Refractory Neuron Circuits

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Abstract

Neural networks typically use an abstraction of the behaviour of a biological neuron, in which the continuously varying mean firing rate of the neuron is presumed to carry information about the neuron's time-varying state of excitation. However, the detailed timing of action potentials is known to be important in many biological systems. To build electronic models of such systems, one must have well-characterized neuron circuits that capture the essential behaviour of real neurons in biological systems. In this paper, we describe two simple and compact circuits that fire narrow action potentials with controllable thresholds, pulse widths, and refractory periods. Both circuits are well suited as high-level abstractions of spiking neurons. We have used the first circuit to generate action potentials from a current input, and have used the second circuit to delay and propagate action potentials in an axon delay line. The circuit mechanisms are derived from the behaviour of sodium and potassium conductances in nerve membranes of biological neurons. The first circuit models behaviours at the axon hillock; the second circuit models behaviour at the node of Ranvier in biological neurons. The circuits have been implemented in a 2-micron double-poly CMOS process. Results are presented from working chips.
1 Introduction

Biological neurons typically communicate with each other via narrow fixed-amplitude pulses in membrane potential known as action potentials, nerve spikes, or spikes. In this discrete-amplitude continuous-time communication mechanism, noise immunity is achieved by a coarse amplitude quantization, while detailed timing information is preserved. The neurons have a practical upper limit on their firing frequency, which is controlled by their refractory period, a period following the firing of a pulse during which the threshold for the firing of a subsequent pulse is increased. The spikes propagate like waves along lengths of nerve fiber called axons, the output wires of neurons. The spikes originate at the start of the axon in the axon hillock, the part of the axon close to the soma or cell body of the neuron. The axons are typically insulated with myelin all along their length except at certain sites along their length called nodes of Ranvier. The action potentials propagate down the axon, being constantly boosted and prevented from dying out by regenerative circuits at the nodes of Ranvier.

Artificial neural networks typically use an abstraction of real neuron behaviour, in which the continuously varying mean firing rate of the neuron is presumed to carry the information about the neuron’s time-varying state of excitation [1]. This useful simplification allows the neuron’s state to be represented as a time-varying continuous-amplitude quantity. However, spike timing is known to be important in many biological systems. For example, in nearly all vertebrate auditory systems, spiral ganglion cells from the cochlea are known to phase lock to pure-tone stimuli for all but the highest perceptible frequencies [2]. The barn owl uses axonal delays to compute azimuthal spatial localization [3]. Studies in the cat [4] have shown that relative timing of spikes is preserved even at the highest cortical levels. Studies in the visual system of the blowfly [5] have shown that information in just three spikes is enough for the fly to make a decision to turn, if the visual input is sparse.

Interest in modeling pulsed neural systems in silicon has been growing. Early work on pulse coding and pulse computation in the neural-network context was done by Murray and Smith in 1987 [6]. In 1988, Ryckebusch, Mead, and Bower [7] described small oscillatory networks, consisting of simple spiking neurons, to model central pattern generators in invertebrates. The neuron circuit that they used allowed control of the firing frequency and pulse-width of the action potential; it is analyzed by Mead [8]. This neuron circuit has been used successfully in silicon models of auditory localization [9] and the jamming-avoidance response of the weakly electric fish Eigenmannia [10]. A sophisticated silicon neuron model was introduced by Mahowald and Douglas in 1991 [11]; it modeled in great detail the behaviour of cortical neurons, including specific circuits for different ion conductances and adaptation mechanisms.

An important issue in silicon neuron modeling is the tradeoff between the degree to which biological behavior is realistically modeled and the number of parameters that must be specified for each neuron. Each parameter is usually specified by an externally applied bias voltage, which must be routed to the neuron on a wire of nonzero width. To prevent the layout area of a large network from becoming dominated by these bias wires, it is desirable to minimize the number of parameters required for each neuron. At the same time, the abstraction of neural behaviour
must not be so simple that essential biological characteristics are lost in the abstraction. In this paper, we describe two simple and compact neuron circuits that feature biologically realistic spiking behaviour. The circuits use eight and nine transistors, respectively, and have three bias-control knobs to set the threshold for firing, pulse-width, and refractory period of the action potential. We have used the first circuit to generate action potentials and have used the second circuit to delay and propagate them in an axon delay line.

The circuits are inspired by models of conductances in biological nerve membranes as modeled by Hodgkin and Huxley in 1952 [12] for a squid giant axon, and by Chiu et al. in 1979 [13] for a rabbit axon. We emphasize at the outset that the circuits are not intended to be detailed models of the biology, but rather are efficient silicon implementations of the ideas in these models. In Section 2, we describe the mechanisms behind the operation of the biological conductances. In Section 3, we describe and analyze the two neuron circuits. We present data from working chips for both of these circuits. In Section 4, we discuss applications of the two circuits. Finally, in Section 5, we conclude by summarizing the contributions of this work.

2 Biological Neurons

Hodgkin and Huxley [12] described the generation of action potentials in the surface membrane of a giant nerve fiber in the squid under the injection of a sufficiently large current. They showed that the dynamics behind the action potential could be explained completely by the presence of a quickly activating, transient sodium conductance that was responsible for the excitatory rise of the action potential and by a slowly activating, persistent potassium conductance that was responsible for the fall of the action potential. The extent to which these conductances were turned on (near their maximum values) was membrane-potential dependent. Further, the time constants governing the activation and inactivation of these conductances also were membrane-potential dependent. In this section, we give an intuitive and simplified explanation of their model as adapted from Kandel and Schwarz [14].

Figure 1 is a schematic of the conductances in the Hodgkin–Huxley model. The sodium conductance, may be viewed as being controlled by two gates in series, an \( m \) gate and an \( h \) gate. For the sodium conductance to turn on and to supply excitatory current, both the \( m \) and \( h \) gates must be open. The \( m \) gate opens rapidly at high membrane potentials and closes quickly at low membrane potentials. The \( h \) gate closes somewhat less quickly at high membrane potentials and opens slowly at low membrane potentials. If the sodium conductance is strongly turned on, the membrane potential is driven towards the sodium reversal potential, \( E_{Na} \), the \( V_{DD} \) rail of neurobiology.

