PERSPECTIVE
Models for Spiking Neurons: Integrate-and-Fire Units and Relaxation Oscillators

Kevin Crisp
Biology Department, St. Olaf College, Northfield, MN 55057.

Relaxation oscillators are nonlinear electronic circuits that produce a repetitive non-sinusoidal waveform when sufficient voltage is applied. In this fashion, they are reminiscent of integrate-and-fire neuron models, except that they also include components with hysteresis, and thus require no threshold rule to determine when an impulse has occurred or to return the voltage to its reset value. Here, I discuss the pros and cons of teaching elementary neurophysiology using first-order linear integrate-and-fire neurons versus relaxation oscillator circuits. I suggest that the shortcomings of both types of models are useful in order to foster a critical understanding of the neurophysiology underlying the firing dynamics of biological neurons.

Key words: integrate-and-fire model, relaxation oscillator, python programming, transistor, teaching lab

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The linear integrate-and-fire neuron is a simple spiking model that produces many of the features characteristic of real neurons (Gerstner & Kistler, 2002). For this reason, they make very practical and useful models in teaching the basic principles of neurophysiology to undergraduate students. However, as they are described simply by a first-order differential equation and have no intrinsic fast dynamics (i.e., action potential) or after-hyperpolarization voltage, they really only describe the slow, subthreshold membrane properties prior to an action potential. On the other hand, relaxation oscillators are electrical circuits with far greater complexity and can be used as spiking neuron models that include both the supra-threshold fast depolarization of the action potential and the voltage reset of the after-hyperpolarization. In this paper, I will discuss the potential strengths and weaknesses of both models as teaching tools for undergraduate neuroscience students. I will show that there are teachable moments associated with the shortcomings of each model and will suggest that both are therefore useful comparisons when teaching the physiological basis of neuronal firing dynamics.

The Linear Integrate-and-Fire (IF) Neuron

The simplest mathematical model of the nerve cell is the linear IF neuron. This model is derived from an equation describing the change in voltage as a function of time observed when current is applied to a simple electrical circuit consisting of a resistor and a capacitor in parallel. The resistor represents the membrane resistance, which for a cell with a diameter of approximately 20 µm maybe be in the ballpark of 256 MΩ. This resistance is due to a leak conductance of 0.3 mS*cm^2. The capacitance may be 80 pF, or 1 µF/ cm^2. In parallel, these two components thus represent the ion channels and phospholipid bilayer of the membrane, respectively (Figure 1). A battery is often added in series with the resistor in order to give the simple model a biologically-plausible resting membrane potential when no current is applied. Unless the voltage of this battery exceeds the voltage threshold for an impulse, this cell will not spike without injected current. Thus a current source (I_inj) is also included in the schematic to represent the injection of current by an intracellular recording electrode. Vm indicates

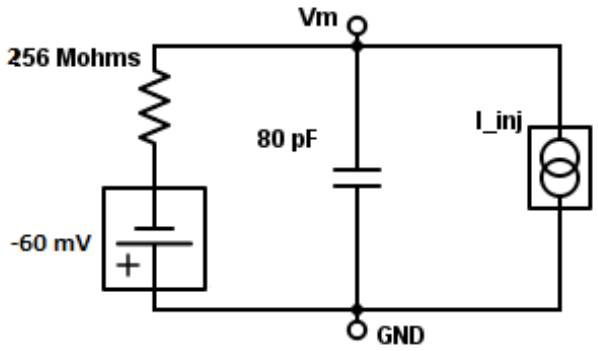

Figure 1. A schematic diagram representing the electrical circuit from which the IF neuron equation is derived.

the point at which the membrane potential is recorded with respect to ground (GND).

