Brain lesion associated with impaired sensorimotor processing of speech auditory feedback in aphasia

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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 to use behavioral measures of speech production combined with lesion anatomy to address the following questions:

1 – How sensorimotor mechanisms of speech auditory feedback processing is impaired in aphasia?
2 – What are the lesion predictors of impaired sensorimotor integration for speech production and motor control?

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.

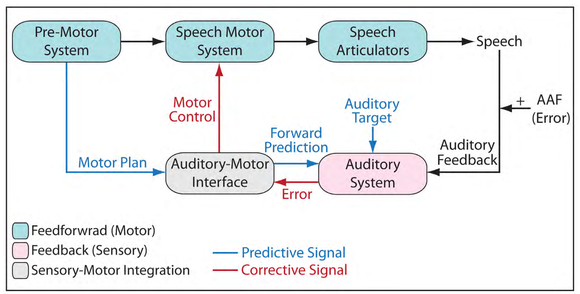


Figure 1. The auditory-motor integration model of speech. In this model, the auditory-motor interface transforms speech motor plans into forward prediction of auditory feedback. The auditory system compares forward predictions with actual speech feedback to detect prediction errors in response to altered 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**).

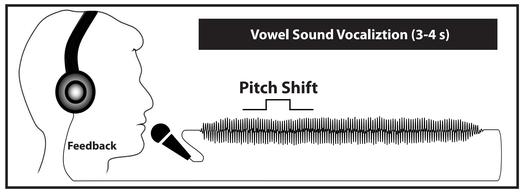


Figure 2. Auditory feedback perturbation paradigm.

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:

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

Regions of interest:

The analysis related z-score-transformed mean image intensities (corrected for multiple comparisons) in 12 regions of interest (**Table 1**) in the left hemisphere to the behavioral measures of speech compensation to AAF.

The regions of interest were selected based on a review of the relevant literature, to encompass areas that have been historically associated with dorsal stream network for speech processing and sensorimotor integration[10-12].

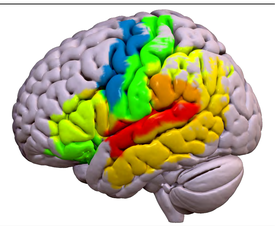
Lesion-symptom-mapping:

A univariate lesion-symptom-mapping (LSM) analysis was completed to identify localized brain damage within the selected ROIs that was associated with speech sensorimotor impairments, as indexed by diminished speech compensation responses to AAF. Only ROIs where at least five participants had damage were included in our analysis.

The LSM analysis procedures were implemented in the NiiStat toolbox for MATLAB (www.nitrc.org/projects/niistat) in which the cortical regions comprised a neuroanatomical model where the mean intensity value in each ROI was entered into a regression analysis to predict diminished speech compensation responses to AAF.

Table 1. Left-hemisphere regions of interest used in lesion-symptom-mapping analysis

Inferior frontal gyrus (pars opercularis)
Inferior frontal gyrus (pars orbitalis)
Inferior frontal gyrus (pars triangularis)
Precentral gyrus
Postcentral gyrus
Rolandic operculum
Supramarginal gyrus
Angular gyrus
Superior temporal gyrus
Middle temporal gyrus
Heschl's gyrus
Inferior parietal gyrus



Results

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**).

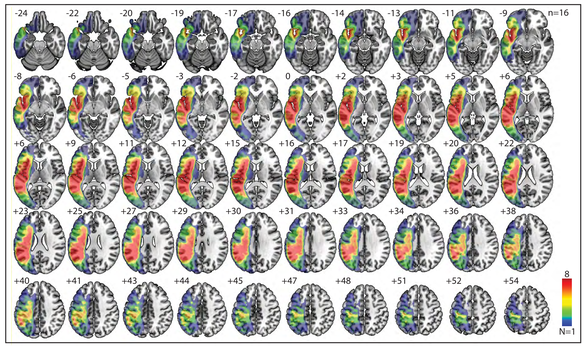


Figure 3. Lesion overlap maps in aphasic speakers (n=16). The maps show lesion distribution on coronal slices in MNI space for the sample, with warmer colors representing more lesion overlap across aphasic speakers (dark red areas represent lesion overlap across at least N=8 individuals).

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. 4**).

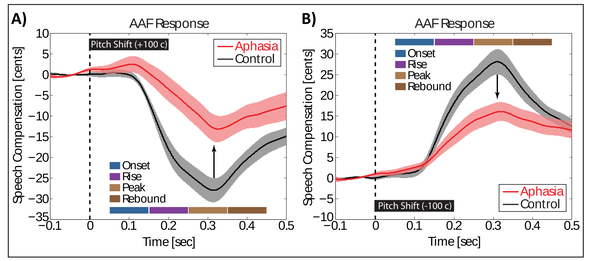


Figure 4. The overlaid profiles of speech compensation responses to altered auditory feedback (AAF) in 16 speakers with aphsia and 16 neurologically intact control individuals.

Lesion predictors of impaired speech:

Results revealed that diminished responses at the onset, rise, and peak of speech compensation to AAF in aphasia were predicted by damage to the superior and middle temporal gyrus, inferior frontal gyrus, and supramarginal gyrus areas, respectively ($z < -2.57$, $p_{corr} < 0.05$) (**Fig. 5**).

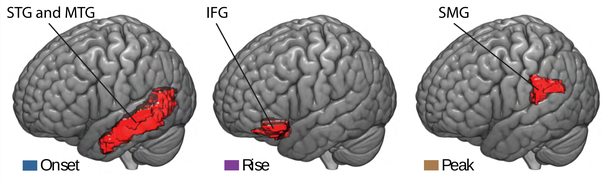


Figure 5. Lesion predictors of diminished speech compensation responses to AAF in aphasia.

Discussion

Our novel approach delineated neural structures in multimodal sensory, motor, and sensorimotor integration networks that are crucial for neural processing of speech auditory feedback.

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 conclude that damage to sensory, motor, and sensorimotor integration networks can disrupt speech error processing in aphasia due to impaired sensory detection of feedback errors, inaccurate motor predictions, or because detected errors are not translated into corrective commands due to damage to the sensorimotor integration interface.

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