ELSEVIER

How is dysfluent reading reflected in the ERP?

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Abstract

The present study focused on early ERP differences between dyslexic and fluent readers which may reflect a deficit in letter string processing. We compared the ERPs of dyslexic and fluent readers (20 German-speaking boys in each group, age 13–14 years) in response to words and consonant strings. Due to the regularity of German orthography, our dyslexic readers suffered mainly from impaired fluency and not from errors. In a word task each trial presented short, highly frequent words and the occurrence of a pseudoword among the eight items of a trial had to be reported. In a corresponding string task each trial presented consonant strings and the occurrence of a word among the strings had to be reported. The blocked presentation allowed expectations about the nature of the stimuli and their timing. Dyslexic readers exhibited a reduced CNV in anticipation of words and strings, which in the case of words was followed by a compensatory enhanced N220. This pattern was limited to left posterior sites and may reflect a sluggish response of brain regions involved in visual letter string processing.

1. Introduction

Dyslexia is a learning disorder that affects the acquisition of reading in a substantial number of children despite normal intelligence and adequate schooling. In regular orthographies, different from English, dyslexic readers exhibit relatively few reading

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errors and suffer mainly from massively reduced reading speed (e.g. Landerl, Wimmer, & Frith, 1997; Zoccolotti et al., 1999). Of importance is that the reading speed problem is not limited to frequent words, where it could be interpreted as reflecting a problem with the storage of letter patterns (i.e. surface dyslexia), but was found similarly for pseudowords with little similarity to existing words (Wimmer, 1993, 1996). A detailed analysis of the reading speed deficit of dyslexic readers was recently presented by Ziegler, Perry, Ma-Wyatt, Ladner, and Schulte-Körne (2003). They found that even for short frequent words, dyslexic German readers (11-year-old) showed an increase in reading time of about 300 ms per additional letter, whereas fluent readers showed no effect of word length. This length effect on reading time resembles a similar, although more dramatic length effect observed for acquired cases of letter-by-letter readers (Behrmann, 1999; Farah, 1999), which for the acquired cases is interpreted as reflecting a visual attentional problem with the parallel processing of several letters (i.e. simultanagnosia). Recently, Hari and Renvall (2001) presented a review of behavioural findings suggesting sluggish focussing and shifting of visual attention (i.e. minineglect) as underlying problem of the impaired reading speed of developmental dyslexia cases.

Consistent with the position that the difficulty of dyslexic readers resides in the early phase of letter string processing are MEG findings with Finnish dyslexic readers (Salmelin, Service, Kiesilä, Uutela, & Salonen, 1996). The Finnish dyslexic readers similar to their German counterparts suffer from a reading speed impairment and not from errors. The latter is not surprising as Finnish is maybe the most regular of all alphabetic orthographies. In response to words and letter strings, Finnish dyslexic readers showed a rather different activation pattern compared to fluent readers. Of particular importance is the absent activation within 200 ms after stimulus onset in left occipitotemporal regions and within 200 and 400 ms in left temporal regions. For the former, that is the left occipitotemporal regions, the left fusiform gyrus was identified by Cohen and Dehaene in a series of ERP and fMRI studies as being specifically involved in letter string processing (see review by McCandliss, Cohen, & Dehaene, 2003). A further finding of the Finnish MEG study was that the dyslexic Finnish readers compensated their early deficient letter string processing by enhanced activation of inferior frontal regions (including Broca) between 200 and 400 ms. Further studies of the Finnish group supported the finding of abnormal dyslexic activation patterns in the early time window where letter string processing is to be expected (Helenius, Salmelin, Service, & Connolly, 1999; Helenius, Tarkiainen, Cornelissen, Hansen, & Salmelin, 1999).

The present study attempted to examine the generality of the Finnish findings for German dyslexic readers, but we had to rely on ERPs. Several ERP studies found a sharp negative ERP deflection peaking around 200 ms (N200) post-stimulus in response to letter strings (e.g. Bentin, Mouchetant-Rostaing, Giard, Echallier, & Pernier, 1999; Nobre, Allison, & McCarthy, 1994). We assumed that this N200 may correspond to the early activation in the Finnish MEG findings. The N200 in the mentioned ERP studies generally was found to be insensitive to differences in the lexical status of the stimuli and, therefore, is thought to reflect visual letter string processing. However, in designing the present study we considered an extreme variation of the lexical status of the stimuli to be of interest and, therefore, contrasted short, highly frequent words with consonant strings. Obviously, the letter strings of the highly frequent words must be familiar visual

objects at least for fluent readers, whereas the consonant strings must be unfamiliar for both groups. Some studies did in fact find an early effect of letter string familiarity in the MEG and ERP (e.g. Assadollahi & Pulvermüller, 2001; Sauseng, Bergmann, & Wimmer, 2004).

