Simple central pattern generator model using phasic analog neurons

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Many biological neurons (called phasic or adapting neurons) display neural adaptation: their response to a constant input diminishes with time. A simple method of adding adaptive firing thresholds to existing analog (or graded-response) neural models is described. A half-center central pattern generator is modeled using two mutually inhibitory phasic analog neurons. Hopf bifurcation analysis shows that oscillatory solutions will arise if the mutual inhibition is sufficiently strong, and allows us to characterize the stability of the cycles which arise. [S1063-651X(99)01006-5]

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I. INTRODUCTION

Animals are capable of remarkable feats of perception, learning, and cognition. The neuron is the building block out of which the networks generating animal behavior are made, and there has therefore been considerable interest in modeling the function of neurons, as a step toward understanding (and perhaps mimicking) the capabilities of living creatures.

Broadly speaking, neurons respond to stimuli by generating action potentials: voltage spikes which travel down the cell's axon. Arriving at synaptic junctions, these spikes influence, typically through neurotransmitters diffused across a synaptic gap, the states of other neurons (or of muscles or other tissues). To model the behavior of a neuron, we may work at the level of the biochemistry of the cell, or we may propose simplified models which capture various aspects of the dynamics. Several popular neural models are, in order of increasing abstractness, the Hodgkin-Huxley equations [1], the FitzHugh-Nagumo equations [2], and integrate-and-fire models (see, for example, Refs. [3,4]).

At a higher level of abstraction, we may replace the individual spiking times with a time-averaged firing rate. Information is lost in this process (see Ref. [5] for a discussion of this point); the result is a considerably simplified model in which each neuron may be considered to output an analog value, its firing rate. Such ''analog'' or ''graded-response'' neural models were proposed by Hopfield [6] and Cohen and Grossberg [7], and may be applied in cases where the time scale of interest is long relative to the typical interspike time. Analog models may be explicitly derived from spiking-time models by carrying out the time averaging process; Bressloff and Coombes [4], for example, generated an analog integral equation from an integrate-and-fire model by convolving the postsynaptic potential with a firing rate function. Analog neurons have proven useful in modeling associative memory [6,8], as behavior controllers for autonomous robots [9], and in solving optimization problems [10].

In associative memory or optimization problems, networks of analog neurons produce their ''answer'' by converging to a fixed-point attractor. In the memory problem we create an attractor corresponding to each stored pattern, and expect the network to recover the original pattern when presented with a noisy version of it. For such applications, we always want the network to converge to a fixed point, and oscillatory solutions are to be avoided. Extensive analysis has been performed on networks of the types introduced in Refs. [6,7], and it has been shown (see, in addition to the original papers, Refs. [11–16]) that they do indeed have the property of always converging to a fixed point.

There are many biological situations, however, in which oscillations are necessary, for example, to drive autonomic functions and in locomotion (see Ref. [17] and references therein). It is thus of interest to examine situations in which the much-studied analog neuron model may be made to generate oscillatory solutions. Many of the oscillatory neural signals seen in biology are generated by central pattern generators (CPG's): networks of neurons whose interconnections are such that the neurons collectively produce rhythmic outputs. CPG's often work on the principle of mutual inhibition, in which neurons (or groups of neurons) are reciprocally connected so that the output of each neuron inhibits the other [17]. Perhaps the earliest description of a CPG of the type shown in Fig. 1 was Brown's ''half-center model'' [18]. As Brown noted, oscillations in two mutually inhibitory neurons can occur if the inhibition is limited in duration. If an initial asymmetry allows the first neuron to dominate, it will ''gain the upper hand,'' suppressing the other while firing strongly itself. If this inhibition is of limited duration, the second neuron will eventually cease to be suppressed, allowing it to dominate and inhibit the first, and so on, yielding a cycle of alternating bursts of activity in the two neurons. Despite its simplicity, the half-center model does capture the essential dynamics of CPG's actually observed in biology: Satterlie [19], for example, described the signals used in swimming in the pteropod mollusk *Clione limacina* as being generated by this mechanism.

