High-Resolution Surface-Coil MR of Cortical Lesions in Medically Refractory Epilepsy: A Prospective Study

P. E. Grant, A. J. Barkovich, L. L. Wald, W. P. Dillon, K. D. Laxer, and D. B. Vigneron

PURPOSE: To determine the role of surface-coil MR imaging in evaluating medically refractory neocortical partial epilepsy. **METHODS:** A prospective study of 25 patients with medically refractory neocortical partial epilepsy was performed. Head- and surface-coil images were reviewed by two neuroradiologists to determine the clarity with which cortical lesions were depicted. The ability of imaging, combined with surface electroencephalograhy (EEG), to locate the suspected epileptogenic zone was evaluated. **RESULTS:** Compared with head-coil studies, surface-coil studies showed four more lesions, caused the most probable diagnosis to be altered in five patients, and better defined the lesions in four patients. Of 11 patients with lobar EEG abnormalities, imaging showed focal cortical abnormalities within the same or adjacent lobe in five and multifocal abnormalities in two. Of six patients with EEG abnormalities restricted to two adjacent lobes, imaging showed focal cortical abnormalities in one of these lobes in five patients and multifocal abnormalities in one patient. Of eight patients with a nonfocal EEG, imaging showed focal cortical abnormalities in five and multifocal cortical abnormalities in one. In two of 13 patients, video/EEG telemetry improved seizure location whereas surface-coil imaging showed focal cortical lesions in six and provided relevant prognostic information in five. **CONCLUSION:** Compared with head-coil studies, surface-coil imaging of the cerebral cortex improved detection and differentiation of focal cortical lesions in 64% of patients. Video/EEG telemetry improved location in 15% of patients, and surface-coil imaging combined with EEG results provided improved location of the suspected epileptogenic zone or relevant prognostic information in 85%.

Index terms: Magnetic resonance, surface coils; Seizures

It is estimated that in the United States approximately 400 000 persons have poorly controlled partial seizures (1). Recurrent, poorly controlled seizures can disrupt quality of life and, in children, can severely impair development (2–5). To improve outcome in medically refractory seizures, surgical intervention is considered. Magnetic resonance (MR) imaging has begun to play an important role in identifying the presumed epileptogenic zone by identifying cortical abnormalities that lie within the region of an electrophysiological abnormality (6). In fact, the degree of resection of the cortical abnormality defined on MR images is a more important factor in surgical outcome in neocortical epilepsy than is the degree of resection of the apparent seizure focus defined by scalp or invasive electroencephalography (EEG) (7–9). Conversely, if an MR examination reveals multiple cortical abnormalities or a large region of cortical involvement or involvement of eloquent areas of brain, surgical outcome is worse (7, 8). Thus, the goal in performing MR imaging is to maximize our ability to detect and define the extent of cortical lesions and to help determine the presence of multiple abnormalities.

Recently, a technique was described that improves visibility of focal cortical abnormalities through the use of high-resolution volumetric MR imaging techniques with isotropic voxels and multiplanar reformations (10). In that study, which was performed with a routine head coil, the limiting factors were signal-to-noise

ratio and postprocessing time. To improve the signal-to-noise ratio further in the cerebral cortex and subcortical white matter, we developed phased-array surface coils that are fitted to the head. The increase in signal-to-noise ratio obtained with these surface coils allows us to increase the spatial resolution and further optimize the contrast-to-noise ratio (11).

In this article we describe the use of phased-array surface coils in the examination of patients with medically refractory partial seizures that are not mesial temporal in origin. This excludes patients with features of mesial temporal sclerosis and means that our study was limited to patients with features of neocortical epilepsy. We evaluate our ability to detect and confidently diagnose lesions involving the cortex with the use of standard head- and surface-coil images and discuss the ability of surface-coil imaging to define the suspected epileptogenic zone further when combined with electrophysiological testing, and to provide useful prognostic information, such as the presence of multiple or extensive abnormalities.

Patients and Methods

Twenty-seven patients referred for MR evaluation of nonmesial temporal medically refractory partial seizures were examined between September 1994 and November 1995. Two examinations were omitted because of motion artifacts. The remaining 25 patients, eight male and 17 female, 1 to 44 years old (average age, 23 years), constitute our study group.

MR examinations were performed on one of four 1.5-T imagers capable of phased-array imaging from four independent receivers. All patients initially underwent a routine MR examination with a quadrature head coil that included an axial dual-echo T2-weighted sequence with parameters of 2500/30,80/0.75 (repetition time/echo time/excitations), 256 × 192 matrix, 5-mm section thickness, and 2.5-mm skip. An axial spin-echo sequence with parameters of 500/minimum/2, 256 × 192 matrix, 5-mm section thickness, and 1-mm skip was performed in two patients; and a coronal volumetric three-dimensional Fourier transform (3DFT) gradient-echo sequence with radio frequency spoilers used to eliminate steady-state magnetization was performed in the remaining 23 patients. The volumetric 3DFT gradient-echo sequence was obtained with the following parameters: 33–36/minimum/0.75–1, theta of 35, 256 × 192 matrix, 20 × 15 to 22 × 16-cm field of view (FOV), and 124 partitions. With a partition size of 1.5 mm, this resulted in a resolution of 0.78 to 0.86 mm in plane and a voxel size of 0.92 to 1.07 mm³.

