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Temporal Lobe Epilepsy: The Various MR Appearances of Histologically Proven Mesial Temporal Sclerosis

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PURPOSE: To determine the frequency of appearance of various MR signs in mesial temporal sclerosis, to determine the optimal scanning planes for their visualization, and to propose a histologic explanation for the diminished demarcation between gray and white matter in the temporal lobe, a frequent MR finding in patients with mesial temporal sclerosis. **METHODS:** MR scans of 14 surgically treated patients with epilepsy and histologically proven mesial temporal sclerosis were assessed for the presence of six features: feature 1, high signal intensity in the hippocampus; 2, reduced hippocampal size; 3, ipsilateral atrophy of the hippocampal collateral white matter; 4, enlarged temporal horn; 5, reduced gray–white matter demarcation in the temporal lobe; and 6, decreased temporal lobe size. **RESULTS:** Feature 1 was present in 14 patients and was best appreciated on the T2-weighted images in planes parallel to the long axes of the hippocampi. Feature 2, present in 12 patients, and feature 6, present in 9 patients, were optimally seen in the coronal planes and on the inversion-recovery sequences in particular. Feature 3, present in 12 patients, was optimally seen on the coronal T2-weighted images. Feature 4, seen in 11 patients, was equally well seen in all planes (transverse, coronal, and parallel to the long axes of the hippocampi). Feature 5, seen in 10 patients, was best appreciated on the T2-weighted images in the planes of the long axes of the hippocampi. Histologic investigation of the temporal lobe white matter in the 10 patients with feature 5 demonstrated on the MR scan showed abnormalities in 7 cases. Oligodendroglia cell clusters were found in 6, with concomitant corpora amylacea in 1 case and perivascular macrophages with pigment a sole finding in another case. **CONCLUSION:** Of the six features found in cases of mesial temporal sclerosis on MR, increased hippocampal signal intensity is the most consistent. A decreased gray–white matter demarcation in the temporal lobe parenchyma is also a frequent feature of this disease. A combination of multiple scanning planes results in an optimal demonstration of lesions.

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

The purpose of our study was to investigate the incidence of different magnetic resonance (MR) features in histologically proven mesial temporal sclerosis and to investigate which imaging planes optimally demonstrate these lesions. We also sought a histologic explanation for the diminished gray–white matter demarcation on MR in the ipsilateral temporal lobes in patients with mesial temporal sclerosis.

Mesial temporal sclerosis is one of the most frequent findings in patients operated on for drug-resistant epilepsy. Thus it is important to demonstrate the lesion preoperatively with an imaging modality with high senstivity and specificity. The features of mesial temporal sclerosis on MR may be subtle. Previous studies (1–7) have reported an increased signal intensity in the hippocampus, a reduced hippocampal size,

an enlarged temporal horn, a decreased gray–white matter distinction in the temporal lobe neocortex, an atrophy of the parahippocampal collateral white matter, a decreased size of the temporal lobe, and a verticalization of the Sylvian fissure. All features except the last were assessed in this study.

Patients and Methods

The MR findings in 14 patients with histologically proven mesial temporal sclerosis were assessed in the present study. The group consisted of 4 men and 10 women with a median age of 32 years (range, 14 to 43 years). Patient selection criteria for surgical treatment consisted of intractable focal epilepsy, originating from one temporal lobe. Seizures had been resistant to multiple antiepileptic drug trials for at least 2 years.

All patients had been evaluated with prolonged interictal and ictal electroencephalographic recording, including sphenoidal recordings with video monitoring and neuropsychological investigations. If surface electroencephalographic recordings and the imaging studies did not suggest localization to one temporal lobe, intracranial electroencephalographic recordings were obtained with subdural and intracerebral electrodes (8).

MR images were made on a 1.5-T system using a regular head coil. The protocol included: (a) sagittal T1-weighted (750/30/1 [repetition time/echo time/excitations]) acquisitions; (b) axial T2-weighted (2000/50,100/1) acquisitions through the entire brains with a section thickness of 8 mm; (c) coronal images through the temporal lobes with a section thickness of 5 mm using a T2-weighted sequence and an inversion-recovery sequence (1537/30/1, inversion time 650); and (d) T2-weighted sequences (2000/50,100/2) in the axial planes through the temporal lobes, parallel to the long axes of the hippocampi, using 4-mm sections. A field of view of 225 mm^2 with a matrix of 256 × 256 was used for all images.

