PERGAMON

Vision Research 41 (2001) 2799–2810

Vision Research

www.elsevier.com/locate/visres

A longitudinal study of the biometric and refractive changes in full-term infants during the first year of life

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Received 26 September 2000; received in revised form 16 February 2001

Abstract

Changes in ocular axial dimensions and refraction were followed longitudinally, using ultrasonography and retinoscopy, during the first year of life (mean ages 4–53 weeks) of a group of 20 full-term infants (10 male, 10 female). Using a mixed-model regression analysis, axial length changes as a function of time were found to be best described by a quadratic expression ($AL = 17.190 + 0.128x - 0.0013x^2$, where AL is the axial length in mm and x is the age in weeks), while anterior chamber depth changed linearly ($ACD = 2.619 + 0.018x$, where ACD is the anterior chamber depth in mm): lens thickness was essentially constant. Spherical equivalent refraction through most of the first year showed a steady reduction in hypermetropia ($SER = 2.982 - 0.032x$, where SER is the spherical equivalent refraction in dioptres): astigmatism also tended to diminish. Mean hyperopic refractive errors through the year were negatively correlated with corresponding axial lengths ($SER = 12.583 - 0.541AL$), but some individual subjects showed marked departures from this pattern. These results are discussed in relation to concepts of emmetropization.

Keywords: Axial length; Emmetropization; Hypermetropia; Infant; Refraction

1. Introduction

Recent years have seen increasing acceptance of the view that the refractive state of the adult eye is the product of both genetic and environmental influences (e.g. McBrien & Barnes, 1984; Young & Leary, 1991; Troilo, 1992). Moreover, although refractive change can occur throughout life, the most critical developments occur during infancy, when the various ocular biometric parameters are changing most rapidly (e.g. Larsen, 1971a,b,c,d). These changes in growth are accompanied by a large change in the overall dioptric power of the eye which decreases from about 90 dioptres (D) at birth to 75 D at 12 months of age (e.g. Luyckx, 1966; Grignolo & Rivara, 1968). This raises the question of how these various changes are orchestrated to bring about a refractive state of near-emmetropia in adulthood.

Although many authors have found that the distribution of refractive errors at birth is approximately normal (e.g. Cook & Glasscock, 1951; Banks, 1980; Hirsch & Weymouth, 1991), by 12 months of age, the distribution has become highly curtosed with a peak close to emmetropia (Ingram, 1979; Gwiazda, Thorn, Bauer, & Held, 1993; Ehrlich, Atkinson, Braddick, Bobier, & Durden, 1995; Wood, Hodi, & Morgan, 1995). This remains so at 6 years (Sorsby, Benjamin, Davey, Sheridan, & Tanner, 1957; Gwiazda et al., 1993) and in adulthood (Stenstrom, 1946; Sorsby, Sheridan, Leary, & Benjamin, 1960), even though individual ocular components remain approximately normally distributed (Stenstrom, 1946; Sorsby, Benjamin, Sheridan, & Leary, 1961). Thus, longitudinal information on the biometric changes occurring in the individual infant

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0042-6989/01/$ - see front matter
PII: S0042-6989(01)00169-9

eye, and their relationship to the overall refraction of the eye, is of great interest because there must be some combination of passive and active emmetropization mechanisms that tends to correlate components in such a way as to produce approximate emmetropia.

There have been a number of cross-sectional studies concerned with the ocular biometry of both premature (O'Brien & Clark, 1994) and full-term (Larsen, 1971a,b,c,d; Blomdahl, 1979) babies. However, only Hirano, Yamamoto, Takayama, Sugata, and Matsuo (1979) have carried out limited longitudinal measurements on axial length and other biometric changes (but not refraction) during the first year of life. For reliable results to be obtained, free of artefacts due to varying levels of accommodation, it is important that measurements be carried out under cycloplegia.

The aim of the present study was, then, to obtain longitudinal data on the growth and refraction under cycloplegia of the normal, full-term infant eye during the first year of life.

2. Methods

2.1. Subjects

Twenty full-term white infants (10 male, 10 female) were recruited on a voluntary basis at the antenatal unit at the University Hospital, Aintree (formerly Fazakerley Hospital). All infants had an uneventful birth history and were born between 39 and 42 weeks of gestation: in no case was ocular pathology present. Parents received a written explanation of all procedures, and informed consent was obtained at the first examination when each infant was approximately 4 weeks old. Subsequent appointments were scheduled for 13, 26, 39 and 52 weeks of age. All measurements were made after cycloplegia: this was achieved with 0.5% cyclopentolate at weeks 4 and 13 and 1% cyclopentolate at the remaining visits.

2.2. Measurement techniques

Axial length (AL), anterior chamber depth (ACD) and lens thickness (LT) were measured using through-the-eyelid ultrasonic biometry as validated by Twelker, Kirschbaum, Zadnik, and Mutti (1997) and Laws, Laws, Wood, and Clark (1998). This is better tolerated by infants than conventional methods and requires no corneal anaesthetic. This method produces reliable and valid measurements: for adults, the difference in readings between visits has a mean and standard deviation of 0.01 ± 0.16 mm, while the mean difference in axial length AL* from the posterior surface of the cornea between through-the-lid and contact biometry is 0.05 ± 0.16 mm (Laws et al., 1998). The instrument employed was a Zeiss–Humphrey ultrasonic biometer, model 820. Readings through the eyelid were obtained using a commercial tear substitute (Visco Tears) as a contact solution. The aim was to record three satisfactory single traces for each eye, although this was not always achieved, due to such reasons as the baby crying. Preliminary experiments showed that, for infants and with careful selection of A-scan traces, through-the-eyelid measurements of lengths from the posterior surface of the cornea (AL* and ACD*) could be corrected to axial lengths and anterior chamber depths (AL and ACD) from the anterior surface of the cornea, as normally specified, by adding 0.70 mm. This factor represents a combination of corneal thickness and small systematic errors in the method (Laws et al., 1998). Corneal thickness is known to decrease from a value of about 0.61 to 0.66 mm at birth (Autzen & Bjornstrom, 1991; Remon et al., 1992) to about 0.57 mm over the first few months of life (Autzen & Bjornstrom, 1991) (although Ehlers, Sorensen, Bramsen, & Poulsen, 1976 give the smaller figures of 0.54 mm at birth, decreasing to 0.52 mm by the age of about 3 years). The mean adult thickness is about 0.52–0.56 mm (Martola & Baum, 1968; Waltman & Hart, 1987). Inter-subject variation in corneal thickness occurs at all ages. Thus, the use of a constant correction factor for all AL* and ACD* data necessarily involves possible errors of a few hundredths of a millimetre, but this was felt to be justifiable in relation to the much greater magnitude of the general biometric changes under investigation.

