Auditory Cortical Responses to Speech-Like Stimuli in Dyslexic Adults

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Abstract

■ Auditory cortical processing of speech-like sounds was studied in 9 dyslexic and 11 normal-reading adults. Noise/square-wave sequences, mimicking transitions from a fricative consonant to a vowel, were presented binaurally once every 1.1 sec and the cortical responses were recorded with a whole-scalp neuromagnetometer. The auditory cortices of both hemispheres were less reactive to acoustical changes in dyslexics than in controls, as was evident from the weaker responses to the noise/square-wave transitions. The results demonstrate that dyslexic adults are deficient in processing acoustic changes presented in rapid succession within tens to hundreds of milliseconds. The observed differences could be related to insufficient triggering of automatic auditory attention, resulting, for instance, from a general deficiency of the magnocellular system. ■

INTRODUCTION

Developmental dyslexia (DD), or specific reading impairment, is a disorder affecting the subject's ability to learn to read, in spite of good motivation, adequate teaching, and normal intelligence. Dyslexia has been associated with a phonological core deficit that impairs mapping of letters to the corresponding sounds of spoken language (Frith & Frith, 1998; Liberman & Shankweiler, 1985; Bradley & Bryant, 1983). Children with reading problems frequently fail to develop adequate phonological awareness, manifested as a disability to analyze and segment the phonemic components of words. Reading-disabled children have problems also in identifying and discriminating consonant–vowel syllables (Reed, 1989; Godfrey, Syrdal-Lasky, Millay, & Knox, 1981), which suggest disorders in the phonemic representations.

A rather different view to reading disorders has emerged from experiments reporting on several mild sensory deficits in many dyslexic subjects. For instance, an auditory deficit at time scales of up to a few hundreds of milliseconds has been documented with sounds presented in rapid succession, and with sounds containing fast frequency transitions (Farmer & Klein, 1995; Tallal & Piercy, 1973, 1975; Tallal, 1980). Consequently, the phonological processing deficit has been speculated to derive, at least in part, from a low-level auditory dysfunction, manifested as impaired temporal processing of sounds (Tallal, 1980).

The first studies reporting on this type of auditory deficits were run on language-learning impaired (LLI) children who fail to develop normal oral language and thus differ from DD subjects in whom the failure is limited to normal reading development. As many children have problems in both oral language and reading, LLI and DD have been suggested to be different manifestations of the same disorder (Tallal, Allard, Miller, & Curtiss, 1997). However, although both disorders are associated with problems in literacy, the underlying mechanisms may differ. For example, limitations in phonological awareness seem to be crucial in DD, whereas in LLI, other language problems, such as weak vocabulary, can influence literacy development (Snowling, Bishop, & Stothard, 2000). The LLI and DD children—at least the subgroups tested so far—seem to differ in the severity of the associated auditory processing deficit: Whereas all LLI children failed in discriminating the order of two different-pitch tones at interstimulus intervals (ISIs) shorter than 150 msec (Tallal, Miller, & Fitch, 1993), 55% of DD children performed within normal limits (Tallal, 1980).

Despite the abundant evidence for auditory deficits in at least a subgroup of dyslexic subjects, the relationship of these deficits to the phonological problems is by no means settled. For instance, the problems of dyslexics in discriminating tones and speech sounds have been claimed to reflect independent deficits (Studdert-Kennedy & Mody, 1995). Moreover, the problems in differentiating, for example, /ba/–/da/ syllables have been suggested to reflect perceptual confusion between phonetically similar syllables rather than a difficulty in perceiving rapid spectral changes (Mody, Studdert-Kennedy, & Brady, 1997).

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It is possible that the neuronal basis of processing of fast frequency transitions and of sounds presented in rapid succession differs. Our recent studies on illusory directional hearing and on auditory stream segregation in dyslexic adults (Helenius, Uutela, & Hari, 1999; Hari & Kiesilä, 1996) are in line with the proposal that dyslexics have a longer-than-usual time window within which successive stimuli may interfere (Cutting & Pisoni, 1978). In the auditory saltation illusion (Hari, 1995), four left-ear leading clicks were followed by four right-ear leading ones; interaural time differences of 0.8 msec were used to produce the lateralized percepts of the single binaural clicks. When presented at long ISIs, the binaural clicks were perceived as four left-sided clicks followed by four right-sided clicks. However, when the ISI was shortened below 150 msec, a saltatory percept emerged, with the sounds appearing to jump from left to right at equidistant steps.

Hari & Kiesilä (1996) demonstrated that dyslexic adults perceive the saltation at significantly longer ISIs than the normal readers. Further evidence for the prolonged processing window was obtained from an auditory stream segregation experiment (Helenius et al., 1999) in which high and low tones were presented alternately. When such a sequence is presented with a long ISI, a continuous sequence of high–low–high–low... tones is heard. When the ISI is shortened, the streams segregate and two separate streams, high–high–high... and low–low–low..., are perceived. Helenius et al. (1999) observed that the ISI leading to segregation was almost double in dyslexic adults compared with control subjects. The results from these two studies agree with the idea of sluggish processing of rapid stimulus sequences in dyslexics. They further indicate that the difficulties in perceiving sounds presented at rapid rates persist to adult age, as do phonetic deficits (Cornelissen, Hansen, Bradley, & Stein, 1996), although many dyslexics eventually overcome their reading difficulties.

The functions of the human auditory cortex can be noninvasively and selectively studied with magnetoencephalography (MEG). The prominent 100-msec auditory responses (N100m) are elicited by various changes in the acoustic environment and can thus be used as indicators of the reactivity of the auditory cortex (for a review, see Hari, 1990). The source location of N100m suggests main contribution from areas in the supratemporal auditory cortex immediately posterior to the primary auditory cortex in the Heschl's gyrus, thereby including the planum temporale (PT) (Godey, Schwartz, de Graaf, Chauvel, & Liégeois-Chauvel, 2001; Lütkenhöner & Steinsträter, 1998; Pantev et al., 1995; Pelizzone et al., 1987); intracranial recordings agree with this view (Liégeois-Chauvel, Musolino, Badier, Marquis, & Chauvel, 1994).

