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# Left-Hemisphere Control of Oral and Brachial Movements and Their Relation to Communication

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## Left-hemisphere control of oral and brachial movements and their relation to communication

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The left cerebral hemisphere of the brain in man is known to be involved in both vocal and manual communication, as evidenced by speech and manual sign language disorders after left-hemisphere pathology. The left hemisphere also has important motor control functions in certain kinds of non-communicative oral and manual movements. Several tasks requiring oral and manual-brachial movements were presented for reproduction to patients with unilateral restricted lesions of the left or right hemisphere. Patients with left-hemisphere lesions were impaired relative to patients with equivalent right-hemisphere damage, thus confirming and extending earlier reports on the effects of more widespread left-hemisphere damage.

Within the left hemisphere, frontal and parietal lesions had the most severe effect, with lesions in the central, occipital or temporal areas producing no or minimal impairment. The left frontal area was paramount in the control of oral movements, and the parietal lobe in control of hand movements, but both regions were required for the more demanding oral and manual tasks. Oral and manual control systems appeared more separable in the frontal than in the parietal region, suggesting that the latter may play a general programming role that is then enacted through the left frontal region.

Speech-disordered (aphasic) patients were inferior to non-aphasic patients on oral and manual tasks. There was a close association between aphasia and the presence of oral or manual defects in left frontal and parietal lobe lesions, but not in temporal-lobe lesions. In addition, a speech task requiring reproduction of a single syllable closely paralleled the reproduction of a non-verbal oral movement, in that both depended critically on the left anterior region; aphasic patients with parietal or temporal lesions were relatively unaffected on either task. However, on reproduction of multisyllabic familiar phrases, there was no association with oral motor deficits. Aphasic patients with anterior and parietal lesions could reproduce these phrases relatively well, but patients with temporal-lobe lesions (and lesser impairment on motor tasks), had great difficulty.

It is suggested that oral and manual apraxia, as well as aphasia, may be a manifestation of a basic motor selection problem in lesions of frontal and parietal lobes, but that the temporal region has some important acoustic-motor function in speech. When this temporal system is intact, it can, at least with overlearned material, bypass defective frontal or parietal oral motor systems.

### INTRODUCTION

It is well known that the left cerebral hemisphere of the brain in man plays an important role in communication. We know this from the fact that speech disorders are a common consequence of damage or disruption to the left hemisphere, and that such disorders are rare after right-hemisphere damage. The precise nature of the left hemisphere's contribution to normal communicative function is, however, not clear. It used to be thought that the left hemisphere was particularly important for symbolic function, i.e. that it was specialized for representational or linguistic behaviours.

Several investigators had, however, noted that neurological patients with left-hemisphere pathology not only showed speech disorders (called 'aphasias') but they also had difficulties in performing some oral and manual movements on command, such as smiling, protruding the tongue, showing how to salute, or how to hammer a nail. These motor difficulties (termed 'apraxias') were often discounted as being due to the language comprehension difficulties, or were interpreted as further evidence of the representational function of the left hemisphere. As early as 1908, however, Hugo Liepmann had suggested that the left hemisphere in man had critical control over what he termed purposive movements. In careful clinical examinations he showed that apraxic patients were not only impaired in responding to commands, but that they could generally not imitate the movements either. Thus, the arms might show good strength and were obviously mobile, and the patient might clearly understand what was required of him, but he could still be incapable of showing how to use a key (in its absence), even when this was demonstrated for him. Often such apraxic patients could, in fact, use the objects fairly well when actually placed in their hands, and Liepmann suggested that the visual, tactual and kinaesthetic cues provided by the object could thus compensate for a loss of what he called 'movements from memory'.

Since apraxia tended to be present in the left arm as well as the right, Liepmann proposed that the left hemisphere controlled such movements for both left and right arms, and also for the oral musculature, although he dwelt less on the latter. Much of his work was concerned with the role of the major commissure between the hemispheres, the corpus callosum, in mediating the bilateral control to left and right arms from the left hemisphere. He pointed out that damage to the motor cortex, which typically results in a weakness and loss of fine movement in the hand opposite the lesion, does not necessarily result in apraxia (Kimura 1980, pp. 26 and 35). Thus the pyramidal, or direct cortico-spinal, system, which mediates movements of the contralateral arm and especially of the contralateral hand and fingers (Brinkman & Kuypers 1973), is apparently not critically involved in the praxis system (Kimura 1979).

The demonstrated failure of apraxic patients to imitate movements correctly was a key fact in prompting the idea that their incapacity was, to some extent at least, a kind of motor control problem. Our earlier research took this idea a step farther and asked whether the defect would be present when the movements to be copied were meaningless or unfamiliar, that is, in no way representational. When patients with pathology of the left hemisphere are required to copy unfamiliar movements, they do indeed have serious difficulties, relative to patients with right-hemisphere pathology. This is true whether one requires a series of movements of the hands and arms (Kimura & Archibald 1974) or of the mouth (Mateer & Kimura 1977). In fact, meaningless non-verbal movements are even more difficult for such patients to copy than are familiar movements. The unfamiliar movements are generally more difficult for all patients to copy, probably because they are less practised. But the fact that apraxic patients fail to copy meaningless movements is a finding hard to reconcile with the idea that the left hemisphere is specialized for symbolic function. Neither does it fit easily with the idea that it is movements initiated from memory that depend critically on the left hemisphere. Instead, the findings were interpreted as a general difficulty in selecting the correct posture via a pre-programmed plan (Kimura 1977), and the fact that the defect showed up most clearly when a series of movements was required was taken to mean that this put the greatest demands on the selection system.

