Ophthalmic findings in dyslexic schoolchildren

M-L Latvala, T T Korhonen, M Penttinen, P Laippala

Abstract

The ophthalmic findings of 55 dyslexic 12 to 13-year-old Finnish schoolchildren and 50 age, sex, and social class-matched control children were evaluated. On a neuropsychological basis the children could be divided into six subgroups: general deficiency, general language, visuomotor, naming, mixed, and normal. The two groups did not differ significantly from each other in visual acuity, cycloplegic refraction, the amount of phorias and tropias, stereo acuity, fusion, or accommodation. Convergence near point ≥8 cm was, however, statistically more frequent in the dyslexic group. This finding was also significant in the general deficiency subgroup compared with the other subgroups. The most conspicuous common denominator in those with dyslexia was revealed to be the convergence insufficiency type of exodeviation, occurring in 38% of the general deficiency dyslexic subgroup and in 36% of the visuomotor dyslexic subgroup. This finding suggests a low accommodative convergence/accommodation ratio in these children.

(*Br J Ophthalmol* 1994; 78: 339–343)

Approximately 5% to 10% of schoolchildren are in need of special education services for specific difficulties in reading and writing. This is in spite of their normal intelligence, apparent health, profitable sociocultural environment, and conventional instruction. Neuroanatomical studies[1] and neuroradiological studies on people with dyslexia[2–4] have revealed cortical cytoarchitectural changes and exceptional symmetry or reversed asymmetry in the language associated planum temporale or angular gyrus regions in the brains of those with dyslexia. Functional studies using positron emission tomography[5] and brain electrical mapping techniques[6] have, in addition, shown changes in the frontal motor areas and in the inferior, visually related regions of both occipital lobes. The active brain areas are also mutually different in different dyslexic subtypes.[6,7]

Many researchers have considered the basic difficulty in dyslexia to be in the processing of phonemic information or in retrieving information from the verbal memory.[8–13] The latest findings have also shown sluggish visual information transfer at least in some dyslexics.[14] References to this are seen in some previous visual evoked potential (VEP) studies.[15,16] Underdevelopment of the magnocellular pathway, which carries transient visual information in the low and middle spatial frequency ranges, has been revealed also neuroanatomically.[14]

Clinically, dyslexic children have been subgrouped according to psychoeducational or neuropsychological measures, achievement test results, or the types of reading and spelling errors. Two of the latest studies come from Scandinavia. In the Norwegian study[17] dyslexic children were divided into four subgroups on the basis of educational psychological tests and examined also ophthalmically. In Finland, Korhonen evaluated learning disabled schoolchildren and matched controls on a neuropsychological basis.[18] These children could be divided into six subgroups, resembling those found earlier among English speaking children.

The ophthalmic and orthoptic studies on dyslexic children are contradictory. In many studies dealing with dyslexics and controls without subgrouping them, no essential differences in visual acuity, refraction, or strabismus have been found.[19–22] Some studies have shown weak evidence of poor binocularity or deficient oculomotor control mechanims.[23–30] Vellutino found no basic differences between the visual perception of dyslexic and normal children,[9] neither did Valtin.[31] Haddad reported reduced fusion ability among many individual poor readers.[29] Orthoptic exercises improved fusion and relieved subjective troubles without any effect on dyslexia. Stein *et al*[32] found unstable vergence control for small targets in 64% of 10-year-old dyslexic children compared with stable ocular motor dominance in nearly all normal readers. Their findings were supported by Bigelow and McKenzie[33] but were considered questionable by Newman *et al*[34] and Bishop.[35] Geiger and Lettvin[36] found that dyslexic people identified letters extrafoveally better than normal readers and showed parafoveolar masking. Their research has been criticised for study arrangements, however.[37] Abnormalities in the eye tracking movements which have sometimes been observed probably reflect the difficulties which many dyslexics possess when reading a strange text.[38,39] Hyvärinen and Laurinen[40] found several abnormal ophthalmic findings such as great refractive errors, poor binocularity, or abnormal contrast transfer in severely dyslexic children.

The purpose of the present study was to evaluate whether ophthalmic findings could have contributed to the learning disability of schoolchildren, subgrouped on a neuropsychological basis, and whether some differences in that respect could be found between the subgroups.