Speech sounds typically contain acoustic transitions at approximately 100-msec intervals. Kaukoranta, Hari, & Lounasmaa (1987) showed that the onset of the Finnish word /hei/ (pronounced [hay]) elicits a typical N100m response, followed by N100m′ triggered by the transition from the fricative consonant to the vowel. As a similar N100m–N100m′ sequence was elicited by a stimulus in which a noise burst was immediately followed by a square wave, these responses seemed to be related to the nonspeech acoustic parameters common to both stimuli (Mäkelä, Hari, & Leinonen, 1988). N100m′ typically peaked 100–120 msec after the transition, on average 12 msec later than the N100m response with respect to the sound onset, probably because of masking by the preceding noise.

Although the N100m′ response is elicited by purely acoustic features of the stimuli, it still could reflect mechanisms of transient detection essential for proper acoustic analysis of speech sounds (Kaukoranta et al., 1987). N100m′ might thereby provide a tool to study how the human auditory system processes speech-like stimuli in the absence of linguistic content, and thus help to further characterize possible low-level auditory deficits in dyslexic subjects.

In the present study, we examined whether the N100m′ responses to noise/square-wave transitions would be disturbed in adult subjects suffering from DD. Normal-reading adults served as the control group. A preliminary report of this study has been presented in abstract form by Koivikko(Renvall), Mäkelä, & Hari (1999).

RESULTS

Behavioral Tasks

The dyslexics were significantly slower than the normal-reading adults in reading speed (dyslexics 97 ± 6 words/min, controls 160 ± 6 words/min; $p < .0001$) and in word recognition (dyslexics 862 ± 65 msec, controls 511 ± 10 msec; $p < .001$); the values of all dyslexic subjects were at least two standard deviations below the mean values of the control subjects. The dyslexics were also significantly slower in naming colors, letters, and numbers (dyslexics 712 ± 60 msec/item, controls 496 ± 25 msec/item; $p < .01$). The groups differed significantly also in backward digit span (dyslexics 4.3 ± 0.5, controls 6.5 ± 0.5; $p < .007$).

MEG Responses

Figure 1 shows the responses of control subjects C1 and C6 and of dyslexic subjects D3 and D6 to all stimuli at one channel over the left hemisphere (LH). Onsets of the square waves presented alone (top traces) evoked a prominent N100m (marked with the open circle) at 103 ± 2 msec in both control and dyslexic subjects. The noise/square-wave transitions elicited an additional response, N100m′ (marked with the filled

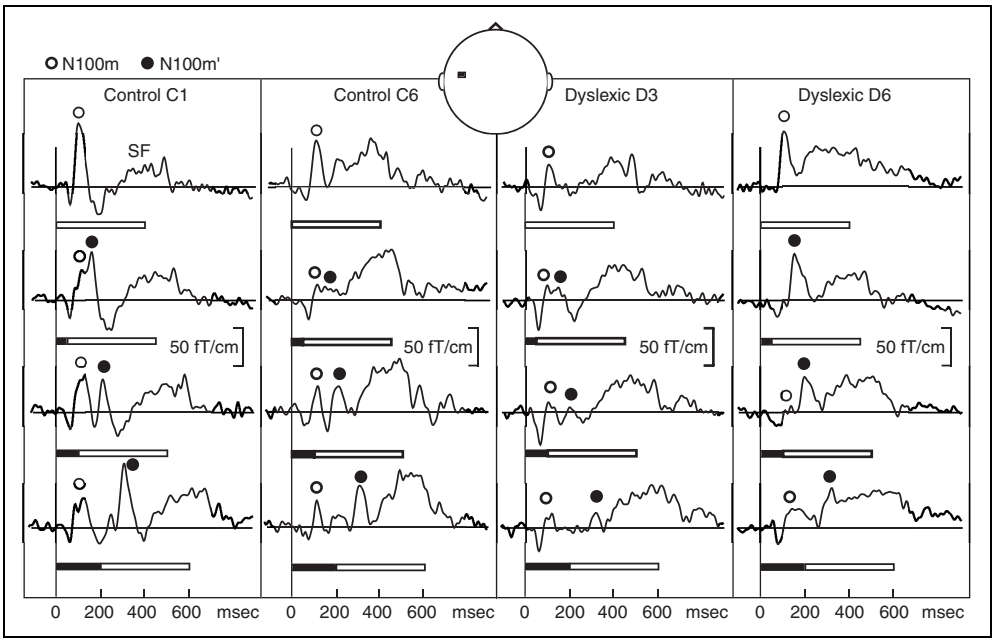

Figure 1. Evoked responses at one channel in the LH for two control and two dyslexic subjects. Open circles indicate the N100m responses, filled circles the N100m′ responses, and SF refers to sustained field. In the horizontal bars below the traces, black bars refer to noise and white bars refer to square-wave parts of the stimuli.

circle) 107 ± 2 msec after the transition of noise to square wave. Sustained fields (SFs) were elicited by all stimuli 300–500 msec after the square-wave onset.

In both control subjects, all stimulus onsets elicited clear N100m responses. In subject C1, the transition-triggered N100m′ response increased by 34% when the noise duration increased from 50 to 200 msec. In subject C6, N100m′ was small for stimuli with 50-msec noises, but increased to 2.9-fold at the longest noise duration. In contrast, the dyslexic subject D3 exhibited small N100m and N100m′ to all noise/square-wave combinations. He also showed prominent P60m responses that peaked about 60 msec after stimulus onsets. In the dyslexic subject D6, N100m did not exceed the prestimulus baseline for stimuli with 50 and 100 msec noise bursts, and N100m′ decreased from 60 to 53 fT/cm when the noise duration increased from 50 to 200 msec. SFs were prominent in all subjects and for all stimuli.

