SEIZURE ANALYSIS OF NEWBORN EEG USING A MODEL BASED APPROACH

Mark Roessgen and Boualem Boashash

Signal Processing research Centre, QUT
GPO Box 2434, Brisbane, Q. 4001, Australia
m.roessgen@qut.edu.au

ABSTRACT

This paper considers the problem of seizure detection in the neonate based on electroencephalogram (EEG) data. It will be shown that by using a histologically and biophysically justifiable model for the generation of the EEG, the detection of electrographic seizure is greatly improved. The model is presented along with an estimator for the model parameters. Then a simple seizure detection scheme based on the model parameter estimates is suggested. It is shown that this scheme is superior in performance to spectral analysis techniques such as the periodogram when used to analyse both simulated and real EEG data.

1. INTRODUCTION

The electroencephalogram (EEG) plays an important role in the assessment of various neonatal or newborn neurological disorders. Its current principal application is in the diagnosis and prognosis of newborns with seizures [1]. It is important to detect seizures and determine their precise aetiology as early as possible in order to administer the correct treatment and therapy. This will ensure that further long term brain injury is minimised. However, this is often difficult as the manifestation of seizure within the EEG is highly variable.

Seizure is an episodic event with a definite beginning and termination. The EEG during this period is termed the ictal EEG. Due to its episodic nature, assessment of the EEG often involves the examination of the between seizure EEG, termed the interictal EEG. Transient EEG behaviour such as spikes and sharp waves are a specific indication of interictal EEG associated with an epileptic process in children and adults. Clancy [2] reports that the situation is far more complex in neonates, where sharp EEG transients (SETS) such as spikes and sharp waves, are present in neurologically healthy newborns as well. This makes seizure detection in the neonate a difficult task as SETS associated with normal EEG must be discriminated from similar waveforms associated with the abnormal case.

In this paper a model based approach for the detection of seizure in the neonate is presented. The model used is based on the histology and biophysics of a localised portion of the brain and as such its parameters can be explicitly related to the mechanisms causing seizure. A simple classification procedure based on two of the model parameters is first suggested for seizure detection. It will be shown that this method offers superior detection performance of low frequency seizure, when compared with spectral analysis techniques such as the periodogram or autoregressive moving average (ARMA) model based techniques.

2. NEONATAL SEIZURE

A seizure occurs when there is an excessive synchronous discharge of neurons within the central nervous system [1]. This manifestation in the EEG, known as electrographic seizure, consists of paroxysmal events which are best described as stereotyped repetitive waveforms that evolve in amplitude and frequency before eventually decaying [2]. In contrast, the normal background EEG consists of irregular activity with no clear periodicity, and occasionally containing SETS (see Fig. 4).

The rate of repetition of the characteristic seizure waveform is not constrained to any specific frequency range. Similarly the amplitude of the signal may be greater or smaller than that of the normal background EEG. Because of this, a simple spectral amplitude and frequency criterion for detection is inadequate, often missing events and giving rise to false alarms. This is confirmed in [3] where significant spectral overlap between normal background EEG and seizure EEG was reported.

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3. THE EEG MODEL

The model for the generation of the EEG studied in this paper is based on that originally proposed by Lopes Da Silva et al [4]. It considers the neurophysiology of a localised portion of the outer cortex of the brain. Its parameters reflect the number of excitatory as well as inhibitory neurons, their interconnectivity, and the characteristics of the corresponding post-synaptic potentials, as well as firing densities (see [4] for more details). The model considered in this paper, is shown in Fig. 1.

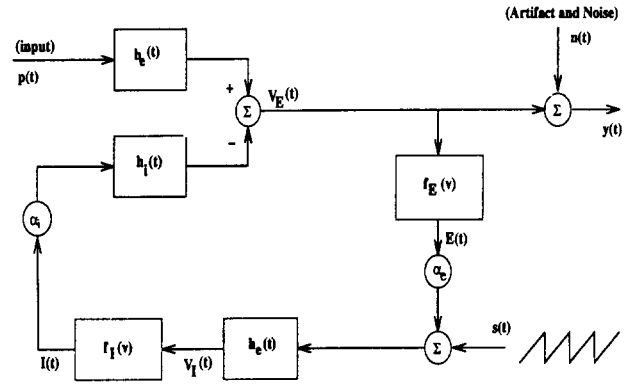

Figure 1. Model for seizure showing EEG, artifact and noise.

