

ELECTROPALATOGRAPHIC ANALYSIS OF APRAXIA OF SPEECH IN A LEFT HANDER AND IN A RIGHT HANDER

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SUMMARY

Two cases with 'pure' apraxia of speech are reported. The articulatory disturbances were quite similar. One of the two cases was a left-handed male with a subcortical haemorrhage and the other a right-handed male with a cerebral infarct. The MRI and CT scans showed that the first case had a lesion that mainly involved the right precentral gyrus and its deep white matter, and that the second had a lesion mainly affecting the lower parts of the left precentral and postcentral gyri and their deep white matter. These findings and a literature review suggest that a corticosubcortical lesion of the lower part of the left precentral gyrus in most right handers and a lesion of the symmetric region in the right hemisphere in some left handers cause apraxia of speech.

The omission errors for sounds articulated by the tongue and the hard palate were analysed using electropalatography, which records visually the dynamics of the palatolingual contact. The results demonstrated that there were three kinds of omission errors: true omissions (no palatolingual contact); omissions with incorrect contact (palatolingual contact for a different sound or undifferentiated sound); and omissions with correct contact (correct palatolingual contact for a target sound). The latter two types of omission error were observed for initial consonants and they were probably caused by a delay in air flow. The patients also showed a tendency to substitute one of the two consonants /t, tʃ/ for other sounds, which suggested that they had difficulty in the inhibition of tongue activity.

INTRODUCTION

It has been known that a unilateral hemisphere lesion uncommonly causes disturbance limited to speech output involving neither auditory nor reading comprehension nor writing disturbance. The syndrome has been variously termed aphemia (Broca, 1861; Bastian, 1898), pure anarthria (Marie, 1906), pure word dumbness (Wernicke, 1906) or pure motor aphasia (Dejerine, 1914). We will use the term 'aphemia' for brevity.

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Aphemia represents at least two types of disturbance. Some patients with aphemia show an inability to articulate all except a very limited set of phonemes or words (Dufour, 1895; Ladame and von Monakow, 1908; Souques, 1928), while others are able to utter most phonemes but show many articulatory errors such as substitutions, additions, omissions and distortions (Denny-Brown, 1965; Lecours and Lhermitte, 1976; Tonkonogy and Goodglass, 1981). These errors are characterized by inconsistency in two ways: phonemes are articulated incorrectly at one time but correctly at another; and errors are not always the same. The second type of disturbance has been reported after recovery from the first (Denny-Brown, 1965; Lecours and Lhermitte, 1976) but not in others (Dufour, 1895; Souques, 1928). F. L. Darley (unpublished) designated the second type 'apraxia of speech'. These two types of disturbance are different from dysarthria arising from bilateral damage of the corticobulbar tracts, the cerebellum or the lower motor neuron whose speech has normal length and where errors tend to be consistent.

Although the articulatory characteristics of apraxia of speech associated with aphasia have been thoroughly studied by systematic phonetic analysis (Shankweiler and Harris, 1966; Johns and Darley, 1970; Trost and Canter, 1974), those of 'pure' apraxia of speech have not been well studied. Even in the well described case with pure apraxia of speech (Lecours and Lhermitte, 1976), the size of the speech sample was not large enough for systematic phonetic analysis.

In the present paper, 2 cases of aphemia (pure apraxia of speech) will be reported. They uttered a very limited number of phonemes at an early stage. At a later stage, they began to produce most phonemes but with numerous articulatory errors. Thus their disturbance at its later stage is well covered by the term 'apraxia of speech'. The utterances of the 2 cases were studied systematically by auditory phonetic analysis to elucidate the articulatory characteristics of apraxia of speech.

Among the various kinds of articulatory errors in apraxia of speech, substitution errors have attracted most attention in the literature. Omission errors, however, are also supposed to characterize apraxia of speech. Electropalatography was employed to observe directly the palatolingual contact during omission errors of our 2 cases in order to clarify the characteristics of this type of error.

The location of the lesion responsible for aphemia or apraxia of speech is still uncertain. Lecours and Lhermitte (1976) have suggested that, in the right-handed, a lesion of the lower part of the left precentral gyrus causes the syndrome. However, a number of cases are needed to confirm the site of the lesion responsible for the syndrome in the right-handed. In the left-handed, so far as we know, no study has established the site of the responsible lesion. One of the 2 cases in this study was left-handed, and the other right-handed. Magnetic resonance imaging (MRI) and computerized tomography (CT) were administered to localize the site of their lesions.

CASE HISTORIES

Case 1 (K. O.)

A 27-yr-old left-handed man, a high school graduate, without prior medical problems, had a subcortical haemorrhage in the right frontal lobe on June 2, 1984. The patient was left-handed from infancy. The left hand was used for all activities except writing and eating with chopsticks. As for these two activities, he had used his left hand at first, but was forced to change to his right hand in childhood. He was left-handed except for writing on the Oldfield Handedness Battery (Oldfield, 1971). His parents and 3 siblings were all right-handed.

Neurological examination on admission (June 2, 1984) revealed impaired consciousness, a left hemiplegia including the lower face, deviation of the tongue to the left, and a left hemihypaesthesia primarily affecting the left upper limb. Two days later he was alert, but with a mild hemiparesis predominantly in the left upper limb. He uttered only /o/, /N/ and /hai/, the latter two meaning 'yes' in Japanese. Angiography disclosed an arteriovenous malformation, which was fed from the right precentral artery and was drained to the right superficial sylvian vein.

The patient was operated upon on June 6, 1984. A craniotomy was performed in the right frontoparietal region. A semiliquid haematoma ($6 \times 5 \times 4$ cm) and the nidus were removed. Neurological examination 1 month after onset showed a mild left-sided lower facial weakness, deviation of the tongue to the left and a left arm monoparesis. There were no signs of left hemihypaesthesia. Two months after onset, neurological examination showed a mild left-sided lower facial weakness and a mild paresis of the left arm. Otherwise examination was normal. Tongue deviation was not present. Pure tone threshold audiometry showed normal hearing levels in both ears despite a 40 dB loss in the left ear and a 15 dB loss in the right at 8000 Hz. Speech audiometry with monosyllabic speech sounds (67-A list a standardized single-syllable list for speech discrimination test by the Japanese Audiological Society) was within normal limits in both ears.

Neurological findings. MRI (Picker International Ltd, VISTR-MR 0.5T) was carried out with a 0.26 Tesla superconducting magnet, using the inversion recovery technique, 10 months after onset. The images (fig. 1) showed an abnormal low intensity region mainly in the right precentral gyrus and its underlying deep white matter such as the middle region of the corona radiata and the right arcuate fasciculus. CT scans parallel to the orbitomeatal line were administered with Toshiba Medical Equipment TCT-60A 1 month and 18 months after onset. They revealed findings compatible with MRI although contaminated with artefacts from the surgical clips (fig. 1).

Language testing. For 10 days after onset, his spontaneous speech was limited to /a/, /i/, /u/, /o/, /N/, /uN/ and /hai/ the latter three meaning 'yes' in Japanese. The patient could not even say his name. Oral repetition was severely disturbed. He could not repeat very familiar phrases such as /ohajo:/ (good morning), for which he uttered /ohooow/. However, he correctly understood questions about his illness and family. He could also comprehend oral commands, such as 'Close your eyes' and 'Open your mouth' and could carry out these commands. He had no difficulty in writing the names of common objects, although he showed a slight disturbance in writing sentences.

The Japanese edition of the Western Aphasia Battery (WAB) (Kertesz, 1982; Sugishita *et al.*, 1986) 0.8 month after onset revealed a marked discrepancy between his speech disturbance and other abilities, namely those of auditory comprehension, reading comprehension and writing (Table 1). In the spontaneous speech task, his disturbance was severe. His production was effortful and slow. Substitution, omission and distortion errors were numerous. He could utter only 9 different phonemes. Among them, 3 different phonemes happened to occur twice in his answers. These were articulated correctly at one time whereas they were misarticulated at another. However, his speech consisted of the correct number of syllables with an approximation of vowels when the target utterances were short. For example, when he was asked 'What is your name?', he uttered '/ooajanieii/', the vowels of which were all correct, though his utterance included 3 omission errors on consonants. To the question 'What is your occupation?', he responded with /anikjaNN/ for /kaifaiN/ (businessman).

