Chapter 13

Pulsed Neuron Models: The New Generation

Neural Networks: A Classroom Approach
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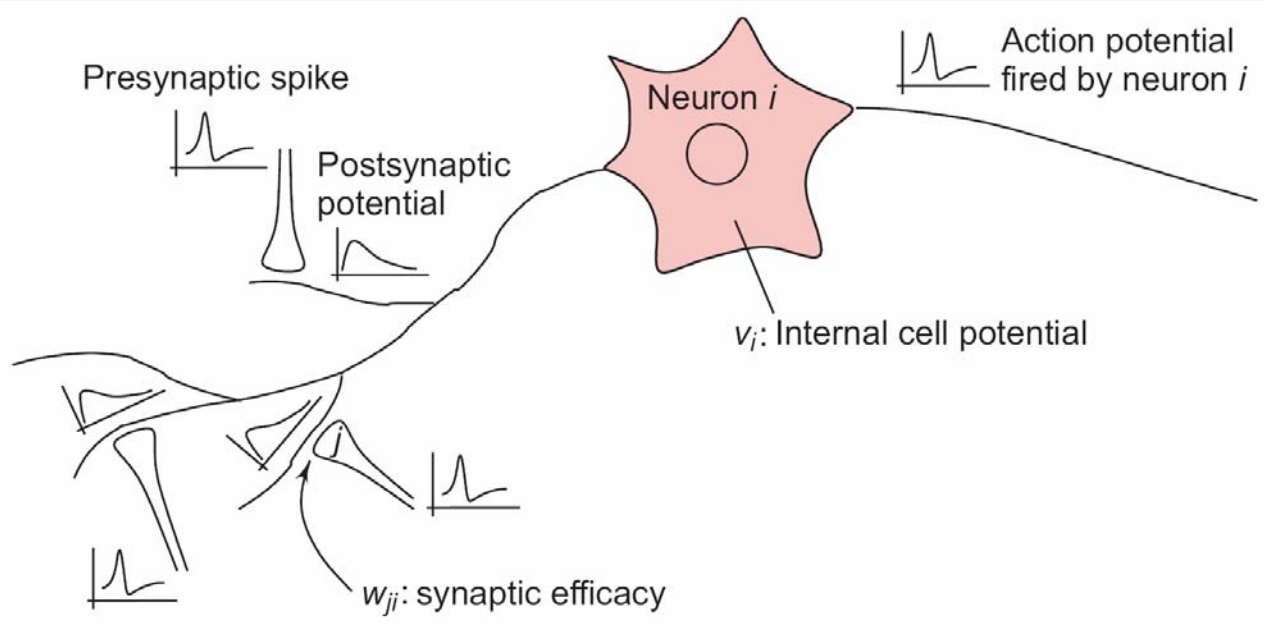
Biological Plausibility

- Artificial neuron models are simplified versions of their biological counterpart
- However, to exploit the real power of the brain metaphor in intelligent systems, biological accuracy must be improved in the basic model of the neuron
- Neuron models which incorporate biologically realistic properties of neuron behavior include
  - Spiking neuron model
  - Conductance based models (Hodgkin-Huxley model)

Biological Neuron



- Presynaptic action potentials fired along axon of neuron j evoke postsynaptic potentials (PSP) in the dendrite of neuron i
- Cell soma integrates spatio-temporal PSPs and if the cell potential exceeds a threshold V_θ, neuron i fires an action potential

Spiking Neuron Model

- Basic idea: represent each PSP by a kernel function and superpose various such functions appropriately depending upon the firing times and physical locations of presynaptic neurons
- The set of firing times of a presynaptic neuron j is

$$\mathcal{T}_j = \{t_j^k, 1 \le k \le n\} = \{t | v_j(t) = V_\theta\}$$

firing times where k indexes the time

- With t_j^n as the most recent firing time of the neuron. When the neuron's cell potential $v_j(t)$ equals threshold V_θ, it fires.

PSP Kernel Functions

- Assumption: time course of the PSP is described by a kernel function κ_{ji} which must vanish before the firing time t_j^k of the presynaptic neuron j

$$\kappa_{ji}(t) = \left[\exp\left(-\frac{t}{\tau_m}\right) - \exp\left(-\frac{t}{\tau_s}\right)\right] u(t)$$

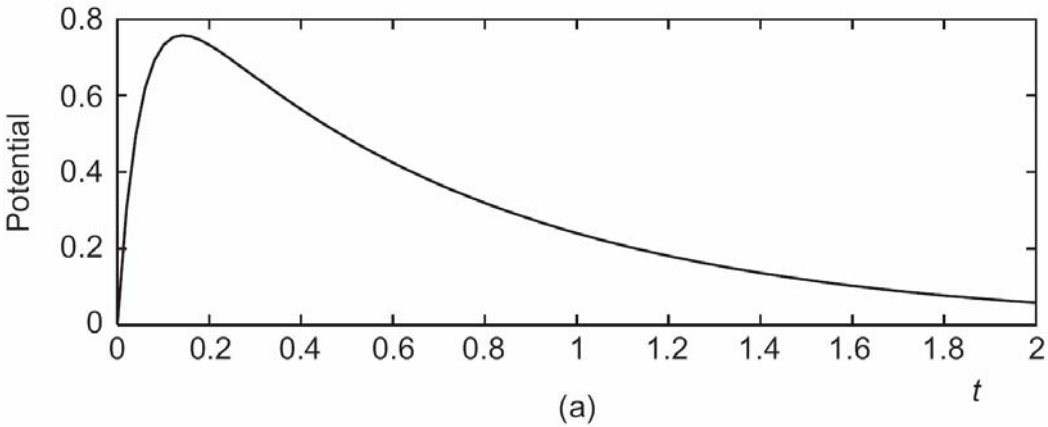
This equation assumes that presynaptic neuron fires at t = 0

Time constants that control the rise and fall of the function

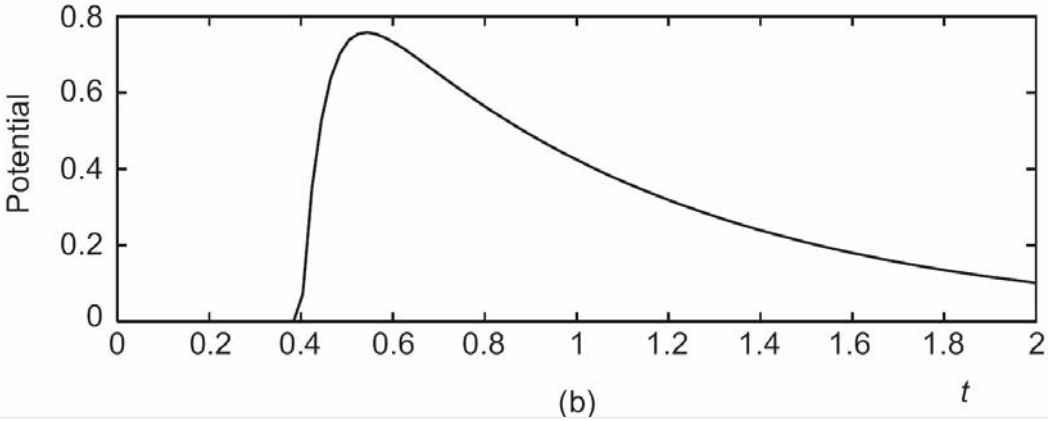
- Axonal and synaptic transmission delays can be designed by introducing an axonal time delay Δ

$$\kappa_{ji}(t-\Delta) = \left[\exp\left(-\frac{t-\Delta}{\tau_m}\right) - \exp\left(-\frac{t-\Delta}{\tau_s}\right)\right] u(t-\Delta)$$

Examples of PSP Kernel Function



(a) Excitatory postsynaptic potential (EPSP) kernel function



(b) Kernel function with axonal time delay

Inhibitory postsynaptic potential (IPSP) can be generated by reversing the sign of the kernel function

Introducing Weight

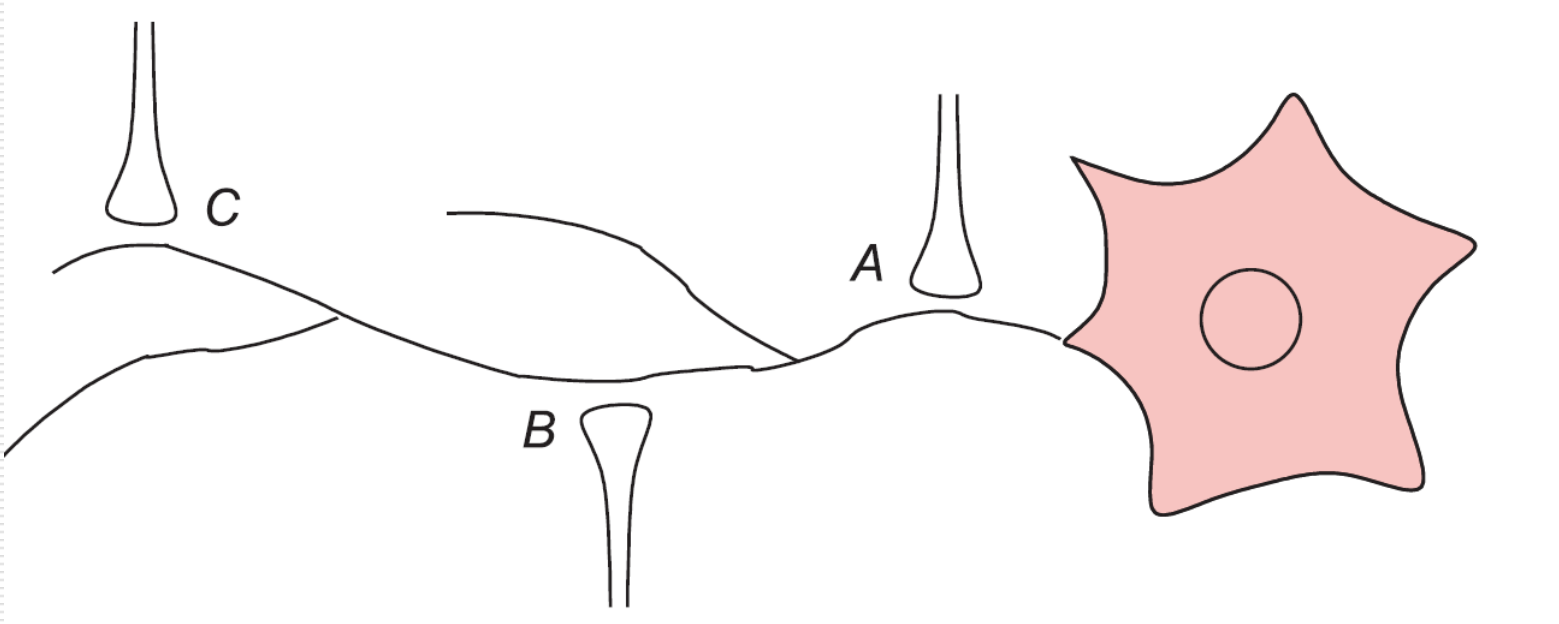
- Appropriately scales the PSP kernel to account for any attenuation effects occurring due to the variable synaptic efficacy and the distance of synapse from the cell soma
- Negative weight will generate an IPSP and a positive weight an EPSP
- EPSP or IPSP will either increase or decrease the postsynaptic soma potential by an amount that depends on the magnitude and sign of the synaptic efficacy
- Various PSPs superposed in a weighted summation decide the actual time course for the cell potential

$$v_i(t) = \sum_j \sum_{t_j^k \in \mathcal{T}_j} w_{ji} \kappa_{ji}(t - t_j^k)$$

Relative Locations of Synapses and Firing Sequence

- The time course of the cell potential depends on two factors
  - the relative location and efficacy of the synapses of firing presynaptic neurons
  - the sequence in which the presynaptic neurons fire

