The Changing Profile of Astigmatism in Childhood: The NICER Study

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PURPOSE. We performed a prospective study of the changing profile of astigmatism in white school children in Northern Ireland.

METHODS. Of the 399 6- to 7-year-old and 669 12- to 13-year-old participants in Phase 1 of the Northern Ireland Childhood Errors of Refraction (NICER) study, 302 (76%) of the younger and 436 (65%) of the older cohort were re-examined three years later (Phase 2). Stratified random cluster sampling was used. Following cycloplegia (cyclopentolate HCl 1%) refractive error was recorded by the Shin-Nippon-SRW-5000 autorefractor. Astigmatism is defined as ≥1.00 diopters cylinder (DC). Right eye data only are presented.

RESULTS. The prevalence of astigmatism was unchanged in both cohorts: younger cohort 17.1% (95% confidence intervals [CIs], 13.3–21.6) were astigmatic at 9 to 10 years compared to 22.9% (95% CIs, 18.3–28.2) at 6 to 7 years; older cohort, 17.5% (95% CIs, 13.9-21.7) of participants were astigmatic at 15-16 years compared to 18.4% (95% CIs, 13.4–24.8) at age 12 to 13 years. Although prevalence remained unchanged, it was not necessarily the same children who had astigmatism at both phases. Some lost astigmatism (10.0%; CIs, 7.5–13.3 younger cohort and 17.4%; CIs, 13.5–22.2 older cohort); others became astigmatic (9.1%; CIs, 6.7–12.2 younger cohort and 11.6%; CIs, 8.4–15.8 older cohort).

CONCLUSIONS. This study presents novel data demonstrating that the astigmatic error of white children does not remain stable throughout childhood. Although prevalence of astigmatism is unchanged between ages 6 and 7 to 15 to 16 years; during this time period individual children are developing astigmatism while other children become nonastigmatic. It is difficult to predict from their refractive data who will demonstrate these changes, highlighting the importance of all children having regular eye examinations to ensure that their visual requirements are met.

Keywords: astigmatism, childhood, prevalence

Astigmatism is a common refractive error and an important cause of visual impairment. The high prevalence of astigmatism at birth decreases throughout infancy and during the school years prospective studies have reported that the astigmatic profile of a population does not change substantially. However, although the distribution and prevalence of astigmatism in a population or cohort may remain unchanged throughout later childhood, it is not always clear from such studies what happens to an individual’s astigmatic error over this time period.

Data from Phase 1 of the Northern Ireland Childhood Errors of Refraction (NICER) Study, a population-based survey of the prevalence of refractive error in white schoolchildren in the UK, reported a high prevalence of astigmatism (≥1.00 diopters cylinder [DC]) in the right eye of 6- to 7-year-olds (24%; 95% confidence intervals [CIs], 19–30) which was not significantly different from the prevalence in 12- to 13-year-olds (20%; 95% CIs, 14–25). Astigmatic errors of 1.00 DC or more were significantly associated with myopia and hyperopia.

These cross-sectional data cannot assess how the presence of astigmatism in childhood impacts the subsequent development of ametropia. Prospective studies have demonstrated equivocal findings: astigmatism has been demonstrated to be associated with childhood myopia development and progression, but myopia progression over a 3-year period also has been shown to be unrelated to the magnitude of lower degrees of astigmatism (<2 DC). Compared to the large amount of available data on the change with age of refractive parameters, such as spherical errors and spherical equivalent refractive error, there is a paucity of prospective information on the changing profile of individual astigmatism during the school years and it currently is unclear as to whether astigmatism is a cause or effect of ametropia.

The current study compares data from Phases 1 and 2 of the NICER study to describe how the profile of astigmatism changes over a 3-year period in white school children in Northern Ireland and how astigmatism is associated with changes to the spherical component of the refraction.

