Right anterior superior temporal activation predicts auditory sentence comprehension following aphasic stroke

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Previous studies have suggested that recovery of speech comprehension after left hemisphere infarction may depend on a mechanism in the right hemisphere. However, the role that distinct right hemisphere regions play in speech comprehension following left hemisphere stroke has not been established. Here, we used functional magnetic resonance imaging (fMRI) to investigate narrative speech activation in 18 neurologically normal subjects and 17 patients with left hemisphere stroke and a history of aphasia. Activation for listening to meaningful stories relative to meaningless reversed speech was identified in the normal subjects and in each patient. Second level analyses were then used to investigate how story activation changed with the patients’ auditory sentence comprehension skills and surprise story recognition memory tests post-scanning. Irrespective of lesion site, performance on tests of auditory sentence comprehension was positively correlated with activation in the right lateral superior temporal region, anterior to primary auditory cortex. In addition, when the stroke spared the left temporal cortex, good performance on tests of auditory sentence comprehension was also correlated with the left posterior superior temporal cortex (Wernicke’s area). In distinct contrast to this, good story recognition memory predicted left inferior frontal and right cerebellar activation. The implication of this double dissociation in the effects of auditory sentence comprehension and story recognition memory is that left frontal and left temporal activations are dissociable. Our findings strongly support the role of the right temporal lobe in processing narrative speech and, in particular, auditory sentence comprehension following left hemisphere aphasic stroke. In addition, they highlight the importance of the right anterior superior temporal cortex where the response was dissociated from that in the left posterior temporal lobe.

Keywords: aphasia; auditory sentence comprehension; stroke

Abbreviations: CAT = comprehensive aphasia test; fMRI = functional MRI; SPM = statistical parametric mapping

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Introduction

The neural basis of speech recovery following aphasic stroke remains unknown. It is generally accepted that there will be some degree of functional reorganization following cerebral damage. This will either involve changes within the pre-existing language network (e.g. Leff et al., 2002) or the recruitment of supplementary brain areas in the left or right hemisphere. Clinical observations combined with structural neuroimaging have suggested that if the lesion is large the prognosis is poor; particularly, it has been proposed, if there is ventral extension of the infarct into the middle and inferior temporal gyri (Selnes et al., 1983; Naeser et al., 1987; Hart and Gordon, 1990). The inverse correlation between the size of the left hemisphere lesion and the degree of language recovery (Deumeurisse et al., 1985; Heiss et al., 1993) has led some to suggest that it is the left rather than right hemisphere that plays an important role in recovery. However, there are also examples of patients with left hemisphere strokes, whose language impairments were impaired further when a second stroke damaged the right hemisphere (Basso et al., 1989). This suggests that, at least in these patients, the right hemisphere was also contributing to language processing.
Functional imaging studies of aphasics’ comprehension have differed in their conclusions concerning the contribution of left and right hemisphere activation. This inconsistency may partly reflect the tasks tested. During auditory repetition tasks, for example, the majority of functional imaging studies have emphasized that good language recovery depends on left hemisphere activation, although right hemisphere activation may be observed when left hemisphere regions are no longer available (Heiss et al., 1997, 1999; Karbe et al., 1998; Kessler et al., 2000). In contrast, during auditory comprehension tasks, a greater emphasis has been placed on the role of the right hemisphere. For example, when patients with left temporal lobe damage listened to words presented at varying rates, right posterior temporal activation was more correlated with increasing word rate than in control subjects (Leff et al., 2002). Right hemisphere activation in patients with left temporal damage has also been shown to correlate with response accuracy when subjects respond to simple and repetitive auditory instructions (Musso et al., 1999) or perform semantic decisions on auditory words (Sharp et al., 2004). Critically, however, these results do not necessarily reflect a ‘laterality shift’ because the same right hemisphere areas are also engaged by neurologically normal subjects (Warburton et al., 1999; Zahn et al., 2002, 2004). This is clearly illustrated in the study by Sharp et al. (2004) who found that right anterior fusiform activation correlated with performance accuracy in neurologically normal controls as well as the patients.

An outstanding question that we wish to address is whether right hemisphere activation contributes to long-term language functioning after aphasic stroke. Previous studies have addressed this issue by investigating how activation changes over the course of recovery (Cardebat et al., 2003; Fernandez et al., 2004); by correlating activation with behavioural responses on the task used during scanning (Musso et al., 1999; Blasi et al., 2002; Sharp et al., 2004) or by correlating activation with language assessments conducted outside the scanner (Rosen et al., 2000; Blank et al., 2003; Noppeney et al., 2005). Although significant activation changes over time or within scanning session have been observed, correlations between activation and language performance outside the scanner have not revealed significant effects in aphasic patients. For example, neither Blank et al. (2003) nor Rosen et al. (2000) observed a relationship between right hemisphere activation and speech production ability measured outside the scanner. These authors, therefore, concluded that right hemisphere activation may represent behavioural strategies (Rosen et al., 2000) or transcallosal disinhibition (Blank et al., 2003) that do not necessarily contribute to the recovery of speech production processes. The role of the right hemisphere, however, is likely to be critically dependent on the task. For example, according to the model of right hemisphere language proposed by Zaidel (1976), the right hemisphere should selectively contribute to the recovery of language comprehension but not to the recovery of language production.

