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A distribution-based model of the dynamics of neural networks in the cerebral cortex

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Abstract. Usual neural networks simplify temporal details in order to emphasize aspects of information processing and learning. This paper presents a complementary approach emphasizing temporal aspects at the expense of informational details. A model is proposed to take account of the variability of neuronal signals in the cerebral cortex and extract valuable information from the distributions of temporal characteristics in data obtained with single neuron recordings.

1. Introduction

Artificial neural networks are usually static: one input signal corresponds to one stable output. Even if the stabilization of this solution takes some time, there is no significant output during this period. An increasing number of models try to include time, but it is mainly for the analysis of temporal patterns and is obtained by using an external clock. It is likely that the dynamics of real neurons are much more complex. At least, the continuous evolution of neuronal signals is a data that is experimentally available and is not thoroughly exploited in current models of neural networks.

Neuronal signals recorded in the cerebral cortex are indeed difficult to handle because they seem very noisy and vary greatly from trial to trial in the same experiment. Statistical tools like peri-event time histograms (PETH's) are often used to filter this noise. However, these methods only give time-averaging for neurons recorded separately over several trials. In contrast, models often suppose space-averaging over many neurons at the same time: the information content of the signal carried by each individual neuron is very poor but the global activity of a population of neurons can be more stable and significant.

In this paper, we show how we have tried to extract meaningful information on the population behaviour from the variations of individual neuron signals. The model is based on distributions of neurons with different parameters and produces distributions of output signals, both for individual neurons and for neuronal populations. These distributions can be compared with experimental data on a single-trial basis.

2. Dynamic model

The structure of the model is derived from the multilayer neural network, with specific adaptations to introduce dynamic aspects (Fig. 1).

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2.1 Cascaded modules

In an ordinary multilayer network, each neuron of a layer receives inputs from all neurons of the previous layer with a given synaptic weight. The task that must be performed is learned by adjusting these weights to obtain the desired outputs for given inputs. Each neuron of a layer then receives its own subset of neuronal signals from the previous layer and performs a different operation compared to other neurons in the same layer. However, we are not interested here in the learning process as the experimental data are for a fixed task for which the subject has been trained. We assume, thus, that the learning phase is over and all synaptic weights are fixed. We also simplify the connection scheme by introducing a global activity *G(t)* for each layer. This activity is defined as the sum of the activities of all neurons in the layer and characterises the whole layer as a local population of neurons. The global activity is input to each neuron of the following layer with different synaptic weights *w*.

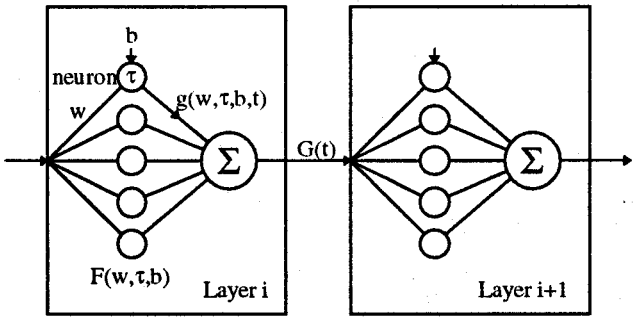

Figure 1: Structure of the model

2.2 Dynamic activation

The network defined so far acts like a filter transforming the input signal into an output signal. The same input always gives rise to the same signals in the network, in contrast to experimental data. Moreover, one can argue that the cerebral cortex is not a passive filter but an active processor that generates activities instead of simply transforming them. We have, thus, introduced a dynamic activation scheme based on signals from other brain areas to modulate the responsiveness of the network. We model this activation with a background of neuronal signal input to each neuron with a constant frequency of spikes *b*. The data signal is superimposed on this background.

2.3 Single neuron

To characterise the responsiveness of individual neurons, we must examine the dynamics of the neuronal signal at the level of the cell membrane potential. We use a simple leaky-integrator model [1] to describe the generation of excitatory postsynaptic potentials (EPSP's) following spikes in presynaptic neurons. In the cerebral

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cortex, it has been estimated that, as sources of input, there are in the order of 10^4 synapses on a single neuron [2], and that the total frequency of incoming spikes is high compared to the time constant of the membrane. Thus, we approximate the total incoming neuronal signal with a continuous function $f_n(t)$ (Hz) giving the instantaneous frequency of spikes. The response of the cell membrane to this input is:

$$u_n(t) = u_{0n} + \frac{v_s w_n}{\tau_n} \int_{t'=0}^{t} f_n(t').e^{(t'-t)/\tau_n}.dt' \qquad (1)$$

where v_s (mV) is the intensity of the EPSP created by a single spike on a synapse of weight 1, w_n is the synaptic weight, and τ_n is the time constant of neuron n.

