Regional Distribution of MR Findings in Hippocampal Sclerosis

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PURPOSE: To investigate the distribution of MR findings in the hippocampus and amygdala in patients with hippocampal sclerosis. **METHODS:** We blindly evaluated MR scans for atrophy and signal changes occurring in the amygdala, hippocampal head, hippocampal body, and hippocampal tail in 57 consecutive patients with hippocampal sclerosis proved by pathologic analysis. **RESULTS:** Regional atrophy or signal change was present in limbic structures. Atrophy was detected in 52 patients, occurring in the amygdala in 7 (12%), hippocampal head in 29 (51%), hippocampal body in 50 (88%), and hippocampal tail in 35 (61%). Hyperintense signal on long-repetition-time images was observed in 49 patients and involved the amygdala in 2 (4%), hippocampal head in 22 (39%), hippocampal body in 46 (81%), and hippocampal tail in 28 (49%). Thirty patients (53%) had abnormal MR findings distributed through the entire ipsilateral hippocampus, 25 (44%) had regional rather than widespread involvement of limbic structures, and 2 (3%) had no MR abnormalities. **CONCLUSION:** Signal and volume changes associated with hippocampal sclerosis affect the entire hippocampus in most patients. However, a substantial number of patients have MR abnormalities that are regional, involving only portions of the hippocampus and amygdala. The most frequently affected region was the hippocampal body. These findings can have important implications for surgery and quantitative image analysis, if the seizure generator is related to MR changes.

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

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Hippocampal sclerosis, also known as mesial temporal sclerosis, is a disorder characterized by hippocampal neuronal loss and medial temporal lobe seizures. Although it is the most common entity associated with medically intractable temporal lobe epilepsy, it is potentially curable by surgery. Identification of the seizure-onset zone and its complete removal are crucial for surgical therapy to be successful. Invasive electroencephalography and magnetic resonance (MR) imaging can accurately predict hippocampal sclerosis, with implications for surgical success, but there still are patients who are not cured by surgery (1–7). One factor that may be related to surgical failure is the type of surgery performed (5, 6, 8). A variety of surgical approaches have been advocated for mesial temporal lobe epilepsies with varying degrees of success (1–7). Some investigators favor resection of the entire medial (limbic) temporal lobe (6, 7), whereas others resect primarily either the anterior (amygdala) (3) or posterior (hippocampus) (4) portions. Because sclerotic tissue correlates with the epileptogenic focus (9) and surgical success is dependent on excision of all epileptogenic tissue (10–12), precise definition of the epileptogenic tissue is the goal in these patients. Several reports have noted that MR findings in mesial temporal sclerosis vary in the amount and uniformity of tissue affected (13–18). The hippocampal body appears to be affected more often than other hippocampal segments. The amygdala also has been implicated in this disorder. Demonstration of regional involvement by hippocampal sclerosis could have important implications for surgery if the

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seizure generator is related to the MR changes. We sought to investigate these regional changes in patients who had hippocampal sclerosis verified by histologic analysis.

Materials and Methods

All patients who underwent epilepsy surgery between July 1986 and January 1990 were enrolled in this study. Patients who had previous surgery or who could not have MR studies done because of contraindications, such as pacemaker or shrapnel, were excluded. In the remaining 144 patients a neuroradiologist, blinded to all clinical and pathologic data, retrospectively evaluated MR scans for hippocampal and amygdala atrophy and signal changes, based on visual inspection. The medial temporal limbic structures were divided into four segments as designated by Duvernoy: the amygdala, hippocampal head, hippocampal body, and hippocampal tail (19–21). The amygdala is distinguished from the hippocampal head by the temporal horn. The hippocampal body was designated as those segments of hippocampus adjacent to the brain stem. The hippocampal tail was designated as that section of hippocampus that started at the posterior aspect of the brain stem and was seen on the coronal section that included the superior colliculus (19–21). The junctions between the amygdala and hippocampal head, hippocampal head and body, and hippocampal body and tail also were evaluated. Signal hyperintensity on long-repetition-time images and atrophy detected on long- or short-repetition-time images were graded absent or present for all four limbic segments in all 144 patients. This report evaluates those 57 patients with histologic evidence of hippocampal sclerosis. There were 31 female and 26 male subjects. An additional 11 patients who did not have hippocampal sclerosis by histology underwent temporal lobectomy for temporal lobe seizures that were presumed before surgery to be attributable to hippocampal sclerosis. The remaining patients, 76 of 144, had either corpus callosotomy surgery or resective surgery for a focal lesion or atrophic region unrelated to hippocampal sclerosis. These patients were included in the MR analysis to limit bias that occurs normally with a retrospective study.