In the present study, we investigated the brain regions that contribute to speech comprehension processes by regressing activation evoked when patients listened to short stories with (i) auditory sentence comprehension and (ii) subsequent memory abilities, measured outside the scanner. The functional MRI (fMRI) paradigm engaged subjects in automatic ‘on-line’ narrative speech comprehension without any requirement to make a response. The intention was to reduce activation related to working memory and other executive functions that are not directly related to normal, on-line comprehension of narrative speech. We predicted that the activation pattern would vary, across subjects, according to their auditory comprehension abilities. In addition, we assessed how activation changed with auditory recognition memory as measured by a post-scanning surprise story recognition test. This allowed us to distinguish linguistic (auditory comprehension) from metalinguistic executive (auditory recognition memory) processes that are not directly related to normal, on-line narrative speech comprehension. Although speech comprehension proficiencies may well depend on working memory capacity (Just and Carpenter, 1992; MacDonald et al., 1992) and the speech comprehension problems of some patients with aphasia have been attributed to verbal working memory deficits (Hermann et al., 1992; Wilson and Baddeley, 1993; Aziz et al., 2002) previous functional imaging studies have not dissociated activation that is related to understanding speech (primarily at a linguistic level) from executive (metalinguistic) aspects of speech processing.

In summary, previous functional imaging studies have reached divergent conclusions concerning the neural mechanisms of recovery and this may reflect the very variable stimuli, tasks and patient groups that have been investigated. It, therefore, remains unknown as to whether right hemisphere activation reflects a maladaptive strategy or a beneficial one. In non-fluent patients, it has been proposed that the pattern of overactivation may limit, rather than account for, aphasia recovery (Belin et al., 1996; Rosen et al., 2000). Each hemisphere may be important, depending on the type of language behaviour and when it was examined (Weiller et al., 1995; Basso et al., 1996; Belin et al., 1996; Mimura et al., 1998; Cao et al., 1999; Ansaldo et al., 2002; Hund-Georgiadis et al., 2002; Ansaldo et al., 2004). In this study we aimed to differentiate auditory sentence comprehension (linguistic) processes from auditory recognition memory processes (executive, metalinguistic) in a group of chronic aphasic patients by regressing activation evoked when patients listened to short stories with (i) auditory sentence comprehension and (ii) subsequent memory abilities, measured outside the scanner.

**Methods**

**Participants**

Seventeen patients (12 males, mean 62 years, SE 2.7) and 18 neurologically normal subjects (8 males, mean 58 years, SE 2.8) were studied. Each had English as their first language, was right-handed and gave informed consent to participate in the study.
All patients acquired their aphasia following a single left hemisphere stroke. They had a volumetric MRI to aid co-registration with their fMRI scan and to define their allocation to the appropriate patient group based on the site of their lesions (Fig. 1). One group of eight patients had left temporal lobe damage (Temporal patients); mean age 58 years, mean time since infarct 41 months. The remaining nine patients who had left hemisphere lesions sparing the temporal lobes acted as an aphasia control group (Control patients); mean age 66 years, mean time since infarct 48 months. There was no significant difference between the groups in terms of age ($P = 0.09, \text{SE} = 4.3$) or time of study after infarct ($P = 0.69, \text{SE} = 17.9$).

Patients’ language abilities were assessed using the comprehensive aphasia test (CAT) (Swinburn et al., 2005). For single words, subsets of items manipulate variables such as word frequency, imageability, animacy and length, with other confounding variables controlled. Imageability values were drawn from the MRC Psycholinguistic Database (Coltheart, 1981) and word frequency from Francis and Kucera (1982). In the sentence level subtests, clausal and syntactic complexities have been manipulated (varying factors like sentence ‘reversibility’ and active/passive structures). With all spoken output tasks in the CAT the focus of interest is in the linguistic rather than motor aspects of speech production. Therefore, articulatory errors not affecting the perceptual identity of the target are scored correct so that patients with dysarthria would not be penalized.

**Scanning stimuli**

Subjects listened to two conditions during their fMRI scans: stories (St) and reversed version of the same stories (RSt). The length of each story was 64 words (25 s duration). All stories were recorded by a single female narrator. A typical example is:

‘Yesterday one person was killed. There was a big storm. The storm blew down many trees. John was driving home after work. The storm was very bad. A tree fell on the road. John did not see the tree. John’s car hit the tree. The police found John. The police called the doctor. It was too late. John died on the way to the hospital.’

Using SoundEdit16™ computer software the waveform for each story was reversed (RSt) and used as the baseline condition. Reversing the speech signal destroys intelligibility while retaining the overall spectrotemporal complexity of the original speech signal. The reversed versions of the narratives were expected to control for early acoustic processing of the speech signal in both left and right superior temporal cortex. However, as reversing speech destroys normal segmental stress patterns, suprasegmental prosody and voice identity, it was expected that the activation patterns in the contrast of St with RSt would not reflect linguistic processing alone.

During each subject’s study they heard 32 stories (St) and 8 reversed stories (RSt), with each story and its reversed version used only once per subject. The order of presentation was randomized, both within and between subjects. Presentation was binaural with the volume set at a comfortable level for each subject. The subjects were asked to simply listen and try to understand the story. They were aware that the reversed stories were unintelligible but were asked to pay attention to the sounds.

