Estimation of Intracortical Connectivity in a Dynamical Neural Field Model

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Abstract

Will save this for last.

Index Terms

Integro-Difference Equation (IDE), Neural Field Model, Cortical Connectivity.

I. Introduction

The human brain is arguably the world's most complex system. Approximately 100 billion neurons and 60 trillion synapses operate in concert to process information, resulting in the cognition that determines our behavior. The brain's efficiency, robustness, and adaptability are unparalleled by any man-made device and, despite decades of concerted research, our understanding of its complex dynamics remains modest. This has led researchers to study different scales of dynamics: starting from individual proteins, synapses, and neurons; moving through to neuronal networks and ensembles of neuronal networks. While our understanding of the function of neurons is well developed, the overall behavior of the brain's meso and macro-scale dynamics remains largely a mystery. Understanding the brain at this level is extremely important since this is the scale where pathologies such as epilepsy, Parkinson's disease and schizophrenia are manifested.

To date, there has been a considerable volume of work in generating physiologically plausible neural field models to fill the void of understanding brain dynamics at the meso/macroscopic scale. Mathematical neural field

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models provide insights into the underlying physics and dynamics of electroencephalography (EEG) and magnetoencephalography (MEG) (see [1] [2] for recent reviews). These models have demonstrated possible mechanisms for the genesis of neural rhythms (such as the alpha and gamma rhythms) [3] [4], epileptic seizure generation [5], [6] and [7] and insights into other pathologies [8] [9] that would be impossible to gain from experimental data alone. Unfortunately, to date the use of these models in the clinic has been limited, since the neural field model are constructed for a general brain dynamics and pathologies almost always have unique underlying patient specific causes. Data from EEG and functional magnetic resonance imaging (fMRI) offers the patient specific link to macro-scale cortical dynamics, making these tools readily applicable to the clinic. However the underlying system properties, or system states, are hidden from EEG and fMRI data, making predictions of the underlying physiology inherently difficult.

For models to be clinically viable they must be patient specific. A possible approach to achieve this would be to use a general neural field model, like the Wilson and Cohen [10] model or a neural mass model like the Jansen and Ritt model [11], and fit it to a patients EEG data. Fitting the neural models to individuals is a highly non-trivial task, and until very recently this has not been reported in the literature. An estimation frame work for neural field model known as dynamical causal modeling (DCM) [2] [12] has recently been proposed for studying evoked potential dynamics. Via a Bayesian inference scheme, DCM estimates the long range connectivity structure between the specific isolated brain regions that best explains a given data set. The interconnected brain regions are modeled by the Jansen and Ritt equations. The approach has proven very useful in understanding specific hierarchical networks of neural information processing. Another recent publication describing a parameter estimation method with a neural field model used an unscented Kalman filter with the Wilson-Cowan neural field equations [13]. This work takes a more system theoretic approach to the neural estimation problem and marks a first step in what has the potential to revolutionize the treatment of many neurological diseases where therapeutic electrical stimulation is viable.

Generating patient specific models allows the application of a range of techniques from control systems theory, where tailored electrical stimulation could be used therapeutically in a closed loop fashion. Currently available epileptic seizure prediction and control devices (i.e., the vagal nerve stimulator) are implemented in an “open loop”. That is, the therapeutic electrical stimulation waveforms are adjusted for each patient by trial and error, disregarding the patient’s neurodynamics and information about their particular pathologies. Given access to an accurate model, the application of control theory in these circumstances would allow for robust therapeutic stimulations. The work from Schiff and Sauer [13] successfully demonstrated it is possible to estimate parameters from the WC equations. This motivates the question, what parameters are the most important? How patient specific do the assumed parameters have to be for the model to be useful? In this paper, we address both of these questions and provide a theoretical platform to perform patient-specific grey-box modeling of the human neocortex.

II. The Importance of Cortical Structure

Neural field models use statistics of the cortex to relate mean firing rates of pre-synaptic neural populations to post-synaptic mean membrane potentials. Each neural population represents a functional cortical processing

unit referred to as a column. The columnar organization of the cortex is not discrete, but is continuous, where pyramidal cells are members of many columns. In general, cortical structure can be modeled in a physiologically plausible manner as being locally homogeneous (in short range intracortical connectivity) and heterogeneous (in long range corticocortical and corticothalamic connectivity) [14] [15]. Locally, each column is thought be connected via symmetric short range local excitation, with surround inhibition [16]. For example, this structural organization is most studied in the visual system, where the surrounding inhibition effectively tunes a cortical column to a particular receptive visual field [17].

