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Temporal-Lobe Epilepsy: Comparison of CT and MR Imaging

W. Schörner, H.-J. Meencke and R. Felix

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Comparison of CT and MR Imaging

W. Schörner[1]
H.-J. Meencke[2]
R. Felix[1]

In 50 patients with temporal-lobe epilepsy, CT and MR findings were compared. Axial CT scans were obtained before and after administration of contrast material. Coronal MR imaging was carried out with two spin-echo (SE) sequences with a repetition time of 1600 msec and echo times of 35 or 70 msec (SE 1600/35, SE 1600/70). A focal lesion was detected by CT in 12 cases and by MR in 16 cases. If discrete attenuation or signal abnormalities are also taken into account, CT provided a positive finding in 13 cases and MR imaging in 20 cases. With the exception of a small calcification, all the lesions revealed on the CT scans were also detected on the MR images. Among the examinations assessable for temporal-lobe asymmetry, signs of a unilateral reduction in temporal-lobe size were seen on two of 35 CT scans and on 15 of 38 MR images. In three patients who had temporal-lobe resection, a subsequent comparison was made between CT, MR imaging, and pathology. Histologically proven glial reactions that could not be detected on CT were demonstrated as high-signal-intensity lesions on the SE 1600/70 image.

We conclude that MR scanning, with its higher sensitivity, superior image quality, and ability of multiplanar imaging, should be the imaging technique of choice in the diagnosis of temporal-lobe epilepsy.

[1]Department of Radiology, Freie Universität Berlin, Klinikum Charlottenburg, Spandauer Damm 130, 1000 Berlin 19, W. Germany. Address reprint requests to W. Schörner.

[2]Department of Neurology, Freie Universität Berlin, Klinikum Charlottenburg, 1000 Berlin 19, W. Germany

Despite optimal pharmacotherapy, about 30% of patients with temporal-lobe epilepsy do not become seizure-free [1]. In these patients, surgery represents the best possible form of treatment. Provided that the indication is stringently applied, up to 50% of drug-resistant patients can be cured of their seizures by surgical intervention [2]. Good operative results, however, require previous accurate qualitative and topographic diagnosis [3]. Of importance here are determination of the unilaterality of the epileptogenic lesion, differentiation between temporal and frontal processes, and accurate establishment of the size and topography of the lesion. Frequently, such points can be clarified only through such invasive procedures as the EEG examination with subdural or depth electrodes [4].

To date, CT has been the most important imaging procedure in the investigation of temporal-lobe lesions [5–8]. However, in regions close to the base of the skull, the CT image is impaired by bone artifacts, which considerably reduce the quality of the image. Nor does selection of particular patient positioning or special angulation of the CT gantry (for example, 15–20° from Reid's baseline [6]) eliminate the limitations of CT in the evaluation of temporal-lobe lesions.

MR imaging permits direct visualization of the temporal lobe without impairment by the bone artifacts typical on CT. Initial investigations appear to indicate that MR imaging has a greater sensitivity than CT in the diagnosis of temporal-lobe epilepsy [9–12].

In our present study of 50 patients with temporal-lobe epilepsy, the diagnostic values of CT and MR imaging were compared. A major aim of this study was to describe the different presentations of temporal-lobe lesions on CT and MR.

Subjects and Methods

We studied 50 patients (26 male and 24 female) with temporal-lobe epilepsy. The patients were 16–62 years old (mean ± SD = 33 ± 10 years). The onset of temporal-lobe epilepsy extended back to the first decade of life in 21 patients and to the second decade in 16 of the patients; in 13 patients epilepsy did not occur until after the age of 20 years.

The diagnosis of temporal-lobe epilepsy was established clinically on the basis of the course of the seizure. The diagnosis was further supported by the results of the EEG examination: EEG detected a unilateral temporal abnormality in 26 cases and a bilateral abnormality in 15. In four patients, the abnormal electrical activity extended beyond the temporal lobe. In five patients, the EEG studies were without focal findings. Patients in whom the neurologic workup suggested the presence of a large focal temporal-lobe process (for example, a tumor) were excluded from the study.

Treatment with drugs rendered 11 patients free of seizures and 20 patients proved resistant to drug therapy; in the remaining 19 patients the influence of medication on the temporal-lobe epilepsy had not been established at the time of the MR imaging. In three patients with refractory temporal-lobe epilepsy, partial lobectomy of the temporal lobe was carried out after the MR examination.

All the patients were referred for a plain CT examination. With the exception of four cases, all patients had a contrast-enhanced CT study (100-ml rapid drip infusion of an ionic or anionic contrast material). The CT studies were regularly conducted in an axial imaging plane with different units (Somatom 2, Somatom DR 2, EMI Head Scanner 1010, GE 8800). In addition, CT scans were obtained in a coronal imaging plane in six patients. When MR imaging indicated definitely or possibly abnormal findings and CT revealed no corresponding findings, CT was repeated on a third-generation scanner (usually the Somatom 2).

