

It is of interest that the two patients we have reported were women and both showed endocrine problems. One had been on ovulatory suppressants since age 16, until her death at age 22 with FMD. The second patient had a long history of chronic functional uterine bleeding, subsequently leading to a hysterectomy. Even though we too are unable to define the etiology of this disease process in our patients, nevertheless, the evidence that hormonal influences may have played a part seems to us to be more than circumstantial.

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Aphasia Outcome in Stroke: A Clinical Neuroradiological Correlation

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SUMMARY Fourteen aphasic patients with acute onset of thromboembolic cerebrovascular insults demonstrable by angiography or radioscintigrams who were available for long-term follow-up have been studied. Their aphasia evolution was compared with acute angiographical and radioisotopic findings, and the lesions shown by follow-up computerized axial tomography (CT).

Angiographical site of occlusion, evidence of early reopening of occluded vessels, and radioisotopic flow asymmetries including the "hot-stroke" luxury perfusion failed to correlate with aphasia out-

come. Radioisotopic static images were more helpful by depicting lesion location and number but lacked the definition seen on the CT scan.

The long-term CT scan by showing the size, location and number of lesions had a good correlation with aphasia outcome. Those patients with large dominant hemisphere involvements, either one large or many smaller lesions, fared poorly while those with lesser lesions did better. Bilateral lesions, at times evasive clinically, helped to account for significant aphasia residuals.

APHASIA, or failure of communicative skills in symbolic language, has traditionally been studied by careful clinical observations, followed by pathological correlations. Indeed, the main historical proponents of aphasia localization, Broca and Wernicke, pioneered this approach in the second half of the nineteenth century.¹⁻³ Recently, investigators have been interested in the in vivo anatomical correlation of

aphasias using neuroradiological tools. Benson and Patten⁴ have used radioisotopic images in localizing aphasias. They have disparaged the role of cerebral angiography, but others have sought to study aphasia with this modality.⁵ Most recently, Mohr et al.^{6,7} have made use of computerized axial tomography (CT) to more clearly define aphasia syndromes.

Compilation of serial aphasia evaluations, acute angiograms and radioscintigrams, and the late follow-up CT scans was done. The aphasia evaluation and the neuroradiological information then were correlated in an attempt to find in vivo prognostic parameters.

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Methods

Fourteen patients who had aphasia as a result of cerebral infarction and who had dominant-hemisphere lesions identifiable either on angiography and/or radioscinigraphy during their acute hospitalization were studied acutely and followed for at least eight months. Aphasia tests were done acutely and again when the patient's condition had stabilized. The final follow-up aphasic evaluation was coupled with a CT scan for clinical anatomical correlation.⁸

Radioscinigraphy was performed using the gamma camera and an intravenously injected bolus of ^{99m}Tc-sodium pertechnetate. An initial flow study was obtained in the vertex position to compare the relative radioisotopic perfusion of the two hemispheres. Delayed static images were obtained to seek evidence of localized radioisotopic uptake. All patients except No. 3 had serial acute and follow-up radioscinigrams using the serial study method previously discussed.⁹ Patient No. 3 had only follow-up studies.

Angiography was performed to study the extracranial and intracranial vasculature of the involved hemisphere. In 11 of 14 patients, including those with carotid occlusion, the contralateral hemisphere also was studied. Repeat angiography within a month of the acute study was performed in six of these patients. The angiographical site of maximal pathology was taken as the largest observable vessel significantly involved (i.e., extracranial or intracranial carotid artery, middle cerebral artery [MCA], main trunk or MCA branches).

The acute radiological findings of Patients Nos. 7, 8 and 13 were mentioned in a prior publication⁹ while those of Patients Nos. 2 and 11 were discussed in another article.¹⁰

The CT scans were done on an EMI-scanner (Emitronics Inc.) without contrast enhancement. CT scans were obtained in all the patients at the time of final communication evaluation. Lesions were graded as small, moderate or large without knowledge of the patient's aphasia outcome.

