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The value of intraoperative electrocorticography in surgical decision making for temporal lobe epilepsy with normal MRI

Neal Luther^{*}, Elayna Rubens[†], Nitin Sethi[†], Padmaja Kandula[†], Douglas R. Labar[†], Cynthia Harden[‡], Kenneth Perrine[‡], Paul J. Christos[§], J. Bryan Iorgulescu^{*}, Guido Lancman^{*}, Neil S. Schaul[¶], Dmitriy V. Kolesnik[¶], Shahin Nouri[#], Andrew Dawson[#], Apostolos J. Tsiouris^{**}, and Theodore H. Schwartz^{*,†}

^{*}Department of Neurological Surgery, Weill Cornell Medical College, New York, New York, U.S.A.

[†]Department of Neurology and Neuroscience, Weill Cornell Medical College, New York, New York, U.S.A.

[‡]Department of Neurology, University of Miami Miller School of Medicine, Miami, Florida, U.S.A.

[§]Department of Biostatistics and Epidemiology, Weill Cornell Medical College, New York, New York, U.S.A.

[¶]Department of Neurology, The New York Hospital Medical Center of Queens, Queens, New York, U.S.A.

[#]Department of Neurology, New York Methodist Hospital, Brooklyn, New York, U.S.A.

^{**}Department of Radiology, Weill Medical Cornell College, New York, New York, U.S.A.

SUMMARY

Purpose—We hypothesized that acute intraoperative electrocorticography (ECoG) might identify a subset of patients with magnetic resonance imaging (MRI)–negative temporal lobe epilepsy (TLE) who could proceed directly to standard anteromesial resection (SAMR), obviating the need for chronic electrode implantation to guide resection.

Methods—Patients with TLE and a normal MRI who underwent acute ECoG prior to chronic electrode recording of ictal onsets were evaluated. Intraoperative interictal spikes were classified as mesial (M), lateral (L), or mesial/lateral (ML). Results of the acute ECoG were correlated with the ictal-onset zone following chronic ECoG. Onsets were also classified as “M,” “L,” or “ML.” Positron emission tomography (PET), scalp-EEG (electroencephalography), and Wada were evaluated as adjuncts.

Key Findings—Sixteen patients fit criteria for inclusion. Outcomes were Engel class I in nine patients, Engel II in two, Engel III in four, and Engel IV in one. Mean postoperative follow-up was 45.2 months. Scalp EEG and PET correlated with ictal onsets in 69% and 64% of patients, respectively. Wada correlated with onsets in 47% of patients. Acute intraoperative ECoG correlated with seizure onsets on chronic ECoG in all 16 patients. All eight patients with “M” pattern ECoG underwent SAMR, and six (75%) experienced Engel class I outcomes. Three of eight patients with “L” or “ML” onsets (38%) had Engel class I outcomes.

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Address correspondence to Theodore H. Schwartz, Department of Neurological Surgery, Weill Cornell Medical College, 525 East 68th St., Box #99, New York, NY 10065, U.S.A. schwartz@med.cornell.edu.

DISCLOSURE

None of the authors has any conflicts of interest to disclose. We confirm that we have read the Journal’s position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

Significance—Intraoperative ECoG may be useful in identifying a subset of patients with MRI-negative TLE who will benefit from SAMR without chronic implantation of electrodes. These patients have uniquely mesial interictal spikes and can go on to have improved postoperative seizure-free outcomes.

