Verbal memory deficits after left temporal lobectomy for epilepsy

Mechanism and intraoperative prediction

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Verbal memory deficits remain a major complication of dominant hemisphere temporal lobectomy for epilepsy. The extent of this deficit was assessed preoperatively and 1 month and 1 year postoperatively with the Wechsler Verbal Memory Scale (WMSV) in 14 adults undergoing left temporal lobectomy. Intraoperative localization of language and verbal memory was also performed by electrical stimulation mapping. The WMSV score decreased an average of 22% at 1 month (13 cases), and 11% at 1 year (10 cases), even though in the majority of cases the medial extent of the resections had been significantly modified as a result of preoperative memory changes in response to intracarotid amobarbital perfusion testing. Memory decline was greater in patients who were not seizure-free, and correlated with the lateral (but not the medial) extent of the resection. The memory deficit could be predicted intraoperatively with 80% accuracy from the relationship of the resection to sites identified by electrical stimulation mapping as essential to naming or input or storage aspects of memory. This technique was applied prospectively in two additional cases with left temporal epileptic foci and complete verbal memory loss with left hemisphere amobarbital inactivation. These resections were tailored to avoid the essential naming and memory sites; the WMSV score increased 1 month postoperatively in both cases. This study identifies a lateral cortical component for verbal memory. Sites essential for that component can be localized intraoperatively with stimulation mapping; when they are spared in a resection, verbal memory deficit following dominant hemisphere temporal lobectomy can be prevented even in high-risk cases.

KEY WORDS: memory, temporal lobe, epilepsy, electrical stimulation

A relationship between human temporal lobe resections and memory deficits was established nearly three decades ago by Penfield, Scoville, and Milner. Their studies indicated that the deficit involved recent post-distractional short-term memory, rather than immediate undistracted memory or long-term memory (beyond a period of retrograde amnesia of as much as 3 years). A severe memory deficit resulted after bilateral temporal lobe lesions; unilateral temporal lobe lesions produced more subtle deficits that differed between the right and left sides. The deficits following left-sided lesions involved short-term memory for verbal material. Subsequent investigation suggested that it was specifically hippocampal damage that accounted for this memory loss. The patients reported by Penfield had surgical temporal lobe removal undertaken as treatment for medically intractable epilepsy. The Montreal group suggested that patients likely to have clinically significant memory deficits after that operation could be identified preoperatively by the presence of verbal memory deficits when the side of the proposed resection was inactivated by intracarotid amobarbital perfusion. Based on these findings and following the suggestion of others, the technique of temporal lobectomy for epilepsy at the University of Washington was modified to vary the medial extent of the resection depending on the degree of verbal memory deficit on the preoperative intracarotid amobarbital study. The medial temporal resection was confined to the amygdala and uncus, with little or no hippocampal removal, when intracarotid amobarbital perfusion on the side of the resection was associated with a large verbal memory deficit.

Nevertheless, verbal memory deficits remain a major complication of anterior temporal lobectomy of the dominant hemisphere. Substantial verbal memory deficits were recently reported after a 1-year postoperative neuropsychological assessment of patients who had undergone a left temporal lobectomy for epilepsy at two other centers. A recent review of "treatable epilep-
sies" suggested that temporal lobectomy should not be performed when preoperative intracarotid amobarbital assessment showed that the remaining temporal lobe could not support memory, and that left temporal lobectomy was contraindicated in patients whose livelihood depended on a facile memory. Thus, the present study was undertaken to: 1) determine the extent of any verbal memory deficit after a modified left dominant hemisphere temporal lobectomy; 2) identify characteristics of the patients and resections that correlate with those deficits; and 3) determine if the deficits can be predicted by changes on intraoperative electrical stimulation mapping of the lateral temporal cortex during language or memory tasks.