Communication skills testing involved a tape-recorded verbal battery, parts of the Minnesota Test for Differential Diagnosis of Aphasia (MTDDA),¹¹ and the Porch Index of Communicative Ability (PICA)¹² and was performed in the first month of the illness and again at final follow-up. All the patients received serial speech therapy as well. The tape-recorded sample included tests for oral facial apraxia (cough, blow, stick out your tongue . . .), verbal apraxia (say "catastrophe, artillery, Methodist, Episcopal . . ."), dysarthria ("count to 20"), and overall oral communication ability ("describe what happened to bring you to the hospital . . .").

The MTDDA is divided into five sections comprising tests for auditory disturbances, visual and reading disturbances, speech and language difficulties, visuomotor and writing problems, and disturbances in numerical relations. Reading and writing subtests were utilized to evaluate higher level reading comprehension and written communication abilities.

The PICA test is designed to assess and quantify certain verbal, gestural and graphic abilities. The index is made up of 18 subtests: eight gestural, four verbal and six graphic. We were able to complete scores for the acute and follow-up stages of illness in 12 patients. Patient No. 7 could not be tested, while Patient No. 9 had only follow-up PICA scores.

On the basis of the PICA, parts of the MTDDA and the

tape-recorded sample, a judgment was made by a certified speech pathologist as to descriptive categories and degrees of severity. The descriptive categories included: (1) auditory comprehension — does the patient receive, perceive and associate meaning with what he hears? (2) Reading comprehension — does the patient receive, perceive and associate meaning with what he reads? (3) Oral communication — is the patient's speech fluent (free flowing, normal rate and prosody) or nonfluent (limited, halting, hesitant, abnormal prosody)? Is he dysarthric (faulty articulation due to impairment of the muscles of articulation)? Does he have oral apraxia (loss of memory for oral nonverbal motor movements)? Does he have verbal apraxia (loss of memory for verbal motor movements)? Is he mute or speechless? (4) Is global aphasia present (severe receptive and expressive impairment in all modalities)? (5) Does he have anomia (loss of power to name objects or to recall names)? (6) Does he demonstrate paraphasia (sound or word reversals, substitutions and/or omissions)? (7) Is he able to perform the motor act of writing? (8) Written communication — is he able to communicate meaning through writing?

The degrees of severity of impairment are as follows: (1) extremely severe (81% to 100%), (2) severe (61% to 80%), (3) moderately severe (41% to 60%), (4) moderate (21% to 40%), (5) mild (11% to 20%), and (6) minimal (1% to 10%). Aphasia outcome was graded as good (minimal-to-mild impairment), fair (moderate-to-moderately severe impairment), or poor (severe or greater impairment).

Results

Table 1 lists the patient's clinical course with the acute and follow-up communication skills evaluations. Table 2 categorizes the parameters of aphasia outcome, angiograms, radioscinigrams and CT scans for each patient.

The aphasia outcome was judged good in six patients (Nos. 1, 6, 9, 10, 12, and 14), fair in three (Nos. 7, 8 and 11), and poor in five (Nos. 2, 3, 4, 5, and 13). Good outcome implied adequate communication skills in social relationships.

Angiography defined the site of vascular occlusion. Of the two patients with acute internal carotid occlusion (Nos. 4 and 10), one made good language recovery (No. 10) while the other did poorly (No. 4). Of the patients with main trunk MCA occlusion (Nos. 1, 2, 3 and 6), two (Nos. 2 and 3) did poorly and two (Nos. 1 and 6) had good aphasia recovery. Five patients had secondary MCA trunk or branch occlusions (Nos. 8, 9, 11, 12 and 14). Two of these patients had only fair language recovery (Nos. 8 and 11), while the other three did well. Good collateralization, as evidenced by retrograde filling of most of the Sylvian triangle branches seen on the lateral angiogram and the absence of mass effect (Patients Nos. 1, 6 and 10), correlated with a better outcome. Patient No. 4 had sparse collaterals and a slight mass effect, and fared poorly. Dramatic reopening of the majority of occluded vessels seen on repeat early angiography¹⁰ in Patients Nos. 2, 11 and 14 as contrasted with essentially unchanged vascular occlusions on early repeat angiography in Patients Nos. 1, 6 and 10 did not correlate with aphasia outcome. Patient No. 2 did poorly and Patient No. 11 had a fair recovery, while the others had good outcomes.