What could cause the limited duration of inhibition which the half-center model assumes? There are several possible

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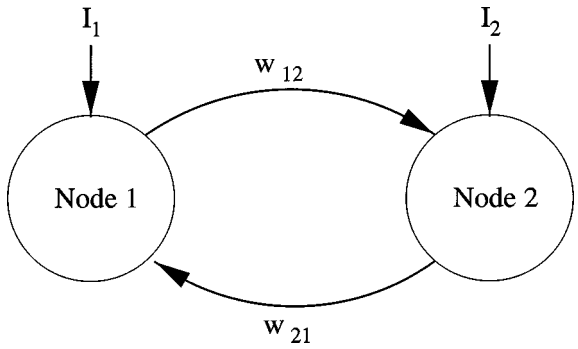

FIG. 1. A two-neuron network, representing Brown's half-center model [18]. Standard analog neuron models [6,7] will not yield oscillatory solutions in this configuration, but the addition of neural adaptation to these models allows oscillations to occur, making the network into a simple central pattern generator.

neurophysiological mechanisms, including fatigue, post-inhibitory rebound, and neural adaptation [17,20]. We shall focus on the last of these. While some biological neurons are ''tonic,'' responding with a steady firing rate output when stimulated with a constant input, many others are ''phasic'' or ''adapting,'' initially responding to a constant stimulus, but gradually ceasing to respond as the stimulation persists [21]. (Fig. 2 shows the different responses of tonic and phasic neurons to a constant input.) Clearly if the two neurons in Fig. 1 are phasic, oscillations become possible; once a given neuron has come to dominate, its input becomes constant and it will eventually ''adapt out,'' reducing its output and allowing the other neuron to take over. (The term ''adaptation'' is used in many different contexts in biology. In this paper, we shall always use adaptation to refer to the neural behavior just described.)

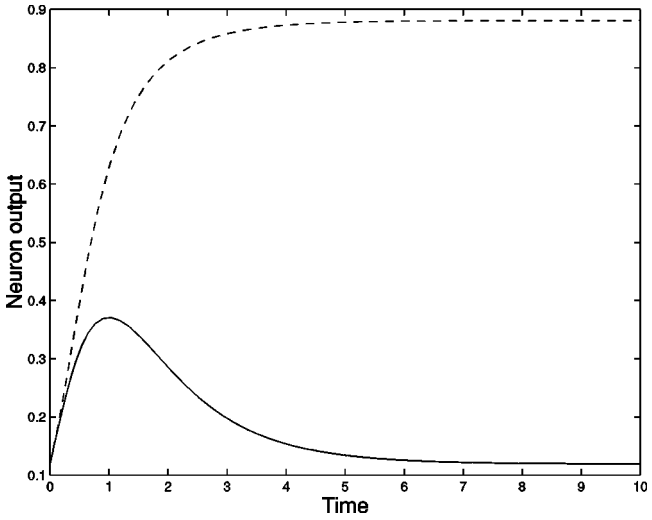

FIG. 2. The different responses of tonic and phasic analog neurons. The plot shows the time integration of Eq. (1) for a single neuron with no self-connection, receiving a constant input $I=1$, with parameters $\tau=1$, $\gamma=4$, and $\theta=-2$. The neuron's output $y=f(\gamma(x-\alpha)+\theta)$ is shown for $k=0$ (dashed line; a tonic neuron) and $k=1$ (solid line; a phasic neuron). The output may be seen as a fraction of the neuron's maximum firing rate, with 0 representing no action potentials being produced, and 1 representing spiking at the highest attainable rate. Here the sigmoidal firing function $f_1(q)=1/[1+\exp(-q)]$ has been used.

Suppose that we wish to model the half-center CPG using analog neurons connected as in Fig. 1. If we use two standard analog neurons [6,7], the system will converge to a fixed point, and no oscillations will occur. If we wish this simple two-neuron system to oscillate, we must introduce some mechanism to limit the inhibitory duration. We shall do this by proposing a simple means by which the qualitative dynamics of neural adaptation may be added to existing analog neuron models. Beyond allowing us to model the half-center CPG, the addition of neural adaptation to existing analog neurons enriches their dynamics, and extends the range of neurological phenomena to which they may be applied.

We begin by introducing our ''phasic analog neurons,'' then proceed to discuss a model of the half-center CPG formed by connecting two such neurons with mutual inhibition. A Hopf bifurcation analysis of the model reveals the inhibitory connection strength at which oscillations will occur, and shows us how to tune the system parameters to yield cycles with desired characteristics.