Clinical Material and Methods

Subjects

As of July, 1983, 14 adults who had undergone left temporal lobectomy for medically intractable epilepsy at the University of Washington also had: 1) pre- and postoperative assessment using the Neuropsychological Battery for Epilepsy; 2) left hemisphere speech based on intracarotid amobarbital assessment (10 cases) or clinical evidence such as an aphasia with seizures localized by electroencephalography (EEG) to the left hemisphere; and 3) craniotomy under local anesthesia with intraoperative electrical stimulation mapping during a test of short-term memory. These 14 patients formed the basis of this study. The age range was 17 to 49 years (mean 28.9 years), and 10 were female. Preoperative Wechsler Adult Intelligence Scale (WAIS) Full Scale Intelligence Quotient (FSIQ) scores ranged from 76 to 115 (mean 98.4). Seizure onset occurred at a mean age of 9.75 years (range 1 to 19 years). Memory testing during intracarotid amobarbital perfusion was obtained in 10 of these cases using a verbal memory measure adapted by one of us (C.B.D.) to this situation. This task consists of consecutive trials each including the naming of an object, followed by reading a nine-word sentence from a card, and then, at a verbal cue, recalling aloud the name of the object presented in this trial. Performance on this test during intracarotid amobarbital perfusion was videotaped for later scoring. The mean baseline error rate on the recall portion of this test was 5.1% (range 0% to 18%). After left hemisphere amobarbital inactivation, recall errors increased in all cases to a mean of 56.5% (range 23% to 100%) in the 5 minutes following recovery from any speech blockage.

Pre- and Postoperative Testing

Postoperative assessment at 1 month was available for 13 cases, and at 1 year for 10 patients, one of whom did not have a 1-month assessment. The memory measurement system was derived from the pre- and postoperative testing Form 1 of the Logical Memory and Visual Reproduction portions of the Wechsler Memory Scale (Wechsler Verbal Memory Scale: WMSV, and Wechsler Nonverbal Memory Scale: WMSN, respectively). The WMSV represents the number of essential elements recalled from two orally presented prose passages with the recall delayed after the completion of the passages only long enough to give a final one-sentence instruction. The WMSN represents the number of features recalled following 10-second exposures to four drawings and following a final instruction. The change in these scores postoperatively was evaluated statistically with the Wilcoxon matched-pairs test. Changes in the WMSV scores at 1 month and 1 year postoperatively were then separately computed as a percent of the preoperative performance for each case. Cases were then ranked from greatest to least deficit at one follow-up assessment and correlated with a number of patient and resection characteristics using the Spearman rank order correlation coefficient (Rs). An assessment of language was also obtained at each testing, in the form of the number of receptive and expressive errors on Reitan's modification of the Halstead-Wepman Aphasia Screening test that is also included in the Neuropsychological Battery for Epilepsy.

Intraoperative Testing

Intraoperative mapping of language and verbal memory by electrical stimulation involved previously described techniques. The test used with stimulation mapping consists of multiple trials presented by slides. Each trial is a sequence of three slides. The first is a line drawing of an object with a common name. In the 4-second period during which this is shown, the patient names the object aloud. The second slide contains an 8- to 10-word sentence that is read aloud during the 8 seconds that this slide is shown. The third slide, shown for 4 seconds, has the word "recall" on it. This acts as a cue for retrieval of the object name originally presented at that trial. Electrical stimulation occurs during showing of the first slide in some trials, and the second or the third in others, all pseudo-randomly interspersed with trials without stimulation that serve as measures of control performance. A predetermined sequence is used, so that each cortical site sampled is stimulated three times under each test condition, but never consecutively.

A mean of 8.4 sites in the lateral temporal, inferior parietal, and posterior frontal cortex were stimulated in the 14 cases of this study (range four to 13 sites). The location of each site was identified by a sterile numbered ticket and recorded photographically. Stimulation was by 60-Hz biphasic square-wave pulses, 2.5 msec total duration, delivered from a constant-current stimulator in a bipolar manner through electrodes separated by 5 mm, in trains lasting for the duration of each slide. Stimulation was at the largest current that did not evoke afterdischarge in the sampled area of cortex. Mean stimulating current in these cases was 4.8 mA, between pulse peaks (range 2.5 to 8 mA).

Performance on the test was separately analyzed for each condition, using the single-sample binomial test.
Left temporal lobectomy and memory deficits to compare performance on the multiple samples with stimulation at a particular cortical site to performance on the large number of control trials. Control trial error rates for the 14 cases in this study were 2.8% on object naming (range 0% to 22%) and 12.75% on memory retrieval (range 0% to 35%). A site was related to a language or memory function when errors in that function during stimulation at that site exceeded control performance at p < 0.05. For each site, the effect of stimulation on object naming was first determined. Any trials with errors in object naming were then excluded from the analysis, and the effects on memory retrieval separately determined for trials with stimulation at the time of input to memory (object naming), during the time the memory was stored (over the standard distraction provided by sentence reading), or at the time of retrieval. Patients were then divided into those with and those without sites related to naming or memory with stimulation during input, storage, or retrieval that were in or within 2 cm of the resection margin (as determined from the intraoperative photographs), and the severity of the postoperative WMSV deficit in these two series of cases was determined.