Sources of N100m and N100m′

In agreement with previous studies (for a review, see Hari, 1990), the N100m and N100m′ responses were adequately explained by two equivalent current dipoles (ECDs), one in the left and the other in the right supratemporal auditory cortex. Figure 2 shows the current dipoles and the corresponding N100m source waveforms of control subject C10 for the square waves presented alone, superimposed on his MR images. In

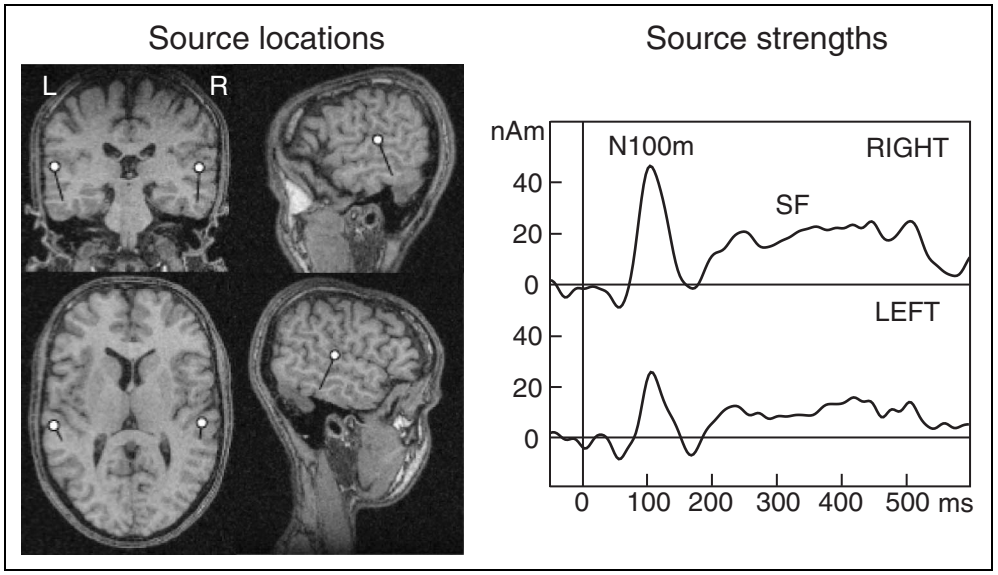

Figure 2. N100m source strengths as a function of time in control subject C10 for square waves presented alone, and the locations (dots) and the orientations (bar) of the current dipoles used to model the responses superimposed on subject's MR images.

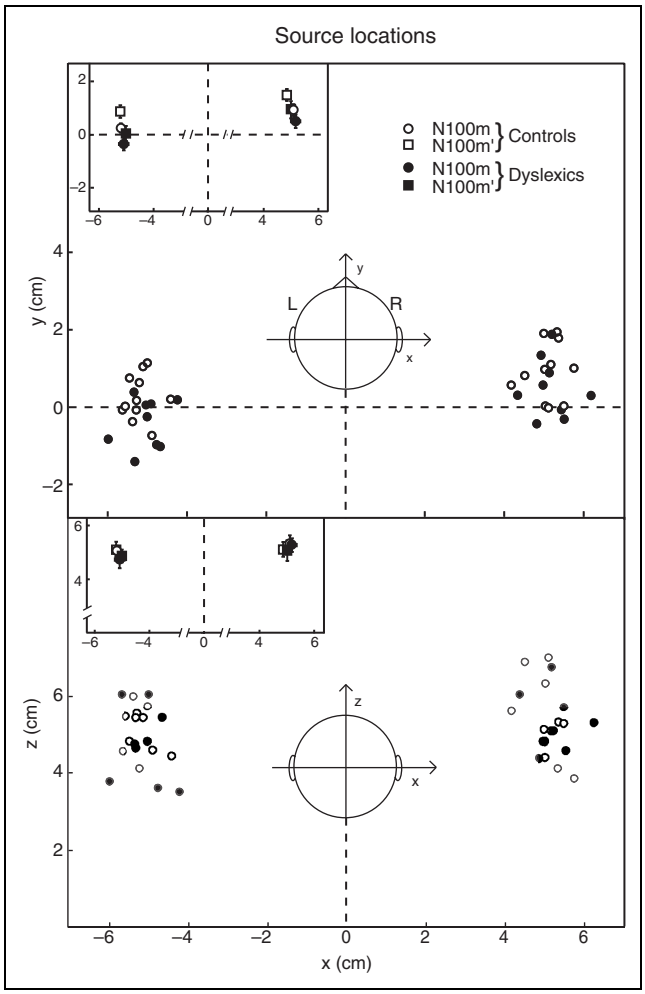

Figure 3. The locations of individual N100m ECDs in both subject groups (open symbols for controls, filled for dyslexics). The inserts show the mean ± *SEM* locations for N100m and N100m′. In the coordinate system the *x*-axis goes through periauricular points from left to right, the *y*-axis from the back of the head to the nasion, and the *z*-axis points to the vertex.

both hemispheres, the dipoles were located in the anterior part of the PT.

Figure 3 shows the individual three-dimensional locations of the ECDs for N100m to square waves presented alone; the inserts show the average locations of N100m and N100m′ sources in both subject groups. In agreement with earlier studies (Kaukoranta et al., 1987), the sources were on average 7–9 mm more anterior in the right hemisphere (RH) than the LH ($p < .005$); the groups did not differ in this respect. In the LH, the sources tended to be more posterior for dyslexics than controls (mean difference 6 mm for N100m, $p = .06$, and 8 mm for N100m′, $p < .05$), but otherwise, the groups did not differ. In control subjects, the sources were on average 6 mm more anterior for N100m′ than for N100m in both hemispheres ($p < .05$), in agreement with previous studies (Loveless, Levänen, Jousmäki, Sams, & Hari, 1996). In dyslexics, a similar tendency was seen in the RH (mean difference 4 mm, $p = .07$).

N100m Latencies and Amplitudes

In the control group, N100m peaked statistically significantly earlier in the RH than in the LH for stimuli with noise durations of 100 msec (mean difference 11 msec, $p < .05$) and 200 msec (mean difference 12 msec, $p < .01$), whereas in dyslexics, no such differences were observed between the hemispheres (see Table 1). As the only statistically significant difference between the groups, the N100m responses to square waves presented alone peaked earlier in the RH of dyslexic than control subjects (mean difference 10 msec, $p < .01$).

