

SEVERE NONFLUENCY IN APHASIA

ROLE OF THE MEDIAL SUBCALLOSAL FASCICULUS AND OTHER WHITE MATTER PATHWAYS IN RECOVERY OF SPONTANEOUS SPEECH

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SUMMARY

The relationship between location and extent of lesion on CT scan and limitation in spontaneous speech was examined. The severity of spontaneous speech ranged from cases with no speech or only verbal stereotypies (first major group) to those with reduced, hesitant, poorly articulated, agrammatic speech (nonfluent Broca's aphasia, second major group). CT scan analysis revealed no *single* neuroanatomical area that contained an extensive lesion which could be used to discriminate the most severe cases from the least severe. The two groups were separable, however, on the basis of the CT scan when the extent of the lesion in *two* subcortical white matter areas were *combined*: (1) the most medial and rostral portion of the subcallosal fasciculus plus (2) the periventricular white matter near the body of the lateral ventricle, deep to the lower motor/sensory cortex area for the mouth.

The most rostral portion of the medial subcallosal fasciculus, located in the lateral angle of the frontal horn (extremely deep to Broca's area), contains projections from the cingulate gyrus (area 24) and the supplementary motor area, to the caudate nucleus. We suggest that one explanation for the more severe limitation in spontaneous speech in the first group is the extensive white matter lesion in these two subcortical pathways had interrupted a large number of connections for (1) initiation and preparation of speech movements, and limbic aspects of speech (lesions in the medial subcallosal fasciculus), and (2) motor execution and sensory feedback for spontaneous speech (lesions in periventricular white matter deep to the motor/sensory cortex area for the mouth). Extensive lesion in only one of these two white matter pathway areas, alone was not sufficient to produce long-lasting severe limitation in spontaneous speech and could not be used to discriminate the two groups on the basis of the CT scans. The patients with less severe limitation in spontaneous speech (nonfluent Broca's aphasia) had less extensive lesion within these two white matter areas combined, and had interrupted a smaller number of these subcortical connections. The sites of the lesions in subcortical white matter in CT scans in Broca's original case who could only produce a verbal stereotypy are similar to those in our first group with the most severe limitation in spontaneous speech. The presence or absence of hemiplegia was not related to severity or recovery of spontaneous speech. Careful examination of lesion extent in these two areas of subcortical white matter on CT

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scanning appears to be relevant in predicting potential for recovery of spontaneous speech in some stroke patients.

INTRODUCTION

The anatomical pathways involved in spontaneous speech are not yet well defined, although they have been studied since 1861 when Broca first suggested the importance of the foot of the third left frontal convolution (Broca, 1861*a, b*). Aphasic patients with little or no speech or only stereotypies are particularly challenging cases to study from a neuroanatomical perspective. Bonhoeffer (1914) attributed severely limited spontaneous speech in a patient to subcortical lesions that blocked outflow pathways from Broca's area across (1) the anterior corpus callosum and (2) the anterior limb of the internal capsule. More recently, neuroanatomical pathways associated with speech have been examined through the use of CT scans in stroke patients. Most of these studies (Mohr *et al.*, 1978; Brunner *et al.*, 1982; Knopman *et al.*, 1983; Poeck *et al.*, 1984) have focused primarily on the extent of cortical lesions. Research by ourselves and others has shown that damage in subcortical areas can severely limit spontaneous speech (Hier *et al.*, 1977; Naeser *et al.*, 1982; Alexander *et al.*, 1987).

The purpose of this study was to examine more closely the relationship between location and extent of lesion on CT scans, and the severity of impairment in spontaneous speech ranging from no speech or only stereotypies where no meaningful verbal information is conveyed, to nonfluent agrammatic speech (nonfluent Broca's aphasia), where at least some meaningful verbal information is conveyed.

METHOD

Subjects

The study included 27 right-handed patients with aphasia (24 men, 3 women) with a single episode of left hemisphere occlusive vascular stroke (thromboembolic infarct). Their mean age at onset was 57.6 yrs (SD 7.6; range 35-69 yrs). Each patient had a CT scan performed at either the Boston VA Medical Center (Ohio Nuclear Delta 2010 CT scanner) or the Palo Alto VA Medical Center (Syntex System 60 CT scanner). The CT scans were obtained from 2 months to 9 yrs following stroke onset and were produced at 15-35 deg from the canthomeatal line.

The severity of impairment of speech was determined from the elicited spontaneous speech sample for description of the Cookie Theft Picture from the Boston Diagnostic Aphasia Examination (BDAE; Goodglass and Kaplan, 1972). These samples obtained at the latest testing time following stroke onset (6 months to 9 yrs) were used to assign patients to one of four groups (*see below*), based on type and severity of impairment of spontaneous speech. The classification of patients according to severity of impairment of spontaneous speech was carried out independently from the CT scan analysis.

Group 1 (no speech or only a few irrelevant words)

Group 1 consisted of 7 patients (6 men, 1 woman) who were able to produce either no speech or only a few irrelevant words in describing the Cookie Theft Picture (*see Appendix for speech*

samples). These patients were tested from 9 months to 8 yrs following stroke onset. Their BDAE auditory comprehension z scores ranged from -0.11 to -1.7 (a moderate to severe deficit, mean -0.91 , SD 0.64). Their word repetition ranged from 0-8/10 words (mean 3.7, SD 3.4) and their visual confrontation naming from 0-19/105 (mean 3.6, SD 7.2). Thus not all cases were globally aphasic in all areas of language. (See also Table 1.)

Group 2 (only stereotypies)

Group 2 consisted of 10 patients (all men) who were able to provide only stereotypies in describing the Cookie Theft Picture (see Appendix for speech samples). These patients were tested from 6 months to 9 yrs following stroke onset. Their BDAE auditory comprehension z scores ranged from $+0.09$ to -1.95 (mild/moderate to severe deficit, mean -0.80 , SD 0.74). Their word repetition ranged from 0-7/10 words (mean 2.2, SD 2.7) and their visual confrontation naming ranged from 0-24/105 (mean 2.8, SD 7.6). This group, like Group 1, included cases who were not globally aphasic in all areas of language. (See also Table 1.)

Group 3 (a few words and/or some overlearned phrases)

Group 3 consisted of 5 patients (4 men, 1 woman) who were able to provide a few words and/or some overlearned phrases in describing the Cookie Theft Picture (see Appendix for speech samples). Their spontaneous speech was more difficult to classify and was considered 'borderline' between the most severe cases in Groups 1 and 2, and the least severe cases in Group 4. These patients were tested from 7 months to 4.5 yrs following stroke onset. Their BDAE auditory comprehension z scores ranged from $+0.29$ to -2.1 (mild to severe deficit, mean -0.77 , SD 0.87). Their word repetition ranged from 0-9/10 words (mean 4.2, SD 4.0) and their visual confrontation naming ranged from 0-42/105 (mean 11.8, SD 17.7). This group was similar to Groups 1 and 2 in that not all cases were globally aphasic in all areas of language. (See also Table 1.)

Group 4 (nonfluent Broca's aphasia)

Group 4 consisted of 5 patients (4 men, 1 woman) who were able to provide verbal information relevant to the Cookie Theft Picture with reduced, hesitant, poorly articulated, agrammatic speech (see Appendix for speech samples). The test scores for these patients (other than spontaneous speech) were taken from 5 months to 6 yrs following stroke onset. Their BDAE auditory comprehension z scores ranged from $+0.93$ to $+0.38$ (only mild deficits, mean $+0.69$, SD 0.22). Their word repetition ranged from 6-9/10 words (mean 7.8, SD 1.5) and their visual confrontation naming ranged from 58-101/105 (mean 77.8, SD 17.7). This group was milder in all language modalities than the other three groups. (See also Table 1.)

When t tests were used to compare BDAE scores among the groups, the patients in Group 4 had significantly higher ($P < 0.005$) auditory comprehension z scores and naming scores than the patients in Groups 1, 2 or 3. In addition, the patients in Group 4 had significantly higher ($P < 0.005$) word repetition scores than the patients in Group 2. There were no other significant differences in auditory comprehension, word repetition or naming among the groups.

Hemisphere asymmetries on CT scans

In order to examine whether the severity of impairment in speech was related to some form of 'anomalous dominance', the occipital and frontal length asymmetries in CT scans were measured using a technique previously published by our laboratory (Pieniadz and Naeser, 1984). When all cases were examined together ($n = 27$), the distribution of the occipital length asymmetries was as follows: 65% of the cases had the typical left occipital length; 8%, equal and 27%, right. Those 7 patients with atypical right occipital length asymmetry, who may have some form of anomalous dominance (Pieniadz *et al.*, 1983), were distributed evenly across the groups as follows: Group 1,1; Group 2,2; Group 3,2 and Group 4,2.

TABLE 1. PATIENT DATA AND BOSTON DIAGNOSTIC APHASIA EXAMINATION TEST SCORES, GROUPS 1-4

Case	Sex	Age at onset (yrs)	CT scan (time post-onset)	Occipital length asymmetry	Total lesion extent in subcallosal fasciculus plus middle 1/3 PVWM	Testing (time post-onset)	BDAE auditory comprehension z score	Words (72)	Commands (15)	Word repetition (10)	Visual confrontation naming (105)
Group 1											
1 (S.F.)	F	66	5 mos	L	10	27 mos	-1.60	28.5	2	5	0
2 (L.P.)	M	61	7 mos	R	10	8 yrs	-1.7	30	1	8	6
3 (H.J.)	M	35	9 mos	=	9.95	7 mos	-0.21	51.5	10	0	0
4 (T.F.)	M	65	4 yrs	L	9.25	4 yrs	-1.19	15.5	9	5	0
5 (M.W.)	M	68	2 mos	L	9	2.5 yrs	-0.58	42.5	10	7	19
6 (L.N.)	M	52	16 mos	L	9	15 mos	-1.0	22.5	6	0	0
7 (H.L.)	M	53	9 mos	L	7.98	9 mos	-0.11	60	13	1	0
Group 2											
8 (W.C.)	M	53	3 mos	R	10	3 mos	-1.9	11	4	0	0
9 (D.A.)	M	54	15 mos	L	10	18 mos	-1.34	47	4	0	0
10 (D.E.)	M	56	11 mos	R	10	13 mos	-0.60	53	7	1	0
11 (G.P.)	M	55	9 yrs	L	9.85	9 yrs	+0.05	60	12	5	24
12 (H.M.)	M	58	3 mos	CNM	9.75	8 yrs	-0.90	35	9	5	4
13 (A.G.)	M	64	15 mos	L	8.88	15 mos	-0.94	43	4	0	0
14 (J.N.)	M	53	28 mos	L	8.1	2 yrs	-0.21	50	9	0	0
15 (E.H.)	M	55	7 mos	L	7.88	6 mos	-1.95	13.5	4	4	0
16 (K.M.)	M	59	33 mos	L	7.25	33 mos	-0.33	60	8	0	0
17 (G.J.)	M	59	8 mos	L	7.13	13 mos	+0.09	57	10	7	0
Group 3											
18 (H.D.)	M	63	3 yrs	L	8.73	35 mos	+0.29	57	12	6	42
19 (C.A.)	M	69	5 mos	L	7.43	7 mos	-2.1	10.5	2	0	0
20 (K.W.)	M	61	4 mos	L	6.25	15 mos	-0.90	55.5	5	0	4
21 (A.A.)	M	58	52 mos	R	6	52 mos	-0.70	41	8	9	13
22 (Z.J.)	F	64	11 mos	R	4.25	11 mos	-0.44	57	5	6	0
Group 4											
23 (W.A.)	M	50	44 mos	L	5.88	7 mos	+0.75	66.5	14	9	83
24 (M.E.)	M	58	4 mos	R	5	5 mos	+0.55	60	13	6	62
25 (M.L.)	F	56	16 mos	R	4.75	24 mos	+0.93	71	15	9	85
26 (B.J.)	M	67	12 mos	=	4	17 mos	+0.84	70	15	DNT	101
27 (T.H.)	M	42	6 yrs	L	2.75	6 yrs	+0.38	58.5	12	7	58

The distribution of the frontal length asymmetries was as follows: 36% of the cases had right frontal length; 52%, equal and 12%, left. The 3 patients with atypical left frontal length asymmetry who may have some form of anomalous dominance were distributed evenly across the groups as follows: Group 1,1; Group 2,1; Group 3,0; Group 4,1. This indicates that hemisphere asymmetries were not unusual in each of the groups. It does not appear that severity of impairment in spontaneous speech was related to anomalous dominance as measured by hemisphere asymmetries as demonstrated on CT scanning.

Analysis of CT scan lesion sites

Fig. 1 shows the neuroanatomical areas that were examined by CT scanning for extent of lesion for each patient. The CT scan slice labels B, B/W, W, SM and SM + 1 refer to the Naeser and Hayward (1978) labelling system. Slice B included the following *cortical* areas: Broca's area and the temporal lobe anterior-inferior to Wernicke's area. Slice B also included the following *subcortical* areas: the temporal isthmus (Nielsen, 1946; Naeser *et al.*, 1982); the insular area, putamen, globus pallidus; anterior and posterior limbs of the internal capsule (ALIC, PLIC); caudate nucleus; subcallosal fasciculus (Muratoff, 1893) and extra anterior lesion extension beyond the frontal horn interrupting fibres of the genu of the corpus callosum (Naeser *et al.*, 1982). All these areas are commonly outlined in CT scan atlases (Hanaway *et al.*, 1977; DeArmond *et al.*, 1976; Matsui and Hirano, 1978) or have been discussed in our previous publications on subcortical aphasias (Naeser *et al.*, 1982; Alexander *et al.*, 1987) with the exception of the subcallosal fasciculus (Sc F).