Depolarizing current imposed upon this circuit (by the current source) will decrease the magnitude of the potential difference between Vm and GND. When the voltage (measured at point Vm) reaches threshold (typically a

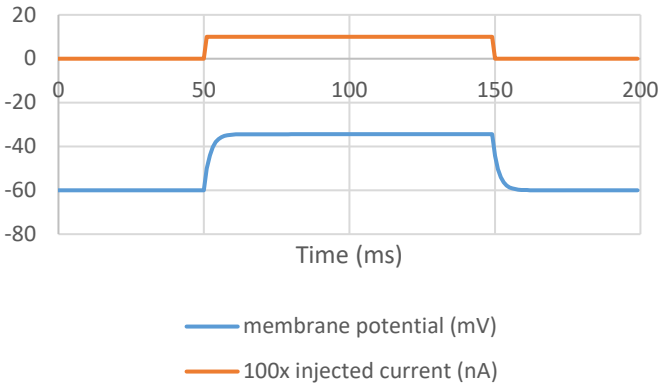

Figure 2. A graph showing the change in voltage over time (blue) of the circuit depicted in Figure 1 when current (0.1 nA, orange) is applied. X-axis is in ms, y-axis is in mV for membrane potential, 10^{-9} A for injected current. (See Appendix A for Python code.)

voltage some 15 mV depolarized from rest), this indicates that an action potential has occurred. The voltage of the circuit is then reset to an arbitrary voltage, typically near the Nernst equilibrium potential for potassium. Note, however, that nothing in the circuit shown in Figure 1 provides for the threshold device or accounts for hyperpolarized reset voltage that occurs after an impulse. Rather, the circuit really only describes the rate at which the cell, under the influence of imposed current, depolarizes toward threshold, or the rate at which the cell hyperpolarizes back to rest from a subthreshold voltage owing to a change in the amplitude of the imposed current.

Students will immediately notice the effects of the membrane time constant on the charging curve in Figure 2. The equation that governs this system (Stein, 1967) is

Equation 1. $$\tau \frac{\partial V}{dt} = I - g(V - E)$$

where V is membrane potential, τ is the membrane time constant (τ = R*C), I is the imposed current, g is the membrane conductance (1/R) and E is the Nernst equilibrium potential for the leak conductance. The graph above was generated using the following numerical solution to Equation 1:

Equation 2. $$V_\infty = (I + gE)/g$$

Equation 3. $$V_{t+\partial t} = V_\infty + (V_t - V_\infty)e^{\frac{-\partial t}{\tau}}$$

where V_t is the membrane potential at time t. To transform this model membrane into a spiking neuron model, it is necessary to add a rule that states that if the voltage exceeds threshold, then $V_{t+dt} = V_{reset}$.

At this point, the student becomes somewhat indignant, not only because a rule beyond the laws of physics has been miraculously imposed upon an otherwise well-behaved physical system, but also because the action potentials produced by the IF neuron lacks the pronounced overshoot and reversal of membrane polarization that characterizes

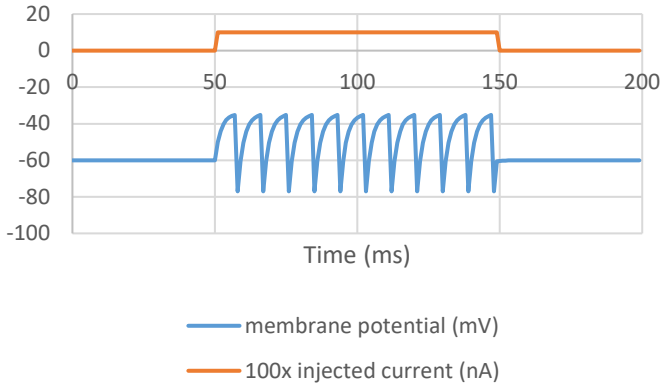

Figure 3. A graph showing a linear IF neuron based on the electrical equivalence circuit shown in Figure 1 and similar to the one shown in Figure 2, but with a "rule" imposed on the system that whenever membrane potential exceeds threshold (-35 mV in this case), the membrane potential suddenly (and inexplicably, based on the circuit in Figure1 and Equation 1-3) becomes -77 mV (approximately the Nernst equilibrium potential for potassium).

the action potential. The linear IF neuron describes only slow, subthreshold membrane dynamics, while the fast, supra-threshold behavior of the neuron is blatantly disregarded. (Most often graphs of the activity of IF neurons are aesthetically augmented by drawing vertical lines that connect threshold to the equilibrium potential for sodium whenever an action potential has occurred.)

This system is far simpler and easier to visualize and understand than the much more physiologically-realistic Hodgkin-Huxley equations, from which even determining the time constant of the membrane takes a bit of algebra.

