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Control of bursting properties in a silicon neuron CPG

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Abstract

We have developed a silicon neuron based on a heart interneuron of the leech. We created a half-center oscillator composed of two silicon neurons connected via inhibitory synapses implemented through dynamic clamp. We investigated the effects of symmetrical variations of maximal conductances on bursting behavior. Burst period, average burst spike frequency, and duty cycle were chosen as major characteristics of the bursting waveform. Burst period and spike frequency showed similar dependencies on the varied parameters in both neurons; duty cycles of the two neurons, however, diverged when the parameters were varied, reflecting mismatch in parameters between chips.

Keywords: aVLSI; Half-center oscillator; Central pattern generator; Motor control

1. Introduction

We have developed a multi-conductance analog very large-scale integrated (aVLSI) model neuron that mimics the dynamics of the leech oscillator heart (HN) interneuron. The dynamical behavior of the silicon neuron is described by a mathematical model [10] that is based upon the Hodgkin–Huxley formalism [4]. Our goal is to use this silicon model neuron to study principles of biological motor pattern generation and apply them to engineered systems. Here, we examined how complex behaviors such as

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bursting can be controlled through parameter variation within a half-center oscillator composed of two silicon neurons.

2. Silicon neuron design

Dynamics of the silicon neuron is based on seven ionic conductances, which are summed onto a membrane capacitor. The currents which are based on studies of a mathematical model of the oscillator HN interneuron [3,6,7] were chosen because they are most important in determining the neuronal dynamics. Specifically, these currents are: (1) a sodium current with instantaneous activation and fast inactivation, I_{Na}; (2) a persistent, non-inactivating sodium current with slow activation, I_{P}; (3) a potassium current with fast activation and inactivation, I_{K1}; (4) a non-inactivating potassium current with slow activation, I_{K2}; (5) a slowly activating and slowly inactivating calcium current, I_{Ca}; (6) a hyperpolarization-activated current with slow activation, I_{H}; and (7) a leak current, I_{leak}.

The summation of these currents onto the membrane capacitor causes a variation in membrane voltage described by

$$\frac{\mathrm{d}V_{\mathrm{m}}}{\mathrm{d}t} = \frac{1}{C_{\mathrm{m}}}[I_{\mathrm{Na}} + I_{\mathrm{P}} + I_{\mathrm{K1}} + I_{\mathrm{K2}} + I_{\mathrm{Ca}} + I_{\mathrm{H}} + I_{\mathrm{leak}}],$$

where V_{m} is the membrane potential and C_{m} the membrane capacitance. Each of the voltage-dependent currents identified by subscript i is described by

$$I_i = g_i m_i h_i (E_i - V_{\mathrm{m}}),$$

where g_i is the maximal conductance, m_i and h_i the activation and inactivation state variables, respectively, and E_i the reversal potential. For non-inactivating currents h_i is equal to 1, and for I_{leak} both m_i and h_i are equal to 1.

The steady-state activation and inactivation functions are given by

$$m_{\infty i} = \frac{1}{1 + \exp(S_{\mathrm{m}}(V_{mi} - V_{\mathrm{m}}))},$$

$$h_{\infty i} = \frac{1}{1 + \exp(-S_h(V_{hi} - V_{\mathrm{m}}))}.$$

The dynamics of the activation and inactivation state variables are given by

$$\frac{\mathrm{d}m_i}{\mathrm{d}t} = \frac{1}{\tau_{mi}}(m_{\infty i} - m_i),$$

$$\frac{\mathrm{d}h_i}{\mathrm{d}t} = \frac{1}{\tau_{hi}}(h_{\infty i} - h_i),$$

where τ_{mi} and τ_{hi} are the time constants for activation and inactivation, respectively. It is notable that there are two distinct differences in the way voltage-gated currents are represented in our silicon neuron compared to the Hodgkin–Huxley representation. First, the time constants in our silicon neuron are constant and not voltage-dependent, and second, the activation state variable are always raised only to the first

