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Clinical value of magnetoencephalographic spike propagation represented by spatiotemporal source analysis: Correlation with surgical outcome

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Abstract

Objective—To investigate the correlation between spike propagation represented by spatiotemporal source analysis of magnetoencephalographic (MEG) spikes and surgical outcome in patients with temporal lobe epilepsy.

Methods—Thirty-seven patients were divided into mesial (n=27) and non-mesial (n=10) groups based on the presurgical evaluation. In each patient, ten ipsilateral spikes were averaged, and spatiotemporal source maps of the averaged spike were obtained by using minimum norm estimate. Regions of interest (ROIs) were created including temporoparietal, inferior frontal, mesial temporal, anterior and posterior part of the lateral temporal cortex. We extracted activation values from the source maps and the threshold was set at half of the maximum activation at the peak latency. The leading and propagated areas of the spike were defined as those ROIs with activation reaching the threshold at the earliest and at the peak latencies, respectively. Surgical outcome was assessed based on Engel's classification. Binary variables were created from leading areas (restricted to the anterior and mesial temporal ROIs or not) and from propagation areas (involving the temporoparietal ROI or not), and for surgical outcome (Class I or not). Fisher's exact test was used for significance testing.

Results—In total and mesial group, restricted anterior/mesial temporal leading areas were correlated with Class I ($p < 0.05$). Temporoparietal propagation was correlated with Class II-IV ($p < 0.05$). For the non-mesial group, no significant relation was found.

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Conclusions—Spike propagation patterns represented by spatiotemporal source analysis of MEG spikes may provide useful information for prognostic implication in presurgical evaluation of epilepsy.

Keywords

magnetoencephalography (MEG); temporal lobe epilepsy; epilepsy surgery; spike propagation; minimum norm estimate (MNE)

INTRODUCTION

Investigating the origin and propagation of epileptic spikes is important for understanding the pathophysiology of epilepsy. Patterns of spike propagation may directly reflect the abnormal neural networks underlying epilepsy (Spencer, 2002). These patterns may also be correlated with outcome of epilepsy surgery and clinically informative in presurgical evaluation (Alarcon et al., 1997, Hufnagel et al., 2000, Schulz et al., 2000). Previous studies have reported the propagation patterns observed on intracranial electroencephalography (IEEG) (Lieb et al., 1991, Alarcon et al., 1994, Alarcon et al., 1997), however, IEEG has limited coverage and the risk of complications.

Magnetoencephalography (MEG) is a non-invasive method that records neuromagnetic fields from the whole brain. Previous studies have reported its usefulness in presurgical evaluation of epilepsy by using conventional single dipole analysis of interictal MEG spikes (Iwasaki et al., 2002, Assaf et al., 2004), whereas spatiotemporal distributed source analysis such as minimum norm estimate (MNE) provides more accurate localization of spikes (Shiraishi et al., 2005, Tanaka et al., 2009). Several studies of scalp electroencephalography (EEG) (Waberski et al., 2000, Huppertz et al., 2001, Zumsteg et al., 2006) and MEG (Tanaka et al., 2010) have shown that spatiotemporal source distribution accurately represents interictal spike propagation observed on IEEG. However, these studies focused on the accuracy of the analysis compared with IEEG, and the correlation of the EEG/MEG findings with surgical outcome has not been investigated, i.e., the feasibility of this method in presurgical evaluation has not been well established.

Interictal temporal spikes are commonly observed in patients with mesial and non-mesial temporal lobe epilepsy, which is often medically intractable and requires surgery. Outcome of temporal lobe surgery results in seizure freedom in 60-70% of cases, however, 30-40% experience seizure recurrence, despite resection of the epileptogenic zones, including mesial structures and the anterior part of the temporal lobe (Radhakrishnan et al., 1998, Wiebe et al., 2001). Previous studies have reported several prognostic factors, such as duration of epilepsy and the presence of hippocampal atrophy (Radhakrishnan et al., 1998, Wiebe et al., 2001). Several researchers have suggested a relation between spike propagation and surgical outcomes, reporting that 1) interictal spikes restricted to the mesial/anterior temporal areas (Iwasaki et al., 2002, Sylaja et al., 2004, Chen et al., 2006), and 2) ipsilateral temporo-parietal involvement (Holmes et al., 2000, Aghakhani et al., 2004) may be predictive for favorable and unfavorable outcomes, respectively. However, the observations of these studies were based on visual inspection, and a quantitative approach may be necessary for understanding clinical values of spike propagation.