Refractive error was determined 30 min after instillation of the cycloplegic, using streak retinoscopy and hand-held lenses. Several measurements were taken (typically four) to ensure that the result remained stable and to confirm that an adequate depth of cycloplegia had been achieved (Hodi & Wood, 1994; Wood et al., 1995). In cases where changes in the refractive finding suggested the presence of residual accommodation, a further drop of cycloplegic was inserted, and the refractive measurements were repeated after a suitable time interval. Results were recorded in sphero-cylindrical form and transformed as required to spherical equivalent refractions (SER) using the formula $SER = S + C/2$, where S and C were, respectively, the spherical and cylindrical components of the refraction.

The ocular components and refraction were measured by one examiner (FP) and confirmed by a second (ICJW). Agreement was normally within ± 0.25 D for both sphere and cylinder components. In the few cases where larger discrepancies were found, measurements were repeated until the examiners' results converged.

2.3. Statistical methods

Longitudinal data present special challenges for analysis because different observations from the same child

may be correlated, and simple regression does not account for this potential lack of independence. Many common methods for analysing longitudinal data require that the same number of observations be gathered from each subject. Obvious problems therefore arise if there is a failure of all infants to attend all the scheduled sessions or an absence of some data due to the lack of cooperation of the infants on some visits. While it is possible to overcome these difficulties by interpolating any missing measurements or omitting subjects with incomplete data sets, neither of these approaches is very satisfactory. A mixed model analysis was therefore used (Diggle, Liang, & Zeger, 1994). This accounted for correlation between observations, while still using all available information from each subject.

The biometric data were entered into a spreadsheet and analysed using SAS PROC MIXED statistical library program (SAS Institute, Cary, NC). Both linear and quadratic mixed-model regressions were used. The linear model allowed the intercept and slope to vary across the children, while in the quadratic model, the curvature could also vary. The models allowed, as appropriate, the intercept and the regression coefficients of the linear and quadratic terms to vary across the children.

3. Results

3.1. Attendance

Potentially, there were 100 subject visits. One subject dropped out after the first visit. Including this case, there was a total of 16 episodes of non-attendance, the numbers of children attending the sequence of five visits being 20, 19, 18, 15, and 12. In addition, on a few occasions, lengths could not be recorded because of lack of cooperation from the infant (see Table 1). In practice, post-birth visit times varied slightly from the intended values and were 4.3 ± 0.9, 14.0 ± 1.9, 27.3 ± 1.7, 40.0 ± 1.9 and 53.1 ± 1.6 weeks (ranges 3–7, 11–18, 25–31, 37–44, 52–56 weeks, respectively).

3.2. Correlation between eyes

Analysis of the differences in axial lengths (AL*) between the right and left eyes and also those between the spherical equivalent refractions (SER), using two-way ANOVAs, showed that there were no statistically significant differences between the eyes ($P = 0.884$ for axial length and $P = 0.977$ for SER) when the data were controlled for patient effect and age. For this reason, all subsequent analyses were in terms of the data for right eyes only.

3.3. Changes in axial length

Fig. 1 shows the axial length of the right eye, AL*, as measured from the posterior surface of the cornea to the anterior surface of the retina, as a function of age for individual subjects. The overall means and standard deviations are given in Table 1: note again that different numbers of subjects contribute to each mean. Examination of Fig. 1 suggests that there is variation in the initial AL*. Growth rates appear broadly similar for all subjects but may slow with increasing age.

The mixed-model regression fits to the data over the first year of life are:

$$\text{Linear } \mathrm{AL}^* = 17.004 + 0.063x \quad r^2 = 0.88$$

$$\text{Quadratic } \mathrm{AL}^* = 16.490 + 0.128x - 0.0013x^2 \quad r^2 = 0.96$$

where x is the age in weeks, and AL* is in millimetres. The quadratic model produces a substantially higher r^2 value, supporting the suggestion that growth slows with increasing age.

Using the quadratic model, it can be seen that

$$\mathrm{d}(\mathrm{AL}^*)/\mathrm{d}x = 0.128 - 0.0026x.$$

Thus the quadratic model indicates that the growth rate decreases from about 0.12 mm/week at 5 weeks of age to almost zero by the end of the first year of life. In practice, growth undoubtedly continues well beyond the first year into adolescence (e.g. Sorsby et al., 1961; Larsen, 1971d; Gordon & Donzis, 1985; Young & Leary, 1991), so that the quadratic model has no valid-

Table 1
Means and standard deviations for the biometric parameters and spherical equivalent refractions of the right eyes of all subjects at different ages

PRIVATE Age (weeks)	Axial length, AL* (mm)	Anterior chamber depth, ACD* (mm)	Lens thickness, LT (mm)	Spherical equivalent refraction (D)
4.3 ± 0.9 (20)	17.01 ± 0.41 (20)	1.91 ± 0.27 (19)	3.70 ± 0.22 (19)	$+2.81 \pm 0.94$ (20)
14.0 ± 1.9 (19)	17.99 ± 0.67 (19)	2.24 ± 0.31 (19)	3.65 ± 0.25 (19)	$+2.74 \pm 1.46$ (19)
27.3 ± 1.7 (18)	19.07 ± 0.75 (17)	2.47 ± 0.28 (16)	3.63 ± 0.23 (16)	$+1.91 \pm 1.31$ (18)
40.0 ± 1.9 (15)	19.46 ± 0.90 (13)	2.63 ± 0.30 (12)	3.58 ± 0.24 (12)	$+1.76 \pm 1.50$ (15)
53.1 ± 1.6 (12)	19.71 ± 0.87 (10)	2.81 ± 0.26 (10)	3.65 ± 0.14 (10)	$+1.50 \pm 1.42$ (12)

Note that the values for axial length, AL*, and anterior chamber depth, ACD*, are measured from the posterior surface of the cornea, rather than the anterior surface, due to the use of through-the-eyelid ultrasound biometry. It was not possible to measure ocular dimensions for every infant on every visit, and the bracketed figures give the number of infants contributing to each mean.

