



# Can intraoperative electrocorticography patterns predict surgical outcome in patients with temporal lobe epilepsy secondary to unilateral mesial temporal sclerosis?

Pedro A.L. Oliveira<sup>a</sup>, Eliana Garzon<sup>a</sup>, Luís O.S.F. Caboclo<sup>a</sup>,  
Patrícia S. Sousa<sup>a</sup>, Henrique Carrete Jr.<sup>b</sup>, Ricardo S. Centeno<sup>c</sup>,  
José M.P. Costa Jr.<sup>d</sup>, Hélio R. Machado<sup>e</sup>, Elza M.T. Yacubian<sup>a</sup>,  
Marino M. Bianchin<sup>f</sup>, Américo C. Sakamoto<sup>a,f,\*</sup>

<sup>a</sup> Department of Neurology and Neurosurgery, Division of Neurology, Federal University of São Paulo, São Paulo, Brazil

<sup>b</sup> Department of Radiology, Federal University of São Paulo, São Paulo, Brazil

<sup>c</sup> Department of Neurology and Neurosurgery, Division of Neurosurgery, Federal University of São Paulo, São Paulo, Brazil

<sup>d</sup> Department of Anesthesiology, Federal University of São Paulo, São Paulo, Brazil

<sup>e</sup> Department of Surgery, Division of Neurosurgery, University of São Paulo, Ribeirão Preto Medical School, São Paulo, Brazil

<sup>f</sup> Department of Neurology, Psychiatry and Clinical Psychology, University of São Paulo, Ribeirão Preto Medical School, São Paulo, Brazil

Received 4 February 2006; received in revised form 15 June 2006; accepted 28 June 2006

## KEYWORDS

Electrocorticography;  
Temporal lobe  
epilepsy;  
Hippocampus sclerosis

## Summary

*Introduction:* Intraoperative electrocorticography (ECoG) can be performed in cases of temporal lobe epilepsy due to hippocampal sclerosis (TLE-HS). However, its significance and correlation with surgical outcome are still controversial.

*Objectives:* To analyze the electrophysiological characteristics of temporal lobe structures during ECoG of patients with TLE-HS, with emphasis on the comparison between pre- and post-resection recordings and surgical outcome.

*Patients and methods:* Seventeen patients with refractory TLE-HS submitted to corticoamigdalohipocampectomy were included in the study. Clinical variables included age at the onset, duration of epilepsy and seizure outcome. The post-operative follow-up ranged from 24 to 36 months. According to outcome subjects

\* Corresponding author at: Campus Universitário, Ribeirão Preto, SP 14048-900, Brazil. Tel.: +55 16 3911 1902; fax: +55 16 3911 1903. E-mail address: sakamoto@fmrp.usp.br (A.C. Sakamoto).

were divided in two subgroups: (A) individuals free of seizures (Engel 1A), and (B) individuals not-free of seizures (Engel 1B–IV). Four patterns of ECoG findings were identified: isolated discharges; high frequency spikes (HFS); continuous discharges; combination of isolated discharges and HFS. According to predominant topography ECoG was classified as mediobasal, lateral (or neocortical), mediobasal and lateral. *Results:* The progressive removal of the temporal pole and the hippocampus was associated with significant decrease of neocortical spikes. No correlation between clinical variables and seizure outcome was observed. Patients who only had isolated spikes on intraoperative ECoG presented a statistical trend for excellent surgical control. Patients who presented temporal pole blurring on MRI also had better post-surgical seizure outcome.

*Conclusions:* This study showed that out of diverse clinical and laboratory variables, only isolated discharges on intraoperative ECoG and temporal pole blurring on MRI predicted excellent post-surgical seizure outcome. However, other studies with larger number of patients are still necessary to confirm these findings.

© 2006 British Epilepsy Association. Published by Elsevier Ltd. All rights reserved.

## Introduction

Intraoperative electrocorticography (ECoG) can be performed in cases of temporal lobe epilepsy due to hippocampal sclerosis (TLE-HS), in order to determine the extent of the neocortical resection.<sup>1</sup> However, the significance of the origin and propagation of the discharges, of the presence or absence of residual spikes, and the correlation of these findings with surgical outcome is still controversial and remains under debate in the literature.<sup>2</sup>

In previous reports, authors observed that approximately 70% of the patients with no post-resection spikes reach seizure-free status,<sup>3,4</sup> while persistence of at least 50% of the pre-resection spikes predicts poor seizure outcome.<sup>5</sup> However, many others have failed to establish correlations between spike quantification and seizure outcome.<sup>6–10</sup>

Despite its classical use in epilepsy surgery, most centers have abandoned intraoperative ECoG in cases of TLE-HS. However, ECoG analysis nowadays should probably include parameters other than post-resection spike quantification, in order to permit a more complete study of the pathophysiology of TLE, and in the search of patterns of prognostic value that could eventually influence the surgical technique.

There are few studies in the literature defining ECoG patterns in patients with TLE-HS.<sup>11</sup> More recently, interest in the neurophysiological study of temporal lobe structures has renewed, after the characterization of the so-called high frequency oscillations (HFOs).<sup>12–18</sup> Such HFOs appear to arise in close proximity to sites of seizure onset, and therefore, might be a significant indicator of the location of the epileptic focus. Activities over 40 Hz have been recorded in the vicinity of the seizure focus and localized in time close to seizure onset in various pathologies.<sup>19</sup> Spontaneous runs above

100 Hz were additionally observed in the hippocampus and entorhinal cortex of epileptic patients.<sup>20,21</sup>

The objective of the present study was to analyze pre- and post-resection electrophysiological characteristics of temporal lobe structures during intraoperative ECoG of patients with TLE-HS, with emphasis on the analysis of patterns of ECoG and on the assessment of their implication for post-surgical seizure outcome.