MR imaging was performed on a supraconducting unit operating at 0.35 T (Siemens Magnetom). A special head coil (25 cm in diameter) was used for brain imaging. MR scans were produced in the coronal plane (10-mm slice thickness) by using a multiple-slice spin-echo (SE) technique. In all patients, SE pulse sequences were performed with a repetition time (TR) of 1600 msec and echo times (TEs) of 35 and 70 msec (double-echo technique) (abbreviated SE 1600/35 and SE 1600/70, respectively). In most patients further coronal images were obtained with a short TR of 400 msec and a TE of 35 msec (SE 400/35). A matrix of 256 × 256 pixels was used, which permits a nominal resolution of 1 × 1 mm in the plane imaged. With twofold data acquisition, the imaging times for the above-mentioned long TR and short TR SE pulse sequences were about 13.7 and 3.4 min, respectively.

CT scans and MR images were reviewed independently by two observers. First, the images were evaluated with respect to a clearly defined abnormality. Focal abnormalities were classified according to their CT density and MR signal intensity relative to the density and signal intensity of normal brain tissue (hyper-/iso-/hypodense on CT and hyper-/iso-/hypointense on MR), and also with respect to the appearance (homogeneous/inhomogeneous). The studies were categorized as normal, possibly abnormal, and abnormal.

A second criterion for image interpretation was the symmetry of the temporal lobes. Here, the sizes of the two temporal lobes were compared on the axial (and when available the coronal) CT image and the coronal SE 1600/35 scan. The smaller of the two temporal lobes was designated as atrophic. The findings were classified as normal, atrophic, or nonevaluable. Under nonevaluable were all those examinations in which, owing to inadequate image quality in the vicinity of the base of the skull or to oblique positioning of the patient's head, assessment of symmetry was difficult.

Results

CT Findings

The CT examination led to the detection of a focal lesion in 11 patients. In one of these, two lesions were found, one in each hemisphere (Table 1). The locations of the 12 lesions were temporal in five; temporoparietal in one; and temporooccipital, occipital, and parietal in two each. Seven of the 12 lesions were hypodense on plain CT scans. Calcified lesions were detected in five cases. Scans obtained after administration of contrast agent revealed an accumulation of contrast material in the lesion in one case (case 8); the other 11 lesions did not enhance after administration of contrast material.

In one female patient, a lesions located in the frontal lobe was suspected. However, no reliable assessment was possible because of marked artifacts adjacent to the frontal sinuses. Thus, the CT finding was classified as possibly abnormal.

In none of the 11 patients with signs of a focal abnormality (12 demonstrated lesions, one suspected lesion) (Table 1) was temporal-lobe asymmetry diagnosed from the CT scan. Among the other 39 patients with no evidence of a focal lesion on the CT scan, unilateral temporal-lobe atrophy was found in two cases; in 22 cases, the temporal lobes were thought to be of approximately equal size on both sides. In 15 cases, no reliable assessment of temporal-lobe atrophy was possible. In six cases in which axial and coronal CT scans were available, both techniques showed normal findings which respect to the size of the temporal lobes on both sides.

MR Findings

MR revealed an abnormal finding in 15 patients. In 14 patients, a solitary lesion was detected; in one patient (case 8), two lesions were observed, one in each hemisphere. The locations of the lesions were temporal in seven, adjacent to the temporal lobe in four, occipital in three, and frontal and parietal in one each.

On T2-weighted SE 1600/70 images, all the lesions were seen as producing a signal different from that of normal brain tissue. In 13 cases, the lesions were homogeneous. In 11 of these cases the lesions showed an increased intensity (Figs. 1–3); in two calcified lesions they showed a reduced signal intensity. In three cases, the lesions were inhomogeneous with a hypointense center and a marginal zone of high signal intensity.

On SE 1600/35 images, an abnormality was seen in 12 of 16 cases. In four cases in which a focal abnormality was demonstrated on T2-weighted SE 1600/70 scans, MR images were normal on the SE 1600/35 sequence. Of the 12 lesions detected in this sequence, six were homogeneous and six inhomogeneous. Of the homogeneous lesions, four were hyperintense and two hypointense. In all six cases, the inhomogeneous lesions revealed a low-intensity central portion and a signal-intense marginal zone.

On the T1-weighted SE 400/35 sequence, 12 of 16 lesions were homogeneous and hypointense. In three patients, the

TABLE 1: CT Attenuation and MR Signal Abnormalities in Temporal-Lobe (TL) Epilepsy

