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Amnesia After Unilateral Temporal Lobectomy: A Case Report

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Summary: We report a mixed handed (L>R) patient with exclusive right cerebral language representation who developed a permanent anterograde amnestic syndrome after right anterotemporal lobectomy. Preoperative neuropsychological performance consisted of impaired verbal memory and normal nonverbal memory. Wada memory performance was asymmetrical for objects presented soon after amobarbital injection in conjunction with no memory asymmetry for items presented later in the Wada evaluation. Pre- and postoperative magnetic resonance imaging (MRI) scans showed no structural lesions; however, postoperative MRI hippocampal volume measurements suggested decreased hippocampal volume for the nonresected temporal lobe. These results confirm the risk of anterograde amnesia after unilateral temporal lobectomy and demonstrate that baseline neuropsychological testing may falsely literalize material-specific memory functions in patients with atypical cerebral language dominance. **Key Words:** Epilepsy—Neurosurgery—Temporal lobe—Amnesia—Wada test.

The most vivid clinical example of the importance of mesotemporal lobe structures in memory acquisition is patient H.M., who developed severe and persistent amnesia after bitemporal lobectomy (Corkin, 1984). However, significant memory impairment after unilateral resection has also been reported. Penfield and Milner (1958) postulated that anterograde amnesia after unilateral temporal lobectomy results from contralateral hippocampal dysfunction in addition to the effects of the temporal lobe surgery. Their hypothesis was confirmed in 1 patient who at autopsy had a pale and shrunken hippocampus contralateral to the operation (Penfield and Mathieson, 1974).

Despite the many reports of the mild to moderate memory changes that may occur after temporal lobectomy, few accounts adequately describe postresection amnesia. Furthermore, the magnitude and permanence of the postoperative memory impairment reported differs.

Baldwin (1956) described memory impairment following unilateral temporal lobectomy in 4 of 65 patients. However, Baldwin reported that the initial postoperative memory deficit resolved over time in 2 of the patients. Furthermore, the 2 other cases successfully returned to work. Thus, more of Baldwin's patients appear to have developed a permanent postoperative amnestic syndrome.

Penfield and Milner (1958) described 2 patients who developed significant memory impairment after dominant hemisphere temporal lobectomy. Wechsler Memory Quotient (MQ) decreased from 94 to 72 in the first patient. The second patient developed memory impairment after the second of two temporal lobe operations. When tested 5 years after the second operation, the patient had an MQ of 97, significantly lower than his WAIS Full-Scale IQ of 120. Although this patient eventually returned to work, he was demoted from a civil engineer to a draftsman owing to the magnitude of the postoperative memory impairment (Milner, 1966). A third patient with significant memory decrease after dominant hemisphere temporal lobectomy was also described (Milner, 1966), although in contrast to the previous 2 patients, the patient was right hemisphere language dominant.

Walker (1957) described 4 patients with postop-

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erative memory impairment, although none appeared to be amnestic. The first 2 patients successfully returned to work after right temporal lobectomy. The third patient had borderline intelligence preoperatively, and memory after left temporal lobectomy was variable. In the fourth patient, the left temporal lobe was resected to expose an aneurysm, and Walker (1957) believed that the deficits could be either verbal memory difficulty or mild aphasia. However, Walker (1957) was the first investigator to provide an estimate of memory impairment (not amnesia) after temporal lobectomy (i.e., 10–15%), and these figures subsequently were used to validate results of Wada memory studies (Kløve et al., 1969).

Serafetinides and Falconer (1962) reported that 7 of 34 patients who had undergone right temporal lobectomy displayed recent memory impairment at follow-up. Four of these patients had preoperative memory deficits, however, and a decrease was noted in only 1 of 4 patients administered the Wechsler Memory Scale both pre- and postoperatively. The researchers concluded that "the type of memory defect we are now considering does not correlate with the more formal psychological test results" (p. 254), and stated that the memory deficit was "usually compensated for," indicating an absence of an amnestic syndrome.