From the Finnish MEG findings of a reduced early activation to letter strings, one would expect that the classic N200 response in our German dyslexic readers might as well be reduced. However, a review of studies which provide information on differences between dyslexic and fluent readers on the early ERP to words (i.e. N100/P100, N200/P200) does not offer strong support for this expectation. Some studies did find reduced early ERP components in dyslexic readers (Brandeis, Vitaccio, & Steinhausen, 1994; Neville, Coffey, Holcomb, & Tallal, 1993; Wimmer, Hutzler, & Wiener, 2002) or abnormal morphology (Helenius, Salmelin, et al., 1999). However, other studies did not find any differences in the early ERP (Robichon, Besson, & Habib, 2002; Stelmack, Saxe, Noldy-Cullum, Campbell, & Armitage, 1988). Differences in the timing of presentation, nature of stimuli, task requirements and/or characteristics of the dyslexic samples may be responsible for this limited convergence of results.

2. Method

2.1. Participants

Twenty dyslexic and 20 fluently reading male adolescents of about 13–14 years of age were recruited from a large longitudinal sample of 530 boys. To be included in the dyslexic group, their present reading rate score (see below) was at or below percentile 10 and they all had already scored below percentile 15 on both a reading fluency test and a spelling test in Grade 3. Exclusion criterion was a low nonverbal IQ (85 or lower) based on three scales (spatial sequences, spatial integration, and spatial concepts) from the Primary Test of Cognitive skills (Huttenlocher & Cohen-Levine, 1990) which was administered at the end of Grade 1. The control group included boys who both in Grade 3 and presently showed reading and spelling scores above percentile 20. Furthermore, both groups only included boys with a clear right hand performance.

The present group selection was based on a standardized sentence reading test, which requires to quickly mark each item of a list of sentences as correct or incorrect. The means of correctly marked sentences within 3 min were 28 (SD=4) and 43 (SD=6) for dyslexic and fluent readers corresponding to percentiles of 5 and 60, respectively. Since wrong markings hardly occurred, the poor performance of the dyslexic participants reflects the reading speed impairment characteristic for dyslexia in regular orthographies. The slow reading of the present dyslexic readers was also evident from two reading aloud tasks, one requiring fast reading of a rather difficult text, the other fast reading of a list of pseudowords. For text reading, the mean syllable per minute scores were 144 (SD=32) and 241 (SD=18) for dyslexic and fluent readers, respectively; for pseudoword reading the corresponding means were 84 (SD=17) and 124 (SD=16), respectively. The error rates of the dyslexic readers tended to be low with 18 and 9% for text and pseudoword reading, respectively, and were largely due to complying with the instruction of reading

as quickly as possible. The dyslexic readers did not differ from the fluent readers with respect to nonverbal IQ (means: 102 and 104, respectively) and age (means: 13; 11 years and 13; 9 years, respectively).

2.2. Tasks and stimuli

The experimental setup intended to elicit cognitive processes similar to the ones involved in fast word processing in natural reading situations. The instruction in the word task was to read the eight items of a trial and examine whether or not a 'nonsense' word without meaning was included. After a 'yes' response, participants were asked for the 'nonsense' word. Correspondingly, in the consonant string task the instruction was to examine whether or not a real word was included among the consonant strings and after a 'yes' trial were asked for the word. Each task consisted of 24 trials (i.e. 192 items) with half of the trials including an odd which was presented varying between fourth and eighth item position. Maximal one odd was presented in a trial. The order of the task was counterbalanced. The large majority of the trials were judged correctly by the dyslexic readers (90% correct yes/no responses for word task trials and 96% correct for the string task trials). Fluent readers were close to perfect (99% correct for both tasks).

The sequence of events of an exemplary trial of each task is given in Fig. 1. Stimulus presentation and timing was done with the Gentask Software from Neuroscan. The items of a trial followed each other in immediate succession. The presentation duration of an item varied between 700 and 750 ms to prevent phase locking on stimulus onset. Participants were familiarized with each task and with the procedure by three practice
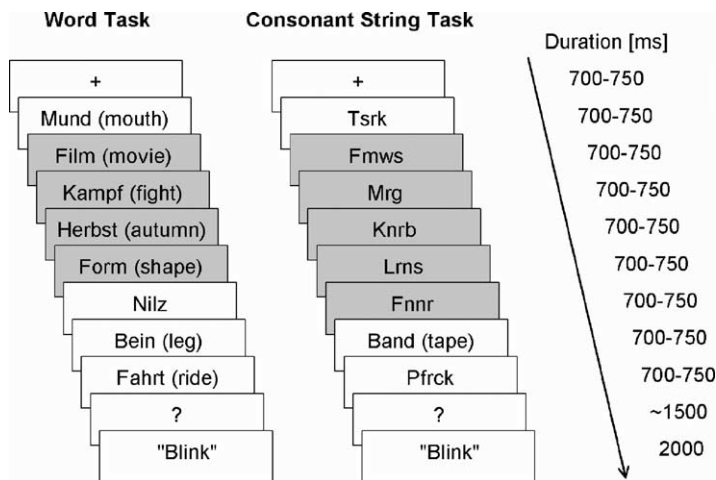

Fig. 1. Item sequence and item duration of a trial for the word and the consonant string task. To the question mark participants had to verbally report ('yes'/'no') the presence/absence of an odd among the items and had to name the odd. 'Blink' stimulated eye blinks before the following trial. The trials include an odd (pseudoword and word, respectively) and require a 'yes' response. Items in grey rectangles were used for calculating the ERPs.

trials before each task. Positive feedback and encouragement was given frequently after the largely correct responses.