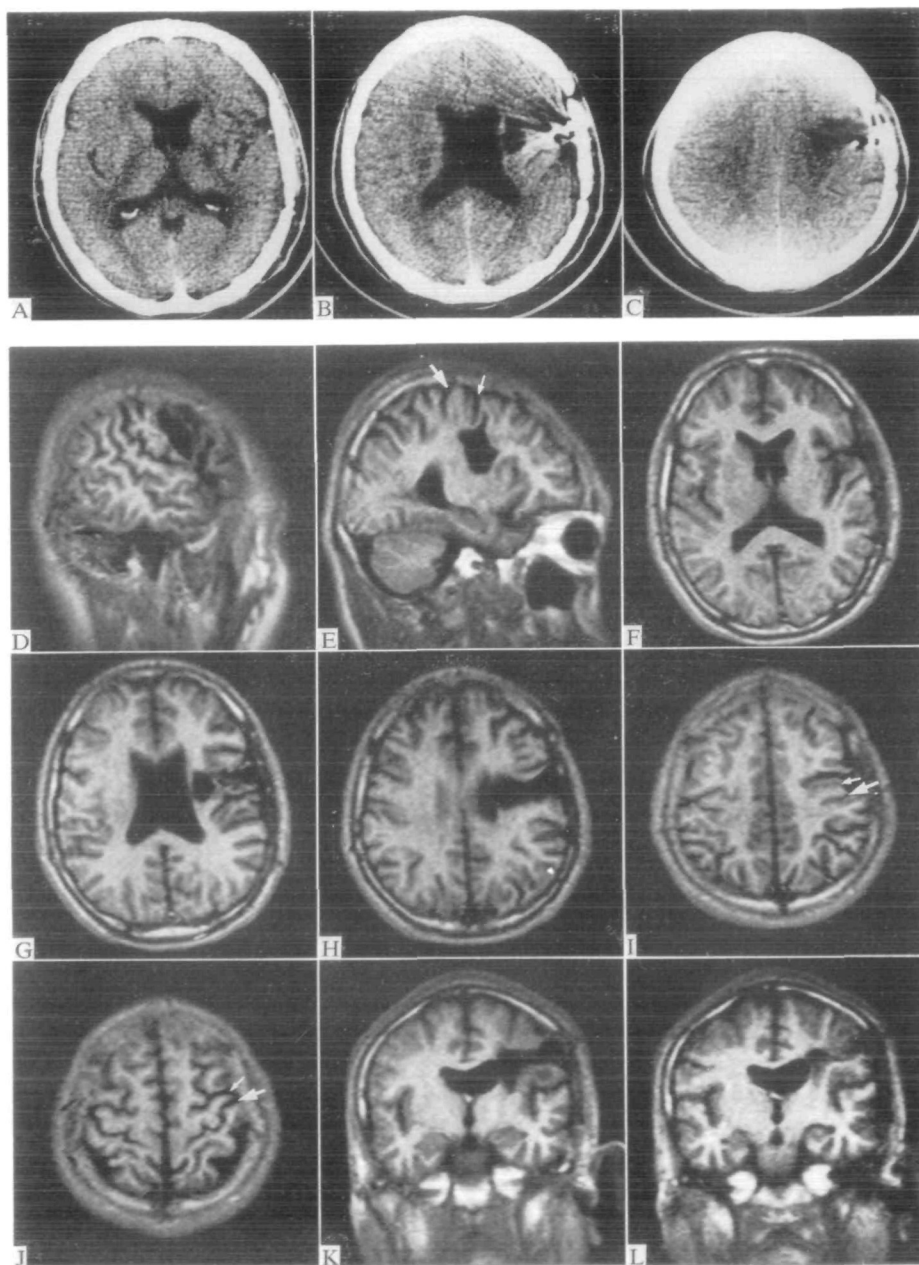


FIG. 1. A-C, CT scans from *Case 1*, 18 months after onset showing an abnormal area of low density mainly affecting the right precentral gyrus extending deeply into the underlying white matter, although artefacts from the surgical clips make the scans unclear. D-L, MRI from this case, 10 months after onset, revealing an abnormal low intensity area in the right precentral gyrus including the middle region of the right corona radiata and the right arcuate fasciculus. Right central sulcus (large arrows); right precentral sulcus (small arrows).

TABLE 1 WESTERN APHASIA BATTERY SUBSCORES (WAB)

	Case 1		Case 2	
	Interval postonset tested			
Subtest category (maximum)	0.8 mo.	2.8 mo.	1.3 mo.	9.6 mo.
Spontaneous speech				
Information content (10)	3	9	0	8
Fluency (10)	1	9*	0	9*
Comprehension (10)	9.6	10	9.6	10
Repetition (10)	3.6	6.7	0	5
Naming (10)	6**	6.8**	0.1	6.5**
Reading (10)	8.2	9.7	6.9	9.4
Writing (10)	8.5	9.9	6	8.7
Praxis (10)	10	10	9.9	10
Construction				
Drawing (3)	3	3	3	3
Block design (0.9)	0.9	0.9	0.9	0.9
Calculation (2.4)	2.4	2.4	2.4	2.4
Raven's score (3.7)	3.4	3.5	2.9	2.8

*Fluency evaluation in the WAB includes phrase length, melody, syntax, paraphasia and word-finding difficulty. The high scores in fluency are due to lack of agrammatism and word-finding difficulty. **The relatively low scores in the naming task are due to substitution errors in sounds.

His abilities to repeat and to read aloud were also severely impaired. During the repetition task, he uttered /anana/ for /banana/ (banana) and /enwaanatte:a:sw/ for /denwaganatteimasw/ (The telephone is ringing). He had no response (naming difficulty) to 1 out of 20 objects in the naming task and articulatory errors to 19 out of the 20 objects. As illustrated in these examples, vowels were less affected than consonants. The patient showed many articulatory errors, such as substitutions, omissions, additions and distortions of sounds. Self-correction was seldom observed.

On the other hand, auditory comprehension, reading comprehension and writing were intact. Auditory comprehension of words was tested with 76 items, and in this he had a perfect score. With 'Yes-No questions' to assess auditory comprehension of sentences such as 'Do you eat a banana before you peel it?' and 'Is a horse larger than a dog?', he correctly responded to 19 out of 20 questions. Oral sequential commands varied from relatively short and easy commands, such as 'Raise your hand', to rather complicated ones such as 'Put the comb on the other side of the pen and turn over the book.' Ten of these 11 commands were correctly executed. His reading comprehension was also nearly intact. In the task where he read sentences and then chose the missing words or phrases, he correctly answered 7 out of 8 items. His writing ability was also quite well preserved, and he wrote 3 sentences describing 10 objects with only 2 spelling errors when requested to describe a presented picture.

The WAB was retested 2.8 months after onset (Table 1). Though his speaking ability improved to the extent that he often uttered short sentences, articulatory errors such as substitution, omission, addition and distortion errors were still numerous. Compared with those of 0.8 month after onset, omission and substitution errors for consonants 2.8 months after onset decreased, while distortion errors for consonants increased. Substitution and distortion errors for vowels decreased. His speech showed prosodic abnormalities, such as the slow speaking rate resulting from a prolongation of sounds and inappropriate intersyllabic pauses, and monotonous pitch. These disturbances were observed not only in spontaneous speech but also in repetition and reading aloud. Syntax was normal. He had no response to 1 out of 20 objects in the naming task and had articulatory errors to 14 of these objects.

On the other hand, he had perfect scores for auditory comprehension of sentences and of oral

sequential commands. He had no errors for reading comprehension at the two to four-sentence level or for executing written commands that included complicated commands such as 'Pick up the pencil, knock 3 times and put it back.' His written description for a presented picture was adequate and accurate using 3 sentences within 3 min. In the Token Test 2 months after onset the patient performed perfectly. Since his disturbance was limited to oral expression with auditory and reading comprehension and intact writing ability, he was diagnosed as having aphemia. His disturbance at the 2.8 months stage where most of Japanese phonemes appeared in his utterance constituted a pure form of apraxia of speech. His apraxia of speech was still observed 2 yrs after onset, though it had been moderately alleviated.

Praxis and gnosis testing. At 0.8 month after onset, he performed perfectly the drawing, block design and praxis subtests of the WAB. There were no indications of constructional, ideomotor or ideational apraxia. He correctly performed the 5 items of the orofacial region in the praxis subtest and the other oral commands such as 'Swallow', 'Puff out your cheeks' and 'Make your teeth chatter'. In contrast, he had some difficulty in executing the commands 'Cough' and 'Click your tongue', indicating a mild orofacial apraxia. At 2.8 months after onset, he perfectly performed the latter two commands. He did not show unilateral hemineglect in drawing pictures. Other apraxias or agnosias were not observed.

Intelligence testing. One month after onset, he attained a verbal IQ of 74 and a performance IQ of 85 on the Wechsler Adult Intelligence Scale. His best performance was on the block design subtest (scaled score: 14).

Case 2 (T. I.)

A 58-yr-old right-handed man, a middle school graduate, suddenly suffered from weakness and numbness on the right side of his body and speech disturbance on January 4, 1984. He was right-handed, as were his parents, 5 siblings and 2 children. He had been hypertensive for 20 yrs.

Neurological examination 1 month after onset showed right-sided lower facial weakness, deviation of the tongue to the right, a right hemiparesis predominantly affecting the right arm, and right hypaesthesia. The plantar response was extensor on the right. Neurological examination was otherwise negative. A gag reflex was present and there was no difficulty in swallowing. The visual fields were normal. The right hypaesthesia cleared by 2 months after onset. Fibrescope examination revealed that movements of the larynx and pharynx were normal. Six months after onset, neurological examination showed mild right-sided lower facial weakness and a mild right hemiparesis predominantly affecting the right arm. The plantar response remained extensor on the right. Sensory function and the visual fields were normal. Deviation of the tongue was no longer present. Pure tone audiometry and speech audiometry with the 67-A list were within the normal range.

