Non-equilibrium Statistical Mechanics of Recurrent Networks with Realistic Neurons

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Abstract. Experimental evidence suggests that spike timing might be used by neurons to process and store information. Unfortunately, the mathematical analysis of recurrent networks with spiking neurons is highly non trivial. Most analytical studies have therefore focused on rate-based models, whereas spiking models tend to be studied numerically. In order to bridge this gap, we propose an effective spiking neuron model which still allows for the application of non-equilibrium statistical mechanical techniques. The model is flexible and its parameters can be adjusted in order to match real data. We analyze the population dynamics in the simple case of constant excitatory synapses.

Keywords: Fokker Planck equation, spiking neurons, population dynamics.

1. Introduction

The nature of the neuronal code is one of the most intensively debated issues in modern neurosciences. In particular, the question of whether the exact timing of single spikes is important for transmitting information is still open (see [1] for an overview of neural coding). Synchronization and temporal correlations are observed experimentally in different brain areas and in different behavioral contexts [2, 3, 4, 5], yet the role of such phenomena is still to be understood. Information estimates from real data are often in favour of a negligible role for spike timing in coding [6]. On the other hand in the framework of synaptic plasticity the exact timing of pre and post synaptic spikes seems a crucial factor [7].

From a theoretical point of view rate models are much easier to study than spiking neuron models, and gave rise to a large body of theoretical research on many classes of recurrent neural networks with often sophisticated synapses, using tools from statistical mechanics (see e.g. [8, 9] for overviews). When faced with the need to incorporate spikes, the difficulty of the theorist is to find a compromise between mathematical complexity and biological realism. The Hodgkin-Huxley model includes the detailed dynamics of the conductances generating the action potential, and there is no need to impose resets or time delays by hand [10]. However, solving analytically the equations of a recurrent network of such neurons is out of the question. Even at a the reduced level of two-dimensional spiking neuron models (see e.g. [11] and [12]) we are not aware of attempts to apply the techniques of statistical mechanics to recurrent networks.

Most studies of recurrent networks with spiking neurons have been based on numerical simulations of integrate-and-fire type neurons, including versions with additional conductances to reproduce different modes of firing ([13] and see [14] for an excellent overview). Attempts to carry out also a mathematical analysis are hampered by the presence in such models of a hard reset and refractory period, imposed by hand, which cause discontinuities in the dynamical laws (in contrast to the Hodgkin-Huxley equations, where such phenomena are generated automatically by the non-linear equations). Those statistical mechanics analyses which have been published on networks of integrate-and-fire neurons (e.g. [15, 16]) have been restricted to relatively simple synapses and uncorrelated synaptic currents.

We propose here a new spiking neuron model which is sufficiently realistic to describe a qualitatively correct dynamics of the membrane potential. At the same time the equations are simple enough as to allow for a mathematical analysis of the network dynamics. The model is flexible and it is characterized by parameters which can be tuned to represent specific experimental data. Our final goal is to relate the network structure and the time constants characterizing the model to the emergency of collective phenomena, like synchronizations or oscillatory behaviour, and to examine the role of spike timing in that.

From a mathematical point of view the single neuron dynamics is described by coupled continuous differential equations which are linear in the corresponding variables (except for the non linearities present in the synaptic current). Although many such

neuron models have been proposed over the years, none appear to be characterized by differential equations that are simultaneously linear and continuous: the models with continuous equations, e.g. [11, 12], are all fundamentally non-linear; integrate-and-fire type models require the membrane potential reset after the spike. Continuity and linearity (except for the currents) are exactly the two requirements that will allow us to perform the mathematical analysis using non equilibrium statistical mechanics.

