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Detailed neurologic studies, high-field-strength MR imaging, and CT scanning were performed preoperatively in 53 patients with intractable complex partial seizures who underwent surgical treatment for epilepsy. Macroscopic structural (tumoral or vascular) lesions were found in 28% of patients. The remainder had pathologic findings consistent with mesial temporal gliosis. Tumors were found in 22% of the patients and were benign or of low-grade malignancy in every case. MR was accurate in the preoperative diagnosis of structural lesions, including very small occult tumors and cryptic vascular malformations. In patients with mesial temporal gliosis, there was correlation between the MR observation of a unilaterally dilated anterior temporal horn and the EEG-identified seizure focus and side of temporal lobectomy. However, MR demonstrated T2-weighted signal abnormalities correlating with the epileptogenic focus in only 8% of cases of mesial temporal gliosis.

MR provided useful information in 28% of patients who underwent surgery for refractory complex partial epilepsy. MR obviated invasive EEG monitoring in 93% of the patients with structural lesions. MR was useful in only 8% of the patients with pathologic changes of mesial temporal gliosis.

Previous reports have evaluated the contributions of low- and mid-field-strength MR imaging in patients with partial epilepsy and have compared the diagnostic efficacy of MR and CT [1–6] or of MR, CT, and positron emission tomography [7–9] in these patients. The efficacy of MR performed at a high field strength vs CT in a group of epilepsy patients, including a subgroup of 26 surgical cases of complex partial seizures, was reported recently [10]. We report the results of preoperative high-field-strength MR examinations in 53 patients who underwent surgery for intractable complex partial seizures; surgical and pathologic correlations were available for each case.

Materials and Methods

Fifty-three patients with complex partial seizures refractory to medical management underwent surgery during the period between October 1985 and October 1988. The patients were 7–54 years old (average, 26 ± 12) and included 23 males and 30 females. The average duration of seizure disorder was 18 years (range, 1–52 years). Pertinent medical history in the subgroup of 38 patients with mesial temporal gliosis included seizure disorders that began with a clinically documented episode of high fever and febrile seizures in two patients and a history of meningoencephalitis in three other patients whose seizures began within the first decade. There was a history of perinatal intracranial hemorrhage in one patient in whom seizures began at age 16. Finally, there was a history of severe head trauma in four other patients. Seizures began in late childhood in one of these four patients, in whom a subdural hematoma had been evacuated at age 18 months. This was the only patient in this series in whom cranial surgery had been performed previously. In two patients, seizures began 2 and 12 months after closed head injury, at ages 7 and 16 years, respectively. In the fourth patient,

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a head injury with associated loss of consciousness and no other sequelae occurred at age 5; complex partial seizures began at 37 years of age.

Preoperative evaluation in all patients included history and physical examination, neuropsychological testing, continuous video and EEG monitoring with scalp and sphenoidal electrodes, and angiography with intracarotid amobarbital sodium (Wada) testing. Invasive EEG monitoring was required for definitive localization of the seizure focus in 41 of the patients; this was achieved with depth electrodes in 39 patients and with multicontact subdural grid electrodes in two children. Invasive EEG monitoring was omitted when obvious structural lesions were seen on neuroimaging studies or the noninvasive presurgical assessment was already definitive for a unilateral ictal focus.

Patients with a seizure focus in the vicinity of a functional area underwent craniotomy under local anesthesia with awake stimulation mapping of local functioning areas. The surgical technique in all cases consisted of the subpial method of resection. En bloc resection of several centimeters of the anterior hippocampus was also included in all anterior temporal lobectomies whenever either presurgical or intraoperative testing confirmed that the opposite mesial temporal structures would support recent memory function. The extent of resection was guided by intraoperative electrocorticography in all patients.

Cranial CT without and with IV contrast enhancement was performed on a General Electric 9800 scanner in 48 patients. Standard transaxial nonenhanced scans were obtained with contiguous 10-mm sections. Contrast-enhanced CT was performed by using a modified technique with gantry angulation to obtain optimal temporal-lobe views [11]. Section planes parallel to the long axis of the temporal lobe were obtained with 10 contiguous 3-mm slices; the remainder of the brain was scanned with 10-mm-thick sections at 10-mm intervals.