II. PHASIC ANALOG NEURONS

Networks of the Hopfield [6] or Cohen-Grossberg [7] types capture the essential dynamics of temporal summation: biological neurons maintain a decaying trace of their past excitation levels [21]. Models of this type generally omit, however, the dynamics of neural adaptation: many real neurons (called ''phasic'' or ''adapting'') respond only at the onset of a constant or slowly varying stimulus, then cease responding as the stimulus persists [21]; biological neurons which respond steadily to constant input also exist, and are called ''tonic.'' We propose a simple method by which a form of neural adaptation may be added to existing analog neuron models.

We will consider the analog equation $\tau\dot{x}=-x+I$ [6,9]. Here x represents the degree of excitation of the neuron, corresponding to some generalized ''tendency to fire.'' In the real biological system, this is a complex function of many factors, including the membrane potential and various ionic concentrations. The term I is the net input to the neuron. The neuron's output is a firing rate $y=f(x)$, where $f(\cdot)$ is some (generally nonlinear) function mapping the cell's excitation to the rate of production of action potentials; we assume that $f(\cdot)$ maps all values onto the range $[0,1]$, so we may view y as a fraction of the neuron's maximum firing rate.

Neural adaptation is often attributed to changes in the behavior of ion channels in the cellular membrane, for example the activation of Ca^{2+}-dependent K^+ channels or the inactivation of Na^+ or Ca^{2+} channels [22–24]. It is possible to construct detailed models which capture the dynamics of adaptation at the ionic level; see Wang [24] for an example. Here we are concerned only with the qualitative dynamics: we want adaptation to act so as to limit the duration of a neuron's firing in response to steady or slowly varying inputs. We expect the adaptive firing threshold to increase as the neuron becomes more excited, to decay back to rest in the absence of excitation, and to reach an upper limit set by the excitation. The simplest way to meet these criteria is with an equation of the form $\dot{\alpha}=k(x-\alpha)$, where α is the firing threshold. This equation excludes any nonlinear effects, and produces the desired behavior.

Augmenting each neuron's description with a second differential equation, we now write the dynamics of a network of n neurons as

$$\tau_i \dot{x}_i = -x_i + \sum_j w_{ji} y_j + I_i$$
$$\dot{\alpha}_i = k_i (x_i - \alpha_i) \tag{1}$$

for $i = 1, \ldots, n$. x_i represent excitation levels with time constants $\tau_i > 0$. α_i are firing thresholds with rate constants $k_i \geq 0$. y_i represent output firing rates, and are functions of the difference between x and α: we use $y_i = f(\gamma(x_i - \alpha_i) + \theta)$, where $f(\cdot)$ is some firing rate function and $\gamma > 0$ and θ are scaling and shifting parameters. We will not specify the firing rate function at this point; in Sec. III we will discuss the effects of two different choices of this function. We take the connection strengths (w_{ij} from neuron i to neuron j) to be constant. Each neuron receives an external input I_i, which may be time varying. Figure 2 shows the result of integrating Eq. (1) for a single node with no self-connection ($w_{11} = 0$).

Matsuoka [25] proposed a similar approach to adding neural adaptation to an analog model, but one in which an adaptation term is incorporated directly into the activation equation; in our model, the $\dot{\alpha}$ equation may be appended to any form of activation equation [for concreteness, we will use the form in Eq. (1) throughout this paper]. Horn and Usher [26] described a form of adaptation for discrete-time, binary-state neurons, as did Halperin [27].

The effect of neural adaptation is high-pass filtering of the input signal [28], and the addition of the $\dot{\alpha}$ equation to an analog neural model is equivalent to passing x through an RC high-pass filter circuit, with $k = 1/RC$. Since the effect of temporal summation is low-pass filtering of the input [29], a phasic neuron acts as a band-pass filter. Consider a single neuron of the type given in (1), with no self-connection: $\tau \dot{x} = -x + I(t)$, $\dot{\alpha} = k(x - \alpha)$. We take the input to be $I(t) = \cos \omega t$. The steady-state output is then $(x - \alpha)(t) = D \cos(\omega t + \psi)$, with

$$D = \omega [\omega^2 (1 + k\tau)^2 + (k - \omega^2 \tau)^2]^{-1/2} \tag{2}$$

and

$$\psi = \tan^{-1} \left[\frac{k - \omega^2 \tau}{\omega (1 + k\tau)} \right]. \tag{3}$$