Case No.	Age	Gender	Location of Lesion/ Size (cm)	CT Density of Lesion	MR Signal Intensity of Lesion			Remarks
					SE 1600/70	SE 1600/35	SE 400/35	
1	24	F	R TL (basal region)/2 × 1.5 × 4	Isodense	Hyperintense	Hyperintense	Hypointense	Focal gliosis
2	25	M	R TL (middle temporal gyrus)/1.5 × 1.5 × 2	Isodense	Hyperintense	Isointense	Isointense	. . .
3	15	F	L TL (mediobasal region)/1.5 × 2 × 3	Hypodense	Hyperintense	Mixed	Hypointense	. . .
4	23	M	L TL (basal region)/ 2.5 × 3 × 3.5	Hypodense	Mixed	Mixed	Hypointense	Cystic lesion surrounded by gliosis
5	18	F	L TL (mediobasal region)/2 × 2 × 2	Hypodense	Hyperintense	Isointense	Hypointense	. . .
6	51	M	R TL (middle temporal gyrus)/2 × 1 × 1	Hyperdense	Mixed	Mixed	Not done	. . .
7	33	M	L TL (basal region)/2 × 2 × 2	Hyperdense	Hyperintense	Hyperintense	Hypointense	Hamartoma, centrally calcified
8	31	F	L temporoparietal lobe/1.5 × 2 × 2	Hyperdense	Hypointense	Hypointense	Hypointense	Suspected AVM
			R frontal lobe (basal region)/0.7 × 2 × 1	Possibly hypodense	Hyperintense	Hyperintense	Hypointense	Suspected posttraumatic scar
9	35	F	L temporooccipital lobe (basal region)/ 0.5 × 1 × 4	Isodense	Hyperintense	Isointense	Isointense	. . .
10	22	M	R temporooccipital lobe/2.5 × 2 × 2	Hypodense	Hyperintense	Mixed	Hypointense	. . .
11	37	M	L occipital lobe (basal region)/2 × 0.5 × 2	Isodense	Hyperintense	Isointense	Isointense	. . .
12	30	M	R occipital lobe (basal region)/2.5 × 2 × 2	Hypodense	Mixed	Mixed	Hypointense	. . .
13	21	M	L occipital lobe (basal region)/2.5 × 2 × 2	Hypodense	Hyperintense	Mixed	Hypointense	. . .
14	17	M	L TL (mediobasal region) 2 × 1.5 × 1	Isodense	Possibly hyperintense	Isointense	Isointense	. . .
			L temporooccipital lobe/2 × 1 × 1	Hypodense	Hyperintense	Hyperintense	Hypointense	. . .
15	19	F	L parietal lobe (subcortical)/2 × 1 × 1	Hyperdense	Hypointense	Hypointense	Hypointense	Calcification, probably caused by infection
			R parietal lobe (subcortical)/0.5 × 0.5 × 0.8	Hyperdense	Isointense	Isointense	Isointense	Calcification, probably caused by infection
16	31	M	L TL (mediobasal region)/1.5 × 1 × 2	Isodense	Possibly hyperintense	Isointense	Isointense	. . .
17	16	F	L TL (mediobasal region)/0.5 × 1.5 × 1	Isodense	Possibly hyperintense	Isointense	Isointense	. . .
18	35	F	L TL (medial region)/ 0.5 × 1 × 1	Isodense	Possibly hyperintense	Isointense	Isointense	. . .

Note.—R = right; L = left. In CT, an isodense finding was considered normal. In MR, an isointense finding was considered normal. In MR, a lesion of mixed signal intensity had a hypointense center and hyperintense periphery.

lesion was isointense and undetectable. In one patient, no image from the SE 400/35 sequence was available in the plane of the lesion.

In four cases, the MR examination revealed a possibly abnormal finding. In each of these cases there was an area of slightly increased signal intensity in the mediobasal portion of the temporal lobe on the SE 1600/70 image (Fig. 4).

Among the 18 patients with a demonstrated or suspected focal lesion, signs of unilateral temporal-lobe atrophy were found in four cases (cases 1, 11, 16, and 18). "Atrophy" of the temporal lobe was localized on the same side as the lesion in every case (Figs. 3 and 4). In 11 patients, the size of the temporal lobe was the same on both sides. In three patients, no assessment of the symmetry of the temporal
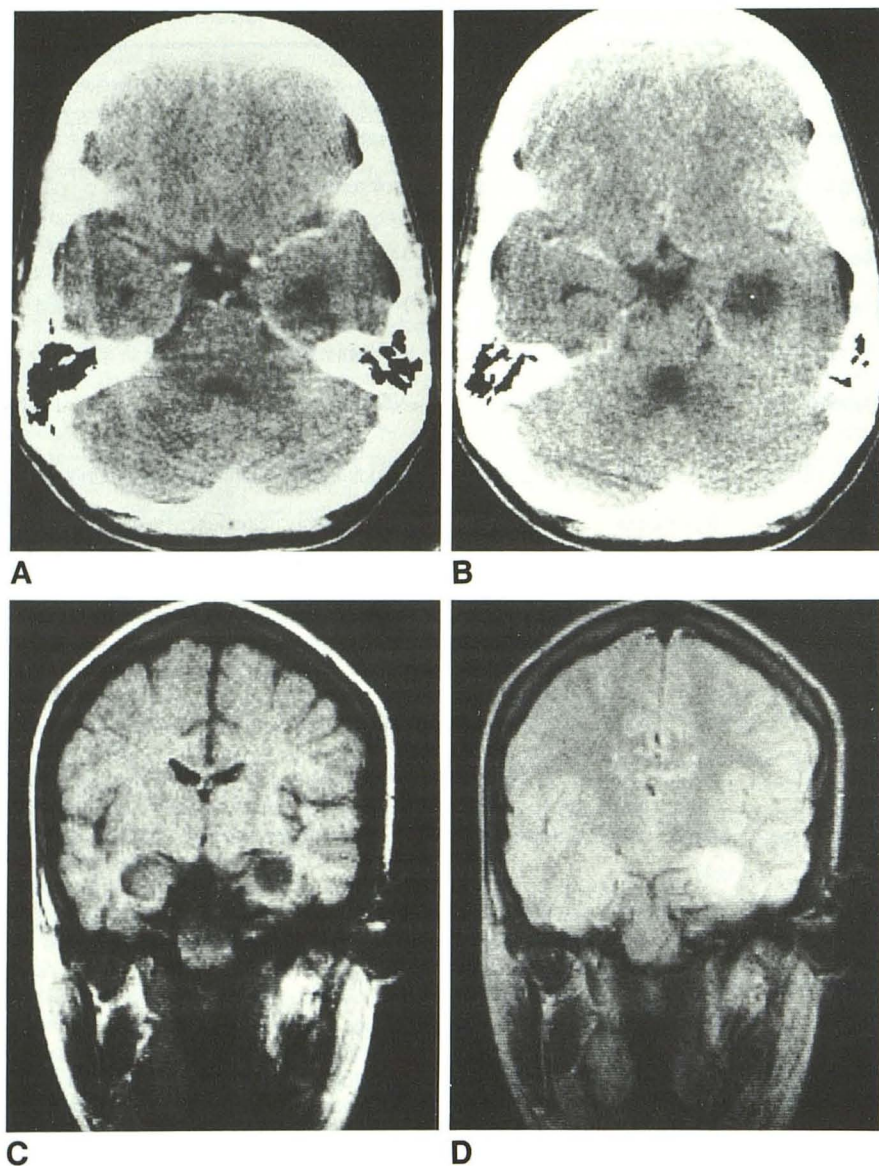
A B

