Article abstract—Analysis of language profiles and CT anatomy in transcortical motor aphasia (TCMA) suggests that the essential lesion is disruption of connections at sites between the supplementary motor area and the frontal perisylvian speech zone. If the lesion is extended, there may also be poor articulation (lesion deep to motor strip for face), impaired auditory comprehension (lesion in anterior head of caudate, anterior limb internal capsule, anterior putamen, and anterior portion of external capsule, claustrum, extreme capsule, and insula), or stuttering (lesion in pars opercularis and lower third of premotor region). This concept unifies disparate anatomic and psychophysiologic observations about three syndromes: classical TCMA, aphasia after left medial frontal infarction, and TCMA during recovery from Broca's aphasia.

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Anatomic basis of transcortical motor aphasia

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Most texts on aphasia approach language disorders by identifying several distinct syndromes,[1-3] including transcortical motor aphasia (TCMA). However, there have been few reports of both clinical description and anatomic data. Either language information is scant, or the cases were caused by trauma or tumors with poor localizing value. We studied 15 patients with TCMA or near-variants of the syndrome. These near-variants were associated with impaired articulation or auditory comprehension problems, or with stuttering.

Patients. *Patient description.* Fifteen right-handed aphasic men were selected from the Boston or Palo Alto Veterans Administration Medical Centers (table 1). Seven had classical TCMA, and eight had a near-variant, as described below. Twelve of the fifteen cases had a Boston Diagnostic Aphasia Exam (BDAE)[4] 1 week to 10 months after onset.

Classical TCMA. The criteria for the diagnosis of classical TCMA consisted of (1) limited spontaneous speech, (2) intact repetition, (3) normal articulation, and (4) good auditory comprehension. Seven patients met these requirements. The following two samples of an oral description of the Cookie Theft Picture of the BDAE[4] are illustrative of their speech output. The examiner's comments are shown in parentheses. The dots indicate long pauses.

Patient 1 (1 month after onset): "No I can't . . . I can't . . . water comin' out of the sink . . . (what else?) . . . Boy fallin' off stool . . . (What's he doing?) . . . Stealing cookies . . . (from?) . . . cookie jar . . . (What's she doing [girl]) . . . eatin' . . . (Mother?)."

Patient 6 (6 weeks after onset): "Well . . . it's . . . (pointed to water) trouble with this . . . (laughed) . . . I can't tell . . . having trouble . . . (What is that?) . . . It's an . . . I'm having trouble . . . (What about this?) . . . I understand what it is . . . it's just that I . . . can't . . . logically . . . break . . . down the ah . . . I just don't understand and . . . at the point . . . don't understand . . . ."

Five of the seven patients with classical TCMA had paraphasia (literal and verbal) and word-finding problems. In these five patients, visual confrontation naming was impaired. Word list generation was very poor. Writing samples (available in four) all showed problems, from ability to write only isolated words to production of paragrammatical sentences with spelling errors and semantic substitutions. Oral reading and reading comprehension, tested in three subjects, was only mildly impaired or normal. Language abnormalities could be magnified by asking the patient to explain proverbs, to describe some simple sequential task (eg, changing a tire), or to tell a story. Under these conditions, word-finding difficulty, perseveration, problems of initiation, and frequent blocking all became more prominent.

The other two of the seven patients with classical TCMA (patients 1 and 7) had little abnormality, other than difficulty initiating speech. They made no

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Table 1. Clinical information

	Case	Sex	Age	Etiology	Hemiparesis	Grasp reflex	Time to 1st exam (days)
Classical TCMA	1	M	66	Infarction	Severe (predominantly leg)	Yes	29
	2	M	56	Infarction	Absent	No	6
	3	M	60	Hemorrhage	Mild	Yes	19
	4	M	60	Infarction	Absent	No	15
	5	M	59	Infarction	FW	No	3
	6	M	53	Hemorrhage	Mild	No	49
	7	M	56	Infarction	Absent	?	116
Impaired articulation	8	M	38	Hemorrhage	Mild	No	70
	9*	M	65	Infarction	Moderate	No	35
	10*	M	64	Infarction	Absent	?	37
	11	M	59	Infarction	FW	No	7
Impaired comprehension	9*	M	65	Infarction	Moderate	No	35
	10*	M	64	Infarction	Absent	?	37
	12*	M	54	Infarction	FW	No	12
	13	M	60	Infarction	FW	No	5
Stutterers	12*	M	54	Infarction	FW	No	12
	14	M	65	Infarction	FW	No	5
	15	M	42	Infarction	Absent	?	28

* Patients belonging to more than one group.
FW Facial weakness only.

overt linguistic errors. Naming was flawless in patient 1 and mildly impaired in patient 7. Word list generation was poor in both.

