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A Random Behavior of Spiking Neurons: Towards Associative Memory

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Abstract. We simulate an associative memory using spiking neurons. Such models in which interaction among neurons are determined by, for example, a Hebbian like learning algorithm already exist. The Hopfield model is one of these examples. However, although we still have many interesting unknown issues in these models, we have some crucial drawbacks as well such as small storage capacity. To overcome these drawbacks, and more importantly, to pursue more biologically plausible models, we study dynamics of spiking neurons.

1. Introduction

Associative memory is a memory system in which we can store information and later we can recall the stored information from its partial and/or imperfect stimuli. An information is stored as a number of stable states with a domain of attraction around each of the stable states. If the system starts with any stimuli within the domain it will converge to the attractor following hopefully a short trajectory. This models human memory in the sense that we can recognize our friends even without meeting for a long time, or we can recall a song immediately after listening to a very beginning of the song. Hopfield (1992) proposed a fully connected neural network model of associative memory in which a set of binary patterns is stored distributedly among binary neurons as attractors. The model had been fairly well studied for more than a decade since then, and we now know it is not so practical partly due to its small storage capacity. Though we still have some interesting unknown issues in the model such as the number and distribution of the solutions which give a network a function of associative memory and these issues still attract interests of some physicists, we study another model using spiking neurons. The goal is to overcome issues like small storage capacity and, more importantly, to look for more biologically plausible models of human memory.

2. Spiking Neurons under Hodgkins-Huxley Model

Some regions in our brain such as *neocortex* or *hippocampus* are said to be made up of two categories of neurons, that is, *pyramidal cells* and *interneurons*. The pyramidal cells typically send signals to both pyramidal cells and interneurons via *excitatory synapses* (positive influences), while interneurons send signals to pyramidal cells via *inhibitory synapses* (negative influences). We now assume N pyramidal cells and implicit number of interneurons. We simulate these pyramidal cells with spiking neurons which interact with each other using electric current i^{pyr} via plastic synapses J_{ij}. Pyramidal cells are also interacted from interneurons by global inhibition i^{int}. Note that when we say simply *"neuron"* hereafter, it implies *"pyramidal cell."*

The membrane potential $v_i(t)$ of neuron i is expressed as:

$$\frac{dv_i(t)}{dt} = f(v_i(t), a_{i1}(t), \cdots, a_{in}(t)) + i_i(t)$$

using n parameters a_{ij} specified as:

$$\frac{da_{ij}(t)}{dt} = g_j(v_i(t), a_{i1}(t), \cdots, a_{in}(t)) \quad (j = 1, 2, \cdots, n)$$

which are necessary to determine the neuron's spiking behavior, and electric current $i_i(t)$ defined as:

$$i_i(t) = i_i^{\text{pyr}}(t) + i^{\text{int}}(t) + i_i^{\text{ext}}(t)$$

which will be described more in detail later.

The number of parameters n, the function form $f(\cdot)$ and $g_j(\cdot)$ depend on the model, such as Hodgkins-Huxley or FitzHugh-Nagumo. Here we assume Hodgkins-Huxley model in which $n = 3$ and $f(\cdot)$ and $g_j(\cdot)$ are given as

$$\begin{aligned} &f(v(t), a_1(t), a_2(t), a_3(t)) \\ &\qquad = \frac{G_{\text{Na}} a_2^3 a_1 (V_{\text{Na}} - v(t)) + G_{\text{K}}\, a_3^4 (V_{\text{K}} - v(t)) + G_{\text{L}}\, (V_{\text{L}} - v(t))}{C_m} \end{aligned}$$

and

$$\begin{aligned} &g_1(v(t), a_1(t), a_2(t), a_3(t)) \\ &\qquad = (1 - a_1(t)) \cdot \frac{0.01(10 - v(t))}{\exp\{(10 - v(t))/10\} - 1} - a_1(t) \cdot 0.125 \exp\{-v(t)/80\} \end{aligned}$$

$$\begin{aligned} &g_2(v(t), a_1(t), a_2(t), a_3(t)) \\ &\qquad = (1 - a_2(t)) \cdot \frac{0.1(25 - v(t))}{\exp\{(25 - v(t))/10\} - 1)} - a_2(t) \cdot 4 \exp\{-v(t)/20\} \end{aligned}$$

$$\begin{aligned} &g_3(v(t), a_1(t), a_2(t), a_3(t)) \\ &\qquad = (1 - a_3(t)) \cdot 0.07 \exp\{-v(t)/20\} - a_3(t) \cdot \frac{1}{\exp\{(30 - v(t))/10\} - 1)} \end{aligned}$$

where a_1 and a_2 are called *activation* and *inactivation* parameters of the sodium current, respectively, and a_3 the activation potassium of the potassium current.

The values of the constants are given here as: $V_{\mathrm{Na}} = 50\ (mV)$, $V_{\mathrm{K}} = -77\ (mV)$, $V_{\mathrm{L}} = -54.4\ (mV)$, $G_{\mathrm{Na}} = 120\ (mS/cm^2)$, $G_{\mathrm{K}} = 36\ (mS/cm^2)$, $G_{\mathrm{L}} = 0.3\ (mS/cm^2)$, and $C_m = 1\ (\mu F/cm^2)$.

Firing times of neuron i is defined as the time when the membrane potential $v_i(t)$ exceeds the threshold value $v_{\mathrm{threshold}}$, and the k-th firing time of neuron i is denoted by $t_i(k)$ which will be used for the following definitions of electric currents.

