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Crossed Aphasia in a Patient with Complex Partial Seizures: Evidence from Intracarotid Amobarbital Testing, Functional Cortical Mapping, and Neuropsychological Assessment*

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ABSTRACT

We report a right-handed patient who became transiently aphasic following a right temporal lobectomy for control of intractable complex partial seizures. Preoperative intracarotid amobarbital testing revealed right-hemisphere language dominance, although bilateral language representation was present. Memory testing during unilateral electrical hippocampal simulation with depth electrodes indicated reliance on left-hemisphere mesial temporal lobe structures for verbal memory. Functional mapping for language during surgery established several right perisylvian regions that, when stimulated, produced speech arrest and/or paraphasic substitution. One-year follow-up neuropsychological assessment demonstrated an increase in verbal learning and decrease in visual memory, a pattern associated with patients who have undergone right temporal lobectomy. These data demonstrate that (1) right cerebral language dominance can be observed when ipsilateral seizure onset is present (2) verbal memory and language dominance are not necessarily linked, and (3) some reported cases of crossed aphasia may in fact have bilateral language representation.

Crossed aphasia refers to language disturbance in right-handed individuals following injury to the right cerebral hemisphere (Bramwell, 1899). Schweiger, Wechsler, and Mazziotta (1987) summarized the possible mechanisms underlying crossed aphasia. These include: (1) control of right hand dominance

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by the right hemisphere which is also dominant for language, (2) a simultaneous lesion of the left hemisphere that produces language disturbance but is insufficiently large to be detected by CT, (3) an arrested developmental stage of language lateralization, and (4) bilateral language representation.

A fundamental issue remains, consequently, whether crossed aphasics represent a normal, albeit small, variant of the population, or whether an occult left-hemisphere injury that cannot be detected contributes to the language disturbance. In a caveat regarding reports of crossed aphasia, Boller (1973) stressed that an overrepresentation of tumor and trauma etiologies suggests that many cases actually reflect bilateral damage. We report an individual who underwent a right temporal lobectomy and who became transiently aphasic following surgery. Convergent evidence was obtained to indicate right-hemisphere language representation, allowing the hypotheses of Schweiger et al. (1987) to be tested.

CASE REPORT

The patient was a 37-year-old, right-handed, white female with 14 years of education with no history of familial sinistrality. Handedness was determined on the basis of the Benton Handedness Inventory (Benton, Meyers, & Polder, 1962). She was a full-term child and was delivered by Caesarian section. At age 3, she had a febrile illness during which she had several convulsions. No further seizures were present until age 5, when she began having spells in her sleep which consisted of lip smacking and grunting noises with labored respirations. She was started on phenobarbital and hydantoin at age 6, and had approximately two generalized tonic-clonic seizures per year until approximately 13 years of age. Phenobarbital was discontinued while she was a teenager due to allergic reaction. She then began experiencing different kinds of spells. The first type consisted of a few seconds of queasiness accompanied by a "butterfly" sensation in her stomach and a bitter taste in her mouth. These episodes occurred 3-5 times/day and lasted several seconds to 1 min. A second type of seizure began with a prodrome of feeling ill and "unreal." During these episodes, she would walk awkwardly and speak incoherently. These episodes lasted between 2-5 min. At the time of her evaluation for surgery, she had not experienced a generalized seizure for 5 years. Preoperative medication included carbamazepine (1400 mg/day) and phenytoin (600 mg/day). MR scan revealed no evidence of a structural lesion.

EEG and Prolonged Monitoring Investigation.
The patient underwent presurgical EEG evaluation with standard sphenoidal and depth electrodes. Interictally, there were frequent sharp waves from the right mesial temporal region with phase reversal at the right sphenoidal electrode. However, EEG seizure onsets during noninvasive recordings were

localized poorly. Depth electrodes subsequently were implanted via a vertex trajectory to traverse the amygdalae and anterior hippocampi bilaterally (Flanigin & Smith, 1987). Interictally, there were bilateral independent epileptiform discharges from the mesial temporal regions which were much more prominent from the right side. Two clinical and four subclinical seizures were recorded, all of which originated simultaneously from the right amygdala and right hippocampus. In the one clinical seizure with adequate behavioral data, the patient stopped conversing and was unable to respond to verbal stimuli.

Language Testing.
The language screening was designed to assess all major language domains (Benson & Geschwind, 1985). To test confrontation naming, four line drawings were presented, and the patient was to name either the picture (e.g., coat), or a portion of the picture (e.g., sleeve). Eight possible points were available. Descriptive naming was assessed by asking the patient to name eight items following a brief description. Comprehension was measured using a modified version of the Token Test and having the patient execute commands of increasing syntactic complexity beginning with simple commands (e.g., "touch the white circle") and progressing to inverted syntax (e.g., "point to the white triangle after you point to the black circle"). Six comprehension commands were presented.

