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A low cost, modular, and physiologically inspired electronic neuron

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We describe a low cost design of an electronic neuron, which is designed to represent the dynamical properties of the membrane potential of biological neurons by modeling the states of the membrane channels. This electronic neuron can be used to study the nonlinear properties of the membrane voltage dynamics and to develop and analyze small neuronal circuits using electronic neurons as building blocks. © *2010 American Association of Physics Teachers.*
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I. INTRODUCTION

The study of nonlinear systems and their properties has gained much importance in physics and biology teaching laboratories. An important example is the dynamics of the membrane voltage of neurons. The rich emerging properties of this system due to the opening and closing dynamics of the membrane channels[1] make it a good choice. However, the biophysical properties of these cells make the setup of electrophysiology experiments difficult to implement.

The modeling of the dynamics of neurons and the possibility of interfacing electronic neurons and living tissues is of increasing interest. The original phenomenological model of Hodgkin and Huxley[2] provides a prescription for the dynamics of the ionic conductances underlying the membrane voltage changes. A simplified and popular model was proposed by FitzHugh[3] and Nagumo *et al.*[4] The formal spiking neuron models are another important approach for modeling neurons. These simple phenomenological models (for example, integrate-and-fire[5] and the spike response models[6]) have the main properties of membrane voltage dynamics and are popular for the studies of neural coding, memory, and network dynamics.

The construction of an analog physical device that integrates the functions of a given neuron according to a particular model has been extensively analyzed. Mahowald and Douglas[7] developed a silicon integrated electronic neuron that efficiently emulates the ionic currents in the cell membrane and captures the membrane potential dynamics of real neurons. This approach introduces key elements such as calcium dynamics and stochastic noise.[8,9] A different approach was taken to build a circuit that integrates, in real time, the differential equations that model the membrane dynamics.[10,11]

One of the main advantages of using analog electronic devices is that they can be used in mixed electronic-biological systems. Thus, it is possible to use real time presynaptic signals of biological neurons to stimulate the electronic neuron and/or the output signal of the electronic neuron to stimulate postsynaptic biological neurons.[11,12]

In this paper, we discuss a low cost and biological inspired electronic neuron, which is capable of reproducing the opening and closing dynamics of the channels present in the membrane. The circuit is a simplified model of a neuron that captures the main properties of the action-potential generating mechanism. To model the dynamics of the biological channels, field effect transistors (FETs) are used. The electronic circuit exhibits the nonlinear patterns of excitability and oscillations that are equivalent to those of biological neurons.[1] One of the principal advantages of this approach is that it lets us measure the effect of each of the components in the general dynamics of the neuron. In this way, the dynamics of the membrane potential can be studied as well as the current in each of the channels. We also developed electronic synapses using the same methodology. Electronic synapses are key to interconnecting electronic neurons. By using neurons and synapses we also discuss an example of a small neural circuit. Building details of the neuron and further project proposals for students can be found in Appendixes A and B.

II. THE NEURON CIRCUIT AND ITS DYNAMICS

The electronic neuron consists of a simple circuit (see Fig. 1) inspired by the Hodgkin–Huxley conductance model, which describes the channel conductance dynamics. In our simplified approach, we focus on representing the channels' final states, without taking into account the detailed dynamical evolution of each channel. V_m and V_i are the membrane and input voltages, respectively, $R_i=39$ kΩ, and $C_m=47$ nF. Sodium (R_{Na}) and potassium (R_{K}) channels are constructed using FETs (2N7000). These FETs act as voltage-controlled switches, alternating the conductance of the channels between low and high values. Each value represents the conductance of the channel in the open (high conductance) and closed (low conductance) states.

The sodium channel schematic is shown in Fig. 2(a). Like the sodium channel present in biological cells, the electronic version has three states: Open (with high conductance), closed, and inactive (both with low conductance). The open state occurs when both FET1 and FET2 are in the high conductance state. For this state to occur the FETs gate-source voltage V_{gs} has to be at a high value (+12 V). The closed state is generated with FET1 in the low conductance state (low V_{gs}) and FET2 in the high conductance state. The inactive state is produced with FET1 with high conductance and FET2 with low conductance.

