Language Deficits After Apparent Clinical Recovery from Childhood Aphasia

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Twenty-seven children with childhood injury to the left hemisphere were tested for language function and compared with appropriate controls. Eleven children had incurred their lesions before the age of 1 year, 16 afterward. The group with perinatal injury to the left hemisphere did not show a specific aphasic deficit even though they were mildly cognitively impaired. The group of children with later injury to the left hemisphere showed aphasic deficits if the original injury had caused a language defect; otherwise the left hemisphere injury was not associated with specific disturbances in language function. The average age at time of lesion in those children who had recovered from aphasia was 4.7 years. We conclude that even when childhood aphasia results from a unilateral nonprogressive lesion, recovery of language is less complete than has been generally supposed.

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In spite of its relative infrequency, acquired aphasia of early childhood has been a subject of great interest to those studying language and the brain. Observations on the incidence, duration, prognosis, and anatomical correlations of such early aphasia have formed the basis for theories on the origin and development of hemispheric specialization. It has been widely accepted that childhood-onset aphasias differ in four respects from adult-onset aphasia, in that the childhood form: (1) occurs with greater relative frequency with right hemisphere lesions; (2) is motoric, or expressive, in type; (3) is ordinarily of short duration; and (4) does not persist if it occurs before a certain age threshold [2, 8, 15].

Woods and Teuber [14], analyzing 64 cases of early-onset aphasia of their own and reviewing reports of 760 cases from the literature, questioned several of these generalizations. They noted that the frequency of aphasia with well-localized right hemisphere lesions was low in the ten recent series they analyzed, that there are cases of jargon aphasia in early childhood, and that the duration of aphasia before ultimate recovery can be more than two years in the worst cases.

The results of that study were consistent with at least the concept of an age threshold for recovery, in that no patient who was under 8 years of age when the lesion occurred was still clinically aphasic when examined neurologically at the time of the study. Nevertheless, passing note was made of two difficulties with the age threshold concept. First, estimates of the threshold vary from as late as the onset of adolescence to as early as age 5 years. Second, it is possible that if speech measures utilizing normal controls as a standard were used to look at a group of patients, subtle language deficits might be detected. Alajouanine and L'Hermitte's [1] data on acquired aphasia in children had suggested that in spite of apparent clinical recovery, deficits in the use of language in a school setting still persisted. And Dennis and Kohn [5] believed that language cannot fully develop in the absence of an intact left hemisphere. The latter authors compared patients who had had early left and right hemispherectomies and showed that even when verbal IQ scores were the same, and in the normal range, the left hemispherectomy group had more difficulty on complex language tasks. In these cases the original lesions antedated speech acquisition and involved the whole left hemisphere, so there was no question of incomplete recovery from late-acquired aphasia.

The goal of the present study was to look specifically for the presence of subtle language deficits that persist after left hemisphere lesions incurred during infancy or childhood (prior to adolescence), but two further questions were addressed: Are such deficits more pronounced when the damage to the left hemisphere is incurred after, rather than before, the age of onset of speech acquisition (taken here to be 1 year of age)? And are such deficits dependent on previous aphasia, or can they arise from the general effects of left hemisphere lesions, even

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lesions that did not cause an initial clinical aphasia? Patients with early left hemisphere brain lesions were given a series of special language tests in an attempt to answer these questions.

Materials and Methods

The study population consisted of individuals who had sustained a single, unilateral, nonprogressive cerebral lesion in the perinatal period, in infancy, or in childhood [13]. Twenty-seven patients with left hemisphere lesions were available for clinical neurological examination and special language testing. They were divided into one group of 11 whose lesions had occurred before their first birthday (presumed perinatally), and another group of 16 whose lesions were incurred later. This dividing point was chosen as a conservative lower limit to the time during which a child's readily observable language acquisition takes place. Tables 1 and 2 describe the patients' handedness, sex, age at lesion and at testing, etiological factors, localization, verbal and performance IQ scores, degree of paresis, and presence or absence of sensory deficits.

Several points should be noted. For the group with early lesions the mean age at time of lesion was estimated at 0 years; the mean age at testing was 17.8 years (median, 16.4; range, 10.2 to 25.5 years). For the group with later lesions the mean age at time of lesion was 5.7 years (range, 1.2 to 15.1 years), while the mean age at time of testing was 15.3 years (median, 13.8; range, 8.6 to 24.5).

All patients were examined neurologically by one of the authors (B. T. W.) at the time of the study. This examination included assessment of language comprehension, naming, repetition, spontaneous speech, reading, and writing. Two of the 16 patients with late lesions were noted on the basis of this examination to be aphasic. (This group of patients largely overlaps those described in a previous report [14] but differs slightly because 1 patient previously noted to have persistent aphasia was not available for the special tests of language.)