CT scans 1 month after onset and MRI 1 yr after onset showed an infarct mainly affecting the left precentral and postcentral gyri and their underlying deep white matter, such as the middle region of the corona radiata and the left arcuate fasciculus. The left insula was also involved (fig. 2).

Language testing. For 1.3 months after onset, spontaneous speech was effortful and slow and consisted of only 1 to 3 syllables. It was limited to /a/, /i/, /o/, /N/, /afi/ (foot) and /ohajo:/(good morning). The patient was unable even to say his name.

The WAB was carried out 1.3 months after onset (Table 1). The patient was able to utter only 1 or 2 /N/ to each of the 7 questions in the spontaneous speech task. In the naming task, he had no responses to any of the 20 objects. When the initial syllable of the names were given as cues, he said /boi/ for /bo:l/ (ball) and /maio/ for /naiΦw/ (knife). In the repetition task, substitution, omission and distortion errors were observed. For example he said /ahaha/ for /banana/ (banana). He uttered only 6 different phonemes. The phoneme /n/ occurred four times in his utterances and was articulated correctly once and variously misarticulated three times. There were few self-corrections in his utterances.

He also showed a severe disturbance for reading aloud. On the other hand, auditory comprehension

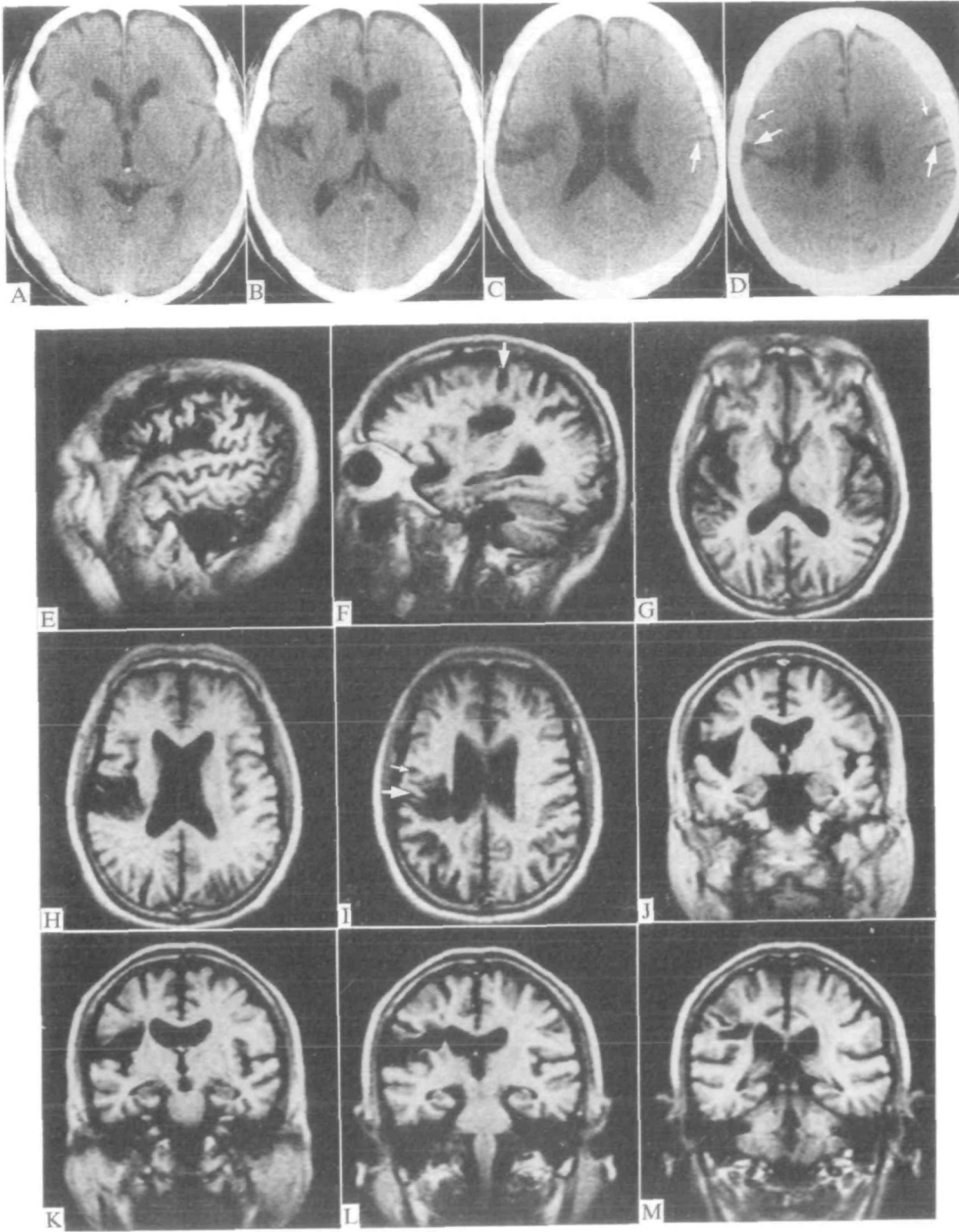


FIG. 2. A–D, CT scans from *Case 2*, 1 month after onset demonstrating an abnormal low density area in the lower parts of the left precentral and postcentral gyri extending deeply into the middle region of the corona radiata. The left arcuate fasciculus and the left insula were also affected. E–M, MRI from this case 1 year after onset revealing an abnormal low intensity area in almost the same area of the left hemisphere. Left central sulcus (large arrows); left precentral sulcus (small arrows).

was well preserved. He responded correctly by gesture to 19 out of 20 'Yes-No' questions and understood and performed correctly all 11 oral sequential commands. In the reading comprehension task, he executed correctly all of the 6 written commands, although he erred on 2 out of the 8 questions which required comprehension of a paragraph. When asked to write a description of a picture, he wrote only 9 nouns with no sentences. His written naming of a stamp, a watch, a pencil and woollen yarn was correct in ideograms (kanji), but showed a mild disturbance in phonograms (kana). However, there was a striking discrepancy between his writing disturbance and that of his speech. In summary, the patient showed severe speech disturbance and mild disturbances in writing and in reading comprehension 1.3 months after onset.

The WAB was readministered 9.6 months after onset. He made no errors in reading comprehension task or dictation of words such as stamp and woollen yarn (Table 1). In the written description of a presented picture he wrote 2 correct sentences having a total of 22 words in 3 min with 2 minor mistakes. He made no errors in writing 25 phonograms. He had perfect scores in the auditory comprehension task and in sequential commands. On the other hand, his speech disturbance was severe. Spontaneous speech and oral repetition was severely hampered by substitution, omission, distortion and addition of sounds, although he had improved to the extent that he often uttered short sentences. In the oral repetition task, omission errors for consonants 9.6 months after onset had decreased compared with the result 1.3 months after onset. Substitution and distortion errors for vowels also decreased. Abnormal prosody characterized by a prolongation of sounds, syllabification and monotonous pitch was apparent. In the naming task, he had no response to 1 out of 20 objects and had articulatory errors to 19 objects. Syntax was within normal limits.

In the Token Test during the same period, the patient had errors on only 2 items in Section F. In summary, his deficits, characterized by isolated severe speech disturbance, was classified as aphemia. His disturbance at the 9.6 month stage, when most phonemes appeared in his speech, constituted an apraxia of speech. The WAB was also tested at 5.3 months after onset in addition to 1.3 months and 9.6 months after onset. The results obtained were very close to those at 9.6 months after onset except that he wrote only one sentence plus 1 word within 3 min in the written description of a picture. His disturbance at this stage is adequately termed a pure apraxia of speech. His apraxia of speech was still present 2 yrs after onset.

Praxis and gnosis testing. At both 1.3 months and 9.6 months after onset, he performed the drawing, block design and praxis subtests in the WAB perfectly, which suggested that he did not have constructional, ideomotor or ideational apraxias. One month and 2 months after onset, he correctly performed commands such as 'Open your mouth' and 'Stick out your tongue.' However, he could not 'Cough' or 'Click his tongue', which suggested a mild orofacial apraxia. These two commands were correctly carried out 6 months after onset. Unilateral hemineglect was not observed when drawing pictures. Other apraxias or agnosias were not observed.

Intelligence testing. He attained a performance IQ of 105 and a verbal IQ of 81, aided by writing, 1.3 months after onset. Five and a half months after onset, his verbal IQ was 96, and his performance IQ was 104.

EXPERIMENTAL INVESTIGATIONS

Three articulation tests were employed to obtain speech samples of the 2 patients. Auditory phonetic analysis and electropalatography were used to analyse the speech samples.

Articulation

Three articulation tests were administered to assess the speech disturbance of the 2 patients. One was the Articulation Test developed by the National Rehabilitation Center for the Disabled (ATNR). The second test, the Articulation Test of the Tokyo Metropolitan Institute for Neurosciences (ATTM), had been used for patients before the ATNR was adopted. Most of the test items of the

ATTM were included in the ATNR. The third was a 100-syllable repetition test. The three tests will be briefly described.

The ATNR. This test included 82 common nouns of from 2 to 6 syllables in length such as dog, glasses and newspaper. It was administered both as a naming task and for oral repetition. In the naming mode, 82 pictures representing the 82 words were presented, and the patient was asked to name each of them three times successively. In the oral repetition mode, the patient repeated each word three times after the examiner had pronounced it once.