To ensure that the subjects had attended to the stimuli, an eye-tracker was used to check that the subjects were awake throughout scanning acquisition; and a surprise story recognition test was presented to each subject after scanning. In this post-scanning test,
Right activity predicts aphasics’ comprehension

subjects were presented with 38 individual phrases and asked to indicate after each one whether they recognized it as being part of a story played to them in the scanner or not.

fMRI scanning
A Siemens 1.5 T scanner was used to acquire T₁-weighted echo-planar images with BOLD contrast. Each echo-planar image comprised 35 axial slices of 2.0 mm thickness with 1 mm inter-slice interval and 3 × 3 mm in-plane resolution. Volumes were acquired with an effective repetition time (TR) of 3.15 s/volume and the first six (dummy) volumes of each run were discarded to allow for T₁ equilibration effects. A total of 460 volume images were taken in two separate runs, each of 10 min duration. After the two functional runs, a T₁-weighted anatomical volume image was acquired for all subjects.

Behavioural analyses
All patients’ individual performance on the comprehensive battery of language tests and the recognition memory test post-scan are tabulated in Table 1. To investigate group differences, the data were entered into SPSS 10.0 (SPSS, Inc., Chicago, IL) and group means for each behavioural variable were compared using independent sample t-tests. Bivariate correlations estimated significance of Pearson correlations between the behavioural variables. Significance was set at $P < 0.05$ (two-tailed) for all analyses.

Statistical parametric mapping
Statistical parametric mapping was performed using SPM2 software (Wellcome Department of Imaging Neuroscience, London, UK), running under Matlab 6.5 (Mathworks Inc., Sherborn, MA, USA). All volumes from each subject were realigned using the first as reference and resliced with sinc interpolation. Within SPM, using Imcalc an 8 mm smoothed study specific template was created using the images from both the normals and patients to aid the accuracy of the normalization of this subject group rather than using the standard SPM template based on normal healthy young volunteers. The functional images were then spatially normalized to the study specific template (Friston et al., 1995a) with SPM2 normalization software, using non-linear-basis functions with high regularization that limits the local distortions within the image. Functional data were spatially smoothed, with a 12 mm full width at half maximum isotropic Gaussian kernel, to compensate for residual variability after spatial normalization and to permit application of Gaussian random field theory for corrected statistical inference.

First, the statistical analysis was performed in a subject-specific fashion. To remove low-frequency drifts, the data were high-pass filtered using a set of discrete cosine basis functions with a cut-off period of 128 s. Each experimental condition was modelled independently by convolving each block with a synthetic haemodynamic response function. The parameter estimates were calculated for all brain voxels using the general linear model, and contrast images comparing each story against reversed stories (i.e. the baseline) were computed (Friston et al., 1995b). These subject-specific contrast images were then entered into three separate second level ANOVAs that identified:

(i) Group differences and commonalities in story activation. Differences were identified by comparing story activation in neurologically normal subjects, patients with left temporal lobe damage and patients without left temporal lobe damage. Commonalities were identified with the conjunction of patient activation and the normal activation inclusively masked ($P < 0.001$ uncorrected) with the same contrasts.
(ii) The impact of auditory sentence comprehension on story activation. Within a single analysis, each patient group was modelled independently and story activation was regressed (in each group separately) on the auditory sentence comprehension score from the CAT. This multiple regression within ANOVA analysis enabled us to find common effects of auditory sentence comprehension and differences between patient groups. For both common and differential effects, we identified the effect of sentence comprehension in conjunction with the main effect of story activation, in order to limit the search to areas that responded positively to intelligible speech. As neurologically normal control subjects are all at ceiling on the CAT auditory sentence comprehension test, they were excluded from the analysis.

Unless otherwise indicated, we report and discuss regions that showed significant effects at $P < 0.05$ (corrected for multiple comparisons across the whole brain) with an extent threshold, for each cluster, of 5 voxels.
(iii) Activation associated with good story recognition memory. Within a single analysis, each patient group and the neurologically normal controls were modelled independently and story activation (for each subject within each group) was regressed on subsequent story recognition memory established from the surprise recognition test. This multiple regression within ANOVA analysis enabled us to find common effects of subsequent recognition memory as well as group differences. For both common and differential effects, we identified the effect of subsequent story recognition in conjunction with the main effect of story activation, in order to limit the search to areas that responded positively to the narrative speech.

Results

Behavioural data
The effect of left hemisphere lesions on language function
Although many of the patients continued to have significant difficulties on tests of speech production and verbal fluency, all patients performed above chance on tests of auditory sentence comprehension (minimal score 73% accuracy) (see Table 1).

A direct comparison of the patient groups indicated lower performance for patients with left temporal lobe damage on both auditory ($P = 0.009$, SE: 1.9) and written sentence comprehension ($P = 0.002$, SE: 2.7) tasks (see Table 2). However, there was considerable variability within groups and no clear relationship between performance on the language tests, and the site and extent of damage (see Table 1). For example, Patients 7 and 5 (WC and GH) had extensive left temporal lobe damage but still scored 28/30 and 30/30 on single word comprehension (chance = 7.5/30) and 25/32 and 23/32 on auditory sentence comprehension (chance = 8/32).

