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Neurophysiological correlates of word recognition in dyslexia

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Summary. The neurobiological basis of learning word spellings and recognition of recently learned words was assessed in a learning experiment in 9 dyslexics and 9 controls male adolescents. In a recognition paradigm previously learned pseudowords and graphic symbols were presented 50 times each interspersed pseudo-randomly between 3 unlearned items which were repeated 50 times and 150 filler pseudowords. The electrophysiological correlate of recognition of learned pseudowords the amplitude of this ERP component was significantly attenuated in the dyslexic group, no differences between the groups were found for recognition of graphic material. These data suggest that dyslexic children are able to learn the spelling of simple words, however, the neurophysiological correlate of recognition of these learned words is significantly attenuated. This result strengthens the view that dyslexic children are not generally impaired in recognition memory but specific for linguistic material like words.

Keywords: Dyslexia, learning, visual evoked potentials, word recognition, P600.

Introduction

Dyslexia is a specific disorder in learning to read and spell in spite of adequate educational resources, a normal IQ, no obvious sensory deficits, and adequate sociocultural opportunity (Dilling et al., 1991). Dyslexia occurs in all languages and especially spelling disorder often persists into adulthood (Schulte-Körne et al., 2003). 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 (Schulte-Körne, 2001).

There is an ongoing discussion about the etiology of dyslexia. A great amount of research has focused on basic perceptual deficits (Habib, 2000; Ramus et al., 2003) which yielded conflicting results. It has been demonstrated

that dyslexics are handicapped with processing of auditory (Ramus et al., 2003) and visual stimuli (Amitay et al., 2002). However, in a recent review it was concluded that sensory and motor coordination deficits are of only minor relevance for the causal explanation of dyslexia (Ramus, 2003).

Therefore, this paper follows a different approach. The central aspects of spelling acquisition, learning to spell and recognition of recently learned words and its neurobiological correlates, have been hardly considered by research on dyslexia.

One common practice in the teaching of spelling involves memorizing weekly lists of spelling words (Graham et al., 1994). During elementary years a child is explicitly taught approximately 3800 words (Graham et al., 1996). The main assumption is that spelling ability develops by learning weekly word lists and by exposure to written text (Henderson and Chard, 1980; Stanovich, 1986). Although some researchers found that learning weekly spelling lists was not a major factor in the development of good spellers (Hughes and Searle, 1997), memorizing word lists is recommended as a part of an effective spelling curriculum and also as a coping strategy for learning disabled children (Graham et al., 1994; Scott, 2000). For dyslexic children the number of required weekly spelling words is lower than for children without a learning disorder, though (Graham and Voth, 1990).

Teachers and parents know the phenomenon that spelling disabled children are able to spell a word correctly immediately after having learned it, but memory of the correct spelling fades rapidly. Sometimes, just one or two hours after having learned a word, dyslexics are unable to spell the word correctly. Dreyer et al. (1995) found that poor spellers have more difficulties remembering than learning correct spellings. They found that immediately after a spelling lesson above and below average spellers did not differ. However, one week later both groups differed significantly because the accuracy of spelling in the below average spellers group declined significantly, while the scores of the above average spellers remained largely stable. These observations raise the question why dyslexics seem to forget learned spellings so quickly, and they lead to the hypothesis that dyslexics have a memory deficit for word spellings.

One procedure to examine memory of word learning is word recognition. Especially the examination of the underlying physiology of word recognition (which might be disturbed in dyslexics) could help to better understand the etiology of reading and spelling disorder. Event related potentials are best suited for temporal analysis of cognitive processes, yielding information not available from behavioral measures. A number of studies dealt with electrophysiological correlates of recognition of previously studied material. A component of the event-related potentials (ERPs) between 300 and 800 ms post stimulus onset were found to have a larger positive amplitude to studied than to new stimuli. The P600 has been repeatedly found to be a neurophysiological correlate of word recognition (Curran, 1999; Allan et al., 1998).

