Which Model to Use for Cortical Spiking Neurons?

Eugene M. Izhikevich

Abstract—We discuss the biological plausibility and computational efficiency of some of the most useful models of spiking and bursting neurons. We compare their applicability to large-scale simulations of cortical neural networks.

Index Terms—Chaos, Hodgkin–Huxley, pulse-coupled neural network (PCNN), quadratic integrate-and-fire (I&F), spike-timing.

I. INTRODUCTION

DURING last few years we have witnessed a shift of the emphasis in the artificial neural network community toward spiking neural networks. Motivated by biological discoveries, many studies (see this volume) consider pulse-coupled neural networks with spike-timing as an essential component in information processing by the brain.

In any study of network dynamics, there are two crucial issues which are: 1) what model describes spiking dynamics of each neuron and 2) how the neurons are connected. Inappropriate choice of the spiking model or the connectivity may lead to results having nothing to do with the information processing by the brain. In this paper, we consider the first issue, i.e., we compare and contrast various models of spiking neurons.

In Section II and Fig. 1, we review important neuro-computational features of real neurons and their contribution to temporal coding and spike-timing information processing. In Section III, we consider various models of spiking neurons and rank them according to: 1) the number of neuro-computational features they can reproduce; and 2) their implementation efficiency, i.e., the number of floating-point operations (addition, multiplication, etc.) needed to simulate the model during a 1 ms time span. The results of our comparison are summarized in Fig. 2. We compare the utility of the models to large-scale simulations of cortical networks in Section IV.

II. NEURO-COMPUTATIONAL FEATURES

In Fig. 1, we review 20 of the most prominent features of biological spiking neurons. The goal of this section is to illustrate the richness and complexity of spiking behavior of individual neurons in response to simple pulses of dc current. What happens when only tens (let alone billions) of such neurons are coupled together is beyond our comprehension. Using some of the models discussed in Section III, the reader can simulate thousands of cortical neurons in real time with 1 ms resolution.

A. Tonic Spiking

Most neurons are excitable, that is, they are quiescent but can fire spikes when stimulated. To test this property, neurophysiologists inject pulses of dc current via an electrode attached to the neuron and record its membrane potential. The input current and the neuronal response are usually plotted one beneath the other, as in Fig. 1(a). While the input is on, the neuron continues to fire a train of spikes. This kind of behavior, called tonic spiking, can be observed in the three types of cortical neurons: regular spiking (RS) excitatory neurons, low-threshold spiking (LTS), and fast spiking (FS) inhibitory neurons [1], [6]. Continuous firing of such neurons indicate that there is a persistent input.

B. Phasic Spiking

A neuron may fire only a single spike at the onset of the input, as in Fig. 1(b), and remain quiescent afterwards. Such a response is called phasic spiking, and it is useful for detection of the beginning of stimulation.

C. Tonic Bursting

Some neurons, such as the chattering neurons in cat neocortex [7], fire periodic bursts of spikes when stimulated, as in Fig. 1(c). The interburst (i.e., between bursts) frequency may be as high as 50 Hz, and it is believed that such neurons contribute to gamma-frequency oscillations in the brain.

D. Phasic Bursting

Similarly to the phasic spikers, some neurons are phasic bursters, as in Fig. 1(d). Such neurons report the beginning of the stimulation by transmitting a burst.

There are three major hypothesis on the importance of bursts in the brain which are: 1) bursts are needed to overcome the synaptic transmission failure and reduce neuronal noise [20]; 2) Bursts can transmit saliency of the input, because the effect of a burst on the postsynaptic neuron is stronger than the effect of a single spike; and 3) bursts can be used for selective communication between neurons [14], where the interspike frequency within the bursts encodes the channel of communication. A good model of a cortical neuronal network cannot neglect bursting neurons.