Either at the same time as or within 1 month of the head-coil study, all patients were examined with high-

resolution phased-array surface coils built specifically for evaluating cortical lesions (11). Four types of phased-array coils were available: 1) two independent figure-eight coils used in quadrature for the vertex, 2) four overlapping phased-array coils for the side of the head, 3) two overlapping circular coils for bilateral imaging of the parietal and temporal lobes, and 4) four overlapping octagonal coils curved to wrap symmetrically around the frontal or occipital lobes. Surface coils were operated in receive-only mode, with the body coil providing homogeneous excitation. Because of the limited coverage of the surface coils, two acquisitions with different coil positioning were occasionally used to make the entire brain visible. Otherwise, studies were limited to the region thought most likely to contain the epileptogenic focus. The presumed site of the focus was determined by a combination of clinical data (seizure semiology), electrophysiological data (EEG, videotelemetry), and, if the head-coil images were abnormal, the location of the abnormality on the head-coil images. In each case, a coronal volumetric 3DFT gradient-echo sequence was performed with the following parameters: 33/7/0.75, theta of 35 to 45, 256 × 224 matrix, 16- to 18-cm FOV, 16-Hz bandwidth, and 124 partitions. With a partition size of 0.7 to 1.0 mm, the resultant voxel size was 0.31 to 0.57 mm³, which is approximately one third the size of the voxel obtained with the head coil. In cases in which T2 abnormalities were identified on the head-coil studies, a fast spin-echo sequence (4000/112/4, 512 × 512 matrix, echo train of 8 to 16, 12- to 18-cm FOV, 3-mm section thickness) or a T2-weighted sequence (2500/81/0.75, 256 × 256 matrix, 12- to 18-cm FOV, 3-mm section thickness) was obtained with the surface coils. An automated intensity-correction algorithm, which divided the image by an edge-completed low-pass filtered version of the original image, was used to greatly reduce intensity variation caused by the reception profile of the coils (11).

Images were analyzed independently by two neuroradiologists for the presence, extent, and type of cortical abnormality. Volumetric images were studied on a console that allowed reformation of the data in any plane. All questionable regions were supplemented by T2-weighted fast spin-echo images that allowed better analysis of any regions of prolonged T2 relaxation in the cerebral cortex and underlying white matter. The images obtained with the use of the quadrature head coil were analyzed first and classified as normal, indeterminate, or abnormal. The indeterminate category included studies with findings of unknown clinical significance, such as foci of T2 prolongation in the deep white matter and gyri that looked abnormal in some planes but normal or nearly normal in others. If a site of abnormal brain was identified, its location and likely pathologic substrate (dysplasia versus neoplasm versus focal atrophy, for example) were recorded. The surface-coil sequences were analyzed after the head-coil studies in the same manner. If the two neuroradiologists disagreed, the images were reanalyzed and discussed until a consensus was reached.

The locations of hemispheric and lobar cortical lesions on the head- and surface-coil studies were compared with each other and with the location of the epileptogenic focus as determined by seizure semiology and electrophysiological testing. To date, six patients have had surgical resection of the presumed seizure focus and one patient has had a cortical biopsy. Imaging findings were compared with pathologic specimens and with postsurgical outcome in these patients.

Results

Epilepsy History and Electrophysiological Results

All patients had to have medically refractory partial seizures (either simple, complex, or both) to be entered into this study. They also had to have clinical and EEG features of neocortical epilepsy. In no patient was mesial temporal sclerosis suspected. The average age of seizure onset was 12 years (range, of 1.5 to 44 years). Seven patients were globally delayed (patients 2, 5, 10, 13, 14, 15, and 24) and 18 were developmentally normal. Three patients had mild focal neurologic abnormalities, including mild left-sided hemiparesis in patient 14, mild right-sided hemiparesis in patient 21, and mild aphasia in patient 7. Three had mild bilateral neurologic abnormalities (patients 13, 15, and 24). The remaining 19 patients were neurologically normal. Results of seizure semiology and interictal EEG performed with scalp electrodes were available in all cases. Videotelemetry was also performed in 13 patients, three with subdurally placed electrodes. Interictal EEG and seizure semiology located the ictal onset in one lobe in 11 patients and to two adjacent lobes in six patients. These results were defined as well-localized and poorly localized, respectively. In the remaining eight patients, the seizure focus could not be restricted to two adjacent lobes on interictal EEG or seizure semiology, and the results were defined as nonlocalizing. Video/EEG telemetry was performed in 13 patients and in only two patients was seizure location improved. Diffuse slowing over a region or spike wave activity was considered indicative of an epileptogenic focus in the corresponding region.

Head-Coil Results

Findings on head-coil studies were normal in seven patients, indeterminate in eight patients, and abnormal in 10 patients. In the indeterminate category, a gyrus with atypical morphology was detected in patient 10, but the cortical thickness, gray–white matter junction, and subcortical white matter were normal. Therefore, the significance of this finding was uncertain and this study was rated as indeterminate. In the other seven patients, the morphology of the cortex and the character of the gray–white junction were not adequately defined to either definitely include or exclude a cortical lesion (Fig 1A). In the 10 abnormal studies, the most probable diagnoses were neoplasm (in three), focal cortical dysplasia (in three), heterotopia (in two), both a focal cortical dysplasia and a heterotopia (in one), and a diffuse migration abnormality (in one). Results of head-coil studies are presented in the Table.

Surface-Coil Results

Findings on surface-coil studies were normal in five patients, indeterminate in two patients, and abnormal in 18 patients. A 2-mm-diameter cystic lesion was detected in the cortex of the right superior temporal gyrus of patient 1 but was of uncertain significance and therefore this examination was rated as indeterminate. The unusual gyrus in patient 10 was again identified, but as no abnormalities were detected in the cortical thickness, gray–white matter junction, or subcortical white matter, this study remained indeterminate. The most probable diagnoses in the abnormal studies included focal cortical dysplasia (in eight patients), heterotopia (in three), and diffuse cortical dysplasia (in two). One patient had both a focal cortical dysplasia and a heterotopia and was therefore counted twice. One patient in the focal cortical dysplasia category had multifocal areas of involvement. The most probable diagnoses in the remaining abnormal studies were neoplasm (two patients), encephalomalacia (two patients), nonspecific foci of T2 prolongation in the white matter (one patient), and vascular malformation (one patient). Results of surface-coil studies are also presented in the Table.

