Class 1 Neural Excitability, Conventional Synapses, Weakly Connected Networks, and Mathematical Foundations of Pulse-Coupled Models

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***Abstract*—Many scientists believe that all pulse-coupled neural networks are toy models that are far away from the biological reality. We show here, however, that a huge class of biophysically detailed and biologically plausible neural-network models can be transformed into a canonical pulse-coupled form by a piece-wise continuous, possibly noninvertible, change of variables. Such transformations exist when a network satisfies a number of conditions; e.g., it is weakly connected; the neurons are Class 1 excitable (i.e., they can generate action potentials with an arbitrary small frequency); and the synapses between neurons are conventional (i.e., axo-dendritic and axo-somatic). Thus, the difference between studying the pulse-coupled model and Hodgkin–Huxley-type neural networks is just a matter of a coordinate change. Therefore, any piece of information about the pulse-coupled model is valuable since it tells something about all weakly connected networks of Class 1 neurons. For example, we show that the pulse-coupled network of identical neurons does not synchronize in-phase. This confirms Ermentrout's result that weakly connected Class 1 neurons are difficult to synchronize, regardless of the equations that describe dynamics of each cell.**

***Index Terms*—Canonical model, class 1 neural excitability, conventional synapses, desynchronization, integrate-and-fire, saddle-node on limit cycle bifurcation, weakly connected neural networks.**

I. Introduction

MANY scientists believe that pulse-coupled neural networks are toy models; that is, even though they are based on abstractions of important properties of biological neurons, they are still far away from the reality (despite the fact that we have no idea what the reality is). As a consequence, all results obtained by studying pulse-coupled neural networks might be irrelevant to the brain.

There are many pulse-coupled models. Among the four considered in this paper, the simplest one has the form

$$\varphi_i' = \omega_i + (1+\cos\varphi_i)\sum_{j=1}^{n} s_{ij}\delta(\varphi_j - \pi)$$

where $\varphi_i \in \mathbb{S}^1$ is the phase variable that represents activity of the ith neuron, $\mathbb{S}^1 = \{e^{i\varphi} \in \mathbb{C}\}$ is the unit circle, $\omega_i > 0$ is the frequency, $-1 < s_{ij} < 1$ is the synaptic coefficient, and δ is the Dirac delta function. The jth neuron fires when φ_j crosses π; at this moment it increments activity of the ith neuron by $(1+\cos\varphi_i)s_{ij}$. The term $1+\cos\varphi_i$ takes into account the absolute and relative refractory period, since $1+\cos\varphi_i \approx 0$ after φ_i fired (crossed π).

This pulse-coupled model, as well as models (7), (10), and (11) below, has a universal property: a huge class of biologically plausible and biophysically detailed neural models taking into account dynamics of all ions, channels, pumps, etc., can be converted to this model by a suitable piece-wise continuous change of variables provided that certain conditions are satisfied. Thus, the question whether or not the pulse-coupled model above is close to biological reality is replaced by the question whether or not the conditions are biologically plausible.

The present paper is devoted to discussion of these conditions. Among them the most important are the following.

1) Neurons are Class 1 excitable, which implies that the neuron activity is near a transition from quiescent state to periodic spiking, and the emerging spiking can have an arbitrary small frequency. If we consider codimension 1 bifurcations, then such a transition corresponds to saddle-node bifurcation on a limit cycle, but not to an Andronov–Hopf bifurcation.
2) Neurons are weakly connected, which follows from the *in vitro* observation that the amplitudes of postsynaptic potentials (around 0.1 mV) are much smaller than the amplitude of an action potential (around 100 mV), or the mean EPSP size necessary to discharge a silent cell (around 20 mV).
3) Synaptic transmission has an intermediate rate, which is slower than the duration of an action potential, but faster than the interspike period.
4) Synaptic connections between neurons are of conventional type, which implies that the synapses under consideration must be either axo-dendritic or axo-somatic, but they cannot be axo-axonic or dendro-dendritic.
5) Synaptic transmission is negligible when presynaptic neurons are at rest; that is, spontaneous release of neurotransmitter does not affect significantly spiking of postsynaptic neurons.