The purpose of this study is to investigate the propagation patterns of interictal MEG spikes represented by MNE, and to correlate these patterns with surgical outcome in patients with temporal lobe epilepsy. Specifically, we focused on the leading and propagated areas of ipsilateral temporal spikes, and hypothesize that:

1. Presence of spikes with a leading area restricted to the anterior and mesial temporal lobe correlate with favorable outcome.
2. Presence of spikes which propagate to temporoparietal area correlate with unfavorable outcome.

PATIENTS AND METHODS

Patients

We retrospectively selected 37 patients (17 male, 20 female, age 9-68, mean 26 years old) with mesial or non-mesial intractable temporal lobe epilepsy who underwent both presurgical evaluation with MEG and subsequent surgery between 2002 and 2012. The diagnosis was based on comprehensive evaluation, including long-term video-EEG monitoring, magnetic resonance imaging (MRI), neuropsychological testing, interictal/ictal single photon emission computed tomography (SPECT) and positron emission tomography (PET). MEG was performed as a part of the presurgical evaluation. In all patients, ictal EEG patterns were found in the affected temporal lobe, while interictal EEG showed uni- or bilateral spikes with ipsilateral predominance. These patients were classified into mesial (n=27) and non-mesial (n=10) groups based on the clinical diagnosis. Patients were post-operatively followed up at 12 to 114 months (mean: 41 months) and surgical outcome of each patient was assessed based on Engel's classification at time of last follow-up (Engel et al., 1987). The detailed clinical data are shown on Table 1. The study complies with the Declaration of Helsinki. All aspects of the study were approved by the institutional review board of Massachusetts General Hospital. Informed consent was obtained from all patients or their guardians.

Data acquisition

MEG was recorded with a 306-channel (204 gradiometers and 102 magnetometers), whole-head MEG system (Elekta-Neuromag, Helsinki, Finland). EEG was simultaneously recorded according to International 10-20 system. The sampling rate was 600Hz with a band-pass filter of 0.1-200Hz. In all patients, anatomical MRI data were acquired with magnetization-prepared rapid acquisition gradient-echo sequences (MPRAGE; TE=3.37 ms, TR=2000 ms, voxel size=1×1×1 mm) with a high-resolution 3T scanner (TIM TRIO, Siemens AG, Erlangen, Germany).

Preprocessing

MEG data were digitally filtered at a bandpass of 1-40 Hz in off-line data analysis. Bad channels with frequent mechanical artifacts were manually removed and the data were processed with temporally extended signal space separation method for further reduction of artifacts (Taulu et al., 2004, Taulu and Simola, 2006). We visually examined the preprocessed data and selected 10 ipsilateral spikes with similar morphology, which appeared most frequently in each patient. This selection and further analysis were performed blindly to the surgical outcomes for avoiding selection bias. These spikes were averaged by using peaks at the channel showing the maximum amplitude as a trigger (0ms) with a time window between -50 and +50ms.

Source analysis (Fig. 1A, top panel)

Spatiotemporal source distribution was calculated by using an MNE (Hämäläinen and Ilmoniemi, 1994). MNE calculates the time course of source distribution constrained to the cortex by deploying unit dipoles on the numerous cortical patches, and creates dynamic activation maps of epileptic spikes (Tanaka et al., 2010). Theoretically, MNE represents all magnetic fields by assuming that the sources are distributed on the cortical surface, and

previous studies demonstrated an excellent concordance between the simulated and calculated sources with this model (Hämäläinen and Ilmoniemi, 1994, Silva et al. 2004). A realistic head model, such as the boundary elemental method, is frequently used for obtaining precise cortical surface to constrain the activation, providing accurate source localization in simulation studies (Silva et al., 2004, Soufflet and Boeijinga, 2005) and in patients with epileptic spikes (Shiraishi et al., 2005, Tanaka et al., 2009, 2010).

In the analysis, we obtained a single-layer head model from MPRAGE by using a boundary elemental method (Hämäläinen and Sarvas, 1989). The inverse solution was calculated from the forward solution by using a noise covariance matrix obtained from the empty room data, modeling the signal pattern generated by a unit dipole distributed on the MRI-derived cortical surface (Dale et al., 1999, Fischl et al., 1999). The activation at each vertex of the cortical surface was mapped at each time point of the averaged spikes by using this inverse solution. Thus, MNE maps represent the cortical activation derived by the spikes both spatially and temporally with distributed sources. Methodological details of spike analysis and MNE are described in the previous studies (Tanaka et al., 2009, Tanaka et al., 2010).