The idea that it was a selection mechanism that was disrupted was greatly strengthened by

finding that even severely apraxic patients, if given a repetitive task, could perform quite well, while an apparently simple motor task that required constant changes in hand posture could often not be learned (Kimura 1977, 1979). This is best illustrated by comparing two tasks, one in which a nut is screwed up and down a bolt as quickly as possible, using fingers and thumb. This requires a fair amount of finger dexterity, but once the actual rotation movement has been made, no further motor selection is necessary, because the movement is simply repeated over and over. Patients with left- or right-hemisphere damage may be slightly slower on the hand opposite the damage, as would be expected from the effect on corticospinal systems.

The other task requires the patient to face a box on which he is shown how to press a button with the index finger, pull a vertical bar forward with the whole hand, and then press down on a horizontal bar with the thumb. Clearly this second task, which a normal person can learn to a strict criterion in ten or fifteen seconds, requires changes in hand posture from one position to another. Patients with left-hemisphere damage are severely impaired on both hands on the latter task, but not on the screw rotation task.

The difficulty seen after left-hemisphere damage on tasks requiring a change in the position of the musculature was not restricted to manual movements. Patients with left-hemisphere damage also have such difficulty with a series of oral movements, even when they are able to reproduce a single oral movement. This principle extends also to the oral movements of speech and is best seen in aphasic patients with a kind of aphasia called 'fluent'. In fluent aphasia, speech may be replete with errors but its production appears effortless, in that there is no laborious articulation problem, as happens in non-fluent aphasia. In fluent aphasics in particular, one might consider that there is a selection deficit, in that individual syllables appear to be produced normally and easily, but in connected discourse, the selection of the syllable to be uttered is apparently impaired. In such patients, repetition of a single consonant-vowel syllable such as 'ba' over and over can be done as quickly as by a non-aphasic patient. But when the fluent aphasic is required to say three different syllables in series repeatedly, such as 'ba-da-ga', he has great difficulty (Mateer & Kimura 1977). This is true even when he can say the syllables over once or twice slowly, indicating that they have been perceived correctly.

This general problem in producing a series of movements, then, can be seen for the oral and brachial musculature, and in both verbal and non-verbal or meaningless kinds of movements. There are, however, also patients who have difficulty in the production of even a single movement, usually in the reproduction of a single hand posture but sometimes also of a single oral movement. Since the labelling of movements as single or multiple is of course somewhat arbitrary, it could be that we are simply seeing a more severe form of the selection problem in such cases.

Our published analyses to date have been concerned with whether there is damage to the left or right hemisphere, without being concerned with the extent or locus of such damage, since restricted lesions are not very common. After several years of investigation, we now have a sufficient number of cases with restricted locus of damage within a hemisphere, to enable us to look profitably at the effects of intrahemispheric organization of function for certain motor tasks. Such organization, and how it relates to communication, is the focus of the present study.

## COMPARISON OF LEFT AND RIGHT HEMISPHERES ON MOTOR TASKS

The 118 patients from whom these data were collected had sustained brain damage largely as a result of cerebrovascular accidents or tumours. As in past studies, data analysis was performed only on those patients whose damage was restricted to one hemisphere of the brain, left or right. In addition, within the cases of unilateral damage, data for the present study were selected from those patients whose lesions were contained within anterior or posterior regions of one hemisphere. Anterior was defined as in front of the central fissure of Rolando, and posterior as behind the central fissure and including the temporal lobes. The sample size in each group as well as the mean age and the aetiology are shown in table 1. The actual sample size on each test was usually smaller than this, since not all patients received all tests.

TABLE 1. STATISTICS ON PATIENT GROUPS

locus of damage	<i>n</i>	male, female	tumour, vascular	mean age years	performance I.Q.
left anterior	22	13, 9	15, 7	51.6	89.2†
left posterior	50	31, 19	20, 26	43.9	95.6†
right anterior	15	9, 6	6, 7	55.1	98.2
right posterior	31	14, 17	11, 16	37.0	95.5

† Overestimated because some aphasics were omitted.

The motor tasks on which data are reported are as follows:

(a) *Single Oral Movements*. The patient is asked to reproduce, by imitation, a series of relatively simple single oral movements, presented one at a time. They include tongue lateralization, tongue protrusion, chattering the teeth, and blowing. The maximum possible score is 17.

(b) *Multiple Oral Movements*. This is a modification of the test devised by Mateer & Kimura (1977), in which three oral movements in a row are presented for imitation. Only at the end of the three may the patient begin to reproduce the movements, some of which consist of the single movements from the task above, and some of which are new movements, e.g. one item is to lateralize the tongue, open the mouth, protrude the lips. A practice item precedes the test items. In the abbreviated version reported here, three items of three movements each are presented, making a maximum possible score of 9.

(c) *Hand Postures*. This task is an abbreviated version of that described in Kimura & Archibald (1974). The patient is asked to copy a static hand posture after it is presented. If unsuccessful, the model posture is presented again and kept in view for the patient to copy. There are five such postures, with a maximum possible score of 10.

(d) *Multiple Hand Movements*. This is an abbreviated form of a movement copying task (Kimura & Archibald 1974). Three series of movements involving one hand and arm are presented for imitation. They are essentially meaningless, e.g. one item consists in bringing the extended fingers and thumb with tips together, to the forehead between the eyes; then rotating and moving the hand forward in a straight line, opening the hand as it moves. There is a detailed scoring system for components of the movement, and a practice item before the test. The maximum score is 24.

