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VOICE ONSET TIME CHARACTERISTICS OF APRAXIA OF SPEECH PART II

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Our previous study (Itoh et al., 1979) examining the voice onset time (VOT) in the speech of four apraxic patients revealed that the VOTs of these patients differed markedly from those of the normal control subjects. We interpreted the result to indicate that the control over the timing of laryngeal and supra-laryngeal articulatory events was disturbed in this subject group.

However, two of the four apraxic patients were within six months after onset of their illness. Clinical observations suggest that these patients with relatively "recent" onset tend to show marked improvement of speech as time goes by. Accordingly, it is expected that these "recent" apraxic patients may demonstrate some changes in their VOT distribution patterns over a certain period of time. Thus, the present study was undertaken to examine whether abnormal VOT distribution patterns of these apraxic patients would change with the lapse of time (Experiment 1).

In addition, VOT data were also obtained from fluent aphasic patients in order to compare their performance with that of apraxic patients, and to elucidate the nature and underlying mechanism of nonfluent speech in apraxia of speech (Experiment 2).

1. Experiment 1: Longitudinal observation of VOTs of apraxic patients

1-1 Method

The subjects were the four apraxic patients mentioned above and two young adult normals. All were male and were the same subjects examined in our previous study.

We obtained VOT data twice from each subject over an interval of more than four months. Table 1 summarizes the post onset time at the first and the second measurements of VOT for each of the four apraxic patients, as well as the age and interval between the two measurements of VOT for all subjects.

The procedure of gathering and analyzing data was the same as used in our previous study. That is, each subject read a randomized list of the Japanese monosyllables /de/, /te/, /ge/ and /ke/. The list contained 25 occurrences of each syllable, for a total of 100 test items. The subjects were instructed to indicate when they misread and noticed the error. In such cases, the subject was asked to re-read the misread syllables.

Wide-band spectrograms of the 25 productions of each syllable were

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Table 1. The post onset time at the first and second measurements of VOT for apraxic subjects, and the age and interval between the two measurements for all subjects.

	Subject	Age (ys)	Post Onset Time 1st measurement	2nd measurement	Interval between 1st and 2nd Measurements
APRAXIC	C1	61	6 years, 7 months	9 years, 7 months	3 years
	C2	66	4 years, 11 months	7 years, 4 months	2 years, 5 months
	C3	45	6 months	3 years, 3 months	2 years, 9 months
	C4	40	2 months	6 months	4 months
NORMAL	A1	32	——	——	3 years, 1 month
	A2	28	——	——	3 years, 2 months

made from the recordings. The VOT was measured as the interval between the first vertical striation representing the vocal fold vibration and the onset of energy "burst" representing the release of an articulatory constriction. VOT measurements obtained from the spectrograms of each syllable were grouped into 10 msec intervals, and frequency distributions were plotted for each subject.

1-2 Results

Figure 1 presents the VOT production distributions of the normal subjects for /de/ and /te/ (left), and /ge/ and /ke/ (right). The abscissa represents the VOT values in msec and the ordinate represents the total number of responses. The black columns indicate the VOT values for the voiced targets /de/ and /ge/, and the blank columns indicate those for the voiceless targets /te/ and /ke/. "1st" means the results of the first measurement and "2nd" means those of the second measurement.

As Figure 1 indicates, the VOT distribution patterns of the first and the second measurements are similar for Subjects A1 and A2. Namely, in the case of A1, it is noted that the VOT values associated with the voiced stops are dispersed over a wide range but mainly in the areas of voicing

