

Phonological Agraphia After Superior Temporal Gyrus Infarction

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Background: Phonological agraphia refers to a condition in which the ability to write nonwords to dictation is impaired, while writing words to dictation is preserved, as is oral repetition of the words and nonwords. This condition has been regarded as reflecting a disconnection within the phonological writing system, and previous neurolinguistic correlations suggested that the anterior-inferior supramarginal gyrus was a crucial link within the system.

Setting: A neurology department of a university hospital.

Patient: A 51-year-old right-handed man presented with speech disturbances. On initial evaluation of his language, his deficit was consistent with that of conduction aphasia, which improved rapidly to an apparently

normal level. A subsequent detailed examination of oral and written repetition of words and nonwords revealed a rather selective form of phonological agraphia. A magnetic resonance imaging scan of his brain showed a focal ischemic lesion at the left posterior superior temporal gyrus and at the underlying white matter.

Conclusions: In contrast to most previously described patients, this patient showed a selective impairment of phonological agraphia in association with a focal infarction restricted to the left posterior superior temporal gyrus, suggesting that this region of the brain is an important node within a wider network of areas that subserve the phonological route for writing.

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PHONOLOGICAL AGRAPHIA is a spelling disorder characterized by the inability to write nonwords to dictation, whereas the ability to write words to dictation and to orally repeat words and nonwords is intact.¹ This condition is thought to be caused by a selective impairment in associating input phonological representations with output orthographic representations and, thereby, a forced reliance on lexical memory for accessing orthographic word-form representations.² Because persons with phonological agraphia rely on lexical information in writing, they usually show errors in writing affixes and nonwords, which presumably are not stored in the mental lexicon, and in writing low-frequency words, for which the access to the lexicon is less efficient.²

Classically, the anterior-inferior part of the left supramarginal gyrus has been pointed to as the lesion site underlying phonological agraphia.³ However, there is considerable variability in the clinical manifestations among described patients.^{1,3-8} Some patients had additional difficulties in oral repetition of nonwords,³ and others showed serious problems in writing real words and nonwords to dictation.^{3,4,7} The patient described by Bud and

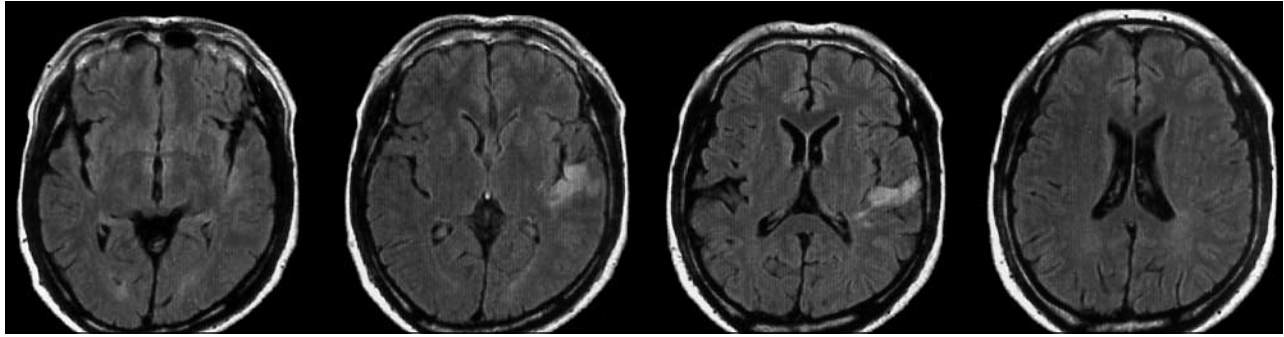
Kertesz⁶ showed significantly higher performance in writing words than nonwords to dictation, but oral naming was extremely poor as well. The reliability of lesion localization in described patients is also quite variable. Some reports in the pre-magnetic resonance imaging era used computed tomographic scans, which tended to be less sensitive than magnetic resonance imaging scans in demarcating the lesion extent. The variability of clinical findings and lesion localization in previous reports has hindered a more precise symptom-lesion correlation. We recently encountered a patient with pure phonological agraphia after infarction in the left posterior superior temporal gyrus (pSTG). The selective impairment and precise lesion localization observed in this patient may lead us a step further in understanding the neurolinguistic mechanisms for writing.

REPORT OF A CASE

CASE DESCRIPTION

A 51-year-old right-handed Korean man who had been healthy, except for a 2-year history of well-controlled hypertension, presented with speech disturbance. Three

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Fluid-attenuated inversion recovery images obtained on the first hospital day show the high signal intensities restricted within the left posterior superior temporal gyrus and the underlying white matter.

days before hospital admission, he found himself speaking not as fluently as before. He had difficulties in finding appropriate words and initiating sentences. However, his difficulties were not so severe as to cause problems in usual conversation. Comprehension was not affected. On neurological examination on hospital admission, he was alert, with intact orientation. Verbal fluency in spontaneous speech was preserved, except for occasional interruptions due to word-finding difficulties, and no paraphasia was observed. He could obey 3-step commands perfectly, suggesting grossly normal auditory comprehension. His spontaneous and dictatorial writing of words and short sentences showed no error in spelling. His reading comprehension was intact, even with rather long and complex sentences. The only deficit observed in language function was an impairment of repetition. No abnormality was found in tests of other cognitive functions, such as praxis, left-right orientation, calculation, finger naming, and spatial attention. Other parts of the neurological examination, including examination of the cranial nerves, motor and sensory functions, and reflexes, were unremarkable.

