

The Aphasia Syndrome of Stroke in the Left Anterior Cerebral Artery Territory

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• **Two patients with transcortical motor aphasia are described. Both had infarcts in the distribution of the left anterior cerebral artery confirmed by computerized tomography (CT). The distinctive language abnormality, unusual EEG pattern, and nuclide and CT scan abnormalities all allow ready diagnosis of the syndrome of infarction in this distribution. The mechanism of the language disturbance is reviewed and implications for treatment and prognosis are discussed.**

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Captured under the general heading of nonfluent aphasia are several distinct syndromes. These include global aphasia and three additional syndromes in which auditory comprehension is relatively or totally preserved: aphemia (known also by other names, including pure word dumbness, but implying a limited disorder of speech secondary to a cortical lesion), Broca's aphasia, and transcortical motor aphasia. The first two have been well reviewed recently and their pathoanatomic bases made clearer.^{1,2} Transcortical motor aphasia is a clinical

entity less well recognized by neurologists and, we suspect, often placed under the general and inaccurate rubric of motor aphasia or expressive aphasia. It is characterized by nonfluent conversational speech, preserved auditory comprehension, and near normal repetition.³ A few reports document the nature of this disturbance⁴ and outline its clinical setting.⁵⁻⁷ We believe that the description of transcortical motor aphasia in one specific clinical setting, left anterior cerebral artery distribution infarction, is useful for several reasons: (1) this setting, while not common, offers a unique and easily recognized clinical picture; (2) computerized tomography (CT) may clearly reveal the lesion and either the EEG or the nuclide brain scan may present distinctive abnormalities; (3) the method of speech therapy used for other nonfluent aphasics may have to be altered; and (4) the prognosis for language recovery appears relatively good.

REPORT OF CASES

CASE 1.—A 56-year-old, fully right-handed man was admitted to a local hospital with a history of dizzy spells for several days and syncope that day. He had a history of alcohol abuse, diabetes mellitus, and hypertension. On examination, the patient was alert, with impaired speech and right-sided weakness. The results of a nuclide brain scan performed on the tenth hospital day were negative. The patient was transferred to Braintree Hospital for evaluation of speech and rehabilitation. Neurological evaluation performed 25 days after onset of illness revealed an alert,

cooperative, and oriented man. Carotid pulses were equal without bruits. Language examination results (see below) showed very limited speech output, marked difficulty in initiating speech, and perseveration of words; the auditory comprehension was normal. Repetition and series speech were dramatically preserved. There was moderate buccofacial apraxia. Right-left orientation and finger recognition were normal. There was a flaccid right hemiparesis of unusual distribution, maximal throughout the leg, moderate in the shoulder, and very mild in the hand, but facial movements were normal. There was mild bilateral and equal decrease in vibratory sense over both feet, and ankle jerks were absent. Otherwise reflexes were slightly brisker on the right; the toe response was flexor bilaterally. There was marked groping and a grasp reflex on the right side. An EEG obtained on day 30 revealed a left frontal and frontopolar semirhythmic slow wave focus. Computerized tomography scan revealed an infarct in the distribution of the left anterior cerebral artery (Fig 1). On follow-up evaluation at day 60, speech was improved, and buccofacial apraxia was minimal. Careful testing revealed a tendency to use a body part as an object in tasks of limb praxis, but only with the left hand. The pattern of hemiparesis was unchanged, but rigidity (paratonia) had developed in the right arm.

Formal Language Evaluation.—The Boston Diagnostic Aphasia Examination⁷ was first administered 25 days after onset. Spontaneous output consisted of short, effortful, telegraphic speech with perseveration and difficulty in initiating all phases. Occasional verbal paraphasias, often representing perseveration, were heard. Auditory comprehension was fully functional, with minimal breakdown for complex material. Naming was mildly impaired. A marked inability to generate

