

# Mesoscopic Neurodynamics

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## Abstract

An overview is given on the history, theory, and applications of mesoscopic neural models. It is shown why and how we can describe the behavior of a large population of neurons by the methods of statistical neurodynamics. Normal and epileptic propagation of cortical activity have been simulated.

Keywords: neurodynamics, statistical approach, epileptic activity

## 1 Introduction

The interaction between different levels of biological organization was one of the main fields of interest of Michael Conrad: “Biological systems are treated as percolation networks in which processes at all scales participate. Macroscopic inputs are transduced to microphysical events through an interleaved hierarchy of structures and processes and microphysical events are amplified to control macroscopic structures and functions. Integrity and adaptation are achieved through self-consistency dynamics operating at all levels of organization” (Conrad, 1996).

In the same spirit, in this paper we discuss the questions of whether, why, and how it is possible to integrate microscopic and macroscopic levels of neural organization by applying mesoscopic modeling strategy.

Structure-based bottom-up modeling has two extreme alternatives, namely multicompartmental simulations, and simulation of networks composed of simple elements. There is an obvious trade-off between these two modeling strategies. The first method is appropriate to describe the electrogenesis and spatiotemporal propagation of the action potential in single cells, and in small and moderately large networks based on data on detailed morphology and kinetics of voltage- and calcium-dependent ion channels. The mathematical framework is the celebrated Hodgkin-Huxley model (Hodgkin and Huxley, 1952) supplemented with the cable theory (Rall, 1962, 1977). Neural simulation software

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such as NEURON (Hines, 1984, 1993), and GENESIS (Wilson and Bower, 1989; Bower and Beeman, 1994) have been constructed to simplify the efficient simulation of neurons with branching patterns. The second approach offers a computationally efficient method for simulating a large network of neurons where the details of single-cell properties are neglected. There is a series of cell models with different levels of abstraction. While multicompartmental models take into account the spatial structure of a neuron, neural network techniques are generally based on integrate-and-fire models. The latter is a spatially homogeneous, spike-generating device. For a review of ‘spiking neurons’, see Gerstner (1999).

There is a long tradition to try to connect the ‘microscopic’ single-cell behavior to the global ‘macrostate’ of the nervous system analogously to the procedures applied in statistical physics. Global brain dynamics are handled by using continuous (neural field) description instead of the networks of discrete nerve cells. Both deterministic, field-theoretic (Griffith, 1963; Seelen, 1968; Wilson and Cowan, 1973; Amari, 1983), and more statistical approaches have been developed.

In a long series of papers starting from 1974 (Ventriglia, 1974, 1988, 1990, 1994), Ventriglia constructed a neural kinetic theory of large-scale brain activities. Ventriglia’s kinetic theory is a statistical field theory. He assumed two types of entities: spatially fixed neurons and spatially propagating spikes (‘impulses’). In our own laboratory this model framework was adopted and modified.

In this paper, first we discuss why it is necessary to use mesoscopic neural modeling strategy (Section 2). Then some specific model frameworks are reviewed (Section 3). Finally, several illustrative examples of the potential applications are given (Section 3.1).

## 2 Why meso-?

Mesoscopic methods bind the local (microscopic) and global (macroscopic) scales. The adjective ‘local’ means point-like here, and ‘global’ may be interpreted as the whole. Obviously, local descriptions may have very different (and relative) meanings, even if we do not go below the single-cell level. A single neuron, which is certainly a submicroscopic unit relative to Freeman’s microscopic model, may be described by a spatially extended (global) model by using a multicompartmental technique. But Freeman’s model for neuron populations is local, since the whole population is lumped into a ‘point’. In addition to speaking about ‘local’ and ‘global’ (Freeman and Kozma, 2000), we should remember the whole hierarchy of the neural organization. A step in this direction is the statistical theory of neural fields for describing the global brain activities in terms of interacting subfields.

