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What the Hands Reveal About the Brain.
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Chapter 6

Apraxia and Sign Aphasia

American Sign Language displays complex linguistic structure but does so by means of gestures (primarily of the arms and hands). Gesture and language are transmitted in the same modality. Is the breakdown of sign language dissociated from disorders of movement and gesture? That is, is sign language represented in the brain in a different way from that of learned movement in general? Investigators have raised much the same question with regard to speech, but there the issue is more difficult to address because most movements of the speech articulators are hidden from view. The movements of the hands and arms, however, are directly observable.

6.1 *Apraxia: Motor Disorder or Symbolic Disorder?*

In attempting to understand the principles of neural organization underlying language, some investigators have tried to root language in movement control and others have tried to base it in the human capacity to convey meaning through symbols. Both sets of investigators have linked the apraxias (neural disorders of purposive movement) with the aphasias.

Kimura (1976, 1979), for example, considers the left hemisphere to be specialized for positioning the oral and manual articulators rather than for symbolic functioning per se. The system of control in the left hemisphere apparently depends on the accurate representation of moving body parts, not on sensory feedback, and thus it is fundamental to the production of a series of self-generated movements (Kimura 1979). Kimura and her colleagues find that aphasics, unlike patients with damage to the right hemisphere, are unable to copy sequences of meaningless movements of the hands or mouth (Kimura 1976). In further support of a link between left-hemisphere dominance for language and left-hemisphere control of movement, Kimura notes that there is a close connection between brain lateralization for speech and hand dominance, that disorders in manual communi-

cation quite often result from left-hemisphere lesions in deaf people, and that persons with left-hemisphere dominance for speech show frequent occurrence of certain right-hand movements during speaking.

Other investigators have proposed a common basis for movement and speech disorders on quite different grounds: They attribute both apraxia and aphasia to an underlying inability to express or comprehend symbols (see Feyereisen and Seron (1982) for a review). The type of apraxia most pertinent to language is ideomotor apraxia, the inability to make purposive movements with either hand when the associated object is absent; for example, a patient able to use a hammer she is holding is unable to pretend to use a hammer. Here, the movement disorder is not explicable by weakness, lack of coordination, sensory loss, or incomprehension of commands (Geschwind 1975). Ideomotor apraxia unequivocally signals symbolic involvement resulting from a lesion in the left hemisphere, and it frequently co-occurs with aphasia. Also, impairments in the comprehension of meaningful gestures and pantomime occur almost exclusively in association with aphasia. These considerations have led to the proposal that aphasia is a disorder in conveying and comprehending symbols of any kind (Goldstein 1948).

Aphasia and ideomotor apraxia do not, however, invariably occur together, suggesting that they may be independent disorders, not manifestations of the same underlying defect in symbolization (Marshall 1980). Some investigators therefore postulate that aphasia and apraxia often occur together because of the anatomical proximity of the neural substrates responsible for language and gestural behavior (Goodglass and Kaplan 1979). Although the neural substrates of praxis are not well known, it does seem clear that both the left frontal and the left parietal lobes are particularly important for the control of learned motor activities. Geschwind (1965) proposed that (visual) imitation of gestures or the following of (auditory) verbal commands is first processed in their respective receptive areas; then messages are relayed to the motor association area of the left frontal lobe by means of the arcuate fasciculus. The left motor association area is connected to a similar area on the right by means of the corpus callosum, and each motor association area is connected to the primary motor area on the same side, which in turn affects the movement of the opposite limbs. Lesions that destroy the left motor association cortex or the anterior portion of the corpus callosum would "disconnect" the right premotor and motor areas from the left hemisphere, resulting in apraxia of the left hand. Apraxia may also result from a lesion in the left parietal lobe, a region that is thought to store visuokinesthetic

motor learning and to program the motor association cortex of the left frontal lobe for the necessary movements (Heilman 1979a). Apraxia, then, may result from a parietal or a frontal lesion in the left hemisphere or from a lesion disconnecting the left parietal from the left frontal lobe or from a corpus callosum lesion disconnecting the right premotor and motor areas from the left hemisphere.