Figure 4 (top) shows the N100m source strengths at different noise durations in both subject groups, measured at the response peaks from the source waveforms. The largest N100m responses were elicited by square waves presented alone, with no amplitude differences between the groups or hemispheres. For the noise/square-wave stimuli, N100m increased with increasing noise duration; this effect was apparently due to the associated prolonged mean-ISI preceding the stimulus.

The results showed a statistically significant effect of Subject Group on N100m across both Hemispheres and all stimuli [$F(1,18) = 5.0$, $p < .04$]: The LH N100m was significantly smaller in dyslexics than in controls at 200 msec noise duration (t test; $p < .05$), and the RH N100m was smaller at 100 msec ($p < .04$) and 200 msec noise duration ($p < .03$). Noise Duration had a significant effect on response amplitudes in the LH of control subjects [controls $F(2,20) = 9.5$, $p < .002$; dyslexics $F(2,16) = 1.5$, $p = .25$] and in the RH of both groups [controls $F(2,20) = 6.3$, $p < .008$; dyslexics $F(2,16) = 8.4$, $p < .004$]: The responses were stronger for stimuli with longer noise bursts.

N100m′ Latencies and Amplitudes

N100m′ latencies did not differ between subject groups in either hemisphere (see Table 1). In both groups, N100m′ peaked significantly (9–23 msec) later than N100m to square wave presented alone (RH: $p < .001$–$.05$ for all noise stimuli; LH: $p < .05$ for 200 msec noise, and in dyslexics $p < .03$ also for 100 msec noise). N100m′ peaked significantly later for 200 than 100 msec noise in the LH of both groups ($p < .05$), and in the RH of control subjects ($p < .04$). The latencies did not significantly differ between hemispheres in either group.

Figure 4 (bottom) shows the N100m′ source strengths at different noise durations in both subject groups. For the 50-msec noise condition, the N100m′ responses of the control subjects were small. Noise Duration had a significant effect on N100m′ amplitudes in both hemispheres [LH: $F(2,16) = 25.7$, $p < .001$; RH: $F(2,16) = 14.3$, $p < .001$]: N100m′ amplitude increased with increasing noise duration and was significantly larger at 200 than at 50 msec noise in both hemispheres ($p < .001$).

Table 1. The Peak Latencies of P60m, N100m, and N100m′ Responses in Both Subject Groups

	Left Hemisphere		*Right Hemisphere*	
	Controls	*Dyslexics*	*Controls*	*Dyslexics*
P60m				
Square wave alone	55 ± 2	53 ± 5	50 ± 4	51 ± 3
Noise duration				
50 msec	53 ± 4	58 ± 4	54 ± 3	52 ± 4
100 msec	56 ± 3	60 ± 4	59 ± 4	56 ± 4
200 msec	58 ± 4	57 ± 5	58 ± 4	58 ± 4
N100m				
Square wave alone	111 ± 4	104 ± 3	107 ± 3	97 ± 2
Noise duration				
50 msec	119 ± 9	105 ± 8	109 ± 7	109 ± 14
100 msec	113 ± 5	108 ± 5	102 ± 5	109 ± 4
200 msec	116 ± 3	111 ± 6	104 ± 5	107 ± 5
N100m′				
Noise duration				
50 msec	109 ± 4	114 ± 7	119 ± 4	108 ± 4
100 msec	111 ± 3	116 ± 5	113 ± 3	118 ± 4
200 msec	115 ± 3	112 ± 4	125 ± 5	118 ± 4

In dyslexic subjects, N100m′ was small for the 50-msec noise. As is evident from Figure 4 (bottom), the RH responses of dyslexics tended to increase from 100 to 200 msec noise duration, but this effect did not reach statistical significance ($p = .07$). Noise Duration had no significant effect on N100m′ amplitude in either hemisphere [LH: $F(2,14) = .27$, $p = .77$; RH: $F(2,14) = 3.2$, $p = .07$]: N100m′ amplitude at 200 msec noise did not statistically significantly differ from the response at 50 msec noise in either hemisphere.

The effect of Subject Group on N100m′ amplitudes across both Hemispheres and Noise Durations was statistically significant [$F(1,15) = 4.8$, $p < .05$]: The responses were statistically significantly smaller in dyslexic than control subjects at 200 msec noise in the LH (t test, $p < .03$) and at 100 msec ($p < .04$) and 200 msec noise ($p < .02$) in the RH. Also the Subject Group × Noise Duration interaction was significant [$F(2,30) = 7.0$, $p < .004$].

Figure 5 shows the individual N100m′ amplitude changes as a function of noise duration. In the LH, the responses increased (>2 nanoamperemeters [nA·m]) in all control subjects but only in 3 of the 8 dyslexic subjects when the noise was prolonged from 50 to 200 msec. It is worth noting that 2 of these 3 dyslexics, and 3 of the controls, did not have a detectable N100m′ response to stimuli with 50 msec noise. The change in source amplitude as a function of noise duration was statistically significantly smaller ($p < .05$) in dyslexics than in control subjects. The main effect was seen from 100 to 200 msec noise ($p < .03$): N100m′ increased in all controls, but only in 3 dyslexics.

In the RH, the responses increased from 50 msec noise to 200 msec noise in 8 of 9 controls, and in all these subjects the increase exceeded 5 nA·m. Responses also increased in 4 of 9 dyslexic subjects but the increase exceeded 5 nA·m only in 2. The mean changes in source strengths were larger in the control group ($p < .005$). In the RH, the groups differed most clearly in change from 50 to 100 msec noise ($p < .03$): N100m′ increased in 6 controls, but only in 1 dyslexic subject.

P60m latencies did not differ between groups in either hemisphere (Table 1). Neither did the two subject groups differ in P60m amplitudes across both hemispheres and all stimuli [$F(1,18) = .80$; $p = .38$].

