Reading without the left ventral occipito-temporal cortex

Seghier, Neufeld, Zeidman, Leff, Mechelli, Nagendren, Riddoch, Humphreys & Price, 2012
Introduction

- Damage to LvOT typically causes alexia without agraphia
- Recovery despite damage to read short, familiar words.
- Neural pathway to rapidly identify whole words, independently of LvOT.

*How is this possible at the neural level?*
Overview

- Case study of LvOT impaired patient
- fMRI and DCM methods to identify most likely w/o LvOT pathway.
- Control group of skilled readers
Authors’ hypothesis:

Connection between left inferior occipital cortex and the left superior temporal temporal cortex.
Method:

- **Subjects**: 29 healthy right-handed native English speakers (16 female, ages 33 ± 18 years) with no neurological or psychiatric history, and normal/corrected to normal vision.

Assessment of AH, 46 months post stroke:

- Identification of letters and numbers
- RT of 160 words manipulated in length (3, 5, 7, 9) and frequency (high, low).
- Familiar vs. unfamiliar non-words:
- Exception word reading: folk, doubt
- Picture naming from BORB

In summary: reading was more difficult for unfamiliar non-words and atypically spelled words.
Functional Imaging Paradigm

- **Stimuli:** short (3-5 letters), familiar object names with high frequency
- **Four conditions:** reading aloud, picture naming aloud, *semantic decisions on written words* and *semantic decisions on pictures of objects*.
- **Four baseline conditions:** saying 1,2,3 to meaningless Greek strings of letters, saying 1,2,3 to meaningless non objects, *perceptual decisions on Greek letter strings*, *perceptual decisions on non objects*.
- **Stimuli presentation:** relatively fast- 4.5s
DCM analyses

- Region selection: 15 regions more activated in patient than subjects when reading out loud.
- Step by step model investigation, plausible models selected by a Bayesian model selection.
- For controls: 31 models for each one, varying in connections to LvOT.

1. How does increasing connections to LvOT influence?
2. Compare connection strengths in controls and patients.

<table>
<thead>
<tr>
<th>Regions</th>
<th>Controls coord.</th>
<th>Pt &gt; Ct Z score</th>
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<tbody>
<tr>
<td>Ventral occipital cortex (v)</td>
<td>22 - 94 2</td>
<td>4.5</td>
</tr>
<tr>
<td>Dorsal occipital cortex (d)</td>
<td>16 92 6</td>
<td></td>
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<tr>
<td>Ventral occipito-temporal</td>
<td>12 - 88 8</td>
<td></td>
</tr>
<tr>
<td>Superior temporal sulcus</td>
<td>26 92 16</td>
<td>4.2</td>
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<tr>
<td>Planum temporale (PT)</td>
<td>30 - 72 32</td>
<td></td>
</tr>
<tr>
<td>Ventral premotor cortex (</td>
<td>30 - 88 22</td>
<td></td>
</tr>
<tr>
<td>Motor cortex (M)</td>
<td>44 - 76 - 12</td>
<td>1.9</td>
</tr>
<tr>
<td>Globus pallidus (GPe)</td>
<td>44 - 62 - 16</td>
<td></td>
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<td></td>
<td>36 - 44 - 18</td>
<td></td>
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<tr>
<td></td>
<td>54 - 30 4</td>
<td>4.3</td>
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<tr>
<td></td>
<td>64 - 26 8</td>
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<td></td>
<td>48 - - 36 10</td>
<td>2.7</td>
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<td>56 - 36 12</td>
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<td>60 12 0</td>
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<td></td>
<td>50 - 10 42</td>
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<td></td>
<td>42 - 12 36</td>
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<td></td>
<td>54 - 16 4</td>
<td>n.s.</td>
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<td>4.8</td>
</tr>
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</table>
Results

- **Step One:** Which visual processing area is the input to the rest of the system of the patient? **Left vOCC** as the input region.
- **Step two:** Which of the connections best explains the patient's data in the left hemisphere 5-region model? A model in which both STS and PT were connected to vOCC, vPM and M.
- **Step three:** How does evidence for the 5 region model change when it is connected to LvOT? **As connections to LvOT increase, model evidence decrease.**
- **Step four:** Does the right hemisphere contribute in the patient?
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Model A: 6 region model of the right homologs with vOCC as the driving region

Model B: same as model A with right GPe included.

Model C: 
same as A, included 6 LH homologs. 12 region model, with all interhemisphere connections homotopic.

Three versions- input to LvOCC, RvOCC and both vOCC.
Summary:

1. Left vOCC as driving input region.
2. STS and PT were involved in linking visual information from LvOCC to the articulation areas in the motor cortex.
3. LvOT did not contribute to the reading network.
4. RH activation was significantly influenced by LH homotopic activation, with insignificant interregion connections in the RH.
DCM analyses of Control Data:

- LvOT connectivity was strongly favored.
- vOCC $\rightarrow$ STS, STS $\rightarrow$ vPM/M pathway connections were significantly stronger in patient.
- Controls also used the non-LvOT route:
  a) Connections modulate by naming and reading
  b) Correlation between the connection strengths of vOCC $\rightarrow$ STS and STS $\rightarrow$ vPM/M ($0.72 < r < 0.93$)
Intersubject variability in the reading pathways used by controls:

- vOCC→STS→vPM/M highly dissimilar from LvOT pathway. Good compensatory route when LvOT is damaged.
- Subjects with weaker LvOT connections relied more on STS pathway. However, they were not better/worse on easy reading tasks.
Discussion

- Left STS route can support accurate reading of rapidly preserved words.
- Not claiming normal reading!
- STS pathway observed in skilled readers, and dissociable from LvOT pathways
- Claim: Left STS pathway is involved in the integration of semantics with phonology
Left STS pathway is involved in the integration of semantics with phonology:

1. Location
2. Tasks that showed left STS activation in fMRI
3. Modulation of connections to and from lefts STS in DCM results
4. Recent MEG study of written word processing
5. Recent fMRI study of silent reading in normal readers
6. fMRI studies show activation in left STS correlated with word length, naming latencies and RT during mono-syllabic reading aloud
Unexpected findings:

1. No abnormal involvement of superior parietal lobe. Longer words? Pseudowords?
2. Lack of Right Hemisphere involvement in patient.
   a) different paradigm
   b) 4 years after stroke
   c) non-optimal conditions, constraints in model.
STS pathway

- Insufficient for patient, but enough for some controls.
- Controls had a processing power that was unavailable to the patient.
- Low connection in LvOT is enough?
- Pathways in the vicinity of LvOT damaged
- Keep in mind:
  1) patient’s lesion included surrounding white matter pathways
  2) bottom-up and top-down interactions between LvOT and STS
  3) Recovery can occur even decades after stroke
My thoughts

- Complexity
- Stimuli
- Future research in normal readers
- More case studies