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A new algorithm for epilepsy seizure onset detection and spread estimation from EEG signals

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Abstract.

Appropriate diagnosis and treatment of epilepsy is a main public health issue. Patients suffering from this disease often exhibit different physical characterizations, which result from the synchronous and excessive discharge of a group of neurons in the cerebral cortex. Extracting this information using EEG signals is an important problem in biomedical signal processing. In this work we propose a new algorithm for seizure onset detection and spread estimation in epilepsy patients. The algorithm is based on a multilevel 1-D wavelet decomposition that captures the physiological brain frequency signals coupled with a generalized gaussian model. Preliminary experiments with signals from 30 epilepsy crisis and 11 subjects, suggest that the proposed methodology is a powerful tool for detecting the onset of epilepsy seizures with his spread across the brain.

1. Introduction

The term epilepsy derives from the Greek term *epilambanein* which means to seize, and it denotes the predisposition to have recurrent, unprovoked seizures. Seizures can be symptomatic; that is, result from specific precipitants such as fever, strokes, metabolic disturbances (e.g. hypoglycaemia, drug abuse/withdrawal), trauma, infections in the central nervous system, and acute head injury. In epilepsy, however, seizures are unprovoked and expected to be recurrent [46]. Appropriate diagnosis and treatment of epilepsy is a main public health issue. According the World Health Organization [1], there are more than 50 millions people worldwide that suffer from some form of epilepsy, nearly 80% of them are in developing regions, where it is believed that 3 out of 4 people with this conditions do not get appropriate diagnostic and treatment [1].

The International League Against Epilepsy (ILAE) [10] defines "epileptic seizure as a transient occurrence of signs and/or symptoms due to abnormal excessive or synchronous neuronal activity in the brain". The elements defining an epileptic seizure include its mode of onset and termination, its clinical manifestations, and its abnormal enhanced synchrony [32]. Physical manifestations of epilepsy result mainly from the synchronous and excessive discharge of

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electricity by a group of neurons behaving abnormally in the cerebral cortex. Epileptic seizures usually have a sudden onset, they spread within seconds and, in most cases, are brief. The precise manifestation of a seizure depends on the location in the brain where it originates, and on how far and fast it spreads. The correct identification of this location and spread information is key to a proper treatment.

Electroencephalography (EEG) is a non-invasive and widely available biomedical modality that can be used to diagnose epilepsy and plan treatment. One particular task that has received attention during the last years is the detection of epileptic seizures from EEG signals measured on the brain scalp. It is widely acknowledged that detection can be improved by summarizing the EEG signals using relevant descriptors; that is, quantities computed from the EEG signals that capture the main relevant features of the seizure, and whose values help discriminating between normal and abnormal brain activity. The most relevant EEG features studied in the literature can be classified in three groups: a) frequency spectral properties such as band power and edge frequency; b) signal morphological properties such as wave amplitude, sharpness, line length, and zero crossings; and c) statistical descriptors such as statistical moments, correlation times and information entropies.

In this work we study a new algorithm for epilepsy seizure onset detection and spread estimation. The algorithms is based on a statistical generalised Gaussian model that represents the activity in each of the brain's main frequency bands, which are identified by using a multi-resolution temporal wavelet representation. The remainder of this document is structured as follows. Section 2 describes the state of the art in seizure onset detection (SOD). Section 3 defines the univariate generalised Gaussian statistical model. Section 4 describes the proposed the epilepsy seizure onset detection and spread estimation algorithm. In Section 5 the proposed methodology is demonstrated on real EEG signals from patients suffering from epileptic seizures. Discussions and conclusions are finally reported in Section 6.

2. Seizure Onset Detection

Seizure onset detection (SOD) methods stem from the observation that EEG descriptors allow discriminating between normal and abnormal brain activity; that is, between the expected rhythms of the brain and varying degrees of thalamocortical interdependence, and the activity resulting from seizures and burst suppressions. Epileptic attacks have two clinical manifestations that may reflect abnormal brain activity: *Ictal* and *Inter-Ictal* discharges, in which the impaired consciousness plays an important role (this is the inability to respond normally to exogenous stimuli by virtue of altered awareness and/or responsiveness) [24]. The Interictal discharges are a positive indicator of epilepsy and, depending on the duration of EEG recordings and the inclusion of different states of vigilance, they can be observed in up to 90% of cases [37].