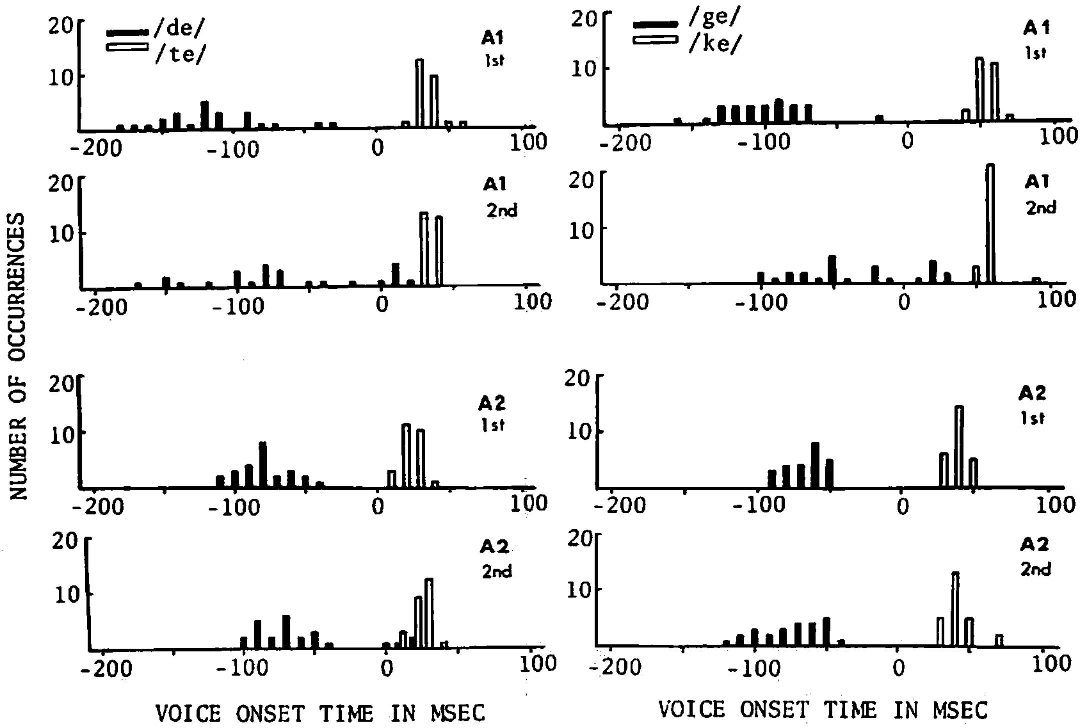


Fig. 1 VOT distributions of the two young adult normals at the first and second measurements for /de/ and /te/ (left), and /ge/ and /ke/ (right).

lead (i. e., glottal pulsing precedes the burst) at the first as well as the second measurements. On the other hand, the voiceless productions are distributed into relatively narrow areas of voicing lag (i. e., glottal pulsing follows the burst) on the two occasions of measurement.

The VOT distribution patterns of Subject A2 for /ge/ and /ke/ are about the same on both occasions of measurement. However, there is a minor overlap of the VOT values between /de/ and /te/ on the second measurement, i. e., some of the productions of /de/ lie in the voicing lag category, as can be seen from Figure 1.

As a whole, however, the normal subjects' voiced and voiceless productions are distributed into relatively discrete areas of VOT and this tendency is maintained across the two occasions of measurement.

Figures 2 and 3 compare the distribution patterns of the VOT values of the first and the second measurements for each of the four apraxic subjects for /de/ and /te/, and /ge/ and /ke/, respectively.

As can be seen from Figure 2, Subjects C1 and C2's VOT distribution patterns for /de/ and /te/ are about the same on both measurements. The VOT productions of these subjects exhibit considerable overlaps between the two phonetic categories, and these abnormal VOT distribution patterns do not change with the passage of time.

