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Original Research

EEG-Triggered Functional MRI in Patients With Pharmacoresistant Epilepsy

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Functional magnetic resonance imaging (fMRI) triggered by scalp electroencephalography (EEG) recordings has become a promising new tool for noninvasive epileptic focus localization. Studies to date have shown that it can be used safely and that highly localized information can be obtained. So far, no reports using comprehensive clinical information and/or long-term follow-up after epilepsy surgery in a larger patient group have been given that would allow a valuable judgment of the utility of this technique. Here, the results of 11 patients with EEG-triggered fMRI exams who also underwent presurgical evaluation of their epilepsy are given. In most patients we were able to record good quality EEG inside the magnet, allowing us to trigger fMRI acquisition by interictal discharges. The fMRI consisted of echoplanar multislice acquisition permitting a large anatomical coverage of the patient's brain. In 8 of the 11 patients the exam confirmed clinical diagnosis, either by the presence (n = 7) or absence (n = 1) of focal signal enhancement. In six patients, intracranial recordings were carried out, and in five of them, the epileptogenic zone as determined by fMRI was confirmed. Limitations were encountered a) when the focus was too close to air cavities; b) if an active epileptogenic focus was absent; and c) if only reduced cooperation with respect to body movements was provided by the patient. We conclude that EEG-triggered fMRI is a safe and powerful noninvasive tool that improves the diagnostic value of MRI by localizing the epileptic focus precisely. J. Magn. Reson. Imaging 2000;12:177–185. © 2000 Wiley-Liss, Inc.

Index terms: epilepsy; epileptic focus; functional MRI; EEG

PRECISE LOCALIZATION of the epileptic focus is a prerequisite for good surgical outcome in patients with pharmacoresistant epilepsy. This is still a challenge, especially in extratemporal lobe epilepsy, in which localization and extent of the epileptogenic zone are more difficult to determine. In these patients, surgical outcome is less favorable compared with patients with temporal lobe epilepsy (1). Several noninvasive imagery methods are routinely used in the presurgical evaluation of epilepsy, such as positron emission tomography (PET) or single-photon emission computer tomography (SPECT). Although PET and SPECT, in particular ictal SPECT, has become a useful tool for localization of the epileptic focus (2–4), these techniques are rarely sufficiently precise in patients with extratemporal epilepsy (5). Newer analysis procedures, such as comparison of ictal and interictal SPECTs, will show whether the yield of these techniques can be augmented (6).

Recently, several case reports using functional MRI (fMRI) during simple partial seizures reported blood oxygenation level-dependent (BOLD) effects concordant with the brain regions harboring the epileptic focus (7–10). However, prolonged seizure activity that is not accompanied by body movements and thus allows a good quality MRI examination, is rarely found in clinical practice. More often, interictal epileptic electrical activity, such as spikes, can be observed in scalp electroencephalography (EEG). By recording the EEG in the magnet (11), fMRI acquisition can be triggered by these spikes in order to visualize brain regions related to the generation of the spikes (12–14).

To date, no patient series and postoperative outcomes with respect to localized regions using fMRI have been presented. Here, we report on the results of 11 patients with pharmacoresistant epilepsy who were investigated in our laboratory using presurgical evaluation of epilepsy with a comprehensive battery of more established exams as well as EEG-triggered fMRI. Our fMRI findings were compared with the results of the presurgical workup as well as with intracranial recordings, if they could be performed.

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MATERIALS AND METHODS

Subjects

Eleven patients (mean age 26.5 years, range 13–41 years) participated in the present study. They suffered from pharmacoresistant epilepsy and were referred to our laboratory for presurgical epilepsy evaluation. The battery of exams applied during the (noninvasive) phase I workup includes a high-resolution MRI, long-term video-EEG monitoring, neuropsychological testing during the postictal and interictal period, PET, and, if possible, ictal and interictal SPECT. Table 1 gives patient clinical data, such as age at evaluation, age of epilepsy onset, MRI abnormalities, and nuclear imagery and EEG findings. Epileptic foci were localized as follows: in the frontal region in two patients, in the posterior region in five, in the anterior temporal region in one, and in the anterior temporal and frontal region in one. One patient suffered from multifocal epilepsy and one from a genetic epilepsy syndrome. Informed consent was obtained for all subjects in accordance with the protocol accepted to the ethics committee of our institution.

EEG Recording and Analysis

Sixteen gold-coated silver scalp electrodes (Neuroscan, Sterling, VA) were applied with conductive paste according to the standard positions of the 10/20 system. Data were recorded continuously on a 64-channel EEG machine (Deltamed, Paris, France) with a sampling rate of 128 Hz. The EEG was displayed on a 14-inch monitor in a bipolar montage, minimizing noise artifacts that potentially interfere with on-line interpretation.

