Review of children referred from the school vision screening programme in Kettering during 1976-8

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Abstract

The progress of 108 children who were identified by the vision screening programme in school as having defective vision (excluding those with puberty onset myopia) was reviewed. Treatment of these children resulted in improvement in visual acuity of the worst eye (two lines or better) for 16 children. Eighteen children had severe amblyopia (6/24 or worse). Among these the vision of only five was improved by treatment. Two thirds of the children had refractive errors in the better eye which required correction.

It seems sensible to identify and treat children with bilateral refractive errors, but the need to treat children with lesser degrees of amblyopia is questioned.

Introduction

Screening of vision in schoolchildren started in 1908 because it was thought that defective vision impeded education. In the postwar years priority has been given to treating children with amblyopia (with or without squint) and myopia. But repeated calls for vision screening before entry to school suggest that many ophthalmologists consider the results of treating amblyopia discovered at this age to be unsatisfactory. In this study the ophthalmological findings in a group of children (excluding those with puberty onset myopia) who were identified at school age by the school vision screening programme in Kettering were analysed, and the results, in terms of the acuity of the worse seeing eye, of treating those who had defective vision are reported.

Patients and methods

SAMPLE

All the children were registered with one of seven general medical practices in and around the town of Kettering in 1976-8 at the time they "failed" the school eye test. The criteria for referral were an uncorrected visual acuity of 6/18 or worse in either or both eyes with or without a "fail" on a test for muscle balance. These tests were performed on a Keystone machine. The years 1976-8 were chosen because detailed records were not kept before 1976, and the pattern of those referred after 1978 probably altered because children born from January 1974 onwards were screened in infancy and were included in other studies.

Excluding those with puberty onset myopia, a total of 204 children, none of whom had previously been identified as having a possible visual defect, were referred to the children's eye clinic. Eighty four (41%) of these children had normal visual acuity, no squint, and normal refraction. Twelve others were excluded for the following reasons: records could not be traced (five), never reattended for full assessment of vision (two), non-accidental injury in infancy (one), unilateral toxoplasmosis lesion (one), posterior uveitis where vision was not improved by treatment (one), uniocular congenital cataract (one), and Duane's syndrome with normal visual acuity and refraction (one).

The remaining 108 children with defective vision had the following diagnoses: reduced uncorrected acuity but neither a squint nor amblyopia (36), "straight eyed" amblyopia (49), and squint (23).

PROCEDURE

Visual acuity without spectacles and the presence of squint, diagnosed by the cover test, were recorded at the first attendance, and each child's refraction was tested after cycloplegia with cyclopentolate 1%. A child was regarded as having defective vision if the uncorrected acuity of either eye was worse than 6/9, if there was more than one line difference between the acuity of the eyes, or if there was a squint. Appropriate spectacles were ordered, and the acuity was reassessed three months later with a linear Sheridan-Gardiner or Snellen test. Acuity on this occasion was recorded in the same way as the initial acuity. The children were treated on conventional lines with occlusion and surgery when this was advisable and practical. They were followed up for varying intervals according to their age and treatment. The last known acuity of the eye with the worst vision was recorded. Nine children were not treated because it was considered for a variety of reasons that they would be unlikely to benefit.

Results

The initial and last known acuities of the 108 children with defective vision are shown in table I. These are summarised in table II to show the numbers of children whose acuity changed by one, two, or three lines. Improvement of two or more lines occurred in

TABLE I—*Visual acuity in 108 children: initial and last known*

	Last visual acuity						
Initial visual acuity	6/6	6/9	6/12	6/18	6/24	6/36	6/60
6/6	20	1	1				
6/9	4	19	1	1			
6/12	3	4	9	4			
6/18	1	7	6	8	1		
6/24			3	1	3		
6/36		1			2		
6/60		1				1	6

TABLE II—*Change in visual acuity*

Changes	Better	Same	Worse
1 line or more	34	65	9
2 lines or more	16	90	2
3 lines or more	3	105	

No of children with 6/24 or less initially=18.
No of children with last known acuity of 6/24 or less=13.

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TABLE III—*Refraction and initial visual acuity*

	6/6	6/9	6/12	6/18	6/24	6/36	6/60
Abnormal (meridional) hypermetropia	6	7	6	14	5	3	5
Normal hypermetropia:							
Bilateral astigmatism ± anisometropia	4	7	6	2			
Unilateral astigmatism ± anisometropia	7	6	2	5			
Anisometropia (<1·5D astigmatism)	1	4	1				1
Squint, normal refraction	3		1	2	1		1
No squint, normal refraction	1	1	3				
High myopia			1		1		1

only 16 children. Thirteen of 18 children with severe amblyopia (6/24 or worse) were not improved.