MR studies were performed in all patients on a 1.5-T superconducting magnet (Signa, General Electric Medical Systems, Milwaukee, WI). Sagittal T1-weighted images, 800/20/4 (TR/TE/excitations), were acquired with a 24-cm field of view, 3-mm slice thickness with 0.6-mm interslice gap, and 256 × 256 acquisition matrix. Intermediate and T2-weighted cardiac-gated coronal and transaxial images, 2000/20,70/1, were acquired by using a 5-mm slice thickness with 2.5-mm interslice gap and 256 × 256 acquisition matrix. The field of view was 24 cm for the transaxial and 20 cm for the coronal images. Approximately one half of the MR studies performed before August 1987 did not have cardiac gating; in these cases the number of excitations used was two. In obtaining the transaxial images, an attempt was made to position the patient with the chin up so as to correspond as closely as possible to the transaxial temporal-lobe CT images. The imaging parameters provided pixel dimensions of approximately 1 × 1 mm in the imaging plane.

MR interpretation was performed without knowledge of the EEG findings; in the majority of patients, MR was performed before admission for electrophysiological monitoring. CT also was performed either on an outpatient basis shortly before admission or on the day of admission before beginning the EEG studies. All patients had surgery after definitive identification of a unilateral seizure focus; pathologic correlation was available in every case.

Results

The resected tissue showed important pathologic changes in all patients. Fifteen of the 53 patients had tumors or vascular malformations (Table 1). Thirty-eight patients had pathologic changes consistent with mesial temporal gliosis

(neuronal degeneration and loss, neuronal heterotopia, and fibrillary astrocytic proliferation).

Tumor histology is summarized in Table 2. Benign or low-grade malignant tumors were present in all 12 cases. Tumors were located in the temporal lobe in eight of 12 cases (Figs. 1–3). One patient had a ganglioglioma in the right posterior frontal operculum. One patient with tuberous sclerosis had a giant cell astrocytoma in the right posterior frontal region (Fig. 4). One patient had a juvenile fibrillary astrocytoma predominantly in the occipital lobe (Fig. 5) and one patient had a grade II astrocytoma in the right putamen and insula (Fig. 6). Vascular malformations were found in three patients. One patient had a left temporal thrombosed arteriovenous malformation (Fig. 7) and one a left temporal and another a right hippocampal cavernous hemangioma.

Preoperative MR was correct in the diagnosis of tumor in 11 of 12 cases and suggested either posterior cerebral infarct or low-grade tumor in the remaining patient with occipital juvenile fibrillary astrocytoma (Fig. 5). MR correctly diagnosed the three patients with vascular malformations. Thus, MR diagnosis was specific for diagnoses of tumor or vascular malformation in 14 (93%) of 15 patients with structural lesions and showed abnormal signal changes in all 15.

CT scans without and with contrast enhancement were available in 10 of the 15 patients with tumors and vascular malformations. CT correctly diagnosed low-grade glioma in two of four patients with gangliogliomas and was unavailable in the other two cases with this diagnosis. In the patient with a right putaminal and insular astrocytoma, the CT scan was interpreted as tumor or vascular malformation. CT missed the diagnosis of tumor in one patient each with oligodendroglioma, giant cell astrocytoma, and benign fibrillary astrocytoma. In the patient with occipital juvenile fibrillary astrocytoma, CT favored the diagnosis of infarct. In the three patients with vascular malformations, CT diagnosis of a calcified vascular

TABLE 1: Surgical Findings in 53 Patients with Intractable Complex Partial Seizures

Finding	No. (%)
Tumors	12 (22)
Vascular malformations	3 (6)
Pathologic changes of gliosis	38 (72)
Total	53 (100)

