

Three Variant Forms of Subcortical Aphasia in Chinese Stroke Patients

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Five right-handed patients with subcortical aphasia that involved the left hemisphere subcortical lesion sites were subjected to CT scans. Given their etiology, two cases were infarctions and the other three were hemorrhages. Two of the patients presented an involvement of the anterior limb of the internal capsule and of the basal ganglia and an anterior superior white-matter lesion extension. In both cases slow scanty dysarthric speech was noted; one had markedly impaired auditory comprehension, and the others were only partially impaired. The third patient presented an involvement of the posterior limb of the internal capsule and of the thalamus and a posterior paraventricular white-matter lesion extension. He had poor auditory comprehension, echolalia, and fluent speech. The last two patients presented an involvement of the internal capsule, the basal ganglia, and

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the thalamus and an anterior posterior paraventricular white-matter lesion extension. The latter two showed poor auditory comprehension with nonfluent and scanty spontaneous speech. The speech sounds were nonsensical monosyllabic words with a pattern similar to that of global aphasia. All patients had lasting right hemiplegia. © 1989 Academic Press, Inc.

INTRODUCTION

In recent years, the relationship between aphasia syndrome and lesion site has become clearer with the development of the CT scan (Naeser & Hayward, 1978; Wallesch, 1985). The subcortical aphasia or atypical aphasia syndrome (Naeser et al., 1982; Damasio, 1981; Damasio & Rizzo, 1982) has been reported previously in association with a pathological foci in the left hemisphere of the basal ganglia, internal capsule, thalamus, and paraventricular white matter (PVWM). This syndrome differs from the classical types of aphasia (Kertesz, 1983), such as Broca's or Wernicke's aphasia.

In 1972, Goodglass and Kaplan described the aphasia test battery BDAE (Boston Diagnostic Aphasia Examination) which is now in common use. Recently, the BDAE has been translated into Chinese with slight modifications, and thus can be used as an evaluation tool in the Republic of China. In our research, the CT scan was used to define the anatomical localization and size of lesions (Naeser, Hayward, & Laughlin, 1981) in the deeper and smaller areas of the brain. This gave us a clearer etiology of the infarction or hemorrhage. The BDAE was used as a speech evaluation model to detect speech deficits and give a more precise and uniform description of the problems.

This paper reports the study of five patients who had three different forms of aphasia syndrome and lesions that extended into the internal capsule, basal ganglia, thalamus, and the PVWM area.

SUBJECTS AND METHODS

Five patients with motor disabilities were tested for aphasia syndrome at the Rehabilitation Department of Mackay Memorial Hospital in Taipei, Taiwan, R.O.C. The basic data are presented in Table 1 and include age at onset, sex, neurological deficit, handedness, and etiology as evident in the CT scans.

These cases were selected on the basis of CTs that demonstrated similar lesion localizations in the subcortical area: namely, the basal ganglia, internal capsule, thalamus, and a PVWM extension in the left hemisphere. They were then divided into three groups according to their similar anatomical localization as revealed by the CT scan findings.

The CT scans (Schnitzlein & Murtagh, 1985) were performed with the canthomeatal line (CML) as the baseline and with 10-mm-slice thicknesses at 10-mm intervals which moved from the baseline toward the vertex. Slice 1 served as the baseline, with subsequent slices designated consecutively. To diagnose the brain lesion objectively and anatomically, the areas of major involvement were read and calculated by the radiologists using a double blind method. A digital planimeter was used to measure the lesion. Three measurements were averaged to produce a mean value, which was multiplied by the magnification factor of 8.40 to obtain the actual area of the brain lesion in every slice. The size of the lesion in the left hemisphere according to each slice is given in Table 2.

TABLE 1
MAJOR CLINICAL NEUROLOGIC FINDINGS FOR SIX SUBJECTS

Case/age at onset/sex	Etiology	Time of CT examination	Deficit		Consciousness at onset	Handed
			Motor	Sensory		
1/55/M	Hemorrhage	6 days after stroke	R hemiplegia arm > leg	OK	Clear	Right
2/61/F	Hemorrhage	Immediately	R hemiparesis leg > arm	OK	Clear	Right
3/56/M	Hemorrhage	Immediately	R hemiplegia	OK	Clear	Right
4/71/M	Infarction	6 days after stroke	R hemiplegia arm > leg	Response to pain	Clear	Right
5/51/F	Infarction	8 days after stroke	R hemiplegia leg > arm	Response to pain	Drowsy	Right

TABLE 2
 LESION SIZE AT EACH CT SLICE OF APHASIA PATIENTS

	Case 1	Case 2	Case 3	Case 4	Case 5
			Area (cm ²)		
Slice B (S4)	—	—	2.13	—	2.13
Slice B/W (S5)	6.39	7.10	3.55	4.26	7.10
Slice W (S6)	6.39	9.23	4.26	4.97	15.62
Slice SM (S7)	7.10	2.84	2.84	5.68	16.33
Slice SM + 1 (S8)	11.36	—	—	3.91	4.61
Slice SM + 2 (S9)	6.39	—	—	—	—

Note. B, Broca's; W, Wernicke's; SM, supramarginal gyrus; SM + 1, 1-cm slice above the level of SM; SM + 2, 2-cm slice above the level of SM.

