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Stroke 2010;41;325-330; originally published online Dec 31, 2009;

DOI: 10.1161/STROKEAHA.109.570374

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ISSN: 1524-4628

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Ischemia in Broca Area Is Associated With Broca Aphasia More Reliably in Acute Than in Chronic Stroke

Elisa Ochfeld; Melissa Newhart, BA; John Molitoris, BS; Richard Leigh, MD; Lauren Cloutman, PhD; Cameron Davis, MS; Jennifer Crinion, PhD; Argye E. Hillis, MD

Background and Purpose—We aimed to determine if ischemia involving Broca area predicts Broca aphasia more reliably in acute or chronic stroke.

Methods—We included consecutive right-hand-dominant patients with left hemisphere ischemic stroke (<48 hours from onset for acute stroke or >6 months after stroke for chronic stroke). MRI scans were analyzed for ischemic lesions or hypoperfusion in Broca area (Brodmann areas 44 and 45). Patients were scored on the Western Aphasia Battery to classify aphasia syndromes; χ^2 tests were used to identify significant associations.

Results—The presence of infarct involving any part of Broca area and the presence of Broca or global aphasia was much stronger in acute ($\chi^2=38.1$; $df1$; $P<0.0001$) than in chronic stroke ($\chi^2=0.54$; $df1$; $P=0.46$; not significant). The association between infarct or hypoperfusion covering all of Broca area and the presence of Broca or global aphasia was much stronger in acute ($\chi^2=35.8$; $df1$; $P<0.0001$) than in chronic stroke ($\chi^2=1.2$; $df1$; $p=0.27$; not significant). In a subset of 20 patients studied longitudinally, the associations were significant only acutely, not chronically ($\chi^2=20$; $df1$; $P<0.0001$ vs. $\chi^2=0$; $df1$; $p=1$; not significant for ischemia involving part of Broca area, and $\chi^2=16.4$; $df1$; $P<0.0001$ vs $\chi^2=3.2$; $df1$; $p=0.08$; not significant for ischemia covering all of Broca area).

Conclusions—Broca aphasia is more reliably associated with infarct/ hypoperfusion of Broca area in acute stroke. Many chronic stroke patients with damage to part or all of Broca area had neither Broca nor global aphasia. Broca or global aphasia was sometimes present initially in these patients but resolved by 6 months. Our results indicate that the acute aphasia syndrome may allow the clinician to predict the compromised vascular territory, even when structural imaging shows only a small (or no) infarct. (*Stroke*. 2010;41:325-330.)

Key Words: acute stroke ■ aphasia ■ brain imaging ■ cognitive impairment ■ ischemia ■ magnetic resonance

It is commonly taught that aphasia syndromes are not reliably associated with particular lesion locations in acute stroke because patients' performance on tasks can vary from day to day in the acute period. However, these fluctuations in language performance may reflect important changes in blood flow,¹ and the aphasia syndrome at a given point of time may actually be an excellent predictor of the vascular territory that is ischemic (hypoperfused or infarcted). If this is the case, then rapid assessment of language might help direct clinical decision-making in acute stroke. For example, consider 2 patients who have acute left subcortical infarcts on diffusion-weighted imaging (DWI, but do not have CT or MR perfusion imaging because of relative contraindications for contrast (severe renal failure). One has no aphasia; the second has Wernicke aphasia. If the aphasia syndrome reliably predicts the vascular territory that is ischemic in the acute stage of stroke, then the clinician can assume that in patient 2

only the inferior division of the left middle cerebral artery is at risk, requiring aggressive intervention.

It is commonly believed that damage to Broca area underlies Broca aphasia. However, the 2 are not always paired exclusively.^{2,3} Many patients with damage to Broca area have language deficits immediately after stroke but recover over time. This recovery may be attributable to reorganization, in which other brain areas assume the functions of lost tissue, or achieved through rehabilitation, which allows alternative strategies to be used when confronting language difficulties. In both circumstances, language tasks that previously depended on Broca area are now accomplished using different brain regions or different cognitive mechanisms (despite the lesion in Broca area).⁴ These transformations require time; they do not occur in the first few days of stroke. Rather, recovery in the first few days requires restoration of blood flow.⁵ Thus, the association between

Received October 13, 2009; final revision received October 20, 2009; accepted November 5, 2009.

