Association between visual impairment and functional and morphological cerebral abnormalities in full-term children

Sonia Grönqvist[1], Olof Flodmark[2], Kristina Tornqvist[3], Göran Edlund[4] and Ann Hellström[5]

Department of Ophthalmology, Central Hospital[1], Karlstad, Department of Neuroradiology, Karolinska Institute, Karolinska University Hospital[2], Stockholm, Department of Ophthalmology, University Hospital[3], Lund, Department of Radiology, Central Hospital[4], Karlstad, Department of Clinical Neuroscience, Section of Ophthalmology, Department of Paediatrics, International Paediatric Growth Research Centre, The Queen Silvia Children's Hospital[5], Göteborg University, Gothenburg, Sweden

ABSTRACT.

Purpose: **To characterise the nature and degree of ocular disorders and cerebral morphological and functional abnormalities in a population-based group of visually impaired full-term pre-school children.**

Methods: **Forty-five children who were born at full-term between 1989 and 1995 in Värmland, Sweden, were reported as being visually impaired. An ophthalmological examination was performed and clinical data regarding mental development and neurological disease were obtained for all children. Cerebral imaging was performed in 35 children.**

Results: **Twenty-six per cent of the children were found to have ocular disorders only. Forty-two per cent had cerebral morphological abnormalities, verified by cerebral imaging, and 65% had signs of cerebral functional abnormalities. In total, 74% were found to have cerebral morphological and/or cerebral functional abnormalities.**

Conclusion: **The majority of children with visual impairment, including children with ocular disorders, were found to have cerebral morphological and/or cerebral functional abnormalities. We suggest that any child with visual impairment should therefore undergo cerebral imaging and be examined by a paediatrician in order to establish the correct diagnosis.**

Key words: cerebral lesions – full-term children – ocular disorders – visual impairment.

Acta Ophthalmol. Scand. 2001: 79: 140–146

The causes of visual impairment in children have changed over recent years. In the past, ocular disease was the predominant cause of reduced vision (Lindstedt 1972), however, as medical, ophthalmological and surgical treatment have improved, there has been a decrease in the proportion of children with this type of visual impairment (Blohmé & Tornqvist 1997). Improved general medical care has increased the number of very pre-term and severely sick children who survive, resulting in a greater proportion of children who have visual impairment due to brain damage (Rosenberg et al. 1996; Blohmé & Tornqvist 1997).

Several recent neuroradiological studies have demonstrated that periventricular leukomalacia (PVL) is the most common cause of visual dysfunction in children born pre-term (Eken 1995; Jacobson et al. 1998). However, to our knowledge, the types and extent of cerebral lesions that are associated with visual impairment in full-term children have yet to be described.

The purpose of this study was to characterise the nature and degree of ocular disorders, cerebral morphological and functional abnormalities in visually impaired full-term children.

Definitions

Visual impairment (VI) was defined as an optotype acuity ≤0.3 with the best correction, or a visual acuity above 0.3 in combination with visual field restriction. In cases in which the child was unable to co-operate (due to young age and/or mental incapacity) a subnormal grating acuity or persistently abnormal fixation behaviour were considered as visual impairment.

Disorders of the eyes and anterogeniculate visual pathways are referred to as "ocular disorders" in the text.

Patients

The county of Värmland in Midwest Sweden has 282,000 inhabitants (Befolkningsstatistik 1994). During the period January 1, 1989 to December 31, 1995,

25,000 children were born in the county. Ninety-nine per cent of all children in Sweden attend routine visual screening at local Child Care Units (Kvarnström et al. 1998). The ability to fixate was evaluated at infancy in all children, and optotype acuity was assessed for each eye at 4 years of age. Infants with deviant fixation behaviour and/or subnormal acuity were referred to the regional ophthalmology service for further assessment.

Among the children born between 1989 and 1995 in Värmland, a total of 66 were found to be visually impaired and reported by the regional ophthalmologists between February 1991 and April 1998. Eighteen of these children, who have been described in a previous paper (Jacobson et al. 1998), were born preterm. Subsequently, three more pre-term children were identified. One had a cerebral malformation, the second had PVL and cerebral atrophy confirmed by neuroradiology, and the third had no ocular disorder but strabismus and mental retardation associated with cerebral palsy, a clinical picture resembling PVL. The parents of this child, however, did not agree to cerebral imaging.

