Fast Spin-Echo MR in Hippocampal Sclerosis: Correlation with Pathology and Surgery

Jae Hyoung Kim, Robert D. Tien, Gary J. Felsberg, Alan K. Osumi, Namsoo Lee, and Allan H. Friedman

PURPOSE: To identify the extent of hippocampal sclerosis in temporal lobe epilepsy with fast spin-echo MR and correlate it with histopathologic findings and surgical outcome. METHODS: MR images of 30 patients with temporal lobe epilepsy and pathologically proved hippocampal sclerosis and 30 control subjects were obtained using a fast spin-echo technique with 4000/100/4 (repetition time/echo time/excitations), 16 echo train, 2- to 3-mm section thickness with interleave, 256 × 256 matrix, and 18-cm field of view. Criteria for MR diagnosis of hippocampal sclerosis included hippocampal atrophy diagnosed with MR volumetry and/or T2-weighted signal change. Hippocampal sectional areas were plotted, and T2 signal changes were topographically evaluated to identify the extent of hippocampal sclerosis, which was subsequently correlated with histopathologic findings and surgical outcome. RESULTS: Hippocampal sclerosis was diffuse, involving both hippocampal head and body, in 96.7% of patients (29 of 30 patients). One patient had normal MR findings. Focal hippocampal sclerosis was not seen. Histopathologic findings of hippocampal sclerosis were present in all 29 patients who had abnormal MR findings. Eighty-six percent of patients (18 of 21 patients), who were followed for at least 1 year after temporal lobectomy, were seizure free (81%, 17 of 21 patients) or significantly improved (5%, 1 of 21 patients). CONCLUSION: Fast spin-echo MR enables accurate definition of the extent of hippocampal sclerosis in patients with temporal lobe epilepsy. All cases of hippocampal sclerosis identified in this study involved the hippocampus diffusely. However, leaving the posterior portion of the hippocampus during surgery does not seem to be a major factor influencing surgical outcome.

Index terms: Brain, magnetic resonance; Brain, temporal lobe; Sclerosis, hippocampal; Seizures


Hippocampal sclerosis refers to neuronal loss and gliosis of the hippocampus. Hippocampal sclerosis is the most common pathologic abnormality associated with medically intractable temporal lobe epilepsy (1, 2). The radiologic diagnosis of hippocampal sclerosis is based on the magnetic resonance (MR) findings of atrophy and/or T2 signal change of the hippocampus (3–10). Neuropathologically, the distribution and extent of neuronal loss and gliosis within the hippocampus (topographical pattern of hippocampal sclerosis) may be related to different surgical outcomes in patients with temporal lobe epilepsy treated by surgery (2, 11, 12). Several distinct topographical types of hippocampal atrophy in patients with temporal lobe epilepsy have been described with MR imaging (13).

Recently, fast spin-echo MR imaging has been used to obtain high-resolution anatomic information and T2 signal character of the hippocampus with one pulse sequence (3, 14). In this study, we identified the extent or topographical distribution of hippocampal sclerosis in patients with temporal lobe epilepsy by fast spin-echo MR examination and correlated the extent of hippocampal sclerosis with histopathologic examination and with patient outcome after surgery.
Subjects and Methods

During the period of 2.6 years, 33 patients, who had no focal epileptogenic extrahippocampal lesions, underwent anterior temporal lobectomy for the treatment of intractable temporal lobe epilepsy caused by presumed hippocampal sclerosis. Of these 33 patients, 30 had diagnoses of hippocampal sclerosis, and 3 patients had normal findings on histopathologic examinations. The pathologic diagnosis of hippocampal sclerosis was made qualitatively without hippocampal neuronal counting by an experienced neuropathologist. Thirty patients with pathologically proved hippocampal sclerosis were included in this study. During the surgical procedure, the hippocampal head and anterior body were resected en bloc, and the posterior body was then removed by subpial aspiration. This technique leaves a small portion of the posterior body and tail of the hippocampus remaining. There were 16 male and 14 female patients with a mean age of 31.7 years (range, 13 to 57 years). All patients underwent a standardized preoperative protocol for location of seizure focus (15). Intracranial depth electroencephalography was used in several patients. A control group consisted of both volunteers and patients without clinical history of seizures. They were 15 male and 15 female patients with a mean age of 33.2 years (range, 14 to 56 years). All patients and control subjects underwent MR examinations on a 1.5-T system. The imaging protocol included conventional spin-echo T1- and T2-weighted axial images and T1-weighted coronal images. Additionally, high-resolution coronal T2-weighted fast spin-echo imaging of the temporal lobe was performed as previously described (3, 14): 4000/100/4 (repetition time/echo time/excitations), 16 echo train length, 2- to 3-mm section thickness with interleave, 256 × 256 matrix, and 18-cm field of view. The imaging plane was perpendicular to the long axis of the hippocampus.

