Results of anterior temporal lobectomy that spares the amygdala in patients with complex partial seizures

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During the decade from December, 1980, to December, 1990, 70 patients with complex partial seizures at our institution underwent an anterior temporal lobectomy that spared the amygdala. Although there was a growing consensus that lobectomies that included the hippocampus and amygdala resulted in better surgical outcomes, there was no evidence to indicate that removal of one or the other or both was necessary to achieve a good result. We were also influenced by the relative paucity of pathology in the amygdala compared to that seen in the hippocampus. We present evidence that the amygdala need not be included in the temporal lobe resection to obtain a good result.

Clinical Material and Methods

Patient Population

A total of 70 patients whose age at the time of surgery ranged from 9 to 60 years (mean 26 years) underwent an anterior temporal lobectomy that spared the amygdala. They have been followed for intervals ranging from 1.5 to 9 years (mean 4.9 years). Except for seven cases, these patients did not have a history of or an existing encephalopathy. In 67 (96%) of the 70 patients, the initial seizure occurred in infancy or childhood; in 51 (73%), the first seizure was a generalized tonic-clonic convulsion, frequently associated with fever. Additional generalized seizures occurred in some cases. Complex partial seizures sometimes began after a latent seizure-free period of months to years. Of the remaining patients, 15 had complex partial seizures from the start of their illness and in four the initial seizure pattern was unknown.

Imaging Procedures

All patients studied prior to November, 1984, underwent preoperative imaging with high-resolution computed tomography (CT). Those treated subsequent to 1984 were studied by magnetic resonance (MR) imaging. The MR image or CT scan was abnormal in 16 patients. Seven cases showed asymmetry of the temporal horns, either anteriorly in the region of the anterior hippocampus or affecting the entire temporal horn, especially posteriorly involving the trigone. There was a small focus of increased signal involving the uncus in one case and the hippocampus in another. In seven cases, the abnormal finding was considered to be coincidental; for example, the pathology was a venous angioma in the hemisphere of the nonaffected tem-
temporal lobe, or a finding consistent with demyelination in the region of the trigone bilaterally, or cerebellar atrophy.

**Positron Emission Tomography**

Positron emission tomography (PET) scans using 15O to measure cerebral blood flow were obtained in 29 patients (P Fox, et al., unpublished data). In 17 patients, the PET scan correctly identified the symptomatic temporal lobe as judged by the results of epidural electrocorticographic (ECoG) recording, pathological study, and clinical outcome. However, in 12 patients who also had a good outcome, the PET scan showed no asymmetry in blood flow between the two temporal lobes.

**Electroencephalographic Recordings**

All 70 patients had several sleep and waking electroencephalographic (EEG) recordings obtained prior to surgery; 35 were also studied with nasopharyngeal electrodes. Combined scalp EEG and videotape monitoring was used infrequently. All patients underwent combined videotape and epidural ECoG monitoring extraoperatively. In 43 of the 70 patients, the EEG and epidural ECoG studies identified the same temporal lobe as giving rise to the seizures. Of the remaining 27 patients, the EEG signal was nonlateralized in 26 and implicated the side opposite to that identified by the epidural ECoG monitoring in five.

Although MR images, scalp EEG studies, and PET scans were all considered in the evaluation, the decision for anterior temporal lobectomy was determined primarily by the epidural ECoG results, recorded both interictally and incident to clinical seizures. Bilateral carotid angiography, Wada tests, and a test for recent recall were performed on every patient. Neuropsychological testing to identify the symptomatic temporal lobe was not employed.

**Operative Procedure**

The anterior temporal lobectomy procedure is carried out with the patient under general anesthesia. The middle temporal gyrus is incised transversely 4 to 4.5 cm posterior to the anterior temporal tip (the incision is kept closer to 4 cm on the side concerned with language function). The incision is extended obliquely in a posterior direction through the inferior temporal and fusiform gyrus and the lateral aspect of the parahippocampal gyrus. The incision through these gyr is extended to the white matter. Next, the bottom of the sulcus between the superior and middle temporal gyri is exposed and divided posteriorly back to the transverse incision in the middle temporal gyrus. Anteriorly, the incision is carried beyond the sulcus through the anterior tip of the temporal lobe. The white matter is then undercut, leaving the residual parahippocampal gyrus and its arachnoid holding the otherwise mobilized lateral temporal cortex. This attachment is divided longitudinally and the lateral temporal cortex and associated white matter are delivered en bloc.