Operative Technique
The technique of temporal lobectomy used in these cases was based on that of Penfield and Jasper, though our modifications have been described previously. These operations were conducted under local anesthesia of either 0.5% lidocaine alone or with 0.25% bupivacaine, in most cases supplemented by 1 cc of intravenous Innovar (fentanyl and droperidol) just before the operation and about 3 to 4 hours before electrical stimulation mapping. Electrocochleography (ECOG) preceded mapping, and the extent of the resection was tailored to the extent of the ECOG interictal epileptic focus and the location of sites considered important for language, based on repeated errors in object naming during stimulation. The resection involved subpial suction techniques, and included the uncus and amygdala tailored to the extent of the ECoG interictal epileptic operation and about 3 to 4 hours before electrical stimulation mapping. Electrocorticography (ECoG) preceded mapping, and the extent of the resection was tailored to the extent of the ECOG interictal epileptic focus and the location of sites considered important for language, based on repeated errors in object naming during stimulation. The resection involved subpial suction techniques, and included the uncus and amygdala in all cases, but the extent of hippocamal removal was varied, depending on the severity of the memory deficit with preoperative left intracarotid amobarbital perfusion. In all cases, two standard measurements of the resection were obtained: from the tip of the middle fossa to the resection margin in the middle temporal gyrus, and to the resection margin on the medial temporal lobe.

Results
Postoperative Memory Change
Table 1 indicates the WMSV and WMSN values before and 1 month after left temporal lobectomy in the 13 patients with such assessment. The verbal memory score was significantly reduced postoperatively, representing a 22% mean reduction (range 82% decrease to 0% increase); eight of the 13 patients showed a reduction, two no change, and three an improvement. One year after operation the verbal memory deficit was somewhat reduced, to a WMSV score of 17.8 (range 4 to 26), compared to a preoperative value of 21.8 (range 12 to 37) in the 10 patients with 1-year evaluations. This change, representing an 11.4% mean reduction (range 66% increase to 80% decrease) is at the margin of statistical significance (p = 0.065). The verbal memory deficit at 1 year was highly correlated with the 1-month score in the nine cases with testing at both time intervals (Table 2), five cases showing a greater deficit at the 1-year than at the 1-month testing. By contrast, the nonverbal memory scores increased slightly at 1 month and 1 year after operation, although not to a statistically significant extent (mean WMSN score at 1 month was 9.7 (range 3 to 13), and at 1 year was 9.2 (range 4 to 12), compared to the preoperative WMSN value of 8.6 (range 3 to 13) in the same 10 cases). Thus, in the majority of our patients, after left temporal lobectomy there was a verbal memory deficit that was not reflected in nonverbal memory, and did not clear completely by 1 year.

Correlation with Patient and Resection Characteristics
The verbal memory deficit was smaller in the eight patients who had been seizure-free between operation.
Evaluating the temporal lobe resection log, we observe a significant correlation between the WMSV score and the lateral extent of the resection. Large lateral temporal resections are associated with large postoperative memory deficits. This correlation is independent of seizure control, as seen in cases where seizure-free outcomes were noted from operation to testing (WMSV correlation with medial resection 0.73, with lateral resection 0.67, and with lateral extent the correlation is 0.73 (p < 0.05) in those cases alone). As expected, the medial, but not the lateral, extent of the resection had been altered depending on the severity of the memory deficit on preoperative left intracarotid amobarbital perfusion. A large memory deficit on left intracarotid amobarbital perfusion has a rank order correlation with the medial extent of the resection (ranked large to small) of 0.67 (p < 0.05) and with the lateral extent the correlation is 0.41 (p > 0.10). Thus, modifying only the medial extent of the resection has not eliminated the verbal memory deficit after left temporal lobectomy. The significant remaining deficit is not predicted by preoperative memory performance or memory deficits within the temporal lobe. Large lateral temporal lobectomy-related memory impairment is related to the extent of damage to medial temporal structures. Rather, it seems to reflect damage to a lateral cortical memory system.