C D

Fig. 1.—Diagnostically equivalent demonstration of cystic lesion on axial CT scans and coronal MR images (case 5).

A and B, Contrast-enhanced CT scans show round, hypodense lesion in mediobasal portion of left temporal lobe.

C, T1-weighted MR scan (SE 400/35). Hypointense lesion has appearance similar to that on CT scans.

D, T2-weighted scan (SE 1600/70). Lesion has high signal intensity. (Temporal-lobe symmetry was not assessed because of oblique positioning of patient's head.)

lobe was possible owing to the oblique positioning of the patient (Fig. 1).

Among the other 32 patients with no demonstrated or suspected focal temporal-lobe lesion, signs of asymmetry in the size of the temporal lobe were found in 11 cases by both observers, while in eight cases both temporal lobes were found to be equal in size. In four cases, the evaluators differed in their assessment of temporal-lobe symmetry. In nine cases, no evaluation of temporal-lobe symmetry was possible owing to the oblique positioning of the patient.

Comparison of CT and MR Findings

Of 17 definitely abnormal findings, MR revealed 16 focal processes, whereas CT showed focal lesions in 12 cases.

With a single exception—a tiny calcification—all of the lesions diagnosed from the CT scan were also detected by MR imaging. On the other hand, four lesions detected by MR imaging were completely missed on the CT scan.

In another case, classified on the basis of the CT scan as having a possibly abnormal finding in the region of the frontal lobe, the MR image revealed a definitely abnormal finding. In another four patients, in whom the CT scan revealed no abnormal finding, a possibly abnormal lesion was detected on the MR image.

All of the lesions that completely escaped detection on the CT scan were processes that, in comparison with the remaining lesions, were relatively difficult to detect on the MR image because of their small size (cases 9 and 11) or their behavior with regard to signal intensity (cases 1 and 2). In these cases, the scans obtained in the SE 400/35 and SE 1600/35 se-

Fig. 2.—Superior sensitivity of MR imaging relative to CT scanning. Focal lesion of right temporal lobe is visible only on T2-weighted MR image (case 2).

A and B, Normal findings on contrast-enhanced CT scans at different levels.

C, SE 1600/35 image shows no obvious abnormality in signal intensity. However, outer contour of right middle temporal gyrus seems to be more prominent.

D, SE 1600/70 image clearly shows focal lesion of high signal intensity in middle temporal gyrus (arrow).

A   B

C   D
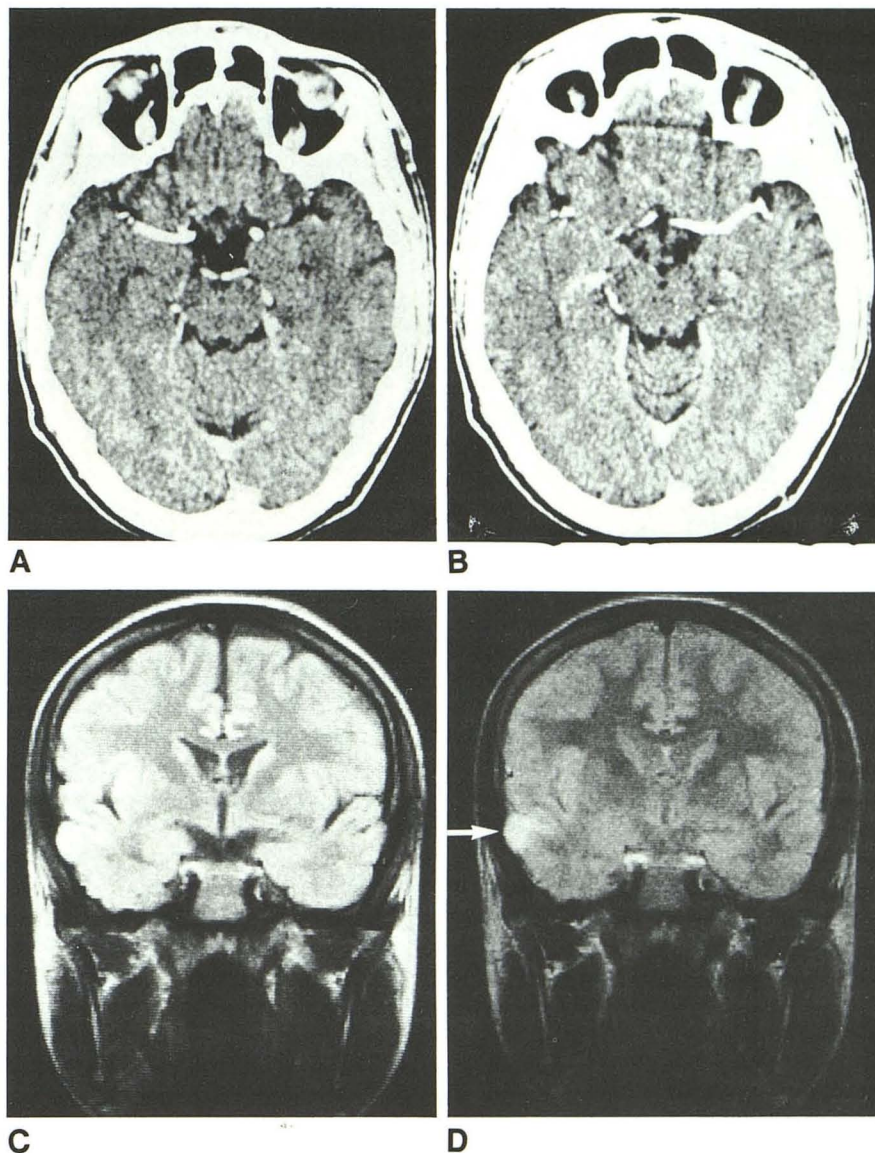
quences did not reliably demonstrate the presence of a lesion, but the T2-weighted images displayed the focal lesions as areas of slightly or moderately increased signal intensity (Figs. 2 and 3).