Next, the white matter is incised to reveal the anterior temporal horn, and the opening is extended to expose the anterior 2 to 3 cm of the hippocampus. The residual parahippocampal gyrus and hippocampus are divided transversely approximately 2.5 cm posterior to the anterior tip of the hippocampus. This incision is carried down to the arachnoid overlying the cerebral peduncle. The choroid plexus is exposed and gently retracted medially to reveal the choroidal fissure, which is opened along its entire extent. With the aid of bipolar coagulation, suction, and sharp dissection, and beginning at the previously made transverse cut, the hippocampus and adjacent residual parahippocampal gyrus are then gently rolled off the arachnoid, which protects the cerebral peduncle, the third and fourth cranial nerves, and the posterior cerebral artery. The hippocampal and parahippocampal tissue is removed en bloc to permit adequate pathological examination for the presence of hippocampal sclerosis. The amygdala is not included in the resection (Figs. 1 and 2).

**Results**

**Surgical Outcome**

The surgical outcome in the 70 patients undergoing anterior temporal lobectomy is listed in Table 1. At a mean follow-up interval of 4.9 years, 55 (79%) of the patients have benefited from surgery. Of these, 23 patients have been seizure-free since their lobectomy (mean follow-up interval 4.1 years). Thirteen of this group are no longer receiving medication, six are receiving a significantly reduced dosage, and five are on the same anticonvulsant regimen as preoperatively. By significant reduction, we mean elimination of one or more drugs, or a regimen of no more than one-half the amount of medication taken preoperatively. Nineteen of the 55 patients who benefited from surgery have experienced only one to several seizures over the entire postoperative period (mean follow-up interval 6.3 years); two are no longer receiving medication, 15 are receiving a significantly reduced amount, and two are on the same regimen as preoperatively. The remaining 13 patients in the good-outcome group have had a greater than 90% reduction in seizure frequency at a mean follow-up interval of 4.4 years; seven are taking a significantly reduced amount of medication and six

<table>
<thead>
<tr>
<th>TABLE 1</th>
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<td><strong>Surgical outcome in 70 patients undergoing anterior temporal lobectomy</strong></td>
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<table>
<thead>
<tr>
<th>Postoperative Group</th>
<th>No. of Cases</th>
<th>Follow-Up Interval (yrs)</th>
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<tbody>
<tr>
<td>seizure-free since surgery</td>
<td>23</td>
<td>1--9</td>
</tr>
<tr>
<td>one to several seizures</td>
<td>19</td>
<td>2.75--9.5</td>
</tr>
<tr>
<td>&gt; 90% reduction in seizures</td>
<td>13</td>
<td>2--5.6</td>
</tr>
<tr>
<td>no benefit</td>
<td>15</td>
<td>—</td>
</tr>
<tr>
<td>total cases</td>
<td>70</td>
<td>—</td>
</tr>
</tbody>
</table>
Anterior temporary lobectomy sparing amygdala

**FIG. 1.** Photographs showing dissection of two human cadaver brains (A and B from one and C and D from the other). The pertinent medial temporal lobe anatomy encountered in anterior temporal lobectomy that spares the amygdala is illustrated. A: A major portion of the parahippocampal gyrus has been resected, and the ventricle is entered. The brain has been positioned to expose the intraventricular anatomy. *Arrows* point to the digitations of the hippocampus (H); A = caudal end of the amygdala; V = the ventricle. The *rostral broken line* represents the incision made through the anterior tip of the hippocampus. The *caudal broken line* represents the incision that is carried down to the pia arachnoid, which is left intact. These incisions free the rostral and caudal ends of the block of hippocampus and associated parahippocampal gyrus that is to be removed en bloc. *Bar* = 1 cm. B: The brain is positioned so that the ventral surface is viewed straight on. Additional hippocampus (H) and parahippocampal gyrus (PHG) have been cut away for better exposure of the amygdala (A, the dark area spanned by *arrows*), which is bulging into the ventricle. This excision is not an intermediate step in the resection of the block of hippocampus and parahippocampal gyrus described in A; it is shown to demonstrate the intraventricular relationship between the amygdala and the hippocampus. During surgery, the hippocampus is retracted medially so that the ventricular surface of the amygdala is always in view and left undisturbed. The uncus (U, highlighted by *dots*) is part of the hippocampal formation. The medial, convex portion of the parahippocampal gyrus rostral to the uncus overlies the amygdala. If any amygdala is included in the block resection, it would be in the amygdala-hippocampal transition zone and represent only a small fraction of the amygdala. C: The bed of resection is shown following excision of inferior fusiform and parahippocampal gyri. The ventricle has been entered. H = hippocampal digitations; A = tail end of the amygdala. D: Section in which the anterior 1.5 cm of hippocampus and associated parahippocampal formation have been resected. The dark area (A) is the amygdala; H = the cut end of the remaining hippocampus; *arrow 1* = the temporal pole; *arrow 2* = the posterior cerebral artery; *arrow 3* = the third intracranial nerve; *asterisk* = the basis pedunculi.

