

Aphasia Resulting From Occlusion of the Left Anterior Cerebral Artery

Report of a Case With an Old Infarct in the Left Rolandic Region

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• A 62-year-old right-handed woman was admitted to the hospital with a cerebrovascular accident in the left hemisphere. She was aphasic as well as hemiplegic on the right. She had had a left cerebrovascular accident 18 years earlier that caused weakness in the right hand, but no grossly detectable speech problems. Autopsy disclosed an old infarct along the left Rolandic area, and a recent infarct in the territory of the left anterior cerebral artery. The clinical picture and pathologic changes are discussed in an attempt to relate the findings at autopsy to the recently developed aphasia.

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Aphasia as the result of injury to the mesial portion of the left frontal lobe is a recognized syndrome.^{1,2} Characteristically, patients suffering from such a lesion show very poor spontaneous speech output with preservation of comprehension and repetition, namely a transcortical motor aphasia. On the other hand, a lesion involving the left peri-Rolandic operculum leads to a conduction aphasia,³ also referred to as central aphasia, characterized by poor repetition in the face of preserved comprehension and spontaneous speech. The basis for the findings in conduction aphasia is presumed to be interruption of the arcuate fasciculus linking receptive to motor speech areas. Recovery is conceivable if connection between the sensory and motor areas for speech is reestablished via transcallosal pathways.

In this case report, a patient who

had an old infarct in the left Rolandic area showed no apparent communication deficit when reexamined years later, but became suddenly aphasic from a recent infarct within the distribution of the left anterior cerebral artery.

REPORT OF A CASE

A 62-year-old right-handed woman was admitted to George Washington University Hospital, Washington, DC, on Oct 10, 1975, with right hemiplegia and aphasia. She had been followed up at the hospital's outpatient clinic for diabetes mellitus, hypertension, and chronic heart failure. In 1957, she had suffered a stroke with right hemiparesis followed by a relatively good recovery. Details of that event are not available, but she resumed an independent life. A neurological examination in 1971 disclosed right-sided hyperreflexia with weakness, especially in the arm, and right-sided hemisensory impairment to touch, position, and vibration. She could walk unassisted and had no apparent communication deficit. Three days prior to admission, she was seen in the clinic because of increasing shortness of breath. Again, no

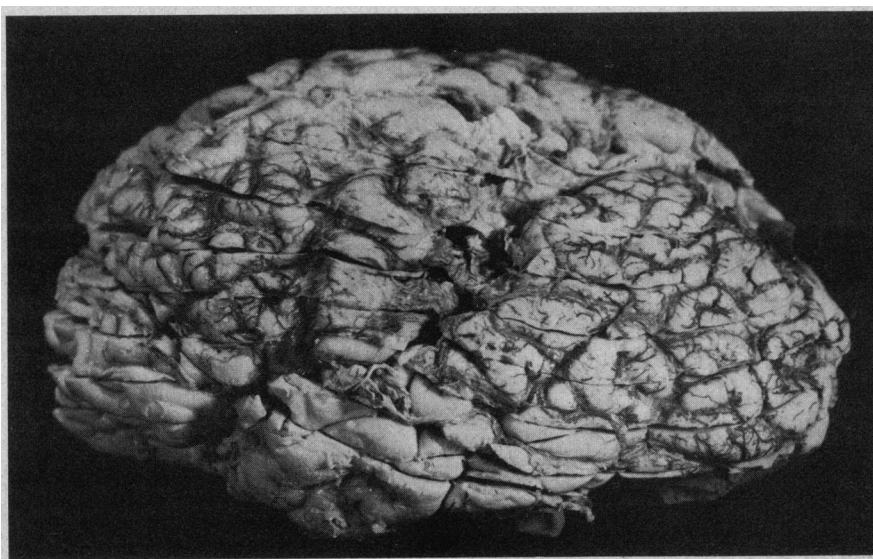
language deficit was observed. On the eve of her admission, she complained of nausea. The next morning, awakening, she was unable to converse or to move her right extremities.

