Modeling Minimal Hodgkin–Huxley Type Neurons

Regular Spiking, Intrinsically Bursting, and Low-Threshold Spiking

Course: Neural Information Processing

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1 Motivation and Introduction

For my project I decided to tackle the Hodgkin–Huxley task: modeling minimal Hodgkin–Huxley-type models for different classes of neurons. While in this course, and also in Neurodynamics, I learned that complex firing behavior can also be modeled even with simpler models than the HH-type, such as the Leaky Integrate-and-Fire (LIF), Izhikevich, or FitzHugh–Nagumo models. These models are simpler in structure as they use fewer parameters. Therefore, they are computationally more efficient and easier to analyze, for example via phase plane analysis, while still modeling similar mechanisms, albeit in an abstracted form.

However, my main interest lies in the biological mechanisms underlying neural computation—how neurons or the brain physically implement different computational functions. This is why I chose to work with biologically more plausible, less simplified models: Minimal Hodgkin–Huxley-type models as described in Pospischil et al. (2008). These models, while still abstract, reproduce key firing behaviors using ion current dynamics grounded in experimentally measured ion channel kinetics.

On the other hand, they are still simplified. They are single-compartment models, meaning they treat the entire neuron (axon, soma, and dendrite) as a single electrical unit, not accounting for spatial gradients or dendritic computation. Furthermore, real neurons typically express 10–20 or more different ion channel types, far more than these minimal models include. Still, they capture the qualitative mechanisms underlying firing behavior, which I aimed to explore.

For this endeavor I implemented three neuron types with different firing behaviors using the models and parameters provided by Pospischil et al. (2008):

1. Excitatory Regular Spiking (RS) neurons with spike-frequency adaptation,
2. Intrinsically Bursting (IB) neurons with bursting and spike-frequency adaptation,
3. Low-Threshold Spiking (LTS) neurons with rebound bursting caused by hyperpolarization.

In the following report, I first explain the conceptual background of HH-type models, specifically for the three neuron types modeled. After that, I briefly describe the implementation, present my results, and discuss how these different dynamics might support distinct computational functions in neural circuits. Finally, I conclude with a reflection and summary.

2 Conceptual Background

The foundation of minimal HH-type models is Hodgkin and Huxley's groundbreaking work (1952), where they measured and mathematically described how ionic conductances

generate action potentials. Their original model included three types of ion channels: sodium (Na^+), potassium (K^+), and a leak channel.

The model treats a patch of axon membrane as an electrical circuit. A capacitor (C_m) represents the lipid bilayer, which is impermeable to ions, and variable resistors represent voltage-gated ion channels for Na^+ and K^+. Electromotive forces (E_{Na}, E_K, E_L) correspond to equilibrium (Nernst) potentials that result from the balance of concentration and electrical forces across the membrane.

The basic membrane equation is:

$$C_m \frac{dV}{dt} = -\sum_k I_k(t) + I_{inj}(t) \tag{1}$$

Each ionic current I_k follows Ohm's law:

$$I_k = g_k(V - E_k) \tag{2}$$

where g_k is the conductance, which for voltage-gated channels depends on activation and inactivation variables governed by first-order kinetics:

$$\frac{dx}{dt} = \frac{x_\infty(V) - x}{\tau_x(V)} \tag{3}$$

In the original Hodgkin–Huxley model, the total ionic current is:

$$I_{ion} = g_{Na} m^3 h (V - E_{Na}) + g_K n^4 (V - E_K) + g_L (V - E_L) \tag{4}$$

where m, h, and n are gating variables controlling sodium activation/inactivation and potassium activation.

Minimal HH-type Models

Real mammalian neurons, however, exhibit much richer dynamics due to additional ion channel types. Pospischil et al. (2008) proposed a family of *minimal HH-type models* that include only a few essential channels to reproduce experimentally observed firing patterns:

$$C_m \frac{dV}{dt} = -(I_{Na} + I_{Kd} + I_{add} + I_L) + I_{inj}(t) \tag{5}$$

where I_{add} represents additional currents such as I_M, I_T, or I_L (Ca^{2+}).

- **Regular Spiking (RS):** Includes the slow K^+ current I_M, which activates during depolarization. This causes spike-frequency adaptation, where firing slows over time—typical of cortical pyramidal neurons.
- **Intrinsically Bursting (IB):** Adds both I_M and a high-threshold Ca^{2+} current (I_L) that provides a slow depolarizing drive, leading to alternating bursts and silent

phases, characteristic of layer 5 pyramidal neurons.

- **Low-Threshold Spiking (LTS):** Incorporates the low-threshold Ca^{2+} current I_T, which activates after hyperpolarization, producing rebound bursts without the voltage exceeding threshold.

3 Implementation and Results

The models were implemented in Python using NumPy, SciPy's `solve_ivp`, and Matplotlib. Each model is defined as a system of coupled ordinary differential equations (ODEs) describing membrane potential and gating variables.

Although I initially considered using the Euler method, I ultimately employed the Runge–Kutta method via `solve_ivp` for improved numerical stability and speed, especially necessary for the F–I (frequency–current) analysis.

