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Speech-induced suppression of evoked auditory fields in children who stutter

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ABSTRACT

Auditory responses to speech sounds that are self-initiated are suppressed compared to responses to the same speech sounds during passive listening. This phenomenon is referred to as speech-induced suppression, a potentially important feedback-mediated speech-motor control process. In an earlier study, we found that both adults who do and do not stutter demonstrated a reduced amplitude of the auditory M50 and M100 responses to speech during active production relative to passive listening. It is unknown if auditory responses to self-initiated speech-motor acts are suppressed in children or if the phenomenon differs between children who do and do not stutter. As stuttering is a developmental speech disorder, examining speech-induced suppression in children may identify possible neural differences underlying stuttering close to its time of onset. We used magnetoencephalography to determine the presence of speech-induced suppression in children and to characterize the properties of speech-induced suppression in children who stutter. We examined the auditory M50 as this was the earliest robust response reproducible across our child participants and the most likely to reflect a motor-to-auditory relation. Both children who do and do not stutter demonstrated speech-induced suppression of the auditory M50. However, children who stutter had a delayed auditory M50 peak latency to vowel sounds compared to children who do not stutter indicating a possible deficiency in their ability to efficiently integrate auditory speech information for the purpose of establishing neural representations of speech sounds.

Introduction

Stuttering is a developmental disorder defined by frequent and involuntary repetitions and/or prolongations of sounds as well as silent blocks that disrupt speech fluency and is prevalent in approximately 5% of preschool children (Yairi and Ambrose, 1999). The onset of the disorder typically occurs between 2 and 5 years of age (Bloodstein and Ratner, 2008). There is evidence for a genetic aetiology of developmental stuttering (Ambrose et al., 1997; Howie, 1981; Kang et al., 2010; Kidd et al., 1981; Lan et al., 2009; Riaz et al., 2005; Suresh et al., 2006; Wittke-Thompson et al., 2007). There are also various neuroanatomical (Beal et al., 2007; Foundas et al., 2001, 2004; Jäncke et al., 2004; Kell et al., 2009; Sommer et al., 2002; Song et al., 2007; Watkins et al., 2008) and neurophysiological (Blomgren et al., 2003; Braun et al., 1997; Chang et al., 2009; De Nil et al., 2000, 2001, 2008; Fox et al., 1996, 2000; Giraud et al., 2008; Kell et al., 2009; Lu et al., 2009; Neumann et al., 2003, 2005; Preibisch et al., 2003; Watkins et al., 2008) differences that have been observed in adults who stutter relative to fluent speakers. To our knowledge, only two studies have examined the neural correlates of stuttering in children (Chang et al., 2008; Weber-Fox et al., 2008). Given that stuttering typically has its onset in the preschool years there is a great deal to be gained from increasing our understanding of the neural signatures of this disorder early in its presentation and development.

Chang et al. (2008) investigated neuroanatomical differences in children who stutter relative to non-stuttering and recovered-from-stuttering peers. Similar to adults who stutter, children who stutter were found to have deficient white matter connectivity, as measured by fractional anisotropy, underlying areas near the left ventral premotor and motor cortices. However, children who stutter also differed from their age-matched fluently speaking peers in a unique

Abbreviations: fMRI, functional magnetic resonance imaging; MEG, magnetoencephalography; PET, position emission tomography.

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way relative to previous reports of differences between adults who stutter and their fluently speaking peers. Chang et al. (2008) reported that children who stutter had reduced grey matter volume compared to children who do not stutter in the left inferior frontal gyrus and bilateral middle temporal regions. Conversely, adults who stutter have been found to have increased grey matter in the left inferior frontal gyrus and bilateral superior temporal regions, including the primary auditory cortex (Beal et al., 2007; Song et al., 2007). However, Kell et al. (2009) found reduced grey matter in the left inferior frontal gyrus in adults who stutter as well as in former stutterers who had recovered from stuttering.

Weber-Fox et al. (2008) measured event-related potentials (ERPs) of children who stutter and fluent children in a visual rhyming task. Children who stutter demonstrated lower accuracy on rhyming judgments relative to fluent children. However, the children who stutter did not differ from fluent children in the ERP component associated with the rhyming effect in this task. Instead, children who stutter demonstrated differences from fluent children in the contingent negative variation and N400. These components reflect anticipation and semantic incongruity. Weber-Fox et al. (2008) concluded that the neural profile of children who stutter suggested inefficient phonological rehearsal and target anticipation for rhyming judgment, and that children who stutter may have difficulty forming the phonological neural representations needed for accurate and efficient rhyming judgments. Further exploration is required to understand if differences in neural functioning between children who stutter and fluent children impact the early auditory processing for integrating feedback into upcoming speech-motor commands.