Mathematical technique to study Class 1 excitable systems was developed by Ermentrout and Kopell [2] who studied parabolic bursters. Later Ermentrout [3] used assumption 2) to analyze rigorously the behavior of two weakly connected Class 1 neurons. He confirmed numerical results of Hansel *et al.* [4]

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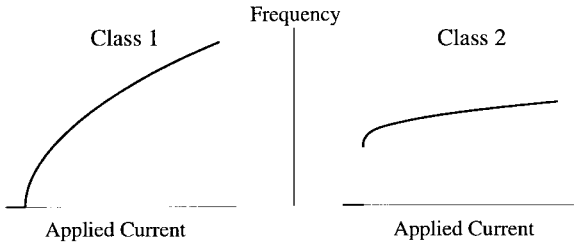

Fig. 1. Dependence of frequency of oscillations on the strength of applied current (the parameter ρ_x) in the Wilson–Cowan model (from [6]).

that Class 1 neurons are difficult to synchronize. In the present paper we use the other assumptions to extend Ermentrout's result to networks of *many* neurons. We achieve this goal by showing that an arbitrary neural network satisfying the conditions above can be transformed to a pulse-coupled canonical model by a suitable change of variables (such a pulse-coupled model was not written explicitly by Ermentrout [3], but was used implicitly when he employed the phase-resetting curve arguments). Then we show that in-phase dynamics of such a pulse-coupled network spontaneously desynchronize, which implies that desynchronization is a genuine attribute of Class 1 neurons that is relatively independent of the equations that describe the neuron activities.

We cannot afford to render the proofs here, since they involve invariant manifold reduction and a number of singular transformations. Thus, we refer a mathematically oriented reader to the book by Hoppensteadt and Izhikevich [6], who provide the necessary background information and the proofs.

II. The Assumptions

A. Neural Excitability

There are two phenomena associated with the generation of action potentials by neurons—neural excitability and transition from rest to periodic spiking activity. The former is a single response to external perturbations, the latter is a qualitative change in dynamic behavior. The type of neural excitability depends intimately on the type of bifurcation from quiescent to oscillatory activity. To classify the spiking mechanism, we must identify the bifurcation.

To study the transition from rest to periodic spiking, Hodgkin [5] performed the following experiment. He applied a weak current of increasing magnitude. When the current became strong enough, the neuron started to generate spikes repetitively with a certain frequency. Hodgkin suggested the following classification (see Fig. 1).

- *Class 1 Neural Excitability:* Action potentials can be generated with arbitrarily low frequency, depending on the strength of the applied current.
- *Class 2 Neural Excitability:* Action potentials are generated in a certain frequency band that is relatively insensitive to changes in the strength of the applied current.

Class 1 neurons in Hodgkin experiments fired with a frequency that varied smoothly over a range of about 5 to 150 Hz. The frequency band of the Class 2 neurons was usually

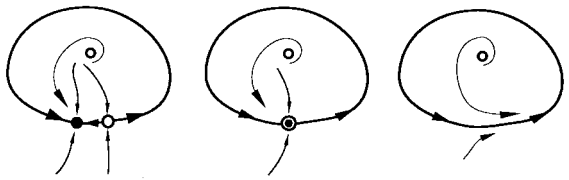

Fig. 2. Saddle-node bifurcation on a limit cycle (from [6]).

75 to 150 Hz, but it could vary from neuron to neuron. The exact numbers are not important to us here. The qualitative distinction between Class 1 and 2 neurons is that the emerging oscillations have zero frequency in the former and nonzero frequency in the latter. This is due to different bifurcation mechanisms.

Let us consider the strength of applied current in Hodgkin's experiments as being a bifurcation parameter. When the current increases, the rest potential bifurcates, which results in its loss of stability or disappearance, and the neuron activity becomes oscillatory. The bifurcation resulting in transition from a quiescent to an oscillatory state determines the class of neural excitability. Since there are only two codimension 1 bifurcations of stable equilibria, it is not surprising that [6], [10]:

- *Class 1:* Neural excitability is observed when a rest potential disappears by means of a *saddle-node bifurcation*, also known as fold bifurcation.
- *Class 2:* Neural excitability is observed when a rest potential loses stability via the *Andronov–Hopf bifurcation.*

The saddle-node bifurcation may or may not be on a limit cycle. The Andronov-Hopf bifurcation may be either subcritical or supercritical. The bifurcational mechanism describing spiking activity must explain not only the appearance but also the disappearance of periodic spiking activity when the applied current is removed. This imposes some additional restrictions on the bifurcations, which are scrutinized in [6].

In this paper we consider Class 1 neural excitability that arises when the rest potential is near a saddle-node bifurcation on a limit cycle, which is also referred to as being saddle-node bifurcation on an invariant circle; see Fig. 2. Looking at the figure from right to left suggests the mechanism of disappearance of the periodic spiking activity. The saddle-node bifurcation on a limit cycle explains both appearance and disappearance of oscillatory activity, and no further assumptions are required.