The items of the word task were short one-syllable German nouns (3–6 letters long) of high frequency with a mean of 1513 occurrences per million according to the CELEX database (Baayen, Piepenbrock, & van Rijn, 1993). Examples are given in Fig. 1. German nouns start with an upper-case letter. Consonant strings were matched to words in letter length and care was taken to avoid visual similarity with existing words. Stimuli were presented on a CRT monitor (refresh rate of 100 Hz) in yellow on a black background in a dimly illuminated room. The letter size of 10 mm (for upper-case letters) at a distance from the monitor of 120 cm was chosen in such a way that it corresponded to a natural reading situation with a letter size of 3 mm at a reading distance of 35 cm, which makes a visual angle of 0.5°.

2.3. EEG recording

The EEG was recorded from 12 Ag/AgCl electrodes mounted to an elastic cap which was fitted to the scalp. The scalp sites contained six standard international 10–20 system locations (P7, P8, PO7, P8, O1 and O2) and six nonstandard locations. The nonstandard locations were chosen to cover language related cortical areas and their right hemisphere homologue. These sites are termed Broca (BL/BR, positioned halfway between FC5 and F7), Wernicke (WL/WR, halfway between CP5 and T7) and angular gyrus (AL/AR, halfway between P3 and CP5). Eye movement artifacts were recorded bipolarly with electrodes placed above and below the left eye (vertical eye movements) and beside the right and the left eye (horizontal eye movements). Electrode impedances were kept below 5 kΩ. EEG was recorded against a common linked earlobe reference using a Neuroscan 32-channel biosignal amplifier system (bandpass 0.15–50 Hz, sampling rate 250 Hz, 16 bit resolution). Eye blink and muscle artifacts were rejected based on computer algorithms using individually adjusted voltage thresholds. Frequencies lower 0.5 Hz and higher 30 Hz were digitally filtered out.

2.4. Data analysis

The ERPs were calculated from 100 ms prior to stimulus onset up to 600 ms following stimulus onset. The first item of each trial was excluded to avoid an effect of the large luminance change from the fixation cross to the first item. Obviously, the odd items were excluded from the calculation too, but we also excluded all items of a trial which followed an odd. This prevented that the large electrophysiological effect of recognizing the odd may be imported into the ERP of the critical non-target items. Thus, all items contributing to the ERP were preceded by a non-target item (see Fig. 1). The mean number of word items contributing to the ERP was 114 and 115 for dyslexic and fluent readers, respectively. The mean number of string items was 109 and 115, respectively.

