Targeted Review

Magnetoencephalography in the presurgical evaluation of epilepsy

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Abstract

Magnetoencephalography (MEG) is an important tool in the presurgical evaluation of patients with medically refractory epilepsy. The appropriate utilization and interpretation of MEG studies can increase the proportion of patients who may be able to further pursue surgical evaluation, refine surgical planning, and potentially increase the probability of seizure freedom after surgery. The aim of this paper is to provide the reader with a comprehensive but accessible guide to MEG, with particular emphasis on acquiring a working knowledge of MEG analysis, identifying patient groups that are most likely to benefit, and clarifying the limitations of this technology.

Key questions

1. What do MEG scanners detect?
2. How is MEG data obtained and processed?
3. How accurately does MEG localize the “epileptogenic zone”?
4. Which patients should get a MEG as part of the presurgical workup?
5. How should MEG results be interpreted and utilized by the referring neurologist/epileptologist?

1. Introduction

The utility of magnetoencephalography (MEG) in surgical evaluation is often debated, despite numerous publications demonstrating that it is useful for this purpose. Some clinicians are suspicious of the complex and seemingly opaque nature of MEG analysis. Other clinicians believe that it does not independently add anything of value to the discussion. Another possible source of dissatisfaction with MEG may be irrational exuberance when first introduced to the technique, which has been subsequently over dampened as the limitations of the technique became clearer.

Although MEG analysis is complex, a working knowledge of this process can be easily acquired. Magnetoencephalography is a supplementary noninvasive test for epilepsy surgery planning that can provide clinically valid, novel localizing information in appropriately selected patients. The aim of this manuscript is to clarify the role of MEG in epilepsy surgery for the practicing clinician while avoiding technical jargon.

2. Questions

2.1. What do MEG scanners detect?

Magnetoencephalography scanners detect magnetic fields produced by the brain’s electrical activity. Electrical charges moving from one point to another generate a magnetic field at a right angle to the flow of current, the direction of which can be determined by the right-hand rule.

Magnetoencephalography signals, like EEG, are produced by excitatory and inhibitory postsynaptic potentials (EPSPs/IPSPs) rather than by action potentials (APs). Unlike EPSPs/IPSPs, APs involve a very small area of the cell membrane and do not summate by overlapping with one another [1]. Dendrites arranged in parallel columns near the cortical surface (Fig. 1A) are the major generator of summed electromagnetic signals. Synaptic activity and axonal activity are not seen on the EEG/MEG. Synapses are randomly oriented so that their fields tend to cancel each other out. Axons are influenced by APs rather than by EPSPs/IPSPs, and the symmetric transmembrane currents in axons produce fields that cancel each other out [2].
The likelihood that brain activity will be detected by MEG depends on a number of factors:

1) Depth of the source: The strength of the magnetic field is inversely proportional to the square of the distance from the electrical source. This precipitous drop-off occurs because of natural laws governing electromagnetism (the Biot–Savart law) and not because of attenuation by tissues/fluids surrounding the brain. Deeper sources may also be more likely to be affected by signal cancelation from other sources and to be suboptimally oriented in relation to the MEG sensors. Deeper structures such as the basal ganglia and cingulate gyrus would need to produce many-fold stronger electrical energy of the superficial cortex to have an equivalent probability of detection by MEG [3].

2) Orientation of the source: MEG can only detect electrical sources that are tangential to the skull surface (Fig. 1E) or sources that are “tilted” so that they have a tangential component. Purely radial current sources (orthogonal to the skull surface) do not produce a detectable magnetic field. Because of the orientation of the apical dendrites, MEG is less sensitive to electrical activity at the gyral crests and sulcal depths and more sensitive to that from the sulcal walls (Figs. 1B and C). In practice, depth is more important than orientation in determining whether an electrical discharge will be detected by MEG [3,4]. Hillebrand calculated that MEG has poor sensitivity to a very thin strip of 2 mm along the crowns of the convexity gyri, but cortical areas in the immediate vicinity produce tilted dipoles that are easily detectable (Fig. 1D) [3].

3) Interaction between multiple electrical sources in the brain: These interactions may be clinically important. For example, some researchers have proposed that opposing electrical discharges produced by the spiral anatomy of the hippocampus produce magnetic fields that cancel each other out, partly accounting for the low sensitivity of MEG for mesial TLE [5].