Dimsdale et al. (1964) reported a case of amnesia after right temporal lobectomy in a woman who had previously been hospitalized for depression. Her preoperative MQ was 80. Postoperatively, she became confused and paranoid and was admitted to a mental hospital. Three weeks postoperatively, her MQ increased 21 points to 101. The researchers reported that she had other memory difficulties, although she was reportedly oriented for time and place 8 months postoperatively. Owing to her significant psychiatric history, it is difficult to attribute the postoperative memory deficit to a single cause. Furthermore, a sclerotic lesion of the unoperated left hippocampal formation was noted at autopsy (Warrington and Duchen, 1992). Warrington and Duchen (1992) acknowledged the difficulty of determining the age of old inactive lesions, and the temporal lobectomy was performed 25 years before autopsy.

Rausch et al. (1985) described a patient who became amnestic after left temporal lobectomy. Postoperative MRI showed that the nonresected hippocampus was grossly intact, although there was evidence of damage to the internal capsule in the nonresected hemisphere (Rausch et al., 1983).

There appears to be a spectrum of postoperative memory decrease. In some cases, transient memory impairment or subjective memory decline without a prominent correlate with formal neuropsychological testing has been described. In other cases with significant postoperative memory decrease, the magnitude of the impairment was not as severe as that of patient H.M.

We report a case of postoperative amnesia after right temporal lobectomy in a patient with right cerebral language dominance. The patient underwent comprehensive preoperative evaluation at two epilepsy surgery centers, including invasive electrode implantation, MRI, pre- and postoperative neuropsychological assessment, and Wada language and memory testing.

CASE REPORT

A 27-year-old mixed-handed (L>R) man with a high school education involving some special education classes wrote with his right hand but used his left hand for most remaining activities. He had no history of familial sinistrality. The patient had drunk an average of 12 beers each weekend since his early twenties.

Seizure history

The patient had a febrile convulsion at age 9 months and was treated with phenobarbital (PB). He remained seizure-free until age 3–4 years, at which time he developed episodes of staring associated with arrest of activity and eye blinking. He was treated without success with all major antiepileptic drugs (AEDs).

Three different seizure types were documented. The first consisted of impaired consciousness, prominent picking automatism, and purposeless ambulation lasting 1–3 min. The second seizure type consisted of unresponsive staring and lip smacking without prominent automatism, generally lasting <1 min. The third type of spell began with an initial brief "wavy" feeling followed by generalized tonic-clonic seizures (GTC). Seizure frequency for the nongeneralized spells varied from 5 to 20 seizures a month, and generalization occurred 3–5 times a year.

EEG

Interictal EEGs showed either rare epileptiform discharges from the temporal regions without clear predominance or diffuse right-sided spike and wave activity. During his noninvasive evaluation at the Medical College of Georgia (MCG) (October 25–31, 1987), seven seizures were recorded. The seizures all appeared clinically similar and consisted of whistling at seizure onset, head and body turning to the left, and circular motions of the left arm.

Because the seizures were not localized with

scalp recordings, bilateral intracranial electrodes were implanted on April 7, 1988 to record stereotaxic EEG (SEEG), and the patient was monitored through April 12, 1988. A vertex trajectory was used, with six contacts per electrode separated by 1 cm and the most inferior contacts traversing the hippocampus (Flanigin and Smith, 1987). Interictal SEEGs showed bilaterally independent mesiotemporal epileptiform discharges including both spike and spike and slow wave discharges (R>L). Three clinically similar seizures were recorded, beginning with staring and whistling, followed by head and body turning to the left, followed in turn by circling left and right hand movements (L>R). SEEG during these seizures consisted of bilateral rhythmic beta recorded from contacts 1 and 2 in the mesiotemporal region. During the first of these seizures, bilateral rhythmic beta activity appeared simultaneously. With the other two seizures, the electrographic seizures developed in the right hippocampal region and spread 2–3 s later to left hippocampus.

