Some Theoretical Investigations in EEG Studies

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Abstract

It is well known that Electroencephalography (EEG) and the respective evoked potentials have deep implications corresponding to specific cognitive tasks and in the diagnosis of several diseases such as epilepsy and schizophrenia. Some recent experimental results have already shown some evidence of chaotic activity in the brain. The Hodgekin-Huxley (HH) models, may yield geometrical solutions in terms of limit cycles and basins of attractors, but its implementation requires a priori knowledge of the kinetics of the innumerable conductances acting in a given set of cells. We are of the opinion that the EEG data should reflect the neuronal dynamics, and there should be some mechanism at the neuronal level which generates stochasticity compatible with the recorded data. In this paper we develop a theoretical framework to show that EEG dynamics may be governed by a suitably biased Vander-Pol oscillator which is closely related with the modified version of the FitzHugh-Nagumo (FN) model making extension of the ideas of dynamic causal modelling (DCM). Eventually we also give a prescription to compute the correlation matrices which may be tested empirically, for some small values of the parameters.

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1 Introduction

That the human brain is a complex system with significant spatiotemporal dynamics is beyond doubt. EEG, a noninvasive technique for probing the dynamics of brain provides a direct measure of cortical activity with millisecond temporal resolution. It should be mentioned here that EEG data is a set of changes in voltage potential [1, 2] which is generated when neuronal activities are activated. From the functional point of view EEG studies have been engaged in a variety of brain activities including cognitive tasks, showing the memory contents which are activated showing the information processing operations, but most abundantly, or to say clinically the research has remained confined [3] with the analysis of several epileptic and schizophrenic patients with comparative EEG analysis. Recently some studies have also been initiated [4] in analyzing the emotional states of patients by studying brain electric fields from the EEG data.

For the analysis of EEG data, representations based on a Fourier transform have been most commonly applied. These methods have proved to be effective for various EEG characterizations, but fast Fourier transform (FFT), suffer from large noise sensitivity. Another approach based on neural network detection systems have been proposed [5, 6] but with a false detection rate. Interestingly [7] have evaluated different parametric models on a fairly large database of EEG segments. Using inverse filtering, white noise tests, and 1-s EEG segments, they found that autoregressive (AR) models of orders between 2 and 32 yielded the best EEG estimation. Recent techniques include methods measuring chaos theory and the energy measure. The measure of complexity used in chaos include parameters like correlation dimension, similarity, synchrony and Lyapunov exponent. It has been overemphasized over the years to find global solutions to cable equations which represent long wavelength EEG standing waves. It should be mentioned here that a nonlinear component is apparent in all analyzed EEG records [8, 9, 10]. The time series obtained by theoretical investigations [11] belonged to four classes, suggesting a chaotic activity with correlation of 5.5. EEG was characterized by inspiratory bursts of oscillations that disappeared during expiration and simulations suggested that this state corresponds to a limit cycle attractor that is specific to a given stimulus.

EEG’s represent the integral output of a large number of neurons, with a complicated dynamics of subsystems with innumerable degrees of freedom. In addition the presence of noise of unknown origin makes it hopeless to reinterpret the data within the framework of chaos theory. Despite this difficulties epilepsy, one of the cherished arenas of investigation of EEG remained to be a recognized model of neuronal synchronization and it is now widely believed [12] that seizure episodes are characterized by bifurcations to system states of low complexity. To cite an example, epileptic bursts produced in CA3 region of the rat hippocampal slices are exposed to $K^+$ enriched extracellular medium by electrical simulation of the mossy fiber inputs
Time series of the evoked field potentials were analyzed and the conclusion was that of undoubted evidence for chaos. Dimensional analysis of an epileptic EEG again provided a hypothesis of an existence of chaotic attractor being the direct consequence of the deterministic nature of brain activity. So by now it can be well inferred that EEG recordings which are the marker for the neuronal activity which show many characteristics of chaotic activity.

