Broca's area aphasias:
Aphasia after lesions including the frontal operculum

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Article abstract—We report 9 cases of aphasia following lesions in the region of the left frontal operculum. It is not possible to capture their variety of clinical manifestations with the simple labels of "Broca's aphasia" or "Broca's area aphasia." Analysis of the breakdown of various components of speech and language in these cases suggests that the operculum, lower motor cortex, and subjacent subcortical and periventricular white matter contain critical parts of different language systems. These systems can be independently impaired. There are several common language syndromes that follow damage that includes the left frontal operculum. These syndromes reflect the effects of the direction and extent of the lesion in the various language systems.

NEUROLOGY 1990;40:353-362

Broca's aphasia is a syndrome of disordered language characterized by effortful speech production, impairments in melodic line and articulation, semantic and phonemic paraphasias, telegraphic or at least shortened phrase lengths, reduced and abnormal grammatical form, and a comprehension deficit most apparent when tested with material dependent upon understanding syntax.[1] For decades, controversy has surrounded the psycholinguistic validity of this syndrome and its pathologic anatomy. Mohr[2] has summarized this often-trod ground. In the past 15 years attention has shifted from classical Broca's aphasia, and, beginning with the reports and reviews of Mohr[2,3] and colleagues,[4] current thinking has come to 2 revised ideas about classical Broca's aphasia: 1st, that the pathologic anatomy, in keeping with the multidimensionality of the language disturbance, usually involves damage to several areas across a large portion of the left hemisphere, including but never limited to frontal operculum;[4,5] and 2nd, that it is usually a disorder of incomplete recovery from more severe aphasia.

Perhaps in part because of these revised notions and perhaps because of improved neuroimaging of small lesions, there have been more recent reports of smaller lesions of the frontal operculum (Brodmann's areas 44 and 45) and adjacent structures. These reports have defined much more restricted clinical-anatomic relationships than those of classical Broca's aphasia. Analysis of these relationships suggests that there are 3 distinct aphasia profiles following lesions of the posterior, inferior frontal region.[6-10] One profile is focused on impaired speech and language initiation; among classical diagnoses, this is approximately synonymous with transcortical motor aphasia and occurs with lesions centered on the operculum. A 2nd profile is focused on disturbed articulatory function; it follows lesions in the lower motor cortex immediately posterior to the operculum. A 3rd profile includes some elements of the 1st 2 plus some additional elements of classical Broca's aphasia. It follows lesions that include both the operculum and lower motor cortex as well as pathways deep to rolandic cortex.

This partition into related but differing case types is important for 2 reasons. First, it partially clarifies the semiologic muddle of Broca's area aphasia.[2] Second, it defines, through examples, the principle that all aphasic syndromes emerge from the anatomic propinquity and convergence of different functional systems. We herein report an analysis of cases with lesions in operculum and lower motor cortex that further illuminates the architecture of the functional systems involved in "Broca's area aphasia" as well as "classical Broca's aphasia."

Methods. *Patient selection.* Patients were selected by clinical and CT criteria (table 1). Only patients with single, left hemisphere ischemic strokes were included. All cases had a CT demonstrating lesion of frontal operculum,[11] or lower motor cortex,[12] or the immediately subjacent subcortical white matter. Patients with any clinical history or CT evidence for right hemisphere lesion were excluded. Nine cases met these combined criteria. None of the patients had been previously reported. We did not evaluate any of the patients in the acute phase of their illnesses; all were referred to the Boston VAMC for speech and language therapy.

Speech and language assessment. The Boston Diagnostic Aphasia Examination (BDAE)[13] was used in 8 patients; 1 patient (no. 6) received a very detailed clinical examination that was approximated to a BDAE profile. Apraxia was assessed

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Supported in part by USPHS Grant NS 06209 and by the Research Service of the Veteran's Administration.

Received November 10, 1987. Accepted for publication in final form July 17, 1989.

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Table 1. Demographics, neurologic and language exams

	Patient								
	1	2	3	4	5	6	7	8	9
Gender/Age	M/68	M/61	M/61	M/54	M/64	F/40	M/53	M/66	M/64
Weeks post-onset to 1st aphasia exam	8	3	5	5	18	20	8	9	20
BDAE output ratings									
Articulatory agility*	7	5	5	2	1	1	3	4	Unratable
Melodic line (prosody)*	7	4	4	1	2	1	4	2	Unratable
Phrase length*	7	7	7	7	7	1	7	7	Unratable
Grammatical form*	7	7	7	7	7	1	7	7	Unratable
Paraphasias	Semantic only	Phon > Sem	Phon > Sem	Phon > Sem	Phon > Sem	Unratable	Phon > Sem	Phon > Sem	Unratable
BDAE comprehension									
Complex-ideational (maximum score, 12)	6	6	9	7	5	6	12	6	4
Overall (Z-score)	0.3	0.3	0.5	0.5	0	0.5	1	0.5	0.3
BDAE writing									
Words to dictation (maximum score, 12)	13	10	13	10	13	13	15	14	9
Narrative (rating, 1-4)	1	1	1	1	0	2	4	0	1
Ideomotor apraxia									
Buccofacial	Moderate	Moderate	Severe	Moderate	Severe	Severe	Mild	Severe	Severe
Limb	None	None	Mild	None	Mild	Mild	None	None	Mild

* BDAE ratings are 1 (severely impaired) to 7 (normal).

