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A physiologically motivated ECoG segmentation method for epileptic seizure onset zone detection

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***Abstract*—In this paper we propose a novel segmentation method based on the relative frequency contributions of ictal multichannel ECoG data. Segments with predominant ϑ-activity are classified as epileptic. The seizure onset zone is determined by the temporal delay of the epileptic ϑ-activity (4-9Hz) on the different channels. We apply this methodology to three seizures of one patient suffering from focal epilepsy. The resulting segments reflect the visual characteristics of the ictal ECoG well. The seizure onset zone identified by the proposed method is in very good accordance with the clinical findings.**

I. Introduction

A. Background

Epilepsies, defined as disorders with recurrent unprovoked seizures, affect approximately 0.7% of the general population (cf. [1]). Seizures are characterized by abnormal synchronized neuronal discharge of networks in both hemispheres (generalized seizures) or in circumscribed networks in one hemisphere (focal seizures). About one third of patients with focal epilepsies develop drug resistant seizures. If a clear-cut definition of the seizure onset zone is possible, resective epilepsy surgery is a valuable treatment option for these patients (cf. [2] and [3]). Up to 70% of patients with drug resistant focal epilepsy become seizure free following surgery. Presurgical evaluation comprises prolonged video-EEG recording, high resolution brain imaging as well as neuropsychological tests. If scalp EEG cannot provide sufficient information on the seizure onset zone, invasive recording using subdural strip electrodes (*electrocorticography*, *ECoG*), which are placed directly on the brain surface, is performed in order to define the epileptic focus (cf. [4]).

The ECoG-based analysis of the initial seizure propagation including the determination of the seizure onset zone can be carried out from two different points of view: the visual inspection of the raw ECoG recordings by clinical experts (currently regarded as gold standard, cf. [5] and [6] for two recent studies) or a computational data analysis based on mathematical algorithms. The latter approach includes various technical methods, e.g. causality measures (cf. e.g [7]) or source analysis methods (cf. e.g. [8]). Instead we propose a different, physiologically motivated computational approach, based on the propagation of ϑ-activity.

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B. Contributions and state of the art

In this paper we analyze ictal ECoG recordings (i.e. during a seizure) with respect to seizure spread; seizure onset time is provided by clinicians. Our contributions to the area of ictal ECoG data analysis are twofold:

1) We propose a novel physiologically motivated segmentation method based on the relative frequency contributions of ictal ECoG data (cf. Subsection II-A).
2) Based on this segmentation we classify each segment with respect to its epileptic character. The temporal delay of the onset of epileptic activity on the different channels is an indicator for seizure propagation, thus revealing the seizure onset zone (cf. Subsection II-B).

In both cases literature offers a wide variety of related ideas:

In order to cope with the instationarities of EEG and ECoG signals, various segmentation methods have been developed in the last decades: The prominent *Spectral Error Measure (SEM)* was introduced in [9], a nonlinear energy operator was used in [10] and a generalized Kolmogorov-Smirnov-statistics in [11]. Other segmentation approaches involve the use of information criteria [12] or the Itakura distance [13].

Seizure detection analysis aims at the temporal detection of epileptic seizures in long-term EEG recordings by evaluating the epileptic character of the combined channel set. We refer to [14] and [15] for an overview of common detection approaches. Other methods include non-linear approaches like entropies [16] or the Recurrence Quantification Analysis (RQA) [17].

II. Method

In our analysis we consider multivariate signals consisting of K channels $x_k[n]$, $k = 1..K$, where n denotes the time index. We assume the signals to be zero-mean and stationary in a sufficiently short data window.

Our proposed methodology consists of two consecutive steps, the segmentation of the ECoG data (cf. Subsection II-A) and the subsequent classification of the resulting segments regarding their epileptic character (cf. Subsection II-B). As the segmentation and classification are applied channel-wise, we will explain them for an arbitrary single channel $x_k[n]$ in the following.

We initially compute power spectral densities varying over time using a sliding window of length T_{win} where the window

is moved by T_{res} seconds. In each window we associate the spectrum with the window center on the time axis. Thus we obtain a sequence of power spectral densities $S(f)[m]$ with time index m (new temporal resolution $1/T_{res}$) which will be the basis of all subsequent calculations.

From neurophysiological considerations we obtain the following frequency bands: δ_{low} (1.0-1.5Hz), δ_{up} (2.0-3.5Hz), ϑ (4.0-9.0Hz), α (9.5-13.5Hz), β (14.0-30.0Hz). For each time step m, we calculate the power of these bands, e.g. $P_\alpha[m] = \int_{9.5}^{13.5} S(f)[m]\,df$, as well as the total power of all these bands $P[m]$. The split of the δ-band was due to technical reasons.

