Major Review

The Impact of Refractive Errors on Strabismus

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Abstract

Strabismus is a very common clinical problem. It is often assumed by the parents of strabismic children and general ophthalmologists, that this is basically a muscular problem, and surgical correction will make the patient's eyes straight for good. It is very important to understand that only good and equal vision in both eyes will lock them into alignment, by virtue of sensory fusion in the brain. This article highlights how uncorrected refractive errors result in amblyopia and strabismus, the significance of their timely and appropriate correction can not only prevent strabismus but restore ocular alignment thus avoiding the need for surgery.

Introduction

The human eye is an optical instrument[1] which allows light to enter the eye and focus at a spot on the retina, the foveal pit, (which has a maximum concentration of photoreceptors, the cones). This maximal stimulation of cones sends impulses via the three neuronal visual pathway to the occipital cortex where a perfect image is perceived, and analysed by the prefrontal cortex. This optical system has certain imperfections in the form of aberrations as well as refractive errors, which not only degrades the retinal image but limits its spatial resolution and clarity.

The aberrations in the optical system of the eye[2,3] occur due to a curved shape of the cornea, its anterior and posterior refracting surfaces, and the bi-convex lens. In bright light, the pupil constricts and blocks the peripheral, divergent rays, thus minimising the effect of spherical aberration. As the pupil enlarges in dim light or at night, more peripheral rays enter the eye and focus anteriorly, making the eye slightly more myopic.[4] The effect of spherical aberrations increases as the fourth power of the pupil's diameter; if the diameter of the pupil doubles, it increases the spherical aberration 16 times. This causes night myopia and haloes around point images, the affect being exaggerated after LASIK or surface ablation for myopia. In addition, in most young eyes, the lens plays a significant role in compensating for corneal aberrations, neutralising them, and improving the quality of retinal image.[5,6] As the lens hardens with age and looses its elasticity, this compensatory mechanism fails; rather it adds to the optical aberrations by the cornea and results in a poor quality of image.

Some degree of refractive errors are seen in all most all babies due to a mismatch between the optical components of the eye so that the resultant retinal image is out of focus. About

90-95% of babies are born with some degree of hypermetropia. Emmetropia has been described by Grosvenor[7] as the "normal" state of the eye in which parallel rays of light focus on the retina while the accommodation is fully relaxed. Others consider emmetropia to range between hypermetropia of +0.50 - +1.50 diopters.[8]

The process of emmetropization occurs during the first 12- 16 months after birth and by the first 5 years of life, both myopic and hypermetropic children become emmetropic in 80% cases.[9]

This process has been postulated to occur by an active as well as a passive mechanism. The active mechanism[10] is a neural process regulated by the degree of blurring of the retinal image: the eye analyses the degree of retinal blur and elongates or shortens proportionately (by changing the axial length of the eye), till the image and retina are conjugate.

A critical factor regulating the axial elongation of eyeball is by altering the amount and composition of the extracellular matrix of the sclera. The image clarity or its defocus at the neuroretina results in release of neurotransmitters by the retinal amacrine cells[11]; Dopamine reduces the axial growth by increasing DNA production and synthesis of proteins and proteoglycans in the sclera[12-14] making it thick and less stretchable thus reducing its elongation. Vasoactive Intestinal Peptide secretion (VIP)[15] stimulates choroidal blood flow and thickening of the spongy choroid while at the same time, thinning and elongation of the sclera. Hence, the axial length of eyeball increases posteriorly. The sclera also has the ability to alter its growth in a sector while the remainder is unaltered. If a portion of the retina has a defocussed image, that part continues to grow and becomes myopic while the remainder with a clear image remains emmetropic, resulting in myopic astigmatism.

This active neural control requires a feedback from the brain, where the image perceived by the retina is analysed. The evidence of such a control was provided by Troilo and Wallman. [16] They severed the optic nerves of chicks and found that this resulted in a reversal of the original refractive error: myopic eyes became hypermetropic while hypermetropic eyes became myopic; emmetropization was more accurate in chicks with an intact optic nerve, meaning that the brain feed back is essential for regulating this process. They further noted that severing the Edinger Westphall nucleus, which controls accommodation, slowed the process of emmetropization but it was not a prerequisite for it. Sorsby[17] has suggested that high level of ametropia results from increase in axial length under a genetic influence. If one parent is myopic, the chance of a myopic child 22.5% while it increases to 42% if both parents are myopic.[18]