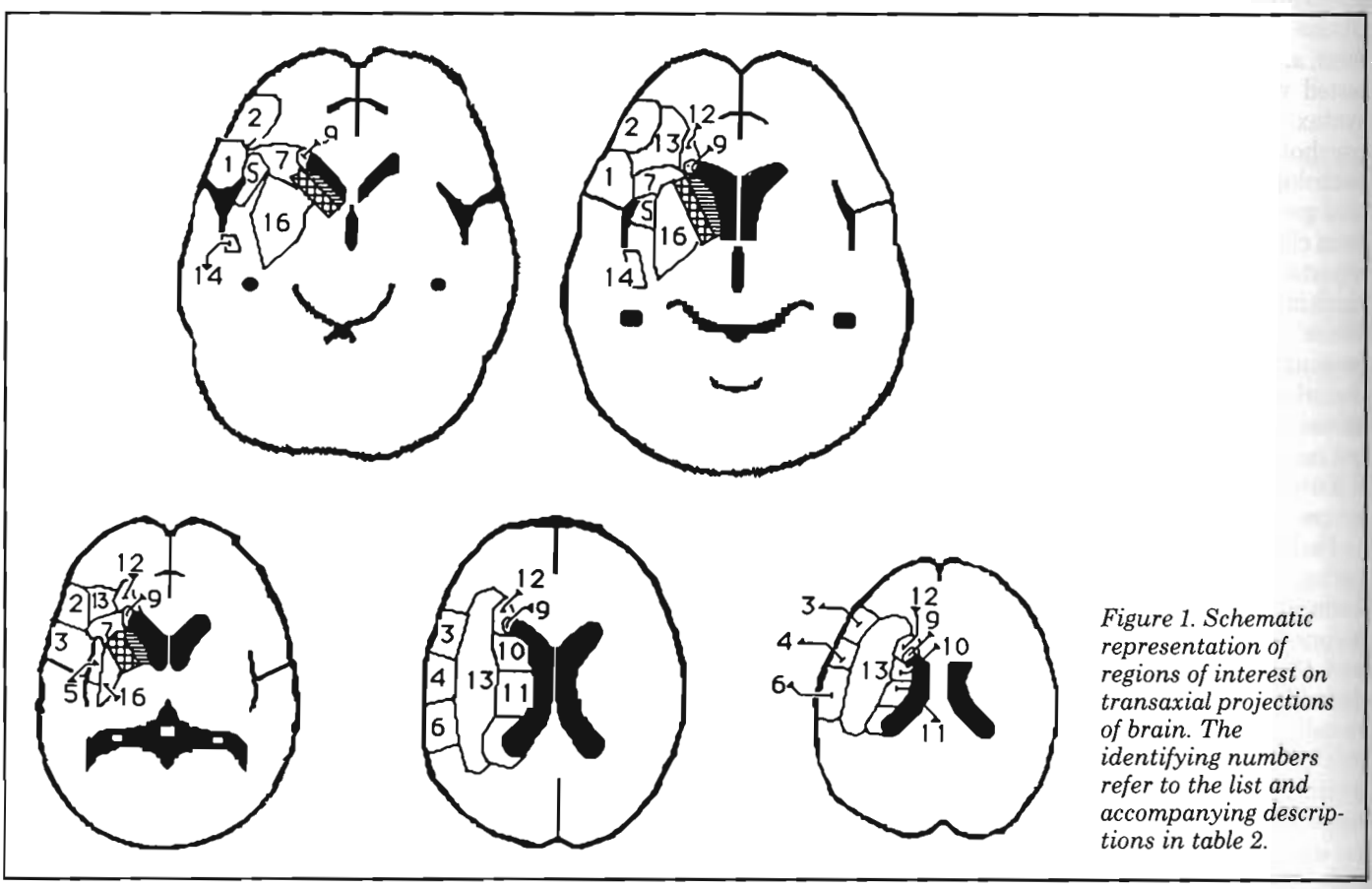

Figure 1. Schematic representation of regions of interest on transaxial projections of brain. The identifying numbers refer to the list and accompanying descriptions in table 2.

with a battery validated at the Boston VAMC[14]; all results are to command. The test results were obtained 1 to 5 months after onset of illness (table 1). All speech and language evaluations were performed by examiners blind to CT findings.

CT studies. All CTs were unenhanced and performed more than 3 weeks after onset. Lesions were visually assessed with a neuroanatomic checklist by 2 reviewers experienced in CT localization. Scans were specifically analyzed for lesion in a number of discrete cortical and subcortical regions of potential functional significance. Schematics of CT slices demonstrating the areas that were rated are displayed in figure 1; the areas are further defined in table 2. Some of the areas are present on more than 1 slice and, for this study, some have been given labels which are not part of standard terminologies. We have used a similar technique with similar labels in previous reports.[15,16]

Table 2. Abbreviations and operational definitions of the anatomic regions rated for lesion