The results of routine blood tests were all within normal limits. A transcranial Doppler study revealed a mild increase in the flow velocity at the proximal trunk of the left middle cerebral artery. Magnetic resonance imaging of the brain performed on the second hospital day revealed a hyperintense lesion in the left pSTG and underlying white matter on T2-weighted and fluid-attenuated inversion recovery scans (**Figure**). Magnetic resonance angiography showed decreased vascularity distal to the left middle cerebral artery bifurcation.

FURTHER ASSESSMENT OF LANGUAGE FUNCTION

Language function was further assessed with the Korean version of the Western Aphasia Battery on the fourth hospital day. While spontaneous speech, auditory comprehension, and naming remained intact, the patient's oral repetition was significantly improved in that he had difficulties only in repeating sentences composed of 5 or more words. His repetition of single words or shorter sentences was normal. His aphasia quotient was 98.6. Whereas his initial language deficit was definitely compatible with conduction aphasia, the result of the Korean version of the Western Aphasia Battery on the fourth

hospital day revealed a performance within normal range using quantitative criteria.

On the same day, to evaluate his performance in repetition more thoroughly, we administered more detailed tests of repetition in auditory and written forms. First, the patient was asked to orally repeat 15 high-frequency words, 15 low-frequency words, and 15 nonwords immediately after each item was spoken to him. The syllable length of the words and nonwords ranged from 1 to 4. The patient repeated all words and nonwords correctly. Second, he was asked to write the words and nonwords as soon as he heard them. He correctly wrote all 15 high-frequency words, 14 low-frequency words, and 7 nonwords. From these tests, selective impairment of writing nonwords to dictation was observed, compatible with phonological agraphia. He wrote nonword syllables that were similar but different from what was spoken to him, while he wrote words correctly. Furthermore, he made errors only in dictatorial writing, but not in oral repetition. Although no normal control data are available, a person of his education and job level would be expected to perform perfectly on these tests. From a detailed inspection of his errors in writing nonwords to dictation, it was observed that all but 1 errors were made on second or later syllables of the nonwords.

COMMENT

Three parallel routes may be involved in writing: a phonological route, a lexical route, and a semantic route.^{2,3} In writing nonwords, only the phonological route can be used because nonwords have no lexical or semantic representation. Roeltgen et al³ suggested that the phonological route is mediated by 2 sequential processes: (1) a segmentation process, in which syllable sequences of words or nonwords are broken down into phonemes; and (2) the phoneme-to-grapheme conversion, in which the segmented phonemes are translated into graphemes. They suggested that selective loss of either of these 2 processes or both could lead to phonological agraphia. We could not determine definitely which of the 2 components was impaired in our patient because we did not evaluate his ability to write a single phoneme to dictation, which would be impaired in the case of a phoneme-to-grapheme conversion defect. However, from the finding that our patient erred mostly at the second or later

syllables of the presented nonwords, we suggest that his deficit was more of segmentation than of phoneme-to-grapheme conversion. Presumably, if the deficit had been in phoneme-to-grapheme conversion and not in segmentation, he would have erred regardless of the position of a phoneme within the nonwords.

For neurolinguistic correlates, the left anterior-inferior supramarginal gyrus has been suggested as the anatomical substrate for phonological agraphia,³ because it was the only area that was damaged in all patients when their computed tomographic scans were superimposed. However, the 4 patients of the study showed considerable anatomical and clinical variability, and for each patient, the lesion extended over a larger area into the frontal operculum, STG, postcentral gyrus, and/or superior parietal lobule. Alexander et al⁹ suggested that, from the analysis of their 2 cases of phonological agraphia and other published cases, phonological agraphia could be produced by lesions in a wide range of perisylvian cortical regions. Phonological agraphia following a large right frontal infarction, superior temporal and anterior parietal destruction,⁵ an extensive left perisylvian infarction,¹ a left insula and basal ganglia infarction,⁷ and a focal left anterior insulo-opercular infarction⁴ has also been reported. However, as previously noted, all these patients showed language disturbances beyond the restrictive criteria of phonological agraphia, and lesions in most patients were quite extensive and presumably encompassed neural substrates of many linguistic functions. In contrast to these patients, our patient had a selective impairment limited only to writing nonwords to dictation and a restricted lesion in the left pSTG and underlying white matter, indicating the importance of the area in writing via the phonological route. However, the route was not completely destroyed in the patient because he was able to write about 47% of the nonwords to dictation. This partial impairment may have resulted because of either incomplete damage to the left pSTG or the widely distributed nature of the network areas involved in the function, as suggested by the published reports previously mentioned.

The role of the left pSTG in language function has been suggested by reports^{10,11} that a pSTG lesion was associated with conduction aphasia and by studies^{12,13} that showed that pSTG stimulation produces aphasic symptoms comparable to conduction aphasia. According to these studies, pSTG dysfunction leads to impairments of phonological output lexicon,¹² phonological loading process during speech production,¹³ or phonological judgment of presented stimuli,¹⁴ which then may result in conduction aphasia. Contrary to these reports, our patient showed no impairment in phonological input processing and phonological output processing, as evidenced by normal performance on oral repetition of nonwords. The deficit in our patient was limited to phonological writing to dictation. We believe this discrepancy between our case and the previously reported cases of conduction aphasia is because of a difference in the exact location of the lesions within the left pSTG. Given that the region seems to be operative in a wider range of phonological processes, not limited to phonological output in auditory modality, it may as well be composed of several subdivi-

sions with roles slightly different from each other. This possibility of functional parcellation of the area, however, requires further studies armed with better spatial resolution than was possible in the present study.

In summary, the present case, in which a selective lesion in the left pSTG was associated with phonological agraphia with little or no impairment in other aspects of the linguistic functions, indicates that this area plays quite an important role in phonological linkage of auditory input with orthographic output, as needed for writing nonwords to dictation.

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