E. Mixed Model (Bursting Then Spiking)

Intrinsically bursting (IB) excitatory neurons in mammalian neocortex [1] can exhibit a mixed type of spiking activity depicted in Fig. 1(e). They fire a phasic burst at the onset of stimulation and then switch to the tonic spiking mode. It is not clear what kind of computation such a neuron can do in addition to detecting the onset and reporting the extent of stimulation.
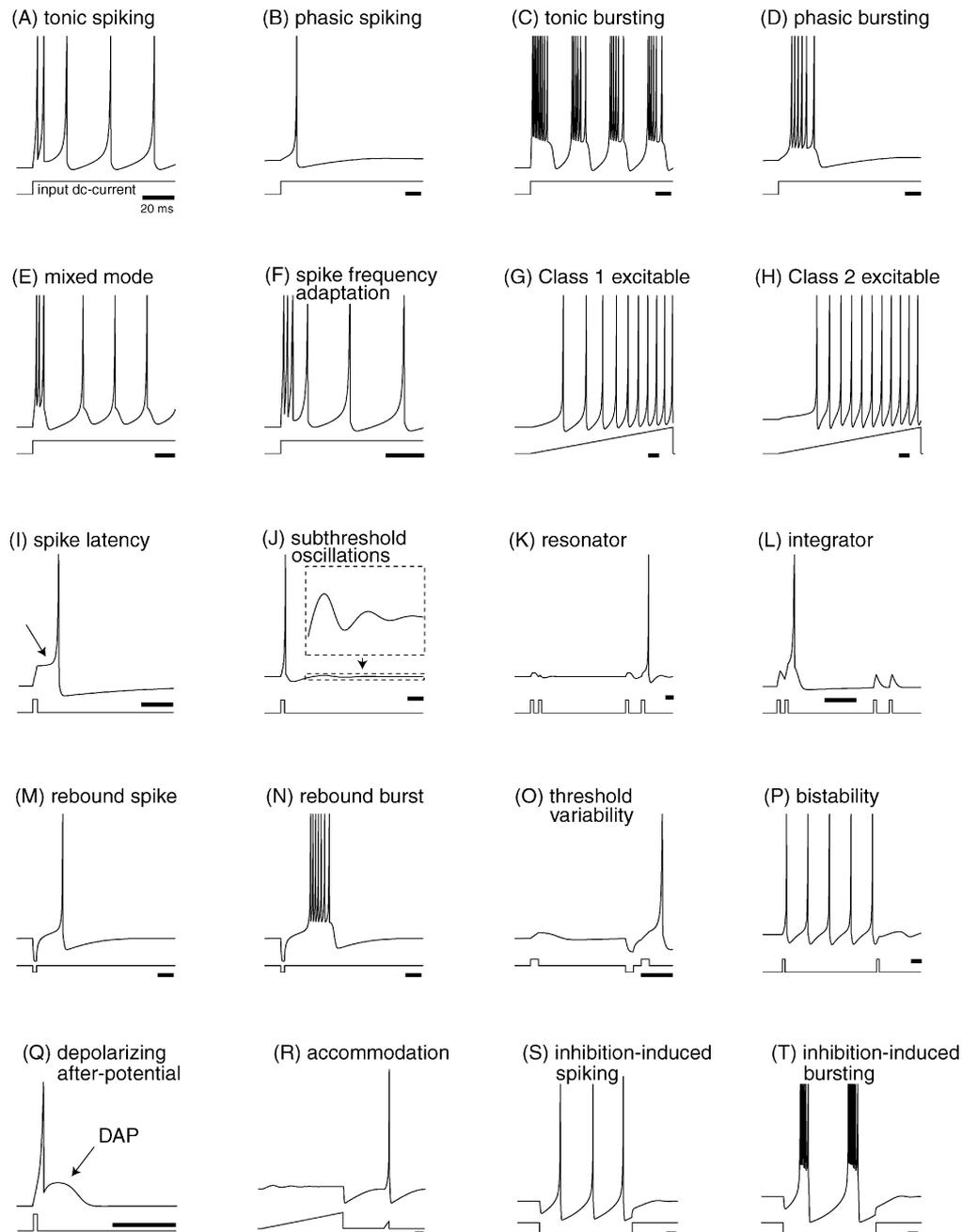
Fig. 1. Summary of the neuro-computational properties of biological spiking neurons. Shown are simulations of the same model (1) and (2), with different choices of parameters. Each horizontal bar denotes a 20-ms time interval. The MATLAB file generating the figure and containing all the parameters, as well as interactive matlab tutorial program can be downloaded from the author's website. This figure is reproduced with permission from www.izhikevich.com. (Electronic version of the figure and reproduction permissions are freely available at www.izhikevich.com).