2. The model and its properties

Full details of our model and the rationale behind its ingredients will be given elsewhere [17]. For each neuron i we introduce a phase ϕ_i and an instantaneous firing rate r_i, which evolve in time according to the following equations:

$$\frac{d}{dt}\phi_i = r_i \qquad\qquad \tau_r \frac{d}{dt} r_i = F[I_i] - r_i + \tau_r \xi_i \tag{1}$$

where τ_r is the time scale at which the rate reaches a steady value for a constant input current, and ξ_i denotes Gaussian white noise, with $\langle \xi_i(t) \rangle = 0$ and $\langle \xi_i(t)\xi_j(t') \rangle = (2T/\tau_r)\delta_{ij}\delta(t-t')$. The membrane potential U_i^m is seen as the sum of a passive part U_i^p and a 'spike' part U_i^s. The latter is a periodic function of the phase ϕ_i (peaked at $\phi_i = 0, 1, 2, 3, \ldots$), the former evolves according to a leaky integrator equation, complemented by a term which generates a reset upon spike emission:

$$U_i^m = U_i^s + U_i^p \tag{2}$$

$$U_i^s = U_i^s(\phi_i, r_i) = \tau_s \Delta_u \; G_{\tau_s}\left[\frac{\sin(\pi\phi_i)}{\pi r_i}\right] \tag{3}$$

$$\tau_m \frac{d}{dt} U_i^p = RI_i - U_i^p - 2\tau_m (U_i^p + U_{\rm hyp})\ G_{\tau_s}\left[\frac{\sin(\pi\phi_i)}{\pi r_i}\right] \tag{4}$$

The function $G_\epsilon(x)$ is a Gaussian distribution with zero mean and standard deviation $\frac{1}{2}\epsilon$. The resting potential is defined as zero. $F[I]$ is the current-to-rate curve, with a treshold at $I = U_{th}/R$ and saturating at the maximum rate equal to the inverse of the refractory period $1/\tau_{ref}$:

$$I < U_{\rm th}/R: \quad F[I] = 0 \tag{5}$$

$$I > U_{\rm th}/R: \quad F[I] = \{\tau_{ref} - \tau_m \log[1 - U_{\rm th}/RI]\}^{-1} \tag{6}$$

$I_i = I_i^{syn}(t) + I_i^{ext}(t)$ in eqs.(1),(4) is is the sum of the synaptic current due to recurrent connectivities $I_i^{syn}(t)$ and of any input current external to the network $I_i^{ext}(t)$. Under the assumption that the synaptic current does not depend on the potential of the post-synaptic neuron we can write:

$$I_i^{syn}(t) = \sum_j J_{ij} U_j^s(\phi_j, r_j) = \sum_j J_{ij} \tau_s \Delta_u\ G_{\tau_s}\left[\frac{\sin(\pi\phi_j)}{\pi r_j}\right]; \tag{7}$$

where J_{ij} is the synaptic strength between neurons i nd j.

Fig.(1) shows the response of an isolated neuron ($I_{syn} = 0$) to the injection of a constant input current $I_{ext} = const.$, compared to a Hodgkin Huxley neuron. In both cases the equations were integrated numerically using MATLAB software.

3. Population dynamics in presence of uniform excitatory synapses

As a first test analysis, we examine the dynamics at a population level in the simple case of uniform excitatory connectivities: J_{ij}=J/N, for each pair of neurons i,j. Since