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are on a regimen comparable to the one they were on preoperatively. Fifteen patients failed to benefit from surgery.

Pathological Examination

The abnormalities encountered in the 70 patients are listed in Table 2. In 56 patients (80%), histological examination of the resected tissue showed frank (54 cases) or suggestive (two cases) hippocampal sclerosis. Of the 55 patients who benefited from surgery, 49 (90%) showed frank (47 cases) or suggestive (two cases) hippocampal sclerosis. Of the 15 patients who failed to respond to surgery, seven showed frank hippocampal sclerosis and seven showed only gliosis. It is noteworthy that only one of the 55 patients who benefited from surgery showed the nonspecific finding of gliosis.

Epidural Recordings

The epidural electrode arrays were positioned bilaterally to record the temporal EC0G signal from the anterior parahippocampal and fusiform gyri medially and to the inferior and middle temporal gyri laterally. In 49 (89%) of the 55 patients who benefited from surgery, the clinical seizure was ushered in by a sudden unilateral suppression of the EC0G signal, associated with the appearance of low-voltage fast activity and usually appearing first in the most medial leads. This

Fig. 2. Magnetic resonance images of the fixed human brain shown in Fig. 1C and D. A: Coronal section at the plane of the amygdala (A). The right (to the reader's left) amygdala has not been compromised by resection of the right anterior hippocampus and associated parahippocampal gyrus. The different darkness between the right and left amygdala is probably related to the air gap created by excision of the anterior hippocampus and associated parahippocampal gyrus. A similar difference exists between the adjacent cortex of the right and left parahippocampal gyrus. B: Coronal section, 8 mm caudal to the section shown in A. The right anterior hippocampus and associated parahippocampal gyrus have been removed. Arrow on intact side points to hippocampus (H). C and D: Sagittal sections of the right (C) and left (D) temporal lobes through the temporal horn of the lateral ventricles. Comparison of the two sides shows excision of the right anterior hippocampus (H) that has spared the amygdala (A). V = the ventricle.
suppression gave way to rhythmic seizure activity which intensified and spread to the contralateral temporal lobe. In the remaining six cases, the electrographic onset showed rhythmic seizure activity of variable frequency rather than suppression and was more prominent in the medial leads.

In the patients who had a good result, the onset of electrographic seizure activity appeared consistently on the same side from seizure to seizure, and appeared clearly before the onset of the clinical seizure. Absence of these last two features was the most common finding in the patients who failed to respond to surgery. In 11 of the 15 who failed to respond to surgery, the pattern of electrographic onset was similar to that occurring in the patients who had a good result, but it appeared either after the clinical onset, simultaneously on both sides, or independently on either side. In the four remaining failures, the electrographic onset was similar in every respect to that in the patients who benefited from surgery. Not only were the electrographic features the same but the onset occurred consistently on one side and preceded the clinical seizure.

Complications

Five patients sustained significant complications: one patient had a mild to moderate monoparesis of the right lower extremity manifested mainly by circumduction; one an impairment of position sense in the left hand; one, who preoperatively had difficulty with recent recall, had worsening of memory impairment; one a homonymous hemianopia; and one case of meningitis. The latter responded readily to antibiotics and the patient recovered without any sequelae. A total of 13 patients incurred a persistent superior homonymous quadrantanopsia. They were aware of this defect only on specific testing of the visual fields.