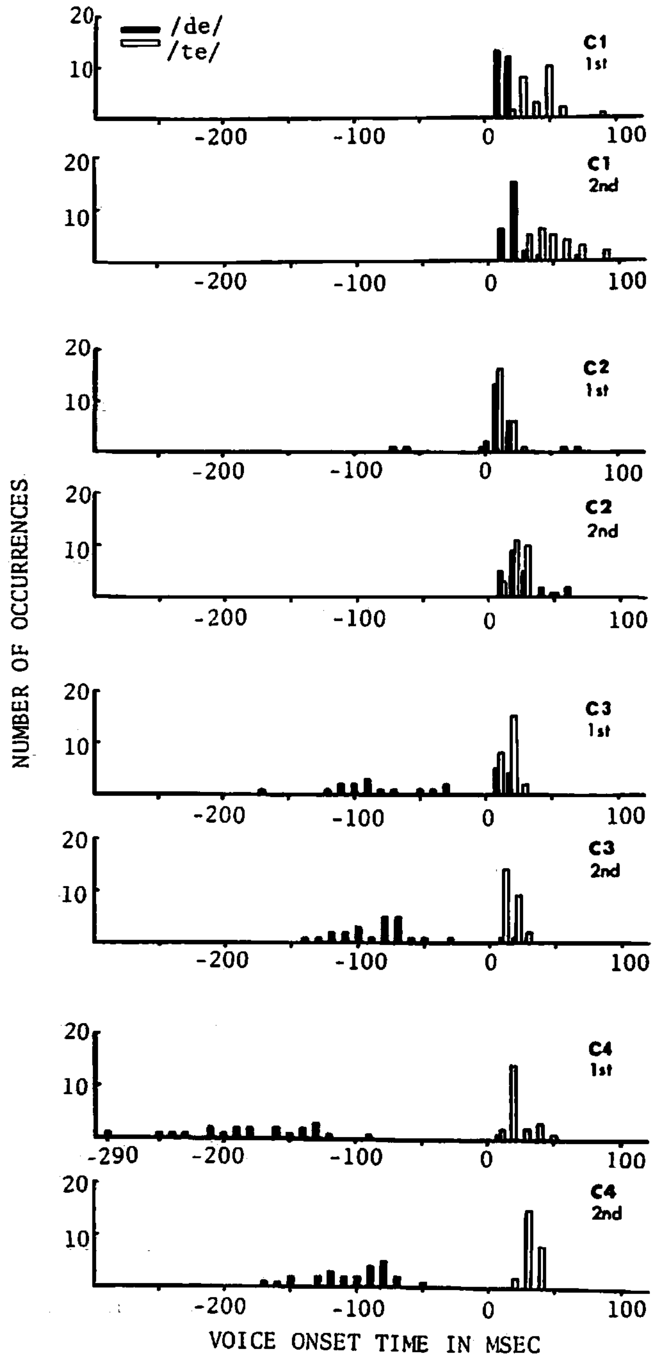
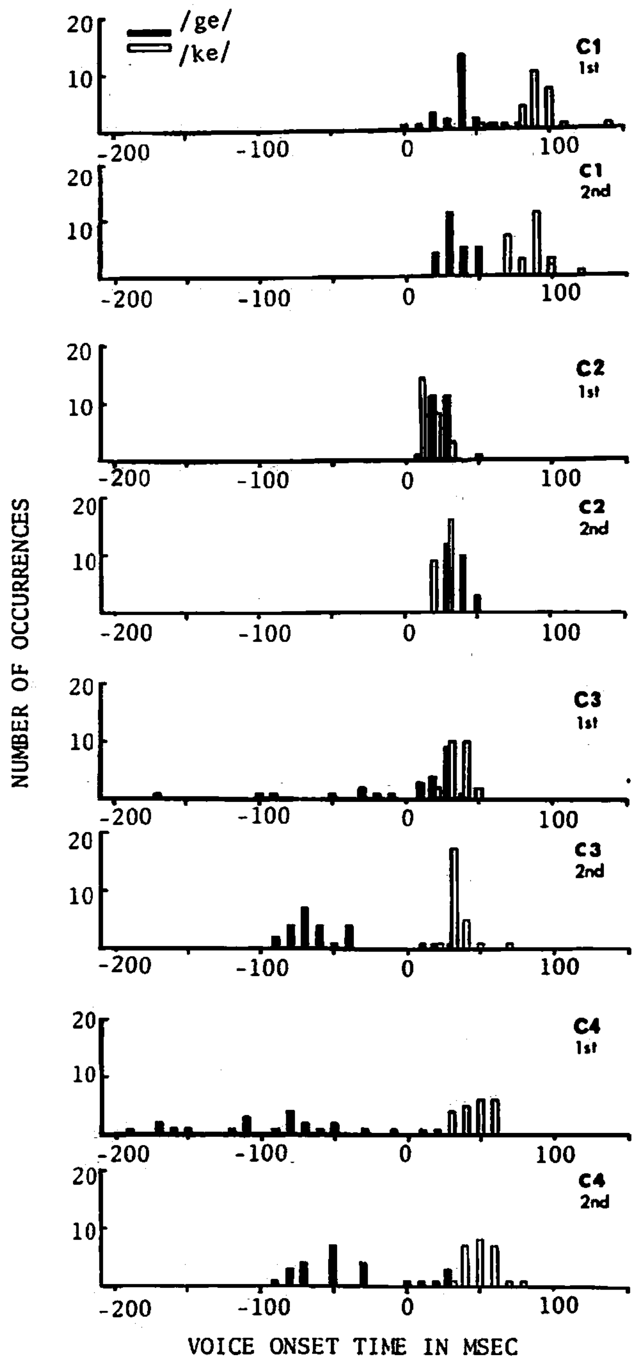


