Chapter 3

General Overview of Neuron Modeling
3.1 Introduction:

Neuron models are used in neuromorphic engineering aiming at understanding of real neuronal systems and are gaining better, possibly brain like performance for systems being built. In recent times the term neuromorphic has been used to describe analog, digital or mixed-mode analog/digital VLSI systems that implement models of neuronal systems (for perception, motor control, or sensory processing) as well as software algorithms. A key aspect of neuromorphic design is to understanding how the morphology of individual neurons, circuits, and overall architectures create desirable computations, affect how information is represented, influences robustness to damage, incorporates learning and development, and facilitates evolutionary change. Neuromorphic engineering takes inspiration from biology, physics, mathematics, computer science and engineering to design artificial neural systems, such as vision systems, head-eye systems, auditory processors, and autonomous robots, whose physical architecture and design principles are based on those of biological nervous systems. Neuromorphic systems are basically artificial systems and can be realized in analog VLSI, and they range from vision chips to synaptic conductances. By building and operating neuromorphic systems, we can have depth knowledge of computational principles of neural networks and systems. Here we reviewed of some publications concerning the development of model of neuron based on Hodgkin – Huxley equations and subsequent electronic models based on the similarities between biological and semiconductor physics.
Neuroengineering, or more precisely Bio-neuroengineering which is inseparable part of Bioelectronics, is an interdisciplinary area, with the common goal of analyzing the function of the nervous system, developing methods to restore damaged neurological function & creating artificial neuronal systems by integrating physical, chemical, mathematical & engineering tools.

The development of artificial circuit models that simulate the behavior of biological neuron is one of today's most promising directions of investigation in the field of neurobio and neuromorphic engineering. Two main classes of models of spiking neurons have been implemented in silicon, namely conductance based model, such as Hodgkin and Huxley (H-H) model and Integrate and fire (I-F) model such as McGregor model. I-F model is employed to reproduce some phenomenological properties of biological neurons that are specially focused on the designers. The latter is employed to design the electrophysiological properties of biological neuron accurately. Conductance based model details allow for a dynamics of a neuron membrane potential and spike generation.

3.2 Conductance based model:

These types of silicon neurons contain analog circuits that emulate the physics of a real ionic conductance and are typically based on prototypical ion conductance models that obey Hodgkin-Huxley principles. The Conceptual basis of the H-H model equation is given by the stochastic motion of ions through channel proteins with the cell membrane, driven by potential difference across the cell membrane and regulated by potential dependent conductances. In 1952 H-H explained how action potentials
are generated through the electrical excitability of neuronal membrane. Action potential are arises from the synergistic action of sodium and potassium channels, each of which open and close in a voltage dependent fashion.

Conductance based models allow for detailed dynamics of a neuron's membrane potential and spike generation. They imitate the biological neurons' response by modeling several ion channels, like sodium, potassium, calcium, etc. Advantages of conductance models are the possibility of modeling post-inhibitory rebounds, bursting, and the multiple-compartmental structure of a single neuron. Thus Hodgkin-Huxley conductance based models provides an excellent description of the electrical behavior of the neuron. The Hodgkin and Huxley (H-H) equation is generally used by biomedical engineers and neuroscientist to model analog circuit for axonal membrane. If a neuron is considered as a signal processing unit, the synapses and dozens of different types of neurotransmitters discovered thus far that can act on transmitter-gated ion channels in synapses must be examined. Thus far, many neuron models were proposed for explaining the active principle of neuron such as the famous Hodgkin-Huxley (H-H) model.