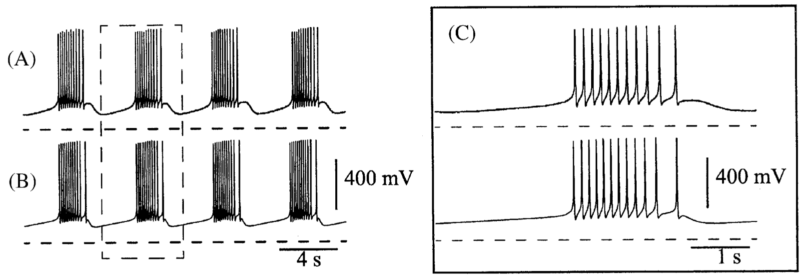

Fig. 1. Silicon neuron membrane voltage waveform (A) compared to mathematical simulation of the silicon neuron (B), and the number of spikes in the zoom of the bursts (C).

power, whereas in the standard Hodgkin–Huxley representation they can be raised to power > 1.

3. Silicon neuron performance

In Fig. 1, we compare the performance of the silicon neuron to a mathematical simulation of the silicon neuron at canonical parameter values. Fig. 1 clearly illustrates that the burst periods are very close. The zoom of the bursts (Fig. 1C) demonstrates that the number of spikes per burst and spike frequency are also very similar. Intrinsic bursting of this type is predicted by the mathematical model of an HN oscillator interneuron and is observed in extracellular recordings of HN neurons [1,2].

In addition, we note that the burst period observed here is close to that observed in a living HN cell.

4. Half-center oscillator design

Through the use of dynamic clamp [8], we implemented mutually inhibitory synaptic connections between two silicon neurons, creating a half-center oscillator schematically depicted in Fig. 2.

These inhibitory synaptic currents are described by

$$I_{\mathrm{syn}} = g_{\mathrm{syn}} s (E_{\mathrm{syn}} - V_{\mathrm{m}}),$$

$$s_{\infty} = \begin{cases} 0, & V_{\mathrm{pre}} < V_{\mathrm{th}}, \\ \tanh(S_{\mathrm{syn}}(V_{\mathrm{pre}} - V_{\mathrm{th}})), & V_{\mathrm{pre}} \geqslant V_{\mathrm{th}}, \end{cases}$$

$$\frac{\mathrm{d}s}{\mathrm{d}t} = \frac{1}{\tau_{\mathrm{syn}}}(s_{\infty} - s),$$

where I_{syn} is the post-synaptic current, g_{syn} the maximal post-synaptic conductance, E_{syn} the post-synaptic reversal potential, s_{∞} the steady-state activation function, S_{syn}

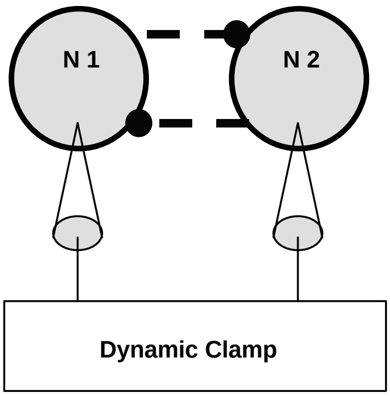

Fig. 2. Schematic design of half-center oscillator composed of two silicon neurons N1 and N2 connected via inhibitory synapses (dashed lines) implemented through dynamic clamp.

the slope of this function, s the synaptic state variable, τ_{syn} the synaptic time constant, V_{pre} the pre-synaptic membrane potential, and V_{th} the synaptic threshold voltage.

5. Half-center oscillator performance

The neurons were initially biased equally with parameters that were based on the leech HN mathematical model [10]. However, because of mismatch in the transistors of the silicon neurons, the parameters g_{K1}, g_{K2}, τ_{mCa} and τ_{hCa}, were adjusted slightly to make the bursting properties, in particular duty cycles, the same. We call the whole set of adjusted parameter values the canonical set. From the canonical set we varied two maximal conductances of two slow currents, I_{Ca} and I_{H}, to determine their feasibility as control parameters for bursting behavior; specifically, we studied their effect upon three major characteristics of bursting waveform: burst period, average spike frequency during the burst, and duty cycle. Fig. 3 shows the effect of varying solely g_{Ca} on the bursting behavior of the two-neuron system. The burst period of the neurons is similar to both our mathematical model of an HN oscillator interneuron and physiological recordings.