TABLE 2: Tumors in 12 Patients with Intractable Complex Partial Seizures

Tumor	No.
Ganglioglioma	4
Benign fibrillary astrocytoma	3
Fibrillary astrocytoma grade II/IV	2
Oligodendroglioma	1
Juvenile fibrillary astrocytoma	1
Giant cell astrocytoma	1
Total	12

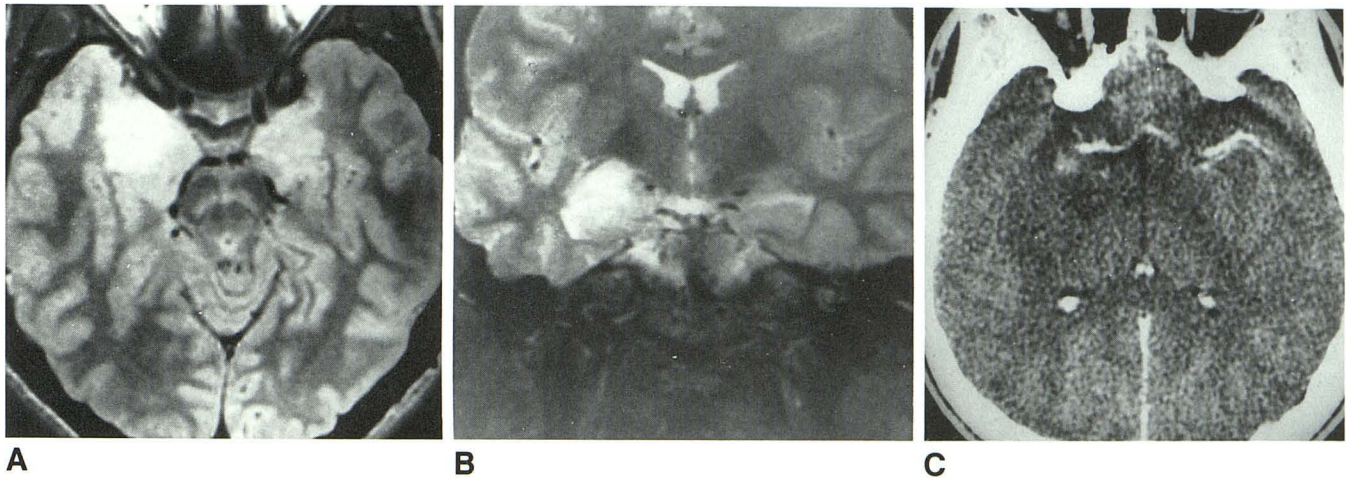**A**   **B**   **C**

Fig. 1.—Right temporal ganglioglioma in a 19-year-old woman.
A and B, Intermediate-weighted transaxial (A) and T2-weighted coronal (B) MR images show abnormal high signal of tumor anterior and superior to temporal horn.
C, Focal small abnormal tumoral enhancement on transaxial CT might not have been apparent without use of angled temporal-lobe CT technique.
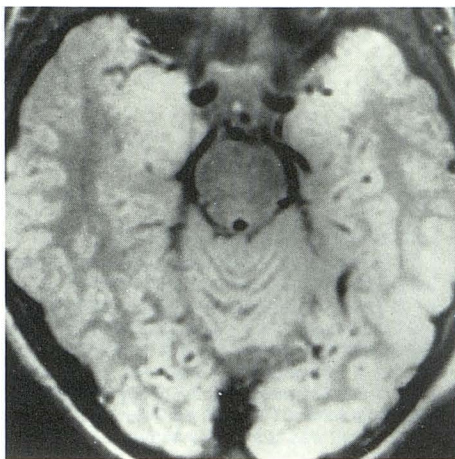

Fig. 2.—Oligodendroglioma in an 11-year-old boy. Intermediate-weighted transaxial MR image shows right mesial temporal abnormal high signal involving gray and white matter diffusely. Pathologically, tumor was cellular without calcification and involved all of the amygdala and uncus with extension into hippocampus.

malformation was made in one case (Fig. 7) and the diagnosis of vascular malformation or vascular tumor was suggested in the other two cases. CT was therefore "positive" for a structural lesion in seven of 10 cases, providing a specific diagnosis of tumor or vascular malformation in three of these and a false-negative interpretation in three patients with tumors.