3.2.1 **Hodgkin-Huxley Membrane Model:**

Hodgkin- and Huxley showed that the current can be carried through the membrane either by charging the membrane capacity or by movement of ions through the resistances in parallel with the capacity. Hodgkin- and Huxley described that the electrical behaviour of membrane may be represented by the equivalent circuit shown in Fig 3.1.
As described by Hodgkin and Huxley, the current passing through the biological membrane can either be the capacitive current, which is by charging the membrane capacitance or ionic current which is due to movement of ions through the resistances in parallel with the capacitance. So, for analysis of Hodgkin and Huxley model, the total current is divided into capacitive current
and ionic current. The expression of capacitive current described by Hodgkin and Huxley is given by-

\[ I_C = C_m \left( \frac{dV}{dt} \right) \]  \hspace{1cm} (3.1)

And the ionic part of the total current is composed of sodium ions \((I_{Na})\) current, potassium ions \((I_K)\) current and other ions \((I_l)\) current and given by-

\[ I_i = I_{Na} + I_K + I_l \] \hspace{1cm} (3.2)

Thus the total membrane current is

\[ I = I_C + I_i \] \hspace{1cm} (3.3)

In the capacitive current, \(V\) is the displacement of the membrane potential from its resting value, \(C_M\) is the membrane capacitance per unit area which is assumed to be constant, \(t\) is time. Hodgkin and Huxley in their series of papers showed that the ionic permeability of the membrane can be expressed in terms of ionic conductances \((g_{Na}, g_K\) and \(g_l)\). The respective ionic currents given by Hodgkin and Huxley are –

\[ I_{Na} = g_{Na} \left( E - E_{Na} \right) \] \hspace{1cm} (3.4)

\[ I_K = g_K \left( E - E_K \right) \] \hspace{1cm} (3.5)

\[ I_l = g_l \left( E - E_l \right) \] \hspace{1cm} (3.6)

Where \(E_{Na}\) and \(E_K\) are the equilibrium potentials for the sodium and potassium ions. \(E_l\) is the potential at which the leakage current due to chloride and other ions is zero. For practical application the above three equations can be written in the form –

\[ I_{Na} = g_{Na} \left( V - V_{Na} \right) \] \hspace{1cm} (3.7)
\[ I_K = g_K (V - V_K) \]  
(3.8)

\[ I_I = \bar{g}_I (V - V_I) \]  
(3.9)

Where,

\[ V = E - E_r \]  
(3.10)

\[ V_{Na} = E_{Na} - E_r \]  
(3.11)

\[ V_K = E_K - E_r \]  
(3.12)

\[ V_I = E_I - E_r \]  
(3.13)

And \( E_r \) is the absolute value of the resting membrane potential. \( V, V_{Na}, V_K \) and \( V_I \) can be measured as displacement from the resting membrane potential.

At steady state condition the capacitive component of total current is zero since at steady state \( (dV/dt) = 0 \). The individual ionic currents are related with their respective ionic conductances \( (g_{Na}, g_K \text{ etc}) \), equilibrium potential of respective ions \( (E_{Na}, E_K \text{ and } E_I) \) and membrane potential \( (E) \) [1]. The total membrane current is a function of time and voltage and is given by –

\[ I = C_m (dV/dt) + g_K m^4 n^4 (V - V_K) + g_{Na} h^3 n (V - V_{Na}) + \bar{g}_I (V - V_I) \]  
(3.14)

In order to introduce the concept of resting and action potential related to biological neurons, the general equation for the total current passing through the patch of membrane given by Hodgkin and Huxley can be expressed as -

\[ I_{in} = C_m (dV/dt) + g_K (V - E_K) + g_{Na} (V - E_{Na}) + I_p \]  
(3.15)
Where, $I_p$ is current due to Na–K pump, which can be split into two separate currents, Sodium pump current $I_p(Na)$, and Potassium pump current $I_p(K)$. Because of its small contribution, Na-K pump current can be neglected.

From the current expression, the resting potential expression can be written as-

$$V_m = \frac{\left( E_K + NK \cdot E_{Na} \right)}{\left( 1 + NK \right)}$$ (3.16)

Where, $NK = \frac{(g_{Na})}{(g_{K})}$ is the sodium/potassium conductance ratio.

