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Testing hypotheses about the underlying deficit of apraxia of speech through computational neural modelling with the DIVA model

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Abstract

Purpose: A recent behavioural experiment featuring a noise masking paradigm suggests that Apraxia of Speech (AOS) reflects a disruption of feedforward control, whereas feedback control is spared and plays a more prominent role in achieving and maintaining segmental contrasts. The present study set out to validate the interpretation of AOS as a possible feedforward impairment using computational neural modelling with the DIVA (Directions Into Velocities of Articulators) model.
Method: In a series of computational simulations with the DIVA model featuring a noise-masking paradigm mimicking the behavioural experiment, we investigated the effect of a feedforward, feedback, feedforward + feedback, and an upper motor neuron dysarthria impairment on average vowel spacing and dispersion in the production of six/bVt/speech targets.
Result: The simulation results indicate that the output of the model with the simulated feedforward deficit resembled the group findings for the human speakers with AOS best.
Conclusion: These results provide support to the interpretation of the human observations, corroborating the notion that AOS can be conceptualised as a deficit in feedforward control.

Keywords: *apraxia of speech; computational modelling; vowel acoustics; feedback masking*

Introduction

Apraxia of Speech (AOS) is a neurogenic motor speech disorder that is defined as an impairment in the planning and/or programming of speech movements (Deger & Ziegler, 2002; Duffy, 2005; Van der Merwe, 1997). The speech of people with AOS is characterised by slow speech rate, abnormal prosody, abnormal speech sound and syllable segmentation, speech sound distortions, and speech errors that are inconsistently present but relatively consistent in type and location (Duffy, 2005; Maas et al., 2008). AOS typically results from brain lesions to the left cerebral hemisphere, but more specific lesion locations reported in the literature diverge. Most reports indicate lesions in left inferior frontal regions (e.g. Dronkers, 1996; Graff-Radford et al., 2014; Hickok et al., 2014; Hillis, et al., 2004; Itabashi et al., 2016; Richardson, Fillmore, Rorden, LaPointe, & Fridriksson, 2012), however, other regions have also been reported, including the parietal cortex (e.g. Hickok et al., 2014; McNeil, Weismer, Adams, & Mulligan, 1990), basal ganglia (Seddoh et al., 1996), and right frontal cortex and basal ganglia structures (Balasubramanian & Max, 2004). The precise location of the lesion responsible for AOS thus remains subject of debate. Likewise, the precise nature of the disorder remains poorly understood.

One of the main difficulties in isolating the underlying deficit(s) is diagnostic circularity. The ability to investigate the characteristics underlying AOS requires *pure* cases of AOS selected on the basis of clear-cut criteria, which are only available as a result of research. As lesion inducing medical accidents such as strokes, brain injuries, or tumours rarely produce isolated and one-dimensional deficits, pure cases are rare and symptom profiles show considerable variation between individuals as well as a large overlap in symptomatology with other speech disorders. Additionally, when confronted with a partial breakdown, the

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