# Effect of Altered Auditory Feedback on Sensorimotor Deficits in Aphasic Individuals

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## BACKGROUND

Post stroke aphasia is associated with decline in sensorimotor integration during speech production. However, the underlying neural mechanisms of this impairments is not fully understood.

The auditory-motor integration model of speech consists of speech error detection and correction (Figure 1). Under altered auditory feedback (AAF), using a pitch-shift stimulus, the researchers were able to detect sensorimotor impairments by measuring the individual's compensatory responses to external error signals.

For individuals with aphasia, research has shown that certain areas of the brain impact sensorimotor interactions for speech error processing. However, there is limited evidence regarding the relationship between underlying lesion characteristics and sensorimotor impairments for a variety of aphasias.

The current study investigated the neural and behavioral correlates of impaired sensorimotor processing for speech production in aphasic patients.

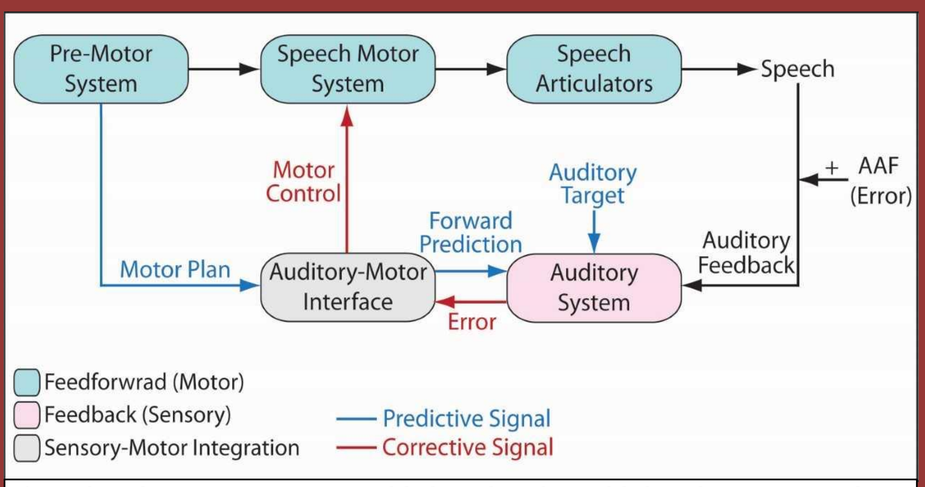


**Fig. 1.** The auditory-motor integration model of speech. In this model, the auditory-motor interface transforms speech motor plans into forward prediction of auditory feedback. The auditory system compares forward predictions with actual speech feedback to detect prediction errors in response to altered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

## METHODS

**Participants:** 16 participants with post-stroke aphasia and 16 neurologically intact control subjects with no history of speech, language, or neurological disorders were recruited for the study. The distribution of aphasia types were as follows: anomic: 5, Broca's: 6, conduction: 5. All participants passed a binaural hearing screening.

**Altered Auditory Feedback (AAF) Task**: Participants were instructed to produce a steady vowel sound of /a/ at their normal conversational pitch and loudness for 2 to 3 seconds. A short altered auditory feedback pitch shift of 200 ms was randomly applied to each trial at different time intervals between 750 and 1250 ms (Figure 2)

**Data Analysis**: Pitch frequency was segmented into epochs from -100 ms to 500 ms and were then converted from Hertz to Cents to calculate compensation magnitude. Pitch contours were averaged across all trials for pitch shifts and individual pitch contours were averaged across all subjects.