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DOI: 10.1161/STROKEAHA.109.570374

damage to Broca area and Broca aphasia may be higher in acute than in chronic stroke because of structure/function reorganization in the chronic stage.

Broca area itself is controversial because it is defined using different neuroanatomical boundaries. It is most often defined as Brodmann areas 44 and 45, areas of cytoarchitecture commonly found in the left posterior inferior frontal cortex.⁶ Broca area has been implicated in a variety of language functions, including grammatical speech production, verb naming, comprehending syntactically complex sentences (eg, passive voice), phonological working memory, and orchestrating speech articulation.^{7–10}

The complete vascular syndrome of Broca aphasia (with impaired fluency, word-finding, articulation, repetition, and comprehending and producing complex grammatical structures) is thought to require not only damage to Broca area but also infarct of the insula and adjacent cerebrum.^{10,11} Damage to Broca area alone may produce an isolated impairment of motor speech, sometimes known as apraxia of speech, and not the full syndrome of Broca aphasia.^{10,12} This observation may not be true in the first day of stroke, however, when tissue dysfunction relatively restricted to Broca area results in the full clinical (vascular) syndrome of Broca aphasia.¹²

We aimed to test the hypothesis that ischemia involving Broca area is more strongly associated with Broca aphasia in acute stroke than in chronic stroke. If confirmed, results would indicate that the vascular aphasia syndromes, including Broca aphasia, may be more reliably associated with the area of ischemia (hypoperfusion or infarct) in the acute stage, and that day-to-day behavioral variations reflect changes in blood flow in the acute period that can be monitored using perfusion-weighted imaging (PWI).

Subjects and Methods

Participants

We included consecutive patients who were right-hand-dominant with left hemisphere ischemic stroke (<48 hours from onset of symptoms or >6 months after stroke) and native speakers of English. Exclusion criteria included inability to complete language testing or MRI within 24 hours of hospital admission, previous symptomatic stroke or other neurological disease, hemorrhage on initial CT (computed tomography scan) or MRI, impaired level of consciousness, intravenous sedation, uncorrected hearing or visual loss, or inability to provide informed consent or indicate a family member to provide consent. We attempted to follow-up all patients who were studied acutely. However, only 20 of 50 returned for retesting at 6 months. An additional 10 patients were studied only chronically.

Imaging

MRI scans, including T2, DWI, and PWI, were obtained on a 3-Tesla Philips magnet (in a few cases scans were obtained on a 1.5-Tesla GE or Siemens magnet, but the variability across patients' brains far exceeds the variability between scanners). For PWI, 20 mL GdDTPA (Gadolinium) was power-injected at 5 mL/sec. Voxel size for PWI and DWI was 4.4 mm³. Slices were 5 mm, with whole brain coverage. Images were analyzed by technicians blinded to the language data and aphasia classification for the presence of dense ischemia (bright on DWI, dark on apparent diffusion coefficient [ADC]) in the acute stage or infarct (on T2 in the chronic stage) or hypoperfusion (on time-to-peak maps derived from PWI) involving Broca area, with Broca area

defined as Brodmann area 44 and/or 45 using a probabilistic cytoarchitectural map.⁶ Time-to-peak maps were registered to T2 images, which have better spatial resolution, to provide anatomic landmarks. Each patient's scans were classified according to the presence or absence of ischemia (on DWI) or infarct (on T2) or hypoperfusion, defined as >4-second delay in time to peak relative to the homologous region in the right hemisphere in any part of Broca area or covering the entire region of Broca area.

Language Assessment

The Western Aphasia Battery–Revised (WAB-R)¹³ was administered to all patients. WAB-R results were used to classify patients as having Broca aphasia, Wernicke aphasia, global aphasia, anomic aphasia, conduction aphasia, transcortical motor aphasia, transcortical sensory aphasia, isolation aphasia, or as being nonaphasic or unclassifiable by a research assistant blinded to the imaging results.

Statistical Analysis

Because dysfunction in Broca area is classically associated with both Broca aphasia and global aphasia, we evaluated this combined aphasia syndrome/lesion association at the acute and chronic stages of stroke. We identified the relationship between the dichotomous lesion classifications (presence vs absence of ischemia anywhere in/covering all of Broca area) and dichotomous aphasia classifications (presence vs absence of Broca or global aphasia) with χ^2 tests. We used an alpha level of $P<0.05$ after correction for 4 comparisons ($P<0.0125$).