Keywords

Electroencephalography; Hippocampus; Resection; Surgery

The role of surgical resection in temporal lobe epilepsy (TLE) is well-established (Wiebe et al., 2001; Jutila et al., 2002; Spencer et al., 2003; Yoon et al., 2003; McIntosh et al., 2004; Spencer et al., 2005). Although historically patients with TLE have been categorized as a single group, recent work has shown subclassifications based on clinical features, imaging, histology, and electroencephalography (EEG) (Du et al., 1993; Burgerman et al., 1995; Gil-Nagel & Risinger, 1997; Bertram & Scott, 2000; Bertram, 2009; Ogren et al., 2009; Stefan et al., 2009). One of the main challenges facing clinicians is the patient with presumed TLE but a normal magnetic resonance imaging (MRI) scan. Unlike patients with hippocampal atrophy, who have a seizure-free rate after surgery approaching 70–80%, TLE with normal MRI has a 40–60% rate of surgical cure (Berkovic et al., 1995; Holmes et al., 2000; Ozkara et al., 2000; Cohen-Gadol et al., 2005; Alarcón et al., 2006; Tatum et al., 2008; Bell et al., 2009). Ambiguities about seizure focus localization pose a challenge to resection, since onsets may be either neocortical or mesial and be clinically indistinguishable (Sylaja et al., 2004; Chapman et al., 2005; Bell et al., 2009). For these patients, chronic intracranial electrocorticography (ECoG) is often employed to acquire ictal data to guide temporal lobe resection (Cascino et al., 1995; Siegel et al., 2001; Kuruvilla & Flink, 2003; Cohen-Gadol et al., 2005; Oliveira et al., 2006).

Although relatively safe, electrode implantation significantly increases length of stay and is associated with a 10–26% rate of complications, including hematoma and infection (Van Gompel et al., 2008; Wong et al., 2009). The risks and costs are higher than a single-stage surgery (Hamer et al., 2002). For this reason, techniques to localize seizure foci such as positron emission tomography (PET), ictal single-photon emission computed tomography (SPECT), functional (f)MRI, and magnetoencephalography (MEG) have been pursued (Assaf et al., 2003, 2004; Stephen et al., 2005; Willmann et al., 2007; Boling et al., 2008; Chassagnon et al., 2009; la Fougere et al., 2009; Kobayashi et al., 2009). Clinical history, neuropsychological, and Wada testing can also assist in this distinction (Loring et al., 1994; Sperling et al., 1994; Sylaja et al., 2004). However, none of these adjuncts have high enough sensitivity and specificity to completely obviate the need for ECoG in these patients.

Another useful technique for localizing epileptogenic cortex is acute (intraoperative) ECoG. The main limitation of acute ECoG is that usually only interictal data is recorded. Acute ECoG has been proven helpful in defining the limits of neocortical lesional epilepsy surgery; however, its use in TLE is controversial (Schwartz et al., 1997; Holmes et al., 2000; Oliveira et al., 2006). Studies have evaluated the predictive value of postresection spikes or preresection spike-tailored resections (Schwartz et al., 1997; McKhann et al., 2000; Chen et al., 2006; Oliveira et al., 2006), but few have correlated the results of acute ECoG with chronic ECoG. Likewise, there are few data on the utility of intraoperative ECoG in MRI-negative TLE. A recent report showing a strong correlation between the distribution of intracranially recorded interictal spikes and ictal onsets in TLE supports an interest in evaluating acute ECoG efficacy in MRI-negative TLE (Goncharova et al., 2009).

The purpose of this study was to determine if acute ECoG could screen patients with MRI-negative TLE to select who would require chronic implantation of electrodes and who could

move directly to a standard anteromedial resection (SAMR) (Spencer et al., 1984). We also hypothesized that these patients might have better outcomes with respect to seizure control.

Methods

Patient selection

We retrospectively examined a prospectively acquired database of patients who underwent epilepsy surgery for medically refractory seizures between 2002 and 2008 by the senior author (THS). Weill Medical College of Cornell University Institutional Review Board approval was obtained for this study. Of a total of 291 operations, we identified a subgroup with presumed TLE by scalp EEG and a normal MRI scan. The clinical, imaging, and scalp EEG findings were first reviewed in a multidisciplinary epilepsy surgery conference, in which patients were identified as having both TLE and a normal MRI. Scalp EEG recordings or reports (in cases where EEG data were not available) were rereviewed by a single blinded epileptologist (EOR) who confirmed the diagnosis of TLE and characterized the seizure type as one of the following: “M” for mesial onset pattern, “L” for temporal neocortical onset pattern, or “ML” if the recording did not meet criteria for M or L or if onsets contained features of both. Mesial “M” onset pattern was defined as a 5–9 Hz anterior temporal or centrottemporal rhythmic activity with a sustained, regular morphology at the seizure onset. Neocortical, “L,” onset patterns were defined as a slow (<5 Hz), irregular or periodic, or temporal or hemispheric rhythm that was poorly sustained at ictal onset (Ebersole & Pacia, 1996; Dantas et al., 1998; Foldvary et al., 2001).