Imaging Parameters

MR studies were performed on a 1.5-T magnet. All patients had axial and coronal long-repetition-time images with the following parameters: 2000–3000/20–30,80–100/1–2 (repetition time/echo time/excitations), 20- to 24-cm field of view, a matrix of either 128 × 256 or 256 × 256, and 3- to 5-mm section thickness with a gap of 0.9 to 3 mm. Twenty-seven patients had additional T1-weighted coronal images with parameters of 400–600/20/4, 128 × 256 matrix, 16-cm field of view, and 5-mm-thick contiguous sections. These were the optimal pulse sequences for imaging patients with epilepsy during the period from 1986 through 1989.

Surgery and Pathology

Patients with medial temporal lobe epilepsy had the hippocampus removed en bloc using the anteromedial temporal lobectomy with radical hippocampectomy procedure described by Spencer et al (6). Fifty-seven patients were found to have hippocampal sclerosis by pathologic analysis. Five of these patients had findings in addition to hippocampal sclerosis, the so-called dual pathology. Two patients had ipsilateral hippocampal tumors, one had a temporooccipital vascular malformation (and a second excisional surgery), one had an ipsilateral frontal lobe cyst (excised by a frontotemporal lobectomy), and one had adjacent meningeal fibrosis. Diagnostic criteria for hippocampal sclerosis consisted of both qualitative and quantitative hippocampal cell loss. A marked decrease of hippocampal neurons was detected visually by a neuropathologist in all 57 patients. Cell counting was performed by two investigators on 6-μm coronal paraffin sections stained with either Nissl or hematoxylin, using five consecutive sections from the anterior hippocampal body. Neuronal density (mean number of cells per cubic millimeter) was determined for each hippocampal subdivision, the cornu ammonis fields (CA1–CA4), and the dentate granular layer as described by Kim et al (22). Patient densities were compared with the mean density from 20 nonepilepsy control patients matched for age and sex. Percentage of total hippocampal neuronal density is expressed as:

$$(\mathrm{pCA1/cCA1} + \mathrm{pCA2/cCA2} + \mathrm{pCA3/cCA3} + \mathrm{pCA4/cCA4} + \mathrm{pDG/cDG})/n$$

where p indicates patient neuronal density; c, mean control neuronal density; DG, dentate granular layer; and n, number of hippocampal subdivisions available for quantitation. Percentage of total hippocampal neuronal density was calculated only if CA1, CA4, and at least one other hippocampal subdivision was available for counting. Two patients did not have adequate tissue available for quantitative studies, but pathologic examination revealed patchy loss of neurons and gliosis within the hippocampus. In the other 55 patients, percentage of total hippocampal neuronal density was decreased more than two standard deviations compared with controls, which is our criterion for defining hippocampal sclerosis (2). We prefer the term *hippocampal sclerosis* rather than *mesial temporal sclerosis*, because our basis for this entity is derived from hippocampal histologic data (23).

Postoperative seizure outcome was based on the classification scheme of Engel et al (24). Mean follow-up interval was 4.0 years after surgery, with a range of 0.6 to 6.1 years. Outcome could not be determined in two patients: one patient was lost to follow up; the other died 1 year after surgery. We defined successful postoperative outcome as Engel class I (seizure-free), class II (rare seizures), or class III (worthwhile improvement, as defined by greater than 90% reduction in seizure frequency).