Radioscinigrams consisted of a flow study and then a delayed static study. These studies fell into three categories:

TABLE 1 *Clinical Course With Acute and Follow-Up Communication Skills Evaluation of Patients*

Clinical	Communication
<p>1: 52-year-old R-handed Caucasian salesman, 15 mo. post-total recovery from an acute R subdural hematoma, on prophylactic A/C because of an aortic valve prosthesis, was admitted drowsy with a mild R hemiparesis, R ignoral and global aphasia. During hospitalization, communication and focal signs markedly improved except for increased upper extremity weakness. At follow-up, he had mild gait difficulty and a weak proximal R upper extremity with better distal movements; he felt he could no longer work as a salesman.</p>	<p><i>Acutely:</i> Moderate nonfluent output with moderate comprehension lack in all modalities; severe impairment of written communication ability; moderate oral apraxia. <i>Follow-up</i> (14 mos.): Minimal speech hesitations and word-finding difficulties with normal comprehension; mild-to-moderate written communication deficit; minimal oral apraxia.</p>
<p>2: 62-year-old R-handed Caucasian office worker with a remote MI and transient hypertension collapsed at work; R hemiparesis (greater in arm), R-sided inattention, poor R gaze, L Horner's syndrome and aphasia were noted. Subacutely, a benign-appearing L carotid bifurcation plaque was removed. A few months later a reversible L-hand paresis occurred. At follow-up, he had marked R-hand paresis; was independent in self-care, but limited due to his language deficit.</p>	<p><i>Acutely:</i> Very severe nonfluency with mild-to-moderate comprehension difficulty in all modalities; no written communication ability; severe oral apraxia. <i>Follow-up</i> (14 mos.): Very severe nonfluent output with moderate reading comprehension ability; mild auditory comprehension difficulty; severely impaired written communication ability; severe oral apraxia.</p>
<p>3: 27-year-old R-handed black banker had a dense R hemiparesis and aphasia several weeks after chest trauma. Three months prior a reversible R hemiparesis and fluent paraphasic aphasia had correlated with L MCA posterior branch occlusions. A similar, but not medically observed, episode had occurred 4 years before. He has recovered without gross paresis and is independent in self-care, but is very limited by his aphasia.</p>	<p><i>Acutely:</i> Severe global aphasia with low output and total comprehension loss; severe oral apraxia. <i>Follow-up</i> (3½ years): Severe nonfluent output with a moderately severe auditory comprehension lack, but with better relative ability to follow written commands; persistent severe deficit in written communication ability; mild oral apraxia.</p>