During EEG recording, the EEG monitor was placed next to the MRI console to allow rapid manually triggered image acquisition by an experienced EEG reader. In a first series of 40–60 images, the acquisition was initiated whenever a discharge was noted on the screen ("activated condition"). Another set of 60 images was obtained after intravenous injection of 1–1.5 mg of Clonazepam, which eliminated all or the majority of discharges on the EEG ("control condition"). For this data set, the images were acquired every 15 seconds to ensure fully relaxed image acquisition.

The recorded EEG was reviewed off-line to reject those sequences unrelated to clear epileptiform activity.

MRI

A 1.5-T ECLIPSE system (Marconi Medical Systems, Cleveland, OH), equipped with fast gradients (26 mT/m with a slew rate of 72 mT/m/msec), was used that permits single-shot echoplanar imaging (EPI). The standard head coil configuration was used (body coil transmit, head coil receive). The following sequence parameters were used: spin echo (SE) with TR/TE 400/16 msec; gradient echo (GRE) with TR/TE 15/4.47 msec, flip angle 25°; single-shot EPI with TE 40 msec, flip angle 90°, 11–15 contiguous 5-mm slices. In one patient, multishot EPI was used with TR/TE 552/40 msec, number of slices 6, slice thickness 6 mm, gap 1 mm.

MRI Analysis

Data analysis was performed off-line using Interactive Data Language (Research Systems, Boulder, CO) on a DEC alpha UNIX station. The analysis was carried out by two of us (F.L., I.Z.) who were blinded to the morphological MRI results or other clinical findings. All studies were first corrected for head motion (15). An intensity threshold of 20% of the maximum image intensity was used to remove the noise. The skull signal was removed manually. After images with motion artifact or those that were triggered improperly were removed, the images corresponding to the two conditions were compared statistically using a cross-correlation computation of each individual pixel with a two-level (activated/control) step function as reference (16). Bonferroni correction was applied to eliminate false positives derived from multiple comparisons. The remaining pixels showing statistically significant signal enhancement were considered and displayed as signal percent change from the baseline. Stand-alone pixels were discarded. For interpretation of the results, only clusters of more than 10 pixels were considered. The fMRI results are displayed using a color scale representing percent change of signal intensity.

RESULTS

No patient reported discomfort of any kind. Recording of the EEG inside the magnet allowed the reliable detection of typical spike activity and (consecutively) proper fMRI acquisition related to these spikes.

Figure 1 compares the standard 16-channel recordings made outside the magnet (Fig. 1a) with the 16-channel EEG recorded inside the magnet (Fig. 1b). Characteristic spike activity in the left temporal lobe (patient #11) can be seen in both recordings (straight arrows) and was used to trigger the EPI acquisition. fMRI analysis revealed signal enhancement adjacent to the lesion (Fig. 1c). However, a cardiac pulse-synchrone artifact was seen in several EEG channels (eg, C3–P3, curved arrow in Fig. 1b), which corresponds to the ballisto-cardiogram as described by Ives and coworkers (11). It was observed in some, mostly rather lean patients, but did not prevent spike identification except in one patient.

Overall, in 8 of the 11 patients, EEG-triggered fMRI confirmed the clinical diagnosis either by the presence ($n = 7$) or absence ($n = 1$) of distinct areas of signal enhancement. The results are summarized in Table 2. In 5 patients (#1, 3, 4, 8, and 11), the results could be confirmed by intracranial EEG recordings. Patient #1 was extensively discussed in a case report (13). Essentially three pixel groups were found, ie, in the left, right frontal, and mesial frontal structures. EEG source localization techniques, extensively discussed by Michel et al (17), revealed onset of these discharges in the left frontal lobe. Focus localization in the left frontal lobe was also suggested by the other phase I exams and was finally confirmed by subdural electrode recordings.

Figure 2 is a volume rendering image that shows the superposition of the area of signal enhancement in the