When the membrane potential $u_n(t)$ rises above a given threshold voltage θ (mV), an action potential is fired. After that, the membrane potential returns to the initial value u_{0n}, then rises again following the same equation, but with different initial conditions. To compute the point where the membrane potential crosses the threshold, we approximate this potential by its tangent at the time t_n^i when the previous action potential occurred. Doing so, we derivate the integral in eq. 1 and come back to the input signal, multiplied by a constant factor. As a result, the times of occurrence of output spikes is given by the following recurrence relation:

$$t_n^{i+1} = t_n^i + \frac{\theta - u_{0n}}{v_s w_n} \frac{\tau_n}{f_n(t_n^i)} \qquad (2)$$

We assume that the initial value u_{0n} of the membrane potential is the potential maintained by the background input of dynamic activation signal. In a simple one-compartment integrator model, an action potential would clear all previous information. In the present model, it is as if the background input was made directly to the cell body, while signal inputs are made on the dendrites. The dendritic inputs are cleared by the firing of an action potential, but inputs onto the soma regain influence soon after the action potential to restore the base potential u_{0n}.

2.4 Neuronal signal

We consider that the unit of a neuronal signal is a peak in the instantaneous frequency curve, and we use triangular signals with the time of occurrence and amplitude of peak as the main characteristics. The input of the network is a triangular signal starting at t=0 and ending at t=t_E, with a peak at t=t_P. Each neuron of the first layer outputs a train of spikes as described by eq. 2. The instantaneous frequency of spikes is computed as the inverse of the time interval between two successive spikes and assigned to a point in between:

$$g_n\left(\frac{t_n^{i+1}+t_n^i}{2}\right) = \frac{1}{t_n^{i+1} - t_n^i} = \frac{v_s w_n}{\tau_n(\theta - u_{0n})} f_n(t_n^i) \qquad (3)$$

This function can then be extended to a continuous function of time. As the input is made of successive ramp signals, this implicit relation leads to a second-degree

equation that can easily be solved. When the output signal $g_n(t)$ of all neurons of the layer are summed, we obtain a global response that we approximate with a new triangular signal, starting at a time t_B and with new characteristics t_P and t_E. This triangular signal is input to the next layer.

2.5 Distributions

The model of individual neurons described above is characterised by several parameters. Some of them are common to all neurons of the network, but others are different from neuron to neuron in each layer. We define a distribution density function of the parameters that vary. We use continuous distributions, assuming a large number of neurons per layer. The summing of the activity of all neurons in a layer can then be performed as an integral of functions $g_n(t)$ weighted by the distribution density. There are three parameters to consider: the synaptic weight w, the time constant τ, and the frequency of the background signal b. The response of a layer is thus expressed as a triple integral:

$$G(t) = N.\int_w \int_\tau \int_b F(w,\tau,b).g(w,\tau,b,t).db.d\tau.dw \qquad (4)$$

where N is the number of neurons in the layer, $F(w,\tau,b)$ is the distribution function, and $g(w,\tau,b,t)$ is the output $g_n(t)$ of individual neurons expressed as a function of the three parameters and time.

As we have no *a priori* information on the shape of the distributions, we use normal distributions. $F(w,\tau,b)$ is thus a product of three normal curves, one for each parameter. The distribution functions for the synaptic weight w and the time constant τ are fixed, but the distribution function of the background frequency b is modified for each trial. The width of the distribution is fixed but the whole curve is moved between two limits. For each trial, we use the same distributions for all layers of the network.

3. Analysis

The model does not include any loop to control the stability of the network as in the case of real systems. We must manually tune each of its parameters so as to obtain realistic results. However, the overall characteristics of these results do not critically depend on the parameter values. An entire set of simulation results contains the activity profile of each neuron of the network for each trial. This set of data can be analysed with the same tools as experimental data, while giving additional information that cannot be inferred from real data. In particular, histograms of the activity of a single neuron for several trials can be broken down to individual curves.