Results

A consecutive series of patients who met inclusion and exclusion criteria participated in this study. There were a total of 50 patients studied acutely (<48 hours after onset) and 30 studied chronically (>6 months after onset). A subset of 20 patients was studied at both time periods. There were no significant differences between acute and chronic patients regarding demographics. Acute patients were 47% female, ages 31 to 83 years, with 7 to 24 years of education. Chronic patients were 53% female, ages 24 to 85 years, with between 12 and 20 years of education. Mean age for acute patients was 59.6 years (SD, 12.1) and 58.8 years (SD, 15.4) for chronic patients. Mean education was 14.0 years (SD, 3.7) for acute patients vs 15.3 years (SD, 2.6) for chronic patients. Acute stroke patients, not surprisingly, had more severe aphasia. Mean WAB-R aphasia quotient was 70.0 (SD, 30.7) for acute vs 82.1 (SD, 22.6) for chronic stroke patients; the aphasia quotient ranges from 0 to 100, with 100 being nonaphasic. The subset of 20 patients studied at both time periods was similar to the entire group: age 39 to 80 (mean, 56.6; SD, 11.9); education 12 to 20 years (mean, 15.4; SD, 2.8); and 55% female. Their acute aphasia quotient range was 2 to 97.6 (mean, 41.5; SD, 36.1); chronic aphasia quotient range was 1.6 to 100 (mean, 84.5; SD, 22.3).

Ischemia Involving Any Part of Broca Area

The association between infarct and hypoperfusion anywhere within Broca area and the presence of Broca or global aphasia was much stronger in acute ($\chi^2=38.1$; $df1$; $P<0.0001$) than in chronic stroke ($\chi^2=0.54$; $df1$; $P=0.46$; not significant). All acute stroke patients with Broca aphasia or global aphasia had infarct/hypoperfusion that included at least part of Broca area. In contrast, 1 chronic stroke patient with Broca aphasia had an infarct that did not include Broca area. Moreover, 80% of acute and 16.6%

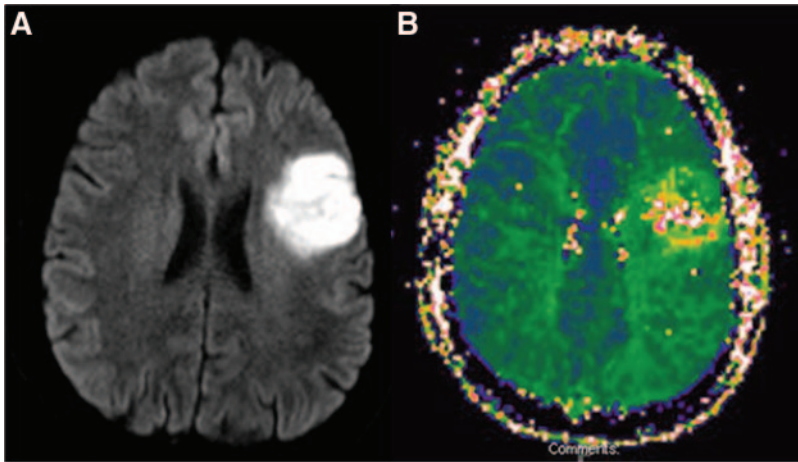


Figure 1. DWI (A) and PWI (B) scans of an acute patient with infarct/hypoperfusion involving Broca area who did not have Broca or global aphasia. This patient was classified as having transcortical motor aphasia on the WAB-R.

of chronic stroke patients with infarction/hypoperfusion involving Broca area had either Broca aphasia or global aphasia. Only 2 acute patients with infarct/hypoperfusion involving Broca area did not have Broca or global aphasia. These patients were both classified as having transcortical motor aphasia on the WAB-R (Figure 1). However, the other acute patients with transcortical motor aphasia ($n=3$) had more classical lesions in the watershed area between the anterior cerebral artery and middle cerebral artery territories (Figure 2).

In the 20 patients studied longitudinally, the association between ischemia involving part of Broca area and Broca or global aphasia was significant only acutely ($\chi^2=20$; $df1$; $P<0.0001$), not chronically ($\chi^2=0$; $df1$; $p=1$; not significant). Eleven of these patients had ischemia in Broca area acutely and chronically; all 11 had Broca or global aphasia acutely, and only 1 had global aphasia (and none had Broca aphasia) at 6 months.