In this model the notation e and i refers to excitatory and inhibitory respectively. The EEG, $V_E(t)$, is given by a linear feedback system whose input driving process $p(t)$ is assumed to be stationary white Gaussian noise (WGN) with zero mean and variance σ^2. The post-synaptic potentials, $h_e(.)$ and $h_i(.)$, are modelled by the difference of two decaying exponentials

$$\begin{aligned} h_e(t) &= A\left(\exp(-a_1 t) - \exp(-a_2 t)\right) \\ h_i(t) &= B\left(\exp(-b_1 t) - \exp(-b_2 t)\right) \end{aligned} \quad (1)$$

where the a_i's and b_i's control the shape of the pulses. The functions $f_E(.)$ and $f_I(.)$ are static threshold functions which relate the average level of the membrane potential to the pulse density. It is assumed they are in their linear region of operation such that $f_E(V) \approx a_{e1}V$ and $f_I(V) \approx a_{i1}V$. The α_e and α_i multipliers relate the interconnectivity of the excitatory and inhibitory neurons.

In this paper the original model as proposed in [4] has been extended to account for seizure activity through the use of a second input waveform $s(t)$. Since seizure involves the synchronous depolarisation of many neurons, we have chosen to model the seizure by a periodic waveform driving the excitatory neurons within the local mass of all neurons. The waveform suggested, $s(t)$, will be a saw-tooth waveform parametrised by its peak-peak amplitude, period and DC level. One period of the signal $s(t)$ is given by

$$f(t) = \begin{cases} C(2t/T_s + D - 1) & 0 < t < T_s \\ 0 & \text{otherwise} \end{cases} \quad (2)$$

where T_s is the period, C is the amplitude and D is the DC level. Although other waveforms are equally possible candidates, the saw-tooth was considered for its simplicity and its applicability to modelling mass depolarisation of neurons with slow repolarisation rate. This scenario is hypothesised to occur during seizure.

In order to estimate the model parameters using the technique to be described in the next section, the discrete time spectrum of the EEG, $S_{V_E V_E}(\lambda)$, is required. Using pre-warping and the bilinear transform [5]

$$\begin{aligned} S_{V_E V_E}(\lambda) = &\left| \frac{A\sigma(a_2 - a_1)(s + b_1)(s + b_2)}{d(s) + (a_2 - a_1)(b_2 - b_1)K_1} \right|^2_{s=\frac{2(1-e^{-j\lambda})}{T(1+e^{-j\lambda})}} \\ &+ \left| \frac{K_2(a_2 - a_1)(b_2 - b_1)H(s)}{d(s) + (a_2 - a_1)(b_2 - b_1)K_1} \right|^2_{s=\frac{2(1-e^{-j\lambda})}{T(1+e^{-j\lambda})}} \end{aligned} \quad (3)$$

where $K_1 = \alpha_e \alpha_i AB/(a_{i1}a_{e1})$, $K_2 = \alpha_i ABC/a_{i1}$, T is the sample period, $H(s) = \frac{(D-1)}{s} + \frac{2}{T_s s^2} - \frac{2e^{-T_s s}}{s(1-e^{-T_s s})}$ and $d(s) = (s + a_1)(s + a_2)(s + b_1)(s + b_2)$.

Defining $C_1 = A\sigma$ and $C_2 = \alpha_i ABC/a_{i1}$ the model parameter vector to be estimated is given by $\theta_E = [a_1, a_2, b_1, b_2, K, C_1, C_2, T_s, D]$.

The described EEG model is both histologically and biophysically justifiable as well as parsimonious with its use of parameters. The estimates of these parameter values from the EEG will be used for seizure diagnosis.

4. ESTIMATION OF THE PARAMETERS

The aim is to estimate the EEG model parameters from a record of EEG in order to detect for seizure. The maximum likelihood (ML) approach is preferred [6]. However, the analytic expression for the log-likelihood function of the model is extremely cumbersome, making it difficult to explicitly derive the estimators. Alternatively it is proposed to maximise an asymptotically close approximation to the log-likelihood function. Known as Whittle's approximation, this approximate log-likelihood is more manageable and the estimator derived from its maximum should retain all the advantages of the ML estimator [6].