Gestational age was estimated by fetal ultrasonography, performed at week 17 of gestation (post-menstruation). Forty-five children had gestational age at birth ≥37 weeks. Thus, 45 full-term children (26 boys and 19 girls), median age 2 years, (range 2 months to 7 years and 2 months), with visual impairment were included in the present study.

Methods

Visual function and ocular examination
Visual acuity was assessed by methods suitable for the age and ability of the child. Sixteen children were identified as visually impaired because of low linear optotype acuity, nine because of subnormal grating acuity and twenty because of abnormal fixation behaviour. Visual fields were assessed by the confrontation method.

Inspection of the anterior segment and fundus examination with binocular ophthalmoscopy were performed in all children. Refraction was carried out and appropriate glasses prescribed if needed. Thirty-seven out of the 45 children were examined by one of the authors (SG). Information on the remaining seven children was obtained from thorough file investigations and discussions with the treating ophthalmologist.

Cerebral imaging (n=35)
Cerebral imaging was performed in all children, except ten who had obvious ocular reasons for visual impairment (Table 1; patients 1–3, 6–8, 10, 12, 23 and 25). In those children who had been submitted to repeated imaging, all examinations were reviewed but the findings from the latest available investigation were used. The brain was imaged using computed tomography (CT) in eight children and by magnetic resonance imaging (MRI) in 27 children.

Clinical data
The attending paediatrician or paediatric neurologist of each child completed a data sheet requesting information on mental development, cerebral palsy, shunt-treated hydrocephalus and epilepsy.

Results

Table 1 and Figure 1 summarise the results of this study.

In the following, the material will be described along three lines: 1) ocular manifestations, 2) mental development and 3) neuroradiological findings (Fig. 1).

Children with ocular disorders (n=30)
Thirty (66%), 14 boys and 16 girls, of the 45 children had an ocular disorder (patients 1–30).

Children with ocular disorders and normal intellectual development (n=13)
Thirteen of these children (patients 1–13) had normal intellectual development. Eleven (patients 1–11) were considered healthy and had no other diagnoses. Neuroradiology was performed in four of the children and was found to be normal. In addition, one child had severe myopia, abnormal optic discs and craniostenosis but showed otherwise normal development (patient 12). Thus 12 children had an ocular disorder as the main reason for their visual impairment.One girl with optic nerve hypoplasia (patient 13) had congenital ataxia; severe visual perceptual disturbances and neuroradiologically demonstrated cerebellar vermis hypoplasia, but otherwise normal cerebral morphology. Her intellectual development was, however, normal.

Children with ocular disorders and impaired mental development (n=17)
The remaining 17 (56%) of the 30 children with ocular disorders (patients 14–30) had impaired mental development. Five of these children (patients 14–16, 18 and 19) had additional cerebral palsy where neuroradiology revealed abnormal cerebral morphology. One child (patient 21) had epilepsy, but normal cerebral morphology. One boy (patient 17) with optic nerve hypoplasia and impaired mental development, but without additional neurological symptoms, also had radiological findings of PVL.

Eight further children in this group had additional diagnoses. One boy (patient 20) had neurofibromatosis type I with cerebral morphological abnormalities. One girl (patient 22) was diagnosed as having homocysteinuria and retinitis pigmentosa verified by analysis of the blood and by ERG, respectively, but with no pathological findings on cerebral imaging. Six of the children had a chromosomal aberration, five with Down syndrome (patients 24–26, 29 and 30) and one with trisomy 13 (patient 23). Neuroradiology was performed in four of these patients and was found to be normal.

The remaining two children (patients 27 and 28) with impaired mental development, had ocular fundus colobomata, but no other diagnoses and normal neuroradiological findings.

Children without ocular disorders (n=15)
Fifteen (33%), 12 boys and 3 girls, of the 45 children had no ocular disorders (patients 31–45).

Children without ocular disorders with normal intellectual development (n=3)
Three children without ocular disorders had normal intellectual development. Two boys (patients 31 and 32) had PVL, diagnosed by neuroradiology. The third child (patient 33) had attention deficit disorder, and neuroradiology revealed bilateral occipital atrophy.