Region of interest (ROI) (Fig 1A, second panel)

We selected regions of interest (ROIs) based on the cortical parcellation of the ipsilateral hemisphere (Dale et al., 1999, Fischl et al., 1999) as follows; 1) Temporoparietal: Supramarginal gyrus and inferior parietal cortex. 2) Inferior frontal: opercular, triangular and orbital part of the inferior frontal gyrus. 3) Mesial temporal: entorhinal cortex and parahippocampal gyrus. 4) Anterior temporal: Anterior part of the lateral temporal cortex, consisting of superior, middle, inferior, transverse temporal gyri and bank of the superior temporal sulcus. 5) Posterior temporal: Posterior part of the lateral temporal cortex described above. 4) and 5) were divided by a plane through the posterior commissure, oriented perpendicularly to the long axis of the temporal lobe.

Determination of the leading and propagated areas of spike

For each patient, we extracted activation values of all unit dipoles in the ROIs within the whole time course of the averaged spike (Fig. 1A, third and fourth panel, Fig. 1B). The maximum activation was obtained at the peak latency (0ms). We set a threshold as half of the maximum activation and obtained unit dipoles with activation reaching the threshold at the earliest latency. The ROIs including these unit dipoles were considered the leading areas. ROIs including unit dipoles with activation over the threshold at 0ms were considered to be propagated areas (Fig. 1B).

Statistics

Binary variables were created from leading areas (restricted to the anterior and mesial temporal ROIs or not) and from propagation areas (involving the temporoparietal ROI or not), and for surgical outcome (Class I or not). Fisher's exact test was used for significance testing. Sensitivity, specificity, positive and negative predictive values were also obtained for both factors.

RESULTS

Table 2 gives the results of leading areas and surgical outcomes. In total, 16 out of 37 patients showed leading areas restricted to the anterior/mesial temporal ROIs, and the surgical outcome of all 16 patients (100%) was classified as Class I. Other types of leading area were seen in 21 patients, and only 13 (62%) had Class I outcome. The anterior/mesial leading area was significantly correlated with Class I outcome ($p < 0.05$). In the mesial group, a leading area restricted to the anterior/mesial temporal ROIs was seen in 15 out of 27

patients, all of which (100%) had Class I outcome. For 12 patients with other leading areas, only 7 (58%) had Class I outcome. Again, the anterior/mesial leading area was significantly correlated with Class I outcome ($p < 0.05$). The sensitivity, specificity, positive and negative predictive values were 68%, 100%, 100%, 42% in the mesial group and 55%, 100%, 100%, 38% in the whole patient group. In the non-mesial group, no correlation between leading area and surgical outcome was found. Temporoparietal propagation was seen in 18 out of 37 patients, 11 (61%) of which had a Class I outcome (Table 3). For 19 patients without temporoparietal propagation, all but one (95%) had a class I outcome. The temporoparietal propagation was significantly correlated with Class II-IV outcome ($p < 0.05$). In the mesial group, 10 out of 27 showed temporoparietal propagation, only 6 of which (60%) had class I outcome, compared to 16 out of 17 patients (94%) without propagation. Again, the temporoparietal propagation was significantly correlated with Class II-IV outcome ($p < 0.05$). The sensitivity, specificity, positive and negative predictive values were 80%, 73%, 40%, 94% in the mesial group and 88%, 62%, 39%, 95% in the whole patient group. In the non-mesial group no relation was found between presence of propagation and outcome. Source distribution maps of typical cases in the mesial and non-mesial groups are shown on Fig. 2 and Fig. 3, respectively. Detailed results of each patient are shown on Table 1.

DISCUSSION

In this study, we examined the propagation patterns of interictal MEG spikes with MNE and correlated it with surgical outcome. All patients with a leading area restricted to the anterior/mesial temporal lobe had a Class I surgical outcome, and the presence of such a restricted leading area was significantly correlated with Class I outcome in the whole patient and mesial group. Conversely, seven out of eight patients with Class II-IV outcome showed temporoparietal propagation, and a significant correlation was found between temporoparietal propagation and poor outcomes. These results demonstrate the clinical utility of examining origin and propagation of epileptiform activity on MEG in the presurgical evaluation of epilepsy. Previous studies using MNE (Tanaka et al., 2009) and dynamic statistical parametric mapping (Shiraishi et al., 2005) investigated spike propagation by visual inspection of distributed source maps. This visual inspection is qualitative and subjective, and the findings may vary since the maps of cortical activation are dependent on arbitrary thresholding. Our procedure evaluates spike propagation more quantitatively and objectively than visual inspection of the source distribution maps.