Subjects and methods

The subjects in the present study came from a follow up study of empirically derived subgroups of children with learning disabilities (LD). In the initial study,[18] a sample of 82 LD children and 84 sex, age, and social class-matched control children was drawn from the entire population of 1607 third grade, Finnish speaking, 9 to 10-year-old children. To be included subjects had to have difficulties with reading and/or spelling to such

Community Health Center, Ophthalmology Unit, Koulukatu 9D, 33200 Tampere 20, Finland
M-L Latvala
T T Korhonen
M Penttinen
P Laippala

Correspondence to:
Dr Marja-Leena Latvala.

Accepted for publication
21 October 1993

an extent that their teacher and a special teacher for reading and spelling problems agreed on the need of remedial teaching. Moreover, these difficulties had to have continued from the second school year into the middle of the third school year. Altogether, 56 teachers from 27 schools reported such children. When evaluating the learning disabilities the teachers used a rating scale from 1 to 4. A rating of at least 3 in reading and/or writing or a rate of 2 in both of these areas was used as the criterion for the inclusion of a subject in the study.

To avoid extreme cases, which may strongly have affected the method (cluster analysis) used to subgroup the subjects, children with a WISC-R full scale IQ of less than 80, children with neurological diseases, and children coming from very extreme family situations (for example, asocial families) were excluded. The learning disabilities in the present sample were relatively mild, and followed the definition of learning disability stated by the Joint Committee on Learning Disability.[41] Only four of the LD children studied in a class for perceptual disorders; all others studied in the regular third grade classes. However, for 80% of the 82 LD children the reading and/or the writing error sum score on a reading and writing test[42 43] was higher than the mean (+2 SD) of the control group.

A panel of 19 neuropsychological tests for a total of 32 measures was administered to the subjects. All children were also submitted to a conventional paediatric and neurological examination. Using cluster analysis[44] the sample could be divided into six subgroups (Table 1). The labels of the subgroups describe the neuropsychological weaknesses typical for each group.

About 3 years later, at the age of 12–13 years, 74 learning disabled and 57 control children participated in a neuropsychological as well as a neuropaediatric follow up examination.[45] In this phase they were also asked to undertake an ophthalmic evaluation; 55 learning disabled and 50 control children did so. The subgroups of these subjects are shown in Table 1. The proportion of boys to girls was 1·8:1 or about the same as in the initial study.

Visual acuity with best correction was tested both before and after cycloplegia, using a Snellen E-chart at 6 metres. Before cycloplegia, near visual acuity was also tested using a standardised reading card (Instru-card) at 33 cm distance. Eye movements were checked and the convergence ability was measured. Possible strabismus was evaluated by the cover test and by Maddox wing near and Maddox rod far. The near point of accommodation was measured using an RAF ruler, with push up method, and the refraction corrected. Fusion amplitude was registered by prism bar at 6 metres and 33 cm distance, with a finding of unrecovering diplopia or turning out of one eye as the end point. Stereo acuity was examined by the Titmus test. Contrast sensitivity and central visual fields were evaluated using the LH contrast test and tangent screen, respectively. Colour vision was screened by the Panel D-15 test in artificial daylight illumination (Airam 60W), both eyes separately. Eyes in which visual acuity was ≤0·7 or spherical refraction ≥3 dioptres myopic were excluded to avoid misinterpretation of the results. The refractive power was determined by streak retinoscopy with and without cycloplegia – to test for cycloplegia, two drops of 1% cyclopentolate were instilled twice with a 5 minute interval and the measurement was carried out after at least 40 minutes. Ophthalmoscopy was performed through the dilated pupils. The anterior segments were examined using a Haag-Streit biomicroscope.

To evaluating the exophoria findings, the classification by Daum[46] was followed:

1 Equal exodeviation (EE): a change between the distant and near values of less than 4 Δ (prism dioptres), if the distance angle of exodeviation is from 0 to 5 Δ, and a change of less than 10 Δ if the distance angle of exodeviation is 6 Δ or more.

2 The convergence insufficiency type (CI): near exodeviation 4 Δ or greater than the distance deviation when the distance exodeviation is between 0 and 5 Δ, and the near deviation 10 Δ or greater if the distance exodeviation is 6 Δ or more.

3 The divergence excess type (DE): the same limits of change as in the convergence insufficiency type, but with the distance exodeviation being greater.

The statistical analysis is based on cross tabulations and χ^2 tests. We also performed logistic regression modelling, but the results were analogous to cross tabulations.