Similarly, it has been observed that visual deprivation in neonates from severe congenital ptosis[19-21], corneal opacity[22] , congenital cataract, vitreous haemorrhage or its opacification[23], and retinopathy of prematurity[24,25] causes the eye to elongate and become increasingly myopic. Another observation suggesting that emmetropization has an active component is the association of myopic progression in response to sustained near vision.[26]

This active mechanism is coupled with the passive process of emmetropization in which appropriate and proportional interactive changes occur between the refractive components of the eye and the change in its axial length.[27] In a study on chicks, Troilo and Wallman[28],

concluded that the corneal curvature is a major contributor of astigmatic emmetropization but it doesn't play a major role in spherical emmetropization.[29] Gernet and Olbrich[30] suggest that lenticular changes are responsible; myopic children have thin crystalline lenses, suggesting a mechanical relationship between eye growth and lens compensation. Larger eyes have a larger equatorial diameter, causing more tension and stretching of the zonular fibres. These stretched fibres consequently flatten the lens and reduce its optical power. Another factor is the changing thickness of choroid during the active emmetropization process and it results in altering the vitreous chamber depth (a passive phenomenon).[39] In myopia, the choroid is thin and the retina moves backwards with it, thus the vitreous chamber depth increases; in hypermetropia, the choroid thickens and pushes the retina forwards, thereby reducing the depth of vitreous chamber. The ocular structures responsible for causing large changes in refractive error are the cornea, lens and the depth of vitreous chamber[40], which is determined by the posterior growth of sclera.

The Natural History of Refractive Errors

Most infants are born with a mild hypermetropia (+2.00D), while a small number have a moderate to high range (>3.5D). This is due to a smaller size of the eyeballs as compared to the rest of the body. By the age of 4 years, the eyeballs and the brain attain 85% of their adult size as compared the rest of the body which has grown to only 20%.[41] With the growth of eyeballs, emmetropization process results in a gradual decrease in the level of hypermetropia in most patients[42] so that during the first 3 to 5 years of life and at the most by 6 years, 80% of the children are found to be emmetropic.[43] Ingram and Barr[44,45] stated that a child born with less than +2.50 diopters of hypermetropia is likely to become emmetropic. Whereas a child born with more than 2.50 diopters of hypermetropia will become more hypermetropic by the age of 3.5 years.[46,47] By the age of 5 years, though the prevalence of refractive errors is reduced, its distribution still peaks towards a mild hypermetropia 48. Over the next 10-15 years of life, the prevalence of hypermetropia decreases further while an increase in the frequency of myopia is seen.[49,50]

A small number of infants (<5%), particularly those with a family history of myopia, become myopic as the axial length of eyeball continues to increase under the genetic influence. There is a shift towards emmetropia by 6 months of age for children with an initial myopic spherical equivalent.

On average this group does not reach the same level of hyperopia as those with an initial hypermetropic spherical equivalent. Infants with high myopia also later retained most of it. However, after 5 years of age, the refractive status tends to return to the original level of myopia. Therefore, it might be possible to predict refractive status in older children based on the earliest manifest refraction, with one year being optimal.[51] Myopic progression has been found to be associated with other factors like female gender, ethnicity, younger age of onset, high IQ score, and prolonged study hours (by increasing the accommodative demand and eye strain).[52,53] The evidence has been provided by Chua et al.[54], who found that elongation of axial length and myopic progression can be slowed down by reducing accommodation with 1% atropine eyedrops once a day.

At birth, the average amount of astigmatism is 2.00 D which decreases to 1.00 D by 2.5-5 years of age[55,56] due to the emmetropization process; 1/3 of their spherical equivalent and 2/3 of the astigmatism reduces due to flattening of cornea during the first two years of life. This has been found in 90% of the children[57] though not in all races. Astigmatism present prior to the age of 6 months usually disappears by the age of 1 year but if it appears in the second six months of life, and is more than 1.50 D, then it is likely to persist and can lead to amblyopia if not corrected with glasses. Emmetropization process corrects "With"-the-rule astigmatism more than "Against"-the-rule astigmatism which is also a risk factor for becoming myopic at an earlier age and amblyopia. According to Abrahamsson and Sjostrand[57], low amounts of anisometropia (<2.50 D) is commonly found during the normal growth period of the eye[58]; children with 3.00 D or more anisometropia at one year of age have a 90% chance of retaining it at the age of 10, and a 60% risk of developing amblyopia.[59] The emmetropization process cannot correct >5.00 D of anisometropia[60] and may result in juvenile microtopia.