Table 1
Patient Data

Patient no.	Age (yr)	Age of onset (yr)	MRI abnormality	Scalp EEG	PET[a]	SPECT[b]	Neuropsychology
1	18	7	Suspicion of left dorsolateral frontal dysplasia; otherwise normal	Interictal: mostly left frontal parasagittal, rarely right frontal; ictal: left frontal onset	Left frontal hypermetabolism	Not performed	Multifocal deficits with predominance of left hemispheric deficits
2	36	31	Transitory left occipital hyperintensity on FLAIR images (small dysplastic lesion)	Interictal: left temporo-occipital spikes, ictal: left posterior onset	Left occipital	Left occipital	Aphasic signs, verbal memory deficits
3	41	3	Right temporal porencephalic cavity, atrophy of the residual right temporal lobe and hippocampus	Interictal: right occipital and right temporal spikes, ictal: right temporal-occipital onset	Extensive region in the right posterior quadrant	Not performed	Personality disorder with schizoid thoughts
4	14	1	Suspicion of left occipital and left frontal dysplasia	Interictal: right temporo-occipital > right and left frontotemporal discharges, ictal: nonlateralizing, left and right hemispheric onset	Right temporo-occipital and right mesiofrontal	Right lateral temporal	Multifocal deficits, moderate mental retardation
5	28	21	Right > left hemispheric cortical atrophy	Interictal: continuous right temporoparietal slowing, ictal: right temporoparietal onset	Right parietal	Right posterior temporoparietal	Normal
6	23	14	Microgyria in the left basal and lateral aspect of the frontal lobe	Interictal: mostly left temporal, rare right temporal spikes, ictal: left temporal or bifrontal onset	Left temporal	Left temporal	Frontal deficits, discrete verbal memory
7	28	11	None	Interictal: no discharges, ictal: left posterior, left hemispheric or nonlateralizing onset	Left temporo-occipital	Left temporooccipital	Discrete left hemispheric deficits; otherwise normal
8	41	17	Left frontal insular atrophy	Interictal: left anterior to mid-temporal, rare right temporal spikes ictal: left mid- to posterior temporal onset	Left mid- to posterior temporal	Lateral aspect of the left temporal lobe	Left temporoparietal dysfunctions, verbal and visuospatial memory deficits
9	16	2	Discrete right frontal atrophy; otherwise normal	Interictal: mostly right frontoparietal spikes, more rarely right, left temporal, left frontoparietal; ictal: right frontal onset	Right frontotemporoparietal	No hyperperfusion visualized	Moderate mental retardation, discrete right frontal deficits
10	13	8	Discretely enlarged left temporal horn	Interictal: generalized with right predominance, ictal: without lateralization or right hemispheric onset	Right > left temporoparietal metabolism	Right lateral temporal	Discrete visuospatial memory deficits, otherwise normal
11	33	12	Left temporal porencephalic cavity, with residual anterior temporal and lateral neocortex, absence of the amygdala and hippocampus	Interictal: left anterior and midtemporal spikes, ictal: left temporal onset	Left temporal	Discrete enhancement of residual left temporal tissue	Normal

[a]Area of hypometabolism.
[b]Area of hyperperfusion (ictal SPECT in comparison with the interictal SPECT).

