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ACQUISITION OF SIGNS FROM AMERICAN SIGN LANGUAGE IN HEARING INDIVIDUALS FOLLOWING LEFT HEMISPHERE DAMAGE AND APHASIA

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Abstract—Three severely aphasic hearing patients with no prior knowledge of sign language were able to acquire competency in aspects of American Sign Language (ASL) lexicon and finger spelling, in contrast to a near complete inability to speak the English counterparts of these visuo-gestural signs. Two patients with damage in left postero-lateral temporal and inferior parietal cortices mastered production and comprehension of single signs and short meaningful sign sequences, but the one patient with damage to virtually all left temporal cortices was less accurate in single sign processing and was unable to produce sequences of signs at all. These findings suggest that conceptual knowledge is represented independently of the auditory–vocal records for the corresponding lexical entries, and that left anterior temporal cortices outside of traditional "language areas" are part of the neural network which supports the linkage between conceptual knowledge and linguistic signs, especially as they are used in the sequenced activations required for production or comprehension of meaningful sentences.

UNTIL RECENTLY, virtually all information regarding the brain basis of linguistic abilities has come from the study of spoken languages. There is little doubt that aspects of any given language are shaped by the sensory and motor apparatus involved in the expression of that language, and that many questions remain regarding the role of the auditory–vocal channel in the linguistic structures, abilities, and impairments investigated after brain damage. However, by studying the processing of a language based on visual signs following brain damage, we hoped to obtain an additional perspective on the neural basis of language. We focused on American Sign Language (ASL), an autonomous language with organizational principles similar to those of English and other auditory–vocal languages, but with a mode of transmission and with linguistic mechanisms that have evolved within the framework of a visual–gestural symbolic system.

Two of the primary levels of linguistic organization in ASL are the combining of sublexical elements into meaningful units, and spatially organized syntax. The former refers to the composition of individual, meaningful signs from combination of a limited set of recurring sublexical components. The latter refers to the specification of semantic and syntactic relations among signs by means of manipulating signs in space. The present study focuses primarily on the first level of ASL organization.

The combining of sublexical units is analogous to the combination of phonemes into meaningful units in spoken languages. In ASL, individual signs which serve as referents for specific objects, actions, relationships, and properties, comprise combinations of handshapes, hand movements, and locations relative to the body.

It is known that deaf signers become aphasic in ASL, as in auditory based languages, following damage to selective areas of the left hemisphere, but not following right hemisphere damage, and that the patterns of ASL impairment in left hemisphere damaged signers are similar to those of aphasia in auditory–vocal languages [1]. Furthermore, it was found that a left language-dominant hearing signer became severely aphasic in both English and ASL during a Wada test with Amytal injection in the left carotid [8].

To explore further the relationships among sign language, auditory–vocal languages, and language-related left hemisphere cortices, we have begun to study the ability of hearing individuals to produce and comprehend visual–gestural signs from ASL following damage to left hemisphere cortices resulting in aphasia for English. We hypothesized that these subjects would be able to acquire aspects of an ASL lexicon, and that the ability to use visual–gestural signs to communicate would show at least partial dissociation from the impaired ability to communicate such references in English, depending on the specific locus of the aphasia producing lesion. We present here the findings on our first three subjects.

SUBJECTS

The subjects were three severely aphasic patients referred to the Division of Behavioral Neurology and Cognitive Neuroscience, Department of Neurology, The University of Iowa Hospitals and Clinics. All were fully right-handed (+100 on the Geschwind–Oldfield questionnaire [16]), and their aphasias corresponded most closely to the traditional diagnostic category of Wernicke's aphasia, according to the Boston aphasia classification system [13]. None was familiar with sign language prior to the onset of aphasia.

The first subject (referred to as NE1188) was a 28-year-old male graduate student. His primary language had been English for most of his life. NE1188 was in good health until the abrupt onset of aphasia and mild right hemiparesis caused by stroke. The hemiparesis resolved within the first days after onset, but the aphasia persisted. The studies reported here were conducted between 5 and 8 weeks post-onset.

The second subject (referred to as RZ1365) was a 56-year-old man whose native language was English. He holds a doctoral degree and was formerly an active professional. Seven years prior to our observations, he had a left middle cerebral artery infarction which resulted in a severe and chronic aphasia. He began participating in weekly sign language instruction approx. 1 year prior to our contact with him.

