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Editorials

Preschool visual screening

Visual screening has an intuitive appeal. Amblyopia in preschool children is asymptomatic and, if it is not due to (or accompanied by) strabismus, will only be detected fortuitously unless screened for. The visual outcome is thought to be better the earlier treatment is initiated, so to the author of an editorial in 1977, preschool screening of all children was mandatory.[1] The only question was how it could best be achieved. Over the past 10 years and, to the credit of the profession, before the introduction of formal systems of audit or evidence based medicine, these certainties have been challenged.[2 3] Concern has focused on the effectiveness of the tests used to identify amblyopia and of the treatment used to reduce it. The cost of mounting a nationwide preschool visual screening programme also needs to be considered, as well as the implications, both social and financial, for the amblyopic child and his or her family. How much does it matter if amblyopia is not detected before starting school?

Williamson and colleagues from Glasgow have made an interesting and important contribution to this debate. The findings of their study published in this issue of the *BJO* (p 1068) may not be universally applicable as the setting was an inner city. This may affect not only the social class mix, with an overrepresentation of social class 3, but also the ease of access to the clinic for treatment. Furthermore, the study design meant that false negatives from the screen could not be identified. However, a number of lessons emerge. Of the total population at risk of amblyopia, about 90% were sent appointments but only 57% attended for screening. A battery of tests was used with visual acuity of less than 6/6 (Snellen) being the best predictor of the presence of amblyopia. At this level, there was a high false positive referral rate, with abnormal refraction (especially myopia) the commonest anomaly. If a cut off of less than 6/9 was used, nearly 20% of the amblyopes would have been missed. Other variables tested at screening (cover test, 20 dioptre prism test, etc) added little additional information. Only 65% of the amblyopes completed the full programme of treatment with the default rate increasing as socioeconomic status reduced. Even in social class 1, non-attendance was greater than 20%. The visual acuity was nearly twice as likely to improve in those who continued to attend but was very unlikely to deteriorate in either group.

The authors have clearly shown the value of reduced visual acuity as the best predictor of amblyopia. Because of the crowding phenomenon, unless Snellen line acuities are tested, cases will be missed. Also, because the visual acuity of children increases with age when tested longitudinally by the same method, if less than 6/6 is used as the cut off point, the test is a better discriminator as the child gets older. Recent information from other sources is relevant. Woodruff and colleagues have also demonstrated that good compliance with treatment is associated with a better visual outcome.[4] About half the children treated by this group achieved 6/9 or better visual acuity but this compares poorly with results from elsewhere with 80%[5] or nearly 100%[6 7] of patients improving to this level with intensive treatment. The financial burden of frequent clinic attendances for those in low socioeconomic groups, especially if successive children in the family have amblyopia, is considerable if the best results are to be obtained. It is also notable that in a comprehensive survey of outcome, the success rate of occlusion treatment for amblyopia varied little with the age of starting treatment.[8] There is no significant association between young age at presentation and better outcome. The worse the initial visual acuity – that is, the more severe the amblyopia, the more difficult it is to treat. Against this, more severe amblyopia is more likely to be strabismic or have a strabismic element and therefore be detected by the parents.[5] The cases that remain undetected in the community are more likely to be anisometropic amblyopes with less severe visual deficits that may be more likely to respond to treatment in the first year at school.[9]

Williamson and colleagues make the point that although the only effective screening test for amblyopia is visual acuity, it is not particularly efficient because of the high number of children with refractive problems referred. Refraction of those with reduced visual acuity at the time of screening would overcome this problem. This could be done by autorefraction or after cycloplegia by orthoptists[10] with the referral of non-amblyopes to opticians.

Current debates between purchasers and providers of healthcare offer the opportunity to change practice for the better. Provider units have tended thus far to react to demands or suggestions rather than proactively offer new approaches. The literature reviewed above indicates that unselective preschool visual screening is inefficient and unnecessary. Resources should be targeted to the screening of children in their first year at school, with refraction of children with reduced visual acuity before referral to the hospital eye service.