The period of 100 ms prior to stimulus onset was originally planned to be used for baseline correction. However, as evident from Fig. 2, a slow negative deflection
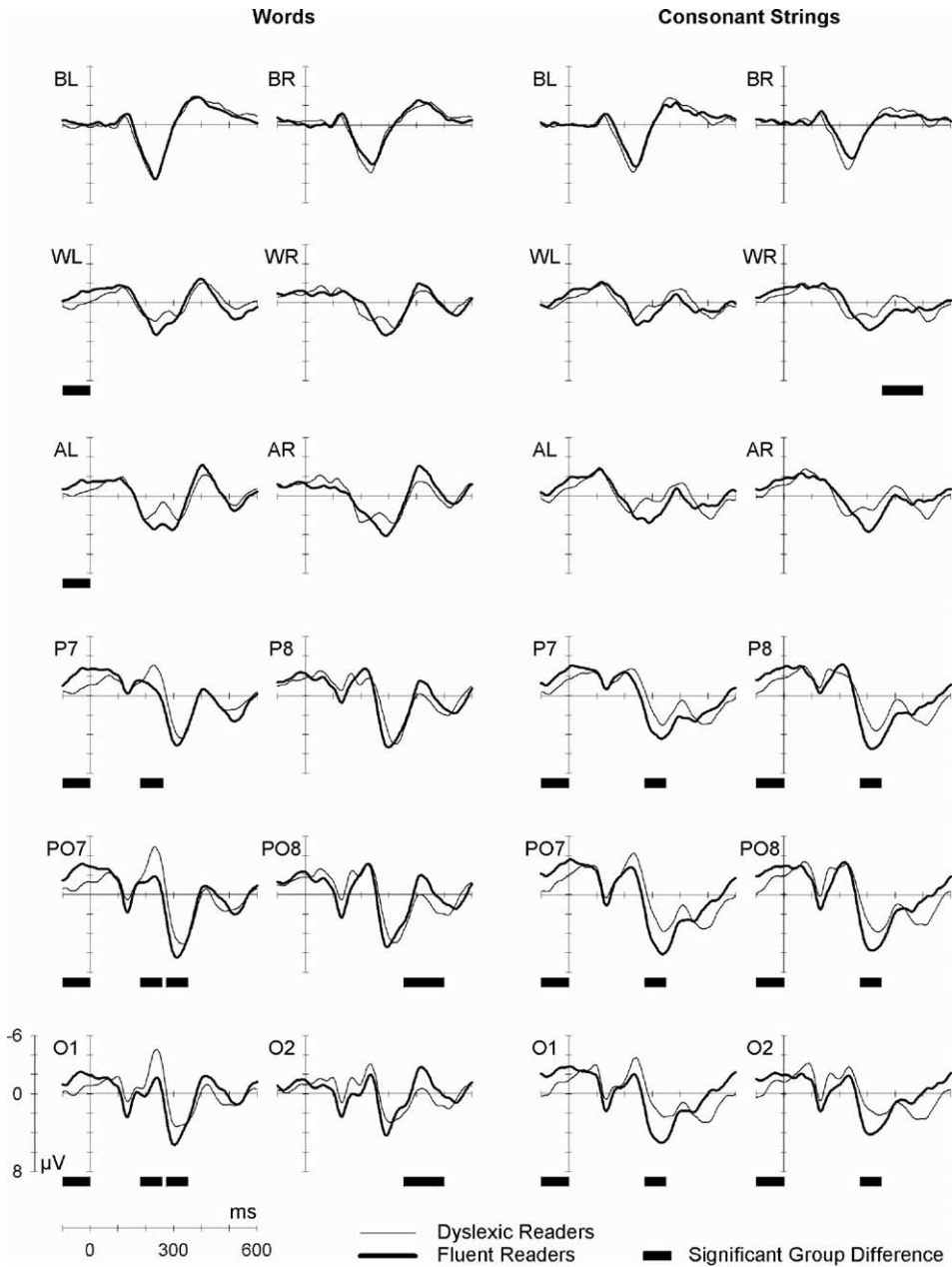
Fig. 2. Grand average ERPs for words and consonant strings. Black bars below the ERPs indicate significant differences ($p < .05$) between ERPs of dyslexic and fluent readers. The length of the bar indicates the latency window for which the mean amplitude was calculated. Electrode sites different from the standard 10–20 system notation are BL/BR—Broca left/right, WL/WR—Wernicke left/right and AL/AR—angular gyrus left/right. Negative voltage is plotted upwards and stimulus onset is at 0 ms.

(CNV)—particularly marked at posterior sites—was present in this period and it differed between dyslexic and fluent readers. Because a 'true' baseline period immediately preceding stimuli was not realized in our paradigm and the CNV appeared to be an essential component of the ERP, no baseline correction was applied. Therefore, the ERPs presented in Fig. 2 reflect the absolute potential differences between the recording electrodes and the referential electrode (common linked earlobe). To examine whether the groups differed before the start of a trial, we compared the mean amplitudes during the last 200 ms of the presentation of the fixation cross, which preceded the start of a trial (Fig. 1). This comparison was done separately for the word and the string task and it was found that the mean amplitudes of the two groups in this baseline period were rather similar and did not differ from each other at any electrode site, all $ts(38) < 0.57$.

Beside the CNV, which was present prior to stimulus onset, the following ERP components were observed after stimulus onset. At posterior sites, there was first a sharp positive deflection with a latency of about 130 ms, followed by a negative deflection peaking at 220 ms. The P130 was relatively small probably due to the fact that stimuli followed in immediate succession with little accompanying change in gross physical characteristics such as luminance. Deflections of the early components at anterior sites were opposite to those at posterior sites. However, at sites over Wernicke and angular gyrus regions there was hardly any N130 and, furthermore, dyslexic readers exhibited a double peak deflection instead of a single P220 peak. Therefore, the analysis of group differences in early components was limited to the posterior electrode sites showing clearly detectable peaks. Further, only at posterior sites a positive deflection peaking at 310 ms was observed. A broad negative deflection peaking at 400 ms was found at all recording sites with larger amplitudes at anterior sites. For words this component was present for both groups. For strings, as expected, the N400 was absent or diminished for fluent readers, but, unexpectedly, it was present for dyslexic readers.

The ERP was quantified by measuring individual peak latencies and calculating mean amplitudes in five latency windows according to the mentioned components: CNV (-100 to 0 ms), P130 (100–160 ms), N220 (180–260 ms), P310 (270–350 ms) and N400 (350–500 ms). These latency windows were chosen according to the peak latency in the grand average. For the sharp early components, narrow latency windows (60 and 80 ms) were applied, whereas for the broad N400 a wider window of 150 ms was used to calculate the mean amplitudes. Measurement of individual peak latencies was based on a computer algorithm finding the most prominent peak in a range of ± 100 ms around the peak latency measured in the grand average ERP.

