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Physiology of Astigmatism

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1. Introduction

We know that the expression emmetropia is a conventional one and that in fact all normal human eyes have mild degrees of spherocylindrical errors (Shilo, 1997) or consist of a bitoric optical system, i.e. have principal meridians of relatively higher and lower powers at right angles. It is generally accepted that genetic factors have a significant role in determining ocular refractive status as well as astigmatism (Hammond et al., 2001) but many conditions and procedures such as surgery, suturing, wound healing, and ocular comorbidities modify the cylindrical status of the eye. Induced astigmatism and surgical correction of astigmatism are extensively addressed in other chapters of this book.

Manifest astigmatism is the vectorial sum of anterior corneal toricity and internal astigmatism. A variety of factors change the magnitude and shift the meridians of these cylindrical components and the perceived subjective astigmatism throughout life. Astigmatism is an extremely dynamic phenomenon, and changes in the shape of optical interfaces, refractive index, optical aperture, eyeball-extraocular structures (eyelids and extraocular muscles) interaction, visual tasks, accommodation, binocularity, tear film status, and even body position induce and modify baseline ocular astigmatism. In this chapter we shall focus on factors that determine baseline, diurnal, functional, and dynamic aspects of 'physiological astigmatism'.

2. Natural course of astigmatism in normal eyes

2.1 Age

Age-related evolution of ocular astigmatism in terms of power and axis has been observed in epidemiologic studies (Abrahamsson et al., 1988; Atkinson et al., 1980; Attebo et al., 1999; Baldwin & Mills, 1981; Ehrlich et al., 1997; Gwiazda et al., 1984; Hirsch, 1963; Kame et al., 1993; Sawada et al., 2008; Stirling, 1920). It is well documented that a high degree of astigmatism is present in neonates and infants; however, the reported amounts show discrepancies (Abrahamsson, et al. 1988; Howland & Sayles, 1985; Isenberg et al., 2004; Kohl & Samek, 1988; Varughese et al., 2005; Wood et al., 1995). The degree of astigmatism is even higher in preterm newborns and has an inverse association with postconceptional age and birth weight (Friling et al., 2004; Varghese et al., 2009). In near retinoscopy without cycloplegia, Gwiazda and colleagues found astigmatism of at least 1 D in about 55% of infants younger than 5 months, 10% of whom displayed a cylinder power of 3 D or more

(Gwiazda et al., 1984). In another study, photorefractive techniques showed that almost all infants at the age of 3 months had at least 1 D of astigmatism, which had decreased to adult levels by the age of 18 months (Atkinson et al., 1980). Likewise, a longitudinal study found astigmatism of at least 1 D in about 40% of infants at 3 months of age with a significant decrease to 4% by the age of 36 months. This reduction appears to be caused by the decrease in toricity of the cornea and the anterior lens (Mutti et al., 2004). Several studies have suggested that corneal shape changes throughout life. The linear reduction of the astigmatism to lower values with age is apparently a part of normal eye maturation (Friling et al., 2004) and emmetropisation. It has been suggested that the high astigmatism in early life induces and activates accommodation (Campbell & Westheimer, 1959; Howland, 1982).

Reports on the axis of astigmatism in infants are contradictory. According to Gullstrand, the natural form of the cornea is against the rule (Gullstrand 1962). Several studies have found a plus cylinder axis at 180±20 (i.e. against the rule) in the majority of infants (Abrahamsson et al., 1988; Baldwin & Mills, 1981; Dobson et al., 1984; Gwiazda et al., 1984; Saunders 1986, 1988). while recent studies have raised questions about the reliability of previously used techniques. These studies have shown that with-the-rule astigmatism is more frequent among infants (Ehrlich et al. 1997; Isenberg et al., 2004; Mutti et al., 2004; Varghese et al., 2009).

As the child grows older, much of the early astigmatism will gradually disappear and transform into with the rule owing to eyelid pressure (Gwiazda et al. 1984). Most of the changes occur at ages 1-3 years, when the vertical and horizontal diameters of the cornea and its elasticity attain adult size and amount (Karesh, 1994). This with-the-rule astigmatism in preschool children is stabilized towards adolescence (Goss, 1991; Huynh et al., 2007; Shankar & Bobier, 2004). However, this is not always true, and a role for myopia development has been attributed to ocular astigmatism, as it may degrade optical blur cues and disrupt emmetropisation, which can lead to axial myopia progression in school-aged children (Gwiazda et al., 2000).

