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Visual evoked potentials elicited by coherently moving dots in dyslexic children

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Abstract

The magnocellular deficit theory is one of the prominent hypotheses in dyslexia research. However, recent studies have produced conflicting results. Ten dyslexic children and 12 controls were examined with visual evoked potentials elicited by random dot kinematogram. The experiment comprises two sequences, one with randomly moving dots (control condition) and a second sequence where a fraction of the dots were moved coherently at the left or right side (depending on the level of coherence, 10%, 20%, and 40% of the dots). Randomly moving dots elicited two components, a P100 and P200, which were not different between the groups. Coherently moving dots elicited a late positivity between 300 and 800 ms, which was significantly attenuated in dyslexic children. The area of this component becomes larger at a higher level of coherence. This study supports the hypothesis of an impairment of a specific magnocellular function in dyslexia.

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Children affected with developmental dyslexia have difficulty learning to read and spell despite adequate intelligence and educational opportunity, and in the absence of any profound sensory or neurological impairment [8]. Dyslexia has been described in all languages, and the prevalence estimates range between 4% and 9%. Particularly spelling problems often persist into adulthood [8]. Dyslexia is known to be a hereditary disorder that affects about 5% of school-aged children, making it the most common of childhood learning disorders [16].

There is an ongoing discussion about the aetiology of dyslexia [14]. A great amount of research has focused on basic auditory and visual perceptual deficits which yielded conflicting results [1,14]. Visual abnormalities have been found to be associated with dyslexia. However, the exact nature of this deficiency and its potential relationship to dyslexia is not yet clear [1,16]. The most widely discussed theory is that dyslexics suffer from a deficit in the magnocellular system [19].

The magnocellular system responds to stimuli of low spatial frequency and low contrast and moving stimuli [11].

The results particularly regarding contrast sensitivity have led to inconsistent results and challenge the magnocellular deficit assumption in dyslexia [18]. Another functional sensitivity of the magnocellular system – the perception of coherent moving stimuli – might be more relevant for dyslexia. Coherent motion sensitivity – elicited by random dot kinematogram (RDK) – was repeatedly examined in dyslexic children. Evidence was found that dyslexics are less sensitive to coherent motion than controls (i.e. the threshold of the perception of coherent motion was significantly higher in dyslexics) [6,20]. This deficit was related to impaired sensitivity of cells within the retino-cortical magnocellular pathway and extrastriate areas in the dorsal stream to which they project. However, the mechanism by which the putative M-pathway deficit results in disrupted motion perception is still unclear. Since coherent motion has not yet been examined by neurophysiological methods in dyslexic children, we chose visual evoked potentials (VEP) to study the influence of coherent motion perception on cortical activity.

The neural basis of coherent motion perception has been examined by VEP elicited by a RDK paradigm [12]. Two components of motion onset were differentiated, one component evoked by motion onset [12]. The second component is evoked by coherent motion onset. These

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components correspond to different functional properties of motion processing neurons and cortical areas which are essential for the analysis and perception of motion. Motion onset can primarily be related to local motion detectors which are located in V1 [15]. Coherent motion perception is mainly related to cortical activity outside the primary visual cortex, mainly in the middle temporal visual area (MT, V5) and the region of the border between temporal, parietal, and occipital lobes, respectively [13]. Since behavioural data suggest a coherent motion perception deficit in dyslexic children, we examined coherent motion onset VEPs in dyslexic children and controls to verify the magnocellular deficit hypothesis. Our hypothesis is that dyslexics have an attenuated VEP elicited by coherent motion onset.

The threshold of perception of coherent motion was found to be a distinguishing variable between dyslexics and controls, and therefore we examined different levels of coherence (10%, 20% and 40%). As a control condition we examined the VEP elicited by motion onset (a moving pattern in which each dot was displaced in a random direction).

Twenty-two children (ten dyslexics, male/female 8:2; 12 controls, male/female 9:3) participated in the study. The two groups were selected from a pool of potential participants (see below) so that group differences in IQ and age were minimized (see Table 1).

The dyslexic children visited a special boarding school for the reading and spelling disabled which is associated with a public school; thus, dyslexics and controls visited the same school. Due to the lack of a standardized German reading test for this age group, dyslexia was solely defined by spelling (discrepancy of at least 1.5 standard deviations between actual spelling and expected spelling based on IQ [17]). Administration of non-standardized word and non-word lists revealed though that the dyslexic group was also characterized by significantly poorer word decoding and phonological decoding abilities, respectively (one sided *t*-tests, $P \leq 0.0001$ for word reading and non-word reading). In the control group, spelling ability was in the normal range for all subjects. According to the teachers, none of the controls were suffering from reading problems. Additional inclusion criteria were to be a native monolingual German speaker, to have normal or corrected visual acuity, and for the dyslexic group 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.