Taking all these into consideration we in this paper are mainly concerned to show whether there exist any mathematical model to fit into these scheme of things in explaining EEG phenomena. It should be admitted here that we have not done any numerical simulation, but mainly followed a mathematical formalism to show whether the desired response of the EEG phenomena may be generated using this mathematical structure. We have tried to investigate the FitzHugh-Nagumo(FN) model though not in the original form. The FN model is based on the premise that changes in the membrane potential is related to sodium activation, inactivation and potassium activation. It is a two variable model, but in essence with our early comments this model unlike HH models is unable to generate chaos. Though it should be mentioned here that some modifications to the FN model has already been done giving rise to Hindmarsh-Rose (HR) model, which is essentially a 3 dimensional model which has the ability to generate chaos.

The non linearity of the active membrane, the brain’s high degree inhomogeneity makes the electric field in the brain difficultly complicated. Our approach is mainly based on the fact that EEG data is measured as the difference in electric potential between one and more other electrodes given by

$$\Phi(\vec{r}, t) = \frac{1}{4\pi\sigma} \Sigma_{i=1}^{n} \frac{I_i(t)}{R_i}$$

where $I_i(t)$ is the fluctuating current moving from the $i$’th current source into a medium of conductivity $\sigma$. $R_i$ is the distance of the $i$’th source from the field point $\vec{r}$. The potential difference $V$ across the membrane follows the diffusion equation given by

$$\lambda^2 \frac{\partial^2 \Phi}{\partial \vec{r}^2} - \tau \frac{\partial \Phi}{\partial t} - \Phi = J$$

where $\lambda$ is the space constant of the axon, which is determined by the electrical and geometrical properties of the axon and the surrounding nerve cells, $\tau$ is the time constant of the membrane and $J$ is the current source term. Some experiments have already been done for some simple systems which show that if we take, say two neurons with both excitatory and inhibitory synaptic activity the potential measured at an intermediate position (assuming dipole currents with fluctuating frequencies $f_a, f_b$) and the temporal component can be written as

$$\Phi(t) = \frac{1}{4\pi\sigma} \left[ \frac{I_a(t)}{R_1} \cos(2\pi f_a t + \alpha_a) - \frac{I_a(t)}{R_2} \cos(2\pi f_a t + \alpha_a) + \frac{I_b(t)}{R_3} \cos(2\pi f_b t + \alpha_b) - \frac{I_b(t)}{R_4} \cos(2\pi f_b t + \alpha_b) \right]$$
So essentially we see that EEG phenomena is a form of some disturbance, or mainly a wave phenomena which may or may not be stochastic in nature. The reason which motivated us to write an article on the analysis of EEG is crucially based on the observation that the EEG potential may be regarded as a function of surface coordinates, which may give rise to stochasticity due to the intrinsic network dynamics and may be represented as a superposition of a number of travelling waves, given by

$$\Psi(t, x, y) = \sum_{l} \sum_{m} \sum_{n} C_{l,m,n} \sin(2\pi f_{n} t - k_{x} x - k_{y} y)$$ (4)

which can be argued to be analogous to Eq. (3) if we include the spatial degrees of freedom.

So the goal of this paper is to identify an underlying scheme of events which may play a crucial role in determining the EEG patterns as depicted above and we will try to show how nonlinearity may be fitted in those models. In the next section we discuss about some general considerations on EEG and a brief review of the existing models. In section 3 we show the analogy of a simple nonlinear oscillator, the biased Vander-Pol, with some extra parameters, which is proposed to be a good candidate as the FN model of neuronal behavior and show with the help of Langevin formalism, that an equivalent Fokker-Plank (FP) equation corresponding to it may be developed and show how to define the EEG potential through the probability distribution and define some measurable quantities which may have the required stochastic response to be fitted into the recently made experiments of EEG data.

## 2 Some notes on EEG and it’s extensions

As we have already stated in the introduction in general all EEG phenomena have a temporal behavior with different categories. A spectral analysis may be done on any one of the categories. To be a bit quantitative a sinusoidal function $\Psi(t)$ is completely described by three parameters as $\Psi(t) = C \sin(2\pi ft + \phi)$ and in general the epoch will be a superposition of the form

$$\Psi(t) = \Sigma_{n}^{k} C_{n} \sin(2\pi f_{n} t + \phi_{n})$$

The spectral density function $Q(f)$, a measure of the $C^{2}_{n}$ is obtained as a Fast Fourier Transform (FFT) with the following prescription

