Etiology of Stroke in Patients With Wernicke’s Aphasia

Laurie E. Knepper, MD, José Biller, MD, Daniel Tranel, PhD, Harold P. Adams Jr., MD, and E. Eugene Marsh III, MD

We reviewed 49 patients with Wernicke’s aphasia resulting from a stroke. Their aphasia was classified on the basis of comprehensive neuropsychological testing. Wernicke’s aphasia was more common in older patients and in men. Cerebral infarction occurred in 38 patients (78%); the remaining four patients (8%) developed aphasia after surgery for aneurysmal subarachnoid hemorrhage. Embolic events were the most common etiology of Wernicke’s aphasia in the 38 patients with cerebral infarction, with cardiac emboli in 40% and large-vessel atheroemboli from a carotid source in 16%. In patients with Wernicke’s aphasia secondary to infarction, an embolic source should be sought. Patients with Wernicke’s aphasia should have computed tomography to exclude intracerebral hemorrhage before institution of anticoagulant therapy. (Stroke 1989;20:1730–1732)

Wernicke’s aphasia usually results from a lesion in the posterior superior temporal gyrus of the dominant hemisphere (area 22). Lesions often extend into the supramarginal (area 40) and angular (area 39) gyri and into area 37. These regions are perfused by branches of the inferior division of the middle cerebral artery. Cerebral infarction is the leading cause of Wernicke’s aphasia, whereas intracerebral hemorrhage is relatively rare. To define further the types and causes of vascular events that result in Wernicke’s aphasia, we studied a series of patients with this language deficit to determine the etiology of their stroke.

Subjects and Methods

We evaluated 49 patients with Wernicke’s aphasia secondary to stroke at the University of Iowa Hospitals and Clinics between 1980 and 1988. The University of Iowa Hospitals and Clinics serve as the state of Iowa’s comprehensive tertiary health care center and serve primarily the state of Iowa and the northwestern part of Illinois. All patients underwent comprehensive neuropsychological testing within 14 days after the onset of stroke by an investigator who was blinded to their pathophysiological and neuroanatomic status. Speech and language probes from the Multilingual Aphasia Examination and the Boston Diagnostic Aphasia Examination were used to classify the aphasia, and the diagnosis of Wernicke’s aphasia was applied to cases of fluent (i.e., utterances of >7 words, noneffortful speech) paraphasic speech when there were severe (i.e., below the first percentile) defects of comprehension and repetition. All patients also had moderate to severe naming impairments, and most had some degree of reading and writing defects (alexia and agraphia).

All but one patient had cranial computed tomography (CT) within 7 days after the onset of aphasia, and many had serial CT scans. Eleven patients had cranial magnetic resonance imaging (MRI). All radiologic studies were reviewed to evaluate the size and location of the vascular lesion.

Thirty patients had M-mode and two-dimensional echocardiography, 24 had cerebral angiography, and 22 had duplex ultrasound studies of the extracranial carotid arteries. The most likely etiology of the stroke was determined using the criteria developed for the Harvard Cooperative Stroke Registry.