Analysis of the extent and site of the lesion was, therefore, not a good predictor of language comprehension. There were no significant correlations between age of the patients or time post-stroke, and performance on any of the language tests.
## Table 1 Patients' behavioural profiles

<table>
<thead>
<tr>
<th>Patient group</th>
<th>Patient number</th>
<th>Patient initials</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Lesion location</th>
<th>Months post-stroke</th>
<th>Auditory word comprehension</th>
<th>Auditory sentence comprehension</th>
<th>Naming</th>
<th>Written word comprehension</th>
<th>Written sentence comprehension</th>
<th>Repetition single word</th>
<th>Recognition memory post-scan</th>
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<tbody>
<tr>
<td>Temp. 1</td>
<td>1</td>
<td>GE</td>
<td>64</td>
<td>M</td>
<td>pST</td>
<td>4</td>
<td>20*</td>
<td>21*</td>
<td>4*</td>
<td>6*</td>
<td>7*</td>
<td>32</td>
<td>18</td>
</tr>
<tr>
<td>Temp. 2</td>
<td>2</td>
<td>GB</td>
<td>69</td>
<td>M</td>
<td>pST</td>
<td>7</td>
<td>30</td>
<td>27*</td>
<td>16*</td>
<td>28</td>
<td>7*</td>
<td>30</td>
<td>28*</td>
</tr>
<tr>
<td>Temp. 3</td>
<td>3</td>
<td>VW</td>
<td>48</td>
<td>M</td>
<td>pST</td>
<td>6</td>
<td>27</td>
<td>29</td>
<td>48</td>
<td>30</td>
<td>30</td>
<td>28*</td>
<td>21</td>
</tr>
<tr>
<td>Temp. 4</td>
<td>4</td>
<td>LH</td>
<td>34</td>
<td>F</td>
<td>aST</td>
<td>125</td>
<td>30</td>
<td>29</td>
<td>46</td>
<td>30</td>
<td>24</td>
<td>24</td>
<td>27</td>
</tr>
<tr>
<td>Temp. 5</td>
<td>5</td>
<td>GB</td>
<td>67</td>
<td>M</td>
<td>a + pST</td>
<td>17</td>
<td>30</td>
<td>23*</td>
<td>47</td>
<td>30</td>
<td>24</td>
<td>30</td>
<td>27</td>
</tr>
<tr>
<td>Temp. 6</td>
<td>6</td>
<td>VG</td>
<td>41</td>
<td>F</td>
<td>a + pST</td>
<td>80</td>
<td>30</td>
<td>16*</td>
<td>18*</td>
<td>27*</td>
<td>9*</td>
<td>0*</td>
<td>12</td>
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<tr>
<td>Temp. 7</td>
<td>7</td>
<td>WC</td>
<td>75</td>
<td>M</td>
<td>a + pST</td>
<td>36</td>
<td>28</td>
<td>25*</td>
<td>37*</td>
<td>24*</td>
<td>29</td>
<td>32</td>
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<tr>
<td>Temp. 8</td>
<td>8</td>
<td>DD</td>
<td>64</td>
<td>M</td>
<td>a + pST</td>
<td>51</td>
<td>26</td>
<td>16*</td>
<td>28*</td>
<td>22*</td>
<td>12*</td>
<td>25</td>
<td>30</td>
</tr>
<tr>
<td>Control 9</td>
<td>9</td>
<td>AK</td>
<td>66</td>
<td>M</td>
<td>Frontal</td>
<td>86</td>
<td>28</td>
<td>26*</td>
<td>48</td>
<td>28</td>
<td>29</td>
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<td>Control 10</td>
<td>10</td>
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<td>63</td>
<td>F</td>
<td>Frontal</td>
<td>42</td>
<td>30</td>
<td>32</td>
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<tr>
<td>Control 11</td>
<td>11</td>
<td>DR</td>
<td>75</td>
<td>F</td>
<td>Frontal</td>
<td>37</td>
<td>29</td>
<td>30</td>
<td>31*</td>
<td>27*</td>
<td>30</td>
<td>15*</td>
<td>16</td>
</tr>
<tr>
<td>Control 12</td>
<td>12</td>
<td>DC</td>
<td>69</td>
<td>M</td>
<td>Parietal</td>
<td>78</td>
<td>30</td>
<td>24*</td>
<td>45</td>
<td>28</td>
<td>28</td>
<td>24*</td>
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<tr>
<td>Control 13</td>
<td>13</td>
<td>PB</td>
<td>71</td>
<td>M</td>
<td>Parietal</td>
<td>95</td>
<td>26</td>
<td>30</td>
<td>48</td>
<td>30</td>
<td>29</td>
<td>29*</td>
<td>31</td>
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<tr>
<td>Control 14</td>
<td>14</td>
<td>RJ</td>
<td>59</td>
<td>M</td>
<td>Parietal</td>
<td>38</td>
<td>30</td>
<td>32</td>
<td>48</td>
<td>30</td>
<td>32</td>
<td>32</td>
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<tr>
<td>Control 15</td>
<td>15</td>
<td>SB</td>
<td>59</td>
<td>F</td>
<td>Subcortical</td>
<td>20</td>
<td>30</td>
<td>32</td>
<td>48</td>
<td>30</td>
<td>32</td>
<td>32</td>
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<tr>
<td>Control 16</td>
<td>16</td>
<td>TS</td>
<td>66</td>
<td>M</td>
<td>Subcortical</td>
<td>13</td>
<td>28</td>
<td>48</td>
<td>29</td>
<td>31</td>
<td>32</td>
<td>32</td>
<td>33</td>
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<tr>
<td>Control 17</td>
<td>17</td>
<td>AM</td>
<td>66</td>
<td>M</td>
<td>Subcortical</td>
<td>24</td>
<td>28</td>
<td>48</td>
<td>29</td>
<td>31</td>
<td>32</td>
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<tr>
<td>Non-aphasic</td>
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<td></td>
<td></td>
<td></td>
<td>Mean = 29.15</td>
<td>Mean = 30.17</td>
<td>Mean = 46.37</td>
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<td>Mean = 31.73</td>
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<td>performance</td>
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<td>SD = 1.35</td>
<td>SD = 1.85</td>
<td>SD = 1.6</td>
<td>SD = 0.79</td>
<td>SD = 2.5</td>
<td>SD = 0.67</td>
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<td>Cut off = 25</td>
<td>Cut off = 27</td>
<td>Cut off = 43</td>
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<td>(max = 30)</td>
<td>(max = 32)</td>
<td>(max = 38)</td>
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</tr>
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</table>

