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CONTRAST SENSITIVITY, OCULAR DOMINANCE AND SPECIFIC READING DISABILITY

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Summary—1. We have investigated the possibility that reduced flicker contrast sensitivity and unstable ocular dominance, which is revealed by failure in the Dunlop Test, may be associated in children with specific reading disability (SRD). We measured childrens' contrast sensitivity in two experiments. In Expt 1, we measured the flicker and static contrast sensitivity of 11 SRD children who passed the Dunlop Test, 11 SRD children who failed the Dunlop Test and 11 normal, control children, all of whom were matched for chronological age. We confirmed that, on average, all SRD children were less sensitive to flicker than normals at all spatial frequencies. But SRD children who failed the Dunlop Test were significantly less sensitive to the flickering gratings than those who passed it.

2. We wanted to be sure that these findings could not be attributed to systematic differences in chronological age, reading ability or intelligence. Therefore in Expt 2 we measured the flicker and static contrast sensitivities of two groups of children who differed only in their Dunlop Test performance. Thus the two groups were matched as closely as possible for age, reading age and IQ. Despite these more stringent controls, Dunlop Test failure was still significantly correlated with reduced flicker contrast sensitivity at all spatial frequencies.

3. Together these results suggest that flicker contrast sensitivity and the stability of ocular dominance may be linked in SRD children. Moreover, we suggest that the reduced flicker contrast sensitivities we observed could be caused by reduced magnocellular sensitivity. Finally, our findings support the idea that abnormal visual processing could affect how children read.

Key words—Specific reading disability; Dunlop test; ocular dominance; contrast sensitivity.

INTRODUCTION

In the last ten years, two kinds of experiment have suggested that children with specific reading disability (SRD) process visual information differently from normal children of the same age.

The first technique has used spatial frequency analysis of vision (Campbell and Robson, 1968). Using static grating stimuli, Lovegrove *et al.* (1982) and Martin and Lovegrove (1984) found that groups of SRDs, aged between 12 and 14 y, showed a moderate reduction in contrast sensitivity (*c.* 0.1 log units) at low spatial frequencies (<2 c/deg) when they were compared with age matched normal controls. The same subjects also showed slightly increased sensitivity (<0.1 log units) for higher spatial frequency stimuli (>6 c/deg) than normal controls. Stronger effects have been found in the temporal domain. For example, Martin and Lovegrove (1987) compared the contrast sensitivities of groups of 13 and 14 y old SRDs with normal children, using counterphase modulated gratings. SRD children showed reduced sensitivity to flickering gratings at all spatial frequencies. This effect was most marked at temporal frequencies of 20 Hz and above.

Psychophysical evidence from humans suggests that early visual processing is shared between a "sustained" and a "transient" system. The sustained channel is a high acuity system which is most sensitive to high spatial and low temporal frequencies. In contrast, the transient system is most sensitive to low spatial and high temporal frequencies. The latter is thought to respond mainly to stimulus movement and flicker (Tolhurst, 1975). Thus, the data described above concerning reduced flicker contrast sensitivity has been interpreted as revealing a transient system deficit in SRD children (Lovegrove *et al.*, 1986, 1990).

Type I visible persistence is defined as the "continued visible response to a stimulus after stimulus offset that is indistinguishable from that occurring during actual presentation of the

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stimulus" (Bowling and Lovegrove, 1981). The duration of visible persistence is thought to depend upon how long it takes transient system activity (triggered by stimulus offset) to inhibit activity in the sustained system (Breitmeyer *et al.*, 1981). Visible persistence normally increases as a direct function of spatial frequency. However, Lovegrove *et al.* found that the slope of the function relating persistence duration to the spatial frequency of test stimuli was much flatter in SRDs than in age matched normals, the curve being elevated at low spatial frequencies and lowered at high spatial frequencies (Lovegrove *et al.*, 1980). This result is also consistent with a transient system deficit in SRDs. Finally, Lovegrove *et al.* (1986) performed a discriminant function analysis on the results of their visible persistence data. They showed that 46/61 (75%) of their SRD sample fell into the "weak transient" system category as compared with only 5/61 (5%) of normals.