Near-variant syndromes. The patients with near-variant syndromes had (1) limited spontaneous speech, indistinguishable in language content from the classical TCMA cases, and (2) intact repetition. They failed to meet the complete criteria for TCMA because of one or more of the following problems: (1) impaired articulation (four patients), (2) mild auditory comprehension problem (four patients), or (3) stuttering (three patients).

Impaired articulation was defined as consistent awkwardness in the production of individual phonologic elements, simplification of consonant blends, or consistent nonparaphasic sound substitutions (table 2).

The following sample is given to illustrate the similarity of the limited language content of those with classical TCMA to those with poor articulation. No attempt was made to transcribe the articulatory errors:

Patient 10 (2 months postonset): "I was retam ... I was retam ... I was retired. We repoy ... repoy ... (What did you retire from?) ... We retired from people who ... no ... (Did you work for yourself?) ... no ... work for (unintelligible) ... for the government ... (federal or state?) ... yes federal government ... (what kind of work did you do for the federal government?) ... We took money and ...."

In patients with impaired articulation, writing varied from no relevant narrative production to simplified but correct sentences (samples available in three cases). Oral reading (four cases) ranged from impaired with word omissions and literal paraphasias to totally intact. Reading comprehension was normal or impaired on only lengthy complex material. Visual confrontation naming was moderately impaired in two patients and good in two. Word list generation was poor.

Impaired auditory comprehension was defined as a deficit in understanding oral verbal material at the single-word or sentence-length level (table 3). On bedside testing,[3] patients were most impaired on the more complex and lengthy verbal material.

Spontaneous speech had the same qualities as in the classical cases.

Writing varied from a total impairment to production of single words. Oral reading ranged from only single words to good on short sentences. Reading comprehension ranged from impaired at the single-word level to impaired only on more lengthy complex material. Visual confrontation naming ranged from moderate impairment to poor. Word list generation was very poor.

Stuttering was defined as the involuntary repetition, prolongation, or arrest of speech sounds.[5] In the patients who stuttered (table 4), stuttering was the only speech characteristic that differentiated them from the classical TCMA patients. A sample of speech follows:

Patient 14 (1 week postonset, describing Cookie Theft Picture): "Oh well . . . cookie the ah . . . k-k . . . S-well somebody's spelling something and somebody wanted to get a cookie jar and fell off and ah . . . s . . . was quite a mess s . . . and ah . . . the the . . . k-k- cookie there . . . ah . . . .s-well, etc."

Writing ranged from an inability to write more than the first name to the production of paragraphic

Table 2. Articulation

	Case	Lesion*	Articulation		Time post onset† (days)
			Bedside testing	Articulatory agility score (BDAE, max = 7)	
Infarction	1	0	Good	7	29
	2	0	Good	—	—
	4	0	Good	—	—
	5	0	Good	7	129
	7	0	Good	6	116
	12	0	Good	7	36
	13	0	Good	7	42
	14	0	Good	7	10
	15	0	Good	7	313
	9	1	Impaired	3	42
	10	1	Impaired	2	46
	11	1	Impaired	4	90
Hemorrhage	3	0	Good	7	26
	6	0	Good	7	73
	8	1	Impaired	—	—

*1 Lesion in PVWM deep to motor strip for face.
0 No lesion in area listed above.
† Earliest BDAE evaluation (obtained at least 1 month postonset) was used for statistical analysis; evaluation prior to 1 month postonset was used only if no later data were available.

Table 3. Auditory comprehension

	Case	Lesion*	Auditory comprehension		Time postonset† (days)
			Bedside testing	Auditory comprehension Z score (BDAE)	
Infarction	1	0	Good	+0.97	29
	2	0	Good	—	—
	4	0	Good	—	—
	5	0.5	Good	+0.94	129
	7	0	Good	+0.60	116
	11	0	Good	+0.75	57
	14	0	Good	+0.84	10
	15	0	Good	+0.98	313
	9	1	Impaired	+0.28	42
	10	0	Impaired	−0.05 (+0.89 at 3 yr)	46
	12	1	Impaired	−0.99 (+0.5 at 4 mo)	36
	13	1	Impaired	−1.32 (+0.05 at 4 yr)	42
Hemorrhage	3	0	Good	+0.72	26
	6	0	Good	+0.09	73
	8	0	Good	—	—

* 1 Lesion in anterior head of caudate, anterior limb internal capsule, anterior putamen, anterior portion of external capsule, claustrum, extreme capsule, insula.
0.5 Patchy lesion in areas listed above.
0 No lesion in areas listed above.