The amplitude D drops to zero as $\omega \to 0$ and as $\omega \to \infty$, reaching a maximum value of $D = 1/(1 + k\tau)$ at $\omega = \sqrt{k/\tau}$. The phase ψ is zero at $\omega = \sqrt{k/\tau}$, approaches $\pi/2$ as $\omega \to 0$, and approaches $-\pi/2$ as $\omega \to \infty$.

Adaptation is most often discussed in relation to sensory neurons, so it is perhaps worth pointing out that motor neurons can also display this behavior. Atwood and Nguyen [30], for example, discussed phasic and tonic motor neurons in crayfish.

III. HALF-CENTER CPG: HOPF BIFURCATION ANALYSIS

We now consider the behavior of two phasic neurons, reciprocally connected as shown in Fig. 1. This represents the dynamics of a simple CPG, the half-center model [17,18,20], and we will show that oscillatory solutions arise for sufficiently strong mutual inhibition. Consider the case of two identical neurons ($\tau_1 = \tau_2 = \tau$, $k_1 = k_2 = k$) with a symmetric connection ($w_{12} = w_{21} = w$) and no self-connections ($w_{11} = w_{22} = 0$). The system has a single fixed point; we shift this point to the origin and write

$$\tau \dot{\tilde{x}}_1 = -\tilde{x}_1 + w f(\gamma(\tilde{x}_2 - \tilde{\alpha}_2) + \theta) - w f(\theta),$$
$$\dot{\tilde{\alpha}}_1 = k(\tilde{x}_1 - \tilde{\alpha}_1),$$
$$\tau \dot{\tilde{x}}_2 = -\tilde{x}_2 + w f(\gamma(\tilde{x}_1 - \tilde{\alpha}_1) + \theta) - w f(\theta),$$
$$\dot{\tilde{\alpha}}_2 = k(\tilde{x}_2 - \tilde{\alpha}_2), \tag{4}$$

where we have defined $\tilde{x}_i = x_i - I_i - w f(\theta)$ and $\tilde{\alpha}_i = \alpha_i - I_i - w f(\theta)$. Here we assume that the inputs I_i are constants.

Treating w as a bifurcation parameter, we perform a Hopf bifurcation analysis of Eq. (4) using standard techniques. See Ref. [31] for more information. (Also, Collins and Stewart [32] discussed Hopf bifurcations in a general class of coupled nonlinear oscillators, and Atiya and Baldi [3] demonstrated the bifurcation in a pair of asymmetrically connected neurons with self-connections.)

Evaluating the Jacobian of Eqs. (4) at the origin, we seek values of w for which a pair of purely imaginary eigenvalues exists; this is the first condition of the Hopf bifurcation theorem. At $w = \pm w^*$, where $w^* = (1 + k\tau)/\gamma f'(\theta)$ with $f'(q) = \partial f(q)/\partial q$, we have the eigenvalues $\lambda_{1,2} = [-(1 + k\tau) \pm \sqrt{1 + k\tau + (k\tau)^2}]/\tau$ and $\lambda_{3,4} = \pm \sqrt{-k/\tau} = \pm i\omega$, where $\omega \equiv \sqrt{k/\tau}$. Note that λ_1 and λ_2 are real and both strictly negative for $k > 0$.

The second condition of the Hopf bifurcation theorem requires that the eigenvalues cross the imaginary axis with nonzero ''velocity:''

$$d[\mathrm{Re}(\lambda)]/dw|_{w = \pm w^*} \equiv d \neq 0. \tag{5}$$

For our system, we find $d = \pm \gamma f'(\theta)/2\tau$. For $f'(\theta) \neq 0$, both conditions of the Hopf theorem are satisfied at $w = \pm w^*$. We now examine the stability of the periodic solutions which emerge after the bifurcation.

