

# A Longitudinal Examination of Crossed Aphasia

Gustavo J. Rey, PhD; Bonnie E. Levin, PhD; Raul Rodas, MD; Brian C. Bowen, MD, PhD; Kester Nedd, DO

**Objective:** To longitudinally examine neuropsychological performance in an adult dextral man with crossed aphasia after cerebrovascular accident.

**Design:** Case report using longitudinal neuropsychological, neurological, and radiological examinations performed in close temporal proximity to one another.

**Setting:** The patient was seen on both an inpatient and an outpatient basis by members of the Departments of Neurology and Radiology of the University of Miami (Fla) School of Medicine.

**Patient:** Thirty-four-year-old right-handed monolingual Hispanic man without family history of left-handedness.

**Results:** Initial neuropsychological testing revealed clas-

sic Broca's dysphasia, visual neglect, and visuospatial disturbances. The visuospatial disturbance resolved within 6 months whereas expressive language remained severely impaired. There was a dissociation between praxis and language. Mood was jovial with indifference toward his neurologic and cognitive limitations. Serial magnetic resonance imaging studies unequivocally localized the lesions to the right hemisphere, involving the right frontal, anterior parietal, and subcortical white matter.

**Conclusions:** The clinicopathologic correlation is compatible with the view that crossed aphasia is a "mirror" representation of that seen in cases of uncrossed aphasia. The course of recovery suggests complete lateralization of language to the right hemisphere with bilateral or crossed representation of nonverbal skills.

(Arch Neurol. 1994;51:95-100)

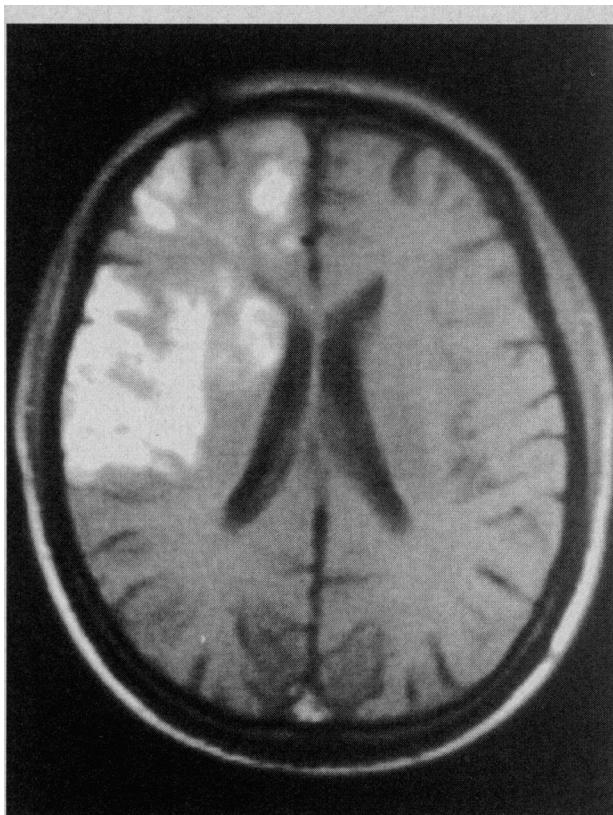
From the Departments of Neurology (Drs Rey, Levin, Nedd, and Rodas), Psychology (Dr Levin), Radiology (Dr Bowen), and Rehabilitation (Dr Nedd), University of Miami (Fla).

**C**ROSSED APHASIA refers to disturbances of language following a right-hemisphere lesion in right-handed individuals. Although it is estimated to occur in 1% to 2% of all dextral aphasics, well-documented cases of crossed aphasia continue to be rare.<sup>1,2</sup> Published studies frequently lack longitudinal evaluation of the aphasic disturbance and/or associated neuropsychological findings, and do not include correlative neurodiagnostic procedures. These studies are frequently confounded by cases with familial history of sinistrality or the inclusion of patients with chronically debilitating neurologic diseases or neurologic conditions that result in diffuse brain abnormalities. This article presents detailed neuropsychological testing and magnetic resonance imaging data in a young adult dextral man with