A. Segmentation

Our segmentation method is based on the temporal changes of the relative frequency contribution of the individual physiological bands. We choose an initial reference point m^*, and for increasing $m > m^*$ we calculate a novel statistics termed *Band Power Measure* ($BPM[m]$) as

$$BPM[m] = \left(\frac{P_{\delta low}[m]}{P[m]} - \frac{P_{\delta low}[m^*]}{P[m^*]}\right)^2 + \ldots + \left(\frac{P_{\beta}[m]}{P[m]} - \frac{P_{\beta}[m^*]}{P[m^*]}\right)^2.$$

If the Band Power Measure exceeds a given threshold th, i.e. $BPM[m] > th$, we start a new segment by updating the reference point $m^* = m + 1$ and continue the calculation for increasing m. The set of reference points obtained by this algorithm gives the boundary points for our segments, i.e. each resulting segment is limited by two subsequent reference points.

Due to its construction, the BPM ignores small contribution shifts within a frequency band. Note that this is in contrast to the SEM-based segmentation in [9].

According to [18] ictal EEG (as well as ECoG) in temporal lobe epilepsy patients is often characterized by its distinct rhythmic ϑ-activity. As our algorithm tends to yield segments with one predominant frequency band by construction, we consider it to be appropriate for the segmentation of these ictal signals.

B. Onset zone analysis

In the second step we decide whether a segment represents epileptic activity. For this purpose we propose a simple and intuitive rule which focuses on neurophysiological aspects of TLE patients: A segment is classified as epileptic if ϑ-activity is dominant within this segment (dominant ϑ-contribution in more than half of the segment points or maximal spectral peak in the ϑ-band in most of the segment points) due to the good localizing value of ϑ-activity, cf. [18]. The start of the epileptic activity on a single channel is indicated by the beginning of the first epileptic segment.

In order to draw conclusions on the initial seizure propagation we apply the segmentation and subsequent classification channel-wise to all channels. The temporal delay of the start of the epileptic ϑ-activity over the different channels is used for describing seizure propagation. The first channels showing epileptic ϑ-activity indicate the seizure onset zone.

C. ECoG data acquisition

The ECoG data used in this study are taken from a patient (male, 43 years) suffering from therapy-resistant focal epilepsy. The patient underwent a presurgical long-term video EEG monitoring at the Hospital Hietzing with Neurological Center Rosenhügel. Three subdural strip electrodes with a total of 25 channels were implanted, and the electrode B1 (outside the seizure focus) was chosen as reference. Compare the MRI (magnetic resonance imaging) scan in Fig. 4 for details.

Recording was performed using a Micromed® system at a sampling frequency of 1024Hz. Four seizures occurred within 2 hours during the monitoring period, but due to decreasing data quality we only analyzed the first three.

After recording, the ECoG data were preprocessed in Matlab®: Line interference was removed using a notch filter at 50 Hz, and a high-pass filter at 1Hz was applied in order to get rid of physiologically irrelevant low-frequency contributions. The signals were low-pass filtered at 64 Hz in order to avoid aliasing and then downsampled to 128 Hz sampling rate.

III. Results

As we are interested in the initial spread of the rhythmic ϑ-activity, we investigate the first 20 seconds of each of the three seizures (cf. Subsection II-C for the ECoG data acquisition). We start one second prior to paroxysmal fast activity (30Hz) or high-frequency oscillations (75Hz) according to the visual analysis of clinicians. Rhythmic ϑ-activity starts approximately 10 seconds later. Note that the detection of the initial fast activity is not in the focus of this study.

We apply the proposed methodology with the following set of parameters: $T_{win} = 1.5\,\mathrm{s}$, $T_{res} = \frac{1}{16}\mathrm{s}$. Power spectral densities are estimated using the non-parametric Welch method (128-point FFT).

A. Segmentation

For the segmentation we employ an empirically determined threshold $th = 0.07$ and an initial reference point m^* at 0.75s. We apply our segmentation to the full 24-channel set $x_k[n]$, $k = 1..24$, of all three seizures, but due to space limitations we only present results of seizure 1 in this subsection.

Fig. 1 displays the segmentation of an exemplary channel, A11, in detail. On top (Fig. 1 (a)) the ECoG data are shown. For a better comprehension the temporal evolution of the relative frequency contributions is detailed in the middle graph (Fig. 1 (b)) and the corresponding BPM statistics at the bottom (Fig. 1 (c)). Segment boundaries are marked in all three graphs to simplify their comparison.

