A Continuum Theory of Electro-Cortical Activity

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Abstract

A set of non-linear continuum field equations are presented which describe the macroscopic dynamics of neural activity in cortex. Numerical solutions of the coupled non-linear system of partial differential equations show properties analogous to cortical evoked potentials, oscillations at the frequency of the mammalian alpha rhythm and non-stationary epileptic spikes.

Key words: Electro-encephalogram, Neural Network, Cortical Field Theory, Evoked Potential

1 Introduction

Spatially continuous descriptions of the activity of cortical tissue are complementary to the more traditional and familiar neural network models which emphasise the discrete distribution of neurons in cortex. Such continuum descriptions can be motivated both physiologically and anatomically [2]. Further these descriptions allow mathematically tractable and computationally inexpensive solution of large scale models of neural tissue and are especially useful in the modeling, description and putative explanation of macroscopic electrocortical activity (e.g electro-encephalogram and evoked potentials). Indeed under certain restrictions and approximations pseudo-analytical solutions can be obtained [4]. Theories resulting from assuming spatial continuity are often referred to as *cortical mean field theories* in that populations of neurons generate a field of neural activity which can be approximated to first approximation by the mean activities of densely interconnected local neuronal aggregates. Such local neuronal aggregates, for the purposes of illustration,

may be thought of as corresponding to some putatively identified elementary structural or functional columniation of cortex.

Cortical field theories have been developed as coupled sets of integro-differential equations [8,5], coupled sets of non-linear ordinary differential equations [2,9], and more recently as coupled sets of non-linear partial differential equations [3,7]. However all have been of limited utility because of the absence of a clear relationship between variables and parameters of the theory and what is experimentally measurable. Presented here are are a set of equations that by the inclusion of anatomically derived neuronal connectivities, ionic reversal potentials and fast excitatory and inhibitory channel kinetics ameliorate many of these problems.

2 Theory

In order to avoid details of cellular and cortical geometry the state variable to be modeled is the *mean soma membrane potential*, h_j $(j = e, i)$, which for excitatory neurons is considered to be linearly related to the associated local field potential and hence the electro-encephalogram [2,5], and hence will be continuous in space. Each neuron is considered as a single RC compartment into which all efferent synaptic activity terminates. Further it is assumed that:

- the model neural tissue consists of two functionally distinct, homogeneous, excitatory and inhibitory neuronal sub-populations.
- there are two, isotropic and homogeneous, scales of neuronal interaction - cortico-cortical (long-range) and intra-cortical (short-range).
- cortico-cortical fibers are exclusively excitatory and synapse on both excitatory and inhibitory cell populations.
- cortico-cortical fiber density falls off exponentially with distinct characteristic scales for excitatory-excitatory and excitatory-inhibitory interactions.
- intra-cortical axonal conduction delays are negligible.
- relative refractory periods are ignored to first approximation.
- synaptic and conduction delay distributions are assumed sharply peaked about central values.
- the effect of pre-synaptic activity on the post-synaptic cells membrane potential is described in terms of a source term driving a second order system with two real and equal eigenvalues with the time scales of such effects corresponding approximately to "fast" excitatory (AMPA/kainate) and "fast" inhibitory (GABA_A) neurotransmitter kinetics.
- the output of each functionally distinct neural mass is defined in terms of its average instantaneous action potential firing rate, S_j $(j = e, i)$, which is a sigmoidal function of the mean soma membrane potential, h_j.

Based on the above assumptions a pair of coupled integro-differential equations can be derived [4], which contain kernels of convolutions corresponding to transmitter kinetics and cortical connectivity. By taking the Fourier Transform of these kernels of (Greens functions) integer derivative terms in space and time can be identified thus allowing the integro-differential equations to be rewritten as a coupled set of non-linear partial differential equations. Based on this result and under the restriction that S_j and h_j do not vary significantly over the characteristic scales of intra-cortical connectivity (*spatial coarse graining*), it can be shown that the following are valid *one dimensional mean field dynamical equations*

$$\tau \frac{\partial h(x,t+\overline{\xi})}{\partial t} = h^r - h(x,t+\overline{\xi}) + \Psi_e(h) I_e(x,t) + \Psi_i(h) I_i(x,t) \quad (1)$$