DISCUSSION

Our MEG recordings illustrated marked differences between dyslexic and normal-reading control subjects in the auditory cortical responses to noise/square-wave transitions that mimicked the fricative consonant–vowel combinations of speech sounds. In control subjects, the transition-triggered N100m′ increased as a function of increasing noise duration, similarly as happens when the duration of the fricative consonant is increased in fricative/vowel combinations (Kaukoranta et al., 1987). In dyslexic subjects, however, no enhancement was observed in the LH; in the RH, a subtle enhancement occurred from 100 to 200 msec noise stimuli but the increase from 50 to 200 msec noise was significantly weaker than in the control subjects.

The Magnocellular Deficit Hypothesis of Dyslexia

In addition to deficits demonstrated in auditory processing, dyslexic subjects display various visual abnormalities (Stein & Walsh, 1997), and deficits in the tactile sense have been reported as well (Laasonen, Tomma-Halme, Lahti-Nuuttila, Service, & Virsu, 2000; Grant, Zangaladze, Thiagarajah, & Sathian, 1999). A general deficit in the magnocellular neural pathways, the so-called M-deficit, has been suggested as one possible unifying explanation underlying the widely varying small sensory and behavioral abnormalities in dyslexics; for recent reviews, see Habib (2000) and Stein & Walsh (1997). The magnocellular pathways consist of large and fast-conducting fibers that can reliably carry transient signals that, as the fastest volleys, may serve as ''time markers'' for cortical processing.

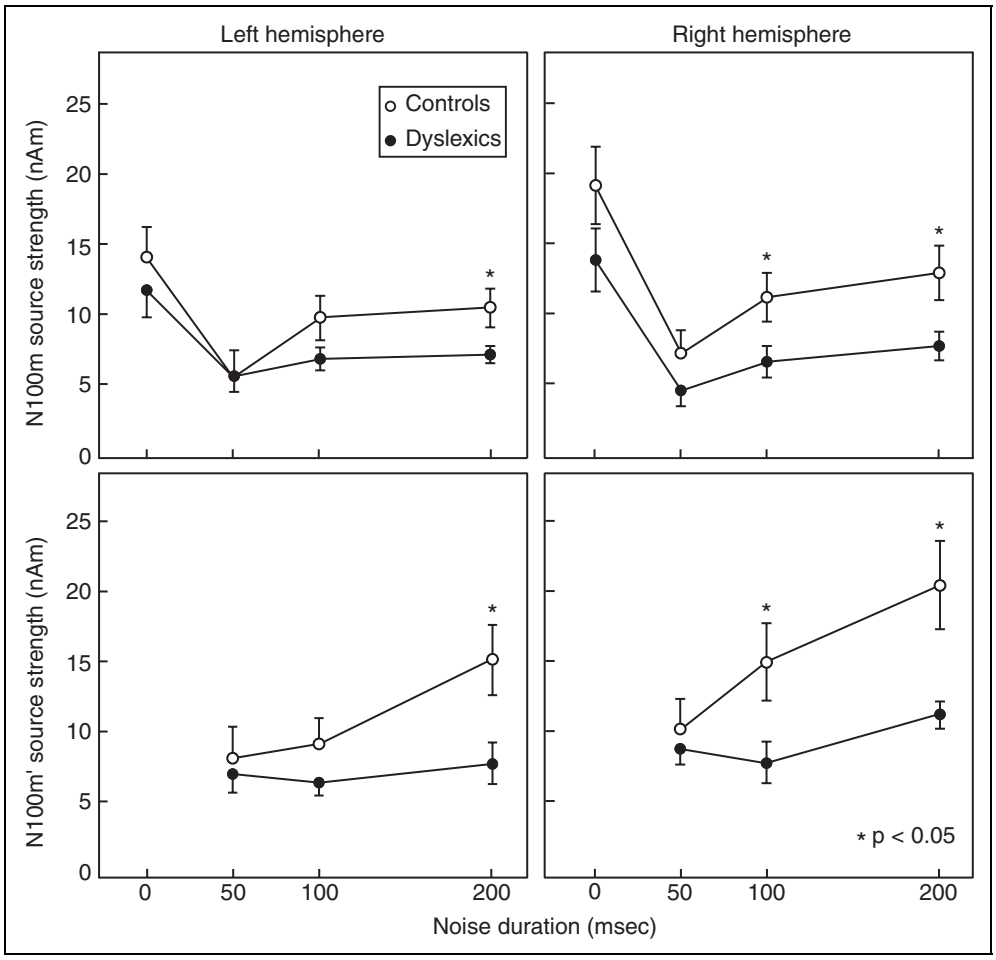

Figure 4. Top: The N100m source strengths to noise/square-wave onsets at different noise durations in both hemispheres (noise duration 0 msec refers to square waves presented alone). Bottom: The N100m′ source strengths to noise/square-wave transitions as a function of noise duration. One dyslexic and two control subjects were excluded from the N100m′ analysis because of inadequate dipole modeling. For the 50-msec noise condition, baseline noise level (see Methods) was used as the amplitude value in 3 control and 3 dyslexic subjects. N100m peaked 20 msec later to stimuli with 50 msec noise than with longer noise bursts in the LH of one control subject, bilaterally in another control subject, and in the RH of one dyslexic subject. The asterisks indicate statistically significant ($p < .05$) differences between the two groups of subjects.

