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Analysis of Hodgkin Huxley neuron model using LTspice and MATLAB

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ABSTRACT

Neuromorphic engineering popularly called as neuromorphic computing is a study used to represent analog, digital and software system based artificial neuron that replicate the behavior of biological neuron in terms of perception and motor control. In this paper, first we are going to study the mathematical model of neuron, understanding the Hodgkin Huxley equation to represent the biological neuron and based on this we have simulated the basic pulse type hardware neuron model on LTSpice using Transistors, resistances, capacitances and externally injected input stimulus current. We have also analyzed the Hodgkin Huxley neuron using MATLAB to understand and explore the basic features of biological neuron like its beating nature, action potential, gating variables and conductance of ions in HH equation.

Keywords— ***Neuromorphic Engineering, Biological Neuron, Hodgkin Huxley Equation, Pulse Type Hardware Neuron, Beating Nature, Action Potential, Gating Variables***

1. INTRODUCTION

Spiking neural network (SNN) has come a long way since its origination way back in 1940s. Regardless of the changes, the neuron- representing node remains one of the most essential constituent of neural networks. The most fundamental elements of nervous system are neurons. They are responsible for gathering information from the external environment, send motor commands to our muscles, and transform and relay electrical signals at every step along the way. A typical neuron consists of soma (cell body), dendrites and axon[1][2].

Soma: It is a part of neuron that contains nucleus of the cell and is responsible for collecting the information.

Dendrites: They are the input part of neuron. Soma receives information from other neurons via these thin filaments.

Axon: This is a long cable that delivers the information from soma to the neighbouring cells. It usually ends with so many synapses interacting to adjacent neuron dendrites.

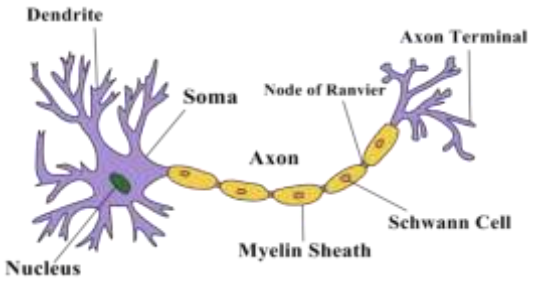

Fig1. Basic Structure of Neuron

When a large number of inputs from other neurons are being collected at one neuron, the signals build up to a certain point. When this signal strength reaches a particular cut-off (threshold), the neuron communicates an impulse through its axon, called action potential. As electrically charged ions moves through the membrane of axon, it gives rise to an action potential. The membrane potential is usually around -70mV[3].

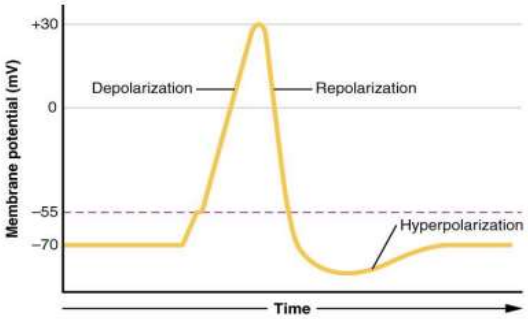

Fig2. Action Potential Graph

As the action potential is reached, sodium(Na^+) channels gets opened and as the concentration of Na^+ is higher outside the cell, a flood of sodium ions begin to enter the cell thus making it more

positive putting it into depolarization state causing the spike to be generated. Although potassium ions(K^+) also move out of the cell, however the voltage required by its channels are higher than Na^+ channels. Therefore, first Na^+ enters cell and creates a rapid increase in potential. This increase causes the K^+ channel to get open and allow the K+ ions to move out of cell . But since the amount of entering Na^+ into the cell is higher than amount of moving K^+ out of cell thus, the membrane potential experiences some net gain. After achieving peak, a refractory period of sodium ion follows and no further sodium ions enter the cell. This means that sodium channels gets shut down and potassium channels kept open thus allowing the membrane voltage to decay causing it to enter into the repolarization state. The K^+ ion channel shuts down when the membrane potential reaches the resting state and at this point sodium channels start leaving the refractory period and reset to its initial phase. After this, potassium and sodium pumps move K^+ back into the cell and Na^+ out of the cell therefore preparing the cell to repeat same phenomena[4][5].