The children were separated into three groups according to whether they had squint, straight eyed amblyopia, or neither, and each group was subdivided into those who did and those who did not have occlusion. Analysis of the results expressed in this way showed that those whose initial acuity was poor and those who had squint or straight eyed amblyopia were more likely to have been treated with occlusion, which is what might be expected. Nineteen children with unilateral amblyopia had astigmatism in their better seeing eye, and 19 with hypermetropia also had hypermetropia or astigmatism in the better eye.

Since there is a possibility that the method of screening children's vision in school might change from a test based on abnormal visual acuity to one based on abnormal refraction the refractions of these 108 children are related to their initial acuities in table III. The abnormal refractions listed are those most likely to be identified by a photorefractor or an autorefractor. The results showed that 90% of children with defective vision also had abnormal refraction.

Discussion

Children's acuity varies from one consultation to another irrespective of treatment, but if an improvement of two or more lines represents a real change this was achieved in 16 cases. Over 900 children are born each year in the practices from which this sample was drawn. It can therefore be calculated that the acuity of the worse seeing eye of about 0·6% of these children improved by two or more lines after defective vision was identified at school age by the vision screening programme in school, while 13 of the 18 children who initially had severe amblyopia were not improved at all. Do these results justify the expense of identifying and treating these children? If allowance is made for children's intelligence there seem to be no educational advantages in treating amblyopia,[1] and Taylor has rightly asked whether it really is necessary to do so.[2] Some job opportunities are lost to people with severe amblyopia, but these children are hypermetropic and have probably been so since infancy. In another sample of children treatment from the age of 1 year did not reduce amblyopia[3] so it would seem pointless (in the view of an ophthalmologist) to identify them when they have started school.

Attention to the problem of unilateral amblyopia should not, however, blind us to the plight of the other eye. Nineteen of these children had astigmatism in the better seeing eye, and 19 of those found to have hypermetropia also had appreciable astigmatism or hypermetropia in their better eye. Thus 38 (35%) of these 108 children needed to be identified because they had a refractive problem in the better eye, which almost certainly required correction.

In a recent review a wide range of practices in the vision screening in schools was reported.[4] It is not surprising therefore that two fifths of all the children referred in this district had absolutely nothing wrong with their sight. Of those who had defective vision, 90% had abnormal refraction (table III). The refractions should theoretically be identifiable by either a photorefractor[5,6] or an autorefractor, and these machines should now be assessed to see if they identify those who need treatment more selectively. It would be sensible to identify children with bilateral refractive errors, but we need to debate the need to treat (and therefore to identify) children with lesser degrees of amblyopia.

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ANY QUESTIONS

What can be done about cold sores that recur in the week before the patient's menstrual period? Prophylactic use of acyclovir has not helped.

Menstruation is a recognised trigger for reactivation of labial herpes simplex virus, but recurrence with each menstrual period is fairly rare. When episodic treatment is ineffective, and when the recurrences are sufficiently frequent and troublesome to warrant long term systemic treatment, oral acyclovir 200 mg four times a day may be used as a prophylactic suppressive agent. Though expensive, it is effective in about 75% of patients. There are no other specific antiherpetic agents marketed for use in this manner, but several other drugs would be worth trying.[1,2]

Inosine pranobex 1 g four times a day for seven to 14 days may be used to treat herpes simplex virus infections and, in a patient with a predictable recurrence, might be started in the premenstrual week. Lysine 1000-1500 mg daily has been used for acute or prophylactic treatment of cold sores, as have various antiulcer drugs such as cimetidine. Drugs used throughout the second fortnight of the menstrual cycle in infection with herpes simplex virus triggered by menstruation include chlorpheniramine (4 mg three times a day) and also aspirin. Aspirin was thought to act by preventing the postovulation rise in body temperature, but recent studies have shown that inhibitors of prostaglandin synthesis reduce replication of herpes simplex virus in vitro.

Physical treatments, such as liquid nitrogen cryotherapy, Grenz ray treatment, or superficial epidermal subsection, all have their advocates. The disadvantages are that cryotherapy is not very successful, Grenz rays are neither popular nor readily available now, and the surgical approach (also not readily available) is not effective unless lesions are always in exactly the same site. Finally, on the basis that female sex hormones affect various aspects of immunological function, hormonal manipulation might inhibit the premenstrual reactivation of herpes simplex virus in this patient. Long term treatment with an oral contraceptive or even with danazol would not be warranted here.—NEIL H COX, *senior registrar in dermatology, Newcastle upon Tyne*

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