

CHAPTER

6

**Left-Hemisphere
Lesions: A
Comparison of
Subjects with and
without Aphasia**

John M. Pettit
Joseph R. Duffy

Numerous studies have explored the relationship between localization of cerebral damage and aphasia (Cappa and Vignolo, 1983; Damasio et al., 1979; Dewitt et al., 1985; Metter et al., 1981). Most of them begin from a theoretical perspective that aphasia can be classified according to major types, such as Broca's, Wernicke's, transcortical motor, and so on. Patients are often classified according to these basic types of aphasia and then brain-imaging information is collected, analyzed and correlated with the types of aphasia under investigation. Many of these studies support the view that lesions in different sites result in clinically different types of aphasia. Naeser and Hayward (1978) were among the first investigators to use computed tomography (CT) scan data to verify lesion sites for five groups of aphasic patients (Broca's, Wernicke's, global, conduction, and transcortical motor) as classified by the Boston Diagnostic Aphasia Examination (BDAE) (Goodglass and Kaplan, 1972). One of their conclusions was that "it was possible to predict the general lesion site from knowing the BDAE aphasia type. The reverse correlation is not yet as well defined" (p. 551).

Basso et al. (1985) used CT scans to examine "the exceptions to classical aphasia localizations" in 267 native Italian speakers. Among the 207 for whom language and CT scan data were compared, 36 represented "exceptions" to the expected type of aphasia based on the lesion data. Three were nonaphasic, even though each had extensive lesions in the classical speech area; seven had fluent aphasia with damage only to Broca's area; eight were fluent with massive anterior and posterior lesions; and six were nonfluent with lesions in Wernicke's area. This study, as well as others, indicates that the relationship between the classical aphasia types, lesion site, and language localization is, in their words, "still largely unknown" (p. 226).

It is likely that the most important barrier to more explicitly defining the relationship between cerebral damage and aphasia is not localization but the failure to adequately describe, in objective terms, those deficits which tell us *what* is being localized. It should be obvious that standardized, objective testing of speech and language functions in all modalities, and in as isolated a form as possible, is a necessity if we are to describe adequately what is impaired and what is intact (hence *what* is being localized).

In this investigation, we administered a large battery of speech and language tests to individuals with positive findings of a left-hemisphere lesion as determined by CT scan. The battery of tasks used in this investigation represents a significant methodological improvement over previous studies, in which tests have most often been used either to determine the presence or absence of aphasia or to classify patients according to different types of aphasia.

Three major issues were explored. The first was to compare in several

modalities the speech-language performance of individuals with and without aphasia who had lesions in the left frontal and frontal-parietal lobes to a control group of normal adults. Second, we examined the differences in performance across several speech-language tasks between aphasic subjects with left frontal lobe lesions and aphasic subjects with left frontal-parietal lobe lesions. Third, we explored the locus of lesions (lesion type, depth, and so on) that result in the presence or absence of aphasia in these two regions of the brain.

METHOD

SUBJECTS

Thirteen subjects with frontal lobe lesions (6 with and 7 without aphasia), 15 subjects with frontal-parietal lobe lesions (6 with and 9 without aphasia), and 7 normal non-brain-damaged adults were tested. Specific information regarding sex, age, etiology, the presence or absence of aphasia, and mean overall speech-language battery score [based on the Porch Index of Communicative Ability (PICA) (Porch, 1967) scoring] are summarized in Tables 6-1 and 6-2. Subjects with lesions were chosen for the study only if they had a positive CT scan with a single left-hemisphere lesion, no evidence of bilateral cortical damage, including cerebral atrophy, and were native English speakers who were 18 years of age or older and able to tolerate testing for a minimum of 45 minutes.

The age range for the 12 aphasic subjects was from 27 to 72 years, with a mean age for the group of 54 years. The age range for the 16 nonaphasic subjects was from 26 to 66 years, with a mean age of 46 years. The age range for the 7 subjects in the control group was from 40 to 72 years, with a mean age of 55 years.

Subjects who were diagnosed as having aphasia were seen by one of four speech pathology consultants at the Mayo Clinic. Owing to individual preferences, more than one test of aphasia was used in the diagnosis by a particular consultant. However, all diagnoses were made independently of the speech-language battery devised for this study.