Comparison of Head-Coil and Surface-Coil Results

The results are grouped according to the interpretation of the head-coil studies. This em-
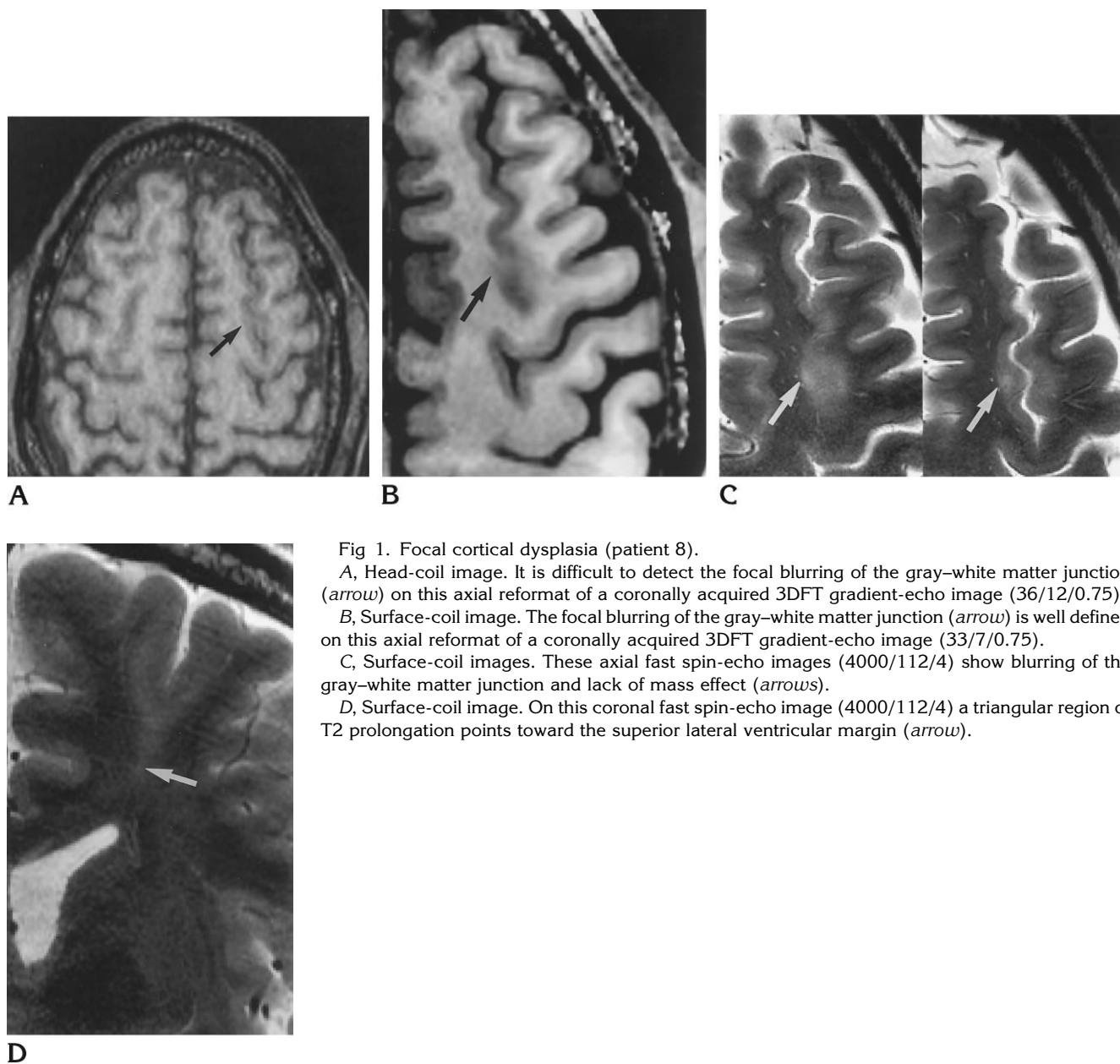
Fig 1. Focal cortical dysplasia (patient 8).

A, Head-coil image. It is difficult to detect the focal blurring of the gray–white matter junction (*arrow*) on this axial reformat of a coronally acquired 3DFT gradient-echo image (36/12/0.75).

B, Surface-coil image. The focal blurring of the gray–white matter junction (*arrow*) is well defined on this axial reformat of a coronally acquired 3DFT gradient-echo image (33/7/0.75).

C, Surface-coil images. These axial fast spin-echo images (4000/112/4) show blurring of the gray–white matter junction and lack of mass effect (*arrows*).

D, Surface-coil image. On this coronal fast spin-echo image (4000/112/4) a triangular region of T2 prolongation points toward the superior lateral ventricular margin (*arrow*).

phasizes the changes in interpretation that occurred with the additional information provided by the surface-coil studies.

Normal Head-Coil Study (Seven Patients).— New findings were detected in three patients (patients 1, 3, and 4) on surface-coil studies. In patient 3, a previously undetected small heterotopia was identified in the right posterior temporal lobe, resulting in abnormal findings on the surface-coil study. A focal cortical lesion less than 5 mm in diameter was identified in the left frontal lobe of patient 4 (Fig 2) that was difficult to detect on the head-coil images, even in ret-

rospect. Owing to the presence of subtle mass effect and sharpness of the gray–white junction, this lesion was suggestive of a neoplasm. In patient 1, a 2-mm-diameter cyst was identified in the right superior temporal gyrus, but the significance of this finding is unknown. This surface-coil study was rated as indeterminate. No abnormalities were detected in the remaining four patients on surface-coil studies.

Indeterminate Head-Coil Study (Eight Patients).—In four patients (patients 8, 11, 12, and 13), the surface-coil studies showed a blurred gray–white matter junction and absence

Electrophysiological, imaging, and pathologic findings in 25 patients with neocortical partial epilepsy