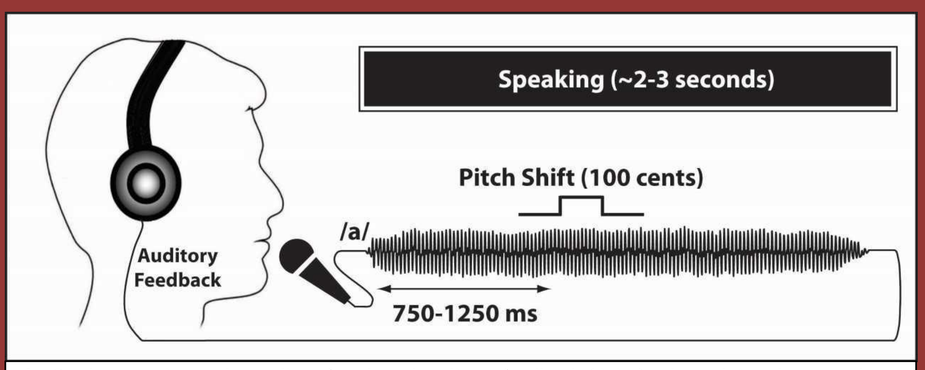


**Fig. 2.** The experimental paradigm for altered auditory feedback (AAF). The task involves steady production of the speech vowel sound /a/ for approximately 2–3 s while a pitch-shift stimulus (PSS) at ±100 cents alters the online auditory feedback of speech. The PSS stimuli are delivered at a randomized time between 750 and 1250 ms after the onset of speech.

## METHODS CONTINUED

**MRI Data Analysis**: Using a 3 T Siemens Trio System and head-coil, two magnetic resonance imaging sequences were collected of the 16 aphasic patients. A univariate lesion-symptom-mapping analysis was performed. In each region of interest, a mean intensity value analyzed with voxel-based permutation thresholding to predict diminished speech compensation responses. The correlation between diminished speech altered auditory feedback response at four points in time and location of the lesion was analyzed (Figure 3)

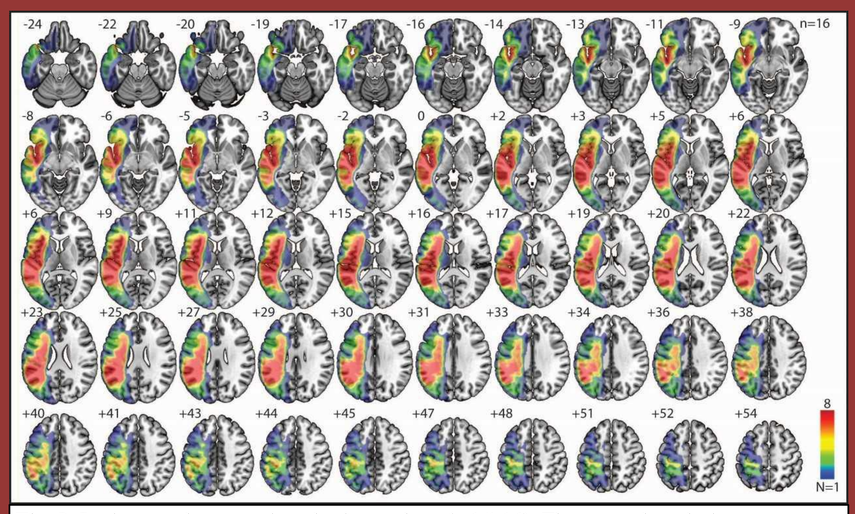


**Fig. 3.** Lesion overlap maps in aphasic speakers (n 1⁄4 16). The maps show lesion distribution on coronal (top) slices in MNI space for the sample, with warmer colors representing more lesion overlap across aphasic speakers (dark red areas represent lesion overlap across at least N 1⁄4 8 individuals).

## RESULTS

Both aphasia and control groups generated compensatory speech responses that opposed AAF direction for upward (Figure 4a) and downward (Figure 4b) pitch-shift stimuli.

There were no significant effects of group or AAF direction on the onset and peak latencies of speech compensation responses.

0-500 ms post stimulus revealed a significant main effect that the magnitude of speech compensation responses to AAF was larger in the control group.

The ROI-based LSM analysis revealed localized brain damage was associated with diminished compensatory speech responses to AAF.

No significant results were found for the rebound time window of 350-450 ms.