Blood pressure was 130/60 mm Hg, with a pulse rate of 60 beats per minute, and a respiratory rate of 20/min. The lungs were clear, and cardiac examination showed increase in heart size, with a systolic murmur. Results of the remainder of the examination, except for the neurological findings, were unremarkable.

Neurological examination disclosed an alert woman with a profound speech defect, who perseverated in her speech and could not utter meaningful sentences. She was unable to follow simple commands. She had a right-sided hemiplegia, with hyperreflexia of 3+ /4 compared to 2+ /4 on the left side. A questionable right Babinski's sign was described. The visual fields were grossly intact. She was incontinent of urine.

A complete blood cell count showed normal hematocrit and hemoglobin levels, a normal platelet count, and a WBC count of 11.3×10^3 /cu mm, with 80% neutrophils. Prothrombin time and partial thromboplastin time were normal. Urinalysis

Fig 1.—Reconstructed external view of left cerebrum, showing collapse of postcentral gyrus and preservation of frontal and temporal areas.



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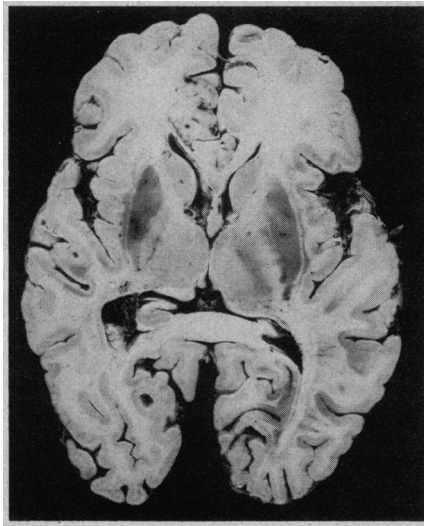


Fig 2.—Horizontal section of cerebrum, showing recent infarct involving corpus callosum in region of genu and rostrum, as well as adjacent mesial frontal lobe, with preservations of Broca's and temporal areas (top view).

showed 3+ sugar. Serum glucose level varied between 268 and 516 mg/dl. The electrolyte values were normal, the BUN level was 15 mg/dl, and creatine level 1.0 mg/dl. Serum cholesterol level was 377 mg/dl. Chest roentgenograms were normal. The ECG showed sinus bradycardia (55 beats per minute). Thyroid function studies gave normal findings.

One week following admission, the patient underwent a formal language evaluation. Her blood pressure was well controlled and there was no evidence of increasing cardiac failure. Her diabetes was difficult to control. She died suddenly on Oct 27, 1975. A complete autopsy was performed.

Aphasia Testing

The Boston Diagnostic Aphasia Examination was administered one week after her most recent stroke. She was alert and responsive throughout the evaluation, but made few attempts to initiate conversation. Her verbal output was nonfluent even when she was asked to repeat words or phrases. Considerable perseveration was noted. Groping was evident before she spoke, but all speech was clearly articulated, without any paraphasic errors. She had difficulty executing voluntary oral movements, particularly lateral and upward movement of the tongue.

She was able to correctly identify most pictures of objects and geometric figures and to point to body parts when named, but she had great difficulty in recalling the names herself. She had difficulty associating actions depicted in pictures, such as "running" and "smoking," when the word for that action was furnished by the examiner. She did carry out simple commands such as "make a fist" and "point to the ceiling," but could not go beyond the first part of multiple-step commands. She also