Parameters for each cell type were taken directly from Pospischil et al. (2008), fitted to experimental recordings (see their Figs. 2, 6, and 8). One main challenge was ensuring unit consistency and correct parameter scaling.

Example Output

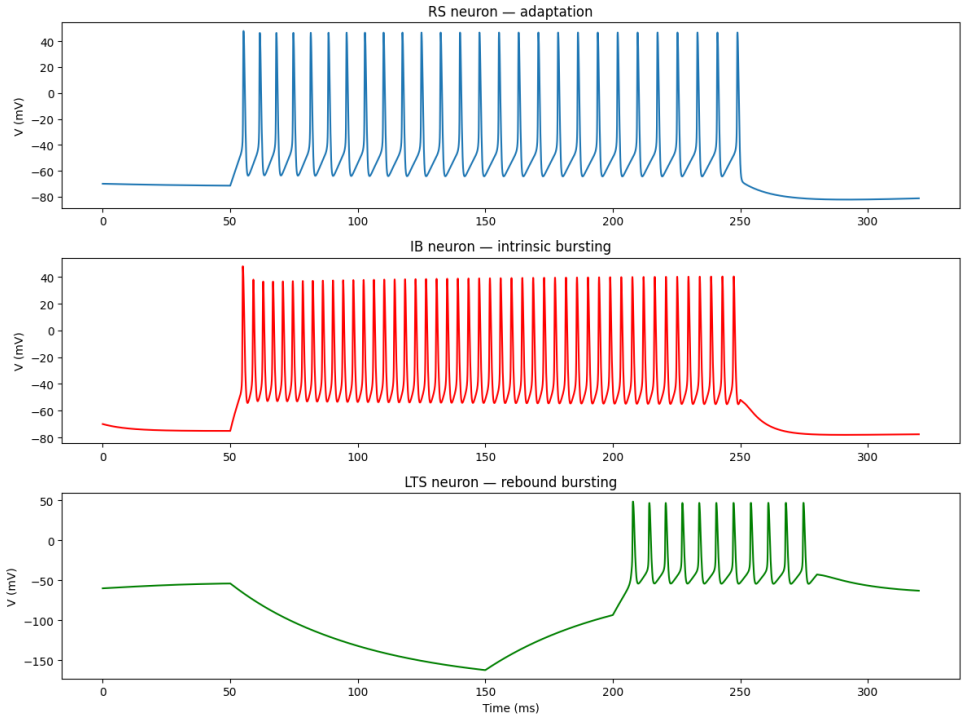

Figure 1: Membrane potential of Regular Spiking neuron showing spike-frequency adaptation with parameters from the paper.

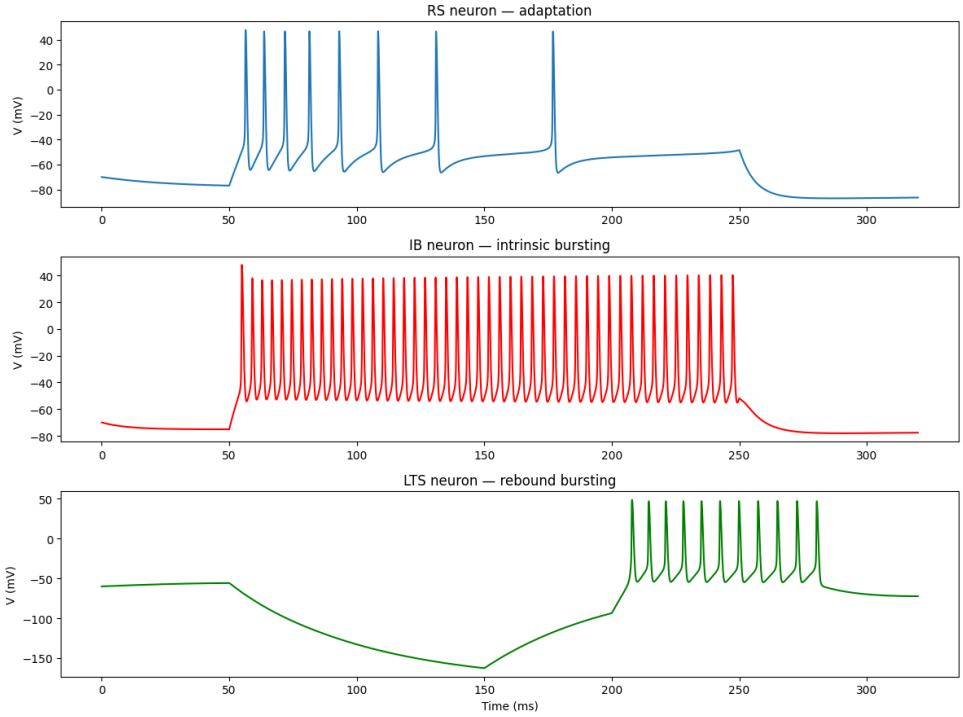

Figure 2: Membrane potential of Regular Spiking neuron showing spike-frequency adaptation with adjusted parameters for more visible behaviour.