Japanese is an open-syllable language. That is, syllables consist of one consonant and one vowel with or without an intervening /j/ after the consonant. The syllable can, however, be closed with the nasal sound /N/. Therefore there are no final consonants except for /N/ in syllables. /N/ is a unique phoneme which is realized as [m, n, ŋ] and the nasalization of the previous vowel. In this paper, therefore, /N/ was excluded from the analyses.

The total number of phonemes in the 246 responses (82 words \times 3) was 1176, with 537 consonants excluding /N/, 615 vowels and 24 consonant-semivowel clusters. The total numbers of initial consonants and medial consonants in test words were 210 and 327, respectively. All of the Japanese consonants (21 in number): /p, t, k, s, ʃ, Φ, ç, h, b, d, g, ts, tʃ, r, m, n, ɲ, dz, dʒ, j, w/ were tested both in initial and medial positions in words. Four consonant-semivowel clusters, /kj/, /bj/, /gj/, and /rj/, appeared 15 times in initial positions in words, and 3 consonant-semivowel clusters, /pj/, /bj/, and /gj/, appeared 9 times in noninitial positions. All of the Japanese vowels (5 in number): /a, o, e, u, i/ were tested in initial, medial and final positions. The number of vowels tested was 615 (21 initial, 399 medial and 195 final vowels).

The ATTM. This test consisted of 32 common words with 2 to 5 syllables. The consonants involved included all of those in the ATNR except for /tʃ, ɲ, dz/. The number of phonemes in the 32 words, excluding /N/, was 163. The patient was asked to name each word 3 times, so that the total number of phonemes tested was 489 (163 \times 3). The number of consonants was 225 (81 initial and 144 medial consonants) and the number of vowels 255 (9 initial, 153 medial and 93 final). The number of consonant-semivowel clusters was 9. This test was administered only to Case 2 and only in the naming mode.

The 100-syllable repetition test. This test consisted of 100 monosyllables which included all of the possible syllabic combinations of Japanese phonemes. The patient was asked to repeat each syllable twice in succession.

Auditory phonetic analysis

The responses of the patients were recorded with a cassette tape recorder (Sony TC-D5M) and narrow phonetic transcription using the International Phonetic Alphabet was carried out. The error responses were categorized into two types according to Trost and Canter (1974): inadequate responses and phoneme errors. Inadequate responses included (1) random phonemes, where phoneme strings failed to yield any kind of word approximation; (2) perseverative responses; (3) paraphasic responses, a semantic substitution such as 'hat' for 'coat'; (4) recurrent utterances; and (5) no response (in this study, naming difficulty). Phoneme errors were those responses which were close to the target word but had some errors in terms of phonemes. The phoneme errors were divided into the following four types of errors: (1) substitutions, as in /t/ for /p/; (2) omissions, as in /a/ for /na/; (3) additions, as in /to/ for /o/; and (4) distortions as in /d/ for /d/. Compound errors, such as distorted substitution or distorted addition errors, were categorized as substitution or addition errors, respectively. The occurrence of sequential errors and self-corrections were also analysed separately. Sequential errors included anticipatory errors such as '/to:to/' for '/no:to/' (note book), perseverative errors like '/memane/' for '/megane/' (glasses) and metathetic errors like '/tsukw/' for '/kwtsu/' (shoes).

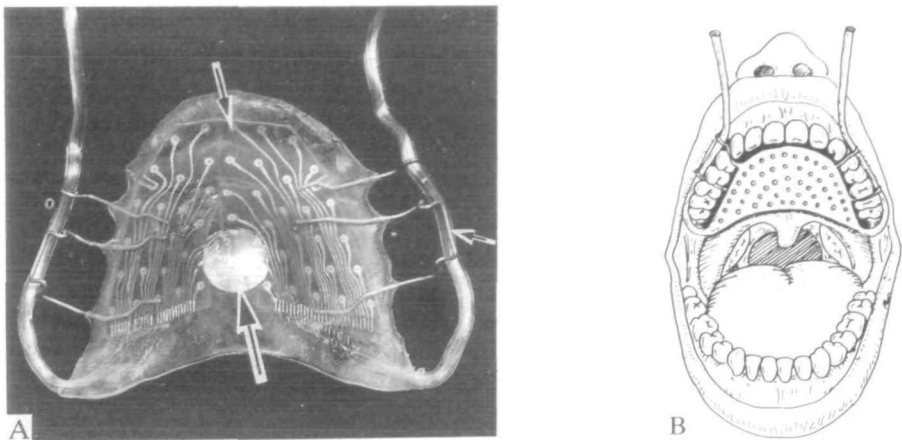


FIG. 3. Artificial palate. A, photograph of an artificial palate. Receiving electrode (large arrow); one of the transmission electrodes (middle-sized arrow); lead wire (small arrow). B, artificial palate fitted against the patient's palate.

Electropalatography

Electropalatography (Rion Co. Ltd) was employed to observe the palatolingual contact during the entire ATNR and ATTM naming tests. The system of electropalatography used in this study included an artificial palate, a display panel with a memory system, a recording adapter connected to a dual-channel tape recorder (Sony TC-D5M) and a graphic printer. The artificial palate made of transparent plastic (1 mm in thickness) was formed after a plaster model of the palate of each patient. It had 63 gold transmission electrodes (2 mm diameter each) implanted on its under surface and 1 gold receiving electrode (8 mm diameter) on the upper surface (fig. 3A). The output from a signal source was applied to each transmission electrode through a distributor controlled synchronously by a clock oscillator. The artificial palate was fitted against the patient's hard palate (fig. 3B). When the tongue touched the transmission electrodes the current ran to the receiving electrode and was sensed by detectors. The signals were then transmitted to the memory system which processed the signals at a rate of 64 frames/s; 63 light emission diodes on a display panel showed the positions of the electrodes which the tongue touched. Both the signals and speech were stored on a dual-channel cassette tape via a recording adaptor. The data on the cassette tape could be reproduced and observed in slow motion on the display panel (fig. 4).

The patterns of the palatolingual contacts for the 2 patients were then printed out and compared with those of 5 normal subjects who spoke each word 3 times, giving 15 normal patterns. The data for omission errors were classified into three types of palatolingual contact: correct contact, incorrect contact and no contact. The palatolingual contact patterns of the phonemes omitted by the patients were judged correct when they did not exceed the maximum or minimum contact range of the normal patterns of target sounds. They were judged incorrect when they were within the maximum or minimum contact range of different sounds or they were undifferentiated contact patterns, that is, exceeding the maximum or minimum contact range of any sound. They were judged no contact when they had no palatolingual contact.

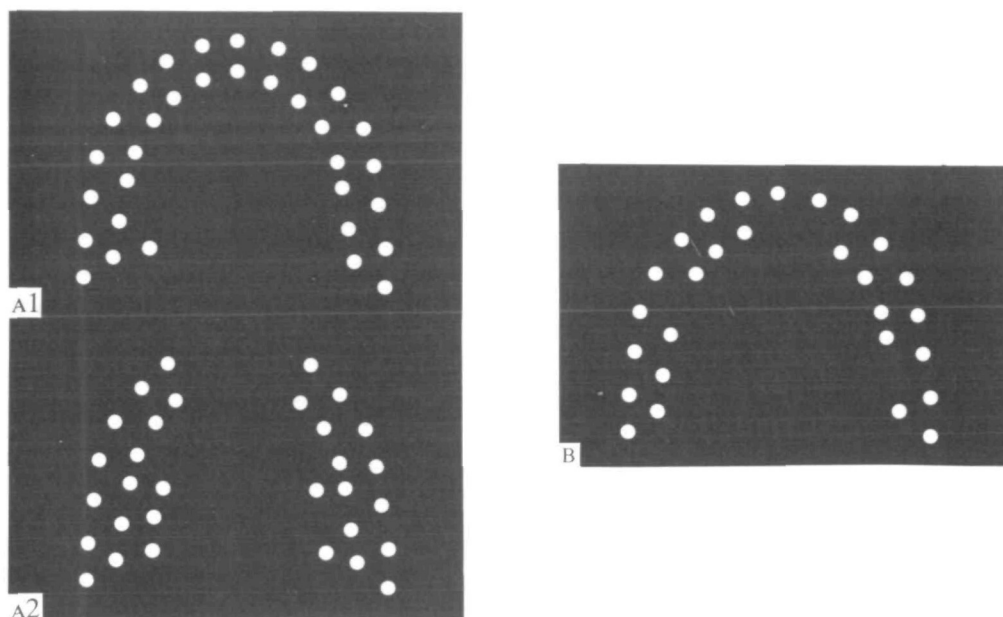


FIG. 4. Electropalatograms from *Case 1* shown on the display panel. Transmission electrodes in contact with the tongue are indicated by the light emission diodes of the display panel which are small circles in the electropalatograms. A1, A2, correct contact of palatolingual pattern for the target sound /dʒ/. A1 is its closure and A2 is its release following closure. B, substituted pattern of palatolingual contact for the target sound /j/ to lingual-alveolar sounds such as /t/, /d/ or /n/. Both (A, B) were perceived as omission errors

RESULTS

Case 1

Auditory phonetic analysis

ATNR naming test. This test was carried out in the naming mode 2.3 months after onset. Out of the 82 test words, 2 (pencil and telephone) were not tested. The 80 pictures representing the 80 words were named 3 times, so that the patient produced 240 responses (80 words \times 3) on which auditory phonetic analysis was performed.