VEPs were elicited by RDK. The stimuli comprised a rectangular patch containing 300 randomly arranged white dots on a black background. At 60 cm viewing distance the patch of dots subtended $8 \times 12^\circ$. The luminance of the dots was 86 cd/m^2 and background luminance was 1.2 cd/m^2 yielding a Michelson contrast of 97%. The angular size of each pixel was 0.03° and the speed of moving dots was $5^\circ/\text{s}$. Each dot had a limited lifetime of 100 ms after which it would disappear and reappear at a random location within the stimulus patch. In order to minimize smooth tracking eye movements which have been found to be abnormal in dyslexics [2], a dot lifetime of 100 ms was chosen. This corresponds to the finding that no deficient eye movements were found in dyslexics if stimuli were shortened to 105 ms [10]. The experiment comprises two sequences in order to differentiate motion onset and coherent motion onset. In sequence 1, each dot was moved independently of the others in a random direction for 1000 ms. In sequence 2, a fraction of the dots were moved coherently (depending on the level of coherence, 10%, 20%, or 40% of the dots) to the left or right side horizontally for 420 ms. The direction of motion and the level of coherent motion were presented randomly, and there were 35 trials for each direction and each level of coherent motion, respectively.

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 International 10% System: 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 (linked mastoid electrodes were used as reference, ground electrode at Fpz). The EEG was amplified with Neuroscan amplifiers, with a low frequency cut-off at 0.1 Hz and an 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 analyzed using the Brainvision Analyzer (<http://www.brainproducts.com>). The signals were averaged into two epochs of 1000 ms each, including a prestimulus baseline of 100 ms. Grand averages were computed over all subjects separately for sequences 1 and 2. Motion onset amplitudes of the P100 and P200 at O1 and O2 were analyzed for sequence 1. These electrodes were chosen because we expect that motion onset primarily activates neurons at V1. The inspection of coherent motion onset VEPs revealed a positivity at 500 ms which was analyzed using TP7, CP3, CP4, TP8, T5, P3, P4, T6, O1, and O2 because we expected that coherent motion onset primarily activates neurons at MT. The mean area ($\mu\text{V} \times \text{ms}$) for P500 was calculated. A lateralization of motion onset and coherent motion onset VEPs was found [12]; thus, lateralization was incorporated in our analyses. Huynh-Feldt correction of *P* values was applied when the sphericity

Table 1
Descriptive statistics on psychometric tests (values are mean \pm SD)

	Controls ($n = 12$)	Dyslexics ($n = 10$)
IQ	106.5 \pm 6.6	103.0 \pm 9.1
Age (years)	12.5 \pm 0.4	12.7 \pm 0.9
Spelling (<i>T</i> value)	54.0 \pm 6.0	28.0 \pm 7.0
Reading words*	53.0 \pm 11.8	19.8 \pm 12.8
Reading non-words*	32.4 \pm 7.3	15.4 \pm 11.4

*Number of words and non-words read in 1 min.

assumption was rejected (Mauchly's test), and the reported P values are one-sided if they refer to our hypotheses.

The grand averages of the motion onset VEPs (control condition) revealed two peaks, a P100 and a P200 amplitude, which were analyzed first (see Fig. 1A). A repeated measures ANOVA with between-subjects factor group (dyslexics vs. controls) and within-subjects factor lateralization (O1 vs. O2) was carried out. The analysis yielded no significant effects for the P100 (group, $P = 0.41$;

lateralization, $P = 0.81$; and group \times lateralization, $P = 0.34$) and the P200 (group, $P = 0.17$; lateralization, $P = 0.07$; and group \times lateralization, $P = 0.9$) peaks.

The grand average of the coherent motion onset VEPs revealed a positivity between 300 and 800 ms (P500). Because of the lack of a clearly defined peak in the range from 300 to 800 ms in the individual data sets, the mean area under the curve in this interval was analyzed. Factors were group (between-subjects, dyslexics vs. controls), level of coherence (10%, 20%, and 40% within-subjects) and lateralization (left hemisphere TP7, CP3, P3, T5, O1, right hemisphere TP8, CP4, P4, T6, O2). There was no evidence of different VEPs depending on the movement direction of the dots, and therefore the data of these conditions were collapsed.

The ANOVA for the P500 area (see Fig. 1B) yielded two significant effects at the $P < 0.05$ level, the main effects of group ($P = 0.0134$) (attenuated area in the dyslexic group) and level of coherence ($P \leq 0.0001$) (larger area at a higher level of coherence, Fig. 1C). The other effects including all interactions were not significant.

We investigated coherent motion VEPs in dyslexic children and controls. In support of the hypothesis of a magnocellular deficit in dyslexia, we found a significantly attenuated area of coherent motion P500 in dyslexic children.

