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Neonatal at Risk Factors, Visual Defects and the Preschool Child: A Report from the Queen Mary Hospital Multidisciplinary Child Development Study

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SUMMARY

An experimental group of 142 children aged four years who had experienced neonatal at risk factors and a control group of 112 children whose perinatal histories were optimal had vision screening tests to detect defective vision or strabismus. Five (3.5 percent) in the at risk group and 10 (8.9 percent) in the control group (total 15, 5.9 percent) were found to have a visual defect. Of those, six had already been identified because of a manifest squint. Nine children with defective vision were first identified through the study.

The importance of the early identification and treatment of visual disorders, particularly amblyopia, is emphasised, and recommendations are made for more widespread vision screening of preschool children.

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INTRODUCTION

It has been reported in the United Kingdom (Chief Medical Officer, Department of Education and Science, 1966) that with the exception of dental disease, defective vision is the most prevalent defect of child development. The report further stated that in the United Kingdom failure to make satisfactory progress at school is frequently due to an undetected visual defect.

The prevalence of visual defects reported in the literature varies according to the definitions of defect used, the screening and assessment techniques used, and the age and location of the sample. There have been no published figures on the prevalence of visual defects in New Zealand preschool children. The New Zealand Health Department (Otago Office) has made available recent figures on the incidence of confirmed visual defects found among 12 522 Otago primary school children. The percentage of confirmed defects in this sample, assessed during 1971 and part of 1972, was 2.94 percent (Morganty, Personal Communication, 1972).

It is well known that the earlier visual defects are recognised and treated the more favorable the prognosis. This is particularly so in the treatment of squint and amblyopia by spectacles, occlusion and orthoptic therapy (WHO, 1967).

While the causes of visual problems are complex, a World Health Organization working group suggested that there are four outstanding factors which place a child at risk of developing a defect. These include a family history of visual defect, virus infection during pregnancy, prematurity and difficult or assisted labour. They suggested that the early screening of those youngsters at risk would identify many who later develop a visual defect (WHO, 1967).

There is general agreement that a very low birth weight premature infant is more likely than a full term infant to develop a visual defect (McDonald, 1962; Lubchenco, 1963; Dann, Levine, New, 1964; Walsh, 1969). Earlier data on these very small premature infants, however, included many cases of retrolental fibroplasia, a condition caused by excessive oxygen therapy until its effects were recognised about 1953 (Harper, Wiener, 1965). Myopia of prematurity is regarded as an abortive form of retrolental fibroplasia (Duke-Elder, 1970). More recent studies suggest that the prognosis for the very small premature infant may be improving. A comparison of Drillien's 1958, 1964 and 1967 figures show a drop from 50 percent to 37 percent to a 14 percent incidence of visual defects. This may well reflect improvements in neonatal care.

The literature on the visual defects of larger premature infants is less clear. Gastren (1955) found a group of large ex-prematures to have significantly more amblyopia and strabismus than a control group, although Caplan and others (1963) found no differences between their premature sample and a control group.

Apart from the well known effect of maternal rubella on the developing fetus, Gardner, James (1960) in an important retrospective study, have implicated maternal diseases, particularly the toxaemias, as a possible cause of myopia. Toxaemia, also, can result in premature labour (Townsend, 1964).

Other neonatal problems have been suggested as possible causes of defective vision and strabismus. Hunter (1968) found a high incidence of squints in infants who had been hypoxic and hypoglycaemic, yet Fraser, Wilks (1959) found no differences in vision and squints between their hypoxic and non-hypoxic sample. McKinna (1966) from her series noted an association between hypoglycaemia and ocular defects, and argued that hypoglycaemia damages the nervous system and therefore the ocular system. Griffiths, Bryant (1971) in a more carefully controlled study, on the other hand, found no association between neonatal hypoglycaemia and ocular defects.

One of the major problems in studying outcomes of adverse perinatal or neonatal events is that of multiple factors interacting in ways that are not yet fully understood. This can be illustrated by Koch's (1964) study where he found that 28 percent of his series of mainly very small premature children with hyperbilirubinaemia had visual acuity of less than $^{6}/_{18}$. In this case it was not clear whether the prematurity, the hyperbilirubinaemia or some other prior factor was responsible. It does seem clear, however, that some of the antecedents of brain damage and brain dysfunction are also antecedents of ocular defects. This relationship

is discussed by Fantl, Perlstein (1967) referring to their study of a large series of cerebral palsied cases. They concluded that ocular abnormalities of the cerebral palsied child "appear to be caused by brain damage occurring before or around the time of birth" (page 863). Recent evidence of a relationship between squints and general physical co-ordination (sometimes considered a component of minimal brain dysfunction, Clements (1966)) has emerged from the British Child Development Study (Davie and others, 1972) further suggesting a link between brain dysfunction and ocular defects.