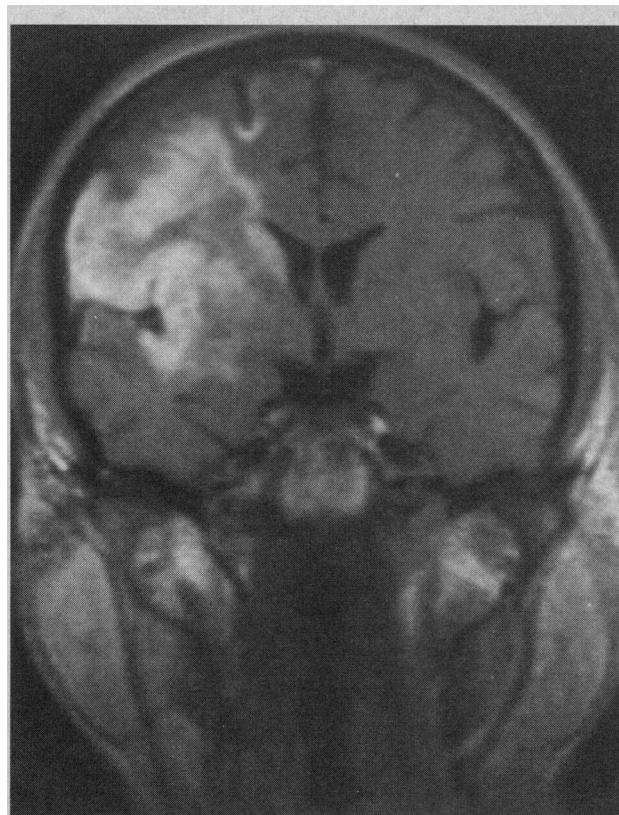
crossed aphasia at 1 week and at 1, 6, and 18 months after suffering a stroke.

## REPORT OF A CASE

The patient was a 34-year-old fully right-handed (Edinburgh handedness inventory, 10),<sup>3</sup> monolingual (Spanish) white man with a ninth-grade education. His medical history was significant for a 28-year history of diabetes requiring split dosing of mixed insulin preparation, a 2-year history of well-controlled hypertension, cigarette smoking, and a 4-year history of headaches (HAs). There was no history of perinatal complications or learning disability. Psychiatric history was significant for depression from 12 to 15 years of age, secondary to difficulty accepting and adjusting to the diagnosis of diabetes. Family history was noncontributory. There were no known left-handed relatives.



**Figure 1.** Axial  $T_2$ -weighted image on initial magnetic resonance study.



**Figure 2.** Postgadolinium  $T_1$ -weighted coronal image 1 week after cerebrovascular accident.

## CLINICAL PRESENTATION AND INITIAL FINDINGS

History of the present illness dates to August 30, 1989, when he was awakened from sleep by a severe right frontotemporal HA, associated with mild left upper extremity weakness and expressive language difficulty with preserved comprehension. He was seen in the emergency department the same day and was found to be alert and oriented. General and physical examinations revealed dystrophic skin changes of the lower extremities and familial proptosis. The initial neurologic examination demonstrated left facial weakness, left upper extremity hemiparesis, and expressive aphasia. Sensory examination was normal, pupils were equal and reactive, and intact visual acuity, ocular motility, and color discrimination were documented. There was no evidence of visual neglect on bedside neurologic examination. A thorough cardiovascular evaluation failed to reveal any abnormalities. A grade 2 diabetic retinopathy was evident and, throughout the hospitalization, control of his diabetes was difficult and managed by the endocrinology service. An electroencephalogram showed abnormal slowing over the right parietal area without epileptiform activity. Neuroradiologic results are presented in **Figure 1** and **Figure 2**. Initial magnetic resonance imaging showed areas of prolonged  $T_1$ - and  $T_2$ -relaxation time in the right frontal and anterior parietal lobes involving cortex and subcortical white matter, as well as the right caudate and anterior lenticular nucleus, associated with mass

effect. Postgadolinium images showed minimal peripheral enhancement in the involved areas. A repeated magnetic resonance imaging 5 days later again showed similar signal abnormalities on long TR images but with decreased mass effect. Postgadolinium images demonstrated prominent gyral enhancement. These findings were consistent with evolving infarcts in the right middle cerebral and anterior cerebral arterial distribution. No signal abnormalities were identified in the left hemisphere or posterior fossa. The neuropsychologic test results are summarized in **Table 1** and **Table 2**. Initial neuropsychologic examination was performed 1 week after admission and the following results were obtained.