$$Q(f) = \frac{2\Delta t}{N} \| \sum_{l=0}^{N-1} \Psi_{l} exp(-i2\pi lf \Delta t) \|^{2}$$ (5)

where $\Psi_{l}$ refers to the EEG sampled at times given at discrete time intervals $l$. Similarly we can define the cross spectral density functions for two channels of EEG,

$$Q(f) = \frac{2\Delta t}{N} \left[ \sum_{l=0}^{N-1} \Psi_{1l} exp(-i2\pi lf \Delta t) \sum_{l=0}^{N-1} \Psi_{2l} exp(-i2\pi lf \Delta t) \right]$$ (6)
to get a coherence function which will give us a measure of correlation between the signals. Now if we include the spatial coordinates where the basic epoch of EEG is given by Eqn. (4), then the covariance matrix consisting of $M$ epochs of EEG from the cross spectral density functions Eqn. (5) of each epoch may be written as

$$q_{rs}(f) = \frac{1}{M} \sum_{l=1}^{M} Q_{rs}^l(f)$$

where $r, s$ are as usual channel numbers and $l$ is the epoch number. The frequency wavenumber spectrum, $T(f, k_x, k_y)$ a measure of $C_{lmn}$ can be obtained from the covariance matrix. So the basic scheme is that simple, analyze the EEG for say two cases, one normal and another epileptic, find out the spectral density in both cases, analyze the results, make a statistical inference and go out clinically. But the actual point here is that as far as the comparison is concerned at a particular scale it may be fine, but does the analysis take into account the real dynamical features of the brain. In other words the basic epochs which we have considered may be constructed assuming a basic circuit model which generate a travelling wave solution as is the case in Eqn. (2). What we are more importantly concerned over here is to take into account the recent experimental results [17, 18, 19] and to propose an underlying dynamics for EEG phenomena. Recent models of EEG studies [20] has given place to fMRI analysis which are executed as a general linear model

$$y = X\beta + \eta$$

which measures the experimentally controlled investigations to the observed blood oxygen level dependent responses (BOLD) [21], $y$ in a voxel specific fashion with the design matrix $X$ and a gaussian noise $\eta$. There has been extensions of this idea in terms of Multivariate autoregressive models (MAR) [22], modelling the vector of regional BOLD signals at time $t(y_t)$ as a linear combination of $k$ past data vectors, with an weighted contribution of the parameter matrices $A_i$ and a noise term $\eta$

$$y_t = \sum_{i=1}^{k} y_{t-i}A_i + \eta_t \quad (7)$$

The drawback of the MAR models is that they do not involve biophysical forward terms, which enable inferences about neural parameters. This model has been extended by DCM [23, 24], which constructs a reasonably realistic neuronal model of interacting cortical regions with neurophysiologically meaningful parameters. In DCM, neural dynamics in several regions, represented by a neural state vector $z$ are driven by experimentally designed inputs. In DCM the change in neural states is a non linear function of the states ($z$), the inputs ($u$), and the neuronal parameters ($\theta^n$), which are the connectivity matrices defining the functional architecture and interactions among brain regions at a neuronal level. The general equation may be written as

$$\dot{z} = F(z, u, \theta^n) \quad (8)$$
It should be noted here that the DCM model may be extended to include background noise which is correlated in space and time. We emphasize this point as we will proceed with this formalism in our case. It may be assumed that the recorded signal is a simple superposition of the brain response and the background noise which is the signal plus noise (SPN) model, where the measured signal $\Lambda'_{rs}$ at channel $r$ and time sample $s$ in trial $t$ is formulated as $\Lambda'_{rs} = \Lambda_{rs} + \eta'_{rs}$, where $\Lambda_{rs}$ is the brain response caused by the stimulus and $\eta'_{rs}$ is the measured noise, which is correlated in space and time as a covariance matrix $\Sigma = X \otimes T$, where $X, T$ are spatial and temporal covariance matrices respectively.