## Patients and methods

A series of 17 surgically treated patients with refractory TLE secondary to unilateral HS was included in the study. All individuals were submitted to comprehensive presurgical assessment, including 1.5-T MRI, video-EEG monitoring with ictal recordings, neuropsychological tests, and psychiatric and psychosocial evaluations. The surgical technique consisted of standard corticoamygdalohippocampectomy, with resection of 2.5–3.5 cm of the temporal cortex measured from the pole in cases of the dominant hemisphere, and 4.5–5.0 cm in cases of the non-dominant hemisphere.

## Anesthesia

All procedures were performed under general anesthesia. During the ECoG isoflurane concentrations were reduced to 0.2 and 0.3% within an interval of 15–20 min before acquisition of the ECoG recording and the nitrous oxide (N<sub>2</sub>O) levels being maintained at 50–60%.

## Clinical data

Clinical variables included age at the onset of epilepsy, duration of epilepsy, and seizure outcome.

The post-operative follow-up ranged from 24 to 36 months. For seizure prognosis assessment subjects were divided in two groups according to Engel classification<sup>22</sup>: (A) individuals free of seizures (Engel class 1A); (B) patients not free of seizures (Engel classes 1B–IV).

### Neuroimaging data

Structural neuroimaging data included visual analysis of the temporal lobe, measurement and comparison of hippocampus volumes, and assessment of temporal pole signal abnormalities, including loss of gray-white matter definition and increased T2 and FLAIR signals within the white matter. The temporal pole blurring was classified as diffuse, when involved the lateral and mesial structures (including parahippocampal gyri), or mesial, when affected exclusively the mesial parts of the anterior temporal lobe.

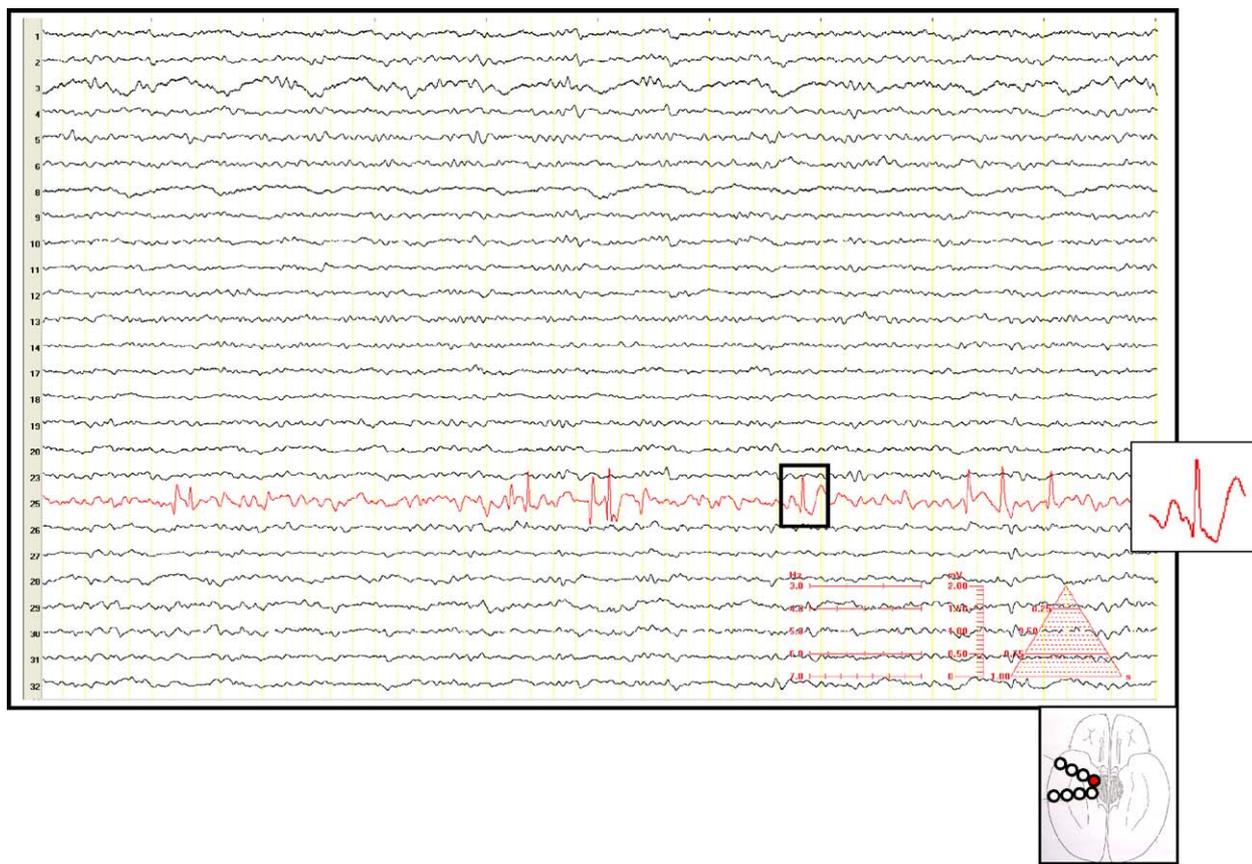
### ECoG recordings

Recordings were obtained through platinum Ad-Tech subdural strips in a 32 channels Nihon-Kohden digital EEG, Neurofax EEG-1000, sampling rate of 1000 Hz.