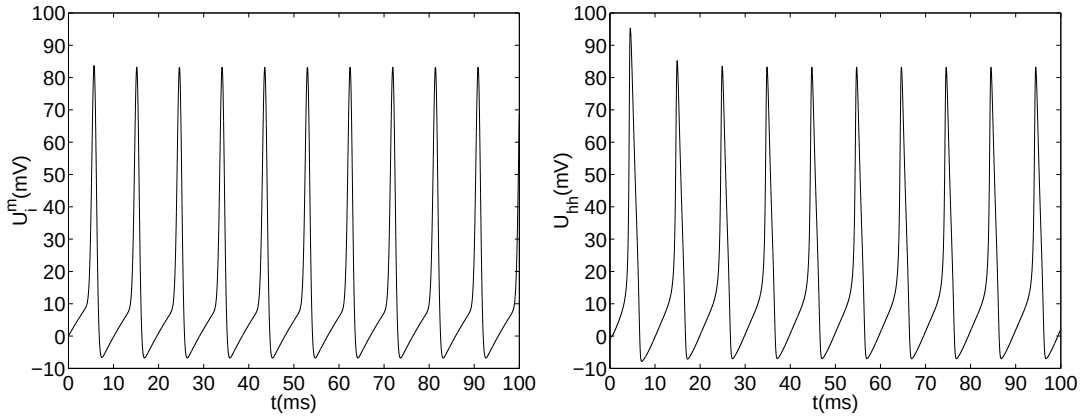

Figure 1. Phase rate model (left) vs Hodgkin-Huxley model (right) after injection of a constant current. I=10 mA; τ_r=1 ms; τ_m=20 ms; τ_s=1 ms; R=5 Ω; Δ_u=110 mV; τ_{ref}=5 ms; U_{th}=10 mV; U_{hyp}=15 mV. For the Hodgkin Huxley equations standard parameters as in [10]. I= 32 mA

the synaptic current (7) is a function of phases and rates only, the population dynamics will be described by the time dependent probability for the network configuration of phases and rates, $p_t(\boldsymbol{\phi}, \mathbf{r})$; its temporal evolution is fully determined by a Fokker Planck equation (see [18, 9]):

$$\frac{d}{dt}p_t(\boldsymbol{\phi}, \mathbf{r}) = -\sum_i \frac{\partial}{\partial \phi_i}\left[F_i^{\phi}(\boldsymbol{\phi}, \mathbf{r}, t)p_t(\boldsymbol{\phi}, \mathbf{r})\right] - \sum_i \frac{\partial}{\partial r_i}\left[F_i^{r}(\boldsymbol{\phi}, \mathbf{r}, t)p_t(\boldsymbol{\phi}, \mathbf{r})\right] + \frac{T}{\tau_r}\sum_i \frac{\partial^2}{\partial r_i^2}p_t(\boldsymbol{\phi}, \mathbf{r}) \tag{8}$$

$$F_i^{\phi}(\boldsymbol{\phi}, \mathbf{r}, t) = r_i \qquad F_i^{r}(\boldsymbol{\phi}, \mathbf{r}, t) = \tau_r^{-1}\left\{F[I_i(\boldsymbol{\phi}, \mathbf{r}, t)] - r_i\right\} \tag{9}$$

Unfortunately solving eq.(8) is an extremely hard computational task. Let us consider the average distribution of rates and phases instead:

$$p_t(\phi, r) = \int d\boldsymbol{\phi} d\mathbf{r} p_t(\boldsymbol{\phi}, \mathbf{r}) \frac{1}{N}\sum_j \delta[\phi - \phi_j]\delta[r - r_j] \tag{10}$$

In the limit of a large number of neurons it can be shown [17] that from eq.(8) one can extract the temporal evolution for $p_t(\phi, r)$, which takes the form of a Time Dependent

Ornstein Uhlenbeck process leading to a gaussian solution:

$$\frac{d}{dt}p_t(\phi,r) = -\frac{1}{\tau_r}\frac{\partial}{\partial r}\left\{\left[F[J\int d\phi' dr'\ p_t(\phi',r')U(\phi',r') + I_{\rm ext}(t)] - r\right]p_t(\phi,r)\right\} - \frac{\partial}{\partial\phi}\left\{r p_t(\phi,r)\right\} + \frac{T}{\tau_r}\frac{\partial^2}{\partial r^2}p_t(\phi,r) \quad (11)$$

Details of the analytical treatment will be given elsewhere [17]. The final solution can be expressed by the following system of equations:

$$p_t(\phi,r) = p_t(r)p_t(\phi|r);\ \ p_t(r) = \frac{e^{-\frac{1}{2}[r-\overline{r}(t)]^2/\Sigma^2(t)}}{\Sigma(t)\sqrt{2\pi}};\ \ p_t(\phi|r) = \frac{e^{-\frac{1}{2}[\phi-a(t)-b(t)r]^2/\sigma^2(t)}}{\sigma(t)\sqrt{2\pi}}; \quad (12)$$

$$\overline{r}(t) = \overline{r}(0)e^{-t/\tau_r} + \int_0^t ds\ e^{-(t-s)/\tau_r}F[JU(s) + I_{\rm ext}(s)] \quad (13)$$

