The leaky integrate and fire model

This note is about the dynamics of a single neuron, it will cover one of the simplest such models: the leaky integrate and fire model.
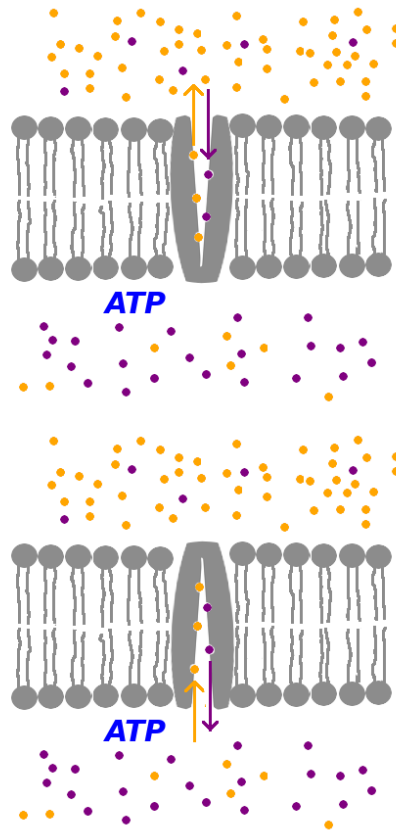

Figure 1: The ion pump pumps three sodium ions out for each it two potassiums it pumps in.

Electrical properties of a neuron

The potential inside a neuron is lower than the potential on the outside; this difference is created by ion pumps, small molecular machines that use energy to pump ions across the membrane seperating the inside and outside of the cell. One typical ion pump is Na+/K+-ATPase (Sodium-potassium adenosine triphosphatase); this uses energy in the form of ATP, the energy carrying molecule in the body, and through each cycle it moves three sodium ions out of the cell and two potassium ions into the cell, see Fig. 1 for a cartoon. Since

Figure 2: A cartoon to help you remember that there is more sodium outside the cell and more potassium inside.

both sodium and potassium ions have a charge of plus one, this leads to a net loss of one atomic charge to the inside of the cell lowering its potential. It also creates an excess of sodium outside the cell and an excess of potassium inside it. We will return to these chemical imbalances later. The potential difference across the membrane is called the **membrane potential**. At rest a typical value of the membrane potential is $E_L = -70$mV. It is useful to remember that the excessive sodium is outside the cell and potassium inside; I think of islands which are surrounded by salty water, as in Fig. 2.

Spikes

So the summary version of what happens in neuons is that **synapses** cause a small increase or decrease in the voltage; **excitatory synapses** cause an increase, **inhibitory synapses** a decrease. This drives the internal voltage dynamics of the cell, these dynamics are what we will learn about here. If the voltage exceeds a threshold, say $V_T = -55$ mV there is a nonlinear cascade which

produces a **spike** or **action potential**, a spike in voltage 1-2 ms wide which rises above 0 mV before, in the usual description, falling to a reset value of $V_R = -65$ mV, the cell then remains unable to produce another spike for a **refractory period** which may last about 5 ms. We will examine how spikes are formed later, this involves the nonlinear dynamics of ion channels in the membrane; first though we will consider the integrate and fire model which ignores the details of how spikes are produced and simplifies the voltage dynamics.

The bucket-like equation for neurons

We will now try to extend the bucket-like equation we looked at before so that it applies to neurons. First off we replace h, the height of the water, by V the voltage in the cell and C will be replaced by C_m, the capacitance of the membrane, the amount of electrical charge that can be stored at the membrane is $C_m V$. The amount of electrical charge is the analogue of the volume of water. Thus, voltage is like height, charge is like the amount of water.

The leak is a bit more complicated, because of the chemical gradients, that is the effects of the differing levels of ions inside and outside the cell along and their propensity to diffuse, the voltage at which there is no leaking of charge is not zero, it is $E_L = -70$mV, roughly. This is an important aspect of how neurons behave, and one we will encounter again looking at the Hodgkin-Huxley equation: you might at first expect that if the voltage inside the cell was, say, -60 mV then even if there was a high conductivity for potassium at the membrane, the potassium ions would stay in the cell: they are positive ions after all and so a negative voltage means the electrical force is attracting them to the inside of the cell. However, this isn't quite what happens, there is a high concentration of potassium inside the cell and because of the random motion of particles associated with temperature, these have a tendency to diffuse, that is to increase the entropy of the situation by spreading out. It takes a force to counteract this; see Fig. reffig:children for a whimsical illustration of this. This is the reversal potential, E_L, the voltage required for zero current even if there is some conductivity. It turns out that the normal Ohm's law applies around the reversal potential so that the current out of the cell is proportional to $V - E_L$.