Mean amplitudes and latencies were analysed with repeated-measure ANOVAs (using Greenhouse-Geisser correction where appropriate) for each latency window separately. Factors were stimulus type (word/consonant string), electrode site (Broca left/right, Wernicke left/right, angular gyrus left/right, P7/8, PO7/8, O1/2) and group (dyslexic/fluent readers). For the analyses of the P130, N220 and P310 only the six posterior electrodes showing these components were included. Given the interest in group differences and space limitation, only ANOVA findings showing a main effect of group or an interaction effect including group are reported.

3. Results

3.1. Latencies

The only group difference approaching significance was found for the N220, $F(1,28) = 4.07$, $p = .053$ with dyslexic readers exhibiting a higher latency than fluent readers (230 vs. 217 ms). Additional inspection of means showed tendencies towards prolonged latencies of the P310 and the N400 in dyslexic readers, but these differences were small (around 10 ms) and not reliable.

3.2. Mean amplitudes

3.2.1. CNV

Relevant findings were a reliable main effect of group and a reliable three-way interaction between group, stimulus type and electrode site, $F(1,38) = 6.48$, $p < .05$, and $F(11,418) = 4.52$, $p < .01$, respectively. Dyslexic readers exhibited a smaller (less negative) mean amplitude than controls (-0.53 vs. -1.30 μV). The interaction was further analysed by post hoc tests. Reliable group differences ($p < .05$) are represented in Fig. 2 as bars below the ERP component. The length of the bars indicates the latency window for which the mean amplitude was calculated. Fig. 2 shows that for words, the reduced CNV amplitudes of the dyslexic readers were limited to the left hemisphere sites WL, AL, P7, PO7 and O1. In contrast, for consonant strings dyslexic readers exhibited reduced amplitudes at left and right hemispheres sites P7, P8, PO7, PO8, O1 and O2.

3.2.2. P130 (100–160 ms)

Neither the main effect of group nor any interaction with group was reliable, Fs < 2.7. As shown in Fig. 2, there was a tendency towards a lower P130 in dyslexic readers.

3.2.3. N220 (180–260 ms)

Only the three-way interaction between group, stimulus type and electrode site was reliable, $F(5,190) = 4.90$, $p < .01$. Fig. 2 shows that for words, dyslexic readers exhibited reliably larger N220 only at the left hemisphere sites P7, PO7 and O1. At these sites, the pattern of group differences is inverse to the one observed for the CNV, that is, the lower CNV for dyslexic readers was followed by the higher N220. For strings there only was a tendency towards a larger N220 at these left posterior sites. Interestingly, at the corresponding right hemisphere sites there was no tendency at all towards a higher N220 for dyslexic readers. The inverse relation between the CNV and the N220 was also evident from reliable negative correlations between these two ERP measures at all left sites: P7, PO7, and O1 (words: $-.34$, $-.32$, $-.33$, ps $< .05$; strings: $-.44$, $-.41$, $-.45$, ps $< .01$). At right sites, a reliable correlation was only present at the occipital site O2 (words: $-.33$, $p < .05$; strings: $-.48$, $p < .01$).

3.2.4. P310 (270–350 ms)

Both the main effect of group and the interaction between group and stimulus type was reliable, Fs$(1,38) = 8.91$ and 8.31, ps $< .01$, respectively. Only for strings, but not for

words did dyslexic readers exhibit a reliably smaller P310 than fluent readers (strings: 1.88 vs. 4.13 µV, $p < .001$; words: 3.12 vs. 4.03 µV). Also there was a tendency towards a reliable three-way interaction involving site, $F(1,190) = 2.31$, $p = .105$. Fig. 2 shows that for words, dyslexic readers exhibited reliably smaller amplitudes only at PO7 and O1, whereas for consonant strings, the difference was reliable for all posterior sites.

3.2.5. N400 (350–500 ms)

Here the finding of interest is the interaction between group and stimulus type, $F(1,38) = 8.62$, $p < .01$ and a tendency towards a reliable three-way interaction including electrode site, $F(11,418) = 2.25$, $p = .099$. For words dyslexic readers showed a smaller N400 than fluent readers (-0.01 vs. -0.60 µV), whereas for strings the opposite was observed (0.73 vs. 1.07 µV), but none of these group differences reached significance. Fig. 2 shows that for words the smaller N400 of the dyslexic readers was reliable only at right posterior hemisphere sites (PO8 and O2). For strings, the higher N400 of dyslexic readers was only reliable at WR. However, as evident from Fig. 2, these group comparisons of mean amplitudes may mask the apparent group difference in ERP morphology in response to consonant strings. Here dyslexic readers—particularly at posterior sites—exhibited an unexpected N400, which was nearly absent for fluent readers who instead exhibited a slow negative going deflection. Formulated differently, dyslexic readers showed a N400 response to both words and strings, whereas fluent readers showed a marked N400 response only to words.

