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Impairment Of Nonverbal Oral Movement After Damage To The Left Cerebral Hemisphere

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IMPAIRMENT OF NONVERBAL ORAL MOVEMENT AFTER DAMAGE TO THE LEFT CEREBRAL HEMISPHERE

by

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Submitted in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy

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ABSTRACT

The defects in language seen after left hemisphere damage in man have traditionally been interpreted as verbal and/or symbolic-representational impairments. A series of studies was conducted on the relationship between oral apraxia and speech disorders which suggested that the speech impairments seen after left hemisphere damage are accompanied by impairment in the production of nonverbal oral movements.

Patients with left hemisphere damage were classified into aphasic or nonaphasic groups on the basis of standard aphasia tests. Aphasic patients were further classified as fluent or nonfluent. Tests given the patients required: (1) the imitation of relatively simple single movements of the oral musculature, of the kind used in traditional testing of oral apraxia, for example, protrusion of the tongue, blowing, etc., and (2) the imitation of three such movements in a sequence. In agreement with the literature, impairment on the single oral movements was evident in nonfluent, but not fluent, aphasics. However, fluent aphasics were impaired in the imitation of multiple nonverbal oral movements, relative to patients with right hemisphere damage and normal control subjects. It thus appears that all patients with aphasic defects also have difficulty in performing nonverbal movements of the oral musculature if the movements are sufficiently demanding.

Analysis of the errors made on the multiple oral movements task revealed that left, but not right, hemisphere damage is highly

associated with perseverative responses. Despite the salient relationship between left hemisphere damage and impairments in the production of both verbal and nonverbal oral movement, no differential effects of left or right hemisphere lesions were found on the degree or pattern of oral sensory deficits.

The results were taken to suggest that there is a primary defect of oral motor control after left hemisphere damage and that the symbolic or representational nature of the motor task is largely irrelevant to the impairment. A fundamental role of the left hemisphere and a possible basis for hemispheric specialization of speech function appears to be the control of complex movement.

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PAPER NO. 1

IMPAIRMENT OF NONVERBAL ORAL MOVEMENTS IN APHASIA

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ABSTRACT

Thirty-six patients with unilateral cerebral vascular damage were required to perform a number of oral motor tasks, both verbal and nonverbal. Nonfluent aphasics were impaired in the imitation of single oral movements, as previously reported. However, on the imitation of complex nonverbal oral movements fluent aphasics were impaired as well as nonfluents. This impairment was not explicable on the basis of visual memory or perceptual deficits, nor on the basis of sensory thresholds as measured on the tongue. The findings suggest that deficits in co-ordinating oral movements are fundamental to most aphasic impairments, the meaningfulness of the responses not being a critical factor in the appearance of the defect.

The association with aphasia of defects in the production of nonverbal movements termed "oral apraxia" has been known since the time of Hughlings Jackson (1878). He described a patient who was unable to protrude the tongue on command, although at other times spontaneous licking movements occurred. In a recent systematic study, De Renzi, Pieczuro and Vignolo (1966) found oral apraxia to be highly associated with Broca's aphasia, but not with Wernicke's aphasia. Poeck & Kerschensteiner (1975) also found imitation of oral movements to be most impaired in Broca's aphasics. Employing the classification of aphasias into "fluent" and "nonfluent" (Benson, 1967), one might say that oral apraxia has been found to be present especially in cases of nonfluency, where speech is characterized by disturbed effortful phoneme production, and where the oral apraxia is seen as another manifestation of a primarily motor disturbance.

On the other hand, the speech production errors emitted by a fluent aphasic are not generally considered as motor impairments, but as linguistic impairments. This position has recently been explicitly stated by Martin (1974), who proposes that the selection and ordering of phonemes must operate in reference to the phonological rules of language and as such are linguistic events rather than motor events. Clearly, one would not expect defects in the production of nonverbal movements to occur in association with a primarily linguistic defect, and as we have noted, oral apraxia is not typically seen in association with fluent aphasia.

In contrast, Liepmann (1913) long ago suggested that "Aphasia (not only motor aphasia but every expressive-aphasic disturbance of speech and writing) and apraxia are, however, essentially similar." From this point of view, both fluent and nonfluent aphasias may be usefully regarded as motor defects, which differ only in the kind or complexity of the motor impairment. Tests for oral apraxia typically employ fairly simple, usually single, movements, e.g., protruding the tongue, blowing, chattering the teeth, etc. A fluent aphasic by definition does not have particular difficulty in initiating or producing individual speech sounds, so that it is perhaps not surprising that he has no difficulty in producing relatively simple nonspeech movements. However, if one regards fluent aphasia as an impairment in putting several speech sounds (movements) together according to some required pattern, then one might expect the reproduction of more complex nonverbal movements also to be impaired. Such an interpretation would be consistent with the suggestion that the left hemisphere is specialized primarily for certain kinds of motor function, and that the verbal or nonverbal nature of the motor task is not critical to the appearance of a defect after left-hemisphere damage (Kimura & Archibald, 1974).

This hypothesis was put to the test in the present study, in that a series of oral motor tasks, ranging in complexity from single movements to complex (multiple) movements, both verbal and nonverbal, was presented for reproduction to neurological patients.

It was expected that nonfluent aphasics would have difficulty on all types of oral movement, simple and complex, verbal and non-verbal. It was also expected that fluent aphasics, while they might have no difficulty with the simple oral movements, should have difficulty with the more complex movements, whether verbal or not.

METHOD

Subjects

Subjects were 13 patients (7 females, 6 males) with right-hemisphere damage and 23 patients (7 females, 16 males) with left hemisphere damage admitted to the University Hospital, London, Ontario. Most patients were located in the Neurology and Neuro-surgery ward but some were in Rehabilitation Medicine. The sample includes all patients seen within a twelve month period who showed evidence of strictly unilateral vascular damage to one hemisphere of the brain. No patient with evidence of bilateral damage was included. All patients were right-handed. The mean age of the patients was 50.3 years with a range of 22 to 72 years.

Selected portions of the Minnesota Test for Differential Diagnosis of Aphasia were used to classify patients as aphasic or nonaphasic. Six tests chosen to yield measures of both comprehension and expression were administered to all patients. The tests of comprehension consisted of A.1 - recognizing common

words (max score = 18), A.4 - identifying items named serially (15) and A.5 - understanding sentences (10) for a maximum comprehension score of 43. The tests of expression consisted of C.5 - counting to 20 (max score = 20), C.6 - naming days of the week (7), and C.13 - naming pictures (20), for a maximum expression score of 47. The maximum possible combined score was 90. The mean score for the thirteen patients in the right-hemisphere group was 89.8 with a range of 89-90. Using 88 as a cut-off score, all of the left hemisphere damaged patients were classified as either aphasic or nonaphasic. Ten of the 23 left hemisphere damaged patients, designated nonaphasic, obtained scores of 89 or 90. The remaining thirteen patients in this group obtained a mean score of 67.8 with a range of 24 to 87 and were classified as aphasic.

Phrase length, the number of utterances per verbal response, is a variable found to provide a high power of discrimination between fluent and nonfluent aphasic speech (Goodglass, Quadfasel & Timberlake, 1964; Kerschensteiner, Poeck & Brunner, 1972). In order to classify the aphasics as fluent or nonfluent, the mean phrase length of a series of verbal responses was determined for all aphasic patients. Responses to the Picture Description task (C.12) or the aphasia test cited above were recorded on tape and transcribed. Phrase length was measured by the number of utterances, whether syllables or unintelligible vocalizations, between

two distinct interruptions in the flow of speech. Interruptions were defined as the initiation of a new sentence or a temporal break of greater than two seconds. The first five consecutive phrases produced by the patient were analyzed. On the basis of the analysis two distinct groups of aphasics emerged. Four patients had a mean phrase length of 1.8 utterances with a range of 1 - 2.8, while the remaining eight had a mean phrase length of 9.0 utterances with a range of 7.0 - 11.2. Patients in the first group were designated as nonfluent aphasics, those in the second group as fluent aphasics. Mean phrase length was also determined for seven of the nonaphasic patients with left-hemisphere damage and was found to be 9.84 (range 5.6 - 13.4), not significantly different from that of the fluent aphasic group.

Thus, using the variables of lesion laterality, aphasia and fluency, four groups of patients were designated (see Table 1): a) 13 right-hemisphere damaged patients, b) 10 left-hemisphere damaged, nonaphasic patients, c) 9 left-hemisphere damaged, fluent aphasic patients, and d) 4 left-hemisphere damaged, nonfluent aphasic patients.

Analysis of variance yielded no significant differences in age ($F[3,32] = 1.02$) or in time of testing after onset ($F[3,32] = 1.13$) between the groups. (Table 1). The median time between onset of symptoms and test date was eight weeks with a range of 1 to 208 weeks. The characteristics of the patient groups are summarized in Table 1.

All but four patients demonstrated unilateral paresis and/or visual field defects at the time of testing. Three of these, in the left-hemisphere damaged nonaphasic group, had shown one or more of the above clinical signs during transient ischemic attacks and all three demonstrated angiographic evidence of left cerebral vascular abnormality. The fourth patient presented no clinical signs other than aphasia, but a left temporal lesion was confirmed by brain scan and electroencephalography. Of the remaining 33 patients with lateralized clinical signs, all but three demonstrated hemispheric abnormality on one or more of the following medical tests; angiography, electroencephalography, brain scan. A more detailed breakdown of etiological factors can be found in Table 1.

(Insert Table 1 about here)

Procedure

Intellectual Testing

The Wechsler Adult Intelligence Scale was administered to 34 patients and the Wechsler Bellevue II was administered to 3 patients, although in eight cases, aphasia precluded determination of the Verbal IQ. To minimize the effect of dominant hand paresis, a prorated Performance IQ which did not include the Digit-Symbol subtest was used.

Nonverbal Motor Tasks

Single oral movements. The patients were instructed to imitate each of 12 simple movements using the oral-facial musculature,

TABLE 1

Characteristics of Patient Groups

Lesion Group	N	Mean Age in Years	Test Time After Onset		Clinical Evidence		Etiology		
			Under 8 Weeks	Over 8 Weeks	Contralateral Hemiparesis	Visual Field Defect	Completed CVA	TIA	Aneurysm (+ Surgery)
Right damage	13	45.9	7	6	10	6	9	-	4
Left-nonaphasic	10	52.7	5	5	6	1	4	6	-
Left-fluent aphasic	9	50.1	4	5	7	2	7	2	-
Left-nonfluent aphasic	4	59.0	0	4	4	3	4	-	-

demonstrated by the experimenter: (a) mandibular depression, (b) midline lingual protrusion, (c) lingual lateralization left, (d) lip protrusion, (e) lingual lateralization right, (f) upper teeth on lower lip, (g) bilateral lip retraction, (h) blow, (i) clear throat, (j) clench teeth, (k) hum, and (l) whistle. Care was taken to stay within the patient's good visual field and to ensure that he was looking at the experimenter's face before the movement was made. A score of 1 was given for each movement correctly reproduced, for a maximum score of 12 on the task. Reduction in range of movement due to obvious paresis of the facial musculature was not penalized. All responses were videotaped to provide a check on scoring accuracy. (Appendix 1).

Multiple oral movements. In this task, the experimenter demonstrated a sequence of movements of the oral-facial musculature, and the patient copied the sequence as soon as the experimenter had completed it. A movement sequence consisted of three of the single movements described above, presented at one second intervals. Five such sequences were presented. The following are brief verbal descriptions (not given to the patient) of the five sequences: (a) bilateral lip retraction, lingual protrusion, upper teeth on lower lip, (b) lingual sweep across upper lip, clench teeth, clear throat, (c) lingual protrusion, lip protrusion, hum, (d) lingual lateralization, mandibular depression, lip protrusion, and (e) lingual protrusion, clench teeth, upper teeth on lower lip. Again, all responses were videotaped. Each of the component movements was individually scored. Thus a max-

imum score of 15 (3 x 5) was obtainable. Secondly, a score reflecting accuracy in the correct sequencing or ordering of the movements was determined. After eliminating response trials in which the patient produced only one movement, the number of movements produced in the correct order on remaining trials was divided by the number of movements produced, to yield a percentage score.

Verbal Tasks

Single phonemes. The patients were instructed to produce single phonemes which the experimenter presented while sitting across a table in full frontal view of the patient. Eighteen consonants, nine vowels and three diphthongs were presented for imitation. The patients' recorded utterances were transcribed by a phonetically-trained listener (senior author) into the alphabet of the International Phonetic Association. Correct production was judged on the basis of examiner-perceived acoustic accuracy. A score of 1 was given for each phoneme correctly produced for a maximum score of 30.

Multiple phonemes → meaningless syllables. In this task the patients were required to imitate a sequence of three meaningless consonant-vowel syllables (six phonemes); e.g., tʌkiso, daʃɛgu. The task consisted of six different sequences, constructed so that each consonant occurred only once during the task. Only the first six phonemes produced by the patient on each trial were scored. A score of 1 was given for each phoneme correctly produced, re-

gardless of order, allowing a maximum score of 6 on each sequence, a score of 36 on the task. Secondly, in a manner similar to that used in the multiple oral movements task, the percentage of phonemes produced in the correct order when two or more were produced was determined.

Multiple phonemes - words and phrases. In this task the patients were instructed to repeat 12 single words (42 phonemes) and 4, two-word phrases (18 phonemes). Each phoneme was scored separately, and order of production was not considered. A maximum score of 60 (42 + 18) could be obtained on the task.

Speeded syllable repetition.

(1) Repeated production of a single consonant-vowel syllable: The patient was asked to repeat the syllable /bʌ/ as quickly as possible. The number of correct productions of the syllable /bʌ/ over a five-second interval was determined from tape recordings. The same measure was taken for rapid repetition of the syllables /dʌ/ and /gʌ/. Since individual phoneme comparisons were not of interest, a mean rate of production was determined over the three syllables.

(2) Repeated production of a trisyllabic sequence: The patient was asked to repeat a sequence of three consonant-vowel combinations used in the above task; i.e., /bʌdʌgʌ/. To ensure that the patient knew the task, the sequence was presented slowly by the examiner before the first repetition request and repeated slowly until the patient produced at least two successive correct productions of the entire sequence. The time between intitial

presentation of the sequence by the examiner, and the completion of two correct sequences by the patient was recorded in each case. The examiner then demonstrated rapid repetition of the sequence for a seven second period, and the patient was asked to produce the sequence as rapidly as possible. The number of correct productions of the entire sequence produced during a five-second interval was counted, again from tape recordings. As this task was initiated later in the study, data are reported for only 32 patients.

Visual sequential memory test. Since the movement copying task obviously calls upon some visual memory skill, a visual sequential memory test was given. A modified version of the Visual Sequential Memory Test, part of the Illinois Test of Psycholinguistic Ability (Kirk, McCarthy & Kirk, 1968) was presented. This test is similar to short term memory tasks such as digit span but presented nonverbally and in a visual mode. In this test, a card depicting black geometric forms in a particular sequence was presented for ten seconds. The patients were then required to order by hand a series of black geometric forms presented on 1" x 1" white plastic chips. Two forms were initially presented for ordering. If the patient responded correctly, three forms were presented on the next trial. If the response was incorrect at any level, a second trial with different but equal numbers of forms was given. Testing proceeded until the patient failed both trials at a particular level. The score given was the highest

number of forms correctly sequenced.

Sensory tests. Cutaneous two-point discrimination thresholds were determined on the left and right sides of the tongue at a site 1 cm posterior to the tongue tip using an aesthesiometer (Lafayette Instruments). Patients were asked to judge whether one or two points had been applied to the tongue surface and respond by raising either one or two fingers. The trials began with a separation between the points well above threshold for discriminating them and decreased in 0.3 cm increments until a criterion level of 75% correct judgments was not met. An ascending trial, begun with a point separation well below threshold, proceeded with increasing increments of separation until the 75% correct criterion level was met. The two-point threshold at each site was the mean of the last descending and ascending value at which criterion was reached. The patient was considered to demonstrate a lingual sensory deficit when the mean of the left- and right-sided thresholds exceeded that of any of seven normal control subjects tested with the same apparatus. Control subjects were of comparable age and had no history of neurological involvement.