Breakdowns in sign language and in nonlinguistic gesture suggest several new ways to investigate apraxia and its relation to aphasia. Because gesture and linguistic symbol are transmitted in the same modality in sign, the breakdown of the two can be directly compared.¹ The breakdown of speech, by contrast, involves disruption of a different channel (the vocal tract) from that of gesture (the hands). Therefore sign language lends itself to a more direct determination of whether or not both aphasia and apraxia result from an underlying asymbolia.

The multilayered nature of ASL provides a second vehicle for assessing the relation between apraxia and aphasia. A pervasive principle in ASL is the concurrent (rather than linear) conveyance of information. For example, it is superimposed changes in movement and spatial contouring of a sign stem that convey inflectional and derivational processes in ASL. A sign and its inflection co-occur in time rather than follow each other in linear succession. Because grammatical and lexical structures are displayed concurrently, grammatical errors within inflected signs allow a unique test of the hypothesis that aphasia is the result of an inability to program complex movements in sequence.

A third way in which the study of sign language might clarify the relationship between aphasia and apraxia comes from the fact that the movements of the articulators in sign are open to view and thus make language production directly available for analysis. By relating impairments in sign language to patients' control of nonlanguage movement and comprehension of gestures, we shed new light on the relationship between aphasia and apraxia.

6.2 *Deficits in Sign Language*

Although the signing of the three patients with left-hemisphere damage is clearly aphasic, their linguistic disorders are quite different,

1. Deaf ASL signers can easily distinguish meaningful gestures that are ASL signs from those that are not. The signal made by a policeman holding his hand up, palm forward, to indicate "stop" is a symbolic gesture for hearing and for deaf people alike, but it is clearly not a sign of ASL. The ASL sign STOP is entirely different. Thus one can distinguish gesture and language within one and the same channel.

involving impairment at different structural layers of the language. Even though the patients with right-hemisphere damage show severe left-sided neglect or serious impairment in their visuospatial capacities, all three are fluent and normal in their sign production.

As we have seen, Paul D.'s aphasia is shown primarily in an abundance of semantic and grammatical paraphasias and errors in spatial syntax. He often uses semantically bizarre constructions. He tends to make inappropriate use of morphologically complex forms where simple ones are the norm. Sometimes he substitutes one inflectional form for another. Grammatical and semantic paraphasias abound. Furthermore, Paul D. tends to avoid using spatial indexes, and when he does use them, he does so inconsistently, disregarding the requirement of the system of verb agreement in ASL.

Karen L.'s signing output is also rich and fluent. Her deficits in expression are confined primarily to two domains: sublexical structure and nominal reference. We did not find any tendency to make semantic or grammatical errors in her ongoing conversation; in this respect she is different from Paul D. In many ways her signing appears to be the least impaired of the left-hemisphere-damaged patients. However, she frequently fails to specify who or what is the subject of her freely and correctly used indexical pronouns; that is, she establishes indexes at abstract points in space but often fails to specify the nominals associated with the spatial indexes. Furthermore, Karen L. has a marked comprehension impairment.

Gail D.'s expressive signing output is the most severely impaired of the patients we have studied; her utterances are often limited to single signs. Her output is effortful, and she often gropes for the sign. There is no trace of the grammatical apparatus of ASL in her signing; signs are made singly and in uninflected form, with selection almost exclusively from referential open-class signs. On a variety of sign language tests, we found marked differences in her skills: Her comprehension of sign language is nearly normal, as is her visuospatial nonlanguage processing. Yet her expression of sign language is grossly impaired, in fact, agrammatic.

6.3 *Apraxia and Deaf Signers*

We administered two tests of apraxia: For nonrepresentational movements we used Kimura's Movement Copying Test (Kimura and Archibald 1974; Kimura 1982); for symbolic movements we used the ideomotor apraxia tests of the BDAE adapted for deaf signers. These tests evaluate movements of the cheeks and mouth (buccofacial) and

arm and hand movements; the arm and hand movements tested are both transitive (related to object manipulation) and intransitive (not involving objects, for example, waving goodbye). As before, all instructions came from a native ASL signer.

