ORAL APRAXIA AND APHASIA

Toshiko S. Watamori*, Motonobu Itoh*, Yoko Fukusako**
and Sumiko Sasanuma***

Introduction

Oral apraxia has been known since the time of Jackson (1878), who described a patient who was unable to protrude his tongue on command, although was able to move it spontaneously to lick his lips after drinking.

Since then, oral apraxia has been described by various authors (Nathan, 1947; Bay, 1957; Alajouanine and Lhermitte, 1960; Luria, 1970). According to Bay (1957), oral apraxia is constantly present in aphasics with cortical dysarthria. On the other hand, Alajouanine and Lhermitte (1960) state that it may also be found in Wernicke's aphasia.

Systematic investigation of oral apraxia¹⁾, however, has begun only recently. Two main issues of interest for the researchers have been 1) the relative incidence of oral apraxia among different types of aphasia; and 2) the presence of qualitatively different patterns of oral apraxia corresponding to different lesions and/or types of aphasia.

De Renzi, Pieczuro and Vignolo (1966) investigated the incidence of oral apraxia among different types of aphasia, finding that oral apraxia was highly associated with Broca's aphasia and phonemic jargon aphasia (a subtype of Wernicke's aphasia characterized by a phonemic-articulatory disorder) followed by conduction

¹⁾ According to De Renzi et al. (1966), oral apraxia may be defined as follows: "the inability to perform voluntary movements with the muscles of the larynx, pharynx, tongue, lips and cheeks, although automatic movements of the same muscles are preserved" This condition is sometimes called "facial apraxia", "oralfacial apraxia" or "bucco-facial apraxia".

^{*} Tokyo Metropolitan Institute of Gerontology

^{**} Tokyo Metropolitan Geriatric Hospital

^{***} Yokohama National University/Tokyo Metropolitan Institute of Gerontology

aphasia but not with the classical Wernicke's aphasia. Poeck and Kerschensteiner (1975) found that imitation of oral movement was most impaired in Broca's aphasia. They found that Wernicke's aphasics also exhibited impaired oral movement, although to a lesser degree. Geschwind (1965) called attention to the common occurrence of facial apraxia (= oral apraxia) in patients with conduction aphasia. He explained this association in terms of the disconnection hypothesis: i.e., facial movements cannot be carried out in response to verbal commands due to damage of the arcuate fasciculus.

Poeck and Kerschensteiner (1975) analyzed movement errors on an oral apraxia test and found no distinctly different profiles corresponding to aphasia subgroups. On the other hand, Brown (1977) described two different forms of facial apraxia possibly attributable to different lesions – anterior lesion apraxia being more in the nature of dyspraxia (clumsy errors), and posterior lesion apraxia being more in the nature of parapraxia (substitution of target action).

Some researchers have been interested in multiple sequential oral movements since speech is a complex sequential motor act of multiple articulators. LaPointe and Wertz (1974) found an impairment of oral motor sequencing, as well as isolated oral movements, among a brain-injured group with articulatory disorders (no information was given as to the hemispheric location of the lesions).

In a more recent study, Mateer and Kimura (1977) investigated simple and multiple, unfamiliar oral movements among brain damaged patients (13 right+ and 23 left-hemisphere damaged patients), finding that only nonfluent aphasics were impaired in imitating relatively simple discrete movements, while both fluent and nonfluent aphasics showed lower scores than nonaphasic left-hemisphere damaged and right-hemisphere damaged groups on the multiple, unfamiliar oral movement tasks. Since this impairment was not explicable on the basis of visual memory or perceptual deficits, nor on the basis of lingual sensory defects, the authors suggested that deficits in reproducing nonverbal oral movements were fundamental to most aphasic impairments. They further suggested that at least two systems were operating in the motor control of speech, one involved in the production of relatively discrete oral movements and the other operating to effect the transition from one discrete movement to another in a smooth and orderly way.

anatomical areas corresponding to such schema, according to the authors, would be the anterior and posterior speech zones, respectively. Further evidence for the presence of heterogeneous oral apraxia was provided by Ohigashi, et al. (1980). They compared performance on isolated movement tasks with that on combined oral movement tasks in fluent and nonfluent aphasics and identified two heterogeneous patterns corresponding to anterior and posterior lesions.