To minimize flow artifacts originating from the circle of Willis and projecting over the temporal lobes on the axial images, the phase-encoding gradient was in the anteroposterior direction. To maximize image quality and to increase the signal-to-noise ratio, the coronal T2-weighted images were made with a chemical shift of 6 pixels (instead of 2 normally used).

The MR images were reviewed by three radiologists with knowledge of the fact that the patients subsequently had temporal lobectomies but blinded for the electroencephalograms and surgical lateralization. The images were examined for six features: feature 1, increased signal intensity in the abnormal hippocampi on T2-weighted images; 2, reduction in size of the pathologic hippocampi; 3, atrophy of the white matter abutting the hippocampi in the parahippocampal gyri; 4, larger temporal horns on the abnormal sides; 5, altered gray–white matter demarcation in parenchyma of the affected temporal lobes; and 6, decrease in sizes of the ipsilateral temporal lobes. The radi-

ologists evaluated these signs in all planes and assessed whether the features occurred unilaterally or bilaterally. The final judgment was reached by consensus.

Histologic samples were obtained during temporal lobectomy from the hippocampus and the neocortical areas of the temporal lobe. The specimens were fixed in phosphate buffered 4% formaldehyde and embedded in paraffin. Histologic sections were stained with hematoxylin and eosin.

The histologic diagnosis of mesial temporal sclerosis was made on the basis of a decrease in the number of neurons in the CA3 or CA4 zones of the hippocampus with concomitant gliosis (9, 10).

Results

Mesial temporal sclerosis was histologically confirmed in all 14 patients.

Table 1 presents the abnormalities found on MR and the results of the histologic examination of the temporal lobe neocortical white matter in each patient. Table 2 shows the planes in which each feature was seen, as recorded using visual assessment. Figures 1 through 5 demonstrate the six different mesial temporal sclerosis findings on MR.

Six of the 10 cases with abnormal signal intensities in the hippocampi and decrease in gray–white matter demarcation had oligodendroglia cell clusters in the white matter of the temporal neocortices. One patient in this group also had perivascular macrophages with pigment in the temporal lobe white matter, and 1 patient had corpora amylacea in the neocortical specimen. However all 4 patients with increased signal intensities in the hippocampi, but without reduced demarcation, also showed oligodendroglia cell clusters.

Discussion

In the present study the MR scans of 14 patients with known histologically proven mesial temporal sclerosis were retrospectively reviewed. Although prospectively, before surgical treatment, 13 of these 14 patients were correctly diagnosed as having mesial temporal sclerosis on their MR scans, and 1 scan was then considered normal, a blinded study would be necessary to rule out the possible bias in the MR reviewing.

Mesial temporal sclerosis is a common histologic finding in patients undergoing surgery for temporal lobe epilepsy. It has been postulated that mesial temporal sclerosis may be related to a complicated delivery (11), febrile convulsions

TABLE 1: Number of patients, abnormal hippocampal side, MR features found per patient, and histologic findings in the temporal lobe white matter

| | Side | MR Features | | | | | | Histology |
		1	2	3	4	5	6	
Patient								
1	L	+	+	+	+	+	+	No abnormality
2	R	+	+	+	+	+	+	No abnormality
3	R	+	+	+[a]	+	+	+	OCC
4	R	+	+	+	+	+	−	OCC
5	R	+	+	+	+	+	−	OCC
6	R	+	+	+[a]	+	+	−	No abnormality
7	L	+	+	+[a]	+	−	+	OCC
8	L	+	+	+	+	−	+	OCC
9	L	+	+	+	+	−	−	OCC
10	R	+	+	+	−	+	+	OCC + CA
11	R	+	+	+	−	+	−	Limited OCC + PVM with pigment
12	L	+	+	−	+	+	+	OCC
13	R	+	−	+	−	+	+	No abnormality
14[b]	L	+	−	−	+	−	+	OCC
Total		14	12	12	11	10	9	

Note.—Feature 1 is high signal intensity in the hippocampus; 2, smaller size of hippocampus; 3, collateral white matter atrophy; 4, enlarged temporal horn; 5, diminished gray–white matter demarcation; and 6, smaller temporal lobe. OCC indicates oligodendroglia cell clusters; PVM, perivascular macrophages.
[a] Bilateral atrophy of collateral white matter.
[b] Decreased temporal lobe white matter volume.

during childhood (12, 13, 14), and status epilepticus (15). These circumstances are believed to cause metabolic disturbances in neurons in the hippocampus, which may disappear and subsequently be replaced by gliosis (15). This may lead to a changed signal intensity on MR and a reduction in size of the hippocampus.