The Ictal discharges are clinical signs used to detect the onset seizure from the epileptogenic zone in the brain cortex, SOD may enable physicians to better therapy with drug treatment, diagnostic and alert procedures, since is intended to recognize the start of a seizure, with the shortest possible delay, but not necessarily with the highest possible accuracy. The SOD when used with intracranial recordings, for seizure identification and retrospective analysis of seizures, often in the context of presurgical evaluations, it can be relatively straightforward to detect with reasonable sensitivity and specificity since it is dealing with events often lasting over a minute [22]. Another use of SOD with extracranial recordings to distinguish between primary and secondary irritative areas may be difficult, because a interictal discharge can spread via normal anatomical connections between cortical areas, through the commissural fibers or via subcortical structures, finally this may lead to widespread or bilateral occurrence of interictal discharges [45].

Seizure onset detection was first investigated in the seventies by Viglione et al. [50] and Liss et al. [30], and with later contributions by Ktonas et al. [27] and Gotman et al. [14]. Moreover,

several works studied linear and nonlinear prediction techniques to separate transients from background activity. For example, filter techniques [3], power spectrum techniques [11], crosscorrelation techniques [52], principal and independent component analysis techniques [33], [20] have been investigated. Other examples include techniques based on wavelet representations [15], state space reconstruction [35], correlation measures [43], signal dimension [17], density [29], integral [41], mutual prediction [44], Lyapunov exponents [19], synchronization [40], similarity measures [18], recurrence quantification measures [47], and nonlinear predictability [4]. We refer the reader to [49] for a comprehensive treatment of measurement, models, detection and prediction. Other important surveys of the literature in this topic can be found in [23], [15], [26], [7], [21], [53], [34], [28], [8], [48].

Modern SOD methods can be grouped into the following categories: 1) Template matching: These are techniques based on finding events tha match previously selected spikes; the detection is made whenever the cross correlation of the EEG with a template exceeds a threshold [31]; 2) Parametric methods: These techniques are based on traditional signal processing and consider that a seizure has occurred when the difference between the EEG signal and a predicted value (based on the assumption that the background is stationary) exceeds a threshold [2]; 3) *Mimetic* Methods: these techniques seek to copy the human expert (i.e. neurophysiologist) and operate by monitoring the value of parameters computed from each wave and applying thresholds [13], [6], [25]; 4) Morphologic Analysis: these techniques are based on the characterisation of the waveforms in terms of sharpness, amplitude, duration, convexity, frequency bands or timefrequency representations of spikes [13], [16]; 5) Syntactic methods: these techniques are based on the detection of the presence of structural features [51]; 6) Neural Networks: this approach adopts a machine learning perspective to learn transients related to epileptic seizures [42], [38]; 7) Expert systems: this approach detects seizures by mimicking an expert's knowledge and reasoning process[12]; 8) Data mining techniques: this approach also adopts a machine learning perspective to train a classifier [8], [9], [48]; 9) Clustering Techniques: in this case detections are based on hierarchical agglomerative processes and self-organising maps [3], [48]; 10) Knowledgebased rules: these techniques seek to incorporate knowledge from neurophysiologists who adopt spatial and temporal rules [5], [7], [5], [48].

3. Generalized Gaussian Distribution

The univariate generalised Gaussian distribution (GGD) is a flexible statistical model that has found numerous applications in science and engineering, and which recently was use to model epilepsy signals in [39]. This statistical model has 3 parameters, with a probability density function (PDF) given by:

$$f(x;\mu,\sigma,\mathcal{B}) = \frac{\mathcal{B}}{2\mathcal{A}(\sigma)\Gamma(\frac{1}{\mathcal{B}})}exp - \frac{|(x-\mu)|^{\mathcal{B}}}{2\sigma^2}$$
(1)

with

$$\mathcal{A}(\sigma) = \sigma \sqrt{\frac{\Gamma(\frac{1}{\mathcal{B}})}{\Gamma(\frac{3}{\mathcal{B}})}}, \quad \Gamma(z) = \int_0^{+\infty} e^t e^{-z-1} dt, \quad z > 0$$

