A short summary of biophysics

Peter Latham, January 15, 2026

1 Introduction

The brain consists of a large number of neurons – about 100 billion in humans – that mainly communicate via spikes. (The "mainly" qualifier is because neurons can directly communicate their voltage via gap junctions. Here we ignore gap junctions, as they don't play much of a role in mature mammals.) The communication is kind of complicated, as neurons consist of multiple parts: a soma (cell body), as well as dendrites, axons and synapses (Fig. 1).

The goal here is to understand how neurons communicate via spikes. We'll do this in stages: we'll first consider the soma, then dendrites and axons, and, finally, the synapses. We'll start, though, with a brief introduction to biophysics in general.

2 Biophysics

In biophysics the main thing we're interested in is the membrane potential, $V(t)$, which is the voltage difference between the inside and outside of a neuron. As shown in Fig. 1, neurons have three main parts: soma, dendrites, and axons, and the membrane potential

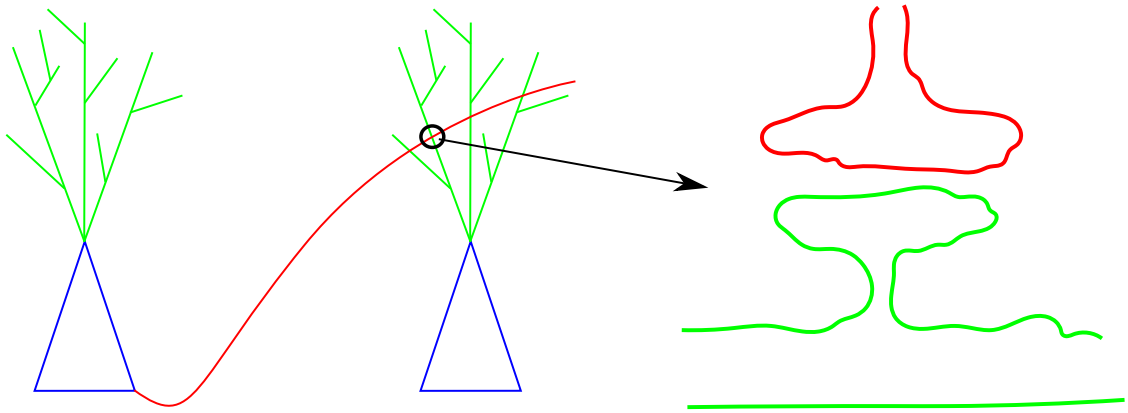

Figure 1: Coupled neurons. The two objects on the left are neurons (which don't really all look alike; I was just too lazy to make them different). The neurons have three main parts: soma (blue), dendrites (green) and axons (red). The dendrites are much, much bigger than shown (50-100 times the size of the soma, which is on the order of 10-20 microns), and so are the axons, which branch (because they connect to about 1,000 other neurons), and can travel long distances (up to a meter). Neurons communicate via synapses, which connect axons to dendrites (usually; axons can also connect directly to the soma). A typical synapse is shown on the right: the presynaptic terminal (red) connects to a spine (green), which is a small structure that sticks out of the dendrites. This being biology, a spine is not always present; the conventional wisdom is that excitatory neurons connect to spines and inhibitory neurons connect directly to dendrites or to the soma. But, this being biology, that conventional wisdom is often violated.

isn't the same everywhere. Ultimately we have to derive separate equations for the membrane potential on the different parts of the neurons. But in this section we'll just think of $V(t)$ as the voltage difference between the inside and outside of a membrane. Given that, we'll derive a general equation for its time derivative.

Essentially, we use two equations. The first is $Q = CV$ where V is voltage and Q is the net charge on the inside of the membrane. Taking a time derivative (and noting that $dQ/dt =$ current) gives us

$$C\,\frac{dV}{dt} = -I\,. \tag{1}$$

Here I is, by convention, the outward current – the current flowing from inside to outside. The sign should make sense: if I is positive, current flows out and the voltage goes down; if I is negative, current flows in and the voltage goes up.

Equation (1) is absolutely fundamental. OK, sort of absolutely fundamental: it ignores magnetic fields, and assumes that the voltage is the same everywhere inside the membrane, which isn't always the case (in particular, it's not the case for dendrites and axons, but we have ways of dealing with that). For now, though, we'll assume that Eq. (1) holds.

So what's the current? If charge were carried by electrons, the current would be computed from $V = IR$ where R is resistance, and if R were constant, we would have a classic RC circuit,

$$C\,\frac{dV}{dt} = -V/R \tag{2}$$

which has the solution $V(t) = V(0)e^{-t/RC}$. However, neurons are not nearly this simple, so the equations are a bit more complicated. For several reasons.

