SCIENTIFIC CORRESPONDENCE

Effect of spectacles on changes of spherical hypermetropia in infants who did, and did not, have strabismus

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Table 1 Effect of spectacles on changes in spherical hypermetropia in normal children and those with strabismus

Group	No	Fixing eyes					
		Start		End		Change	
		Mean	SE	Mean	SE	Mean	SE
Normal	189	+4.57	0.07	+3.33	0.08	-1.24	0.09
T0	89	+4.47	0.11	+3.13	0.08	-1.34	0.13
T+/-	55	+4.60	0.10	+3.25	0.13	-1.35	0.15
T+	45	+4.74	0.15	+3.85	0.20	-0.89	0.18
Strabismus	100	+5.15	0.13	+4.87	0.16	-0.27	0.14
T0	53	+4.85	0.15	+4.69	0.21	-0.16	0.19
T+/-	25	+5.14	0.22	+4.76	0.29	-0.38	0.27
T+	22	+5.85	0.31	+5.43	0.42	-0.42	0.35
Total	289	+4.77	0.06	+3.87	0.09	-0.90	0.08
Group	No	Non-fixing eyes					
		Start		End		Change	
		Mean	SE	Mean	SE	Mean	SE
Normal	189	+4.65	0.07	+3.51	0.08	-1.15	0.09
T0	89	+4.57	0.11	+3.33	0.09	-1.24	0.13
T+/-	55	+4.55	0.12	+3.42	0.14	-1.13	0.15
T+	45	+4.96	0.14	+3.97	0.21	-0.98	0.18
Strabismus	100	+5.37	0.12	+5.53	0.15	+0.16	0.13
T0	53	+5.08	0.16	+5.36	0.21	+0.28	0.18
T+/-	25	+5.36	0.22	+5.37	0.31	+0.01	0.28
T+	22	+6.07	0.28	+6.09	0.33	+0.02	0.26
Total	289	+4.90	0.07	+4.21	0.09	-0.70	0.08

Abstract

Aim—To explore why emmetropisation fails in children who have strabismus.

Methods—289 hypermetropic infants were randomly allocated spectacles and followed. Changes in spherical hypermetropia were compared in those who had strabismus and those who did not. The effect of wearing glasses on these changes was assessed using *t* tests and regression analysis.

Results—Mean spherical hypermetropia decreased in both eyes of “normal” children ($p<0.001$). The consistent wearing of glasses impeded this process in both eyes ($p<0.007$). In the children with strabismus, there were no significant changes in either eye, irrespective of treatment ($p>0.05$).

Conclusions—In contrast with normal infants, neither eye of those who had strabismus emmetropised, irrespective of whether the incoming vision was clear or blurred. It is suggested that these eyes did not “recognise” the signal of blurred

vision, and that they remained long sighted because they were destined to squint. Hence, the children did not squint because they were long sighted, and glasses did not prevent them squinting.

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Children who have strabismus fail to emmetropise,¹⁻⁴ and we found⁴ deficient resting,⁵ or tonic,⁶ accommodation in both eyes before infants squinted. This complemented other reports⁷⁻¹⁵ of “weak” accommodation in such eyes and was probably not the result of “failed” emmetropisation.⁴ Although reduced accommodation has been thought to impede emmetropisation,^{2,16} the clarity of visual input is considered^{17,18} to be the main factor controlling eye growth. In order to explore further the association between deficient accommodation, emmetropisation, and strabismus, the data from a randomised trial¹⁹ of treating hypermetropic infants with glasses have been re-examined. We have assumed that the prime effect of glasses was to clear vision, and we have concentrated on changes in the spherical refraction, because infants’ eyes naturally focus on the less hypermetropic meridian.²⁰ Changes in astigmatism are considered separately (Ingram RM, Gill LE, Lambert TW. Reduction of astigmatism after infancy in children who did who did and did not have strabismus. In preparation).

Sample and methods

In all, 615 (9.18%) of 6700 unselected 6 month old infants who attended for “screening” after instillation of cyclopentolate 1% had more than +5.25 D hypermetropia in at least one meridian. Approval was obtained for 372 (60.5%) of these to be randomly prescribed glasses (2.00 D less than the retinoscopy figure for each meridian) for constant wear.¹⁹ Those wearing glasses had non-cycloplegic retinoscopy, through their glasses, at all follow up attendances, and the prescription was amended until the cycloplegic retinoscopy of both eyes indicated less than +4.25 D in one meridian and less than +1.50 D astigmatism, when glasses were discontinued. They were subdivided according to whether they were deemed (by observations at each attendance)

to be wearing glasses consistently (T+, n=67) or not (T+/-, n=80). A total of 77 had a squint (five exotropia) diagnosed by the cover test and 23 a microtropia diagnosed by the 4 dioptre prism test.²¹ The mean initial astigmatism of the fixing eyes of those who had strabismus was significantly ($p<0.001$) less (0.31 D) than in those who did not, but there was no difference ($p>0.50$) in the non-fixing eyes.