METHODS
The NICER Study is an ongoing study of the prevalence and progression of refractive error in school children in Northern Ireland. Participants were 6 to 7 and 12 to 13 years old at Phase 1 and at Phase 2 participants were re-evaluated three years after the initial cross-sectional data had been collected. The protocols at Phase 2 were identical to those from Phase 1 and have previously been described in detail. The study was
approved by University of Ulster’s Research Ethics Committee and adhered to the tenets of the Declaration of Helsinki. Stratified random cluster sampling (level of economic deprivation and urban/rural residence) was used to identify schools to participate in the study. Written consent was obtained from parents/guardians. The protocol for data collection included measures of logMAR monocular distance visual acuity and assessment of ocular posture at distance and near using a cover/uncover test. Following cycloplegia (achieved using one drop of cyclopentolate HCl 1%, minims single dose; Chauvin Pharmaceuticals, Romford, UK) refractive error was recorded in negative cylindrical format to the nearest 0.25 D by the Shin-Nippon SRW-5000 autorefractor (Shin-Nippon, Tokyo, Japan), with the representative value from five measurements being used for subsequent analysis. The representative value (RV) is provided by the proprietary software of the Shin-Nippon SRW-5000 autorefractor and although not an arithmetic mean of the readings taken, provides reliable measurements for use in clinical practice and vision science research. The Zeiss IOLMaster (Carl Zeiss, Jena, Germany) was used to measure at least three axial length measurements, five anterior depth measurements, and three corneal radii of curvature (CRC) measurements. Parental myopic status was established using a questionnaire, completed by parents of participants at Phase 1. This questionnaire has been shown to be valid for self-identification of myopia.

Definitions

As all right and left eye refractive data were correlated (Spearman’s $\rho$ 0.24–0.89, all $P < 0.001$), right eye data only have been presented.

Refractive astigmatism, referred to throughout this paper as astigmatism, was provided from the RV of the autorefractor output.

There is no standard definition as to what constitutes a significant level of astigmatism. Currently the American Association for Pediatric Ophthalmology and Strabismus Vision Screening Committee guidelines recommend that for children greater than 49 months old, astigmatism greater than 1.50 D should be detected; however, a survey of UK hospital optometrists reported that 50% of practitioners would consider prescribing for nonoblique astigmatism of $\geq 1.00$ D. In the current study astigmatism is defined as $\geq 1.00$ DC as this also facilitates comparison with previous prevalence data.

Astigmatism was used to subdivide subjects into four categories: subjects who were not astigmatic at Phase 1 or Phase 2, subjects who remained astigmatic between Phases 1 and 2, subjects who became astigmatic between Phases 1 and 2, and subjects who became nonastigmatic between Phases 1 and 2. Change in astigmatism between Phases 1 and 2 for all subjects also is presented. Where astigmatism is increasing, the change is described in terms of a positive figure, where it is decreasing, values are negative. Change in corneal astigmatism is similarly defined.

Change in refraction is presented as change in the spherical component (the least myopic meridian), with negative values indicating a myopic shift and positive values indicating a hyperopic shift in refraction. Change in spherical equivalent refraction (sphere $+ \frac{1}{2}$ cyl, SER) is not used as it is dependent on the astigmatic component of the refraction.

The cylinders and their axes also were converted into vectors. A positive $J_0$ indicates with-the-rule astigmatism and a negative $J_0$ indicates against-the-rule astigmatism. A positive $J_{45}$ indicates the power is greatest at $135^\circ$ and a negative $J_{45}$ indicates the power is greatest at $45^\circ$.

A Geographical Information Systems approach, using unit postcode address information and the Northern Ireland multiple deprivation measure, was applied to assign an area based rank measure of economic deprivation to each child. Population density of the area where the child resided was defined as urban if there were $\geq 10$ persons per hectare and rural if the population density was $< 10$ persons per hectare.

Participants aged 6 to 7 years at Phase 1 and 9 to 10 years at Phase 2 are described as the younger cohort; those aged 12 to
TABLE 1.

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Prevalence (95% CIs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Younger Cohort</td>
<td>Older Cohort</td>
</tr>
<tr>
<td>Prevalence of Astigmatism</td>
<td>65.5 (60.7 to 70.4)</td>
</tr>
<tr>
<td>Between Phase 1 &amp; 2</td>
<td>5.5 (3.0 to 7.8)</td>
</tr>
<tr>
<td>Remained Astigmatic</td>
<td>11.6 (7.4 to 15.8)</td>
</tr>
<tr>
<td>Between Phase 1 &amp; 2</td>
<td>17.4 (15.9 to 20.9)</td>
</tr>
</tbody>
</table>