Pertinent CT and MR findings in patients with mesial temporal gliosis are summarized in Table 3. CT studies in 32 (89%) of 36 patients were either normal or showed nonspecific findings such as generalized or cerebellar atrophy. Two patients had a CT diagnosis of unilateral dilatation of a temporal horn that was contralateral to the side of epileptogenic

focus and surgery in both. In one other patient CT was thought to show dilatation of both temporal horns. In a single patient with left mesial temporal gliosis, CT revealed an area of low attenuation in the anteromesial left temporal lobe in the same location that MR demonstrated a high-signal abnormality on T2-weighted images.

Major MR findings in the 38 patients with pathologically proved mesial temporal gliosis are also summarized in Table 3. Thirty (79%) of 38 patients had either normal studies or nonspecific findings such as cerebral or cerebellar atrophy. In contradistinction to CT, MR detection of unilateral dilatation of the temporal horn was ipsilateral to the side of seizure focus and surgery in each of six cases. Only three (8%) of 38 patients had MR signal changes in the mesial temporal region (Fig. 8). Pathology in these cases showed moderate gliosis in one case and severe gliosis in the other two.

Clinical follow-up information at intervals ranging from 6 months to 1 year was available for 35 of the 53 surgical patients. Twenty-six patients (74%) were seizure-free; another four patients (11%) had rare seizures, defined as no more than one or two seizures per year. Five patients (14%) had less than a 90% reduction in seizure frequency.

Discussion

Partial seizures begin in a localized area of cerebral cortex, most often the temporal lobe, and are considered complex if there is associated alteration of consciousness. A significant number of patients in this subcategory of seizure diagnosis are refractory to medical management [12, 13]. Since the early 1950s, temporal lobectomy has provided a cure or a substantial improvement for many of these patients. Mesial temporal sclerosis or gliosis is the most common pathologic abnormality found [13]. Theories as to its cause include hypoxic damage to the hippocampus and parahippocampal
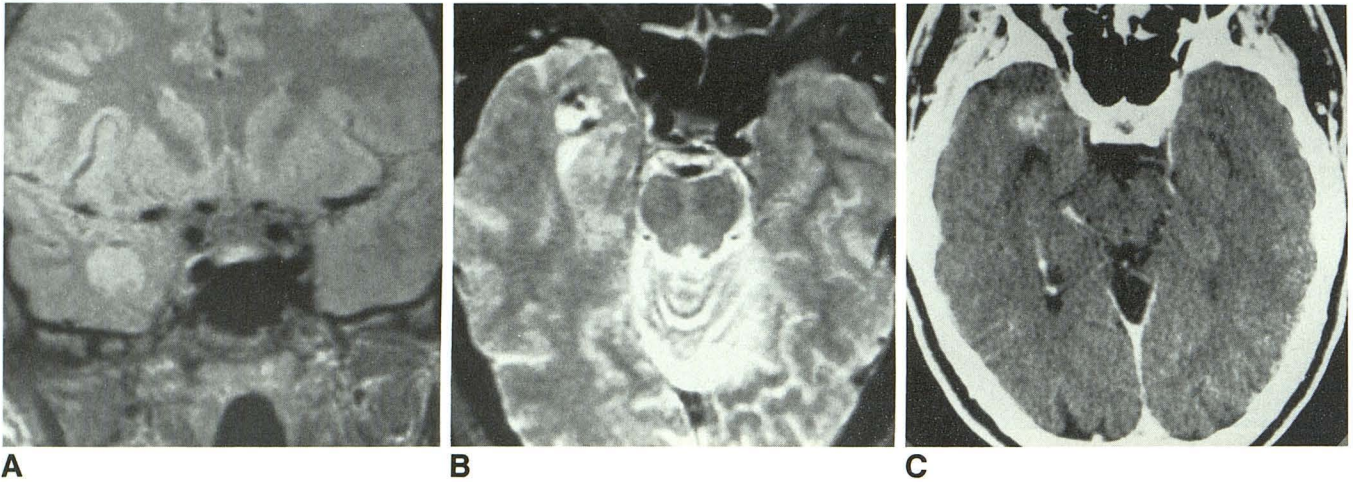
Fig. 3.—Benign fibrillary astrocytoma.
A–C, Intermediate-weighted coronal (A) and T2-weighted transaxial (B) MR images show well-defined round tumor containing calcifications (B). Mass effect produces deformity with posterior displacement of right temporal horn evident on both transaxial MR (B) and contrast-enhanced CT (C).
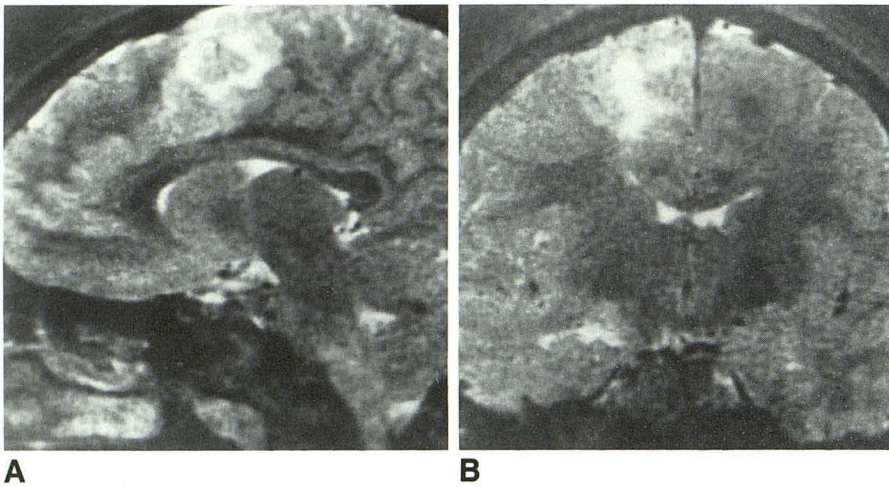