First, charge is not carried by electrons, it's carried by ions. And, because neurons have ion pumps, the ions have different concentrations on the inside and outside of the cell. In particular, the concentrations of sodium and chloride (abbreviated Na and Cl) are high on the outside of the cell, while the concentration of potassium (abbreviated K) is high on the inside. (If you ever become a neuroscientist you should memorize that; but if not I wouldn't bother; it's one of those facts you can always look up.) What's important is the effect of an ion imbalance: even when the membrane potential, V, is zero, an ion imbalance will cause a current to flow (for example, an inward Na current, because there's a lot more sodium on the outside than inside). That rules out $V = IR$, and it means we need something more complicated. The thing we use is

$$I_x = g_x(V - \mathcal{E}_x) \tag{3}$$

where x refers to the ion, so it could be Na, Cl or K (other common ions used in the brain are Ca, for calcium, and Mg, for magnesium, but we won't worry about either, at least for now). The parameter g_x is the conductance of a channel that allows ion x to pass through (it's the inverse of the resistance, R_x: $g_x = 1/R_x$), and $\mathcal{E}_x$ is the reversal potential. The reversal potential needs to be included because of the concentration imbalance. For example, the reversal potential for Na is about 20 mV, which means the voltage on the inside of the cell has to be about 20 mV higher than the voltage on the outside to keep the sodium current from flowing.

Notice that the conductance depends on the ion. That's because channels, which are holes in the cell that ions can flow through, can be ion specific. For example, a channel

may allow only Na, or only Cl, to flow through it. But because this is biology, which is inherently complicated, some channels aren't ion specific, and they let any ion flow through them (although often with different conductances). And, of course, there's the in-between case: channels that let a few ions through, like Na and K but nothing else. But that doesn't really matter; g_x measures the total conductance of ion x taken over the whole membrane.

A nice thing about conductances is that they add, which should be kind of intuitive: adding more channels gives you more current (remember parallel circuits?). Thus, the total current is

$$I = \sum_x g_x(V - \mathcal{E}_x)\,. \tag{4}$$

It is useful to combine Eqs. (1) and (4), which gives us

$$C\,\frac{dV}{dt} = -\sum_x g_x(V - \mathcal{E}_x)\,. \tag{5}$$

This is the starting point for pretty much all of biophysics, and is the main equation we'll use.

All the interesting behavior that we see in the brain is due to the behavior of the conductances, g_x. They can – and do – depend on just about anything. In the simplest case, they're constant, which gives us a passive neuron. Passive neurons are simple, but not very useful as computing devices. Consequently, evolution invented voltage-dependent conductances (to generate spikes) and concentration-dependent conductances (to allow communication across synapses). We'll consider those next; after that, we'll examine how all this changes for extended objects like dendrites and axons.

3 The Hodgkin-Huxley model

Here we consider active channels. Channels themselves are very small, consequently, they're stochastic. Typically we model them as being either open or closed. Importantly, the probability of opening or closing depends on voltage, which we write

$$\begin{aligned} \alpha_x(V) &= \text{probability per unit time that channel } x \text{ goes from closed to open} \\ \beta_x(V) &= \text{probability per unit time that channel } x \text{ goes from open to closed.} \end{aligned} \tag{6}$$

where x refers to channel type. This is a Markov model, so if x is the probability that the channel is open, then it is is (relatively) easy to show that x obeys the equation

$$\tau_x(V)\,\frac{dx}{dt} = x_\infty(V) - x \tag{7}$$

where

$$\tau_x(V) = \frac{1}{\alpha_x(V) + \beta_x(V)} \tag{8a}$$

$$x_\infty(V) = \frac{\alpha_x(V)}{\alpha_x(V) + \beta_x(V)}\,. \tag{8b}$$

OK, this isn't quite the whole story; a channel typically consists of more than one x, and they all have to be open for current to flow. For the Hodgin-Huxley model, there are two kinds of active channels: sodium and potassium. The probability that the active sodium channel is open is m^3h and the probability that the active potassium channel is open is n^4, where m, h and n obey Eq. (7), but with x replaced with the appropriate variable. For this model, the voltage evolves according to

$$C\,\frac{dV}{dt} = -g_L(V-\mathcal{E}_L) - g_{Na}m^3h(V-\mathcal{E}_{Na}) - g_K n^4(V-\mathcal{E}_K) \tag{9}$$

where g_L is the leak (meaning passive) conductance and m, h and n are the probability of channels being open. We typically divide by g_L to give us the equation

$$\tau\,\frac{dV}{dt} = -(V-\mathcal{E}_L) - \rho_{Na}m^3h(V-\mathcal{E}_{Na}) - \rho_K n^4(V-\mathcal{E}_K) \tag{10}$$

