Sensorimotor Impairment of Speech Production and Motor Control in Post-Stroke Aphasia: Evidence from Behavioral and Neurophysiological Biomarkers

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

[1] Speech Neuroscience Lab, Department of Communication Sciences and Disorders, University of South Carolina, 915 Greene Street, Columbia, SC 29208, USA [2] The Aphasia Lab, Department of Communication Sciences and Disorders, University of South Carolina, 915 Greene St., Columbia, SC 29208, USA
[3] Department of Neurology, Medical University of South Carolina, Charleston, SC 29425, USA [4] Department of Psychology, University of South Carolina, Columbia, SC 29208, USA [5] Department of Cognitive Sciences, University of California, Irvine, Irvine CA 92697, USA

Introduction

Background:

Aphasia is an acquired speech-language disorder commonly resulting from post-stroke damage to the left-hemisphere. Depending on factors such as the size, location, and type of the stroke, individuals with aphasia exhibit a wide range of behavioral variability including, but not limited to, impairments in speech fluency, auditory comprehension, word-finding, and speech repetition that impact everyday communication ability.

Evidence from several studies has suggested the notion that certain aspects of behavioral impairment in aphasia may be accounted for by damage to the sensorimotor network that supports auditory feedback processing during speech[1-5].

Objectives:

The present study was a systematic investigation toward understanding the impairment of sensorimotor integration mechanisms that underlie speech auditory feedback processing in patients with post-stroke aphasia. Our goal was investigate behavioral, neurophysiological, and lesion correlates of impaired sensorimotor integration of speech in aphasia:

1 – How speech auditory feedback processing is impaired in aphasia?
2 – What are the lesion predictors of impaired speech sensorimotor integration in aphasia?
3- How neurophysiological measures represent speech impairment?

Speech sensorimotor integration:

The principles of integrative models of speech are centered around the idea of an internal forward model that estimates the dynamical states of speech articulators based on learned and internally maintained associations between motor commands and their actual sensory (e.g., auditory) feedback[6-9].

According to these models, speech control is not directly mediated by incoming sensory feedback, but rather via internal representations of predicted sensory consequences of motor commands even before sensory feedback has become available(**Fig. 1**)

During overt production, sensory feedback can be used to correct for speech feedback errors and update the internal forward model.

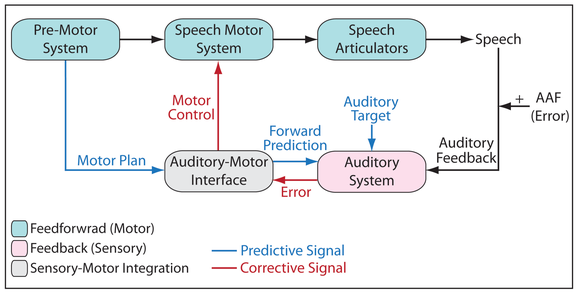

Figure 1. The auditory-motor integration model of speech. In this model, the auditory-motor interface transforms speech motor plans into forward prediction of auditory feedback. The auditory system compares forward predictions with actual speech feedback to detect prediction errors in response to altered auditory feedback (AAF). The auditory system also detects sensory prediction errors in response to AAF by comparing the intended auditory target with actual feedback from speech. The generated sensorimotor errors are translated into corrective signals by the auditory-motor interface to adjust the speech motor parameters in response to AAF.

Materials and methods

Experimental task:

Sixteen patients with post-stroke aphasia (6 Broca's, 5 anomic, 5 conduction) and 16 neurologically intact control individuals completed a speech vowel production task under altered auditory feedback (AAF) condition. During vowel production, a randomized (up or down) pitch-shift stimulus perturbed speech auditory feedback at 100 cents (**Fig. 2**).

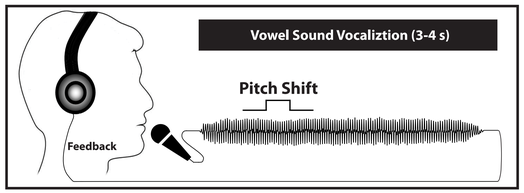

Figure 2. Auditory feedback perturbation paradigm.

Speech compensation:

For each aphasic patient, speech compensation responses to AAF were calculated based on the log-transformed ratio of speech compensation magnitude normalized to the mean of the control group response for both upward and downward pitch-shift stimuli:

$$Speech\ Compensation\ Ratio = 10 \times \log_{10}(X_{Aphasia}/\overline{X}_{Control})$$

Lesion maps:

Neuroimaging data in stroke survivors were used to determine lesion predictors of impaired sensorimotor function associated with diminished compensatory responses to errors in speech auditory feedback (**Fig. 3**).