Another kind of visual attribute studied in SRD children has been the stability of ocular dominance, as measured by the Dunlop Test. This test is thought to assess how reliably retinal information is associated with extra-retinal information about the position of the two eyes in the head. Stein and Fowler (1981, 1985) have suggested that stable ocular dominance is important to allow accurate estimation of the visual direction of small binocularly viewed targets, such as letters on a page (Stein and Fowler, 1981). They have found that as many as 65% of 7–11 y old SRD children may experience unstable ocular dominance, as revealed by their inconsistent responses in the Dunlop Test (Stein and Fowler, 1985). (Also see Methods for description of the Dunlop Test.) By comparison, only 20% of unselected primary school children of the same age show unstable responses in the Dunlop Test (Stein *et al.*, 1986), 3–10% of whom would be expected to be reading disabled anyway (Rutter and Yule, 1975). The Dunlop Test has proved controversial in that some investigators have failed to replicate Stein and Fowler's findings (Newman *et al.*, 1985; Bishop *et al.*, 1979), while others have succeeded (Bigelow and MacKenzie, 1985; Masters, 1988). Nevertheless, the fact that 75% of SRDs show evidence consistent with reduced transient function while as many as 65% may have unstable ocular dominance, raises the question of whether these two phenomena could be associated in reading disabled children.

Therefore, we measured childrens' static and flicker contrast sensitivities in two experiments in which we made different kinds of group comparison. In Expt 1, we followed the same protocol as Lovegrove *et al.* but compared three groups of chronologically age-matched children: primary school children with normal binocular vision, SRDs who passed the Dunlop Test (i.e. they had stable ocular dominance), and SRDs who failed the Dunlop Test (i.e. they had unstable ocular dominance). In Expt 2, we wanted to ensure that any differences we observed in contrast sensitivity could not be attributed to differing levels of reading experience. We therefore matched children for reading age, as well as chronological age and IQ, and measured the static and flicker contrast sensitivities of children who passed the Dunlop Test and compared them with children who failed the Dunlop Test.

EXPERIMENT 1

Methods

Subjects. We selected subjects from a population of children who had been referred to the Royal Berkshire Hospital, Reading, for orthoptic assessment because of reading difficulty. Children were defined as SRD if their reading age measured on the British Ability Scales (B.A.S.) reading test fell two or more SDs behind that predicted from their age and B.A.S. IQ. The experimental groups comprised 11 SRD children who failed the Dunlop Test and 11 who passed it. The two groups of SRD children were compared with 11 children from a local primary school, who had normal binocular vision. All three groups of children were matched as closely as possible for chronological age and IQ, as summarized in Table 1.

Orthoptic and psychological assessment. Every child was examined to exclude orthoptic and gross ophthalmological pathology, before s/he performed the Dunlop Test. Assessment included separate measurements of the Snellen acuities of the two eyes. In addition we measured each child's stereoacuity using the Randot test, and his/her near point for convergence and accommodation using the R.A.F. rule. In this test, a letter target (for accommodation) or small dot (for convergence) is moved smoothly in the sagittal plane towards the subject who states when the target becomes blurry (*c.* 6–8 cm for near point of accommodation) or diplopic (*c.* 6–8 cm for near point of

Table 1. Age, reading age and IQ for children taking part in Expt 1

	Normal ($n = 11$)	Dunlop Test pass ($n = 11$)	Dunlop Test fail ($n = 11$)
Age (months)			
Mean	112	108	106
SD	10.4	13.5	14.4
Range	93–125	93–130	85–125
Reading age (months)			
Mean	133	91	88
SD	27.8	16.3	15.4
Range	86–173	72–118	65–111
IQ (B.A.S.)			
Mean	112	110	116
SD	16.9	14.0	16.6
Range	78–137	92–136	88–149

Analysis of variance with Scheffe multiple comparisons failed to show any significant effects of group (i.e. Normal, Dunlop Test pass and Dunlop Test fail) on age and IQ.

convergence). Summary data for all these measures are shown in Table 2.

In the Dunlop Test children viewed two almost identical macular size fusion scenes through a synoptophore. The slide viewed by the right eye had a house with an arrowheaded post to the left of the front door, while the left eye saw a house with a post with a circle on top to the right of the front door. The angle of the synoptophore tubes was adjusted until children fused the two scenes. Then the tester abducted the synoptophore tubes (at 1.5 deg/s), and the children attempted to diverge their eyes to maintain fusion. When children understood clearly what they had to do, most gained a clear impression that one of the posts moved towards the door during this procedure. After about 5 deg divergence, diplopia intervened. The test was repeated 10 times, the slides being changed over frequently to try to prevent children guessing.

Table 2. Results of orthoptic assessment of children taking part in Expt 1

Subjects		SN R	SN L	CON (cm)	ACC (cm)	RAN (s)
Normals	Mean	4.36	4.27	6.00	6.73	35.9
($n = 11$)	SE	0.20	0.19	0.00	0.56	8.06
Dunlop Test passed	Mean	4.37	4.37	6.00	6.83	23.3
($n = 11$)	SE	0.19	0.19	0.00	0.30	2.56
Dunlop Test failed	Mean	4.62	4.54	6.17	7.00	37.1
($n = 11$)	SE	0.24	0.21	0.17	0.39	4.71

SN R and SN L, denominators of Snellen acuity for left and right eyes; CON, near point of convergence; ACC, near point of binocular accommodation; RAN, stereoacuity.

