Spiking Neuron Model and Spike-Timing Dependent Plasticity

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1 Spiking neuron model – spike response model (SRM)

Let us introduce a spiking neuron model according to (Maass and Bishop 1999, Gerstner and Kistler 2002). The state of neuron i is described by a state variable $u_i(t)$ that can be interpreted as an effective electrical membrane potential or postsynaptic potential (PSP). When $u_i(t)$ reaches a threshold ϑ, a neuron fires, i.e. emits a spike. The moment of ϑ crossing defines a firing time $t_i^{(f)}$ of the f^{th} spike. The set of all n firing times of neuron i is denoted by $F_i = \{t_i^{(f)};\ 1 \leq f \leq n\} = \{t | u_i(t) = \vartheta\}$. Two different processes contribute to the value of the state variable $u_i(t)$:

$$u_i(t) = \sum_{t_i^{(f)} \in F_i} \eta_i(t - t_i^{(f)}) + \sum_{j \in \Gamma_i} \sum_{t_j^{(f)} \in F_j} w_{ij} \epsilon_{ij}(t - t_j^{(f)}) \tag{1}$$

First, immediately after firing an output spike at $t_i^{(f)}$, u_i is lowered or "reset". Mathematically, this can be done by adding an exponential negative kernel $\eta_i(t - t_i^{(f)})$ to the state variable $u_i(t)$, with the boundary conditions of $\eta(s) = 0$ for $s < 0$, and $\eta(s) \to 0$ for $s \to \infty$ (see Fig. 1):

$$\eta_i(s) = -\vartheta \exp\Big(-\frac{s}{\tau}\Big) \tag{2}$$

where τ is a time constant of the order of 1 – 10 ms. The u_i reset mathematically corresponds to the rise and fall of ϑ that occurs in real neurons after firing a spike.

Second, a neuron receives input from presynaptic neurons $j \in \Gamma_i$, where Γ_i is a pool of all neurons presynaptic to neuron i. A presynaptic spike at time $t_j^{(f)}$ increases (or decreases) $u_i(t)$ by an amount $\epsilon_{ij}(t - t_j^{(f)})$. The weight of synaptic connection(s) from neuron j to neuron i is denoted by w_{ij}. It takes positive (negative) values for excitatory (inhibitory) connections. The positive kernel $\epsilon_{ij}(t - t_j^{(f)})$ takes usually different exponential forms with boundary conditions of $\epsilon_{ij}(s) = 0$ for $s < 0$, and $\epsilon_{ij}(s) \to 0$ for $s \to \infty$. This kernel expresses an individual postsynaptic potential (PSP) evoked by neuron j on neuron i. An axonal transmission delay Δ^{ax} can be included in the definition of ϵ_{ij}. Often used formula for the PSP kernel is the double exponential (see Fig. 1)

$$\epsilon_{ij}(s) = \Big[\exp\Big(-\frac{s - \Delta^{ax}}{\tau_m}\Big) - \exp\Big(-\frac{s - \Delta^{ax}}{\tau_s}\Big)\Big] \tag{3}$$

where $\tau_m \approx \tau$ and $\tau_s < \tau_m$ are time constants of the order of 1 – 10 ms, and Δ^{ax} ($\geq$ 1 – 2 ms) is the axonal transmission delay from neuron j to neuron i. In electronic circuits, all $\Delta^{ax} = 0$. Another popular PSP kernel is the so-called α-function, $x \exp(-x)$, i.e.

$$\epsilon_{ij}(s) = \Big(\frac{s - \Delta^{ax}}{\tau_s}\Big) \exp\Big(-\frac{s - \Delta^{ax}}{\tau_s}\Big) \tag{4}$$

External inputs can be added to the right hand side of eq.(1), thus incorporating for instance background oscillations, electrode stimulation, and so on.

