Motion-onset VEPs in dyslexia. Evidence for visual perceptual deficit

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The magnocellular deficit theory is one of the prominent theories in dyslexia. However, recent studies have produced conflicting results. In order to assess the validity of this theory, 8 dyslexic children and 14 controls were examined with a motion-onset visual evoked potential (VEP) paradigm at three different velocities (2, 8, and 16 deg/s). VEP elicited by stationary gratings served as a control condition. Amplitudes of motion-onset VEP components (P100, P200) but not of the stationary VEP are significantly attenuated in dyslexic children. Further, there is an interaction of group and velocity for the P200 in the way that group differences are more pronounced for higher velocities than for lower velocities. These results support the hypothesis of an impairment of a specific magnocellular function in dyslexia. *NeuroReport* 15:1075–1078

Key words: Dyslexia; Magnocellular function; Motion onset; P100; P200; Visual evoked potential (VEP)

INTRODUCTION

Dyslexia is a specific disorder in learning to read and spell in spite of adequate educational resources, a normal IQ, no obvious sensory deficits, and adequate sociocultural opportunity [1]. Dyslexia occurs in all languages and is known to be a hereditary disorder that affects about 5% of school-aged children, making it the most common of childhood learning disorders [2].

There is an ongoing discussion about the aetiology of dyslexia [3]. Visual abnormalities have found to be associated with dyslexia. However, the exact nature of this deficiency and its potential relationship to dyslexia is not yet clear [4]. Visual processing is currently seen as comprising two separate but interactive subsystems with different 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 fast temporal resolution, low contrast, and low spatial frequencies. The dorsal pathway is believed to be concerned with visual attributes related to movement and positional relationships. 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 sensitive to medium and high spatial frequencies and has moderate temporal resolution [5]. The ventral stream into which most of the parvocellar information is channelled is mainly dealt with object discrimination based on colour, form, and texture [5]. The most widely discussed theory is that dyslexics suffer from a deficit in the magnocellular system [6]. However, the results of contrast sensitivity in particular have led to inconsistent results and challenge the magnocellular deficit assumption in dyslexia [7]. Another functional sensitivity of the magnocellular system, the perception of moving stimuli, might be more relevant for dyslexia. Eden *et al.* [8] and Demb *et al.* [9] found that activation of the motion-specific MT area was reduced in dyslexics. However, others [10,11] did not find evidence for a reduced cortical activation or lower motion perception in dyslexic adults. The fact that the velocities of the moving stimuli used varied across studies might explain the conflicting results.

The neural basis of motion perception has been studied repeatedly using visual evoked potentials [12,13]. Visual motion-onset evokes two major components of the VEP: At occipital sites, a positive component at a latency around 100–130 ms (P100), and at central-parietal leads a positivity with a latency about 230 ms (P200). The amplitude of this latter component varies with stimulus velocity [14]. We examined motion-onset VEP in dyslexic children and controls to confirm the magnocellular deficit hypothesis. Because the magnocellular system is in contrast to the parvocellular system specialised for detecting visual motion [5] we used moving sine wave gratings. As a control condition we investigated VEP elicited by stationary gratings. Since magnocellular neurones are sensitive to rapid moving stimuli, group differences between dyslexics and controls should only occur for rapid moving stimuli. Thus our hypothesis is that dyslexics have an attenuated P100 and P200 elicited by motion onset of sine wave gratings. In addition, the group differences should become larger with increasing velocity values.

MATERIALS AND METHODS

Twenty-two children, 8 dyslexics (male:female 6:2) and 14 controls (male:female 10:4) participated in the study. The two groups were selected from a pool of potential participants so that group differences in IQ and age were minimised (Table 1).

The dyslexic children visited a special boarding school for dyslexic children which is associated with a public school. Dyslexics and controls visited the same public school. Dyslexic children and controls were examined by standardised spelling [15] and IQ test [16] and non-standardised word and pseudoword reading tests [17]. Due to the lack of standardised German reading test for this age group, dyslexia was defined by spelling (discrepancy > 1.5 s.d. between actual spelling and expected spelling based on IQ [18]). Administration of non-standardised word and pseudoword lists revealed that the dyslexic children were also characterised by significantly poorer word decoding and phonological decoding abilities, respectively (one sided *t*-tests, $p=0.0003$ for word reading and $p < 0.0001$ for pseudoword reading). In the control group, spelling ability was in the normal range for all subjects (Table 1).

Additional inclusion criteria were to be a native monolingual German speaker, to have normal or corrected visual acuity, and 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 [19]. Subjects sat in a dental chair with neck support to reduce head movement in a darkened room (average luminance of 1.2 cd/m^2) at 60 cm viewing distance from an EIZO 21 inch computer monitor, the gaze was fixed on a cross in the centre of the screen. VEP were elicited by sine wave vertical gratings (2 cycles/deg visual angle, contrast=0.8, average luminance 12 cd/m^2) in a circle of 3° visual angle diameter on a dark background [20]. Tests started with a stationary phase. Subsequently the stimuli were moved randomly with 3 different velocities (2, 8, 16 deg/s) horizontally (18 times in each direction). Stimuli were presented for 600 ms in each phase. Participants indicated which direction (left or right) they had perceived by pressing one of the two buttons of a computer mouse.