<p>4: 50-year-old R-handed Caucasian woman with rheumatic heart disease, atrial fibrillation and a prior valvulotomy was found at home with a flaccid R hemiplegia and global aphasia. Intermittent L ptosis was noted. She made minimal improvement during her 1-mo. acute hospitalization and subsequently.</p>	<p><i>Acutely:</i> Severe global aphasia with only simple phrase perseveration and severe failure of all modality comprehension; severe oral apraxia. <i>Follow-up</i> (2¼ years): Severe nonfluent output with a moderately severe comprehension lack; written comprehension markedly impaired; severe oral apraxia.</p>
<p>5: 69-year-old R-handed black man, with arteriosclerotic heart disease, had an acute language disturbance after awakening. No focal motor or sensory deficits were demonstrable. There was a vague history of prior strokes without residual. On follow-up, he was able to live at home with much support from his family. Persistent dense language disability remained with a suggestive R-sided field defect on double simultaneous stimulation.</p>	<p><i>Acutely:</i> Severe fluent output with multiple paraphasias, neologisms (jargon speech); no comprehension ability; severe written impairment; no oral apraxia. <i>Follow-up</i> (8 mos.): Severe fluent aphasia with marked paraphasia, little (if any) evidence of self-monitoring; minimal comprehension ability; severe written communication deficit.</p>
<p>6: 48-year-old R-handed Caucasian woman (community leader and housewife), 5 years post-MI, had acute onset of R-sided weakness, sensory and field deficits and aphasia while shopping. Initial bilateral limb apraxia was seen. At follow-up, she walked with an R hemiparetic gait and had a plegic R hand; she needed slight assistance in ADL but had no apraxia. An intensive total rehabilitation program was instituted.</p>	<p><i>Acutely:</i> Very severe nonfluent output with intact comprehension in all modalities (could do receptive vocabulary tests well); no written communication ability; severe oral apraxia. <i>Follow-up</i> (13 mos.): Mild nonfluent output with hesitations; mild word-finding problems; normal comprehension; normal written communication ability; no oral apraxia.</p>
<p>7: 32-year-old L-handed Caucasian man (superior drafting student) with rheumatic heart disease, chronic atrial fibrillation and mitral valve prosthesis had acute onset of speech loss, R field cut and mild R hemiparesis. Four years prior, a L hemiparesis and dysarthria episode fully resolved. At follow-up, there were no motor or visual deficits; he was independent in ADL, but unable to do his prior work or communicate beyond a simple level.</p>	<p><i>Acutely:</i> Global aphasia, uttering garbled sounds; no auditory comprehension; moderately severe reading comprehension loss; could follow some simple written commands; no written communication skill; severe oral apraxia. <i>Follow-up</i> (2½ years): Remained without recognizable speech output; cortically deaf; moderate reading comprehension ability; can respond to some written phrases; moderate writing impairment (writes short telegraphic phrases); severe oral apraxia.</p>