It should be pointed out, however, that the items these authors categorized as simple or isolated oral movements included those tasks that Luria (1970) called "symbolic acts". According to Luria, "an apraxia of the speech apparatus does not affect the execution of 'postural' movements²⁾ of the lips and tongue as much as it does the execution of simple acts which are symbolic in nature but dissociated from the system of speech. 3)" While Luria considers such acts as being symbolic, they can be analyzed in another way. For example, the act of "whistling" involves several movements smoothly coordinated temporally as well as spatially; i.e., inhalation, proper positioning of the lips and tongue, releasing the breath stream while controlling the air flow and adjusting the movements of the lips and tongue. As such, Luria's "symbolic acts" demand more coordination of different muscles than In fact, Johns and LaPointe (1976) stated "postural movements". that tasks such as "cough", "clear throat", "blow" and "whistle" were the most susceptible items in their oral apraxia test possibly because they demanded the coordination of the breath stream and/or phonation with oral movements.

The results of research, thus far, have been rather inconclusive as to the appearance of oral apraxia among different types of aphasia as well as the presence of different types of oral apraxia. Thus, it seems necessary that a study of oral apraxia be undertaken under strictly controlled stimulus conditions, followed by a scrupulous analysis of patients' performances. The purpose of this investigation is threefold: First, to determine the relative incidence of oral apraxia among the different aphasia

^{2) &}quot;postural" movements: e.g. touch the upper lip with the tip of the tongue, place the tongue between the upper lip and teeth, etc.

^{3) &}quot;symbolic" acts: e.g. to produce the oral movements appropriate to spitting, kissing, whistling, clicking the tongue, etc.

subgroups with lesions identified by the CT scan. Second, to identify the different error patterns of oral apraxia associated with each subgroup of aphasia. Third, to determine the effects of task differences, i.e., "postural" (elemental) vs. "symbolic" (coordinated) tasks, on performance among the different aphasia subgroups.

Method

Subjects

The subjects were 35 aphasic patients (27 males and 8 females) with left hemisphere damage and 10 healthy persons of matched age as controls. Table 1 shows the characteristics of each patient group. Brain lesions were identified by the CT scan for all of the aphasic patients. All of the patients had unilateral left hemisphere lesions, and the localization of the lesions in each aphasia subgroup was similar to that reported in Totsuka et al., 1979. The Roken⁴ Test for the Differential Diagnosis of Aphasia (RTDDA) was used to classify the aphasic patients into each aphasia subgroup.

Table 1
Characteristics of each patient group

Group	N	Mean Age	Test Time a (months po	Etiology CVA Other		
			≤3	3<		
Broca I*	10	52.5	0	10	10	O
Broca II**	10	55.1	2	8	10	0
Conduction	5	60.4	2	3	5	0
Wernicke	10	57 .7	1	9	10	Ó

(Mean age for the patient group was 55.8 and mean age for normal Ss was 58.6)

- * Broca I: Patients have mild language impairment accompanied by mild to severe apraxia of speech, corresponding to 'aphasia with persisting disfluency' in Schuell's classification.
- ** Broca II: Patients have moderate to severe language impairment complicated by apraxia of speech, corresponding to 'aphasia with sensorimetor impairment' in Schuell's classification.

⁴⁾ Tokyo Metropolitan Institute of Gerontology

Procedure

The oral apraxia test items are given in Table 2. The subjects were instructed to make the required movements first on verbal command (verbal mode) and then on imitation (imitation mode). Conventionally, oral apraxia tests have been given solely in the imitation mode, lest a verbal comprehension deficit interfere with the results. However, since the disconnection hypothesis of apraxia crucially involves language as a stimulus condition, we felt that it was essential to test oral praxis using the two modalities i.e., verbal and imitation. The oral movements exhibited by the subjects in response to each test item was carefully described on the spot. These descriptions were then coded and scored according to the examples shown in Table 3. These codes were a modified version of the ones developed by Poeck and Kerschensteiner (1975).

