Original article

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Pre-surgical epilepsy evaluation using 3T MRI. Do surface coils provide additional information?

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ABSTRACT – *Purpose.* To assess if 3T MRI can be further improved by adding surface coil imaging, in the context of detection and characterization of cerebral lesions in patients with drug-resistant epilepsy. *Methods.* Twenty five patients with drug-resistant epilepsy undergoing evaluation for epilepsy surgery were examined with high resolution 3T MRI. The patients were MRI-negative (n = 15), or had unclear findings (n = 10), on previous MRI at 1.0-1.5T. Surface coils were applied over the suspected epileptogenic zone after imaging in the head coil. In MRI-negative patients, placement of the coils was defined by semiological analysis, extracranial video-EEG, and, in selected cases, subtraction ictal SPECT co-registered with MRI and PET. Coil placement was re-analyzed and graded, based on the degree of convergence between different investigational modalities. *Results.* Surface coil MRI allowed visualization of the cortical lesions with somewhat better demarcation and detail, but did not contribute to detection of previously undiagnosed lesions and did not provide additional information regarding type of lesion. Possible epileptogenic lesions were detected on 3T MRI in 12 patients. No abnormalities were found in the remaining 13 patients. 3T MRI provided new or additional information about the cortex, compared with reports from previous 1.0-1.5T MRI in 5 patients (20%). *Conclusion.* 3T MRI with high resolution is valuable for lesion detection, especially MCD, in patients with drug-resistant epilepsy. We question the additional contribution from supplementary surface coil imaging at 3T MRI.

Key words: 3T MRI, surface coils, epilepsy surgery, malformation of cortical developement

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Epilepsy surgery renders approximately 50-60% of patients seizure-free. High quality neuroradiology contributes both to etiological diagnosis and a favourable prognosis following surgery. The likelihood of seizure freedom increases if an epileptogenic lesion has been identified on magnetic resonance imaging (MRI) during the pre-surgical work-up (Kuzniecky 1996, Ruggeri 2000, Kuzniecky and Knowlton 2002, Kral *et al.* 2003). Malformation of cortical development (MCD) is a common finding in surgical series (Li *et al.* 1995, Wieshmann 2003, Lüders and Schuele 2006). The

detection and delineation of MCD in brain imaging is crucial for complete resection and is a significant factor for favourable, long term outcome (Hamiwka *et al.* 2005).
Despite advances in imaging technology, previous studies have shown that not all lesions can be visualized with MRI as histopathological examination has revealed a higher proportion of epileptogenic lesions than was detected by pre-surgical radiology (Raymond *et al.* 1995, Von Oertzen *et al.* 2002). Furthermore, MRI often showed only part of the lesion in patients with MCD.
Improved image quality in selected regions of the cerebral cortex can potentially be achieved by using surface coils provided by the MRI scanner manufacturers. There are two previously published studies examining an epilepsy population with 1.5T MRI and surface coils (Gomez-Hassan *et al.* 2004, Grant *et al.* 1997). Gomez-Hassan *et al.* advocated the use of surface coils, and Grant *et al.* found the method to have great potential for the evaluation of patients with neocortical, partial epilepsy.
Knake *et al.* (2005) examined patients with focal epilepsies with 3T MRI, and added an eight-channel, phased array coil which was specifically built, and not a readily available surface coil provided by the manufacturer. The study showed improved lesion detection rate compared with 1.5T MRI. To our knowledge, there is no systematic, clinical observational study comparing 3T MRI with and without added surface coil examination.
Potential problems with surface-coil imaging include limited brain coverage and an overall increase in scan time (Moore *et al.* 2002, Bronen *et al.* 2002). It is therefore essential to establish if the use of surface coils improves the detection of epileptogenic lesions compared to head coil 3T MRI without additional surface coils.
In this study, we evaluate whether supplementary imaging using surface coils at 3T can improve the detection and delineation of epileptogenic lesions in a selected group of patients with pharmacoresistant epilepsy.

Methods and materials

Patient selection

Patients were recruited from the epilepsy surgery program at Lund University Hospital, Sweden. From our database, we retrospectively identified and included 25 patients (median age 15 years, range 4-51 years, 14 females and 11 males), who had been examined with 3T MRI, with and without adjunctive surface coils, during the clinical evaluation for epilepsy surgery. Disease duration ranged from 2-38 years (median 10 years) and both adults and children were included. Twenty-three patients had focal epilepsy and two were unclassified at referral to the University Hospital.

