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MR Findings after Depth Electrode Implantation for Medically Refractory Epilepsy

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PURPOSE: To evaluate using MR imaging chronic changes in the brain parenchyma after electroencephalography depth electrode placement in patients with medically refractory epilepsy. METHODS: A retrospective review of MR scans in 57 patients who underwent stereotactic placement of 210 depth electrodes was performed. Scans were evaluated for evidence of aliosis, hemorrhage, or infection along the probe tracts. RESULTS: Signal abnormalities along the probe tracts were seen in 38 patients (67%). Of the 210 probe tracts evaluated on long-repetition-time images, 85 (41%) were associated with punctate hyperintensity and four (2%) with punctate hypointensity; 120 (57%) showed no MR changes to suggest prior electrode placement. One probe placement was complicated by a significant intraparenchymal hemorrhage. CONCLUSIONS: Depth electrode implantation for electroencephalography monitoring results in imperceptible or minimal chronic changes as detected by MR in almost all patients. The punctate hypersensitivity on long-repetition-time images is thought to be caused by gliosis along the probe tracts. These signal changes should not be confused with the seizure focus. Significant hemorrhage or infection is a rare complication of probe placement. Incorporation of MR angiographic data with conventional spin-echo images at the time of stereotactic probe placement may further reduce the low incidence of probe-related hemorrhage.

Index terms: Seizures; Electroencephalography; Brain, magnetic resonance

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Depth electrode electroencephalography (EEG) monitoring plays an important role in the evaluation and selection of patients with medically intractable epilepsy for surgical treatment. Compared with scalp monitoring, depth electrode EEG may better locate a single epileptogenic region, or detect unsuspected multiple foci (1–4). However, it is invasive and subject to more patient complications than either scalp or sphenoidal EEG. We studied a group of patients who had undergone depth electrode placement with fol-

low-up magnetic resonance (MR) scans to evaluate evidence of histologic change, hemorrhage, or infection along the probe tracts.

Material and Methods

MR examinations of 57 patients with a clinical diagnosis of medically refractory epilepsy who underwent implantation of 210 depth electrodes were retrospectively reviewed by two experienced neuroradiologists. There were 30 men and 27 women, 16 to 50 years of age (mean 29.8 years). All MR examinations were performed on a 1.5-T superconductive magnet and were obtained from 1 to 64 months (mean, 18.6 months) after probe removal. A multisection short repetition time sagittal sequence (600/30/2 [repetition time/echo time/excitations]) and a multisection, dual-echo axial and/or coronal (2000-2500/30,80/1) sequence were performed in each case. Nineteen patients also had an axial (600/15/2) sequence after intravenous administration of 0.1 mmol/kg gadopentetate dimeglumine. Two different stainless steel depth electrodes were used. A semirigid probe constructed of no. 24 needle stock wrapped with 90% platinum and 10% iridium wires was

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used in 33 patients before April 1988. Subsequently, a flexible probe consisting of helically wound wires with a 1mm external diameter was used in 24 patients (3, 4). Depth electrode implantation was performed using a modified Todd-Wells frame and a predominantly parasagittal placement with MR images used to determine end point targets. From one to eight electrodes were implanted per patient. The most common regions studied by depth electrodes were the temporal and frontal lobes (Fig 1). The temporal lobe and hippocampus were evaluated with sagittal probe insertion from the medial occipital lobe through the hippocampus to the amygdala. This was often done in conjunction with insertion of frontotemporal electrodes. When the frontal lobe was suspected as the probable seizure source, medial frontal lobe probes were placed (1).

Fig. 1. Depth electrode implantation. Sagittal (600/11) (A) and axial (600/20) (B) images demonstrate depth electrodes in situ. These probes were used to evaluate the hippocampus and temporooccipital lobes (straight arrow), the frontotemporal region (curved arrow), and the supplementary motor cortex (open arrow).

Results

A total of 210 probe tracts were evaluated; 120 (57%) showed no MR abnormality, whereas 89 (43%) demonstrated signal changes (Table 1).

The most common pattern of signal abnormality seen in 85 (41%) of these probe tracts were punctate hyperintensities on both the shortand long-echo-time images of the long-repetitiontime sequences (Fig 2). These were 1 to 2 mm in diameter and were best demonstrated in the imaging plane perpendicular to the probe trajectory. Along four (2%) probe tracts, there were punctate foci of signal void that demonstrated magnetic

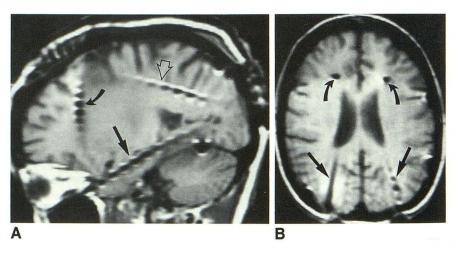
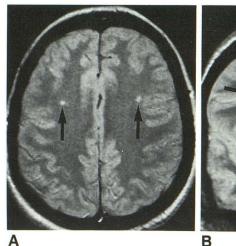
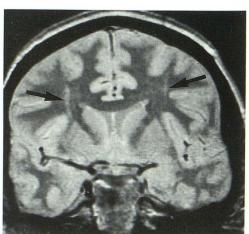


TABLE 1: MR findings after 210 depth electrode implantations

No. of Probe Tracts (%)	Findings on Long- Repetition-Time Scans	Interpretation
120 (57%)	None	Normal
85 (41%)	Punctate hyperintensity	Gliosis
4 (2%)	Punctate signal void	Hemosiderin
1 (<1%)	Hyperintense mass	Hematoma

Fig. 2. Pattern of hyperintense signal abnormality. Axial (2000/30/1) (A) and coronal (2000/30/1) (B) images illustrate hyperintense signal changes along the site of the depth electrodes, which are thought to represent regions of gliosis. These signal abnormalities (arrows) conform to the shape of the depth electrode, punctate in cross-sectional (A), and linear in the in-plane (B), images. The depth electrodes were removed 23 months before MR examination. Fortyone percent of all electrode tracts had this appearance.