Verbal fluency was tested using the Controlled Oral Word Association subtest from the Multilingual Aphasia Examination (Benton & Hamsher, 1978); the reported score has been adjusted for years of education. Semantic fluency consisted of animal or vegetable name generation (60 s). Repetition was assessed employing three sentences. Four reading tasks were administered, beginning with a single word and concluding with three simple sentences. Five writing to dictation items were presented, examples of which can been seen in Figure 4. Other tests of language function included the Visual Naming and Token Tests subtest from the Multilingual Aphasia Examination (Benton & Hamsher, 1978).

Additional Neuropsychological Tests.
The patient was administered the WAIS-R as a measure of general cognitive ability. Other tests of neuropsychological function included Visual Form Discrimination (Benton, Hamsher, Varney, & Spreen, 1983), Judgement of Line Orientation (Benton et al., 1983), and the Rey-Osterrieth Complex Figure (CF; Rey, 1941). Verbal learning was assessed with the Selective Reminding test (Form 2; Hannay & Levin, 1985) and Serial Digit Learning (SD9; Benton et al., 1983). Consistent Long-Term Retrieval (CLTR) was our dependent measure for Selective Reminding. CLTR is a measure of the patient's ability to retrieve information from memory without the need of subsequent prompting by the examiner. Serial digit learning is a digit supraspan learning task. Visual-spatial

memory was assessed using immediate (30-s) and delayed (30-min) CF recall.

Neuropsychological Tests Results.

Neuropsychological performance levels are depicted in Table 1, and language screening results are presented in Table 2. Her spontaneous speech was fluent, prosodic, and free from aphasic errors. Performance on language testing was normal. Naming was intact on both the MAE naming subtest and on our language screening battery. The patient was able to adequately execute complex two-stage commands involving inverted syntax. Generative verbal fluency was at the 11 th centile for Controlled Oral Word Association, and appeared normal for animal name generation. Repetition was intact. Reading and writing were normal. Praxis and right-left orientation were normal.

Memory was borderline for both verbal and visual-spatial material. Selective Reminding CLTR performance was severely impaired using the normative tables of Larrabee, Trahan, and Levin (1986). In contrast, serial digit learning was at the 10th centile. Immediate (30-s) and delayed (30-min) CF recall was normal. However, there was qualitative evidence on CF delayed recall to suggest right-temporal dysfunction (Loring, Lee, & Meador, 1988). These qualitative errors included diamond (element 14; see Lezak, 1983) on a stem, misplacement of the diamond (element 14), and inversion of the upper triangle (element 9).

Table 1

Results of pre- and postoperative neuropsychological assessments. With the exception of IQ scores and generative fluency measures, performances are expressed as number correct/total possible points.

	9-17-86	10-22-87
Full Scale IQ	92	89
Verbal IQ	85	88
Performance IQ	95	97
Multilingual Aphasia Examination Naming	44/60	38/60
Multilingual Aphasia Examination Verbal Fluency[1]	26	24
Multilingual Aphasia Examination Token Test	44/44	44/44
Visual Form Discrimination	29/32	26/32
Judgement of Line Orientation	20/30	25/30
Facial Recognition	40/54	39/54
Selective Reminding CLTR (6-trial)	15/72	26/72
Serial Digit Learning	6/24	--
Complex Figure Immediate Recall	15/36	6/36
Complex Figure Delayed Recall	16/36	0/36
Logical Memory Immediate	--	22/50
Logical Memory Delay	--	15/50
Visual Reproduction Immediate	--	36/41
Visual Reproduction Delay	--	19/41

[1] Corrected score for three 60-second trials

Table 2
Results of serial language screening assessments.
Scores are expressed as the number of correct responses over the number of trials.
Fluency measures reflect number of words generated for given interval.

	Baseline	Day 1	Day 4	1-year
Visual Naming	8/8	2/8	4/8	8/8
Descriptive Naming	8/8	0/8	2/8	7/8
Comprehension	6/6	2/6	2/6	6/6
Verbal Fluency[1]	26	0	9	24
Semantic Fluency[2]	16	6	6	11
Repetition	5/5	2/5	5/5	5/5
Reading	4/4	3/4	3/4	4/4
Writing	5/5	3/5	3/5	4/5

[1] Corrected score for three 60-second trials
[2] Generated samples for a single 60-second interval

Angiography/Intracarotid Amobarbital Testing.
Angiography performed immediately prior to intracarotid amobarbital testing revealed normal vasculature with only slight crossflow in the anterior cerebral arteries. The amount of crossflow was symmetrical for left and right arteriograms. The amobarbital testing was videotaped which allowed detailed review.