Now, if an ionic current is injected-

$$I_{ext} = C_m \left( \frac{dv}{dt} \right) + g_K \left( V - E_K \right) + g_{Na} \left( V - E_{Na} \right)$$ (3.17)

inside the cell through the membrane neglecting the Na-K active pump current $I_p$, it will shift the resting potential of the membrane to a new steady value. Therefore, constant current $I_{ext}$ applied for a short time $\Delta t$ will shift the membrane potential to a new steady value given by-

$$V_{m}^* = \left[ \frac{\left( E_K + NK \cdot E_{Na} \right)}{\left( 1 + NK \right)} \right] + \frac{I_{ext}}{(g_K + g_{Na})}$$ (3.18)

Now if the $I_{ext}$ current is turn off, the membrane current will decay back to its original resting value $V_m$. The simulated output profile of H-H model is shown in Fig 3.2.
After these historical developments, many researchers in the field of bio-
neuroengineering proposed different models of biological neurons. The
dynamics of those models is quantitatively and qualitatively similar to
Hodgkin-Huxley mechanisms which implement their specific equations.

3.2.2 Lewis Membrane Model:

Edwin R. Lewis published several electronic membrane models that are based
on the Hodgkin-Huxley equations. Lewis membrane model proposed by
Edwin R. Lewis, uses discrete transistors and associated components for the
sodium and potassium conductances, synaptic connections, and other
functions of electronic membrane model [2]. All these are parallel circuits
connected between nodes representing the inside and outside of the
membrane. Lewis implemented the electronic membrane model as shown in

Fig. 3.2: The simulated output of H-H model
Fig. 3.3, with active filters for sodium and potassium conductances and multipliers.

Since the output of the model is the transmembrane voltage $V_m$, the potassium current can be evaluated by multiplying the voltage corresponding to $G_K$ by $(V_m - V_K)$. The Lewis model is an accurate physical analogy to the Hodgkin-Huxley expressions and the behavior of the output voltage $V_m$ corresponds to that predicted by the Hodgkin-Huxley equations. The electronic circuits in the Lewis neuron model have provision for inserting (and varying) not only such constants as $G_{K_{\text{max}}}$, $G_{Na_{\text{max}}}$, $V_K$, $V_{Na}$, $V_{Cl}$, which enter the Hodgkin-Huxley formulation, but also $\tau_{ls}$, $\tau_m$, $\tau_v$, which allow modifications from the Hodgkin-Huxley equations. In the following, the components of the model are discussed separately.
3.2.2.1 Potassium Conductance of Lewis Membrane model:

The circuit simulating the potassium conductance of Lewis membrane model is shown in Fig 3.4. The potassium conductance function $G_K(V_m, t)$ is generated from the simulated membrane voltage through a nonlinear active filter according to the Hodgkin-Huxley model (in the figure separated with a dashed line). The three variable resistors in the filter provide a control over the delay time, rise time, and fall time. The value of the potassium conductance is adjusted with a potentiometer, which is the amplitude regulator of a multiplier. The multiplier circuit generates the function $G_K(V_m, t)\cdot v_K$, where $v_K$ is the difference between the potassium potential ($V_K$) and membrane potential ($V_m$). The multiplier is based on the quadratic function of two diodes.

![Fig. 3.4: The circuit simulating the potassium conductance of the Lewis membrane model.](image)
3.2.2.2 Sodium Conductance of Lewis Membrane model:

In the Lewis circuit simulating the sodium conductance, Lewis omitted the multiplier on the basis that the equilibrium voltage of sodium ions is about 120 mV more positive than the resting voltage. Because of the interest in small membrane voltage changes, the gradient of sodium ions may be considered constant. The circuit simulating the sodium conductance is shown in Fig 3.5. The time constant of the inactivation is defined according to a varistor. The inactivation decreases monotonically with the depolarization, approximately following the Hodgkin-Huxley model.

![Circuit Diagram](image)

Fig. 3.5: The circuit simulating the sodium conductance of the Lewis membrane model.
3.2.2.3 Simulated Action Pulse of Lewis Membrane model:

By connecting the components of the membrane model as in Fig 3.6 and stimulating the model analogously to the real axon, the model generates a membrane action pulse. This simulated action pulse follows the natural action pulse very accurately. Fig 3.7 illustrates a single action pulse generated by the Lewis membrane model. The goal of Lewis model is to simulate the behavior of a neuronal network, including coupled neuron.