All patients in both groups were tested at the time of the study with the Wechsler Intelligence Scale for Children (WISC) [11] or, if they were 16 years of age or older, with the Wechsler-Bellevue Intelligence Scale, Form II [10]. The two groups had closely comparable mean full-scale IQ scores (early group, 86.5 ± 20.1 SD; later group, 89.5 ± 22.1); they had almost identical mean verbal IQ scores (early group, 90.3 ± 19.5 SD; later group, 90.1 ± 24.8).

Controls for the special language tests were 48 normal children of average intellectual ability as judged by their teachers and guidance counselors. They were in grades 5, 7, 9, and 11 when tested, and the sexes were equally represented at each grade level. While some of the tested patients were older than the range covered by the controls, they were all 25 years or younger, and intellectual functions of the type tested are known to be stable in this age range [3]. Accordingly, for purposes of matching patient ages to control ages, the patients between 18 and 25 years were considered to be 17. It was not possible to obtain Wechsler IQ scores for the controls.

Procedures

There were eight separate probes for language deficits.

PICTURE NAMING. The subjects were shown the Oldfield-Wingfield line drawings [9]. All subjects saw the

Table 1. Study Patients with Hemiparesis Acquired in the Perinatal Period[a]

Patient No. and Sex	Handedness After Lesion	Age at Testing (yr and mo)	Possible Etiological Factors	Findings When Tested				
				VIQ	PIQ	Hemiparesis	Somatosensory Loss	VFD
1, F	L	10.2	Neonatal seizures	60	47	Moderate	Yes	RUQ
2, F	L	16.4	Difficult labor and delivery	94	103	Mild	Yes	No
3, M	L	17.1	Premature	86	90	Severe	Yes	No
4, M	L	15.3	?Early trauma; congenital	70	78	Severe	Yes	No
5, F	L	21.9	Idiopathic	101	89	Severe	Yes	No
6, M	R	23.3	Difficult labor and delivery	96	78	Mild	Yes	No
7, M	L	15.4	Difficult labor and delivery	91	80	Severe	Yes	No
8, F	L	25.5	Premature	110	104	Severe	Yes	RVF
9, M	L	14.3	Seizures and hemiparesis	60	62	Severe	Yes	No
10, M	L	21.9	Idiopathic	112	110	Mild	Yes	No
11, M	L	14.6	Difficult labor and delivery	113	97	Mild	Yes	No

[a]In all 11 patients the lesion was localized in the left cerebral hemisphere.

VIQ = verbal intelligence quotient; PIQ = performance intelligence quotient; VFD = visual field defect; RUQ = right upper quadrant; RVF = right visual field.

Table 2. Study Patients with Hemiparesis Acquired After the First Birthday[a]

Patient No. and Sex	Handedness		Age at Lesion (yr and mo)	Age at Testing (yr and mo)	Cause of Lesion	Lesion Localization	Findings When Tested				
	Before Lesion	After Lesion					VIQ	PIQ	Hemiparesis	Somatosensory Loss	Aphasia Status
12, F	?	L	1.2	8.6	CVA	LMCA	74	82	Moderate	Yes	Never aphasic
13, F	?	L	1.2	10.4	Seizures of unknown cause	LCH	58	62	Mild	No	Never aphasic
14, F	?R	L	1.7	24.5	Tuberculous meningitis with CVA	LMCA	61	81	Severe	Yes	Recovered
15, F	R	L	2.3	9.2	CVA	LICA	89	94	Mild	No	Recovered
16, F	?L	L	2.5	16.0	CVA	LMCA	126	117	Mild	Yes	Never aphasic
17, F	R	L	2.8	10.6	Tetralogy of Fallot; hemiparesis 11 days after Blalock shunt	LMCA	85	80	Severe	Yes	Recovered
18, F	?	R	3.5	11.8	R weakness after T&A; ?hypotension	Left cerebral watershed	90	90	Mild	No	Never aphasic
19, M	?L	L	5.1	22.4	CVA	LMCA	82	89	Severe	Yes	Recovered
20, F	?	L	5.2	13.6	?Contusion; ?mild CVA; ?earlier lesion	LCH	105	111	Mild	Yes	Never aphasic
21, M	R	L	6.3	21.0	Carotid artery clamped to treat LMCA aneurysm	LICA	82	83	Moderate	Yes	Recovered
22, F	R	L	7.2	13.8	CVA	LICA	105	94	Severe	Yes	Recovered
23, M	R	R	7.8	13.6	CVA	LMCA	147	111	None	No	Recovered
24, F	R	L	8.1	19.3	Seizures; hemiparesis	LMCA	59	63	Mild	Yes	Still aphasic
25, M	R	L	8.6	12.8	CVA	LMCA	72	86	Severe	Yes	Still aphasic
26, M	R	R	13.8	17.0	Abscess, surgically drained	Left anterior frontal region	90	85	None	Yes	Never aphasic
27, M	R	R	15.1	20.7	CVA; history of migraine	?LMCA	117	104	Mild	Yes	Never aphasic

[a]None of the 16 patients had visual field defects.

