offer prophylactic preconceptual counselling to all nulligravidas.

We thank John Summerfield and Martin Johnson for constructive criticism of the manuscript, Janet Cooper for keeping track of the patients, and the women who took part, without whose cooperation and enthusiasm the study would not have been possible. The study was supported by a programme grant (G 8302273) from the Medical Research Council to PRB and Dr M H Johnson.

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(Accepted 26 June 1989)

Vision screening at 8 and 18 months

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Abstract

Objective—To determine the effectiveness of an existing screening programme based in the community for ocular and vision defects in infants considered at increased risk of such defects.

Design—Children with ocular or vision defect by the age of 2 were ascertained by searching records. Those from populations at high risk were matched with their results from screening tests. The characteristics of the cases among this population were compared with those of the cases in the remainder of the population. Patterns of referral and age at referral were studied in both groups.

Setting—The study was conducted within Oxfordshire Health District.

Subjects—433 Children at high risk born in 1984 to mothers living in the health district at delivery and who either weighed less than 2000 g or weighed 2000 g and over and required admission to a special care nursery for longer than 24 hours. The low risk population (6254) were infants without these characteristics who were resident in the health district at the time of referral.

Interventions—Screening tests for vision or ocular defects already routinely used were applied by health visitors at 8 and 18 months to the children at high risk.

Main outcome measure—Comparison of results of screening tests with vision and ocular defects detected by the age of 2.

Results—Screening tests in current use for vision loss and squint in this age group were insensitive and had a low positive predictive value when applied to a high risk population. Defects that were not apparent on direct inspection were unlikely to be detected by these tests. In the high risk group the relative risk of having a defect was 2.8 (95% confidence interval 1.8 to 4.5) but 85% of all cases detected by the age of 2 were in children at low risk. Referral patterns and age of referral differed in the two groups.

Conclusions—Screening by health visitors of high risk populations contributes little to the detection of vision and ocular defects. This type of evaluation needs to be applied also to low risk populations, who have different referral patterns and contribute most of the cases.

Introduction

Vision screening of preschool children has become a much debated and contentious issue. Earlier claims of the likely benefits of screening¹ have been challenged by ophthalmologists, paediatricians, and community doctors.^{2,4} The term vision screening covers several

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Br Med J 1989;299:545-9

different tests carried out at a wide range of ages to identify various ocular conditions.⁵ The tests may be applied to either the whole population or a subgroup. The broad aim of a programme of vision screening is to identify children who are likely to have treatable vision or ocular defects that would otherwise remain undetected.⁶

In the first 18 months of life the vision screening tests most commonly used by health visitors, general practitioners, and clinical medical officers are aimed at detecting reduced visual acuity and squint. During the examination structural abnormalities of the eye, periorbital defects, and abnormal visual behaviour may become apparent.

To assess a vision screening programme answers are needed to three main questions. Firstly, Are the tests valid, differentiating between children who do and do not have the condition? To measure this a standard needs to be defined. Secondly, Is the screening programme effective? What is the contribution of a screening programme carried out by health visitors when applied at a particular age and to a particular population to the early detection and management of vision problems and ocular defects in the total population? Thirdly, Is the screening programme efficient? Would the resources needed to support the programme be better spent in other ways? What is the impact of the screening programme on the diagnostic services?

An opportunity arose to study the first two questions when a screening programme was offered to a subpopulation of infants considered at particular risk of vision and ocular defects. This formed part of the Oxford region child development project.⁷ Subsequently, a search was made of all referrals in Oxfordshire Health District for specialist eye care to ascertain the numbers and characteristics of all children presenting with an eye problem by the age of 2.

The relation between the results of the screening test in the high risk subpopulation and the presence or absence of a vision or ocular defect at the age of 2 was then determined. By examining referral patterns and age at referral in this group we assessed the contribution of the screening programme to the detection of defects. Comparison of the type of ocular and vision defects in this group and in the remainder of the population allowed us to assess the likely overall contribution of a vision screening programme of the total population to the detection of these defects by the age of 2.

Subjects and methods

Infants born in 1984 to mothers living in the Oxfordshire Health District who either weighed less than 2000 g or weighed 2000 g or more and were admitted to a special care nursery for longer than 24 hours were enrolled into the screening programme. These infants were considered to be at increased risk of vision and ocular defects⁸ and were designated high risk. Infants were screened at the age of 8 and 18 months (uncorrected for gestational age at birth) by health visitors. Several questions related to vision and ocular function were included (see box). The tests were among those already used by health visitors.