Patient	Location of Seizure Focus		Head-Coil		Surface-Coil		Pathologic Findings
	EEG/Seizure Semiology	Ictal Video/EEG Telemetry	Localization	Diagnosis	Localization	Diagnosis	
Localized surface EEG/semiology							
1	R temporal	R frontotemporal	Not localized	Normal	R temporal	Indeterminate	Hamartoma
2	R temporal	...	Not localized	Normal	Not localized	Normal	Normal
6	R occipital	R > L occipital	Not localized	Normal	Not localized	Normal	Normal
7	L central	...	Not localized	Normal	Not localized	Normal	Rasmussen's encephalitis
11	L temporal	Frontal, probably L	L frontal	Indeterminate	L frontal	FCD	FCD
14	R central	...	R parietal	Indeterminate	Not localized	Normal	Normal
17	R central	R central	R frontoparietal	FCD	Multifocal	MCD	FCD*
19	L parietal	...	L parietal	Tumor	L parietal	Encephalomalacia	
20	R frontal	R frontal	R frontal	FCD	R frontal	FCD	FCD
23	L temporal	...	L frontal	FCD	L frontal	Tumor	
25	L temporal	L temporal	Multifocal	Heterotopia	Multifocal	Heterotopia	Mesial temporal sclerosis
Poorly localized surface EEG/semiology							
4	R > L frontal	R > L frontal	Not localized	Normal	L frontal	Tumor	
8	L frontotemporal	L > R frontal	L frontal	Indeterminate	L frontal	FCD	
9	L frontotemporal	...	L frontal	Indeterminate	L frontal	FCD	
12	L frontotemporal	Not localized	L frontal	Indeterminate	L frontal	FCD	
21	L frontotemporal	...	L frontal	Tumor	R frontal	Vascular malformation	
22	Bilateral parietal	Bilateral parietal	R parietal, multifocal	FCD, heterotopia	R parietal, multifocal	FCD, heterotopias	
Nonlocalized surface EEG/semiology							
3	Not localized	Not localized	Not localized	Normal	R temporal	Heterotopia	
5	Not localized	...	Not localized	Normal	Not localized	Normal	
10	Not localized	...	L frontoparietal	Indeterminate	L frontoparietal	Indeterminate	
13	Not localized	Generalized	L frontal	Indeterminate	L frontal	FCD	
15	Not localized	...	Multifocal	Indeterminate	R parietal	Encephalomalacia	
16	Not localized	...	L frontal	Tumor	L frontal	FCD	
18	Not localized	...	R frontal	Heterotopia	Bilateral frontal	NSWM	
24	Not localized	Not localized	Bilateral diffuse	DCD	Bilateral diffuse	DCD	

Note.—FCD indicates focal cortical dysplasia; MCD, multifocal cortical dysplasia; DCD, diffuse cortical dysplasia; NSWM, nonspecific foci of T2 prolongation in the white matter.
* Pathologic finding of the one lesion resected.

Fig 2. Small cortical lesion (patient 4).

A, Head-coil image. The small cortical lesion (*arrow*) is difficult to detect, even in retrospect, on the coronally acquired 3DFT gradient-echo image (36/12/0.75).

B, Surface-coil image. The improved signal-to-noise ratio and spatial resolution of the coronally acquired 3DFT gradient-echo image (33/7/0.75) acquired with surface coils resolve this small lesion (*arrow*).

C, Head-coil image. On this axial reformat of the coronally acquired 3DFT gradient-echo image (36/12/0.75), the small cortical lesion (*arrow*) is again difficult to detect.

D, Surface-coil image. On this axial reformat of the coronally acquired 3DFT gradient-echo image (33/7/0.75), this small lesion (*arrow*) is again resolved.
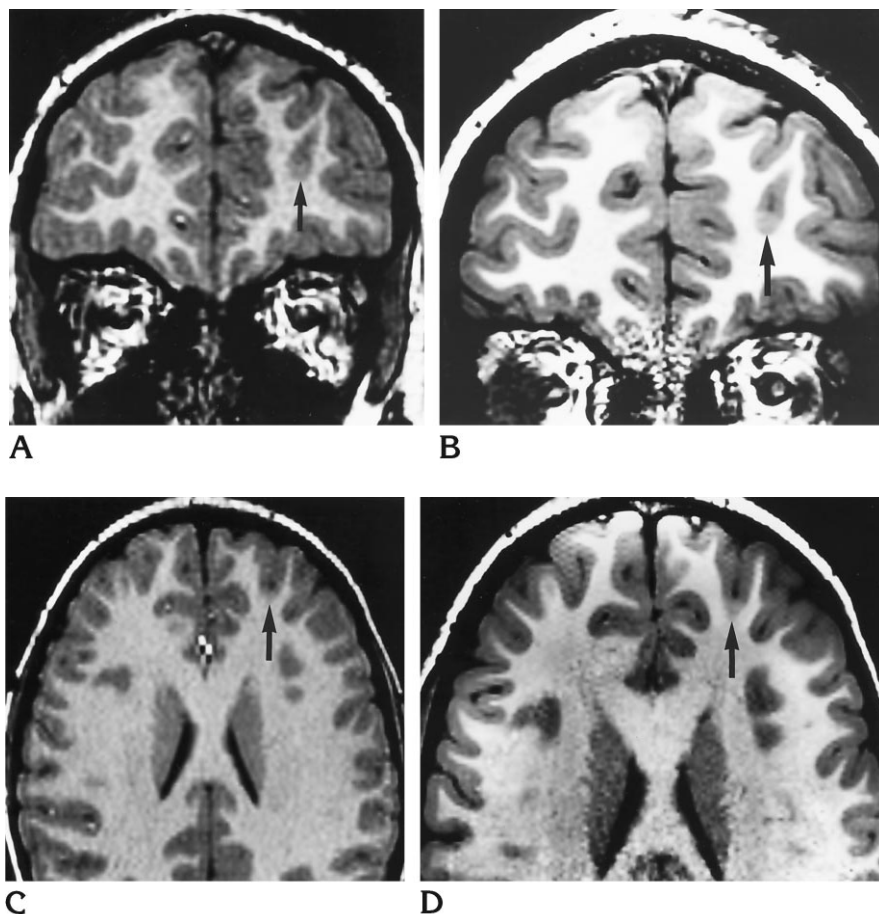
of mass effect, converting the diagnosis from an indeterminate finding to a focal cortical dysplasia (Fig 1). In three patients (patients 9, 14, and 15), regions of possible irregularities in the gray–white matter junction were seen better on surface-coil studies; the surface-coil study was interpreted as normal in patient 14 but abnormalities were confirmed on the studies in the remaining two patients. Interpretation of both the surface- and head-coil studies was indeterminate in patient 10, and the significance of the unusual gyrus remains unknown. Thus, surface-coil imaging caused six studies originally classified as indeterminate to be reclassified as abnormal and one study originally classified as indeterminate to be reclassified as normal.