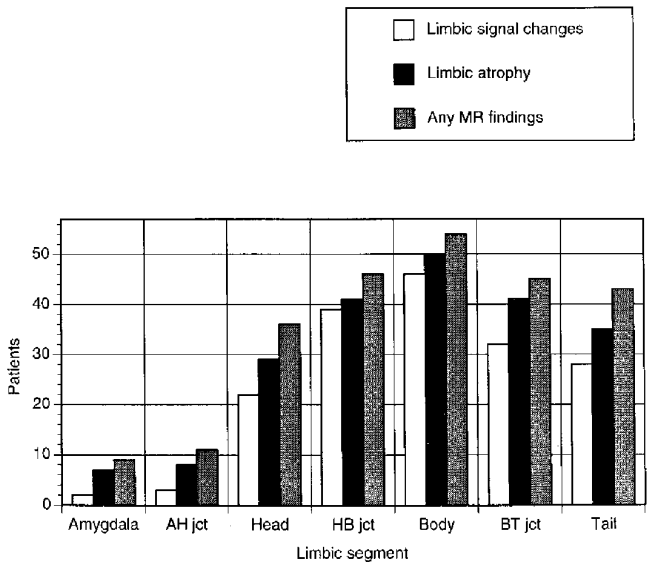


Fig 1. Regional MR findings in 57 patients with hippocampal sclerosis. "Any MR findings" represents either signal changes or atrophy occurring at that segment. *AH jct* indicates amygdala/hippocampal head junction; *head*, hippocampal head; *HB jct*, hippocampal head/hippocampal body junction; *body*, hippocampal body; *BT jct*, hippocampal body/hippocampal tail junction; and *tail*, hippocampal tail.

Results

Atrophy ipsilateral to the side of hippocampal sclerosis was seen in 52 of the 57 patients, occurring within the amygdala in 7, the hippocampal head in 29, the hippocampal body in 50, and the tail in 35. Ipsilateral hyperintense signal changes on long-repetition-time images was observed in 49 of the 57 patients, involving the amygdala in 2, the hippocampal head in 22, the body in 46, and the tail in 28, as seen in Figures 1 through 4. The limbic segment most frequently affected was the hippocampal body. Atrophic changes were seen slightly more frequently than signal abnormalities in each segment (Fig 1). However, signal changes also occurred in the absence of atrophic changes, as noted in Figure 1.

MR findings (either atrophy *or* hyperintensity) were observed in 55 of the 57 patients. The hippocampal body and an adjacent limbic segment (head, tail, or junctions) were affected in 50 patients (Fig 4). Twenty-one patients had abnormal MR findings distributed through the entire hippocampus. In nine additional patients with involvement of the entire hippocampus, MR findings extended anteriorly at least to the

