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Leaving tissue associated with infrequent intracranial EEG seizure onsets is compatible with post-operative seizure freedom

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Abstract

Identify seizure onset electrodes that need to be resected for seizure freedom in children undergoing intracranial electroencephalography recording for treatment of medically refractory epilepsy. All children undergoing intracranial electroencephalography subdural grid electrode placement at the Children's Hospital of Philadelphia from 2002-2008 were asked to enroll. We utilized intraoperative pictures to determine the location of the electrodes and define the resection cavity. A total of 15 patients had surgical fields that allowed for complete identification of the electrodes over the area of resection. Eight of 15 patients were seizure free after a follow up of 1.7 to 8 yr. Only one seizure-free patient had complete resection of all seizure onset associated tissue. Seizure free patients had resection of 64.1% of the seizure onset electrode associated tissue, compared to 35.2% in the not seizure free patients (p=0.05). Resection of tissue associated with infrequent seizure onsets did not appear to be important for seizure freedom. Resecting ≥ 90% of the electrodes from the predominant seizure contacts predicted post-operative seizure freedom (p=0.007). The best predictor of seizure freedom was resecting ≥ 90% of tissue involved in majority of a patient's seizures. Resection of tissue under infrequent seizure onset electrodes was not necessary for seizure freedom.

Keywords

Epilepsy; epilepsy surgery; cortical dysplasia; neocortical epilepsy; intracranial electroencephalography

1. Introduction

For children with medically refractory focal epilepsy, surgical intervention often offers the best hope for long-term seizure freedom. The central question for surgical management is

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determining what tissue must be resected in order for a patient to be seizure free post surgery. Surgical evaluation includes multiple non-invasive studies, including long-term video electroencephalography (EEG) monitoring, imaging and neuropsychological evaluations. Unfortunately, invasive subdural intracranial EEG recordings to define the seizure onset region and tailor the resection are still required in some patients. In clinical practice defining the tissue that must be removed for seizure freedom is challenging as the frequency or characteristics of interictal spiking, the presence of secondary ictal foci, extent of interictal slowing are subjective criteria that may differ between and within epilepsy centers [1,2 4,7]. Using intracranial EEG the Duchowny group [4] determined that a complete resection of the electrodes involved in seizure onset, as well as regions of frequent abnormal interictal epileptiform discharges was the best predictor of a good outcome after epilepsy surgery [2-6]. Even with the use of invasive and non-invasive techniques seizure freedom following focal cortical resections remains low (37-55%) especially in patients with normal magnetic resonance imaging's (MRI's) [2,8,9]. Recently it was shown that patients can be seizure free with more limited resections, though determining why a limited resection can work in some patients is not well understood [10].

To improve surgical outcome while limiting resection size it would be helpful to develop methods for determining how to deal with multiple intracranial EEG seizure onset locations and relate these features to resection and outcome. Here we set out to quantify, in pediatric patients undergoing intracranial EEG, the completeness of the resected seizure onset associated tissue and determine the relationship to seizure outcome. Using this data, we have identified a relationship between the resection of the electrodes associated with the most common seizure onsets and surgical outcome. Long-term these results will hopefully, help us design larger surgical trials to improve outcomes and ultimately help us better understand the epileptic network.

2. Material and methods

Children's Hospital of Philadelphia Institutional Review Board approved the study. Between 2002 and 2008, 42 patients underwent intracranial EEG recordings for medically intractable epilepsy and were enrolled with informed consent in a study of intracranial EEG analysis and relationship to resection and outcome. We retrospectively reviewed all patients studying intra-operative photographs of their pre-grid placement, post-grid placement, post-resection and included patients that had the complete surgical resection field within the picture, where a relationship between the electrodes and tissue resected could be confidently established. Of 32 patients, 15 met all of these criteria and could be included in the analysis (Fig. 1). Most of the patients were excluded because the complete surgical field was obscured by the dura and skull. Nineteen of the 32 patients (60%) enrolled were seizure free similar to the cohort analyzed.

Presurgical evaluation for placement of the intracranial grids included scalp EEG recordings of interictal, ictal events and MRI for all patients. Single photon emission computed tomography, positron emission tomography and magnetoencephalography were carried out on a small subset of the patients.

The decision on what tissue to resect was made by the treating group of epileptologists, independent of the current study, taking into account seizure onsets, interictal abnormalities, motor and language mapping, and data from the non invasive studies (scalp EEG, MRI, and magnetoencephalography). The extent of clinical resection was limited in some patients by the desire to avoid resection of language and motor cortex.