G is now G_m, a conductance, measuring the porousness of the membrane to the flow of ions, in other words, it gives the constant of proportionality for the leak current: the leak current out of the cell is $G_m(V - E_L)$. We actually divide across by the conductance, and write $R_m = 1/G_m$, the resistance. Finally, we write $\tau_m = C_m/G_m$ to get

$$\tau_m \frac{dV}{dt} = E_L - V + R_m I \tag{1}$$

I might end up being synaptic input, but traditionally we write the equation to match the *in vivo* experiment where I is an injected current from an electrode, so we write I_e, 'e' for electrode. τ_m is a time constant, using the notation of dimensional analysis we have $[\tau_m] = T$. To check this note that the units of capacitance are charge per voltage: $[C_m] = QV^{-1}$, the units of resistance is
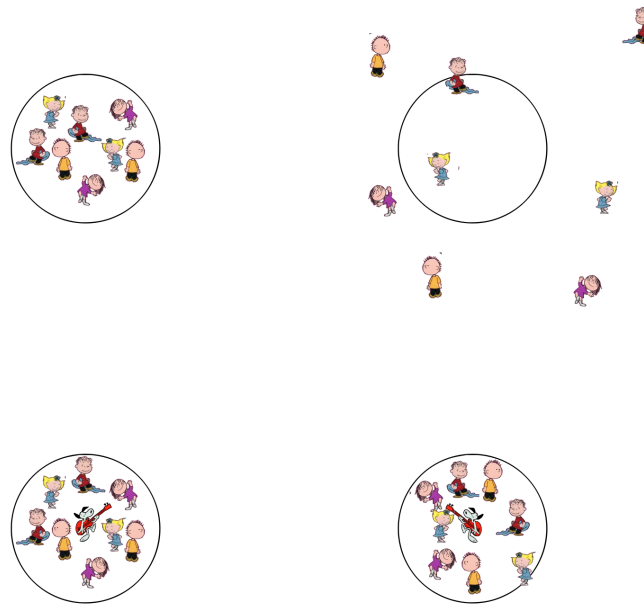
Figure 3: In the first panel the children are in the circle, however random movement quickly disperses them across the playground, in the second panel; however, in the final two panels the force exerted on the children by the musical dog counteracts the diffusion pressure; in the neuron, the ions are children and the role of snoppy is played by voltage.

voltage per current $[R_m] = VI^{-1}$ and current is charge per time, $[I] = QT^{-1}$ so $[C_m R_m] = T$, time.

The equation above leaves out the possibility that there are other non-linear changes in the currents through the membrane as V changes. This is a problem since there are other non-linear changes in the currents through the membrane as V changes. The equation above leaves these out, in fact, the nonlinear effects are strongest for values of V near where a spike is produced, so one approach is to use the linear equation unless V reaches a threshold value and then add a spike 'by hand'. This has the effect of changing the voltage to a reset value, this mimics what happens in the neuron, or in the Hodgkin Huxley model which we will look at next and which includes the full non-linear dynamics which makes the spike. Anyway, in summary

- V satisfies

$$\tau_m \frac{dV}{dt} = E_L - V + R_m I_e \tag{2}$$

- If $V \geq V_T$ a spike is recorded and the voltage is set to a reset value V_R.

The reset value, the voltage after the spike is often set equal to the leak potential. This is the **leaky integrate and fire model**, a surprisingly old model first introduced in [1]. It lacks lots of the details important in the dynamics of neurons, but is useful and often used for modeling the behavior of large neuronal networks or for exploring ideas about neuronal computation in a relatively straight-forward setting.

This model is easy to solve; if I_e is constant we have already solved it above up to messing around with constants:

$$V(t) = E_L + R_m I_e + [V(0) - E_L - R_m I_e]e^{-t/\tau_m} \tag{3}$$

If I_e is not constant it may still be possible to solve the equation, but in any case the equation can be solved numerically on a computer. An example in given in Fig. 4.

