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Sensorimotor impairment of speech auditory feedback processing in aphasia

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ABSTRACT

We investigated the brain network involved in speech sensorimotor processing by studying patients with post-stroke aphasia using an altered auditory feedback (AAF) paradigm. We combined lesion-symptom-mapping analysis and behavioral testing to examine the pervasiveness of speech sensorimotor deficits and their relationship with cortical damage. Sixteen participants with aphasia and sixteen neurologically intact individuals completed a speech task under AAF. The task involved producing speech vowel sounds under the real-time pitch-shifted auditory feedback alteration. This task provided an objective measure for each individual's ability to compensate for mismatch (error) in speech auditory feedback. Results indicated that compensatory speech responses to AAF were significantly diminished in participants with aphasia compared with control. We observed that within the aphasic group, subjects with lower scores on the speech repetition task exhibited greater degree of diminished responses. Lesion-symptom-mapping analysis revealed that the onset phase (50–150 ms) of diminished AAF responses were predicted by damage to auditory cortical regions within the superior and middle temporal gyrus, whereas the rising phase (150–250 ms) and the peak (250–350 ms) of diminished AAF responses were predicted with damage to the inferior frontal gyrus and supramarginal gyrus areas, respectively. These findings suggest that damage to the auditory, motor, and auditory-motor integration networks are associated with impaired sensorimotor function for speech error processing. We suggest that a sensorimotor integration network, as revealed by brain regions related to temporal specific components of AAF responses, is related to speech processing and specific aspects of speech impairment, notably repetition deficits, in individuals with aphasia.

Introduction

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. There is growing evidence that speech production impairments may be impacted by injury beyond the dedicated language production system, including lower-level speech motor mechanisms that are not directly influenced by language-dependent neural processes (Josephs et al., 2006; Whitwell et al., 2013; Basilakos et al., 2015). However, the underlying mechanisms of speech and language are often conflated, and challenges have persisted in providing definitive distinction between the neural processes that subserve these functions in the human brain (Fridriksson et al., 2013, 2015a). In addition, due to the large degree of variability in lesion anatomy and its behavioral consequences, a common and unified account of lesion-behavior relationship has not been well-established for aphasia. These challenges have been aggravated by the lack of consensus regarding how speech-language deficits should be qualified (or quantified), and by the fact that there are several combinations of characteristics that define specific behavioral impairments in post-stroke individuals with aphasia.

Converging evidence from several studies has corroborated the notion that certain aspects of behavioral impairment in aphasia are accounted for by damage to the sensorimotor network that supports auditory feedback processing during speech (Anderson, 1997;

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Fridriksson et al., 2010, 2013, 2015b; Buchsbaum et al., 2011). Deficits in sensorimotor integration have been implicated in conduction aphasia because of the hallmark difficulty with speech repetition, which requires the interfacing of incoming sensory information (i.e. the auditory target) with the outgoing motor production. Despite relative strengths in comprehension and fluency, individuals with conduction aphasia typically exhibit significant difficulty in correcting their speech errors during a speech repetition task, representing a severe impairment of sensorimotor integration mechanisms (Hickok et al., 2000; Josephs et al., 2006; Fridriksson et al., 2010; Buchsbaum et al., 2011). This notion is corroborated by findings that speakers with conduction aphasia are less sensitive to the disruptive effects of delayed auditory feedback as predicted by damage to the auditory-motor integration network (Boller and Marcie, 1978; Boller et al., 1978). Errors in production, or paraphasias, as well as less severe degrees of impairment with repetition are also common in other aphasia subtypes such as Broca's, Wernicke's, or anomic (Fridriksson et al., 2008, 2009, 2016). These overlapping behavioral symptoms are in support of the assumption that similar speech-language impairments result from similar underlying patterns of brain damage, and are indicative of a common neural substrate underlying speech sensorimotor integration. However, individuals with aphasia may show variable degrees of impairment along the continuum depending on the severity of stroke and lesion characteristics.

