Association Between the Beta Band Neural Response and the Behavioral Performance in Aphasic and Neurologically Intact Individuals

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Background

The complex motor act of speech requires sensorimotor integration. The integration of online auditory feedback provides one source of sensory input for fluent production of speech. In addition, auditory feedback is critical for detecting production errors and speech motor control [1]. Although the influence of auditory feedback on speech has long been demonstrated by previous studies, more recent evidence has also supported the notion that the motor system is involved in speech perception [2]. These findings emphasize the importance of studying the underlying mechanisms of sensorimotor integration in the auditory and speech motor systems.

The role of auditory feedback in speech control is supported by data showing that human subjects produce compensatory motor responses to pitch-shift alterations (i.e. error) in their vocalization auditory feedback [3-5]. The contemporary models of speech suggest that an internal model generates forward predictions about sensory input based on a copy of motor commands ("efference copies") and compares them with the incoming auditory feedback for error detection and motor control [6]. These processes are proposed to be carried out by predominantly left-lateralized brain networks implicated in speech sensorimotor integration [7].

Previous research has shown that individuals with left-hemisphere stroke leading to speech and language impairment due to aphasia generate diminished speech compensation responses to pitch-shift alterations in their vocalization auditory feedback [8]. In the current study, we used left-hemisphere stroke as a model to study the underlying neural mechanisms of speech sensorimotor integration in participants with the clinical diagnosis of post-stroke aphasia.

Study Goal

The present study aims to examine the association between brain neural activity and the ability for speech auditory feedback error correction in both post-stroke aphasia and neurologically intact individuals.

Methods

This study included data from 34 individuals with aphasia (age: 61+/-11.2 years) and 46 neurologically intact subjects (age: 64+/-7.9 years). As shown in Figure 1, participants were asked to produce a vowel sound /a/ under randomized up and down pitch-shifted (+/- 100 cents) altered auditory feedback (AAF). To probe the measure of brain activity, electro-encephalography (EEG) signals were simultaneously recorded from 64 scalp electrodes following a standard 10-10 montage during the vocalization task under pitch-shifted AAF conditions. A multivariate linear regression model was fitted to examine the association between the EEG beta band power (13-25 Hz) and the vocal compensation responses within a time window from 0 to 500 ms following the onset of pitch-shift AAF stimuli.

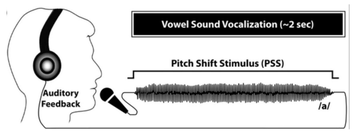

Figure 1. The experimental paradigm for altered auditory feedback (AAF).

Results

According to the t-statistics of 2.4778 (p = 0.0138), we have enough evidence to support that the magnitude of vocal compensation is significantly diminished in aphasia vs. the control group under the upward pitch-shift condition (**Figure 2A**). However, with the t-statistics of 0.2430 (p = 0.8082), we do not have enough evidence to support a significant difference in vocal compensation magnitude between two groups under the downward pitch-shift AAF condition (**Figure 2B**).

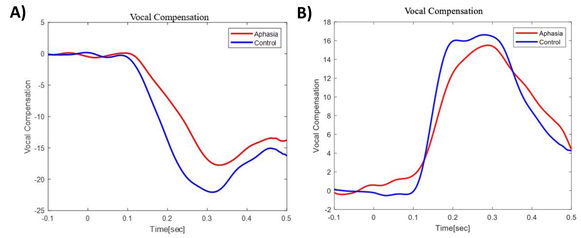

Figure 2. The overlaid speech compensation responses in aphasia and control groups in response to **A)** upward, and **B)** downward pitch-shift stimuli in -100 to 500 ms time window.

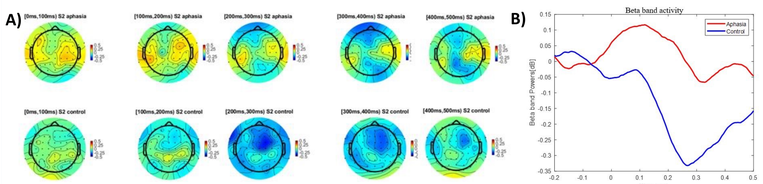

Figure 3. A) The head plots display the topographical distribution maps for grand-average beta band power for the upward pitch-shifts in aphasia (top) and control groups (bottom). We observed that beta band power was more strongly de-synchronized (i.e. attenuated) in the control vs. aphasia group in electrodes over the fronto-central region. The beta band desynchronization reached the peak at the time window from 200 to 300 ms. **B)** The overlaid beta band power contours for aphasia and control within -200 to 500 ms relative to pitch-shift stimulus onset.

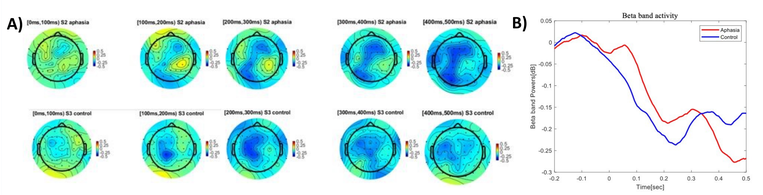

Figure 4. A) The head plots display the topographical distribution maps for grand-average beta band power for the downward pitch-shift in aphasia (top) and control groups (bottom). We observed that beta band power was more strongly de-synchronized (i.e. attenuated) in the control vs. aphasia group in electrodes over the centro-parietal region. The beta band desynchronization reached the peak at the time window from 200 to 300 ms. **B)** The overlaid beta band power contours for aphasia and control within -200 to 500 ms relative to pitch-shift stimulus onset.

