

## REVIEW ARTICLE

**A REVIEW OF ASTIGMATISM: A REFRACTIVE ERROR CORRECTABLE BY CYLINDRICAL GLASSES****<sup>1</sup>DR. SHAH JAY RAJANIKANT\*, <sup>2</sup>DR. DHIMAN K. S.**<sup>1</sup>Ph.D. (Ayu.) Scholar, Shalakyta Tantra Department, I.P.G.T. & R.A., Jamnagar, India<sup>2</sup>Professor and HOD, Shalakyta Tantra Department, I.P.G.T. & R.A., Jamnagar, India

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**ABSTRACT**

**Background:** Astigmatism is a type of refractive error. Some type of it can be corrected by Cylindrical type of glasses. It is found in clinical practice commonly. This review presents an overview of research that has been carried out examining various aspects of this refractive error. **Objective:** The present study was aimed to examine the components of astigmatism and the research into the prevalence and natural course of astigmatic refractive errors throughout life. **Materials and Methods:** Study The prevalence of astigmatism in various diseases and syndromes is also discussed. We highlight the extensive investigations that have been conducted into the possible aetiology of astigmatism, however, no single model or theory of the development of astigmatism has been proven conclusively. **Discussion:** Observations and evidence from the literature supporting and contradicting these hypotheses are presented. Recent advances in technology such as wavefront sensors and videokeratoscopes have led to an increased understanding of ocular astigmatism. With continued improvements in technology, our knowledge of astigmatism and its genesis should continue to grow. **Conclusion:** With continued improvements in technology, our knowledge of astigmatism and its genesis should continue to grow.

**Keywords :** Review, Astigmatism, Cylindrical glass, Refractive Error.**INTRODUCTION**

Astigmatism is a commonly encountered refractive error, accounting for about 13 per cent of the refractive errors of the human eye.<sup>1</sup> Our knowledge of astigmatism appears to have begun in the early 1800s when Thomas Young reported on his own astigmatism but it was not until 1825 that the first cylindrical lens was used by George Airy for the purpose of correcting his own astigmatic refractive error.<sup>2</sup> Since these early explorations, there has been a great deal of research carried out into various aspects of astigmatism. One reason for this research interest is the fact that the presence of astigmatism appears to have the potential to influence normal visual development. The presence of high degrees of astigmatism is associated with the development of amblyopia<sup>3</sup> and some associations have also been noted between astigmatism and the development of Myopia.<sup>4</sup> Advances in technology and instrumentation mean that our ability to measure, define and analyse the eye's optical and shape properties (including astigmatism) have improved markedly in recent years.

In spite of extensive research, the exact cause of astigmatism is still not known. One possible reason for astigmatic development would be a genetic aetiology. Other possible causes include mechanical interactions between the cornea and the eyelids and/or the extraocular muscles or a visual feedback model in

which astigmatism develops in response to visual cues. In this review we will consider the various hypotheses regarding the aetiology of astigmatism and examine the evidence in the literature for these theories. We will also present some new evidence from recent research in our laboratory that has investigated the role of near work and eyelid forces on corneal shape and refractive error development.

**MATERIALS AND METHODS INCLUDING DISCUSSION****Components of Astigmatism**

Ocular astigmatism can occur as a result of unequal curvature along the two principal meridians of the anterior cornea (known as corneal astigmatism) and/or it may be due to the posterior cornea, unequal curvatures of the front and back surfaces of the crystalline lens, decentration or tilting of the lens or unequal refractive indices across the crystalline lens (known as internal or residual astigmatism).

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The combination of the corneal and the internal astigmatism gives the eye's total astigmatism (that is, total astigmatism equals corneal astigmatism plus internal astigmatism). Corneal astigmatism is often classified according to the axis of astigmatism as being either with-the rule (WTR), oblique or against-the-rule (ATR). In the past, astigmatism has been defined as 'regular' or 'irregular'.

A recent study investigating corneal topography has classified astigmatism according to the changes occurring in the astigmatism of the peripheral cornea.<sup>5</sup> Corneal astigmatism was classified as being stable, reducing or increasing in the peripheral cornea. Of the subjects with significant corneal astigmatism tested in this study, astigmatism was found most commonly to be reducing (47 per cent of astigmatic subjects) or stable (44 per cent) in the peripheral cornea illustrates these forms of corneal astigmatism.