Among the 11 cases in which both the CT scan and MR image revealed a definite abnormal finding, the lesions were better defined by MR in six cases (cases 3, 4, 6, 10, 12, and 13). In these six patients, the lesions were homogeneous on the CT scan, but the SE 1600/35 image (in individual cases also SE 1600/70 images) showed an inhomogeneous lesion with areas of centrally reduced signal and a periphery of increased signal. In one patient (case 4), the process appeared appreciably larger on the MR image than on the CT scan.

In five cases, the CT scan demonstrated a calcification. This finding was indirectly demonstrable on the MR image—in three cases it was seen as an area of low signal intensity

on all sequences. In one case, the MR image revealed an abnormal finding that, however, was not typical of calcification because it presented a high signal. One small calcification seen on CT was not seen on MR.

Comparison of MR and Histologic Findings

In three drug-resistant patients with temporal-lobe epilepsy and definite MR evidence of a focal lesion, a partial temporal lobectomy was carried out. In one case, the histologic workup revealed the presence of focal gliosis, while in two cases a hamartoma was diagnosed.

A venous infarction was suspected as the cause of the gliosis detected histologically (case 1). The MR finding of a focal homogeneous lesion with reduced signal intensity on
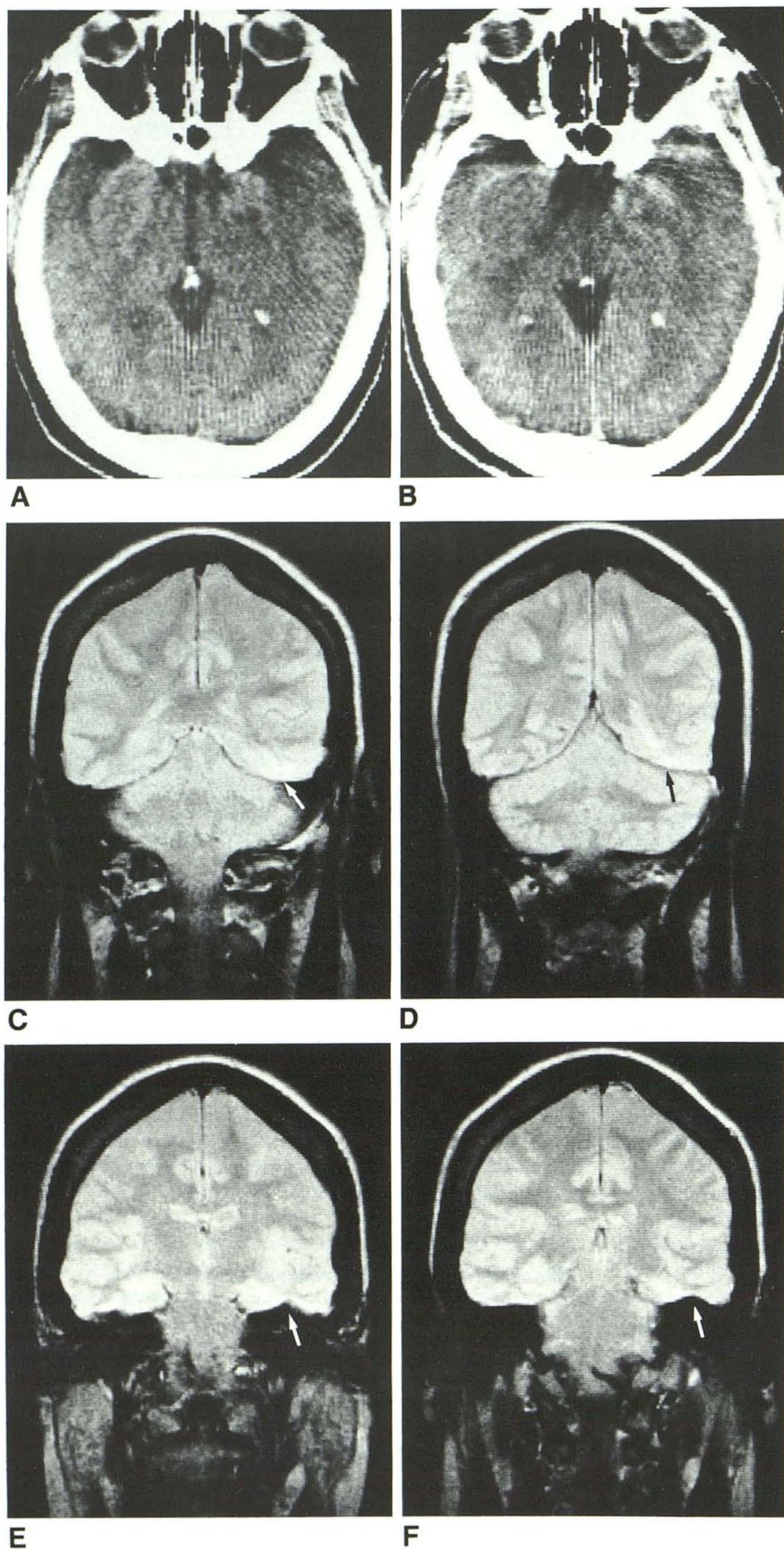
Fig. 3.—Focal lesion is detectable only on T2-weighted scans and is associated with ipsilateral "atrophy" of temporal lobe (case 9).