The landmarks used were as follows:

Slice 4 (S4) is 40 mm above the CML and is similar to slice B. (Note that Broca's area can be seen lateral to the left frontal horn.)

Slice 5 (S5) is 50 mm above the CML and is similar to slice B/W. (Note that the anatomical area at the third ventricle quadrigeminal cistern can be seen.)

Slice 6 is 60 mm above the CML cutting and is similar to slice W. (Note the butterfly-shape horns and strium with calcified choroid plexus. Also the internal capsule-putamen and thalamus can be seen in both S5 and S6.)

Slice 7 is at the 70-mm cutting and is similar to slice SM (the body of the lateral ventricle can be seen), and slice 8 is similar to slice SM + 1 (paraventricular white-matter can be seen most clearly in slice SM + 1). Slice 9 is similar to slice SM + 2.

After the patients' general condition stabilized, language performance was formally evaluated with the translated and slightly modified BDAE test which is commonly used in general hospitals. The results of the testing and patient's education level are given in Table 3. According to the patients' performances on the BDAE test, their speech characteristics were rated on a 7-point scale (Figs. 1b, 2b, and 3b). For the last item, there is a mean of the percentiles of the four auditory comprehension subtests. Four of the five patients took and completed the second examination during the follow-up study. Due to personal reasons case 3 was unable to come to the hospital for a follow-up examination, but was tested over the telephone.

Report of Cases

Case 1. This 55-year-old right-handed male patient developed aphasia, right hemiplegia, and facial palsy of the central type in August 1985 (Table 1). His speech was nonfluent, difficult, and agrammatical with phonemic paraphasia. However, he had fair articulation. He could repeat four-character phrases, but his auditory comprehension was markedly impaired (Table 3). One week after the initial evaluation, his auditory comprehension was much improved. The CT scan (Fig. 1) obtained immediately after the stroke showed a hemorrhage involving the left basal ganglia, a compression to the anterior limb of the internal capsule, and an extension to the anterior-superior paraventricular white-matter extension.

The patient was therapeutically treated in the Rehabilitation Department with physical, occupational, and speech therapy, and his auditory comprehension quickly improved. Six months later, he could walk with a little support. However, the right hand still could not function, but he could elevate it. He had almost completely recovered his auditory comprehension. He repeated well at word-level, and attained 8/8 in the repetition of high-

TABLE 3
 BOSTON DIAGNOSTIC APHASIA EXAMINATION (BDAAE) TEST SCORES FOR SIX SUBJECTS

No. of case	1	2	3	4	5
Testing, months after stroke					
Education (1st/2nd)	1/6	1/2	1	1/3	1/3
Spontaneous speech	Primary school Nonfluent	High school Grammatical	Primary school Fluent with mild dysarthria	Primary school No speech or only stereotyped monosyllable	Primary school
No. of words per phrase (7)	2/7	1/6	5	0/0	0/0
Articulatory agility (7)	7/7	2/4	5	0/0	0/0
Grammatical form (7)	0/7	0/6	6	0/0	0/0
Timed verbal oral agility (2 × 7 = 14)	0/7	0/6	6	0/0	0/0
Timed nonverbal agility (2 × 6 = 12)	0/9	0/5	0	0/0	0/3
Comprehension (2 × 36 = 72)	4/59	25/62	2	0/1	0/1
Body part (20)	0/18	19/18	5	0/0	0/3
Commands (15)	1/10	8/13	3	0/0	0/2
Repetition words (10)	6/10	0/10	8	0/0	0/0
High-probability phrase (8)	0/8	0/8	3	0/1	0/0
Visual confrontation naming (3 × 38 = 114)	0/96	0/96	0	0/0	0/0

probability phrases task. The patient scored 7/14 in the timed verbal oral agility task and 9/12 in the timed nonverbal oral agility task. Except for slight difficulty in color-naming, he performed very well in the visual confrontation naming task. His spontaneous speech was grammatical but slow.

Case 2. This 61-year-old right-handed female patient developed aphasia, right hemiparesis, and mild right facial palsy of the central type in November 1985. Tables 1 and 3 summarize the neurological findings and the results of the aphasia testing. In the initial assessment, her comprehension was partially impaired. Her singing and automatic speech ability (e.g., days of the week, numbers from 1 to 21, and the 12 animal codes corresponding to the lunar years) were preserved, but she had severe dysarthria. She could speak a few words, but she was unable to calculate. The CT scan (Fig. 1) obtained immediately after stroke showed a hemorrhage in the left basal ganglia, in the anterior limb of the internal capsule, and in the anterior superior part of PVWM.

The patient was treated in our Rehabilitation Department with physical, occupational, and speech therapy. Two months later, she could walk without support. The right hand still could not function, but she could elevate it in a synergic pattern. Her comprehension returned to near normal levels, and her phrase length increased. Her repetition and visual confrontation naming were also good, and her spontaneous speech was grammatical. The articulation improved, but the pitch was low and inflexible. Her calculating ability also improved.