For the manual tasks, although each hand is tested whenever possible, some patients with anterior lesions have a paralysis of the hand or arm on the side opposite the damaged hemi-



sphere. For this reason, the data on manual-brachial tasks are always reported for the hand on the same side of the lesion; where strength will typically be unaffected. That is, data are given for the left hand of the left-hemisphere group and the right hand of the right-hemisphere group. Past studies have shown no significant difference between left and right hands within patients in performance on these tasks (Kimura & Archibald 1974; Kimura 1979).

A comparison of patients with damage to the left or right hemisphere, anterior or posterior is shown in figure 1. Patients with either anterior or posterior left-hemisphere lesions are inferior

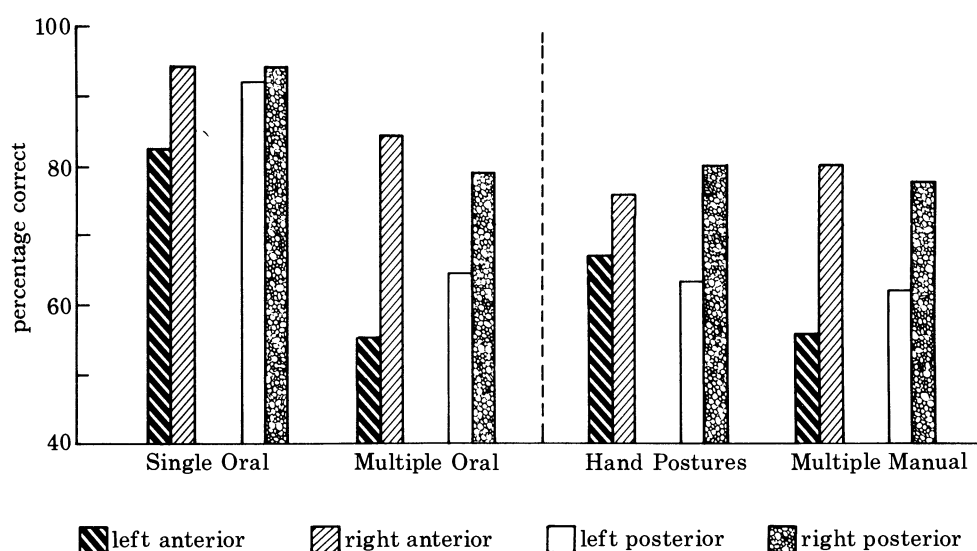


FIGURE 1. Comparison of the effects of right and left hemisphere lesions on oral and manual tasks.

to those with equivalent right-hemisphere damage on both of the tasks that involve making several movements (Multiple Manual, Multiple Oral). However, on single movements a somewhat different pattern emerges. On Single Oral Movements the only group significantly impaired is the left anterior group ( $p < 0.05$ ), whereas on Single Hand Postures the only group impaired is the left posterior group ( $p < 0.005$ ).

Normative data are not available on the oral movements, but on the two manual tasks a group of elderly normals performed at a level quite comparable with the right-hemisphere group (Hand Postures: normals, 72%; right-hemisphere lesions, 79%. Multiple Manual: normals, 85%; right-hemisphere lesions, 78%). It therefore seems reasonable to assume that performance by the right-hemisphere group on the oral movements is also near normal, despite the report (Kolb & Milner 1981) that copying of similar multiple oral and manual movements is impaired by right frontal lesions.

#### INTRAHemispheric ORGANIZATION OF MOTOR FUNCTION

Confirming previous reports in our laboratory and elsewhere, then, it is clear that the left hemisphere plays a critical role in the control of certain oral and brachial movements. This is especially noticeable with multiple movements, where greater demands would be made on

selection, but it does also appear in production of single movements. However, with only one movement, there appears to be a degree of dissociation between anterior and posterior systems in the left hemisphere, in that single oral movements depend more on the left anterior region, and single manual postures on the left posterior region, for accurate execution.

The organization of these functions within the left hemisphere was further pursued by breaking the posterior group down into several subgroups, a subclassification permitted by the

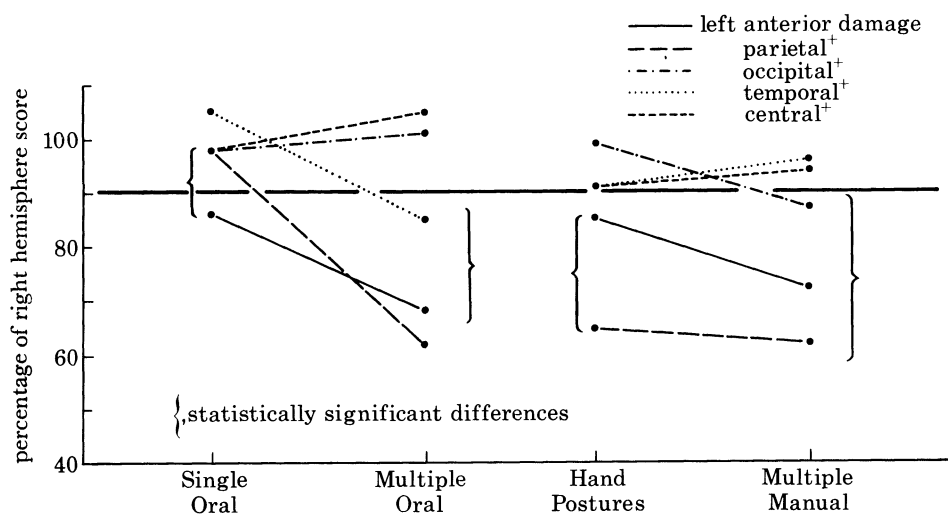


FIGURE 2. The effects of locus of lesion within the left hemisphere on oral and manual tasks.

large size of this group. Within the left posterior group, subgroups could be determined in which the cortical extent of the damage was primarily temporal ( $n = 18$ ), primarily parietal ( $n = 14$ ), or primarily occipital ( $n = 7$ ). With vascular lesions and tumours, the white matter beneath the cortex is almost invariably affected as well. A further group with lesions affecting primarily the left central (Rolandic) region (and thus not classifiable as anterior or posterior) was added for left intrahemispheric comparisons. There were 18 patients in this left central group. These groups of patients were so classified as to be mutually exclusive.