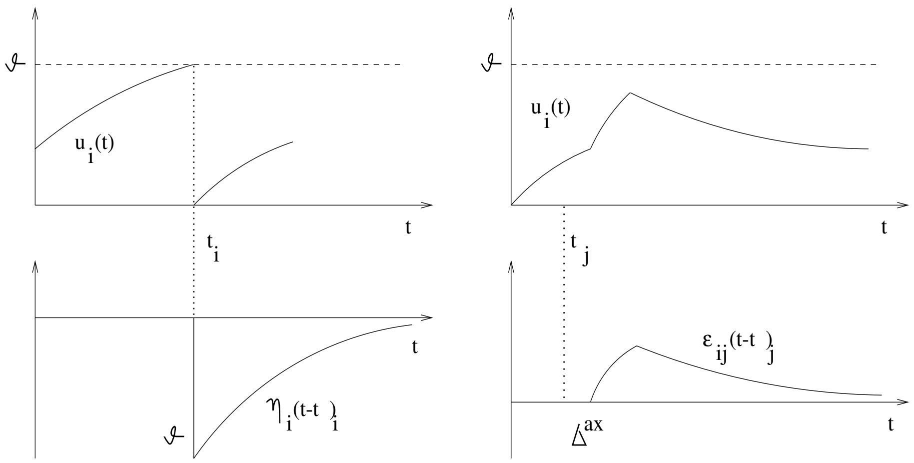

Figure 1: When the state variable $u_i(t)$ of a spiking neuron reaches the firing threshold ϑ at time t_i, it is reset to zero by means of the reset kernel $\eta_i(t-t_i)$. The kernel ϵ_{ij} describes the response of $u_i(t)$ after some axonal delay Δ^{ax} to a presynaptic spike at time t_j. In electronic circuits, axonal delays are equal to zero.

1.1 Integrate-and-Fire model

"Integrate-and-Fire" (I&F) neuron model is basically a resistor R and capacitor C in a parallel circuit, and can be directly electronically implemented on a chip (Maass and Bishop 1999). Eq.(1) holds also for the I&F model with the synaptic weights $w_{ij} = Rc_{ij}/\tau_m$, where R is the electrical resistance, c_{ij} is the charge deposited on the capacitor C by a single presynaptic spike of neuron j, and $\tau_m = RC$ is the membrane time constant. In the I&F model,

$$\eta_i(s) = -(\vartheta - u_r)\exp\Big(-\frac{s}{\tau_m}\Big) \tag{5}$$

where $u_r = u_i(t^{(0)})$ is the initial or resting membrane potential. Next,

$$\epsilon(s) = \frac{1}{1-(\tau_s/\tau_m)}\Big[\exp\Big(-\frac{s}{\tau_m}\Big) - \exp\Big(-\frac{s}{\tau_s}\Big)\Big] \tag{6}$$

1.2 Noisy spiking neuron

A deterministic spiking neuron fires whenever $u_i \geq \theta$. In reality, the noise is omnipresent and causes different responses of neurons to the same input. Thus, in the presence of noise, we can no longer predict the exact time of spike firing. The simplest and most popular way of introducing noise into spiking neurons is adding a stochastic variable $noise(t)$ with zero mean and finite variance to the right hand side of eq.(1).

In (Maass and Bishop 1999), the authors favor the noise in reset. Namely, the reset kernel in eq.(2) changes to

$$\eta_i(s) = -\vartheta\exp\Big(-\frac{r}{\tau}\Big)\exp\Big(-\frac{s}{\tau}\Big) \tag{7}$$

where r is a random variable with zero mean and finite variance. After each spike, a new value of r is drawn randomly from a Gaussian distribution $g(0,\sigma)$ with $\sigma << \tau$. Paramaters of noise are adjusted in such a way that the distribution of values of postsynaptic interspike intervals (ISI) has a Poisson character. Noisy reset has the advantage over other noise models that changing the level of noise does not alter this ISI distribution.

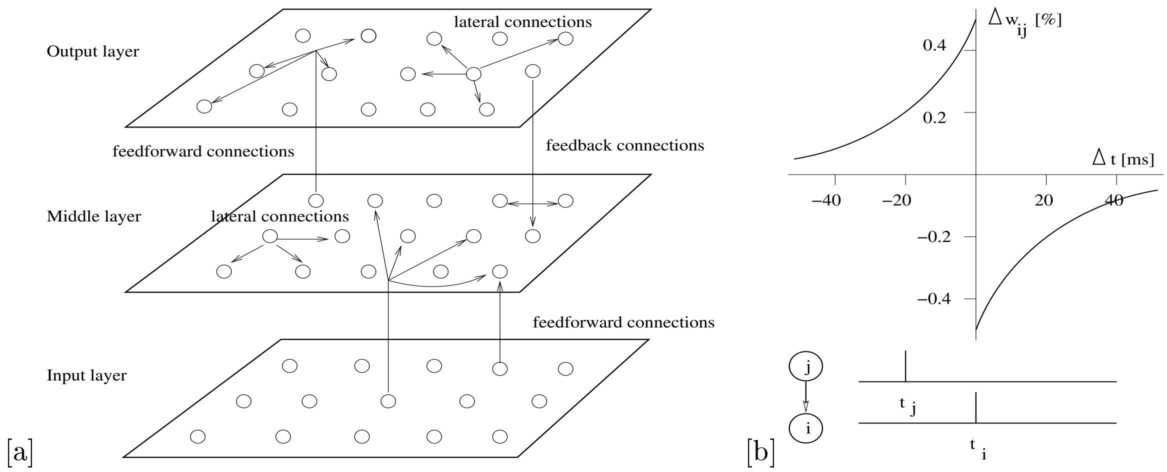

Figure 2: [a] Between neurons, there are feedforward, feedback and lateral connections at every level, local and global. [b] The STDP modification function. The change of w_{ij} due to a single pre- and postsynaptic spike pair expressed as a percentage of w_{max}. (Redrawn according to Song et al. 2000.)

