Visual information processing in dyslexic children

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Abstract

Objective: Several studies presented evidence for magnocellular deficits in dyslexics both in behavioural as well as in electrophysiological data of local electrode sites. We investigated two well-known paradigms (motion-onset and random-dot-kinematogram) with regard to global electrophysiological parameters.
Methods: Twenty-one-channel event-related potentials (ERPs) of 16 dyslectic and 15 control children were analyzed with reference-independent methods. For each paradigm quasi stable microstates were identified by means of a data-driven segmentation procedure and compared between both groups.
Results: Differences in global ERP responses between dyslexic and control children could be found for rapid moving gratings but not for the dot coherence.
Conclusions: Dyslexic children seem to have some highly specific visual deficits in processing moving stimuli. These deficits can be related to the magnocellular system.

1. Introduction

Literary language is one of the most important cultural techniques. And dyslexia – defined according to ICD-10 (Dilling et al., 1991) as a specific disability in reading or spelling in spite of a normal IQ – is an enormous problem in many languages. It seems to be associated with reduced phonological awareness and multisensory deficits. One of these lacks has been suggested to result from abnormal visual pathways: Most dyslexic studies (Lovegrove, 1996) indicate a defect in the magnocellular components of the visual processing streams (Livingstone et al., 1991; Brannan et al., 1998). The magnocellular system is, in addition to the parvocellular system, one of the two interactive subsystems for visual processing, arising from cells widely distributed across the retina and projecting to the visual cortex via the ventral lateral geniculate nucleus. It detects fast temporal resolution, low contrast, and low spatial frequencies. An irregular magnocellular system could reduce the visual sensitivity for moving or flickering stimuli (Cornelissen et al., 1998a) and therefore to interfere with lexical decision tasks (Cornelissen et al., 1998b) or the detection of small letters (Stein and Walsh, 1997) for example. Since the earliest studies (Lovegrove et al., 1980, 1982, 1986) sinus-gratings have been one of the most popular experimental means. Depending on the spatial frequency (cycles of the bright and dark bars per degree visual angle) dyslexics showed a reduced sensitivity (Evans et al., 1994; Kubová et al., 1995) for motion detection. A second experimental design includes the so-called random dot kinematogram (Cornelissen et al., 1995; Eden et al., 1996): A lot of small pixels move on the screen in random directions. Suddenly a variable percentage of these dots moves in one coherent direction. For the correct judgement of this event (cf. Slaghuis and Ryan, 1999) a minimum extent of coherence is needed which might be higher for dyslexics. Perhaps this effect could be found in electrophysiological data too.

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Some of theses experiments included not only psycho-physiological but also electrophysiological parameters (amplitudes and latencies of visual evoked potentials (VEPs) at selected electrode sites, see e.g. Livingstone et al., 1991; Johannes et al., 1996). Consequently dyslexics showed smaller evoked potentials caused by the assumed deficit in the visual subsystem, although some other studies yielded incompatible results (cf. Schulte-Körne et al., 1999 or Greatrex and Drasdo, 1995).

One aim of this study was to show any alteration in global electrophysiological responses (see Lehmann and Skrandies, 1984 for details) dependent on specific visual stimuli. Global parameters as the global field power (GFP) or the so-called centroids allow conclusions to be drawn with regard to overall electric brain activities, topographic displacements or asymmetries of brain potentials (Strik et al., 1994) and can also be used for various source localization methods (Pascual-Marqui, 1999). Based on its extraordinary high stability (Fallgatter et al., 2000) we focused on the P300-amplitude and time range. As suitable experimental setups addressing the magnocellular subsystem, the above-mentioned sinus-gratings (Slaghuis and Ryan, 1999) and random dot kinematograms (Cornelissen et al., 1995) as visual stimuli were used. We hypothesize that both paradigms might show reduced global amplitudes for dyslexics as compared to controls due to a reduced brain activity.