Analysis of variance with Scheffe multiple comparisons failed to show any significant effects of group (i.e. Normal, Dunlop Test pass and Dunlop Test fail) on any of these measures.

In order to categorize children as having "stable" or "unstable" ocular dominance, we treated the Dunlop Test as a pass/fail visual task (see Stein and Fowler, 1982). Children passed the test if they saw the post move on the same side in eight or more trials out of 10, i.e. they had a stable response to the test. Otherwise they failed the test, and were said to have an unstable response.

Children's IQs were measured with the B.A.S. They were calculated from the mean of the matrices and similarities subtest T-scores.

Apparatus. Children viewed a Joyce Electronics CRT display from a distance of 1.5 m, through a set of neutral density filters, with natural pupils and without a fixation target. With this arrangement screen luminance averaged 3.8 cd/m^2. The mean room luminance was 2.7 cd/m^2. The edges of the CRT were masked with dark card, so that only a central, circular portion of the screen, subtending 8 deg, was visible. The CRT's screen refresh rate was set at 200 Hz.

A signal generator was used to present sinusoidal grating patterns of 0.5, 1.5, 3 and 6 c/deg. Stimuli were sinusoidally modulated in counterphase at 20 Hz for the flicker contrast measurements. In both experiments, the tester pressed a button to trigger an electronic timer which in turn allowed the grating patterns to appear for exactly 1000 ms. Therefore, children saw a grey field, then a grating pattern with the same mean luminance, and then a grey field again. Stimulus onset and offset had a square-wave profile. Grating contrast was modulated manually by means of a signal attenuation unit. The smallest attenuation step which this unit could make was 1 dB.

Procedure. To identify childrens' thresholds, contrast was systematically reduced by means of a staircase procedure (cf. Levitt, 1971). At each point on the staircase, a grating appeared in one of four randomly selected orientations (horizontal, vertical, 45 deg upwards to the left and 45 deg upwards to the right); children had to decide which orientation they thought they had seen. In a pilot study, we found that the smallest change in contrast that children could detect reliably was 3 dB. So, each staircase was terminated two reversals after this step size was reached. We defined threshold as the average contrast at the last two reversals. The order in which spatial frequencies were presented was always the same (0.5, 1.5, 3.0 and 6.0 c/deg). Although this may have generated a learning bias, we assumed that any effect would have been the same for each comparison group.

Results and discussion

Figure 1 shows mean contrast sensitivity plotted against spatial frequency, where sensitivity is the reciprocal of threshold. In Fig. 1(a) the data from the normal primary school children ($n = 11$) are compared with the pooled data from both groups of SRD children ($n = 22$). The flicker contrast sensitivities of our normal control subjects were very similar to those measured by Robson (1966), who asked adult subjects to view grating stimuli modulated at 22 Hz. This suggests that our measuring technique was reasonably reliable. By comparison, we found that the sensitivity of SRDs at all spatial frequencies tested was reduced by about 0.25 log units. Analysis of variance, including interactions, confirmed that the main effects of spatial frequency and group (i.e. normals vs SRDs) were significant ($F_{3,124} = 58.6$, $P < 0.0005$; and $F_{1,124} = 66.0$, $P < 0.0005$, respectively). Thus we repeated Martin and Lovegrove's (1987) finding that SRDs show reduced flicker contrast sensitivity over this range of spatial frequencies, when they are compared with age matched normals.

Figure 1(b) extends this result by subdividing the SRD children according to their performance in the Dunlop Test. Thus SRD children who failed the Dunlop Test had significantly lower (*c.* 0.15 log units) flicker contrast sensitivity at all spatial frequencies than those who passed the Dunlop Test. Analysis of variance, including interactions, showed that both main effects of spatial frequency and group (i.e. normals, SRDs who passed the Dunlop Test and SRDs who failed the Dunlop Test) were significant ($F_{3,120} = 64.2$, $P < 0.0005$; and $F_{2,120} = 43.9$, $P < 0.0005$, respectively). No significant interaction between spatial frequency and group was found ($F_{6,120} = 0.21$, $P > 0.9$). Finally, Scheffe multiple comparisons confirmed that the mean contrast sensitivity of each of the three groups (pooling data across spatial frequencies) was significantly different from the other two, where each comparison exceeded the critical values $\alpha = 0.005$ and $F_{2,120} = 5.54$.

Figure 1(c) shows the contrast sensitivities of the three groups for static stimuli. Unlike the flicker data, the static contrast sensitivity curves do not lie one under the other in parallel.