1.3 Network of spiking neurons

After choosing the model(s) for basic elements of the network, one has to specify the network's architecture in terms of different layers and connectivity within and between layers (Fig. 2[a]). Let us assume that a subset of neurons $I_{input} \subset I$ represents input neurons, and another subset of neurons $I_{output} \subset I$ represents output neurons. A spike train emitted by neuron i is fully characterized by the set of firing times $F_i = \{t_i^{(1)}, ..., t_i^{(n)}\}$. The firing times F_i of the neurons from I_{input} constitute the network input. They may represent a random activation or some pattern. The firing times F_i of the neurons from I_{output} constitute the network output. These firing times are computed according to the previously described rules and according to the network architecture. From a mathematical point of view, a network of spiking neurons computes a function which maps a vector of several time series $\langle F_i \rangle_{i \in I_{input}}$ onto a vector of several other time series $\langle F_i \rangle_{i \in I_{output}}$.

2 Spike-timing dependent plasticity of synapses

Experimental neuroscientists discovered a form of long-term synaptic plasticity that depends on the relative timing of pre- and postsynaptic spikes at an excitatory synapse. It has been named the spike-timing dependent plasticity (STDP) (Song et al. 2000). Long-term potentiation of excitatory synapses occurred if presynaptic spikes preceded postsynaptic spikes by no more than $\sim$40 ms. On the other hand, presynaptic spikes that followed postsynaptic spikes produced long-term depression (weakening) of synapses. The largest changes (in both directions) occurred when the time difference between pre- and postsynaptic spikes was small. Further, there was a sharp transition from weakening to potentiation as the time difference passed through zero. Based on experimental measurments, a mathematical formula for STDP has been proposed (Song et al. 2000). A function $\Delta w_{ij}(\Delta t)$ determines the value of synaptic change resulting from a single pair of pre- and postsynaptic spikes separated by a time $\Delta t = t_j - t_i$ (Fig. 2[b])

$$\Delta w_{ij} = A_+ w_{max} \exp(\Delta t / \tau_+) \quad if \ \Delta t < 0$$

$$\Delta w_{ij} = A_- w_{max} \exp(-\Delta t / \tau_-) \quad if \ \Delta t > 0 \tag{8}$$

where w_{max} is the maximal limiting value of the synaptic weight. In case of $\Delta t = 0$, one of the two cases can be chosen at random. The parameters $\tau_+ > 0$ and $\tau_- > 0$ determine the ranges of pre- to postsynaptic interspike intervals over which synaptic potentiation and weakening occur, respectively. $A_+ > 0$ and $A_- > 0$ determine the maximum relative amounts of synaptic modification, which occur when Δt is close to zero.

Based on measurments made on mammalian cortical cells, Song et al. (2000) used these values of synaptic modification parameters: $\tau_+ = \tau_- = 20$ms (though these values do not necessarily have to be the same), $A_+ = 0.005$, $A_-/A_+ = 1.05$ (a slight dominance of synaptic weakening over potentiation), $w_{max} = 0.015$. They simulated an I&F model neuron with 1000 excitatory and 200 inhibitory synapses. Inputs were stimulated with independent Poisson spike series at different rates (inhibitory trains were fixed to an average rate of 10 Hz). Only excitatory synapses were modified, while inhibitory synapses were fixed. The steady state was usually achieved after about 100 s.

What is the role of STDP for one neuron? Song et al. (2000) showed that STDP has a strong regulatory effect on the steady-state firing rate ν_i of the postsynaptic neuron. It increases only by about 1 Hz for each 5 Hz increase in the input firing rates ν_j, whereas for nonplastic synapses it increases by about 100 Hz, which is a very unrealistic situation.

What is the role of STDP at a network level? Inputs that fire the postsynaptic neuron with short latency develop stronger synapses than inputs that fire the postsynaptic neuron with longer latencies. When some presynaptic spikes arrive after postsynaptic spikes, meaning that these presynaptic neurons did not cause postsynaptic firing, their synapses weaken. For uncorrelated stochastic presynaptic spike trains, chance determines whether given synapses will become weaker or stronger. However, when the presynaptic inputs are correlated (i.e. arrive at about the same time instant), they increase their ability to fire a postsynaptic neuron. Through the STDP synaptic potentiation correlated groups of synapses are strenghtened. Thus, the STDP displays the basic feature of Hebbian learning (Hebb 1949), that is the creation of synchronized cell ansambles. This should allow for fast binding of features belonging to the same object.

References

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