Ischemia Covering All of Broca Area

The association between infarct and hypoperfusion covering all of Broca area and the presence of Broca or global aphasia was much stronger in acute ($\chi^2=35.8$; $df1$; $P<0.0001$) than in chronic stroke ($\chi^2=1.2$; $df1$; $p=0.27$; not significant). All of the acute stroke patients with dysfunction of all of Broca area were classified as either Broca or global aphasics. Among

chronic stroke patients, 83.3% with infarct covering all of Broca area did not have either Broca or global aphasia.

In the 20 patients studied longitudinally, the association between ischemia covering all of Broca area and Broca or global aphasia was significant only acutely ($\chi^2=16.4$; $df1$; $P<0.0001$), not chronically ($\chi^2=3.2$; $df1$; $p=0.08$; not significant).

Classical Vascular Aphasia Syndrome/Lesion Associations

In acute stroke, 6 patients had infarct (or dense ischemia) on DWI covering only part of Broca area, and 3 had infarct or dense ischemia covering all of Broca area. Of these, 6 had matched perfusion defects, 1 had no perfusion defect, and 2 with infarcts involving part of Broca area had hypoperfusion of all of Broca area. Another 2 patients had no infarct in Broca area but hypoperfusion of part of Broca area or all of Broca area (Figure 3). The patient with hypoperfusion only in Broca area had Broca aphasia; the patient with hypoperfusion of the entire middle cerebral artery territory had global aphasia. That is, the area of hypoperfusion, but not the infarct in these 2 cases, predicted the classical vascular aphasia syndrome in the acute phase after stroke.

Most of the chronic stroke patients (selected for having had a left hemisphere stroke, not for having aphasia) showed anomia or no aphasia. However, 1 chronic stroke

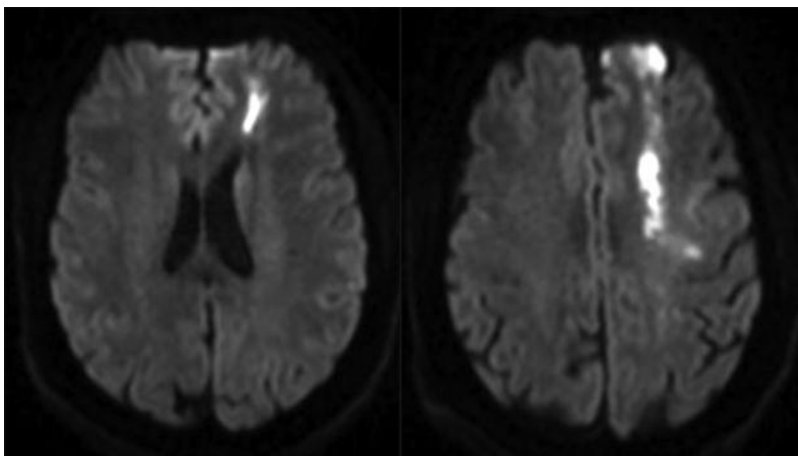


Figure 2. DWI of a patient with transcortical motor aphasia and the more typical lesion location.

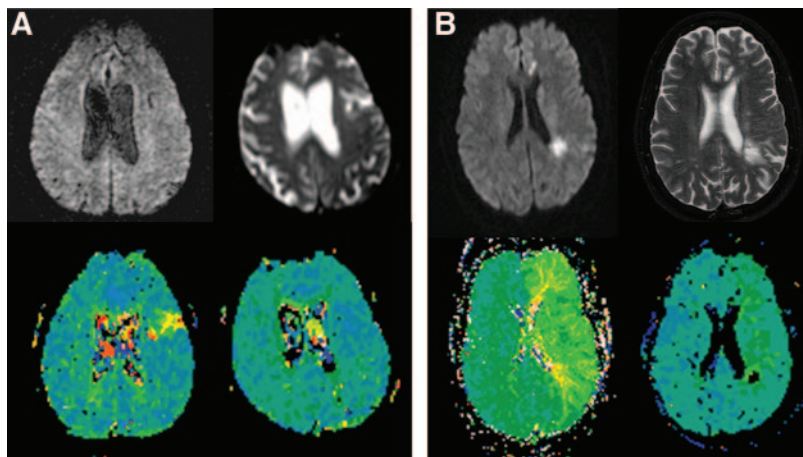


Figure 3. A, DWI or T2 (top) and PWI (bottom) at day 1 (left) and follow-up (right) in a patient who had Broca aphasia at day 1 when he had hypoperfusion of Broca area, which resolved with reperfusion of Broca area (before day 2; he remained nonaphasic at 6-month follow-up). B, DWI or T2 (top) and PWI (bottom) at day 1 (left) and follow-up (right) in a patient who had global aphasia at day 1 when she had hypoperfusion of the entire left middle cerebral artery and anterior cerebral artery territories, which resolved with reperfusion (at day 10; she remained nonaphasic at 6-month follow-up).