The third subject (referred to as BH1490) was a 28-year-old woman whose native language was English. She was a factory worker with a high school education, and was in good health until the onset of herpes simplex encephalitis at age 26. Her neurologic recovery was complete with the exception of the cognitive impairments described below. The studies reported here were conducted 2 years post-onset.

NEUROANATOMICAL STUDIES

Neuroanatomical analysis of magnetic resonance images (MRI) was performed according to our standard template charting technique [10]. The extent of cortical involvement is plotted in normal diagrams of cytoarchitectural fields in Fig. 1, for comparison across the three subjects.

NE1188: MRI obtained 1 year post-stroke showed an area of infarction in the territory of the left middle cerebral artery. The damage involves part of the temporal and parietal cortices, the insular cortex, and the basal ganglia. In the temporal region, the posterior half of the superior gyrus and the posterior third of the middle gyrus, are involved. In the parietal region, the posterior segment of the supramarginal gyrus and the anterior segment of the angular gyrus are damaged. The insula is virtually destroyed, and there is partial damage to the head of the caudate nucleus and the outermost portion of the putamen.

RZ1365: MRI obtained 7 years post-stroke showed an area of infarction in the territory of the left middle cerebral artery. The damage involved the frontal, parietal, and temporal cortices, as well as the insula. The temporal damage involves the posterior half of the superior gyrus and the posterior third of the middle gyrus. In the parietal cortex, the damage involved most of the supramarginal and angular gyri. There was also subcortical damage at the level of the postcentral gyrus. There is some damage to the most posterior segment of the frontal operculum. The insula is destroyed. No damage can be seen in the caudate, but the lateral ventricle is enlarged at the level of the frontal horn.

BH1490: MRI obtained 2 years after herpes simplex encephalitis showed a lesion involving most of the left

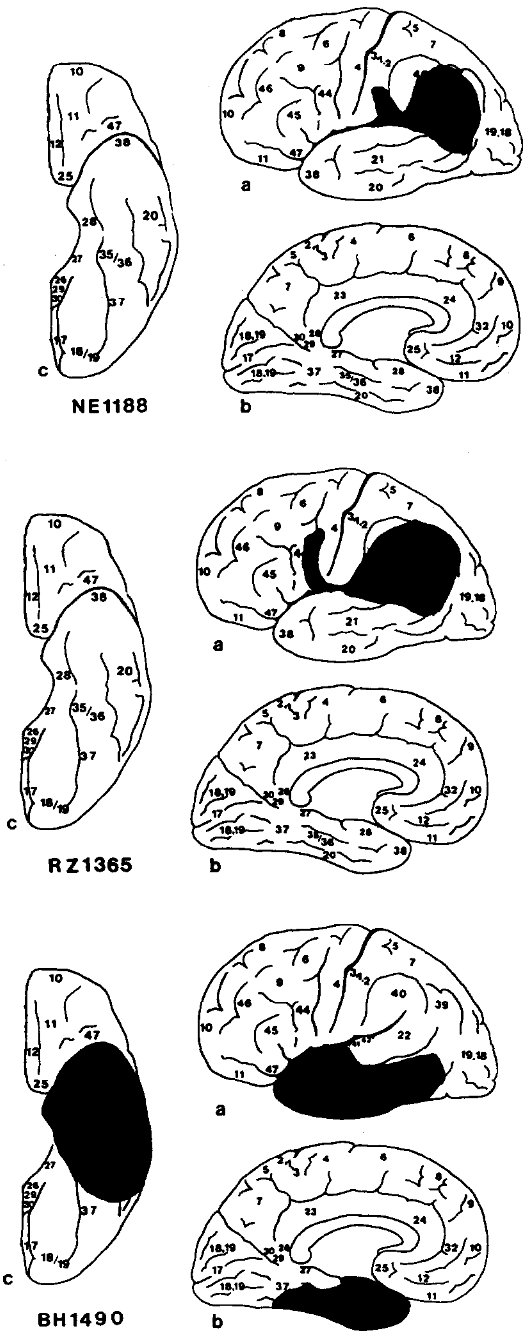

Fig. 1. Diagrams showing the cortical extent of the lesions in the three subjects. Panels a, b and c are views of the left hemisphere, respectively, lateral, medial, and inferior. Note that the lesion pattern in BH1490 is virtually complementary to that of the two other subjects. Most of her left temporal cortices are destroyed except for the posterior half of the superior temporal gyrus. In patients NE1188 and RZ1365 the lesions only involve part of the posterior half of the superior temporal gyrus and spare all other temporal cortices.

temporal lobe. Only the posterior segment of the superior temporal gyrus, including the transverse gyrus of Heschl, was spared. The insula was also damaged. There was no evidence of damage to the right hemisphere.