† Criteria for choosing scores at specified time postonset are the same as for articulation.

Table 4. Stuttering

	Case	Lesion*	Stuttering
Infarction	1	0	0
	2	0	0
	4	0	0
	5	0.5	0
	7	0	0
	9	0	0
	10	0	0
	11	0	0
	13	1	0
	12	0.5	1
	14	1	1
	15	1	1
Hemorrhage	3	0	0
	6	0	0
	8	1	0

* 1 Lesion in pars opercularis and lower third of premotor area.
0.5 Small or patchy lesion in areas listed above.
0 No lesion in areas listed above.

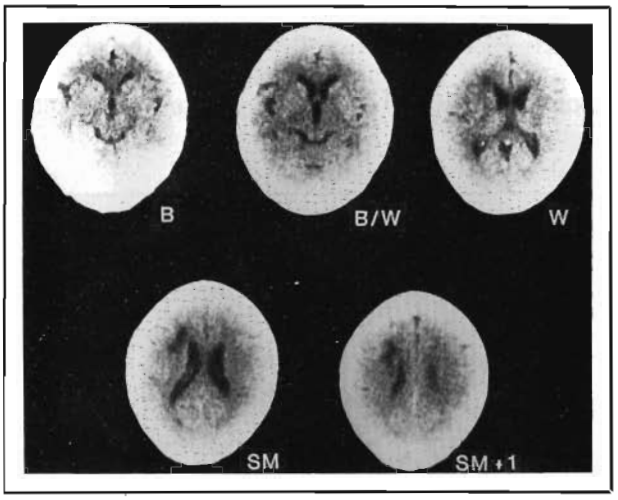

Figure 1. CT of classical TCMA case, in which frontal lobe lesion is anterolateral to the left frontal horn. Subcortical white matter lesion begins at slice W deep to left middle frontal gyrus area and continues with superior lesion extension, which is largest at slices SM and SM+1. There is no lesion in Broca's area (slices B and B/W); rather, lesion is deep and superior to Broca's area. Patient 4, aged 60, 7 days postonset.

sentences and phrases. On oral reading (two cases tested), the patients stuttered. Reading comprehension varied from being impaired at the single-word level to being good on sentence-length material. Visual confrontation naming ranged from poor (patient 12) to very good. Word list generation was severely impaired in the patient with poor visual confrontation naming.

Summary of patients. The fifteen patients (classical TCMA and near-variants) were essentially the same in their spontaneous spoken language and repetition, but varied in articulation, auditory comprehension, and stuttering.

In addition to the criteria by which we selected our patients, several other common features emerged. There were problems with initiation of speech, maintenance of a continuous fluid speech line, and production of narrative utterances of any complexity of content. Spontaneous speech was telegraphic in some cases, but was more often grammatical. Patients spoke in full sentences if encouraged; at least, they produced grammatical phrases and sentence fragments. Visual confrontation naming was variable, but could be very good. Word list generation (eg, animal naming) was highly impaired, particularly when compared with confrontation naming. Writing with either hand was often impaired. There were numerous paralinguistic features (dependent on language, but not strictly linguistic). There was a profound reduction in the capacity to generate an organized sequential narrative, even about a grossly automatized activity such as changing a tire or fixing an omelet. There was difficulty relating the abstract meaning of idiomatic expressions or proverbs. Perseveration was prominent across all language tasks, but distractability was not a prominent feature. Examination usually revealed no significant sensory, motor, or visual impairment other than a right central facial paresis or right leg paresis.