## LANGUAGE EXAMINATION

Language was assessed with the Multilingual Aphasia Examination-Spanish (MAE-S),<sup>4</sup> the Test de Vocabulario en Imágenes Peabody,<sup>5</sup> and translated portions of the Boston Diagnostic Aphasia Examination.<sup>6</sup> Oral expressive language skills were severely compromised, with no spontaneous intelligible verbalizations. He was unable to perform simple orolingual manipulations, recite automatized series, or repeat simple words. Written expressive skills were also severely compromised, as he was unable to write simple words to copy or to dictation (**Figure 3**, left). He managed to write letters and numbers to dictation with approximately 50% accuracy. Simple written arithmetic calculations were markedly defective.

**Table 1. Language-Related Test Results at 1 Week, and at 1, 6, and 18 Months After Cerebrovascular Accident\***

	Time After Cerebrovascular Accident			
	1 wk	1 mo	6 mo	18 mo
<b>Receptive Language</b>				
Receptive vocabulary (TVIP SS)	106	...	118	117
Auditory comprehension of complex ideational information (BDAE raw score)	8/8	...	8/8	8/8
Aural comprehension (MAE-S percentile)	50	...	90	90
Written comprehension (MAE-S percentile)	<1†	...	30	90
Token test (MAE-S percentile)	<1†	1†	2†	2†
<b>Expressive Language</b>				
Nonverbal oral agility (BDAE raw score)	3/12†	...	6/12‡	6/12‡
Visual naming (MAE-S percentile)	<1†	...	10‡	5‡
Sentence repetition (MAE-S percentile)	<1†	<1†	2†	2†
Controlled word association (MAE-S percentile)	<1†	...	<1†	<1†
Writing words to dictation (raw score)	0/9†	2/9†	2/9†	2/8†
Writing letters to dictation (raw score)	6/14†	14/14	14/14	13/13
Writing sentences to copy (raw score)	0/3†	2/3‡	3/3	3/3

\*TVIP indicates Test de Vocabulario en Imágenes Peabody; BDAE, Boston Diagnostic Aphasia Examination; and MAE-S, Multilingual Aphasia Examination-Spanish.

†Moderately to severely impaired performance.

‡Mildly impaired performance.

Receptive skills were relatively preserved. Identification of body parts, ability to follow 1-, 2-, and 3-step commands, and comprehension of complex ideational information were performed with 85%, 67%, and 100% accuracy, respectively. Receptive vocabulary on the Test de Vocabulario en Imágenes Peabody was average. The MAE-S Aural Comprehension of Words Phrases was also well within normal limits, but aural comprehension on the MAE-S Token Test was severely impaired. Written comprehension of simple words and phrases in the MAE-S was defective.

## ASSOCIATED COGNITIVE SKILLS

Right-left orientation<sup>7</sup> to self was normal but impaired in extrapersonal space. Mild left visual field neglect was evident on a line bisection test and a simplified letter cancellation task. He was unable to spontaneously draw a clock. The copy condition of the Rey-Osterreith Complex Figure<sup>8</sup> and clock drawing to copy revealed distinct left visual field inattention (Figure 4, top). Reproduction of the Rey figure was impaired and segmented, with omissions and distortions of left-sided details. Angular judgments<sup>9</sup> and discrimination of simple geometric designs<sup>10</sup> were impaired.