6.3.1 *Nonrepresentational Movement*

We used the slightly abbreviated form of Kimura's Movement Copying Test described in Kimura (1982). The task is to imitate movements of the hands and arms in unfamiliar and meaningless sequences (figure 6.1). These sequences are meaningless to signers as well as to nonsigners. The subject sees three movements to be imitated, all involving only one hand and arm. The first movement has an open hand with all fingers spread, positioned perpendicular to the body in front of the opposite arm. The hand is swept across the body from one side to the other. As the hand sweeps across the body, the extended fingers move from spaced apart to touching each other (figure 6.1a). This movement is scored for initial hand posture, initial hand orientation, lateral and straight movement, and proper hand closing. In the second movement the extended fingers and thumb of the hand

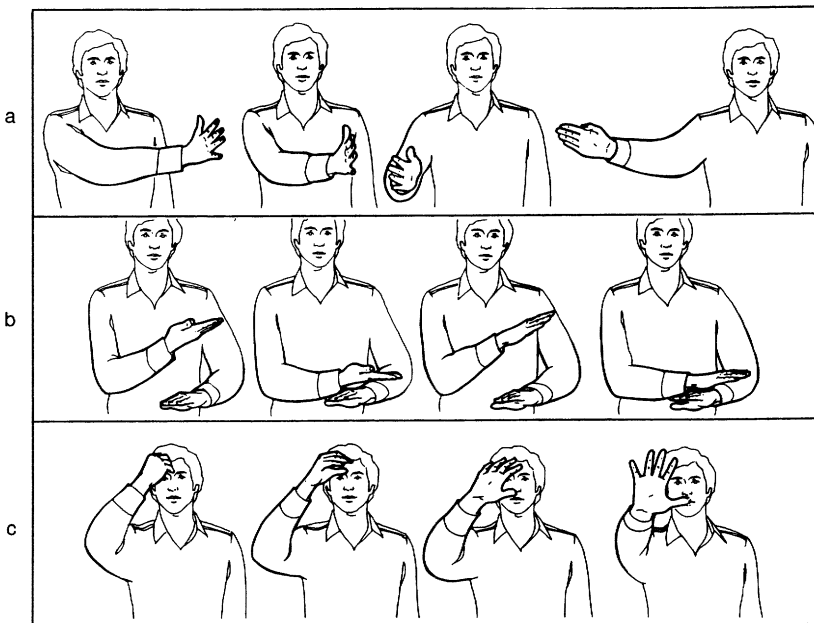


Figure 6.1
Nonrepresentational movements: Kimura Movement Copying Test.

are in contact, the back of the hand slaps the other forearm, rotates, and then the palm slaps the forearm (figure 6.1b). This movement is scored for hand posture, back slap, forearm rotation, and front slap. The final movement in the series starts with the fingertips and thumb together in a ring, all touching the forehead; then the hand moves out and away from the forehead, rotating and opening as it moves (figure 6.1c). This movement is scored for starting posture, forward and linear movement, forearm rotation, and hand opening. Two trials are given for each sequence. Each component of each sequence is given a score of two if performed correctly on the first trial, a score of one if performed correctly on the second trial, and a score of zero if not performed correctly on either trial. Each of the three sequences has four components that can be scored, so the maximum possible score is 24 points per hand.

Kimura (1982) reports data from 118 hearing patients with unilateral brain damage: 72 patients with left-hemisphere damage and 46 patients with right-hemisphere damage. Because many patients have one hand or arm paralyzed, we follow Kimura in reporting only scores for the hand on the same side as the lesion, where strength is typically unaffected. Table 6.1 presents the mean scores of the hearing patients from Kimura (1982) and the scores of our six deaf patients. The mean score of Kimura's hearing patients with left-hemisphere damage is 59 percent correct, whereas the mean score of the hearing patients with right-hemisphere damage is 78 percent correct, significantly higher. Kimura indicates that scores falling below a level of 90 percent of the mean score of the patients with right-

Table 6.1
Performance on the Kimura Movement Copying Test

Subject	Left-hemisphere-damaged patients' scores (percent correct)	Right-hemisphere-damaged patients' scores (percent correct)
Hearing patients		
(from Kimura (1982))	59 (mean)	78 (mean) ^a
Deaf patients		
Paul D.	92	
Karen L.	63	
Gail D.	71	
Brenda I.		83
Sarah M.		75
Gilbert G.		70.8

a. Ninety percent of this level is 70.2 percent; patients with scores below 70.2 percent are considered to be impaired.

hemisphere damage should be considered impaired. Table 6.1 shows that on the basis of this cutoff value only Karen L. is impaired; the other deaf patients are not.