### Prevalence of Astigmatism and changes with Age

There have been many studies that have attempted to define the prevalence of astigmatism in the population and to illustrate the typical changes that occur in astigmatism throughout life. These investigations provide some clues to the possible causes of astigmatism.

Generally, studies have shown that in the first months of life, infants exhibit a high prevalence of significant degrees of astigmatism,<sup>6</sup> which appears to be corneal in origin. The cornea of newborns is steep and exhibits large degrees of astigmatism. Isenberg and co-workers<sup>7</sup> used video keratoscopy to measure the corneal curvature of newborns (up to eight days after birth) and found an average of six dioptres of corneal astigmatism. Studies have also shown that the steepest, most astigmatic corneas occur in the newborns with the lowest birth weight and lowest post-conceptual age. While studies have consistently found high degrees of corneal astigmatism to be present in infancy, there is some conflicting evidence of the most common axis of astigmatism. Perhaps indicating the difficulties of obtaining accurate measurements on newborn infants or suggesting a large amount of variability in the corneal shape of infants, some studies have found a predominance of WTR corneal astigmatism, while others have shown a predominance of ATR corneal astigmatism in infants.

As infants grow older, the prevalence of high degrees of astigmatism typically reduces or, in other words, an emmetropisation of the astigmatic refractive error occurs. Dobson, Fulton and Sebris<sup>8</sup> found a shift in astigmatism from a predominance of higher degrees of ATR astigmatism in children younger than four years, to a predominance of low levels of WTR astigmatism in children older than four years. Gwiazda and colleagues<sup>9</sup> postulated that pressure from the eyelids on the cornea over time may be causing the shift in astigmatic axis from ATR to WTR in children.

Studies of preschool-age children generally show a relatively low prevalence of high degrees of astigmatism (that is, greater than one dioptre) that is

predominantly WTR in nature.<sup>10</sup> Huynh and associates<sup>11</sup> investigated a large population of six-year-old children and found that only 4.8 per cent of children exhibited greater than one dioptre of ocular astigmatism and 75 per cent of subjects exhibited WTR corneal astigmatism.

In summary, at birth children exhibit a high incidence of astigmatism that is corneal in origin. As children grow older, the cornea flattens with significantly reduced astigmatism. Over the age of four years, the prevalence of large amounts of astigmatism is low, with small amounts of WTR astigmatism being found most commonly.

### Astigmatism in adults

Astigmatism in young adults (younger than 40 years) occurs commonly but in relatively low amounts.<sup>12</sup> In an investigation of young adults aged 20 to 30 years, Satterfield found that 63 per cent of subjects exhibited 0.25 D or more of ocular astigmatism, however, the majority of subjects with measurable astigmatism exhibited less than one dioptre. In a cross-sectional study, Fledelius and Stubgaard found that 46 per cent of the total population had corneal astigmatism of greater than 0.5 D but only 4.7 per cent of the population exhibited greater than 1.5 D of corneal astigmatism. Generally, studies have shown that in young adults, WTR astigmatism occurs most commonly.<sup>13</sup>

With increasing age, a general shift in the axis of astigmatism is found from a predominance of WTR astigmatism (in adults younger than 40 years) to a predominance of ATR astigmatism (in adults older than 40 years). This shift in astigmatism axis in older age appears to be due to changes in corneal curvature. In a cross-sectional study of corneal and total astigmatism, Anstice found that internal astigmatism remained relatively stable over time and that changes in astigmatism throughout life were due primarily to changes in corneal curvature. Baldwin and Mills<sup>39</sup> investigated longitudinal changes in corneal and total astigmatism in patients over a 40-year period and found a steepening of the cornea and an increase in ATR astigmatism with aging. The majority of this change in astigmatism was due to corneal change, that is, a steepening of the horizontal meridian of the cornea. As will be discussed later, this change in corneal curvature may be related to the reduction in tension of the eyelids that typically occurs with age.<sup>14</sup>

In summary, young adult subjects typically display small degrees of WTR astigmatism and in older adult years a shift in astigmatism occurs where ATR astigmatism becomes more prevalent. Astigmatism most commonly occurs due to the curvature of the cornea and the changes in astigmatism that occur throughout life also appear to be due primarily to corneal change.