Abnormal Head-Coil Study (10 patients).— All patients with an abnormal quadrature head-coil study had an abnormal surface-coil study. However, surface-coil studies resulted in a change in the most probable diagnosis in five patients. In three of these five patients (patients 16, 19 and 21), abnormalities thought to represent tumors on head-coil studies were shown to be more likely nonneoplastic because of lack of mass effect on surface-coil studies (Fig 3). In one of the five patients (patient 18), areas interpreted as heterotopias on the head-coil images were shown to have poorly defined margins on surface-coil images and were therefore more compatible with indeterminate foci of T2 prolongation. In the last of these five (patient 23), an area of presumed focal cortical dysplasia was more suggestive of a tumor on surface-coil images, owing to the detection of mild mass effect. Although not altering the diagnosis, surface-coil images increased the specificity of the diagnosis in patient 17 by illustrating the multiple, small, fused polymicrogyri that appeared as an indeterminate area of focal cortical dysplasia on head-coil studies (Fig 4). The surface-coil images also showed a second, much smaller focus of polymicrogyria in the contralateral hemisphere (Fig 4C). In the remaining four patients (patients 20, 22, 24, and 25), there was no change in interpretation with the addition of the surface-coil study.
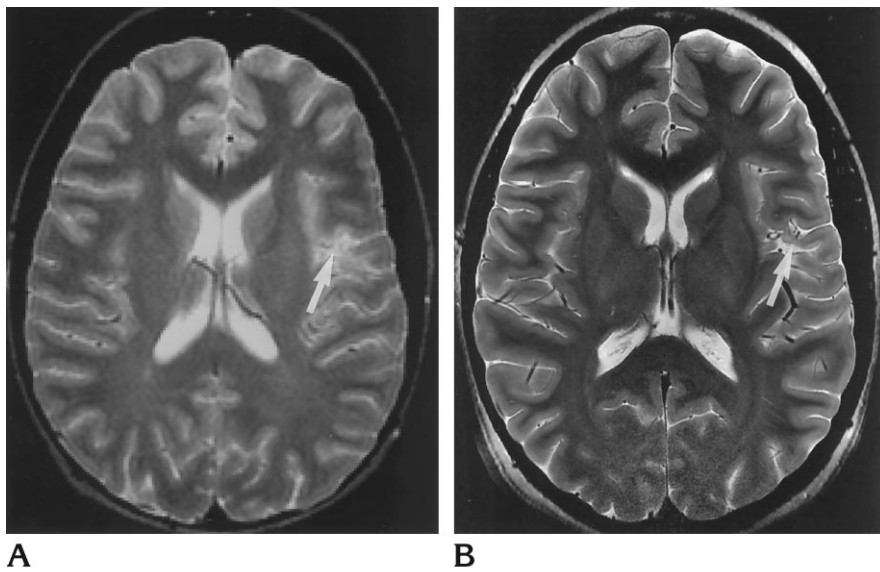
Fig 3. Focal encephalomalacia (patient 16).

A, Head-coil image. A neoplastic lesion cannot be ruled out on this axial T2-weighted image (2500/80/0.75) (*arrow*).

B, Surface-coil image. The axial surface-coil fast spin-echo image (400/112/4) shows focal volume loss with thinning of the cortex (*arrow*), consistent with encephalolmalacia.
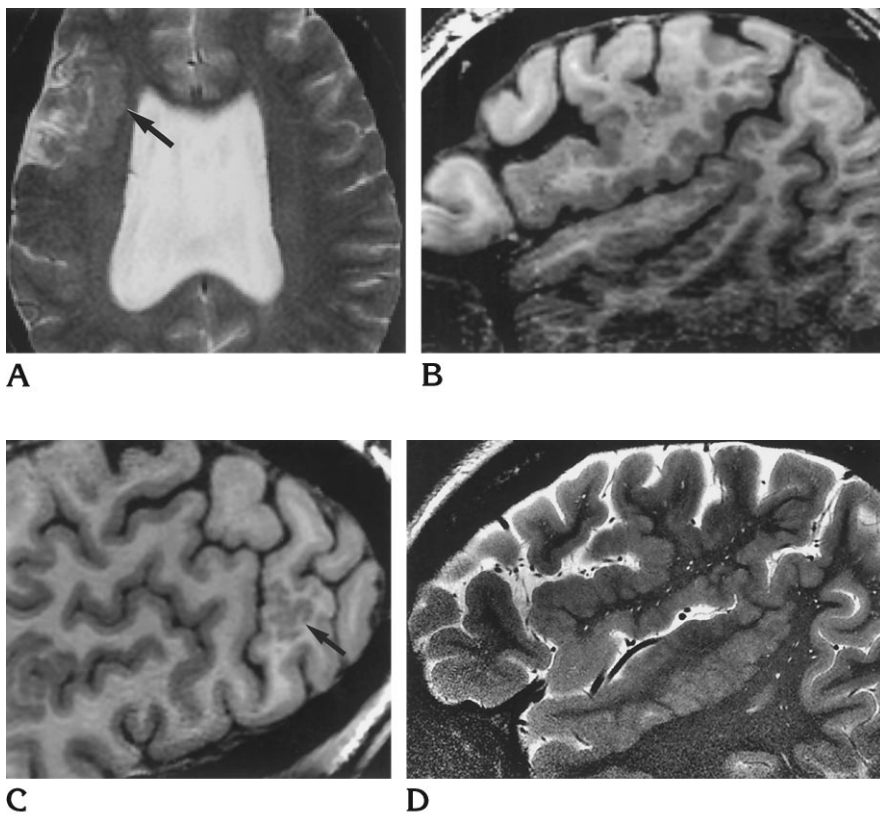

Fig 4. Multifocal polymicrogyria (patient 17).

A, Head-coil image. The multiple small gyri of polymicrogyria in the right hemisphere are difficult to resolve on the axial T2-weighted image (2500/80/0.75) (*arrow*).