Although MR has proved much more sensitive than computed tomography in the detection of mesial temporal sclerosis (16), the incidence of mesial temporal sclerosis on MR differs significantly between studies investigating patients with drug-resistant temporal lobe epilepsy (8% found by Brooks et al [2]; Gates and Rodriguez, 55% [3]; Heinz, 62% [4]; Dowd, 64% [5]; Kuzniecky, 70% [1]; and 93% in a study by Jackson et al [6]).

Various authors have proposed criteria for the diagnosis of mesial temporal sclerosis on MR (1–3, 7, 17). The optimal planes and sequences for the depiction of the hippocampus suggested by different authors (6) are the coronal T2-weighted images and the axial images parallel to the temporal fossa (18) or to the hippocampus using a T2-weighted sequence (6). The features assessed in our study and the optimal planes for their visualization will be discussed separately.

MR Features

1. Increased Signal Intensity in the Hippocampus (Fig 1). This MR criterion in the pathologic hippocampus is the most common

TABLE 2: Number of times the six criteria, used in the diagnosis of mesial temporal sclerosis on MR, were noted on the different planes and sequences

| | MR Features | | | | | |
	1	2	3	4	5	6
Axial T2	9/14	2/12	. . .	7/11	0/10	0/9
Coronal T2	8/14	9/12	12/12	6/11	5/10	6/9
Coronal inversion-recovery	0/14	12/12	11/12	8/11	0/10	8/9
T2 parallel to the long axis of the hippocampus	10/14	3/12	. . .	6/11	10/10	5/9

Note.—Feature 1 is high signal intensity in the hippocampus; 2, smaller size of hippocampus; 3, atrophy of the collateral white matter; 4, enlarged temporal horn; 5, diminished gray–white matter demarcation; and 6, smaller temporal lobe.
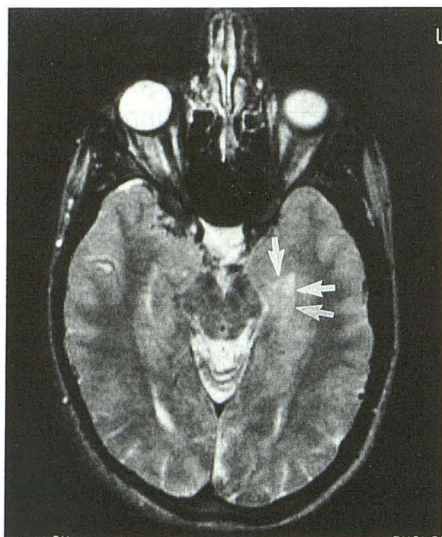
Fig 1. Case 14. Axial spin-echo 2000/100 image angulated parallel to the long axis of the hippocampus. A slightly increased signal intensity is present in the left hippocampus (*arrow*).

finding in this study. It was seen in all 14 cases. Kuzniecky et al (1) found high signal in 71% of patients with severe gliosis and in 50% of patients with moderate gliosis. They state that in most cases the severity of pathologic changes corresponded to the intensity of the abnormal signals on T2-weighted images. However, in some cases no agreement was found, and the location of the abnormal signal intensity did not always correlate with the distribution of the histologic changes (1). Heinz et al (4) found increased signal intensity in the mesial structures of 62% of patients with mesial temporal sclerosis using transverse (angulated along the temporal horn) and coronal T2-weighted images. Berkovic (12) found in all 10 presented patients increased hippocampal signal suggestive of mesial temporal sclerosis, using transverse T2-weighted images in planes parallel to the floors of the middle cranial fossae and coronal T2-weighted images.

The plane parallel to the long axis of the hippocampus used in our study provided good visualization of this feature, which is in agreement with Jackson et al (6). A pitfall encountered in the plane parallel to the long axis is caused by cerebrospinal fluid in the choroid fissure, which presents as a triangular area of increased signal intensity on the long-repetition-time long-echo-time sequence in the area of the head of the hippocampus. This area is usually located one section cranial to the hippocampus on the im-

ages in the plane parallel to the long axis with a section thickness of 4 mm.

Another pitfall in the plane parallel to the long axis may be caused by a skewed position of the patient's head, resulting in a partial volume inclusion of cerebrospinal fluid in the temporal horn, cranial to the hippocampus.

Contrary to the suggestions of other authors (2, 4, 5, 19), the coronal T2-weighted images in our study seemed to be less sensitive for this feature (it was noted in 7 of 12 cases), probably because of flow artifacts originating from the circles of Willis and projecting over the temporal lobes. Cardiac gating has been suggested as a means to reduce these artifacts (5) but was not used in our study. A caudal-cranial phase-encoding direction, wraparound suppression, and a caudally placed presaturation slab may provide another option to improve the image quality. This is time consuming and was not used in our study.