Optics of Hypermetropia

In emmetropia, parallel light rays from a distant object are focused by the lens in the absence of accommodation onto the retina (fovea) and a clear image is formed. In hypermetropia, because of a short axial length, light rays are focused behind the retina resulting in a blurred image. Hence accommodation is called upon which increases the curvature/thickness of the lens, thereby increasing its refractive power so that distant light rays are brought to focus upon the retina. The rays coming from a near object are more divergent. As more accommodative effort is needed to focus the near object, the eye converges as well due to accommodation/convergence synkinesis resulting in an Esotropia (ET). According to Ingram, et al.[61], infants with an esotropia or a microtropia did not show a spontaneous reduction of hypermetropia by the emmetropization process and were more likely to have accommodative problems. Both fixing and non-fixing eyes demonstrated accommodative abnormalities with poor convergence, showing that the underlying defect was congenital rather than refractive.

Children have a sufficient accommodative reserve to maintain a clear retinal image without producing asthenopia.[62] However a constant need for accommodative effort for near work results in tearing eyes, squinting and facial contortions while reading, frequent blinking, constant or intermittent blurring of vision, focusing problems, difficulty with or aversion to reading, decreased binocularity and eye-hand coordination. The presence and severity of these symptoms is variable and depends upon the degree of hypermetropia. Hypermetropia usually stabilises by the age of 6-8 years[63] and starts reducing in amplitude with time. However, in children, who are constantly reading or doing close work for long periods, constant accommodation results in spasm of ciliary muscle and artificial myopia.

Impact of Uncorrected Hypermetropia on

Vision & Strabismus:

1. A mild to moderate degree of hypermetropia or astigmatism is present in 90% cases of infantile esotropia and should be fully corrected as it may lead to amblyopia. A child with true, essential infantile ET should have a surgical correction of strabismus by 18 months age so that stereopsis can develop. Any astigmatic error should be corrected by glasses.

2. Anisometropic hypermetopia (> 1.5D) or hypermetropic astigmatism of 1D, persisting beyond 2 years of age, results in amblyopia.[64]
3. If both eyes have the same degree of hypermetropia, then an alternating ET develops. Since the child fixates alternately with either eye, amblyopia does not develop.

4. If one eye is more hypermetropic, it accommodates more and converges more resulting in a unilateral, constant ET. A constantly in-turned eye loses foveal fixation, the child tends to prefer the emmetropic or less hypermetropic eye for seeing and the more hypermetropic eye (with a blurred image) is neglected by the brain. It is suppressed by the good eye, its neuronal connections to the brain shrink and it becomes amblyopic.

5. A large, constant ET results in an eccentric fixation as it never straightens to focus image on the fovea.
6. In a constantly esotropic eye, the MR never relaxes as the eye never assumes a primary position; its constant contraction results in its hypertrophy and contracture. Even if such an eye is given full hypermetropic correction, the hypertrophic muscle does not relax fully, a small amount of ET still persists and is erroneously labelled as a partially accommodative ET.

7. Uncorrected hypermetropia (>3.5 D in one meridian) results in blurring of vision, reduced binocular vision, constant accommodative effort, fatiguability. These factors contribute to poor motor and cognitive development in younger children (9 months to 5.5 years) and poor performance at school in older children. Screening by visual acuity testing in all preschool children is very important.

8. Full optical correction of significant hypermetopia during infancy in the absence of strabismus, may interfere with the process of emmetropization[65] but partial spectacle correction is safe and reduces the incidence of subsequent strabismus. 9. In the presence of an esophoria or ET, no matter of how small a magnitude, full hypermetropic correction is

mandatory to achieve a foveal fixation binocularly. This can only be achieved by full cycloplegia with atropine eyedrops as it neutralises even the latent hypermetropia (due to the tone of ciliary muscle = 1-1.50D). Cycloplegia with cyclopentolate eyedrops does not neutralise the latent hypermetropia but only the manifest hypermetropia which an individual can correct by accommodating and is called Facultative Hypermetropia. If the error is large, then even by fully accommodating, the objects are not seen clearly, especially for near. This remaining amount of Hypermetropia that still remains uncorrected by accommodation is called Absolute and needs correcting glasses (Manifest= facultative + absolute).