The data that was used for the study included multiple photographs taken in the operating room, during the placement of subdural grids and at the time of resection, for comparison of

grid placement and pre- and post-resection cortex. All pictures were taken by the neurosurgeon standing directly above the operative field. Images of the grids on the cortex and the postoperative resection cavity were aligned by rotating the pictures using Adobe Photoshop (San Jose, CA). Major landmarks within the pictures, including large blood vessels, gyral patterns and the edges of the craniotomy were used to align the images of the cortex, grids, and resection cavity. Two electrophysiologists retrospectively reviewed all intracranial EEG recordings and identified the seizure onset electrodes by consensus, with no prior knowledge of tissue resected and blinded to patient outcome. To identify seizure onset electrodes, the unequivocal electrical onset was determined and each electrode was traced back to the earliest electrical change. The earliest electrical change was defined as electrodes in which there was a clear change in the frequency, amplitude or morphology of the background EEG leading up to the unequivocal electrical onset. All seizures, including clinical, subclinical, and atypical seizures were used for seizure onset marking.

Post-operative seizure evaluations were performed by the treating neurologist and outcome was recorded using a modified Engel [11] classification: Class 1 -seizure free; Class 3-worthwhile improvement; Class-4 no worthwhile improvement, follow-up duration is as listed in table 1. All groups were compared using a Fisher exact test using InStat software (GraphPad Software, LaJolla, CA).

3. Results

Clinical characteristics of all 15 patients are shown in table 1. Eight of the patients were seizure free (class 1), and seven were not seizure free, four-class 3 and three-class 4. The seizure free and not seizure free groups were similar in most respects including age at surgery; surgical sites, follow-up duration and all but two of the patients had cortical dysplasia as their underlying pathology on histological examination (Table 1). The numbers of electrodes implanted per patient varied from 68 to 128, but the mean number of implanted electrodes was 103 for both the seizure free and not groups (Tables 2 and 3). There was no difference in the average number of electrodes resected (23.5±14, Mean±standart deviation [SD] for seizure free or 24.2±12, Mean±SD for not seizure free) (Table 3). Thirteen of 15 patients had cortical dysplasia, but only six had an MRI detectable lesion (Table 1). Four of the six who's post-operative MRI had no residual detectable lesion were seizure free, while the two patients with residual lesion had persistent seizures.

There was no differences in the numbers of electrodes implanted, number of electrodes resected, or pathology, between the seizure free and non-seizure free patients. Number of seizure captured per patient varied widely, between 5-201, with no statistically significant difference noted between the seizure free and not seizure free groups. The number of seizure onset electrodes varied per patient 3-47. There was no difference, however, in the numbers of electrodes involved in seizure, onsets between the seizure free (17.6±12, mean±SD) and not seizure free patients (24.7±13, mean±SD). The seizure free patients had more tissue underlying the seizure onset region resected, than non-seizure free patients (64.1% versus 35.2%, t-test P=0.05). Of the eight seizure-free, patients only one, patient 7, had complete resection of all tissue associated with seizure onset electrodes. This suggests that complete resection of all tissue associated with all the seizure onsets is not necessary for seizure freedom (Fig. 2).

Each patient had considerable variability in the number of onset electrodes involved in each seizure (Fig. 2). For example, a patient could have seizures arising from eight electrodes (Fig. 2-patient 1), with four electrodes 97,98,102,103 involved in the majority of seizure onsets and four electrodes 94,95,96,99 infrequently involved. Qualitative review of all patients suggested that both seizure free and not seizure free patients had tissue from rare

seizure onset electrodes that was not resected. These results are illustrated by the graph of each patient shown in figs 2 and 3. Plotted on the x-axis are any electrodes associated with seizure onsets; plotted on the y-axis are number of seizures with onset from that electrode. The colors of the bars represent if an electrode was resected: red- completely resected, yellow-edge of the resection directly under the electrode, blue- not resected (Figs 2 and 3). The seizure free patients in contrast to the not seizure free patients had almost all of the seizure onset tissue associated with the frequent seizure onsets resected. We compared the seizure free and non-seizure free patients (Figs 2 and 3) and attempted to identify the surgically necessary seizure onset zone that would distinguish the two populations. Six of eight seizure free patients had resection of almost all, ≥ 90%, of tissue associated with the most frequent seizure onset electrodes (Table 3). In contrast, 0/7 non-seizure free patients had ≥90% resection of the tissue associated with predominant seizure onsets captured. Therefore, resection of ≥90% of tissue associated with the predominant seizure onsets was a significant predictor of surgical success (Fisher exact test, P=0.007). Two seizure free patients had 12% and 75% of the major seizure onset electrodes removed. These two patients further reinforce the notion that not all seizure onset contacts need to be removed to render a patient seizure free.