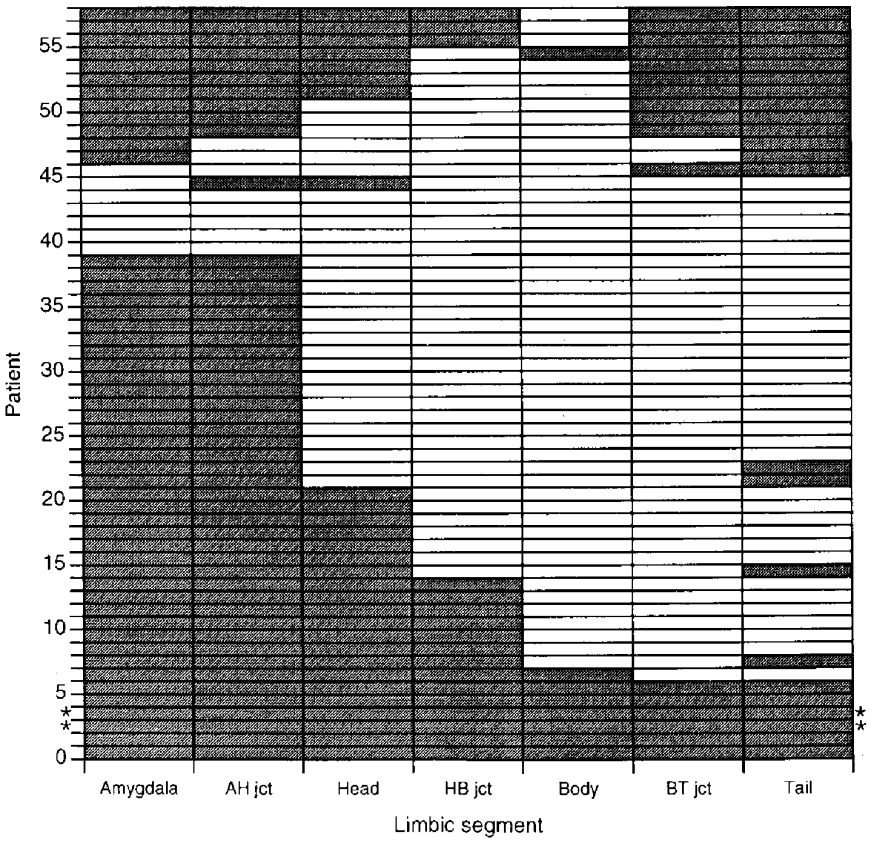


Fig 2. Limbic segments with atrophy. *White rectangles* represent atrophic limbic segments. Patient number was sorted by segmental distribution, thus patient number does not correspond to that in Figure 3 or 4. *AH jct* indicates amygdala/hippocampal head junction; *head*, hippocampal head; *HB jct*, hippocampal head/hippocampal body junction; *body*, hippocampal body; *BT jct*, hippocampal body/hippocampal tail junction; *tail*, hippocampal tail; and *asterisk*, ipsilateral tumor.