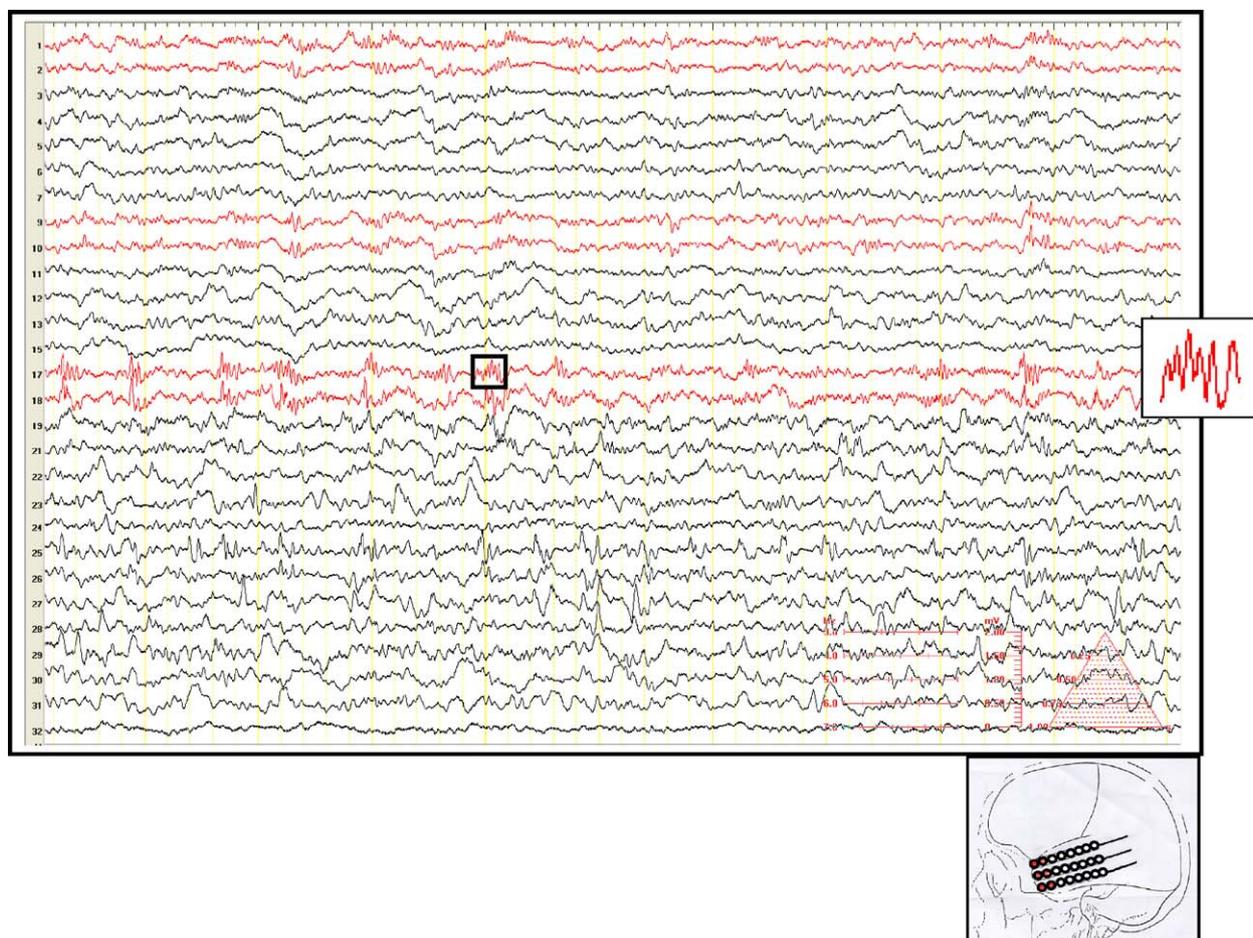
EEG data were analyzed on a low cut filter of 1 Hz and high cut filter of 300 Hz. For the analysis we used referential montage with a reference placed in the duramater. EEG acquisition was performed in three stages, each lasting 10 min: (a) phase I—pre-resection: three 8-contacts strips were placed over the surface of the lateral temporal lobe covering the superior, middle and inferior temporal gyri, respectively, plus two 4-contacts strips placed on the mediobasal region; (b) phase II—post-resection of the temporal pole: three 8-contacts strips placed on the margins of the resection, and one 4-contacts strip placed over the longitudinal surface of the hippocampus; (c) phase III—post-resection of mesial structures: three 8-contacts strips placed at the margins of the resection. All EEG records were visually and independently analyzed by two observers with expertise in presurgical evaluation. In case of disagreement between the two observers a consensual meeting was held.

### ECoG patterns

ECoG patterns were classified according to the morphology of the discharges and their topography. Four



**Figure 1** ECoG type I (isolated spikes). Channels 1–8: superior temporal gyri; 9–14: middle temporal gyrus; 17–22: inferior temporal gyrus; 25–28: anterior basal temporal; 29–32: posterior basal temporal.



