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Language impairment is reflected in auditory evoked fields

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Abstract

Specific language impairment (SLI) is diagnosed when a child has problems in producing or understanding language despite having a normal IQ and there being no other obvious explanation. There can be several associated problems, and no single underlying cause has yet been identified. Some theories propose problems in auditory processing, specifically in the discrimination of sound frequency or rapid temporal frequency changes. We compared automatic cortical speech–sound processing and discrimination between a group of children with SLI and control children with normal language development (mean age: 6.6 years; range: 5–7 years). We measured auditory evoked magnetic fields using two sets of CV syllables, one with a changing consonant /da/ba/ga/ and another one with a changing vowel /su/so/sy/ in an oddball paradigm. The P1m responses for onsets of repetitive stimuli were weaker in the SLI group whereas no significant group differences were found in the mismatch responses. The results indicate that the SLI group, having weaker responses to the onsets of sounds, might have slightly depressed sensory encoding.
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1. Introduction

Children with Specific Language Impairment (SLI) have problems in speaking or understanding despite normal cognitive development and peripheral hearing. Quite a large number (around 5%) of otherwise normally developing children suffer from this problem. The cause of SLI is not yet known. However, a variety of impairments potentially related to primary underlying deficits have been associated with SLI. These impairments, occurring at different cognitive levels, include deficits in discrimination of speech sounds (Burlingame et al., 2005), in working memory and phonological short-term memory, with a particularly marked deficit in non-word repetition (Bishop et al., 1999; Gathercole and Baddeley, 1990; Archibald and Gathercole, 2006; van Daal et al., 2007).

Many studies have revealed difficulties in lower-level auditory processing in SLI children. It has been claimed that the underlying problem is in processing rapidly changing temporal sequences (Tallal and Piercy, 1973; Tallal et al., 1985; Benasich and Tallal, 2002). Children with SLI also show a deficit in frequency discrimination (McArthur and Bishop, 2004a,b; Hill et al., 2005; Mengler et al., 2005). These problems, however, are not present in all children with SLI (e.g., Rosen, 2003; McArthur and Bishop, 2004a). In addition, other kinds of auditory processing deficits, such as problems in detecting amplitude envelope rise time and duration of simple tones may occur (Stollman et al., 2003; Corriveau et al., 2007).

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One way to address the underlying impaired brain mechanisms in SLI is to investigate the evoked brain responses of these children. Auditory evoked responses are an objective way to study the reactivity of the brain to sound. They can be measured without the subject paying attention to the stimuli, which is advantageous in studies of young children, particularly children with problems in understanding the test instructions. Both auditory evoked potentials (AEPs) and magnetic fields (AEFs) reflect the brain's activation pattern with millisecond time resolution. MEG is insensitive to inhomogeneities between the brain and the sensors, and selectively sensitive to tangential currents (Hämäläinen et al., 1993). MEG is particularly advantageous in studies where the activity from left and right auditory cortices is compared, because of the orientation of the magnetic field patterns, which are orthogonal to the patterns of potential distribution. Consequently, whereas electric potentials evoked by activity in the auditory cortices summate at the vertex and generate only one maximum on the head surface, magnetic field patterns are limited to one hemisphere.

The evoked auditory response sequence is characterized in scalp recordings as a series of positive and negative waves (P1-N1-P2-N2) that are robust and easily identified in adults. The most studied deflection is called N1 (N1m in MEG recordings, the 'm' referring to 'magnetic'). The N1/N1m is evoked by various changes in the auditory environment and it occurs at about 100 ms after the stimulus onset (Näätänen and Picton, 1987) with its generation site at the supratemporal auditory cortex (Hari et al., 1980).