Children without ocular disorders with impaired mental development (n=12)
Twelve (80%) of the 15 children without ocular disorders (patients 34–45) had impaired mental development. Two of them (patients 34 and 35) had PVL, but no additional neurological symptoms or other diagnoses. Five children (patients 36–39 and 43) had additional neurological

Table 1. Visual function, ocular disorders, mental development, neurological symptoms and neuroimaging findings in 45 fullterm children with visual impairment.

Patient no.	Birth year	Sex	Binocular visual acuity at presentation	Visual field defect	Ocular disorders	Nystagmus	Mental disability	Neurological symptoms	Other diagnose	Neuro-imaging modality	Cerebral imaging findings
Children with ocular disorder and normal mental development (n=13)											
1	1990	F	0.2	−	ocular albinism	+	−	−	−	−	−
2	1990	M	1.8 CPD	+	retinitis pigmentosa	+	−	−	−	−	−
3	1991	F	no fixation	−	ocular albinism	+	−	−	−	−	−
4	1991	M	no fixation	not performed	ocular albinism	+	−	−	−	CT	normal
5	1992	F	no fixation	+	optic nerve hypoplasia	+	−	−	−	CT	normal
6	1992	F	0.2	−	Peters' anomaly	−	−	−	−	−	−
7	1994	M	no fixation	not performed	congenital nyctalopia	−	−	−	−	−	−
8	1994	M	no fixation	−	aphakia	−	−	−	−	−	−
9	1995	F	9.8 CPD	−	fundus coloboma	−	−	−	−	MRI	normal
10	1995	F	no fixation	not performed	vitreoretinal dysplasia	rowing eye	−	−	−	−	−
11	1995	M	no fixation	−	essential congenital nystagmus	+	−	−	−	MRI	normal
12	1991	F	0.3	−	optic disc anomaly	−	−	−	craniostenosis	−	−
13	1993	F	0.15	−	optic nerve hypoplasia	−	−	congenital ataxia	−	MRI	cerebellar vermis hypoplasia
Children with ocular disorder and impaired mental development (n=17)											
14	1991	M	no fixation	not performed	optic nerve hypoplasia	rowing eye	+	cerebral palsy	microcephaly,deletion chromosome 9	MRI	generalized cerebral atrophy
15	1994	F	no fixation	not performed	optic nerve atrophy	rowing eye	+	cerebral palsy, epilepsy	−	MRI	severe generalized cerebral atrophy
16	1994	M	no fixation	not performed	optic nerve atrophy	−	+	cerebral palsy, epilepsy	−	MRI	severe generalized cerebral atrophy
17	1994	M	0.4	+	optic nerve hypoplasia	−	+	−	−	MRI	moderate posterior periventricular leukomalacia R=L
18	1994	M	no fixation	not performed	optic nerve atrophy	rowing eye	+	cerebral palsy	shunted hydrocephalus	MRI	severe bilateral occipital atrophy
19	1993	M	0.03	−	optic nerve hypoplasia	−	+	cerebral palsy	trisomy 4	MRI	hypoplasia of corpus callosum
20	1990	M	0.25	−	optic nerve atrophy	−	+	−	Neurofibromatosis 1	MRI	optic nerve glioma
21	1992	M	no fixation	not performed	essential congenital nystagmus	+	+	epilepsy	−	MRI	normal
22	1990	F	0.04	−	retinitis pigmentosa pseudophakia	+	+	−	homocysteinuri	MRI	normal
23	1991	M	no fixation	not performed	bilateral microphthalmus sclerocornea	−	+	−	trisomy 13	CT	normal
24	1990	F	0.97 CPD	+	optic nerve hypoplasia	+	+	−	Down syndrome	CT	normal
25	1994	F	0.01	−	essential congenital nystagmus	+	+	−	Down syndrome	MRI	normal
26	1990	F	no fixation	−	essential congenital nystagmus	+	+	−	Down syndrome	CT	normal
27	1994	F	4.9 CPD	−	right fundus coloboma left microphthalmus	−	+	−	−	MRI	normal
28	1991	F	no fixation	+	bilateral funduscoloboma	+	+	−	−	MRI	normal
29	1990	F	no fixation	−	aphakia	+	+	−	Down syndrome	−	−
30	1990	M	0.32	−	macular anomaly	−	+	−	Down syndrome	−	−

Table 1 (contd).