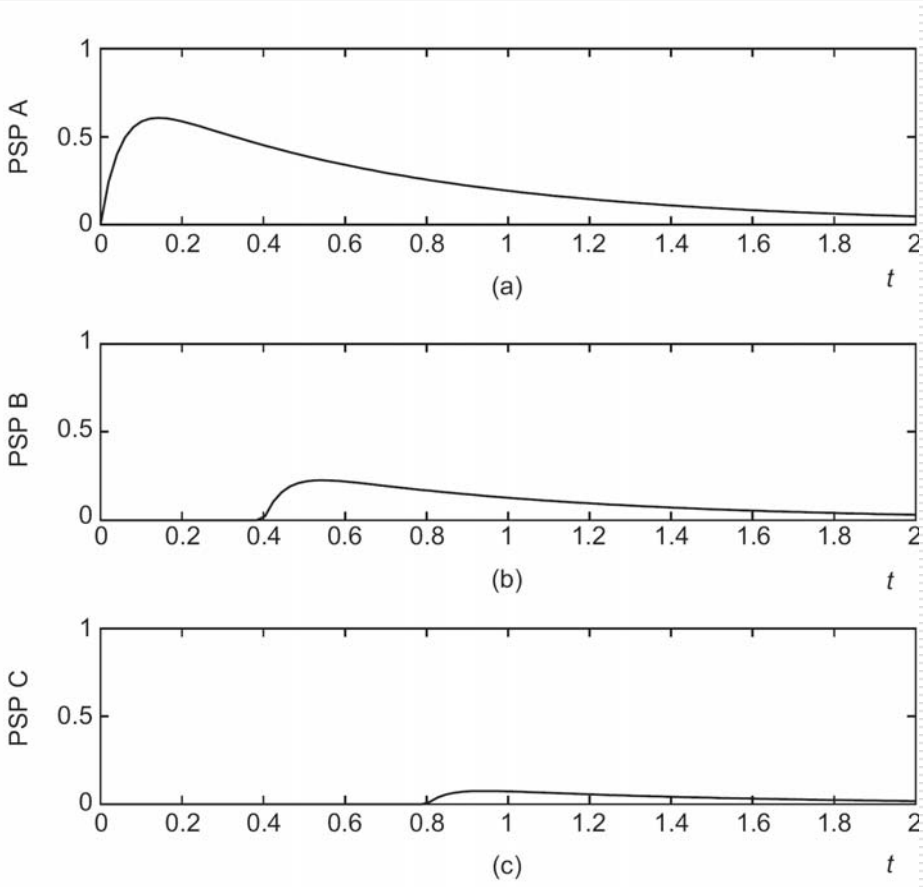
Relative Locations of Synapses and Firing Sequence



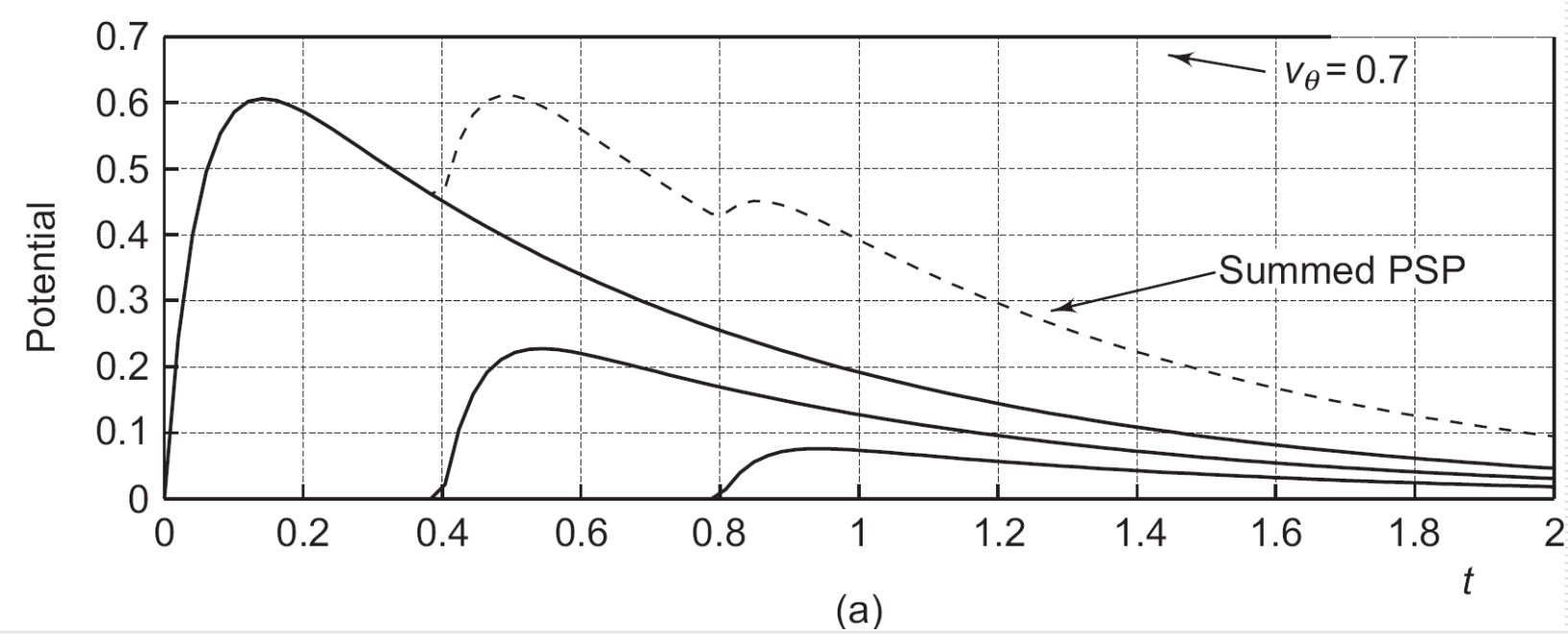
- Assumptions
  - synapses A, B, and C are all excitatory
  - they all have the same kernel function

Time Course of Synapses A, B and C

- Synapse A receives a spike at t = 0; synapse B at t = 0.4; synapse C at t = 0.8
- The selection of firing times imply a firing sequence $A \rightarrow B \rightarrow C$

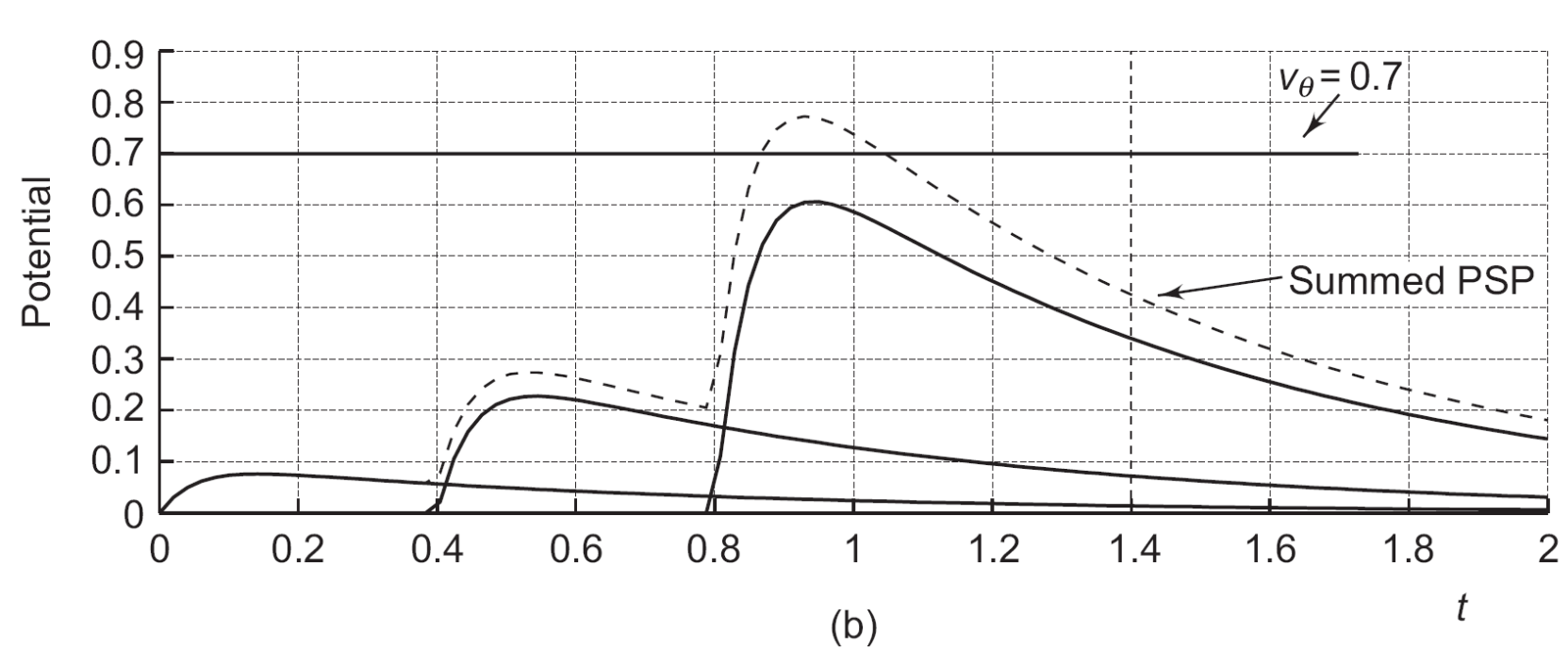


Firing Sequence A → B → C



- The neuron does not fire an action potential for the cell's threshold of 0.7

Firing Sequence C → B → A



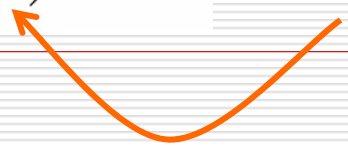
- In this case the neuron fires!