Intracarotid amobarbital testing was conducted with the patient in the supine position on the angiography table. At the beginning of the procedure, the patient was instructed to count repeatedly from 1-20. Following demonstration of a contralateral hemiplegia, multiple cognitive tasks were presented to assess comprehension, naming, repetition, and memory.

An initial injection of 100 mg was administered via the right internal carotid artery.Following injection, the patient displayed single number perseveration (i.e., "3,3,3,3,") without complete speech arrest. Ten seconds following the injection, she was completely unable to execute simple single-stage commands. Forty-five seconds post injection, she was still unable to respond to command, and she began counting after each request for performance. Her ability to name, tested at 145 s post injection, was characterized by phonemic substitution (e.g., "pine" for "pipe"), and repetition was impaired ("Mary had a lit dram, ... Mary lad..."). She correctly solved only one of the two visual discrimination items presented.

Following return to baseline strength and language function, the left hemisphere was tested. An injection of the left internal carotid artery, also 100 mg, produced an arrest of counting accompanied by an inability to execute simple commands. However, she was able to provide her name when questioned at 45 seconds. At 100 seconds, she made comprehension errors when attempting to execute two-stage commands involving inverted syntax. All subsequent verbal tasks, involving naming, comprehension, and repetition, were normal. Both visual discrimination items were correctly solved. The amobarbital evaluation suggested bilateral language, with greater language representation in the right hemisphere.

Hippocampal Stimulation/Memory Testing.
The contribution of each hippocampus to verbal memory function was tested by assessing verbal memory during unilateral electrical hippocampal stimulation with depth electrodes (Loring et al., 1988). Hippocampal stimulation was applied using a balanced square wave pulse (60 Hz, 1 ms/phase) with a Grass constant current generator. A 7-s train, followed by 3 s of no stimulation, was passed repeatedly between contacts 1-3 which were separated by a total of 10 mm. The current was 0.45 mA/phase for both left and right hippocampal stimulation.

Verbal memory functioning was evaluated using the Brown-Peterson distractor technique employing an interpolated delay of 20 s. Ten trials, consisting of 3 words each, were administered during each condition yielding a total possible score of 30. Initial depth electrode stimulation was performed on the left hippocampus, with a 30-min delay between left and right hippocampal simulation. The following performance levels were obtained (correct/total words presented): Baseline = 18/30; Left Stimulation = 13/30; Right Stimulation = 21/30.

Functional Cortical Mapping.
Functional mapping for localization of the facial sensorimotor cortex, and frontal and temporal language cortices was carried out after exposure of the right lateral anterior temporal lobe and fronto parietal opercular area prior to resection (see Figures 1 and 2). Stimulation was provided by a Grass constant current generator. The stimulus was a 60 Hz, balanced square wave pulse of 1 ms per phase, with 1 ms between phases. Stimulation was at 1.5 mA/phase, 3 mA peak-to-peak. Results of both sensorimotor and speech mapping are presented in Table 3. For mapping of the posterior frontal lobe, the patient was instructed to count repeatedly from 1-20. Bipolar stimulation trains of approximately 3-s duration were applied repeatedly to all areas of exposed inferior and middle frontal gyri. Temporal lobe mapping was performed while the patient repeatedly recited a nursery rhyme (i.e., "Mary had a little lamb"). Repeated 3-s stimulus trains were applied to all exposed areas of the superior, middle, and inferior temporal gyri. The patient's level of arousal and cooperation was sufficient to allow satisfactory mapping of cortical areas. With the exception of position 10, her pattern of cortical language localization was similar to that which we observe during functional mapping of the left hemisphere in most patients.

In addition, the right sylvian fissure displayed a more inferior slope as it approached its termination at the sylvian point than is standard for the right hemisphere (LeMay & Culebras, 1972). Although the orientation was not typical of the right hemisphere, its slope was similar to the left sylvian fissure in