The pattern of damage in BH1490 is remarkably different from that in the other two patients. Unlike NE1188 and RZ1365, BH1490 has (a) damage to all the anterior temporal cortices, and (b) sparing of the auditory (primary and association) cortices in the left superior and posterior temporal gyrus. This pattern virtually complements and hardly overlaps the pattern of the other two which are, in turn, remarkably similar.

NEUROPSYCHOLOGICAL STUDIES

Linguistic and other cognitive abilities were evaluated with a battery of standardized neuropsychological tests probing aspects of intellect, memory, visual perception, visual construction, and language. The battery included selected subtests of the Wechsler Adult Intelligence Scale—Revised [19], Revised Benton Visual Retention Test [2], Facial Recognition Test [4], Rey–Osterrieth Complex Figure Test [15], Multilingual Aphasia Examination [3], and subtests of the Boston Diagnostic Aphasia Examination (BDAE) [13]. Scores obtained on the standardized tests are presented in Table 1.

Table 1. Neuropsychological test scores

	NE1188	RZ1365	BH1490
Multilingual Aphasia Examination			
COWA	1*	1*	1*
Visual naming	1*	1*	1*
Sentence repetition	1*	1*	1*
Token test	2*	1*	1*
Aural comprehension	2*	2*	2*
Reading comprehension	17	59	1*
Oral spelling	1*	1*	1*
Boston Diagnostic Aphasia Examination			
Responsive naming	20*	73*	42*
Repeating phrases	20*	20*	1*
Automatized sequences	10*	40*	1*
Reading sent. and paragraphs	60*	95	1*
WAIS—R			
Information	25	13	—
Block design	16	84	25
Picture arrangement	84	13	16
BVRT (Form C, Administration A)			
Number correct	8	6	4*
Number of errors	2	4	9*
Facial Recognition Test	90	99	15
Rey–Osterrieth Complex Figure Test			
Copy	99	99	60
30 min recall	30	10	15

Note: All scores are presented as percentile scores, except for the BVRT, for which raw scores are presented.

*Scores which are in the Defective range according to published norms.

NE1188's nonlinguistic cognitive abilities were well-preserved, as evidenced by normal performances on all administered tests of nonverbal intellect, memory, visual perception, and visual construction. There was no evidence of impairment with either hand in performing transitive or intransitive gestures to copy or instruction.

His speech was fluent and well-articulated, with a paucity of content words and frequent paraphasic errors, including phonemic, semantic, and neologistic errors. There was a near complete inability to produce nouns, verbs, adjectives, or adverbs. Production of pronouns and functors was relatively preserved, although they were often used incorrectly. There were no evident impairments of prosody, volume, speech initiation, or length of utterance. He was unable to repeat single words or isolated speech sounds. Aural comprehension was severely impaired at the level of single words and short phrases. Reading comprehension was somewhat better, but still defective. Writing was agraphic, with well-formed letters, but frequent letter and word substitutions. Similar to his speech, writing was characterized by a paucity of content words. There were slight improvements in NE1188's linguistic abilities during the course of this study. However, the pattern of defects remained unchanged, and the impairments described above remained severe.

RZ1365's nonlinguistic cognitive abilities were also well-preserved. Transitive and intransitive gestures were performed normally with both hands. Speech was fluent and well articulated, but highly paraphasic (primarily phonemic and semantic errors). Speech prosody, volume, initiation, and length of utterance were normal. Naming, repetition, writing, and aural comprehension were all severely impaired. Reading comprehension was relatively better than aural comprehension, but still defective.

BH1490's performances on tests of nonverbal intellect and visual perception were within normal limits. Memory for geometric designs was mildly impaired. Although copy of a complex figure was within normal expectations, three-dimensional block construction was mildly impaired. These mild cognitive impairments do not interfere with her ability to drive, orient herself, learn new routes, or learn to recognize new faces and objects. Transitive and intransitive gestures were performed normally with both hands.