2. Methods

Sixteen dyslexic children (ICD-10: F 81.0 and F 81.1; gender distribution: 12 boys and 4 girls; age: 8.5–10.6 years, mean: 9.3 years; IQ measured by CFT > 85, mean: 107 ± 10) and 15 control children (11 boys and 4 girls; age: 8.3–9.9 years, mean: 9,0 years; IQ measured by CFT > 85, mean: 105 ± 9) at German school grades 2, 3 or 4 were assessed. All were native monolingual speakers of German, had no hearing or uncorrected visual problems. The child psychiatric assessments for the dyslexic children included psychopathological classification, physical and neurological examination. They were done by child psychiatrists and disclosed no neurological, emotional or behavioural deficits. Attention deficits were ruled out by the CBCL-scale; handedness was not registered. The criterion for dyslexia was a discrepancy of 1.5 standard deviations between actual and (based on IQ) expected spelling ability and T-value of < 45 (Warnke, 1992). According to standardized interviews with their parents the control children had neither neurological deficits nor attention problems. All children underwent two examinations (separated psychological and electrophysiological sessions of about 2 h each) and got a small present at the end. Seven additional children had undergone the investigation but were excluded due to unfulfilled group criteria or missing data but not due to too many blinks during EEG-recordings.
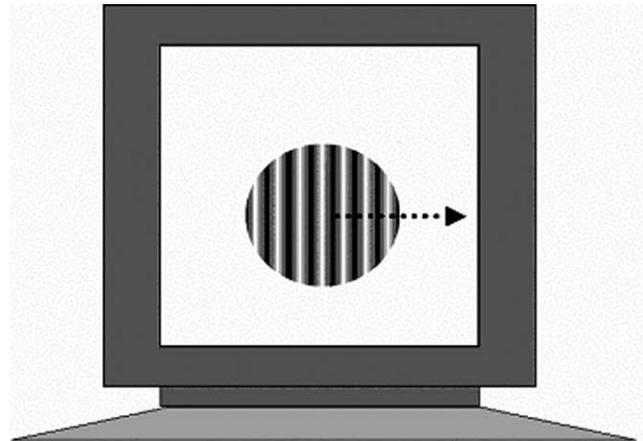

Fig. 1. Motion-onset paradigm.

During the motion-onset paradigm children looked to a 21-inch PC-monitor in the viewing distance of 60 cm. In two runs a vertical sine wave grating pattern (Fig. 1) was faded in and moved with 3 different velocities (2, 8, 16 deg/s) to the right or left side (brightness of pattern: 12 cd/m^2; brightness of background: 2 cd/m^2; contrast: 0.8; diameter of grating: 3.2° visual angle; spatial frequency: 2 cycles/deg). Each condition was presented on the screen 35 times per run (the duration of initial non-moving grating was 600 ms, the duration of the moving grating 500 ms and the duration of the empty screen (only a small central fixation cross) randomly distributed between 900 and 1900 ms.

In the coherent-motion condition they saw in two runs a random-dot kinematogram (Fig. 2) with a variable percentage (10, 20, 40, 80%) of dots moving coherently to the left or right side (brightness of the dots: 86 cd/m^2; angular velocity of the dots: 5 deg/s; size of each dot: 0.3° visual angle, visible in a rectangular of 12° height and 8° width). Each condition was presented on the screen 35 times per run (duration of the coherent motion: 420 ms, times between the coherent motions: randomly distributed between 1080 and 2080 ms).

In both paradigms the children had to press the left or right mouse-button, depending on the moving direction.