Fig. 3.6: The complete Lewis membrane model
3.2.3 Roy Membrane Model:

Guy Roy published an electronic membrane model in 1972 and gave it the name "Neurofet". His model, analogous to Lewis's, is also based on the Hodgkin-Huxley model. Guy Roy used Field Effect Transistors (FETs) to simulate the sodium and potassium conductances. FETs are well known as adjustable conductors. So the multiplying circuit of Lewis may be incorporated into a single FET component (Fig 3.8). In the Roy model the conductance is controlled by a circuit including an operational amplifier, capacitors, and resistors. This circuit is designed to make the conductance
behave according to the Hodgkin-Huxley model. Roy's main goal was to achieve a very simple model rather than to simulate accurately the Hodgkin-Huxley model. Nevertheless, the measurements resulting from his model, shown in Fig 3.9 and 3.10, are reasonably close to the results obtained by Hodgkin and Huxley. Fig 3.9 illustrates the steady-state values for the potassium and sodium conductances as a function of applied voltage. Note that for potassium conductance the value given is the steady-state value, which it reaches in steady state. For sodium the illustrated value is $G_{Na} = G_{Na max} m^3 h_0$; it is the value that the sodium conductance would attain if $h$ remained at its resting level ($h_0$). The full membrane model was obtained by connecting the potassium and sodium conductances in series with their respective batteries and simulating the membrane capacitance with a capacitor of 4.7 nF and simulating the leakage conductance with a resistance of 200 kΩ. The results from the simulation of the action pulse are illustrated in Fig 3.10 [3]

Fig. 3.8: The circuits simulating (A) sodium and (B) potassium conductances in the Roy Membrane Model
Fig. 3.9: Steady-state values of the (A) $G_K$ and (B) $G_{Na}$ as a function of membrane voltage clamp in the Roy model (solid lines), compared to the measurements of Hodgkin and Huxley (dots). $V_{m}$, the transmembrane voltage, is related to the resting value of the applied voltage clamp.

Fig. 3.10: Voltage-clamp measurements made for (A) potassium and (B) sodium conductances in the Roy model. The voltage steps are 20, 40, 60, 80, and 100 mV. (C) The action pulse simulated with the Roy model.
3.2.4 Farquhar and Hasler Membrane Model:

There is clear analogy between biological neuron and semiconductor physics. Hodgkin-Huxley realized non-linear conductance using resistors. But biological channels have exponential current relationship to the voltage on the membrane which can not be simply realized using resistor. E. Farquhar and P. Hasler [4] proposed a model of sodium and potassium channel type neuron circuit which can generate action potential. The proposed model of E. Farquhar and P. Hasler is shown below in Fig. 3.11.

![Diagram of Farquhar and Hasler Membrane Model](image)

*Fig. 3.11 The figure shows the model proposed by E. Farquhar and P. Hasler*
The Fig. 3.11 shows the transition from bio-physics to semiconductor physics. The proposed model of E. Farquhar and P. Hasler can be realized for linear conductances using Bipolar Junction Transistor (BJT) or a sub-threshold mode Metal Oxide Semiconductor Field Effect Transistor (MOSFET). MOSFET has higher preference over BJT because of its extremely low power dissipation in sub-threshold mode. As mentioned earlier, the proposed model of neuron circuit of E. Farquhar and P. Hasler consists of sodium and potassium channel circuits. The sodium channel circuit of E. Farquhar and P. Hasler is shown in Fig. 3.12 below, and it contains a band pass filter and a band pass control circuit which controls the sodium channel transistor and it is observed that the response of band pass filter is that of sodium channel response. Similarly, the potassium channel circuit of E. Farquhar and P. Hasler is shown below in Fig. 3.13. By combining the two channel circuits of E. Farquhar and P. Hasler a complete neuron circuit model is proposed, which is shown in Fig. 3.14. The results obtained by E. Farquhar and P. Hasler closely matches with the biological data.
Fig. 3.12: The sodium channel circuit model of E. Farquhar and P. Hasler