Electrodes were placed at 30 scalp sites based on the 10% System of the American Electroencephalographic Society: Fp1, Fp2, F7, F3, Fz, F4, F8, FT7, FC3, FCz, FC4, FT8, T3, C3, Cz, C4, T4, TP7, CP3, CPz, CP4, TP8, T5, P3, Pz, P4, T6, O1, Oz, O2 (referred to left mastoid, ground electrode at Fpz). Horizontal and vertical eye movements and blinks were detected with two additional electrodes placed below the subjects' right and left eyes and the Fp1 and Fp2 electrodes. The EEG was amplified with Neuroscan amplifiers, low frequency cut-off at 0.1 Hz; upper frequency cut-off at 70 Hz. The EEG was recorded continuously and A/D converted at a sampling rate of 256 Hz. EEGs were analysed using the Brainvision Analyzer (www.brainproducts.com). The signals were averaged into epochs of 1100 ms, including a prestimulus baseline of 100 ms. EEG epochs in which either the EEG or electro-ocular (EOG) activity exceeded $\pm$ 100 μV or the gradient of the EOG exceeded 100 μV were automatically excluded from averaging. Peak amplitudes of the P100 at O1, Oz, and O2, and of the P200 at O1, Oz, O2, P3, Pz, P4, C3, Cz, and C4 were exported and analysed with SAS software. Huynh-Feldt correction of *p*-values was applied when the sphericity assumption was rejected (Mauchly's test), and the reported *p* values are one-sided if they refer to our hypotheses.

Table 1. Descriptive statistics on psychometric tests.

	Controls (n=14)	Dyslexics (n=8)
IQ	106.5 ± 7.5	103.3 ± 10.6
Age (years)	12.5 ± 0.4	12.7 ± 0.8
Spelling (Tvalue)	54.0 ± 6.0	29.0 ± 7.2
Word reading*	46.6 ± 2.9	19.0 ± 12.3
Non-word reading*	33.3 ± 6.3	14.3 ± 12.3

*Number of words or non-words read in 1 min.
Values are mean ± s.d.

RESULTS

VEP (P100) elicited in the stationary phase was analysed first (Fig. 1). A repeated measures ANOVA with between-subjects factor group (dyslexics *vs* controls) and within-subjects factor lateralisation (O1 *vs* O2) was carried out. The analysis yielded no significant effects (group $p=0.85$, lateralisation $p=0.28$, and group × lateralisation $p=0.46$). Consequently, VEP (P100 and P200) elicited in the moving phase was analysed second (Fig. 2). Two repeated measures ANOVAs were carried out for the motion-onset P100 and P200 amplitudes, respectively, with the factors group (between-subjects, dyslexics *vs* controls) and velocity (2, 8, and 16 deg/s, within-subjects). There was no evidence for different VEPs for left or right sided stimulus movements, therefore the data of these conditions were combined. Since there was no evidence for lateralisation effects, the data of all electrodes (P100: O1, Oz, and O2; P200: O1, Oz, O2, P3, Pz, P4, C3, Cz, and C4) were averaged for each condition.

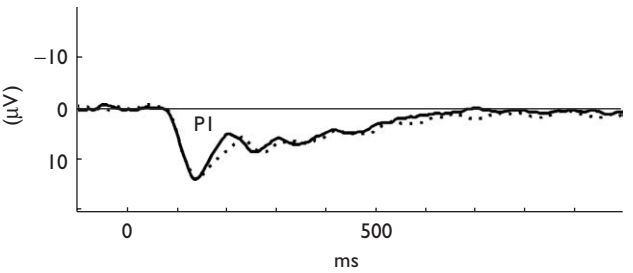

Fig. 1. Grand mean VEP (stationary phase) for dyslexics (dashed line) and controls (bold line) at occipital lead.

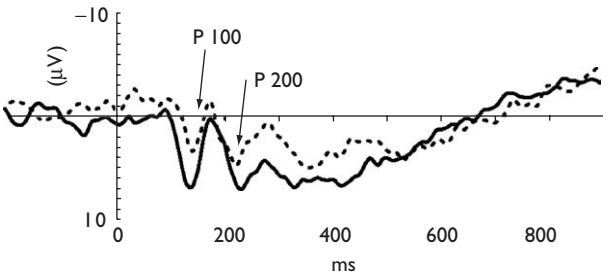

Fig. 2. Grand mean VEP (motion-onsets P100 and P200) at 16 deg/sec for dyslexics (dashed line) and controls (bold line) at left occipital (O1) lead.

