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IN order to investigate the relationship between dyslexia and central auditory processing, 19 children with spelling disability and 15 controls at grades 5 and 6 were examined using a passive oddball paradigm. Mismatch negativity (MMN) was determined for tone and speech stimuli. While there were no group differences for the tone stimuli, we found a significantly attenuated MMN in the dyslexic group for the speech stimuli. This finding leads to the conclusion that dyslexics have a specific speech processing deficit at the sensory level which could be used to identify children at risk at an early age.

Auditory processing and dyslexia: evidence for a specific speech processing deficit

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Introduction

Dyslexia is a specific disability in learning to read and spell in spite of adequate educational resources, a normal IQ, no obvious sensory deficits and adequate sociocultural opportunity.¹ Dyslexia occurs in all languages and spelling disability in particular often persists into adulthood.⁵ Prevalence estimates range from 4 to 9%.²

A consensus has emerged that a deficit in phonological processing is a major cause for dyslexia.³ Phonological processing involved different aspects of linguistic awareness, including phoneme identification, phoneme discrimination and verbal short-term memory.

For all phonological processing tasks, speech perception is a prerequisite condition. If dyslexics have difficulties in speech perception, a less precise phonological representation may be the result and would lead to further difficulties in complex verbal processing tasks. In several studies, significant group differences between dyslexic children and normals have been found regarding the categorical perception of synthetic /ba/–/da/–/ga/ syllables mainly differing in the transitions of the second and third formant.^{4–6} The procedures in these studies were stimulus identification and discrimination, which required subjects to focus their attention on the relevant stimulus dimension, especially if stimuli were masked by amplitude-matched noise.⁷

The question arises whether the speech perception deficit of the dyslexics already occurs at the level of sensory perception which is characterized by pre-attentive and automatic processing.

A neurophysiological paradigm that is best suited to examine pre-attentive and automatic central auditory processing is the mismatch negativity (MMN). The MMN is a negative component of the ERP, elicited when any discriminable change occurs in a sequence of repetitive homogeneous auditory stimuli.⁸ The MMN occurs ~100–300 ms post-stimulus onset and is elicited by changes in frequency, intensity, or duration of tone stimuli, or changes in complex stimuli such as phonetic ones. The MMN is assumed to be a result of a mechanism that compares each current auditory input with a trace of recent auditory input stored in the auditory memory. The MMN usually reaches its amplitude maximum over the fronto-central scalp.

The aim of this study was to determine the relationship between dyslexia and central auditory processing. To examine whether the speech perception deficits of dyslexics are pre-attentive and automatic, we used a passive oddball paradigm, which requires the subjects to focus their attention on a sensory modality (i.e. watching a silent movie) other than that of the test stimuli. To elicit an MMN we used speech stimuli as well as tone stimuli. The tone stimuli serve as a control condition to examine whether the central auditory perception dysfunction

is specific for speech stimuli. The hypothesis is that the MMN of dyslexics, compared with that of controls, is attenuated in the speech condition but not in the sine wave condition.

Materials and Methods

Nineteen spelling-disabled (mean age 12.5 ± 0.3 years) and 15 control children (mean age 12.6 ± 0.8 years) at grades 5 and 6 were assessed (only boys). The two groups did not differ regarding their IQs (IQ of spelling disabled 104 ± 9.7 ; IQ of controls 104.6 ± 14). The spelling-disabled children visited the same high school as the control children and were ascertained through a special boarding school for dyslexics. Inclusionary criteria were to be a native monolingual speaker of German, no middle-ear infection within the week of testing, no hearing problems and no uncorrected visual acuity, no apparent neurological, emotional or behavioural deficits or unusual educational circumstances that could account for poor reading and spelling ability. Spelling disability was assumed if there was a discrepancy of ≥ 1 s.d. between actual spelling ability and expected spelling based on IQ.⁹

Additionally, the spelling disabled group had a significantly lower word decoding ability than the controls ($p = 0.01$). This group is referred to as 'dyslexic'.