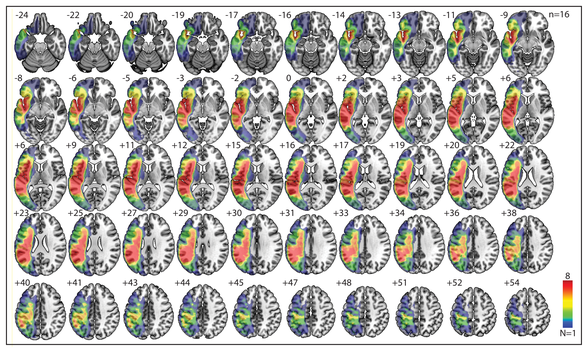

Figure 3. Lesion overlap maps in aphasic speakers (n=16). The maps show lesion distribution on coronal slices in MNI space for the sample, with warmer colors representing more lesion overlap across aphasic speakers (dark red areas represent lesion overlap across at least N=8 individuals).

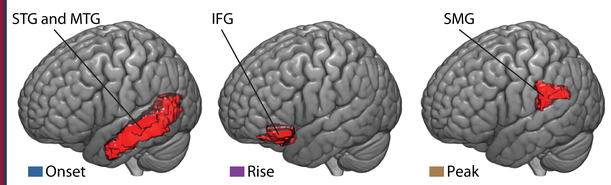

Figure 4. Lesion predictors of diminished speech compensation responses to AAF in aphasia.

Results

Behavioral responses:

Results of the analysis revealed a significant main effect of group ($F(1,30) = 16.02$, $p < 0.001$), indicating that the magnitude of speech compensation responses to AAF was diminished in aphasia vs. control (**Fig. 5**).

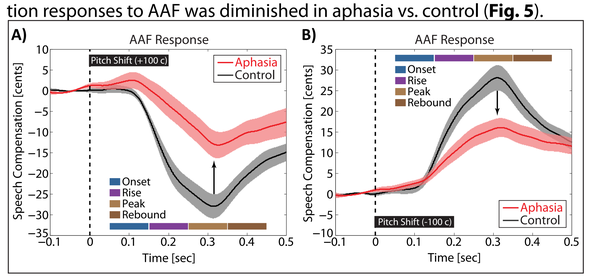

Figure 5. The overlaid profiles of speech compensation responses to altered auditory feedback (AAF) in 16 speakers with aphsia and 16 neurologically intact control individuals.

ERP Responses:

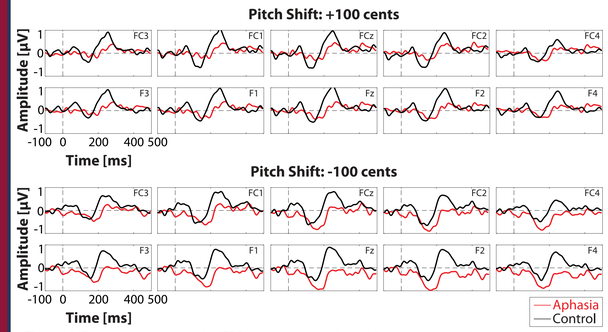

Figure 6. Event-related potential (ERP) responses to pitch shifts across aphasia and control groups.

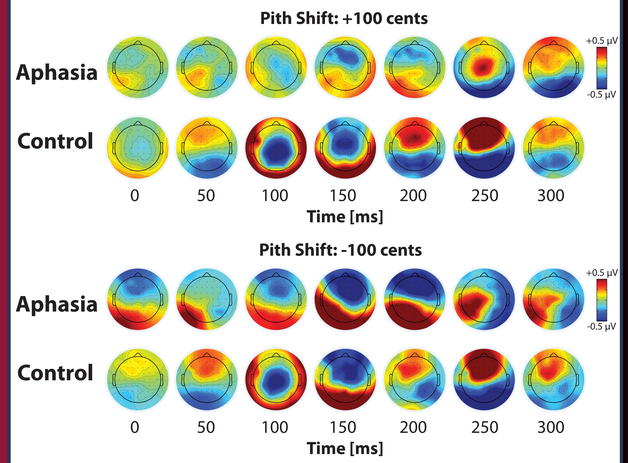

Figure 7. Topographical distribution maps of ERP responses to pitch-shift stimuli.

Discussion

Our approach combined behavioral, neurophysiological, and lesion correlates to charactrize impaired sensorimotor integration of speech in post-stroke aphasia.

We propose that our findings support the following notions:

The measure of compensation responses to altered auditory feedback (AAF) provided an objective biomarker to probe the integrity of speech sensorimotor mechanism and identify its impaiment in patients with post-stroke aphasia.

The temporal-specific pattern of diminished compensatory responses to speech errors in auditory feedback in aphasia is influenced by damage to distinct neural networks within sensory, motor, and sensorimotor integration networks.

These findings emphasize the role of cortical auditory areas in speech monitoring and sensory detection of feedback errors in the early phase of speech motor control. We argue that the IFG and SMG subserve functions associated with motor predictions and sensorimotor integration for detection and correction of speech errors in auditory feedback.

We argue that ERP measures provide a neurphysiological biomaker to identify deficits in neural processing of speech auditory feedback and its impairment in post-stroke aphasia. Our findings support the notion that modulation of specific ERP components (e.g., N1, P2) highlight sensory, motor, and sensorimotor aspects of speech impairment in aphasia.

References

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