Her speech was fluent and well-articulated, but was highly paraphasic (more so than either NE1188 or RZ1365). Semantic substitutions were the most frequent error type, but there were also phonemic and neologistic errors. Speech prosody, volume, initiation, and length of utterance were not impaired. Overlearned phrases were used frequently and inappropriately. BH1490 was rarely able to produce words on command. She was unable to repeat single words or isolated speech sounds. Aural and reading comprehension were severely impaired at the single word level. She was virtually unable to write any word, but could often produce the first few letters.

EXPERIMENT USING SIGNS FROM ASL

The three aphasic subjects were next evaluated for the ability to learn visual–gestural signs to communicate references to specific items and events. The ASL training portion of the experiment applied only to subjects NE1188 and BH1490, because RZ1365 had participated in ASL instruction for 1 year prior to our contact with him. These two aphasic subjects were matched with two normal control subjects. The control subjects (NC1 and NC2) were individually matched to NE1188 and BH1490 for age, education, and gender, and like the patients had no previous familiarity with sign language. NC1 was a 26-year-old, right-handed male graduate student. NC2 was a 30-year-old, right-handed woman who had a high-school education and was employed as a housekeeper. The aphasic and control subjects were individually provided with systematic instruction in aspects of ASL and finger spelling in 10 sessions conducted over a period of approx. 14 days. In addition to the 26 letters of the alphabet and the numbers 1–20, a pool of over 100 signs depicting common nouns, pronouns, verbs, adjectives, adverbs and prepositions was selected for instruction, with an emphasis on words with practical utility in normal daily activities.

Training sessions

The 10 training sessions were 30–60 min in length, and were conducted by a deaf sign language instructor and two hearing technicians fluent in ASL. In conjunction with the appropriate sign, individual words, letters, and numbers were presented in several modalities. These included words printed on cards and spoken, as well as the use of objects, pictures, and gestures. Following presentation of a sign, the subject would then attempt to form the sign, and was corrected if necessary. This procedure was repeated until an individual sign was reliably produced at the criterion level of three successive correct ASL responses to printed, pictorial, or aural stimuli. After a sign had been learned at this level, it was entered into a group for later rehearsal, and the next sign was presented. Signs were rehearsed in groups of 2–20, in formats which required both production and comprehension.

Test sessions

Test sessions were conducted following training sessions (except for subject RZ1365, who participated only in the test portion). Each subject was tested individually. NE1188 and BH1490 were tested on a set of 100 signs which had been learned at the criterion level and subsequently rehearsed. These signs were from the lexical categories of nouns ($n=41$), verbs ($n=22$), modifiers (adjectives and adverbs, $n=18$), and functors ($n=19$) (listed in the Appendix). RZ1365 was tested on a different set of signs, comprising the same lexical categories, selected to probe his more complex sign vocabulary.

To assess ASL production, each subject was presented with individual English words, letters, and numbers printed on cards, and required to make the appropriate sign. If a subject was unable to comprehend a printed word, the word was also spoken or conveyed through pictures. To assess ASL comprehension, individual ASL signs were presented in random order, and the subject was required to write all or part of the associated English word, or to sketch a pictorial representation.

RESULTS

Production

Individual signs. In contrast to their near complete inability to produce content words in English, all aphasic subjects were able to acquire a relatively large repertoire of signs (Table 2). All subjects except BH1490 were able to correctly produce the appropriate ASL sign for more than 90% of the words presented, and even BH1490 was correct on 81 of 100 signs. Note, however, that her matched control (NC2) was correct on all 100 signs.

For all subjects, most errors consisted of either complete inability to recall a sign, or sublexical errors (one component of the hand shape or movement was incorrect). There were few semantic (whole sign) substitutions. Because BH1490 was the only subject who did not approximate 100% correct in the production of individual signs, we further analyzed her performances across grammatical categories (Table 3). Her performance was good for nouns and functors, but less so for verbs and modifiers.

All subjects were able to produce individual signs representing the 26 letters of the alphabet (presented in random order) at or near 100% correct. This was also true for the numbers 1–20 presented in random order, except for subject BH1490, who was only able to learn the numbers 1–10 during the course of the training sessions.