The individual scores for each patient on the language subtests of the comprehensive aphasia test battery (CAT) and the surprise story recognition memory test post-fMRI-scanning. The cut-off where mentioned is the score that at least 95% of normal subjects exceed; as a result the scores that are below the cut-off represent aphasic performance. Individual test scores highlighted in bold italic typeface with an asterisk (*) represent aphasic performance. aST = anterolateral superior temporal lobe; pST = posterior superior temporal lobe; a + pST = damage to both anterior and posterior superior temporal cortices; Recognition memory post-scan = surprise recognition memory test post-scanning; Temp. = patients with left temporal lobe damage; n/c = not collected.
Table 2 Group language comprehension data

<table>
<thead>
<tr>
<th>Patient group</th>
<th>Auditory word comprehension</th>
<th>Auditory sentence comprehension</th>
<th>Written word comprehension</th>
<th>Written sentence comprehension</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SE</td>
<td>Mean</td>
<td>SE</td>
</tr>
<tr>
<td>Temporal (n = 8)</td>
<td>25.9</td>
<td>1.8</td>
<td>23.5*</td>
<td>1.7*</td>
</tr>
<tr>
<td>Control (n = 9)</td>
<td>28.8</td>
<td>0.5</td>
<td>29.2*</td>
<td>0.9*</td>
</tr>
<tr>
<td>Maximum score</td>
<td>30</td>
<td>32</td>
<td>30</td>
<td>32</td>
</tr>
</tbody>
</table>

The table displays mean scores and standard error of the patient groups with left superior temporal lobe damage (Temporal patients) and those without damage to the left temporal lobe (Control patients) on auditory and written single word and sentence comprehension testing. We performed independent t-tests for each variable, and significance was set at $P < 0.05$ (two-tailed). The Control patients were as a group significantly better on tests of auditory and written sentence comprehension than the Temporal patients ($P = 0.009$, SE: 1.9; $P = 0.002$, SE: 2.7, respectively), these scores are highlighted in bold italic typeface with an asterisk (*). There was no difference between the patient groups on tests of single word comprehension, with both groups’ mean performance >80% accuracy on these tests.

Recognition memory performance on the surprise story recognition test

The Normal group’s mean performance on the surprise recognition test post-scan (mean $= 32.4/38$; SE 0.8) was significantly higher ($P < 0.0001$) than the group of 16 patients (mean $= 24.4/38$; SE 1.7). Note: Patient 2, GB from the group of patients with temporal lobe damage did not participate in the surprise recognition test post-scan. The mean performance of the seven patients with temporal lobe damage was lower (20.9/38; SE 2.3) than the nine patients without temporal lobe damage (27.1/38; SE 2.1) but this mean difference did not quite reach significance ($P = 0.06$) due to the wide variability within each group.

There were no significant correlations between recognition memory scores and

(i) the age of the patients and normal subjects, ($P = 0.45$, $n = 34$),
(ii) the time post-infarction in the patients ($P = 0.27$, $n = 16$) or
(iii) sentence comprehension ($P = 0.41$, $n = 16$).

However, patient performance on the post-scan recognition test was significantly correlated with auditory single word comprehension ($P = 0.04$, $n = 16$).

Functional imaging data

Activation for listening to stories relative to baseline

In the Normal group, listening to stories relative to baseline, (reversed stories) activated lateral left temporal cortex, with five main peaks extending from posterior lateral to anterolateral temporal lobe centred on the superior temporal sulcus (STS), and at the junction of the superior temporal gyrus (STG) with the inferior parietal lobe (IPL). Activation in the right superior temporal cortex did not extend into the STG/IPL junction, with four main peaks in the lateral temporal lobe. Additional activations were observed bilaterally in the ventral temporal lobe, within the occipito-temporal sulcus separating the inferior temporal gyrus (ITG) and fusiform gyrus (FG), and in five regions of the left frontal and bilateral motor cortices (Fig. 2, a; Table 3, a).

The Control patients, i.e. those without temporal lobe damage, activated the same system as the Normal group, apart from two regions in bilateral motor cortices that the Normal group activated significantly more (Fig. 2, b1 and b2; Table 3, b). The controls activated any region significantly more than the group of 16 patients (mean $= 23.5/38$; SE 0.8) was significantly lower (20.9/38; SE 2.3) than the nine patients without temporal lobe damage (27.1/38; SE 2.1) but this mean difference did not quite reach significance ($P = 0.06$) due to the wide variability within each group.

Recognition memory performance on the surprise story recognition test

The Normal group’s mean performance on the surprise recognition test post-scan (mean $= 32.4/38$; SE 0.8) was significantly higher ($P < 0.0001$) than the group of 16 patients (mean $= 24.4/38$; SE 1.7). Note: Patient 2, GB from the group of patients with temporal lobe damage did not participate in the surprise recognition test post-scan. The mean performance of the seven patients with temporal lobe damage was lower (20.9/38; SE 2.3) than the nine patients without temporal lobe damage (27.1/38; SE 2.1) but this mean difference did not quite reach significance ($P = 0.06$) due to the wide variability within each group.