Some recent theories in interpreting the set of images in terms of visual signals corresponding to brain activities may be given by a Markov Random Field (MRF) [26], implying that probability of a pixel, assuming a particular value is dependent on the values of the neighbouring pixels. It is governed by a joint probability distribution

$$p(x) = \frac{1}{Z} \exp(-\beta U(x))$$

where $Z$ is the normalization constant, $\beta$ the inverse of temperature, $x$ is a vector denoting an array of pixel values and $U(x)$, the Potential. Once the choice of the potential is made the method of simulated annealing (SA) [27] may be used to employ a stochastic differential equation, which gives the dynamics of the image formation

$$dy(t) = -\nabla U(y) + \sqrt{\frac{2}{\beta}} dW$$

where $y$ denotes the image, $U$ the potential and $W$ the Brownian process. Now we have some points to make regarding the Eqn. (10) which will be pertinent for our purpose. The equation is essentially the Itô interpretation [28] of the standard Langevin equation

$$\dot{y} = A(y) + D(y)L(t)$$

We will be considering equations of the form (10) and to avoid the IS dilemma we will be interpreting the noise as an external one which is essentially created in an otherwise deterministic system (though may not be the case with brain, but at least as of now we don’t have a choice) by a random force whose stochastic properties are assumed to be known.

3 Biased Vander-Pol Equation as an aid to understand EEG

It is beyond doubt that a number of cellular and combined cellular network mechanisms generate collective behavior of neurons. Cells which may not be intrinsically oscillatory may become so as a consequence of network properties [29]. As there are multiple interacting levels of
organizational hierarchy in the human brain there are suitable forms of cooperativity and synchronization. It is a possible gesture that the mean field dynamics \cite{30} may give us an useful insight into the proper realm of things. So in our objective for a proper understanding of the dynamics which make the brain visible through EEG, which is the analysis of brain waves we first of all postulate that that the EEG is represented by the following expansion

$$
\Psi(t, x, y) = \sum_{n} \sum_{l} C_{nl} E[H_{nl}(x, y)] e^{(i2\pi f_{nl}t)}
$$

Here $H_{nl}(x, y)$ are the empirical orthogonal functions which reflect the spatial properties corresponding to the n’th temporal frequency component of the EEG and $M$ denotes the number of EEG channels. It should be mentioned here that $E[H_{nl}(x, y)]$ is the expectation value of the functions with respect to a particular distribution function. As we will see shortly that the distribution function arises as a solution to a stochastic differential equation which as we understand should be governed by the underlying cortical dynamics. The functions $H_{nl}(x, y)$ are the eigenvectors of the covariance matrix $q_{rs}(f_{n})$ and the coefficients $C_{nl}$ can be related to it’s eigenvalues. Again the estimate for the frequency-wavenumber spectrum $T(f, k_{x}, k_{y})$ may be obtained by giving the following definition

$$
T(f, k_{x}, k_{y}) = \frac{1}{M^2} \sum_{r=1}^{M} \sum_{s=1}^{M} q_{rs}(f) E[H_{nl}(x, y)]
$$

So essentially in understanding and interpreting the electric fields or EEG data Eqn. (13) tells us all. The covariance matrix is the matrix formed by the cross spectral density functions and contains complex numbers as they contain the relative phase of EEG. So this part is purely a part of the experimental data, but the other part is dependent on the underlying mechanics and cortical activity. The better the guesswork made in devising a cortical model we will be able to get a better fit.

Now to analyze the dynamics of the cortical network which may give rise to oscillations we consider a particular generalization of the Van der Pol (VP)oscillator. It is well known that VP models are special case of the Lienard equations

$$
\ddot{x} + f(x)\dot{x} + g(x) = 0
$$

which with some stringent conditions \cite{31} on the functions $f, g$ may lead to unique, stable limit cycle surrounding the origin in the phase plane. The VP equation in it’s original form

$$
\ddot{x} + \mu(x^2 - 1)\dot{x} + x = 0
$$

has a degenerate Hopf bifurcation at $\mu = 0$, which implies the vanishing of the nonlinear term, but if we scale the variables as $z = \mu^{1/2}x$ we may be able to get a Hopf bifurcation without the vanishing of the nonlinear term. So it may be anticipated that the VP models may be used
to study bursting behavior of the neurons, which eventually leads to evoked spike trains which appears in phase space to undergo a transition from a steady state to a repetitive limit cycle via Hopf bifurcations \[32\]. So we extend the biased VP model as

\[
\ddot{x} + \mu(x^2 - 1)\dot{x} + \nu Z(x) = a + \Lambda(t)
\]  