RESULTS

Intellectual Testing

Analysis of intellectual testing yielded results consistent with the literature (Table 2). Patients with right hemisphere damage, as a group, had a lower performance IQ relative to their Verbal IQ. Patients with left hemisphere damage demonstrated a lower Verbal IQ relative to their Performance IQ, particularly in the case of aphasic patients. There was no significant difference between the Performance IQ's of the right hemisphere damaged group and the three left hemisphere damaged groups ($F[3,32] = 0.93$), suggesting comparable levels of nonverbal visuospatial abilities.

(Insert Table 2 about here)

Nonverbal Motor Tasks

Because several of the tasks resulted in ceiling effects in one or more of the groups, reducing the variance in these cells, all such groupwise comparisons were done using the non-parametric Kruskal-Wallace One Waynalysis of Variance. When ceiling effects were not encountered, scores were subjected to one-way analysis of variance. Pairwise comparisons were made using the Mann Whitney U test or the t test. Significance levels were adjusted for use of these tests in multiple comparisons. Although the tabled data is presented as percentage of items correct for easy across task comparisons, statistics were based on raw scores.

TABLE 2

IQ Data on Patient Groups

Group	N	Full Scale IQ	Verbal IQ	Performance IQ (Prorated)
Right hemisphere damage (nonaphasic)	13	101.2	107.5	89.7
Left hemisphere damage (nonaphasic)	10	99.7	97.9	101.9
Left hemisphere damage (fluent aphasic)	9	77.3 (n=6)	71.0 (n=6)	91.3 (n=9)
Left hemisphere damage (nonfluent aphasic)	4	-	-	89.3 (n=4)

Single oral movements (Table 3). The nonfluent aphasic group was significatnly impaired on this task relative to the fluent aphasic group ($t = 3.83$, $df = 11$, $p < .005$), the left hemisphere damaged nonaphasic group ($t = 4.18$, $df = 17$, $p < .005$) and the right hemisphere damaged group ($t = 4.33$, $df = 20$, $p < .005$). There were, however, no significant differences between the latter three groups. Thus, only the nonfluent aphasics were impaired in imitating relatively simple discrete movements of the oral musculature. Two additional nonfluent aphasics not included in the study because there was angiographic evidence of right hemisphere as well as left hemisphere involvement also obtained scores on this task below the range of all other groups.

(Insert Table 3 about here)

Multiple oral movements (Table 3). Fluent aphasics, who did not show a defect on producing single oral movements demonstrated a highly significant impairment on this task relative to each of the two nonaphasic groups, left hemisphere damaged ($t = 4.88$, $df = 17$, $p < .005$) and right hemisphere damaged ($t = 5.18$, $df = 20$, $p < .005$). As would be expected from the results of the single oral movements, the nonfluent aphasics were impaired on this task as well. Although there was a trend for the nonfluent aphasic group to perform more poorly than the fluent aphasics, this did not reach significance. There was no significant difference between the performance of the two nonaphasic groups on this task. Thus, both aphasic groups were clearly impaired relative to nonaphasic left-hemisphere and right-hemisphere damaged groups in their ability

TABLE 3

Nonverbal Tasks: Percentage of Items Correct

	Group	N	Single Oral Movements (Maximum = 12)	Multiple Oral Movements (Maximum = 15)
Right damage:	nonaphasic	13	95	77
Left damage:	nonaphasic	10	96	78
Left damage:	fluent aphasic	9	90	41 *
Left damage:	nonfluent aphasic	4	58 *	22

Single Oral Movements: $F[3,32] = 13.30$, $p<.01$

Multiple Oral Movements: $F[3,32] = 15.91$, $p<.01$

*$p<.005$

to produce a sequence of nonverbal oral movements.

Percentage scores, reflecting the proportion of movements which are produced in the correct sequence or order, are listed in Table 5. Although the component score indicates the significantly fewer correct movements were made by patients in the two aphasic groups, the proportion of movements which were produced in the correct order did not differ between groups. Thus, if a movement was produced at all, it was likely to be in the correct sequence.

Verbal Tasks

Single phoneme production (Table 4). The nonfluent aphasic group obtained a significantly lower mean score than the fluent aphasics on this task ($U = 2$, $p < .01$). The fluent aphasic group was also impaired relative to the left hemisphere damaged nonaphasics ($U = 9$, $p < .01$) and to the right hemisphere damaged group ($U = 0$, $p < .01$). Nonfluent aphasics and to a lesser extent fluent aphasics thus had difficulty imitating single speech movements.

(Insert Table 4 about here).

Multiple phonemes - meaningless syllables (Table 4). Relative to the single phoneme task, this multiple phoneme task disclosed a more striking imapirment in the fluent aphasics relative to both the nonaphasic left hemisphere damaged and right hemisphere damaged patients ($U = 4$, $p < .001$ for each comparison). Although both aphasic groups, fluent and nonfluent, were significantly impaired on this task, the difference between the performance of the

TABLE 4

Verbal Tasks: Percentage of Phonemes Correct

Group		N	Single Phonemes (Max = 30)	Multiple Phonemes Syllables	Multiple Phonemes Words & Phrases (Max = 60)
Right damage:	nonaphasic	13	97	99	100
Left damage:	nonaphasic	10	97	99	100
Left damage:	fluent aphasic	9	77 *	67 **	78 **
Left damage:	nonfluent aphasic	4	48 *	43	52

Single phonemes: $H[3,32] = 20.02$, $p < .001$

Syllables: $H[3,32] = 23.73$, $p < .001$

Words & phrases: $H[3,32] = 24.37$, $p < .001$

* $p < .01$

** $p < .001$

nonfluent aphasics and that of the fluent aphasics did not reach significance. The left and right nonaphasic groups both perform near ceiling on this task. As was found on the multiple oral movements task the percentage of phonemes produced in the correct order when two or more were produced was high (greater than 90) for all groups (Table 5).

(Insert Table 5 about here)

Multiple phonemes - words and phrases (Table 4). The results of this task are essentially the same as those found in the meaningless multiple phoneme task reported above ($H[3,32] = 24.37$, $p<.001$). That is, performance of fluent and nonfluent aphasic groups did not differ significantly while each group is impaired relative to the nonaphasics who perform at ceiling. The product-moment correlation co-efficient for the left hemisphere damaged aphasic patients ($N = 13$) between performance on the meaningless and meaningful multiple phoneme tasks was .82 ($df = 11$, $p<.005$).

Speeded syllable repetition (Table 6).

(1) Rapid repetition of single CV combinations: The nonfluent aphasics produced significantly fewer repetitions of a single consonant vowel combination in five seconds than any other group (each $t \geq 4.35$, $p<.005$). There were no significant differences between the other groups, fluent aphasics producing the series as rapidly and accurately as the nonaphasic left- and right-hemisphere damaged groups.

(2) Rapid repetition of a series of three consonant-vowel

TABLE 5

Percentages of Responses Made in the Correct Sequence

Group		N	Multiple Oral Movements	Multiple Phonemes Syllables
Right damage:	nonaphasic	13	96.2	100.0
Left damage:	nonaphasic	10	96.6	100.0
Left damage:	fluent aphasic	9	93.3	90.6
Left damage:	nonfluent aphasic	4	100.0	95.3

combinations: Before a five second repetition rate was determined on this task, the patient was required to produce at least two correct /bʌdʌgʌ/ sequences. Not one of the four patients demonstrating nonfluent aphasia produced two correct sequences within a 30 second practice period. Three of the eight fluent aphasics tested did not produce two correct sequences, while each of the twenty nonaphasic patients tested did so. The five fluent aphasics who achieved criterion took a mean of 13.4 seconds to produce the required two correct sequences. This was significantly longer ($p<.01$) than the mean times (5.6; 5.7 seconds) taken by the left and right hemisphere damaged nonaphasics.

Analysis of group performance based on those patients who reached the criterion of two correct sequences yielded a significant difference in the number of completed sequences during a five second interval ($F[2,22] = 11.05$, $p<.01$). The fluent aphasics completed a mean of 3.0 sequences, significantly fewer ($p<.01$) than the means of the left-hemisphere damaged nonaphasics and right-hemisphere damaged patients, 8.5 and 8.3 respectively. Thus, though the fluent aphasics repeated a single consonant vowel combination as rapidly and accurately as the nonaphasic groups, they were clearly impaired in the production of a sequence of different consonant vowel combinations.

(Insert Table 6 about here)

TABLE 6

Production of Syllables Over 5-Second Interval

Group		N	Single Consonant-Vowel Combinations /baba/,/dada/,/gaga/	Sequences of Three Consonant-Vowels /badaga/	Time to Criterion (sec)
Right damage:	nonaphasic	12	24.1	8.3	5.7
Left damage:	nonaphasic	8	28.2	8.5	5.6
Left damage:	fluent aphasic	8	25.3	3.0 *	13.4 *
Left damage:	nonfluent aphasic	4	15.3 *	-	-

Single: $F[3,28] = 5.33$ $p<.01$

Sequences: $F[2,22] = 11.05$ $p<.01$

*$p<.01$

Visual Sequential Memory Task (Table 7).

Analysis of variance yielded no significant differences between the groups on this task ($F[3,27] = 1.33$). Thus, there was no evidence that the groups differed in their ability to manually order visual symbols from memory.

(Insert Table 7 about here)

Sensory Testing.

The relationship between lingual sensory deficit, based on two point discrimination thresholds and impaired nonverbal oral movement was investigated. The greatest mean two point threshold for left and right lingual sites combined for a control subject was 1.25 cm, with a mean threshold in controls of .96 cm. Of the patients for whom thresholds could be obtained, both of two nonfluent aphasics, four of eight fluent aphasics and five of thirteen right hemisphere patients, had mean thresholds greater than 1.25 cm and, on that basis, were considered to show a lingual sensory defect. There was no significant difference between the mean discrimination threshold of the fluent aphasics (1.49 cm) and the right hemisphere damaged patients (1.25 cm), who scored significantly higher on the nonverbal multiple oral movements task. If only the fluent aphasics are considered, both the four patients demonstrating a sensory defect and the four without a defect obtain mean scores on the multiple oral movements task (4.5 and 7.3 respectively) which are lower than the mean scores of the right hemisphere damaged patients with or without sensory

TABLE 7

Visual Sequential Memory

Group	N	Score
Right hemisphere lesions (nonaphasic)	12	4.7
Left hemisphere lesions (nonaphasic)	7	4.4
Left hemisphere lesions (fluent aphasic)	7	3.9
Left hemisphere lesions (nonfluent aphasic)	5	4.8

defect (10.6 and 12.1 respectively). Thus, lingual sensory defects did not necessarily occur in conjunction with impaired nonverbal oral movement.

Summary of Results (See Fig. 1)

Nonfluent aphasics were impaired relative to nonaphasic groups on all oral movement tasks, and relative to the fluent aphasic group on single oral movements, single phonemes, and speeded repetition of a single syllable. Fluent aphasics were impaired relative to nonaphasics on the imitation of multiple nonverbal oral movements as well as on all verbal tasks except speeded repetition of a single syllable. The left and right hemisphere damaged nonaphasic groups did not perform significantly differently on any task.

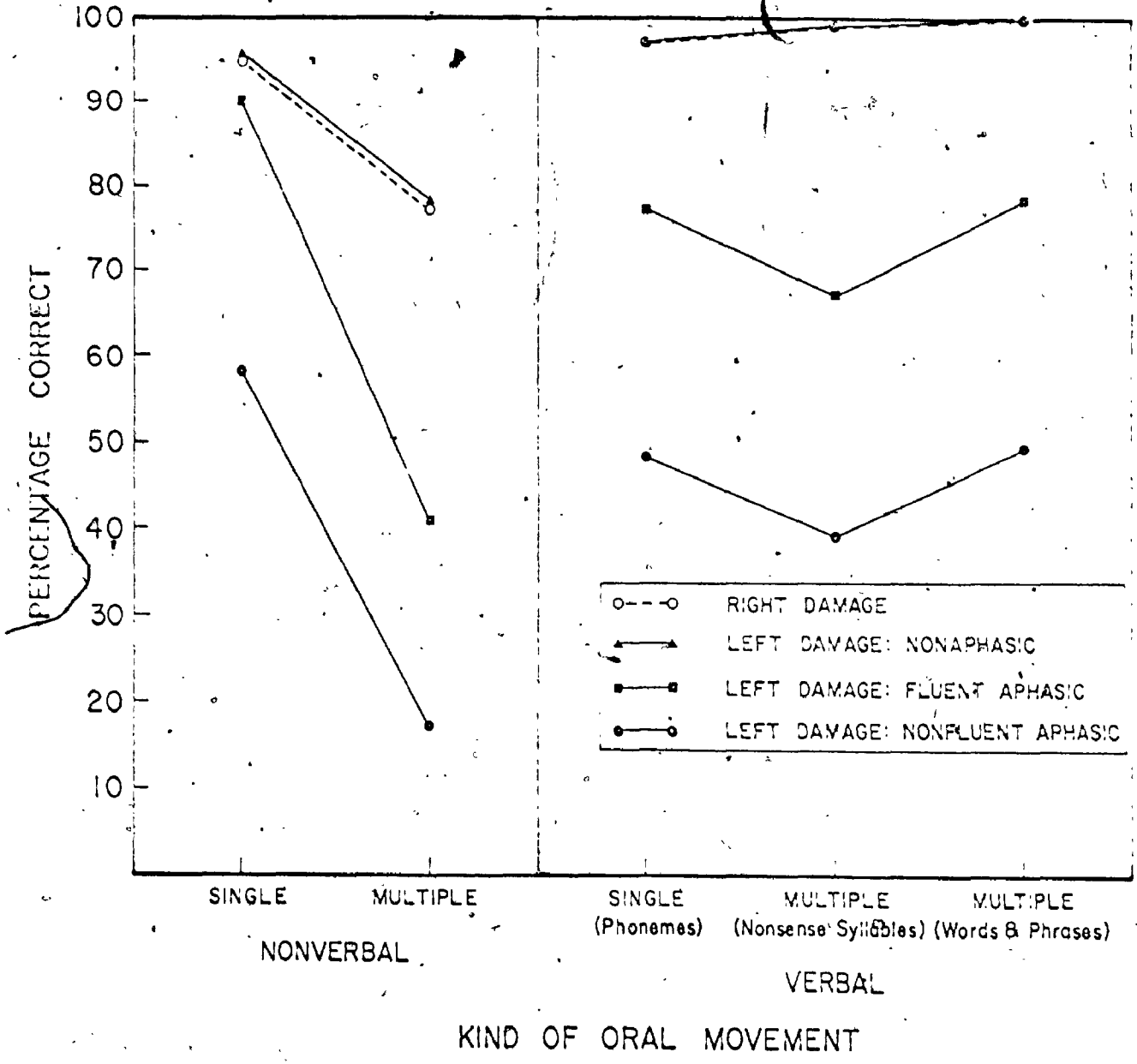
(Insert Figure 1 about here)

DISCUSSION

In agreement with earlier reports in the literature, nonfluent aphasics were impaired in copying simple nonverbal oral movements, but fluent aphasics were not. However, fluent aphasics did show significant deficits in the production of nonverbal oral movements when the task was more complex. Thus although fluent aphasics could imitate single oral movements adequately, they were clearly impaired in imitating multiple oral movements, where the single movements were presented in groups of three and had to

Figure 1

Summary of Results: Performance of Groups on single and multiple, nonverbal and verbal tasks.


100
90
80
70
60
50
40
30
20
10
PERCENTAGE CORRECT
RIGHT DAMAGE
LEFT DAMAGE: NONAPHASIC
LEFT DAMAGE: FLUENT APHASIC
LEFT DAMAGE: NONFLUENT APHASIC
SINGLE
MULTIPLE
SINGLE (Phonemes)
MULTIPLE (Nonsense Syllables)
MULTIPLE (Words & Phrases)
NONVERBAL
VERBAL
KIND OF ORAL MOVEMENT

be reproduced in the presented order.