CT SCANS

The diagnosis of a single left-hemisphere lesion was made by a Mayo Clinic neuroradiologist. The neuroradiologist measured and re-

TABLE 6-1. SUMMARY DATA FOR 28 SUBJECTS WITH A LEFT-HEMISPHERE LESION IN THE FRONTAL AND FRONTAL-PARIETAL LOBE AND THE MEAN OVERALL SCORE ON THE SPEECH-LANGUAGE BATTERY (PICA SCORING)

<i>Subject</i>	<i>Sex</i>	<i>Age</i>	<i>Lesion site</i>	<i>Etiology</i>	<i>Aphasia</i>	<i>Mean overall score</i>
1	M	63	Frontal	Infarct	Yes	13.33
2	M	71	Frontal	Infarct	Yes	13.37
3	M	33	Frontal	Tumor	Yes	13.00
4	F	27	Frontal	Cyst	Yes	13.77
5	M	53	Frontal	Infarct	Yes	13.33
6	F	62	Frontal	Infarct	Yes	12.87
7	F	62	Frontal	Infarct	No	14.33
8	F	53	Frontal	Tumor	No	14.60
9	F	47	Frontal	Tumor	No	14.42
10	M	66	Frontal	Infarct	No	14.05
11	F	43	Frontal	Infarct	No	14.28
12	F	35	Frontal	Tumor	No	14.67

13	M	30	Frontal	Tumor	No	14.39
14	M	53	Frontal parietal	Infarct	Yes	8.07
15	M	55	Frontal parietal	Tumor	Yes	11.42
16	F	41	Frontal parietal	Infarct	Yes	12.75
17	F	59	Frontal parietal	Infarct	Yes	9.41
18	M	55	Frontal parietal	Infarct	Yes	12.82
19	M	72	Frontal parietal	Hem.	Yes	11.12
20	M	48	Frontal parietal	Tumor	No	14.22
21	F	56	Frontal parietal	Tumor	No	14.05
22	M	62	Frontal parietal	Tumor	No	14.40
23	F	55	Frontal parietal	Infarct	No	13.59
24	F	47	Frontal parietal	Tumor	No	14.42
25	M	35	Frontal parietal	Tumor	No	13.87
26	F	43	Frontal parietal	Tumor	No	14.34
27	F	32	Frontal parietal	Tumor	No	14.48
28	F	26	Frontal parietal	Infarct	No	14.65

TABLE 6-2. DATA FOR SEVEN NORMAL NON-BRAIN-DAMAGED SUBJECTS AND THE MEAN ON SPEECH-LANGUAGE BATTERY (PICA SCORING)

<i>Subject</i>	<i>Sex</i>	<i>Age</i>	<i>Mean overall score</i>
1	F	56	14.48
2	M	40	14.65
3	F	72	14.20
4	F	41	14.65
5	F	54	14.63
6	M	61	14.50
7	F	61	14.30

corded the depth, size, and locus of the lesion, noted the structures involved in the lesion, and drew the lesion on a neurologic topographic sheet. All subjects with lesions also had a complete neurologic examination.

LANGUAGE BATTERY

The language battery in this study was administered within 48 hours of the subject's CT scan. It consists of 26 subtests, some of which use the 10 objects from the Porch Index of Communicative Ability (PICA) (Porch, 1967). Scoring is based on the PICA 16-point multidimensional scoring system. There are, however, a number of important differences between the present battery and the PICA. For example, there are tasks on the battery that involve written and verbal descriptions of actions performed with objects, tasks that assess motor speech ability, and tasks that assess auditory and visual discrimination.

RELIABILITY MEASURES

Ten subtests from the speech-language battery were correlated with 10 subtests on the PICA for nine subjects. Pearson product-moment correlation coefficients for these data on nine subjects ranged from .83 to

.98, with an overall (data combined) correlation coefficient of .91 ($p < .001$).

Interjudge reliability was addressed by having one of the two investigators score tape-recorded responses and the written data on five subjects. Interjudge reliability (Pearson r) for this procedure was .89 ($p < .01$).

It appears that the speech-language battery used in this investigation (although *not* intended as a test of aphasia) does measure similar language tasks as the PICA. Furthermore, investigators trained in the use of the multidimensional scoring system can use this speech-language battery reliably.