patient showed a classic lesion–deficit association: global aphasia associated with infarct involving both Broca area and Wernicke area (Figure 4). Another chronic stroke patient had Broca aphasia with a lesion involving both Broca and Wernicke areas. This patient initially had global aphasia but had recovered comprehension of words and sentences with simple syntax. Nevertheless, speech remained halting with impaired articulatory planning.

Exceptions to the Classical Vascular Aphasia Lesion Associations

Scans of the only 2 acute stroke patients who had ischemia in Broca area but not the classically associated syndrome (ie,

had transcortical motor aphasia rather than Broca aphasia) are shown in Figure 1. Several patients with chronic stroke with lesions involving or covering all of Broca area had anomia or no aphasia (Figure 5).

Discussion

In our patient sample, Broca aphasia was more consistently associated with infarction/hypoperfusion of Broca area in acute stroke than in chronic stroke. Broca aphasia is a vascular syndrome. Many patients with damage to all of Broca area at the chronic stage had neither Broca nor global aphasia. These patients may have had Broca aphasia initially, but it may have resolved over time because of reorganization of structure/function relationships (as confirmed in the longitudinal study of 20 patients).

Our results differ from those reported in early CT studies of classical aphasia in 2 ways. First, we selected patients on the basis of having a first symptomatic ischemic stroke in the left hemisphere, either in the past 48 hours or at least 6 months previously. Earlier studies selected patients who had chronic aphasia only. Consistent with the majority of our patients, it is highly likely that those patients with chronic Broca aphasia do have lesions involving Broca area, at least in most cases.^{14,15} Second, by including PWI, we were able to identify dysfunctional brain tissue that would not have been visible on acute CT or structural MRI that likely contributed to the patients' language impairments in the acute stage (Figure 3). Our results do not address the issue of whether dysfunctional tissue restricted to Broca area more commonly results in the full syndrome of Broca aphasia¹² or a more restricted motor speech disorder.¹⁰

Nonfluent aphasias including Broca aphasia are reported in higher frequencies in younger patients. In the acute phase, this has been attributed to a higher proportion of anterior lesions in younger patients and higher proportion of posterior lesions in older patients.¹⁶ The age influence on location of infarct may reflect stroke mechanism, with more frequent cardioembolic strokes (which are associated with inferior division middle cerebral artery strokes)¹⁷ in older individuals, and more frequent carotid dissection (which often causes infarcts that include superior division middle cerebral artery territory, including Broca area) in younger individuals. Irrespective of the explanation for the age effect on lesion

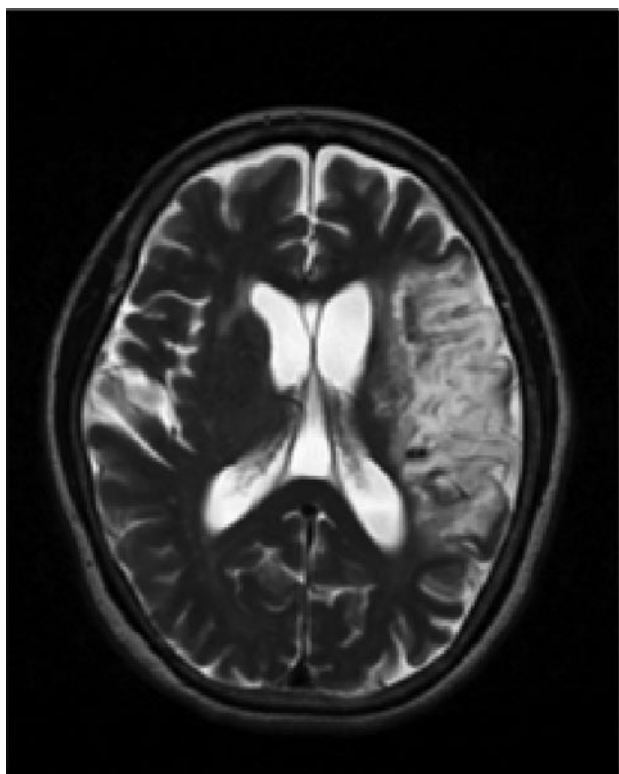


Figure 4. The 1 chronic stroke patient who showed a classic lesion–deficit association. This patient had global aphasia associated with infarct involving both Broca area and Wernicke areas.