Kernel Reset

- Once the cell potential reaches the threshold, the cell potential undergoes a reset to model the refractory nature of the firing of an action potential
- Introducing another kernel function $\gamma_i(t - t_i^k)$ adds a negative contribution to the cell potential at time t_i^k and resets it to the resting value
- An example refractory function

$$\gamma_i(t) = -V_\theta \exp\left(-\frac{t}{\tau}\right) u(t)$$

time constant controls the rate of fall of the exponential



Spike Response Model of neuron

- The introduction of the refractory function modifies the cell potential to

$$v_i(t) = \sum_{t_i^k \in \mathcal{T}_i} \gamma_i(t - t_i^k) + \sum_j \sum_{t_j^k \in \mathcal{T}_j} w_{ji} \kappa(t - t_j^k)$$

- Assuming that only the most recent spike fired by neuron i contributes a refractory function

$$v_i(t) = \gamma_i(t - t_i^n) + \sum_j \sum_{t_j^k \in \mathcal{T}_j} w_{ji} \kappa(t - t_j^k)$$

reset kernel

weighted PSP kernels

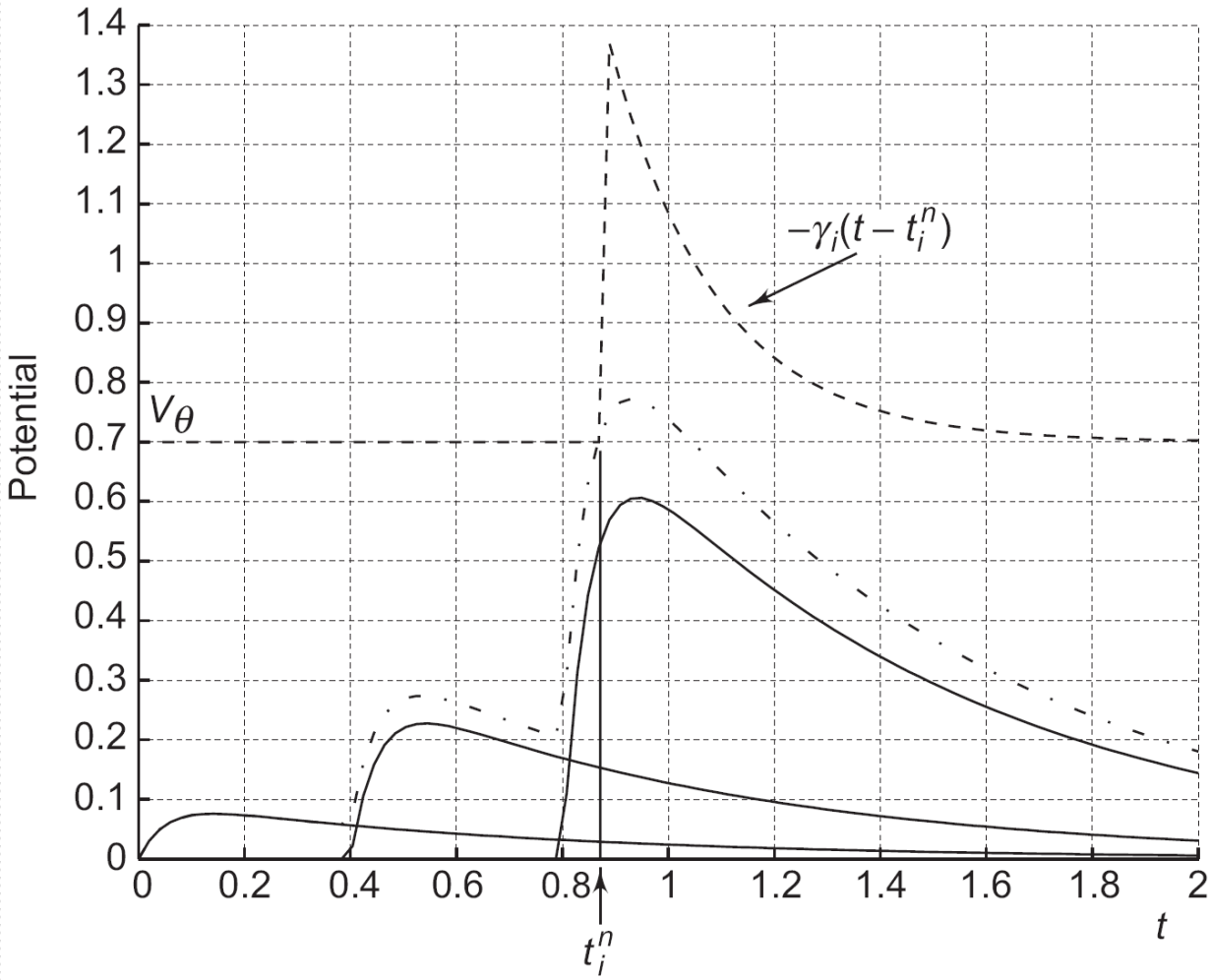
Kernel Function as a Dynamic Threshold

- The next spike will occur when the given cell potential equals the threshold

$$\sum_j \sum_{t_j^k \in \mathcal{T}_j} w_{ji}\kappa(t - t_j^k) = V_\theta - \gamma_i(t - t_i^n) \quad \leftarrow \text{dynamic threshold}$$

$$= V_\theta + V_\theta \exp\left(-\frac{t - t_i^n}{\tau}\right) u(t - t_i^n)$$

Kernel Function as a Dynamic Threshold



After the most recent firing time, threshold suddenly increases from V_θ to $2V_\theta$ making it more difficult for neuron to fire

As threshold gradually decays back to its resting value V_θ , the possibility of neuron firing increases thus modeling the refractory nature of neuron firing

Spike Response Model: General Version

- In reality:
  - A neuron fires a spike whenever its cell potential crosses the threshold value
  - The potential of the cell continues to rise to a peak and then falls suddenly to undershoot its resting value.
  - It then gradually decays towards its resting value
- Spike after-potential crosses threshold twice
  - once on the way up to the peak
  - once on the way down to the undershoot

Spike Response Model: General Version

- The entire time course of the spike after-potential can be described by the kernel function

$$\mathcal{T}_i = \{t | (v_i(t) = V_\theta) \wedge (v_i'(t) \geq 0)\}$$

This ensures that a firing time is recorded only when the spike crosses the threshold on the way up to the peak

- The shape of the after-potential in the cell and the action potential generated is almost the same

Refractoriness

- *Absolute refractory period* – no other spike can be fired while the action potential rises or falls
- *Relative refractory period* – difficulty in firing for the neuron during the undershoot
- Refractoriness decides the maximum frequency at which a neuron can fire spikes

Accounting for Refractoriness

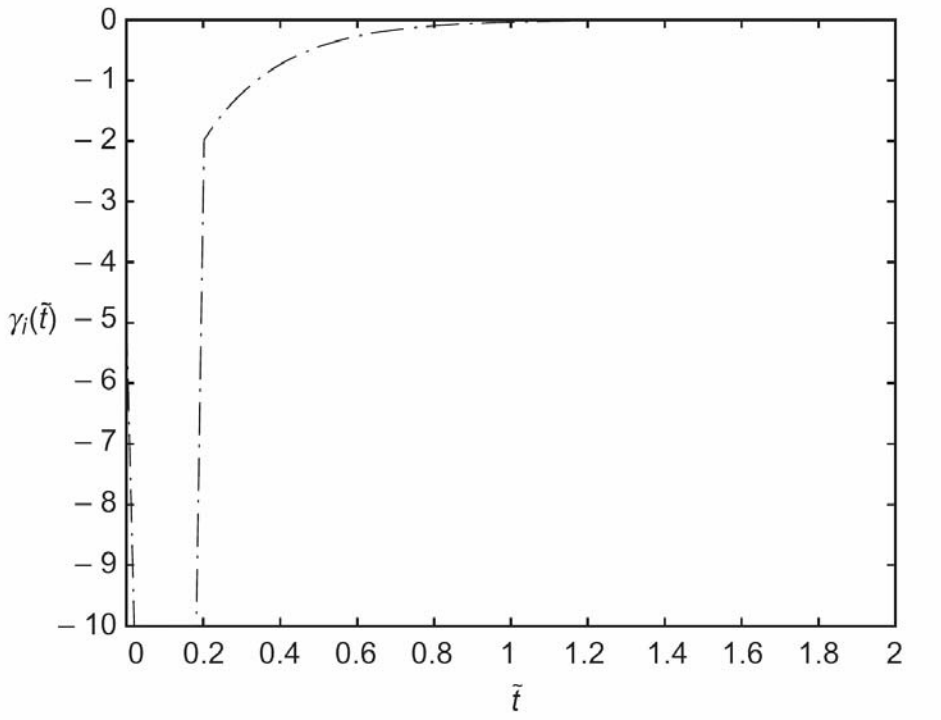
- When both the refractory periods are introduced into the Spike Response model, the reset kernel function becomes

$$\gamma_i(\tilde{t}) = \underbrace{-Ru(\tilde{t})u\left(-(\tilde{t}-\Delta_R)\right)}_{\text{Negative going pulse of magnitude } -R \text{ (absolute refractory period)}} \underbrace{- R_0 \exp\left(-\frac{\tilde{t}-\Delta_R}{\tau_r}\right)u(\tilde{t}-\Delta_R)}_{\text{An exponential that decays from } -R_0 \text{ delayed In time by } \Delta_R}$$

- where $\tilde{t} = t - t_i^k$

Accounting for Refractoriness

- The kernel function $\gamma_i(\tilde{t})$ has a large negative pulse followed by an exponential decay
- Assume:
  - all EPSPs and IPSPs have identical shapes and their amplitude and sign is decided by the premultiplying W_{ji}
- Cell potential can be written as



$$v_i(t) = \sum_{t_i^k \in \mathcal{T}_i} \gamma_i(t - t_i^k) + \sum_j \sum_{t_j^k \in \mathcal{T}_j} w_{ji} \kappa(t - t_j^k)$$

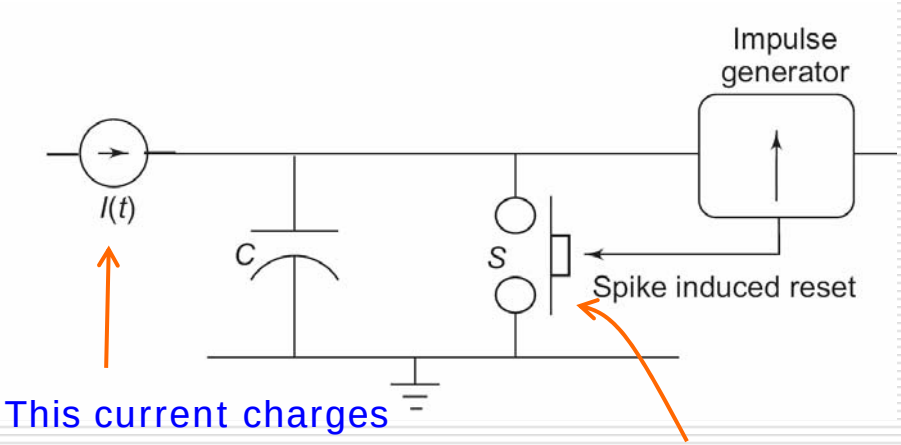
Integrate and Fire (IF) Neurons

- Simple and powerful spiking neuron model
- Various versions based on sub-threshold operation and a potential threshold for spike generation, motivated from an electrical model of the neuron membrane
- Two types
  - Ideal non-leaky IF neuron
  - Ideal leaky IF neuron

Non-leaky IF Neuron

- Capacitor is responsible for sub-threshold generation
- For constant I capacitor voltage increases linearly in time.
- When capacitor voltage equals threshold V_θ, neuron is assumed to fire an output pulse
- This initiates a reset action that brings the capacitor voltage back to the rest value (assumed to be zero)