The results demonstrated that an increase of the percentage of coherently moving dots has an effect on VEP. With an increasing level the P500 area increases. This effect replicates the findings of Niedeggen and Wist [12] and is consistent with the response characteristics of neurons in area MT. By applying magnetoencephalography combined with functional magnetic resonance tomography (fMRT), it was found that coherent moving dots activate the region of temporo-parietal-occipital cortex, basically MT/V5 [3,5]. In a single unit study evidence was found that the firing rate of recorded MT neurons increases linearly with increasing percentage of moving pixels [4].

Our VEP data did not reveal hemispheric differences. A possible reason is that we did not use lateralized stimulus presentation [9].

In contrast to coherent motion we did not find evidence for different VEPs elicited by motion onset. Although the inspection of the data suggests a group difference for the P200, this difference was not statistically significant. Motion onset might be more related to magnocellular functions located at striate cortex (e.g. V1). Recent findings support this view. Results of an fMRT study suggest that V1 was better activated by noise than by coherent motion, possibly reflecting activation of neurons with a wider range of motion selectivities [3].

In conclusion, the present experiment provides evidence that VEP components are related to processing of motion. Undirected motion onset VEPs did not differ in dyslexics and controls, whereas coherent motion onset VEP clearly did so. Furthermore, these results suggest that magnocel-

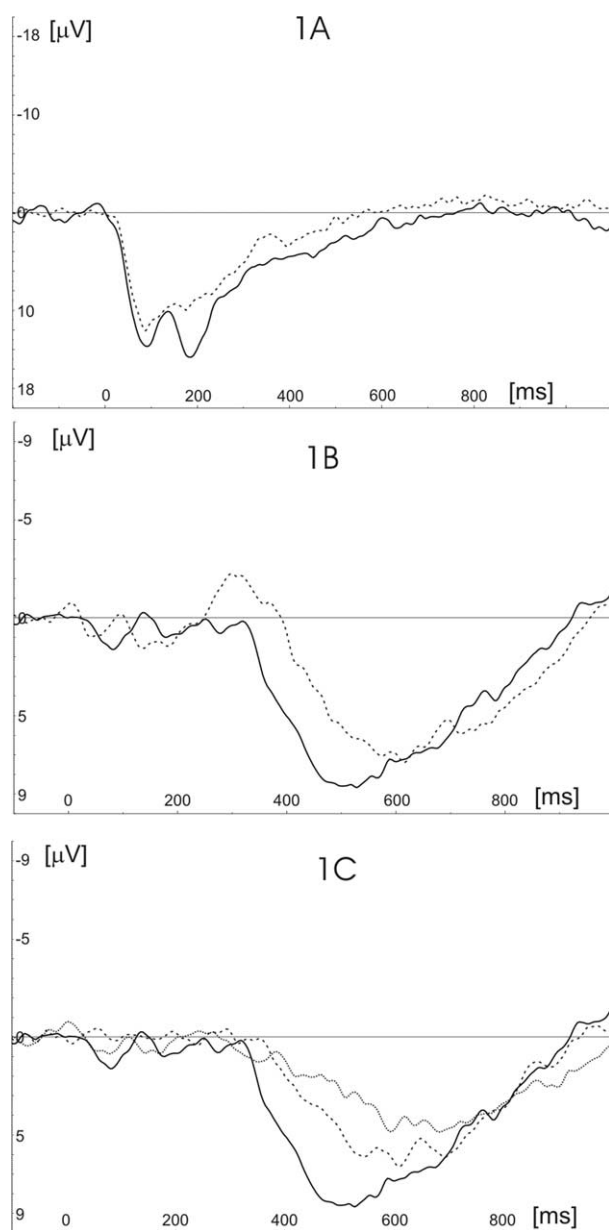


Fig. 1. (A) Grand mean VEPs (P100 and P200) for dyslexics (dashed line) and controls (bold line) at left occipital lead (O1). (B) Grand mean VEP (coherent motion P500) at right parietal (P4) lead at a 40% level of coherence for dyslexics (dashed line) and controls (bold line) at left occipital lead (O1). (C) Illustration of the main effect of level of coherence: grand mean VEPs for controls at P4 at three levels of coherence: 40%, bold line; 20%, dashed line; and 10%, dotted line.

ular functions are affected in dyslexia and, moreover, that specific regions, namely area MT/V5, are relevant for the aetiology of dyslexia.

The clinical relevance of coherent motion for reading came from a behavioural study [7]. The authors found evidence for a correlation of coherent motion detection and letter position encoding. This result suggests that an impaired magnocellular function could lead to uncertainty about where letters and letter features are positioned with respect to each other, subsequently leading to reading errors.

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