F. Spike Frequency Adaptation

The most common type of excitatory neuron in mammalian neocortex, namely the regular spiking (RS) cell, fires tonic spikes with decreasing frequency, as in Fig. 1(f). That is, the frequency is relatively high at the onset of stimulation, and then it adapts. Low-threshold spiking (LTS) inhibitory neurons also have this property. The interspike frequency of such cells may encode the time elapsed since the onset of the input.

G. Class 1 Excitability

The frequency of tonic spiking of neocortical RS excitatory neurons depends on the strength of the input, and it may span

the range from 2 Hz to 200 Hz, or even greater. The ability to fire low-frequency spikes when the input is weak (but superthreshold) is called Class 1 excitability [8], [17], [22]. Class 1 excitable neurons can encode the strength of the input into their firing rate, as in Fig. 1(g).

H. Class 2 Excitability

Some neurons cannot fire low-frequency spike trains. That is, they are either quiescent or fire a train of spikes with a certain relatively large frequency, say 40 Hz, as in Fig. 1(h). Such neurons are called Class 2 excitable [8], [17], [22]. Their firing rate is a poor predictor of the strength of stimulation.

I. Spike Latency

Most cortical neurons fire spikes with a delay that depends on the strength of the input signal. For a relatively weak but superthreshold input, the delay, also called spike latency, can be quite large, as in Fig. 1(i). The RS cells in mammalian cortex can have latencies of tens of ms. Such latencies provide a spike-timing mechanism to encode the strength of the input.

J. Subthreshold Oscillations

Practically every brain structure has neurons capable of exhibiting oscillatory potentials, as in Fig. 1(j). The frequency of such oscillations play an important role and such neurons act as bandpass filters, as we discuss next.

K. Frequency Preference and Resonance

Due to the resonance phenomenon, neurons having oscillatory potentials can respond selectively to the inputs having frequency content similar to the frequency of subthreshold oscillations. Such neurons can implement frequency-modulated (FM) interactions and multiplexing of signals [14], [16]. In Fig. 1(k), we stimulate such a neuron with two doublets (pairs of spikes) having different interspike frequencies. The neuron responds only to the doublet whose frequency resonates with the frequency of subthreshold oscillations. Such neurons are called resonators.

L. Integration and Coincidence Detection

Neurons without oscillatory potentials act as integrators. They prefer high-frequency input; the higher the frequency the more likely they fire, as in Fig. 1. This can be useful for detecting coincident or nearly coincident spikes.

M. Rebound Spike

When a neuron receives and then is released from an inhibitory input, it may fire a post-inhibitory (rebound) spike, as in Fig. 1(m). This phenomenon is related to the anodal break excitation in excitable membranes. Many spiking neurons can fire in response to brief inhibitory inputs thereby blurring the difference between excitation and inhibition.

N. Rebound Burst

Some neurons, including the thalamo-cortical cells, may fire post-inhibitory bursts, as in Fig. 1(n). It is believed that such bursts contribute to the sleep oscillations in the thalamo-cortical system.

O. Threshold Variability

A common misconception in the artificial neural network community is the belief that spiking neurons have a fixed voltage threshold. It is well-known that biological neurons have a variable threshold that depends on the prior activity of the neurons. In Fig. 1(o), we first stimulate a neuron with a brief excitatory pulse of current that produces 10 mV depolarization. The neuron does not fire, hence, the input is subthreshold. Then, we apply a brief inhibitory input and then exactly the same "subthreshold" pulse of current. The neuron fires the second time because its "threshold" was lowered by the preceding inhibitory input. Hence, the same 10-mV depolarization can be subthreshold or superthreshold depending on the prior activity. Interestingly, a preceding excitatory pulse might raise the threshold and make the neuron less excitable.

P. Bistability of Resting and Spiking States

Some neurons can exhibit two stable modes of operation: resting and tonic spiking (or even bursting). An excitatory or inhibitory pulse can switch between the modes, as in Fig. 1(p), thereby creating an interesting possibility for bistability and short-term memory. Notice that to switch from the tonic spiking to resting mode, the input must arrive at an appropriate phase of oscillation, thereby emphasizing the importance of spike-timing in such information processing.