$$\left(\frac{\partial}{\partial t} + \gamma_e\right)^2 I_e(x,t) = \Gamma_e \gamma_e \mathrm{e}\{N_e^\beta S_e(h_e) + \phi(x,t) + p_e(x,t)\} \quad (2)$$

$$\left(\frac{\partial}{\partial t} + \gamma_i\right)^2 I_i(x,t) = \Gamma_i \gamma_i \mathrm{e}\{N_i^\beta S_i(h_i) + p_i(x,t)\} \quad (3)$$

$$\left(I\frac{\partial}{\partial t} + \overline{v}\Lambda\right)^2 \phi(x,t) - \overline{v}^2 \frac{\partial^2 \phi(x,t)}{\partial x^2} = \overline{v}\Lambda N^\alpha \left(\overline{v}\Lambda + I\frac{\partial}{\partial t}\right) S_e(h_e) \quad (4)$$

where $h = (h_e, h_i)^T$, $h^r = (h_e^r, h_i^r)^T$, $I_e = (I_{ee}, I_{ei})^T$, $I_i = (I_{ie}, I_{ii})^T$, $N_e^\beta = (N_{ee}^\beta, N_{ei}^\beta)^T$, $N_i^\beta = (N_{ie}^\beta, N_{ii}^\beta)^T$, $N^\alpha = (N_{ee}^\alpha, N_{ei}^\alpha)^T$, $\phi = (\phi_e, \phi_i)^T$, $\Lambda = \mathrm{diag}(\Lambda_{ee}, \Lambda_{ei})$, $\tau = \mathrm{diag}(\tau_e, \tau_i)$, $\Psi_j(h) = \mathrm{diag}(\psi_j(h_e), \psi_j(h_i))$, $p_e = (p_{ee}, p_{ei})^T$, $p_i = (p_{ie}, p_{ii})^T$ and I is the identity matrix, with

$$S_j(h_j) = S_j^{max}(1 + r_{abs} S_j^{max} \exp[-g_j(h_j - \theta_j)])^{-1} \quad (5)$$

$$\psi_j(h_{j'}) = (h_j^{eq} - h_{j'})/|h_j^{eq} - h_{j'}^r| \quad (6)$$

where $j, j' = e, i$. Table 2 defines all symbols used. Equation 1 corresponds to an average neuron into which all the synaptic "currents" terminate. Equations 2 and 3 correspond to the activation of post-synaptic receptors by incoming pre-synaptic activity. Equation 4 describes the propagation of neural activity (in terms of action potential firings) by the cortico-cortical (long-range fibers).

3 Numerical Solutions

A one-dimensional von Neumann-Richtmyer finite difference solver [6] coupled with a fourth-order Runge-Kutta method was used for the numerical

e, i	excitatory, inhibitory	τ_j	membrane time constant
h_j	mean soma membrane potential	$\overline{\xi}$	mean synaptic delay
γ_j	transmitter rate constant	$\overline{v}$	mean cortico-cortical conduction velocity
Γ_j	post-synaptic potential amplitude	h_j^r	resting cell membrane potential
N_{ej}^{α}	total number of connections that a cell of type j receives from excitatory cells via cortico-cortical fibers	h_j^{eq}	reversal potential associated with synapses of type j
$N_{j'j}^{\beta}$	total number of connections that a cell of type j receives from cells of type j' via intracortical fibers	S_j	mean firing rate
Λ_{ej}	characteristic scale of cortico-cortical fibers		

solutions of equations (1)-(4). Figure 1 illustrates three oscillatory modes for the mean soma membrane potential of excitatory cells in response to variations in the mean amplitude of an homogeneously applied spatio-temporally band-limited white noise driving excitatory cells - (i) low amplitude oscillation at $13 - 20\ Hz$ (mammalian beta), (ii) moderate amplitude oscillation at $8 - 13\ Hz$ (mammalian alpha) and (iii) high amplitude oscillations (analogous to epileptic spikes) associated with the regenerative spread of excitation (figure not shown).