**Data Analysis**

All statistical analyses were done using Intercooled Stata 13 (StataCorp, College Station, TX, USA). As much of the refractive data are not normally distributed nonparametric analyses have been used where possible. To explore differences between participants and nonparticipants in Phase 2, the χ² test was used for categorical data (sex, spectacle wear at Phase 1, grammar school education, urban residence, and parental myopic status) and the Mann-Whitney U test (equivalent to the Wilcoxon rank sum test) was used for continuous data (Phase 1 SER, sphere, and astigmatism). The Mann-Whitney U test also was used to explore differences in the time interval (from Phase 1 to Phase 2) and the change in J₀ and J₄₅ between the two age groups.

Prevalence estimates with 95% CIs were adjusted for the cluster design. Estimated prevalence data are from participants where data are available from Phases 1 and 2. For each comparison the 95% CIs were compared; if they overlapped prevalence was determined not to have changed significantly. Spearman’s rank correlation was used to assess the strength of the association between the continuous variables.

Results were considered statistically significant if P < 0.05.

**RESULTS**

Of the 399 6- to 7-year-old and 669 12- to 13-year-old children who participated in Phase 1 of the NICER study, 302 (76%) of the younger and 436 (65%) of the older cohort were re-examined at Phase 2. As over 98% of participants at Phase 1 were white, this study presents data from white participants only (n = 295 younger cohort; n = 429 older cohort). Although for both cohorts females were more likely than males to participate at Phase 2 (younger cohort, P = 0.04; older cohort, P < 0.001), there was no statistically significant difference between participants and nonparticipants in Phase 2 with respect to Phase 1 SER (younger cohort, P = 0.75; older cohort, P = 0.33), sphere (younger cohort, P = 0.43; older cohort, P = 0.61), astigmatism (younger cohort, P = 0.32; older cohort, P = 0.68), and spectacle wear (younger cohort, P = 0.10; older cohort, P = 0.06), economic deprivation (younger cohort, P = 0.79; older cohort, P = 0.51), urban/rural classification (younger cohort, P = 0.31; older cohort, P = 0.28), parental myopic status (at least one myopic parent; younger, P = 0.19; older, P = 0.99), or attendance at grammar school (academically selected schools for children aged 11 years and older; older cohort only, P = 0.67). For further analysis, Phase 1 results pertain only to data from those who participated in Phases 1 and 2.

The younger cohort had a statistically significantly greater follow-up interval compared to the older cohort (younger median 35.9 months, interquartile range [IQR], 35.7–36.4; older 35.7 months, IQR, 34.6–35.9; z = −9.34, P < 0.001).

Of the 724 participants, only two had an increase in astigmatism greater than 2.00 DC over the 3-year period and both of these had a change of at least 3.00 DC. For one of these outliers the change in corneal curvature data suggests that the change in astigmatism may be pathological in origin. The second outlier had a right esotropia and available aberration data from Phase 2²⁷ suggest that the Phase 2 refractive error measurement may have been made off axis. These two outliers (both of which were from the younger cohort) have been removed from subsequent analyses. To be representative of the normal population, data from all other participants with
strabismus were included in subsequent analyses (strabismus younger cohort \( n = 8 \), 2.6%; older cohort \( n = 11 \), 2.5%).

Analyses also were replicated after data from strabismic participants had been removed, but no noteworthy changes were identified. The spherical component of the refraction at Phase 1 ranged from \(-1.00\) to \(+10.75\) DS for the younger cohort and from \(-5.50\) to \(+11.25\) DS for the older cohort. Although astigmatism increased and decreased over the 3-year period (Fig. 1), there was no statistically significant difference in the median amount of change in astigmatism between the two cohorts (younger cohort median \( 0.25 \) D, IQR \(-0.50\) to \(+0.25\); older median \( 0 \) D, IQR \(-0.25\) to \(+0.25\); \( z = 1.4 \), \( P = 0.16 \)). The overlapping 95% CIs show that the prevalence of astigmatism also was unchanged in both cohorts: at 15 to 16 years 17.5% (95% CIs, 13.9–21.7) of participants were astigmatic compared to 18.4% (95% CIs, 13.4–24.8) at age 12 to 13 years; at 9 to 10 years 17.1% (95% CIs, 13.3–21.6) were astigmatic compared to 22.9% (95% CIs, 18.3–28.2) at 6 to 7 years.