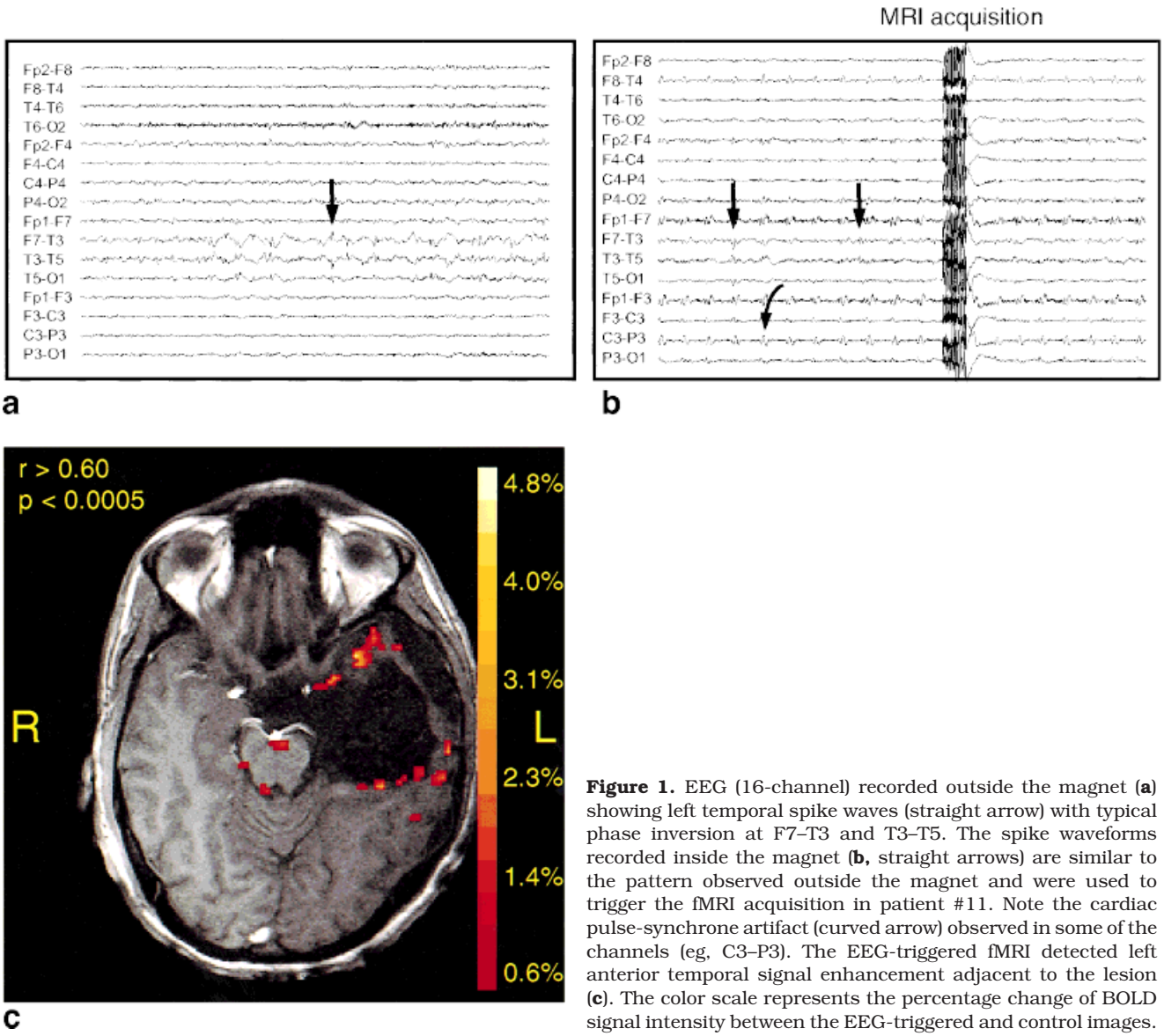

Figure 1. EEG (16-channel) recorded outside the magnet (**a**) showing left temporal spike waves (straight arrow) with typical phase inversion at F7–T3 and T3–T5. The spike waveforms recorded inside the magnet (**b,** straight arrows) are similar to the pattern observed outside the magnet and were used to trigger the fMRI acquisition in patient #11. Note the cardiac pulse-synchrone artifact (curved arrow) observed in some of the channels (eg, C3–P3). The EEG-triggered fMRI detected left anterior temporal signal enhancement adjacent to the lesion (**c**). The color scale represents the percentage change of BOLD signal intensity between the EEG-triggered and control images.

EEG-triggered fMRI with the subdural electrodes related to seizure onset in the left frontal lobe in this patient. The discrete discordance between both areas is probably because the seizure focus seems to be localized in the depth of the frontal sulcus. In these cases, the maximal activity as recorded on the surface might be somewhat displaced due to neuronal or volume conduction, ie, not reflecting the activity directly underneath. This potential limitation of the localizing value of subdural recordings is well recognized (18,19). A subtotal resection of the left frontal lobe including most of the area of signal enhancement was carried out, sparing its posterior part due to the presence of speech-related cortex as determined by electrical stimulation. Surgical intervention resulted in a more than 80% seizure reduction, which was considered significant in a patient with up to three seizures a day and frequent secondary generalizations.

Figure 3 illustrates the findings in patient #4, who suffered from multifocal epilepsy together with moderate mental retardation. A seizure frequency with up to 10 partial seizures and one to two secondary generalizations a week was noted. Ictal scalp recording showed no consistent focal onset; in the interictal EEG, frequent spikes over the right mid- to posterior temporal region but also over the left temporal and frontal regions were seen. Imagery (MRI, PET, ictal and interictal SPECTs), as well as neuropsychology showed left and right hemispheric dysfunctions. Depth electrode recordings revealed independent left and right temporal seizure onset with predominance of the right side and rapid propagation to ipsi- and contralateral frontal and temporal regions. Although interictally most spikes were found in the posterior aspect of the right temporal neocortex, thus confirming the scalp EEG data, seizure onset implicated the mesial temporal structures. Apart from the right posterior temporal spikes, somewhat fewer independent spikes were seen in the left temporal as well as mesial and lateral frontal structures during depth EEG recording. These findings may correspond to the multiple areas of signal enhancement in the frontal and temporal lobes; however, the strongest signal