A Nonlinear Integrate-and-Fire (IF) Neuron

The linear IF neuron's greatest short-coming is its linearity, which comes from the representation of the leak conductance as a constant resistance. Membrane resistance, of course, is not constant but changes as a function of voltage. The IF neuron can be made somewhat more realistic by adding a voltage-dependent inward current (for detailed explanations, see Friesen & Friesen, 2010):

Equation 4. $$\tau \frac{\partial V}{dt} = I - g_L(V - E_L) - g_{Na}(V - E_{Na})$$

where g_L is the leak conductance (1/R) and g_{Na} is a voltage-dependent conductance that is described by the equation:

Equation 5. $$g_{Na} = \frac{\bar{g}_{Na}}{1 + e^{\frac{h-V}{s}}}$$

This alters the circuit diagram shown in Figure 1, adding a second battery (with reverse polarity) and a variable resistor, in parallel with the capacitor (Figure 4). However, it must be noted that the variable resistor is only conceptual in nature, in the sense that it will not respond automatically to changes in voltage; if this circuit were actually constructed, this potentiometer dial would have to be manipulated manually.

There are some satisfying improvements to this model. First of all, the student no longer has to fret as to the absence of the hallmark spike component of the action potential (Figure 5). Whereas the linear IF neuron is characterized by negative feedback (which will be demonstrated below), the nonlinear IF neuron has positive feedback in the form of an

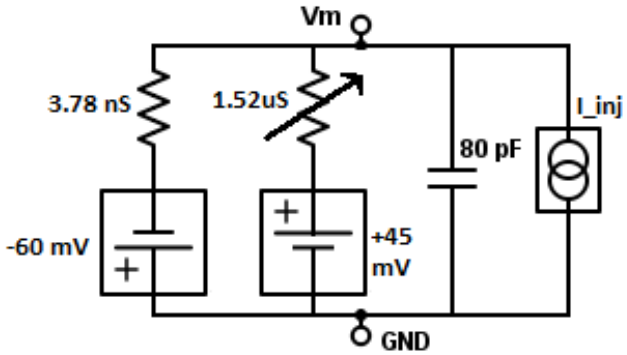

Figure 4. The electrical equivalence circuit for a nonlinear IF neuron with dynamics described by Equation 4-5. Note that resistors are now marked with associated conductances (g), and that the two batteries have opposite polarities, representing ENa (45 mV) and EL (-60 mV), respectively. (See Appendix B for photographs.)

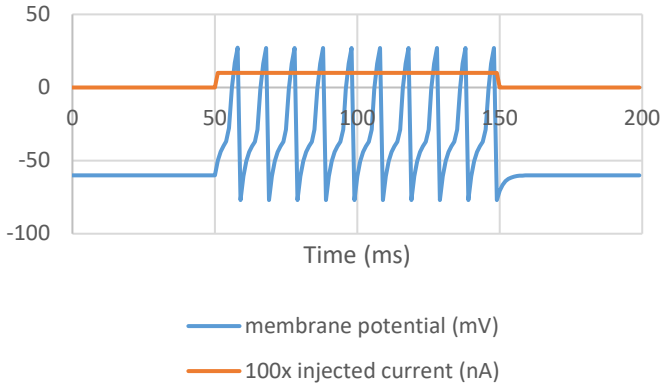

Figure 5. The firing properties of the nonlinear IF neuron described in Figure 4 and by Equations 4-5. This model's positive feedback makes it a truly spiking neuron model, although it still requires a voltage reset rule so that the action potential terminates in an after-hyperpolarization. The rule resets the voltage (again inexplicably according to the circuit in Figure 4 and Equations 4-5) to -77 mV when the membrane potential exceeds a threshold of +30 mV. [From Equation 5, h = 30 mV, s = 1 mV).]