Fig. 2 VOT distributions of the four apraxic subjects at the first and second measurements for /de/ and /te/.

Fig. 3 VOT distributions of the four apraxic subjects at the first and second measurements for /ge/ and /ke/.



In contrast, Subjects C3 and C4 show a marked change in terms of VOT distribution patterns between the first and the second measurements. In the case of Subject C3, there is a considerable overlap between the two phonetic categories at the first measurement, but such an overlap decreases considerably at the second measurement. Subject C4's productions of /de/ at the first measurement are characterized by abnormally long voicing lead, but such abnormal VOT values of /de/ are not observed at the second measurement. Similar results can be seen from Figure 3 for /ge/ and /ke/.

In summary, the results of the first experiment demonstrated that two of the four apraxic patients showed a marked change in terms of VOT distribution patterns with the lapse of time, while the rest of the apraxic patients and two normal young adult subjects maintained their VOT distribution patterns across the two measurements.

2. Experiment 2: VOTs in fluent aphasic patients

2-1 Method

We obtained VOT data from six fluent aphasic patients of the Tokyo Metropolitan Geriatric Hospital. All were male and between the ages of 49 and 66.

Table 2 summarizes the age, type of aphasia, cause of brain damage, site of lesion and post onset time for the six fluent aphasic patients.

As the table indicates, all subjects have site of lesion at the temporal and/or parietal lobes of the left hemisphere.

The procedure of gathering and analyzing data was the same as used in the first experiment.

2-2 Results

Figure 4 presents individual VOT distribution patterns of each of the fluent aphasic subjects for /de/ and /te/ (left), and /ge/ and /ke/ (right).

It can be seen from the figure that the discreteness of the VOT distribution patterns between the voiced and voiceless cognates sharing the same place of articulation is clearly maintained in all six subjects, although there are minor overlaps of VOT value distributions between the two phonetic categories in the productions of Subjects D3, D5 and D6. It is also evident that there is no obvious difference concerning the VOT distribution patterns between Wernicke's aphasic subjects (D1, D2, D3 and D4) and conduction aphasic subjects (D5 and D6).

Figure 5 presents the pooled data of the normal young adult group, the normal aged group, the apraxic group and the fluent aphasic group in the form of accumulated frequency distributions of the VOT values for /de/ and /te/ (left), and /ge/ and /ke/ (right). The descriptive statistics for the VOT distributions associated with productions of /de/, /ge/, /te/ and /ke/ for each subject group are included in Table 3. The data of the normal young adult group, the normal aged group and the apraxic group were reproduced from Itoh et al. (1979).