The performance of the left-hemisphere subgroups on oral and manual tasks is shown in figure 2. In order to equate for difficulty level of the various tasks, the scores are shown here and in subsequent figures as a percentage of the score for the entire right-hemisphere group. The 90% performance level of the right-hemisphere group is indicated by a line. As a rough guide, patient groups that fall below this level can be taken to be impaired, but specific statements concerning statistical levels of significance will be made where appropriate, to supplement the figures.

Figure 2 indicates exactly as in figure 1 that the left anterior group is impaired on tasks involving both oral and manual multiple movements, but is also impaired in producing single oral movements. However, within the posterior group, only the left parietal subgroup is impaired on any of these tasks, and in fact it almost entirely accounts for the left posterior deficit seen in figure 1. On Single Hand Postures, the left parietal group is significantly impaired even relative to the left anterior group, whereas these two groups do not differ significantly on Multiple Manual Movements or on Multiple Oral Movements. There is no impairment in left

occipital or left central groups, and only a suggestion of an impairment in the left temporal group on Multiple Oral Movements.

It appears, then, that when the selection of more than one movement is required, both left frontal and left parietal lobes play a significant role, whether the movements are oral or manual. However, oral movement control is critically dependent on left anterior systems, and in fact the multiple movement deficit in patients with left anterior lesions may reflect the importance of this region in producing a single oral movement. A parallel statement for hand and arm movements may be made of patients with left parietal damage, in that they show the greatest impairment of any group on manual tasks. Moreover, the difficulty in executing a series of hand movements appears to be not much greater in this group than is that of executing a single movement.

TABLE 2. ASSOCIATION BETWEEN ORAL AND MANUAL MOVEMENTS  
(Product-moment correlation and percentage association.)

	Single Oral	Single Manual	Multiple Oral
<i>left anterior lesions</i>			
Single Manual	0.32 (10%)	— (—)	— (—)
Multiple Oral	0.87** (76%)	0.64* (41%)	— (—)
Multiple Manual	0.20 (4%)	0.57* (32%)	0.49 (24%)
<i>left parietal lesions</i>			
Single Manual	0.46 (21%)	— (—)	— (—)
Multiple Oral	0.53 (28%)	0.68** (46%)	— (—)
Multiple Manual	0.56* (31%)	0.86** (74%)	0.85** (72%)

\*  $p < 0.05$ ; \*\*  $p < 0.01$ .

In order to further explicate the movement control systems in left anterior and parietal regions, intercorrelations were done among the four motor tasks. The sample size is not the same for all tasks, but varies from 13 to 18 in the left anterior group and from 11 to 16 in the left parietal group. The correlations are shown in table 2. The high correlation in the left anterior group between single and multiple oral movements (accounting for approximately 75% of the variance) does indeed confirm the suggestion that the difficulty with multiple oral movements in this group is related to the difficulty with single oral movements. This oral movement control system apparently does not overlap extensively with the manual control system in the left anterior region, since scores for multiple oral and multiple manual movements do not correlate significantly.

In contrast, the left parietal group shows not only a very high correlation between single and multiple manual scores (accounting for approximately 75% of the variance), but also a very high correlation between multiple manual and multiple oral scores (accounting for over 70% of the variance). The overall pattern suggests greater overlap in the left parietal region between oral and manual control systems than is present in the left anterior region.



## MOTOR SKILLS AS A BASIS FOR COMMUNICATIVE FUNCTION

The two major natural systems of communication in man are speaking and manual signing. Complex manual sign languages are of course most prevalent among deaf populations, but rudimentary manual communication systems are employed within some hunting groups during hunting, and were employed at one time across North American Indian tribes.

*Manual communication*

Several cases of sign language disorders in the deaf after central nervous system damage have now been reported in the literature, and it is clear that these typically occur after left-hemisphere damage (table 3) (Kimura 1982). Disorders in manual signing, or sign language aphasias, have

TABLE 3. MANUAL SIGNING DISORDERS IN LEFT- AND RIGHT-HANDED SUBJECTS ( $n = 11$ )  
(Cases reviewed in Kimura (1982).)

	left-hemisphere lesion	right-hemisphere lesion
right-handed	9	0
left-handed	1	1

always been interpreted as linguistic disorders (as indeed have vocal aphasias). Some of these cases do not have obvious difficulty in performing familiar movements to command, i.e. to show how to use a hammer, to salute, etc. Since this is the usual method of testing for manual apraxia, it seemed reasonable to conclude that there was no apraxia and that the defect was restricted to linguistic movements. However, it is problematic whether such well practised movements are in fact a good means for testing for a motor control problem. Many more patients have difficulty with the less practised unfamiliar series of movements described earlier under Multiple Manual Movements than they do in demonstrating object use or showing familiar intransitive movements (Kimura & Archibald 1974). The familiar and unfamiliar movements are highly correlated, but the familiar movements are easier for all subjects and may thus be considered to be less sensitive to an apraxic impairment (Kimura 1979). This would be particularly true for patients with a manual signing history, where the familiar movements would be frequently employed in pantomime in conjunction with the sign language.