This inability to imitate a series of oral movements is not readily explicable on the basis of defects in visual perception or memory. For example, there were no differences in Performance IQ, which is one measure of visuospatial function, between the aphasics and patients with right-hemisphere damage, yet the latter could do the task quite well. It will also be recalled that the four groups of patients did not differ in their ability to manually order visually presented nonsense designs, a measure of visual sequential memory. Finally, some preliminary data not reported in the Results section, suggest that fluent aphasics can select up to three demonstrated movements from a series of photographs of the movements, as well as patients with right hemisphere damage. Thus, neither visual perceptual deficits nor visual memory span deficits appear to account for the poorer performance of the fluent aphasics on the nonverbal oral movements task.

One study (Rosenbeck, Wertz, and Darley, 1973) has suggested that severity of articulation disorder may be related to the degree of oral sensory deficit. The limited results available on lingual sensory deficits in our study, however, do not appear to account for the movement impairments. The two-point threshold for the tongue does not differentiate between fluent aphasics and nonaphasic patients with right-hemisphere damage, though these two groups differ markedly in their reproduction of oral movements.

Moreover, fluent aphasics without lingual sensory deficit are almost as impaired in the production of nonverbal oral movements as those with sensory defect. Thus, while sensory defects may impair movement control, the bulk of the deficit seen in the present study must be attributable to other factors.

Given that the poor performance by the fluent aphasics on the nonverbal oral movements task is not reducible to visual or sensory deficits, what is the nature of the impairment? The nonverbal movement deficit only appeared when the task required several changes in the positioning of the oral musculature, single oral movements being unimpaired. Instead of making three distrete movements, the patients often produced random, continuous, amorphous oral posturing, as if searching for correct placements. Occasionally, there was perseveration on a movement which had occurred earlier in the same sequence or on previous trials, or a totally unique oral movement might be made. Frequently, no more than one or two movements appeared to be attempted at all.

Although the requirement of more than one change in the target configuration of the muscles maximizes the movement deficit, the impairment does not appear to be one of ordering or sequencing alone. Both nonverbal and verbal movements which are produced by the fluent aphasics are likely to be produced in the correct order; the lowered scores on the multiple oral movements task and multiple phoneme imitation task represent fewer

total correct movements. In fact, over 90% of the movements produced for all patients on both these tasks are in the correct order. These results are in agreement with those of Weiner (1969) who demonstrated that although dysphasics were impaired on sequential oral-movement tasks, the problem was not, as suggested by LaPointe and Wertz (1974), one of incorrect ordering of the movements. This is perhaps surprising in view of the prevalent opinion that speech makes heavy demands on a serial ordering process (see MacNeilage, 1970, for review). However, many of the identifiable errors in aphasic speech are not primarily sequencing defects. Thus a substitution of one consonant for another in a word represents rather an incorrect selection of a target phoneme than an error in phoneme order. We have seen no aphasic pattern in which there are sequencing defects independent of errors in target sounds. In our study, even when the situation was optimal for the production of errors in sequencing alone, such as in the rapid repetition of three different syllables (badaga), after rehearsal, errors were not exclusively sequential. Often there was instrusion of extraneous syllables or sounds, or only two of the syllables were repeated. In other words, the deficit appears in a failure to achieve target motor responses when more than one is required rather than just an improper ordering of those targets.

The defect in the reproduction of nonverbal oral movements, seen in almost all our aphasic patients, is all the more striking

when one considers that the presentation of the movements was entirely in the visual modality, whereas the movements involved in speaking are closely tied to, and monitored by, auditory characteristics. Given the auditory-vocal nature of our speech tasks, receptive deficits would be expected to interact with motor deficits in aphasic patients to affect performance more severely on the verbal than on the nonverbal tasks. In fact, just the opposite occurred, with scores on the nonverbal oral movements task being lower for all groups. From a motor control explanation, this would be expected, however, since the multiple oral movements are clearly unfamiliar and unpractised, relative to the verbal oral movements, and would thus be more sensitive to central nervous system damage than the production of the overlearned speech movements. The fact that the familiar (speech) patterns (which also have greater representational content) are spared in aphasics relative to the unfamiliar, is of course not in accord with the idea that the primary function of the left hemisphere is language.

It will be recalled that the nonfluent aphasics were impaired relative to fluent aphasics in the imitation of single oral movements. On the verbal tasks, the fluent and nonfluent aphasics differed significantly only in the production of single phonemes and the repetition of single consonant-vowel combinations (bababa), nonfluent aphasics again demonstrating greater impairments. It is tempting to draw a parallel between verbal and nonverbal tasks in the apparent dissociability of the control of

single and multiple movements. Such dissociability might suggest at least two systems operating in the motor control of speech, one which is involved in the production of relatively discrete oral movements, perhaps of phonemes, and other operating to effect the transition from one discrete movement to another in a smooth and orderly way. Presumably this latter system could also be involved in the selection or programming of the movements into longer sequences. The anatomical areas corresponding to such a schema would be the anterior speech area for control of single units, and the posterior speech zone for the programming of such units. At present, however, we must keep in mind the distinct possibility that the single and multiple oral movements represent simply degrees of difficulty along a unitary dimension, and that the difference between fluents and nonfluents on these tasks represents a difference in degree, rather than in the nature of the impairment.

Classification in Aphasia

We have found the dimension of fluency in aphasia (Goodglass, Quadfasel and Timberlake, 1964) to be useful in several respects. First, the criterion employed, mean length of response in syllables is easily applied and usable across examiners, a characteristic notably lacking in most classification systems. Second, in our sample there was a clear separation of groups, with no overlap, and no tendency to overlap. If this pattern is maintained with future groups, it will indeed suggest that there

is a dichotomy here, rather than a unitary dimension. Third, in the analysis of our findings, there was a meaningful relationship between the fluent/nonfluent classification and results on other tests. Finally, there may be an anatomical correlate to this distinction, in that the brain scan localization study by Benson (1967), and indeed many clinical observations from the time of Broca (1861), would implicate the anterior speech area of the left hemisphere in the nonfluent deficit, while the posterior speech area would be involved in the fluent deficits.

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APPENDIX 1

Scoring Procedure Used in the Evaluation of Performance on the Oral Movement Tasks

During the actual testing session each oral movement correctly produced was indicated as such on a response sheet. A correct judgement was assigned when the examiner felt there was no doubt that a movement in the required direction was made by the required oral-facial structure. Movements restricted in apparent force or extent were not penalized. Judgements were based on the examiners observation of the response. Maximum distance between the examiner and the subjects face was two feet.

All responses were recorded on videotape (including audio track). Focal length was adjusted so that a full frontal face view extending from the neck to the top of the head was recorded. The full test sessions for each patient were recorded consecutively until a tape was full. After each test session the tape was reviewed. The accuracy of each response previously scored as correct was checked and a verbal description of each incorrect movement made was recorded.

At completion of the study the portion of tape containing the single oral movements task and the multiple oral movements task for each subject was spliced into a master tape (no speech tasks were included). Since subjects represented patients consecutively seen, selected only for medical evidence of strictly unilateral hemisphere dominance, the master tape provided a random

ordering of patients with right or left hemisphere damage and with or without aphasia. It was felt the scoring of responses on this tape, with patients identified only by number, would provide a blind condition score. Both the correctness/incorrectness score and the verbal description of incorrect movements on the initial score sheets and blind scoring procedure were compared. Any discrepancies were again reviewed on videotape and an independent judgement made by another observer was recorded.

PAPER NO. 2

IMPAIRMENTS OF NONVERBAL ORAL MOVEMENTS AFTER LEFT HEMISPHERE DAMAGE: A FOLLOWUP ANALYSIS OF ERRORS

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ABSTRACT

Acquisition of a task requiring the imitation of a series of nonverbal oral movements is most severely impaired in patients with aphasic disturbance. However, nonaphasic patients with left hemisphere damage also perform more poorly than patients with right hemisphere damage or normal control subjects. Analysis of the errors made on the multiple oral movements task reveals that left, but not right, hemisphere damage is highly associated with perseverative responses. Patients with right hemisphere damage do not differ significantly from normal control subjects in the acquisition of or in the type of errors made on the task. It is suggested that the left hemisphere plays an important role in the control of nonverbal oral movement production.

Mateer and Kimura (in press) have shown that nonfluent aphasics were significantly impaired in their ability to imitate relatively simple single movements of the oral musculature, movements of the kind used in traditional testing of oral apraxia. When the imitation of a series of nonverbal oral movements was required, both nonfluent and fluent aphasics were significantly impaired, relative to nonaphasic patients with left or right hemisphere damage. It was suggested that a fundamental aspect of both fluent and nonfluent aphasia may be deficient co-ordination of oral movements.

The purposes of this study were to: 1) determine whether patients with left and right hemisphere damage and normal control subjects differ in the types of errors made on the multiple oral movement task, and 2) to present previously unreported data on the acquisition of the task over successive trials. The results presented in the initial paper (Mateer and Kimura, in press) were based on performance on only the first trial of this multiple oral movement task.

Distinct differences between the groups in the prominence of particular error types, as well as in the overall incidence of errors were apparent. Analysis of task acquisition disclosed two important findings: 1) the impairment in aphasia is a consistent one with little improvement in performance over repeated trials, and 2) patients with left hemisphere damage, but no obvious aphasia, demonstrate significantly poorer acquisition over repeated trials

than do patients with right hemisphere damage or normal control subjects.

METHOD

Subjects

There were five subject groups: a) 21 patients with right hemisphere damage, b) 21 nonaphasic patients with left hemisphere damage, c) 12 fluent aphasics with left hemisphere damage, d) 8 nonfluent aphasics with left hemisphere damage and e) 27 age-matched normal control subjects. Of the total 62 patients with unilateral hemispheric damage, 45 had vascular and 17 had neoplastic lesions. The criteria for unilateral hemispheric damage, aphasia and fluency were the same as those presented elsewhere in detail (Mateer and Kimura, in press).

Procedure

In the multiple oral movements task, the subject must imitate a sequence of three relatively simple oral movements produced by the examiner. (The task is described in detail in Mateer and Kimura, in press.) Five different sequences were presented. If all three movements within one sequence were not produced or were produced in the wrong order, the examiner repeated the same sequence for imitation up to two more times. Thus, the number of trials administered to each subject could range from 5, in the case of all five sequences correctly produced on the first trial, to 15,

in the case of the maximum three trials taken for each of the five sequences. The patient was videotaped (full face and neck view) throughout administration of the task. Each oral movement produced was described and recorded at the time of testing and accuracy of scoring was checked by at least two reviews of the videotape. The analysis of error types was based on all trials administered.

As a measure of task acquisition of an oral movement sequence over repeated trials, the number of correct completed sequences produced within one, two and three trials was determined for each subject. Using this type of analysis, a patient who produced a correct oral movement sequence on the first trial was also given credit for having produced it within two and three trials, although these trials were not actually administered.

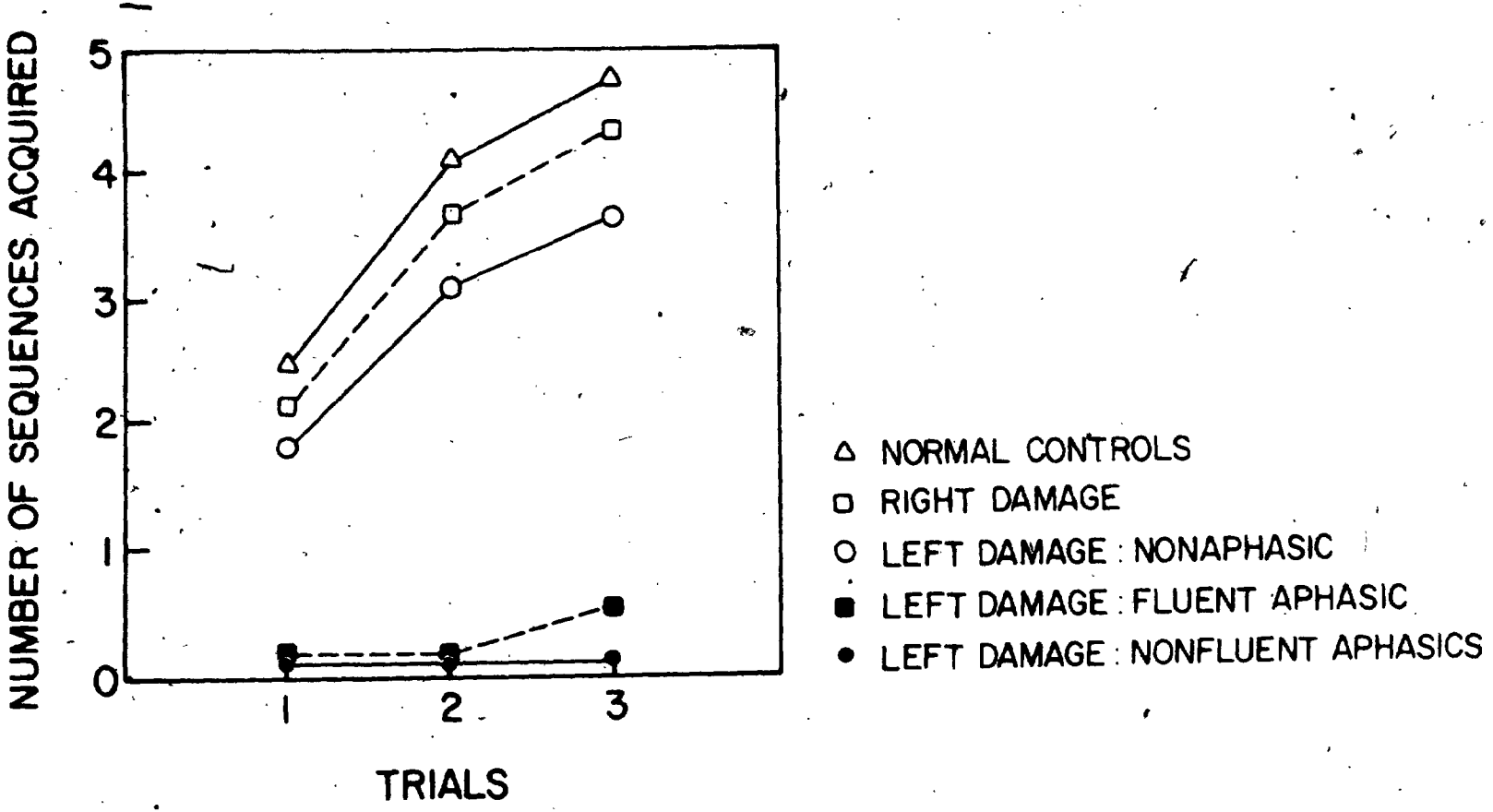
RESULTS

Acquisition of the multiple oral movement sequences

The mean numbers of correct oral movement sequences completed within one, two or three trials by subjects across the five groups are graphed in Figure 1. It is obvious that neither nonfluent nor fluent aphasics show any appreciable acquisition, at least in terms of correctly completed sequences, over the three possible trials. In fact, only three of the fluent aphasics and one nonfluent aphasic produced even one of the five sequences correctly within three tri-

Figure 1

Mean numbers of correct oral movement sequences acquired within the three available trials.


NUMBER OF SEQUENCES ACQUIRED
0
1
2
3
4
5
1
2
3
TRIALS
NORMAL CONTROLS
RIGHT DAMAGE
LEFT DAMAGE : NONAPHASIC
LEFT DAMAGE : FLUENT APHASIC
LEFT DAMAGE : NONFLUENT APHASICS

als. This difference between the two aphasic groups is not significant. In contrast, the three nonaphasic groups (left hemisphere damaged, right hemisphere damaged and normal controls) appear to demonstrate a fairly consistent pattern of acquisition of the oral movement sequences over a series of trials.

Although the shape of the acquisition curve is similar across the nonaphasic groups, inspection of the data revealed that only 33% of left hemisphere damaged nonaphasics successfully completed all five sequences within three trials versus 67% of the right damaged patients and 85% of normal control subjects who did so. This difference between the groups in the incidence of acquisition of all five sequences is significant [X^2 = 13.9 df, $p<.001$].