and where $\mu \in \mathcal{R}$ is a location parameter, $\sigma \in \mathcal{R}^+$ is a scale parameter and $\mathcal{B} \in \mathcal{R}^+$ is a shape parameter that controls the shape of the density tail. The density (1) has many properties that are useful for modelling signals: i) the value of (1) approaches zero as x approaches positive and negative infinity, ii) the mean and mode of (1) coincide and are given by μ , iii) (1) is symmetric with respect to the mean value μ . Notice that the GGD parametric family comprises many popular distributions that are commonly used in biomedical signal processing. For example, setting $\mathcal{B} = 1$ leads to a Laplacian or double-exponential distribution, $\mathcal{B} = 2$ to a Gaussian or normal distribution, and $\mathcal{B} \to \infty$ to a uniform distribution. We refer the reader to [36] for a comprehensive treatment of the mathematical properties of the GGD statistical model.

4. Methodology

The first step of the proposed methodology is to represent the EEG signals in a time-frequency domain. This step allows decomposing each one of the EEG channels in the signal array into five frequency bands associated with the five brain rhythms that clinicians typically use to capture different types of neurological activity. We believe that this decomposition is very useful for SOD because there are rhythms or frequency patterns that appear often at the beginning of the epileptic crisis and that are difficult to quantify visually. We have used a time-frequency representation associated with the following bands: Delta: 0.5-4Hz, Theta: 4-8Hz, Alpha: 8-13Hz, Beta: 13-30Hz and Gamma: 30-45Hz. In order to achieve this we used a multilevel 1-D wavelet decomposition filter constructed using a Dauchebies wavelet decomposition with five resolution scales. From the wavelet decomposition it is then possible to determine how the energy in the signal is spread over the different *brain rhythms*, and how this distribution of energy evolves over time. Precisely, this information is captured by modelling the signals associated each each brain rhythm with a GGD statistical model, whose parameters \mathcal{A} and \mathcal{B} are estimated from the observed data, see equation (1). The shape parameter \mathcal{A} is closely related to the variability of the brain activity and is therefore a good descriptor for performing SOD detection.

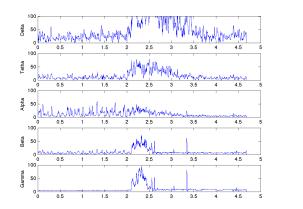
We evaluated the performance of the proposed seizure onset detector and spread estimation algorithms by using the Children's Hospital Boston database [45], which consists of 36 EEG recordings from paediatric subjects with intractable seizures. In this work we used 30 crisis from 11 subjects. A set including two to five bipolar EEG recordings sampled at 256Hz were available for each subject. Each recording contained a seizure event with a labeled onset that was detected by an experienced neurologist, who worked backward from the observed clinical onset to find the electrographic onset. Each signal was edited to have an epoch with the following characteristics: 2 minutes before crisis, crisis at minute 2 and 2 minutes after crisis.

For each epoch we know where the crisis begins and can calculate the onset delay by comparing the average amplitude with the background, similar to [16]. Once onset delay estimation has been performed for each brain rhythm and each channel (by performing SOD with the respective parameter \mathcal{A}), we collect this information in a table that allows identifying channels with low delay and thus understanding how the seizure originated and propagated. This is illustrated in Figures (1) to (6) and Table (1). The proposed algorithm can be summarised as follows:

- (i) Separate each EEG signal in the array of signals into the different physiological brain frequency bands.
- (ii) Estimate the GGD parameters \mathcal{A} and \mathcal{B} , see eq. (1).
- (iii) Use the parameter A to calculate the delays of each SOD for each channel and each frequency bands.
- (iv) Create a table with the information and calculate each delay for each channel by frequency bands.
- (v) Organize the table ascendently.
- (vi) Calculate the SOD candidate channels for the different brain areas (i.e. Frontal, Parietal, Temporal and Occipital).
- (vii) The candidates for the onset are the channels that are common to all other channels by frequency band in a determined brain area; and the other channels are the possible spread accompanying the crisis.
- (viii) Repeat these steps for all the rhythms bands.

Table 1. Onset Delay (in seconds) by Frequency Bands, the symbol (-) means that there is no clear difference in the beginning of the onset. In this example, according to the data, the channels (13) FP2-F8, (14) F8-T8 and (15) T8-P8 are the best candidates by brain area for SOD because it have minor delays; in this case F8 is common to all channels in this brain area, therefore is the best option for be the onset and the other channels form the spread.