(14)

Here the parameters \(\mu, \nu, a\) though independent of spatio-temporal variables, yet may depend on some other variables linked with the cortical architecture and function (for example if we take the neurons as point particles, they may be related to their field, mass etc), \(Z(x)\) a choice function and \(\Lambda(t)\) is a noise term. Now it is important to mention here that the above model may be implemented as a equivalent circuit with several components and storage devices which may be a sufficient criterion to generate chaos, by the Poincare-Bendixon theorem \[28\]. Now as has been already advocated \[33\] that Eqn. (14) can be related to a modified FN model which by the previous argument may give rise to chaotic behavior. It can be shown by the Lienard plane analysis that when \(\mu >> 1\) the system has a stable limit cycle for a critical value of the parameter \(a\), which show relaxation oscillations phenomena. But it should be be mentioned here that in the modified version of our VP model, for some suitable choice function we do propose the possibility of bifurcations. In this context to gain some more grounds into the proposal, we can’t resist the temptation of introducing another oscillator, a special case of the Forced Duffing (FD) oscillator with weak parameters as

\[
\ddot{x} + \dot{x} + x + \epsilon x^3 = F(t)
\]  

(15)

The above system may be interpreted as a weak perturbations of the harmonic oscillator and gives rise to saddle node bifurcations of cycles. But unfortunately we don’t find any relaxation processes associated with it and thereby could’t be guaranteed that it may serve as a viable neuronal architecture model. With this digression we would like to visit our modified VP model Eqn. (14). As in our formalism we have to compute the expectation values of the empirical orthogonal functions we would like to recast Eqn. (14) in the Langevin approach where we get an equivalent two dimensional system given by

\[
\dot{x} = f(x) + y \\
\dot{y} = -\gamma y + \epsilon \Omega(x, \epsilon y) + \Lambda(t) + a
\]  

(16)

with \(f(x) = (\gamma + \mu)x - \frac{4\mu x^3}{3}\). It should be noted here that \(\gamma\) is defined in terms of the given parameters in the modified VP equation and the \(\Omega\) is determined in terms of \(f(x)\) and \(Z(x)\), though an arbitrary \(\epsilon\) dependence may not be removed. Here we take \(\epsilon << 1\) which ensures the equivalence of the two systems up to \(O(\epsilon^2)\). We assume a non gaussian noise term, to have a viable chaotic interpretations and it is assumed to have the following properties

\[
< \Lambda(t) > = 0
\]
\[ <\Lambda(t_1)\Lambda(t_2)> = \vartheta \delta(t_1 - t_2) \]
\[ <\Lambda(t_1)\Lambda(t_2)\cdots\Lambda(t_m)> = \vartheta_m \delta(t_1 - t_2)\delta(t_1 - t_3)\cdots\delta(t_1 - t_m) \] (17)

for \( m \geq 1 \). It should be mentioned here that \( \vartheta_m \) for \( m = 2 \) determines the size of the fluctuating term and related to the thermal noise [34] in the problem. So essentially the noise term is essentially determined by the constants \( \vartheta_m \). To get the master equation for this system, if we consider a Poisson process \( Z(t) \) given by
\[ Z(t) = \int_0^t \Lambda(t')dt', \quad (t > 0) \]
which is characterized by the transition probability \( T_{\tau}(z) \) defined as the product of convolutions as
\[ \int e^{ikz}T_{\tau}(z)dz = \exp[\tau \sum_{m=1}^{\infty} \frac{(ik)^m}{m!} \vartheta_m] = \exp[\rho \tau \int (e^{ikz} - 1)w(z)dz] \] (18)
with \( \vartheta_m = \rho \int z^mw(z)dz \).