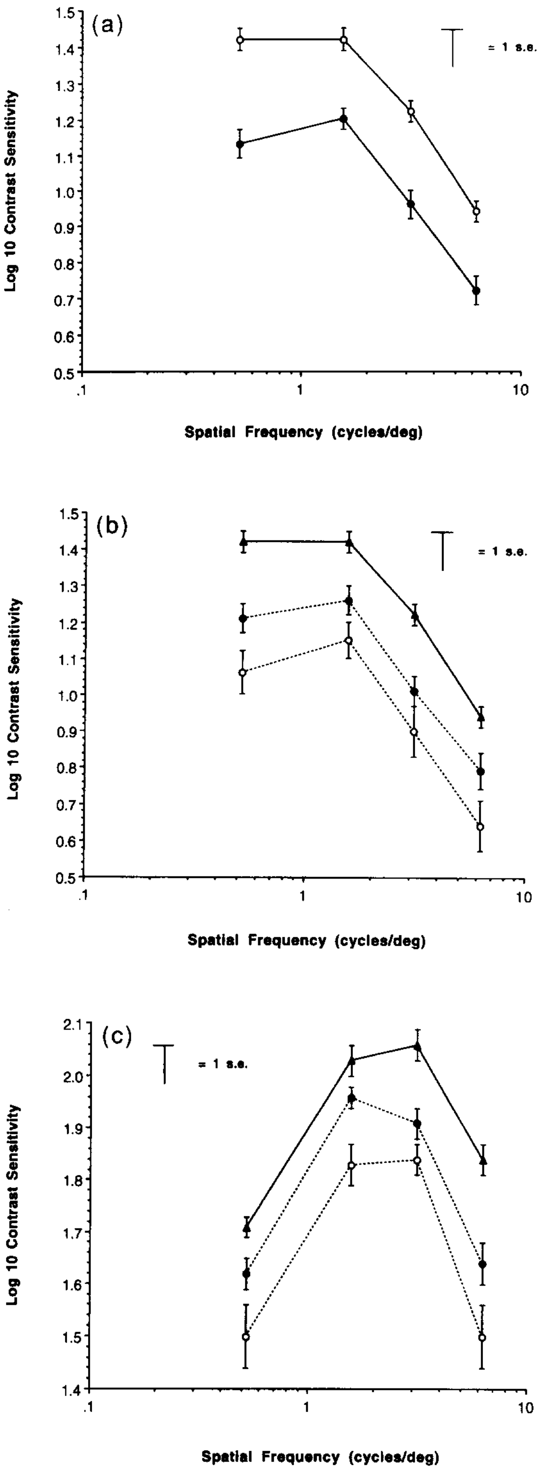

Fig. 1. (a) Plot of flicker contrast sensitivity against spatial frequency. Open circles represent normal children ($n = 11$), solid circles represent SRDs ($n = 22$). (b) Plot of flicker contrast sensitivity against spatial frequency. Normals, solid triangles with solid line. SRDs who passed the DT, solid circles with dashed line. SRDs who failed the DT, open circles with dashed line. (c) Plot of static contrast sensitivity against spatial frequency. Normals, solid triangles with solid line. SRDs who passed the DT, solid circles with dashed line. SRDs who failed the DT, open circles with dashed line.

Instead, the sensitivity difference between normals and SRDs who passed the Dunlop Test was 0.09 log units at 0.5 c/deg compared with 0.20 log units at 6.0 c/deg. However, analysis of variance comparing just these two groups (i.e. normals and SRDS who passed the Dunlop Test) did not show a significant interaction between spatial frequency and group ($F_{3,87} = 1.99$, $P = 0.12$). We made two other comparisons; SRDs who passed vs SRDs who failed the Dunlop Test and SRDs who failed the Dunlop Test vs normals. Neither comparison revealed significant interactions between Dunlop Test and spatial frequency.

In the Introduction to this paper, we described how two kinds of experiment have shown differences in the way SRDs process visual information when they are compared with age matched normal controls. Until now, there has been no suggestion that these two approaches might be associated. But we have found that the reduction in flicker and static contrast sensitivity in reading disabled children depended on whether they had stable or unstable ocular dominance. This suggests that these contrast sensitivity measures and the Dunlop Test are indeed related in reading disabled children.

EXPERIMENT 2

It is one thing to show that SRD children exhibit visual processing differences when they are compared with age matched normals, but it is quite another to determine whether these differences may affect the way children read. In their present form, the results from Expt 1 represent little more than a physiological correlate of reading difficulty (Seymour, 1986). They can not address the issue of causality.

In principle, to show that some aspect of visual processing may affect reading, it is necessary to compare groups of children with and without a visual problem who are at least matched for reading age (Bryant and Bradley, 1985). This ensures that any group differences in visual performance can not be attributed to systematic differences in reading experience. Therefore, in Expt 2, we compared the flicker contrast sensitivities of children who failed the Dunlop Test with children who passed the Dunlop Test, and who were matched as closely as possible for age, reading age and IQ.