1						T
Channel Number	Channel Name	Delta	Theta	Alpha	Beta	Gamma
1	FP1-F7	-0.099	.001	.002	.13	.131
2	F7-T7	-	.069	.234	.131	.058
3	T7-P7	-	.037	.215	.369	.371
4	P7-O1	-	-	-	.224	.162
5	FP1-F3	-	.037	.011	.133	.133
6	F3-C3	-	-	-	.221	.132
7	C3-P3	-	-	-	.378	.14
8	P3-O1	-	-	-	.534	.552
9	FP2-F4	.083	.06	.037	.212	.098
10	F4-C4	-	.001	-	.125	.110
11	C4-P4	-	.018	.029	-	.103
12	P4-O2	-	.035	-	-	-
13	FP2-F8	.105	.012	.025	.021	.101
14	F8-T8	.073	.087	.134	.097	.1
15	T8-P8	.081	.018	.013	.099	.103
16	P8-O2	-	.036	.016	.253	.086
17	FZ-CZ	-	-	-	.034	.141
18	CZ-PZ	-	-	-	-	-
19	P7-T7	.086	.085	.04	2.36	.35
20	T7-FT9	-	.069	-	.372	.377
21	FT9-FT10	.005	.001	.016	.098	.117
22	FT10-T8	.075	.066	.01	.102	.101
23	T8-P8	.065	.032	.015	.098	.117



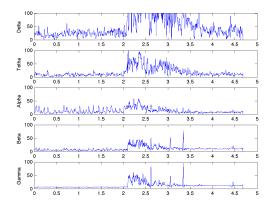


Figure 1. \mathcal{A} for Channel 13.

Figure 2. \mathcal{A} for Channel 14.

No distinctions regarding the types of seizure onsets were considered; the data contains focal, lateral, and generalized seizure onsets. Furthermore, the recordings were made in a

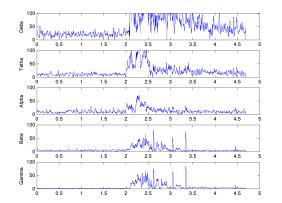


Figure 3. \mathcal{A} for Channel 15.

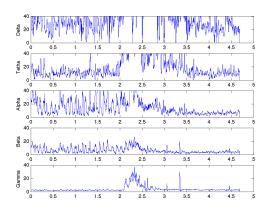


Figure 5. \mathcal{A} for Channel 10.

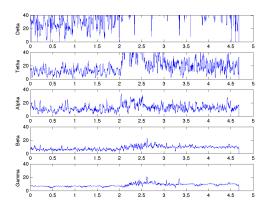


Figure 4. \mathcal{A} for Channel 16.

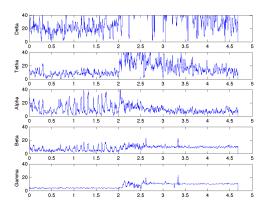


Figure 6. \mathcal{A} for Channel 11.

routine clinical environment, so non-seizure activity and artifacts such as head/body movement, chewing, blinking, early stages of sleep, and electrode pops/movement are present in the data. The set of recordings lasted on average 35 minutes for 30 subjects in total; 2 hours for 4 subjects; and 12 hours for 2 other subjects. Taken together the recordings account for 60 hours of EEG recordings and 139 seizures [45].

5. Results

The performance of the algorithm proposed for seizure onset detector and spread estimation was assessed using the Children's Hospital Boston database described above, and by comparing results with those obtained by visual inspection by an experimented neurologist relying on EEG and MRI data.

For illustration figure (8) shows the EEG and MRI image for one patient; this information allows the reconstruction of a model of the brain and the determination of the location where the seizure was originated, as well as how is spread throughout the brain, which allows is the information that our algorithm seeks to estimate directly from the EEG.

In this SOD example, we can see the \mathcal{A} parameter in the x-axis, se Figures (1) to (6), which correspond to right temporal brain area. Our algorithm shows that the seizure starts in channel F8 and then spreads across the channels F8-T8-P8, F4-C4-P4, as is shown the data in table (1)

and Figure (7).

Channels with the minor delay in the right temporal brain area: (13) FP2-F8, (15) T8-P8, (22) FT10-T8, (14) F8-T8, (12) P4-O2.