### Astigmatism and other Refractive Errors

There is some evidence to suggest that the presence of astigmatism may be associated with the presence of spherical refractive errors. The presence of astigmatism has been found to be associated with

myopic refractive errors, that is, astigmatism was associated with higher degrees of Myopia. Fulton, Hansen and Petersen suggested that uncorrected astigmatic errors influenced the development of Myopia and that the optical blur from uncorrected astigmatism may be a trigger for myopic development. The presence and changes in astigmatism have been found by some investigators to be associated with an increased progression of Myopia.<sup>15</sup> In a longitudinal study of refractive error, Gwiazda and colleagues found that their subjects exhibiting significant ATR astigmatism and myopia in infancy were more likely to develop myopia at school age. In contrast with these studies, other investigators<sup>15</sup> have found little to no association between the presence of astigmatism and the presence and progression of myopic refractive errors.

While there is some equivocal evidence, there does appear to be an association between astigmatism and the development and progression of myopia. The exact nature of this relationship and the mechanisms underlying it are not fully understood.

#### **Astigmatism resulting from Ocular Surgery**

Ocular surgery can lead to significant changes in Astigmatism. The fact that some surgical procedures can cause highly significant changes in corneal curvature and Astigmatism provides information regarding the biomechanical properties of the cornea and may also give clues to the aetiology of Astigmatism. Meek and Newton<sup>16</sup> suggested that the structural and mechanical properties of the cornea (including the arrangement of collagen fibrils in the cornea and sclera) can explain the alteration in corneal curvature and the resulting Astigmatism changes following some ocular surgery.

Modern surgical techniques for cataract involve a small incision in the cornea and can lead to alterations in astigmatism. Incisions made in the cornea generally cause a flattening in the incised corneal meridian (and a subsequent steepening of the orthogonal meridian), thus leading to astigmatic change.<sup>17</sup> Therefore, the location of the corneal incision made during cataract surgery will influence the induced astigmatism, whereby a superior incision typically results in a flattening of the vertical meridian (and an increase in ATR astigmatism) and temporal incision placement leads to a flattening of the cornea in the horizontal meridian (that is, an increase in WTR astigmatism). The size of the incision (with larger incisions causing greater astigmatic change) and the location of the incision in relation to the corneal centre (with incisions closer to the centre of the cornea being associated with greater astigmatic change) will influence the induced astigmatism. The changes in corneal astigmatism following cataract surgery appear to be related to the anatomical and biomechanical properties of the cornea. By understanding these corneal changes following incisional surgery, strategies are now employed that take advantage of these astigmatic changes in the cornea to reduce the overall level of refractive astigmatism following cataract surgery.

Retinal detachment surgery involving scleral buckling causes significant changes in astigmatism and corneal curvature. This appears to be due to indentation of the sclera by the buckle leading to alterations in corneal curvature, which can result in either regular or irregular corneal astigmatism. The specific buckling procedure influences the changes in corneal curvature. Local or segmental buckles lead to a local steepening in the corneal quadrant adjacent to the buckle and encircling buckles lead to a more generalised peripheral flattening and central steepening of the cornea. Uneven tightening or asymmetric placement of encircling scleral buckles may also lead to marked asymmetric astigmatic change. Most studies have noted these corneal changes to be transient but significant change can persist up to six months following surgery.<sup>18</sup>

Trabeculectomy for glaucoma can cause significant corneal change, which is typically a steepening in the vertical meridian, leading to an increase in regular WTR and irregular astigmatism.<sup>19</sup> The size of the incision appears to be correlated to the amount of induced astigmatism with smaller incisions (as used in 'microtrabeculectomy' procedures) leading to a smaller astigmatic change. The exact cause of the corneal change following trabeculectomy is not known, although it has been postulated that it relates to tension from sutures used in the surgery, cauterisation of the wound or wound healing factors leading to steepening of the cornea in the superior meridian.