All subjects had normal hearing and reported themselves to be strongly right-handed according to a handedness questionnaire.¹⁰ Acoustic stimuli were produced by 90 ms of 1000 Hz ('standard', $p = 0.85$) and 1050 Hz (deviant, $p = 0.15$) sine waves (including 3 ms rise and 3 ms fall time) and were presented in a pseudorandom order (at least five standards between two deviants) with a constant ISI of 590 ms (from onset to onset). Speech stimuli (standard /da/, deviant /ba/), adopted from Heinz and Stark¹¹ were synthesized with the Computerised Speech Research Environment.¹²

The auditory stimuli were presented binaurally by headphones. To control for level of arousal and to minimize subjects' attention to the stimuli, they were instructed to watch videotaped silent movies and to ignore the test stimuli. Subjects were instructed to follow the screen play and to answer several questions on topics of the movies after the EEG recording.

Electrodes were placed at 19 scalp sites based on the International 10-20 System: Fp1, Fp2, F7, F8, F3, F4, Fz, C3, C4, Cz, T3, T4, T5, T6, P3, P4, Pz, O1, O2 (referred to linked ears, ground electrode at Fpz). Eye movements and blinks were monitored by two electrodes placed below the subjects' right and left eyes and the Fp1 and Fp2 electrodes. The EEG was

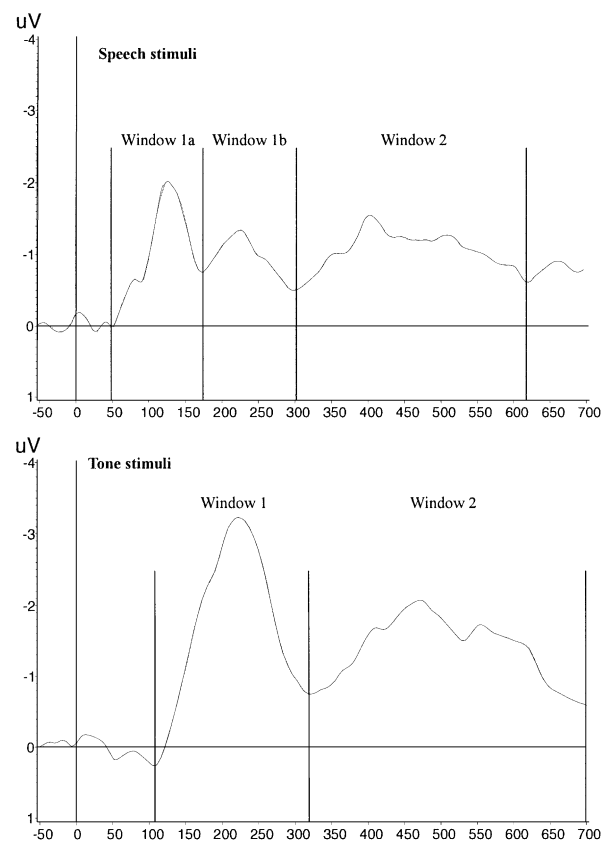


FIG 1. Grand average curves of the MMN for speech and tone stimuli.

amplified with Schwarzer amplifiers, time constant 0.6 s; upper frequency cut-off at 85 Hz. The EEG was recorded continuously and A/D converted at a sampling rate of 172 Hz. The signals were averaged into epochs of 750 ms, including a prestimulus baseline of 50 ms. Difference waveforms were calculated by subtracting ERPs to standards from those to deviants.

In order to define the time window for the MMN potential, the difference curves (standard-deviant) at Fz were plotted (grand average of all subjects).