The most medial Sc F is a narrow white matter area surrounding the lateral angle of the frontal horn containing a pathway through which fibres pass from the cingulate gyrus and supplementary

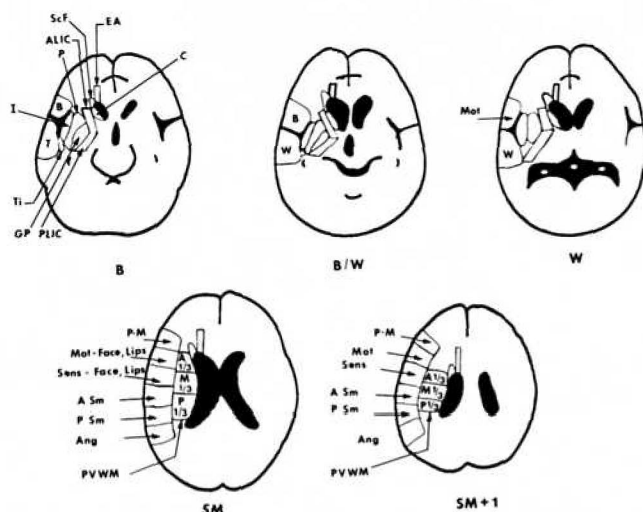


FIG. 1. Location of specific neuroanatomical areas on CT scan slices B, B/W, W, SM and SM + 1 (Naeser and Hayward, 1978, slice labelling system). Each neuroanatomical area was examined for extent of lesion using a 0-5 point scale (0 = no lesion; 5 = entire area has solid lesion, *see text*). B = Broca's area (45 on slice B; 44 on slice B/W); T = temporal lobe anterior-inferior to Wernicke's area on slice B; Ti = temporal isthmus; I = insular structures including insula, extreme capsule, claustrum, external capsule; P = putamen; GP = globus pallidus; ALIC = anterior limb, internal capsule; PLIC = posterior limb, internal capsule; Sc F = medial subcallosal fasciculus; C = caudate; EA = extra anterior extension beyond the frontal horn; W = Wernicke's area (22); Mot = motor cortex; P-M = premotor cortex; Sens = sensory cortex; A Sm = anterior supramarginal gyrus; P Sm = posterior supramarginal gyrus; Ang = angular gyrus; PVWM = periventricular white matter area (A 1/3, anterior 1/3 PVWM; M 1/3, middle 1/3 PVWM; P 1/3, posterior 1/3 PVWM).

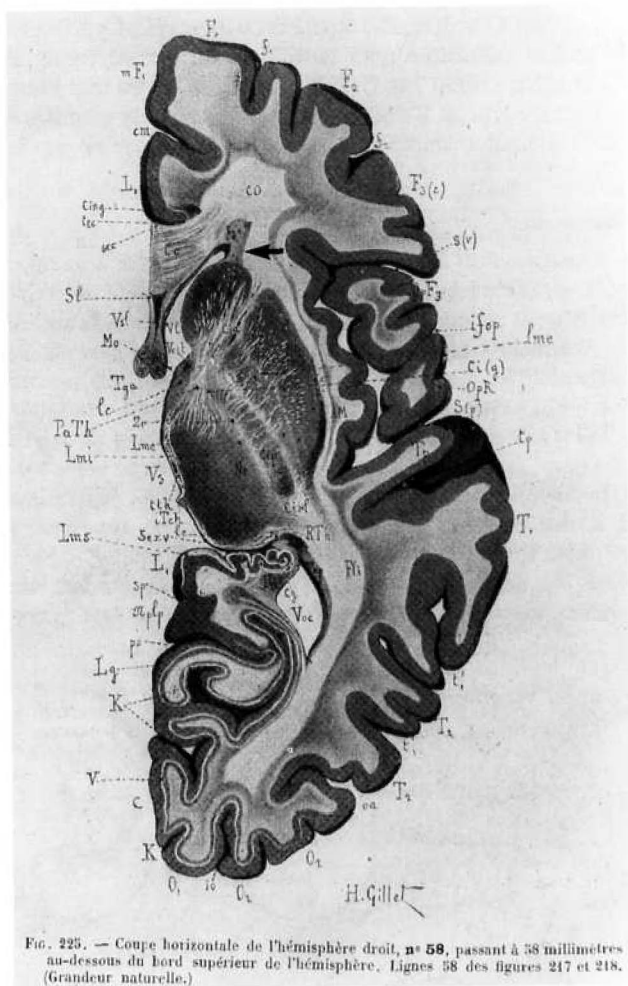


FIG. 225. — Coupe horizontale de l'hémisphère droit, n° 58, passant à 58 millimètres au-dessous du bord supérieur de l'hémisphère. Lignes 58 des figures 217 et 218. (Grandeur naturelle.)

FIG. 2. Drawing in horizontal plane from Dejerine (1895, fig. 225), showing location of the medial subcallosal fasciculus, Sc F (substance grise sous-ependymaire) in the lateral angle of the frontal horn (arrow). Note the small size; the width of the medial Sc F is only approximately one-tenth of the distance from the lateral border of the frontal horn to the cortical mantle.

motor area (SMA) to the caudate. The subcallosal fasciculus was first described by Muratoff (1893) in the dog brain as the 'fasciculus subcallosus'. It is located under the corpus callosum. Dejerine (1895) diagrammed it in the human brain and labelled the most medial portion as substance grise sous-ependymaire (Sge, see fig. 2). This most medial portion is very narrow and, in fact only one-tenth of the distance from the lateral border of the frontal horn to the cortical mantle. (This represents only a few millimetres on the CT scan.) Yakovlev and Locke (1961) have diagrammed these cingulate and SMA projections to the caudate in more detail in the monkey brain. In their work the most medial portion of the Sc F is labelled stratum subcallosum (St Sbc, see fig. 3).

The cortical and subcortical areas examined on slices B/W (mid-third ventricle level) and W (roof of third ventricle level) are similar to most of those examined on slice B with the exception of the

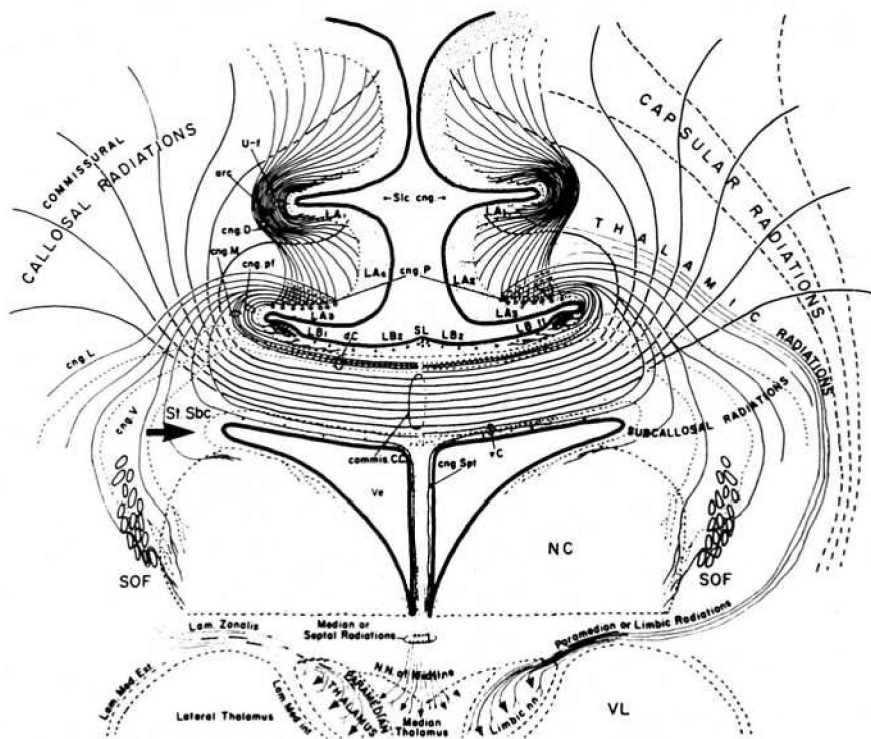


FIG. 3. Drawing in coronal plane from Yakovlev and Locke (1961, fig. 6), showing location of the medial subcallosal fasciculus (stratum subcallosum St Sbc) in the lateral angle of the frontal horn (arrow). Note that the connections from the cingulate gyrus and supplementary motor area to the head of the caudate are located within the St Sbc area immediately lateral to the frontal horn. (Reprinted with permission.)

addition of Wernicke's area on slices B/W and W and motor cortex on slice W; the remainder of the subcortical areas are self-explanatory.

Slice SM (bodies of lateral ventricle level) included the following cortical areas: premotor; motor; sensory; anterior supramarginal; posterior supramarginal; angular. The motor and sensory cortices at this slice level primarily represent the mouth area. Slice SM also included the following subcortical areas: extra anterior lesion extension; Sc F; anterior one-third periventricular white matter (PVWM); middle one-third PVWM and posterior one-third PVWM. Ross (1980) has shown how the descending pyramidal tracts are located immediately adjacent to the body of the lateral ventricle. Thus the deepest periventricular white matter areas, especially the middle-to-posterior one-third PVWM areas contain, in part, these descending pyramidal tract pathways. These PVWM pathways are diagrammed coronally in fig. 4.

The cortical and subcortical areas examined on slice SM+1 are similar to those examined on slice SM.

Assessment of extent of lesion within each neuroanatomical area

Each neuroanatomical area was assessed visually for extent of lesion within that area using the following scale: 0 = no lesion; 1 = equivocal lesion; 2 = small, patchy or partial lesion; 2.5 = patchy, less than half of area has lesion; 3 = half of area has lesion; 3.5 = patchy, more than half of area has lesion; 4 = more than half of area has solid lesion; 5 = total area has solid

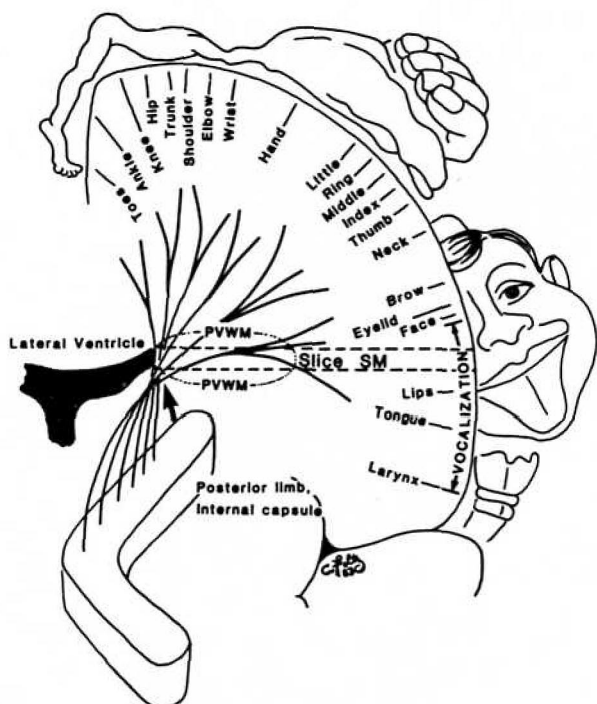


FIG. 4. Coronal diagram showing location of descending pyramidal tract pathways in the deepest, subcortical periventricular white matter (PVWM) area immediately adjacent to the body of the lateral ventricle (arrow). On CT scan these descending pyramidal tract pathways are located in the PVWM on slices SM and SM + 1. On the CT scan slices inferior to these, the pyramidal tract pathways are located in the posterior limb of the internal capsule (CT scan slices W, B/W and B). See also fig. 1.

lesion. We have used a similar rating scale in previous CT scan studies with an interrater reliability coefficient of 0.93 (Borod *et al.*, 1984).

The lesion extent data were compared between each group for each neuroanatomical area using Mann-Whitney U tests. These data were compared between each group for each neuroanatomical area on each separate CT scan slice, as well as on combined CT scan slices when a given neuroanatomical area was represented on more than one CT scan slice. For example, Broca's area is represented on slices B and B/W; the extent of lesion in Broca's area was therefore examined on slice B alone, as well as slice B/W alone, as was the mean extent of lesion on slices B and B/W combined.

RESULTS

No significant differences ($P < 0.01$) were observed in lesion site data between the aphasia patients with No Speech (Group 1) versus those with Stereotypies (Group 2). The lesion site data from these two groups were therefore combined, forming a No Speech/Stereotypies Group ($n = 17$) for comparison with the Nonfluent Broca's Group ($n = 5$). (The lesion site data for patients who used only a few words and/or some overlearned phrases, Group 3, are discussed later.)

Significant differences ($P < 0.01$) in extent of lesion were observed between

the No Speech/Stereotypies Group vs the Nonfluent Broca's Group for the neuroanatomical areas listed in Table 2. The No Speech/Stereotypies Group always had greater extent of lesion in the specific neuroanatomical areas than did the Nonfluent Broca's Group.

TABLE 2. SIGNIFICANT DIFFERENCES (MANN-WHITNEY U-TESTS, $P < 0.01$) IN LESION SITE DATA BETWEEN THE NO SPEECH/STEREOTYPIES GROUP VS THE NONFLUENT BROCA'S GROUP

	<i>No Speech/ Stereotypies Group</i>		<i>Nonfluent Broca's Group</i>		<i>P value</i>
	<i>X</i>	<i>SD</i>	<i>X</i>	<i>SD</i>	
Slices B and B/W (Mean lesion extent, 2 slices)					
*Subcallosal fasciculus	4.41	0.81	2.03	1.68	< 0.005
Anterior limb, internal capsule	4.88	0.21	2.5	1.61	< 0.009
Putamen	3.64	1.37	1.2	1.50	< 0.009
Posterior limb, internal capsule	2.40	1.83	0	0	< 0.009
Temporal isthmus	3.69	1.36	0.25	0.5	< 0.0002
Slice W					
Putamen	4.26	1.46	2	1.41	< 0.009
Slice SM					
*Middle 1/3 PVWM	4.71	0.47	2.45	0.87	< 0.0003
Posterior 1/3 PVWM	3.85	1.42	0.4	0.8	< 0.0009
Anterior supramarginal gyrus	3.5	1.92	0.4	0.8	< 0.009
Slice SM + 1					
Middle 1/3 PVWM	4.59	0.71	1.78	1.91	< 0.003
Posterior 1/3 PVWM	4.00	1.66	0.8	1.6	< 0.005
Anterior supramarginal gyrus	3.37	1.58	0.2	0.4	< 0.002

Means lesion extent in each area: 0 = no lesion; 5 = total lesion.

* Areas which when summed, produce no overlap between the no speech/stereotypies cases vs the nonfluent Broca's cases.

Each of these areas where significant differences were observed is a subcortical lesion site area with the exception of the anterior supramarginal gyrus area. When the lesion extent values for each individual case were examined in each of these significantly different neuroanatomical areas, there was no single neuroanatomical area which alone could discriminate the 17 No Speech/Stereotypies cases vs the 5 Nonfluent Broca's cases. There were, however, two lesion site areas which, when summed, produced no overlap between the No Speech/Stereotypies cases vs the Nonfluent Broca's cases. These two lesion site areas were subcortical areas including (1) the medial Sc F (mean lesion extent over slices B and B/W) plus (2) the middle one-third PVWM (slice SM). These two areas are shown in the shaded areas in fig. 5. No other lesion site combination could be used to discriminate these 22 cases into their respective groups.