R = right, L = left, A/C = anticoagulation, MCA = middle cerebral artery, mo. = month, MI = myocardial infarction, ADL = activities of daily living, SLE = systemic lupus erythematosus.

TABLE 1 (continued)

Clinical	Communication
<p>8: 52-year-old R-handed Caucasian man (computer scientist) with mild hypertension had transient headaches and diplopia. These fully subsided, but a few hours later, global aphasia and R hemiparesis developed and resolved quickly (within hours) to a R-sided ignoral and jargon aphasia. At follow-up, he was fully independent in ADL, and could partake in sports, but was limited by his communication deficit from doing computer programming. He complained of poor R-hand sensory discrimination and paresthesias.</p>	<p><i>Acutely:</i> Severe fluent jargon speech with moderately severe auditory and reading impairment; very severe written communication impairment; no oral apraxia. <i>Follow-up (3 years):</i> Moderate fluent output deficit with paraphasias; moderate reading comprehension loss with somewhat better auditory comprehension; moderate-to-severe written communication impairment.</p>
<p>9: 65-year-old R-handed Chicano housewife, an insulin-dependent diabetic with a history of multiple prior MIs, lost her speech in the midst of a bingo game. She was aphasic and extinguished her R side tactically and visually. At follow-up, she had regained a normal existence but complained of visual distortions.</p>	<p><i>Acutely:</i> Severe fluent meaningless output with poor auditory and reading comprehension; written communication severely impaired; no oral apraxia. <i>Follow-up (14 mos.):</i> Speech had minimal paraphasias; normal all-modality comprehension; normal written communication.</p>
<p>10: 30-year-old R-handed Caucasian housewife, 20 days post-caesarean delivery of her fourth child, had R hemiparesis, R field cut, L Horner's syndrome and aphasia after awakening. At follow-up, there was slight R-hand clumsiness in fine movements; she functioned normally as a housewife, mother and part-time office worker.</p>	<p><i>Acutely:</i> Moderate nonfluent output with mild comprehension loss in auditory and reading spheres; moderate written communication lack; moderate oral apraxia. <i>Follow-up (2 years):</i> Minimal speech hesitations; occasional mild auditory comprehension loss (subjective only); rare mild written language difficulty; no oral apraxia.</p>
<p>11: 61-year-old R-handed Caucasian man had R paresis, L gaze deviation and muteness 3 days following coronary artery surgery. Past history included a remote MI with persistent angina, and a traumatic R-leg amputation. At follow-up, intact motor functions enabled him to do his hobby (cartoon drawing) well; was able to perform ADL (receives disability support); his tongue deviated slightly to the R and nonfluent speech persisted.</p>	<p><i>Acutely:</i> Very severe (mute) nonfluent output; mild auditory comprehension lack (can follow simple commands); moderate reading comprehension loss; severe written communication loss; severe oral apraxia. <i>Follow-up (10 mos.):</i> Severe nonfluent impairment with only rare carrier phrases; normal auditory comprehension; moderate reading comprehension ability; mild written communication impairment; severe oral apraxia.</p>
<p>12: 18-year-old R-handed Caucasian man with known SLE had sudden onset of R-sided numbness, weakness and dysphasia. A week prior, transient L body weakness had occurred; two weeks prior he had transient painless gross hematuria. Examination revealed R hand and central face weakness, decreased R body sensation, nonfluent aphasia, and a new cardiac murmur. At follow-up, he had regained normal motor function with intact handwriting and sports ability; he had resumed his prior activity levels but had difficulty with higher level abstraction; he felt he could no longer grasp college mathematics.</p>	<p><i>Acutely:</i> Mild nonfluent output with intact comprehension; minimal written language difficulty; no oral apraxia. <i>Follow-up (8 mos.):</i> Mild nonfluent output; intact comprehension; trace of written communication difficulty.</p>
<p>13: 24-year-old R-handed Chicano woman with rheumatic heart disease and mitral valve prosthesis had an acute R hemiparesis, R-sided ignoral and global aphasia. She improved from dense to moderate motor deficits during her 2-week hospitalization. At follow-up, she had fair fine motor movements and intact gait, was able to help out in caring for young siblings, but was markedly limited by communication and psychological problems.</p>	<p><i>Acutely:</i> Severe global aphasia with muteness and all-modality comprehension loss; no written language ability; severe oral apraxia. <i>Follow-up (3 years):</i> Moderate nonfluent speech with short phrases only; moderate comprehension ability (both auditory and reading); moderately severe written language impairment; persistent moderate oral apraxia.</p>
<p>14: 58-year-old R-handed Caucasian man (industrial designer) with electrical evidence of prior MIs had sudden onset of R arm paresthesias and speech difficulty. Examination revealed an aphasia and R lower quadrantanopia and moderate R hand clumsiness. At follow-up, he had no focal motor or visual deficit; he could draw well but appeared to have difficulty obtaining work; he was fully independent.</p>	<p><i>Acutely:</i> Moderately nonfluent output; mild comprehension difficulty with higher level abstractions; moderate written language difficulty; mild-to-moderate oral apraxia. <i>Follow-up (11 mos.):</i> Minimal speech hesitations; intact comprehension; mild-to-moderate written language difficulty; no oral apraxia.</p>

TABLE 2 *Categorization of Aphasia Outcome, Angiograms, Radioscintigrams and CT Scans for Each Patient*