Cortical		
1. Frontal operculum	FOP	Posterior, inferior frontal gyrus including pars opercularis and pars triangularis
2. Middle frontal gyrus	MFG	Areas above and anterior to FOP; probably includes the part of inferior frontal gyrus immediately anterior to pars triangularis
3. Lower motor cortex	LMC	Lower 50% of prerolandic gyrus
4. Lower sensory cortex	LSC	Lower 50% of postrolandic gyrus
5. Anterior, superior insula	ASI	Suprasylvian, prerolandic insula
6. Anterior supramarginal	ASMG	SMG anterior to the posterior limit of the sylvian fissure
White matter (WM)		
7. Frontal isthmus	FI	White matter confluence anterior to the ALIC
8. Anterior limb internal capsule	ALIC	
9. Subcallosal fasciculus	SCF	WM forming the lateral angle of the frontal horn of the lateral ventricle
10. Anterior, superior paraventricular WM	ASPVWM	Innermost 1/3 of WM adjacent to the anterior 1/3 of body of lateral ventricle
11. Middle, superior paraventricular WM	MSPVWM	Innermost 1/3 of WM adjacent to the middle 1/3 of body of lateral ventricle
12. Anterolateral periventricular WM	ALPVWM	WM around and superior to the frontal horn; anterolateral to the medial subcallosal fasciculus
13. Subcortical WM	SCWM	Moving from medial to lateral, the middle 1/3 of the WM
14. Temporal isthmus	TI	WM pathways to temporal lobe passing behind the sylvian fissure; the anterior 1/2 contains the medial geniculotemporal connections
Basal ganglia		
15. Caudate	CAUD	Head of caudate nucleus
16. Lenticular nucleus	PUT	May involve the anterior or lateral segments or the entire putamen; globus pallidus on lower sections may rarely be involved

Results. Language and praxis findings are reported in table 1. CT lesions are reported in table 3, with reference to neuroanatomic regions displayed in figure 1 and summarized in table 2. The findings are most easily outlined by dividing the patients into a few types based upon lesion topography, but these groups are not to be taken as immutable sets of signs.

Clinical-anatomic correlations. Patient 1 is the only case of the 1st type in the present report. He had a lesion in frontal operculum, middle frontal gyrus, and white matter deep to frontal operculum including the frontal isthmus, but no involvement of lower motor cortex, of subcortical white matter deep to sensorimotor cortex, or of the periventricular or paraventricular white matter (PVWM) other than the frontal isthmus (figure 2). (Note that throughout the text and in the tables, "PVWM" can stand for either para- or periventricular white matter. When "PVWM" is unmodified, it refers to both.) He is the only case with *no* elemental motor deficits, with entirely normal speech quality (articulation and melodic line), with normal repetition (not shown in table 1), and with no ideomotor apraxia. Language was terse and often delayed in initiation, but grammatically correct. There were word-finding deficits and semantic paraphasias.

Patients 2 through 6 had lesions very similar to each other and constitute the 2nd general type of case. These patients had substantial cortical involvement of frontal operculum, lower motor cortex, and anterior superior insula, involvement of subcortical white matter deep to those regions, and further deep extension into the frontal isthmus, anterolateral periventricular white matter, anterosuperior paraventricular white matter, putamen, anterior limb of internal capsule, and either the anterolateral periventricular white matter or the anterosuperior paraventricular white matter, or both. (CT findings are demonstrated in a composite in figure 3.)

All patients had central right facial paresis and mild right hand weakness. By history, cases 2, 4, 5, and 6 were mute for days. At the time of our evaluation speech quality was impaired in all 5 patients, from moderately defective articulation and prosody to severe dysarthria and dysprosody. Patients 2 through 5 had sentence-length, grammatically normal utterances despite initiation struggle and impairments in speech production. Phonemic paraphasias were prominent. Repetition and oral reading were comparable to spontaneous output—dysarthric, dysprosodic, with phonemic paraphasias but normal grammar. Only patient 6 had more severe spoken aphasia. She could produce only single word, telegraphic utterances, not improved in repetition or oral reading.

All 5 patients had severely impaired written language. Writing of primer dictation words was mildly impaired, but narrative writing capacity was very reduced with only single words or shortened phrases and agrammatical constructions ("I go and tennis"). Patient 6 made much more use of writing to communicate, presumably because spoken output was so limited, but her writing was qualitatively similar to the others.

All 5 patients had substantial buccofacial apraxia but mild or no limb apraxia.

Patients 2, 3, 5, and 6 were followed for over 1 year. All improved. Patients 2, 3, and 5 continued to have dysarthria, mild deficits in speech initiation, and overall terse responses. There were still word-finding problems and predominantly phonemic paraphasias. Communication was functional. Patient 6 received several months of Melodic Intonation Therapy[17] beginning 5 months after the stroke. Spontaneous output im-

Table 3. CT lesions

Pt no.	Cortical lesion							SCWM					
	MFG	FOP	LMC	LSC	ASMG	ASI	Other	MFG	FOP	LMC	LSC	ASMG	Other
1	±	+	—	—	—	±	—	—	+	—	—	—	—
2	—	+	+	—	—	+	—	—	+	+	—	—	—
3	—	±	+	+	—	+	—	—	+	+	+	—	—
4	—	+	+	—	—	+	—	—	+	+	—	—	—
5	—	+	+	+	—	+	—	—	+	+	+	—	—
6	—	+	+	—	—	+	Post-SMG	—	+	+	—	—	—
7	—	—	+	+	+	+	—	—	—	+	+	+	—
8	—	—	—	—	—	±	—	—	—	+	+	—	—
9	—	—	+	—	—	+	—	—	—	+	—	—	—

See table 2 for definitions of heading abbreviations.

\+ Major lesion (more than half of the area has lesion).
± Small or patchy lesion.
— No lesion.

ant Anterior putamen only.
lat Lateral putamen only.
SMG Supramarginal gyrus.