There were no significant correlations between recognition memory scores and

(i) the age of the patients and normal subjects, ($P = 0.45$, $n = 34$),
(ii) the time post-infarction in the patients ($P = 0.27$, $n = 16$) or
(iii) sentence comprehension ($P = 0.41$, $n = 16$).

However, patient performance on the post-scan recognition test was significantly correlated with auditory single word comprehension ($P = 0.04$, $n = 16$).

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However, patient performance on the post-scan recognition test was significantly correlated with auditory single word comprehension ($P = 0.04$, $n = 16$).
Positive correlations between activation and performance on the surprise story recognition test post-scan, common to controls and patients, were observed in the left prefrontal cortex ($x = -42, y = +8, z = +20; Z = 5.32$) and right cerebellum ($x = +16, y = -80, z = -40; Z = 5.17$) (Fig. 4). There were no significant differences between subject groups. There were no significant negative correlations.

**Discussion**

Clinical observations over many decades have demonstrated that verbal comprehension, both lexical and sentential semantics, is largely lateralized to the left perisylvian cortices in most individuals. Nevertheless, speech comprehension abilities can be very well preserved following left hemisphere lesions. We investigated whether good speech comprehension was related to left or right hemisphere activation. One group of patients had left temporal lobe damage (Temporal patients); another had left hemisphere damage that spared the left temporal lobe (Control patients). We found that auditory sentence comprehension ability was positively correlated with (i) bilateral temporal activation when the temporal lobes were spared; but (ii) only a right anterior temporal region when the left temporal lobe was damaged. Below, we discuss the implications of our results for understanding speech comprehension following aphasic stroke.

**Lesion site and speech comprehension deficits**

Auditory sentence comprehension was significantly more impaired in the group of patients with temporal damage compared with the group of patients without temporal damage. However, within the group of patients with temporal damage, there was variability in both the lesion site and the speech comprehension scores. On auditory single word comprehension tasks, all patients scored significantly above average, but the severity of the impairment varied widely. This suggests that the extent of damage to the left hemisphere, particularly in the anterior temporal region, is a critical factor in determining speech comprehension. The findings highlight the importance of individual variation in identifying the specific regions involved in speech processing and suggest that rehabilitation strategies may need to be tailored to the individual patient.
Table 3: Regional activation for stories > baseline

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<tr>
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<th>(b) Control pts</th>
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The first three columns show the coordinates and Z-scores for the condition effect (St > RSt) in (a) the Normal group (visualized in Fig. 2a), (b) the patient Control group (visualized in Fig. 2b), and (c) the patients with temporal lobe damage (visualized in Fig. 2c). The next three columns (b', c' and c') show the coordinates and Z-scores for the condition effect (St > RSt) where the Normal group activated significantly more than Control patients (b') and Temporal patients (c'), and where the Control patients activated significantly more than the Temporal patients (c'), visualized in Fig. 2 (b', c' and c'), respectively. Coordinates are given as x, y and z according to the atlas of Talairach and Tournoux (1988). Normal = normal subject group; Control pts = patients whose lesions spared the left temporal cortices; Temporal pts = patients whose lesions involved the left superior temporal cortices. Significant effects at P < 0.05 (corrected for multiple comparisons across the whole brain) are shown in bold typeface.
chance (8/32 = 25%) with the lowest score (16/32 = 50%) coming from Patient 6 (MG) who had both anterior and posterior superior temporal lobe damage. On the auditory sentence comprehension task, again, all patients scored above chance with the lowest score (16/32) coming from Patient 8 (DD) following extensive left superior temporal damage. The remainder of the patients made relatively few errors on our word and sentence comprehension tests even when left temporal lobe damage was extensive. For example, sentence comprehension remained excellent following damage to either posterior (Patient 1: GE) or anterior (Patient 4: LH) temporal cortices as well as in Patient 7 (WC) who had lost virtually all his anterior and posterior superior temporal cortices. These observations do not reveal any appreciable relationship between site of damage and speech comprehension ability. Instead, they suggest that, when the left temporal lobe is damaged, areas outside the left temporal lobe may be sustaining speech comprehension.

**Narrative speech activation in control subjects**

When listening to narrative speech compared with reversed speech, our neurologically Normal subjects and Control patients activated bilateral posterior and anterior temporal regions and the left inferior frontal and premotor cortices. The role of the right temporal activation in speech comprehension remains unclear. As the reversal of speech destroys normal stress patterns, intonation, prosody and many features that signal unique voice identity, the bilateral anterior temporal signals may not be attributed exclusively to linguistic processing of speech. Many authors have associated right temporal lobe activation in normal subjects predominantly with processing of the non-verbal components (intonation, prosody, voice identity, etc.) of speech (Buchanan et al., 2000; Belin et al., 2002; Mitchell et al., 2003; Wildgruber et al., 2004). This lateralization of different functions is the usual clinical view. However, there is

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**Fig. 3** Location of temporal damage and abnormal activation in patients GE and LH. Sagittal (x = −58) and coronal (y = −14 and −50) slices are from the MRI scans of each patient after spatial normalization and indicate the extent of the lesions and the validity of the normalization. Arrows highlight the areas of temporal damage. Reduced activation (Stories > baseline) compared with neurologically Normal subjects are shown below in yellow on the same MRI slices (Normal > GE/LH).
good evidence for participation of the right hemisphere in lexical semantic functions in normal subjects (Ellis and Shepherd, 1974; Hines, 1975; Day, 1977, 1979; Young and Ellis, 1985) and in patients with semantic dementia who are more impaired in lexical semantic processing when anterior and ventral temporal atrophy is bilateral (Chan et al., 2001; Galton et al., 2001; Lambon Ralph et al., 2001).

