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Roozbeh Behroozmand[1]*, Yilun Zhang[2], Yuan Wang[2], and Julius Fridriksson[1]

[1] Department of Communication Sciences and Disorders, University of South Carolina, Columbia, SC, United States
[2] Department of Epidemiology and Biostatistics, University of South Carolina, Columbia, SC, United States

***corresponding author, r-behroozmand@sc.edu**

Introduction

Aphasia is an acquired communication disability commonly resulting from post-stroke damage to the left-hemisphere brain networks. Depending on the size, location, and type of the stroke, individuals with aphasia exhibit a wide range of behavioral symptoms such as disorders in speech fluency, auditory comprehension, word-finding, and speech repetition. Recent investigations have provided evidence that such deficits in aphasia may result from damage to lower-level brain networks implicated in speech production and motor control mechanisms that are not directly influenced by language-related neural processes [1-3]. In the present study, we investigated the neural oscillatory correlates of speech impairment in individuals with post-stroke aphasia.

Methods

A total of 34 subjects with post-stroke aphasia (22 males; age range: 42-80 yrs; mean age: 61.2 yrs), and 46 neurologically intact control subjects (23 males; age range: 44-82 yrs; mean age: 63.6 yrs) completed a speech vowel production task under altered auditory feedback (AAF) while EEG signals were simultaneously recorded from 64 scalp electrodes following a standard 10-10 montage. All subjects with aphasia were tested at least 6 months post stroke and had undergone testing with the Western Aphasia Battery (WAB) [4]. Based on the WAB aphasia classification system, the distribution of aphasia types across the 34 subjects was as follows: Anomic = 7; Broca's = 18; Conduction = 8; and Global = 1. Subjects in the control group had no history of speech, language, or neurological disorders. Subjects in both groups passed a binaural hearing screening and had thresholds of 40 dB or less at 500, 1000, 2000, and 4000 Hz. For this study, EEG data were analyzed to measure modulation of beta band power of neural oscillatory activities within 13-25 Hz frequency ranges before and after the onset of speech vowel production under normal auditory feedback (NAF).

Results

Results of the preliminary analysis indicated deficits in the neural oscillatory mechanisms during the planning phase of speech production in individuals with post-stroke aphasia compared with controls. This effect was indexed by the reduced magnitude of beta band de-synchronization (i.e. power reduction) before the onset of speech as well as an earlier onset of power reduction in aphasia vs. controls.

Conclusions

Beta band de-synchronization has been suggested to play a key role in regulating motor planning and production neural processes in a wide range of behaviorally relevant tasks. This effect is proposed to arise from the interplay between thalamo-cortical networks that selectively activate task-relevant motor areas by priming prefrontal cortical neurons via reducing their excitability threshold before the onset of movement [5]. Findings from the present study suggest that individuals with aphasia exhibit deficits in engaging such neural processes to activate cortical motor neurons for speech production as indexed by their pathologically altered patterns of neural oscillatory mechanisms driving beta band de-synchronization. Our preliminary analysis show that individuals with aphasia have deficits in regulating both the timing and the overall power of beta band de-synchronization before the onset of vowel sound production, suggesting deficit in the underlying neural mechanisms during the planning phase of speech.

References

[1] Basilakos, A., Rorden, C., Bonilha, L., Moser, D., & Fridriksson, J. (2015). Patterns of Poststroke Brain Damage That Predict Speech Production Errors in Apraxia of Speech and Aphasia Dissociate. *Stroke*, *46*(6), 1561–1566. https://doi.org/10.1161/STROKEAHA.115.009211

[2] Behroozmand, Roozbeh, Phillip, L., Johari, K., Bonilha, L., Rorden, C., Hickok, G., & Fridriksson, J. (2018). Sensorimotor impairment of speech auditory feedback processing in aphasia. *NeuroImage*, *165*(July 2017), 102–111. https://doi.org/10.1016/j.neuroimage.2017.10.014

[3] Johnson, L. P., Sangtian, S., Johari, K., Behroozmand, R., & Fridriksson, J. (2020). Slowed Compensation Responses to Altered Auditory Feedback in Post-Stroke Aphasia: Implications for Speech Sensorimotor Integration. *Journal of Communication Disorders*, *88*(July), 106034. https://doi.org/10.1016/j.jcomdis.2020.106034

[4] Kertesz, A. (2007). *The Western Aphasia Battery - Revised.* New York: Grune & Stratton.