The potassium conductance may be viewed as being controlled by a single \( n \) gate. For the potassium conductance to turn on and to supply inhibitory current, the \( n \) gate must be open. The \( n \) gate opens with a delay at high membrane potentials and closes slowly at low membrane potentials. If the potassium conductance is strongly turned on, the membrane potential is driven towards the potassium reversal potential, \( E_K \), the ground rail of neurobiology.
Table 1: Approximate time constants for gate opening and closing (in ms.).

<table>
<thead>
<tr>
<th>Gate/Action</th>
<th>m</th>
<th>h</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opening</td>
<td>0.1</td>
<td>8</td>
<td>1</td>
</tr>
<tr>
<td>Closing</td>
<td>0.5</td>
<td>1</td>
<td>6</td>
</tr>
</tbody>
</table>

Besides the active (voltage-dependent) sodium and potassium conductances, there is also a passive (non-voltage-dependent) conductance present that ensures that the membrane potential remains at a low resting value, $E_R$, when the active conductances are turned off. This conductance is referred to as a leak conductance. Typically, the membrane resting potential, $E_R$, is close to the potassium reversal potential, $E_K$.

The time constants for the opening and closing of the $m$, $h$ and $n$ gates are taken from Hille [15] and tabulated in Table 1. Note that these numbers refer to the time constants at the extremes of the membrane potential, that is with the membrane potential at the low resting value near $E_K$, and with the membrane potential near the high sodium reversal potential $E_{Na}$; they do not represent the actual times taken to open or close the gates.

Before the start of an action potential, the membrane is at the resting potential, $E_R$ and the $m$ gate of sodium and the $n$ gate of potassium are closed, whereas the $h$ gate of sodium is open. If the membrane is driven sufficiently positive by an excitatory input, the $m$ gate opens rapidly and sodium current rushes in to drive the membrane even more positive. The resulting positive-feedback action drives the membrane potential toward the high sodium reversal potential, $E_{Na}$. The sodium $h$ gate now closes, because of the high membrane potential. The closure of the $h$ gate shuts off the sodium conductance. Meanwhile, the more slowly activating $n$ gate opens and, along with the passive leak, brings the membrane potential back to its low resting value, $E_R$. The low membrane potential closes the $m$ gate quickly, but the $n$ gate is slower in closing and closes only some time later. The sodium conductance cannot turn on until the $h$ gate opens again. The $h$ gate is slow to open, and does not open until well after the action potential is over and consequently causes a refractory period. Note that the refractoriness arises from two sources—the lack of excitability from the sodium channel since the $h$ gate is slow to open, and the presence of inhibition from the potassium channel since the $n$ gate is slow to close. The presence of potassium inhibition increases the refractoriness after the action potential, but the lack of sodium excitability is the primary determinant of the refractory period as the latter has a slower time course and persists even after the potassium inhibition has turned off.

Chiu et al. [13] showed that, at the nodes of Ranvier of a myelinated rabbit axon, action potentials were generated when a sodium conductance and a strong passive leak conductance with no potassium conductance were present. The transient sodium conductance was responsible for the excitatory rise of the action potential as before, and the strong leak conductance was responsible for quickly restoring the membrane potential to rest and keeping the action potential brief. Thus, the need for potassium conductances is obviated if strong leak conductances are
present.

The first of our neuron circuits in Section 3 is loosely like the Hodgkin–Huxley sodium-potassium conductance pair. We refer to it as the “sodium-potassium neuron”. We primarily designed it as an efficient engineering solution to the problem of creating a refractory period after the firing of a pulse. The biological equivalent of this neuron would be as follows. A persistent sodium conductance (one that has only an $m$ gate and no $h$ gate) causes the rising phase of the action potential. The sodium conductance activates, after a delay, a potassium conductance that is coupled to it. This potassium conductance restores the membrane potential to rest and causes the falling phase of the action potential. The potassium conductance inactivates slowly, so the persistence of potassium inhibition causes a refractory period after the firing of the action potential. Sodium and potassium conductances are usually present at axon-hillock locations in biological neurons, and one can generate action potentials by injecting current at these locations. The circuit, therefore, bears some similarity to biological axon-hillock circuits.

The second of our neuron circuits described in Section 3 models a transient sodium conductance and a leak to generate action potentials. We refer to it as the “sodium-leak neuron”. The transient sodium conductance in the circuit has close similarities to the Hodgkin–Huxley sodium-conductance model, and the $m$ and $h$ gates are modeled directly by transistors in the circuit. The overall circuit is quite like the biological rabbit node of Ranvier circuit of Chiu et al. [13], which also has only leak and transient sodium conductances, and no potassium conductance.

3 Silicon Neurons

In this section, we describe, analyze and present data for the two neuron circuits. In Section 3.1 we review briefly the equations in the subthreshold region of operation of an MOS transistor, which is where we primarily operate our circuits. In Section 3.2, we discuss the sodium-potassium neuron circuit. In Section 3.3 we discuss the sodium-leak neuron circuit. Both the sodium-potassium neuron circuit and the sodium-leak neuron circuit have three bias voltages to control the threshold, pulse width, and refractory period of the action potential.

The sodium-potassium neuron circuit takes as its input a current, and generates as its output a train of pulses from ground to $V_{DD}$. The pulses are fired only if the current charges the input (and output) node higher than the threshold voltage. The firing frequency increases with input current until the upper frequency limit set by the refractory period is reached; for larger inputs the firing frequency saturates.