FET1 is controlled by operational amplifier 1 (OA1), which acts as a voltage comparator. $V_{\text{ref-Na-1}}$ is the threshold voltage for the neuron to spike. If V_m is below threshold, V_{gs} for FET1 equals −12 V and the FET is in the low conductance state. When V_m crosses the threshold, V_{gs} rapidly equals +12 V, and the FET state switches to high conductance.

The inactivation of the channel is controlled by FET2. The switching dynamics of this FET is controlled by OA2–OA5. OA2 acts as a buffer to measure V_m without extracting current from the circuit. OA3 is an integrator that produces a delay signal of V_m. The characteristic time delay is deter-

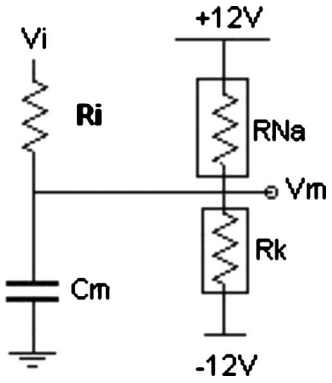

Fig. 1. Schematics for the electronic neuron circuit. V_m represents the membrane voltage, C_m the membrane capacity, V_i the stimuli signal, and R_i the input impedance. R_{Na} and R_{K} represent each of the circuits that emulate the sodium and potassium channels, respectively.

mined by $R_{d\text{-Na}}$ and $C_{d\text{-Na}}$. OA4 is a signal inverter, and OA5 acts as a voltage comparator. When the neuron is not spiking, the input signal of this comparator is less than $V_{\text{ref-Na-2}}$, and thus V_{gs}=+12 V and the FET is in the high conductance state. When the input signal is greater than $V_{\text{ref-Na-2}}$, V_{gs} switches to −12 V and the FET turns to the low conductance state, thereby inactivating the channel.

The potassium channel schematic is shown in Fig. 2(b). In this case, the channel has only two possible states: Open (FET3 with high conductance) and closed (FET3 with low conductance). The control circuit of FET3 is similar to the control circuit of FET2. Here, the natural state of the channel is closed. When the delay signal (produced by OA6–OA8) is greater than $V_{\text{ref-K}}$, the comparator (OA9) switches its output, and the channel turns to the high conductance state.

The neuron dynamics is shown in Fig. 3. A schematic of the sequence of events following a stimulus is presented in Fig. 3(a). The stimulus can be an injection of current through R_i, or the activation of an excitatory synapse, as will be shown in Sec. III. If the stimulus is enough for V_m to exceed the threshold, an action potential starts, and if not, V_m shows only an RC-like behavior.

The first step in the action potential is the opening of the sodium channel. This opening produces a positive feedback for V_m to rapidly increase its value. Two events eventually finish this process: The inactivation of the sodium channel and the opening of the potassium channel. Both events pro-

