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Attenuated hemispheric lateralization in dyslexia: evidence of a visual processing deficit

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THERE is controversial evidence that deficits in the processing of low contrast and low spatial frequency stimuli are of importance in the pathogenesis of dyslexia. Fifteen adult dyslexics and 19 controls were examined using visual evoked potentials (VEP) at varying spatial frequencies (2 and 11.33 cpd) and contrasts (0.2, 0.4, 0.6, 0.8). Our results show that the amplitude of VEPs following different spatial frequencies and contrasts did not differentiate between dyslexics and controls. Further, we found significantly higher amplitudes of the P1 and P2 over the right occipital cortex. For the P2, this hemispheric asymmetry was not found in the dyslexic group suggesting a specific low level visual processing deficit in the right occipital region in dyslexia. *NeuroReport* 10:3697–3701 © 1999 Lippincott Williams & Wilkins.

Key words: Dyslexia; Hemispheric asymmetry; Magnocellular and parvocellular visual system; P1; P2; Visual evoked potentials

Introduction

Dyslexia is a disorder resulting from a developmental impairment in the ability to read and spell despite adequate educational resources, a normal IQ, no obvious sensory deficits and adequate sociocultural opportunity [1]. Research into the aetiology of dyslexia has looked at visual [2] and auditory processing functions [3], and recent linkage analysis has begun to delineate the genetic basis of dyslexia [4].

Visual processing is currently seen as comprising two separate but interactive subsystems with different spatiotemporal response characteristics [5]. The magnocellular system, which arises from cells widely distributed across the retina, projects via the ventral lateral geniculate nucleus (LGN) to the visual cortex and thereafter largely to the parietal cortex. It preferentially mediates movement, fast temporal resolution, low contrast, and low spatial frequencies. The parvocellular system originates in cells concentrated in the fovea and projects via the dorsal LGN to the visual cortex and then mainly to the temporal cortex. It is responsible for colour resolution, high contrast and high spatial frequencies [5].

Considerable evidence has been put forward in favour of the magnocellular deficit theory in dyslexia [2]. Contradictory findings, however, have resulted in continuing debate as to its role in the pathogenesis of dyslexia. According to the magnocellular system deficit theory, the processing of low spatial frequency and low contrast stimuli presented with high temporal frequency is disturbed in dyslexics. This theory could be confirmed by psychophysiological and neurophysiological studies. Lovegrove *et al.* [6] found reduced contrast sensitivity at low spatial frequencies. This result could recently be confirmed by Slaghuis and Ryan [7]. A number of visual evoked potential (VEP) studies have provided further evidence of a magnocellular dysfunction in dyslexics [8,9]. For instance, Livingstone *et al.* [8] and Lehmkuhle *et al.* [9] found smaller VEPs following low spatial frequency stimuli at high temporal frequencies and low contrast.

However, some studies have yielded incompatible results with the magnocellular deficit theory [10–12]. The findings that dyslexics have a reduced contrast sensitivity only at high spatial frequencies [10] and that the P1 latency is significantly greater at high contrasts [11] suggest a parvocellular deficit rather than either a magnocellular or the absence of a magnocellular deficit. The functions of the magnocellular and parvocellular systems have been shown to have a lateralized representation [13]. Psychophysiological investigations suggest that the magnocellular system projects preferentially to the right hemisphere [14]. Evoked potential studies in normal subjects have also suggested a functional asymmetry of the visual cortex [14,15]. The right occipital cortex appears to be more sensitive to the magnocellular functions of low spatial frequency and fast temporal resolution [13]. Furthermore, lateralization

has been demonstrated with neuropsychological paradigms, e.g. the right hemisphere preferentially undertakes tasks such as processing of patterns and specific shape information, and word recognition [16,17].

Given the presence of visual hemispheric asymmetries in normal readers, it seems promising to assess the VEPs of dyslexics with a visual processing paradigm in order to clarify the role of a visual processing deficit in dyslexia. Until now, no studies have looked at visual processing hemispheric differences in dyslexics, VEP studies on dyslexics have only been examined at central electrode positions such as Oz or Cz [8,9,11,12].

In this study, we have investigated VEPs at a number of lateralised electrode positions over a range of contrasts and spatial frequencies to test both magno- and parvocellular systems, in order to investigate hemispheric asymmetry in a group of adult dyslexics and controls. The hypothesis is that the amplitude of the early components of the VEP following low contrast and low spatial frequency stimuli in dyslexics as compared to controls is attenuated. Since magnocellular functions are represented over the right hemisphere, group differences between dyslexics and controls should mainly occur over the right occipital region.