Multiple sign sequences. We also assessed the ability to combine multiple signs in a meaningful sequence. First, in a repetition task, short ASL sequences were presented and the subjects were asked to repeat each sequence in ASL. The longest sequences consisted of four signs, for example, "I need practice sign", and "If rain, baseball cancel". All subjects except BH1490 performed well on this task. BH1490 had extreme difficulty combining signs. She was able to repeat only four of 24 sequences, and all of those were under four signs in length.

Ten to thirteen action sequences of the form "subject–verb–object" were acted out with miniature figures, and the subjects were required to produce the appropriate ASL sequence. An example of such a sequence is "Man kick horse". None of the subjects performed as well as the controls on this task, but NE1188 and RZ1365 were able to produce the correct series of ASL signs for at least 70% of the action sequences. In contrast, BH1490 was able to produce the correct sign sequence for only 2 of 10 action sequences. Errors included subject–object reversals, as well as verb and noun substitutions.

Table 2. Sign production

	Percent correct	Number correct	Number attempted
Individual signs			
NE1188	98	98	100
RZ1365	94	127	135
BH1490	81	81	100
NC1	97	97	100
NC2	100	100	100
Letters			
NE1188	96	25	26
RZ1365	100	26	26
BH1490	100	26	26
NC1	100	26	26
NC2	100	26	26
Numbers			
NE1188	100	20	20
RZ1365	90	18	20
BH1490	100	10	10
NC1	95	19	20
NC2	100	10	10
Repetition			
NE1188	85	17	20
RZ1365	100	12	12
BH1490	17	4	24
NC1	90	18	20
NC2	100	24	24
Sequences			
NE1188	77	10	13
RZ1365	70	7	10
BH1490	20	2	10
NC1	92	12	13
NC2	100	10	10

Table 3. Sign production and comprehension across grammatical categories: subject BH1490

		Percent correct	
Lexical category	*N*	Production	Comprehension
Nouns	41	88	90
Verbs	22	68	68
Modifiers	18	72	67
Functors	19	89	89

The impairment of sign sequencing shown by BH1490 did not appear to be secondary to a general sequencing defect. For example, she obtained a block pointing span of six blocks forward and six blocks backward on the Wechsler Memory Scale—Revised, and performed at the mean level for normal subjects on the Memory for Objects subtest of the Stanford–Binet Intelligence Scale: Fourth Edition.

Comprehension

Individual signs. To assess comprehension of ASL, subjects were presented with individual signs and asked to write the associated word or draw a pictorial representation. Words which were misspelled but easily recognizable were considered to be correct. All subjects except BH1490 performed this task at or near 100% correct (Table 4). BH1490 was correct on 81 of 100 signs. A breakdown of BH1490's sign comprehension performances across grammatical categories (Table 3) showed that she was correct on nearly 90% of nouns and functors, but less than 70% correct on verbs and modifiers.

Table 4. Sign comprehension

	Percent correct	Number correct	Number attempted
Individual signs			
NE1188	100	100	100
RZ1365	97	97	100
BH1490	81	81	100
NC1	100	100	100
NC2	100	100	100
Letters			
NE1188	100	26	26
RZ1365	100	26	26
BH1490	88	23	26
NC1	100	26	26
NC2	100	26	26
Numbers			
NE1188	80	16	20
RZ1365	100	20	20
BH1490	100	10	10
NC1	100	20	20
NC2	100	10	10
Sequences			
NE1188	90	9	10
RZ1365	100	10	10
BH1490	20	2	10
NC1	100	10	10
NC2	100	10	10

As with the production of signs associated with individual letters, all subjects were able to learn to comprehend signs representing the letters of the alphabet. Only BH1490 made any errors on this task (23 of 26 correct). There was somewhat more variability in the ability to comprehend signs representing numbers. Subjects were presented with individual signs and required to hold up the appropriate number of fingers. NE1188 was able to learn the numbers 1–20, and was correct on 80% of those in the test session. RZ1365 was correct on 20 of 20 numbers. BH1490 was able to learn only the numbers 1–10 during the training sessions, but she was 100% correct on those numbers when presented in a random order in a test session.

