Deficits of Nonverbal Communication in Aphasic Patients: A Test of Some Current Causal Theories

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The aphasiology literature of the past 100 years contains numerous observations that patients with aphasia exhibit impaired communication in the use of gestures, pantomime, and other nonverbal behaviors (e.g. Alajouanine and Lhermitte, 1963; Bay, 1964; Critchley, 1939, 1970; Finkelnberg, 1870; Goldstein, 1948; Head, 1926; Jackson (Head, 1915); Marie (Cole and Cole, 1971); Sarno and Sarno, 1969). However, only a small number of recent studies have systematically investigated nonverbal communication in aphasia. While this minority status of the nonverbal area is understandable because of our traditional linguistic view of aphasia and because verbal impairments constitute the most obvious, crippling aspect of the disorder, the study of nonverbal symbolic communication has theoretical and clinical implications of sufficient merit that it should not be ignored.

Experimental studies of nonverbal communication in aphasia have attempted to answer two questions. First, are patients with aphasia deficient in the area of nonverbal symbolic communication? And second, if they are deficient, why? Although it has been suggested that nonverbal symbol systems should be adopted to circumvent the verbal deficits imposed by aphasia (Chen, 1968; Eagleson, Vaughn, and Knudson, 1967; Gardner, Zurif, Berry, and Baker, 1976; Glass, Gazzinaga, and Premack, 1973; Goldstein and Cameron, 1952; Schlanger and Schlanger, 1970; Teitelbaum, 1950), the answer to the first question is a rather clear "yes." That is, aphasic patients, as a group, are deficient in nonverbal symbolic areas. Studies by Goodglass and Kaplan (1963) and Pickett (1974) have found aphasic patients to be inferior to non-brain injured and/or brain injured, non-aphasic patients on measures of pantomime expression. Duffy, Duffy, and Pearson (1975), Gainotti and Lemmo (1976), and Pickett (1974) have found patients with aphasia to exhibit deficiencies in the area of pantomime comprehension. To our knowledge, there are no published experimental findings which contradict these results.

The answer to the second question, "why are aphasic patients deficient in these nonverbal areas?," is not nearly so clear-cut. Currently, there are three primary theories which attempt to explain the nature of the relationship between aphasia and pantomime deficits. These theories are illustrated in the model shown in Figure 1. The first and most common interpretation is one proposed by Goodglass and Kaplan (1963). This theory states that the presence of nonverbal expressive deficits in aphasic patients merely reflects the presence of a co-occurring, but independent,