Materials and Methods

Thirty-four adults (15 dyslexics, all male, mean age 25.9 ± 4.2 years) and 19 controls (14 male, five female, mean age 22.3 ± 6.6) participated in the study. The two groups did not differ with regard to their IQs (mean IQ of spelling disabled was 123.2 ± 8.8; IQ of controls was 117.5 ± 14.2). The dyslexics had either completed or were from the final class of a boarding school for dyslexics and were selected as a result of their continuing spelling disability. Spelling disability was defined by the presence of a discrepancy of $\geqslant 1$ s.d. between actual spelling and expected spelling based on IQ [18]. The spelling disabled group also had a significantly lower word decoding ability in comparison to the controls, with significant differences between the groups on both reading test scores: word reading accuracy (one sided *t*-test, $p = 0.0046$) and reading speed ($p = 0.0003$). The control group were undergraduate psychology students. The spelling ability of the control group was in the normal range.

Inclusion criteria were to be a native monolingual German speaker, to have normal corrected visual acuity and no hearing problems, with no neurological, emotional or behavioural deficits or unusual educational circumstances that could account for poor reading and spelling ability. All subjects were strongly right-handed according to a self-report handedness questionnaire [3].

Subjects sat in a darkened room (average luminance 1.2 cd/m^2) at a 60 cm viewing distance to an EIZO 21 computer monitor. The visual stimuli presented consisted of sine wave vertical gratings in a circle on a dark background at a 3° visual angle. Background luminance was 2 cd/m^2 and grating luminance was 20 cd/m^2. Eight separate conditions, comprised of the combination of two spatial frequencies (2 and 11.33 cycles per degree of visual angle, cpd) and four contrast levels (0.2, 0.4, 0.6 and 0.8), were presented in random order. These contrasts and frequencies were chosen to optimise the chance of differentiating between dyslexics and normals and are similar to those used by other researchers [6]. Three hundred gratings were presented under each condition, for 200 ms each with an interstimulus interval of 600 ms.

Electrodes were placed at 19 scalp sites based on the International 10-20 System: Fp1, Fp2, F7, F8, F3, F4, Fz, C3, C4, Cz, T3, T4, T5, T6, P3, P4, Pz, O1, O2 (referred to linked ears, ground electrode at Fpz). Eye movements and blinks were monitored by two electrodes placed below the subjects' right and left eyes and the Fp1 and Fp2 electrodes. The EEG was amplified with Schwarzer amplifiers, time constant 0.6 s; upper frequency cut-off 85 Hz. The EEG was recorded continuously, A/D converted at a sampling rate of 172 Hz and transferred for further analysis to a DEC Alpha computer. The signals were averaged into epochs of 750 ms, including a prestimulus baseline of 50 ms. Epochs with artefacts were excluded from averaging. Peak amplitudes of the components were measured with an event related potential parameter programme developed at our Institute. The peak amplitudes of the VEPs were assessed separately for the P1 and P2 components, which are the first and second visible positivity after stimulus presentation (see Fig. 1).

Results

Two MANOVAs were carried out for the P1 and P2 amplitudes, respectively. Four factors were analysed: group (dyslexics *vs* controls), lateralization (O1 *vs* O2), spatial frequency (2 *vs* 11.33 cpd) and contrast (0.2, 0.4, 0.6, 0.8).

The MANOVA for the P1 amplitude (see Table 1) yielded three significant effects. The main effect spatial frequency (higher amplitude at the low spatial frequency), the main effect lateralization (higher amplitude over the right hemisphere) and the interaction between lateralization and spatial frequency were significant (the spatial frequency effect being larger for right occipital cortex). The

