

## Simple Model of Spiking Neurons

Eugene M. Izhikevich

**Abstract**—A model is presented that reproduces spiking and bursting behavior of known types of cortical neurons. The model combines the biological plausibility of Hodgkin–Huxley-type dynamics and the computational efficiency of integrate-and-fire neurons. Using this model, one can simulate tens of thousands of spiking cortical neurons in real time (1 ms resolution) using a desktop PC.

**Index Terms**—Bursting, cortex, Hodgkin–Huxley, PCNN, quadratic integrate-and-fire, spiking, thalamus.

### I. INTRODUCTION

To understand how the brain works, we need to combine experimental studies of animal and human nervous systems with numerical simulation of large-scale brain models. As we develop such large-scale brain models consisting of spiking neurons, we must find compromises between two seemingly mutually exclusive requirements: The model for a single neuron must be: 1) computationally simple, yet 2) capable of producing rich firing patterns exhibited by real biological neurons. Using biophysically accurate Hodgkin–Huxley-type models is computationally prohibitive, since we can simulate only a handful of neurons in real time. In contrast, using an integrate-and-fire model is computationally effective, but the model is unrealistically simple and incapable of producing rich spiking and bursting dynamics exhibited by cortical neurons.

In this paper, a simple spiking model (1), (2) is presented that is as biologically plausible as the Hodgkin–Huxley model, yet as computationally efficient as the integrate-and-fire model. Depending on four parameters, the model reproduces spiking and bursting behavior of known types of cortical neurons, as we illustrate in Fig. 1 and summarize in Fig. 2.

Mathematical analysis of the model will be published in the monograph by Izhikevich [8]. The derivation of the first (1) is based on bifurcation theory and normal form reduction [2], [5], and the part  $v' = v^2 + I$  is sometimes referred to as being a quadratic integrate-and-fire neuron. The full model was first published in [10, eqns. (4) and (5) with voltage reset discussed in Sect. 2.3.1] in a trigonometric form more suitable for mathematical analysis. The form presented here is more suitable for large-scale simulations.

### II. THE MODEL

Bifurcation methodologies [8] enable us to reduce many biophysically accurate Hodgkin–Huxley-type neuronal models to a two-dimensional (2-D) system of ordinary differential equations of the form

$$v' = 0.04v^2 + 5v + 140 - (u + I) \quad (1)$$

$$u' = a(bv - u) \quad (2)$$

with the auxiliary after-spike resetting

$$\text{if } v \geq 30 \text{ mV, then } \begin{cases} v \leftarrow c \\ u \leftarrow u + d. \end{cases} \quad (3)$$

Here,  $v$  and  $u$  are dimensionless variables, and  $a$ ,  $b$ ,  $c$ , and  $d$  are dimensionless parameters, and  $' = d/dt$ , where  $t$  is the time. The variable

Manuscript received February 2003; revised April 2003. This work was supported in part by the Neurosciences Research Foundation and by a Grant from the Alafi Family Foundation.

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Digital Object Identifier 10.1109/TNN.2003.820440