Results

Vascular causes of Wernicke’s aphasia were cerebral infarction in 38 patients (Group 1) and intracerebral hemorrhage in seven patients (Group 2); the remaining four patients (Group 3) had the onset of aphasia after aneurysm surgery following subarachnoid hemorrhage. These 49 patients (30 men and 19 women) ranged in age from 25 to 89 years; 46 were right-handed, two were left-handed, and handedness was unknown in one. Neuroimaging studies were
was presumed in a second elderly patient who had cerebral amyloid angiopathy in one patient and unknown in six patients. In addition to aphasia, 17 patients had minor motor deficits, four had hemisensory loss, and one had a homonymous hemianopsia. Twenty-two patients were hypertensive. CT revealed left temporal parietal infarction in 35 patients, left frontal infarction in one, and multiple subcortical infarctions in one; the other patient was not studied with CT. Arteriography was abnormal in 13 of the 16 patients studied; two had branch occlusions of the left middle cerebral artery (one with associated vasculitic changes), one had an embolus in the posterior parietal artery, three had complete left internal carotid artery occlusions, and seven had atherosclerotic 75% stenosis, ulceration, or both. Echocardiography was abnormal in 16 of the 28 patients studied, demonstrating a left ventricular thrombus in two, mitral valve vegetations in one, left atrial enlargement in 11, left ventricular hypertrophy or decreased left ventricular function in seven, and other valve abnormalities in 10 (aortic valve calcification in five, mitral annular calcification in four, aortic stenosis in one, and probable mitral valve prolapse in one). Many patients had several echocardiographic abnormalities. Embolic stroke was diagnosed in 21 of the 38 patients, 15 with cardioembolic strokes and six with large-vessel atheroembolic events (Table 1). Large-vessel atherothrombotic stroke was diagnosed in three patients (two men and one woman, mean age 65 years) with angiographic evidence of internal carotid artery occlusion. We were unable to establish a definite etiology in 13 Group 1 patients.

The seven patients in Group 2 (three men and four women) had intracerebral hemorrhage causing Wernicke’s aphasia. All were right-handed, and the mean age was 66 (range 38–89) years. Three also had a hemiparesis, and three had a homonymous hemianopsia. All seven patients had left temporal-parietal hemorrhages demonstrated by CT scan. Four patients underwent arteriography; one had a dural arteriovenous malformation (AVM) and the other three had avascular masses. The etiologies of intracerebral hemorrhage were cerebral amyloid angiopathy in two, dural AVM in one, hypertension in one, anticoagulant therapy in one, and unknown in two patients. Two patients had surgical evacuation of the hematoma. Cerebral amyloid angiopathy was pathologically documented in one patient and was presumed in a second elderly patient who had mild well-controlled arterial hypertension and recurrent lobar intracerebral hemorrhages.

The four patients in Group 3 (one man and three women) developed Wernicke’s aphasia after aneurysm surgery following subarachnoid hemorrhage, immediately after in three and 2 days after in the fourth. The mean age was 63 (range 55–76) years. Three patients were right-handed, and handedness was unknown in the other. Locations of the operated aneurysms were the left internal carotid artery in one, the internal carotid artery–posterior communicating artery junction in one, the giant intercavernous carotid artery aneurysm in one, and the left middle cerebral artery in one patient. Three patients had direct aneurysmal clipping. The patient with the giant intercavernous carotid artery aneurysm had carotid ligation. One patient who had repeat angiography after the onset of aphasia had vasospasm of the left anterior and middle cerebral arteries and occlusion of a posterior branch of the middle cerebral artery. The presumed causes of postoperative infarction in the other three patients were vasospasm in two and hemodynamic ischemia in one.

### Discussion

Aphasia classification may change in patients with acute stroke; thus, CT and neuropsychological testing should be done during the same period following a stroke to obtain the best correlation. In 45 of our patients CT scans revealed appropriately located lesions and were performed during the same period as the neuropsychologic testing. Both investigations were performed within 14 days after stroke onset, validating our clinical diagnosis of Wernicke’s aphasia.