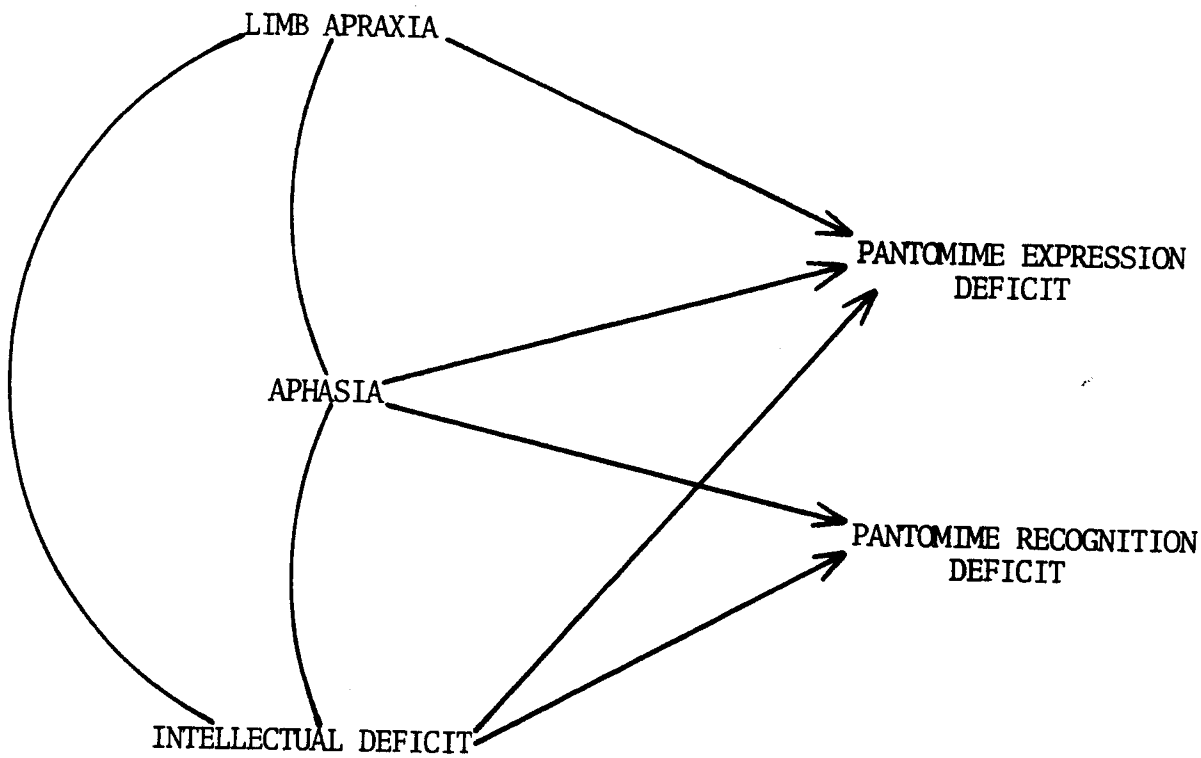

Figure 1. Model of explanations for pantomime deficits in patients with aphasia.

problem of motor programming—a limb apraxia. Therefore, as shown in the model, limb apraxia is said to cause, or explain, the presence of pantomime expression deficits. Since this theory does not claim that limb apraxia causes pantomime recognition deficits, limb apraxia and pantomime recognition are not connected in the model. The second and less commonly accepted interpretation states that nonverbal symbolic expression and recognition deficits are an integral part of the aphasic syndrome—a view which supports the notion that aphasia is a central symbolic deficit which is reflected in both verbal and nonverbal modes of communication. As shown in the model, then, aphasia is said to cause, or explain, the presence of pantomime expression and recognition deficits. A third and perhaps minor theory is one that states that expressive and receptive nonverbal deficits are the result of general intellectual deficit. In the model, therefore, intellectual deficit is shown to cause, or explain, pantomime expression and pantomime recognition deficits.

It also should be noted that limb apraxia, aphasia, and intellectual deficit are connected by curved lines in the model. These curved lines indicate that while limb apraxia, aphasia, and intellectual deficit may co-occur or be associated with one another, the relationships among them are not presumed to be causal ones.

The current study was directed toward clarifying the nature of the relationship between nonverbal (pantomime) deficits and aphasia by testing the validity of the three theories which are summarized in the model just presented. We will return to the model later to see which of the three theories is supported best by our data.

Methodology

Subjects

Although the study's primary focus was on aphasic patients, three groups of subjects were tested:

1. Twelve (12) non-neurologically impaired hospitalized adults.
2. Twenty-seven (27) patients with medical evidence of unilateral right hemisphere damage.
3. Forty-seven (47) patients with medical evidence of unilateral left hemisphere damage and a diagnosis of aphasia.

Data descriptive of the aphasic group can be found in Tables 1 and 2. There were no significant differences among the three groups of subjects in age, education, or handedness, and no significant differences between the aphasic and right hemisphere damaged group in terms of time-post-onset or distribution of etiologies.

Table 1. Descriptive Data for Age, Education, Time Post-Onset, Etiology and Handedness for the Aphasic Group.