2. MATHEMATICAL MODEL OF A NEURON

We know that working phenomena of a neuron is very complicated. So, to study it effectively, it is good to start with a simple model consisting of electric components. Given below in the Figure 3 is the electric circuit model of a neuron given by Hodgkin and Huxley[6][7].

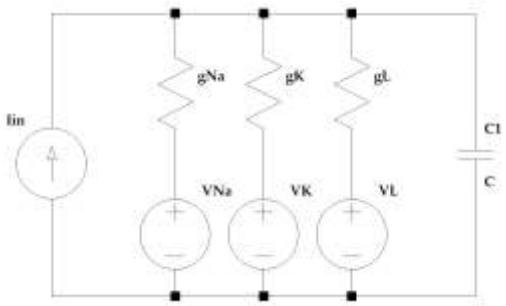

Figure 3: Hodgkin-Huxley's neuron's electrical model

Here, g_{Na} and g_K indicate the ion channel conductances and V_{Na} and V_K indicate sodium and potassium Nernst equilibrium potential[8].

V_L and g_L accounts for the leak currents which is the current obtained as a contribution of all the gates that are always open. Sodium and Potassium gates are membrane potential gated, i.e., their conductances is a function of membrane potential V_m. Leaky gates have some contribution of sodium and potassium ions but also of other ions like calcium, chlorine etc[9]. Therefore,

$$I_{total} = I_K + I_{Na} + I_L \quad -(1)$$

where,

$$I_{Na}(V_m,t) = (V_{Na} - V_m).g_{Na}(V_m,t) \quad -(2)$$

$$I_K(V_m,t) = (V_K - V_m).g_K(V_m,t) \quad -(3)$$

$$I_L(V_m,t) = (V_L - V_m).g_L \quad -(4)$$

As leak gates are not voltage dependent, the conductance g_L remains constant. The various conductances in this model varies as follows:-

$$g = g_{max}.f(V_m,t) \quad -(5)$$

where,

f is the fraction of channels open, which depends on V_m and t, g is the conductance of a particular ion at any given V_m and t, and g_{max} is the maximum conductance of that particular ion channel.

If all the channels for a particular ion are open, the value of f for it will be 1 and its minimum value is 0, when no channel is open. We have,

$$I_{in} = C\frac{dV}{dt} + (V_{Na} - V_m).g_{Na}(V_m, t) + (V_K - V_m).g_K(V_m, t) + (V_L - V_m).g_L \quad -(6)$$

In equation (5), the variable f is also known as the gating variable, which is a key concept in understanding the functionality of a neuron[10]. So, if f determines the portion of open channels, (1-f) will determine the portion of closed channels. These open and closed channels are interlinked by two rate coefficients α and β, which are itself dependent upon V_m.[11]

We have,

$$\frac{df}{dt} = \alpha_f(1-f) - \beta_f(f) \quad -(7)$$

α models the rate of opening of closed channels and β models the rate of closing of open channels.

As the same equation (7) holds true for all channels, from now on in this paper, f will be indicated as m, h and n for Na^+ activation, Na^+ inactivation, and K^+ activation respectively, so they can be distinguished properly[12][13][14].

Potassium Activation

$$G_K = g_{K,max}.n^4(V_m,t)$$

In this equation, n is raised to 4th power, because each potassium channel has 4 activation gates. These 4 gates open and close independently of each other, so to get the probability of each gate to be open, we need to multiply them. Also,

$$\frac{dn}{dt} = \alpha_n(1-n) - \beta_n(n)$$

α_n and β_n, here are chosen so potassium gates begin to activate at or above ~0mV and begin to de-activate at or below ~-20mV[15].