Q. Depolarizing After-Potentials

After firing a spike, the membrane potential of a neuron may exhibit a prolonged after-hyperpolarization (AHP) as, e.g., in Fig. 1(b), I or M, or a prolonged depolarized after-potential (DAP), as in Fig. 1(q). Such DAPs can appear because of dendritic influence, because of a high-threshold inward currents activated during the spike, or because of an interplay between subthreshold voltage-gated currents. In any case, such a neuron has shortened refractory period and it becomes superexcitable.

R. Accommodation

Neurons are extremely sensitive to brief coincident inputs, but may not fire in response to a strong but slowly increasing input, as we illustrate in Fig. 1(r). The slowly ramped current in the figure does not elicit a spike, while a smaller but sharply ramped current elicits a spike. During the slow ramp, the inward currents have enough time to inactivate and outward currents have enough time to activate, so the neuron accommodates, becomes less excitable and cannot generate a spike.
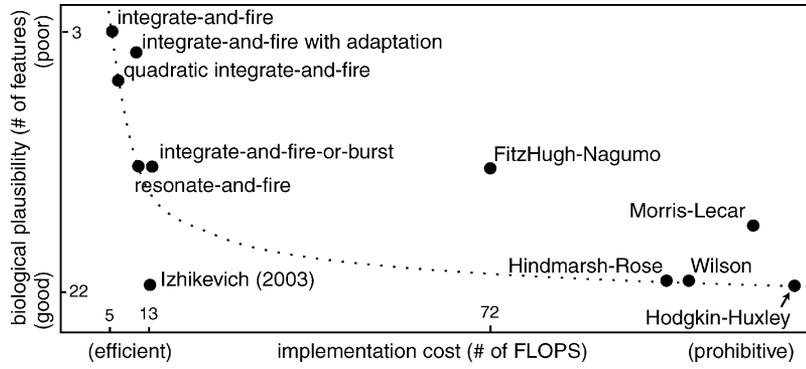
S. Inhibition-Induced Spiking

A bizarre feature of many thalamo-cortical neurons is that they are quiescent when there is no input, but fire when hyperpolarized by an inhibitory input or an injected current, as we illustrate in Fig. 1(s). This happens because the injected current activates the h-current and deinactivates calcium T-current, leading to tonic spiking.

T. Inhibition-Induced Bursting

Instead of spiking, a thalamo-cortical neuron can fire tonic bursts of spikes in response to a prolonged hyperpolarization, as in Fig. 1(t). It is believed that such bursting takes place during spindle wave oscillations in the thalamo-cortical system and it plays an important role in sleep rhythms.

No model should exhibit all these 20 neurocomputational properties simultaneously simply because some of the properties are mutually exclusive. For example, a neuron cannot be an integrator and a resonator at the same time. However, there are models that can easily be tuned to exhibit each such property. For example, all of the neuronal responses in Fig. 1 were obtained using a simple spiking model having four easily tunable parameters [15].

Scatter plot — axes: biological plausibility (# of features) (poor → good, values 3 … 22) versus implementation cost (# of FLOPS) (efficient → prohibitive, values 5, 13 … 72). Plotted models:

- integrate-and-fire
- integrate-and-fire with adaptation
- quadratic integrate-and-fire
- integrate-and-fire-or-burst
- resonate-and-fire
- Izhikevich (2003)
- FitzHugh-Nagumo
- Morris-Lecar
- Hindmarsh-Rose
- Wilson
- Hodgkin-Huxley