RESULTS

One-way analysis of variance for the two groups of left-hemisphere lesion subjects and the control group were performed for each subtest. The Duncan multiple-range test was performed for subjects with and without aphasia for each lesion site (frontal and frontal-parietal) and between both experimental groups and the control group for each of the 26 subtests (Winer, 1971).

NORMAL SUBJECTS

Results indicated statistically significant differences ($p < .01$) between aphasic and nonaphasic subjects and the normal control group for the subtests on the language battery. Figure 6-1 summarizes the performance across all 26 subtests for the group of seven normal (control) subjects. There are six subtests that were more difficult than others for the normal subjects. These include number 17—repeating a series of words; number 6—describing the manipulation of the objects; number 7—describing the function of the objects; number 9—writing a description of the action in manipulating the objects; number 12—pointing to the objects presented in a series; and number 15—pointing to objects in a series after the subject reads them aloud. Three of these six subtests require short-term memory and two require a description of the manipulation of the objects (similar to the Reporter's Test; DeRenzi and Ferrari, 1978).

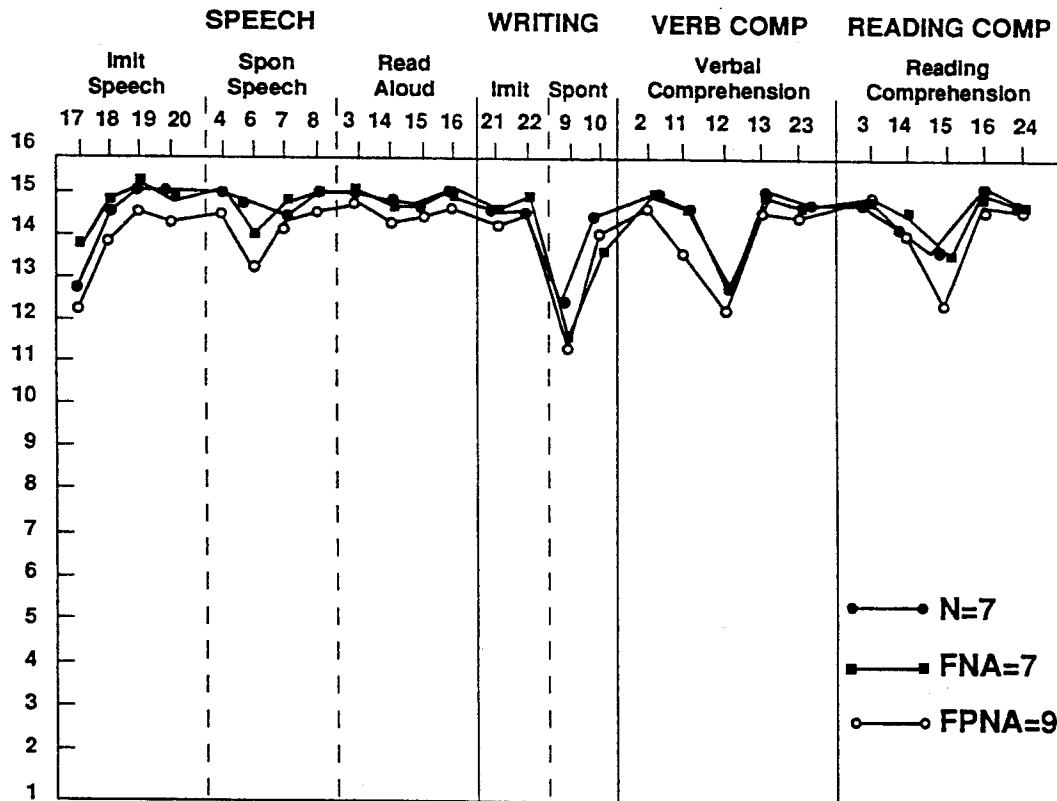


Fig. 6-1. Profiles for N = normal (control); FNA = frontal lobe, not aphasic; $FPNA$ = frontal-parietal lobe, not aphasic subjects.

APHASIC AND NONAPHASIC SUBJECTS WITH FRONTAL LOBE LESIONS

Results of the Duncan's multiple-range tests revealed statistically significant differences ($p < .05$) on 8 of the 26 subtests between aphasic and nonaphasic subjects with frontal lobe lesions. Figure 6-2 shows that the nonaphasic group performed significantly better than the aphasic group on three imitative speech tasks (number 17—repeating a series of words; number 18—repeating commands; and number 20—motor speech tasks), one spontaneous speech task (number 6—describing an action performed with the objects), and one reading aloud task (number 15—reading a series of two to six words). A significant difference in favor of the nonaphasic subjects was found for only one writing task (number 9—a written description of the movement of the objects), one verbal comprehension task (number 12—touching a series of objects on command), and one reading comprehension task (number 15—pointing to objects in a series read by the subject).