One thing to notice is that there are no spikes for low values of the current. Looking at the equation

$$\tau_m \frac{dV}{dt} = E_L - V + R_m I_e \tag{4}$$

so the equilibrium value for constant I_e, the value where V stops changing, is

$$\bar{V} = E_L + R_m I_e \tag{5}$$

Now if this value $\bar{V} > V_T$ then as the neuron voltage increased towards its equilibrium value, $\bar{V}$, it would reach the threshold, V_T, and spike. Hence, if $\bar{V} > V_T$ the neuron will spike repeatedly. However if $\bar{V} < V_T$ then the neuron will not spike for that input because it will never reach threshold. We won't do it here[1], but, in fact, since we can solve the equations for constant I_e we can work out the $f - I$ curve, the relationship between the firing rate and the input current. It is plotted in Fig. 5.

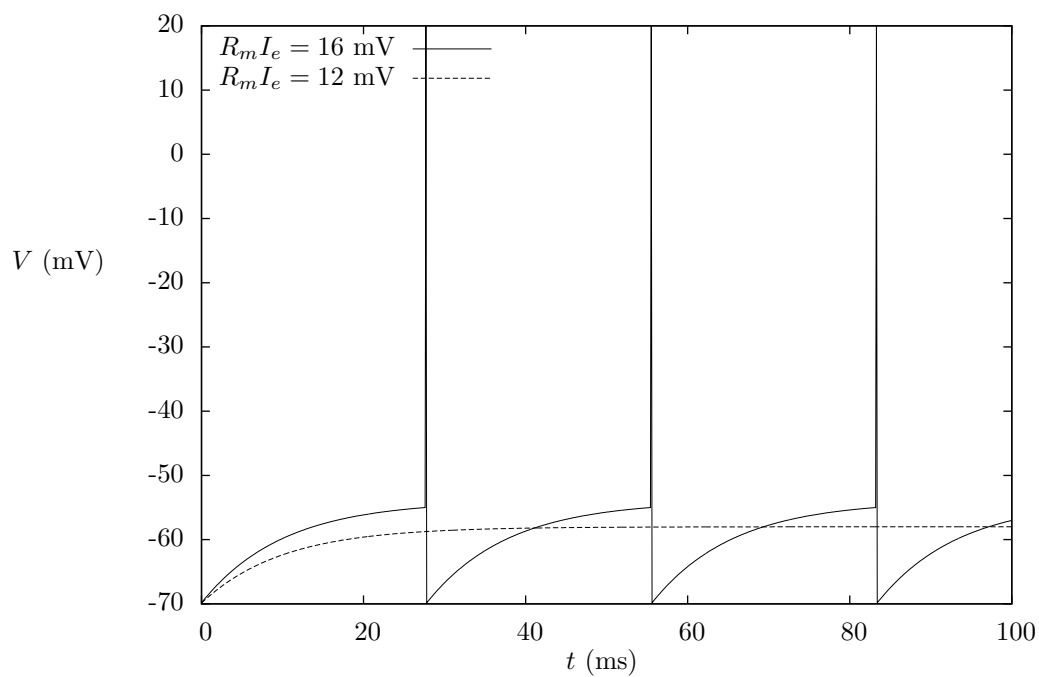[1]This calculation is described in `note_on_the_f-I_curve.pdf`

Figure 4: An integrate and fire neuron with different inputs. For $R_mI = 12$mV the voltage relaxes towards the equilibrium value $V = E_L + R_mI_e = -58$ mV. It never reaches the threshold value of $V_T = -55$mV. For $R_mI = 16$ mV the voltage reaches threshold and so there is a spike; the spike is added by hand, in this case by setting V to 20 mV for one time step. The voltage is then reset. Here $\tau_m = 10$ ms.
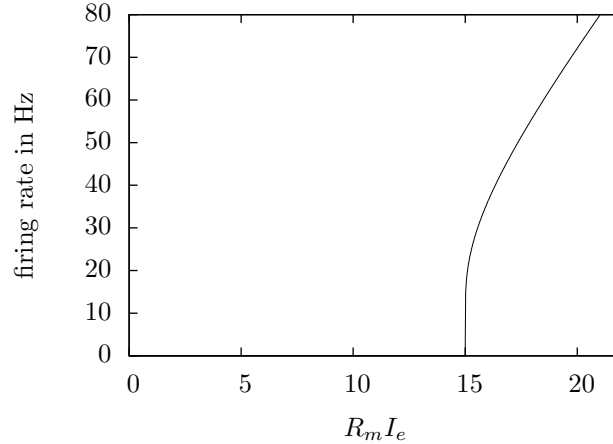
Figure 5: The firing rate, that is spikes per second, for the integrate and fire neuron with different constant inputs with $\tau_m = 10$ ms, $V_T = -55$ mV and both the leak and reset given by -70 mV. Notice how there is no firing until a threshold is reached and after that the firing increases very quickly.