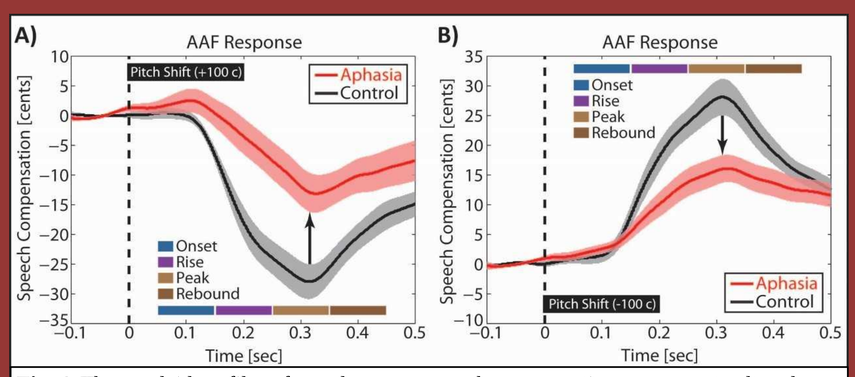


**Fig. 4.** The overlaid profiles of grand-average speech compensation responses to altered auditory feedback (AAF) for A) upward (þ100 cents) and B) downward ( 100 cents) pitch-shift stimuli in 16 speakers with aphasia and 16 control individuals. The temporal profile of responses are divided into onset (50–150 ms), rise (150–250 ms), peak (250–350 ms), and rebound (350–450 ms) time periods relative to the onset of pitch-shift stimuli at 0 ms.

## RESULTS CONTINUED

Diminished speech responses were found to be negatively correlated or best predicted from damage to the superior and middle temporal gyri for the time window of 50-150 ms, to the pars orbitalis at 150-250 ms, and damage to the supramarginal gyrus at 250-350 ms.

A significant positive correlation was found between overall aphasia severity and speech responses to AAF within the peak time window. This effect is from the correlation between speech AAF responses and the WAB scores on the speech recognition task.

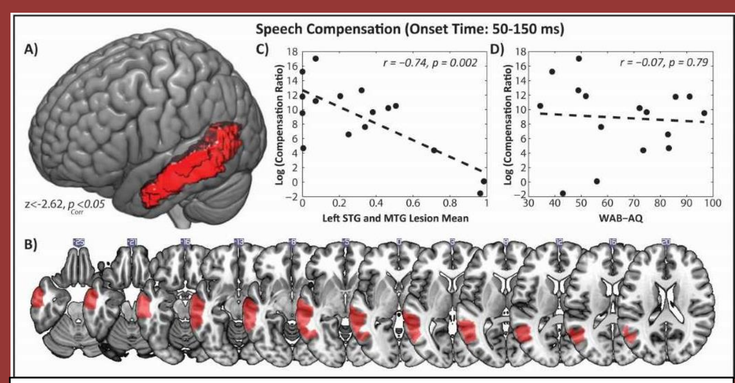


**Fig. 5.** Anatomical representation of lesion on the superior and middle temporal gyrus associated with diminished responses to AAF at the onset phase of speech compensation (50–150 ms) overlaid on A) reconstructed brain surface and B) coronal slices in MNI space. C) Demonstrates correlation between speech compensation ratio in aphasia normalized to the mean of control group and lesion volume on the superior and middle temporal gyrus. D) Demonstrates correlation between speech compensation ratio and patients' scores on the Western Aphasia Battery (WAB).

## DISCUSSION

Damage to the neural networks within the superior temporal gyrus, middle temporal gyrus, inferior frontal gyrus, and supramarginal gyrus are predictive of impaired sensorimotor function for speech error processing.

Early phase of speech motor output is dependent upon input from the auditory cortex to the motor system.

Deficits in rising phase of speech compensation magnitude dependent on damage to the inferior frontal gyrus associated with motor planning, programming, and execution of speech.

Damage to the supramarginal gyrus damage was the best predictor of impaired sensorimotor function for speech compensation magnitude associated with impaired speech error detection and correction of intended target.

Damaged areas correlated to WAB scores for aphasia participants.

## REFERENCES

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