The sodium-leak neuron circuit takes as its input a positive step change in voltage, and generates as its output a pulse from ground to $V_{DD}$. The pulse is generated only if the height of the input step is greater than a threshold voltage. The voltage input is coupled capacitively to the input (and output) node of the neuron. The circuit is thus like a thresholding positive-edge triggered oneshot; it fires a pulse on every positive edge of a square-wave input and remains dormant on every negative edge. However, because of the circuit’s refractory properties, as the frequency of the square wave is increased, the firing shifts from occurring on every edge to on
3.1 Review of Transistor Operation in Subthreshold

In the subthreshold region of operation, the current, \( I_{ds} \), flowing through an nMOS transistor from drain to source is given by

\[
I_{ds} = I_0 e^{\frac{\kappa V_g}{U_T}} \left( e^{-\frac{V_s}{U_T}} - e^{-\frac{V_d}{U_T}} \right)
\]  

(1)

where \( V_g \) is the gate voltage, \( V_d \) is the drain voltage, \( V_s \) is the source voltage, \( U_T = \frac{kT}{q} \) is the thermal voltage, and \( I_0 \), and \( \kappa \) are constants [8]. For pMOS transistors, Eq. 1 is valid if all voltages are measured downward from \( V_{DD} \) (\( V \to V_{DD} - V \)). The parameter \( \kappa \) is usually lower for transistors in the well than for transistors in the substrate, and typically lies between 0.5 and 0.9. The parameter \( I_0 \) is different for every transistor, and scales with the width-to-length ratio of the transistor. If \( V_d - V_s > 5U_T \), Eq. 1 simplifies to

\[
I_{ds} = I_0 e^{\frac{\kappa V_g - V_s}{U_T}}
\]  

(2)

The subthreshold equations are valid in the region where

\[
\kappa V_g - V_s < V_T.
\]  

(3)

The parameter \( V_T \) is called the transistor threshold voltage. For the process in which our chips are fabricated, the threshold voltage \( V_T \) was approximately 0.9 V for both n- and p-channel transistors.

3.2 The Sodium-Potassium Neuron

In Sections 3.2.1 and 3.2.2, we describe and analyze the sodium-potassium neuron circuit. In Section 3.2.3 we present experimental data for this neuron.

3.2.1 Circuit Description and Operation

The sodium-potassium neuron circuit is shown in Figure 2. The capacitance \( C_m \) represents the neuron membrane capacitance. The voltage \( V_m \) represents the membrane potential. The input current to the circuit is \( I_{in} \). The sodium conductance behaviour is modeled by transistors \( ND \), \( NM_1 \), \( NM_2 \), \( PM_1 \) and \( PM_2 \). A higher membrane potential increases \( I_{M_1} \), which is mirrored to form the excitatory sodium current \( I_{Na} \). The mirror transistors are sized such that \( I_{Na} = 3I_{M_1} \) and \( I_{MC} = \frac{I_{M_1}}{3} \). The voltage \( V_{Na}^{th} \) sets the threshold of activation of the sodium conductance and thereby controls the threshold for firing of the neuron. The current \( I_{Na}^{max} \), set by the voltage \( V_{Na}^{max} \), controls the maximum sodium current and determines the pulse width of the action potential.
The potassium-conductance behavior is modeled by transistors $NK$ and $NR$. The voltage $V_n$ on capacitor $C_N$ represents the state of activation of the potassium conductance. The larger $V_n$ is, the stronger is the inhibitory potassium current, $I_K$, and the more refractory is the neuron. The transistor $PMC$ couples the sodium and potassium conductances, such that $C_N$ is charged high whenever there is sodium current present. The current $I_R$, set by the voltage $V_R$, controls the refractory period of the neuron.

Initially, we assume that the membrane capacitance is discharged ($V_m = 0$) and that the neuron is not in a refractory state ($V_n = 0$). Since $V_m < V_{Na}^{th}$, nearly all the current $I_{Na}^{max}$ flows in the right side of the differential pair. The input current $I_{in}$ charges $C_m$ until $V_m > V_{Na}^{th}$. At this point, the current $I_{Na}^{max}$ flows predominantly in the left side of the differential pair, so $I_{M1} = I_{Na}^{max}$, and the current mirror causes the additional current $I_{Na} = 3I_{Na}^{max}$ to charge $C_m$. After the threshold voltage $V_{Na}^{th}$ is reached, the resulting positive feedback charges $V_m$ quickly toward $V_{DD}$.

The current $I_{MC}$ is activated at the same time as $I_{Na}$; this current causes $V_n$ to rise by charging $C_N$. When $V_n$ is large enough to cause a current $I_K > I_{Na} + I_{in}$ to flow, $I_K$ pulls $V_m$ down toward ground, completing the action potential. The mirror transistor ratio $I_{Na}/I_{MC} = 9$ ensures that a full-blown action potential occurs. Once $V_m < V_{Na}^{th}$, both $I_{Na}$ and $I_{MC}$ are deactivated. As long as $V_n$ is large enough to keep $I_K > I_{in}$, $V_m$ remains discharged and the neuron is in its refractory period. $V_n$ is leaked away to ground by the current source $I_R$, which controls the duration of the refractory period. Once $V_n$ becomes small enough that $I_K < I_{in}$, the refractory period is over and $I_{in}$ is capable of initiating another action potential.

### 3.2.2 Circuit Analysis

The controlling parameters for the circuit are the voltage $V_{Na}^{th}$, which controls the threshold voltage, the current $I_{Na}^{max}$ which controls the pulse-width of the action potential, and the current $I_R$, which controls the duration of the refractory period. We expect that, under normal operation, the current $I_{Na}^{max} \gg I_{in}$, i.e. that the positive-feedback current will overwhelm the input current when an action potential is initiated.

We define the threshold for firing an action potential $V_{TH}$ as the value of $V_m$ such that the positive feedback current $I_{Na}$ is equal to the input current $I_{in}$. It can easily be shown that

$$V_{TH} = V_{Na}^{th} - \frac{U_T}{\kappa} \ln \left( \frac{3I_{Na}^{max}}{I_{in}} - 1 \right),$$

where we assume that all transistors are operating below threshold. In general, $V_{TH} < V_{Na}^{th}$, because the positive-feedback current begins to make its contribution before $V_m$ actually reaches $V_{Na}^{th}$.

