

### Neurobasis of Impaired Voice Motor Control in Parkinson's Disease

Erin Hurst<sup>1</sup>, Meg Johnson<sup>1</sup>, Sarah Neal<sup>1</sup>, Karim Johari<sup>1</sup>, Priyantha Herath<sup>2</sup>, Roozbeh Behroozmand<sup>1</sup>

<sup>1</sup>Department of Communication Sciences and Disorders, University of South Carolina, Columbia, SC <sup>2</sup>Department of Neurology, University of South Carolina, Columbia, SC



# Background

- In efforts to further explore the voice mechanism, Guenther and colleagues developed the  $\bullet$ DIVA model<sup>1</sup> to outline the neural correlates associated with speech. The model includes two subsystems that control voicing: feedforward and feedback control system.
- Evidence in the speech neuroscience lab has proven that patients with Parkinson's disease (PD) have sensorimotor deficits in speech processing that specifically affect the feedforward and feedback control subsystems. Therefore, these patients suffering from

# Results

- Our analysis indicated that Parkinson's patients and control group controlled their voice in response to pitch-shifted feedback alterations by generating compensatory vocal responses. • PD patients generated compensatory vocal responses that were significantly larger that those generated by the control group. This overshooting pattern of vocal motor compensation was observed in response to both upward and downward pitch-shift stimuli and were consistent with findings of previous studies<sup>2-3</sup> (Figure 3a).
- Consistent with data from one previous study in PD<sup>4</sup>, the overshoot in vocal responses in PD

PD have impaired ability to generate and control motor actions. Previous studies have detected auditory-vocal integration neural abnormalities in voice motor control of these individuals. However, it is unclear as to how these patients compensate for this abnormal presentation.



were accompanied with a significant reduction in the amplitude of the P1-N1-P2 ERP components compared with the control group (Figure 3b). • Control subjects generated stronger neural activities as indexed by fronto-central ERP components shown in the topographical distribution maps (Figure 3c).



Figure 3. a) Vocal compensation responses to upward and downward pith shift in PD (red) and control (black) groups. b) Temporal profile of ERP responses to pitch-shift stimuli overlaid for PD (red) and control (black) groups. c) Topographical distribution maps of the P1, N1, and P2 ERP components.

### Methods

**<u>Purpose</u>**: The purpose of this study was to determine if Parkinson's patients have reduced voice motor control and if they do, how do they compensate for this reduced control?

**<u>Participants</u>**: We recruited 16 patients with PD and 16 age-matched control subjects with no history of neurological disease and speech, hearing and cognitive impairment. All participants were right-handed, speaker of English, and over the age of 50.

Participants were tested for measuring their brain neural activity using EEG while they performed a voice motor control task under altered auditory feedback.

### <u>Altered Auditory Feedback (AAF) Task:</u>

•Participants were visually cued to produce and maintain a steady vocalization of the vowel sound /a/ for approximately 2-3 seconds (Figure 2).

• During each vocalization trial, a brief (200 ms) pitch shift stimulus altered the voice auditory feedback randomly in upward and downward directions with a magnitude of  $\pm 100$  cents. •A total number of 200 trials (100 shifted up, 100 shifted down) were recorded for the analysis of behavioral and neural responses.

#### **Data Analysis:**

## Discussion

- Our findings revealed impairment of sensorimotor mechanisms for voice motor control in PD. • We found that PD exhibited an overshooting pattern of behavioral vocal compensation in
- response to alterations in the auditory feedback. This effect indicated that the feed-forward motor mechanisms of voice are impaired in PD.

• As a results, PD patients showed a greater degree of over-sensitivity to feedback alterations, which was reflected in their de-stabilized vocal motor reactions in response to pitch-shift stimuli.

• In addition, we argue that the sensorimotor neural mechanisms of voice motor control are impaired in PD. This notion is supported by our findings demonstrating that the amplitude of the P1-N1-P2 ERP components were significantly reduced in PD compared with the control group.

• The pattern of topographical distribution maps revealed the contribution of frontal cortical mechanisms for voice motor control. Our data suggest that these mechanisms are normally functional in control group, whereas they are impaired in patients with PD.

• Overall, our findings provided new insights into the behavioral and neural correlates of sensorimotor impairment for voice motor control in PD.

• Event-related potentials (ERPs) and measures of vocal compensation were extracted in response to upward and downward pitch-shift stimuli in the auditory feedback.



Figure 2. Experimental paradigm for the altered auditory feedback task. Participants were cued to produce a steady vocalization of the vowel sound /a/ while they received pitchshift alterations in their voice auditory feedback.

• We propose that the behavioral measure of vocal compensation and ERP responses to altered auditory feedback can be used as powerful objective biomarkers of voice motor disorder in PD. • These biomarkers can be developed into a clinical assessment battery for diagnosis and treatment

of voice motor disorders in PD.

## References

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