### Rat's motor cortex

### Model

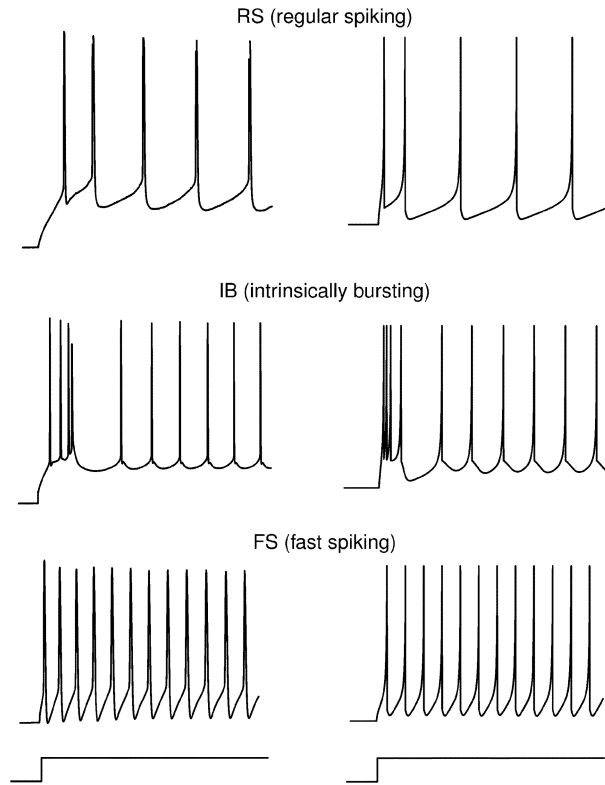


Fig. 1. The simple model (1), (2) can reproduce firing patterns of neurons recorded from the rat's motor cortex. Data are kindly shared by N. Desai, model parameters as in Fig. 2.

$v$  represents the membrane potential of the neuron and  $u$  represents a membrane recovery variable, which accounts for the activation of  $K^+$  ionic currents and inactivation of  $Na^+$  ionic currents, and it provides negative feedback to  $v$ . After the spike reaches its apex (+30 mV), the membrane voltage and the recovery variable are reset according to the (3). Synaptic currents or injected dc-currents are delivered via the variable  $I$ .

The part  $0.04v^2 + 5v + 140$  was obtained by fitting the spike initiation dynamics of a cortical neuron (other choices also feasible) so that the membrane potential  $v$  has  $mV$  scale and the time  $t$  has  $ms$  scale. The resting potential in the model is between  $-70$  and  $-60$  mV depending on the value of  $b$ . As most real neurons, the model does not have a fixed threshold; Depending on the history of the membrane potential prior to the spike, the threshold potential can be as low as  $-55$  mV or as high as  $-40$  mV.

- The parameter  $a$  describes the time scale of the recovery variable  $u$ . Smaller values result in slower recovery. A typical value is  $a = 0.02$ .
- The parameter  $b$  describes the sensitivity of the recovery variable  $u$  to the subthreshold fluctuations of the membrane potential  $v$ . Greater values couple  $v$  and  $u$  more strongly resulting in possible subthreshold oscillations and low-threshold spiking dynamics. A typical value is  $b = 0.2$ . The case  $b < a$  ( $b > a$ ) corresponds to saddle-node (Andronov–Hopf) bifurcation of the resting state [10].
- The parameter  $c$  describes the after-spike reset value of the membrane potential  $v$  caused by the fast high-threshold  $K^+$  conductances. A typical value is  $c = -65$  mV.