Within the health district infants can be referred for a specialist eye opinion by several routes, including direct referral by general practitioners, paediatricians, and other specialists. Health visitors can refer patients directly to the specialist eye service through general practitioners or through an orthoptic vision screening service.⁹

Information on infants referred from multiple sources for an opinion from an eye specialist by the age

Protocol for vision screening at 8 and 18 months

Questions asked to health visitor

- 1 Do the parents think the baby sees normally?
- 2 Are there immediate family members with poor vision?
- 3 Do the eyes look normal? If no, specify.
- 4 Does the baby look at a small object?*
- 5 Does the baby rake up a small object?
- 6 Are the eye movements normal?
- 7 Are torch reflections in same position in each eye?
- 8 Is the cover test normal?
- 9 Does the baby object to one eye being covered more than the other? (only at 18 months)

*At 8 months small object=raisin; at 18 months small object="hundreds and thousands" cake decorations
Answers: Yes; no; uncertain; not tested

of 2 was obtained by searching records from eye clinics and orthoptic records within the district. None of the information was available on computer. Further data were obtained from the paediatric assessment centre; special needs advisers to the Department of Education and Department of Social Services, who held the "blind register"; the eye operation register; and birth registers. The following information was abstracted from the records: health district of birth; birth weight; admission to special care nursery; age at referral; source of referral; and outcome of referral. Information on outcome was also sought for infants in the screened population who were known to have left the district and had been referred for an eye check to a centre outside the district. Information on referred infants was matched with the results of screening tests by using name and date of birth.

A case was defined as a child with a recognised or suspected vision or ocular abnormality requiring treatment or surveillance. (For classification of cases see table IV.)

Information was also abstracted on the outcome of referral by the age of 2 of infants who were not included in the screening programme (low risk group). These infants were living in the district at the time of referral, and many would have undergone routine screening.

Results

In 1984 there were 6687 live births to mothers living in the district at the time of delivery. Overall, 433 infants were defined as high risk; 35 of these died in the neonatal period and a further six died before the age of 2. Results of vision screening tests were available for 381 infants in the first year of life and 361 infants in the second year. Table I shows the reasons for loss of information. The figure shows the age at which the 8 and 18 month tests were actually given. Table II gives the numbers of infants who failed each test.

The tests were divided into three groups for analysis: non-specific tests (questions 1, 2, and 3 in box); tests

TABLE I—Number of high risk infants in screening programme

	No of infants
Infants eligible for screening	398
Parents refused	6
Died before tests at 8 months	2
Infants available for screening	390
Moved house and not located	8
Health visitor refused	1
8 Month screening results obtained	381
Died between 8 and 18 months	4
Other	16
18 Month screening results obtained	361*

*One infant subsequently died before 2 years.

for squint (questions 6, 7, and 8); and tests for visual acuity (questions 4, 5, and 9). The search of records showed that 31 infants in this high risk screened group were referred for an eye opinion before the age of 2. Twenty one of the 31 were designated as cases (see table IV). All 21 cases were used as the standard for the first triad of tests, 15 cases of squint detected by age 2 for the second triad, and 10 cases of reduced visual acuity for the third triad; these included six children

Distribution of ages at which 8 month and 18 month tests for screening vision were applied

TABLE II—Number (percentage) of high risk infants who failed tests for vision screening at 8 and 18 months

Question*	At 8 months	At 18 months
1	12/377 (3.2)	14/343 (4.0)
2	154/375 (41.1)	133/349 (40.0)
3	25/372 (6.7)	28/346 (8.1)
4	23/379 (6.1)	11/328 (3.4)
5	52/379 (13.7)	9/348 (2.6)
6	19/377 (5.0)	14/346 (4.0)
7	19/362 (5.2)	16/331 (4.8)
8	35/356 (9.8)	43/320 (13.4)
9		25/318 (7.9)

*See box for details of questions.

