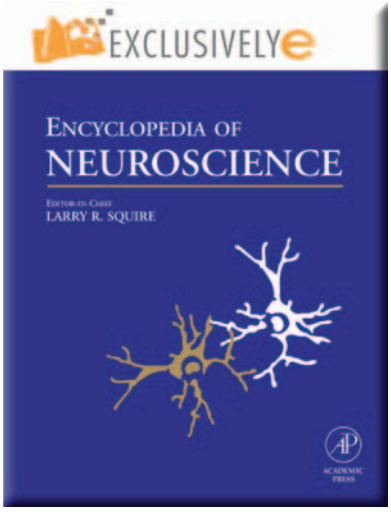

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Spiking Neuron Models

W Gerstner, Ecole Polytechnique Fédérale de Lausanne, Lausanne, Switzerland

Introduction: Spikes and the Question of Neural Coding

Neurons communicate with each other by electrical pulses, called action potentials or spikes. Whether the exact timing of action potentials or only the mean firing rate plays a role in neuronal communication is a question of intense debate, often referred to as the problem of neural coding. The question is further complicated by the fact that neuronal firing rates can be defined in at least three different ways: (1) the mean firing rate of a single neuron in a single trial, defined as a temporal average over a sufficiently long time (e.g., number of spikes in a time window of 500 ms, divided by 500 ms); (2) the firing density of a single neuron averaged across several repetitions of the same stimulus, typically defined via the amplitude of a peristimulus-time-histogram as a function of time; and (3) the population rate in a group of neurons with similar properties, defined as an average across the group. From reaction time experiments, it is clear that the first definition of a rate (average across time) cannot be the code used by the neurons. Averaging across several repetitions, as in the second definition, is a useful experimental method but cannot be the strategy of an animal which has to respond to a new stimulus. Finally, while the concept of averaging over groups of equivalent neurons is theoretically appealing, it is hard to see how such groups could be defined in a living brain since the organization into groups might change from one moment to the next, depending on task and stimulus demands.

If rates are such a difficult concept, does that imply that the exact timing of individual spikes matters? Not necessarily. A typical cortical neuron receives input from thousands of other neurons. What matters is probably the number (and spatial distribution across the dendrite) of spike arrivals averaged over an interval of one or a few milliseconds. However, if one spike arrival at an excitatory synapse were removed and replaced by a spike arrival at a different excitatory synapse at a similar dendritic location, the response of the neuron would hardly change. Since the question of coding and of the relevance of exact spike timings for neuronal response is still an open issue, researchers in theoretical neuroscience often use models that take the pulsed (i.e., 'spiking') nature of neuronal signals into account. These are so-called spiking neuron models, as opposed to rate models, in which the activity of neurons is described only in terms of firing rates.

Rate Models in Theoretical Neuroscience

In the field of artificial neural networks, the traditional way of describing neuronal activity has been by means of rate models. In this framework, each unit in a neuronal network is characterized by its rate $r(t)$, which depends nonlinearly on the total stimulation I it received immediately before. In a standard model in discrete time, the rate $r_i(t)$ of a neuron i is given by

$$r_i(t) = g(I_i(t - \mathrm{d}t)) \qquad [1]$$

where $\mathrm{d}t$ is the time step of the simulation and g the nonlinear gain function. Typically, the function g takes a value of zero for a stimulation I below some threshold value and saturates at a maximum value $r^{\max}$ for very strong stimulation. The total stimulation I_i of neuron i has two contributions, that is, the sum over all inputs converging onto neuron i plus potentially some external input I^{ext}:

$$I_i(t) = \sum_k w_{ik} r_k(t) + I^{\mathrm{ext}}(t) \qquad [2]$$

where r_k are the firing rates of other neurons k. In continuous time, eqn [1] is often replaced by a differential equation:

$$\tau \frac{\mathrm{d}r_i}{\mathrm{d}t} = -r_i + g(I_i) \qquad [3]$$

with some time constant τ that describes the response time of the unit to a change in stimulation.

While artificial neural networks using rate models have been successfully used in tasks describing cortex development or memory retrieval, the biological interpretation of the network units is unclear. Each unit i and k in eqn [2] could be interpreted as a single neuron and its rate as a temporally averaged firing rate. In view of the limitations of the firing rate concept discussed above, it must then be concluded that such a single-neuron rate model cannot be used to describe the fast neuronal dynamics during, for example, the rapid reaction of an organism to a new stimulus. Alternatively, each unit could be interpreted as a population of cells and its activity as the population rate. However, in that case the connectivity matrix w_{ij} would refer to the connectivity between populations rather than neurons, and the relevance of model results for electrophysiological measurements is questionable.