where

$$\tau = \frac{C}{g_L} \approx 10\text{ms} \tag{11a}$$

$$\rho_{Na} = \frac{g_{Na}}{g_L} \approx 400 \tag{11b}$$

$$\rho_K = \frac{g_K}{g_L} \approx 240\,. \tag{11c}$$

To understand the behavior of these equations, recall first of all that $\mathcal{E}_{Na} \approx +20$ mV and $\mathcal{E}_K \approx -80$ mV. Second, we need to know how $m_\infty(V)$, $h_\infty(V)$, and $n_\infty(V)$, The first and last, m and n, are increasing functions of V, while h is a decreasing function of V. Thus, when the voltage increases past a threshold, the m-channels open, which raises the voltage even more, which causes them to open even more. This leads to a rapid increase in voltage. However, with a slight delay, the h-channel closes, pushing the voltage toward the leak potential, and at the same time the n-channel opens, pushing the voltage down even more, toward -80 Mv. For this to work, the time constant of the m-channel must be much smaller than for the other two, which is it: $\tau_m < 1$ ms, while τ_h and τ_n are both on the order of 1-2 ms.

We would like to make this quantitative picture more quantitative, but that's hard – the Hodgkin-Huxley equations is a nonlinear differential equation with four variables, and as far as anybody knows there's no analytic solution. So we do what any self-respecting theorist does: we change the problem. Because the m-channel is fast, we replace m in Eq. (10) with $m_\infty(V)$. And because the potassium channel just causes the voltage to undershoot, we get rid of it altogether. This gives us the two-variable system

$$\tau\,\frac{dV}{dt} = -(V-\mathcal{E}_L) - \rho_{Na}m_\infty(V)^3h(V-\mathcal{E}_{Na}) + V_0\,. \tag{12a}$$

$$\tau_h(V)\,\frac{dh}{dt} = h_\infty(V) - h\,. \tag{12b}$$

Note that we have added an external voltage (which is really an external current times some conductance), because we may want to drive the neuron). To analyze these equations we draw the nullclines: curves along which either $dV/dt = 0$ (the V-nullcline) or $dh/dt = 0$ (the h-nullcline). These are shown in Fig. 2. For the reversal potentials I used $\mathcal{E}_L = -70$ mV

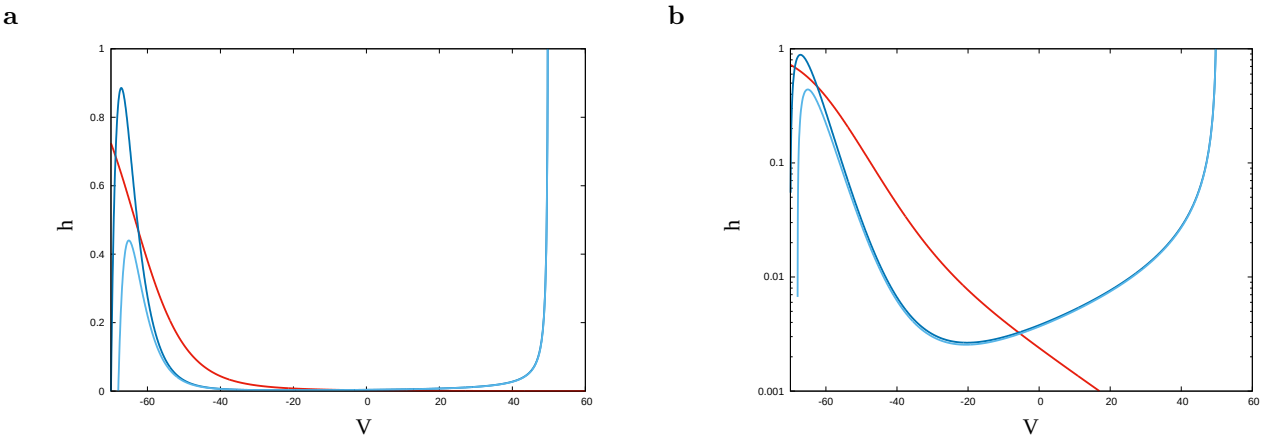

Figure 2: Nullclines for the approximate model given in Eq. (12). Red: h-nullcline. Dark blue (higher curve): V-nullcline with $V_0 = 0$. Light blue (lower curve): V-nullcline with $V_0 = -2$.

and $\mathcal{E}_{Na} = 50$ mV, and for the ratio of peak sodium to leak conductance I used $\rho_{Na} = 400$. Finally, for $h_\infty(V)$ and $m_\infty(V)$ I used,

$$h_\infty(V) = \frac{0.06e^{-0.05(V+65)}}{0.06e^{-0.05(V+65)} + 1/(1+e^{-0.1(V+35)})} \tag{13a}$$

$$m_\infty(V) = \frac{0.1(V+40)(1-e^{-0.1(V+40)})}{0.1(V+40)(1-e^{-0.1(V+40)}) + 4e^{-0.0556(V+65)}}\,, \tag{13b}$$

taken from Dayan and Abbott (I think).