TABLE III—Characteristics of vision screening tests in high risk infants

Question*	No of infants tested		Sensitivity (%)		Specificity (%)		Positive predictive value		Negative predictive value	
	8 Months	18 Months	8 Months	18 Months	8 Months	18 Months	8 Months	18 Months	8 Months	18 Months
<i>General</i>										
1	377	343	35	58	99	99	58	78	96	98
2	375	349	48	39	60	61	6	5	95	95
3	372	346	55	67	96	95	44	43	97	98
<i>Squint</i>										
6	377	346	50	70	97	98	42	50	98	99
7	362	331	53	87	97	97	42	44	98	99
8	356	320	61	43	92	87	23	7	98	99
<i>Vision</i>										
4	379	328	70	62	96	98	35	45	99	99
5	379	348	70	62	88	99	13	55	99	99
9		318		40		93		8		99

*See box for details of questions.

TABLE IV—Numbers of infants with diagnosed vision and ocular defects at 2 years

	Total infants	High risk infants	Low risk infants	Relative risk	95% Confidence interval of relative risk
Manifest squint (intermittent or constant)	97	14	83	2.7	1.5 to 4.7
Neurological disease affecting visual system	16	9	7	20.5	7.7 to 54.8
Periorbital disease	13		13		
Refractive error (without manifest squint)	13	2	11	2.6	0.6 to 11.8
Nystagmus	11	3	8	6.0	1.6 to 22.5
Intraocular disease	10	1	9	1.8	0.2 to 14.0
Latent squint	9		9		
Total No with vision and ocular defects*	139	21	118	2.8	1.8 to 4.5
Total No alive at 2 years	6634	391	6243		

*Some infants had more than one defect.

with severe reduction of vision in both eyes and four children who were later shown to be amblyopic.

Table III shows the characteristics of these screening tests. Most tests at both 8 and 18 months showed a low sensitivity—that is, their ability to detect the presence of a defect was poor—and a low positive predictive value—that is, failing the test was poorly predictive of a defect. Generally the tests had a high specificity—that is, their ability to exclude a defect was good—and high negative predictive value—that is, passing the test carried a high probability that an eye defect would not become apparent by the age of 2. The exception to this was the question on family history; failure to ask specifically for a history of defects with onset in early childhood probably accounted for this.

Three other points emerged. Of the 15 children who had a squint detected by the age of 2, the 10 who failed the cover test at 8 months also looked abnormal on inspection. Of the five children with squint detected by the age of 2 who looked normal on examination at 8 months, only one failed the cover test. Performance of the cover test by health visitors at 8 months added minimally to the detection of squint over the impression gained on overall inspection. Secondly, among infants with reduced visual acuity detected by the age of 2 the six infants with severe vision loss all looked abnormal on inspection. Of the four with subsequent amblyopia, only one failed the tests applied. Performing these screening tests for reduction of visual acuity (triad 3 on table III) at 8 and 18 months added little to the information gained on overall inspection and observation of visual behaviour. Finally, only five of 21 parents of infants with an eye defect thought that their infant did not see normally at 8 months (question 1 in box). These parents all had infants with severely reduced vision in both eyes, which had already been investigated.

Assuming that population movement in and out of the district was equal during the study,¹⁰ we considered the total number of infants at low risk to be 6254 (total live births (6687) minus high risk screened infants

(433)). Eleven of these died before the age of 2. A total of 221 infants in the low risk population were referred: 118 of these were classified as cases. Table IV shows the numbers and characteristics of the children with eye defects in the high and low risk populations. Although several children were identified as probably amblyopic, diagnosis at the age of 2 is often uncertain. These children were included in other categories—for example, strabismus or refractive error—as appropriate. The risk of an eye defect, particularly manifest squint, and an eye problem associated with neurological disease was considerably increased in the high risk group. This group, however, accounted for only 15% of all the cases in the total population.

TABLE V—Age at first attendance for specialist eye opinion of infants at low and high risk of developing ocular and vision defects

Age (months)	Low risk infants	High risk infants
<3	4	0
3-	13	4
6-	15	8
9-	27	4
12-	16	0
15-	7	0
18-	13	3
21-	13	0
≥24	10	2*

*Includes one child for whom data not known.

Over half the screened infants (11/21) in the high risk group who were designated as cases were already attending an eye department by 8 months (the recommended age for screening under the child development project and routine screening in this health district) compared with 27% (32/118) of cases in the low risk group (table V). There was a marginal increase in the rate of referral among the low risk population aged 9-11 months, possibly attributable to screening. The extent and outcome of screening were not known for this low risk group, but it was part of the routine child health surveillance for all infants at the time of the study.