Visual analysis of all fast spin-echo MR images was performed to assess the T2 signal change of the hippocampus. The presence or absence of increased T2 signal at each image was visually determined by the consensus of three neuroradiologists. The location of fast spin-echo images demonstrating T2 signal change was recorded for correlation with the topographical distribution of hippocampal atrophy.

All fast spin-echo MR images were loaded on the Signa console (General Electric, Milwaukee, Wis) for volumetry. In a blinded fashion, a neuroradiologist with experience in MR volumetry measured the cross-sectional areas of each hippocampus manually, tracing the hippocampus from the head to tail on each successive section as previously reported (3). Hippocampal volumes were then calculated by summing areas and multiplying by section thickness. To determine unilateral hippocampal atrophy, a mean volume difference (right hippocampal volume minus left hippocampal volume) and standard deviation were used. Values beyond 2 SD of mean volume difference of control subjects were determined to be abnormal.

To identify the topographical distribution type of hippocampal sclerosis, diagrams of hippocampal successive cross-sectional areas measured on MR images were plotted and combined with the T2 signal information of the hippocampus. Successive cross-sectional areas of both hippocampi were plotted against the long axis of the hippocampi to compare easily both hippocampal volumes section to section as previously performed (13). Because visual comparison of cross-sectional areas of both hippocampi was sensitive to the patient's head rotation in the coronal plane, the degree of head rotation was estimated by comparing the bilateral symmetric structures of the gyrus intralimbicus, which is an internal marker separating the head and body of the hippocampus. When the degree of head rotation in the coronal plane was more than one section thickness, the area curve plots were shifted accordingly to correct for rotation. The gyrus intralimbicus is a small structure composed of the cornu Ammonis subfields 3 and 4 and constitutes the medial portion of the most posterior segment of the hippocampal head. When tracing the coronal MR images from the hippocampal head toward the tail, the level of the gyrus intralimbicus can be identified by recognizing the absent digitations and the bulbous medial mass of the hippocampal head (Fig 1).

The extent of hippocampal sclerosis was determined, by the consensus of three neuroradiologists, by visually comparing area-curve plots of the patients with those of the 30 control subjects. The topographical distribution of hippocampal atrophy was classified into diffuse (involving the hippocampal head and the body), anterior focal (involving the head to the level of the gyrus intralimbicus), and posterior focal types (only involving the body and/or tail). The level of the gyrus intralimbicus and that of the quadrigeminal plate were considered the most caudal sections of the hippocampal head and body, respectively. The distribution of T2 signal change was also classified similarly. These two parameters (hippocampal atrophy and T2 sig-
nal change) were then incorporated into a final determination of the topographical distribution of hippocampal sclerosis. On any section of an individual hippocampus, if either hippocampal atrophy or T2 signal change existed, that individual section was considered positive for hippocampal sclerosis.

The fast spin-echo MR-derived topographical types of hippocampal sclerosis were compared with the histopathologic extents of hippocampal neuronal loss. The presence or absence of hippocampal neuronal loss was qualitatively determined by a neurologist experienced in neuropathologic examination of hippocampi, without quantitative neuronal counting, by reviewing specimens of both the hippocampal head and body. The hippocampal head and body were differentiated by histologic characteristics; the hippocampal head has the folding of the granular cell layer of the dentate gyrus and the pyramidal cell layer of the cornu Ammonis, which are absent in the hippocampal body. Because of the method of surgical removal, an exact section-to-section comparison between the fast spin-echo MR images and the histopathologic specimen was not possible.

Surgical outcomes were assessed in all patients who were evaluated postsurgically for at least 1 year and were compared with the fast spin-echo MR-derived extent of hippocampal sclerosis. Surgical outcomes were classified into seizure free, significantly improved (defined as 10 or less seizures per year and more than a 75% decrease in seizure rate), and not significantly improved (defined as more than 10 seizures per year or less than a 75% decrease in seizure rate) as previously defined (15).

Results

The data of MR-based topographical types, T2 signal change, histopathologic examinations, and surgical outcomes of hippocampal sclerosis are summarized in the Table.