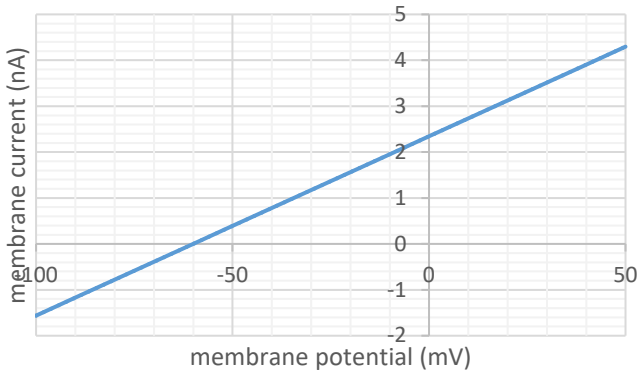

Figure 6. The IV plot of the linear integrate-and-fire neuron described by Equation 1. Note that only one equilibrium exists (the x-intercept at the membrane's resting leak potential), and that this equilibrium is stable (positive slope).

inward current that is activated by depolarization. By changing the rule to reset the voltage when it reaches a positive value (I used 20 mV), a satisfactory approximation of the action potential is elicited.

The superiority of the nonlinear IF model is especially evident by comparing its IV plot (Figure 7) to that of the linear IF neuron (Figure 6). The linear IF model simply illustrates Ohm's law: voltage is equal to current times resistance. Since the resistance is constant (voltage-independent), the IV plot is simply a straight line with a positive slope.

The x-intercept is the resting membrane potential for this cell. On its own, a resistor would have an x-intercept at the origin, but the battery in series with the resistor in Figure 1 shifts the x-intercept to the voltage of that source (the equilibrium potential of the leak current in Equation 1. The positive slope means that this model is homeostatic. If the membrane is depolarized above the resting membrane potential of -60 mV, a positive (outward) current develops in proportion to the degree of depolarization, driving the membrane potential in the negative direction back down to rest. Conversely, if the membrane potential becomes more hyperpolarized than -60 mV, a negative (inward) current develops that depolarizes the membrane back to rest. It is easy to see from Equation 1 that there is no change in the membrane potential (i.e., the derivative is zero) when membrane potential is equal to the equilibrium potential of the leak current.

The IV plot of the nonlinear IF neuron is much more interesting (Figure 7). It has two stable equilibria (x-intercepts with positive slopes), representing the two equilibrium potentials in Equation 4 (leak and sodium). It also has an unstable equilibrium characterized by an x-intercept with a negative slope. This state of the system is unstable because it will show positive feedback in response to perturbations in either direction. If it tips toward the negative direction, a positive current will develop that drives it more negative. If it trends in the positive direction, a negative current will develop that drives it ever more positive. This point is a graphical representation of the action potential threshold. Note that the inward sodium current so dwarfs the resting membrane potential that the current looks zero toward the left. The inset is a blow up of the two negative equilibria.

Both IF neurons represent systems inspired by electrical circuits with known physical properties. However, the student who attempts to build these circuits will be disappointed by the results. The electrical equivalence circuits will show no threshold, no action potential, and no intrinsic capacity for reset, except by manual manipulation of the variable resistor in the case of the circuit shown in Figure 4. Thus, the students are asked to use the properties of an analogous physical system, which itself is insufficient to account for the most salient characteristics of the biological system. Einstein's Constraint (whether truly attributable to Einstein or not) argues that the goal of theory is to reduce a complex system to the fewest simple

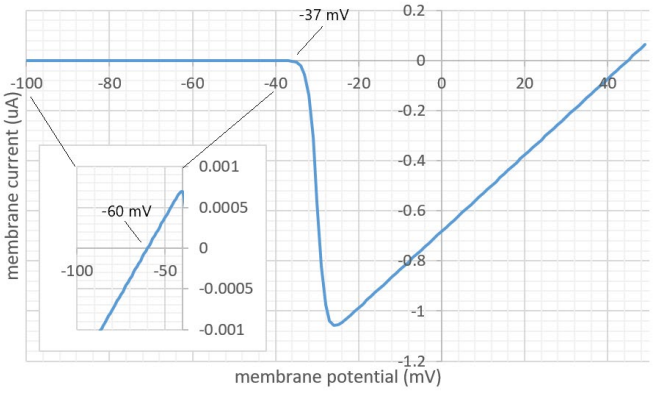

Figure 7. The IV plot of the nonlinear IF neuron described by Equations 4-5. The inset is included to show that a stable equilibrium still exists at the resting membrane potential (-60 mV). Note that there is also an unstable equilibrium at -37 mV and a pronounced region of negative slope resistance between -37 and -25 mV. There is also a second stable equilibrium at +45 mV (equilibrium potential for sodium). It is worth reminding the students that in a real cell, this stable equilibrium is ephemeral due to fast sodium channel inactivation and the opening of delayed-rectifier potassium channels.

components possible, *without compromising the fidelity of the model to the natural system's behavior*. IF neurons have merit in teaching temporal integration, stable and nonstable equilibria, IV curves and other challenging neurophysiological concepts, concepts which are challenging for undergraduates to extract from the shear complexity of the full Hodgkin-Huxley model. Nevertheless, they require the undergraduate to accept the imposition of paraphysical phenomena upon an otherwise well-behaved physical system.