Transient Deficits

Five patients exhibited either partial or complete paresis of the third or fourth nerve. The symptoms and signs completely resolved over a period of weeks to several months. Six patients exhibited a transient hemiparesis which disappeared in a few days to several weeks, and eight exhibited a transient dysphasia which resolved in a few days. These symptoms developed on the 1st or 2nd postoperative day. Eleven patients showed a superior quadrantanopsia which cleared by the time of discharge. One patient developed a seventh nerve peripheral facial palsy which cleared in several weeks.

Discussion

It is clear from the imaging studies, epidural ECoG recordings, and pathological studies that the patient population in this study consisted predominantly of cases with a temporal mesiobasal seizure focus of nonneoplastic origin. Considering that our resections did not include the amygdala, the results take on an added

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**TABLE 2**

Pathology and outcome in 70 patients*

<table>
<thead>
<tr>
<th>Pathology</th>
<th>Outcome</th>
<th>Total Cases</th>
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<tbody>
<tr>
<td></td>
<td>Good</td>
<td>Poor</td>
</tr>
<tr>
<td>hippocampal sclerosis†</td>
<td>47</td>
<td>7</td>
</tr>
<tr>
<td>suggestive hippocampal sclerosis</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>focal lateral temporal sclerosis</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>focal cortical dysplasia</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>cortical malformation &amp; suggestive hippocampal sclerosis</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>leptomeningeal fibrosis</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>perivascular lymphocytic infiltrate in lateral temporal cortex, hippocampus, &amp; amygdala (5 x 5 x 3-mm biopsy specimen)</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>nonspecific astrocytosis, subpial in lateral temporal cortex, or in cortex &amp; white matter of both lateral temporal cortex &amp; hippocampus</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>total cases</td>
<td>55</td>
<td>15</td>
</tr>
</tbody>
</table>

* All patients had mild to moderate subpial astrocytosis.
† Three patients had associated leptomeningeal fibrosis.

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**TABLE 3**

Patients in current series with either a history of or an existing encephalopathy

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs) at Surgery</th>
<th>Follow-Up Interval (yrs)</th>
<th>Encephalopathy</th>
<th>Outcome</th>
<th>Pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>12</td>
<td>3</td>
<td>retardation</td>
<td>failure</td>
<td>hippocampal sclerosis</td>
</tr>
<tr>
<td>2</td>
<td>10</td>
<td>3</td>
<td>learning disability</td>
<td>&gt; 90% seizure reduction</td>
<td>hippocampal sclerosis</td>
</tr>
<tr>
<td>3</td>
<td>25</td>
<td>3.5</td>
<td>viral encephalitis at 27 mos</td>
<td>almost seizure-free</td>
<td>hippocampal sclerosis</td>
</tr>
<tr>
<td>4</td>
<td>9</td>
<td>4</td>
<td>birth injury, retardation, &amp; lt hemiparesis</td>
<td>&gt; 90% seizure reduction</td>
<td>hippocampal sclerosis</td>
</tr>
<tr>
<td>5</td>
<td>14</td>
<td>4</td>
<td>Staphylococcus sepsis at 4 days; residual moderate retardation &amp; clumsiness</td>
<td>failure</td>
<td>gliosis</td>
</tr>
<tr>
<td>6</td>
<td>14</td>
<td>5</td>
<td>viral encephalitis at 12 yrs, residual memory impairment &amp; behavior problem</td>
<td>&gt; 90% seizure reduction</td>
<td>hippocampal sclerosis</td>
</tr>
<tr>
<td>7</td>
<td>19</td>
<td>2</td>
<td>head injury at 15 yrs</td>
<td>seizure-free</td>
<td>hippocampal sclerosis</td>
</tr>
</tbody>
</table>