Since the half-center model relies on mutual inhibition, we will consider $w = -w^* - \mu$, and examine what occurs as μ crosses from negative to positive values. Applying a linear change of coordinates, we bring Eqs. (4) into the normal form

$$\begin{pmatrix} \dot{z}_1 \\ \dot{z}_2 \end{pmatrix} = \begin{pmatrix} \lambda_1 & 0 \\ 0 & \lambda_2 \end{pmatrix} \begin{pmatrix} z_1 \\ z_2 \end{pmatrix} + \begin{pmatrix} \phi_1(z) \\ \phi_2(z) \end{pmatrix},$$
$$\begin{pmatrix} \dot{z}_3 \\ \dot{z}_4 \end{pmatrix} = \begin{pmatrix} 0 & -\omega \\ \omega & 0 \end{pmatrix} \begin{pmatrix} z_3 \\ z_4 \end{pmatrix} + \begin{pmatrix} \phi_3(z) \\ \phi_4(z) \end{pmatrix}. \tag{6}$$

We define the abbreviations $A \equiv k\tau$, $B \equiv \sqrt{1+A+A^2}$, $C \equiv B(A+1)$, $D \equiv B(A-1)$, $E \equiv C-(A+1)^2$, $F \equiv D+A^2+1$, $G \equiv D-A^2-1$, $H \equiv C+(A+1)^2$, $\chi_1 \equiv \gamma((A-B)z_1/(1+A)+(A+B)z_2/(1+A)-z_3/\sqrt{A})+\theta$, $\chi_2 \equiv \gamma((A-B)z_1/(1+A)+(A+B)z_2/(1+A)+z_3/\sqrt{A})+\theta$, $J_{12} \equiv f(\chi_1)+f(\chi_2)-2f(\theta)$, and $J_{21} \equiv f(\chi_2)-f(\chi_1)$. We may then write the nonlinear terms in Eqs. (6) as

$$2B\tau\phi_1(z) = Ez_1 + Fz_2 + wJ_{12}(B-1)/2,$$

$$2B\tau\phi_2(z) = Gz_1 + Hz_2 + wJ_{12}(B+1)/2,$$

$$\tau\phi_3(z) = -z_3(A+1) + wJ_{21}\sqrt{A}/2, \tag{7}$$

$$\phi_4(z) = 0.$$

The stability coefficient for the periodic solutions, commonly denoted a, is defined in terms of partial derivatives of the functions (7); see Ref. [31] for the expression. We approximate the center manifold by a power series near the origin, obtain the coefficients, evaluate the derivatives, and after some algebra find that

$$a = \frac{\gamma^2}{16(k\tau)\tau}\frac{N_1 - N_2}{D_1}, \tag{8}$$

where

$$N_1 = f'''(\theta)f'(\theta)(16(k\tau)^3 + 57(k\tau)^2 + 57k\tau + 16),$$

$$N_2 = 8[f''(\theta)]^2((k\tau)^3 + 3(k\tau)^2 + 3k\tau + 1), \tag{9}$$

$$D_1 = [f'(\theta)]^2(16(k\tau)^2 + 41k\tau + 16).$$

Both supercritical ($a<0$; stable oscillatory solutions) and subcritical ($a>0$; unstable oscillatory solutions) Hopf bifurcations occur for our system, depending on the choice of the parameters k, τ, γ, and θ. The sign of a is a function only of θ (associated with the baseline output of each neuron), and the product $k\tau$ (which is the ratio of the time scales of the excitation and adaptation equations). The magnitude of a depends on all four parameters; note from Eq. (8), however, that the dependence on γ is a simple proportionality to γ^2.

The details of the stability of the oscillatory solutions depend on the form of the firing rate function, $f(\cdot)$. First we consider a common sigmoidal choice, $f_1(q) = 1/[1 + \exp(-q)]$. We have $f'(\theta) > 0$ for all θ, and thus the transversality condition (5) is satisfied for any choice of θ. The stability boundaries for this case are shown in Fig. 3. Note that for this firing function, whether the bifurcation is supercritical or subcritical depends mainly on the value of θ, which is associated with the spontaneous firing rate of each neuron ($f(\theta)$ is the firing rate when the neuron is either unexcited or fully adapted). Figure 4 shows a plot of $a(\theta)$ with the other parameters fixed at $k=\tau=1$ and $\gamma=4$.