Suppose the observed EEG data are corrupted by independent additive noise, $n(t)$, such that $y(t) = V_E(t) + n(t)$, here $n(t)$ is assumed to be stationary zero mean WGN with variance σ_n^2. Given the observations $\mathbf{Y} = [y(1), \cdots, y(N)]$ Whittle's approximation to the log-

likelihood function for $\mathbf{Y}$ is written:

$$\tilde{L}_N\{\mathbf{Y}\} = -\frac{N}{4\pi}\int_{-\pi}^{\pi}[\log(2\pi)^2\{S_{V_E V_E}(\lambda)+\sigma_n^2\} + \frac{I_{\mathbf{Y}}(\lambda)}{S_{V_E V_E}(\lambda)+\sigma_n^2}]d\lambda \quad (4)$$

where $I_{\mathbf{Y}}(\lambda)$ is the periodogram of $\mathbf{Y}$.

Defining the parameter vector to be estimated as $\boldsymbol{\theta} = [a_1, a_2, b_1, b_2, K, C_1, C_2, T_s, D, \sigma_n]$, an approximate ML estimate $\hat{\boldsymbol{\theta}}$ of the parameter vector $\boldsymbol{\theta}$ is then:

$$\hat{\boldsymbol{\theta}} = \underset{\boldsymbol{\theta}}{\operatorname{argmax}}(\tilde{L}_N\{\mathbf{Y};\boldsymbol{\theta}\}, \theta \in \Theta) \quad (5)$$

where Θ is the parameter space of physically reasonable values. The maximisation will be achieved using an iterative numerical optimization procedure based on work by Powell [7].

5. EXPERIMENTS AND DISCUSSION

Monte Carlo simulations were performed where 2000 realisations of 512 points of simulated background EEG data with no seizure component i.e. $s(t) = 0$, were generated with a known parameter vector. The model parameters were then estimated using the above approach. It was found that given a good initial parameter vector, the method worked well. However when the initial starting vector was a far from that corresponding to the global maximum, the numerical maximisation routine often converged to a local maximum giving poor results.

The method was next used to analyse two specific records of 512 points of simulated EEG data. The control or normal EEG had the parameter vector

$$\boldsymbol{\theta}_c = [2, 400, 15, 20, 500, 30, 0, 0, 0, 0.1]$$

and contained no seizure, the seizure EEG had the parameter vector

$$\boldsymbol{\theta}_s = [2, 400, 15, 20, 500, 30, 900, 0.96, 0, 0.1].$$

The two time traces are displayed in Fig. 2. Taking into account the patient history, the EEG on other channels as well as the surrounding EEG, the neurologist would make as objective a judgement as possible of these two records.

Welch's averaged periodogram with a window length of 196 data points and an overlap of 66%, was then used to analyse both records (Fig. 3). Although the spectral peak associated with the seizure data has a larger magnitude than that associated with the control, without further analysis it is not possible to make an objective distinction between the two. This is because there is significant spectral overlap between the control and seizure EEG. Spectral analysis using ARMA based techniques would give similar results.

Using the model based method, the two records were analysed. From the results in Table 1 it is suggested that the magnitude of the ratio of the parameters C_2 and C_1 be used for detection. From the model, it is to be expected that when C_2 is large with respect to C_1 seizure is likely to be present. Since for the control, $C_2/C_1 = 0$, and for the seizure $C_2/C_1 = 32.5$, it may be concluded in the latter that the likelihood of seizure having occurred is large.

Real records of EEG data recorded from a neonate were then analysed. The first record (which was normal), was chosen to be the control, while the second was chosen from a portion undergoing seizure as scored by a trained neurologist (Fig. 4). Although clear repetitive waveforms are present in the seizure EEG, periodogram analysis reveals very little of this when compared with the control EEG (Fig. 5). The control EEG is of a much larger magnitude than the seizure EEG, and also there is significant spectral overlap.

Table 2. presents the results of analysing the data using the method described above. Although convergence of the maximisation routine to the global maximum could not be guaranteed, choosing multiple starting points helps to achieve this. The results show that for the control EEG $C_2/C_1 = 0.51$ and for the seizure record $C_2/C_1 = 12.03$. The large magnitude of the latter ratio indicates the presence of seizure.