The standard axial thick-section image seemed equally sensitive in the detection of this feature; it was seen in nine patients. It must be emphasized that this is the case only when the phase-encoding gradient is in the anteroposterior direction.

2. Reduction in Size of the Hippocampus (Figs 2 and 3). The decreased hippocampal size was the next most common finding (12 of 14 patients). From Table 2 it seems that this criterion may be best assessed on the coronal images, the inversion-recovery sequence being slightly better than the others. However, care has to be taken to obtain the images in an exact coronal plane, if necessary, using images angulated along the left-right axis as well as the caudal-cranial axis. The axial plane is inferior to the coronal plane for the depiction of this feature. This is in agreement with the findings of Berkovic et al (12).

The reduced hippocampal size would be expected to be present in all cases, as a result of scarring and retraction. However, it was not seen in all of our patients. Jack et al (20) used a volumetric technique to assess the hippocampi in patients with drug-resistant temporal lobe epilepsy. Of 40 patients with unequivocal lateralization, this technique showed the abnormal hippocampus in 85%, was indeterminate in 12.5%, and was incorrect in 2.5%. Mesial temporal sclerosis was considered present if the sole finding was a smaller hippocampal volume.
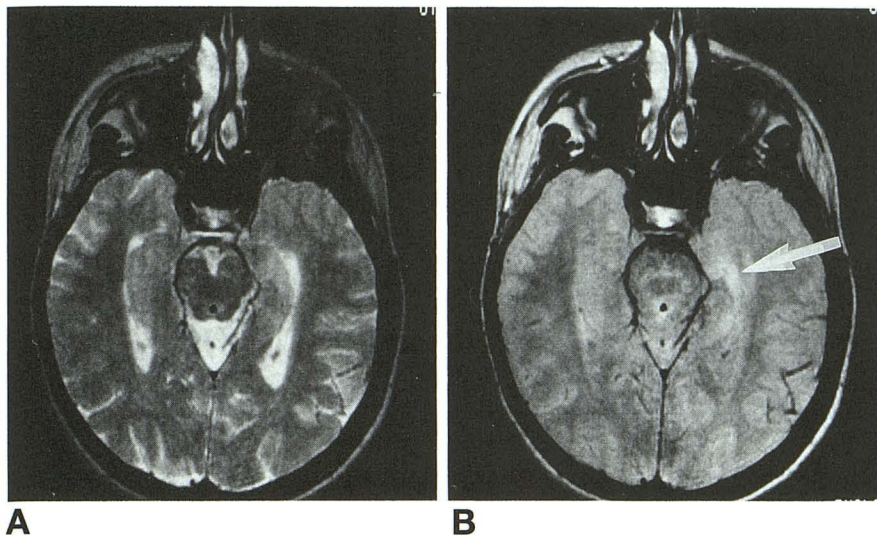
Fig 2. Case 2. A, Spin-echo 2000/100 image angulated parallel to the long axis of the hippocampus demonstrates a high signal in a smaller left hippocampus with an enlarged temporal horn.

B, Typical comma-shaped high signal in the left hippocampus (*arrow*) on the first echo of the T2-weighted spin-echo 2000/50 image angulated parallel to the long axis of the hippocampus.

Using three-dimensional fast low-angle shot imaging and computerized volume measurement of the hippocampi in patients with chronic epilepsy of the temporal lobe, Ashtari et al (21) also found that unilateral temporal lobe seizures were accompanied by significant reductions in hippocampal volume ipsilateral to the seizure focus. In our study all patients with decreased hippocampal size showed concomitant ipsilateral increased signal on T2-weighted images.