MRI protocol and evaluation

The patients had all been examined according to the same protocol, from November 2004 to June 2005, in the department of radiology at our hospital. All patients had previously undergone MRI at 1.0-1.5T, either at a University hospital or other referring centres in Sweden.

Head coil acquisition

MRI was performed with a 3T scanner (Philips Intera) using a 4-channel head coil and parallel imaging. The protocol for presurgical evaluation included a coronal, T2-weighted, fluid attenuated, inversion recovery (FLAIR) sequence (31 slices, thickness 5 mm, FOV 230 mm, matrix 288x384, TR 11000 ms, TE120 ms, TI 2800 ms, acceleration factor 2), a coronal, T1-weighted, 3D gradient, echo sequences covering the entire brain (200 partitions, thickness 1 mm, FOV 256 mm, matrix 256x256, TR 8 ms, TE 3-4 ms, TI 2800 ms, acceleration factor 2.5) and a T1-weighted inversion recovery (IR) sequences in 2 planes (slices 33, thickness 2 mm, FOV 220 mm, matrix 268x336, TR shortest, TE15 ms, TI 400 ms, acceleration factor 1) covering the region where the suspected epileptogenic zone was thought to be located. The scan time for the IR sequence was nine minutes for each plane.

Surface coil acquisition

The head coil was exchanged for two flexible surface coils (SENSE-flex-M, Philips), applied bilaterally on the head of the patient and centred according to the description below. The surface coil scans comprised a T1-weighted inversion recovery (IR) in two planes, without parallel imaging (33 slices, thickness 1.2 mm, FOV 220 mm, matrix 268x336, TR shortest, TE 15 ms, TI 400 ms, acceleration factor 1.2). The scan time was six minutes for each plane.
Real, as well as modulus images were reconstructed from the head coil IR sequence, whereas only modulus images could be reconstructed from the surface coil images with the software version available. On the real images, white matter has high signal intensity (white in the images), and grey matter has a lower signal intensity (grey), and the image background is grey. On modulus images, the white matter has low signal (black) and the grey matter has higher signal intensity (grey); the image background is black.
In the clinical, presurgical, MRI protocol used for the patients in this study, the T2-weighted FLAIR sequence and the T1-weighted, 3D, gradient echo (T1 3D GRE) sequence, both covering the whole brain, were used to detect signal changes and morphological abnormalities, respectively. The T1-weighted, inversion recovery (T1-IR) sequence with only subtotal brain coverage (for scan time reasons) had higher resolution and better grey/white matter differentiation. An inversion time of 400 ms was used to optimize grey-/white matter contrast on the T1-IR images. The in-plane spatial resolution was identical for head

and surface coil T1-IR images, 0.7 mm x 0.8 mm reconstructed to 0.4 mm x 0.4 mm, which is superior to the T1-weighted, 3D, gradient echo (T1 3D GRE) sequence, where it was 1 mm x 1 mm. The latter sequence had a slice (partition) thickness of 1 mm, whereas the T1-IR images obtained with the head coil had a slice thickness of 2 mm and the TI-IR- surface coil images had a thickness of 1.2 mm. Because of the thicker slices, the T1-IR sequences were acquired in two perpendicular planes, to optimize the spatial resolution in all planes. In order to maximize the SNR, no acceleration factor was used for the head coil T1-IR imaging. A low acceleration factor (1.2) was used for the corresponding surface coil imaging in order to somewhat reduce scan time without reducing SNR too much.

3T images were re-evaluated by an experienced neuroradiologist with knowledge of only the lateralization of the suspected epileptogenic zone. Pathological findings were noted with regard to type and localization. They were divided into malformations of cortical development, abnormalities of the hippocampus, white matter signal abnormalities and parenchymal loss. The malformations of cortical development were classified according to the well established Barkovich classification system (2001, 2005, 2004). Additional information from the surface coil images was described.

Surface coil placement

Surface coils were applied over the suspected epileptogenic brain region to further improve the image quality in selected regions of the cerebral cortex. In patients referred from other centers, the clinical data provided were used to guide coil placement. Discussions during the epilepsy surgery management rounds with the multidisciplinary epilepsy surgery team provided localizing information for surface coil placement for patients referred from the surgical team in Lund. In MRI-negative patients, placement of the coils was defined by localizing data from semiological analysis, extracranial video-EEG, ictal SPECT, SISCOM (subtraction ictal SPECT co-registered on MRI) and PET-findings. In MRI-positive patients, and patients with diffuse or non-specific lesions, placement of the coils was guided by previous MRI findings and additional localizing information.