4. Discussion

There are two important findings in this study. First, a complete resection of all seizure onset zones is not necessary for seizure freedom. Second, identifying the electrodes involved in the onset of the most frequent seizures and resecting ≥90% of the region is sufficient for seizure freedom post epilepsy surgery.

Using pictures taken in the operating room to directly visualize the electrode placement and resection cavity, we quantified the extent of seizure onset zone that must be resected for seizure freedom. We were surprised to see that most patients who achieved seizure freedom had less than a total resection of all the seizure onset associated electrodes. In particular the tissue underlying electrodes that were associated with rare seizure onsets did not have to be resected for seizure freedom. However, identifying the number of seizures arising from each electrode and focusing on resecting almost all (≥90%) of the predominant seizure onset region could predict a high likelihood of postoperative seizure freedom. There was even a single patient that had as little as 12% of the seizure onset cortex resected and still experience seizure freedom. Several prior studies, have suggested that complete resection of all the epileptic activity is the best predictor for seizure freedom [2-6]. However, a more recent study of Perry et al., 2010, [10] showed a more limited resection is successful in some patients. Our data would agree with both studies but extends the analysis to suggest that resection of infrequent seizure onset regions are not necessary for seizure freedom.

There are limitations of using the current method of operating room photos and prior methods using MRI to determine the extent of the resection. These include shifts in electrodes or swelling of the brain over the duration of the implantation or during removal of electrodes and tissue. Another limitation is due to limited exposure in the operating room of the resection area. In our study, more than half of the patients could not have the resection mapped. Though MRIs obtained during implantation and post-resection may have problems with swelling and shifting of the brain to fill the resection vacuum; MRI visualization post operatively is the preferred method for identifying residual dysplastic or gliotic tissue. Overall, we believe that our direct visualization method allowed for an accurate determination of the relationship between the subdural electrodes and the underlying tissue resected in the 15 patients where complete visualization of the resective field was possible.

Prior studies have suggested that areas of consistent seizure spread also need to be resected to improve seizure freedom [2-6]. While our data suggests that there is a difference in outcome based on resection of tissue from frequent and infrequent seizure onset regions, it is possible that the infrequent seizure onset electrodes were misidentified. Instead the infrequent seizure onset may be secondary ictal spread that does not need to be resected. A future study would need to be carried out to address this question more specifically.

A number of studies have documented significant variability amongst epileptologists in seizure detection and localization on surface and intracranial EEG [12-14]. Osorio et al. 2002, [14] had expert electrophysiologists review intracranial EEG for the presence of seizures and showed poor inter rater reliability. While this has not been done for seizure onset electrodes, variability between and within epileptologists might contribute to the difficulty in distinguishing onset electrodes versus ictal spread. Potential confounders for this work include variable seizure onset morphology and artifacts at the start of seizures.

The difficulty in distinguishing seizure onset from spread may explain why the electrodes infrequently associated with seizure onset seem much less important for seizure freedom. It would not, however, explain patient 8's seizure freedom following resection of 12% of seizure onset electrode associated tissue. While a number of explanations could account for this patient's good outcome, particularly the complete resection of the dysplasitc lesion on MRI, this patient and our data as a whole raise the intriguing idea that seizures have a critical epileptogenic region necessary for generation of a seizure. We hope that in the future new techniques with higher frequency recordings or computer analysis can help distinguish seizure generators from areas of propagation.

While most patient's histopathologic diagnosis was cortical dysplasia (13/15) only six had a dysplastic lesion identified on MRI. This suggests that MRI used in a clinical setting is not highly sensitive for identifying cortical dysplasia. In this subgroup, removal of the complete MRI lesion was associated with seizure freedom in four patients. Conversely, the two patients with remaining lesional tissue were not seizure free. This data agrees with prior studies, which have suggested that leaving an MRI lesion is associated with poor outcome and might even trump electrographic-based resections [4].

We have not addressed the need to resect interictal epileptic activity. Several groups, including our own, have previously shown that interictal sharps and spikes on intracranial EEG correlate with seizure onset in a subset of patients [15,16,7]. The resections performed for this study were done based strictly on the impression of the treating physician and likely, both seizure onset and interictal data were taken into account. Our retrospective analysis of the seizure onset data suggests that tailored resections focused on frequent seizure onset regions can be sufficient.

