

Case Reports

Mixed Transcortical Aphasia Without Anatomic Isolation of the Speech Area

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We report two patients with mixed transcortical aphasia following left frontal lobe infarctions. Although there was no evidence of anatomic isolation of the speech area on computed tomograms or magnetic resonance imaging scans, single-photon emission computed tomography in one case demonstrated diminished blood flow over the left parietal convexity suggestive of "functional isolation" of the posterior perisylvian language zone. (*Stroke* 1990;21:953-956)

Mixed transcortical aphasia is characterized by reduced or absent spontaneous speech, severely impaired language comprehension, and preserved repetition.¹⁻³ Although the localization of lesions producing mixed transcortical aphasia is variable,^{4,5} the syndrome is seen most often with diffuse or multifocal pathologic changes that involve both anterior and posterior left hemisphere cortical association areas but spare the perisylvian language core.³ According to Goldstein,¹ the critically placed lesions produce an "isolation of the speech area" posteriorly from the "ideational field" of the parietal association cortex and anteriorly from frontal areas important for the production of volitional speech. Since Broca's area, Wernicke's area, and the arcuate fasciculus are usually spared, repetition remains intact.

Acute mixed transcortical aphasia is uncommon.⁶ Recently, Bogousslavsky et al⁶ described four cases of acute mixed transcortical aphasia in the setting of internal carotid artery occlusion with computed tomographic (CT) evidence of simultaneous anterior precentral-central sulcus artery territory embolic infarction and posterior watershed infarction related to hemodynamic insufficiency. These cases support Goldstein's contention by demonstrating both anterior and posterior anatomic isolation of the speech area.

We report two cases of mixed transcortical aphasia in which CT and magnetic resonance imaging (MRI) showed only frontal left hemisphere infarcts.

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Case 1

A 79-year-old right-handed woman was admitted for the sudden onset of right-sided weakness and the inability to speak. On admission, spontaneous speech was reduced to the repetitive utterance "wah-wah-wah." She was unable to follow any commands and could not name or repeat. Four days later echolalic repetition of sentences was noted. Although spontaneous speech was still virtually absent, she could now repeat words and sentences, completed open-ended sentences, and was able to sing. Auditory comprehension remained severely impaired even at the single-word level. She could name only four of the first 30 items on the Boston Naming Test.⁷ She was able to read aloud single words without evidence of comprehension.

CT 4 days after admission (Figure 1) and MRI 2 weeks later showed a left frontal infarct involving the territories of both the middle and anterior cerebral arteries. The lesion destroyed Broca's area and extended superiorly and anteriorly into the premotor and prefrontal regions of the left hemisphere. The lower precentral gyrus was also involved, but there were no retrorolandic lesions.

Single-photon emission computed tomography (SPECT) with intravenous iofetamine performed 3 weeks after admission showed an area of low blood flow corresponding to the frontal lesion (Figure 2) but also revealed an area of diminished blood flow over the parietal convexity of the left hemisphere. Carotid Doppler study was unremarkable.

The patient's condition remained unchanged until 4 weeks after admission, when she suddenly developed left-sided weakness. Although awake and alert, she could no longer repeat and was essentially mute. CT showed a new right frontoparietal infarct in the territory of the middle cerebral artery. The patient