Additional evidence on the importance of lateral temporal structures in this memory deficit comes from the significant correlation between that deficit and performance on an aphasia battery (Table 2). The language deficit in these cases was a mild one, clinically noticeable in only two cases who were seizure-free from operation to testing (WMSV correlation with medial resection 0.07, with lateral resection 0.73 (p < 0.05) in those cases alone). As expected, the medial, but not the lateral, extent of the resection had been altered depending on the severity of the memory deficit on preoperative left intracarotid amobarbital perfusion. A large memory deficit on left intracarotid amobarbital perfusion has a rank order correlation with the medial extent of the resection (ranked large to small) of 0.67 (p < 0.05) and with the lateral extent the correlation is 0.41 (p > 0.10). Thus, modifying only the medial extent of the resection has not eliminated the verbal memory deficit after left temporal lobectomy. The significant remaining deficit is not predicted by preoperative memory performance or memory deficits within the temporal lobe. Large lateral temporal lobectomy-related memory impairment is related to the extent of damage to medial temporal structures. Rather, it seems to reflect damage to a lateral cortical memory system.

Intraoperative Prediction of Memory Deficit by Electrical Stimulation Mapping

Figure 1 indicates the proportion of sites in different zones of the lateral temporal, inferior frontal, and parietal lobes related to naming or memory by electrical stimulation mapping in these 14 cases. At least one temporoparietal site with naming and/or memory changes was present in each case: 11 patients had such sites related to naming errors and 13 to memory changes. Within the temporoparietal cortex, the largest proportion of naming sites was in the posterior superior temporal gyrus zone, while the largest proportion of memory sites were in the middle superior temporal and inferior parietal zones. Memory sites extend into the anterior middle temporal lobe zones as well, and thus are likely to be included in an anterior temporal lobectomy. However, there is considerable variability in the exact location of these memory sites in different patients, analogous to the variability in the lateral temporal lobe. Stimulation sites in individual cases were aligned in a graph, as shown in Figure 1, indicating the proportion of cases with errors after stimulation at sites within that zone. The inner circle graph indicates the proportion of cases with naming errors evoked at sites within that zone. The outer circle graph shows the proportion of cases with short-term memory errors evoked at sites within that zone. The filled outer portion of these graphs indicates the proportion of cases with errors after stimulation during input or storage aspects of memory, the dotted outer portion cases with errors after stimulation at the time of retrieval. The fine dashed lines in the anterior temporal lobe delineate the lateral extent of the resections in the two additional prospectively studied cases. Below is the mean and range of the extent of the anterior temporal resection of the 14 retrospective cases, measured at the level of the middle temporal gyrus.
29 memory sites were related only to input or storage, 12 were related only to retrieval, and only two were common to both.

Cases were divided into those with or without sites related to language or memory within 2 cm of the margin of, or actually in, the anterior temporal resection (the "resection zone"). Most cases of postoperative memory deficits occurred when sites related to naming or memory after stimulation during input or storage (but not retrieval), were within the resection zone (l-month postoperative WMSV performance in the eight cases with such sites differed from that in the five cases without: \( p = 0.004 \), Mann-Whitney U-test). Seven of the eight patients with such sites within the resection zone had memory deficits at 1 month and four of the five patients without such sites did not (the probability of seeing such a separation on a chance basis is 0.05, Fisher's exact test). This type of error on stimulation mapping was equally effective in identifying the postoperative memory deficit at 1 year. The presence of such sites in the resection zone identified five of the six cases with WMSV deficits at 1-year postoperatively; the absence of such errors identified three of the four cases without such a deficit. As with all stimulation mapping, significance should be attached to sites without any stimulation-evoked changes only when such changes are present elsewhere, as they were in each of our cases. Thus, the postoperative memory deficit was predicted with about 80% accuracy by mapping the effects of intraoperative electrical stimulation on language and memory.

