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Modulation of auditory processing during speech movement planning is limited in adults who stutter

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ABSTRACT

Stuttering is associated with atypical structural and functional connectivity in sensorimotor brain areas, in particular premotor, motor, and auditory regions. It remains unknown, however, which specific mechanisms of speech planning and execution are affected by these neurological abnormalities. To investigate pre-movement sensory modulation, we recorded 12 stuttering and 12 nonstuttering adults' auditory evoked potentials in response to probe tones presented prior to speech onset in a delayed-response speaking condition vs. no-speaking control conditions (silent reading; seeing nonlinguistic symbols). Findings indicate that, during speech movement planning, the nonstuttering group showed a statistically significant modulation of auditory processing (reduced N1 amplitude) that was not observed in the stuttering group. Thus, the obtained results provide electrophysiological evidence in support of the hypothesis that stuttering is associated with deficiencies in modulating the cortical auditory system during speech movement planning. This specific sensorimotor integration deficiency may contribute to inefficient feedback monitoring and, consequently, speech dysfluencies.

1. Introduction

Stuttering is a disorder of speech fluency associated with abnormal brain *activation* in a widespread network of pre-motor, motor, and sensory regions (Braun et al., 1997; Chang, Kenney, Loucks, & Ludlow, 2009; De Nil, Kroll, Kapur, & Houle, 2000; De Nil, Kroll, Lafaille, & Houle, 2003; Fox et al., 1996; Neumann et al., 2003; Watkins, Smith, Davis, & Howell, 2008). Across individual stuttering subjects, the involvement of specific brain regions appears to vary considerably (Ingham, Wang, Ingham, Bothe, & Grafton, 2013; Wymbs, Ingham, Ingham, Paolini, & Grafton, 2013). Nevertheless, several *structural* brain abnormalities have been reported, and these abnormalities include atypical white matter in pathways suggested to connect speech motor and auditory regions (Chang, Erickson, Ambrose, Hasegawa-Johnson, & Ludlow, 2008; Chang, Horwitz, Ostuni, Reynolds, & Ludlow, 2011; Cykowski, Fox, Ingham, Ingham, & Robin, 2010; Foundas et al., 2003; Sommer, Koch, Paulus, Weiller, & Buchel, 2002; Watkins et al., 2008). Accordingly, some of the most prominent contemporary theoretical views of stuttering suggest that the disorder may result from deficits in specific processes of sensorimotor integration that are critical for both early speech motor learning and mature speech motor control (Beal et al., 2010; Brown, Ingham, Ingham, Laird, & Fox, 2005; Cai et al., 2012; Chang et al., 2011; Daliri, Prokopenko, & Max, 2013; Hickok, Houde, & Rong, 2011; Kell et al., 2009; Liotti et al., 2010; Max, 2004; Watkins et al., 2008).

One aspect of sensorimotor integration that may be of particular theoretical importance in this regard is the central nervous system's (CNS) *prediction* of the sensory consequences (or, more generally, movement outcomes) of planned motor commands. In a recent study (Daliri, Prokopenko, Flanagan, & Max, 2014), we found that individuals who stutter accurately predict specific movement consequences in a ballistic reaching task (i.e., arm movements completed without relying on afferent feedback) in which those consequences could be fully compensated through anticipatory adjustments during movement planning. However, based on our overall theoretical framework (Max, 2004), stuttering individuals may be more likely to have difficulties with appropriately using such predictions to successfully "prime" task-relevant *sensory systems* for their subsequent role in (a) closely monitoring afferent inputs for online feedback control while (b) simultaneously preventing feedback-based motor responses that are undesirable during self-generated voluntary movements

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(note that the latter part of this hypothesis overlaps with ideas proposed by Zimmermann, 1980). Limited evidence consistent with this hypothesis was already provided by McClean (1996) who demonstrated that, as compared with fluent speakers, stuttering adults show less attenuation of mechanically-evoked lip muscle reflexes prior to the onset of speech (with lip muscle activity measured in speech trials vs. no speech trials). When participating sensory systems (auditory, somatosensory) are insufficiently modulated in terms of their response to self-generated afferent inputs, the triggered motor responses may interfere with, and disrupt, ongoing movements. To date, however, it remains completely unknown (a) whether stuttering individuals' atypical sensorimotor responses at speech onset are in fact due to a lack of *central modulation* of sensory neural systems, and, if so, (b) whether stuttering individuals show a lack of pre-speech sensory modulation in the *auditory cortical regions* that have been implicated in several, although not all, neuroimaging studies (see above).

Here, we addressed both these questions directly by using electroencephalographical (EEG) data and auditory evoked potential analyses to investigate, in stuttering vs. nonstuttering adults, the modulation of auditory cortical activity in response to probe tones presented prior to speaking (i.e., during speech movement planning) and in control conditions without preparation for motor activity.[1] Using an experimental paradigm that we previously developed for work with typically fluent speakers (Max, Daniels, Curet, & Cronin, 2008), we recorded long latency auditory evoked potentials (LLAEPs) in response to auditory stimuli presented during the delay phase of a delayed-response *speaking* task (seeing a word on a monitor, silently reading the word, and saying it aloud after a *go* signal), a *silent reading* task (seeing a word and silently reading it), and a *seeing* task (seeing nonlinguistic symbols). We also recorded the same LLAEPs in a standard eyes-closed rest condition to compare both groups in terms of basic auditory processing in the absence of an active task, and to verify, through comparison with these reference data, the validity of data processing and analysis procedures used in the three active tasks. Analyses focused on the amplitude and latency of the LLAEP components N1 and P2.[2] We hypothesized that if stuttering is associated with a lack of modulation of auditory cortical regions prior to speech onset, the stuttering group would fail to show the typical within-subject N1 amplitude attenuation that we have previously documented for normally fluent speakers (Max et al., 2008).