Only two electrophysiological studies examining word recognition in a learning paradigm in dyslexics have been published (Stelmack et al., 1988; Stelmack and Miles, 1990). They examined the ERPs elicited by hits and correct rejections in word recognition in dyslexics and controls. The P600 was found to be a correlate of word recognition over central, temporal, parietal, and

occipital leads. In the first study, Stelmack et al. (1988) found no group differences in P600 amplitude or latency at Pz elicited by hits of word recognition. In the second study (picture prime condition) the amplitude of the P600 for hits in word recognition was significantly larger in controls than in dyslexics. This ERP finding – although in contrast to the first study – suggests a long-term memory deficit for recently seen linguistic material in dyslexics.

However, there are some shortcomings of these studies. The experiments do not reflect a natural (ecological) learning situation because the words which had to be remembered were shown only once. Normally school children are presented new words repeatedly to learn the spellings.

Further the error rates in reading words were significantly larger for the dyslexics than for controls in both studies. This could mean that the observed group difference in word recognition is partly based on the dyslexics' word reading deficit and can not be differentiated from a word recognition deficit of recently seen words.

The present experiment exploits the neurophysiological correlates of word recognition of previously intensively studied words in dyslexic and control school age children. Several methodological constraints of the above mentioned studies were considered and the experiment was configured to provide conditions which give students best opportunities to learn the spellings of new words. This means that first, the word material chosen for learning was easy to read, consequently dyslexics were able to read these words without problems, avoiding an influence of reading deficits on word recognition.

Second, words could be intensively studied without a time constraint. Since dyslexic students often need more time to learn new words, this procedure provides enough time to learn the new spellings.

Third, we used pseudowords as material because an influence of word frequency has been found on the P600 in word recognition (e.g. Rugg, 1990). A word frequency effect has been found on ERPs in dyslexics (Johannes et al., 1995), and dyslexics differ from controls regarding their familiarity with word material due to their reading disorder. This fact might lead to an underestimation of the dyslexics' word recognition ability.

Fourth, we used a control condition with graphic material that could not be phonologically decoded.

Our first hypothesis (hypothesis 1) was that dyslexics have an attenuated amplitude of the P600 elicited by recently learned pseudowords, but not for graphic symbols. Based on the clinical observation of a rapidly fading memory in dyslexics for recently learned word spellings, the same procedure was carried out again after a two hours break. This was to investigate whether there were increasing group differences of the P600 amplitude for pseudowords (hypothesis 2).

Methods

Subjects

Dyslexic adolecents were ascertained through a special boarding school for dyslexics and visited the same high school as the control children. From this high school 26 adolescents (dyslexics and

controls) from 10th grade were chosen to participate in the study. Controls were selected by high school teachers based on students performance in reading and spelling. Dyslexic adolescents were chosen by teachers from the boarding school.

Inclusionary criteria were to be a native monolingual speaker of German, no uncorrected visual acuity, and no apparent neurological, emotional, or behavioral deficits (e.g. ADHD) or unusual educational circumstances that could account for poor reading and spelling ability. Spelling was measured by an age-appropriate German spelling test (Jäger and Jundt, 1981) and spelling disorder was diagnosed if there was a discrepancy of at least 2 standard deviations between actual spelling ability and that predicted on the basis of IQ (linear regression model, Schulte-Körne et al., 1996, 2001a). The spelling ability of the control group was in the normal range (see below). Reading ability could not be assessed because there are no standardized German reading tests for adolescents or adults. However, the dyslexics had been assessed with a reading test when they had entered the boarding school (5th grade), and at that time word decoding had been below average. Control subjects reported never to have had difficulties in word decoding and reading comprehension. All subjects reported themselves to be strongly right-handed. From the 26 adolescents only 9 students fulfilled the inclusionary criteria for dyslexia and 9 for controls (see above).

9 dyslexics (mean age 17.4 ± 0.6 , mean IQ 114.5 ± 8.8 , mean percentile rank in spelling test 4,7) and 9 controls (mean age 16.7 ± 0.7 , mean IQ 112.0 ± 19.6 , mean percentile rank in spelling test 42) were assessed (only boys).

Both groups were very similar regarding their non-verbal IQ (measured by German adaptation of the Culture Fair Intelligence Test, CFT20; Weiß, 1987) and their age.

Material

Two types of stimuli were used in two experiments: pseudowords and graphic symbols.