Beyond the integrate and fire equations

The leaky integrate and fire model is very useful; from a practical point-of-view it can be used in modelling, either for modelling large networks of spiking neurons for neuroscience, for example, when examinging the statistics of spiking or synapse strengths under various regimes, or when investigating spiking neural networks in machine learning. It also clarifies the role of the membrane time constant, measuring this is a useful and important step in the experimental exploration of a neuron's properties. From a theoretical point-of-view; it gives important insight into what a neuron does.

The model has limitations; it does not incorporate the dynamics of the active ion channels. Ultimately, this means it does not model the production of the spike; in the integrate and fire model the spike is included using the threshold condition, when the voltage exceeds the threshold a spike is said to occur, but there is no indication of how that happens. The **Hodgkin-Huxley equation** includes some active channels and will demonstate the dynamics that can produce a spike. However, it is possible to consider other, smaller, modifications of the leaky integrate and fire model.

One issue with the leaky integrate and fire model is that it does not model behaviour near the threshold, even before the neuron starts to spike, when its voltage approaches the threshold where a spike becomes inevitable, some of the active channels do start to open altering the dynamics. Since this only applies

very close to threshold it might not seem a significant drawback of the model; however, in some descriptions of neuronal networks some homeostatic principles of balanced excitation and inhibition mean that neurons are often near threshold making the dynamics near threshold important. There are extensions to the leaking integrate and fire model that seek to address this, usually by adding extra non-linear terms to the differential equation; one successful example is the adaptive exponential leaking integrate and fire model which adds an exponential term.

Another more straight-forward difference between the integrate-and-fire neuron and real neurons is refractoriness; most neurons are unable to fire directly after a spike, there is a rich discription of this **refactory period**, many neurons have a short period where they are unable to spike and another, longer, period where spike is less likely to occur. The refractory period is often taken to be around 10 ms, but this does vary a lot between neurons, Purkinje cells for example seem to have little or no refractory period. This refactoriness is related to the active channels, for example, during spiking potassium channels open, as we will see this is part of the dynamics which produces the down-swing of the spike and, while they are open, these potassium channels will push the voltage towards the potassium reversal potential. This affect can easily be added to the integrate-and-fire model, for example, by adding to the spiking rule to stipulate that the voltage remains fixed that the reset value for some specified time after a spike.

One final example is **spike rate adaptation**, in the integrate-and-fire model a neuron with a constant input will spike at a constant rate: in fact, the model resets completely with every spike so the neuron never shows any effect of its previous behaviour. Real neurons often show more complex behaviour, often, for example, a neuron will spike slower as it continues to spike, or its second spike will happen very easily, something called pair-pulse excitation but will then slow down. There are even neurons, such as the mossy cells of the hilar region of dentate gyrus, which spike faster and faster the longer they are excited. This can be easily incorporated into the integrate-and-fire neuron using an extra current:

$$\tau_m \frac{dV}{dt} = E_L - V + R_m I_e - W \tag{6}$$

where W is itself a current which decays to zero:

$$\tau_w \frac{dW}{dt} = -W \tag{7}$$

and has its own rule for what happens when there is a spike:

$$W \to \delta W \tag{8}$$

Thus, if δW is positive, W will increase if there is a spike, this gives a negative contribution to dV/dt, slowing down spiking. W then decays to zero with a timescale τ_w. These dynamics would model spike rate adaptation and the W current itself can be thought of as the effect of a slow potassium current. In fact, spike rate adaptation is not explained by the Hodgkin-Huxley, but a slow potassium current could be added there as well.

github.com/LaurenceA/COMS30017_2022/

References

[1] Lapicque, L. (1907). Recherches quantitatives sur l'excitation électrique des nerfs traitée comme une polarisation. J. Physiol. Pathol. Gen, 9:620–635.