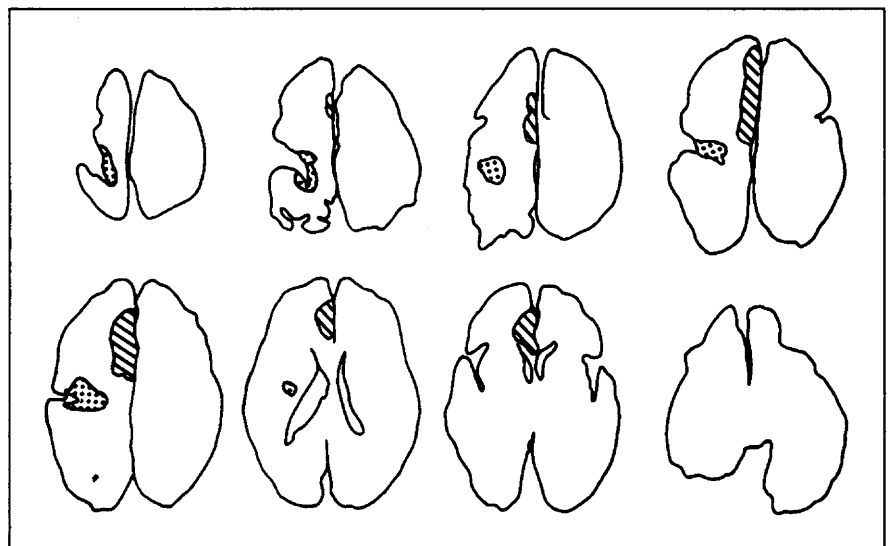
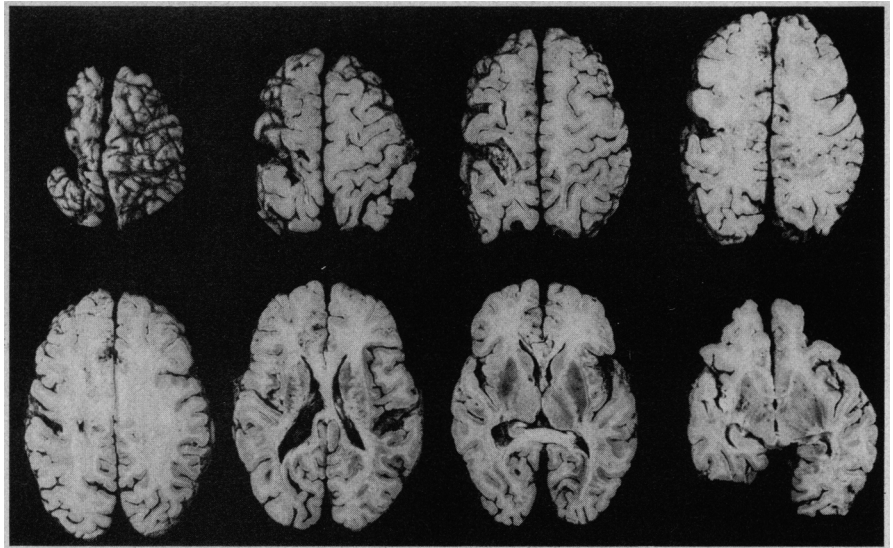


Fig 3.—Photograph (top) and diagram (bottom) of complete set of horizontal slices of cerebrum, showing location and extent of both recent (hatched) and healed (stippled) infarcts in left hemisphere.

showed left-right disorientation. When given a series of questions requiring a yes-no answer, she perseverated in saying "yes" to all questions such as "Is a hammer good for cutting wood?" or "Will a board sink in water?"

She managed to complete the nursery rhymes given to her, such as "Jack and Jill..." and "There was an old woman who lived in a shoe..." She could correctly repeat short phrases such as "you know how," "down to earth," and "go ahead and do it if possible," but failed on longer sentences such as "They heard him speak on the radio last night." When asked to complete a series such as the days of the week, the alphabet, or numbers from 1 to 20, she perseverated on one day, one letter, or one number. She could correctly associate most letters or words written in different scripts. When shown a series of words to associate with verbal counterparts, she made the correct choice in 60% of them. Despite her ability to pick out words,

she could not understand written words or sentences. She could not read aloud. When asked to write, she managed to correctly scribble her first name, using the left hand, but further testing had to be abandoned because of fatigue and frustration.