3. Atrophy of Collateral White Matter Adjacent to the Hippocampus (Fig 4). Bronen et al

(7) first described this finding on MR in six of eight patients with high signal in the hippocampus and suggested this finding to be a possible MR correlate of sclerosis that extends pathologically beyond the hippocampal gray matter. In our study the distinction between the collateral white matter and the abutting gray matter is diminished on coronal T2-weighted images. The inversion-recovery sequence demonstrates a local reduction of the volume of the adjacent collateral white matter; however, it is well demarcated from the neighboring gray matter (Fig 4).
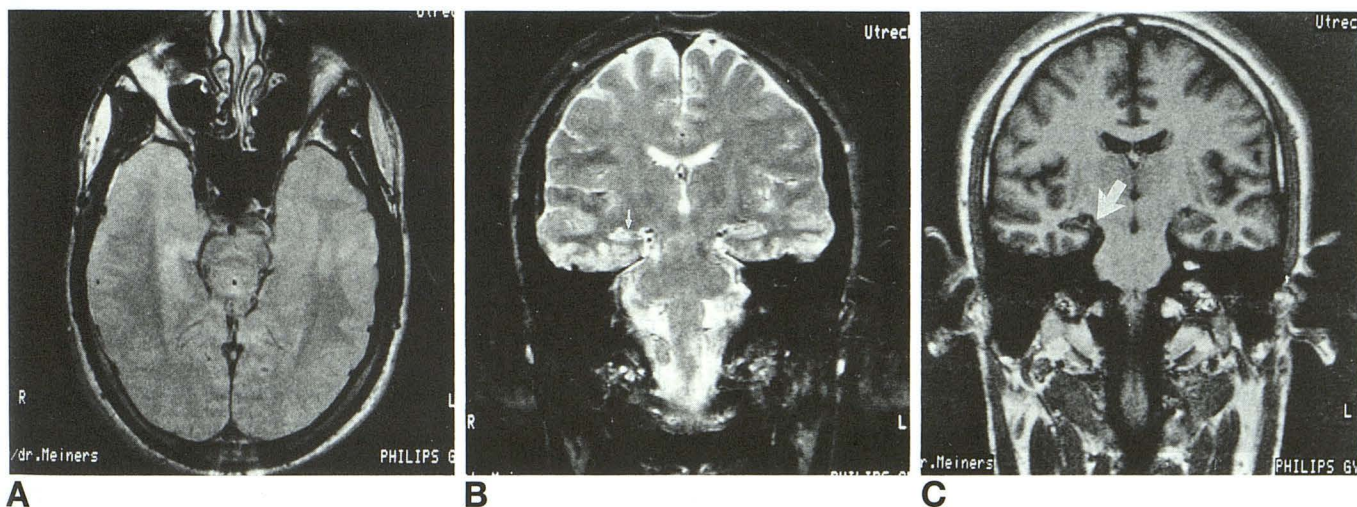

Fig 3. Case 5. A, Spin-echo 2000/50 image angulated parallel to the long axis of the hippocampus demonstrates a typical comma-shaped high signal in the right hippocampus, with a diminished gray–white matter demarcation in the right temporal lobe (*arrow*).

B, The coronal spin-echo 2000/100 image confirms the decreased hippocampal size and the high signal in the right hippocampus (*arrow*).

C, Coronal inversion-recovery 1537/575/30 image demonstrates the decreased size of the hippocampus more clearly than the the coronal T2-weighted image in B. The choroid fissure is indicated with an *arrow*.

Fig 4. Case 1. Coronal spin-echo 2000/ 100 image demonstrates collateral white matter adjacent to the hippocampus (*arrows*). The collateral white matter on the left (*L*) is atrophic. A coronal inversion-recovery 1537/575/30 image in this case also shows the left (*L*) collateral white matter to be atrophic (*arrow*).

A   **B**
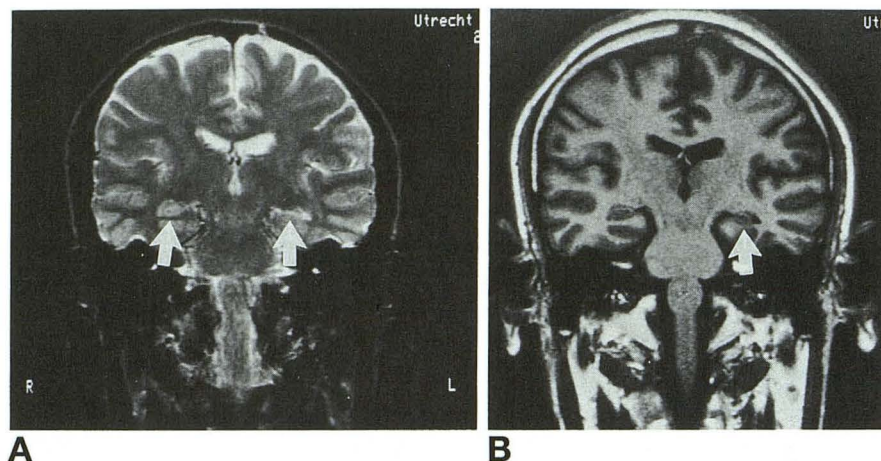
In our opinion this feature may be subject to erroneous judgment because of partial volume effect of gray matter in sulci or in the case of asymmetry of the sulci adjacent to the hippocampus, resulting in an asymmetry of the volume of collateral white matter. Also, a rotated position of the patient's head may influence this finding. These explanations may account for the bilateral collateral white matter atrophy found in three of our cases (patients 3, 6, and 7 in Table 1). This also leads us to advocate the use of this feature only in combination with the other criteria for mesial temporal sclerosis.