Fig. 4.—Giant cell astrocytoma in a 10-year-old girl with tuberous sclerosis.
A and B, T2-weighted sagittal (A) and coronal (B) MR images show posterior frontal parasagittal mass extending to level of corpus callosum. Tumor was composed of dysplastic cerebral tissue containing many giant astrocytes and a few giant neurons. CT without and with contrast enhancement was completely negative.
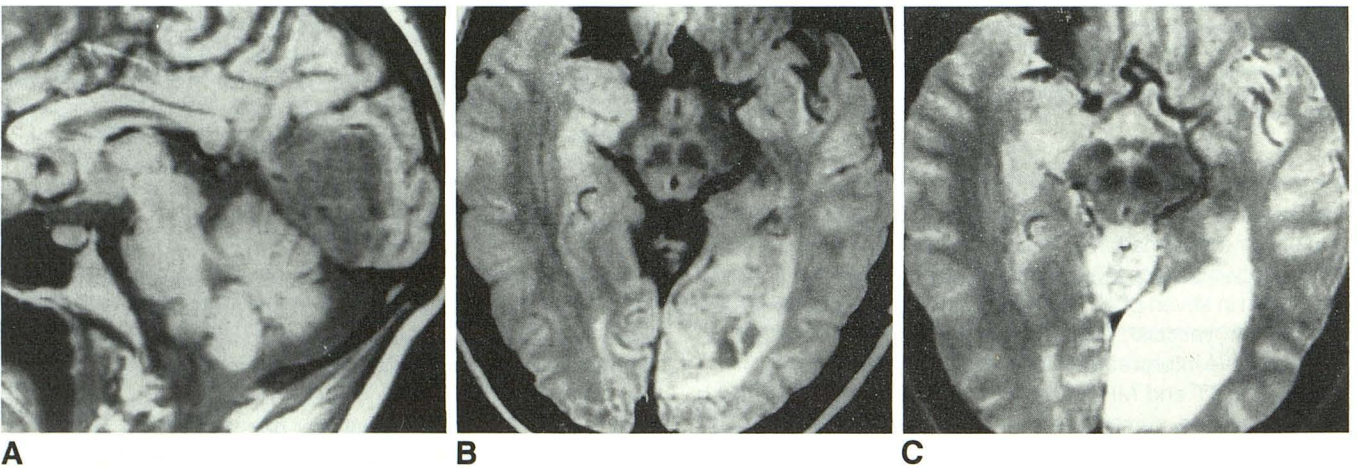