Table 2
Oral Apraxia Test

- 1 tongue protrusion
- 2 tongue protrusion-retraction
- 3 put the tongue in the center of upper lip
- 4 put the tongue in the center of lower lip
- 5 lateral movement of the tongue
- 6 pucker lips

Elemental Movement

- 7 click tongue
- 8 puff out cheeks
- 9 whistle
- 10 blow
- 11 chew
- 12 cough
- 13 smile
- 14 chatter teeth

Coordinated Movement

Table 3

Coding of errors

(Example: "Move your tongue to either end of your mouth.")

P	Abbreviation	Code	Description of Behavior			
	of Code	code	bescription of behavior			
	NR	No Response	No movement was observable.			
	I	Irrelevant	No oral movement was executed, but the whole body other than the oral-facial structure became tense.			
	T	Talk instead of moving	Repeated command "Move your tongue to either end of your mouth."			
	P	Perseveration	Gestures elicited by preceding items were performed.			
134	S	Substitution of movements	Discrete oral-facial movement other than the one requested was performed. (e.g., move the eyes horizontally instead of moving the tongue)			
4	F	Fragmental movements	Some part of the requested gesture was lacking (e.g., move tongue only to one end of the mouth)			
	Α	Additional movements	Requested movements were executed with some additional oral-facial structure movements.			
	An	Additional Noise	Requested movements were executed with additional vocal overflow (e.g., move tongue while vocalizing "aQ,aQ".)			
	N	Noise instead of movement	Made noise instead of movement (e.g., vocalizes "eh,eh" without oral-facial structure movements)			
	D	Delay and Self-Correction	Requested movement was performed after some pause or unsuccessful movements.			
_	Score	Code				
	2	Correct Movement				
	1 0	D NP T T D S F A An and N				
	U	NR, I, T, P, S, F, A, An and N				

Results

Group performance on the oral apraxia test

The mean performance by the control and aphasic subgroups on the oral apraxia test in the verbal and imitation modes is shown in Table 4. All of the aphasic subgroups had more difficulty making oral movements than the control group in the verbal mode. Median tests to determine whether each aphasic subgroup differed from the control group in terms of central tendency were significant (p $\langle .01 \rangle$) for all of the aphasia subgroups. However, in the imitation mode, only the Broca II patients differed significantly from the control group.

Table 4

Group performance on the oral apraxia test in the verbal and imitation modes

		Ve	rbal Mod	đe	Im	itation	Mode
Group	N	$\overline{\mathbf{x}}$	SD	Range	$\overline{\mathbf{X}}$	SD	Range
Broca I	10	18.4	5.17	12-27	23.3	3.71	15-28
Broca II	10	11.0	6.93	2-24	18.5	7.42	8-25
Conduction	5	17.6	2.79	15-21	25.8	1.92	23-28
Wernicke	10	10.6	5.68	0-17	22.5	7.82	8-28
Normal	10	24.9	2.60	21-28	27.0	1.70	24-28
(The maximum score on this test in each mode was 28.)							

Incidence of disordered oral movement in the aphasia subgroups

Table 5 shows the number of patients exhibiting disordered oral movement in each aphasia subgroup. Disordered oral movement was defined as a score at least two standard deviations below the mean of the normal control group.

Chi square tests were performed to determine the differences between the two stimulus modes, i.e., verbal vs. imitation. The difference was significant only for the Wernicke's subgroup ($X^2 = 7.0$, p<.01). To determine the influence of auditory comprehension deficits upon the performance on the oral apraxia test, correlation coefficients were calculated between the score on the auditory processes section of the RTDDA and the oral apraxia test score in the verbal mode for each aphasia subgroup. Table 6 shows that

the correlation coefficient was significant only for the Wernicke's patients, indicating that the low performance on the oral apraxia test in the verbal mode could be accounted for in terms of an auditory comprehension deficit in this group. The number of patients showing disordered oral movement decreased to three in the imitation mode in the Wernicke's subgroup. Two of the three patients who showed disordered oral movement upon imitation were those with phonemic jargon aphasia.