Optics of Myopia

The term myopia means “I close eyes”, a myopic person sees distant objects with half closed eyes. Because of a large axial length, parallel rays of light from a distant object are focused in front of the retina resulting in a blurred distant image. Half-closed eyes create the affect of a pinhole, thus reducing the extent of blurred image. Since the rays from a near object are divergent, they focus on the retina, producing a clear image. Hence a myopic person can see near objects clearly (short-sightedness); the farthest distance at which the vision is clear is called the Far Point (Punctum Remotum). In an emmetrope, the far point is at infinity while in a myope, the higher the degree of myopia, shorter is this distance e.g. in myopia of 1D, far point is at 1 meter, in myopia of 2D, far point is 1⁄2 meter.

Impact of Myopia on Vision & Strabismus:

1. Myopia is mostly due to an increase in axial length of the eyeball. 1mm increase in axial length causes myopia of 3D. Increase in corneal curvature by 1mm causes a myopia of 6D but it is seen less frequently as the normal emmetropization process encourages corneal flattening. It is seen in pathological conditions like keratoconus, keratoglobus. Index myopia (due to increased refractive index of lens) is seen in diabetes and nucleus sclerosis.
2. A Limited Horizon; a myope can only see clearly till the far point. Hence his whole world is limited to that distance. This can result in psycho-social problems in children with uncorrected myopia.
3. Eyestrain, diplopia for near work. The eyes normally converge when focussed at a near object as in reading, writing; convergence causes accommodation as well because of accommodation/convergence synkinesis in the brain (both reflexes operate together). The divergent rays from near objects focus clearly on the retina without the need for accommodation, but because of the synkinetic reflex, this extra accommodation focus rays in front of the retina resulting in a blurred near image. Hence the myope gives up the effort to converge allowing one eye to deviate outwards intermittently, resulting in diplopia for near and an exophoria initially, progressing later to exotropia (XT), when the convergence effort is totally abandoned. In a study by Noha et.al.[66], myopia was associated with intermittent XT in 90% cases by the age of 20 years. Intermittent XT is seen in 1% of healthy children in USA while esodeviation is more common, though the reverse is seen in Asian populations. Another study also showed a strong association between myopia and XT. The explanation given by the authors is that intermittent XT promotes the development of myopia through increasing accommodative demand, as the reduction of accommodation slows myopic progression, thereby claiming that intermittent XT is a risk factor for myopia.This view does not sound rational as myopia sets in first and exophoria, progressing to an intermittent XT, and then a constant XT is noted at a later age.

4. In a study by Kushner [67], intermittent exotropia was treated with overcorrecting minus lens therapy, on the principle that minus lenses in spectacles stimulate accommodative convergence, thereby, reducing an exotropic deviation. According to previous studies, excessive accommodation can result in myopic progression. However, in this study, it was found that the overcorrecting minus lens therapy did not result in myopic progression over time. This study again refutes the view by Noha et al., that excessive accommodation results in myopic progression.

5. It has been found that increased outdoor activities[68], including sports and leisure time, were associated with less myopia and a more hypermetropic mean refraction. Hence myopic children should be discouraged using cell-phones, laptops for long periods of time and promoted to spend more time outdoors. They should be encouraged to take a break intermittently during prolonged study hours.

6. Bad reading habits result in myopic progression by causing a constant eyestrain, increasing the IOP and stretching the coats of the eyeball, thereby increasing the axial length. Children should be discouraged to read lying in bed, or stooping over their books, laptops, mobile phones.

7. Uncorrected anisometropic myopia of >4.50 D results in amblyopia because of a constantly blurred image in that eye. 8. Correcting glasses prescribed should be in the weakest minus lens that gives a 6/6 vision for distance. Over- correction of myopia should be avoided as it interferes with the emmetropization process. Children should be encouraged to wear the refractive glasses constantly to restore the convergence/accommodation balance for near as well.

9. Myopia is fully corrected in the presence of an exophoria or XT to stimulate accommodative convergence.
10. If an Esophoria is noted with the patient wearing his myopic correction, then it is important to reduce the minus correction.

11. In adults, it is important to decrease the minus correction for near as the amplitude of accommodation decreases with age
12. High degrees of myopia at birth can result in esotropia in early childhood. In this case, the infant's far point is very close to the eyes making the eyes converge all the time to see clearly at this distance; the vision for more remote distance is poor so convergence is not relaxed on looking further away resulting in constant esotropia.

Conclusion

Uncorrected refractive errors can cause a lot of visual morbidity in children and interfere with their development. They can result in amblyopia and strabismus, which can

totally be avoided by early and appropriate spectacle prescription. Except in congenital strabismus, surgery is not the answer but appropriate refractive correction with glasses which not only restore a normal visual acuity in either eye but also the muscle balance.

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