Simultaneously the event-related potentials (ERPs) were recorded from 24 scalp electrodes (covering all 10–20 sites
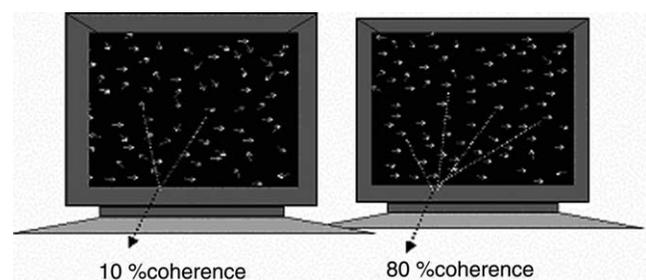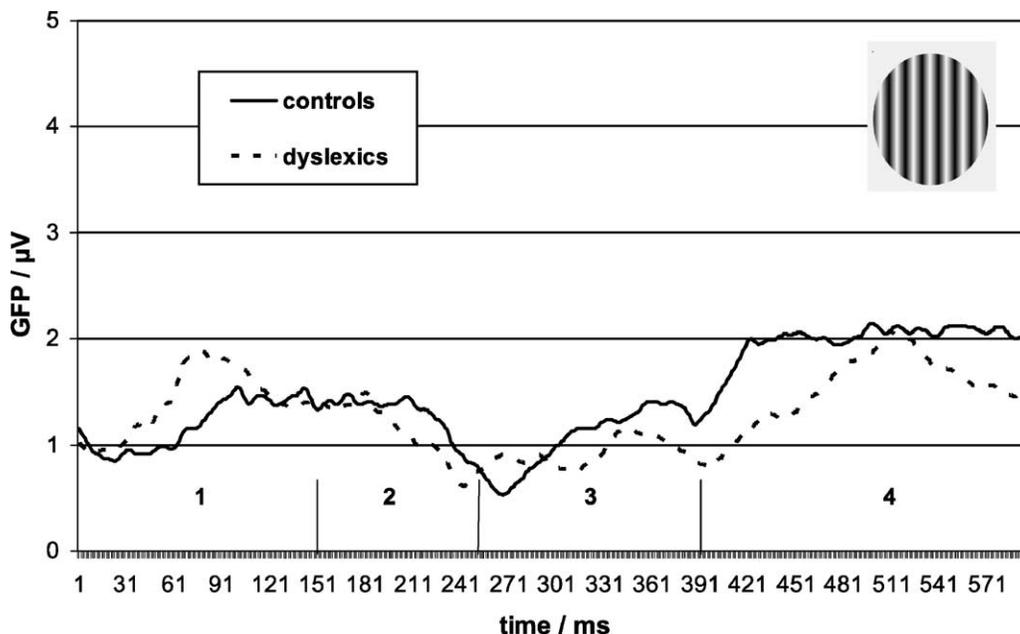

Fig. 2. Coherent-motion paradigm

Fig. 3. Motion-onset at 2 deg/s: global amplitudes (GFP) of dyslexics versus controls.

plus Oz, FPz and EOG-electrodes). All impedances were below 5 kΩ. ADC-rate was 1000 Hz with an amplifier bandpass filter of 0.1–70 Hz. All data were re-referenced to average reference. Each analyzed sweep started with the onset of the motion and ended 600 ms afterwards. The artefact-free samples (rejection criteria were voltages exceeding 100 μV or voltage rises of more than 50 μV/ms in any channel) were averaged (in all trials at least 20 artefact-free EEG epochs per condition in a single subject were achieved). The data were analyzed according