A and B, Normal appearance of temporal lobes on contrast-enhanced CT scans at different levels.

C–F, Contiguous coronal SE 1600/70 scans from occipital to temporal. Small lesion of high signal intensity is visible in left occipital lobe, directly above tentorium (arrows C and D), and is associated with ipsilateral focal reduction in size of temporal lobe (arrows, E and F).

Fig. 4.—Suspected lesion in anterior hippocampus visible on T2-weighted MR image (case 18).
A and *B*, Normal contrast-enhanced CT scans at different levels.
C, SE 1600/35 image. No abnormality is seen.
D, SE 1600/70 scan. Small area in left mediobasal temporal lobe is seen with slightly increased signal intensity (*arrow*).

A   B

C   D
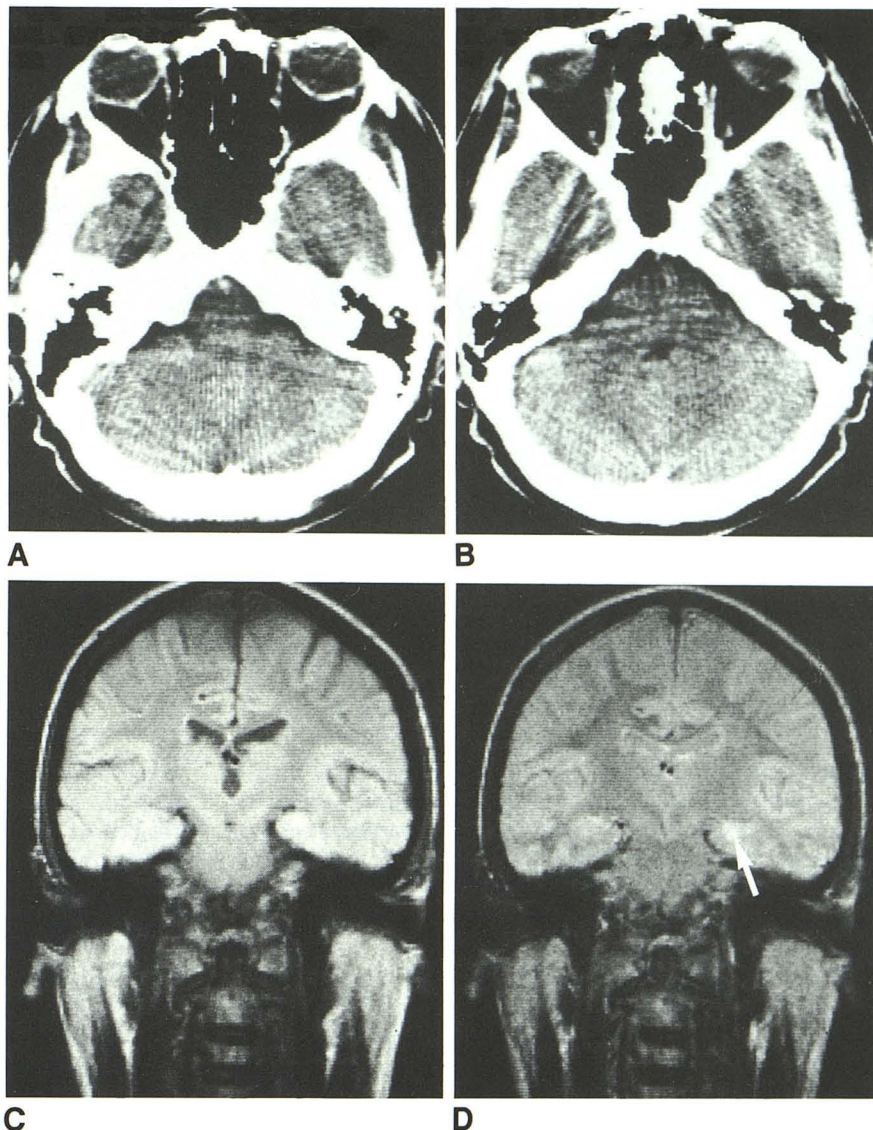
SE 400/35 images and increased signal intensity on SE 1600/35 and SE 1600/70 images (Fig. 5) was compatible with the histologic finding of gliosis. CT findings were normal.