Table 2. The age, type of aphasia, cause of brain damage, site of lesion and post onset time for the six fluent aphasic patients.

Subject	Age (ys)	Type of Aphasia	Cause of Brain Damage	Site of Lesion*	Post Onset Time
D1	50	Wernicke's	CVA	Temporal lobe of the left hemisphere	3 months
D2	50	Wernicke's	CVA	Temporal lobe of the left hemisphere and the internal capsule of the right hemisphere	11 months
D3	50	Wernicke's	Operational removal of ruptured aneurysm	Posterior region of the temporal lobe of the left hemisphere	1 year, 6 months
D4	66	Wernicke's	Operational removal of hematoma	Superior temporal gyrus, optic radiation, angular gyrus and corona radiata of the left hemisphere	2 years, 11 months
D5	65	Conduction	CVA	Temporal and parietal lobes of the left hemisphere	2 years, 3 months
D6	49	Conduction	Operational removal of hematoma	Temporal and parietal lobes of the left hemisphere	4 years, 6 months

* Identified by CT scan

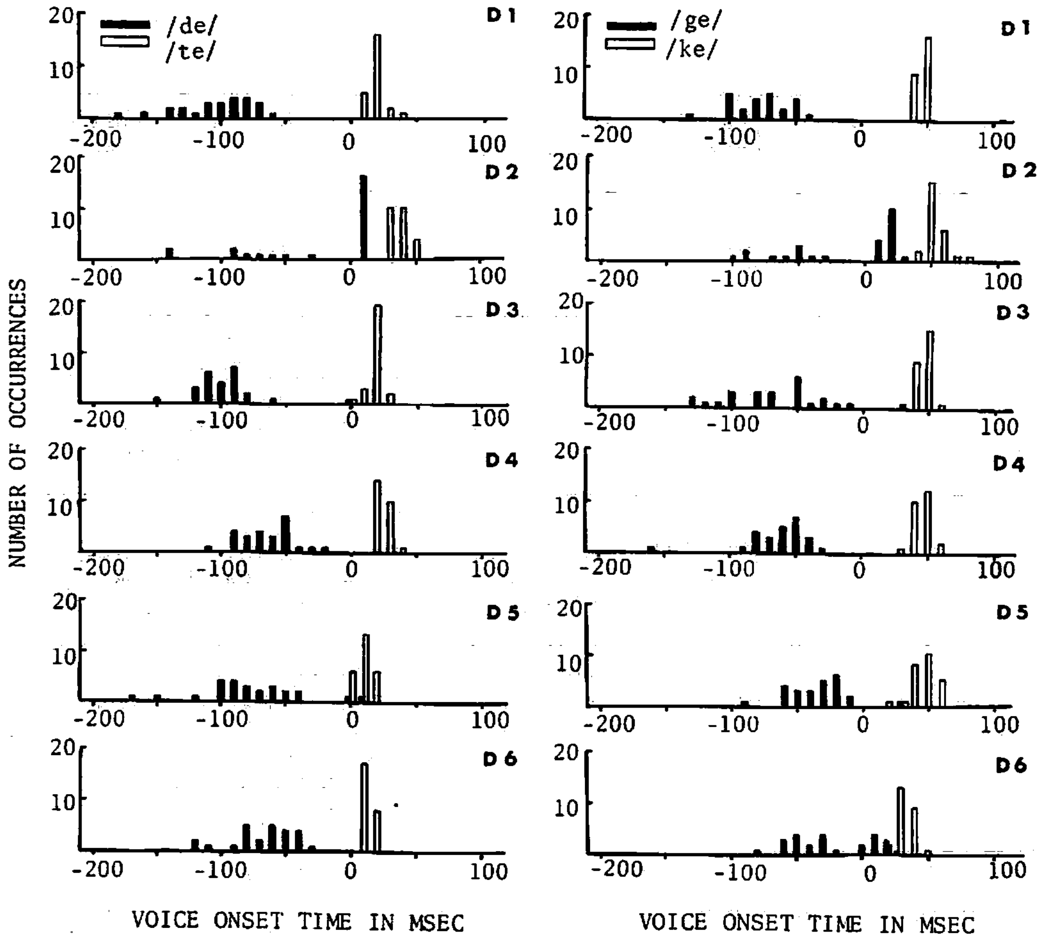


Fig. 4 VOT distributions of the six fluent aphasic subjects for /de/ and /te/ (left), and /ge/ and /ke/ (right).