In early adulthood, astigmatism of more than one diopter is infrequent and is still with the rule. Lin and colleagues found a slight increase in the amount of astigmatism in medical students after five years (Lin et al., 1996). Other cross-sectional studies have indicated that mean total astigmatism changes with age, varying from as much as 0.62 D with the rule during adolescence to as much as 0.37 D against the rule in older ages (Baldwin & Mills, 1981). Baldwin and Mills found that steepening of the cornea in the horizontal meridian accounts for a major proportion of the increase in against-the-rule total astigmatism among older patients (Baldwin & Mills 1981). In the Blue Mountains Eye Study (Attebo et al., 1999), mean total astigmatism increased with age from 0.6 D to 1.2 D in youngest (49-59) to oldest (80-97) age groups. From a related study, an average of 1.6 D rise in total corneal astigmatism is documented for each five years of increase in age (Ho et al., 2010). Nuclear sclerosis cataract and the change in refractive index of the crystalline lens at older ages may contribute to myopic astigmatism (Fotedar et al., 2008).

Anterior corneal (and total) astigmatism shows flattening in the vertical meridian with aging, in contrast to a trend towards with-the-rule astigmatism on the posterior corneal surface (Ho et al., 2010). Against-the-rule astigmatism is the most common type of astigmatism in adults over 40 years of age. Interestingly, men are significantly more likely to develop against-the-rule astigmatism (Goto et al., 2001). In general, corneal toricity accounts

for the major component of total astigmatism (Asano et al., 2005; Ho et al., 2010); it is suggested that, with aging, upper eyelid pressure on the cornea and the tone of orbicularis muscle decrease (Marin-Amat, 1956). It has also been demonstrated that with-the-rule astigmatism decreases when eyelids are retracted from the cornea (Wilson et al., 1982). When relative steepening in the vertical meridian is abated, the intrinsic lenticular against-the-rule astigmatism will manifest. Decreases in action of extraocular muscles, especially the medial rectus (Marin-Amat, 1956), and vitreous syneresis and liquefaction may also contribute (Mehdizadeh, 2008).

The contribution of the lens to the ocular astigmatism is relatively constant throughout life (Hofstetter & Baldwin, 1957). Development of this lenticular astigmatism may be due to an emmetropisation phenomenon, as it effectively decreases manifest astigmatism in the early decades of life. But in older ages, lenticular astigmatism is manifested as an against-the-rule astigmatism when the corneal astigmatism is decreased (Artal et al., 2000, 2001; Ehrlich et al., 1997).

2.2 Diurnal changes of astigmatism in the normal eye

The magnitude and axis of astigmatism vary during the day; this variation can be described with regard to changes in eyelid pressure, extraocular muscle tension, pupil size and accommodation. From previous reports, it is postulated that generally the cornea has its flattest shape on awakening and steepens slightly until the evening (Manchester, 1970). Kiely et al. reported fluctuations in corneal asphericity during the day without recognizing a specific pattern (Kiely et al., 1982). Recently, diurnal variations in corneal topography have been studied by Read et al.: corneal wavefront error analysis revealed significant changes in astigmatism during the day (Read et al., 2005); see below.

2.2.1 Lid pressure and muscle tension in near tasks

Changes in corneal contour exerted through eyelid pressure have been widely discussed since the mid-1960s, and transient bilateral monocular diplopia after near work due to temporarily induced toricity in the cornea has been reported by a number of investigators (Bowman et al., 1978; Golnik & Eggenberger, 2001; Knoll, 1975; Mandell, 1966). It is agreed that visual tasks with significant downward gaze, such as reading, can alter corneal curvature owing to eyelid pressure (Collins et al., 2006; Read et al., 2007a). This will lead to horizontal bands on red reflex during retinoscopy (Ford et al., 1997) with concomitant topographic changes and corresponding distortions in Zernike wavefront analysis. Buehren et al. have reported changes towards against-the-rule astigmatism (Buehren et al., 2003).

In a recent study by Shaw et al., the average trend in the astigmatism axis due to near work was said to be against the rule, with approximately 0.25 D change within 15 minutes of 40° downward gaze, where both the upper and lower eyelids are in contact with the central 6 mm of the cornea. They also reported that eyelid tilt, curvature and position are important in the magnitude of corneal changes (Shaw et al., 2008).

Collins et al. demonstrated greater topographical changes in astigmatism during downward gaze with a larger angle (45° versus 25°) and with lateral eye movements (Collins et al., 2006). Studies on the time course of astigmatism regression have revealed slower recovery after longer periods of reading. Moreover, patterns of regression are similar among individuals, with a rapid recovery within the first 10 minutes after reading, and resolution takes between 30 to 60 minutes (Collins et al., 2005).