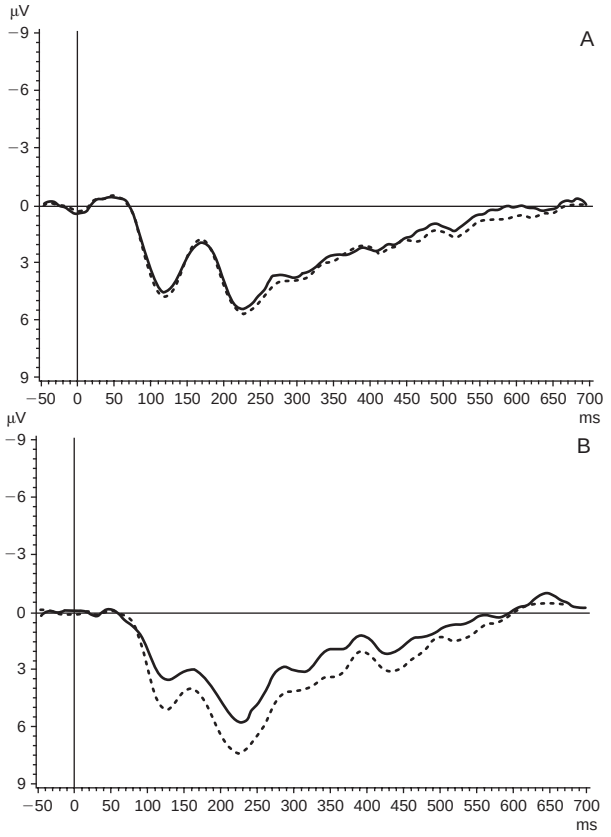

FIG. 1. A typical grand average for dyslexic (**A**) and control (**B**) groups. The VEPs were elicited with a grating of 11.33 cpd and a contrast level of 0.6. at electrode positions O1 (left, solid line) and O2 (right, dotted line). It is not possible for reasons of space to show all conditions.

Table 1. MANOVA results for the P1

Effect	F value	*p* value
Lateralization	7.76	0.0089
Spatial frequency	7.18	0.0115
Lateralization × spatial frequency	5.35	0.0273

MANOVA for the P2 potential yielded five significant effects (Table 2). Three of the four main effects were significant: lateralization (amplitude larger over right occipital cortex), spatial frequency (amplitude larger for low spatial frequency), and contrast (amplitude larger for low contrast). The interaction between lateralization and spatial frequency was in the same direction as the respective interaction for P1, i.e. the spatial frequency effect was larger on the right than the left hemisphere. Thus for both the P1 and the P2 the amplitudes elicited by a low spatial frequency stimulus were larger over the right occipital cortex. A significant interaction between lateralization and group was found. The dyslexic group failed to show this asymmetry which was clearly seen in the control group (Fig. 2).

Figure 3 shows two examples for the distributions

Table 2. MANOVA results for the P2

Effect	F value	*p* value
Lateralization	18.51	0.0001
Spatial frequency	12.47	0.0013
Contrast	5.03	0.0061
Group × lateralization	6.14	0.019
Lateralization × spatial frequency	9.87	0.0036

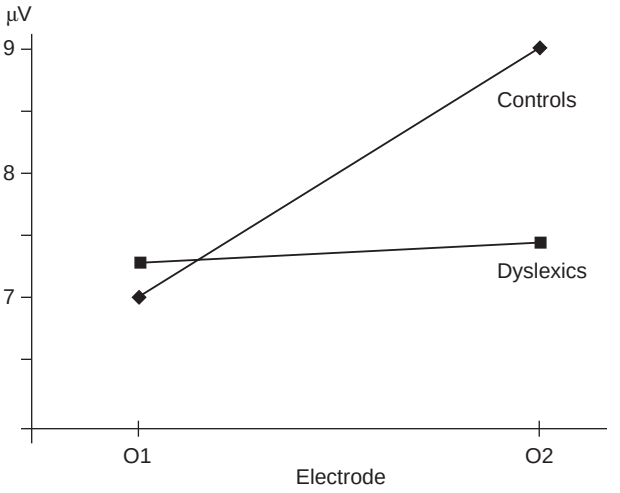

FIG. 2. P2 means for the interaction between group and lateralization.

of the brain activities. Brain maps for the other conditions look very similar. As shown in Fig. 3, the main positivity was found at occipital regions and that activation was higher over the right occipital region (O2) in the control group.