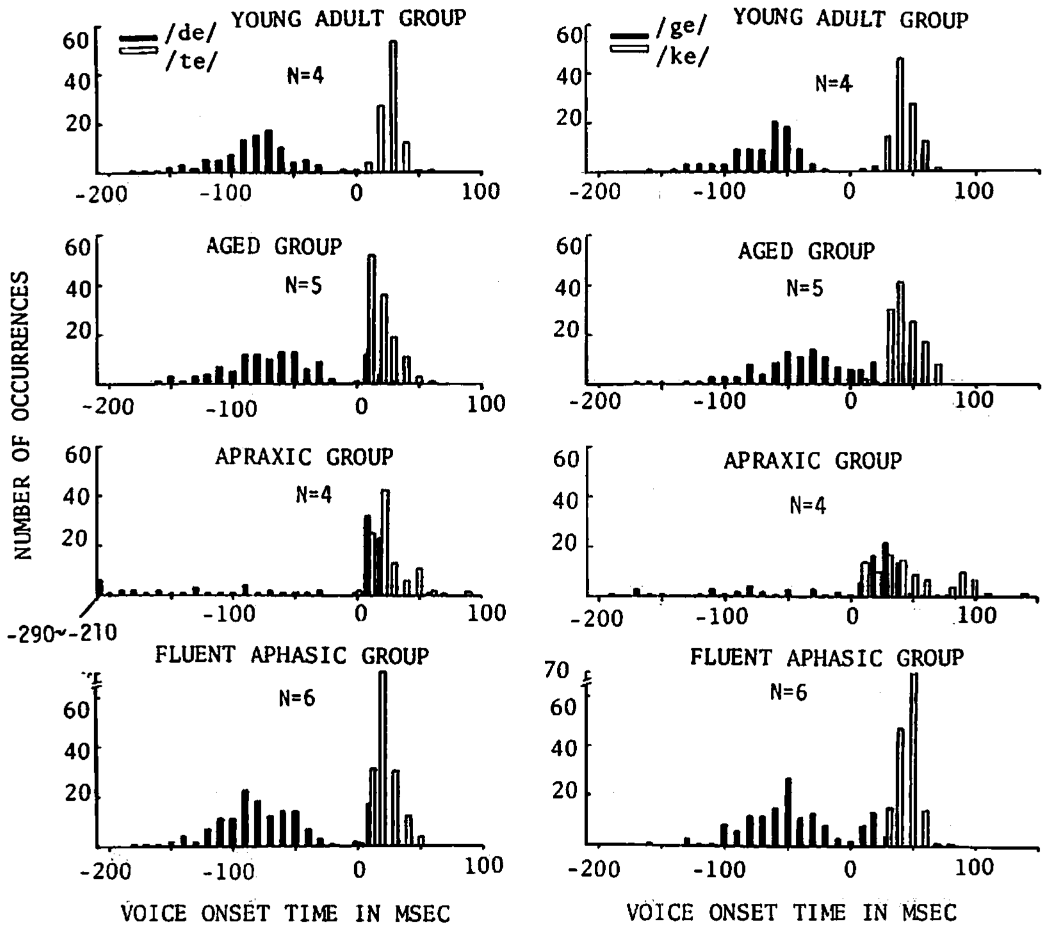


Fig. 5 VOT distributions for four subject groups for /de/ and /te/ (left), and /ge/ and /ke/ (right).