$$\Sigma(t) = \sqrt{e^{-2t/\tau_r}\Sigma^2(0) + T(1-e^{-2t/\tau_r})} \quad (14)$$

$$\sigma^2(t) = \sigma^2(0) + \frac{2T}{\tau_r}\int_0^t ds\ b^2(s) \quad (15)$$

$$U(t) = \int\frac{d\phi dr}{2\pi\sigma(t)\Sigma(t)}\ e^{-\frac{1}{2}[r-\overline{r}(t)]^2/\Sigma^2(t) - \frac{1}{2}[\phi-a(t)-b(t)r]^2/\sigma^2(t)}\ U(\phi,r)$$
$$= \tau_s\Delta_u\int\frac{d\phi dr}{2\pi\sigma(t)\Sigma(t)}\ e^{-\frac{1}{2}[r-\overline{r}(t)]^2/\Sigma^2(t) - \frac{1}{2}[\phi-a(t)-b(t)r]^2/\sigma^2(t)}\ G_{\tau_s}\left[\frac{\sin(\pi\phi)}{\pi r}\right]$$

and the coefficients $a(t)$,$b(t)$ in eq.(12) obey the following equations:

$$\frac{d}{dt}a(t) = \frac{b(t)}{\tau_r}\left\{\frac{2T\overline{r}(t)}{\Sigma^2(t)} - F[JU(t) + I_{\rm ext}(t)]\right\} \quad (16)$$

$$\frac{d}{dt}b(t) = 1 + \frac{b(t)}{\tau_r}\left\{1 - \frac{2T}{\Sigma^2(t)}\right\} \quad (17)$$

The numerical solution has been evaluated using an algorithm written in C. Fig.(2) shows the average rate $\overline{r}$ and the average phase $a(t)+b(t)\overline{r}$ as a function of time. As one

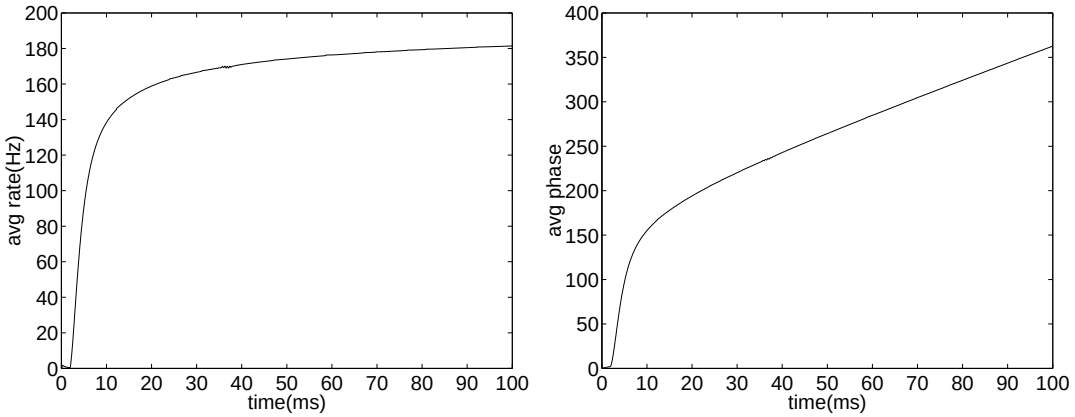

Figure 2. Time course for the average rate (left) and phase (right) with constant synapses. Parameters as in fig.(1). T=1; J=0.1.

could expect, since each neuron receives only excitatory inputs with uniform synaptic strength, the average rate grows rapidly saturating at the inverse of the refractory period. The average phase grows rapidly at first, and finally it reaches an asymptotic linear growth. Moreover we find that the rate variance saturates rapidly to its asymptotic value T, while the phase variance σ^2 grows linearly in time, starting from the initial condition of zero (full synchronization). Neurons increase their firing rates and gradually desynchronize.

4. Conclusions

We have presented a new spiking neuron model allowing for a mathematical analysis of the population dynamics. The model has parameters which can be adapted for application to specific data. We have solved the population dynamics in the case of constant excitatory recurrent synapses. Future developments include the use of plastic, structured synapses in presence of external stimuli.

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