While the electrophysiological results obtained by intracellular recordings

have a proper corresponding theory based on the Hodgkin-Huxley equations, data deriving from the brain imaging devices lack coherent theoretical approaches. Though it became clear that the gap between conventional neural modeling techniques and brain imaging data should be narrowed (Horwitz et al., 2000), there is much to be done.

Just as collective phenomena emerging in physical systems made from large number of elementary components (spins, molecules, etc.) are treated by statistical mechanics, analogously have statistical dynamical theories of neural populations been established (Beurle, 1956; Griffith, 1963; Seelen, 1968; Wilson and Cowan, 1973; Amari, 1974; Ventriglia, 1974, 1988, 1994; Ingber, 1982; Clark et al., 1985; Peretto, 1984, 1992). Neuronal population theories established earlier have used oversimplified single-cell models. One important example is the lack of ability to generate bursts mode. Our kinetic population model (Érdi et al., 1997; Barna et al., 1988; Gröbler et al., 1998) has common principles with multicompartmental models in that both models are built from anatomical and physiological data on cell types, network connectivities, unitary synaptic functions. There is, however, a methodological difference between the two approaches. For two reasons, our model does not contain the details of single-cell events (but benefits from the results of single-cell modeling!). First, even if we believed that without very detailed biophysical description of single-cell events the understanding of the functional organization of the whole hippocampus was impossible, we could neither find sufficient elementary data to build the model, nor the large-scale and long-term simulation could be done, even by supercomputers. Second, and more importantly, emphatically, the statistical theory we apply is based on the hypothesis that the behavior of large neuron populations can most adequately be described by the methods of statistical neurodynamics.

### 3 How meso-?

In the previous section we argued for the need of large-scale long-term examinations in the field of realistic neural networks. As we set forth before the statistical formulation of certain phenomena carries some extra information in quantitative respect compared to a system of many interconnected microscopic subsystems. In this section, some mesoscopic methods and models for describing characteristic functions of different cortical brain areas are reviewed.

The basic notion in the followings will be the population of neurons. We assume that a neural population consists of a large number of identical neurons that have similar properties and receive excitatory and inhibitory synaptic input with the same average rate. However, in some cases (e.g. Ormutag et al., 2000) the population is defined on more general basis: the function describing the population's state is based explicitly on an ensemble average of replica systems. The single neuron model to be incorporated into the statistical description may vary from the McCulloch-Pitts neurons via integrate-and-fire models to the

Hodgkin-Huxley model.

Based on the notion of neural populations, Mallot and Giannakopoulos (1996) presented a highly abstract, deterministic mean-field framework for modeling cortical networks. In their model, the state of each population is characterized by an intracellular potential function of a neuron located in the cortical point  $x$  and time  $t$ . This potential value is transformed into a spike rate through two types of point operations: a nonlinear transfer function corresponding to the single neuron model and spatial density function of the cells. Network architecture is implemented by connection layers modeling dendritic and axonal arborization of neurons constituting the population. A neuron in point  $x$  from a population  $p$  on the cortex can spread its activity around a point  $y$  on the connection layer  $l$ . Activity from different populations to a certain layer is summed at all times for every layer. The dendritic arbor of a given population then sums the activity of appropriate layers determining the population's intracellular potential. This model framework is primarily used for simulation of activity dynamics. Interesting dynamical properties, such as multistability and limit cycle behavior of space-invariant layered structures are considered. Layered cortical areas are also studied and the binocular image representation in the ocular dominance stripes of the primary visual cortex are examined using this model.

Nykamp and Tranchina (2000), following the work of Knight et al. (1996), presented a paper that deals with statistical neurodynamics based on the population density approach. As an example, they implemented a population density model of one hypercolumn of the visual cortex and applied it to orientation tuning. In the model, identical integrate-and-fire neurons are grouped to form a large neural population. For each population, they introduced a probability density function (PDF) that represents the distribution of neurons over all possible states. By assuming synaptic transmission with infinite velocity, and adopting an integrate-and-fire single cell model, the interacting population is characterized by a single variable, namely with the membrane potential.

