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Anomaly in neural phase coherence accompanies reduced sensorimotor integration in adults who stutter

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ABSTRACT

Despite advances in our understanding of the human speech system, the neurophysiological basis of stuttering remains largely unknown. Here, it is hypothesized that the speech of adults who stutter (AWS) is susceptible to disruptions in sensorimotor integration caused by neural miscommunication within the speech motor system. Human speech unfolds over rapid timescales and relies on a distributed system of brain regions working in a parallel and synchronized manner, and a breakdown in neural communication between the putative brain regions could increase susceptibility to dysfluency. Using a speech motor adaptation paradigm under altered auditory feedback with simultaneous recording of EEG, the oscillatory cortical dynamics was investigated in stuttering and fluent adults (FA). Auditory feedback perturbation involved the shifting of the formant frequencies of the target vowel sound. Reduced adaptation in response to the feedback error was observed in AWS and was accompanied by differences in EEG spectral powers and anomalies in phase coherence evolving over the course of speech motor training. It is understood that phase coherence possibly captures neural communication within speech motor networks. Thus, the phase coherence network of the two groups exhibited differences involving the EEG frequency bands. These findings in anomalous neural synchrony provide novel evidence for compromised neuronal communication at short time scales within the speech motor network of AWS.

1. Introduction

Stuttering is a communication disorder in which fluency disruptions of syllables are both pathognomonic and symptomatic (Bloodstein and Ratner, 2008). Recent research has provided evidence that stuttering negatively impacts quality of life, personal relationships and socio-economic opportunities (Craig et al., 2009; Yaruss, 2010). Stuttering is widely considered to be a disorder of the central nervous system based on a wide range of structural and functional neuroimaging findings (Watkins et al., 2008; Chang et al., 2009; Choo et al., 2011; Chang and Zhu, 2013; Connally et al., 2014; Chang et al., 2015) rather than a disorder manifested in the peripheral musculature or neuromuscular junctions (Ingham et al., 2009). However, a complete and coherent understanding of its etiology is still lacking particularly in terms of the temporal dynamics of speech preparation and production in adults who stutter (AWS). A number of imaging studies have identified brain areas in which aberrant activity correlates with aspects of dysfluency in AWS (Fox et al., 1996; Jurgens, 2002; Neumann et al., 2003; Preibisch et al., 2003a, 2003b), but the neural dynamics of fluent speech and dysfluency have received far less attention. Findings from recent MEG or EEG studies have shown that silent and overt speech related activities elicit aberrant brain activity (Salmelin et al., 2000; Beal et al., 2010, 2011; Craig-McQuaide et al., 2014; Neef et al., 2015; Joos et al., 2014). The findings from these studies point to a core sensorimotor deficit in stuttering.

Persistent developmental stuttering is found, for example, to be associated with structural deficit in speech related brain areas of left sensorimotor cortex (Sommer et al., 2002). Brain network spanning auditory and motor areas develop differently in children who stutter (Chang and Zhu, 2013) and they exhibit reduced white matter tract in cortical premotor areas (Watkins et al., 2008). Such structural deficits may, thus, lead to impaired sensorimotor integration in AWS.

Human speech is a dynamic process involving distributed brain areas and neural mechanisms unfolding in parallel over rapid timescales with high degree of temporal precision (Chen et al., 2008; Guenther, 2006; Hickok and Poeppel, 2007; Tourville et al., 2008;

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Londei et al., 2010). Sensory, motor, associative and higher areas in the frontal cortex work in concert to invoke the appropriate speech motor routines to elicit an integrated sequence of movements of the articulators towards accomplishing desired speech goals. It is posited that motor preparation and production of fluent speech depends on uninterrupted and synchronized communication between these distributed brain regions, while a breakdown in the relevant neural pathways could lead to speech disruptions as observed in stuttering.

Recent EEG studies have suggested that timing in functional brain networks is accomplished by neural phase coherence, reflecting synchronous firing patterns across distributed brain network subserving a specific mode of behavior such as speech (Fries, 2005, 2015; Schroeder et al., 2008; Varela et al., 2001; Arnal et al., 2011; Giraud and Poeppel, 2012; Siegel et al., 2012). In addition, phase coherence between specific EEG frequency bands is found to be associated with formation of motor memory and neural communications (Fell and Axmacher, 2011). Therefore, neural phase coherence might subserve the formation of new sensorimotor map in speech training. Indeed, distinct coherence patterns have been shown to accompany speech motor training in healthy fluent adults (FA) (Sengupta and Nasir, 2015, 2016). It was found that theta-, beta-, and gamma-band activities during speech planning and production and theta-gamma phase coherence during speech planning contained significant and reliable information about motor speech adaptability. If stuttering is indeed caused by an impaired information flow due to miscommunication within speech motor network, differences in the phase coherence patterns of the distinctive EEG frequency bands – theta, alpha, beta and gamma - between stutterers and fluent adults should likewise emerge.