Table 3. Mode, minimum, maximum and range of VOT distributions for four subject groups.

Subject Group	Syllable	Mode	Minimum	Maximum	Range
Young Adult	de	-70	-180	0	180
N = 4	ge	-60	-160	20	180
(25-32ys.)	te	30	10	60	50
	ke	40	30	70	40
Aged	de	-55	-160	20	180
N = 5	ge	-30	-170	30	200
(66-72ys.)	te	10	10	60	50
	ke	40	10	70	60
Apraxic	de	10	-290	70	360
N = 4	ge	30	-190	80	270
(40-66ys.)	te	20	0	90	90
	ke	30	10	140	130
Fluent Aphasic	de	-90	-180	10	190
N = 6	ge	-50	-160	30	190
(49-66ys.)	te	20	0	50	50
	ke	50	20	80	60

(in msec)

As Figure 5 and Table 3 indicate, the VOT distribution patterns of the fluent aphasic group are quite similar to those of the normal subject groups (especially the aged group) but are in sharp contrast with those of the apraxic group.

3. Discussion

The results of the first experiment clearly demonstrate that the VOT distribution patterns of the normal subjects remained essentially unchanged when the VOTs were measured twice with a certain time interval in between.

On the other hand, two of the four apraxic patients showed a marked change in terms of VOT distribution patterns with the lapse of time.

A comparison among the four apraxic patients revealed that Subjects C3 and C4 whose VOT distribution patterns changed markedly between the two measurements were quite different in several respects from C1 and C2 whose VOT distribution patterns remained unchanged on the two measurements.

First, C3 and C4 were relatively young in age (both were in their forties) and had relatively "recent" onset at the first measurement (six and two months, respectively). Furthermore, clinical observations indicated that C3 and C4 showed marked improvement of articulatory ability over the period during which the two VOT measurements were undertaken. On the other hand, C1 and C2 were older in age (both were in their sixties) and had a "long standing" apraxic impairment which stayed unchanged clinically over the period during which the two VOT measurements were undertaken.

There have been ample clinical observations indicating that improvement takes place much more frequently in younger patients in the early stage of the post onset period than in older patients in the later stage of the post onset period. The results of the first experiment confirm these observations and suggest that the disturbed timing control of the laryngeal and supralaryngeal articulatory adjustments, which is considered to be responsible for inconsistent voicing errors, <u>may</u> improve in the case of younger apraxic patients with recent onset.

In addition, it is also noticeable that the VOT distribution patterns of C3 and C4 differed markedly from those of C1 and C2 (see Figures 2 and 3): that is, C3 and C4 showed voicing lead for voiced stops across the two measurements just as the normal subjects did, while C1 and C2 showed hardly any voicing lead for voiced stops. As described in our previous report, C1 and C2 can be considered to have exhibited a relatively "pure" form of apraxia of speech, while C3 and C4 can be considered to have had mild aphasia in addition to apraxia of speech. Therefore, it may well be that the difference in the VOT distribution patterns between the two groups of apraxic subjects reflects the difference in the nature or underlying mechanism of their speech defects. However, the particular distributions obtained from them may reflect a sampling artifact. Further observation of a larger number of patients is certainly in order to clarify this matter.

Analysis of the data obtained from the second experiment indicated that the VOTs of the fluent aphasics were similar to those of the normal subjects. That is to say, their voiced and voiceless productions were distributed into relatively discrete areas of VOT although there were sporadic occurrences of overlap between the VOT distributions for the two phonetic categories. This result can be interpreted to indicate that the timing relation between the articulatory release and the onset of the vocal fold vibration for the voiced and voiceless cognates is well under control in the fluent aphasics, as in the normals. It would seem then that speech sound errors exhibited by fluent aphasic patients are not attributable to any impairment at the level of articulatory programming at least along the VOT dimension, but attributable to an impairment at another level of speech production process such as the level of phonological processing (phoneme selection or retrieval).

Acknowledgment

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