Electrophysiological studies on visual information processing in dyslexia and ADHD

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Chapter 5

An electrocortical measure of visual orienting discriminates infants at risk for dyslexia from controls at 5 months

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Abstract

The present study was aimed at detecting early signs of visual orienting problems in dyslexia. A group of 28 5-month-old infants at risk for dyslexia (12 girls and 16 boys) and 17 control infants (10 girls and 7 boys) were investigated using a spatial cueing paradigm. Continuous electroencephalogram (EEG) was measured and event-related potentials (ERPs) to spatial cues were obtained. The at-risk group showed smaller ERPs compared to controls, particularly at parieto-occipital sites, corroborating the results of a prior investigation on dyslexic adults. At-risk boys and girls did not differ with respect to visual orienting.
Introduction

A child with developmental dyslexia can experience severe difficulties in acquiring the specific phonological and visual skills necessary for learning to read and write. By definition, reading level of these children is not age-appropriate despite adequate instruction, intelligence, and the absence of physical disability (APA, 2000). The observation that dyslexia runs in families led to genetic studies, demonstrating the contribution of hereditary factors to the occurrence of the disorder with estimates of familial transmission ranging from 50 to 70% (DeFries, Fulker & Labuda, 1987).

Besides evident deficits in phonological processing, deviances have been found in low-level visual processing that are consistent with the transient, or magnocellular, visual system (Jaśkowski & Rusiak, 2005; Lovegrove, 1990; Stein & Walsh, 1997). Neuroimaging and autopsy studies have confirmed the presence of structural defects in cortical and subcortical structures, responsible for processing transient visual as well as auditory information, indicative of magnocellular system defects (Galaburda, Sherman, Rosen, Aboitiz, & Geschwind, 1985; Humphreys, Kaufmann & Galaburda, 1990; Paulesu, Demonet, Fazio, McCrory, Chanoine et al., 2001). The orienting response, also relying heavily on the integrity of magnocellular pathways projecting to the parietal cortex (Johnson, 2002; Posner & Petersen, 1990), is considered important for reading (Casco, Tressoldi, & Dellantonio, 1998). Some behavioural evidence has been found for weaker involuntary orienting of visuo-spatial attention in dyslexic children and adults (Brannan & Williams, 1987; Buchholz & Aimola Davies, 2007; Dhar, Been, Minderaa, & Althaus, 2008; Facoetti, Paganoni, Turatto, Marzola, & Mascetti, 2000). The benefit of orienting attention prior to shifting ones gaze is to facilitate perceptual processing at the attended location (Posner, 1980), thus speeding up reading. This process may be inefficient in developmental dyslexics.

Cortical mechanisms of visual orienting of attention have frequently been studied in normally functioning adults (Eimer, 2000; Fu, Fan, Chen, & Zhuo, 2001; Nobre, Sebestyen, & Miniussi, 2000; Yamaguchi, Tsuchiya, & Kobayashi, 1994). In a previous study on covert orienting, dyslexic adults showed deficient orienting to peripheral cues, as demonstrated by smaller brain potentials to cues in the superior-parietal and parieto-occipital cortices. The largest group differences were found at the location of the precuneus (Dhar et al., 2008). Due to the reflexive nature of visual orienting, it can also be measured in infants. Covert orienting can
be determined by means of a spatial cueing paradigm, whereby shifts of attention are triggered by the onset of a peripheral cue (Posner, 1980). Covert orienting in infants has been measured from the age of 4 months (Johnson, Posner, & Rothbart, 1994; Johnson & Tucker, 1996; Richards, 2000). When the cue is presented briefly, no saccade is elicited to the cue (Johnson & Tucker, 1996). As a result, attention is rapidly shifted to the location of the cue. Keeping in mind that the maturation of cortical and sub-cortical systems may be altered in dyslexic children deviances are expected from an early age.

The present study was part of the Dutch Prospective Dyslexia Study (NWO, 1996) that was launched in attempt to discover early visual, auditory and linguistic precursors of dyslexia that may facilitate early identification of the disorder. The measurement of ERPs provides non-invasive insight into cortical processing during task processing. This technique offers the possibility to obtain information on neural processing in infants in the absence of an overt response. Previously, the general focus has been on the development of auditory discrimination of speech sounds in at-risk children (Lyytinen, Guttorm, Huttunen, Hämäläinen, Leappänen et al., 2005; Molfese, 2000; van Leeuwen, Been, Zwarts, Maassen, et al., 2008). The focus of the present study is to investigate electrocortical deviances in visual orienting in infants with a familial risk of dyslexia using a spatial cueing paradigm. In line with the previous findings from adults with dyslexia, we expect a decreased evoked potential to cues in at-risk infants.