The medial Sc F at slices B and B/W contains the common pathway through which fibres pass from the cingulate gyrus and SMA to the caudate. The middle one-third PVWM at slice SM contains subcortical white matter fibres deep to the lower motor/sensory cortex area for the mouth. In addition, this middle one-third

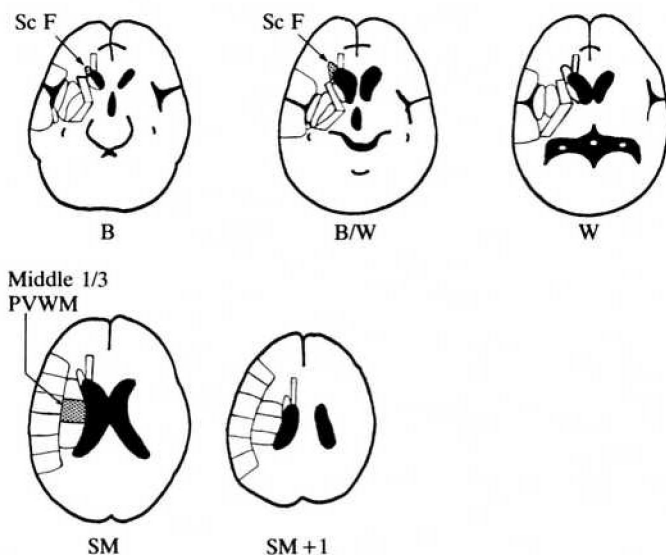


FIG. 5. Location on CT scan slices of the two deep subcortical white matter areas which, when examined for total extent of lesion *combined*, discriminated the cases with no speech or only stereotypies versus those with nonfluent Broca's aphasia. These two deep subcortical white matter areas included (1) the medial Sc F, mean lesion extent at slices B and B/W plus (2) the white matter deep to the lower motor/sensory cortex area for the mouth, the middle one-third PVWM at slice SM.

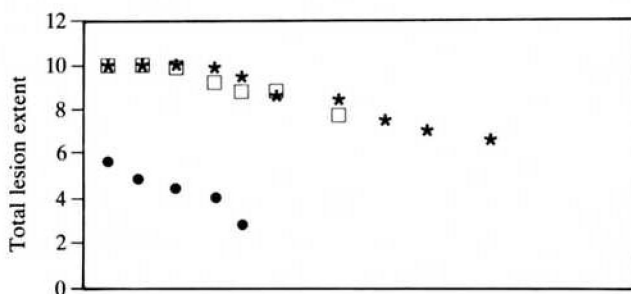


FIG. 6. Values for total lesion extent on the CT scan in the two deep subcortical white matter areas *combined*: (1) the medial Sc F area (mean lesion extent at slices B and B/W) plus (2) the white matter area deep to the lower motor/sensory cortex area for mouth, middle one-third PVWM at slice SM, for individual cases in three groups. Note that all cases with the most severe limitation in speech (Groups 1 and 2) had total lesion extent values above 7; all cases with the least severe limitation in speech (Group 4, Broca's aphasia) had total lesion extent values below 6: The summed maximum lesion extent value of 10 on the graph represents maximum lesion extent ratings of 5 (entire area has solid lesion) in each of the two deep subcortical white matter areas combined. Open squares = no speech; stars = stereotypies; closed circles = nonfluent Broca's aphasia.

PVWM area contains, in part, corona radiata, arcuate fasciculus and additional pericingulate fibres projecting to perisylvian cortical language areas.

The combined lesion extent values for these two subcortical areas are plotted for each No Speech/Stereotypies case and each Nonfluent Broca's case in fig. 6. This graph shows that there was no overlap in these combined lesion extent

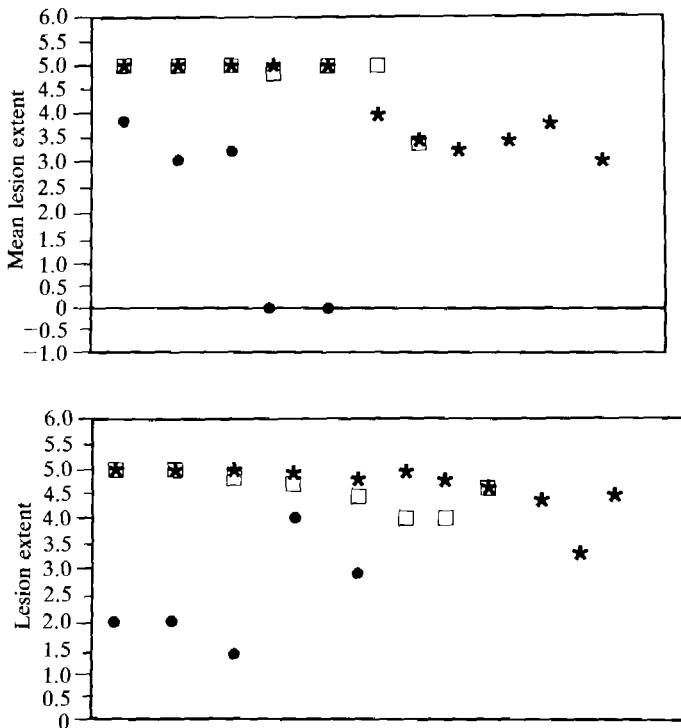


FIG. 7. *Top*, lesion extent values *only* in the medial Sc F area (mean lesion extent at slices B and B/W) for individual cases in three groups. Note that there was complete overlap in the lesion extent values for the medial Sc F for cases with the most severe limitation in speech (Groups 1 and 2) versus those with the least severe limitation in speech (Group 4, Broca's aphasia). *Bottom*, graph showing lesion extent values *only* in the white matter deep to the lower motor/sensory cortex area for mouth, middle one-third PVWM at slice SM, for the same three groups. Note that there was complete overlap in the lesion extent values for the middle one-third PVWM for cases with the most severe limitation in speech (Groups 1 and 2) versus those with the least severe limitation in speech (Group 4, Broca's aphasia). These two graphs show why it is necessary to *combine* the lesion extent values in the two deep subcortical areas mentioned here (*see* fig. 6).

values between the No Speech/Stereotypies cases and the Nonfluent Broca's cases. All the No Speech/Stereotypies cases had summed lesion extent scores above 7 and all the Nonfluent Broca's cases had summed lesion extent scores below 6.

The extent of lesion values in the medial Sc F alone (mean lesion extent values on slices B and B/W) are plotted in the top half of fig. 7 for the No Speech/Stereotypies cases and the Nonfluent Broca's cases. This graph shows that there was overlap in the lesion extent values for the medial Sc F between the two groups; that is, the mean lesion extent values in the medial Sc F alone, were not adequate to discriminate these two very different groups of patients.

The lesion extent values in the middle one-third PVWM alone (white matter deep to the lower motor/sensory cortex area for mouth at slice SM) are plotted in the lower half of fig. 7 for the No Speech/Stereotypies cases and the Nonfluent Broca's cases. This graph shows that again there was overlap in the lesion extent

values between the two groups. Thus the lesion extent values in the middle one-third PVWM, alone, also were not adequate to discriminate these two very different groups of patients. It was only when the lesion extent values were combined for these two lesion site areas, medial Sc F at slices B and B/W plus the middle one-third PVWM at slice SM (fig. 6), that the two groups were successfully discriminated on the basis of CT scan lesion extent values.

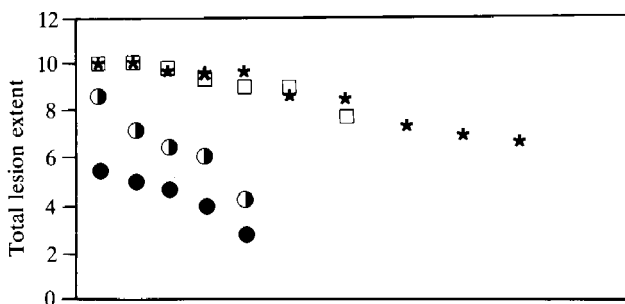


FIG. 8. Graph showing total lesion extent values on the CT scan in two deep subcortical white matter areas combined (1) the medial Sc F area (mean lesion extent at slices B and B/W) plus (2) the white matter area deep to the lower motor/sensory cortex area for the mouth, middle one-third PVWM at slice SM, for cases in all four groups. Note that most cases in Group 3, the group with speech which was better than that of the first two groups but worse than that of the fourth, had total lesion extent values which were often between those for the first two groups and the fourth group. Open squares = no speech; stars = stereotypies; closed circles = nonfluent Broca's aphasia; half-filled circles = few words, overlearned phrases.

Fig. 8 shows the combined lesion extent values for the two most important areas, medial Sc F area (mean at slices B and B/W) plus the middle one-third PVWM area at slice SM, for all four groups in this study. As mentioned above for fig. 6, this shows that the No Speech/Stereotypies cases had the highest lesion extent values in these two areas combined, and the Nonfluent Broca's, the lowest values. The combined lesion extent values for these two most important areas for the cases in Group 3 (those with a few words and/or some overlearned phrases) basically fell between the combined lesion extent values for the No Speech/Stereotypies cases and those values for the Nonfluent Broca's cases, although a few of these Group 3 cases overlapped into the range for the most severe cases (Groups 1 and 2) and the least severe cases (Group 4). These borderline Group 3 cases are discussed further, below.

The 2 more severe cases in Group 3 (Cases 18, 19) were perhaps 'borderline' in their classification into that group (*see* speech samples in Appendix). These 2 more severe cases in Group 3 also had larger combined lesion extent values for the medial Sc F area plus the middle one-third PVWM area (8.73 and 7.43). These combined lesion extent values overlapped into the range for the No Speech/Stereotypies cases (values above 7).

The least severe case in Group 3 (Case 22) was also perhaps 'borderline' in her classification. Her speech was hesitant, poorly articulated and agrammatic, more

compatible with the Nonfluent Broca's Group; however, her comprehension was too severely impaired (BDAE auditory comprehension z score of -0.44 for her to be categorized as having classical Broca's aphasia. The combined lesion extent value for the medial Sc F area plus the middle one-third PVWM area was only 4.25 in this case, a value which overlapped into the range for the Nonfluent Broca's cases (combined lesion extent values below 6).

In summary, the cases with the most severe impairment in spontaneous speech, those with No Speech or only Stereotypies (Groups 1 and 2), had combined lesion extent values for these two areas of above 7, and cases with the least severe impairment, those with Nonfluent Broca's aphasia (Group 4), had combined values of below 6. Those cases who fell between these two groups in terms of severity of impairment of spontaneous speech, namely those with a few words and/or some overlearned phrases, basically fell between these two groups in terms of combined lesion extent values (around 6). There were exceptional cases at either extreme within Group 3. A few sample cases and CT scans from each of the four groups are discussed separately, below.

ILLUSTRATIVE CASES

Group 1 (no speech or only a few irrelevant words)

Case 3

H.J., a 35-yr-old male, at 9 months following stroke onset still had no speech, although he could phonate and produce grunt-like sounds. He had dense right hemiplegia. The CT scan in fig. 9 shows a primarily subcortical infarct which included a large lesion in the medial Sc F at slices B and B/W and large lesion in the middle one-third PVWM at slice SM. The total lesion extent in the two areas combined was 9.95 (see fig. 9).

H.J. has subcortical global aphasia and has not previously been reported. The subcortical lesion and spontaneous speech in this case are compatible with our previously published cases of subcortical global aphasia who had lesions in the capsular-putaminal area with anterior-superior lesion extension into PVWM, posterior lesion extension across the temporal isthmus interrupting auditory pathway fibres to the temporal lobe, and extra anterior lesion extension beyond the frontal horn interrupting fibres of the genu of the corpus callosum (Naeser *et al.*, 1982). These cases may have some cortical involvement in the insula, as does this case; however, the recent study by Alexander *et al.* (1987) has shown that cortical lesions in the insula and subjacent white matter produce only a mild conduction aphasia, and are not associated with global aphasia. The spontaneous speech in these subcortical global aphasia cases is not different from those with large cortical/subcortical lesions (see fig. 10). This case is a good example of how a small primarily subcortical lesion can produce severe deficits in terms of language behaviour, and that it can be lesion site, not lesion size, which is critical in determining the resultant language behaviour.

Case 4

T.F., a 65-yr-old male, at almost 4 yrs postonset, still had no speech and produced only unintelligible speech sounds, e.g., 'juh . . . ah . . . jou . . .', etc. (see Appendix). A dense right hemiplegia was present. The CT scan in fig. 10 shows a large cortical/subcortical infarct which included a large lesion in the medial Sc F at slices B and B/W and large lesion in the middle one-third PVWM at slice SM. The total lesion extent in the two areas combined was 9.25 (see fig. 10).

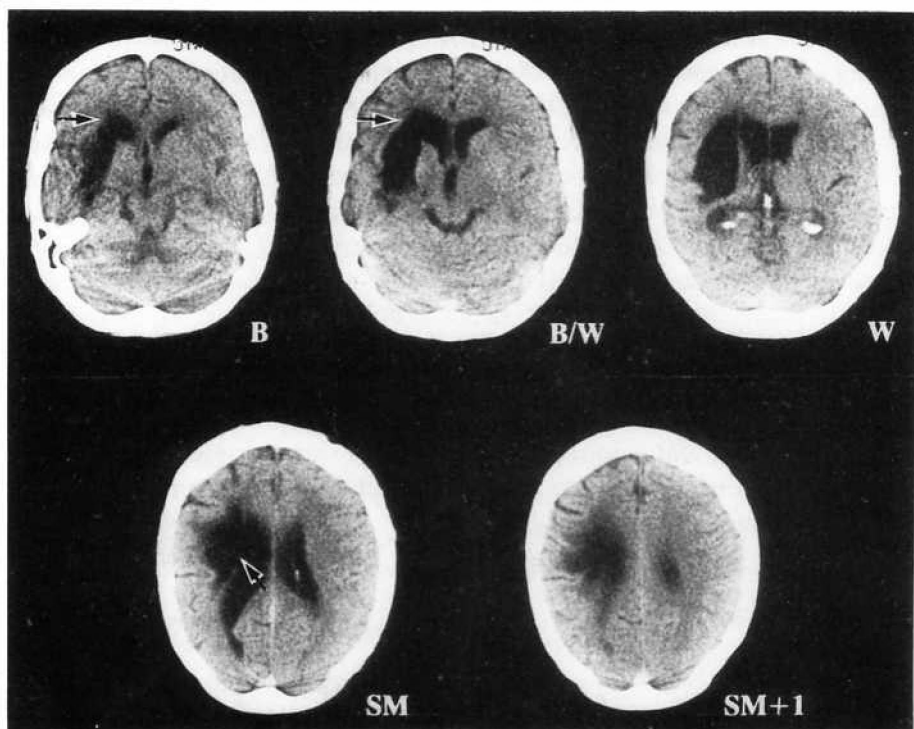


FIG. 9. CT scan at 9 months following stroke onset in *Case 3* (Group 1). A dense right hemiplegia was present. The left hemisphere lesion is on the left side of the CT scan. Lesion extent in the medial Sc F at slice B was rated 5; and at slice B/W, also 5 (see arrows); mean 5. Lesion extent in the middle one-third PVWM at slice SM was rated 4.95 (see arrow); total lesion extent, 9.95. Note that the entire lesion is primarily subcortical.

