Auditory Processing in Dyslexia:
Evidence from Psychophysical Studies and
Event-Related Magnetic Fields
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I. General Introduction

Developmental dyslexia is a language-based learning disorder affecting the written language skills of roughly 7% of the population. Although several behavioral and some neurobiological correlates of dyslexia have been identified and investigated, large ‘scalp’ studies using magnetoencephalography (MEG) in children are rare. There is ample evidence that many children with dyslexia are impaired in their perception of speech sounds, in particular stop consonants, such as /b/, /d/, and /g/. The present thesis investigates auditory processing in individuals with dyslexia. Using behavioral and magneto-cortical measures, the focus is on perception of stop-consonant syllables in children and adolescents.

This chapter begins with a description of the phenomenology of developmental dyslexia. Then, current etiological theories of dyslexia are addressed. Neurobiological correlates of the disorder follow. In reviewing findings in the field of electrophysiology the aims and assumptions inherent in the present work are outlined.

PHENOMENOLOGY OF DEVELOPMENTAL DYSLEXIA

Developmental dyslexia has most often been defined on the basis of a specific reading disorder (American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, DSM-IV; 315.00) or as a combined specific reading and spelling disorder (World Health Organization: International Classification of Diseases, ICD-10; F81.0). According to such definitions, dyslexia is a disability in learning to read, spell, and write despite normal intellectual capacity and educational resources, as well as adequate sociocultural opportunity. At the same time sensory deficits, neurological pathology, and other impediments to attaining literacy skills are absent. Disturbances in reading and spelling significantly interfere with academic achievement or activities of daily living requiring reading or spelling skills. Estimates of prevalence rates vary between 3 and 10% (e.g., Rutter et al., 1976; Shaywitz et al., 1990; Haffner et al., 1998). While several epidemiological studies have found dyslexia to be two to four times more common in males than in females (Rutter & Yule, 1975; Finucci & Childs, 1981; Esser & Schmidt, 1994; Lewis et al., 1994), others have revealed a more balanced gender ratio (Shaywitz et al., 1990; Wadsworth et al., 1992; Flynn & Rahbar, 1994). Research in behavioral genetics indicates that dyslexia is familial, heritable, and genetically heterogeneous (for reviews see Pennington, 1995, 1999; Schulte-Körne et al., 1998b).

1 See Miles et al. (1998) for a comparative study and critical discussion.
As provided by the ICD-10 diagnostic guidelines, difficulty in oral reading and spelling is characterized by omissions (e.g., ‘ply’ instead of ‘play’ for the English language and ‘lesen’ instead of ‘lesen’ for the German), substitutions (e.g., ‘house’ for ‘home’, German: ‘Haus’ for ‘Heim’), inversions (e.g., ‘aks’ instead of ‘ask’ for the English language and ‘baul’ instead of ‘blau’ for the German), or additions of words or fragments of words. Decoding is frequently slow. Passage reading is associated with problems dyslexic children had keeping their place in lines of text, as are errors in comprehension. At the beginning of first grade, there may be weakness in learning the alphabet, naming letters, producing rhymes, and categorization of speech sounds (i.e., detecting which sounds in the language imply a difference in meaning, called phonemic perception\(^2\)). In secondary school and adulthood, deficits in spelling are often more salient than reading problems. It deserves mention, however, that frequency and types of errors in literacy components vary as a function of skills learned and the difficulty of the task (Grissemann, 1972).

Although symptoms of the disorder (e.g., poor sense of rhyme) may be present as early as kindergarten age, dyslexic individuals are not identified until they have tried and failed to learn to read. Especially in children demonstrating above-average intellectual ability, the disorder may not be manifest before the fourth grade, or even later (DSM-IV).

In addition to difficulties in the literacy domain, dyslexia may be associated with psychosocial problems, abnormalities in cognitive processing, and clinically relevant conditions. As described in DSM-IV and ICD-10, children with dyslexia may suffer from demoralization, low self-esteem, and deficient social skills. Problems in school adjustment or even an increased risk for dropping out of formal schooling have been reported. [In Germany, a number of dyslexic schoolchildren failed to reach class level (Warnke, 1999), many attend schools for special education (Esser & Schmidt, 1993), and less than 2% of the dyslexic students complete high school (Haffner et al., 1998)]. Adult dyslexics may have significant problems in occupational functioning or social mobility. [Data from a German follow-up study revealed that dyslexic participants were likely to take up occupations demanding practical rather than literacy skills. Moreover, dyslexic adults finishing 9 years of school, which is the minimum requirement of formal education in Germany, tended to be less satisfied with their jobs than those with higher educational levels (Strehlow et al., 1992).]

Deficits in cognitive processing that often precede or are associated with dyslexia include \textit{inter alia}: poor visual discrimination, weakness in auditory segmenting, limitations in

\(^2\) Phonemes are the smallest elements of speech which, if changed, would change the meaning of a word (e.g., Liberman et al., 1967).
working memory, linguistic disturbances (e.g., misarticulation of sounds, impairment in receptive and/or expressive language abilities), or a combination of these. Dyslexia may be associated with a higher rate of attention-deficit/hyperactivity disorder, behavior disorders (e.g., conduct disorder), emotional disorders (e.g., dysthymic disorder), or developmental coordination disorder.

In their New Zealand longitudinal study, Ferguson and Lynskey (1997) showed that associations between early reading difficulties and later conduct problems arise because those children displaying reading delays are characterized by a number of disadvantageous conditions which were already present at preschool age. A person’s general intellectual ability, amount of support received, and socioeconomic status have been identified as factors influencing the course of dyslexia (Naylor et al., 1990). Nevertheless, research has shown that many problems of the dyslexic population persist into adulthood (e.g., Maughan, 1995; Boetsch et al., 1996).

As stated above, the definition of dyslexia implies an impairment in specific achievement. In the two classification manuals, DSM-IV and ICD-10, the diagnosis of dyslexia requires reading and writing achievement significantly below the performance expected given the person’s chronological age, intelligence, and educational level. The ICD-10 research criteria recommend a discrepancy of at least 2 standard deviations (SD) between achievement and both age norm and general intelligence quotient (IQ). However, it is commonly intelligence that has received the most attention in diagnostics and research. Since a 2 SD achievement-IQ discrepancy would exclude a large percentage of dyslexic individuals, in many studies, the criterion has been lowered to 1-1.5 SD (see Schulte-Körne et al., 2001c for a recent discussion).

The definition and use of the term dyslexia has recently been controversial as Anglo-American and European researchers have argued that the standard discrepancy model is empirically unfounded and theoretically inadequate (e.g., Stanovich, 1996; Fletcher et al., 1998; Gustafson & Samuelsson, 1999). The functional significance of this definition is that children with relatively low IQ scores (i.e., between 71-85) who cannot learn reading and writing are not considered dyslexic. According to the Anglo-American literature, they are labeled ‘backward readers’ (e.g., Rutter & Yule, 1975), ‘garden-variety poor readers’ (e.g., Stanovich, 1991), or ‘low achievers’ (Fletcher et al., 1994). In most countries these children are not eligible for special educational services since their failure to initiate reading and spelling is accounted for by their general pattern of performance. The German educational system represents one exception offering special classes (within regular schooling) or special
schools for children who suffer from literacy problems but do not show the achievement-IQ discrepancy.

Some researchers hold the opinion that developmental dyslexia is not a single clinical entity. Therefore, several attempts have been made to classify dyslexic samples into relatively homogeneous subtypes. For instance, Boder (e.g., Boder, 1973; Flynn & Boder, 1991) diagnosed dyslexic children as ‘dysphonetic’, ‘dyseidetic’, or ‘mixed’ by observing qualitative differences in their reading and spelling performances. Children with *dysphonetic dyslexia* (63% of the sample) have difficulty learning sound-symbol relationships, thus committing phoneme/grapheme errors. As preliterates, they demonstrate deficits while analyzing spoken language, e.g., deleting syllables and phonemes of aurally presented words. Dysphonetics prefer to use global decoding strategies and read words as visual gestals. Their typical misreadings are word substitutions based on minimal cues (e.g., ‘dress’ for ‘diesel’), gestalt (e.g., ‘horse’ for ‘house’), or semantic association (e.g., ‘funny’ for ‘laugh’). Dysphonetic dyslexics generally read quickly but inaccurately. When spelling unfamiliar words, typical errors occurred are letter/syllable omissions or letter-order confusions.

Children with *dyseidetic dyslexia* (9% of the sample) generally use phonetic strategies in written language (i.e., grapheme-phoneme/phoneme-grapheme correspondence rules) but show difficulty in perceiving whole-word configurations. As a consequence, they read and spell phonetically regular words relatively accurately, albeit slowly, but show poor performance on phonetically irregular words. Phonetic renditions of irregular words (e.g., ‘toc’ for ‘talk’) and visuospatial letter reversals (e.g., ‘bib’ for ‘did’) are typical errors present both in their reading and spelling.

Children with *mixed dyslexia* (21% of the sample) display problems in both phonetic integration and gestalt function. Children of this subtype experience the most severe impairment in academic skills.

Another approach to subtyping adheres to the dual-route model of reading (e.g., Coltheart, 1978; Morton & Patterson, 1980) which is based upon case studies reporting several profiles of acquired dyslexia. This model posits that reading aloud entails two separate procedures, the so-called ‘lexical’ and ‘sublexical’ procedures. The lexical procedure (direct route) involves using the orthographic form of a word to retrieve an associated phonological representation stored in a mental lexicon. Since the mental lexicon only contains words which the reader has previously encountered, this procedure cannot be utilized in

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3 Acquired forms of dyslexia occur in premorbidly competent readers as a consequence of brain disease or injury.
decoding pseudowords.\textsuperscript{4} The sublexical procedure (indirect route) involves the use of grapheme-phoneme conversion rules. It produces correct responses for items whose phonological codes are specified by the rules (phonetically regular words and pseudowords) and it would lead to incorrect responses for irregular words which have to be pronounced \textit{via} the lexical route.

The dual-route model accounts for two profiles of acquired dyslexia. One profile is referred to as ‘surface dyslexia’ and the other as ‘phonological dyslexia’. Whereas surface dyslexics show a specific difficulty in reading \textit{via} the lexical procedure (e.g., Marshall & Newcombe, 1973; Coltheart et al., 1983), phonological dyslexics display the deficit in the sublexical procedure (e.g., Shallice & Warrington, 1980; Patterson, 1982). Castles and Coltheart (1993) identified developmental forms of surface and phonological dyslexia in children by using pseudowords and irregular words. Fifty-five percent of the children performed worse when reading pseudowords compared to irregular words (phonological dyslexic profile). Thirty percent of the children showed the reverse pattern of performance (surface dyslexic profile). Nine percent of the cases scored low on both irregular and pseudowords characterizing a mixed deficit profile, and 6% showed no discrepancy between the tasks. This finding was largely supported and extended by the study of Manis et al. (1996)\textsuperscript{5}: Whereas the surface dyslexics’ performance resembled that of younger normal readers, 71% of the phonological dyslexics demonstrated poorer pseudoword decoding than the reading-level matched control children. Manis and colleagues concluded that the phenomenology of surface dyslexia may be associated with a developmental delay, whereas phonological dyslexia rather reflects a developmental disorder.

To illustrate some characteristics of developmental dyslexia let us now turn to a related condition termed \textbf{specific language impairment} (SLI). SLI is diagnosed when children fail to develop language at the expected rate for no apparent reason. Typical symptoms are limited vocabulary, syntactic and morphological deficits, and impairment in language comprehension (see Leonard, 1998). After reaching school age, children with SLI are at risk for learning problems similar to those seen in dyslexics; conversely, children with developmental dyslexia typically have been found to be deficient in some linguistic tasks (e.g., Aram et al., 1984; Kamhi & Catts, 1986; Tallal et al., 1988; Bishop & Adams, 1990; Scarborough, 1990; Catts, 1993). On the basis of such performance profiles it has been

\footnotesize{\textsuperscript{4} Pseudowords are arbitrary phonetically regular letter combinations. Examples for the English and German language are ‘fleast’ and ‘fliest’, respectively (Landerl, 1996).}

\footnotesize{\textsuperscript{5} It should be mentioned that Manis et al. (1996) preferred to interpret their findings with the Seidenberg and McClelland connectionist model rather than the dual-route model. For further particulars on this topic the reader is referred to the work of Seidenberg and McClelland (1989).}
proposed that dyslexia, at least for some dyslexics, may be a weak form of SLI (Kamhi & Catts, 1986). Although there is supporting evidence from neurophysiological studies (Neville et al., 1993; McAnally & Stein, 1997), this assumption is still a matter of intense debate (see e.g., Leonard, 1998; Snowling et al., 2000). Nevertheless, the striking convergence between the characteristics of developmental dyslexia and SLI has given rise to the classification **language-based learning impairment** (see e.g., Tallal et al., 1998).

**Etiology of Dyslexia: Theories**

Given the complexity of cognitive processes involved in reading and writing, it is not surprising that a wide range of possible etiological models has been offered for dyslexia. A plethora of research exists supporting three theories that associate dyslexia with (1) a specific linguistic problem due to a deficit in **phonological processing**; (2) a general auditory processing impairment, specifically in **temporal processing**; and (3) impaired visual processing arising from abnormalities of the **magnocellular pathway** of the visual system.

**Phonological processing deficit**

The most widely accepted theory of dyslexia is that it is a linguistic problem and specifically due to a deficit in phonological processing (for reviews see e.g., Wagner & Torgesen, 1987; Goswami & Bryant, 1991; Rack, 1994; Snowling, 1995; Frith, 1998). Phonological processing refers to “the use of phonological information (i.e., the sounds of one’s language) in processing written and oral language” (Wagner & Torgesen, 1987, p.192) and encompasses at least three components: (a) phonological or phonemic awareness, (b) phonological recoding in lexical access, and (c) phonetic recoding in working memory (Wagner & Torgesen, 1987).

**(a) Phonological or phonemic awareness** is defined as “conscious access to the phonemic level of the speech stream and some ability to cognitively manipulate representations at this level” (Stanovich, 1986, p.362). Only when the conscious awareness of the sound structure has been developed can a child grasp the principle of an alphabetic language, in which linguistic segments are represented visually by graphemes (Gleitman & Rozin, 1977). Phonemic awareness tasks involve alliteration, counting, deleting, and substituting phonemes within words or syllables, reversing the order of phonemes within words or syllables, blending phonemes presented in isolation to form a word, and segmenting words into phonemes (e.g., Wagner & Torgesen, 1987; Rack, 1994). Other tasks are
concerned with awareness of broader sound structures such as words and syllables; these include rhyming, counting or deleting syllables in words, and segmenting words into subsyllabic units (e.g., Wagner & Torgesen, 1987; Rack, 1994). The former skills have been referred to as phonemic awareness in the narrow sense, the latter as phonemic awareness in the broad sense (Skowronek & Marx, 1989).

In their classical study, Bradley and Bryant (1978) presented 10-year-old dyslexic children with alliteration and rhyme tasks (phonemic awareness in the narrow and broad sense, respectively). The children were required to indicate which was the odd one out in a sequence of four spoken words (e.g., sun see sock rag or weed peel need deed or nod red fed bed). Dyslexic children performed more poorly than younger reading-level matched normal readers. This comparison suggests that dyslexic children’s poor awareness of syllabic-rhyme and alliteration may have had a role in their reading impairment. Comparable evidence was obtained by other researchers, who found that dyslexic children have difficulty in deciding whether two words rhyme or identifying the non-rhyming word of a short item sequence (e.g., Rack, 1985; Holligan & Johnston, 1988; Wimmer, 1993).

A large number of studies has shown that children with dyslexia are impaired on a variety of phonological awareness skills in the narrow sense (e.g., Manis et al., 1988; Bruck & Treiman, 1990; Olson et al., 1990; Bruck, 1992; Swan & Goswami, 1997b; Joanisse et al., 2000). The dyslexic children were not only outperformed by their age-matched controls but were worse as well when compared with younger reading-level matched normal readers. Thus, performance on these phonemic awareness tasks was not simply a function of reading level.

(b) **Phonological recoding in lexical access** refers to “recoding written symbols into some kind of sound-based representation, and then using this sound-based representation to access the lexicon” (Wagner & Torgesen, 1987, p.203). Tasks designed to estimate phonological recoding for lexical access typically include rapid naming of series of common objects, or other kinds of stimuli, naming pictures of familiar objects, and deciding whether a letter string constitutes a real word or a pseudoword (e.g., Wagner & Torgesen, 1987; Rack, 1994). Phonological recoding seems to be an important tool at early stages of reading acquisition (e.g., Ehri & Wilce, 1979) and even useful in the sophisticated reader for less familiar or unknown words (see the sublexical *versus* the lexical process of the dual-route model under ‘Phenomenology of developmental dyslexia’, this chapter).

Denckla and Rudel (1976) tested rapid automatized naming in a sample of 248 7- to 13-year-olds comprising dyslexic, non-dyslexic learning disabled, and normally literate
control subjects. The dyslexic children were found to be slower at naming series of objects, colors, digits, and letters compared to both age controls and generally learning-impaired children. Similar results obtained by comparing naming rate in dyslexics and age-matched controls were reported by other researchers (e.g., Wolf, 1986; Lovett, 1987; Bowers & Swanson, 1991; Wimmer, 1993; Semrud-Clikeman et al., 2000).

Katz (1986) observed dyslexic children to be inferior to normally literate age controls when naming familiar pictures, in particular those with polysyllabic and/or low frequency names (e.g., pyramid, rhinoceros, stethoscope). Further studies using reading-level match comparisons confirm an association between picture naming and reading ability (Wolf 1991; Swan & Goswami, 1997a,b).

Poor phonological recoding in children with dyslexia has also been reported by Nicolson and Fawcett (1994). In a lexical decision task, subjects were required to judge as quickly as possible whether a monosyllabic letter string (e.g., shop, thop) represents a word or not. Dyslexic children were not only impaired in comparison to their age-matched controls but performed even more slowly than their reading-level matched controls for access to words.

Within the reading domain, pseudoword decoding has proved to be a particularly valid measure of phonological processing (for a review see Rack et al., 1992). Decoding of a pseudoword requires the generation of a sound-based representation via the application of grapheme-phoneme correspondence rules (e.g., Snowling, 1995). This skill has variously been designated a measure of phonological recoding (e.g., McCusker et al., 1981; Wagner & Torgesen, 1987; Vandervelden & Siegel, 1996) or phonemic awareness (e.g., Frith, 1985; Witton et al., 1998). There is an abundance of evidence that accuracy and speed of reading pseudowords clearly differentiate skilled from less skilled readers and that dyslexic individuals are significantly impaired in performing the task (e.g., Snowling, 1981; Stanovich, 1986; Siegel, 1986, 1992; Landerl, 1996; Joanisse et al., 2000). However, as was described earlier in this chapter, there are also dyslexic children who appear to have mastered the alphabetic principle, and thus have no problems in decoding pseudowords. These children have sometimes been classified as surface dyslexics (see under ‘Phenomenology of developmental dyslexia’, this chapter). Interestingly, Sprenger-Charolles et al. (2000) reported that deficits in phonemic awareness and phonological short-term memory (see under

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6 Stimuli used in the Denckla and Rudel (1976) study were the use objects comb, key, watch, scissors umbrella, the colors red, green, black, blue, yellow, the digits 2, 6, 9, 4, 7, and the high-frequency lower-case letters p, o, d, a, s.
‘Phonetic recoding in working memory’, next paragraph) were even detectable in (French) children with surface dyslexia.

(c) **Phonetic recoding in working memory** denotes “recoding written symbols into a sound-based representational system that enables them to be maintained efficiently in working memory during ongoing processing” (Wagner & Torgesen, 1987, pp.192-193). Efficient phonetic recoding appears to be an important tool for the novice reader. Unlike the able reader, the beginner devotes the maximum amount of cognitive resources possible to the process of blending phonemes to generate words (Wagner & Torgesen, 1987). A working-memory model applied to the mechanism of learning to read is that developed by Baddeley and colleagues (e.g., Baddeley, 1990). This model is conceptualized as encompassing at least three interrelated components, a central executive and its two subsidiary systems, a visuo-spatial sketchpad for setting up and manipulating visual images and a phonological loop for encoding verbal materials. The phonological loop is of primary interest to us here. This loop is assumed to comprise a phonological store that represents verbal information in a phonological form and a subvocal articulatory rehearsal process that maintains the material in the phonological store. Auditory speech gains obligatory access to the phonological store, whereas written language has to be phonologically recoded by the articulatory rehearsal process in order to enter the store (Baddeley, 1990). Measures of phonetic recoding in working memory usually involve serial recall of diverse speech materials and verbal repetition of words, pseudowords, or sentences (e.g., Wagner & Torgesen, 1987; Rack, 1994).

Early studies have shown that elementary-school children with difficulty in word recognition tended to make more errors in serial-recall tasks including auditorily presented letter names (Shankweiler et al., 1979), consonant-vowel syllables (Brady et al., 1987), and words (Mann et al., 1980; Brady et al., 1983) as compared to their normally reading peers. Poor readers were also distinguished from good readers in their memory for printed letter strings (Shankweiler et al., 1979). In addition, poor readers were found to show a reduced or even absent phonological similarity effect (i.e., superior recall performance for phonemically dissimilar versus rhyming items) that is evident in normal individuals even at early reading stages (e.g., Shankweiler et al., 1979; Mann et al., 1980; Brady et al., 1983). On the other hand, no performance differences between good and poor readers were observed on tasks tapping visuo-spatial working memory (for a review see Vellutino, 1980). These findings suggest that poor readers do not rely on phonological codes in working memory. However, it was subsequently demonstrated that poor readers show a phonological similarity effect comparable to normal controls when they (i) were presented with list lengths adjusted to their
memory spans\(^7\) (Hall et al., 1983; Holligan & Johnston, 1988), (ii) were comparable or equated on memory span (Johnston et al., 1987; Irausquin & de Gelder, 1997), or (iii) were matched on reading level (Johnston et al., 1987; Holligan & Johnston, 1988). This indicates that poor readers use phonetic recoding in working memory, but less efficiently and hence have more limited capacity to remember linguistic material. The possible assumption that this inefficiency may have its roots in slower articulatory rehearsal speed could not be confirmed (e.g., Brady et al., 1989; Irausquin & de Gelder, 1997).

Further evidence that poor reading may be associated with impaired use in phonological recoding or phonetic representation comes from studies examining verbal repetition performance. For example, Snowling and colleagues (Snowling, 1981; Snowling et al., 1986) found dyslexic individuals to be poorer than normal reading level-matched controls at repeating pseudowords (e.g., *karpinular, bagmivishent*). Similarly, Mann and associates (Mann et al., 1980, 1984) observed that poor reading elementary-school children did less well than normally reading age controls on single sentence repetition.

Prospective longitudinal studies including children sampled at random suggest that phonological processing (i.e., phonemic awareness, phonological recoding in lexical access, and phonetic recoding in working memory) can play a causal role in reading deficits (for reviews see Torgesen & Wagner, 1987; Rack, 1994). For instance, Bradley and Bryant (1983, 1985) conducted a 4-year longitudinal study providing data from 368 children ranging from 4 to 5 years at initial screening. Phonological awareness was measured using the Bradley and Bryant (1978, see above) oddity-detection task (NB: the 4-year-old children listened to series of three words and the 5-year-olds to series of four).\(^8\) Results indicate a significant relationship between pre-reading children’s sensitivity to alliteration and rhyme and later performance on reading and spelling tests. Holding constant general factors such as age at initial testing, IQ, and memory for word lists, alliteration and rhyming performance accounted for 4-10% of the variance in reading and 6-10% of the variance in spelling. The influence of alliteration and rhyming performance seemed to be specific since it accounted for less of the variance (1-4%) in later math ability.

Further evidence for the importance of phonological skills in early reading acquisition is provided by experimental training studies. In a quantitative meta-analysis of controlled U.S.

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\(^7\) One often-quoted characteristic associated with dyslexia is impaired performance on memory span tasks. For instance, scores on Digit span subtest of the Wechsler Intelligence Scales for Children (WISC) tended to be lower in poor readers than in normal readers, even when matched on overall IQ (for a review see Rugel, 1974).

\(^8\) Wagner and Torgesen (1987) raised the objection that the task used by Bradley and Bryant (1983, 1985) might be a better test of (phonetic recoding in) working memory than of phonemic awareness.
studies, Bus and van IJzendoorn (1999) confirmed that training phonological awareness reliably improves children’s phonological and reading skills. The combined effect sizes for phonemic awareness and reading were .73 (N = 739) and .70 (N = 745), respectively. Thus, the meta-analytic results documented the causal role of phonemic awareness in the process of learning to read. However, it has also been shown that the relationship between phonological skills and reading ability is reciprocal (for reviews see Wagner & Torgesen, 1987; Rack, 1994). Using an extensive battery of tasks, Wagner et al. (1994) assessed phonological processing abilities (tapping phonemic awareness, phonological recoding in lexical access, and phonological memory) and reading-related knowledge of 244 children from kindergarten through second grade. While there were considerable causal influences of phonological processing abilities on word decoding, letter-name knowledge exerted a causal influence on subsequent phonological skills.

Studies including children at high risk for dyslexia suggest that phonological problems emerge early in childhood. For example, Scarborough (1990) reported that 65% of a group of children from families with a history of dyslexia could be diagnosed as reading disabled at the end of Grade 2. As 2½-year-olds, these children were impaired in consonant pronunciation accuracy (an index of phonological production) and produced shorter, less syntactically complex utterances; at 3 years of age, object-naming abilities (a measure of phonological recoding in lexical access) and receptive vocabulary were less developed; and at 5 years these children showed problems in tasks requiring object naming, rhyming (an index of phonemic awareness), and letter-sound knowledge. In a more recent study by Gallagher et al. (2000), 63 children who all had a first-degree dyslexic relative and 34 control children of similar non-verbal intelligence from non-dyslexic families were followed from 3 to 6 years of age. Fifty-seven percent of the at-risk-for-dyslexia group showed a delay in literacy development at 6 years, compared with 12% of the control group. Retrospective analyses revealed that at 3 years of age, literacy-delayed at-risk children scored more poorly than both controls (n = 30) and literacy-normal at-risk children on general language tests, letter knowledge, and phonological tasks (nursery rhyme knowledge, novel-stimulus word repetition, memory span for digits). The studies by Scarborough (1990) and Gallagher et al. (2000) indicate that language difficulties in general and phonological deficits in particular occur early in children with literacy problems. Whereas Scarborough interpreted the results as a general verbal limitation associated with dyslexia, Gallagher and colleagues discussed an interactive framework in which semantic and phonological skills promote early reading acquisition.
Elbro et al. (1998) followed Danish children of dyslexic and normally reading parents from the beginning of kindergarten until the beginning of the second grade. Their data revealed that the occurrence of dyslexia in a child was significantly predicted by letter-naming ability, phoneme identification performance, and a measure of the precision of phonological production skills.

At this point, similarities of dyslexia to the dyslexia-related condition SLI (see under ‘Phenomenology of developmental dyslexia’, this chapter) become apparent in two ways: Firstly, language-disordered children were found to be deficient on a series of phonological tasks (e.g., Kamhi & Catts, 1986; Gathercole & Baddeley, 1990; Catts, 1993; Leitao et al., 1997). Secondly, Catts (1993), studying children with SLI longitudinally, reported a significant association between measures of receptive and expressive language abilities, phonological awareness (deleting and blending of syllables/phonemes), and phonological recoding in lexical access (rapid automatized naming) at kindergarten and reading outcome in the first two grades. The phonological tasks, however, were observed to be the strongest predictors of single-word and pseudoword reading.

The studies by Scarborough (1990), Gallagher et al. (2000), Elbro et al. (1998), and Catts (1993) indicate a changing pattern of language difficulties from generally language to specifically phonologic difficulties over time for children who later become reading impaired. It was further shown that phonological processing difficulties endure into adulthood and even exist in dyslexic individuals who had become good readers (e.g., Pennington et al., 1990; Bruck, 1990; 1992; Elbro et al., 1994; Gallagher et al., 1996). In line with behavioral data, functional brain-imaging studies have revealed atypical cortical activation patterns in dyslexic adults during engagement in various phonological tasks (e.g., Rumsey et al., 1992, Paulesu et al., 1996; Shaywitz et al., 1998; Pugh et al., 2000a). Distinct brain-activation profiles associated with phonological processing have also been reported in children and adolescents with dyslexia (Georgiewa et al., 1999; Temple et al., 2001).

In summary, there is substantial evidence that many dyslexic or reading-impaired individuals are affected by phonological processing deficits. The underlying mechanism of these deficits is still unclear. One candidate source is impaired perception of speech at the phoneme level, since dyslexic and SLI children have frequently been reported to show difficulty in segmenting, discriminating, and identifying speech sounds (for reviews see Tallal et al., 1993; Farmer & Klein, 1995; Bishop, 1997). However, its causal primacy is under debate as well. Contrary to the proponents of the phonological deficit hypothesis, others hold the opinion that such problems derive from a more fundamental auditory perceptual deficit.
that affects processing of all sounds, not just speech (for reviews see Tallal et al., 1993; Farmer & Klein, 1995; Tallal et al., 1998). This approach is discussed in the following section.

**Auditory temporal processing deficit**

Research on auditory perception in language-based learning disorders has particularly been influenced by the work of Tallal and her colleagues. Tallal (1984; Tallal et al., 1993) has proposed that dyslexics’ phonological impairments may result from an underlying deficit in auditory temporal processing. **Temporal processing** refers to the rate of information processing essential for encoding brief and rapidly changing or rapidly occurring successive events (Tallal, 1984). Much of Tallal’s research has involved children with SLI. However, it was the striking similarity between SLI and developmental dyslexia that led her to assume that the temporal processing deficit may be causally related to both conditions. In what follows, relevant studies giving rise and lending support to the hypothesis of an auditory temporal processing deficit in language-based learning disorders are described. Conflicting findings are then discussed briefly regarding the nature and generality of the auditory deficit.

Within the framework of four studies, Tallal and Piercy (1973a,b; 1974; 1975) reported data from 12 SLI children ranging from 6 to 9 years and 12 age-matched control children who developed language normally. In the first study, Tallal and Piercy (1973a) employed two different complex tones, each of 75 ms in length. The children were trained to associate each tone, presented in isolation, with a corresponding response panel. Once the association had been learned, sequences of two tones (1-1, 1-2, 2-1, or 2-2) were presented, and the child was asked to push the respective panels in the correct order (= repetition task requiring sequencing). The SLI children performed above the level of chance when the tones were separated by an interstimulus interval (ISI) exceeding 300 ms, but their performance deteriorated when shorter ISIs were used. The control group, on the other hand, maintained high-level performance at shorter ISIs, and scored above-chance levels with ISIs as brief as 8 ms. A similar pattern of results was demonstrated when the child was asked to indicate whether two tones in a series were the same or different (= same-different task requiring discrimination). This suggests that the sequencing difficulty experienced by the children with SLI was secondary to their impairment in discriminating rapidly occurring tones.

In their second study, Tallal and Piercy (1973b) manipulated the tone duration in addition to the ISI. When the tone duration was 250 ms, however, the SLI children repeated the two-element patterns as accurately as the control children at all ISIs tested (8-428 ms).
When the tone duration was 175 ms, the SLI children were outscored by the controls at ISIs of 15 ms or less. Tones of 125 ms or 75 ms differentiated the two groups even for ISIs less than or equal to 150 ms. Thus, the total duration of the stimulus pattern appeared to be critical to the SLI children’s performance.

On the basis of the results of these two studies, Tallal and colleagues concluded that children with SLI exhibit a deficit in perceiving auditory events occurring in rapid succession – within tens of milliseconds. This brief time scale led the researchers to focus on the phonemic level of speech. Consonant and vowel phonemes are perceived by different temporally coded acoustic cues (e.g., Liberman et al., 1967). The acoustic cue for vowels is the relation among the frequencies (called formants) which remain uniform throughout the stimulus and have a relatively long duration. Stop consonants, in contrast, are characterized by brief transitional periods during which the frequencies of the formants change very rapidly over time. Figure I.1 illustrates the acoustic spectra that will, when converted to sounds, be perceived as the vowel phonemes /ɛ/ and /æ/, and the stop-consonant syllables /ba/ and /da/.

![Figure I.1.](image)

The formant frequencies of the two vowels are stationary and differ from each other by an approximately constant amount throughout their entire 250-ms duration. The two syllables

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9 Stop consonants or plosives are produced by closing the vocal tract. Most languages have six stop consonants in common: b, d, g, p, t, k. The first three stops are voiced (i.e., they cause vibration of the vocal cords), the latter are unvoiced (i.e., vocal cords are not vibrating) (e.g., Liberman et al., 1967).
share identical stationary formants of the vowel /a/ for most of the 250 ms and differ only in the initial portion, during which the frequencies change within some 40 ms. Consequently, the perception of a difference between stop-consonant syllables crucially depends on the accurate analysis of the very brief formant transitions.

Given the phonemes’ temporal characteristics, Tallal and Piercy (1974) had hypothesized that children with SLI (i) would be unimpaired in distinguishing the vowel phonemes /ɛ/ and /æ/ of 250 ms, and (ii) would be impaired in distinguishing the stop consonant-vowel syllables /ba/ and /da/, which have a total duration of 250 ms including a formant transition period of 43 ms. The experimental results confirmed their predictions: No differences were observed between SLI and control groups in sequencing the two vowels. Whereas all control children succeeded in distinguishing /ba/ and /da/, only two out of 12 SLI children could discriminate and sequence the syllables correctly.

In their final study, Tallal and Piercy (1975) aimed at investigating whether SLI children’s poor performance on tests with stop-consonant syllables was due to an inability to utilize brief cues within phonemes or rather derived from an impairment in processing transitional speech elements *per se*. The children were presented with two pairs of synthesized speech stimuli: (i) the vowel-vowel pairs /ɛI/ and /æI/, in which the first (stationary) vowel was 43 ms in duration, followed by the second of 207 ms; and (ii) the consonant-vowel pairs /ba/ and /da/, in which the initial transitional period was extended from 43 ms to 95 ms and the duration of the stationary vowel was reduced to 155 ms. Now, the SLI children displayed problems with the (temporally reduced) vowel-vowel syllables, but performed as accurately as the age controls did on the (temporally extended) stop-consonant pairs. This finding was assumed to confirm the *brevity* of the contrastive information – independent of its transitional or stationary character – as the major player in SLI children’s sound difficulties.

Tallal et al. (1980a) administered a perceptual-constancy task to 34 SLI children ranging from 5 to 9 years and 38 age-matched control children who were normal language learners. The perceptual-constancy task required the child to push one panel for stimuli comprising the phoneme /b/ (viz., /ba/, /be/, /bi/), and another panel for stimuli comprising /d/ (viz., /de/, /dae/, /di/). The performance of the SLI group was inferior to that of the control group: 41% of the SLI children versus 63% of the controls managed this task. The children who succeeded participated in another task (Tallal et al., 1980b) in which bisyllabic words

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10 In speech, perceptual constancy refers to the ability to detect acoustic information that remains constant over changes in phonetic context, characteristics of the speaker (e.g. gender), and speaking rate (Liberman et al., 1967; Strange et al., 1983). Perceptual constancy for the phonemes /b/ and /d/ occurring in various vowel environments (i.e., phonetic context) was assessed in the Tallal et al. (1980a) study.
were approximated by presenting the syllables adjacent in time (e.g., /dæ/ 50-ms interval /di/, for daddy). The children with SLI had greater difficulty in sequencing the syllables than the control children. The researchers concluded that the added linguistic redundancy available in these stimuli was not sufficient to counteract the auditory processing deficit observed in children with SLI.

In a study by Tallal and Stark (1981), 35 SLI children and 38 control children with normal language skills between the ages of 5 and 8 years were exposed to a variety of speech sound contrasts. The task required the child to press a panel when the target syllable was heard, and to make no response when the other sound occurred. The SLI children performed below the level of the age-matched control group on the syllable contrasts /ba/-/da/, /da/-/ta/, and /sa/-/ʃa/. No group differences were obtained for the contrasts /dab/-/dæb/, /sa/-/ʃa/, and /ε/-/æ/. This pattern of results is interesting in two different ways: Firstly, the two vowel stimuli /ε/ and /æ/ were only 40 ms in duration. Tallal and Piercy (1975) had employed the same vowels and durations in the concatenations /εI/ and /æI/, and found the SLI children to be significantly impaired in perceiving these stimuli (see above). Tallal and Stark (1981) concluded that rather than being impaired in processing all brief cues, SLI children have particular constraints in responding correctly to stimuli that not only include brief acoustic cues but also are followed in rapid succession by other acoustic information. Thus, the SLI group had difficulty with the stop-consonant contrasts (viz., /ba/-/da/, /da/-/ta/), but performed normally on the isolated brief vowels as well as on syllables in which the relatively long lasting cues provide the contrastive information (viz., the vowels in /dab/ versus /dæb/, each of some 170 ms and the 100-ms silence between the offset of the fricative\textsuperscript{11} consonant and the onset of the vowel formants in /sta/ versus /sa/). Secondly, the syllables /sa/ versus /ʃa/ differed spectrally throughout the initial 130 ms of the fricative consonants. Since the SLI children were found to have problems with these syllables, though their contrastive cue is not brief in nature, Tallal and Stark suggests that children with SLI may also be impaired in discriminating certain spectral cues. Given these findings, the researchers hypothesize that deficient auditory processing in children with SLI may be the result of aberrant acoustic masking mechanisms.

Supportive evidence for this assumption was offered in a masking experiment conducted by the Merzenich group (Wright et al., 1997). Detection of an auditory (test) stimulus – in this case a brief simple tone of 20 ms – is suppressed when followed

\textsuperscript{11} Fricative consonants are produced by constricting the vocal tract. For example, to generate /s/, air is pushed between the tip of the tongue and the ridge above the upper teeth (e.g., Liberman et al., 1967).
immediately by a another (masking) stimulus – in this case a hissing sound. This phenomenon, known as backward masking, was found to be massively enhanced in school-age children with SLI: for them the test stimulus had to be nearly twice as loud as for the age controls, in order to be detected. Importantly, the result could not be traced to generally elevated perceptual thresholds or to attentional lapses, because the SLI and control groups performed at equivalent levels when a long tone of 200 ms was embedded in the masker. Furthermore, it was shown that backward masking in SLI children was greatest when the two stimuli were composed of similar frequencies, i.e., shared a common spectral context.

Thus far, many children with SLI have difficulty with tasks involving rapid auditory processing, though at least some of these children exhibit impaired frequency discrimination abilities as well (see also Stark & Heinz, 1996b). It deserves mention, however, that some children with SLI also have been reported to be impaired in sequencing more slowly presented sounds (Tallal et al., 1981; Bishop et al., 1999).

The same series of auditory tasks described previously (Tallal & Piercy, 1973b, 1974) was administered to children diagnosed with developmental dyslexia. Using the Tallal and Piercy (1973b) task, Tallal (1980) examined discrimination and sequencing skills in 20 dyslexics averaging 9 years of age. She observed that dyslexic children, as a group, performed as well as younger normally developing controls (n = 12) provided that the tones were presented slowly (ISI = 428 ms) but did less well when the presentation rate was increased (ISIs declining from 305 to 8 ms). In a further step, Tallal (1980) tested whether the performance of the dyslexic children at shorter ISIs is related to their reading abilities. Errors in auditory rate processing were found to correlate significantly with errors in tests of spelling ($r = .67$), word discrimination ($r = .64$), word knowledge ($r = .64$), and pseudoword reading ($r = .81$). The latter highly positive correlation is of particular importance since it supports an association between auditory temporal processing and phonological decoding (i.e., the use of grapheme-phoneme conversion rules). More to the point, efficient processing of rapidly changing acoustic input has been assumed to affect the ability to learn to use phonetic codes correctly (Tallal, 1980).

Reed (1989) confirmed and extended the findings of Tallal (1980) in groups of dyslexic and normally reading children with an average age of 9 years. Sequencing performance at ISIs varying between 10 and 400 ms was assessed using pairs of isolated vowels and pairs of stop consonant-vowel syllables with a duration of 250 ms (as in the Tallal & Piercy, 1974, study) and pairs of complex tones with a duration of 75 ms (as in the Tallal, 1980, study). Reed found that the dyslexic children were increasingly impaired relative to
their normally reading peers as ISIs decreased for tones and consonant syllables, but performed within the normal range when vowels were employed. The dyslexic children’s difficulty with brief and rapidly changing cues and their mastery with long-duration stimuli were also seen in tasks simulating rather natural speech conditions: The dyslexic children were impaired when they were asked to match pictures with auditory words differing in their initial stop consonant, but had no unusual problems in sequencing the 250-ms vowels presented in a white noise background. Interestingly, the performance profile on vowel stimuli embedded in noise matches precisely that observed in the tonal masking experiment by Wright et al. (1997; see above). Similar to Tallal (1980), Reed (1989) aimed at exploring whether temporal processing deficits and phonological impairments are characteristic of the same children with dyslexia. Therefore, the children also participated in a categorical perception task, which included a nine-item continuum varying from /ba/ at one endpoint to /da/ at the other endpoint. The results revealed that the dyslexic children were impaired at discriminating syllable pairs that cross the boundary of the phonetic categories /ba/ and /da/. Furthermore, they were less consistent than normal readers in identifying syllables near the phonetic boundary. Thus, the dyslexic group appeared to show a less sharply defined boundary between the phonological categories /b/ and /d/. According to Reed (1989), the perceptual difficulty with brief and rapidly changing cues may contribute to inadequately defined sound representations and hence interfere with the processing of phonological information.

The studies of Tallal (1980) and Reed (1989) indicate that an impairment in temporal processing is present in children with dyslexia, as it might be co-occurring with a phonological deficit. However, Tallal (1980) observed that performance in pseudoword reading and auditory rate processing varied considerably within her dyslexic group. In particular, 55% of the dyslexic children scored within normal limits when presented with rapidly occurring tones, while 45% of the dyslexics showed a similar, though less impaired, pattern of performance to that observed in SLI children (see Tallal & Piercy, 1973b). Given the strong correlation between rapid auditory processing and pseudoword decoding (see above), Tallal inferred that this variability suggest two subgroups of developmental dyslexia: one group of dyslexic children with concomitant oral language delay, who exhibit both deficits in temporal processing and “phonics skills” (Tallal, 1980, p.188) and another group of

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12 Categorical perception denotes that discrimination of different sounds sharing the same phonological category is poor while discrimination of sounds belonging to different phonological categories is quite good, even though the physical differences between the two types of sound pairs are identical (e.g., Liberman et al., 1967). The mechanism of categorical perception is necessary to support perceptual constancy in speech (see footnote 10).
dyslexics with normal language skills showing none of these deficits. In supporting this, Tallal and Stark (1982) referred to the finding that dyslexic children without oral language delay differed from normal readers neither in sequencing rapid tones nor in their knowledge of word parts, which is a measure of phonics skills. However, to completely support Tallal’s subgroup hypothesis, dyslexic children with normal oral language should be compared both with normally developing peers and dyslexic children with concomitant oral language weakness. Exactly that was carried out by Heath et al. (1999), whose findings provide no evidence for the subgroup’s profile proposed by Tallal.