Methods. *Analysis.* CT. All patients had CT performed at least 2 months postonset, with the exception of a single patient (4), who had a scan at 1 week. CTs of the patients within each of the four clinical categories were examined to determine whether there was any consistent correlation between anatomic lesion site and the language abnormalities in each category. The technique used for lesion localization has been described.[6]

Classical TCMA. In all seven patients with classical TCMA, CT demonstrated a small lesion in the left frontal lobe (figures 1 and 2). The most common site was in the white matter anterolateral to the left frontal horn (figure 3). This lesion was usually largest at the level of the bodies of the lateral ventricles (slices SM and SM + 1), superior to the pars triangularis and pars opercularis of Broca's area (slices B and B/W). Only one patient (5) had a cortical and deep lesion in the pars triangularis area (slice B) and a patchy lesion in the pars opercularis area (slice B/W). None of the other six cases had any lesion in the pars triangularis or pars opercularis region of Broca's area. Three patients (1, 6, and 7) had lesions in the distribution of the anterior cerebral artery (ACA); one included the supplementary motor area (patient 7, figure 2), and two were deep to it (patients 1 and 6.) Patients 1 and 7 had virtually no language distur-

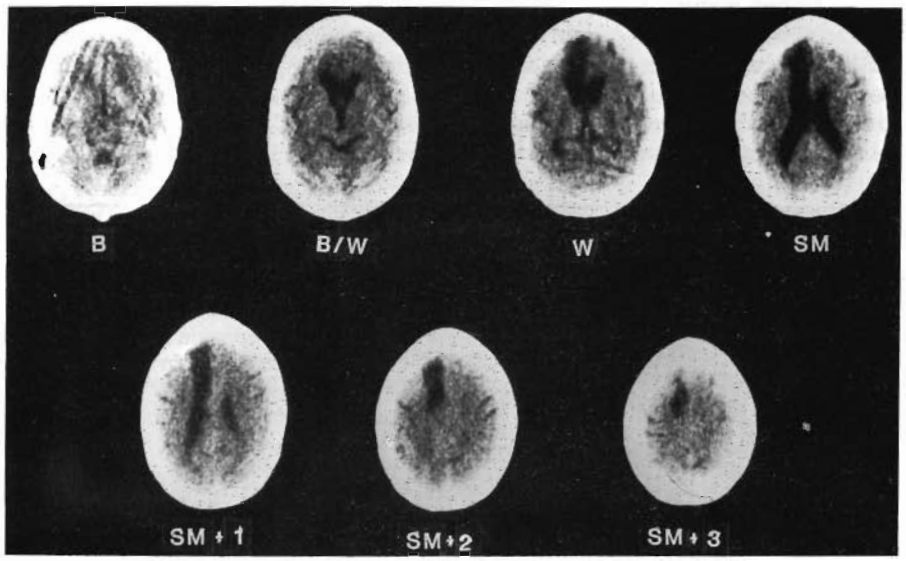

Figure 2. CT of very mild classical TCM aphasia case, in which frontal lobe lesion is directly anterior to the left frontal horn in the distribution of the left anterior cerebral artery. Lesion is cortical and subcortical in the region of the superior frontal gyrus (beginning at slice W) and extending into the supplementary motor area (slices SM+2 and SM+3). Patient 7, aged 56, 1 month postonset.

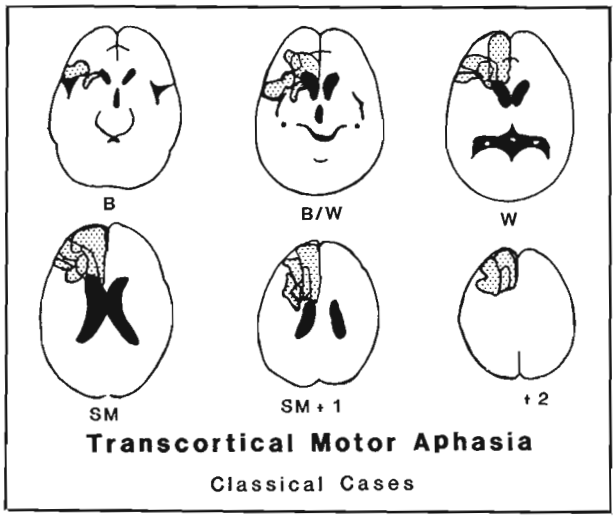

Figure 3. Composite of CT lesion sites for seven cases of classical TCM aphasia. Note the major lesion site is either a small lesion anterolateral to the left frontal horn (slices B/W, W, SM, SM+1, and SM+2) or a lesion directly anterior and superior to the left frontal horn (slices W, SM, SM+1, and SM+2), which includes the supplementary motor area (slice SM+2).

bance other than difficulty in speech initiation. Patient 6 had a hemorrhage and was clearly aphasic. Patients 1, 6, and 7 showed no akinesia or mutism at the time they were studied, which was 29 days postonset at the earliest. There was no specific lesion site that was found in all cases of classical TCMA.