The auditory evoked responses mature with age. The main pattern of the AEF response sequence seen in adults, P1m-N1m-P2m, with deflections peaking at about 50, 100 and 200 ms, respectively, is different in early childhood. The broad P1m deflection in newborns, peaking at around 250 ms (Lengle et al., 2001; Huotilainen et al., 2003; Pihko et al., 2004), becomes smaller with age and its latency decreases (Paetau et al., 1995). Paetau et al. (1995) studied children from 3 months to 15 years. A biphasic response, with P1m peaking at about 100 ms and an N1m peaking at about 260 ms occurred in children up to 12 years. The source strengths of P1m were two to three times stronger in children than in adults. The strength of the N1m in children depends on the inter-stimulus interval (ISI) (Rojas et al., 1998; Takeshita et al., 2002). N1m was not elicited in 21% of the subjects between 6 and 11 years with 1-kHz 100-ms tone delivered at 1.6-s ISI (Takeshita et al., 2002). The development of AEFs with age is in line with the maturation of the evoked potential components P1 and N1b with tangential sources (Sharma et al., 1997; Cunningham et al., 2000; Ponton et al., 1996, 2000, 2002). Consequently, between-studies comparison in young children of the effect of SLI on these brain responses, reflecting the processing of sensory stimulus features, may be complicated by developmental transition of AEPs and AEFs, and of their sensitivity to stimulation parameters.

The effect of SLI on the N1 has been investigated in several studies in children of varying ages. The results indicated mostly either enhanced N1 responses in children with SLI compared to control children, or no differences in the N1 of the two groups (Neville et al., 1993; Lincoln et al., 1995; Tonnquist-Uhlén et al., 1996; Shafer et al., 2000; Marler et al., 2002; Ors et al., 2002). The effect of SLI on P1 has been less frequently studied; however, the results are also inconsistent across various studies. Rinker et al. (2007) did not find differences in the sensory responses (P1 and N250) of SLI and control groups (7–11 years) either to the standards (700 Hz) or deviants (750 Hz). However, SLI children did not show latency decrease with age whereas the control children did. Oram Cardy et al. (2005) studied AEFs to paired tones separated by a 150 ms gap and found that the M50 and M100 (corresponding to P1 and N1) evoked by the second, but not the first tone, were identified in significantly fewer 8–17-year old children with SLI than children with typical language development. Mills and Neville (1997) studied P100 to speech sounds in infants 13–30 months of age, and found that the laterality effect present in control children was not found in late talkers, considered to be at risk for SLI.

Cunningham et al. (2000) studied the maturational change of the P1-N1-N2 response sequence as a function of age and found no differences in the P1 and N1 responses between normal and learning impaired children (8–15 years old, not strictly SLI); neither was there a relation between behavioral discrimination along a /da/–/ga/ continuum and P1-N1-N2. However, the P1-N1-N2 parameters were significantly correlated with standardized tests of spelling, auditory processing, and listening comprehension in the learning impaired children. McArthur and Bishop (2004b, 2005) found, however, age-inappropriate N1-P2 responses in SLI children regardless of their auditory frequency discrimination performance, and suggested that maturational status might be a key factor in explaining differences between SLI children.

Another widely studied evoked component is the mismatch negativity (MMN). It occurs after or parallel with N1 and reflects automatic sound discrimination (Näätänen, 1992). It can be recorded as an enhanced negativity evoked by an occasional deviating stimulus in a sequence of frequently repeated 'standard' stimuli. MMN has recently gained attention in studies on language problems. As its amplitude has been shown to correlate, for example, with sound frequency discrimination (Tiitinen et al., 1994; Novitski et al., 2004) a lower MMN amplitude would reflect poorer sound discrimination. Indeed, reduced MMN amplitude to tone frequency changes (standard 500 Hz, deviant 553 Hz) in children with SLI was reported by Korpilahti and Lang (1994) and in subsequent replications of the study with subjects of varying ages (Korpilahti, 1995; Holopainen et al., 1997, 1998). Uwer et al. (2002) found an attenuated MMN to syllables (/da/ as standard, /ga/ and /ba/ as deviants), but not to a frequency change of tones (standard 1000 Hz, deviant 1200 Hz). Rinker et al. (2007) suggested that the magnitude of the frequency change, 200 Hz, in the study by Uwer et al. (2002), was too large to be difficult for the SLI group. In their own study the tone change from 700 Hz to 750 Hz evoked an MMN at about 200 ms in SLI children only at Fz but not at Cz, whereas the MMN was seen in both electrode locations in the control group (Rinker et al., 2007). Marler et al. (2002) found differences in the MMN latency between SLI and control children, whereas the attenuation of the MMN amplitude in the SLI children, evident in the grand averages, was not statistically significant.

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