We retrospectively re-analyzed the basis for surface coil localization to assess the accuracy of placement. The zones providing independent localizing information were defined: 1) the symptomatogenic zone, 2) the ictal onset zone by extracranial video-EEG and/or ictal SPECT or SISCOM; 3) the irritative zone by interictal EEG; and 4) the functional deficit zone by interictal SPECT and PET (Lüders and Carreño 2001). Authors were blinded to other investigational results during the re-evaluation of each separate modality, but as both epileptologists are involved in the surgical program they may have recognized some patients.

Semiological analyses

Semiological seizure classification was performed by two epileptologists. Information from video-EEG recorded seizures was available for 18 patients. For seven patients with normal 1.0-1.5T MRI findings, we had no access to video-EEG as they were referred to our institution primarily for high field strength imaging, and for these patients, seizure classification was done by review of medical records (*table 1*; patients # 12, 13, 15, 21-23 and 25). We used the semiological seizure classification proposed by Lüders *et al.* (1998). Anatomical correlates to the symptomatogenic zones were categorized by lobe when localizing seizure semiology was found; in patients with focal motor seizures, the symptomatogenic zone was localized to the fronto-central region, visual auras to the occipital lobe, sensory auras to the parietal lobe, automotor seizures to the temporal lobe, and hypermotor seizures to the frontal lobe.

EEG analyses from video monitoring

Each patient's ictal EEG was recorded during the video-EEG monitoring and was reviewed and classified for clinical purposes. For study purposes, the VEEG performed locally was re-evaluated by a neurophysiologist. EEG onset patterns were classified as regional by lobe or brain region, lateralized by hemisphere, or non-localizing/generalized. Interictal EEG was classified in the same manner.

Ictal SPECT, SISCOM and PET analyses

In six cases, complementary localizing data were obtained by subtraction ictal SPECT co-registered with MRI (SISCOM). Functional imaging was reviewed by a neurophysiologist, and localization of significant hotspots and significant hyperperfusions were defined by lobe or brain region. Five patients had undergone ictal SPECT examination at other University hospitals (*table 1*; patients # 12, 21-23 and 25). SISCOM was not performed at the external institutions. In four patients, interictal positron emission tomography (PET) investigations were available. All PET scans were carried out at Uppsala University Hospital, Sweden. Externally performed functional imaging results were taken from patient records.

We graded the level of evidence for coil positions into three categories depending on the degree of convergence between different investigational modalities and coil placement:

I. good support for coil placement. Convergence between coil position and localizing information from investigational modalities existed, indicating a certain region where no previous pathology had been found on 1.0T-1.5T MRI;

II. partial support for coil placement. When surface coils were positioned over one or multiple lobes or regions, in convergence with other localizing findings, even though

Table 1. Patient demographics - results from pre-surgical evaluation (video-EEG, functional and anatomical imaging) and histopathology.

Pat #	Age (y/sex)	Disease duration (y)	Seizure semiology	Video-EEG sz onset	Ictal SPECT/SISCOM/PET	Type of lesion on 1.0-1.5 T MRI*	Type of lesion on 3T MRI	Location of lesion	Histo-pathology
1	51/F	29	Automotor sz	R temporal	-	MCD*	MCD: polymicrogyria	R fronto-temporal + insula	-
2	49/M	38	Bilat tonic sz	R fronto-central	SISCOM: L parietal, bilat medial fronto-parietal	Unspecific*	MCD: focal transmantle cortical dysplasia	L fronto- parietal	MCD
3	30/F	12	Visual aura → automotor sz, + R hand sensory aura	R + L temporal	-	Normal	Normal	-	-
4	27/F	16	Automotor sz	Lateralized L	SISCOM: L + R temporal, L front temporal	MCD*	MCD: subcortical (transmantle) heterotopia + cortical thickening	R parietal	-
5	27/F	13	GTC sz	L temporal + R occipito-temporal	-	Normal	Normal	-	Unspecific
6	24/F	21	Automotor sz	L occipito-temporal	SISCOM: L temporal + L occipital	HS	HS	L medial temporal	-
7	32/F	18	Aura	L temporal	-	Normal	Normal	-	-
8	37/M	27	Hypermotor	Non-localized	SISCOM: L temporal	Heterotopic tissue	MCD: Subcortical heterotopia	Bilat temporal	HS + MCD (micro-dysgenesis)
9	17/M	17	Automotor	R fronto-central	-	Cortical atrophy*	MCD: Focal cortical dysplasia	R parietal	-
10	17/F	14	Psychic aura	L temporal	-	L HS + increased signal	L HS + increased signal	L medial temporal + L occipital	-
11	14/M	9	Aura	No sz during monitoring	SISCOM: L temporal	Normal*	Normal	-	-
12	14/M	7	Atonic szs, aphasic szs	R + L frontal	SPECT: L frontal	Normal	Parenchymal loss, possibly post-traumatic	R frontal	-
13	13/F	10	L motor sz	-	-	Normal	MCD: focal transmantle CD	R frontal (precentral gyrus)	-
14	13/F	3	Abdominal/olfactory + visual aura	L temporal	-	Normal	Normal	-	-

Table 1. suite.