The acquisition data for these three nonaphasic groups were then subjected to a three by three, two way analysis of variance. Using the variables of group (left hemisphere damaged non-aphasic, right hemisphere damaged and control) and number of sequences acquired within available trials (one, two, and three trials, repeated measure). There were significant main effects between acquisition over the available trials [$F = 160.28$, df = 2,132, $p<.02$] and between groups [$F = 4.43$, df = 2,66, $p<.02$] with no significant interaction between these two variables. Multiple comparisons over trials revealed, as would be expected, that significantly more sequences were acquired as the number of available trials used increased. Comparison of group means revealed that the nonaphasic patients with left hemisphere damage produced significantly fewer

completed sequences across trials than patients with right hemisphere damage or control subjects ($p<.005$ for each comparison). The right hemisphere damaged and control groups did not differ significantly.

Thus, although they do not show the marked impairments observed in aphasic patients, even nonaphasic patients with left hemisphere damage are impaired on acquisition of the task over several trials relative to patients with right hemisphere damage and normal control subjects.

Analysis of error types

Nonfluent aphasics rarely attempted to make more than a single oral posture, and it was often a slow amorphous movement which was difficult to categorize. This was in contrast to more describable errors in movement observed in all other groups. Nonfluent aphasics, therefore, were not included in the analysis of error type, although they would of course differ from every other group.

Analysis of responses on the multiple oral movements task revealed four major kinds of errors: a) Omission errors -- production of fewer than three movements on a trial or partial responses in which some aspect of the required movement was missing, b) Unique errors -- including movements which were unrelated to the required ones in any obvious way, movements bearing some relation to the movements required but using an alternative oral-facial

structure, e.g., lateralizing the jaw rather than the tongue and the production of phonemic or nonphonemic vocalizations, c) Perseverative errors -- movements which were produced as correct movements on previous trials or earlier in the same trial and d) Reversals in sequence -- instances where two correct movements were produced but in the wrong order.

Since the task was, in part, designed to measure acquisition of the movement sequences, there were differing numbers of trials (5 to 15) across subjects. In order to achieve a common base for comparison of error types, the number of errors of each type was divided by the number of trials administered to obtain a mean number of errors per trial. (See means presented in Table 1).

Fluent aphasics made more errors per trial than any other group. An analysis of variance using the variables of group and the mean number of errors of each type per trial yielded both main effects and a significant interaction between the two variables [F, group x error type = 6.26, df = 9,231, $p<.01$]. Post hoc multiple comparisons (Newman-Keuls, Kirk, 1968) supported the following conclusions: a) all groups produced more omission errors per trial than errors of any other type; fluent aphasics showed the most omission errors and controls showed the fewest, b) fluent aphasic and nonaphasic patients with left hemisphere damage produced significantly more perseverative errors per trial than unique or reversal errors; both these groups produced more perseverative errors than right hemisphere damaged or control groups, c) fluent aphasics

TABLE 1

Mean Number of Errors Per Trial by Error Type and Group

Error Type	Group			
	Control N=27	Right Damage N=21	Left Damage Nonaphasic N=21	Left Damage Fluent Aphasic N=12
Omission	0.25	0.49	0.49	1.00
Perseverative	0.08	0.07	0.28	0.61
Unique	0.05	0.05	0.04	0.31
Reversal	0.03	0.06	0.01	0.05
TOTAL ERRORS	0.51	0.67	0.82	1.97

F, group x error type = 5,26, df = 9,231, $p<.01$

produced more unique than reversal errors per trial; this group also produced more unique errors than any other group, d) all groups produce fewer reversal errors than errors of any other type; the groups did not differ from each other on the mean number of reversals produced per trial. In summary, fluent aphasics appear to be differentiated from all other groups on the basis of unique errors, while both fluent aphasics and left hemisphere damaged nonaphasics are differentiated from non-left hemisphere damaged groups on the basis of perseverative errors. While these statistics are based on the number of errors of each type per trial, the interaction between group and error type is also readily apparent when the percentage of total errors accounted for by each error type within each of the groups is graphed (Figure 2).

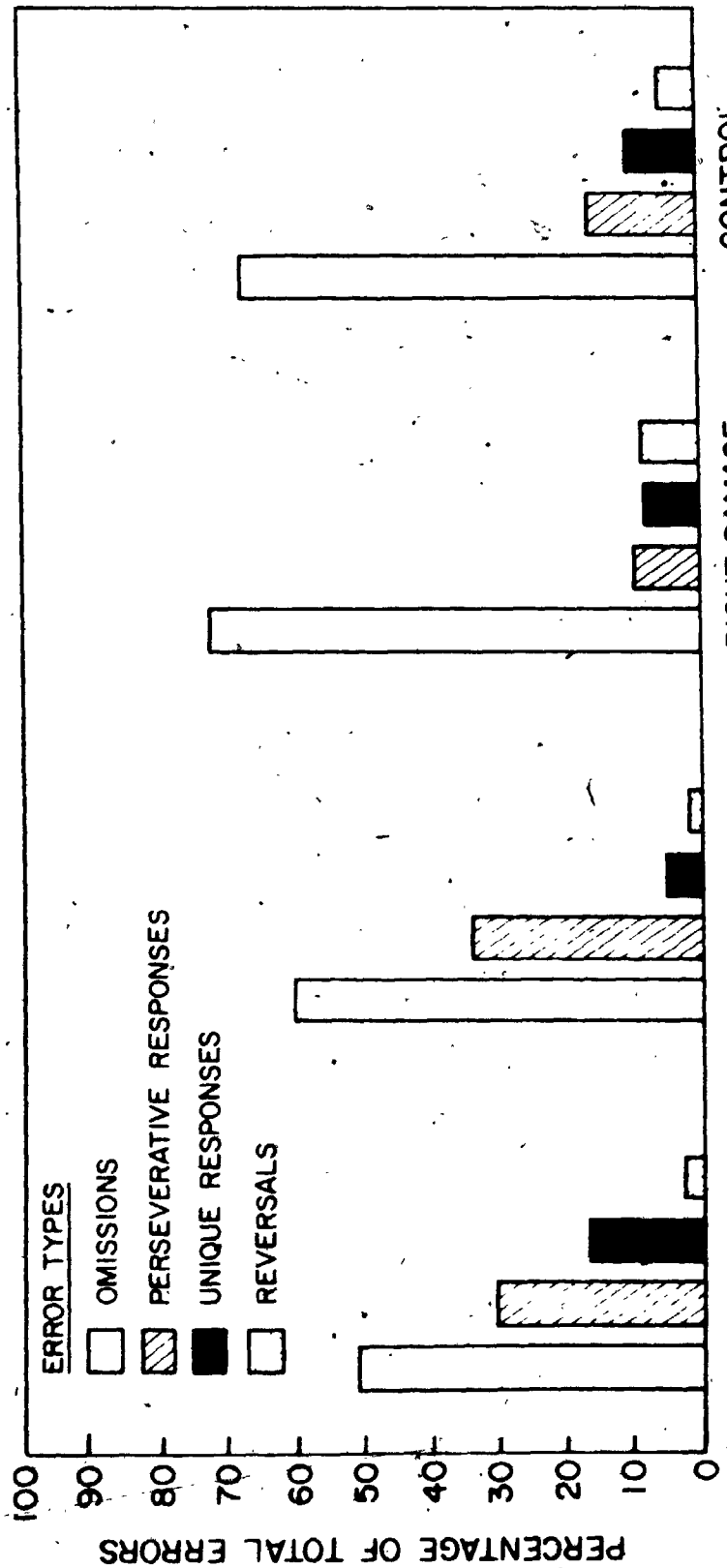
By definition, the perseverative error category included movements which had been produced correctly on previous trials as well as movements which were the same as movements which immediately preceded them. These latter "immediate" perseverations represented less than 22% of the total number of perseverative errors for each group.

DISCUSSION

The findings suggest that although aphasic patients are clearly the most severely impaired group in the acquisition and production of a series of nonverbal oral movements, nonaphasic

Figure 2

Percentage of total errors accounted for by each error type within each subject group.


PERCENTAGE OF TOTAL ERRORS
100
90
80
70
60
50
40
30
20
10
0
ERROR TYPES
OMISSIONS
PERSEVERATIVE RESPONSES
UNIQUE RESPONSES
REVERSALS
LEFT DAMAGE: FLUENT APHASIC
LEFT DAMAGE: NONAPHASIC
RIGHT DAMAGE
CONTROL
GROUP

patients with left hemisphere damage do not acquire the task as readily as do patients with right hemisphere damage or control subjects. In addition, the high proportion of perseverative errors demonstrated by nonaphasic patients with left hemisphere damage represents an error pattern more like fluent aphasics with left hemisphere damage than like the two groups without left hemisphere damage. It may be that the difficulty level, unfamiliarity and/or strict scoring criteria of the oral movements task contribute to revealing subtle deficits in motor control in the patients with left hemisphere damage. The relatively undemanding tests for aphasia and highly practised nature of speech may have prevented detection of subtle verbal impairments in left hemisphere damaged patients without obvious aphasia. In general, results could be taken to suggest some commonality underlying the motor deficits in both left hemisphere damaged groups, which is not related to a strict separation along the lines of aphasic impairment.

Analysis of the kind of errors made by all groups on the task suggests several factors which may relate to the observed performance. First, since the task involved the imitation of a series of movements after presentation by the examiner, the stimuli were no longer present during the subject's response, thus requiring some storage of the input. The fact that all groups, including the aphasics, made more omission errors than errors of any other type probably reflects the importance of this storage

factor. Both patient and control subjects frequently verbalized being unable to remember the movements, in association with omission errors. Although the patient groups did not differ, in the initial study, in their scores on a visual sequential memory task, the visual stimuli to be ordered or selected remained in view, reducing visual storage requirements. Since aphasic patients frequently hesitated or revised their oral movements, the latencies between the presentation of the oral movements for imitation and production of responses were increased, placing greater demands on memory function, and possibly contributing to the higher number of omissions per trial.

The perseverative errors made by the left hemisphere damaged group are of special interest. The term perseveration has most often been used to describe the repetition of an already executed action when a new movement is required (and perhaps intended). Wilson (1908) cites Pick's classic example of a patient who blew out a candle and then blew on all objects subsequently presented to him. Many of the errors made by nonfluent aphasics on the imitation of single oral movements (Mateer and Kimura, in press) were of this type, persistence on an <u>immediately</u> preceding movement. Unlike these errors, most perseverative errors produced on the multiple oral movements task by fluent aphasics and nonaphasic patients with left hemisphere damage were repetitions of movements which had been produced on a previous trial or movement sequence. Although some movements may be somewhat easier to pro-

duce or more familiar than others, results of the previous study (Mateer and Kimura, in press) showed that these groups were able to produce in isolation all the single movements which make up the movement sequences. Although transitions from one movement to another can be made, the probability of selecting or evoking certain previously produced movements is increased. Certainly perseveration is a frequently noted aspect of aphasic speech. Longstanding production of a single stereotypic utterance may be manifested in severe nonfluent aphasia. The high frequency production of specific words or phrases introduced at some point in a speaking session, that is, perseveration on previously produced responses, is often seen in association with fluent aphasia.

Unique errors produced by the fluent aphasic patients most closely resemble the random amorphous oral posturing of nonfluent aphasics, but without the obvious slowness and weakness demonstrated by the latter group. In some cases, a vocalization or movement of another oral structure, in addition to the required one, suggested a difficulty in moving an oral structure discretely or in isolating a motor response. Many patients appeared very uncertain about when an oral configuration had been achieved and continued making "searching" or "placing" movements. One patient described haltingly "losing my way around my mouth". The low frequency of reversal errors suggests that the deficit is not one of ordering movements per se.

These findings suggest that the left hemisphere has a specialized capacity for the production and sequencing of correct nonverbal as well as verbal oral movements. A similar left hemisphere function with respect to the production of limb movements has been reported. Kimura and Archibald (1974) found that patients with left hemisphere damage, whether aphasic or nonaphasic, were impaired on the production of a sequence of meaningless hand and arm movements. Impairment on this task correlated well with impairments on traditional tests of oral apraxia. In a more recent study, Kimura (1976), reported impairments in left hemisphere damaged patients on the acquisition and subsequent performance of a motor skill involving several changes in hand and limb posture. Analysis of the errors on the manual skill task revealed that perseveration and unrelated errors accounted for the major proportion of errors produced by the left, but not by the right, hemisphere damaged group. These are, of course, errors of a type similar to those seen on the complex oral movement task reported here.

Kimura suggests that a left hemisphere based system might be involved particularly in the movements of limbs or oral structures relative to some general body schema rather than to visually or otherwise externally guided targets. Certainly, the achievement of articulatory targets for speech requires movement of oral structures relative to one another for the attainment of oral tract configurations which correspond to specific acoustic pro-

ductions. MacNeilage and MacNeilage (1973) propose that control of speech depends on achieving such target configurations rather than on coding invariant movement patterns for the production of specific phonemes. Sussman (1972) suggests the motor system must be informed of the current spatial location of the articulators, as well as the next desired spatial location, to effect a movement which will reduce the discrepancy between the two. Perhaps requiring the production of oral movements in a series places greater demands on the analysis of changing spatial configurations of the articulators than the production of a single target posture from a "rest" position. The data presented in this study suggest that the achievement of successive target oral configurations, even those unrelated to phoneme production, is particularly dependent on the left hemisphere.

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PAPER NO. 3

PATTERNS OF ORAL-FACIAL SENSORY IMPAIRMENT AFTER UNILATERAL HEMISPHERIC DAMAGE

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Abstract -- Pressure sensitivity and two point discrimination thresholds obtained at four oral-facial sites are reported for 41 patients with cerebral vascular damage, and for 24 normal subjects. Although there is bilateral sensory representation for the face and mouth in each hemisphere, significantly more contralateral than bilateral oral-facial sensory deficits were found after unilateral damage. Despite the previously reported relationship between left hemisphere damage and impairments in the production of both verbal and nonverbal oral movement, there appear to be no differential effects of left or right hemisphere lesions on the degree or pattern of oral-facial sensory deficits. In the normal control group, elderly subjects demonstrated higher two point discrimination thresholds, but not pressure sensitivity thresholds, than young subjects.

Oral-facial sensory impairments are often cited as indicators in the diagnosis of nervous system pathology. However, very little data is actually available on either normal oral-facial sensory capacities, particularly in older subjects, or on the degree and nature of impairment associated with different forms and sites of neurological involvement.

Extensive bilateral representation of oral-facial structures in thalamo-cortical sensory pathways and cortex has been well documented (see review after Results). This pattern is in marked contrast to the primarily contralateral sensory representation of the hands. Therefore, one might expect the incidence and patterns of oral-facial sensory deficit to be quite different from that seen for hand sites, the area tested most extensively in a sensory examination. Several authors have warned against using the side of the body ipsilateral to a lesion as a baseline of comparison for the contralateral side. The argument for using, instead, a comparison with normal persons is even more compelling in evaluating oral-facial sensory function, given the bilateral organization of these areas.

There has been disagreement in the literature with regard to hemispheric asymmetries in the processing of sensory information. Sporadic reports of bilateral sensory loss after unilateral cerebral lesions have appeared for over 100 years (see Corkin [1], for review). Semmes, Weinstein, Ghent and Teuber [2], in the first systematic study in this area, reported that bilateral sensory

loss in the hands occurred quite frequently after unilateral hemispheric damage and was more often related to left hemisphere damage than to right in subjects with penetrating brain wounds. Other studies have not supported such a differential effect of right and left lesions on sensory threshold data [3,4,5]. Although all studies revealed some incidents of sensory loss on the hand ipsilateral to the side of lesion, these deficits occurred as often with right as with left hemisphere damage. At least two variables, sensory measure and type of lesion may be operating here. Carmon and Dyson [4] report a high incidence of ipsilateral deficit on two-point discrimination in patients with unilateral cerebral damage of tumor, vascular and traumatic origin. Corkin, Milner and Taylor [5] report ipsilateral deficits particularly on point localization in patients with discrete cortical excisions. Comparable studies have not been conducted which focus on the oral-facial structures. Given the apparent salience of the left hemisphere in the control of nonverbal as well as verbal oral motor function, recently suggested by Mateer and Kimura [6], a greater role of the left hemisphere in mediating oral sensory function might be predicted.

