Regions of Dysfunctional Neural Tissue Associated with Impairment of the Graphemic Buffer in Spelling

Summary

The graphemic buffer is a working memory component of the spelling system that temporarily stores the sequence of graphemes while each grapheme is written or spelled aloud. We evaluated 331 patients with left hemisphere stroke on oral and written spelling to dictation, and written picture naming tasks and MRI including within 48 hours of stroke onset. Spelling performance was examined to identify presence or absence of graphemic buffer deficit. A voxel-wise chi-square map revealed that graphemic buffer deficits were associated with ischemia in pre- and post-central gyri, posterior inferior frontal and lateral occipital cortex.

Spelling is a complex process, requiring a number of cognitive processes and representations to accurately spell familiar and unfamiliar words. Evidence for distinct processes and representations that can be selectively impaired has come from detailed analysis of spelling performance by patients with focal brain damage whose pattern of performance across tasks can be explained by assuming damage to a single component [1-6] Additional evidence comes from computational models of spelling that can be disrupted at specific levels of processing and replicate (more or less) the patterns of performance of patients with neurological impairment [7]. Such studies have provided evidence that spelling irregular familiar words from dictation requires at least the following processing components: auditory recognition of the word to be spelled, comprehension of the word, access to the orthographic representation (stored spelling of the word), holding the word in a short-term "graphemic buffer" while the word is spelled aloud (which also requires access to the letter names that represent each grapheme, as well as motor speech processes) or while the word is written (which also requires converting the abstract grapheme to a specific letter shape with a particular case and font, as well as motor programs for writing the letter). Spelling an unfamiliar word (e.g. pseudoword or surname, such as Obama) from dictation requires holding the phonological representation in a "phonological buffer" while each phoneme is converted to an appropriate grapheme (e.g. [ou] -> O) and holding the sequence of graphemes in the "graphemic buffer" while each letter is named (for oral spelling) or written (for written spelling). Note that the single common component to oral and written spelling of familiar and unfamiliar words is the graphemic buffer.

Evidence for the role of the graphemic buffer in spelling has come from detailed single case studies of patterns of performance on various spelling tasks by patients with focal brain injury. Caramazza and colleagues [1] proposed that selective damage to the graphemic buffer should result in nearly identical spelling errors in all spelling tasks (oral and written spelling from dictation, written naming, delayed copy transcoding from upper to lower case or vice versa) because it is a component of each of these tasks. Furthermore, they proposed that spelling accuracy should not be affected by word frequency, familiarity, grammatical word class, orthographic regularity, or concreteness, since all words need to be held in the buffer regardless of these parameters. However, spelling accuracy should be affected by word length, because longer words need to be held in the buffer for a longer period of time. Furthermore, errors should include deletions, insertions, transpositions, and substitutions of letters, resulting in phonologically implausible nonword errors (e.g. pencil spelled pedol rather than spelled plausibly, such as pensal), because errors result from impaired short-term storage of the correctly spelled word. Several patients with performance that conforms to this pattern of errors have been reported.

The patients reported to have selective damage to the graphemic buffer have had a variety of lesions, usually large infarcts in the distribution of the middle cerebral artery [1-5]. Many of these patients initially had other spelling deficits, but had residual selective damage to the graphemic buffer months or years after brain damage, after other regions of the brain had assumed some functions of the damaged part. Many, but not all, have had damage in the inferior parietal cortex, an area that has been implicated in short-term memory. Other lesions have involved frontal cortex (another region often associated with short-term memory function) and occipital cortex (an area that is activated in visual memory). The graphemic buffer might require a network of brain regions that support short-term storage of a string of abstract letter shapes – e.g. areas critical for working memory executive system and areas critical for a visual-spatial scratchpad to hold representations with spatial extent. The purpose of this study was to identify areas of the brain critical for function of the graphemic buffer, by identifying voxels where infarct or hypoperfusion (tissue dysfunction caused by inadequate blood flow) is associated with impaired graphemic buffer function in acute stroke – before reorganization, recovery, or therapy.

Methods

We studied spelling of words and pseudowords in a series of right-handed patients who met the following inclusion criteria: (1) able to complete testing and MRI within 48 hours acute ischemic stroke; (2) able to provide informed consent or identify a family member to do so); (3) at least 10th grade education; (4) native speaker of English. Patients were also excluded if they were sedated or had premorbid difficulty spelling by self or family report.

Language Testing

Patients were administered a battery of lexical tests described previously [8]. Here we focus on tests or oral and written spelling of 34 words and 26 pseudowords to dictation. We categorized performance of all patients who completed testing as having (1) intact graphemic buffer; (2) impaired graphemic buffer; or (3) indeterminate. See Table 1 for criteria.