Models	biophysically meaningful	tonic spiking	phasic spiking	tonic bursting	phasic bursting	mixed mode	spike frequency adaptation	class 1 excitable	class 2 excitable	spike latency	subthreshold oscillations	resonator	integrator	rebound spike	rebound burst	threshold variability	bistability	DAP	accommodation	inhibition-induced spiking	inhibition-induced bursting	chaos	# of FLOPS
integrate-and-fire	-	+	-	-	-	-	-	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	5
integrate-and-fire with adapt.	-	+	-	-	-	-	+	+	-	-	-	-	+	-	-	-	-	+	-	-	-	-	10
integrate-and-fire-or-burst	-	+	+		+	-	+	+	-	-	-	-	+	+	+	-	+	+	-	-	-		13
resonate-and-fire	-	+	+	-	-	-	-	+	+	-	+	+	+	+	-	-	+	+	+	-	-	+	10
quadratic integrate-and-fire	-	+	-	-	-	-	-	+	-	+	-	-	+	-	-	+	+	-	-	-	-	-	7
Izhikevich (2003)	-	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	13
FitzHugh-Nagumo	-	+	+	-		-	-	+	-	+	+	+	-	+	-	+	+	-	+	+	-	-	72
Hindmarsh-Rose	-	+	+	+			+	+	+	+	+	+	+	+	+	+	+	+	+			+	120
Morris-Lecar	+	+	+	-		-	-	+	+	+	+	+	+	+		+	+	-	+	+	-	-	600
Wilson	-	+	+	+			+	+	+	+	+	+	+	+	+	+		+	+				180
Hodgkin-Huxley	+	+	+	+			+	+	+	+	+	+	+	+	+	+	+	+	+			+	1200

Fig. 2. Comparison of the neuro-computational properties of spiking and bursting models; see Fig. 1. "# of FLOPS" is an approximate number of floating point operations (addition, multiplication, etc.) needed to simulate the model during a 1 ms time span. Each empty square indicates the property that the model should exhibit in principle (in theory) if the parameters are chosen appropriately, but the author failed to find the parameters within a reasonable period of time.

III. SPIKING MODELS

Below we review some widely used models of spiking and bursting neurons that can be expressed in the form of ordinary differential equations (ODE) (thus, we exclude the spike response model [5]). In addition to the 20 neuro-computational features reviewed above, we also consider whether the models have biophysically meaningful and measurable parameters, and whether they can exhibit autonomous chaotic activity. We start with the simplest models first. The summary of our comparison is in Fig. 2.

Throughout this section, v denotes the membrane potential and v' denotes its derivative with respect to time. All the parameters in the models are chosen so that v has mV scale and the time has ms scale. To compare computational cost, we assume that each model, written as a dynamical system $\dot{x} = f(x)$, is implemented using a fixed-step first-order Euler method $x(t + \tau) = x(t) + \tau f(x(t))$ with the integration time step τ chosen to achieve a reasonable numerical accuracy.

A. I&F

One of the most widely used models in computational neuroscience is the leaky integrate-and-fire (I&F) neuron

$$v' = I + a - bv, \text{ if } v \geq v_{\text{thresh}}, \text{ then } v \leftarrow c$$

where v is the membrane potential, I is the input current, and a, b, c, and v_{thresh} are the parameters. When the membrane potential v reaches the threshold value v_{thresh}, the neuron is said to fire a spike, and v is reset to c.

The I&F neuron is Class 1 excitable; it can fire tonic spikes with constant frequency, and it is an integrator. It is the simplest

model to implement when the integration time step τ is 1 ms. Indeed, the iteration $v(t+1) = v(t)+(I+a-bv(t))$ takes only four floating-point operations (additions, multiplications, etc.) plus one comparison with the threshold v_{thresh}. Because I&F has only one variable, it cannot have phasic spiking, bursting of any kind, rebound responses, threshold variability, bistability of attractors, or autonomous chaotic dynamics. Because of the fixed threshold, the spikes do not have latencies. In summary, despite its simplicity, I&F is one of the worst models to use in simulations, unless one wants to prove analytical results.

B. I&F with Adaptation

The I&F model is one-dimensional (1-D), hence it cannot burst or have other properties of cortical neurons. One may think that having a second linear equation

$$v' = I + a - bv + g(d - v)$$
$$g' = \frac{(e\delta(t) - g)}{\tau}$$

describing activation dynamics of a high-threshold K current can make an improvement, e.g., endow the model with spike-frequency adaptation. Indeed, each firing increases the K activation gate g via Dirac delta function δ and produces an outward current that slows down the frequency of tonic spiking. Simulations of this model take 10 floating point operations/1 ms time step, yet the model still lacks many important properties of cortical spiking neurons.