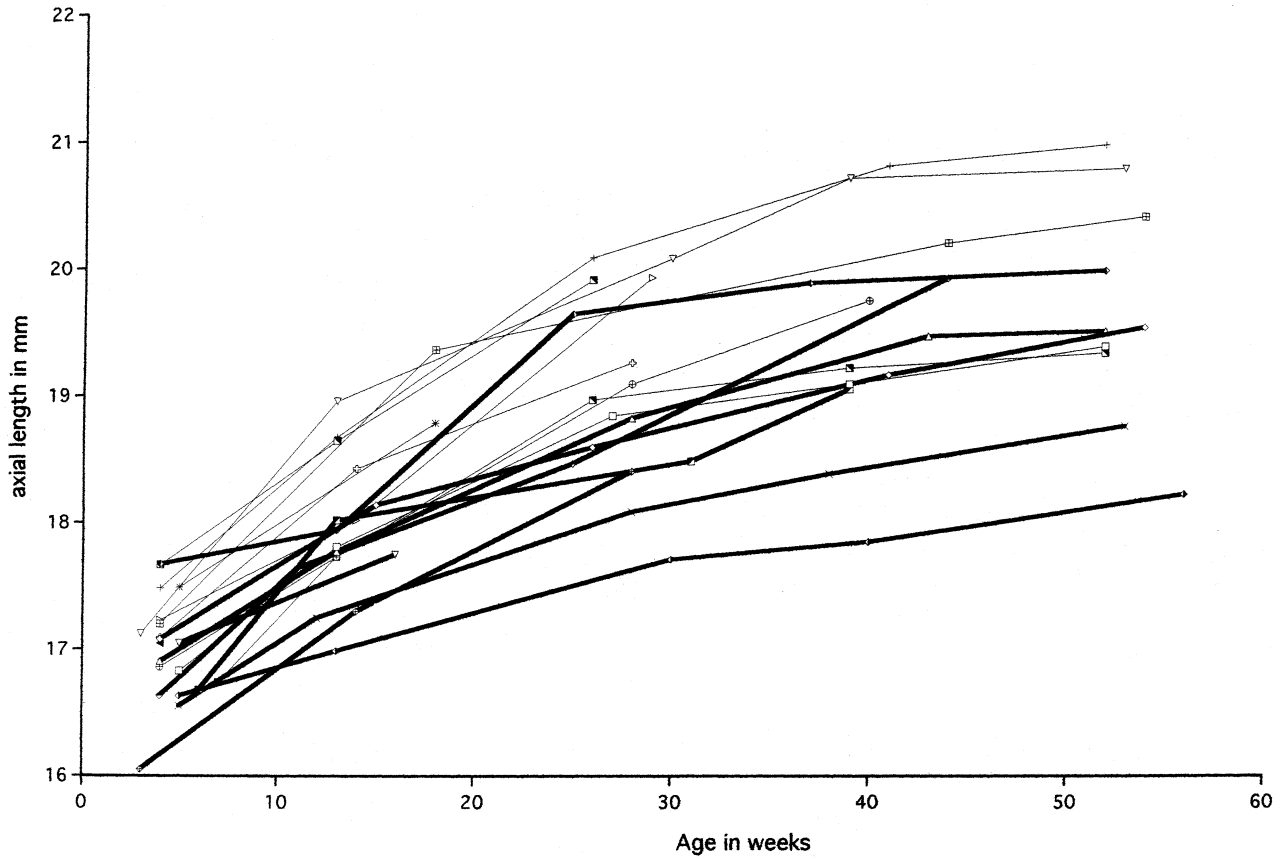

Fig. 1. Axial length, AL*, as a function of age for the right eyes of individual subjects. AL* is the axial length as measured from the posterior surface of the cornea. Thicker lines identify female subjects.

ity beyond the timespan of the data. This model suggests that the initial axial lengths differ significantly among the children ($P = 0.013$), but that the growth rates do not ($P = 0.999$).

Analysis of the data in terms of the gender of the infants suggests that while the axial lengths were very similar at the time of the first measurements (AL* = 17.09 ± 0.46 mm for the boys and 16.91 ± 0.33 mm for the girls, not significantly different in either mean or variance), initial eye growth was somewhat slower for girls than boys. The quadratic regression equations are:

$$\text{Males } AL^* = 16.486 + 0.154x - 0.002x^2$$

$$\text{Females } AL^* = 16.493 + 0.102x - 0.0012x^2.$$

There was no correlation between birthweight and axial length at the first visit.

3.4. Changes in anterior chamber depth, ACD*

Values of ACD*, as measured from the posterior surface of the cornea, for individual subjects are illustrated in Fig. 2. Both the individual data and their means (Table 1) suggest that the age trends are roughly linear. The mixed-model linear regression equation is:

$$ACD^* = 1.919 + 0.018x \quad r^2 = 0.78.$$

There are statistically significant differences between the initial values of ACD* for different subjects ($P = 0.019$) but not between their growth rates ($P = 0.47$). A quadratic model gives a negligible improvement in r^2 (to 0.80), so that the linear model appears adequate.

When the data were divided according to gender, there were no significant differences with respect to anterior chamber depth.

3.5. Lens thickness, LT

The individual data are shown in Fig. 3: note the expanded ordinate scale. Although, on this expanded scale, there appear to be substantial changes in thickness between visits, these changes are generally no larger than would be expected from the reliability of the measurement technique. Unlike axial length and anterior chamber depth, there appears to be no systematic change with age in the lens thickness over the age range studied (see also Table 1 for mean values). The slope of the linear regression equation

$$LT = 3.684 - 0.002x$$

does not differ significantly from zero. Dividing the data according to the gender of the subjects showed that there were no significant differences between the lens thicknesses of boys and girls.

3.6. Refraction

Changes in spherical equivalent refraction, SER, with age are illustrated in Fig. 4, mean values being given in Table 1. For most subjects, there is a tendency for the early hyperopic refractive error to decrease towards emmetropia over the first year of life, but there are some obvious exceptions to this trend. In particular, the refraction of one subject with a modest amount of hypermetropia at the age of about 5 weeks increases to a relatively high level of hypermetropia (about +5 D) by 3 months and then remains essentially stable through the rest of the first year of life, while another has become slightly myopic by about 9 months.