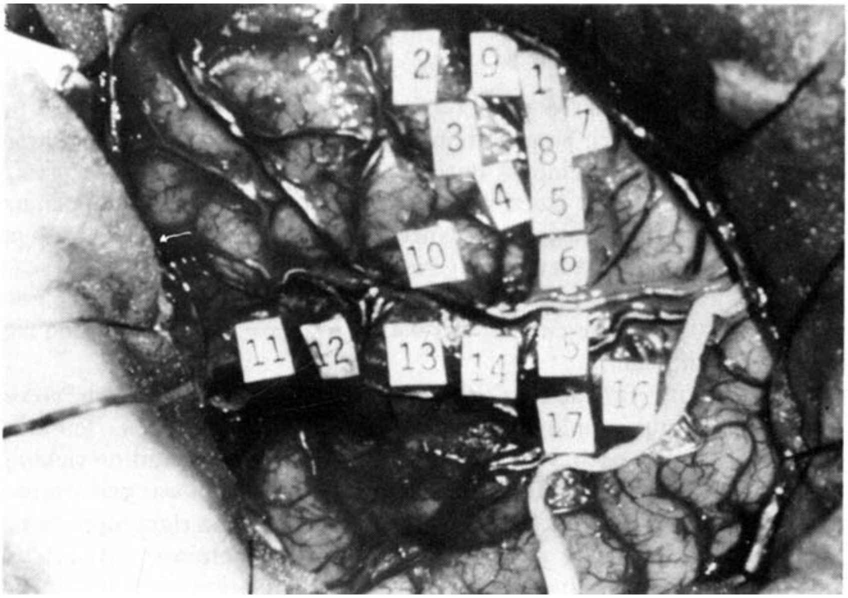

Figure 1. Electrical stimulation sites that produced functional deficits from the patient.

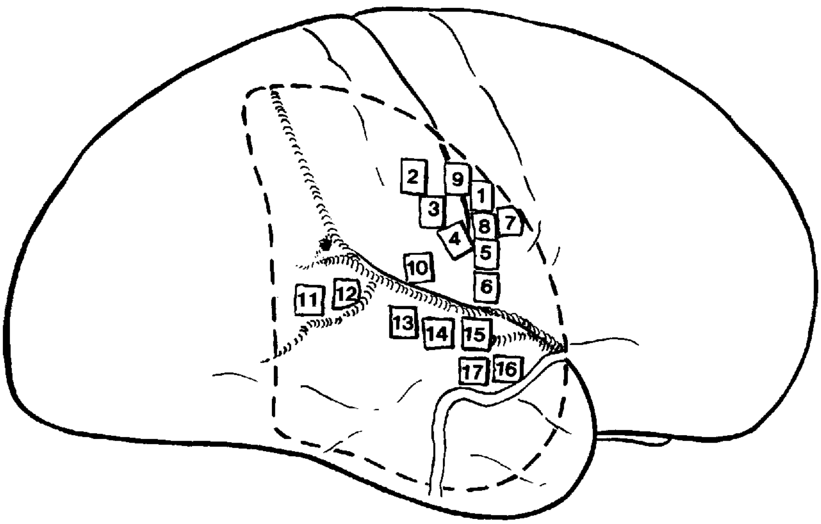

Figure 2. Stylized representation of lateral brain surface indicating position of stimulation sites relative to standard landmarks.

Table 3
Results of functional cortical mapping

Stimulus Number	Experience
SENSORIMOTOR MAPPING	
1	Inability to open mouth
2	Funny sensation in left side of mouth
3	Tingling sensation in both sides of mouth
4	Tingling sensation in left side of mouth
5	Tingling sensation in both sides of mouth
6	Tingling sensation in both sides of mouth
7	Drawing up of mouth bilaterally
8	Generalized mouth movement
9	Slow drawing of mouth, slow opening and closing
SPEECH MAPPING	
10	Interruption of counting. Stimulation anterior to 5,6,7, and 8 failed to produce any further interruption of counting.
11	Recitation interruption with phonemic paraphasic substitution
12	Perseveration of recitation
13	Speech interruption and phrase repetition
14	Arrest in recitation
15	Arrest in recitation
16	Arrest in recitation, one word after stimulus onset
17	Semantic paraphasia. Arrest in recitation, one word after stimulus onset. Stimulation anterior to 16 and along the third temporal convolution failed to produce any arrest of recitation.

left - language - dominant patients. The posterior resection margins extending from the most anterior temporal fossa measured 4.3 cm along the first temporal convolution, 5.5 cm along the third temporal convolution, and 5.2 cm along the parahippocampal gyrus (which included removal of approximately 2 cm of anterior hippocampus). The uncus extended 14 mm across the tentorial margin.

Immediate postoperative CT.

CT performed on the first postoperative day revealed, in addition to the right temporal lobectomy, mild compression of the right lateral ventricle which was believed due to edema (See Figure 3). Mild hemorrhage was observed along the posterior transection margin and medial pia in the right temporal lobe, a finding commonly present following temporal lobectomy.

Immediate postoperative language.

Language assessment using our screening test was conducted the first day following surgery, and again on the fourth postoperative day. Results of these language assessments are included in Table 2. An impairment on all core linguistic functions was observed, with significant reductions seen during naming, comprehension, fluency, and repetition. Less severe impairments were observed for reading and writing, and her errors typically had a perseverative quality to them. This perseveration is clear when examining her writing (Figure