The critical value for $V_n$ is that value for which the pull-down current $I_K = I_{in} + 3I_{Na}^{max}$; when $V_n$ reaches this critical voltage, $V_n^{hi}$, $I_K$ will discharge $C_m$ and cause the end of the action potential.

$$V_n^{hi} = \frac{U_T}{\kappa} \ln \left( \frac{3I_{Na}^{max} + I_{in}}{I_0} \right).$$
The pulse width, \( t_p \), or the duration of the action potential, is the time required to charge up the capacitance \( C_N \) from ground to the critical value \( V_n^{hi} \); it is given by

\[
t_p = \frac{C_N V_n^{hi}}{I_N^{max} / 3 - I_R}.
\] (6)

The circuit is usually operated in the region where \( I_N^{max} \gg I_R \), so the pulse-width is determined primarily by \( I_N^{max} \).

The refractory period is the time required for \( I_R \) to discharge \( C_N \) to a value \( V_n^{lo} \) such that the current \( I_K \) is equal to \( I_{in} \). It is easy to show that \( V_n^{lo} \) is given by

\[
V_n^{lo} = \frac{U_T}{\kappa} \ln \left( \frac{I_{in}}{I_0} \right).
\] (7)

The refractory period is

\[
t_R = \frac{C_N \left( V_n^{hi} - V_n^{lo} \right)}{I_R} = \frac{C_N U_T}{I_R} \frac{\kappa}{\kappa} \ln \left( \frac{3I_N^{max} + I_{in}}{I_{in}} \right),
\] (8)

where \( V_n^{hi} \) and \( V_n^{lo} \) are given by Eqs. 5, and 7, respectively.

3.2.3 Chip Data

Figure 3 shows the trajectories of the two voltages \( V_m \) and \( V_n \). At the beginning of the trace, \( V_m \) increases linearly due to the constant current \( I_{in} \), until \( V_m \) approaches \( V_{Na}^{th} \), the threshold voltage; at this point, \( V_m \) is quickly charged up toward \( V_{DD} \), and \( V_n \) begins to rise. A short time later, \( V_n \) is large enough to cause \( V_m \) to be pulled hard to ground, completing the action potential. \( V_n \) is then discharged slowly to ground by \( I_R \), during which time the cell is in its refractory period and \( V_m \) is held low by the transistor NK.

Figure 4a shows the spike rate versus the input voltage \( V_{in} \) for several values of the refractory voltage \( V_R \). Leakage currents place a lower limit on the spike rate of the neuron of about 0.03 Hz. As expected, the refractory period places an upper limit on the spike rate of the neuron.

Figure 4b shows the refractory period versus the control input \( V_R \). As expected, the refractory period is an exponential function of \( V_R \) in the subthreshold region, with a slope of \( \kappa q/(kT) = 27.16 V^{-1} \), corresponding to \( \kappa = 0.706 \).

3.3 The Sodium-Leak Neuron

In Sections 3.3.1 and 3.3.2, we describe and analyze the circuit. In Section 3.4 we present experimental data for the circuit.
3.3.1 Circuit Description and Operation

Figure 5 shows the sodium-leak neuron circuit. The circuit has strong similarities to the Hodgkin–Huxley model of sodium conductance. The membrane potential, referred to ground, is denoted by $V_m$. The $m$ gate is represented by transistor $NM$. The $h$ gate is represented by transistor $NH$. The membrane potential $V_m$ controls the state of the $m$ gate such that, the larger $V_m$ is, the more strongly turned on the $m$ gate is. The membrane potential $V_m$ controls the $h$ gate through transistors $NC$ and $PC$ such that, the larger $V_m$ is, the less strongly turned on the $h$ gate is. The voltage on capacitor $C_H$ represents the state of the $h$ gate; it is denoted by $V_h$. The input to the circuit is $V_{in}$; it is capacitively coupled to the membrane potential via $C_e$. The voltages $V_L$, $V_D$, and $V_R$ set the maximum currents flowing through transistors $NL$, $ND$ and $PR$ to be $I_L$, $I_D$, and $I_R$, respectively. The current $I_L$ represents the passive leak current and determines a voltage threshold and rate threshold for firing an action potential, i.e., both the input voltage change, $\Delta V_{in}$, and the input voltage's rate of change, $\frac{dV_{in}}{dt}$, must be sufficiently high for an action potential to be fired. Both these thresholds are increased if $I_L$ is increased. The current $I_D$, along with capacitor $C_H$, determines the rate of closing of the $h$ gate and thus sets the pulse width. The current $I_R$, along with capacitor $C_R$, determines the rate of opening of the $h$ gate and thus sets the refractory period. The transistors $PM_1$ and $PM_2$ form a current mirror and represent the positive feedback action of the the sodium conductance. Note that, for excitatory current to flow out of $PM_2$ and to raise the potential of $V_m$, both the $m$ gate and $h$ gate must be turned on as transistors $NM$ and $NH$ are in series.

Experimental data in Figure 6 shows the dynamic behaviour of nodes $V_{in}$, $V_m$, and $V_h$ while the circuit was generating a series of action potentials. When the circuit is at rest, the membrane potential $V_m$ is at ground, $V_{in}$ is unchanging, and $V_h$ is at $V_{DD}$. Thus, as in the Hodgkin–Huxley sodium-conductance model, the $m$ gate $NM$ is closed and the $h$ gate $NH$ is open. If there is a positive step input of magnitude $\Delta V_{in}$ at the input, a capacitive surge of current will charge the membrane potential to $\Delta V_{in}$. If $\Delta V_{in}$ is large enough, the current through the series combination of $NM$ and $NH$ will be larger than $I_L$ and will be mirrored back through $PM_1$ and $PM_2$ to charge the membrane potential node. The rise in membrane potential will increase the current still further, since $NM$ is turned on even more strongly. The resulting positive-feedback action will cause the membrane potential to be driven almost all the way to $V_{DD}$. If, on the other hand, $\Delta V_{in}$ is not large enough, the current that is mirrored back through $PM_1$ and $PM_2$ will be smaller than $I_L$ and the membrane potential will fall, causing transistor $NM$ to be turned on more weakly. The positive feedback will now turn off $NM$ completely and $I_L$ will discharge the membrane potential back to ground. Thus, there is a threshold for firing at the input—a minimum $\Delta V_{in}$ is needed to excite the circuit to $V_{DD}$. This threshold is controlled by $I_L$ or equivalently by $V_L$. A larger $I_L$ implies a larger threshold. In addition to the voltage threshold discussed above, the input voltage's rate of change, $\frac{dV_{in}}{dt}$, must be sufficiently high so that voltage increases in $V_{in}$ cause voltage increases in $V_m$ through capacitor $C_e$, in spite of the inhibiting