Pt. no., age, sex	Oral aphasia outcome	Angiography	Radioscintigraphy	CT scanning
1, 53/M	Good	L MCA main trunk occlusion, persistent; good collaterals	Decreased L flow, persistent; slight diffuse L uptake	Small L deep frontoparietal caudate area lesion
2, 63/M	Poor	L MCA main trunk occlusion, reopening later with branch occlusions	"Hot-stroke," L frontoparietal and anterofrontal uptakes	Large L frontoparietal, small L anterofrontal lesions
3, 30/M	Poor	L MCA main trunk occlusion; fair collaterals	Decreased L flow, persistent; static—not done	Moderate-sized L temporal, frontoparietal lesion; small R posterotemporal lesion
4, 52/F	Poor	L distal cervical ICA occlusion; sparse collaterals; mass effect	Decreased L flow, persistent; large L parietotemporal uptake	Large L holotemporal, posterofrontal, anteroparietal lesion
5, 69/M	Poor	Not done	Symmetrical flow, L temporoparietal uptake	Large L posteroparietal-superior temporal lesion
6, 49/F	Good	L MCA main trunk occlusion, persistent; good collaterals	Decreased L flow, persistent; L posterofrontal, temporoparietal uptake	Moderate-sized L posterofrontal temporoparietal lesion
7, 35/M	Fair*	Not done	"Hot-stroke," L mid-to-posteroparietal uptake	Moderate-sized bilateral symmetrical temporoparietal lesions
8, 55/M	Fair	L parietal branch occlusions and stain	"Hot-stroke," L parietal uptake	Moderate-sized L posteroparietal lesion
9, 66/F	Good	L posteroparietal branch occlusion and stain	Symmetrical flow, L temporoparietal and R occipital uptakes	Moderate-sized L posteroparietal and R occipital lesions
10, 32/F	Good	L ICA occlusion above ophthalmic artery, persistent; good collaterals	Decreased L flow, persistent; L posteromedial frontal uptake	Small L caudate head area lesion
11, 61/M	Fair*	L MCA posterior trunk occlusion, reopening later with branch occlusions	"Hot-stroke," L anteroparietal and R posterior fossa uptakes	Moderate-sized L posterofrontal, anteroparietal lesion
12, 18/M	Good	L posterofrontal occluded vessel and stain	"Hot-stroke," L frontoparietal uptake	Small L anteroparietal temporal lesion
13, 27/F	Poor	Not done	"Hot-stroke," L parietotemporal uptake	Large L temporoparietal lesion, ? R parietal lesion
14, 58/M	Good	L anteroparietal branch occlusions with stain, later normal	"Hot-stroke," L parietal uptake	Small L midparietal lesion

*Nonoral only.

M = male, F = female, L = left, R = right, MCA = middle cerebral artery, ICA = internal carotid artery. Patient's age is at follow-up.

(1) the persistent relative decreased dominant hemispheric arterial phase flow with positive static images seen in Patients Nos. 1, 3, 4, 6 and 10, (2) the symmetric flow, positive static images seen in Patients Nos. 5 and 9, both having dominant-hemisphere posteroparietal lesions, and (3) the "hot-stroke" phenomenon⁹ of transiently relative increased radioisotopic perfusion to the side of infarction seen in Patients Nos. 2, 7, 8, 11, 12, 13 and 14. Category 1 had Patients Nos. 1, 6 and 10 with good aphasia outcome and Patients Nos. 3 and 4 with poor outcome. In the symmetric flow group (Category 2), Patient No. 5 did poorly while Patient No. 9 did well in communication outcome. The "hot-stroke" group (Category 3) yielded two good aphasia outcomes (Nos. 12 and 14), three fair outcomes (Nos. 7, 8 and 11), and two poor outcomes (Nos. 2 and 13). Evidence of multiple lesions on the acute static images, at times clinically unsuspected, was matched with a poor outcome in Patient No. 2, a fair outcome in Patient No. 11 and a good aphasia recovery in Patient No. 9.

The CT scan lesions in our patients were all demonstrated in long-term follow-up. The times ranged from eight months to three and one-half years after the acute aphasia-producing episode.

Multiple lesions were demonstrated in four patients (Nos. 2, 3, 7 and 9) with poor outcome in Patients Nos. 2 and 3, fair in Patient No. 7 and good aphasia recovery in Patient

No. 9. Patient No. 11, who had a posterior fossa lesion besides his left hemispheric lesion, demonstrable by radioscintigrams, had only the latter lesion seen on his CT scan. Large single CT scan dominant-hemisphere infarcts were seen in Patients Nos. 4, 5, and 13 (fig. 1) with poor outcomes in all. Small single CT scan lesions in the dominant hemisphere were seen in Patients Nos. 1, 10, 12 and 14 with good aphasic outcomes in all.