Case 3. Tables 1 and 3 summarize the neurologic findings and aphasic testing for this 56-year-old right-handed male patient. The patient's speech was fluent and grammatical with mild dysarthria. Spontaneous speech and visual confrontation naming showed paraphasia, such as phonemic paraphasia (e.g., "ng" was omitted in "Hee-yang" and "Hee-yang" became "Hee-Ya"), semantic paraphasia (e.g., "grape" was substituted by "papaya"), and preservative paraphasia (e.g., he added "monkey, dog, pig" at the end of every recitation test). In the Chinese calendar, "monkey, dog, and pig" are the last 3 of 12 animals that correspond to the lunar years, and were spoken correctly during the recitation test. His auditory comprehension was markedly impaired, but he could do well on the word levels. The repetition of high-probability phrase score was 3/8. The patient's speech output was also echolalic when the therapist asked questions in the responsive naming section of the test. The CT scan (Fig. 2) was given immediately after the stroke, and indicated a hemorrhage involving the thalamus, the posterior limb of the internal capsule, and the paraventricular white-matter.

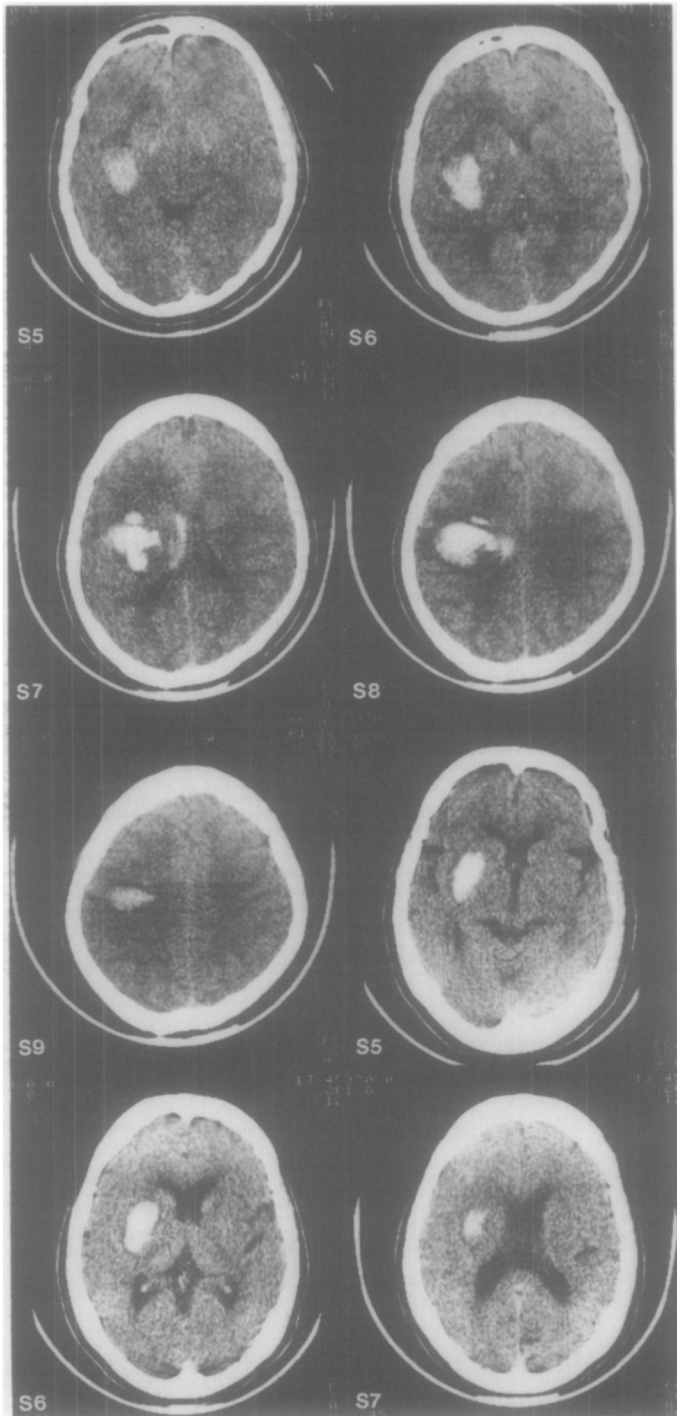
Two to three months later, marked auditory comprehension recovery was noted. The motor function was also much improved. The patient now could ambulate without support and could use utensils with his right hand on a functional level, but was still weak. Four months later, his speech recovered to nearly a normal level. He could describe things grammatically on the phone without paraphasia. He could also calculate plus and minus.

Case 4. This 71-year-old right-handed male patient developed global aphasia and right hemiplegia in December 1985. At that time, he could not speak. The first speech assessment was given 2 weeks after onset.

The CT scan (Fig. 3) that was obtained 6 days after the patient's stroke showed an infarction involving the left caudate nuclei, the anterior limb of the internal capsule, and the putamen nuclei. The CT scan also showed an infarction in the region of the anterior posterior paraventricular white-matter.

The patient was reevaluated in the third month after the stroke, but his comprehension had not improved. He uttered many stereotyped monosyllables (e.g., "wa," "going," "won"), and possibly the result of his previous habit of swearing when he felt uncomfortable.

FIG. 1. Computed tomographic scans of case 1 (S5-S9, top) and case 2 (S5-S7, bottom). Hemorrhagic lesion over capsular-putaminal area with anterior-superior lesion extension.