Fig. 4 demonstrates the effect of equally varying g_{Ca} and g_{H} in both neurons on burst period, average burst spike frequency, and duty cycle. Burst period and spike frequency show a non-linear dependence upon g_{Ca} and g_{H}, and were very closely matched between silicon neurons N1 and N2. It is not surprising that burst periods of the two cells are matched, as this is a necessary result of alternating periodic bursting in a half-center oscillator.

Duty cycle, however, varies noticeably between the two neurons with variation of g_{H} (Fig. 4D) and is not as pronounced for variation of g_{Ca} (Fig. 3). This disparity is most likely due to a combination of the unequally adjusted parameters in the canonical set and fabrication mismatch.

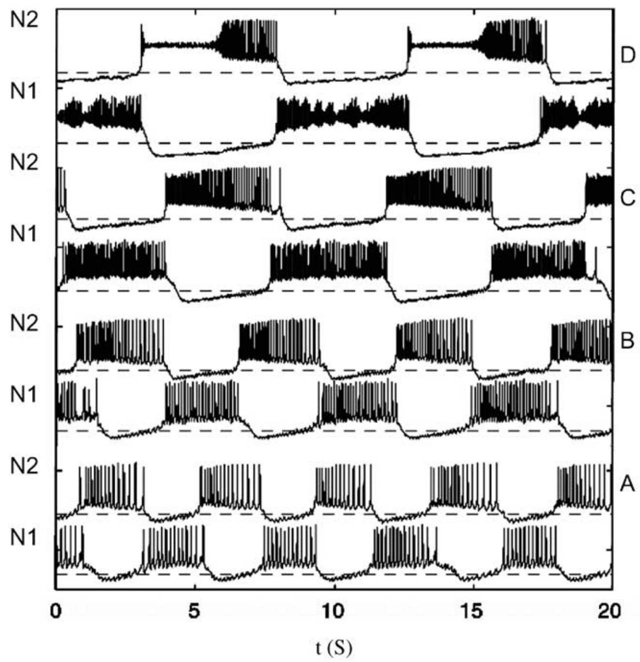

Fig. 3. Examples of bursting waveforms in half-center oscillator composed of silicon neurons N1 and N2, corresponding to different values of g_{Ca}. The silicon neuron can generate membrane potential between 0.5 and 1.5 V. Dashed lines mark leak reversal potential ($E_{\mathrm{leak}} = 0.6$ V, which corresponds to $E_{\mathrm{leak}} = -50$ mV in a biological neuron). (A) $g_{\mathrm{Ca}} = 0.76$ nS (canonical); (B) $g_{\mathrm{Ca}} = 1.01$ nS; (C) $g_{\mathrm{Ca}} = 1.35$ nS; (D) $g_{\mathrm{Ca}} = 1.79$ nS.

6. Discussion

Period, spike frequency, and duty cycle are all important characteristics for the control of motor systems both biological and engineered. Understanding how underlying neuronal parameters effect these characteristics is critical for building motor systems controlled by silicon neurons that seek to match the effectiveness of biological systems. We have demonstrated two parameters which are useful for control of these characteristics, and have identified limitations due to differences between the elements. Both biological and engineered systems share the problem of disparate parameters among fundamental operating units. Biological systems can overcome this problem through self-adaptation of these units.

Studies of activity-dependent adaptation in neurons [5,11,12] have determined potential mechanisms by which problems due to parameter mismatch can be overcome. Some of these mechanisms have been implemented in previous versions of silicon neurons [9].