Regarding the role of extraocular muscles on corneal astigmatism, Lopping has mentioned that continuous use of the medial rectus muscle, especially during near tasks, imposes a force on the cornea which increases its radius of curvature in the horizontal meridian resulting in a shift towards against-the-rule astigmatism (Lopping & Weale, 1965).
These observations have implications for clinical testing, and it would be prudent that examinees avoid near tasks at least 30 minutes prior to refractive and topographic assessments.

2.2.2 Eyelid slant and tension

Apart from temporary changes of corneal curvature due to eyelid pressure, the cumulative effect of the eyelids contributes to naturally occurring astigmatism in healthy adults (see 2.1 above).
Slanting of the palpebral fissure is an important factor affecting corneal toricity (Read et al., 2007b). The magnitude of astigmatism increases as the palpebral fissure diverges from the horizontal plane. Male subjects show more downward fissure slanting, whereas female subjects show more upward fissure slanting (Garcia et al., 2003). People with Down's syndrome (Akinci et al., 2009; Little et al., 2009) or Treacher Collins syndrome (Wang et al., 1990) will show oblique astigmatism partly due to upward or downward slanting of the palpebral fissure.
Thicker or tighter eyelids tend to correspond with higher degrees of astigmatism as well. Asians and Native Americans show higher degrees of corneal astigmatism than other races (Osuobeni & Al Mijalli, 1997).
Corneal rigidity can also contribute to the amount of astigmatism caused by eyelid pressure. For instance, nutritional deficiencies are presumed to decrease corneal rigidity and flatten the horizontal meridian while steepening the vertical one (Lyle et al., 1972).

2.2.3 Pupil dynamics

We know that the optical system of the eye is not coaxial and at least three important axes have been described: optical axis (corneal optical center to lens's optical center), visual axis (object of regard to fovea; line of sight), and pupillary axis. There is a mild physiological pupil decentration in the nasal direction. Such physiological asymmetries, which have long been described (Walsh, 1988), induce coma (Wilson et al., 1992).
The pupil is the aperture for light entrance into the eye; excluding pharmacologic changes, pupil size and its (centroid) lateral position around the optical axis of the eye change according to ambient light (Walsh, 1988; Wilson et al., 1992), accommodative effort, and emotional status (Wilson et al., 1992).
Pupil size correlates with both the magnitude and orientation of astigmatism. Larger mesopic pupil sizes are detected with higher cylinder powers and are also associated with with-the-rule astigmatism rather than against-the-rule and oblique astigmatism (Cakmak et al., 2010). Larger pupil sizes—in low lighting conditions— increase the amount of higher order aberrations such as coma and may intensify the cylinder power in subjective/manifest refraction. Coma has been shown to be correlated with greater amounts of astigmatism (Hu et al. 2004). On the contrary, pupillary accommodative constriction reduces higher order aberrations including lenticular astigmatism (Sakai et al., 2007).
About 0.4 mm temporal pupil centroid shift in darkness was first reported by Walsh (Walsh, 1988); Wilson and Campbell (Wilson et al., 1992) then found shifts of up to 0.6 mm with decreased illumination, in nasal or temporal directions.

2.2.4 Accommodation and convergence

Three decades ago, Brzezinski introduced the expression 'accommodative astigmatism' and claimed that changes in lenticular astigmatism can neutralize corneal astigmatism and reduce the eye's overall toricity (Brzezinski, 1982). Other investigators have suggested that astigmatism increases as the accommodative response becomes larger (Denieul, 1982; Ukai & Ichihashi, 1991). According to Brzezinski, accommodative astigmatism is related to lens distortion due to inhomogeneous lens elasticity, variable constriction in ciliary muscles (which itself changes the lens power), and nonhomogeneous tension of the extraocular muscles during convergence (which causes corneal distortion). These may explain 'lag of accommodation', the phenomenon of less accommodative response than the accommodative stimulus in the horizontal meridian and the resultant with-the-rule astigmatism (Tsukamoto et al., 2001). Pupillary constriction may contribute to such changes as well (see above).

In a more recent study, Tsukamoto et al. found that all emmetropic subjects became astigmatic during accommodation, 93% with the rule (mean -1.96 D). Corneal astigmatism of with-the-rule orientation with mean values of 0.84 D and 0.91 D, respectively, for right and left eyes was detected without a direct association with the amount of accommodation. The eyes became emmetropic just after relaxation (Tsukamoto et al., 2000). Cheng et al. examined wavefront aberrations in a large adult population and found changes in astigmatism towards with the rule with an average of -0.1 D during maximum accommodation (Cheng et al., 2004). The mentioned pupillary and accommodative effects interact with the factors considered above during near tasks (see above).