Conflicting reports have also appeared regarding asymmetries in normal subjects in pressure sensitivity and two point discrimination thresholds. Semmes, Weinstein, Ghent and Teuber [2] report significant but small differences in pressure sensitivity for the hands in the direction of greater left-sided sensitivity. Wein-

stein [7] reports a similar finding for the index finger but not for the cheek, upper lip, or 17 other body parts tested. Other investigators [8,9], using similar procedures, failed to find such left-right differences in pressure sensitivity thresholds. A consistent laterality difference seems just as elusive when two point discrimination data are considered. Weinstein [7,10] failed to find consistent left-right differences in normal subjects across a broad range of body parts, including cheek and upper-lip sites. Lass, Kotchek and Deem [11] and McCall and Cunningham [12] reported that two point discrimination thresholds in various oral structures were higher on the right in some subjects, on the left in others and were equal in the remaining. In an analysis of group data, Ringel and Ewanowski [13] noted no consistent patterns among two point discrimination thresholds at various oral sites, except that midline regions were more discriminate than either right or left sides.

In this study, pressure sensitivity and two point discrimination thresholds were determined at four oral-facial sites on left and right sides in control subjects and in patients with unilateral cerebral lesions. The purpose was to establish normative oral-facial sensory data applicable to an older patient population and to investigate the incidence and pattern of oral-facial sensory impairment after hemispheric damage. In view of the obviously greater role of the left than the right hemisphere in the control of verbal (speech) as well as nonverbal oral movements, oral sen-

sory function may be differentially impaired in left and right hemisphere damaged patients.

METHOD

Subjects

Subjects were 15 patients (7 males, 8 females) with right hemisphere damage and 26 patients (19 males, 7 females) with left hemisphere damage admitted to the University Hospital, London, Ontario. The patients were selected on the basis of evidence of strictly unilateral vascular damage to one hemisphere of the brain. All but four of the patients showed positive signs of unilateral cerebral involvement on at least one of an electroencephalogram, brain scan or angiogram. The remaining four demonstrated clear clinical signs of hemiplegia and/or visual field defect. Any patient with evidence of bilateral damage, or with a peripheral neuropathy as judged by the neurological exam was excluded from the study. There were no significant differences between the left and right hemisphere damaged groups in the incidence of hemiparesis (80% in the left versus 88% in the right hemisphere damaged group) or in the incidence of visual field defect (31% in the left versus 38% in the right hemisphere damaged group). Approximately one half the patients in each group was tested less than eight weeks after onset of neurological symptoms. Thirteen of the 26 left hemisphere damaged patients were designated aphasic on the basis of a combination of expressive and receptive speech tasks. Ex-

tremely inconsistent responding by 3 of these aphasic patients precluded reliable determination of sensory thresholds. All tests were also administered to 24 control subjects having no history of neurological disease. These were volunteers from a senior citizens community centre, and students from the university community. Analysis of variance yielded no significant differences between the ages of the right hemisphere damaged group (mean = 47.4 years), the left hemisphere damaged group (53.2 years) and the control group (48.4 years), [F = 0.669, df = 59, NS]. All subjects, patients and controls, were right-handed.

Procedure

Pressure thresholds

Pressure thresholds were obtained with a Semmes Weinstein Pressure Aesthesiometer (Research Media, Model PR-11), which consisted of twenty nylon filaments, equal in length but varying in diameter, each implanted at one end of a plastic rod. The filaments are assigned a value corresponding to the common logarithm (expressed as log units) of the force required to bend it maximally by pressing against the tip. The values ranged from 1.65 (most sensitive) to 6.65 (least sensitive). The subject was instructed to raise the hand ipsilateral to the lesion immediately upon feeling something touch him. All subjects were blindfolded to eliminate visual cues from movements of the experimenter. Using the method of limits, one descending series and one ascending series were administered, the threshold estimate being the

mean value of the last filament perceived in the descending series and the first perceived in the ascending series. Each stimulation lasted approximately one second, the interval between stimulations being varied to facilitate detection of false positive responses.

Four oral-facial sites were tested; the upper lip, the lower lip, the tongue and the cheek (Figure 1). Testing of the lip sites was performed in the pigmented region midway between the vermilion border and the orifice of the mouth, 5.0 mm to the left and right of midline. Both upper and lower lip sites were used as test sites as they are innervated by different branches of the trigeminal nerve. Measurements on the tongue were made on the anterior dorsum (blade) at a point 5.0 mm posterior to the tongue tip, 3.0 mm lateral of midline. Buccal thresholds were obtained in the horizontal plane 5.0 mm lateral to the mouth edge. The order of facial sites tested was constant for all subjects: cheeks, upper lip; lower lip, and tongue. At each site, the side ipsilateral to the presumed lesion was tested first for all patients. Order of testing right and left sides was counterbalanced in control groups.

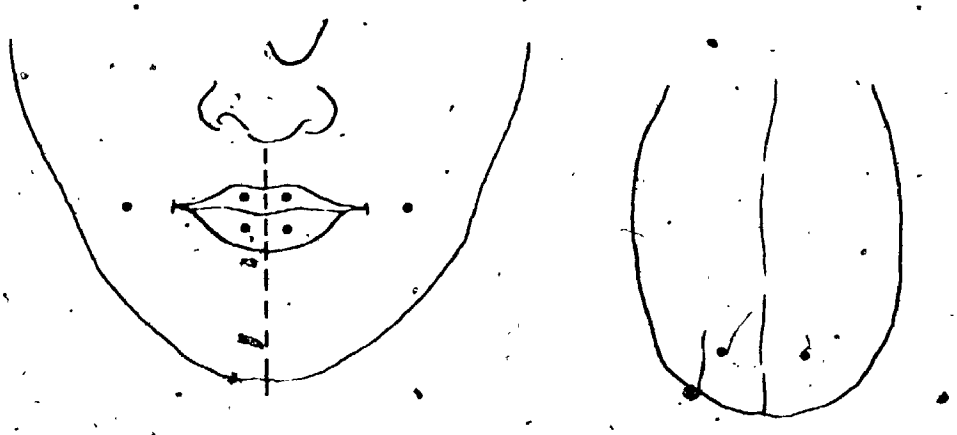
(Insert Figure 1 about here)

Two point discrimination

Two point discrimination thresholds were determined for the same four oral-facial sites, in the transverse plane, using an aesthesiometer (Lafayette Instrument). Subjects were asked to indicate whether one or two points had been applied by raising either

Figure 1

The dots on the facial and lingual schemata indicate the site at which pressure sensitivity thresholds were obtained. Two point discrimination thresholds were obtained in a transverse plane using the same site as the point midline between the two stimulation points.



one or two fingers of the hand ipsilateral to the lesion. Each trial consisted of four randomly presented episodes of stimulation, two single point applications and two applications of two points separated by a certain distance. The trials began with a separation between the points well above thresholds for discriminating them and decreased in 0.3 mm increments until a criterion level of 75% (3 out of 4) correct judgements was not met. An ascending series, begun with a point separation well below threshold, proceeded with increasing increments of separation until the 75% correct criterion level was met. The two point threshold at each site was the mean of the last descending and ascending value at which criterion was reached.

Double simultaneous stimulation

The object of double simultaneous stimulation is to detect sensation patterns in which each of two stimuli applied to homologous body parts would be reported if each were applied alone but in which only one of the two stimuli is reported when both are applied together. Such failures to report have been designated as extinction, neglect or inattention [14]. In the task, two wooden sticks were used to apply supraliminal pressure at the selected oral facial sites, cheeks, upper lip, lower lip and tongue, for a one second interval. At each site, single left and right sided stimulation trials were randomly interspersed with two bilateral stimulation trials. Thus, eight bilateral stimulation trials were presented. The blindfolded subject was asked to point to all

points of stimulation and responses were recorded for each trial.

RESULTS

Patterns of sensitivity in the control group

Since previous investigators had determined oral-facial sensory thresholds only in relatively young persons, it was appropriate to determine the effect of inclusion of older subjects on the resultant means. The control subject group was divided into two groups with no age overlap, one with a mean of 24.3 years (N = 12) and one with a mean age of 70.9 years (N = 12). In addition to age (young/old), the variables of side of testing (left/right, repeated measure) and anatomical site tested (four oral-facial sites, repeated measure) were used in an analysis of variance to evaluate the results of both sensory measures.

Pressure sensitivity thresholds. Analysis of the pressure sensitivity thresholds did not support any significant differences among the four oral-facial sites tested [F, site = 2.923, df = 3,66, NS], between the left and right sides [F, side = 0:540, df = 1,22, NS], or between the two age groups [F, age = 0.026, df = 1,22 NS]. Since there were no significant left/right differences, threshold values were collapsed over side tested at each site. The mean pressure sensitivity thresholds for control subjects in the two age groups are given in Table I.

Two point discrimination thresholds. The two point discrimination data (Table 1) also did not support any significant difference between left and right sides [F, side = 0.736, df = 1,22, NS]. There were, however, significant effects of both site [F, site = 188.140, df = 3,66, $p<.01$] and age [F, age = 13.435, df = 1,22, $p<.01$]. While there were no significant differences between the lingual and labial sites, the two point discrimination threshold for the cheek was significantly greater than for any other oral-facial site [$p<.005$ for each] and, in fact, was invariably at least three times as great. Older subjects demonstrated significantly higher two point discrimination thresholds than younger subjects at all oral-facial sites tested [$p<.01$ for each].

(Insert Table 1 about here)

Patterns of sensitivity in patient groups

A three way analysis of variance using the variables of side of lesion (left or right), side of structure tested with reference to the side of the presumed lesion (contralateral or ipsilateral, repeated measure) and anatomical site of testing (four sites, repeated measure) was performed for threshold values obtained on both sensory measures.

Pressure sensitivity thresholds. Analysis of the pressure sensitivity data (Table 2) revealed no effect of side of lesion [$F = 2.24$, df = 1,36, NS]. There were, however, main effects of both side of testing and site, and a significant interaction between these two variables [F, side of testing x site = 6.95, df = 3,108,

TABLE 1

Mean Thresholds for Two Age Groups of Normal Control Subjects
Collapsed Over Left and Right Sides

	Pressure Sensitivity (Log Units)		Two Point Discrimination (mm)	
	Young ($\bar{X}$=24.3 years, N=12)	Old ($\bar{X}$=70.9 years, N=12)	Young ($\bar{X}$=24.3 years, N=12)	Old ($\bar{X}$=70.9 years, N=12)
Upper Lip	2.43	2.33	3.71	4.76
Lower Lip	2.44	2.38	3.44	4.63
Tongue	2.53	2.62	3.64	5.42
Cheek	2.43	2.58	14.09	17.27

Pressure sensitivity:

F, side = 0.540, df = 1,22, NS
F, site = 2.923, df = 3,66, NS
F, age = 0.026, df = 1,22, NS

Two point discrimination:

F, side = 0.836, df = 1,22, NS
F, site = 188.140, df = 3,66, $p<.01$
F, age = 13.435, df = 1,22, $p<.01$

$p<.001$]. Multiple comparison tests (Newman-Keuls) revealed that mean threshold values were higher on the side contralateral to the lesion than on the side ipsilateral to the lesion at all oral-facial sites ($p<.01$ for each). Thresholds for the contralateral tongue and cheek sites were significantly greater than for the contralateral upper and lower lips ($p<.01$), while there were no differences between the pressure sensitivity thresholds across all four sites ipsilaterally. The mean thresholds for controls given in Table 2 are collapsed over left and right sides and age groups.

(Insert Table 2 about here)

Two point discrimination thresholds. Analysis of the two point discrimination data (Table 3) also revealed no significant effect of side of lesion on the threshold values [$F = 0.53$, $df = 1,36$, NS]. Again, in addition to significant main effects of side and site of testing, there was a significant interaction between the two variables [F, side of testing x site $= 15.59$, $df = 3,108$, $p<.001$]. Mean thresholds for contralateral structures were significantly higher than those for ipsilateral structures across all four oral-facial sites tested ($p<.01$ for each site). When either contralateral or ipsilateral measures were considered, only the two point discrimination value for the cheek site differed significantly from the other sites tested. Consistent with the main effect of site in the control subject data, cheek thresholds were several times higher than lip or tongue thresholds, which did not

TABLE 2

Mean Pressure Sensitivity Thresholds (Log Units)

Site	Left Lesion		Right Lesion		Controls L-R Mean
	Contra	Ipsi	Contra	Ipsi	
Upper Lip	2.90	2.51	2.64	2.45	2.39
Lower Lip	2.87	2.54	2.60	2.29	2.41
Tongue	2.37	2.91	3.28	2.56	2.60
Cheek	3.62	2.68	3.14	2.26	2.51

F, side of lesion = 2.24, df = 1,36, NS

F, side of testing (contra/ipsi) x site = 6.95, df = 3,108, p .001

Contra threshold > Ipsi threshold at all sites.
Ipsilateral thresholds are not significantly different across sites.
Contralateral tongue and cheek thresholds > contralateral lip thresholds.

differ significantly from each other.

(Insert Table 3 about here)

Incidence of pressure sensitivity and two point discrimination deficits in patient groups. Because an elevated mean threshold value does not speak directly to the actual incidence of deficit, the thresholds of the control subjects were used to set up quantitative criteria for defining sensory deficits. Using the procedure of Semmes, Weinstein, Ghent and Teuber [2], both pressure sensitivity and two point discrimination deficits were defined as a significant deviation from the mean threshold of the control group. The t value appropriate to a one-tailed test with 24 degrees of freedom, 2.492, was obtained from the t-distribution. The product of this value and the standard deviation was added to the mean threshold value at each site to determine the lower limits of a sensory deficit corresponding to 0.01 level of significance for each of the two tests (Table 4).

(Insert Table 4 about here)

Using this criterion for determination of deficit, the number of patients in each group who demonstrated impairment was determined for all sites, contralaterally and ipsilaterally. Tables 5 and 6 give the incidence, corresponding percentage of the group it represents and the mean values of the elevated thresholds. Although the percentage of left hemisphere damaged patients demonstrating ipsilateral deficits was higher than right hemisphere

TABLE 3

Mean Two-Point Discrimination Thresholds (mm)

Site	Left Lesion		Right Lesion		Controls L-R Mean
	Contra	Ipsi	Contra	Ipsi	
Upper Lip	7.67	4.50	7.52	4.13	4.29
Lower Lip	6.90	4.71	7.84	4.24	4.13
Tongue	8.43	5.19	8.78	5.07	4.61
Cheek	27.34	17.67	27.53	14.72	16.67

F, side of lesion = 0.53, df = 1,36, NS

F, side of testing (contra/ipsi) x site = 15.59, df = 3,108, $p<.001$

Contra threshold > Ipsi threshold at all sites:
Contra and Ipsi cheek threshold > thresholds at all other sites.

TABLE 4

Significantly Elevated Threshold Values Derived from Normative Data of 24 Controls

	Pressure Sensitivity (Log Units)			Two Point Discrimination (mm)		
	Control L/R Mean	δ	$\bar{X} + (2.492)\delta$*	Control L/R Mean	δ	$\bar{X} + (2.492)\delta$*
Upper Lip	2.39	0.36	3.29	4.29	1.14	7.13
Lower Lip	2.41	0.39	3.38	4.13	1.17	7.04
Tongue	2.60	0.38	3.55	4.61	1.48	8.30
Cheek	2.51	0.43	3.58	16.67	3.29	24.87

*The numbers in this column were subsequently used as cut off thresholds. Patients having thresholds above these values were considered to show a significant sensory deficit on the particular task and site. The constant, 2.492, is the t value which corresponds to a 0.01 level of significance for a one-tailed test (degrees of freedom = 24).

damaged patients in all cases, chi-square analyses did not support a statistically significant difference between left and right hemisphere damaged groups in the incidence of either contralateral or ipsilateral deficits at any site tested, consistent with results of the overall analysis of variance. This was true for measures of both pressure sensitivity and two point discrimination.