The cortical/subcortical lesion sites and language behaviour in *Case 4* are compatible with previously published cortical/subcortical global aphasia cases (Naeser and Hayward, 1978; Kertesz, 1979; Damasio, 1981; Naeser, 1983). The subcortical CT scan lesion sites in *Case 3*, fig. 9, can be superimposed over the cortical/subcortical lesion sites in *Case 4*, fig. 10. Although the cortical lesion in *Case 4* increased the overall lesion size, the severity of the deficit in spontaneous speech was equal in both cases. The vascular aetiology in *Case 3* was likely to have been an occlusion of the left internal carotid artery (Naeser *et al.*, 1982); that in *Case 4* was probably total occlusion of the left middle cerebral artery, including both superior and inferior divisions.

Group 2 (only stereotypies)

Case 16

K.M., a 59-yr-old male, at 33 months following stroke onset, could produce only the stereotypy 'morning, morning' or 'boy, boy'. He had only mild incoordination of fine right finger movements, without hemiparesis. The CT scan in fig. 11 shows an extensive lesion in the medial Sc F at slice B, and only a patchy lesion at slice B/W. An extensive lesion was also present in the lateral portion of the middle one-third PVWM (patchy, greater than half of the area affected). The total lesion extent for the two areas was 7.25 (see fig. 11).

It is probable that this case had no hemiparesis because the lesion did not extend completely across the middle-to-posterior one-third PVWM adjacent to the body of the lateral ventricle at

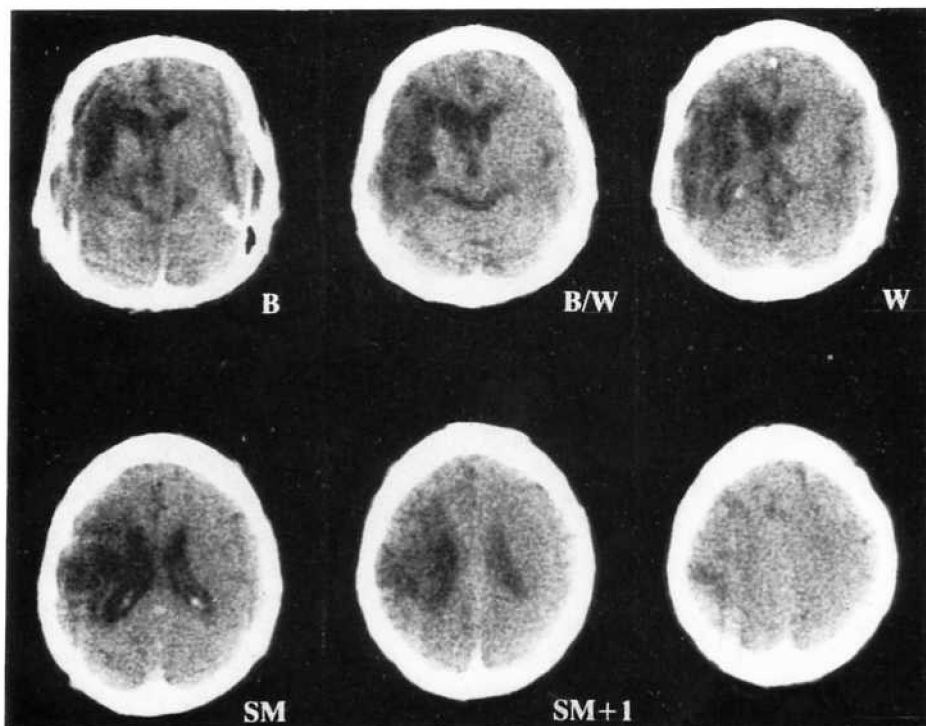


FIG. 10. CT scan at 47 months following stroke onset of *Case 4* (Group 1). A dense right hemiplegia was present. Lesion extent in the medial Sc F at slice B was rated 5; slice B/W, 4.5; mean 4.75. Lesion extent in the middle one-third PVWM at slice SM was rated 4.5; total lesion extent, 9.25. The lesion was a large frontal, parietal, temporal lobe infarct involving cortical as well as subcortical structures in the territory of the entire left middle cerebral artery.

slices SM or SM+1. The deepest subcortical descending motor pathways were therefore not completely interrupted. Note the depth of the location of the PVWM motor pathways in fig. 4. This case emphasizes the importance of examining the deepest few mm in the PVWM on the CT scan at slices SM and SM+1 when assessing potential for recovery of hemiplegia. Of course no lesion was present in the PLIC or lower motor pathways on the lower CT slices W, B/W or B, etc. This case makes the important point that the presence or absence of hemiplegia is not always a good prognostic indicator of potential for recovery of spontaneous speech. At almost 3 yrs following stroke onset there was no hemiplegia but he produced no relevant speech.

Case 17

G.J., a 59-yr-old male, at 13 months following stroke onset could only use the stereotypies, '1, 2, 3, 4, 5' or 'boom, boom'. A dense right hemiplegia was present. The CT scan in fig. 12 shows a patchy but definite lesion in the medial Sc F at slices B and B/W and an extensive lesion in the middle one-third PVWM at slice SM. The total lesion extent was 7.13 (*see* fig. 12).

This case exemplifies the necessity of examining extremely carefully the few mm in the lateral angle of the left frontal horn at slices B and B/W. This case had essentially no lesion in Broca's cortical area and the only area where a lesion was present on slices B and B/W included the small

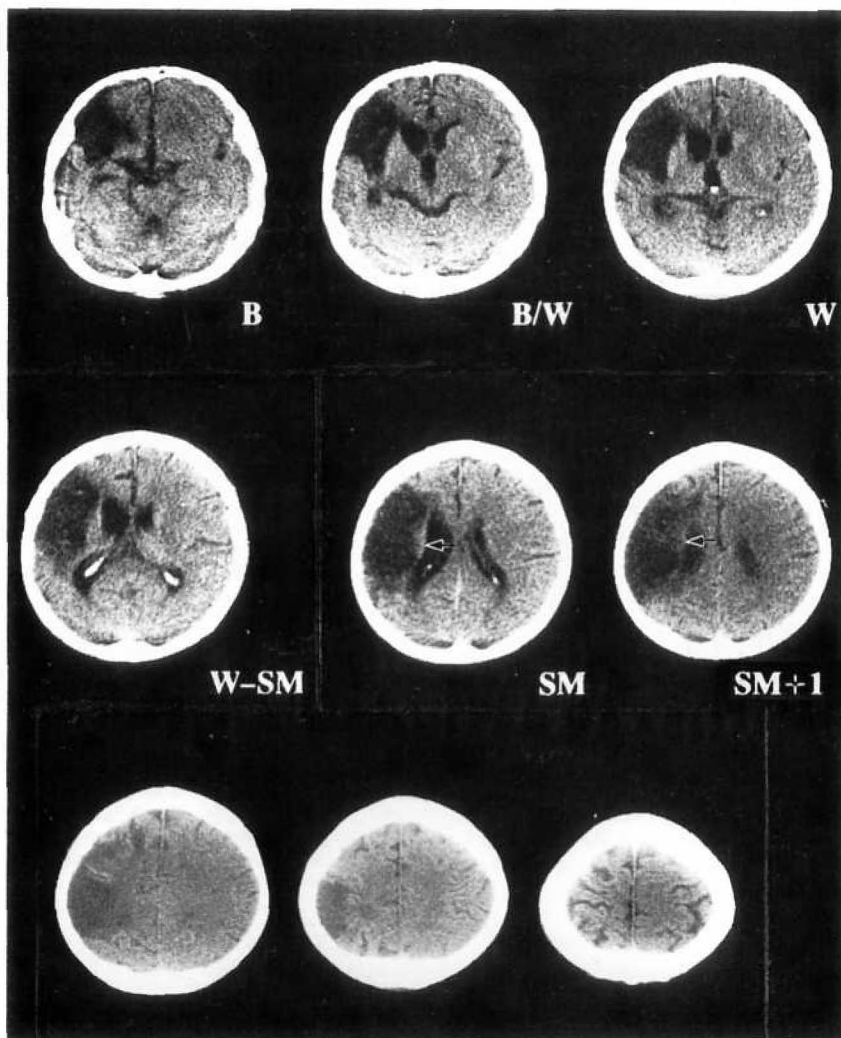


FIG. 11. CT scan at 33 months following stroke onset of *Case 16* (Group 2). No right hemiparesis was present. Lesion extent in the medial Sc F at slice B was rated 5; slice B/W, 2.5; mean 3.75. Lesion extent in the middle one-third PVWM at slice SM was rated 3.5; total lesion extent 7.25. The absence of hemiparesis in this case was compatible with the absence of a lesion in the deepest middle-to-posterior PVWM area adjacent to the body of the lateral ventricle at slices SM and SM+1 (arrows). This deepest PVWM area contains, in part, the descending pyramidal tract pathways (see also fig. 4). No cortical or subcortical lesion was present in arm or leg motor areas near the vertex.

deep subcortical white matter in the area of the medial Sc F, extremely deep to Broca's area, adjacent to the lateral angle of the frontal horn.

Group 3 (a few words and/or some overlearned phrases)

Case 20

K.W., a 61-yr-old male, at 15 months following stroke onset was able to say a few phrases such as 'There, too . . . there too . . . um . . . I don't know . . .', etc. (see Appendix). A right hemiplegia

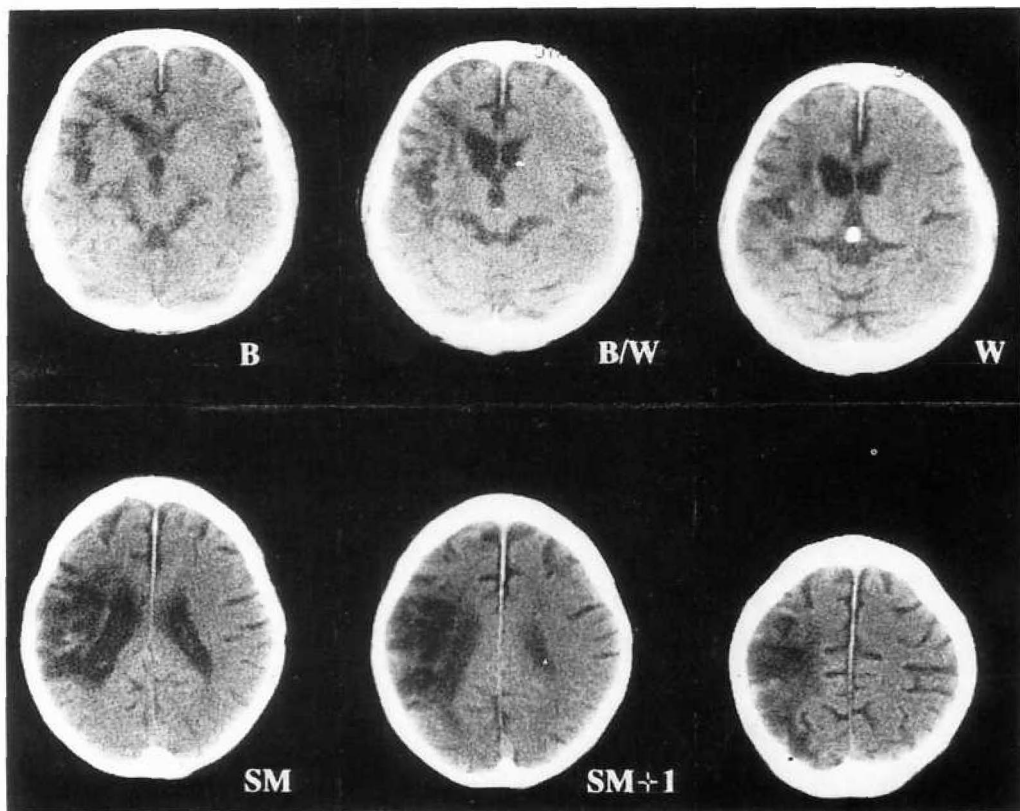


FIG. 12. CT scan at 8 months following stroke onset in *Case 17* (Group 2). A dense right hemiplegia was present. Lesion extent in the medial Sc F at slice B was rated 3.75; slice B/W, 2.5; mean 2.63. Lesion extent in the middle one-third PVWM at slice SM was rated 4.5; total lesion extent 7.13. Note that there was no cortical or immediately subcortical lesion in Broca's area on slices B or B/W.

was present. The CT scan in fig. 13 shows only a partial lesion in the medial Sc F, especially at slice B/W-W and an incomplete lesion (patchy, greater than half of area affected) in the middle one-third PVWM at slice W-SM. The total lesion extent for the two areas was 6.25 (see fig. 13).

This is a good example of a case whose speech was between that of the cases in Groups 1 and 2 (no speech or only stereotypies) and that of the cases in Group 4 (nonfluent Broca's) and whose total lesion extent in the two areas also was between that of the other two groups, i.e., Groups 1 and 2, above 7 and Group 4, below 6.

Case 22

Z.J., a 68-yr-old female, at 8 months following stroke onset had speech compatible with nonfluent Broca's aphasia, but too severe a comprehension deficit to be diagnosed as typical Broca's aphasia (see Appendix). No hemiparesis was present. The CT scan in fig. 14 shows only a minimal lesion in the medial Sc F at slice B and no lesion in the medial Sc F at slice B/W. In fact, the medial Sc F is elegantly spared at slice B/W, with a solid lesion lateral to it, extending to, and including, Broca's cortical area. A lesion was present in only about half of the middle one-third PVWM area at slice SM-SM+1. The total lesion extent for the two areas was only 4.25 (see fig. 14).