Autopsy Findings

The fresh brain weighed 1,150 g. The dura and sinuses were unremarkable. External examination of the fixed brain disclosed symmetrical cerebellar hemispheres; thin and translucent leptomeninges; normal cerebral veins; scattered atheromata of the large arteries with moderate narrowing of the lumina; and an almost complete occlusion of the left middle cerebral artery near its origin. An occlusion in the anterior cerebral arteries could not be found. A slightly swollen and softened cortex was found in the mesial and parasagittal area of the left frontal lobe, extending from near the frontal pole back to the level of the central sulcus. The

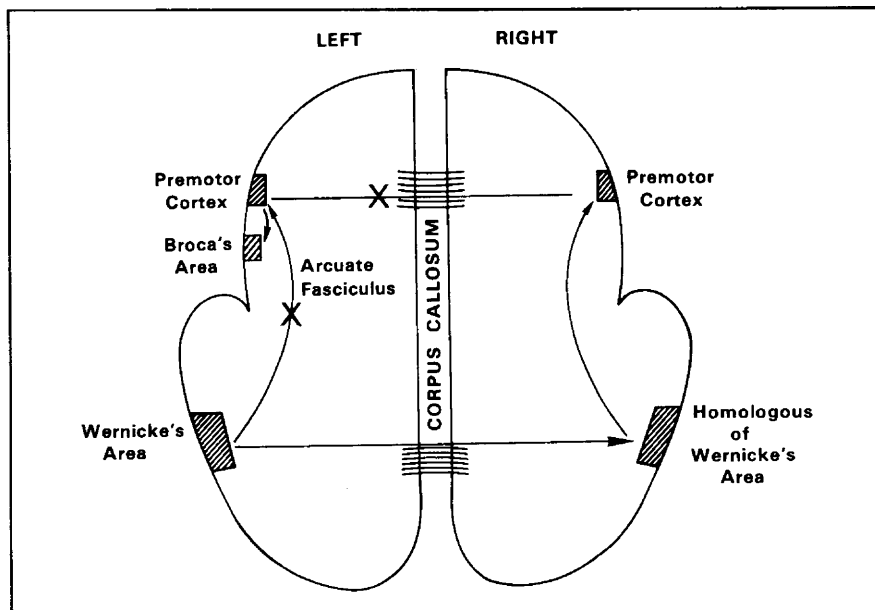


Fig 4.—Schematic diagram advanced to explain disconnection of receptive from expressive speech areas. X indicates lesions interrupting arcuate fasciculus and corpus callosum (modified from Geschwind¹⁹).

left postcentral gyrus showed collapse and softening extending from the Sylvian fissure up over the convexity to within 2 cm of the midline. The left inferior frontal gyrus (Broca's area), the left angular and supramarginal gyri, and the left temporal lobe including the planum temporale were normal (Fig 1).

Multiple horizontal sections of the fixed brain showed recent necrosis with coagulative softening, slight swelling, and punctate hemorrhages, involving parasagittal and mesial portions of the left frontal lobe within the distribution of the left anterior cerebral artery; the area of necrosis extended from near the frontal pole back to the central sulcus, and included cortex as well as subjacent white matter to a maximal depth of approximately 2.5 cm, not quite reaching the mediolateral midpoint of the left hemisphere itself. Broca's area with its subjacent white matter was not involved (Fig 2). This recent necrosis extended to include adjacent portions of the left side of the rostrum, genu, and anterior parts of the body of the corpus callosum (Fig 2). Microscopic sections from this region showed recent coagulative necrosis of all tissue elements with punctate perivascular hemorrhages, scattered gitter cells, rarefaction of neuropile, and a narrow rim of slight astrogliosis, consistent with a stage of evolution of approximately two to three weeks. The horizontal sections also displayed cystic rarefaction and collapse of the left postcentral gyrus, resulting in virtual disappearance of this gyrus through most of its extent; this cystic destruction extended into subjacent parietal white matter, involving extensively the region of the arcuate fasciculus as well as undercutting portions of the left precentral gyrus. The angular and supramarginal gyri with their subjacent white matter were intact, as was the temporal

lobe (Fig 2). Microscopic sections from this region showed the healed endstage of an old infarct, with disappearance, collapse, and cystic formation of neural parenchyma, a few remaining gitter cells, and surrounding astrogliosis, consistent with a stage of evolution of many years.