The mixed model gives a linear regression equation:

$$\mathrm{SER} = 2.982 - 0.032x \qquad r^2 = 0.59.$$

Analysis in terms of gender showed that there was no statistically significant variation between the sexes with respect to SER.

Amounts of astigmatism recorded were generally modest (≤ 2.00 DC for all subjects at all times), and no infant had clinically significant (≥ 1.00 DC) astigmatism at the end of their first year of life. In most instances, any astigmatism was with-the-rule (Table 2).

4. Discussion

This study has obvious limitations imposed by the size and nature of the subject sample, particularly the limited refractive range present, and the problem of missed visits. However, the results give, for the first time, a reasonably clear longitudinal picture of the associated changes in axial length, anterior chamber depth, lens thickness and refraction that occur during the first year of life. This picture complements that already obtained for slightly older children (e.g. Sorsby et al., 1957, 1961; Sorsby & Leary, 1970; Ehrlich 1995, 1996)

4.1. Axial dimensions

The results for axial dimensions emphasise the well-recognised fact that growth in the young eye is not a simple increase in scale but rather that different parts of

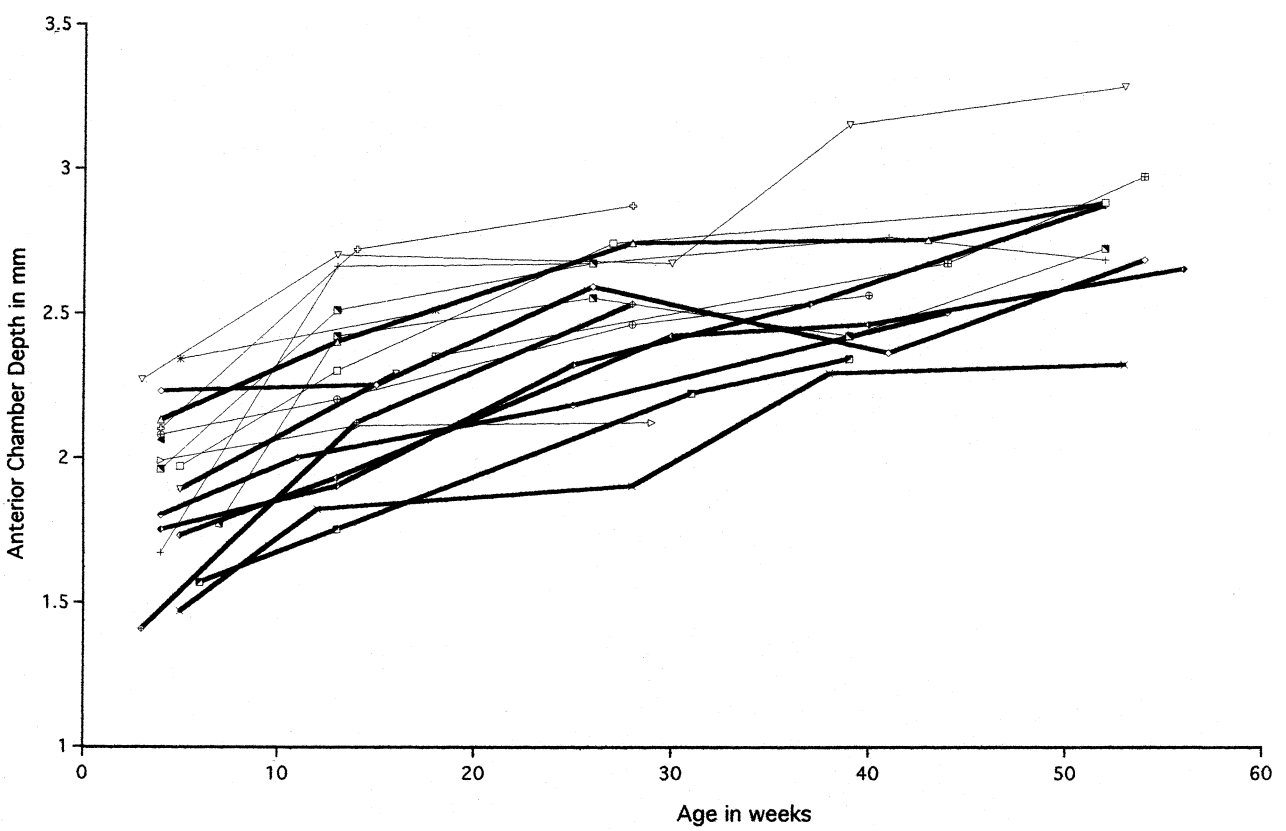

Fig. 2. Anterior chamber depth, ACD*, as a function of age for the right eyes of individual subjects. ACD* is the anterior chamber depth as measured from the posterior surface of the cornea. Thicker lines identify female subjects.