Recent studies using neural field models have demonstrated the theoretical implications of specific connectivity structure of the cortical columns in neural field models, where the connectivity kernel governs the bifurcation points of the system [18] and types of oscillations that can be generated [19]. This implies that if we could estimate the connectivity structure for an individual, then we could capture the essential patient specific neurodynamics that lead to various oscillatory states. Estimating functional cortical connectivity via EEG measures is currently a highly active area of research. There is a lot of interest in understanding the hierarchy of brain regions that are involved in specific tasks, motivating the use of techniques like DCM. Other comparable methods are based on auto-regressive (AR) modeling of the EEG, MEG, or fMRI times series. Studies have concentrated on finding functional connectivity patterns using information contained in the AR coefficients using techniques like Granger causality [20], the direct transfer function [21], and partial directed coherence [22]. Again, like the DCM approach, these methods estimate long range connectivity patterns that does not provide a clear relationship between the continuum field models and data.

Until now, estimation of local intracortical connectivity structure has not been attempted. Recently, it has been shown that it is possible to estimate local coupling of systems governed by integro-difference equations by formulating a state-space model [23]. The key development in this work was to define a state-space model with an order independent of the number of observations (or ECoG recording electrodes in this case). In addition, the appropriate model selection tools have been developed [24] allowing for the application of this technique to neural fields. Modeling the neural dynamics within this framework has a distinct advantage over AR models, such that the number of parameters to define the connectivity basis functions (three in the paper) is considerably smaller than the number of AR coefficients typically required to achieve the relevant information criteria (AIC or BIC). In this paper, we demonstrate for the first time how intracortical connectivity can be inferred from ECoG data, based on a variant of the Wilson and Cowan neural field model [10]. This work provides a fundamental link between the theoretical advances in neural field modeling and patient specific data.

III. Neural Field Model

In this section, we describe a variant of the Wilson and Cowan neural field model [10] that will be used to create our intracortical connectivity estimator. This model is descriptive of a range of neurodynamics of the cortex such as evoked potentials, visual hallucinations, and epileptic behavior. It is also capable of generating complex patterns of activity such as Turing patterns, spirals, and traveling oscillations. The neural field model is popular due to being

parsimonious yet having a strong link with the underlying physiology. The model relates the average number of action potentials g arriving at r to the local post synaptic membrane voltage. The post-synaptic potentials generated at a neuronal population at location r by action potentials arriving from all other connected populations at locations r' can be described by

$$v(r,t) = \int_{-\infty}^{t} h(t-t')\, g(r,t')\, dt' + e(r,t). \tag{1}$$

The term $e(r,t)$ can be considered the potential generated by unmodeled inputs and spontaneous background activity. The post-synaptic response kernel $h(t)$ is described by

$$h(t) = \begin{cases} \exp(-\alpha t) & t \geq 0 \\ 0 & t < 0 \end{cases} \tag{2}$$

where $\alpha = \tau^{-1}$ and τ is the synaptic time constant. The synaptic response kernel can be more elaborate, using rise and fall time constants, however, this simple form serves our purposes. Nonlocal interactions between cortical populations are described by

$$g(r,t) = \int_{\Omega} w(r,r')\, f(v(r',\bar{t}))\, dr' \tag{3}$$

where $\bar{t} = t - d\,|r - r'|$, d is the propagation delay of action potentials, f is spiking rate of populations r', w is the spatial connectivity kernel, and Ω is the spatial domain, representing a cortical sheet or surface.

The average firing rate of the presynaptic neurons is related to the postsynaptic membrane potential by the sigmoidal activation function

$$f(v_t(r')) = \frac{\nu_0}{1 + \exp(\beta(v_0(r) - v(r',\bar{t})))}. \tag{4}$$

Even though we will model v_0 as being homogeneous across the cortical sheet we will express it as being spatially dependant allowing for generality when constructing the state-space representation in the next section. The parameters in equation 4 are detailed in Table 1. By combining equations 1 and 3 we get the spatiotemporal model

$$v(r,t) = \int_{-\infty}^{t} \int_{\Omega} h(t-t')\, w(r,r')\, f(v(r',\bar{t}))\, dr' dt' + e(r,t) \tag{5}$$