<table>
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<th>Etiology</th>
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<tr>
<td>Cardiogenic</td>
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<tr>
<td>Recent myocardial infarction (2 with left ventricle thrombus on echocardiogram)</td>
<td>5</td>
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<td>Atrial fibrillation</td>
<td>5</td>
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<tr>
<td>Infective endocarditis</td>
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<td>Post-coronary artery bypass surgery</td>
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<td>Right-to-left intracardiac shunt (paradoxical embolism)</td>
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<tr>
<td>Carotid atherosclerotic disease</td>
<td>9</td>
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<td>L ICA occlusions (large-vessel atherothrombosis)</td>
<td>3</td>
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<tr>
<td>L ICA stenosis or ulceration (large-vessel atheroembolism)</td>
<td>6</td>
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<tr>
<td>Undetermined</td>
<td>10</td>
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<tr>
<td>Meningovascular syphilis with positive human immunodeficiency virus titer</td>
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<tr>
<td>Equivocal</td>
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<tr>
<td>Probable mitral valve prolapse</td>
<td>1</td>
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<tr>
<td>Following surgery involving resection of malignant neoplasm</td>
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L ICA, left internal carotid artery.
Approximately 20–25% of patients with acute stroke present with aphasia, and approximately 13% of these patients have Wernicke’s aphasia. Little has been written about the etiology of strokes that cause Wernicke’s aphasia. In our patients with stroke and Wernicke’s aphasia, 78% had cerebral infarction, 14% had intracerebral hemorrhage, and 8% developed aphasia after aneurysm surgery following subarachnoid hemorrhage. Of our patients with cerebral infarction, 55% had embolic infarction, 39% had embolic infarction from a cardiac source. The etiology of infarction was not determined in 26% of our Group 1 patients, probably reflecting the limitations of a retrospective study.

Tonokonogy reported that posterior aphasia (including Wernicke’s and transcortical sensory aphasias) occurs most often in patients with carotid stenosis, presumably atheroembolic, and rarely from carotid occlusion. He concluded that 20–25% had cardioembolic strokes; however, detailed cardiac evaluations were not provided. Caplan and Stein reported Wernicke’s aphasia to occur secondary to embolism (65%), thrombosis (15%), intracerebral hemorrhage (15%), and subarachnoid hemorrhage (6%) in 54 patients in the Harvard Cooperative Stroke Registry. These data were presented in tabular form, and the extent of evaluation and the basis of the diagnosis of etiology were not provided. These data are comparable to ours. Zaraspe-Yoo et al reviewed 103 patients with aphasia and reported embolism to cause Wernicke’s aphasia in 67%. In a retrospective review of 94 aphasic stroke patients, Harrison and Marshall concluded that 15% (four of 27 patients) with stroke secondary to cardioembolism developed Wernicke’s aphasia compared with 4.5% (3 of 67 patients) with stroke secondary to “carotid disease.” CT scans were not done in all patients, and it is difficult to draw conclusions from these data.

Intracerebral hemorrhage occurred in 14% of our patients with Wernicke’s aphasia. This incidence of hemorrhage is comparable to the data of Caplan and Stein from the Harvard Cooperative Stroke Registry. Previously, Tonokonogy and Stolyarova had reported intracerebral hemorrhage to be a rare cause of Wernicke’s aphasia, occurring in none of 25 post-mortem cases of aphasia resulting from intracerebral hemorrhage. Three of our seven Group 2 patients had a visual field defect, present in only one of our 38 Group 1 patients, possibly reflecting a more extensive lesion in those with intracerebral hemorrhage.

Wernicke’s aphasia occurred following a subarachnoid hemorrhage and aneurysm surgery in 8% of our patients, primarily secondary to vasospasm. Caplan and Stein reported that 6% (3 patients) developed Wernicke’s aphasia after subarachnoid hemorrhage.

In our series, there was a male predominance, primarily among patients with embolic cerebral infarction. Similar data have been previously reported. Our patients with Wernicke’s aphasia were older than patients with vascular Broca’s aphasia (unpublished data), which is in concordance with previously reported series. Some authors have believed that there is a change in speech organization or a change in cognitive function with increasing age, such that Wernicke’s aphasia could result in an older patient with a lesion in various areas in the language zone. A more plausible hypothesis is that older patients tend to have more posterior infarcts and subsequently develop Wernicke’s aphasia. With increasing age the proximal middle cerebral artery becomes more horizontal and may course inferiorly, possibly resulting in more posterior infarctions because of emboli occurring preferentially to the inferior division.

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