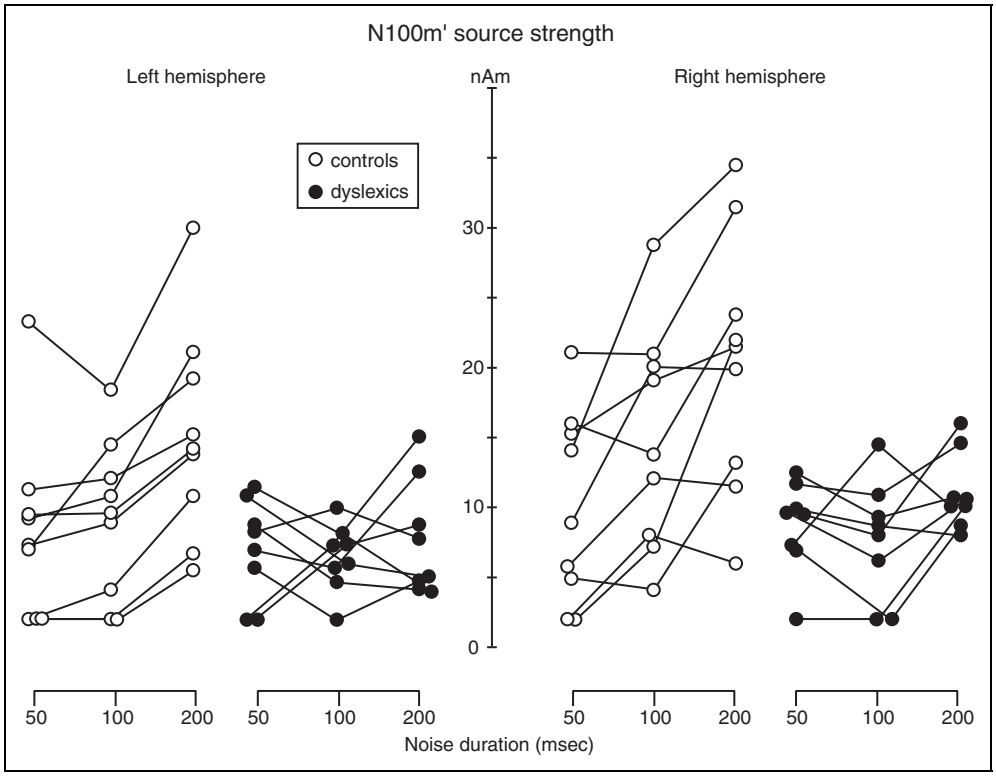

Figure 5. The absolute changes in N100m′ source strength in both hemispheres when the noise of the stimuli was prolonged from 50 to 100, and from 100 to 200 msec.

Evidence for visual M-deficit in dyslexic subjects has been reported in histological, electrophysiological, brain imaging, and psychophysical studies (Hari, Valta, & Uutela, 1999; Hari, Renvall, & Tanskanen, 2001; Demb, Boynton, & Heeger, 1998; Eden et al., 1996; Cornelissen, Richardson, Mason, Fowler, & Stein, 1995; Livingstone, Rosen, Drislane, & Galaburda, 1991; Lovegrove, Bowling, Badcock, & Blackwood, 1980). However, the findings differ considerably between studies, tasks, and subject groups, to the extent that the whole evidence for the magnocellular deficit has been disputed (see, e.g., Skottun, 2000a). The M-deficit hypothesis has evoked further distrust because some of the detected sensory deficits (e.g., the contrast sensitivity problems) seem unlikely to directly cause the reading disorder. Consequently, the connection between the possible M-deficit and the reading disorders is under continuous debate (Skottun, 2000b; Stein, Talcott, & Walsh, 2000).

Postmortem studies of brain morphology of 5 dyslexic subjects (Galaburda, Menard, & Rosen, 1994; Livingstone et al., 1991) showed smaller magnocellular layers in the lateral geniculate nuclei (LGN) and in the left-sided medial geniculate nuclei (MGN) of the thalamus than observed in normal-reading subjects. These anomalies in the visual and auditory pathways are in line with the observed behavioral deficits in dyslexics, and are often taken as a confirming evidence of the M-deficit. Unfortunately, these findings have not been replicated or expanded to a larger number of subjects. Recently, Jenner, Rosen, & Galaburda (1999) demonstrated that the thalamic changes in these five brains of dyslexic subjects were not associated with any changes in the layers with magnocellular input at the primary visual cortex.

Segregated magno- and parvocellular processing routes are well documented in the visual system up to the level of the primary visual cortex V1; at later processing stages, the inputs are intermingled to a large extent. Large neurons with thick axons are efficient in processing and transmitting transient signals. Although similar magno/parvo distinction is not typically made in the auditory system, "magno" cells exist also in the MGN of the auditory thalamus, and these cells were smaller and more disorganized in the postmortem brains of dyslexic than of normal-reading subjects (Galaburda et al., 1994). Moreover, the auditory system is divided already at the level of nucleus cochlearis into two pathways: one rather direct and fast relay system via the ventral cochlear nucleus, and a more complex processing stream via the dorsal cochlear nucleus. At the cortical level, auditory analogies to dorsal and ventral auditory processing streams have been suggested, although it is still unclear to which extent these would be organized to "what," "where," and "how" pathways (Maeder et al., 2001; Zatorre & Belin, 2001; Belin & Zatorre, 2000; Kaas & Hackett, 1999; Romanski et al., 1999), and how this division would be related to the possible auditory fast-conducting magnocellular pathways.

Auditory Responses and Stimulus-driven Automatic Attention

Although transient N100m-like responses can be elicited by various abrupt changes in the auditory environment, they also reflect stimulus-specific neural activity, and the stimulus specificity increases when the ISI gets shorter (Hari, 1990). Näätänen & Picton (1987) suggested that one functional role of the (electric) N100-type responses is related to nonspecific attention-triggering processes in the auditory cortices. Such relationship is in line with the increase of N100m amplitude when the ISI is prolonged (Hari, Kaila, Katila, Tuomisto, & Varpula, 1982; Hari et al., 1987), that is, when the behavioral saliency of the stimulus increases.

The N100m amplitude can increase as a result of either increased synchrony or number of the activated neurons (Hari, 1990), or as a sign of reduced active inhibition (Loveless, Hari, Hämäläinen, & Tiihonen, 1989). In the present study, the diminished N100m and N100m′ amplitudes in dyslexic subjects were not accompanied by delayed latencies, and therefore, increased active inhibition, rather than a decrease in neuronal synchrony, could underlie these effects. This view agrees with the study by Nagarajan et al. (1999) who found that N100m to the second sound of a pair is, at short stimulus onset asynchronies, smaller in dyslexic than normal-reading adults. Interestingly, Frenkel, Sherman, Bashan, Galaburda, & Loturco (2000) showed in mice with cortical ectopias a very similar result: Responses to the second tone were smaller at short ISIs. Thus, there might be a close connection between the functional and structural changes because cortical ectopias have been reported in the brains of dyslexic subjects (Galaburda, Sherman, Rosen, Aboitiz, & Geschwind, 1985).