Pat #	Age (y/sex)	Disease duration (y)	Seizure semiology	Video-EEG sz onset	Ictal SPECT/SISCOM/PET	Type of lesion on 1.0-1.5 T MRI*	Type of lesion on 3T MRI	Location of lesion	Histo-pathology
15	10/M	5	Myoclonic sz + absences	-	PET: R temp-occipital + L temporal	Normal	Normal	-	-
16	9/F	9	Bilat tonic sz	R TPO-junction	PET: R parietal	Normal	Normal	-	-
17	8/M	7	Hypomotor sz	R occipital	-	Increased signal on FLAIR sequences	Increased signal on FLAIR sequences	Bilat subcortical occipital	-
18	6/F	4	Automotor sz	R occipital	-	R HS + parenchymal loss	R HS + parenchymal loss	R medial temporal + R temporo-occipital	-
19	20/M	14	L arm sensory aura	Non-localizing	-	Normal*	Normal	-	-
20	19/M	15	Bilat tonic sz	R fronto-central	SISCOM: R central	Increased signal (changes after previous subpial transection)	MCD: Focal cortical dysplsia, + postop subpial transection	R frontal	-
21	11/M	6	R motor sz	-	SPECT: L frontal PET: L frontal	Normal	Normal	-	MCD (biopsy only)
22	15/F	9	R arm motor sz → mouth sensory-motor sz	-	SPECT: L frontal	Normal	Normal	-	-
23	11/F	6	L leg motor sz	-	SPECT: R parietal	Normal*	Normal	-	MCD (micro-dysgenesis)
24	8/F	3	Myoclonic sz	Generalized	-	Normal	Normal	-	-
25	4/M	2	Bilat tonic sz	-	SPECT: L frontal PET: L frontal	Normal	Normal	-	-

Pat: patients; #: number; y: years; sz: seizure, R: right; L: left; M: male; F: female; GTC: generalized tonic clonic; sz: seizure; TPO: temporo-parieto-occipital; MCD: malformation of cortical development; CD: cortical dysplasia; HS: hippocampal sclerosis; SPECT: single photon emission computed tomography; SISCOM: subtraction ictal SPECT co-registrated on MRI; PET: postitron emission tomography;* examined with 1.0T.

there was the indication of other possible epileptogenic regions, *i.e.* uncovered separate regions of interest;
III. no support for epileptogenicity in the underlying region where coils were applied.

Ethics committee approval and funding

This study was performed retrospectively and the study subjects were not submitted to any additional investigation other than that which the referring physician had originally requested. The study was performed in accordance with the regulations of the local ethics committee. This material has not been previously published. No potential for commercial bias was declared by the authors.

Results

New lesions identified on 3T MRI

3T MRI provided new or additional information about structural grey matter abnormalities, compared to reports from 1.0-1.5T MRI, in five patients (20%). In one 1.5T MRI-negative patient, an MCD was identified (*table 1*; patient # 13), and in four patients with unspecific findings on 1.0T or 1.5T MRI, MCDs were identified (*table 1*; patients # 2, 8, 9 and 20) on 3T MRI.
Table 1 provides demographics for the entire study group, results available from presurgical evaluation and histopathology from patients who underwent surgery. *Table 1* also shows the type and the location of findings on 3T MRI and 1.0-1.5T. The lesions identified on 3T MRI were malformations of cortical development in seven, hippocampal sclerosis in three, white matter lesions in one, and regional atrophy in one patient.

Surface coil examination

Surface coil 3T MRI visualized the cortical lesions with somewhat better demarcation and detail *(figures 1, 2, 3)*, but did neither contribute to detection of previously undiagnosed lesions nor add information regarding type of lesion in any of the cases reviewed in this study. The improved details using surface coils only pertained to cortical structures situated close to the coils. Better demarcation of cortical structures was a general finding wherever pathology was delineated. This was particularly noted in four patients with MCDs (*table 1*; patients # 1, 4, 9 and 20).
Table 2 shows the level of evidence for surface coil placement and type of investigational modalities providing localizing information for each patient with normal 1.0-1.5T MRI.