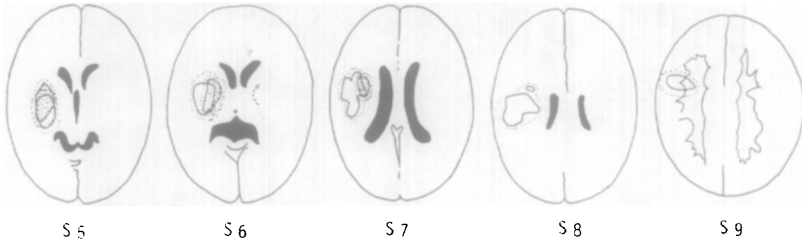


Fig. 1a. Composite CT scan lesion sites for cases 1 and 2, with basal ganglia, anterior limb of internal capsule, and anterior or superior extension of PVWM from S5 to S9. No cortical involvement was found. Dotted lines indicate the brain edema area.

In the second assessment, he could still only repeat two words and a one high-probability phrase. There was slight motor recovery, but he still could not walk. Although his auditory comprehension did not improve, his family felt that he exhibited understanding by facial expressions, gestures, and environmental cues. In other words, he could use nonverbal communication.

Case 5. This 51-year-old right-handed female patient had a minor stroke due to a very small left basal ganglia infarction in September 1982. Without sequelae, she could work normally. In December 1985, she again suffered a stroke. At this time, the auditory comprehension was completely impaired, and she became mute. The CT scan (Fig. 3) which was obtained 8 days after the stroke showed a cerebral infarction involving the left

RATING SCALE PROFILE OF SPEECH CHARACTERISTICS

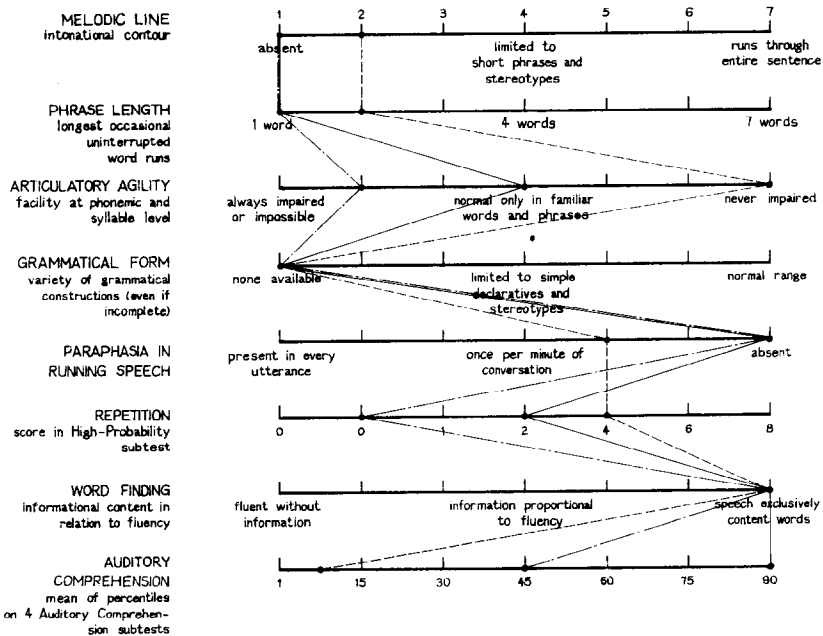


Fig. 1b. BDAE rating scale of speech characteristics. Typical Broca's aphasia characteristics are indicated by a solid line; characteristics of case 1 (-----) and case 2 (.....) are shown with broken lines.