The location and extent of both infarcts is shown in the diagram and photograph of the complete set of horizontal brain slices (Fig 3).

Other neuropathologic features included almost complete stenosis of the left middle cerebral artery by atheroma, and atrophy of the left corticospinal tracts in the brain stem.

Other autopsy findings included marked coronary and aortic atherosclerosis; recent and healed subendocardial myocardial infarcts; multiple, bilateral pulmonary emboli; and a hemorrhagic cyst of the right thyroid.

In summary, the final neuropathologic diagnoses were as follows: recent infarct of the left cerebrum, in the distribution of the anterior cerebral artery, involving mesial and parasagittal frontal lobe and anterior corpus callosum; healed infarct within the territory of the middle cerebral artery, involving postcentral gyrus and subjacent white matter including arcuate fasciculus and white matter subjacent to the precentral gyrus; and marked atherosclerosis of intracranial arteries. Noteworthy is the preservation in the left hemisphere of Broca's area, Wernicke's area, and other parts of the temporal lobe.

COMMENT

Most reported cases of aphasia resulting from damage to the mesial part of the left frontal lobe have been caused by tumors,^{1,2,4-8} with improvement in many following excision.

Notwithstanding the fact that a detailed description of the aphasia is lacking in the majority of these reports, it is difficult to estimate the effects of edema and displacement of brain tissue by a neoplasm. Thus, some of Cushing's patients, reported by Chusid et al,⁴ showed dysnomia, dyslexia, dyscalculia, and even receptive aphasia (case R-B), while others had difficulty only in initiation of spontaneous speech. Reports of patients with dysphasia due to occlusion of the left anterior cerebral artery are relatively few.^{1,2,9-12} Most cases in the older literature also lack details of the aphasic syndrome. Most recently, Kyörney¹ described three patients with pathologic confirmation of occlusion of the anterior cerebral artery. The first was a 71-year-old woman who survived for 12 days after her stroke. Her speech output was reduced, but she demonstrated pronounced echolalia even without comprehending the content of what she said and could repeat long sentences with verbal and prosodic fidelity. At autopsy, in addition to the above mentioned infarct, there were three small areas of softening deep in the white matter of the left hemisphere. The second patient was a 64-year-old woman who had a left hemianopsia, then developed a right hemiplegia with global aphasia. At autopsy, her brain showed occlusion of the left anterior cerebral artery as well as old infarcts in both occipital lobes. Patient 3 was a 61-year-old man who developed a right hemiplegia of sudden onset with bilateral long tract signs and an expressive dysphasia with preservation of repetition and comprehension. He could name objects put in his left hand but not the right. He showed a strong tendency to repeat whatever was said to him. Eighteen months later, a right homonymous hemianopsia developed. At autopsy, in addition to softening in the territory of the left anterior cerebral artery, there were numerous small infarcts in the territory of the right anterior cerebral artery as well as in the insular portion of the left temporoparietal area. The parasagittal infarcts were pathologically older than the others and the author concluded that these were at the basis of the speech defect, prior to the onset of the right hemianopsia.

Rubens² described in detail two patients with sudden onset of right crural hemiplegia, aphasia, and incontinence and whose radionuclide brain scans, three weeks after onset, showed uptake in the distribution of the left

anterior cerebral artery territory. Both patients had an acute stage of aphonia lasting several days, followed by a prolonged period lasting several weeks during which there was marked diminution of spontaneous speech, with a remarkably intact ability to repeat even long and complex sentences. The first patient even showed an interesting inability to refrain from completing open-ended sentences and series of days or numbers. Both patients showed good comprehension and lack of paraphasic errors, and the second had persistent left-sided apraxia and tactile anomia.