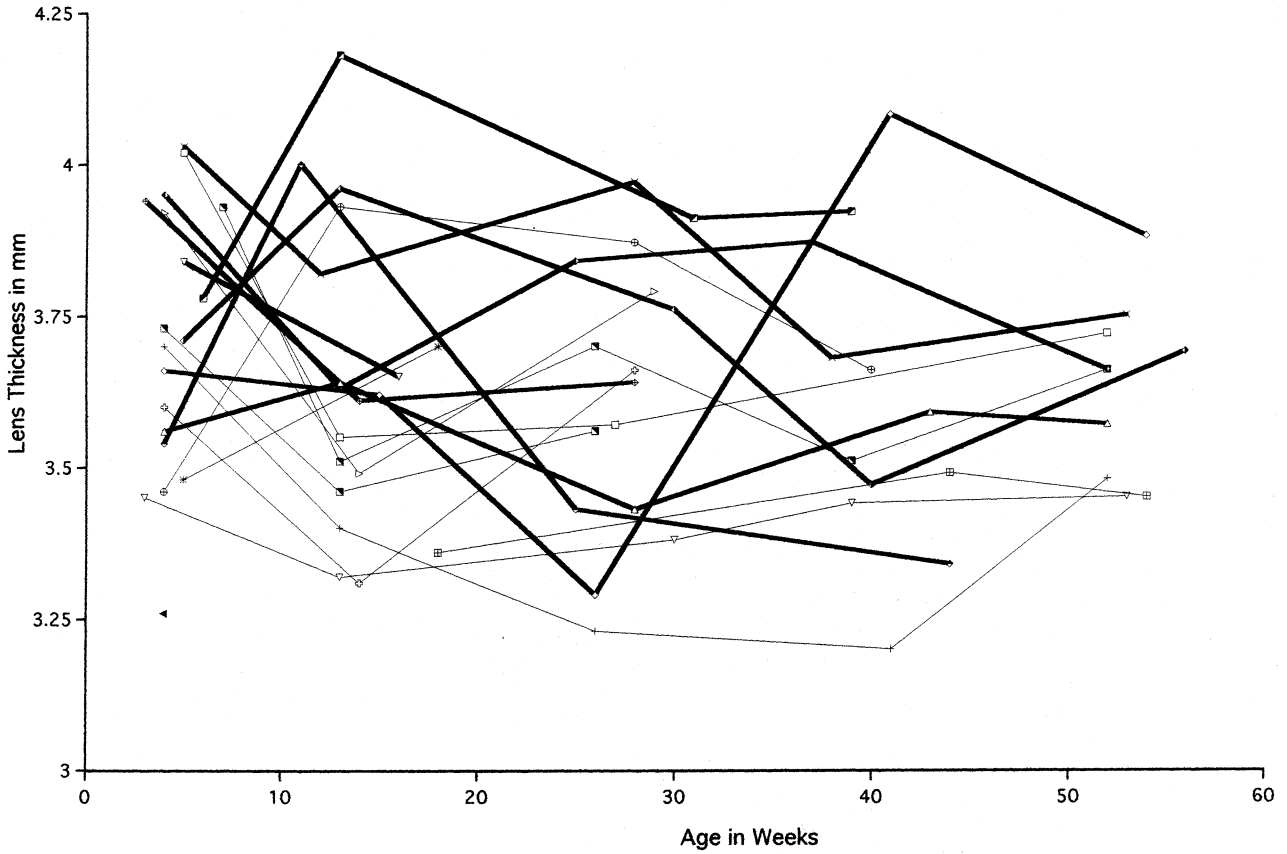

Fig. 3. Lens thickness, LT, as a function of age for the right eyes of individual subjects. Note that the ordinate scale is expanded in comparison with earlier figures. Thicker lines identify female subjects.

the eye grow at different rates (e.g. Spooner, 1957; Lotmar, 1976; Day, 1997; Mutti & Zadnik, 1997; Oyster, 1999). The regression analyses suggest that axial length growth is curvilinear, while the anterior chamber depth grows linearly. Moreover, if allowance is made for the fact that to give true axial lengths, AL, and anterior chamber depths, ACD, the values of AL* and ACD* should each be increased by 0.7 mm (Laws et al., 1998), between the ages of 5 and 50 weeks, true axial length increases by about 18%, while the true anterior chamber depth increases by 34%. The study found no evidence for any associated change in lens thickness. Perhaps unsurprisingly, axial lengths for boys tended to be slightly greater than those for girls (Sorsby et al., 1961; Larsen, 1971d).

To illustrate the lack of simple scaling more clearly, Fig. 5 shows the relative changes in AL, ACD and LT in terms of both the regression equations and the mean data. The changes have been normalised to unity at birth, although strictly, the validity of the regression equations only extends over the age range (approximately 4–53 weeks) during which measurements were made. Note that because different numbers of subjects contribute to each mean, the mixed-model regression equations should give a more reliable indication of age trends than the means.

Evidently, the finding that the older eye is not simply a scaled up version of the infant eye considerably weakens the argument that the reduction in hypermetropia occurring with growth simply reflects the scaling down of a proportional error as the overall power of the eye reduces (Hofstetter, 1969; Edwards, 1992). Some form of mechanism to appropriately correlate the differential growth of different components must be required.

Considering now the individual parameters, when corrected as necessary for corneal thickness, our mean values of 17.7 ± 0.4, 2.5 ± 0.3 and 3.7 ± 0.2 mm at the age of about 4 weeks for AL, ACD and LT, respectively, agree reasonably well with those elsewhere in the literature for neonates in the first month of life. Direct comparison is difficult, since different investigators use different methodology and infants of varying ages, genders and racial origins. However, mean axial lengths recorded, almost entirely by ultrasound, for groups of full-term infants of ages between 1 day and 3 weeks lie between 16.4 and 18.1 mm (Sorsby & Sheridan, 1960; Gernet, 1964; Luyckx, 1966; Grignolo & Rivara, 1968; Larsen, 1971d; Blomdahl, 1979; Hirano et al., 1979; Gordon & Donzis, 1985; Fledelius, 1992; Isenberg et al., 1995). In some instances, the values in these studies

may be a little low, since applanation of the cornea by the direct contact of the ultrasound probe may have resulted in a shortening of the measured length by about 0.2 mm (Shammas, 1984).

The growth rate at birth deduced from the present data, of about 0.13 mm/week, is very similar to the value of 0.14 mm/week found by Fledelius (1992).

Only Hirano et al. (1979) give longitudinal data on change in axial length over the first 16 months of life, for a group of full-term Japanese infants. A curvilinear regression fit to their data gives an equation

$$AL = 17.89 + 0.147x - 0.0015x^2.$$

Apart from the slightly higher constant term, this is very similar to our own regression when corrected for the effect of corneal thickness, i.e.

$$AL = 17.19 + 0.128x - 0.0013x^2.$$

The rate of growth given by the quadratic regression equation for the Hirano data is

$$d(AL)/dx = 0.147 - 0.003x.$$

Interestingly, this declines to zero at about 49 weeks, almost the same value as can be deduced from the quadratic fit to our own data. Given the fact that, in practice, the eye continues to grow well beyond one