The growth of a pterygium onto the cornea can lead to significant changes in corneal astigmatism. Typically, the shift in astigmatism is an increase in (asymmetric) WTR astigmatism brought about by a flattening of the cornea that occurs in the horizontal meridian between the corneal apex and the head of the pterygium. The specific cause of this corneal flattening is thought to be a tear pooling effect near the head of the pterygium, mechanical tractional forces on the cornea from the pterygium or a combination of these factors. The size of the pterygium appears to be related to the magnitude of the induced astigmatism. illustrates the corneal topographical changes typically brought about by a pterygium. Surgery to remove pterygia typically leads to a reduction of the induced WTR astigmatism and an increase in the regularity and symmetry of corneal topography. The amount of astigmatic change brought about by surgery for pterygium appears to be related to the preoperative size of the pterygium.

Another ocular surgical procedure that can result in significant amounts of corneal astigmatism postoperatively is penetrating keratoplasty. Troutman and Lawless reported an average level of corneal astigmatism of 4.3 D (range of 0–10.5 D) in subjects following penetrating keratoplasty. A number of different procedures has been suggested to reduce the level of astigmatism post-keratoplasty. Selective manipulation of sutures whereby the sutures along the steepest corneal meridian are loosened has been shown to reduce the astigmatism.<sup>20</sup> Belmont, Troutman and Buzard<sup>21</sup> found intra-operative monitoring of corneal curvature resulted in less astigmatism following surgery. The difference between the graft and recipient corneal size may also influence the post-surgical

corneal curvature. Newer surgical techniques, such as non-mechanical trephination with the Excimer laser, also appear to lead to a reduction in post-operative astigmatism compared to traditional mechanical trephination techniques.

### Causes of Astigmatism

While much research has been carried out into the prevalence and changes in astigmatism throughout life, questions still remain of the causes of astigmatism. As astigmatism most commonly has a corneal origin, the following sections will concentrate on the research that has been conducted into the possible causes of corneal astigmatism.

### Genetics and astigmatism

One possible explanation of the aetiology of astigmatism is that astigmatic refractive errors are genetically determined. Numerous studies have been undertaken to investigate the influence of genetics on astigmatic development.

In an early study, Wixson<sup>22</sup> investigated the heritability of corneal power by comparing corneal power in a group of parents and children with a group of husbands and wives. He concluded that both parents seem to participate in determining the corneal power characteristics of the child. Wixson suggested that the inheritance of corneal power appeared to be best approximated by an autosomal recessive pattern.

Several studies comparing monozygotic and dizygotic twins have investigated the genetic influence on astigmatic refractive errors, including Teikari and O'Donnell,<sup>23</sup> Teikari and associates and Valluri and colleagues. All of these studies found significant differences in the intra-pair correlations for spherical refractive errors between monozygotic and dizygotic twins, suggesting that genetic influences on myopia and Hyperopia are strong. The correlations between monozygotic twins for astigmatism were not significantly different from the correlations between dizygotic twins in these studies. This suggests that the genetic contribution to astigmatism is low, with environmental factors being the major contributors. In another large study, Hammond and co-workers<sup>24</sup> investigated the refractive error of 506 female twins (226 monozygotic and 280 dizygotic). In contrast to the previous studies of twins, Hammond and co-workers found the correlations for monozygotic twins for astigmatism to be greater than the correlations for dizygotic twins. This suggests more significant genetic effects on astigmatism than previous studies. The authors concluded that the heritability of astigmatism was 50 to 65 per cent (that is, 50 to 65 per cent of the variance in astigmatic refractive error was attributed to genetic effects) and that the heritability predominantly involved dominant genetic effects.

Clementi and associates<sup>25</sup> analysed data from 125 Italian families affected by astigmatism (476 subjects). Refractive error and corneal astigmatism were measured using automated techniques. When the data were analysed for astigmatism as a qualitative trait (that is, affected/unaffected), no definite model of inheritance

was found to best fit the data. When the severity of astigmatism was included in the analysis, an autosomal dominant model of inheritance provided the best fit to the data. The authors estimated that the frequency of the putative gene was low and that it had more effect on the presence of astigmatism than on its severity. Clementi and associates suggested that bias (in subject selection and analysis) in previous studies may have led to inconsistent results.

Lee and colleagues investigated the refractive errors of a large population of families in the Beaver Dam eye study (440 family groups). While strong aggregation of Myopia and hyperopia was found among siblings in this study (suggesting a potential genetic influence on these refractive errors), minimal associations were found between family members for astigmatism. This suggests minimal genetic influence on Astigmatic refractive error.