Fig. 5.—Juvenile cystic fibrillary astrocytoma in a 25-year-old woman.
A–C, Sagittal T1-weighted (A) and transaxial intermediate- (B) and T2- (C) weighted images reveal occipital tumor with posterior temporal extension that has very high signal on T2-weighted series (C). Microcysts were found to be a major component pathologically.
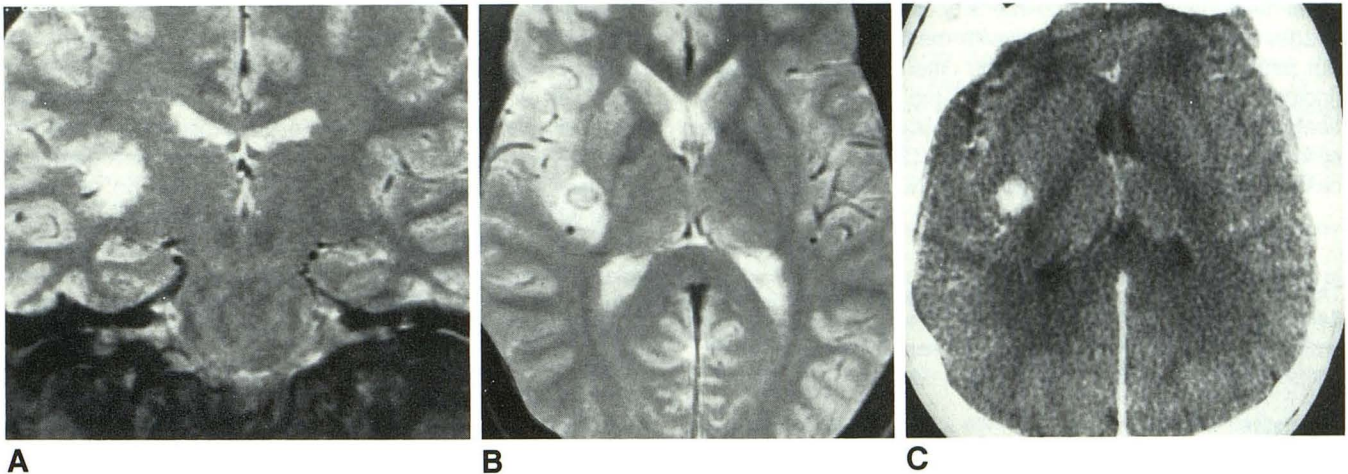
Fig. 6.—Right putaminal and insular astrocytoma, grade II/IV.
A–C, T2-weighted coronal (A) and transaxial (B) MR images and contrast-enhanced transaxial CT scan (C). Tumor with surrounding edema is best delineated on transaxial MR.
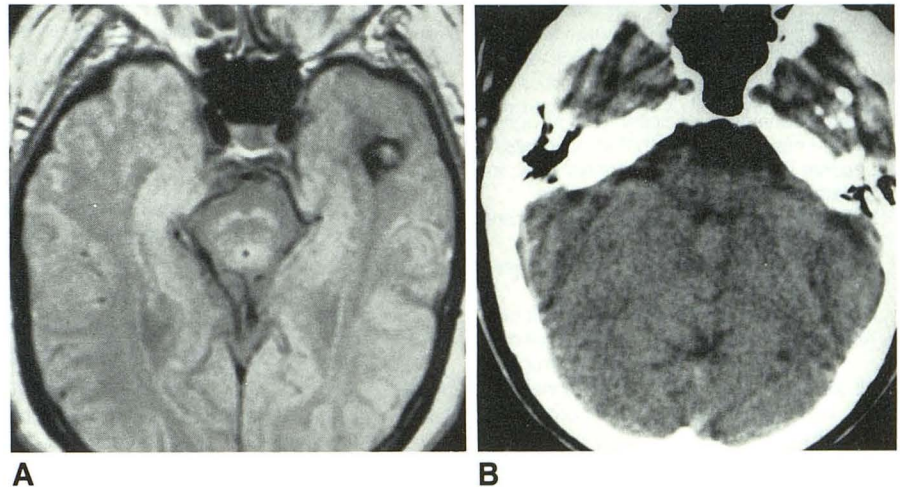
Fig. 7.—Thrombosed arteriovenous malformation in a 52-year-old man.
A, Intermediate-weighted transaxial MR image reveals lesion adjacent and anterolateral to left temporal horn.
B, Unenhanced CT scan shows popcorn calcifications.
Calcifications and old hemorrhage of surrounding tissue were noted at pathologic examination.