In order to investigate whether pressure sensitivity or two point discrimination deficits, when they occurred, were more severe in left or right hemisphere damaged subjects, the elevated discrimination values were compared across the two groups. No significant differences in magnitude of deficit were found between the groups at any of the sites tested (see Tables 5 and 6). Ipsilateral deficits were seen only on tongue and cheek sites for pressure sensitivity thresholds, but were seen at all sites for two point discrimination thresholds.

(Insert Tables 5 and 6 about here)

One might argue that instances of apparent ipsilateral deficit on either task could be accounted for by aphasic involvement, either in terms of failure to comprehend the task or an inability to respond appropriately. This notion was not supported. Four of the instances of markedly increased pressure sensitivity bilaterally and four of the cases of markedly increased two point discrimination thresholds bilaterally were seen in nonaphasic left hemisphere damaged patients or right hemisphere damaged patients. Although the remaining bilateral sensory deficits were seen in six

TABLE 5

Pressure Sensitivity: Incidence of Contralateral and Ipsilateral Defects

Site	Lesion Group											
	Left N=23						Right N=15					
	Contralateral			Ipsilateral			Contralateral			Ipsilateral		
	N	%	$\bar{X}$*	N	%	$\bar{X}$	N	%	$\bar{X}$	N	%	$\bar{X}$
Upper Lip	6	26	4.15	0	0	-	2	13	4.01	0	0	-
Lower Lip	5	22	4.26	0	0	-	1	7	3.84	0	0	-
Tongue	11	48	4.25	6	26	3.61	6	40	4.34	2	13	3.72
Cheek	10	43	5.08	3	13	3.61	6	40	4.18	0	0	0

*Mean of significantly elevated threshold values.

TABLE 6

Two Point Discrimination: Incidence of Contralateral and Ipsilateral Defects

Site	Lesion Group											
	Left N=23						Right N=15					
	Contralateral			Ipsilateral			Contralateral			Ipsilateral		
	N	%	$\bar{X}$*	N	%	$\bar{X}$	N	%	$\bar{X}$	N	%	$\bar{X}$
Upper Lip	5	22	17.15	1	4	7.95	5	33	13.08	0	0	-
Lower Lip	8	35	12.47	3	13	7.95	4	27	16.28	0	0	-
Tongue	9	39	13.92	2	9	9.53	6	40	15.05	1	7	9.25
Cheek	9	39	46.39	4	17	34.10	7	47	51.69	0	0	0

*Mean of significantly elevated threshold values.

of the left hemisphere damaged patients with aphasia, in every instance a threshold value within normal limits was obtained for at least one of the sites, suggesting adequate comprehension of task instructions and effective response capabilities.

It might also be argued that those patients demonstrating ipsilateral as well as contralateral sensory deficits actually have undetected bilateral hemispheric damage. In order to investigate this possibility, those patients with ipsilateral sensory deficits were compared with those without such deficits on an independent measure, grip strength of the ipsilateral hand determined with a dynamometer. The difference on this measure was not significant and was not in the predicted direction if bilateral deficits are hypothesized (mean ipsilateral hand grip strength of patients with ipsilateral oral-facial sensory deficits = 35.78 Kg, mean of those without ipsilateral sensory deficit = 29.48 Kg [$t = 1.76$, $df = 36$, NS]).

Despite no obvious differences in the overall incidence of deficit on each of the two sensory measures, the relationship between the two measures, within patients, is of interest. Forty of the 47 cases of pressure sensitivity deficit (85%) were associated with a two point discrimination deficit at the same site while only 18 of the 53 cases of two point discrimination deficit (34%) were associated with a pressure sensitivity deficit.

Results of the double simultaneous task. No control subject ever failed to report that both sides were touched on bilateral stimulation trials. Because this task was introduced later in the investigation, results are available for only 15 right hemisphere damaged cases and 19 of the left hemisphere damaged cases. Six right hemisphere damaged patients failed to identify the contralateral stimuli on bilateral stimulation at least four out of eight times. Two of these never reported the contralateral side on bilateral stimulation accurately. Only one left hemisphere damaged patient demonstrated a failure to report bilateral stimulation in the presence of accurate single stimuli identification. This difference in incidence between the left and right hemisphere damaged groups is significant at the 0.025 level ($X^2 = 6.19$, df = 1). One of the cases in the right hemisphere damaged group who failed to identify contralateral stimulation when bilateral stimulation was applied, failed to report two points at any degree of separation on the two point discrimination task at any site on either side. His results are not included in the two point discrimination analysis.

ANATOMICAL ORGANIZATION

There is a large body of anatomical, neurophysiological and behavioral evidence confirming bilateral sensory representation of the face and oral structures. The trigeminal (cranial nerve V) sensory root, formed by fibers from the skin of the face and oral

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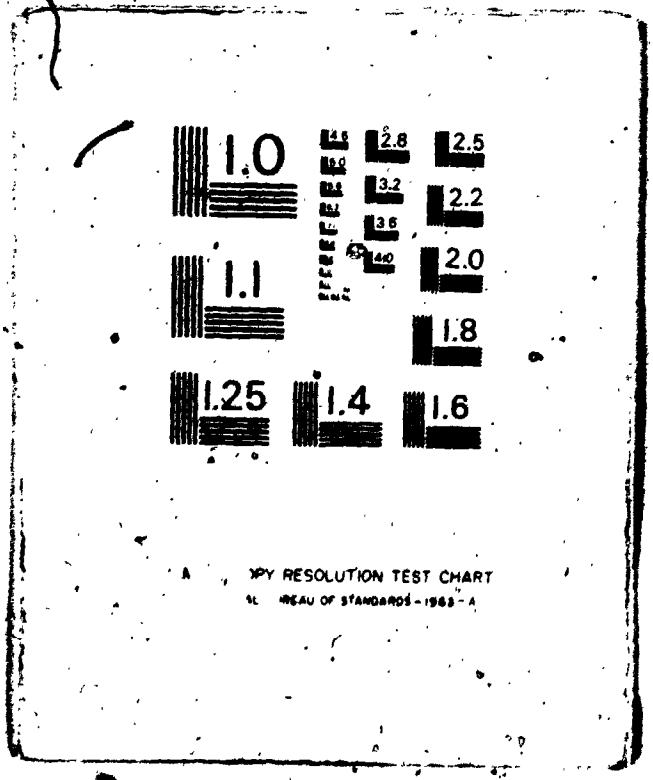
1.0
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mucosa, enters the pontine tegmentum where the root divides into short ascending fibers terminating in the main sensory nucleus and long descending fibers comprising the spinal trigeminal tract. (See Figure 2). Clinical evidence suggests that pain and temperature sense are handled entirely by the spinal N. V tract and its contralateral thalamic projections via the ventral secondary ascending tract. Light touch and two point discrimination are primarily mediated by the main sensory nucleus [15]. Secondary fibers from this nucleus ascend, after partial decussation, as the dorsal secondary ascending tract of N. V, to terminate in the ventral posteromedial nucleus of the thalamus. Tertiary fibers from this nucleus project via the internal capsule and corona radiata to the inferior postcentral gyrus of the cerebral cortex and from there to other cortical areas [16]. Although often represented as a crossed tract, degeneration studies in the cat and monkey have presented evidence that fibers from the main sensory nucleus ascend bilaterally in the dorsal central trigeminal tracts [17,18, 19].

In addition to direct ipsilateral connections, there is anatomical and electrophysiological evidence for interhemispheric connections between the proximal limb, trunk and head regions (including the face and mouth areas) of primary somatosensory cortex, via the corpus callosum in cats, racoons and monkeys [20,21]. Similar callosal connections have not been demonstrated between cortical areas having sensory representation for the hands, feet or

digits.

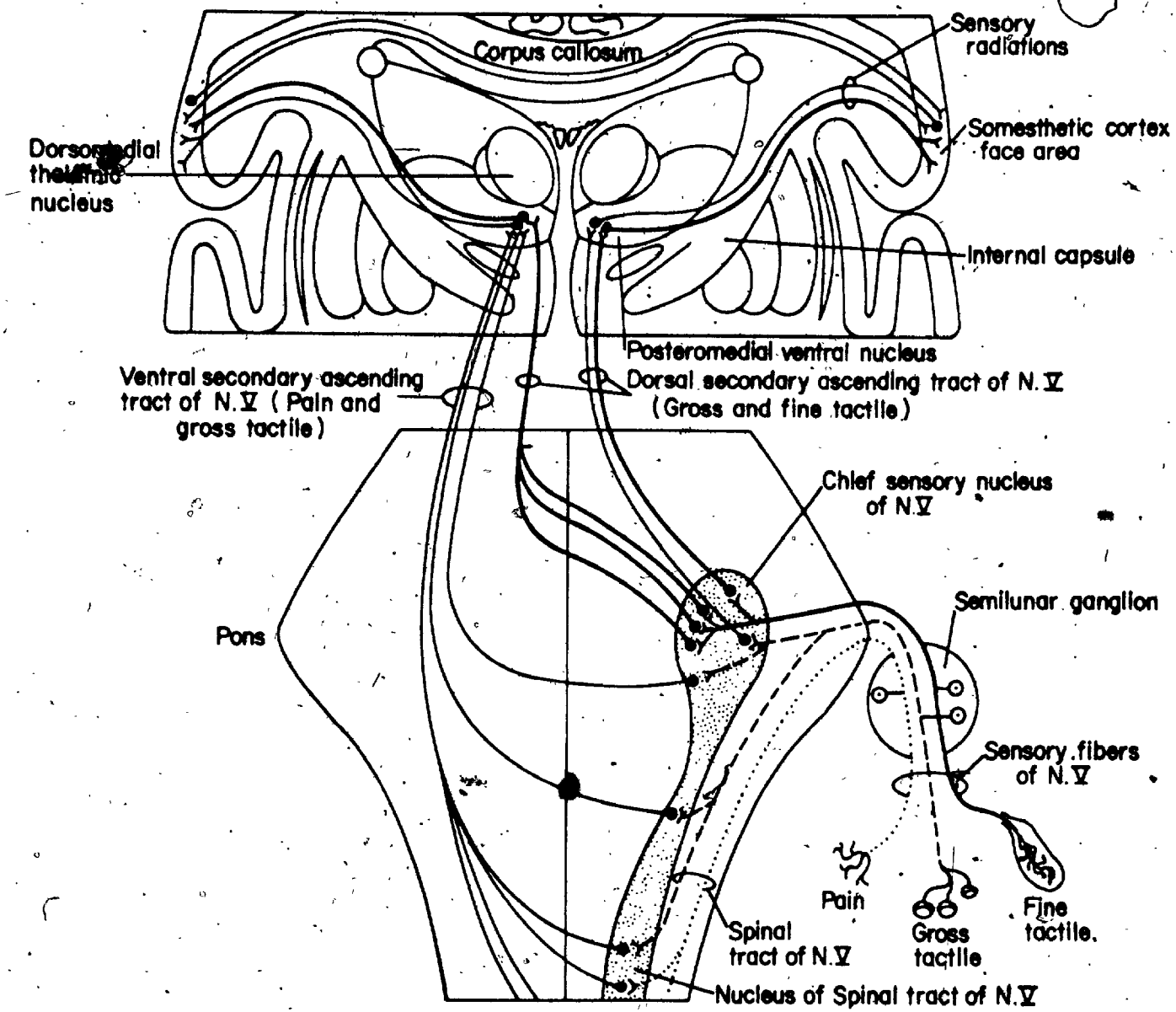
(Insert Figure 2 about here)

Somesthetic function has been studied in patients who have undergone sections of the corpus callosum, a procedure which presumably prevents transfer of somesthetic input. If such a patient is able to cross-localize, that is, use the left hand to touch a point stimulated on the right side of the body, and vice versa, the functional use of ipsilateral projection systems can be demonstrated. Gazzaniga, Bogen and Sperry [22] discuss a commissurotomized patient who was able to cross-localize with stimulation points on the head and face, but not on other body parts.

Electrical stimulation of the primary sensory cortex in man has been associated with the report of sensory experiences. Contralateral, bilateral and ipsilateral sensations have been reported after postcentral face area stimulation, whereas stimulation of all other body areas resulted in strictly contralateral effects [23,24]. In addition, bilateral, contralateral and ipsilateral responses for non-facial as well as facial body parts have been obtained after stimulation of a second sensory area on the superior border and bank of the sylvian fissure in both hemispheres. A secondary sensory area in this region in animals, from which responses to bilateral stimulation can be recorded, has been reported to receive input from the primary sensory area (SI) on the same side, and from the contralateral primary and secondary areas via the corpus callosum [25]. Van Buren and Baldwin [26] reported

Figure 2

Diagram to illustrate the projections of exteroceptive impulses from the face carried over the somatic sensory branches of the trigeminal nerve. Note that impulses set up by fine tactile stimuli (as in pressure sensitivity and two point discriminations) are transmitted by the main sensory nucleus which has bilateral thalamic projections via the dorsal secondary ascending tracts of N. V. Also note callosal connections between the primary face area of each somatosensory cortex. (Adapted from Fig. 144, Chapt. 3, Crosby, E.C., Humphrey, T. and Lauer, E.W., Correlative anatomy of the nervous system, MacMillan, New York, 1962.)


Corpus callosum
Sensory radiations
Somesthetic cortex face area
Dorsomedial thalamic nucleus
Internal capsule
Posteromedial ventral nucleus
Ventral secondary ascending tract of N.V (Pain and gross tactile)
Dorsal secondary ascending tract of N.V (Gross and fine tactile)
Chief sensory nucleus of N.V
Semilunar ganglion
Pons
Sensory fibers of N.V
Pain
Gross tactile
Fine tactile.
Spinal tract of N.V
Nucleus of Spinal tract of N.V

the incidence, laterality and quality of sensations about the mouth, face and head-neck regions elicited by cortical stimulation in man. Crude sensation was most often referred to the mouth (predominantly contralaterally) after stimulation of the lowest 3 to 4 cm of the postcentral area. Sensation after temporal lobe stimulation was most often referred to the ipsilateral hand and neck region followed in frequency by bilateral referral. Some sensations at the contralateral mouth and face area were also reported. Precentral sensory responses were less common but often characterized by sensation of head and mouth movement contralaterally or bilaterally.

DISCUSSION

Effects of age

The tabled threshold values on the pressure sensitivity and two point discrimination measures in control subjects should provide useful normative data applicable to older neurological populations. It was felt that the slightly higher thresholds on the two point discrimination task in comparison to values previously cited in the literature [13] might be due to the inclusion of non-neurological control subjects up to 70 years of age. A significant effect of age on this task was confirmed statistically. This was in contrast to a lack of significant effect of age on the

pressure sensitivity task. MacDonald and August [27] also reported a decrease in accuracy on an oral form recognition task in a geriatric population as compared to young adults. Age certainly appears to interact with some measures of discriminative sensibility of the face and mouth. The normative values obtained in this study thus provide conservative estimates of deficit and are appropriate to many patient populations.

Sensory threshold patterns in normal population

There was no support in the normal control data for left/right differences in sensitivity, since left and right thresholds are not significantly different at any of the oral-facial sites on either of the sensory measures. The discrepancy between these results and those of previous investigators who reportedly have found stable left/right differences, [7,28] may reflect a real difference in sensory laterality effects at oral-facial versus other body part sites. Other investigators have also used multiple ascending and descending trials in the determination of thresholds. Such procedures are difficult to use when normative data on oral-facial sites applicable to neurologically impaired patients is of interest, but may be instrumental in detecting what are usually the small left/right differences reported for normal subjects.