**Table 2. Associated Cognitive Skills at 1 Week, and at 1, 6, and 18 Months After Cerebrovascular Accident\***

	Time After Cerebrovascular Accident			
	1 wk	1 mo	6 mo	18 mo
<b>Visuospatial</b>				
Line Bisection Test	L neglect†	WNL	WNL	WNL
Benton's R/L-personal (raw score)	8/8	...	12/12	12/12
Benton's R/L-extrapersonal (raw score)	1/6‡	...	6/8	6/8
Benton's Facial Recognition (raw score)	41	...	39‡	43
Benton's Line Orientation (raw score)	3/10‡	9/10	7/10	4/10‡
Rey-Osterreith Figure (percentile)	<1‡	<1‡	9‡	20
WAIS-R Block Design (SS)	5	8	7	7
<b>Memory</b>				
WMS-R				
Logical memory (raw score)				
Immediate			10/25	9/25
Delayed			8.25	4/25
Paired Associates (WMS) (total score)			7.5	5.5
Visual reproduction (raw score)			27/41	30/42
Immediate			27/41	28/41
Delayed				
Wepman's Visual Memory (raw score)	9/16‡	11/16‡	14/16	12/16‡
<b>Praxis</b>				
Buccofacial (raw score)	3/3	...	3/3	3/3
Limb gesture (raw score)	2/3†	...	3/3	3/3
Ideomotor (raw score)	2/3†	...	3/3	3/3

\*WNL indicates within normal limits; WAIS-R, Wechsler Adult Intelligence Scale-Revised; SS, age-corrected standard score; and WMS, Wechsler Memory Scale.

†Mildly impaired.

‡Moderately to severely impaired.

Visuospatial assembly of block designs<sup>11</sup> was borderline. Facial recognition<sup>7</sup> was low average. Visual scanning speed on Trails A was severely impaired and he could not perform Trails B.<sup>12</sup> Verbal memory was not assessed due to the severity of his language disturbance. Simple visual recognition memory<sup>13</sup> was defective. Buccofacial praxis, limb gestures, and ideomotor praxis were all performed without difficulty.<sup>6</sup>

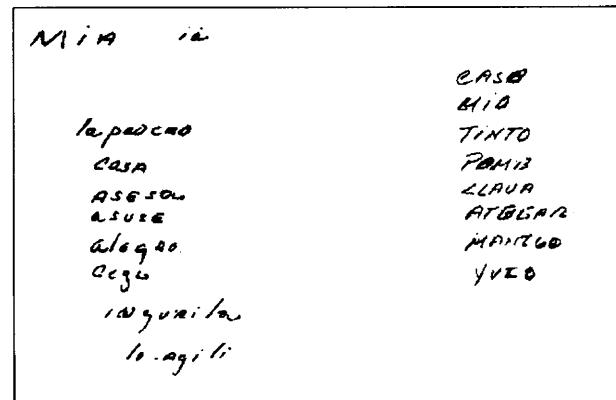
There were no indications of depression or subjective complaints of mood disturbance. His affective presentation was characterized by a jovial mood, with diminished concern regarding his neurologic symptoms and communication impairments.

## COURSE AND FOLLOW-UP EXAMINATIONS

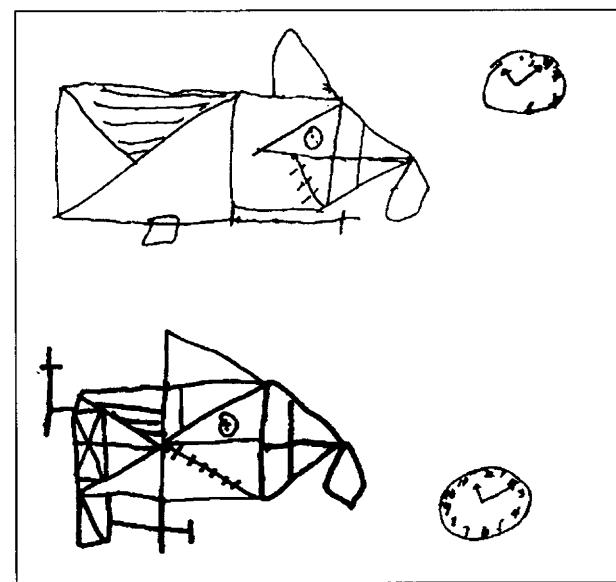
The patient's medical and neurologic condition gradually stabilized and he was transferred to the rehabilitation center 3 weeks after his cerebrovascular accident. On admission to the neurologic rehabilitation service, the left upper extremity was hypotonic with a documented strength of 1/5

