Revisiting speech repetition with lesion-symptom mapping: contributions of insula, temporo-parietal cortices and the arcuate fasciculus

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Introduction

Patients with aphasia following left-hemisphere stroke typically show impaired speech repetition. This impairment has classically been associated with lesions to a prominent white matter tract - the arcuate fasciculus (AF). More recent lesion-symptom mapping (LSM) studies in acute aphasia have additionally implicated cortical grey matter in the left inferior parietal (IP) and insular cortices (e.g., Fridriksson et al., 2010; Kümmerer et al., 2013). By contrast, a left posterior superior temporal cortex lesion, without involvement of the AF, has been implicated in chronic aphasia (e.g., Baldo et al., 2012).

Aside from varying in terms of chronic/acute strokes, these studies have also employed different assessment measures (repetition subtests from aphasia batteries vs experimental tasks) and analysis techniques, especially relating repetition score as regressor (e.g., Baldo et al., 2012) or additionally covarying for comprehension and/or lesion volume in regression analyses (e.g., Fridriksson et al., 2010; Kümmerer et al., 2013). We therefore investigated repetition deficits in a sample of chronic stroke patients using a standardised aphasia battery, and tested the effects of including comprehension and lesion volume as covariates in the LSM analyses.

Experimental design and analysis

We assessed 30 patients (15 male, mean age 62, SD 9.7 years) on the Western Aphasia Battery - Revised (WAB-R; Kertesz, 2007). Inclusion criteria include: (i) ability to give consent; (ii) single left-hemisphere cortical stroke; (iii) English as first language; (iv) time post-stroke > 6 months; (v) no prior neurologic, psychiatric, or substance abuse history. Aphasia Quotient scores on the WAB-R and lesion volumes are displayed in Table 1 below, indicating a relatively wide range of severity.

<table>
<thead>
<tr>
<th>Aphasia Quotient</th>
<th>Mean [SD]</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Repetition</td>
<td>8.36 (1.84)</td>
<td>3.8 - 10</td>
</tr>
<tr>
<td>Comprehension</td>
<td>9.1 (1.31)</td>
<td>5.5 - 10</td>
</tr>
<tr>
<td>Spontaneous Speech</td>
<td>16.47 (3.54)</td>
<td>7 - 20</td>
</tr>
<tr>
<td>Naming</td>
<td>8.26 (1.3)</td>
<td>2.4 - 10</td>
</tr>
<tr>
<td>Aphasias Quotient</td>
<td>85.5 (14.83)</td>
<td>43.6 - 100</td>
</tr>
<tr>
<td>Lesion Volume (ml)</td>
<td>11.6157</td>
<td>-5.0 - 5.14</td>
</tr>
</tbody>
</table>

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Conclusion

Our results confirm repetition impairments in chronic aphasia are predominantly associated with a lesion of the left posterior superior temporal cortex (e.g., Baldo et al., 2011), without significant involvement of the AF fibre tract, IP or insular cortices (cf. Fridriksson et al., 2010; Kümmerer et al., 2013). This relationship remained significant when lesion volume was covaried (cf., Kümmerer et al., 2013), although not when comprehension was covaried (cf. Fridriksson et al., 2010).

The lesions responsible for repetition impairments in acute and chronic stages of recovery may therefore differ. Repetition and auditory comprehension impairments are typically highly correlated in aphasia. Thus, null results when comprehension is employed as a covariate might reflect the influence of multicollinearity that can result in a increased type II error rate in regression analyses.

References


Acknowledgements

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Figure 2. LSM for WAB-R Repetition subtest scores rendered on the average control template, threshold at p < .001 (uncorrected) for visualisation.

Figure 3. (A) LSM for WAB-R Repetition subtest score with lesion volume covered, rendered on the average control template, threshold at p < .001 (corrected) for visualisation. (B) Cutaway showing LSM with AF fibre tract overlaid in cyan.