Figure 2 illustrates the temporal response of excitatory neurons at $x = 0$ for two excitatory impulses of differing durations. The form of the damped oscillatory responses resembles the middle and late components found to exist in a variety of cortical event-related potentials (ERP). Of particular note is the notch on the first "wave" of excitation at about 25 ms. Such a notch is found in average evoked potential recordings from pre-pyriform cortex when the lateral olfactory bulb is stimulated. Freeman explained this as the result of re-excitation of pyramidal cells by pyramidal cells [2]. This result is the first unequivocal replication of this phenomenon, which demonstrates the mutually excitatory connections that are crucial for learning [1].

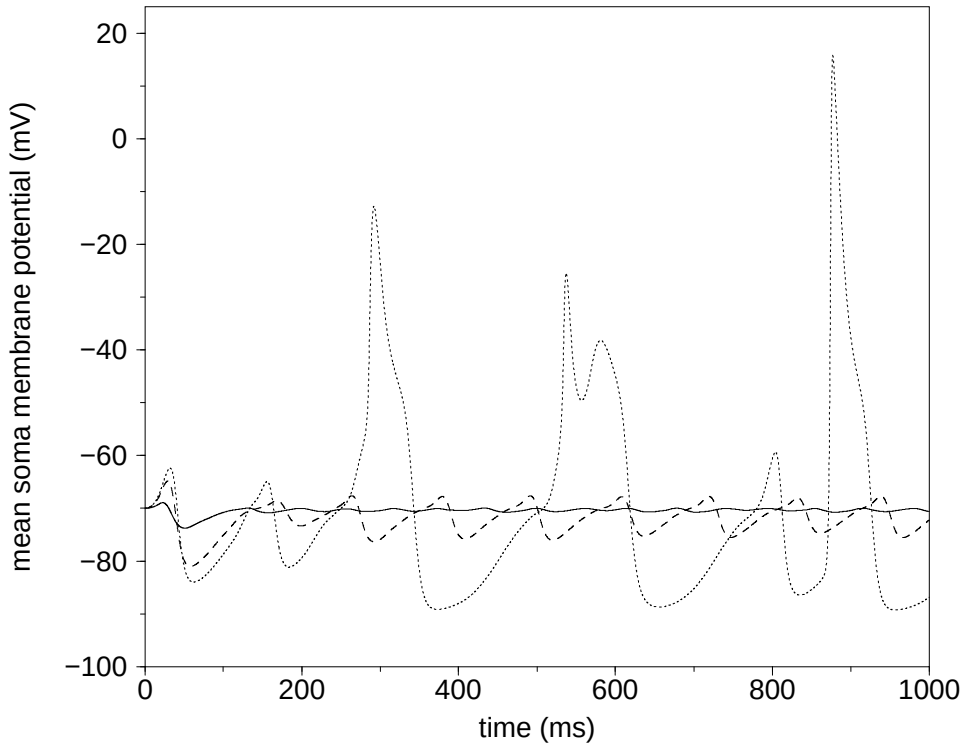

Fig. 1. Illustration of three oscillatory modes of the mean soma membrane potential of excitatory cells at $x = 0$ in response to homogeneous driving of inhibitory and excitatory cells with spatio-temporally band-limited ($< 6.2\ rad\, cm^{-1}$, $< 620\ rad\, s^{-1}$) white noise. (i) $\langle p_{ee}(x,t)\rangle = 1.1\ ms^{-1}$ (solid line) (ii) $\langle p_{ee}(x,t)\rangle = 1.4\ ms^{-1}$ (dashed line) (iii) $\langle p_{ee}(x,t)\rangle = 1.5\ ms^{-1}$ (dotted line). Other parameters for the driving noise were $\mathrm{var}[p_{ee}(x,t)] = 1.0\ ms^{-2}$, $\mathrm{var}[p_{ei}(x,t)] = 1.6\ ms^{-2}$ and $\langle p_{ei}(x,t)\rangle = 1.6\ ms^{-1}$. Boundary conditions were null flux, simulation time step was 0.1 ms, $\tau_{e,i} = 5\ ms$, $\Gamma_e = 0.18\ mV$, $\Gamma_i = 0.37\ mV$, $\gamma_e = 0.3\ ms^{-1}$, $\gamma_i = 0.065\ ms^{-1}$, $h^r_{e,i} = -70\ mV$, $h^{eq}_e = 45\ mV$, $h^{eq}_i = -90\ mV$, $N^{\beta}_{ee,ei} = 3034$, $N^{\beta}_{ie,ii} = 536$, $N^{\alpha}_{ee} = 4000$, $N^{\alpha}_{ei} = 2000$, $\Lambda_{ee} = 0.4\ cm^{-1}$, $\Lambda_{ei} = 0.65\ cm^{-1}$, $\overline{v} = 0.7\ cm\, ms^{-1}$, $r_{abs} = 1\ ms$, $\theta_{e,i} = -50\ mV$, $g_e = 0.28\ mV^{-1}$, $g_i = 0.14\ mV^{-1}$, E_{max}, $I_{max} = 1\ ms^{-1}$ and $\overline{\xi} = 0\ ms$. Further details about the parameter values used can be found in [4].

