

# The Resolving Stroke and Aphasia

## A Case Study With Computerized Tomography

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• A 39-year-old man suffered an intracerebral hemorrhage in the region of the left internal capsule deep to Wernicke's area. The location of the lesion was confirmed by computerized tomography (CT) performed two days postictally. Two weeks after admission, the Boston Diagnostic Aphasia Examination (BDAE) disclosed Wernicke's aphasia. We hypothesize that the hematoma exerted pressure on Wernicke's cortical area, thus causing the resulting Wernicke's aphasia at that time. A CT scan three months later showed absorption of the hematoma, with a residual low-density lesion deep to Wernicke's area, in the region of the arcuate fasciculus. At that time, BDAE testing disclosed a mild conduction aphasia. Serial CT scanning combined with discriminating clinical evaluation of aphasia provides a valuable opportunity for study of the processes underlying stroke resolution and aphasia.

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We have been investigating the relationship between types of aphasia, as demonstrated with the Boston Diagnostic Aphasia Examination (BDAE), and site of anatomic lesion inferred from computerized tomography (CT) in stable stroke patients.<sup>1,2</sup> Recently, we have studied

a patient whose aphasia syndrome and CT findings changed concomitantly, with improvement, over a three-month period.

### REPORT OF A CASE

A 39-year-old right-handed man was admitted to Stanford University Medical Center, Palo Alto, Calif, after the sudden onset of headache, right hemiplegia, and aphasia. The history was negative for hypertension, diabetes, or previous neurological symptoms. The patient was alert. All pulses were normal; there were no bruits. Blood pressure was 160/106 mm Hg; a sinus rhythm was present. Neurological examination demonstrated a right homonymous hemianopsia, right central facial weakness, and inability to protrude the tongue. A right hemiplegia and hyporeflexia were present, with decreased sensation to pain and touch on the same side. A weak right extensor plantar response was elicited.

Computerized tomography performed

two days after admission (Fig 1) demonstrated a small intracerebral hematoma in the region of the left caudate nucleus, with compression of the adjacent frontal horn of the lateral ventricle. A second, larger hematoma was present in the region of the posterior limb of the left internal capsule, with compression of the atrium and body of the left lateral ventricle. Cerebral angiography disclosed a mass effect in the left internal capsule. No aneurysm, arteriovenous malformation, or extracranial vascular disease was demonstrated. The patient was transferred to the Palo Alto Veterans Administration Hospital for convalescence and rehabilitation nine days after onset.

### Initial Aphasia Testing

The BDAE utilizes a test profile (Fig 2) to aid in classification of aphasia.<sup>3</sup> Initial testing carried out two weeks after admission disclosed a moderate to severe fluent Wernicke's aphasia. Spontaneous speech was fluent (items 1 through 6, Fig 2) and marked with multiple verbal paraphasias.