Dynamics given by the time evolution of the PDF are based on probability conservation. Probability contained in a given interval  $(a, b)$  can only change due to probability flux across the endpoints of the interval. Probability flux in the model results from three sources: (i) the leakage flux is due to the exponential decay of the integrate-and-fire neurons' voltage towards the resting potential; (ii) excitation and inhibition flux are results of conductance change due to synaptic input; (iii) probability flux across the firing threshold potential of neurons giving the firing rate. The partial differential equations governing the time evolution of the PDF can efficiently be solved numerically, however the authors show that a diffusion approximation is more suitable for numerical analysis. The results given by this approximation are good if the voltage jumps due to synaptic conductance changes are small compared to the voltage interval determined by the resting and threshold potentials.

In a much earlier work, also based on the probability density function approach, Ventriglia constructed a neural kinetic theory of large-scale brain activities that he presented in a series of papers starting from 1974 (Ventriglia, 1974, 1982, 1990, 1994). His statistical theory is based on two entities: spatially fixed neurons and spatially propagating impulses. Neurons might be excitatory or inhibitory and their states are characterized by their subthreshold membrane potential or inner excitation, threshold level for firing, a resting level of inactivity state, maximum hyperpolarization level, absolute refractoriness period and a synaptic delay time. Under some conditions they emit impulses. Neurons are grouped in populations, state of the neurons in the population is described by the population's PDF. Impulses move freely in space (in the numerical implementation some rule should be defined due to treat the effects due to spatial discretization), and might be absorbed by neurons changing their inner excitation. Impulses are distributed in velocity-space according to the corresponding PDF.

Gröbler, Barna, and Érdi (Gröbler et al., 1998; Barna et al., 1988) extended this theory by the following method: they present a diffusion theory in two different senses. Both the dynamical behavior of neurons in their state-space is and the movement of spikes in the physical space are considered as diffusion process. The state-space in the model consists of the two-dimensional space coordinate  $\mathbf{r}$  for both neurons and spikes, a membrane potential coordinate  $u$  for all types of neurons, and an intracellular calcium-concentration coordinate  $\chi$  for pyramidal neurons only. Both cell types, the inhibitory and excitatory ones are described by ionic conductances specific to neuronal type. Instead of a fixed firing threshold, a soft firing threshold is realized by voltage-dependent firing probability. Absorbed spikes induce time-dependent postsynaptic conductance change in neurons, expressed by the alpha-function.

Denoting the PDF of neurons by  $g_s(\mathbf{r}, u, \chi, t)$  (illustration on fig 1, right panel) and the PDF of spikes by  $f_s^{(\alpha)}(\mathbf{r}, t)$  at time  $t$  and for population  $s$  time-evolutions are formulated by balance equations. For neurons the left-hand side of equation (1) describes the interspike dynamics containing the drift terms and the diffusion terms for the membrane potential ( $u$ ) and the intracellular calcium concentration ( $\chi$ ) variables while the right-hand side contains a sink and a source term corresponding to neurons returning from firing and those starting to fire, respectively.

$$\begin{aligned} & \frac{\partial g_s(\mathbf{r}, u, \chi, t)}{\partial t} + \frac{\partial}{\partial u} (\varepsilon_s(\mathbf{r}, u, \chi, t) \cdot g_s(\mathbf{r}, u, \chi, t)) + \\ & + \frac{\partial}{\partial \chi} (\eta_s(\mathbf{r}, u, \chi, t) \cdot g_s(\mathbf{r}, u, \chi, t)) - \frac{D_u}{2} \cdot \frac{\partial^2 g_s(\mathbf{r}, u, \chi, t)}{\partial u^2} - \\ & - \frac{D_\chi}{2} \cdot \frac{\partial^2 g_s(\mathbf{r}, u, \chi, t)}{\partial \chi^2} = b_s(\mathbf{r}, u, \chi, t) - n_s(\mathbf{r}, u, \chi, t) \end{aligned} \quad (1)$$