Why is it that we can leave electrodes associated with seizure onset zone and still obtain seizure freedom? Though our findings cannot answer this question, the finding of seizure freedom without complete resection is in keeping with previous studies that suggest interrupting the epileptic network or removal of the ictal generators could be sufficient to render a patient seizure free [5,10]. Future work should be aimed at identifying markers of an ictal generator to determine the minimal amount of brain tissue necessary for removal to make patients seizure free.

In conclusions, in children requiring intracranial EEG to better define the epileptogenic zone, large amounts of ictal and interictal EEG information must be analyzed to determine the resection most likely to produce seizure freedom. We found that almost none (1/8) of our seizure free patients had the entire seizure onset zone resected from all the seizures captured. Resection of tissue associated with regions of frequent seizure onset predicted a good

outcome and tissue rarely involved in seizure onsets did not have to be resected for post-operative seizure freedom. Due to the small number of patients in our cohort a repeat analysis with a second group of children needs to be performed.

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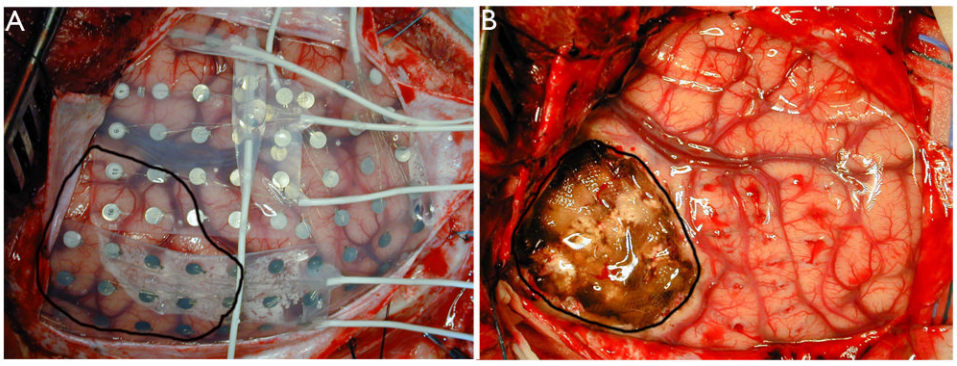

Fig 1.

(A) Pre-resection picture taken in the operating room with electrodes on the surface of the brain. (B) Picture taken in the operating room post-resection. Black line is our approximation of the resection cavity.