Patient no.	Birth year	Sex	Binocular visual acuity at presentation	Visual field defect	Ocular disorders	Nystagmus	Mental disability	Neurological symptoms	Other diagnose	Neuro-imaging modality	Cerebral imaging findings
Children without ocular disorder and normal mental development (n=3)											
31	1992	M	0.25	–	–	–	–	–	shunted hydrocephalus	CT	severe posterior periventricular leukomalacia L>R
32	1995	M	0.25	+	–	+	–	–	–	MRI	mild posterior periventricular leukomalacia R=L
33	1992	M	0.25	–	–	+	–	–	attention deficit disorder	MRI	bilateral occipital atrophy
Children without ocular disorder with impaired mental development (n=12)											
34	1992	M	0.25	–	–	–	+	–	–	MRI	moderate posterior periventricular leukomalacia R=L
35	1995	M	7.4 CPD	–	–	–	+	–	–	MRI	moderat posterior periventricular leukomalacia R=L
36	1989	M	no fixation	+	–	+	+	cerebral palsy	–	MRI	severe periventricular leukomalacia L>R
37	1989	F	no fixation	+	–	–	+	epilepsy	–	MRI	left congenital occipital lesion
38	1995	M	4.9 CPD	+	–	+	+	cerebral palsy, epilepsy	microcephaly	MRI	bilateral parietal and occipital atrophy L>R
39	1992	M	0.97 CPD	not performed	–	+	+	epilepsy	microcephaly	MRI	pathological myelination
40	1991	F	0.23 CPD	–	–	–	+	–	trisomy 16,microcephaly	CT	moderat cerebral artophy
41	1989	M	no fixation	–	–	–	+	–	multiple extracerebral malformations	CT	agenesis of corpus callosum
42	1990	M	1.8 CPD	+	–	–	+	–	XYY Syndrome	MRI	normal
43	1992	F	no fixation	not performed	–	+	+	epilepsy	trisomy 16	MRI	normal
44	1989	M	0.04	not performed	–	–	+	–	Down syndrome	MRI	normal
45	1992	M	0.25	not performed	–	–	+	–	–	MRI	normal

Fig. 1. Venn diagram of 45 full-term visually impaired children, with regard to impaired mental development (n=29), abnormal neuroradiological findings (n=19) and ocular disorders (n=30).

symptoms; four had epilepsy (one of whom also had cerebral palsy), and one had cerebral palsy only. Neuroradiology revealed cerebral abnormalities in four of these children.

Among the remaining five children, with impaired mental development, three had chromosomal aberrations. One had trisomy 16, one had XYY and one had Down syndrome (patients 40, 42 and 44, respectively). One boy (patient 41) had multiple organ malformations, and neuroradiology demonstrated agenesis of the corpus callosum. One child (patient 45) had no additional diagnoses.

Children with impaired mental development (n=29)

Twenty-nine (65%) of the 45 children had impaired mental development. Twelve (41%) of these children had no ocular disorder and 4 of these 12 children had normal neuroradiological findings (patients 42–45). Seventeen children had both an ocular disorder and impaired mental development (patients 14–30). Among these, seven had abnormal neuroradiological findings. Altogether, 27 of 29 children with impaired mental development had undergone neurological imaging, which revealed cerebral pathology in 15 of these children.

Children without impaired mental development (n=16)

Of the 16 children without impaired mental development, 13 (81%) had an ocular disorder (patients 1–13). One girl in this group had abnormal neuroradiological findings demonstrating cerebellar vermis hypoplasia.