Multiple sign sequences. Ten ASL sequences depicting "subject–verb–object" action sequences were presented, and subjects were required to select the appropriate miniature figures from an array of 25 figures and then act out the sequence. NE1188 was correct on 9 of 10 sequences. The single error resulted from a subject–object reversal. RZ1365 and the control subjects were correct on 100% of the sequences. BH1490 was correct on only two sequences. Her errors included subject–object reversals, verb substitution, and object substitution.

Use of sign in communication. Both NE1188 and RZ1365 used ASL in conjunction with English to communicate in nontest situations in our laboratory and with other people outside the laboratory. Patient BH1490 differed from the other two aphasic patients in that she rarely used signs to help her communication. Although she practiced signs at home and occasionally used signs to refer to objects around the house, she rarely used signs to communicate, even in situations where she was unable to express herself in English, her audience knew ASL, and she had appropriate signs in her repertoire.

DISCUSSION

These results show that hearing individuals with severe aphasia for English caused by damage to language cortices are able to learn and execute numerous signs from ASL in spite of their inability to produce the equivalent items in English. References to objects, actions, properties and relations may be expressed by such patients through the newly acquired visual–gestural signs. Furthermore, patients with lesions in left posterior temporal and parietal regions can combine the newly learned signs in meaningful sequences and use signs as a means to assist their impaired communication. Only the patient with damage to virtually all left temporal cortices was limited to the use of one sign at a time in both production and comprehension, and was unable to use signs to help her communication. The ease with which the patients acquired and used the visuomotor signs suggests that they were connecting them with preexisting semantic knowledge (conceptual, nonverbal memoranda regarding objects, properties, etc.). This not only implies that semantic knowledge is represented independently of the auditory–vocal records that embody the corresponding lexical entries, but also suggests that the two patients who used signs for communication were relying on the preexisting frame that bound conceptual representations and linguistic representations.

The differences in the performances of subject BH1490 relative to those of NE1188 or RZ1365 provide intriguing clues regarding the location of networks necessary for acquiring visual–gestural signs and using them in communication. Subject BH1490 was not only less successful than the other two in single sign acquisition, but clearly failed to use the extensive set of signs she learned. Turning to the anatomical specifications of the cases, it seems that damage to either left posterotemporal cortices or inferior parietal cortices is compatible with: (a) the learning of single signs across varied lexical classes, (b) the use of those signs in brief but meaningful sequences, and (c) the use of signs to help communication. On the other hand, destruction of the entire left temporal cortices (seen only in BH1490) precludes the production or comprehension of meaningful sequences, and precludes the use of the newly learned signs as a means of communication.

The findings may be interpreted in the context of previous evidence that experienced signers, deaf or not, develop ASL aphasia in association with selective left hemisphere dysfunction [1, 8]. The previous evidence (see Ref. [17] for review) combined with current knowledge from experimental neuroanatomy, neurophysiology and neuropsychology (see Refs [5]–[7] and [9] for reviews) has led to some working hypotheses regarding the neural substrate of sign acquisition and processing. In brief, the key hypotheses are as follows: (1) The visuomotor patterns contained in ASL signs can be acquired on the basis of networks which include (a) visual association cortices concerned with shape and motion processing (located in posterior temporal/occipital regions), and (b) somatosensory and motor association cortices concerned with hand movement (in parieto-frontal regions). We presume the left side of this bilateral network is dominant. (2) The bond between isolated signs and the conceptual knowledge that they refer to is rooted in higher order association cortices of both temporal and frontal regions. Again we presume the network in the left side is dominant. In individuals such as our patients, who were proficient in English and who learned signs as part of a second language, we propose that the binding depends on the neural structures that were previously responsible for the bond between concepts and English lexical entries. Elsewhere we have proposed the concept of "lexical access convergence zone", a third party mediating device that performs this binding function and that is based on left anterior temporal cortices, as far as English nouns are concerned. Naturally, in deaf signers acquiring ASL as a first language, these devices would be dedicated to ASL mediation alone and might be placed in a slightly different anatomical position. (3) The sign sequencing process depends on the action of left frontal cortices over the structures designated under (1) and (2).