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word lists was in direct contrast to functional responsive naming. Repetition was well articulated and done well to long phrase lengths. Automatic speech required initial cues but was then completed with intact articulation. Reading aloud was minimally impaired, while reading comprehension of sentences was moderately impaired. With the nondominant left hand, writing was almost unintelligible and functional only for name and address. Buccofacial apraxia was prominent; nonverbal agility tasks were performed slowly and with effort. Reevaluation at 75 days, following two months of speech therapy, showed continuing marked difficulty in initiating speech, but frequent attempts were now made to produce spontaneous responses in a group setting. Most spontaneous productions were grammatically complete sentences, and speech was virtually fluent except for persistent hesitations, increased latencies, and word-finding pauses. Perseveration had decreased. Auditory comprehension was normal except for very complex material where failure may have represented concreteness rather than language failure. Responsive naming had improved and remained greatly superior to generation of word lists (eight items in 1½ minutes). Repetition was flawless and well articulated. Reading aloud was excellent. Reading comprehension was within normal limits. Writing was intact for short phrases with verbal cueing. Buccofacial apraxia was much decreased. Speech therapy had focused on compensation for anomia through use of appropriate circumlocutions, and alleviation of speech hesitancy through a strategy in which the patient was encouraged to continue and complete any self-initiated production. Group therapy served as a forum for arousing this patient's interest and motivation to communicate. It provided a structural setting for learning facilitating techniques that the patient later used in other settings.

CASE 2.—A 62-year-old woman was admitted to a local hospital with the abrupt onset of right-sided weakness and difficulty speaking. She had a long history of treated hypertension, a myocardial infarction in 1965, treated hypothyroidism, and mild diabetes mellitus. An EEG and nuclide flow study revealed a nonlocalized disorder of the left hemisphere. The patient was transferred on day 15 for rehabilitation. On examination, she was alert and cooperative. The left carotid pulse was diminished, but without bruit. Spontaneous speech was nonfluent with some echoing. Comprehension was normal. Confrontation naming was very good. Repetition, reading aloud, and serial speech were perfect with normal articulation (results of formal language testing are described below). There was mild buccofacial apraxia and a tendency to substitute body part as object bilaterally on praxis testing. There was a flaccid right hemiparesis of unusual distribution, dense in the leg, moderate in the proximal arm, and minimal in the distal arm and lower face. Stretch reflexes were brisker on the right; both toe responses were flexor. There was a strong right-sided

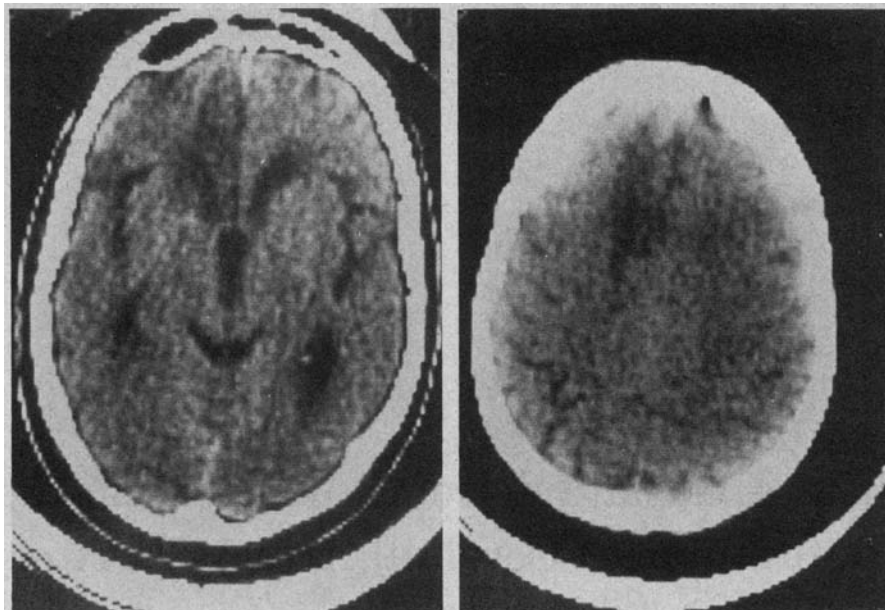


Fig 1.—Noncontiguous sections from nonenhanced computerized tomographic scan reveal large area of decreased density in medial left frontal region (case 1).