and 4/5 for the left lower extremity. There was an impairment of fine and gross motor control of the left lower extremity associated with increased tone, but he managed to ambulate without an assistive device. Reflexes were exaggerated over the left side of the body to 3/4. Sensory examination remained unchanged. Measurable improvement was noted during this admission and he was subsequently followed up by outpatient neurology and neurorehabilitation services. Follow-up neuropsychologic examination at 1 month revealed a persistent and severe expressive language deficit. Spontaneous speech was minimal, and characterized by effortful, nonintelligible output. Repetition, naming, and word writing to dictation were similarly defective. Aural comprehension of syntactical relations also remained severely impaired, but improved performance was noted in writing numbers and letters to dictation, sentence copying, and recitation of simple overlearned material (ie, days of the week). Left hemispatial attention also improved on the letter cancellation test. He was again unable to spontaneously draw a clock, but reproduction to copy now resulted in appropriate spatial distribution of numbers. Reproduction of the Rey figure revealed improved attention to left visual field details, but he exhibited perseverative errors, and his overall score remained within the impaired range. Immediate and delayed incidental memory for the Rey figure were low average. Line bisection, assembly of block designs, and judgment of line orientation were within normal limits. Visual discrimination of geometric designs and visual recognition memory remained impaired. Again, it was not possible to formally assess verbal memory due to the severity of his dysphasia. There were no indications of depression. Affect was jovial, and unsuccessful attempts to communicate were accompanied by spontaneous laughter and jocularity.

A 6-month follow-up neurologic examination demonstrated increased tone of the left upper extremity with diminished fine and gross motor skills. Movement of this extremity occurred in a flexor-synergy pattern. Independent isolated movements were evident in the left lower extremity with minimally increased tone and mild circumduction gait. A magnetic resonance imaging scan again showed areas of prolonged T<sub>1</sub>- and T<sub>2</sub>-relaxation time involving the frontal and anterior-parietal lobes, as well as the insula and striatum. The involved areas were now more sharply defined and there was enlargement of the frontal horn and body of the right lateral ventricle. The findings were consistent with chronic infarction in the distribution of the anterior and middle cerebral arteries. Again, no signal abnormalities were identified in the left hemisphere. Concurrent neuropsychologic testing revealed subtle improvement in expressive skills. Spontaneous verbalizations were slow, effortful, agrammatic, and characterized by brief utterances (2- to 3-word phrases) and phonemic paraphasias. Verbal and nonverbal orolingual manipulations were also executed slowly and effortfully. He recited the days of the week effortfully but accurately. However, he was unable to recite the days backward or count from 20 backward to 1. The MAE-S performance scores revealed perva-



**Figure 3.** Left, Word writing to dictation on initial neuropsychologic examination. Dictated words are as follows: *mio, tira, casa, perro, primo, llave, yate, entregar, manchado*. Right, Word writing to dictation on 6-month follow-up neuropsychologic examination. Dictated words are as follows: *casa, mia, tira, perro, llave, entregar, manchado, and yate*.



**Figure 4.** Top, Reproduction of the Rey-Osterreith Complex Figure and spontaneous clock drawing on initial neuropsychologic examination. Bottom, Reproduction of the Rey-Osterreith Complex Figure and spontaneous clock drawing on 6-month follow-up neuropsychologic examination.

sive residual expressive impairments in visual naming, sentence repetition, and word fluency. Spontaneous writing and writing words to dictation also remained defective (Figure 3, right). He was able, however, to write numbers and letters to dictation and copy sentences with 100% accuracy. Written comprehension of words and phrases improved to within normal limits, but he continued to exhibit marked difficulty in the Token Test. His ability to perform simple arithmetic calculations improved, but was still below expectation.

Within the visuospatial domain, right-left orientation, Block Design, line bisection, spontaneous clock drawing, facial recognition, and judgment of line orientation were scored within the low average to average range. Copy of the Rey figure improved to the borderline range (Figure 4, bottom). He continued to use a segmented approach in the reproduction of this figure, and subtle omis-

sion of left-sided details and perseverative errors were still evident. However, the line bisection test did not reveal left visual field inattention. Visual psychomotor scanning on Trails A was slow but accurate. Shifting of letter-number sets on Trails B remained severely impaired.