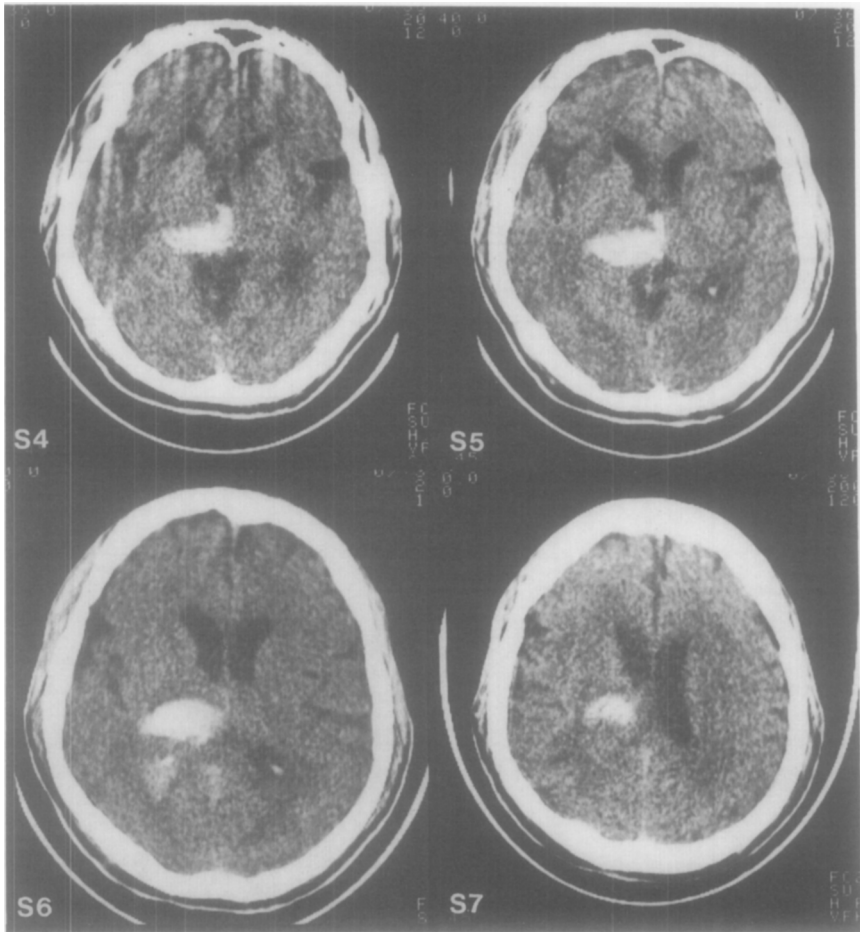


FIG. 2. Computed tomographic scans of case 3 (S4–S7). Hemorrhagic lesion of case 3 over capsular-putaminal area with marked edema to posterior area. Note the compression of the left posterior lateral ventricle.

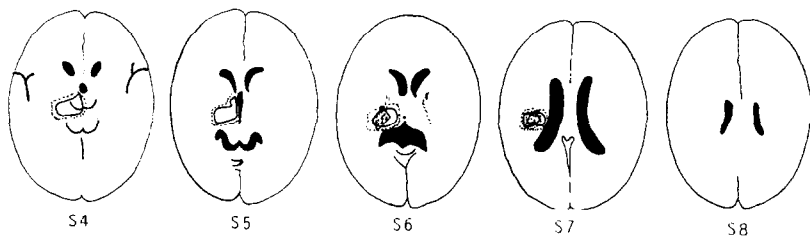


FIG. 2a. Composite CT scan sites for case 3, with left posterior limb internal capsule thalamus and posterior extension of PVWM from S4 to S7.

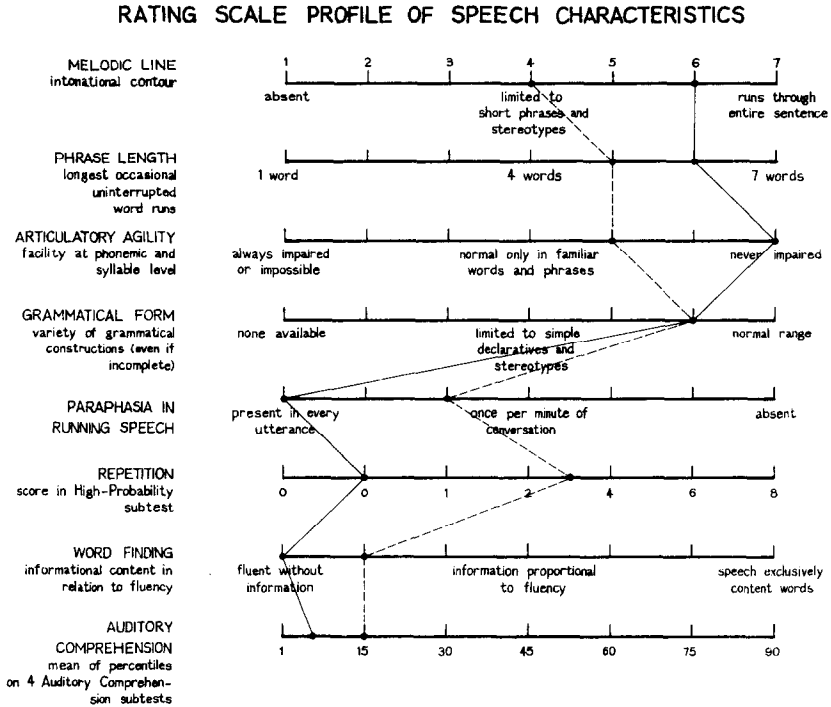


Fig. 2b. BDAE rating scale of speech characteristics. Typical Wernicke's aphasia characteristics are indicated by a solid line; characteristics of case 3 (-----).

putamen, the anterior and posterior limbs of the internal capsule, the thalamus (small lesion), and the anterior posterior paraventricular white-matter.

The follow-up assessment was made the third month after the stroke when she could walk with a little support. At this time, the right upper extremity still could not function, but her auditory comprehension had improved slightly. She could now recognize some body parts (e.g., ear, nose, neck, palm, shoulder, and knee) and obey some simple commands (e.g., "Make a fist," "Keep your eyes closed"). In spontaneous speech, only meaningless, stereotyped monosyllables were produced, although she could communicate with her family nonverbally.

RESULTS

In cases 1 and 2, the CT scans showed a lesion of the anterior internal capsule and the basal ganglia and an anterior superior extension of the PVWM (Fig. 1a). Some surrounding hypodense area was noted, and it was very marked in case 1. The aphasia pattern as shown in Fig. 1b had the following characteristics: the auditory comprehension was partially affected; there was nonfluent speech; there were differing degrees of repetition; the oral ability was affected; and both cases 1 and 2 had lasting hemiplegia. The prognosis was good, and both cases experienced