4. Enlargement of the Temporal Horn (Fig 5). Based on findings in our 14 patients, we think that the criterion of a larger temporal horn is relevant for the diagnosis of mesial temporal sclerosis only if the ipsilateral hippocampus is smaller. If the hippocampi are symmetrical in size and signal intensity, an enlarged temporal horn may be considered a normal variant or the result of loss of temporal lobe parenchyma from causes other than mesial temporal sclerosis. McLachlan et al (22) found temporal horn size to have a lack of predictive value for location of the seizure focus and even for diagnosis of temporal lobe epilepsy. Both healthy control subjects and patients with multiple sclerosis had enlarged temporal horns, usually asymmetric and sometimes larger than those seen in patients with temporal lobe epilepsy (22, 23).

The enlarged temporal horn was well appreciated in all planes and on T2-weighted and inversion-recovery sequences, the latter being slightly better. It was present in 11 of 14 patients. This high incidence in our patient group is possibly related to the age of the patients.

Kuzniecky et al (24) suggest that hippocampal atrophy with enlargement of the temporal horn may be progressive. In their study 2 patients younger than 10 years of age in a group of 12 patients did not show as much atrophy as the rest of the group of pediatric patients older than 10 years of age. They suggest that a longitudinal MR study will be necessary to clarify this issue.

5. Diminished Gray–White Matter Demarcation in the Temporal Lobe (Figs 5 and 6). In the present study the reduced distinction between gray and white matter in the temporal lobe was a frequent finding in combination with a pathologic hippocampus (10 of 14 cases). It was seen in the planes parallel to the to the long axes of the hippocampi in all 10 cases and 5 times in the coronal planes. The sensitivity of the coronal plane may be reduced because of flow artifacts.

In all 10 cases the T2-weighted images show blurred gray–white matter borders in the pathologic temporal lobes in their anterior regions. An interesting observation is the absence of this abnormality on the inversion-recovery images. These may serve as "normal" images for comparison (Figs 5C and D). The underlying cause of the altered gray–white matter distinction is not clear.