Electrical model of an integrate and fire neuron



This current charges the capacitor

A switch that models the reset action by short circuiting the capacitor

Time dependence of the capacitor voltage

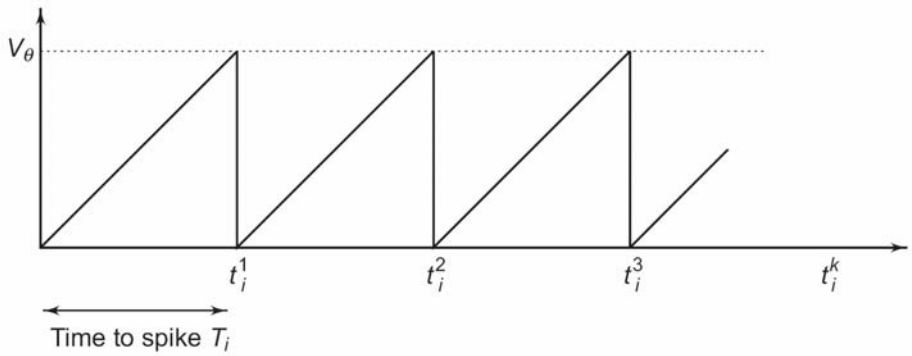
$$C\dot{v}_i = I_i(t)$$

Assuming the current to be constant → $v_i(t) = \frac{I}{C}t$

Non-leaky IF Neuron

- Charge that accumulates between two firing times is CV_θ
- Any arbitrarily small input current will increase the charge of the capacitor and will eventually cause the capacitor to reach the threshold causing the neuron to fire.
- No forgetting of information

Voltage waveform of cell potential $V_i(t)$



The time to spike T_i is

$$T_i = \frac{CV_\theta}{I}$$

assuming that neuron starts from zero potential. So neuron firing frequency

$$f = \frac{1}{T_i} = \frac{I}{CV_\theta}$$

When neuron current varies in time, successive occurrence of spikes can be computed from

$$\int_{t_i^k}^{t_i^{k+1}} I_i(t)\,dt = CV_\theta$$

Non-leaky IF Neuron

- The absolute refractory period is modelled by setting the capacitor voltage to zero for fixed time Δ_R immediately following spike generation. Any current that is coming during this time interval is shunted by the switch opened after a time $t_i^n + \Delta_R$
- This has the effect of reducing the frequency of generation spikes as

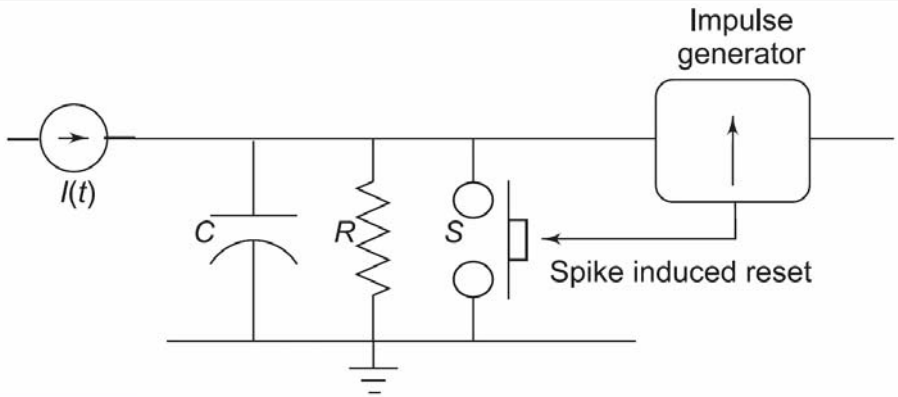
$$f = \frac{1}{\frac{CV_\theta}{I} + \Delta_R}$$

$$= \frac{I}{CV_\theta + I\Delta_R}$$

Leaky IF Neuron

- In reality, the neuron membranes leak and so the cell potential has a tendency to decay back towards its resting value
- A leakage resistance in parallel with the capacitor models this aspect

Electrical model of a leaky IF neuron



Resistor allows the capacitor voltage to discharge with a time constant

$$\tau_m = RC$$

Capacitor voltage is

$$C\dot{v}_i + \frac{v_i}{R} = I_i(t)$$

$$\tau_m \dot{v}_i = -v_i(t) + RI_i(t)$$

$\tau_m \rightarrow$ membrane time constant

Leaky IF Neuron

- The cell potential becomes

$$v_i(t) = v(0^-)\exp\left(-\frac{t}{\tau_m}\right) + IR\left[1 - \exp\left(-\frac{t}{\tau_m}\right)\right]$$

Leakage term: initial voltage across the capacitor leaks to zero

Capacitor charges up to its asymptotic value IR exponentially with time constant τ_m

- In the absence of inputs, capacitor voltage will decay to zero.
- A spike is fired once voltage crosses threshold and the neuron goes into reset condition; switch is closed and capacitor voltage is short circuited to zero
- Now, small currents will not be able to charge capacitor fast enough to overcome the leakage through resistance. There has to be a minimum constant current, threshold current I_θ which satisfy the condition $I_\theta R = V_\theta$
- Only currents larger than I_θ are capable of generating spikes

Leaky IF Neuron

- Starting from reset condition, a spike will occur at a time T_i such that

$$IR\left[1-\exp\left(\frac{-T_i}{\tau_m}\right)\right]=V_\theta \quad \Rightarrow \quad T_i=-\tau_m \ln\left(1-\frac{V_\theta}{IR}\right)$$

- Firing frequency of leaky IF neuron becomes

$$f=\frac{1}{T_i}=\frac{1}{-\tau_m \ln\left(1-\frac{V_\theta}{IR}\right)}$$

- To model refractoriness, it is assumed that after a spike is generated any input currents are shunted out by the switch for a fixed time interval Δ_R

$$f=\frac{1}{T_i+\Delta_R}=\frac{1}{\Delta_R-\tau_m \ln\left(1-\frac{V_\theta}{IR}\right)}$$

External Current

- Note: external current I(t) is to be generated by the pulses of other presynaptic neurons in a network
- Assumption: form of spikes in the IF class of models is the delta function $\delta(\cdot)$
- Input current as a function of presynaptic firings of delta functions

$$I_i(t) = \sum_j q_{ji} \sum_{t_j^k \in \mathcal{T}_j} \delta(t - t_j^k)$$

Quantum of charge deposited on the capacitor from presynaptic neuron j to postsynaptic neuron i

External Current

- Replacing the $\delta(\cdot)$ functions with current pulses of finite width changes current to

$$I_i(t) = \sum_j q_{ji} \sum_{t_j^k \in \mathcal{T}_j} \alpha(t - t_j^k)$$

- One of the various forms of above α functions is

$$\alpha(t) = \frac{t - \Delta}{\tau_s^2} \exp\left(\frac{-t - \Delta}{\tau_s}\right) u(t - \Delta)$$

axonal transmission delay

time constant of the order of few milliseconds

External Current

- Current form is chosen to model the low pass characteristics of a synapse with simple exponential decay

$$\alpha(t) = \frac{1}{\tau_s} \exp\left(-\frac{t}{\tau_s}\right) u(t)$$

- The current thus is

$$I_i(t) = \sum_j q_{ji} \sum_{t_j^k \in \mathcal{T}_j} \exp\left(-\frac{t - t_j^k}{\tau_s}\right) u(t - t_j^k)$$

- Note the correspondence between external current and finite pulse width current

IF Neuron Model: A Special Case of Spiking Neuron Model

- Reset of cell potential after firing time t_i^k can be modelled by assuming an outgoing δ-current pulse

$$I_i^O(t) = -\underbrace{C(V_\theta - v_r)}\sum_{t_i^k \in \mathcal{T}_i} \delta(t - t_i^k)$$

Resting potential of the neuron

Each time neuron fires, this quantum of charge will be removed from capacitor causing neuron to reset

- Capacitor voltage for IF neuron becomes

$$\tau_m \dot{v}_i = -v_i(t) + RI_i^I(t) + RI_i^O(t)$$

IF Neuron Model: A Special Case of Spiking Neuron Model

- Assuming constant input current $I_i^I(t) = I$ and considering only the last firing time of neuron i makes the capacitor voltage

$$v_i(t) = v_i(0)\exp\left(-\frac{t}{\tau_m}\right) + RI\left(1 - \exp\left(-\frac{t}{\tau_m}\right)\right) \underbrace{-(V_\theta - v_r)\exp\left(-\frac{t - t_i^n}{\tau_m}\right)}$$

Assuming v_r=0, this term makes the cell potential decrease suddenly at the instant of firing by V_θ .This decrement gradually decays to zero with time constant τ_m

- Assuming the input current as a function of current pulses of finite width, capacitor voltage can be recast into

$$v_i(t) = \sum_{t_i^k \in \mathcal{T}_i} \gamma_i(t - t_i^k) + \sum_j \sum_{t_j^k \in \mathcal{T}_j} w_{ji}\kappa(t - t_j^k)$$

Spiking neuron model form!

IF Neuron Model: A Special Case of Spiking Neuron Model

- Assuming the input current as a function of current pulses of finite width, capacitor voltage can be recast into

$$v_i(t) = \sum_{t_i^k \in \mathcal{T}_i} \gamma_i(t - t_i^k) + \sum_j \sum_{t_j^k \in \mathcal{T}_j} w_{ji} \kappa(t - t_j^k)$$

Spiking neuron model form!

- where

$$\gamma_i(t - t_i^k) = -(V_\theta - v_r) \sum_{t_i^k \in \mathcal{T}_i} \exp\left(-\frac{t - t_i^k}{\tau_m}\right)$$

Hence IF neuron model is a special case of the spiking neuron model

$$w_{ji} = \frac{R q_{ji}}{\tau_m}$$

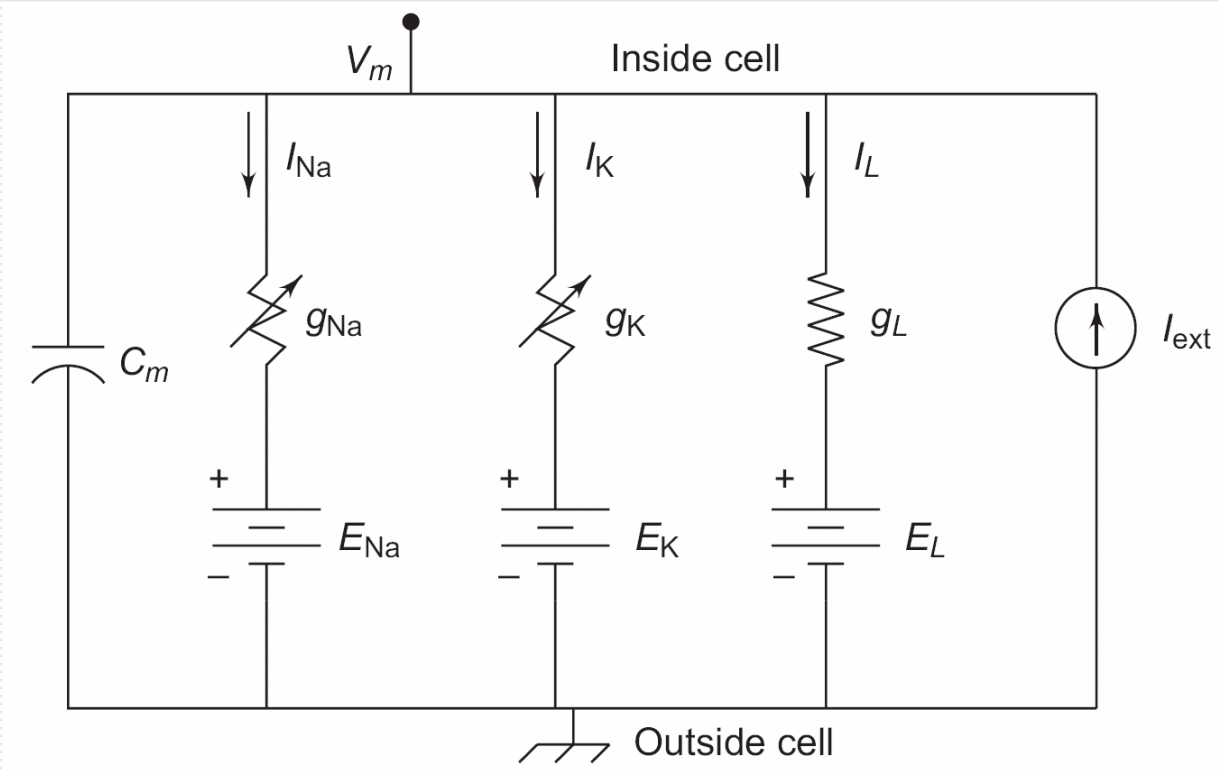
$$\kappa(t - t_j^k) = \frac{1}{1 - (\tau_s / \tau_m)} \left[\exp\left(-\frac{t - t_j^k}{\tau_m}\right) - \exp\left(-\frac{t - t_j^k}{\tau_s}\right)\right] u(t - t_j^k)$$