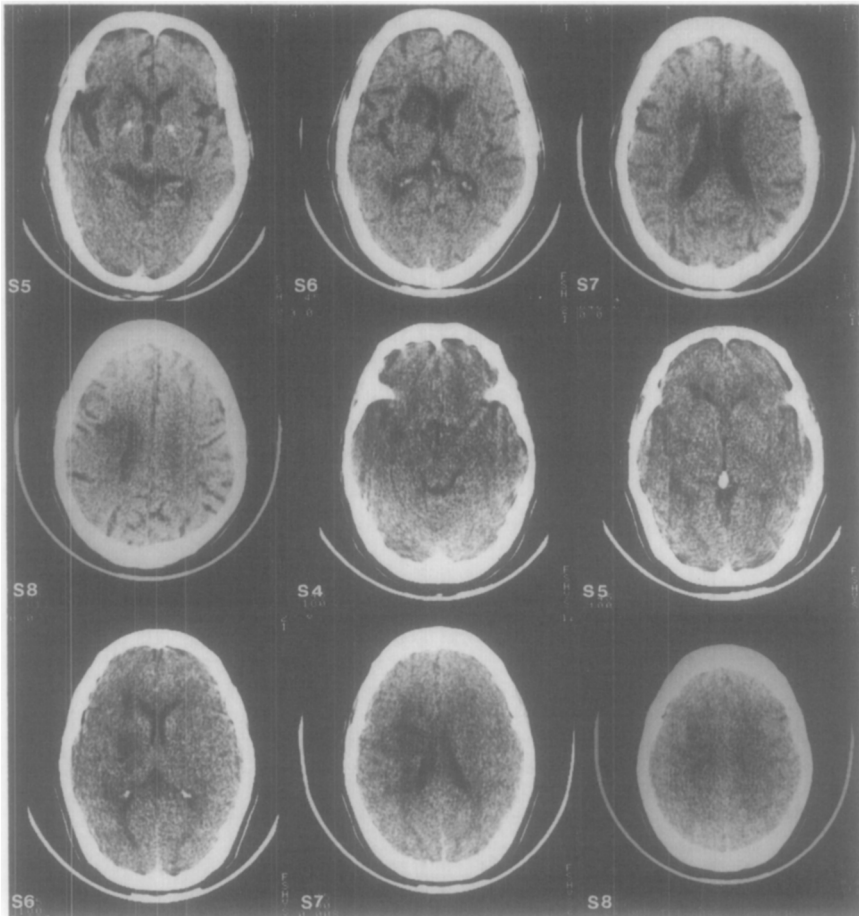


FIG. 3. Computed tomographic scans of case 4 (S5-S8, top) and case 5 (S4-S8 bottom). Marked infarcted lesion over capsular-putaminal area with both anterior superior and posterior extension.

good recovery of auditory comprehension and grammatically correct speech. However, dysarthria was found.

In case 3, the CT scan showed that the lesion had an involvement of the posterior internal capsule, the thalamus, and a posterior PVWM extension (Fig. 2a). The aphasia pattern shown in Fig. 2b had the following characteristics: auditory comprehension was severely affected; there was fluent speech with phonemic, semantic, and preservation paraphasia; good grammatical form was present; there was good articulation; the patient had lasting hemiplegia. The follow-up study showed marked speech and motor recovery.



Fig. 3a. Composite CT scan lesion sites for cases 4 and 5, with caudate nucleus. Anterior limb, posterior limb internal capsule, putamen and anterior or superior extension of PVWM from S4 to S8.

In cases 4 and 5, the CT scans showed a lesion with an internal capsule and the basal ganglia lesion had anterior posterior PVWM involvement (Fig. 3a). The aphasia shown in Fig. 3b had the characteristics of global aphasia, poor auditory comprehension, and nonfluent speech with some monosyllabic sounds. The follow-up poststroke study at 3 months revealed

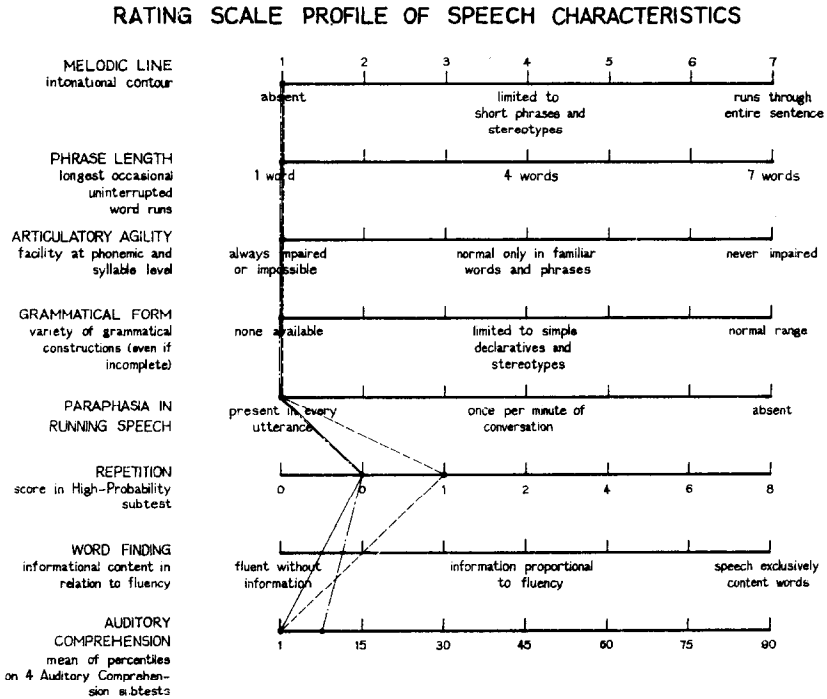


Fig. 3b. BDAE rating scale of speech characteristics. Typical global aphasia characteristics are indicated by a solid line; characteristics of case 4 (-----) and case 5 (.....) are shown with broken lines.

similar findings with no speech recovery, but the patients did exhibit slight motor recovery of the lower extremities.