There are, of course, many other nonlinear IF models with interesting properties. Several are derived from quadratic functions, and even have a second differential equation that describes activity-dependent changes in firing, such as spike frequency adaptation and bursting (Izhikevich 2003). However, such quadratic models are often too arbitrary for the students, concealing such fundamentals as membrane resistance and capacitance, so these are not necessarily the best starting point for the undergraduate.

Relaxation Oscillators as Model Neurons

The relaxation oscillator represents perhaps the exact opposite end of the model neuron spectrum. Work on relaxation oscillators as models of nerve activity dates back more than 80 years (Hill, 1933). Perhaps the most common example of a relaxation oscillator as a model neuron is the FitzHugh-Nagumo oscillator (FitzHugh 1961; Nagumo et al., 1962). This class of oscillator circuit produces a non-sinusoidal oscillation of electrical potential when constant current is applied of sufficient amplitude. Depending on how it is designed, the circuit could produce a saw-toothed wave, but in the circuit diagram shown here I have used an LED in order give the circuit a voltage spike that resembles an action potential (Figure 8). The behavior of this system is compellingly neuron-like. The electrolytic capacitor and resistor provide the RC elements that give the circuit its subthreshold time constant.

The resistor value is 470 Ohms, which is not at all comparable to the membrane resistance of a nerve cell, but was selected as a current limiting resistor when considering the powering of the LED. Likewise, an electrolytic capacitor, some 5 orders of magnitude greater than would be reasonable of the cell described above, was selected in order to give the "cell" a desired firing frequency range. The time constant for the "cell" is approximately 10-fold higher than would be reasonable for a nerve cell, and this is because on close inspection, the spike is about an order of magnitude too broad in duration. The chosen parameters allow for compression of the time axis such that the spike form and firing rate appear to be biologically realistic.

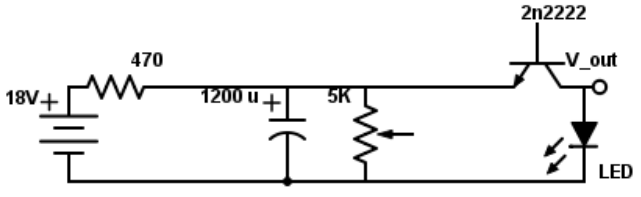

Figure 8. A simple relaxation oscillator as an alternative electrical model of a spiking nerve cell. Note that the 2n2222 transistor is wired in a reverse-biased configuration, and that the base pin is intentionally floating (unconnected). V_out represents the point at which voltage is measured and fed to the data acquisition system or oscilloscope.

The best aspect of the relaxation oscillator is that it has an intrinsic threshold, upon which a spike is generated, and after which an after-hyperpolarization develops. This is achieved by using a transistor as a switch. The 2N2222 is a general purpose NPN transistor, but in the reverse-biased mode shown in Figure 8, it has a low breakdown voltage and a negative slope resistance. It also has varactor-like properties, a varactor (or variable capacitance diode) being an electronic component with a voltage-dependent capacitance. Other general purpose transistors (such as the 2N3904 and the 2N4401) were not found to be adequate substitutes in this circuit. The "action potential" is viewed by recording the voltage drop across the visible-light LED, which is in series with the reverse-biased transistor.

The current delivered from the series 9V batteries to the transistor is controlled by the potentiometer in Figure 8, but both the batteries and the potentiometer could be eliminated if a quality variable power supply is available. At subthreshold voltages, a small depolarization is observed across the LED. However, when voltage reaches threshold, the system enters into an oscillatory state during which impulses are repeatedly fired, at a frequency proportionate to the intensity of the stimulation.