Among the 252 children referred, 202 referrals originated from the primary health care team.

TABLE VI—Source of referral to specialist eye service for infants at low and high risk of developing ocular and vision defects

Source	Low risk infant	High risk infant
Paediatrician	9	11
General practitioner	52	6
Health visitor plus orthoptic screening	48	2
Others	4	2
Not known	5	

Of these, 130 were direct referrals from a general practitioner, of which 58 proved to be cases. The remaining 72 were among the 763 children who were referred by the health visitor to an orthoptic secondary screening service and thence to the specialist eye service; 50 of these proved to be cases. Although most cases overall (107/139) originated from the primary health care team (table VI), most of the high risk cases had been referred directly from paediatric or other specialist clinics.

Discussion

Several factors may have contributed to the insensitivity of routinely used screening tests in our high risk population. We used eye defects detected by the age of 2 as the standard, and, as eye conditions change over time, the time lag between screening and ascertaining cases may have led to an underestimate of sensitivity. Also the age at testing may have been inappropriate, the state of maturity may have contributed, and the tests may have been incorrectly applied. Probably, however, much of the problem lay with the tests themselves, which were unable to detect conditions that were not already apparent on direct observation.

SCREENING FOR LOW VISION

With the screening tests that are routinely available accurate assessment of visual acuity in each eye before the age of 2 is not possible. Though techniques of forced preferential looking can be used for infants and children under the age of 2,¹¹ these are not yet considered suitable for screening by health visitors. Therefore screening tests for vision used at this age will probably detect only bilateral loss of vision that is sufficiently severe to alter behaviour patterns or to be associated with overt signs such as wandering eye movements, avoidance of gaze, and nystagmus.

Amblyopia is unlikely to be detected in children under the age of 2 unless associated with a manifest squint¹² or other ocular abnormality such as ptosis. When amblyopia is associated with a refractive error it may be detected with retinoscopy¹³ or photo-refraction.¹⁴ Neither of these techniques is currently used routinely within the community. The issue is further complicated by changes in refractive error, particularly in infancy, and disagreement over the

possible benefits of early correction of refractive errors in preventing amblyopia and squint.^{15 16}

SCREENING FOR SQUINT

All of the tests for squint at 8 months had a low sensitivity, and the cover test was the least specific. Thirty five (10%) 8 month old infants and 43 (14%) 18 month old infants failed the cover test. As this technique seems useful in experienced hands for detecting both latent and manifest squint¹⁷ and eliminating pseudosquint, possibly further training of health visitors in giving the test will improve its performance. After seeing a training videotape, many health visitors said that they had never previously seen an abnormal cover test. Many squints, particularly accommodative squints, do not become apparent until well after the second birthday, and the ability of the commonly used screening tests, applied before the age of 2, to detect these late onset squints is uncertain. Overall, unless a squint was apparent on inspection it was unlikely to be detected by a cover test applied by the health visitor on this young population.⁴

EFFECTIVENESS OF SCREENING PROGRAMME

The high risk population contributed only 15% of all infants with defects identified by the age of 2. The children tended to be referred early to diagnostic eye services by paediatric and other specialist services, reflecting the current practice of close surveillance in hospital clinics of infants who have caused concern in the perinatal period. From a community perspective infants with eye defects who are most likely to be missed are those within the large group with a low prevalence of defect but who contributed 85% of the cases. Whatever the quality or choice of test or tester a screening programme confined to a high risk population will miss most cases in the total population.

After completing the formal screening procedure health visitors are asked, "Do you think everything is all right with this child?" The answer to this question was assumed to be based not only on the test but on previous knowledge of the infant's development and wellbeing, on observations at home, and on discussion with parents. Although the response to this question taps concerns in many aspects of development and hence is rather non-specific, it proved more sensitive in detecting impairments that included vision and ocular problems at 8 months than any of the tests for vision screening evaluated individually in this population.

Health visitors acquire an experience of the development and abilities of normal children that is unique in the primary health care team. Although individual screening tests may prove inaccurate on formal evaluation, this ability to identify correctly a high proportion of cases should not be ignored.