To arrive at the final form of the model we shall state the synaptic response kernel as a Green's function

$$Lh(t) = \delta(t), \tag{6}$$

where L is a temporal differential operator and $\delta(t)$ is the Dirac-delta function. This provides the most general form

$$L\left(\frac{\partial}{dt}\right) v(r,t) = \int_{\Omega} w(r,r')\, f(v(r',\bar{t}))\, dr' + e(r,t), \tag{7}$$

where L is a polynomial of order n with constant coefficients that provides an n^{th} order model. Most studies considering this model use first or second order time derivatives. We shall use the first order model giving

$$\frac{dv(r,t)}{dt} + \alpha v(r,t) = \int_{\Omega} w(r,r')\, f(v(r',\bar{t}))\, dr' + e(r,t). \tag{8}$$

A. Multi-Layer Model

$$\frac{dv_e(r,t)}{dt} + \alpha_e v_e(r,t) = \int_\Omega w_{ee}(r,r') f(v_e(r',\bar{t})) + w_{ei}(r,r') f(v_i(r',\bar{t}))\, dr' + \varepsilon_e(r,t) \tag{9}$$

$$\frac{dv_i(r,t)}{dt} + \alpha_i v_i(r,t) = \int_\Omega w_{ie}(r,r') f(v_e(r',\bar{t})) + w_{ii}(r,r') f(v_i(r',\bar{t}))\, dr' + \varepsilon_i(r,t) \tag{10}$$

The subscript ei denotes connectivity from an inhibitory neural element to an excitatory neural element. We can write the model in matrix form

$$\begin{bmatrix} \frac{dv_e(t)}{dt} \\ \frac{dv_i(t)}{dt} \end{bmatrix} = \int_\Omega \begin{bmatrix} w_{ee}(r,r') & w_{ei}(r,r') \\ w_{ie}(r,r') & w_{ii}(r,r') \end{bmatrix} \begin{bmatrix} f(v_e(r',\bar{t})) \\ f(v_i(r',\bar{t})) \end{bmatrix} dr' - \begin{bmatrix} \alpha_e v_e(r,t) \\ \alpha_i v_i(r,t) \end{bmatrix} + \begin{bmatrix} \varepsilon_e(t) \\ \varepsilon_i(t) \end{bmatrix} \tag{11}$$

$$\frac{d\mathbf{V}(r,t)}{dt} = \int_\Omega \mathbf{W}(r,r') f(\mathbf{V}(r,t))\, dr' - \mathbf{A}\mathbf{V}(r,t) + \mathbf{E}(r,t) \tag{12}$$

The iEEG observation equation is

$$y(k,t) = \int_\Omega m(k,r)\left(v_e(r,t) + v_i(r,t)\right) dr' + w(k,t) \tag{13}$$

B. Alternate Model Description I

An alternate and equivalent way to remove the temporal integration using the Laplace transform . We start by considering a discrete neural mass i with inputs with zero delay from another discrete mass j and

$$v_i(t) = \int_{-\infty}^{t} h_j(t-t') w_{ij} p(v_j(t))\, dt', \tag{14}$$

where w_{ij} can be considered the synaptic efficiency from j to i. Now we take the Laplace transform $\mathcal{L}(v_i(t)) = V(s)$ yielding

$$\begin{aligned} V_i(s) &= H_j(s) w_{ij} P(V_j(s)) \\ &= (s+\alpha)^{-1} w_{ij} P(V_j(s)) \qquad (15) \\ \Rightarrow sV_i(s) + \alpha V_i(s) &= w_{ij} P(V_j(s)). \qquad (16) \end{aligned}$$

Now taking the inverse Laplace transform we get

$$\frac{dv_i(t)}{dt} + \alpha v_i(t) = w_{ij} p(v_j(t)). \tag{17}$$

Next we convert the model from discrete neural masses to a continuum by

$$\frac{dv(r,t)}{dt} + \alpha v(r,t) = \int_\Omega w(r,r')\, p(v(r',\bar{t}))\, dr' + e(s,t). \tag{18}$$

IV. STATE-SPACE MODEL

We begin the derivation of the state-space model with the discrete time, continuous space IDE neural field model. A one-step Euler method was used to transform the ODE to a discrete time form.