The sector of the networks required for acquisition of the shapes and motion in signs was spared in all three patients. The sector of the networks required for binding signs to conceptual knowledge was spared in NE1188 and RZ1365 (in both hemispheres), but significantly damaged in BH1490 on the left. While patients NE1188 and RZ1365 were able to connect their new visuomotor signs to the preexisting system that bound concepts and English words, patient BH1490 no longer had available such a core frame for lexical access. She had to connect signs to concepts, on a one by one basis, rather like a paired associate learning task, and not as part of language operation. The failure to create sequences of signs stems from this defect. The networks required for sign sequencing were intact in NE1188 and RZ1365, but significantly damaged in BH1490 because the action of left frontal cortices on left anterotemporal cortices was no longer possible.

In the final analysis, the situation of the three patients may be characterized as follows. In patients NE1188 and RZ1365, sign provided them with input and output channels for a language system whose core lexical access frames must have been relatively intact. In BH1490, the preexisting language system was largely damaged and signs only made contact with knowledge *outside* of that preexisting lexical access system. These contacts appear to have been of little practical use to this subject.

Further evidence is needed to validate this preliminary account. It will be important for future research to probe the acquisition of signs by patients with single and multiple lesions in various aspects of the left and right hemispheres, including lateral occipitotemporal cortices, hand-related sectors of the frontal and parietal areas, anterotemporal cortices, and premotor/prefrontal cortices. Electrophysiological and PET studies will also be required to complement lesion studies in the evaluation of processing lateralization. Future findings are likely to complement additional evidence on the neural basis of sign processing coming from studies of sign aphasia in the deaf, and will help decide whether or not the networks used by deaf and hearing signers are largely the same or different.

The findings of the present study are of practical interest with regard to neuropsychological rehabilitation. Previous research has indicated that some aphasic patients are able to learn aspects of nonverbal communication systems. For instance, SKELLY *et al.* [18] found that visual–gestural signs from American Indian Sign facilitated verbalization in verbal apraxic patients. HELM-ESTABROOKS *et al.* [14] demonstrated that global aphasics were able to improve their pantomime and aural comprehension ability through a training system in which gestures were used to represent objects. GLASS *et al.* [12] found that seven global aphasics were able to learn some aspects of an artificial language based on a modification of a system originally designed for chimpanzees (symbols cut out from colored paper were used to express various relationships, such as same–different, and subject–verb–object sequences).

GARDNER *et al.* [11] attempted to teach eight severely aphasic patients a visual communication system which consisted of geometric or ideographic symbols drawn on notecards, with each symbol representing a meaningful unit (word or phrase). Five patients (three global, one Broca/mixed and one Wernicke/transcortical aphasia) were able to acquire various degrees of competence with the system, including at least one patient who used it to communicate with family members. Three other global aphasics were unable to master the system after 3 months of training. Recently, a computerized version of this visual communication system has been taught to a globally aphasic patient [20]. This patient was able to comprehend reversible prepositional phrases expressed in the visual communication system, in contrast to his inability to comprehend these phrases in printed English. It is interesting to note that this patient, like our patient BH1490, had greater difficulty learning verbs than nouns in the alternative communication system.

In conclusion, all of these studies offer converging evidence that hearing aphasics have a greater or lesser ability to learn a new system of visually based signs. Clearly, dysfunction of the primary mode of communication (the auditory–vocal system) does not predict equal dysfunction in alternate modes (e.g. the visual–gestural system). In general, the prior studies differ from ours in that the patterns of patient performance have not been related to neuroanatomical data, and the role of neural factors in the profile have not been considered.

Another difference between the studies reviewed above and the present study is that our aphasic patients were able to learn aspects of a biologically valid, standard, and widely-used non-vocal language. To be sure, none of the three subjects had been taught the subtleties of ASL syntax. However, two of the three subjects found ASL signs to be useful in certain situations in their daily activities outside of the laboratory, and all reported that the acquisition of signs significantly reduced their feelings of disability following stroke.

As a well-established language, ASL has advantages over other nonvocal communication systems in the rehabilitation of aphasic patients who have been unable to regain functional auditory–vocal languages. There are communities of people in all parts of North America who know and use ASL (comparable, but separate sign languages are used in Europe and other parts of the world). Courses teaching sign language are readily available for family members, and sign language interpreters are frequently used in religious services, television programs, and other public events. For some aphasic patients, the ability to produce and comprehend at least some elements of sign language may facilitate their adjustment following brain damage.