Sodium activation

$$g_{Na} = g_{Na,max}.m^3(V_m,t).h(V_m, t)$$

In this equation, m is raised to 3rd power for similar reasons as given for potassium activation.

$$\frac{dm}{dt} = \alpha_m(1-m) - \beta_m(m)$$

α_m and β_m, here are chosen so sodium gates begin to activate at or above ~-50mV and begin to de-activate at or below ~-30mV.

Sodium inactivation

$$\frac{dh}{dt} = \alpha_h(1-h) - \beta_h(h)$$

α_h and β_h, here are chosen so sodium gates begin to slowly inactivate at ~-20mV and very slowly begin to de-inactivate at or below ~-40mV[15].

3.PULSE TYPE HARDWARE NEURON MODEL

This model have many kinds of variations designed over time for an excitable membrane [16]. With reference to the electrical equivalent circuit model for HH equation, there are mainly four branches in the neuron model. The membrane capacitance, membrane voltage, leakage Nernst equilibrium potential, injected DC current and leakage resistance and are depicted by *C, V, E_L, I_{ext}, R_L,* respectively.[17][18]

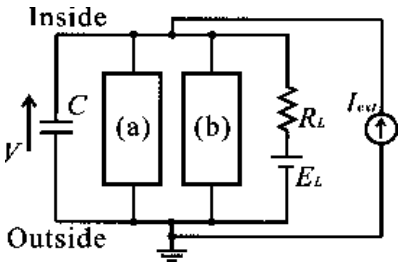

Fig4. Model for Hardware neuron

The branch (a) represent the voltage dependent inward sodium(Na^+) ion channel and the branch (b) represent outward potassium (K^+) ion channel. Replacing the branches (a) and (b) with their transistor model, we get the simulated circuit for hardware neuron.

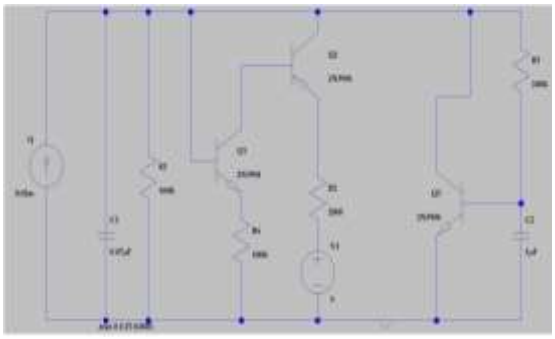

Fig5. Pulse Hardware neuron model

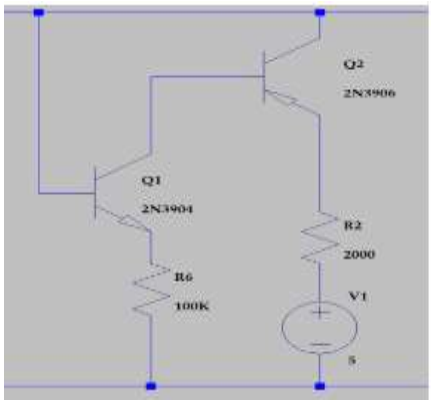

Figure 6(a): Sodium ion channel path

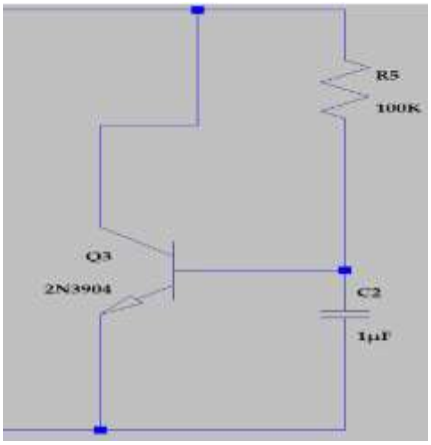

Fig 6(b). Potassium ion channel path

In this neuron model, the cut-off voltage of transistor *Q1* acts as a threshold voltage for the neuron. When the DC input current starts flowing across the circuit, it starts building the voltage across the capacitor *C1* i.e. membrane potential. Once this voltage gets above the cut-off voltage of transistor *Q1*, it gets on and sodium ionic current gets switched on. The potassium (K^+) ionic current gets switched on when the voltage across capacitor *C2* gets above the cut off voltage of transistor *Q3*.