Table 5
Incidence of disordered oral movement in the aphasia subgroups

No. of patients showing disordered oral movement*

Group	No. of pt.	Verbal Mode	Imitation Mode
Broca I	10	5	5
Broca II	10	8	6
Conduction	5	3	2
Wernicke	10	10	3

^{*} More than two SDs below mean of normal control subjects.

Table 6

Correlation coefficient between the score on the auditory processes section of the RTDDA and the score on the oral apraxia test in response to verbal command

Group	N	r	. significance
Broca I	10	0.20	NS
Broca II	10	0.45	NS
Conduction	5	0.42	NS
Wernicke	10	0.67	S p<0.01

Error profile and aphasia subgroup

The errors on the oral apraxia test were classified according to the coding system shown in Table 3. The proportion of each error type was calculated for each aphasia subgroup. As shown in Fig. 1, the error types differed among the different aphasia subgroups.

In the verbal mode, the normal subjects showed three peaks in their error profile - "Substitution (S)", "Additional Movements (A) and "Delay (D)". The error profiles for the Broca I and the

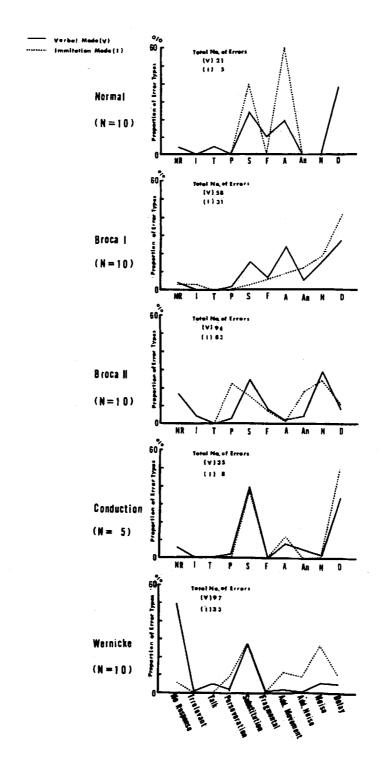


Fig. 1 Error profiles for normal and aphasic subjects. The ordinate represents the proportion of error types. The abscissa represents error types.

conduction patients resembled that of the normal Ss in this respect. However, the most distinguishing difference between the normal Ss and the Broca I and the conduction patients was the presence of "Noise" errors, i.e., "Noise (N)" and "Additional Noise (An)" in the latter two groups. Although these three groups resembled each other in terms of the presence of a high proportion of "Delay (D)" errors, the quality of the "Delay (D)" errors in the normal Ss was also somewhat different from that in the Broca I and the conduction patients. "Delay (D)" for normals includes either pauses, during which no movement was attempted, or the achievement of the required movements after a repetition or clarification of the command. "Delay (D)" for the Broca I and the conduction patients includes the achievement of the required movements after several unsuccessful movement attempts in addition to pauses. The profile for the Broca II patients had peaks at "No Response (NR)", "Substitution (S)" and "Noise (N)". The Wernicke's patients' most prevalent error type in the verbal mode was "No Response (NR)", followed by "Substitution (S)".

In the imitation mode, the normal subjects made only a few errors which belonged to either-"Substitution (S)" or "Additional Movements (A)". For the Broca I patients, while "Noise" errors increased only slightly, the proportion of "Delay (D)" errors increased considerably in this mode. For the Broca II patients, while the proportion of "No Response (NR)" errors decreased greatly, only a small decrease was noted for "Substitution (S)" and "Noise (N)" errors. On the other hand, "Perseveration (P)" and "Additional Noise (An)" increased considerably in this mode. Since the oral apraxia test was administered in a fixed order (verbal mode to imitation mode), perseverative errors were more likely in the imitation mode. Although the increase in "Perseveration (P)" in the imitation mode was also observed in the Wernicke's patients, the degree of increment was small compared to that in the Broca II patients. Thus, this feature seems to be a phenomenon unique to the Broca II patients. The conduction aphasics showed an almost identical error profile in both the verbal and imitation modes. More than 70% of the errors for the conduction aphasics were either "Substitution (S)" or "Delay (D)". In the Wernicke's patients, the proportion of "No Response (NR)" errors decreased drastically in the imitation mode. In contrast,