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1 The voltage $V_{in}$ is driven to a value that is a few mV lower than $V_{DD}$, say, $V_{DD} - \epsilon$. The value $\epsilon$ is such that the current flowing through transistor $PM_2$, with a strongly turned on gate voltage, $V_{mir}$, and a small drain-to-source voltage, $\epsilon$, is equal to $I_L$. 

9
influence of $I_L$. The smaller the capacitor $C_C$ and the larger the current $I_L$, the higher is the threshold on the rate of change of input voltage.

When $V_m$ is at $V_{DD}$, transistor $NC$ is turned on strongly and transistor $PC$ is turned off. Thus, the current, $I_D$, discharges the capacitor $C_R$, and drives $V_h$ away from $V_{DD}$ and toward ground. Eventually, $V_h$ will be low enough that the current through $NM$ and $NH$ will be less than $I_L$. The membrane potential will then begin to fall, and will be discharged back to ground by $I_L$. Thus, in response to a step of sufficient height, an action potential pulse will be generated. The pulse width is controlled primarily by the current $I_D$, or equivalently, by the voltage $V_D$, with a larger $I_D$ implying a shorter pulse width. The pulse width is also weakly dependent on $I_L$, since a larger $I_L$ will cause $V_m$ to start falling sooner and at a higher value of $V_h$, so the pulse width will be shorter.

When $V_m$ is near ground, transistor $NC$ is turned off and transistor $PC$ is turned on strongly. Now node $V_h$ is charged back up to $V_{DD}$ by the current $I_R$. The threshold for firing is high right after the firing of the action potential, since $V_h$ is close to ground and $NH$ is shut off. As $V_h$ approaches $V_{DD}$, the threshold for firing decreases progressively. Thus, the action potential has a refractory period. The refractory period is controlled by $I_R$, or equivalently, by the voltage $V_R$, with a high $I_R$ implying a short refractory period.

The circuit should be operated in the range where $\frac{I_D}{C_C} \gg \frac{I_P}{C_H} > \frac{I_R}{C_R}$. The inequality $\frac{I_D}{C_C} \gg \frac{I_P}{C_H}$ ensures that the falling edge of the action potential is brief and sharp, as compared with the rest of the pulse. The inequality $\frac{I_P}{C_H} > \frac{I_R}{C_R}$ makes the refractory period longer than the pulse width as it is in a biological action potential.

3.3.2 Circuit Analysis

In this section, we derive expressions for the dependence of the voltage threshold $V_{TH}$ on $V_L$, the pulse width $t_p$ on $V_D$, and the refractory period $t_R$ on $V_R$. We always operate the circuit with voltages $V_D$ and $V_R$ at subthreshold voltages. It is important in this circuit, as in biology that the leak current be high so that the action potential is brief. For this reason, the leak voltage $V_L$ is sometimes an above-threshold voltage. Our analysis is quantitative for the case where $V_L$ is a subthreshold voltage and is qualitative for the case where $V_L$ is an above-threshold voltage. We assume in the analysis below, that the step changes in the input are sufficiently steep so that

$$C_C \frac{dV_{in}}{dt} \gg I_L. \tag{9}$$

In effect, we are only modeling voltage threshold effects and neglecting rate threshold effects.

The threshold voltage, $V_{TH}$, is determined by that voltage, $V_m$, at which the current flowing through transistors $NH$, $NM$, $PM_1$, and $PM_2$ is $I_L$, given that $V_h$ is at $V_{DD}$ (if $V_h$ is below $V_{DD}$, the circuit is within its refractory period and the threshold is higher than the value we are computing). If $V_h$ is at $V_{DD}$, the source of transistor $NM$ will be approximately at ground, since transistor $NH$ behaves like a switch. The approximation that the source voltage of transistor
NM is at ground is an excellent approximation if \( V_L \) is a subthreshold voltage. If \( PM_1 \) and \( PM_2 \) are matched and \( NH \) and \( NL \) are matched, therefore, \( V_m = V_L \) at threshold.

Due to the presence of parasitic capacitance present at the membrane potential node, the change in membrane potential, \( \Delta V_m \), due to a step voltage change of \( \Delta V_{in} \) at the input node is given by

\[
\Delta V_m = \left( \frac{C_c}{C_c + C_{par}} \right) \Delta V_{in}, \tag{10}
\]

where \( C_{par} \) is the parasitic capacitance at the membrane potential node. Thus, the threshold measured at the input, \( V_{TH} \), is given by

\[
V_{TH} = \left( \frac{C_{par} + C_c}{C_c} \right) V_L. \tag{11}
\]

If \( V_L \) is an above-threshold voltage, we can no longer make the approximation that transistor \( NH \) is a switch, and \( NH \) behaves like a nonlinear source-degeneration resistor that reduces the transconductance of transistor \( NM \). For the current flowing through \( NM, NH, PM_1 \) and \( PM_2 \) to be \( I_L \), therefore, the voltage \( V_m \) has to higher than it was in the subthreshold case. Thus, above threshold the \( V_{TH} \) vs. \( V_L \) curve is expected to be steeper than that predicted by Eq. 11 for the subthreshold region.