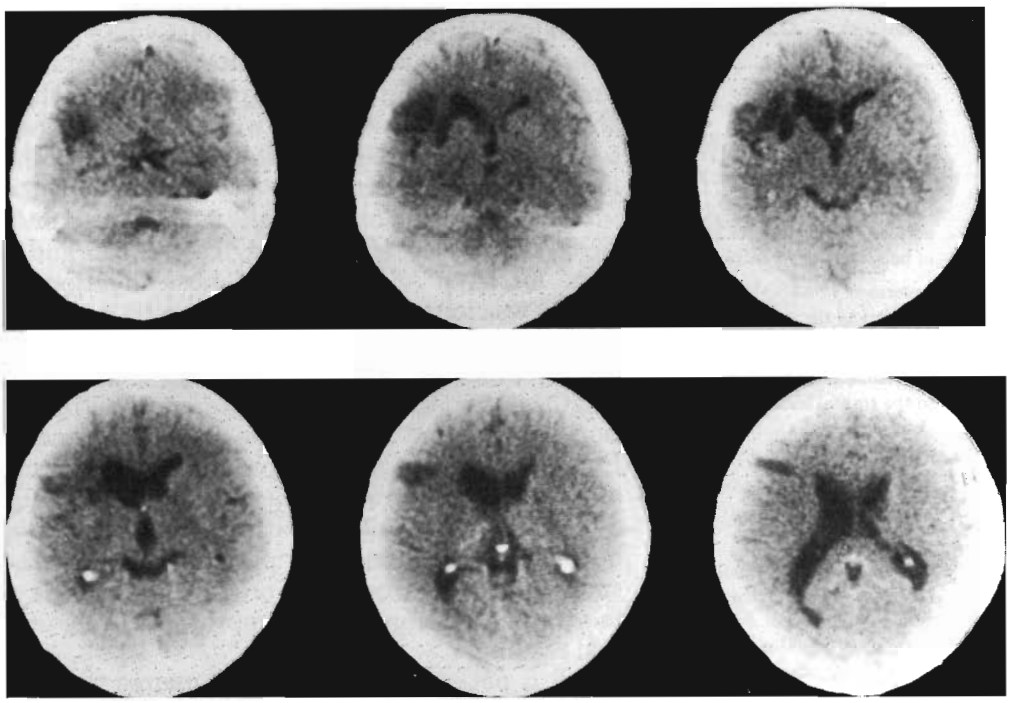

Figure 2. CT of patient 1. Lesion in operculum and middle frontal gyrus but with deep white matter extension only to the frontal isthmus.

proved over the next 6 months. Phrases became often sentence-length; grammatical form was reduced, but agrammatical structures were infrequent. Dysarthria, dysprosody, speech initiation delay, and phonemic paraphasias remained prominent.

Patients 7 to 9 had smaller lesions without any direct involvement of frontal operculum; they represent a 3rd lesion type. All 3 had damage to lower motor cortex or subcortical white matter deep to lower motor cortex, damage to anterior superior insula, and to lateral putamen. Patients 7 (figure 4) and 8 (figure 5) had substantial subcortical white matter damage deep to perirolandic regions. Deep damage was restricted to anterolateral periventricular white matter in patients 8 and 9 (figure 6). Right central facial paresis was the only elemental abnormality. Patients 7 and 8 had aphasia profiles quite similar to patients 2 through 5: dysarthria and dysprosody, but grammatical, sentence-length utterances with word-finding problems and paraphasic errors, predominantly phonemic. There was less initiation delay for patients 7 and 8. Repetition and oral reading produced abundant phonemic paraphasias. Patient 7 had strikingly good writing (sentence length with phonemic paragraphias; ie, identical to spoken output) and strikingly little buccofacial apraxia compared with the other cases. He was also the only left-hander in the study. Patient 9 is similar to the others in many measures—auditory comprehension, primer dic-

PVWM					Capsule/Striatum		
FI	AL	AS	MS	TI	PUT	CAUD	ALIC
+	—	—	—	—	+	—	+
+	—	±	±	—	ant	+	+
—	+	+	—	—	lat	—	+
+	+	±	—	—	ant	—	+
+	+	±	±	—	+	—	+
+	+	±	—	—	lat	—	+
—	—	—	—	—	lat	—	—
—	+	—	—	—	lat	—	—
—	+	—	—	—	lat	—	—

tation writing, and praxis—but he had, and continues to have at 4 years post-stroke, virtually no speech output. Despite intensive therapy, spoken output has changed very little, but apraxia has improved. He uses elaborate gestures to communicate.

Discussion. We believe that there is evidence that the so-called nonfluent aphasias, including classical Broca's aphasia, have identifiable constituent elements and that it is analysis of the anatomy of those constituent elements—not of the entire syndrome—that will best define brain-behavior relationships. The literature on aphasia contains considerable support for this premise.

Mohr et al[3,4] were instrumental in the 1970s in refocusing investigation of Broca's aphasia on its constituent elements and off the complete syndrome. The strategy of that research was to select cases with lesions in and around the frontal operculum, and the main result was the demonstration that lesions of Broca's area (taken to be the frontal operculum) did not result in classical Broca's aphasia.[4] Although the format of their reports allow only limited case-by-case clinicoanatomic analysis and Mohr et al seemed satisfied with characterizing these milder cases under the general rubric of Broca's area aphasia, there are unequivocal examples of the different profiles that we are reporting here to be found within the Broca's area aphasia reports. All examples had published CT schematics or postmortem findings similar to the ones we have reported here. Subsequent reports from other investigators demonstrated the same profiles.[18,19] The formats of the reports again made case-by-case comparisons impossible, and patients were labelled only by syndrome designation.

We propose that the Broca's area aphasias actually subsume 3 separate profiles that may overlap in any combination. Each profile has its own pathologic anatomy.