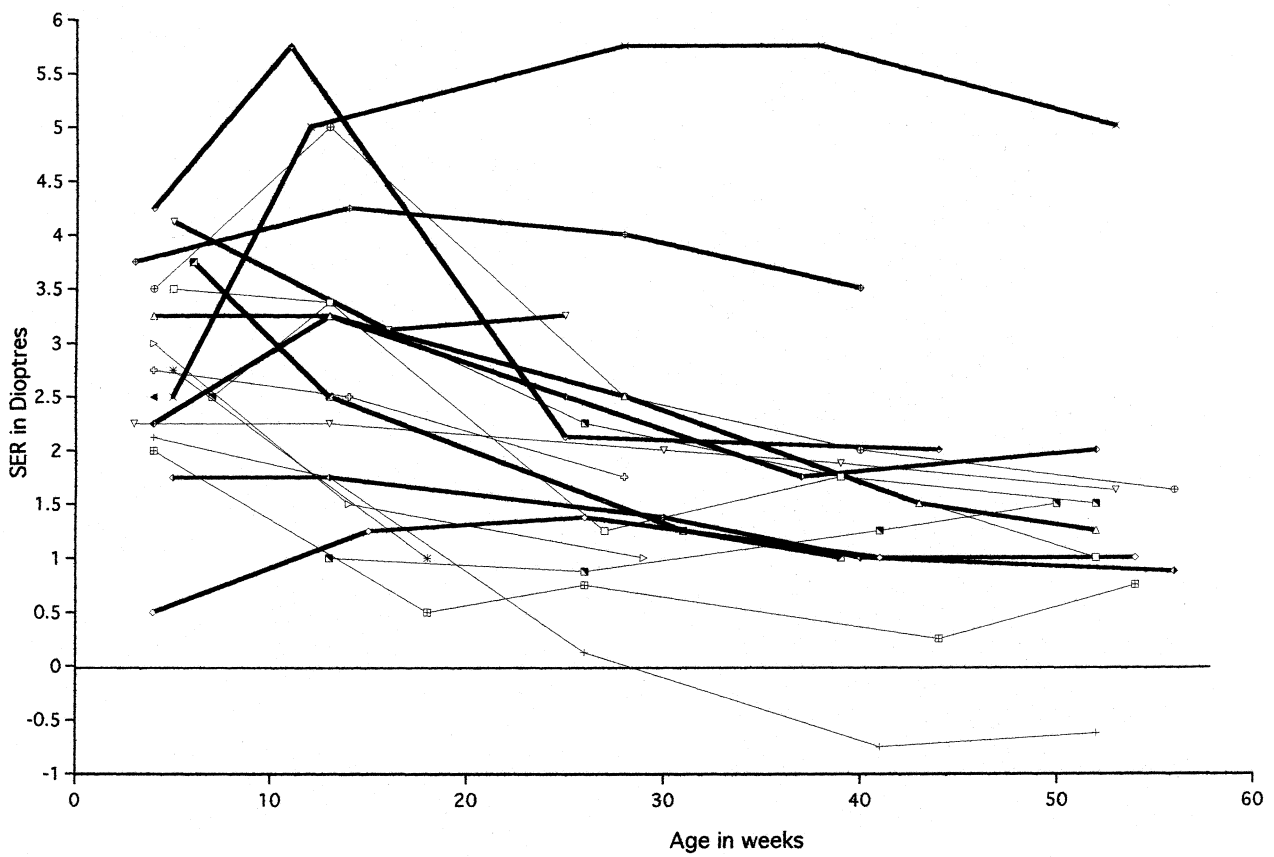

Fig. 4. Changes in mean spherical equivalent refraction, SER, for the right eyes of individual subjects. Thicker lines identify female subjects.

Table 2
Characteristics of infant astigmatism as a function of age

PRIVATE Age (weeks)	Percentage of subjects with astigmatism $\geq$0.25 DC	Percentage of with-the-rule astigmatism	Percentage of subjects with astigmatism $\geq$1.00 DC	Mean astigmatism (DC)	S.D. (DC)
4.3 $\pm$ 0.9 (20)	74	80	21	0.53	0.42
14.0 $\pm$ 1.9 (19)	74	92	32	0.66	0.59
27.3 $\pm$ 1.7 (18)	76	92	29	0.72	0.66
40.0 $\pm$ 1.9 (15)	60	78	20	0.42	0.40
53.1 $\pm$ 1.6 (12)	42	40	0	0.21	0.28

The bracketed figures in the age column give the number of subjects of each age. Note that the individual values of astigmatism for each group are not normally distributed.

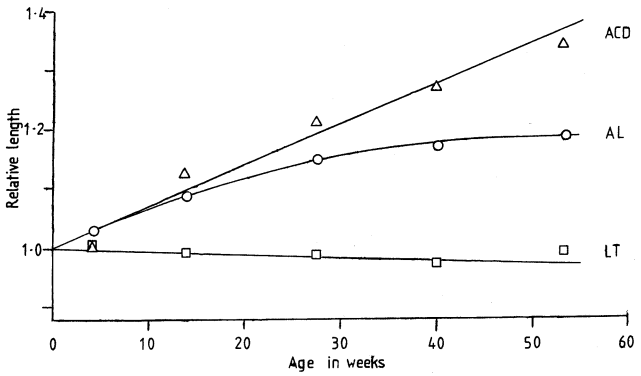

Fig. 5. Relative changes in axial length (AL), anterior chamber depth (ACD) and lens thickness (LT) during the first year of life. The data are normalised to the values at birth obtained from the regression equations (see text). The smooth curves are from the regression equations, and the data points are mean experimental values.

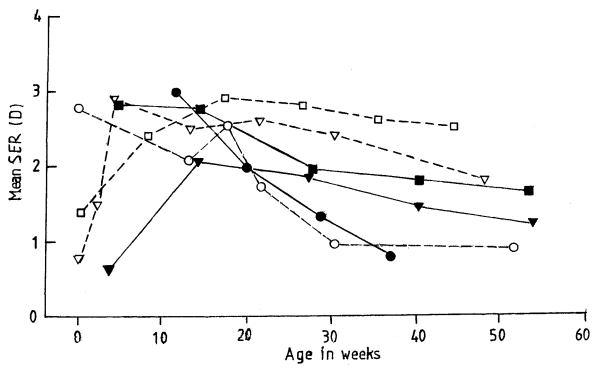

Fig. 6. Spherical equivalent refraction as a function of age for full-term infants, including the results of the present study. Full lines represent longitudinal studies, dashed lines transverse studies. ■: present study; ●: Edwards (1991); ▼: Wood et al. (1995); ▽: Santonasto (1930); □: Grignolo and Rivara (1968); ○: Thompson (1987).

year, it thus appears that the quadratic fits to axial growth are not valid beyond the time span of the data. It is clear from Fig. 4 that growth rates are slowing through the first year of life, so that a linear fit is inappropriate. As the quality and time span of the available data improve, it is, then, likely that a higher-order model will be found to be optimal.