This study may also provide a quantitative basis for visual inspection of spatiotemporal source maps. We found two typical patterns in the mesial group exemplified by Patient 6 and 21 (Fig. 2). In Patient 6, source distribution maps showed anterior temporal activation in the early latency of spike without temporoparietal propagation at peak. This pattern (Type I map) corresponds to the presence of restricted leading area and the absence of temporoparietal ROI propagation determined by the quantitative ROI analysis, which is correlated with a favorable surgical outcome. In Patient 21, the inferior frontal lobe, as well as the anterior/mesial temporal cortex, is activated in the early latency and the peak activation involves temporoparietal area. These findings of spatiotemporal source maps (Type II map) are quantified and confirmed by the ROI analysis, which shows a correlation with a poor surgical outcome. Thus, visual inspection of source distribution maps are likely informative in clinical setting even without formal quantification.

In the non-mesial group, neither presence of an anterior/mesial leading area nor temporoparietal propagation was statistically correlated with surgical outcome. There are several possibilities explaining this negative finding, including the small number of patients and the heterogeneity of both etiology (lesion types) and spike localization in this group. Indeed, in non-mesial TLE, the relation between spike localization and surgical outcome is

still debated. Hennessy et al. (2001) reported that interictal EEG spikes restricted to the ipsilateral temporal lobe were not predictive of surgical outcome in patients with lesional temporal lobe epilepsy, while Janszky et al. (2006) found that localized ictal EEG discharges were associated with favorable outcome in such patients. Heers et al. (2010) studied several cases with lesional lateral temporal lobe epilepsy, showing single dipole localization of MEG spikes close to the lesion was associated with a favorable surgical outcome. In our cases, both Patient 30 and 32 showed widespread activation at the peak of spikes. For early latency, Patient 30 showed activation only in the posterior temporal ROI and had Class I outcome. In Patient 32, early activation was seen in the posterior temporal and temporoparietal ROIs and the outcome was Class IV (Fig. 3). A restricted leading area might be more important for a favorable surgical outcome than a specific location with propagation. Further investigation in a larger patient group may clarify the different impact of spike propagation on surgical outcome compared with mesial group.

In this study we visually examined the MEG data and selected the most frequent type of spikes in each patient. We consider these spikes demonstrate the representative propagation patterns that may have a clinical significance in each patient. The spike selection and analysis were blinded to the surgical outcomes for avoiding selection bias. However, analyzing spikes with a single morphology may result in missing information of spikes with different morphology. Indeed, we found a few spikes which showed widespread cortical activation including the temporoparietal ROI (Supplementary Fig. 1) in Patient 24, who was presented to have Class III outcome without temporoparietal propagation in Table 1. These rare spikes were more predictable for surgical outcome than the frequent spikes in this case. Here, inpatient variation in spike morphology may be informative for clarifying the correlation between spike propagation and surgical outcome in detail. On the other hand, clarification of all types of spike morphology by visual inspection requires a great effort and is time-consuming, and automated spike detection will be useful for revealing the variation by analyzing numerous spikes. Nonclercq et al. (2012) have reported that cluster-based spike detection demonstrates such variation consistently with visual inspection of spikes on EEG. Application of such methods in a patient cohort will lead further investigation and provide more detailed information regarding spike propagation patterns. There are several limitations in this study that needs further investigation. First, we used a threshold determined by half of the maximum peak of spikes for deciding the leading/propagated areas, however, the best procedure for obtaining such a threshold has not been well established (Tanaka et al., 2009, Tanaka et al., 2013). Investigating the statistical distribution of spike activation in detail will be helpful for resolving this issue. Second, we did not investigate the relationship between spatiotemporal source distribution and resection volume since postsurgical MRI was not available in this study. Previous MEG studies have demonstrated that resection of the cortex with single dipole distribution is associated with a favorable surgical outcome in both temporal and extratemporal lobe epilepsy (Genow et al., 2004, Fischer et al., 2005). Third, the retrospective design of the study does not allow for controlling all possible confounders. There are several prognostic factors of temporal lobe surgery, including clinical profiles and EEG findings (Radhakrishnan et al., 1998, Wiebe et al., 2001), and the present study does not clarify the relationship of these factors and spike propagation. On the other hand, this study provides sufficient encouragement for a prospective data collection effort using better-controlled, larger cohorts.