Summary of MR volumetry, topographical type, and surgical outcome

<table>
<thead>
<tr>
<th>Patient</th>
<th>MR Volumetric Lateralization*</th>
<th>MR-Based Topographical Type</th>
<th>Pathologic Correlation?</th>
<th>Surgical Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Atrophy</td>
<td>T2 signal</td>
<td>Combined</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>R (-0.666)</td>
<td>P</td>
<td>D</td>
<td>D</td>
</tr>
<tr>
<td>2</td>
<td>R (-1.086)</td>
<td>D</td>
<td>D</td>
<td>D</td>
</tr>
<tr>
<td>3</td>
<td>R (-1.281)</td>
<td>D</td>
<td>D</td>
<td>D</td>
</tr>
<tr>
<td>4</td>
<td>R (-0.854)</td>
<td>D</td>
<td>D</td>
<td>D</td>
</tr>
<tr>
<td>5</td>
<td>R (-0.774)</td>
<td>D</td>
<td>D</td>
<td>D</td>
</tr>
<tr>
<td>6</td>
<td>R (-0.570)</td>
<td>D</td>
<td>D</td>
<td>D</td>
</tr>
<tr>
<td>7</td>
<td>R (-1.161)</td>
<td>D</td>
<td>D</td>
<td>D</td>
</tr>
<tr>
<td>8</td>
<td>R (-0.680)</td>
<td>D</td>
<td>D</td>
<td>D</td>
</tr>
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<td>9</td>
<td>R (-0.843)</td>
<td>D</td>
<td>D</td>
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</tr>
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<td>10</td>
<td>R (-0.372)</td>
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<td>11</td>
<td>R (-1.236)</td>
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<td>D</td>
<td>D</td>
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<td>12</td>
<td>R (-0.528)</td>
<td>D</td>
<td>NS</td>
<td>D</td>
</tr>
<tr>
<td>13</td>
<td>R (-0.759)</td>
<td>D</td>
<td>NS</td>
<td>D</td>
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<td>14</td>
<td>R (-1.022)</td>
<td>D</td>
<td>D</td>
<td>D</td>
</tr>
<tr>
<td>15</td>
<td>L (0.981)</td>
<td>D</td>
<td>D</td>
<td>D</td>
</tr>
<tr>
<td>16</td>
<td>L (0.720)</td>
<td>D</td>
<td>D</td>
<td>D</td>
</tr>
<tr>
<td>17</td>
<td>L (0.584)</td>
<td>D</td>
<td>D</td>
<td>D</td>
</tr>
<tr>
<td>18</td>
<td>L (0.891)</td>
<td>D</td>
<td>D</td>
<td>D</td>
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<td>19</td>
<td>L (0.986)</td>
<td>D</td>
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<td>20</td>
<td>L (1.215)</td>
<td>D</td>
<td>D</td>
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<td>21</td>
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<td>D</td>
<td>D</td>
<td>D</td>
</tr>
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<td>22</td>
<td>L (1.090)</td>
<td>D</td>
<td>D</td>
<td>D</td>
</tr>
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<td>23</td>
<td>L (1.060)</td>
<td>D</td>
<td>D</td>
<td>D</td>
</tr>
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<td>24</td>
<td>L (0.764)</td>
<td>D</td>
<td>D</td>
<td>D</td>
</tr>
<tr>
<td>25</td>
<td>L (1.224)</td>
<td>D</td>
<td>D</td>
<td>D</td>
</tr>
<tr>
<td>26</td>
<td>NL (0.222)</td>
<td>NA</td>
<td>NS</td>
<td>N</td>
</tr>
<tr>
<td>27</td>
<td>R (-0.420)</td>
<td>P</td>
<td>Db</td>
<td>Db</td>
</tr>
<tr>
<td>28</td>
<td>NL (0.156)</td>
<td>Db</td>
<td>Db</td>
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<td>29</td>
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<td>Db</td>
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<td>Db</td>
</tr>
<tr>
<td>30</td>
<td>NL (0.172)</td>
<td>Db</td>
<td>D‡</td>
<td>Db</td>
</tr>
</tbody>
</table>

Note.—D indicates diffuse; Db, bilaterally diffuse; N, normal; NA, no atrophy detected; NL, no lateralization detected; NS, no T2 signal change detected; NSI, not significantly improved; P, posterior; SF, seizure-free; and SI, significantly improved.