All preoperative MRIs were then rereviewed by a single blinded neuroradiologist (AJT) to confirm that there was no pathology on the MRI. Manual volumetrics were performed to rule out mesial atrophy. PET scans and Wada testing results were reviewed when available. The PET results were classified by visual analysis as “M” if hypometabolism involved the mesial structures with or without lateral cortex hypometabolism, since the area of hypometabolism is known to be larger than the ictal onset zone in mesial onset TLE (Carne et al., 2004; Schwartz, 2005). They were classified as “L” if only the lateral neocortex was hypometabolic, or “N” if normal. The Wada was classified as either “M” mesial if there was a significant memory disparity implying decreased memory (defined as recall of <50% of items tested) in the epileptic hemisphere or “L” if there was no disparity between hemispheres or a disparity implying decreased memory in the nonepileptic hemisphere (Perrine et al., 1995). These latter patients, of which there were none in this study, would need to pass a superselective Wada to be considered for surgery at our center.

Surgical procedure and electrocorticography

All patients underwent implantation of electrodes for chronic ECoG monitoring of ictal onsets. An 8 × 6 contact grid was placed over the temporal lobe, extending from the sphenoid wing and from the inferior temporal gyrus to just above the sylvian fissure. Six-contact strip electrodes were positioned in the following locations: (1) parallel to the sylvian fissure, around the temporal tip, extending to the amygdala; (2) perpendicular to the sylvian fissure, underneath the temporal lobe and in front of the petrous bone, extending to the tentorium and anterior mesial structures; (3) perpendicular to the sylvian fissure, underneath the temporal lobe and behind the petrous bone, extending towards the tentorium and posterior mesial structures; (4) toward the parietooccipital lobes; and (5) underneath the frontal lobe (Fig. 1). Intraoperative ECoG proceeded for a period of 5 min. The only anesthetics used during, and for 20 min prior to, the recording were remifentanyl (0.05–0.25 µg/kg per minute) and <0.2% isoflurane. The ECoG was divided by the surgeon at the time of surgery into the following groups: “M” spikes arising only from the mesial structures (including parahippocampal gyrus and temporal tip, represented by the most medial three

contacts of electrodes 1, 2, and/or 3 described above), “L” spikes coming only from the lateral cortex (any other contacts than those described above), or “ML” if spikes were coming from both areas. Following the ECoG recording, all patients had a stereotactic depth electrode placed into the midportion of the hippocampus, tangentially through the inferior temporal gyrus. Additional grids and strips were added as needed if spikes were recorded from any of the lateral contacts, thereby ensuring that the interictal spikes were not located at the edge of a grid or strip. Patients were brought to the epilepsy monitoring unit, where doses of antiepileptic medications were slowly lowered and monitored using the XLTEK system (XLTEK, Toronto, ON, Canada) until at least three seizures were recorded. Patients underwent postoperative skull x-ray, CT scan, and MRI of the brain to confirm electrode placements.

Ictal onsets were then categorized by the surgeon and epileptologist prior to surgery in the same fashion as the acute data had been: either “M,” “L,” or “ML,” based on the determination of the region of seizure onset. SAMR at our institution is a slight modification of the technique described by Spencer et al. (1984), namely, removal of the anterior 3 cm of middle and inferior temporal lobe neocortex, the anterior temporal tip, entorhinal cortex, and parahippocampal gyrus, combined with a complete amygdalohippocampectomy. Because the purpose of this study was to determine which patients could have these structures removed without the need for chronic ECoG, we categorized the ictal onsets as “M” if they arose from either the amygdalohippocampal complex, parahippocampal gyrus/entorhinal cortex, or the anterior temporal tip. Although onsets from the temporal tip are not by strict definition truly mesial in onset, for the purposes of this study such a classification facilitates data analysis because these patients undergo the same surgical procedure as patients with truly mesial onsets. Onsets were categorized as “ML” if they simultaneously occurred mesially and laterally.