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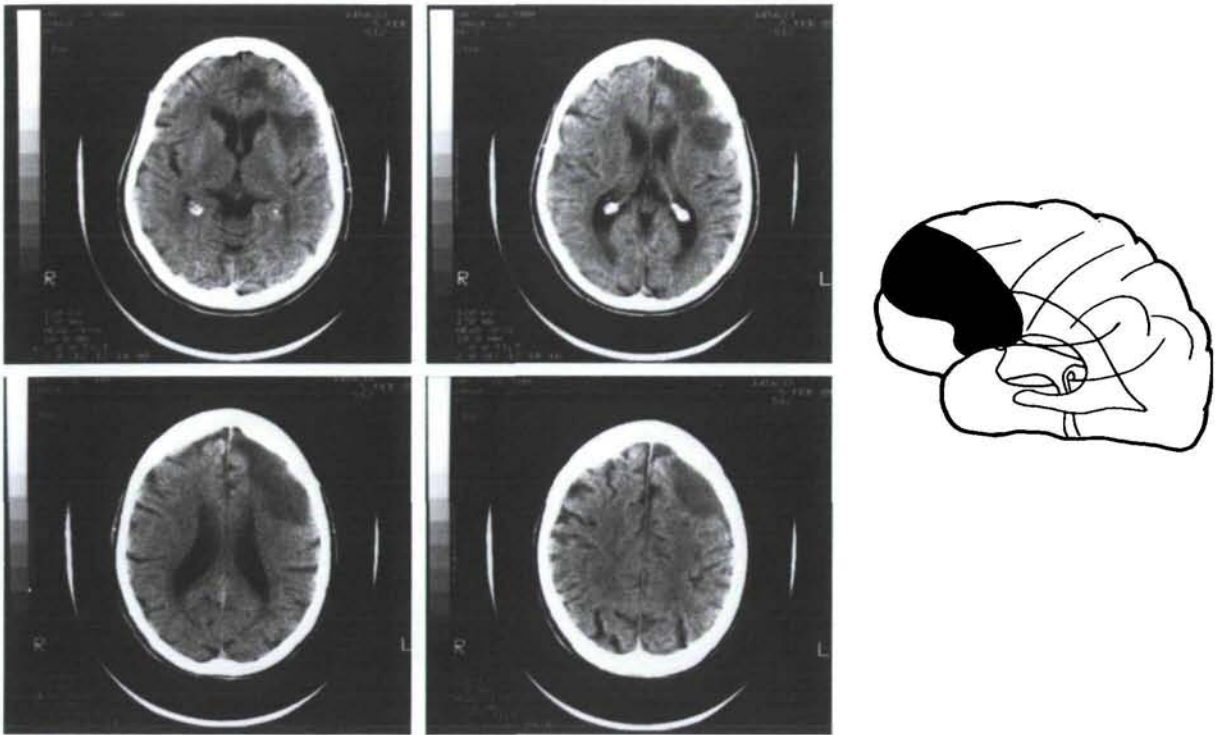


FIGURE 1. Computed tomograms and diagram showing left frontal lesion in Case 1.