The strengths of the relaxation oscillator model are obvious. This is a buildable electrical circuit which produces many of the most salient behaviors of real neurons. The "cell" has a time constant and fires impulses at frequencies that are related to the amplitude of current injected (Figure 9, bottom trace), just as is the case for the IF neurons. However, unlike the IF models, the relaxation oscillator model also exhibits both a threshold and a visible "action potential" when that threshold is reached. Following the dramatic action potential, there is a voltage reset "after-hyperpolarization". These latter three properties are faked using a rule in the IF neuron, such that the electrical

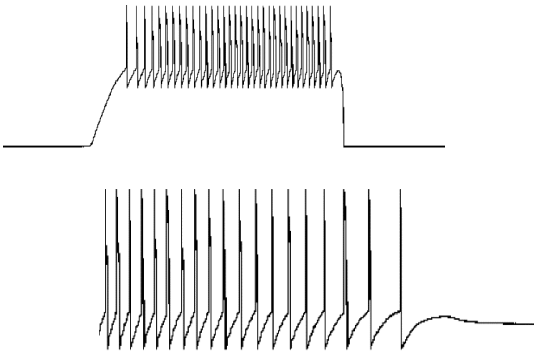

Figure 9. Spiking behavior of the relaxation oscillator. Upper trace shows the resting state, depolarization as current is applied by varying the potentiometer, the transition to a regenerative spiking state, and cessation of spiking upon lowering of the applied current back toward the minimum. Bottom trace shows near threshold spiking behavior as current is slowly lowered, with faster firing toward the left of the plot and a cessation of spiking just below threshold to the right. Note the subthreshold charging curve with measurable time constant.

equivalence circuit for the relaxation oscillator actually works (generates the spiking behavior it is intended to describe), while the electrical equivalence circuits for the IF neurons will only show charging curves related to the time constant attributable to the RC components of the circuit.

However, the relaxation oscillator model is not without its shortcomings. None of the parameters in the system resemble biologically plausible values. The LED imposes one physical limit, not turning on until approximately 1.5V is applied to it, while the breakdown voltage for the emitter-base junction in the transistor is huge on the neuronal scale (6 volts). The kinetics of spike termination in the resonance oscillator model are too slow, so that spikes are quite broad, and variable in duration, ranging from 15 ms at the lowest achievable firing rates to more than 25 ms at higher frequencies. In fact, at the highest firing rate (only about 10-15 Hz), the depolarizing phase of the action potential actually has a duty cycle of 0.38; an actual nerve cell with a 2-ms duration action potential occurring at a rate of 120 Hz would have a duty cycle of only 0.25. Furthermore, while a depolarization to about 1.94 volts (in the circuit shown in Figure 9) is necessary to trigger spiking behavior, the peak depolarization during the spike is only 1.56 V above this. If in a normal cell, the voltage threshold is approximately 25% depolarized from rest, the voltage change during the depolarizing phase of the action potential should be approximately 7 times that difference, not less than twice. This gives the relaxation oscillator model the appearance of a recording from a cell with an electrically inexcitable soma and an action potential that occurs at an electrotonically substantial distance from that soma. For a leech neurobiologist like myself, this appears normal, but for students learning neurophysiology from textbooks, it may appear strange. But they may forgive the model for this as the after-hyperpolarization gives the model's action potential a relatively normal appearance (if we ignore the y-axis).

The biggest shortcoming of the relaxation oscillator model, however, is that apart from having resistive and capacitive elements that endow it with a membrane potential, and having a threshold voltage that determines whether the model is in a static or regenerative state, there is nothing about this model that in any way represents biology. There are several factors contributing to the oscillations in this circuit, including voltage-dependent changes in *capacitance* which have no parallel in biological membranes. The termination of the action potential is due to an inactivation of sorts (the transistor is acting as a switch that controls the flow of current to the LED), but the after-hyperpolarization is attributed to a discharging of capacitance, not the interplay between two batteries and the relative resistances between them and the voltage-measuring point. While the depolarization and repolarization of the action potential depends on a complex dance between sodium and potassium ions, the relaxation oscillator system is a single charge system that oscillates. For an analogy, imagine a cell with voltage-gated sodium channels that permitted an influx of sodium so great that ENa hyperpolarized below the resting membrane potential, and that this was the reason for spike termination. The relaxation oscillator model does many of the right things, but mostly for the wrong reasons.