The basic mechanism of how the above circuit is used to generate the action potential is described below:

I1 is used to charge the capacitor *C1* until the voltage across it exceeds the cut-off voltage of transistor *Q1*. As *Q1* gets on, *Q2* also gets on and thus it provides the direct path from voltage *V1* (sodium Nernst equilibrium potential) to charge the membrane capacitor *C1* to more positive voltage. Current via sodium channel path consisting of transistor *Q1* and *Q2* charges *C2* slower in comparison to the *C1*(Membrane capacitor). The voltage across *C2* rises until it exceed the turn-in voltage of transistor *Q3*. As *Q3* gets on, membrane voltage goes down directly by potassium current through transistor *Q3*. Consequently, membrane voltage (V_m) abruptly drops towards potassium Nernst voltage.

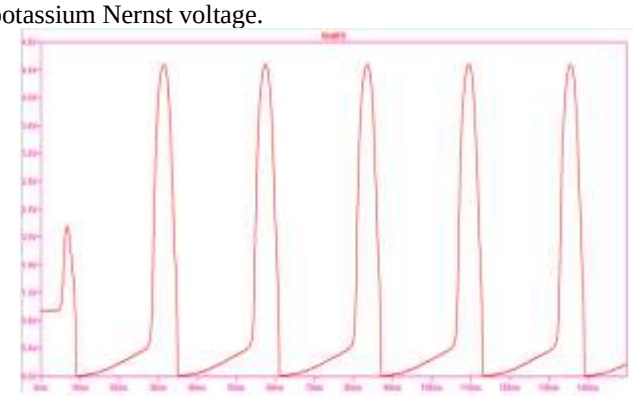

Fig7. Simulated output of hardware neuron

4. NUMERICAL COMPUTATION OF HODGKIN-HUXLEY NEURON MODEL USING MATLAB

We simulated the MATLAB code for a neuron for an injected current of 15mA.

The experimental values used for simulations are given in Table1 below.

Table1: Experimental values used for simulations

Parameters	Values
g_{Na_max}	120 S
g_{K_max}	36 S
g_L	0.3 S

V_{Na}	60 mV
V_K	-77 mV
V_L	-54.4 mV
V_{rest}	-65 mV
α_m	$\frac{2.5 - 0.1(V - V_{rest})}{e^{(2.5-0.1(V-V_{rest})} - 1}$
β_m	$4e^{\frac{-(V-V_{rest})}{18}}$
α_n	$\frac{0.1 - 0.01(V - V_{rest})}{e^{(1-0.1(V-V_{rest})} - 1}$
β_n	$0.125e^{\frac{-(V-V_{rest})}{80}}$
α_h	$0.07e^{\frac{-(V-V_{rest})}{20}}$
β_h	$\frac{1}{e^{(3-0.1(V-V_{rest})} + 1}$

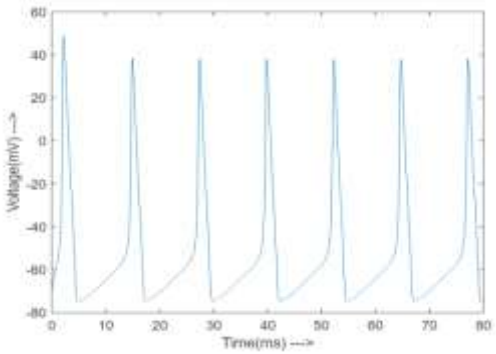

Fig8. Membrane Potential spikes in neuron

We can see in Figure 8, that the action potential start at approximately -70mV and goes upto 45mV. The rapid increase and decrease in voltages in the range of few milliseconds for any particular spike can be clearly observed.