When deriving an analog version of their integrate-and-fire model, Bressloff and Coombes [4] used a nonsigmoidal firing rate function. A simplified version of their function is $f_2(q) = \Theta(q)/[1 + \ln(1+1/q)]$, where $\Theta(q) = 1$ for $q>0$, and is zero otherwise. For this choice of firing function, we must consider only $\theta > 0$; otherwise we have $f'(\theta) = 0$, which renders the bifurcation value $w^* = (1+k\tau)/\gamma f'(\theta)$ undefined, as well as violating the transversality condition (5). Figure 5 shows the stability boundaries when this firing function is used. Note that this choice of firing rate function makes the product $k\tau$ the main factor in determining the stability of solutions.

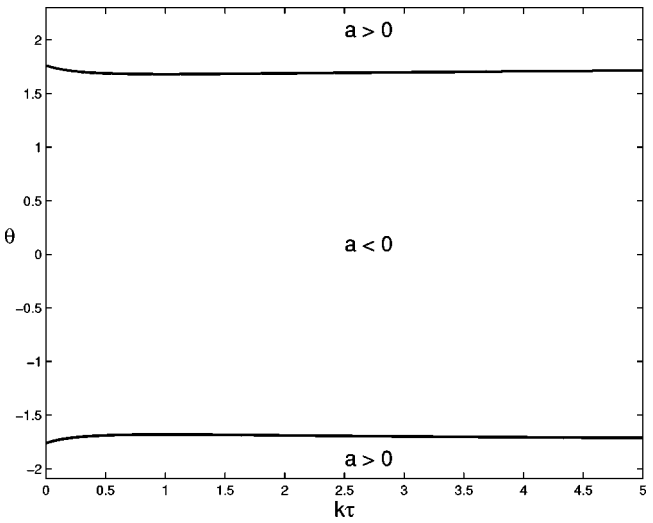

FIG. 3. Stability boundaries for Eqs. (4), using the sigmoidal firing rate function $f_1(q) = 1/[1 + \exp(-q)]$. The periodic solutions arising at the Hopf bifurcation point are stable in the central region ($a<0$), and unstable above and below it ($a>0$).

Numerical simulation has been used to test the algebraic results. [All numerical results have been generated using the sigmoidal firing rate function $f_1(\cdot)$, introduced above.] With $k=\tau=1$, $\gamma=4$, and $\theta=0$, we find $w^*=2$, and $a=-1$. With $w=-w^*-\mu$, the Hopf theorem predicts oscillations with radius $r=\sqrt{-d\mu/a}=\sqrt{-\gamma f'(\theta)\mu/2\tau a}=\sqrt{\mu/2}$. Setting $\mu=0.02$ and integrating Eqs. (6) numerically, we find that the projection of the trajectory onto the z_3-z_4 plane converges to a circle of radius $r=\sqrt{0.02/2}=0.1$, as expected. Note that Hopf bifurcation analysis is strictly local: it tells us that oscillatory solutions will arise in the vicinity of the ori-

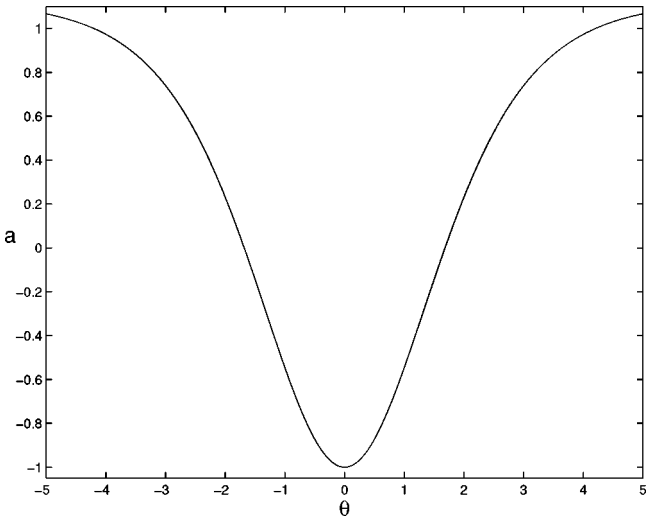

FIG. 4. Stability coefficient a [for sigmoidal firing rate function $f_1(\cdot)$, as in Fig. 3], plotted as a function of parameter θ (the other system parameters are fixed at $\tau=k=1$ and $\gamma=4$). A transition from stable to unstable oscillations occurs at $\theta=\pm 1.68$.