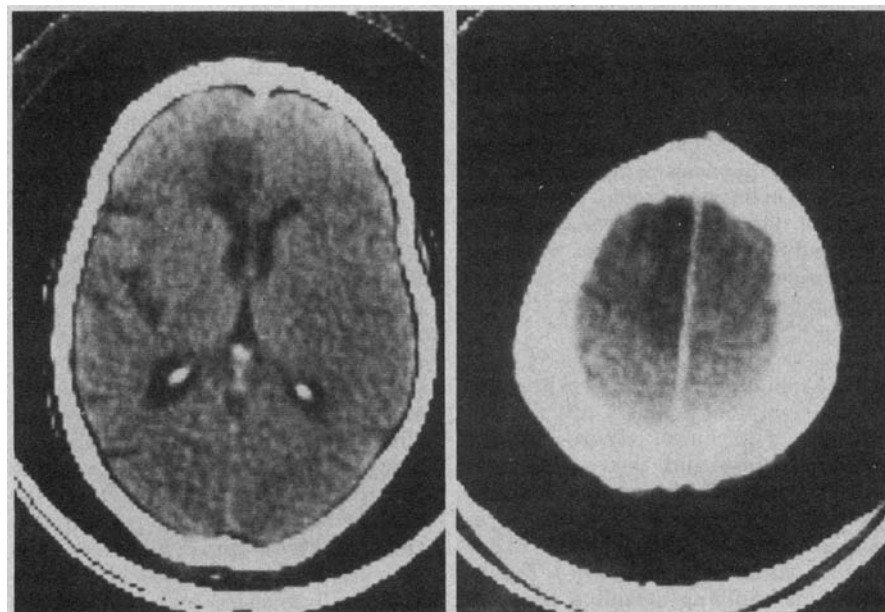


Fig 2.—Similar lesion in the medial left frontal region (case 2).

grasp reflex. Sensory examination results, visual fields, and optokinetic nystagmus were normal. An EEG obtained on day 22 revealed a striking frontopolar slow wave focus with spread in the left hemisphere. Computerized tomography scan revealed an infarct in the distribution of the left anterior cerebral artery (Fig 2). On reevaluation at day 90, speech and buccofacial praxis had improved. Right-sided limb praxis was normal, but clumsiness and posturing of the left hand in transitive praxis tests (eg, pretend to comb your hair) persisted. An attempt to use external sequential stimuli to improve speech initiation (eg, pointing to successive papers, rhythmically patting her hand, etc) was

unsuccessful. The right hemiparesis remained, affecting primarily the leg and the shoulder. Tone and sensation were normal and the right grasp reflex persisted.

Formal Language Evaluation.—The Boston Diagnostic Aphasia Examination⁷ was first administered 19 days after onset. Conversational speech was telegraphic, consisting of fragmented sentences and incomplete phrases (ie, "think, sink's overflowing") with severely impaired speech initiation. Auditory comprehension was intact, although with increased latencies of response and echoing on directional tasks. Responsive and confrontation naming were intact, in contrast to formulating word lists in which the patient produced

only four items in 1½ minutes. Repetition was excellent and was normally articulated, in contrast to the severely limited spontaneous output. Automatic speech was normal, although an initiating cue was required. Oral sentence reading was normal. Reading comprehension was within normal limits. Writing with the dominant right hand was limited to production of name and address. Buccofacial apraxia was present. Reevaluation 90 days after onset revealed persisting difficulty initiating conversational speech but with improved performance when cued. Speech was hesitant and effortful, often telegraphic, but well articulated. There was less perseveration and much less echoing. Occasional fluent, grammatically normal utterances were produced (eg, "She has an 18-year-old daughter who is in college"). The striking discrepancy persisted between the labored spontaneous speech and the normal, well-articulated repetition, reading aloud, serial speech, and confrontation naming. Ability to generate word lists had improved but was significantly poorer (12 items in 1½ minutes) than intact responsive naming skills. She could write short, grammatically correct sentences. During narrative writing, the patient spontaneously verbalized the words she was writing and had great difficulty producing words when asked to remain silent during the task. Speech therapy focused on extending the patient's use of complete sentences, and at discharge, the patient was capable of producing sentences in response to direct questions and in approximately 60% to 70% of spontaneous utterances.