* The number in parentheses is the hippocampal volume difference (right minus left) in cubic centimeters.
† The patient showed mild hippocampal neuronal loss compatible with hippocampal sclerosis but had normal MR findings.
‡ Diffuse type of T2 signal change was noted in the resected hippocampus.
Fast Spin-Echo MR Volumetric Lateralization

The mean difference of hippocampal volumes of controls was 0.079 cm³ (SD, 0.177 cm³). Lateralization of the abnormal hippocampus with fast spin-echo MR volumetry was performed in 26 of the 30 patients with hippocampal sclerosis. There was no false lateralization. Four patients were not lateralized. Three of these four patients (patients 28, 29, and 30) had evidence of bilateral hippocampal atrophy based on MR volumetry. All three had corroborative evidence of bilateral hippocampal sclerosis by depth electroencephalographic findings, and the dominant seizure-producing hippocampus was resected. One patient (patient 26) with pathologically proved hippocampal sclerosis showed no lateralization and bilaterally reduced hippocampal volumes, although the right hippocampus was resected based on the depth electroencephalographic finding.

Topographical Distribution of Signal Abnormality

Of the 26 patients who were lateralized by fast spin-echo MR volumetry, 23 also had visually identified T2 signal change ipsilaterally; the signal change was diffuse in all 23. Another patient (patient 27) had bilaterally increased T2 signal suggesting bilateral hippocampal sclerosis; the right hippocampus was resected based on MR evidence of unilateral atrophy and the consistent depth electroencephalographic finding of right-sided seizure activity. Two other patients (patients 12 and 13) who were lateralized by volumetry showed no signal abnormality. Of the 3 patients with definite bilateral hippocampal atrophy, 2 patients (patients 28 and 29) showed bilateral diffuse T2 signal change, and 1 patient (patient 30) showed unilateral diffuse signal change in the dominant seizure-producing hippocampus. One patient (patient 26), who was not lateralized and did not show bilaterally reduced hippocampal volumes, did not show signal abnormality.

Topographical Distribution of Atrophy

From the area curves of the 30 control subjects, a close approximation between the right and left hippocampal area curves was demonstrated against the entire hippocampal axis (Fig 2), although a minimal discrepancy between both curves was often found at the level of the hippocampal head (Fig 3). Twenty-six patients with fast spin-echo MR volumetric lateralization showed definite discrepancies between both area curves, suggesting hippocampal atrophy; the atrophy was of the diffuse type in 24 (Figs 4 and 5) and the posterior focal type in 2 (patients 1 and 27) (Fig 6). Three patients (patients 28, 29, and 30) showed bilateral diffuse atrophy; there were no definite discrepancies between both area curves but definitely reduced absolute volumes bilaterally when compared with controls (Fig 7). One patient (patient 26) did not show any discrepancy suggesting atrophy.

Combination of Topographical Distribution of Atrophy and Signal (Topographical Distribution of Hippocampal Sclerosis)

Of the 30 patients, 22 showed both unilateral diffuse hippocampal atrophy and T2 signal change. Two patients (patients 12 and 13)
showed unilateral diffuse hippocampal atrophy but no T2 signal change. Two patients had posterior focal atrophy; 1 (patient 1) had ipsilateral diffuse T2 signal change, and the other (patient 27) had bilaterally diffuse T2 signal change suggesting bilateral hippocampal sclerosis.

Three patients had bilateral diffuse hippocampal atrophy; 2 (patients 28 and 29) also had bilateral diffuse T2 signal abnormality, and 1 (patient 30) had diffuse signal only in the dominant seizure-producing hippocampus. One patient had normal fast spin-echo MR findings. Overall, considering both hippocampal atrophy and T2 signal change, 29 of the 30 patients were determined to have the diffuse type of hippocampal sclerosis (25 unilateral diffuse type and 4 bilateral diffuse type).

**Histopathologic Correlation**

Sixteen patients with intact histopathologic specimens of both the hippocampal head and body showed good correlations between the MR-determined and histopathologic extent of hippocampal sclerosis. Thirteen patients with intact histopathologic specimens of either the hippocampal head or body also demonstrated positive correlations between the MR-determined extent of hippocampal sclerosis and histopathologies; all showed neuronal loss on each available specimen (of the hippocampal head in five patients and the body in eight patients). One patient (patient 26) with normal MR findings showed mild neuronal loss on the histopathologic specimen of only the hippocampal body.