regions and pathologic change that occurs in response to repeated seizures [14, 15].

In our series, mesial temporal gliosis was present in 38 (72%) of 53 patients. Macroscopic structural lesions were present in the remaining 28% of cases. Tumors constituted 22% of cases and included four cases of ganglioglioma, a relatively rare tumor; ganglioglioma, therefore, represented 33% of the tumors in this series. It is of interest that gangliocytoma, a closely related tumor, was reported recently in three patients with childhood epilepsy [16]. In addition, several patients with benign fibrillary astrocytomas were noted to have scattered mature ganglion cells within what was predominantly a fibrillary astrocytic tumor. Neuronal heterotopia is a pathologic change associated with mesial temporal sclerosis, and the concurrence of tumor and changes of mesial temporal gliosis in seizure patients has been noted previously [13].

MR was successful in detecting the structural lesions, and provided a diagnosis of tumor in three patients that was missed by CT. However, the insensitivity of MR in patients with complex partial seizures and mesial temporal gliosis is also clearly demonstrated in this series. MR failed to demonstrate signal abnormalities in 35 of 38 patients with subsequent pathologically proved mesial temporal gliosis. The literature has been inconsistent in regard to the correlation of mesial temporal gliosis and the prevalence of abnormal high-intensity signal changes on T2-weighted MR images. In MR studies at low and mid field strengths, temporal-lobe T2 signal abnormalities were reported in several isolated cases [17, 18] and in 11 of 14 patients with mesial temporal gliosis in one previous series [19]. However, a similar study by Sperling et al. [8] found no MR abnormalities in 18 patients with mesial temporal gliosis and intractable complex partial seizures. One previous study with high-field-strength MR [20] found tem-

poral mediobasal high-signal-intensity areas on T2-weighted images in three of 12 patients with mesial temporal gliosis, but pathologic studies revealed no differences between the findings in these three cases and in the remaining nine patients. In another series of 31 patients with temporal-lobe epilepsy who were examined at 1.5 T [21], only one of 14 cases with mesial temporal gliosis showed a high-intensity

TABLE 3: CT and MR Findings in 38 Intractable Complex Partial Seizure Patients with Mesial Temporal Gliosis

Study/Finding	No.
CT	
Negative	25
Generalized cerebral and/or cerebellar atrophy	6
Dilatation of one or both temporal horns	3
Unilateral low-attenuation anteromesial temporal lobe	1
Posterior cranial hemiatrophy	1
Not available	2
Total	38
MR	
Negative	19
Cerebellar atrophy	4
Generalized cerebral atrophy	2
Posterior cranial hemiatrophy	1
Frontal bone flap[a]; occipital focal atrophy	1
Chiari I malformation with hydrocephalus	1
Lacunar infarcts/white-matter ischemic foci	2
Unilateral dilatation of temporal horn	2
Unilateral dilatation of temporal horn; cerebellar atrophy	2
Unilateral dilatation of temporal horn; generalized cerebral atrophy	1
Abnormal mesial temporal high signal on T2	1
Abnormal mesial temporal high signal on T2; diffuse cerebral and cerebellar atrophy	1
Abnormal mesial temporal high signal on T2; unilateral dilatation of temporal horn; cerebellar atrophy	1
Total	38

[a] Caused by evacuation of a traumatic subdural hematoma during infancy.