Methods

Subjects. Subjects were drawn from the clinical population referred to the same orthoptic clinic in Reading. But this time we did not attempt to discriminate between SRDs, normal readers and children who were poor readers because of generally low ability. Instead we only divided children according to their performance on the Dunlop Test, taking care to match the two groups of children as closely as possible for age, IQ and reading age. Thus we made two comparisons. In the first, we measured flicker contrast sensitivity of 15 children who passed and 15 children who failed the Dunlop Test. In the second, we measured static contrast sensitivity in 34 children who passed and 30 children who failed the Dunlop Test. The two pairs of comparison groups were of mixed abilities as summarized in Table 3 below.

Table 3. Age, reading age and IQ for children taking part in Expt 2

	Flicker		Static	
	Dunlop Test pass ($n = 15$)	Dunlop Test fail ($n = 15$)	Dunlop Test pass ($n = 34$)	Dunlop Test fail ($n = 30$)
Age (months)				
Mean	109	107	109	109
SD	20.4	14.7	20.4	20.0
Range	79–148	85–125	73–159	73–147
Reading age (months)				
Mean	92	97	90	92
SD	17.2	27.2	16.4	18.6
Range	70–118	65–168	60–126	60–136
IQ (B.A.S.)				
Mean	108	115	109	109
SD	16.6	15.1	14.4	14.1
Range	72–136	88–149	78–137	73–147

Analysis of variance failed to show significant effects of Dunlop Test on age, IQ and reading age in both groups of children.

Table 4. Results of orthoptic assessment of children taking part in Expt 2

Subjects		SNR	SNL	CON (cm)	ACC (cm)	RAN (s)
Static contrast sensitivity						
Dunlop Test passed	Mean	4.71	4.67	6.00	7.26	27.1
($n = 34$)	SE	0.27	0.27	0.00	0.23	1.1
Dunlop Test failed	Mean	4.71	4.70	6.23	7.35	36.9
($n = 30$)	SE	0.21	0.21	0.11	0.23	1.1
Flicker contrast sensitivity						
Dunlop Test passed	Mean	4.53	4.37	6.00	6.93	23.6
($n = 15$)	SE	0.17	0.16	0.00	0.33	1.1
Dunlop Test failed	Mean	4.67	4.60	6.27	7.07	37.7
($n = 15$)	SE	0.21	0.18	0.18	0.33	1.1

SN R and SN L, the denominators of the Snellen acuities for left and right eyes; CON, near point of convergence; ACC, near point of binocular accommodation; RAN, stereoacuity.

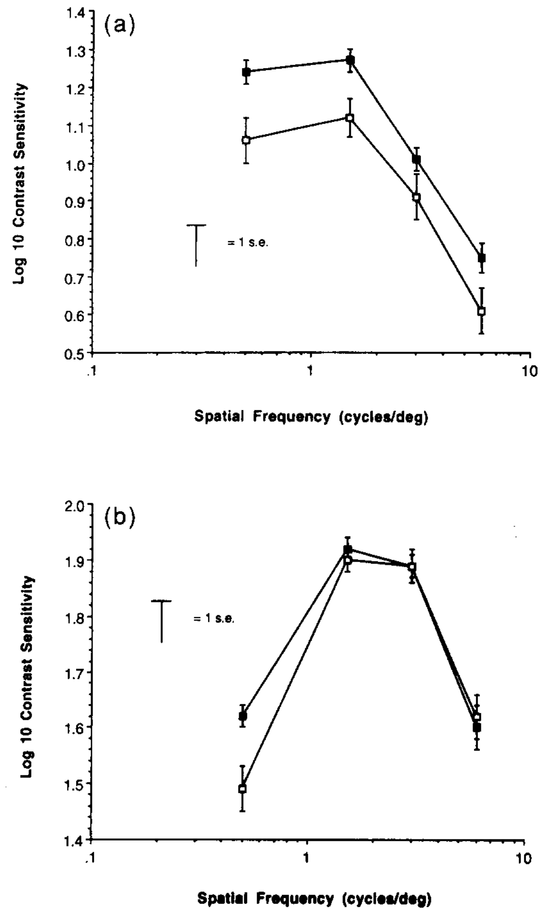

Fig. 2. (a) Plot of flicker contrast sensitivity against spatial frequency. Mixed ability children who passed the DT, solid squares. Mixed ability children who failed the DT, open squares. (b) Plot of static contrast sensitivity against spatial frequency. Mixed ability children who passed the DT, solid squares and solid line. Mixed ability children who failed the DT, open squares and dashed line.

Procedure. The apparatus, clinical assessment and method of contrast sensitivity measurement were identical to Expt 1. Table 4 summarizes the orthoptic findings of the children tested in Expt 2.