DISCUSSION

Traditional forms of classifying aphasia cases, such as the system that breaks aphasia into six subgroups (Wernicke's Broca's, transcortical motor, transcortical sensory, conduction, and global) (Damasio, 1981; Naeser & Hayward, 1978), have been subject to new findings that are now being made available with the CT scan. Moreover, variant types of aphasia induced by the deep-seated brain lesion were also noted with the CT scan (Damasio, Damasio, Rizzo, Varney, & Gersh, 1982; Naeser et al., 1982). When we reviewed these findings in subcortical aphasia, several observations of our patients caught our interest and were seen to be noteworthy.

Upon reviewing and comparing the CT scans, we found that the hemorrhagic area in the patients demonstrated markedly different patterns. In case 1, the hemorrhagic area had a very distinct and marked area of hypodensity that surrounded it, especially when it was compared to cases 2 and 3. In case 2 and 3, the hemorrhagic area was about the same as that of case 1, but the hypodense area was not evident and this may have been due to the fact that the area had not yet fully developed since the examination was made on onset of the disease. In case 4 and 5, the hypodense area was more pronounced than in cases 2 and 3. Specifically, the hypodense area was evident, but the interface was not definitely delineated.

In retrospect and given the clinical course of the disease, the authors felt that the large surrounding hypodense area that was demonstrated in case 1 as well as the less marked hypodense area in cases 4 and 5 seemed to be the result of brain edema. This conclusion was drawn from several observations. First and foremost, the clinical course of the disease in the first patient indicated that it was brain edema versus hemorrhagic infarction. In hemorrhagic infarction cases, the patient often regresses after the onset which is often due to the onset of a secondary hemorrhage. But in this situation, the patient steadily improved. Second, the dark surrounding hypodense area in case 1 did not exhibit any of the "salt and pepper" characteristics that indicate a hemorrhagic infarction. Third, the CT scans were taken at different times and this seemed to play a major role in the differences between cases 2 and 3 and the other cases. Specifically, the CT scans were taken immediately on onset in cases 2 and 3, but were taken on the sixth, seventh, and eighth days respectively in cases 1, 4, and 5. The earlier pictures in cases 2 and 3 could easily account for the lack of brain edema. Thus, it was deduced that the

surrounding hypodense area in cases 1, 4, and 5 could be attributed to brain edema.

In the five cases, all of the patients experienced hemiparesis or hemiplegia. This may be due to the fact that the pathways for motor function of limbs lie close to the motor or sensory speech pathways. Thus they can be affected both jointly as well as individually. (Tanridage & Kirshner, 1985).

In 1981, Damasio found that some patients exhibited symptoms of Broca's aphasia even though the subcortical area rather than the cortical area was affected. In like manner, the authors found that the patients in the first two cases suffered from subcortical damage and yet exhibited some symptoms that were similar to Broca's aphasia and had nonfluent speech.

In both cases, there was involvement of the anterior limb of the internal capsule and the basal ganglia and an anterior superior extension to the paraventricular white-matter in the left hemisphere. However, the lesion extension in case 1 (S5-S9) was more extensive than that in case 2 (S5-S7). While the lesions in both cases involved the subcortical area in the left hemisphere, the area of involvement in case 1 was slightly different and more extensive. Also, while some of their respective manifestations were similar to nonfluent aphasia, others were different. In auditory comprehension, case 1 revealed a transcortical feature with severe involvement. However, case 2 revealed partial involvement of auditory comprehension.

In reviewing the data from these two cases, we might conclude that even though the lesions may be relatively similar in size and in a similar location, they may show themselves differently. This difference may be due to the fact that the motor speech pathways are close to each other and were affected in one case but not the other. These motor pathways extend through or involve the internal capsule, the basal ganglia, and the anterior-superior extension of the PVWM. In 1982, Naser et al. also proposed a similar finding to explain different clinical manifestations of similarly located and sized lesions.

Another major characteristic of these two cases was that they both made remarkable progress after the onset of the diseases, and this is vividly seen in Table 3. Specifically, they both demonstrated tremendous progress in spontaneous speech, auditory comprehension, repetition of words, and visual confrontation and naming. In auditory comprehension, cases 1 and 2 had impairments of different types and severity. The auditory comprehension of case 1 jumped radically during the first week after the evaluation, and then made steady improvement. But in case 2, the auditory comprehension just improved steadily. In looking at these two cases, the rapid and mark improvement in case 1 may be attributed to the fact that the initial evaluation was held 1 week earlier than that

of case 2. Given the early marked improvement of case 1, one could infer that it was the result of the brain edema subsiding at an early stage.