Table 2
Findings on Examination

Patient no.	Age (yr)	Age of onset (yr)	Epileptic focus location	fMRI-EEG localization	Intracranial EEG	Surgery
1	18	7	Left frontal	Left frontal	Chronic subdural electrodes	Yes
2	36	31	Left temporo-occipital	Left occipital (calcarine fissure)	Not performed	No
3	41	3	Right occipitotemporal	Right occipital and temporal, adjacent to the cavity	Perioperative electrocorticography	Yes
4	14	1	Multifocal with predominance of the right posterior temporal region	Multifocal, but with predominant right posterior	Depth electrodes	Yes
5	28	21	Right parietotemporal	Right posterior temporal	Not performed	No
6	23	14	Left temporal and left frontal	No focal signal enhancement	Depth electrodes	Yes
7	28	11	Left posterior temporal	No focal signal enhancement	Not performed	No
8	41	17	Left posterior temporal	Left posterior temporal	Subdural electrodes	No
9	16	2	Right frontal	No focal signal enhancement	Not performed	No
10	13	8	Diffuse (genetic disorder)	No focal signal enhancement	Not performed	No
11	33	12	Left temporal	Left anterior and mid temporal SE, adjacent to cavity	Perioperative electrocorticography	Yes

enhancement was noted in the right posterior cortex. Although epileptogenic discharges were frequently seen in this area as well as metabolic and perfusion changes, no structural MRI anomaly could be detected.

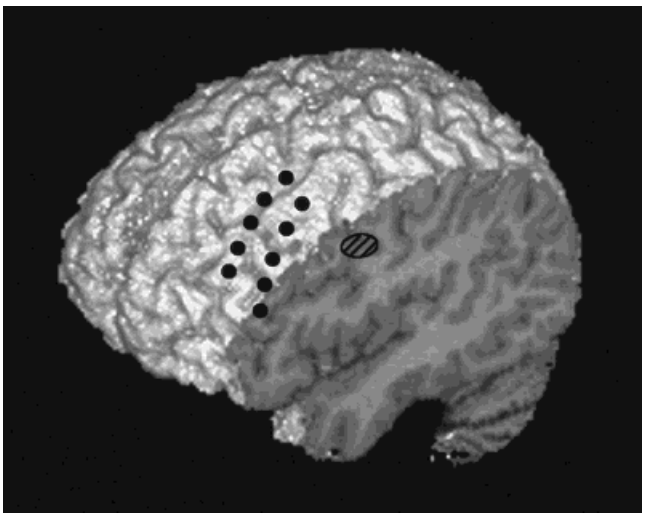

Figure 2. Volume rendering brain image of patient #1 showing the area of signal enhancement (circle) found by EEG-triggered fMRI. This area corresponds well with the epileptogenic region as found by subdural EEG recording using a 64-electrode grid. Only the electrodes showing epileptic activity are shown (black dots).

Based on the depth electrode findings, suggesting a major implication of the right mesial temporal structures, and the fact that resection of the temporo-occipital lobe would result in a major neurological handicap, it was decided to carry out a palliative anterior 2/3 temporolobectomy. During the first 3 postoperative months, a major seizure reduction was observed. After this period, the frequency of the complex partial seizures rose again to almost the preoperative seizure frequency; however, a major reduction in secondary generalized seizures persisted (one every 2 months or less). The postoperative result only partially confirms the fMRI findings: since the most suspicious area was not resected, this may explain the continuation of complex partial seizures. However, the patient suffered from multifocal epilepsy, a seizure disorder that is difficult to treat surgically.

In two patients (#3 and 11), presurgical evaluation suggested that the focus lay adjacent to a porencephalic cyst. In patient #3, a 41-year-old woman, the porencephalic cyst resulted from an operation for a meningioma in the right posterior region at the age of 11, which did not relieve her seizure disorder. Apart from the porencephalic cyst, residuals of the right hippocampus, temporal neocortex, and occipital tissue were seen on MRI. EEG and nuclear imagery suggested both temporal and occipital dysfunction independently, which was confirmed by the fMRI findings and perioperative electrocorticography. Figure 4 illustrates the findings in patient #3; areas of acti-