Results

In the control group, all children had visual acuity >0·7 in both eyes. In the dyslexic group, two children (3·6%) had bilateral visual acuity ≤0·7 (one 0·6, the other 0·7) and two children (3·6%) had unilateral amblyopia ≤0·7. This difference was not statistically significant. Figure 1 presents the spherical refraction in the dyslexic and control groups. In the dyslexic group, two subjects (3·6%) had astigmatism ≥1 D and two (3·6%) had anisometropia ≥1 D. None in the control group had astigmatism of that magnitude, three (6%) had anisometropia ≥1 D. Three children in both groups had an accommodation near point ≤7 D, and six subjects in both groups had some abnormality in colour vision by Panel D-15 test. Only one dyslexic child, with septum pellucidum anomaly and bilateral visual acuity of 0·6, had clearly abnormal contrast sensitivity. Four children in the dyslexic group had bilaterally abnormal optic discs: one was the above

Table 1 The proportion of ophthalmically examined dyslexic and control children in the six subgroups

	Dyslexics			*Controls*			*Total*	
Subgroup	*n*	*%*	*(%)**	*n*	*%*	*(%)†*	*n*	*%*
General deficiency	13	24	(21)	1	2	(1)	14	13
General language	11	20	(22)	2	4	(5)	13	12
Visuomotor	11	20	(22)	12	24	(21)	23	22
Naming	8	14	(11)	0	0	(1)	8	8
Mixed	11	20	(18)	5	10	(10)	16	15
Normal	1	2	(6)	30	60	(62)	31	30
	55	100	(100)	50	100	(100)	105	100

*Percentage of the subgroup in the original group of dyslexic children (n=82). The difference between the percentage is not statistically significant.
†Percentage of the subgroup in the original group of control children (n=84). The difference between the percentages is not statistically significant.

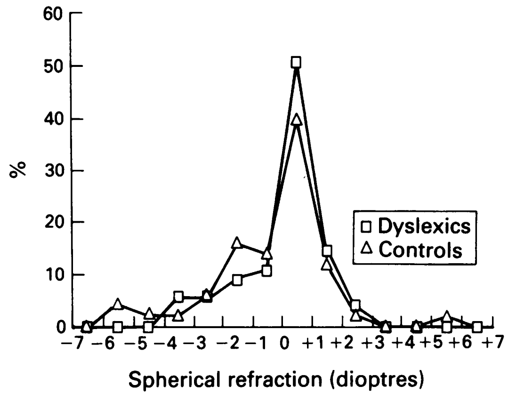

Figure 1 The distribution of cycloplegic refraction in dyslexic and control groups (right eye, spherical equivalent).

mentioned girl with septum pellucidum anomaly, having hypoplastic discs; two had small optic discs but good visual acuities, and one had markedly tilted discs and good visual acuities. One child in the control group had pseudostasis papillae with good vision in both eyes. Table 2 presents the binocular function in both groups.

When the above mentioned variables were compared between the six neuropsychologically based subgroups consisting of dyslexic and control children, no remarkable differences were found. Nine variables were selected as being the most relevant to reading and writing abilities (referred to later as reading variables) (Table 3). At this stage, two dyslexic children with bilaterally reduced visual acuities were omitted: the girl from the general deficiency subgroup who had hypoplastic discs and visual acuity of 0·6 bilaterally, and another girl from the mixed subgroup who had astigmatism 3·5 D/2·0 D and bilateral acuity of 0·7. Distribution of the abnormal reading findings in the subgroups of dyslexic and control subjects is shown in Table 3. The differences were of no statistical significance. The number of children in the dyslexic group was too small to allow reliable statistical evaluation of the reading findings between only the dyslexic subgroups. When the occurrence of individual reading variables in five other subgroups was compared with that of the normal subgroup, two statistically significant differences were found: convergence near point ≥8 cm in the general deficiency subgroup (p=0·0470) and unilateral visual acuity ≤0·7 in the naming subgroup (p=0·0497). When the exophorias at reading distance were evaluated further, some interesting observations were made. As Table 4 shows, the type of exophoria in the dyslexic group was invariably convergence insufficiency. In the control group it was more heterogeneous. In the dyslexic group the near exophoria was combined with hyperopic or emmetropic refraction in 9/13 (69%) of the cases, and with myopia in 4/13 (31%). In the control group the refraction was equally distributed.