Out of the 240 responses, 55 responses were correct and 185 responses were judged as having phoneme errors. There were no inadequate responses such as no responses (naming difficulties). The 185 phoneme-error responses had 290 erroneous phonemes, that is, 25% of the total number of phonemes (1149). The phoneme errors included 63% (182/290) substitution errors, 22% (64/290) omission errors, 14% (41/290) distortion errors and 1% (3/290) addition errors. There were no omission or addition errors of syllable units. Self-correction responses were only 2% (7/290) of the total errors. Nine sequential errors (8 anticipatory, 1 perseverative) were observed. They accounted for only 3% (9/290) of the total

errors. Further analysis for consonant, consonant-semivowel cluster and vowel errors was as follows.

1. Consonants. The patient made 261 consonant errors, involving 50% (261/525) of the total consonants used in the test (Table 2). There were no significant differences in the numbers and the types of errors between the first, second and third responses. The percentage of errors was 74% (153/207) for initial consonants and 34% (108/318) for medial consonants, indicating that initial consonants elicited more errors than medial consonants ($\chi^2 = 80.1$, $P < 0.01$). Errors were observed for all 21 consonants. The consonant with the lowest percentage of errors was /ɲ/ (17%) followed by /tʃ/ (29%), /m/ (29%), /w/ (33%), /k/ (40%) and /t/ (41%). Those with higher percentages were /dz/ (95%) /ts/ (88%) /s/ (81%) /d/ (73%) and /dʒ/ (73%) (fig. 5). This indicated that the most difficult consonants were affricates and fricatives.

Out of 261 consonant errors, 63% were substitutions, 23% omissions, and 14% distortions, with no errors of addition (Table 2). Substitution errors occurred for all 21 Japanese consonants, and were highly inconsistent. Seventeen consonants which elicited more than 3 substitution errors (i.e., /j, p, t, k, b, d, g, m, n, s, h, ts, dz, r, ʃ, tʃ, dʒ/) were substituted by more than 2 different consonants. These consonants were changed to 18 different consonants (/p, t, k, ʃ, Φ, ɕ, h, b, d, g, ts, tʃ, r, n, ɲ, dz, dʒ, j/), with substitutions of /t/ (20), /tʃ/ (18), /k/ (14) and /n/ (13) occurring most frequently.

When analysing the consonants for consonant-substitution errors using the four subphonetic dimensions of Trost and Canter (1974) — that is, place, manner, voicing and oral-nasal — 60% (91) were off by one dimension, followed by those off by two dimensions 29% (45), three dimensions 8% (12) and four dimensions 3% (4). Place was the most frequent error, 48% (111), followed by manner errors 26% (59), voicing errors 19% (43) and oral-nasal errors 7% (16). There were more voiceless substitution errors for voiced consonants, 81% (35), than the reverse type of error, 19% (8), indicating a strong tendency toward devoicing.

Sixty omission errors were observed for 17 different consonants among the 21 different test consonants (fig. 5). While the omission error percentages varied from 29% for /dz/ to 6% for /ʃ/ and /j/, there were no significant differences between

TABLE 2 CONSONANT, VOWEL AND CONSONANT-SEMI VOWEL CLUSTER ERRORS, AND EACH TYPE OF ERROR IN CASE 1, 2 3 MONTHS AFTER ONSET*

	Consonant	Vowel	Consonant-semivowel	Total
Sound tested	525	600	24	1149
Errors (error rate)	261(50%)	17(3%)	12(50%)	290(25%)
Error type				
Substitution	165(63%)	9(53%)	8(67%)	182(63%)
Omission	60(23%)	0(0%)	4(33%)	64(22%)
Addition	0(0%)	3(18%)	0(0%)	3(1%)
Distortion	36(14%)	5(29%)	0(0%)	41(14%)

*The ATNR naming test was employed.

any consonants. Most of the omission errors, 88% (53/60) occurred for initial consonants with 12% (7/60) for medial consonants.

Thirty-six distortion errors occurred for 15 different consonants (fig. 5). The error percentages varied from 29% for /dz/ to 2% for /k/. The distortion errors included palatalization 19% (7), partial devoicing 17% (6), partial omission of sounds 42% (15) and others 22% (8).

2. **Vowels.** Vowel errors occurred much less often than consonant errors ($\chi^2 = 330.8$, $P < 0.01$), only 3% of the total number of vowels (17/600) (Table 2). These vowel errors occurred most frequently for initial vowels at 22% (4/18), compared with medial vowels at 2% (9/393) and final vowels at 2% (4/189) (Fisher's

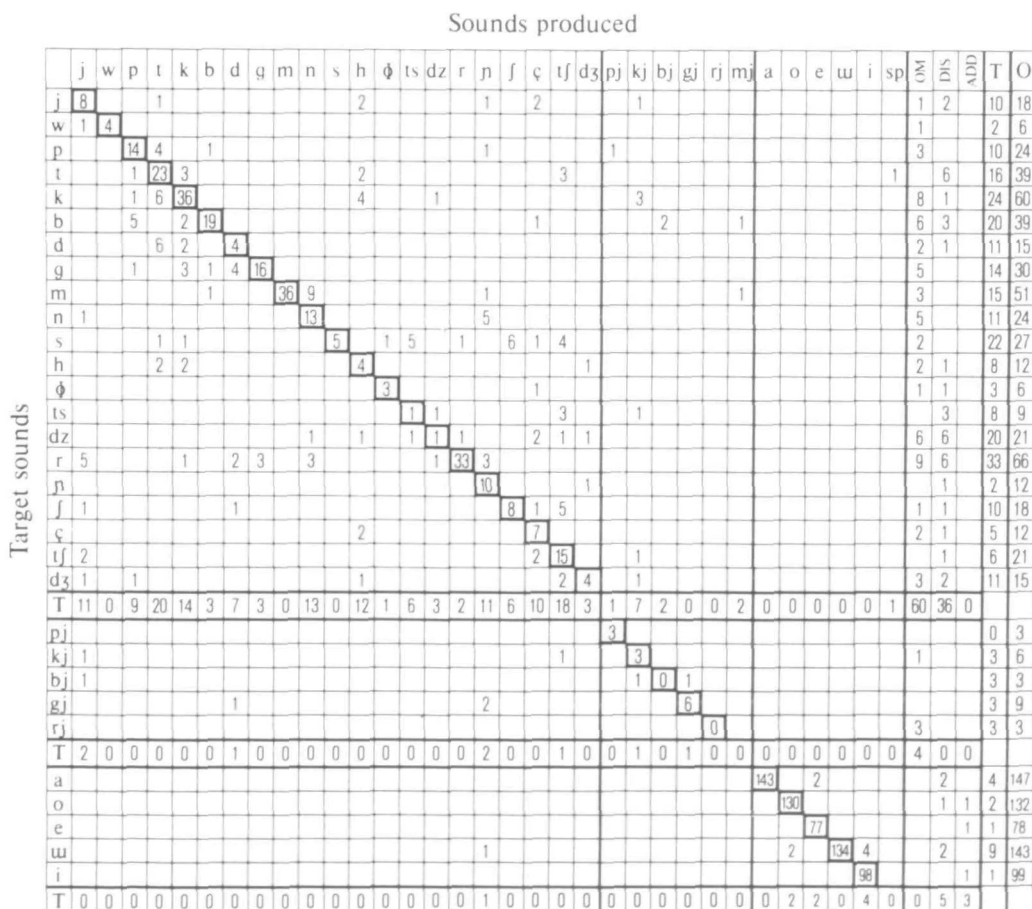


FIG. 5. Confusion matrix of consonants, consonant-semivowel clusters and vowels for *Case 1*, 2.3 months after onset. Each line of the matrix shows the distribution of sounds which the patient produced in response to the target sounds as indicated on the left margin. Cells forming the diagonal are correct productions. SP=sounds not existing in Japanese language but possible to transcribe by International Phonetic Alphabet. OM=omission; DIS=distortion; ADD=addition; T=total number of errors; O=total number of occurrences of each target sound.

exact test, $P < 0.01$). Errors occurred for all 5 vowels. The highest error rate was 6% for /u/ and the lowest was 1% for /i/. Less substitution, omission and addition errors were observed for vowels than for consonants (substitution errors: $\chi^2 = 191.8$, $P < 0.01$; omission errors: Fisher's exact test, $P < 0.05$; distortion errors: Fisher's exact test, $P < 0.01$). Substitution errors accounted for 53% of the total number of vowel errors, followed by distortion errors 29% and addition errors 18%, with no omission errors (Table 2). Eight of the 9 substitution errors were substituted vowels, and only 1 was a consonant.

3. Consonant-semivowel clusters. Out of 24 consonant-semivowel clusters tested, 50% were judged to have errors. The error rate 100% for /bj/ (3/3) and /rj/ (3/3). The lowest error rate was 0% for /pj/ (0/3). Substitution errors were 67%, followed by omission errors at 33%. There were no addition or distortion errors (Table 2).