Materials and methods

Participants
The participant group consisted of 28 5-month-old infants at genetic risk for dyslexia (12 girls and 16 boys) and 17 controls (10 girls and 7 boys). Another 14 children were too sleepy or tired to complete the experiment. Infants were recruited from the Dutch population and were included in the Dutch Prospective Dyslexia Study. Inclusion in the at-risk group was based on (1) a family history of the parents with respect to reading problems and dyslexia and (2) scores of the affected parents and of an additional first- or second-degree relative of these parents on a test battery including speed tests on real word reading, non-word decoding and verbal IQ (Kuijpers, van der Leij, Been, van Leeuwen, ter Keurs, et al., 2003).
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Inclusion of the infants was only possible if at least one parent and one relative were classified as severely dyslexic. Dyslexia was not present in the control group families and both parents had to score above criterion on our dyslexia test. All participating families received oral and written information about the study and all gave informed consent. The study protocol was approved by a medical ethics committee.

Stimuli, paradigm and procedure
The task began with a picture of a colourful computer-drawn face presented centrally on a monitor for 1000 ms to draw the infant’s attention to the screen. Each subsequent trial consisted of a cue-stimulus sequence. Another picture of a colourful computer-drawn face, now in a vertical bar of 2 by 8 cm appearing on a light background, was used for both the cue and the stimulus, which were presented at a visual angle of 1° to the left or right of the centre of the screen. The cue was presented for a short duration of 46 ms whereas the stimulus was presented for 500 ms with an inter-stimulus interval of 75 ms. The stimulus appeared either at the same or the opposite visual field as the cue. The following four trial types were presented randomly: left cue-left stimulus, right cue-right stimulus, left cue-right stimulus, and right cue-left stimulus.

Infants were placed in a child safety seat at a distance of 55 cm from a monitor on which the stimuli were presented. The experimenter was seated in a separate room and was not visible to the child during the experiment. Trials were manually presented when the child’s eyes were focussed on the screen. A direct video link from a video camera, focussing on the infant’s eyes, allowed the experimenter to observe when the child was actually viewing the screen, as the monitor was then reflected in the infant’s pupils. When it was necessary to attract the infant’s attention back to the screen, scenes from Sesame Street were presented in addition to the stimuli. A minimum of in total 40 successful trials was required.

Electrophysiological recording and analysis
Continuous EEG was sampled at 500 Hz, using a SynAmps model 5083 amplifier (Neuroscan) with an input impedance of 10 MOhm, from 64 Ag/AgCl-sintered ring electrodes embedded in an EEG recording cap (EASYCAP GmbH). Reference electrodes were attached to the mastoids. A ground electrode was placed on the right cheekbone. Electrodes were arranged according to the international 10/20 system (Jasper, 1958). Horizontal electro-oculogram (EOG) was recorded from the
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outer canthus of each eye. Vertical EOG was recorded from infraorbital and supraorbital electrodes placed in line with the pupil of the left eye. Electrodes were filled with conductive gel using a syringe without a needle. Cotton buds were used to ensure that the impedance was maintained below 15 kΩ. A band-pass filter (0.5 – 30 Hz, 48 dB/oct) and notch filter (50 Hz) were applied to the raw data. The Gratton and Coles algorithm (Gratton, Coles, & Donchin, 1983) was used to correct ocular artefacts. For all trials the EEG was segmented from -200 to 1400 ms from cue onset and was subsequently averaged. An interval from 140 to 290 ms was divided into three 50 ms bins, the areas (μV x ms) of which were exported for analyses.

A repeated measures analysis was applied with the between-subjects factors group (two levels: at-risk and control) and gender (two levels: boy and girl). Two within-subjects factors were applied: bin (with three levels: bin 1 ranging from 140 - 190 ms, bin 2 from 190 - 240 ms, and bin 3 from 240 – 290 ms) and electrode. As the largest effects were expected at parieto-occipital and central parietal sites, the following 16 electrodes were entered: CP3, CP1, CPZ, CP2, CP4, P3, P1, Pz, P2, P4, PO3, POZ, PO4, O1, OZ, and O2. The dependent measure was the mean area in each of the three bins.