Conclusions

I have expressed in the past my strong desire to introduce my students to basic electronics and instrumentation in the teaching of neuroscience (Crisp et al., 2016; Crisp, 2018). I especially rely on these types of simulations and circuit-models in my upper division neurobiology seminar and in my computational neuroscience course. But they are also very useful in working one-on-one with research students on computational neuroscience projects. However, they are generally for the more advanced neuroscience student, or for any students who have a good deal of comfort with interdisciplinary material (physics, math, computer science and biology).

Part of the appeal of the relaxation oscillator as a spiking neuron model is that it gives the students a hands-on introduction to some basic electrical components (like resistors and capacitors) that are critical for the understanding of how neural membranes work. To build a simple circuit and see it behave like a spiking neuron makes for a fun and easy demonstration that sets the stage for the more abstract mathematical models of spiking neurons such as the linear and nonlinear IF neurons. The student who truly understands the similarities and shortcomings of each of these models will understand quite a bit about neurophysiology and modeling so the flaws of each models herein identified can serve as teachable moments.

REFERENCES

Crisp KM (2018) Recording EMG signals on a computer sound card. J Undergrad Neurosci Educ 16:A210-216.

Crisp KM, Lin H, Prosper I (2016) Breadboard amplifier: building and using simple electrophysiology equipment. J Undergrad Neurosci Educ 14: A124-131.

FitzHugh R (1961) Impulses and physiological states in theoretical models of nerve membrane. Biophys J 1:445-466.

Friesen WO, Friesen JA (2010) Neurodynamix II: Concepts of Neurophysiology Illustrated by Computer Simulations. Oxford, UK: Oxford University Press.

Gerstner W, Kistler WM (2002) Spiking neuron models: single cells, populations, and plasticity. Cambridge, MA: Cambridge University Press.

Hill AV (1933) Wave transmission as the basis of nerve activity. Cold Spring Harbor Symp Quant Biol 1: 146-151.

Izhikevich EM (2003) Simple model of spiking neurons. IEEE Transactions on Neural Networks 14:1569-1572.

Nagumo J, Arimoto S, Yoshizawa S (1962) An active pulse transmission line simulating nerve axon. Proc IRE 50:2061-2070.

Stein RB (1967) Some models of neuronal variability. Biophys J 7:37-68.

Received December 5, 2018; revised February 18, 2019; accepted February 19, 2019.

Address correspondence to: Dr. Kevin Crisp, Biology Department, 1500 St. Olaf Avenue, St. Olaf College, Northfield, MN 55057. Email: crisp@stolaf.edu

APPENDIX A

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# Python 2.7 code used to produce data in Figures 2,3,5,6 & 7
from math import *

# change include_fast_sodium variable = (True or False)
# to switch between linear and nonlinear IF units

include_fast_sodium = False

# simulation variables:
R = 256e6 # Ohms, measured with negative current at rest
I = 0.1e-9 # A
gl = 1/R # mhos (siemens)
El = -0.06 # V
Ena = 0.045 # V
gna_max = 1.52e-6 # mhos (siemens)
Cm = 80e-12 # F
tau = Cm*R # S
v = El # V
dt = 1e-2 # S
if include_fast_sodium: vth = 0.03 # V
else: vth = -0.035 # V
vreset = -0.077 # V
h = -0.04
s = 0.001

# compute voltage as a function of time
print "ms \t mV \t nA"
tsteps = 20000 # time-steps to simulate
for t in range(int(tsteps*dt)):
    if t > 50 and t < 150: i = I
    else: i = 0
    if include_fast_sodium:
        gna = gna_max / (1 + exp((h - v)/s))
    else:
        gna = 0
    G = gl + gna
    vinf = (i + gl*El + gna*Ena)/G
    v = vinf + (v - vinf)*exp(-dt/tau)
    if v > vth: v = vreset
    print str(t) + "\t" + str(v*1000) \
    + "\t" + str(i*10e10)
print "----------------------"