Of the remaining three children (patients 31–33), all had cerebral pathology

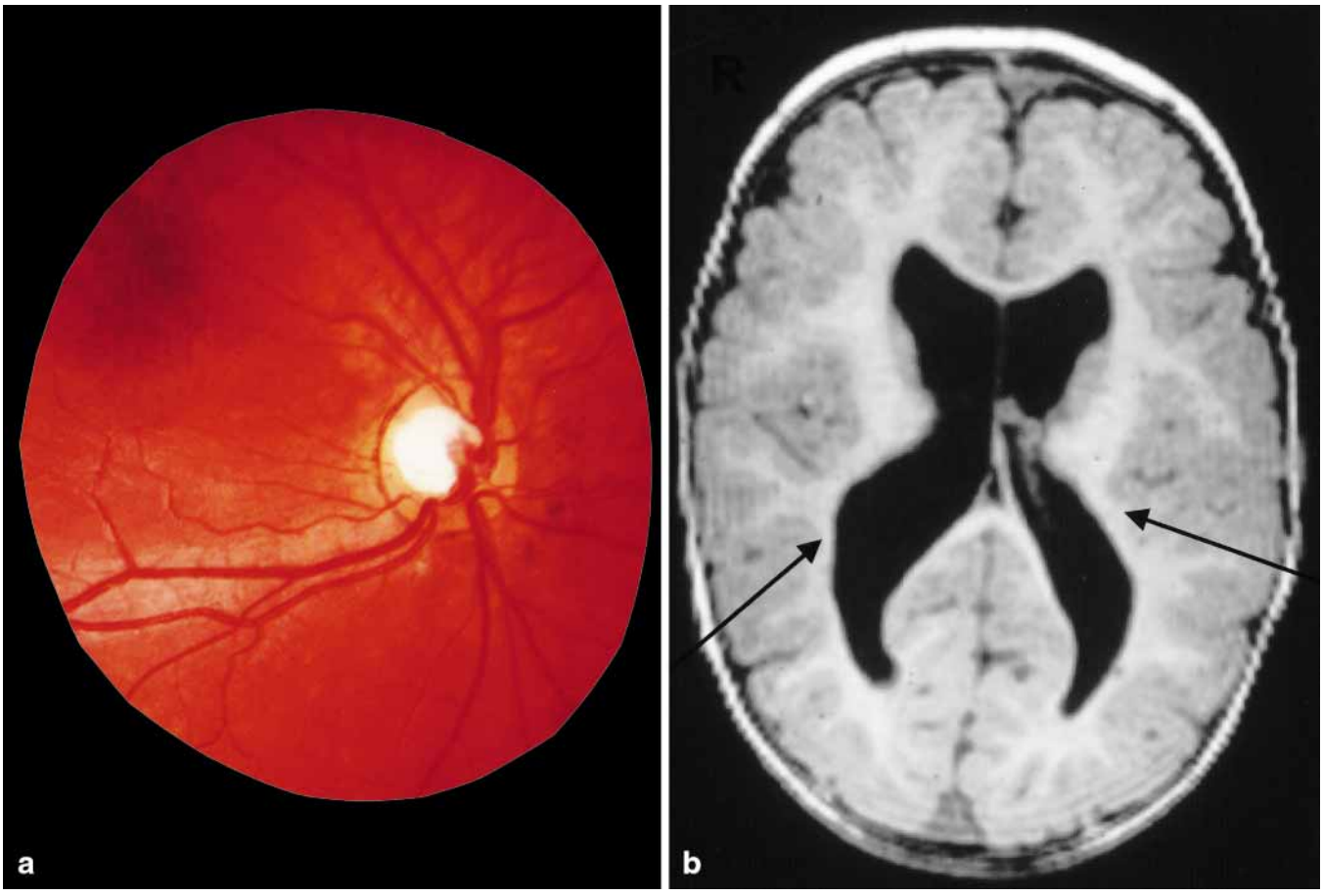

Fig. 2. Fundus photograph of the right eye (a) in a 6-year-old boy with periventricular leukomalacia (patient 17, Table 1), demonstrating a large optic cup in a normal sized optic disc. T1-weighted axial image showing bilaterally reduced periventricular white matter (arrow). This and other images from the same study also shows global reduction of the amount of white matter. The ventricles are dilated secondary to this focal as well as general loss of white cerebral matter. These findings are pathognomonic for Periventricular Leukomalacia.

affecting the posterior visual pathways (two with PVL and one with bilateral occipital atrophy).