The types of error that the deaf signing patients made are revealing. The left-hemisphere-damaged patients made errors on all components (hand configuration, movement, location, and orientation), but Brenda I., a right-hemisphere-damaged patient, made only spatial errors. For the first movement sequence (figure 6.1a), Brenda I. began the movement in the space near her midline rather than far to her left. Right-lesioned Sarah M. also made this error. Because both Brenda I. and Sarah M. manifest severe left hemispatial neglect in other situations, their spatial errors may be related to this condition. Brenda I.'s only other error was in the orientation of the hand with respect to the body, another spatial error.

6.3.2 *Representational Movement*

As in the BDAE, our adaptation of the tests of apraxia is divided into three sections: buccofacial movements, intransitive limb movements, and transitive limb movements. When subjects were unable to carry out a movement to command ("Show me how you would . . ."), the experimenter demonstrated the movement and asked the subject to copy it. We selected commands that would elicit gestures that differ markedly from corresponding signs, for example, "Show me how you would write your name." The gesture should involve configuration of the hand as if holding a pen or a pencil and movement characterizing writing. The ASL sign is radically different. When a subject was unable to copy a transitive limb movement, he or she was given the actual object and asked to show its use. Figure 6.2 presents the results of the ideomotor apraxia testing for each patient to each movement.

In making buccofacial movements, only left-lesioned patient Gail D. had difficulty. She failed to perform correctly four of the five movements to command and two of the movements to copy. When asked to demonstrate how to cough, she opened her mouth, signed VOMIT, then explosively mouthed "pop" as she moved her hand outward from her mouth. In trying to demonstrate a sneeze, she produced the sign SNEEZE (an opening and downward movement of the hand from the nose). For the movement for a kiss she mouthed "kiss," and in demonstrating chewing, she pursed her lips. The only gesture that she correctly performed to command was moving her eyes up; eye movement, however, forms a special category because it is represented primarily by the nonpyramidal motor system and is

	Left Hemisphere Damaged Patients						Right Hemisphere Damaged Patients						Controls					
	PD	KL	GD	BI	SM	GG	GW	RA	LL	W/Obj.	Imitation	Command	W/Obj.	Imitation	Command	W/Obj.	Imitation	Command
Buccofacial																		
cough			●															
sneeze			●															
kiss			●															
chew			●															
move eyes up			●															
Intransitive Limb																		
wave goodbye																		
signal "stop"																		
call a dog																		
Transitive Limb																		
throw a ball																		
clean a bowl																		
write name																		
start a car																		
cut meat																		


pass  fail 

Figure 6.2
Results for left- and right-lesioned signers and controls for the ideomotor apraxia test, which evaluates representational movements.

often preserved in hearing apraxic patients (Geschwind 1975). The right-hemisphere-damaged patients and the controls had no difficulty with buccofacial movement.

For intransitive limb movements Gail D. again had some difficulty, whereas the other left-hemisphere-damaged patients, the right-hemisphere-damaged patients, and the controls did not. Gail D. failed to perform two of the four movements to command correctly, although she was able to imitate these gestures correctly. As for the other patients, Paul D. performed all the gestures correctly; Karen L. and right-lesioned Brenda I. and Gilbert G. failed to perform one gesture to command ("Signal to stop"), as did Sarah M. ("Call a dog") and one elderly control patient ("Signal to stop"). Finally, Gail D. had severe difficulty with transitive limb movements, whereas the other patients did not. She correctly gestured only one of the five movements to command ("Clean a bowl"). Furthermore, her errors to two of the items were the classic apraxic error of using a body part as an object. When asked to write her name and to cut meat in gestures, Gail D. extended her index finger from her fist, as if representing an implement: a pen for carrying out a writing motion, and a knife for demonstrating how to cut meat. Gail D. was also unable to make correct imitations of the movements that she had failed to produce to command. And even in her imitations of the movements, she produced body-part-as-object errors for the commands "Write your name" and "Cut meat." She was easily able to make the movements when given the actual objects, so it was clear that she had no elementary motor disorder; rather, she quite clearly exhibited ideomotor apraxia.