	AGE	EDUCATION	TIME POST-ONSET (wks.)	ETIOLOGY	HANDEDNESS
N	46*	34*	46*	47	41*
	62.96	11.21	134.65	CVA=43	R=39
$\overline{X}$	62.96	11.21	134.65	CVA=43	R=39
s.d.	11.59	3.23	225.44	TRAUMA=3	L=2
Range	32-82	6-19	3-998	UNKNOWN=1	

*data unavailable for all patients

The data in Table 2, summarizing the PICA performance of our aphasic group, indicates that the group was probably quite representative of the aphasic population on the basis of its range, variability, and average performance on the PICA.

Table 2. Summary of Performance of the Aphasic Group (N=47) on the Porch Index of Communicative Ability.

	OVERALL PICA SCORE	OVERALL PICA PERCENTILE
$\overline{X}$	10.16	47.96
s.d.	2.57	25.99
Range	5.10-14.44	7-96

Test Measures

Five tests were used to generate information about the deficits under study:

Pantomime Recognition Test (PRT). This was a slightly shorter but otherwise identical measure of pantomime recognition ability described by Duffy, Duffy, and Pearson (1975). The test contains 46 items whose stimuli consist of pantomime actions performed by the examiner. The patient demonstrates recognition of the pantomimed act by pointing to a picture of the object (whose use was demonstrated without the actual object) from among four choices.

The performance of the aphasic group on the PRT was found to be significantly inferior ($p < .01$) to that of the control group and right hemisphere damaged group. This result is not surprising and supports the previously mentioned findings that aphasic patients are deficient in their nonverbal symbolic recognition ability.

Pantomime Expression Test (PET). On this measure patients were nonverbally conditioned to pantomime the use of 23 common objects when shown pictures of the objects. Responses were scored on a 16-point multidimensional scoring scale similar to that used for the PICA (Porch, 1967).

While the control and right hemisphere damaged groups performed similarly on the PET, the aphasic group was found to be significantly inferior ($p < .01$). Again, this finding is in agreement with the previously mentioned studies that have found patients with aphasia to exhibit pantomime expression deficits.

Manual Apraxia Test (MAT). This measure of the presence and severity of limb apraxia (Duffy, 1974) consists of 80 items which require the patient to imitate a variety of unilateral upper limb movements. A total of 252 movement components are scored on a 21-point multidimensional, interval level scoring scale.

Once again, the aphasic group performed more poorly ($p < .01$) than the control group and the right hemisphere damaged group. This finding is in agreement with several studies which have found left hemisphere damaged patients to exhibit limb apraxia in comparison to control subjects and/or right hemisphere damaged patients (DeRenzi, Pieczuro, and Vignolo, 1968; Duffy, 1974; Hecaen and de Ajuriaguerra, 1964; Liepman, 1913).

Raven's Progressive Matrices (RPM). This standardized intelligence test (Raven, 1940) was chosen as our measure of general intelligence. It is a non-verbal test which requires the patient to make visual discriminations and engage in visually-based reasoning processes, but it does not require verbal output or assembly capacity. Only simple pointing responses are required and nonverbal procedures were used to condition patients as to the nature of the task.

There were no significant statistical differences ($p > .05$) among the control, right hemisphere damaged and aphasic groups on this measure of intelligence. While this finding tends not to support the theory that intelligence is causal of pantomime deficits, it does not preclude the existence of a relationship between intelligence and pantomime deficits within the aphasic group.

Porch Index of Communicative Ability (PICA). The PICA (Porch, 1967) was used as our measure of general aphasic impairment. Only the aphasic group was given the PICA and its results were summarized in Table 2.

Results

The results of the group comparisons which were just presented support the conclusion that aphasic patients are deficient in pantomime abilities. They do not, however, answer the question about why pantomime deficits exist, although they make aphasic impairment and limb apraxia more likely explanations than general intelligence. To approach an answer to this question it is necessary first to examine the interrelationships among pantomime expression and recognition and the potential explanatory variables of aphasia, apraxia, and intelligence. The relevant correlations can be found in Table 3. As can be seen, all of these correlations are significant and several

Table 3. Pearson r Correlations Among the Raven's Progressive Matrices (RPM), Manual Apraxia Test (MAT), Porch Index of Communicative Ability (PICA), Pantomime Recognition Test (PRT), and Pantomime Expressions Test (PET) Based on the Performance of the Aphasic Group.