Table 2 Binocularity in the dyslexic and control groups

	Dyslexics (n=55)		*Controls (n=50)*		
	%	*n*	%	*n*	
Heterophoria at 33 cm:					
esophoria ≥3Δ	5·8	3/52	6·1	3/49	
exophoria ≥6 Δ	25·0	13/52	12·2	6/49	
vertical phoria ≥2 Δ	9·4	5/53	6·1	3/49	
Heterotropia at 33 cm	3·6	2/55	2·0	1/50	
Heterophoria at 6 m:					
esophoria ≥3 Δ	11·3	6/53	8·2	4/49	
exophoria ≥6 Δ	1·9	1/53	2·0	1/50	
vertical phoria ≥2 Δ	3·8	2/53	4·1	2/49	
Fusion amplitude:					
≤32 Δ at 33 cm	7·5	4/53	6·1	3/49	
≤15 Δ at 6 m	9·4	5/53	12·2	6/49	
Convergence near point ≥8 cm	12·7	7/55	2·0	1/50	p=0·0385
Stereo acuity lacking or deficient (>60 seconds of arc)	9·1	5/55	8·0	4/50	

Discussion

When defining specific dyslexia, clear external causes for reading difficulties such as significant uncorrected hypermetropia must be excluded. Many authors have failed to find any positive correlation between conventional ophthalmic status and dyslexia, and instead have found evidence of overtreatment of minor findings with unnecessary glasses, unnecessary orthoptic exercises, etc.[21 47] Comparing dyslexic and control persons as groups has, in general, not produced any remarkable differences. But this kind of approach has aroused criticism.[31 48 49] The same finding may be more important for one individual, meaning, for example, a different binocular situation.[48] The crowding of many minor findings may also be important.

In this study, when comparing the two groups, slightly more cases with convergence insufficiency, exophoria at near, or a slight hypermetropia were found in the dyslexic group (Table 2, Fig 1). In this respect, the findings are comparable with, for example, those of Eames[24] and Bishop *et al.*[50] The dyslexics also had unilateral or bilateral amblyopia or mild bilateral disc anomalies more often. The number of colour confusions was unexpectedly high in both groups, most of the children, especially in the

Table 3 Reading variables in the dyslexic and control children

		Number of abnormal reading variables				
Abnormal reading variables	*Subgroup*	*0*	*1*	*2*	*≥3*	*n*
	Dyslexics:					
1 Refraction (right eye), spherical equivalent, ≥+2 D	general deficiency	3 (25%)	4	3	2 (17%)	12
2 Anisometropia ≥1 D	general language	7 (64%)	2	1	1 (9%)	11
3 Astigmatism (right eye) ≥1 D	visuomotor	5 (45%)	4	2	0	11
	naming	5 (63%)	2	0	1 (13%)	8
4 Horizontal phoria/tropia at 33 cm	mixed	7 (70%)	3	0	0	10
5 Fusion ≤32 Δ at 33 cm	normal	0	1	0	0	1
6 Convergence near point ≥8 cm	total	27 (51%)	16 (30%)	6 (11%)	4 (8%)	53
	Controls:					
7 Reduced stereo acuity	general deficiency	0	1	0	0	1
8 Accommodation ≤7 D	general language	1 (50%)	1	0	0	2
9 Visual acuity ≤0·7 (unilateral)	visuomotor	10 (83%)	1	0	1 (8%)	12
	mixed	4 (80%)	1	0	0	5
	normal	18 (60%)	7	3	2 (7%)	30
	total	33 (66%)	11 (22%)	3 (6%)	3 (6%)	50

Table 4 Evaluation of dyslexic and control children with near exopheria

Patient No	Refraction (spherical)	Type of exophoria	Reduced stereo acuity	Fusion ≤32	Deficient convergence ≥8 cm	Accommodation ability ≤7 D	Subgroup	Proportion of convergence insufficiency type of exophoria in the subgroup
Dyslexics:								
1	−2·37/−1·75	CI					General deficiency	
5	+1·0/+1·0	CI	+				General deficiency	
8	+0·75/+0·75	CI					General deficiency	5/13=38% of the subgroup
9	−2·75/−2·5	CI					General deficiency	
10	+1·0/+0·82	CI	+	+	+		General deficiency	
14	+1·0/+1·0	CI					Visuomotor	
15	+0·5/+0·5	CI			+		Visuomotor	4/11=36% of the subgroup
18	±0/+0·25	CI					Visuomotor	
20	+1·37/+1·12	CI					Visuomotor	
29	−1·25/−1·5	CI					Mixed	1/11=9% of the subgroup
37	−2·87/−3·0	CI					General language	2/11=18% of the subgroup
42	+2·25/+2·0	CI					General language	
55	+0·75/+0·5	CI					Normal	1/1=100% of the subgroup
Controls:								
69	+1·0/+1·0	CI					Mixed	1/5=20% of the subgroup
97	+1·12/+0·87	EE					Normal	
99	+0·75/+0·75	EE				+	Normal	
100	−0·87/−0·37	EE	+			+	Normal	
103	−5·0/−4·5	CI		+	+		Normal	2/30=6% of the subgroup
105	−2·5/−2·5	CI					Normal	