$$\frac{v_{t+1}\left(\mathbf{r}\right)-v_t(\mathbf{r})}{T_s}=\int_\Omega k\left(\mathbf{r},\mathbf{r}'\right)f\left(v_t\left(\mathbf{r}'\right)\right)d\mathbf{r}'-\alpha v_t\left(\mathbf{r}\right)+e_t\left(\mathbf{r}\right), \tag{19}$$

$$v_{t+1}\left(\mathbf{r}\right)=T_s\int_\Omega k\left(\mathbf{r},\mathbf{r}'\right)f\left(v_t\left(\mathbf{r}'\right)\right)d\mathbf{r}'-T_s\alpha v_t\left(\mathbf{r}\right)+v_t(\mathbf{r})+T_se_t\left(\mathbf{r}\right), \tag{20}$$

$$v_{t+1}\left(\mathbf{r}\right)=T_s\int_\Omega k\left(\mathbf{r},\mathbf{r}'\right)f\left(v_t\left(\mathbf{r}'\right)\right)d\mathbf{r}'+(1-T_s\alpha)v_t\left(\mathbf{r}\right)+T_se_t\left(\mathbf{r}\right), \tag{21}$$

$$v_{t+1}\left(\mathbf{r}\right)=T_s\int_\Omega k\left(\mathbf{r},\mathbf{r}'\right)f\left(v_t\left(\mathbf{r}'\right)\right)d\mathbf{r}'+\lambda v_t\left(\mathbf{r}\right)+T_se_t\left(\mathbf{r}\right). \tag{22}$$

where $e_t(r)\sim\mathcal{GP}(0,R)$, $\lambda=1-T_s\alpha$ and T_s is the time step or sampling step.

The observation equation for the model is

$$y_t(k)=\int_\Omega m(k,\mathbf{r})v_t(\mathbf{r})\mathrm{d}\mathbf{r}+\varepsilon(\mathrm{k}), \tag{23}$$

where $\varepsilon(k)\sim\mathcal{N}(0,\Sigma)$, k indexes the iEEG electrodes, and $m(k,r)$ is the observation kernel relating the neural field to EEG measurements accounting for electrodes characteristics.

In order to implement standard estimation techniques we propose a decomposition of the The field using a set of Gaussian basis functions. Decomposition allows a continuous field to be represented by a finite dimensional state vector. This allows for the application of standard nonlinear, non-Gaussian state estimation methods such as sequential Monte-Carlo techniques like particle filtering. The field decomposition is described by

$$v_t\left(\mathbf{r}\right)=\phi^T\left(\mathbf{r}\right)x_t. \tag{24}$$

where $\phi(r)$ is the Gaussian basis functions that are scaled by the the state vector. The connectivity kernel can all be decomposed into a set of basis functions

$$k(\mathbf{r},\mathbf{r}')=\theta^T\psi(\mathbf{r},\mathbf{r}') \tag{25}$$

Will will assume we know the parametric form of the basis functions, where the parameter θ is unknown. Each connectivity basis function can be considered a layer in the WC model, representing short range excitation, surround inhibition and long range excitation. Making substitutions we get

$$\phi^T\left(\mathbf{r}\right)x_{t+1}=\theta^T\int_\Omega\psi\left(\mathbf{r},\mathbf{r}'\right)f\left(\phi^T\left(\mathbf{r}'\right)x_t\right)d\mathbf{r}'-\alpha\phi^T(\mathbf{r})x_t+\varepsilon_t\left(\mathbf{r}\right). \tag{26}$$

To isolate the state we cross multiply by $\phi(\mathbf{r})$ and integrate over $\mathbf{r}$

$$\int_\Omega\phi\left(\mathbf{r}\right)\phi^T\left(\mathbf{r}\right)d\mathbf{r}x_{t+1}=\int_\Omega\phi(\mathbf{r})\theta^T\int_\Omega\psi\left(\mathbf{r},\mathbf{r}'\right)f\left(\phi^T\left(\mathbf{r}'\right)x_t\right)d\mathbf{r}'d\mathbf{r}-\alpha\int_\Omega\phi(\mathbf{r})\phi^T(\mathbf{r})d\mathbf{r}x_t+\int_\Omega\phi\left(\mathbf{r}\right)\varepsilon_t\left(\mathbf{r}\right)d\mathbf{r}. \tag{27}$$

Now we define

$$\Gamma=\int_\Omega\phi\left(\mathbf{r}\right)\phi^T\left(\mathbf{r}\right)d\mathbf{r}. \tag{28}$$

Substituting equation 28 into 27 and rearranging gives

$$x_{t+1} = \Gamma^{-1} \int_{\Omega} \phi(\mathbf{r}) \theta^T \int_{\Omega} \psi(\mathbf{r}, \mathbf{r}') f\left(\phi^T(\mathbf{r}') x_t\right) d\mathbf{r}' d\mathbf{r} - \alpha x_t + \Gamma^{-1} \int_{\Omega} \phi(\mathbf{r}) \varepsilon_t(\mathbf{r}) d\mathbf{r}. \tag{29}$$