In the context of experimental training, Merzenich and Tallal investigated school-age children who demonstrated a severe delay in oral language, reading problems, and “marked” (Merzenich et al., 1996, p.80) deficits in temporal processing. Exercises designed to train children’s auditory temporal processing skills resulted not only in improvements of these skills but also of their performance on phonological and language tests (Merzenich et al., 1996; Tallal et al., 1996). This finding indicates the co-occurrence of oral language deficits, reading disability, and impaired temporal and phonological processing, but the relationship of auditory temporal processing to reading disability has nevertheless been blurred by the researchers’ designation of the children as ‘language learning impaired’. Thus, it remains unclear whether the children tested are dyslexic with concomitant oral language delay or primarily language-impaired with signs of dyslexia.

Although it has been reported that problems in auditory processing can change with age (Bernstein & Stark, 1985; Lincoln et al., 1992), adults with developmental dyslexia were found to be impaired relative to normally literate controls on several tasks involving reception of rapidly changing or rapidly successive acoustic inputs. For example, Hari and Kiesilä (1996) employed trains of binaural clicks – four left-ear clicks followed by four right-ear clicks – which produce an illusory perception of saltatory sound movement – from left to right – at short ISIs. In controls, this movement illusion disappeared at ISIs exceeding 90-120 ms. However, in dyslexics, who as a group were inferior to the controls on different measures of phonological processing, the illusion persisted up to ISIs of 250-500 ms. “Dyslexic adults thus seem to have a deficit in the processing of rapid sound sequences, which is also manifested in significant delays in their conscious auditory percepts.” (Hari & Kiesilä, 1996, p.138).

Helenius et al. (1999a) observed aberrant auditory stream segregation in adults with dyslexia. Rapid alternation of high and low tone sequences led to the perception of two separate streams, i.e., a high- and a low-pitched stream. In the control group, the stream
segregation occurred at stimulus onset asynchronies falling below 130 ms, while in the dyslexic group this was true at 210 ms. In addition, stream segregation correlated significantly with pseudoword recognition time \( (r = .72) \) in dyslexics. That is, dyslexic adults who were slow in accessing phonological information tended to display abnormal processing of rapid tone sequences.

Witton et al. (1998) reported adult dyslexics to have a specific deficit in detecting low rates of auditory frequency modulation (2 Hz and 40 Hz) that might reflect a weakness in response to rapidly successive acoustic cues relevant for speech discrimination. Furthermore, less sensitivity to low-rate frequency modulations was associated with poor phonological decoding in both dyslexic and normally reading adults \( (r = .41 \text{ for } 2 \text{ Hz}, r = .60 \text{ for } 40 \text{ Hz}) \).

Taken together, an auditory temporal dysfunction may be present and associated with phonological deficits in a proportion of dyslexic individuals. Findings provided by the adult literature on auditory perception seem to be less conflicting than those in the child population. However, an influence of test method cannot be ruled out. Usually, the tasks administered to adult dyslexics are more challenging than those presented to children. Tallal’s (1984; Tallal et al., 1993) assumption that a temporal processing deficit is causally related to phonological impairments in dyslexia is in its strong form not tenable. Relationships between the two variables are correlational at best. Valuable information could come from prospective longitudinal studies including children who are at high familial risk for dyslexia. Indeed, Benasich and Tallal (1996; Benasich, 1998) have shown that 55% of the infants born into families with a history of language-based learning impairments are distinguished from babies of non-language/learning-impaired families when they are 7 months old (the average age at which they were first assessed) in their ability to successfully discriminate rapidly successive sounds \( (\text{ISI} < 300 \text{ ms}) \). No performance differences were observed on slow sequences \( (\text{ISI} = 500 \text{ ms}) \). The performance profile of the at-risk infants resembles the deficits recorded in school-age children with SLI and dyslexia (see above). When the children are followed over time they are consistently language delayed, whereby infant measures on auditory temporal processing were proven to be significant predictors of language outcome at 2 years of age. It remains to be seen whether the auditory temporal dysfunction predicts phonological deficits or even dyslexia or whether – according to the proponents of the phonological deficit theory – the perceptual problems are rather associated symptoms that are milder and less consistent in occurrence (for a review see Studdert-Kennedy & Mody, 1995).

According to the current version of the temporal processing hypothesis, the deficit is not limited to the auditory modality. Similar constraints in coping with brief and rapidly
occurring events have also been reported for the visual and tactile modality in children with SLI and dyslexia (for reviews see Tallal et al., 1993; Farmer & Klein, 1995). A multimodal or “pansensory” (Tallal et al., 1993, p.27) temporal processing deficit – though still highly controversial – is assumed to have a particularly severe impact on the development of spoken and written language (Tallal, 1984; Tallal et al., 1993). Since the 1980s, temporal sensitivity in the visual domain has been investigated primarily in terms of the magnocellular deficit hypothesis for dyslexia. This hypothesis is considered next.

**Magnocellular deficit**

About 1900, Morgan, Hinshelwood, and Orton described the first cases of developmental dyslexia (Warnke, 1990). At the time the children were thought to be suffering from 'word blindness' as perceptual problems, which were caused mainly by visual confusion, were assumed to be at the root of the children's difficulty with written language. Since the 1970s, research on the etiology of dyslexia has focused on verbal, specifically phonological, skills (see above). However, in the last 10-15 years the idea that dyslexia is associated with impaired visual processing has been making a comeback. Especially, the work of Lovegrove and colleagues in the 1980s has stimulated further research on visual processing in developmental dyslexia (e.g., Lovegrove et al., 1982, 1986; Martin & Lovegrove, 1984, 1987, 1988; Slaghuis & Lovegrove, 1984, 1985, 1986).

Lovegrove’s experiments were designed to test for visible persistence¹³ and contrast sensitivity differences between normal and dyslexic readers using sinusoidal waveform gratings. The series of studies indicate that children with dyslexia display (a) longer-lasting visible persistence at low spatial frequencies (i.e., coarse gratings) and (b) less sensitivity to gratings, particularly at low spatial frequencies, low contrasts, low luminances, and high temporal frequencies (i.e., fast flickering gratings) in comparison to normal readers. In contrast, dyslexic children often showed shorter visible persistence and slightly elevated contrast sensitivity at higher spatial frequencies (Lovegrove et al., 1982, 1986; Martin & Lovegrove, 1984, 1987, 1988; Slaghuis & Lovegrove, 1984, 1985, 1986). Furthermore, it was found that abnormalities in visual processing occur in approximately 75% of a dyslexic group

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¹³ “Visible persistence is defined as any continued visible response occurring after stimulus offset that is phenomenally indistinguishable from that occurring during the actual presence of the stimulus.” (Lovegrove et al., 1982, p.309). In Lovegrove’s experiments (e.g., Martin & Lovegrove, 1984), visible persistence was typically assessed by presenting sine wave gratings in close succession. Subjects were required to report whether they observed a blank interval between the gratings or not. Failure to detect a blank interval indicated that the image (or visible persistence) of the first stimulus was still apparent at the onset of the second.
Auditory Processing in Dyslexia – General Introduction

(Slaghuis & Lovegrove, 1985; Lovegrove et al., 1986), are present before children commence reading practice (Lovegrove et al., 1986), and continue into adulthood (Slaghuis et al., 1996).

Psychophysical studies of different laboratories have confirmed Lovegrove’s findings (e.g., Felmingham & Jakobson, 1995; Borsting et al., 1996). Evidence is also provided by electrophysiological studies to the effect that visual evoked potentials in dyslexic individuals were reduced or delayed for stimuli with low spatial and high temporal frequencies (e.g., Livingstone et al., 1991; Lehmkuhle et al., 1993; Kubová et al., 1995).

Lovegrove and colleagues interpreted these results in the framework of the transient and sustained channels of the visual system. The transient system responds rapidly to low spatial and high temporal frequencies, low contrasts, and low luminances. The sustained system responds more slowly to medium and high spatial frequencies, low temporal frequencies, medium and high contrasts, as well as color differences (e.g., Maunsell & Van Essen, 1983; Shapley, 1990; Merigan & Maunsell, 1993; Jenner et al., 1999). Transient visual processing is mainly mediated by large magnocells that comprise the magnocellular pathway between retina and cortex, whereas sustained processing depends on the sensitivity of the smaller parvocells comprising the retino-cortical parvocellular pathway (e.g., Merigan & Maunsell, 1993; Milner & Goodale, 1995). On the basis of primate and human visual studies, Breitmeyer and Ganz (1976) suggested that with each saccadic eye movement transient/magnocellular channels normally inhibit sustained/parvocellular channels so that the eidetic image of the previous fixation does not persist and mask the subsequent fixation. During reading, such masking would cause visual confusion (i.e., the letters would jumble up). Lovegrove and colleagues (e.g., Lovegrove et al., 1986) therefore proposed that the visual deficit in developmental dyslexia reflects a failure of the transient system on sustained inhibition. A number of independent research groups, however, have found the magnocellular/transient system rather than the parvocellular/sustained system to be suppressed during saccades (e.g., Burr et al., 1982; 1994). In addition, several psychophysical and electrophysiological studies failed to confirm that reduced contrast sensitivity is associated with dyslexia (e.g., Victor et al., 1993; Gross-Glenn et al., 1995; Walther-Mueller, 1995; Hayduk et al., 1996; Johannes et al., 1996). Two aspects are discussed in this context: (a) spatial-temporal contrast sensitivity tasks are not sensitive enough for stimulating the magnocellular retino-cortical stream; and (b) only a subgroup of the dyslexic population exhibits abnormal visual magnocellular function.

Ad (a) task sensitivity: Experiments in monkey visual cortex indicate that motion stimuli are the most selective for the magnocellular system (Newsome & Paré, 1988). Indeed,
children and adults with developmental dyslexia have been shown to be less sensitive than controls to moving stimuli both in psychophysical (e.g., Cornelissen et al., 1995; Witton et al., 1998; Everatt et al., 1999; Slaghuis & Ryan, 1999; Talcott et al., 2000) and brain-imaging (Eden et al. 1996; Demb et al., 1997, 1998) studies. Furthermore, performance in pseudoword reading – a measure of phonemic awareness – was found to be correlated with the sensitivity of detecting visual coherent motion in dyslexic adults (Witton et al., 1998). Similarly, Cornelissen et al. (1998) observed that dyslexic children’s letter errors were best explained by independent contributions from motion detection and phonemic awareness.

Unlike grating contrast-sensitivity measurements which were dominated by the retino-thalamic pathway, motion detection involves more centrally functions of the magnocellular system (see Stein et al., 2000b). Centrally functions are assumed to be closer to cognitive skills required for reading. Therefore, alternative more centrally located mechanisms by which a magnocellular/transient-system deficit could cause visual confusion during reading have been postulated (see Stein & Walsh, 1997), such as attentional deficits or destabilized binocular fixation, to name just two, whereby the latter mechanism has been supported by several studies (e.g., Stein & Fowler, 1981, 1993; Stein et al., 2000a).

Ad (b) subgroup-specific phenomenon: Borsting et al. (1996) found that in dyslexic adults contrast sensitivity abnormalities are only apparent in a subgroup of Boder’s mixed dysphonetic-dyseidetic dyslexia (see under ‘Phenomenology of developmental dyslexia’, this chapter). This finding was supported by Slaghuis and Ryan (1999) in a sample of children: In comparison to a normally reading control group, the mixed-dyslexia subgroup displayed a wide range of impaired transient processing manifest itself in reduced contrast sensitivity at low spatial frequencies, elevated motion detection thresholds, and longer duration of visible persistence. Cestnick and Coltheart (1999) observed that children classified as phonological dyslexics (see under ‘Phenomenology of developmental dyslexia’, this chapter) performed abnormally in a visual motion detection task, whereas surface dyslexics did not differ from the control children. Thus, it seems that only one subgroup of dyslexic individuals displays deviations from normal magnocellular functioning.

NEUROBIOLOGICAL CORRELATES OF DYSLEXIA

Dyslexia has long been assumed to have a neurodevelopmental origin (Hynd & Semrud-Clikeman, 1989). During the past 30 years several neurobiological correlates of dyslexia have been reported by a number of research teams. In what follows, relevant findings from post-mortem, neuroimaging, and electrophysiological studies are reviewed.
Post-mortem studies

Galaburda and colleagues have performed the only comprehensive post-mortem studies to date of diagnosed cases of developmental dyslexia (Galaburda et al., 1985; Humphreys et al., 1990; Livingstone et al., 1991; Galaburda & Livingstone, 1993; Galaburda et al., 1994; Jenner et al., 1999). All five cases (one female and four males) showed evidence of small areas of cortical dysgenesis (called microdysgenesis) including ectopias (small nests of abnormally placed neurons) and dysplasia (focally distorted cortical lamination). The microdysgenesis varied in number and location from brain to brain and tended to involve the language-relevant perisylvian cortex. Furthermore, the structural deviations tended to be lateralized to the left hemisphere in male specimens (Galaburda et al., 1985), whereas in the female brain a fairly symmetric distribution was observed (Humphreys et al., 1990; NB: specimen ORT-20-87). Because ectopias or dysplasias are found only rarely in routine autopsy analyses (or in other developmental disorders), usually omit perisylvian regions, and are located more frequently in the right side of the brain than in the left, Galaburda (1988, 1989, 1993; Sherman & Galaburda, 1999) considered the malformations to be specifically associated with dyslexia. According to Galaburda, the microdysgenesis might reflect neuronal migration errors that occurred during fetal development.

Changes in the pattern of hemispheric asymmetry of the planum temporale also were seen in dyslexic brains. The planum temporale is a triangular landmark situated on the supratemporal surface just posterior to the first Heschl’s gyrus, inside the sylvian fissure. The left planum coincides with part of Wernicke’s speech comprehension area (e.g., Galaburda, 1993; Shapleske et al., 1999). Large post-mortem studies (Geschwind & Levitsky, 1968; Wada et al., 1975) each including 100 normal adult brains found that it was symmetrically sized between the hemispheres in 16%, whereas 10.5% showed a rightward asymmetry and 73.5% a leftward. Corresponding figures reported on 307 ordinary fetal or neonatal specimens were 29%, 16%, and 54% (Wada et al., 1975; Chi et al., 1977). Consequently, the planum temporale is thought to be an important substrate of left-hemispheric language lateralization (Geschwind & Levitsky, 1968; Wada et al., 1975). Going back to the five dyslexic brains (Galaburda et al., 1985; Humphreys et al., 1990) none was reported to show the typical planar asymmetry favoring the left side; instead these autopsy specimens exhibit the symmetrical

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14 Details concerning the male subjects examined in the above-mentioned six studies are somewhat inconclusive.

15 Anatomic asymmetry of the planum temporale is not unique to humans. For instance, Gannon et al. (1998) demonstrated left-hemispheric size predominance of the planum temporale in chimpanzees. Thus, language lateralization may have been based on this physiogenetically older asymmetry.
type due to an enlarged right-hemisphere planum. Galaburda et al. (1987) have proposed that symmetry reflects reduced cell death in the right planum temporale during late fetal development, which leads to enhanced survival of neurons, forming improper connections and resulting in a redefinition of the cortical architecture.\footnote{So far, animal models have lent no support to Galaburda’s failure-of-neuronal-loss hypothesis (Galaburda, 1994).}

Another set of post-mortem examinations was performed on thalamic structures, i.e., the lateral geniculate nucleus (LGN) of the visual pathway and the medial geniculate nucleus (MGN) of the auditory pathway. The magnocellular layers of the (‘visual’) LGN were found to be more disorganized in dyslexic than in non-dyslexic brains (Livingstone et al., 1991). Furthermore, magnocell bodies were on average 27% smaller and appeared more variable in size and shape in the brain of dyslexic individuals relative to those of controls. Neither the parvocellular lamination nor the parvocell sizes of the LGN differed between the population specimens. In the auditory system, Galaburda et al. (1994) reported significantly smaller MGN neurons on the left side compared with the right in the same dyslexic autopsy specimens. No hemispheric asymmetry in MGN neuronal size was observed in ordinary brains. In addition, brains of dyslexic individuals were said to exhibit a relative excess of small neurons and a relative paucity of large neurons on the left side as compared to control brains. According to Galaburda et al. (1996), the structural deviances found in the LGN of dyslexic brains were associated with a slowness in early segments of the magnocellular channels (see under ‘Magnocellular deficit’, this chapter), whereas the MGN differences may be related to the auditory temporal processing abnormalities (see under ‘Auditory temporal processing deficit’, this chapter) described in language-impaired children.

Autopsy data on neuronal tissue in the primary visual cortex (area 17) were presented in a recent work by Jenner et al. (1999). In contrast to the atypical organization in the magnocellular layers of the LGN, the (five) dyslexic brains did not show consistent changes in the size of cortical neurons receiving thalamic magno input. The researchers suggested that this inconsistency may in part be due to blending of magnocellular and parvocellular pathways or functional effects of cortico-cortical top-down projections. On the other hand, another example of changes in hemispheric asymmetry similar to that of the planum temporale was observed. That is, brains of non-dyslexic individuals comprised larger neurons in the left hemisphere than in the right, whereas dyslexic brains showed no lateralization. According to Jenner et al. (1999), the neuronal symmetry in primary visual cortex is associated with abnormality in circuits involved in reading.
To date, Galaburda’s group has presented autopsy data on nine brains of individuals (six males and three females) with a history of developmental dyslexia. Three of the male and one of the female patients were reported to have histories of delayed language acquisition (Galaburda et al. 1985; Humphreys et al., 1990). All dyslexic brains have displayed evidence of symmetric *plana temporalis* (Galaburda, 1988, 1989; Humphreys et al., 1990). Neuronal ectopias and architectonic dysplasias were observed in all male cases and two of the females (Galaburda, 1993). Other cerebrocortical deviations in dyslexic autopsy specimens such as microgyria and cortical scars were less uniform than the pattern of microdysgenesis (Galaburda, 1993). Overall, dyslexic female brains showed fewer and differently located microcortical malformations when compared to male brains (Humphreys et al., 1990). Histological differences in thalamic structures and the primary visual cortex are hitherto limited to reports on five dyslexic brains versus five (Livingstone et al., 1991; Jenner et al., 1999) or seven control brains (Galaburda et al., 1994). In interpreting the study results, Galaburda (1988, 1989, 1993; Galaburda et al., 1996) has hypothesized that dyslexia is an outcome of anomalous neural development which might derive from brain injury during the prenatal stage. Here, the chemical environment and maturation rate of relevant brain areas are assumed to interact.

To complete the picture, two post-mortem findings in language-impaired children should be briefly mentioned here. Landau et al. (1960) observed bilateral perisylvian cystic lesions with surrounding dysplasias and a severe retrograde degeneration in the MGN in a male brain. In a female autopsy specimen, Cohen et al. (1989) found a dysplastic microgyrus in the left insular cortex and decreased asymmetry of the *plana temporalis*.

Despite the robustness of most of the findings provided by Galaburda and colleagues, there are methodological issues complicating the interpretation of the results. For instance, many subjects with dyslexia had a history of comorbid disorders or prior head injuries which would have prevented their participation in neuroimaging studies (Galaburda et al., 1985; Humphreys et al., 1990). Further, the number of autopsy specimens examined so far is small. In post-mortem studies, reliable identification of microanatomical deviations in general and

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17 Figures were taken from various journals and books indexed by the bibliography databases *Medline* and *Psychinfo*.

18 In the two studies, the diagnostic terms ‘congenital aphasia’ (Landau et al., 1960) and ‘developmental dysphasia’ (Cohen et al., 1989) were used to describe the language impairment of the young patients.

19 Galaburda’s research team has shown that induced cortical lesions in newborn animals can have behavioral and anatomical effects similar to those observed in humans with dyslexia. For instance, induction of microgyria in the developing rat brain causes an impairment in fast auditory temporal processing in males. In addition, microgyric male rats exhibited more small and fewer large MGN cells when compared to sham operated males. Microgyric female rats exhibited neither neuronal MGN anomalies nor auditory temporal processing deficits (Herman et al., 1997).
the boundaries of the *planum temporale* in particular has often proved to be difficult (e.g., Kirch & Weinberger, 1986; Shapleske et al., 1999). I will return to the latter point when reviewing structural brain-imaging studies (see next section).

**Neuroimaging studies**

Based on neurobiological and cognitive theories, structural as well as functional brain-imaging studies in people with dyslexia have focused on areas subserving language. Because findings of atypical cortical asymmetry in known language regions may be related to deviances in interhemispheric transfer of information, the morphology of the *corpus callosum* has been another point of interest.

**Structural neuroimaging**

Magnetic Resonance Imaging (MRI) studies have shown that individuals with dyslexia have a higher incidence of reduced or reversed asymmetry of temporo-parietal language regions than the normal population (Rumsey et al., 1986; Hynd et al., 1990; Larsen et al., 1990; Duara et al., 1991; Kushch et al., 1993; Dalby et al., 1998; Robichon et al., 2000). Similar to the post-mortem findings by Galaburda and colleagues (see under ‘Post-mortem studies’, this chapter), in vivo MRI studies demonstrated unusual asymmetry (i.e., right = left or right > left) of the *planum temporale* in people with dyslexia (Hynd et al., 1990; Larsen et al., 1990; Flowers, 1993). Larsen et al. (1990) found that 13 out of 19 dyslexic adolescents displayed symmetric plana compared to only 5 out of 17 normal readers. Among the dyslexic readers exhibiting “pure phonological dysfunction” (p.297), all showed absence of typical leftward asymmetry of the *planum*. This led the authors to hypothesize that symmetrical *plana temporali* might be a possible neurobiological substrate for phonological processing impairment in developmental dyslexia. While in the studies of Larsen et al. (1990) and Flowers (1993) atypical asymmetry of the *planum temporale* was due to an increase in size on the right side (which is consistent with Galaburda’s post-mortem results), Hynd et al. (1990) have shown differences due to a shorter left *planum* length.

More recent MRI research has challenged the view of altered planar asymmetry in dyslexia (Leonard et al., 1993; Schultz et al., 1994; Rumsey et al., 1997b; Best & Demb, 1999; Heiervang et al., 2000; Robichon et al., 2000). For instance, Leonard et al. (1993) reported an exaggerated leftward asymmetry in a small group of compensated dyslexics compared with unaffected relatives and controls. Best and Demb (1999) observed that
dyslexic adults with a magnocellular pathway deficit (see under ‘Magnocellular deficit’, this chapter) did not depart from the left-lateralized planum temporale type. According to Best and Demb, planar asymmetry may be associated with a subgroup of dyslexia.

Concerning the dyslexia-related condition SLI, only a few MRI studies have included the planum temporale as a direct measurement. Gauger et al. (1997) found a trend towards atypical planum temporale asymmetry in a group of 11 children with SLI, whereas Preis et al. (1998) could not confirm deviances from the left-right pattern among 21 language-impaired children. To circumvent the obstacles inherit in the shape and location of the planum temporale, Plante et al. (1991) evaluated MRI scans of the broader perisylvian-language area in 16 boys with and without SLI. (The measured perisylvian area in each hemisphere contained portions of the frontal and parietal operculae, and superior temporal gyrus including the planum temporale.) While 75% of the SLI children departed from the usual left-greater-than-right asymmetry, only 25% of the controls showed atypical perisylvian configuration. Deviant asymmetry patterns were also observed more frequently in first-degree relatives of children with SLI than in control subjects (Plante, 1991).

Structures of the perisylvian area other than the planum temporale have also been found to be different both in SLI and dyslexia (Jackson & Plante, 1996; Gauger et al., 1997; Clark & Plante, 1998; Heiervang et al., 2000; Robichon et al., 2000). Participants with SLI in the Gauger et al. (1997) investigation demonstrated greater rightward asymmetry of the so-called planum+ (i.e., planum temporale and posterior ascending sylvian ramus measurements summed) and a tendency towards atypical left-right configuration of the pars triangularis (i.e., the gyrus formed by the atypical ascending and anterior horizontal sylvian rami which coincides with part of Broca’s speech production area). As to Broca’s area, a similar result was obtained by Robichon et al. (2000) in 16 adult male dyslexics. Other MRI studies targeting primarily posterior language regions, revealed no changes in planum+ lateralization in either disorder (Preis et al., 1998; Heiervang et al., 2000). Analyzing the vertical part of the planum+, the posterior ascending ramus or so-called planum parietale, Heiervang et al. (2000) found that dyslexic boys were less likely to show the expected rightward asymmetry

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20 The presentation of the results in the Gauger et al. (1997) study appears to be somewhat inconsistent: On page 1276 the authors stated that “Children with SLI … were less likely to have leftward asymmetry of the … planum temporale, … than control children” whereas on page 1277 they wrote that “With regard to interhemispheric asymmetries, the planum temporale … [was] not significantly different for the two subject groups”.

21 Gauger et al. (1997) did not make clear that the group differences in pars triangularis asymmetry represented only a trend (see also footnote 20): Under ‘Results’ on page 1276 the authors wrote that “Children with SLI … were less likely to have leftward asymmetry of the pars triangularis, … than control children”. Statistical analysis of the asymmetry coefficient presented on page 1277 did not indicate significant results, however.
than normally reading controls. Several studies in dyslexia and SLI reported no alterations in planum parietale asymmetry, however (Leonard et al., 1993; Gauger et al., 1997; Rumsey et al., 1997b; Preis et al., 1998).

Furthermore, a less common type of gyral morphology (i.e., polygyria) in the posterior sylvian region was far more likely to occur in children with SLI and their first-degree relatives than in control subjects (Jackson & Plante, 1996). Clark and Plante (1998) found that an extra sulcus in the anterior perisylvian-language area was associated with poor language skills but not with a positive family history of developmental language disorders. Comparable to the Jackson and Plante (1996) findings in children with SLI, Leonard et al. (1993) reported a higher incidence of added gyri between the postcentral sulcus and posterior ascending ramus in a group of compensated dyslexics. In contrast, the results by Hiemenz and Hynd (2000) and Robichon et al. (2000) failed to support a possible association between sulcal pattern morphology and diagnosis of developmental dyslexia.

In summary, changes in perisylvian-language regions have been reported for dyslexia as well as SLI. A very small number of studies including families affected by language-based learning impairments suggests that certain perisylvian deviances might put language learning at risk. However, there has been no brain structure for which MRI findings have been replicated consistently. The planum temporale has been the most prominent landmark investigated in dyslexia. While some studies indicated reduced or absent left-right planar asymmetry in people with dyslexia others did not. The inconsistency of the studies examining the planum temporale may be attributed to several factors: (1) Research groups disagree on how to define the boundaries of the planum temporale; its structural ambiguity has led to some imaging measurements of unidimensional lengths rather than surface area (for a review see Shapleske et al., 1999). (2) Different measurement techniques used to acquire images and to measure anatomical regions are associated with considerable variability in planar surface areas across studies. For example, Best and Demb (1999) compared three measurement methods on the planum temporale in five dyslexic and five normally literate adults. The first two methods adopted from relevant MRI studies included the tissue between Heschl’s sulcus and the terminal upswing of the posterior ascending sylvian ramus, though the second one took into account neither the shape of the planum nor the small sulci on its surface. The third method approximated those used in Galaburda’s post-mortem work revealing solely the bidimensional area on the superior surface of the temporal lobe. The results showed that both participant groups became less left-lateralized using the second and third procedure that exclude sulcul tissue to an increasing degree. (3) Variation in certain characteristics of the
participants (e.g., handedness, gender, intellectual capacity, oral language skills, or socioeconomic background) across studies might obscure the relation between planar asymmetry and dyslexia (Eckert et al. 2001; for reviews see Beaton, 1997; Lambe, 1999; Shapleske et al., 1999; Eckert & Leonard, 2000). For instance, given that non-right-handedness is related to reduced or reversed asymmetry of the planum temporale and given that most studies reported normal distributions of handedness within the dyslexic group, careful control for handedness is essential in imaging studies of dyslexia. (4) Finally, certain methodological flaws, such as small sample sizes, criteria used to define dyslexia, the heterogeneity of the disorder, and codiagnoses (e.g., attention-deficit/hyperactivity disorder) might also contribute to conflicting information regarding morphometric changes in this landmark (e.g., Shapleske et al., 1999; Eckert & Leonard, 2000).

Although still highly controversial, it has been proposed that reported findings of reduced cerebral asymmetry in dyslexia (and SLI) may be associated with anomalous interhemispheric pathways coursing through the corpus callosum to the perisylvian-language regions (Filipek, 1995). The corpus callosum subserves communication and integration between the hemispheres and has been shown to be topographically organized with projections from specific cortical areas to specific callosal regions (De Lacoste et al., 1985; Pandya & Seltzer, 1986). Based on animal models, Galaburda’s group (Rosen et al., 1989; Galaburda et al., 1990) has hypothesized that the commissural connections between the hemispheres are inversely related to cerebral asymmetries. Thus, more symmetric brains have a stronger interhemispheric connectivity, which may be reflected by a larger size of the corpus callosum and vice versa. To date, there are five studies using MRI techniques in order to compare corpus callosum size between individuals with dyslexia and non-dyslexic controls (Duara et al., 1991; Larsen et al., 1992; Hynd et al., 1995; Rumsey et al., 1996; Robichon & Habib, 1998). Duara et al. (1991) observed that the most posterior segment of the corpus callosum termed ‘splenium’ was larger in a group of 21 dyslexic adults than in 29 controls. However, this effect was primarily accounted for by dyslexic female participants. In addition, both the (most anterior) genu area and the corpus callosum in general were larger in female than in male dyslexic adults. Larsen et al. (1992) failed to find differences of the total callosal area or the splenium in a predominantly male sample of 19 dyslexic adolescents and 17 normal readers. They also reported no deviances in size of the corpus callosum in subgroups of dyslexia related to reading profile or symmetry/asymmetry of the planum temporale. Studying children, Hynd et al. (1995) noted completely different results with the dyslexic group (n = 16) showing a smaller genu region than the equally-sized control group.
Furthermore, moderate positive correlations were found between overall reading achievement and the (region of interest) measurements for the genu \( (r = .40) \) and splenium \( (r = .35) \) in these children. Rumsey et al. (1996) reported an increase in the area of the posterior third of the corpus callosum – roughly corresponding to the splenium and its rostrally adjacent segment, the isthmus – in dyslexic men \( (n = 21) \). Likewise in a group of 16 adult male dyslexics, Robichon and Habib (1998) showed a larger total callosal area, in particular in the isthmus but found that this result was accounted for by right-handed participants.

Taken together, of the five studies reviewed above three found an increase in size of the corpus callosum in adults with dyslexia, especially in the splenium (Duara et al., 1991; Rumsey et al., 1996) and the isthmus (Rumsey et al., 1996; Robichon & Habib, 1998). The isthmus contains fibers from the superior temporal and posterior parietal regions; the splenium involves all of the fibers connecting occipital cortex, but also links the superior parietal lobules and the temporo-parieto-occipital junctional area, the region including the planum temporale (De Lacoste et al., 1985; Pandya & Seltzer, 1986). Thus, these callosal segments are associated with posterior language regions, in which atypical cerebral asymmetries and other cytoarchitectonic deviances have been reported in dyslexia (e.g., Hynd et al., 1990; Larsen et al., 1990; Flowers, 1993; Leonard et al., 1993; Kushch et al., 1993; Dalby et al., 1998). The works of Larsen et al. (1992) and Hynd et al. (1995) on corpus callosum morphometry in dyslexia provide conflicting results, however. In explaining this, differences in subject characteristics (e.g., age, gender, handedness, comorbidity, or intellectual ability) as well as procedural variations in the methods used to acquire the scans and to define and measure the callosal subregions (of interest) may play an important role (Filipek, 1995; Beaton, 1997; Lambe, 1999).

There are only three MRI studies presenting corpus callosum data in language-impaired children (Njiokiktjien et al., 1994; Gauger et al., 1997; Preis et al., 2000). Gauger et al. (1997) mentioned normal total callosum size in children with SLI, although they tended to be less likely to show typical asymmetry patterns in perisylvian-language regions. In a large study including 110 children, Njiokiktjien et al. (1994) found the corpus callosum to be larger in an SLI group exhibiting both familial dysphasia and dyslexia compared with non-familial cases. The only study, to date, measuring subregions of the corpus callosum in language-impaired children is that of Preis et al. (2000). Evaluating the MRI scans of the same children showing the usual anatomical asymmetry of the temporal and parietal plana (Preis et al., 1998; see above), no differences for absolute corpus callosum size and its subareas were
observed between the SLI and the control group. In view of the limited studies it is not possible to draw firm conclusions regarding callosal morphology in SLI.

As repeatedly stressed, it is apparent that no consistent structural correlates have been associated with developmental dyslexia (or SLI) using MRI techniques. Several factors possibly accounting for the inconsistent findings have been outlined earlier. Because the relationship between neurostructural deviances and behavioral measures is under discussion, more insight has been expected from functional brain-imaging methods.

**Functional neuroimaging**

Among the developmental disorders, functional measures of cerebral blood flow or metabolism such as Positron Emission Tomography (PET) and functional Magnetic Resonance Imaging (fMRI) have been applied most often in studies of (adults with) dyslexia (Filipek, 1999). As for the condition SLI, neuroimaging articles primarily report on Single Photon Emission Computer Tomography (SPECT) data (Denays et al., 1989; Tzourio et al., 1994; Chiron et al., 1999). Their findings could be summarized to the effect that they further support the hypothesis of deviant language laterализation in at least one subtype of SLI. A number of functional neuroimaging studies, however, has targeted some of the underlying processes assumed to be compromised in dyslexia (see under ‘Etiology of dyslexia: Theories’, this chapter), and has attempted to locate brain regions reflecting atypical activation in dyslexic individuals. Here, I report PET and fMRI studies examining differences in phonological, auditory temporal, or magnocellular processing between dyslexic and non-impaired readers. For further review of functional neuroimaging research in dyslexia the reader is referred to Habib (2000), Pugh et al. (2000b), and Grigorenko (2001).

**Brain activation during phonological tasks.** One of the first functional imaging studies examining phonological processing (see under ‘Phonological processing deficit’, this chapter) in dyslexia was that of Rumsey et al. (1992). PET scans were obtained from 14 adult male dyslexics and 14 normally literate controls while performing two tasks: a phonemic awareness task in which participants were asked to press a button if two auditorily presented words rhymed with each other, and a non-phonologic attentional task in which they were required to push a key whenever a target tone in a series of (simple) tones was detected. In normal literates, the left temporo-parietal cortex (angular/supramarginal gyrus) was activated during rhyme judgement but not during tone detection. Dyslexic individuals showed reduced

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22 In the present thesis, only technologies measuring regional cerebral blood flow or metabolism were embraced by the term ‘functional neuroimaging’.
blood flow in the left temporo-parietal regions activated in controls while performing the phonological task but did not differ from controls in these regions during rest or attentional testing. Thus, dyslexic individuals demonstrated a left temporo-parietal dysfunction associated with phonological demands of the rhyming task.

A subsequent PET study (Paulesu et al., 1996) employed two visually presented phonological tasks: a rhyming task (Does the letter rhyme with B?) and a short-term memory task (Was K among the last 6 letters you saw?). In normally literate men (n = 5; all right-handed), both tasks activated a number of perisylvian structures of the left hemisphere including Broca’s area, Wernicke’s area, and the insula, whereas parietal operculum activation was specific to the phonological memory task. In dyslexic men (n = 5; all right-handed), only a subset of brain regions normally involved in phonological processing was activated: Broca’s area during rhyme judgement, left temporo-parietal cortex during short-term memory demands, but the insula of the left hemisphere never. Paulesu and co-workers thought the left insular cortex to be crucial to convert whole-word phonology (temporo-parietal regions) to segmented phonology (inferior-frontal regions). They speculated that phonological deficits in dyslexia may result from a weak connectivity between anterior and posterior language areas.

The study of Paulesu et al. (1996) support the findings of Rumsey et al. (1992) to the effect that they found reduced activity in the left temporo-parietal regions in dyslexic adults while performing simple rhyming tasks, but extended it in showing task-dependent activations of only a subset of left-hemispheric perisylvian-language areas. It deserves mention, however, that the two PET studies used different methodological approaches. While Rumsey et al. (1992) employed a region of interest method which is governed by preconceived anatomical considerations, Paulesu et al. (1996) exploited whole-brain scanning and voxel-based image analysis permitting more detailed investigation of brain areas.

Using whole-head PET scanning, Rumsey et al. (1997a) compared 17 right-handed dyslexic men and 14 non-impaired controls who performed two kinds of print tasks with stress on phonological or orthographic features. The first type of task, referred to as ‘pronunciation’ included (phonological) decoding of pseudowords (e.g., phalbap, chirl) and (orthographic) reading of low-frequency irregularly spelled words (e.g., pharaoh, choir). The second type of task involved lexical decision making with a phonological instruction (Which one sounds like a real word?; e.g., jope-joak) or an orthographic one (Which one is a real word?; e.g., thurd-third). In comparison to normally literate controls, males with dyslexia displayed reduced blood flow in temporal regions bilaterally and in inferior parietal cortex,
mainly on the left, during both pronunciation and lexical decision making. However, their activation of left inferior frontal cortex (Broca’s area) during both phonological- and orthographic-decision making did not differ from the control group. Thus, the Rumsey et al. (1997a) results contrast with the data of their earlier study (Rumsey et al., 1992) as well as with those of Paulesu et al. (1996). Rumsey et al. (1997a) comment on the absence of different activation loci in phonological versus orthographic tasks which might result, they suggest, from more basic deficits in phonemic awareness. They further hypothesized that the dyslexic group may have approached unknown irregular words in a manner closely resembling the letter-by-letter reading of unfamiliar pseudowords.

In line with the latter interpretation by Rumsey and associates, Shaywitz et al. (1998) designed a set of hierarchically organized print tasks, thought to make progressively greater demands on phonological analysis. The tasks required same-different judgements concerning: (i) line orientation (e.g., $V/V-V/V$), presumed to reflect visual-spatial processing; (ii) letter case (e.g., $BbBb-BbBb$), thought to predominantly explore orthographic processing; (iii) single-letter rhyme (e.g., $T-T$); as well as (iv) pseudoword rhyme (e.g., $leat-jete$), assumed to add increasingly more phonological processing demands; and (v) semantic category (e.g., $corn-rice$), believed to make demands on transcoding from print to phonology, but requires also activation of the mental lexicon to get the meaning of the word.

Brain activation patterns of 17 regions of interest per hemisphere were measured by means of fMRI in 29 right-handed dyslexic adults and 32 controls. On tasks making explicit demands on phonological processing (e.g., pseudoword rhyming), dyslexic individuals showed a relative underengagement of left posterior perisylvian and occipital sites (Wernicke’s area, the angular gyrus, and striate cortex), coupled with a disproportionately elevated response in a left anterior region (inferior frontal gyrus) than non-impaired readers. According to Shaywitz and colleagues, the findings suggest a functional disruption in those posterior cortical systems that are engaged by phonological decoding, and, possibly, a compensatory cortical reliance on Broca’s area.

In a further analysis of the Shaywitz et al. (1998) data, Pugh et al. (2000a) turned their attention to the functional connectivity of the angular gyrus. Their reasons were twofold: First, the (left) angular gyrus is considered pivotal in mapping visually presented inputs onto phonologic representations. Second, dyslexic males$^{23}$ have been reported to show a functional disconnection between the left angular gyrus and related posterior regions during reading (Horwitz et al., 1998). However, it could not be determined whether this disruption is specific

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$^{23}$ Horwitz et al. (1998) used data from the Rumsey et al. (1997a) PET study.
to phonological decoding engaged by reading tasks. The re-analyzed data by Pugh et al. (2000a) revealed significant correlations between angular gyrus and occipital and temporal-lobe sites on pseudoword rhyme and semantic category judgements in controls, but not in the dyslexic group. In the right hemisphere, corresponding correlations were significant for both reading groups. Thus, this pattern of results suggests a breakdown in left-hemisphere connectivity in reading, when substantial phonological decoding (or “phonological assembly”, Pugh et al., 2000a, p.51) was required, whereas right-hemisphere homologues seem to work in a compensatory manner for dyslexic readers.

Comparable evidence for an atypical left-hemispheric brain activation pattern thought to reflect a fundamental disruption of phonological processing for poor reading was offered from the PET study by Brunswick et al. (1999). The researchers compared six non-impaired adult readers with six readers having a childhood history of developmental dyslexia (all right-handed males) on word and pseudoword naming. Dyslexic individuals showed less activation in ventral occipito-temporal sites and greater engagement of left inferior frontal gyrus than non-impaired controls. Using identical sets of stimuli, the same laboratory tested verbal repetition in eight dyslexic men and six controls (McCrory et al., 2000). Here, the dyslexic group demonstrated less hemodynamic response compared with the control group in the right superior temporal and right post-central gyri. Since studies in healthy individuals indicate that attending to the phonetic structure of speech is associated with a decrease in right-hemisphere processing, McCrory and colleagues concluded that reduced right-hemisphere activation in the dyslexic group indicate an attentional bias towards phonetic elements of the auditory input. That is, less processing of non-phonetic aspects of speech may favor greater salience of the phonological structure of attended speech for dyslexic readers.\(^{24}\)

Regarding the findings of an atypical brain activation profile observed either in the left hemisphere (Brunswick et al., 1999) or the right (McCrory et al., 2000), the authors proposed that the neural manifestation of phonological disruption in dyslexia is task-specific, i.e., functional rather than structural in nature.

Taken together, during print tasks tapping phonological processing, dyslexic adults have shown usual or enhanced activity in left-hemisphere frontal-lobe language regions, but reduced or absent activity in left temporo-parietal language areas (Paulesu et al., 1996; Rumsey et al., 1997a; Shaywitz et al., 1998; Brunswick et al., 1999). Furthermore, the left

\(^{24}\) In the Brunswick et al. (1999) as well as McCrory et al. (2000) study, no group differences were observed between the word and pseudoword versions of the tasks. The authors pointed out that the pseudowords used in the studies were highly word-like (e.g., carrot vs. cappot) and that differences might emerge when the lexical credibility of the pseudowords is reduced.
angular gyrus has been found to be functionally disconnected from related temporal and occipital regions (Horwitz et al., 1998; Pugh et al., 2000a).

In order to clarify whether atypical cerebral responses in individuals with dyslexia reflect a fundamental deficit of phonological processing or rather a compensation for poor reading in adulthood, Temple et al. (2001) conducted an fMRI study in children. Whole-brain imaging data were acquired from 24 dyslexic and 15 normally reading children (8-12 years old) during phonological and orthographic tasks of rhyming and matching visually presented consonant letter pairs (e.g., *Do T and D rhyme?* and *Are P and P the same?*, respectively). During letter rhyming, activity in left frontal-lobe regions was evident in both groups with the dyslexic children displaying larger activation than their normally reading peers. Activity in left temporoparietal cortex associated with phonological processing was only observed in normally reading controls, however. During letter matching, the control group demonstrated activity throughout extrastriate cortex, whereas the dyslexic group showed reduced extrastriate occipital responses to orthographic processing. Thus, altered temporoparietal activation probed by rhyme letters in dyslexic children parallel prior findings in dyslexic adults pointing to their core phonological deficits. Moreover, childhood dyslexia may be characterized by impaired extrastriate activity thought to be important for orthographic processing.