	RPM	MAT	PICA	PRT
MAT	.49			
PICA	.60	.64		
PRT	.49	.35*	.72	
PET	.51	.70	.89	.68

*p <.01 (for all other r's, p <.001)

are quite large. The PET is very highly correlated with the PICA (.89), but it is also strongly related to the MAT (.70). And the PET also is not insignificantly related to the Raven's (.51). Thus, these correlations not only lend support to the "aphasia theory" as the explanation for pantomime expression deficits, but also lend some credibility to the "apraxia theory," and somewhat less to the "intelligence theory." Likewise, the PRT is strongly related to the PICA (.72), which supports the "aphasia theory" as the causal explanation for pantomime recognition deficits, but intelligence cannot be ruled out as a possible contributor as well (.49). In addition, it can be seen that the PICA, the Raven's, and the MAT are all significantly related to one another. In sum, these simple correlations do not clarify the nature of the underlying relationships in question. They do make it clear, however, that the nature of the underlying interrelationships is quite complex.

We found three forms of data analysis which were particularly helpful to our understanding of the difficult-to-interpret correlations which were just presented. They were stepwise multiple regression, partial correlations, and a technique called path analysis. Time permits us to talk only about the results of partial correlations and path analysis, although all three forms of analysis are consistent in the types of conclusions which may be drawn from them.

Partial Correlations

Partial Correlations were useful in interpreting our findings because they allowed us to look at the relationship between two variables while controlling for the effects of other variables. Table 4 presents a number of the most important partial correlations which help to clarify the relationships among pantomime deficits, aphasia, limb apraxia, and intellectual deficit.

Table 4. Partial Correlations Examining the Relationships Among the Pantomime Recognition Test (PRT), Pantomime Expression Test (PET), Manual Apraxia Test (MAT), Raven's Progressive Matrices (RPM), and Porch Index of Communicative Ability (PICA).

CONTROL VARIABLE		PRT	PET
MAT	PICA	.69	.81
RPM	PICA	.61	.85
PICA	MAT	-.21*	.38
PICA	RPM	.11*	-.07*
PICA	PET	.12*	
MAT	PET	.65	
RPM	PET	.57	

*p > .05

When the correlation between the PRT and the PICA is controlled for the effects of limb apraxia or intelligence, there is very little change from the original, simple (non-partial) correlation. That is, when limb apraxia is controlled for, the correlation drops only to .69 (from .72), and when intelligence is controlled for, the correlation drops only to .61. These partial correlations suggest that aphasia and pantomime recognition are truly associated and are not related merely because of a common association they both may have with limb apraxia or intelligence. Also, when the relationship between pantomime recognition and limb apraxia and between pantomime recognition and intelligence is controlled for the effects of aphasia, the original, significant correlations drop very noticeably to nonsignificant levels (-.21 and .11 respectively). This suggests that the associations between pantomime recognition and limb apraxia and intelligence are due to the fact that pantomime recognition, limb apraxia and intelligence all share a good deal of common variance with aphasia, and not because limb apraxia or intelligence are causally related to pantomime recognition.

Similar conclusions are drawn when the partial correlations associated with the PET are examined. That is, the relationship between pantomime expression and aphasia is hardly affected when limb apraxia and intelligence are controlled for (r drops to .81 when limb apraxia is controlled for, and drops to only .85 when intelligence is controlled for). Also, when the effect of aphasia on the relationship between the PET and intelligence is controlled for, the original correlation drops to insignificant levels (-.07). Of even greater interest, when the relationship between pantomime expression and limb apraxia is controlled for the effects of aphasia, the correlation drops drastically from .70 to .38, indicating that the original strong relationship between limb apraxia and pantomime expression was obtained because they share a large common variance with severity of aphasia, and not because they have a strong cause-effect relationship with one another. This finding seriously challenges the Goodglass and Kaplan (1963) notion that pantomime deficits in patients with aphasia are primarily the result of limb apraxia.