Conductance Based Models

- Models based on the actual workings of excitable membranes
- Dynamic nature of membrance conductances that take place during an action potential have been understood from two fundamental techniques the *space clamp* and *voltage clamp*
- In an axon, the membrane potential is determined by three conductances
  - Voltage dependent leak conductance g_L
  - Voltage dependent sodium conductance g_{Na}
  - Voltage dependent potassium conductance g_K

Hodgkin-Huxley Model of an Axon

- Based on an electrical equivalent circuit that incorporates a capacitance with three conductances



Electrical Equivalent Circuit of Axonal Membrane

- The current flow across the membrane has two major components: one that charges membrane capacitance and other that is generated by the movement of specific ions across the membrane
- The latter ionic current include 3 components
  - A sodium current I_{Na}
  - Potassium current I_k
  - A small leakage current by chloride ions I_L
- Currents are assumed to be controlled by batteries E_{Na} , E_K , E_L that correspond to the equilibrium potentials for each of the ions

Electrical Equivalent Circuit of Axonal Membrane

- The batteries for E_{Na} and E_K are placed in series with variable conductances g_{Na} , g_K and the battery for E_L is placed in series with a passive conductance g_L
- KCL at the inside node is $C_m \dot{v}_m + I_{\text{ion}} = I_{\text{ext}}$
- Hodgkin-Huxley equation describing the circuit is

$$C\dot{v}_m = g_L(E_L - v_m) + g_{\text{Na}}(E_{\text{Na}} - v_m) + g_{\text{K}}(E_{\text{K}} - v_m) + I_{\text{ext}}$$

Gating the Channel Access

- Hodgkin and Huxley suggested a simple model to account for voltage sensitivity of the sodium and potassium conductances with the following assumptions:
  - Each channel has two conducting states – an open state in which ions are free to cross through the pore, and a closed state in which the pore is blocked
  - Many individual ion channels with small ionic conductances determine the measurable behaviour of the whole membrane

Gating the Channel Access

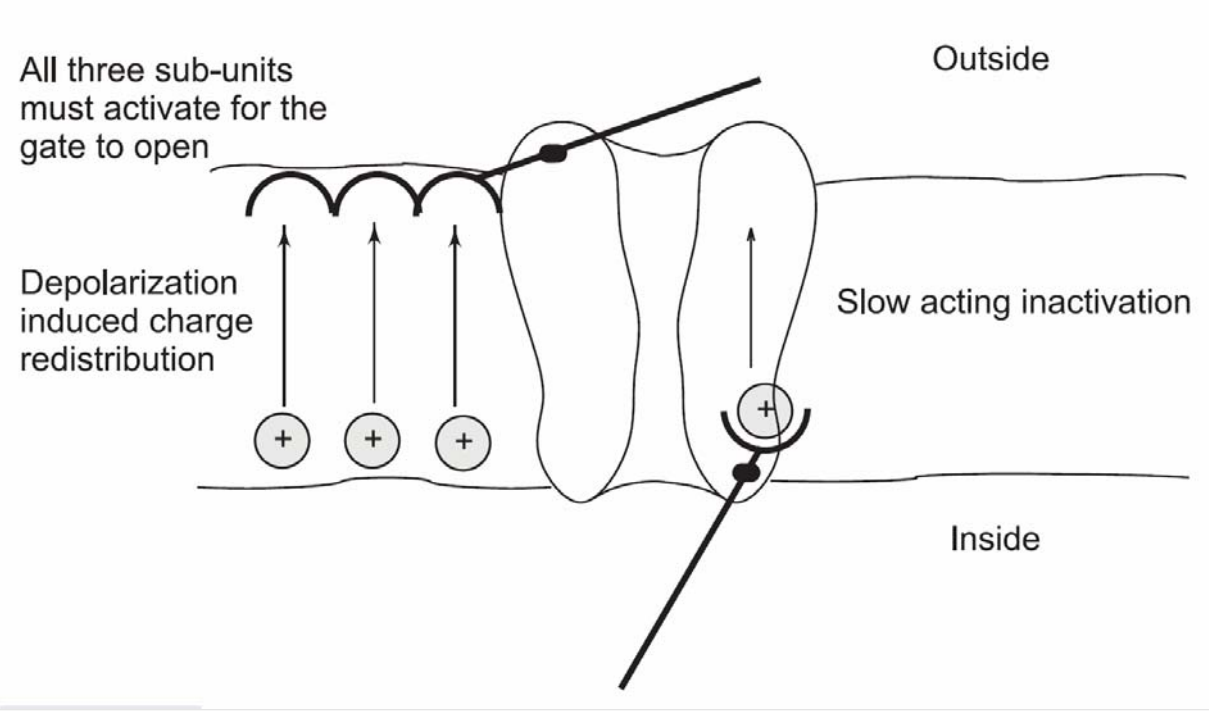
- Channels behave as though free passage through the pore were controlled by different *gates*
- Any gate can be in one of two states – *permissive* or *non-permissive*
  - When all gates for a channel are in permissive state, the channel is *open* and ions can pass through it
  - If any one of the gates is in non-permissive state, ions cannot flow and the channel is *closed*
- A change in membrane potential alters the probability p of an individual gate to be in the permissive or non-permissive state, and thus decides whether a channel will be in the open or closed state

Gating the Channel Access

- P is the fraction of gates in a population of channels that are in the permissive state
- P-1 is the fraction that are in the non-permissive state
- When a larger fraction of the total population of channels is open, the total membrane conductance to that ion increases
- The maximum conductance $\overline{G}$ for an ion is reached when all the channels for that ion are open

Sodium Channel Activation Gates

- **Assumption:** Sodium channel protein with an activation gate comprising three identical sites



Sodium Channel Activation Gates

- The sites are activated when positive charges attach to the sites
- While the activation gate is normally closed, all three sites must activate for the gate to undergo a structural change and open
- Depolarization causes positive charges residing on the inside surface of the membrane to redistribute to the outer surface, thereby activating the activation gate sites

Sodium Channel Activation Gates

- m = (0, 1) be the probability that a particular site is active
- Rate of change of probability is decided by the following
  - Sites that are inactive can activate
  - Sites that are active can become inactive
- Transition from inactive to active state is governed by a voltate dependent rate $\alpha_m(v)$
- Transition from active to inactive state is proportional to a voltage dependent rate $\beta_m(v)$
- $(1-m)\alpha_m(v)$ gives the probability that the site activates over a small interval of time
- $(1-m)\beta_m(v)$ gives the probability that the site inactivates over a small interval of time

Sodium Channel Activation Gates

- The rate of change of probability becomes

$$\dot{m} = \alpha_m(v)(1-m) - \beta_m(v)m$$

- With

$$m(t) = m_\infty(V) - (m_\infty(V) - m_0)e^{-t/\tau_m(V)}$$

Initial and final values of m

Time constant

- Rise in g_{Na} exhibits considerable delay following depolarization and there is an S-shaped increase in that can be explained if more that one binding site must be occupied by positive gating particles before the channel can open
- If there are x sites, the probability of channel open would be proportional to m^x
- The actual rise in sodium conductance following a depolarizing step suggests that x =3 for the sodium channel i.e. three binding sites must be occupied by gating particles before the channel will conduct

Sodium Channel Inactivation Gates

- The inactivation gate normally remains open
- This gate is controlled by a single site at which a positive gating charge is already bound, causing the site to be active
- When the cell depolarizes, positive charges redistribute towards the outer membrane surface
- When the charges detach, the site becomes inactive and the gate closes

Sodium Channel Inactivation Gates

- h be the probability that the site is active
- As cell undergoes depolarization, m increases and h decreases
- During hyperpolarization, m decreases and h increases
- The rate of change of probability of the inactivation gate to be active is

$$\dot{h} = \alpha_h(v)(1-h) - \beta_h(v)h$$

h declines exponentially at a rate governed by the individual rates $\alpha_m(v)$ and $\beta_m(v)$

- which gives

$$h(t) = h_\infty(V) - (h_\infty(V) - h_0)e^{-t/\tau_h(V)}$$

Time constant

Sodium Channel Inactivation Gates

- The closing of the inactivation gate is slower than the opening of the activation gate
- The probability P_{Na} that the sodium channel is open is governed by the *joint probability* that the activation and inactivation gates are simultaneously open
- This is the product of the probability that the three sites of the activation gate and the single site of the inactivation gate are active together
- It can be put as

$$P_{\mathrm{Na}} = m^3 h$$

Potassium Channel Activation Gates

- For potassium ions, there is only an activation gate and no inactivation gate
- The potassium activation gate is assumed to be normally closed and is opened when a requisite number of sites on the gate are activated
- Let n be the probability of activation of a site of the activation gate for potassium channel
- As before, the probability that the channel is open in n^x
- An analysis carried out for the charge in potassium conductance following a step depolarization suggests that $x = 4$ for the voltage sensitive potassium channel i.e. the rise in potassium conductance is found to be proportional to n^4

Potassium Channel Activation Gates

- Assumption: the channel protein has an activation gate comprising four identical sites, which are activated when positive charges attach to these sites
- The rate of change of probability n is

$$\dot{n} = \alpha_n(v)(1-n) - \beta_n(v)n$$

- which gives

$$n(t) = n_\infty(V) - (n_\infty(V) - n_0)e^{-t/\tau_n(V)}$$

Time constant

- The probability P_K that the potassium channel is open is then given by the joint probability that the four sites are simultaneously active: $P_K = n^4$

Difference between Potassium and Sodium Channels

- The time constant τ_m is almost an order of magnitude smaller than τ_h and τ_n
- This fast activation-slow inactivation for sodium channels along with the slower activation of potassium channels together accounts for the positive going action potential observed

Rate Constants

- The sodium and potassium conductances can be modelled as

$$g_{\mathrm{Na}} = \overline{G}_{\mathrm{Na}} \cdot m^3(t)h(t)$$

$$g_{\mathrm{K}} = \overline{G}_{\mathrm{K}} \cdot n^4(t)$$

Maximal sodium and Potassium conductances

- And the rate constants after careful experimental procedures are as follows

$$\alpha_m(v) = \frac{0.1(v-40)}{\exp(\frac{v-40}{10}) - 1} \qquad \alpha_h(v) = 0.07\exp\left(\frac{v-65}{20}\right) \qquad \alpha_n(v) = \frac{0.01(v-55)}{\exp(\frac{v-55}{10}) - 1}$$

$$\beta_m(v) = 4\exp\left(\frac{v-65}{18}\right) \qquad \beta_h(v) = \frac{1}{\exp(\frac{v-35}{10}) + 1} \qquad \beta_n(v) = 0.125\exp\left(\frac{v-65}{80}\right)$$