To complete the overview of functional neuroimaging research on phonological processing in dyslexia, the second fMRI study (Georgiewa et al., 1999) conducted in young developmental dyslexics should be briefly mentioned here. Thirty-four (German) dyslexic and normally literate adolescents with an average age of 14 years (all right-handed) were scanned while silently performing several tasks: viewing of letter strings, reading of nonwords (e.g., *bnams*) and frequent words (e.g., *Blume*, engl.: *flower*), and phonological transformation (*Move the first letter to the end of the word and add the common German suffix ‘-ein’*; e.g., *Blume → lume-bein*). Dyslexic adolescents were found to show reduced activation in inferior frontal regions (in particular Broca’s area) and in left-hemisphere inferior temporal-lobe sites during tasks that invoke substantial grapheme-phoneme conversions and phonological awareness (i.e., nonword reading and phonological transformation). Neither group displayed temporoparietal activity, however. These results differed from the brain activation pattern observed in dyslexic children (Temple et al., 2001) as well as the previous cited adult findings. As suggested by Temple and associates (2001), task differences (covert behavioral response in the adolescent study versus overt response in the other studies) and the variation in image analysis procedures (in adolescents only a limited number of brain regions were
imaged) might have played a significant role in accounting for the deviant finding by Georgiewa et al. (1999).

**Brain activation during auditory temporal processing tasks.** To date, there are two functional neuroimaging studies reporting on auditory temporal processing (see under ‘Auditory temporal processing deficit’, this chapter) in dyslexia (Rumsey et al., 1994; Temple et al., 2000). Rumsey et al. (1994), employing PET contrasted brain activation in 15 right-handed dyslexic men and 18 normal readers during performance of a tonal matching task. The task demanded the participants to press a button if tonal sequences (3-4 tones) in a pair were identical. During tonal matching, dyslexic and normally reading adults displayed similar left-hemisphere temporal activation, but the dyslexic group exhibited reduced blood flow in right fronto-temporal regions. Along with this physiological difference, the dyslexic group was significantly impaired in performing the task. Since the task involved fast-paced stimulus presentation (16 tonal pairs/min), the authors considered the finding of impaired right-hemisphere activation as being consonant with hypothesized deficits in rapid temporal processing in dyslexia. Concerning the fact that many subjects had participated in the Rumsey et al. (1992) PET study of phonological processing (see above), Rumsey and colleagues proposed that dyslexic individuals may have more widespread deficits encompassing left- as well as right-hemisphere temporal cortex.

In a recent study employing fMRI, Temple et al. (2000) have examined whether adults with dyslexia exhibit deviances in the neural response to rapidly changing acoustic information. Stimuli employed were non-speech analogues of consonant-vowel-consonant syllables with either brief (= rapid) or temporally extended (= slow) acoustic transitions. In each stimulus condition, subjects were asked to press a key for high-pitched but not for low-pitched sounds. While normal readers displayed increased activity in the left prefrontal cortex in response to rapid relative to slow non-speech analogues, dyslexic individuals showed no differential left-frontal activity for the two stimulus types. Furthermore, differential left-frontal responsiveness was inversely correlated with rapid auditory processing performance (i.e., the lower the threshold ISI needed for sequencing three 20-ms tones, the greater the difference between activity for rapid compared with slow stimuli). According to Temple and colleagues, these results point to the role of left prefrontal regions as normally mediating rapid auditory processing and being disrupted in dyslexic readers. In supporting their conclusion, the researchers refer to the PET study of Belin et al. (1998) in right-handed

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25 It should be noted that the PET study by Rumsey et al. (1994) was not explicitly designed to test auditory temporal processing.
healthy men. Using comparable non-verbal stimuli, they found left prefrontal activation for rapid changing stimuli relative to a resting baseline, but no activation for slow stimuli relative to rest.

The two studies of Rumsey et al. (1994) and Temple et al. (2000) provide contrasting results. However, differences in imaging technologies, stimulus materials, tasks, and brain regions of interests between the studies as well as the lack of further research aggravate concluding remarks on neuronal responses to auditory temporal processing in dyslexia.

**Brain activation during visual motion perception.** Three published fMRI articles presented evidence for a selective deficit in the magnocellular system (see under ‘Magnocellular deficit’, this chapter) in adults with dyslexia (Eden et al., 1996; Demb et al., 1997, 1998). To contrast cerebral activation in six right-handed dyslexic males and eight normally reading controls, Eden et al. (1996) measured local blood-oxygenation level-dependent (BOLD) contrast signals, while the participants passively viewed either a coherently moving random-dot stimulus (magnocellular stimulus) or a stationary pattern (parvocellular stimulus). Moving stimuli are supposed to elicit strong responses in an area labeled V5 (MT) which is located in an extrastriate region at the junction of occipital and temporal lobes (Zeki, 1993). While in normal readers, the magnocellular stimulus activate V5/MT bilaterally, viewing moving dots failed to activate this area in dyslexic readers. In contrast, both groups demonstrated the expected activation of posterior occipital cortex (V1/V2) and extrastriate regions (inferior temporal/fusiform gyrus) during the presentation of parvocellular stimuli. According to Eden and colleagues, these data provide a neurophysiological basis of reported visual perception deficits in dyslexia that have implicated magnocellular dysfunction.

Demb et al. (1997, 1998) measured BOLD signals in response to low-luminance moving gratings (magnocellular stimuli) as opposed to control stimuli “designed to stimulate multiple pathways” (Demb et al., 1997, p.13363). Dyslexic individuals (n = 5; all right-handed) showed reduced activity relative to normal readers both in primary visual cortex (V1) and several extrastriate regions (inter alia MT+) in response to moving gratings of various contrasts. Moreover, there was a significant three-way correlation between brain activity, perceptual thresholds, and reading rate in both dyslexic and normal readers. Thus, participants exhibiting stronger V1 and MT+ activity demonstrated better moving discrimination performance and tended to be faster readers.

In summary, the fMRI results obtained in the visual system in dyslexia point to a functional deficit in the magnocellular pathway. However, as for the previous imaging
findings on auditory and phonological processing, brain areas showing significantly atypical responses vary between the studies. Thus, discrepancies might be attributable to differences in the subject populations, the stimuli, or the procedures used for localizing visual brain areas. While the phonological hypothesis of dyslexia has received valuable support from recent PET and fMRI research, hemodynamic neuroimaging studies on both magnocellular and auditory temporal processing are limited. Neurologic investigations of visual and auditory processing in dyslexia have been carried out primarily by means of electroencephalography. Targeting auditory processing as of most interest for the issue of the present thesis, this topic is considered next.

**Electrophysiological studies**

Electrophysiological recording techniques namely electroencephalography (EEG) and magnetoencephalography (MEG) excel in examining brain processes with high temporal resolution. Event-related potentials (ERPs) of the EEG elicited by various verbal and non-verbal stimuli have been analyzed in numerous studies of language-based learning impairments. For a review of auditory ERPs in dyslexia and SLI the reader is referred to Leppänen and Lyytinen (1997). A brief survey of ERPs to visually and auditorily presented stimuli in these populations is provided by Habib (2000). For more recent studies on visual ERPs in the framework of the magnocellular deficit theory of dyslexia the reader is referred to Kubová et al. (1995), Johannes et al. (1996), and Schulte-Körne et al. (1999b).

Among the limited number of MEG studies in language-based learning impairments, auditory event-related field (ERF) investigations constitute the minority. Instead, MEG research in dyslexia has been primarily concerned with magnetic source imaging during performance of various reading tasks (Salmelin et al., 1996; Helenius et al., 1999b,c; for a review see Salmelin et al., 2000; Simos et al., 2000a,b). Here, neuronal source activity within a predefined time window of visual ERPs was determined and (if available) projected onto structural brain images. In general, these MEG studies support the findings of hemodynamic deviances in language-related brain sites in dyslexia (see under ‘Functional neuroimaging’, this chapter). For instance, Salmelin et al. (1996) found that print processing was associated with enhanced source activity in the left-hemisphere inferior temporo-occipital border at about 180 ms after word onset in normally reading controls but not in dyslexic adults. Also a subsequent activation of the left temporal region between 200 and 400 ms was only evident in normal readers; instead dyslexic participants, but not controls, showed neuronal responses in the left inferior frontal cortex (approximately in Broca’s area) in this same time interval.
Reminiscent of the fMRI findings in children and adults with dyslexia (Shaywitz et al., 1998; Brunswick et al., 1999; Temple et al., 2001), this response pattern was suggested to reflect posterior cortical anomaly and compensatory reliance on frontal-lobe systems. Atypical source activity in posterior brain sites was also detected in children with dyslexia during engagement in printed pseudoword rhyme-matching and word-recognition tasks (Simos et al., 2000a,b). Dyslexic children displayed reduced activity in left temporo-parietal cortex between 300 and 1200 ms post-stimulus onset, coupled with a high density of source clusters in homologous right-hemisphere regions as compared to normally reading children. Using an auditory version of the word-recognition task, Simos and colleagues (2000b) found no group differences in the number of activity sources in temporo-parietal regions. It deserves mention, however, that the auditory task was only run “to rule out the hypothesis that hypoactivation of left temporo-parietal areas in dyslexics was due to a more general cerebral dysfunction in these areas” (Simos et al., 2000b, p.810). Thus from this study, it is not possible to draw conclusions about deviances in neuronal source activity associated with auditory event-related processing in dyslexia.

In the following sections, auditory ERP and ERF studies of language-based learning impairments are reviewed. Because of the wealth of ERP components examined in these populations, the survey is limited to the N100 and mismatch negativity (MMN). Both the N100 and MMN have been intensively studied in basic research as well as in clinical populations. Moreover, their magnetic counterparts, termed N100m and mismatch field (MMF), have also been addressed in two auditory ERF papers including reading-impaired individuals.

**Auditory event-related potentials**

**The N100.** The N100 (or N1) is the most prominent peak of auditory ERPs elicited by simple repetitive stimuli such as tones or syllables. Differences in latency or amplitude of the auditory N100 have been reported in children with reading difficulties (Pinkerton et al., 1989; Neville et al., 1993; Brunswick & Rippon, 1994) as well as in children with language impairments (Dawson et al., 1989; Neville et al., 1993; Lincoln et al., 1995; Tonnquist-Uhlén et al., 1996). Amplitude reduction of the N100 was found in a group of 14 boys with difficulties in reading, writing, and spelling (designated ‘poor readers’) as compared to 18 ‘good readers’ (all 8-9 years old) in a study by Pinkerton et al. (1989). Cortical auditory ERPs were recorded in response to 2000-Hz tone bursts while participants watched silent films. Reduced N100 amplitudes (around 160 ms) in poor readers were observed at three of four
scalp locations. For the whole sample, N100 amplitude was correlated positively with performance IQ, spelling scores, reading accuracy and comprehension, as well as arithmetics. In interpreting the data, Pinkerton and colleagues suggested that the decreased N100 magnitude could be associated with impaired processes mediating selective attention.

Brunswick and Rippon (1994) contrasted 15 dyslexic boys (7-11 years old) and 15 normally reading controls (8-10 years old) on ERPs to stop consonant-vowel syllables presented in a dichotic listening paradigm. The participants were asked to report simultaneously presented syllables as accurately as possible. No significant group differences were observed either in the right or in the left ear responses. However, normally reading children exhibited larger N100 amplitudes at left temporal-electrode sites than the dyslexic children who showed less lateralized temporal N100 magnitude. N100 lateralization was also found to be positively related to performance on a phonological awareness task, viz., rhyme oddity detection among words differed in their last sounds (e.g., pin, win, sit, fin; see the Bradley and Bryant task under ‘Phonological processing deficit’, this chapter). According to Brunswick and Rippon, the deviances in N100 laterality are associated with abnormal cerebral lateralization of language functions in dyslexia. The failure of the dichotic listening task to discriminate between dyslexic and normal readers in spite of the N100 laterality differences was suggested to indicate that laterality does not affect processing of the stimuli per se but appears to be associated with later aspects of phoneme analysis. However, in view of the fact that the N100 has been considered a basic index of adequate sensory registration, Leppänen and Lyytinen (1997) proposed that an altered N100 response might reflect inaccurate tuning of sensory information resulting in less reliable auditory representations, that are, in turn manifested in poor performance on language tests.

Yingling et al. (1986), on the other hand, did not find any differences between 38 severely dyslexic boys (mean age 13.3 years) and 38 non-impaired peers in ERPs following stimulation with auditory clicks. Bernal et al. (2000) observed no deviations of the N100 to pure tones in a group of 20 poor readers (10-12 years old), but reported larger amplitudes in two later components, the N200 and the P200 as compared to 20 normally reading children.26

In a recent study, Molfese (2000) presented evidence that auditory ERPs recorded within 36 h of birth discriminated between newborns who 8 years later would be classified as dyslexic, poor, or normal readers. The auditory ERPs analyzed by Molfese included the N1-

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26 Unfortunately, Pinkerton et al. (1989), Brunswick and Rippon (1994), and Yingling et al. (1986) did not present any figures depicting grand-average or individual subject ERPs.
P2-N2 waves elicited by speech and non-speech syllables with mean peak latencies of 174, 309, and 458 ms, respectively. The left-hemisphere N1 latency at birth was found to be shortest for the normally reading children and longest for the poor readers. Neither the dyslexic nor the poor readers displayed a well-defined N1 component. Right-hemisphere N2 peak amplitudes were largest for the dyslexic children and smallest for the poor readers. In particular the group differences in the N1 latency might point, as suggested by Molfese, to an underlying perceptual mechanism upon which some aspects of later developing verbal and cognitive processes are based.

As for SLI, Dawson et al. (1989) reported atypical hemispheric asymmetry of N100 in response to a simple speech stimulus. In this study, 10 children with SLI ranging from 6 to 15 years were compared to 10 children with autism and 10 language-normal controls (aged 8-13 years). Children were presented with series of auditory stimuli involving 80% clicks, 10% syllables (viz., /da/), and 10% piano chord stimuli and asked to indicate whenever the /da/ stimulus occurred. Based on the analysis of right-hemisphere-minus-left-hemisphere scores, both the SLI and autistic group showed the reversed pattern of N100 asymmetry that characterized the controls (i.e., smaller left-than-right amplitude and shorter left-than-right latency). Furthermore, in children with autism, language abilities were associated with right-hemisphere activity measures: while performance on three out of six verbal tests correlated positively with the N100 latency, correctness on all six tests were negatively related to the N100 amplitude. In children with SLI, on the other hand, impaired performance on four of the language measures was associated with longer left-hemisphere N100 latency. No statistical relationship between N100 and language measures was obtained for the control children. According to Dawson and colleagues, the pattern of hemispheric activity found in children with SLI coincides with a deficit in processing sequential information, for which the left hemisphere is thought to be pivotal.

Lincoln et al. (1995) studied children with SLI, autism, or normal language skills (10 in each group; aged 8-14 years) running two experiments. The first experiment involved passive listening to series of pure tones which differed in frequency and intensity (1000 Hz/60dB vs. 70 dB and 3000 Hz/63 dB vs.73 dB, all with equal probability). No group

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27 It should be noted that the equivalents of the adult components are unclear (Čeponienė et al., 1998). For example, the N1/N100 component is not consistently present until age 9, although it can be seen more readily when stimulus presentation rate is reduced or multi-channel recording techniques are implemented (Bruneau et al., 1997; Čeponienė et al., 1998; Ponton et al., 2000).

28 Autism is a pervasive developmental disorder that usually appears during the first three years of life. The disorder is characterized by qualitative impairments in social and communicative skills, with restricted and repetitive activities and interests (DSM-IV; 299.00). Autistic individuals typically have problems processing auditory information (see e.g., Courchesne, 1987).
differences in latency or amplitude of the auditory N100 were obtained. However, unlike the children with autism or SLI, the control participants did show an increase in N100 amplitude to increases in stimulus intensity. In the second experiment, two pure tones were presented with the following variation on the so-called oddball paradigm under two different conditions. The active or response condition required the child to press one button to each frequent tone (probability = 70%) and another button to each infrequent tone (probability = 30%). In the passive or no-response condition, children simply listened to the stimuli. Both in the active and passive condition, N100 amplitude was found to be generally larger in SLI children compared to control subjects (and nearly significant larger compared to autistic children). The N100 latencies were similar in autistic and control children, but differed from the SLI group. Lincoln and colleagues concluded that the N100 deviances are consistent with theories of SLI related to ineffective regulation of sensory input. They speculated that the brief (50-ms) duration of the tones employed in their study may have been too short for full processing without SLI children having to allocate further attentional resources as mirrored by the enhanced N100. Alternatively, the increased N100 peak has been suggested to be associated with impaired encoding of auditory information in short-term memory. Thus, for some of the SLI children the 2-second ISI might be too long to maintain the internal reference indicating whether the tone designated the frequent or infrequent stimulus.

Neville et al. (1993) reported N100 deviances in a subset of SLI children who exhibit deficits in auditory temporal processing. Twenty-two SLI children with concomitant reading disability (RD) and 12 controls who evidenced normal language development and academic achievement (all 8-10 years old) were compared on auditory and visual ERPs. The auditory paradigm involved an active oddball task in which a 1000-Hz tone was presented as the target stimulus (10% probability) among 2000-Hz standard stimuli at one of three ISIs (200, 1000, and 2000 ms) and at one of three different stimulus positions (left ear, both ears, and right ear). Since no group differences were obtained for the auditory ERPs to either stimulus, the SLI/RD children were subclassified into two subgroups according to their performance on an auditory rapid sequencing test (see under ‘Auditory temporal processing deficit’, this chapter). SLI/RD children performing below the median level were classified as ‘low repetition’ (i.e., displaying auditory temporal processing problems) while those scoring above were classified as ‘high repetition’. Following that, the N140 component to standard tones was found to be significantly diminished over the right hemisphere at the shortest ISI in the low-repetition group compared to both the language-normal controls and the high-repetition

29 Subgroup sizes are not specified in the article.
SLI/RD group. In addition, the latency of the standard N140 was significantly delayed in the low-repetition SLI/RD group especially over temporal and parietal sites of the left hemisphere. Neville and co-workers considered the N140 component equivalent to the adult N100. A contralateral (to the stimulated ear) and anterior distribution of the N140 response suggests to them that it reflects activity generated in the superior temporal gyrus encompassing primary and secondary auditory areas. Hence, these findings were assumed to indicate that in SLI/RD children with auditory temporal processing problems, the reduced and slowed activity within these cortical sites contributed to their language symptoms. The authors’ interpretation is not to be taken as a single-factor account of the deficits of language- and reading-impaired children, however. Thus, various deviations on visual ERPs to both language and non-language stimuli were also reported for either the whole SLI/RD group or only a subset of it.

Finally, Tonnquist-Uhlén et al. (1996; see also Tonnquist-Uhlén, 1996) observed significantly delayed N100 latency and a tendency towards a higher incidence of unusual topographic maps in 20 children with severe SLI (9-15 years old) when contrasted to an age-matched control group (n = 20). Using a passive listening paradigm, pure-tone stimuli of 500 Hz were delivered to the left and right ear separately. The peak latency of the vertex-recorded N100 was longer in the SLI children (on average 110 ms) than in the healthy controls (on average 100 ms) following right-ear stimulation. Both left-ear and right-ear elicited N100 responses tended to decline with increasing age in the control children but not in the SLI group. While the delayed N100 latencies were presumed to be due to slower processing in central auditory pathways, the lack of an age-related latency decrease was considered to indicate that the disturbance persists rather than reflects a pure maturational delay. Furthermore, the SLI children showed a trend towards a greater number of deviating or non-focal topographical maps after left-ear stimulation. According to Tonnquist-Uhlén et al. (1996), atypical N100 topography may be accounted for by a lack of synchronization, that is due to immature or poor connections between different cortical areas and deeper structures.

Taken together, the auditory ERP studies cited above indicate differences in N100 features between groups of children designated SLI, dyslexia, or poor readers and healthy controls. While latency deviations in language-based learning impairments may be associated with a common timing deficit, N100 amplitude differences have been related to attentional factors or inadequate sensory processing. Great individual subject variability coupled with recording techniques using only a limited number of electrodes have not seldom led to negative results or only non-significant trends, however. Furthermore, variations in stimulus
materials (e.g., clicks, tones, or syllables), task paradigms (e.g., response/active vs. no-response/passive task, oddball paradigm vs. repetitive unchanging stimuli), and interstimulus intervals (for this issue see e.g., Čepioniene et al., 1998) across the ERP studies aggravate a comparison of the findings. In the next section, ERP studies of dyslexia and SLI analyzing the MMN will be presented. These have at least in common that they employed the same paradigm because the MMN is elicited by a passive or ‘unattended’ oddball task. 

The MMN. The MMN is a fronto-centrally negative component of the auditory ERP, usually peaking between 100 and 250 ms post-stimulus onset. It is thought to reflect a pre-attentive neuronal change-detection mechanism, occurring when an infrequent physically ‘deviant’ sound encounters a well-established sensory memory trace of a frequently presented ‘standard’ sound (e.g., Näätänen, 2001). The MMN has proven to be a suitable tool for studying auditory discrimination in both adults and children (for a review see e.g., Cheour et al., 2000; Kraus & Cheour, 2000; Näätänen, 2001). Unlike the N100 which shows morphological changes through the second decade (e.g., Courchesne, 1990; Ponton et al., 2000), the MMN is relatively mature at the age of 6 (Csépe, 1995; Kraus et al., 1999). Response of this kind was reported in neonates (e.g., Cheour-Luhtanen et al., 1995; Cheour et al., 1999) and even in pre-term infants (Cheour-Luhtanen et al., 1996). The MMN has been demonstrated as a sensitive measure for distinguishing individuals with language-based learning impairments from healthy peers (e.g., Korpilahti & Lang, 1994; Kraus et al., 1996; Holopainen et al., 1997, 1998; Schulte-Körne et al., 1998a, 1999a, 2001a; Baldeweg et al., 1999; Bradlow et al., 1999; Kujala et al., 2000; for a review see Kujala & Näätänen, 2001).

Kraus and colleagues were among the first to investigate auditory phoneme processing in children with language-based learning disorders using MMN. Kraus et al. (1996) sought to determine whether deficits in perception of rapid spectro-temporal changes experienced by children with learning problems derive from aberrant neuronal representations of acoustic events prior to conscious reception or from higher-level dysfunctions. Behavioral discrimination abilities and MMN responses to synthetic consonant syllables were evaluated in 6- to 15-year-old controls with normal academic performance (n = 90) and children exhibiting learning problems (n = 91). The learning-impaired children displayed a discrepancy between intellectual capacity and psychoeducational achievement. As a group, these children showed \textit{inter alia} problems on measures of listening comprehension, sound blending, reading, and spelling. First, both groups of children were asked to discriminate

\footnote{In order to control for attention, MMN paradigms frequently involve the presentation of silent films or cartoons.}
behaviorally along two continua of speech contrasts, /ba/-/wa/ and /da/-/ga/ with the former varying exclusively in the duration of the formant transition period (10–40 ms) and the latter in the spectral content with the transition duration held constant at 40 ms (see under ‘Auditory temporal processing deficit’, this chapter). Individuals with learning problems performed less well than the control children on both continua, and both groups had more difficulty in discriminating /da/-/ga/ than /ba/-/wa/. However, there was a greater difference between the learning-impaired group and the controls for /da/-/ga/ discrimination than for /ba/-/wa/. Then, the MMN was evaluated in two subgroups of the children – one comprising 21 ‘good /da/-/ga/ perceivers’ and the other 21 ‘poor /da/-/ga/ perceivers’. A prominent MMN in response to just-perceptibly different variants of /da/ and /ga/ was evident in good /da/-/ga/ perceivers, but not in poor perceivers. Correlational analyses performed for all the 42 children revealed moderate but significant relationships between behavioral /da/-/ga/ discrimination scores and both MMN duration and mean amplitude ($r = -0.40$ and $r = -0.42$, respectively). That is, accurate discrimination on /da/ versus /ga/ was associated with robust MMNs; limited performance, on the other hand, was related to diminished mismatch responses. In addition, both good and poor /da/-/ga/ perceivers were easily able to discriminate a /ba/-/wa/ contrast and, as was evaluated in 14 children of each subgroup, displayed a robust MMN to just-perceptibly different variants of /ba/ and /wa/. According to Kraus and colleagues, the findings indicate that the speech-sound discrimination deficits exhibited by children with learning problems probably have their origins in the auditory pathways and may be pre-attentive in nature. Moreover, the selective impairment in neuronal representation and behavioral discrimination of the /ba/-/wa/ syllables compared to the /da/-/ga/ stimuli, suggested to them that the two rapid spectro-temporal contrasts tap separate and distinct neuronal mechanisms which may be differentially vulnerable to disruption. Identification of disturbed mismatch responses may thus have implications for differential diagnosis and targeted intervention strategies for children with learning impairments and attentional disorders (Kraus et al., 1996).

In a subsequent study of the same research team, Bradlow et al. (1999) aimed at investigating the precise acoustic-phonetic features that pose perceptual difficulties for some children with learning problems. Seventy-two controls with normal academic achievement and 32 learning-impaired children ranging in age from 6 to 16 years participated here. The learning-impaired group demonstrated similar difficulties as described in the paper of Kraus et al. (1996). Behavioral discrimination performance on two synthesized /da/-/ga/ continua with either short (40 ms) or extended (80 ms) formant transitions was compared; MMN
responses to just-perceptibly different syllables of each continuum were evaluated. Consistent with previous findings (Kraus et al., 1996), children with learning problems displayed smaller MMNs relative to their non-impaired age-mates to the /da/-/ga/ pair when the formant transition duration was short. In the learning-impaired group, larger mismatch activity to temporally extended compared to short transitional syllables were found, although discrimination performance remained significantly impaired irrespective of formant transition length. In accord with their performance levels, normally learning children showed similar MMN responses to both short- and lengthened-transition /da/-/ga/ pairs. While extending the formant transition duration did not improve behavioral discrimination, the MMN data were thought to indicate that, at a pre-attentive neural level, the long-transition syllables were represented more accurately than the short-transition stimuli in children with learning problems. In further interpreting the results, Bradlow et al. (1999) refer to the speech training program by Merzenich and Tallal (e.g., Merzenich et al., 1996; Tallal et al., 1996) in which formant transitions of consonant stimuli had been lengthened in time so as to make them more intelligible during training (see under ‘Auditory temporal processing deficit’, this chapter). The authors suggested that the better neural representation of the longer duration syllables may underlie the success of acoustically modified speech training, whereas short-transition stimuli – which are poorly encoded – may be difficult for children to access for learning purposes.

Risk for language-based learning impairments also has been studied exploiting the MMN paradigm in infants. Leppänen and Lyytinen (1997) compared 6-month-old infants born into families with a history of dyslexia (n = 18) and control babies with no such background (n = 17). ERP differences were found in response to the (Finnish) nonsense word /atta/ while the standard was the shorter-duration nonsense word /ata/: infants with a positive family history showed a smaller MMN-like response over the left, but not right, hemisphere than the control group. This longitudinal study is still in progress and it will be interesting to see whether the attenuated MMN-like component signals an elevated risk of developmental dyslexia.

Schulte-Körne and co-workers (1998a, 1999a, 2001a) have used the MMN to address the question of whether the perceptual deficits in people with dyslexia are of a general

31 This behavioral result diverges from the findings of Tallal and Piercy (1975) who reported enhanced perception in children with SLI of synthetic stop-consonant syllables (viz., /ba/-/da/) when the formant transition duration was extended from 43 to 95 ms (see under ‘Auditory temporal processing deficit’, this chapter). According to Bradlow et al. (1999), differences in subject, task, and stimulus characteristics may account for the conflicting results.

32 This term was chosen as Leppänen and Lyytinen (1997) pointed out that only a proportion of the ERP waveform in the study could be interpreted to reflect the MMN.
auditory or speech specific nature (see under ‘Etiology of dyslexia: Theories’, this chapter). In the Schulte-Körne et al. (1998a) study, 19 dyslexic boys and 15 normal spellers (mean age 12.6 years) were presented with either synthetic stop-consonant syllables (standard /da/ vs. deviant /ba/) or pure tones (standard 1000 Hz vs. deviant 1050 Hz). While there were no group differences to the frequency change in tones, the dyslexic children showed a significantly reduced MMN amplitude to the change in syllables. Consequently, the results were assumed to point to a specific deficit at a pre-attentive sensory level. Comparable results were obtained when contrasting 12 dyslexic adults and 13 controls with normal spelling skills on stimulus series encompassing either the synthetic stop-consonant syllables /da/ and /ga/ or the 2200-Hz and 2640-Hz sinusoidal tones (Schulte-Körne et al., 2001a). MMN responses differed between the two adult groups only in the syllable condition. The researchers conclude that speech perception at a pre-conscious stage constitutes one major player in dyslexia not only in children but also in adults. However, as conceded by Schulte-Körne et al. (1999a, 2001a) the findings leave open the question whether the group differences in the speech condition were due to the temporal information embedded in consonant stimuli.

Pursuing this issue further, Schulte-Körne et al. (1999a) evaluated the mismatch activity to rapidly changing tone-burst patterns in 15 dyslexic adults and 20 normal spellers. The tone-burst patterns consisted of four frequency segments, 720-815-1040-815 Hz. In the standard pattern, the duration of the single frequencies was 50-90-25-50 ms, respectively. In the deviant pattern, the two segments of identical frequency (viz., 815 Hz) had been exchanged resulting in the duration sequence 50-50-25-90 ms. Dyslexic adults were observed to show an attenuated MMN relative to non-impaired controls. Schulte-Körne et al. (1999a) concluded that impaired neural discrimination of temporal, rather than phonetic, information may be pivotal for the findings of reduced MMN to stop-consonant syllables in dyslexia.

One of the most informative studies in testing the major competing etiology hypotheses in dyslexia – a linguistic versus a more general processing deficit – is provided by Kujala et al. (2000). Both behavioral and MMN responses to tone-pattern contrasts and tone-pair changes were obtained in eight dyslexic adults and eight normal readers. The tone patterns consisted of four 500-Hz tones with silent intertone intervals of either 200, 150, and 50 ms (standard pattern) or 200, 50, and 150 ms (deviant pattern). That is, in the standard pattern, the third tone was close to the fourth, and in the deviant pattern closer to the second tone. The tone pairs were composed of two 500-Hz segments separated by either 150 ms (standard pair) or 50 ms (deviant pair). These intervals matched those between the two middle segments of the standard and deviant tone pattern, respectively. Kujala and colleagues found
no group differences in the MMN amplitude to the temporal change in the tone-pair condition. In the tone-pattern condition, two consecutive MMNs were elicited in the normally reading controls, whereas dyslexic readers showed only the second MMN to the shortening of the intertone interval. The occurrence of a biphasic MMN suggested to the researchers that the auditory system in controls reacted to the two deviations in the pattern, a too-early tone, which first corresponds to an addition and then to an omission of a tone. The auditory system of the dyslexic readers, on the other hand, might discriminate the second, but not the first change. In agreement with the MMN data, behavioral discrimination performance was found to be normal in the tone-pair task but impaired in the tone-pattern task. Kujala and colleagues concluded that dyslexic adults have problems in processing temporal information only when surrounded by other sounds, as is customary in the linguistic domain (cf., phonemes in words). The behavioral and MMN findings were thought to be consistent with a basic auditory dysfunction in dyslexia.

Other MMN studies have found a selective deficit in frequency discrimination both in adults with dyslexia (Baldeweg et al., 1999) and children with SLI (Korpilahti & Lang, 1994). Baldeweg et al. (1999) contrasted 10 dyslexic adults and 10 normal readers on their MMN responses to either frequency changes or duration changes in pure tones. The frequency condition included a 1000-Hz standard tone and four deviants of 1015, 1030, 1060, and 1090 Hz with a constant stimulus duration of 50 ms. The duration condition involved 1000-Hz tones with the standard being 200 ms long, and the four deviants being 160, 120, 80, or 40 ms in length. Mismatch activity to duration decrement did not differ between the two participant groups. In the frequency condition, however, dyslexic adults showed delayed and reduced MMN potentials relative to normal readers. This neuronal dysfunction was mirrored in a similarly specific behavioral impairment in discriminating tone frequency, but not tone duration. Furthermore, the frequency-discrimination deficit and MMN delay correlated with the degree of impairment in phonological skills, as reflected in reading errors of regular words and pseudowords. Although the authors pointed out that the study was not designed to investigate the ability to process rapidly presented auditory stimuli, some physical features of auditory events (viz., frequency) were assumed to add more than others (viz., duration) to the temporal processing deficit observed in some dyslexic individuals.

Similar results were reported by Korpilahti and Lang (1994) in 8- to 13-year-old SLI children (n = 14) when compared to 12 controls with normal language skills. In the SLI group, the peak amplitude of frequency-change MMN (500 vs. 533 Hz) was found to be significantly smaller than in the control group. The duration-change MMN (50 vs. 110 ms or
50 vs. 500 ms), however, showed a significant group difference only for sinusoidal stimuli with highly-contrasting values (50 vs. 500 ms). In the control subjects, unlike in the children with SLI, the latency of the frequency MMN was negatively correlated with age. MMN lateralization to the right hemisphere was observed in the language-normal children, especially for the frequency stimuli, whereas, in the SLI children the MMN was lateralized to the left hemisphere. According to the authors, two possible hypotheses may account for the attenuated MMN in the frequency-change condition: (i) abnormally quick fading of the sensory memory trace or (ii) decreased attention to auditory events. The latter assumption seems somewhat surprisingly, since neither a sign of an attention-switching mechanism was evident in the ERP waveform (P300) nor an active oddball paradigm was employed (see Leppänen & Lyytinen, 1997).

The results concerning the frequency MMN amplitude were replicated in younger children with SLI (3-6 years) by Holopainen et al. (1997). In a subsequent study, Holopainen et al. (1998) found an attenuation of the MMN amplitude for frequency change not only in children with SLI (aged 5-9 years), but also in mentally retarded children with delayed development of speech and language (aged 5-8 years). Here, attenuated frequency MMN was related to the impairment of linguistic skills irrespective of the child’s cognitive level (Holopainen et al., 1998). However, MMN to duration changes was not investigated in either study.

The survey of MMN studies indicates that the mismatch response provides a powerful method for studying auditory discrimination and memory in children and adults with language-based learning disorders. Some of the experiments were designed to test whether the neuronal mismatch pattern favors a general auditory dysfunction hypothesis or one of a linguistic processing deficit. Other MMN studies sought to determine those precise acoustic features which provoke the perceptual difficulties experienced by some individuals with language-based learning disabilities. Taken together, the findings suggest deviances in the accuracy of the neuronal representation of speech and non-speech sounds as well as of certain auditory features in the language learning-impaired population.

Auditory event-related fields

Only two peer-reviewed articles have studied auditory ERFs in individuals with language-based learning problems so far (Nagarajan et al., 1999; Heim et al., 2000c). Both studies recorded magnetic responses from the left-hemisphere auditory cortex. Nagarajan et al. (1999) compared seven adult poor readers and seven normally reading controls on tone-
sequence perception. Sequences of two brief (20-ms duration) sinusoidal tones (high-high, high-low, low-high, or low-low) were presented at each of three different ISIs (100, 200, and 500 ms), and participants were asked to press two buttons in the correct order. The ERF response amplitudes over a 150-200-ms post-stimulus time range were found to be generally stronger in poor readers than in normal readers. Furthermore, $N_{100m}$ amplitudes for the second stimulus of a sequence were weaker in poor readers than in control participants, for ISIs of 100 or 200 ms, but not for the ISI of 500 ms. This neuronal deviance was corroborated by a similar performance profile on tasks measuring perceptual interference between rapidly successive stimuli. The findings were interpreted as further evidence that “most” (Nagarajan et al., 1999, p.6487) reading-impaired individuals exhibit an enduring dysfunction in the cortical integration of brief and rapidly successive sound inputs.\footnote{Another finding which was thought to indicate different cortical response dynamics to brief and rapidly successive stimuli was observed in MEG spectra. The average distributed response coherence was weaker in the $\beta$- and $\gamma$-band frequency ranges (20-60 Hz) for shorter, but not for longer, ISIs in poor readers compared with normally reading controls (Nagarajan et al., 1999).}

A study by Heim et al. (2000c) – which will be presented in detail in Chapter II of this thesis – included 10 children with dyslexia and 9 normally literate controls (aged 8-14 years). Using a passive oddball paradigm, the children were stimulated with frequency changes (1000-Hz standard vs. 1200-Hz deviant) or naturally produced stop consonant-syllable contrasts ([da] standard vs. [ga] deviant and \textit{vice versa}). Contrary to the results obtained in electric MMN studies (see above), the magnitude of the MMF did not differ significantly between the participant groups either in the frequency- or in the syllable-change condition. However, the sources of the magnetic waves in response to both speech and non-speech (standard) events 210 ms after stimulus onset were found to be located about 1.5 cm more anterior in children with dyslexia than in controls. The magnetic response at 210 ms was assumed to be equivalent to the adult $N_{100m}$, which has recently been located to the planum temporale (Lütkenhöner & Steinsträter, 1998, Ohtomo et al., 1998). Heim and colleagues speculated that the source configuration of the 210-ms responses might index activity within the planum temporale in the control children, whereas in the dyslexic children sources might be tied to sites anterior to the planum.

To summarize, these MEG studies indicate deviant cortical auditory processing in poorly reading adults and children with dyslexia. Nevertheless, in order to draw firm conclusions further research is needed.
AUDITORY PROCESSING IN DYSLEXIA: THE AIMS OF THE PRESENT THESIS

Psychoacoustic studies have shown that many children with dyslexia or SLI are at least mildly impaired in their ability to detect stop consonants, such as /b/, /d/, /g/, and /k/ (for reviews see Tallal et al., 1993; Farmer & Klein, 1995; Bishop, 1997). While Tallal and colleagues have proposed that the deficit in stop-consonant perception results from impaired integration of brief and rapidly changing sounds (for reviews see Tallal et al., 1993; Farmer & Klein, 1995), other researchers have ascribed the specific difficulty to the stop consonants’ spectral similarity (e.g., Mody et al., 1997; for a review see Studdert-Kennedy & Mody, 1995). Whatever the cause, proponents of both hypotheses have associated impaired consonant perception in dyslexia and SLI with distorted or ‘noisy’ phonological representations of speech sounds. Indeed, findings in the field of auditory ERP research provide evidence for inaccurate cortical neuronal representation of speech syllables in individuals with language-based learning impairments (see under ‘Electrophysiological studies’, this chapter).

Comparable MEG studies evaluating auditory ERFs and its underlying sources in these populations are quite rare, however. The MEG is a very appropriate technique to study auditory processing and cortical organization, especially in developmental disorders such as dyslexia or SLI. The reasons are manifold: (i) Neuromagnetic fields are generated by intracellular currents in pyramidal neurons of fissural cortex flowing tangentially to the scalp. Hence, the supratemporal auditory cortex, located as it is in the sylvian fissure, is well-positioned for MEG recordings. (ii) The high temporal resolution of MEG is capable of reflecting central auditory processing with an accuracy of the order of milliseconds. Thus, MEG is a good technique to address ideas about auditory processing deficits in dyslexia or SLI as suggested by Tallal. (iii) At instances in time when there is only one focal source of activation per hemisphere, MEG allows the determination of the source location with an accuracy of a few millimeters. Under such circumstances, this is not only superior to EEG-based source analysis but also affords a higher spatial resolution than hemodynamic techniques such as PET or SPECT. (iv) MEG recordings are non-invasive and free of bodily contact, i.e., require no electrode placement on the scalp as required for the EEG. Therefore, MEG is a procedure well-suited for children, especially those with language-based learning impairments, who often show stress from a prolonged history of medical and psychological testing. (For details on MEG and MEG-based methods the reader is referred to Elbert, 1998.)

The present thesis sought to characterize the cortical neurophysiology of auditory processing in children and adolescents with dyslexia by utilizing the benefits of MEG-based
methods. The focus will be on auditory ERFs and corresponding sources elicited by stop-
consonant syllables in a passive task paradigm. Findings of psychophysical experiments are
expected to complement magneto-cortical data and in so doing could bring valuable
information to light. The present thesis aims at examining the following questions:

(1) Are there differences in the brain’s automatic change detection response –
the neuromagnetic MMF – to stop consonant-vowel syllables between children
and adolescents with dyslexia and normally literate controls? If so, is the
observed neuronal activity pattern associated with behavioral discrimination
performance?

(2) Are there differences in source location of ERFs in response to stop
consonant-vowel syllables between children and adolescents with dyslexia and
normally literate controls? If so, is this a maturational phenomenon or rather a
stable characteristic of dyslexia?

(3) Do children and adolescents with dyslexia exhibit an auditory temporal
processing deficit? If so, does this deficit co-occur with a similar dysfunction
in the visual modality?

(4) Are improvements in literacy skills following linguistic training mirrored in
an altered magnetic-brain activity in SLI children with symptoms of dyslexia?
In other words, are there training-associated changes in the MMF and source
configuration of ERFs to stop consonant-vowel syllables?

In order to examine these experimental questions six studies were conducted, all of which are
presented in chapter II of this thesis.
II. Studies

Here I will present six studies (A-F) examining specific experimental questions on auditory processing in dyslexia. The general implications provided by these studies are outlined in chapter III of this thesis.

STUDY A: ATYPICAL ORGANIZATION OF THE AUDITORY CORTEX IN CHILDREN WITH DYSLEXIA

Introduction

Anatomical and functional studies of dyslexic brains suggest striking alterations in the temporal bank of the left-hemisphere sylvian fissure (Galaburda, 1993; Steinmetz, 1996). The region within and surrounding the sylvian fissure contains structures playing a specialized role for the analysis of acoustic signals. One prominent structure is the planum temporale, a plane caudal to the first Heschl’s gyrus (von Economo & Horn, 1930). Because the left-hemispheric planum is a part of the Wernicke’s speech comprehension area and because it is, on average, larger in size in normal right-handed adults than the corresponding structure of the right hemisphere (see Steinmetz et al., 1990a), the planum temporale has often been considered an important biological substrate of language lateralization.

The impetus for focusing attention on the planum temporale in dyslexic individuals derives from post-mortem studies of Galaburda and colleagues (for reviews see Galaburda, 1989, 1993). A number of post-mortem and in vivo studies have revealed either reduced or absent left-right asymmetry of the planum temporale in people with dyslexia (Galaburda et al., 1985; Lubs et al., 1988; Humphreys et al., 1990; Hynd et al., 1990; Larsen et al., 1990). A Magnetic Resonance Imaging (MRI) study by Larsen et al. (1990) suggests that dyslexic subjects with phonological processing deficits might display symmetrical planum. Galaburda et al. (1987) presented evidence that the symmetry is due to a larger right rather than a smaller left planum. Generalizing this finding Galaburda and associates proposed that symmetrical brains of dyslexics result from a diminished neural loss in the right hemisphere during prenatal development. It deserves mention, however, that more recent MRI techniques have failed to find significant changes in normal planum temporale asymmetry in people with dyslexia (Leonard et al., 1993; Schultz et al., 1994; Rumsey et al., 1997b; Best & Demb, 1999; Heiervang et al., 2000; Robichon et al., 2000). Variations in subject characteristics (i.e., criteria used to define dyslexia, heterogeneity of the dyslexic population, inadequate control

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34 This part of the thesis largely corresponds to the paper of Heim et al. (2000c).
of sex and handedness) and differences in anatomical definition of the planum temporale may play an important role in explaining these conflicting results (for reviews see Shapleske et al., 1999; Eckert & Leonard, 2000).