Children with cerebral imaging (n=35)

Twenty of the 35 children in whom neuroradiology was performed had an ocular disorder. Neuroradiological findings were abnormal in 8 of these children (patients 13–20). Of the remaining 15 imaged children who had no ocular disorders, 11 had abnormal neuroradiological findings and 8 of these children had impaired mental development. Thus, 4 children had neither ocular nor cerebral morphological abnormalities. Three of these children had chromosomal aberrations (patients 42–44) and one boy had impaired mental development only (patient 45).

Cerebral imaging affecting retrogeniculate visual pathways and/or the visual cortex (n=15)

Cerebral abnormalities affecting the retrogeniculate visual pathways and/or the visual cortex were seen in 15 individuals (12 boys and 3 girls). Six (13%) of the 45 children, all boys, had periventricular leukomalacia (patients 17, 31, 32 and 34–36) and four children had occipital cortical abnormalities (patients 18, 33, 37 and 38). Four children had generalised cerebral atrophy (patients 14–16 and 40) and one boy had pathological myelination (patient 39).

Children without cerebral imaging (n=10)

Neuroradiology was not performed in ten children (patients 1–3, 6–8, 10, 12, 29

Table 2. Ocular disorders in 45 children born full-term, 1989–1995, reported as visually impaired.

Patient number(s)	Type of ocular disorder	Number
5, 13, 14, 17, 19, 24	optic nerve hypoplasia	6
15, 16, 18, 20	optic nerve atrophy	4
11, 21, 25, 26	essentiel congenital nystagmus	4
1, 3, 4	ocular albinism	3
9, 27, 28	fundus coloboma	3
8, 29	aphakia	2
2, 22	retinitis pigmentosa	2
23, 27	microphthalmus	2*
6	Peter's anomaly	1
7	congenital nyctalopia	1
10	vitreoretinal dysplasia	1
30	macular anomaly	1
12	optic disc anomaly	1
31–45	none	15

* Patient 27 in addition with fundus coloboma.

Table 3. Cerebral imaging findings in 35 children born full-term, 1989–1995, reported as visually impaired.

Patient number(s)	Type of cerebral lesion	Number
17, 31, 32, 34–36	periventricular leukomalacia	6
18, 33, 37, 38	occipital atrophy	4
14–16, 40	general cerebral atrophy	4
19, 41	abnormalities corpus callosum	2
39	pathological myelination	1
13	vermis hypoplasia	1
20	optic nerve glioma	1
4, 5, 9, 11, 21–28, 42–45	normal cerebral imaging	16

and 30). All had an ocular disorder explaining their visual impairment. Seven children (patients 1–3, 6–8 and10) had no additional diagnoses, while two of the children (patients 29 and 30) had Down syndrome, and one girl (patient 12) had craniostenosis.

Discussion

Among visually impaired children in the present study, 42% had cerebral morphological abnormalities verified by cerebral imaging, and 66% had signs of cerebral functional abnormalities (impaired mental development and/or neurological symptoms). Twenty-six per cent of the visually impaired children were found to have ocular disorders only.

Absence or impairment of visual fixation and visual behaviour were criteria for inclusion in this study. This report is from a regional centre in Sweden without immediate access to vision-evoked potentials or electroretinograms to validate the clinical observations, however, it was clear from detailed clinical observation and assessment that all the children reported in the present study had visual impairment.

The finding that a large proportion of the visually impaired children also had impaired mental development reflects the association between mental ability and visual performance. It should be noted that accurate visual evaluation is difficult to perform in a child with reduced mental capacity, including poor concentration, poor co-operation and limited communication. In addition, a well functioning visual system includes the ability to process, sort and interpret the visual input, functions that have been shown to be affected in children with impaired mental development. Children with severe visual impairment or blindness also have more severely impaired mental development than sighted children (Warburg 1982). In addition, children with additional impairments, including mental impairment, suffer from the most severe visual impairment (Rosenberg et al. 1996; Blohmé 2000). In the present study there were five children (patients 41–45) who had neither ocular disorders nor abnormal cerebral imaging affecting the retrogeniculate visual pathways or the visual cortex. All of these children had impaired mental development and it may be speculated that their reduced mental capacity is the most likely explanation for their poor visual performance. Children with impaired mental development also often have a delayed appearance of their visual response (Uemura 1979). In the present study, 36 children were below 5 years of age at the time of their visual examination and most of these children had impaired mental development. This may suggest that some of these children will improve their visual capacity later in life, a proposition supported by the studies of Blohmé & Tornqvist 1997 and Blohmé 2000.