Results and discussion

In Fig. 2(a), mean flicker contrast sensitivity is plotted against spatial frequency. As in Expt 1, the children who failed the Dunlop Test showed reduced (*c.* 0.15 log units) contrast sensitivity for flickering gratings at all spatial frequencies tested. Analysis of variance confirmed that the main effects of spatial frequency and Dunlop Test were significant ($F_{3,115} = 48.9$, $P < 0.0005$; and $F_{1,115} = 18.1$, $P < 0.0005$, respectively).

Figure 2(b) shows the static contrast sensitivities of 34 children who passed and 30 children who failed the Dunlop Test. The most noticeable effect is that children who failed the test showed a small reduction in sensitivity at 0.5 c/deg (0.13 log units). Analysis of variance revealed a significant interaction between spatial frequency and Dunlop Test ($F_{3,242} = 2.95$, $P < 0.05$).

Recently, Cornelissen *et al.* (1991, 1992) used a design in which children were matched for both reading age and chronological age to compare reading performance in children who passed and failed the Dunlop Test. In both studies, only children who failed the Dunlop Test read differently if print size was reduced, or if they were asked to read with both eyes as opposed to one; more of their reading errors became nonwords (neologisms). In these experiments, the only factor which differentiated the groups was their performance on a visual task (the Dunlop Test), and the only factor which was manipulated during the experiments was the visual appearance of text. Therefore, the change in the pattern of children's reading errors must have occurred for visual reasons alone, supporting the idea that unstable ocular dominance may directly affect how children read.

In Expt 2 we also controlled for age, reading age and IQ and found that Dunlop Test failure still correlated with reduced flicker contrast sensitivity. Therefore the present findings add further support for the idea that differences in visual performance may causally influence the way children read.

GENERAL DISCUSSION

Like Lovegrove *et al.* the most impressive effects we have found are in the temporal domain; not only did SRDs show lower flicker contrast sensitivity than normals but the size of the reduction depended on the outcome of their Dunlop Test performance. Moreover these findings were upheld even when age, reading age and IQ were accounted for. One peculiar feature of these results is the way that the flicker contrast sensitivity curves lie one under the other, almost in parallel. This might suggest that attentional factors alone could explain the group differences that we found. However, in Expt 1, the difference in mean static contrast sensitivity between normals and SRDs was greater at 6.0 than at 0.5 c/deg. Furthermore, in Expt 2, the difference in static contrast sensitivity between children who passed or failed the Dunlop Test was restricted to 0.5 c/deg. These assymetries suggest that the flicker contrast findings are unlikely to be due to attentional factors alone.

Unlike Lovegrove *et al.*, in Expt 1 we found that SRDs were less sensitive to static gratings than normals at all spatial frequencies, though the difference was most marked at 6.0 c/deg. This high spatial frequency loss is unlikely to be due to optical blurring since we did not find systematic differences in Snellen acuity or near point of accommodation when SRDs and normals were compared (see Table 2). However, this effect could be explained if SRDs experienced more fixational instability (Dickinson and Abadi, 1985).

What are the implications of reduced sensitivity to flickering gratings? Kulikowski described how grating stimuli of $< c.$ 20 c/deg, counter-phased at temporal frequencies $< c.$ 8 Hz appear to move or drift (Kulikowski, 1971), thereby suggesting an inter-relationship between motion processing and temporal modulation of a stimulus. Outside this large range of spatiotemporal conditions, stimuli no longer appear to drift. Other psychophysical evidence suggests that motion detection is carried out by two (Kulikowski and Tolhurst, 1973; Holliday and Ruddock, 1983) or possibly three channels (Hess and Snowden, 1992). For example, Anderson and Burr (1989) used a masking paradigm to measure the spatial frequency, orientation and temporal frequency selectivity of motion detector units in human vision. Their data could be accounted for with only two classes of motion detector; one with band-pass characteristics, peaking at 8 Hz, and the other low-pass, extending to about 8 Hz before attenuation.

The neurophysiology of flicker and movement detection have been extensively studied in the macaque, whose visual system is analogous to that of humans (Derrington and Lennie, 1984; Merigan and Eskin, 1986; Shapley and Perry, 1986; Merigan and Maunsell, 1990). For example, Merigan and Maunsell (1986) chemically ablated the magnocellular portion of the lateral geniculate nuclei (LGN) of macaques. Three threshold measures were clearly disrupted by the magnocellular lesions. Contrast sensitivities for a 1 c/deg grating which drifted at 10 Hz and for 10 Hz counterphase modulated 1 c/deg gratings were reduced, though the effect was twice as large for the drifting stimulus. Sensitivity to a low spatial frequency Gaussian blob, flickering at 10 Hz was virtually abolished. In addition, critical fusion frequency was greatly reduced. By comparison, contrast sensitivity for static 2 c/deg gratings remained unaffected by the magnocellular lesions. These findings are in accord with the study conducted by Schiller *et al.* (1990) who also showed that magnocellular, but not parvocellular lesions dramatically reduced movement detection and discrimination thresholds, assessed with random-dot displays.