In one patient (case 4), a hamartoma with a centrally located cyst was detected grossly. Histologically, the periphery of the lesion revealed an extensive glial reaction. On the MR images, the cyst had a reduced signal intensity. On SE 400/35 images it was not possible to decide reliably whether the peripheral glial portion of the lesion produced a signal different from that of surrounding brain tissue; but on SE 1600/35 and 1600/70 scans a broad, homogeneous zone of increased signal intensity was observed. CT showed an area of diminished density (this correlated with the histologically confirmed cyst), but did not reveal the surrounding area of gliosis.

In the third patient (case 7), the histologic workup revealed a calcified hamartoma. Histologically, in addition to the centrally located calcifications, an extensive glial reaction was also recognized in the periphery of the lesion. In the MR examination, the applied pulse sequences each showed the lesion (both calcifications and peripheral gliosis) as a homogeneous area with reduced and increased signal intensity, respectively (Table 1). CT demonstrated only the calcifications, but was not able to show the glial component in the periphery of the lesion.

Discussion

Psychomotor attacks are related to functional disturbances of the temporal lobe [13]. As the pathologic investigations of resected temporal-lobe material show, a specific morphologic lesion is found in roughly 75% of patients with psychomotor attacks [2, 14, 15]. In about 25% of such cases the lesions are already grossly detectable (for example, tumors), and in roughly 50% of the patients the pathologic process is hippo-
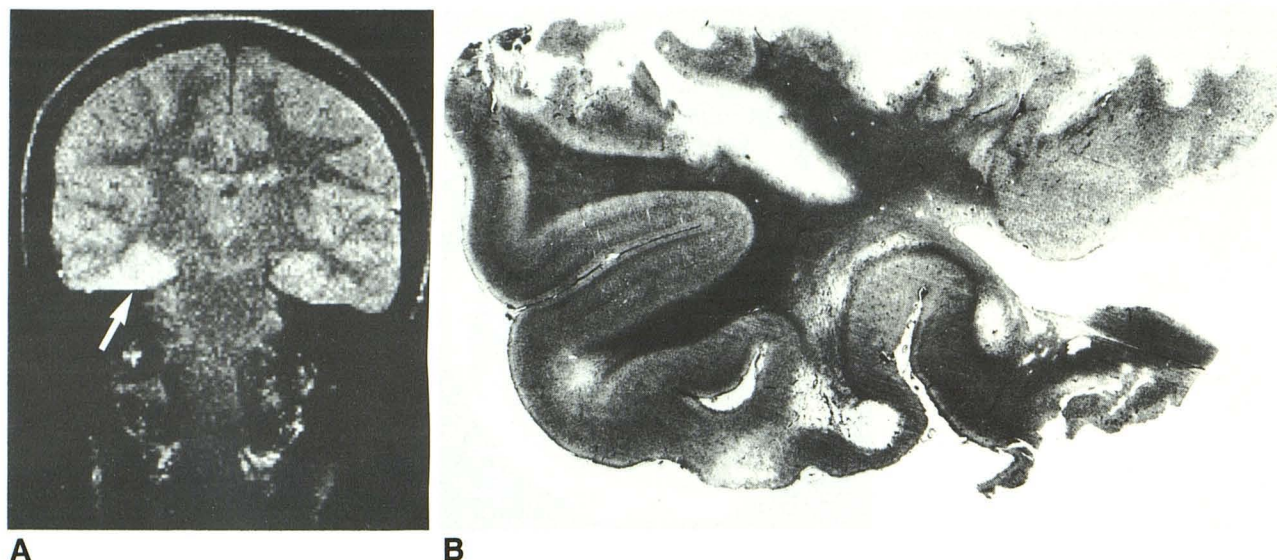
Fig. 5.—Correlation between pathologically proven gliosis and signal-intensity increase on T2-weighted MR image (case 1).
A, Coronal SE 1600/70 scan. Small area of definitely increased signal intensity in basal portion of right temporal lobe (arrow). (Reprinted from [11].)
B, Cross section of resected right temporal lobe. Macroscopically visible changes, predominantly in mediobasal portion of right temporal lobe (histologically proven gliosis within hippocampus).

campal sclerosis, which is defined predominantly by the microscopic appearance. Hippocampal sclerosis is characterized by elective neuronal necrosis, and a varyingly marked increase in the number of glial cells in the mediobasal portion of the temporal lobe [16]. Hippocampal sclerosis is frequently accompanied by atrophy or hypoplasia of the ipsilateral temporal lobe [8, 17, 18].

Depending on the composition of the case material, the CT investigation of patients with psychomotor attacks produces evidence of a focal lesion in 15–45% of patients [6, 11, 19]. These findings represent gross structural lesions, such as tumors, infarctions, cysts, or posttraumatic lesions [6, 10, 11, 13]. But even gross structural lesions of relatively large size can escape detection on the CT scan owing to the presence of bone artifacts, which make assessment of the temporal lobe difficult [6].