The effect of left temporal lobe damage on narrative speech activation

As expected, left temporal lobe damage significantly reduced left superior temporal activation compared with the neurologically Normal subjects and the Control patients. More remarkably, analyses of individual patients revealed that left anterior temporal activation was significantly reduced even when this region was spared in patients with posterior temporal damage (see Patient GE in Fig. 3). Likewise, left posterior temporal activation was significantly reduced even when this region was spared in patients with anterior temporal damage (see Patient GH in Fig. 3). The results from these two patients whose lesions involved either posterior or anterior superior temporal cortices suggest a mutual dependence between left anterior and posterior superior temporal lobe activation when subjects are listening to narrative speech.

The consequence of left temporal lobe damage was also reflected in the observation that activation for narrative speech was strongly right lateralized (see Fig. 2, c). This apparent laterality shift (left dominant activation in Normals...
and Control patients right lateralized activation following left temporal lobe damage), did not reflect compensatory right hemisphere activation because examination of activation in right hemisphere regions revealed that the patients with left temporal lobe damage activated within the normal range. This cannot be attributed to abnormalities in the BOLD response, because the structural integrity of the right hemisphere was not compromised in any of the patients. The results, therefore, suggest that right superior temporal activation was dissociated from that in the left superior temporal lobe. This contrasts with previous observations that have shown how damage to the left frontal operculum can enhance activation in the right frontal operculum during speech production tasks (Rosen et al., 2000; Blank et al., 2003).

The effect of auditory sentence comprehension score on narrative speech activation

Right lateralized activation following left temporal lobe damage suggests that narrative speech comprehension might be sustained by the right hemisphere. Moreover, in some patients (e.g. Patient 7, WC), who had little remaining left temporal cortex, right hemisphere activation appeared to be sufficient for good auditory single word and sentence comprehension. However, there are well-recognized difficulties when drawing such inferences. First, the absence of left hemisphere activation may be due to a lack of sensitivity in damaged cortex (i.e. it could be a null result). Second, there is always a trade-off between engaging patients in an executive paradigm that requires a motor response versus engaging patients in more naturalistic speech comprehension paradigms that do not involve a motor response. The disadvantage of the former is that performance on the decision-making aspects of the task cannot be controlled across subjects. The disadvantage of the latter is that we do not have a measure of how much of our narratives were understood while the subjects were in the scanner.

Our conclusion that the right hemisphere contributes to speech comprehension is, therefore, dependent on our observation that right anterior temporal activation could be predicted not by lesion site but on the basis of auditory sentence comprehension ability measured outside the scanner (see Fig. 4). This correlational approach is becoming increasingly popular in functional imaging studies of patient populations. For example, Shaywitz et al. (2003) have shown that left occipito-temporal activation increases with reading ability in neurologically normal and dyslexic children; while Noppeney et al. (2005) have shown that right hemisphere activation correlates with reading ability following left anterior temporal lobe resection. Perhaps surprisingly, the aphasia literature has not previously reported correlations between right hemisphere activation and speech comprehension and production abilities measured outside the scanner. For example, neither Rosen et al. (2000) nor Blank et al. (2003) found any relationship between right frontal activation and speech production measures. Our findings, in the context of speech comprehension, therefore, support previous suggestions that the right hemisphere might be able to sustain speech comprehension better than speech production (Zaidel, 1976; Zaidel et al., 2000).

In patients with left temporal lobe damage, auditory sentence comprehension ability was only predicted by activation in a right lateral superior temporal region, anterior to primary auditory cortex. Activation in the same right anterior temporal area also predicted auditory sentence comprehension ability in patients with lesions outside the left temporal lobe, but in addition, these patients also showed a correlation between left posterior superior temporal activation and auditory sentence comprehension performance. One interpretation of these findings could be that auditory sentence comprehension relies on a bilaterally-distributed system. This is demonstrated in terms of bilateral activations in Normals and Control patients. The reduced comprehension performance of the group of patients with left temporal lobe damage would, therefore, be reflected in reduced, total temporal lobe activation—in this case the remaining activation of the right temporal region, with sentence comprehension performance predicted by the sum of left and right temporal activation.

However, this does not explain our data. In the patients with left temporal lobe damage there was no negative correlation between the remaining activation in the left temporal lobe with the degree of right temporal activation. Patients with left temporal lobe damage and good sentence comprehension did not have higher right temporal responses than the Control patients (who had both left and right activation), and patients with bilateral temporal activation (e.g. Patients 16 and 17) did not have better sentence comprehension than patients with only right temporal activation (e.g. Patients 3 and 4). An alternative interpretation is that, in this group of chronic patients with left temporal damage, the response in the right temporal lobe is independent of that in the damaged left temporal lobe. In other words, the results suggest that in the chronic phase post-stroke the effect in the right anterior temporal lobe is independent of that in the left posterior temporal lobe: the Control patients showed both effects while patients with left temporal lobe damage only showed the effect in the right hemisphere.