In the Figure 9 below, we closely examined one particular spike and what happens to the gating variables as the membrane potential rise and fall.

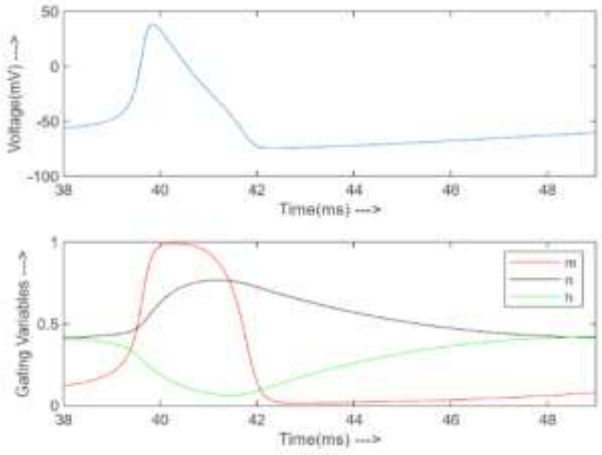

Fig9. Variation of gating variables with membrane potential

We can see, as the V_m rises, the value of *m* also rapidly rises from almost 0.2 to 1. Thus *m* gates are swinging open at a very high rate in this region. As *m* gates account for sodium activation, this causes the sodium ions to rush into the cell, which results in increment of V_m.

After V_m attains peak, *m* value goes down, decreasing V_m. If we observe the *h* gates, its value is decreasing from around 0.4 to more lower value after V_m attains peak. Therefore, it blocks the more sodium ions to enter the cell hence decreasing V_m. For n gates, its value is increasing gradually from 0.4 upto 0.8. As this account for potassium ion activation, this causes potassium ions' movement from inside to the outside of the cell as concentration of potassium ion is more inside the cell as compared to outside. This movement of positive ions, causes V_m to decrease. After a few milliseconds, the gating variables return to their steady state variable.

The conductances of potassium(g_K) and sodium ions (g_{Na}) has been plotted along with leak conductance (g_L) in Figure 10 to observe the effect of gating variables on them.

The conductances of potassium and sodium ions fluctuate in relation to their gating variables which is shown in Figure 9. As stated earlier, leak conductance does not depend upon membrane potential, thus it remains constant.

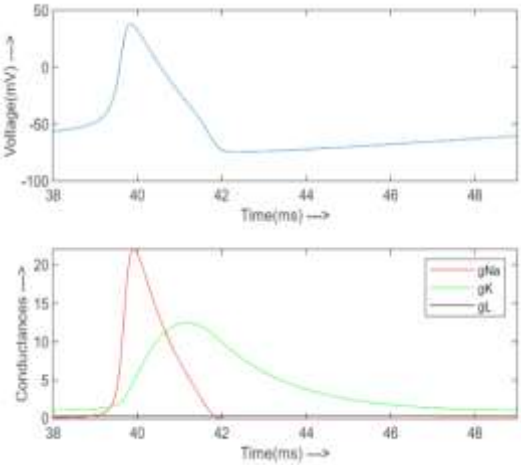

Fig10. Variation of conductance with V_m

5. CONCLUSION

The neuron model proposed by Hodgkin Huxley is one of the most used model in various fields of research in spiking neural networks. In this paper, we conducted a detailed analytical study of this model, exploring its different characteristics and aspects. Apart from studying it through the circuit parameters in Ltspice, we also plotted its characteristics numerically in MATLAB using the mathematical equations given by Hodgkin and Huxley. The variations in gating variable and ionic conductances were aligned with the spiking characteristics of neuron.

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