The 1st profile is impaired articulation and prosody. Déjérine[20] was correct when he argued that damage to lower motor cortex or to the subcortical white matter efferent projections of lower motor cortex could produce dysarthria, with or without aphasia. There are several recent reports of small, shallow lesions in lower motor cortex, not involving frontal operculum.[8-10,12] These patients have had primarily motor speech deficits that have been elegantly analyzed in 1 report.[8] We have previously reviewed[12] the various notions about this motor deficit of speech. It has been labelled aphemia,[12] cortical dysarthria,[21] subcortical dysarthria,[22] and even apraxia.[23] Whatever the label and whatever the rostral (lower motor cortex) or caudal (genu of internal capsule) location of the lesion, these reports have all described an efferent motor speech disturbance. Damage to this efferent motor system presumably underlies the articulation/prosody disturbances that were observed in all our patients except case 1.

The 2nd profile is delayed initiation of language with sparse but grammatical and, at their best, sentence-length utterances with anomia, semantic paraphasias, and preserved repetition. This profile fits criteria[13,24] for transcortical motor aphasia (TCMA). Two major recent studies of TCMA[25,26] include cases with lesions centered in frontal operculum without involvement of lower motor cortex or PVWM. In fact, patients 2, 4, and 5 of our earlier report[25] had clinical-anatomic profiles *precisely* like patient 1 in the current report. There are 2 additional case reports with similar clinico-anatomic relationships.[7,9] Both cases had laconic, terse, grammatical, sentence-length utterances with semantic paraphasias. Articulation was normal. Both had almost complete recovery within a month, perhaps confirming Kleist's assertion that extension to PVWM is essential for lasting aphasia after lesions in the frontal operculum.[27]

The 3rd profile within the Broca's area aphasias includes impaired articulation and prosody *and also* phonemic errors in all output; this profile is accompanied by buccofacial apraxia. This profile occurs after lesions in lower motor cortex, subcortical white matter, and PVWM structures deep to lower motor cortex including anterosuperior paraventricular white matter and middle superior paraventricular white matter . In addition to our patients 2 through 8, the reports of Mohr et al[3,4] and of Tonkonogy and Goodglass[9] unequivocally identify patients of this type.

It is an important observation that the 4 defining features of classical Broca's aphasia or, at least, of nonfluency[13]—articulatory struggle, impaired prosody, shortened phrase length, and reduced grammatical form—do not occur together in these 3 more restricted profiles. When the features of nonfluency are dissociated, there are specific implications about the anatomy of the lesion and perhaps about the prognosis of the aphasia. We have previously described similar feature dissociations in cases with primarily subcortical lesions.[15,28] Knopman et al[29] have also described the dissociation of the features of nonfluency after lesions of operculum or of paraventricular white matter. The methodology of CT analysis in that report[29] does not allow complete comparison with our cases nor ready division into the profiles that we propose here. The interaction of lesions in lower motor cortex, in frontal operculum, and in the various portions of the para-

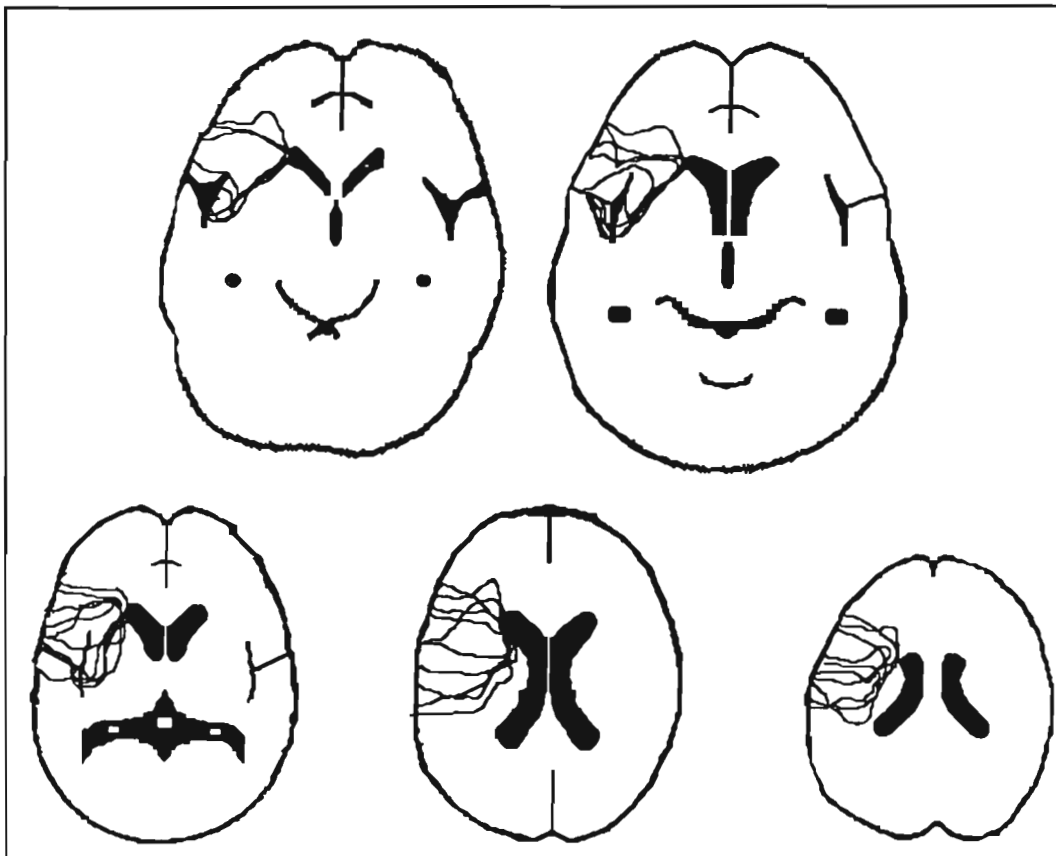

Figure 3. Composite of CT studies from patients 2 through 6. Note consistent and extensive direct and undercutting involvement of operculum and lower motor cortex. There is deep white matter involvement to the periventricular region anterolateral to the frontal horn, as well as at least partial involvement of the paraventricular white matter adjacent to the anterior superior portion of the body of the ventricle.