"Noise (N)", "Additional Noise (An)" and "Additional Movements (A)" increased. While "Noise (N)" was identified as the most distinguishing error in the Broca II patients, the Wernicke's (and also the conduction patients') "Noise (N)" errors were quite different from the ones made by the Broca's patients. The Wernicke's patients produced mainly onomatopoeic words (e.g., said "fuh, fuh" instead of blowing, or said "gohon, gohon" instead of coughing), while the Broca's patients made fragmental and often undifferentiated sounds (e.g., "paQ, paQ" instead of blowing, or "kuQ, kuQ" instead of chewing). Thus, it can be said that when model movements were demonstrated in the imitation mode, the Wernicke's patients tended to sustitute semantically associated words for the required movements.

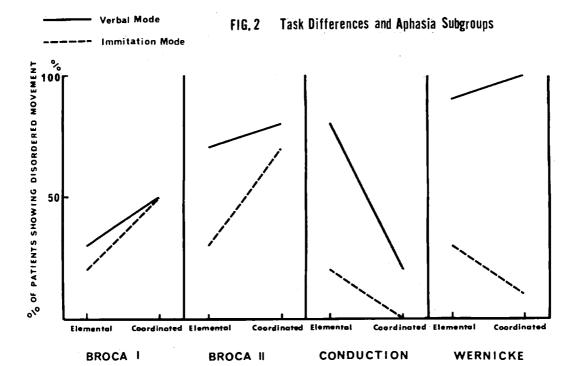
The analysis of error types seems to indicate that types of errors are important for differentiating between normals vs. aphasics, as well as among the subgroups of aphasia. Although care should be taken in generalizing these results because of the limited number of subjects in each subgroup, it is tempting to say that the nature, not the number, of the patients' errors should be examined carefully.

The relationship between elemental and coordinated movements in the different aphasia subgroups

The fourteen test items were divided into two categories: six elemental (what Luria terms "postural") and eight coordinated (what Luria terms "symbolic") movements (Table 2). The patients scoring more than two standard deviations below the mean of the normal control group in each of the two categories in the verbal and imitation modes were classified as showing disordered oral movement for that category. Fig. 2 shows the percentage of patients belonging to the different aphasia subgroups who showed disordered oral movement in either category.

Both of the Broca's aphasic groups exhibited a similar tendency, i.e., a fewer number of patients had difficulty with elemental movements in either mode compared with coordinated movements, and the discrepancy between the two categories increased in the

⁵⁾ and 6): These onomatopoeic expressions are commonly used in written Japanese.



imitation mode. In contrast to the Broca's patients, the conduction aphasics showed the reverse tendency. In the verbal mode, the discrepancy between elemental and coordinated movements was quite large. Although the discrepancy became smaller in the imitation mode, the direction of the discrepancy remained the same. The Wernicke's aphasics showed little difference between the two categories in the verbal mode. The high percentage of patients who showed disordered oral movement in this group seems to indicate a comprehension difficulty, as stated earlier. In the imitation mode, the Wernicke's patients showed the same tendency as the conduction aphasics.

Discussion

In this study, we attempted to analyze the oral movement disorder of different aphasia subgroups in relation to stimulus mode as well as the nature of the task. As was pointed out by Poeck and Kerschensteiner (1975), oral apraxia was found in all the aphasia subgroups studied, with Broca's aphasia exhibiting the highest proportion of instances of apraxia. Three Wernicke's patients showed disordered oral movement upon imitation. Two of them had phonemic jargon aphasia. This subtype of Wernicke's

aphasia has been identified as having a high incidence of oral apraxia in a previous study by De Renzi et al. (1966).

The analysis in relation to stimulus mode revealed that the two Broca groups and the conduction aphasics did not show a significant difference between the verbal and the imitation modes, although more patients exhibited oral movement disorder in the verbal mode than in the imitation mode exept in the Broca I group. On the other hand, the Wernicke's aphasics displayed clear discrepancy between the two stimulus modes - verbal and imitation. An auditory comprehension deficit was positively correlated with the score on the oral apraxia test in the verbal mode only in the Wernicke's aphasics, thus indicating an influence of auditory comprehension deficits upon the execution of movement in the verbal mode.