# compute current as a function of voltage
def float_range(start, stop, step):
    i = start
    while i < stop:
        yield i
        i += step
print "membrane potential (mV)" + "\t" \
      + "membrane current (nA)"
for v in float_range(-0.1,0.05,.01):
    if include_fast_sodium:
        gna = gna_max / (1 + exp((h - v)/s))
    else: gna = 0
    i = gl*(v - El) + gna*(v - Ena)
    print str(v*1000) + "\t" + str(i*10e9)
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APPENDIX B

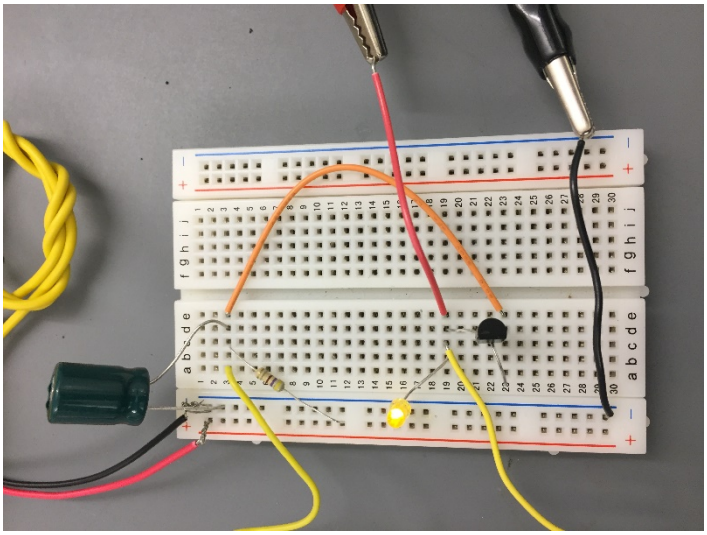

Figure A1. Photograph of a prototyping board with the circuit shown in Figure 8. Note that pin 2 of the 2n2222 is open (not connected to anything); it is actually bent upward toward the ceiling. The two yellow wires at the bottom of the image are connected to the potentiometer. The red and black leads at the top of the image are connected to the oscilloscope. The red and black wires at the left of the image are from the batteries.

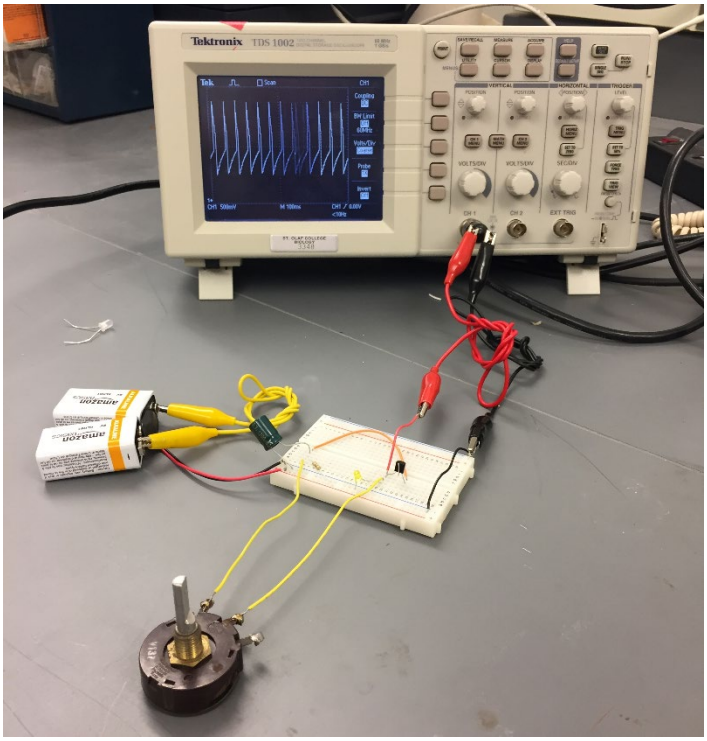

Figure A2. Photograph of the experimental apparatus used to visualize the spiking activity of the resonance oscillator depicted in Figures 8 and A1. Note that 2 9V batteries are connected in series to provide power. The potentiometer appears near the bottom left corner of the image. The exact value of the potentiometer is not critical, but something in the range of 5-20 kOhms should allow for a range of spiking activity from the resonance oscillator as the potentiometer is adjusted. Note that the LED will blink every time a spike occurs, but the oscilloscope is used to visualize the time-dependent changes in voltage (across the LED).