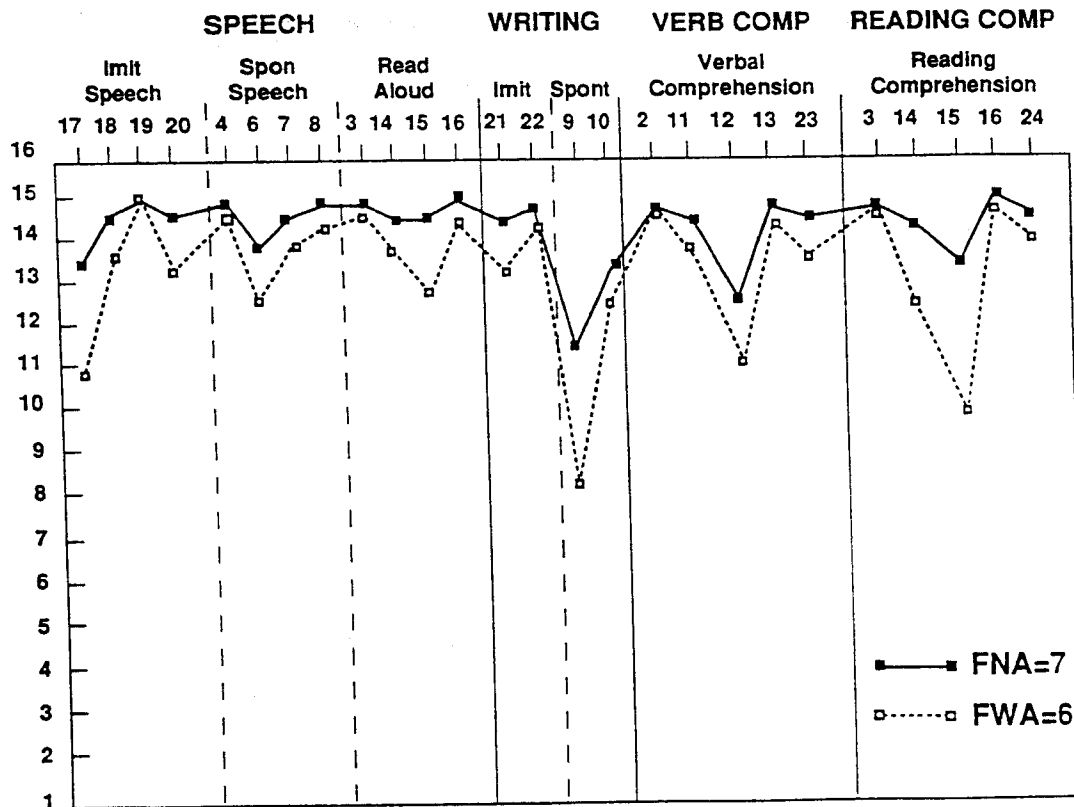


Fig. 6-2. Profile for *FNA* = frontal lobe lesion, not aphasic subjects and *FWA* = frontal lobe lesion, with aphasia subjects.

APHASIC AND NONAPHASIC SUBJECTS WITH FRONTAL-PARIETAL LOBE LESIONS

Analysis of the data for the subjects with frontal-parietal lobe lesions indicated that there were significant differences ($p < .05$) between aphasic and nonaphasic subjects on 24 of 26 subtests. These included the same 8 subtests that were found to be significantly different between aphasic and nonaphasic subjects with frontal lobe lesions as well as 16 other subtests. These differences are illustrated in Figure 6-3. The difference between those with and without aphasia was not found to be significant ($p < .05$) for subtests number 2 (pointing to single words) and number 3 (pointing to single words after reading words aloud).