Pseudowords were randomly constructed out of four letters (consonant – vowel – consonant – vowel). Pseudowords resembling real words were manually excluded. In a pre-test with dyslexics of the same age group as our probands we ensured that the pseudowords were easy enough to read so that even dyslexics had no reading problems with these stimuli.

To avoid any similarities with letters, the graphic symbols of the control condition consist of 4 * 3 squares with a total of 4 squares containing a dot (see Fig. 1). Items resembling familiar symbols (e.g. cross) or letters were excluded.

Procedure

Subjects sat in a darkened room (average luminance of 1.2 cd/m^2) with 60 cm viewing distance to an EIZO 21' computer monitor.

The following procedure was carried out for pseudowords and graphic symbols in the same way. The order of the type of material was counterbalanced between groups and conditions.

In part one of the experiment (learning phase), three items (pseudowords: GIMO, BATU, MOFO) were presented simultaneously on the computer screen. The subject was first instructed to memorize the three items (pseudowords or graphic symbols, and then asked to reproduce them (pseudowords: spell them by clicking with the cursor on letters of a keyboard presented on the screen; graphic symbols: placing the dots in the correct cells by clicking on the cells of an empty 4 * 3 squares grid on the screen). Half of the probands started with the pseudowords, the other half



Fig. 1. Three graphic symbols which have to been learned

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with the graphic material. The learning phase ended when all three pseudowords or graphics symbols had been correctly reproduced in three successive trials.

In part two of the experiment (recognition phase 1), 450 stimuli were presented to the subject in random order. 150 stimuli comprised the previously learned 3 items, each presented 50 times (targets); 150 comprised 3 unlearned items, each presented 50 times (non-targets). The non-targets were used to control for unspecific effects which might have influenced the neurophysiological correlates of word recognition. 150 unlearned items were presented as filler words.

Each item was presented for 300 ms, followed by an ISI of 1700 ms in which the subjects had to press the left mouse button if they recognized a previously learned item, or the right mouse button for a new, unlearned item.

Since we were specifically interested in the long-term aspect of this learning paradigm, this part of the experiment was repeated after 2 hours. Thus, part three of the experiment (recognition phase 2) was the same as part two, but conducted two hours after the end of part two of the study. In the meantime the probands were offered food and beverages.

It is important to keep in mind that all probands were able to reproduce the stimuli after the learning phase. All but one subject managed to reproduce all pseudowords correctly at the first attempt, and all subjects managed to reproduce all graphic symbols correctly at the first attempt. This means that the type of material and number of items chosen was easy enough to ensure that even dyslexics could learn the stimuli in a short time. Also, the behavioral data (correctly identified targets and correctly identified non-targets) were analyzed (Repeated Measures ANOVA, factors group (dyslexics vs. controls), time (recognition phase one vs. two), and material (pseudowords vs. graphic symbols). There were no significant effects, indicating that the material was indeed easy enough to learn so that the dyslexics could gain the same results as the controls.

Measurements and analyses

EEG was measured with additional markers for stimulus onset and the subject's reaction (mouse button pressed). Electrodes were placed at 30 scalp sites based on the International 10-20 System: Fp1, Fp2, F7, F3, Fz, F4, F8, FT7, FC3, FCz, FC4, FT8, T3, C3, Cz, C4, T4, TP7, CP3, CP2, CP4, TP8, T5, P3, Pz, P4, T6, O1, Oz, O2 (referred to left mastoid, ground electrode at Fpz). Eye movements and blinks were monitored by four additional electrodes. The EEG was amplified with Neuroscan amplifiers, lower frequency cut-off 0.1 Hz; 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 (www.brainproducts.com). The signals were averaged into epochs of 1100 msec, including a prestimulus baseline of 100 msec. Artefact-free epochs of correctly identified items were averaged into the categories targets and non-targets. Peak amplitudes and latency of the P600 ERP component were exported and analyzed with SAS software. Huynh-Feldt correction of p values was applied when the sphericity assumption was rejected (Mauchly's test), and the reported p values are one-sided if they refer to our hypotheses. Since we were expecting maximal ERP amplitudes over central-parietal leads, ERPs over central (C3, Cz, C4), central-parietal (CP3, CPz, CP4), and parietal leads (P3, Pz, P4) were analysed. Mean amplitude and mean latency (mean value of all nine electrodes) were analysed.