However, while prevalence remained unchanged, it was not necessarily the same children who had astigmatism at Phases 1 and 2. Table 1 shows that while most children were not astigmatic at either Phase 1 or Phase 2, some children were losing astigmatism, while others were becoming astigmatic over the study period. Figure 2 shows the amount by which astigmatism changed across the different classifications (never astigmatic, remained astigmatic, became astigmatic, and became nonastigmatic).

At Phase 1, of those with astigmatism \( \geq 1.00 \) DC, 21% of the younger cohort and 48% of the older cohort reported having a current refractive correction compared to 28% of the younger cohort and 55% of the older cohort at Phase 2. However, poor compliance with spectacle wear has been reported previously for this population\(^{28}\) so accurate assessment of the impact of refractive correction on the changing profile of astigmatism cannot be made.

Separate analyses were done on the 19 participants (younger cohort, \( n = 8 \); older cohort, \( n = 11 \)) with strabismus. Although astigmatism \( \geq 1 \) DC was more common within this subgroup (not astigmatic at Phase 1 or 2, \( n = 6 \), 32%; remained astigmatic, \( n = 5 \), 26%); astigmatism increased and decreased over the 3-year period (median change in astigmatism, \(-0.25\) D, IQR \(-0.50\) to \(+0.50\); \( n = 4 \), 21% became astigmatic and \( n = 4 \) lost astigmatism).

Table 2 shows that 7.5% \( (n = 23) \) of the younger cohort and 4.7% \( (n = 20) \) of the older cohort had a change in astigmatism (either increasing or decreasing) of at least 1 DC.

Although there was no statistically significant association between change in refractive astigmatism and change in corneal astigmatism overall (younger cohort Spearman’s \( \rho = -0.07 \), \( P = 0.25 \); older cohort Spearman’s \( \rho = -0.08 \), \( P = 0.09 \)), the correlation between change in refractive and corneal astigmatism was greater among the participants who had refractive astigmatism \( \geq 1 \) DC at Phase 1 and was significant for the older cohort (younger cohort Spearman’s \( \rho = -0.18 \), \( P = 0.15 \); older cohort Spearman’s \( \rho = -0.27 \), \( P = 0.02 \)). There also was a statistically significant correlation between change in refractive and corneal \( J_{45} \) for both cohorts (younger cohort Spearman’s \( \rho = 0.24 \), \( P < 0.001 \); older cohort Spearman’s \( \rho = 0.25 \), \( P < 0.001 \)) and between change in refractive and corneal \( J_{45} \) for the younger cohort (younger cohort Spearman’s \( \rho = 0.14 \), \( P = 0.01 \); older cohort Spearman’s \( \rho = 0.02 \), \( P = 0.73 \)).

Figure 3 shows the relation between the change in astigmatism and the change in the spherical component of the refraction: increasing astigmatism is associated with a hyperopic shift in refraction (younger cohort Spearman’s \( \rho = 0.15 \), \( P = 0.01 \); older cohort Spearman’s \( \rho = 0.22 \), \( P < 0.001 \)).

Figure 4 illustrates that in the younger cohort high levels of astigmatism (>2.50 DC, \( n = 3 \)) both increased \((n = 1)\) and decreased \((n = 2)\) between Phases 1 and 2, but in the older

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**Figure 2.** Change in astigmatism between Phases 1 and 2. Where astigmatism is increasing, the change is described in terms of a positive figure, where it is decreasing, values are negative. The line in the gray box marks the median; lower and upper edges of the box mark the lower and upper quartiles, and the whiskers mark the range of the data with the outliers shown as gray dots.
Table 2. Changing Profile of Astigmatism in the 3-Year Period