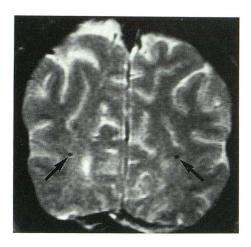


Fig. 3. Hemosiderin along probe tracts. Coronal (2000/30/1) image demonstrates punctate hypointense foci (*arrows*) consistent with hemosiderin from microhemorrhage. MR imaging was obtained 7 months after removal of occipitotemporal probes.

susceptibility, consistent with hemosiderin related to prior microhemorrhage (Fig 3). The only abnormality detected on the short-repetition-time images was a right occipital lobe hematoma, which resulted from placement of one occipito-temporal probe (Fig 4). There was no enhancement of any of the tracts in the 19 patients who had gadopentetate dimeglumine-enhanced scans after probe removal.

Discussion

In studies performed before the present MR imaging era, invasive monitoring of medically intractable epilepsy was found to provide additional information and potentially alter surgical management in one-third to one-half of patients so studied (1, 2). Depth electrode implantation may provide more precise location of an epileptogenic region than scalp EEG data in patients in whom noninvasive evaluation is nonconcordant. Invasive monitoring may also detect unsuspected multiple epileptogenic foci. This allows better surgical management, and more complete resection of the seizure focus while creations of neurologic deficits are minimized.

The risks of depth electrode placement are primarily those of intracranial infection and hemorrhage. The reported incidence of infection is 5% (1) and is usually manifested as a meningitis, and more rarely as an encephalitis. Although we did not detect any parenchymal changes to suggest infection, our evaluation for infection was limited by both the lack of gadopentetate dimeglumine administration in 38 patients and the

long time interval between probe removal and MR scanning. Although most patients with meningoencephalitis would have been diagnosed and treated in the short term, none showed clinical evidence of infection. One patient developed an occipital lobe hematoma after probe placement, presumably the result of a small venous laceration. Avoidance of the vascular structures is key to minimizing this complication. In the past, we used arterial, venous, and bony anatomy from preoperative angiography to elucidate the surface venous anatomy and arterial anatomies. Recently, we have supplemented conventional angiography with a two-dimensional time-of-flight MR angiogram of arterial and venous anatomy with the patient in a stereotactic frame. By superimposing the anatomic and angiographic information, we hope to further reduce the small incidence of probe-related hemorrhage.

The pattern of linear hyperintensities seen along the majority of probe tracts on the longrepetition-time images is consistent with mild gliotic changes, although histologic confirmation was not available. We realize there might be a systematic bias in this study in those patients imaged after large brain resections, because the evaluation of those areas that were resected would be limited. This occurred in patients who undersent anterior temporal lobectomy and radical hippocampectomy (n = 30) when we were evaluating occipitotemporal depth electrodes (n = 30). However, we were able to image the posterior aspect of these depth electrode tracts, and in those cases in which surgery had not been performed, it was unusual to see signal abnormality localized to only one region of the tract. Therefore, we feel that it is unlikely that a significant systematic undercounting of the incidence of these changes resulted from interval surgery.

The pattern of punctate signal voids, seen in association with four probe tracts, was most likely related to microhemorrhage. An alternative cause for this latter pattern might be metal deposition within the brain parenchyma from alloy impurities within the probes themselves. This seems less likely, given the relative infrequency with which this pattern was observed.

The lack of enhancement of the brain or pia meter in the 19 patients who received intravenous gadopentetate dimeglumine may be a reflection of the relatively long interval between probe placement and MR imaging, which averaged 18.3 months. Ten of these 19 were imaged within 1 year. Elster and DiPersio (5) demonstrated in a



Fig. 4. Significant hemorrhage complicating probe placement. CT with acute hematoma surrounding the right occipitotemporal probe posteriorly (A); sagittal (600/11/1) (B), and axial (2000/80/1) (C) images obtained 1 week after hemorrhage and probe removal demonstrate evolving occipital hematoma.

group of 46 patients who had undergone major intracranial surgery that enhancement of the brain or pia mater does not normally last beyond 1 year. On the basis of this data, one might predict normal enhancement in probe tracts in the immediate postimplantation period, which could be difficult to distinguish from infection.

In summary, one should expect either a normal study or punctate hyperintensities on long-repetition-time images after depth electrode placement in the vast majority of cases. Anything else is abnormal. Evidence of significant hemorrhage or infection related to probe placement is rare. In those patients who are imaged after depth electrode placement, one should not confuse these

punctate hyperintensities with the source of seizure activity.

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