Near-variants. The patients with near-variants of TCMA all had a left frontal lobe lesion that spared at least part of Broca's area, as did the classical TCMA cases. However, each of these patients had additional lesion extension into neighboring regions.

(1) Impaired articulation. In addition to the typical lesions anterolateral to the left frontal horn, the lesion in all these cases extended into the periventricular white matter area deep to the precentral gyrus region, representing face, lips, and tongue[7,8] at slice SM (figure 4). Two of the four patients (8 and 9) also had a patchy lesion in the pars triangularis or pars opercularis (slice B/W in figure 4, patient 8). Patient 11 also had a lesion deep to pars opercularis. No case with intact articulation had lesion extension into periventricular white matter (PVWM) deep to precentral gyrus area for face, at slice SM.

(2) Mild auditory comprehension problems. In addition to typical lesions anterolateral to the left frontal horn, three of the four patients (9, 12, and 13) had extension of the lesion completely across the anterior portion of the head of the caudate, anterior limb of the internal capsule, anterior putamen, and anterior portion of external capsule, claustrum, extreme capsule, and insula (figure 5). The fourth patient had, in addition to lesion anterolateral to the left frontal horn, lesions in the head of caudate, part of the anterior limb internal capsule, and PVWM deep to face area, as well as a questionable small lesion in the region of the angular gyrus.

(3) Stuttering. All three patients had lesions in the pars opercularis and lower third of the premotor region (figure 6). Two of them had lesions limited to these areas only (patients 14 and 15).

Statistical analysis. The clinical-anatomic findings suggest that extension of the lesion associated with classical TCMA may result in predictable deviations from the typical syndrome: (1) Involvement of the PVWM deep to the motor strip for face produces impaired articulation. (2) Extension across anterior head of the caudate, anterior limb of the internal capsule, anterior putamen, and anterior portion of external capsule, claustrum, extreme capsule, and insula produces impaired auditory comprehension. (3) Involvement of the pars opercularis and

lower third of the premotor region causes acquired stuttering.

To evaluate the statistical reliability of these predictions, we analyzed the relationships between quantitative measures of articulation, auditory comprehension, and stuttering and the lesion sites by Pearson product-moment correlation coefficients. Articulation and auditory comprehension were scored using the articulatory agility and auditory comprehension Z scores of the BDAE, respectively (tables 2 and 3). Stuttering was rated as "0" or "1", where "0" represented no stuttering, and "1" indicated that stutter-

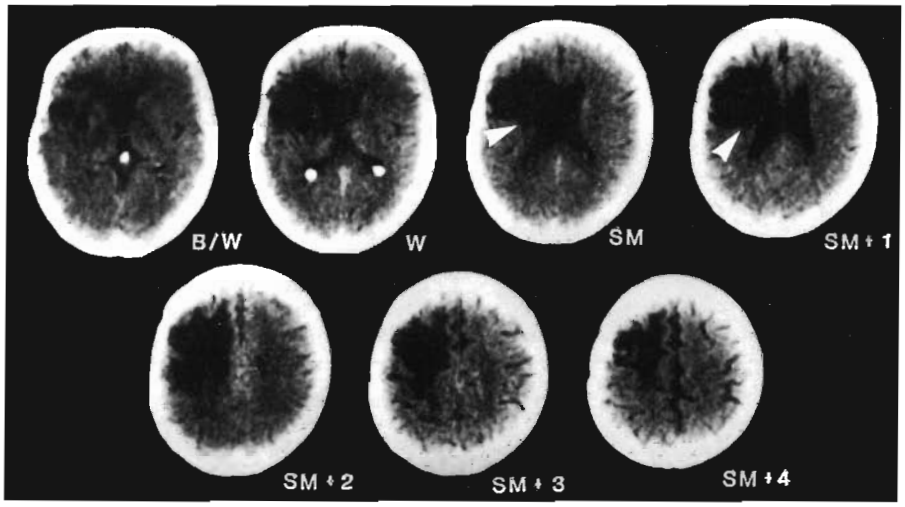

Figure 4. CT of near-variant TCM aphasia case with poor articulation. In addition to the frontal lobe lesion, which is anterolateral to the left frontal horn (all slices), there is superior lesion extension into the descending pyramidal pathways for face, lips, and tongue area in the periventricular white matter at slices SM and SM+1 (arrows). Patient 8, aged 54, 16 years postonset of intracerebral hemorrhage, which occurred at age 38.