THE FUTURE OF VISION SCREENING

In the much needed critical appraisal of formal screening procedures and tests that are currently underway¹⁸ it is tempting to discard tests that individually are insensitive and non-specific. In doing so it would seem important not to discard the framework within which the experienced primary health care worker can regularly and systematically observe and evaluate an infant. At present there is no system for routinely monitoring the effectiveness of current preschool screening practices in the community. Results of screening tests are not kept centrally and searches for cases are usually manual, time consuming, and expensive.

We suggest that as new routine information systems are computerised evaluation of screening programmes in current use and new screening tests will be essential. Finally, one of the requirements for a screening programme is that treatment or management should be

available for the conditions being sought, resulting in overall benefit for the patient. While we accept that changes in the practices for preschool vision screening are obviously needed, the "knock on" effect of such changes on diagnostic services in terms of workload and cost will need to be taken into account. More importantly, the effects of early treatment of low vision, squint, refractive error, and amblyopia in very young children need to be clarified.

We thank the Oxfordshire health visitors who performed the screening tests and Ms Hazel Ashurst for help with analysis of screening data. The Oxford region child development project was funded jointly by the Department of Health and Social Security and Oxford Regional Health Authority. Maureen Stayte was supported by a grant from Oxfordshire Health Authority (locally organised research grant).

The steering committee of Oxford region child development project comprises Mrs J Catterson (chairman), Oxford Regional Health Authority; Dr M Goldacre, Oxford record linkage study; Miss R King, administrative coordinator; Ms A J Macfarlane, national perinatal epidemiology unit; Dr J A Macfarlane, department of community medicine; Professor Sir Alexander C Turnbull, department of obstetrics and gynaecology; Dr A R Wilkinson, department of paediatrics.

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(Accepted 14 June 1989)

Death rates and causes of death in 284 consecutive patients with giant cell arteritis confirmed by biopsy

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Br Med J 1989;299:549-50

Although giant cell arteritis affects the arteries,¹ the statistical survival rate in several studies has been shown to be the same as that of the general population.^{2,3} These series have been comparatively small, however, and possibly not large enough to detect minor differences in survival rates.

Patients, methods, and results

All temporal artery biopsies performed in Göteborg over 10 years (1 January 1977-31 December 1986) were identified, and 284 subjects were diagnosed as having histologically verified giant cell arteritis. A follow up investigation of all patients was performed in December 1987. The causes of death were obtained from death certificates and the National Bureau of Statistics. The calculations of the expected number of deaths and comparison with the observed number have been described.²

By December 1987, 82 of the 284 patients had died (64 women and 18 men). The expected number of deaths in an age and sex matched control group was 68.16, and the difference was not significant. The mean age at death was 79.5 years (59-90 years), and the mean time from diagnosis to death was 3.3 years (<0.5-108 months).

The observed numbers of deaths from vascular and malignant disorders at follow up were 62 and 9 respectively, compared with the expected numbers of 43 ($p<0.05$) and 12.9 (NS) (figure). One year after diagnosis the observed number of deaths from vascular disorder was 21 and the expected number 7.01 ($p<0.001$). Seventeen of these 21 subjects died within the first four months, eight of cerebrovascular disease,

three from myocardial infarction, three from cardiac failure, two from rupture of a dissecting aneurysm of the aorta, and one from pulmonary embolism. From the second year after diagnosis to follow up, there was no significant difference between the observed and the expected number of deaths from vascular disorders.

Three of the 17 patients displayed symptoms typical of polymyalgia rheumatica, seven had only cranial symptoms (temporal arteritis), two suffered from both polymyalgia rheumatica and temporal arteritis, and five had only systemic symptoms. Their mean age at death was 76.1 years (61-89 years) and, in comparison with the total numbers of deaths, there was no sex difference.

All 17 patients had been treated with corticosteroids. In 13 of them, however, treatment had been insufficient to suppress disease activity as judged from clinical symptoms and the return of the erythrocyte sedimentation rate to normal. The cause was either too low a dose of corticosteroids, premature discontinuation of treatment, or simply too short a time (arbitrarily defined as less than one month) to suppress disease activity before death occurred.

Necropsy was performed in seven of the 17 patients.

Number of deaths from vascular disorders in patients with giant cell arteritis proved by biopsy compared with an age and sex matched control population