The pulse width \( t_p \), is determined by the time taken to discharge \( V_h \) from \( V_{DD} \) to the value at which the current through \( NM, NH, PM_1 \), and \( PM_2 \) falls to \( I_L \). Just before the pulse ends, \( V_m = V_{DD} \). Thus, we may treat transistor \( NM \) as a switch, and approximate the current just before the pulse ends to be determined by the voltage \( V_h \). If all transistors are matched, the point at which the current through transistors \( NM, NH, PM_1 \), and \( PM_2 \) falls to \( I_L \), therefore occurs when \( V_h = V_L \). This is an excellent approximation if \( V_L \) is a subthreshold voltage. Further, we know that \( V_h \) is discharged at the rate given by \( \frac{I_D}{C_H} \). Thus, the pulse width is computed to be

\[
t_p = \frac{C_H(V_{DD} - V_L)}{I_D}. \tag{12}
\]

The current \( I_D \) is given by the subthreshold Eq. 2 for transistor \( ND \). As in the case for determining \( V_{TH} \), if \( V_L \) is above threshold, the approximation of transistor \( NH \) being a switch is no longer valid and the pulse width predicted from Eq. 12 is an overestimate, since \( V_h \) is greater than \( V_L \) at the point where \( V_m \) begins to fall.

The refractory period, \( t_R \), is determined by the time taken to recharge \( V_h \) from its low value, \( V^{lo}_h \), at the end of the pulse to a value, \( V^{hi}_h \), that is sufficiently high that the current flowing through the chain of transistors \( NM, NH, PM_1 \) and \( PM_2 \) is \( I_L \); that is,

\[
t_R = \frac{C_H(V_h^{hi} - V_h^{lo})}{I_R}. \tag{13}
\]

In subthreshold, the current \( I_R \) is given by

\[
I_R = I_0^{PR} e^{\frac{V_{DD} - V_R}{V_T}}, \tag{14}
\]
where $\kappa_p$ and $I_0^{PR}$ are the $\kappa$ and $I_0$ parameters for transistor $PR$. From the analysis to determine $t_p$, we know that $V_{h}^L = V_L$ is an excellent approximation if $V_L$ is a subthreshold voltage; it is an underestimate if $V_L$ is an above-threshold voltage. The voltage $V_h^{hi}$ is a function of how close $\Delta V_{in}$ is to the threshold $V_{TH}$. We now compute $V_h^{hi}$ as a function of $\Delta V_{in}$ for $V_L$ being a subthreshold voltage. We assume, for simplicity, that $C_{par} = 0$, $V_{OS} = 0$, and all transistors are matched. The current flowing through $NM$ and $NH$ may be shown to be

$$I_{mh} = \frac{I_0 e^{\frac{\Delta V_{in}}{U_T}} e^{\frac{V_h^{hi}}{U_T}}}{e^{\frac{\Delta V_{in}}{U_T}} + e^{\frac{V_h^{hi}}{U_T}}} \tag{15}$$

where $V_m$ and $V_h$ are both sub-threshold voltages, Eq. 1 is valid for transistors $NM$ and $NH$, and $V_{mir} \gg \frac{5kT}{q}$. The voltage $V_h^{hi}$ is then the solution to the equation

$$I_{mh} = I_0 e^{\frac{V_h^{hi}}{U_T}} \tag{16}$$

with $I_{mh}$ as defined in Eq. 15, $V_m = \Delta V_{in}$ and $V_h$ being the unknown variable for which we must solve. Some algebraic manipulation yields,

$$V_h^{hi} = \Delta V_{in} - \frac{U_T}{\kappa} \ln \left( e^{\frac{\Delta V_{in} - V_L}{U_T}} - 1 \right) \tag{17}$$

with $\Delta V_{in} > V_L$, so an action potential exists. The limiting cases of Eq. 17 yield

$$\lim_{\Delta V_{in} \to \infty} V_h^{hi} = V_L, \quad \lim_{\Delta V_{in} \to -V_L} V_h^{hi} = \infty. \tag{18, 19}$$

Note that the R.H.S. of Eq. (17) is a very steep function of $\Delta V_{in}$. Within approximately $\frac{5kT}{q}$, which is about 0.2 V, it changes from $\infty$ to $V_L$. The two limiting cases are consistent with physical intuition about the circuit: If $\Delta V_{in}$ is very large, the point at which the current through transistors $NM$, $NH$, $PM_1$ and $PM_2$ is $I_L$ occurs when $V_h \approx V_L$, since $NH$ limits the current and $NM$ is a switch. If $\Delta V_{in}$ is very near $V_L$, the point at which the current through transistors $NM$, $NH$, $PM_1$, and $PM_2$ is $I_L$ occurs when $V_h$ is very large, since $NM$ limits the current and $NH$ must be as perfect a switch as it can be. In practice, since we can never attain $\infty$, but rather can attain only $V_{DD}$, the lowest obtainable threshold has to be slightly greater than $V_L$; similarly, the lowest $V_h^{hi}$ will have to be slightly greater than $V_L$. If $V_L$ is an above-threshold voltage, Eq. 17 is not valid. The functional relation between $V_h^{hi}$ and $\Delta V_{in}$, however, has a similar form and Eqs. 18 and 19 are still true. The voltage $V_h^{hi}$ is a monotonic decreasing function of $\Delta V_{in}$ that decreases from $V_{DD}$ at $\Delta V_{in} \approx V_L$ to $V_L$ at $\Delta V_{in} \approx V_{DD}$.