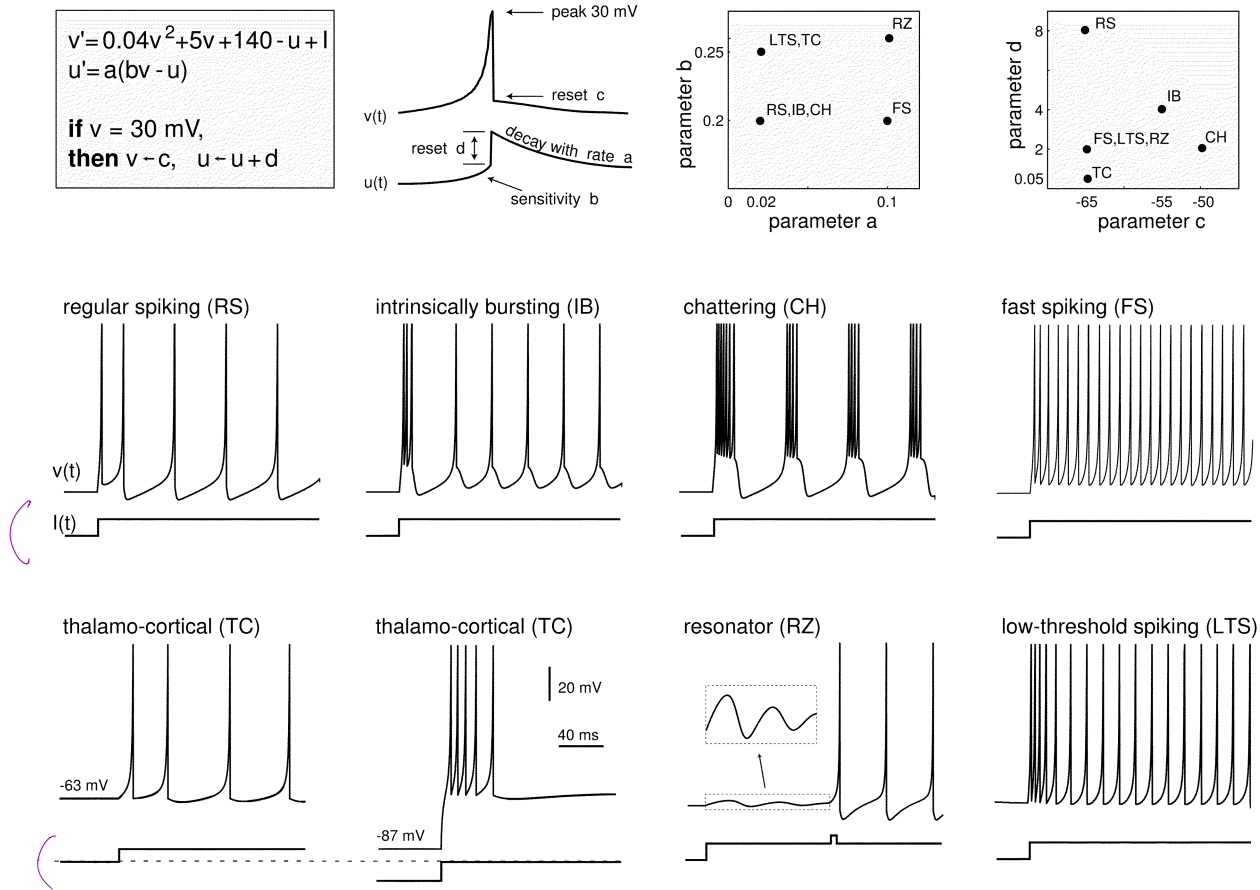


Fig. 2. Known types of neurons correspond to different values of the parameters  $a$ ,  $b$ ,  $c$ ,  $d$  in the model described by the (1), (2). RS, IB, and CH are cortical excitatory neurons. FS and LTS are cortical inhibitory interneurons. Each inset shows a voltage response of the model neuron to a step of dc-current  $I = 10$  (bottom). Time resolution is 0.1 ms. This figure is reproduced with permission from [www.izhikevich.com](http://www.izhikevich.com). (Electronic version of the figure and reproduction permissions are freely available at [www.izhikevich.com](http://www.izhikevich.com).)

- The parameter  $d$  describes after-spike reset of the recovery variable  $u$  caused by slow high-threshold  $\text{Na}^+$  and  $\text{K}^+$  conductances. A typical value is  $d = 2$ .

Various choices of the parameters result in various intrinsic firing patterns, including those exhibited by the known types of neocortical [1], [3], [4] and thalamic neurons as summarized in Fig. 2. A possible extension of the model (1), (2) is to treat  $u$ ,  $a$  and  $b$  as vectors, and use  $\sum u$  instead of  $u$  in the voltage (1). This accounts for slow conductances with multiple time scales, but we find such an extension unnecessarily for cortical neurons.

### III. DIFFERENT TYPES OF DYNAMICS

Neocortical neurons in the mammalian brain can be classified into several types according to the pattern of spiking and bursting seen in intracellular recordings. All excitatory cortical cells are divided into the following four classes [1], [3]:

- *RS (regular spiking)* neurons are the most typical neurons in the cortex. When presented with a prolonged stimulus (injected step of dc-current in Fig. 2RS, bottom) the neurons fire a few spikes with short interspike period and then the period increases. This is called the spike frequency adaptation. Increasing the strength of the injected dc-current increases the interspike frequency, though it never becomes too fast because of large spike-afterhyperpolarizations. In the model, this corresponds to  $c = -65$  mV (deep voltage reset) and  $d = 8$  (large after-spike jump of  $u$ ).