In our dyslexic subjects, the smaller transition-related N100m′ responses at the 100–200-msec noise durations might thus reflect weakened attentional capture by the auditory changes, resulting from increased inhibition of the corresponding neuronal pool. At present, it remains unknown whether the dyslexics' N100m′ responses would reach the normal level at longer noise durations than those used in this experiment. In line with the hypothesis of weakened attention triggering in dyslexic subjects, infrequent deviant sounds in an otherwise monotonous stimulus sequence elicit smaller mismatch responses in LLI/dyslexic than normal-reading subjects (Kujala et al., 2001; Koivikko(Renvall) & Hari, 2000; Baldeweg, Richardson, Watkins, Foale, & Gruzelier, 1999; Kraus et al., 1996).

Reading-disabled children perform below controls in a dichotic listening task that requires selective attention to either ear (Asbjornsen & Bryden, 1998), but generally,

little is known about the auditory attention in dyslexia. In the visual modality, magnocellular pathway is considered to be crucial for attention capturing and focusing (Vidyasagar & Pammer, 1999; Steinman, Steinman, & Lehmkuhle, 1997). Interestingly, the attentional dwell times were in a dual-target "attentional blink task" about 30% longer in dyslexic than control adults (Hari et al., 1999), and dyslexics also displayed signs of weakened attentional capture in both visual hemispaces (Hari et al., 2001). At a more general level, we recently suggested that regulation of stimulus-driven automatic attention could serve as the causal link between the magnocellular deficit and the observed temporal processing problems at the time scales of up to a few hundreds of milliseconds (Hari & Renvall, 2001; Hari et al., 2001).

Hemispheric Differences in Rapid Auditory Processing

Several studies have shown lack or even reversed asymmetry in the visual and auditory areas of dyslexic brains (Jenner et al., 1999; Hynd, Semrud-Clikeman, Lorys, Novey, & Eliopulos, 1990; Galaburda et al., 1985). For example, anomalous symmetry of the PT has been frequently reported (for a recent review, see Eckert & Leonard, 2000) instead of the typically larger left PT in 65% of normal population (Geschwind & Levitsky, 1968). Anatomical abnormalities of the PT could have contributed to the results of our dyslexic subjects, as well, although we were not able to confirm any gross anatomical differences since MR images were available only for one dyslexic subject. However, some lack of a normal hemispheric asymmetry is suggested by the finding that N100m peaked 10–11 msec earlier in the RH than in the LH in control subjects, whereas no such asymmetry was observed in dyslexics.

Processing of auditory transients is crucial for determining the temporal order of speech elements (phonemes) and for the segmentation of speech sounds. Thus, some speech-dominance-related hemispheric specialization for processing of rapid auditory transients has been intensively searched for. Indeed, recent studies have implied that the LH-dominant activation in linguistic tasks may extend to the processing of some rapid transient nonspeech sounds. For example, LH advantage has been found in PET studies for processing of nonverbal sounds containing rapid frequency transitions (Belin et al., 1998) and of temporal variations of pure tones (Zatorre & Belin, 2001). On the other hand, fast frequency modulations in the middle of sound produced larger N100m responses in the RH than in the LH (Pardo, Mäkelä, & Sams, 1999), and in a recent fMRI study (Poldrack et al., 2001), the activations of auditory cortices decreased symmetrically when speech was temporally compressed. These findings illustrate the complexity of hemispheric balance in acoustic processing. In accordance with many other imaging studies (Klingberg et al., 2000; Brown et al., 2001; Eden et al., 1996), the present study revealed differences between dyslexic subjects and normal readers in both hemispheres.

Auditory Masking and Cortical Responses

The stimuli used in the present study resemble those used in forward-masking experiments: Noise can be considered the masker of the following square-wave onset. Both the decrease of the N100m′ amplitude compared with the onset response without the preceding noise and the longer latency of N100m′ than of N100m suggest that some masking occurs (Mäkelä et al., 1988). If N100m was attention-related and dyslexics had problems in attention triggering, masking with, for example, intermittent noise or speech (Hari & Mäkelä, 1988) should have more distracting effects on N100m in dyslexics than in control subjects. So far, auditory masking between LLI/dyslexic children and normal readers has been studied only by behavioral measures (McArthur & Hogben, 2001; Wright et al., 1997).

Conclusion

The auditory cortices of dyslexic adults reacted less vigorously than those of control subjects to acoustical changes mimicking transitions from a fricative consonant to a vowel. We propose that the observed impairment is related to weakened triggering of stimulus-driven automatic auditory attention, resulting from, for example, deficient function of the magnocellular system. This deficit could contribute to problems in the phonological processing and thus play an important role in the genesis of reading disability.

METHODS

Subjects and Behavioral Tasks

We studied 9 dyslexic adults (mean ± *SEM* age 31 ± 2 years; 5 women, 4 men; all right-handed) and 11 healthy control subjects (29 ± 2 years; 5 women, 6 men; 10 right-handed, 1 ambidextrous with −9 laterality index in the Edinburgh handedness test in which the left- vs. right-handedness ranges from −100 to +100). The dyslexics were selected on the basis of a stated diagnosis of DD, and 6 of them had participated in special tutoring at school age. One dyslexic subject had suffered from a left-sided noise trauma that had affected hearing at high frequencies (maximum at 4 kHz), but he reported equal loudness of the stimuli in both ears. None of the subjects were known to suffer from any neurological or psychiatric disorder, and they were not on continuous medication. The lowest level of education in dyslexics was 11 years;

one subject had a university degree, one was studying toward it, two were studying for an academic-level professional degree, and the others had successfully finished vocational education.