VIQ = verbal intelligence quotient; PIQ = performance intelligence quotient; CVA = cerebrovascular accident; LMCA = left middle cerebral artery territory; LCH = left cerebral hemisphere; LICA = left interior carotid artery territory; T&A = tonsillectomy and adenoidectomy.

pictures in the same order and were asked to name each object. Responses to both the 10 warm-up pictures and the 26 test pictures were recorded and scored as right or wrong. No attempt was made to analyze type of error, and latencies were not recorded.

SPELLING. Subjects were asked to spell aloud eight words: *tag, year, house, story, borrow, stretch, disaster,* and *parallel.* The response for each word was simply recorded as right or wrong.

RHYMES. Subjects were asked to complete the line, or give the next line, or six orally presented nursery rhymes, as follows:

1. Jack be nimble _____
2. Mary had _____
3. Hickory, dickory, dock _____
4. Georgie Porgie _____
5. Little Boy Blue _____
6. Little Jack Horner _____

Responses were judged as right or wrong.

SENTENCE COMPLETION. Subjects were given the sentence completion section of the Boston Diagnostic Aphasia Examination [7], in which they were instructed to read sentences silently and select one of four proffered choices of a word or phrase to complete the sentence.

RELATIONS TASK. Subjects were instructed that the examiner would read aloud a series of statements about family relationships in the *examiner's* family, the task being to decide whether the statement could be true or was certainly false. The subjects were then read twelve sentences, each of which was to be judged (possibly) true or (certainly) false. Subjects could request that the sentences be read again. The answers to all statements could be determined on semantic or syntactical grounds alone and did not require any specific knowledge about the examiner's family. Examples: My father's sister is my aunt, or My brother's father is the same person as my father's brother. Responses were scored as correct or incorrect.

THAT-CLAUSE SYNTAX. A series of twenty sentences, each containing a relative clause beginning with *that*, was read to the subjects. Twelve of the sentences were anoma-

lous because of subject-object reversal either within or across relative clause boundaries (e.g., The boat that built the boy went in for lunch) while the other eight were correct. Subjects had to say whether a sentence was correct or incorrect ("OK" or "silly") and correct the anomaly. Scores analyzed in this report were simply the number of sentences correctly determined to be acceptable or unacceptable (i.e., the sum of false positive and false negative responses).

ASK-TELL DISTINCTION. This task is based on the work of Chomsky [4] on the developmental stages children go through in learning the distinction between usage of the verbs *ask* and *tell*. For the version used in this study, puppets were used as props, and the subjects were instructed first to ask or tell the puppets various things, and then to assume the part of the puppet and indicate how the puppet would ask or tell something. The test was scored on items correct as well as levels of discrimination achieved (as per Chomsky).

TOKEN TEST. The full token test of DeRenzie and Vignolo, in its English form [6], was given to each subject. The test consists of 62 commands related to cut-out paper shapes of different color, size, and geometric form. A single command might be: Touch the large red circle and the small blue triangle. Scoring was on the basis of sentence errors, meaning the number of commands executed incorrectly in any respect.

Data Analysis

The two groups of patients, who differed in mean age at time of testing, were compared on each test to school controls who most closely matched them in age. In this matching, all patients over 17 years were counted as 17 (the oldest patient was, in fact, 25 when tested). This was viewed as a conservative approach because the patients were thus compared to a younger control group on tests in which performance tends to improve with age. The group of patients with early lesions, whose mean age at testing was in this way set at 15.8 years, was compared to the 24 oldest controls (mean age, 15.7 years), while the group with later lesions, with a mean age set at 13.9, was compared to the 48 oldest controls (mean age, 14.0). Comparison was by the Mann-Whitney test.

Results

Table 3 shows the Z-scores and levels of significance when the two patient groups are compared to the appropriate controls. The group with later lesions was significantly impaired ($p < 0.05$) relative to controls on six of the tests, while the group with early lesions was significantly worse on only one test—spelling. Even if the 2 patients who were still clinically aphasic when tested are excluded, the significantly low scores of the group with later lesions are still maintained on all six tests. The difference between the two patient groups indicates that childhood lesions of the left hemisphere have a greater effect on language function when they come after rather than before the first birthday; that is, during or following the period of rapid speech acquisition.