Fig. 3.13: The potassium channel circuit model of E. Farquhar and P. Hasler
The experimentally measured action potential generated by the circuit model developed by E. Farquhar and P. Hasler is shown in Fig. 3.15. The action potential generated is quite similar to the biological one.
The physical principles governing ion flow in biological neurons share some interesting similarities to electron flow through channels of a MOS transistor. A simple electrical circuit can be designed which seeks to exploit these similarities to produce an artificial silicon neuron circuit which behaves in a manner similar to a biological neuron. Artificial neurons are designed to process information in a way that directly mimics the processing that is found in biological neurons.

### 3.3 Integrate and Fire (I&F) model:

Integrate and fire (I&F) models are less realistic than conductance-based ones, but require fewer transistors and less silicon real-estate. They allow for the implementation of large, massively parallel networks of neurons in a single VLSI device. I&F neurons integrate pre-synaptic input currents and generate a
voltage pulse analogous to an action potential when the integrated voltage
reaches a spiking threshold. Networks of I&F neurons have been shown to
exhibit a wide range of useful computational properties, including feature
binding, segmentation, pattern recognition, onset detection, and input
prediction. Many variants of these circuits were built during the 1950s and
1960s using discrete electronic components.

3.3.1 I & F model of Mead:

The first simple VLSI version was probably the Axon-hillock circuit,
proposed by Carver Mead and colleagues in the late 1980s (Mead, 1989) [5].
It reduces the complexity of a single component with a single conductance.
The Integrate & Fire model of Carver Mead is shown in Fig 3.16.

![Fig. 3.16: The Axon Hillock Integrate and Fire circuit of C. Mead.](image-url)
In this model, a capacitor that represents the neuron's membrane capacitance integrates current input to the neuron. When the capacitor potential crosses the spiking threshold a pulse $V_{out}$ is generated and the membrane potential $V_{mem}$ is reset. This circuit captures the basic principle of operation of biological neurons, but cannot faithfully reproduce all of the dynamic behaviors observed in real neurons. In addition it has the drawback of dissipating non-negligible amounts of power while the membrane potential $V_{mem}$ crosses the amplifier's switching threshold [6].

The basic circuit of this model consists of a capacitor $C$, parallel with a resistor $R$, driven by current $I$. The basic Integrate and Fire model of neuron is shown in Fig 3.17.

Fig 3.17 Basic Integrate and Fire circuit
The driving current is split into two components:

\[ I = I_R + I_C \]  

(3.19)

Where \( I_R \) is the resistive current passing through the \( R \), and \( I_C \) is the capacitive component.

Thus, \( I = V/R + C \left( \frac{dv}{dt} \right) \)  

(3.20)

Where \( V \) is the membrane potential. A spike will occur when \( V \) reaches a threshold \( V_{TH} \). After the occurrence of spike, next spike cannot occur due to refractory period [7].

### 3.3.2 Leaky integrate and fire model:

The family of leaky integrate-and-fire (LIF) neuron models has a simplified spike generation mechanism while providing an accurate approximation of the membrane potential and other neuron properties like refractory properties and adaptation to stimuli. Simplification of spike generation allows for improved computation speed as compared with conductance based models. This model is a very simple mechanism of spike generation and dendritic integration. Abbott & Kepler (1990) have shown that the more complex H-H model can be reduced to an LIF model in the limit that the membrane loading time is the dominant time scale of the neuronal dynamics [8]-[9]. The model provides a good description of subthreshold integration of synaptic input and injection current that the neuron received. The Leaky Integrate and Fire model of neuron is shown in Fig 3.18.
The resistor $R$ models the overall leakage of the membrane, and the $C$ models the overall capacitance of the membrane. The time constant $\tau = RC$. When a spike occurs the capacitor will be discharged. In this neuron model, the dendrites are modeled as a single point at which the synaptic inputs are summed, while current leaks away linearly. The voltage like state variable at point $A$ is described by:

$$\frac{dA}{dt} = -\frac{A}{\tau} + I(t)$$  

(3.21)