<table>
<thead>
<tr>
<th>No Change in Astigmatism</th>
<th>Increase in ≥1 DC</th>
<th>Decrease in ≥1 DC</th>
<th>Increase in &lt;1 DC</th>
<th>Decrease in &lt;1 DC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Younger Cohort</td>
<td>Older Cohort</td>
<td>Younger Cohort</td>
<td>Older Cohort</td>
<td>Younger Cohort</td>
</tr>
<tr>
<td>Proportion</td>
<td>20.8</td>
<td>21.2</td>
<td>32.4</td>
<td>36.8</td>
</tr>
<tr>
<td>95% CIs</td>
<td>15.2–27.8</td>
<td>17.7–25.2</td>
<td>26.1–39.5</td>
<td>30.4–43.8</td>
</tr>
<tr>
<td>n</td>
<td>61</td>
<td>91</td>
<td>95</td>
<td>158</td>
</tr>
<tr>
<td>Median phase</td>
<td>0.50</td>
<td>0.25</td>
<td>0.25</td>
<td>0.50</td>
</tr>
<tr>
<td>Astig D (IQR)</td>
<td>(0.25–0.75)</td>
<td>(0.00–1.00)</td>
<td>(0.00–1.00)</td>
<td>(0.00–1.00)</td>
</tr>
</tbody>
</table>

DISCUSSION

Few studies, with the exception of surveys within nonwhite populations,24 have examined the prevalence of astigmatism in childhood beyond the age of 12 to 13 years. The current study confirms previous longitudinal7 and cross-sectional11 reports that prevalence of astigmatism remains relatively static throughout childhood and also demonstrates that prevalence of astigmatism remains stable beyond 12 to 13 years. However, these prevalence data are misleading as results from this study showed that a noteworthy minority of individuals’ astigmatic profiles are dynamic within this period. Of the younger cohort in this study, the 3-year incidence of astigmatism of 11.6% is very similar to that reported for similarly aged children in Singapore,10 and astigmatic errors ≥1 DC are likely to develop in approximately 10% of children during early teenage years. This finding has important public health implications: the changing profile of childhood astigmatism must be considered when devising recommendations for appropriate eye examination intervals as uncorrected astigmatism can cause a reduction in vision.30

Although there was a weak association between increasing astigmatism and a hyperopic shift in the spherical component of the refraction in both cohorts and an association between increasing with-the-rule astigmatism and a myopic shift in refraction in the older cohort, as the correlation was low, neither the amount of astigmatism at Phase 1 nor the change in astigmatism over the 3-year period, are useful clinical predictors of the change in the spherical component of the refractive error over the same time frame. Associations between astigmatism and ametropia have been reported previously with astigmatism (especially against the rule astigmatism) being associated with the development and progression of myopia8,10,16,31 and it has been suggested that astigmatic blur in early life may have an effect on the emmetropization process.32 The NICER study Phase 1 reported a high prevalence of astigmatism at 6 to 7 years and 12 to 13 years, which was associated with hyperopia and myopia,11 both of which are more common in white children in Northern Ireland than in white children in Australia.34 If this high prevalence of astigmatism is present in early childhood it may be that the disruptive effect of astigmatic blur on emmetropization has manifested at an earlier age and could explain the differences in
prevalence between the two populations and why no strong association has been found in later childhood between astigmatism and changing refraction in Northern Ireland. Further studies of astigmatism from birth, through infancy and childhood would assist in confirming whether astigmatism is a cause or effect of ametropia in this population. Data from Phase 3 of the ongoing NICER study, six years after Phase 1, also will help establish if the lack of an association between astigmatism and changing refractive status continues into adulthood.

**FIGURE 3.** Scattergraph of the relation between change in astigmatism and change in spherical component between Phases 1 and 2. Where astigmatism is increasing, the change is described in terms of a positive figure, where it is decreasing, values are negative. Noise (5%) has been added to the data (using the jitter function on STATA) to allow for representation of overlapping points. Younger cohort $r = 0.15, P = 0.01$; older cohort $r = 0.22, P < 0.001$.

**FIGURE 4.** Scattergraph of the relation between change in astigmatism between Phases 1 and 2 and amount of astigmatism at Phase 1 for those with astigmatism $\geq 1$ DC at Phase 1. Where astigmatism is increasing, the change is described in terms of a positive figure, where it is decreasing, values are negative. Noise (5%) has been added to the data (using the jitter function on STATA) to allow for representation of overlapping points. Younger cohort, coefficient $= -0.28, P = 0.02$; older cohort, coefficient $= 0.17, P = 0.14$. 
For both cohorts there was a correlation between change in refractive and corneal J0. However, change in refractive and corneal J45 was correlated only in the younger cohort. As with other studies, the measures of corneal astigmatism in the current study are made solely on the basis of changes to the anterior curvature of the central cornea and we cannot disregard that the posterior and peripheral curvature of the cornea also may have changed. Future refractive error studies on this population also would benefit from assessment of corneal topography and intraocular parameters, such as lens curvature, which has been proposed as a contributory source of myopic astigmatism, to help ascertain the origin of these astigmatic changes.