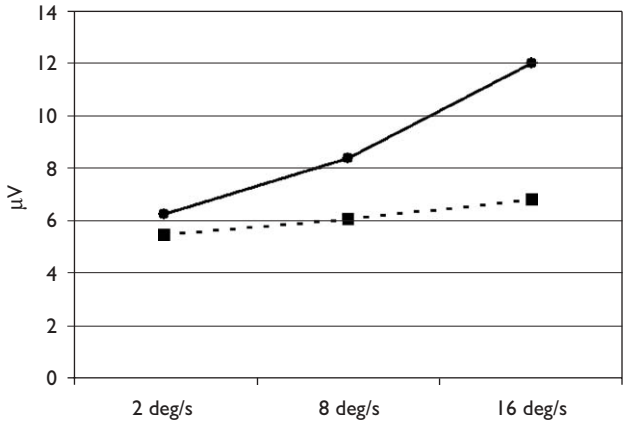

Fig. 3. Graphical illustration of the group by velocity interaction for the P200. X-axis: amplitude in µV, y-axis: velocity in deg/sec, dyslexics (rectangles), controls (circles).

The ANOVA for the P100 amplitude (Fig. 2) yielded two significant effects, the main effects group (p=0.034; attenuated amplitude in the dyslexic group) and velocity (p=0.008; higher amplitude at higher velocity). The interaction between group and velocity was tersely not significant (p=0.058; group effect being larger for higher velocity). The ANOVA for the P200 amplitude yielded three significant effects (Fig. 2), a main effect group (p=0.015; attenuated amplitude in the dyslexic group), main effect velocity (p=0.0001; larger amplitude at higher velocity) and the interaction between group and velocity (p=0.003; group effect being larger for higher velocities; Fig. 3).

DISCUSSION

We investigated motion-onset VEPs in dyslexic children and controls. In support of the hypothesis of a magnocellular deficit in dyslexia, we found a significantly attenuated amplitude of the motion-onset VEP components P100 and P200 in dyslexic children. We also found that the group difference (P200) becomes larger if the stimuli were moved faster (increased velocity). This result further encourages a magnocellular deficit in dyslexia. The finding that the VEP elicited by stationary gratings did not reveal a significant group difference replicates our previous finding [20] and met our expectation because magnocellular neurones are mainly sensitive for moving stimuli. This study contributes to the discussion about the relevance of magnocellular functions in dyslexia. Recent results on motion perception in dyslexia were inconsistent [10,11,21]. This can be solved by considering different task characteristics such as velocity of moving stimuli: group differences are larger for higher velocities. Thus the finding of no evidence for a motion perception deficit in dyslexia in the Raymond and Sorensen [10] (1.1 deg/s) and Vanni *et al.* [11] (2.5 deg/s) studies might be explained by the fact that they used moving stimuli with quite low velocities. Another relevant aspect to explain the conflicting results might be the measurement level (behavioural *vs* neurophysiological). Evidence for a motion perception deficit was found with fMRT [8,9] and VEPs (our results), whereas behavioural data did not support such a deficit [10,21]. The rationale behind this could be that behavioural data are more influenced by variables such as cognitive strategy, motivation, and attention. We found two components of the motion-onset VEP, a positivity at a latency of 100 ms and a positivity at a latency of 200 ms related to motion processing. These two components were related to motion perception and the amplitude of these were attenuated in dyslexic children. The peak maximum of the P100 amplitude was found over occipital leads, whereas for the P200 the peak maximum was found over parietal leads. Thus the P200 might be a neurophysiological correlate of neuronal activity from neurones within the dorsal stream of the magnocellular pathway. The significance of a functional impairment of parietal brain areas for dyslexia could be related to the neuronal model of attentional spotlight [22]. This theory of attentional feedback control proves that the magnocellular dominated dorsal stream gates the parvocellular inputs into the ventral stream. It has been shown in macaques that an attentional feedback from the magnocellular system selectively influence neuronal responses in the attended visual field location in the primary visual cortex [23]. Vidyasagar suggested that learning to read will be a special instance of training the dorsal stream to perform the spotlighting in a spatially sequential manner [22]. Recent behavioral studies provide evidence for a attention related spatial and temporal deficit in dyslexia which might be correlated to magnocellular functions in the parietal cortical areas [24].

In summary, we find clear evidence for a disruption of motion processing mechanisms which are dominated by input from the magnocellular system in dyslexic children. The clinical relevance of this finding has yet to be examined. Some researchers found that motion processing and encoding of letter position are associated [25]. Others suggest the significance of attentional related perceptual processing deficits [22]. However further work has to be done to explain the specific functional role of the magnocellular system for dyslexia.

CONCLUSION

The importance of magnocellular functions in the pathogenesis of dyslexia is discussed controversially. We investigated the hypothesis that dyslexic children have a specific magnocellular deficit and found evidence to support the view that processing of rapidly moving stimuli is disturbed in dyslexics. Our finding of attenuated visual evoked potentials in dyslexics only for rapidly moving stimuli stresses the functional role of the magnocellular system in dyslexia.

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