J ELSTON

Radcliffe Infirmary,
Oxford OX2 6HE

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Topical aldose reductase inhibitor

Polymegathism is a term used to describe increased variability in the size of corneal endothelial cells. Polymegathism came to notice when the advent of the clinical specular microscope made the close examination of the corneal endothelium a relatively simple clinical procedure. With the addition of digital morphometric analysis systems to clinical specular microscopes, quantification of variations in cell size and shape also became simple and reliable. Polymegathism and pleomorphism (increased variability in cell shape) occur with age,[1–3] after trauma (including cataract surgery),[4 5] with contact lens wear,[6–10] with a number of disorders affecting the anterior segment of the eye,[11–14] and after keratorefractive procedures.[15–17] They also occur in diabetes.[18–20]

Polymegathism is often seen in diabetics when no other ocular disease is apparent. The precise mechanism is yet to be determined. It has been attributed to the accumulation of sorbitol in endothelial cells which in turn has been linked to the presence in corneal endothelial cells of aldose reductase, the rate limiting enzyme in the polyol pathway associated with the conversion of glucose to sorbitol.[21–23] In addition to corneal endothelial cells, the enzyme is present in the lens and the retina.[24–26] Experiments in diabetic animals have implicated sorbitol accumulation with osmotic changes and the development of cataracts.[27 28] A similar process is proposed to explain irregularities in the size of corneal endothelial cells. When blood glucose levels are high, there is sufficient substrate for the polyol pathway to become important and for the intracellular osmolarity to rise and water to accumulate and alter cell size and shape. Whatever the mechanism, inhibition of aldose reductase reverses endothelial polymegathism.[21 22 29 30]

It has been demonstrated that galactose fed dogs developed polymegathism and that the concomitant administration of aldose reductase inhibitors prevented this.[22] Furthermore, it has also been shown that topical administration of aldose reductase inhibitors could reverse polymegathism in animals.[21 23 29 30] More recently it has been shown that corneal epitheliopathy and decreased sensation in human diabetics can be reversed using the same therapeutic approach.[31 32] The paper by Ohguro *et al* in this issue of the *BJO* (p 1074) confirms that endothelial polymegathism will also respond to this treatment.

The clinical significance of endothelial polymegathism is uncertain. Although it is generally acknowledged that polymegathism occurs in diseased corneas, the precise pathology has not been identified. Until now nobody has been able to relate polymegathism to essential endothelial structures such as pump sites. There is some evidence, at least at an anecdotal level, that corneas exhibiting polymegathism have a reduced functional reserve capacity and that they are more prone to develop oedema after cataract surgery.[4] The decreased functional reserve has also been demonstrated in controlled experiments on humans subjected to hypoxic stress.[1] Uncertainty about the clinical significance of polymegathism is due to the paucity of reports of corneal disease associated with the phenomena. Polymegathism is common in contact lens wearers and in diabetics, but it is very unusual for patients with polymegathism as a result of hard contact lens wear to go on to develop corneal failure and bullous keratopathy, or for patients with diabetes and polymegathism to suffer a similar fate.

The findings set out in the paper by Ohguro *et al* may have direct clinical benefits. If the decreased functional reserve capacity ascribed to patients with polymegathism is real then the risk of anterior segment surgery to the cornea may be reduced by the use of topical preparations of aldose reductase inhibitors. However, the importance of the observations may go beyond this.

Firstly, there is the prospect of the development of pharmacology relevant to the complicated and important metabolism of the corneal endothelium pump; even though the precise physiological mechanisms of this are not fully understood, the prospects of pharmacological manipulation are tantalising. Secondly, the prospect of reducing the systemic complications of diabetes by medical means opens the way to new opportunities on a broader front.[28]

If we are to explore these possibilities further, more needs to be known about the basic biology of the cornea. Much has been learned recently, but much more remains unknown.

DOUG COSTER

Department of Ophthalmology,
Flinders Medical Centre,
South Australia

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J Elston

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