**Surgical Outcomes**

Clinical follow-up after temporal lobectomy for the 30 patients ranged from 6 months to 2.6 years. Surgical outcomes were assessed in 21 patients who were postsurgically evaluated for
at least 1 year (mean, 1.8 years) with 17 patients seizure free, 1 patient significantly improved, and 3 patients (patients 24, 26, and 27) not significantly improved. Of these three, 1 patient (patient 24) had definite MR evidence of unilateral hippocampal sclerosis; another patient (patient 27) had bilateral diffuse T2 signal change; and the other patient (patient 26) had normal fast spin-echo MR findings.

Discussion

Hippocampal sclerosis refers to an entity of neuronal loss and atrophy with associated gliosis involving the hippocampus and is the most common cause of medically intractable temporal lobe epilepsy (1, 2). Temporal lobectomy with hippocampectomy is a widely accepted surgical treatment for patients with hippocampal sclerosis (16–18). Accurate decision as to which hippocampus to resect is essential before surgical treatment of hippocampal sclerosis. MR imaging has been used to assess the hippocampus for the evaluation of patients with temporal lobe epilepsy and demonstrated its valuable role in depicting the abnormalities of the hippocampus (atrophy and/or increased T2 signal of the hippocampus) and lateralizing the seizure focus (3–10). Significant correlations between the hippocampal size measured on MR imaging and hippocampal neuronal density in surgical specimens have been found in patients with hippocampal sclerosis (9, 19, 20) (Lee N, Lewis D, Tien R, et al, “Hippocampal Sclerosis: Fast Spin Echo MRI Volumetry and Neuronal Loss” [abstract], Epilepsia 1993;34 [suppl 6]:129–130). Increased T2 signal of the hippocampus, reflecting gliosis, is another important indicator of hippocampal sclerosis (3, 7, 10, 21, 22). Previous MR imaging studies have described a variable frequency of T2 signal change of the hippocampus, occurring in 12% to 65% of patients with hippocampal sclerosis (4, 10, 23). However, with the improvement of

Fig 5. A, Hippocampal area curves of the diffuse type of hippocampal sclerosis (patient 1). Right hippocampal atrophy is noted predominantly in the body level; however, T2 signal was diffusely increased throughout the right hippocampus (arrows): sections at the levels of the hippocampal head (B), the body (C), and the tail (D).
MR imaging techniques, several recent MR studies have verified T2 signal change of the hippocampus to be a highly sensitive MR finding suggesting hippocampal sclerosis, occurring in 84% to 100% (3, 22) (Ojemann LM, Tsuruda JS, Holmes MD, Alvord EC, Ojemann GA, Hayes CF, “Comparison of Clinical Features, Histology and High Resolution Fast Spin MRI Using a Phased Array Coil in Patients Undergoing Surgery for Temporal Lobe Epilepsy” [abstract], Epilepsia 1993;34 [suppl 6]:136).

Several topographical types of hippocampal atrophy in patients with temporal lobe epilepsy were described by the MR volumetric and morphometric method (13). In this method, right and left hippocampal volumes could be easily compared with each other along the hippocampal long axis. We combined the method of MR volumetry and T2 signal change of the hippocampi to identify a more accurate topographical distribution of hippocampal sclerosis. This information could be obtained by fast spin-echo coronal hippocampal MR images, and with this technique, all patients but one showed evidence of diffuse hippocampal sclerosis. Posterior focal atrophy of the hippocampus with diffuse T2 signal change was found in two patients. Additionally, because hippocampal sclerosis was found in our study to be a diffuse process, commonly observed minor variations in hippocampal anatomy from side to side on MR should not indicate hippocampal sclerosis.

MR volumetry for the classification of topographical distribution of hippocampal sclerosis has some technical drawbacks. First, mild discrepancy between area curves bilaterally was often found at the level of the hippocampal head in the control group. This finding reflects the complex morphology of the hippocampal head as opposed to the uniform morphology of the hippocampal body. A discrepancy in the hippocampal head area curves was also noted even with slight rotation of the patient’s head.
Therefore, when the degree of patient head rotation was more than one section thickness (2 to 3 mm) by comparing the gyrus intralimbicus bilaterally, an appropriate correction was made on the corresponding area curve. Second, the discrepancy in the anterior-to-posterior dimensions of both hippocampi occurred normally, which may result in difficulty in matching the hippocampal area curves. Therefore, we used the gyrus intralimbicus as an anatomic landmark for matching both hippocampal area curves. However, mild focal atrophy of the hippocampal head may not be detected easily in this manner, so the consideration of T2 signal becomes a significant parameter in the diagnosis of hippocampal sclerosis.