### 3.4 Chip Data

Data were taken from a chip containing the circuit of Figure 5. The data are shown in Figures 6–9. Circuit waveform data are shown in Figure 6 and were discussed in Section 3.3.1. The $V_{TH}$
versus $V_L$ curve of Figure 7 was fit by the expression $1.6(V_L - 0.05)$ for the subthreshold range of voltages $0.5 < V_L < 0.85$ V. The data are consistent with Eq. 11, except for the presence of a small offset of 50 mV. The offset is partly due to transistor mismatch, partly because the function generator used to collect data had a slight overshoot in its square wave output and partly because $V_m$ does not rest exactly at ground. We obtained independent measurements of the parasitic capacitance attenuation from $V_{in}$ to $V_m$ by taking the ratio of the peak-to-peak amplitudes of a sine wave at $V_{in}$ and $V_m$ respectively, with the circuit turned off ($V_L$ at ground, $V_D$ at $V_{DD}$, $V_R$ at $V_{DD}$); they yielded a value of 1.58. The value of 1.58 compares very favorably with the value of 1.6 from data. Above threshold, the $V_{TH}$ versus $V_L$ curve deviates from the subthreshold fit curve and becomes increasingly steep as expected from the discussion that follows Eq. 11 in Section 3.3.2.

Equation 12 predicts that the $t_p$ vs. $V_D$ curve should decrease exponentially in the subthreshold regime; Figure 8 shows that this prediction is correct. The data points were fit to an exponential in the range $0.450 < V_D < 0.800$ with a $\kappa$ of 0.68, which compares favorably with the $\kappa = 0.66$ value determined by an independent measurement on a test nMOS transistor on the same chip.

We obtained refractory period data by measuring the critical frequency, $f_R$, at which the circuit transitioned from firing on every rising edge of the input $V_{in}$ (a square-wave input with frequency $f_{in}$) to firing on every other rising edge of the input, as a function of $V_R$. This frequency-halving behaviour is shown in Figure 9a. The experiment was performed with the magnitude of the positive step change in the input $\Delta V_{in}$ approximately equal to the threshold for firing, $V_{TH}$. This procedure yields the maximum possible refractory period as discussed in Section 3.3.2. The refractory period, $t_R$ was then computed to be

$$t_R = \frac{1}{f_R} - t_p.$$  

(20)

While taking these measurements, we always ensured that the input period, $\frac{1}{f_R}$, was greater than the pulse width, $t_p$; that is, as $f_R$ increased with decreasing $V_R$, we simultaneously decreased $t_p$ by increasing $V_D$. Figure 9b shows a plot of the data obtained for $t_R$ versus $V_R$. As expected from Eq. 13, the data are exponential in the subthreshold regime. The data points in the range $4.25 < V_R < 4.65$ fit an exponential, with the $\kappa$ parameter being 0.62. The $\kappa$ determined by an independent measurement on a test pMOS transistor on the same chip was 0.56. This discrepancy was caused by the reduction of the threshold for firing with increasing frequency: As the frequency of the input is increased, the voltage at the gate of the mirror transistor $PM_1$, $V_{mir}$, has less time between successive inputs to charge back up to $V_{DD}$. Consequently, at higher frequencies, $V_{mir}$ is at a lower voltage between action potentials and the current subtracted from transistor $NL$ by transistor $PM_2$ is higher so the threshold is systematically lower. The lower threshold greatly reduces $V_{th}$ and, consequently, $t_R$, so the decrease of $t_R$ with increasing frequency or decreasing $V_R$ is made steeper. The $\kappa$ parameter obtained from the data on $t_R$ versus $V_R$ is thus higher than one would expect from single-transistor measurements.
4 Circuit Applications

We have used the sodium-potassium neuron circuit in a number of applications that involve the processing of information from electronic cochleas [16] [17]. Phase and amplitude information from signals in the cochlea are processed by small neuronal-network circuits that model inner hair cells and spiral ganglion cells in real cochleas, and are encoded as phase-locked neuronal discharges, just as in biological auditory nerve fibers. The sodium-potassium neuron has been used in an auditory center-surround circuit to provide frequency sharpening, and in small networks that model the central pattern generator circuits involved in motor control in invertebrates [18].

Whereas the sodium-potassium neuron is naturally suited for generating spike trains and building networks, the sodium-leak neuron is naturally suited for propagating and delaying action potentials, and for doing delay computations. By inverting the output (the $V_m$ node) of the sodium-leak neuron we obtain an active low positive-edge triggered oneshot. A cascade of such one-shots builds an axon delay line where the pulses are delayed at each succeeding stage by one pulse width. Data from such an axon delay line are shown in Figure 10. We have used the onset-detecting and delay-generating properties of the neuron to build a working chip that computes velocity estimates of locally moving edges in a 1D visual image [19]. The neuron has also been used in a working chip that recognizes sequences of two tones in an auditory input.

5 Conclusions

The two biologically inspired spiking neuron circuits that we have described and analyzed in this paper are robust, compact (eight to nine transistors), easily controllable, and operate over wide ranges in firing frequency, pulse width, refractory period and threshold. The firing frequency can be varied from a few tenths of 1 Hz. to 100 kHz. In the subthreshold region of operation, the pulse widths and/or refractory periods of the action potentials can be varied from a few tens of μsecs. to hundreds of msecs. The threshold for firing can be varied from approximately 0.7 V to $V_{DD}$. The power consumption of these circuits is low, since the circuits operate primarily in the subthreshold regime. The circuits are thus eminently suited for use as fundamental modules in pulsed-mode neural-network circuitry or as building blocks in the silicon modeling of neurobiological systems. We have used these circuits to build working electronic auditory nerve fibers, central pattern generators, axon delay lines, and visual motion detectors.

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References


Figure Captions

Figure 1—The Hodgkin–Huxley Conductance Model: The sodium conductance has \( m \) and \( h \) gates whose extent of opening is controlled by the membrane potential, \( V_m \). The more open the \( m \) and \( h \) gates are, the larger the sodium conductance is. Similarly, the more open the \( n \) gate is, the larger the potassium conductance is. The leak conductance is non-voltage-dependent. The sodium, potassium and leak conductances are connected to voltage sources of value \( E_{Na} \), \( E_K \) and \( E_L \) respectively.