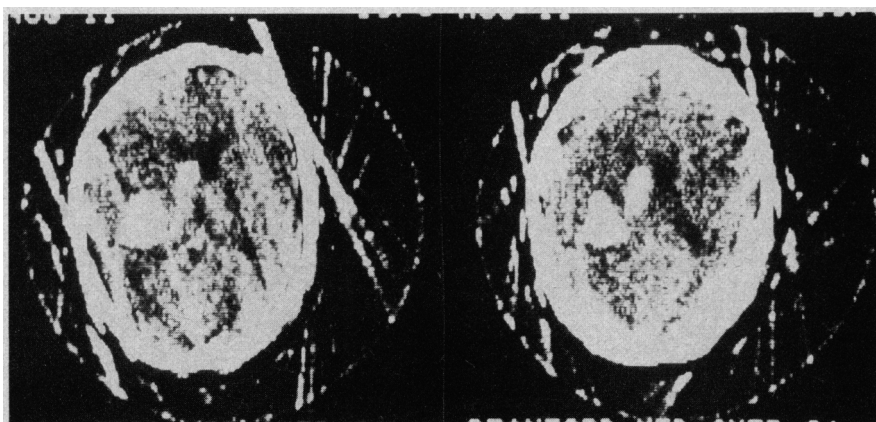


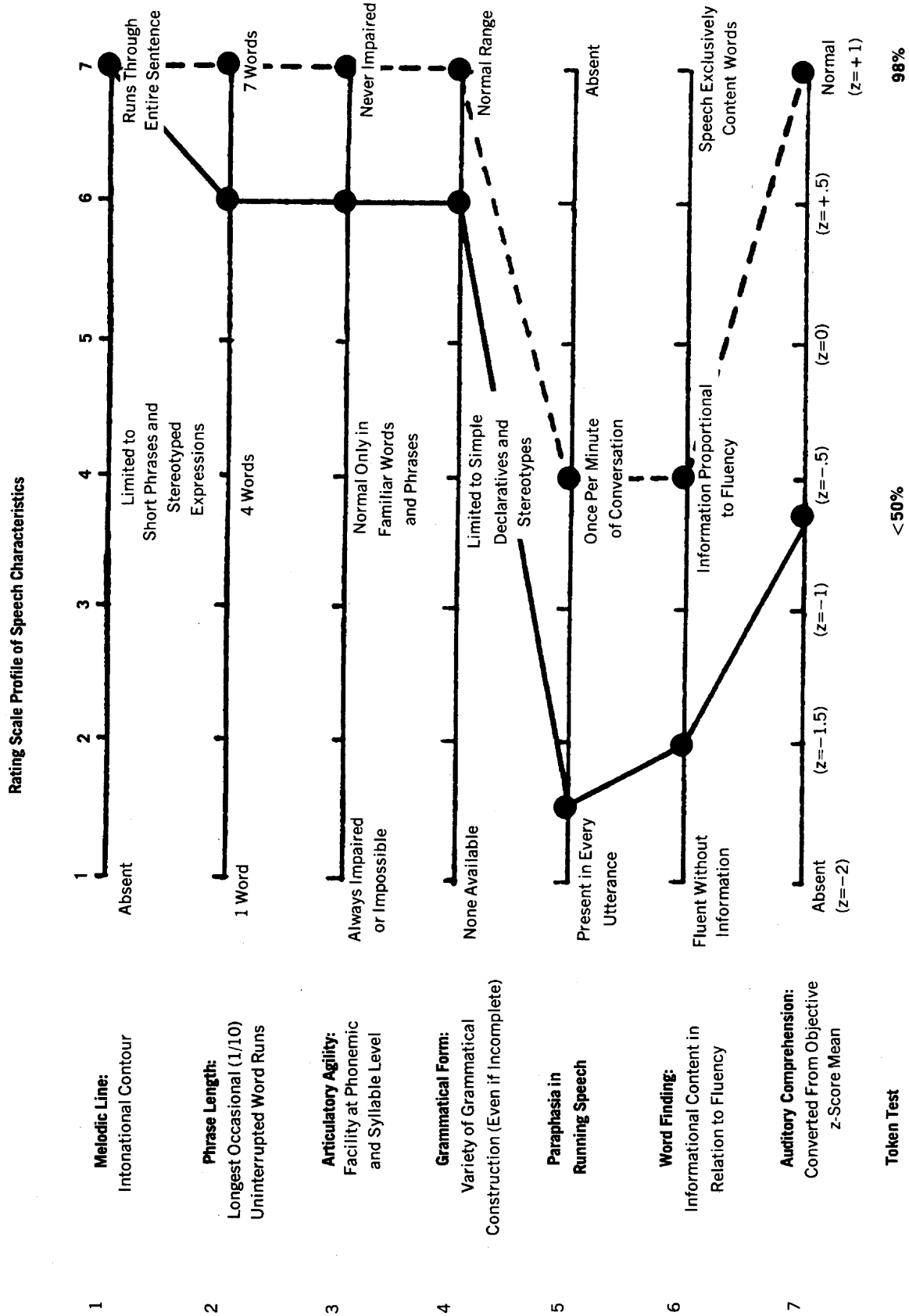
Fig 1.—Computerized tomographic scan two days after stroke. Small hematoma near head of left caudate nucleus. Larger hematoma in region of posterior limb of internal capsule, anterior to atrium of left lateral ventricle (left) and lateral to body of left lateral ventricle (right).

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Fig 2.—Boston Diagnostic Aphasia Examination profile used to classify aphasia patients as to aphasia type. Items 1 through 6 reflect characteristics of patient's spontaneous speech output item 7 reflects language comprehension ability. Solid line indicates patient's initial aphasia profile (Wernicke's aphasia), broken line, follow-up aphasia profile (conduction aphasia).