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REFERENCES

1. BELLUGI, U., POIZNER, H. and KLIMA, E. S. Language, modality, and the brain. *Trends Neurosci.* **12,** 380–388, 1989.
2. BENTON, A. L. *The Revised Visual Retention Test: Clinical and Experimental Applications.* The Psychological Corporation, New York, 1974.
3. BENTON, A. L. and HAMSHER, K. *Multilingual Aphasia Examination Manual.* AJA Associates, Inc., Iowa City, 1978.
4. BENTON, A. L., HAMSHER, K., VARNEY, N. R. and SPREEN, O. *Contributions to Neuropsychological Assessment.* Oxford University Press, New York, 1983.
5. DAMASIO, A. R. Time-locked multiregional retroactivation: A systems level proposal for the neural substrates of recall and recognition. *Cognition* **33,** 25–62, 1989.

6. DAMASIO, A. R. The brain binds entities and events by multiregional activation from convergence zones. *Neural Computation* **1,** 123–132, 1989.
7. DAMASIO, A. R. Concepts in the brain. *Mind Lang.* **4,** 24–28, 1989.
8. DAMASIO, A. R., BELLUGI, U., DAMASIO, H., POIZNER, H. and VAN GILDER, J. Sign language during left-hemisphere Amytal injection. *Nature* **322,** 363–365, 1986.
9. DAMASIO, A. R., DAMASIO, H., TRANEL, D. and BRANDT, J. The neural regionalization of knowledge access: Preliminary evidence. *The Brain Symposium Volume.* Cold Spring Harbor Laboratory, 1990.
10. DAMASIO, H. and DAMASIO, A. R. *Lesion Analysis in Neuropsychology.* Oxford University Press, New York, 1989.
11. GARDNER, H., ZURIF, E. B., BERRY, T. and BAKER, E. Visual communication in aphasia. *Neuropsychologia* **14,** 275–292, 1976.
12. GLASS, V. A., GAZZANIGA, M. S. and PREMACK, D. Artificial language training in global aphasics. *Neuropsychologia* **11,** 95–103, 1973.
13. GOODGLASS, H. and KAPLAN, E. *The Assessment of Aphasia and Related Disorders,* 2nd edn. Lea & Febiger, Philadelphia, 1983.
14. HELM-ESTABROOK, N., FITZPATRICK, P. M. and BARRESI, B. Visual action therapy for global aphasia. *J. Speech Hearing Disord.* **47,** 385–389, 1982.
15. LEZAK, M. D. *Neuropsychological Assessment,* 2nd edn. Oxford University Press, New York, 1983.
16. OLDFIELD, R. C. The assessment and analysis of handedness: The Edinburgh inventory. *Neuropsychologia* **9,** 97–113, 1971.
17. POIZNER, H., KLIMA, E. S. and BELLUGI, U. *What the Hands Reveal About the Brain.* MIT Press/Bradford Books, Cambridge, MA, 1987.
18. SKELLY, M., SCHINSKY, L., SMITH, R. W. and FUST, R. S. American Indian Sign (AMERIND) as a facilitator of verbalization for the oral apraxic. *J. Speech Hearing Disord.* **39,** 445–456, 1974.
19. WECHSLER, D. *Wechsler Adult Intelligence Scale—Revised.* The Psychological Corporation, New York, 1981.
20. WEINRICH, M., STEELE, R. D., CARLSON, G. S., KLECZEWSKA, M., WERTZ, R. T. and BAKER, E. Processing of visual syntax in a globally aphasic patient. *Brain Lang.* **36,** 391–405, 1989.

APPENDIX

Nouns

glasses	car	hospital	tomato	boy	rain
tomorrow	sign	dress	father	man	table
home	girl	squirrel	horse	monkey	onion
woman	dog	cat	water	word	hour
house	minute	tree	Sunday	Monday	Tuesday
Wednesday	Thursday	Friday	Saturday	moon	yesterday
fork	sentence	bird	key	friend	

Verbs

sleep	drink	draw	love	break	want
practice	like	need	eat	kick	hit
push	teach	close	remember	forget	watch
open	sit	write	know		

Modifiers

tired	happy	sad	yellow	orange	blue
purple	white	green	red	same	different
old	right	left	small	angry	black

Functors

and	or	if	on	off	under	above		around		after
in	you	your	I, me	who	them, they	us	his	hers	my	