Altogether, the results from human psychophysical experiments suggest that flicker sensitivity is related to motion detection and that there are probably two classes of motion detector. Furthermore, lesion studies suggest that the magnocellular system provides important input for flicker and movement detection. However, recent anatomical and electrophysiological evidence suggests that magno- and parvocellular input may converge upon the same cells even as early as V1 (Maunsell *et al.*, 1992; Casagrande and Lachica, 1992). Certainly, there is known to be a high degree of interconnectivity in the extra-striate visual areas beyond V2 (Douglas and Martin, 1991). Therefore, since psychophysical measurements represent the consequence of activity throughout large parts of the visual system, it is not clear what relative contributions the magno- and parvocellular systems might make to psychophysically defined motion channels. Nevertheless, it seems likely that flicker contrast sensitivity and motion detection are intimately linked with magnocellular function.

In the experiments reported in this paper, we found that children with SRD showed reduced sensitivity to flickering stimuli, especially if they also had unstable ocular dominance. In the light of psychophysical and neurophysiological findings, it is plausible that unstable ocular dominance and reduced flicker contrast sensitivity in SRDs could be related to abnormal magnocellular function and/or abnormal motion detection. Indeed this conclusion was reached by Livingstone *et al.* in a recent paper (Livingstone *et al.*, 1991). These authors measured visually evoked potentials in SRD and normal adult subjects. They used high and low contrast checkerboard stimuli of high and low spatial frequencies which were contrast reversed at 0.5 Hz. The earliest negative component in the VEP at low spatial frequencies and low contrast has been attributed to activity in the magnocellular pathway (Jones and Keck, 1978). This component was missing in all SRD subjects, consistent with magnocellular dysfunction. The electrophysiological results were complemented by post-mortem anatomical comparisons of the lateral geniculate nuclei of SRD and normal brains. Whereas the dorsal, parvocellular layers appeared normal in both SRDs and normals, the ventral magnocellular layers of the SRD brains showed a preponderence of abnormally small cell bodies.

In conclusion, we have found evidence that reduced flicker contrast sensitivity and unstable ocular dominance may be related in SRD children. However, the physiological mechanisms underlying this association remain to be explored.

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REFERENCES

Anderson S. J. and Burr D. C. (1989) Receptive field properties of human motion detector units inferred from spatial frequency masking. *Vision Res.* **29,** 1343–1358.

Bigelow E. R. and McKenzie B. E. (1985) Unstable ocular dominance and reading ability. *Perception* **14,** 329–335.

Bishop D. V. M., Jancey C. and Steel A. McP. (1979) Orthoptic status and reading disability. *Cortex* **15,** 659–666.

Bowling A. and Lovegrove W. (1980) The effect of stimulus duration on the persistence of gratings. *Percept. Psychophys.* **27,** 574–578.

Breitmeyer B. G., Levi D. M. and Harweth R. S. (1981) Flicker masking in spatial vision. *Vision Res.* **21,** 1377–1385.

Bryant P. E. and Bradley L. (1985) *Childrens Reading Problems.* Blackwell, Oxford.

Cambell F. W. and Robson J. G. (1968) Application of Fourier analysis to the visibility of gratings. *J. Physiol.* **197,** 551–566.

Casagrande V. A. and Lachica E. A. (1992) What are cytochrome oxidase blobs and interblobs really segregating. *Invest. Ophthal. visual Sci.* **33,** 900.

Cornelissen P. L., Bradley L., Fowler M. S. and Stein J. F. (1991) What children see affects how they read. *Devl Med. Child Neurol.* **33,** 755–762.

Cornelissen P. L., Bradley L., Fowler M. S. and Stein J. F. (1992) Covering on eye affects how some children read. *Devl Med. and Child Neurol.* **34,** 296–304.

Derrington A. M. and Lennie P. (1984) Spatial and temporal contrast sensitivities of neurones in lateral geniculate nucleus of macaque. *J. Physiol.* **357,** 219–240.

Dickinson C. M. and Abadi R. V. (1985) The influence of nystagmoid oscillation on contrast sensitivity in normal observers. *Vision Res.* **25,** 1089–1096.

Douglas R. J. and Martin K. A. C. (1991) Opening the grey box. *Trends Neurosci.* **14,** 286–293.

Hess R. F. and Snowden R. J. (1992) Temporal properties of human visual filters: number shapes and spatial covariation. *Vision Res.* **32,** 47–59.