**FIGURE 5.** Scattergraph of the relation between change in J0 between Phases 1 and 2 and the change in the sphere. Noise (5%) has been added to the data (using the jitter function on STATA) to allow for representation of overlapping points. Younger cohort, coefficient = –0.08, P = 0.20; older cohort, coefficient = –0.31, P = 0.006.

**FIGURE 6.** Scattergraph of the relation between change in J45 between Phases 1 and 2 and the change in the sphere. Noise (5%) has been added to the data (using the jitter function on STATA) to allow for representation of overlapping points. Younger cohort, coefficient = –0.03, P = 0.57; older cohort, coefficient = –0.002, P = 0.97.
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Strengths

Identical robust protocols for the measurement of refractive error and ocular components were used within both phases of the NICER Study and participation at Phase 2 was good. Furthermore, with the exception of sex, there were no statistically significant differences between those who participated in both phases and those who only participated at Phase 1. Although there was a significant difference in follow-up interval between the two cohorts, the median difference of 0.2 months equates to less than 1 week and, therefore, is unlikely to have had a major influence on the development or progression of refractive error.

Limitations

As with all prospective studies, the data must be considered with respect to the repeatability of the instrumentation being used. The 95% limits of agreement for cylindrical power using the Shin-Nippon autorefractor have been calculated as −0.50 to +0.44 DC from data reported by Mallen et al. and many of the participants within the current study showed a change in astigmatism that should be disregarded as possibly being due to the repeatability limitations of the instrument. Of the younger cohort, 39% (n = 115) displayed a change in astigmatism outside of the 95% repeatability limits of the instrument, compared to 21% (n = 92) of the older cohort. It also is clear from Figure 2 and Table 1 that many of those becoming astigmatic or losing astigmatism have a clearly defined change beyond the repeatability limits of the autorefractor.

The different sample sizes of the two cohorts, reflects the fact that the primary aim of Phase 1 of the NICER study was to establish the prevalence of myopia in 6- to 7-year-olds and 12- to 13-year-olds. This study of the changing profile of astigmatism would have more statistical power had there been an increased sample size at Phase 1 and greater participation at Phase 2.

The current study did not explore risk factors for the development or progression of astigmatism. Astigmatism is a genetic condition and it is difficult to obtain reliable data on family history of astigmatism without directly assessing the refractive status of family members. Exploring risk factors for astigmatism without adjusting for family history is likely to result in erroneous conclusions. This study did not assess whether participants were contact lens wearers so we cannot examine any potential effect on corneal shape and astigmatic refractive error.

Previous studies have used a variety of methods and definitions of refractive error to present data on the change in astigmatism, and there does not appear to be a consensus as to the most appropriate way in which to analyze the data. In the current study, data were presented illustrating the change in spherical component of the least myopic meridian, rather than change in spherical equivalent refraction as the latter is influenced by the amount of astigmatism. The authors explored presenting the data using different methods of analysis, including describing how astigmatism was associated with change in classification of refractive error using the Collaborative Longitudinal Evaluation of Ethnicity and Refractive Error (CLEERE) system to describe the change in refractive status between Phases 1 and 2. Using this system children were classified as myopic if they had −0.75 D or more myopia in both meridians and hyperopic if they had +1.25 D or more hyperopia in both meridians. Utilizing the CLEERE methodology did not alter the findings reported: neither astigmatism at Phase 1 nor change in astigmatism between Phases 1 and 2 were significantly associated with categorical change in refraction.

Conclusions

Although the prevalence of astigmatism is similar at ages 6 to 7 years and 15 to 16 years, during childhood individual children continue to develop astigmatism while other children become nonastigmatic. Results from the present study suggested that we cannot currently predict from their refractive data which children will demonstrate these astigmatic changes, highlighting the importance of all children having regular eye examinations to ensure that their visual requirements are being met.

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References