The patients (Nos. 5, 8 and 9) who initially clearly fell into the fluent aphasia categorization of Benson and Geschwind¹³ all had at least moderate-sized left posteroparietal lesions (fig. 2). This correlated with their acute static radioscintigrams. Patient No. 9 also had a right occipital infarct. Those patients with initial severe global aphasia (Nos. 3, 4, 7 and 13) had either bilateral lesions (Nos. 3 and 7), temporal in part, or large lesions (Nos. 4 and 13). Three of these patients had poor outcomes while Patient No. 7 (fig. 3) made a fair nonoral recovery. Three patients with acute output difficulties much greater than comprehension deficits (Nos. 10, 12 and 14) had small dominant lesions while two patients (Nos. 6 and 11) in this language category had moderate-sized dominant-hemisphere lesions. Four of these patients (Nos. 6, 10, 12 and 14) made good communication skills recoveries, while Patient No. 11, who also had a right posterior fossa lesion on radioscintigrams, made only a fair nonoral recovery.

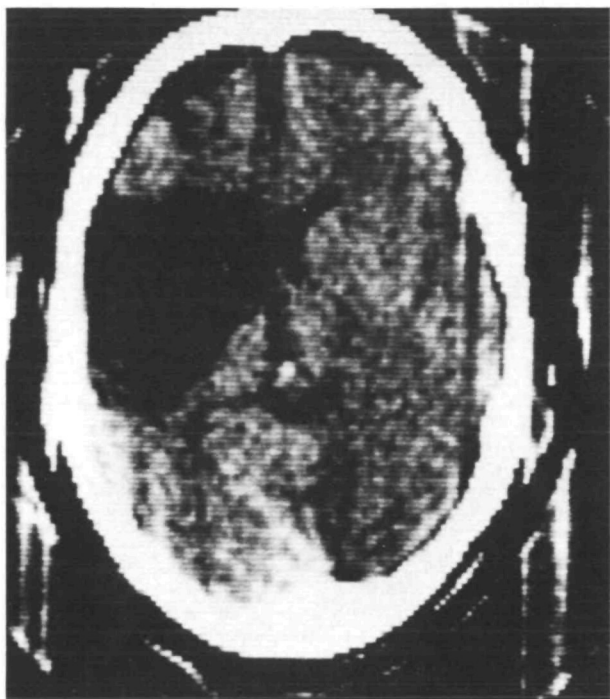


FIGURE 1. CT scan (Patient No. 4) demonstrating a large left posterofrontal, holotemporal, anteroparietal infarct resulting from acute internal carotid occlusion. The patient had severe aphasia at two and one-fourth years' follow-up.



FIGURE 2. CT scan (Patient No. 9) demonstrating a moderate-sized left posteroparietal infarct resulting from middle cerebral artery branch occlusions. The right occipital lesion is seen on a more inferior slice. The patient made a good recovery from fluent aphasia at 14-months' follow-up.

Comments

The patients reported were selected from neurological admissions with acute thromboembolic cerebrovascular episodes, causing aphasia and neuroradiologically defined lesions. Long-term follow-up availability was a prerequisite for inclusion in the study. Thus, the study is biased in excluding patients with rapidly progressive diseases and patients with non-demonstrable lesions radiologically, and in being confined to cerebral infarction patients. Different emphasis may occur in other aphasia etiologies such as primary hemorrhage⁷ or head trauma.^{14, 15}

Despite attempts at subjective and objective evaluations, aphasic recovery encompasses many variables difficult to evaluate. In the good recovery group, no patient felt fully returned to his or her prior state. Hesitancies, exacerbated by emotion, slowness in transforming thoughts to words and, in some, difficulty with formerly mastered concepts were noted. Alternately, the families of the poor outcome patients often felt that their communication abilities were better than the low testing scores implied. This is probably an effect of environmental, gestural and other non-verbal cues used in familiar home situations. Thus, the ratings of aphasia outcome must be considered relative at present.