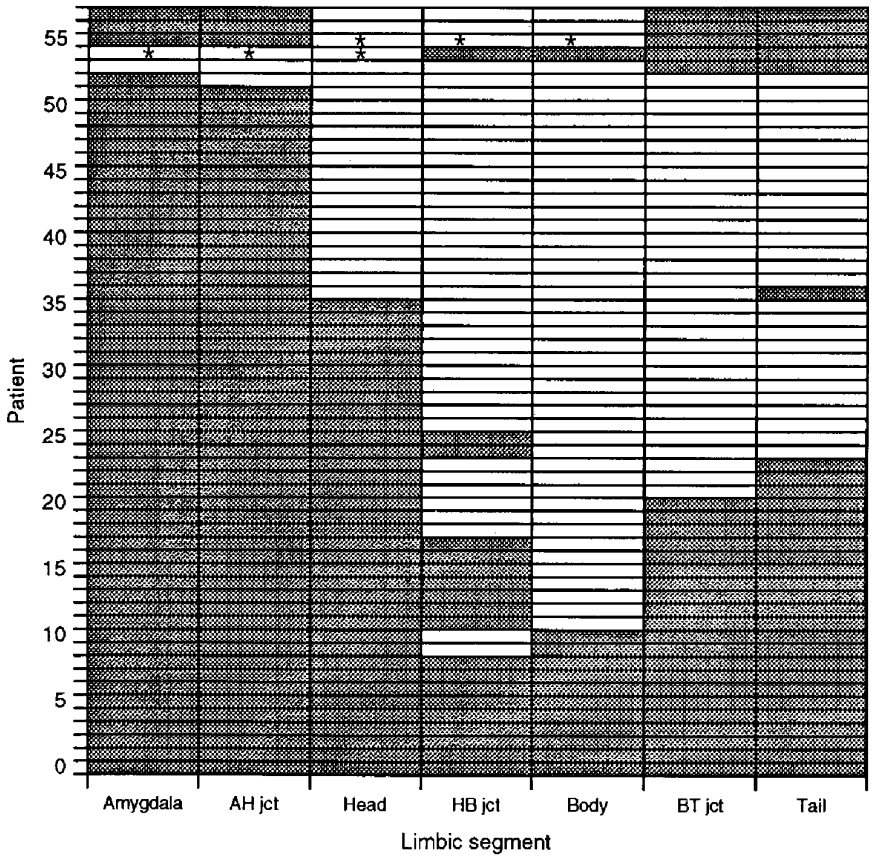


Fig 3. Limbic segments with hyperintense signal. *White rectangles* represent limbic segments that were hyperintense on long-repetition-time images. Patient number was sorted by segmental distribution, thus patient number does not correspond to that in Figure 2 or 4. *AH jct* indicates amygdala/hippocampal head junction; *head*, hippocampal head; *HB jct*, hippocampal head/hippocampal body junction; *body*, hippocampal body; *BT jct*, hippocampal body/hippocampal tail junction; *tail*, hippocampal tail; and *asterisk*, ipsilateral tumor.

hippocampal-amygdaloid junction (Fig 5). Thus, of the 57 patients, 30 had uniform involvement of at least the entire hippocampus, 25 had nonuniform involvement of limbic structures, and 2 had no imaging abnormalities. With the exception of 1 patient with an ipsilateral neoplasm, no patient had findings affecting the anterior limbic structures (amygdala or hippocampal head) without also affecting the hippocampal body. Atrophic or signal changes affecting the hippocampal body alone or in conjunction with hippocampal tail was seen in 8 cases (Fig 6).

A successful postoperative seizure outcome occurred in 52 (95%) of the 55 patients in whom follow-up data were available. Fifty patients were seizure-free (class I), 1 had rare seizures (class II), 1 had greater than 90% reduction in seizure (class III), and 3 had less than a 90% decrease in seizures postoperatively (class IV). Of those patients with dual pathology, 1 patient had a class IV outcome, 3 had class I outcomes, and 1 was undetermined because of premature death.

Of the 11 temporal lobe epilepsy patients who were found to not have hippocampal sclerosis at temporal lobectomy, 1 patient had questionable hippocampal signal changes, whereas another had questionable atrophic changes. Findings in both cases were thought to be normal variants and not diagnostic of hippocampal sclerosis by MR criteria.

Discussion

The syndrome of medial temporal lobe epilepsy associated with hippocampal sclerosis consists of a number of fairly well-defined attributes, but the pathogenesis of this disorder and the mechanism by which it causes seizures are still unclear. Patients with prototypical hippocampal sclerosis have predominantly unilateral temporal lobe seizures originating in the medial temporal lobe that can be treated successfully by temporal lobectomy. This syndrome is associated with hippocampal abnormalities including granule cell hyperexcitability, marked neuronal cell depletion, selective hilar interneuron loss, selective axonal sprouting, reorganization of neurotransmitter receptors, increases in second messenger and sodium pump systems, and MR evidence of atrophy and T2