Here  $\varepsilon_s$  and  $\eta_s$  describes the electric and the calcium current to the cell, respectively. These functions are determined solely by the single-cell model. Generally, neurons in the interspike dynamics receiving ample excitation – which is determined by the voltage-dependent firing probability,  $p_s(u)$  – emit a spike and get into refractory period. After spending  $T_s$  amount of time in refractoriness cells return to the interspike dynamics into a point in phase space determined by the functions  $U_s$  and  $\chi_s$ . The amount of neurons going to and returning from refractoriness is given by equations (2) and (3), respectively.

$$n_s(\mathbf{r}, u, \chi, t) = \begin{cases} p_s(u) \cdot \varepsilon_s(\mathbf{r}, u, \chi, t) \cdot g_s(\mathbf{r}, u, \chi, t) & \text{if } \varepsilon_s(\mathbf{r}, u, \chi, t) > 0 \\ 0 & \text{if } \varepsilon_s(\mathbf{r}, u, \chi, t) \leq 0 \end{cases} \quad (2)$$

$$b_s(\mathbf{r}, u, \chi, t) = \int_{-\infty}^t dt' \int_{-\infty}^{\infty} du' \int_0^{\infty} d\chi' n_s(\mathbf{r}, u', \chi', t') \cdot \delta(u - U_s(u', \chi', t')) \cdot \delta(\chi - \chi_s(u', \chi', t')) \cdot \delta(t - T_s(u', \chi', t')) \quad (3)$$

Similarly, the time evolution of the PDF of spikes is given by equation (4). Its left-hand side accounts for drift and diffusion of spikes in space, the right-hand side for spike absorption, which is proportional to the spike number, and emission, which is proportional to the number of firing cells:

$$\begin{aligned} \frac{\partial f_{s'}^{(\alpha)}(\mathbf{r}, t)}{\partial t} + (\mathbf{v}_{s'}^{(\alpha)} \nabla) f_{s'}^{(\alpha)}(\mathbf{r}, t) - \frac{D_r}{2} \cdot \Delta f_{s'}^{(\alpha)}(\mathbf{r}, t) = \\ = -\sigma_{s'} f_{s'}^{(\alpha)}(\mathbf{r}, t) + \lambda_{s'}^{(\alpha)} \cdot \int_{-\infty}^{\infty} du' \int_0^{\infty} d\chi' n_{s'}(\mathbf{r}, u', \chi', t) \end{aligned} \quad (4)$$

where  $\mathbf{v}_{s'}^{(\alpha)}$  is the spike propagation velocity in the direction  $\alpha$ ,  $\sigma_{s'}$  and  $\lambda_{s'}^{(\alpha)}$  are the absorption and emission coefficients, respectively.

Information exchange between populations  $s$  and  $s'$  is realized by synaptic interaction when an impulse emitted by  $s'$  is absorbed by  $s$ . Postsynaptic conductance change is proportional to the integral of spikes absorbed ( $a_{s's}$ ) in the past weighted by an alpha function:

$$\gamma_{s's} = \frac{\overline{\gamma_{s's}}}{\tau_{s's}} \int_0^{\infty} dt' a_{s's}(\mathbf{r}, t - t') \cdot t' \cdot \exp\left(1 - \frac{t'}{\tau_{s's}}\right) \quad (5)$$

where  $\overline{\gamma_{s's}}$  is the maximal synaptic conductance and  $\tau_{s's}$  is the time to peak. The postsynaptic conductance is used to calculate the synaptic current which

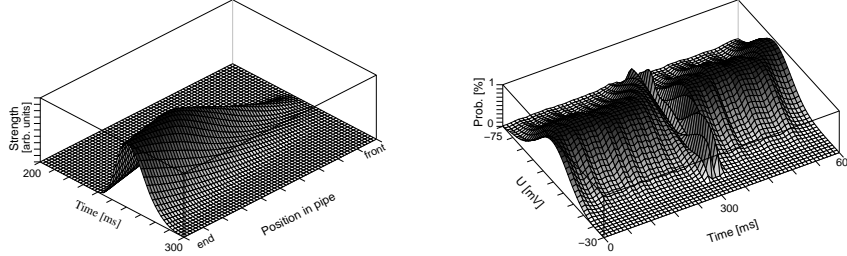


Figure 1: Left: an illustration for activity propagation through fibers. Direct connections between cortical areas are realized by pipes. Activity from firing cells of the source population are fed into the pipe which transmits it to cells of the target population. (Strength is given in arbitrary units.) Right: Time evolution of populational PDF. As an illustration a one dimensional distribution of neural states along the membrane potential axis is plotted against time.

is an additive term in  $\varepsilon_s(\mathbf{r}, u, \chi, t)$ .