Immediate and delayed memory for logical discourse information on the Wechsler Memory Scale-Revised subtest<sup>14</sup> were low average, but paired associate learning was moderately impaired. On this latter task, he managed to recall correctly all semantically related word associations, but none of the unrelated word pairs. Immediate and delayed visuographic memory on the Wechsler Memory Scale-Revised were low average. Visual recognition memory was within normal limits. Incidental recall of the Rey figure decayed to the mildly impaired range. All buccofacial, limb gestures, and ideomotor praxis items were performed with 100% accuracy.

Affect remained jovial, with indifference toward his neurologic limitations and communication problems.

Neurologic progress was complicated by the development of partial motor seizures involving the left side of his face and left hand, with secondary generalization on June 12, 1990. He was effectively treated with carbamazepine and maintained at therapeutic levels. A follow-up electroencephalogram on July 24, 1990, demonstrated persistent slowing over the right hemisphere with no epileptiform activity.

An 18-month follow-up neurologic examination showed persistent left facial and upper extremity weakness, associated with increased tone. Other aspects of his neurologic examination remained unchanged, but continued functional improvement in ambulation capacity was documented. Serum carbamazepine levels were normal, without clinical or laboratory evidence of toxicity. Neuropsychologic testing revealed a stable pattern of language performance. Spontaneous speech was minimal, with short, effortful utterances containing phonemic substitutions. Oral agility in the performance of simple orolingual manipulations remained impaired. A subtle improvement was evident in the recitation of automatized sequences, as he recited the days of the week correctly and made one error reciting the months. Mental tracking also improved. He recited the days backward and made two errors counting from 20 backward to 1. On the MAE-S subtests, persistent deficits were evident in word retrieval, sentence repetition, controlled word association, and aural comprehension on the Token Test. Word writing to dictation was also defective, but he managed to write letters and numbers to dictation without difficulty, and copy sentences with 100% accuracy. Simple arithmetic calculations continued to be performed with 50% accuracy. All praxis items were performed without errors.

Visuospatial skills also remained relatively stable, with the exception of a decline in the judgment of angles. No significant changes were evident in right-left orientation, constructional praxis, visual discrimination, line bisection, and facial recognition. Memory testing revealed decreased performance in the delayed recall of logical discourse informa-

tion, new verbal learning, and incidental memory for the Rey figure. Visuographic memory on the Wechsler Memory Scale-Revised and visual recognition memory remained within the low average range. Affect remained unconcerned and jovial.

## COMMENT

Our case study is unique in that it used neuropsychologic and magnetic resonance imaging data to examine longitudinally the course of crossed aphasia in a young dextral monolingual man without a familial history of left handedness. In this patient, primary compromise of the right frontal, anterior-parietal, and underlying brain regions resulted in a classic Broca's type dysphasia. This clinicopathologic correlation is compatible with the view that the language disturbance in crossed aphasia is a "mirror" representation of that seen in cases of uncrossed aphasia.<sup>15</sup> This clinical presentation has been found to occur more frequently than other forms of anomalous language profiles in patients with crossed aphasia.<sup>2</sup>

Visuoperceptual disturbances and left hemifield inattention have been reported in case studies of crossed aphasia,<sup>15-20</sup> but most studies do not address the nature and evolution of these impairments over time. In our case, initial evaluation 1 week after cerebrovascular accident revealed left visual field inattention and pervasive visuospatial/constructive deficits that resolved gradually over time. Segmentation, perseverative errors, and overall preservation of spatial relationships in the reproduction of a complex line drawing suggest that subtle residual constructional dyspraxia was partly related to an "executive" disturbance. While it is possible that both language and visuospatial functions are lateralized exclusively to the right hemisphere, this is considered to be a remote possibility since there are no radiologic or electrophysiologic data to indicate dysfunction of the left hemisphere. Conversely, the remarkable recovery of nonverbal cognitive functions in the context of a large right-hemispheric lesion raises the possibility of bilateral representation or crossed hemispheric dominance for visuospatial skills. This pattern of reversed lateralization of nonverbal cognitive skills has been recently reported in patients with lesions to the left hemisphere.<sup>21</sup>