Holliday I. E. and Ruddock K. H. (1983) Two spatio-temporal filters in human vision—I. Temporal and spatial frequency response characteristics. *Biol. Cybernet.* **47,** 173–190.

Jones R. and Keck M. J. (1978) *Invest. Ophthal. visual Sci.* **17,** 652–659.

Kulikowski J. J. (1971) Effect of eye movements on the contrast sensitivity of spatio-temporal patterns. *Vision Res.* **11,** 261–273.

Kulikowski J. J. and Tolhurst D. J. (1973) Psychophysical evidence for sustained and transient detectors in human vision. *J. Physiol.* **232,** 149–162.

Levitt H. (1971) Transformed up-down methods in psychoacoustics. *J. acoust. Soc. Am.* **49,** 467–477.

Livingstone M. S., Rosen G. D., Drislane F. W. and Galaburda A. (1991) Physiological and anatomical evidence for a magnocellular deficit in developmental dyslexia. *Proc. natn. Acad. Sci. U.S.A.* **88,** 7943–7947.

Lovegrove W. J., Garzia R. P. and Nicholson S. B. (1990) Experimental evidence for a transient system deficit in specific reading disability. *J. Am. Optom. Assoc.* **61,** 137–145.

Lovegrove W. J., Martin F. and Slaghuis W. (1986) A theoretical experimental case for a visual deficit in specific reading difficulty. *Cognit. Neuropsychol.* **3,** 225–267.

Lovegrove W. J., Martin F., Bowling A., Blackwood M., Badcock D. and Paxton S. (1982) Contrast sensitivity functions and specific reading disability. *Neuropsychologia* **20,** 309–315.

Martin F. and Lovegrove W. (1984) The effects of field size and luminance on contrast sensitivity differences between specifically reading disabled and normal children. *Neuropsychologia* **22,** 73–77.

Martin F. and Lovegrove W. (1987) Flicker contrast sensitivity in normal and specifically disabled readers. *Perception* **16,** 215–221.

Masters M. C. (1988) Orthoptic management of visual dyslexia. *Br. orthop. J.* **45,** 40–48.

Maunsell J. H. R., Nealey T. A. and Ferrera V. P. (1992) Magnocellular and parvocellular contributions to neuronal responses in monkey visual cortex. *Invest. Ophthal. visual Sci.* **33,** 901.

Merigan W. H. and Eskin T. A. (1986) Spatio-temporal vision of macaques with severe loss of Pβ retinal ganglion cells. *Vision Res.* **26,** 1751–1761.

Merigan W. H. and Maunsell J. H. R. (1990) Macaque vision after magnocellular lateral geniculate lesions. *Visual Neurosci.* **5,** 347–352.

Newman S. P., Karle H., Wadsworth J. F., Archer R., Hockly R. and Rogers P. (1985) Ocular dominance reading and spelling: a reassessment of a measure associated with specific reading difficulties. *J. Res. Reading* **8,** 127–138.

Robson J. G. (1966) Spatial and temporal contrast sensitivity function of the visual system. *J. opt. Soc. Am.* **56,** 1141–1142.

Rutter M. and Yule W. (1975) The concept of specific reading retardation. *J. child Psychol. Psychiat.* **16,** 181–197.

Schiller P. H., Logothetis N. K. and Charles E. R. (1990) Role of the color-opponent and broad-band channels in vision. *Visual Neurosci.* **5,** 321–346.

Seymour P. H. K. (1986) *Cognitive Analysis of Dyslexia.* Routledge (& Kegan Paul), London.

Shapley R. and Perry V. H. (1986) Cat and monkey retinal ganglion cells and their functional roles. *Trends Neurosci.* **9,** 229–235.

Stein J. F. (1991) Visuospatial sense hemispheric asymmetry and dyslexia. In *Vision and Visual Dysfunction, Vol.* 13. *Vision and Visual Dyslexia,* edited by Stein J. F. Macmillan, London.

Stein J. F. and Fowler M. S. (1981) Visual dyslexia. *Trends Neurosci.* **4,** 77–80.

Stein J. F. and Fowler M. S. (1982) Diagnosis of dyslexia by means of a new indicator of eye dominance. *Br. J. Ophthal.* **66,** 332–336.

Stein J. F. and Fowler M. S. (1985) Effect of monocular occlusion on visuomotor perception and reading in dyslexic children. *Lancet* **ii,** 69–73.

Stein J. F., Riddell P. M. and Fowler M. S. (1986) The Dunlop Test and reading in primary school children. *Br. J. Ophthal.* **70,** 317–320.

Tolhurst D. J. (1975) Reaction times in the detection of gratings by observers: a probabilistic mechanism. *Vision Res.* **15,** 1143–1149.