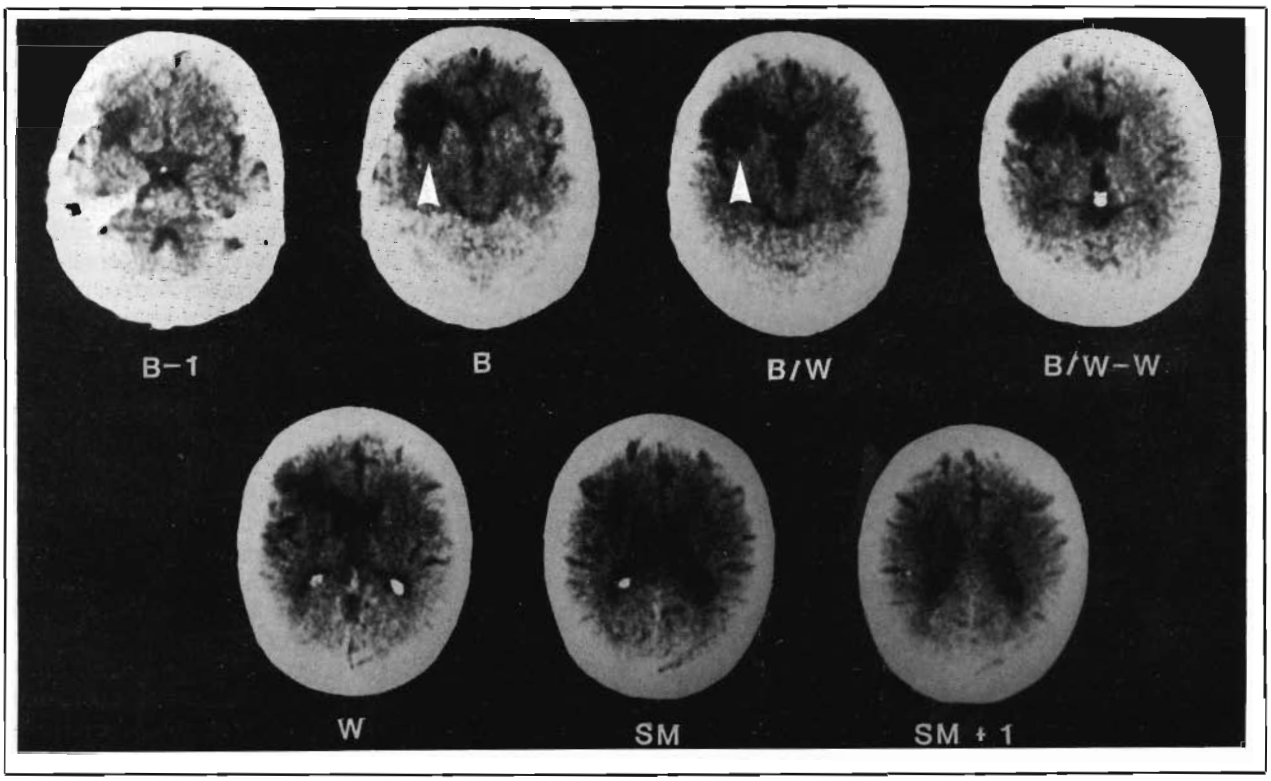

Figure 5. CT of TCM aphasia case with mild auditory comprehension problem. In addition to lesion, which is anterolateral to left frontal horn, there is lesion in the anterior portion of the head of the caudate (slice B/W-W) and lesion extension across the anterior limb internal capsule, anterior putamen, and anterior portion of the external capsule, claustrum, extreme capsule, and insula (slices B and B/W, arrows). Patient 9, aged 65, 1 month postonset. This patient, in addition to having a mild comprehension problem, also had poor articulation. Note superior lesion extension into the descending pyramidal pathways for face, lips, and tongue area in the periventricular white matter at slices SM and SM+1. (See also figure 4.)

ing was present (table 4). Lesion site was scored as "0" or "1", where "0" represented no lesion at the relevant site, and "1" indicated that a lesion was present. Statistical correlations were performed only on data from the patients with cerebral infarction—not on those with intracerebral hemorrhage.