4. Discussion

A first finding was the failure to detect substantial ERP latency deficits of dyslexic readers. Such a deficit would be expected from the general slow reading performance of our dyslexic participants. A group difference of borderline reliability was only found for the latency of the N220, but even here the dyslexic delay was small (13 ms). For the other components (P130, P310 and N400) the latency differences were negligible. These findings are consistent with several other negative findings on latency differences between dyslexic and fluent readers (Brandeis et al., 1994; Neville et al., 1993; Robichon et al., 2002). However, other studies found massively delayed latencies (Breznitz & Leikin, 2001; Breznitz & Misra, 2003; Csepe, Szücs, & Honbolygo, 2003; Leikin, 2002). The reasons for this divergence are not obvious. A rather trivial one could be that despite the general massive behavioral difference in reading time, the two groups may not have differed in reading the short and highly frequent words of the present task. This possibility is not plausible, but cannot be ruled out as our paradigm did not allow to measure reaction times to each stimulus. A further, more general explanation for the absent dyslexic ERP latency deficit in the present and some other ERP studies is offered by the position of Klimesch et al. (2004) that ERPs reflect a synchronization of brain oscillations, like alpha and theta. For example, the present P130 component in response to letter strings may arise from the reset and synchronisation of the continuous alpha and theta rhythms to the onset of the stimulus. If this is the case, and, furthermore, if the extent and type of resetting and

synchronisation is not differentially affected by the present task, then no latency delay is expected when dyslexic readers exhibit alpha and theta frequencies in the normal range.

According to the MEG findings of Salmelin et al. (1996) we expected a reduced N220 which may reflect a visual attentional dysfunction in letter string processing of dyslexic readers. However, the opposite was found as dyslexic readers exhibited a clearly enhanced N220 in the case of words and a tendency in this direction for strings. This finding is also contrary to the tendency among ERP studies towards reduced early components for dyslexic readers (Brandeis et al., 1994; Helenius, Salmelin, et al., 1999; Neville et al., 1993; Wimmer et al., 2002). However, our finding of an enhanced N220 among dyslexic readers has to be related to the reduced CNV. This pattern suggests a compensatory relationship, that is, the reduced anticipatory activation of brain regions involved in letter string processing was compensated for by the enhanced activation of the very same brain regions after arrival of the letter string. This compensatory relationship finds support not only in the comparison of group means, but also in the negative correlations between CNV and N220. Of particular interest is the topography of the compensatory relationship, which for our short, highly frequent word stimuli was largely limited to left posterior sites. This topography is in general correspondence with the results of neuroimaging findings reviewed by Pugh et al. (2001) and Temple (2002). The reduced anticipatory activation of brain regions involved in letter string processing was also observed in response to consonant strings but it was topographically more diffuse in that the reduced CNV of dyslexic readers was not limited to left posterior sites but was observed for all posterior sites. Again, as in the case of words, negative correlations between CNV and N220 were suggestive of a compensatory relationship between these two components.

Although the main interest of the present study were early ERP manifestations which may reflect dyslexic difficulties with letter string processing we also note group differences in late ERP components. These were specifically marked for consonant strings where dyslexic readers exhibited a reduced P310 followed by an unexpected N400. This reduced positivity around 300 ms and in particular the occurrence of an N400 suggests that dyslexic readers failed to quickly recognize the input as a nonreadable consonant string and attempted to read it in a phonological manner. A similar observation and interpretation was made by Coch, Maron, Wolf, and Holcomb (2002) who found a marked N400 to consonant strings in less competent readers (10 and 11-year-old children).

For the interpretation of our main finding, that is, the reduced CNV of dyslexic readers, one has to note that the setup of our tasks allowed definite expectations about the timing and the nature of the stimuli. For the expectation about arrival of the stimulus it is relevant that all stimuli of a trial were of rather short duration (725 ± 25 ms) and followed each other in immediate succession. Apparently, the dyslexic readers made less use of the stimulus timing as they did less effectively activate relevant brain regions. The finding that the group difference in the case of words was limited to left posterior sites, whereas in the case of strings it was present at both left and right posterior sites speaks for the topographical specificity of this anticipatory brain activation. The topographical specificity of the CNV was stressed by Rösler, Heil, and Röder (1997) who suggested that the CNV reflects modulation of distinct cortical cell assemblies before they become involved in actual stimulus processing. In studies with dyslexic readers the CNV did not find much attention. However, one study did find a reduced CNV in reading impaired

adolescents (McPherson, Ackerman, Holcomb, & Dykman, 1998) and reduced CNVs were also found in cases of acquired dyslexia (Cohen et al., 2001; Dobel et al., 2002). Further, in a large sample of unimpaired English readers the CNV was found to increase with age and reading competence (Grossi, Coch, Coffey-Corina, Holcomb, & Neville, 2001). The mentioned studies typically used judgment tasks in which a first stimulus signaled arrival of a second stimulus which, for example, has to be judged as rhyming or not.