Combining the information obtained from the area-curve plots and T2 signal change, diffuse hippocampal sclerosis was the only topographical type seen in our study. Similar findings have been reported (Ojemann et al, “Comparison of Clinical Features”; Kuzniecky R, Faught E, Morawetz R, Black L, “MRI Patterns of Mesiotemporal Atrophy in Intractable Temporal Lobe Epilepsy” [abstract], Epilepsia 1993;34 [suppl 6]:141); however, a recent investigation, although described in patients who did not undergo surgery, reported that the anterior focal type was more frequent (60%) than the diffuse type (35%) (13). This discrepancy may result from differences in patient selection, the definition of hippocampal head and body, and the scan technique (eg, the lack of T2-weighted scans). Our results are supported by correlative histopathologic examination in 16 patients, although our technique was a qualitative method without exact section-to-section comparison between the MR sections and histopathologic sections. The validity of our technique is also supported by previous studies describing that the hippocampal size or volume correlates well with hippocampal neuronal density (9, 19, 20) (Lee et al, “Hippocampal Sclerosis”).

Most patients with hippocampal sclerosis become seizure free or significantly improved after surgical treatment; however, some patients do not improve. Two patterns of hippocampal sclerosis have been classified by means of histopathologic examination with implications concerning surgical outcomes: the diffuse pattern (neuronal loss in the hippocampal head and body equally) with a worse surgical outcome than that of the focal pattern (neuronal loss in the hippocampal head predominantly) (2, 11, 12). Better surgical outcomes have also been reported in patients whose resected hippocampi showed definite neuronal loss and atrophy than in the patients without those findings (24–26). The persistence of the most posterior portion of the hippocampus (presumably containing similar neuronal loss and gliosis) in patients after temporal lobectomy has been postulated to have an influence on surgical outcomes (2, 12). However, most of the patients with the diffuse type of hippocampal sclerosis in our study were seizure free or significantly improved during at least a 1-year follow-up period despite the remainder of the presumably atrophied and gliotic posterior hippocampus. Further follow-up is needed, because seizures may recur in a minority of patients after a longer postoperative period (15). Our results are supported by several reports in which similar surgical outcomes of hippocampal sclerosis have been documented regardless of the extent of the hippocampal resection (16, 25, 27–29). It has been postulated that sufficient removal of the entorhinal cortex is a major factor to control seizure activity, because it is a functional area (Brodman’s area 28) conducting the propagation of seizures arising from the hippocampus (30). This area is located in the anterior portion of the parahippocampal gyrus, but its posterior extension along the parahippocampal gyrus is uncertain (31). Therefore, it may be that the posterior extent of hippocampal sclerosis will not greatly influence surgical out-

![Fig 7. Atrophic change is noted in right hippocampus; however, T2 signal change is not remarkable (arrows) (patient 12).](image-url)
comes if the surgical procedure includes removal of the anterior hippocampus and a sufficient amount of the entorhinal cortex. Insufficient resection of the entorhinal cortex may have been the cause of poor surgical outcome in one patient (patient 24). Another possible cause is propagation of the seizure activity along the fornix to the frontal cortex or the cingulate gyrus instead of propagating via the entorhinal cortex to other cortical areas (30). Preliminary investigation suggests surgical failure may occur if the seizure focus is bilateral or widespread in the temporal lobe (2), and this may be the cause of the poor postoperative outcome of another patient (patient 27). The third patient (patient 26) with poor outcome had a normal fast spin-echo MR finding, and histopathological examination of this case revealed only very mild hippocampal neuronal loss. Prior reports have shown that surgical outcome is not optimal in patients whose resected hippocampi do not show significant neuronal loss and atrophy (24–26).

Our study has identified the relationship among the extent of hippocampal sclerosis determined by fast spin-echo MR, pathologic findings, and surgical outcome. All cases of hippocampal sclerosis identified on fast spin-echo MR involved the hippocampus diffusely from the head to the posterior body or tail, and focal involvement was not seen. The surgical outcome of patients with the diffuse type of hippocampal sclerosis was excellent in our study, and therefore it can be assumed that persistence of the posterior portion of the pathologic hippocampus is not a factor influencing surgical outcome. Awareness of the topographical distribution of hippocampal sclerosis within the hippocampus aids in the interpretation of hippocampal sclerosis of hippocampal MR images in patients with seizure and in the presurgical evaluation of these patients.

References