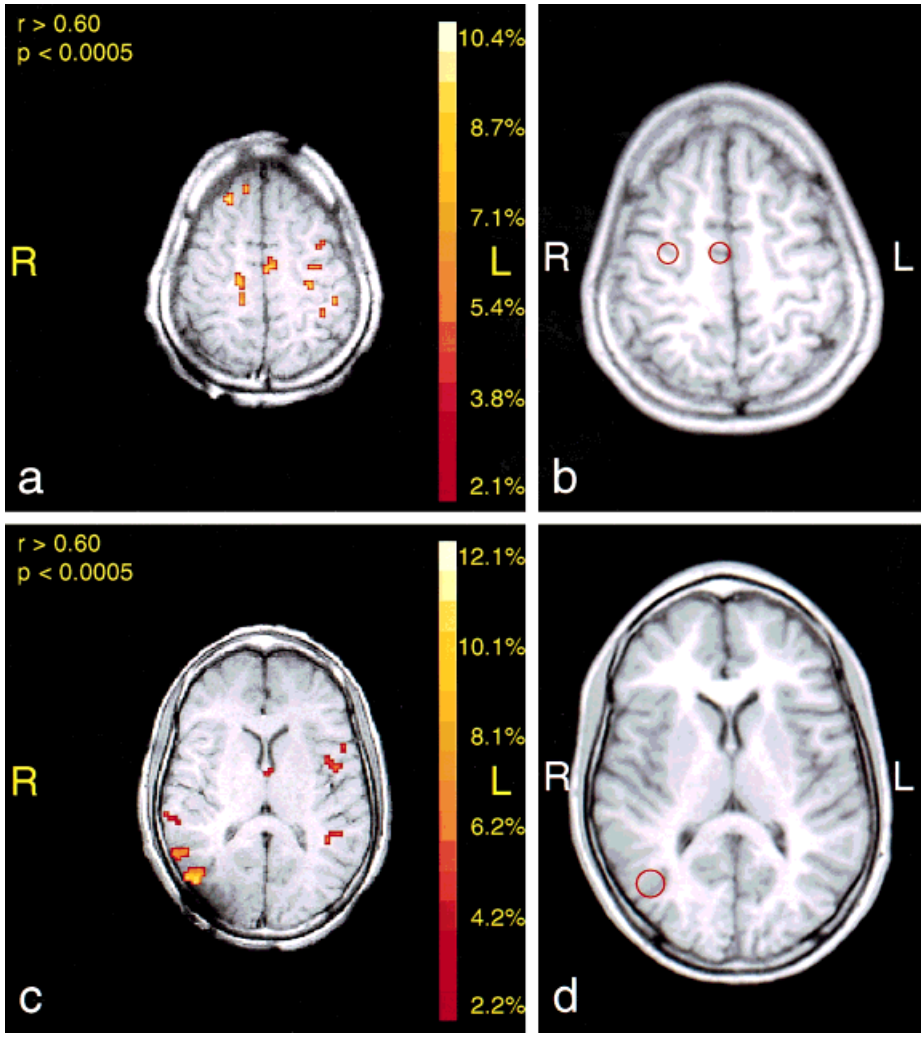

Figure 3. Patient #4: The multiple areas of signal enhancement found by EEG-triggered fMRI (**a** and **c**) corresponded well with activities found by depth EEG recording (**b** and **d,** circles). Temporal analysis of the EEG found spike onset in the right posterior temporal region (d).

vation around the lesion can be seen, with a predominance in the anterior region corresponding to residual temporal neocortex and hippocampal tissue. Surgery included removal of the remnants of temporal and occipital tissue, which has rendered the patient seizure free for more than 1 year.

The MRI of patient #11 also revealed a porencephalic cavity, due to factor V Leiden mutation, which was located in the left anterior temporal lobe (Fig. 1c). The residual anterior and mid-temporal tissue appeared atrophied and dysplastic, and the absence of the left amygdala and hippocampus was noted. Very frequent spikes in the anterior and mid-temporal region allowed EEG-triggered fMRI examination. Signal enhancement was found around the lesion, ie, anterior, lateral, and posterior to the cavity. EEG source localization, as used in patient #1, showed that the discharges originated in the anterior aspect and propagated to the tissue posterior to the cyst. Abundant spikes from the residual lamina were confirmed by intraoperative electrocorticography; these were less frequent from the posterior margin of the lesion. After resection of the lamina, leaving the posterior margin of the lesion intact, the patient has been seizure free (follow-up at 1 year).

Patient #2 had frequent simple partial seizures consisting of visual phenomena in the right visual field. The presurgical workup allowed the unequivocal diagnosis of left occipital epilepsy. Figure 5a represents the fMRI findings, which were concordant with the presence of a transient hyperintense signal in the lower part of the calcarine fissure as retrieved by structural MRI (Fig. 5b). Follow-up MRI showed disappearance of the hyperintense signal; it was concluded that the hyperintense signal reflected a transitory edema after prolonged seizure activity, probably related to an underlying dysplasia (20–22). Due to major neurological deficits after a possible operation and the fact that most of the patient's seizures are of the simple partial type, surgery was not considered an option, and drug treatment was pursued. Although in this patient no invasive EEG data were obtained, the clinical picture and the results of all