Also of interest is the finding that when the original strong relationship between the PET and the PRT (.68) partials out the effects of the PICA (aphasia), the correlation drops to insignificant levels (.12). This suggests that the relationship between pantomime expression and pantomime comprehension exists not because of their common link to the nonverbal mode of communication, but rather because of their common association with aphasia.

Path Analysis

Another way of looking at the data is through a technique known as path analysis (for a more complete description of the technique see, for example: Duncan, 1966, 1970; Heise, 1969). While path analysis has not, to our knowledge, been used by aphasiologists, it is a very useful tool for theory testing because it allows for the testing of hypotheses about causal relationships among variables. While path analysis does not provide proof of a proposed causal relationship, it is quite valuable in assessing the plausibility of causal sequences which are generated by alternative theories. Statistically, the technique requires the interpretation of regression equations whose dependent and independent variables are determined by what is known as a path diagram. A path diagram is simply a model which lays out the presumed sequence of "cause" and "effect."

The model presented earlier in Figure 1 was essentially a path diagram whose assumptions could be tested through path analysis. Path analysis allowed us to test the validity and completeness of that model and to determine which, if any, of the proposed "causal" variables are the most potent cause of pantomime deficits in patients with aphasia.

The results of the path analysis are illustrated in Figure 2. The numbers on the curved lines are merely the simple correlations among the PICA, MAT, and RPM and represent the strength of their noncausal interrelationships. They are not particularly important to understanding the most salient results of the analysis. What are important are the numbers along each of the proposed causal paths. These numbers are know as path coefficients and their magnitude is indicative of the degree of causal relationship between the connected variables. E_1 and E_2 are estimates of residual or unknown influences on PRT and PET.

As can be seen, the path coefficients leading to PRT and PET show that intellectual deficit has a negligible effect on the two pantomime variables (PET = -.07; PRT = .09) and that limb apraxia has only a small effect on pantomime expression (.24). Aphasic impairment, on the other hand, can be seen as producing strong changes in pantomime ability in both the recognition