At this stage, it would be reasonable to consider the sensitivity of MEG for electrical activity in different areas of the brain.

• Superficial cortex, including the lateral temporal cortex: MEG is more sensitive than EEG in this region. Only 3–4 cm² of synchronized epileptiform cerebral activity is required for producing an MEG signal [6,7], while 6–10 cm² is required for producing an EEG signal [8,9]. Overall, between 50 to 90% of spikes detected on electrocorticography (ECoG) are also detected by MEG [5,10,11].

• Mesial temporal structures: These structures are far from the MEG sensors. Previous literature regarding MEG sensitivity is inconsistent. Santisteban et al. [12] found that 50% of ECoG spikes originating in the amygdala and 63% of hippocampal ECoG spikes were detected by MEG. On the other hand, other similar studies did not detect any MEG spikes corresponding to mesial temporal ECoG spikes [5,13]. A recent study without simultaneous ECoG detected spikes in 86% of patients with presumed mesial TLE [14]. There is probably more than one reason for these inconsistencies: greater sensitivity of some MEG sensors (axial gradiometers or magnetometers) to deep sources [14], inadequate differentiation between epileptiform discharges that originate and are limited to the mesial structures versus those that spread to the temporal pole or basal or lateral cortex, and the abundance of interictal activity in TLE are all plausible reasons. At this point, it should be mentioned that EEG is also insensitive to these discharges [13,15].

• Basal temporal cortex: Again, this area is far from the MEG sensors, but the orientation of this cortex may be advantageous for MEG detection because it produces currents that are vertically oriented (i.e., tangential to the skull). Magnetoencephalography can detect spikes from this region if a large area (between 3 and 8 cm²) is involved in epileptic activity [6,7,15].

• Insula: The insular cortex is relatively far from the MEG sensors and may produce mostly radial currents [16,17]. However, distortion of the insular cortex by epileptogenic structures [17] and nonradial currents associated with normal insular gyration make detection of spikes more probable than expected. Recent studies have convincingly demonstrated that epileptic activity from the insula can be detected by MEG [16–19].

• Posterior intrasylvian cortex including Heschl’s gyrus and planum temporale: MEG sensitivity in these regions is better than expected. Even more so than basal cortices, the horizontal orientation of intrasylvian cortices facilitates MEG sensitivity. Evoked potentials from Heschl’s gyrus are detectable even to the level of tonotopic mapping with millimeter precision [2]. Posterior intrasylvian spikes are detectable in 68–100% of patients with Landau–Kleffner syndrome [20–22], and MEG-directed surgery can result in dramatic clinical improvement [21,23].

• Interhemispheric fissure: MEG sensitivity is good for a length of cortex that extends about 1/3rd of the way to the corpus callosum from the convexity (Fig. 8 in Hillebrand and Barnes [3]) but is probably low for
regions very deep in the interhemispheric fissure, e.g., cingulate gyrus. Magnetoencephalography may help correctly lateralize electrical activity in the more superficial interhemispheric regions that is either difficult to lateralize (frontal lobe epilepsy) or lateralizes incorrectly (e.g., visual-evoked potentials) using EEG. In a recent study, MEG detected 90% of interhemispheric spikes recorded on subsequent ECoG, and 80% of these MEG spikes colocalized with ECoG [10].

- Orbitofrontal cortex: MEG sensitivity in this region is likely low overall, decreasing as a function of distance from the lateral surface. Magnetoencephalography detection may be facilitated by the orientation of this cortex.

2.2. How is MEG data obtained and processed?

Magnetoencephalography studies aim to capture interictal activity. A small number of patients have seizures while the MEG is being recorded. This ictal information is rare; hence, it is not discussed further.

2.2.1. Data acquisition

Magnetoencephalography scanners look like giant hair dryers (Fig. 2A). The patient lies down (or sits down in some scanners), with their head inside the scanner. The inside of the hair dryer is lined by saline-filled sphere [27] or a skull phantom [4]. Magnetoencephalography can be challenged to find the location of artificial electrical currents passed through electrodes either implanted in the human brain [25,26] or placed in an artificial construct such as a saline-filled sphere [27] or a skull phantom [4]. Magnetoencephalography is very accurate in localizing these discharges. The error in localization by MEG was between 3 to 8 mm for most studies but was 17 mm for one outlying study [26].