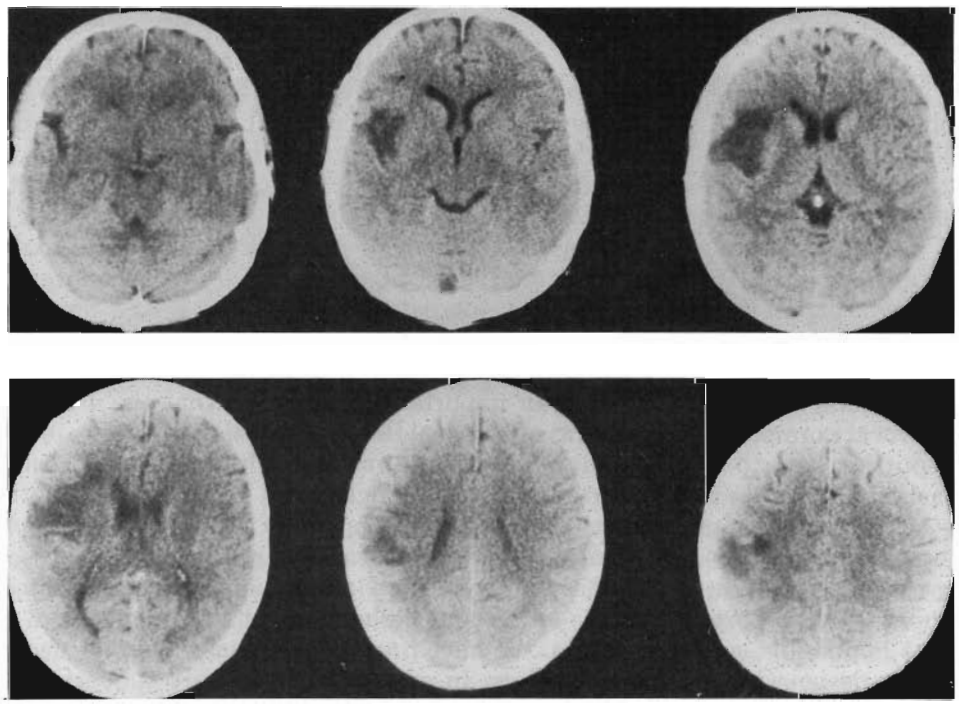

Figure 4. CT from patient 7. Involvement of lower motor and sensory cortex regions as well as the anterior, inferior parietal lobule and the subcortical white matter deep to those regions. There is no significant involvement of the PVWM.

ventricular white matter seem critical to dissociated features of nonfluency in their cases as well. That features of nonfluency dissociate has obvious implications for investigations of aphasia.[30]

These are then 3 functional systems damaged by lesions in Broca's area. The systems can be damaged in isolation with lesions restricted to (1) operculum[7,9,26] (initiation and formulation), (2) lower motor cortex[8,9,12] (articulation and prosody), or (3) temporoparietal to opercular connections[10] (phonemic production). The

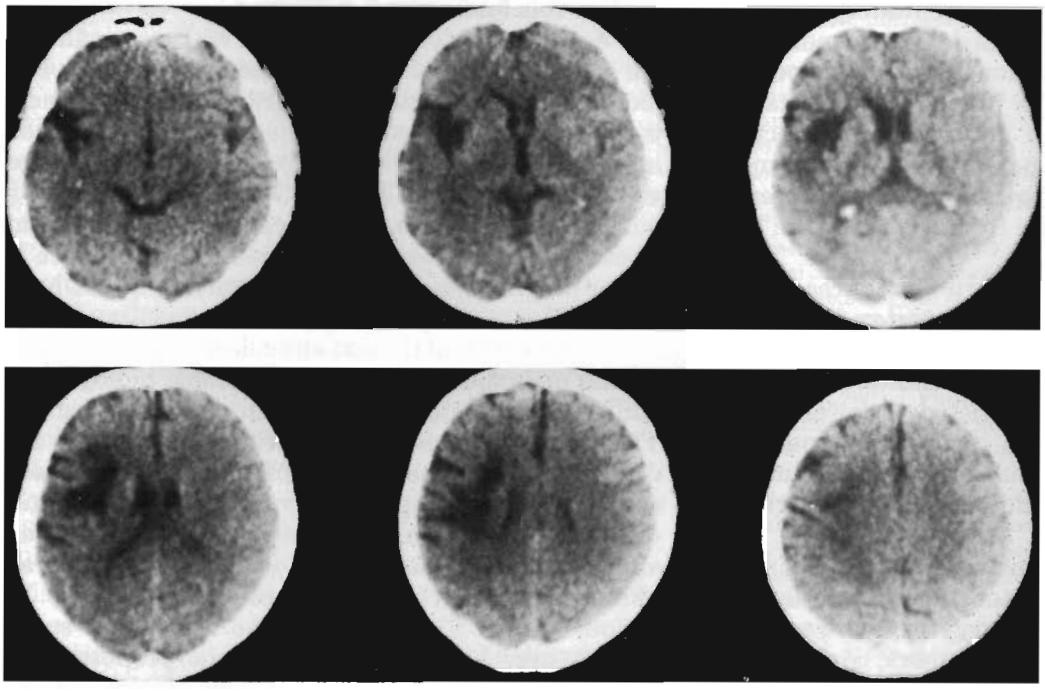

Figure 5. CT from patient 8. Minimal cortical and PVWM involvement, but extensive damage to the subcortical white matter deep to lower motor and sensory regions.