**Figure 2** ECoG type II: high frequency spikes (HFS). Channels 1–7: superior temporal gyrus; 9–15: middle temporal gyrus; 17–24: inferior temporal gyrus; 25–28: anterior basal temporal; 29–32: posterior basal temporal.

patterns were identified: (a) type I: isolated discharges—characterized by isolated spikes or sharp waves (Fig. 1); (b) type II: high frequency spikes (HFS)—characterized by trains of high frequency spikes defined as above 30 Hz (Fig. 2); (c) type III: continuous discharges—characterized by repetitive discharges lasting longer than 10 s (Fig. 3); (d) type IV: combination of isolated discharges and high frequency spikes in the same recording. According to the predominant topography (predominance of more than 70%) these patterns were further subdivided into: (a) type A: mediobasal; (b) type B: lateral or neocortical; (c) type C: mediobasal and lateral. Quantification of discharges was also performed and the records were accordingly classified as very frequent ( $>20/\text{min}$ ), frequent (5–20/min) or rare ( $<5/\text{min}$ ).

### Statistical analysis

We studied numerical variables with independent-samples *T*-test, using Levene's robust tests for examining the homogeneity of the samples and these

results are presented as mean (S.D.). For categorical variables, we applied Fisher's exact test. These analyses were performed using SPSS 10.0 for Windows (SPSS Inc., USA). A  $\chi^2$ -test for trend was applied for assessment of a possible trend for reduction of neocortical spikes after progressive removal of the temporal pole and of the hippocampus. This analysis was performed using GraphPad InStat (GraphPad Software Inc., San Diego, USA). Values were considered statistically significant when  $p < 0.05$ .

## Results

### Clinical and neuroimaging data

Seventeen patients (5 men, 12 women), ranging from 29 to 59 years (average 39.8 years, median 37 years) were included. Five patients had right HS and 12 had left HS. The main clinical variables are shown in Table 1, including age, sex, age at epilepsy onset, duration of epilepsy, initial precipitant event, MRI findings and post-surgical seizure



**Figure 3** ECoG type III: continuous discharges. Channels 1–7: superior temporal gyrus; 10–15: middle temporal gyrus; 17–23: inferior temporal gyrus; 25–28: anterior basal temporal; 29–32: posterior basal temporal.

outcome. Overall 12 individuals were seizure-free of disabling seizures (Engel 1A–1B) and seven individuals were completely seizure-free (Engel 1A).

### ECoG patterns

The main ECoG findings are shown in [Table 2](#). The discharges on pre-resection ECoG showed the following distribution: type I, nine patients; type II, three patients; type III, two patients; type IV, three patients. HFS ranged from 80 to 104 Hz and were observed in six patients. Regarding the topographic distribution of type I pattern, 4/9 patients had type A (mesial) discharges, 3/9 had type B (lateral or neocortical) and 2/9 mesial and lateral distribution. Regarding types II–IV, only types B and C distributions were seen ([Table 2](#)). Regarding the quantification of the pre-resection discharges, in type I we had 4/9 patients with frequent discharges, 3/9 with very frequent discharges, and 2/9 with rare discharges. In type II group, 2/3 had rare discharges, and 1/3 had frequent discharges. In the subgroups types III and IV, all patients had very frequent discharges.

The ECoG findings after the resection of the temporal pole are also shown in [Table 2](#). Remarkably, five out of the seven patients that did not have mesial discharges on pre-resection ECoG (type B) presented hippocampal discharges after the initial resection: type I, two patients; type II, two patients; type III, one patient. However, this difference was not significant ( $p = 0.15$ ). Regarding the morphology of the discharges, four out of these five patients showed hippocampal discharges similar to pre-resection neocortical discharges, while one patient that previously had HFS (type II) over the neocortex showed different pattern of hippocampal discharges, a combination of isolated spikes and HFS (type IV). Between the 10 patients that previously presented mesial discharges on pre-resection ECoG (types A and C), two patients had increase in the frequency of the discharges while one patient, contrarily, showed disappearance of the discharges. Regarding the ECoG findings at the margins of the neocortical resection, of the 13 patients that had neocortical spikes on pre-resection ECoG (types B and C), seven showed disappearance of the spikes after

**Table 1** Clinical and neuroimaging data

Patient	Age (years)	Sex	Epilepsy age of onset	Length of illness (years)	Initial event	Type of initial event	Side of HS	Visual analysis of HS	Rate of HA (%)	Blurring	Localization of anterior temporal lobe with blurring	Outcome
1	53	F	14	39	Yes	Afebril Seizure	L	Severe	61–80	Yes	Diffuse	Engel ID
2	33	M	8	25	Yes	Status	L	Moderate	Not performed	No	–	Engel IIIA
3	50	F	1	49	No	–	L	Moderate	61–80	Yes	Mesial	Engel IB
4	40	M	15	25	No	–	L	Moderate	61–80	No	–	Engel IIB
5	29	F	15	14	Yes	Febril seizure	L	Moderate	10–30	Yes	Mesial	Engel IA
6	29	F	18	11	Yes	Febril seizure	L	Discrete	31–60	No	–	Engel IB
7	33	M	12	21	No	–	R	Severe	61–80	Yes	Mesial	Engel IA
8	37	F	19	18	Yes	Status	L	Moderate	10–30	Yes	Mesial	Engel IB
9	48	M	18	30	No	–	R	Severe	31–60	No	–	Engel IB
10	29	F	10	19	No	–	L	Severe	61–80	Yes	Mesial	Engel IA
11	50	F	32	18	No	–	R	Discrete	10–30	No	–	Engel IIB
12	37	F	2	35	Yes	Febril seizure	R	Discrete	31–60	Yes	Diffuse	Engel IA
13	40	F	25	15	No	–	L	Discrete	31–60	Yes	Mesial	Engel IA
14	33	F	9	24	Yes	Febril seizure	L	Moderate	31–60	Yes	Mesial	Engel IA
15	34	F	12	22	No	–	L	Moderate	31–60	Yes	Diffuse	Engel IA
16	59	F	2	57	Yes	Febril seizure	L	Moderate	Not performed	Yes	Diffuse	Engel IIB
17	43	M	8	35	No	–	R	Severe	31–60	No	–	Engel IIIA

F: female; M: male; L: left; R: right; HS: hippocampus sclerosis; HA: hippocampus asymmetry.