C. Integrate-and-Fire-or-Burst

Smith and coauthors [24] suggested an improvement—integrate-and-fire-or-burst (I&FB) model

$$v' = I + a - bv + gH(v - v_h)h(v_T - v)$$

if $v = v_{\text{thresh}}$, then $v \leftarrow c$

$$h' = \begin{cases} \frac{-h}{\tau^-}, & \text{if } v > v_h \\ \frac{(1-h)}{\tau^+}, & \text{if } v < v_h \end{cases}$$

to model thalamo-cortical neurons. Here h describes the inactivation of the calcium T-current, g, v_h, v_T, τ^+, and τ^- are parameters describing dynamics of the T-current, and H is the Heaviside step function.

Having this kind of a second variable creates the possibility for bursting and other interesting regimes summarized in Fig. 2. But this comes with a price: It takes between 9 and 13 operations (depending on the value of v) to simulate 1 ms of the model.

D. Resonate-and-Fire

The resonate-and-fire neuron [16] is a two-dimensional (2-D) analogue of the I&F neuron

$$z' = I + (b + \iota\omega)z$$
$$\text{if } \text{Im } z = a_{\text{thresh}}, \qquad \text{then } z \leftarrow z_0(z)$$

where the real part of the complex variable z is the membrane potential. Here b, ω, and a_{thresh} are parameters, and $z_0(z)$ is

an arbitrary function describing activity-dependent after-spike reset. The resonate-and-fire model is simple and efficient—it takes 10 operations to simulate 1 ms. When the frequency of oscillation $\omega = 0$, it becomes an integrator. Its neuro-computational properties are summarized in Fig. 2.

E. Quadratic I&F

An alternative to the leaky I&F neuron is the quadratic I&F neuron, also known as the theta-neuron [2], [3] or the Ermentrout–Kopell canonical model [10] (when it is written in a trigonometric form). We present it here following [19]

$$v' = I + a(v - v_{\text{rest}})(v - v_{\text{thresh}})$$

if $v = v_{\text{peak}}, \qquad$ then $v \leftarrow v_{\text{reset}}$

where v_{rest} and v_{thresh} are the resting and threshold values of the membrane potential. This model is canonical in the sense that any Class 1 excitable system described by smooth ODEs can be transformed into this form by a continuous change of variables [18]. It takes only seven operations to simulate 1 ms of the model, and this should be the model of choice when one simulates large-scale networks of integrators. Unlike its linear analogue, the quadratic I&F neuron has spike latencies, activity-dependent threshold (which is v_{thresh} only when $I = 0$), and bistability of resting and tonic spiking modes.

F. Spiking Model by Izhikevich (2003)

All of the responses in Fig. 1 were obtained using a simple model of spiking neurons proposed recently by Izhikevich [15]

$$v' = 0.04v^2 + 5v + 140 - u + I \tag{1}$$
$$u' = a(bv - u) \tag{2}$$

with the auxiliary after-spike resetting

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

Here variable 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 $(+30 \text{ mV})$, the membrane voltage and the recovery variable are reset according to the (3). If v skips over 30, then it first is reset to 30, and then to c so that all spikes have equal magnitudes. The part $0.04v^2 + 5v + 140$ is chosen so that v has mV scale and the time has ms scale. Geometrical derivation of the model based on fast and slow nullclines can be found in [11].

The model can exhibit firing patterns of all known types of cortical neurons with the choice of parameters a, b, c, and d given in [15]. It takes only 13 floating point operations to simulate 1 ms of the model, so it is quite efficient in large-scale simulations of cortical networks. When $(a, b, c, d) = (0.2, 2, -56, -16)$ and $I = -99$, the model has chaotic spiking activity, though the integration time step τ should be small to achieve adequate numerical precision.

We stress that $+30$ mV in (3) is not a threshold, but the peak of the spike. The threshold value of the model neuron is between -70 and -50, and it is dynamic, as in biological neurons. To build intuition and understanding of the dynamics of the model, the reader is advised to download an interactive MATLAB tutorial program from the author's webpage and play with the model and its parameters. In particular, the reader could explore all 20 neuro-computational properties in Fig. 1.