Children with cerebral visual impairment have been shown to have complex disorders in cognitive visual functions in addition to visual acuity and/or visual field defects (Good et al. 1994; Ahmed & Dutton 1996; Dutton et al. 1996; Jacobson et al. 1996). Thus, a visually impaired child with cerebral pathology often manifests more severe visual function impairment than a child who is visually impaired due to ocular disorders only (Ek 2000).

In addition, many of the children with abnormal cerebral morphology had additional neurological symptoms, such as epilepsy, cerebral palsy, attention deficit disorder, and ataxia. Among the 16 children with normal imaging, 12 had chromosomal aberrations and/or impaired mental development. These findings further support the frequent finding of additional handicaps among children with visual impairment, and are in accordance with other studies (Rosenberg et al. 1996; Blohmé & Tornqvist 1997a).

In our group of full-term children, 15 (33%) had abnormal cerebral morphology affecting the retrogeniculate visual pathways, while 12 (26%) had ocular disorders only that could explain the impaired vision.

Of the 15 children with abnormal morphology of the retrogeniculate visual pathways, six had PVL. In the visually impaired children born preterm during the same period, analysed and reported previously (Jacobson et al. 1998), ten children had PVL. Another child with PVL was later defined as preterm and a further preterm child had the clinical picture of PVL (cerebral palsy and strabismus) although not verified by neuroradiology. Thus, altogether 18 (27%; 15 boys and 3 girls) out of 66 visually impaired children born in our county between 1989 and 1995, had periventricular leukomalacia. Five of the full-term children with retrogeniculate visual pathway abnormalities also had an ocular diagnosis; namely, optic nerve atrophy and optic nerve hypoplasia. Among the ten preterm children with periventricular leukomalacia, two children had marked optic disc cupping and three had optic nerve atrophy.

In the present study, 26% of the patients with ocular disorders had abnormal neuroradiological findings. This reflects the high association between ocular and cerebral abnormalities. In a study by Waugh et al. (1998), 51% of the children with congenital disorders of the peripheral visual system, i.e. disorders of the anterogeniculate visual pathways and the globe, had brain anomalies confirmed by neuroradiology. In the present study, eight (26%) of the 30 children with ocular disorders had brain anomalies confirmed by neuroradiology. This is a smaller proportion than that reported by Waugh et al., however, when the eight children with ocular disorders and impaired mental development (a sign of brain dysfunction despite normal neuroradiology) are included, the total proportions become comparable. It should be stressed that ten of the children with ocular disorders in our study had not been examined by neuroradiology.

Four of the children had the diagnosis essential congenital nystagmus, a term that in fact is an indication of "cause un-

known". All four children had normal neuroradiological findings and no other ocular manifestations.

The habilitation of children with cerebral visual impairment differs from that of children with ocular visual impairment (Jan & Freeman 1998), not only because they often have multiple disabilities, but also because they often have visual perceptual disturbances in addition to their low visual acuity and/or visual field restriction (Dutton et al. 1996; Jacobson et al. 1996). There are also reasons to believe that even if the child has normal visual acuity and no visual field defects, she or he may still have problems employing the visual system optimally on account of visual perceptual disturbances. Like others, we suggest that the definition of visual impairment ought to be broadened (WHO, Bangkok 1992; Valberg & Fosse 1999), particularly because financial support for habilitation is often dependent upon the definition of visual impairment.

Conclusion

The majority (74%) of children with visual impairment were found to have cerebral morphological and/or cerebral functional abnormalities. It should be stressed that 60% of children with ocular disorders had cerebral morphological and/or cerebral functional abnormalities. We therefore recommend that children with visual impairment should undergo neuroradiological assessment and paediatric examination as a routine component of their work up in order to establish the correct diagnosis, thus allowing children with functional visual impairment access to optimal habilitation strategies.

Acknowledgements

The authors are grateful to Gordon Dutton, Professor, for constructive criticism.