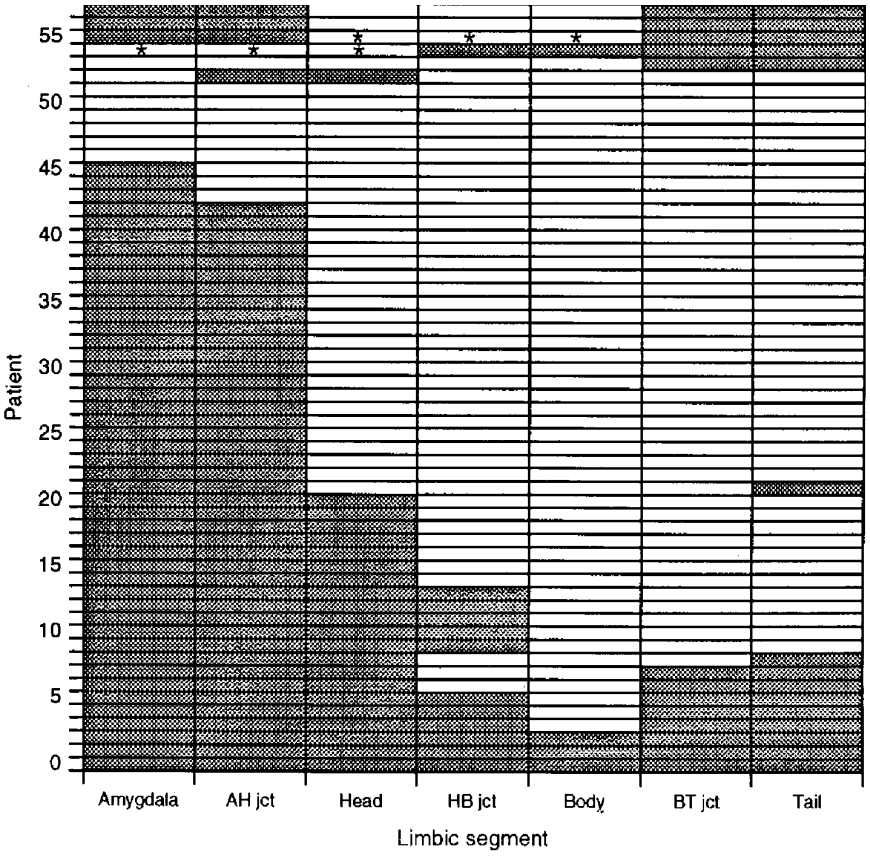


Fig 4. Limbic segments with MR abnormality (signal or atrophy). *White rectangles* represent limbic segments that were either hyperintense on long-repetition-time images or showed atrophy by MR. Patient number was sorted by segmental distribution, thus patient number does not correspond to that in Figure 2 or 3. *AH jct* indicates amygdala/hippocampal head junction; *head*, hippocampal head; *HB jct*, hippocampal head/hippocampal body junction; *body*, hippocampal body; *BT jct*, hippocampal body/hippocampal tail junction; *tail*, hippocampal tail; and *asterisk*, ipsilateral tumor.

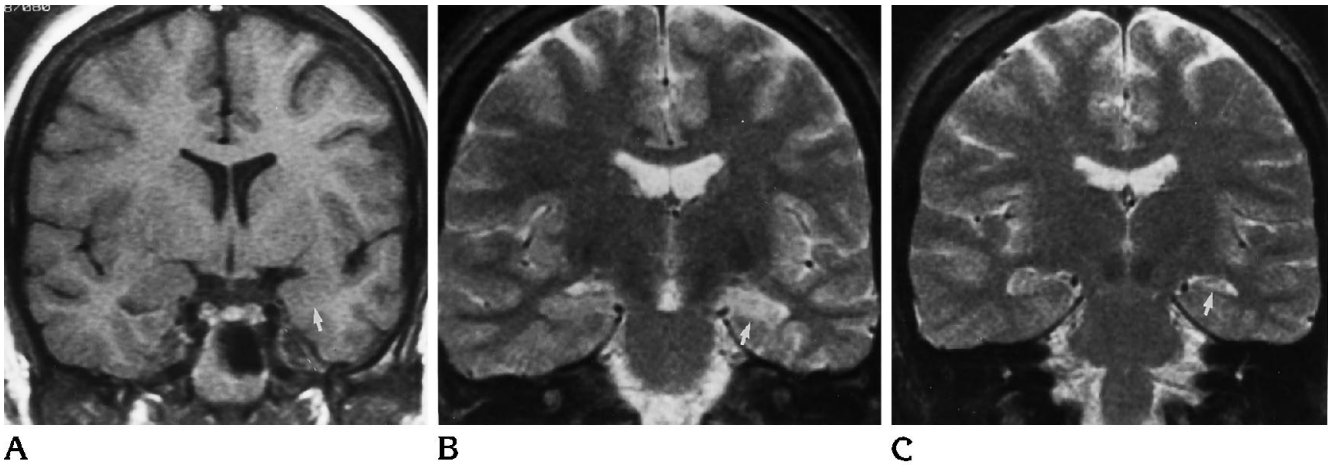
prolongation (23, 25–29). A large proportion of patients have had complicated febrile seizures in early childhood.