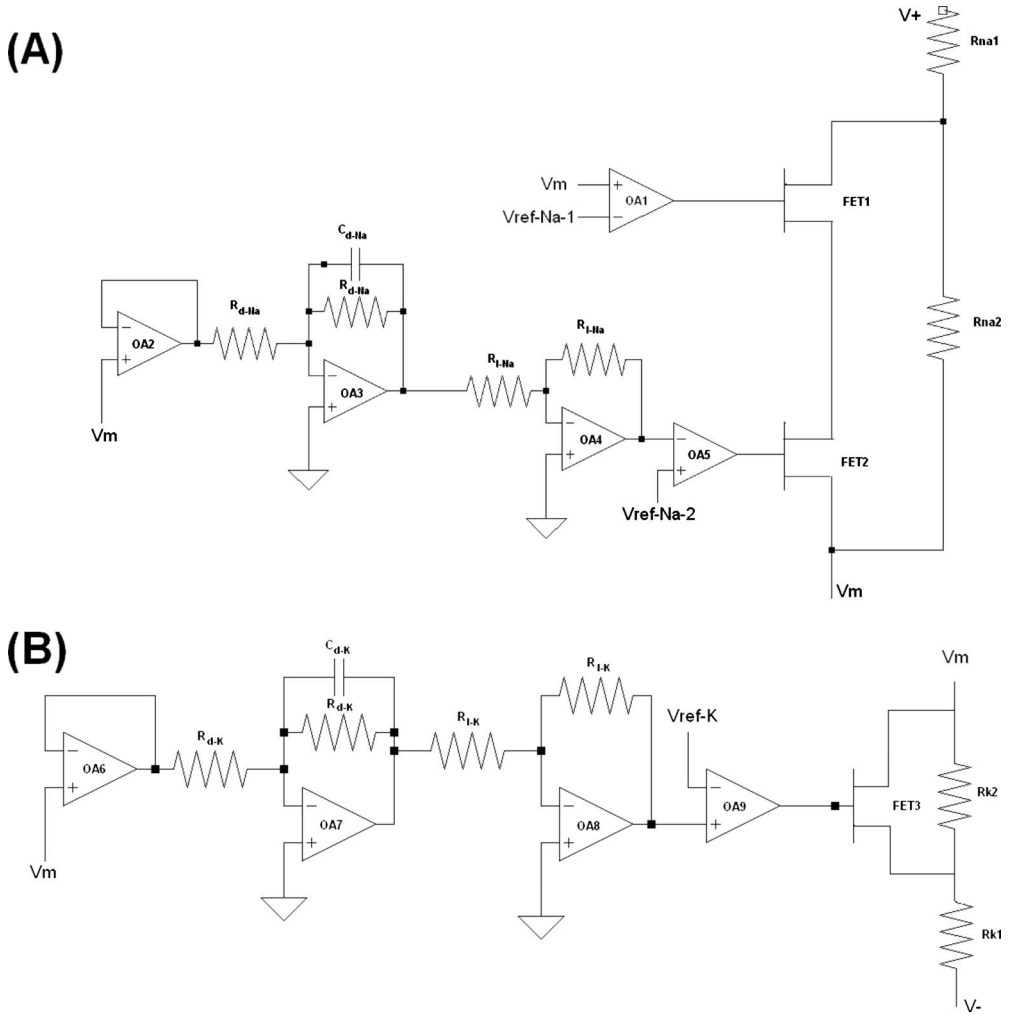

Fig. 2. Schematics for the channels. (a) Sodium channel. Two FETs control the state of the channel. FET1 rapidly opens the channel when V_m exceeds the threshold reference $V_{\text{ref1-Na-1}}$. FET2 uses a V_m delay signal produced by OA2–OA4 to inactivate the channel: $R_{d\text{-Na}}$=$R_{I\text{-Na}}$=47 kΩ, $C_{d\text{-Na}}$=100 nF, R_{Na1}=47 kΩ, and R_{NA2}=330 kΩ. (b) Potassium channel. FET3 uses a V_m delay signal to open the channel: $R_{d\text{-K}}$=$R_{I\text{-K}}$=47 kΩ, $C_{d\text{-K}}$=100 nF, R_{K1}=47 kΩ, and R_{K2}=330 kΩ.