Both cases achieved near total improvement in auditory comprehension, but still had some deficit in word discrimination. Word repetition was normal in both cases, but there was still a little deficiency in visual confrontation naming. Their spontaneous speech also showed steady improvement to nearly a normal level. However, the rate of improvement was different in each case, with case 1's achievement occurring over 6 months and that of case 2 over a 2-month period. The difference in these two improvement rates of their total speech patterns may be due to the different size of lesions. It should be noted that this substantial improvement pattern differs radically from Broca's and Wernicke's aphasia cases. Knopman et al. (1983) found that some patients who had subcortical aphasia demonstrated significant improvement. We also had similar findings. This marked improvement may be attributed to any one or more of the following reasons. First, this may be due to the natural reabsorption of the hemorrhage. Second, compensation may be easier if the damaged area involves the underlying white-matter and basal ganglia but not the cortical area. Third, the therapy program might have also been a contributing factor to the remarkable speech and motor recovery. Shewan and Kertesz (1984) felt that some of the improvement in aphasia cases might be attributed to the therapy factor.

In case 3, the patient had an involvement of the posterior limb of the internal capsule and the thalamus and a posterior extension of the PVWM that demonstrated itself in poor auditory comprehension. However, he was a fluent speaker with mild dysarthria and semantic paraphasia. The clinical findings in this case were similar to those found in Wernicke's aphasia cases, but in this case the grammatical form of speech was good. Given this point, it differs from Wernicke's aphasia as cited by Goodglass and Kaplan in 1972, but concurs with the Glosser, Kaplan, Lo Verme study in 1982 (Glosser, Kaplan, & Lo Verme, 1982) on left thalamic-putaminal lesion. Also the repetition ability in case 3 was much better preserved and is similar to that of patients with "transcortical sensory features." Finally, in the follow-up telephone conversation, it was found that the spontaneous speech also improved markedly to a near normal level.

Given these motor and speech manifestations, case 3 demonstrated transcortical sensory features that may have been due to the involvement of the left thalamus as it was delineated by Alexander and Lo Verme in 1980. The cause of poor auditory comprehension with fluent speech could be due to involvement of the posterior extension of PVWM and/or the posterior limb of the internal capsule. Recently, Lo Verme (1980) and Wallesch et al. (1983) reported similar findings and conclusions. This possibly means that the cortical-subcortical-cortical loop with its double

projection to the neostriatum might well participate in producing responses among the corticocortical precesses. Selnes, Knopman, Niccum, Rubens, and Larson (1983) and Wallesch (1985) support this proposition. The marked recovery in auditory comprehension over the first 3 months may have been due to the disappearance of the hemorrhage pressure and the compensation by other subcortical fibers that were not damaged. Also it is important to remember that lesions in the postlimb of the internal capsule, thalamus, and the PVWM can induce hemiplegia and fluent aphasia.

Cases 4 and 5 had similar infarctions in areas over the left basal ganglia and the left internal capsule and an anterior/posterior extension of the paraventricular white-matter. They were similar in that they both clinically displayed symptoms of global aphasia. Both patients exhibited no speech or only stereotyped monosyllables. In fact, their spontaneous speech, auditory comprehension, repetition of word, and visual confrontation naming were nonexistent. The follow-up of these two cases during the third month indicated that no significant improvement in spontaneous speech or auditory comprehension occurred.

The severely limited speech output of these two cases of subcortical global aphasia may have been the result of the isolation of Broca's/Wernicke's areas from the internal capsule, basal ganglia, and the genu of corpus callosum as implied by Kertesz (1983). Although the pathology of cases 1, 2, and 3 (hemorrhage) was different from that of cases 4 and 5 (infarction), in retrospect, the damage in the respective areas determines whether it is partially involved as in cases 1, 2, and 3 or totally involved as in cases 4 and 5. Similarly, if the lesion involves only the anterior or posterior area, it is more likely to be less severe than a lesion that longitudinally affects the anterior/posterior portion of the brain. This may mean that deep-seated structures are also very important in speech manifestations (Brunner, Kornhuber, Seemuller, Suger, & Wallesch, 1982; Alexander & Naeser, 1984). Although the person's natural compensation mechanism is directly affected by the location of the lesion (Knopman, Selnes, Niccum, Rubens, Yock, & Larson, 1983), we found that the anterior-posterior lesion was difficult to compensate in our cases—the mechanism may be equally affected by lesion pathology due to different recovery processes. In our hemorrhagic cases we had an earlier and better recovery rate than in the infarcted cases. Scott and Miller (1985) had similar observations on their patients' rapid recovery from small supratentorial intracerebral hemorrhages, and they also noted no rapid improvement in thrombotic or embolic events. It should be noted however that how the natural pathophysiological compensation mechanism works is unclear. Still, the pathology of the brain lesion plays as important a role in affecting the natural compensatory mechanism as the location of the brain lesion does.

In conclusion, the CT scan had provided us with more detailed information than ever before, especially in localizing the lesion, helping to clarify the symptoms of aphasia, and classifying patients. And yet, definitive correlations regarding size and severity, location and symptoms, etc. cannot be conclusively made in every situation (Kertesz, 1983; Reinvang & Dugstad, 1981; Freedman, Alexander, & Naeser, 1984). For example, the size of the lesion can generally be correlated with the severity of the disease, but there are still many exceptions. In our study, the lesion area was mainly located in the B/W slice and the SM slice of the five cases with variant forms of subcortical aphasia; the size of the lesion was the largest in case 5 and there were severe symptoms of total global aphasia. But the lesion in case 4 was smaller than that in case 1, and yet the patient didn't recover. In some instances, the size of the lesion resulting in partial involvement of the speech mechanism may be larger than that of the lesion resulting in total involvement of the same mechanism. In other words, the site of the lesion may be more important than the size of the lesion in producing symptoms.

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