Where $\tau$ is the time constant of the point neuron (i.e. a reciprocal) measure of its leakiness), and $I(t)$ is the total external input to the neuron. In the presence of positive input, the activity $A$ can rise to the threshold $\theta$. When this is crossed from below, the neuron emits a spike, and $A$ is reset to some initial value. The mechanism of spike generation is generally ignored in the model, and the output is characterized entirely by the sequence of spike times. This type of neuron is sometimes known as a point neuron, because all the...
geometry of the dendrite has been shrunk to a single point. If $R$ is infinite, then
the neuron is not leaky, and it simply integrates its input until this reaches
threshold. If $\tau$ is small, then more recent inputs have a larger effect on $A$. If $I(t)$
is made up of a number of excitatory synaptic inputs, each of which is not
large enough to cause $A$ to exceed $\theta$, then the neuron will act as a coincidence
detector, firing when a number of its excitatory inputs occur at about the same
time, allowing $A$ to reach $\theta$ in spite of the leakage.

As integrate and fire model is described by the dynamics of membrane
potential $V(t)$.

$$C_m \frac{dv}{dt} = - \frac{C_m}{\tau_m} \left[ v(t) - V_0 \right] + I_S(t) + I_{inj}(t) \quad (3.22)$$

Where $C_m$ is the membrane potential, $V_0$ is the resting potential, $\tau_m$ is the
passive membrane time constant, $I_S(t)$ a current describing the effect of
synaptic input. $I_{inj}(t)$ is the current injected in to the neuron. The first term in
the right is the current due to the passive leak of the membrane, and the
passive membrane time constant is related to the neuron’s capacitance and
leak resistance $R_m$ of the membrane potential by $\tau_m = R_m C_m$. For
subthreshold potentials the respond of the model to periodic deterministic
input is

$$v(t) = V_0 + e^{-\frac{t}{\tau_m}} \int_0^t \frac{I_{inj}(t')}{C_m} e^{\frac{t'}{\tau_m}} \, dt' \quad (3.23)$$

Where it is assumed that the membrane potential at the initial time $t_0$ is at the
resting potential $v(t_0) = V_0$. When the membrane potential reaches the
threshold $V_{th}$ a spike is generated and the membrane potential is reset to its
initial value $V_{reset}$. The main interest in the integrate-and fire neuron model is
stochastic synaptic input. In the case of current synapses, the synaptic current
is described by

\[ I_S(t) = C_m \sum_{k=1}^{N_x} a_{E,k} S_{E,k}(t) + C_m \sum_{k=1}^{N_x} a_{I,k} S_{I,k}(t) \] (3.24)

As in the case of homogeneous synaptic input, the excitatory and inhibitory
synaptic inputs, \( S_{E,k}(t) \) and \( S_{I,k}(t) \), are described as a series of \( \delta \)-function inputs
to each synapse.

\[
\begin{align*}
S_{E,k}(t) &= \sum_{t_{E,k}} \delta(t-t_{E,k}) \text{ and} \\
S_{I,k}(t) &= \sum_{t_{I,k}} \delta(t-t_{I,k})
\end{align*}
\] (3.25)

where \( t_{E,k} \) and \( t_{I,k} \) are the times of the synaptic input spikes for the excitatory
and inhibitory synapses, respectively. Thus leaky integrate fire model gives
the membrane potential for injected current and details of the synaptic input
for current synapses and conductance synapses [10].

Nonlinear integrate-and-fire models have been considered in different
regimes. These can roughly be categorized by three criteria; whether they are
deterministic or stochastic, whether the applied signal is static or time
dependent, and whether this is a sub- or suprathreshold signal, i.e. whether the
signal may drive the membrane voltage above the threshold in absence of
random noise.

**3.4 References:**

Membrane Current And Its Application To Conduction And