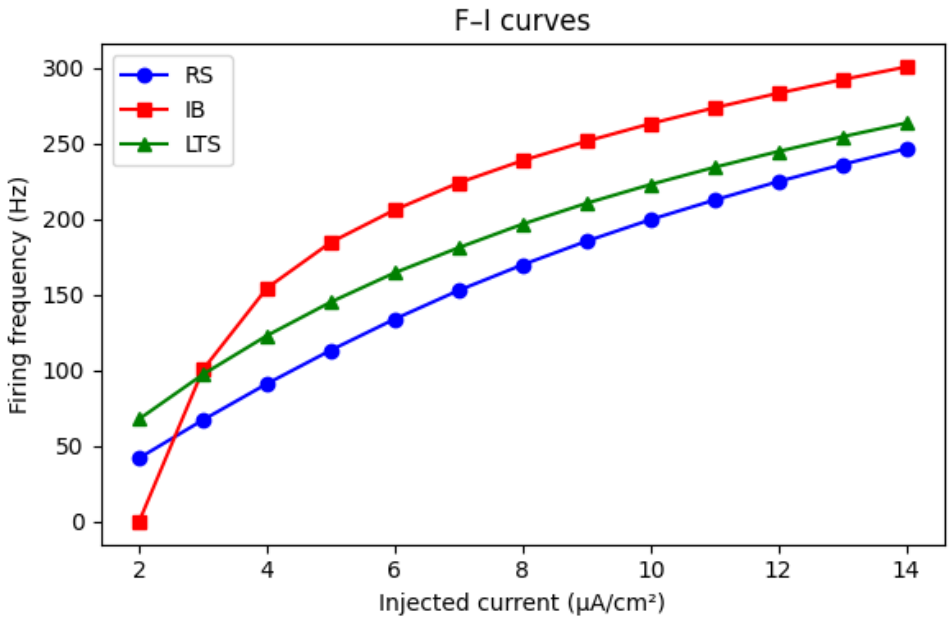

Figure 3: Frequency–current (F–I) curves for each neuron type with paper parameters .

The expected behaviors—frequency adaptation (RS), bursting (IB), and rebound activation (LTS)—were all reproduced. By adjusting parameters such as g_M and $\tau_{\max}$, I could make adaptation more pronounced, gaining intuition for how each parameter shapes firing behavior.

Additionally, I computed frequency–current (F–I) curves for each neuron type to quan-

tify how firing rate depends on input strength.

4 Computational Relevance of Firing Behaviors

The distinct firing dynamics of the RS, IB, and LTS models imply specialized computational roles in neural circuits.

For one, the **Regular Spiking (RS)** neuron can be used for rate coding because its firing rate gradually adapts to a steady input current (via the slow $\mathbf{I_M}$ current), effectively mapping input amplitude to output firing rate. This allows the neuron to act as a gain controller, encoding sustained inputs efficiently while preventing saturation. This behavior can also be used for temporal filtering—detecting temporal changes by passing rapid changes while filtering out slow fluctuations. This is because, due to the slow $\mathbf{I_M}$ current, the neuron is less responsive to sustained or low-frequency inputs but more responsive to transient or high-frequency changes.

The **Intrinsically Bursting (IB)** neurons produce rapid sequences of spikes that convey a strong, temporally precise signal. These bursts are computationally advantageous because they are more likely to drive postsynaptic firing, activate NMDA receptors by maintaining depolarization that lifts the Mg^{2+} block, and thereby trigger dendritic NMDA spikes. This, for example, supports mechanisms such as synaptic plasticity or synchronization in rhythmic or sensory circuits.

The **Low-Threshold Spiking (LTS)** neuron's rebound bursting, mediated by the T-type calcium current $\mathbf{I_T}$, enables firing after inhibition, allowing it to encode not the input itself but the timing of inhibitory release. This property supports oscillatory coordination and phase coding, as seen in thalamocortical and inhibitory networks where timing and rhythmic structure carry critical information.

5 Conclusion and Reflection

In this project, I implemented and explored minimal Hodgkin–Huxley–type models for three neuron classes—RS, IB, and LTS—based on Pospischil et al. (2008). Despite their simplicity, these single-compartment models successfully reproduced key experimental firing behaviors.

Even though we had implemented the basic HH model before in the course, this implementation was demanding, even though almost everything was given by the paper, though now I feel I got better at implementing dynamical systems. I am definitely not done with these models and would like to get better intuition and control to get very specific firing patterns. I played around a lot with the parameters, and in hindsight, I wish I had tracked these better in the Notebook. The implementation has shown me the

usefulness of the simple but biologically more plausible model types for getting a deeper understanding of our underlying biological mechanisms.

References

[1] Pospischil, M., Toledo-Rodriguez, M., Monier, C., et al. (2008). *Minimal Hodgkin–Huxley type models for different classes of cortical and thalamic neurons.* Biological Cybernetics, 99(4), 427–441.

[2] Gerstner, W., Kistler, W. M., Naud, R., & Paninski, L. (2014). *Neuronal Dynamics: From Single Neurons to Networks and Models of Cognition.* Cambridge University Press.