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Speech and non-speech processing in people with specific language impairment: A behavioural and electrophysiological study

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Abstract

McArthur and Bishop (2004) found that people with specific language impairment (SLI) up to 14 years of age have poor behavioural frequency discrimination (FD) thresholds for 25-ms pure tones, while people with SLI upto 20 years of age have abnormal auditory N1–P2–N2 event-related potential (ERP) responses to the same tones. In the present study, we extended these findings to more complex non-speech and speech sounds by comparing younger (around 13 years) and older (around 17 years) teenagers with SLI and controls for their behavioural FD thresholds and N1–P2 ERPs to 25 and 250-ms pure tones, vowels, and non-harmonic complex tones. We found that a subgroup of people with SLI had abnormal responses to tones and vowels at the level of behaviour and the brain, and that poor processing was associated with the spectral complexity of auditory stimuli rather than their phonetic significance. We suggest that both the age of listeners and the sensitivity of psychoacoustic tasks to age-related changes in auditory skills may be crucial factors in studies of sound processing in SLI.

Keywords: Specific language impairment; Frequency discrimination; Vowel discrimination; Event-related potentials

1. Introduction

Around 5% of children have problems understanding or producing spoken language despite having normal general cognitive abilities and no medical problems. The cause of this condition, known as specific language impairment (SLI), is not yet known. In fact, there may be no single cause. SLI may result when a number of risk factors co-occur (Bishop, Carlyon, Deeks, & Bishop, 1999). These risk factors may include deficient short-term verbal memory (Gathercole, 1993), a limited processing capacity (Leonard, 1996), and an impaired ability to acquire grammatical rules (Rice, Wexler, & Redmond, 1999). Another potential risk factor for SLI is poor auditory processing, which could affect the ability to discriminate between speech sounds. This may result in less stable or specific neural representations of speech sounds, which may ultimately interfere with perceiving and producing spoken language.

One aspect of auditory processing that is receiving increasing attention in studies of language impairment is frequency discrimination (FD). Although most research has focused on discrimination of brief or rapid stimuli (see Tallal, 2000; for review), some recent studies have found clear evidence for poor FD in children with SLI when there is no stress on rapid processing (Korpilahti, 1995; Ors et al., 2002; Uwer, Albrecht, & von Suchodoletz, 2002). For example, Mengler, Hogben, Michie, and Bishop (in press) found that a group of 9- to 12-year-olds with SLI were significantly poorer than age-matched controls at discriminating between the frequencies of 100-ms pure tones. When most of these children were re-tested $2\frac{1}{2}$ years later by Hill, Hogben, and Bishop (in press), the FD thresholds of the SLI group

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had improved, but were still significantly poorer than the control group.

Other behavioural studies have found less clear evidence for poor FD in SLI. McArthur and Bishop (2004) found that only five out of sixteen 9- to 20-year-old teenagers with SLI had poorer FD thresholds than controls. When they re-tested most of these people $1\frac{1}{2}$ years later, two people with poor FD thresholds were still impaired while two others had improved to reach the normal range (Bishop & McArthur, in press). These variable findings could reflect heterogeneity of SLI: An FD deficit may be only one of a number of risk factors for SLI, so only a subgroup of people with SLI would perform poorly on behavioural tests of FD. However, another possibility suggested by our data (Bishop & McArthur, in press; Hill et al., in press) is that poor FD improves with age. A similar argument was recently put forward by Wright and Zecker (2004) to account for varied behavioural findings on auditory deficits in children with language and learning disabilities. They suggest that these children have a maturational lag in auditory discrimination of about 3–4 years. Whether an auditory deficit is seen in SLI will depend both on the age of the sample and the developmental trajectory of the specific auditory function that is tested. Wright and Zecker (2004) further postulated that auditory maturation ceases at puberty. This would explain why delayed auditory processing in some people with SLI does not catch up by early adulthood.