4 Conclusions

Inhomogeneities are easily added to the equations derived if gradient information is ignored and areal variations occur at characteristic scales large compared to numerical discretization. Further the issue of cortical lamination can be most profitably addressed, within this theory, by considering more than two local neuronal populations. Equations (1)-(4) allow the easy inclusion of any number of spatial scales and neuronal sub-populations and may be especially

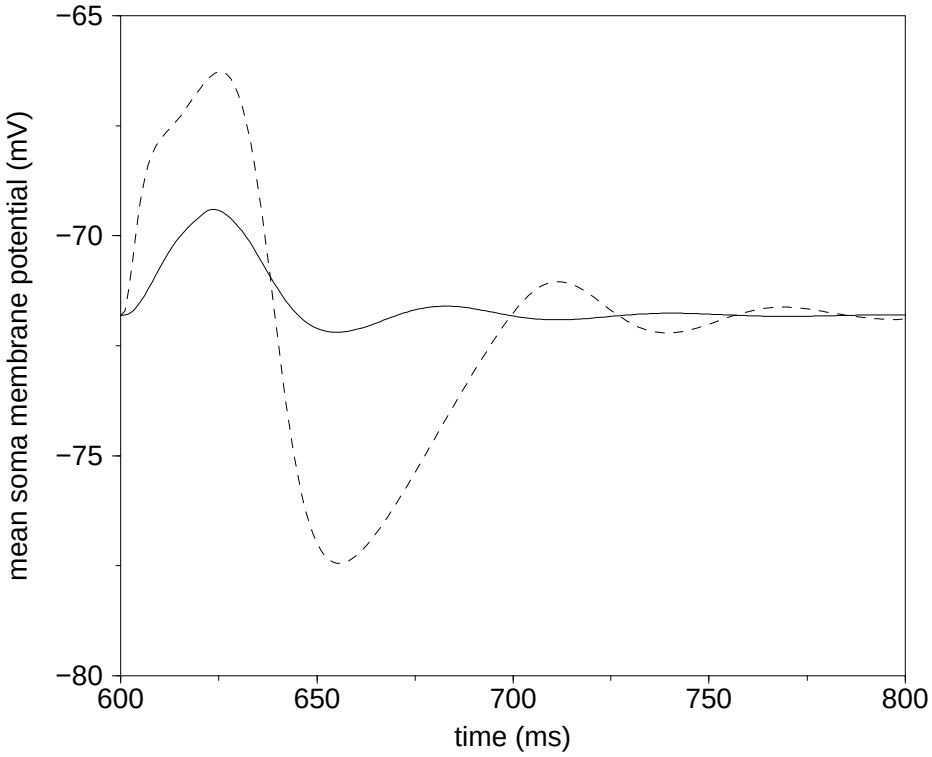

Fig. 2. Temporal response of excitatory neurons at $x = 0$, to two different impulses applied at $x = 0$ - (i) 2 ms^{-1} (height), 20 ms (width) (solid line), and (ii) 40 ms^{-1}, 1 ms (dashed line). $g_{e,i} = 0.28\ mV^{-1}$ otherwise all simulation parameters are the same as Figure 1.

useful in the modeling of distributed cortical systems such as the olfactory system (see for example [9]).

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