Using functional Magnetic Resonance Imaging, Shaywitz et al. (1998) observed a relatively reduced blood flow during the performance of phonological tasks in Wernicke’s area, angular gyrus, and striate cortex (Area 17) and a relative increase in frontal brain regions (e.g., Broca’s speech area) of dyslexic as compared to normal adults. A similar left-hemispheric activation pattern was obtained with Positron Emission Tomography by Brunswick et al. (1999) during an explicit reading experiment. Relative to normal controls, dyslexic readers showed reduced activation in the left posterior inferior temporal cortex (“Wernicke’s Wortschatz or thesaurus”, p.1913), left cerebellum, left thalamus, and medial extrastriate cortex and enhanced activation in a pre-motor region of Broca’s area. When reading was implicit, dyslexic subjects also demonstrated diminished activation in Wernicke’s Wortschatz, but no elevated activation in anterior parts of the brain.

Auditory event-related potential (ERP) components like the P50 and N100 constitute additional tools for the evaluation of information processing in children with different handicaps (e.g., Pinkerton et al., 1989; Brunswick & Rippon, 1994; Stein et al., 1995; Oades et al., 1996; Tonnquist-Uhlén, 1996). For example, Pinkerton et al. (1989) found reduced amplitudes in children with poor literacy skills in ERPs to repetitive 2000-Hz tone bursts, for the N100 at three of four scalp locations and for the P50 at the left temporal site. Furthermore, Brunswick and Rippon (1994) reported an amplitude reduction of the left temporal N100 to stop consonant-vowel syllables in dyslexic boys, when compared to normally reading controls.

The ERP component ‘mismatch negativity’ (MMN) has proven to be a suitable index for studying auditory discrimination in children. Importantly, the mismatch response has been demonstrated as a sensitive measure for distinguishing individuals with language-based learning disorders from healthy peers (for reviews see e.g., Cheour et al., 2000; Kraus & Cheour, 2000; Kujala & Näätänen, 2001; Näätänen, 2001). For instance, Kraus et al. (1996) observed a nearly absent mismatch response to deviant stop consonant-vowel syllables in children with learning problems. Moreover, the neuronal dysfunction was correlated with behavioral discrimination of these syllables (viz. /da/ versus /ga/) leading Kraus and colleagues to suggest that the speech-sound discrimination deficits probably have their origins in the auditory pathways and may be pre-attentive in nature. Schulte-Körne et al. (1998a)
found that the MMN to the stop-consonant syllable contrast /ba/-/da/ in dyslexic boys was attenuated but not absent.

Particular attention has been paid to the source locations of the P50, N100, and MMN, especially by means of magnetoencephalography (MEG). MEG studies have demonstrated that corresponding auditory event-related fields (ERFs) – P50m, N100m, and ‘mismatch field’ (MMF) – have sources in the supratemporal auditory cortex (Pantev et al., 1986; Yoshiura et al., 1996; for a review see Hari, 1990). However, it is important to distinguish between the structures generating the event-related components and the underlying causes for a deviance in these responses. Sources in the supratemporal cortex, for instance, can be influenced by bottom-up processing in the auditory pathway or top-down processing occurring in other parts of the brain. Taken together, high-temporal resolution electrophysiological procedures are particularly able both to detect atypical information processing in children and to reveal general functional principles of the brain.

The purpose of the present study was to investigate the left-hemisphere auditory cortex response to speech sounds in children with dyslexia by using MEG technique. Based on anatomical and physiological findings, we put forward two hypotheses: (i) there is a difference in the organization of the auditory cortex revealed particularly by differences in source locations of M80 and M210 components between dyslexic children and normally literate controls; and (ii) the MMF to stop-consonant syllable contrasts is attenuated in children with dyslexia.

Methods

Subjects

Eleven developmentally dyslexic children (2 females) were identified from the records of the outpatient clinic at the Department of Phoniatics and Pedaudiology of the Münster University (Münster, North Rhine-Westphalia). Diagnosis of dyslexia was based on a discrepancy (≥ 1 SD) between actual spelling scores and spelling predicted by general mental ability (Schulte-Körne et al., 1998a); dyslexic subjects were also required to show poorer than normal reading achievement. Nine normal controls were matched for age, sex (1 female), mother tongue (all native speakers of German), and handedness (all right-handed). One dyslexic male was excluded from further analyses because of insufficient data quality (movement artifacts during recording). Mean age was 10.50 years (range: 8-13 years; SD =
1.72) and 10.78 years (range: 8-14 years; SD = 2.28) for dyslexic and control groups, respectively ($t_{17} = -0.3$, $p > .05$, n.s.).

Subjects had no history of neurological, otological, or psychiatric disease. Furthermore, none of the children was on medication which might have affected the central nervous functioning. As shown in Table II.A.1, written language skills were documented by pseudoword reading\(^{35}\) (Welte, 1981), standard reading [Zürcher Lesetest (Linder & Grissemann, 1980)], and spelling tests [Diagnostischer Rechtschreibtest for grade 2, 3, 4, or 5 (Meis, 1970; Müller, 1983a,b; Grund et al., 1994); Westermann Rechtschreibtest for grades 6 to 8 (Rathenow et al., 1981)]. Mental ability was assessed by an age-appropriate German adaptation of Cattell’s Culture Fair Intelligence Test, CFT 1 (Weiss & Osterland, 1979) or CFT 20 (Weiss, 1987). The CFT provides a non-verbal measure on intelligence, thus reducing the influence of verbal abilities and education on IQ test performance.

The nature of the study was fully explained to the subjects’ parents and their consent obtained. Every child received a cinema ticket and a surprise trinket for her/his voluntary participation.

**Table II.A.1: Psychometric data for study groups**

<table>
<thead>
<tr>
<th></th>
<th>Control Ss (n = 9)</th>
<th>Dyslexic Ss (n = 10)</th>
<th>$U^c$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-verbal intelligence</td>
<td>IQ(^a)</td>
<td>120</td>
<td>111.5</td>
<td>27.5</td>
</tr>
<tr>
<td>Written language scores</td>
<td>% Rank(^b) (bandwidth)</td>
<td>50-100</td>
<td>0-25</td>
<td></td>
</tr>
<tr>
<td>Standard reading</td>
<td>Errors(^a) (max. = 30)</td>
<td>1</td>
<td>12</td>
<td>8.5</td>
</tr>
<tr>
<td>Pseudoword reading</td>
<td>% Rank(^a)</td>
<td>81</td>
<td>13</td>
<td>0</td>
</tr>
</tbody>
</table>

\(^a\)median; \(^b\)mode; \(^c\)Mann-Whitney U-test; n.s., not significant ($p > .05$).

\(^{35}\)Normally the administered test demands verbal repetition of spoken pseudowords, two to six syllables in length.
Magnetoencephalographic measures

Stimulation

Three different blocks of stimuli were presented through an ear tube (see Pantev et al., 1995) to the subject’s right ear (contralateral to the MEG-investigated hemisphere) with a constant intertrial interval (stimulus onset to stimulus onset) of 1 s and a stimulus duration of 200 ms. Blocks 1 and 2 included 1000 stimuli each of natural spoken syllables [da] and [ga], while 500 sine wave tones (1000 Hz and 1200 Hz, with 10 ms rise and fall times) were presented in the third block (see also Table II.A.2). The stimulus intensity was at 65 dB sensation level.

In each block, an oddball paradigm was employed in which a deviant stimulus (D, probability of occurrence 10%) was presented in a series of standards (S, probability of occurrence 90%): Block 1: [da] = S, [ga] = D; Block 2: [ga] = S, [da] = D; Block 3: 1000 Hz = S, 1200 Hz = D. Stimuli were presented in a pseudorandom order with at least six standard stimuli separating presentations of two deviants. To achieve a better signal-to-noise ratio, the two blocks with verbal material were presented a second time, resulting in five blocks altogether. The order of the blocks was pseudorandomized, with no two identical blocks occurring consecutively.

To control the level of arousal as well as to constrain eye movements and blinks during recording, subjects watched silent cartoons or movies displayed on a special magnetic-field free video screen. In addition, concentrating on the film helped the children to lie still during recording, which lasted ≈45 min. The participants were instructed to attend to the video program and to ignore the auditory stimuli. They were also informed that the examiner would enter the recording room after every block and that slight movements of the lower body were permitted at this time. Two-way speakers and a video camera allowed the researchers to communicate with the child, who stayed alone in the magnetically shielded room during data acquisition.

Data acquisition

Recordings were performed inside a magnetically shielded room (Vacuumschmelze GmbH, Hanau, Germany), using a 37-channel biomagnetometer (Magnes™, Biomagnetic

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36 Previous studies have shown that the strongest auditory ERFs are recorded on the side contralateral to the side of handedness (Elberling et al., 1980, 1981) and stimulation (Elberling et al., 1980, 1981; Pantev et al., 1986).

37 The frequency-dependent representation of pure tones in primary auditory area should serve as a landmark (e.g., Pantev et al., 1995).
Technologies, Inc., San Diego, CA, USA). The detection coils of the system are arranged in a circular concave array with a diameter of 144 mm, and a spherical radius of 122 mm. The axes of the detection coils are normal to the surface of the sensor array. The distance between the centers of two adjacent coils measures 22 mm; each coil has a diameter of 20 mm. The sensors are configured as first-order axial gradiometers with a baseline of 50 mm. The spectral density of the intrinsic noise of each channel was between 5 and 7 fT/√Hz in the frequency range > 1 Hz. The children rested on their right side with their head, neck, and upper body supported by a specially fabricated vacuum mattress. A sensor position indicator system determined the spatial locations of the sensors relative to the head and indicated if head movements occurred during recordings. The neuromagnetic field pattern was recorded over the left supratemporal cortex. The measurement system was centered over a point ≈1.5 cm superior to position T3 of the 10-20 system for electrode placement (corresponding approximately to the midpoint of the sylvian fissure) and was positioned as close as possible to the subject’s head. Using a bandwidth of 0.01-100 Hz for filtering and a sampling rate of 297.6 Hz, five blocks of 500 stimulus-related epochs, each of 700 ms (including 100 ms prestimulus baseline), were recorded and stored for further analysis.

Data analysis

For every block of data, selective averages were computed from the responses to the standards and deviants separately for each stimulus class. Stimulus-related epochs that were contaminated by muscle or eye blink artifacts (signal variations of more than 3.5 pT) were automatically rejected from the averaging procedure. A baseline correction was carried out for all channels by subtracting the mean value of the signal during the 100 ms prior to the stimulus. The data were low-pass filtered using a 20 Hz second-order zero-phase shift Butterworth filter (12 dB/oct).

In order to analyze the MMF, a difference wave was computed for each of the five blocks by subtracting the selective average responses to the standard stimulus from the selective average responses to the deviant stimulus (see Table II.A.2). Due to the low strength of the five difference waves around 40 fT in comparison to the biological noise, we decided not to assess the source locations of the mismatch generator. For comparisons of the mismatch waves, two evaluation intervals (A = 180-280 ms and B = 280-380 ms) were selected.
A single moving equivalent current dipole (ECD) model in a spherical volume conductor was used for source analyses. Data quality sufficient for this data analysis technique was achieved for standard stimuli. Therefore, source analysis was calculated for the M80 and M210 components in response to standard stimuli. Source parameters included location, dipole moment as well as parameters estimating the quality of the dipole fit, i.e., goodness of fit and confidence volume. Source locations were computed in a head-based Cartesian coordinate system. The origin of this coordinate system was set at the midpoint of the medial-lateral ($y$-) axis, the axis that joined the center points of the entrance to the acoustic meatuses of the right and left ear (positive towards the left ear). The posterior-anterior ($x$-) axis extended from the origin to the nasion (positive towards the nasion) and the inferior-superior ($z$-) axis was perpendicular to the $x$-$y$ plane (positive towards the vertex). While the dipole moment ($q$) reflects field strength (in nAm), the goodness of fit value (in %) describes how much of the measured magnetic field is accounted for by the ECD.

Analyses of the five standard response waves ([da]-S and [ga]-S first and second occurring block, 1000 Hz) led to the estimated dipole locations of the M80 and M210. For each standard evoked magnetic field a post-stimulus time interval containing the field maximum and minimum (= evaluation interval in ms), the local root mean square (RMS) maximum (in fT), and the local goodness of fit maximum were chosen. The spatial ECD coordinates ($x, y, z$) for every component were assigned to the corresponding averaged values of 5-8 adjacent sampling points (16.75-26.80 ms) around the center of the obtained evaluation intervals. Only those $x$-, $y$-, and $z$-values were included in the average which fulfilled the following additional criteria based on statistical and anatomical considerations: (a) goodness of fit $> 90\%$, (b) distance of ECD to midsagittal plane $> 1.5$ cm, (c) inferior-superior value $> 3$ cm, and (d) confidence volume $< 0.1$ cm$^3$. Since the measured parameters ($x, y, z, q, \text{RMS}$, 

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**Table II.A.2: The five difference ($\Delta$) waveforms**

<table>
<thead>
<tr>
<th>No.</th>
<th>Difference waveforms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1a.</td>
<td>$\Delta ([ga]-D - [da]-S)$; first block</td>
</tr>
<tr>
<td>1b.</td>
<td>$\Delta ([ga]-D - [da]-S)$; second block</td>
</tr>
<tr>
<td>2a.</td>
<td>$\Delta ([da]-D - [ga]-S)$; first block</td>
</tr>
<tr>
<td>2b.</td>
<td>$\Delta ([da]-D - [ga]-S)$; second block</td>
</tr>
<tr>
<td>3.</td>
<td>$\Delta (1200 \text{ Hz-D} - 1000 \text{ Hz-S})$</td>
</tr>
</tbody>
</table>

*Note. $D = \text{deviant}, S = \text{standard}.$*
and latency) of the M80 and M210 components in the two identical syllable blocks were very similar (test/retest criterion) the data of these blocks were averaged for further analysis.

**Statistics**

The latencies, the RMS amplitudes, the dipole moment, and the ECD locations (anterior-posterior, medial-lateral, inferior-superior) of the M80 and M210 were used as dependent variables in three-way analyses of variance (ANOVA). Group (dyslexic vs. control) was treated as between-group factor, Component (M80 vs. M210) and Stimulus ([da]-S vs. [ga]-S, averaged over two identical syllable blocks vs. 1000 Hz) as repeated measurement factors.

In the case of the MMF, a three-way ANOVA was computed with Group treated as a between-group factor, Interval (A vs. B) and Block (1-5, see also Table II.A.2) as repeated measures. The dependent variable was the RMS amplitude.

Probability effects of more than two-level repeated measures factors were Greenhouse-Geisser adjusted. Post-hoc comparisons were carried out using the Scheffé test.

**Results**

**M80 and M210**

**Latencies**

In response to the standard stimuli, a sequence of two prominent waves was easily detectable in all subjects. We labeled these deflections M80 and M210, as the latencies were distinctly longer than the corresponding set of deflections (P50m and N100m) in adults. An analysis of the latencies of the M80 (mean latency = 80 ms, range = 67 ms) and M210 (mean latency = 211 ms, range = 105 ms) attained a significant main and interaction effect for the factors Component and Stimulus. The Component \( \times \) Stimulus interaction \( F(2,34) = 5.6, p < .02, \epsilon = .685 \) as well as the main effect of the factor Stimulus \( F(2,34) = 10.7, p < .001, \epsilon = .777 \) result from shorter M210 latency to the standard tone (mean latency = 194 ms) than to the standard syllables [da] (mean latency = 221 ms) and [ga] (mean latency = 219 ms), as indicated by significant post-hoc comparisons \( ps < .05 \). The difference between the consonant-vowel syllables was insignificant. For the component M80 the tone-[da]-[ga]

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38 In their MEG study, Paetau et al. (1995) have demonstrated major effects of interstimulus interval on the N100m, with delayed or absent N100m in healthy children at rapid stimulation rates and adult-type N100m responses at slower stimulation rates (1.2-2.4 s).
contrasts (mean latency = 76, 81 and 84 ms, respectively) did not reach significance. There were no significant group differences.

Figure II.A.1 demonstrates sets of magnetic field waveforms in response to the standard syllable [da] for an individual dyslexic and control subject. Clear extrema and polarity reversals of the M80 (first deflection) and M210 (second deflection) are evident.

**Figure II.A.1.** Sets of 37 averaged waveforms in response to the standard syllable [da] (second occurrence, see Table II.A.2) measured over the left hemisphere in one dyslexic and one control subject. The center of the sensor array corresponds to the point =1.5 cm superior to the position T3 (approximately the midpoint of the sylvian fissure) of the 10-20 system for electrode placement of the subject. The responses are presented according to the placement of the pickup coils relative to the child’s head. Flux leaving the skull is positive (outgoing magnetic field); flux entering the skull is negative (ingoing field). The first (positive) deflection represents the M80, the second (negative) deflection the M210. The epoch length is 700 ms.

**Magnetic field amplitudes and dipole moments**

The three-way ANOVA of M80 and M210 RMS values obtained for the standard stimuli revealed significant main effects for the Component \([F(1,17) = 9.7, p < .006]\) and Stimulus \([F(2,34) = 9.3, p < .002, \epsilon = .798]\) factors. The field amplitude of the M210 was larger than that of the M80 (mean RMS value = 101 vs. 71 fT, respectively). Finally, the two components exhibited greater signal power to syllables (mean RMS value ± SEM for standard [da] = 88 ± 5.47 fT and standard [ga] = 92 ± 5.52 fT) than to the tone (78 ± 5.78 fT; ps < .05). The group factor had no statistically meaningful influence on field amplitudes. Possible interactions between the Group, Component, and Stimulus factors remained insignificant.
The results of the statistical analysis of the RMS field amplitudes were confirmed by the corresponding dipole moment values. First, for both the dyslexics and controls, the dipole moment of the M210 (mean $q$-value = 33 nAm) was nearly twice as large as that of the M80 (mean $q$-value = 18 nAm) elicited by standard stimuli [$F(1,13) = 14.2, p < .002$]. Second, the dipole moment was generally stronger in the syllable conditions (mean $q$-value for standard [da] = 28 nAm and standard [ga] = 30 nAm) than in the tone condition [mean $q$-value = 19 nAm; $F(2,26) = 7.4, p < .01, \epsilon = .648$]. Third, the group effect and possible interactions among the Group, Component, and Stimulus factors failed to attain significance.

**Source locations**

The $x$-, $y$-, and $z$-coordinates of the estimated source locations of the M80 and M210 obtained for the non-verbal and verbal standards are presented in Figure II.A.2.

![Figure II.A.2](image)

**Figure II.A.2.** Source locations of the M80 and M210 to the different standard stimuli for the dyslexic and control groups. The left 2D-plots represent anterior-posterior and inferior-superior axes, the right plots medial-lateral and inferior-superior axes.

Within each group, the two-dimensional plots indicate source clusters for the different M80 waves on the one hand and M210 waves on the other, i.e., three-way ANOVAs of the $x$-, $y$-, and $z$-values yielded no significant Component $\times$ Stimulus interactions. Interestingly, the statistical analysis of the $x$-coordinate (posterior-anterior direction) revealed a highly
significant Group × Component interaction \([F(1,13) = 12.4, \ p < .004]\). The overall significance was due entirely to the group contrast of the M210 localization \((p < .004)\) indicating that in the dyslexics the sources of the M210 were considerably more anterior than in the controls (mean \(x\)-value = 1.26 cm and \(-0.21\) cm, respectively). This result is illustrated in Figure II.A.2 (left plots). For the \(y\)- and \(z\)-axis values, only the factor Component had a pronounced impact \([F(1,14) = 6.8, \ p < .02\] and \(F(1,14) = 8.0, \ p < .01]\): As can be seen in Figure II.A.2 (right plots), the sources of the M210 are located more medially (mean \(y\)-value = 3.97 cm) and more inferiorly (mean \(z\)-value = 5.52 cm) than the corresponding equivalent sources of the M80 (mean \(y\)-value = 4.38 cm, mean \(z\)-value = 6.27 cm).

Localizations of the M210 generator to tonal and verbal standard stimuli obtained from individual subjects in each group are displayed in Figure II.A.3a. To account for a possible effect of neuroanatomical variability (e.g., size of the head) on M210 source locations, localizations were normalized with respect to M80 as the difference \(\Delta(M210 - M80)\). These relative source locations are presented for each dyslexic and control subject in Figure II.A.3b. Figure II.A.3b (left) emphasizes the group difference of the M210 locations in the anterior-posterior direction. The majority of the dyslexic subjects processed the standard tone and syllables at a more anterior position [mean group difference\(^{39}\) = 1.27 cm; \(F(1,17) = 6.3, \ p < .02\)]. On the other hand, similar locations of acoustic processing of dyslexic and normally literate children in medial-lateral [mean group difference = \(-0.04\) cm; \(F(1,17) = 0.02, \ p > .05, \ n.s.\)] and inferior-superior directions [mean group difference = \(-0.42\) cm; \(F(1,17) = 0.7, \ p > .05, \ n.s.\)] again become obvious in Figure II.A.3b (right).

\[^{39}\] Mean group differences were calculated by subtracting the mean relative source locations in the control group from those in the dyslexic group.
Figure II.A.3. Individual subject data from 10 dyslexics (filled circles) and 9 controls (open triangles) for source locations of the M210 averaged across tonal and verbal standard stimuli. The left 2D-plots represent anterior-posterior (x) and inferior-superior axes, the right plots medial-lateral (y) and inferior-superior axes (z). (a) Absolute locations of the M210 generator. (b) Localizations of the M210 relative to the source location of the M80. Mean coordinates (SEM) are \( x, y, z = [0.81, -0.39, -0.85 (0.40, 0.18, 0.31)] \) and \( x, y, z = [-0.46, -0.35, -0.43 (0.28, 0.25, 0.39)] \) for dyslexic and control groups, respectively.
Mismatch field

In all subjects, standard and deviant waveforms in both the tone and speech blocks could be derived. Figure II.A.4 reveals the syllable data (standard and deviant waveforms) averaged for the dyslexics (left) and the controls (right). Both groups showed a significant difference between deviant and standard.

![Figure II.A.4](image_url)

*Figure II.A.4.* Grand average of the RMS waveforms elicited by the syllables [da] and [ga] in the dyslexic and control groups. The dashed line characterizes the answer to the standard [da], the solid line the response to the deviant [ga]. The pronounced difference between deviant and standard responses becomes evident within the latency range 280 to 380 ms.

The three-way ANOVA of the RMS values did not indicate a group main effect implying that the mismatch responses of the dyslexics and controls did not differ in size. A statistically meaningful effect was yielded for the factor Interval \[F(1,17) = 5.6, p < .03\], i.e., the MMF activity was most prominent in the second evaluation window B (mean RMS value = 41 vs. 46 fT for interval A and B, respectively). The non-significant Group × Interval interaction emphasized the generality of the interval effect. The factor Block (see Table II.A.2) gained neither a significant influence by itself nor as an interaction with the variables Group and/or Interval indicating that all subjects could automatically distinguish between simple and complex auditory stimuli.
Discussion

The present study examined two hypotheses: (i) dyslexia is characterized by a variant in the organization of the left-hemisphere auditory cortex, as marked by the sources of M80 and M210; and (ii) the MMF to deviant consonant-vowel syllables is attenuated in dyslexic children. The experimental results confirmed the first assumption. The M210 generator was localized more anterior in the left temporal lobe of the dyslexic than of the control children. The magnitude of this effect was considerable with an average group difference of 1.47 cm for the absolute location of the M210 source. The effect was also clearly evident relative to the location of the M80 generator, although the latter did not systematically differ between the participant groups.

In adults, the middle-latency ERP components, like P50 to tones, are directly associated with Heschl’s gyrus (Liégeois-Chauvel et al., 1994), whereas the N100m arises from the depolarization of pyramidal neurons in the planum temporale (Elbert, 1998; Lütkenhöner & Steinsträter, 1998). This view of generator locations is consistent with the present finding in the control children, with the middle-latent M80 being located anterior to the subsequent M210. Probably in both the dyslexic and control children, primary auditory cortex was activated in the M80 latency range. In the subsequent processing stage, characterized by the M210, however, only the controls shifted activation to the posterior regions of the planum temporale. In contrast, in dyslexics, brain structures tended to be activated anterior to the M80 in Heschl’s gyrus, i.e., in regions that are part of Wernicke's area but do not lie within the planum temporale.

One may suppose that, given no difference was found for the localization of the M80 generator, the difference in M210 source cannot be attributed to gross anatomical variations. It deserves mentioning, however, that certain regions of the auditory cortex may have greater intersubject or developmental variability than others. Assuming that the M210 but not the M80 is generated within a region with more natural variability, it is possible that the present group difference simply results from a difference in anatomy. Whether the origin of the atypical source configuration in children with dyslexia is structural or functional in nature cannot be settled at this point. Nevertheless, MEG technique provides a promising tool for revealing atypical organization of the auditory cortex in children with dyslexia.

The second hypothesis that dyslexic individuals reveal an attenuated MMF to deviant consonant-vowel syllables could not be confirmed by the present analyses. Both groups produced significant and normally pronounced MMFs. In both groups and across all stimulus
conditions, the MMF was more prominent in the later time window B (280-380 ms), which is consistent with the rather long M210 latency.

The fact that an MMF group effect was not observed contrasts with the results reported by Kraus et al. (1996) and Schulte-Körne et al. (1998a), showing an absent/attenuated MMN to deviant syllables in learning disabled and dyslexic children. A factor possibly accounting for the different finding might be the characteristics of speech sounds. While Kraus’ and Schulte-Körne’s group presented synthetic syllables mainly differing in the frequency transitions of the second and third formant, we intended, by using natural language, to improve the ecological validity of our study. The spoken syllables [da] and [ga] diverged over and above that in the slowly changing parameters of the fundamental frequency providing information about the pitch of the speaker. As stated by Tallal and co-workers (e.g., Tallal et al., 1993) dyslexic children do not have problems understanding sounds when presented slowly but do in the perception of brief transient elements which may characterize certain stop consonants. Consequently, the normal mismatch response in our dyslexic sample might reflect variations in pitch rather than in the rapid changes of spectral characteristics in consonants. Further MEG studies utilizing carefully generated synthetic consonant-vowel syllables are needed in order to clarify the diverging mismatch results.

Finally, it should be kept in mind that Kraus et al. (1996) and Schulte-Körne et al. (1998a) examined the electric MMN, whereas we report findings for its magnetic counterpart. It may well be that the MMF reveals differences between the responses to standard and deviant stimuli more clearly that are blurred in the electrical recordings due to greater biological noise. Whenever the current sources in the cerebral cortex are predominantly tangential to the scalp, the MEG is likely to have a signal-to-noise ratio superior to that generated in an electroencephalogram (EEG), since the biological noise sums to a radially oriented source (Eulitz et al., 1997; Elbert, 1998). The limited number of electrodes (9-19) typically used in EEG studies could be another reason why a MMN has gone undetected in subjects with reading and spelling impairments: The different source configurations in dyslexic and control children, as found in the present study, might lead to a different scalp distribution of mismatch activity and consequently, an electrode array optimized for normal MMN topography may fail to detect deviant distributions. To resolve this issue, future studies should include either a high-density electrode array or a combination of MEG and EEG.

In conclusion, our data suggest that dyslexics and normally literate children differ as to the organization of their left-hemisphere auditory cortex. Whether this atypical configuration is specific to the auditory cortex of the left hemisphere or is paralleled by a similar pattern in
the right hemisphere should be examined in future studies monitoring the activation of both hemispheres.

**Summary of study A:** Neuroanatomical and -functional studies have converged to suggest an atypical organization in the temporal bank of the left-hemisphere sylvian fissure for dyslexia. Against the background of this finding, we applied high temporal resolution magnetoencephalography to investigate functional aspects of the left-hemisphere auditory cortex in 10 right-handed developmentally dyslexic children (aged 8-13 years) and 9 matched normal subjects (aged 8-14 years). Event-related field components during a passive oddball paradigm with pure tones and consonant-vowel syllables were evaluated. The first major peak of the auditory evoked response, the M80, showed identical topographical distributions in both groups. In contrast, the generating brain structures of the later M210 component were located more anterior to the earlier response only in children with dyslexia. Control children exhibited the expected activation of more posterior source locations of the component that appeared later in the processing stream. Since the group difference in the relative location of the M210 source seemed to be independent of stimulus category, it is concluded that dyslexics and normally literate children differ as to the organization of their left-hemisphere auditory cortex.
STUDY B: BEHAVIORAL AND NEURONAL ASPECTS OF LANGUAGE DYSFUNCTION IN DYSLEXIA

Introduction

There is ample evidence that a fair proportion of children with language-based learning impairments has difficulty in perceiving certain speech sounds (for reviews see Tallal et al., 1993; Farmer & Klein, 1995; Bishop, 1997). Studies on categorical perception of synthetic stop-consonant continua indicate that children with dyslexia may be less accurate than normal readers on cross-category, but not on within-category discrimination (Godfrey et al., 1981; Werker & Tees, 1987; Serniclaes et al., 2001). For example, Serniclaes et al. (2001) reported reduced discrimination of cross-category changes on a /ba/-/da/ continuum coupled with enhanced discrimination of acoustic differences belonging to the same phoneme category in dyslexic children versus normally reading controls. Thus, dyslexic children were assumed to be less successful in exploiting phonological contrasts that usually promote discrimination across phoneme boundaries.

Tallal and Piercy (1974, 1975) observed that school-age children with specific language impairment may succeed in discriminating the stop-consonant syllables /ba/ and /da/ when the fast (43-ms) transitional elements were artificially lengthened (to 95 ms). The finding was one impetus for developing a training program which exposes children with language-based learning disorders to speech that has been acoustically modified so that the transitional elements are temporally extended and amplified (Merzenich et al., 1996; Tallal et al., 1996; for a survey see Tallal et al., 1998).

In a group of learning-disabled children, Bradlow et al. (1999) investigated the effects of lengthened formant transition duration not only on behavioral discrimination but also on neural representation of stop-consonant syllables. They found elevated discrimination thresholds on both rapid and extended transition duration /da/-/ga/ contrasts. At the neural level, however, the electrophysiological mismatch negativity (MMN, see under ‘Auditory event-related potentials’, chapter I) to short-duration /da/-/ga/ contrasts was significantly attenuated, whereas the MMN to the temporally extended across-category changes were more pronounced and resembled those of their normally learning age-mates. According to Bradlow et al. (1999) the MMN data indicate that, at a pre-attentive neural level, the long-transition syllables were represented more accurately than the short-transition stimuli in children with learning problems.

In the present study, we aimed at replicating Tallal and Piercy’s (1975) findings of superior discrimination performance on temporally extended relative to rapid formant
transition consonant syllables in a group of children and adolescents with dyslexia. The German syllables [baː] and [daː] were presented following the same-different procedure of Tallal and Piercy (1974, 1975). In particular, we sought to determine the neuronal correlates of processing short and long transitional [baː]-[daː] contrasts using the magnetic counterpart of the MMN, the mismatch field (MMF). Motivated by the findings on categorical perception suggesting enhanced within-category discrimination on stop-consonant continua in dyslexia, MMF was also evaluated in response to short and long transition duration contrasts belonging to the category [baː] (see Table II.B.2).

**Methods**

**Subjects**

A total of 43 children and adolescents participated in the study. Twenty-two participants (aged 8.6-16.5 years, 6 females) had been diagnosed with developmental dyslexia, 21 participants (aged 8.5-17.8 years, 9 females) served as normally literate controls. Study groups did not differ significantly on age (see Table II.B.1). All subjects evidenced at least normal non-verbal intelligence (IQ > 85). On average, controls displayed significantly higher non-verbal IQs than the dyslexic subjects (see Table II.B.1). All participants were native speakers of German. Handedness was assessed according to the Edinburgh Handedness Questionnaire (Oldfield, 1971). Subjects with a laterality quotient (LQ) ≥ +70 were designated right-handers. One of the dyslexic and two of the control subjects were ambidextrous (−60 ≥ LQ ≤ +60); in each group there was one left-hander (LQ ≥ −70). Subjects were reported free of otological, neurological, or psychiatric disease. Furthermore, none of the participants was on medication which might affect central nervous functioning.

Dyslexic subjects were recruited by advertisement from among members of the German Dyslexia Association and by recommendations of the local educational counseling service (Schulpsychologische Beratungsstelle Konstanz, Baden-Württemberg). As shown in Table II.B.1, their performance in language-related tests (reading/phonological decoding, spelling, and verbal memory span) consistently differentiated them from the control group.

Written informed consent was obtained from all participants and their parents. Subjects received shopping vouchers and/or cinema tickets for participating in the study.
Table II.B.1: Sample characteristics (mean ± SD)

<table>
<thead>
<tr>
<th></th>
<th>Control Ss (n = 21)</th>
<th>Dyslexic Ss (n = 22)</th>
<th>t-test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>12.64 ± 2.47</td>
<td>12.31 ± 2.77</td>
<td>n.s.</td>
</tr>
<tr>
<td>Non-verbal IQ</td>
<td>114.43 ± 14.49</td>
<td>104.64 ± 16.05</td>
<td>p &lt; .04</td>
</tr>
<tr>
<td>Standard word reading</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Errors (z-scores)</td>
<td>−0.60 ± 0.16</td>
<td>0.58 ± 1.12</td>
<td>p &lt; .001a</td>
</tr>
<tr>
<td>Time (s)</td>
<td>73.24 ± 24.66</td>
<td>123.59 ± 57.74</td>
<td>p &lt; .001b</td>
</tr>
<tr>
<td>Standard passage reading</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Errors (z-scores)</td>
<td>−0.70 ± 0.19</td>
<td>0.67 ± 1.00</td>
<td>p &lt; .001a</td>
</tr>
<tr>
<td>Time (s)</td>
<td>147.10 ± 52.61</td>
<td>281.41 ± 132.40</td>
<td>p &lt; .001b</td>
</tr>
<tr>
<td>Word reading</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Points (max. = 300)</td>
<td>287.24 ± 9.77</td>
<td>226.68 ± 48.49</td>
<td>p &lt; .001a</td>
</tr>
<tr>
<td>Time (s)</td>
<td>147.00 ± 53.24</td>
<td>245.73 ± 95.94</td>
<td>p &lt; .001b</td>
</tr>
<tr>
<td>Pseudoword reading</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Points (max. = 300)</td>
<td>260.29 ± 16.44</td>
<td>159.64 ± 51.11</td>
<td>p &lt; .001a</td>
</tr>
<tr>
<td>Time (s)</td>
<td>221.48 ± 61.51</td>
<td>328.77 ± 101.24</td>
<td>p &lt; .001b</td>
</tr>
<tr>
<td>Standard spelling</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Errors (z-scores)</td>
<td>−0.85 ± 0.32</td>
<td>0.81 ± 0.70</td>
<td>p &lt; .001a</td>
</tr>
<tr>
<td>Digit span forwards</td>
<td>5.91 ± 0.84</td>
<td>5.37 ± 0.67</td>
<td>p &lt; .03c</td>
</tr>
<tr>
<td>Digit span backwards</td>
<td>4.57 ± 0.66</td>
<td>3.92 ± 0.86</td>
<td>p &lt; .01c</td>
</tr>
</tbody>
</table>

*a* t-test for unequal variances; *b* statistical analysis is based on logarithmic data; *c* group comparison included data from 20 out of 22 dyslexics; n.s., not significant (p > .05). Non-verbal intelligence was assessed with Raven’s Standard Progressive Matrices (Heller et al., 1998). Oral reading was examined using word and passage reading of the Zürcher Lesetest (Linder & Grissemann, 1998). In addition, reading and phonological decoding skills were documented using non-standardized word- and pseudoword-reading tests (points system: 0 = no response or error, 1 = self-correction, 2 = complete/partial repetition of an item, 3 = correct response; full listings of the reading-test items are included in the Appendix). Depending on the subject’s grade, the Diagnostische Rechtschreibtest, DRT 2, 3, 4, and 5 (Müller, 1983a, 1997; Grund et al., 1994, 1995) or Westermann Rechtschreibtest, WRT 6+ (Rathenow et al., 1981) was administered to estimate spelling abilities. Due to a lack of German normative data for older children, we decided not to transform reading and spelling raw scores into %-ranks; z-scores (mean = 0, SD = 1) are therefore given. Digit span forwards and backwards were determined using a one-up one-down staircase procedure; item presentation was auditory, recall was verbal (for further details see study F of this thesis).

**Stimuli**

Two 10-item stimulus continua varying in equal steps from [ba:] to [da:] were created using Speechlab software (Diesch, 1997) based on a Klatt cascade/parallel formant
synthesizer (Klatt, 1980). All syllables were generated in the cascade branch of the synthesizer at a 10 kHz sampling rate. Each syllable had a duration of 250 ms including a formant transition (FT) period of either 40 ms (rapid FT continuum) or 90 ms (extended FT continuum). Stimuli of the continua were composed of three formants (F1-F3) and differed in the onset frequencies of the second and third FT. The end points of each continuum were modeled after the stimuli from Tallal and Piercy’s (1974) study; stimulus frequencies were adjusted for the German language. The starting frequencies of the second and third FT were 1095 Hz and 2100 Hz for the end point syllables [ba:] and 1702 Hz and 2633 Hz for the end point syllables [da:]. The steady-state formant frequencies of the vowel [a:] were 770, 1340, and 2400 Hz for F1, F2, and F3, respectively. The fundamental frequency of each syllable started at 128 Hz and decreased linearly to 109 Hz at stimulus offset. The amplitude of voicing was constant at 54 dB and fell linearly to 11 dB during the last 25 ms of each stimulus.

For the present study, five stimuli of each speech continuum were selected based on the results of a categorical perception experiment. Sixteen school-age children (3 females, aged 8-16 years) and 17 adults (6 females, aged 18-48 years) with normal hearing and literacy skills participated in two experimental conditions. Condition 1 contained the rapid FT 10-item [ba:]-[da:] continuum, condition 2 the extended FT continuum. The order of the conditions was balanced across subjects. Syllables of the continua were delivered to both ears via Quart Phone IMP50 headphones at ≈72 dB sound pressure level. The ISI (defined as syllable offset to onset) was 1 s. In every condition, subjects were asked to classify 10 random presentations of each stimulus (= 100 trials) as [ba:] or [da:] by pressing a corresponding key on the computer keyboard (left cursor key = [ba:], right cursor key = [da:]). Figure II.B.1 illustrates the mean identification functions for both speech continua of the adult and the children’s group. In the rapid FT condition, adults perceived stimuli 1-6 as [ba:] and 8-10 as [da:] with at least 87% accuracy. Stimulus 7 was identified near chance level and thus formed the category boundary. In the extended FT condition, adults’ classification of stimulus 6 was not as clear as for the rapid FT continuum; here stimuli 1-5 were identified as [ba:] and 7-10 were identified as [da:] in at least 82% of the time (Figure II.B.1, top). In both the rapid and extended FT condition, children classified stimuli 1-5 as [ba:] and 7-10 as [da:] with at least 77% accuracy; stimulus 6 was ambiguously perceived and represented the category boundary (Figure II.B.1, bottom).
Figure II.B.1. Mean identification functions for the rapid and temporally extended FT [ba]-[da] continua in 17 adults (top) and 16 children (bottom) with normal literacy skills.

Based on these identification functions, four pairs of stimuli – each two corresponding pairs from the rapid and extended FT continuum – were chosen for the present neurophysiological experiment: (i) stimuli 5 and 8 which occurred near the category boundaries and were identified as different phonemes [ba:] and [da:], respectively, and (ii) stimuli 1 and 4 which were identified within the same phonemic category [ba:]. The former pairs are referred to as ‘across-categories’ pairs and the latter, as the ‘within-category’ pairs. The acoustic differences between stimuli 5 vs. 8 and stimuli 1 vs. 4 were equivalent. The starting frequencies of the second and third FT were 1567 Hz and 2515 Hz for stimulus 8, 1365 Hz and 2337 Hz for stimulus 5, and 1297 Hz and 2278 Hz for stimulus 4 (for frequency parameters of the end point stimulus 1, see above).
For the *psychoacoustic experiment* the endpoint syllables [ba:] (stimulus 1) and [da:] (stimulus 10) of the rapid and extended FT continua were used. Identification scores up to 99% in the adult and 98% in the children’s group (Figure II.B.1) indicated that the end point stimuli represent excellent examples of each phonetic category. Table II.B.2 provides an overview of selected stimuli for the present study.

Table II.B.2: Selected stimuli from the rapid and extended FT [ba:]-[da:] continuum

<table>
<thead>
<tr>
<th>Experiment</th>
<th>Stimulus pairs from the [ba:]-[da:] continua</th>
</tr>
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<tbody>
<tr>
<td>Psychoacoustic: Same-different task</td>
<td></td>
</tr>
<tr>
<td>Neurophysiological: Mismatch field</td>
<td></td>
</tr>
<tr>
<td>Psychoacoustic: Same-different task</td>
<td>1-10 [ba:]_1-[da:]_10</td>
</tr>
<tr>
<td>Neurophysiological: Mismatch field</td>
<td>1-4 [ba:]_1-[ba:]_4 within-category pairs</td>
</tr>
<tr>
<td></td>
<td>5-8 [ba:]_5-[da:]_8 across-categories pairs</td>
</tr>
</tbody>
</table>

*Psychoacoustic experiment: the same-different task*

Subjects were seated in front of a 17 in. NEC monitor in series with an IBM compatible 486-Personal Computer. Auditory signals were transmitted through headphones (Quart Phone IMP50). Two keys (left and right cursor keys) on the computer keyboard were used to collect subjects’ responses. Each participant was tested individually in a quiet room. The experimental session lasted ≈35 min.

The experiment consisted of two conditions: The rapid FT condition, in which the end point syllables [ba:]_1 and [da:]_10 of the rapid FT continuum were used and the extended FT condition, in which the endpoint syllables of the extended FT continuum were presented. The experimental procedure was identical for both conditions. The order of the conditions was counterbalanced across subjects; conditions were separated by a short break. The syllables were presented binaurally at ≈72 dB sound pressure level. The task required the subject to press the right (green) key if two successive syllables were the same ([ba:]-[ba:], [da:]-[da:]) and the left (red) key if they were different ([ba:]-[da:], [da:]-[ba:]) by using the index finger of her/his right hand. Feedback was provided after each stimulus pair (= trial) by a happy or unhappy face on the computer screen. The intertrial interval was 2 s. The training phase consisted of a maximum of 48 trials (12 for each possible pair combination, randomly intermixed) and finished earliest if a criterion of 20 correct responses in 24 consecutive trials was achieved. The task was terminated for subjects who did not meet the criterion. During
training an ISI (defined as syllable offset to onset) of 428 ms was employed; in the immediately following testing phase syllables were presented at six different ISIs: 8, 15, 30, 60, 150, and 305 ms (Tallal and Piercy, 1974, 1975). Testing included 48 trials, eight (2 × 4 syllable-pair combinations) for each ISI, with a randomized presentation of the different intervals. The percentages of correct trials were measured at each ISI.