2.2.2. Coregistration

Coregistration (registration between functional and structural modalities) enables MEG activity to be displayed on the patient’s MRI. Before each recording run, key scalp landmarks (e.g., nasion and the preauricular points) and the location of small coils placed at 3–5 positions on the scalp are digitized with a hand-held wand or a camera system. The MEG can identify coil location within the “hair drier” because of the fact that they generate small magnetic fields of known strength. After MEG recording is complete, the key scalp landmarks are identified on a previously obtained MRI, completing the process.

2.2.3. Source localization

Epileptiform spikes are identified on the MEG using the same common sense rules that apply to EEG spikes. Artifacts produced by the heartbeat/pacemakers/VNS, etc. can usually be filtered out, e.g., in the 34 patients with VNS monitored at UAB since 2006, the study was severely limited because of VNS artifact in only 5 patients.

“Source localization” or “modeling” attempts to mathematically identify electrical sources that are consistent with the detected magnetic field. Although models assuming a perfectly spherical head are easier to compute, rapid advancements in computing have ensured that more complex models that accurately reflect patient head geometry (e.g., the “boundary element model (BEM)”) can be used instead.

For epileptiform spikes, the most common source localization used is calculation of a single equivalent dipole. The single electrical dipole (SED) is a theoretical electrical discharge, which, if it were to be present, would best explain the measured magnetic field at that instant. Although this is a theoretical construct, one could argue it is not more theoretical than the mental image that an experienced epileptologist builds in his mind after studying an interictal spike on the EEG. In both cases, we assume (explicitly in one case and implicitly in the other case) that the recordings are produced by a single epileptogenic discharge in a restricted area of the brain (SED models assume this to be a point source), although the reality may be much more complicated [24]. Occasionally, no dipole that explains the magnetic field can be calculated. This likely reflects complex distributed epileptic activity.

Alternative techniques for source localization may be used (Fig. 3). Distributed source models (e.g., LORETA and EPICFOCUS) divide the brain into many small pieces and calculate the charge at each piece so that the combination of these charges best explains the measured magnetic field. Beamforming techniques (e.g., synthetic aperture magnetometry — SAM) estimate the electrical charges in a prespecified area such that this calculation is consistent the observed magnetic fields. They are especially useful for noisy data, especially when the region of interest is known (e.g., putative epileptogenic locus, Wernicke’s area).

2.3. How accurately does MEG localize the “epileptogenic zone”?

Empiric evidence suggests that MEG is very reliable in localizing the epileptogenic zone.

2.3.1. MEG localization compared to known position of an artificial source

Magnetoencephalography can be challenged to find the location of artificial electrical currents passed through electrodes either implanted in the human brain [25,26] or placed in an artificial construct such as a saline-filled sphere [27] or a skull phantom [4]. Magnetoencephalography is very accurate in localizing these discharges. The error in localization by MEG was between 3 to 8 mm for most studies but was 17 mm for one outlying study [26].

Fig. 2. MEG scanners: (A) a MEG scanner at UAB; (B) a MEG scanner with the hood removed: the head cavity is lined by multiple (148 in this case) sensors; (C) an individual SQUID. Images courtesy of MEG International Services Ltd. and Dr. Jean-Michel Badier, Institut de Neurosciences des Systèmes, France.
error is smaller than EEG localization error (8 mm versus 10 mm in one study, 3 mm versus 7 mm in the other study) [4,25].

2.3.2. MEG localization compared with electrocorticography (ECoG)
Magnetoencephalography shows good agreement with simultaneous ECoG [6,7,28,29]. Magnetoencephalography results correlate with ECoG more consistently than EEG [28,29]. Magnetoencephalography shows good agreement with subsequently performed ECoG [30–36]. These studies indicate that all spikes have an ECoG correlate, and about 2/3rd of all ECoG spikes are detected by MEG [37]. Studies validating MEG using subsequent ECoG should be interpreted with care since epileptic discharges can vary in time and space within the irritative/epileptogenic zone [15].