The axial lengths at 26 and 39 weeks quoted by Baldwin (1990) and Larsen (1971d) as a result of transverse studies are slightly lower than those found by either Hirano et al. (1979) or us, but are based on very small numbers of subjects.

The present mean values of anterior chamber depth (ACD) and lens thickness (LT) in the first few weeks of life (about 2.5 ± 0.3 and 3.7 ± 0.2 mm, respectively) are compatible with those found by earlier authors (ACD 2.4–2.9 mm, LT 3.4–4.0 mm, e.g. Gernet, 1964; Luyckx, 1966; Larsen, 1971a,b; Blomdahl, 1979; Hirano et al., 1979; Fledelius, 1992). Not all authors used cycloplegia: it is possible that this may have affected their LT results, since an eye under cycloplegia might be expected to have flatter lens curvatures and reduced LT. The longitudinal data of Hirano et al. (1979) suggest that ACD increases at an almost constant rate (about 0.024 mm/week, somewhat faster than our value of 0.018 mm/week) up to the age of about 8 months and then tends to stabilise. The more limited results of Larsen (1971a,b) are again compatible with ours. Our finding of an apparent lack of change in LT concurs with the result found by Hirano et al. and presumably implies that as the lens grows in equatorial diameter, its surfaces become flatter (Mutti et al., 1998).

4.2. Refraction

Although there are individual exceptions (Fig. 4), our results accord with the well-established trend of generally reducing hypermetropia during the first year of life (see, for example, Saunders, 1995 for a review). The r^2 value for the mixed model linear regression fit is lower ($r^2 = 0.59$) than those for the dimensional parameters. This is not surprising, since it reflects the fact that refractive error depends upon the combined interaction of a large number of parameters (dimensional, curvature and index), leading to greater individual variation in the pattern of age-related change. Glickstein and Millodot (1970) argued that the retinoscopic finding of hypermetropia might be an artefact caused by the primary reflecting layer of the retina lying anterior to the receptor layer, but comparisons of refractive results using both retinoscopy and visual evoked potentials in various animals (Meyer & Schwassmann, 1970; Millodot & Blough, 1971; Millodot, 1971; Hughes, 1977; Mutti, Zadnik, Johnson, Howland, & Murphy, 1992) suggest that any such effect is likely to be small and that it would not affect the age trend of the results.

Fig. 6 compares the changes in our mean spherical equivalent refractions with those found by earlier authors in both transverse and longitudinal studies.

When comparing the various results illustrated in Fig. 6, the existence of standard deviations of at least 1 D on all the data points must be remembered, although for clarity, these uncertainties are omitted from the figure. Absolute values of refractive error may also be affected by the nature of the cycloplegics used (e.g. Ingram, 1979; Ingram & Barr, 1979b). With these provisos, the present data do not confirm the relatively large increase in hypermetropia over the first three months of life that is recorded in some studies (Santonasto, 1930; Grignolo & Rivara, 1968; Wood et al., 1995), although some of our individual subjects do show this effect (Fig. 4). Saunders (1995) has discussed possible reasons for such discrepancies in terms of differences in the ways in which infants are classified into age groups and the problems of retinoscopy on

very young infants. Although the study by Wood et al. (1995) used very similar techniques to those employed in the present work, the mean age of their subjects at the first visit was only 2 weeks, whereas that in the present study was 4.3 weeks. We cannot exclude, then, the possibility of a rapid early increase in hypermetropia around birth, as was also observed in the transverse study of Gordon and Donzis (1985). Ehrlich et al. (1995, 1997) have hypothesised that 'emmetropization' is, in fact, more of a trend towards low hyperopia (+0.50 to +1.00 D) rather than towards zero refractive error. Hence initial increases in hypermetropia (Santonasto, 1930; Grignolo & Rivara, 1968; Wood et al., 1995) are more likely to be recorded if the population includes a large proportion of infants who are born with myopia or very low hypermetropia: this was not the case with the present population sample.

Although Saunders, Woodhouse, and Westall (1995) suggested that the rate of decrease of hypermetropia between the ages of about 3 and 15 months increases linearly with the initial refractive error, the individual patterns of change shown in Fig. 4 suggest that this may be an over-simplification, possibly because the rates of change in the Saunders et al. study were based on only two measurements of refraction for each subject. Similarly, the suggestion by Ehrlich et al. (1997) that the refractive change after 9 months is proportional to the initial error may be more true of the group than the individual. Ehrlich et al. (1997) found that infants tend to lose about one-third of their spherical equivalent refraction over the age interval 9–21 months, a figure in satisfactory agreement with our observed loss of about half the initial error over the first year when the slowing of refractive change with age is taken into account. Ingram and Barr (1979a) have suggested that if, at the age of 1 year, hypermetropia in one principal meridian exceeds +2.5 D, there will be no later systematic trend towards emmetropia, emmetropization only occurring for lower levels of hypermetropia or if either meridian is myopic.

If our admittedly limited data on astigmatism are considered, the percentages of subjects of various ages with astigmatism equal or greater than 1 DC (Table 2) are compatible with those found in earlier studies (see, for example, Saunders, 1995 for review). The results for astigmatic axes support some recent findings that early infant astigmatism is mostly with-the-rule (e.g. Edwards, 1991; Hopkisson, Arnold, Billingham, McGarrigle, & Shribman, 1992; Saunders et al., 1995) However several other previous studies (e.g. Howland, Atkinson,