The MR image provides an artifact-free presentation of the temporal lobe and is thus better suited than is CT for the detection of lesions close to the base of the skull. As Ormson et al. [10] reported, the preoperative CT scan in 12 patients with pathologically confirmed gross temporal-lobe lesions was positive in only seven cases, as opposed to 10 cases imaged by MR. In our case material the ratio of positive findings on CT and MR was similar to that of Ormson et al. (12 positive CT findings vs 16 abnormal MR findings). Of our three patients who had surgery, CT was positive in two, whereas MR showed an abnormal process in all three. With the exception of the limited detectability of small calcifications, we found that MR imaging has a greater sensitivity than CT scanning in the diagnosis of patients with psychomotor attacks.

As a rule, temporal-lobe gliosis cannot be recognized on CT [6]. Only in a single case in which the temporal-lobe gliosis was associated with a coarse calcification did CT reveal a positive finding [20]. Otherwise, temporal-lobe gliosis is probably too small or produces insufficient attenuation changes relative to normal brain tissue to be detected on CT. The extent to which temporal-lobe gliosis is directly detectable by MR imaging has already been considered, but the findings have been contradictory [10, 21–23]. For example, Ormson et al. [10] found no abnormal MR findings in patients in whom mild temporal-lobe gliosis had been confirmed histologically. In contrast, McLachlan et al. [22] found a prolongation of T2 in the region of the temporal lobe in four of eight patients with hippocampal gliosis. However, the correlation between the degree of severity of temporal-lobe gliosis and its detectability on the MR image was low. Ethier et al. [23] reported patients in whom minimal to moderate temporal gliosis was found histologically and in whom MR imaging revealed an abnormal finding in 10 of 12 cases. Here, a focal increase in signal intensity was detected over mesiotemporal structures on T2-weighted images. In our patient population, too, we were able to find a slight increase in signal intensity in a focal area of the mediobasal temporal lobe in four patients with a negative CT scan. The localization and extent of the abnormal findings appear compatible with temporal-lobe gliosis. Because to date a correlation with neuropathologic findings is lacking, we decided to classify the MR findings as possibly abnormal.

Glial reactions of the temporal lobe, as mesial temporal sclerosis or as an accompanying reaction of gross structural lesions, are detectable with MR imaging, as confirmed by one surgically proven case. In two patients with focal lesions, both of which were detectable by CT and MR, the glial marginal zone was detectable only on the MR image. Our present findings and the data in the literature indicate that gliosis can be detected by MR imaging with varying frequencies.

Because temporal-lobe gliosis is not usually visible on CT,

detection of unilateral atrophy by CT or MR—which was assumed to be an indirect correlate of regional gliosis in the temporal lobe—received particular attention. Conventional radiographic and pneumoencephalographic procedures—today largely replaced by CT studies—were earlier used to demonstrate unilateral temporal-lobe atrophy [8, 18, 24]. The reliability with which unilateral atrophy of the temporal lobe can be recognized on CT is variable and depends on the CT technique [6, 8]. Wyler and Bolender [8] reported a complete correlation between preoperatively predicted temporal-lobe atrophy and pathologic findings in 17 patients with temporal-lobe epilepsy when CT examinations were performed by a high-resolution technique with metrizamide-enhanced CSF and planimetric analysis. In reviewing 40 CT examinations that were routinely performed in an axial imaging plane and occasionally in a coronal imaging plane, Blom et al. [6] found that CT, even if performed with thin slices and coronal scans, did not provide an accurate demonstration of temporal-lobe asymmetry.

In our study we did not obtain coronal CT scans routinely, because artifacts from minor patient motion, especially when the patient's head was stretched backward to an extreme degree and occasionally from dental fillings, often make interpretation of coronal CT scans difficult.

MR imaging does, indeed, appear to be very sensitive for the detection of asymmetry in the size of the temporal lobes [11, 25]. Unilateral atrophy of the temporal lobe in patients with psychomotor attacks is generally reported more often after MR than after CT scanning [11, 21, 22]. In our own case material, signs of unilateral temporal-lobe atrophy were found in 15 of 38 assessable MR studies, but in only two of 35 CT examinations. The reasons for this are probably the superior imaging quality of MR in the region of the temporal lobe close to the base of the skull, together with the possibility of multiplanar imaging, with the aid of which partial-volume effects can better be avoided.

Unilateral temporal-lobe atrophy often is difficult to assess on MR images, primarily because even slight degrees of rotation of the patient's head can lead to considerable variation in the asymmetry of the images. Possibly, quantitative planimetric studies, as have been used in CT scanning [8], may be helpful in MR imaging of temporal-lobe asymmetry.

In summary, MR imaging seems qualitatively better than CT in the evaluation of temporal-lobe lesions in psychomotor seizure disorders. Moreover, there is evidence to suggest that temporal-lobe gliosis, which frequently represents the morphologic substrate of a psychomotor epilepsy and which cannot be detected with CT scanning, can be shown directly on the MR image (or indirectly because of temporal-lobe asymmetry). MR imaging must, therefore, be considered the preferred imaging procedure for studying a patient with psychomotor attacks.

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