Results

The number of trials upon which averages were based did not differ between the groups. The average number of trials was 74 in the at-risk group and 72 in the control group.

A group x bin x electrode interaction was revealed (F(30,12) = 2.94, p = .026), suggesting that the group difference varied per bin and electrode. Further analyses revealed the greatest group differences in bin 3 (240 – 290 ms) for the electrodes CP4 (F(1,41) = 5.07, p = .03), P1 (F(1,41) = 6.18, p = .017), PO3 (F(1,41) = 7.62, p = .009), POZ (F(1,41) = 5.72, p = .021), and O1 (F(1,41) = 6.82, p = .013). The results suggest a reduced mean area in the at-risk group compared to controls in response to the cue for these electrodes. No main effect for gender was found (F(1,41) = .17, p = ns), nor were there any interactions of gender with group, bin or electrode.

Figure 1 depicts ERPs in the central, parietal, and occipital cortex. The larger ERP is apparent for the control group compared to at-risk infants.
ERPs to cues in the at-risk and control group

Figure 1. The figure represents the average cue-evoked potential in at-risk and control infants, measured from the parieto-occipital cortex. The vertical dotted line corresponds to cue onset.
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Discussion

Reading requires rapid visual processing that is controlled by the magnocellular stream (Vidyasagar, 1999). The aim of the present study was to investigate the efficiency of covert visual orienting, relying on magnocellular processing, in infants at risk for developmental dyslexia. The present data demonstrated a distinctly smaller electrocortical response to peripheral cues in at-risk children, suggesting that the development of visual orienting in children with a familial risk for dyslexia is disturbed from a very early age and that these deviances can be non-invasively measured at an age of 5 months. It should, however, be noted that in infants as young as 5 months the variability of ERPs is far greater than in older children and adults (deRegnier, 2005), thus enhancing the potency of the findings in the present study.

The present study is in line with a previous study on adults with dyslexia, who also showed a diminished cortical response to peripheral cues on a similar visuo-spatial orienting paradigm (Dhar et al., 2008). The attenuated cortical responses to cues were found mainly in the right superior parietal hemisphere and the precuneus, the activation of which has been related to the processing of spatial cues (Giesbrecht, Woldorff, Song, & Mangun, 2003), lending further support for the involvement of the parietal cortex in dyslexia (Jaśkowski & Rusiak, 2005).

Surprisingly, no differences were found between girls and boys in the present study. Prior studies on normal brain development, as well as structural and functional brain studies predict that gender differences in dyslexia are to be expected (Lambe, 1999).

The largest group differences were found in the interval from 240 to 290 ms. This interval overlapped the onset of stimulus presentation. Therefore, one may question whether the differences are related to cue processing or stimulus processing. If the ERP differences in bin 3 are related to stimulus processing, we would expect to find group differences after 290 ms as well. Therefore, an additional 2 bins were analysed ranging from 290 - 340 and from 340 - 390 ms. As no group differences were found in these bins, we propose that the group differences are related to cue processing.

Exploring the neurochemical mechanisms underlying attention may assist in understanding the deficits seen in dyslexia. Acetylcholine receptors in visual pathways play an important role in selective attention (Beane & Marrocco, 2004), and diminished acetylcholine has been found to dramatically slow orienting of
attention (Mentis, Sunderland, Lai, Connolly, Krasuski, et al., 2001). In a model simulation of auditory neuronal processing in the dyslexic brain (Been & Zwarts, 2003), it was demonstrated how a hypothetically lowered cortical neuronal density in the dyslexic model, could result in a lowered amount of available acetylcholine. Simulating a rise in the release of acetylcholine caused the auditory evoked potential to reach a normal level. Similarly, decreased acetylcholine levels may be involved in visual processing deficits in dyslexia.

Due to the relatively small number of trials in each condition, we were unable to analyse left and right cues separately. Future investigations should endeavour to include analyses of visual field effects, as these may provide more information on possible differences in hemispheric function.

The infants in this study have an elevated risk for dyslexia. Only when they are older, will the children be tested for dyslexia. When this information is available, the analyses will be repeated with the pure dyslexic group. Therefore, the group differences seen now are expected to become more pronounced in the future.
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