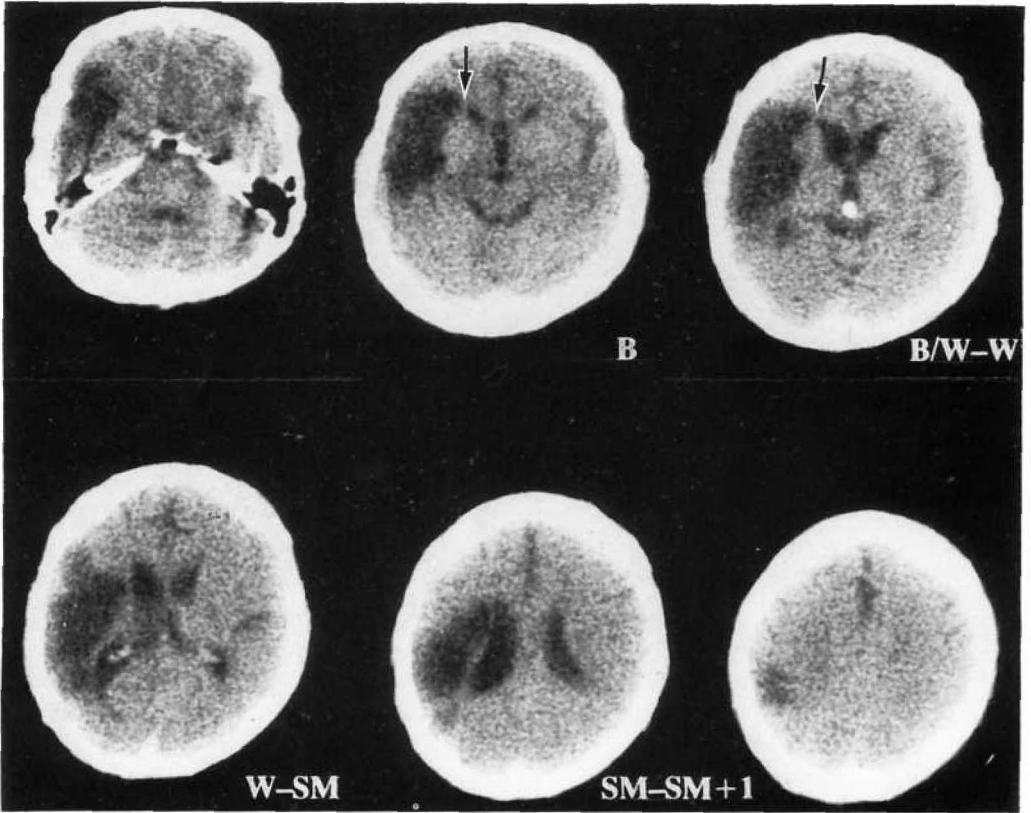


FIG. 13. CT scan at 4 months following stroke onset in *Case 20* (Group 3). A right hemiplegia was present. Note the deep PVWM lesion at slice SM-SM+1. Arrows show increased sparing in the medial Sc F at slices B and B/W-W. Lesion extent in the Sc F at slice B was rated only 2.5; slice B/W-W, only 2.5; mean 2.5. Lesion extent in the middle one-third PVWM at slice W-SM was rated 3.75; total lesion extent 6.25.

It is probable that this case had no hemiparesis because the lesion did not extend completely across the middle-to-posterior one-third PVWM adjacent to the body of the lateral ventricle at slice SM-SM+1. The deepest subcortical descending motor pathways were not completely interrupted (similar to *Case 16*, fig. 11.) Group 2, *Case 16*, fig. 11, however, had a more extensive lesion in the medial Sc F at both slices B and B/W, than did this *Case 22* from Group 3, fig. 14. *Case 16* was not hemiparetic, but he produced only stereotypies. *Case 22* also was not hemiparetic, and she had nonfluent agrammatic speech which was relevant to the cookie theft picture. These 2 cases differ in lesion site primarily in the deepest subcortical white matter surrounding the lateral angle of the left frontal horn at slices B and B/W, i.e., the medial Sc F at this level (compare figs 11, 14).

In summary, *Case 22* is important because it shows how close a lesion can be to the border of the lateral angle of the left frontal horn and still spare the medial Sc F (see slice B/W) as well as sparing the deeper portion of the middle one-third PVWM area at slice SM, and thus allowing the patient to recover some spontaneous speech. The sparing of the deepest portion of the middle-to-posterior one-third PVWM adjacent to the body of the lateral ventricle at slice SM-SM+1 is also likely to have spared the deepest descending motor pathways for the arm and leg, so that the patient was not hemiparetic.

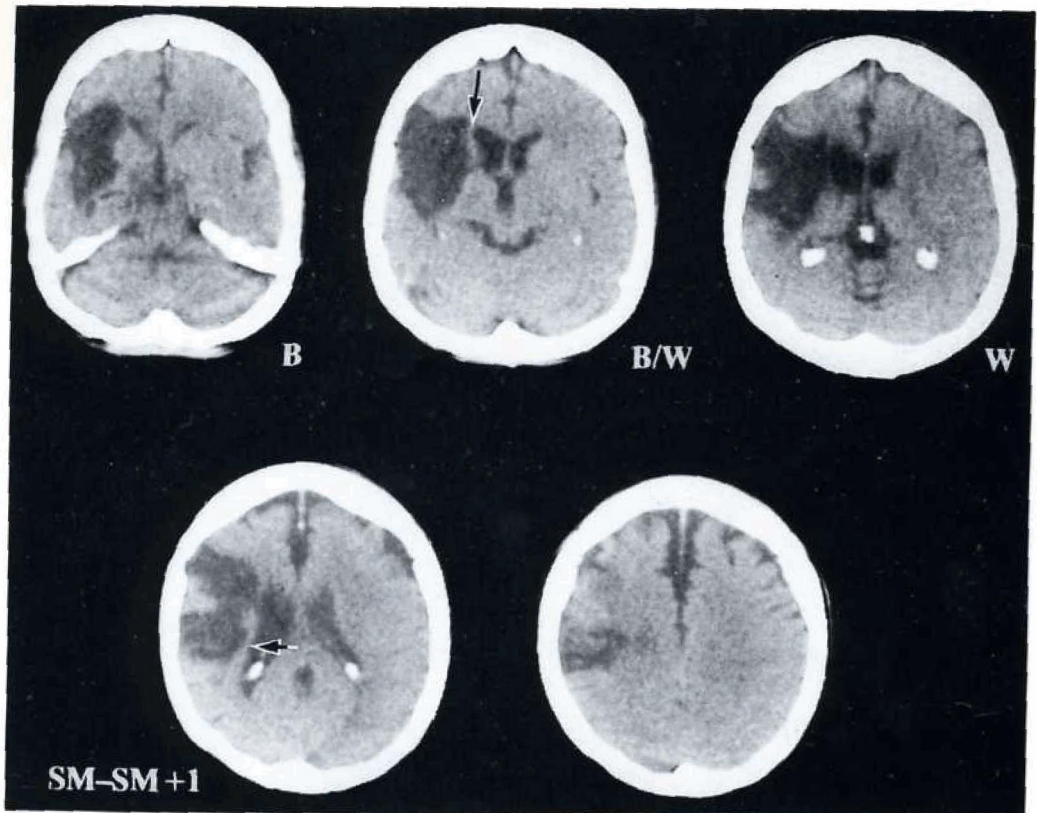


FIG. 14. CT scan at 11 months following stroke onset in *Case 22* (Group 3). No right hemiparesis was present. Lesion extent in the medial Sc F at slice B was rated only 2.5; slice B/W, 0; mean 1.25. Arrow shows no lesion in the medial Sc F at slice B/W, despite a large lesion lateral to it, extending outwards to include Broca's cortical area. Lesion extent in the middle one-third PVWM at slice SM-SM+1 was rated 3; total lesion extent 4.25. The absence of hemiparesis in this case was compatible with the absence of a lesion in the deepest middle-to-posterior one-third PVWM area adjacent to the body of the lateral ventricle at slice SM-SM+1 (arrow). This deepest PVWM area contains, in part, the descending pyramidal tract pathways (see also figs 4, 11).

Group 4 (nonfluent Broca's aphasics)

Case 23

W.A., a 50-yr-old male, at 7 months following stroke onset produced nonfluent agrammatic speech which was compatible with Broca's aphasia (see Appendix). A mild hemiparesis was present and there was good recovery. The CT scan in fig. 15 shows that although there was an extensive lesion in the medial Sc F at slices B and B/W, there was only a minimal lesion in the middle one-third PVWM at slice SM (only a small patchy lesion). The total lesion extent in the two areas was 5.88 (see fig. 15).

This case has the typical lesion distribution associated with longer-lasting Broca's aphasia which we have repeatedly observed in our laboratory. (The Broca's aphasics who were included in this study were still nonfluent and agrammatic at 7 months to 6 yrs following stroke onset.) This lesion distribution usually includes infarction in parts of Broca's area which extends across to the border of the frontal horn (including medial Sc F, slices B and/or B/W), plus superior lesion extension into

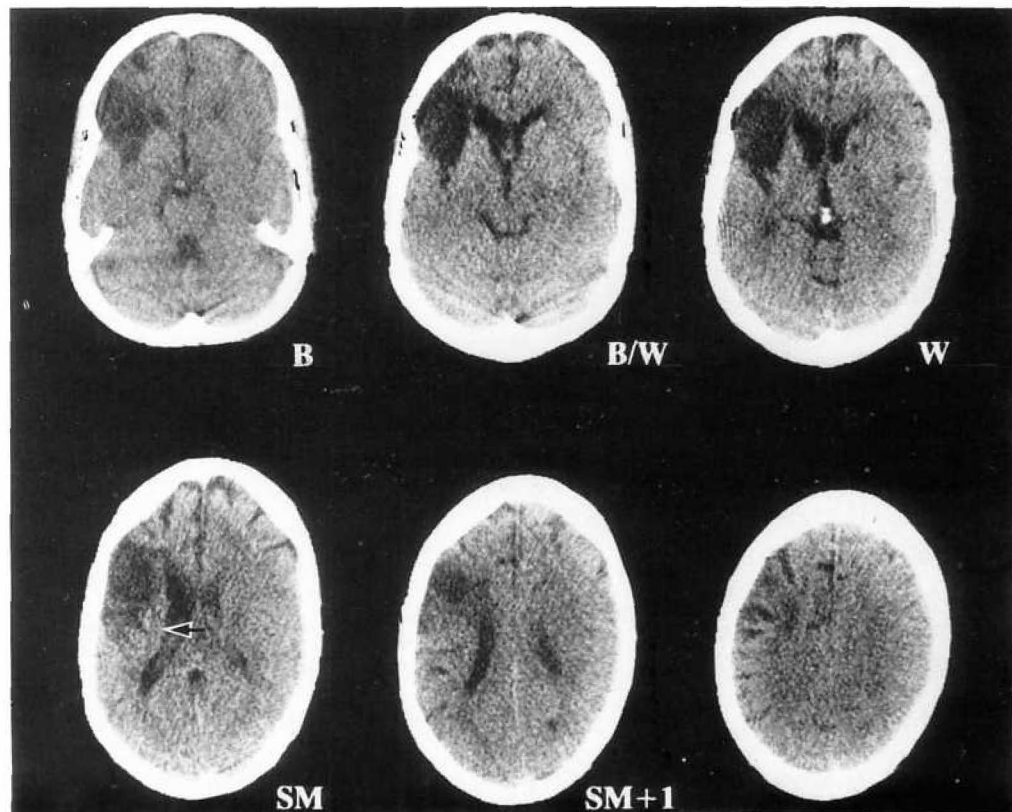


FIG. 15. CT scan at 44 months following stroke onset in *Case 23* (Group 4). A mild hemiparesis was present and there was good recovery. Lesion extent in the medial Sc F at slice B was rated 4; slice B/W 3.75; mean 3.88. Lesion extent in the middle one-third PVWM at slice SM was rated only 2; total lesion extent 5.88. The arrow at slice SM shows a minimal lesion in the middle one-third PVWM which greatly reduced the combined total lesion extent to below 6, a value compatible with his mild limitation in speech. The mild hemiparesis with good recovery in this case was compatible with the smaller lesion in the deepest PVWM area immediately adjacent to the body of the lateral ventricle at slices SM and SM+1. This deepest PVWM area contains, in part, the descending pyramidal tract pathways (*see also* figs 4, 11, 14).

the lower motor cortex area for the mouth (slices W and SM), which extends into the deep anterior one-third PVWM area, and sometimes part of the middle one-third PVWM area (slice SM). In some cases the lower motor cortex lesion is absent (slices W and SM). The deep subcortical PVWM lesion, however, is usually always present. The cortical portions of this lesion are compatible with lesion sites in longer-lasting Broca's aphasia cases previously published by Mohr *et al.* (1978).

Case 25

M.L., a 56-yr-old female, at 24 months following stroke onset produced nonfluent agrammatic speech compatible with Broca's aphasia (*see* Appendix). No hemiparesis was present. The CT scan in *fig. 16* shows a patchy lesion in the medial Sc F at slice B and a more extensive lesion in the medial Sc F at slice B/W. Only a minimal lesion was present in the middle one-third PVWM at slice SM-SM+1. The total lesion extent value for the two areas was only 4.75 (*see* *fig. 16*).

This case demonstrates that it is possible to have Broca's aphasia and not have a hemiplegia.

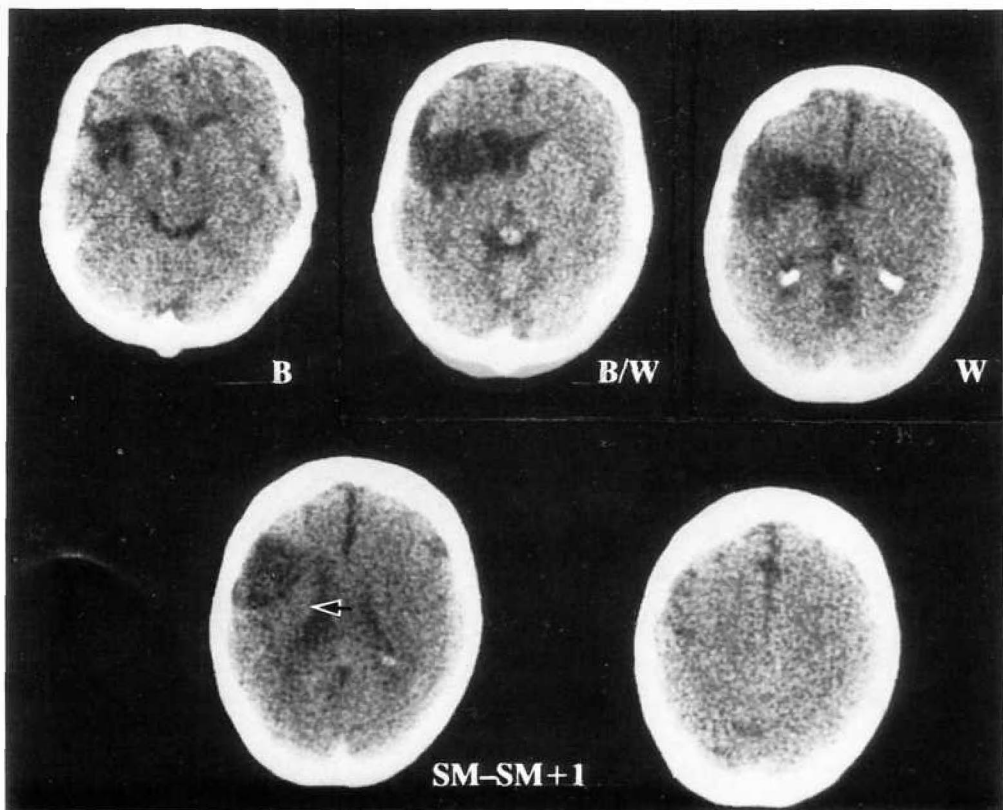


FIG. 16. CT scan at 16 months following stroke onset in *Case 25* (Group 4). No right hemiparesis was present. Lesion extent in the medial Sc F at slice B was rated only 2.5; slice B/W, 4; mean 3.25. Lesion extent in the middle one-third PVWM at slice SM-SM + 1 was rated only 1.5; total lesion extent 4.75. The arrow at slice SM-SM + 1 shows the minimal lesion in the middle one-third PVWM which greatly reduced the combined total lesion extent in this case to below 6, a value compatible with her mild limitation in speech. The absence of hemiparesis in this case was compatible with the absence of a lesion in the deepest middle-to-posterior one-third PVWM area adjacent to the body of the lateral ventricle at slice SM-SM + 1. This deepest PVWM area contains, in part, the descending pyramidal tract pathways (see also figs 4, 11, 14).