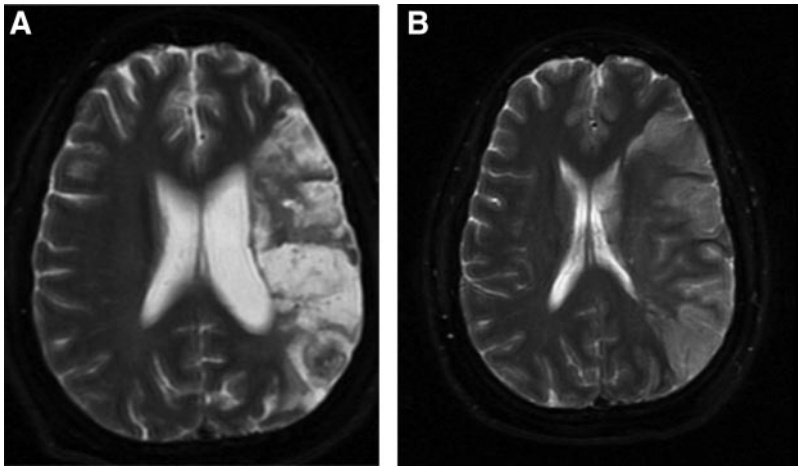


Figure 5. Exceptions to the classical vascular aphasia lesion associations in chronic stroke. A, Patient with anomic aphasia but infarct involving both Broca and Wernicke areas. B, Patient with no aphasia but infarct involving both Broca and Wernicke areas.

location, the effect can account for the different frequency of aphasic vascular syndromes at different ages.¹⁸ This conclusion augments our finding that infarct location may be more closely related to the aphasia syndrome in acute than chronic stroke.

Our patients were relatively young (on average ≈ 59 years) but not atypical for stroke patients admitted to our hospital. In a recent study, we reported data from 226 white and 284 black patients admitted to our stroke service.¹⁹ There was a significant difference in age across racial groups by ANOVA ($F=4.2$; $P=0.016$). Mean age was 63.4 ± 16.1 years for white vs 59.8 ± 14.4 for black patients. Slightly more than half of our patients were black, so it is not surprising that our patients were, on average, closer to 59 years. Also, we excluded patients with previous strokes, dementia, and uncorrected hearing or visual loss, which would have been more common in older stroke patients.

Lesion location has long been acknowledged as the major determinant of aphasia attributes. However, the methods used to determine the aphasia classification are important. A large percentage (50%–60%) of aphasias are not classifiable in either the acute stage¹⁸ or the chronic stage,²⁰ using classic descriptions or the Boston Diagnostic Aphasia Examination. In contrast, nearly all patients can be classified using the WAB-R, which groups patients into 1 syndrome classification or another based on scores on fluency, naming, comprehension, repetition, and auditory comprehension. The resulting aphasia classification, however, does not always match the clinical impression.²¹ In our patient sample, the 2 patients with acute ischemia in Broca area who did not have Broca aphasia by the WAB-R had transcortical motor aphasia on the WAB-R. These patients had relatively high scores on sentence repetition attributable to partial credit on most items, but they had halting, agrammatic sentence repetition, which is more consistent with the classical description of Broca aphasia. Nevertheless, we used the WAB-R for classification in this study because it is relatively objective and reproducible.

Conclusion

Recognizing the limitations of WAB-R classification and the relatively small sample size of our study, our results chal-

lenge the commonly held belief that acute aphasia does not reflect classic lesion–deficit associations, at least when “lesion” is defined as ischemic tissue that can be visualized with PWI. This result is clinically important because it indicates that the acute aphasia syndrome allows the clinician to predict the vascular territory that is compromised, even when structural imaging shows only a small (or no) infarct. For example, if the patients whose scans are shown in Figure 3 had not undergone PWI, then their vascular syndromes would have indicated that there was eloquent brain tissue at risk, indicating a need for aggressive intervention.