Discussion

In our study, surface coils did not provide additional information over and above that provided by head coil 3T MRI for the detection and characterization of MCDs in patients with drug-resistant epilepsy. Surface coil MRI visualized the cortical lesions with somewhat better demarcation and detail *(figures 1, 2, 3)*, but did not improve lesion detection or provide information regarding type of lesion in any of the cases reviewed.
High resolution MRI is able to detect MCD in an increasing number of patients, as previously described by several authors (Barkovich and Kuzniecky 1996, Grant *et al.* 1998, Colliot *et al.* 2006, Knake *et al.* 2005). In our study population, 3T MRI revealed MCDs that had not been detected on clinical 1.0-1.5T MRI in five out of the 25 patients. MCDs are subtle abnormalities that can easily be missed without the use of high resolution imaging and sequences with good grey/white matter differentiation (Vattipally and Bronen 2004). We consider this finding clinically important, although it was not the primary topic of our study.
Grant *et al.* (1997) found improved detection and differentiation of focal cortical lesions in 64% of patients (16/25). In that study, two acquisitions with different coil positioning were occasionally used to increase coverage. Knake *et al.* (2005) demonstrated an increased number of MRI findings in focal epilepsy using 3T, phased-array MRI in previously 1.5T MRI-negative patients, with a 37.5% (15/40) new lesion detection rate. One limitation of that study was, as the authors pointed out themselves, the inability to separate the effects of 3T imaging and phased array coil imaging.
In our patients, there was a 20% (5/25) detection rate of new lesions, but our patients were retrospectively selected from our pre-surgical MRI database and only patients with results from 3T MRI, with and without adjunctive surface coil investigation, were included. Therefore, MRI-positive patients with new and clear-cut epileptogenic lesions on 3T without surface coils compared to lower field strength MRI were not included in our study. Thus, the difference in detection rate may be explained by the disparity in study design and selection bias. Furthermore, two patients (*table 1*, patients # 15 and # 24) had an unspecified epilepsy syndrome diagnosis at referral, but were later determined to have primary generalized epilepsy, and in an unlikely category for cortical pathology.
A crucial question is the placement of the surface coils, as it is only possible to cover a limited volume of the cerebral cortex. A clear hypothesis must be provided to guide coil placement, taking into account all the localizing data for zones of interest pointing to the epileptogenic focus. From our re-evaluation, we concluded that localizing information was not taken into full account in several patients (*table 2*; level of evidence II), *i.e.* when surface coils were positioned over one or multiple lobes or regions in convergence with localizing findings, even though there was indication for non-covered separate zones of interest. It is a weakness in our study that all possible epileptogenic regions were not adequately covered by surface coils.