In order to account for possible confounding variables which might influence the hypothesized group differences a difference curve was analysed. For this analysis the ERPs of correctly recognized non-target were subtracted from correctly recognized target ERPs. This procedure takes care of two effects we do not want to interfere with our main analysis: first, if a stimulus is presented repeatedly, this is likely to change the processing of the stimulus even if there is no conscious memory (Wiggs and Martin, 1998). This effect has been called implicit memory or repetition priming (Rugg et al., 1998). The neurophysiological correlate of implicit memory is a positive going amplitude 250–600 ms post stimulus, which is larger for repeated compared to new stimuli (Rugg, 1990). Since we are interested to investigate explicit word recognition, i. e. the recognition of recently learned words and not of repeatedly presented word, the calculation of the difference curve reduced the influence of repeatedly presented material (see Results, Fig. 2). Second, children had to differentiate learned from unlearned material. This requires attention allocation and stimulus discrimination. One component which is most associated with this cognitive process is the P300 (Johnson, 1985). Again because we are interested to examine the

neurophysiological correlate of recognition of recently learned material and not of the discrimination process of learned and unlearned words the statistical analyses is based on the difference curve.

Results

Figure 2 shows the ERPs for the three pseudoword conditions: recognition of recently learned words, of unlearned words (repeated like learned words) and unlearned words (not repeated). In all three conditions a clear component at a latency of 300 ms is apparent. The amplitude and latency of the P300 seem to be very comparable for all three pseudoword categories, but only with the recognition of learned pseudowords a late positive component with a latency about 600 ms (P600) with maximum amplitude over Pz can be identified.

Because probands had to discriminate repeatedly presented learned from unlearned stimuli and this discrimination process reveals a P300 as a neurophysiological correlate of this discrimination process, the difference curve of correctly recognized pseudowords (learned) minus correctly recognized unlearned pseudowords (repeatedly presented, unlearned) (Figs. 3 and 4, Table 1) was analysed. The inspection of the difference curve (Figs. 3 and 4) reveals only one component of the ERP with a latency at around 600 ms (P600). The positivity at around a latency of 300 ms disappears. The amplitude of the late positivity was analysed first.



Fig. 2. Grand mean ERP (control group) for recognition of learned pseudowords (bold line), unlearned pseudowords repeatedly presented (dashed line) und unlearned pseudowords presented once (dotted) at recognition phase 1

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Fig. 3. Grand mean ERP (difference curve of correctly recognized pseudowords minus correctly recognized unlearned pseudowords) for dyslexics (dashed line) and controls (bold line) at recognition phase 1 for pseudowords



Fig. 4. Grand mean ERP (difference curve of correctly recognized pseudowords minus correctly recognized unlearned pseudowords) for dyslexics (dashed line) and controls (bold line) at recognition phase 1 for graphic symbols

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		Recognition phase 1		Recognition phase 2	
		Controls	Dyslexics	Controls	Dyslexics
Pseudowords	amplitude latency	7.0 ± 2.1 609.8 ± 61.6	5.0 ± 1.3 609.8 ± 40.1	9.3 ± 3.1 571.2 ± 17.7	7.0 ± 2.8 598.5 ± 52.1
Graphic Symbols	amplitude latency	$5.6 \pm 1.3 \\ 689.9 \pm 50.2$	$\begin{array}{c} 7.1 \pm 2.6 \\ 680.0 \pm 45.4 \end{array}$	7.4 ± 2.4 675.8 ± 47.0	6.8 ± 4.4 690.9 ± 37.8

 Table 1. Means and standard deviations of peak amplitudes and peak latencies (difference curves) of all experimental conditions (pseudowords and graphic symbols, recognition phase 1 and 2, controls and dyslexics)

The analysis of the amplitude yielded no significant main effects group (p=0.39) and material (p=0.58). In support of hypothesis 1 (attenuated amplitude of the P600 in dyslexics specific for pseudoword recognition) we found a significant interaction between material and group (p=0.032). Looking at the means (Table 1) one sees that there is a comparably large group difference for the pseudoword condition, but no such difference for the graphic symbols condition. Hence, the significant interaction stems from a significant group effect for pseudowords, and the absence of such an effect for graphic symbols. Posthoc t-Tests support this view: the p values are 0.031 (pseudowords) and 0.73 (graphic symbols). Figure 5 illustrates this effect.