The patients were subsequently brought to the operating room for resection guided only by the results of the chronic ECoG. SAMR was performed on “M” onsets, regardless of the interictal data. If the onsets were lateral, then the resection consisted of removal of the ictal onset zone and areas of frequent interictal spiking, unless extraoperative stimulation mapping demonstrated critical language areas within 1 cm of the resection, in which case the resection was limited to preserve these areas (Ojemann, 1983). In addition, mesial structures of the nondominant side were removed if involved early in the seizure spread, whereas the hippocampus was spared on the dominant side. If onsets were “ML,” then both the neocortical and mesial regions were resected, with sparing of critical language areas in the dominant temporal lobe. Engel classification was employed to categorize seizure outcome (Engel et al., 1993). Kaplan-Meier survival analysis was performed to evaluate seizure-free survival following surgery, and the log-rank test was performed to compare seizure-free survival between patients with “M” versus “ML” or “L” acute ECoG recordings. All p-values are two-sided with statistical significance evaluated at the 0.05 alpha level. Ninety-five percent confidence intervals were calculated to assess the precision of the obtained estimates. All analyses were performed in STATA Version 11.0 (StataCorp, College Station, TX, U.S.A.).

Results

Patient demographics and results are summarized in Table 1. In total, 16 patients met the criteria for inclusion, of which 8 were male. The patients’ ages ranged from 20–47 years (median age 35 years). Median seizure frequency preoperatively was five per month. Scalp EEG demonstrated “M” onsets in two patients, compared with “L” onsets in 10 patients, and “ML” in four patients. PET scan reports were available for 14 patients, and were categorized as “M” in 10 patients, five of whom had “M” ictal onsets on chronic intracranial

ECoG. PET scans showed hypometabolism classified as “L” in two patients, both of whom had “L” ECoG onsets, whereas two patients had normal scans. Wada revealed that surgery was carried out on the dominant hemisphere in 10 patients. Memory scores were classified as “M” in six patients, only two of whom had ECoG ictal onsets classified as “M.” Overall, localization from scalp EEG and PET hypometabolism correlated with ictal onsets from chronic ECoG in 56% and 64% of patients, respectively. Wada memory scores correlated with onsets in 47% of patients. In no patients were the scalp EEG, PET, and Wada all concordant. Antiepileptic medications given just before the start of surgery were consistent with the patient’s preoperative regimen, which included levetiracetam in two patients, carbamazepine in four, lamotrigine in four, phenytoin in three, zonisamide in three, valproate in two, topiramate in one, and oxcarbazepine in one. Ten patients were on combination therapy; in all instances medications were withheld temporarily during chronic ECoG recordings.

Intraoperative ECoG revealed interictal activity arising uniquely from the mesial contacts (M) in eight patients, from the lateral contacts (L) in four patients, and from both mesial and lateral contacts (ML) in four patients. Of the eight patients whose acute ECoG demonstrated “M” spiking, ictal onsets were “M” in all patients, localized to the hippocampus in five patients and parahippocampal gyrus or temporal tip in three patients. All eight patients whose acute ECoG was “ML” or “L” had ictal onsets corroborating intraoperative recordings (Table 1). Overall, intraoperative ECoG correlated with chronic intracranial recordings in all 16 patients (100% sensitive and specific). There were two operative morbidities and no mortalities associated with all surgery (6% complication rate). This included one case of a superficial wound infection and another case of a hematoma (both epidural and subdural) postelectrode placement, which required operative evacuation on postimplant day 3. Both patients ultimately recovered well without permanent sequelae.

Median and mean postoperative follow-ups were 41.5 and 45.2 months, respectively (range 9–86 months). Of the 16 patients total, nine (56%) had Engel class I outcomes, two had class Engel II outcomes, four had Engel class III outcomes, and one had an Engel IV outcome. Kaplan-Meier analysis of seizure-free survival after surgery is plotted in Fig. 2. Median seizure-free survival time after surgery was 55 months [95% confidence interval (CI) 3 months, upper limit not estimated]. Of the five patients with Engel class III or worse outcome, three had no abnormalities on histopathologic evaluation of resected specimens. Eight of the remaining 11 patients with Engel class I and II outcomes had abnormal pathology.