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significance. The surgical outcome (79% of the patients benefited from surgery) is similar to that achieved with the procedure of amygdala-hippocampectomy for temporal lobe epilepsy caused by a mesiobasal focus.28,29 That excision spares virtually all of the medial and lateral temporal cortex, removing only the amygdala, hippocampus, and parahippocampal gyrus. Taken together, the results of that procedure and ours strongly suggest that, in the majority of patients with a temporal mesiobasal focus, the hippocampus is at the hub of epileptogenic propagation, and its removal or the neutralization of its influence is key to a successful outcome. Yet, similar outcomes are documented for a variety of anterior temporal lobectomies in which the amount of hippocampus removed varies from excision of the entire hippocampus25,29 to removal of tissue measuring 2.5 to 3 cm,5,7,12,18,20 1 to 1.5 cm,22,23 or less than 1 cm * to no tissue removal.14

**Neuroanatomical Considerations**

Recent neuroanatomical studies on the connectivity between the hippocampus and the neo- and limbic cortices provide a possible explanation of these results.1,2,18,26,27 The anterior portion of the parahippocampal gyrus, which overlies the anterior part of the hippocampus and amygdala, is entorhinal cortex. It receives a major input from polysensory associational areas of the temporal, frontal, parietal, and limbic cortices, mainly via the posterior parahippocampal gyrus and perirhinal area. This input converges on islands of cells in layer 2 of entorhinal cortex and from there projects massively, as the excitatory perforant pathway, to the dentate gyrus and CA regions of the hippocampus. The output from the hippocampus projects back to the entorhinal cortex, from which there is a reciprocal projection back to polysensory associational and limbic cortices. Thus, the entorhinal cortex is in the position of gatekeeper for the reciprocal flow of information between the hippocampus and the neo- and limbic cortices. A lesion of the entorhinal cortex effectively isolates the hippocampus, because the latter has no direct input from the cerebral cortex (Figs. 3 and 4).

We suggest that removal of the entorhinal cortex might be the common denominator that explains the similar results among the varieties of anterior temporal lobectomies. If one assumes that the seizure activity begins in the hippocampus, the site of pathology, but clinical expression of the seizure requires spreading to areas outside the hippocampus where the necessary mass of neural tissue can be recruited to trigger a clinical seizure, then removal of the entorhinal cortex could be as effective for preventing a seizure as excision of the hippocampus. Thus, the similar surgical outcomes among the varieties of temporal lobe resection may reflect excision of the entorhinal cortex that was included.5,7,12,18,20,22,23 or might have been included,14 in all of those procedures. With respect to the neocortical excision, in which both the amygdala and the hippocampus are spared and only temporal lobe cortex is removed, the good results have been attributed to the site of pathology, to a neuronal dysgenesis, or to ectopia of the temporal neocortex.14 It is proposed that the epileptogenic focus in those cases was limited to the temporal neocortex without involvement of the temporal mesiobasal lobe, and that is why they achieved good results with excisions that spared both the hippocampus and amygdala. However, if their resections included the anterior parahippocampal gyrus, then that could be an alternative explanation of the surgical outcome.

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* W Feindel, et al.: Data presented as Poster 1138 at the 1991 American Association of Neurological Surgeons annual meeting.

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**Fig. 3.** Diagrams summarizing cortical afferent and efferent connections of the entorhinal cortex, which is the predominant source of projections to the hippocampus. The major cortical input to entorhinal cortex (black area in ventral view of monkey brain, center) originates in the adjacent parahippocampal gyrus, areas TF/TH of von Economo's atlas (stippled area), and perirhinal cortex, area 35/36 of Brodmann's atlas (cross-hatched area). These regions in turn receive projections from several polysensory associational regions in the frontal, temporal, and parietal lobes (upper). The entorhinal cortex also receives cortical inputs directly from other presumed polysensory regions and one unimodal input from the olfactory bulb (stippled area, lower). With the exception of the olfactory projection, these connections are reciprocal. STG = superior temporal gyrus. (Modified from Squire LR, Zola-Morgan S: Memory: brain systems and behavior. Trends Neurosci 11:170-175, 1988.)
Anterior temporary lobectomy sparing amygdala