The suggested ratio of the two parameters, C_2 and C_1, for detection is suboptimal for classification but was suggested as a first approach. Further research into classification schemes based on the complete parameter set is necessary to improve upon this.

6. CONCLUSIONS

In this paper a new approach to the analysis of the neonatal EEG for seizure was presented. The technique involved performing detection based on estimating the parameters of a physiologically valid model.

It was shown that for low-frequency seizures this approach was superior to spectral approaches such as the periodogram. This is because the parameters from the model can be explicitly related to the mechanisms causing the seizure. The ratio of two of the model parameters was suggested as a first approach for seizure detection. Further research is needed for optimal detection based on the complete model parameter set. This method was presented for the case of neonatal EEG but is equally applicable to adult EEG as well.

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7. REFERENCES

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Parameter	a_1	a_2	b_1	b_2	K
Control EEG	9	169.7	9.64	10.86	276
Seizure EEG	3.52	197	14.7	19.832	428.3
Parameter	C_1	C_2	T_s	D	σ_n^2
Control EEG	37	0.0	0	0	0.0001
Seizure EEG	32	1041	0.98	0.12	0.0002

Table 1.

Parameter	a_1	a_2	b_1	b_2	K
Control EEG	0.016	159.9	8.8	12.5	143.6
Seizure EEG	3.358	10	15.7	1164.1	243.6
Parameter	C_1	C_2	T_s	D	σ_n^2
Control EEG	18.8.	9.5	3.62	2.91	2e-5
Seizure EEG	15.3	183.8	1.419	0.024	0.18

Table 2.

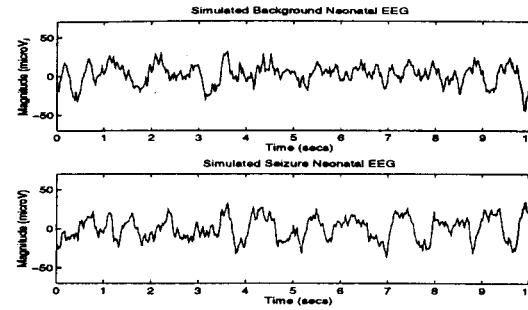

Figure 2. Simulated control and seizure data records.

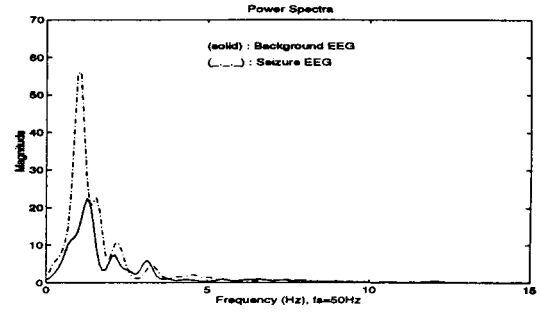

Figure 3. Spectra of simulated control and seizure records.

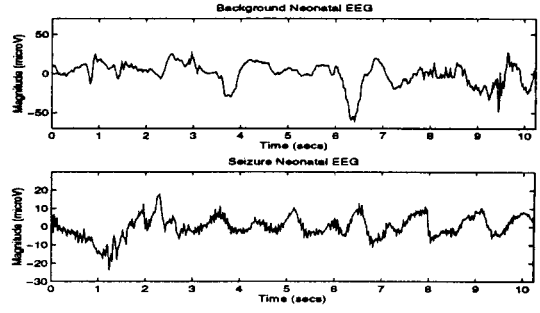

Figure 4. Control and seizure EEG records.

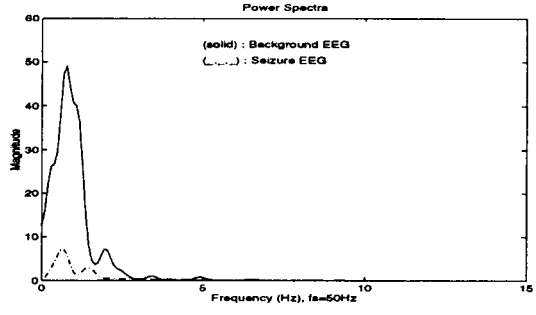

Figure 5. Spectra of control and EEG records.