All eight patients with “M” pattern acute ECoG proceeded to have a SAMR, and six (75%) of these patients experienced Engel class I outcomes. Of the eight patients with “M” acute pattern ECoG, six had lateral neocortical interictal spikes on chronic ECoG, of whom five of six had Engel class I outcome following SAMR. In contrast, in the “L” and “ML” subgroups only, three of eight patients (38%) had Engel class I outcomes. The log-rank test demonstrates that seizure-free survival was significantly longer in patients with “M” versus “ML” or “L” acute ECoG recordings ($p = 0.03$, median seizure-free survivals of 73 and 23 months in the two groups, respectively). These Kaplan-Meier curves are plotted in Fig. 3.

Discussion

In patients with normal MRI scans, identifying the anatomic etiology of the seizures and the potential target for surgical intervention can be a challenge. When seizures arise from radiographically normal mesial structures, this syndrome has been labeled “paradoxical TLE” and shown to have lower incidence of febrile seizures, higher incidence of secondary generalized seizures, and less evidence of cell loss on histology (Cohen-Gadol et al., 2005).

These patients often undergo chronic implantation of electrodes to identify the ictal onset zone prior to resective surgery (Cascino et al., 1995; Siegel et al., 2001; Kuruvilla & Flink, 2003; Cohen-Gadol et al., 2005; Oliveira et al., 2006). Despite these invasive measures, rates of seizure freedom are still lower than those in patients with hippocampal sclerosis on MRI (Schwartz et al., 2006). Although relatively safe, the risks of chronic electrode implantation include hemorrhage, infection, and wound breakdown, all of which can also cause psychological stress to the patient (Hamer et al., 2002; Van Gompel et al., 2008; Wong et al., 2009). Inpatient hospitalization can be lengthy, accompanied by substantial costs. Complications are more commonly associated with grid implantation than seizure focus resection. Complication rates have been cited in large series as high as 15–26% for grid implantation and 2–4% for resection, further evidencing the need for alternative methods to chronic grid recordings (Lee et al., 2000; Hamer et al., 2002). Both complications in the current series resulted from grid implantation.

In this retrospective study, we have shown that acute intraoperative ECoG can be used as a screening tool to identify a subset of patients, with TLE and normal MRI scans, who may avoid chronic implantation of electrodes and proceed directly to SAMR. These patients have large, frequent interictal spikes arising uniquely from the mesial structures during the brief sample acquired in the operating room, while lacking interictal spikes generated by other locations. In this series, none of these patients went on to have lateral ictal onsets on their chronic ECoG. All ultimately underwent SAMR, including removal of the parahippocampal gyrus and anterior temporal tip, which theoretically could have been performed at their first operation had they been identified at this earlier stage. Lateral interictal spiking on chronic ECoG did not correlate with worsened outcome following SAMR. Surgical outcome in these patients is comparable to that in patients with hippocampal sclerosis. On the other hand, patients with any acute ECoG interictal spikes outside of the mesiobasal region, or without large frequent uniquely mesial spikes, still require chronic implantation of electrodes to guide their ultimate surgical resection, and may have a worse outcome.

Chronic implantation of intracranial electrodes has contributed enormously to our understanding of TLE. In recent years such studies have demonstrated that the temporal lobe consists of a complex network of potentially epileptogenic structures, all of which interact in a dynamic fashion. As a result of these studies, several subtypes of TLE have been identified. In one such categorization, five major etiologies have been characterized: (1) The mesial subtype, in which seizures arise from the amygdalohippocampal complex, parahippocampal gyrus, or entorhinal cortex; (2) The temporopolar subtype, in which seizures arise from the temporal pole alone, or concurrently with the mesial structures; (3) The mesiolateral subtype, in which seizures arise simultaneously from both mesial and lateral structures; (4) The lateral subtype, in which seizure arise only from the lateral temporal neocortex; and (5) The temporal “plus” subtype, in which seizures arise from a complex network including not only the temporal lobe, but also the insula, orbitofrontal region, frontal and parietal operculum, and temporal parietal junction (Kahane & Bartolomei, 2010). Using this schema, we have shown that we can identify types 1 and 2 using acute intraoperative ECoG. After additional validation of this technique, we can potentially move these patients directly to SAMR, whereas all other patients will undergo chronic implantation of electrodes to further characterize their seizure onsets and direct surgical planning.