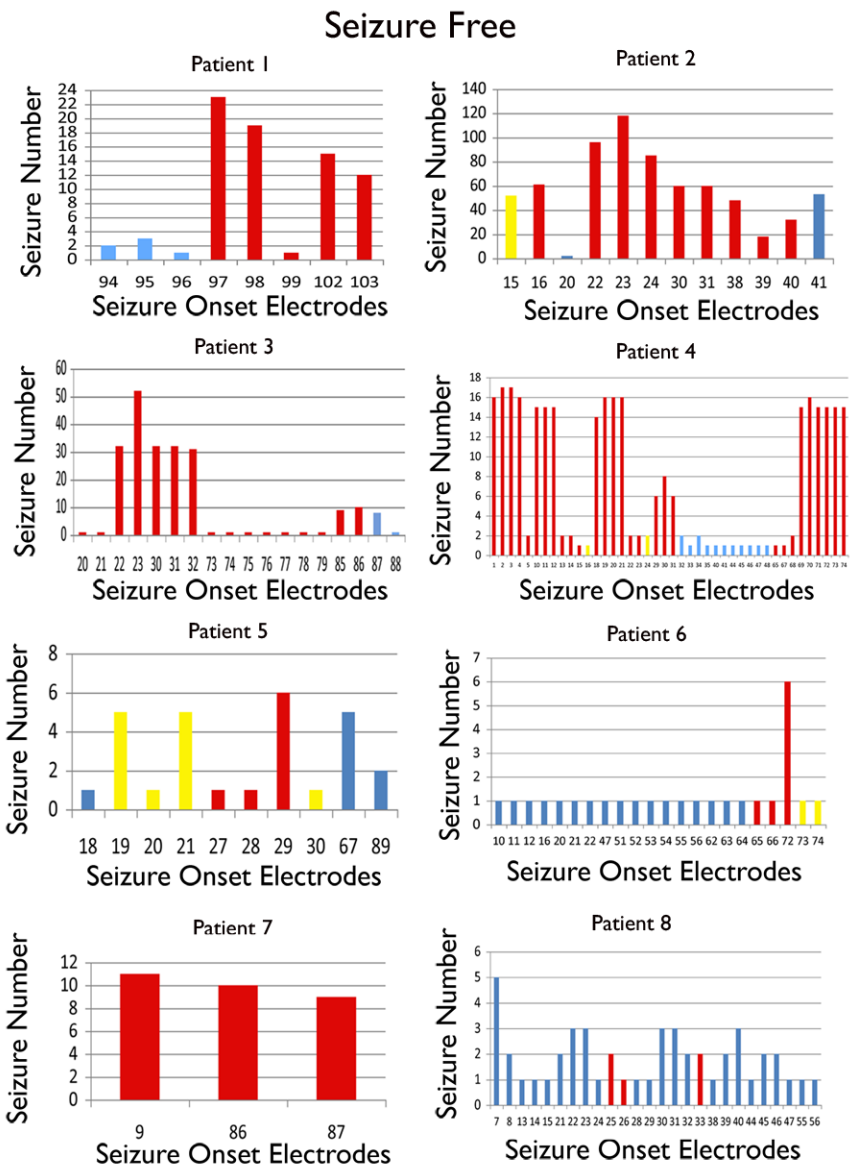

Fig. 2.

Graphs from the seizure free patients showing number of seizure onsets on the Y-axis arising from each electrode on the X-axis. The color of each bar represents if the tissue underlying the electrode was resected. Red-resected; yellow-the edge of the resection was immediately under the electrode; blue-unresected. Patient 7 is the only patient with a complete resection of all the seizure onset electrode associated tissue. Patients 1,2,3,4 and 6 had ≥ 90% of the associated tissue resected from the most common seizure onset electrodes. Patients 5 and especially patient 8 had < 90% of the associated tissue resected from the most common seizure onset electrodes but was still seizure free.

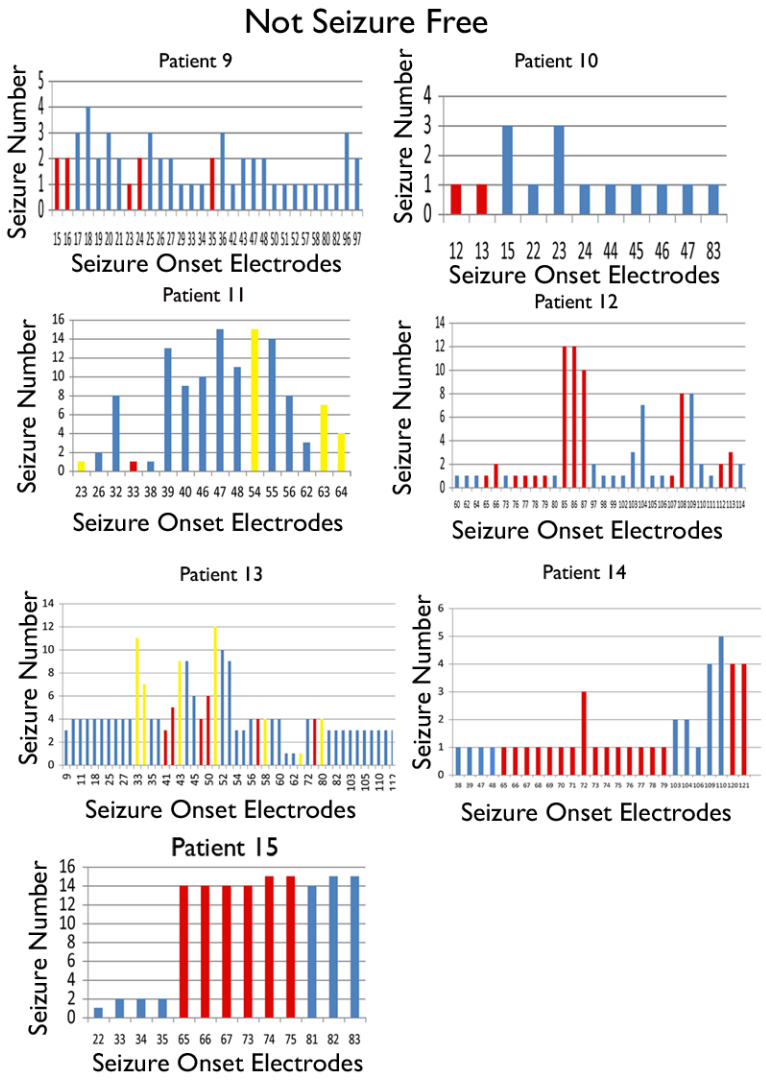

Fig. 3.