Acknowledgments

The authors gratefully acknowledge the support and the participation of the patients in the study.

Sources of Funding

The research reported in this article was supported by NIH R01 DC05375 to AH.

Disclosure

None.

References

1. Croquelois A, Wintermark M, Reichhart M, Meuli R, Bogousslavsky J. Aphasia in hyperacute stroke: language follows brain penumbra dynamics. *Annals of Neurology*. 2003;54:321–329.
2. Basso A, Lecours AR, Moraschini S, Vanier M. Anatomical correlations of the aphasia defined through computerized tomography: Exceptions. *Brain Language*. 1985;26:201–229.
3. Dronkers NF, Shapiro JK, Redfern B, Knight RT. The role of Broca area in Broca aphasia. *J Clin Exp Neuropsychol*. 1992;14:52–53.
4. Kertesz A. Recovery of aphasia. In: Feinberg TE, Farah MJ, eds. *Behavioral Neurology and Neuropsychology*. New York: McGraw Hill; 1997:167–182.
5. Hillis AE, Heidler J. Mechanisms of early aphasia recovery: Evidence from mr perfusion imaging. *Aphasiology*. 2002;16:885–896.
6. Amunts K, Schleicher A, Burgel U, Mohlberg H, Uylings HBM, Zilles K. Broca region revisited: Cytoarchitecture and intersubject variability. *J Comparative Neurol*. 1999;412:319–941.
7. Grodzinsky Y. The neurology of syntax: Language use without Broca area. *Behavioral Brain Sci*. 2000;23:1–71.
8. Paulesu E, Frith DC, Frakowiak RS. The neural correlates of the verbal component of working memory. *Nature*. 1993;362:342–345.
9. Stowe LA. Sentence comprehension and the left inferior frontal gyrus: Storage, not computation. *Behavioral Brain Sci*. 2000;23:51.
10. Mohr JP, Pessin MS, Finkelstein S, Funkenstein HH, Duncan GW, Davis KR. Broca aphasia: Pathologic and clinical. *Neurology*. 1978;28:311–324.

11. Dronkers NF, Plaisant O, Iba-Zizen MT, Cabanis EA. Paul Broca historic cases: High resolution MR imaging of the brains of leborgne and lelong. *Brain*. 2007;130:1432–1441.
12. Davis C, Kleinman JT, Newhart M, Gingis L, Pawlak M, Hillis AE. Speech and language functions that require a functioning Broca area. *Brain Language*. 2008;105:50–58.
13. Kertesz A. *Western aphasia battery-revised*. Austin, TX: Pro-Ed; 2006.
14. Naeser MA, Hayward RW. Lesion localization in aphasia with cranial computed tomography and Boston Diagnostic Aphasia Examination. *Neurology*. 1978;28:545–551.
15. Alexander MP. Aphasia: Clinical and anatomical aspects. In: Feinberg TE, Farah MJ, eds. *Behavioral Neurology and Neuropsychology*. New York: McGraw Hill; 1997:133–150.
16. Ferro JM, Madureira S. Aphasia type, age and cerebral infarct localization. *J Neurol*. 1997;244:505–509.
17. Knepper LE, Biller J, Tranel D, Adams HP Jr, Marsh EE III. Etiology of stroke in patients with Wernicke's aphasia. *Stroke*. 1989;12:1730–1732.
18. Godefroy O, Dubois C, Debachy B, Leclerc M, Kreisler A. Vascular aphasias: Main characteristics of patients hospitalized in acute stroke units. *Stroke*. 2002;33:702–705.
19. Gildersleeve KL, Heidler-Gary J, Newhart M, Davis C, Kannan V, Cloutman L, Hillis AE. Racial differences in stroke characteristics: a retrospective cohort and imaging study. Abstract presented at the Annual Meeting of the American Academy of Neurology. Seattle, WA: American Academy of Neurology; April 2009.
20. Goodglass H, Kaplan E. *Boston Diagnostic Examination for Aphasia*. Philadelphia: Lea & Febiger; 1972.
21. Swindel CS, Hollan AL, Fromm D. Classification of aphasia: WAB type vs. Clinical impression. In: Brookshire RH, ed. *Clinical aphasiology*. Minneapolis: BRK Publishers; 1984:48–54.