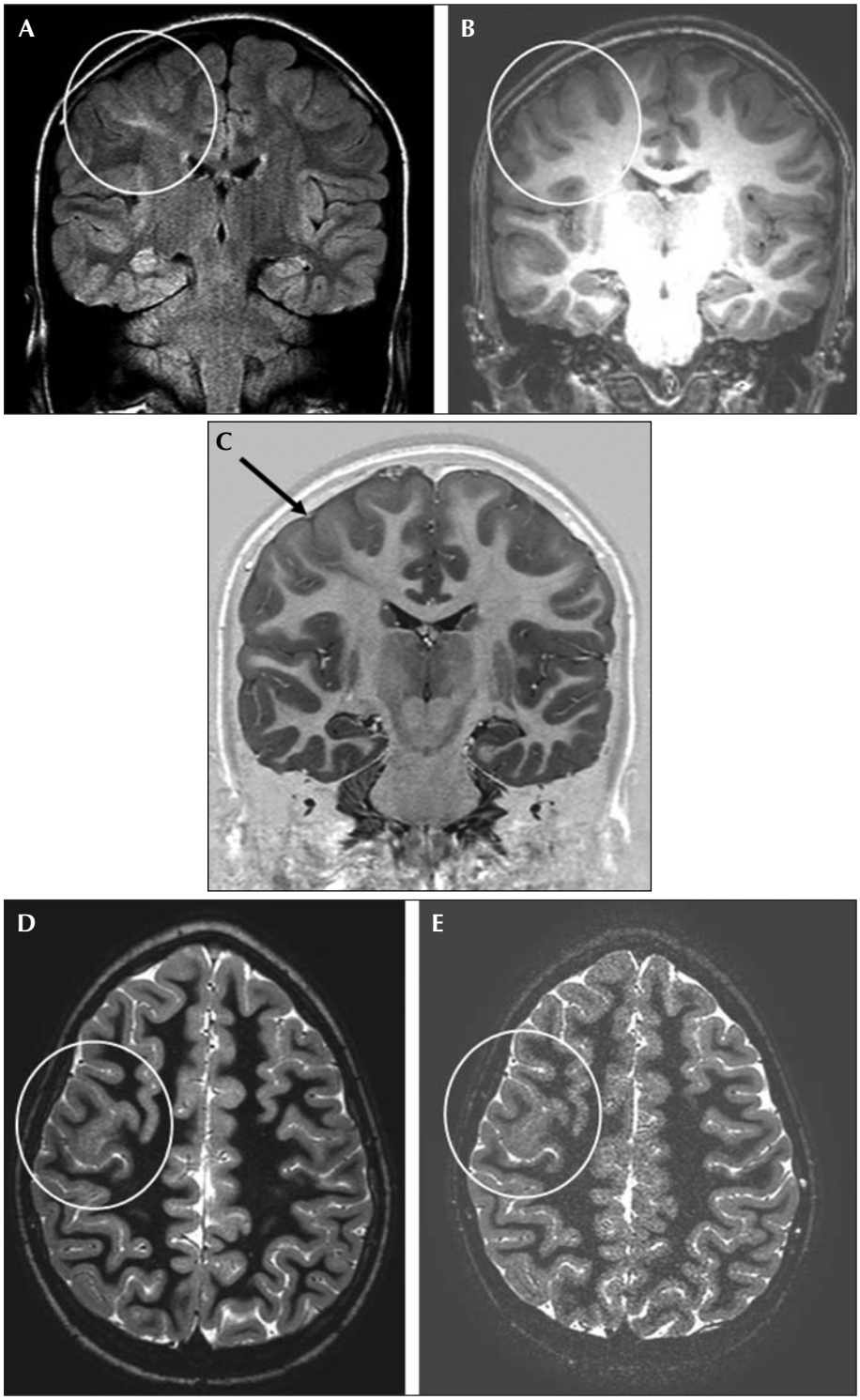

Figure 1. 13-year-old girl with right frontal lobe epilepsy (pat # 13 in *table 1*) and focal transmantle cortical dysplasia on MRI. **A**) 3T, coronal, T2-w FLAIR sequence shows right-sided, subcortical, hyperintensity extending radially towards the lateral ventricle (white circle). **B**) 3T, coronal, T1-w 3D GRE sequence confirms cortical abnormality (white circle) in the same region. **C**) 3T, head coil, coronal T1-w IR real image better depicts the cortical thickening and blurring of the cortical-white matter junction (arrow). Tissue with grey matter signal extends radially towards the lateral ventricle. **D**) Axial head coil, T1-w IR modulus image confirms the cortical thickening and blurring on the right side (white circle). **E**) The corresponding surface coil, T1-w, modulus image provides a few more details, but is noisier. Decreasing signal-to-noise ratio and thereby image quality with increasing distance from the surface coils.