NORMAL SUBJECTS AND NONAPHASIC BRAIN-DAMAGED GROUPS

Figure 6-1 summarizes the profiles for normal subjects and nonaphasic subjects with frontal lobe and frontal-parietal lobe lesions. There were

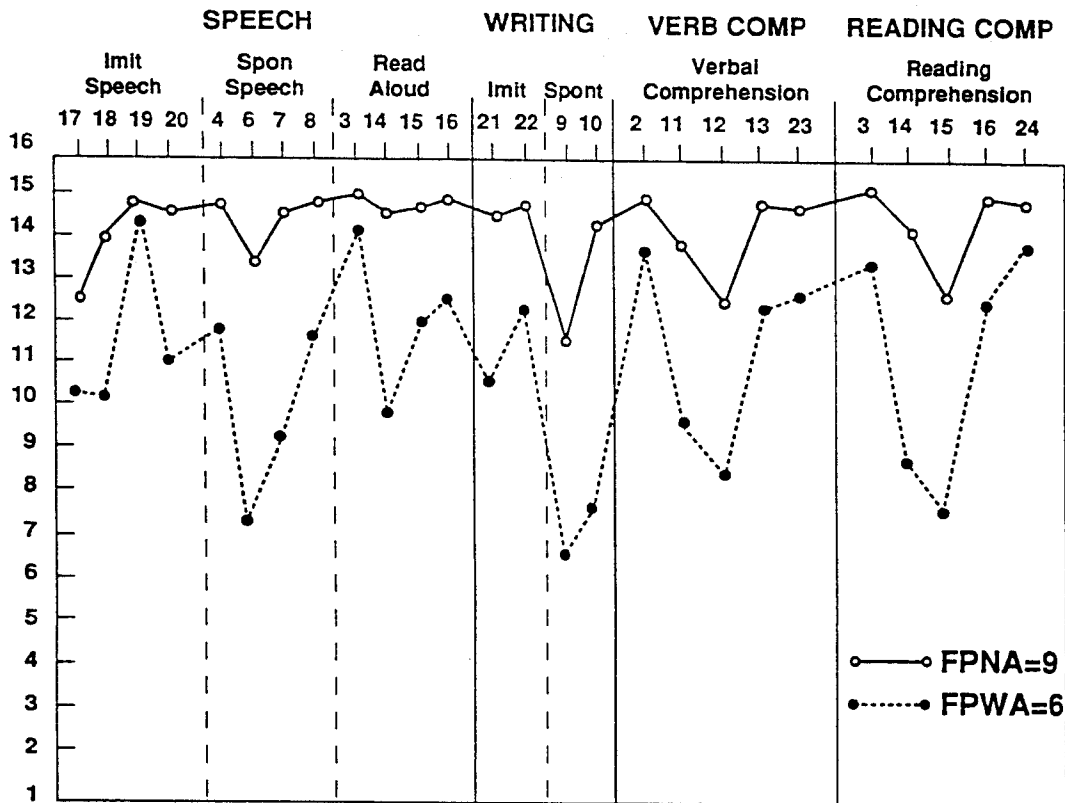


Fig. 6-3. Profile for FPNA = frontal-parietal lobe lesion, not aphasic subjects and FPWA = frontal-parietal lobe lesion, with aphasia subjects.

no statistically significant differences ($p < .05$) in the performance of the nonaphasic left-hemisphere lesion subjects and subjects in the normal non-brain-damaged group on any of the 26 subtests.

APHASIC SUBJECTS WITH FRONTAL AND FRONTAL-PARIETAL LOBE LESIONS

Statistically significant differences ($p < .05$) were found between aphasic subjects with frontal lobe lesions and aphasic subjects with frontal-parietal lobe lesions on 21 of the 26 subtests. That is, the aphasic subjects with frontal-parietal lobe lesions performed less efficiently on 21 of the subtests than did aphasic subjects with frontal lobe lesions. However, no statistically significant differences ($p < .05$) between the performance of the two aphasic groups were found for subtests number 17, 15, 2, 23, and 3.

DIFFERENCES BETWEEN BRAIN-DAMAGED GROUPS

The data were averaged for each major category on the battery that included the subtests for speech, writing, verbal comprehension, and reading comprehension for the two brain-damaged groups. These data were then submitted to the Duncan multiple-range test. A statistically significant difference ($p < .05$) between aphasic and nonaphasic subjects with frontal lobe lesions was found for speech but not for the other three major areas. Significant differences ($p < .05$) were found for all four major language areas between aphasic and nonaphasic subjects with frontal-parietal lobe lesions.