Cable Equation

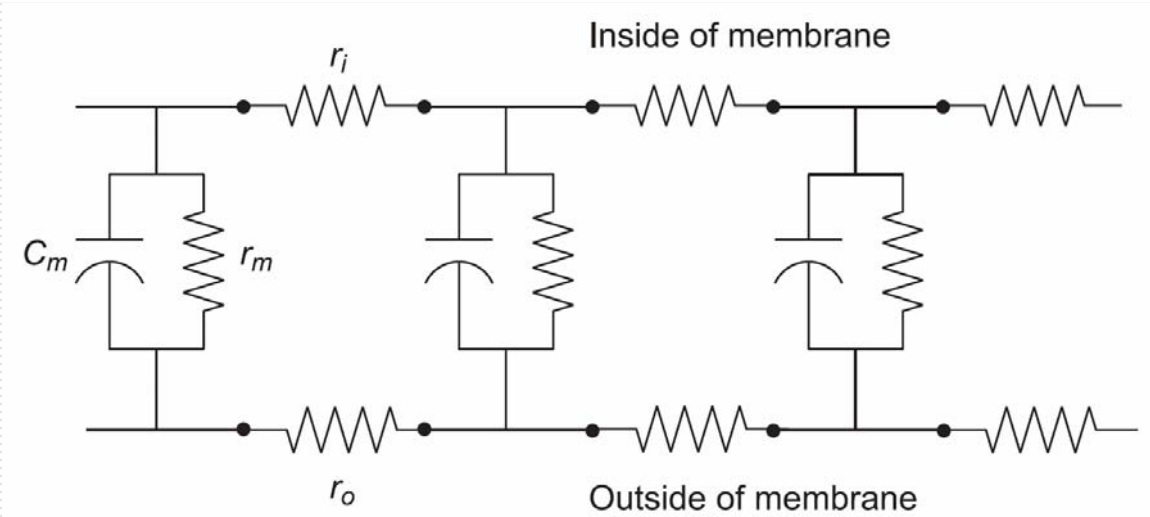
- Neuron models discussed so far focussed on sub-threshold integration and spike generation and have no spatial structure
- Wilfrid Rall addressed the following questions
  - How inputs to the neuron affect the potential in the soma?
  - How does the PSP spread through the dendritic tree?
- Result: Cable equation which describes how local changes in membrane potential spread along the dendritic branches, and how this spread depends on the electrical properties of the membrane

Dendritic Behaviour ≡ Electric Cable

- The behaviour of dendrites is much like that of an electrical cable on various counts
  - There is an axial resistance that manifests itself due to the poor conducting properties of the axoplasm
  - The membrane is a leaky insulator
  - There is a capacitance that arises because of the charges that accumulate on either sides of the insulating membrane. This capacitance can be of the order of 1 $\mu F/cm^2$

One-dimensional Cable Theory

- The sub-threshold operation of IF neuron was modelled with the help of a capacitor in parallel with a resistance
- It was assumed while modelling a membrane that the behaviour can be approximated by a series of *compartments* (capacitor-resistor combination in parallel) connected by axial resistances



One-dimensional Cable Theory

- KCL for a compartment is

Membrane current ⟶ $i_m = \frac{v}{r_m} + C_m \dot{v}$

Membrane capacitance

Membrane resistance

- The axial current i_a that arises along an infinitesimal distance Δx that causes a voltage drop Δv can be written as

$$i_a = \frac{\Delta v}{(r_o + r_i)\Delta x} = \left(\frac{1}{r_o + r_i}\right)\frac{dv}{dx}$$

One-dimensional Cable Theory

- Any changes in the axial current can arise only from the membrane current i_m . So,

$$i_m = \frac{di_a}{dx} = \left(\frac{1}{r_o + r_i}\right)\frac{d^2v}{dx^2}$$

- Equating two membrane current expressions

$$\frac{v}{r_m} + C_m\frac{\partial v}{\partial t} = \left(\frac{1}{r_o + r_i}\right)\frac{\partial^2 v}{\partial x^2}$$

One-dimensional Cable Equation

- In a more compact form the voltage can be written as

$$v = \lambda^2 \frac{\partial^2 v}{\partial x^2} - \tau \frac{\partial v}{\partial t}$$

One dimensional cable equation

- Where $\lambda^2 = \left(\frac{r_m}{r_o + r_i} \right)$ and $\tau = r_m C_m$

Two Special Cases: Case 1

- Assumption: A voltage $v = V$ has been established using a voltage clamp i.e. the membrane voltage v is maintained constant in time at a particular point in space ($\frac{\partial v}{\partial t} = 0$)
- Cable equation reduces to

$$v = \lambda^2 \frac{\partial^2 v}{\partial x^2}$$

- With solution $v(x) = V \exp(-x/\lambda)$

- Inference: the voltage V falls of exponentially with distance from the point where it is held constant

Two Special Cases: Case 2

- Consider a space clamp, where the membrane voltage is held constant in space and so partial derivatives in space vanish
- Cable equation therefore reduces to

$$v = -\tau \dot{v}$$

- With solution

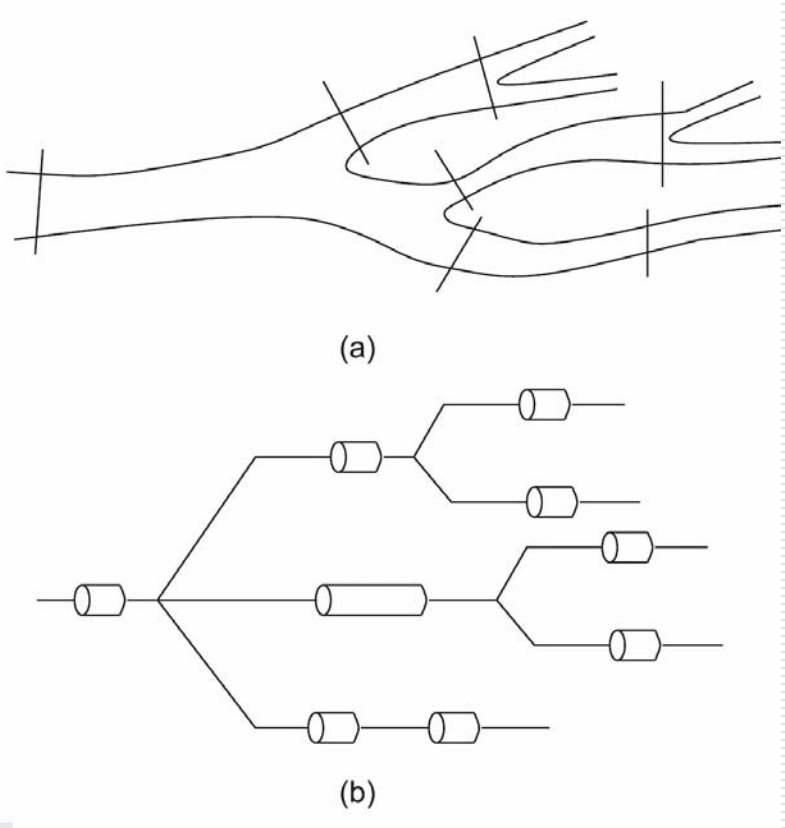
$$v(t) = v(0^-)\exp(-t/\tau)$$

An exponential decay in time with time constant τ

- Inference: the voltage perturbations will decay in time exponentially, if the membrane is space clamped. If a step input is given then the voltage will rise exponentially to the step value

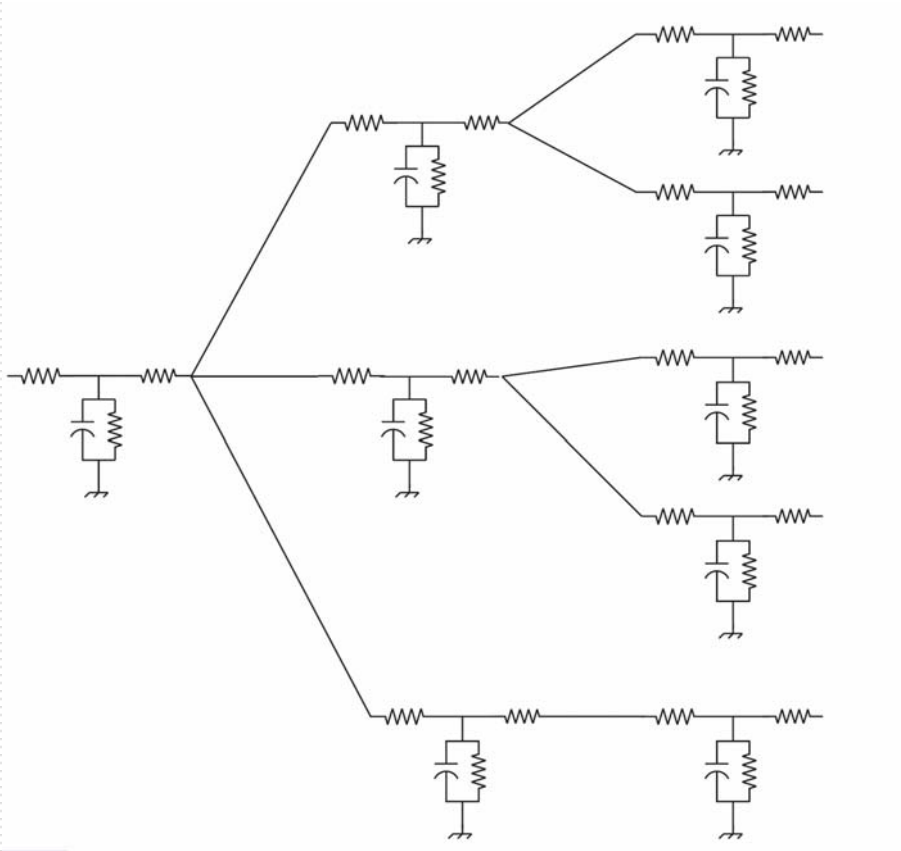
Compartmental Models

- Introduced by Rall
- Dendritic tree
  - a collection of short cylindrical segments, where the tree attached at the end of each segment acts as a sink for the longitudinal current
  - divided into *compartments* whenever branching takes place or dendritic diameters change significantly
- Compartmental modelling approach
  - a finite-difference or discrete approximation to the non-linear cable equation



Compartmental Models

- A single dendrite tree model can include thousands of compartments and as many equations for each time step
- Dendritic segments that are physically short are assumed to be equipotential and are modeled by a single RC membrane compartment



Compartmental Models

- Compartments are connected to each other in accordance with the topology of the tree with the help of resistances that model the longitudinal resistivity
- Differences in physical properties of membranes and differences in potential are shown by a change in compartment
- A dendritic tree divided into sufficiently small compartments has a solution to the compartmental model that is close to that of the continuous cable model
- A compartment can represent a patch of membrane with a variety of voltage-gated (excitable) and synaptic (time-varying) channels

Compartments yield Linear Differential Equations

- KCL for a segment of a dendritic tree is

Sum over all axial current that flow into the region through cross section boundaries

$$\sum i_a - \int_A i_m dA = 0$$

Transmembrane current density

- In computer simulation the neuron is divided into compartments that are small enough so that the spatially varying transmembrane current i_m in any compartment j is well approximated by its value i_{mj} at the center of the compartment

$$i_{mj} A_j = \sum i_a$$

Compartments yield Linear Differential Equations

- From ohm's law, the voltage drop $(V_k - V_j)$ between the centers of two compartments k and j, divided by the resistance r_{jk} of the path between them yields the axial current

$$i_{mj} A_j = \sum_k \frac{V_k - V_j}{r_{jk}}$$

- Membrane current $i_{mj} A_j$ is the sum of capacitive and ionic components

Membrane capacitance of compartment j

$$i_{mj} A_j = C_j \frac{dV_j}{dt} + I(V_j, t)$$

Models the effect of ionic conductances

Compartments yield Linear Differential Equations

- Combining membrane current equations yield

$$C_j \frac{dV_j}{dt} + I(V_j, t) = \sum_k \frac{V_k - V_j}{r_{jk}}$$

- Adding injected currents I_{ext} result in

$$C_j \frac{dV_j}{dt} + I(V_j, t) = \sum_k \frac{V_k - V_j}{r_{jk}} + I_{\text{ext}}$$

- More the number of compartments the greater the simulation accuracy. Doubling the number of compartments increases the accuracy by four!