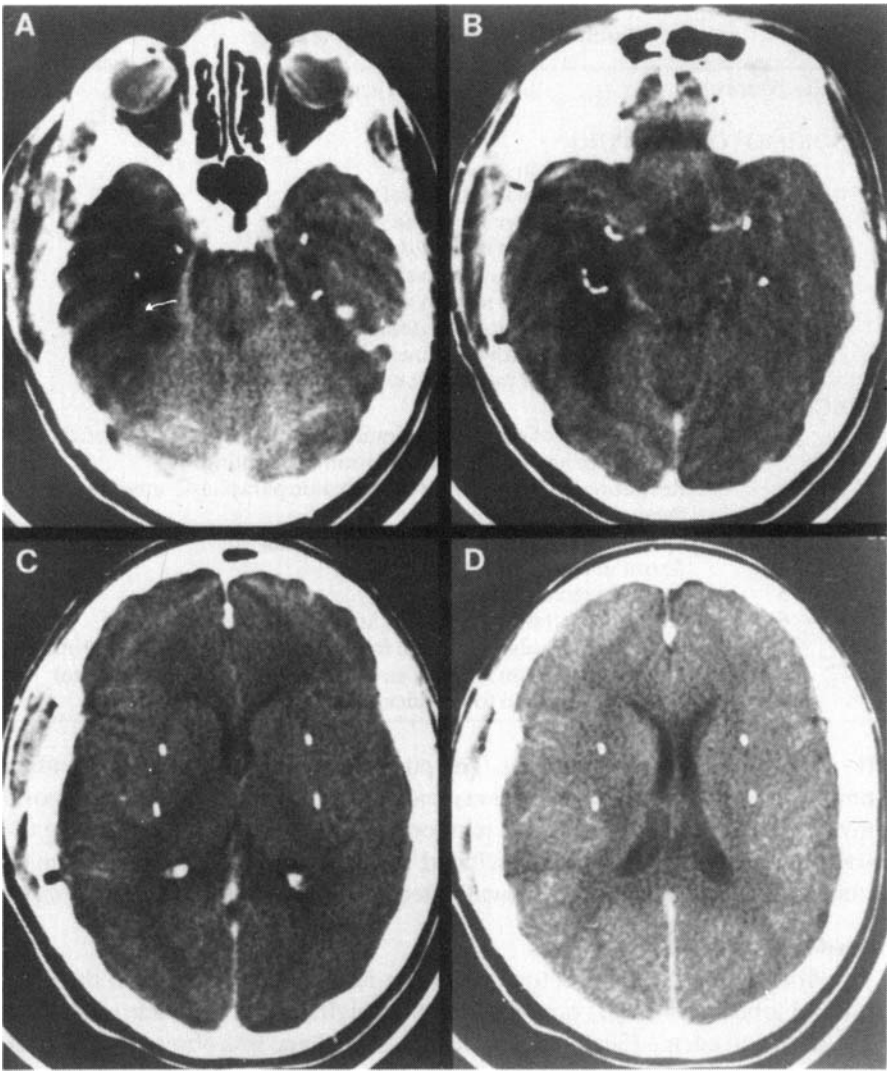

Figure 3. CT scan 1 day post right temporal lobectomy. Note decreased density in area of resection and four symmetric hyper-densities which are depth electrodes. On the right, the electrodes hang free in the resection cavity.

4). After correctly writing "The cat is black" to dictation, her first error was present writing "The weather is cloudy."As can be seen in her attempt to write "The knight slew the dragon," this same error is observed on both attempts. Her writing on the fourth postoperative day was still very perseverative. After she wrote "The knight is cloudy!," she was presented with a new sheet of paper, and she again repeated the same incorrect response. Asked how to spell "dragon," she wrote "Floudys." She then spontaneously remarked, "I spelled