LESION DATA

Lesion data tend to support the observation that tumors are less likely to result in aphasia than infarcts. There were 13 subjects with tumors and 13 subjects with infarcts. Two aphasic patients had tumors (15% of the total), while 11 nonaphasic patients had tumors (85% of the total). Conversely, 8 of the aphasic patients had infarcts (62% of the total) and 5 of the nonaphasic patients had infarcts (38%).

Measurement of the lesions was calculated for the largest diameter in the axial plane. The x diameter for nonaphasic subjects with a lesion in the frontal lobe was 29.2 mm, while the x diameter for the aphasic subjects was 42.2 mm. For subjects with frontal-parietal lobe lesions, the x diameter for the nonaphasic subjects was 26.7 mm, and for the aphasic subjects it was 28.4 mm.

DISCUSSION

LESION DATA

In the case of frontal lobe lesions, larger lesions were found for nonaphasic subjects, but in frontal-parietal lobe lesions, larger lesions were found for aphasic subjects. Although no statistical treatment was performed on these data, the frontal lobe lesion subjects without aphasia had lesions that were most often anterior and superior, but in the aphasic subjects, the lesions were more posterior and inferior. Further-

more, the majority of the nonaphasic patients had tumors, while the majority of the aphasic patients had infarcts.

In the case of the two groups of subjects with frontal-parietal lobe lesions, the lesion size differences were not as great, but the aphasic subjects in this group were more severely aphasic than the aphasic group with frontal lobe lesions. One might suggest that the combination of the etiology (an infarct), involvement of both frontal and parietal lobes, and locus could account for the greater severity of aphasia in this group. As in the case of the aphasic frontal lobe patients, aphasic subjects with frontal-parietal lobe lesions had lesions that were more inferior than they were for the nonaphasic frontal-parietal lobe patients.

THE SPEECH-LANGUAGE BATTERY

In general, the speech-language tasks that provided the best discrimination between aphasic and nonaphasic subjects with left-hemisphere lesions were those requiring the repetition, reading, or comprehension of a series of words (pen-key-knife, etc.) and those requiring the subject to verbally describe, repeat, or write a description of an action performed by the experimenter. Some of these same tasks also were found to be more difficult for the control subjects.

DIFFERENCES BETWEEN BRAIN-DAMAGED GROUPS

Data examined in terms of the four major categories (speech, writing, verbal comprehension, and reading comprehension) would support the conclusion that aphasic subjects with frontal lobe lesions have a greater impairment in speech than in other language areas. Furthermore, aphasic subjects with frontal-parietal lobe lesions have difficulty with all four major language areas of speech, writing, verbal comprehension, and reading comprehension.

When the data were averaged for each of the four major areas, no significant differences were found among the nonaphasic frontal-lobe-damaged subjects, nonaphasic frontal-parietal-lobe-damaged subjects, and normal (control) subjects for the four major areas of speech, writing, verbal comprehension, and reading comprehension. These results tend to support the contention that a lesion in the classical speech area does not inevitably result in aphasia or even in a significant impairment in language functions.

CONCLUSIONS

Based on the results of this study, we offer the following conclusions. First, relatively large lesions in areas of the brain associated with language functions do not necessarily result in aphasia. Second, damage to the frontal and frontal-parietal lobes does not inevitably result in significantly impaired language functions compared to those of normal non-brain-damaged subjects. Third, tumors are less likely to result in aphasia than infarcts. Fourth, aphasic subjects with frontal lobe lesions demonstrate more impairment in speech tasks than in writing, verbal comprehension, and reading comprehension than nonaphasic frontal lobe lesion subjects. Fifth, aphasic subjects with frontal-parietal lobe lesions demonstrate more impairment across speech, writing, verbal comprehension, and reading comprehension than nonaphasic frontal-parietal lobe lesion subjects.

Clinical applications of this study suggest that tasks such as those on the speech-language battery used in this investigation could be included in aphasia testing for better differentiation of the aphasic from the nonaphasic patient. Specifically, tasks that require the subject to describe the manipulation of objects by writing or describing what and how objects were manipulated as well as the repetition, reading, and pointing to a series of objects should be considered as stimuli to help differentiate the aphasic from the nonaphasic patient. Additional research similar to the present study with subjects having other types of lesions, locations, and extent of cortical involvement will add to our understanding of the relationship of aphasia, lesion site, and language localization.

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