4 Dendrites and axons

For dendrites and axons, we can no longer assume that $V(t)$ is constant everywhere on the inside of the membrane. Which makes things a bit more complicated; see biophysics.pdf.

5 Synapses

The conductances can also depend on the concentration of a neurotransmitter in the synaptic cleft (the area between the red presynaptic terminal and the green spine in Fig. 1). In that case the channels are on the spine, and we have

$$I_s = g_s(V - \mathcal{E}_s) \tag{14a}$$

$$g_s = \overline{g}_s s \tag{14b}$$

where s (which stands for "synaptic"), is between 0 and 1. It obeys the equation

$$\frac{ds}{dt} = c(1-s) - \beta s\,. \tag{15}$$

Here c is the neurotransmitter concentration in the synaptic cleft (which is generally near zero, but goes up when a spike arrives at the presynaptic terminal), and β tells us how fast the synaptic conductance decays when the concentration drops back to near zero.

There is a bit of a subtlety associated with Eq. (14). The voltage should really refer to the voltage in the spine, not at the soma. However, to model networks, we often pretend that it's the voltage at the soma; basically, we pretend that dendrites don't exist (this is the point neuron approximation). In that case, the current, $I_s = s\overline{g}_s(V - \mathcal{E}_s)$, is the current that flows into the soma.

6 A model of networks of neurons, on fast timescales

With this approximation, we can combine Eq. (14) with the Hodgkin-Huxley equation, Eq. (10), to give us a set of equations describing a network of neurons. Using the subscript i to label neurons, and letting $\mathcal{E}_s \to \mathcal{E}_j$, $s(t) \to s_j(t)$ and $\overline{g}_s \to W_{ij}$ (and summing over j), we have a set of equations that looks like

$$C\,\frac{dV_i}{dt} = -g_L(V_i - \mathcal{E}_L) - g_{Na}m_i^3h_i(V_i - \mathcal{E}_{Na}) - g_K n_i^4(V_i - \mathcal{E}_K) - \sum_j W_{ij}(V_i - \mathcal{E}_j)s_j(t)\,. \tag{16}$$

We say "on fast timescales" because it ignores the fact that the weights change, and weight changes depend on activity.

There are several things to note about this equation. First, the reversal potential, $\mathcal{E}_j$, depends on the presynaptic neuron – something that evolution gave us. Second, we should be aware that the weights, W_{ij}, are very sparse: each neuron makes only about 1,000 connections, and a brain the size of, say, a human, contains 100 billion neurons, so most of the weights are zero. Third the very last term, $s_j(t)$, determines the shape of the PSP (postsynaptic potential) associated with neuron j. It obeys something like Eq. (15), but we often assume it has a stereotyped shape, and write

$$s_j(t) = \sum_k f_j(t - t_j^k) \tag{17}$$

where t_j^k is the time of the k^{th} spike on neuron j and $f_j(t)$ is a function that rises rapidly and decays slightly more slowly than it rises. It is sometimes modeled as a double exponential,

$$f_j(t) = \frac{e^{-t/\tau_j^s} - e^{-t/\tau_j^f}}{\tau_j^s - \tau_j^f}\,\Theta(t)\,. \tag{18}$$

Here τ_j^s and τ_s^f are fast and slow time constants; for fast synapses, τ_f^f is in the range 1-5 ms and τ_j^s is in the range 3-10 ms (and they can be many 10s of ms for slow synapses), and $\Theta(t)$ is the Heaviside step function: $\Theta(t) = 1$ if $t > 0$ and 0 otherwise. However, we could swap in just about any function and that wouldn't have much effect on the network dynamics.

7 Summary

As you can see, things are relatively complicated. But just keep in mind two things:

1. All of biophysics comes from Eq. (5).

2. Conductances, g_x, are the interesting part of Eq. (5). So far we have seen that they can depend on voltage and the concentration of a neurotransmitter. (They can, of course, depend on both – this being biology, evolution has thought of just about anything we can imagine, within reason.) But that's not all. For instance, for very early sensory processing, conductances can depend on the outside world: photoreceptors in the retina have conductances that respond to light; hair cells in the ear have conductances that respond to mechanical vibration; the olfactory receptor neurons in the nose have conductances that respond to chemicals, and so on. So, if we want to know the fundamental equations describing the brain, we need to focus on conductances!