A decision regarding operation was deferred, and the family pursued operation at a different institution. The patient underwent additional invasive monitoring at Baptist Memorial Hospital (BMH) (November 11–15, 1988) with eight subdural strips of electrodes. The electrodes were placed bilaterally in laterofrontal, mesiofrontal, laterotemporal, and mesiotemporal areas. Interictal recordings showed bilateral independent discharges, with greater discharges recorded from the right hemisphere. The discharges were recorded maximally from the mesio- and laterotemporal strips. Four seizures were recorded, two with clear onset from the right mesiotemporal region and two with unclear onset characterized by rhythmic discharges on the right with simultaneous decremental event on the left. One of these seizures spread to involve the left laterofrontal area partially.

Radiology

Preoperative MRIs using standard T_1-, proton density-, and T_2-weighted images showed no structural abnormality. Although one MRI suggested possible left temporal horn dilatation and mild deep atrophy, two repeat preoperative MRIs were interpreted as normal. Postoperative MRIs performed in June 1989 and in October 1992 reflected the partial right anterotemporal lobectomy with no other significant intracranial abnormalities (Fig. 1). Volume measurement from the MRI performed in October 1992 with the temporal lobe imaging protocol developed by Jack et al. (1989) indicated a left hippocampal volume of 1,456 mm^3.

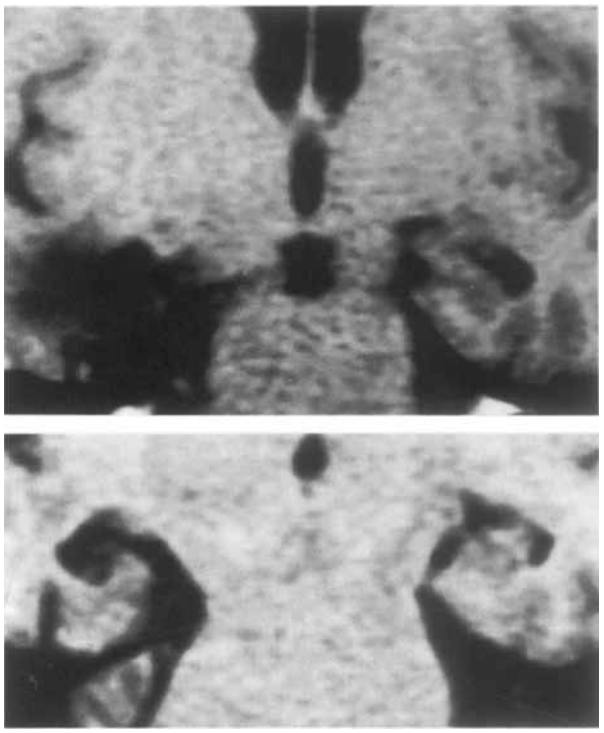

FIG. 1. Postoperative magnetic resonance imaging (MRI) scan of mesiotemporal lobe shows evidence of previous right sided temporal lobectomy and absence of gross lesion of contralateral hippocampus. With quantitative volume measurements, the patient showed left hippocampal volume of 1,456 mm^3. Images from adjacent MRI slices (5 mm thick).

Neuropsychological evaluation

Baseline neuropsychological results from tests administered at both institutions that have been repeated postoperatively are summarized in Table 1. Neuropsychological testing was performed on October 28, 1987 at MCG, and on October 3, 1988 at BMH. The patient had a low-average IQ, and no evidence obtained suggested dysfunction in a region other than the temporal lobe. Preoperative neuropsychological evaluation indicated generally impaired verbal memory in conjunction with normal nonverbal memory. On the Wechsler Memory Scale-Revised (WMS-R, Wechsler, 1987), he obtained a Verbal Memory Index of 74 and a Visual Memory Index of 104. His WMS-R General Memory Index was low average (84, and he was able to retain information adequately during a 30-min delay (Delay Index = 83).