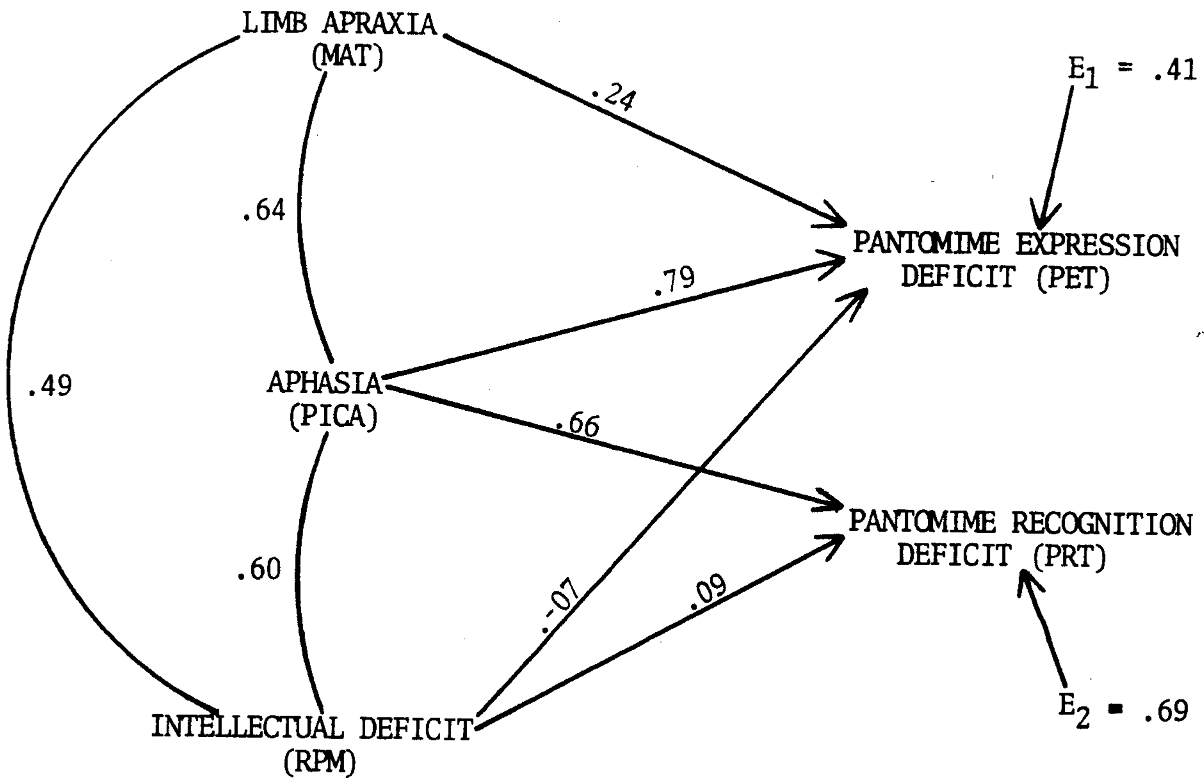

Figure 2. Results of path analysis of data relevant to explanations of pantomime deficits in patients with aphasia.

mode (.66) and expression mode (.79). Of the variables examined by this path analysis, then, aphasic impairment is shown clearly to have the strongest influence on pantomime deficits.

Summary and Conclusions

To summarize, the results of partial correlations and path analysis fail to support the notion of a strong causal relationship between limb apraxia and pantomime impairment, or between general intelligence and pantomime impairment. Our results do provide strong support for the existence of a potent relationship between pantomime deficits and verbal aphasic impairment.

It would appear from these findings that we need to seriously consider the idea that these nonverbal deficits are a reflection of a central symbolic disorder, and that aphasia, or at least some forms of aphasia, are not primarily, or exclusively, linguistic deficits. If this notion is correct, and aphasic impairment is reflected in both verbal and nonverbal symbolic areas, we should view with a healthy skepticism the rather numerous, undocumented recommendations to train aphasic patients in the use of signs, gestures, pantomime, drawing, etc. in an effort to circumvent their impaired verbal communication.

Finally, from the theoretical perspective, it seems that while the study of aphasia may contribute to our understanding of language, a purely linguistic model may not provide us with a complete understanding of aphasia. Luria (1973), for example, suggested that a complete understanding of aphasia is more likely to be found in the study of the psychological structures underlying the communicative process. For these reasons, it would appear that the continued study of nonverbal deficits in aphasia is warranted and that such study can make important contributions to our fuller understanding of the nature of the disorder.

References

Alajouanine, T. and Lhermitte, F. Non-verbal communication in aphasia. In A. DeReuch and M. O'Connor (Eds.), Disorders of Language. Boston: Little, Brown (1963).

Bay, E. Present concepts of aphasia. Geriatrics, 19, 319-331 (1964).

Chen, L.Y. Manual communication by combined alphabet and gestures. Archives of Physical Medicine and Rehabilitation, 52, 381-384 (1971).

Cole, M. and Cole, M. Pierre Marie's Papers on Speech Disorders. New York: Hafner (1971).

Critchley, M. The Language of Gesture. London: Arnold (1939).

Critchley, M. Aphasiology and Other Aspects of Language. London: Arnold (1970).

DeRenzi, E., Pieczuro, A., and Vignolo, L.A. Ideational apraxia: a quantitative study. Neuropsychologia, 6, 41-52 (1968).

Duffy, J.R. Comparison of brain injured and non-brain injured subjects on an objective test of manual apraxia. Unpublished Ph.D. dissertation, University of Connecticut (1974).

Duffy, R.J., Duffy, J.R., and Pearson, K.L. Pantomime recognition in aphasics. JSHR, 18, 115-132 (1975).

Duncan, O.D. Path analysis: sociological examples. American Journal of Sociology, 72, 1-16 (1966).