COMMENT

The clinical syndrome of anterior cerebral artery occlusion has long been known⁹ and is described in modern textbooks on neurology.¹⁰ The deficits include hemiparesis with crural predominance, often flaccid, variable sensory loss in the lower extremity, left sided ideomotor apraxia and involuntary motor activity in the paretic arm, such as grasping and paratonia. Critchley's review of the necropsy-confirmed cases up to 1930⁹ establishes that a speech disturbance occurs with left anterior cerebral artery occlusion, but he describes the nature of this deficit only as "speech disturbances of various types." Modern texts expand little on this definition, and language disturbances seen in this setting generally remain unrecognized. Since Critchley's review, the pertinent literature on language disturbances following left frontal lesions that spare Broca's area has followed several independent threads.

Numerous reports characterize the alterations in verbal output secondary to manipulation of the supplementary motor area, that medial frontal pre-

motor region which lies entirely within the distribution of the anterior cerebral artery. Either spontaneous seizures arising from or electrical stimulation of either supplementary motor area produce repetitive vocalizations, speech arrest, and bilateral mouth movements.¹¹⁻¹⁶ Surgical ablation or manipulation of the supplementary motor area produces transient speech disturbances described as failure of initiation, "motor aphasia," or mutism.^{13,15,17} Permanent speech disorders include difficulty in initiating speech and anomia but are rare residuals of damage to this area.^{14,18}

Reports of aphasia following damage in the distribution of the left anterior cerebral artery have been infrequent. Poppen reported the results of anterior cerebral artery ligation during surgery for frontal tumors; in two patients, aphasia developed that was "practically complete" initially but apparently cleared rapidly.¹⁹ Guidetti described a patient whose left anterior cerebral artery was ligated to control bleeding from a ruptured aneurysm; postoperatively there was "severe motor aphasia" that resolved over several months.¹⁵ According to Goodglass and Kaplan,⁸ transcortical motor aphasia (TCMA) is marked by an absence of spontaneous speech, with difficulty in initiating speech and limited ability to name or to make brief responses. Auditory comprehension, reading comprehension, and reading aloud are relatively intact. "Repetition is remarkable in that it is prompt, well articulated, grammatically intact and free of difficulty of initiation which marks all other speech."

There are a few cases, labelled TCMA and caused by a single vascular episode, with which our cases may be compared. Atkinson⁶ briefly described one patient with TCMA following a large left frontal infarct but with scattered left parietal infarction as well and an atypical clinical syndrome. Rubens⁵ described two patients with TCMA and infarcts in the left anterior cerebral artery territory seen on brain scan. The clinical syndromes were typical except for the absence of left body apraxia in one patient. In another report, Rubens mentions nine patients with TCMA, three with anterior cerebral artery distribution infarcts and all but one of the others with large lesions in the left frontal lobe.¹ Von Stockert described a woman with a brain scan-proven infarction of the left medial frontal lobe.²⁰ She could not initiate speech although she could repeat. By three weeks after

onset, all language skills were within normal limits. In a larger study of localization in aphasia, Kertesz and McCabe briefly mention three patients with TCMA, one of whom had a left anterior cerebral artery distribution infarct.²¹ A recent report on TCMA includes one case with left medial frontal infarction.⁷ Only two of the reports include detailed language studies,^{5,20} and neither was published in a neurological journal.