Detailed Models of a Spiking Neuron

The classical model of a spiking neuron is the mathematical description of Hodgkin and Huxley of action potential generation in the giant axon of the squid. Generation of spikes in the model arises through the interplay of four nonlinear differential equations. The first equation summarizes the current conservation on a small piece of membrane. A current I^{ext} injected onto the membrane can either charge the capacity C of the membrane or pass through one of the ion channels. With three different ion channel types (one for sodium, one for potassium, and one for all remaining 'leak' components), the change of the voltage is given by

$$C\frac{\mathrm{d}u}{\mathrm{d}t} = I^{\text{ext}} - g_{\text{Na}}m^3h(u - E_{\text{Na}}) - g_{\text{K}}n^4(u - E_{\text{K}}) - g_L(u - E_{\text{L}}) \qquad [4]$$

where g_{Na} and g_{K} are the maximum conductance of the sodium and potassium channel, respectively; E_{Na} and E_{L} are their reversal potentials; and m, h, and n are additional 'gating' variables that describe the state of the channel as a function of time. Each of the three variables m, n, and h follows an equation of the form

$$\frac{\mathrm{d}x}{\mathrm{d}t} = -\frac{x - x_0(u)}{\tau(u)} \qquad [5]$$

with empirical function $x_0(u)$ and $\tau(u)$ derived from the experiments (and different for m, n, and h).

Computer simulations as well as mathematical analysis of the system of four nonlinear differential equations shows that action potentials are generated only if the total stimulation I^{ext} reaches a critical value. To a first degree of approximation, one may therefore conclude that action potential generation is an all-or-none process. However, a closer examination of the behavior shows that there is neither a strict voltage threshold nor a strict current threshold. In particular, for time-dependent inputs (e.g., current steps and ramps or randomly fluctuating current as input), not only the momentary voltage or current amplitude matters, but the stimulation history as well.

Formal Spiking Neuron Models

Although neither the Hodgkin–Huxley model nor real neurons have a strict firing threshold, in practice the process of neuronal action potential generation can often be well approximated as a threshold process. The simplest model in the class of formal spiking neuron models is the leaky integrate-and-fire model. In this model, spikes are triggered whenever the membrane potential u reaches a given threshold θ. Below threshold, the membrane potential is described by its capacity C and input resistance R. Current conservation gives the linear differential equation.

$$C\frac{\mathrm{d}u}{\mathrm{d}t} = -\frac{u}{R} + I^{\text{ext}} \qquad [6]$$

If u reaches the threshold θ, the spike time t^f is noted and the membrane potential reset to a fixed value u_{reset}, before integration of eqn [6] is resumed. The essence of the leaky integrate-and-fire model is a clear separation between a completely linear (passive) subthreshold regime and a strict firing threshold. The leaky integrate-and-fire model is today the standard neuron model in many simulation studies concerning the dynamics of large neuronal networks or the question of neural coding.

Generalizations of the leaky integrate-and-fire model have been proposed in several directions. First, since some neurons exhibit in the subthreshold regime a damped oscillatory response to changes in the input (as opposed to a simple exponential decay), the voltage eqn [6] can be coupled with a second equation. As long as this second equation is linear, the subthreshold behavior of the neuron is still completely linear, that is, doubling the amplitude of an input generates a subthreshold response which is twice as large. Second, refractory effects (i.e., reduced responsiveness of a neuron immediately after firing) have been included. The combination of refractory effects with a linear subthreshold behavior leads to the spike response model with a membrane potential

$$u(t) = \eta(t - \hat{t}) + \int \kappa(t - \hat{t}, s)I^{\text{ext}}(t - s)\mathrm{d}s \qquad [7]$$

where $\hat{t}$ is the firing time of the last spike of the neuron, η describes the form of the action potential and its spike after-potential, and κ the linear response to an input pulse. The next spike occurs if the membrane potential u hits a threshold θ from below, in which case $\hat{t}$ is updated. Hence the main characteristics of the Spike Response Model are identical to those of the leaky integrate-and-fire model, that is, a linear subthreshold regime in combination with a strict firing threshold. However, the spike response model allows refractory effects to be included (since the functions η and κ depend on the time since the last firing) as well as subthreshold oscillations (expressed through an appropriate shape of η and κ).

A third generalization of the standard leaky integrate-and-fire model concerns adaptation. By introduction of a second equation that summarizes processes on a slower timescale, it is possible to describe the slow adaptation of the neuron after a step in the stimulating current.

Fourth, the spike triggered by a strict threshold process at the soma can be combined with one or several additional equations describing the (passive) properties of the dendrite. Finally, the strict threshold can be replaced by a smooth threshold process if eqn [6] is turned into a nonlinear equation:

$$C\frac{du}{dt} = -f(u) + I^{ext} \qquad [8]$$

where $f(u)$ describes the nonlinearities of the membrane in the vicinity of the firing threshold. Two standard choices of $f(u)$ are the quadratic integrate-and-fire model $f(u) = a\ (u-u_1)\ (u-u_2)$ and the exponential integrate-and-fire model $f(u) = -(u/R) + b\ \exp[(u-\theta)/\Delta]$. Both models have free parameters, that is, a, u_1, and u_2 for the quadratic and b, θ, Δ, and R for the exponential integrate-and-fire model, that can be used to adapt the model and put it into a desired firing regime.