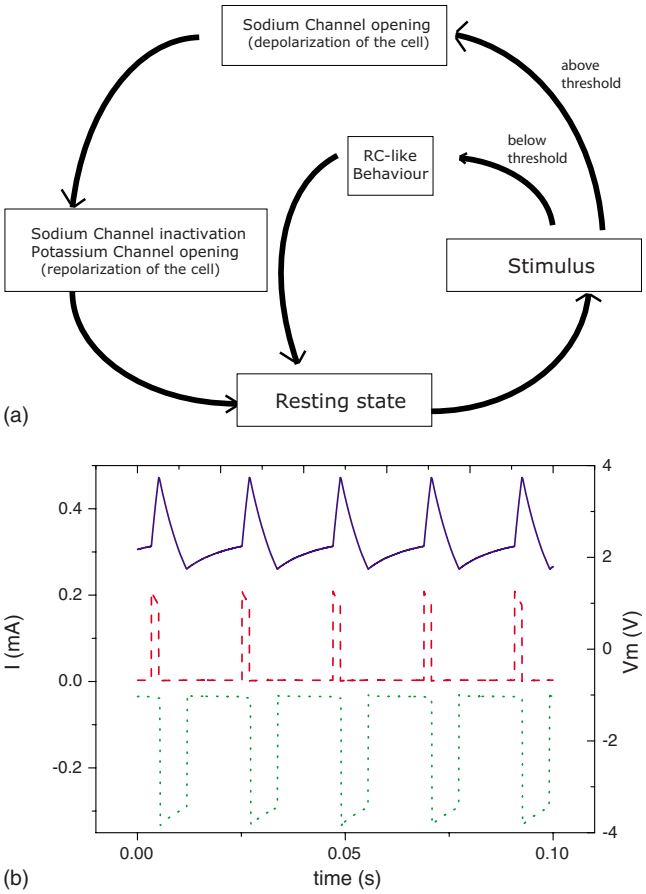

Fig. 3. Neuron excitability dynamics. (a) Schematic of the evolution of the response to a stimulus. If the stimulus is less than the threshold, the membrane potential returns to the resting value with typical RC-like behavior. If the stimulus is above threshold, an action potential is triggered. The stimulus forces the neuron to depart from the resting state. This event opens the sodium channel, thus starting the action potential, which finishes by inactivating the sodium channel and opening of the potassium channel. (b) Membrane voltage (V_m, upper continuous line) and channel currents during action potentials. During the action potential, the sodium current (middle dashed line) increases V_m, and the potassium current (bottom dotted line) decreases V_m. The sodium current starts immediately with the beginning of the action potential and finishes before the action potential ends (due to the inactivation of the channel). The potassium current starts a few milliseconds after the action potential (delay signal) and finishes when the action potential ends.

duce negative feedback for V_m, thus decreasing its value. The action potential concludes with the closing of the potassium channel and the return of the cell to its resting state. Figure 3(b) shows the dynamics of V_m, as well as the currents going through the sodium and potassium channels during the action potential.

III. SYNAPSES AND SIMPLE NEURONAL CIRCUITS

Neurons transmit information from one neuron to another principally through synapses.[1] There are two forms of synaptic transmission: Electrical and chemical. One important aspect of synaptic transmission is that the strength of the connection can be enhanced or diminished by neuronal activity. This property, called plasticity, is crucial to memory and other higher brain functions.

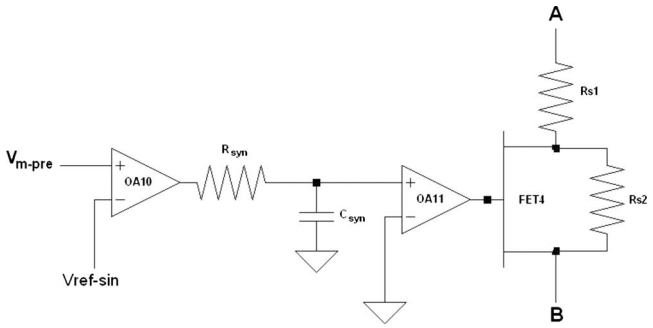

Fig. 4. Synapse circuit. The first OA acts as a comparator and starts charging the capacitor as soon as the V_m of the presynaptic cell crosses the threshold ($V_{\text{ref-syn}}$). If V_C is greater than GND, the second OA activates the FET, and thus opens the synaptic channel. The channel can be configured as excitatory or inhibitory, depending on the connection of terminals A and B (see Sec. III): R_{syn}=47 kΩ, C_{syn}=100 nF, R_{s1}=18 kΩ, and R_{s2}=470 kΩ.

Electrical synapses are fast and are characterized by having little structural and temporal variation among them. They can be described as ion channels that connect the pre- and postsynaptic neurons. In our case, this type of synapse can be represented as a simple resistance connecting V_m in both neurons.