Karen L. was unable to produce two of the gestures to command ("Write your name" and "Start a car"). She repeatedly used the sign SIGNATURE rather than the gesture but correctly copied the gesture. Karen L. seemed to show great difficulty comprehending the item "Show me how you start a car." She repeatedly tried to relate a story about the time she first started driving at age 12. She was, however, able to copy the gesture correctly.

Finally, right-lesioned Brenda I. was unable to perform correctly two of the five movements to command ("Write your name" and "Cut meat"). For these two commands she gave tangential descriptions without producing the correct gestures. For one of them, however, "Write your name," she did gesture correctly when the examiner demonstrated the starting position of the hand. For the other gesture, "Cut meat," we note that two control subjects also failed to produce the gesture correctly to command. Right-lesioned Sarah M. was also unable to produce the latter gesture to command,

but she easily imitated it. Right-lesioned Gilbert G. had no difficulty with performing any of the gestures to command.

6.3.3 *Pantomime Recognition*

Varney and Benton's (1978) Pantomime Recognition Test assesses the ability to understand meaningful, nonlinguistic gestural communication. The test consists of four practice items followed by a series of thirty videotaped pantomimes of a man miming the use of some common objects, such as a spoon, a pen, or a saw. From a test booklet containing four response choices per item, the patients are asked to point to the drawing depicting the object pantomimed. The four drawings for each test item include the correct choice (saw, for example), a semantic foil (an axe), a regular foil (an object whose use is pantomimed elsewhere on the test), and an odd foil (a train). Varney and Benton provide hearing patient norms for forty aphasic patients and for twenty control subjects without brain damage. Defective performance on the test is defined as performance below the level of the poorest control patient, which was 86.7 percent correct. By this criterion, 35 percent of the hearing aphasic patients had defective performance in pantomime recognition. The performance of all of our deaf patients fell in the normal range (figure 6.3): Paul D. scored 90 percent, Karen L. 86.7 percent, and Gail D. 100 percent correct. The two right-hemisphere-damaged patients who received the test, Brenda I. and Gilbert G., scored 93.3 percent and 100 percent correct, respectively.

6.4 *The Separability of Apraxia and Sign Aphasia*

In a long-standing controversy over the nature of aphasic disorders, certain investigators have proposed a common underlying basis for disorders of gesture and disorders of language. In this view disorders of language result from more basic disorders of movement control. The data we have obtained on apraxia and aphasia from six brain-damaged signers do not support either those who attribute the specialization of the left hemisphere specifically to the control of changes in the position of both oral and manual articulators (Kimura 1976, 1979) or those who claim that both apraxia and aphasia result from an underlying deficit in the capacity to express and comprehend symbols (Goldstein 1948). Instead, our findings suggest that sign language can break down along linguistic lines, independently of disorders of movement and gesture (both symbolic and meaningless).