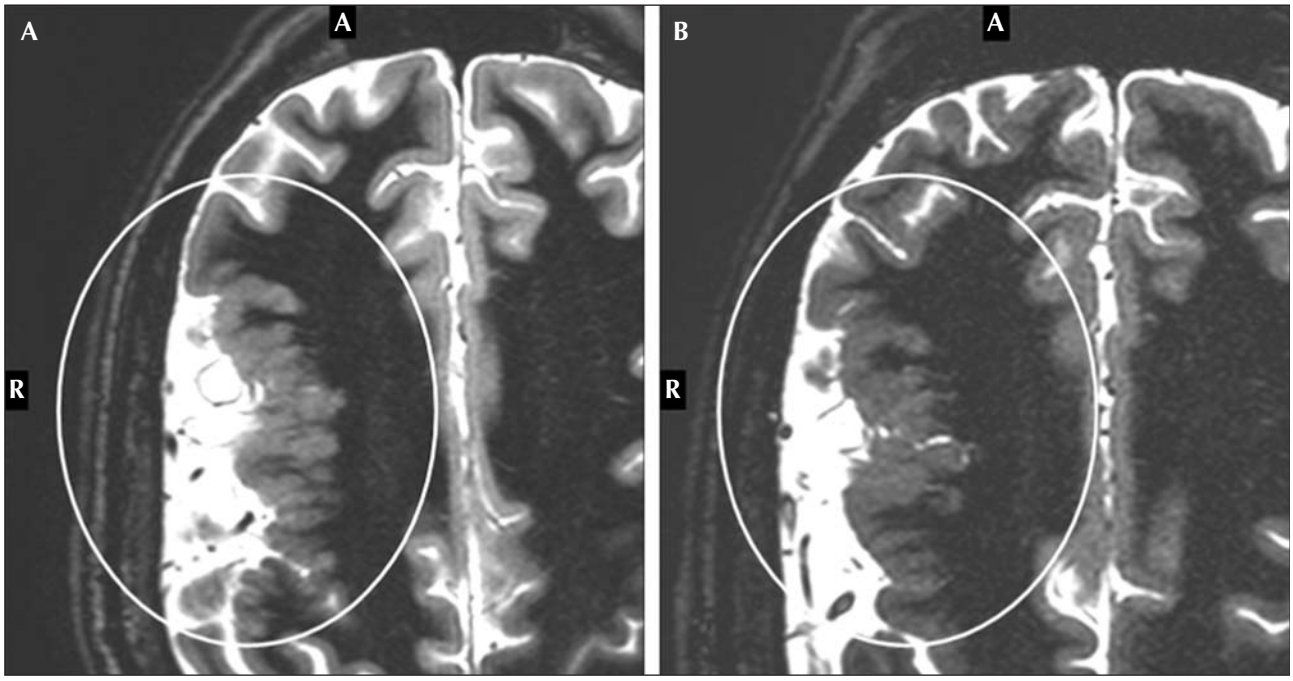

Figure 2. 51-year-old woman with right temporal lobe epilepsy (pat # 1 in *table 1*) and polymicrogyria in the right frontal and temporal lobes on MRI. **A**), **B**) Magnified, axial T1w IR modulus images obtained with head coil (**A**) and surface coils (**B**) show the slightly superior demarcation on the surface coil image.

A different choice of coil placement, taking in account all localizing data, may have contributed to a more positive outcome.

Furthermore, in four cases, surface coils were placed over the temporal lobes bilaterally illustrating an attempt to cover deeper structures not perfectly suited for this method (*table 2*; patients # 3, 5, 6 and 12). Patient # 5 experienced an abdominal aura at seizure-onset, a symptom closely linked to mesial temporal lobe epilepsy. Even if there were a good rationale for examining the mesial structures in the

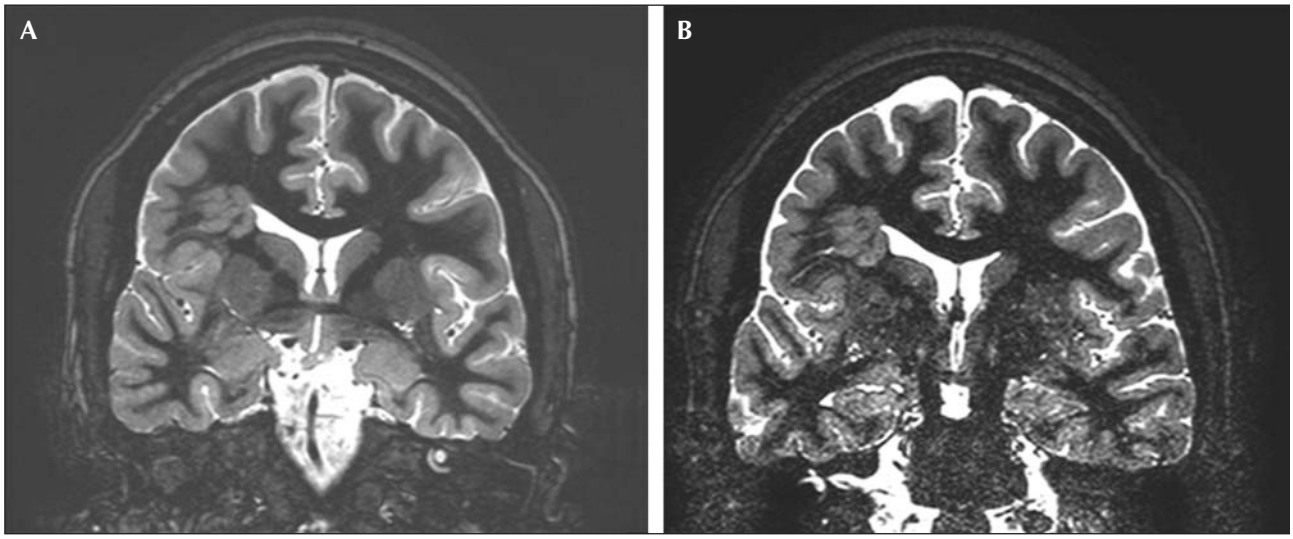

Figure 3. 28-year-old woman with partial epilepsy (pat # 4 in *table 1*) and a right parietal, subcortical (transmantle) heterotopia on MRI. **A**) 3T, T1-w IR modulus image shows the right-sided malformation (white circle). **B**) 3T, surface coil T1-w IR modulus image provides slightly better demarcation of subtle details in the right-sided, heterotopic grey matter. Decreasing signal-to-noise ratio at a distance from the surface coil with markedly deteriorated image quality in the inferior portion of the image.