The value of scalp and intracranially recorded interictal spikes in the presurgical work-up of patients with TLE is well-researched but controversial. Although some have shown that spikes can occur in widespread areas of brain despite focal epilepsy (Lieb et al., 1980; Lange et al., 1983; Kanazawa et al., 1996; Alarcon et al., 1997; Hufnagel et al., 2000), others report a good concordance between interictal spikes and seizure onset, depending on specific

parameters (Katz & Spencer, 1989; Alarcon et al., 1997; Hufnagel et al., 2000; Carreno&Luders, 2001; Goncharova et al., 2009). More precisely, higher amplitude, higher frequency spikes may be predictive of seizure onset zone, and identification of the initiation site using computer algorithms may correlate with the ictal onset zone and outcome (Alarcon et al., 1997; Hufnagel et al., 2000; Goncharova et al., 2009). Using both acute intraoperative and extraoperative ECoG, respectively, Alarcon et al. (1997) and Hufnagel et al. (2000) showed that despite widespread interictal activity, quantification of the earliest spike provided the best correlation with the ictal onset zone (84%), and resection of these areas correlated with favorable outcome in 93% of patients. Amplitude and frequency were less predictive, although contralateral hippocampal spikes were the most commonly located extraictal site in patients with mesial TLE, an area that would not have been recorded in our patients. Goncharova et al. (2009) recently reported on the distribution of intracranially recorded interictal spikes during 4 h epochs in a series of 21 patients with TLE. Although patients with mesial and lateral onsets both had a wide distribution of spikes, using criteria for dominant high-frequency spikes, they were able to distinguish between these two groups based on the spatial distribution and frequency of dominant spikes. However, no previous investigator has investigated preresection ECoG in patients with MRI-negative TLE as a screening tool or to predict outcome.

Holmes et al. (2000) demonstrated that scalp-recorded mesiobasal interictal spikes predict better outcome after tailored temporal lobectomy in patients with normal MRI. However, they did not study the predictive value of the distribution of spikes recorded from the acute intraoperative ECoG. Prior reports have also shown that anterior temporal scalp-recorded interictal spikes or ictal rhythmic temporal theta ("M" type onset) predict positive outcomes in patients with TLE and normal MRI, but again, ECoG was not used in these studies (Sylaja et al., 2004; Tatum et al., 2008; Bell et al., 2009). PET scans have also been used to select a subgroup of TLE patients with normal MRI that have a better outcome after surgery (Carne et al., 2004). SPECT as well has been shown to be useful (Bell et al., 2009). We did not routinely perform SPECT; however, the other adjuncts were all less effective than acute intraoperative ECoG in selecting patients with MRI-negative TLE for SAMR and predicting postoperative outcome.

Limitations

The main criticism of this paper is the potential for miscategorizing a patient based on such a brief intraoperative recording under anesthesia. Two errors can be made. It is known that some patients with mesial TLE will have spikes recorded from the lateral neocortex during ECoG (Schwartz et al., 1997). Although these patients might benefit from immediate SAMR, chronic ECoG would be prudent to rule out neocortical onsets. The use of light anesthesia, which could be criticized, may dampen these small infrequent propagated spikes and reduce the size of this misclassified group. The second possible error would be misclassifying a patient with true lateral onsets or dual pathology as having mesial epilepsy and treating them prematurely with SAMR. How frequently such patients would display uniquely mesial interictal spikes on ECoG is unknown. However, this situation did not occur in the present study. Such a patient would likely fail anteromesial temporal resection based on the existence of an unidentified, widespread epileptic focus. Of the eight patients we identified with "M" pattern ECoG, a 75% Engel I outcome was demonstrated, indicating that the frequency of a non-mesial-only onset misclassification event may be rare. Finally, it is important to realize that in this subgroup of MRI-negative patients with TLE, the laterality of onsets was known from the scalp EEG. Hence, our findings may apply only to a subset of MRI-negative TLE patients.

Another potential criticism may center on the use of light anesthesia during acute ECoG. Most general inhalational anesthetics will reduce frequency and amplitudes of spikes, and

must be used sparingly during ECoG recordings (Gloor, 1975; Sloan, 1998; Kuruville & Flink, 2003). Alternatively, recordings might be more sensitive if performed under local anesthesia, as done in traditional tailored surgery (Ojemann, 1987). It is our hope that other groups who have performed intraoperative ECoG in patients with MRI-negative TLE, under either light general or local anesthesia, will review their data to either corroborate or discredit our proposal.

