Introduction to Computational Neuroscience

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Resumen

This document introduces fundamental concepts in computational neuroscience, with a particular focus on the role of modeling in understanding brain function. The purpose is to get enough deep to create new insights by the hand of investigation.

Índice

1. Generalities

1. Santiago Ramón y Cajal established that the brain is composed of discrete units—neurons—rather than forming a continuous network.

2. Computational modeling in neuroscience has evolved significantly. Early models were rooted in nonlinear dynamical systems (e.g., Hodgkin-Huxley models), followed by the development of stochastic models, neural coding frameworks, unsupervised learning paradigms, and logical or symbolic systems. More recent approaches include connectionist models such as autoencoders, pattern association networks, and manifold-based representations. Each class of model captures different aspects of neural computation or cognition.

3. **Neuron**: A neuron is an electrically excitable cell that processes and transmits information through signals. It has three main components:

   - **Dendrites**: Receive input signals from other neurons.
   - **Soma (cell body)**: Acts as a nonlinear integrator. If the combined input exceeds a certain threshold, the neuron emits a spike.
   - **Axon**: Conducts the output spike to other neurons, often over long distances.

4. **Synaptic Communication**:

   - Synapses are the junctions where neurons communicate. The presynaptic terminal releases neurotransmitters that bind to receptors on the postsynaptic neuron.
   - **Action Potentials**: If the membrane potential crosses a threshold, an all-or-none electrical event known as an action potential is generated. Its shape and amplitude remain constant as it travels down the axon.
   - A sequence of action potentials generated by a neuron over time is known as a **spike train**.

2. Model 1: Nernst Potential

The Nernst potential arises from the interplay between diffusion and electrostatics at equilibrium. When ions diffuse from a region of high concentration to low concentration—e.g., K^+ moving out of a neuron—the net movement carries positive charge away, leaving behind excess negative charge. This charge imbalance generates an electric potential that increasingly opposes the diffusion. At equilibrium, the chemical and electrical driving forces exactly cancel. The resulting voltage is the Nernst potential, so $n(x) = Np(x)$

$$p(x) \propto \exp(-B \cdot u(x)) \quad \Rightarrow \quad \frac{n_1(x)}{n_2(x)} = \exp(-B \cdot \Delta u)$$

Solving for the potential difference:

$$\Delta u = \frac{1}{B} \log\left(\frac{n_2(x)}{n_1(x)}\right)$$

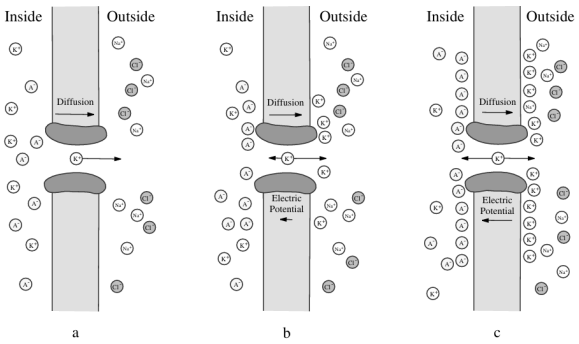

Figura 1: Visualization of membrane with two densities and equilibrium of two forces.

In biophysical terms, this yields the Nernst equation $E = k_B T \log\left(\frac{[\text{ion}]_{\text{out}}}{[\text{ion}]_{\text{in}}}\right)$ which is valid for a single ion type.

Insight: We model the system under thermal equilibrium, which balances the diffusive and electrical forces.

3. Model 2: Hodgkin-Huxley Model

The Hodgkin Huxley (H-H) model improves the real neurons understanding. First, it describes the membrane potential as an electrical circuit consisting of a capacitor in parallel, where ion channels act as voltage-dependent gates. This arises non-equilibrium behavior.

Main Assumption: The action potential is the macroscopic answer to the sum of all the individual currents done by the ion channels. These channels arises are modeled as gates with RC dynamics.

1. **Capacitor - Membrane:** The neuron's membrane is modeled as a capacitor that stores energy in the form of an electric field:

$$C = \frac{Q}{U}$$

The capacitor current is nonzero only when the membrane potential changes over time:

$$I_C(t) = C\frac{du(t)}{dt}$$

2. **Current Conservation (Kirchhoff's Law):** The total membrane current $I(t)$ splits and sums:

$$I(t) = I_C(t) + \sum_k I_k(t)$$

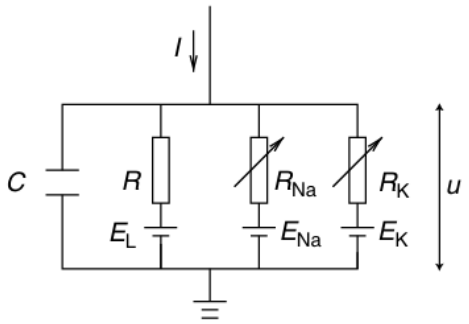

Figura 2: Electrical circuit representation of the Hodgkin-Huxley model showing membrane capacitance and gates dynamics.

Ionic Currents: Each ion species contributes to the total current based on its own gate:

$$\sum_k I_k = g_L(u - E_L) + g_{Na} \cdot m^3 h \cdot (u - E_{Na}) + g_K \cdot n^4 \cdot (u - E_K)$$

Gate Dynamics: Each gating variable $x \in \{m, h, n\}$ evolves over time as a first-order differential equation. Gating is sensitive to the membrane potential, so gates are classified as active or inactive. This mechanism is modeled by the probability that an active gate is open m (or n for K^+ channels) and the probability that an inactive gate is open is h. Then, a proportion of open & close channels is given by $p = m^a h^b$

$$\dot{x} = \frac{1}{\tau_x(u)}(x - x_0(u))$$

There are two figures important to understand the H-H model:

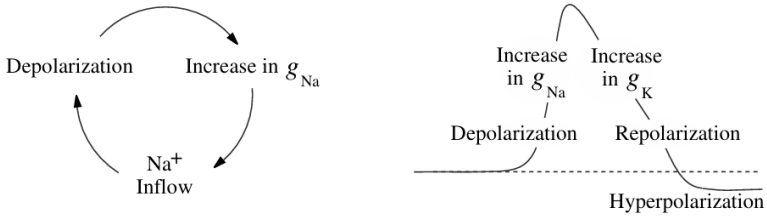

Figure 2.16: Positive and negative feedback loops resulting in excited (regenerative) behavior in neurons.

Figura 3: Figure taken from Izhikevich's book

The Hodgkin-Huxley model exhibits two key feedback loops: (1) A fast positive feedback loop where Na^+ channels open rapidly causing depolarization, and (2) A slower negative feedback loop where K^+ channels gradually open to repolarize the membrane. The temporal difference between these loops creates the characteristic spike.