G. FitzHugh–Nagumo

The parameters in the FitzHugh–Nagumo model [4]

$$v' = a + bv + cv^2 + dv^3 - u$$
$$u' = \varepsilon(ev - u)$$

can be tuned so that the model describes spiking dynamics of many resonator neurons. Its neuro-computational properties are summarized in Fig. 2. Since one needs to simulate the shape of each spike, the time step in the model must be relatively small, e.g., $\tau = 0.25$ ms. It takes 18 floating point operations/0.25 ms, hence 72 operations/1 ms of simulation. Since the model is a 2-D system of ODEs without a reset, it cannot exhibit autonomous chaotic dynamics or bursting. Adding noise to this, or some other 2-D models, allows for stochastic bursting.

H. Hindmarsh–Rose

The Hindmarsh–Rose model of thalamic neuron [23] can be written

$$v' = u - F(v) + I - w$$
$$u' = G(v) - u$$
$$w' = \frac{(H(v) - w)}{\tau}$$

where F, G, and H are some functions. Depending on their choice, the model can, in principle, exhibit all of the neuro-computational properties in Fig. 1. The problem is, of course, how to find the functions to model, say RS or LTS neurons. Let us assume that this problem is somehow solved and that the functions are polynomials of the third degree (in the best case). Since we need to simulate the shape of the action potential, the maximal time step is 0.25 ms. Since it takes 30 floating point operations/0.25 ms of simulation time, it would take 120 operations to simulate 1 ms of the model. Again, this is an optimistic assessment that might never be achieved.

I. Morris–Lecar

Morris and Lecar [21] suggested a simple 2-D model to describe oscillations in barnacle giant muscle fiber. Because it has biophysically meaningful and measurable parameters, the model became quite popular in computational neuroscience community. It consists of a membrane potential equation with instantaneous activation of Ca current and an additional equation describing slower activation of K current

$$C\dot{V} = I - g_L(V - V_L) - g_{Ca}m_\infty(V)$$
$$\times (V - V_{Ca}) - g_K n(V - V_K)$$
$$\dot{n} = \lambda(V)(n_\infty(V) - n)$$

where

$$m_\infty(V) = \frac{1}{2}\left\{1 + \tanh\left[\frac{(V - V_1)}{V_2}\right]\right\}$$
$$n_\infty(V) = \frac{1}{2}\left\{1 + \tanh\left[\frac{(V - V_3)}{V_4}\right]\right\}$$
$$\lambda(V) = \bar{\lambda}\cosh\left[\frac{(V - V_3)}{(2V_4)}\right]$$

with parameters: $C = 20\ \mu\text{F/cm}^2$, $g_L = 2\ \text{mmho/cm}^2$, $V_L = -50$ mV, $g_{Ca} = 4\ \text{mmho/cm}^2$, $V_{Ca} = 100$ mV, $g_K = 8\ \text{mmho/cm}^2$, $V_K = -70$ mV, $V_1 = 0$ mV, $V_2 = 15$ mV, $V_3 = 10$ mV, $V_4 = 10$ mV, $\bar{\lambda} = 0.1\ \text{s}^{-1}$, and applied current $I(\mu\text{A/cm}^2)$.

The model can exhibit various types of spiking, but could exhibit tonic bursting only when an additional equation is added, e.g., slow inactivation of Ca current. In this case, the model becomes equivalent to the Hodgkin–Huxley model discussed below (both have transient inward and persistent outward currents).

Because one needs to simulate the shape of the action potential in the Morris–Lecar model, the time step must be significantly smaller than 1 ms. We found that $\tau = 0.1$ ms is the largest step that gives reasonable results when the model is used to simulate cortical spiking neurons. Since the model involves hyperbolic tangents and exponents, it takes around 60 floating point operations to evaluate one 0.1-ms time step, which leads to 600 operations/1 ms of simulation.

J. Wilson Polynomial Neurons

Wilson [25] suggested to model cortical neurons using polynomial equations. His model consists of four differential equations, which we do not provide here. It can exhibit all neuro-computational properties in Fig. 1, provided that the parameters are chosen appropriately, which is not an easy task. The suggested time step in the model was 0.1 ms, though it could be pushed up to 0.25 ms without significant loss of precision or noticeable distortion of the shape of the action potential. It takes 45 floating point operations to evaluate 0.25 ms of the model, hence 180 operations/1 ms.

K. Hodgkin–Huxley

The Hodgkin–Huxley model [9] is one of the most important models in computational neuroscience. It consists of four equations and tens of parameters, not provided here, describing membrane potential, activation of Na and K currents, and inactivation of Na current. Though the model has quite limited behavior for original values of parameters, it can actually exhibit all properties in Fig. 1, if the parameters are tuned.