The studies into genetics and astigmatism do present some conflicting results. Certain studies indicate some degree of heritability of astigmatism and also tend to favour an autosomal dominant mode of inheritance. Other studies favour a stronger environmental influence. It would appear that both genetic and environmental factors have roles in the development of astigmatism. The exact nature of these mechanisms is still not fully understood.

### Astigmatism and extraocular muscles

Howland and Sayles suggested that corneal astigmatism may develop as a result of unequal tension exerted on the cornea by the extraocular muscles (EOMs) (for example, an increased degree of tension in the horizontal recti muscles may lead to a bending of the cornea in the horizontal meridian thus leading to ATR corneal astigmatism). They proposed that changes in the tension of the EOMs throughout life might lead to subsequent changes in corneal astigmatism. The effect of extraocular muscle (EOM) tension on corneal shape is a subject that has received limited coverage in the literature and the exact influence that contraction and relaxation of the EOMs has on corneal topography is still not fully understood.

Early research into this topic investigated the changes occurring in corneal curvature during convergence. Some studies have found that a slight flattening of the cornea (in the horizontal meridian) accompanies the act of convergence.<sup>26</sup> Other investigators found that no significant changes in corneal curvature (as measured with a photokeratoscope) occur with convergence or with changes in fixation (that is, changes in EOM tension do not cause a change in corneal shape). All of these studies were limited by the technology of their time and the techniques used may not have provided a sufficiently accurate assessment of the periphery of the cornea to describe any changes occurring during convergence.

More recent reports of the effect of EOM tension on astigmatism have centred on changes occurring in corneal topography and refraction following EOM surgery. Kwitko and colleagues<sup>27</sup> found that surgery on rabbit EOM's caused significant changes in corneal

topography in some cases. There have also been several reports of highly significant changes in astigmatism and corneal topography following surgery for strabismus in human subjects.

The exact cause of this change in corneal topography following EOM surgery is still not known. Alteration in muscle tension (and subsequent alteration in the force applied by the muscles to the anterior globe) or changes in tractional forces due to surgery have both been suggested as possible causes of these topographical changes. Factors relating to surgical recovery (for example, inflammation in and around the globe) may also influence the corneal topography following strabismus surgery. It remains to be seen whether alterations in EOM tension from everyday tasks such as convergence and eye movement also cause significant changes in corneal topography and astigmatism.

### **Eyelid pressure and astigmatism**

Pressure from the eyelids on the cornea has been implicated as a possible factor in the development of corneal astigmatism. Grosvenor proposed a theory for the aetiology of astigmatism, whereby the band-like pressure from the upper eyelid on the cornea causes the eye to exhibit WTR astigmatism (as occurs in the majority of young adults). Grosvenor suggested that the tightness of the eyelids and the rigidity of the ocular surface interacted to produce corneal astigmatism. The typical shift in astigmatic axis from WTR in young adults to ATR in older adults was also explained through a reduction in lid tension with age, leading to a reduction in WTR corneal astigmatism.

There has been a number of experiments examining the effect of the eyelids on corneal Astigmatism. Wilson, Bell and Chotai<sup>28</sup> investigated changes in corneal astigmatism brought about by lifting the eyelids. Eighteen subjects had their corneal astigmatism measured (by keratometry) with lids in normal position and retracted (with a speculum). Subjects with more than one dioptre of WTR astigmatism showed a systematic change in the direction of less WTR astigmatism when the lids were retracted. Those subjects showing changes exhibited a steepening of the horizontal meridian of the cornea but not a flattening of the vertical meridian as may have been expected. The results of this experiment suggest that the position of the lids has an influence on the degree and direction of corneal astigmatism.

In a cross-sectional study, Vihlen and Wilson investigated the changes in eyelid tension and corneal toricity that occur with age. A definite reduction in lid tension, and a change in corneal toricity towards ATR were found with increasing age. No association was found between eyelid tension and corneal toricity (that is, tighter lids did not correlate with more astigmatism). Longitudinal studies investigating eyelid tension and corneal toricity may be required to shed more light on whether eyelid tension does play a role in the typical changes in corneal astigmatism found with age.

The influence of lid position on astigmatism was also studied by Grey and Yap. Ocular astigmatism was

measured on patients who adopted three different lid positions (that is, deliberately widened, normal position and deliberately narrowed lids). A significant increase in ocular astigmatism was found for the deliberately narrowed eyelid position with subjects showing an increase of WTR astigmatism.