Imaging

All patients underwent MRI, including diffusion-weighted imaging (DWI), which is sensitive to infarct within minutes from onset, and perfusion-weighted imaging (PWI), which shows areas of hypoperfusion that correspond to dysfunctional tissue [8,9]. Areas of ischemia (on DWI) and hypoperfusion (on PWI) were mapped onto the MNI atlas as a single region of dysfunctional tissue for each patient, using MRICron [10]. With this program we then generated probability maps showing voxels most associated with impairment in the graphemic buffer.

Results

A total of 331 patients were able to complete spelling tests; 21 met criteria for impairment at the level of the graphemic buffer; 48 met criteria for intact graphemic buffer. Remaining patients met neither criteria, so their performance was considered indeterminate (and were excluded from the voxel based analysis). Figure 1 shows voxels where ischemia was associated with the presence of impaired graphemic buffer. Disrupted processing at the level of the graphemic buffer was associated with ischemia in pre- and post-central gyri and subcortical white matter underlying prefrontal cortex, using a False Discovery Rate (FDR) correction for multiple comparisons at $p < 0.01$. Tissue dysfunction in voxels in posterior inferior frontal and lateral occipital gyri were also associated with impaired graphemic buffer, but less consistently (FDR $p < 0.05$).

Discussion

Our results provide a voxel-based analysis of cortical regions associated with impairment of the graphemic buffer. Not surprisingly, results revealed that damage or dysfunction in any one of several regions can result in disruption of spelling at the level of the graphemic buffer, indicating a network of brain regions is essential to maintaining the sequence of graphemes while a word is spelled. Results complement functional imaging studies that show activation associated with verbal working memory tasks in these areas in prefrontal cortex and parietal cortex [11-16] and visual working memory in occipital cortex [17].

Limitations of the study include the fact that that we were not able to complete lengthy tests to carry out detailed analyses of spelling to rule out some influence of lexical or sublexical processes (e.g. as indicated by an effect of orthographic regularity or concreteness). Therefore, some of our patients may have had additional deficits in the spelling process. However, we believe our criteria at least suggest that impairment of the graphemic buffer contributed to their spelling.

Table 1.

Criteria for Impairment at the Level of the Graphemic Buffer

1) Majority (>75%) of errors were phonologically implausible nonwords;
2) No significant difference (by chi-square) between spelling tasks (oral and written spelling to dictation, written naming) or stimuli (words versus pseudowords), comparing subset of items matched in length
3) total error rates on long words (5+ letters) at least 10% greater than short (3-4 letters) words, coupled with an average error rate per letter in the word that was greater for long than short words (number of incorrect or omitted letters, divided by the number of letters in the target

Criterion for No Impairment at the Level of the Graphemic Buffer:

1) >75% real word errors (visually similar words, semantic errors, and/or morphological errors); or,
2) >75% phonologically plausible errors or
3) normal performance (<10% total errors, based on norms for our stimuli) in spelling words or nonwords.

References

1. Caramazza A, Miceli G, Villa G, Romani C. The role of the graphemic buffer in spelling: Evidence from a case of acquired dysgraphia. Cognition 1987;26: 59-85.

2. Posteraro L, Zinelli P, Mazzucchi A. Selective impairment of the graphemic buffer in acquired dysgraphia: A case study. Brain Lang 1988;35: 274-286.

3. Schiller NO, Greenhall JA, Shelton JR, Caramazza A. Serial order effects in spelling errors: Evidence from two dysgraphic patients. Neurocase 2001;7: 1-14.

4. Bormann T, Wallesch C-W, Blanken G. "Fragment errors" in deep dysgraphia: Further support for a lexical hypothesis. Cogn Neuropsychol 2008;25: 745-764.

5. Hillis AE, Caramazza A. The graphemic buffer and attentional mechanisms. Brain Lang 1989;36: 208-235.

6. Tainturier MJ, Rapp BC. Complex graphemes as functional spelling units: Evidence from acquired dysgraphia. Neurocase 2004;10: 122-131.

7. Glasspool DW, Houghton G. Serial order and consonant-vowel structure in a graphemic output buffer model. Brain Lang 2005;94: 304-330.

8. Hillis, A.E., Kane, A., Tuffiash, E., Ulatowski, J.A., Barker, P., Beauchamp, N., Wityk, R. (2002). Reperfusion of specific brain regions by raising blood pressure restores selective language functions in subacute stroke. Brain and Language, 79, 495-510.

9. Hillis AE, Kane A, Tuffiash E, et al. Neural substrates of the cognitive processes underlying spelling: Evidence from MR diffusion and perfusion imaging. Aphasiology 2002;16: 425-438.