Some patients with medial temporal lobe epilepsy, however, do not have all the attributes associated with hippocampal sclerosis. Hippocampal sclerosis may represent the end stage of a number of different disorders. If it is the common final pathway for several entities causing epilepsy, then regional variations in its manifestations may help characterize these entities and offer insights into treatment. This concept is supported by studies indicating that the primary source of seizures may be the amygdala (3, 16), hippocampal head (1, 4, 8, 9), hippocampal body, hippocampal tail (6, 8), entorhinal cortex (4), or throughout these structures (8, 9). If the MR findings of regional limbic structure involvement are reflective of the epileptogenic zone, MR may help our understanding of the pathophysiology of this disorder and help tailor surgical approaches.

This study proves that signal and volume changes affect the full extent of hippocampus in most patients with hippocampal sclerosis. Twenty-one (37%) of 57 patients had MR changes (atrophy or signal changes) that involved the entire hippocampus. In 9 additional patients (16%), MR changes were distributed throughout the hippocampus with extension to the amygdala. Although nonuniform involvement of the hippocampus occurred in 25 (44%) of 57, 12 patients had changes that were widespread, affecting 4 of the 7 segments listed in Figure 4. Regional involvement limited to either anterior or posterior limbic segments was less common. MR abnormalities occurred in the anterior (amygdala-head-body) limbic segments in 5 (9%) of 57 and the posterior (body-tail) limbic segments in 8 (14%) of 57.

We also examined the frequency of limbic segment involvement and the relationship of atrophy to signal changes. The hippocampal body was the region most frequently affected, occurring in 54 (95%) of 57. Atrophy occurred more frequently than signal changes, but signal changes were found independent of atrophy. Jackson et al confirmed that hyperintense signal changes may occur without hippocampal atrophy (30).

Although several temporal lobe epilepsy studies suggest that MR hippocampal findings



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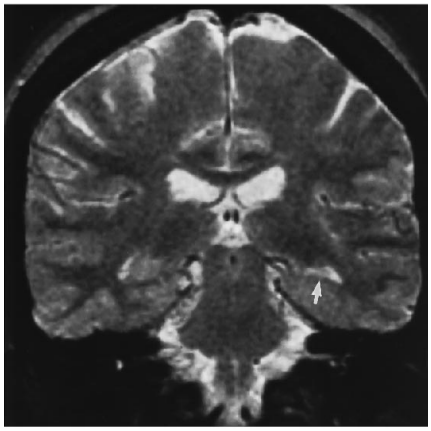