There were no significant differences between the pressure sensitivity thresholds obtained at any of the oral-facial sites tested. In contrast to this homogeneity of pressure sensitivity thresholds, two point discrimination thresholds differed signifi-

cantly across test sites. While the thresholds were equal at the lip and tongue sites, the threshold at the cheek was several times higher. Other investigators [13] have found the tongue to be significantly more sensitive on this task than other oral sites. Failure to find such an increased lingual sensitivity in this study can be explained by the data of Lass, Kotchek and Deem [11] which strongly supports an increased sensitivity on the tongue tip as compared to the tongue dorsum, the lingual site used in this study. In contrast to the finding of increased thresholds on the cheeks as compared to lips and tongue in this study, Weinstein [7] did not find a significant difference between the upper lip and cheek on the two point discrimination task. Two methodology differences may relate to the discrepant results. First, Weinstein reports using the nonpigmented upper lip area rather than the pigmented as used in this study, and secondly, his thresholds were determined along the longitudinal rather than the transverse axis. Ladd and Woodworth [29] suggest that sensitivity on two point discrimination tasks may be greater in the transverse than in a longitudinal axis on both arms and legs.

The significant effect of site on the two point discrimination thresholds, but not on the pressure sensitivity threshold, demonstrates the lack of consistent correspondence between these two measures. Weinstein [7] reported a very low nonsignificant correlation between pressure sensitivity and two point discrimination in normal subjects across a broad range of body sites tested.

He suggests that the correlation between a measure of size of somatosensory cortex subserving a body part and the sensitivity threshold at that body part is highly significant for two point discrimination data but nonsignificant for pressure sensitivity data. With respect to the sites used in this study, the cheek certainly appears to be related to a much smaller area on the sensory homunculus than are the lips and tongue [30] and is the site at which significantly higher two point but not pressure sensitivity thresholds were found.

Effects of lesions

Corkin, Milner and Rasmussen [31] reported convincing evidence relating sensory deficits on the hands to inclusion of the postcentral gyrus in cortical excisions for focal epilepsy. While contralateral two point discrimination deficits on the palm were always related to excisions invading postcentral gyrus, pressure sensitivity deficits, although most common after postcentral invasion, were also seen after frontal, temporal and more posterior parietal excisions. Ruch [32] argues that thalamic relays are also important in integrating patterns of touch sensation before projection to the somatosensory cortex for two point discrimination. What is suggested here is that discrimination between two points is related more specifically to thalamo-cortical function than is appreciation of light touch, which may be more diffusely represented.

The data presented in this study do not at first inspection suggest any overall differences in the incidence of contralateral pressure sensitivity or two point discrimination deficit at any site. If, however, the relationship between the two kinds of deficit is considered, 85% of the instances of pressure sensitivity deficit are associated with a two point discrimination deficit at the same site whereas only 34% of the instances of elevated two point thresholds are associated with a pressure sensitivity decrement. Apparently, either deficit can occur in isolation, that is, without the other, but two point discrimination is more often selectively impaired. Corkin, Milner and Rasmussen [31] found that pressure sensitivity deficits on the hands associated with non-postcentral gyrus excisions were not as severe as the pressure sensitivity deficits seen after postcentral gyrus excision and most often were not associated with two point discrimination deficits. In this study, too, the values of the pressure sensitivity deficits which were not associated with two point discrimination deficits were always lower than the mean of the elevated pressure sensitivity thresholds. Several neural models of two point discrimination incorporate an element of intensity discrimination as a basis for the spatial discrimination [32]. It might be postulated, then, that damage to somatosensory cortex could selectively impair two point discrimination while more diffuse cortical and subcortical damage could selectively impair appreciation of light touch. If the deficit in pressure sensitivity was great enough, there might be insufficient information, perhaps less than some

minimal level, for effective two point discrimination. This model would provide an explanation for the findings of isolated deficits on either task, yet higher incidence of two point discrimination deficits given a pressure sensitivity deficit. The vascular etiologies of these cases preclude accurate determination of site or extent of damage but variable size lesions of both cortical and subcortical structures must be postulated.

Overall the incidence of ipsilateral deficit was quite high, 44% of the patients demonstrating an ipsilateral deficit on at least one site for at least one of the tasks, 60% a contralateral deficit. It might be argued that the instances of ipsilateral deficit are related to undetected bilateral hemispheric damage. The finding of equivalent performance on an independent motor test of grip strength on the ipsilateral hand for patients with and without ipsilateral oral-facial sensory deficits, however, argues against more bilateral impairment in the former group. There was also no suggestion in the data that bilateral deficits are actually due to aphasic involvement, although it is true that in some instances aphasic impairment can preclude reliable testing of sensory function.

In every case patients demonstrating an ipsilateral deficit had a contralateral deficit as great or greater than the ipsilateral one. Thus, ipsilateral deficits are functionally synonymous with bilateral impairment. This suggests that discrete areas or tracts are not devoted strictly to ipsilateral information but rather that

this information is carried with afferent contralateral information at cortical and probably thalamic levels. The higher incidence of contralateral deficit also suggests that although oral-facial sensory representation may be bilaterally organized, the contralateral connections are more salient.

Pressure sensitivity thresholds are more often severely elevated contralaterally at the tongue and cheek sites than at the upper and lower lip. The lingual and buccal sites were also the only ones involved in cases of bilateral pressure sensitivity deficits. Although some bilateral two point discrimination deficits were seen at all sites tested, the highest percentages of contralateral deficits on this task were also on the tongue and cheek. The lips appear to have a larger area of representation on the somatosensory cortex than the tongue or cheek, so that small vascular lesions might less often produce a critical extent of damage for these sites. Thus, the tongue and cheek sites appear to be more discriminating sites for the detection of sensory deficits.

There was no support in the patient data for different effects of right versus left hemisphere lesions on the incidence, severity or pattern of sensory deficits. Although left hemisphere lesion cases contribute a higher proportion of the cases of bilateral sensory loss, the difference did not reach statistical significance. Consistent vascular etiologies and equivalent proportions of hemiparesis and visual field defects in the right and left hemisphere damaged groups would suggest that lesion size and site is

similar.

All of the patients used in the study were also given a broad range of both verbal and nonverbal motor tasks, the results of which have previously been reported [6]. The highly significant association between left hemisphere damage and impairment on both the verbal (speech) and nonverbal oral movement tasks stands in marked contrast to the nonsignificant effect of left versus right hemisphere lesions on the incidence or pattern or oral-facial sensory deficits. Whatever oral motor functions are being disrupted by left hemisphere damage, they do not seem to be ones primarily under the guidance of or dependent on external tactile feedback.

The only measure on which side of lesion was a significant factor was the double simultaneous stimulation task. Significantly more right hemisphere damaged patients fail to identify contralateral stimulation on bilateral stimulation than do left hemisphere damaged patients. This probably reflects a generalized multimodal neglect or inattention factor often cited in association with right parietal lobe damage [14] rather than differences in oral-facial sensory organization per se.

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PAPER NO. 4

APHASIA AND ORAL APRAXIA - A REVIEW OF THE LITERATURE AND IMPLICATIONS FOR HEMISPHERIC SPECIALIZATION

Hughlings Jackson (1878) is usually credited with the first observations linking an impairment in nonverbal oral movement with speech disorders, or aphasias. He described a patient who was unable to protrude his tongue on command, although at other times spontaneous licking movements occurred. Such an inability to produce an oral movement, not due to paralysis of the oral musculature, has been termed oral apraxia.

Liepmann (1913) described apraxia as an impairment in the production of acquired movements of the hands and mouth. Just as hand and arm movements used in the handling of objects are acquired, movements of the oral musculature used in the production of speech are acquired. In grouping impairments of acquired movement under the term apraxia, Liepmann suggested that "aphasia (not only motor aphasia, but every expressive-aphasic disturbance of speech and writing) and apraxia are essentially similar". That is, they result from the same kind of underlying deficit in motor control.

Despite these early and clearly articulated associations between disordered oral movement and disordered speech, subsequent research has done little to clarify the nature of the relationship. The purposes of this paper are: 1) to discuss problems in the investigation of speech and non-speech oral motor impairments, and 2) to review the evidence for a relationship between such impairments. The conclusions lend support to the hypothesis that a fun-

damental function of the left hemisphere is the control of certain kinds of complex movements, (Liepmann, 1913; Kimura, 1974). Although some complex movements, i.e. speech, have acquired symbolic, representational functions, such functions do not appear to be the underlying basis of left hemisphere specialization.

PROBLEMS IN INVESTIGATION OF SPEECH AND NON-SPEECH ORAL IMPAIRMENTS

Problems in Describing and Interpreting Speech Disturbances

There are many terminological problems in reviewing the literature of aphasia. A large number of labels have been applied to disturbances of speech behavior, most of which are poorly defined and used differently by various authors.

All errors in aphasic speech can, at some level, be considered as errors in the selection, production and ordering of speech sounds according to appropriate patterns. A patient may have difficulty producing even a single phoneme or may demonstrate many errors in phoneme selection during speech attempts. Such "phonemic" disorders of speech production have been variously termed verbal apraxia (Canter, 1967), apraxia of speech (Johns and Darley, 1970), phonetic disintegration (Alajuanine and Lhermitte, 1964), anarthria (Critchley, 1952), apraxic dysarthria (Nathan, 1947), motor aphasia (Liepmann, 1913), cortical dysarthria (Bay, 1965), sensorimotor impairment (Schuell; Jenkins and Jimenez-Pabon, 1964), and literal paraphasia (Goodglass and Kaplan, 1972). Other patients dem-

onstrate many errors in the selection and production of words or phrases. This second category includes anomia, verbal paraphasia (substitution of a semantically closely related word, e.g., mother for father), and grammatic or syntactic errors (correct words produced out of order or words produced without appropriate grammatic inflection).

Although a classification into two different types of aphasia is often proposed, the criteria for distinguishing between phoneme and higher level errors are rarely given or not easily applied. For example, Darley (1970) has strongly urged the separation of apraxia of speech from aphasia. He defines apraxia of speech as "an impairment in the capacity to program the positioning of the speech musculature and the sequencing of movements for the production of phonemes", aphasia as "an impairment in symbolic processing". However, the criteria he uses for inclusion in the apraxia of speech category would be met by most if not all aphasic patients. Thus, expression in most classifications of aphasia contains initial phoneme errors on some occasions, more phoneme substitutions and omissions than distortions and "islands of error-free speech" (Darley, 1970), although the latter might be merely the same profanity uttered repeatedly.

Darley (1970) has also proposed that apraxia of speech can be distinguished from aphasia on the grounds that apraxia is not a word-finding difficulty. Unfortunately, the decision on this point

is often very inferential, e.g., the patient who produces "tup" for "cup" is said not to have word-finding difficulty, whereas the patient who produces "spag" for "cup" does have such difficulty. Without knowing something about the reliability of a particular misnaming or whether the patient can perhaps write it, though not be able to speak it, there is little basis for such inferences. Since assumptions regarding such a separation of apraxic and aphasic speech are not based on empirical evidence, it seems more parsimonious at present to consider the whole range of expressive speech disruption under the term aphasia.

Broca's aphasia (also termed expressive aphasia, aphemia, motor aphasia and anarthria) is characterized by laborious articulation even on a single phoneme level, with a severe reduction in the flow of speech. In other types of aphasia, speech may seem fluently and effortlessly articulated but produced with many errors. This dimension of fluency has been shown to discriminate usefully between what have classically been called Broca's (expressive) and Wernicke's (receptive) aphasias (Goodglass, Quadfasel and Timberlake, 1964; Kerschensteiner, Poeck and Brünner, 1972; Mateer and Kimura, in press).

Fluency is usually defined by the mean length of verbal responses (in syllables or words) in a picture description task. The criterion is easily applied and usable across examiners, a characteristic notably lacking in most aphasia classification sys-

tems. There may also be an anatomical correlate to the fluency distinction, in that the brain scan localization study by Benson (1967), and indeed many clinical observations from the time of Broca (1861), would implicate the anterior speech area of the left hemisphere in the nonfluent deficit, while the posterior speech area would be involved in the fluent deficits. The fluent/nonfluent classification will be used in general discussions throughout this paper, but review of specific studies will employ the investigator's terminology and, where possible, a description of its use within that study.

Problems in Investigating Nonverbal Oral Movement Impairments

The terms "oral apraxia" (DeRenzi, Pieczuro and Vignolo, 1966), "nonverbal apraxia of the oral mechanism" (Eisenson, 1962), "facial apraxia" (Nathan, 1947) and "faciolingual apraxia", (Woltman, 1923) have been used to describe impaired movements of the oral musculature in the absence of significant paresis. Most studies, however, have failed to give detailed descriptions of how such impairments have been measured. The following are some of the major problems:

1) Control for perceptual deficits. It is commonly accepted that oral movement to command is more difficult than to imitation or in response to environmental objects or situations, yet such interpretations have been confounded with the possibility of an impairment in auditory comprehension as a basis for the difficulty. Often, sufficient information regarding auditory comprehension has

not been given. Similarly, accurate visual perception of movements which are presented for imitation has rarely been ascertained (for example, by the use of recognition tasks).

2) Control for task difficulty. Performance in response to command, imitation, and object use have sometimes been used in a single test (i.e., scoring a response to imitation if a response to command is failed). Reporting responses to different modes of stimuli separately (Poeck and Kerschensteiner, 1975) is necessary for interpretation of the deficits.

Tests for oral apraxia have typically required fairly simple, usually single movements, e.g., protruding the tongue, blowing, chattering the teeth, etc. These movements might, in terms of their difficulty in production, be thought of as comparable to single speech sounds. Since most aphasics have more difficulty producing several speech sounds together than producing a single sound, the production of a single non-speech oral movement might not be particularly difficult. Recent studies (Lapointe and Wertz, 1974; Mateer and Kimura, in press) have used nonverbal oral motor tasks which require the imitation of several oral movements in a series. This kind of systematic increase in response length provides some parallel to the motor requirements of speech movements.

3) Limitation of tests to familiar movements. Russell and Espir (1961) suggested that apraxia results from a loss of "memory" for motor patterns rather than an impairment in the actual

execution of the motor response. Presumably, one has a "memory" only for previously executed movements. Many nonverbal oral movements used in traditional apraxia tests, such as blowing, sucking, whistling and licking the lips are familiar practised movements. Before making the inference that it is the mnemonic substrates of a movement that are lost, testing of movement patterns which could not have been committed to "memory" through practice or familiarity is required. Impairment on such tasks would suggest a fundamental deficit in the execution of movement per se.

4) Criteria for scoring. Scoring criteria have rarely been described. Often a simple correct/incorrect judgement, with little description of the basis for either decision, has been used. Some authors have included slowness, weakness and limited range of excursion of the oral movements as an indication of apraxic disturbance (LaPointe and Wertz, 1974). However, the classical definitions of apraxia have always stated that paresis is definitely not a primary feature of the disorder. If severe, it would, in fact, preclude determination of apraxic impairment. The recent use of multidimensional scoring systems for oral movement tests (Rosenbeck, Wertz and Darley, 1973; LaPointe and Wertz, 1974; Poeck and Kerschensteiner, 1975; Mateer, 1976) will hopefully lead to a more comprehensive classification of the types of errors that occur.

RELATIONSHIP BETWEEN NONVERBAL AND VERBAL ORAL MOTOR IMPAIRMENTS

Coexistence of the Disorders

The predominant clinical impression in the literature is that the underlying deficit in nonfluent aphasia is a motor one, an inability to produce or integrate movements of the organs of articulation for speech (Corbin, 1951; Eisenson, 1962; Bay, 1965; DeJong, 1967; Darley, 1967). Presumably, the motor-based interpretation of nonfluent speech deficits is suggested by the obvious difficulty and effort, even at a phoneme level, with which nonfluent aphasic speech is often produced.

Few studies have, however, systematically investigated the relationship between oral apraxia and type of aphasia. Nathan (1947) reported six cases who demonstrated oral apraxia and, from his description, nonfluent speech disturbances, after penetrating brain wounds. Alajuanine (1956) examined 22 patients with oral apraxia, 11 of whom, even after a period of years, continued to produce only a small repertoire of recurring utterances and verbal stereotypes and 11 of whom developed some degree of limited but functional expressive ability. Those cases in which the verbal stereotype did not change continued to manifest an "important (severe) oral apraxia". Although apraxic errors can be associated with longstanding aphasia, Alajouanine and Lhermitte (1960) suggested that nonverbal oral movement impairments are sometimes only

a transitory symptom because they are less complex than speech.