[5] Pfurtscheller, G., & Lopes Da Silva, F. H. (1999). Event-related EEG/MEG synchronization and desynchronization: Basic principles. *Clinical Neurophysiology*, *110*(11), 1842–1857. https://doi.org/10.1016/S1388-2457(99)00141-8

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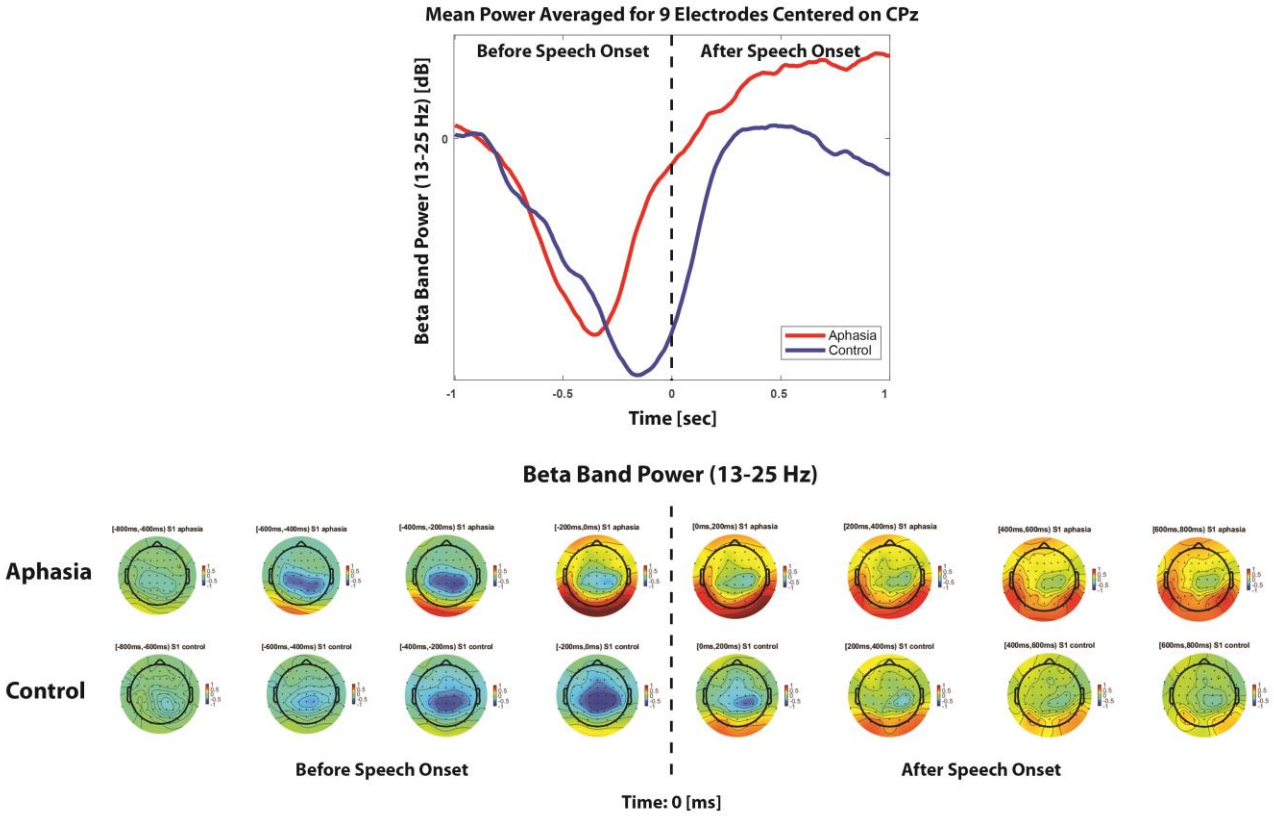

Figure 1. Top panel: the overlaid profile of mean beta band power (13-25 Hz) for aphasia (n=34) and control (n=46) groups before and after the onset of speech averaged across 9 EEG channels over the centro-parietal area centered on the CPz electrode. Bottom panel: the top view of scalp showing the topographical distribution maps of the mean beta band power for 64 EEG electrodes before and after the onset of speech averaged across aphasia and control groups.