Again, the lesion did not extend completely across the middle-to-posterior one-third PVWM adjacent to the body of the lateral ventricle at slice SM-SM + 1 so that the deepest subcortical descending motor pathways were not completely interrupted (similar to *Case 16*, Group 2, fig. 11, and *Case 22*, Group 3, fig. 14). Each of these 3 cases without hemiplegia had three different types of language involvement. *Case 16* could produce only stereotypies; *Case 22* could produce nonfluent agrammatic speech but had a moderate comprehension deficit and *Case 25* could produce nonfluent agrammatic speech and had good comprehension. All 3 of these cases underscore the point that the presence or absence of hemiplegia is not a good prognostic sign for recovery of speech.

Case 26

B.J., a 67-yr-old male, at 17 months following stroke onset produced nonfluent agrammatic speech which was compatible with Broca's aphasia (see Appendix). A dense right hemiplegia was

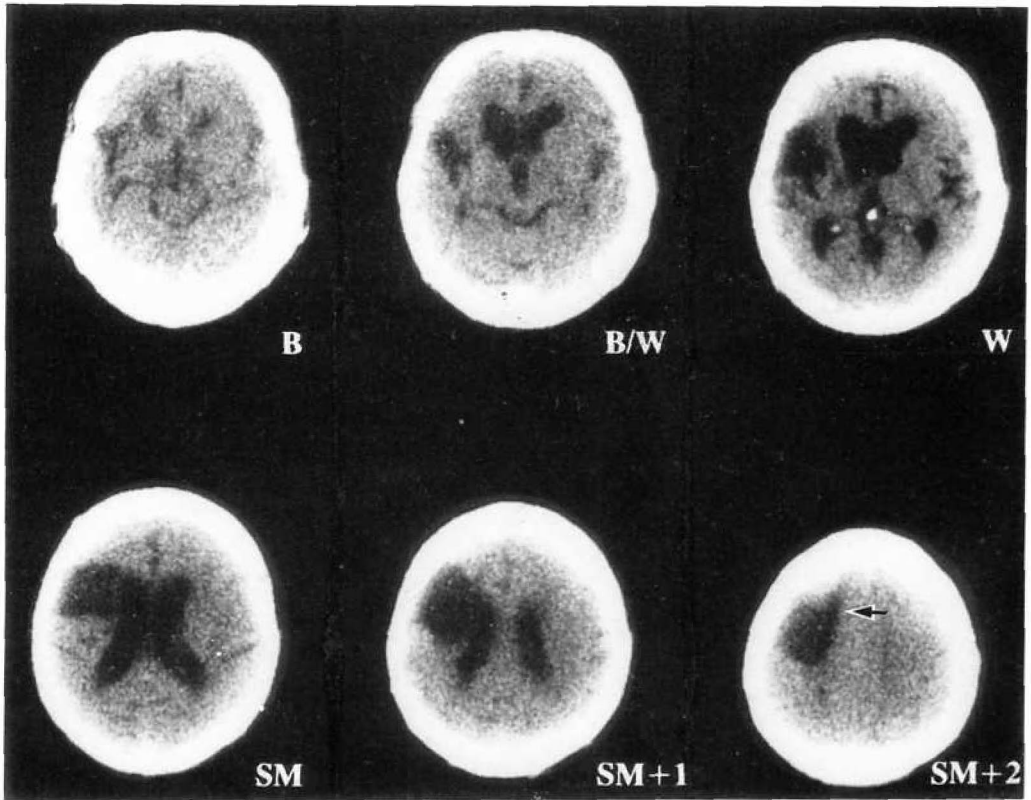


FIG. 17. CT scan at 1 yr following stroke onset in *Case 26* (Group 4). A dense right hemiplegia was present. Note deepest PVWM lesion at slice SM+1. No lesion was present in the medial Sc F at slices B or B/W, although a lesion was present deep to the supplementary motor area (SMA) and cingulate gyrus on slice SM+2 (arrow). Lesion extent in the middle one-third PVWM at slice SM was rated 4; total lesion extent, 4. The overall lesion site distribution for this case is unusual as it involves portions of both the distribution of the left middle cerebral artery and the anterior cerebral artery. This case exemplifies how lesion in the middle one-third PVWM can be combined with lesion outside the distribution of the left middle cerebral artery (anterior cerebral artery) to still produce nonfluent, agrammatic speech when there is no lesion in Broca's area and there is no lesion deep to it in the medial Sc F.

present. The CT scan in fig. 17 shows no lesion in the medial Sc F but lesion in greater than half of the middle one-third PVWM area. The total lesion extent was 4 (see fig. 17).

This was the only case in this study that had a lesion which included not only portions of the left middle cerebral artery distribution but also portions of that of the left anterior cerebral artery. This case had a lesion at slice SM+2 which undoubtedly interrupted projections from the SMA and cingulate gyrus to the caudate. Hence, although this Broca's aphasic had no lesion in the medial Sc F or Broca's cortical area, projections for speech initiation, preparation for speech movements and limbic aspects of speech were still interrupted, albeit from a different lesion source (deep to SMA and cingulate gyrus). It is probable that the same pathways were involved in this case, as other Broca's aphasics; however, in this case, the lesion was closer to the origin of the SMA/cingulate gyrus projections rather than being near their more distant projection sites in the medial Sc F deep to Broca's area near the caudate, as seen in most other Broca's aphasia cases.

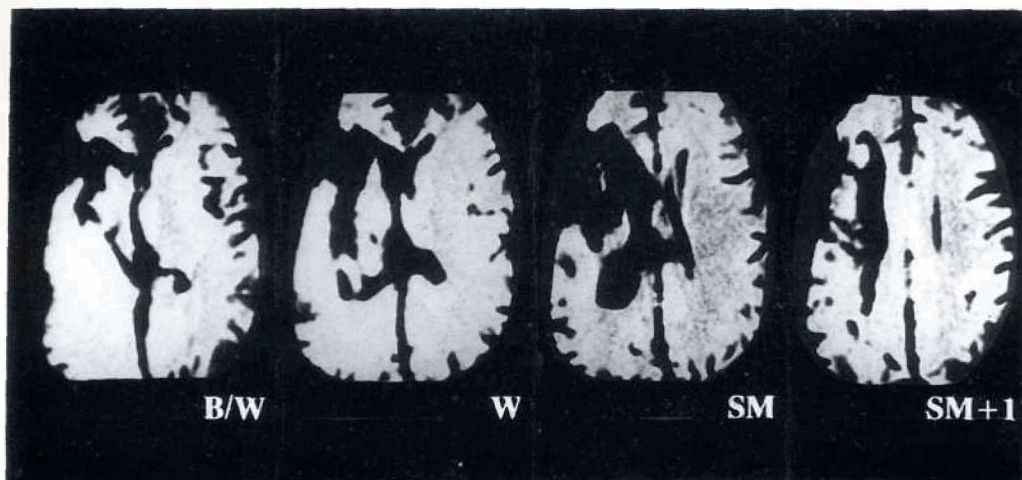


FIG. 18. CT scan of Broca's original case, Leborgne, from Castaigne *et al.* (1980), who at age 51, 21 yrs after stroke onset could produce only the stereotypy, 'tan, tan'. This case was similar to the Group 2 cases in the present study. A dense right hemiplegia was present. Lesion extent in the medial Sc F at slice B/W was rated 5; although slice B was not available, it was assumed that because the lesion was so extensive on slice B/W, it was equally extensive on slice B(5); thus, the mean lesion extent of the medial Sc F was 5. Lesion extent in the middle one-third PVWM at slice SM was rated 4; total lesion extent, 9. This total lesion extent value of 9 in these two deep subcortical white matter site areas was well within the range for cases with severe limitations in speech (total lesion extent values greater than 7). The dense hemiplegia in this case was compatible with a large PVWM lesion which extended to the deepest portion, immediately adjacent to the body of the lateral ventricle, especially at slice SM+1. (Reprinted with permission.)

Sample cases from previously published studies

Broca's original case, Leborgne

The results from the 27 cases examined in the present study indicate that when a deep subcortical lesion in the medial Sc F at the level of Broca's area is combined with a large lesion in the middle one-third PVWM deep to the lower motor/sensory cortex area for mouth, there is no spontaneous speech or only stereotypies. These results are supported further by examination of the CT scan of Leborgne's brain.

Leborgne was 30 yrs old at the time of his stroke and he died 21 yrs later. His spontaneous speech was limited to the stereotypy 'tan, tan'. His auditory comprehension was reported to be good. He had a dense right hemiplegia. Broca attributed his poor speech to a lesion in the cortical region of the foot of the third left frontal convolution (1861*a, b*). Broca himself never observed the depth of the lesion in Leborgne's brain because it was never cut, as was common practice at that time. Recently a CT scan was performed on the preserved brain at 140 yrs post-onset (Castaigne *et al.*, 1980; Signoret *et al.*, 1984; see fig. 18).

Fig. 18 shows slices B/W, W, SM and SM+1 (no slice B was available) of Leborgne's brain. Examination of the deep subcortical white matter surrounding the lateral angle of the left frontal horn reveals an extensive lesion in the medial Sc F at slice B/W. Because the lesion in the medial Sc F is so extensive at slice B/W, it is assumed there was a similarly extensive lesion in the medial Sc F at slice B. There is also an extensive lesion in the middle one-third PVWM at slice SM. The total lesion extent for the two areas was estimated to be 9. At slice SM+1 the PVWM lesion is contiguous with the lateral border of the body of the lateral ventricle and is compatible with the dense right hemiplegia reported by Broca. (There was no lesion in the PLIC.)

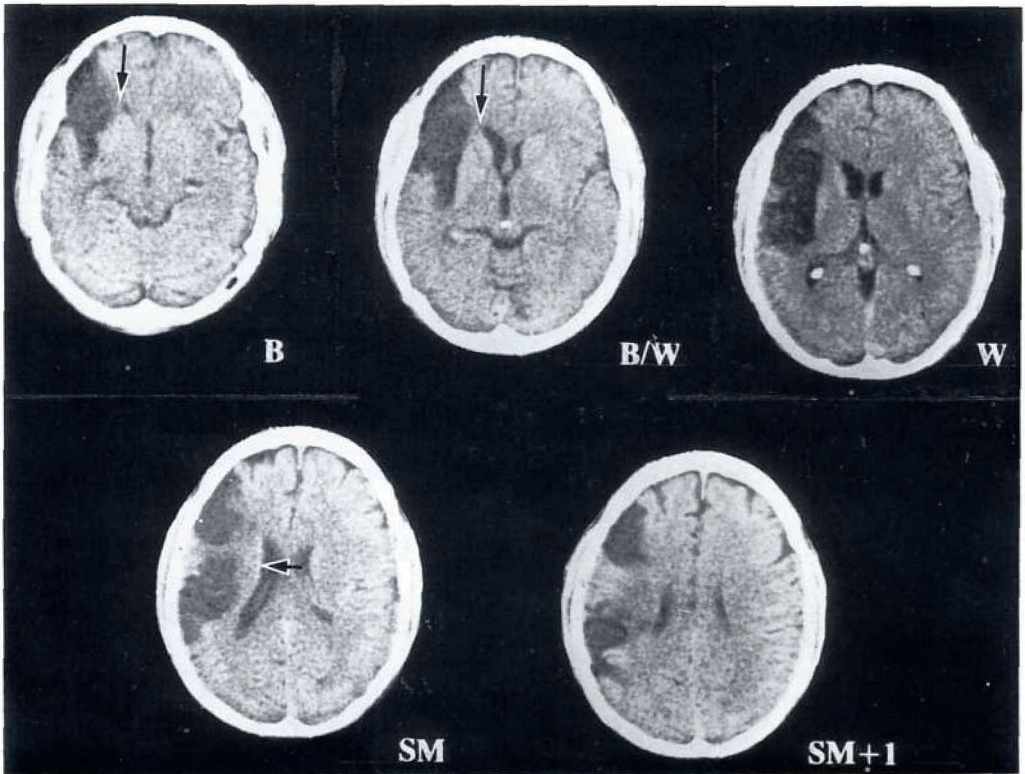


FIG. 19. CT scan at 5 months following stroke onset in *Case 73* from Knopman *et al.* (1983) (fig. 8), who at 6 months after stroke onset produced well articulated, grammatically correct speech. No right hemiparesis was present. Despite a large lesion in Broca's area, surface and deep (slices B and B/W) and a large lesion in the lower motor cortex area for the mouth, surface and deep (slice SM), there was no lesion in the medial Sc F at slices B or B/W (arrows) and there was almost no lesion in the middle one-third PVWM at slice SM (arrow). The absence of hemiparesis in this case was compatible with the absence of a lesion in the deepest PVWM area adjacent to the body of the lateral ventricle at slices SM and SM+1 (*see also* figs 4, 11, 14, 16). There also appeared to be almost no lesion in the lower sensory cortex area for the mouth. (Reprinted with permission.)

The occipital length asymmetry in Leborgne's CT scan appears to be the typical left occipital length. There appears to be no evidence for any form of anomalous dominance as measured by the CT scan hemisphere asymmetry.

