

A Multivariate Behavioural and Lesion-Symptom Mapping Investigation of the Core Aspects of Fluent Speech Production

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A multivariate behavioural and lesion-symptom mapping investigation of the core aspects of fluent speech production

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Introduction

Fluent speech production requires the rapid coordination of multiple cognitive and neural systems, including syntax, sentence planning, working memory, semantics, phonology, and articulatory motor control (e.g., Gordon, 2020; Matchin & Hickok, 2020; Mirman et al., 2019; Nozari & Faroqi-Shah, 2017). Building on prior data-driven work, we used Quantitative Production Analysis (QPA; Rochon et al., 2000; Saffran et al., 1989) to derive a diverse and comprehensive set of narrative speech production measures that include fluency and syntax at lexical, utterance, and sentence levels. These measures were combined with general measures of language impairment (aphasia quotient, naming accuracy) and measures of semantic and phonological deficits to situate fluency and syntax deficits in the context of well-established dimensions of aphasia variability: overall severity, semantic deficit, and phonological deficit.

Methods

Behavioural data consisted of 13 QPA measures plus 8 additional measures (see Figure 1), which were available for 69 participants with aphasia following left hemisphere stroke. All participants were right-handed native English speakers. The behavioural measures were entered into a principal component analysis (PCA) with bifactor rotation – a latent structure model where each item loads on a general factor that reflects what is common among the items, and two or more orthogonal factors that explain variance not accounted for by the general factor (e.g., Reise et al., 2010). Lesion data were available for 58 of the participants and each factor score was analysed with multivariate lesion-symptom mapping using SCCAN (Pustina et al., 2018). Sparseness was optimised independently for each analysis using 8-fold cross-validation.

Results

The PCA produced a four-factor solution that accounted for 70.6% of the variance in the data, with factors corresponding to (1) length and complexity of output (mean sentence and utterance length, proportion of words in sentences, infection and embedding); (2) lexical syntax (proportions of verbs, closed class words, and pronouns); (3) semantics (CCT, semantic discrimination, semantic errors); (4) phonology (word and nonword repetition, phonological errors). Figure 1 shows the factor loadings and Figure 2 shows the SCCAN LSM results. Deficits on the lexical-syntax factor were associated with damage in a relatively small set of fronto-parietal regions (optimal sparseness = 0.216, CV correlation = 0.517, p < 0.001): IFG pars triangularis, precentral gyrus, and dorsal portion of the inferior parietal lobule. Deficits in length and complexity of speech output were associated with a somewhat larger temporo-parietal region (optimal sparseness = 0.576, CV correlation = 0.603, p <

0.001): SMG and postcentral gyrus, posterior STG and MTG, and Heschl's gyrus. For comparison (and not surprisingly), overall aphasia severity (WAB AQ) was associated with damage to a large portion of the MCA territory (optimal sparseness = 0.798, CV correlation = 0.653, p < 0.001) that included parietal, temporal, and frontal regions.

Conclusions

These results suggest a cognitive and neural dissociation between lexical-level syntactic production (use of pronouns, closed class words, verbs) and the length, complexity, and structure of sentences, utterances, and phrases; and both of these functions are relatively separate from lower-level phonological and semantic abilities.

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Figure 1. Bifactor PCA loadings. Each factor is shown in a separate panel with positive loadings in blue and negative loadings in red. Note: Sent = Sentence(s), Len = Length, Utter = Utterance, Prop = Proportion, Aux = Auxiliary.



Figure 2. SCCAN LSM results for lexical-syntax (top row), length/complexity of speech output (middle row), and aphasia severity (bottom row).