Duncan, O.D. Partials, partitions and paths. In Borgatta, E.F. and Bohrnstedt, G.W. (Eds.), Sociological Methodology 1970. San Francisco: Josey-Bass (1970).

Eagleson, H., Vaughn, G., and Knudson, A. Hand signals for dysphasia. Archives of Physical Medicine and Rehabilitation, 48, 410-414 (1967).

Finkelnberg, F. Niederrheinische Gesellschaft, Sitsung vom 21. Marz 1870 in Bonn. Berl. klin. Wschr., VII, 449-450, 460-462 (1870).

Gainotti, G. and Lemmo, M.A. Comprehension of symbolic gestures in aphasia. Brain and Language, 3, 451-460 (1976).

Gardner, H., Zurif, E.B., Berry, T., and Baker, E. Visual communication in aphasia. Neuropsychologia, 14, 275-292 (1976).

Glass, A.V., Gazzaniga, M.S., and Premack, D. Artificial language training in global aphasics. Neuropsychologia, 11, 95-103 (1973).

Goldstein, H. and Cameron, H. New method of communication for the aphasic patient. Arizona Medicine, 8, 17-21 (1952).

Goldstein, K. Language and Language Disturbances. New York: Grune and Stratton (1948).

Goodglass, H. and Kaplan, E. Disturbance of gesture and pantomime in aphasia. Brain, 86, 703-720 (1963).

Head, H.H. Aphasia and Kindred Disorders of Speech Vol. I. Cambridge, Eng.: Cambridge University Press (1926).

Head, H.H. Jackson on aphasia and kindred affections of speech. Brain, 38, 1-189 (1915).

Hecaen, H. and de Ajuriaguerra, J. Left Handedness. New York: Grune and Stratton (1964).

Heise, D.R. Problems in path analysis and causal inference. In Borgatta, E.F. (Ed.) Sociological Methodology 1969. San Francisco: Josey-Bass, 38-73 (1969).

Liepman, H. Motor aphasia, anarthria, and apraxia. Transactions of the 17th International Congress of Medicine, Sect. XI, Part II, 97-106 (1913).

Luria, A.R. The Working Brain. New York: Basic Books (1973).

Pickett, L. An assessment of gestural and pantomimic deficit in aphasic patients. Acta Symbolica, 5, 3, 69-86 (1974).

Porch, B. Porch Index of Communicative Ability. Palo Alto, Calif: Consulting Psychologists Press (1967).

Raven, J.C. Progressive Matrices: Instructions, Key, and Norms. London: H.K. Lewis and Co. (1940).

Sarno, J. and Sarno, M. Stroke: The Condition and the Patient. New York: McGraw-Hill (1969).

Schlanger, P.H. and Schlanger, B.B. Adapting role-playing activities with aphasic patients. JSHD, 35, 229-235 (1970).

Teitelbaum, I. Communication for the paralyzed. Medical Times, Nov. (1950).

Discussion

Q. Could you differentiate the kind of gesturing that goes on for pantomime purposes versus other kinds of gesturing that people might use naturally to communicate?

A. We define pantomime as the deliberate use of bodily or manual movement to convey a message in the absence of speech. Gesture is a somewhat broader term and would include movements, particularly of the hands and face, which accompany speech for purposes of emphasis.

Q. Which hand did your people use for their pantomime activity?

A. All of our aphasic subjects used their left hand.

Q. Is there any reason to believe that performance with one or the other hand might be different?

A. There are some theories which would predict occasional differences in spite of equal neuromuscular function of the two limbs. Geschwind, for example, would predict unequal performance of the two limbs in certain disconnection syndromes. But he speaks primarily of disconnections of verbal processing areas from areas where motor commands are carried out. Our procedure for eliciting pantomime avoided these problems becuase our patients were nonverbally trained as to the nature of the task and had only to respond to picture, not verbal, stimuli. We do not presently have data to demonstrate that there was or was not a difference in performance between the two limbs but, considering our methodology, we have no reason to suspect that significant differences would have occurred in those patients who were not hemiplegic or hemiparetic.