Now with all these definitions and some suitable restrictions on the noise function it could be seen that Eqn. (16) is equivalent to the FP equation given by
\[ \frac{\partial P(x, y, t)}{\partial t} = -y \frac{\partial f(x)P}{\partial x} + \frac{\partial (\gamma y + e\Omega x)P}{\partial y} + \rho \int w(\zeta)(P(x, y + \zeta x, t) - P(x, y, t))d\zeta \] (19)
with some suitable initial conditions on the Probability distribution dictated by the linearized version of the DCM Eqn. (8). So essentially with some approximations we have found out the stochastic differential equation determining the probability distribution of the underlying relaxation oscillator process driven by the modified VP equation, which may in some sense characterize the neuronal processes. So the task which remains in hand is to calculate the EEG and the frequency-wavenumber spectrum which requires the knowledge of the expectation values which is defined by
\[ E[H(x(t), y(t))] = \int \int H(x, y)P(x, y, t)dxdy \] (20)

It should be noted here that we may define the time correlation matrix of any quantity \( G(x, y) \) between any two time intervals by
\[ \chi(t) = \int_{t_1}^{t_2} d\zeta \delta G(x(t + \zeta), y(t + \zeta))\delta G(x(\zeta), y(\zeta))^T \]
where \( \delta G = G - E[G] \)

It is to be noted here that the Langevin formalism in one dimensional formulation with a color noise in the joint variables \( x, \xi \)

\[ \dot{x} = f(x) + \xi \]
\[ \dot{\xi} = -\gamma \xi + L(t) \] (21)
gives rise to a FP equation of the form
\[ \frac{\partial P}{\partial t} = \frac{\partial}{\partial x}f(x)P + \frac{1 - e^{-(1+\gamma)t}}{1 + \gamma} \frac{\partial^2 P}{\partial y^2} \] (22)
Corresponding to this, it is worthwhile to mention that an action functional has been proposed in some input \( i(x) \) output \( o(x) \) variables as

\[
S[o(x), i(x)] = j \int dt i(x)(o(x) \dot{x} - f(x)) + \vartheta \int dt i^2(x) \tag{23}
\]

Expanding \( S \) in a Volterra expansion upto the second order may give rise to a probability functional

\[
P[o(x, t), i(x, t)] \sim e^{-\frac{1}{2} \int d^2x d^2x' \int dt dt' o(x, t)(o(x', t')K(x, x', t, t') + i(x', t')M(x, x', t, t'))} \tag{24}
\]

where \( K, M \) are functions, which are dependent on the neuronal architecture. This formalism has been applied to the Limulus eye, \[37\] whose equation is already known to get hold of a specific form of these functions. Though this is not the probability distribution we were talking about, but it will be interesting to see in this case that under what approximations the distribution matches with the functional.

An important point which is to be noted here is that the modified VP Eqn. \[14\] which we have used here is a complicated equation and in general the solution is in many instances difficult to determine. But if the parameters are small we may bypass the solution to find out the expectation values by perturbation theory. To give an example in the case of the FD oscillator Eqn. \[14\], with \( F(t) \) interpreted as white noise the FP equation is given by

\[
\frac{\partial P}{\partial t} = -\dot{x} \frac{\partial P}{\partial x} + \frac{\partial(x + \epsilon x^3)P}{\partial \dot{x}} + \frac{\partial^2 P}{\partial x^2} \tag{25}
\]

The equation for the expectation values show that a complete analytical solution is possible when \( \epsilon \) is small and we can treat it perturbatively.

4 Conclusions

We have tried in this article to give a theoretical framework to model EEG data which takes into account the underlying neuronal dynamics. As the EEG data is essentially thought to be consisting of travelling waves which is inherently nondeterministic, the formalism is based on finding out an analysis based on a suitable relaxation oscillator which has the potential to generate stochasticity. We also give a theoretical scheme of how to compute the EEG spectrum, the correlation and covariance matrices based on the modified version of the FN model. One can make some numerical estimates of the spectrum based on some fixed small values of the parameters and an assumed Gaussian probability distribution in the simplest case and see how it matches with the experimental data, based on FFT which in turn may give some definite indications about the correctness of the assumed model. So based on this hypothesis we hope that experimental studies of EEG, apart from clinical importance have a large role to play in
brain modelling. Recent experimental results indicate that wavelet transforms (WT) \[38\] are much suited for EEG studies, which depends on the scaling and shifting properties of the initial wavelet. It will be interesting to explore the consequences and extrapolations of the proposed theoretical framework in the context of the WT model.

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