B, Surface-coil image. The multiple small gyri in the right hemisphere are resolved on the sagittal reformats of the 3DFT gradient-echo image (33/7/0.75) acquired with surface coils.

C, Surface-coil image. The sagittal reformat of the coronally acquired 3DFT gradient-echo image (33/7/0.75) shows a second, previously undetected focus of polymicrogyria (*arrow*) in the contralateral (left) parietal lobe.

D, Surface-coil image. The sagittal fast spin-echo image (4000/112/4) also resolves the multiple small gyri of polymicrogyria in the right hemisphere.

Comparison of Surface-Coil Results and Interictal Electrophysiology/Seizure Semiology

The results are grouped according to the location of the seizure focus by interictal scalp EEG/seizure semiology. In each section the location as determined by interictal EEG/seizure semiology is compared with the location deter-mined by MR imaging. This emphasizes the impact that imaging studies have on determining the site of ictal onset. Results are summarized in the Table.

Good Localization with EEG/Seizure Semiology.—In 11 patients, the interictal EEG/seizure semiology localized the epileptogenic focus to

one lobe (patients 1, 2, 6, 7, 11, 14, 17, 19, 20, 23, and 25). Although the interictal EEG in patient 17 was not located, seizure semiology resulted in location of the epileptogenic focus in the right central region. Four of these patients had normal MR findings (patients 2, 6, 7, and 14). Five had focal cortical abnormalities on MR images limited to one lobe (patients 1, 11, 19, 20, and 23), with the same lobe involved as on EEG in three patients (patients 1, 19, and 23) and with the adjacent lobe in the same hemisphere involved in two patients. These focal cortical lesions are strong candidates for the true site of ictal onset. In the remaining two patients (patients 17 and 25) with good EEG/seizure semiology, MR showed unsuspected multifocal abnormalities involving more than one lobe. In patient 17, two regions of polymicrogyri were identified, with the larger polymicrogyric area corresponding to the lobe identified with EEG/seizure semiology. In patient 25, one heterotopia was identified in each occipital lobe and neither location corresponded to the lobe identified with EEG/seizure semiology. In these two cases, the imaging findings decrease the probability that the patients will be seizure free with resection of the electrophysiologic focus.

Poor Localization with EEG/Seizure Semiology.—In six patients (patients 4, 8, 9, 12, 21, and 22), the EEG/seizure semiology restricted the epileptogenic focus to two adjacent lobes. Five patients (patients 4, 8, 9, 12, and 21) had MR abnormalities localized to one lobe and in each case that lobe was one of the two identified with EEG/seizure semiology. In patient 22, the surface-coil study did not improve location, but multifocal heterotopias were identified as well as a region suggestive of focal cortical dysplasia in the right parietal lobe.

No Localization with EEG/Seizure Semiology.—In eight patients the EEG/seizure semiology was not restricted to two adjacent lobes (patients 3, 5, 10, 13, 15, 16, 18, and 24). Patient 5 had normal MR findings. Four patients (patients 3, 13, 15, and 16) had abnormalities limited to one lobe on MR images, three of which involved the cortex and were therefore strong candidates for the ictal focus. Patient 24 had diffuse polymicrogyria. Patient 10 had an unusual-appearing gyrus that was of uncertain clinical significance. In patient 18, bilateral frontal abnormal foci of T2 pro-

longation were identified by MR imaging, but EEG/seizure semiology suggested a focus in the right frontoparietal region. No cortical abnormalities or definite anatomic cause of the seizures were identified.

Comparison of Surface-Coil Results and Video/EEG Telemetry with and without Subdural Electrodes

Thirteen patients had video/EEG telemetry after interictal EEG, but video/EEG telemetry improved location of the suspected epileptogenic zone in only two (15%) of them (patients 11 and 25). In the same group of 13 patients, surface-coil imaging identified a focal cortical abnormality that was a strong candidate for the ictal focus in six (46%) (patients 4, 8, 11, 12, 13, and 20) and provided useful prognostic information in another five (38%) (patients 3, 17, 22, 24, and 25). Useful prognostic information included the identification of heterotopias or diffuse/multifocal cortical dysplasias. Note that even in the two patients in whom video/EEG telemetry provided better location of the ictal focus (patients 11 and 25), surface-coil imaging provided addition useful information by identifying a focal cortical lesion in patient 11 and by identifying the presence of multiple cortical abnormalities in patient 25. In only two patients did surface-coil imaging fail to provide additional useful information (patients 1 and 6). Therefore, surface-coil imaging provided clinically relevant information in 85% of patients, whereas video/EEG telemetry provided clinically relevant information in only 15% (see Table).

Comparison of Surface-Coil Results, Pathologic Findings, and Surgical Outcome

Six patients (patients 1, 2, 11, 17, 20, and 25) had surgical resection and one patient (patient 7) had a cortical biopsy. Patient 1 had a right temporal focal topectomy with histologic sections revealing a hamartomatous lesion with no clear posterior margin. Although no abnormalities were identified in the anterior temporal lobe with surface-coil imaging, a small cyst was identified in the cortex of the right superior temporal gyrus. This cyst was approximately 1 cm posterior to the posterior resection margin and was within remaining, possibly abnormal, cortex. Patient 2 had a right temporal focal topectomy with normal pathologic findings consistent

with the normal surface-coil study. A left temporal focal topectomy was performed on patient 25, in whom pathologic specimens showed only mild gliosis in the left hippocampus. The MR examination in this patient showed one heterotopia in each occipital lobe but notemporal abnormalities. Patient 11 underwent a left frontal topectomy, patient 17 a right posterior frontal lobectomy, and patient 20 a partial right frontal lobectomy. In all three cases the pathologic diagnosis of cortical dysplasia was consistent with the MR interpretation. Because of rapid progression of symptoms in patient 7, a cortical biopsy was performed 2 months after a normal MR examination. The biopsy results were suggestive of Rasmussen encephalitis.