To illustrate the distribution of brain activity for the two conditions (pseudowords and graphic material) spherical spline interpolation was used to generate scalp topographies of ERP measures (Figs. 6 and 7). The brain maps show that



Fig. 5. Illustration of the material by group interaction, data of recognition phase 1 and 2 and of electrode position were averaged

Fig. 6. Scalp topography for the difference curve in the pseudoword condition, above controls, below dyslexics at recognition phase 1

Fig. 7. Scalp topography for the difference curve in the graphic material condition, above controls, below dyslexics at recognition phase 1



the main activity at the latency between 600 and 700 ms was registered over parietal cortical areas. The distribution is comparable between the two conditions, however the group differences in the pseudoword condition is clearly visible.

The main time effect is also significant (p = 0.0019) indicating that amplitudes of the P600 are generally larger at recognition phase 2. Further we found a significant interaction between material and time (p = 0.032) indicating that the amplitude of the P600 is larger for pseudowords and graphic symbols at recognition phase 2 than recognition phase 1.

We did not find evidence for our second hypothesis (larger difference between the groups only for pseudowords at recognition phase 2) because the three-fold interaction group * material * time was not significant (p = 0.073, one-sided test).

Second we analyzed the latency of the P600 latency which yielded one significant effect, a main effect material (p < 0.0001). This means that the latency of the late positivity is significantly shorter in the pseudoword condition in comparison to the graphic material condition.

Discussion

Summary of the results

Aim of this study was to examine the neurophysiological correlates of word recognition of recently learned new words in dyslexic adolescents and controls. This implied that the learning situation and the complexity of the items had to be chosen so that dyslexics were able to learn the items sufficiently well. Therefore three simple pseudowords (four letters: C-V-C-V) had to be learned to spell with no time constraints. This procedure makes sure that even the dyslexics were able to learn the items in order to be able to analyze the correlates of recognition of recently learned material. Thus, we assessed dyslexics in a situation where they did not actually fail to perform a task.

As a neurophysiological correlate of pseudoword and graphic material recognition we found a late positivity at a latency of 600 ms. While the design of the study aims at explicit memory, the experimental condition of repeated presentation of items inevitably produces effects of repetition priming, which overlay explicit memory ERPs. In order to be able to separate these effects, we also ran a non-target condition (previously unlearned stimuli) which produces repetition priming ERPs. Furthermore, recognition of learned and unlearned stimuli require discrimination processes. A neurophysiological correlate of this is the P300 which we found in the target as well as in the non-target conditions. Therefore statistical analysis was based on difference curves (learned minus unlearned stimuli). These difference curves (for pseudowords and graphic material) are expected to represent explicit memory for recently learned material only.

With respect to our first hypothesis, the analysis yielded a two-fold interaction of material and group: P600 elicited by correctly recognized pseudowords is larger in controls than in the dyslexic group, while the P600 for graphic symbols does not significantly differ between the groups. The dyslexics had an attenuated P600 to pseudowords although they had learned the items successfully before. The largest group differences in the pseudoword condition were found over parietal leads. This findings corresponds well to the findings of Rüsseler et al. (2003) who found that the ERP component in a latency window of 450–650 ms for correctly recognized words was attenuated over centroparietal electrodes in dyslexic adults (Rüsseler et al., 2003).

The second hypothesis (increasing group differences over time) could not be confirmed. A possible evaluation of this result is that an interval of two hours is too short to alter the memory traces significantly. The short interval was chosen on the basis of clinical experience. The only study that explored this effect used a much longer interval though (one week; Dreyer et al., 1995). Therefore, it could be that despite clinical intuition the interval chosen was too short to prove such an effect.