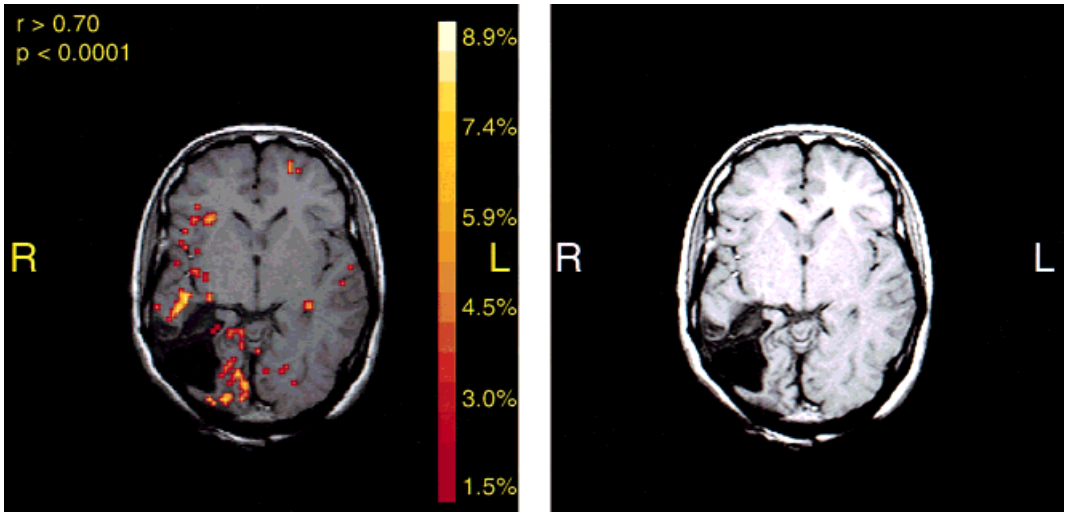

Figure 4. Patient #3: EEG-triggered fMRI results demonstrating remaining area of activation around the porencephalic cavity corresponding to epileptic electrical activity as demonstrated by perioperative electrocorticography.

exams were in agreement and in concordance with the fMRI findings.

Patient #5 suffered from frequent somatosensory simple partial seizures of the left hemibody, in most cases starting in the left hemiface. Structural MRI revealed global cortical atrophy with discrete predominance of the right hemisphere. In ictal and interictal EEGs as well as in nuclear imagery exams, a right temporo-parietal dysfunction became evident. EEG-triggered fMRI revealed a right posterior area of signal enhancement located at the medial temporal gyrus according to the Talairach and Tournoux atlas (23), which most likely corresponds to the presumable site of epilepsy onset as determined by phase I exams and the seizure semeiology. Invasive evaluation was first proposed but finally was not performed because the patient became seizure free under a newly introduced bitherapy.

In one patient (#10), no distinct area of signal enhancement was noted, although the workup suggested a right hemispheric or right temporal dysfunction. It turned out that this patient suffered from a rare genetic disorder (ring chromosome 20), which seems not to be related to a discrete dysfunctional brain region, thus—on the basis of the absence of focal findings—confirming the clinical diagnosis. Brain surgery does not seem to be helpful in this condition (24), so this treatment option was abandoned.

Overall, in 3 of the 11 patients, localization of the epileptic focus was not possible. In one case (patient #9) the ballisto-cardiogram artifact, together with strong movement artifacts, prevented proper spike identification and therefore the data could not be analyzed. In the other two cases, focus identification was hindered by the absence of interictal epileptogenic activity (patient

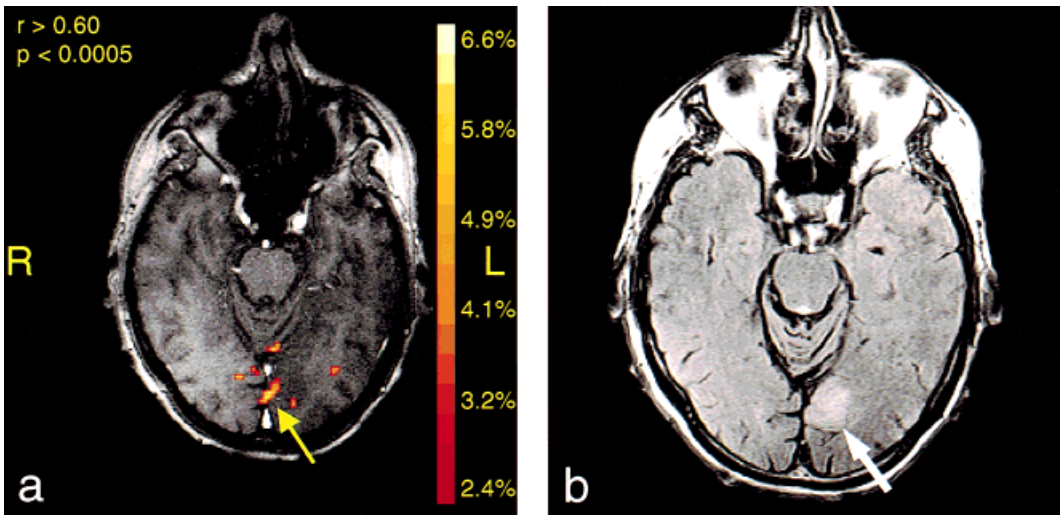

Figure 5. Patient #2: EEG-triggered fMRI obtained during frequent simple partial seizures causing visual phosphenes in the upper right visual field. The fMRI finding (**a**) was confirmed by MRI signal abnormality (arrows) obtained with a FLAIR sequence (**b**). The FLAIR sequence parameters were as follows: inversion time 1700 msec, TR/TE 7000/83 msec, echo train length 8.