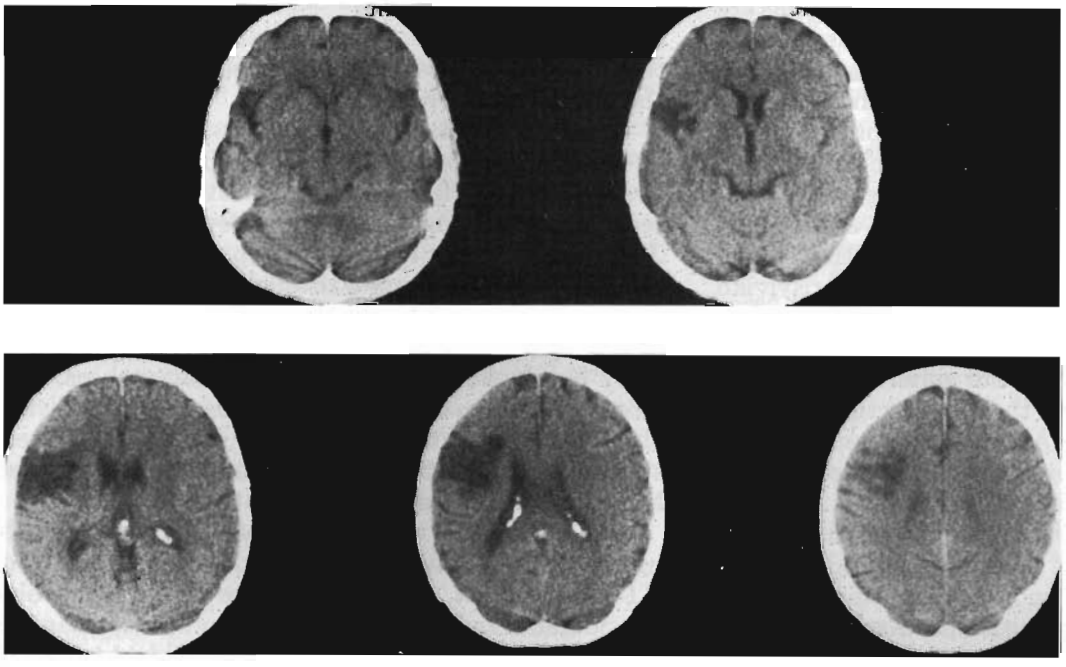

Figure 6. CT from patient 9. Lesion in lower motor cortex, white matter deep to motor cortex and anterolateral PVWM.

specific pattern of injury determines the exact language result.

The nature of the underlying brain-behavior relationships is illuminated by evidence from other sources. Cortical stimulation studies[31,32] have indicated that stimulation of the frontal operculum produces naming impairments and speech arrest. Ojemann and Whitaker[32] have suggested that frontal operculum seems to serve as the intersection between the cerebral system for language and the system for motor speech activity. Frontal operculum is the center of a broad region of frontal lobe which is the terminal projection area of the supplementary motor area,[33,34] and 1 major avenue of the activation to speak comes to the left frontal region from the limbic system through the supplementary motor area.[16,25] The frontal operculum also has bidirectional connections with key posterior cortical regions for language, the superior temporal gyrus, and the neighboring inferior parietal lobule.[35]

In sum, frontal operculum serves as an area which is situated to integrate the activational or facilitatory (limbic) aspects of the language, the semantic (posterior

connections) aspects of language, and the motor planning aspects of language.[32] This facilitatory capacity may be *centered* in operculum, but damage to more than operculum alone is necessary for deficits to be permanent. More persistent deficits in language facilitation (sparse, hesitant utterances) and in lexical selection (mild anomia and semantic paraphasias) require more extensive damage in the deep frontal white matter to the projections to dorsolateral frontal cortex from limbic and posterior structures.

Stimulation of lower motor cortex produces speech production deficits.[32] Lower motor cortex in the left hemisphere has particular importance for articulation despite the bilateral cortical representation of bulbar function.[12] Shallow lesions restricted to lower motor cortex produce almost pure dysarthria and dysprosody.[8,9,11] Lesions of the subcortical white matter below lower motor cortex produce the same dysarthria/dysprosody.

In the cases with more extensive lesion in subcortical white matter, deep to lower motor cortex, lower sensory cortex, or anterior supramarginal gyrus, there will also be damage to the longer intrahemispheric connections. Among these connections is the arcuate fasciculus, which runs from the posterior perisylvian region (temporal and parietal) to the posterior, inferior frontal region in the subcortical white matter (not the PVWM) above the extreme capsule.[36] Phonemic paraphasias are prominent in patients with damage to this system, whether the lesion is in Wernicke's area or the temporofrontal connections in subcortical white matter, and whatever the resulting aphasic syndrome.