Our main finding—the reduced CNV of dyslexic readers specifically at posterior left hemisphere sites—has to be interpreted cautiously. A less interesting possibility is that it simply reflects prolonged letter string processing. For example, such prolonged processing may prevent that regions get activated in anticipation of the next stimulus because they are still engaged in processing the given stimulus. The present data do not allow exclusion of this possibility definitively, but also do not provide support. For example, visual inspection of the ERPs in Fig. 2 shows that the build-up of the CNV, which followed the offset of the N400, occurred at about the same time in dyslexic and fluent readers. This suggests that the reduced CNV of dyslexic readers may not simply reflect prolonged word processing, but does reflect an original dysfunction of left posterior brain regions involved in letter string processing. One interpretation of this dysfunction would be that it reflects a biological abnormality of the left posterior brain regions involved in letter string processing. This interpretation would be in line with structural anatomical findings (review by Habib, 2000), however, it is hard to reconcile with the finding that the N220 was enhanced over left posterior regions and no other ERP deficits were observed at these sites. A more functional interpretation would be that the reduced CNV reflects no gross anatomical abnormality, but a sluggish response of left posterior brain regions to the timing of stimulus presentation. More specifically, for fluent readers there is a quick increase in brain activation starting before stimulus onset, whereas for dyslexic readers this increase is slower and prolonged. The slower increase would explain the reduced CNV of the dyslexic readers and the prolonged increase would explain their enhanced N220. In conclusion, our findings suggest that under certain conditions the reduced CNV at distinct brain regions may serve as ERP marker of dysfluent reading. However, these findings are certainly in need of replication and the validity of the interpretation has to be examined.

References

Assadollahi, R., & Pulvermüller, F. (2001). Neuromagnetic evidence for early access to cognitive representations. *Neuroreport, 12*, 207–213.

Baayen, R. H., Piepenbrock, R., & van Rijn, H. (1993). *The CELEX lexical database (CD-ROM)*. Philadelphia, PA: Linguistic Data Consortium, University of Pennsylvania.

Behrmann, M. (1999). Pure Alexia: Underlying mechanisms and remediation. In R. M. Klein, & P. McMullen (Eds.), *Converging methods of understanding reading and dyslexia* (pp. 153–189). Cambridge: MIT Press, 153–189.

Bentin, S., Mouchetant-Rostaing, Y., Giard, M. H., Echallier, J. F., & Pernier, J. (1999). ERP manifestations of processing printed words at different psycholinguistic levels: Time course and scalp distribution. *Journal of Cognitive Neuroscience*, *11*(3), 235–260.

Brandeis, D., Vitaccio, D., & Steinhausen, H. C. (1994). Mapping brain electric micro-states in dyslexic children during reading. *Acta Paedopsychiatrica*, *56*, 239–247.

Breznitz, Z., & Leikin, M. (2001). Effects of accelerated reading rate on processing word's syntactic functions by normal and dyslexic readers: Event related potential evidence. *Journal of Genetic Psychology*, *162*(3), 276–296.

Breznitz, Z., & Misra, M. (2003). Speed of processing of the visual-orthographic and auditory-phonological systems in adult dyslexics: The contribution of 'asynchrony' to word recognition deficits. *Brain and Language*, *85*, 486–502.

Coch, D., Maron, L., Wolf, M., & Holcomb, P. J. (2002). Word and picture processing in children: An event-related potential study. *Developmental Neuropsychology*, *22*(1), 373–406.

Cohen, R., Dobel, C., Berg, P., Köbbel, P., Schönle, P. W., & Rockstroh, B. (2001). Event-related potential correlates of verbal and pictorial feature comparison in aphasics and controls. *Neuropsychologia*, *39*(5), 489–501.

Csepe, V., Szücs, D., & Honbolygo, F. (2003). Number-word reading as challenging task in dyslexia? An ERP study. *International Journal of Psychophysiology*, *51*, 69–83.

Dobel, C., Cohen, R., Berg, P., Nagl, W., Zobel, E., Köbbel, P., et al. (2002). Slow event-related brain activity of aphasic patients and controls in word comprehension and rhyming tasks. *Psychophysiology*, *39*(6), 747–758.

Farah, M. J. (1999). Are there orthography specific brain regions? Neuropsychological and computational investigations. In R. M. Klein, & P. McMullen (Eds.), *Converging methods of understanding reading and dyslexia* (pp. 221–243). Cambridge: MIT Press, 221–243.

Grossi, G., Coch, D., Coffey-Corina, S., Holcomb, P. J., & Neville, H. J. (2001). Phonological processing in visual rhyming: A developmental ERP study. *Journal of Cognitive Neuroscience*, *13*(5), 610–625.