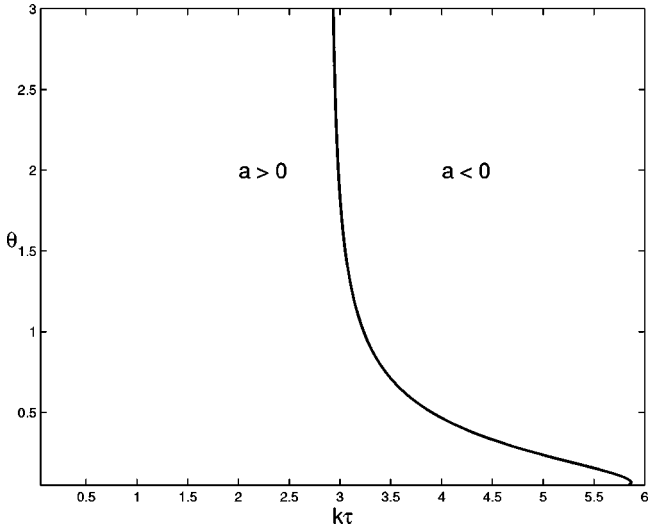

FIG. 5. Stability boundaries for Eqs. (4), using the nonsigmoidal firing rate function $f_2(q)=\Theta(q)/[1+\ln(1+1/q)]$, where $\Theta(q)=1$ if $q>0$, and is zero otherwise. Oscillatory solutions are stable to the right of the boundary line ($a<0$) and unstable to the left ($a>0$).

gin of Eqs. (4) when $|w|$ exceeds w^*. It does not guarantee that oscillatory solutions will *not* occur prior to this point. With $a>0$, a large-magnitude limit cycle appears; this cycle is globally stable for $\mu>0$, while for $\mu<0$ the system becomes multistable, with some solutions converging to the origin and some to the limit cycle. Figure 6 shows such a case. With $a<0$, the simulations indicate that the origin is globally stable for $\mu<0$.

This analysis allows us to choose the system parameters in Eq. (4) to yield the type of oscillatory solutions we desire. Selecting parameters for which $a<0$, and taking μ to be small, we obtain small limit cycles in the vicinity of the fixed

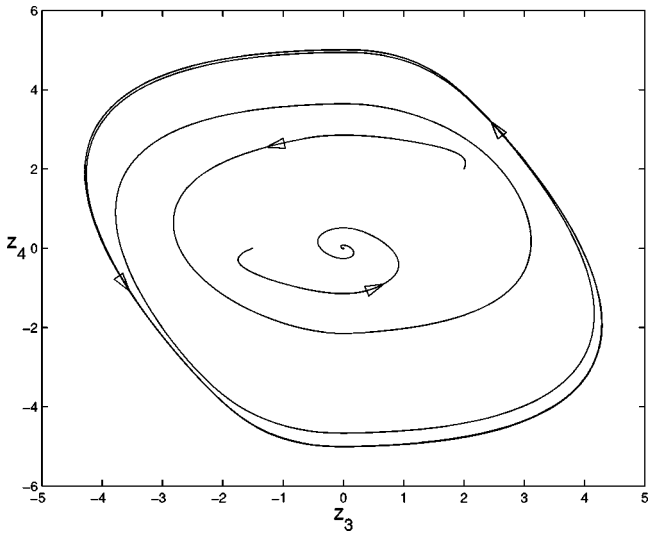

FIG. 6. Trajectories obtained by numerical integration of Eqs. (6), with $\tau=k=1$, $\gamma=4$, and $\theta=-4$. The bifurcation point is $w^*=28.308$, and the stability coefficient is $a=0.973$. We take $w=-w^*-\mu$. The plot shows trajectories (projected onto the z_3-z_4 plane) for $\mu=-15$. Note that the system is multistable, with coexistence between a stable fixed point and a stable limit cycle.