Additional tests of material specific memory shown in Table 1 included the Selective Reminding test (Buschke and Fuld, 1974), California Verbal Learning Test (Delis et al., 1987), and Digit Supraspan Memory (Benton et al., 1983). Memory for visual-spatial material was tested with the Rey-Osterrieth Complex Figure delayed recall (Lezak, 1983), and Form Sequence Learning (Hamsher et al., 1983).

TABLE 1. *Pre- and postoperative neuropsychological test results*

Test	Preoperative	March 1989 (2 months postoperative)	June 1989 (5 months postoperative)	September 1989 (8 months postoperative)	March 1992 (38 months postoperative)
Full Scale IQ	87	—	80	—	81
Verbal IQ	79	—	75	—	74
Similarities	5	—	3	—	6
Comprehension	8	—	6	—	6
Information	5	—	5	—	5
Digit Span	9	—	10	—	7
Arithmetic	6	—	6	—	5
Performance IQ	96	—	94	—	90
Block Design	9	—	9	—	8
Picture Arrangement	9	—	8	—	8
Picture Completion	9	—	11	—	12
Digit Symbol	11	—	9	—	6
Language					
Controlled Oral Word Association	35	24	31	38	29
MAE Visual Naming	50/60	26/60	32/60	30/60	28/60
Token Test	41/44	42/44	42/44	—	43/44
Memory testing					
WMS-R General Memory Index	84	—	<50	58	59
WMS-R Verbal Memory Index	74	—	51	<50	55
Logical Memory I	15/50	—	4/50	3/50	5/50
Logical Memory II	3/50	—	0/50	0/50	0/50
WMS-R Visual Memory Index	104	—	77	85	93
Visual Reproduction I	38/41	—	34/41	35/41	38/41
Visual Reproduction II	34/41	—	5/41	6/41	16/41
WMS-R Delayed Recall Index	83	—	<50	<50	50
Selective Reminding CLTR	53/144	—	—	29/144	3/72
Complex Figure 30-s recall	17.5/36	—	—	4/36	4/36
Complex Figure 30-min recall	15/36	—	—	4/36	0.36
Serial Digit Learning	17/24	—	—	1/24	—
California Verbal Learning Test					
Total Trial 1–5	40/80	14/80	23/80	24/80	31/80
Trial 5	9/16	4/16	4/16	6/16	6/16
Short Delay Free Recall	4/16	3/16	4/16	0/16	2/16
Attention/concentration					
WMS-R Attention/Concentration	104	—	85	87	81
Trail Making A	22 s	27 s	17 s	—	—
Trail Making B	77 s	105 s	131 s	—	—
Visual spatial functions					
Complex Figure Copy	36/36	—	36/36	36/36	35/36
Judgment of Line Orientation	22/30	—	25/30	—	—
Facial Recognition	45/54	—	49/54	—	—
Visual Form Discrimination	32/32	—	30/32	—	—
Achievement testing					
WRAT-R Reading	63	51	53	—	—
WRAT-R Spelling	59	59	57	—	—
WRAT-R Arithmetic	76	82	76	—	—

Wada evaluation

The patient underwent Wada testing on October 30, 1987. Angiogram performed immediately before Wada assessment showed normal cerebral vasculature. After left hemisphere injection of amobarbital 100 mg, counting ability remained unaffected and a mildly agitated affect change was noted. Receptive language for complex comprehension was normal. Naming, repetition, and expressive speech were all normal. Recognition memory for eight objects presented soon after demonstration of hemiplegia and for five items presented several minutes after initiation of the procedure was tested after behavioral return to baseline. The patient was able to recognize 8 of 8 early objects and 3 of 5 late items (2 objects and a nursery rhyme recognized, 0 of 2 visual-spatial designs recognized).