to the adaptive segmentation (Lehmann and Skrandies, 1984; Brandeis and Lehmann, 1986) yielding different time segments. Details are outlined in Fallgatter et al. (1997, 1999). The resulting GFP (Lehmann and Skrandies, 1980) shows the map strength, the resulting centroids illustrate the map topography (Pascual-Marqui et al., 1995). Statistical analyses based on an analysis of variance (one between measure: two groups; one repeated measure: 3 conditions for the motion onset paradigm/4 conditions for the coherent motion paradigm) and post hoc t tests.
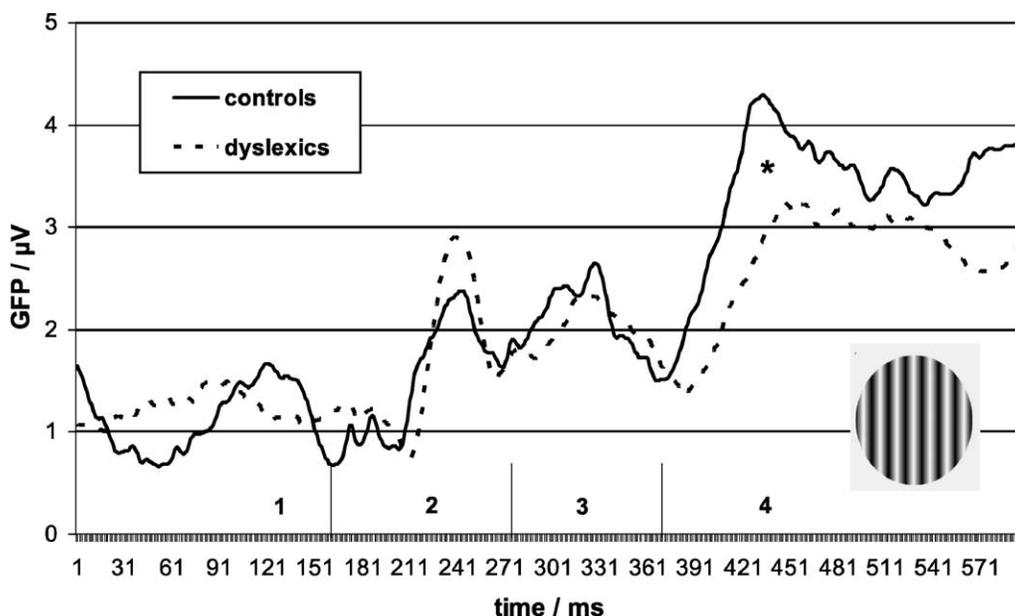

Fig. 4. Motion-onset at 16 deg/s: global amplitudes (GFP) of dyslexics versus controls. $*P < 0.05$.
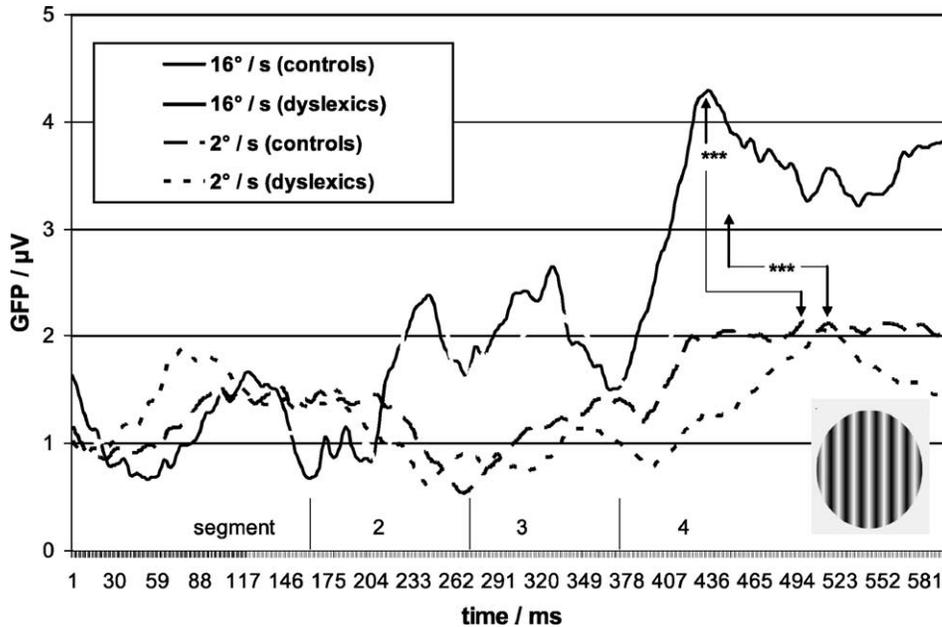
Fig. 5. Motion-onset: global amplitudes (GFP) of dyslexics and controls at two different angular velocities, ***$P < 0.001$.

