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SHORT COMMUNICATION

Visual Evoked Potential Evidence for Magnocellular System Deficit in Dyslexia

Z. KUBOVÁ[1], M. KUBA[2], J. PEREGRIN[3], V. NOVÁKOVÁ[4]

Departments of [1]Physiology, [2]Pathophysiology and [3]Ophthalmology, Charles University, Faculty of Medicine, Hradec Králové; [4]Psychological and Pedagogical Centre, Hradec Králové, Czech Republic

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Summary

Some recent studies on dyslexia have suggested a selective abnormality in the magnocellular visual pathway. To verify this hypothesis, we investigated motion-onset visual evoked potentials (VEPs) (predominantly testing the magnocellular system) as well as pattern-reversal VEPs (presumably testing the parvocellular system) in 20 dyslexics and 16 controls (both groups with a mean age of 10.0 years). Although the latencies and amplitudes of the main positive peak of pattern-reversal VEPs did not differ between the dyslexic and control group, the motion specific negative peak of motion-onset VEPs was significantly delayed ($p<0.001$) in dyslexics. Our results confirm a selective magnocellular pathway disorder in dyslexics and indicate that the motion-onset VEPs might serve as an objective method for early diagnosis of dyslexia.

Key words

Dyslexia – Visual evoked potentials – Motion – Magnocellular system

In the past, the most commonly accepted view concerning the origin of dyslexia (impairment of reading skills despite normal intelligence, social environment, visual acuity and motivation) was that reading disability was not attributable to defects in visual processing (e.g. Vellutino 1979). However, it has been suggested in a number of recent studies (e.g. Livingstone *et al.* 1991, Mason *et al.* 1993, Chase and Jenner 1993, Lovegrove 1993) that dyslexia might result from a disorder in one of the two basic parallel visual pathways, namely the magnocellular one, which is believed to be predominantly a flicker and motion detecting system.

To test this hypothesis, we examined visual evoked potentials (VEPs) in dyslexics to the onset of movement that seem to be specific for evaluation of the magnocellular system function (Kuba and Kubová 1992, Kubová *et al.* 1995). Simultaneously, VEPs to conventional pattern-reversal stimulation (presumably testing the parvocellular system – Kubová *et al.* 1995) were also analyzed.

Both types of VEPs were tested in 16 normal readers (8 boys and 8 girls, with the mean age of 10 years) and in 20 age-matched dyslexic (but not dysgraphic) children (14 boys and 6 girls). Dyslexia was diagnosed by specialists in the Psychological and Pedagogical Centre. The dyslexic children, although having normal IQ (PD Wechsler test), displayed reading skills at least two years behind the skills of children of the same age. All of them had visual acuity of 6/6 or better (with correction if needed) and showed no other apparent visual abnormalities when tested by an ophthalmologist.

The visual stimulus consisted of either square-wave black and white checkerboard with an element size of 40' (pattern-reversal) or isolated checks with 120' check-to-check distances (motion stimulation). The mean luminance was 17 cd/m^2 and contrast of either 96 % (pattern-reversal stimulation) or 10 % (motion stimulation) was used. The stimuli were generated on a computer monitor (ViewSonic 21"; 100 frames/s; total display size 30 x 40 deg) under

computer control (IBM compatible PC 486). For pattern-reversal VEPs the checkerboard reversed at a rate of 2 rev/s, for motion-onset VEPs the pattern was displaced at a velocity of 10 deg/s in the direction varying randomly from trial to trial (left, right, up or down). The movement lasted 200 ms and the interstimulus interval (during which the pattern was presented stationary) was 1 s. Monocular and binocular VEPs were recorded in the bipolar lead O_Z-C_Z and in three unipolar leads with the electrodes placed at O_Z and 5 cm to the right and to the left from this point – O_R and O_L. Linked earlobes served as reference. After amplification (Tektronix AM 502) in the 0.1–100 Hz band, 40–100 epoches (according to the signal-to-noise ratio) of 400 ms duration were averaged on a PC with a sampling rate of 500 Hz. In the pattern-reversal VEPs latency and amplitude of the main P100 peak were evaluated and in the motion-onset VEPs those of the motion specific N160 peak.