DeRenzi, Pieczuro and Vignolo (1966) found an oral apraxia in 90% (N = 42) of patients with Broca's aphasia and 83% (N = 6) of patients demonstrating phonemic jargon (meaningless syllables articulated in quick succession with approximately normal speech prosody). Poeck and Kerschensteiner (1975) found the imitation of oral movements to be severely impaired in patients with Broca's aphasia, although occasional errors were found in patients with Wernicke's aphasia as well.

Mateer and Kimura (in press) found that although patients with fluent aphasia were not impaired on the imitation of single oral movements, they were impaired when the imitation of a series of three such movements was required. Nonfluent aphasics demonstrated difficulty on the imitation of even single oral movements.

These studies all suggest a strong association between impaired production of simple nonverbal oral movements and nonfluent speech. In addition, fluent aphasics have difficulty in the imitation of nonverbal oral movements when more than one is required in a series. Thus, nonverbal oral movement impairments are seen in both fluent and nonfluent aphasia and, as Mateer and Kimura (in press) have suggested, may provide the underlying basis for the speech disorder.

Similarities in the Nature of "Spared" Verbal and Nonverbal Oral Function

In his early observations, Hughlings Jackson (1878) discussed the voluntary-automatic dissociation seen in impairments of both nonverbal oral movement and speech. Patients who may be unable to blow on command or even on imitation often do so quickly and easily when a lit match is presented. Patients who are unable to produce an utterance on command or to repeat it may produce the utterance with no apparent difficulty in an appropriate situation. He argued that one response, the automatic one, is "nonpropositional", while the ordered or imitated one is "propositional". Yet, if the response (for example saying goodbye in response to someone leaving) is appropriate to the situation, it is difficult to see in what sense the term propositional is used.

Speech movements which are preserved in aphasic disturbance are often either very brief high frequency words ("yes", "no", "Goddam") or longer highly practised sequences (counting, saying the days of the week). So-called automatic nonverbal oral movements (e.g., licking the lips, blowing out a match) are also familiar high frequency responses. Practice has empirically been shown to be a factor in the survival of a behavior after brain damage. Responses which have been overtrained preoperatively are better retained than those trained just to criterion (Chow and Survis, 1958; Orbach and Fantz, 1958; Lukaszewska and Thompson, 1967). Oral motor responses (both verbal and nonverbal) which

have been frequently produced appear to be the ones least affected by brain damage. It is, moreover, important to question whether apparently recovered or spared motor responses are performed with the same degree of speed, accuracy or organization as they were prior to the lesion (Goldberger, 1974).

Correspondence Between Types of Errors Made on Verbal and Nonverbal Motor Tasks

Errors on nonverbal oral movement tasks. Rosenbeck, LaPointe and Deal (1973) list common errors made on tests of oral apraxia; delayed responses, multiple response attempts, movements in the wrong direction, partial responses, substitution of alternate oral-facial structures to produce the movement, substitution of verbal or nonverbal vocalization and perseveration on previously produced movements.

Poeck and Kerschensteiner (1975) presented a detailed analysis of errors on a test of oral apraxia in aphasic/apraxic patients. Most errors were substitutions of oral facial movements other than the ones presented (37%), fragmentary execution of movements (14%), and the substitution of verbal movements (speech sounds or words) for nonverbal movements (11%). (Percentages are approximations based on figured data.) These authors also report an extremely high proportion of errors (44% analyzed across other error types) with an element of perseveration from previous movements. The general pattern of errors, including perseveration, was not different across four designated types of aphasia (Broca's, Wernicke's,

amnesic, global), although the frequency of errors, highest in Broca's aphasia, differed markedly across the aphasic groups.

Mateer (1976) found that perseverative errors accounted for a higher proportion of the total errors in patients with left hemisphere damage than in patients with right hemisphere damage or normal control subjects. Movements unrelated to the required ones in any obvious way, movements using alternate oral-facial structures and the substitution of vocalizations for movements were frequently demonstrated by fluent aphasics with left hemisphere damage. Often patients made continuous "searching" movements, revising the configuration of the oral structures as if they were unsure when the appropriate target position had been reached. In contrast, sequencing errors, (production of two correct movements out of order) rarely occurred.

Errors on tests of speech production. Shankweiler and Harris (1966), Deal and Darley (1972) and Trost and Canter (1974) investigated phoneme accuracy in patients with Broca's aphasia. Phoneme production was better in repetition than in naming tasks. Many errors were close approximations (in terms of the number of differing distinctive features) to target phonemes. Phonemes with a high frequency of occurrence in spoken English were more accurately articulated, while phonemes with a low frequency of occurrence were least accurately produced. There was marked inconsistency in production on repeated attempts, with frequent revision

of responses. Often, unrelated nonverbal oral movements were observed on verbal tasks, curiously reminiscent of the verbal intrusions on non-speech oral apraxia tests.

Production errors which appear to be at word and sentence levels, rather than at phoneme levels (the difficulty with such a dichotomy has been discussed) have been analyzed primarily in linguistic terms. Viewed very simply, however, the error patterns include the addition of inappropriate words and substitution of perhaps related but incorrect words or phrases. Wepman (1956) demonstrated that difficulty with word usage in aphasics was related to low word frequency in the language.

Perseverative errors are as salient with regard to disordered verbal responses as they are to disordered nonverbal responses. Within the nonfluent classification, responding can consist of constant repetition of a single sometimes meaningless utterance (Alajuanine, 1956). Fluent aphasics rarely demonstrate the kind of immediate perseverations seen in nonfluent aphasia, but often demonstrate repeated intrusions of particular phonemes, word or phrases throughout a speaking session (Buckingham and Kertesz, 1976).

The serial ordering of phonemes and words is frequently reported to be disturbed. Luria (1964) and Lashley (1951) discussed the expressive disorders of speech as a breakdown in the serial or sequential ordering of behavior. Mateer and Kimura (in press) reported, however, that although aphasic patients made many

omission and substitution errors when repeating a series of three consonant-vowel syllables, reversals, changing the order of phonemes per se, rarely occurred.

In summary, although evidence in many cases is limited, errors on both nonverbal and verbal oral motor tasks share many of the same characteristics:

1) Production of single oral movements and single phonemes is often impaired in the same patient. In other patients, impairments are seen only when a series of verbal or nonverbal oral movements is required. (Mateer and Kimura, in press).

2) Substitution of one recognizable oral movement or phoneme for another (sometimes a close approximation to the target) rather than weak production characterizes performance on both verbal and nonverbal tasks. (Trost and Canter, 1974; Poeck and Kerschensteiner, 1975; Mateer, 1976).

3) Perseverative responding is a major source of error on both verbal and nonverbal tasks. (Mateer, 1976; Alajuanine, 1956; Buckingham and Kertesz, 1976).

4) There are intrusion of nonverbal oral movements on verbal tasks and intrusion of verbalization on nonverbal tasks. (Rosenbeck, LaPointe and Deal, 1973; Poeck and Kerschensteiner, 1975).

5) When a series of nonverbal or verbal movements is required,

selection of appropriate movements seems to be impaired, while few errors are made in correct ordering of the movements per se (Mateer and Kimura, in press).

The similarity of error response characteristics on verbal and nonverbal oral motor tasks suggests a common mechanism underlying both kinds of deficits.

NEUROANATOMICAL SYSTEMS IN ORAL APRAXIA

Nathan (1947) found only one area of lesion common to five patients demonstrating oral apraxia and aphasia after missile wounds of the brain -- the inferior part of the left precentral gyrus (face area) or the region deep to this area. In three of the five cases, Broca's area is not involved and in two the supramarginal gyrus (portion of posterior speech area) could not have been involved. All of the cases had an upper motor neuron right facial weakness, but only two demonstrated a sensory loss on the face. Mateer and Kimura (in press) reported right sided hemiparesis (including the face) in all patients with single nonverbal oral movement impairments, a finding suggesting at least left inferior precentral involvement. Damage to the motor face area appears to be associated with oral apraxia for even simple oral movements.

Nathan reviews a number of studies (not available in English) which implicate, in addition to inferior precentral gyrus (Bonovici,

1914; Pussep and Levin, 1923), the corpus callosum (Mingazzini and Claria, 1920) and even right hemisphere lesions (Lewandowsky, 1907; von Monakow, 1914; Hartman, 1907) in cases of oral apraxia. Although Penfield and Rasmussen (1950) reported arrest of speech and production of nonverbal oral movements after stimulation of either the left or right inferior precentral cortex, Mateer and Kimura (in press) found no oral movement deficits after right hemisphere lesions, some of which necessarily involved the face area. Production of multiple oral movements was, however, impaired in fluent aphasia, the type of aphasia most often associated on brain scan with left hemisphere lesions posterior to the central sulcus (Benson, 1967).

RELATIONSHIP BETWEEN PRODUCTION AND COMPREHENSION DEFICITS IN APHASIA

It may be argued that a model of aphasia which views impairment in motor control as the fundamental deficit, ignores the obvious, often severe impairments in the comprehension of speech which may accompany the cerebral injury. A complete discussion of the classification of receptive impairments is beyond the scope of this paper (see Brown, 1974 for review).

Most studies which have investigated receptive speech impairments have varied the parameters of stimulus length, vocabulary difficulty, and syntactic complexity, although only a few have done so in a systematic fashion (Shewan and Canter, 1971). The

interpretation of tests for comprehension at the word, phrase or sentence level is, it seems, beset with the difficulty that speech is redundant. Only portions of the actual acoustic signal need be perceived for adequate comprehension, and the contribution of extraphonemic factors such as rate and inflection are infrequently controlled or measured. Boller and Green (1972), for example, found that even the most severe aphasics maintain an ability to discriminate whether they have heard a question or command. Tests for perception of single speech sounds have only rarely been conducted. Since each individual phoneme or vocalization produced by a subject can be transcribed, impairments in expression can be much more easily quantified than can impairments in reception (Kimura, 1974).

Despite these difficulties, many studies have found a high degree of correlation between measures of speech expression and reception in aphasics (Karis and Horenstein, 1976; Schuell and Jenkins, 1959; Kimura and Archibald, 1974). Although historically Broca's aphasia has been associated with good comprehension, patients with Broca's aphasia demonstrate a significant and almost constant impairment in verbal comprehension on the sensitive Token Test (DeRenzi and Vignolo, 1962).

Cases of pure motor aphasia or pure anarthria, i.e., without receptive difficulty have been reported in the literature (Lecours and Lhermitte, 1976). Although tests of auditory comprehension

have not been detailed enough in such cases to rule out receptive deficits, they point out the fact that there can be a major discrepancy between the effect of cerebral damage on expression and reception of speech. A very old idea, recently supported, to some extent, by the studies of "split-brain" patients (Sperry and Gazzaniga, 1967), is that speech expression is strictly lateralized in the left hemisphere, while reception of speech may be somewhat more bilaterally organized.

A strict separation of the expression and reception of speech, however, may not be a realistic point of view. Liberman, Cooper, Shankweiler and Studdert-Kennedy (1967) have outlined evidence for a motor theory of speech perception which holds that some components of speech are perceived with reference to the mechanisms by which they are produced, that speech input is processed via a motor command system. They suggest that perception is more closely related to the articulatory movements associated with speech than it is to the acoustic characteristics of speech. If perception depends, at some level, on integrity of the motor control system, an impairment of that motor control system might result in comprehension deficits.

To some extent, the level of receptive impairment appears to follow the level of expressive impairment in aphasia. In fluent aphasics, whose expression is not typically disturbed at a phoneme level, the comprehension of word and sentence length material is

impaired. In nonfluent aphasics, whose expressive difficulties are at a phoneme level, it seems reasonable to expect that the perception of phonemes might be impaired. Despite the frequent claim that such patients have only minimal receptive difficulty, the redundancy of the speech signal could conceivably allow for good speech comprehension even if the perception of individual units was impaired.

Carpenter and Rutherford (1973) reported that four of nine Broca's aphasics failed a task requiring a discrimination between acoustic cues of the kind found to be important for phonemic differentiation. In these four patients, however, comprehension of meaningful speech was better than all of five Wernicke's aphasics. Liberman (1974) cites an unpublished study by L. Taylor, B. Milner and C. Darwin in which nonaphasic patients with excisions of the face area of the left sensorimotor cortex were found to be severely impaired on the identification of stop consonants in a nonsense carrying phrase context, despite good pure tone acuity and speech comprehension.

LEFT HEMISPHERE SPECIALIZATION FOR COMPLEX MOTOR CONTROL

Other evidence suggests a special function of the left hemisphere in the control of nonverbal oral movement in normal subjects. Sussman and MacNeilage (1971), in a dichotic pursuit auditory tracking task, found a significant right ear advantage when the

signal to be varied was dependent on non-speech movements of the tongue or jaw. The laterality effect was interpreted as evidence for a left hemisphere mechanism relating oral motor responses to auditory input, a mechanism which could be functional for speech control.

A number of studies has supported a cerebral asymmetry in manual motor control. Kimura and Archibald (1974) found that left hemisphere damaged patients were impaired on the imitation of a sequence of meaningless hand and arm movements. The degree of impairment on this task was highly correlated with more traditional measures of manual apraxia employing the demonstration of gestures or object use. Milner (1976) found that the production of a previously trained sequence of arm movements was almost always impaired after sodium amytal was injected into the arterial system of the side of the brain where speech was represented, but rarely impaired when the non-dominant hemisphere was injected. Heilman (1975) reported impairments on a pursuit-rotor task after left hemisphere damage. Kimura (1976) found that patients with left hemisphere damage were impaired on the acquisition and performance of a manual motor skill. Kimura, Battison and Lubert (1976) reported that a deaf man, aphasic for sign language after a left hemisphere stroke, was impaired on the imitation of complex non-linguistic (meaningless) hand and arm movements.

It thus appears that the control of complex movement requiring changes in the position of both the limbs and oral musculature is closely linked to function of the left hemisphere. Since speech consists of a series of complex oral movements, perhaps an important aspect of "hemisphere dominance" for speech is just a capacity for complex sequential motor control (Liepmann, 1913; Mateer and Kimura, in press). Relevant to this kind of asymmetry for human motor function, is the greater role of the left motor nucleus in the control of song production, a learned communicative behavior, in canaries (Nottebohm, 1976).

Motor control of speech appears to be an extremely complex case of human motor control in general and as yet, the neural processes underlying it are poorly understood. Some degree of hearing is obviously requisite to the development and long-term maintenance of normal speech production (Chase, 1967; Hardy, 1970; Ringel, 1970; Abbs and Hughes, 1975). Although a number of studies has suggested a major role of tactile and proprioceptive feedback in the control of speech on the basis of speech defects after oral anesthesia (Scott and Ringel, 1971; Hutchinson and Putnam, 1975), the use of such procedures is doubtful because motor innervation has been shown to be affected as well (Abbs, Folkins and Sivarajan, 1975). Mateer and Kimura (in press) reported that two point discrimination deficits on the tongue did not correlate with verbal or nonverbal oral motor impairments. The role of afferent feedback in the production of speech is obviously not well understood.

MacNeilage (1970) proposed that the neural mechanism for speech control does not produce invariant movement patterns, but rather produces movements of the articulators for the attainment of relatively invariant target configurations. These target positions of the oral musculature are associated with particular phoneme productions, while the actual movement pattern depends on the preceding and following phoneme targets.

Kimura (1976) suggested that there may be a normal dependency on the left hemisphere for accurate positioning of the brachial and oral musculature. This special capacity for complex motor control could provide the underlying basis of left hemisphere specialization for speech. The impairments in production of non-verbal and verbal oral movements after left hemisphere damage certainly reflect an inability to achieve successive target configurations of the oral musculatures (Mateer and Kimura, in press). Expressive aphasic deficits may result from damage to complex motor control mechanisms on which such movements depend, the symbolic associations of the movements not being fundamentally related to the impairment.

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