Lieberman and Grierson<sup>29</sup> measured corneal topography in subjects with and without the lids touching the cornea. They found changes in corneal shape occurred when the lids were retracted from the cornea. This further confirms that the position of the eyelids can influence the shape of the cornea.

If pressure from the eyelids leads to WTR corneal astigmatism, then one may expect that certain correlations exist between the axis and magnitude of astigmatism and the angle and position of the eyelids. It should be noted that the existence of correlations between eyelid morphological features and astigmatism does not prove that eyelid pressure is causing astigmatism. Garcia and associates<sup>45</sup> studied a population of children with high astigmatism (greater than 1.5 D). Cycloplegic retinoscopy, corneal topography and palpebral fissure slant were measured. Most astigmatism was WTR. The majority of subjects also displayed an up-slanting of the palpebral fissure (that is, temporal canthus higher than the nasal canthus). A significantly higher proportion of patients with high corneal and total astigmatism also displayed abnormally slanted palpebral fissures. The axis of astigmatism was found to be significantly correlated with the degree of palpebral fissure slant. The steeper corneal axis was found to be oriented perpendicular to the horizontal axis of the palpebral fissure. Garcia and associates suggested two possible mechanisms for the association between palpebral fissure slant and Astigmatism axis: developmental factors may lead to correlated growth between the lids and the cornea or the mechanical effects of the slanting eyelid caused alterations in the corneal shape.

Read, Collins and Carney recently carried out an experiment investigating corneal astigmatism and eyelid morphology in a group of 100 young normal adult subjects. Corneal astigmatism was assessed through the analysis of corneal topographical data and eyelid morphological information was ascertained through analysis of digital images taken of the anterior eye and adnexae (in primary gaze, 20 degrees downgaze and 40 degrees downgaze). In this group of normal subjects, significant correlations were found between the parameters describing the axis of corneal astigmatism and the angle of the upper and lower eyelid and palpebral fissure in primary gaze.

### **Astigmatism in diseases and syndromes**

There are several genetic syndromes that are associated with eyelid abnormalities and also with an increased prevalence of astigmatism. Studies into populations of subjects with these syndromes tend to support the notion that pressure from the eyelids may contribute to corneal astigmatism.

Down syndrome has been associated with significant ocular abnormalities. Da Cunha and de Castro

Moreira noted that 82 per cent of the Down syndrome patients exhibited up-slanting palpebral fissures (that is, temporal canthus is higher than nasal canthus) and 60 per cent exhibited astigmatism. Astigmatism (greater than 0.5 D) was the most common refractive error found in the population and severe astigmatism (greater than 3 D) was found in 20 per cent of the children. Haugen, Hovding and Lundstrom<sup>30</sup> presented longitudinal data on 60 children with Down syndrome. They found 57 per cent of the children have astigmatism (the majority being WTR). The reduction in infant astigmatism seen in the first years of life in a normal population was not exhibited by the Down syndrome population (that is, there was a failure of the emmetropisation process in this population). Eleven children had oblique astigmatism, which displayed distinct mirror symmetry of axes between right and left eyes. The authors suggested that this dramatic mirror symmetry for oblique astigmatism may be caused by mechanical factors exerted on the cornea by the up-slanting of the palpebral fissures. Cregg and colleagues<sup>31</sup> also found a failure of emmetropisation in Down syndrome children. Of those children with oblique axes, the majority showed mirror symmetry of the axes between eyes.

Wang and associates reported on the ocular findings of 14 patients with Treacher Collins syndrome, a rare congenital disorder. The patients generally exhibited a downward slanting of the palpebral fissure. Corneal astigmatism (greater than 2 D) was present in five of the 14 patients. There was an overall correlation between the degree of facial deformity and the presence of astigmatism. Generally, the axis of astigmatism was found to be in the same quadrant as the horizontal palpebral fissure axis.

Ocular abnormalities are also found in spina bifida. Paysse and co-workers<sup>32</sup> found exaggerated up-slanting of the palpebral fissure to be a common finding in their 73 subjects with spina bifida. A high prevalence of oblique astigmatism was also found. The majority of the patients with up-slanting palpebral fissures exhibited astigmatism (greater than 0.75 D). Of the patients with astigmatism and up-slanting palpebral fissures, the axis of astigmatism tended to be oriented perpendicular to the angle of the palpebral fissure, similar to the trends reported with Down syndrome.