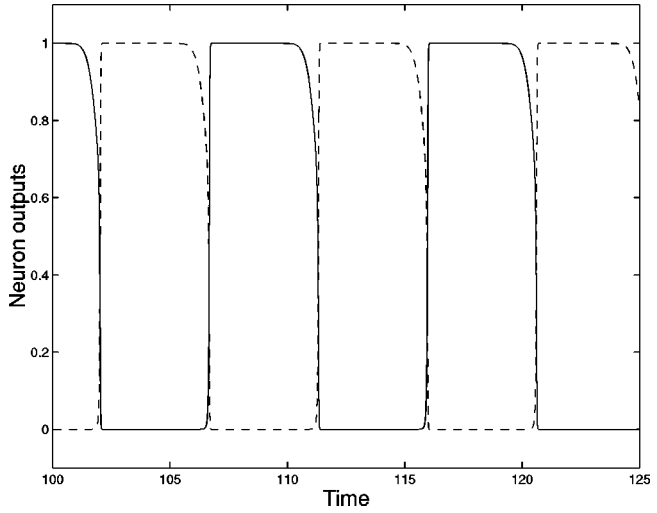

FIG. 7. Numerical integration of Eqs. (4), with $\tau=k=1$, $\gamma=4$, $\theta=-4$, and $w=-w^*-0.02$, where $w^*=28.308$. The plot shows the firing rate outputs $y_i=f(\gamma(\tilde{x}_i-\tilde{\alpha}_i)+\theta)$ for node 1 (solid line) and node 2 (dashed line). As in Fig. 2, the neural outputs may be taken to be fractions of the maximal firing rate. The oscillations seen here correspond to the large-magnitude limit cycle seen in Fig. 6.

point, corresponding to small fluctuations in the base firing rate of the two neurons. With $a>0$ and $\mu>0$, we obtain a large limit cycle in which the two neurons are alternately strongly activated and strongly inhibited, and this is the case we select to represent the half-center CPG. See Fig. 7 for a plot of the outputs of the two nodes for one possible set of system parameters. The transitions here are very sharp, making the network's output resemble a square wave, but we may tune the abruptness of the transitions by changing a; for example, this may be accomplished by varying θ while holding the other parameters fixed.

We have considered only the vicinity of $-w^*$, but another Hopf bifurcation with identical stability properties occurs for $w=w^*+\mu$. The oscillatory solutions arising for this case have the two neurons becoming active in phase with each other, rather than being activated in alternation as in the half-center model.

IV. SUMMARY AND CONCLUSIONS

We have introduced a simple means of adding the qualitative dynamics of neural adaptation to any existing analog (also known as graded-response) neuron model. Using these phasic analog neurons, we have shown that we may model the dynamics of the simplest central pattern generator, the half-center model: two phasic neurons connected in a mutually inhibitory fashion, producing alternating bursts of activity. A Hopf bifurcation analysis shows us the inhibitory strength past which oscillatory solutions will certainly arise, and allows us to produce oscillations of a desired type by tuning the system parameters.

In the absence of neural adaptation, two mutually inhibitory neurons will end up with one neuron fully inhibiting the other, a situation known as ''oscillator death.'' This was discussed, in the context of both analog and integrate-and-fire neural models, Atiya and Baldi [3] and Bressloff and

Coombes [4]. As we have seen in this paper, mutual inhibition can in fact lead to oscillatory behavior in a pair of neurons, provided that the inhibitory effect is of limited duration.

The limited duration of the inhibition is the key to the appearance of oscillatory solutions. Thus, although it may be of interest to consider a more physiologically detailed model of the neural adaptation effect, we would not expect such extensions to affect the existence of the Hopf bifurcation. The precise time course of the adaptation is less significant than the fact that each neuron eventually ceases to fire, allowing the other to become active.

Figures 3 and 5 make it clear that the choice of firing rate function has a significant effect on the behavior of the system. It is not clear that we can describe either $f_1(\cdot)$ or $f_2(\cdot)$ as more biologically realistic for all cases. In attempting to model a particular set of neurons, one should ideally examine the firing rate experimentally and construct a function from that data.

Analog neurons have proven to be a useful tool both in modeling some of the functions of the brain, and in attempting to reproduce animal behavior in the context of robotics and artificial intelligence. The addition of neural adaptation to these models may enhance their usefulness in each of these areas.

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