10. Rorden C, Karnath H-O, Bonilha L. Improving lesion-symptom mapping. J Cogn Neurosci 2007;19: 1081-1088.

11. McNab F, Leroux G, Strand F, et al. Common and unique components of inhibition and working memory: An fMRI, within-subjects investigation. Neuropsychologia 2008;46: 668-2682.

12. Tsukiura T, Fujii T, Takahashi T, et al. Neuroanatomical discrimination between manipulating and maintaining processes involved in verbal working memory; a functional MRI study. Cogn Brain Res 2001;11: 13-21.

13. Crottaz-Herbette S, Anagnoson RT, Menon V. Modality effects in verbal working memory:Differential prefrontal and parietal responses to auditory and visual stimuli. Neuroimage 2004;21: 340-351.

14. Jansma JM, Ramsey NF, Slagter HA, Kahn RS. Functional anatomical correlates of controlled and automatic processing. J Cogn Neurosci 2001;13: 730-743.

15. Wendelken C, Bunge SA, Carter CS. Maintaining structured information: An investigation into functions of parietal and lateral prefrontal cortices. Neuropsychologia 2008;46: 665-678.

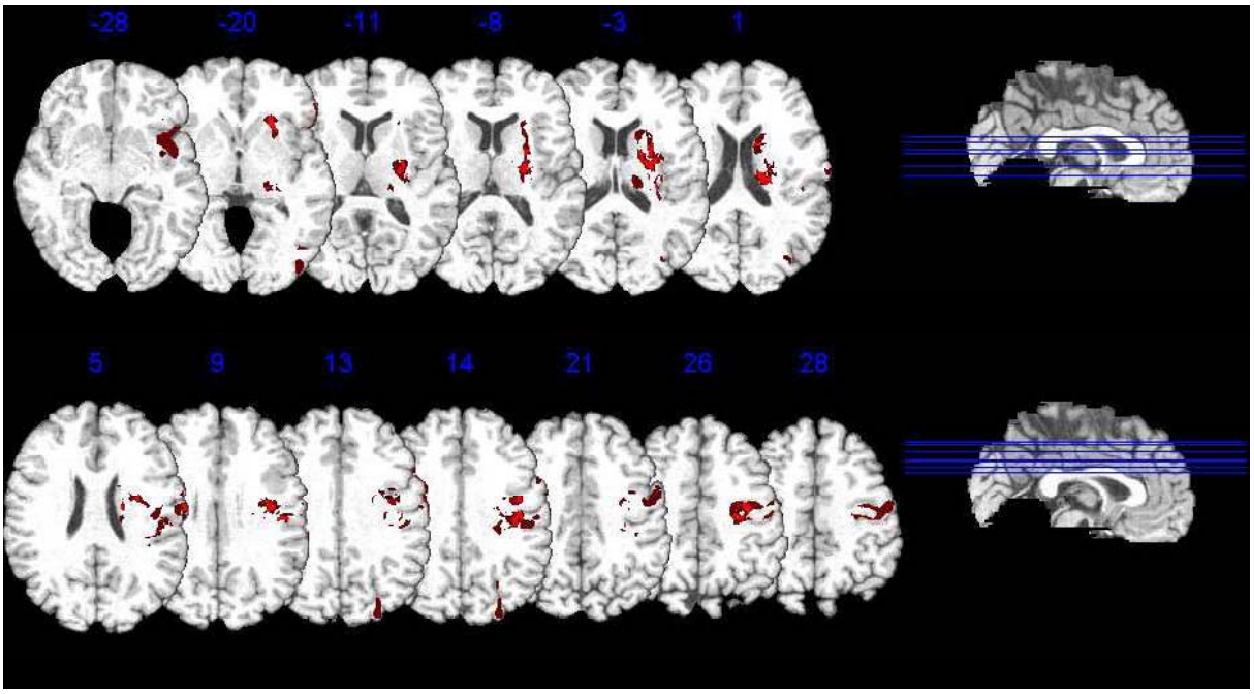
16. Jonides J, Schumacher EH, Smith EE, et al. The role of parietal cortex in verbal working memory. J Neurosci 1998;18: 5026-5034.

17. Xu Y, Chun MM. Dissociable neural mechanisms supporting visual short-term memory for objects. Nature 2006;440: 91-95.

Figure Legend

Figure 1.

Voxels of the MNI atlas where tissue dysfunction (hypoperfusion and/or dense ischemia or infarct, as defined bright on DWI or >4 sec delay in time to peak arrival of contrast relative to the homologous voxels in the right hemisphere on PWI) was associated with a graphemic buffer deficit. Images are presented in radiological convention (left hemisphere on right). Areas in bright red indicate $p < 0.01$ FDR ($Z > 2.34$), while areas in dark red indicate $p < 0.05$ FDR ($Z > 1.73$).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