Graphs from the non-seizure free patients showing number of seizure onsets on the Y-axis arising from each electrode on the X-axis. The color of each bar represents if the tissue underlying the electrode was resected. Red-resected; yellow-the edge of the resection was immediately under the electrode; Blue-unresected. While several patients had tissue resected that was associated with a large number of seizure onset electrodes, see 12 and 14. None of the patients had ≥ 90% of the tissue associated with the most frequent seizure onset electrodes.

Table 1

Clinical Summary

Patient number #	Age at surgery (yr)	Resection	Pathology	Magnetic resonance imaging lesion / resected	Follow-up duration (yr)	Outcome	Seizure #
1	7	Right frontal	Cortical dysplasia - type 2B	No	8	1	32
2	5	Right frontal	Cortical dysplasia - type 2B	Yes/yes	2.9	1	201
3	11	Right frontal	Cerebral vascular accident	Yes	2.4	1	53
4	5	Left frontal	Cortical dysplasia - type 2A	Yes/yes	2	1	22
5	16	Right frontal	Cortical dysplasia - type 2A	No	2	1	6
6	3	Right temporal	Cortical dysplasia - type 3B	Yes/yes	2	1	8
7	8	Left temporal	Cortical dysplasia - type 2A	No	1.9	1	13
8	14	Right frontal	Cortical dysplasia - type 2B	Yes/yes	1.7	1	5
9	18	Left frontal	Cortical dysplasia - type 2A	No	6.7	3	8
10	16	Right frontal	Cortical dysplasia - type 2A	No	4.9	4	5
11	15	Right frontal	Cortical dysplasia - type 2A	Yes/no	3.2	3	17
12	7	Right temporal	Cortical dysplasia - type 2B	No	2.1 and 2nd surgery	3	14
13	5	Right frontal	Cortical dysplasia - type 2A	Yes/no	0.5 and 2nd surgery	4	13
14	11	Left frontal	Cortical dysplasia - type 2A	No	6	3	13
15	12	Right temporal, parietal, occipital	Cortical dysplasia - type 2A	No	3	4	16

Cortical dysplasia typing is according to (Blumcke et al., 2011) [17], Class 1 -seizure free; Class 3-worthwhile improvement; Class-4 no worthwhile improvement,

#-Number

Table 2

Intracranial EEG Summary

Pt. #	Total # electrodes implanted	Electrodes removed / total electrodes %	# Onset electrodes all seizures	Onset electrodes resected / all seizures %	# Onset electrodes predominant seizures	# Onset electrodes resected / predominant seizure (%)
1	120	11 %	8	63 %	4	4 (100%)
2	92	24 %	12	83 %	11	10 (91%)
3	116	41 %	18	89 %	5	5 (100%)
4	92	46 %	42	74 %	20	20 (100%)
5	126	11 %	10	70 %	4	3 (75%)
6	74	16 %	22	23 %	1	1 (100%)
7	116	16 %	3	100 %	3	3 (100%)
8	84	18 %	26	12 %	6	0 (0%)
9	100	8 %	30	17 %	18	4 (22%)
10	94	18 %	11	18 %	2	0 (0%)
11	68	23 %	16	31 %	10	2 (20%)
12	112	14 %	30	43 %	6	4 (66%)
13	116	30 %	47	27 %	7	4 (57%)
14	128	30 %	26	65 %	5	3 (60%)
15	96	34 %	13	46 %	9	6 (66%)

#-Number

Table 3

Summary Comparison of Seizure Free and not Seizure Free

Analysis	Seizure free	Not seizure free	Statistic
Average # of electrodes / patient	103 ± 19 (SD)	103 ± 20 (SD)	Not significant
Average # of seizures / patient	42.5 ± 66 (SD)	12.8 ± 4 (SD)	Not significant
Average # of electrodes removed / patient	23.5 ± 14 (SD)	24.2 ± 12 (SD)	Not significant
Average # of onset electrodes / patient	17.6 ± 12 (SD)	24.7 ± 13 (SD)	Not significant
Average % onset electrodes resected / patient	64.1% ± 31 (SD)	35.2% ± 20 (SD)	P=0.05
Resected >90% of predominant seizure onset electrodes	6/8	0/7*	*P=0.007

#-Number,

SD: Standard deviation,

*- Significant,