The membrane equation

Using Kirchhoff’s law we can write down the membrane equation (see P Dayan and L.F. Abbott, Theoretical Neuroscience)

\[ I_{\text{inj}} \]

\[ C \frac{dV_m}{dt} + \frac{(V_m - E_L)}{R} = I_{\text{inj}} \]

\[ \tau \frac{dV_m}{dt} = E_L - V_m + RI_{\text{inj}} \]

\[ \tau = RC \]

membrane time constant (typically 3-100 ms)
Response to a current pulse

\[ \tau \frac{dV_m}{dt} = E_L - V_m + RI_{inj} \]

\[ V_\infty = E_L + RI \]

\[ V_m(t) = V_\infty + (V_m(0) - V_\infty) e^{-t/\tau} \]
Spikes

Injection of a current pulse:

The membrane equation:

\[ \tau \frac{dV_m}{dt} = E_L - V_m + RI \]

« Integrate-and-fire » model:

If \( V = V_t \) (threshold)
then: neuron spikes and \( V \rightarrow V_r \) (reset)

(phenomenological description of action potentials)
Synapses
Synapses

- **Dendrites**
- **Axon**
- **Cell body**
- **Synaptic space**

- **Presynaptic bouton**
- **Transmitter**
- **Receptor**
- **Postsynaptic membrane**

**Action potential**
Synaptic currents

Transfer of charge (=ions): $Q = C \cdot \Delta V$

$$\tau \frac{dV_m}{dt} = E_L - V_m$$

$V_m \rightarrow V_m + \frac{RQ}{\tau}$  at spike time

$\Delta V = Q/C = RQ/\tau$

$$V_m(t) = E_L + \frac{RQ}{\tau} e^{-\frac{t}{\tau}}$$
Excitation/inhibition

In fact, it depends on the equilibrium potential of the synaptic channel.
## Major synapse types

<table>
<thead>
<tr>
<th>Neurotransmitter</th>
<th>Receptor</th>
<th>$E_{rev}$</th>
<th>Properties</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glutamate</td>
<td>AMPA</td>
<td>0 mV</td>
<td>fast</td>
</tr>
<tr>
<td></td>
<td>NMDA</td>
<td>0 mV</td>
<td>slow, voltage-dependent</td>
</tr>
<tr>
<td>GABA</td>
<td>GABA-A</td>
<td>-70 mV</td>
<td>fast</td>
</tr>
<tr>
<td></td>
<td>GABA-B</td>
<td>-100 mV</td>
<td>slow</td>
</tr>
</tbody>
</table>

Also: glycine, acetylcholine…

can change because it depends on ionic concentrations

Dale’s principle: a neuron only expresses one type of neurotransmitter
Several spikes

\[ \tau \frac{dV_m}{dt} = E_L - V_m \]

\[ V_m \to V_m + w \] at spike time

\[ w = \text{« synaptic weight »} \]

Many spikes at times \( t_i^k \) (\( i = \text{synapse}; \ k = \text{spike index} \)):

\[ V_m \to V_m + w_i \] at time \( t_i^k \)

\[ V_m(t) = E_L + \sum_{i,k} PSP_i(t - t_i^k) \]

Where: \[ PSP_i(t) = H(t) w_i e^{-\frac{t}{\tau}} \]

\( H = \text{Heavyside} \)
Coincidence detection
Coincidence detection: principle

\[ \tau \frac{dV}{dt} = E_L - V \]

\[ V \rightarrow V + w \]

Threshold \( V_t \)

Threshold

Spike if \(|d| < -\tau \log \left( \frac{V_t - E_L}{w} - 1 \right)\)
Example in sound localization

Interaural time difference (ITD) depends on sound direction.

sound

spikes
The Jeffress model of sound localization

When the sum of acoustic and neural transmission delays of one side equals that of the other side, the coincidence detector fires maximally.
Integration
The firing rate

Firing rate

\[ F = \frac{1}{\langle T_n \rangle} \]

\[ (T_n = t_{n+1} - t_n) \]
Firing rate for constant current

\[ \tau \frac{dV_m}{dt} = E_L - V_m + RI \]

- Firing condition:
  \[ E_L + RI = V_t \]
  \[ I \geq \frac{V_t - E_L}{R} \]

- Time to threshold:
  \[ T = \tau \log \frac{E_L + RI - V_m(0)}{E_L + RI - V_t} \]
  \[ T = \tau \log \frac{E_L + RI - V_r}{E_L + RI - V_t} \]
  from reset

threshold \( V_t \)
reset \( V_r \)
Current-frequency relationship

\[ F = \frac{1}{T} = \left( \tau \log \frac{E_L + RI - V_r}{E_L + RI - V_t} \right)^{-1} \]
The refractory period

For a few ms after a spike: the neuron cannot produce a spike.
Firing rate with a refractory period

\[ T = \Delta + \tau \log \frac{E_L + RI - V_r}{E_L + RI - V_t} \text{ from reset} \]

\[ F = \frac{1}{T} = \left( \Delta + \tau \log \frac{E_L + RI - V_r}{E_L + RI - V_t} \right)^{-1} \quad \text{max } 1/\Delta \]
Rank order coding
Prey localization by the sand scorpion

Inhibition of opposite neuron

→ more spikes near the source

(polar representation of firing rates)

Conversion rank order code → rate code

Stürtzl et al. (2000). Theory of arachnid prey localization. PRL
Learning
Hebb’s rule

When an axon of cell A is near enough to excite cell B and repeatedly or persistently takes part in firing it, some growth process or metabolic change takes place in one or both cells such that A’s **efficiency**, as one of the cells firing B, is **increased**. (1949)

Neuron A and neuron B are active: $w_{AB}$ increases

Physiologically: « synaptic plasticity »

PSP size is increased
(or: transmission probability is increased)
Synaptic plasticity at spike level

Presynaptic action potential

(f) Synaptic plasticity at spike level

pre → post: potentiation
post → pre: depression

(STDP = Spike-Timing-Dependent Plasticity)

- causal rule
- favors synchronous inputs

Dan & Poo (2006)