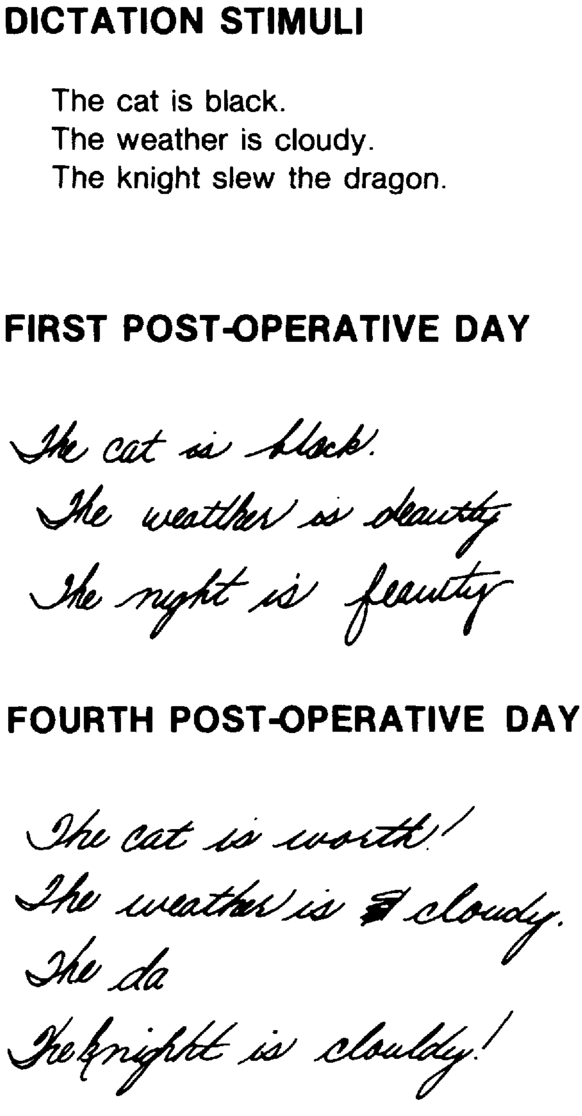

Figure 4. Post-operative writing to dictation.

that wrong. It should be f-o-u-l-d-i-e-s." Her spontaneous speech was fluent but circumlocutious, and her paraphasic responses were typically semantic errors.

No evidence of dyspraxia was present during execution of transitive limb commands. In addition, basic visual-spatial constructional skills were intact, with the patient able to copy a cross and necker cube.

Follow-up Examination.
The patient was seizure free at 1-year follow up. At the time of her assessment, her medication included phenytoin (400 mg/day) and carbamazepine (1200 mg/day). Neuropsychological evaluation was performed with parallel forms of memory tests. Form 4 of the Selective Reminding test from Hannay and Levin (1983) was used to assess verbal memory, and the Taylor Complex Figure (Taylor, 1969) was employed for visual memory assessment. Serial digit learning performance was not obtained. However, the Logical Memory and Visual Reproduction subtests from the Wechsler Memory Scale-Revised (WMS-R; Wechsler, 1987) were administered.

Results of the 1-year follow-up neuropsychological assessment are presented in Table 1, with the follow-up language screening performance included in Table 2. IQ measures were not significantly different from her presurgical performance. Language was essentially at preoperative levels (see Table 2), although several errors were observed. Performance on the Visual Naming subtest from the Multilingual Aphasia Examination (Benton & Hamsher, 1978) was impaired.

Memory evaluation revealed an increase in performance on the Selective Reminding test, and a decrease in CF recall (Table 1). In addition, scores on the Logical Memory Subtest from the WMS-R declined 32% from immediate to delayed recall. This decline was associated with a change in centile ranking from 33 to 25. In contrast, the Visual Reproduction declined 47%, with a corresponding change from the 79th to 12th centile.

DISCUSSION

The present case illustrates that right-hemisphere language dominance can be observed in a right-handed patient with complex partial seizures originating from the right temporal lobe. Although this patient displayed right-hemisphere language dominance, intracarotid amobarbital testing also revealed evidence of left-hemisphere language representation. The language disturbance following left amobarbital injection cannot be explained on the basis of crossflow from the language-dominant right hemisphere. Angiography utilizes pressure injection of dye, increasing the amount of observed crossflow, whereas the intracarotid amobarbital injections are by hand over a 4-s interval. The amount of crossflow in this patient is commonly present in other patients who display unilateral language representation following amobarbital injection.

Of the four hypotheses presented by Schweiger et al. (1987) to account for crossed aphasia, atypical ipsilateral control of right-hand dominance was not supported based upon the demonstration of contralateral flaccid hemiplegia after right amobarbital injection. Simultaneous bilateral lesions cannot be ruled out as contributing to the language disturbance because mesial temporal sclerosis may be present bilaterally. However, for this explanation to be tenable, the left-hemisphere mesial temporal sclerosis would have to be responsible for the language disturbance. This is unlikely because of the longstanding seizure history, and because the postoperative deficits were the result of a right temporal lobectomy. Further, all available evidence confirmed language function and language dominance in the right hemisphere for this patient (e.g., intracarotid amobarbital testing, functional cortical mapping, postoperative language testing). The language impairments during electocortical stimulation were not the result of generalized cerebral disruption. We have mapped the right hemisphere of a patient in whom language dominance could not be determined by intracarotid amobarbital testing due to marked crossflow, and no disruption of language was observed. We have also demonstrated that errors on our language screening test are not observed following right temporal lobectomy in patients who are left hemisphere dominant for language (Loring, Meador, Martin, & Lee, 1989).

Since our patient experienced a febrile seizure at age 3, it is possible that this may have interrupted the normal process of increasing lateralization with age and would support the arrested developmental stage hypothesis. Brown and Hécaen (1976) hypothesized that the degree of lateralization and localization present at the time of injury determines the type of language disturbance. Thus, the early spell may have produced an arrested stage of language lateralization. Although we cannot rule out this possibility, it is an unlikely explanation since a single febrile seizure generally has no appreciable effect on subsequent development (Livingston, 1972). Further, although bilateral speech might be observed with an arrested stage of cerebral language lateralization, it is unlikely that there would be greater language in the right cerebral hemisphere.