It is clear from these cases that the cardinal features of the dysphasia associated with infarction in the territory of the left anterior cerebral artery are the following: a marked reduction in spontaneous speech, preservation of comprehension and repetition, and absence of paraphasia. Echolalia may be present. These findings are consistent with those described following ablation of parasagittal epileptic foci¹² and penetrating missile injuries in the same area,¹³ where the major deficit is initiation of speech. Stimulation of the parasagittal prefrontal cortex on either side induces vocalization and repetitive utterances or words.¹⁴

It is well known that injury of the posterior portion of the left inferior frontal gyrus (Broca's area) also results in paucity of spontaneous speech, with groping and monitoring behaviors. The difference with the mesial, parasagittal lesion is the preservation of repetition and absence of paraphasic errors. Goldstein¹⁵ used the term "transcortical motor aphasia" to describe this type of communication deficit while Luria and Tsvetkova¹⁶ called it "dynamic aphasia." It is important to mention

that occlusion of the main trunk of the left anterior cerebral artery may, indeed, produce a motor aphasia that is indistinguishable from Broca's aphasia. This is probably due to undermining of Broca's area by an infarct deep in the territory of Heubner's artery.^{1,17} This type of aphasia is usually transient, with recovery of spontaneous speech¹⁸ or persistence of a transcortical motor aphasia (TMA).

Although one cannot put too much weight on findings only one week after the onset of illness as they may be quite different from the outcome in a chronic stable patient, the case described in this report is nonetheless interesting in that some features of TMA were present whereas other findings did not fit with only one form of aphasia, probably because of the combined effects of the old and the more recent infarct. Those elements that fit a TMA syndrome are the reduction of spontaneous speech output, groping and monitoring behaviors, and lack of paraphasic errors. These findings can be explained by the fresh infarct in the territory of the left anterior cerebral artery. In contrast to the other features of TMA, this patient demonstrated impairment of repetition, although repetition was better than spontaneous speech. There was no echolalia, which is commonly found in TMA. It is thought that the discrepancy can be accounted for by the presence, in addition to the recent parasagittal lesion, of an old infarct undermining the parietal opercular area that interrupted the arcuate fasciculus connecting the sensory association cortex with the motor association area. It is unfortunate that no data are available on any language difficulties that may have accompanied the first stroke. It is tempting to speculate that a tran-

sient dysphasia may have developed and that recovery took place via transcallosal pathways, as shown in the diagram (Fig 4). Instead of being conveyed intrahemispherically from Wernicke's area to Broca's area, auditory information could be relayed through posterior callosal fibers to the right auditory association cortex, from there to the right premotor cortex, and finally to Broca's area via the anterior portion of the corpus callosum. The recently infarcted area, as can be seen in the brain sections, does in fact involve the anterior part of the corpus callosum. One would expect, then, to find difficulty in repetition of auditory material as well as dysnomia. This was indeed the case, for the patient could not name objects or figures, but could point to them (using the left hand) when the names were supplied. Her spoken language did not by any means match her comprehension. Thus she perseverated in answering "yes" to questions such as "Is a hammer good for cutting wood?" or "Does a board sink in water?" while, on the other hand, she did carry out at least simple commands, identify objects in pictures, and use tools such as a knife or fork to feed herself. The problem, then, was not primarily one of comprehension or of association of auditory stimuli with visual ones, but rather of transferring auditory and visual information to the motor speech area. It is obvious that her language deficits did not fit into any well-defined category. The combined effects of the two lesions resulted in a mixed pattern that is clearly different from what one would predict in a left anterior cerebral artery syndrome alone.

Craig Linebaugh and Penelope Myers reviewed the manuscript.

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