Discussion

We have investigated the influence of high and low spatial frequencies and contrasts on the VEP in dyslexics and controls. Previous researchers have looked at the VEP in dyslexics, but have not examined inter-hemispheric differences [8,9,11,12]. We have demonstrated lateralization of two components of the VEP, the P1 and P2. These positive components of the VEP with latencies of 100–250 ms reflect early perceptual processing and pattern recognition [19]. For all contrasts and spatial frequencies, activity in the right hemisphere was larger than in the left. This result suggests that the magno- and parvocellular processing of contrasts and spatial frequencies is preferentially located in the right hemisphere. In addition, the significant interaction of lateralization with spatial frequency demonstrates that this functional asymmetry is even greater for low spatial frequencies, which means that this magnocellular function (sensitivity to low spatial frequencies) is preferentially processed in the

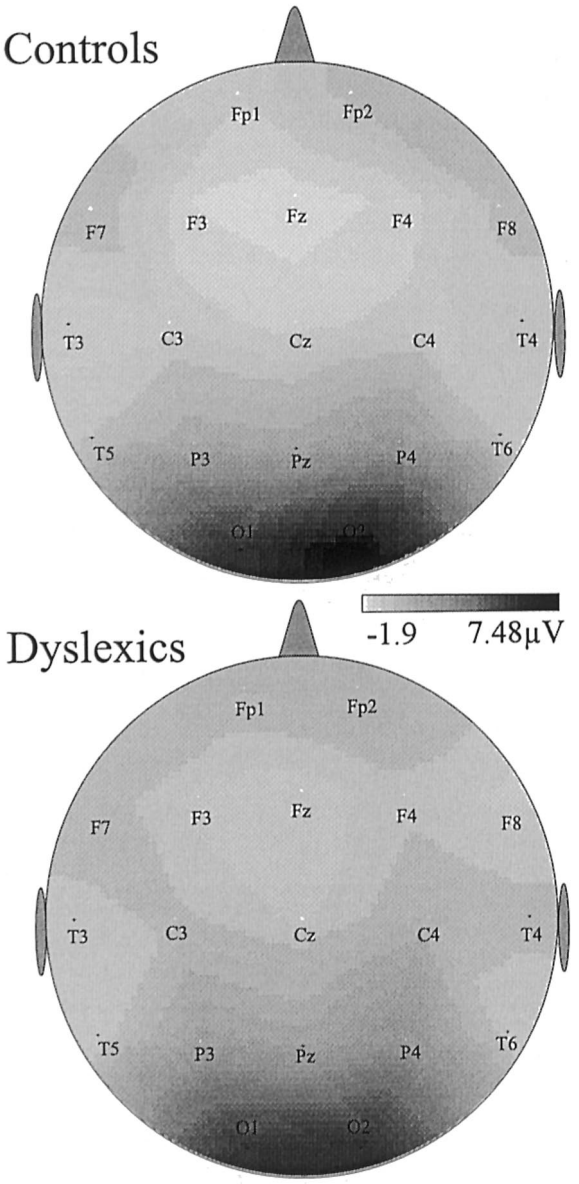

FIG. 3. Typical brain maps of the P2 for one condition (in this case spatial frequency = 11.3 cpd and contrast s = 0.6). It is not possible for reasons of space to show all conditions.

right hemisphere. These results confirm the results of Rebai *et al.* [13] that the hemispheres differ with regard to their sensitivity to the physical characteristics of visual stimuli. Recent work by Rebai *et al.* [15] found that the amplitude of an early component of the VEP (C1) elicited by sine wave gratings at different spatial frequencies was larger in the right hemisphere. However, while Rebai *et al.* [15] found the right hemisphere to be more responsive to high spatial frequencies, we found the reverse result. This discrepancy might have arisen as a result of the small sample size in the study of Rebai *et al.* ($n = 5$), or may result from the difference in luminance: while Rebai *et al.* used high high luminance, our study was performed at low luminance.

Our results show no influence of contrast and spatial frequency variations on VEP amplitudes of dyslexics. This is in line with the work of Victor *et al.* [12]. The fact that we found no significant difference between dyslexics and controls in terms of contrast sensitivity or spatial frequency did not support the magnocellular deficit theory in dyslexia.