Gröbler et al. (1998) performed various numerical simulations to study the behavior of the model. Results were obtained for large populations of neurons as well as for an ‘average cell’, which is a hypothetical neuron receiving the average synaptic input calculated by the statistical approach. They showed that decreasing inhibition in the model can result in synchronized population activity or for minimum inhibition in fully synchronized population burst due to recurrent excitation of pyramidal cells. Spatial activity propagation was also found numerically by solving the time evolution equations for a 0.4 mm x 1.6 mm sized hippocampal CA3 slice. Without inhibition propagating burst activity was observed with highest velocity of  $0.08 \frac{m}{s^2}$ .

The theory proposed by Gröbler et al. (1998) is capable of capturing spatiotemporal activity evolution in slices of cortical areas where synaptic connections are assumed to be randomly distributed and where the long-distant specific connections play negligible role. To study large system of interacting cortical areas (where the interaction is established by long-range, specific connections) this model has been extended (Kiss, 2000). A new term to the synaptic current in  $\varepsilon_s(\mathbf{r}, u, \chi, t)$  has been added:

$$\gamma_{s's} = \gamma_{s's}^{old} + \frac{\overline{\gamma_{s's}^t}}{\tau_{s's}^t} \int_0^\infty dt' \int_{\Omega(\mathbf{r}')} \kappa(\mathbf{r}, \mathbf{r}') \cdot a_{s's}(\mathbf{r}, t - t_d - t') \cdot t' \cdot \exp\left(1 - \frac{t'}{\tau_{s's}^t}\right), \quad (6)$$

where the  $\kappa(\mathbf{r}, \mathbf{r}')$  function determines the source and target cortical area between which information exchange occurs. Activity produced by the source

population influences the target population after  $t_d$  time delay giving account of signal propagation delay in fibers (see fig. 1, left panel).

The diffusion theory described above follows the “bottom-up” concept. It first exploits knowledge acquired from physiological single cell studies in creating the primary building blocks. Second, anatomical information on connections between specific brain areas is built into the model. At the same time this diffusion theory is a constructed theory and not a derived one, which means that the final form of equation (1) is constructed using basic principles of random processes and not derived from the equations describing single neurons and connections. However, efforts are done presently to derive macroscopic equations from microscopic ones and relate them to the diffusion theory.

As the diffusion theory accounts for an infinite number of neurons its applicability was tested. Comparing both the activity-time graph and the evolution of the PDF obtained by numerical simulations of the diffusion theory to the averaged result of a network with variable size of single cells, we found that they were in good accordance even for relatively small networks (about 1000 cells) and as the network size increased the deviation of between different methods decreased (data not shown).

### 3.1 Applications

Simulations of activity propagation in the feline cerebral cortex under normal and locally disinhibited conditions has been performed using a model built on the last described one. Connections between cortical areas were taken from the database reported by Scannell et al. (1995). Single-cell modules constituting the basic elements of neural populations are described by their membrane potential. In simulations, PDFs of neuron’s alone were taken into account, spike propagation were modeled by using direct connections only.