2.3.3. MEG localization compared to location of epileptogenic tissue
Magnetoencephalography dipoles cluster around known epileptogenic abnormalities [31,33,35,38,39], providing strong proof for the accuracy of MEG and the computational methods for source localization. In apparently “nonlesional” cases, MEG can identify areas that, on retrospective MRI review, show epileptogenic abnormalities in oft-overlooked areas such as the insula in almost 50% of cases [18,40]. In truly cryptogenic cases (MRI true negatives), MEG findings have been

Fig. 3. Source localization: a 17-year-old male with medically intractable seizures consisting of rightward head turn followed by right arm extension and/or intermittent clonic movements. Panel 1: Spike appearance on raw EEG and MEG waveforms. Panel 2: Electrical field contour maps can be generated for EEG as well. Panel 3: Source localization results using only the MEG (magnetic) contour maps. Note the relative consistency of all source localization techniques: single equivalent dipole model (top), An example of beamforming (SAMg2) (middle). An example of distributed source modeling (SWARM). SED — single equivalent current dipole.
shown to be consistent with postresection histopathology in up to 70% of patients [36].

2.3.4. MEG localization compared to seizure freedom after resection

Incomplete resection of MEG spike clusters is associated with a worse outcome [11,41,42], and having a single well-defined cluster is a strong predictor of seizure freedom [43]. In patients with multiple epileptogenic lesions (e.g., tuberous sclerosis), MEG clusters identify the active lesion with accuracy exceeding that of ictal scalp EEG [44]. Magnetoencephalography spikes may extend beyond the MRI-visible epileptogenic lesion in about 1/2 of all FCD cases [41]. The relevance of these spikes depends on whether they cluster. Tightly clustered spikes indicate epileptogenic tissue involved in seizure onset [45]. Scattered MEG spikes (formally defined as a group of less than six spikes or spikes more than 1 cm away from other spikes) represent irritated cortex, the resection of which is not essential for seizure freedom [46].

2.4. Which patients should get a MEG as part of the presurgical workup?

Currently, MEG is utilized as a tool for presurgical workup. In this section, we present some scenarios that leverage the unique strengths of MEG (Table 1, Fig. 4) and discuss some aspects of MEG utilization in greater detail (text below). No list or discussion can fully capture the numerous permutations of clinical variables in these patients, but we hope that this section will aid the reader in evaluating whether a MEG study will help his/her individual patient.

A negative scalp EEG should prompt, rather than preempt, thoughts of getting a MEG. If we explore the data from 173 patients with varying epilepsies from three large studies [47–49], 54% (93/173) of the patients had spikes on both EEG and MEG, 7% (12/173) had spikes only on the EEG, 18% (32/173) had spikes only on MEG, and 21% (36/173) did not have spike on either modality. We can use these numbers to calculate a statistic that is more important, given the usual sequence of EEG and MEG: almost half (47%, 32/68) of all patients who do not have spikes on EEG will have spikes on MEG. Although MEG has low sensitivity to mesial temporal-only discharges, it shares this weakness with EEG [13,15].

Magnetoencephalography is not as sensitive or reliable as ECoG. However, ECoG may not define the ictal onset completely (or at all) depending on the adequacy of the preimplantation hypothesis for suspected site of seizure onset. Magnetoencephalography can be especially helpful in guiding electrode placement [55,56] to compensate for the limited spatial coverage offered by ECoG. In cases where the primary intracranial implantation does not capture ictal onset, secondary MEG-guided intracranial implantation has been shown to correctly identify epileptogenic zones not considered initially [55]. Magnetoencephalography is superior for this purpose compared to interictal PET, since PET hypometabolism is almost always vastly larger than the epileptogenic zone [57,58].

Insular epilepsy may be underdiagnosed and responsible for many temporal lobe epilepsy surgery failures [59]. Epileptiform activity from the insula is nearly impossible to see on the scalp EEG. Invasive monitoring is complicated by the myriad branches of the middle cerebral artery overlying the insula. Magnetoencephalography can be used to confirm that the likelihood of insular epilepsy is high enough to justify invasive and potentially life-threatening monitoring with insular depths. Magnetoencephalography spikes in the insula can be seen in 60–100% of patients with insular epilepsy detected by other means [16,17,19]. These retrospective analyses probably overestimate MEG sensitivity since it is likely that insular epilepsy is frequently never detected. Magnetoencephalography can be used to estimate the minimum contribution of the insula to nonlocalizable epilepsy. For example, Heers et al. [18] found insular MEG spikes in 2 out of 3 (66%) patients with no clear hypothesis regarding seizure onset. Both had good surgical outcome.

2.5. How should MEG results be interpreted and utilized by the referring neurologist/epileptologist?

It is important to remember that like all interictal studies, the MEG is a supplementary test. In the following section, we present some tips and recommendations for utilizing MEG reports.