Figure 2—The Sodium-Potassium Neuron Circuit: The voltage, \( V_m \), on capacitor \( C_m \) is the membrane potential, and the voltage \( V_n \) on capacitor \( C_N \) is the voltage that determines the state of refractoriness of the neuron. The bias voltages \( V_{Na}^{th} \), \( V_{Na}^{max} \) and \( V_R \) set the threshold for firing, pulse width, and refractory period of the action potential, respectively. The input current, \( I_{in} \), determines the frequency of firing of the neuron, as long as it is not limited by the neuron's refractory period.

Figure 3—Circuit Waveforms for the Sodium-Potassium Neuron Circuit: The voltage \( V_m \) increases linearly with time, as the capacitor \( C_m \) is charged up by the input current from ground to the threshold for firing. When the threshold for firing is reached, the positive feedback in the circuit is activated and \( V_m \) quickly rises toward \( V_{DD} \). At the threshold for firing, the voltage, \( V_n \), also rises quickly from ground as capacitor \( C_N \) is charged up by a scaled copy of the positive-feedback current. The rise in \( V_n \) activates a discharging current at the membrane potential node that returns \( V_m \) to ground. The capacitor \( C_N \) is then slowly discharged by a passive leak current at the \( V_n \) node, which returns \( V_n \) linearly to ground. Meanwhile, the input current charges \( C_m \) back up to the threshold for firing, and the cycle of activity is periodically repeated. These waveforms were obtained with \( V_{Na}^{th} = 2.50 \) V, \( V_{Na}^{max} = 1.20 \) V, \( V_R = 0.99 \) V, and \( V_{in} = 0.81 \) V.

Figure 4—Input Output Characteristics of the Sodium-Potassium Neuron Circuit: Figure 4a shows that the firing frequency or spike rate of the neuron is linear with input current as long as the frequency of firing is not limited by the refractory period of the neuron. The data displayed for Figure 4a were taken with \( V_{Na}^{th} = 2.50 \) V, and \( V_{Na}^{max} = 1.72 \) V, for all curves.

Figure 4b shows the refractory period \( t_{ref} \) as a function of \( V_R \), for a given input current \( I_{in} \). In subthreshold, \( t_{ref} \) decreases exponentially with \( V_R \). Data for Figure 4b were taken with \( V_{Na}^{th} = 2.50 \) V, \( V_{Na}^{max} = 1.72 \), and \( V_{in} = 1.15 \).
Figure 5—The Sodium-Leak Neuron Circuit: The voltage, $V_m$, is the membrane potential, and the voltage $V_h$ on capacitor $C_h$ is the voltage that determines the state of refractoriness of the neuron. The bias voltages $V_L$, $V_D$ and $V_R$ set the threshold for firing, pulse width, and refractory period of the action potential, respectively. The input $V_m$ is capacitively coupled to the circuit by capacitor $C_C$. An action potential is fired on every positive edge of a square-wave input, as long as the period of the input is greater than the neuron's refractory period.

Figure 6—Circuit Waveforms for the Sodium-Leak Neuron Circuit: In response to a positive edge of sufficient height at the input, the positive feedback in the circuit is activated, so the membrane potential, $V_m$, is driven from ground to $V_{DD}$. The capacitor $C_h$ is then discharged from $V_{DD}$ by a current source that is switched on when $V_m$ reaches $V_{DD}$; $V_h$ falls linearly toward ground. Eventually, $V_h$ gets close enough to ground, that the inhibitory leak current at the membrane potential node exceeds the excitatory positive feedback current; the leak current, then, discharges $V_m$ to ground. When $V_m$ is at ground, the discharging current source at the $V_h$ node is switched off and a charging current source is switched on. The charging current source starts charging $C_h$ back up toward $V_{DD}$, so $V_h$ rises linearly toward $V_{DD}$. After $V_h$ has reached $V_{DD}$, the cycle of activity repeats again on the next positive edge in the input. These waveforms were obtained with $V_L = 1.516$, $V_D = 0.656$, $V_R = 4.40$, and $V_i$ being a square-wave input of frequency 100 Hz and step height 2.630 V. Note that these waveforms are representative of a case where the period of the input is greater than the neuron’s refractory period.

Figure 7—Threshold Voltage Characteristics for the Sodium-Leak Neuron Circuit: The threshold voltage, $V_{TH}$, is the height of the smallest positive step at the input that can cause an action potential. The threshold is linear with $V_L$ for subthreshold values of $V_L$ and deviates from linearity for above-threshold values of $V_L$. The data above were taken for $V_D = 0.502$ V, $V_R = 4.63$ V, with the input being a square wave of frequency 0.91 Hz.
Figure 8—Pulse Width Characteristics for the Sodium-Leak Neuron Circuit
The pulse width, $t_p$, decays inversely with $I_D$, and thus exponentially with $V_D$ for subthreshold values of $V_D$. The data above were taken for $V_L = 1.50\, V$, $V_R = 4.63\, V$, with the input being a square wave of frequency 0.91 Hz.

Figure 9—Refractory Period Characteristics for the Sodium-Leak Neuron Circuit
Figure 9a shows the frequency halving behavior of the sodium-leak neuron circuit. As the period of the square wave input is less than the refractory period of the neuron, the circuit fires an action potential only on every other positive edge of the square-wave. The refractory period, $t_R$, is the inter-pulse interval at which frequency halving just begins to occur. Figure 9b shows that the refractory period, $t_R$, increases exponentially with $V_R$ for subthreshold values of $V_R$. The data above were taken for $V_L = 1.50\, V$, with the input being a square wave of step height $2.57\, V$.

Figure 10—The Axon Delay Line
The figure shows the input to an axon delay line and the outputs from the first five taps on the line. The delay line is built by having a chain of sodium-leak neurons coupled to one another via inverters. Each output is delayed from its predecessor by a pulse-width. The curves have been offset for clarity. The input p.p. value is $2.240\, V$ and the pulse-widths are between 30 and 50 ms.
Figure 1
Figure 3
Figures 4a (top) and 4b (bottom)
Figure 5
Figure 6
Figure 8