In 1964 Falconer et al (10) suggested that the process of sclerosis, although mainly affecting the mesial gray matter in the temporal lobe, may spread widely throughout the lobe, leading to a generalized atrophy and gliosis of both cortex and white matter; possibly the reduced gray–white matter demarcation on MR is the imaging correlate of this histologic finding.
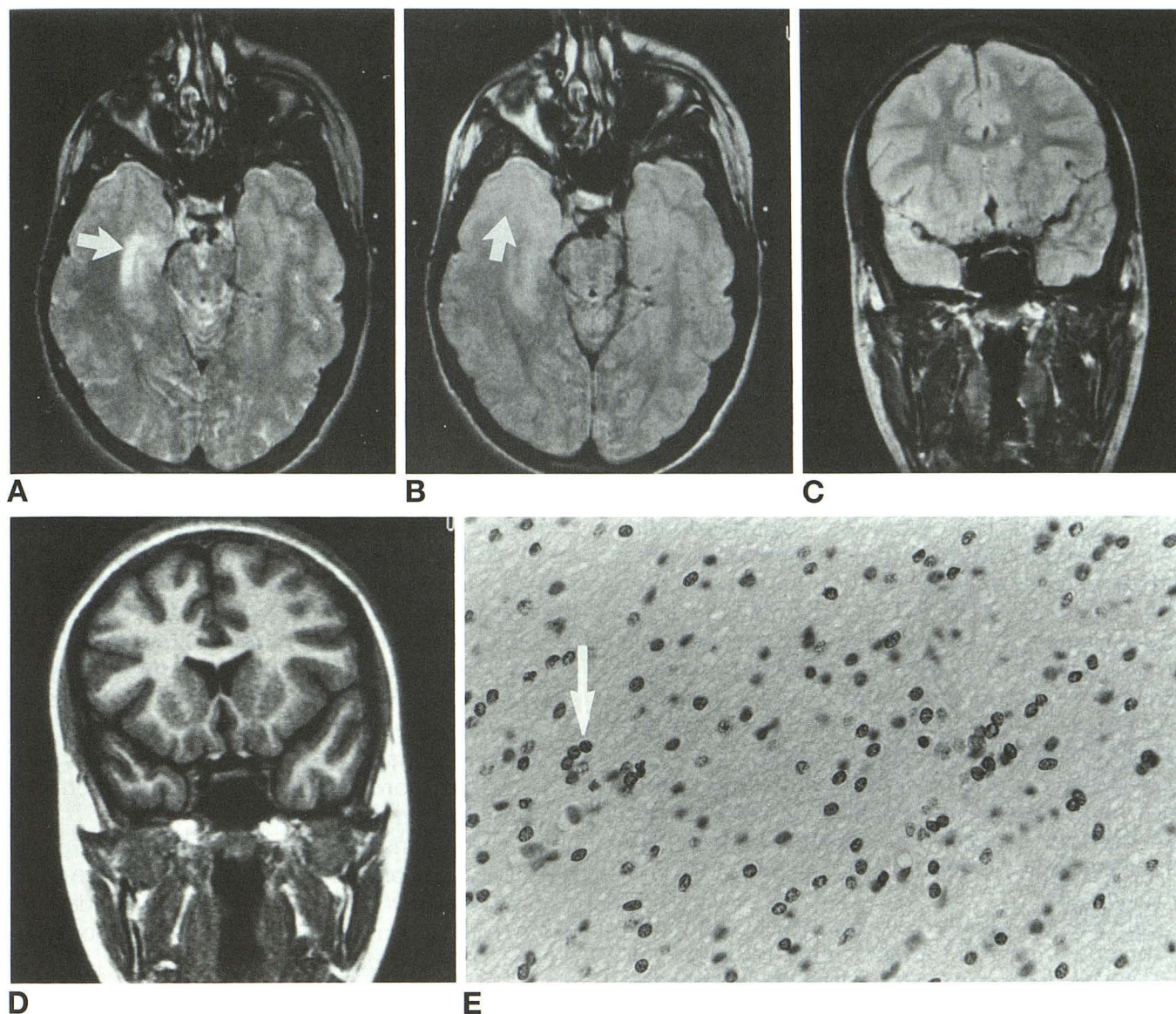
Fig 5. Case 4. *A*, Spin-echo 2000/100 image angulated parallel to the long axis of the hippocampus shows a high signal intensity in a smaller hippocampus with an an enlarged temporal horn on the right (*arrow*), with a decrease in gray–white matter distinction in a smaller temporal lobe.

B, The diminished gray–white matter demarcation is better seen on the first echo of the T2-weighted spin-echo 2000/50 image (*arrow*).

C, Coronal spin-echo 2000/50 image shows diminished gray–white matter demarcation in the right temporal lobe compared with the left.

D, On a coronal inversion-recovery 1537/650/30 image with a similar position, the assymetry between the gray–white matter demarcation in the temporal lobes is not appreciated.

E, Histologic specimen of the temporal lobe white matter shows an increased number of oligodendroglia cell clusters (*arrow*) (hematoxylin and eosin staining, original magnification 9.3×).

Froment et al (25) believe this finding suggests extensive gliosis; Jackson et al (6) found corpora amylacea in one patient with this feature, similar to one of our patients. However, corpora amylacea may develop after the use of depth electrodes as a result of damage to brain tissue.

Kuzniecky et al (26) recently described one case with cortical dysplasia showing a diminished gray–white matter demarcation; the remaining six of their seven cases with cortical dysplasia showed high signal intensities on the T2-weighted images in gray and/or white matter of the temporal lobes.

Fig 6. *A*, Spin-echo 2000/50 image angulated parallel to the long axis of the hippocampus demonstrates a reduced distinction between gray–white matter in a smaller left temporal lobe in a patient without hippocampal sclerosis (case not included in Table 1).

B, The diminished gray–white matter demarcation is confirmed on the coronal spin-echo 2000/50 image. The histologic section resembled the white matter seen in Figure 5E.