T2-weighted signal abnormality in the corresponding temporal lobe. MR was also negative in eight of 12 patients in the series of Heinz et al. [10] who were operated on for complex partial seizures and did not have tumors. A diagnosis of mesial temporal sclerosis was proved in three of these, while the surgical technique of subpial aspiration did not permit a definitive diagnosis in two; in the other three no diseased tissue was found. Surgical and pathologic correlation was not mentioned for the four cases in which MR was positive. The unilateral temporal-lobe T2 high-signal focus of mesial temporal gliosis may be a small and relatively subtle abnormality. It is possible that previous studies performed at low and mid field strengths, where the ratio of image contrast to noise is lower compared with high-field-strength examinations, were relatively more susceptible to interpretation error from "shading" artifacts and from carotid pulsation artifacts, especially on the coronal projection. In these previous studies, motion suppression techniques were not yet available or were not used.

Previous attempts with pneumoencephalography, and more recently with nonionic contrast CT cisternography [22], to define an atrophic temporal lobe or uncal herniation have met with varied success. Although the number of cases in our current series is small, the data suggest that MR may accurately detect a unilateral, focally dilated anterior temporal horn in patients with a unilateral seizure focus in the same temporal lobe. This may be a subtle or "soft" sign but can provide ancillary evidence that may be helpful in the surgical decision-making process in some borderline cases. Further correlation of this particular finding in a larger number of temporal lobectomy patients should prove of interest.

In summary, MR provided an important contribution in the preoperative diagnosis of tumor or vascular malformation in 28% of 53 patients operated on for refractory complex partial epilepsy. MR afforded a surgical decision without the need for invasive EEG monitoring in 14 of 15 patients with structural lesions. MR was found to be contributory in only a small number of patients (8%) with pathologic changes of mesial temporal gliosis.
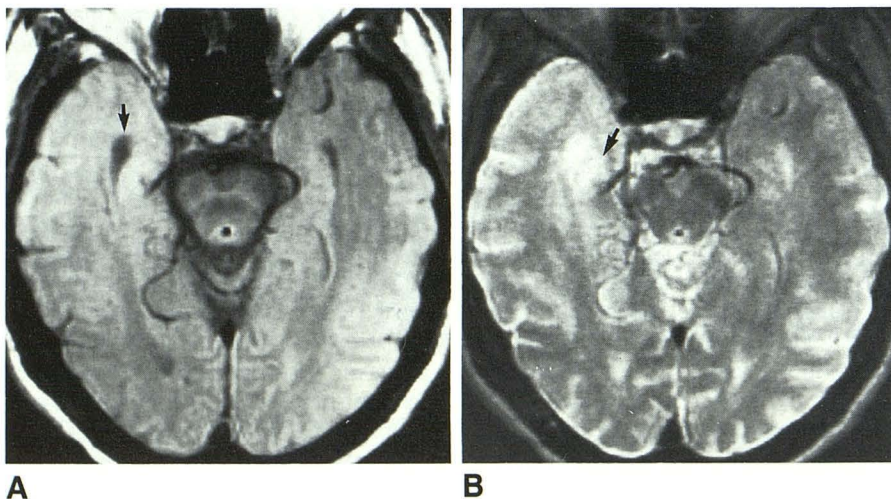

Fig. 8.—Mesial temporal gliosis in a 47-year-old woman.
A, Intermediate-weighted transaxial MR image shows focal dilatation of right anterior temporal horn (arrow) and mild increased signal intensity of right uncus.
B, T2-weighted transaxial image shows abnormal high-intensity signal (arrow) medial to dilated temporal horn.

A　　　　　　B

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