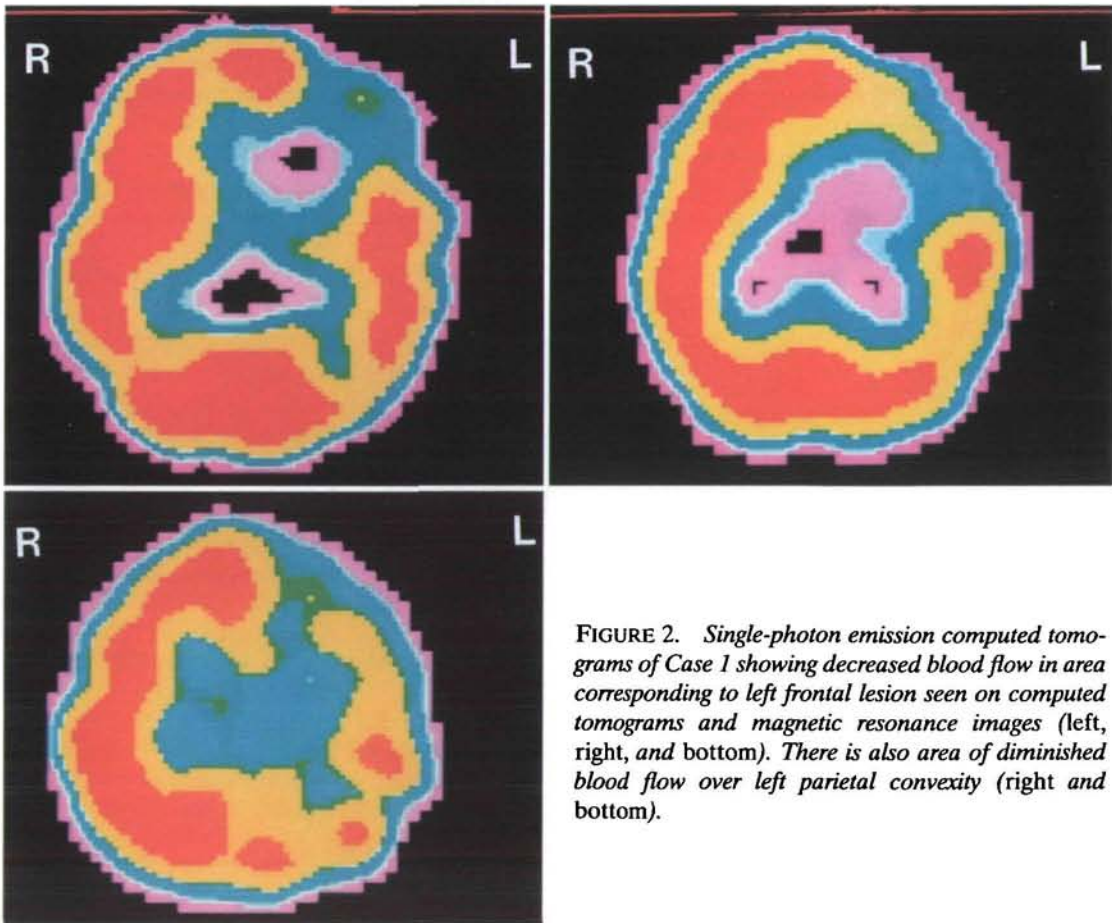


FIGURE 2. Single-photon emission computed tomograms of Case 1 showing decreased blood flow in area corresponding to left frontal lesion seen on computed tomograms and magnetic resonance images (left, right, and bottom). There is also area of diminished blood flow over left parietal convexity (right and bottom).

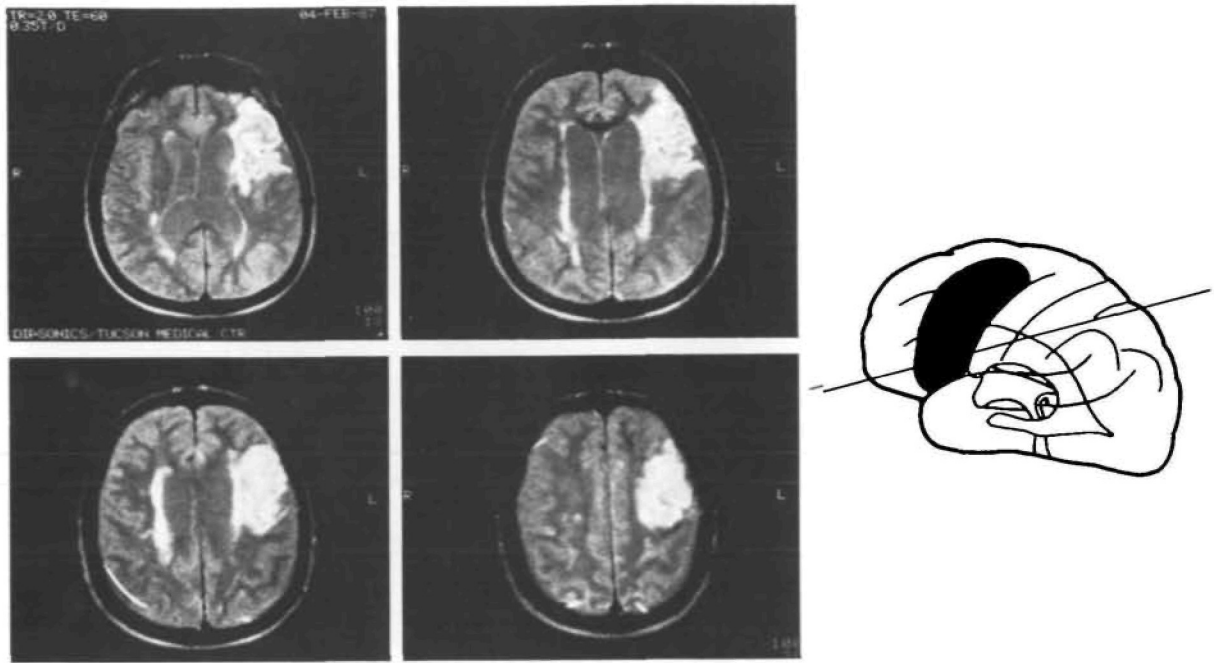


FIGURE 3. Magnetic resonance images and diagram demonstrating left frontal lesion in Case 2. Solid line indicates angle of magnetic resonance imaging scan.

died of a cardiac arrest 4 days later. Request for autopsy was denied.