The use of visual identification of spikes on acute ECoG rather than a more objective computer algorithm represents another shortcoming. Further studies of the value of interictal spike distribution as a screening tool in this particular subgroup of patients will benefit from a more objective analysis. It is important to note that centers that perform a selective amygdalohippocampectomy, either trans-sylvian or transtemporal, will perform an inadequate resection in a subset of patients with onsets in the anterior temporal tip. For this reason, we favor a modified Spencer technique as described previously.

Lastly, sample size must be considered as a limitation of this study. The survival curves in Figure 3 should be interpreted with caution given the small number of failures in the “M” acute ECoG recording subgroup. Although this analysis had sufficient power to detect a difference between median seizure-free survival between the “M” and the “ML/L” subgroups ($p = 0.03$), the width of the resulting confidence intervals for the median seizure-free survival times (i.e., upper limits not estimated) indicate the lack of precision in the log-rank analysis. A larger sample size would be necessary to estimate the median seizure-free survival times with a greater level of precision. Sixteen patients comprise a relatively large series of operative MRI-negative patients with TLE, and the findings of the acute ECoG were strongly predictive of whether patients would ultimately demonstrate lateral onsets, but it is important to recognize that it is not expected that 100% of patients with mesial onsets will be appropriately identified using this technique, as was the case in our experience.

Conclusions

In summary, we propose that acute intraoperative ECoG can be used in patients with MRI-negative TLE to screen for patients who will benefit from SAMR and avoid the need for chronic implantation of electrodes. These patients will not benefit from chronic implantation of electrodes and their outcome rivals patients with hippocampal sclerosis. On the other hand, patients whose acute ECoG demonstrate any spike outside the mesial structures or anterior temporal tip should be implanted with electrodes, although their outcome may not be as favorable as the former group.

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Figure 1.

Lateral (A) and ventral (B) schematic diagrams of the brain showing electrode locations used for intraoperative ECoG as well as chronic monitoring. A depth electrode was additionally placed in the hippocampus for chronic monitoring. “M” denotes contacts used to determine mesial interictal spikes. Spikes at any other contact were considered lateral.

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Figure 2.
Kaplan-Meier analysis of failure-free survival as defined as first postoperative seizure.
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Figure 3.
 Failure-free survival as defined as postoperative seizure in patients with “M” onsets (blue line) versus those with “L” or “ML” onsets (red line).
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Table 1

Patient characteristics

Sex	Age	Lateral	Pathology	Frequency ^a	EEG	PET	Wada	Intraoperative ECoG	Chronic ECoG	Engel class
M	38	L	Infarction, hemorrhage	3	M	M	L	M	M (AH)	I
F	30	L	Gliosis	4	L	L	M	L	L	III
F	32	R	Normal	2	L	M	M	L	L	III
M	45	L	Normal	6	L	M	M	M	M (PHG/TT)	II
M	47	L	Normal	4	L	M	M	M	M (PHG/TT)	III
F	26	L	Inflammation	8	L	M	L	M	M (AH)	I
F	38	R	Sclerosis, gliosis	20	L	M	L	L	L	I
F	20	L	Sclerosis, gliosis	8	L	NA	L	M	M (PHG/TT)	I
M	46	L	Normal	13	L	N	M	M	M (AH)	I
F	35	L	Neuronal loss, gliosis	10	M	M	L	M	M (AH)	I
M	28	L	Normal	4	ML	NA	L	ML	ML	IV
M	40	R	Neuronal loss, gliosis	5	L	L	M	L	L	I
F	22	R	Neuronal loss, gliosis	10	ML	N	Fail	ML	ML	III
M	35	R	Normal	12	ML	M	L	ML	ML	I
F	40	R	Neuronal loss, gliosis	16	L	M	L	ML	ML	II
M	45	L	Sclerosis	4	ML	M	L	M	M (AH)	I

PHG/TT, parahippocampal gyrus/temporal tip; AH, amygdalohippocampal complex; NA, not available.

^aFrequency is in seizures per month.