When praxis performance is studied in patients with crossed aphasia, buccofacial skilled movements are typically impaired, with relative preservation of limb praxis.<sup>2,22,23</sup> Our findings of severe impairment in simple orolingual agility and language skills, with preserved buccofacial and ideomotor praxis indicates a dissociation between praxis and language<sup>24</sup> and supports the view that ideomotor praxis may be independent of hemispheric language dominance.<sup>2</sup>

Memory functioning in crossed aphasia has been sparsely investigated and findings have been inconsistent. In our patient, performance on nonverbal memory tasks followed a course similar to that described above for visuospatial skills, with recovery to relatively normal levels of performance by

the 6-month follow-up. It was not possible to formally assess verbal memory during the acute and subacute stages of recovery due to the severity of his language disturbance. At 6 months after cerebrovascular accident, the patient demonstrated relatively preserved memory for logical discourse information. On paired associate learning, our patient exhibited selective difficulty learning semantically unrelated word pairs, a pattern of performance that may also represent "executive" dysfunction rather than a true memory disorder.<sup>25,26</sup> The observation that verbal memory was disproportionately compromised relative to visual memory raises the possibility of crossed lateralization of memory skills. Although controversy exists as to the neurologic substrates involved in the expression and comprehension of emotional responses, hemispheric differences in the regulation of emotional behaviors are well established. Patients with left anterior cerebral involvement and nonfluent dysphasia frequently experience a "catastrophic" reaction characterized by profound depression.<sup>27,28</sup> Conversely, patients with left posterior cerebral lesions and those with right-hemispheric compromise typically show indifference, apathy, and diminished insight into their cognitive alterations.<sup>28</sup> Our patient's affective presentation was not typical of that seen in cases of Broca's dysphasia following left anterior cerebral involvement in that he consistently displayed an inappropriate jovial mood and indifference toward his neurologic and cognitive limitations. At no time were depressive symptoms clinically evident or reported by the patient. Of particular interest is the observation that his unsuccessful attempts to communicate, typically a source of great frustration in patients with Broca's aphasia, were accompanied by spontaneous laughter and jesting. These findings suggest that depression in patients with Broca's dysphasia may be independent of the language disturbance and reflects compromise of underlying brain systems responsible for the regulation of mood. Regarding affective components of communicative behavior, our patient's facial expression was normal and he managed to identify emotional facial expressions to confrontation without difficulty.

The mechanisms involved in atypical cerebral localization are yet to be determined. The presence of perinatal or early developmental trauma has been related to the crossed lateralization of language and other cognitive functions.<sup>29</sup> Similarly, disorders of neuronal migration during early stages of fetal development can result in a wide range of neurologic disturbances, which may be associated with anomalous patterns of cognitive hemispheric dominance.<sup>30</sup> However, in cases of crossed aphasia where there is no history of neuropathologic insult or aberrant prenatal development, the cause of crossed hemispheric dominance remains poorly understood. Longitudinal case studies of similar patients with crossed lateralization of cognitive functions may provide important insight regarding the clinical manifestations of these rare disorders.

Accepted for publication December 8, 1992.

We thank Arthur Benton, PhD, and Rachel Tomer, PhD, for reviewing the manuscript and Esther Feldman, PhD, for assisting in the administration of neuropsychological measures.

Reprint requests to the Division of Neuropsychology, Department of Neurology, University of Miami School of Medicine, 1150 NW 14th St, Suite 715, Miami, FL33136 (Dr Rey).