Habib, M. (2000). The neurological basis of developmental dyslexia: An overview and working hypothesis. *Brain*, *123*(12), 2373–2399.

Hari, R., & Renvall, H. (2001). Impaired processing of rapid stimulus sequences in dyslexia. *Trends in Cognitive Sciences*, *5*, 525–532.

Helenius, P., Salmelin, R., Service, E., & Connolly, J. F. (1999). Semantic cortical activation in dyslexic readers. *Journal of Cognitive Neuroscience*, *11*(5), 535–550.

Helenius, P., Tarkiainen, A., Cornelissen, P., Hansen, P. C., & Salmelin, R. (1999). Dissociation of normal feature analysis and deficient processing of letter-strings in dyslexic adults. *Cerebral Cortex*, *9*(5), 476–483.

Huttenlocher, J., & Cohen-Levine, S. (1990). *Primary test of cognitive skills*. Monterey, CA: Macmillan.

Klimesch, W., Schack, B., Schabus, M., Doppelmayr, M., Gruber, W., & Sauseng, P. (2004). Phase-locked alpha and theta oscillations generate the P1–N1 complex and are related to memory performance. *Cognitive Brain Research*, *19*(3), 302–316.

Landerl, K., Wimmer, H., & Frith, U. (1997). The impact of orthographic consistency on dyslexia: A German-English comparison. *Cognition*, *63*, 315–334.

Leikin, M. (2002). Processing syntactic functions of words in normal and dyslexic readers. *Journal of Psycholinguistic Research*, *31*(2), 145–163.

McCandliss, B. D., Cohen, L., & Dehaene, S. (2003). The visual word form area: Expertise for reading in the fusiform gyrus. *Trends in Cognitive Sciences*, *7*(7), 293–299.

McPherson, W. B., Ackerman, P. T., Holcomb, P. J., & Dykman, R. A. (1998). Event-related brain potentials elicited during phonological processing differentiate subgroups of reading disabled adolescents. *Brain and Language*, *62*, 163–185.

Neville, H. J., Coffey, S. A., Holcomb, P. J., & Tallal, P. (1993). The neurobiology of sensory and language processing in language impaired children. *Journal of Cognitive Neuroscience*, *5*(2), 235–253.

Nobre, A. C., Allison, T., & McCarthy, G. (1994). Word recognition in the human inferior temporal lobe. *Nature*, *372*, 260–263.

Pugh, K. R., Mencl, W. E., Jenner, A. R., Katz, L., Frost, S. J., Lee, J. R., et al. (2001). Neurobiological studies of reading and reading disability. *Journal of Communication Disorders*, *34*(6), 479–492.

Robichon, F., Besson, M., & Habib, M. (2002). An electrophysiological study of dyslexic and control adults in a sentence reading task. *Biological Psychology*, *59*, 29–53.

Rösler, F., Heil, M., & Röder, B. (1997). Slow negative brain potentials as reflections of specific modular resources of cognition. *Biological Psychology*, *45*, 109–141.

Salmelin, R., Service, E., Kiesilä, P., Uutela, K., & Salonen, O. (1996). Impaired visual word processing in dyslexia revealed with magnetoencephalography. *Annals of Neurology*, *40*, 157–162.

Sauseng, P., Bergmann, J., & Wimmer, H. (2004). When does the brain register deviances from standard word spellings?—An ERP study. *Cognitive Brain Research*, *20*, 529–532.

Stelmack, R. M., Saxe, B. J., Noldy-Cullum, N., Campbell, K. B., & Armitage, R. (1988). Recognition memory for words and event-related potentials: A comparison of normal and disabled readers. *Journal of Clinical and Experimental Neuropsychology*, *10*(2), 185–200.

Temple, E. (2002). Brain mechanisms in normal and dyslexic readers. *Current Opinion in Neurobiology*, *12*(2), 178–183.

Wimmer, H. (1993). Characteristics of developmental dyslexia in a regular writing system. *Applied Psycholinguistics*, *14*, 1–33.

Wimmer, H. (1996). The nonword reading deficit in developmental dyslexia: Evidence from children learning to read German. *Journal of Experimental Child Psychology*, *61*(1), 80–90.

Wimmer, H., Hutzler, F., & Wiener, C. (2002). Children with dyslexia and right parietal lobe dysfunction: Event-related potentials in response to words and pseudowords. *Neuroscience Letters*, *331*, 211–213.

Ziegler, J. C., Perry, C., Ma-Wyatt, A., Ladner, D., & Schulte-Körne, G. (2003). Developmental dyslexia in different languages: Language-specific or universal? *Journal of Experimental Child Psychology*, *86*, 169–193.

Zoccolotti, P., De Luca, M., Di Pace, E., Judica, A., Orlandi, M., & Spinelli, D. (1999). Markers of developmental surface dyslexia in a language (Italian) with high grapheme–phoneme correspondence. *Applied Psycholingusitics*, *20*, 191–216.