A central finding of previous functional neuroimaging studies of speech production in adults who stutter is a reduction in auditory cortex activation, in the presence of increased speech-motor cortex activation, relative to that of fluently speaking adults (De Nil et al., 2008; Fox et al., 1996, 2000; Watkins et al., 2008; but see Kell et al., 2009). Consequently, several researchers have posited that the interaction between motor and auditory cortices may be abnormal in adults who stutter (Brown et al., 2005; Ludlow and Loucks, 2003; Max et al., 2004; Neilson and Neilson, 1987). Specifically, some studies have proposed that stuttering may arise from difficulties controlling speech acts due to faulty neural representations of speech processes in the brain (Corbera et al., 2005; Max et al., 2004; Neilson and Neilson, 1987). A crucial aspect of normal speech acquisition is the gradual transition of control of speech-motor movement from a feedback-biased to feedforward-biased mechanism during development (Bailly, 1997; Guenther and Bohland, 2002; Guenther, 2006). Difficulty developing the neural processes for speech in childhood may interfere with the transition of speech-motor control from a predominant feedback to a more feedforward mode and contribute to the onset of stuttering (Civier et al., 2010; Max et al., 2004; Neilson and Neilson, 1987).

Further insight into the relation between motor and auditory cortical regions may be gained from the study of speech-induced auditory suppression, a mechanism related to this interaction. Speech-induced auditory suppression is a normal neurophysiological process thought to be related to the monitoring, and subsequent modification of, the auditory targets associated with speech-motor acts (Beal et al., 2010; Heinks-Maldonado et al., 2006; Houde et al., 2002; Numminen et al., 1999; Tourville et al., 2008). Various models of speech-motor control posit that projections from motor-related areas to auditory cortex relay information concerning the auditory target region for the speech sound under production (Guenther, 2006; Houde et al., 2002; Kröger et al., 2009; Ventura et al., 2009). The auditory target is compared to the actual auditory feedback and if there is correspondence then the incoming auditory signal is suppressed. If the auditory feedback is outside the range of the predicted auditory target then an error is detected and corrective motor commands are issued to the motor cortex (Heinks-Maldonado et al., 2006; Tourville et al., 2008).

Speech production, from conceptual formulation to articulation, is completed in approximately 600 ms (Levelt, 2004; Sahin et al., 2009). On average, adults are able to produce 5 syllables per second when asked to speak at a fast rate (Tsao and Weismer, 1997). Auditory feedback can be used to modify speech production within a time period ranging from 81 to 186 ms (Tourville et al., 2008). Millisecond level information about the sequence of cortical events comprising speech production is crucial for understanding the interaction between motor execution and auditory feedback of self-generated speech. The aforementioned investigations of speech production in adults who stutter used either positron emission tomography (PET) or functional magnetic resonance imaging (fMRI) which are limited in their ability to resolve temporal events occurring over periods shorter than several seconds. However, magnetoencephalography (MEG) is able to measure neural events with millisecond temporal resolution combined with good spatial resolution. MEG has been used to demonstrate that speech-induced related suppression of auditory activation can be detected as early as within 50 to 100 ms of vocalization in adults (Beal et al., 2010; Curio et al., 2000; Houde et al., 2002; Numminen et al., 1999).

We have reported that adults who stutter had shorter auditory M50 and M100 latencies in response to the self-generated vowel /i/ and vowel-initial words in the right hemisphere relative to the left hemisphere whereas adults who do not stutter showed similar latencies across hemispheres (Beal et al., 2010). These timing differences were observed in adults who stutter despite similar levels of auditory M50 and M100 peak amplitude reduction during active generation relative to controls. In other words, speech-induced auditory suppression resulted in peak latency differences in the adults who stutter relative to fluently speaking adults rather than peak amplitude differences. The neural timing differences may reflect inefficient access to the neural representations of speech processes, or compensation for such a deficit, in adults who stutter.

In adults, the M100 (N1 in EEG/ERP studies) is the most robust and reproducible auditory component across participants (Bruneau and Gomot, 1998). Therefore, the main emphasis of MEG studies of auditory evoked responses has been the M100 (Mäkelä, 2007). However, in children the morphology of the waveforms are often different such that the M50 is at a prolonged latency and more robust and reproducible across child participants relative to adults (Oram Cardy et al., 2004). From early childhood through to adulthood the evoked response morphology in MEG and EEG gradually changes, such that the M50 becomes less robust and reproducible and the M100 becomes more so (Bruneau and Gomot, 1998; Gage et al., 2003; Kotecha et al., 2009; Oram Cardy et al., 2004; Paetau et al., 1995; Picton and Taylor, 2007; Rojas et al., 1998). Furthermore, the M50 and M100 have been shown to have a common source in the primary auditory cortex (Hari et al., 1987; Kanno et al., 2000; Mäkelä and Hari, 1987). Functionally, both the M50 and M100 are known to change in amplitude and/or latency in response to manipulations of auditory stimuli characteristics such as amplitude, pitch or interstimulus interval (Roberts et al., 2000). Given that speech is a rapid and dynamic motor process, it follows that the underlying neural system supporting it must respond in a timely, precise and sequential manner to ensure its correct production (Guenther, 2006; Ludlow and Loucks, 2003; Tourville et al., 2008; Tsao and Weismer, 1997). Therefore, it is reasonable to predict that the neural correlates of auditory feedback processing of self-generated speech will be reflected in the first measureable and reproducible auditory response component across children. The main goal of the current study was to understand the differential effects of speech-induced auditory suppression in children who stutter and in age-matched fluently speaking peers. The first observable and reproducible auditory component, namely the M50, is the focus of investigation as it is most likely to reflect early motor–auditory interaction in children ages 6 to 12 years old.

Despite auditory feedback of self-generated speech signals being crucial to the normal development of speech-motor control (Callan