CI=convergence insufficiency.
EE=equal deviation.

dyslexic group, showing unspecific errors. This may be caused by inattentiveness after numerous other tests. The Panel D-15 test in this age group may not be very reliable, either.[51] Quite a high proportion of children in the control group also had some positive findings. In this respect the conclusions coincide with those by Aasved,[17] Norn,[19] and Blika.[21]

Subgrouping dyslexic children allows a more individual approach to the problem. A neuropsychological basis for subgrouping is relevant, giving comprehensive information of an individual's functional and developmental profile. In the Bergen study,[17] no statistical differences were found between the subgroups. Their subgroups – auditory, audiovisual, visual, and others – were different from those in this study, however. Comparing independent ophthalmic variables between our subgroups did not give remarkable differences either. Only a slight accumulation of the findings in the general deficiency subgroup was noticed. One should, however, realise that the subgroups that emerged were so small that the statistical analysis does not have enough power to detect dependence structures, and, accordingly, certain conclusions must be made without statistical support.

The IQ difference between LD subjects and control subjects in this study (the mean for the LD children being 97·7 and for the control children 110·2) reflects the common fact that most of the LD subjects had specific or, as in the general deficiency subgroup, general neuropsychological and cognitive disorders. This also affected the IQ results, and similarly caused some IQ differences between the subgroups. The general deficiency and the naming subgroups were the most impaired showing also the greatest amount of neurological soft signs (50% and 40% with two or more, respectively).[18] These subgroups also had the most unfavourable prognosis in reading and spelling, the naming subgroup being more specified in its function deficits.[45] The finding that control subjects in the visuomotor subgroup also had visuomotor spatial problems is interesting. It has been hypothesised that mild visuomotor spatial difficulties may not directly cause reading problems.[18] The LD subjects, but not the control subjects, in the visuomotor subgroup also showed attentional and some mild language difficulties, which may primarily have caused the reading problems. Comparison of the eye findings in the dyslexic and normal visuomotor subgroups revealed only slightly more positive findings in the dyslexics. During follow up the visuomotor dyslexic subgroup made good progress in reading.[45]

The most conspicuous common denominator for the dyslexic children was revealed to be the convergence insufficiency type of exodeviation, seen in 25% of these children. It means that their accommodative power is relatively small and the accommodative convergence/accommodation ratio (AC/A) low. However, no regular decrease in accommodation amplitude could be detected in these children. This refers to other aspects in accommodation being probably more important but more difficult to measure as also suggested by Lennerstrand.[39] The four exophoric children in our dyslexic visuomotor subgroup were all also hyperopic or emmetropic while the two esophoric children were myopic. In the control visuomotor subgroup no children had exophoria; one with deviation had esophoria (far) combined with hyperopic refraction and the other had esotropia, myopic anisometropia, and no fusion. References to poor vergence control or slight overpresentation of near exophoria in dyslexic children are not uncommon in previous reports,[19 24-26 30 32] either, although in a recent carefully performed study[52] no statistical differences were found. One could, however, speculate that the combination of hyperopic refraction and low AC/A ratio at the critical age constitutes an unfavourable basis for learning to read. Associated with some developmental delays or mild neurological problems its importance may increase.

The present study shows that ophthalmic factors ought not be overlooked as a contributing factor to dyslexia in at least some individuals. Maybe they sometimes constitute part of the dyslexic syndrome and become more significant when crowding together. A dyslexic child would benefit from individual, also ophthalmic, evaluation. Correcting ophthalmic, abnormalities, when possible, creates more favourable opportunities for special education. This is, by far, the best means of rehabilitation.