**Prospective Application of Stimulation Mapping to Avoid Memory Deficits**

After completion of the previous study, two candidates for left temporal lobectomy were encountered who provided a preliminary test of whether a postoperative memory deficit can be avoided by tailoring a resection around lateral temporal sites related either to naming, or to the input and storage phases of memory. Both patients, right-handed men aged 33 and 31 years, had a left temporal origin for their seizures established by scalp EEG monitoring of the onset of spontaneous seizures. Both also had EEG evidence of right temporal interictal abnormalities. The first patient first suffered seizures at 31 years of age, following recovery from herpes simplex encephalitis. The second patient had seizures dating from the age of 10 months, following possible encephalitis. Preoperative WAIS-Revised FSIQ scores were 107 and 78, respectively. Seizures in both patients were intractable to antiepileptic drugs. Preoperatively, one patient was having an average of 2.5 partial complex seizures weekly, the other had more than one weekly. Both had left hemisphere dominance for language, based on intracarotid amobarbital perfusion testing, and both had 100% memory error rates after left carotid injection. Thus, neither patient would be considered for temporal lobectomy under the criteria presently in use at some other centers. At craniotomy under local anesthesia, sites related by electrical stimulation mapping to naming or to the input and storage aspects of memory were identified in the lateral temporal cortex in each case, and the resection was tailored to avoid these sites by more than 2 cm. Figure 1 shows the lateral extent of these two resections. Medially, the amygdala and uncus (but not the hippocampus) were resected in both cases. However, these were not just small resections, for in one case the inferior temporal gyrus resection extended back 85 mm. Rather, these resections were shaped to remove as much of the ECoG-identified epileptic focus as possible while providing an appropriate margin around the sites related to the naming and input and storage aspects of memory identified by stimulation mapping. One month after operation (when the verbal memory deficit was greatest in our other cases), the WMSV score of verbal memory had increased in both patients, in one from a preoperative value of 16 to 18 and in the other from 7 to 17. (The WMSN score also increased in both, from 10 to 11 in one and 7 to 10 in the other). Thus, the verbal memory deficit after dominant temporal lobectomy can apparently be avoided, even in such high-risk cases, by tailoring the resection to avoid hippocampus and the lateral temporal structures identified by stimulation mapping as essential to language or memory input or storage.

What is presently unknown is whether this procedure will compromise the probability of seizure control in such cases, for these two patients have been followed for only a few months as of this writing. In both cases some lateral temporal cortex demonstrating epileptiform spikes on ECoG had to be left intact, in order to avoid the sites considered essential to memory. Nevertheless, both of these patients had reduction in seizure frequency. Thus, it may be possible to control seizures and avoid the verbal memory deficit with this approach.

**Discussion**

The findings of this study indicate that some reinterpretation of the generally accepted model of verbal memory mechanisms in dominant temporal lobe is in order. This reinterpretation has consequences for modifications of temporal lobectomy for epilepsy directed at reducing postoperative memory deficits. Modification of the dominant temporal lobe resection to spare the hippocampus in our patients with major memory deficits on preoperative intracarotid amobarbital perfusion of that hemisphere has not eliminated the postoperative memory deficit. From our own data, we cannot determine whether such a modification has reduced the deficit. However, there is some indication that it has. Memory assessments preoperatively and 1 year after surgery, with the same verbal memory measure we used (the WMSV), were recently reported from two centers where the medial temporal resection always includes anterior hippocampus, even in patients with poor preoperative intracarotid amobarbital memory testing (although patients with 100% memory errors...
with amobarbital perfusion of the side of the proposed resection are not considered for operation at those centers.\textsuperscript{9,22} In both of these reports, patients who were seizure-free between operation and testing had a smaller memory deficit than those not seizure-free, and this was also true of our patients. In both reports there was some improvement in the WMSN measure of spatial memory after left temporal resections and this too was seen in our patients. However, the seizure-free patients showed a mean 18% reduction in the WMSV score at 1 year in one series where the hippocampus was included in all resections,\textsuperscript{12} and a 30% reduction in the other.\textsuperscript{9} In our series, the medial extent of the resection varied depending in part on our quantified intracarotid sodium amobarbital perfusion procedure, and the resulting deficit was 11% at 1 year. Of course, these are not matched series, so the difference only suggests that modifying the medial extent of the temporal resection may be associated with some reduction in the postoperative memory deficit.

Despite the findings just noted, there is a verbal memory deficit that does not seem to be related to medial temporal structures. Rather, that deficit correlates with the extent of lateral cortical excision, and can be predicted by the effects of lateral cortical stimulation during language and memory tasks. There is additional evidence for the lateral temporal cortex having a role in verbal memory mechanisms. Persisting verbal memory deficits were reported after temporal lobe strokes that left the patients transiently aphasic.\textsuperscript{1,3} "Conduction aphasia," often due to a posterior temporal lobe lesion, has been modeled as primarily a short-term memory deficit.\textsuperscript{24} Extension of a left temporal lobe resection for epilepsy posteriorly into the superior temporal gyrus was associated with the same increase in verbal memory deficit as extension of the resection posteriorly into the hippocampus in an early study by Milner.\textsuperscript{7} Excision of medial but not lateral temporal structures in patients with temporal lobe epileptic foci has not been associated with a memory deficit in several cases.\textsuperscript{28} Verbal memory changes with left lateral temporal electrical stimulation mapping have also been reported by Fedio and Van Buren.\textsuperscript{9} Thus, the model of verbal memory mechanisms in the human brain must be revised to include not only the hippocampus and structures around the third ventricle,\textsuperscript{14} but also the lateral cortex of the dominant hemisphere. It is damage to this lateral temporal cortical component that accounted for the residual memory deficit after left temporal lobectomy in our patients.