Right hemisphere injection of 100 mg amobarbital produced speech arrest and inability to execute simple commands. Two minutes after injection, the patient continued to have severe comprehension impairment and repetition deficits, although simple naming was normal. After return to baseline, his recognition memory was severely impaired for early objects (0 of 8). However, he correctly recognized 3 of 5 late items (2 of 2 objects and rhyme recognition, 0 of 2 design recognition). Results of the Wada evaluation indicated right cerebral lan-

guage dominance, superior early object memory after left hemisphere injection (8 of 8 vs. 0 of 8), but identical late item scores bilaterally (3 of 5).

Postoperative course

A right anterotemporal lobectomy performed at BMH on January 13, 1989 involved a 4.5-cm resection of laterotemporal cortex with subpial aspiration of hippocampus to the posterior margin of the cerebral peduncle. The patient did not undergo intraoperative mapping of language function. Immediately after operation, a prominent recent memory deficit was observed in conjunction with expressive aphasia. The aphasia resolved to a moderate dysnomia by the time of hospital discharge.

There has been no significant reduction in seizure frequency despite treatment with multiple AEDs, including experimental agents, without satisfactory reduction in seizure frequency. At the time of this writing, the patient is being treated with carbamazepine, topiramate, and PB. In the past 3 months, he has experienced 21 complex partial seizures.

Postoperative neuropsychological assessment

Formal neuropsychological assessment initially conducted at BMH 2 months postoperatively documented a substantial decrease in recent memory function. Both WMS-R General Memory and Delayed Recall Indexes decreased to <50. There was a greater negative effect of neurosurgery on Logical Memory than on Visual Reproduction. However, delayed Visual Reproduction, which assesses the ability to retain simple visual during a 30-min delay, decreased significantly. Although prose passage recall was impaired preoperatively, postoperative performance was even poorer. Some of these scores have improved slightly with time, although they continue to be impaired and at a level significantly below the preoperative performances. The patient was also variably oriented to time, misstating either the month, the day of the month, or year. At his most recent follow-up assessment, he was unable to state his age correctly.

Postoperative decline was observed on several language tasks. Visual naming (Benton and Hamsher, 1989) in particular decreased significantly, with no improvement noted with longer follow-up. This was accompanied by a decrease in reading ability (WRAT-R; Jastak and Wilkerson, 1984).

The major memory impairments were obvious to his family. He would go to dinner and a movie with his brother and have no recollection of these events when questioned the next day. Similarly, he would have no recall of events that had occurred earlier in the day (e.g., going to church, shopping, or walking with his family). For activities, the patient enjoyed solving magazine word puzzles, but postoperatively he would solve the same puzzle from different copies of the same puzzle book without realizing that he had previously worked on the identical puzzle.

DISCUSSION

The performance of our patient confirms the risk to recent memory function after temporal lobectomy. Amnesia has persisted for 3 years, although his psychometric memory performances have improved slightly. This slight improvement on measures of neuropsychological memory performance suggests that the magnitude of memory impairment is not as severe as the amnesia experienced by H.M.

Wada memory testing

Milner et al. (1962) first included memory testing during amobarbital anesthesia to predict the presence of contralateral mesiotemporal lobe dysfunction. By producing a state of temporary reversible dysfunction ipsilateral to the side of proposed operation, the potential effects of temporal lobectomy on memory function could be modeled prior to surgical resection. Since then, Wada memory testing has been widely used to identify patients believed to be at risk for postoperative amnesia, although not without controversy (Loring et al., 1992).

Our patient displayed asymmetry in early object memory on Wada testing, but no asymmetry was evident in the patient in his performance on the later Wada items. Although early object Wada memory is superior to the later Wada items in identification of lateralized memory impairment (Loring et al., 1994), early object memory performance alone may falsely identify amnesia risk (Loring et al., 1990*a*). Furthermore, although both the early and later Wada items are sensitive statistically to contralateral dysfunction over a patient series, prediction of an individual is variable and may be altered by the stimuli (early vs. late) or criteria used.