To the best of our knowledge, this is the first demonstration of different VEP amplitudes in dyslexics and controls over the right occipital region. This finding supports the presence of a visual processing deficit in dyslexia, and in particular, a right sided deficit which seems to be independent from spatial frequency and contrast sensitivity. Evidence for a right hemisphere deficit in dyslexia has been reviewed by Stein [20]. Right sided functions like binocular control, visual localization and stereoacuity have been found to be impaired in dyslexics [20]. Furthermore, a failed lateralization in dyslexia has also been demonstrated in research using different methods. In a CT study, parieto-occipital cerebral asymmetry was found to be greater (larger on the right than the left) in dyslexic adults than normals [21]. In addition, an MRI study [22] found a positive correlation between the volume of the right occipital cortex and the severity of reading difficulties in dyslexia. This adds further weight to the argument that the right occipital cortex might be disrupted in dyslexics and strengthens the importance of the right occipitoparietal cortex for the pathogenesis in dyslexia. It has also been demonstrated that the right occipital cortex is preferentially involved in the global integration of visual stimuli [23,24], and we speculate that this could be an important mechanism in dyslexia, such that dyslexics have difficulties integrating visual components into meaningful letters and words. A corresponding deficit has been shown in the perceptual integration of non-lexical information in dyslexics [25] and it may be that an underlying deficit in the global integration of visual information occurs in dyslexia.

Conclusion

The importance of contrast sensitivity and spatial frequency has been always considered controversial in the pathogenesis of dyslexia. We have investigated the hypothesis that dyslexics have a magnocellular deficit and have found no evidence to support the view that processing of low contrast and low spatial frequency is specifically disturbed in dyslexics. Our finding of a selectively attenuated visual evoked potentials in dyslexics is the first electrophysiologi-

cal evidence of a right occipital visual processing deficit.

References

1. Dilling H Mombour W and Schmidt MH. *International Classification of Mental Diseases, ICD-10*. German edition. Bern: Huber, 1991.
2. Stein J and Walsh V. *Trends Neurosci* **20**, 147–52 (1997).
3. Schulte-Körne G, Deimel W, Bartling J *et al. NeuroReport* **9**, 337–340 (1998).
4. Schulte-Körne G, Grimm T, Nöthen MM *et al. Am J Hum Genet* **63**, 279–282 (1998).
5. Merigan WH and Maunsell JHR. *Annu Rev Neurosci* **16**, 369–402 (1993).
6. Lovegrove WJ, Bowling A, Badcock B *et al. Science* **210**, 439–440 (1980).
7. Slaghuis WL and Ryan JF. *Vision Res* **39**, 651–668 (1999).
8. Livingston MS, Rosen GD, Drislane FW *et al. Proc Natl Acad Sci* **88**, 7943–7947 (1991).
9. Lehmkuhle S, Garzia RP, Turner L *et al. N Engl J Med* **328**, 989–996 (1993).
10. Gross-Glenn K, Skottun BC, Glenn W *et al. Vis Neurosci* **12**, 153–163 (1995).
11. Brecelj J, Struel M and Raie V. *Eur J Physiol* **431**, R299–R300 (1996).
12. Victor JD, Conte MM, Burton L *et al. Vis Neurosci* **10**, 939–936 (1993).
13. Rebai M, Mecacci L, Bagot JD *et al. Neuropsychologia* **27**, 315–324 (1989).
14. Kosslyn SM, Chabris CF, Marsolek CJ *et al. Exp Psychol Hum Percept Perform* **18**, 562–577 (1992).
15. Rebai M, Bernard C, Lannou *et al. Brain Cog* **36**, 21–29 (1998).
16. Marsolek CJ, Kosslyn SM and Squire LR. *J Exp Psychol Learn Mem Cogn* **18**, 492–508 (1992).
17. Marsolek CJ, Schacter DL and Nicholas CD. *Mem Cogn* **24**, 539–556 (1996).
18. Schulte-Körne G, Deimel W, Müller K *et al. J Child Psychol Psychiat* **37**, 817–822 (1996).
19. Hillyard SA, Teder-Salejarvi WA and Münte TF. *Curr Opin Neurobiol* **8**, 202–210 (1998).
20. Stein J, Ridell P and Fowler S. *Brain and Reading.* London: Macmillan Press, 1989: 139–157.
21. Hier DB, LeMay M, Rosenberger PB *et al. Arch Neurol* **35**, 90–92 (1978).
22. Duara R, Kushch A, Gross-Glenn K *et al. Arch Neurol* **48**, 410–416 (1991).
23. Vanni S, Revonsu A, Saarinen J *et al. Neuroreport* **8**, 183–186 (1996).
24. Martinez A, Moses P, Frank L *et al. Neuroreport* **8**, 1685–1689 (1997).
25. Raymond JE and Sorensen RE. *Vis Cogn* **5**, 389–404 (1998).

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