Angiography either by demonstrating the site of occlusion or by showing early reopening of occluded vessels¹⁰ did not have prognostic value in aphasia outcome. This is in agreement with the conclusions of other investigators.^{4, 16, 17}

The radioscintigram, our other acute neuroradiological parameter, also had difficulty in correlating with language outcome. The flow asymmetries, whether relatively

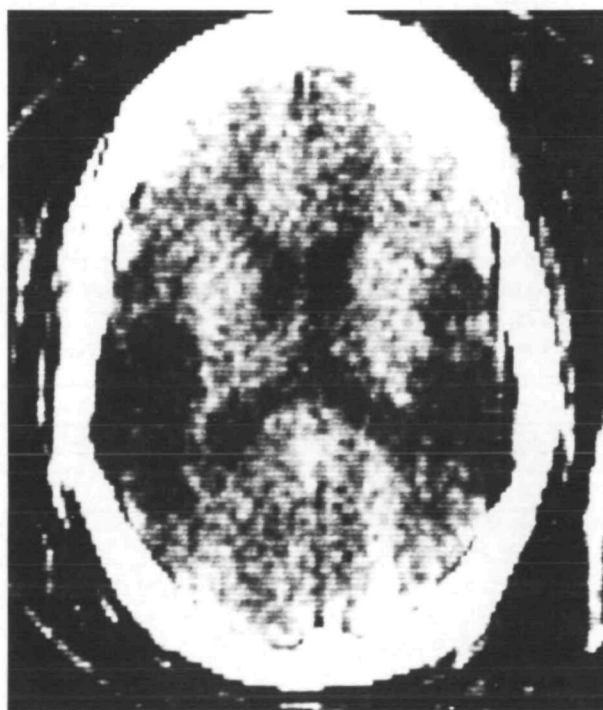


FIGURE 3. CT scan (Patient No. 7) demonstrating symmetrical moderate-sized temporal parietal infarcts presumed secondary to prosthetic mitral valve generated emboli. The patient made a fair nonoral aphasia recovery at two and one-half years' follow-up.

decreased or increased to the infarcted hemisphere, were not useful. The static images were helpful in showing gross size and number of lesions but lacked clear lesion definition. Both angiograms and radioscintigrams are not yet fine enough techniques to elucidate and map clearly persistent regional circulatory defects.

The CT scan has the advantage of presenting relative density measurements that correlate well with the altered tissue seen pathologically.¹⁸ All the lesions demonstrable at long-term follow-up in our patients were low density ones with fairly distinct margins. These lesions are consistent with stationary cerebral infarcts.⁸ Clinically many of the multiple lesions seen on the CT scans were unsuspected either because they were masked by the profound dominant-hemisphere deficit or because they were in relatively silent areas. Large single CT dominant-hemisphere lesions correlated with poorer outcomes than did small single lesions. The CT scan thus was useful in showing the size, location and number of lesions.

In the patients with fluent aphasia, the CT scan showed predominant left posteroparietal lesions. These lesions correlated with the acute phase static radioscintigrams which themselves were in agreement with a prior radioisotopic study of fluent aphasias.¹⁹ Bilateral, in part temporal, or large single dominant-hemisphere lesions correlated with the acute severe global aphasia states. These patients had generally poorer aphasia recoveries. Alternately, relatively small-sized dominant-hemisphere CT lesions correlated with acute expressive much greater than receptive difficulties and a more benign aphasia resolution.

Of interest are the two patients with small CT scan lesions in the region of the caudate head (Nos. 1 and 10) who clinically presented with moderate dominant-hemisphere deficits. They both had relative decreased hemispheric perfusion radioisotopically and significant persistent major vessel occlusion angiographically, left MCA trunk in Patient No. 1 and distal left internal carotid artery in Patient No. 10. Both patients made good symbolic language recoveries.

The CT scan thus most clearly parallels the traditional clinical pathological aphasia methodology with the great added advantage of in vivo examinations.¹⁸ The correlation with aphasia outcome here seems most promising. A larger sample and serial scanning will be necessary to confirm this initial impression.

Other factors such as education, intellect, age, moti-

vation and therapeutic intensiveness also may be very significant parameters in aphasia outcome. These are difficult to fully assess and were beyond the scope of the present report. Studying these factors together with the pertinent clinical and CT scan findings in a large group may help to elucidate their relative importance.

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