Table 1

Sample indications for a MEG study (Fig. 4).

<table>
<thead>
<tr>
<th>Clinical scenario Comments</th>
<th>Justification for a MEG study</th>
<th>Recommendations</th>
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<tbody>
<tr>
<td>No clear hypothesis regarding seizure onset</td>
<td>MEG detects interictal spikes in about half of patients without EEG spikes [47–49]. Retrospective MEG-directed review of MRI may reveal previously undetected epileptogenic lesions in up to 50% of patients [18,40].</td>
<td>MEG results may justify potentially dangerous invasive monitoring.</td>
</tr>
<tr>
<td>Insular onset suspected</td>
<td>See text above.</td>
<td>MEG may be helpful in epilepsy with cingulate features if the focus is superficial, with early spread of seizure activity to deeper cortex.</td>
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<tr>
<td>Interhemispheric (especially frontal) onset suspected</td>
<td>MEG can detect spikes from the interhemispheric area [10,37,50]. MEG can lateralize medial frontal spikes even when the EEG is nonlateralizing because of varying dipole orientation or secondary hyssynhrony [50].</td>
<td>MEG may help to replace planned chronic ECoG with less complicated techniques, e.g., intraoperative ECoG, or (in combination with PET/SPECT/ictalEEG) may provide enough evidence for a standard ATLR.</td>
</tr>
<tr>
<td>Mesial temporal onset suspected, without clear evidence of MTS on MRI</td>
<td>MEG can detect mesial temporal spikes in about 85% of patients with mesial TLE [14]. MEG spike orientation may help in distinguishing mesial TLE from lateral TLE. Vertical or horizontal MEG spikes in the anterior temporal pole indicate a higher chance of mesial TLE [14].</td>
<td>MEG may help to replace planned chronic ECoG with less complicated techniques, e.g., intraoperative ECoG, or (in combination with PET/SPECT/ictalEEG) may provide enough evidence for a standard ATLR.</td>
</tr>
<tr>
<td>Intrasylvian onset suspected</td>
<td>MEG can detect intrasylvian spikes in EGG-negative patients [19] or may identify a single intrasylvian epileptogenic focus even when the EEG abnormalities are widespread, e.g., in LKS [20,21,23,51].</td>
<td>MEG results may justify potentially dangerous invasive monitoring.</td>
</tr>
<tr>
<td>Grid placement is planned</td>
<td>See text above.</td>
<td>MEG results may be sufficient for surgery, e.g., in TS, or may be a guide to grid placement.</td>
</tr>
<tr>
<td>Previous craniotomy including revision epilepsy surgery</td>
<td>Skull defects distort EEG, making spike identification difficult [52]. They also distort electrical fields [53] so that EEG localization may be erroneous [54]. Magnetic fields are unaffected by the structure of the skull. MEG sensitivity and localization accuracy remain unaltered.</td>
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</table>
As mentioned, MEG analysis is mostly limited to interictal spikes. Epileptic fast oscillations detectable on MEG are being investigated [50,51]. Magnetoencephalography reports may contain the results of language and sensory mapping. Magnetoencephalography mapping may be more reliable than fMRI, especially when neurovascular coupling is disturbed, such as in highly vascular tumors. Magnetoencephalography language lateralization has acceptable (but imperfect) concordance with the Wada [52,53]. We still recommend invasive mapping to confirm MEG language mapping.

The MEG report contains figures of epochs with representative epileptiform discharges. For each epoch, MEG, EEG, and flux diagrams indicating the magnetic field at one preselected timepoint in this epoch are included (Fig. 3). The magnetic flux diagrams can be used to estimate the underlying dipole. The direction of this dipole is determined by the right-hand rule applied to the magnetic flux entering and exiting the scalp. Depth can be inferred by the distance between these two flux centers — the further apart, the deeper the source. If more than a single related pair of flux maxima are present, then it should be assumed that more than one major source are present at that instant in time and that a single dipole model may not be appropriate.