An analysis of the error profiles yielded distinctly different profiles corresponding to each of the four aphasic subgroups, as well as according to anterior - posterior classification of aphasia.

In the verbal mode, the Broca I patients showed an error pattern which was similar to that for normals. In the imitation mode, however, "Delay (D)"errors increased considerably, and this feature became the most prominent one in this group. The "Delay (D)" errors of this group consisted of "groping for the target movement" type response - e.g., when the patient attempted to click his tongue, he first moved his tongue aimlessly in the mouth and then touched the alveolar ridge with his tongue tip, and finally succeeded in producing a click. Such groping behavior is commonly seen in Broca I patients when they attempt to produce speech sounds. The high proportion of "Delay (D)" errors in the imitation mode shown by the Broca I patients might have stemmed from the same underlying mechanism as their articulation difficulty - i.e., a motor programming difficulty for oral movement that has possibly resulted from lesions in the left premotor cortex.

The Broca II patients showed considerable derailment from the normal profile in the verbal mode as well as in the imitation mode. This subgroup of patients showed a proportion of "No Response (NR)" errors second only to Wernicke's subgroup. Since auditory comprehension deficits were found to be unrelated to the oral apraxia test score in this group, this high proportion of "No Response (NR)"

errors could be related to a failure in the retrieval of the motor pattern for the target action and/or a more severe problem in motor programming. Another distinct feature of this group was the high proportion of "Noise (N)" and "Additional Noise (An)" errors which was observed in the verbal and the imitation modes. It is difficult to explain the underlying mechanism for such "Noise (N)" errors, surely the most distinctive characteristic of the Broca II group. In the imitation mode, "No Response (NR)" errors decreased, while "Perseveration (P)" and "Additional Noise (An)" increased. The high proportion of "Perseveration (P)" errors could be related to extensive lesions in the frontal lobe, usually encountered in this type of patient, which are larger and deeper than the lesions in Broca I patients (Totsuka, et al., 1979).

In the conduction aphasics, the errors resembled their phonological behavior in speech - "Substitution(S)" and "Delay(D)" were the significant features. In the Wernicke's aphasics, in the verbal mode, the high proportion of "No Response (NR)" errors was explicable from their auditory comprehension deficit. Other than "No Response (NR)" errors, "Substitution(S)" errors were the most prominent feature in the verbal mode for the Wernicke's patients. In the imitation mode, as "No Response (NR)" errors decreased and "Noise (N)" and "Additional Noise (An)" increased, the profile of the Wernicke's patients came closer to the verbal mode error profile of the Broca II patients. However, as stated earlier, the quality of the "Noise (N)" responses were quite different between the Broca II and the Wernicke's patients.

When the tasks were divided in terms of the nature of the movements (elemental vs. coordinated) involved, strikingly different error patterns emerged for the different aphasia subgroups. That is, while the anterior aphasics (both subgroups of the Broca's aphasics) exhibited more difficulty with coordinated movements in the verbal as well as in the imitation mode, the posterior aphasics (conduction and Wernicke's aphasics) exhibited a different pattern from that of the anterior aphasics in relation to tasks and modes. The tendency for cordinated movements to be more impaired in the anterior aphasics can be readily explained by the finding of Itoh, et al. (1979, 1980) that temporal organization among different articulators is disturbed in patients with apraxia of speech. This is also consistent with Luria's finding (1970)