1983 published case

The results from the present study are also supported by a recently published case with good speech recovery by Knopman *et al.* (1983). Their Case 73 supports the findings from the present study because of specific unusual sparing of the two white matter areas outlined here, despite a large extensive cortical/subcortical lesion lateral to these areas. Their case had good recovery of spontaneous speech by 6 months following stroke onset (*see* fig. 19).

The Knopman *et al.* (1983) Case 73 was published as fig. 8 in their paper. This case recovered to scores of 7 on the BDAE in phrase length, grammatical form, articulation and melodic line, by 6 months following stroke onset (D.S. Knopman, personal communication), despite a large lesion in Broca's area, superficial and deep, and a large lesion in the lower motor cortex area for the mouth,

superficial and deep. This fluent speech is compatible with the results of the present study because there was sparing of the medial Sc F at slices B and B/W, and most of the middle one-third PVWM at slice SM. The lower sensory cortex area for the mouth also was largely spared. There was no hemiplegia. The PVWM lesion was not adjacent to the body of the lateral ventricle at either slice SM or slice SM + 1.

This case demonstrates that sparing of the medial Sc F at slices B and B/W, and the middle one-third PVWM at slice SM, despite the presence of a large cortical lesion, may be a good prognostic sign for potential for recovery of speech. The sparing of the lower sensory cortex area for the mouth also may have been important for speech recovery.

Speech/language therapy

All the subjects who were inpatients at the Boston VA Medical Center (21/27 cases) received some form of speech/language therapy at the rate of 5 treatments per week while inpatients. Because of the nature of their communicative deficit, a nonvocal therapy programme that trains aphasic patients to produce representational gestures, Visual Action Therapy (VAT) was used with the most severe patients (Helm-Estabrooks *et al.*, 1982). The VAT programme was used with 5/7 cases in Group 1 (no speech); 9/10 cases in Group 2 (only stereotypies) and 3/5 cases in Group 3 (a few words or overlearned phrases). Following successful completion of the VAT programme some of these patients received a course of speech therapy.

One patient in Group 3 was treated with a method called Voluntary Control of Involuntary Utterances (Helm-Estabrooks and Barresi, 1980) which uses oral reading as a basis for gaining control over involuntarily expressed words and phrases. Three out of 5 patients in Group 4 received only Melodic Intonation Therapy (Sparks *et al.*, 1974), a programme for improving spontaneous speech through the use of melodically intoned, rhythmically tapped phrases and sentences. All BDAE test scores reported here are scores obtained following treatment and during the more chronic stage. The exact relationship between the various speech therapy programmes used and final spontaneous speech ability is unknown, and is beyond the scope of this study.

DISCUSSION

Other CT scan studies of severe limitation in spontaneous speech

CT scan studies of lesion sites producing severe limitation in spontaneous speech were undertaken recently by Brunner *et al.* (1982) and by Poeck *et al.* (1984). Brunner *et al.* concluded that automatisms and recurring utterances occurred only with combined cortical and basal ganglia lesions. No specific subcortical white matter pathway areas, such as the medial Sc F, were examined in that study. Composite lesion diagrams were shown only for one CT scan slice (slice W) and no complete CT scans of individual cases were shown. Although the information provided was limited, it appears that the conclusions from that study are not in agreement with our findings, which suggest that it is involvement of primarily *subcortical white matter* pathways (not basal ganglia and cortical lesions) which produce severe limitation in spontaneous speech.

Poeck *et al.* (1984) specifically examined patients with only stereotypies as compared with other global aphasics and found no differences in CT scan lesion sites between the two groups. The results of our study are in agreement with their study in that no differences were observed in lesion sites between the cases who produced only stereotypies versus those who had no speech. Poeck *et al.*,

however, also found no difference in lesion sites between cases with only stereotypies versus those with other forms of aphasia, including 'Broca's, Wernicke's, transcortical sensory, left middle cerebral artery occlusion and isolated basal ganglia lesions'. That investigation used a technique of superimposing lesions to search for common lesion sites; no specific subcortical pathways such as the medial Sc F were examined. They concluded that superimposing lesions is not an adequate approach and recommended that future anatomical studies attempt to examine disconnections involving critical cortical areas.

Two subcortical white matter pathway areas identified in the present study

The present study focused on specific cortical and subcortical areas and isolated two subcortical white matter pathway areas which, when damaged, resulted in severe limitation in spontaneous speech, that is, no speech or only stereotypies. These two areas, namely (1) the medial Sc F surrounding the lateral angle of the most rostral portion of the frontal horn (CT scan slices B and B/W) and (2) the white matter deep to the lower motor/sensory cortex area for mouth (CT scan slice SM), are discussed in more detail, below.

The Sc F, as mentioned above, was first described by Muratoff in 1893 in the dog brain. Rose (1935) cites four different labels for this area of white matter, comprising those of Muratoff, the fasciculus subcallosus, Anton, the fasciculus longitudinalis medialis, Wernicke, the fasciculus nuclei caudati, and Dejerine, the fasciculus fronto-occipitalis. In Dejerine's 1895 text, the most medial portion of the Sc F is labelled substance grise sous-ependymaire (Sge) and the more lateral portion, fasciculus occipito-frontalis (OF) (fig. 2). In the Yakovlev and Locke's (1961) study with the monkey brain, it is labelled stratum subcallosum (fig. 3).

Benjamin and Van Hoesen (1982), using horseradish peroxidase injections in monkey brains, have shown strong reciprocal connections between area 24 of the cingulate gyrus and the SMA. The importance of the SMA in 'the development of the intention-to-act' has been reviewed by Goldberg (1985). Barnes *et al.* (1980), using autoradiography in monkey brains, have shown that a major entry point for direct projections from the cingulate gyrus (and indirect projections from the SMA due to strong cingulate-SMA reciprocal connections) to the caudate is in the most medial white matter surrounding the lateral angle of the frontal horn in its most rostral portion. Jürgens (1984) has also observed direct connections from the SMA to the caudate. These mesial frontal cortex projections then spread to the ventral and lateral portion of the caudate and to the lateral portion of the putamen. Hence lesions located in the most medial white matter surrounding the lateral angle of the most rostral portion of the frontal horn (medial Sc F) would be interrupting pathways from the cingulate gyrus area and SMA leading into the caudate and putamen. This would have an effect on the initiation, preparation for speech movements, and limbic aspects of spontaneous speech.

The Sc F actually has projections from the cingulate and SMA to the caudate throughout the inferior portion of the corpus callosum. It appears, however, that

it is the most medial and rostral of these connections which are relevant in severely limiting speech when combined with a lesion deep to the lower motor/sensory cortex area for the mouth. In the present study, significant differences in lesion extent in the medial Sc F were only observed between the No Speech/Stereotypies group versus the Nonfluent Broca's group on the lower CT scan slices (B and B/W; CT scan slices at the level of Broca's area), not the higher CT scan slices, W, SM or SM + 1, although clearly the medial Sc F is also present in the lateral angle of the frontal horn and body of the lateral ventricle on these higher CT slices (*see* fig. 1).

Yakovlev and Locke (1961) further studied two parts of the Sc F: (1) the medial division containing corticostriate fibres from the cingulum and (2) the lateral division containing corticocortical fibres of the superior fronto-occipital bundle. It appears that lesions in the most medial portion of the Sc F (i.e., the portion interrupting the corticostriate projections) produce the most severe limitation in spontaneous speech only when combined with white matter lesions deep to the lower motor/sensory cortex area for the mouth (middle one-third PVWM at slice SM).

Additional support for the importance of the most medial white matter area surrounding the lateral angle of the left frontal horn in relationship to spontaneous speech comes from stimulation studies in this area performed by Van Buren (1963, 1966). During surgical procedures for basal ganglia disorders, it was observed that lower range electrical stimulation within or in the vicinity of the head of the caudate nucleus consisted predominantly of speech arrest; patients ceased counting. In 7/10 cases there was speech arrest from stimulation on the left, in 3/10 cases, from the right side. There also was arrest of voluntary movement.

Van Buren (1963) wrote '. . . what has been produced by stimulation in the vicinity of the head of the caudate nucleus is not indeed aphasia, but a more basic disturbance in which the impulse to speak or to continue another task has been dulled or forgotten'. He further described the response as that which resembles most closely the response obtained from the SMA.

It is still not known why the patients in this study who had extensive lesions in the most medial and rostral portions of the Sc F, plus lesions in the white matter deep to the lower motor/sensory cortex area for the mouth, had no speech or only stereotypies. We suggest, however, that each lesion in each subcortical white matter area produced important disconnections. The Sc F lesion interrupted projections from the cingulate and SMA to the striatum. Thus, this lesion appears to have interrupted initiation, preparation for speech movements, and limbic aspects of speech. The lesion in the subcortical white matter deep to the lower motor/sensory cortex area for the mouth may have interrupted the pathways necessary for motor execution as well as possibly those pathways necessary for sensory feedback. Hence we hypothesize that lesions in these deep subcortical areas effectively prevented any relevant speech because there were no available pathways for speech initiation, motor execution or sensory feedback. Undoubtedly,

other language association areas were also disconnected as a result of lesions in these two white matter areas.

When a large lesion was present in *both* the medial Sc F and the white matter deep to the lower motor/sensory cortex area for mouth, spontaneous speech pathways were essentially disconnected in the left hemisphere, and the patient either had no speech or only stereotypies (*see* figs 9, 10, 11, 12, 18.) In 1 case, this severe limitation persisted for at least 9 yrs following stroke onset (Case 11). In patients where nonfluent agrammatic speech was present, there was more sparing of either one of these two subcortical white matter pathway areas. Usually it was the extra sparing of the subcortical white matter deep to the lower motor/sensory cortex area for the mouth (middle one-third PVWM at slice SM) which was associated with improved speech ability resulting in nonfluent agrammatic speech (*see* figs 14, 15, 16). When both of these two subcortical areas were completely spared, despite a large cortical lesion lateral to them including Broca's area and the lower motor cortex area for mouth, there was normal phrase length, grammatical form, articulation and melodic line (*see* fig. 19.)

Importance of the combination effect of these two subcortical white matter areas

It is important to stress the *combination* effect of lesion in these two subcortical areas in relationship to severe limitation in spontaneous speech. Previous research in our laboratory and others has shown that small lesions in the frontal lobe alone, including subcortical lesions involving white matter lateral to the frontal horn, will produce only transcortical motor aphasia (Freedman *et al.*, 1984). The medial Sc F has not been specifically mentioned in earlier reports on transcortical motor aphasia, but interruption of pathways from the SMA has been implicated in the poor initiation of speech observed in this form of aphasia (Rubens, 1975).

Alternatively, it has been our observation that patients who have a lesion which is limited to the subcortical PVWM area at slice SM and/or slice SM + 1 may have only mild transcortical motor aphasia with or without dysarthria, and with or without hemiplegia, depending on the exact location of the PVWM lesion along the lateral border of the body of the lateral ventricle (Freedman *et al.*, 1984; Alexander *et al.*, 1987; Alexander and Naeser, 1987; Naeser, Alexander, Stiasny-Eder *et al.*, unpublished observations).

Lesion in these two white matter areas are contiguous, and additional lesions vary

Although the results of this study have focused on subcortical lesions in the medial Sc F and in the white matter deep to the lower motor/sensory cortex area for the mouth, it should be stressed that obviously the lesions in these two areas are contiguous and many other pathways are also undoubtedly disconnected along with these lesions. These other disconnected pathways may include portions of the fronto-occipital connections in the lateral bundle of the Sc F, frontopontine connections, portions of the arcuate fasciculus, and/or other periculate projections to the perisylvian language areas. The exact course of the lesion between the

most medial and most rostral portion of the Sc F on slices B and B/W to and including parts of the middle one-third PVWM deep to the lower motor/sensory cortex area for the mouth on slice SM, varied from case to case and not all of them included the ALIC, caudate or putamen, for example. All cases did, however, also include the anterior one-third PVWM at slice SM. Variations in lesion direction as well as individual variation in exact location of anatomical areas will, of course, contribute to variations in the language behaviour and perhaps also to potential for recovery. In addition, although the role of the right hemisphere for spontaneous speech is limited and not well understood at this time, this is another variable which must be considered and may contribute to individual variation.

Presence or absence of hemiplegia not associated with recovery of spontaneous speech

Recent studies have suggested that the absence of hemiplegia with acute global aphasia may be a favourable prognostic sign for good recovery from global aphasia (Legatt *et al.*, 1987; Tranel *et al.*, 1987). The results from the present study, however, suggest that absence of hemiplegia is not always a valuable prognostic sign in predicting recovery of spontaneous speech. One case in the present study had no hemiplegia, but also still had no speech almost 3 yrs post-stroke. This case and others examined in our laboratory have suggested that the presence or absence of hemiplegia may be independent of the type or severity of aphasia. Hemiplegia which occurs with lesions in middle cerebral artery territory without involvement of the PLIC appears to be directly related to whether the lesion is in the middle-to-posterior one-third PVWM and whether the lesion is immediately adjacent to the body of the lateral ventricle at slices SM and/or SM + 1.

Subcortical aphasia studies and refinement of subcortical anatomical lesion site labels

The overall mechanism underlying severe and lasting aphasia in cases with subcortical lesions is receiving increasing attention (Crosson, 1985). The importance of subcortical lesions in the striatum and surrounding white matter in producing atypical 'subcortical' aphasias has been stressed by Damasio *et al.* (1982), Naeser *et al.* (1982), and Alexander *et al.* (1987). Although the small, deep subcortical white matter pathway of the medial Sc F had not been isolated for specific examination per se in our previous studies on subcortical aphasia (Naeser *et al.*, 1982; Alexander *et al.*, 1987), it was included in those studies, in part, under the anatomical label 'extra anterior lesion extension' (EALE) beyond the frontal horn. A subcortical lesion in this area was always seen in the most severe cases with little or no speech, although at that time it was assumed that this lesion was interrupting connections across the corpus callosum to the right hemisphere as had been suggested by Bonhoeffer (1914).

The results from the present study suggest, however, that it is connections from the cingulate and SMA to the caudate which were actually interrupted and hence,

initiation and preparation of speech movements were also interrupted. In fact, EALE beyond the frontal horn was not an important lesion site which distinguished severe versus milder limitation in spontaneous speech in the present study, although it still may have interrupted projections across the corpus callosum to the right hemisphere in cases where this lesion was present. Results from the present study suggest that it is the most medial white matter which is immediately lateral to the frontal horn (not the white matter which is immediately anterior to the frontal horn) which is the most critical area to assess in evaluating the extent of lesions within the medial Sc F.