Chemical synapses are capable of more variable interactions, and thus can produce more complex behavior. The functionality of these synapses can be schematically described as a sequence of steps: Detection of an action potential on the presynaptic neuron, releasing of chemical substances (neurotransmitters) into the space between neurons (synaptic cleft), and binding of the neurotransmitters to receptors in the postsynaptic cell. This last step opens ionic channels in the postsynaptic cell and produces changes in the membrane potential of the postsynaptic neuron. Chemical synapses can be either excitatory (excitatory synapse) or inhibitory (inhibitory synapse). Electronic implementations of synapses vary from silicon versions that mimic the detailed dynamics[13] to more functional designs.[10,12]

In our electronic version the synapse is modeled by a simple circuit (see Fig. 4). This synapse senses the presynaptic voltage ($V_{m\text{-pre}}$) with a comparator (OA10). If $V_{m\text{-pre}}$ is greater than the synaptic threshold ($V_{\text{ref-syn}}$), the comparator output switches and C_{syn} starts to charge. As soon as the voltage in C_{syn} crosses the ground voltage, the synaptic channel immediately opens (FET4) and injects a current into the postsynaptic neuron.

This synapse can be either excitatory or inhibitory and is determined by the sign of the injected current when the channel opens. For the synapse to be excitatory, the A terminal should be connected to a positive source (+12 V) and the B terminal to the postsynaptic voltage terminal ($V_{m\text{-post}}$). To be inhibitory, the A terminal should be connected to $V_{m\text{-post}}$ and the B terminal to a negative source (−12 V).

A small neuronal circuit is presented. In this circuit, neuron 1 (N1) is set to constantly spike. N1 is connected to N2 through an excitatory synapse representing a feed-forward connection. As a result, N2 spikes follow the N1 spikes. A schematic of the circuit and the evolution of V_m for N1 and N2 are shown in Fig. 5(a).

The loop can be closed by connecting N2 to N1 with an inhibitory synapse. In this case, every spike in N1 produces a