2. Methods

2.1. Participants

Twelve right-handed stuttering adults (eleven men and one woman; M_{age} = 27.32 years, age range: 18–46 years) and twelve right-handed nonstuttering adults (eleven men and one woman; M_{age} = 27.25 years, age range: 19–45 years) participated in the experiment after providing informed consent. Nonstuttering participants were individually matched with the stuttering participants based on age (±3 years) and sex. All participants were naive to the purpose of the study.

Eligibility criteria for all participants included (a) being a native speaker of American English, (b) self-reported absence of psychological, neurological, or communication disorders (other than stuttering in the stuttering group), (c) not taking any medications with possible effects on sensorimotor functioning, and (d) pure tone behavioral hearing thresholds at or below 20 dB HL at all octave frequencies from 250 Hz to 8 kHz in both ears.

Using the Stuttering Severity Instrument, Fourth Edition (SSI–4; Riley, 2008), each stuttering participant's severity was determined by an American Speech-Language-Hearing Association-certified speech-language pathologist. Individual participant information for the stuttering group (age, sex, handedness, overall SSI–4 score, stuttering severity classification, and frequency of stuttering averaged across the SSI–4 speaking and reading tasks) are presented in Table 1.

2.2. Procedure and instrumentation

The experiment was conducted inside a sound-attenuated room. Wearing an electrode cap (details given below), participants were seated approximately 1 m from a 23-in. liquid crystal display (LCD) monitor with a refresh rate of 60 Hz. Participants' speech output was transduced and amplified (WL185, Shure Incorporated, Niles, IL; DPS II, ART ProAudio, Niagara Falls, NY) and, after amplification by a headphones amplifier (S-phone, Samson Technologies Corp., Syosset, NY), played-back to the participant in real-time through insert earphones (ER-3A, Etymotic Research Inc., Grove Village, IL). The insert earphones were also used to deliver binaural auditory stimuli (1 kHz, 50 ms duration, 10 ms rise/fall time, 75 dB SPL) during some trials. Before each recording session, this feedback system was calibrated such that speech input with an intensity of 75 dB SPL at the microphone (approximately 15 cm from the participant's mouth) resulted in 72 dB SPL output in the earphones (Cornelisse, Gagné, & Seewald, 1991). For calibration, the intensity of the auditory feedback in the earphones was measured using a 2 cc coupler (Type 4946, Bruel & Kjaer Inc., Norcross, GA) connected to a sound level meter (Type 2250A Hand Held Analyzer with Type 4947 ½″ Pressure Field Microphone, Bruel & Kjaer Inc., Norcross, GA).

Continuous EEG was recorded in three conditions: *speaking*, *reading*, and *seeing*. Each condition consisted of 270 trials (3 blocks of 90 trials). In each block, binaural auditory stimuli were delivered through the insert earphones during one third of the trials (tone trials) whereas no auditory stimuli were presented in the remaining trials (no-tone trials). The order of the conditions was counterbalanced across participants in each group.

In the *speaking* condition (Fig. 1A), each trial started with the presentation of a word in white characters on a black background on the display. After 600 ms, the color of the word changed to green. This change of color constituted a *go* signal for the participant to say the word aloud. In the *reading* condition (Fig. 1B), the procedure was the same as in the speaking condition except that participants were instructed to read the word silently without any movements—thus, the motor component of the task was eliminated. In the *seeing* condition (Fig. 1C), the procedure was the same as in the *reading* condition except that nonlinguistic symbols ("++++") were shown rather than words—thus, both the cognitive-linguistic activity associated with reading and the motor activity were eliminated. For the tone trials in all three conditions, auditory stimuli were delivered 400 ms after presentation of the word/symbols in white color (Fig. 1D). Each trial ended 500 ms after the color of the word/symbols changed to green. The interstimulus-interval from the end of a trial to the beginning of the

[1] Note that, by investigating sensory systems during movement planning, this paradigm addresses neural processes that are distinct from those investigated with another recent paradigm in which stuttering and nonstuttering speakers have been compared in terms of auditory responses to their own speech during speech production (Beal et al., 2010, 2011; Liotti et al., 2010).

[2] The prominent LLAEP component that peaks ~70–130 ms after stimulus onset is typically labeled N100 or N1 in EEG recordings and M100 or N1m in magnetoencephalographic (MEG) recordings. This component is known to be generated by neuronal populations in the primary auditory cortex (Godey, Schwartz, De Graaf, Chauvel, & Liegeois-Chauvel, 2001; Näätänen & Picton, 1987; Zouridakis, Simos, & Papanicolaou, 1998). A similarly prominent component that peaks ~150–250 ms after stimulus onset is typically labeled P200 or P2 in EEG recordings and M200 or P2m in MEG recordings. The neural sources of the latter component have been shown to be located more anterior in auditory cortex (Papanicolaou et al., 1990; Ross & Tremblay, 2009).