**Table 2** ECoG findings ranked according to surgical outcome

Patient	Phase I: pre-resection ECoG			Phase II: post-resection of temporal pole ECoG				Phase III: post-resection of hippocampus and temporal pole ECoG				Outcome	
	ECoG pattern	Topography of ECoG pattern	Frequency of discharges	ECoG pattern of HP	Frequency of discharges in HP	Margin of temporal lobe	Frequency of discharges at margin	ECoG pattern at temporal margin	Margin of temporal lobe	Localization of discharges (relationship to margin)	Localization of discharges		Frequency of discharges
5	I	B	R	—	—	—	—	—	—	—	—	—	Engel IA
7	I	B	F	I	R	—	—	—	—	—	—	—	Engel IA
13	I	A	R	I	VF	—	—	—	—	—	—	—	Engel IA
14	I	C	VF	I	R	+	R	—	—	—	—	—	Engel IA
15	I	B	F	I	VF	—	—	—	—	—	—	—	Engel IA
12	II	B	F	IV	VF	—	—	—	—	—	—	—	Engel IA
10	III	B	VF	—	—	+	R	—	—	—	—	—	Engel IA
9	I	A	F	I	F	—	—	IV	+	3 cm	GTM E GTS	R	Engel IB
3	I	C	VF	I	VF	+	VF	IV	+	2 cm	GTI	R	Engel IB
8	II	B	R	II	R	—	—	—	—	—	—	—	Engel IB
6	IV	C	VF	IV	F	—	—	—	—	—	—	—	Engel IB
1	IV	C	VF	IV	VF	+	VF	I	+	1–2 cm	GTM	F	Engel ID
16	I	A	F	I	—	—	—	—	—	—	—	—	Engel IIB
4	II	C	R	II	F	+	R	—	—	—	—	—	Engel IIB
11	III	B	VF	III	F	—	—	—	—	—	—	—	Engel IIB
17	I	A	VF	I	F	—	—	—	—	—	—	—	Engel IIIA
2	IV	C	VF	IV	VF	+	F	IV	+	1 cm	GTI	F	Engel IIIA

ECoG pattern: type I, isolated discharges; type II, high frequency spikes; type III, continuous discharges; type IV, combination of isolated discharges and high frequency spikes. Topography: (A) mediobasal (predominance of more than 50%); (B) lateral predominance of more than 50%; (C) mediobasal and lateral. Frequency of discharges: VF, very frequent (>20/min); F, frequent (5–20/min); R, rare (<5/min). (+) Presence of discharges; (–) absence of discharges; MTG, middle temporal gyrus; STG, superior temporal gyrus; ITG, inferior temporal gyrus.

the initial resection, a not statistically significant difference ( $p = 0.24$ ). Of the remaining six patients, three patients continued to have the same pattern in the same frequency, and three patients continued to have the same pattern but in frequency smaller than before.

The main ECoG findings after the resection of the mesial structures are also exposed in Table 2. Only four patients continued to show discharges at the margins of the resection three of them presented discharges restricted to one gyrus, while one had more widespread discharges, overlying more than one gyrus. All residual discharges were localized within 3 cm of the margin of the resection. Of the four patients that showed residual discharges at the margin of the resection, three had type IV (two of them did not have this pattern on pre-resection recordings), and one had type I pattern. Regarding the frequency of the discharges, all patients had

reduction in their frequency after the resection when compared to pre-resection ECoG.

When analyzed together, the progressive removal of the temporal pole and after, of the hippocampus, caused a marked reduction of the neocortical spikes. Before surgery, 13 out of 17 patients (76.5%) showed neocortical discharges. After removal of the temporal pole, this number dropped to five patients (29.4%). After removal of the hippocampus, the number of patients that still had neocortical discharges was only four out of 17 (23.53%). A  $\chi^2$ -test for trend revealed that the progressive reduction of neocortical discharges had a significant trend ( $p = 0.0019$ ;  $\chi^2$  trend = 9.62).

In summary, the removal of the temporal pole favored the appearance of hippocampal discharges, although this difference was not statistically significant. On the other hand, the progressive removal of the temporal pole and after, of the mesial temporal

**Table 3** Variables according to absolute post-surgical seizure freedom (Engel 1A)

Variable	Group		
	Engel 1A	Engel 1B–IV	p-Value
Age at onset of epilepsy (S.D.)	12.1 (6.96)	13.5 (9.22)	0.75
Duration of epilepsy (S.D.)	21.4 (6.99)	30.7 (14.49)	0.14
IPI			
Yes/no	3/4	5/5	1.00
Gender			
Men/women	1/6	4/6	0.33
Side of surgery			
Left/right	5/2	7/3	1.00
Temporal lobe blurring on MRI			
Yes/no	7/0	4/6	0.035*
ECoG frequency of discharges			
Rare/frequent	4/3	2/8	0.16
Type of discharges			
I/other	5/2	4/6	0.33
II/other	1/6	2/8	1.00
III/other	1/16	1/9	1.00
IV/other	0/7	3/7	0.29
Presence of complex discharges in any of three ECoGs			
Isolated spikes (type-I)/complex patterns (types II–IV)	5/2	2/8	0.058**
Topographic distribution of discharges			
Mesio basal/others	1/6	3/7	0.60
Frequency of hippocampal discharges			
Rare/frequent	4/3	2/8	0.16
Margin discharges after temporal pole removal			
Yes/no	2/5	4/6	1.00
Margin discharges after hippocampus removal			
Yes/no	0/7	4/6	0.10

\* Significant.