that "symbolic" acts are more susceptible to apraxia of the speech apparatus, although the lesion Luria postulates for such a disorder is different. On the other hand, in the conduction aphasics in the verbal mode, coordinated movements were easier than elemental ones. Such a dissociation cannot be readily explained by the disconnection hypothesis. According to this hypothesis, the conduction aphasics cannot carry out oral movements on verbal command, since verbal input is prevented from reaching the premotor area from Wernicke's area due to a disruption of the arcuate fasciculus. If the disconnection hypothesis is correct, the conduction aphasics should demonstrate a difficulty in executing oral movements to verbal commands regardless of the nature of the task, i.e., whether coordinated movements or elemental movements. In fact, no such dissociation was seen in the Wernicke's patients, who showed difficulty comprehending verbal commands in general (Fig. 2). The dissociation between the two types of tasks in the conduction aphasics in the verbal mode could be rather explained in terms of the nature of the verbal commands themselves. For example, for the coordinated movements, commands like "Show me how you cough when you catch cold" were used. This type of command is linguistically more redundant and thus might have aided the patients' comprehension. On the other hand, the commands used for the elemental movements relied heavily on linguistic decoding ability (e.g., "Put your tongue in the center of your upper lip"). The difference in linguistic structures of these two types of commands can possibly account for the dissociation between the patient's performance of coordinated and elemental movements in response to verbal command in the conduction aphasics. For the Wernicke's patients, such linguistic redundancy did not improve their performance in the verbal mode possibly because of the severity of their auditory comprehension deficit. Though conduction aphasics are known to have good comprehension, some researchers have suggested that they seem to have a subtle receptive difficulty, such as a difficulty in grasping syntactic relationships and that their difficulty in repeating sentences could be traced back to this problem (Green and Howes, 1977). Our results for the oral apraxia test also suggest that conduction aphasics' difficulty in executing oral movements, particularly elemental movements, could be related to such a subtle difficulty in the

linguistic processing of auditory stimuli.

In the imitation mode, two posterior aphasics exhibited a similar tendency for coordinated movements to be somewhat easier than elemental ones. This tendency appears to contradict the findings of Mateer and Kimura (1977), who found that posterior aphasics were impaired in coordinating multiple oral movements. Since the tasks used in their study and ours were different, however, it would not be adequate to simply compare the two results. While Mateer and Kimura used unfamiliar, unpracticed movement sequences, we used familiar, overlearned movements. Familiarity seemed to have assisted the posterior aphasics in executing the required movements in our study. On the other hand, such familiarity did not improve the anterior aphasics' performance because their difficulty appeared to involve essentially the coordination of different articulators and/or movements.

In summary, the analysis of the error profiles and the nature of the tasks revealed different patterns for each of the four aphasia subgroups. Although the limited data restricts the generalizability of our present results, it was suggested that two qualitatively different oral apraxias were present corresponding to the two anterior aphasic subgroups (Broca I and II) and the two posterior aphasic subgroups (conduction and Wernicke's). In the anterior aphasics, the ability to perform relatively simple, isolated movements was preserved, while the ability to perform coordinated movements was disturbed. The analysis of the error profiles also suggests that the difficulty encountered by the anterior aphasics resulted from lesions involving the premotor division of the frontal lobe.

In contrast, the posterior aphasics demonstrated linguistic processing problems in the verbal mode. In the imitation mode, the errors were mostly "Substitution(S)" and, in the Wernicke's patients, "Noise (N)", which was regarded as a type of semantic substitution behavior. Furthermore, the task analysis revealed a similar error tendency in both types of posterior aphasics in the imitation mode. These results seem to support Brown's hypothesis as to the different nature of apraxia as a result of anterior and posterior lesions (dyspraxia vs. parapraxia).

Summary

- (1) The present study confirmed that oral apraxia is commonly seen in aphasic patients regardless of the type of aphasia.
- (2) However, Broca's aphasics comprised the highest percentage of orally apraxic patients.
- (3) The analysis of errors revealed different profiles corresponding to anterior (Broca I and II) and posterior (conduction and Wernicke's) aphasics. "Dyspraxia" seems to be an appropriate term to identify anterior aphasics' errors in an oral apraxia test, while "parapraxia" seems to be appropriate for posterior aphasics' errors in an oral apraxia test.
- (4) The task difference (elemental vs. coordinated) affected the patients' performance differently in the anterior and posterior aphasics.
- (5) The anterior aphasics' difficulty in executing coordinated movements seemed to stem from lesions involving the premotor region and, thus, a motor programming difficulty. The posterior aphasics' difficulty in executing oral movements in the verbal mode was in part attributed to a linguistic processing difficulty. In the imitation mode, however, both types of posterior aphasics exhibited a similar tendency contrasting with the one exhibited by the anterior aphasics.