**Neurophysiological experiment: the mismatch field**

**Stimulation**

Syllables were presented binaurally through a magnetically silent and echo-free delivery system with an almost linear frequency characteristic (deviations less than ± 4 dB in the range 200-4000 Hz). The duration of the intertrial interval (defined as stimulus onset to stimulus onset) was constant at 1 s. The stimulus intensity was 60 dB above the individually determined hearing level. In a passive oddball paradigm, repetitive standard stimuli were occasionally (\(p = 20\%\)) replaced by a deviant stimulus. Stimuli were presented in a pseudorandom sequence with no two deviants occurring consecutively. In the within-category pairs, \([ba]_1\) served as the standard and \([ba]_4\) as the deviant stimulus. For the across-categories pairs, \([ba]_5\) was the standard and \([da]_8\) the deviant. Stimulus sequences were presented in four blocks of 650 events (see Table II.B.3); the order of the blocks was counterbalanced between subjects.

**Table II.B.3: Blocks used in the oddball paradigm**

<table>
<thead>
<tr>
<th>No.</th>
<th>Experimental blocks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>([ba]_1-[ba]_4) rapid FTs</td>
</tr>
<tr>
<td>2</td>
<td>([ba]_1-[ba]_4) extended FTs</td>
</tr>
<tr>
<td>3</td>
<td>([ba]_5-[da]_8) rapid FTs</td>
</tr>
<tr>
<td>4</td>
<td>([ba]_5-[da]_8) extended FTs</td>
</tr>
</tbody>
</table>

*Note. [standard]-[deviant]*

To control for level of arousal, participants watched silent movies or cartoons displayed on a special magnetic-field free screen. The subjects were instructed to attend to the video program and to ignore the auditory stimuli. In addition, subjects were asked to prevent unnecessary eye or body movements during recordings. Compliance was verified by video-monitoring. Including preparation and breaks, the experimental session lasted 2-2.5 h.
Data acquisition and analysis

Magnetic responses were measured simultaneously from the left and right hemispheres using a whole-head neuromagnetometer (BTi, MAGNES 2500™, 4D Neuroimaging, San Diego, CA, USA) housed in a magnetically shielded chamber (Vacuumschmelze GmbH, Hanau, Germany). Subjects were seated with their legs extending horizontally on a height-adjustable bed, their backs leaning against a backrest, and their heads inside the helmet-like sensor. Within the sensor 148 superconducting quantum interference detectors (magnetometer-type) are arranged in a circular concave array so that they cover the entire cranium. The distance between two adjacent magnetometers measures 28 mm. The intrinsic system noise is 10 fT/√Hz down to 0.5 Hz.

Prior to the beginning of the MEG experiment, the positions of five indicator coils attached to the subject’s forehead and in front of the tragus of both ears were determined in relation to five anatomical landmarks (nasion, inion, vertex, and preauricular points), using an Isotrak 3-D digitizer (Polhemus Navigator Sciences, Colchester, VT, USA). The position of the subject’s head with respect to the sensor was determined for every experimental block by feeding currents to the indicator coils and locating them before and after each block.

Syllable-evoked brain responses were recorded continuously at a sampling rate of 508.63 Hz with a bandpass of 0.1-100 Hz. Eye movements and blinks were monitored by recording horizontal and vertical electro-oculograms (EOG). In an off-line mode, magnetic signals were first corrected for magnetocardiographic activity by means of a linear regression algorithm included with the BTi software package. Then, averaged waveform s for the standard and deviant syllables were calculated across epochs of 800 ms, including a 100 ms prestimulus baseline. Epochs with a MEG or EOG change > 3.5 pT or > 120 µV, respectively, were omitted from further analysis. The baseline was corrected for each channel according to the mean value of the signal during the 100 ms prior to the stimulus. After that, evoked fields were digitally low-pass filtered to 20 Hz using a second-order zero-phase shift Butterworth filter (filter roll-off: 12 dB/oct).

For analyzing the MMF, difference waves for each of the four blocks (see Table II.B.3) were calculated by subtracting the filtered averaged waves to the standard syllables from those to the deviant syllables. Mean MMF amplitude was measured in the root-mean square (RMS) difference waves scored in a fixed time-window between 150 and 300 ms (e.g., Kraus et al., 1993) for selected channels over the left and right hemispheres (see Appendix).
Results

**Psychoacoustic experiment: Same-different task**

Figure II.B.2 displays the performance in the rapid and extended conditions of the same-different task for the dyslexic and control groups.

![Figure II.B.2. Percentage correct for 21 control (open circles) and 22 dyslexic (filled triangles) subjects on the same-different task at various ISIs in the rapid and extended FT conditions.](image)

Statistical analysis was conducted on subject’s percent correct values using mixed-design analysis of variance (ANOVA) with FT (rapid vs. extended) and ISI (8, 15, 30, 60, 150, vs. 305 ms) as within-subjects factors and Group (dyslexic vs. control) as between-subjects factor. There was a significant main effect of Group \[F(1,41) = 10.6, p < .002\] with the dyslexic subjects having on average lower scores than the controls in both FT conditions across the range of ISIs tested [(mean ± SEM) 82.48 ± 2.81% vs. 93.61 ± 1.89%, respectively]. In addition, a significant Group × FT × ISI interaction was obtained \[F(5,205) = 2.7, p < .02\]. A follow-up test revealed largest significant group contrasts at the 15-ms ISI in the rapid FT condition and at ISIs of 8 and 30 ms in the extended FT condition (Newman-Keuls’ ps < .01; see Figure II.B.2).

Since the subject groups differed for non-verbal IQ, the relationship between this variable and the dependent variables of the same-different task was evaluated by Spearman rank correlations. All of these correlations were low and not significant (min. \(r = .01, p < .94\), n.s.; max. \(r = .18, p < .24\), n.s.) indicating that non-verbal IQ was not a confound for the present psychoacoustic results.
Compared to the controls, the dyslexic group showed poorer performance on both FT conditions. However, closer inspection of the percent correct values in the three shortest ISIs (8, 15, and 30 ms) revealed that some of the dyslexic subjects showed superior performance in the temporally extended versus rapid FT condition. Therefore, the dyslexic subjects were classified into two groups based on the mean difference across the three shortest ISIs between the extended (ex) and rapid (ra) FT conditions:

$$\text{mean } \Delta_{\text{ex-ra}} = \frac{(\text{ISI } 8+15+30)/3}{\text{ex}} - \frac{(\text{ISI } 8+15+30)/3}{\text{ra}}$$

Dyslexic subjects having difference values > 0 were subclassified as ‘benefiters’ (n = 8), while those with difference values ≤ 0 were classified as ‘non-benefiters’ (n = 14).

Figure II.B.3 illustrates the performance pattern of the benefiters, non-benefiters, and control subjects in the rapid and extended FT conditions across the ISIs tested. Since we are interested in whether different perceptual abilities in people with dyslexia are mirrored in distinct neuronal activation patterns, statistical analysis of the MMF data (see below) was based on this classification.41

**Neurophysiological experiment: Mismatch field**

Standard and deviant waveforms were derived in each of the four blocks (see Table II.B.3) and in all of the participants. Figure II.B.4 illustrates the whole-head MEG responses

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40 Brief stimuli presented in rapid succession are particularly demanding and thus are more suitable in discriminating between the groups.

41 The two dyslexic groups did not differ significantly in terms of age and the psychometric measures employed (for all Fs p > .05).
to the standard and deviant syllables with rapid FTs (within-category condition) in one dyslexic subject of the non-benefiter group. In the enlarged representation for one of the anterior channels, the difference between deviant and standard responses becomes evident.

**Figure II.B.4.** Top: Whole-head view of averaged waveforms in response to the standard rapid FT-syllable [ba]\(\text{t}\) (green curve) and the deviant rapid FT [ba]\(\text{t}\) (red curve) in an individual dyslexic subject (9 years) of the non-benefiter group. The measurement helmet, viewed from above, is flattened onto the plane with the nose pointing upwards. Bottom: Enlarged representation of the two waveforms for one of the left anterior channels. The responses are depicted from −100 before to 700 ms after stimulus onset.
Statistical analysis was performed on the mean amplitudes of the MMF (RMS values) by using mixed-model ANOVA with Category (within vs. across), FT (rapid vs. extended), and Hemisphere (left vs. right) as within-subjects factors and Group (benefiter, non-benefiter, vs. control) as between-subjects factor. The ANOVA yielded three significant interaction effects: Group × Category \([F(2,40) = 4.5, p < .02]\), Group × Hemisphere \([F(2,40) = 3.8, p < .03]\), and Group × FT × Hemisphere \([F(2,40) = 5.6, p < .007]\). No other sources of variance reached significance. The interaction effects were examined in detail using planned comparisons analyses.

**Figure II.B.5.** Mean MMF amplitudes (RMS values) common to the controls (open circles), benefiters (filled triangles), and non-benefiters (open triangles) for the two category conditions.

*Ad Group × Category.* Figure II.B.5 depicts the mean MMF amplitudes in response to within- and cross-category contrasts in the three subject groups. In the within-category condition, dyslexic subjects comprising the benefiter group exhibited significantly larger mean MMF amplitudes than the controls did \([60.83 \pm 6.66 \text{ fT vs. } 43.53 \pm 2.60 \text{ fT, respectively; } F(1,40) = 9.6, p < .004]\). Corresponding responses of the non-benefiters \((52.29 \pm 3.27 \text{ fT})\) were in between the RMS values of the benefiter and control groups. There were no significant group differences in the MMF magnitude in the across-categories condition: mean values came to \(48.77 \pm 3.74 \text{ fT}\) in controls, \(53.30 \pm 6.46 \text{ fT}\) in benefiters, and \(46.04 \pm 2.86 \text{ fT}\) in non-benefiters.
Ad Group × Hemisphere. Figure II.B.6 illustrates the mean MMF amplitudes over the left and right hemispheres across the category and FT conditions for the three participant groups. Both dyslexic groups were comparable to control subjects in terms of the left-hemispheric RMS values. While there was no significant interhemispheric difference in MMF amplitudes in either the non-benefiters (left: 49.48 ± 3.18 fT; right: 48.85 ± 3.17 fT) or the controls (left: 48.80 ± 3.37 fT; right: 43.51 ± 3.08 fT), the benefiter group displayed significantly stronger MMF responses over the right hemisphere (61.66 ± 7.54 fT) than over the left [52.47 ± 4.53 fT; \( F(1,40) = 4.2, p < .05 \)]. The enhanced right-hemispheric mismatch activity in the benefiter group was statistically different from those of the control group [\( F(1,40) = 8.5, p < .006 \)].

Ad Group × FT × Hemisphere. Figure II.B.7 shows the mean MMF amplitudes over both hemispheres in response to rapid and extended FT contrasts for the three subject groups. In the control group, the left-hemispheric MMF amplitude was significantly larger in the extended (52.55 ± 3.92 fT) than in the rapid (45.05 ± 3.21 fT) FT condition [\( F(1,40) = 5.8, p < .02 \)]; no FT-associated amplitude change was found in the right hemisphere (43.59 ± 4.02 fT and 43.42 ± 2.74 fT for the rapid and extended FTs, respectively). Furthermore, the controls’ mismatch responses to the temporally lengthened contrasts were significantly attenuated in the right hemisphere relative to the left [\( F(1,40) = 8.6, p < .006 \)].
Both the benefiter and non-benefiter groups showed similar left-hemispheric responses to either FT contrast: in the benefiter group the rapid and extended FT values were 52.84 ± 4.57 fT and 52.11 ± 5.66 fT, respectively; homologous values of the non-benefiter group came to 50.44 ± 3.88 fT and 48.52 ± 4.10 fT. In non-benefiter, almost equally-level responses to rapid (49.88 ± 2.32 fT) and extended (47.82 ± 4.97 fT) stimuli were also true for the right hemisphere. Unlike control subjects, benefiter showed the extended (69.17 ± 8.83 fT) versus rapid (54.15 ± 7.76 fT) FT-associated MMF increase in the right hemisphere \(F(1,40) = 7.0, p < .01\); their responses to the extended FT stimuli were significantly larger than those of the non-benefiter \(F(1,40) = 7.7, p < .008\) and control subjects \(F(1,40) = 12.8, p < .001\). In addition, the benefiter’s MMF to the lengthened FT contrasts was significantly stronger in the right compared to the left hemisphere \(F(1,40) = 11.4, p < .002\). Finally, no significant differences were found in mean amplitude of the right MMF in the non-benefiter group compared to the control response in any FT condition.

To further illustrate the neuronal activity patterns of the three participant groups, MMF responses to rapid and extended FT contrasts were separately plotted for either category condition (Figure II.B.8). For clarity, respective group mean RMS values ± SEMs are summarized in Table II.B.4.
Figure II.B.8. Mean MMF amplitudes (RMS values) over both hemispheres common to the controls (open circles), benefactors (filled triangles), and non-benefactors (open triangles) in response to rapid and extended FT contrasts of (a) the within-category condition ([ba]1-[ba]4) and (b) the across-categories condition ([ba]1-[da]8). For clarity, group mean RMS values ± SEMs are summarized in Table II.B.4.
Table II.B.4: Mean MMF amplitudes (RMS values, fT) for the three participant groups (mean ± SEM)

<table>
<thead>
<tr>
<th></th>
<th>Within category [ba:]-[ba:]</th>
<th>Across categories [ba:]-[da:]</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rapid FT</td>
<td>Extended FT</td>
</tr>
<tr>
<td><strong>Left</strong></td>
<td>43.95 ± 3.22</td>
<td>46.56 ± 3.48</td>
</tr>
<tr>
<td><strong>Right Hemi.</strong></td>
<td>42.07 ± 3.73</td>
<td>41.54 ± 3.57</td>
</tr>
<tr>
<td><strong>Control Ss (n=21)</strong></td>
<td><img src="image" alt="Control Ss" /></td>
<td><img src="image" alt="Control Ss" /></td>
</tr>
<tr>
<td><strong>Left</strong></td>
<td>57.17 ± 4.74</td>
<td>56.99 ± 7.62</td>
</tr>
<tr>
<td><strong>Right Hemi.</strong></td>
<td>57.67 ± 8.14</td>
<td>71.50 ± 14.44</td>
</tr>
<tr>
<td><strong>Benefiters (n=8 dyslexic Ss)</strong></td>
<td><img src="image" alt="Benefiters" /></td>
<td><img src="image" alt="Benefiters" /></td>
</tr>
<tr>
<td><strong>Left</strong></td>
<td>54.73 ± 5.75</td>
<td>53.17 ± 5.91</td>
</tr>
<tr>
<td><strong>Right Hemi.</strong></td>
<td>56.23 ± 3.21</td>
<td>45.03 ± 5.38</td>
</tr>
<tr>
<td><strong>Non-benefiters (n=14 dyslexic Ss)</strong></td>
<td><img src="image" alt="Non-benefiters" /></td>
<td><img src="image" alt="Non-benefiters" /></td>
</tr>
</tbody>
</table>

*Note. Hemi. = hemisphere.*

As shown in Figure II.B.8, both dyslexic groups exhibited similar left-hemispheric MMF responses to rapid and extended FT contrasts in either category condition. In the control group, the larger amplitude to extended relative to rapid syllables over the left hemisphere is mainly due to the cross-category contrasts. The benefiters, on the other hand, displayed their right-hemispheric extended *versus* rapid FT-associated MMF increase in both category conditions. Thus, the right-lateralized activation pattern in the benefiter group (Figure II.B.6) is principally accounted for by the elevated MMF amplitudes to lengthened within-category as well as cross-category contrasts. In non-benefiters, similar MMF responses to both rapid and extended FT changes over the right hemisphere resulted from two opposed activity patterns: in the across-categories condition the amplitudes slightly increased following stimulation with extended FT exemplars (Figure II.B.8b), whereas in the within-category condition a weaker activity to the extended compared to the rapid syllables was visible (Figure II.B.8a). The latter response pattern constitutes the ‘mirror image’ of the one observed in the benefiter group with the axis of symmetry passing through the right-hemispheric RMS means of the rapid FT condition. This (right-hemisphere) ‘mirror image’ configuration jointly contributed to the result that benefiters, but not non-benefiters, exhibited significantly stronger MMF amplitudes in the within-category condition than control subjects did (see Figure II.B.5). Furthermore, the separate illustration of the Group × FT × Hemisphere interaction suggests differences in rapid within-category contrasts: both dyslexic groups
displayed enhanced mean MMF amplitudes relative to control subjects in either hemisphere. However, in the across-category condition the RMS values to rapid syllable changes are lower and closer to the control means resulting in a leveling out of the differences in the Group × FT × Hemisphere interaction.

In a last step the relationship between non-verbal IQ and mean amplitudes of the MMF in each block was evaluated by Spearman rank correlations. None of these correlations was high and/or statistically significant (min. $r = -0.09, p < .58$, n.s.; max. $r = 0.29, p < .06$, n.s.) suggesting that the MMF data were not confounded by non-verbal IQ.

**Discussion**

The current study presents the examination of various behavioral and neuronal aspects of language dysfunction in dyslexia. **Discrimination performance** on the stop-consonant syllables [ba:] and [da:] revealed two subgroups of dyslexic individuals: one group displaying the limited rapid discrimination *versus* ameliorated extended FT syllable discrimination ability that has been observed in children with specific language impairment (Tallal & Piercy, 1974, 1975) and the other group showing a balanced performance profile which was quite similar to those of the normally literate controls (Figure II.B.3). The former group of dyslexic subjects was designated benefiters (n = 8) and the latter non-benefiters (n = 14). Thus, 36% of the dyslexic subjects did benefit from extended FT syllables in a behavioral discrimination task.

**Neuronal processing** of rapid- and extended-transition stimuli that differed either across the speech categories [ba:] and [da:] or within the category boundaries of a single syllable [ba:] was compared between the dyslexic subgroups and the control subjects. Results indicate that the benefiters exhibited significantly stronger mismatch activity to within-category contrasts than the controls did (Figure II.B.5). This is consistent with findings from categorical perception studies showing enhanced discrimination of stop-consonant contrasts constituting the same phoneme category in children with dyslexia (Godfrey et al., 1981; Werker & Tees, 1987; Serniclaes et al., 2001). However, unlike in the behavioral studies which also revealed limited cross-category perception of stop-consonant syllables, no significant group differences in terms of MMF magnitude to the [ba:]-[da:] contrasts were evident. That is, at the pre-attentive neuronal level, even the benefiters can discriminate between the phonemic contrasts. However, their neuronal discrimination ability might not be integrated into a voluntary response to rapid (cross-category) syllables.
In any subject group, the MMF did not differ significantly between the category conditions indicating similar discrimination both across and within phoneme boundaries. This result is in line with some electrophysiological studies in healthy adults in which the MMN to stop-consonant syllables was found to depend more on acoustic changes than on phonemic contrasts (Sams et al., 1990; Sharma et al., 1993; Maiste et al., 1995; Dalebout & Stack, 1999). Other researchers presented stronger MMNs to cross-category than to within-category syllable changes (Dehaene-Lambertz, 1997; Sharma & Dorman, 1999). Here, the reversed response pattern is suggested for the dyslexic subjects, especially of the benefiter type (Figure II.B.5). However, intersubject variability was particularly large in the benefiter group which might have weakened the results.

Dyslexic subjects comprising the benefiter group demonstrated significantly larger MMF amplitudes over the right hemisphere than over the left (Figure II.B.6). This right-lateralized MMF was mainly due to enhanced responses to extended FT contrasts both in the within- and across-category condition (Figures II.B.7 & 8). It thus matches their performance profile in the same-different task. Non-benefitters showed no amplitude increase to extended FT syllables in either hemisphere (Figures II.B.7 & 8) which also corresponds to their behavioral discrimination pattern. On the contrary, their right-hemispheric amplitudes to lengthened within-category syllables decreased and approached the MMF magnitude of the control group. Regarding the within-category condition, the two dyslexic groups exhibited stronger mismatch responses to rapid FT contrasts than the controls did. As mentioned above, this neuronal activity supports the categorical perception findings by other researchers who reported elevated discrimination of within-category stop-consonant syllables in dyslexia (Godfrey et al., 1981; Werker & Tees, 1987; Serniclaes et al., 2001). One may speculate that the neuronal representations of phonetic stimuli belonging to the same category are spatially more distant in dyslexic subjects than in normal controls. It remains unclear, however, why the non-benefitters showed diminished right-hemisphere responses when exposed to the temporally lengthened analogues. Future MMF experiments mapping the phonological feature space42 in dyslexic and normally literate individuals might shed light on these results.

The control subjects displayed significantly larger MMF amplitudes to extended versus rapid FT contrasts over the left hemisphere. This effect was driven predominantly by the cross-category changes (Figures II.B.7 & 8). In the behavioral discrimination task, the control group excelled in the rapid as well as extended FT condition. While the present

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42 For methodological elaboration on this issue the reader is referred to Diesch et al. (1996) and Eulitz et al. (2001).
performance data are consistent with reports of other authors (Tallal & Piercy, 1974, 1975; Bradlow et al., 1999). Bradlow et al. (1999) found no FT-related MMN change in their normally learning control group. However, the amplitude difference to rapid and extended syllable contrasts in our control subjects was less pronounced than the one in the benefiter group (Figure II.B.7). Possibly, the multi-channel whole-head MEG technique may be more sensitive in detecting subtle neuronal processing differences than the limited number of scalp electrodes used by Bradlow and colleagues.

Let us return to the MMF increase which occurred in response to temporally lengthened syllable contrasts in both the controls and the dyslexic subjects of the benefiter type. While the control group displayed the FT-associated amplitude change over the left hemisphere, the benefiter group showed it over the right (Figure II.B.7). The MMF increase was more prominent in the benefiter group whose right-hemispheric amplitudes to rapid FT contrasts were as strong as the controls’ left-hemispheric responses in the extended FT condition. The cortical activity data point to a left-hemisphere deviance in the benefiter group – a finding which was also suggested by dynamic neuroimaging investigations (see under ‘Functional neuroimaging’, chapter I) as well as by our earlier MEG study (see study A, this chapter) in dyslexia. To compensate for this functional impairment, the right cerebral hemisphere of the benefiter may get involved more intensely during the processing of speech sounds. However, the compensatory mechanism seems to be less successful in integrating the pre-attentive neuronal activity into a voluntary behavioral response to rapid-transition contrasts. Several studies in animals and humans have demonstrated that central nervous system mechanisms can be modified by experience (for surveys see Buonomano & Merzenich, 1998; Gilbert, 1998; Elbert et al., 2001). Experience-related changes in sensory cortex appear in response to a heavy training schedule realized in a behaviorally relevant context (e.g., Elbert et al., 2001; Elbert & Heim, 2001). Based on the principles of neuronal plasticity, Tallal and Merzenich developed a training program which yielded considerable improvements in processing rapid transitional elements of speech in children with language-based learning impairments (Merzenich et al., 1996; Tallal et al., 1996). Comparable evidence was found by Habib et al. (1999) in children with dyslexia. It would be interesting to examine whether such training would alter the magneto-cortical activity in the benefiter group.

To conclude, our MMF results are considered once again in light of the MMN findings of Bradlow et al. (1999). Consistent with previous event-related potential studies in language-based learning disorders (Kraus et al., 1996; Schulte-Körne et al., 1998a, 2001a), Bradlow and colleagues reported an attenuated MMN to short-transition syllable contrasts (viz., /da/-
In children with learning problems. In the present work, this has not been observed even in the dyslexic subgroup who may be expected to be impaired regarding their behavioral data. Variations in subject characteristics, stimulus and measurement techniques (multi-channel whole-head MEG vs. sparse-array electroencephalography) across the studies may well account for the different results.

To obtain additional information various elements of the present paradigm could be altered. Ideally, the behavioral data set would have included both across- and within-category syllables delivered in a categorical perception task. For practical reasons, we revert to our data of the same-different task modeled on the one developed by Tallal and Piercy (1974, 1975). This discrimination task involved short- and long-transition syllables that represented ideal exemplars of the [b] and [d] categories and was originally implemented to test the hypothesis of an auditory temporal processing deficit in dyslexia (see e.g., Tallal et al., 1993). Far more informative would be the determination of within- and cross-category discrimination thresholds for each individual subject along rapid and extended FT stop-consonant continua. The individually measured just-noticeable different within- and across-category stimuli should then be presented in an MMF paradigm. This experimental design is planned in a future study.

In summary, the present data suggest two dyslexic subgroups differing in their discrimination ability of short and long transitional stop-consonant syllables both at the behavioral and neuronal level. MMF data indicate that the cortical activity profiles to within- and cross-category contrasts varied across the dyslexic and control groups. The significance of these results should be examined in more detail using a larger group of children and adolescents.
Summary of study B: The present work examined various behavioral and neuronal aspects of speech sound processing in 22 dyslexic subjects and 21 normally literate controls aged 8 to 17 years. Discrimination performance on the stop consonant-vowel syllables [ba] and [da] revealed two subgroups of dyslexic individuals: one group displaying impaired performance on short transitional compared to temporally lengthened syllables and the other group showing a balanced performance profile which was quite similar to those of the normally literate controls. The former group of dyslexic subjects was designated benefiter (36%) and the latter non-benefiter (64%). Neuronal processing of rapid- and extended-transition stimuli that differed either across the speech categories [ba] and [da] or within the category boundaries of a single syllable [ba] was compared between the dyslexic subgroups and the control subjects. The mismatch field (MMF) was recorded simultaneously from both hemispheres by means of whole-head magnetoencephalography. Results indicate that the dyslexic subjects of the benefiter type exhibited significantly stronger mismatch activity to within-category contrasts relative to the controls. This is consistent with reports from categorical perception studies. Furthermore, the benefiter group demonstrated significantly larger MMF amplitudes over the right hemisphere than over the left. This right-lateralized MMF was mainly due to enhanced responses to long transitional contrasts both in the within- and across-category condition. It thus matches their performance profile in the syllable discrimination task. Non-benefiter showed no amplitude increase to temporally extended stimuli in either hemisphere which also corresponds to their behavioral discrimination pattern. The control subjects displayed significantly larger MMF amplitudes to extended versus rapid contrasts over the left hemisphere. The cortical activity data point to a left-hemisphere deviance in the benefiter group – a finding which was also suggested by dynamic neuroimaging investigations and our earlier MEG study in dyslexia. To compensate for this functional impairment, the right cerebral hemisphere of the benefiter may get involved more intensely during processing of speech sounds. However, the compensation mechanism seems to be less successful in integrating the pre-attentive neuronal activity into a voluntary behavioral response to rapid-transition contrasts. It would thus be interesting to examine whether training with acoustic-modified speech would alter the magneto-cortical activity in the benefiter group. In conclusion, the present data suggest two dyslexic subgroups differing in their discrimination ability of short and long transitional stop consonant-vowel syllables both at the behavioral and neuronal level.
STUDY C: ALTERED HEMISPHERIC ASYMMETRY OF AUDITORY P100M IN CHILDREN AND ADOLESCENTS WITH DYSELAXIA

Introduction

Investigation of the neural basis of dyslexia has suggested interhemispheric deviances in the organization of the perisylvian region (see under ‘Neurobiological correlates of dyslexia’, chapter I). Functional neuroimaging studies involving the visual presentation of language tasks have shown typical or enhanced activity in left-hemisphere frontal-lobe language sites, but reduced or absent activity in left temporo-parietal language areas in people with dyslexia (Paulesu et al., 1996; Rumsey et al., 1997a; Shaywitz et al., 1998; Brunswick et al., 1999; Temple et al., 2001). By contrast, McCrory et al. (2000) reported right-hemisphere differences during auditory word repetition: the dyslexic individuals demonstrated less hemodynamic activation than normal readers in the right superior temporal and right post-central gyri. Regarding findings of an atypical brain-activation profile observed either in the left hemisphere or the right, McCrory and colleagues proposed that the neural manifestation of dyslexia is task-specific, i.e., functional rather than structural in nature.

In a magnetoencephalographic (MEG) study (Heim et al., 2000c; see also study A, this chapter), we observed pronounced deviations in the organization of the left-hemisphere auditory cortex in children with dyslexia. The source location of the M210 passively elicited by speech and non-speech stimuli was found to be shifted away from the planum temporale towards anterior regions and probably into the superior temporal sulcus. The dyslexic group did not differ from the control group in the earlier magnetic field (M80) whose center of activity might be tied to the Heschl’s gyrus. Another MEG investigation of our research team (study B, this chapter) indicates differences in the hemispheric mismatch activity pattern between children and adolescents with dyslexia and normally literate controls. While the normal control group displayed an increase in mismatch amplitude to temporally lengthened stop-consonant contrasts over the left hemisphere, a subgroup of the dyslexic individuals showed this effect over the right hemisphere. This finding was interpreted as being consistent with a left-hemispheric functional deviance coupled with a tendency towards compensation by predominantly right-hemisphere involvement for some children and adolescents with dyslexia.

43 A preliminary report on this study was presented at the meetings of the Society for Psychophysiological Research in the year 2001 (Heim et al., 2001b).
As revealed by the hemodynamic and MEG data mentioned above, it would be erroneous to suppose that a deviance in language processing will be consistently located to a single cerebral hemisphere. In study A, magnetic responses were solely recorded over the left supratemporal cortex. Hence, there is no information whether dyslexic individuals and normal readers may also differ in magnetic source configuration of the right hemisphere. In the present study, we aimed to investigate possible interhemispheric source differences of magnetic responses to stop-consonant syllables between children and adolescents with dyslexia and normally literate controls by means of whole-head MEG. Since cortical auditory evoked potentials exhibit lengthy development (Courchesne, 1978; Sharma et al., 1997; Ponton et al., 2000), we were also interested in evaluating the age-related changes of magnetic field parameters.

Methods

Subjects

We tested 14 children and adolescents diagnosed with developmental dyslexia (aged 8.6-15.9 years, 5 females) and 12 control subjects (aged 8.5-15.0 years, 5 females) who had no history of reading and writing impairment. Informed written consent was obtained from all participants as well as their parents before testing began. Subjects received shopping vouchers and/or cinema tickets for participating. All participants had German as a first language, all evidenced normal peripheral hearing and none was reported as having had a history of neurological disease, psychiatric disorder, or psychotropic medication. As assessed with the Edinburgh Handedness Inventory (Oldfield, 1971) one dyslexic subject was left-handed and one control subject was ambidextrous (laterality quotients = −90 and −10, respectively). Subject groups did not differ significantly on age ($t_{24} = 0.8$, $p > .05$, n.s.). Mean age was $12.92 \pm 2.29$ years and $12.27 \pm 2.00$ years for dyslexic and control groups, respectively. While all participants demonstrated at least normal non-verbal intelligence (IQ > 85), non-verbal IQ scores were significantly lower in the dyslexic than in the control group (see Table II.C.1).

Dyslexic participants were recruited by advertisement from among members of the German Dyslexia Association and by recommendations of the local educational counseling service (Schulpsychologische Beratungsstelle Konstanz, Baden-Württemberg). As summarized in Table II.C.1, analysis of the data from language-related tests (reading/phonological decoding, spelling, and verbal memory span) revealed impaired performance of the dyslexic relative to the control group.
| Table II.C.1: Psychometric data across subject groups (median) |
|-----------------------------------|-----------------|-----------------|--------|-------|
|                                  | Control Ss (n = 12) | Dyslexic Ss (n = 14) | U*    | p     |
| Non-verbal IQ                    | 118.38           | 97.38            | 24.5  | .002  |
| Standard word reading            |                  |                  |       |       |
| Errors (max. = 72)               | 0.00             | 7.00             | 1.5   | .001  |
| Time (s)                         | 62.50            | 98.00            | 13.5  | .001  |
| Standard passage reading         |                  |                  |       |       |
| Errors (max. = 261)              | 5.00             | 33.00            | 0     | .001  |
| Time (s)                         | 120.50           | 218.50           | 6     | .001  |
| Word reading                     |                  |                  |       |       |
| Points (max. = 300)              | 290.50           | 232.50           | 2     | .001  |
| Time (s)                         | 131.00           | 235.50           | 11    | .001  |
| Pseudoword reading               |                  |                  |       |       |
| Points (max. = 300)              | 267.00           | 168.50           | 0     | .001  |
| Time (s)                         | 200.00           | 282.50           | 29    | .005  |
| Standard spelling                |                  |                  |       |       |
| % errors                         | 10.00            | 70.00            | 3     | .001  |
| Digit span forwards              | 5.95             | 5.30             | 33.5  | .05b  |
| Digit span backwards             | 4.70             | 3.70             | 19.5  | .04b  |

*aMann-Whitney U-test; ^bstatistical comparison included data from 11 out of 14 dyslexic subjects. Non-verbal intelligence was estimated with Raven’s Standard Progressive Matrices (Heller et al., 1998). Oral reading was examined using word and passage reading of the Zürcher Lesetest (Linder & Grissemann, 1998). In addition, reading and phonological decoding skills were documented using non-standardized word- and pseudoword-reading tests (points system: 0 = no response or error, 1 = self-correction, 2 = complete/partial repetition of an item, 3 = correct response; full listings of the reading-test items are included in the Appendix). Depending on the subject’s grade, the Diagnostische Rechtschreibtest, DRT 2, 3, 4, and 5 (Müller, 1983a, 1997; Grund et al., 1994, 1995) or Westermann Rechtschreibtest, WRT 6+ (Rathenow et al., 1981) was administered to assess spelling abilities. Lacking German normative data for older children, we decided not to transform reading and spelling raw scores into %-ranks; instead absolute error scores and percentage of errors, respectively, are shown. Digit span forwards and backwards were determined using a one-up one-down staircase procedure; item presentation was auditory, recall was verbal (for further details see study F of this thesis).

Magnetoencephalographic measures

The experimental paradigm as well as the recording and averaging procedure of the MEG were as described in study B of this thesis. In the current study, only the responses to
the 40-ms formant transition syllable [ba:] presented as the standard stimulus in the across-categories condition (viz. [ba:]5-[da:]8) will be reported.

Data analysis. In the latency window between 50 and 300 ms from stimulus onset a sequence of two prominent waves was detectable in each participant: one wave with a peak latency between 78 and 139 ms and another peaked between 221 and 288 ms. Rounding mean latencies to tens and considering dipole orientation (see below), these components were labeled as P100m and N260m, respectively.

Source parameters of the magnetic components P100m and N260m were estimated with a single equivalent current dipole (ECD) in a spherical volume conductor using 34 channels separately over the left and right perisylvian sites (see Appendix). An ECD defined by the dipole moment, the orientation, and space coordinates was computed for each sample point by means of a least-squares fit. The location estimates of each ECD were specified with reference to a head-based Cartesian coordinate system. The origin of this coordinate system was set at the mid-point of the medial-lateral (y-) axis interconnecting the center points of the entrance to the auditory meatus of the two ears (positive towards the left ear). The posterior-anterior (x-) axis projecting from the origin to the nasion (positive towards the nasion) and the inferior-superior (z-) axis being perpendicular to the x-y plane (positive towards the vertex).

ECDs of the left and right hemispheres were selected around the root mean square (RMS) maximum of the P100m and N260m. They were accepted when they fulfilled the following criteria based on statistical and anatomical considerations: (a) goodness of fit > 90%, (b) confidence volume < 2000 mm$^3$, (c) RMS > 30 fT, (d) dipole moment > 3 nAm, (e) correct dipole orientation: P100m ECD pointed upwards, N260m ECD pointed downwards, (f) stability of spatial source coordinates (x, y, and z) over a few milliseconds, (g) distance of ECD to midsagittal plane > 2.5 cm, and (h) inferior-superior value 3 < z < 8 cm.

In order to avoid missing cells in the statistical analysis, the selection criteria were watered down for eight out of 26 subjects: In five dyslexic and two control subjects the goodness of fit criterion was violated for the right-hemispheric P100m ECD with values varying between 84 and 89%. In two of these subjects (one dyslexic and one control subject) the respective dipole moment and RMS values fell below the cut-off scores by ≈15%. In another (control) subject the distances of the right-hemispheric P100m and N260m ECDs to the midsagittal plane were < 2.5 cm (viz., y = −1.33 cm and −1.99 cm, respectively). Due to the sufficiently small confidence volumes (all < cut-off score) and the dipolar field distribution of the questionable dipole fits these ECDs were still acceptable.
Table II.C.2 presents the mean goodness of fit and confidence volume for the two components’ ECDs in each subject group. Groups did not differ statistically on either source parameter [goodness of fit: $F(1,24) = 2.3, p > .05, \text{n.s.}$; confidence volume: $F(1,24) = 0.9, p > .05, \text{n.s.}$]. The goodness of fit for the P100m ECD was better in the left than in the right hemisphere (97% vs. 94%) which was mainly due to the eight subjects displaying right-hemispheric fit values below the 90% criterion (see above). Furthermore, in the right hemisphere the ECD of the N260m accounted for on average 3% more of the measured field variance than the P100m ECD [$F(1,24) = 4.8, p < .04$]. The confidence volume was significantly smaller for the N260m ECD than for the dipole fit of the P100m [41.42 mm$^3$ vs. 219.46 mm$^3$; $F(1,24) = 11.6, p < .002$].

<table>
<thead>
<tr>
<th></th>
<th>Goodness of fit (% )</th>
<th>Confidence volume (mm$^3$)</th>
<th>Goodness of fit (% )</th>
<th>Confidence volume (mm$^3$)</th>
</tr>
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<tbody>
<tr>
<td><strong>Left hemisphere</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P100m</td>
<td>97.28 ± 1.00</td>
<td>169.40 ± 80.74</td>
<td>94.82 ± 1.00</td>
<td>338.00 ± 163.18</td>
</tr>
<tr>
<td>N260m</td>
<td>97.63 ± 1.00</td>
<td>65.93 ± 26.03</td>
<td>97.00 ± 1.00</td>
<td>45.64 ± 31.80</td>
</tr>
<tr>
<td><strong>Right hemisphere</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P100m</td>
<td>96.24 ± 1.00</td>
<td>141.84 ± 36.37</td>
<td>92.44 ± 1.00</td>
<td>228.61 ± 65.30</td>
</tr>
<tr>
<td>N260m</td>
<td>97.28 ± 1.00</td>
<td>27.16 ± 5.05</td>
<td>96.76 ± 0.00</td>
<td>26.97 ± 16.80</td>
</tr>
</tbody>
</table>

**Note.** For statistical details see text.

**Statistics**

The peak latencies and RMS values of the P100m and N260m were submitted to two-way analyses of variance (ANOVAs) with Group (dyslexic vs. control) as between-subjects factor and Hemisphere (left vs. right) as repeated measures factor. Three-way ANOVAs were conducted on the strengths (i.e., dipole moments) and locations ($x$, $y$, and $z$) of the ECDs with Component (P100m vs. N260m) and Hemisphere as within-subjects factors and Group as between-subjects factor. Significant results ($p < .05$) were followed by planned comparisons. Rank correlations between the chronological age and magneto-cortical responses were computed using Spearman’s coefficient. Since there was no significant correlation of non-verbal IQ with any of the neuromagnetic parameters analyses were not adjusted for non-
verbal intelligence (for n = 26: min Spearman’s $r = .03, p < .89, n.s.;$ max. Spearman’s $r = −.35, p < .08, n.s.$).

**Results**

Figure II.C.1 shows the grand average responses (RMS) to the syllable [ba:] of the control and dyslexic groups. In both subject groups, two main deflections, peaking at $\approx 100$ and $\approx 260$ ms post-onset of the syllable, were obvious in the responses over each hemisphere. As indicated by the contour maps (Figure II.C.2), the dipoles of the first peak were oriented superiorly, indexing positive polarity (P100m) and the dipoles of the second peak were oriented inferiorly, indexing negativity (N260m).

![Graph of Figure II.C.1](image)

**Figure II.C.1.** Grand mean RMS waveforms for the left and right hemispheres in all control (broken curves) and dyslexic (solid curves) subjects. Peaks are indicated as P100m and N260m.
control subject

P100m

N260m

dyslexic subject

P100m

N260m

Figure II.C.2. Isofield contour maps at the peak latencies of the P100m and N260m in an individual control and dyslexic subject. Solid isocontour lines indicate the outgoing, the dashed lines the ingoing magnetic field, and the thick solid line zero flux. The spacing of the contours is 10 fT. A1-A148 represent the channel numbers. Following the ‘right-hand rule’, the components’ sources in the left and right hemispheres are oriented as follows; the P100m dipoles pointed up (indexing positivity), the N260m dipoles pointed down (indexing negativity).

Latencies and field amplitudes. Table II.C.3 presents the latencies and RMS field amplitudes of P100m and N260m peaks for the dyslexic and control subjects. Two-way ANOVAs provided no significant group differences on the peak latencies of the two components. For the later peak N260m a significant hemisphere main effect was observed with shorter mean latencies in the left (260.70 ms) than in the right (267.88 ms) hemisphere \[ F(1,24) = 5.9, \ p < .02 \]. Mean field amplitudes of P100m and N260m did not differ
statistically between the dyslexic and control subjects. For both groups, significantly larger P100m amplitudes were recorded in the left (83.24 fT) compared to the right (65.63 fT) hemisphere \(F(1,24) = 12.7, p < .002\]. No effect of Hemisphere was observed for the later N260m peak.

<table>
<thead>
<tr>
<th></th>
<th>Left hemisphere</th>
<th></th>
<th>Right hemisphere</th>
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<tbody>
<tr>
<td></td>
<td>Peak latency</td>
<td>Peak amplitude RMS (fT)</td>
<td>Peak latency</td>
<td>Peak amplitude RMS (fT)</td>
</tr>
<tr>
<td><strong>P100m</strong></td>
<td>98.57 ± 2.90</td>
<td>89.94 ± 8.97</td>
<td>94.48 ± 3.62</td>
<td>66.73 ± 7.96</td>
</tr>
<tr>
<td><strong>N260m</strong></td>
<td>260.13 ± 5.50</td>
<td>126.65 ± 11.83</td>
<td>267.18 ± 4.16</td>
<td>124.27 ± 7.62</td>
</tr>
<tr>
<td><strong>Control Ss (n = 12)</strong></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td><strong>Dyslexic Ss (n = 14)</strong></td>
<td></td>
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</tr>
<tr>
<td><strong>P100m</strong></td>
<td>95.72 ± 2.43</td>
<td>76.54 ± 6.77</td>
<td>100.37 ± 4.40</td>
<td>64.53 ± 6.60</td>
</tr>
<tr>
<td><strong>N260m</strong></td>
<td>261.26 ± 4.03</td>
<td>140.33 ± 17.25</td>
<td>268.58 ± 3.78</td>
<td>139.75 ± 12.33</td>
</tr>
</tbody>
</table>

*Note.* For statistical results see text.