Only a few well-described cases of TCMA with clinical findings typical of left medial frontal infarction have been previously reported. The neurologic findings include right crural paresis, involuntary motor activity of the right arm, and variable sensory loss in the right leg. Variable left ideomotor apraxia is seen. If infarction of the anterior 80% of the corpus callosum is complete, callosal signs should be seen.²² Our patients did not have these signs when examined by us. The language findings are poverty of spontaneous speech, slow initiation of speech, some capacity for brief responses and naming, relatively to completely intact comprehension and reading, and excellent, well-articulated repetition. A similar language syndrome may be seen in patients with large lesions elsewhere on the dorsolateral surface of the left frontal lobe anterior to Broca's area⁴ or, rarely, even with lesions including Broca's area,^{4,21} but the accompanying neurological signs are different. The combination of characteristic language and speech changes with other specific neurological findings is a powerful indicator of infarction in left anterior cerebral distribution. The EEG localization is atypical of carotid or middle cerebral artery occlusive disease and, while not well described in standard EEG texts,²³ appears strongly indicative of infarction in anterior cerebral artery distribution. Findings on brain scan may also be diagnostic.

Since first described by Lichtheim,²⁴ a rich literature has developed on the features of TCMA suggesting mechanisms for its production in left frontal lesions, some completely outside of the perisylvian speech area. Lichtheim postulated an interruption of fibers to the motor speech area from a concepts center. This center was not specifically localized but rather represented the combined action of much of the brain converging on Broca's area. Bastian²⁵ theorized that partial damage to the motor speech area caused the syndrome by increasing the threshold for motor speech performance.

Goldstein²⁶ considered the problem

of transcortical aphasias in great depth, suggesting two separate mechanisms for producing the syndrome. In one, partial damage to the motor speech area altered the speech threshold, but in this case, repetition and automatic speech, while better than spontaneous, would not be so clearly normal. A second form of TCMA did not represent a language deficit so much as an "impairment of the impulse to speak at all." Not a global impairment of all verbal movements, as intact repetition, oral reading, etc proved, but an inhibition in initiation of motor activity maximal in speech functions produced by focal left frontal lesion, which may also partially involve the motor speech areas. These possibilities may account for some cases of TCMA in which the lesion affects the frontal convexity in or near Broca's area, but our patients had lesions far removed from the perisylvian region.

Left frontal lesions in nonaphasic patients produce another important related effect. Milner²⁷ demonstrated a deficit in "verbal fluency" in patients following partial frontal lobectomy for control of epilepsy. In addition to diminished spontaneous speech, these patients have markedly diminished ability to form a word list, when compared with patients with

lesions elsewhere in the brain outside the language region. Botez and Barbeau²⁸ in a review of clinical and experimental work concluded that the medial frontal supplementary motor area was part of the more primitive system involved in a "starting mechanism" of speech but also in the maintenance of speech fluency apropos Milner's observations. This deficit was seen in our patients.

Finally, studying cases of trauma or cases not completely described or localized, often with tumors, Luria²⁹ has extracted three apparently independent deficits from the performance of patients with TCMA. In short, there is a combined failure to form propositional units, to maintain fluency and to initiate speech. The deficits stem from large left frontal lesions similar to those seen in our patients. In addition, a lesion in the left anterior cerebral artery distribution may specifically damage a primitive system of speech organization, magnifying the impairment in spontaneous speech production. The surgical literature described earlier indicates that this latter speech disturbance is reversible.

While the implications for speech therapy remain uncertain, traditional therapeutic techniques based on verbal cueing and repetition and newer

therapies based on intoned repetition³⁰ are of limited value.³¹ They address a function that is preserved and performed almost automatically in these patients. Luria has suggested providing an external sequence to speech by relating speech to some other sequential movement, such as pointing to squares or patting one's leg.²⁹ This helped his patients in initiation and maintenance of speech. We attempted this and were unsuccessful, but external rhythmic stimulation remains a plausible approach for these patients.

Regardless of therapy, there are data suggesting that the prognosis of TCMA may be relatively good,^{5,22,32} although some patients have persistent difficulty in initiating and maintaining narrative speech, even if responsive speech is normal. In summary, the deficits produced by infarction in the distribution of the left anterior cerebral artery produce a distinct clinical syndrome. The findings on EEG and nuclide and CT scans may confirm clinical localization. Therapy for the speech disorder may require individualized alterations; the overall prognosis is favorable.

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