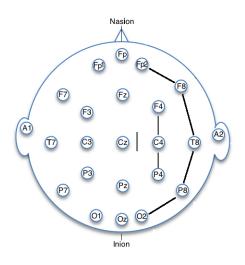


Figure 7. EEG electrodes Array, shows that the seizure starts in channel F8 and then spreads across the channels F8-T8-P8, F4-C4-P4

Spread by Delta Bands: (10) F4-C4, (21) FT9-FT10, (11) C4-P4, (23) T8-P8, (14) F8-T8, (22) FT10-T8, (15) T8-P8, (9) FP2-F4, (19) P7-T7, (1) FP1-F7, (13) FP2-F8.

Spread by Theta Bands: (1) FP1-F7, (21) FT9-FT10, (13) FP2-F8, (15) T8-P8, (11) C4-P4, (23) T8-P8, (16) P8-O2, (3) T7-P7, (5) FP1-F3, (9) FP2-F4, (22) FT10-T8, (2) F7-T7, (20) T7-FT9, (19) P7-T7, (14) F8-T8.

Spread by Alpha Bands: (1) FP1-F7, (22) FT10-T8, (5) FP1-F3, (15) T8-P8, (23) T8-P8, (16) P8-O2, (21) FT9-FT10, (13) FP2-F8, (12) P4-O2, (9) FP2-F4, (19) P7-T7, (14) F8-T8, (3) T7-P7, (2) F7-T7.

Spread by Beta Bands: (13) FP2-F8, (17) FZ-CZ, (14) F8-T8, (21) FT9-FT10, (23) T8-P8, (15) T8-P8, (22) FT10-T8, (10) F4-C4, (1) FP1-F7, (2) F7-T7, (5) FP1-F3, (9) FP2-F4, (6) F3-C3, (4) P7-O1, (16) P8-O2, (3) T7-P7, (20) T7-FT9, (7) C3-P3, (8) P3-O1, 19 P7-T7.

Spread by Gamma Bands: (2) F7-T7, (16) P8-O2, (9) FP2-F4, (14) F8-T8, (13) FP2-F8, (22) FT10-T8, (11) C4-P4, (15) T8-P8, (10) F4-C4, (21) FT9-FT10, (23) T8-P8, (1) FP1-F7, (6) F3-C3, (5) FP1-F3, (7) C3-P3, (17) FZ-CZ, (4) P7-O1, (19) P7-T7, (3) T7-P7, 20 T7-FT9, (8) P3-O1.

6. Discussion

The preliminary results reported in this work suggest that the proposed algorithm is potentially useful for onset detection and spread estimation, and that performing the analysis at the level of the brain activity rhythm bands can improve the identification of the area of the brain affected. This suggests that the algorithm is potentially interesting for devising automatic processing systems.

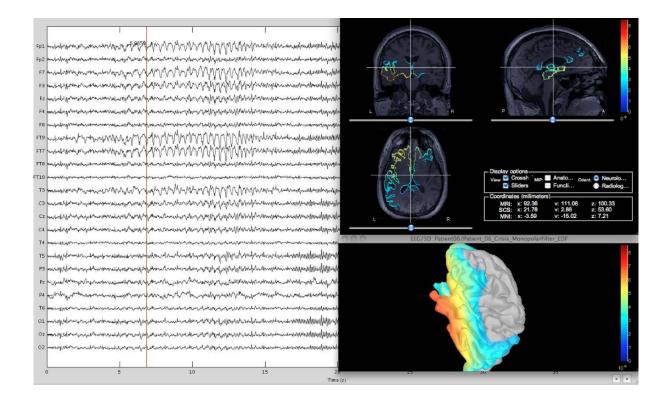


Figure 8. For illustration, the figure shows the EEG of a patient (left) with his corresponding MRI (right top). A reconstruction of the brain is shown with his corresponding brain spread (left bottom). On the right of each image is a color map which indicates the intensity of each sour activation.

Another interesting research questions to explore in more depths, are related with the analysis of the frequency bands detected before the onset seizure, and which channels are active despite not participating directly in the detection of the seizure onset.

Perspective for future work include an extensive evaluation of the proposed methodology, as well as performing comparisons with other detection methods from the state of the art, and the development of fusion techniques to combine detections from several algorithms to increase robustness to noise and to artefacts.

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