Computing with Spiking Neurons

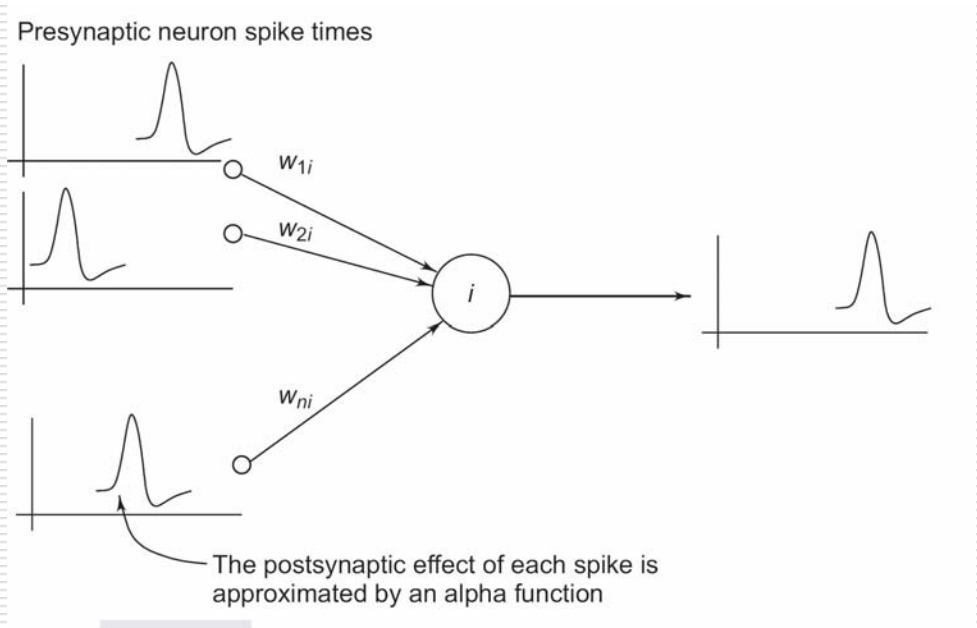
- Focus will be on the nature of computations that can be performed with temporal patterns
- Recent research involve computation in networks of neurons with unreliable synapses that compute using space-rate codes
- There are features of the spiking neuron that have no analog in the artificial neuron world!

Coincidence Detection

- A coincidence detector can check whether the firing times of presynaptic neurons are almost the same or not
- If the firing times of presynaptic neurons encode numbers, then a coincidence detector can tell us whether or not these numbers have almost the same value

Coincidence Detection

- Assumptions:
  - Each of n presynaptic neurons to neuron I has the same transmission delay $\Delta_{ji} = \Delta$ and their weights $w_{ji} = 1$
  - Each presynaptic neuron fires exactly once and generates a PSP
  - They generate identical PSP functions



$$\kappa(t-\Delta) = \frac{1}{1-(\tau_s/\tau_m)}\left[\exp\left(-\frac{t-\Delta}{\tau_m}\right) - \exp\left(-\frac{t-\Delta}{\tau_s}\right)\right]u(t-\Delta)$$

Coincidence Detection

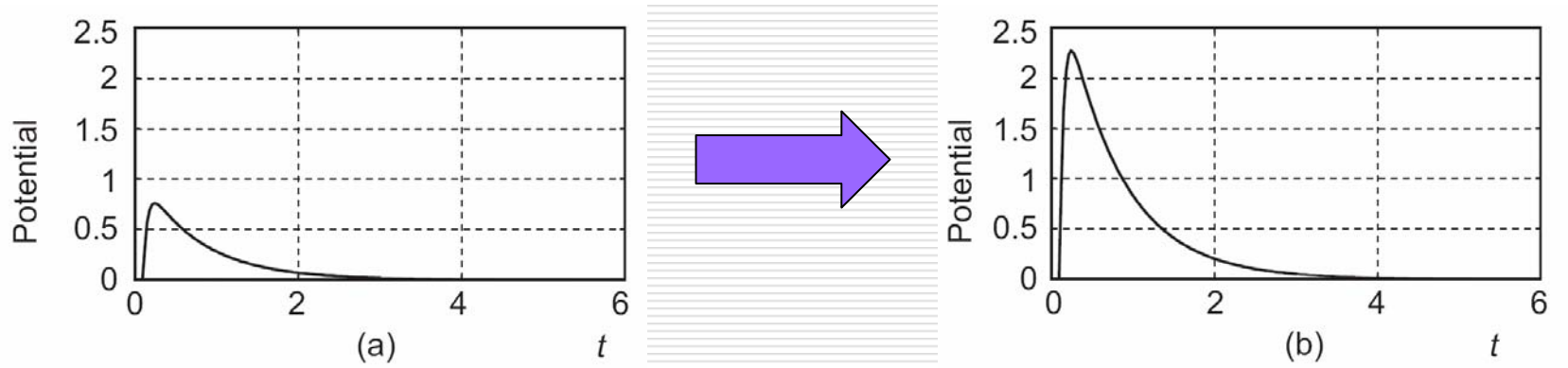
- PSPs are temporarily integrated by the neurons in a weighted fashion
- If the PSPs are fired at intervals that are far apart, the integrated cell potential has a small chance of exceeding the threshold
- If the PSPs are spaced at intervals that are close to one another, they add up and the chances that the integrated cell potential exceeds the threshold is much larger

Coincidence Detection

- If all n presynaptic neurons fire together (at the same instant) at time t_1 then the PSPs are simply superposed in time and the integrated cell potential becomes

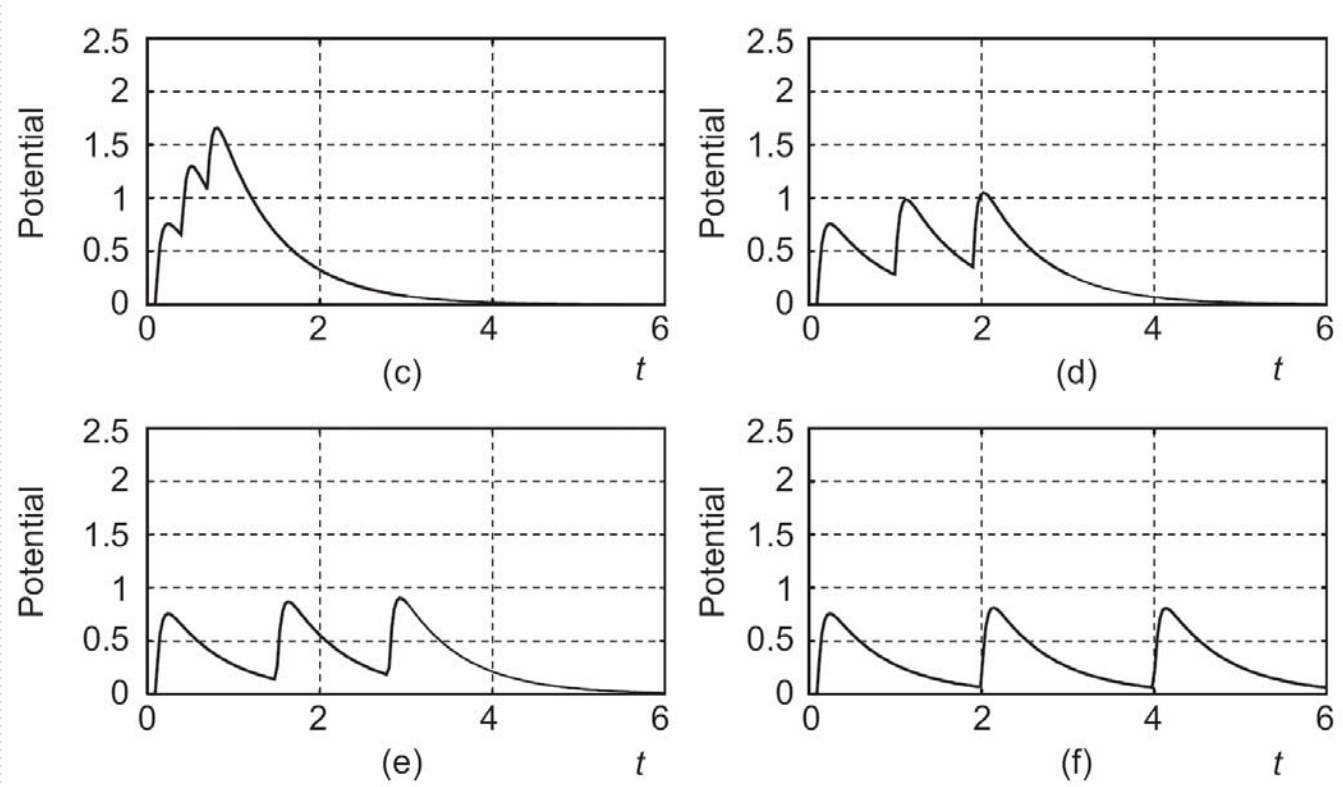
$$v_i(t) = \sum_j w_{ji}\kappa(t) = n\kappa(t)$$

- The resulted integrated cell potential is exactly the same shape, scaled up n times



Coincidence Detection

- As the PSPs get spaced out in time, the peak of the integrated potential falls



Coincidence Detection

- There is an upper bound on the separation allowed that ensures that the integrated potential exceeds a threshold
- For a given set of time constants, one can find values of spike separation d_1, d_2 where $0 < d_1 < d_2$ and a threshold V_θ such that the integrated soma potential $v_i(t)$ remains less than V_θ for any arbitrary number of EPSPs that fire at times separated by a temporal distance greater than or equal to d_2
- $v_i(t)$ will reach a value greater than V_θ for *any two* EPSPs that are separated temporally by a temporal distance that is less than or equal to d_1

Element Distinctness Function ED_n

- The spiking neuron is capable of computing the *element distinctness* function $ED_n: R^n \rightarrow \{0,1\}$
- ED function essentially computes whether the firing times $t_1,\ldots t_n$ of any pair of presynaptic neurons lie within a certain interval. The function can be defined as follows

$$ED_n(t_1, \ldots, t_n) = \begin{cases} 1 & \text{if there are } j \neq j' \text{ such that } |t_j - t_{j'}| \leq d_1 \\ 0 & \text{if } |t_j - t_{j'}| \geq d_2 \ \forall j \neq j' \end{cases}$$