Of the two patients in whom no cortical lesion was detected (patients 2 and 25), one had no change in seizure frequency (patient 2) and one remains seizure free (patient 25). Patient 1, with an indeterminate imaging finding, remains seizure free. The two patients who underwent complete surgical resection of the cortical abnormality identified on MR images (patients 11 and 20) remain seizure free. In patient 17, in whom two cortical lesions were identified, seizures were reduced more than 50% after nearly complete resection of the largest region.

Discussion

The standard method used by epileptologists to locate epileptogenic foci is electrophysiological monitoring (12). The outcome for surgical excision of the presumed epileptogenic focus is improved if an anatomic abnormality matching the suspected area of abnormal electrical activity can be identified on imaging studies (8). In Taylor and Falconer's well-known study of the control of epilepsy by surgical resection of dysplastic cortex, six of 10 patients became seizure free and 20% had marked reduction in seizure frequency after surgical resection of the major portion of the associated lobe (13). Palmini et al (7) showed that 75% of patients with extratemporal cortical dysplasia had at least a 50% reduction in seizure activity; in 56% of the patients, major seizure activity was reduced by at least 90%. Outcome was most strongly correlated with extent of lesion removal; thus, it appears that patients with a focal cortical lesion corresponding to the area of abnormal electrophysiology have an improved prognosis after surgical resection compared with those in

whom a lesion cannot be located. Because locating the lesion is of great consequence, identification and characterization of small lesions by neuroimaging can be of crucial importance. This observation emphasizes the emerging role of imaging both in detecting and portraying the extent of cortical lesions.

Causes of intractable partial neocortical epilepsy include cortical dysplasias—such as layered and unlayered polymicrogyria (14), microdysplasia (15), the Taylor types of focal cortical dysplasia (16), and localized pachygyria—Rasmussen encephalitis, encephalomalacia, vascular malformations, and cortical neoplasms. When small, these lesions are difficult to detect and to differentiate. For example, the subtle mass effect of small neoplastic lesions and the subtle blurring of the gray–white matter junction in small focal dysplasias may be impossible to detect with routine head-coil images (8, 16) (Fig 1A). Cortical dysplasias 1.5 cm in size have gone undetected on routine head-coil MR images, even in retrospect (17), probably because the detection rate on these images is related to the degree of histologic change and, therefore, to the image resolution (18, 19). Moreover, it is sometimes difficult to differentiate polymicrogyria from pachygyria on head-coil studies, since areas of focal polymicrogyria can appear as thickened pachygyric regions owing to an inability to resolve multiple, small, fused gyri (20–22) (Fig 4).

To improve the ability to detect and discriminate among focal lesions causing partial epilepsy, we developed dedicated phased-array surface coils for imaging the brain (11). As described elsewhere (11), the surface coils are placed immediately adjacent to the skin, resulting in an increased signal-to-noise ratio in the underlying brain parenchyma relative to that obtained with a standard head coil. This increased signal-to-noise ratio can be used to improve both spatial and contrast resolution. The major limitation of surface coils, the small spatial coverage, is minimized by using a phased-array construction, which significantly increases the volume imaged (11). Placement of the surface coils was guided by the known seizure semiology, electrophysiological data, and head-coil imaging results.

Surface-coil imaging altered the immediate clinical management of five patients. In patients 16, 19, and 21, the increased spatial resolution allowed us to be confident that no mass effect

was present and therefore to conclude that the lesion was nonneoplastic (Fig 3). The added finding of a thinned cortex in patient 19 was suggestive of encephalomalacia. Excluding tumor from the differential diagnosis altered the clinical management in these patients, as surgical excision was no longer immediately indicated. In patient 18, small foci of abnormal T1 and T2 prolongation in the deep white matter, initially interpreted as heterotopias, were shown to be poorly defined and to have signal intensities different from gray matter. The change in diagnosis to "nonspecific white matter changes" altered the counseling of this patient, who was concerned about the possible hereditary nature of heterotopic gray matter.

Surface-coil imaging also allowed us to identify polymicrogyria more easily (Fig 4) and potentially to differentiate it from pachygyria. The ability of surface-coil imaging to differentiate among various types of cortical dysplasia may allow prospective categorization of patients by specific clinicomorphologic syndrome, which may, in turn, help better determine prognosis. We speculate that a useful classification system for cortical dysplasias may be derived from a combination of gross anatomic data obtained by surface-coil MR imaging and histopathologic examination of the resected tissue. Eventually, it may be possible to determine the type and extent of many cortical dysplasias with imaging alone.

It is of note that we observed three distinct appearances of dysplastic cortex on surface-coil images: 1) irregularity of the gray–white matter junction with associated T2 prolongation in the underlying white matter (patient 21); 2) irregularity of the gray–white matter junction without an associated T2 abnormality in the underlying white matter (patients 9, 15, 17, 22, and 24) (frank polymicrogyria could be reasonably well resolved in patient 17, Fig 4); and 3) focal areas of gray–white matter junction blurring and triangular T2 prolongation in the subjacent white matter with the apex extending toward the ventricle (Fig 1) (patients 8, 11, 12, and 13).

These imaging findings were correlated with pathologic findings in patients 17 and 11, representing the second and third type of dysplasia, respectively. In the third type of focal cortical dysplasia, it might be suggested that the associated T2 prolongation represents wallerian degeneration, but the fact that the tract of T2 prolongation extends to the ventricular surface and not into the internal capsule discounts that possibility. In fact, the tract extends along the course followed by migrating neurons during cortical development, extending radially from the ventricle along the radial glial fibers with horizontal excursion in the subcortical region (23). This appearance, which has also been described in patients with tuberous sclerosis (24, 25), has been postulated to represent an abnormality along the radial glial-neuronal unit resulting from abnormal differentiation in the stem cells within the germinal zone (26). These imaging findings may well give clues to the underlying pathophysiology of these malformations.

Although focal cortical lesions that were strong candidates for the epileptogenic focus were identified in 52% of our patients, only a small fraction have been treated surgically. This is either because the patient is a poor surgical candidate or because the referring clinician has preferred to attempt seizure control by means of new types or combinations of medical therapy. Among the three patients with focal cortical abnormalities on MR images who did have surgery, the two whose lesions were completely resected remain seizure free and the one in whom the larger of two focal abnormalities was resected has had a significant reduction in seizure frequency.