1 Galaburda AM. Neuropathologic correlates of learning disabilities. *Sem Neurol* 1991; **11:** 20–7.
2 Hier DB, LeMay M, Rosenberger PB, Perlo VP. Developmental dyslexia: evidence for a subgroup with a reversal of cerebral asymmetry. *Arch Neurol* 1978; **35:** 90–2.
3 Hynd GW, Semrud-Clikeman M, Lorys AR, Novey ES, Eliopulos D. Brain morphology in developmental dyslexia and attention deficit disorder/hyperactivity. *Arch Neurol* 1990; **47:** 919–26.
4 Duara R, Kushch A, Gross-Glenn K, Barker WW, Jallad B, Pascal S, *et al.* Neuroanatomic differences between dyslexic and normal readers on magnetic resonance imaging scans. *Arch Neurol* 1991; **48:** 410–6.
5 Gross-Glenn K, Duara R, Barker WW, Loewenstein D, Chang J-Y, Yoshii F, *et al.* Positron emission tomographic studies during serial word-reading by normal and dyslexic adults. *J Clin Exp Neuropsychol* 1991; **13:** 531–44.
6 Duffy FH, McAnulty GB. Brain electrical activity mapping (BEAM): the search for a physiological signature of dyslexia. In: Duffy FH, Geschwind N, eds. *Dyslexia. A neuroscientific approach to clinical evaluation.* Boston/Toronto: Little, Brown, 1985: 105–22.
7 Duffy FH, McAnulty G. Neurophysiological heterogeneity and the definition of dyslexia: preliminary evidence for plasticity. *Neuropsychologia* 1990; **28:** 555–71.
8 Flynn JT. Dyslexia. *Int Ophthalmol Clin* 1984; **24:** 177–92.
9 Vellutino FR. Dyslexia. *Sci Am* 1987; **256:** 20–7.
10 Council Report. Dyslexia – Council on Scientific Affairs. *JAMA* 1989; **261:** 2236–9.
11 Dyslexia [Editorial]. *Lancet* 1989; ii: 719–20.
12 Beauchamp GR, Kosmorsky G. Learning disabilities: update comment on the visual system. *Pediatr Clin N Am* 1987; **34:** 1439–46.
13 Rumsey JM. The biology of developmental dyslexia. *JAMA* 1992; **268:** 912–5.
14 Livingstone MS, Rosen GD, Drislane FW, Galaburda AM. Physiological and anatomical evidence for a magnocellular defect in developmental dyslexia. *Proc Natl Acad Sci USA* 1991; **88:** 7943–7.
15 Byring R, Järvilehto T. Auditory and visual evoked potentials of schoolboys with spelling disabilities. *Dev Med Child Neurol* 1985; **27:** 141–8.
16 Cohen J, Breslin PW. Visual evoked responses in dyslexic children. *Ann N Y Acad Sci* 1984; **425:** 338–44.
17 Aasved H. Ophthalmological status of school children with dyslexia. *Eye* 1987; **1:** 61–8.
18 Korhonen T. An empirical subgrouping of Finnish learning-disabled children. *J Clin Exp Neuropsychol* 1991; **13:** 259–77.
19 Norn MS, Rindziunski E, Skydsgaard H. Ophthalmologic and orthoptic examinations of dyslectics. *Acta Ophthalmol* 1969; **47:** 147–60.
20 Cassin B. Strabismus and learning disabilities. *Am Orthopt J* 1975; **25:** 38–45.
21 Blika S. Ophthalmological findings in pupils of a primary school with particular reference to reading difficulties. *Acta Ophthalmol* 1982; **60:** 927–34.
22 Hall PS, Wick BC. The relationship between ocular functions and reading achievement. *J Pediatr Ophthalmol Strabismus* 1991; **28:** 17–9.
23 Evans BJ, Drasdo N. Review of ophthalmic factors in dyslexia. *Ophthalmol Physiol Opt* 1990; **10:** 123–32.
24 Eames TH. Comparison of eye conditions among 1000 reading failures, 500 ophthalmic patients and 150 unselected children. *Am J Ophthalmol* 1948; **31:** 713–7.
25 Hammerberg E, Norn MS. Defective dissociation of accommodation and convergence in dyslectic children. *Acta Ophthalmol* 1972; **50:** 651–4.
26 Bedwell CH, Grant R, McKeown JR. Visual and ocular control anomalies in relation to reading difficulty. *Br J Educ Psychol* 1980; **50:** 61–70.