Accommodation always accompanies convergence during near-vision tasks (Tait, 1933; Rosenfield & Gilmartin, 1988), and it is known that slight changes in cylinder power and axis (towards with the rule) occur during convergence alone (Beau Seigneur, 1946; Lopping & Weale, 1965; Tsukamoto, Nakajima et al., 2000). Seigneur has mentioned that this change is seen in a small percentage of eyes (Beau Seigneur, 1946), and many of the individuals do not experience any discomfort when using the same spectacles for far and near activities; nevertheless, for those who experience such an alteration, separate spectacle prescriptions for near and far distance vision might be beneficial.

2.2.5 Cyclotorsion and binocularity

Eye rotation around the Z axis (rolling or cyclotorsion) modifies the axis of ocular astigmatism in relation to the outside world. There is a complex interaction between accommodation, baseline astigmatism, and torsional alignment (Buehren et al., 2003; Read et al., 2007). These features contribute to eye fusional potential, depth perception, and depth of field (Regan & Spekreijse, 1970).

A number of reasons are implicated for physiological ocular torsion including unmasking of cyclophoria during monocular fixation and fusion loss (Tjon-Fo-Sang et al., 2002; Borish & Benjamin, 2006) and changing of body position from upright to supine (Park et al., 2009; Hori-Komaii et al., 2007; Fea et al., 2006; Chernyak, 2004; Swami et al., 2002); these changes gain remarkable clinical significance when an individual is examined in the seating position but undergoes laser ablation in the supine position. Binocularity is also disturbed during corneal topography and wavefront aberrometry; binocular viewing is not normal during laser ablation in the supine position either.

Although several studies have shown significant incyclotorsion or excyclotorsion of about 2-4 degrees (maximum 9 to 14 degrees) as a result of changing the body position from seated

to supine (Swami et al., 2002; Chernyak, 2004; Fea et al., 2006; Neuhann et al., 2010), a number of investigations have reported insignificant axis changes of less than 2 degrees, which can hardly affect astigmatic correction (Tjon-Fo-Sang et al., 2002; Becker et al., 2004). It has been suggested that axis misalignment of about 4 degrees will lead to 14% cylinder undercorrection during laser ablation (Swami et al., 2002; Neuhann et al., 2010).

As mentioned above, cyclotorsion is frequently seen when switching from binocular to monocular vision, especially in those who have significant cyclophoria (Borish & Benjamin, 2006). Although it has been believed that an occluded eye shows excyclophoria under monocular occlusion of several hours (Graf et al., 2002), it is indeterminate whether ocular torsion resulting from monocular occlusion for a short period during refractive surgery or retinoscopy and monocular subjective refraction is clockwise or counter-clockwise; Hori-Komai et al. and Chang et al. both demonstrated that the magnitude and direction of cyclotorsion is different for each individual (Chang, 2008; Hori-Komai et al., 2007). This has significance in subjective refraction refinement and binocular balancing as well; in fact, a novel position in the phoropter allows maintenance of binocularity (fusion) while clarity of the images of the eyes are independently assessed (Borish & Benjamin, 2006).

Apart from body position and monocularity, which account for static eye rotational alignment, dynamic cyclotorsion also occurs during laser ablation and may result in astigmatic undercorrection and/or induced astigmatism (Neuhann et al., 2010; Chang, 2008; Hori-Komai et al., 2007). Fea et al. showed that blurring of the fixation target happens during ablation (after epithelium removal in surface ablation and following flap lifting in LASIK) and is an important factor for dynamic cyclotorsion, the magnitude of which seems to be significantly higher in the supine position (Fea et al., 2006). Modern eye trackers now are designed to dynamically follow the eye during laser ablation.

2.2.6 Tear film

The tear layer has a refractive index near to that of the cornea (1.33 versus 1.376) and refraction at the air-tear film interface accounts for the majority of refractive power of the anterior ocular surface (Oldenburg et al., 1990). Use of hard contact lenses to correct refractive errors creates a 'tear lens' in the contact lens-cornea interface which resolves the keratometric cylinder (Astin, 1989). This decouples anterior corneal astigmatism from internal astigmatism and manifests as 'residual astigmatism'. The nature of this astigmatism is frequently against the rule and at times can cause eye strain (see above).