Results (continued)

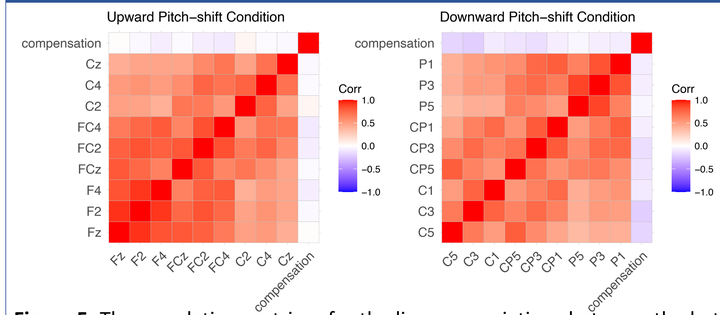

Figure 5. The correlation matrices for the linear associations between the beta band power at different channels and speech compensation. The Wilks' lambda statistics for the MANCOVA test is 0.952. According to the F-value at 2.26 (p = 0.0142), we have sufficient evidence to support a significant linear association between beta band powers and speech compensation under downward pitch-shift condition; however, no such effect is indicated for responses to upward condition.

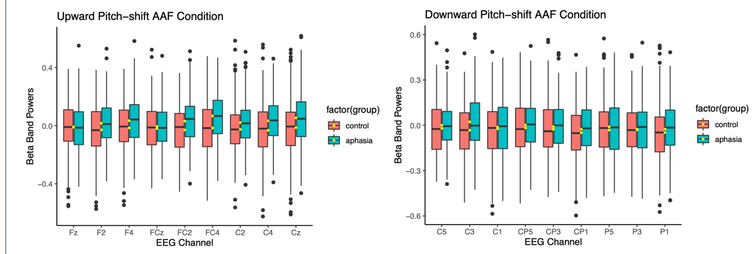

Figure 6. The yellow dots show the mean beta band power for each group. The Wilks' lambda statistics of the MANCOVA test are 0.866 and 0.916 for the group variable under upward pitch-shift and downward pitch-shift conditions, respectively. The according F-values at 5.66 (p < 0.0001) and 3.71 (p = 0.0002), we have enough evidence to reject the null hypothesis that beta band power is not different for the two groups. This evidence suggests that the aphasia group exhibited significantly diminished de-synchronization of the beta band power compared with controls under both upward and downward AAF conditions.

Conclusions

The findings of the current study reveal that diminished neural de-synchronization of the beta band activities is related to the poorer performance on speech auditory feedback error correction under the downward pitch-shift AAF condition. Such relation is not found under the upward pitch-shift AAF condition. The reason might be that the participants failed to reach the desired low F0 trajectory under the upward pitch-shift stimulus. Furthermore, the beta band de-synchronization is diminished for the aphasia group compared to the healthy control group, suggesting that aphasic individuals have deficits in the underlying neural mechanisms for the sensorimotor integration. Our study highlights the abnormal pattern of beta band neural oscillation modulation in individuals with post-stroke aphasia. This finding may help develop methodologies for speech treatment related to normalizing brain activity such as brain stimulation and neuro-feedback training.

References:

[1] Guenther F. H. (2006). Cortical interactions underlying the production of speech sounds. *Journal of communication disorders*, 39(5), 350–365.

[2] Watkins, K. E., Strafella, A. P., & Paus, T. (2003). Seeing and hearing speech excites the motor system involved in speech production. *Neuropsychologia*, 41(8), 989–994.

[3] Elman J. L. (1981). Effects of frequency-shifted feedback on the pitch of vocal productions. *The Journal of the Acoustical Society of America*, 70(1):45-50.

[4] Larson C. R. (1998). Cross-modality influences in speech motor control: the use of pitch shifting for the study of F0 control. *Journal of communication disorders*, 31(6), 489-502.

[5] Burnett T. A, Freedland M. B., Larson C. R., Hain T. C. (1998). Voice F0 responses to manipulations in pitch feedback. *The Journal of the Acoustical Society of America*, 103(6):3153-61.

[6] Hickok, G. (2012) . Computational neuroanatomy of speech production. *Nature Reviews Neuroscience*, 13(2) : 135–145.

[7] Hickok, G. and Poeppel , D. (2007) . The cortical organization of speech processing. *Nature Reviews Neuroscience*, 8(5) :393–402.

[8] Behroozmand, R. , Phillip, L. , Johari , K. , Bonilha, L. , Rorden, C. , Hickok, G. , and Fridriksson, J. (2018) . Sensorimotor impairment of speech auditory feedback processing in aphasia. *NeuroImage*, 165: 102–111.

Acknowledgement

This research was supported by funding from NIH/NIDCD Grants R01-DC018523 and K01-DC015831 (PI: Behroozmand) and R21-DC014170 and P50-DC014664 (PI: Fridriksson). The authors wish to thank Lorelei Phillip Johnson and Stacey Sangtian for their assistance with data collection.