In regard to representational gestures, the data clearly show that of

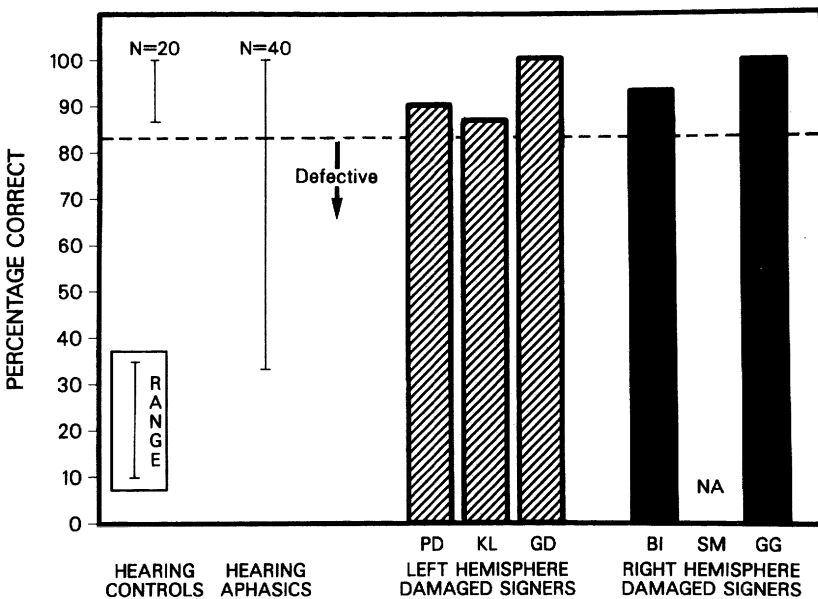


Figure 6.3

Performance of left- and right-lesioned signers on a test of Pantomime Recognition. The range of hearing control scores and of scores of hearing aphasics are shown for comparison. None of the deaf signers is impaired.

our six brain-damaged subjects only Gail D. has ideomotor apraxia. Gail D. has severe difficulty in producing and imitating movements to command. Furthermore, she produces the classic apraxic error of using a body part as an object, both to command and in imitation. It is interesting that her ideomotor apraxia can be predicted from her lesion, according to the Geschwind (1965) model. Her lesion affects the left motor and premotor areas as well as the anterior portion of the corpus callosum, resulting in right hemiplegia and apraxia on the left side. The other deaf patients we studied are generally able to make correct gestures to command, and all correctly imitate gestures presented to them. The few mistakes they did make in gesturing to command actually result from difficulties in comprehension. In fact, these patients make no errors in imitation, a task that does not require intact language comprehension.

Because ideomotor apraxia occurs with sign aphasia only for Gail D. (but not for the other two left-lesioned patients, Paul D. and Karen L.), we can dissociate the capacity for using the linguistic gestures of sign language from the capacity to produce and to imitate communicative but nonlinguistic gestures. In a similar vein, all the patients performed normally on a test of pantomime recognition; yet some showed impairment in the comprehension of ASL (as shown by

performance on the BDAE and on other sign language tests). Hence the deficits in sign comprehension are unlikely to be explained by a general loss in the comprehension of communicative gestures.

With regard to nonrepresentational movements, only Karen L. was impaired on Kimura's movement copying test (with a score less than 90 percent of the mean score of the right-hemisphere-damaged hearing patients). Gail D. scored within the 90-percent level of hearing subjects with right-hemisphere damage, and right-lesioned Brenda I., Sarah M., and Gilbert G. were unimpaired, as was expected. It is significant that Gail D., who is extremely impaired on tests of ideomotor apraxia, performed well on copying nonrepresentational movements. Clearly, her apraxia is based on a motor-symbolic deficit rather than on a motor-sequencing one.

It is interesting that Paul D. showed no impairment when we administered Kimura's Movement Copying Test, whereas four years after his stroke he was tested and reported to be impaired in copying these movement sequences (Kimura, Battison, and Lubert 1976). Because he has recovered his ability to imitate nonrepresentational movements, his remaining aphasia for sign language cannot be due to a more basic incapacity to make nonrepresentational movements of the hands and arms.

Both Karen L. and Paul D. have fluent sign output; yet they show a double dissociation of sign language components. It seems highly unlikely that a movement-sequencing deficit could account for their double dissociation of linguistic structures. Furthermore, Paul D.'s semantic paraphasias and Karen L.'s comprehension deficits are clearly not attributable to a movement disorder. Nor are Karen L.'s sublexical errors and Paul D.'s paragrammatisms in sign the product of a disorder in the sequencing of movements, because the components of a sign (Handshape, Place of Articulation, Movement) co-occur throughout the sign and because grammatical morphemes also occur simultaneously with it; that is, lexical stem and inflection co-occur in time. This is not to say that capacities for movement sequencing are not an important function of the left hemisphere (Kimura and Archibald 1974; Kimura 1979, 1982). Clearly, more cases are needed for a fuller understanding of the relationship between apraxia and aphasia for sign language. The language deficits of the three aphasic signers with left-hemisphere damage, however, are related to specific linguistic components of ASL, rather than to an underlying motor disorder or to an underlying disorder in the capacity to express and comprehend symbols of any kind. This *separation between linguistic and nonlinguistic functioning* is all the more striking because for sign language gesture and linguistic symbol are transmitted in the same modality.

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