3. Results

All figures display the GFP-curves averaged at each recording time, whereas the tables show the statistics of the individual GFP-peaks in time segment 4.

3.1. Motion-onset paradigm

At the moving onset of the slowly moving sinus-gratings only low global amplitudes without any pronounced maxima can be seen (Fig. 3). Contrary to these findings the quickly moving sinus-gratings evoked 4 consistent microstates (data driven time segments based on the grand mean from the controls) with borders around 160, 270 and 375 ms (Fig. 4). Additionally the ERP responses of controls versus dyslexics are plotted for the high and low velocity condition in Fig. 5. The statistical analysis for the amplitude maxima in segment 4 shows significant effects ('velocity': $F = 117$, df $= 2$, $P < 0.001$; 'group': $F = 6.8$, df $= 1$, $P < 0.05$; and interaction: $F = 4.7$, df $= 2$, $P < 0.05$). Post hoc t tests reveal the amplitude differences in detail (Table 1). Controls show also higher global amplitudes in the time segments 2–4 for the high as compared to the low velocity condition.

3.2. Coherent-motion paradigm

Depending on the growing percentage of coherent moving dots the global amplitudes rise both for controls (Fig. 6) and for dyslexics (Fig. 7). A comparison of the global amplitude maxima in the most pronounced time segment 4 (450–600 ms) is plotted in Fig. 8. The 10% and 20% coherence conditions yield only small peaks which differ marginally between groups. A significant amplitude rise occurs at 40% coherence for both groups. Again for controls there is further significant increase at 80% coherence whereas the higher level for dyslexics at 40% coherence only slightly rises up to a bit lower extend at 80% coherence compared to controls (Table 2). The statistical analysis for the amplitude maxima in segment 4 reveals highly significant effects for 'coherence condition' ($F = 38.4$, df $= 3$, $P < 0.001$). The 'group' effect ($F = 0.62$, df $= 1$, $P > 0.05$) and the interaction effect ($F = 1.17$, df $= 3$, $P > 0.05$) are not significant. All results of post hoc t tests can be seen in Table 2.

4. Discussion

4.1. Motion-onset paradigm

The identified ERP microstates especially in the late time segment 4 (the classical P300-region) showed significant amplitude differences between the presented velocities.

Table 1

Statistical t tests between maxima of global amplitudes (GFP) at different motion-onset conditions and groups in time segment 4

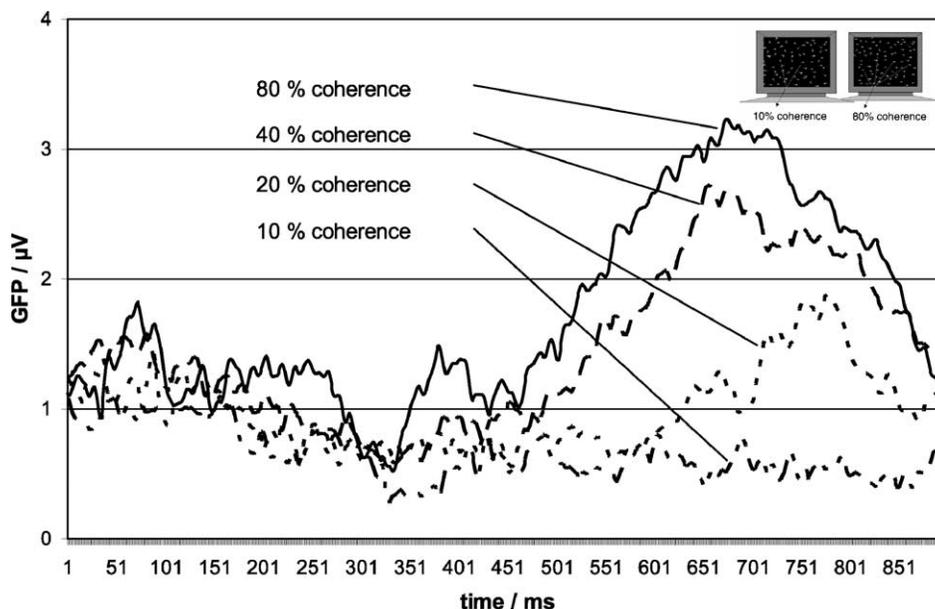| Condition	Group	GFP-maximum (μV)	P	t	df	cf.
2 deg/s	Controls	4.75	> 0.05	1.96	60	Fig. 3
	Dyslexics	4.04				
16 deg/s	Controls	8.06	< 0.05	2.66	60	Fig. 4
	Dyslexics	6.24				
2 deg/s	Controls	4.75	< 0.001	-8.08	29	Fig. 5
16 deg/s		8.06				
2 deg/s	Dyslexics	4.04	< 0.001	-7.09	31	Fig. 5
16 deg/s		6.24				