The superior eyelid exerts pressure on the cornea, and tear accumulates over the lower eyelid margin due to gravity; this combination induces a vertical coma which may manifest as a cylinder (Montés-Micó et al., 2004a). Localized aggregation of lacrimal fluid is also caused by peripheral corneal lesions such as pterygium (Oldenburg et al., 1990; Walland et al., 1994; Yasar et al., 2003), limbal conjunctival carcinoma (Leccisotti, 2005), or nodules (Das et al., 2005). Such changes cause corneal astigmatism and are largely resolved after excision of the lesion or drying of the tear pool (Leccisotti, 2005; Yasar et al., 2003).

The tear film effect can also be discussed with regard to ocular wavefront changes during blinking. It has been agreed that higher order corneal aberrations show micro-fluctuations during the inter-blink interval. These dynamic variations of ocular surface topography have been widely investigated using high speed videokeratoscopes (E. Goto et al., 2003; T. Goto et al., 2004; Koh et al., 2002; Kojima et al., 2004; Montés-Micó et al., 2004a; Németh et al., 2002). Zhu et al. found that the height of the ocular surface increased about 2 mm within 0.5 s after blinking at the upper edge of the topography map. They also declared that absolute values

in horizontal coma and secondary astigmatism at 45° significantly increased during the inter-blink interval, while secondary astigmatism at 0° decreased considerably (Zhu et al., 2006). In another study, irregular astigmatism induced by tear film breakup was measured and significant increases were observed in coma, spherical aberration and total higher order aberrations (Koh et al., 2002).
It is therefore suggested that measurement of corneal wavefront aberrations for refractive surgery purposes should be done at a fixed interval after each blink (Montés-Micó et al., 2004b). Based on a number of studies that evaluated the variability of topography maps (Buehren et al., 2001; Iskander et al., 2005; Montés-Micó et al., 2004b), an interval of 1 to 4 seconds after blinking is suggested as the optimal time (Zhu et al., 2006).

2.2.7 Retinal astigmatism

From a historical point of view, directional variability in photoreceptor arrangement was proposed as a source of astigmatism (Mitchell et al., 1967); in other words, functional retinal elements may be more abundant or thicker in one axis than the other (Shlaer, 1937). More recently, a 'tilted' retina was simulated and it was observed to manifest as some degree of cylindrical error (Flüeler & Guyton, 1995). This could be the result of unequal lengthening of the sclera in different meridians during axial growth.

3. Conclusion

Although most of the materials presented in this chapter are of investigational interest, there is a resurging interest in these physiological issues owing to refractive surgery. Variations in tear film status, torsional alignment, and pupil features are sources of error in ocular refractive assessment and laser photoablation. Our objective should be firstly not to spoil the innate versatility and optical quality of the virgin eye; and secondly, to avoid inconsistencies in the outcome. The available optical models do not simulate optical performance of the eye perfectly, and the refractive surgery technology —in terms of diagnosis and treatment— does not fully follow our optical models either.
On the positive side, if we intend to make 'super vision' a reality (Applegate et al., 2004), we have to better understand the mentioned dynamics and interaction and 'personalize' treatments. Iris registration for example, can be used to avoid the negative effects of pupil centroid and astigmatism axis shifts during excimer laser ablation (Porter et al., 2006; Jing et al., 2008; Khalifa et al., 2009; Park et al., 2009). But this is just the beginning and we need dynamic optical models and advanced simulations to fulfil the mentioned objectives.
Additionally, an in-depth understanding of physiology of ocular astigmatism may throw light on the pathobiology of refractive errors and lead to new avenues for the prevention of clinically significant astigmatism.

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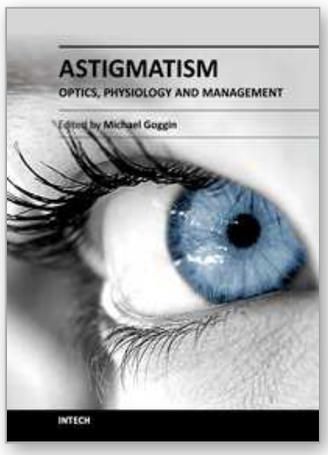

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This book explores the development, optics and physiology of astigmatism and places this knowledge in the context of modern management of this aspect of refractive error. It is written by, and aimed at, the astigmatism practitioner to assist in understanding astigmatism and its amelioration by optical and surgical techniques. It also addresses the integration of astigmatism management into the surgical approach to cataract and corneal disease including corneal transplantation.

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