The correlations between lesion site and language deficit were significant for articulation, auditory comprehension, and stuttering (table 5). To determine whether effects of either age or time after onset could have accounted for these correlations, semipartial correlations were computed to remove the effects of these additional variables, where relevant. Standard multiple regression techniques were used.[9]

The significance of the semipartial correlations remained unchanged for articulation (table 5).

For stuttering, time after onset was not removed, because one patient's stutter first became evident only after speech output began to improve (patient 12). The potential for the delayed appearance of stuttering made the meaning of time after onset difficult to interpret; only the effects of age were removed. The semipartial correlation was significant.

For auditory comprehension, the semipartial correlation with both time after onset and age removed was marginally significant (table 5). In fact, this semipartial correlation would have been stronger, except that the patients with the target lesion were tested relatively early after onset. When two of the other patients were retested at 4 months and 4 years, respectively, their auditory comprehension Z scores were still lower than the scores in six of the seven patients without target lesion (table 3).

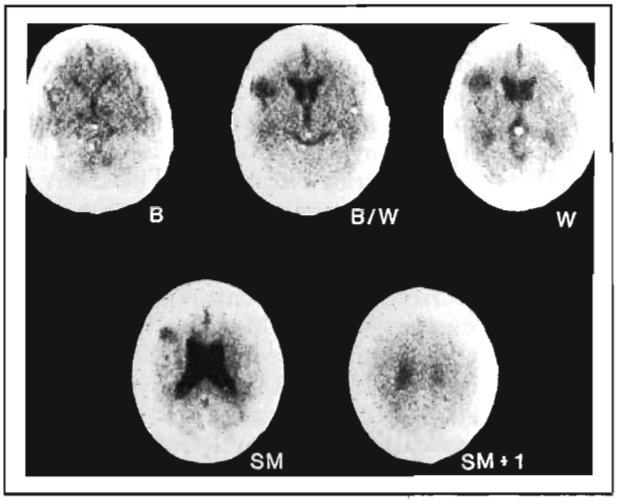

Figure 6. CT of TCM aphasia case with stuttering. Lesion is located in the pars opercularis part of Broca's area (slice of B/W) and in the lower third of the premotor area (slices W and SM). Patient 15, aged 42, 1 year postonset.

Discussion. CT studies demonstrate that the lesions underlying TCMA are predominantly subcortical; the major area of convergence is the white matter anterolateral to the left frontal horn of the lateral ventricle. Extension of the lesions never completely involved the pars triangularis and pars opercularis. The localization seen in our cases is generally compatible with that noted by Rubens,[10] using isotope scans; by Naeser et al, who reported on lesion site[6] and size,[11,12] using CT; and with the pattern described by Damasio and Damasio.[13,14]

Table 5. Statistical correlations

Lesion site	Language parameter	Simple r*	Significance level for simple r	Significance level† for semipartial r
PVWM deep to motor strip for face	Impaired articulation	−0.96	F(1,8) = 87 $p = 0.001$	F(1,6) = 53 $p = 0.001$
Anterior limb of internal capsule, anterior head of caudate, anterior putamen, external capsule, claustrum, extreme capsule, and insula	Auditory comprehension problems	−0.73	F(1,8) = 9.1 $p = 0.05$	F(1,6) = 4.7 $0.05 p = 0.01$
Pars opercularis and lower third of premotor area	Stuttering	+0.68	F(1,10) = 8.6 $p = 0.05$	F(1,9) = 5.5‡ $p = 0.05$

* Lesion site versus language parameter.
† Age and time postonset removed.
‡ Only age removed (see text).

Although none of our patients had complete destruction of Broca's area, extensive lesions of this region on CT may, in exceptional cases, lead to TCMA. Perhaps, in these cases, the cytoarchitectonic field of Broca's area (Brodmann's areas 44 and 45) has been displaced onto the orbital aspect of the frontal operculum. Such displacement would permit entire or partial sparing of this crucial area.[15]

Lesion extension into the periventricular white matter adjacent to the body of the lateral ventricle was associated with impaired articulation. This is the region through which the corticobulbar fibers for face descend from the precentral gyrus en route to the brainstem.[7] Articulatory disturbance in these cases may be secondary to destruction of these fibers.