We have shown that the use of surface-coil imaging in this study improved our ability to detect and differentiate cortical lesions even if the resulting signal intensity change or irregularity of the gray–white matter junction was confined to a region less than 1 cm in length (Figs 1 and 2). However, visual inspection of both head- and surface-coil images is time-consuming and requires significant experience; therefore, automated mathematical methods are being developed that will be able to determine the thickness and signal intensity of the cortex as well as quantify the degree of folding in the gray–white matter junction on 3DFT gradient-echo images.

In summary, surface-coil images were better than head-coil images either in detecting more lesions or in better defining lesions in 64% of the 25 patients in this study. When combined with electrophysiological data, surface-coil images identified a focal cortical lesion that was a strong candidate for the epileptogenic focus in 52% of patients. Of the 13 patients undergoing

video/EEG telemetry, video/EEG telemetry alone improved localization in only 15%, whereas surface-coil imaging showed a strong candidate for the epileptogenic focus in 46% and provided relevant prognostic information in a further 38%. We conclude that surface-coil imaging, guided by results of head-coil and surface EEG studies, has great potential for use in the evaluation of neocortical partial epilepsy.

Acknowledgments

References

1. Hauser WA, Hesdorffer DC. *Epilepsy: Frequency, Causes and Consequences.* New York, NY: Demos Press; 1990
2. Farwell JR, Dodrill CB, Batzel LW. Neuropsychological abilities of children with epilepsy. *Epilepsia* 1985;26:395–400
3. Bradley C. Behavior disturbances in epileptic children. *JAMA* 1951;146:436–441
4. Taylor DC. Epilepsy as a chronic sickness: remediating its impact. In: Engel J Jr, ed. *Surgical Treatment of the Epilepsies.* New York, NY: Raven Press; 1993:11–22
5. Moshe SL, Shinnar S. Early intervention. In: Engel J Jr, ed. *Surgical Treatment of the Epilepsies.* New York, NY: Raven Press; 1993:123–132
6. Kuzniecky RI, Cascino GD, Palmini A, et al. Structural neuroimaging. In: Engel J Jr, ed. *Surgical Treatment of the Epilepsies.* New York, NY: Raven Press; 1993:197–209
7. Palmini A, Andermann F, Olivier A, Tampieri D, Robitaille Y, et al. Focal neuronal migration disorders and intractable partial epilepsy: results of surgical treatment. *Ann Neurol* 1991;30:750–757
8. Palmini A, Gambardella A, Andermann F, et al. Operative strategies for patients with cortical dysplastic lesions and intractable epilepsy. *Epilepsia* 1994;35(Suppl 6):S57–S71
9. Casino G, Boon PAJ, Fish DR. Surgically remediable lesional syndromes. In: Engel J Jr, ed. *Surgical Treatment of the Epilepsies.* New York, NY: Raven Press; 1993:77–86
10. Barkovich AJ, Rowley HA, Andermann F. MR imaging in partial epilepsies: value of high resolution volumetric techniques. *AJNR Am J Neuroradiol* 1995;16:339–343
11. Wald LL, Carvajal L, Moyer SE, et al. Phased array detectors and an automated intensity correction algorithm for high resolution MR imaging of the human brain. *Magn Reson Med* 1995;34:433–439
12. Sharbrough FW, Gotman J. Overview of new diagnostic techniques. In: Engel J Jr, ed. *Surgical Treatment of the Epilepsies.* New York, NY: Raven Press; 1993:155–159
13. Taylor DC, Falconer MA. Focal dysplasia of the cerebral cortex in epilepsy. *J Neurol Neurosurg Psychiatry* 1971;34:369–387
14. Ferrer I. A golgi analysis of unlayered polymicrogyria. *Acta Neuropathol* 1984;65:69–76
15. Meenke HJ. Minimal developmental disturbances in epilepsy and MRI. In: Shorvon SD, Fish DR, Andermann F, Bydder GM, Stefan H, eds. *Magnetic Resonance Scanning and Epilepsy.* New York, NY: Plenum Press; 1994:127–136
16. Barth PG. Disorders of neuronal migration. *Can J Neurol Sci* 1987;14:1–16
17. Desbiens R, Berkovic SF, Dubeau F, et al. Life-threatening focal status epilepticus due to occult cortical dysplasia. *Arch Neurol* 1993;50:695–700
18. Kuzniecky RI. Magnetic resonance imaging in developmental disorders of the cerebral cortex. *Epilepsia* 1994;35(Suppl 6):S44–S56
19. Kuzniecky R, Murro A, King D, et al. Magnetic resonance imaging in childhood intractable partial epilepsies: pathological correlations. *Neurology* 1993;43:681–687
20. Guerrini R, Battaglia A, et al. Epilepsy and focal gyral anomalies detected by MRI: electroclinico-morphological correlations and follow-up. *Dev Med Child Neurol* 1992;34:706–718
21. Otsubo H, Hwang PA, Jay V, et al. Focal cortical dysplasia in children with localization-related epilepsy: EEG, MRI, and SPECT findings. *Pediatr Neurol* 1993;9:101–107
22. Palmini A, Andermann F, Olivier A, et al. Focal neuronal migration disorders and intractable partial epilepsy: a study of 30 patients. *Ann Neurol* 1991;30:741–749
23. Gressens P, Evrard P. The glial fascicle: an ontogenic and phylogenic unit guiding, supplying and distributing mammalian cortical neurons. *Brain Res Dev Brain Res* 1993;76:272–277
24. Iwasaki S, Nakagawa H, Kichikawa K, et al. MR and CT of tuberous sclerosis: linear abnormalities of the cerebral white matter. *AJNR Am J Neuroradiol* 1990;11:1029–1034
25. Braffman BH, Bilaniuk LT, Naidich TP, et al. MR imaging of tuberous sclerosis: pathogenesis of this phakomatosis, use of gadopentetate dimeglumine, and literature review. *Radiology* 1992;183:227–238
26. Seidenwurm D, Barkovich AJ. Understanding tuberous sclerosis. *Radiology* 1992;183:23–24