The anterior location of the right hemisphere correlation is also interesting. The literature on aphasic stroke patients emphasizes the importance of the posterior superior temporal and inferior parietal cortices in speech comprehension (Alexander et al., 1989) as do functional imaging studies of aphasia following left hemisphere lesions (Leff et al., 2002; Musso et al., 1999). However, these studies were not able to segregate speech perception from auditory comprehension processes. In contrast, we focused on auditory sentence comprehension in the regression analysis, which highlighted the role of the right anterior superior temporal cortex (and the left
posterior superior temporal cortex when it was spared in Control patients). The anterior location of the right hemisphere effect is consistent with studies of semantic dementia that show progressive and ultimately profound loss of speech comprehension following bilateral anterior and ventral temporal lobe atrophy (Mummery et al., 1999; Chan et al., 2001; Galton et al., 2001).

The effect of story recognition memory on narrative speech activation

In distinct contrast to the correlation of auditory sentence comprehension with narrative speech activation, performance on the surprise story recognition test post-fMRI-scanning predicted left inferior frontal and right cerebellar activation (see Fig. 5). This was observed across all subject groups, Normal and both patients groups, with no significant differences between groups, even though error rates were higher in the patients than the normal controls. The implication of this double dissociation in the effects of auditory sentence comprehension and story recognition memory is that left frontal and left temporal activations are dissociable. For example, prefrontal and motor activation may be associated with working memory and other executive functions not directly related to normal, on-line speech processing (Crinion et al., 2003). Indeed, left frontal lesions do not typically impair simple narrative speech comprehension; and the Control patients included in the present study, three of whom had left frontal lesions, performed as a group significantly better on language comprehension tests than the patients with left temporal lobe lesions. Together the dissociation in the effects of speech comprehension and story recognition memory suggest that left inferior frontal activation during speech processing may be associated with 'top-down' rather than 'bottom-up' processing.

Fig. 5 Activation increases with story recognition. Regional activation (stories > baseline) increases with performance on the surprise story recognition memory test post-scan for all subject groups are shown in colour on models of the brain from SPM2 (P < 0.05 corrected). Two main peaks were observed, in the right cerebellum (x = 16, y = −80, z = −40) and in the left prefrontal cortex (x = −42, y = 8, z = 20). The x-axes in the graphs indicate the individual subjects’ scores on the surprise story recognition memory test post-scan; while the y-axes indicate the mean centred activation for stories more than baseline. No significant difference in the regression slopes for Normals (green stars), Control patients (black triangles) or patients with temporal lobe damage (red circles) was observed in either region. Green slopes illustrate Normal regression slopes (right Rsq = 0.32; left Rsq = 0.28). Black slopes illustrate patient regression slopes, Control and Temporal lobe damaged patients combined as one group (right Rsq = 0.26; left Rsq = 0.26).
Implications for long-term language functioning after aphasic stroke

Our results indicate that the right superior temporal cortex does play a role in auditory sentence comprehension following aphasic stroke, at least, in the chronic phase. The fact that the patients’ deficits with speech production were far more enduring (see Table 1) is consistent with the model of right hemisphere language proposed by Zaidel (1976) in which the right hemisphere should selectively contribute to the recovery of language comprehension but not to the recovery of language production. Future studies can, therefore, investigate whether right anterior temporal activation changes longitudinally over time during the acute or chronic stage of recovery. For example, it may be the case that recovery of speech comprehension depends on the pre-morbid function of the right anterior temporal lobe. Alternatively, the function of the right anterior temporal lobe may depend on experience/therapy.

It has been proposed that the left posterior STS and supramarginal plane provide a system whereby speech sequences are briefly held in memory and can be repeated by projection forwards of the encoded speech sequences to the caudal frontal lobe via the superior longitudinal fasciculus (Hickok et al., 2001). Thus, the normal left lateralization of speech comprehension systems may be more a reflection of left superior temporal connections with speech production faculties within the left frontal lobe rather than purely auditory speech processing systems per se. To fully dissociate these possibilities will require additional behavioural data from patients with right superior temporal lesions. It will also be important to investigate whether successful speech comprehension rehabilitative techniques are associated with increased responsiveness of the right anterolateral superior temporal lobe.

Conclusions

In this study, we have shown that:

(i) Auditory speech comprehension both at the single word and sentence level can be relatively well preserved following left anterior and posterior superior temporal cortical damage. This suggests that alternative neuronal mechanisms are available to support speech comprehension following aphasic stroke.

(ii) Lesions involving the left superior temporal cortices result in significantly less activation not only in the area of lesioned cortex but also ipsilaterally along the length of the superior temporal lobe even in structurally intact cortices. This suggests that, for processing narrative speech, left posterior and anterior superior temporal cortices respond together.

(iii) Auditory sentence comprehension abilities, as measured outside the scanner, predicted the level of activation in the left posterior and right anterior superior temporal cortices. When the left superior temporal region was damaged, the correlation with sentence comprehension was only observed in the right anterior superior temporal cortex. This suggests that right superior temporal responses are dissociated from those in the left superior temporal lobe.

These results have implications for the development of rehabilitative and pharmacological approaches to the treatment of aphasic stroke. Once cerebral infarction is established, rehabilitative techniques, whether behavioural or pharmacological, must be directed at intact cortices. Following extensive left temporal lobe infarction, we are proposing that right anterolateral, superior temporal cortices, may be a target for such therapy.

Acknowledgements

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Right activity predicts aphasics’ comprehension


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