\*\* Statistical trend.

structures, revealed a statistically significant trend favoring the reduction or disappearance of neocortical discharges.

### Correlation of clinical and imaging data with outcome

There was no correlation between clinical data and post-surgical seizure outcome. However, it is noticeable that in our series, patients presenting blurring of the temporal pole on MRI had better surgical outcome ( $p < 0.04$ ) (Table 3).

### Correlation of ECoG patterns with outcome

Of the four patients with residual spikes after the neocortical and mesial resections, only one remained with disabling seizures during the follow-up (patient number 2). Regarding the ECoG pattern, of the five patients that remained with seizures after surgery, three had types II–IV patterns, respectively, while only one had type I pattern. Regarding the topographic distribution of the discharges in these five patients that did not become seizure-free, two had mesial pre-resection discharges, two had mesial and lateral discharges, and one had exclusively neocortical spikes (Table 2). Interestingly, when considering recordings of all three phases of the study (Table 2), patients who had only isolated spikes on intraoperative ECoG presented a statistical trend for excellent surgical outcome (Engel 1A) when compared to those patients who had more complex patterns. This result might suggest that intraoperative ECoG predicts surgical outcome. However, given the small size of our sample we recognized that our results need further confirmation in larger studies.

## Discussion

In this small series, 71% of the patients became free of disabling seizures after surgery, a result consistent with recent series.<sup>23</sup> Clinical data, including age, duration of epilepsy, presence or absence of initial precipitating injury, clinical history, showed variable correlation with seizure outcome.<sup>24</sup> In our series, patients with temporal pole blurring on MRI had better surgical outcome, thus convergent with recent findings from Chabardes et al.<sup>25</sup>

As reported here, distinct electrophysiological patterns can be encountered on pre-resection ECoG of patients with mesial TLE-HS. Detailed analysis of these patterns is warranted once they might have implications in post-surgical seizure outcome. In the

present study, we performed detailed analysis of ECoG patterns, including morphology, topographic distribution, frequency of discharges, and recordings in pre-resection, partial resection and final resection phases. When globally considered all three recording phases, patients who presented more complex ECoG patterns seem to have worst post-surgical seizure outcome (Tables 2 and 3).

Type II pattern consisting of bursts of very fast activity above 80 Hz was initially described in animals,<sup>12,14</sup> and later also in the cerebral cortex of patients submitted to invasive recordings,<sup>13,16–18</sup> being mainly encountered in the hippocampus and entorhinal cortex of these individuals.

In the present study, activities with frequencies ranging from 80 to 104 Hz were observed in both, the lateral and medial temporal cortex. HFOs were previously characterized as ripples (80–200 Hz) and fast ripples (above 200 Hz). Previous animal studies have suggested that these oscillations derive from distinct sources, but this has not been clearly defined for humans.<sup>18</sup> Ripples apparently originate from inhibitory postsynaptic potentials of hippocampal pyramidal cells,<sup>17,26</sup> while fast ripples have been observed after the induction of epileptogenesis by injection of kainic acid,<sup>17</sup> thus suggesting that they are related to the process of epileptogenesis, possibly associated with the neuronal reorganization underlying epileptogenesis.<sup>27–29</sup> Intracellular recordings also suggest that HFOs might also play a role in the generation of focal epileptic seizures, especially those oscillations above 200 Hz.<sup>30</sup> These oscillations are usually mediated by mechanisms involving a vicious feedback loop in which very fast oscillations reflect synchronous action potentials. The synchronization of these action potentials in adjacent neurons is probably the result of electrical interactions, which were demonstrated in laboratory studies using cat models in the early 1980s.<sup>30</sup>

In our study due to methodological limitations, especially the narrower frequency band of our filter setting (0.1–300 Hz), and more importantly, to the fact that we only placed electrodes overlying the cortical surface, we were just able to record high frequency field potentials that we called HFS. Although it is tempting to consider HFS analogous to HFOs, this association has not been demonstrated in humans so far. Type III, often associated with malformation of cortical development which is usually a more diffuse lesion than HS,<sup>31</sup> was not a frequent pattern in this study, similarly to types II and IV. In our series, HFS were not correlated with post-surgical seizure prognosis (Table 3). Nevertheless, studies with larger number of patients might be necessary before excluding these patterns as predictors of seizure control after surgery.

Regarding the presence of discharges at the margins of the resection most previous studies have not demonstrated such correlation,<sup>32–34</sup> whereas, others found the absence of post-resection discharges to be predictive of a good outcome.<sup>3</sup> The differences in these results are probably due to variations in the methodology, such as the duration of tracing analysis which ranged from 2 to 10 min, and the anesthetic substances, whose influence on epileptiform activity is well-known. In our study, the presence of residual discharges at the border of the resection did not influence the prognosis.

Although not significant, the resection of the temporal pole apparently led to an increase in the hippocampal discharges, or provoked their appearance disclosure in patients who did not have them in the pre-resection ECoG. However, we need to consider technical differences in the recordings: in the pre-resection phase the activity was captured through subdural strips placed subtemporally, opening the possibility that this placement was not ideal, while the post-resection recordings were obtained with subdural strips placed under direct visualization of the mesial structures, including the hippocampus.