The final depiction of findings in an MEG report is the superimposition of source localization results on the patient’s MRI. The single equivalent current dipole (SED) model is an abstract mathematical representation that does not necessarily represent the morphology of the actual source. The computed dipole is often placed “deep” to the actual source generator(s) not infrequently in white matter. Many, if not most, spike sources detected with MEG are generated by at least several square centimeters of cortex, and a deep source indicates that a large region of the overlying cortex is active. The orientation of dipoles (especially temporal) should be noted. All dipoles should be interpreted by using their location and orientation to infer back to what major cortical surface/plane would best generate both of these parameters. Clustering of dipoles should be noted in addition to their location and orientation.

Lastly, the MEG report may sometimes contain diagrams of other localization techniques described earlier in this paper, e.g., beamforming or distributed source modeling (Fig. 3).

We would like to highlight three potential pitfalls in utilizing MEG results:

1. In a patient suspected of having multifocal epilepsy, MEG may not identify all epileptogenic zones, especially if they are deep, i.e., MEG cannot be used to rule out the epileptic potential of areas (e.g., cingulate gyrus) to which its sensitivity is suboptimal. Conversely, MEG may successfully identify epileptogenic tissue that is not responsible for the patient’s habitual seizures, i.e., a second site not primarily responsible for the patient’s epilepsy. Just as with EEG, the referring physician should include all data, especially ictal data, to appropriately interpret MEG results.

2. In a patient suspected of having a single epileptogenic focus (e.g., stereotyped aura), MEG may detect secondary or propagated spike sources without detecting the initiating or primary source(s), resulting in another type of false localization. This scenario usually arises when the primary focus is too deep to be detected by MEG. In our experience, this may occur even when there is a paucity of spikes on the EEG possibly due to higher sensitivity of MEG for lateral cortical discharges.

3. A negative MEG study (no spikes detected) usually indicates that the sources are too small or too deep for detection. EEG–fMRI may be a promising modality for detecting deep-seated epileptic sources and networks in the future, but its utility needs to be clinically validated [54].

When epileptiform discharges are captured, MEG results may prompt one of the following conclusions:

1. Noninvasive testing (including MEG) is enough to justify proceeding straight to surgery.
2. The available evidence (including MEG) supports placement of intracranial electrodes to precisely delineate the epileptogenic zone.

3. Available localization data are insufficient to support unifocal epilepsy that is amenable to surgical resection.

If conclusions (1) or (2) are considered, unifocal and consistently oriented MEG spikes should be targeted for resection or intracranial electrode sampling when the putative localization is supported by other data, particularly ictal semiology or EEG. Scattered/isolated MEG spikes may not need to be targeted since nonresection of such spikes likely does not affect surgical outcome [46]. When conclusion (3) is reached and no other data (neurophysiological or imaging) reveal conclusive clues, the MEg may be a true negative study — the patient’s epilepsy is truly or effectively generalized, or multiple epileptogenic zones are actually present so that focal surgical resection is not likely to be effective. Great caution should be heeded in making this conclusion since the negative predictive value of MEG or any single test is poor [60].

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Disclosure statement

The authors have nothing to disclose.

Key questions (answered)

1. What do MEG scanners detect?
   Magnetoencephalography scanners detect magnetic fields produced by the brain’s electrical activity. It has excellent sensitivity for activity in the superficial areas of the cortex. The ability of MEG to detect electrical activity in deep brain structures is low.

2. How is MEG data obtained and processed?
   There are many small detectors called SQUIDs inside a MEG scanner. They produce electrical currents when the brain’s magnetic field passes through them. These measurements can be used to estimate the location and intensity of electrical sources inside the brain, a process called “source localization.” Single equivalent dipole modeling is the most common form of source localization.

3. How accurately does MEG localize the “epileptogenic zone”?
   Multiple studies have demonstrated that MEG accurately determines the location of artificial sources, ECoG findings, or MRI lesions and aids in delineating the zone required to ensure seizure freedom.

4. Which patients should get a MEG as part of the presurgical workup?
   The decision to get a MEG should be individualized; Table 1 describes some patients for whom it should be especially beneficial. In particular, all patients in whom grid placement is planned and all patients with no clear hypothesis for seizure onset should get a MEG.

5. How should MEG results be interpreted and utilized by the referring neurologist/epileptologist?
   Magnetoencephalography results are almost always intercalar and are meant to supplement other presurgical investigations. Clusters of MEG spikes should be targeted for aggressive evaluation and, possibly, resection. However, MEG may not localize deep epileptogenic lesions, and a lack of MEG clusters does not indicate an absence of surgically resectable focal abnormalities.

References