Limitations of Formal Spiking Neuron Models

Formal spiking neuron models approximate the electrical properties of neurons by one or a few equations that summarize basic features of normal neuronal behavior with a small number of phenomenological parameters. Formal spiking neurons are therefore not suitable for predicting electrophysiological experiments under nonstandard stimulation conditions. In particular, since all electrical properties of a neuron are summarized in one or two phenomenological equations, these models cannot be used to predict changes of behavior caused by, for example, pharmacological blockage of specific ion channels. Moreover, since the spatial structure of real neurons is not represented in detail, all nonlinear dendritic effects cannot be incorporated into formal spiking neuron models (passive properties, however, can be). Similarly, since the model summarizes neuronal behavior under some reference condition, slow changes in neuronal behavior, caused by slow current ramps or accumulation of intracellular calcium or a simple fatigue of the neuron, for example, cannot be captured.

However, under some stimulation conditions, formal spiking neurons perform surprisingly well. A direct comparison of the spike response model with the Hodgkin–Huxley model during stimulation with random current shows that up to 90% of spike times of the Hodgkin–Huxley model are correctly predicted by the spike response model with a temporal precision of 2 ms. Moreover, a spike response model with a second equation describing adaptation has also been used to predict spike times of layer-V pyramidal neurons in rat cortex under random current injection. The same time-dependent input $I^{ext}(t)$ was given to both a model neuron and a pyramidal neuron. If the fluctuating current had large amplitude, the neuron itself generated spikes reliably with the same timing across several repetitions of the experiments, but less so if the fluctuation amplitude was reduced. Similarly, the model neuron was able to correctly predict the spike times of the pyramidal neuron (up to 70%) if the fluctuation amplitude of the current was high, but much less well if it was low. Thus, under random current injection, formal spiking neurons describe action potential generation in pyramidal neurons to a high degree of accuracy.

Spiking Neurons in Large Networks

A major advantage of formal spiking neurons such as the leaky integrate-and-fire model is their simplicity, which has two important consequences. First, it is possible to simulate neural networks with a large number of neurons at a reasonable numerical cost. Second, network properties such as the mean firing rate of neurons in a network of randomly connected integrate-and-fire units can be studied analytically with tools from mathematical probability theory, statistical physics, and bifurcation theory.

One important insight that has arisen from studies with formal spiking neurons is the importance of the subthreshold regime for cortical activity. In order to have large networks of interacting spiking neurons working in a state in which individual neurons show irregular firing with broad interspike interval distributions, it turns out to be necessary that the total drive from thousands of excitatory inputs is approximately balanced by inhibition. If this is the case, the neuronal membrane potential hovers normally in the subthreshold regime but stays close to the threshold so that neurons are very responsive to small changes in the input.

Furthermore, studies with networks of spiking neurons have been used repeatedly to elucidate the potential of different neural codes. For example, the stability and reproducibility of spatiotemporal spike patterns in networks of integrate-and-fire neurons have been studied in the context of synfire chains, a potential coding mechanism relying on precise spike times. Similarly, it can be understood why and under what conditions a population of spiking neurons responds instantaneously to changes in the input, that is, much faster than the membrane time constant that characterizes the passive response of the membrane. Finally, formal spiking neurons have been used for studying the functional consequences of spike timing-dependent plasticity.

The example of oscillatory activity allows one to see how formal spiking neurons can be used to predict by a purely mathematical argument the firing activity in large networks. For the sake of simplicity, we suppose that the network consists of N neurons with identical properties and that every neuron is connected to all other neurons by synapses of the same strength. Since we are interested in oscillatory activity, we assume that all neurons in the network fire at the same time, except one that lags behind by a small amount. Will this neuron eventually join the group of the others? To answer this question, we calculate the total synaptic input to this specific neuron, generated by the action potentials in the group of synchronous neurons. Knowing the input, we can derive from eqn [6] or eqn [7] the time course of its membrane potential, and from the time course, we can predict its firing time, that is, the moment when the membrane potential hits the threshold. An analogous calculation is repeated to predict the firing time of the group of synchronous neurons and hence the timing difference between the activity of the single neuron and that of the group. The one neuron lagging behind will eventually join the group of synchronous neurons (i.e., the oscillation is stable) if the timing difference is reduced from one firing cycle to the next. This is just a simple example, but similar mathematical arguments can be used to predict the population activity in large networks and answer questions such as: What is the mean firing activity of neurons in the network? Do neurons fire asynchronously, or do the firing times tend to cluster? Does the network activity show spontaneous oscillations? Will the network switch to a different state on a new input? If yes, could this explain short-term memory in neural networks?

To summarize, formal spiking neuron models are a highly simplified and compressed way of describing action potential generation in real neurons. While such an approach has obvious limitations and cannot be used to study detailed properties of isolated neurons (such as active dendrites or effects of pharmacological blockage of specific channels), the models have been useful in the past to elucidate the essence of spike generation in single neurons as well as principles of neuronal coding in large neuronal networks, and they will probably remain important tools for modeling studies in the future.

See also: Action Potential Initiation and Conduction in Axons; Hodgkin–Huxley Models; Population Codes: Theoretic Aspects; Spike-Timing Dependent Plasticity (STDP); Spike-Timing-Dependent Plasticity Models.

Further Reading

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