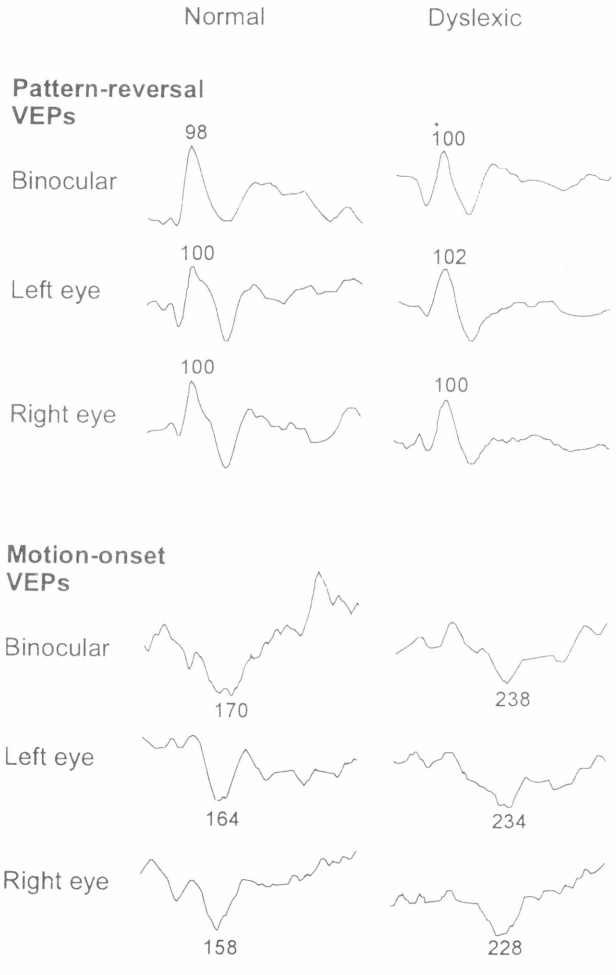

Fig 1
Typical examples of the binocular and monocular pattern-reversal and motion-onset VEPs from one normal reader and from one dyslexic child.

No significant differences in the pattern-reversal VEPs were found between the normal and dyslexic subjects. In contrast, the latencies of the monocular as well as binocular motion-onset VEPs were significantly prolonged ($p<0.001$) in the group of the dyslexics (see original recordings of VEPs in Fig. 1). While the pattern-reversal VEP latencies from dyslexic children never exceeded the upper limit of the norm, 70 % of the dyslexics had abnormally delayed motion-onset VEP latencies (Fig. 2).

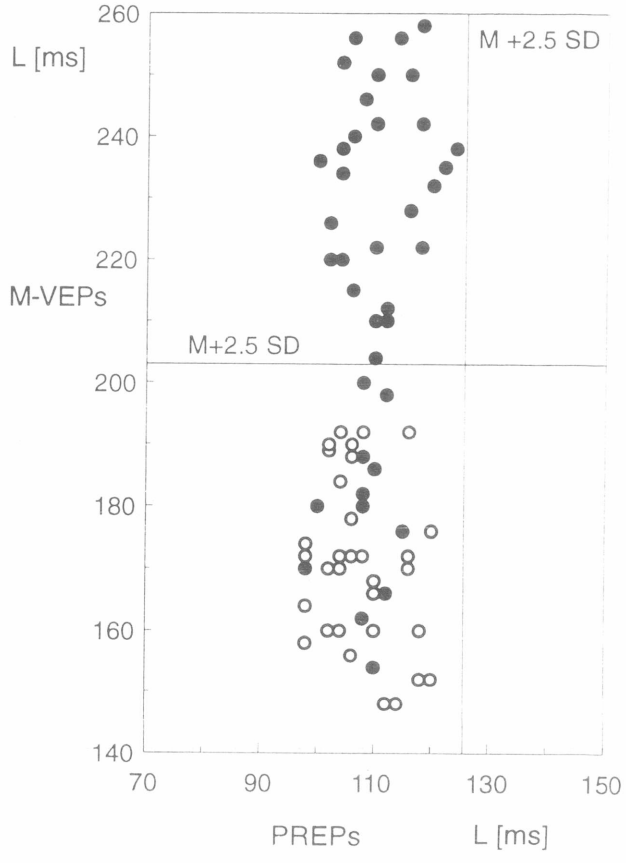

Fig 2
Scatter diagram of the monocular pattern-reversal VEP latencies (PREPs) versus monocular motion-onset VEP latencies (M-VEPs) for dyslexic children (solid circles) and control group (open circles). M + 2.5 S.D. represents Mean + 2.5 Standard Deviation which is the upper limit of the norm (based on findings in the control group of 16 children).

Among many different theories concerning the origin of dyslexia there is an increasing evidence that a slowing down of visual perception through the magnocellular subdivision of the visual pathway (transient deficit) can play a substantial role in about of 75 % of dyslexics (e.g. Lovegrove 1993). Our findings fit well with these data because in the present study we have shown that in 70 % of our dyslexic children a selectively impaired magnocellular (transient) system function (tested by motion-specific VEPs) can be found. The increased latencies of our variant of the motion-onset VEPs indicate that the conduction velocity of the magnocellular visual system is slower

than in normal readers, which can be caused by the reduced size of magnocellular cells in lateral geniculate nucleus found by Livingstone *et al.* (1991). Breitmeyer and Ganz (1976) speculated how a weakness of the transient system can lead to reading problems. It can either impair the normal coordination of saccadic eye movements during reading, or the slow transient system in dyslexics cannot interrupt sufficiently the sustained activity between two successive visual fixations. This might produce an overlapping of the perceived letters.

Although these theories have not been fully verified yet, this study supports the concept of some magnocellular system insufficiency in dyslexia. Therefore, the motion-onset VEPs, so far not commonly used, can represent a very convenient method for objective testing of this defect.

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Reprint Requests

Dr. Z. Kubová, Department of Physiology, Faculty of Medicine, Charles University, Šimkova 870, 500 38 Hradec Králové, Czech Republic.