



Atypical central auditory speech-sound discrimination in children who stutter as indexed by the mismatch negativity



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ABSTRACT

Purpose: Recent theoretical conceptualizations suggest that disfluencies in stuttering may arise from several factors, one of them being atypical auditory processing. The main purpose of the present study was to investigate whether speech sound encoding and central auditory discrimination, are affected in children who stutter (CWS).

Methods: Participants were 10 CWS, and 12 typically developing children with fluent speech (TDC). Event-related potentials (ERPs) for syllables and syllable changes [consonant, vowel, vowel-duration, frequency (F0), and intensity changes], critical in speech perception and language development of CWS were compared to those of TDC.

Results: There were no significant group differences in the amplitudes or latencies of the P1 or N2 responses elicited by the standard stimuli. However, the Mismatch Negativity (MMN) amplitude was significantly smaller in CWS than in TDC. For TDC all deviants of the linguistic multifeature paradigm elicited significant MMN amplitudes, comparable with the results found earlier with the same paradigm in 6-year-old children. In contrast, only the duration change elicited a significant MMN in CWS.

Conclusions: The results showed that central auditory speech-sound processing was typical at the level of sound encoding in CWS. In contrast, central speech-sound discrimination, as indexed by the MMN for multiple sound features (both phonetic and prosodic), was atypical in the group of CWS. Findings were linked to existing conceptualizations on stuttering etiology.

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Educational objectives: The reader will be able (a) to describe recent findings on central auditory speech-sound processing in individuals who stutter, (b) to describe the measurement of auditory reception and central auditory speech-sound discrimination, (c) to describe the findings of central auditory speech-sound discrimination, as indexed by the mismatch negativity (MMN), in children who stutter.

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1. Introduction

Neural bases of stuttering have been intensively studied recently. The pathophysiology and neural bases underlying developmental stuttering, however, still remains poorly understood. Contemporary theories of stuttering incorporate many factors, like atypical neurophysiology, genetics, personality, linguistic factors, and atypical auditory processing (Bloodstein & Bernstein Ratner, 2008; Hall & Jerger, 1978; Liotti et al., 2010). We focus in this study on central speech-sound processing since accuracy of this function is essential for speech acquisition, production, and comprehension (Jansson-Verkasalo et al., 2003, 2010; Kuhl & Rivera-Gaziola, 2008).

Auditory processing difficulties may be subtle in nature in individuals with stuttering, and therefore may not manifest themselves in standardized behavioral tests (Kaganovich, Wray, & Weber-Fox, 2010). Auditory event-related potentials (ERPs) provide the necessary temporal and spatial resolution to detect subtle differences in auditory processing, and can be used to investigate well-defined stages of central auditory processing. Auditory ERPs are minute and discrete electrical potentials in the electroencephalogram (EEG) and are manifestations of neural activity that is specifically related, or time-locked, to sensory stimulation (Stapells & Kurzberg, 1991). The ERP waveform consists of a sequence of positive (P) and negative (N) deflections or peaks that are named according to their polarity and latency (timing relative to the stimulus onset), their serial order or cognitive meaning (Näätänen, 1992). Early components of the auditory ERPs reflect the neural correlates of reception and encoding of a stimulus. A P1-N1b-P2-N2 complex is typical in adults, P1 having a latency of about 50 ms (Ponton, Eggermont, Kwong, & Don, 2000). In children, however, the early stages of sound-feature encoding are reflected by obligatory P1-N2-N4 responses (Choudhury & Benasich, 2011). The P1 response latency decreases rapidly during the first decade of life from about 200–80 ms (Sharma, Dorman, & Spahr, 2002). P1 is followed by a broad negativity, N2, at about 200 ms (Čeponienė et al., 2001; Čeponienė, Rinne, & Näätänen, 2002; Niemitalo-Haapola et al., 2013). N1b is elicited in children only with long interstimulus intervals (Čeponienė et al., 2002) and becomes progressively consistent from the age of ten years onwards (Ponton, Eggermont, Khosla, Kwong, & Don, 2002). The language-related, negative-going N400 wave is an index of lexical access and integration (for review, see Kutas & Federmeier, 2011, for review), while the positive P600 is linked to the processing of syntactic violations (Friederici, 2002) or difficulty of syntactic integrations (Kaan, Harris, Gibson, & Holcomb, 2000). The magnitude (amplitude), speed (latency) and the location of processing reflect the efficacy of neural functions.

Central auditory discrimination can be investigated with the Mismatch Negativity (MMN) component of the auditory ERPs (Näätänen, 1992; Näätänen, Kujala, & Winkler, 2011). MMN is elicited even in inattentive subjects by potentially discriminable deviances in repetitive aspects of auditory stimuli (Näätänen, 1992; Winkler, 2007), and its latency and amplitude correlate with behavioral discrimination accuracy (Kujala & Näätänen, 2010; Kujala et al., 2001; Tiitinen, May, Reinikainen, & Näätänen, 1994).

Neuroanatomical (Beal, Gracco, Lafaille, & De Nil, 2007; Watkins, Smith, Davis, & Howell, 2008), as well as neurophysiological methods (Corbera, Corral, Escera, & Idiazábal, 2005; De Nil, Kroll, & Houle, 2001; De Nil et al., 2008; Hampton & Weber-Fox, 2008; Liotti et al., 2010) have been used to study auditory processing in adults who stutter. ERP studies (Corbera et al., 2005; Hampton & Weber-Fox, 2008; Liotti et al., 2010) have shown that auditory processing in adults who stutter is atypical in response to speech stimuli (Liotti et al., 2010) and in response to tones (Weber-Fox & Hampton, 2008) when compared to the controls. Similarly, a number of brain imaging studies have shown reduced (Chang, Kenney, Loucks, & Ludlow, 2009; De Nil et al., 2008; Fox et al., 2000; Ingham, Fox, Ingham, & Zamarripa, 2000) or increased activation (De Nil, Kroll, Kapur, & Houle, 2000; Kell et al., 2009; Neumann et al., 2003) in auditory areas in adults who stutter when compared to fluent speakers during a variety of speech tasks suggesting altered auditory processing. It has also been found that fluency-inducing therapies increase temporal activations (Fox et al., 1996; Neumann et al., 2003) which further support the assumption that temporal regions are part of a cortical–subcortical system. This suggestion is also corroborated by the study of Chang et al. (2009). Chang et al. (2009) investigated adults who stutter using fMRI during speech and non-speech perception, speech planning, and fluent production without masking. They found that adults who stutter had less activation in the frontal and temporoparietal regions relative to the controls during both speech and non-speech perception and planning. During speech and non-speech production, adults who stutter had less activation than the controls in the left superior temporal gyrus and the left pre-motor areas but greater activation in the right superior temporal gyrus, bilateral Heschl's gyrus, insula, putamen, and precentral motor regions. In addition, hemispheric differences in auditory processing have been reported in a number of studies between individuals who stutter and fluently speaking controls (Chang, Erickson, Ambrose, Hasegawa-Johnson, & Ludlow, 2008; Lu et al., 2010). While the precise nature of these differences is not clear, earlier suggestions that they may be related to atypical auditory inhibition, have not been confirmed (Beal, 2010; Beal et al., 2011). In a

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