For the tones we found two distinguishable components (110–319 ms, window 1, and 320–700 ms, window 2), and for the speech stimuli we found three distinguishable components (47–175 ms, window 1a, 176–302 ms, window 1b, and 303–620 ms, window 2). Visual inspection of the individual MMN curves revealed evidence for more than one peak in window 2 for both tone and speech stimuli, therefore we chose areas and not amplitudes for all windows as measures for further data analyses. Mean areas ($\mu\text{V} \times \text{ms}$) were calculated for each component.

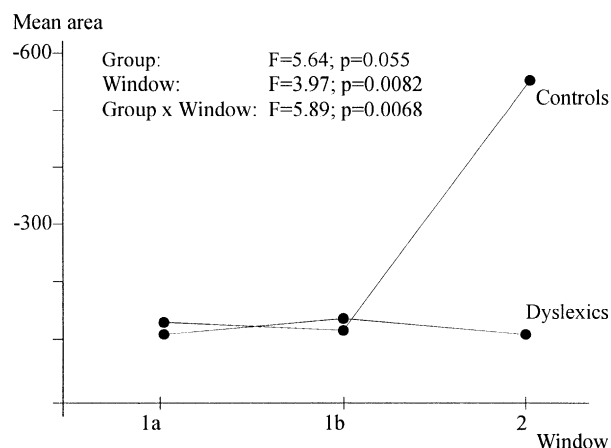
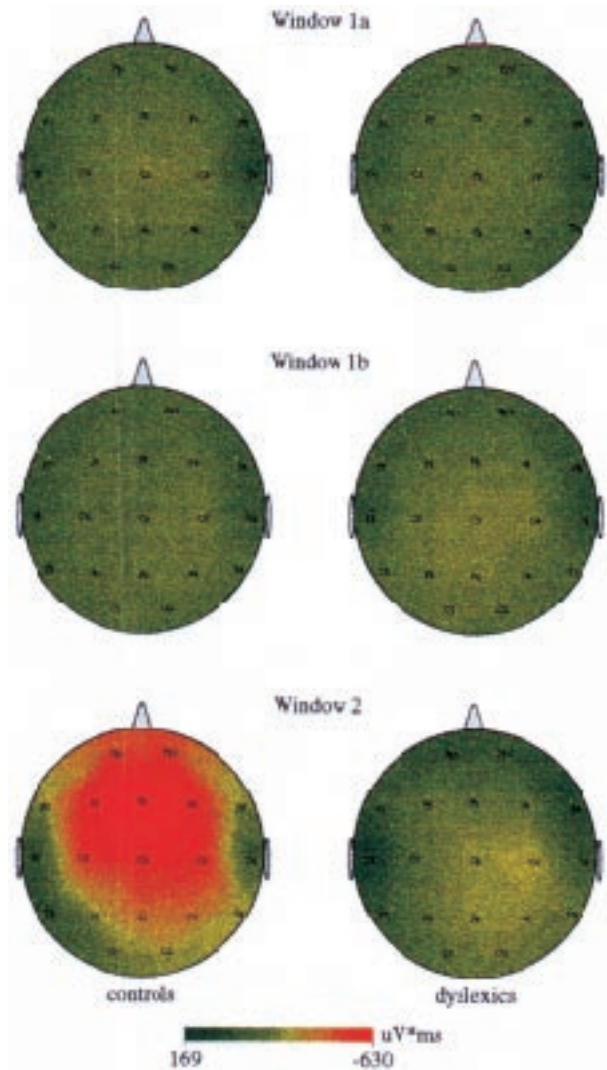
Table 1. Mean values and SD of the MMN area for the different time windows and for speech and tone stimuli

	Speech stimuli		Tone stimuli	
	Controls	Dyslexics	Controls	dyslexics
Window 1a	-125 ± 186	-108 ± 169	-303 ± 194	-311 ± 235
Window 1b	-114 ± 188	-128 ± 188		
Window 2	-555 ± 536	-112 ± 292	-417 ± 529	-467 ± 417

Results

For further statistical analyses, the areas of the electrodes of the fronto-central region (F3, Fz, F4, FP1, FP2, C3, Cz, C4) were averaged. Fz as the assumed centre was given double weight. The resulting mean value was used to examine group differences. Table 1 shows the means and s.d. of the MMN areas for the fronto-central region.