Fig. 6. Coherent-motion: global amplitudes (GFP) of controls at different percentages of coherence.

The physiological responses separate clearly between slow and fast visual pattern. This is true for both groups, whereas the ERP-amplitudes of the control children exceed the amplitudes of the dyslexic children in the fast condition significantly. The measured global ERP-responses of the dyslexics for fast movements are significantly less pronounced and this supports our assumption that the motion detection seems to be underdeveloped in dyslexics. Dyslexic children showed reduced activity compared with control children in a condition that is believed to be strongly influenced by the magnocellular system and thus data would support the hypothesis of abnormalities in

the magnocellular pathway. These findings are in accordance to similar results in ERP (Brannan et al., 1998) or functional magnetic resonance imaging (Demb et al., 1997; Eden et al., 1996) data.

4.2. Coherent-motion paradigm

The identified ERP microstates of the random dot kinematograms in the late time segment 4 showed significant amplitude increases with increasing coherences, at least above a certain threshold. But there were no significant differences between both groups and this
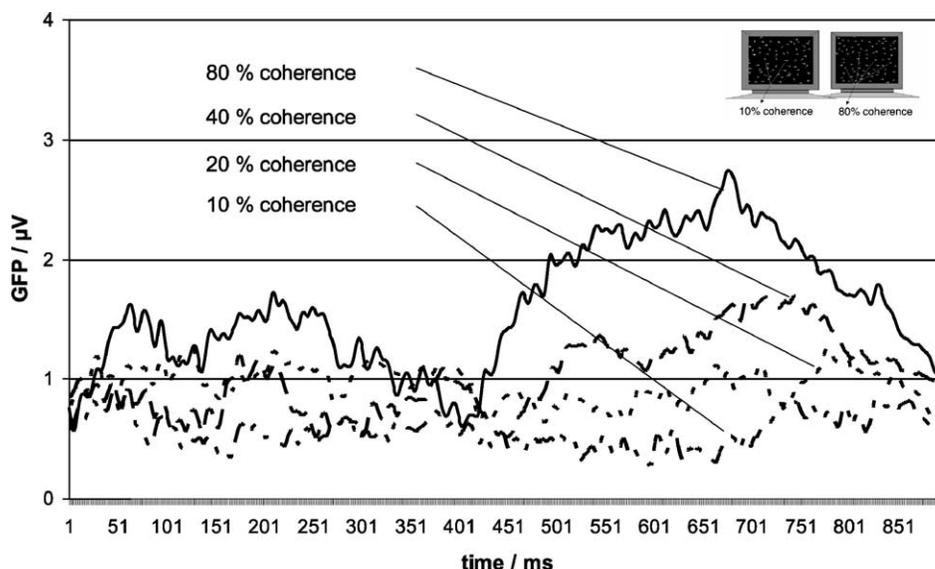