It is therefore critical to the linguistic hypothesis to know whether, if presented with a more difficult non-linguistic series of movements, a patient with sign-language aphasia would be impaired. In fact, this question has been broached in only two cases of signing disorder (Chiarello 1982; Kimura *et al.* 1976). In both cases, there was a significant impairment in reproducing non-linguistic hand movements. In the study by Kimura *et al.*, the same movements presented to non-aphasic deaf signers were performed with no difficulty, though they were not coded as signs. It is thus quite possible that a similar defect went undetected in previous reported cases of sign-language aphasia.

In the case reported by Kimura *et al.* (1976), the lesion involved the distribution of the left middle cerebral artery, and since there was only minimal weakness on the right, may have been primarily posterior. In the case reported by Chiarello the major damage, as indicated by

computed tomography scan, was in the left parietal region, although there was some Rolandic involvement as well. Assuming at least parietal damage in both cases, it is entirely to be expected, from the effect of localized lesions reported above, that a difficulty in producing manual movements would ensue. There seems no reason to suppose that the manual signing difficulty was anything more than a motor selection problem. However, the fact that the breakdown in the sign language was non-random, i.e. that the errors bore some relation to the structure of the sign language, has been interpreted by others as indicating that the defect *in signing* was linguistic (Battison 1978; Chiarello 1982). Whether this is a necessary conclusion will be discussed in a later section.

#### *Vocal communication*

How does the left-hemisphere organization for motor control relate to control of speaking? Some light may be shed on this question by comparing patients with and without speech disorders, on the motor tasks described above (figure 3). There are nine aphasic patients in the left anterior group and seven patients in each of the left parietal and left temporal groups. No

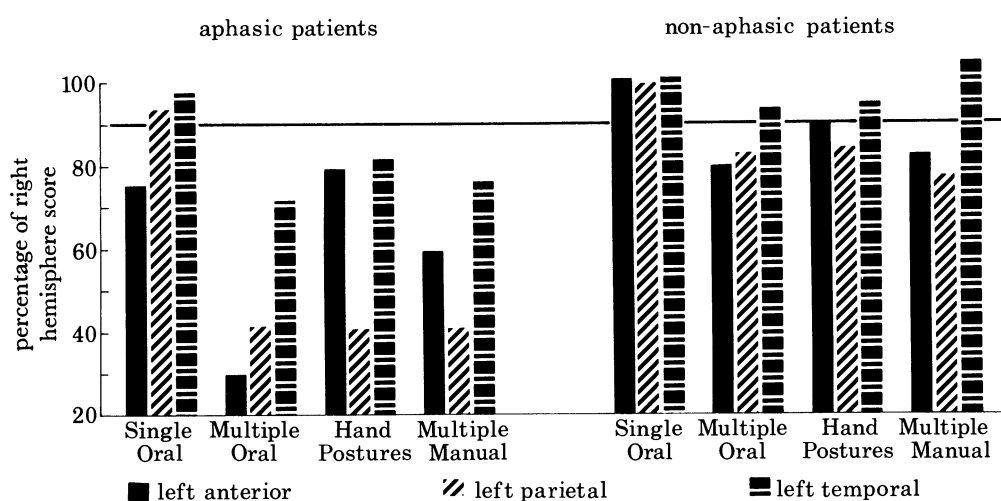


FIGURE 3. Comparison of the performance of aphasic and non-aphasic patients on oral and manual tasks.

patients with occipital lesions were aphasic, and only one patient with a Rolandic lesion was aphasic, so that no comparisons can be made within occipital or central lesions.

It is immediately apparent that all three aphasic subgroups are more impaired than the corresponding non-aphasic groups on almost all motor tasks. It is also worth noting that left anterior and parietal lesions, even when they do not render the patient aphasic, have significant deleterious effects on most motor tasks. Temporal-lobe lesions, however, have such effects only in aphasic patients. Of the four motor tasks, the single exception to this statement appears to be Single Oral Movements, where only the left anterior aphasic group is affected relative to the non-aphasic. This confirms the repeated reports in the literature of the association of oral apraxia with non-fluent aphasia or 'Broca's' aphasia (De Renzi *et al.* 1966; Mateer & Kimura 1977; Poeck & Kerschensteiner 1976). Left parietal and temporal aphasics are not impaired on single oral movements, but they are more impaired on all other tasks than the corresponding non-aphasic group.

There is thus a close but not inevitable association between the presence of aphasia, as clinically defined, and the degree of motor dysfunction. Cases without aphasia but with severe oral or manual apraxia are not common, but they do occasionally occur after anterior or parietal lesions. Typically, these patients have had a transient aphasia, which has cleared. The converse, aphasic patients who are free of motor impairments, happens most often with temporal-lobe lesions, rarely after anterior lesions and essentially never with parietal lesions. When sparing occurs after anterior lesions, it appears to be manual movements that are spared, confirming once again the greater separation of oral and manual control systems in the left frontal region than in left parietal.

It is possible that the accentuated motor impairment in aphasic patients is due to the fact that the lesions are more extensive or severe than in non-aphasics, and thus that they coincidentally also affect motor control systems. Certainly that would appear to be a reasonable inference with the temporal-lobe lesions. However, the inevitability of motor control defects in parietal-lobe aphasics, and of oral motor defects in frontal-lobe aphasics, argues against a simple size-of-lesion explanation in these regions. It seems more probable that in aphasics with anterior or parietal lesions, some very critical motor control system is affected, and that aphasia in these cases is another manifestation of this defective control.

