Introduction

Background:
Aphasias are acquired speech-language disorders 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 studies of aphasia 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.

Objectives:
The present study was a systematic investigation toward understanding the impairment of sensorimotor mechanisms that underlie speech auditory feedback processing in patients with post-stroke aphasia. Our goal was to use behavioral measures of speech production and motor control to identify localized brain damage within the sensorimotor network that supports auditory feedback processing.

Methods:

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 consequences (e.g., auditory feedback). 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.

Experimental task:
Sixteen patients with post-stroke aphasia (6 Broca's, 5 anomic, 5 conductive) and 16 neurologically intact controls completed a 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).

Results:

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 responses for both upward and downward pitch-shift stimuli.

Lesion predictors of diminished speech compensation responses to AAF in aphasia were predicted by damage to the sensorimotor network that supports auditory feedback. These findings emphasize the role of sensorimotor areas in impaired speech production in aphasia due to impaired speech error detection and correction of speech errors in auditory feedback.

Discussion:

Our novel approach delineates neural structures in multimodal sensory, motor and sensorimotor integration networks that are crucial for neural processing of speech auditory feedback. These findings imply that the IFG and SMG subregions associated with motor predictions and sensorimotor integration for detection and correction of speech errors in auditory feedback.

Conclusion:
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 damage to speech motor control is not translated into corrective commands due to damage to the sensorimotor integration interface.

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Acknowledgement:
This research was supported by funding from NIH/NIDCD Grants R21-DC015871-01 (P. Behroozmand) and R21-DC014170 and P20-DC014664 (Pi Fridriksson).