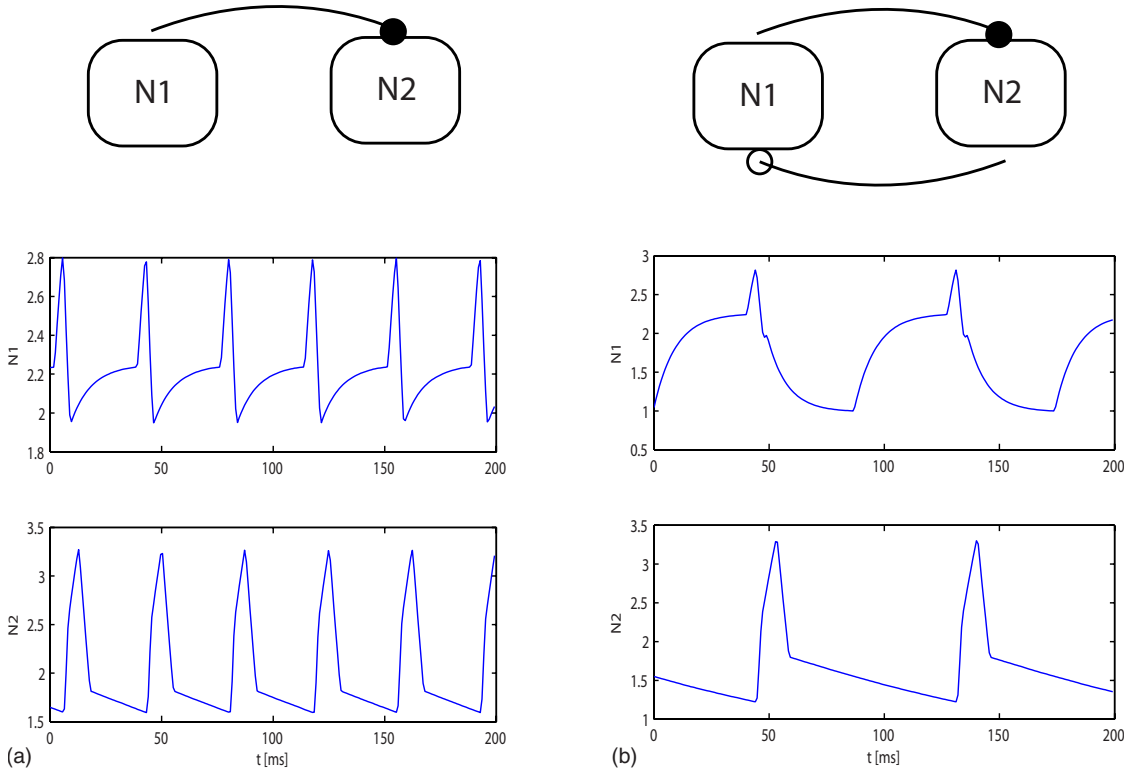

Fig. 5. Basic neuronal circuit. (a) N1 is connected to N2 via a feed-forward connection using an excitatory synapse. Below the schematic of the connection the evolution of V_m in both neurons is shown. In this case, every spike in the presynaptic neuron drives a spike in the postsynaptic neuron. (b) The previous circuit is completed with a feedback inhibitory connection from N2 to N1. Here, the effect from pre- to postsynaptic is preserved, but the action potentials in the postsynaptic neuron inhibit the presynaptic neuron, thus delaying the next spike. Also, the firing frequency of the system is decreased.

spike in N2, but the N2 spikes sequentially inhibits N1. N2 inhibition of N1 retards the appearance of a new spike in N1. As a result, the spiking frequency of the system strongly decreases. The circuit schematic and the time dependence of V_m for both neurons are shown in Fig. 5(b).

IV. DISCUSSION

The circuits are modularly constructed so that each of the modules represents biological substructures fundamental to neuronal dynamics (that is, ionic channels). The circuits permit the simultaneous measurement of variables in each of the modules, thus representing a great advantage for understanding the role played by each of the substructures in the whole neuronal dynamics.

APPENDIX A: BUILDING THE NEURON

The neuron can be built using a regular solderless protoboard distributing the components according to the interconnected electrical terminals. In our case, we used a stripboard with connections similar to a protoboard, but soldering all the components and wires, thus giving more robustness to the design.

The neuron was modularly built using three stripboards, one for the membrane circuit, one for the "sodium" channel, and one for the "potassium" channel.

The power supply of the circuit was +12 and −12 V ($V+$ and $V-$, respectively). The FETs we used are general purpose, 2N7000. The operational amplifiers are low power quad LM324 and the voltage comparator, LM311.

To establish the voltage references ($V_{\text{ref\_Na1}}$, $V_{\text{ref\_Na2}}$,and $V_{\text{ref\_K}}$), we used the three-terminal adjustable regulator, LM317. All the resistors and capacitances used were general purpose components.

APPENDIX B: SUGGESTED PROBLEMS

Problem 1. Determine the characteristics of the impulse stimuli and the outcome response by evaluating the minimum duration and/or amplitude of the impulse stimuli that triggers an action potential.

Problem 2. Study the relation between the firing frequency and the amplitude of a constant stimulus. Can the frequency be arbitrarily low? (See the types of excitability in Refs. 1, 2, and 5.)

Problem 3. Implement simple circuits such as excitatory-excitatory, excitatory-inhibitory, or inhibitory-inhibitory.

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The Astronaut Engineer

I am, and ever will be, a white-socks, pocket-protector, nerdy engineer, born under the second law of thermodynamics, steeped in steam tables, in love with free-body diagrams, transformed by Laplace, and propelled by compressible flow….

Science is about what is, and engineering is about what can be. The Greek letter eta, in lowercase, often shows up in engineering documents. Engineers pay a good bit of attention to improving eta because it is a symbol for efficiency—doing an equivalent or better job with less weight, less power, less time, less cost. The entire existence of engineers is dedicated to doing things better and more efficiently….

The twentieth was a century often punctuated with the terror of war and darkened with societal struggles to overcome injustice. But it was also the first century in which technology enabled the tenets and the images of those traumas to reach across the world and touch people in ways that were previously unimagined. John Pierce, the engineer who fathered Telstar, the first satellite to relay television signals across the Atlantic, said that engineering helped create a world in which no injustice could be hidden….

[Arthur C. Clarke's] third law seems particularly apt today: Any sufficiently developed technology is indistinguishable from magic. Truly, it has been a magical century.

From an address to the National Press Club by astronaut Neil Armstrong on February 22, 2000 reproduced in *Rocket Men* by Craig Nelson, Viking (2009) p. 404. Quote suggested by Bartley L. Cardon.