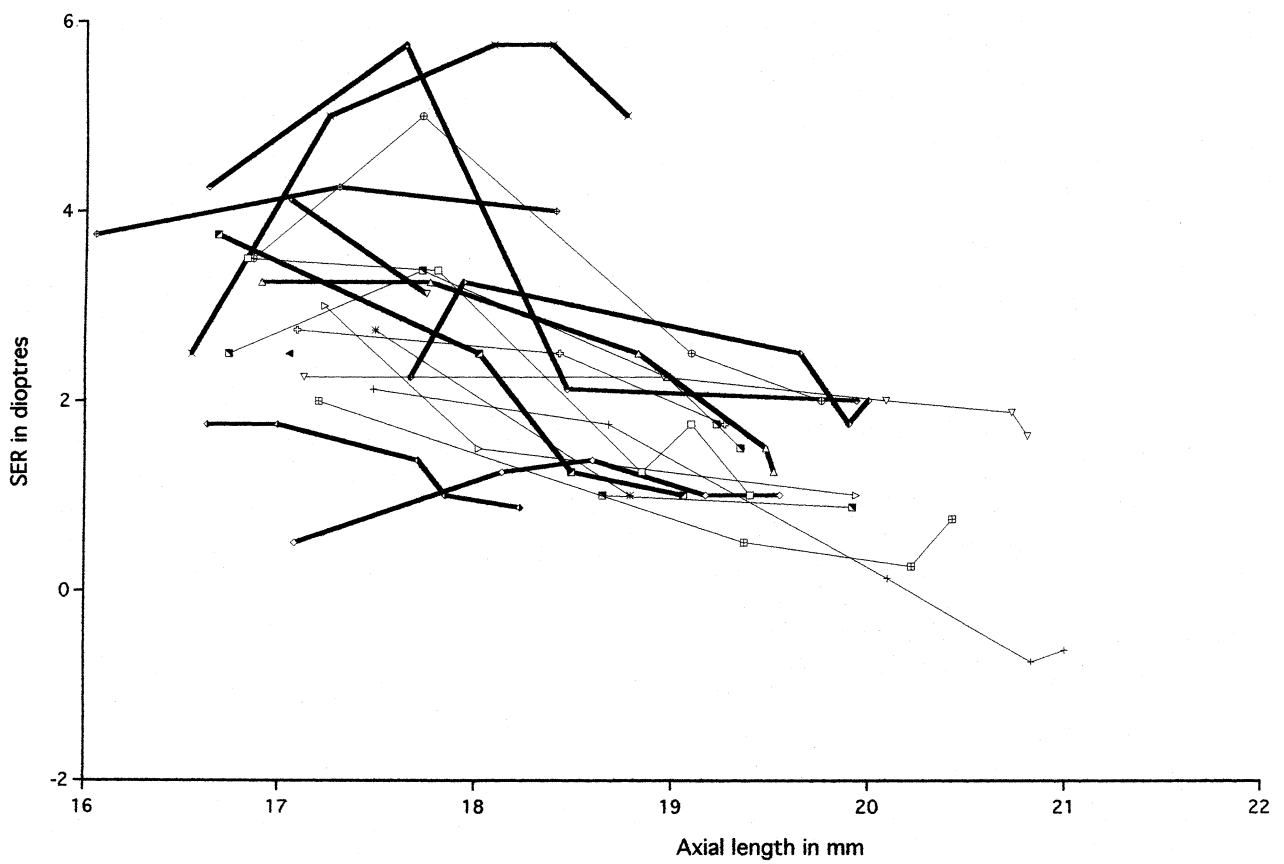

Fig. 7. Longitudinal changes in spherical equivalent refractions, SER, as a function of the axial lengths, AL*, recorded at the same ages, for each subject. As each eye grows, there is a general tendency for hypermetropia to reduce, but some individual eyes do not follow this pattern. Thicker lines identify female subjects.

Braddick, & French, 1978; Mohindra, Held, Gwiazda, & Brill, 1978; Atkinson, Braddick, & French, 1980; Gwiazda, Scheiman, Mohindra, & Held, 1984; Howland & Sayles, 1984, 1985; Gwiazda, Grice, Held, McLellan, & Thorn, 2000) have observed that against-the-rule astigmatism prevails. It may be that these differing findings are due to the different study populations involved or to the use of differing criteria for what constitutes clinically significant amounts of astigmatism (see, for example, Saunders, 1995). All studies agree that the level of astigmatism declines towards adult levels during the first year of life.

In general, the observed, naturally occurring, general trend towards emmetropia (or, possibly, low hypermetropia) over the first year of life supports the view that caution should be exercised when considering the optical correction of refractive errors in infants, since any corrective lenses may interfere with the emmetropization process (e.g. Medina, 1987; Ingram, Arnold, Dally, & Lucas, 1991; Wildsoet, 1997; Smith & Hung, 1999).

4.3. Correlation of changes in spherical equivalent refraction with those in axial length

Stenstrom (1946), in exploring the correlation between his measurements of axial length, corneal curvature and anterior chamber depth with the corresponding refractive errors, concluded that only in the case of axial length was there any meaningful correlation ($r^2 = 0.58$). Young and Leary (1991) suggested that a somewhat better correlation with refraction could be obtained by using vitreous chamber depth rather than the full axial length. Van Alphen (1961) thought that axial length and corneal power combined were the most important determinants of the overall refraction.

Fig. 7 plots the longitudinal values of spherical equivalent refraction, SER, for each subject against the corresponding values of axial length, AL*. The mixed-model linear regression equation describing the data is:

$$\mathrm{SER} = 12.204 - 0.541\mathrm{AL}^*, \text{ giving } r^2 = 0.54.$$

Since $\mathrm{AL}^* = \mathrm{AL} - 0.700$ mm, we can rewrite the equation in terms of the full axial length as

$$\mathrm{SER} = 12.583 - 0.541\mathrm{AL}.$$

If Fig. 7 is examined in more detail, it is of interest that the subject who remains strongly hypermetropic at the age of about 1 year has a slightly shorter eye than is typical for the group, while the subject who becomes myopic has a longer eye. It is evident, however, that possession of a short or long eye at the age of 1 year does not necessarily imply an unusual value of refraction. Further longitudinal studies are in progress, which will yield values for corneal curvature as well as axial dimensions, and these should further clarify the origins of abnormal values of infant refraction.

5. Conclusions

The present longitudinal study confirms that, during the first year of life, growth is not a simple scaling process in which the relative dimensions of all ocular components change equally, but rather one in which different parts of the eye grow at different relative rates. Thus, the proportions of the various components are changing. Growth in axial length is better described by a second-order function of age, while increases in anterior chamber depth are essentially linear: lens thickness is constant. While, over most of the first year, there is an overall trend for the early hypermetropic refractive error to decrease linearly with increase in axial length, there are individual exceptions to this behaviour. Longer or shorter axial lengths than the norm do not necessarily confer an unusual tendency to myopia or hypermetropia.

Acknowledgements

We are grateful for support from the College of Optometrists and the North West Regional Health Authority.

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