Impaired Efference Copy Mechanisms of Speech in Post-Stroke Aphasia and the Role of Dorsal Stream Network

Roozbeh Behroozmand[1], Leonardo Bonilha[2], Chris Rorden[3], Gregory Hickok[4], Julius Fridriksson[5]

[1]Speech Neuroscience Lab, Department of Communication Sciences and Disorders, University of South Carolina, 915 Greene Street, Columbia, SC 29208, USA

[2]Department of Neurology, Medical University of South Carolina, Charleston, SC 29425, USA

[3]Department of Psychology, University of South Carolina, Columbia, SC 29208, USA

[4]Department of Cognitive Sciences, University of California, Irvine, Irvine CA 92697, USA

[5]The Aphasia Lab, Department of Communication Sciences and Disorders, University of South Carolina, 915 Greene St., Columbia, SC 29208, USA

Abstract

The efference copies of motor commands have been proposed to play a key role in generating internal predictions about expected sensory feedback during speech production[1,2]. These predictive signals serve important functional mechanisms such as distinguishing own speech from those of external speakers, online monitoring, and detection and correction of feedback errors in self-produced speech sounds. In the auditory system, this effect was shown to result in suppression of sensory neural responses to self-produced speech sounds that are predicted by the efference copies during active production in comparison with passive listening to the playback of the identical self-produced speech[3,4]. In addition, the representation of efference copies in the auditory system has been associated with speaking-induced enhancement of neural responses to unexpected alterations (errors) in the auditory feedback[5]. In the present study, we used speakers with post-stroke aphasia as a model to investigate how damage to neural structures of the dorsal stream network in the left-hemisphere may impair efference copy mechanisms during speech. The altered auditory feedback (AAF) experimental paradigm was utilized to measure neurophysiological brain responses to online pitch shifts in speech auditory feedback while 34 speakers with aphasia and 46 matched control subjects produced a steady speech vowel sound /a/ at their conversational pitch and loudness and listened to the playback of their self-produced speech under AAF. Results of our analysis on event-related potentials (ERPs) indicated a significant suppression of the N1 ($F=17.8$, $p=0.001$) and P2 ($F=16.4$, $p=0.001$) neural responses during speaking vs. listening in control subjects; however, this suppression effect was reduced for the N1 ($F=8.35$, $p<0.05$) and was eliminated for the P2 component ($p>0.05$). Lesion-mapping analysis of ERP suppression modulation in aphasic relative to control subjects revealed that damage to multimodal dorsal

stream networks within the left frontal and temporal cortical areas predicted impaired efference copy mechanisms in speakers with post-stroke aphasia. We found that such impairments were predicted by damage to Inferior Frontal Gyrus (IFG) Pars Orbitalis, Anterior Cingulate Cortex (ACC), and Supplementary Motor Area (SMA) for the N1, and Precentral Gyrus and Superior Temporal Gyrus (STG) for the P2 component. These findings suggest that the impairment of efference copy mechanisms is represented by damage to widely distributed speech sensorimotor regions within the dorsal stream network in the left hemisphere. At earlier stages of speech auditory feedback processing, such impairments are predominantly predicted by damage to motor networks within the prefrontal cortical areas whereas the later stages of such impairments involve damage to regions within auditory-motor networks in the temporal and frontal lobe cortical areas. These findings demonstrate the contribution of complex multimodal neural substrates and the dynamic temporal aspects of speech sensorimotor processing and its impairment in speakers with post-stroke aphasia.

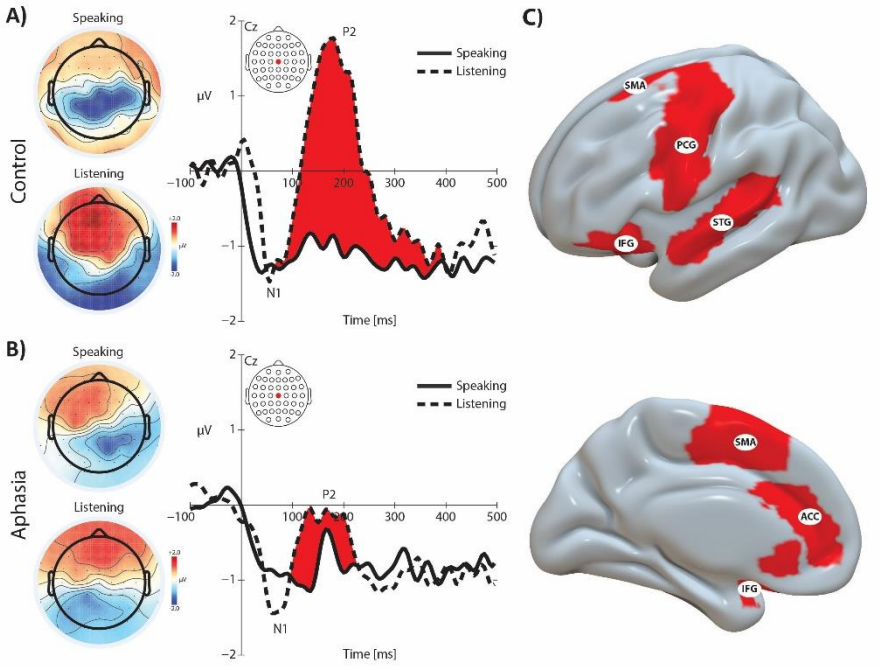

Figure1. Topographical distribution maps and the temporal profiles of ERP activity overlaid for speaking and listening conditions for the P2 ERP component in **A)** control (n=46) and **B)** aphasic speakers (n=34). **C)** Results of lesion-mapping analysis of ERP activity in aphasia: reduced and eliminated suppression of the N1 and P2 ERP components is predicted by damage to a distributed network including the cortical motor areas (IFG, ACC, SMA) for the N1 and auditory-motor cortical areas (STG and PCG) for the P2 component.

References

[1] Hickok G, Houde JF, RongF. Sensorimotor integration in speech processing: computational basis and neural organization. *Neuron* (2011)

[2] Houde JF, Chang EF. The cortical computations underlying feedback control in vocal production. *Current Opinion in Neurobiology* (2015)

[3] Houde JF, Nagarajan SS, Sekihara K, Merzenich MM. Modulation of the auditory cortex during speech: An MEG study. *Journal of Cognitive Neuroscience* (2002)

[4] Behroozmand R, Larson CR. Error-dependent modulation of speech-induced auditory suppression for pitch-shifted voice feedback. *BMC Neuroscience* (2011)

[5] Behroozmand R, Phillip L, Johari K, Bonilha L, Rorden C, Hickok G, Fridriksson J. Sensorimotor impairment of speech auditory feedback processing in aphasia. *Neuroimage* (2018)