Table 3. Results of Mann-Whitney Comparisons of Patient Groups versus Normal Controls, Expressed as Z-Scores

Test	Early Lesion Group (N = 11)	Late Lesion Group (N = 16)
That-clause	0.78 (NS)	2.81[a]
Sentence completion	0.04 (NS)	1.67[b]
Picture naming	−0.56 (NS)	2.97[a]
Token test	1.42[c]	2.21[a]
Spelling	2.82[a]	2.95[a]
Relations	0.02 (NS)	2.02[b]
Ask-tell	0.89 (NS)	1.42[c]
Rhymes	−0.15 (NS)	0.45 (NS)

Levels of significance: [a]$p \leq 0.01$; [b]$p \leq 0.05$; [c]$p \leq 0.10$. NS = not significant.

Table 4. Results of Mann-Whitney Comparisons of Patients Who Recovered from Aphasia and Those Who Were Never Aphasic versus Normal Controls, Expressed as Z-Scores

Test	Recovered Aphasic (N = 7; VIQ = 88.4)	Never Aphasic (N = 7; VIQ = 94.3)
That-clause	2.17[b]	1.54[c]
Sentence completion	2.40[a]	0.09
Picture naming	2.80[a]	1.26
Token test	1.37[c]	1.25
Spelling	2.14[b]	1.50[c]
Relations	0.80	0.70
Ask-tell	0.59	0.53
Rhymes	1.36[c]	1.20

Levels of significance: [a]$p \leq 0.01$; [b]$p \leq 0.05$; [c]$p \leq 0.10$.

Further analysis of the group with later lesions also permits assessment of the specific effects of previous aphasia on current language performance. Of those 16 patients, 2 became aphasic after lesions at ages 8.1 and 8.6 years, and remained aphasic; 7 became aphasic after lesions at ages 1.7, 2.3, 2.8, 5.1, 6.3, 7.2, and 7.8 years but recovered clinically; and the remaining 7 incurred lesions at ages 1.2, 1.2, 2.5, 3.5, 5.2, 13.8, and 15.1 years but were never aphasic. Table 4 shows the language test results for the latter two groups. When compared to controls, the 7 recovered aphasics (mean verbal IQ, 88.4; mean adjusted age at testing, 14.0 years) were significantly impaired ($p < 0.05$) on four tests: picture naming, spelling, that-clause syntax, and sentence completion. In contrast, the 7 patients who were never aphasic (mean verbal IQ, 94.3; mean adjusted age at testing,

13.5 years) did not score significantly below controls on any of the language tests. Thus, in the entire group with later lesions, the special difficulties on the language tests are primarily attributable to those patients who had developed aphasia with their original lesions. These problems may reflect incomplete recovery from aphasia that is not apparent on individual examination. Whether the difference in mean verbal IQ scores, amounting to 5.9, explains the difference in results between the patients who were never aphasic and those who recovered from aphasia or whether language factors not reflected in verbal IQ scores also contribute cannot be determined. This problem was avoided in the initial analyses contrasting patients with earlier lesions and those with later lesions because the verbal IQ scores of the groups were almost identical.

Discussion

The previously reported results of clinical observation of the patients in this study indicated that all those whose lesions had occurred before age 8 years had recovered language function [12], and that result seemed consistent with the classic concept of an age threshold for recovery [8]. The current study, however, raises doubts as to whether the age of 8 years, or any age after the onset of overt speech acquisition, can really be viewed as a threshold before which full language recovery can be expected. The current study indicates that left hemisphere lesions, if incurred before 1 year of age, do not result in significant impairment on a variety of language tasks, but left hemisphere lesions after 1 year, if they cause initial aphasia, leave significant residual impairment on most of the same language tasks. The observation that recovery of language functions after childhood lesions involving the left hemisphere speech areas is best after very early lesions, intermediate after lesions before age 8 years, and least after later lesions suggests that there is an inverse relationship between age at lesion and degree of language recovery which is at least stepwise rather than all-or-none. (We could not, however, demonstrate a significant correlation of age at lesion and level of language test performance for the 7 recovered aphasics, perhaps because of their wide variation in IQ scores and ages at testing.)

It should be noted that the group of patients with left hemisphere lesions incurred prior to the age of 1 year showed evidence of impairment of higher function. Compared to sibling controls, their verbal IQ scores were significantly lower, and the same is true for the group of patients with later left hemisphere lesions [12]. Moreover, other variables such as extent of lesion may also be important, since patients with very early left hemisphere lesions who have had complete hemispherectomies show impairment on special language tests [5].

The assumptions that aphasia is common after right hemisphere lesions in childhood and that there is a threshold age for language recovery led to the hypothesis that both hemispheres initially play an active role in speech, but that the left hemisphere generally assumes more and more dominance until the right hemisphere loses all active role in speech and can no longer compensate for a left-sided lesion. The contrary observation of infrequent aphasia after right hemisphere lesions and of a stepwise decline in language recovery capacities after left-sided injury make it more likely that in most individuals, the left hemisphere has the leading role in speech from the outset; the right hemisphere has only the potential for language, a potential which declines during childhood as the right hemisphere becomes increasingly committed to other functions.

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