Anomalous coherence patterns are, presumably, more likely to be manifest under alterations in sensorimotor context for speech tasks, such as changes in auditory feedback, which can induce fluency or elicit atypical speech production in AWS (Kalinowski et al., 1993; Stuart et al., 1996). Recent studies also found that aberrant sensorimotor integration is closely linked with stuttering (Loucks and De Nil, 2012; Cai et al., 2012, 2014). Here, it is proposed that by assessing adaptation under altered auditory feedback the genesis of sensorimotor disintegration in AWS can be revealed (Houde and Jordan, 1998; Purcell and Munhall, 2006a, 2006b; Villacorta et al., 2007). This paradigm allows one to examine sensorimotor integration with the introduction of auditory perturbation and how the nervous system generates adaptive responses over the course of training. Maladaptive responses to the perturbation are predicted for AWS possibly result from anomalous neural communication in sensorimotor circuits that might be manifested by aberrant neural activities over the centro-parieital scalp areas. This is in line with observed differences in brain activity across different bands between AWS and FA even in the absence of any overt dysfluency (Salmelin et al., 2000). However, what is novel about the current study is that differences in phase coherence between various EEG frequency band-pairs at different stages of adaptive processes can highlight the nature of neural communication breakdown, which is thought be an important factor in stuttering. Any insight into short time scale neural communication in stuttering will elucidate aberrant cortical dynamics associated with the disorder.

2. Methods

2.1. Subjects

Study participants included 9 FA (2 F (females); 21. 3 ± 0.5 years; mean and SE) and 8 AWS (4 F; 26.3 ± 3.1 years;) with persistent stuttering. All subjects were native English speakers; the fluent adults had no known history of hearing or speech disorders. The AWS had undergone at least 3 years of speech therapy and stuttering severity was assessed according to Systematic Disfluency Analysis, a formal analysis tool used by speech-language pathologists to quantify behavioral stuttering and speech disfluency patterns. Frequency of stuttering events (% syllables stuttered, or %SS) ranged from 8.5% to 49% (mean 16%) and included one adult with severe stuttering, one with moderate and the rest with mild stuttering. The Northwestern University Research Ethics Board approved all experimental procedures and informed consent was obtained from all subjects prior to their participation in this study.

2.2. Experimental setup and task

All experiments were carried out in a soundproof booth. The task consisted of a speech motor adaptation protocol under continuous recording of EEG. The target word “Head” was displayed on a computer screen in front of the subjects and they were instructed to read it aloud when it appeared. Each subject repeated the target word for 8 baseline blocks followed by 12 training blocks. Each block consisted of 12 trials, totaling 360 trials per subject. AWS did not show any dysfluency during the production of the target word. The blocks were separated by a small hiatus of 30 s and trials were separated by 2.5 s intervals to prevent task induced fatigue. All subjects received normal auditory feedback in the baseline phase, while in the training phase auditory feedback was perturbed by shifting the first two formant frequencies of the target vowel /æ/ towards /I/ as in “Hid” (Fig. 1A). Estimates of formant shifts were obtained at the start of the experiment for each subject during a screening phase when the vowel space was mapped out. The intensity of the feedback signal played back to participants was adjusted to 80 dB to minimize air-borne unaltered auditory feedback. A masking noise of 60 dB was also delivered through the headphones to minimize any bone-conducted unaltered feedback.

We defined the baseline, early- and late-training phases as the last 50% of baseline trials, first 30% of training trials and the last 30% of the training trials respectively so that all phases contained a roughly equal number of trials. This partitioning allowed us to compare the neural activity of the two groups over the course of training as new sensorimotor maps presumably have been established in response to the perturbation (Sengupta and Nasir, 2015). We also selected a time window extending from 500 msec before to 500 msec after the onset of voice for our analysis. This partitioning within each utterance allowed us to study the neural activity relating to speech planning and motor preparation as well as during production and feedback processing.

2.3. Altered feedback

The formant frequencies of the vowels were altered in real time during speech production following the methods of the altered auditory feedback paradigm (Jones and Munhall, 2005; Purcell and Munhall, 2006a, 2006b) using LabView real time language implemented in the National Instruments PXI system. This approach allows estimation of formant frequencies using the Burg algorithm to update the LPC (linear predictive coding) filter coefficients of the speech signal at a rate of 10 kHz. The participant's voice was recorded at 10 kHz to obtain offline estimates of the formant frequencies.

2.4. Acoustical analyses and learning

The first and second formant frequencies of each utterance of the target sound for each subject were extracted using PRAAT (Boersma, 2001) and customized Matlab routines. The formant frequencies were normalized by subtracting their baseline means. Speech motor adaptation was assessed by focusing only on the produced first formant frequency (f1), since shifts in the first formants are typically much