- A conventional artificial neural network that is to compute element distinctness function can become rather complex

Element Distinctness Function ED_n

- Theorem: Any layered networks of TLNs that computes ED_n needs to have at least $\log(n!) \geq n/2 \cdot \log n$ threshold neurons in its first layer

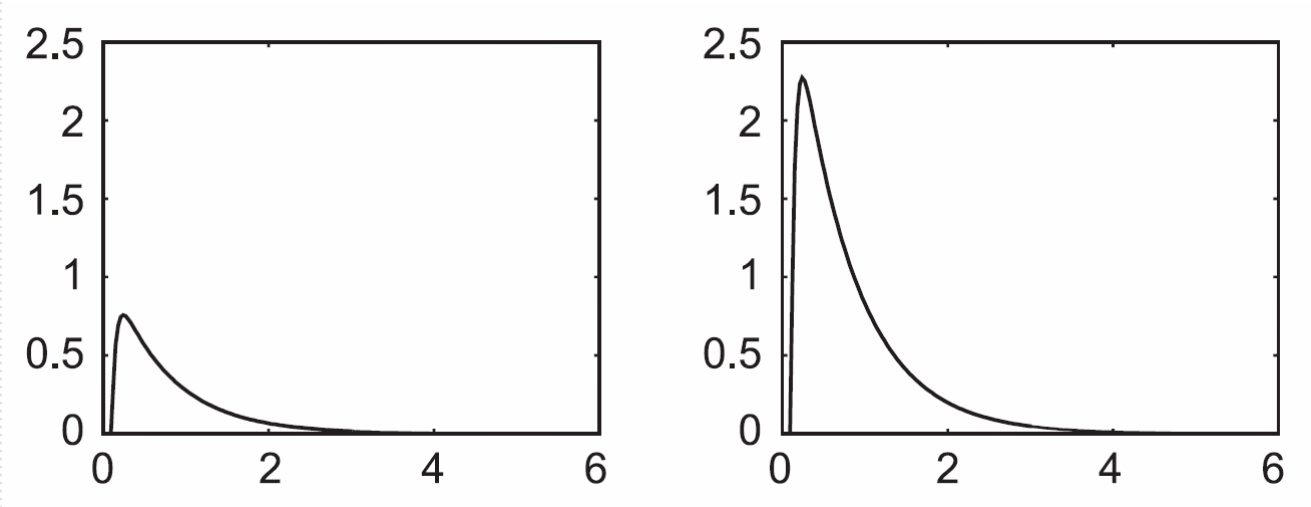
- Theorem: Any feedforward neural network consisting of arbitrary sigmoidal neurons needs to have at least $(n-4)/2$ neurons in order to compute ED_n

Temporal Radial Basis Function Neurons

- Firing threshold V_θ and weights w_{ji} can be tuned in such a way that as to permit the spiking neuron to detect specific temporal patterns in the input
- One can raise the value of V_θ to a point where the neuron fires a spike only if all the presynaptic pulses occur almost simultaneously
- Let us consider three presynaptic neurons as inputs with weights $w_{ji} = 1$

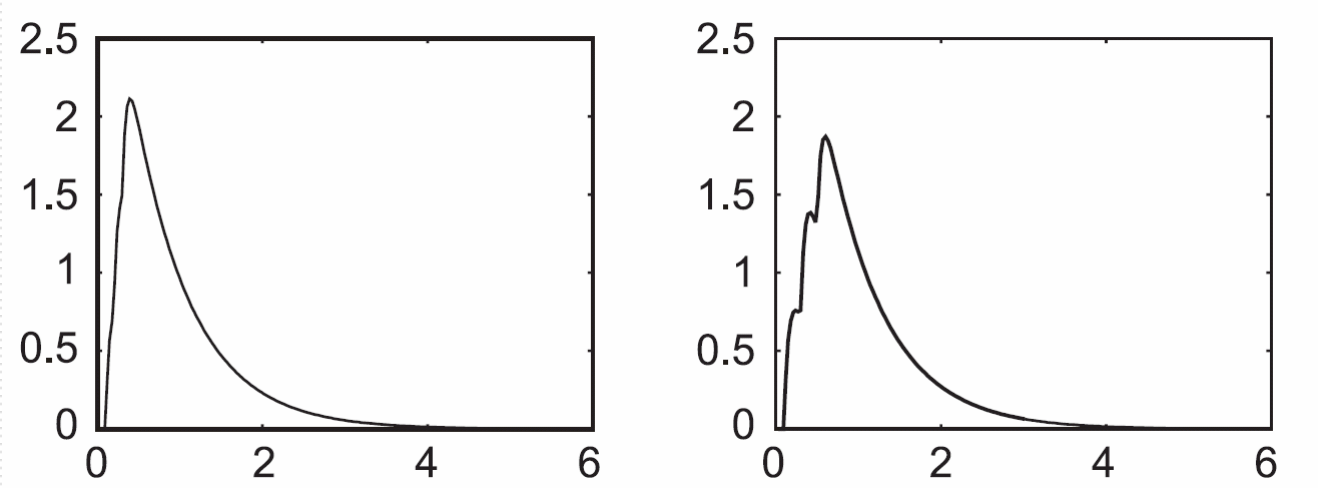
Temporal Radial Basis Function Neurons

- Each neuron fires a spike that results in the EPSP kernel function
- When all three occur simultaneously the resulting soma potential is scaled up three times
- If we fix the threshold to be 2.0 then simultaneous firing makes the soma potential exceed the threshold



Temporal Radial Basis Function Neurons

- A separation of 0.1 between the firing times of the three neurons still make the soma potential exceed the threshold
- A separation of 0.2 between the firing times results in a soma potential that falls short of the threshold



Temporal Radial Basis Function Neurons

- Spiking neuron fires only if *all* the presynaptic neurons fire at approximately the same time
- Infact the neuron is detecting whether or not the firing times of all presynaptic neuron lie within a certain time interval
- This neuron is *temporal radial basis function* (TRBF) neuron
- A TRBF neuron fires only if, for some constant T_I all presynaptic neurons have firing times $t_j \approx T_I - \Delta_{ji}$ with the transmission delay vector $(\Delta_{1i},\ldots,\Delta_{ni})$ behaving like the temporal center of the neuron

Computing Weighted Sums

- For multilayered or recurrent computations with spiking neurons we need to generate analog or numeric outputs that are dependent on the input through a weighted sum $\sum_j \tilde{w}_{ji} x_j$ for numeric inputs $X \in R^n$ and weight coefficients $\tilde{w}_{ji} \in R^n$. This summation has to be computed through the firing time of neuron i
- Therefore the mechanism has to shift the firing time of the neuron in accordance with the magnitude of the weighted sum

Computing Weighted Sums

- Assumptions
  - each PSP kernel has a linearly rising phase that starts after the axonal transmission delay Δ_{ji} elapses
  - The function $\kappa_{ji}(t)$ can be approximated by a straight line equation with slope $m_{ji} \in R$ for a time interval $T_L > 0$
  - Presynaptic neurons j fire at times $t_j = T_I - x_j$

Coding Numeric Inputs

- The numeric inputs x_j are coded into firing times of presynaptic neurons such that the larger the x_j earlier the corresponding presynaptic neuron fires
- Thus a numeric input vector translates to a vector of firing times of presynaptic neurons

Computing with Weighted Sums

- Let the cell potential v_i reaches the threshold V_θ while all the PSPs generated are still in their linear phases
- This allows to compute the cell potential as a superposition of linear functions
- Assuming that neuron i fires at time t_i we can write

$$\sum_j w_{ji}\kappa_{ji}(t_i - t_j) = V_\theta$$

Computing with Weighted Sums

- Linear approximation of the kernel function for the time period $t_i-(t_j+\Delta_{ji})$ gives

$$\sum_j w_{ji}\kappa_{ji}(t_i - t_j) \approx \sum_j w_{ji}m_{ji}\big(t_i - (t_j + \Delta_{ji})\big) = V_\theta$$

- which yields

$$t_i = \frac{V_\theta}{\sum_j w_{ji}m_{ji}} + \frac{\sum_j w_{ji}m_{ji}(t_j + \Delta_{ji})}{\sum_j w_{ji}m_{ji}}$$

Constant that depends on synaptic weights kernel slopes

- with proper substitutions

$$t_i = \frac{V_\theta}{M} + \sum_j \frac{w_{ji}m_{ji}}{M}\left(T_I - x_j + \Delta_{ji}\right)$$

Computing with Weighted Sums

- The neuron firing time equation can be rearranged as

$$t_i = \frac{V_\theta}{M} + \sum_j \frac{w_{ji} m_{ji}}{M} (T_I + \Delta_{ji}) - \sum_j \frac{w_{ji} m_{ji}}{M} x_j$$

$$= T_O - \sum_j \tilde{w}_{ji} x_j$$

$$T_O = \frac{V_\theta}{M} + \sum_j \frac{w_{ji} m_{ji}}{M} (T_I + \Delta_{ji})$$

$$\tilde{w}_{ji} = \frac{w_{ji} m_{ji}}{M}$$

- Clearly, the firing time of neuron i essentially codes a weighted sum of the inputs
- The larger the weighted sum, the earlier the firing time with respect to time T_O and the smaller the weighted sum, the later the firing time

Spiking Neurons are Universal Approximators

- Neural networks with the linear threshold neurons in their hidden layer can universally approximate continuous functions
- Somehow we have to simulate a linear threshold signal function with a spiking neuron
- Then, spiking neurons can approximate continuous functions with arbitrary accuracy

Simulating Linear Threshold Signal Function

- The linear threshold signal function can be simulated using a spiking neuron by coding the function into the firing times of the neuron

$$S(y) = \begin{cases} 0 & y \leq 0 \\ y & 0 < y < 1 \\ 1 & y \geq 1 \end{cases}$$

Simulating Linear Threshold Signal Function

- The neuron i now fires at times t_i with the function

$$t_i = T_O - \mathcal{S}\left(\sum \tilde{w}_{ji} x_j\right)$$

- where presynaptic neurons fire at times $t_j = T_I - x_j$
- The above equation describes the following:
  - Neuron i does not fire before $T_O - T$ to simulate the condition $\sum \tilde{w}_{ji} x_j \geq 1$
  - Neuron i does not fire after T_O to simulate the condition
  - Neuron i adjusts its firing time between T_O-T and T_O in accordance with the value of $\sum \tilde{w}_{ji} x_j$ to simulate the condition $0 \leq \sum \tilde{w}_{ji} x_j \geq 1$

Simulating Linear Threshold Signal Function

- One can therefore construct a layered network of such neurons that simulate the linear threshold signal function
- Remember: the basis of the entire discussion of universal approximation rests on the assumption that the initial segments of the PSP kernels are linear and computation takes place with these approximated kernels

Universal Approximation Theorem for Spiking Neurons

- Theorem: Any feedforward or recurrent analog neural network that consists of s linear threshold neurons can be simulated arbitrarily closely with a spiking neural network of c spiking neurons with analog inputs and outputs encoded by temporal delay of spikes. This holds even if the spiking neurons are subject to noise

Universal Approximation Theorem for Spiking Neurons

- Corollary: Any given continuous function $f : [0, 1]^n \rightarrow [0, 1]^p$ can be approximated arbitrarily closely by a network of spiking neurons with inputs and outputs encoded by temporal delays