The significant time main effect (higher P600 two hours later for both materials and both groups) indicates though that some alteration has taken place. This could be due to memory consolidation or might be a result of higher familiarity with the experimental design. To our best knowledge, this is the first electrophysiological study assessing re-test effects on item recognition. Therefore, it is quite difficult to evaluate this effect since there are no studies we can compare our results with.

Specific memory deficit in dyslexia?

The ERP findings of this study encourage the view of a specific word recognition memory deficit in dyslexia. The P600 was found to be a specific correlate of recognition memory. Because old words misclassified as new and new words misclassified as old did *not* elicit the P600 old/new potential, the P600 is considered an electrophysiological correlate of correctly recognized words only (Smith, 1993; Wilding et al., 1996).

The amplitude of the P600 is supposed to reflect the quality or amount of information retrieved (Rugg et al., 1995). For our results this means that recognition memory for pseudowords is inferior in dyslexics when compared to controls. This fact seems at first sight surprising because both groups were able to learn the items. On the other hand, the ability (or the lack thereof) to learn those items is just a dichotomous behavioral measure of the underlying cortical representation. It can be speculated that the attenuated P600 for pseudowords points to a deficit of word recognition memory, although not to the degree of failing to recognize the items.

The question arises why it is that the P600 in dyslexics is impaired with recognizing pseudowords but not with recognizing graphic symbols. One might argue that reading requires the application of phoneme-grapheme rules because orthography and phonology are closely related and developmentally connected (Ehri, 1980). But as proposed by several researchers (e.g. Seidenberg, 1985), phonology is mainly activated for reading and spelling of orthographically illegal words. The pseudowords used in our study are, however, orthographically legal. Therefore it seems sound to assume that phonology did not play a major role for recognizing the pseudowords in our experiment. If this holds true, it must be the visual recognition of pseudowords that is impaired in our dyslexic group.

Several researchers have proposed the existence of a visual memory for sequences of letters within a word (Ehri, 1980) and that recognition of spelling patterns is mainly based on visual memory for parts of words (Johnstone and Shanks, 2001). In a recent work, Pelli et al. (2003) pointed out that the human brain does not identify entire words, but instead has efficient templates only for recognizing parts of words. Our results suggest that this process of identifying parts of words might be impaired in dyslexics.

Clinical evidence

We find that dyslexics have an attenuated P600 on pseudoword recognition. Inevitably, the material chosen had to be easy enough so that even dyslexics would be able to learn the pseudowords successfully. This was a requirement that had to be met in order to be able to compare ERPs. Therefore, our data provide evidence for a functional deficit in a sub-clinical range where, on a behavioral level, dyslexics did not (yet) fail. It seems plausible though to extend the result to the clinical range, i.e. it can be speculated that an attenuated word recognition potential is also involved when the material is more difficult so that dyslexics are no longer able to learn it as well (on the behavioral level) as controls. If this is true it means that dyslexics are generally inferior to controls with respect to visual pseudoword (and word) recognition.

Spelling is often taught as an intensive exercise in memorizing letter sequences for specific words (Graham et al., 1994) leading to a long term memory for words and their spellings. This could simply be the wrong angle if it is visual word recognition that is impaired. Thus simple training of word memory might not be a useful coping strategy for dyslexics. However, we do agree with several authors (Lovett et al., 1990; Berninger et al., 1998; Graham, 1999) that spelling training should integrate training in subsyllabic segmentation, functional spelling units, and phonological awareness. In addition, rule-based learning has also been recommended and has been proven effective for dyslexic children (e.g. for German language by Schulte-Körne et al., 1998, 2001b).

The finding that the cortical correlates for the recognition of learned graphic symbols are comparable in dyslexic and control children alludes to training of visual discrimination and other solely visual abilities to ameliorate reading and spelling ability. E.g., visual form perception training is an integrated part of the optometric vision therapy which has been recommended for dyslexic children (American Academy of Optometry and the American Optometric Association). Although the graphic material in our study is different from material typically used in figure-ground discrimination tasks, children had to discriminate and remember complex visual patterns which apparently did not raise a problem for the dyslectic group. Thus recommending training of visual spatial discrimination dyslexics should be questioned.

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