The mechanism by which the lesions associated with poor comprehension exert their effect is not known. Van Hoesen et al[16] described pathways going from the auditory cortex through the anterior limb of the internal capsule to the head of the caudate. Damasio et al[17] have suggested that damage to these fibers may explain auditory comprehension deficits with subcortical lesions. Galaburda and Pandya[18] demonstrated the presence of pathways between auditory cortex and the frontal lobe traveling through the extreme capsule. Lesions in these fiber tracts may have produced the comprehension deficits seen in our patients. Alternatively, there may not be a simple anatomic explanation for impaired auditory comprehension in this subgroup. Comprehension is determined by so many factors that several behavioral problems (perseveration, apraxia, or inattention) may produce apparent comprehension deficits.

Too little is known about the mechanisms of acquired stuttering to offer an explanation of its occurrence in TCMA. The disorders in these stutterers may be more closely related to aphemia than to TCMA. This would be compatible with the demonstrated lesion site in aphemia, which is a restricted speech production disturbance without significant aphasia.[3]

Medial frontal lesions and aphasia. An important unresolved issue in clarification of TCMA is the nature of the language defect after medial frontal infarction.

Reviewing cases of infarction in the territory of the left anterior cerebral artery up to 1930, Critchley[19] concluded that there were "speech defects of various types," "partly defects of an aphasic order," "partly those of dysarthria," and "some degree of aphasia of the executive type." Seven additional cases have been described in detail[20-24], and there have been brief reports of 12 more.[25-28]

The seven detailed cases had certain features in common. All patients examined early had a period of muteness for days[24] to weeks.[22] Impaired auditory comprehension was common initially, but rapidly improved. As soon as the muteness cleared, repetition and recitation were normal, although word-finding problems were encountered in spontaneous speech.[20,24] Severe agraphia was common, although some recovery could be seen.[22] Major deficits persisted in formulating answers to open-ended questions,[24] in narrative story-telling,[22,23] and in producing word lists.[24] Anatomic data included postmortem,[20,24] isotope scan,[21,22] and CT.[23] All seven patients had medial frontal infarction with involvement of the supplementary motor area.

For the 12 patients with only brief comments about language, the following features are consistent[25-28]: sudden transient muteness, persistently reduced spontaneous speech, minimal responses, and difficulty initiating speech.

Neuroanatomy. The SMA is architectonically intermediate between neocortex and limbic cortex (allocortex). In many ways, the SMA is functionally similar to the anterior cingulate.[29] The SMA has afferent and efferent connections with the cingulate.[25] The anterior cingulate, in turn, has connections with numerous limbic structures through the thalamus (dorsomedial and ventral anterior nuclei) and hippocampus.[30] The SMA also has extensive afferent and efferent connections with motor and premotor cortex.[25] The SMA may be viewed as the most cephalad portion of an integrated brain mechanism responsible for the initiation of speech.[31] Lesions anywhere in the system (periaqueductal gray matter, reticular thalamus, anterior cingulate, and SMA) cause mutism; stimulation produces vocalization. This system, with its rich limbic connections, could explain why we speak. The dominant hemisphere perisylvian region accounts for the structure and content of language: how we produce language. Finally, a large area of frontal lobe is involved in the complex uses to which we can put language. The studies of Milner,[32] Benton,[33] and Luria[34] support this contention. Left prefrontal lesions that do not cause aphasia affect word list generation, explanations of metaphors and proverbs, sentence arrangement, and sequential narration.

Conclusion. Small lesions limited to SMA cause a pure disorder of speech initiation. Destruction of fibers from SMA to frontal premotor cortex may disconnect the limbic "starter mechanism" of speech from the cortical regions that control motor speech.[31] Initially, there may be mutism. As speech recovers, there may be impaired initiation, prolonged response latency, and fluency, but no language errors.[26]

Larger lesions in the deep white matter, above and lateral to the frontal horn, also disconnect the limbic drive from speech control. In addition, these larger lesions disrupt numerous other projection systems within the left hemisphere. As this occurs, more true language abnormalities are seen. Furthermore, there are more limitations in higher-level language use (eg, verbal abstractions) and more "frontal" qualities to language (eg, perseveration). Because inferior posterior frontal cortex and its direct descending path-

ways are spared, speech quality (articulation) is normal. Because lateral inferior suprasylvian pathways are spared, repetition is preserved. This complex is classical TCMA.

The clinical-anatomic mechanism of TCMA proposed here provides a foundation for the many neuropsychological theories that have been suggested to underlie this syndrome.

Acknowledgments

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