- *IB (intrinsically bursting)* neurons fire a stereotypical burst of spikes followed by repetitive single spikes (Fig. 2IB). In the model, this corresponds to  $c = -55$  mV (high voltage reset) and  $d = 4$  (large after-spike jump of  $u$ ). During the initial burst, variable  $u$  builds up and eventually switches the dynamics from bursting to spiking.
- *CH (chattering)* neurons can fire stereotypical bursts of closely spaced spikes. The inter-burst frequency can be as high as 40 Hz. In the model, this corresponds to  $c = -50$  mV (very high voltage reset) and  $d = 2$  (moderate after-spike jump of  $u$ ).

All **inhibitory** cortical cells are divided into the following two classes [4]:

- *FS (fast spiking)* neurons can fire periodic trains of action potentials with extremely high frequency practically without any adaptation (slowing down), as one can see in Fig. 2FS. In the model, this corresponds to  $a = 0.1$  (fast recovery).
- *LTS (low-threshold spiking)* neurons can also fire high-frequency trains of action potentials (Fig. 2LTS), but with a noticeable spike frequency adaptation. These neurons have low firing thresholds, which is accounted for by  $b = 0.25$  in the model. To achieve a better quantitative fit with real LTS neurons, other parameters of the model need to be changed as well.

In addition, our model can easily reproduce behavior of thalamo-cortical neurons, which provide the major input to the cortex

- *TC (thalamo-cortical)* neurons have two firing regimes: When at rest ( $v$  is around  $-60$  mV) and then depolarized, they exhibit

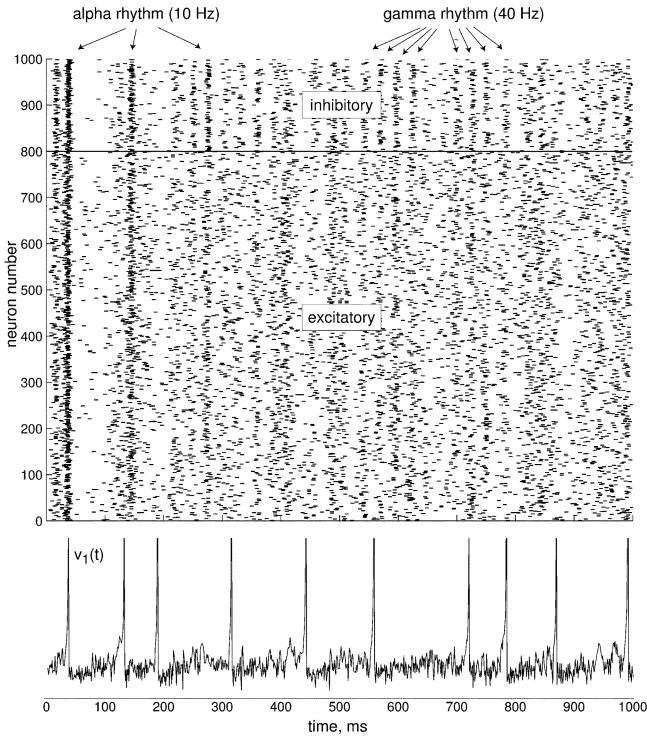


Fig. 3. Simulation of a network of 1000 randomly coupled spiking neurons. Top: spike raster shows episodes of alpha and gamma band rhythms (vertical lines). Bottom: typical spiking activity of an excitatory neuron. All spikes were equalized at +30 mV by resetting  $v_1$  first to +30 mV and then to  $c$ .

tonic firing as in Fig. 2TC, left voltage trace. However, if a negative current step is delivered so that the membrane potential is hyperpolarized ( $v$  is around  $-90$  mV), the neurons fire a rebound burst of action potentials, as in Fig. 2TC, right voltage trace.

The model can exhibit other interesting types of dynamics.

- *RZ (resonator)* neurons have damped or sustained subthreshold oscillations, as in Fig. 2RZ. They resonate to rhythmic inputs having appropriate frequency (as the resonate-and-fire model [9]). This behavior corresponds to  $a = 0.1$  and  $b = 0.26$ . Notice that there is a bistability of resting and repetitive spiking states: The neuron can be switched between the states by an appropriately timed brief stimuli.

Dynamics of other neuronal types, including those in brainstem, hippocampus, basal ganglia, and olfactory bulb, can also be described by our model.