In order to reduce the developmental hazards associated with adverse perinatal and neonatal events, a continuous assessment of the survivors is necessary. The present study is part of an extension of a research project described by Buckfield (1972) which is a multidisciplinary longitudinal study of the total development of children who experienced potentially hazardous neonatal events, compared with a control group of children whose perinatal histories were normal.

This paper examines the prevalence of visual defects in a group of infants who were at risk in the neonatal period because they were premature, of low birth weight, or because they experienced birth hypoxia and other neonatal illnesses. This group is compared with a control group of full-term infants whose perinatal histories were optimal. The paper also presents information on the incidence of visual defects in a sample of preschool children and makes suggestions for improved early vision screening procedures.

THE SAMPLE

The sample is drawn from all children born at or admitted to Queen Mary Hospital (QM) in 1968. The great majority of deliveries in the Dunedin metropolitan area are conducted at QM. In 1968 there were 2013 live births at QM and 19 babies were transferred from outlying areas for intensive care, the majority being of low birth weight. Of the total live born survivors at QM, 72 were premature (gestational age less than 37 weeks) and 41 were considered small for gestational age (SGA defined as a birth weight below the tenth percentile for gestational age based on 6151 consecutive births at QM). There were 13 infants of 2500g or less whose gestational age was uncertain. A further 36 infants were considered hypoxic at birth because they scored less than two on the Apgar score for heart rate, respiration and colour at five minutes after birth. Finally, there was a group of 20 infants who experienced none of the complications above, but who developed either the respiratory distress syndrome (RDS) or hyperbilirubinaemia (15mg/100ml or more). These 182 surviving infants were considered at risk in the neonatal period.

The experimental or at risk group was drawn from all those either born at or admitted to QM during 1968 who experienced any of the above neonatal at risk factors. A control group was randomly selected from all children born during the same year who were full term and of appropriate weight for gestational age. Children with any known maternal or perinatal abnormalities were excluded from the control group. Any child with a major congenital fault was excluded from both experimental and control groups.

27.7 percent of the sample were not assessed because they were untraced (e.g., some adopted children) or because they were known to have moved beyond Otago. There was no significant difference between the percentage of children lost to the experimental group and the control group. The parents of all the children who were traced co-operated in the study.

Table one shows the numbers in the various experimental groups who were actually assessed. Within the premature sample only four children were of less than 1501g of birth weight. Thirteen were between 1501 and 2000g, 16 were between 2001 and 2500g and 25 were greater than 2500g in birth weight.

Table 1.—Subdivisions of the sample

Group	N
Premature	58
Small for gestational age	36
Low birth weight (? gestational age)	11
Hypoxic	36
RDS or hyperbilirubinaemia	11
Total at risk group	142
Control group	112
Total sample	254

METHODS

All children were examined shortly after birth or admission to QM and careful records were kept of all relevant aspects of every infant's perinatal and neonatal history. Gestational age was calculated from the first day of the last menstrual period and if any doubt was felt about the accuracy then this was classified as "uncertain".

The children selected for the study were then examined within one month of their fourth birthdays. The investigators worked blind not knowing the perinatal histories of the children at the time of examination.

An attempt was made to screen the vision of all the children at age four years. Screening assessment was based on the illiterate E test and the cover test (Parr, 1962). The E test was performed at 4m using a reduced Snellen type of chart. An acuity of 6/6 was regarded as normal for four year olds (Parr, 1962) and any child whose uncorrected visual acuity was suspected to be 6/12 or less in one or both eyes was referred to the ophthalmology department for full assessment.

Also referred to the ophthalmology department were all children with a suspected squint, either manifest, or where a cover test (Parr, 1962) suggested a latent squint. Further, except in the case of one youngster (with severe mental retardation and cerebral palsy) all children who did not respond satisfactorily to the screening tests were referred as well. False positive referral rates were unusually high, because any doubts or mild suspicions were acted on with a referral to the ophthalmology department for detailed examination. It was considered more desirable to over refer than under refer.