The half-center oscillator configuration is an important underlying mechanism of pattern generation in biological systems. Our understanding of its operation is critical for

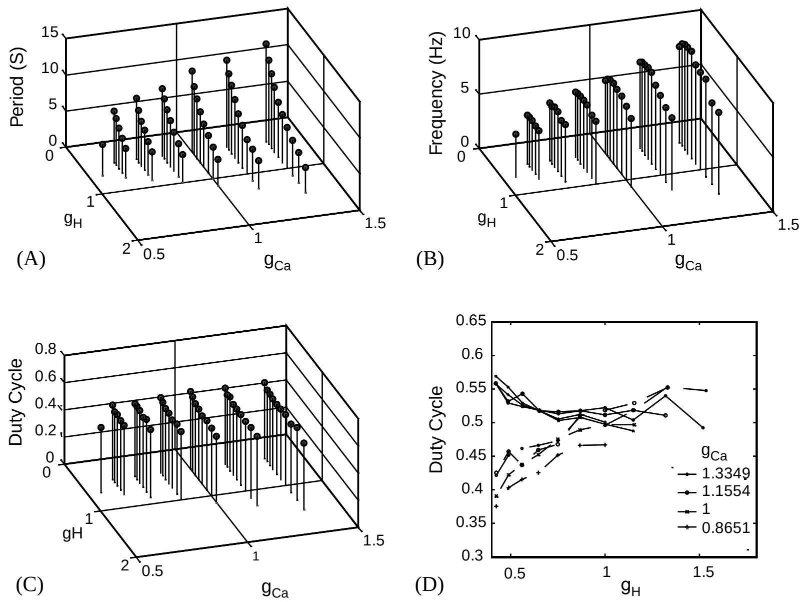

Fig. 4. Effect of equally varying g_{Ca} and g_H in both neurons, N1 and N2, on burst period (A), spike frequency (B), and duty cycle (C), shown for neuron N1. (D) Change in duty cycle with varying g_H. Neurons N1 (solid line) and N2 (dashed line) are shown at several values of g_{Ca} (D). Maximal conductances are shown as a ratio to the canonical value.

understanding many biological problems, and developing engineered systems inspired by biological behavior.

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References

[1] G.S. Cymbalyuk, R.L. Calabrese, Endogenous bursting of leech heart interneurons, Society Neuroscience Meeting, New Orleans, 164.2, 2000.

[2] G.S. Cymbalyuk, R.L. Calabrese, Oscillatory behaviors in pharmacologically isolated heart interneurons from the medicinal leech, Neurocomputing 32 (2000) 97–104.

[3] A.A. Hill, J. Lu, M.A. Masino, O.H. Olsen, R.L. Calabrese, A model of a segmental oscillator in the leech heartbeat neuronal network, J. Comput. Neurosci. 10 (2001) 281–302.

[4] A.L. Hodgkin, A.F. Huxley, A quantitative description of membrane current and its application to conduction and excitation in nerve, J. Physiol. 117 (1952) 500–544.

[5] G. LeMasson, E. Marder, L.F. Abbott, Activity-dependent regulation of conductances in model neurons, Science 259 (1993) 1915–1917.

[6] F. Nadim, Ø.H. Olsen, E. De Schutter, R.L. Calabrese, Modeling the leech heartbeat elemental oscillator. I. Interactions of intrinsic and synaptic currents, J. Comput. Neurosci. 2 (1995) 215–235.
[7] Ø.H. Olsen, F. Nadim, R.L. Calabrese, Modeling the leech heartbeat elemental oscillator. II. Exploring the parameter space, J. Comput. Neurosci. 2 (1995) 237–257.
[8] A.A. Sharp, F.K. Skinner, E. Marder, Mechanisms of oscillation in dynamic clamp constructed two-cell half-center circuits, J. Neurophysiol. 76 (1996) 867–883.
[9] M.F. Simoni, S.P. DeWeerth, Adaptation in a VLSI model of a neuron, IEEE Trans. Circuits Syst. II: Analog Digital Signal Processing 46 (1999) 967–970.
[10] M.F. Simoni, Synthesis and analysis of a physical model of biological rhythmic movement control with sensorimotor feedback. Ph.D. thesis, Georgia Institute of Technology (2002), 16–50.
[11] C. Soto-Trevino, K.A. Thoroughman, E. Marder, L.F. Abbott, Activity-dependent modification of inhibitory synapses in models of rhythmic neural networks, Nat. Neurosci. 4 (2001) 297–303.
[12] G. Turrigiano, L.F. Abbott, E. Marder, Activity-dependent changes in the intrinsic properties of cultured neurons, Science 264 (1994) 974–977.