In the context of the dual-stream model (Hickok and Poeppel, 2004, 2007; Rauschecker and Scott, 2009; Rauschecker, 2011, 2012; Hickok, 2012a), deficits in speech sensorimotor integration have been attributed to damage to neural structures that support the strongly left-hemisphere dominant dorsal stream network. The dorsal stream includes areas within the prefrontal, temporal, and parietal cortices that constitute a sensorimotor network for planning, execution, and motor control of speech. The principles of this model 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 planned motor commands and their actual sensory (e.g., auditory and somatosensory) feedback (Hickok et al., 2011; Houde and Nagarajan 2011; Hickok, 2012a). According to this model, online speech control is not directly mediated by incoming sensory feedback from productions, but rather via internal representations of predicted sensory consequences of planned motor commands that provide rapid corrective feedback to speech controllers in case of erroneous productions even before the actual feedback has become available. During overt production, actual feedback can also be used to correct for speech feedback errors, and subsequently update the internal forward model representation. The schematic of the proposed dorsal stream network model for speech motor control is illustrated in Fig. 1. In this model, the auditory system, which codes the targets for speech gestures, interacts with the premotor and motor systems through a sensorimotor interface, which is proposed to be predominantly localized in the Sylvian fissure at the boundary between the parietal and temporal lobes (i.e., area Spt) (Hickok et al., 2003, 2008). This sensorimotor interface provides two potential sources of feedback control: internal, whereby motor plans are checked against their auditory targets via forward motor-to-sensory prediction and corrected internally if necessary prior to overt production, and external, whereby errors of produced speech can be compared against their targets for correction. Errors are detected via mismatches between sensory targets and motor predictions. In a laboratory environment, overt speech errors can be simulated by applying an online altered auditory feedback (AAF) stimulus to externally induce mismatch between the predicted and overtly detected speech. As a result, the error signal is translated into corrective motor commands via the auditory-motor interface for speech control. Evidence from several studies has supported the role of sensorimotor networks in speech error detection and correction and have shown that speakers generate compensatory motor responses to correct for alterations in their speech auditory feedback (Behroozmand et al., 2009; Chang et al., 2013; Greenlee et al., 2013; Niziolek and Guenther, 2013).

In aphasia, damage to different brain areas in the left hemisphere may disrupt sensorimotor interactions for speech error processing (Fridriksson, 2010; Fridriksson et al., 2013; Basilakos et al., 2014). For example, individuals with conduction aphasia are capable of detecting errors in their own speech due to preserved auditory error detection mechanisms, but they make frequent speech repetition errors possibly because motor speech error processing is disrupted by inaccurate forward predictions, or because detected errors are not translated into corrective commands due to damage to the auditory-motor interface (Baldo et al., 2008; Fridriksson et al., 2010). Historically, damage to the arcuate fasciculus was attributed to this pattern of deficits, but more recent neuroimaging studies have indicated that a posterior region of the parietal-temporal boundary (area Spt) plays an integral role for speech sensorimotor integration (Buchsbaum et al., 2011; Hickok et al., 2011; Hickok, 2012a; Rogalsky et al., 2015). Despite the existing evidence, our understanding of sensorimotor feedback in aphasia has been mostly limited to examining repetition deficits within the conduction aphasia group, and therefore, comprehensive knowledge about the relationship between lesion characteristics and impaired sensorimotor integration in aphasia is scant. To overcome this shortcoming, it is crucial to conduct large-scale investigations that do not rely on data only from a certain group of post-stroke individuals (i.e., conduction aphasia), but rather on

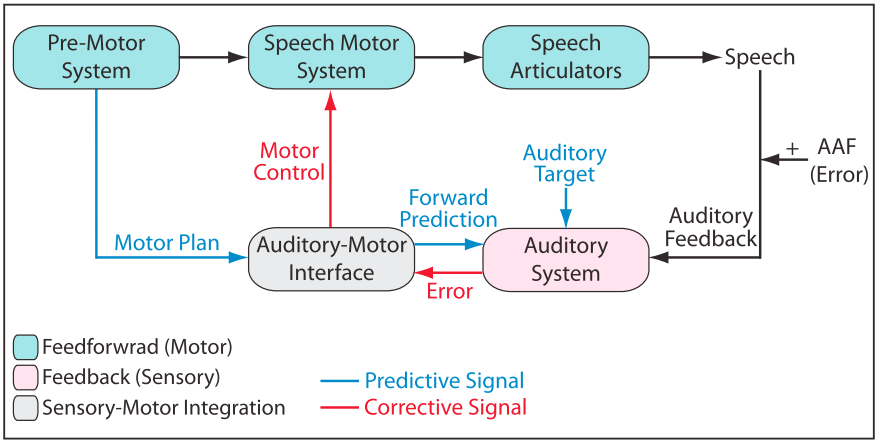

Fig. 1. The auditory-motor integration model of speech. In this model, the auditory-motor interface transforms speech motor plans into forward prediction of auditory feedback. The auditory system compares forward predictions with actual speech feedback to detect prediction errors in response to altered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