The subjects performed five reading-related behavioral tasks. In the oral reading task, the subject had to quickly read aloud a Finnish story, and the reading speed was measured during 1 min in the middle of reading. In a computerized word recognition task, the subject had to decide, as fast as possible, whether a word presented on a computer screen was a real Finnish word or an orthographically legal pseudoword. Correctly recognized words were used for calculating the word recognition speed. Naming speed was measured with a 5 × 10 matrix consisting of numbers, letters, and colors. Working memory was tested with digit spans forwards and backwards by using the standard WAIS procedure (Wechsler, 1955).

Auditory Stimuli

Four different sounds were led to the subject binaurally through plastic tubes and earpieces; the stimuli were presented in random order within the same sequence with a sound onset asynchrony of 1.1 sec; the same stimulus was allowed to occur only once in succession. The ISI (from offset to onset) varied from 0.5 to 0.7 sec. The stimuli consisted of a burst of white noise (0, 50, 100, or 200 msec in duration), followed immediatelly by a 400-msec square wave of 250 Hz. Sound intensity was adjusted to be at a comfortable listening level (65–75 dB SPL), and the rms values of the noise and square-wave bursts were equal. The preferred intensity levels did not differ between the groups ($p = .44$).

The subject was instructed to ignore the sounds, and all subjects except 1 dyslexic individual read a self-chosen text during the measurement to maintain the level of vigilance as stable as possible. This procedure was considered adequate for the present purposes because attention affects the auditory N100m responses only during very demanding tasks, such as tracking duration changes in one of two frequency channels in one ear (Rif, Hari, Hämäläinen, & Sams, 1991). One dyslexic subject who did not read was carefully instructed not to attend to the sounds.

MEG Recording

Whole-scalp neuromagnetic signals were measured in a magnetically shielded room, while the subject was sitting with the head supported against the helmet-shaped bottom of the Neuromag-122 magnetometer (Ahonen et al., 1993). The device comprises 122 planar first-order Superconducting QUantum Interference Device (SQUID) gradiometers covering the whole scalp. Each sensor unit measures two orthogonal tangential derivatives of the magnetic field component B_z normal to the helmet surface. Such gradiometers detect the largest signal just above a local activated brain area.

Four head-position-indicator coils were attached to the scalp, and their positions were measured with a three-dimensional digitizer; the head coordinate frame was specified by the two periauricular points and the nasion. The head position with respect to the sensor array was determined by feeding current to the marker coils. The recording passband was 0.03–100 Hz and the data were digitized at 300 Hz. The responses were digitally low-pass filtered at 40 Hz. Vertical electrooculogram was recorded to discard data contaminated by eye blinks and movements. A minimum of 100 artifact-free epochs was averaged for each stimulus category.

Analysis of the MEG Signals

To locate the cerebral sources of the responses, ECDs were searched by a least-squares fit to the data (Hämäläinen, Hari, Ilmoniemi, Knuutila, & Lounasmaa, 1993). The initial ECDs, one for each hemisphere, were calculated from signals measured by a subset of 10–16 sensors over the temporal lobes. An ECD represents the location, orientation, and strength of current flow in the activated brain area. Only ECDs explaining more than 80% of the local field variance were accepted for further analysis. The analysis was then extended to the entire time period, and all channels were taken into account: The previously found ECDs were kept fixed in orientation and location while their strengths were allowed to change. Only dipoles with strengths $\geq$4 nA·m were accepted; the prestimulus baseline was typically 1–2 nA·m.

For a more reliable quantification of the transient N100m and N100m′ responses, the signals were high-pass filtered at 3 Hz to discard the sustained fields occurring during long sensory stimuli (cf. Hari, Aittoniemi, Järvinen, Katila, & Varpula, 1980; Hari, 1990). The peak amplitudes of source waveforms and signals were then measured with respect to a 200-msec prestimulus baseline.

The N100m sources were searched for responses elicited by the square waves presented alone, and the same ECDs were used to explain the responses in all conditions. In 3 dyslexic and 3 control subjects, a better explanation was obtained by sources identified for responses to stimuli with 100 or 200 msec noise bursts, and those ECDs were used in the further analysis. The three-dimensional coordinates of ECDs did not differ significantly between these and other subjects.

The N100m′ sources were searched for the condition with the largest N100m′ response, and the same ECDs were used to explain responses in other conditions. In control subjects, all except the RH dipole of 1 subject were searched in the 200-msec noise condition, whereas in dyslexics, the sources were obtained in 1 subject

in the 100-msec condition, and in 3 subjects (2 subjects in both hemispheres, 1 subject in the LH) in the 50-msec condition.

The N100m and N100m′ latencies were measured from the source waveforms. If no response could be identified at a typical latency or if the response did not exceed 4 nA·m, the baseline level was used as the amplitude value. If only one response was detected for the 50-msec noise stimuli, the response was considered as N100m′ if it peaked >30 msec later than N100m to other noise/square-wave stimuli and >50 msec later than N100m to square-wave presented alone; otherwise, these responses were considered N100m.

The P60m latencies and amplitudes were measured from the vector sum $\sqrt{\left(\frac{\partial B_z}{\partial x}\right)^2+\left(\frac{\partial B_z}{\partial y}\right)^2}$ of the channel pair showing the maximum signal. The vector sums of the two orthogonal gradients were calculated for each channel pair. In signal strength comparisons, the vector sums simplify the analysis when the orientation of the neural current changes drastically as a function of time, with minor accompanying changes in the source location. In such a case, the amplitude measurements from a single channel could be misleading. If the response could not be identified, the average noise level of the channels (5 fT/cm) was used as the amplitude value.

Statistical Analysis

Two-tailed *t* tests were used for statistical comparisons of the behavioral data, response latencies, N100m and N100m′ source strengths, and N100m′ source strength changes. Mixed-model ANOVA (Subject Group as a between-subjects factor, and Noise Duration and Hemisphere as within-subjects factors) was used for evaluating the group effects across all stimulus conditions in N100m and N100m′ source strengths and P60m amplitudes. Within subject groups, the N100m′ and N100m source strengths across all noise/square-wave stimuli were analyzed with two-factor ANOVA separately for both hemispheres.

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