Electropalatographic analysis of omission errors

In order to clarify the nature of the omission errors, electropalatography was carried out during the entire ATNR naming test, 2.3 months after onset. The phonemes articulated with the tongue against the hard palate in Japanese are 14: /t, d, tʃ, dʒ, ts, dz, ɕ, ʃ, n, ɲ, s, r, j, rj/: 34 of the total of 64 omission errors were for 10 of these 14 phonemes articulated with the tongue against the hard palate and these 34 were submitted to analysis (Table 3). The results revealed palatolingual

TABLE 3 ELECTROPALATOGRAPHIC ANALYSIS OF OMISSION ERRORS IN CASE 1, 2.3 MONTHS AFTER ONSET*

Types of palatolingual contact	Target sounds										Total
	j	d	n	s	dz	r	ʃ	ɕ	dʒ	rj	
Correct contact	1	2	5		4	1		1	1		15
Incorrect contact											
Different sound				1	2	2	0	0	1	3	9 } 14
Undifferentiated sound				1		2	1	1			
No contact						4			1		5

*Scores indicate occurrence of each type of palatolingual contact.

contact for the target phonemes (correct palatolingual contact) in 15 out of the 34 omission errors, and palatolingual contact for different sounds or undifferentiated sounds (incorrect palatolingual contact) in 14 out of the 34 omission errors. On the other hand, those which showed no palatolingual contact — that is, true omission errors — proved to be only 5 out of the 34 (Table 3). All of the omission errors with correct contact (15/15) and most of the omission errors with incorrect contact (13/14) occurred for initial consonants. On the other hand, more than half of the true omission errors (3/5) were observed for medial consonants.

Repetition versus naming

The ATNR was also carried out in the repetition mode 2.3 months after onset. Compared with the result for the naming mode at the same period, no differences were observed either in the error rate or in the distribution of the four types of errors. In the repetition mode, as in the naming mode, no significant differences were found in the rates or types of errors between the first, second and third responses for the test words.

The patient also had great difficulty in doing the 100-syllable repetition test. Sixty-one percent of the phonemes (233/378) were erroneous 0.9 month after onset: consonants 89% (131/148), vowels 36% (70/194), consonant-semivowel clusters 89% (32/36). Four months after onset, the errors had decreased greatly to 27% (106/390): consonants 51% (78/154), vowels 6% (12/200), consonant-semivowel clusters 44% (16/36).

*Case 2**Auditory phonetic analysis*

ATTM naming test. The ATTM, consisting of 32 words, was administered in the naming mode 4.5 months after onset. Thirty-two pictures representing the 32 words were named 3 times, so that 96 responses were obtained. The 96 responses included 6 correct responses and 90 responses judged as having phoneme errors. There were no inadequate responses such as no responses (naming difficulties). One self-correction response was observed. The 90 erroneous responses had 156 phoneme errors, or 32% of the total number of phonemes tested (489) (Table 4). Sequential errors were only 8 (3 anticipatory and 5 perseverative). Errors for consonants were 58% (131/225) and for consonant-semivowel clusters 100% (9/9). Vowel errors were only 6% (16/255). Consonant errors were more frequent than vowel errors ($\chi^2 = 151.8$, $P < 0.01$). Consonant-semivowel cluster errors were more frequent than vowel errors ($\chi^2 = 88.0$, $P < 0.01$). More substitution, omission and addition errors were observed on consonants than on vowels (both substitution errors and omission errors: Fisher's exact test, $P < 0.01$; addition errors, $P < 0.05$). For consonants, substitution errors, at 92% of the total errors, were more frequent than the other types of error. The most frequent for vowels were addition errors (56%) and for consonant-semivowel clusters, substitution errors (78%) (Table 5).

ATNR naming test. The ATNR, consisting of 82 words, was administered in the naming mode 9.6 months after onset, and 246 responses (82 words \times 3) were obtained. Since the patient had no responses (naming difficulties) to 2 pictures representing 2 words (a colouring book and a penguin), 6 responses (2 words \times 3) were judged as inadequate responses. There were 18 correct responses. The remaining 222 responses were judged as having phoneme errors, and they were submitted for auditory phonetic analysis. There were no self-corrections.

We observed 362 phoneme errors in the 222 responses, or 31% of the total 1149

TABLE 4 CONSONANT, VOWEL AND CONSONANT-SEMIVOWEL CLUSTER ERRORS, AND EACH TYPE OF ERROR IN CASE 2, 4.5 AND 9.6 MONTHS AFTER ONSET

	<i>Consonant</i>	<i>Vowel</i>	<i>Consonant-semivowel</i>	<i>Total</i>
4.5 months after onset				
Sound tested	225	255	9	489
Errors (error rate)	131(58%)	16(6%)	9(100%)	156(32%)
Error type				
Substitution	121(92%)	4(25%)	7(78%)	132(84%)
Omission	7(5%)	0(0%)	0(0%)	7(5%)
Addition	1(1%)	9(56%)	0(0%)	10(6%)
Distortion	2(2%)	3(19%)	2(22%)	7(5%)
9.6 months after onset				
Sound tested	525	600	24	1149
Errors (error rate)	301(57%)	41(7%)	20(83%)	362(31%)
Error type				
Substitution	262(87%)	0(0%)	20(100%)	282(78%)
Omission	3(1%)	0(0%)	0(0%)	3(1%)
Addition	0(0%)	30(73%)	0(0%)	30(8%)
Distortion	36(12%)	11(27%)	0(0%)	47(13%)

*The ATTM naming test was employed 4.5 months after onset, and the ATNR naming test 9.6 months after onset.

indicating more errors for initial consonants than for medial ones ($\chi^2 = 72.5$, $P < 0.01$). Errors were observed for all of the 20 consonants except for /*n*/. The error rate was the highest for /*g*/ (27/27) and /*ʃ*/ (6/6) at 100%, followed by /*ç*/ (92%), /*dz*/ (90%), /*f*/ (89%) and /*s*/ (81%), again indicating that the most difficult sounds were fricatives and affricates. The lowest was 0% for /*n*/ (0/12), followed by /*tʃ*/ (14%), /*t*/ (26%), and /*m*/ (29%) (fig. 6). Substitution errors were the most common at 87% (262), followed by distortion errors at 12% (36) and omission errors at 1% (3), with no addition errors.

The substitution errors were inconsistent. Most of the consonants — excluding /*n*/ of no errors and /*ts*/, all of whose substitution errors were to /*tʃ*/ — were substituted for by more than 2 different consonants. Although the direction of the substitution was not consistent, there was a striking tendency for most of the consonants to be substituted for by /*t*/ 26% (68), or /*tʃ*/ 24% (61).

When analysing the consonant-for-consonant substitution errors under the four dimensions proposed by Trost and Canter (1974), most of the errors differed by one dimension 50% (126) or two dimensions 46% (115) from the target, followed by three dimensions 4% (9). Place errors accounted for 46% (175), followed by manner errors at 35% (132), voicing errors at 15% (58) and oral-nasal errors at 4% (16). There was a striking tendency toward devoicing. The patient made more voiceless substitution errors 88% (51) for voiced consonants than the reverse type of error 12% (7).

A total of 3 omission errors was observed for 2 different consonants. Thirty-six distortion errors, which were observed for 9 different consonants, including palatalization 17% (6), partial devoicing 11% (4), partial omission of sounds 3% (1) and others 69% (25).

2. Vowels. Forty-one vowel errors were observed, that is, in 7% of the vowels

tested (41/600). This was much lower than the error rate for consonants ($\chi^2 = 337.5$, $P < 0.01$). These vowel errors occurred most frequently for initial vowels at 90% (19/21), followed by errors for medial ones at 6% (22/387), with no errors for final vowels (0/192) (Fisher's exact test, $P < 0.01$). Errors occurred to all of the 5 vowels. The error rate was the highest for /e/ (9/78) at 12%. The lowest was 3% for /u/ (5/144). Less substitution, addition and distortion errors were observed on vowels than on consonants (Fisher's exact test, $P < 0.01$). Addition errors accounted for 73% of the total number of vowel errors, followed by distortion errors at 27%, while no substitution or omission errors were observed (Table 4). Most of the addition errors occurred for initial vowels. The frequent phonemes added were /t/ (8), /b/ (5), /d/ (4), /dʒ/ (3) and /j/ (3).

3. Consonant-semivowel clusters. The consonant-semivowel clusters having errors numbered 20 (83%) out of the 24 consonant-semivowel clusters tested (Table 4). The error rate was 100% for /kj/ (6/6), /pj/ (3/3) and /bj/ (3/3). The lowest error rate was 0% (0/3) for /rj/. All of these 20 errors were substitution errors.

Electropalatographic analysis of the omission errors

Electropalatography was conducted during the entire ATTM naming test 4.5 months after onset. The results included 7 omission errors for consonants. Six of these were phonemes articulated with the tongue against the hard palate. They were submitted to analysis. The result showed 1 omission error with correct palatolingual contact, 2 with incorrect palatolingual contact and 3 with no palatolingual contact (Table 5). All the omission errors with correct contact and with incorrect contact occurred for initial consonants, while all the errors with no contact — that is, true omission errors — were observed for medial consonants.