In this example a significant change of the BPM statistics can be observed in case of frequency shifts from one physiological band into another. Furthermore the segments coincide well with phases of ϑ-domination.

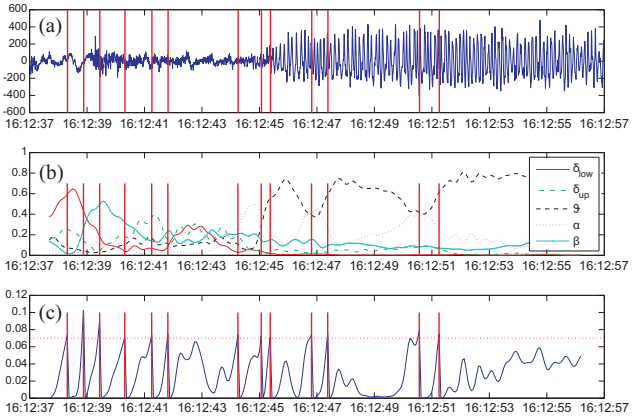

Figure 1. Segmentation of channel A11, seizure 1. (a) ECoG data, (b) Relative contributions of the physiological frequency bands, (c) Band Power Measure and imposed threshold (dotted). Segment boundaries are indicated by vertical lines in all three graphs.

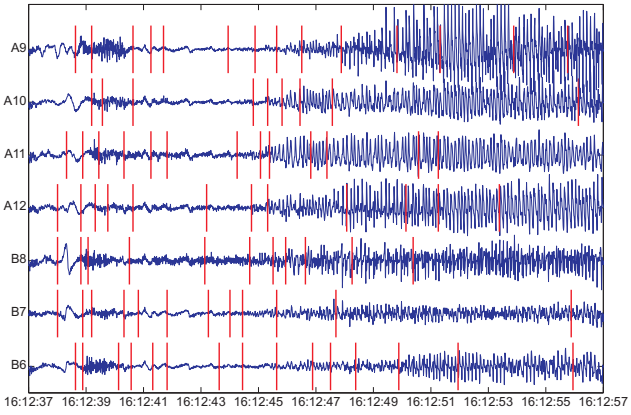

Figure 2. Segmentation of 7 selected channels, seizure 1. ECoG data, segment boundaries are indicated by vertical lines.

In Fig. 2 we highlight the segmentation of 7 selected channels (out of the segmented 24) which include the channels of the seizure onset zone. Segments are longer in the second half of the displayed 20 seconds which is dominated by rhythmic ϑ-activity.

B. Onset zone analysis

The segment classification based on the segmentation of the former step (all 24 channels of the three seizures respectively) allows us to investigate the initial propagation of the ϑ-activity and derive the localization of the seizure onset zone.

In Fig. 3 we exemplarily present our findings for seizure 1: In a four-second-zoom of Fig. 2 it displays the initial ϑ-propagation. For a better visibility segments classified as epileptic are highlighted. Channel B8 shows the first occurrence of dominant ϑ-activity, closely followed by electrodes A10, A11 and A12.

Table I summarizes our findings for all three seizures as well as the visual analysis. Our algorithm indicates that the seizure onset zone comprises the electrodes B8, A10, A11 and A12, which coincides with visual inspection of three experts

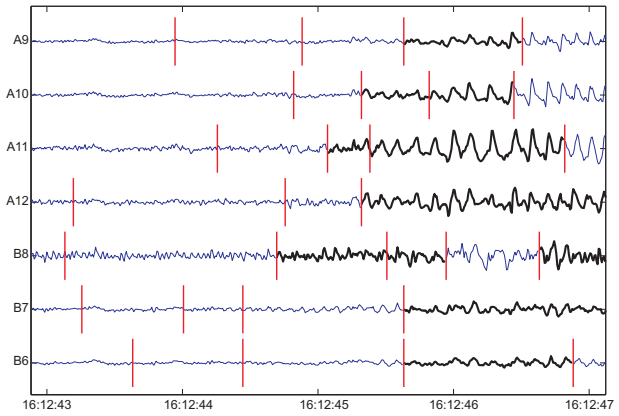

Figure 3. Propagation of ϑ-activity during four seconds in 7 selected channels, seizure 1. Segment boundaries are indicated by vertical lines, segments classified as epileptic are highlighted.