All the refractions were done by RMI, and unaltered cycloplegic retinoscopy figures are quoted. The last retinoscopy was done when a squint was diagnosed (mean 37.99 (SD 11.67) months) or at 42+ months (mean 44.18 (7.21) months). Eyes of "normal" children were designated as "fixing" or "non-fixing" according to their vision or last refraction. If these were equal, they were randomly designated.

The clinical data were analysed using EPI-INFO and SPSS software, t tests, and multiple linear regression.

Results

Because of low numbers, the 23 children with microtropia were combined for analysis with the 73 who had squint, and are referred to as the "strabismus" group. Mean hypermetropia decreased significantly ($p<0.001$ using paired sample t tests) in both eyes of the normal group (see Table 1), but not in the strabismus group ($p = 0.06$ for fixing eyes, $p = 0.24$ for non-fixing eyes).

Each of the three treatment subgroups of the normal children showed a significant reduction ($p<0.001$) of the mean hypermetropia in both eyes, though the reduction was smaller in the T+ group (that is, those who consistently wore glasses). To explore this further, linear regression (see Fig 1) was used to relate the final value of hypermetropia (y) to its starting value (x), with differences in slope of the regression lines being used to compare responses to the three treatments—T0, no

Figure 1 Effect of spectacles on changes in spherical hypermetropia of normal children (linear regression with 95% confidence intervals).

glasses; T+/-, glasses sometimes worn; T+, glasses consistently worn. In the T0 subgroup the slope of the regression lines did not differ significantly from zero ($p=0.72$ for fixing eyes, $p=0.32$ for non-fixing eyes, estimated slopes of 0.03 and 0.09 respectively); in other words, the final value did not depend on the starting value. In the T+/- subgroup the same was true for the fixing eyes ($p=0.14$, estimated slope 0.24) and for the non-fixing eyes there was only marginally significant dependence on the starting value ($p=0.02$, estimated slope 0.37). However, in the T+ subgroup the final value was linearly related to the starting value with a slope of 0.68 ($p=0.001$) for the fixing eyes and 0.75 ($p=0.006$) for the non-fixing eyes. The consistent wearing of glasses (T+) by the normal children was therefore associated with the maintenance of initial high levels of hypermetropia—that is, their emmetropisation was impeded.

In the children who had strabismus there was no significant change in the mean hypermetropia of either eye, both overall and in each treatment subgroup (all $p>0.05$).

Discussion

There was a linear decrease of spherical hypermetropia in both eyes as these normal children grew (Fig 1), and this was impeded by the consistent wear of glasses ($p<0.007$). This confirms the findings of Hung *et al.*,²² but differs from those of Atkinson *et al.*,¹⁴ who did not separate squinters from non-squinters. If blurred vision is the principal stimulus for emmetropisation,^{17,18} its removal (by glasses) would explain the failure of the consistently treated normal infants to emmetropise. Although there is a risk that glasses might leave them hypermetropic, we do not know if this would be permanent.

In the children who had strabismus, there was no significant change in either eye, and if this was due to deficient accommodation, we would have to ask why, in the first instance, accommodation was only deficient in infants who later had strabismus. Both accommodation and emmetropisation are thought to be stimulated by blurred vision, but these strabismic children failed to emmetropise irrespective of whether their vision was blurred, or cleared by glasses. We suggest that deficient emmetropisation and deficient accommodation, found⁴ before children squint, could be the result of an inability to recognise (or respond to) a stimulus of blurred vision, and that both these defects and strabismus might be caused by one, congenital, lesion. This might be situated in the retina of both eyes, because failure to emmetropise has been reported in association with congenital retinal abnormalities.^{23,24} Finally, if these children had squinted because they were long sighted, and blur induced accommodation had “driven” the excess convergence, we have to ask how blurred vision

could “switch on” accommodation when it had previously⁴ not done so, and why did glasses, which should have nullified any need for extra accommodation, not prevent them¹⁹ squinting? We suggest that the sequence of events is that (i) they remained long sighted because they were destined to have strabismus, and (ii) disparity/diplopia initially triggered convergence driven^{25,26} accommodation, which may then interact with accommodative vergence,²⁷ permitting glasses to have some effect, but neither a curative nor a preventive one.

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