Also interestingly, the removal of the temporal pole and hippocampus reduced or abolished the epileptiform discharges at the border of the resection, contrarily to previous findings demonstrating that selective amigdalohippocampectomy increased neocortical spiking.<sup>35</sup> These observations suggest that the discharges observed in neocortical areas are dependent on the mesial temporal structures, a conclusion with good biological plausibility.

Besides morphology, we characterized the ECoG patterns according to their location and frequency. Few studies establishing ECoG patterns in this group of patients are available in the literature. Gómez-Utrero et al.,<sup>11</sup> in a prospective study involving 33 patients with refractory TLE submitted to surgery, proposed a classification of the findings based on the location of the interictal abnormalities, and encountered five patterns: type I (mesial), type II (mesial with neocortical propagation), type III (mesial and independent neocortical locations), type IV (neocortical with mesial propagation), and type V (neocortical). Types I and II were correlated with better surgical outcome, but cases of refractory TLE secondary to various etiologies were included, with no homogeneity regarding the pathology underlying the epileptic syndromes. In the present study, we were not able to find any correlation between ECoG topography of pre-resection discharges and surgical outcome. However, we recognize that this finding needs to be confirmed in further studies with larger number of individuals.

In respect to the structural imaging data, anterior temporal abnormalities previously described in the literature<sup>36</sup> were analyzed. We found these abnormalities in 11 patients (64%). In other studies it was found in 64–71% of individuals with TLE-HS.<sup>36,37</sup> The presence of discharges over the lateral temporal cortex was associated with increased incidence of anterior temporal abnormality (67%) when compared to the group with absence of these discharges (29%). However, the samples were heterogeneous, and this find also needs confirmation with larger number of patients and more homogeneous groups. Pathologic factors associated to these abnormalities still remain unidentified so that neurophysiology data, ECoG in particular, may contribute to clarifying their pathologic substrates. Interestingly, in our series, those patients who presented temporal pole blurring on MRI had better surgical outcome. However, we recognize that this finding needs to be confirmed in further studies specifically designed to evaluate this possibility.

In conclusion, it is possible to establish different ECoG patterns in refractory TLE-HS, on the basis of the dominant graphoelements and their topography. Based in our findings, we suggest that more complex patterns on intraoperative ECoG might predict worst surgical outcome in TLE-HS. However, the small series studied here is a limiting factor and further studies with larger number of patients might be necessary to confirm our findings.

## Acknowledgement

The financial support was provided by FAPESP.

## References

1. Engel Jr J, Ojemann GA. The next step. In: Engel Jr J, editor. *Surgical treatment of the epilepsies*. 2nd ed. New York: Raven Press; 1993. p. 319–29.
2. Alarcon G, Garcia Seoane J, Binnie CD, Martin Miguel MC, Juler J, Poplkey CE, et al. Origin and propagation of interictal discharges in the acute electrocorticogram: implications for pathophysiology and surgical treatment of temporal lobe epilepsy. *Brain* 1997;**120**:2259–82.
3. Bengzon AR, Rasmussen T, Gloor P, Dussault J, Stephens M. Prognostic factors in the surgical treatment of temporal lobe epileptics. *Neurology* 1968;**18**(8):717–31.
4. Maxwell R. Intracranial monitoring and functional localization. In: Apuzzo M, editor. *Neurosurgical aspects of epilepsy*. Park Ridge, IL: American Association of Neurological Surgeons; 1991. p. 103–16.
5. McBride MC, Binnie CD, Janota I, Polkey CE. Predictive value of intraoperative electrocorticograms in resective epilepsy surgery. *Ann Neurol* 1991;**30**:526–32.