At the ophthalmology department the orthoptist reassessed the visual acuities using the E test at 6m, or picture cards (Allen, 1957) in some cases, carried out the cover test and the four diopter prism test. Children whose vision was not clearly normal and had a squint had an atropine refraction and their fundi examined by one of the medical staff. Where treatment was indicated it was carried out and where doubts existed the children were followed up at intervals until a confident assessment could be made.

RESULTS

Fifteen children (5.9 percent of the total) were found to have a visual defect. Of these, five were in the experimental or at risk group (3.5 percent) and 10 in the control group (8.9 percent). Thus, there was no evidence from this study to suggest that the neonatal at risk factors place a child at greater risk of developing a visual defect. On the contrary, in this sample there were more than twice as many defects among the control group than among the at risk group.

Of some interest was the finding that six out of 15 had visual defects that had been recognised prior to this study and all had manifest squints. Two other children, not under treatment, had intermittent left convergent squints with mild hypermetropia. Including these two cases, the study resulted in nine previous undetected defects being identified. Three out of the seven children without squints had amblyopia.

DISCUSSION

This study found more than twice as many children with visual defects in the control group with optimal perinatal histories as in the experimental group who had experienced neonatal at risk factors.

Six children already under treatment before this study commenced all had strabismus and their problem was immediately obvious. It is well accepted that children with a unilateral squint nearly always have amblyopia of some degree because the vision in the deviated eye had been suppressed to prevent diplopia. What is not well known, however, is that children can have amblyopia without a squint. Three of the nine children with visual defects first identified by this study had amblyopia without a squint. This type of amblyopia is caused by a unilateral refractive error, or markedly differing refractive errors (anisometropic amblyopia). This results in the absence of a clear image on the retina of one eye and the visual process in this eye fails to mature. If not corrected by about seven years of age by spectacles and/or occlusive therapy, permanent poor vision in the affected eye in adult life is the rule. It is well known that the earlier the treatment of amblyopia is commenced the greater the chance of success (Parr, 1962). Amblyopia, squints and other visual defects do not vanish spontaneously, nor are they best left until the child is of school age. The need for early recognition and treatment of most visual problems is widely recognised by ophthalmologists.

The findings from this study suggest that the majority of those with visual defects are unlikely to be detected before the Health Department's primary school screening. It is desirable that children with visual defects can be identified before school entry so that treatment can be instituted earlier with more effective results.

The writers consider it advisable to refer any preschool child to an ophthalmologist where there is a suspicion of a visual defect. Suspicion of a defect can be strengthened particularly where there is a family history of myopia or strabismua (WHO, 1967). Ophthalmologists would accept an increased number of false positive referrals if this means that a greater number of young children could be identified and treated earlier than is the case at the present time.

In New Zealand we have a number of agencies and personnel actively involved in visual screening of preschool children. The Plunket Society nurses test the visual acuity of children under their care and some general medical practitioners, as a matter of routine, screen the vision of young children. Also, the Health Department has recently instituted vision screening of children at kindergartens and play centres. However, many children do not benefit from this early screening and are not identified as having visual defects until after school entry.

The writers believe that all children should have their vision screened during their fourth year. Experience from this study has shown that most four-year-olds can respond with practice to the illiterate E test and others can respond successfully to the picture cards. It is recommended that vision screening be made routine at the time the booster immunisation injections are given, prior to school entry. This should be carried out by the general medical practitioner or by a trained assistant. Further, it is recommended that trained personnel, such as kindergarten teachers, play centre supervisors and infant teachers in primary schools, be given the opportunity to learn the skills of administering simple vision screening tests. The tests are not time consuming, are enjoyable for children, and in some cases may well result in a child avoiding a later persisting serious visual problem.

The treatment of a visual defect in young children may result in a dramatic improvement in awareness of the world. One mother wrote commenting on the rapid development her child made after spectacles were prescribed for myopia. This particular child gained 30 IQ points between psychological testing at four years of age before treatment (Stanford Binet intelligence scale) and at five years of age after treatment (Wechler preschool and primary scale of intelligence, full scale IQ). It is intended to reassess the sample at age seven years and the developmental progress of those whose visual defects were treated will be studied. This future study should provide further information on the long-term significance for development of the early recognition and treatment of a visual defect.

CONCLUSIONS

This study found that preschool children who experience neonatal at risk factors are no more likely than other children to develop a visual defect. Overall, an incidence of 5.9 percent with a visual defect were identified. It is recommended that more widespread vision screening of preschool children be carried out.

Further study of the long-term significance of early recognition and treatment of visual defects in preschool children is proceeding.

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