In general, scientists refer to all conductance-based models as being of the Hodgkin–Huxley-type. Such models are important not only because their parameters are biophysically meaningful and measurable, but also because they allow us to investigate questions related to synaptic integration, dendritic cable filtering, effects of dendritic morphology, the interplay between ionic currents, and other issues related to single cell dynamics.

The model is extremely expensive to implement. It takes 120 floating point operations to evaluate 0.1 ms of model time (assuming that each exponent takes only ten operations), hence, 1200 operations/1 ms. Thus, one can use the Hodgkin–Huxley formalism only to simulate a small number of neurons or when simulation time is not an issue.

IV. CONCLUSION

As the reader can see in Fig. 2, many models of spiking neurons have been proposed. Which one to choose? The answer depends on the type of the problem. If the goal is to study how the neuronal behavior depends on measurable physiological parameters, such as the maximal conductances, steady–state (in)activation functions and time constants, then the Hodgkin–Huxley-type model is the best. Of course, you could simulate only tens of coupled spiking neurons in real time.

In contrast, if you want to simulate thousands of spiking neurons in real time with 1 ms resolution, then there are plenty of models to choose from. The most efficient is the I&F model. However, the model cannot exhibit even the most fundamental properties of cortical spiking neurons, and for this reason it should be avoided by all means. The only advantage of the I&F model is that it is linear, and hence amenable to mathematical analysis. If no attempts to derive analytical results are made, then there is no excuse for using this model in simulations.

The quadratic I&F model is practically as efficient as the linear one, and it exhibits many important properties of real neurons, such as spikes with latencies, and bistability of resting and tonic spiking modes. However, it is 1-D, and hence, it cannot burst and cannot exhibit spike frequency adaptation. Thus, it can be used in simulations of cortical neural networks only when biological plausibility is not a great concern.

If the goal is to understand the fine temporal structure of cortical spike trains, and to use spike-timing as an additional variable to understand how the mammalian neocortex processes information, a spiking model that can exhibit all or most of the 20 neuro-computational properties of biological neurons summarized in Fig. 1 is required. The model recently proposed by Izhikevich [15] was developed exactly for these purposes. It is the simplest possible model that can exhibit all the firing patterns in Fig. 1. Indeed, removal of the (2) makes it 1-D with no possibility for bursting, removal of the term v^2 makes it linear and equivalent to the resonate-and-fire model.

The author has used the model to simulate a fully connected network of 1,000 cortical spiking neurons in real time with 1 ms resolution using only modest computational resources (1 GHz desktop PC; MATLAB code is provided in [15]. Using the C programming language, it is possible to speed up simulations by the factor of twenty). The network exhibited rhythms in the alpha and gamma frequency range, transient and sustained spike synchrony, spindle waves, sleep oscillations, and other temporal phenomena.

This model was also used in a simulation of a network of 100 000 spiking neurons with realistic cortical anatomy, axonal delays, and spike-timing dependent synaptic plasticity (STDP) [13]. Due to the interplay between spiking, plasticity, and delays, the neurons self-organized into *polychronous* (i.e., multiple-timing) groups that could generate persistent time-locked firing patterns with ms precision. There are many such polychronous groups coexisting at the same time. In another study [12], we found that each neuron participates in many polychronous groups, so that the total number of groups the model could memorize can significantly exceed the number of neurons, or even the number of synapses in the network, resulting in unprecedented memory capacity.

In conclusion, having a network of computationally efficient and biologically plausible cortical spiking neurons interconnected according to the principles of known anatomy of the neocortex should be the goal of every scientist exploring information processing in the mammalian brain.

ACKNOWLEDGMENT

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Eugene M. Izhikevich was born in Moscow, Russia, in 1967. He received the Master's degree in applied mathematics and computer sciences from Lomonosov Moscow State University in 1992, and the Ph.D. degree in mathematics from Michigan State University, East Lansing, in 1996.

Eugene is an Associate Fellow at The Neurosciences Institute, La Jolla, CA, where he studies nonlinear dynamics of biological neurons and builds large-scale models of the brain.