One limitation of behavioural studies of FD in people with SLI is that poor scores on behavioural FD tests could result from poor attention or low motivation as well as an FD deficit. Event-related potentials (ERPs) can be used to measure auditory processing without the attention of a listener. Auditory ERPs represent the average pattern of electrical activity emitted by groups of neurons in response to a sound. This activity, which is thought to reflect post-synaptic activation, is measured using electrodes that are placed on a listener's scalp. These electrodes detect the electrical activity that is present immediately after the onset of a sound. When many exemplars of the same sound are presented, the ratio of activity generated by a sound (signal) compared to activity generated by other factors (noise) becomes great enough for an auditory ERP waveform to emerge for that sound (see Fig. 2 for examples of ERP waveforms). The first and second positive peaks in the auditory ERP waveform are called P1 and P2, respectively. The first and second negative peaks are called N1 and N2, respectively. The locations of the neurons that produce these peaks are not fully understood. Some evidence suggests that P1 stems from the secondary auditory cortex (Liegeois-Chauvel, Musolino, Badier, Marquis, & Chauval, 1994); N1 originates from multiple sources that include the primary auditory cortex, the posterior–superior temporal plane, and non-specific frontal regions (Bruneau & Gomot, 1998; though cf Liegeois-Chauvel et al., 1994); P2 originates from the mesencephalic reticular activating system (Ponton, Eggermont, Kwong, & Don, 2000); and N2 is produced by cells in the superior temporal gyrus and medial temporal lobes (O'Donnell et al., 1993).

A handful of studies have tested people with SLI for their auditory ERPs to non-speech sounds. Some have found that people with SLI, as a group, had abnormal N1 responses (Lincoln, Courchesne, Harms, & Allen, 1995; Tonnquist-Uhlen, 1996; Tonnquist-Uhlen, Borg, Persson, & Spens, 1996) and P2 responses to tones (Adams, Courchesne, Elmasian, & Lincoln, 1987; Tonnquist-Uhlen, 1996). Neville, Coffey, Holcomb, and Tallal (1993) found that a subgroup of their children with SLI, who had poor performance on a behavioural auditory processing measure, had abnormal N1 and N2 responses to tones under certain conditions. However, other studies of SLI have found normal N1 or P2 responses to tones (Courchesne, Lincoln, Yeung-Courchesne, Elmasian, & Grillon, 1989; Marler, Champlin, & Gillam, 2002; Mason & Mellor, 1984; Ors et al., 2002).

It is difficult to explain these contradictory findings. There is no obvious difference between the studies that did and did not find abnormal auditory ERPs people with SLI in terms of stimulus type, inter-stimulus interval (ISI), or number of stimuli. However, these studies do differ slightly in sample size, with studies finding abnormal ERPs in people with SLI using larger groups. There may also be a difference in the types of people recruited for the SLI groups. Three of the five studies that found abnormal ERPs in SLI noted that their participants had particularly severe learning disabilities (i.e., Tonnquist-Uhlen, 1996; Tonnquist-Uhlen et al., 1996; Neville et al., 1993). For example, the two studies by Tonnquist-Uhlen and colleagues tested children with mild to moderate delays in mental function, whose reference values for their intelligence quotient were adjusted to the 8-year-old level (the mean age of the children was 11.9 years). Nine of their 20 children with SLI had pathological EEGs and two were border-line. Thus, this study may have tested children with more global cognitive impairments who would not usually meet the typical criteria for SLI.

Age is another factor that might explain why some studies find abnormal ERPs in people with SLI while others did not. It has been suggested that an auditory processing deficit present early in life may resolve with age (Farmer & Klein, 1995). We know that the studies that did and did not find abnormal ERPs in people with SLI had comparable age-ranges across studies (8–15 years and 7–16 years, respectively). However, we do not know the proportion of children at each age in each study. It is possible that the studies that found abnormal ERPs in children with SLI had a higher proportion of young children in their SLI group than studies that found normal ERPs in children with SLI.