The early object Wada memory asymmetry of the present patient, if considered in isolation, suggests that the risk of amnesia was predicted accurately by the amobarbital procedure. Because correct prediction not only involves the likelihood of correct identification of risk but also correct identification of risk absence, it is important to identify patients with similar Wada memory performance who did not develop amnesia. Three patients in the MCG series have undergone operation (L = 2, R = 1) after Wada memory testing using amobarbital dosages comparable to those used in the present patient who exhibited pronounced performance asymmetries

(i.e., ≥4) in the wrong direction and who did not become amnestic. Thus, Wada memory results should not be interpreted in isolation and out of the context of other clinically relevant information.

MRI

The original MRI scans of our patient were obtained before implementation of the specialized protocol designed to image the hippocampus. However, visual inspection of postoperative MRI made with hippocampal imaging protocol (Jack et al., 1989) did not show clear atrophy of hippocampus contralateral to the operation; depth electrodes tracks were not visible in the post SEEG MRI. With quantitative volume measures, the patient had a left hippocampal volume of 1,456 mm^3. This value is substantially lower than the average left hippocampal volume in our right temporal lobectomy patients (mean 2,055 mm^3, SD 412) and of right hippocampal volume in our left temporal patients (mean 2,535 mm^3, SD 317). Jack et al. (1989) reported normative values of 2,800 mm^3 (SD 400) for left hippocampus and of 2,400 mm^3 (SD = 400) for right hippocampus [Jack et al. (1989) reported SEM rather than SD; we have converted SEM to SD to use a common metric]. Because a left–right difference exists in hippocampal volumes of nonepilepsy controls we cannot know whether the appropriate comparison for this patient should be the left or right temporal lobe patients, given his reversed language laterality. However, regardless of the comparison group, decreased hippocampal volume of the hippocampus contralateral to operation is suggested. Relative hippocampal asymmetry could not be calculated because of the previous temporal lobectomy.

Neuropsychological testing

Patients believed to be at risk for postoperative amnesia will have significant impairment of both verbal and nonverbal recent memory functions, reflecting bilateral mesiotemporal lobe involvement. Our patient had consistent asymmetry of recent memory tasks, with impaired verbal learning and memory but generally normal visual memory. Because of the relative impairment of the language-dominant hemisphere's temporal lobe function (right) in conjunction with relative sparing of the nonlanguage dominant temporal lobe function (left), no amnesia risk was suggested since the temporal lobectomy was performed on the side associated with the material-specific memory impairment (i.e., right).

One conclusion that we derive in this case concerns the potential dissociation of material-specific memory functions from cerebral language dominance. We have also observed a dissociation between language representation and material-specific memory functions in a patient with bilateral language representation (R>L) who underwent right temporal lobectomy (Loring et al., 1990*b*). Despite the verification of right cerebral language representation by multiple techniques (Wada testing, cortical stimulation mapping at operation, postoperative aphasia), neuropsychological testing showed a material-specific memory impairment for visuospatial material, a pattern associated with right temporal lobe seizure in left language-dominant patients. Although this patient displayed some left cerebral language representation, Wada testing in our amnestic patient showed no evidence of any left cerebral language. Thus, when standard cerebral language representation cannot be established, a mirror representation of material-specific memory function cannot be assumed and the confidence with which inferences are made based on neuropsychological memory testing should be lessened.

Our patient illustrates postoperative amnesia after right temporal lobectomy in the context of right cerebral language dominance. The pattern of preoperative neuropsychological deficits indicated impaired verbal memory in conjunction with normal nonverbal memory. Wada memory performance was asymmetrical for items presented soon after amobarbital injection, with no memory asymmetry for items presented later in the evaluation. Although repeated MRI scans showed no structural lesions, quantitative hippocampal volume derived from the patient's operative MRI suggested reduced hippocampal volume contralateral to the operation. These results confirm the risk to recent memory function after unilateral temporal lobectomy and indicate that baseline neuropsychological testing may falsely lateralize material-specific memory functions in patients with right cerebral language dominance.

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