At the stage of 9.6 months after onset, electropalatography was conducted during the entire ATNR naming test. The results showed 3 omission errors for consonants. The electropalatography showed correct contact in one of the 3 omission errors and no contact in the remaining 2 omission errors, indicating true omissions (Table

TABLE 5 ELECTROPALATOGRAPHIC ANALYSIS OF OMISSION ERRORS IN CASE 2*

Types of palatolingual contact	Interval postonset tested							Total
	4.5 months Target sound					9.6 months Target sound		
	<i>j</i>	<i>d</i>	<i>dʒ</i>	<i>r</i>	<i>t</i>	<i>r</i>	<i>m</i>	
Correct contact					1		1	2
Incorrect contact								
Different sound		1						1
Undifferentiated sound			1					1
No contact	1			2		2		5

*Scores indicate occurrence of each type of palatolingual contact.

5). The omission error with correct contact was again observed for initial consonant and those with no contact were for medial consonants.

Repetition versus naming

The ATNR in the repetition mode was given to the patient 5 months after onset. Compared with the result for the naming mode at the same period, no differences were observed, either in the errors rate or in the distribution of the four types of errors. There were no differences in the rates or types of errors between the first, second and third responses for each word, as in the naming mode.

In the 100-syllable repetition test 1.5 months after onset, the patient produced errors for 324 (85%) out of the 382 phonemes tested: consonants 91% (137/150), vowels 77% (151/196) and consonant-semivowel clusters 100% (36/36). At 8.5 months after onset, the number of errors decreased to 47% (184/390): consonants 73% (112/154), vowels 20% (39/200) and consonant-semivowel clusters 92% (33/36).

DISCUSSION

Our 2 patients, a left-hander with a right hemisphere lesion and a right-hander with a left hemisphere lesion had disturbances limited to speech output, while their auditory and reading comprehension as well as writing remained nearly normal. During the early period following onset, the patients were able to articulate only very limited sounds. Several months following onset, the patients recovered enough to articulate most of the Japanese phonemes, but showed many inconsistent substitution, omission, distortion and addition errors. The disturbance at this stage is well covered by the term 'apraxia of speech' (F. L. Darley, unpublished).

At this stage, the patients had almost no naming difficulties in the naming task of the WAB, the ATTM or ATNR naming test. They pronounced an approximation of each target word in the naming tests. Furthermore, they were able to write the words correctly. These findings suggest that their disturbance was not a naming (word finding) difficulty but an articulatory disturbance.

The articulatory characteristics of apraxia of speech are still controversial in many respects (Wertz *et al.*, 1984). Almost all phonetic studies of apraxia of speech have been based on cases with apraxia of speech associated with aphasia (Shankweiler and Harris, 1966; Johns and Darley, 1970; Trost and Canter, 1974; LaPointe and Johns, 1975). The discrepancies among these findings had probably been caused by differences both in severity and in the type of aphasia accompanying apraxia of speech. There are a few accounts (Sasanuma, 1971; Lecours and Lhermitte, 1976; Square *et al.*, 1981) of phonetically studied cases with apraxia of speech not complicated by aphasia, but these have been preliminary reports and mutually contradictory. More detailed articulation studies of apraxia of speech uncontaminated with aphasia are necessary to clarify the details of the syndrome of apraxia of speech. Since our cases had apraxia of speech not accompanied by aphasia, it is worthwhile to describe their articulatory characteristics.

Articulatory characteristics on auditory phonetic analysis

Although the side of the lesion and the handedness of the 2 cases were different, their articulatory characteristics were almost the same. The systematic phonetic analysis of speech production in the 2 patients several months after onset, when they exhibited a typical apraxia of speech, showed the following 15 common characteristics. (1) Errors occurred for almost all phonemes. There was only one phoneme (/p/ in Case 2) which were always correct among the phonemes tested more than 4 times. (2) Two types of inconsistency were observed in their errors: a phoneme was incorrect at one time but correctly articulated at another; errors for the same phoneme differed from time to time. These two types of inconsistency have been emphasized as an important characteristic of apraxia of speech in contrast to dysarthria arising from damage to the bilateral corticobulbar tracts, the cerebellum or the lower motor neuron, where errors are consistent. However, there was one exception (/g/ in Case 2) to the first type of inconsistency. On the other hand, there was no exception to the second type of inconsistency among the phonemes tested more than 3 times. Even the phoneme /g/ in Case 1 showed the second type of inconsistency.

The following three conditions (3, 4, 5) elicited more errors. (3) Errors occurred more frequently on consonants than on vowels. (4) The most difficult consonants were affricates and fricatives. More substitution, distortion and omission errors were observed on consonants than on vowels. (5) More errors were made for initial sounds than for noninitial sounds, even when repetitions and retrials were excluded.

(6) The tendency in error types was for substitution errors for consonants to be more frequent than omission, distortion or addition errors, both in naming and in oral repetition of words. However, in the single-syllable repetition of Case 1, omission errors exceeded substitution errors.

The following five characteristics (7, 8, 9, 10, 11) on their substitution errors were revealed. (7) There was a tendency to substitute 1 of the 2 consonants /t, tʃ/ for other sounds. This characteristic is quite important for speculation on the nature of apraxia of speech. It suggests that the patients had difficulty in preventing the tip of the tongue from rising up to the alveolar and postalveolar region. Therefore apraxia of speech may involve problems with inhibition of tongue activity. (8) Consonants were seldom substituted for vowels, or vice versa (9) Over half the substitution error sounds were different from the target by one phonetic dimension, and over 90% of the errors were limited to one or two dimensions, using the scale of Trost and Canter (1974). The two characteristics (8) and (9) indicate that the substituted sounds were relatively close to the target sounds. (10) Substitution errors were most likely to involve errors of place, followed in order by errors of manner, voicing and the oral-nasal distinction. (11) Voiceless-for-voiced substitutions were more frequent than voiced-for-voiceless substitution.

The sequential errors and self-corrections, although they were often discussed in the previous literature, were relatively small in number ((12) and (13)). (12) The

percentage of sequential errors — that is, anticipatory, perseveratory and metathetic errors — relative to the total number of errors was low (less than 10%). (13) The number of self-corrections was small in our 2 patients (less than 2%). Some researchers (e.g., Darley, 1982; Wertz *et al.*, 1984) have suggested self-correction as one of the main characteristics of 'apraxia of speech'. However, when they knew that they had made some errors anyway, our 2 patients gave up self-correction and chose to continue their utterances of communicative efficiency without self-correction.

(14) Spontaneous speech (naming) accuracy was not better than oral repetition accuracy. (15) No significant differences in accuracy were observed between the first, second and third response either in the naming or in the oral repetition mode. This characteristic was observed both for initial sounds and for noninitial sounds.

There were a few differences in the disturbance between our 2 cases: omission errors were observed more in Case 1 than in Case 2 (Fisher's exact test, $P < 0.01$); addition errors were more in Case 2 than in Case 1 (Fisher's exact test, $P < 0.01$). Further studies are needed to establish whether these differences are due to differences in their lesions or individual factors.

As well as at the later stage described above, at the very early stage when their articulation was limited to 6 to 9 different sounds, substitution, omission and distortion errors were observed although they were small in number. Phonemes which occurred more than twice were articulated incorrectly at one time but correctly at others. The nature of the errors seems very similar to those of the later stage.

Omission errors

Our electropalatographic results revealed that what were categorized auditorily as omission errors could be classified into three types: omission errors with no palatolingual contact (true omissions); omission errors with incorrect palatolingual contact (palatolingual contact exists but it is for different sounds or for undifferentiated sounds which therefore would be a substitution error or a distortion error with air flow); and omission errors with correct palatolingual contact (palatolingual contact for the target sound exists and would be realized as the correct sound with air flow). Almost all omission errors with correct or incorrect contact were observed for initial consonants, which is important for inferring the causes of this type of omission error as will be discussed below.

Darley (1982) considered omission errors as errors of simplification and thus not characteristic of apraxia of speech. However, our results from electropalatography go against this hypothesis by revealing the existence of omission errors with incorrect contact, which are thus not errors of simplification.

The omission errors with correct or incorrect contact were probably caused by a delay in air flow for the following three reasons. First, they had palatolingual contact. Secondly, almost all omission errors with correct or incorrect contact occurred at the beginning of utterances, that is, for initial consonants. Thirdly, the

distortion errors in our patients included partial omission errors, where a weak and short sound was perceived. These were probably due to a slight delay in air flow.

A delay in air flow also may explain why vowels elicited less omission errors than consonants (*see* Characteristic 3). Air flow almost always starts during a consonant before a vowel. Consequently a delay in air flow occurred less frequently during a vowel.