Our “one-fits-all” choice of the function  $0.04v^2 + 5v + 140$  in (1) is justified when large-scale networks of spiking neurons are simulated, as we discuss below. However, if one is interested in the behavior of a single neuron, then other choices of the function are available, and sometimes more preferable. For example, the function  $0.04v^2 + 4.1v + 108$  with  $b = -0.1$  is a better choice for the RS neuron, since it leads to the saddle-node on invariant circle bifurcation and Class 1 excitability [10].

#### IV. PULSE-COUPLED IMPLEMENTATION

We have used this model to simulate a sparse network of 10 000 spiking cortical neurons with 1 000 000 synaptic connections in real time (resolution 1 ms) using a 1 GHz desktop PC and C++ programming language. The following MATLAB program (also available on author’s webpage) simulates a network of randomly connected 1000 neurons in real time. Motivated by the anatomy of a mammalian cortex,

we choose the ratio of excitatory to inhibitory neurons to be 4 to 1, and we make inhibitory synaptic connections stronger. Besides the synaptic input, each neuron receives a noisy thalamic input.

In principle, one can use RS cells to model all excitatory neurons and FS cells to model all inhibitory neurons. The best way to achieve heterogeneity (so that different neurons have different dynamics), is to assign each excitatory cell  $(a_i, b_i) = (0.02, 0.2)$  and  $(c_i, d_i) = (-65, 8) + (15, -6)r_i^2$ , where  $r_i$  is a random variable uniformly distributed on the interval  $[0, 1]$ , and  $i$  is the neuron index. Thus,  $r_i = 0$  corresponds to regular spiking (RS) cell, and  $r_i = 1$  corresponds to the chattering (CH) cell. We use  $r_i^2$  to bias the distribution toward RS cells. Similarly, each inhibitory cell has  $(a_i, b_i) = (0.02, 0.25) + (0.08, -0.05)r_i$  and  $(c_i, d_i) = (-65, 2)$ .

The model belongs to the class of pulse-coupled neural networks (PCNN): The synaptic connection weights between the neurons are given by the matrix  $S = (s_{ij})$ , so that firing of the  $j$ th neuron instantaneously changes variable  $v_i$  by  $s_{ij}$ .

```
% Created by Eugene M. Izhikevich, February 25, 2003
% Excitatory neurons      Inhibitory neurons
Ne=800;                   Ni=200;
re=rand(Ne,1);            ri=rand(Ni,1);
a=[0.02*ones(Ne,1);      0.02+0.08*ri];
b=[0.2*ones(Ne,1);       0.25-0.05*ri];
c=[-65+15*re.^2;        -65*ones(Ni,1)];
d=[8-6*re.^2;           2*ones(Ni,1)];
S=[0.5*rand(Ne+Ni,Ne),  -rand(Ne+Ni,Ni)];

v=-65*ones(Ne+Ni,1);      % Initial values of v
u=b.*v;                  % Initial values of u
firings=[];              % spike timings

for t=1:1000              % simulation of 1000 ms
    I=[5*randn(Ne,1);2*randn(Ni,1)]; % thalamic input
    fired=find(v>=30);    % indices of spikes
    firings=[firings; t+0*fired,fired];
    v(fired)=c(fired);
    u(fired)=u(fired)+d(fired);
    I=I+sum(S(:,fired),2);
    v=v+0.5*(0.04*v.^2+5*v+140-u+I); % step 0.5 ms
    v=v+0.5*(0.04*v.^2+5*v+140-u+I); % for numerical
    u=u+a.*(b.*v-u);      % stability
end;
plot(firings(:,1),firings(:,2),'.');
```

One can see in Fig. 3 that the network exhibits cortical-like asynchronous dynamics; that is, neurons fire Poisson spike trains with mean firing rates around 8 Hz. Dark vertical lines indicate that there are occasional episodes of synchronized firings in the alpha and gamma frequency range (10 and 40 Hz, respectively). Although the network is connected randomly and there is no synaptic plasticity, the neurons self-organize into assemblies and they exhibit collective rhythmic behavior in the frequency range corresponding to that of the mammalian cortex in the awake state. Changing the relative strength of synaptic connections and the strength of the thalamic drive can produce other types of collective behavior, including spindle waves and sleep oscillations. We can easily observe and study these cortical states because our simple spiking model describes *accurately* dynamics of known types of cortical neurons. Thus, *there is no longer a contradiction between biological plausibility and computational efficiency of model neural networks.*