In conclusion, spatiotemporal maps of interictal spike propagation provide prognostic information in the presurgical evaluation of epilepsy patients. Specifically, this study demonstrates that spike propagation patterns represented by spatiotemporal source distribution of MEG spikes may predict surgical outcomes in patients with temporal lobe epilepsy.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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ABBREVIATIONS

MEG	magnetoencephalography
IEEG	intracranial electroencephalography
MPRAGE	magnetization-prepared rapid acquisition gradient echo
MNE	minimum norm estimate
ROI	region of interest

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Highlights

Magnetoencephalographic spike propagation is informative in presurgical evaluation.

Anterior/mesial temporal loading areas are correlated with favorable outcomes.

Temporoparietal propagation of spikes is correlated with unfavorable outcomes.

Figure 1. Schematic representation of analyzing spike propagation

(A) Top panel: Spatiotemporal source distribution of the averaged spike obtained by minimum norm estimate, projected on the lateral and mesial cortical surface of the right hemisphere. Second panel: Region of interests (ROIs) used for investigating spike propagation. ROIs are obtained from cortical parcellation. Third panel: Unit dipoles distributed on the cortical surface. Spatiotemporal source maps (top panel) consist of the estimated activation in each dipole. Fourth panel: Combined image of top, second and third panel. The activation values at each unit dipole are extracted.

(B) The time course of the activation at each unit dipole is represented. The figure shows examples of anterior temporal, inferior frontal and temporoparietal ROIs, including AT1, IF1 and TP1 shown on (A). When the maximum peak ($=0\text{ms}$) activation within all ROIs appears at a unit dipole AT1 (shown as M), the threshold (Th) is determined by $(1/2) \times M$. The spike activation reaches Th at the latency $t(AT1)$. The latency “ t ” is obtained at each unit dipole of all ROIs in the same manner, such as $t(IF1)$. Note that Th is a constant applied for all unit dipoles. In the figure, $t(AT1)$ is earlier than $t(IF1)$. ROIs containing the earliest “ t ” are considered leading areas of the spike. The peak activation at IF1 is larger than Th and ROIs including such unit dipoles are considered propagated areas. In contrast, the activation at TP1 does not reach Th .

Figure 2.

Spatiotemporal source maps of the averaged spike in early and peak (=0ms) latency obtained from representative patients of mesial group. (A) 50-year-old female (Patient 6). Early activation appears at -14ms in the anterior temporal area. Peak activation is seen in small portions of the mesial and posterior temporal areas as well as the anterior temporal area. The patient has been seizure free after a right temporal lobectomy (Engel's Class I). (B) 18-year-old male (Patient 21). Early inferior frontal activation appears at -12ms as well as the anterior/mesial temporal activation. Peak activation involves posterior temporal and temporoparietal areas in addition to the early activation areas. The patient underwent a right temporal lobectomy but still has occasional disabling seizures (Class II).

Figure 3.

Spatiotemporal source maps of the averaged spike in early and peak (=0ms) latency obtained from representative patients of non-mesial group. (A) 32-year-old male (Patient 30). Early activation at -28ms is seen in the right mid-temporal area. Peak activation extends to the whole temporal region and temporoparietal area. The patient underwent a right temporal lobectomy and only has simple partial seizures with a transient strange sensation in the head (Class I). (B) 22-year-old female (Patient 32). Early activation at -8ms involves right mid-temporal and temporoparietal areas. Peak activation is seen in the same areas with larger strength. The patient underwent a subpial topectomy with transection but still has frequent seizures (Class IV).