If the aphasic disturbances that occur after anterior and parietal lesions are indeed manifestations of a disorder in motor control, then one would expect some differences to be apparent also in the character of the speech disorders, as between anterior, parietal and temporal lesions. A great deal has been written on the nature of aphasic disorders from anterior and posterior lesions, but on the *output* side there appears to be one major difference, the degree of fluency. Aphasic patients with frontal lesions tend to be non-fluent, that is, they have a difficulty at the syllabic-articulatory level that manifests itself also in short utterances (Benson 1967). The question whether there are significant differences in the control of speaking between temporal and parietal lesions has not been extensively discussed.

The potentially different functions of anterior, parietal and temporal lesions in the control of speech output may be further elucidated by two speech-repetition tasks that were administered to all patients. They were intended to be roughly parallel to the oral and manual tasks described above.

*Single syllable reproduction.* An isolated syllable is presented for immediate reproduction by the patient, in full view of the examiner's face. The task includes all of the stop consonants, five vowels, as well as sibilants, fricatives, nasal sounds and glides. Twenty different sounds are presented, with a maximum possible score of 20.

*Multisyllabic speech.* This consists of several familiar phrases that must be reproduced, e.g. 'black sheep'. Credit is given for each individual phoneme correctly reproduced. Maximum score is 54.

These speech tasks are, however, clearly different from the non-verbal oral motor tasks in that (1) they are presented in the auditory, not the visual, modality and (2) the phrases at least are more familiar than the non-verbal tasks, and consequently should be easier for most people.

Since non-aphasic patients have no difficulty with these verbal tasks, the data will be reported only for aphasic patients. Performance on these two verbal tasks and on the parallel non-verbal oral movements are compared in figure 4.

If one looks first at single oral movements and single syllables, it is apparent that the two types of response are closely related in terms of anatomical substrate. The left anterior region is critical for the production of both speech and non-speech single oral movements, with the parieto-temporal region of much lesser significance. This relationship is also confirmed by the significant correlation between the two tasks across all aphasic subjects ( $r = 0.61$ , 21 degrees of freedom,  $p < 0.01$ ), despite their very different modes of presentation. This finding is in agreement with earlier suggestions that the basic elements of both speaking and non-speaking oral movements are mediated by the same or similar mechanisms (De Renzi *et al.* 1966; Poeck & Kerschensteiner 1975).

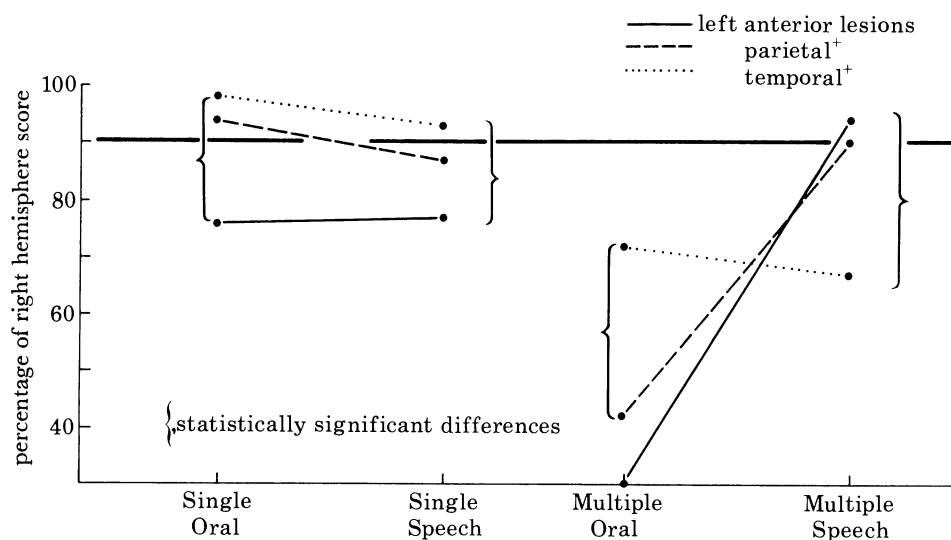


FIGURE 4. The effect of locus of lesion in aphasic patients on verbal and non-verbal tasks.

However, when one looks at the multisyllabic phrases, the picture is quite different. Here it is apparent that significant impairment is produced by lesions of the temporal lobes, and there is minimal impairment after anterior or parietal lesions, even though the latter produce aphasia. This is in sharp contrast to the pattern of deficits on multiple oral movements, where it will be recalled that anterior and parietal-lobe lesions produce the greatest deficit.

This suggests that the mechanism for the reproduction of single syllables is very different from that for multisyllabic speech, at least for familiar overlearned multisyllabic speech. This inference is further strengthened by the lack of correlation between the single and multisyllabic speech tasks ( $r = 0.315$ , 21 degrees of freedom, not significant). Thus the performance on single non-verbal oral movements and the reproduction of single syllables are significantly associated, but the reproduction of two types of speech – single syllables and multisyllabic familiar speech – is not. The anterior system in the left hemisphere is particularly important for mediating the production of single speech sounds, whereas the left temporal region is of critical importance in mediating the reproduction of multisyllabic speech.

This does not appear to be due entirely to the limitation of verbal short-term memory in patients with left temporal lesions. Data were fortuitously available on four of the seven aphasic patients with temporal-lobe lesions, on a task requiring changing syllable production.



That is, the patient was required to reproduce the sequence ba-da-ga over and over as quickly as possible (Mateer & Kimura 1977). Three of the four patients could reproduce the sequence at least once, indicating that perception was sufficiently accurate and that the short-term verbal store was adequate to the performance of the task. However, they were unable to continue with rapid repetition, making intrusive and repetitive errors, much as they did in the reproduction of phrases.