27 Stein JF, Fowler S. Diagnosis of dyslexia by means of a new indicator of eye dominance. *Br J Ophthalmol* 1982; **66:** 332–6.
28 Brinkley JR, Walonker F. Convergence amplitude insufficiency. *Ann Ophthalmol* 1983; **15:** 826–31.
29 Haddad HM, Isaacs NS, Onghena K, Mazor A. The use of orthoptics in dyslexia. *J Learning Disabilities* 1984; **17:** 142–4.
30 Buzzelli AR. Stereopsis, accommodative and vergence facility: do they relate to dyslexia? *Optometry Vis Sci* 1991; **68:** 842–6.
31 Valtin R. Dyslexia: deficit in reading or deficit in research? *Reading Research Quarterly* 1978/79; **14:** 201–21.
32 Stein JF, Riddell PM, Fowler S. Disordered vergence control in dyslexic children. *Br J Ophthalmol* 1988; **72:** 162–6.
33 Bigelow ER, McKenzie BE. Unstable ocular dominance and reading ability. *Perception* 1985; **14:** 329–35.
34 Newman SP, Wadsworth JF, Archer R, Hockly R. Ocular dominance, reading and spelling ability in schoolchildren. *Br J Ophthalmol* 1985; **69:** 228–32.
35 Bishop DV. Unfixed reference, monocular occlusion, and developmental dyslexia – a critique. *Br J Ophthalmol* 1989; **73:** 209–15.
36 Geiger G, Lettvin JY. Peripheral vision in persons with dyslexia. *N Engl J Med* 1987; **316:** 1238–43.
37 Shaywitz BA, Waxman SG. Dyslexia. *N Engl J Med* 1987; **316:** 1268–70.
38 Olson RK, Kliegl R, Davidson BJ. Dyslexic and normal readers' eye movements. *J Exp Psychol* 1983; **9:** 816–25.
39 Lennerstrand G, Ygge J. Dyslexia; ophthalmological aspects 1991. *Acta Ophthalmol* 1992; **70:** 3–13.
40 Hyvärinen L, Laurinen P. Ophthalmological findings and contrast sensitivity in children with reading difficulties. In: Zotterman Y, ed. *Dyslexia – neuronal, cognitive and linguistic aspects.* International symposium, 1980. Oxford, Pergamon, 1982: 117–22.
41 Hammill DD, Leigh JE, McNutt G, Larsen SC. A new definition of learning disabilities. *Learning Disability Quarterly* 1981; **4:** 336–42.
42 Ruoppila I, Röman K, Västi M. KTL:n diagnostisia lukukokeita peruskoulun II ja III luokille (KTL diagnostic reading tests for grades II and III of the comprehensive school). Reports from the Institute for Educational Research 41. University of Jyväskylä 1968. (in Finnish).
43 Ruoppila I, Röman K, Västi M. KTL:n diagnostisia kirjoituskokeita peruskoulun II ja III luokille. (KTL diagnostic writing tests for grades II and III of the comprehensive school). Reports from the Institute for Educational Research 50. University of Jyväskylä 1969. (in Finnish).
44 Dixon WJ. *BMDP statistical software.* Berkeley: University of California Press, 1983.
45 Korhonen T. Neuropsychological stability and prognosis of subgroups of children with learning disabilities. *J Learning Disabilities* 1991; **24:** 48–57.
46 Daum K. Characteristics of exodeviations: I. A comparison of three classes. *Am J Optomet Physiol Optics* 1986; **63:** 237–43.
47 Metzger RL, Werner DB. Use of visual training for reading disabilities: a review. *Pediatrics* 1984; **73:** 824–9.
48 Suchoff JB. Research on the relationship between reading and vision – what does it mean? *J Learning Disabilities* 1981; **14:** 573–6.
49 Temple CM. New approaches to the development dyslexias. *Adv Neurol* 1984; **42:** 223–32.
50 Bishop DV, Jancey C, Steel AM. Orthoptic status and reading disability. *Cortex* 1979; **15:** 659–66.
51 Pinckers A. Color vision and age. *Ophthalmologica, Basel* 1980; **181:** 23–30.
52 Ygge J, Lennerstrand G, Rydberg A, Wijecoon S, Pettersson B-M. Oculomotor functions in a Swedish population of dyslexic and normally reading children. *Acta Ophthalmol* 1993; **71:** 10–21.