**Source strengths and coordinates.** Table II.C.4 summarizes the ECD strengths and coordinates of the P100m and N260m for the dyslexic and control subjects. A three-way ANOVA performed on dipole moment data revealed a significant main effect for the factor Component \(F(1,24) = 30.9, p < .001\]. As was to be expected by the proportions of the RMS peaks (see Table II.C.3), dipole moments of the N260m (27.63 nAm) were on average stronger than those of the earlier component P100m (15.72 nAm). Neither differences between subject groups nor any other effect reached statistical significance. When analyzing the dipole moments of each component separately in two-way ANOVAs, a significant main effect of Hemisphere was yielded for the P100m ECD \(F(1,24) = 5.1, p < .03\]. That is, the P100m source was on average stronger in the left (18.36 nAm) than in the right (13.08 nAm) hemisphere. As with the corresponding RMS field amplitudes, no interhemispheric differences were found for the N260m source strength.
Table II.C.4: The dipole moments and coordinates of P100m and N260m for the two subject groups (mean ± SEM)

<table>
<thead>
<tr>
<th></th>
<th>Left hemisphere</th>
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<th>Right hemisphere</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Dipole</td>
<td>x (cm)</td>
<td>y (cm)</td>
<td>z (cm)</td>
</tr>
<tr>
<td></td>
<td>moment (nAm)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P100m</td>
<td>19.76 ± 2.08</td>
<td>5.05 ± 0.15</td>
<td>5.74 ± 0.22</td>
<td>15.04 ± 4.27</td>
</tr>
<tr>
<td></td>
<td>25.17 ± 2.79</td>
<td>4.98 ± 0.16</td>
<td>5.21 ± 0.17</td>
<td>28.00 ± 4.85</td>
</tr>
<tr>
<td>N260m</td>
<td>5.05 ± 0.15</td>
<td>4.98 ± 0.16</td>
<td>5.21 ± 0.17</td>
<td>5.21 ± 0.17</td>
</tr>
</tbody>
</table>

Control Ss (n = 12)

Dyslexic Ss (n = 14)

<table>
<thead>
<tr>
<th></th>
<th>Dipole</th>
<th>x (cm)</th>
<th>y (cm)</th>
<th>z (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>moment (nAm)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P100m</td>
<td>16.95 ± 1.99</td>
<td>4.83 ± 0.23</td>
<td>5.93 ± 0.18</td>
<td>11.12 ± 1.30</td>
</tr>
<tr>
<td></td>
<td>32.94 ± 5.01</td>
<td>4.77 ± 0.24</td>
<td>5.38 ± 0.25</td>
<td>24.41 ± 3.08</td>
</tr>
<tr>
<td>N260m</td>
<td>5.93 ± 0.19</td>
<td>4.77 ± 0.24</td>
<td>5.38 ± 0.25</td>
<td>5.21 ± 0.17</td>
</tr>
</tbody>
</table>

Note. For statistical results see text.

A three-way ANOVA performed on the x-coordinates yielded significant main effects for the factors Component [$F(1,24) = 6.9, p < .01$] and Hemisphere [$F(1,24) = 9.1, p < .006$]. In addition, there were two significant interaction effects: Group × Hemisphere [$F(1,24) = 14.9, p < .001$] and Group × Component × Hemisphere [$F(1,24) = 7.7, p < .01$]. Figure II.C.3 depicts the three-way interaction which was then evaluated in detail using planned comparisons.

Figure II.C.3. Source locations of P100m and N260m along the posterior-anterior (x-) axis for the control subjects (open circles) and dyslexic subjects (filled triangles). The x-coordinates of the two components were averaged for each group within hemispheres.
Looking at the control group, a similar source configuration was observed in both hemispheres with the N260m source locations being significantly posterior to the P100m sources \([F(1,24) = 7.5, p < .01 \text{ and } F(1,24) = 14.8, p < .001 \text{ for left and right hemispheres, respectively}]. However, the P100m and N260m sources of the right hemisphere (mean \(x\)-values \(\text{right} = 2.23 \text{ cm and } 1.41 \text{ cm}, \text{ respectively}) were located on average more anterior than the corresponding sources of the left hemisphere [mean \(x\)-values \(\text{left} = 1.44 \text{ cm and } 0.87 \text{ cm}, \text{ respectively}; F(1,24) _{\text{P100m}} = 6.7, p < .02 \text{ and } F(1,24) _{\text{N260m}} = 6.4, p < .02). Thus, control subjects displayed a hemispheric source asymmetry for both components. Whereas in the dyslexic group the generators of the N260m followed the more anterior right (1.80 cm) than left (1.15 cm) localization pattern \([F(1,24) = 10.9, p < .003]\), source locations of the P100m were found to be rather symmetrical between hemispheres (mean \(x\)-values = 1.42 cm and 1.26 cm for left and right hemispheres, respectively). The absence of P100m source asymmetry in dyslexic subjects resulted in two significant effects in the right hemisphere: (1) a within-group difference for the N260m ECD located anterior to the P100m ECD [mean \(x\) difference = 0.54 cm; \(F(1,24) = 7.5, p < .01\] and (2) a between-group difference for the P100m ECD located \(\approx 1\) cm more anterior in controls than in dyslexic subjects \([F(1,24) = 6.6, p < .02\]. These right-hemisphere effects are further illustrated in Figure II.C.4 which shows individual subject data projected onto the sagittal plane (= \(x-z\) coordinates).

![Figure II.C.4](image-url)

**Figure II.C.4.** Individual subject data from 12 control (open circles) and 14 dyslexic (filled triangles) subjects for localizations of the P100m source relative to the source location of the N260m in each hemisphere. The abscissa represents the posterior-anterior (\(x\)-) axis, the ordinate the inferior-superior (\(z\)-) axis.
To account for a possible influence of neuroanatomical variability on the components’ ECD locations, P100m source localizations were normalized with respect to the N260m sources according to \( \Delta_{sc} (P100m_{sc} - N260m_{sc}) \), whereby SC = source coordinate on x- or z-axes. As Figure II.C.4 (right) shows, in the right hemisphere of 11 out of 14 dyslexic subjects (= 79%) the P100m source was located posterior to the N260m source (indexed by negative \( \Delta_x \) values). On the other hand, in 10 out of 12 control subjects (= 83%) right-hemispheric P100m generator locations were anterior to those of the right-hemispheric N260m (indexed by positive \( \Delta_x \) values). No such group-specific source configuration was evident in the left hemisphere (Figure II.C.4, left).

Analysis of the source coordinates along the medial-lateral (y-) axis revealed no statistically meaningful effects. Figure II.C.5 depicts the group mean y-values of P100m and N260m ECDs in each hemisphere.

![Figure II.C.5. Source locations of P100m and N260m along the medial-lateral (y-) axis for the control subjects (open circles) and dyslexic subjects (filled triangles). The y-coordinates of the two components were averaged for each group within hemispheres. Negative values denote coordinates of the right hemisphere.](image)

Generator loci along the z-axis (see Figure II.C.6) were found to be significantly influenced by the factor component for the sources of the N260m to be located more inferior than the sources of the P100m [mean z-coordinates = 5.27 cm and 5.77 cm, respectively; \( F(1,24) = 18.8, p < .001 \)]. Other effects failed to reach statistical significance.

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44 Statistical analysis was based on absolute y-values.
To test whether there are age-related changes in the magneto-cortical responses, correlations between age and both latencies and field amplitudes for the P100m and N260m peaks as well as their source strengths were computed. Table II.C.5 presents the correlational data for the whole sample (n = 26). None of the dipole moments or the peak RMS amplitudes was significantly correlated with subjects’ age. Significantly negative age correlations were found for the P100m latency of the left (r = −.77, p < .001) and right (r = −.43, p < .03) hemispheres. Whereas the age-by-left-hemisphere P100m latency correlation remained statistically significant within each subject group (r = −.76, p < .002 and r = −.79, p < .002 for dyslexics and controls, respectively), the smaller correlation with the respective right-hemispheric value approached statistical significance in the dyslexic subjects (r = −.52, p < .06, n.s.) but not in the control subjects (r = −.45, p < .14, n.s.).

Table II.C.5: Rank correlations between age and different parameters of P100m and N260m for the whole study sample (n = 26)

<table>
<thead>
<tr>
<th></th>
<th>Left hemisphere</th>
<th></th>
<th>Right hemisphere</th>
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<tbody>
<tr>
<td></td>
<td>P100m</td>
<td>N260m</td>
<td>P100m</td>
</tr>
<tr>
<td>Lat.</td>
<td>Amp.</td>
<td>q</td>
<td>Lat.</td>
</tr>
<tr>
<td>Age</td>
<td>−.77&lt;sup&gt;b&lt;/sup&gt;</td>
<td>−.13</td>
<td>−.01</td>
</tr>
<tr>
<td></td>
<td>−.43&lt;sup&gt;c&lt;/sup&gt;</td>
<td>−.10</td>
<td>.10</td>
</tr>
</tbody>
</table>
<sup>a</sup> in months, <sup>b</sup>p < .001, <sup>c</sup>p < .05.
Lat. = peak latency (ms), Amp. = peak field amplitude (RMS, fT), q = dipole moment (nAm).
Discussion

In a number of studies, deviances of hemispheric laterality in the organization of the perisylvian region in dyslexia have been suggested (see under ‘Neurobiological correlates of dyslexia’, chapter I). The current study further investigated such differences using auditory event-related fields (ERFs) to the syllable [ba:] in children and adolescents with dyslexia and normally literate controls. In all participants, two main deflections – P100m and N260m – were evident in the ERF from each hemisphere. The validity of the chosen components as dependent variables is briefly discussed in the following paragraph.

Auditory evoked potential peaks, frequently reported in school-age children, are the P85-120 and N200-250 (Courchesne, 1990; Korpilahti & Lang, 1994; Čeponienė et al., 1998, 2001). However, their equivalents among adult auditory event-related potentials (ERPs) are unclear (Čeponienė et al., 1998). An adult-like N1 peak cannot usually be obtained in children before the age of 9 years, although it can be seen more readily when stimulus presentation rate is reduced or multi-channel recording techniques are implemented (Bruneau et al., 1997; Čeponienė et al., 1998; Ponton et al., 2000). Likewise, the magnetic N1m has been shown to be delayed or absent in children at rapid stimulation rates and was more similar to the adult type at slower rates (Paetau et al., 1995; Rojas et al., 1998). Considering the dipole orientation of the present P100m to the syllable [ba:], this might be a correlate of the adult auditory P1/P1m. The speech stimulus might also be of sufficient duration to elicit a sustained field which is characterized by a negative polarity. It would thus be conceivable that the N260m corresponds to the sustained field found in adult samples (e.g., Eulitz et al., 1995; Diesch & Luce, 2000). Disregarding the polarity, the latencies of the P100m and N260m waves are reminiscent of the adult auditory components N1m and P2m. I will return to this issue when discussing the results of the source localizations of the two components.

The peak latencies and field amplitudes of the P100m and N260m did not differ statistically between the dyslexic and control subjects. While the latencies of the P100m were similar for both hemispheres, the N260m peaked significantly earlier over the left hemisphere than over the right. The left-hemispheric P100m amplitude was significantly larger than the corresponding amplitude of the right hemisphere. No interhemispheric difference on amplitude was observed for the later N260m. The field amplitude pattern of the two components was confirmed by the dipole moment data.

The laterality effect of the P100m is concordant with findings of auditory ERP studies suggesting a superiority of the left hemisphere for the perception of stop-consonant sounds (for surveys see Molfese & Betz, 1988; Simos et al., 1997). This superiority has been
attributed to lateralized phonetic decoding mechanisms (e.g., Studdert-Kennedy & Shankweiler, 1970) or to hemispheric dissociations in processing rapidly changing acoustic input (viz., the short formant transition period; see e.g., Tallal et al., 1993, 1998). However, the present experimental design does not permit a decision between the proposed mechanisms. For clarification of this controversy, manipulation of the formant transition duration of the stop-consonant syllables as well as rapid and slowed non-speech analogues would be required. Ongoing work from our laboratory addresses this experimental question both in children and adults with dyslexia. Whatever the underlying processes, the leftward lateralization of the P100m in terms of field amplitude and dipole moment was evident in both groups. Likewise, no group differences were found for the later component N260m which showed a left-hemisphere advantage in terms of latency. The N260m might reflect a faster processing of the vowel (viz., [a:]) in the left than the right cerebral hemisphere.

In order to explore possible age-related changes in latency, field amplitude, and dipole moment of the two components, correlational analyses were performed among these variables. For the whole sample, significantly negative age correlations were obtained for the P100m latency in the left ($r = -0.77$) and right ($r = -0.43$) hemispheres. The age-by-left-hemisphere correlation remained highly significant both in the dyslexic and control subjects, whereas the moderate right-hemisphere coefficients failed to reach significance in either group. In view of the number of tests run, the age-by-right-hemisphere P100m latency correlation obtained for the whole sample should only be considered a trend. Nevertheless, the results are in line with developmental auditory ERP studies in healthy individuals showing latency decreases on the components P1 and N1 up to about 18 years of age (Sharma et al., 1997; Ponton et al., 2000). Similarly, Paetau et al. (1995) presented data showing age-related latency decreases on the neuromagnetic P1m and N1m over both hemispheres in healthy children (0.3-15 years). Unfortunately, no statistical analyses of their data were reported. In the current study, an age-related amplitude reduction on the first auditory response as demonstrated by some ERP studies (Sharma et al., 1997; Ponton et al., 2000) has not been found. Furthermore, no statistical relationship between the participants’ age and P100m dipole moment was obtained. Paetau et al. (1995) reported stronger dipole moments of the magnetic waves P1m and N1m in children than in adults. Because Paetau and colleagues did not present source strength data as a function of age among the group of children, a direct comparison with our results is more difficult.

As for the second magnetic component N260m, no significant age correlations with any of the parameters were obtained. Likewise, Courchesne (1978) found no significant age-
related changes on the electric component P2 in healthy individuals 6-36 years of age. Ponton et al. (2000) observed no consistent age-associated P2 latency changes, but decreases in amplitude across mid-childhood to adulthood. Paetau et al. (1995) merely reported a reduced or absent magnetic P2m in children up to 12 years of age.

Taken together, a comparison of the present correlational data with findings from developmental auditory ERP/ERF studies appears to be difficult. The reasons are manifold and include for instance differences in age groups, subject characteristics, and stimulus material across the studies. Keeping in mind that our sample size was very small, we can conclude that the dyslexic and control subjects did not differ in maturation of central auditory system activity at least as measured by the latency, amplitude, and dipole moments of two magnetic components (viz., P100m and N260m). Changes in event-related cortical responses do not encompass a simple latency decrease due to the ongoing myelinization of nerve fibers or amplitude reduction due to synaptic loss. Instead, they reflect complex processes of sculpturing highly efficient neural networks (Courchesne, 1990). Thus, continued research on the auditory ERF structure in larger groups of dyslexic and normally literate individuals is necessary to confirm and fully understand the present results.

The observation that dyslexic and control subjects did not differ on latency, amplitude, and dipole moment data is consistent with our previous MEG finding (study A, this chapter; see also Heim et al., 2000c). In both the current study and our earlier one, atypical source locations were found in the dyslexic subjects. In the present control sample, the source configurations were similar for both hemispheres with N260m ECDs located significantly posterior to P100m ECDs (see Figure II.C.3). The P100m as well as N260m source locations of the right hemisphere were significantly more anterior than the left-hemisphere homologues. Consequently, the control subjects displayed a hemispheric source asymmetry for either component. This interhemispheric asymmetry is consistent with reports of a more anterior location for adult N1m in the right than in the left hemisphere (e.g., Elberling et al., 1982; Kaukoranta et al., 1987; Nakasato et al., 1995; Ohtomo et al., 1998).

The generators of the P100m and N260m indicate activity in the temporal bank of the sylvian fissure. The source configuration in the control group suggests that the N260m arises from depolarization of pyramidal neurones in the planum temporale, whereas the P100m corresponds to a center of activity in or close to Heschl’s gyrus. In a single-subject design, Lütkenhöner and Steinsträter (1998) localized the adult N1m to the planum temporale and the P2m to Heschl’s gyrus. Taking up the notion that the P100m and N260m might be correlates of the adult N1m and P2m, respectively, one may speculate that in children and adolescents
the magnetic responses were generated in quite the contrary perisylvian structures. Further research is needed in order to examine (i) whether the components P100m and N260m are equivalents of the adult N1m-P2m peaks, and if so, (ii) whether and at which age the generation of the components is ‘undertaken’ by the planum temporale and Heschl’s gyrus, respectively.

In the dyslexic group, the sources of N260m coincide with the typical more-anterior-right-than-left localization pattern, whereas generator loci of the P100m were found to be rather symmetrical between hemispheres (see Figure II.C.3). This absence of asymmetry reflected a striking deviance in the organization of the right hemisphere: the N260m source was located significantly anterior to the P100m source with the latter being positioned ≈1 cm posterior to the P100m ECD in the control group. Thus, in the dyslexic group the right-hemisphere P100m source appears to be outside of Heschl’s gyrus, i.e., probably in the planum temporale. The right-hemisphere deviance of the P100m ECD location might be associated with a premature shift to the planum temporale without however the N260m being shifted to the same extent to Heschl’s gyrus. Continued research in the area of magnetic source imaging in children and adults with dyslexia is necessary to test the validity of the present findings and their implications.

The deviance in the right cerebral hemisphere observed in the dyslexic subjects is in line with the PET findings by McCrory et al. (2000) showing reduced activation in several right-hemisphere regions in general and an area of the right secondary auditory cortex in particular in a group of adult dyslexics. Other functional neuroimaging studies of dyslexia reported atypical activation patterns in the left hemisphere (Paulesu et al., 1996; Rumsey et al., 1997a; Shaywitz et al., 1998; Brunswick et al., 1999; Temple et al., 2001). By the same token, our previous MEG study (Heim et al., 2000c; see also study A, this chapter) revealed deviations in the organization of the left-hemisphere auditory cortex in children with dyslexia. There, an unusual source location was detected for a later response (≈210 ms post-stimulus) relative to the earlier M80 to pure tones and consonant syllables. In the current study, on the other hand, a relative change in generator position of the earlier magnetic P100m to the later N260m was observed in the right perisylvian region. The findings of left- and right-hemisphere deviances by no means contradict each other but characterize the temporal dynamic of the brain processes in individuals with dyslexia. This is not surprising in the context of the notion of the brain as a highly dynamic system (e.g., Elbert et al., 2001).
Our findings, while preliminary, support altered hemispheric asymmetry in persons with dyslexia. Moreover, they emphasize the contribution of the right primary auditory cortex and adjacent regions to the neural correlates of developmental dyslexia.

**Summary of study C:** The present study examined possible interhemispheric source differences of magnetic responses to the synthetic syllable [ba synthetic] in the auditory cortex of 14 dyslexic subjects and 12 normally literate controls aged 8 to 15 years by means of whole-head magnetoencephalography. In all subjects, two main deflections – labeled P100m and N260m – were evident in the responses over each hemisphere. In the control group, a similar source configuration was observed in both hemispheres with the N260m source locations being posterior to the P100m sources. Both the P100m and N260m sources of the right hemisphere were located more anterior than the corresponding sources of the left hemisphere. Thus, control subjects displayed the characteristic hemispheric source asymmetry for both components. Whereas in the dyslexic group the generators of the N260m followed the more anterior right than left localization pattern, source locations of the P100m were found to be rather symmetrical between hemispheres. The absence of P100m source asymmetry in dyslexic subjects resulted in two effects in the right hemisphere: (i) a within-group between-hemispheres difference with the N260m source located anterior to the P100m source and (ii) a between-group difference for the P100m source which was located about 1 cm more anterior in controls than in dyslexic subjects. Our results suggest altered hemispheric asymmetry of auditory P100m due to an atypical organization of the right hemisphere in children and adolescents with dyslexia.
STUDY D: ALTERED HEMISPHERIC ASYMMETRY OF AUDITORY N100m IN ADULTS WITH DEVELOPMENTAL DYSLEXIA

Introduction

Structural and functional studies of the human brain have shown altered hemispheric asymmetry – particularly of temporal lobe structures – in people with developmental dyslexia (see under ‘Neurobiological correlates of dyslexia, chapter I). Magnetoencephalographic (MEG) data from our own laboratory complement these findings: In study C of this thesis (this chapter, see also Heim et al., 2001b), we found that children and adolescents with dyslexia displayed a hemispheric symmetry in the source location of the auditory P100m to the stop-consonant syllable [ba:]. The change in source asymmetry reflected an atypical organization of the right hemisphere.

In the current study, we aimed at examining whether the deviation of hemispheric laterality in the organization of the auditory cortex is a maturational phenomenon or rather a stable characteristic that is also evident in adults with dyslexia. We report the findings from an MEG experiment in which the syllable [ba:] was presented to adult dyslexic subjects and normally literate controls. The latencies, amplitudes, and source parameters of the magnetic wave around 100 ms after stimulus onset were compared between the subject groups. This wave was assigned differently in the adult sample than in the group of children and adolescents participating in study C; here the dipole orientation of this component indexed a negative polarity and is therefore referred to as the N100m.

Methods

Subjects

Ten developmentally dyslexic adults (mean age 38.21 ± 12.46 years, 4 females) and 10 normally literate control subjects (mean age 32.83 ± 13.18 years, 5 females), all native speakers of German, gave informed written consent to participate in this study. They received a small financial bonus for participating. Subject groups were comparable in age ($t_{18} = 0.9$, $p > .05$, n.s.) and intellectual capacity (see Table II.D.1). All subjects had finished the minimum of formal education (9 years in Germany). Handedness was assessed with the Edinburgh Handedness Inventory (Oldfield, 1971). Subjects with a laterality quotient (LQ) ≥ +70 were considered right-handers. Two of the dyslexic and control subjects were ambidextrous (−60 ≥ LQ ≤ +60). Dyslexic and control subjects were free of psychiatric
disorders requiring consultation (present or past) and any psychotropic medication; they had no history of neurological disease and evidenced normal hearing thresholds.

The dyslexic subjects were recruited by advertisement from among members of the German Dyslexia Association. They reported significant difficulties in school lessons including reading, spelling, writing, and learning foreign languages. As a group, the dyslexics still experienced discomfort on measures of reading, phonological decoding, and orthography (see Table II.D.1).

Table II.D.1: Psychometric data for study groups (median)

<table>
<thead>
<tr>
<th></th>
<th>Control Ss (n = 10)</th>
<th>Dyslexic Ss (n = 10)</th>
<th>$U^a$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-verbal intelligence</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Raw scores (max. = 60)</td>
<td>54</td>
<td>51.5</td>
<td>41</td>
<td>n.s.</td>
</tr>
<tr>
<td>Word reading</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Points (max. = 300)</td>
<td>300</td>
<td>286</td>
<td>1</td>
<td>.001</td>
</tr>
<tr>
<td>Time (s)</td>
<td>97.5</td>
<td>140</td>
<td>22.5</td>
<td>.04</td>
</tr>
<tr>
<td>Pseudoword reading</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Points (max. = 300)</td>
<td>277.5</td>
<td>189.5</td>
<td>0</td>
<td>.001</td>
</tr>
<tr>
<td>Time (s)</td>
<td>151.5</td>
<td>296.5</td>
<td>1</td>
<td>.001</td>
</tr>
<tr>
<td>Spelling ability</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Raw scores (max. = 40)</td>
<td>39</td>
<td>31</td>
<td>3.5</td>
<td>.001</td>
</tr>
</tbody>
</table>

*Mann-Whitney U-test; n.s., not significant (p > .05). Non-verbal intelligence was assessed with Raven’s Standard Progressive Matrices, SPM (Heller et al., 1998). Since the SPM provides only percentiles normalized for non-German-speaking adults from the age of 20 up, raw scores are presented. Reading and phonological decoding skills were documented using non-standardized word- and pseudoword-reading tests (points system: 0 = no response or error, 1 = self-correction, 2 = complete/partial repetition of an item, 3 = correct response; full listings of the reading-test items are included in the Appendix). At the time of conducting this study, no standardized spelling test was available for the adult population, in which participants are asked to write down single spoken words. Therefore, the most demanding spelling-assessment tool available for children – the Westermann Rechtschreibtest, WRT 6+ (Rathenow et al., 1981) – was administered.

Magnetoencephalographic measures

The experimental paradigm and magnetic recordings were as described in study B of this thesis (see this chapter). In the present study, only the responses to the 40-ms formant
transition [baː]₁ which served as the standard stimulus in the within-category condition (viz., [baː]₁-[baː]₄) were evaluated.⁴⁵

Data analysis. Artifact-free epochs of 100 ms pre-onset and 700 ms post-onset of the syllable [baː] were obtained using the procedure detailed in study B. The source of the neuronal activity was estimated by determining the equivalent current dipole (ECD) around the N100m root mean square (RMS) maximum using 34 channels separately over the left and right perisylvian regions (see study C, this chapter). The following constraints were placed on the dipole fits a priori: (a) goodness of fit > 90%, (b) confidence volume < 2000 mm³, (c) RMS > 30 fT, (d) dipole moment > 3 nAm, (e) ECD oriented downwards, (f) stability of spatial source coordinates (x, y, and z) over a few milliseconds, (g) distance of ECD to midsagittal plane > 2.5 cm, and (h) inferior-superior value > 3 and < 8 cm. In both subject groups the ECD model explained on average 97% (SEM = 1) of the measured field variance [F(1,18) = 0.2, p > .05, n.s.]. The average confidence volume of the dipole fits was 152.12 mm³ (SEM = 46.28) and 65.13 mm³ (SEM = 20.06) for dyslexic and control groups, respectively [F(1,18) = 3.0, p > .05, n.s.].

Statistics

Statistical analysis of group-specific hemispheric asymmetries was conducted on the latency, field amplitude, dipole moment, and the Cartesian source coordinates of the N100m using mixed-design ANOVA with Hemisphere (left vs. right) as a within-subjects factor and Group (dyslexic vs. control) as a between-subjects factor. Significant effects (p < .05) were followed by planned contrasts.

Results

Figure II.D.1 (left) illustrates a typical averaged neuromagnetic response (overlay of 148 channels) in an individual dyslexic subject. The peak at 120.86 ms post-onset of the syllable (= 139.86 ms minus 19 ms run time correction) corresponds to the component N100m. The isofield contour map (right) and the negative dipole values in x and z directions (bottom) indicate the correct orientation of the ECD with respect to the electric counterpart, the N100 (see e.g., Mäkelä et al., 1993).

⁴⁵ The short-formant transitional [baː]₁ prototype was chosen as the data were to be compared with magnetic-brain responses elicited by this same syllable in schizophrenia patients (see Heim et al., 2000b).
Figure II.D.1. **Left:** Averaged neuromagnetic response (n = 463) to the syllable [ba] in an individual dyslexic subject. Waveforms from all 148 channels are superimposed. The peak at 120.86 ms (≈ 139.86 ms minus 19 ms run time correction) after syllable onset corresponds to the component N100m. **Right:** The respective isofield contour map at the peak latency. Solid isocontour lines indicate the outgoing, the dashed lines the ingoing magnetic field, and the thick solid line zero flux. The spacing of the contours is 10 fT. A1-A148 represent the channel numbers. Following the ‘right-hand rule’, the dipole of each hemisphere points in a postero-inferior direction indicating the negativity of the component. **Bottom:** Dipole fit for the N100m in the left hemisphere at the peak latency. Note the negative dipole values in x and z directions (Qx and Qz, respectively).

*Latenity, field amplitude, and dipole moment of N100m.* There were no significant differences in the latency, field amplitude, or dipole moment of the N100m obtained over left and right temporal cortices for the two subject groups (see Table II.D.2). No significant between-group differences emerged on any of the dependent variables.
Table II.D.2: Latencies, field amplitudes, and dipole moments of N100m across subject groups (mean ± SEM)

<table>
<thead>
<tr>
<th></th>
<th>Control Ss (n = 10)</th>
<th></th>
<th>Dyslexic Ss (n = 10)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hemisphere</td>
<td></td>
<td></td>
<td>Hemisphere</td>
</tr>
<tr>
<td></td>
<td>Left</td>
<td>Right</td>
<td></td>
<td>Left</td>
</tr>
<tr>
<td>Latency (ms)</td>
<td>119.29 ± 2.76</td>
<td>120.09 ± 2.14</td>
<td>118.72 ± 3.81</td>
<td>117.93 ± 3.37</td>
</tr>
<tr>
<td>Field amplitude, RMS (fT)</td>
<td>76.95 ± 6.16</td>
<td>76.92 ± 10.13</td>
<td>64.15 ± 8.19</td>
<td>65.35 ± 7.62</td>
</tr>
<tr>
<td>Dipole moment (nAm)</td>
<td>15.73 ± 3.50</td>
<td>12.68 ± 1.77</td>
<td>13.96 ± 2.37</td>
<td>11.30 ± 1.60</td>
</tr>
</tbody>
</table>

Note. For all Fs(1,18) p > .05.

Source locations of N100m. Table II.D.3 presents the generator loci of the N100m in the head-based Cartesian coordinate system for the dyslexic and control subjects.

Table II.D.3: Source coordinates of N100m across subject groups (mean ± SEM)

<table>
<thead>
<tr>
<th></th>
<th>Control Ss (n = 10)</th>
<th></th>
<th>Dyslexic Ss (n = 10)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hemisphere</td>
<td></td>
<td></td>
<td>Hemisphere</td>
</tr>
<tr>
<td></td>
<td>Left</td>
<td>Right</td>
<td></td>
<td>Left</td>
</tr>
<tr>
<td>x (cm)</td>
<td>1.22 ± 0.39</td>
<td>2.00 ± 0.25</td>
<td>1.33 ± 0.21</td>
<td>1.27 ± 0.22</td>
</tr>
<tr>
<td>y (cm)</td>
<td>5.48 ± 0.39</td>
<td>−5.66 ± 0.21</td>
<td>5.20 ± 0.35</td>
<td>−5.53 ± 0.21</td>
</tr>
<tr>
<td>z (cm)</td>
<td>5.78 ± 0.24</td>
<td>5.74 ± 0.17</td>
<td>5.91 ± 0.24</td>
<td>6.15 ± 0.28</td>
</tr>
</tbody>
</table>

Note. For statistical particulars see text.

The ANOVA performed on the x-coordinates (anterior-posterior axis) revealed a significant main effect for the factor Hemisphere \([F(1,18) = 6.9, p < .02]\) and a significant Group × Hemisphere interaction \([F(1,18) = 9.6, p < .006]\). While the main effect is of minor importance, the interaction (Figure II.D.2) was considered in detail using planned comparisons analyses.
In the control group, the N100m source was located significantly more anterior in the right hemisphere (mean x-value = 2.00 cm) than in the left [mean x-value = 1.22 cm; F(1,18) = 16.3, p < .001]. By contrast, dyslexic subjects did not show hemispheric asymmetry in the location of the N100m ECD in the anterior-posterior direction (mean x-value of the right and left hemispheres = 1.27 cm and 1.33 cm, respectively). While there was no significant between-group difference in the center of activity over the left hemisphere, the dyslexic subjects’ N100m source of the right hemisphere was localized ≈0.70 cm posterior to the source in the control group [F(1,18) = 5.0, p < .04]. As illustrated in Figure II.D.3, this finding was not likely to have resulted from enhanced intersubject variability within the dyslexic group. On the contrary, the variability of the x-values seems to be no greater among the dyslexics than among the controls.

**Figure II.D.2.** Mean source locations of the N100m on the posterior-anterior (x-) axis in each hemisphere for the control (open circles) and dyslexic groups (filled triangles).

**Figure II.D.3.** Individual subject data from all controls and dyslexics for the N100m source localizations along the posterior-anterior (x-) axis in the left and right hemispheres.
Figure II.D.4 depicts the source locations of the N100m on the medial-lateral (y-) axis for the dyslexic and control subjects. The ANOVA run on the y-coordinates provided no significant effects. For both subject groups, the mean y-value was 5.34 cm in the left and –5.60 cm in the right hemisphere.

Analysis of the N100m source locations in the inferior-superior direction (z-axis) revealed no significant results. Across subject groups, mean z-coordinates of 5.85 and 5.95 cm were calculated for left and right hemispheres, respectively (Figure II.D.5).

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46 In each hemisphere, absolute y-coordinates were used for statistical analysis.
Discussion

The present study showed an atypical interhemispheric asymmetry in the positions of the N100m sources to the syllable [ba:] in adults with dyslexia. While in the normally literate control group the right N100m ECD was located more anterior than the corresponding ECD of the left hemisphere, the dyslexic group displayed a rather symmetrical source configuration between the hemispheres. This symmetry reflected a deviance in the right perisylvian cortex for the dyslexic subjects’ N100m generated more posterior than the response in adult controls (Figure II.D.6).

![Figure II.D.6](image_url)

**Figure II.D.6.** Mean source locations of the N100m for the control (blue rhombus) and dyslexic groups (red rhombus) superimposed on the standard magnetic resonance images (MRI) included with the BESA® Source Analysis Module (V.4.00, MEGIS Software GmbH, Gräfelfing, Germany). R and L denote right and left side of the brain, respectively (radiological convention). The combined MEG and MRI information suggests that the N100m is generated in the temporal bank of the sylvian fissure. The two subject groups did not differ in N100m dipole positions along the inferior-superior axis (coronal slice, left) and the medial-lateral axis (see also transversal slice, right). Regarding the anterior-posterior direction, atypical source configuration among dyslexic participants was apparent (transversal slice, right): In the control group, the N100m sources were found to be asymmetrical with a more anterior localization in the right than in the left hemisphere. The N100m dipoles in the dyslexic group did not exhibit the same interhemispheric asymmetry. While there was no significant between-group difference in the center of activity over the left hemisphere, the dyslexic subjects’ N100m source of the right hemisphere was positioned posterior to the control participants’ source.

A number of MEG studies demonstrated interhemispheric source asymmetry for the N100m response in healthy adults (e.g., Elberling et al., 1982; Reite et al., 1989; Nakasato et al., 1995; Ohtomo et al., 1998; Rockstroh et al., 2001). This observation is supported by the present event-related field (ERF) recordings on normally literate adults. The *planum temporale* has been considered one center of activity for the adult N100m (Lütkenhöner & Steinsträter, 1998; Ohtomo et al., 1998; Godey et al., 2001). N100m source asymmetry might
be concordant with anatomical data showing excesses of frontal and peri-rolandic cortex in the left hemisphere and of posterior parietal lobe in the right hemisphere (e.g., Rubens et al., 1976; Steinmetz et al., 1990b; Foundas et al., 1995, 2001; Binder et al., 1996). According to Binder et al. (1996), larger frontal and peri-rolandic mass on the left side pushes the point of upward deflection of the sylvian fissure posteriorly and tilts the planum temporale back. Conversely, larger parietal cortex on the right side might push the point of sylvian deflection anteriorly and tilt the planum forward.

The hemispheric balance in the source locations of the N100m found in our dyslexic group might agree with structural brain studies suggesting atypical asymmetry of the planum temporale in reading disabled individuals (Hynd et al., 1990; Larsen et al., 1990; Flowers, 1993). However, more recent studies have challenged the view of altered planar asymmetry in dyslexia (Leonard et al., 1993; Schultz et al., 1994; Rumsey et al., 1997b; Best & Demb, 1999; Heiervang et al., 2000; Robichon et al., 2000). Less N100m lateralization might also reflect altered neuronal morphology in temporal-plane sites of dyslexic individuals (see Galaburda, 1988, 1989, 1993). It is conceivable that the different morphology interfere with efficient auditory processing, and consequently other (right posterior) perisylvian regions become involved in this type of processing. These substituted regions may not perform the task as efficiently as a normally developed planum temporale would. Traditional views such as this one suggest that a structural deficit is the cause and functional deviance the consequence. In the course of neural plasticity studies of the human brain, the possibility has been acknowledged that functional alterations arising presumably from behavioral or environmental demands trigger morphological changes (see e.g., Elbert et al., 2001). Thus, a different location for the processing of syllables might alter neural morphology or even brain structure. Probably maturational and environmental factors interact to yield a given result. On the basis of the presently available data it is not possible to decide which of the processes might be the major player, however.

In study C (this chapter), we have speculated that the P100m detected in children and adolescents might correspond to the adult N100m. In order to clarify whether the juvenile P100m and adult N100m represent the same or different components, further ERF investigation using cross-sectional and longitudinal data from childhood to adulthood would be required. Herein, the continuous observation of the dipole orientation of the componentry would be indispensable. Nevertheless, the present findings are consistent with our previous study C revealing (i) a more anterior-right-than-left source location of the P100m to the syllable [ba:] in normally literate children and adolescents and (ii) an absence of hemispheric
source asymmetry in their dyslexic peers which was related to (iii) a difference in right perisylvian sites for the P100m dipole located more posterior in young dyslexic individuals than in controls. Consequently, reduced or absent lateralization in the source configuration of auditory ERFs around 100 ms after stimulus presentation appears to be a common feature observed in both children and adults with dyslexia. Moreover, less asymmetry has been associated with a deviance in the auditory cortex of the right hemisphere in both investigations. This finding might be causally related to dyslexia, but might also reflect a compensatory mechanism of a possible left-hemisphere dysfunction. The latter supposition has been implicated in at least a subgroup of dyslexic individuals using the mismatch paradigm (see study B, this chapter). One promising way to clarify this issue would be the implementation of training programs tapping different aspects of literacy skills. MEG recordings before and after the training regimen would then indicate to what extent a specific intervention method might be capable of altering cerebral lateralization in dyslexic individuals.

In summary, reduced or absent hemispheric asymmetry in posterior perisylvian regions appears to be a stable characteristic rather than a maturational phenomenon in dyslexia. In both children and adults with dyslexia the lateral source symmetry reflects an atypical organization in the right hemisphere.

**Summary of study D:** The current auditory event-related field study aimed at determining whether the deviance of hemispheric source asymmetry found in children and adolescents with dyslexia (see study C, this chapter) is a maturational phenomenon or rather a stable characteristic that is also evident in dyslexic adults. Ten adult dyslexic subjects and 10 normally literate controls were presented with the syllable [ba:] while the event-related magnetic activity was recorded from both hemispheres using whole-head magnetoencephalography. In control subjects, the auditory N100m source (corresponding to P100m in children?) was found to be asymmetrical with a more anterior localization in the right hemisphere than in the left. This typical source asymmetry was absent in dyslexic subjects. The results indicate that an absence of hemispheric laterality may reflect a common feature present both in children and adults with dyslexia.
STUDY E: AUDITORY TEMPORAL PROCESSING DEFICIT IN CHILDREN WITH DYSLEXIA IS ASSOCIATED WITH ENHANCED SENSITIVITY IN THE VISUAL MODALITY

Introduction

Psychoacoustic studies have shown that many children with language and literacy problems display limitations in reception of brief and rapidly changing phonemes (e.g., stop consonants) or rapidly successive sound inputs (cf. Tallal et al., 1993; Farmer & Klein, 1995) for which Paula Tallal has been using the term ‘temporal processing deficit’ (see under ‘Auditory temporal processing deficit’, chapter I). Experimental findings in the visual and tactile sensory modalities have led some researchers to conclude that a temporal processing deficit might be pansensory in children with language impairment and people with dyslexia (Tallal et al., 1993; Farmer & Klein, 1995). Because multimodal approaches within a single study have been sparse and inconclusive, the question of whether a temporal processing deficit in these populations might be general or modality-specific has been a subject of intense debate.

The present study aimed to investigate temporal processing abilities in both the auditory and visual modality in children with dyslexia and normally literate controls. The auditory task required a same-different judgment of two successively presented stop consonant-vowel (CV) syllables ([ba] and [da]) which are characterized by rapid frequency changes (formant transitions) that occur during the initial few tens of milliseconds. As a control condition, the same syllables were presented with temporally extended formant transitions (Tallal & Piercy, 1975). This control procedure should provide an evaluation of the auditory temporal processing hypothesis for dyslexia: A relative weakness in discriminating between CV syllables with rapidly changing compared to CV syllables with temporally extended formant transitions would support the view of a deficit in temporal acoustic processing; poor performance on both conditions would suggest that the difficulties experienced by the dyslexic children are speech-specific in general. In accordance with Tallal’s work, performance on the two conditions was measured at different interstimulus intervals (ISIs) varying between 8 and 305 ms (Tallal & Piercy, 1974, 1975). In the visual task, thresholds for the detection of temporal order of two light flashes were determined. The findings of the study should add valuable information to the issue of whether a temporal processing deficit in dyslexia is pansensory or modality-specific.

47 This part of the thesis largely corresponds to the paper of Heim et al. (2001a).
Methods

Subjects

Twenty-two children and adolescents (aged 11.1-15.4 years, 3 females) diagnosed with developmental dyslexia and 11 controls (aged 11.0-15.8 years, 1 female) with normal literacy skills were recruited from the Munich, Bavarian, area. All subjects were native speakers of German. The two groups were very similar on age and non-verbal intelligence (see Table II.E.1).

Table II.E.1: Psychological assessment data across subject groups (mean ± SD)

<table>
<thead>
<tr>
<th></th>
<th>Control Ss (n = 11)</th>
<th>Dyslexic Ss (n = 22)</th>
<th>t-test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>13.6 ± 1.6</td>
<td>13.2 ± 1.4</td>
<td>n.s.</td>
</tr>
<tr>
<td>Non-verbal IQ</td>
<td>106.4 ± 14.0</td>
<td>108.6 ± 11.0</td>
<td>n.s.</td>
</tr>
<tr>
<td><strong>Standard reading</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Errors (z-scores)</td>
<td>−0.9 ± 0.4</td>
<td>0.4 ± 0.9</td>
<td>( p &lt; .001 ) (^a)</td>
</tr>
<tr>
<td>Time (s)</td>
<td>125.0 ± 12.4</td>
<td>184.2 ± 41.6</td>
<td>( p &lt; .001 ) (^b)</td>
</tr>
<tr>
<td><strong>Pseudoword reading</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% errors</td>
<td>5.0 ± 2.5</td>
<td>16.0 ± 5.5</td>
<td>( p &lt; .001 ) (^a)</td>
</tr>
<tr>
<td><strong>Standard spelling</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Errors (z-scores)</td>
<td>−1.2 ± 0.6</td>
<td>0.6 ± 0.5</td>
<td>( p &lt; .001 )</td>
</tr>
</tbody>
</table>

\(^a\) t-test for unequal variances; \(^b\) statistical comparison is based on logarithmic data; n.s., not significant \((p > .05)\). Non-verbal intelligence was assessed with Raven’s Standard Progressive Matrices (Heller et al., 1998). Reading and phonological decoding skills were examined using ‘passage reading’ of the Zürcher Lesetest (Linder & Grissemann, 1998) and a non-standardized pseudoword-reading test (40 items; see Appendix), respectively. Depending on the child’s grade, the Diagnostische Rechtschreibtest, DRT 5 (Grund et al., 1995) or Westermann Rechtschreibtest, WRT 6+ (Rathenow et al., 1981) was administered to evaluate spelling abilities. Due to a lack of German normative data for older children, we decided not to transform reading and spelling raw scores into %-ranks; z-scores \((\text{mean} = 0, \text{SD} = 1)\) are therefore given.