Table I
ONSET ZONE OF ALL THREE SEIZURES. RESULTS BASED ON OUR METHOD AND VISUAL INSPECTION BY CLINICIANS

Seizure	Investigator	Initial Electrodes	Close follow-up
1	*Algorithm*	*B8*	*A10, A11, A12*
	Expert 1	B8	A10, A11, A12
	Expert 2	A11, A12, B8	A9, A10, B7
	Expert 3	A10, A11, A12	B8
2	*Algorithm*	*A10, A11, A12*	*B6, B8*
	Expert 1	A11, A12	A9, A10
	Expert 2	A11, A12	A10
	Expert 3	A11, A12	B8
3	*Algorithm*	*A10, A11, A12*	*B6, C1, C2, C5*
	Expert 1	A9, A10	A8, A11, A12, B6, B7, B8, C1, C4, C5
	Expert 2	A9	A1, A2, A3, C2, C3
	Expert 3	A8, A9	A1, C3, C4, C5

(the two authors SP, CB and a technical assistant). Compare the MR image in Fig. 4 for a visualization of the electrode positions.

IV. Discussion

A. Segmentation

In Subsection III-A we showed that the BPM-based segmentation is an appropriate method for the segmentation of ictal ECoG data. Fig. 1 underlines the advantages of the construction of the BPM statistics: Prior to the rhythmic ϑ-activity (starting at ~16:12:45) we observe quickly interchanging frequency contributions (cf. plot (b)). This results in a BPM statistics with high variations and frequent threshold exceeding (cf. plot (c)). Therefore our algorithm yields short segments in this period. On the other hand, during the rhythmic activity, only small power shifts occur within the physiological frequency bands. Thus the frequency contribution of the respective bands show a constant behavior, namely the ϑ-band on a high level. This results in longer segments.

Fig. 2 as well underlines the ability of the proposed segmentation method to properly reflect the visual characteristics

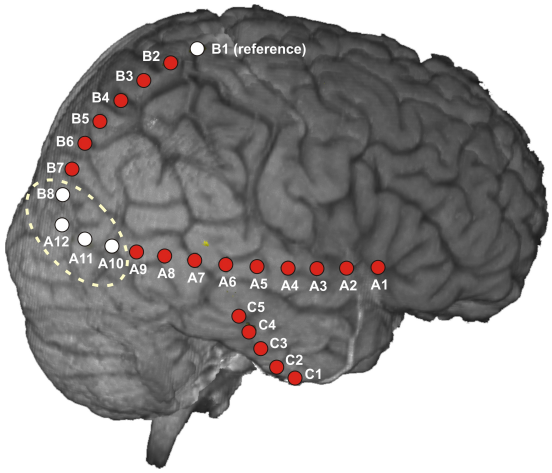

Figure 4. MRI scan with electrode positions. Electrodes of the seizure onset zone according to our method are marked.

of the ECoG data.

Due to the low threshold $th = 0.07$ we obtain a reactive segmentation behavior and short segments. In particular the influence of the initial reference point m^* is almost negligible in this setting.

B. Onset zone analysis

In this pilot study we assume that the initial ϑ-spread represents a valid indicator (among others) for seizure propagation in TLE, in particular for the determination of the seizure onset zone. Our results are in good accordance with the clinical findings (cf. Table I), which supports our assumption.

We would like to point out that our methodology is robust. Small parameter changes of th and m^* lead to a slightly different segmentation and classification, but the indicated seizure onset zone remains constant.

In our analysis we intentionally limit ourselves to tracking ϑ-activity. However, the observed patient also shows intermittent epileptic α-activity. These segments are not characterized as epileptic, cf. Fig. 3 (channels A9, A10, A11 at 16:12:47). For future work we propose patient-specific frequency bands indicated by the clinician.

Another possible amelioration is the improvement of classification rules which additionally consider the signal amplitude and the entropy as measures of rhythmicity (cf. e.g. [14]).

In the authors' opinion it is remarkable that the combination of two simple ideas delivers results which are consistent and well correlated with clinical findings. This might be due to the close relation between the method and neurophysiology.

V. Conclusion

In this paper we proposed a novel seizure onset zone detection method based on segmentation and subsequent classification of ictal ECoG data. This pilot study shows promising first results in tracking the initial propagation of ictal ϑ-activity as an indicator for seizure propagation. It therefore has the potential for an objectivation in the presurgical clinical evaluation of therapy-resistant patients.

However, this requires further research and an application to a broader data basis allowing for a performance evaluation (sensitivity vs. specificity, parameter variation) of the method.

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