Fig. 7. Coherent-motion: global amplitudes (GFP) of dyslexics at different percentages of coherence.
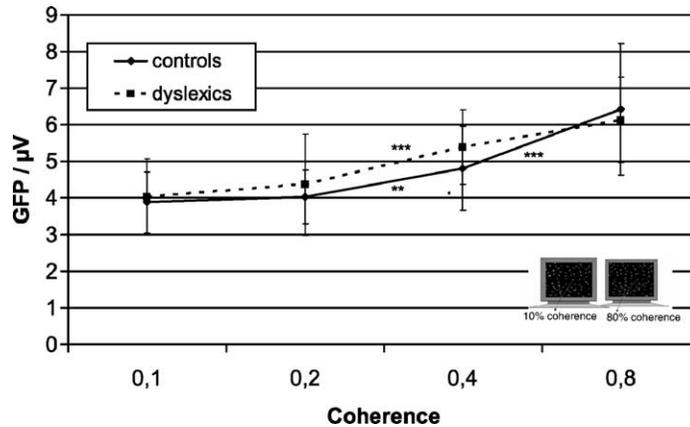
Fig. 8. Coherent-motion: mean of the global amplitude maxima of dyslexics and controls at different percentages of coherence in time segment 4. **$P < 0.01$; ***$P < 0.001$.

refuses our hypothesis. The brain potentials excited by the coherent movement of the dots seem to be in the same order for dyslexics and controls. The general assumption of a "significant reduction in sensitivity to coherent motion in the group with dyslexia" (Slaghuis and Ryan, 1999) could not be reproduced by our electrophysiological data, but in their study Slaghuis and Ryan used behavioural criteria (correct responses on a button pad). On the other hand we replicated the findings of Slaghuis and Ryan (1999) and Raymond and Sorensen (1998) that there are thresholds of motion recognition in the order of 20–40% coherence.

Summing up, it may be said that dyslexic children show attenuated electrophysiological signal responses to moving visual stimuli. This is true not in general but depends strongly on the experimental design and supports the theory that dyslexia is not a general deficit in visual information processing, but depends on highly specific abnormalities in visual recognition.

Table 2
Statistical *t* tests between maxima of global amplitudes (GFP) at different coherence conditions for both groups in time segment 4

Coherence	Group	GFP-maximum (µV)	P	t	df	cf.
10%	Controls	3.88	>0.05	-0.92	29	Fig. 8, solid line
20%		4.03				
20%		4.03	<0.01	-3.39		
40%		4.81				
40%		4.81	<0.001	-4.29		
80%		6.42				
10%	Dyslexics	4.03	>0.05	-1.72	31	Fig. 8, dotted line
20%		4.37				
20%		4.37	<0.001	-4.23		
40%		5.39				
40%		5.39	>0.05	-1.51		
80%		6.13				

References

Brandeis D, Lehmann D. Event-related potentials of the brain and cognitive processes: approaches and applications. Neuropsychologia 1986;24: 151–68.

Brannan JR, Solan HA, Ficarra AP, Ong E. Effect of luminance on visual evoked potential amplitudes in normal and disabled readers. Optom Vis Sci 1998;75:279–83.

Cornelissen PL, Richardson A, Mason A, Fowler S, Stein J. Contrast sensitivity and coherent-motion detection measured at photopic luminance levels in dyslexics and controls. Vis Res 1995;35:1483–94.

Cornelissen PL, Hansen PC, Gilchrist I, Cornack F, Essex J, Frankish C. Coherent-motion detection and letter position encoding. Vis Res 1998a; 38:2181–91.

Cornelissen PL, Hansen PC, Hutton JL, Evangelinou V, Stein JF. Magnocellular visual function and children's single word reading. Vis Res 1998b;38:471–82.

Demb JB, Boynton GM, Heeger DJ. Brain activity in visual cortex predicts individual differences in reading performance. Proc Natl Acad Sci USA 1997;94:13363–6.

Dilling H, Mombour W, Schmidt MH. International classification of mental diseases, ICD-10 (German edition). Bern: Huber; 1991.

Eden GF, VanMeter JW, Rumsey JM, Maisong JM, Woods RP, Zeffiro TA. Abnormal processing of visual motion in dyslexia revealed by functional brain imaging. Nature 1996;382:66–9.