Rausch and Walsh (1984) reported that patients with left-hemisphere epileptogenesis were more likely to be right-hemisphere language dominant. Because this patient's seizures originated in the right-hemisphere, and that left-hemisphere epileptogenicity increases the likelihood of contralateral language representation, one would have expected to observe no right-hemisphere language. Yet, our patient not only exhibited right-hemisphere language representation, but also displayed greater language in the right than in the left hemisphere. Further, the slope of our patient's right sylvian fissure was more typical of the angle commonly seen in the left hemisphere. Since the normal slope asymmetry is present during fetal development (Wada, Clarke, & Hamm, 1975), the slope of our patient's sylvian fissure suggests a hard-wired predisposition for her right-hemisphere to develop language. Thus, to the degree that performance by this patient is generalizable to the population of crossed aphasics, mixed cerebral language dominance is the most plausible explanation for language disturbance in dextrals following right-hemisphere pathology.

In patients with crossed aphasia, the relationship of injury to visual-spatial processing has been unclear. If crossed aphasia suggests a mirror cortical representation (*situs inversus*), then visual-spatial deficits should be observed with the same frequency as with left-hemisphere injuries in the general population. In a review of nonverbal disturbances in crossed aphasia, Castro Caldas, Confraria, and Poppe (1987) reported the presence of constructional deficits in 76% of crossed aphasics. This contrasts with constructional apraxia in 45% of right-handed patients with left-hemisphere injuries. Some reports of crossed aphasia described visual-spatial deficits (e.g., Schweiger et al., 1987), while others suggested no significant impairment in visual-spatial functions (e.g., Larrabee, Kane, & Rogers, 1982). Thus, there appears to be substantial patient variability with respect to degree of lateral asymmetry of visual-spatial functions in relation to language dominance.

Our patient exhibited no postoperative deficits on the constructional praxis screening. On the first postoperative day, she was able to copy all shapes, including the relatively difficult cross and necker cube. However, there was other evidence of deficits in visual-spatial processing. During the preoperative workup, there were qualitative errors on the complex-figure recall that are seen more commonly in patients with right temporal seizure onset (Loring et al., 1988). In addition, the only visual discrimination error during amobarbital assessment was observed during the right-hemisphere injection. Consequently, it appears as if this patient retained substantial right-hemisphere representation for visual-spatial functions, despite significant language representation as well.

Her performance on the postoperative neuropsychological assessment revealed relative improvement on Selective Reminding CLTR, and poorer CF memory. Novelly et al. (1984) reported that right-temporal-lobectomy patients display poorer visuospatial memory and improved verbal memory at 1 year as compared to their preoperative baseline. Further, our patient's percentage retention on the Logical Memory and Visual Reproduction subtests was consistent with non-language-dominant right temporal dysfunction. These latter scores should be interpreted cautiously because preoperative performance levels were not obtained, and because the WMS-R has yet to be appropriately validated in clinical populations. However, Chelune and Bornstein (1988) have suggested that percent decline for the Logical Memory and Visual Reproduction subtests is sensitive to lateralized temporal dysfunction. Consequently, it appears that, although this patient had greater right-hemisphere language representation, the right mesial temporal lobe contributed significantly to recent visuospatial memory function.

Additional evidence of left cerebral dominance for verbal memory was obtained with verbal memory assessment during electrical hippocampal stimulation. A decline in verbal memory was present only following left hippocampal stimulation. This pattern suggests relatively greater left hippocampal contribution to recent verbal memory function. We have previously shown that, in patients with right temporal seizure onset, Brown/Peterson memory performance is decreased only during left hippocampal stimulation, presumably due to relatively intact left mesial temporal lobe (Loring et al., 1988). Thus, the pattern of performance during memory stimulation studies by this patient suggests that her left temporal lobe was primarily responsible for verbal memory function, and is consistent with pre- and postoperative neuropsychological memory test results.

No pronounced language deficits were produced by the surgery, although visual naming was in the impaired range. The concern for postoperative aphasia provides the rationale for intraoperative functional mapping of language prior to temporal lobe resection. The immediate postoperative language deficits are postulated to result from edema and/or diaschisis in the residual right cerebral structures. The language deficits following operation appeared to be linguistic in nature and not due to postoperative confusion and/or disorientation. If this patient had sustained a vascular event involving the distribution of the right middle cerebral artery, she would likely have been considered a case of pure crossed aphasia since the language representation of the left hemisphere would remain unknown. Thus, this case illustrates that some cases of crossed aphasia may in fact have bilateral language representation. In addition, this report demonstrates that right cerebral dominance for language can be observed when seizures originate from the right mesial temporal lobe, and indicates that cerebral dominance for verbal memory and language are not necessarily linked.