A   B
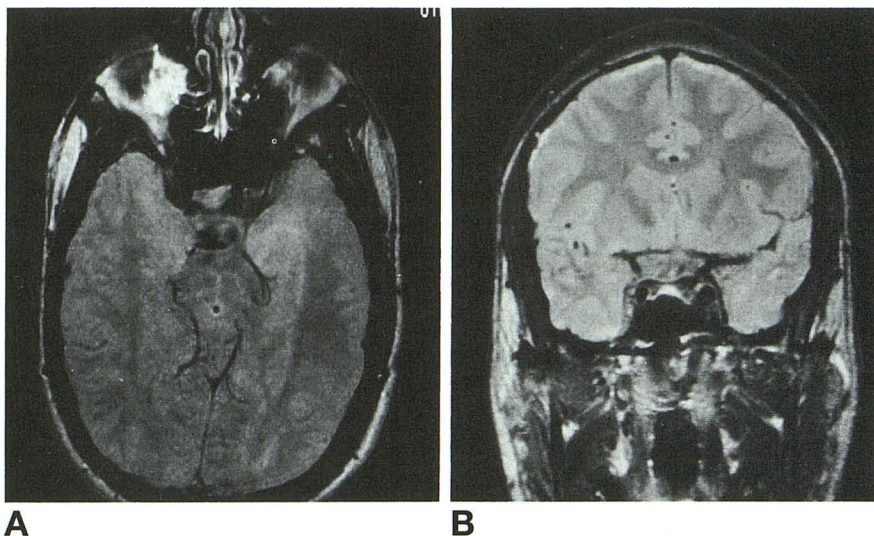
In our study the search for oligodendroglia cell clusters in the histologic specimens of the temporal lobes was initiated by the assessment of a patient operated on for intractable temporal lobe epilepsy, not included in the present study of 14 patients. On MR this patient presented with a decrease in gray–white matter distinction in a smaller temporal lobe, without a signal intensity increase in the hippocampus. Histologic examination of the temporal lobe white matter showed a marked increase of oligodendroglia cell clusters similar to that seen in Figure 5E. The histologic specimen of the hippocampus was normal.

Oligodendroglia cell clusters were also found in the specimens of patients with increased signal in the hippocampi but without decrease in gray–white matter distinction. Furthermore, oligodendroglia cell clusters are frequent findings in normal temporal lobe tissue at autopsy. Whether the finding of increased oligodendroglia cell clusters is relevant in patients with mesial temporal sclerosis with decreased gray–white matter demarcation, and whether the frequency differs from that of patients without epilepsy, remains to be investigated.

Possibly the altered gray–white matter demarcation may not be seen with the normal hematoxylin and eosin staining, or it reflects a chemical change in the white matter, rather than a structural variation. Further investigation using a combination of chemical shift imaging and chemical spectroscopic studies may clarify this issue.

The relevance of the temporal lobe reduced gray–white matter distinction may be twofold:

1. The area of hypometabolism on the 18-F-labeled deoxyglucose positron emission tomography scan is generally found to be larger than the structural lesion on MR (8, 27, 28). This extrahippocampal MR finding has not been consistently used as a criterion in the diagnosis of mesial temporal sclerosis. Whether a correlation exists between the extension of the positron emission tomography abnormality and the temporal lobe tissue changes on MR requires further evaluation.

2. The extension of suggested structural abnormality beyond the boundaries of the hippocampus may be relevant for the surgical approach (ie, a temporal lobectomy as opposed to an amygdalohippocampectomy).

6. Reduction in Size of the Temporal Lobe (Figs 5 and 6). In this study the criterion of a reduced size of the temporal lobe was not considered an indication of mesial temporal sclerosis if it was a solitary finding. It was then considered to signify hypoplasia in accordance with a congenital abnormality. It has been noted in other studies of patients with epilepsy (20), but its significance is not clear. Jack et al (29) have measured the volumes of temporal lobes in patients without epilepsy and came to the conclusion that the left temporal lobe generally is slightly but significantly larger than the right temporal lobe. This also should be taken into account when the difference in size is used as a criterion for mesial temporal sclerosis.

In one patient with symmetrical demarcation of gray–white matter, the total amount of white matter was decreased in the pathologic tempo-

ral lobe, probably accounting for the reduced temporal lobe size (case 14 in Table 1).

Reduced temporal lobe size was noted in 9 of 14 patients and was best appreciated on the coronal inversion-recovery images. However, care must be taken to avoid oblique coronal images caused by a rotated position of the patient's head, with resultant asymmetry of the temporal lobes.

Mesial temporal sclerosis may have different appearances on MR images, resulting in a combination of six features, of which an increased hippocampal signal intensity is the most consistent finding. The subtlety of the abnormalities of mesial temporal sclerosis on MR and the associated artifacts and pitfalls in the different scanning planes necessitate the use of multiple planes for confirmation. Decreased gray–white matter demarcation in the temporal lobe parenchyma seems to be a common feature in patients with mesial temporal sclerosis. This may be related to increased oligodendroglial clusters seen in temporal lobe white matter on pathologic specimens obtained from these patients.

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