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ABSTRACT

The disruption to the emmetropisation process is commonly cited as one important reason for not prescribing glasses for hypermetropia in infants – if, of course, there are no other indicators present (anisometropia or strabismus). The purpose of this case report is to re-visit what is considered to be a 'normal' amount of hypermetropia in infancy and the issues concerned with refractive correction and prescribing in younger children with hypermetropic errors. Using an illustrative case study, it is also aimed to highlight the speed or rate at which emmetropisation can take place.

Keywords: emmetropia, emmetropisation, hypermetropia, refractive error

INTRODUCTION

nfants are generally born hypermetropic, with a gradual decrease in this hypermetropia during the early years of life.¹⁻³ The process by which this change in refractive error occurs is known as emmetropisation. When infants or children are found to be hypermetropic, the clinician is often faced with challenging questions on how to manage the refractive error. The principles behind prescribing glasses in young children are different to those of adults and take into consideration cortical plasticity and emmetropisation or the influence of refractive correction on this process. We report a case study of an infant displaying a rapid change in refractive error towards emmetropia from initial higher levels of hypermetropia to highlight and discuss issues concerned with refractive correction and prescribing in younger children with hypermetropic errors.

CASE STUDY

A 5½ month old baby, N.E, presented for routine eye examination, having a family history of anisometropic amblyopia (father). On examination, no manifest deviation was apparent and ocular movements were normal. A

Correspondence: **Zoran Georgievski** Department of Clinical Vision Sciences, La Trobe University, VIC 3086, Australia. Email: z.georgievski@latrobe.edu.au fusion response was also demonstrated to a 'prism reflex test'. Retinoscopy following cycloplegia (cyclopentolate 1%) revealed a hypermetropic refractive error, $+4.00/-1.00x180^{\circ}$ and $+3.50/-1.00x180^{\circ}$, in the right and left eyes respectively. However, glasses were not prescribed.

On follow-up examination at 17 months of age, N.E.'s refractive errorhaddecreased significantly. Repeat cycloplegic retinoscopy revealed a refraction of $+1.00/-0.50 \times 180^{\circ}$ and $+0.50/-0.50 \times 180^{\circ}$, in the right and left eyes respectively – a decrease in the hypermetropic and astigmatic refractive error initially present. Specifically, there was a decrease of 2.75 diopters in the spherical equivalence of each eye. The remaining ocular examination, which included a cover test, assessment of ocular movements and the prism reflex test, continued to be unremarkable.

At the final follow-up visit at $3\frac{1}{2}$ years of age, N.E. had only a slight further decrease in his refractive error. Cycloplegic retinoscopy yielded refractive error of $+0.75/-0.25\times180^{\circ}$ and +0.50, in the right and left eyes respectively. Further, N.E. demonstrated stereo acuity of 550'' (Lang stereotest) and visual acuity of 6/9 in each eye (Lea Symbols chart).

DISCUSSION

Low levels of hypermetropia are regarded as normal in infant populations. Studies reporting on the distribution of refractive error in healthy infants have reported that over 90% of preschool children have hypermetropia less than approximately 3D and astigmatism of less than $1.75D.^{2-4}$ The most rapid shift in refractive error occurs within the first year of life, particularly between 3 and 12 months^{1,2,5} In fact, Ingram et al⁶ reported that if emmetropisation occurred, it was complete in 82% of infants by 1 year of age. In our case, N.E.'s hypermetropic refractive error was of a moderate amount and decreased from a spherical equivalent of +3.50D to +0.75D and +3.00D to +0.25D in the right and left eyes, respectively, in the space of just less than 1 year.

Studies of infants have shown that the emmetropisation process is closely related to the initial refractive error present in the first 6 months of life^{7,8}. This is such that higher levels of ametropia demonstrate a more rapid decrease in refractive error relative to those with initially lower levels of ametropia. Saunders et al⁸ reported that for each dioptre of hypermetropia present during the first 6 months of life, the level of ametropia present decreased by 0.06D per month between the initial refraction and 12-17 months of age. Mutti et al⁷ also reported on this phenomenon, noting that higher levels of hypermetropia were related to faster rates of axial growth. This growth was subsequently effective in decreasing hypermetropia to emmetropia during the first year of an infant's life. Mutti et al⁷ observed, however, that this was only effective in infants with up to 5D of hypermetropia. Infants with a hypermetropic refractive error greater than this tended not to emmetropise effectively. Conversely, infants with minimal refractive errors close to emmetropia showed little shifts or in some rare instances became myopic.

The precise mechanisms regulating the process of emmetropisation are not well understood. Experimental studies suggest that 'active' visual feedback related to focusing errors drives shifts in refractive error.^{7,9-12} For instance, inducing refractive errors in chicks has been shown to be associated with changes in axial length.¹⁰ Variations to the power of ocular components also play a role in the modulation of refractive error. The power of the cornea and crystalline lens has been shown to be inversely correlated to axial growth, but it is thought that this is most likely related due to the growth of the eye rather than being the mechanism for emmetropisation.⁷

Understanding the emmetropisation process has important clinical implications for the management of refractive errors in infants. Given that mild to moderate amounts of hypermetropia 'emmetropise' in straight-eyed infants, there is little need to prescribe glasses for this population. However, the level of hypermetropia that requires correction is contentious. Despite this, the American Academy of Ophthalmology (AAO)¹³ has provided guidelines for prescribing glasses in young children and suggests that the threshold for prescribing glasses for hypermetropia in children up to 1 year of age is 6D. The threshold for children aged up to 2 years is 5D and for children up to the age of 3, 4.5D. Spectacles are

recommended if correction improves visual acuity or ocular alignment for children 4 years or above.

The AAO further suggests that hypermetropic correction should be reduced by up to 2D in children (except where esotropia is present and full correction is required for optimal alignment). Under-correction of hypermetropia is often recommended on the basis that the emmetropisation process can be impeded by correcting hypermetropic refractive errors.^{6,14} Ingram et al^{6,14} reported that the consistent wearing of hypermetropic spectacle correction from the age of 6 months was associated with the maintenance of high levels of hypermetropia. However, the effect of spectacle correction continues to be debated. Atkinson et al^{15,16} have, for instance, reported that spectacle correction only has a transient effect on refractive error between 9 and 18 months of age and that treatment does not impede emmetropisation in the longer term.

This paper highlights the issues concerned with prescribing hypermetropic correction in the paediatric population. In the case report presented, it is shown that infants with initial moderate hypermetropia levels, similar to those with mild hypermetropia, do emmetropise during their first year of life. This, alongside various cross-sectional and longitudinal studies,^{1,2,4-6} suggest that in the absence of strabismus and/or anisometropia, infants with moderate amounts of hypermetropia can be conservatively managed and monitored by regular cycloplegic refractions, rather than have glasses prescribed. The immediate correction of refractive error should best be avoided. Persistent high levels of hypermetropia beyond the first year may warrant treatment, especially if there is no improvement.

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