Evans BJ, Drasdo N, Richards IL. An investigation of some sensory and refractive visual factors in dyslexia. Vis Res 1994;34:1913–26.

Fallgatter AJ, Brandeis D, Strik WK. A robust assessment of the NoGo-anteriorisation of P300 microstates in a cued continuous performance test. Brain Topogr 1997;9:295–302.

Fallgatter AJ, Mueller TJ, Strik WK. Age-related changes in the brain electrical correlates of response control. Clin Neurophysiol 1999;110: 833–8.

Fallgatter AJ, Eisenack SS, Neuhauser B, Aranda D, Scheuerpflug P, Herrmann MJ. Stability of late event-related potentials: Topographical descriptors of motor control compared with the P300 amplitude. Brain Topogr 2000;12:255–61.

Greatrex JC, Drasdo N. The magnocellular deficit hypothesis in dyslexia: a review of reported evidence. Ophthalmic Physiol Opt 1995;15(5): 501–6.

Johannes S, Kussmaul CL, Münte TF, Mangun GR. Developmental dyslexia: Passive visual stimulation provides no evidence for a magno-cellular processing defect. Neuropsychologica 1996;34(11):1123–7.

Kubová Z, Kuba M, Peregrin J, Nováková V. Visual evoked potential evidence for magnocellular system deficit in dyslexia. Physiol Res 1995;44:87–9.

Lehmann D, Skrandies W. Reference-free identification of components of checkerboard-evoked multichanal potential fields. Electroenceph clin Neurophysiol 1980;48:609–21.

Lehmann D, Skrandies W. Spatial analysis of evoked potentials in man – a review. Prog Neurobiol 1984;23:227–50.

Livingstone M, Drislane F, Rosen G, Galaburda A. Physiological and anatomical evidence for a magnocellular defect in developmental dyslexia. Proc Natl Acad Sci USA 1991;88:7943–7.

Lovegrove B. Dyslexia and a transient/magnocellular pathway deficit: the current situation and future directions. Aust J Psychol 1996;48:167–71.

Lovegrove W, Bowling A, Badcock D, Blackwood M. Specific reading disability: differences in contrast sensitivity as a function of spatial frequency. Science 1980;210:439–40.

Lovegrove W, Martin R, Bowling A, Blackwood M, Badcock D, Paxton S. Contrast sensitivity functions and specific reading disability. Neuropsychologica 1982;20:309–15.

Lovegrove W, Martin R, Slaghuis W. A theoretical and experimental case for a visual deficit in specific reading disability. Cogn Neuropsychol 1986;3:225–67.

Pascual-Marqui RD. Review of methods for solving the EEG inverse problem. Int J Bioelectromagn 1999;1:75–86.

Pascual-Marqui RD, Michel CM, Lehmann D. Segmentation of brain electrical activity into microstates: model estimation and validation. IEEE Trans Biomed Eng 1995;42:658–65.

Raymond JE, Sorensen RE. Visual motion perception in children with dyslexia: normal detection but abnormal integration. Vis Cognit 1998;5(3):389–404.

Schulte-Körne G, Bartling J, Deimel W, Remschmidt H. Attenuated hemispheric lateralisation in dyslexia: evidence of a visual processing deficit. Neuroreport 1999;10(17):1–5.

Slaghuis WL, Ryan JF. Spatio-temporal contrast sensitivity, coherent motion, and visible persistence in developmental dyslexia. Vis Res 1999;39(3):651–68.

Stein J, Walsh V. To see but not to read; the magnocellular theory of dyslexia. Trends Neurosci 1997;20:147–52.

Strik WK, Dierks T, Franzek E, Stöber G, Maurer K. P300 in schizophrenia: interactions between amplitudes and topography. Biol Psychiatry 1994;35(11):850–6.

Warnke A. Legasthenie und Hirnfunktion, 2nd ed. Bern: Huber; 1992.