All of the above populations exhibit a high prevalence of astigmatism. It appears that in some cases, the increased prevalence of astigmatism and axis of astigmatism can be explained by changes in the mechanical interactions of the eyelids with the cornea. Asymmetric corneal growth is also possible.

### **Nystagmus and Astigmatism**

Nystagmus is characterised by rapid involuntary oscillatory eye movements. These back and forth eye movements typically occur horizontally. Nystagmus may occur as a physiological phenomenon or be a congenital or acquired defect. Congenital nystagmus can be associated with many different ocular and neurological defects. Subjects with nystagmus have been shown to display an increased prevalence of high

degrees of astigmatism.<sup>33</sup> The astigmatism in these subjects is generally WTR and corneal in origin.

It appears that the process of emmetropisation (that is, the normal process of reduction of neonatal refractive errors with eye growth) is impaired in subjects with nystagmus. These subjects show a wide spread of refractive errors, with high degrees of WTR corneal astigmatism being particularly common. While the exact cause of the high degrees of astigmatism is not known, mechanical interaction between the eyelids and the cornea (which would be increased as a result of the constant nystagmoid eye movement) may play a role. Changes in the influence of the EOMs on corneal shape or asymmetric visual experience are other possible explanations for the increased prevalence of corneal astigmatism in subjects with nystagmus.

### **Eyelid pathology and Astigmatism**

There have been numerous reports of how certain eyelid pathologies can cause corneal distortions and changes in corneal astigmatism. These reports highlight the influence that changes in eyelid pressure can play on corneal topography and astigmatism.

The presence of a chalazion in the eyelid has been shown to cause significant corneal distortions and resultant changes in corneal topography and astigmatism in some patients. Surgical removal of the chalazion generally leads to resolution of the corneal changes.

Records<sup>34</sup> presented various causes of monocular diplopia. He suggested that external irregularities of the cornea and eyelids including chalazia and unusually tight lids may produce corneal distortions that may lead to monocular diplopia.

Robb reported that 16 of 37 infants with eyelid and orbital haemangiomas exhibited astigmatic refractive errors. In almost all cases of astigmatism, the haemangioma appeared to be in a position where it exerted pressure on the eye in a direction perpendicular to the axis of astigmatism. Hence, the astigmatism was probably related to the pressure exerted by the lesion on the cornea. Plager and Snyder reported three cases in which the surgical resection of eyelid and orbital capillary haemangiomas in infants caused a resolution of astigmatism. Pressure from the haemangioma on the globe was suggested as the cause of the astigmatism in these infants.

Ptosis and the surgical repair of ptosis have been implicated in the development of astigmatism. Patients with congenital ptosis have a higher degree of corneal topographic asymmetry and irregularity, as well as a higher degree of corneal astigmatism. In addition, astigmatism appears to change following surgical repair of congenital ptosis,<sup>35</sup> possibly due to changes in the lid/cornea interaction following surgery. Superior corneal steepening was reported in a 62-year-old man with bilateral blepharoptosis. Repair of the ptosis led to relief of symptoms of monocular diplopia and amelioration of the corneal distortion.

Blepharoplasty surgery in adult patients typically causes an increase in WTR astigmatism. As some of

these changes regressed with time after surgery, Holck, Dutton and Wearly<sup>36</sup> concluded that the astigmatic changes may be due to post-surgical eyelid swelling. Detorakis, Ioannakis and Kozobolis investigated changes in corneal topography following lower eyelid surgery for involuntional ectropion. An increase in the percentage of subjects exhibiting WTR corneal astigmatism was found after blepharoplasty surgery to restore the normal lower eyelid position.

Moon, Lee and Kim<sup>37</sup> investigated the corneal topography of subjects who suffered from essential blepharospasm or hemifacial spasm. These subjects were treated with a botulinum toxin-A injection to relax the eyelid muscles and relieve the blepharospasm. Corneal topography measurements one month after botulinum injection (a time where the Botulinum Toxin is thought to reach its maximum effect) revealed a trend for a reduction in the amount of WTR corneal astigmatism compared to pre-treatment levels. Six months after the injection (where the effects of the toxin are thought to have disappeared), corneal astigmatism was noted to be returning towards pre-treatment levels.