More detailed analyses can be performed than with histograms, but the analysis of experimental data must be refined accordingly. We have, thus, developed a method to extract instantaneous frequency profiles from individual recordings of neuronal spikes. We plot the successive times of occurrence of spikes against the cumulated

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number of spikes. Smoothing this curve conserves the global number of spikes while compensating for the stochastic nature of their times of occurrence. We can then compute a frequency of spike between each pair of successive spikes and assign it to the point in between, as we did for the model. The timing and amplitude of the peaks in this frequency curve can easily be extracted. Fig. 2 shows the result obtained in the case of a neuron with two peaks of activity in most trials.

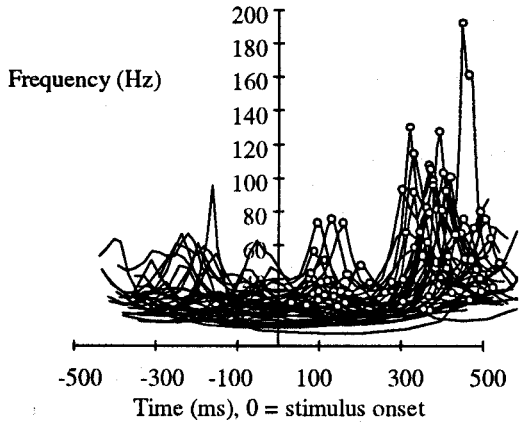

Figure 2: Example of dual-peak responses

The timing and amplitude of peak activity can be studied in scattergrams against other variables such as the total reaction time (RT) of the network. The total reaction time of the simulated network is defined as the onset of activity in the last layer. We can study the influence of each parameter of the model on the shape of the scattergrams. For example, the upper and lower limits of the total reaction time are mainly governed by the range of variation in the base frequency distribution. We can also see that neurons with strong connections with the previous layer, or with long time constants, react faster and more strongly than neurons with weak connections or short time constants. As a consequence, we could infer the static parameters (layer number, synaptic weight, time constant) of the neurons from the characteristics of the whole set of trials with different background frequencies b.

Of course, our simple model cannot account for the complexity of real networks and such a detailed analysis cannot be done for real data. The main discrepancy comes from the single neuron model. The relation between the amplitude P^m of the output peak for layer m and its time of occurrence is uniquely determined by a relation easily derived from eq. 3: the output frequency is maximum when the input signal is maximum and thus:

$$t_P^m = t_P^{m-1} + \frac{1000}{2P^m} \tag{6}$$

A neuron model with more subtle variations of the response time would give more realistic results.

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We can complete the analysis of scattergrams with statistical tools. Fig. 3 shows, for example, the variance of the time of occurrence of peak plotted against the mean time of occurrence normalised with the total reaction time. This figure shows that the cascaded structure of the model is compatible with experimental data. Due to population behaviour, a neuron in one layer can react later than a neuron in the next layer and still participate in data transmission. The variability in neuronal signal occurs at each stage of information processing and cumulates from the stimulus input to the motor output. There is no evidence of a critical decision-making centre causing the whole variability of the total reaction time.

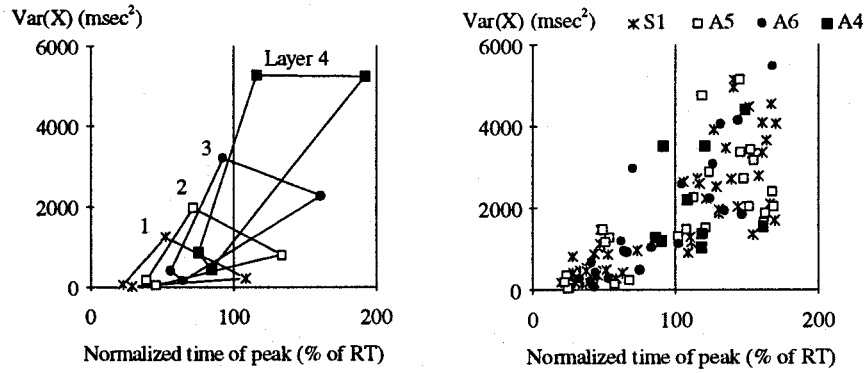

Figure 3: Simulated and experimental scattergrams of the variance of peak time

4. Conclusion

The analysis based on the proposed model is complementary to the usual information-oriented models. The dynamics of the neuronal response during the execution of a task can also give information about the organisation of the network, even though the mechanism by which the information is processed is totally neglected.

The model we present here is only an instance of a more general framework making the link between models at several levels: single cell, local network, global network,... Models of each level can be easily modified and the influence of the parameters observed in the global output. As the results of the simulations can be directly compared to real data, this could lead to a better cohesion between experimental data and theories [3].

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