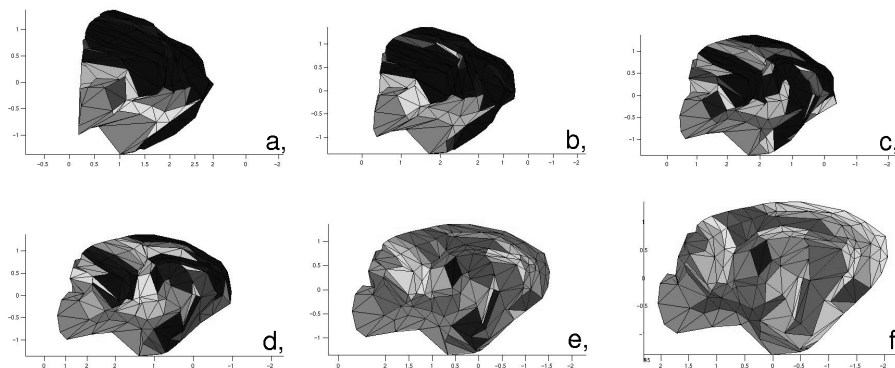


Figure 2: Simulations of epileptiform activity development on the cat’s cortex. Local disinhibition of posterior cingulate cortex resulted in an overall, highly synchronized activity state.



Simulations show that the probability of oversynchronization and epileptiform populational burst activity in the disinhibited brain area is increased as inhibition decreased. The nontrivial spread of activity from the initially bursting area through long range connections connections is shown on figure 2.

To have a better understanding of the mechanisms underlying epileptiform activity development we turned our attention to simulating single cortical areas. We introduced synaptic depletion and refilling into the synaptic conductance equations through a multiplicative resource factor which is described by equation 7.

$$r(t) = \begin{cases} 1 - \exp(-\frac{t}{\tau}) - \rho \sum_{t'=0}^t \delta(t' - t_{firing}) & \text{if } r(t = t_{firing}) > \rho \\ 1 - \exp(-\frac{t}{\tau}) - \sum_{t'=0}^t r(t') \cdot \delta(t' - t_{firing}) & \text{if } r(t = t_{firing}) < \rho, \end{cases} \quad (7)$$

where  $\rho$  is the size of one quantum of transmitter substance and  $\tau$  the refilling time constant.

Two kinds of epilepsy protocols were examined, motivated by experiments of Dóczy et al. (1999). First, we demonstrated that the treatment by the GABA<sub>A</sub> blocker bicucullin which is regarded as an *in vitro* model of focal cortical epileptogenesis, evokes quasi-periodic, large amplitude population bursts with interburst intervals about  $10 \pm 4$  seconds (figure 3, left panel). Second, the effects of magnesium-free solutions were simulated by assuming enhanced recurrent excitatory connections between pyramidal cells of the third layer as shown on figure 3, right panel.

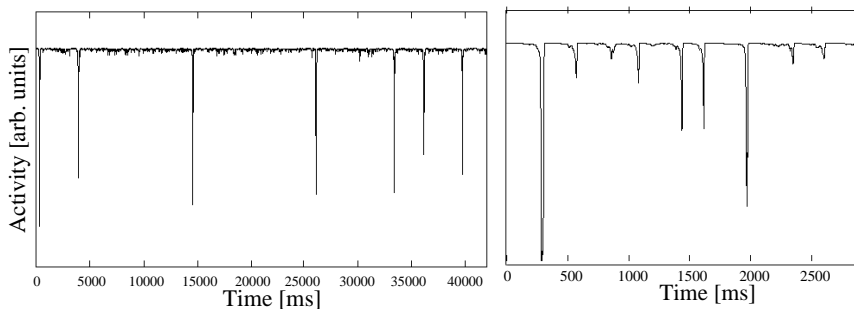


Figure 3: Simulation of epilepsy protocols. Electrical activity following treatment by bicucullin (left panel), and in magnesium-free solution (right panel)

We found that although both these *in vitro* models evoked similar epileptiform electrical activity in simulated slices characteristic timings and the fine

structure of bursts were different.

## 4 Conclusion

Our intention was to prove that mesoscopic neurodynamics is a necessary mathematical device to connect microscopic and macroscopic spatial and temporal scales. Specifically a scale-invariant theory (and software tool) was developed, which gives the possibility to simulate the statistical behavior of large neural populations, and synchronously to monitor the behavior of an ‘average’ single cell. Several illustrative applications have been given.

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