Apraxia

Is the syndrome 'apraxia of speech' really apraxia? Apraxia was defined by Liepmann (1900) as follows: 'Apraxia is, in short, the inability to act, i.e., to move the moveable parts of the body as intended, though motility is preserved. The notion of falsity, unfitness for the purpose, is conveyed by the word "apraxia"'. The motility of the articulatory apparatus of our 2 patients was preserved, since they had neither significant paresis, ataxia nor involuntary movement, and since they articulated correctly target sounds at one time but not at others. Their articulatory errors — such as substitution, omissions, distortions and addition errors — were not their intended responses, which was verified by the fact that they were able to write the target words correctly. Therefore the speech disturbance of our 2 cases seems to be apraxia. The term 'apraxia of speech' can be employed for 'pure' apraxia of speech such as that shown by our cases but not for apraxia of speech complicated with aphasia, where the intention of patients cannot be checked in writing (Sugishita, 1981).

Liepmann (1920) classified apraxia into three types: limb-kinetic, ideomotor and ideational apraxia. The apraxias concern limbs and face movements but do not concern articulatory movements. Though Liepmann mentioned that speech disturbance of expressive aphasia (probably apraxia of speech plus aphasia) was apraxia, he did not specify into which type of apraxia the speech disturbance was classified. Liepmann (1920) described substitution errors of the limbs as errors of ideomotor apraxia, and omission errors as those of ideational apraxia. Limb-kinetic apraxia, defined as a loss of skill in learned movements and inability to form the beginning of movements, may be applicable to distortion errors and null responses. Our patients often had a substitution error on a phoneme but had a distortion, omission or addition error on the same phoneme at other times. Consequently, none of the three types of apraxia seems to fit apraxia of speech.

Many studies (Nathan, 1947; Denny-Brown, 1965; Tonkonogy and Goodglass, 1985) observed that apraxia of speech and oral apraxia coexist, while there were a few cases with apraxia of speech and no oral apraxia (Lebrun *et al.*, 1973). Denny-Brown (1965) observed that there were certain tasks, such as cough or whistle, which are very sensitive to even mild oral apraxia. Both of our 2 patients showed a slight oral apraxia at the early stage: they could not cough and click their tongue. The results suggest that apraxia of speech and oral apraxia coexist early after onset.

The reported cases with apraxia of speech having no oral apraxia had probably been tested without the sensitive tasks.

Localization of the lesion causing apraxia of speech

Lecours and Lhermitte (1976) have indicated that a corticosubcortical lesion in the lower part of the left precentral gyrus causes pure anarthria (apraxia of speech) in the right-handed. Tonkonogy and Goodglass (1981) have argued that a lesion in the lower parts of the precentral and postcentral gyri causes aphemia (apraxia of speech) in the right-handed. These hypotheses are consistent with the lesion of our second patient who was right-handed, although his lesion extended deeply into the white matter underlying the precentral and postcentral gyri, making his syndrome severe.

A left-handed patient with apraxia of speech has not been reported as far as we know. It would be interesting to know in which hemisphere the lesion in such a patient is sited and whether it is focal or diffuse. Most left-handed aphasics have left hemisphere damage, the remainder having right hemisphere damage (Hécaen and Sauguet, 1971). The first case in this study, who was a nonfamilial left-hander, had a focal lesion in the right hemisphere, namely, a corticosubcortical lesion mainly affecting the lower part of the right precentral gyrus and its underlying deep white matter. This finding suggests that, in some left-handers, speech is unilaterally and focally represented in a manner opposite to that of most right-handers, that is, in the right hemisphere. Our first case is therefore not consistent with the hypothesis of Hécaen *et al.* (1981) that, for left-handers, speech is represented in the left hemisphere or bilaterally. Our finding suggests that, in non-familial left-handers, speech is unilaterally and focally represented in the right hemisphere, as proposed by Delis *et al.* (1983).

Our results indicate two possible components of apraxia of speech: one is a delay in air flow, and the other is difficulty in the inhibition of tongue activity. Furthermore, both of these do not always occur, and this inconsistency could be a common characteristic of these disturbances. To clarify the nature of apraxia of speech, further studies of apraxia of speech uncomplicated by aphasia as in our cases will be indispensable.

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REFERENCES

- BASTIAN HC (1898) *A Treatise on Aphasia and Other Speech Defects*. London: H. K. Lewis.
 BROCA P (1861) Remarques sur le siège de la faculté du langage articulé suivies d'une observation d'aphémie. *Bulletins de la Société Anatomique de Paris*, **36**, 330–357.

- DARLEY FL (1982) *Aphasia*. Philadelphia: W. B. Saunders.
- DEJERINE J (1914) *Sémiologie des Affections du Système Nerveux*. Paris: Masson.
- DELIS DC, KNIGHT RT, SIMPSON G (1983) Reversed hemispheric organization in a left-hander. *Neuropsychologia*, **21**, 13–24.
- DENNY-BROWN D (1965) Physiological aspects of disturbances of speech. *Australian Journal of Experimental Biology and Medical Science*, **43**, 455–474.
- DUFOUR M (1895) Hemiplegie droite. Ancienne aphasie motrice pure ayant disparu au bout de deux ans. Tuberculose pulmonaire et renale. *Bulletins de la Société Anatomique de Paris*, **70**, 492–495.
- HÉCAEN H, DE AGOSTINI M, MONZON-MONTES A (1981) Cerebral organization in left-handers. *Brain and Language*, **12**, 261–284.
- HÉCAEN H, SAUGUET J (1971) Cerebral dominance in left-handed subjects. *Cortex*, **7**, 19–48.
- JOHNS DF, DARLEY FL (1970) Phonemic variability in apraxia of speech. *Journal of Speech and Hearing Research*, **13**, 556–583.
- KERTESZ A (1982) *The Western Aphasia Battery*. New York: Grune and Stratton.
- LADAME P-L, MONAKOW C VON (1908) Observation d'aphémie pure (anarthrie corticale). *Encéphale*, **3**, 195–228.
- LAPOINTE LL, JOHNS DF (1975) Some phonemic characteristics in apraxia of speech. *Journal of Communication Disorders*, **8**, 259–269.
- LEBRUN Y, BUYSENS E, HENNEAUX J (1973) Phonetic aspects of anarthria. *Cortex*, **9**, 126–135.
- LECOURS AR, LHERMITTE F (1976) The 'pure form' of the phonetic disintegration syndrome (pure anarthria): anatomo-clinical report of a historical case. *Brain and Language*, **3**, 88–113.
- LEIPMANN H (1900) Das Krankheitsbild der Apraxie ('motorischen Asymbolie') auf Grund eines Falles von einseitiger Apraxie. *Monatschrift für Psychiatrie und Neurologie*, **8**, 15–44, 102–132, 182–197.
- LIEPMANN H (1920) Apraxie. *Ergebnisse der Gesamten Medizin*, **1**, 516–543.
- MARIE P (1906) Revision de la question de l'aphasie: la troisième circonvolution frontale gauche ne joue aucun rôle spécial dans la fonction du langage. *Semaine Médicale*, **26**, 241–247.
- NATHAN PW (1947) Facial apraxia and apraxic dysarthria. *Brain*, **70**, 449–478.
- OLDFIELD RC (1971) The assessment and analysis of handedness: the Edinburgh Inventory. *Neuropsychologia*, **9**, 97–113.
- SASANUMA S (1971) Speech characteristics of a patient with apraxia of speech: a preliminary case report. *Annual Bulletin, Research Institute of Logopedics and Phoniatrics, University of Tokyo*, **5**, 85–89.
- SHANKWEILER D, HARRIS KS (1966) An experimental approach to the problem of articulation in aphasia. *Cortex*, **2**, 277–292.
- SOUQUES A (1928) Quelques cas d'anarthrie de Pierre Marie. *Revue Neurologique*, **2**, 319–368.
- SQUARE PA, DARLEY FL, SOMMERS RK (1981) An analysis of the productive errors made by pure apractic speakers with differing loci of lesions. In: *Clinical Aphasiology: Proceedings of the 11th Conference on Clinical Aphasiology*. Edited by R. H. Brookshire. Minneapolis: BRK Publishers, pp. 245–250.
- SUGISHITA M (1981) Apraxia of speech, aphemia and anarthria (in Japanese). *Higher Brain Function Research*, **1**, 107–111.
- SUGISHITA M, TAGAWA K, NAGATA K, KAGA K, MAKISHITA H, MOTOMURA M, ARIGA K, ENDOU K, KABE S, KAMEWADA F, KAWABATA S, KAWAMURA M, KONNO K, SEKI K, SOMA Y, HASEGAWA K, FUKADA T, YAMAZAKI K. (1986) *The Western Aphasia Battery* (Japanese edition). Tokyo: Igaku-Shoin.
- TONKONOGY J, GOODGLASS H (1981) Language function, foot of the third frontal gyrus, and rolandic operculum. *Archives of Neurology, Chicago*, **38**, 486–490.
- TROST JE, CANTER GJ (1974) Apraxia of speech in patients with Broca's aphasia: a study of phoneme production accuracy and error patterns. *Brain and Language*, **1**, 63–79.

- WERNICKE C (1906) Der aphasische Symptomencomplex. In *Die Deutsche Klinik VI (I)*. Edited by E. von V. Leyden and F. Kemperer. Berlin-Wien: Urban und Schwarzenberg, pp. 487–556.
- WERTZ RT, LAPOINTE LL, ROSENBEK JC (1984) *Apraxia of Speech in Adults: The Disorder and Its Management*. Orlando, FL and London: Grune and Stratton, pp. 52–69.

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