Measures of receptive speech function do not differentiate temporal-lobe aphasics from the other aphasic subgroups in our study. It seems rather that some important acoustic-motor control is vested in the left temporal region. This system appears to be necessary for eliciting speech movements via the auditory mode. As long as this left temporal region is intact, even in aphasic patients, the reproduction of auditorily presented familiar speech material can be achieved with a high degree of accuracy. Aphasic patients with anterior and parietal-lobe lesions, even those who have severe difficulty in reproducing non-verbal oral movements, may reproduce familiar phrases quite well. This suggests that the temporal acoustic-motor system is not only necessary, but may also be sufficient, for the reproduction of this kind of material.

In spontaneous speech, by definition, all aphasic patients make errors. Clearly the frontal and parietal regions do contribute in some way to the correct selection of the movements of speaking, but this may not be via the auditory mode. It appears that in the production of connected discourse, quite apart from the overall content of the communication, there are at least two important avenues of speech selection. One is the selection of the correct movements, or articulatory postures, via a motor programming system; the other is the running off of such articulatory sequences via an acoustic-motor channel. When the basic oral motor selection mechanisms are defective, as one must assume that they are in certain frontal and parietal lesions of the left hemisphere, the acoustic-motor pathway is still able to reproduce segments of overlearned material, suggesting that this pathway can bypass at least one of the motor programming systems in the frontal and parietal lobes.

Not only can an intact temporal-lobe acoustic-motor system function to elicit speech correctly in the presence of oral motor deficits, it can also function in the presence of a defect in reproduction of a single *speech* sound. Several aphasic patients with left frontal damage had difficulty in reproducing a single syllable, but were, surprisingly, much better able to reproduce the multisyllabic phrases (figure 4). This suggests that the reproduction of multisyllabic speech via the acoustic-motor system need not proceed syllable by syllable, but presumably can short-circuit the anterior syllabic system. Whether this short-circuiting proceeds via some minimal replicative function in the parietal lobe or by means of more direct input to lower speech centres is at present an open question.

It might be expected that with difficult verbal material or unfamiliar verbal sequences, the bypassing of oral-motor control systems would be less likely to occur than with overlearned material. Unfortunately, data on the ba-da-ga task were not systematically collected on the present population, so we have insufficient information at the moment to answer this question. We do know from previous work that patients with non-fluent aphasia, and thus with presumably anterior damage (Benson 1967), typically cannot produce even a single syllable repeatedly (Mateer & Kimura 1977). In the present study, aphasic patients with anterior damage are, as expected, less fluent than those with parietal or temporal damage, in spontaneous speech, and should thus have difficulty on the ba-da-ga task. The critical comparison group, therefore,



would be aphasic patients with parietal-lobe damage who presumably have minimal difficulty at the level of producing a single syllable, but might be expected to experience difficulty with several syllables. However, only one such patient was administered the task, so this question must remain a matter of speculation until we have sufficient data to answer it.

#### GENERAL COMMENTS

Evidence has been presented that the midfrontal and parietal regions in the left hemisphere have a critical function in control of oral and brachial movements. This control function appears to some degree complementary, in that oral movements depend more on the anterior region, and manual movements on the parietal region. The specificity of oral and manual control is best seen with single movements, in that single oral movements are affected almost exclusively by left frontal lesions, while single hand postures are affected primarily by parietal lesions. In contrast, when multiple oral or multiple manual movements must be produced, considerably more overlap of function between anterior and parietal lesions appears. This may be due to the fact that multiple movements put greater demands on the control systems than does a single movement, and that consequently the lesser role of each region in the complementary function becomes more apparent.

Intercorrelations among oral and manual tasks within anterior and parietal groups further suggest that there is a sharper separation between oral and manual control systems in the left anterior region than in the left parietal. A reasonable inference from this fact is that the parietal region is concerned more with general programming of movements, and that it subsequently influences the more specific motor control systems in the left frontal region. The direction of this influence, from parietal to frontal, was suggested by Hugo Liepmann many years ago. Aside from the differential specificity of oral and manual control, it is not clear at present what further differentiates the parietal and frontal contributions to movement.

The temporal lobe contribution to motor control appears minimal and is seen only after lesions that result in aphasia. Since these lesions involved the posterior temporal region, it is possible that they encroached somewhat on the parietal lobe, and that the motor deficits seen thus reflect parietal-lobe function. Only the assessment of further cases will provide an answer.

#### *Relation to communication*

The paramount role of the left hemisphere, and in particular the parietal region, in the control of arm and hand movements also manifests itself in sign language disorders after damage to the central nervous system in the deaf (Kimura 1982). Although it is usually claimed that the sign language disorders are linguistic in nature, what few data there are do not unequivocally favour this position. Thus, meaningless, or non-linguistic hand movements have also been found to be defective in the only two cases in whom they have been studied (Chiarello 1982; Kimura *et al.* 1976). Despite this fact, the tendency has been to regard this defect as merely an associated disorder, rather than the basis for the signing disorder (Battison 1978; Chiarello 1982). The argument is that since the errors in signing bear some meaningful relation to the structure of the language, they cannot be motoric in origin. However, this argument ignores the nature of the motor defect in apraxia. Apraxia is not a defect of strength or general motility, but appears to be a motor selection or programming difficulty of some kind. It is to be

expected that such a selection mechanism will have some means of incorporating the past experience of the organism. Therefore, if the mechanism becomes defective, the errors produced in signing would also be expected to bear some relation to the learned pattern of movements in signing.