Table 2. Localizing data from medical history and pre-surgical evaluation in the 1.0-1.5T MRI-negative patients. The level of evidence for coil positions was graded into three categories depending on the degree of convergence between different investigational modalities and coil placement. Grade I = good support; grade II = partial support; grade III = no support for coil placement.

Patient number (corresponding # in *table 1*)	Seizure semiology by medical history and/or video-EEG	Symtomatogenic zone (anatomical correlates)	Seizure onset zone by video-EEG	Irritative zone by interictal EEG duringVEEG/or routine EEG*	Ictal SPECT/SISCOM/PET	Surface coil placement	Evidence grade for coil placement
1 (3)	Visual aura → automotor sz, R hand sensory aura	L or R occipital and temporal lobes + L parietal lobe	R + L temporal	L temporal	-	L parietal	II
2 (5)	Visual aura → GTC sz	L or R occipital lobes	L temporal and R occipito- temporal	L + R temporal	-	L occipito-parietal	II
3 (7)	Psychic aura	TPO-junction	L temporal	L temporal	-	Bilat temporal	I
4 (11)	Aura	Non-localizing sz	No sz during monitoring	- /L + R frontal	SISCOM: L temporal	Bilat frontal + temporal	I
5 (14)	Abdominal/olfactory aura + visual aura	L or R temporal lobe + L or R occipital	L temporal	L temporal	-	Bilat temporal	II
6 (15)	Myoclonic sz	Bilat fronto-central	-	-	PET: R temporo-occipital + L temporal	Bilat temporal	II
7 (16)	Bilat tonic sz	Bilat frontal	R TPO	R temporo-occiptial	PET: R parietal	R hemisphere	II
8 (19)	L arm sensory aura	R parietal lobe	Non-localizing	R temporo-occipital	-	R central region	I
9 (21)	R motor sz	L fronto-central	-	- /L fronto-temporal	SPECT: L frontal PET: L frontal	L fronto-temporal	I
10 (22)	R arm motor sz → mouth sensory-motor sz	L fronto-central	-	- /L fronto-central	SPECT: L frontal	L hemisphere, central region	I
11 (23)	L leg motor sz	R fronto-central	-	- /R parietal	SPECT: R parietal	R frontal	II
12 (24)	Myoclonic sz with L predominance	Bilat fronto-central	Generalized	Bilat, L predominance	-	Bilat temporal	III
13 (25)	Bilat tonic sz	Bilat fronto-central	-	- /L + R frontal	SPECT: L frontal PET: L frontal	Bilat frontal	I
14 (12)	Atonic szs, aphasic szs	L frontal	R + L frontal	- /R + L frontal	SPECT: L frontal	Bilat frontal	I
15 (13)	L motor sz	R fronto-central	-	R frontal	-	Bilat fronto-central region	I

Sz: seizure; R: right; L: left; bilat: bilateral; EEG: electroencephalogram; SPECT: single-photon emission computed tomography; SISCOM: subtraction ictal SPECT co-registrated on MRI; PET: postitron emission tomography; TPO: temporo-parieto-occipital; VEEG: video-electroencephalogram; * VEEG performed locally/previous routine EEG and VEEG performed externally.

temporal lobe in this particular patient, surface coil MRI is not the method of choice for detection of hippocampal sclerosis. Enhanced delineation can only be accomplished in the cortex while there is a rapid decrease in signal intensity from the brain surface close to the coil to the centre of the head, compromising image quality for assessment of deeper, underlying structures. With improved hardware it may be possible to decrease the slice thickness for head coil T1-IR imaging. This will enable further improvement of imaging of the cortex, as well as of the deeper structures of the brain.

In conclusion, surface coils did not provide additional information over and above that provided by head coil 3T MRI in our study of patients with drug-resistant epilepsy. It was confirmed that 3T MRI with high resolution is valuable for lesion detection, especially MCDs. Considering the high quality imaging obtained at 3T when also using T1-weighted inversion recovery sequences with high resolution and excellent grey/white matter differentiation, we question the additional contribution from supplementary surface coil imaging at 3T MRI. □

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