6. Engel J, Driver MV, Falconer MA. Electrophysiological correlates of pathology and surgical results in temporal lobe epilepsy. *Brain* 1975;**98**:129–56.
7. Stefan H, Quesney LF, Abou-Khalil B, Olivier A. Electrocorticography in temporal lobe epilepsy surgery. *Acta Neurol Scand* 1991;**83**:65–72.
8. Fiol ME, Gates JR, Torres F, Maxwell RE. The prognostic value of residual spikes in the postexcision electrocorticogram after temporal lobectomy. *Neurology* 1991;**41**:512–6.
9. Schwartz T, Bazil C, Walczak TS, Chan S, Pedley TA, Goodman RR. The predictive value of intraoperative electrocorticography in resections for limbic epilepsy associated with mesial temporal sclerosis. *Neurosurgery* 1997;**40**(2):302–9.
10. McKhann GM, Schoenfeld-McNeill J, Born DE, Haglund MM, Ojemann G. Intraoperative hippocampal electrocorticography to predict the extent of hippocampal resection in temporal lobe epilepsy surgery. *J Neurosurg* 2000;**93**:44–52.
11. Gómez-Utrero E, Sánchez-Alonso A, Alijarde MT, Navarrete EG. Valor pronóstico de la electrocorticografía en la epilepsia temporal: patrones de la relación mesial y neocortical. *Rev Neurol* 2001;**33**(9):801–8.
12. Buzsáki G, Leung L, Vanderwolf CH. Cellular bases of hippocampal EEG in the behaving rat. *Brain Res Rev* 1983;**6**:139–71.
13. Buzsáki G, Horváth Z, Urioste R, Hetke J, Wise K. High frequency network oscillation in the hippocampus. *Science* 1992;**256**:1025–7.
14. Bragin A, Jandó G, Nádasdy Z, Hetke J, Wise K, Buzsáki G. Gamma (40–100 Hz) oscillation in the hippocampus of the behaving rat. *J Neurosci* 1995;**15**:47–60.
15. Chrobak JJ, Buzsáki G. High frequency oscillations in the output networks of the hippocampal–entorhinal axis of the freely behaving rat. *J Neurosci* 1996;**16**:3056–66.
16. Hirai N, Uchida S, Maehara T, Okubo Y, Shimizu H. Enhanced gamma (30–150 Hz) frequency in the human medial temporal lobe. *Neuroscience* 1999;**90**(4):1149–55.
17. Bragin A, Engel Jr J, Wilson CL, Fried I, Buzsáki G. High-frequency oscillations in human brain. *Hippocampus* 1999;**9**:137–42.
18. Bragin A, Wilson CL, Staba RJ, Reddick M, Fried I, Engel Jr J. Interictal high frequency oscillations (80–500 Hz) in the human epileptic brain: entorhinal cortex. *Ann Neurol* 2002;**52**(4):407–15.
19. Falconer MA, Serafetinides EA, Corsellis JAN. Etiology and pathogenesis of temporal lobe epilepsy. *Arch Neurol* 1964;**10**:233–48.
20. Fisher RS, Webber WR, Lesser RP, Arroyo S, Uematsu S. High-frequency EEG activity at the start of seizures. *J Clin Neurophysiol* 1992;**9**:441–8.
21. Bragin A, Engel Jr J, Wilson CL, Fried I, Mathern GW. Hippocampal and entorhinal cortex high-frequency oscillations (100–500 Hz) in human epileptic brain and in kainic acid-treated rats with chronic seizures. *Epilepsia* 1999;**40**:127–37.
22. Engel Jr J. Outcome with respect to epileptic seizures. In: Engel Jr J, editor. *Surgical treatment of the epilepsies*. New York, NY: Raven Press; 1987. p. 553–71.
23. Jeong SW, Lee SK, Hong KS, Kim KK, Chung CK, Kim H. Prognostic factors for the surgery for mesial temporal lobe epilepsy: longitudinal analysis. *Epilepsia* 2005;**46**(8):1273–9.
24. McIntosh AM, Wilson SJ, Berkovic SF. Seizure outcome after temporal lobectomy: current research practice and findings. *Epilepsia* 2001;**42**(10):1288–307.
25. Chabardes S, Kahane P, Minotti L, Tassi L, Grand S, Hoffmann D, et al. The temporopolar cortex plays a pivotal role in temporal lobe seizures. *Brain* 2005;**128**(8):1818–31.
26. Ylinen A, Bragin A, Nádasdy Z, Jando G, Szabo I, Sik A, et al. Sharp wave-associated high frequency oscillation (200 Hz) in the intact hippocampus network and intracellular mechanisms. *J Neurosci* 1995;**15**:30–46.
27. Babb TL, Brown WJ. Pathological findings in epilepsy. In: Engel Jr, editor. *Surgical treatment of the epilepsies*. New York: Raven Press; 1987. p. 511–40.
28. De Lanerolle NC, Brines ML. Neurochemical remodeling of the hippocampus in human temporal lobe epilepsy. In: Engel Jr J, Wasterlain C, Cavalheiro EA, Heinemann U, Avanzini G, editors. *Molecular neurobiology of epilepsy*. Amsterdam: Elsevier Science; Epilepsy Res 1992 (Suppl. 9) p. 205–20.
29. Mathern GW, Babb TL, Bruton CJ. Hippocampal sclerosis. In: Engel Jr J, Pedley TA, editors. *Epilepsy: comprehensive textbook*. New York: Lippincott-Raven Publishers; 1997. p. 133–55.
30. Grenier F, Timofeer I, Steriade M. Neocortical very fast oscillations (ripples 80–200 Hz) during seizures: Intracellular correlates. *J Neurophysiol* 2003;**89**(2):841–52.
31. Palmieri A, Gambardella A, Andermann F. Intrinsic epileptogenicity of human dysplastic cortex as suggested by corticography and surgical results. *Ann Neurol* 1995;**37**:476–87.
32. Cascino G, Trenerry MR, Jack CR, Dodick D, Shalhough FW, Elson LS, et al. Electrocorticography and temporal lobe epilepsy: relationship to quantitative MRI and operative outcome. *Epilepsia* 1995;**36**(7):692–6.
33. Kanner A, Kaydanova Y, Toledo-Morrell L, Morrell F, Smith M, Bergen D, et al. Tailored anterior temporal lobectomy. *Arch Neurol* 1995;**52**:173–8.
34. Tran TA, Spencer SS, Marks D, Javidan M, Pacia S, Spencer DD. Significance of spikes recorded on electrocorticography in nonlesional medial temporal lobe epilepsy. *Ann Neurol* 1995;**38**:763–70.
35. Cendes F, Dubeau F, Olivier A, Cukiert A, Andermann E, Quesney LF, et al. Increased neocortical spiking and surgical outcome after selective amygdalohippocampectomy. *Epilepsy Res* 1993;**16**:195–206.
36. Mitchell LA, Jackson GD, Kalnins RM, Saling MM, Fitt GJ, Ashpole RD, et al. Anterior temporal abnormality in temporal lobe epilepsy. A quantitative MRI and histopathologic study. *Neurology* 1999;**52**:327–36.
37. Meiners LC, Valk J, Jansen GH, Luyten PR. Magnetic resonance of epilepsy: three observations. In: Shorvon SD, Fish DR, Andermann F, Bydder GM, Stefan H, editors. *Magnetic resonance scanning and epilepsy*. New York: Plenum; 1994. p. 79–82.