The $i_i^{\mathrm{pyr}}(t)$ is defined as a sum of synaptic electric currents from other pyramidal cells via plastic synapses J_{ij}, that is,

$$i_i^{\mathrm{pyr}}(t) = A^{\mathrm{pyr}} \cdot \sum_{j=1}^{N} \sum_{k} J_{ij} S^{\mathrm{pyr}}(t - t_j(k))$$

where J_{ij} is a synaptic strength from neuron j to neuron i and A^{pyr} is a parameter that specifies the intensity of the current, and

$$S^{\mathrm{pyr}} = \begin{cases} 0 & \text{for } t < 0 \\ \{1/(\tau_1^{\mathrm{pyr}} - \tau_2^{\mathrm{pyr}})\} \cdot \{\exp(-t/\tau_1^{\mathrm{pyr}}) - \exp(-t/\tau_2^{\mathrm{pyr}})\} & \text{for } t \geq 0. \end{cases}$$

Here, τ_1^{pyr} and τ_2^{pyr} are set to 10 and 5 (msec), respectively.

Inhibitory postsynaptic potentials are assumed to be given independently on i as

$$i_i^{\mathrm{int}}(t) = \frac{A^{\mathrm{int}}}{N} \cdot \sum_{j=1}^{N} \sum_{k} S^{\mathrm{int}}(t - t_j(k))$$

where A^{int} controls the global inhibition, and

$$S^{\mathrm{int}} = \begin{cases} 0 & \text{for } t < 0 \\ \{-1/(\tau_1^{\mathrm{int}} - \tau_2^{\mathrm{int}})\} \cdot \{\exp(-t/\tau_1^{\mathrm{int}}) - \exp(-t/\tau_2^{\mathrm{int}})\} & \text{for } t \geq 0 \end{cases}$$

with τ_1^{int} and τ_2^{int} being set to 5 and 2.5 (msec) here, respectively.

The membrane potential $v_i(t)$ of all the neurons $(i = 1, 2, \cdots, N)$ are set so that they maintain fixed values with $i_i(t) = 0$ during $t < 0$. Then external electric current i_i^{ext} triggers initial firings at $t = 0$, which evokes the dynamics afterwards. Note that i_i^{ext} are given only at $t = 0$ and then set to be zero again.

Finally, how we represent patterns to be stored among spiking neurons? Our constraint is that every neuron should fire only once in one period T. That is to say, our μ patterns are stored as:

$$s_i^\mu \in [\,0,\ T\,),\quad i = 1, \cdots, N,\quad \mu = 1, \cdots, p.$$

In other words, a pattern is stored as a configuration of N real values between 0 and T.

See Yoshioka (2001) more in detail. All of the definitions in this section is after his paper.

3. Learning by Evolution

The question is then whether the above simulation is biologically plausible indeed.

Yoshioka (2001) proposed the following Hebbian like solution to store a set of patterns.

$$J_{ij} = \frac{1}{N} \sum_{\mu=1}^{p} W(s_i^\mu - s_i^\mu)$$

where

$$W(t) = \frac{1}{\tau_1 - \tau_2} \{ \frac{e^{-t/\tau_1} - e^{-(T-t)/\tau_1}}{1 - e^{-T/\tau_1}} - \frac{e^{-t/\tau_2} - e^{-(T-t)/\tau_2}}{1 - e^{-T/\tau_2}} \}$$

adding a comment that it's not biologically plausible since these solutions for J_{ij} take both positive and negative values, while the J_{ij} should biologically be all positive as mentioned in the previous section.

Hence our proposition is to use an Evolutionary Computation to obtain all positive J_{ij} which store a number of patterns. This is of course under an assumption that solutions are not unique though. As Edleman (1987) once proposed Neuronal Evolution, we assume evolution exists as neurons' level in our brain, which is not like a species' level in the original sense.

Evolutionary computation is a technique to solve problems, typically (but not necessarily) NP-complete optimization problems, using an analogy of biological evolution. See for example Bäck et al. (2000). In our problem, we represent a candidate solution by a string, named *chromosome* or *individual*, which is comprised of N^2 genes each of which represents each of J_{ij}. At the beginning, a population of these candidate solutions, say a hundred, is created at random. Since they are created at random, they do not perform well for the task of storing a set of given patterns. However some individuals are comparatively a little better than others. In order to evaluate the performance, we define a fitness function, or we might call it objective function, which evaluates how good is each individual. Then two individuals are selected as parents with the selection scheme: the better fitness the more likely to be selected. From selected two parents we pick up either of genes with equal probability from cite to cite of the two chromosomes (*uniform crossover*). Thus we obtain one child chromosome. Occasionally we replace some of the genes of the child chromosome with another random value (*mutation*). These procedures of obtaining one child chromosome are repeated until we create the next generation. In this way, we can expect chromosomes with better performance than the ones in the previous generation, and eventually we expect to obtain the solution(s) to a set of J_{ij} which have the perfect performance to store a set of given patterns as associative memory.

4. Concluding Remarks

We have described a realization of associative memory using spiking neurons. The simulation using spiking neurons is more biologically plausible than the traditional binary state neurons. Expecting more biologically plausible model we propose here to employ an evolutionary computation. Since we have just started this study, we have had no experimental result, but we hope many interesting phenomena await our exploration.

Reference

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