

Impaired lexical retrieval is one of the hallmark impairments in aphasia and is particularly evident in confrontational naming. In general, naming errors in aphasia can be categorized as having either a phonological or semantic basis. Numerous studies have focused on naming errors in aphasia and some have related error types to brain anatomy (e.g. Schwartz et al., 2009, 2012).

One of the prevailing models of lexical retrieval was introduced over two decades ago by Dell and colleagues (1986). The dual-route interactive two-step model has been used to explain speech error patterns not only in healthy individuals (Dell, 1986), but also individuals with aphasia (Martin et al., 1994, Schwartz et al., 2006). More recently, the naming errors of individuals with aphasia have been used to support the dual-stream model (Hickok & Poeppel, 2007). The dual stream model of language processing posits two anatomically distinct streams that facilitate language processing, where a bilateral ventral stream maps speech sound to meaning in order to perceive and understand speech and a strongly left-dominant dorsal stream maps speech sounds to motor speech plans to produce and monitor speech production.

Dell and colleagues (2013) reported findings of a voxel-based lesion-parameter mapping study. Naming data from the Philadelphia Naming Test (PNT; Roach, et al., 1996) and the Philadelphia Repetition Test (PRT) were used to compute model parameters based on where in the model the primary impairment was thought to load: semantic-weights (s-weights), phonological-weights (p-weights), and non-lexical weights (nl-weights). These weights are thought to reflect the strength of the connections between semantic, lexical, phonological, and nonlexical levels represented in the dual-route interactive two-step model. Dell and colleagues (2013) mapped the s, p, and nl parameters for naming and word and nonword repetition tasks onto lesion maps of 103 individuals with chronic aphasia. They suggested their voxel-lesion parameter maps (VLP-maps) and behavioral findings, specifically pertaining to word repetition support the model.

We replicated the procedures of Dell and colleagues (2013) for the naming component and mapped the s and p-parameters onto brain maps of persons with aphasia within our dataset. We were particularly interested in the semantic weights to characterize semantic impairments because semantic errors are inherently more easily defined and classified than phonological or nonlexical errors. With regard to the dual stream model, we predicted s-weighting would be predicted within the ventral stream, and p-weights within the dorsal stream.

The PNT was administered to 63 persons with chronic aphasia. Naming attempts were categorized the same way as Dell et al (2013): correct naming, semantic, form-related word, mixed, unrelated word, and non-word errors. Relying on the same algorithm as Dell et al. (WebFit; <http://langprod.cogsci.illinois.edu/cgi-bin/webfit.cgi>), s-weights and p-weights were calculated for each participant based on their naming error pattern from the PNT and PRT.

Structural brain images for each participant were acquired using a Siemens 3T MRI system with a 12-element head coil. All participants underwent a high-resolution T1 and T2 MRI

sequences. Brain damage was demarcated on T2 MRI by experienced clinicians for the purpose of lesion-parameter mapping.

Voxel-based lesion-parameter mapping was used to relate localized brain damage to s and p-weights. Univariate and multivariate linear regression was completed using an in-house code written in MatLab (The MathWorks, Natick, MA) and corrections for multiple comparisons completed using permutation thresholding with 2,000 (univariate) and 3,000 (multivariate) permutations (Rorden et al., 2009).

Consistent with the Dell et al. (2013) and other prior work, we found a minimal correlation between s and p-weights ($r=.162$, $p=.196$). This finding suggests for our dataset, s and p-parameters were independent and parameters would map onto different brain regions.

The univariate analysis revealed damage to six Brodmann areas (BA) associated with semantic weights: BA 20 (inferior temporal gyrus; $z=2.41$), 21 (middle temporal gyrus; $z=3.12$), BA 22 (superior temporal gyrus; $z=3.19$), BA 41 (auditory cortex; $z=-2.71$), BA 42 (auditory cortex; $z=3.28$), and BA 48 (medial surface of temporal gyrus; $z=2.46$). Surprisingly, no regions survived thresholding ($z=1.94$) for p-weights.

The multivariate analysis (where both s and p-weights are entered into the same analysis) revealed five Brodmann areas associated with s-weights: BA 20 (inferior temporal gyrus; $z=-2.02$), BA 21 (middle temporal gyrus; $z=-3.09$), BA 22 (superior temporal gyrus; $z=-3.09$), BA 41 (auditory cortex; $z=-2.31$) and BA 42 (auditory cortex; $z=-3.09$). Again, no regions survived thresholding ($z=-0.79$) for p-weights. In summary, we found damage to the auditory cortex and Wernicke's area were strong predictors of s-weights.

In contrast to Dell et al. (2013), our results did not reveal diffused damage to the left hemisphere to be related to s-parameter weights. Furthermore, we did not find a significant relationship between cortical damage and p-weights. Dell and colleagues' analysis revealed a large portion of the left hemisphere for s-weights; however, after controlling for lesion size, the voxels significant for s-weights were found in the anterior temporal lobe (anterior, superior and middle temporal gyri, and temporal pole), prefrontal cortex (middle and inferior frontal gyri), parietal-temporal junction, and angular gyrus. They did not find significant voxels in the posterior superior temporal gyrus or middle temporal gyrus, which includes Wernicke's area. Based on their findings, Dell and colleagues questioned their model's single function characterization of s-weights given the widespread pattern of VLP-maps, suggesting s-weights may contribute to multiple cognitive functions.

In our study, we found a significant relationship between s-weights and damage to the middle temporal gyrus, superior temporal gyrus, and the primary auditory cortex. Further, these results are consistent with Schwartz et al. (2009), a lesion symptom mapping study that found a concentration of significant voxels in Brodmann area 21 associated with semantic errors on the PNT. However, unlike Schwartz and colleague, damage to regions 37, 45/46 was not

significant in our analyses. Our findings show s-weights are best predicted by damage to auditory cortex and Wernicke's area.

Most surprisingly, we did not find localized brain damage associated with p-weights, whereas Dell and colleagues found significant damage to regions in the anterior part of the dorsal stream (SMG, post- and precentral gyrus and insula), and posterior, superior and middle temporal gyri, including Wernicke's area, to predict p-weights. Based on these findings, Dell et al argued phonological errors are produced in stages after retrieval of the lexical form. This is particularly interesting because the posterior temporal areas found in their p-weights analysis are the same as our findings for the s-weights.

The discrepancies between our findings and those of Dell and colleagues may be due, in part, to failures of the model to accurately predict naming errors which, in turn, can be related to localized brain damage. Our findings for s-weights overlap with a few significant areas for p-weights in the Dell and colleagues' paper, suggesting these areas are either both responsible to some degree for both errors types, in which case the model cannot clearly classify errors within specific and distinct brain regions or there are potential methodological differences that could account for such overlap. Further, we failed to find significant voxels associated with p-weights. These discrepancies highlight the need for replication of studies of this kind.

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