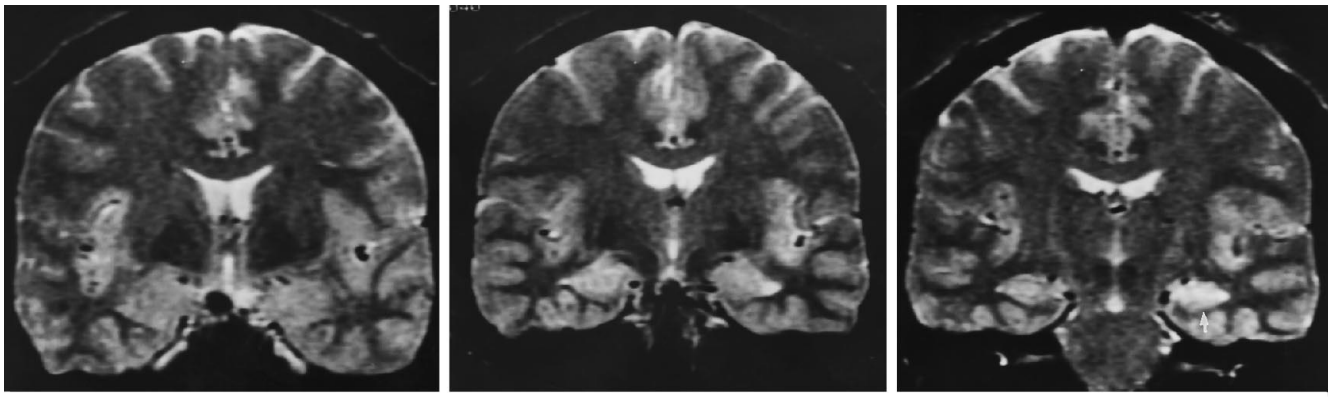
D

Fig 5. Short-repetition-time, short-echo-time coronal image at the amygdala (*A*), and long-repetition-time, long-echo-time images at the hippocampal head/body junction (*B*), body (*C*), and body/tail junction (*D*). Atrophy (*arrows*) is distributed from the amygdala to the hippocampal tail on the left side. Signal hyperintensity is marked at the hippocampal head/body junction. Ipsilateral temporal lobe atrophy also is present.

are not uniform, they did not formerly evaluate regional changes in limbic structures (13, 14, 16–18). One study by Cook et al (15) investigated regional limbic atrophy quantitatively in 20 nonsurgical patients with medial temporal lobe epilepsy. They found that atrophy occurred anteriorly in 12 patients, posteriorly in 1 patient, and throughout the limbic structures in 7 patients. The distribution of MR findings in our study differs from that of Cook et al. This difference may be explained in part by the differences in patient number, patient selection criteria, imaging criteria, and methodology (qualitative versus quantitative). Although our study lacks quantitative MR data, we tried to replicate routine clinical practice, in which MR investigation of temporal lobe epilepsy is done with visual assessment.

If these MR changes are related to the seizure generator, then regional involvement may have important implications for surgery. Because an anterior temporal lobectomy with a radical hippocampectomy (including the hippocampal tail) is performed at our institution in all patients suspected of having hippocampal sclerosis, our excellent outcome data may be attributable to removal of *all* medial temporal limbic structures. However, many different types of temporal lobe surgeries are performed for hippocampal sclerosis worldwide, with varying degrees of success. Limbic epilepsy surgery aimed at sparing the amygdala has been reported as successful in 79% (4), whereas surgery directed at removing the amygdala without hippocampus may be successful in 63% (3). Could outcomes be improved by surgery directed at hippocampus or amygdala, respectively, if involvement is found by MR? Several investigations have found that seizure elimination corresponded to the amount of medial temporal lobe resected, but correlation with preoperative segmental MR changes was not studied (5, 12, 31–33).

Segmental limbic changes also could affect quantitative MR measurements in patients with hippocampal sclerosis. Most quantitative volume measurements include the entire hip-



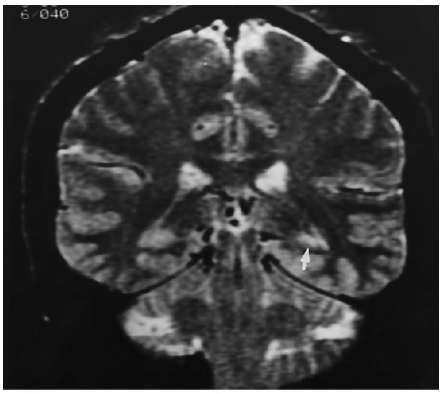


Fig 6. Long-repetition-time/long-echo-time images at the amygdala (*A*), hippocampal head (*B*), body (*C*), and tail (*D*). The left hippocampus was hyperintense from hippocampal body to tail (*arrows* in C and D), but only the tail was atrophic. The amygdala and hippocampal head had no abnormal MR findings.

pocampus (34, 35), although separate evaluation of the amygdala has recently been reported (15, 17, 18). Quantitative volume measurements of the entire hippocampus may obscure segmental involvement (15). Quantitative volume studies based solely on the hippocampal body also have been reported. The anatomic borders are easier to define, and these measurements are quicker to perform than whole limbic measurements. Hippocampal body measurements appear to be accurate and practical for most patients with hippocampal sclerosis (36, 37), which is confirmed by our data, but the anterior or posterior extent of disease may be missed. Quantitative T2 relaxation measurement is another sensitive method for diagnosing hippocampal sclerosis (25, 38). It is important that these measurements be performed at the level of a hippocampal body. Our data suggest that when signal abnormalities occur, part of the hippocampal body is involved in almost all cases.

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