## V. CONCLUSION

In this paper, a simple model that reproduces the rich behavior of biological neurons, including spiking, bursting, and mixed mode firing patterns, post-inhibitory (rebound) spikes and bursts, continuous spiking with frequency adaptation, spike threshold variability, bistability of resting and spiking states, and subthreshold oscillations and resonance is presented (the latter are discussed in [7] and [9]).

Our model is the simplest possible model that can reproduce these types of neuronal behavior: It consists of only two equations and has only one nonlinear term, i.e.,  $v^2$ . Yet, the model is *canonical* in the sense that the difference between it and a whole class of biophysically detailed and accurate Hodgkin–Huxley-type models, including those consisting of enormous number of equations and taking into account all possible information about ionic currents, is just a matter of coordinate change [6].

We show how to use the model to build networks of spiking neurons capable of exhibiting collective dynamics and rhythms similar to those of the mammalian cortex. Due to the extreme computational simplicity of the model, we can simulate thalamo-cortical networks consisting of tens of thousands of spiking neurons in real time with 1 ms resolution using an old 1-GHz desktop PC.

## ACKNOWLEDGMENT

The author thanks F. C. Hoppensteadt, D. Golomb, R. Pichevar, E. W. Gall, A. I. K. Seth, J. R. Iversen, and Y. Chen, who read the first draft of the manuscript and made a number of useful suggestions.

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## Global and Partial Synchronism in Phase-Locked Loop Networks

L. H. A. Monteiro, N. C. F. Canto, J. G. Chaui-Berlinck, F. M. Orsatti, and J. R. C. Piqueira

**Abstract**—We analytically investigate the existence of global and partial synchronism in neural networks where each node is represented by a phase oscillator. Partial synchronism, which is important to pattern recognition, can be caused by increasing the natural frequency of an oscillator and restricting the frequencies of others in certain ranges.

**Index Terms**—Phase-locked loop (PLL), phase oscillator, synchronism, visual cortex.

## I. INTRODUCTION

Hoppensteadt and Izhikevich [1] and Wang [2] have proposed network models where the neural activity is described by differential equations. Both architectures can be used for pattern recognition via associative memory, which occurs when a group of neurons fires synchronously. These models were inspired on findings and ideas about the functioning of the mammalian visual cortex.

It is known that different features of an object appearing in a visual scene, as its color, shape, velocity, and the direction of its motion, are processed in different cortical areas (e.g., [3]). Several authors have proposed that these features are linked through temporal correlations of neural activities (e.g., [4]). Thus, each feature is represented by a neural group oscillating in a synchronized way, and distinct features of the same object are simultaneously represented by distinct synchronous groups. Neural groups corresponding to different objects must be desynchronized from each other, in order to avoid ambiguous conjunctions. Experimental observations in mammals seem to support this theory (e.g., [5]).

In the Wang model [2], the activity of a cortical column is described by two nonlinear differential equations developed by Wilson and Cowan [6] representing the interactions between two populations of neurons that are distinguished by the fact that their synapses are either excitatory or inhibitory.

In the Hoppensteadt-Izhikevich model [1], the activity of a cortical column is represented by the nonlinear differential equation of a phase-locked loop (PLL) (e.g., [7], [8]) describing the temporal evolution of the phase associated to the oscillation.

In both network models, the connectivity among nodes follows simple laws. In this sense, these networks are classified as regular. Despite this regularity, both networks are considered intractable analytically, because it is a very hard task to find expressions relating the parameter values of the oscillators in order to assure global or partial synchronism.

We present analytical results about a simple version of the Hoppensteadt-Izhikevich network. We try to give some hints for answering the

Manuscript received January 31, 2003; revised April 4, 2003. The work of L. H. A. Monteiro and J. R. C. Piqueira was supported by CNPq.

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Digital Object Identifier 10.1109/TNN.2003.820441