There are other functional systems involved in these patients that do not have obvious anatomic bases. All of our cases, like all aphasics, were anomic, and all had semantic paraphasias. This disturbance at the level of semantic retrieval is found in all cases of left perisylvian injury.[37] There may be differences in the exact quality or severity of disturbance in semantic retrieval with different lesion localizations,[38] but apparently all parts of the left central cortex and their connections participate in semantic function. All cases had some impairments in auditory comprehension at the level of complex sentences or multistep commands. It has often been observed that comprehension of sentential or syntactically complex material is impaired in virtually all aphasics.[39] The demands upon working memory, sequential memory, or the cognitive processing required to solve the problems of syntax may involve a network that consists of essentially all portions of the perisylvian region. There are then functional systems for semantic retrieval and for sentential/syntactic comprehension, but they are integrated across so much of the left hemisphere that they have no meaningful localization at the level of the gyrus or the discrete pathway that has been the focus of this report. The agraphia seen in all of our cases may be a phonologic agraphia that has been reported with perirolandic lesions.[40] It could be due to damage to the intrahemispheric connections between the parietal lobe and the frontal lobe that have been proposed to be critical for writing.[41] We did not obtain enough data about the linguistic nature of the writing errors to be able to explore this further.

The problem of variability. Even with our small sample of patients, there was evidence of variability. Four factors may contribute to the variability. The 1st factor is the existence of basic biological differences between individual brains. Left-handedness is sometimes taken as a macromarker of basic biological differences.[42] In our series, the absence of significant apraxia or agraphia in case 7 may be related to anomalous dominance for motor programs, a reported phenomenon in left-handers.[43]

Microanalysis of the size and sulcal complexity of the frontal operculum suggests considerable biologic variability in the basic anatomic structure of the region.[44] The work of Ojemann and Whitaker,[32] showing that different patients have different thresholds for speech arrest or anomic responses after electrical stimulation of frontal operculum, points to basic physiologic variability. There is, then, anatomic and physiologic variability—both beyond CT resolutions—in this key region. Patient 9, who has had no recovery of speech output, may be an exception to the proposed relationships based on a particular biological variability such as that demonstrated by Ojemann and Whitaker.[32] There are no known methods for demonstrating such variability. There are also at present no known hypotheses for the neural basis (or bases) for anomalous or exceptional *intra*hemispheric localizations of functions,[45] although there are emerging hypotheses about the interhemispheric anomalies.[46]

A 2nd factor that may contribute to variability is unequal recovery. Perhaps most brains show the same acute effects of a lesion, but the plasticity necessary to recover, partially or completely, differs among patients. This could account for patient 6, part of whose profile seemed quite appropriate throughout, but other parts only evolved later in the course with specific therapy.[47]

A 3rd factor might be critical but subtle, and presently overlooked, brain-behavior relationships. The size of the lesion does not seem to be the critical variable. Of the patients (6 and 9) with unexpectedly severe spoken output impairments, 1 had a moderate-sized lesion and 1 had a small lesion (patient 9).

A 4th factor of variability may be that CT simply misses the responsible lesion. For this reason, timing of CT is important. Analysis of acute CTs may involve considerable risk of a missing lesion still evolving or overestimating as lesion what is actually just transient edema. Our experience has been that CT performed 2 or more months after onset is optimum for delineation of lesion boundaries necessary for precise brain-behavior correlations. MRI studies will probably reduce this timing problem. Some of the clinical variability in the work of Mohr et al[4] may be due to analysis of only very early scans.

Conclusions. (1) The frontal operculum is involved in speech and language initiation, presumably a reflection of its position as the termination of limbic projections to frontal lobe and as motor association cortex for speech. Shallow lesions of frontal operculum produce

very transient reductions of language output with normal articulation and repetition. Lesions of frontal operculum that reach to the deep white matter in frontal isthmus and anterolateral periventricular white matter have a much more prolonged and severe but similar profile (TCMA in classical terminology[25]), presumably because of much more extensive damage to the limbic-frontal projections[34] and to cingulate-striatal-frontal connections.[48]

(2) The left lower motor cortex is involved in speech production, presumably a reflection of its position as the final pathway of motor control of speech from the language-dominant hemisphere. Shallow lesions of lower motor cortex produce a restricted dysarthric disorder, best characterized by Lecours and Lhermitte[8] as pure phonetic disintegration. In some cases, the impairment in prosody may be as severe as or more severe than the articulatory problem.

(3) Lesions extending more deeply to involve subcortical white matter and PVWM also damage *intra*-hemispheric pathways of language and result in a much more complex syndrome with phonemic substitutions and writing impairment, in addition to the dysarthria.

(4) The combination of lesions—frontal operculum and lower motor cortex plus subcortical white matter and PVWM—result in a more severe aphasia, with effortful initiation, phonemic paraphasias (spontaneous and in repetition), writing impairment, and ideomotor apraxia. In the acute phase, these patients may have traditional, nonfluent aphasia—articulation impairment, prosodic impairment, and agrammatical, shortened utterances. The evolved disorder is, however, much less severe than that; grammatical, sentence-length utterances return, albeit still labored and paraphasic and with speech impairment.

(5) Classical Broca's aphasia follows damage to all of the above systems plus additional damage to the limbic-frontal periventricular pathways of the medial subcallosal fasciculus.[16] With a lesion so large and a syndrome so complex, it is not possible to dissect out the contributions of the various functional systems. In any case, the analysis of the pathologic anatomy of aphasia is most profitably pursued through investigations of the distributed anatomy of the individual performance deficits—*not* the syndromes.[49]

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