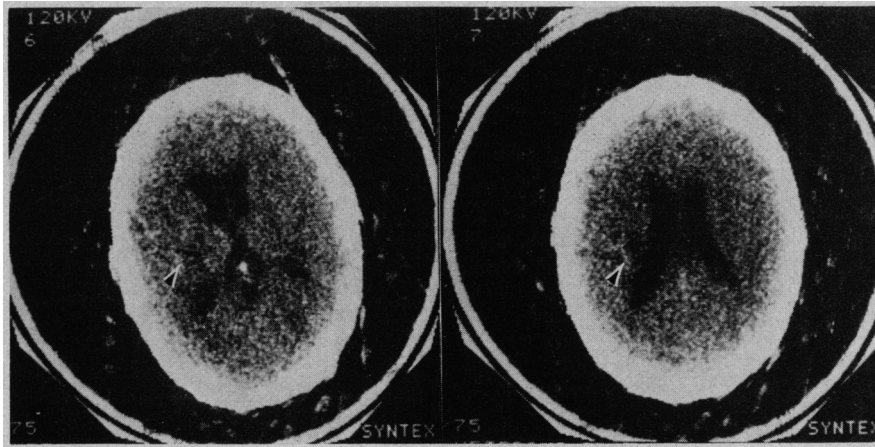


Fig 3.—Computerized tomographic scan three months after onset. Both hematomas have resorbed. Slight enlargement of frontal horn of left lateral ventricle (left). Small low-density lesion deep in posterior portions of arcuate fasciculus anterior to atrium (left scan arrow) and lateral to body of left lateral ventricle (right scan arrow).

Language comprehension was moderately to severely impaired at  $-6$  on the BDAE auditory comprehension z-score (item 7, Fig 2) and less than 50% on the Token Test.<sup>4</sup> These test results are graphically displayed as the solid line in Fig 2. They are compatible with a fluent Wernicke's aphasia.<sup>3</sup> Word and phrase repetition were severely impaired, reading was moderately impaired (seven of ten sentences were comprehended correctly in silent reading), and writing was severely impaired.

After six weeks, the patient was able to walk with a cane, and was discharged with residual weakness of the right extremities.

#### Subsequent Examination

Computerized tomography and BDAE testing were done again three months after onset of aphasia. The CT scan demonstrated resorption of the hematomas (Fig 3). Slight enlargement of the frontal horn of the left lateral ventricle was noted, with a small low-density area, anterior to the atrium and lateral to the body of the left lateral ventricle, that did not extend to the cortical surface.

The BDAE testing disclosed a mild conduction aphasia (Fig 2). Spontaneous speech was substantially improved and near normal (items 1 through 6, Fig 2) with occasional literal paraphasias. Language comprehension was normal, at  $+1$  on the BDAE auditory comprehension z-score (item 7, Fig 2) and 98% on the Token Test. Due to major improvement in auditory comprehension, the BDAE aphasia profile was now compatible with a fluent conduction aphasia (broken line, Fig 2).<sup>3</sup> Although word and phrase repetition had also

improved, there remained a persistent problem with number and low frequency of occurrence phrase repetition. For the standard conduction aphasia test phrase, "No ifs, ands, or buts," he responded, "Nos ifs. . . ." When asked to repeat "1776" he answered "776." Reading ability had improved (nine of ten sentences were comprehended correctly in silent reading) and writing had also improved.

#### COMMENT

In recent CT studies of stable stroke patients with aphasia,<sup>1,2</sup> the patients with Wernicke's aphasia had temporal lobe lesions located anterior to the atrium of the left lateral ventricle that involved cortical as well as deep structures. In our patient, in whom initial BDAE testing demonstrated a typical fluent Wernicke's aphasia, CT done two days after the stroke (Fig 1) showed a large intracerebral hematoma in the posterior limb of the internal capsule (anterior to the atrium of the left lateral ventricle). The mass effect of this intracerebral hematoma might well have exerted pressure laterally onto Wernicke's area, thus causing the characteristic deficits of the usually cortically located lesion.

At the time of BDAE testing three months later, the patient had mild conduction aphasia (graphic profile in Fig 2, broken line). Conduction aphasia is commonly observed with lesions in the arcuate fasciculus, a deep pathway that extends from the supramar-

ginal gyrus and Wernicke's areas to Broca's area.<sup>3</sup> The four patients with conduction aphasia studied with BDAE and CT in our previously reported studies<sup>1,2</sup> showed this type of aphasia to be primarily associated with subcortical lesions in the posterior portions of the arcuate fasciculus (anterior to the atrium, and lateral to the body of the left lateral ventricle). In this patient, CT disclosed a small subcortical lesion anterior to the atrium and lateral to the body of the left lateral ventricle (the residua from the previous hematoma), consistent with involvement of the posterior portions of the arcuate fasciculus (Fig 3). Also present was a slight enlargement of the left frontal horn (the residua from the previous caudate hematoma). The relative contribution of the caudate lesion to the aphasia is not known.

As serial CT scans confirmed the resolution of the hemorrhage and mass effect, concomitant BDAE testing demonstrated a change in aphasia type from severe Wernicke's to mild conduction. Computerized tomography, when correlated with discriminating clinical evaluation, can provide a challenging opportunity to correlate function with anatomic changes. Properly used, such studies may be found to have predictive value in recovery from aphasia.

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