Two MANOVAs were performed, one for speech stimuli and one for tone stimuli, respectively. Effects of two factors were examined: group (dyslexics *vs* controls) and window (repeated measurement of the different windows: three for the speech stimuli and two for the tone stimuli). Analysis of the tone data revealed no significant effects, while analysis of the speech data yielded a significant window main effect and a significant group \times window interaction (Fig. 2). While the significant window main effect is of minor importance, the interaction of window and group should be examined in detail. The non-significant overall group effect shows (Fig. 2) that dyslexics do not have less MMN activity in general and irrespective of the window considered. The significant interaction, however, points to group differences that occur in only one or two of the windows. In order to clarify this, we examined the univariate *p*-values as given within the repeated measurements design. For window 2 the group effect was significant ($F = 9.45$; $p = 0.0043$).

**FIG. 2.** Mean values and *p*-values of the MANOVA for the speech MMN.**FIG. 3.** Brain maps of the speech MMN in the three time windows.

For further illustration of these findings, especially in terms of distribution differences, six brain maps were calculated (two groups \times three windows, Fig. 3). In each figure the maximum of MMN activity is at fronto-central leads. This means that the definition of our region of interest concurs with the actual distribution of MMN activity. We found no evidence for scalp distribution differences of the MMN activity between dyslexics and controls.

Discussion

We examined the hypothesis that dyslexics have an attenuated MMN for speech but not for tone stimuli. This hypothesis could be confirmed because we did not find group differences for the tone stimuli, but significant differences for speech stimuli did occur in window 2. This result of a specific speech processing deficit supports the result of Rumsey *et al.*, who

reported significant lower activation (PET study) of the left temporoparietal cortex in dyslexics during rhyme detection task but no group differences between dyslexics and controls during a simple tone detection task.¹³

Deficits in speech perception, especially the differentiation of phonemes such as /da-/ba/ have been shown to be confounded with reading and spelling deficits.¹⁴ However, this paradigm requires children to actively discriminate phonemes. This cognitive process could be influenced by the level of attention, by motivation and by memory span performance. Thus it remains unclear whether the found deficits in speech perception point to an underlying deficit of dyslexia, or are just a side-effect, or are like dyslexia caused by the same underlying, yet unknown, deficit.

The great advantage of the MMN studied by a passive oddball paradigm is that it examines a very early information process, and thus allows conclusions about the cause of dyslexia, rather than just finding coinciding deficits. Furthermore, the MMN is generally considered pre-attentive,⁸ and thus also rules out lack of attention and/or motivation as cause of poor performance. Our results therefore suggest that the deficits in pre-attentive speech processing can be considered a cause of dyslexia. On the other hand, our results are in line with a memory trace deficit in dyslexia which again was shown on a cognitive processing level by several researchers.^{15,16} The MMN is generated by a process which registers the difference between the present stimulus (deviant) and that represented by the trace (standard). Interestingly, Näätänen *et al.* recently found that the phonemic memory trace revealed by MMN is language specific, i.e. that the memory trace of speech sounds must be developed early in life.¹⁷ As shown by Kuhl *et al.*,¹⁸ speech recognition pattern develops within the first year of life. First this could mean that the memory trace of phonemes arises in the first year of life.

Second, regarding our results this could mean that the phoneme perception deficit is developed very early in life and has severe influence on children's reading and spelling at grade 5 and 6. Therefore, further research using the MMN paradigm should clarify whether this phoneme perception deficit developed in early years of life is a predictor of later reading and spelling disability.

Conclusion

The attenuated MMN for speech stimuli in dyslexics reveals deficits in pre-attentive and automatic information processing which can be considered a cause of dyslexia. This provides a tool for identifying children at risk even in pre-school age.

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