Fig. 4. Photograph, ventral view, of the human brain showing both the gyral topographic anatomy of the medial temporal lobe and the distribution within the gyri of the architectonic areas that correspond to those shown in the diagram of the monkey brain (Fig. 3). Arrow 1 denotes the collateral sulcus; arrow 2 the rhinal sulcus; arrow 3 the third intracranial nerve; and arrow 4 the posterior cerebral artery. The perirhinal cortex (PR) is identified by small circles; much of this lies buried in the walls of the collateral sulcus. The entorhinal cortex (ER) is identified by small crosses, the uncus (U) by dots, and the posterior parahippocampal cortex (PH) by asterisks. We prefer the term "posterior parahippocampal cortex" because the word "gyrus" designates a gross anatomical structure, whereas the terms "entorhinal cortex," "perirhinal cortex," and "posterior parahippocampal cortex" refer to architectonic areas defined by light microscopy. Thus, the parahippocampal gyrus harbors the entorhinal cortex in its approximate anterior two-thirds, the posterior parahippocampal cortex in its approximate posterior one-third, and the perirhinal cortex predominantly in the walls of the collateral sulcus. Bar = 1 cm.

Treatment Failures

There remains for discussion the four patients who failed to respond to surgery, even though the electrographic findings indicated a unilateral mesiobasal focus. The findings in one of these patients provide an explanation, at least in his case. In 1982, monitoring during clinical seizures showed an electrographic profile typical of those patients who benefited from surgery. A sudden right-sided suppression of the ECoG signal in the medial leads ushered in each clinical seizure. The suppression gave way to frank seizure activity which built in amplitude and spread first to the more lateral rightsided leads and then to the left side. At that time a high-resolution CT scan was normal. The patient failed to respond to a right anterior temporal lobectomy, and histological examination of the resected tissue revealed only gliosis of the lateral and medial temporal cortex. There was no hippocampal sclerosis. In 1987, an MR image revealed a lesion which proved to be a ganglioglioma in the right posterior temporal lobe at the level of the trigone. Since the 1987 resection of the tumor, which invaded the posterior hippocampus, the patient has been seizure-free (PJ Kelly, personal communication, 1991). In all probability, the ganglioglioma was present in 1982 and was the cause of the seizures. Computerized tomography scans sometimes miss a ganglioglioma that is readily revealed by an MR image. Yet, the epidural ECoG recording indicated an anterior mesiobasal focus. Probably the electrographic onset of the seizure invaded the anterior and posterior parahippocampal gyrus simultaneously, and the epidural electrodes captured the anterior discharge, leading us to interpret the recording as being consistent with hippocampal sclerosis.

The other three failed cases showed hippocampal sclerosis and require a different explanation. We have considered the following possibilities. First, although hippocampal sclerosis occurs mainly in the anterior hippocampus,17 it may have extended into the posterior hippocampus which was not included in the anterior...
Neural Circuitry of Memory

Our results and their interpretation are reminiscent of recent studies on memory impairment.\(^{15,20,21}\) A pathophysiology that affects some of the neural circuitry as do complex partial seizures. Zola-Morgan, et al.,\(^21\) demonstrated that lesions of the perirhinal and parahippocampal cortex that spare the amygdala and hippocampus produce severe memory impairment. Furthermore, bilateral stereotactic lesions of the amygdala that spare adjacent cortex do not impair memory, nor do amygdalar lesions exacerbate memory impairment due to lesions of the hippocampal formation. These studies suggest that the severe memory impairment in monkeys and humans associated with bilateral medial temporal lesions results from damage to the hippocampal formation and adjacent anatomically related cortex, not from conjoint hippocampus-amygdala damage.\(^{20}\)

Conclusions

On the basis of our data and that in the literature reviewed herein, we believe it is timely to consider collection of a series of patients in whom resections are limited to the anterior hippocampus and/or associated anterior parahippocampal gyrus. The patient population should be limited to individuals who harbor a temporal non-neoplastic mesiobasal focus.

Acknowledgments

The technical assistance of Virginia R. Hobbs, R.N., M.S.N., and Karl L. Probst is gratefully acknowledged. We thank Joseph L. Price, Ph.D., for critically reviewing the discussion on the connectivity of the medial temporal lobe, and Mokhtar Gado, M.D., for the MR images reproduced in Fig. 2.

References

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Manuscript received October 28, 1991. Accepted in final form February 4, 1992. Address reprint requests to: Sidney Goldring, M.D., Department of Neurosurgery, Washington University School of Medicine, 660 South Euclid Avenue, Box 8057, St. Louis, Missouri 63110.