Lid-loading procedures (with metal lid weights) to treat lagophthalmos have also been shown to induce one to two dioptres of WTR astigmatism in some patients.<sup>38</sup> The astigmatism in these cases was attributed to implants that were too heavy or of incorrect radii that caused increased pressure on the cornea.

All of these eyelid pathologies increase the influence of the eyelids on the cornea. Resolution or removal of these pathologies generally leads to a reversal of the corneal changes. These results support the concept of astigmatism development, where eyelid pressure is involved and illustrate how increasing eyelid pressure can lead to alterations in corneal curvature.

### Visual tasks and Corneal Astigmatism

Sustained pressure on the cornea from normal eyelids may also lead to corneal changes. Several reports have appeared in the literature relating episodes of monocular diplopia (caused by corneal distortion) to periods of near work in downward gaze.

Buehren, Collins and Carney<sup>39</sup> found that 12 of 20 subjects showed significant changes in central corneal topography immediately following a 60-minute reading task. The change in corneal shape was described as a wave-like distortion, which corresponded closely to the position and angle of the lids during reading. Significant changes were also found in corneal refractive power and astigmatism. The change in corneal astigmatism following reading was towards ATR. Further to this study, Buehren, Collins and Carney<sup>40</sup> showed that the significant corneal topographical changes that occur as a result of reading in downgaze also lead to significant changes in the eye's total higher order aberrations and astigmatism.

The effects of different visual tasks on corneal topography were investigated by Collins and colleagues.<sup>41</sup> Corneal topography was measured before and after subjects performed a 60-minute reading task, a

microscopy task and a computer task. Eyelid induced corneal topographical changes were evident following each of the tasks. The reading and microscopy generally resulted in larger, more centrally located corneal topographical changes. A number of subjects showed significant changes in corneal astigmatism (in the form of an increase in ATR astigmatism) following reading and microscopy. The pattern of topographical change appeared to be related to the position of the eyelids and the amount of horizontal eye movement involved in the task. Tasks involving more horizontal eye movements (for example, reading) and narrower palpebral apertures exhibited more localised corneal changes.

Evidence indicates that sustained pressure on the cornea from normal eyelids can result in significant corneal change. With recent developments in corneal topography and improved methods of visualising and analysing the shape of the cornea, corneal astigmatic and more complex corneal shape changes have been revealed resulting from the pressure of the eyelids on the cornea in visual tasks involving downgaze. These studies indicate the potential of certain visual tasks to cause short-term corneal topographical and astigmatic changes. Further research is required to explore the influence that these short-term changes may have on longer term refractive change and development of astigmatism.

### CONCLUSIONS / FUTURE RESEARCH DIRECTIONS

The exact cause of astigmatism is still not known. Current research indicates that genetic influences may play a role in the development of astigmatism. Other possible contributing factors include eyelid pressure, EOM tension and visual feedback. Interaction between the cornea and the eyelids seems to be a likely explanation of increased astigmatism in a number of ethnic groups and diseases. Pathology of the eyelids and pressure from the eyelids during reading also produce changes in corneal astigmatism. Recent research into children with high astigmatism indicates that the eyelids influence the axis and degree of corneal astigmatism. While it is clear that the eyelids can influence corneal shape, there is still no conclusive evidence that pressure from the eyelids causes corneal astigmatism.

There are several areas for further research that may help to shed more light on the causes of corneal astigmatism. As interactions between the rigidity of the cornea and the angle, position and tension of the eyelids may all contribute to determining the shape of the cornea, a study that accurately measures all of these factors on a large population of subjects may further our understanding of the causes of astigmatism. Longitudinal studies investigating corneal astigmatism, eyelid morphology and tension in subjects whose corneal astigmatism is known to be changing (for example, populations of older subjects whose astigmatism typically changes towards more ATR) should also increase our understanding of the causes of astigmatism. With advances in technology, our ability to measure and define ocular astigmatism and its components has improved markedly. Therefore, with the rapid technological advances in the ophthalmic

field, our understanding of the aetiology of astigmatism should increase in years to come.

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