This work was supported by grants from the Sigvard & Marianne Bernadotte Research Foundation for Children Eye Care, the Margit Thyselius Foundation for Blind Youth, the Linnea and Josef Carlsson Foundation, the Foundation of Crown Princess Margareta, the County Council of Värmland. The Knut and Alice Wallenberg Foundation, The Swedish Medical Research Council (No #10863 and #13515) and The Göteborg Medical Society.

References

Ahmed M & Dutton G (1996): Case Reports. Cognitive Visual Dysfunction in a Child with Cerebral Damage. Dev Med Child Neurol 38: 736–743.

Befolkningsstatistik (1994): Statistiska Centralbyrån, Örebro, Sweden.

Blohmé J & Tornqvist K (1997a): Visual impairment in Swedish children. I. Register and prevalence data. Acta Ophthalmol Scand 75: 194–198.

Blohmé J & Tornqvist K (1997b): Visual impairment in Swedish children. II. Etiological factors. Acta Ophthalmol Scand 75: 199–205.

Blohmé J & Tornqvist K (1997c): Visual impairment in Swedish children. III. Diagnoses. Acta Ophthalmol Scand 75: 681–687.

Blohmé J (2000): On Visual Impairment In Swedish Children. Thesis, Faculty Of Medicine, Lund University, ISBN 91–628–4063–0.

Dutton G, Ballantyne J, Boyd G, Bradnam M, Day R, McCulloch D, Mackie R, Phillips S & Saunders K (1996): Cortical visual dysfunction in children: A clinical study. Eye 10: 302–309.

Ek U (2000): Children with visual disorders-cognitive development, developmental disorders and consequences for treatment and counselling. Thesis, Department of Psychology, Stockholm University, ISBN 91–7265–051–6.

Eken P, de Vries L, van der Graaf Y, Meiners L & van Nieuwenhuizen O (1995): Haemorrhagic-ischaemic lesions of the neonatal brain: Correlation between cerebral visual impairment, neurodevelopmental outcome and MRI in infancy. Dev Med Child Neurol 37: 41–55.

Good W, Jan J, DeSa L, Barkovich J, Groenveld M & Hoyt C (1994): Cortical Visual Impairment in Children. Surv Ophthalmol 38: 351–364.

Jacobson L, Ek U, Fernell E, Flodmark O & Broberger U (1996): Visual impairment in preterm children with periventricular leukomalacia – Visual, cognitive and neuropaediatric characteristics related to cerebral imaging. Dev Med Child Neurol 38: 724–735.

Jacobson L, Lundin S, Flodmark O & Ellström K (1998): Periventricular leukomalacia causes visual impairment in preterm children. Acta Opthalmol Scand 76: 593–598.

Jan J & Freeman R (1998): Who is a visually impaired child? Dev Med Child Neurol 40: 65–67.

Kvarnström G, Jakobsson P & Lennerstrand G (1998): Screening for visual and ocular disorders in children, evaluation of the system in Sweden. Acta Paediatr 87: 1173–1179.

Lindstedt E (1972): Severe visual impairment in Swedish children. Doc Ophthalmol 31: 173–204.

Rosenberg T, Flage T, Hansen E, Riise R, Rudanko S, Viggosson G & Tornqvist K (1996): Incidence of registered visual impairment in the Nordic child population. Br J Ophthalmol 80: 49–53.

Uemura Y (1979): The assessment of visual ability in children. Paediatr Ophthalmol 329–331.

Valberg A & Fosse P (1999): Synspröving og WHOs kategorier for synshemning. Synsforum 1: 9–13.

Warburg M (1982): Why are the blind and severely visually impaired children with mental retardation much more retarded than the sighted children? Acta Ophthalmol (Copenh) 157: 72–81.

Waugh M, Chong WK & Sonksen P (1998): Neuroimaging in children with congenital disorders of the peripheral visual system. Dev Med Child Neurol 40: 812–819.

WHO publication (1992): Management of low vision in children. Bangkok 93.27 Geneva.

Received on June 20th, 2000.
Accepted on November 30th, 2000.

Corresponding author:
Sonia Grönqvist
Ögonmottagningen Hagahuset
Trekantsgatan 3
SE-652 20 Karlstad
Sweden
Tel: +46 54 617363, Fax: +46 54 183746
e-mail: sonia.gronqvist@liv.se