REFERENCES

Benson, D. F., & Geschwind, N. (1985). Aphasia and related disorders: A clinical approach. In M-M. Mesulam (Ed.), *Principles of behavioral neurology* (pp. 193-238). Philadelphia: F. A. Davis.

Benton, A. L., & Hamsher, K. de S. (1978). *Multilingual aphasia examination.* Iowa City: Department of Neurology, University Hospitals.

Benton, A. L, Hamsher, K. de S., Varney, N. R., & Spreen, O. (1983). *Contributions to neuropsychological assessment.* New York: Oxford University Press.

Benton, A.L., Myers, R., & Polder, G.J. (1962). Some aspects of handedness. *Psychiatrica et Neurologica, 144,* 321-337.

Boller, F. (1973). Destruction of Wernicke's area without language disturbance: A fresh look at crossed aphasia. *Neuropsychologia, 11,* 243-246.

Bramwell, B. (1899). On "crossed" aphasia etc. *Lancet, 1,* 1473-1490.

Brown, J., & Hécean, H. (1976). Lateratlization and language representation. *Neurology, 26,* 183-189.

Castro-Caldas, A., Confraria, A., & Poppe, P. (1987). Non-verbal disturbances in crossed aphasia. *Aphasiology, 1,* 403-413.

Chelune, G. J., & Bornstein, R. A. (1988, August). Memory characteristics of patients with unilateral temporal and nontemporal lesions. Paper presented at the annual meeting of the American Psychological Association, Atlanta, GA.

Flanigin, H. F., & Smith, J. R. (1987). Depth electrode implantation at the Medical College of Georgia. In J. Engel (Ed.), *Surgical treatment of the epilepsies* (pp. 609-612). New York: Raven Press.

Hannay, H. J., & Levin, H. S. (1985). Selective Reminding Test: An examination of the equivalence of four forms. *Journal of Clinical and Experimental Neuropsychology, 7,* 251-263.

Larrabee, G. J., Kane, R. L., & Rodgers, J. A. (1982). Neuropsychological analysis of a case of crossed aphasia: Implication for reversed laterality. *Journal of Clinical Neuropsychology, 4,* 131-142.

Larrabee, G. J., Trahan, D. E., & Levin, H. S. (1986, February). Normative data for the verbal selective reminding test. Paper presented at the annual meeting of the International Neuropsychological Society, Denver, CO.

LeMay, M., & Culebras, A. (1972). Human brain: Morphologic differences in the hemispheres demonstrable by carotid arteriography. *New England Journal of Medicine, 287,* 168-170.

Lezak, M. D. (1983). *Neuropsychological assessment* (2nd Ed.). New York: Oxford University Press.

Livingston, S. (1972). *Comprehensive management of epilepsy in infancy, childhood, and adolescence.* Springfield, IL: Charles C. Thomas.

Loring, D. W., Lee, G. P., Flanigin, H. F., Meador, K. J., Smith, J. R, Gallagher, B. B., & King, D.W. (1988). Verbal memory performance following unilateral electrical stimulation of the human hippocampus. *Journal of Epilepsy, 1,* 79-85.

Loring, D. W., Lee, G. P., & Meador, K. J. (1988). Revising the Rey-Osterrieth: Rating right hemisphere recall. *Archives of Clinical Neuropsychology, 3,* 239-247.

Loring, D. W., Meador, K. J., Martin, R. C., & Lee, G. P. (1989). Language deficits following unilateral temporal lobectomy. *Journal of Clinical and Experimental Neuropsychology, 11,* 41 (Abstract).

Novelly, R. A., Augustine, E. A., Mattson, R. H., Glaser, G. H., Williamson, P. D., Spencer, D. D., & Spencer, S. S. (1984). Selective memory improvement and impairment in temporal lobectomy for epilepsy. *Annals of Neurology, 15,* 64-67.

Rausch, R., & Walsh, G. O. (1984). Right-hemisphere language dominance in right-handed epileptic patients. *Archives of Neurology, 41,* 1077-1080.

Rey, A. (1941). L'examen psychologique dans les cas d'encéphalopathie traumatique. *Archives de Psychologie, 37,* 126-139.

Schweiger, A., Wechsler, A. F., & Mazziotta, J. C. (1987). Metabolic correlates of linguistic functions in a patient with crossed aphasia: A case study. *Aphasiology 1,* 415-421.

Taylor, L.B. (1969). Localization of cerebral lesions by psychological testing. *Clinical Neurosurgery, 16,* 269-287.

Wada, J., Clarke, R., & Hamm, A. (1975). Cerebral hemispheric asymmetry in humans: Cortical speech zones in 100 adults and 100 infant brains. *Archives of Neurology, 32,* 239-246.

Wechsler, D. (1987). *Manual for the Wechsler Memory Scale-Revised.* New York: The Psychological Corporation.