## REFERENCES

1. Zangwill OL. Two cases of crossed aphasia. *Neuropsychologia*. 1979;17:167-172.
2. Alexander MP, Fischette MR, Fisher RS. Crossed aphasia can be mirror image or anomalous: case reports, review and hypothesis. *Brain*. 1989;112:953-973.
3. Oldfield RC. The assessment and analysis of handedness: the Edinburgh inventory. *Neuropsychologia*. 1971;9:97-113.
4. Rey GJ, Benton AL. *Multilingual Aphasia Examination-Spanish: Administration Manual*. Iowa City, Iowa: AJA Associates Inc; 1991.
5. Dunn LM, Padilla ER, Lugo DE, Dunn LM. *Test de Vocabulario en Imágenes Peabody*. Diablo, Calif: Dunn Educational Services Inc; 1986.
6. Goodglass H, Kaplan E. *The Assessment of Aphasia and Related Disorders*. 2nd ed. Philadelphia, Pa: Lea & Febiger; 1983.
7. Benton AL, des Hamsher K, Varney NR, Spreen O. *Contributions to Neuropsychological Assessment*. New York: Oxford University Press Inc; 1983.
8. Osterreith PA. Le test de copie d'une figure complexe. *Arch Psychol*. 1944; 30:206-356.
9. Benton AL, Varney NR, des Hamsher K. Visuospatial judgment: a clinical test. *Arch Neurol*. 1978;35:364-367.
10. Wepman JM, Morency A, Seidl M. *Visual Discrimination Test*. Los Angeles, Calif: Western Psychological Services; 1975.
11. Wechsler D. *WAIS-R Manual*. New York, NY: Psychological Corp; 1981.
12. Reitan RM, Wolfson D. *The Halstead-Reitan Neuropsychological Test Battery*. Tucson, Ariz: Neuropsychology Press; 1985.
13. Wepman JM, Morency A, Seidl M. *Visual Memory Test*. Los Angeles, Calif: Western Psychological Services; 1975.
14. Wechsler D. *Wechsler Memory Scale-Revised Manual*. San Antonio, Tex: Psychological Corp; 1987.
15. Henderson VW. Speech fluency in crossed aphasia. *Brain*. 1983;100:1-8.
16. Carr MS, Jacobson T, Boller F. Crossed aphasia: analysis of four cases. *Brain Lang*. 1981;14:190-202.
17. Hindson DA, Westmoreland DE, Carroll WA, Bodmer BA. Persistent Broca's aphasia after right cerebral infarction in a right hander. *Neurology*. 1984;34:387-389.
18. Yarnell PR. Crossed dextral aphasia: a clinical radiological correlation. *Brain Lang*. 1981;12:128-139.
19. Barraquer-Bordas L, Mendilaharsu C, Peres-Serra J, Mendilaharsu SA, Grau-Veciana JM. Estudio de dos casos de afasia cruzada en pacientes manidextros. *Acta Neurol Latinoam*. 1963;9:140-148.
20. Haaland KY, Miranda F. Psychometric and CT scan measurements in a case of crossed aphasia in a dextral. *Brain Lang*. 1982;17:240-260.
21. Fisher RS, Alexander MP, Gabriel C, Gould E, Milione J. Reversed lateralization of cognitive functions in right handers: exceptions to classical aphasiology. *Brain*. 1991;114:245-261.
22. Basso A, Capitani E, Laiacona M, Zanobio ME. Crossed aphasia: one or more syndromes? *Cortex*. 1985;21:25-45.
23. Wechsler AF. Crossed aphasia in an illiterate dextral. *Brain Lang*. 1976;3:164-172.
24. DeRenzi E, Motti F, Nichelli P. Imitating gestures: a quantitative approach to ideomotor apraxia. *Arch Neurol*. 1980;37:6-10.
25. Levin HS, Goldstein FC, Williams DH, Eisenberg HM. The contribution of frontal lobe lesions to the neurobehavioral outcome of closed head injury. In: Levin HS, Eisenberg HM, Benton AL, eds. *Frontal Lobe Function and Dysfunction*. New York, NY: Oxford University Press Inc; 1991.
26. Stuss DT, Benson DF. The frontal lobes and control of cognition and memory. In: Perecman E, ed. *The Frontal Lobes Revisited*. New York, NY: IRBN Press; 1987.
27. Benson DF. *Aphasia, Alexia, and Agraphia*. New York, NY: Churchill Livingstone Inc; 1979.
28. Heilman KM, Watson RT, Bowers D. Affective disorders associated with hemispheric disease. In: Heilman KM, Satz P, eds. *Neuropsychology of Human Emotion*. New York, NY: The Guilford Press; 1983.
29. Strauss E, Wada J. Lateral preferences and cerebral speech dominance. *Cortex*. 1983;19:165-177.
30. Brown MC, Levin BE, Ramsay RE, Landy HJ. Comprehensive evaluation of left hemisphere type I schizencephaly. *Arch Neurol*. In press.