#7) or a basal focus localization too close to frontal and temporal air cavities (patient #6).

DISCUSSION

In most patients, fMRI triggered by interictal epileptic discharges detected a region of blood flow changes that converged with the epileptogenic focus as determined by a comprehensive battery of noninvasive and (if indicated) invasive exams. Areas in the frontal, occipital, and parietal lobes were reliably detected, with the exception of areas too close to air cavities. Since this is particularly true for the most frequent focal epileptic syndrome, mesial temporal epilepsy, this type of epilepsy is less well suited for EEG-triggered fMRI examination. However, mesial temporal epilepsy is now well described (25) and easily recognized with most routine imagery techniques. Probably due to its good definition, postoperative outcome is usually considered very good, rendering up to 70%–80% of the patients seizure free (1).

It is well known that patients with extratemporal lobe epilepsy, even if they are explored invasively, have lower chances of becoming seizure free postoperatively (1). A precise focus localization is still difficult to obtain, in particular in the absence of an MRI lesion, which may explain the overall worse postoperative outcome in this patient group. Our results demonstrate that fMRI, triggered by interictal discharges, represents a promising tool to help define the epileptic focus in these particular patients. Except for transient fatigue after benzodiazepine injection, no discomfort was reported.

In some cases, areas of signal enhancement extended to adjacent or remote brain regions, leaving open the fundamental question of which area is the most relevant. Additional analysis of the EEG, eg, with EEG source localization techniques, help to define the spatiotemporal pattern of spike origin and propagation (17).

Several limitations were encountered. Like other functional MRI exams, EEG-triggered fMRI is very sensitive to motion. Different approaches have been proposed to correct for motion (15). In our study, we fixed the patient's head either by a bandage on the scanner's head holder or by using a vacuum cushion. Furthermore, we used the automated image registration method, which proved to be accurate to detect motion in a subvoxel range (26,27). Nevertheless, image registration cannot completely recover functional information due to interpolation error and saturation effect on image intensity (28,29). In particular, in patients with mental retardation and/or small children who cannot cooperate sufficiently, movement artifacts represent a major obstacle toward meaningful data acquisition. This could be overcome with sedation (without abolition of the epileptogenic discharges) but would need complementary medical staff on stand by.

Focus localization simply with fMRI is probably not useful if performed on patients not having interictal discharges, even when an active epilepsy can be presumed (ie, repetitive seizures despite drug treatment). Most likely the underlying neurophysiological abnormalities do not recruit enough tissue to generate visible spikes, and thus, the associated signal enhancement may also disappear in the noise of the fMRI data. Since this observation is based on one case, larger patient populations are needed to determine whether fMRI, without EEG triggering, has the potential to localize the focus "blindly."

As already stated above, an important limitation of the method is the sensitivity of long echo time EPI to susceptibility artifacts. The MR signal amplitude of structures lying close to the air-tissue interface is drastically reduced. Anatomic regions that are suspected to be the most affected are the inferior frontal lobe and the anterior and basal temporal lobes. In these cases, other MR sequences are less sensitive to susceptibility artifacts, eg, the PRESTO sequence (30). This sequence has been used in functional imaging of the human visual cortex (31) and needs to be evaluated in future studies.

Despite the limitations of the EEG-triggered fMRI, we believe that it is a potentially valuable tool and should be applied in particular for patients with nonlesional extratemporal epilepsy and frequent discharges. In these cases, the more established (noninvasive) tests often fail to render precise focus localization and, consequently, surgical outcome may not be optimal. Even placement of intracranial electrodes could be done in a more tailored fashion, provided that the most relevant structures are known.

CONCLUSIONS

It is possible to detect interictal epileptogenic activity safely inside a 1.5-T MRI magnet. EEG-triggered fMRI reveals discrete areas of activation in patients with extratemporal lobe epilepsy who have frequent interictal discharges. The technique is strictly noninvasive and allows precise focus identification in adult and pediatric patients, in particular in patients with extratemporal epilepsy, who are known to be difficult surgical candidates. It is less useful in patients whose seizure focus is too close to air cavities, who have too infrequent discharges, or who have difficulties in remaining motionless inside the magnet.

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