Table 1

Patient profile and results

No	Age/Sex	Diagnosis	Interictal EEG	MRI	Leading area(s)	Propagated area(s)	Surgery	Outcome
Mesial group (n=27)								
1	M/14	L	LT	L hippocampal atrophy + previous L posterior temporal glioma resection	AT	MT, AT	L TL+subpial transection	I
2	F/43	L	LT	normal	AT	MT, AT	L TL	I
3	M/33	L	LF, LT	L hippocampal atrophy	AT	IF, AT, PT	L TL	I
4	M/11	R	LT<RT	normal	TP, PT	TP, IF, MT, AT, PT	R TL	IV
5	F/68	L	LT	L hippocampal atrophy	AT	MT, AT, PT	L TL	I
6	F/50	R	RT	R hippocampal atrophy	AT	MT, AT, PT	R TL	I
7	M/13	L	LT, LP	normal	PT	TP, IF, MT, AT, PT	L TL	I
8	M/15	L	LT	normal	AT	AT	L TL	I
9	F/15	R	LT<RT	R hippocampal atrophy	AT	IF, MT, AT, PT	R TL	I
10	M/50	L	LT	L hippocampal atrophy	AT	IF, MT, AT, PT	L TL	I
11	F/35	R	RT	normal	AT	TP, IF, MT, AT, PT	R TL	I
12	F/20	R	RF, RT	normal	IF, PT	TP, IF, MT, AT, PT	R TL	I
13	F/16	L	LF, LT, LP	L hippocampal atrophy	MT	MT, AT, PT	L TL	I
14	F/9	L	LF, LT	normal	IF, MT, AT	IF, MT, AT	L TL	I
15	M/9	R	RT	R hippocampal atrophy	PT	TP, IF, MT, AT, PT	R TL	I
16	F/16	L	LF, LT	L hippocampal atrophy	AT	IF, MT, AT	L TL	I
17	F/15	R	RT	normal	PT	TP, IF, MT, AT, PT	R TL	II
18	M/21	L	LT	normal	AT	IF, MT, AT, PT	L TL	I
19	M/38	R	LT	R hippocampal atrophy	MT, AT, PT	TP, MT, AT, PT	R TL	I
20	F/28	R	RF, RT	normal	MT	MT, AT, PT	R TL	I
21	M/18	R	RT	normal	IF, MT, AT	TP, IF, MT, AT, PT	R TL	II
22	M/59	L	LT	L hippocampal atrophy	MT	MT, AT, PT	L TL	I
23	F/17	L	LF, LT, LP	normal	MT, PT	IF, MT, AT, PT	L TL	I
24	M/15	L	LT	normal	AT, PT	IF, MT, AT, PT	L TL	III
25	M/23	R	LT	R hippocampal atrophy	TP	TP, IF, MT, AT, PT	R TL	I
26	F/18	R	LT, LP	R mesial temporal cortical malformation, periventricular heterotopia	AT	AT, MT	R temporal lesionectomy	I

No	Age/Sex	Diagnosis	Interictal EEG	MRI	Leading area(s)	Propagated area(s)	Surgery	Outcome
27	F/28	R	RF, RT	normal	Non-mesial group (n=11)	TP, IF, AT, PT	R TL	IV
28	M/13	L	LF, LT, LP	L anterior temporal arachnoid cyst	AT	IF, MT, AT	L TL	I
29	M/21	L	LT	normal	PT	TP, MT, AT, PT	L TL	I
30	M/32	R	RT	normal	PT	TP, MT, AT, PT	R TL	I
31	M/27	R	RT	R inferior temporal cortical dysplasia	PT	TP, MT, AT, PT	R TL	I
32	F/22	R	LT<RT	diffuse cortical thickening	TP, AT, PT	TP, AT, PT	R subpial topectomy+transection	IV
33	F/49	R	LT<RT	normal	PT	TP, MT, AT, PT	R TL	I
34	F/9	L	LT	L inferior temporal signal abnormality	TP, AT, PT	TP, IF, MT, AT, PT	L inferior temporal+hippocampectomy	III
35	F/17	L	LT, LP	normal	TP	TP, AT	L lateral temporal resection	III
36	F/18	L	LT	L superior temporal tumor	IF, MT, AT, PT	TP, IF, MT, AT, PT	tumor resection	I
37	M/60	L	LT	L temporoparietal cystic lesion	MT, PT	MT, AT, PT	L temporal lesionectomy	I

M: male, F: female, L: left TLE, R: right TLE, LT(RT): left(right) temporal, LF(RF): left(right) frontal, LP: left(right) parietal, LT<RT: more frequent left than right temporal spikes (LT>RT: vice versa), TP: temporoparietal, IF: inferior frontal, MT: mesial temporal, AT: anterior temporal, PT: posterior temporal, TL: temporal lobectomy, Outcome: represented by Engel's Classification.

Table 2

Leading area and surgical outcomes

	Class I	Class II-IV
Mesial group (n=27)		
AT/MT/AT+MT	15	0
including other areas	7	5
Non-mesial group (n=11)		
AT/MT/AT+MT	1	0
including other areas	6	3

MT: mesial temporal ROI, AT: anterior temporal ROI

Table 3

Propagated areas and surgical outcomes

	Class I	Class II-IV
Mesial group (n=27)		
TP(+)	6	4
TP(-)	16	1
Non-mesial group (n=11)		
TP(+)	5	3
TP(-)	2	0

TP(+): presence of temporoparietal ROI propagation, TP(-): absence of temporoparietal ROI propagation