We can rearrange to get

$$x_{t+1} = \Gamma^{-1} \int_{\Omega} \phi(\mathbf{r}) \int_{\Omega} f\left(\phi^T(\mathbf{r}') x_t\right) \psi^T(\mathbf{r} - \mathbf{r}') d\mathbf{r}' d\mathbf{r} \theta - \alpha x_t + \Gamma^{-1} \int_{\Omega} \phi(\mathbf{r}) \varepsilon_t(\mathbf{r}) d\mathbf{r}. \tag{30}$$

This provides the final state-space model

$$x_{t+1} = \theta^T q(x) - \alpha x + \Gamma^{-1} \int_{\Omega} \phi(\mathbf{r}) \varepsilon_t(\mathbf{r}) d\mathbf{r} \tag{31}$$

with observation equation

$$y_t(k) = \int_{\Omega} m(k, \mathbf{r}) \phi^T(\mathbf{r}) d\mathbf{r} x_t + \varepsilon(k), \tag{32}$$

V. Estimation of the Nonlinear Homogeneous IDE Neural Field Model

VI. Comparison of Estimated and True Connectivity

VII. Discussion

VIII. Conclusion

The conclusion goes here.

Appendix A
Ken's Discretization

Spatio-temporal interactions can be described by the continuous space continuous time stochastic differential equation

$$\tau dv(r,t) = \left(-v(r,t) + \int_{\Omega} w(r,r') f(v(r',t)) dr'\right) dt + de(r,t). \tag{33}$$

where $e(r,t)$ is a zero mean temporal Wiener process with incremental covariance $\Sigma_e(r)dt$. (not too sure about this noise since it needs to be white temporally but coloured spatially - which in continuous space-time I have never seen written ... has anyone?) To simplify the notation, but without loss of generality, 33 can be rewritten as

$$dv(r,t) = q(v(r,t)) dt + de(r,t) \tag{34}$$

Had $q(v(r,t))$ been a linear function in v, then 34 could be rewritten as

$$dv(r,t) = Av(r,t)dt + de(r,t) \tag{35}$$

Considering temporal sampling instances δ apart given by $\{t_k = 0,\ 1,\ 2, \dots\}$, then a solution to 35 at each sampling time is given by

$$v(r, t_k + \delta) = \exp\{A(\delta)\} x(t) + \int_{t_k}^{t_k + \delta} \exp\{A(t_k + \delta - \tau)\} de(r, \tau) \tag{36}$$

Let the discrete time random variable $e'(r,t_k)$ be

$$e'(r,t_k) = \int_{t_k}^{t_k+\delta} \exp\{A(t_k+\delta-\tau)\} de(r,\tau) \tag{37}$$

then, $e'(r,t_k)$ is a zero mean, temporally white noise term with covariance

$$E\left[e'(r,t_k), e'^{\top}(r,t_k)\right] = \int_{t}^{t+\delta} \exp\{A(t_k+\delta-\tau)\}\Sigma_e(r)\exp\{A(t+\delta-\tau)\} d\tau \tag{38}$$

But we are not so luck as to be able to go from 34 to 35 and thus I doubt if we can obtain an exact solution to 34 without an approximation. Agreed?

A possible approximate solution is the one you have already used. Thus, interpreting the difference equation 34 as an integral equation gives

$$v\left(r,t\right) = v(r,t_0) + \int_{t_0}^{t} q\left(v(r,\tau)\right) d\tau + \int_{t_0}^{t} de\left(r,\tau\right) \tag{39}$$

Using an Euler approximation, 39 can be approximated at each sampling time by

$$v\left(r,t_{k+1}\right) = v(r,t_k) + q\left(v(r,t_k)\right)\left(t_{k+1}-t_k\right) + \left(e(r,t_{k+1}) - e(r,t_k)\right) \tag{40}$$

which can be rewritten as

$$v\left(r,t_k+\delta\right) = v(r,t_k) + \delta q\left(v(r,t_k)\right) + e'(r,t_k) \tag{41}$$

where $e'(r,t_k)$ is a zero mean Gaussian random variable with covariance $\delta\Sigma_e$. (Note that, because of the way the noise is defined in 33 it is not δ^2 but δ)

APPENDIX B

Appendix two text goes here.

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