Acknowledgement

The authors wish to express sincere appreciation to Ms. Mariko Yoshino and Ms. Mariko Fujibayashi of Toranomon Hospital, and to the staff of Speech Pathology Services of the Tokyo Metropolitan Geriatric Hospital for their assistance with data collection. We also acknowledge Ms. Shuko Murakami of the Tokyo Metropolitan Institute of Gerontology for her assistance with data collection and data analysis. Portions of this paper were presented to the annual meeting of the Shinkeishinrigakukonwakai at Kyoto, 1980.

This study was supported in part by a grant from the Adult Disease Clinic Memorial Foundation.

References

- Alajouanine, TH., and F. Lhermitte (1960); Les troubles des activités expressives du langage dans l'aphasie. Leurs relations avec les apraxies, Rev. Neurol. 102, 604-633.
- Bay, E. (1957); Die corticale Dysarthrie und ihre Beziehungen zur sogen. motorischen Aphasie. Deutsche Zeitschrift f. Nervenheilkunde, 176, 553-594.
- Brown, J.R. (1977); Mind, Brain, and Consciousness. Academic Press, New York.
- De Renzi, E., A. Pieczuro and L.A. Vignolo (1966); Oral Apraxia and Aphasia, Cortex, 2, 50-73.
- Geschwind, N. (1965); Disconnexion syndromes in animals and man, Brain, 88, 237-294, 585-644.
- Green, E. and D.H. Howes (1977); The nature of conduction aphasia: A study of anatomic and clinical features and of underlying mechanisms, in Whitaker and Whitaker (eds.), Academic Press, New York.
- Itoh, M., S. Sasanuma and T. Ushijima (1979); Velar movement during speech in a patient with apraxia of speech. Brain and Language, 7, 227-239.
- Itoh, M., S. Sasanuma, H. Hirose, H. Yoshioka and T. Ushijima (1980); Abnormal articulatory dynamics in a patient with apraxia of speech: X-ray microbeam observation. Brain and Language, 11, 66-75.
- Jackson, J.H. (1932); Remarks on non-protrusion of the tongue in some cases of aphasia (1878), in Selected Writings of J.H. Jackson, Vol. 2, 153-154, Hodder and Stoughton, London.
- Johns, D.F. and L.L. LaPointe (1976); Neurogenic disorders of output processing: Apraxia of speech. In Studies in Neurolinguistics Vol. 1, Whitaker and Whitaker (eds.), Academic Press, New York.
- LaPointe, L.L. and R.T. Wertz (1974); Oral-movement abilities and articulatory characteristics of brain-injured adults, Perceptual and Motor Skills, 39, 39-46.
- Luria, A.R. (1966); Higher Cortical Functions in Man. Basic Books, Inc., New York.
- Luria, A.R. (1970); Traumatic Aphasia, Mouton, The Hague. Mateer, C. and D. Kimura (1977); Impairment of nonverbal oral movements in aphasia, Brain and Language, 4, 262-276.
- Nathan, P.W. (1947); Facial apraxia and apraxic dysarthria, Brain, 70, 449-478.
- Ohigashi, Y., T. Hamanaka, H. Ohashi, K. Hadano, N. Kato, A. Tomita and K. Asano (1980); A propos de l'hétérogénéité de l'apraxie bucco-faciale. Folia Psychiatrica et Neurologica Japonica, 34, 35-43.
- Poeck, K. and M. Kerschensteiner (1975); Analysis of the sequential motor events in oral apraxia, in Zülch, K.J., Creutzfeldt, O. and Galbraith, G.C. (eds.), Cerebral Localization, Springer-Verlag, Berlin.
- Totsuka, G., H. Funai, M. Fujibayashi, Y. Fukusako and S. Sasanuma (1979); Differential diagnosis of aphasia and lesions in the brain. The Japan Journal of Logopedics and Phoniatrics, 20, 197-205.