Problems with composite lesion site methodology

The methodology used in the present study was unique in that it did not rely on composite lesion sites. Instead, our approach focused on the potential effect of disconnection in specific anatomical pathways. Previous studies which had tried to examine composite CT scan lesion sites in association with severe limitation in spontaneous speech did not obtain meaningful results (Brunner *et al.*, 1982; Poeck *et al.*, 1984). Indeed, if we reexamine our own composite lesion site drawing for 'anterior' capsular/putaminal (C/P) subcortical aphasia cases (fig. 4, Naeser *et al.*, 1982), it appears these cases with grammatical but dysarthric speech had extensive lesions in *both* the medial Sc F *and* the middle one-third PVWM.

When each 'anterior' C/P subcortical aphasia case is examined separately, however, there is variable amount of lesion extension in the medial Sc F lesion and middle one-third PVWM lesion. For example, in a given case, if greater lesion extent is present in the first area, then lesser lesion extent is present in the second area, and vice versa. The individual case in fig. 2, bottom (1982) shows no lesion in medial Sc F at slice B, an extensive lesion in medial Sc F at slice B/W, and a partial lesion in the middle one-third PVWM at slice SM. This variability in lesion extent between the two areas can never be appreciated in a composite lesion site drawing for *all* the cases with the same language behaviour, but different extents of lesion in the two areas. The lesion extent in separate areas must be examined separately, for each individual case, even if the language behaviour is the same. Undoubtedly several different lesion site combinations can produce similar language behaviour.

Two subcortical white matter areas hypothesized to affect specific cortical areas

It is hypothesized from cerebral blood flow studies (Olsen *et al.*, 1986) and positron emission tomography studies (Kushner *et al.*, 1984; Metter *et al.*, 1985) that subcortical white matter lesions have a direct effect on specific cortical areas which have become indirectly involved due to lesions in the subcortical connections to those specific cortical areas. In patients with small subcortical lesions which do not interrupt critical pathways to or from specific cortical areas, there is often recovery, especially when good collateral flow is present (Olsen *et al.*, 1986). None of the subcortical cases in this study, however, had good recovery. Hence it is

assumed that the severe cases in this study who even had small subcortical lesions, but which were located in critical areas (most medial and rostral Sc F plus white matter deep to the motor/sensory area for the mouth) probably had a lesion in white matter pathways which were critical for cortical activation of speech. Thus although the emphasis in this study was on the examination of *subcortical* lesion sites, it was probably the *cortical* pathways which were disconnected by the subcortical lesions, which ultimately produced the severe limitation in spontaneous speech. Future research with PET or rCBF with patients with infarcts in the most medial and rostral portion of the Sc F and white matter deep to the motor/sensory cortex area for the mouth may help to clarify exactly which cortical areas have been disconnected.

Our hypothesis at this time is that at least the cingulate gyrus/SMA cortical area as well as the motor/sensory cortical area for the mouth are two important cortical areas involved in spontaneous speech. In addition, other perisylvian cortical language areas must also be considered such as Broca's or Wernicke's areas and/or the supramarginal gyrus area. The importance of both the cingulate gyrus/SMA as well as the lower motor/sensory cortex for the mouth in spontaneous speech is supported both by the rCBF studies of Larsen *et al.* (1978) and by the electrical stimulation studies of Penfield and Roberts (1959).

Additional neuroanatomical evidence which supports our findings comes from a recent study by Jürgens (1982). He injected horseradish peroxidase into the cortical larynx area within the lower sensorimotor face cortex in 3 squirrel monkeys. He observed retrogradely labelled cells in a continuous band extending all along the upper bank of the sylvian fissure from a homologous Broca's area rostrally, to the parietal association cortex (area 7) caudally. 'In addition, labelled cells were found in the ventrolateral prefrontal cortex, orbital cortex, *anterior cingulate gyrus, supplementary motor area, insula and inferior temporal gyrus*' (p. 377) (italics added by the present authors). In addition, labelled cells were observed in many subcortical nuclear groups. The results of the Jürgens' study thus are particularly relevant to the present study, because he stressed that 'The two medial cortical areas found . . . to contain labelled cells thus must be regarded as the limbic and SMA representation of the face' (p. 383). In his conclusions he stressed that these strong connections between the anterior cingulate gyrus/SMA and the cortical larynx area support previous observations that the anterior cingulate gyrus/SMA is extremely relevant to spontaneous speech disturbance in man (Penfield and Roberts, 1959; Larsen *et al.*, 1978). Hence this recent study by Jürgens (1982) is indirectly supportive of the importance of the two subcortical white matter areas isolated as important in spontaneous speech in the present study (medial Sc F and middle one-third PVWM deep to face area); and directly supportive of the two cortical areas implied to be important in spontaneous speech in the present study (cingulate gyrus/SMA and lower motor/sensory cortex area for mouth).

The present study focused on spontaneous speech and although all cases in

Groups 1 and 2 had severe limitation in spontaneous speech, not all of these cases had complete cessation of speech; 10/17 cases in Groups 1 and 2 could still repeat a few words and 4/17 could correctly name some pictures to visual confrontation. Research by Kirzinger and Jürgens (1982), as well as others (Smith *et al.*, 1981) has shown that lesions in the SMA have a direct effect on the initiation of 'spontaneous' motor behaviour patterns which are triggered internally and not on those triggered directly by external stimuli. Kirzinger and Jürgens observed, for example, that after the SMA was ablated in squirrel monkeys and these monkeys were placed in isolation, the number of vocal 'isolation calls' emitted from the monkeys was reduced, although the acoustic structure remained intact. Thus the absence of internally-generated speech (spontaneous speech) in the presence of some externally-generated speech (word repetition and naming) may be compatible, in part, with lesions directly affecting projections from the SMA. Further, variation in word repetition and naming ability observed across those subjects who otherwise had no meaningful spontaneous speech may have been due, in part, to variation in the extent of the lesion in the projections from the SMA, as well as from other areas. This would require further study.

Use of CT scanning to predict potential for recovery of spontaneous speech

Examination of the two subcortical white matter areas isolated in this study is suggested as a basic starting point for assessing potential for recovery of spontaneous speech in severely nonfluent stroke patients with infarction in the various branches of the left middle cerebral artery. When working with patients who have lesions outside the distribution of this artery, especially in the left anterior cerebral artery, different structures must be examined. For example, in cases with left anterior cerebral artery infarcts, it is possible that cortical lesions in the SMA and/or the cingulate gyrus may combine with subcortical lesions in the middle one-third PVWM to produce long-lasting impairment in speech, even when no lesion is present in the medial Sc F at slices B and B/W. Obviously, other cortical and/or subcortical lesion sites may also combine to produce equally severe limitation in spontaneous speech.

After completing this study with 27 cases, we recently reviewed over 50 other cases in our files and did find 1 case of exceptional recovery in spontaneous speech, despite an extensive lesion in the medial Sc F at slices B and B/W and in the middle one-third PVWM at slice SM (*see* fig. 20). The total lesion extent for these two structures in this case was 7.37, a value which should have placed him in Groups 1 or 2 (values above 7). The patient was a 45-yr-old, right-handed male who was a highly successful lawyer at the time of his stroke. At 1 yr post-stroke, he was able to use 7-word phrases, although they were spoken very slowly and with poor articulation. For example, in describing the cookie theft picture, he said, in part, '. . . she is going du get wet feet because . . . hu . . . uh . . . is going to get . . . the shoe is going to get wet'. He has continued to recover in spontaneous speech until the present (over 5 yrs post-stroke), although the slow rate

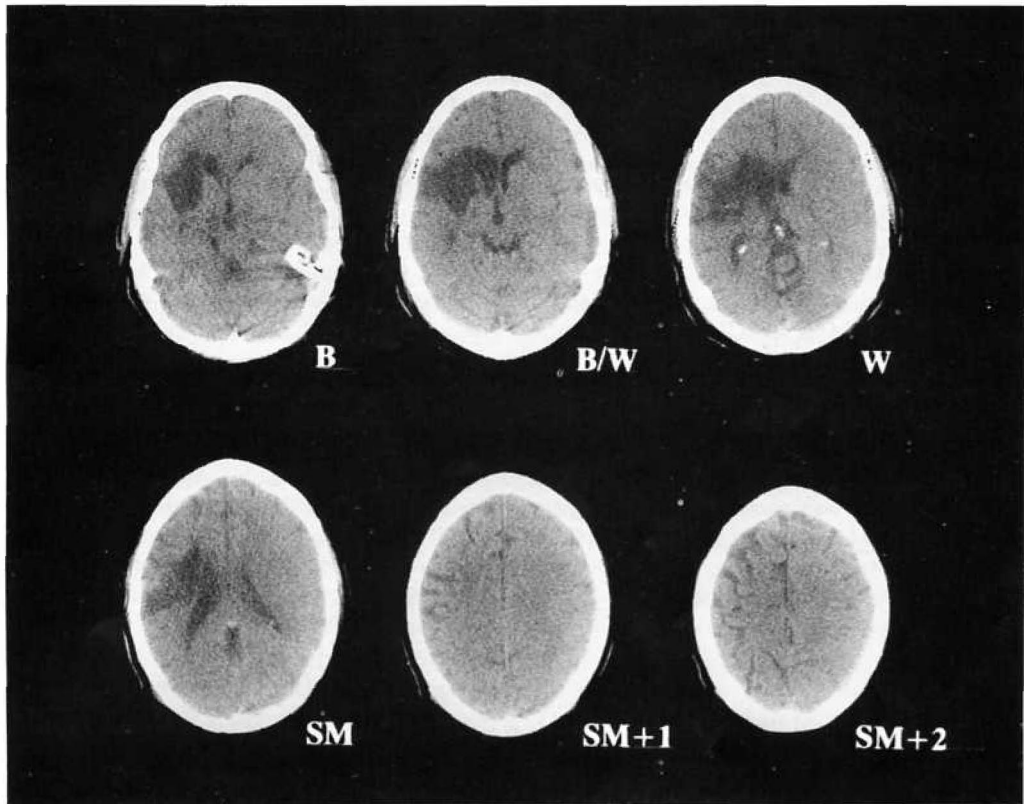


FIG. 20. CT scan at 1 yr following stroke onset in a case who had unexpected recovery of spontaneous speech at 1 yr post-stroke (see text) despite an extensive lesion in the medial Sc F (slice B, 3.75; slice B/W, 5) and the middle one-third PVWM at slice SM (4). The total lesion was rated 7.37. A dense right hemiplegia was present.

articulation have persisted. A dense right hemiplegia is still present. This case exemplifies the fact that exceptions will always be found for patterns of language recovery that are unexpected with specific CT scan lesion sites. In this case, although the combined lesion extents placed him in the range for cases with the most severe limitation in spontaneous speech, his speech exceeded that for even the Broca's aphasics in Group 4. His exceptional recovery may have been related to a relatively young age at onset, a higher educational level and a higher premorbid IQ.

Lastly, examination of the two subcortical white matter areas stressed in the present study may contribute to a better understanding of the mechanisms involved in normal spontaneous speech. In the rCBF studies by Larsen *et al.* (1978), significant increases in blood flow were observed, particularly in the left hemisphere, in the SMA and the motor and sensory cortex area for the mouth during automatic speech. Results from the present study have suggested some specific subcortical

white matter pathways, which although not visible in rCBF studies which focus on cortical cerebral blood flow, appear to be critically important in the final coordinated cortical activation for speech.

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APPENDIX

<i>Case</i>	<i>Time post-onset</i>	<i>Spontaneous Speech Samples for description of the Cookie Theft Picture</i>
Group 1		
1	27 mos	'Yeah . . . yeah'
2	8 yrs	No speech.
3	9 mos	No speech.
4	47 mos	'Juh . . . ah . . . jou . . . juhjuh . . . uhpai . . . uhnouer'
5	2.5 yrs	No speech.
6	15 mos	'No . . . ' (and 'grunts')
7	9 mos	No speech.
Group 2		
8	6 yrs	'Boom . . . boom'
9	18 mos	'Ai . . . da tu . . . dididi'
10	13 mos	'Senny fenny'
11	9 yrs	'I don't know . . . good good . . . yes, yes . . . tu, tu . . . no, no'
12	4 yrs	'Wa, wa . . . for Christ sake'
13	15 mos	'Guhdi, guhdi . . . wazuh waz'
14	2 mos	'Bee bee . . . bye bye'
	(2 yrs)	At this time there was almost no speech.
15	6 mos	'Yes, yes'
16	33 mos	'Morning, morning . . . boy, boy'
17	13 mos	'1, 2, 3, 4, 5 . . . boom, boom'
Group 3		
18	35 mos	Unintelligible vowel sounds. 'Siuhl . . . yeah down . . . un . . . cookies wash um um wash eeah no water here, fuhee, no good over here.'
19	7 mos	'Goddam . . . Chrissakes . . . I forgot it . . . well goddam'
20	15 mos	'There, too . . . there, too . . . um . . . I don't know . . . that's all I guess gee whiz. I don't know, that's all . . . well . . . that, too and there and there'.
21	52 mos	'Well . . . uh . . . Duh um . . . glasses . . . run'
22*	8 mos	'Nothing. The kid break'in an . . . on an that one. He gonna get get'in, gah. Its running. He given one to give one. She's dissing.'
*At this time the patient's speech output was almost compatible with nonfluent Broca's aphasia, however her comprehension was still too impaired for her to be considered a Broca's aphasic.		
Group 4		
23	7 mos	'The wady is doing her dishes. Sink undis over uh . . . The window is open and the "w" won . . . a very funny day outside . . . ook children . . . a boy and a girl'.
24	7 mos	'A kids . . . a cookies . . . and uh, uh, fall down, . . . wash'in de dishes . . . uh runn'in water . . . fish fash . . . uh foor . . . he was . . . girl a cookie'.
25	24 mos	'The girl . . . uh, sh-sh sheez, the boy fall down . . . the ch-ch chair . . . the boy . . . is . . . cookies . . . the boy, the lady . . . is . . . raiping the dishes'.

APPENDIX

<i>Case</i>	<i>Time post-onset</i>	<i>Spontaneous Speech Samples for description of the Cookie Theft Picture</i>
Group 4 (<i>cont.</i>)		
26	17 mos	'Well, wiss . . . watcheez, water, . . . uh, this kaitee jar . . . uh . . . do . . . eee . . . dee . . . deezeez . . . uh, ahniz . . . ahniz, uh, whoops . . . bay . . . birl . . . no . . . girl . . . boy . . . girl, I /ton/ know'.
27	6 yrs	'Dis iz . . . bee out a lawn built up . . . This kid . . . fall down . . . This kid waking up here'.