Case 2

A 62-year-old right-handed man was admitted for the sudden onset of the inability to speak. On admission, spontaneous speech was absent. Although alert and attentive, he did not follow any commands and could not repeat or name. Mild right facial weakness and right upper extremity drift were present. Two days later he was noted to produce single meaningless repetitive utterances and occasional echolalic repetition. Propositional speech was still virtually absent, but he could now follow axial commands. By the fourth day he accurately repeated words, sentences, and meaningless phrases and completed open-ended sentences. Except for axial commands, auditory comprehension remained severely impaired even at the single-word level. He occasionally correctly named a few items on the Boston Naming Test, but would soon start to perseverate. Although he could read words and sentences aloud, he gave no indication that he understood what he read. Spontaneous writing was impossible, but he correctly spelled to dictation several words he did not understand.

CT 4 days after admission and MRI 3 weeks later (Figure 3) showed a large left frontal infarct in the territory of the middle cerebral artery. The lesion destroyed Broca's area and involved the dorsolateral premotor and prefrontal cortex anterior and superior to it. The primary motor cortex was spared, and there were no retrorolandic lesions.

Although some improvement of language comprehension was observed, the patient's condition remained essentially unchanged for the first 6 weeks.

At 3 months' follow-up, spontaneous speech was still sparse but he was able to produce occasional meaningful utterances. A significant improvement of auditory and reading comprehension was noted.

Discussion

These two cases had very similar language profiles. After a brief initial period of global aphasia, the language deficit rapidly evolved into classical mixed transcortical aphasia. CT and MRI showed only left frontal lobe infarcts in both cases. The lesion destroyed Broca's area and involved premotor and prefrontal regions anterior and superior to it. Partial or complete destruction of Broca's area has been described in reports of transcortical aphasia, leading some investigators to believe that repetition was mediated by the intact right hemisphere in these cases.⁸⁻¹⁰ The abolition of repetition following the right hemispheric stroke in Case 1 is consistent with this view.

The lack of CT or MRI evidence for posterior anatomic isolation of the speech area is an unexpected finding in mixed transcortical aphasia. However, in Case 1 SPECT demonstrated an area of diminished blood flow over the left parietal convexity, suggesting that decreased functional activity of this cortical region results in language deficits typically seen with structural lesions that anatomically isolate the posterior perisylvian language zone. Based on these observations, we propose that "functional isolation" of the speech area may be a mechanism of mixed transcortical aphasia in cases without anatomic isolation.

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References

1. Goldstein K: *Language and Language Disturbances*. New York, Grune & Stratton Inc, 1948, pp 292-324
2. Geschwind N, Quadfasel FA, Segarra JM: Isolation of the speech area. *Neuropsychologia* 1968;6:327-340
3. Albert ML, Goodglass H, Helm NA, Rubens AB, Alexander MP: *Clinical Aspects of Dysphasia*. Wien, Springer-Verlag, 1981, pp 99-100
4. Ross ED: Left medial parietal lobe and receptive language functions: Mixed transcortical aphasia after left anterior cerebral artery infarction. *Neurology* 1980;30:144-151
5. McFarling D, Rothi LJ, Heilman KM: Transcortical aphasia from ischemic infarcts of the thalamus: Report of two cases. *J Neurol Neurosurg Psychiatry* 1982;45:107-112
6. Bogousslavsky J, Regli F, Assal G: Acute transcortical mixed aphasia: A carotid occlusion syndrome with pial and watershed infarcts. *Brain* 1988;11:631-641
7. Kaplan E, Goodglass H, Weintraub S: *Boston Naming Test*. Philadelphia, Lea & Febiger, 1983
8. Bastian H: Some problems in connexion with aphasia and other speech defects. *Lancet* 1897;1:933-942, 1005-1017, 1131-1137, and 1187-1194
9. Niessl von Mayendorf E: *Die aphasischen Symptome*. Leipzig, Engelmann, 1911
10. Rubens AB: Transcortical motor aphasia, in Whitaker H, Whitaker HA (eds): *Studies in Neurolinguistics*. New York, Academic Press Inc, 1976, vol 1, pp 293-306

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