The pattern of language and memory changes evoked by stimulation in the present cases, and in other cases previously reported by us,\textsuperscript{10,11,13} provides insight into the organization of this lateral cortical verbal memory system in the dominant hemisphere. Memory and language generally are localized to separate although adjacent sites, with sites related to memory surrounding those related to language not only posteriorly, as demonstrated in the present cases, but also frontally.\textsuperscript{10,11,13} Temporal and parietal memory sites are most often related to the input and storage aspects of memory. Those sites may be the location of the neural events representing the actual storage of short-term verbal memory. We have recently identified an evoked potential pattern on the EEGG recorded from those sites that may reflect these neural events, because those evoked potentials seem to be anatomically specific to such memory sites, and there, behaviorally specific to tasks requiring entry of information into memory.\textsuperscript{16} On the other hand, frontal memory sites are more often related to retrieval by stimulation mapping; thus, the cortical verbal memory mechanisms seem to be organized with temporoparietal storage and frontal retrieval.

Our finding that most of the patients who will have memory deficits after temporal resection can be identified by intraoperative electrical stimulation mapping is further indication that lesions and stimulation identify the same brain areas as essential to a particular function. This provides confirmation for modeling stimulation effects in association cortex as lesions localized in space and time.\textsuperscript{10} The value of "negative" findings with stimulation mapping was also demonstrated as long as sites with "positive" effects are present elsewhere in the patient and stimulation uses currents just below the threshold for afterdischarge. We have reported similar relationships between stimulation-evoked changes in naming and the effects of temporal resections on postoperative language function: the presence of positive naming sites, as determined by stimulation, in or on the margin of the resection is associated with a postoperative aphasia, and the absence of such sites (as long as they are present elsewhere) with the absence of a postoperative language defect.\textsuperscript{11} The considerable variability in the individual location of sites essential to language or memory (Fig. 1)\textsuperscript{12} indicates that they cannot be identified solely on anatomic criteria as would be necessary for operations conducted under general anesthesia. Rather, they must be identified in each case individually by a technique such as stimulation mapping in awake patients under local anesthesia.

The ability to identify patients who will have postoperative memory deficits by the relationship of stimulation mapping changes to the resection, and our ability to avoid such deficits in the two prospectively evaluated high-risk cases by tailoring the resection to avoid such sites indicates a technique for reducing verbal memory deficits after left temporal resections. That technique seems to be effective even when preoperative intracarotid amobarbital perfusion suggests that the temporal lobe contralateral to the resection cannot support memory by itself. However, there are several aspects of our approach to avoiding memory deficits with temporal lobectomy that require further evaluation. Stimulation mapping of memory is quite time-consuming, and requires careful attention to distinguish between random errors (there is often a baseline error rate) and significant, repeatable errors. The use of multiple samples of stimulation effect at each site under each test condition is essential, as is the use of interspersed control trials to assess that baseline error
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rate. Occasional patients will make so many errors on those control trials that even errors on every one of three or four samples at a site still might be a chance occurrence. One must then obtain more samples of stimulation effect at sites considered crucial to planning the resection.

Whether resections tailored around areas important to memory, both medially (sparring the hippocampus) and laterally (sparring naming and memory input and storage sites), will have a reasonable probability of providing long-term seizure control also must be determined. Every limitation on resection of the focus probably reduces the chances of seizure control to some degree below that we have reported with unmodified temporal resection, that is, two-thirds of patients seizure-free and three-quarters of patients with a very substantial improvement or seizure-free 1 year or more postoperatively. Thus, tailoring resections around memory sites might not be used in every case. But if the reduction in probability of long-term seizure control is not too great, with the use of our technique of stimulation mapping of language and memory combined with such tailoring of the resection, temporal lobectomy becomes a therapeutic option for patients with medically intractable epilepsy and temporal lobe foci who otherwise would not be candidates for surgery. Such cases include those who cannot afford any verbal memory loss, or where the other temporal lobe cannot support memory.

Acknowledgments

Drs. I. Fried, C. Mateer, S. Polen, T. Sanquist, and H. Whitaker assisted with portions of this study; some patients were under the care of Drs. L. Ojemann, A. Ward, A. Wilensky, and A. Wyler.

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Manuscript received March 21, 1984. Accepted in final form July 13, 1984.

This work was supported by NIH Grants NS17111 and NS17277.

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