The same claim might be made of vocal aphasia. However, in speaking, the visible mouth movements are not the chief carriers of information. Rather, the output is processed primarily by the auditory modality, and it has generally been assumed that the auditory system plays an important monitoring role in speech production as well. Some clues about how the motor and acoustic components of speech control are organized can be gleaned by considering what happens to the reproduction of oral movements, and of speech sounds, after damage to restricted systems in the left hemisphere.

In the left anterior region, lesions that produce aphasia invariably produce an oral motor control defect. This defect may not always be apparent with single oral movements but is certainly evident when several oral movements are demanded. In the left parietal region, aphasia is accompanied by both oral and manual deficits. The close association between aphasic disorders and motor control problems in these two groups suggests that the aphasia itself is a manifestation of the motor control difficulty. This is less true of aphasia after temporal-lobe lesions.

Let us assume that the way in which the oral motor system is organized has a parallel in speech. This would suggest that the unit of speech, possibly the syllable, is critically dependent on the anterior region, but that running off several such units requires an intact parietal system as well. Thus with an anterior lesion there is hesitancy and articulatory difficulty apparent even at the level of the syllable, and a consequent difficulty in reproducing single speech sounds. Speech is also non-fluent, in that utterances are short. If the programming of several of these units requires the parietal lobe, then with parietal lesions there should be a difficulty apparent primarily at the multisyllabic level. In fact, individual sounds can be produced, but spontaneous speech, although fluent, contains many errors, errors that presumably reflect inaccurate selection of the units based in the frontal region.

So far, this is consistent with the motor defects. However, the difficulty that aphasics with anterior lesions have in producing spontaneous speech does not appear when they have merely to repeat back familiar phrases, even though they may have difficulty repeating back a single sound. Similarly, aphasic patients with parietal lesions, although unable to organize spontaneous speech without error, are able to repeat back such phrases. Yet aphasic patients with temporal-lobe lesions, with minimal motor defects, are clearly impaired on this same task.

There are at least two obvious characteristics that differentiate the repetition of familiar phrases from the spontaneous production of speech. One is the familiarity of the material. It is axiomatic that more familiar overlearned material is less subject to disruption of the central nervous system than is less familiar material. This would suggest that well learned series, such as counting, would be less likely to be disrupted than naming or describing, and this is true for most aphasic patients. However, the other characteristic has to do with the degree of auditory control in speech production. When phrases are to be repeated back, auditory input is paramount in determining the response, a condition considerably less true in spontaneous speech. Although acoustic memory for speech sounds may certainly play a role in organizing normal speech, it is probable that non-acoustic motor control is of at least equal importance.

The left temporal lobe appears to be critically involved in the eliciting of speech via auditory channels. As long as this acoustic-motor region is intact, multisyllabic speech can be reproduced with a fair degree of accuracy. This may be true even when motor impairments are present, and when the reproduction of a single syllable is defective. This suggests that in normal spontaneous speech there are at least two sources of control, one having to do with the programming of the movements of speaking, and one having to do with acoustic triggering. Both systems must be intact to produce error-free speech in connected discourse, but if required only to repeat back a series of well learned phrases the acoustic-motor system appears to be capable of by-passing the defective oral motor control systems in anterior and parietal lobes.

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## REFERENCES (Kimura)

- Battison, R. 1978 *Lexical borrowing in American Sign Language*. Silver Springs, Maryland: Linstock Press.
- Benson, D. F. 1967 Fluency in aphasia: correlation with radioactive scan localization. *Cortex* **3**, 373–394.
- Brinkman, J. & Kuypers, H. G. J. M. 1973 Cerebral control of contralateral and ipsilateral arm, hand and finger movements in the split-brain Rhesus monkey. *Brain* **96**, 653–674.
- Chiarello, C. 1982 Aphasia in a prelingually deaf woman. *Brain* (In the press.)
- De Renzi, E., Pieczuro, A., & Vignolo, L. A. 1966 Oral apraxia and aphasia. *Cortex* **2**, 50–73.
- Kimura, D. 1977 Acquisition of a motor skill after left-hemisphere damage. *Brain* **100**, 527–542.
- Kimura, D. 1979 Neuromotor mechanisms in the evolution of human communication. In *Neurobiology of social communication in primates* (ed. H. D. Steklis & M. J. Raleigh), pp. 197–219. New York: Academic Press.
- Kimura, D. 1980 *Translations from Liepmann's essays on apraxia*. Univ. Western Ont. Psychol. Dept. Res. Bull. no. 506.
- Kimura, D. 1981 Neural mechanisms in manual signing. *Sign Lang. Stud.* **33**, 291–312.
- Kimura, D. & Archibald, Y. 1974 Motor functions of the left hemisphere. *Brain* **97**, 337–350.
- Kimura, D., Battison, R. & Lubert, B. 1976 Impairment of non-linguistic hand movements in a deaf aphasic. *Brain Lang.* **3**, 566–571.
- Kolb, B. & Milner, B. 1981 Performance of complex arm and facial movements after focal brain lesions. *Neuropsychologia* **19**, 491–503.
- Liepmann, H. 1908 *Drei Aufsätze aus dem Apraxiegebiet*. Berlin: Karger.
- Mateer, C. & Kimura, D. 1977 The impairment of nonverbal oral movements in aphasia. *Brain Lang.* **4**, 262–276.
- Poeck, K. & Kerschensteiner, M. 1975 Analysis of the sequential motor events in oral apraxia. In *Cerebral localization* (ed. K. J. Zülch, O. Creutzfeldt & G. C. Galbraith), pp. 98–111. Berlin: Springer-Verlag.