Control subjects were selected from a private school. Sixteen children from the dyslexic group attended a special school for dyslexia, the other 6 having been referred by an independent institute that specializes in diagnosing and treating children with learning disabilities. Although the dyslexic subjects were receiving remedial training and intense
tutoring, they performed significantly poorer on measures of reading, phonological decoding and spelling as compared to the controls (see Table II.E.1). All subjects had normal hearing thresholds and normal or corrected-to-normal visual acuity. Informed written consent was obtained from the children and their parents. All children were rewarded with shopping vouchers or cinema tickets for their voluntary participation.

**Design and Procedure**

All subjects participated in two psychophysical tasks, which were carried out on an IBM compatible 486-Personal Computer. The order of the tasks was counterbalanced between subjects. Each child was tested individually in a quiet room providing by the schools or the institute.

**Auditory same-different task**

The German CV syllables [ba:] and [da:] were created with a sampling rate of 10 kHz in a cascade mode by using Speechlab software (Diesch, 1997) based on a Klatt cascade/parallel formant synthesizer (Klatt, 1980). The total stimulus duration was 250 ms including a formant transition (FT) period of either 45 ms (rapid FT condition) or 95 ms (extended FT condition). The syllables were composed of three formants (F1-F3) and differed in the onset frequencies of the second and third FT. The starting points of the second and third FT were 1095 Hz and 2100 Hz for the syllable [ba:] and 1702 Hz and 2633 Hz for [da:]. The fundamental frequency of each syllable started with a 5-ms delay at 128 Hz and decreased linearly to 109 Hz at stimulus offset. The steady-state formant frequencies of the vowel [a:] were 770, 1340, and 2400 Hz for F1, F2, and F3, respectively. The amplitude of voicing was constant at 54 dB and fell linearly to 11 dB during the last 25 ms of the stimulus.

The experimental procedure was identical for the rapid and extended FT conditions. The sequence of the conditions was counterbalanced across subjects; conditions were separated by a short break. The syllables were delivered to both ears via Sony MDR-CD470 headphones at ≈72 dB sound pressure level. The task required the child to press the right (green) key on the computer keyboard if two successive syllables were the same ([ba:]-[ba:], [da:]-[da:]) and the left (red) key if they were different ([ba:]-[da:], [da:]-[ba:]) by using the index finger of her/his right hand. Feedback was provided after each stimulus pair (= trial) by a happy or unhappy face on the computer screen. The intertrial interval was 2 s. The training phase consisted of a maximum of 72 trials (18 for each possible pair combination, randomly
intermixed) and finished earlier if a criterion of 30 correct responses in 36 consecutive trials was achieved. The task was terminated for subjects who did not meet the criterion. During training an ISI (defined as syllable offset to onset) of 428 ms was employed; in the immediately following testing phase syllables were presented at six different ISIs: 8, 15, 30, 60, 150, and 305 ms (Tallal and Piercy, 1974, 1975). Testing included 48 trials, eight (2 × 4 syllable-pair combinations) for each ISI, with a randomized presentation of the different intervals. The percentages of correct trials were measured at each ISI.

**Visual temporal-order task**

Equiluminant light flashes of green and red were generated by two light-emitting diodes (LEDs). LEDs were mounted side by side (separation 1 cm) on a black surface slanted toward the subject. The LED apparatus was positioned on a table (approx. 70 cm high) with a constant viewing distance of 40 cm. Subjects were asked to press either a red (left) or green (right) key on the computer keyboard with the index finger of their right hand indicating the LED which flashed first. After the second LED flashed, both LEDs remained on for 2 s. Two-element stimulus sequences (red-green, green-red) were randomized across trials. The intertrial interval was 5 s. In order to direct subject’s attention to the LEDs, each trial was announced by a brief tone. During an initial training phase of 10 trials, onsets between two light flashes (SOA, stimulus onset asynchrony) varied between 310 and 400 ms, i.e., rather long intervals, to allow all subjects to understand the task. In the testing phase (= 40 trials) the SOA was adjusted from trial to trial (starting SOA = 300 ms), using an adaptation of the staircase procedure (Cornsweet, 1962). After a correct response in a given staircase, the SOA was shortened, whereas an incorrect answer led to the SOA being lengthened. Sizes of downward or upward steps (= reversals) were 10% of the previous value; below a 10-ms SOA a step size of 1 ms was used. The visual temporal-order threshold in milliseconds, defined as the arithmetic mean of the last 20 reversals, was calculated by the computer program.

**Results**

Univariate analysis of variance (ANOVA) showed no significant group effect on visual temporal-order thresholds, indicating that both the dyslexic (mean threshold = 8.9 ms, SEM = 1.2) and control children (mean threshold = 9.9 ms, SEM = 1.3) managed the visual temporal processing task without difficulty and equally well.
Mixed-design ANOVA performed on the data of auditory processing revealed a significant group main effect \([F(1,31) = 4.2, p < .05]\) for the dyslexics to have lower scores than the controls in both FT conditions across the range of ISIs tested (Figure II.E.1).

Even though no significant interaction was obtained with the factor Group, there was a small tendency for larger group differences in the rapid than in the temporally extended FT condition. As demonstrated by other investigators, some people with dyslexia may have no difficulty in tasks involving rapidly changing acoustic stimuli (Tallal et al., 1993; Farmer & Klein, 1995). That might also apply to a subset of dyslexic children in the present study and in turn have weakened the Group \(\times\) Condition interaction. Therefore, the dyslexic subjects were subclassified into two groups based on their discrimination performance on the rapidly changing syllables in the three shortest ISIs (8, 15, and 30 ms). Dyslexic subjects scoring \(\geq 87.5\%\) in at least two of the shortest ISIs were classified as ‘good perceivers’, otherwise they were classified as ‘poor perceivers’. Mixed-design ANOVA carried out with these groups yielded a significant Group \(\times\) FT condition interaction effect \([F(2,30) = 6.8, p < .004]\).

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48 By definition, temporal processing is called upon in particular when brief stimuli are presented in rapid succession (Tallal et al., 1993; Farmer & Klein, 1995).
As illustrated in Figure II.E.2, the 14 dyslexic subjects comprising the group of good perceivers did not differ from the controls in any of the conditions. However, the poor-perceiver group (n = 8 dyslexic subjects) was significantly less accurate in the rapid FT condition compared to the other groups (Scheffé’s $p < .001$) and their performance level in the extended FT condition (Scheffé’s $p < .02$). This result suggests that at least 8 subjects of the dyslexic group had difficulty in processing rapid temporal acoustic information.

In order to avoid possible ceiling effects, we used the minimum correct percentages of each subject attained at any ISI of the rapid FT condition for analyzing the relationship between auditory and visual temporal processing. Individual subject data for minimum auditory scores in relation to visual temporal-order thresholds are presented in Figure II.E.3.
A bivariate prediction analysis based on weighted Kappa (Hildebrand et al., 1977) indicated a significant distribution pattern for the dyslexic group ($p < .004$). Ten dyslexic subjects whose performance in the same-different task pointed to limitations in auditory temporal processing [i.e., $\leq 75\%$ correct (Tallal & Piercy, 1973a)], demonstrated low temporal-order thresholds ($< 10\, ms$) in the visual sensory modality. In 5 of these children, visual thresholds were at least $1\, SD$ below the group mean of normal controls. In control subjects, the statistical relationship between measures on auditory and visual temporal processing did not reach significance.

**Discussion**

Our results reveal an auditory temporal processing deficit in children with dyslexia whereas temporal sensitivity was enhanced rather than impaired in the visual task. Thus, the current data provide no evidence for a pansensory or general temporal processing deficit in children with dyslexia. On the contrary, poor auditory temporal sensitivity might be compensated by a well-functioning visual sensory modality. Supportive evidence has been provided by a study of Talcott et al. (2000) in a sample of unselected elementary-school children: Auditory and visual temporal processing were found to be differently engaged in phonological and orthographic skills, implying independent candidates in determining a child’s ability to learn to read. Our findings support the view that intervention methods for dyslexia should target the auditory modality (Merzenich et al., 1996; Tallal et al., 1996).

**Summary of study E:** Developmental dyslexia has been associated with a deficit in temporal processing. Controversial is the question whether the postulated deficit is pansensory or limited to the auditory modality. We present psychophysical assessment data of auditory and visual temporal processing abilities in children with dyslexia (aged 11-15 years). While none of the dyslexic children displayed temporal processing abnormalities in the visual sensory modality, dyslexics with poor auditory temporal scores reached a high level of visual performance. Our results do not confirm the hypothesis of a general temporal processing deficit for dyslexia but suggest that limitations in auditory temporal processing might be compensated by a well-functioning visual sensory modality.
STUDY F: SYLLABIC TRAINING, LITERACY SKILLS, AND CORTICAL ORGANIZATION IN CHILDREN WITH SPECIFIC LANGUAGE IMPAIRMENT

Introduction

Psychoacoustic experiments have shown that many children with specific language impairment (SLI) or dyslexia are at least mildly impaired in their reception of stop consonants, such as /b/, /d/, /g/, and /k/ (e.g., Tallal & Piercy, 1974; Brandt & Rosen, 1980; Godfrey et al., 1981; Tallal & Stark, 1982; Werker & Tees, 1987; Elliott & Hammer, 1988; Elliott et al., 1989; Reed, 1989; Sussman, 1993; Stark & Heinz, 1996a; Manis et al., 1997; Mody et al., 1997). Three experimental tasks in particular have been used in studying speech perception abilities in children. In the first type of task, children might be presented with stimulus pairs and asked to discriminate between the two stimuli or reproduce the order of stimulus presentation. Using this task, children with either SLI or dyslexia were found to be outperformed by their normally developed age-mates when exposed to synthesized stop consonant-vowel syllables (viz., /ba/ and /da/) which represented good exemplars of natural speech (Tallal & Piercy, 1974; Tallal & Stark, 1982; Reed, 1989; Mody et al., 1997).

The second kind of task involves a categorical perception paradigm. In a categorical perception experiment, the subject hears a continuum of synthetic sounds ranging in equal acoustic steps from one endpoint stimulus (e.g., /da/) to another (e.g., /ga/). The listener might be asked to identify stimuli along the continuum into phonemic categories (here, /d/ and /g/) and/or discriminate between pairs of sounds as same or different. Aberrant categorical perception of several stop-consonant continua has been reported in children with SLI as well as children with dyslexia: typically, they tended to be less accurate at discriminating sounds across phoneme categories and less consistent in identifying sounds near the category boundaries than controls with normal language and literacy skills (Brandt & Rosen, 1980; Godfrey et al., 1981; Werker & Tees, 1987; Reed, 1989; Sussman, 1993; Manis et al., 1997). This suggests that children with SLI or dyslexia display less sharply defined phonetic boundaries compared to children who experience no difficulty in spoken and written language.

Synthetic speech continua are also included in the third type of task designed to determine the smallest acoustic differences among the sounds (i.e., just noticeable differences) that can be discriminated. Studies employing this task have shown that children

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49 A preliminary report on study F was presented at the meetings of the Society for Psychophysiological Research in the year 2000 (Heim et al., 2000a). Parts of this study were also included in the 2001 Society for Neuroscience Itinerary Planner CD-ROM (Heim et al., 2001c).
with SLI exhibit elevated discrimination thresholds: they require, relative to normal controls, greater acoustic distances between (speech) sounds in order to differentiate them (Elliott & Hammer, 1988; Elliott et al., 1989; Stark & Heinz, 1996a). Similar results were reported by Kraus et al. (1996) and Bradlow et al. (1999) in children with learning problems displaying a discrepancy between intellectual capacity and psychoeducational achievement. As a group, these children showed *inter alia* problems on measures of listening comprehension, sound blending, reading, and spelling (Kraus et al., 1996).

While Tallal and colleagues have proposed that the deficit in stop-consonant perception results from impaired integration of brief and rapidly changing sounds (for reviews see Tallal et al., 1993; Farmer & Klein, 1995), other researchers have ascribed the specific difficulty to the stop consonants’ spectral similarity (e.g., Mody et al., 1997; for a review see Studdert-Kennedy & Mody, 1995). Whatever the cause, proponents of both hypotheses have associated impaired consonant perception in SLI and dyslexia with distorted or ‘noisy’ phonological representations of speech sounds. Malformed representation of phonemes in turn might lead to language acquisition problems and later difficulty in mastering the phoneme-to-grapheme correspondence rules that underlie facile reading and spelling.

A potential limitation of psychophysical measures is that they often involve higher-level processing components, i.e., attentional and cognitive factors. The study by Kraus et al. (1996) sought to determine whether the perceptual difficulties experienced by children with learning problems (see above) originate in aberrant neuronal representations of acoustic events prior to conscious reception or in higher-level processing deficits. Kraus and colleagues observed that elevated discrimination thresholds assessed from a /da/-/ga/ continuum are paralleled by attenuated mismatch negativities (MMNs) to a deviant syllable /da/, in a train of standard syllables /ga/. The researchers concluded that the phonemic discrimination deficits probably have their origins in the auditory pathways and may be pre-attentive in nature. Comparable evidence was obtained by the same laboratory in larger groups of learning-impaired and normal children (Bradlow et al., 1999). Likewise, Schulte-Körne et al. (1998a) reported a diminished magnitude of the MMN to the phonetic contrast /ba/-/da/ in dyslexic boys compared to normally literate peers.

In a magnetoencephalographic (MEG) study, Heim et al. (2000c, see also study A of this thesis) found a difference in the organization of the auditory cortex between children with normal literacy skills and children with dyslexia following stimulation with the stop-consonant syllables [da] and [ga]. However, the organization of functional brain architecture may be modified by learning and experience (Elbert et al., 2001; Elbert & Heim, 2001). In
humans, the MMN and its magnetic counterpart, the mismatch field (MMF) have proved to be valuable tools for assessing neuronal plasticity and cortical re-organization (e.g., Kraus et al., 1995; Näätänen et al., 1997; Tremblay et al., 1997; Cheour et al., 1998; Winkler et al., 1999; Menning et al., 2000). For instance, Kraus et al. (1995) trained adults to discriminate between just-noticeable different variants of the stop consonant-vowel syllable /da/ over the course of six days. The behavioral training resulted in significant increases in the duration and magnitude of the MMN. In a related study, Tremblay et al. (1997) reported that changes in MMN following listening training generalized to novel speech sounds.

Merzenich and Tallal found that impaired phonological processing could be greatly improved in 5- to 10-year-old children with language-learning impairment (Merzenich et al., 1996; Tallal et al., 1996). Considering both the mechanisms of neural plasticity and the inaccurate representation of “rapidly successive phonetic elements” (Merzenich et al., 1996; p.77) in the auditory cortex of language-learning impaired children, they designed a computer-based training program. Children were submitted to a training regimen which included audiovisual games for about 100 min/day extended over four weeks with five training days a week. Rapid transitional speech and non-speech stimuli were initially disambiguated by prolonging them in time and/or amplifying them. As training progressed and the children demonstrated success, the modified acoustic stimuli were presented at rates that became closer and closer to those that occur in natural speech. The effect of training on the brain processes underlying these changes was examined by Temple et al. (2000) in three adult dyslexics using functional Magnetic Resonance Imaging. Normally reading adults displayed increased activity in the left prefrontal cortex in response to rapidly changing relative to slowly changing, non-verbal acoustic stimuli. Before training, dyslexic adults exhibited no differential left frontal response. After training, enhanced left frontal responsiveness during rapid as compared with slow stimuli was observed in two of the dyslexic readers.

Here we investigated the change in performance in SLI children with symptoms of dyslexia using conventional speech and language training. The training based on the syllabic method by Buschmann (1989, 1995; Hofmann, 1998) has been shown to be effective in elementary-school children with poor spelling abilities (Tacke et al., 1993). A recent study of our own laboratory (Schenk, 2001) revealed improvements on measures of reading and spelling in language-impaired children following syllabic training compared to a waiting-list group. In the current study, training-related changes were assessed by means of (i) a categorical perception task on a [ba]-[da:] continuum, (ii) language-related psychometric
measures (verbal memory span/phonological memory, reading/phonological decoding, and spelling/phonological encoding), and (iii) the neuromagnetic MMF. Furthermore, we sought to determine whether improved performance in a therapeutic setting is mirrored in an altered source configuration of event-related magnetic fields indicating cortical re-organization.

**Methods**

**Training group**

The training group comprised 7 SLI children with symptoms of dyslexia (mean age 11.61 ± 0.71 years, 3 females). The children attended a school for special education in the catchment area of Konstanz (Baden-Württemberg) and were referred by the school principal and classroom teachers. The selection criteria used were that (a) the children were diagnosed as SLI; (b) they were aged between 9 and 12 years; (c) they had German as their mother tongue; (d) they were right-handed; (e) their non-verbal IQ was > 85; (f) they were suffering no hearing impairment, brain lesion, or chronic medical condition; and (g) they were not diagnosed as having an attentional and/or hyperactivity disorder or clinically relevant emotional disturbance.

As documented in the school records, non-verbal intelligence was mostly estimated with Cattell’s Culture Fair Intelligence Test (Weiss & Osterland, 1979; Weiss, 1987). Each child’s non-verbal IQ was reassessed by using Raven’s Colored Progressive Matrices (Schmidtke et al., 1980). For details see below. Handedness was measured using the lateralization quotient (LQ) of the Edinburgh Handedness Inventory (Oldfield, 1971). Children with an LQ ≥ +70 were designated right-handers.

The training study was approved by the state school authority (Oberschulamt Freiburg, Baden-Württemberg). All parents of the children were asked to sign a consent form, after the nature of the study had been explained.

**Syllabic training**

The training of reading and writing followed a syllabic principle developed by Buschmann (Buschmann, 1989, 1995; Hofmann, 1998). Writing training had three major characteristics: (1) the child said a word while writing it down (= co-articulation); (2) co-articulation was performed on a syllable-by-syllable basis (= syllabic speaking); and (3) before the child has learned co-articulation, he or she practiced syllabic speaking with flourishes of the writing arm while stepping sideways in the writing direction (= flourishing
exercise). Reading training was similar. Syllabic speaking was the same as during writing training, but the children co-articulated while drawing small inverted arches under the syllables.

The children were trained in groups of two or three for two 1-hr sessions/week and were assigned additional homework for four consecutive weeks. Training sessions took place at school within the normal teaching day. After each session the children received a small reward (candies or stickers). The training schedule is outlined in Table II.F.1.

**Table II.F.1: The training schedule**

<table>
<thead>
<tr>
<th>First week</th>
<th>Session 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Different objects were hidden under a cloth. The experimenter took the first object – for instance a postcard depicting a sunflower (German: <em>Sonnenblume</em> [zɔnənblʊmɛ]) – and asked the children to name it. The experimenter demonstrated the flourishing exercise as follows: A flourish of the writing arm began at eye level, passed in front of the stomach and ended back at eye level. The arch-shaped flourish was accompanied by a step to the right, the left leg was dragged like dancing a waltz. At the same time the syllable was co-articulated. The beginning and end of a phonetic syllable (here, <em>Son-nen-blu-me</em>) was clearly marked by the characteristic performance of the flourishes and steps. Moreover, these motor activities facilitated rhythmical speaking. One of the children drew an object – for instance a potato (German: <em>Kartoffel</em> [kaʁtɔfˈɛl]) –, named it, and practiced the flourishing exercise (<em>Kar-tof-fel</em>). A second, then a third child and finally all the children did the exercise with this object. Then another child took an object, etc. Other words were practiced which has been spontaneously generated by the children or the experimenter (e.g., compound words such as <em>Kar-tof-fel-sup-pe</em> [kaʁtɔfɛlˈzuppa], English: ‘potato soup’). <strong>Homework.</strong> Verbatim: “Practice the flourishing exercise by using the words presented in the session. Choose your favorite word for flourishing.”</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Second week</th>
<th>Session 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Favorite words and words of the previous training session (“old” words) were flourished. “New” words (compound words of variable length) were practiced (e.g., <em>Kau-gum-mi-au-to-ma-len</em> [kaʊkumiautomaten], English: ‘chewing gum vending machines’). Further, the flourishing exercise was supplemented by minor variations: The children drew small inverted arches (∪∪∪) with the finger or chalk on the board while stepping sideways, or, while sitting on their chairs the children drew smaller arches on the table or in the air. For both variations co-articulation was natural. <strong>Homework.</strong> Verbatim: “Practice the flourishing exercises by using the words presented in the session. Choose your favorite word for flourishing.”</td>
<td></td>
</tr>
</tbody>
</table>

| Session 3 |
| Flourishing exercises (≈10 min) with favorite words, some old words (e.g., ‘sunflower’, ‘potato’) or words which were still difficult for the children. Transition to writing: The experimenter wrote a word on the board (e.g., *malen* [maːlɛn], English: ‘to draw’). Each child read it silently and whispered it in the experimenter’s ear. The children flourished the word while sitting at their tables. The experimenter wrote *malen* on the board and co-articulated it (*ma-len*). Each child wrote the word in her/his exercise book and co-articulated it. The experimenter drew inverted arches under the syllables of the word with her finger and thereafter with chalk: ıyordu Each child did the arches in the exercise book first with her/his finger and then with a ball-point pen. The children turned to a new page in their exercise books and were requested to write the word *malen* as before. Again, they drew arches under the syllables with the finger/ball-point pen. The next word (e.g., *re-den* [redɛn], English: ‘to talk’) was introduced, etc. **Homework.** Verbatim: “Practice the flourishing and writing exercises by using the words presented in the session. Choose your favorite word for flourishing.” |

| Session 4 |
| Same procedure as in session 3. |
**Third week**

**Session 5**
Flourishing exercises with favorite and other (old/new) words. In addition, short sentences were flourished (e.g., *Die Bie-nen sum-men*. [diː] [biːnən] [sum-men], English: ‘The bees are humming.’). These exercises lasted ≈5 min. Transition to reading (≈35-40 min): The experimenter wrote the arbitrary syllable combination *nanunana* [nanunana:] on the board. The children read and flourished it (na-nu-na-na). The experimenter and the training participants drew small arches (with the chalk) under the syllables. The experimenter wrote *nemunene* [nemunene:] on the board, proceeding as before (ne-ru-ne-ne). The experimenter wrote *latituli* [latituli:] on the board, again, proceeding as before (la-iti-tu-li). The children were given sheets of paper containing rows of syllables. The rows varied between 16 to 32 letters in length. Adjacent letters were separated by two blanks. The experimenter said: “The magic word *nanunana* will guide you through all the rows.” Again, the children flourished *na-nu-na-na*. Each child read the first row while drawing small arches with the finger/ball-point pen under the syllables. Then the children read the first row in unison. The next rows were treated the same way. Transition to writing: New words were written following the same procedure as in sessions 3 and 4.

**Homework.** The children were given the same reading list and were asked to practice the rows which had been read in the previous session. Additionally, they were asked to choose their favorite word for flourishing.

**Fourth week**

**Session 6**
Favorite and other (old/new) words were flourished (≈5 min). Transition to reading (≈35-40 min): The magic word *nanunana* was repeated. The experimenter wrote the magic word on the board. The children read/flourished it. All the children drew small arches under the syllables on the board. The syllable combinations *nemunene* and *latituli* were repeated in the same way. Then the children went on reading their reading lists according to the procedure introduced in session 5. Transition to writing: see previous sessions.

**Homework.** See session 5.

**Session 7**
Favorite and other (old/new) words were flourished (≈5 min). Transition to reading (≈15 min): The children continued reading their reading lists (see previous sessions). Transition to writing: A “trick” was introduced for the spelling of difficult words with inconsistency in phoneme-grapheme correspondence (e.g., *Pilz* [pilts], English: ‘mushroom’). The experimenter demonstrated the trick on the board. She said: “I am speaking and writing *Pil*, then pause, put the dot on the *i*, flourish *ze* [NB: *Pilze* = plural of *Pilz*] and add the *z*. *Pilz* This trick helped the children to identify the last sound as letter *z* and not *tz* or *is*. Each child practiced the trick with that word. The next irregular word (e.g., *schneller* [schnel], English: ‘quick’) was introduced and practiced. She said: “I am speaking and writing *schnel*, then pause, flourish *ler* [NB: *schneller* = comparative form of *schnell*] and add the *l*, *schnell*.” In this way the trick helped the children to hear the double *l*. Towards the end of the session some new (phonetically regular) words were flourished and written (see previous sessions).

**Homework.** Verbatim: “Practice the trick with the same words as presented in the session. Choose your favorite word for flourishing.”

**Session 8**
Flourishing exercises with favorite and other (old/new) words (≈5 min). Transition to reading (≈15 min): The children continued reading their reading lists (see previous sessions). Transition to writing: The trick was repeated and applied to new words. Towards the end of the session a short sentence (e.g., *Die Bie-nen sum-men auf der Wie-se*. [diː] [biːnən] [sum-men] [auf] [dɔːf] [vɪsə], English: ‘The bees are humming on the meadow.’) was flourished. The children wrote the sentence in their exercise books and did the arches with the finger/ball-point pen.

**Training measures**
Before and after syllabic training, psychometric, psychoacoustic, and MEG measures were obtained. Children were all tested individually. Psychometric tests were administered at school; the psychoacoustic and MEG experiments were conducted at the laboratory of the University of Konstanz.
Psychometric tests. As mentioned above, children in the training group had previously been tested for intellectual capacity. In the current study, children’s non-verbal intelligence was reassessed by using Raven’s Colored Progressive Matrices, CPM (Schmidtke et al., 1980). The CPM test was chosen for reasons of practicability (e.g., brief testing time, attractive stimulus materials). The scores of three children exceeded by one year of age the range within which the test is valid. IQ norms for the oldest age group (i.e., 10;4-11;0 years;months) provided the basis for calculating the intelligence scores of children outside the valid range.

Children’s memory span for digits was assessed using the forwards and backwards method. All subjects received the forwards span test before the backwards span test as is customary (e.g., Tewes et al., 2000). Children were informed that digits between 1 and 9, incorporated in series of variable length, would be presented at a rate of one per second. They were instructed to listen carefully and repeat the digits immediately in either the same (= forwards) or reversed (= backwards) order of presentation. Digit-span testing followed an adaptation of the one-up one-down staircase procedure (Cornsweet, 1962; Bachelder & Denny, 1977a,b). A correct reproduction of an item sequence was followed by an increase in item length, whereas an incorrect answer led to the item length being decreased. In the latter case, a new sequence of the last successfully performed item number was presented. In a preliminary testing phase, the starting item length was two digits, i.e., short enough for all children to manage the task. After a subject had made two incorrect responses the main test began. The main test comprising 10 presentations (= trials) starting at the last successfully performed item length. Digit span forwards and backwards were each determined by taking the arithmetic mean of the item numbers presented in the 10 trials of the main test. This procedure has proven to be both efficient and well-suited for estimating memory spans in learning-impaired children (Heim, 1997).

Children’s reading abilities were estimated using the Zürcher Lesetest, ZLT (Linder & Grissemann, 1998) and a non-standardized word-reading test. The ZLT required the child to read single words (in total 72 items) as well as small passages (3 stories consisting of 261 words). If the subject either did not pronounce an item correctly, corrected her-/himself, or failed to respond, an error was recorded. As a control, all subjects’ performances were audio-taped.

The non-standardized word-reading test, originally comprising 100 words, was modeled on the Word Identification Test of the Woodcock Reading Mastery Tests-Revised (Woodcock, 1987). The words differed in degrees of familiarity, regularity, as well as
complexity of graphemes and syllabic structure. They were grouped in order of difficulty and were 2-15 letters long. The spelling of the words was in accordance with the new German spelling rules in force since August 1, 1998. Due to time constraints and to provide a more reliable training measure, the test was split into two halves (A and B) using the odd-even method (Lienert, 1989). The order of the two forms each containing 50 words was counterbalanced across time of testing and subjects (pre-A vs. post-B, pre-B vs. post-A). The responses of the children were audio-taped and scored according to the following points system: 0 = no response or error, 1 = self-correction, 2 = complete/partial repetition of a word, 3 = correct response. A maximum of 150 points could be achieved in each test form. Full listings of the reading-test items are included in the Appendix.

Three different tests were used to assess children’s phonological skills. The Mottier-Test (Welte, 1981) providing a measure of phonological memory. The test required verbal repetition of spoken pseudowords (i.e., arbitrary, pronounceable letter combinations), two to six syllables in length. A total of 30 items were presented, yielding a maximum score of 30 points. As a control, the children’s performance was audio-taped.

Phonological decoding (i.e., the application of grapheme to phoneme correspondence rules) was estimated using a non-standardized pseudoword-reading test. The test originally comprised 100 pseudowords, 2-15 letters long. Pseudowords were derived from items of the aforementioned non-standardized word-reading test by permuting letters within or between the words. Letter combinations within pseudowords were constructed so as not to form German morphemes. The pseudoword-reading test was split into two halves (A and B) following the method applied to the word-reading test. The order of the two 50-item test forms was counterbalanced across assessment times and subjects (pre-A vs. post-B, vice versa). Children’s reading was audio-taped and scored as for the non-standardized word-reading test. A total of 150 points could be achieved in each test form. Full listings of the pseudoword reading-test items are included in the Appendix.

To assess phonological encoding (i.e., the application of phoneme to grapheme correspondence rules) two comparable forms of phonetic dictation (A and B) were administered (Findeisen & Melenk, 1991). Form A was a story about the European rocket Ariane and contained 83 phonetically regular words. Form B was about long airplane flights between Europe and America and consisted of 85 phonetically regular words. The order of the two test forms was counterbalanced across time of testing and subjects (pre-A vs. post-B, pre-B vs. post-A). The children were required to write the stories from dictation. Subjects were not judged on their use of upper- and lower-case letter rules.
Depending on the child’s class level either DRT 4 or DRT 5 of the Diagnostische Rechtschreibtest (Grund et al., 1994, 1995) was administered to evaluate spelling abilities. For each version two parallel forms (A and B) were used. The sequence of the parallel forms was counterbalanced across assessment times and subjects (pre-A vs. post-B, pre-B vs. post-A). The tests required the children to fill in single spoken words. In total, 42 and 51 items had to be recorded, yielding maximum scores of 42 and 51 for the DRT4 and DRT5, respectively. In contrast to phonetic dictation, the spelling tests emphasized orthographic rather than phonetic items.

**Psychoacoustic experiment.** Phoneme reception abilities in SLI children were assessed by using a categorical perception task with German stop consonant-vowel syllables. A 10-item stimulus continuum varying in equal steps from [baː] to [daː] was created using Speechlab software (Diesch, 1997) based on a Klatt cascade/parallel formant synthesizer (Klatt, 1980). All syllables were generated in the cascade branch of the synthesizer at a 10 kHz sampling rate. The total stimulus duration was 250 ms including a formant transition (FT) period of 40 ms. The stimuli of the continuum were composed of three formants (F1-F3) and differed in the onset frequencies of the second and third FT. The end points of the continuum were defined by good examples of the syllables [baː] and [daː] (see Methods section of study B, this chapter). The starting frequencies of the second and third FT were 1095 Hz and 2100 Hz for the end point syllable [baː] and 1702 Hz and 2633 Hz for the end point syllable [daː]. The steady-state formant frequencies of the vowel [aː] were 770, 1340, and 2400 Hz for F1, F2, and F3, respectively. The fundamental frequency of each syllable started at 128 Hz and decreased linearly to 109 Hz at stimulus offset. The amplitude of voicing was constant at 54 dB and fell linearly to 11 dB during the last 25 ms of the stimulus.

The categorical perception task was carried out on an IBM compatible 486-Personal Computer. The syllables were delivered to both ears via Quart Phone IMP50 headphones at ≈72 dB sound pressure level. The interstimulus interval (defined as syllable offset to onset) was 1 s. Children were asked to classify 10 random presentations of each stimulus (= 100 trials) as [baː] or [daː] by pressing a corresponding key on the computer keyboard (left cursor key = [baː], right cursor key = [daː]) with the index finger of their right hand. No feedback was provided for responses. For each subject, the identification functions \( f \) of the [baː]-[daː]
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continuum were quantified by calculating the proximity of the two function curves according to

\[ f = \sqrt{\sum_{i=1}^{10} (a_i - b_i)^2} \]

where  

- \( i \) = stimulus number along continuum
- \( a_i \) = % identification [ba:]  
- \( b_i \) = % identification [da:]

**Magnetoencephalographic measures.** Neuronal phoneme processing was investigated by recording magnetic-brain activity in response to synthetic syllables. The syllables were selected based on the results of a categorical perception experiment conducted in normally literate subjects (see also Methods section of study B of this thesis). They were asked to identify stimuli in the same 10-item [ba:]-[da:] continuum as described above. The mean identification scores for stimuli 5 and 8 in the continuum were 88 and 91% for children (n = 16) and 96 and 91% for adults (n = 17). These two stimuli occurring near the category boundary of the continuum were referred to as [ba:] and [da:]. The starting points of the second and third formants were 1365 Hz and 2337 Hz for the [ba:] stimulus and 1567 Hz and 2515 Hz for the [da:] stimulus. (For stimulus duration and other parameters, see paragraph ‘Psychoacoustic experiment’.) Variants of the syllables [ba:] and [da:] were preferred to ideal exemplars, as they are more demanding thus enhancing the ecological validity of the speech material.

A series of 650 syllables was delivered binaurally through a magnetically silent and echo-free plastic tubing terminating in ear inserts (see study B, this chapter). The duration of the intertrial interval (stimulus onset to stimulus onset) was constant at 1 s. The stimulus intensity was 60 dB above the individually determined hearing level. A passive oddball paradigm was used in which the syllable [ba:] served as the standard (probability of occurrence = 80%) and [da:] as the deviant stimulus (probability of occurrence = 20%).

To control the level of arousal and to fixate their attention, the children were presented with a silent cartoon/movie displayed on a magnetic-field free screen. Subjects lying supine

51 Subjects were also exposed to a block of 650 syllables with extended formant transitions (90 ms). These data are not reported here. The order of the two blocks was varied between subjects, but within subjects block order was held constant across assessment times (pre- vs. post-training).
were asked to move as little as possible and to avoid excess blinking during recordings. Compliance was verified by video-monitoring.

The MEG signals were recorded in a magnetically shielded room, using a helmet-shaped 148-channel whole-head neuromagnetometer (for technical details see study B of this thesis under ‘Methods’). Prior to the beginning of the MEG measurement, five head position indicator coils were attached to the subject’s forehead and in front of the tragus of each ear. The locations of these coils in relation to five anatomical landmark points (nasion, inion, vertex, and both preauricular points) were measured with an Isotrak 3-D digitizer (Polhemus Navigator Sciences, Colchester, VT, USA). Before and after recording, the positions of the indicator coils with respect to the sensor were determined from the magnetic signals produced by the coils.

Brain responses evoked by syllables were recorded continuously at a sampling rate of 508.63 Hz with a bandpass of 0.1-100 Hz. Eye movements and blinks were monitored by recording horizontal and vertical electro-oculograms (EOG). In an off-line mode, magnetic signals were first corrected for magnetocardiographic activity by means of a linear regression algorithm included with the BTi software package. Then, averaged waveforms for the standard and deviant syllables were calculated across epochs of 800 ms, including a 100 ms prestimulus baseline. Epochs with a MEG or EOG change > 3.5 pT or > 120 µV, respectively, were omitted from further analysis. The baseline was corrected for each channel according to the mean value of the signal during the 100 ms prior to the stimulus. After that, evoked fields were digitally low-pass filtered to 20 Hz using a second-order zero-phase shift Butterworth filter (filter roll-off: 12 dB/oct).

For analyzing the MMF, difference waves were calculated by subtracting the filtered averaged waves to the standard syllable from those to the deviant syllable. Mean MMF amplitude was measured in the root-mean square (RMS) difference wave scored in a fixed time-window between 150 and 300 ms (Kraus et al., 1993) for selected channels over the left and right hemispheres (see Appendix).

Source parameters of the evoked magnetic fields were estimated with a single equivalent current dipole (ECD) in a spherical volume conductor using 34 channels separately over the left and right perisylvian regions (see Appendix). An ECD defined by the dipole moment, the orientation, and space coordinates was computed for each sample point by means of a least-squares fit. The location estimates of each ECD were specified with reference to a head-based Cartesian coordinate system. The origin of this coordinate system was set at the mid-point of the medial-lateral (y-) axis interconnecting the center points of the
entrance to the auditory meatus of the two ears (positive towards the left ear). The posterior-anterior (x-) axis projecting from the origin to the nasion (positive towards the nasion) and the inferior-superior (z-) axis being perpendicular to the x-y plane (positive towards the vertex).

Data quality sufficient for utilizing this source analysis technique was achieved for magneto-cortical responses to the standard syllable in the latency range of 231-278 ms (labeled N260m) for six out of seven SLI children. Optimal fit source estimates around the RMS maximum of the magnetic field were accepted when they satisfied the following statistical and anatomical requirements: (a) goodness of fit > 90%, (b) confidence volume < 2000 mm$^3$, (c) RMS > 40 fT, (d) dipole moment > 5 nAm, (e) stability of spatial source coordinates (x, y, and z) over a few milliseconds, (f) distance of ECD to midsagittal plane > 2.5 cm, and (g) inferior-superior value 3 < z < 8 cm. Accepted ECDs were oriented downward indexing the negative polarity of the magnetic component. The average goodness of fit of the ECD model was 98% before and 97% after (SEMs = 0.4) training; confidence volumes pre- and post-training were 117.18 mm$^3$ (SEM = 111.74) and 86.96 mm$^3$ (SEM = 41.21), respectively [all Fs (1,5) < 1, all ps > .05, n.s.].

**Group of healthy children**

The group of healthy children comprised 7 subjects (mean age 10.83 ± 1.13 years, 2 females) with normal literacy skills, all native speakers of German. They were not significantly different from the training group in terms of age ($t_{12} = 1.5$, $p > .05$, n.s.). All except one healthy subject (LQ = −70 on the Edinburgh Handedness Questionnaire) were right-handers. Subjects were rewarded with shopping vouchers and/or cinema tickets. Informed written consent was obtained from the children and their parents.

**Behavioral measures.** Children’s non-verbal intelligence was estimated using Raven’s Standard Progressive Matrices, SPM (Heller et al., 1998). Digit span forwards and backwards tests were the same as for the training group. To assess reading abilities, the ZLT and the non-standardized word-reading test were administered as well. The word-reading test was given in its full length (100 words); in total 300 points could be achieved. Similar to the training group, phonological skills were documented using the Mottier-Test and non-standardized pseudoword-reading test. The pseudoword-reading test was administered in its full length (100 items, yielding a maximum of 300 points). A phonetic dictation test was not included. Spelling abilities were estimated with the B-forms of the DRT 4 and 5 (see training group); for two healthy subjects having been attended grade 6, the Westermann Rechtschreibtest, WRT6+ (Rathenow et al., 1981) was employed. In the WRT6+, 40 sentences have to be
completed, yielding a maximum score of 40. The categorical perception task was the same as for the training group.\textsuperscript{52}

**Magnetoencephalographic measures.** Magnetic-brain activity was recorded with the same [ba:]-[da:] syllables, experimental paradigm, and MEG device as described under ‘Training measures’.\textsuperscript{53} Using identical data analysis technique, MMFs were calculated for all healthy children. Similar to the training group, the N260m could be modeled with the ECD for six out of seven healthy subjects. Acceptance of dipole fits was based on the same criteria as detailed above; all ECDs were oriented correctly. In healthy subjects, the ECD model explained on average 98\% (SEM = 1) of the measured magnetic field variance; the average confidence volume was 31.51mm\textsuperscript{3} (SEM = 16.51). The two parameters were not significantly different from those of the training group [min. and max. \( F_{s} (1,10) = 0.07 \text{ and } 1.56, \text{ respectively; all } ps > .05, \text{ n.s.} \].

**Statistics**

Group comparisons were carried out for pre- and post-training measures. Digit span values were submitted to a mixed-model univariate analysis of variance (ANOVA) with Group (training vs. healthy) as between-subjects factor and Span method (forwards vs. backwards) as within-subjects factor. Word- and passage-reading scores of the ZLT were subjected to a multivariate ANOVA (\( F \text{ = Wilks’ Lambda} \); Group constituted the between-subjects factor. All other behavioral variables were analyzed using unpaired \( t \)-tests. In cases with unequal population variances (Levene test), \( t \)-tests with separate variance estimates were conducted; adjusted degrees of freedom and \( p \)-values are given below. Magneto-cortical parameters were submitted to a mixed-design univariate ANOVA with Group (training vs. healthy) treated as a between-subjects factor and Hemisphere (left vs. right) as a within-subjects factor.

As described in the Results section, children in the training group tended to have lower IQ scores than healthy subjects. We did not adjust the statistical analyses by covarying non-verbal IQ for the following reasons: (a) Children of the training group have already been tested for non-verbal intelligence by their class and/or remedial teachers. We applied the CPM to yield an additional unique measure of intellectual capacity. Furthermore, improvements in

\textsuperscript{52} Categorical perception was also tested for syllables with 90-ms formant transitions (see training group).

\textsuperscript{53} Healthy subjects were exposed to three further blocks each of 650 stimuli: (1) [ba:]-[da:] contrast with 90-ms formant transitions (see training group), (2) endpoint [ba:] vs. variant [ba:] with 40-ms or (3) 90-ms formant transition periods. The order of the blocks was counterbalanced across subjects.
phonological and/or literacy measures, accompanied by an unchanging non-verbal IQ, would provide suggestive evidence for specific training effects. (b) In small non-equivalent group designs, correlations between covariate and dependent variables are subject to outliers. In small sample sizes as given in the current study, analysis of covariance would lead to a misrepresentation of effects (Dunn & Clark, 1987). (c) The CPM and SPM contain different IQ distributions. While in the CPM up to the age of 9 years a maximum IQ of 132 could be achieved, the SPM provide a maximum value of 145. Thus, the application of the SPM would result in greater intersubject variability.

Pre- vs. post-training scores were compared by using three statistical models. Paired t-tests were performed on behavioral data with the exception of ZLT reading and digit span variables. Word- and passage-reading scores of the ZLT were subjected to an incomplete multivariate repeated measures ANOVA with Time of testing (pre- vs. post-training) constituting the within-subjects factor ($F = $ Wilks’ Lambda). Digit span and magneto-cortical measures were analyzed using complete multivariate repeated measures ANOVA with Time of testing (pre- vs. post-training) as one within-subjects factor and Span method (forwards vs. backwards) or Hemisphere (left vs. right), respectively, as the other.

The Results section describes ANOVA results providing significant interaction effects ($p < .05$) are followed by planned comparisons. Data are presented as mean ± SEM.

**Results**

**Psychometric tests**

*Non-verbal intelligence.* The mean non-verbal IQ of healthy subjects was 116.42 ± 7.67. Within the training group, non-verbal IQ remained stable across the time of testing; the pre- and post-test scores were 97.71 ± 3.39 and 99.29 ± 3.09, respectively. Group analyses revealed a tendency for the training children to have lower IQ scores than the healthy subjects (pre-training: $t_{8.25} = -2.2, p < .06, \text{n.s.}$; post-training: $t_{7.90} = -2.1, p < .07, \text{n.s.}$).

*Digit span.* Figure II.F.1 depicts the digit span forwards and backwards for the SLI children before and after training and for the healthy subjects. Mixed-design ANOVA including pre-training scores of the SLI children provided significant main effects for the factors Span method [$F(1,12) = 79.1, p < .001$] and Group [$F(1,12) = 28.7, p < .001$]. The effects indicate that (a) both groups displayed superior performance in the forwards compared to backwards span test and (b) forwards and backwards spans were significantly lower in the training group (4.16 ± 0.21 and 2.94 ± 0.13) than in the group of healthy children (5.26 ± 0.22
and 4.07 ± 0.12). Repeated measures ANOVA performed within the training group showed significant main effects for Time of testing \[F(1,6) = 12.4, \ p < .01\] and Span method \[F(1,6) = 59.3, \ p < .001\]. In addition, there was a significant Time of testing × Span method interaction \[F(1,6) = 11.0, \ p < .02\]. Post-hoc planned contrasts revealed a significant increase in backwards span (mean difference = 0.59; \(F(1,6) = 17.0, \ p < .006; \) post-score = 3.53 ± 0.22), but no change for forwards span (mean difference = 0.20; post-score = 4.36 ± 0.25) following training. Post-training backwards spans were still below the group mean of healthy children as demonstrated by the respective mixed-model ANOVA: Similar to pre-test group analysis, there were significant main effects of the factors Group \[F(1,12) = 7.5, \ p < .02\] and Span method \[F(1,12) = 57.5, \ p < .001\]; the Group × Span method interaction did not approach significance.

![Digit span forwards (F) and backwards (B)](image)

**Figure II.F.1.** Digit span forwards and backwards for the SLI group (pre-training = blue, post-training = red bars) and for the group of healthy children (green bars).

**Reading and spelling abilities.** Figure II.F.2 illustrates reading and spelling abilities in the SLI children (pre- vs. post-training) and the healthy subjects. To facilitate comparisons across each of the dependent variables, scores were provided as percent correct. Unpaired \(t\)-testing with the pre-training values of the SLI children revealed a significant difference between groups in non-standardized word reading (57.71 ± 7.22% in the training group and 92.86 ± 1.24% in the healthy group; \(t_{6.35} = -4.8, \ p < .003\)). Statistically meaningful gains (\(t_{6} = -3.4, \ p < .01\)) were observed at post-training time (increase = 9.14%; post-test score = 66.86 ± 8.04%). As expected, post-training performance on non-standardized word reading was still below the normal group mean (\(t_{6.28} = -3.2, \ p < .02\)).
The training group’s pre-test scores on standard word (71.03 ± 7.17%) and passage (70.55 ± 6.43%) reading of the ZLT were significantly lower than the group mean of healthy children [96.83 ± 0.99% and 95.73 ± 0.64%, respectively; \(F(2,11) = 0.4, p < .008\)]. This pattern of results also held true when analyzing the post-training data on the same subtests [79.76 ± 7.27% for words and 79.31 ± 5.51% for passages; \(F(2,11) = 0.5, p < .02\)]. However, the SLI children displayed statistically meaningful improvements on both subtests across assessment times [mean differences for words and passages = 8.73% and 8.76%, respectively; \(F(2,5) = 0.1, p < .006\)].

Unpaired \(t\)-tests revealed significant differences between groups in standard spelling (DRT 4/5, WRT 6+). Pre- and post-training scores of the SLI children were 12.97 ± 4.67% and 30.62 ± 6.00%; the mean of the healthy group was 81.60 ± 3.41% (pre: \(t_{12} = −11.9, p < .001\); post: \(t_{12} = −7.4, p < .001\)). Again, the SLI children demonstrated significant gains on literacy skills following training (mean difference = 17.65%; \(t_6 = −3.5, p < .01\)).

**Phonological skills.** Figure II.F.3 shows the performance (in percent correct) on the phonological measures for the SLI children before and after training and for the healthy subjects. The mean of the training group’s pre- (51.90 ± 3.91%) and post-test (62.38 ± 5.02%) scores on pseudoword repetition (Mottier-Test) was significantly lower than the group mean of healthy children (89.52 ± 1.69%; pre: \(t_{12} = −8.8, p < .001\); post: \(t_{12} = −5.1, p < .001\)). However, the SLI children repeated more pseudowords correctly after than before training (mean difference = 10.48%; \(t_6 = −5.0, p < .003\)).
The SLI children did significantly poorer on non-standardized pseudoword reading than the healthy children (84.19 ± 2.49%) both before (35.43 ± 5.73%; $t_{8.20} = -7.8$, $p < .001$) and after training (52.29 ± 7.98%; $t_{7.16} = -3.8$, $p < .006$). Again, the SLI children demonstrated significant improvements at post-training time (mean difference = 16.86%; $t_6 = -3.4$, $p < .02$).

The paired $t$-test performed on the phonetic dictation scores revealed statistically meaningful gains across assessment times. The pre- and post-training values of the SLI children were 65.34 ± 11.31% and 83.36 ± 8.59%, respectively (mean difference = 18.02%; $t_6 = -5.9$, $p < .001$).

**Categorical perception task**

Figure II.F.4 shows the mean identification functions of the [ba]-[da:] continuum for the SLI children (pre- vs. post-training) and the healthy subjects. In healthy children, stimuli 1-5 were identified as [ba:] and 7-10 were identified as [da:] with at least 76% accuracy. Stimulus 6 was perceived as [ba:] 39% of the time and as [da:] 61% of the time; thus, at stimulus 6 a shift to the category [da:] was in the offing. Before training, the SLI children$^{54}$ identified the syllables significantly less reliably than the healthy subjects as indicated by the closer proximity of the identification functions $[f_{\text{SLI-pre}} = 18.59 ± 2.28$ vs. $f_{\text{healthy}} = 26.53 ±$  

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$^{54}$ One male subject with SLI was excluded from categorical perception analyses since he classified each stimulus of the 10-item [ba]-[da:] continuum as [ba].
After training, the mean identification functions were widely spaced, i.e., the SLI children were significantly more accurate in their stimulus classification ($f_{\text{SLI-post}} = 24.69 \pm 1.68$, $f_{\text{SLI} \Delta \text{(post-pre)}} = 6.10$; $t_5 = -2.8$, $p < .04$). Moreover, the identification functions post-training approached the normal categorical perception pattern. The SLI children identified stimuli 1-5 as [ba:] and 7-10 as [da:] with at least 72% accuracy. Stimulus 6 was identified near chance level and thus formed the category boundary.

Figure II.F.4. Mean identification functions for the [ba:]-[da:] continuum in the SLI group (pre-training = blue, post-training = red traces) and in the group of healthy children (green traces).

**Magnetoencephalographic data**

**MMF.** Figure II.F.5A depicts the averaged magneto-cortical responses to the standard and deviant syllables in one subject with SLI before and after training and one healthy subject in anterior and posterior channels of the left and right hemispheres. The channel selection of each hemisphere is based on the maximum outgoing and ingoing magnetic field around 260 ms after stimulus onset. As shown in Figure II.F.5A, the difference between deviant and standard responses in the latency range of 150 to 300 ms is larger in the right hemisphere than in the left in the subject with SLI before training; after training, the activity pattern resembles that of the healthy subject, i.e., the corresponding difference is larger in the left than right.
hemisphere. Figure II.F.5B1 displays the sensor layout (148 channels) for the difference waves (obtained by subtracting responses to standard syllables from those to deviant syllables) in the subject with SLI at pre-training time. The difference waves in the latency range of the MMF (150-300 ms) were more pronounced in the right than in the left hemisphere. Figure II.F.5B2 illustrates the isocontour map of the difference wave at its maximum around 260 ms. The source of the MMF in each hemisphere points upwards, indexing a positive polarity of the MMF.\textsuperscript{55}

\textbf{Figure II.F.5. (A)} Averaged magneto-cortical responses to standard syllables (black curves) and deviants (colored curves) in one subject with SLI before (top) and after (middle) training and one healthy subject (bottom) recorded over one anterior and one posterior channel of the left and right hemispheres. Note, in the SLI subject the difference between deviant and standard responses (latency range: 150-300 ms) is larger in the right hemisphere than in the left before training; after training, the activity pattern resembles that of the healthy subject, i.e., the corresponding difference is larger in the left than right hemisphere. \textbf{(B1)} Whole-head view of the difference waves (obtained by subtracting responses to standard syllables from those to deviant syllables) in the subject with SLI prior to training, projected onto a plane. The waveforms are depicted from −100 before to 500 ms after stimulus onset. Inward going magnetic flux points downwards, outward going flux points upwards. Difference waves are more pronounced in the right hemisphere than in the left. The small vertical bar indicates the maximum of the difference wave, the MMF, at \approx 260 ms in one of the right anterior channels. \textbf{(B2)} The field distribution over the left and right hemispheres is plotted at the peak latency of the difference wave. Solid isocontour lines indicate the outgoing, the dashed lines the ingoing magnetic field, and the thick solid line zero flux. The spacing of the contours is 10 fT. A1-A148 represent the channel numbers. Following the ‘right-hand rule’, the MMF source of each hemisphere is oriented superiorly, indexing positive polarity (see next page).

\textsuperscript{55} This polarity is diametrically opposed to the electric equivalent, the MMN. However, other researchers have also found evoked responses reflecting a positive difference wave in children (e.g., Pihko et al., 1999; Leppänen et al., 1999).
Figure II.F.5. Legend see previous page.
Figure II.F.6 shows the mean amplitude of the MMF (RMS values) in the left and right hemispheres for the SLI children before and after training and for the healthy subjects. Mixed-design ANOVA including pre-training values of the SLI children provided a significant Group × Hemisphere interaction [$F(1,12) = 17.2, p < .001$]. Planned comparisons calculated for this interaction revealed a significantly larger MMF over the left (61.39 ± 9.18 fT) than right (43.90 ± 6.64 fT) hemisphere for the group of healthy children [$F(1,12) = 12.9, p < .004$]. Among the SLI children mismatch activity was significantly stronger in the right relative to the left hemisphere [66.06 ± 8.56 fT; $F(1,12) = 5.2, p < .04$], while the left-hemispheric mean MMF amplitude (55.01 ± 6.23 fT) was similar to that observed in the healthy subjects.

As Figure II.F.6 shows, the right-hemispheric MMF in the SLI group was attenuated at post-training assessment [Time of testing × Hemisphere: $F(1,6) = 6.0, p = .05$]. Mixed-model ANOVA including the post-test values of the training children simply provided a main effect for the factor Hemisphere [$F(1,12) = 11.8, p < .005$]. This indicates that after training the SLI group displayed a left-lateralized mismatch activity pattern (55.25 ± 3.75 fT in the left hemisphere vs. 44.54 ± 4.94 fT in the right), which was not statistically different from that observed in the group of healthy children.

**N260m.** Figure II.F.7 illustrates RMS waveforms in response to the standard syllable [baʃ] averaged for 6 SLI children (pre- vs. post-training) and 6 healthy subjects within hemispheres. The most prominent peak occurred in both subject groups was around 260 ms
following the stimulus (N260m). Mixed-design ANOVA revealed no significant group differences in terms of latency or field amplitude of the N260m (Figure II.F.7, see Table II.F.2). However, as indicated by within-subjects analysis in the SLI group, post-training amplitudes tended to be lower than pre-training amplitudes \( F(1,5) = 6.4, p < .052, \text{n.s.} \). This pattern of results was confirmed by the dipole moment data (see Table II.F.2): While there were neither significant group differences nor other statistically meaningful sources of variance, the SLI children showed a trend for decreasing \( q \)-values across time of testing \( F(1,5) = 4.8, p < .08, \text{n.s.} \).

**Figure II.F.7.** Grand average of the RMS waveforms elicited by the standard syllable [ba:] in the SLI group (pre-training = blue, post-training = red curves) and the group of healthy children (green curves). The most prominent peak occurred in both subject groups around 260 ms after stimulus onset.

**Table II.F.2: Latencies, field amplitudes, and dipole moments of the N260m to the standard syllable [ba:] for the SLI group (pre- vs. post-training) and for the group of healthy children (mean ± SEM)**

<table>
<thead>
<tr>
<th></th>
<th>Training group (n=6)</th>
<th>Healthy Ss (n=6)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td><strong>Latency (ms)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left</td>
<td>264.38 ± 3.63</td>
<td>260.75 ± 4.82</td>
</tr>
<tr>
<td>Right hemisphere</td>
<td>267.33 ± 4.04</td>
<td>267.65 ± 4.95</td>
</tr>
<tr>
<td><strong>Field amplitude (RMS, fT)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left</td>
<td>150.67 ± 26.73</td>
<td>124.33 ± 16.74</td>
</tr>
<tr>
<td>Right hemisphere</td>
<td>166.83 ± 23.25</td>
<td>150.58 ± 18.80</td>
</tr>
<tr>
<td><strong>Dipole moment (q, nAm)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left</td>
<td>45.87 ± 13.10</td>
<td>30.37 ± 5.52</td>
</tr>
<tr>
<td>Right hemisphere</td>
<td>40.40 ± 6.21</td>
<td>28.93 ± 2.86</td>
</tr>
</tbody>
</table>

*Note. For all Fs p > .05.*
Figure II.F.8 shows the source localizations of the N260m along the posterior-anterior (x-) axis for the SLI children before and after training and for the healthy children.

While, in healthy subjects, the sources of the N260m were located more anterior in the right (2.23 ± 0.11 cm) than in the left hemisphere (0.95 ± 0.33 cm), the SLI group displayed different pre-training source locations with reduced source asymmetry between hemispheres (pre-right: 1.71 ± 0.70 cm; pre-left: 1.59 ± 0.27 cm). However, mixed-model ANOVA calculated with the pre-training x-coordinates of the SLI children provided only trend effects for the factor Hemisphere \([F(1,10) = 4.7, p < .06, \text{n.s.}]\) and the Group × Hemisphere interaction \([F(1,10) = 3.3, p < .10, \text{n.s.}]\). As indicated by repeated measures ANOVA, the pre-training symmetric source configuration assumed a slight post-training asymmetry with a shift towards posterior in the left hemisphere (mean difference = 0.40 cm; post-x value = 1.19 ± 0.37 cm). In the right hemisphere alone, no significant change occurred across time of testing [mean difference = 0.10 cm; post-x value = 1.60 ± 0.45 cm; \(F(1,5) = 7.7, p < .04\)]. This result is mirrored in a significant main effect for the factor Group \([F(1,10) = 21.1, p < .001]\) as well as a significant Group × Hemisphere interaction \([F(1,10) = 5.5, p < .04]\) as revealed by a mixed-design ANOVA including the post-training data of the SLI children.

Figure II.F.9 illustrates the source localizations of the N260m along the medial-lateral (y-) axis in the left and right hemispheres for the SLI group (pre- vs. post-training) and the group of healthy children.
Mixed-design ANOVA including the pre-training \(\gamma\)-coordinates\(^{56}\) revealed a significant main effect of Group \([F(1,10) = 9.9, p < .01]\) with healthy subjects displaying more lateral source locations in both hemispheres (left: 4.53 ± 0.35 cm; right: –5.52 ± 0.23 cm) as compared to the SLI children (left: 4.12 ± 0.31 cm; right: –4.59 ± 0.30 cm). Pre- vs. post-training comparisons for the SLI group revealed a shift towards more lateral source locations (left: 4.75 ± 0.28 cm; right: –5.15 ± 0.15 cm), although the main effect for the factor Time of testing was non-significant \([F(1,5) = 5.9, p < .06, \text{n.s.}]\). Subsequent group comparisons including the post-training \(\gamma\)-values of the SLI children failed to reach significance.

Figure II.F.10 depicts the source localizations of the N260m along the inferior-superior (\(z\)-) axis in the SLI group (pre- vs. post-training) and in the group of healthy children.

\(^{56}\) In each hemisphere, absolute \(\gamma\)-coordinates were used for analyses.
Mixed-model ANOVA calculated with the pre-training z-coordinates of the SLI children showed significant main effects of Group \( F(1,10) = 6.3, p < .03 \) and Hemisphere \( F(1,10) = 7.4, p < .02 \). In general, N260m sources were more superior in the left than in the right hemisphere with the group of healthy children (left: 6.02 ± 0.27 cm; right: 5.78 ± 0.23 cm) displaying more superior localizations than the SLI group (left: 5.35 ± 0.20 cm; right: 4.93 ± 0.22 cm). Repeated measures ANOVA within the training group did not provide any statistically meaningful effect. However, in the left hemisphere, sources post-training tended to be located more inferior than the sources pre-training [mean difference = 0.41 cm; \( F(1,5) = 3.8, p < .11, \text{n.s.} \)]. Mixed-design ANOVA including the post-training z-coordinates of the SLI children still revealed significantly inferior sources in the SLI than in the healthy group \( F(1,10) = 6.9, p < .03 \). The post-training z-coordinates for children with SLI were 4.95 ± 0.35 cm and 4.81 ± 0.34 cm for left and right hemispheres, respectively.

Discussion

The present study examined the change in performance in SLI children with concomitant dyslexia following a 4-week (8-day) syllabic-training period. Training-associated changes were assessed by means of language-related psychometric measures, a categorical perception task, and the neuromagnetic components MMF and N260m. Pre- and post-training scores of the children with SLI were compared with data of children who experienced no unusual difficulty in spoken and written language.

We observed significant gains on tests of reading, spelling, and phonological processing skills. Main changes (on average 17.5%) were recorded for measures of spelling in general and phonological tests in particular – measures which the syllabic training was designed to directly address. These results are in line with those obtained in a treatment versus waiting-list group design by Tacke et al. (1993) and Schenk (2001).

Post-training outcomes of verbal memory span achieved significance only in the backwards condition. A study conducted in children with learning problems (Heim, 1997) revealed that recalling digits backward was less sustained than recalling digits forward. Thus, the larger digit span backwards scores after than before training might rather be due to a familiarization effect. Regarding both the short training period and the severity of the disorder, it was not surprising that all post-test scores on language-related psychometric tests were still below the group mean of healthy children. Similar to our previous syllabic-training study in language-impaired children (Schenk, 2001) no improvements on non-verbal
intelligence scores were observed pointing more likely to specific effects on language-related skills.

As reported by several researchers, children with SLI or dyslexia tended to show aberrant *categorical perception* of stop consonants (Brandt & Rosen, 1980; Godfrey et al., 1981; Werker & Tees, 1987; Reed, 1989; Sussman, 1993; Manis et al., 1997). Comparable evidence was found in the present study at pre-training time: the SLI children were significantly less accurate in identifying the stimuli as either [ba:] or [da:] than the group of healthy children. After training, on the other hand, SLI children’s identification functions were no longer different from those of healthy subjects. This outcome might be attributed to three factors: (i) the specific training implemented, (ii) unspecific aspects such as involvement in an active intervention program or receiving individual attention from the experimenter, or (iii) increased familiarity with the task. Considering the systematic change towards a normal categorical perception pattern it seems less likely that the outcome may only be driven by the familiarity with the task. The possibility that keeping the children occupied or giving them one’s attention might have caused the treatment effects cannot be completely rejected, however. Improvements on language-related psychometric tests associated with constant non-verbal intelligence scores provide suggestive evidence for a specific influence but need to be validated by comparing the effects of alternative training methods.

Our main interest was to determine whether improved performance in behavioral tasks was mirrored in a change of magnetic-brain activity indexed by the *MMF* to stop-consonant syllables. The group of healthy children demonstrated a dominant MMF in the auditory cortex of the left hemisphere. Larger mismatch activity over the left compared to the right hemisphere has also been reported in healthy adults following stimulation with native phonemes (Näätänen et al., 1997) as well as with cross-category phonemes (Sharma & Kraus, 1995). Before training, the SLI children displayed a stronger mismatch response in the right hemisphere than in the left, while their left-hemispheric MMF was similar to that observed in healthy subjects. After training, the SLI group did not differ from the group of language-normal children in the magnitude of the right-hemispheric MMF, leading to a comparable left-lateralized mismatch activity pattern. Thus, the children with SLI showed a deviance in the right hemisphere at pre-training time which appears to be normalized at post-training time. Since there was no repeated MEG measurement in the group of healthy children, one could raise the objection that the change in MMF topography might reflect natural variations. Indeed, Uwer and von Suchodoletz (2000) reported only a moderate stability for the electric MMN elicited by stop-consonant syllables in healthy school-age children. However, it seems
unlikely that this should be true solely for the MMF in the right hemisphere. Furthermore, an
instability of the MMF should not be expressed as a shift towards a normalization, and this is
precisely what happened to the MMF activity pattern in the SLI group. Whether the change in
topography is stable or a temporary phenomenon needs to be investigated in follow-up
assessments.

Given the performance profile on the categorical perception task across the two
assessment times, one may speculate that a larger MMF amplitude over the right than left
hemisphere is associated with less reliable identification of the stop-consonant stimuli
whereas a relatively reduced right-hemispheric MMF amplitude support adequate phoneme
identification. Possibly, the alteration of the MMF activity pattern may be associated with an
improvement of the cognitive processes involved in reading and spelling. Supporting
evidence that poor literacy skills may be associated with an atypical functional neuroanatomy
in the right hemisphere is offered from two MEG studies of our laboratory (Heim et al.,
2000b, see also study D of this thesis; Heim et al., 2001b, see also study C of this thesis). We
found that the right-hemispheric sources of the magnetic waves in response to the syllable
[ba] 100 ms after stimulus onset were located more anterior in controls than in subjects with
dyslexia. No such group difference was observed in the left hemisphere.

Analyses of the N260m elicited by the syllable [ba] indicate atypical source locations
in children with SLI both before and after syllabic training: While in the group of healthy
children the N260m source was found to be asymmetrical with a more anterior localization in
the right than in the left hemisphere, source asymmetry was absent or reduced in the SLI
group. In addition, the ECD of the N260m was located more superior in healthy subjects than
in SLI children. Thus, the sustained group difference in the source configuration of the
N260m may point to a neuroanatomical deviance in children with SLI. This finding regarding
the source loci in the SLI group contrasts with a previous report of normal N260m dipole
positions in anterior-posterior and inferior-superior directions in a group of children and
adolescents with dyslexia (Heim et al., 2001b, see also study C of this thesis). It is likely that
differences in subject characteristics such as type of language-based learning impairment
(dyslexia in study C; SLI with concomitant dyslexia in the current study) or chronological age
(range in study C: 8-15 years; range in the present study: 10-12 years) account for the
discrepancy.

To our knowledge, this is the first MEG study investigating changes in magnetic-brain
activity following speech and language training in children with SLI. Examining the effect of
intervention programs on brain processing will constitute a major step forward in our
understanding of neural mechanisms underlying language-based learning impairments. Further studies involving larger groups of children, alternative treatment procedures, and follow-up assessments are necessary in order to evaluate the validity of the present findings.

**Summary of study F:** Electrophysiological studies have provided neuronal evidence for a phonemic perception deficit in children with language-based learning impairments. Recently it was shown that phonological processing in children with specific language impairment (SLI) and dyslexia may rapidly improve with acoustically modified speech training. Using conventional speech and language training, we sought to determine whether the improvement of performance in SLI children is reflected in altered brain activity as measured by the auditory mismatch field (MMF) and N260m. Neuromagnetic responses elicited by the synthetic stop-consonant syllables [ba] and [da] were recorded by means of whole-head magnetoencephalography. Seven right-handed SLI children (aged 10-12 years) participated in training including syllabic speaking, writing, and reading. Training consisted of two sessions per week and additional homework for four consecutive weeks. Performance and cortical activity measures were obtained before and after training. Significant gains on measures of reading, spelling, and phonological skills were observed. Behavioral identification abilities across a [ba]-[da] continuum improved significantly. Moreover, the syllabic training seems to normalize neuronal phoneme processing in children with SLI: Pre-training MMF data revealed a dominant activation pattern in the right-hemispheric auditory cortex, post-training evaluations indicated a similar MMF topography as in normally developing age-mates. However, sustained group differences in the source configuration of the N260m across assessment times may also point to a neuroanatomical deviance in children with SLI. These data suggest that behavioral training in a therapeutic setting may alter neurophysiological parameters of cortical organization as indexed by the auditory MMF.
III. Synthesis and Implications

The present thesis examined behavioral and neurophysiological aspects of auditory processing in people with language-based learning impairments. This was accomplished by conducting a series of psychophysical experiments and magnetoencephalographic (MEG) recordings including stop consonant-vowel syllables. In the following section, the experimental questions inherent in the current work are reviewed. Then, outstanding issues which should be addressed in future studies of language-based learning disabilities are briefly discussed. The chapter concludes by suggesting elements for functional neuroanatomy of developmental dyslexia.

EXPERIMENTAL QUESTIONS AND OUTCOME

Within the framework of six studies (A-F), the primary goal of this thesis was to examine four experimental questions. These are outlined below.

(1) Are there differences in the neuromagnetic mismatch field (MMF) to stop consonant-vowel syllables between children and adolescents with dyslexia and normally literate control subjects? If so, is the observed neuronal activity pattern associated with behavioral discrimination performance?

The first complex of experimental questions has been specifically addressed by study B of this thesis. Tallal and Piercy (1974, 1975) observed that school-age children with specific language impairment (SLI) may succeed in discriminating stop consonant-vowel syllables when the fast transitional elements were artificially lengthened. In study B, we aimed at replicating these findings in a group of children and adolescents with dyslexia using the German syllables [ba:] and [da:]. Psychoacoustic performance data indicate that only a subset of dyslexic individuals (36%, labeled ‘benefiters’) displayed superior discrimination ability on temporally extended, relative to rapid, formant transition consonant syllables. The majority of the dyslexic participants (64%, designated ‘non-benefiters’) exhibited a balanced performance profile which resembled those of the normally literate controls. Neuronal syllable processing was then compared between the dyslexic subgroups and the control subjects. Dyslexic individuals comprising the benefiter group showed an increase in MMF amplitude to extended- versus rapid-transition syllables, namely in the right hemisphere. While normal controls demonstrated this effect over the left hemisphere, dyslexic subjects of the non-benefiter type displayed no formant transition-related MMF enhancement in either
hemisphere. This pattern of findings indicate both (i) differences in MMF to stop-consonant syllable contrasts between dyslexic subjects and normally literate controls and (ii) an association between magnetic activity in supratemporal cortices and performance profile.

(2) Are there differences in source location of event-related fields (ERFs) in response to stop consonant-vowel syllables between children and adolescents with dyslexia and normally literate controls? If so, is this a maturational phenomenon or rather a stable characteristic of dyslexia?

The second complex of experimental questions was examined cross-sectionally by studies A, C, and D of this thesis. Study A recorded auditory ERFs in response to pure tones and stop consonant-vowel syllables (viz., [da] and [ga]) over the left supratemporal cortex in dyslexic and normally literate children. The source location of the magnetic wave at 210 ms post-stimulus was found to be located ≈1.5 cm more anterior in dyslexic subjects than in controls. This difference was also evident relative to the source location of the earlier component at 80 ms which showed identical topographical distribution in both groups.

Study C employed whole-head MEG to elucidate the interhemispheric source configuration associated with auditory syllable processing. Children and adolescents with dyslexia were found to show atypical hemispheric asymmetry in the positions of the P100m sources to the stop-consonant syllable [ba:]. While in the normally literate control group the right P100m dipole was located more anterior than the corresponding dipole of the left hemisphere, the dyslexic group displayed a rather symmetrical source configuration between the hemispheres. This symmetry reflected a deviance in the right perisylvian region for the dyslexic subjects’ P100m generated ≈1 cm more posterior than the response in controls. The deviation was also obvious relative to the source location of the later component N260m which did not systematically differ between the participant groups.

Study D has shown that an atypical cerebral lateralization related to a deviance in the right perisylvian region is also present in adults with dyslexia. In normally literate controls, the N100m sources to the syllable [ba:] were found to be asymmetrical with a more anterior localization in the right than in the left hemisphere. The N100m dipoles in dyslexic adults did not exhibit the same interhemispheric asymmetry. While there was no significant between-group difference in the center of activity over the left hemisphere, the dyslexic subjects’ N100m source of the right hemisphere was positioned ≈0.70 cm posterior to the source in the control participants.
The three studies revealed (i) alterations in source locations of auditory ERFs to stop-consonant syllables in children and adolescents with dyslexia as well as (ii) that such deviations were detectable in adulthood and thus suggested a stable characteristic in dyslexia. Concerning findings offered from intracerebral recordings and magnetic source imaging techniques, the deviances might be tied to Heschl’s gyrus and adjacent regions, in particular the planum temporale (e.g., Liégeois-Chauvel et al., 1994; Lütkenhöner & Steinsträter, 1998; Ohtomo et al., 1998; Godey et al., 2001).

(3) Do children and adolescents with dyslexia exhibit an auditory temporal processing deficit? If so, does this deficit co-occur with a similar dysfunction in the visual modality?

These questions were the subject of behavioral study E. In 45% of the dyslexic participants discrimination performance on rapidly changing stop-consonant syllables pointed to limitations in auditory temporal processing. None of these subjects displayed temporal processing abnormalities in the visual domain. On the contrary, poor auditory temporal processing was associated with enhanced sensitivity in the visual modality. This suggests a specific rather than a general temporal processing deficit for dyslexia. In addition, limitations in auditory temporal processing might be compensated by a well-functioning visual sensory modality.

(4) Are improvements in literacy skills following linguistic training mirrored in an altered magnetic-brain activity in language-impaired children with symptoms of dyslexia? In other words, are there training-associated changes in the MMF and source configuration of ERFs to stop consonant-vowel syllables?

Study F investigated the final experimental question in a small group of SLI children with concomitant dyslexia using syllabic training. Significant gains on measures of reading, writing, and phonological skills were accompanied by changes in neuronal processing of stop-consonant syllables: Although pre-training MMF data revealed a dominant activation pattern in the usually non-dominant right-hemispheric auditory cortex, post-training evaluations indicated an MMF topography similar to that found in normally developing age-mates. However, deviations in the source configuration of an ERF component around 260 ms post-stimulus both before and after training may also point to a neuroanatomical deviance in children with SLI. Study F suggests that linguistic training may alter neurophysiological parameters of cortical organization as indexed by the auditory MMF.
Taken together, the current work targeting the auditory domain mainly suggests deviances in the right supratemporal cortex in people with dyslexia. Functional brain-imaging studies of dyslexia have predominantly found an unusual activation pattern in left-hemisphere perisylvian-language areas, in particular during reading tasks (see under ‘Functional neuroimaging’, chapter I). The findings offered from high-spatial resolution imaging techniques and the current data acquired with high-temporal resolution MEG by no means contradict each other. Instead, the ERF observations on speech syllables elucidate the *temporal dynamic* of this phenomenon.

**OUTSTANDING ISSUES**

Continued research in the area of functional neuroanatomy and behavior in language-based learning disorders is needed to address a range of outstanding issues. These are detailed in the subsequent section.

*Can the findings obtained in our dyslexic samples be ascribed to either a dysfunction of the phonological system or a more general auditory deficit involving the perception of temporal information?*

The nature and origin of speech sound processing problems in dyslexia have been a subject of debate for more than two decades (for recent studies on this topic see Kujala et al., 2000; De Martino et al., 2001; Marshall et al., 2001; Rosen & Manganari, 2001; Waber et al., 2001). Tallal et al. (1993) proposed that the temporal characteristics of auditory stimuli are critical for individuals with language-based learning disorders (see under ‘Auditory temporal processing deficit’, chapter I). More to the point, the persons affected may have difficulty when stimuli are brief or rapid, but display no problems when they are lengthened in time or presented at a slower rate. This impairment is said to apply to both verbal and non-verbal auditory input. A study by Wright et al. (1997) revealed greatly enhanced backward masking effects for brief pure tones (20 ms) in children with SLI relative to language-normal controls. By contrast, the SLI children performed at the level of the control group when a relatively long tone (200 ms) was embedded in the masking stimulus. Wright and colleagues suggest that an abnormal degree of backward masking would be expected to “clearly degrade the perception of the brief acoustic elements of speech” (p.178) and is in accord with the notion “that children with reading difficulties are particularly poor at discriminating words that differ only in their first sound” (p.178).
Other researchers have argued that children’s auditory perceptual problems are associated symptoms which are milder and less consistent in occurrence (e.g., Studdert-Kennedy & Mody, 1995; Snowling, 2001). This view holds that individuals with language-based learning disorders have a deficit in linguistic processing, that is, the individuals affected exhibit difficulty in transforming speech input into the phonological code used in reading and writing (see under ‘Phonological processing deficit’, chapter I).

The question whether there is evidence for a temporal dysfunction in a group of children with dyslexia has been specifically addressed by study E. Here, one aspect of auditory temporal processing, namely the discrimination of short- compared to long-transition speech syllables was investigated. Indeed, a subset of dyslexic children displayed limited performance on rapid stop-consonant syllables. Such problems were also detected in a subgroup of dyslexic individuals in study B: Dyslexic subjects who benefited behaviorally from prolongation of stop-consonant stimuli demonstrated enhanced MMF amplitude to temporally extended versus rapid syllable contrasts. However, this subtype of dyslexia (designated ‘benefiters’) did not show a weaker mismatch response to rapid stimulus changes than their normally literate age-mates. We have hypothesized that the benefiters’ neuronal discrimination ability might not be integrated into a voluntary response to short transitional stop-consonant syllables. Thus, the problem may not be confined to the sensory level, but also linked to output stages. Whether this neural activity profile reflects a deficit in processing temporal aspects of the speech signal or is rather associated with impairment in phonological decoding could not be settled here, however. Likewise, the experimental design used in studies A, C, and D did not permit a decision which of the two competing views accounts for the dyslexics’ atypical source configurations in temporal-lobe sites. For clarification of this controversy, a variety of task paradigms including the manipulation of the formant transition duration of the stop-consonant syllables as well as rapid and slowed non-speech stimuli would be required.

(2) Can the present neural correlates of dyslexia be traced to functional or rather to structural alterations?

Several aspects of the findings make one think of the neural deviances being functional rather than structural. Regarding the MMF results, ‘functionality’ is suggested to the effect that the MMF represents an interaction between neural responses to standard and those to deviant stimuli. Moreover, the changeability in mismatch topography in a small group of language-impaired children following a four-week training period may favor functional elements (see
study F, chapter II). Finally, observations of relative source deviations (i.e., M210 vs. M80 and P100m vs. N260m, see study A and C of this thesis, respectively) in children and adolescents with dyslexia are probably not reminiscent of gross-anatomical alterations. However, investigations at the microscopic level indicate that not only functional but also morphological changes are important when neurons alter their synaptic efficacy (Engert & Bonhoeffer, 1999). Specifically, the emergence of new dentritic spines can occur within only an hour after the induction of long term potentiation in a hippocampal slice culture. Thus, structural and functional elements seem to be interwoven and barely capable of disentanglement.

(3) Are the present cerebro-cortical deviances causally related to dyslexia or do they reflect a compensatory mechanism?

One promising way to clarify this issue would be the implementation of training programs tapping different aspects of literacy skills, such as audio-visual matching (e.g., Kujala et al., 2001), phonological skills (e.g., Hatcher & Hulme, 1999), or orthographic knowledge (e.g., Schulte-Körne et al., 2001b). MEG recordings before and after the training regimen would then indicate to what extent a specific intervention method might be capable of altering cortical organization in dyslexic individuals. This experimental approach will be pursued in our future studies. Children with dyslexia shall be assigned to one of three remediation programs lasting four weeks: (i) auditory sound discrimination following the computer-based training exercises of FastForWord Language™ [http://www.scilearn.com; see e.g., Merzenich et al., 1996; Tallal et al., 1996], (ii) syllabic speaking, reading, and writing according to the principle developed by H. Buschmann (for details see study F, chapter II), and (iii) the rule-based Marburg Spelling Training (German: Das Marburger Rechtschreibtraining) designed by Schulte-Körne and Mathwig (2001). Normally literate children matched for age, non-verbal intelligence, gender, and handedness constitute the control group. Like the training groups, the control subjects are tested twice. Behavioral assessment includes psychometric measures on literacy skills as well as categorical perception tasks. At the neural level, the MMF and other cortical auditory ERFs to stop-consonant syllables and non-speech analogues serve as dependent variables. As for the syllabic training, we expect a replication of the findings presented in study F, that is, significant improvements on language-related performance tests and changes towards a left-dominant MMF topography. The intensive and adaptive computer-based exercises involving modified verbal and non-verbal acoustic stimuli should bring about the most salient gains on phonological measures (e.g., pseudoword reading, categorical
perception of stop-consonant syllables). Moreover, this training is expected to yield changes on lateral asymmetry as indexed by the MMF as well as ERF sources localized in the latency range between 50 and 300 ms from stimulus onset. The rule-based spelling procedure should mainly provoke progresses in orthography and real-word reading, while central auditory functioning is assumed to remain unaffected. Possible cortical changes underlying the alleviation of dyslexia in the course of a specific training then suggest that the observed deviances in hemispheric laterality are causally linked to the disorder. In addition, differences in brain responses to speech syllables relative to non-speech stimuli may offer valuable information relevant to the controversy of whether dyslexia is based on a phonological deficit or a general auditory perceptual dysfunction.

**Implications: Elements for Functional Neuroanatomy of Dyslexia**

Based on the considerations presented above, I derive the following implications with regard to the functional neuroanatomy of developmental dyslexia:

- There is a *subset of dyslexic children and adolescents* who demonstrate limited discrimination performance on stop consonant-vowel syllables, but succeed when the transitional elements of the consonants are artificially lengthened. This may point to a deficit in auditory temporal processing.

- Auditory syllable processing is associated with deviations in *hemispheric asymmetry* in both children and adults with dyslexia. Thus, an absence of cerebral laterality may represent a stable rather than a maturational feature of dyslexia.

- Reduced or absent cerebral lateralization in people with dyslexia reflects deviances in *temporal-lobe sites*, possibly in primary auditory cortex and adjacent areas.

- Atypical organization in temporal regions is observed in both cerebral hemispheres in people with dyslexia, but predominantly in the *right hemisphere*.

- Altered cortical organization in children with language-based learning impairment may be *modifiable* through linguistic training in a therapeutic setting.

- The current work targeting the auditory domain suggests deviances primarily in the right supratemporal cortex in people with dyslexia. These observations complement functional neuroimaging studies indicating mainly a left-hemisphere deficit in dyslexia and characterize the *temporal dynamic* of the relevant brain processes.
IV. Summary

ABSTRACT
Psychoacoustic investigations have shown that many children with language-based learning disorders are at least mildly impaired in their reception of stop consonants, such as /b/, /d/, and /g/. The present thesis sought to examine behavioral and neurophysiological aspects of auditory processing in people with developmental dyslexia compared to normally literate controls. To this end, a series of psychophysical experiments and magnetoencephalographic recordings were conducted, involving stop consonant-vowel syllables. The auditory mismatch field (MMF) and other cortical event-related field (ERF) components served as dependent variables. As an important result, these studies pointed to deviances in hemispheric asymmetry of temporal-lobe sites in children and adolescents with dyslexia. Signal space analyses revealed an altered topographical distribution of the MMF, while source space analyses showed pronounced deviations in source positions of ERFs. These findings may reflect atypical organization of the primary auditory cortex and adjacent areas, in particular the planum temporale. Cross-sectional investigations indicate that a deviance in source configuration of magnetic waves around 100 ms after syllable presentation continue into adulthood. However, differences in hemispheric balance might be altered following linguistic training as suggested by a study in a small group of language-impaired children with symptoms of dyslexia. In addition to significant gains on performance measures of literacy skills, MMF responses showed a trend towards normalization after a four-week training period. A behavioral experiment inspired by the hypothesis of a temporal processing deficit for dyslexia suggests that limitations in auditory processing might be compensated by a well-functioning visual sensory modality: dyslexic children with poor discrimination performance on rapidly changing stop-consonant syllables reached high scores in a visual temporal-order task. Another study revealed two subgroups of dyslexic individuals differing in their discrimination ability of short and temporally extended stop consonant-vowel syllables both at the behavioral and neuronal level. Taken together, the studies indicate predominantly right-hemisphere deviances in children and adults with dyslexia. These observations complement hemodynamic studies of the human brain suggesting mainly a left-hemisphere dysfunction in dyslexia and characterize the temporal dynamic of the neural processes under consideration.
ZUSAMMENFASSUNG

Niños con problemas en el aprendizaje del lenguaje hablado y escrito, presentan con frecuencia dificultad en la percepción acústica de consonantes oclusivas (por ejemplo: /b/, /d/, y /g/). En esta tesis han sido estudiadas la conducta y la neurofisiología del procesamiento auditivo de personas con dislexia, y de un grupo control sin problemas en el dominio ya mencionado. Como métodos de análisis fueron realizados una serie de experimentos psicofísicos y magnetoencefalográficos, utilizando sílabas con consonantes oclusivas. Como variables dependientes fueron definidas el “mismatch field” (MMF) auditivo así como también otros campos magnéticos relacionados con eventos corticales (CRE). Los resultados indican una alteración de la asimetría hemisférica del lóbulo temporal de niños y adolescentes con dislexia. Los análisis del espacio de las señales biomagnéticas, revelaron un cambio en la distribución topográfica del MMF, mientras que los análisis del espacio de la fuente eléctrica demuestran una alteración de las localizaciones de la fuente de los CREs. Estos resultados podrían reflejar una organización atípica en el córtex auditivo primario y las regiones adyacentes, particularmente en la zona del plano temporal. Investigaciones transversales indican que alteraciones en la configuración de las fuentes eléctricas de los componentes magnéticos localizadas aproximadamente 100 ms post stimuli, pueden ser también identificadas en adultos. Sin embargo, las diferencias en la asimetría hemisférica parecen ser modificables por medio del uso de un entrenamiento lingüístico, como lo sugiere el estudio de un grupo pequeño de niños con problemas del lenguaje hablado y escrito. Además del mejoramiento en lectura y escritura, y en el procesamiento fonológico, después de un periodo de práctica de cuatro semanas, la distribución topográfica del MMF demostró una tendencia a la normalización. Un experimento conductual basado en la hipotésis del déficit de procesamiento temporal, sugiere que la reducción del procesamiento auditivo puede ser compensada por medio de una buena modalidad visual: los niños dislécticos con funcionamiento deficitario en la discriminación de sílabas compuestas de consonantes oclusivas rápidamente cambiantes, alcanzaron un alto rendimiento en una tarea visual de orden temporal. Otro estudio sugiere la existencia de dos subgrupos de individuos dislécticos. Éstos se diferenciaron, por una parte, en su capacidad de discriminación de sílabas cortas y temporalmente ampliadas a nivel conductual en consonantes oclusivas, y por otra parte, a nivel neuronal. En su conjunto, los estudios indican predominantemente alteraciones del
hemisferio derecho en niños y adultos con dislexia. Estas observaciones se complementan con aquellos resultados obtenidos en estudios de neuroimagen funcional, los que principalmente sugieren una disfunción del hemisferio izquierdo y además caracterizan la dinámica temporal del fenómeno observado.
V. References


Auditory Processing in Dyslexia – References


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