Saddle-node bifurcations on limit cycles are ubiquitous in two-dimensional systems

$$\begin{cases} \dot{x} = f(x, y) \\ \dot{y} = g(x, y). \end{cases}$$

Let us plot the nullclines $\dot{x} = 0$ and $\dot{y} = 0$ on the xy-plane. Each intersection of the nullclines corresponds to an equilibrium of the model. When the nullclines intersect as in Fig. 3, the bifurcation occurs. The phase portrait in the figure is similar to the one in Fig. 2.

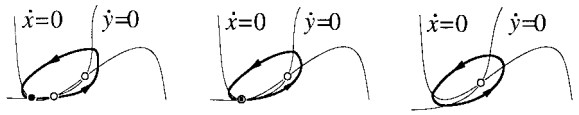

Fig. 3. Saddle-node bifurcation on the limit cycle in Wilson–Cowan model (from [2] and [6]).

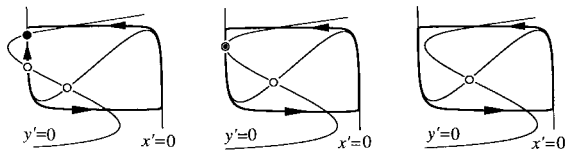

Fig. 4. Wilson–Cowan relaxation oscillator exhibiting saddle-node bifurcation on the limit cycle (from [6]).

Saddle-node bifurcation on a limit cycle can also be observed in relaxation systems of the form

$$\begin{cases} x' = f(x, y) \\ y' = \mu g(x, y), \end{cases} \qquad \mu \ll 1$$

having nullclines intersected as in Fig. 4. Again, the phase portrait of such a system is qualitatively similar to the one depicted in Figs. 2 and 3.

Saddle-node bifurcation on a limit cycle leading to Class 1 neural excitability can be observed in many multidimensional biophysically detailed systems of Hodgkin–Huxley type, such as Connor [1] and Morris–Lecar [9] models (see [3]). Many believe that majority of cortical neurons in mammals are of Class 1 (B. Ermentrout, personal communication). Incidentally, the Hodgkin–Huxley model exhibits Class 2 neural excitability for the original values of parameters. It may though exhibit Class 1 excitability when a transient potassium A-current is taken into account [11].

The Ermentrout–Kopell Canonical Model: Consider a system of the form

$$\dot{X} = F(X, \lambda) \tag{1}$$

describing dynamics of a neuron, where variable X describes its activity (e.g., membrane potential and activity of all ions, currents, channels, pumps, etc.), and λ is a vector of parameters. Since we are far away from understanding all details of neuron dynamics, we do not have detailed information about the function F. Moreover, we do not even know what the dimension of X is.

It is a great challenge to study (1), but much progress can be achieved when (1) has Class 1 neural excitability, i.e., there is a saddle-node bifurcation on a limit cycle for some λ_0. In this case we can use the Ermentrout–Kopell theorem [6, Th. 8.3] combined with the invariant manifold reduction [6, Th. 4.2] to find a continuous noninvertible change of variables $(\varphi, r) = h(X, \lambda)$, which exists for all λ near λ_0, that transforms *every* system of the form (1) to the Ermentrout–Kopell canonical model

$$\varphi' = (1 - \cos\varphi) + (1 + \cos\varphi) r \tag{2}$$

where $\varphi \in \mathbb{S}^1$ is a phase variable that describes activity of the neuron along the limit cycle, $\mathbb{S}^1 = \{e^{i\varphi} \in \mathbb{C}\}$ is the unit circle,

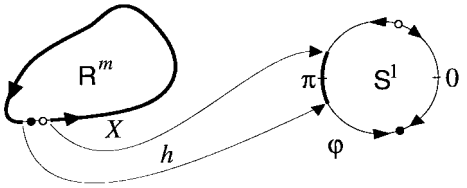

Fig. 5. The transformation h maps solutions of (1) to those of (2).

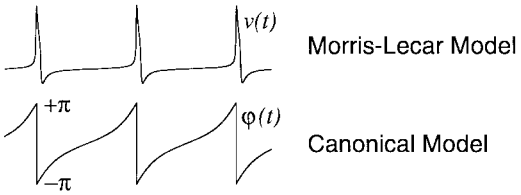

Fig. 6. Spiking activities of the Morris–Lecar system (see [3]) and the Ermentrout–Kopell canonical model (2) are related as $\varphi(t) = h(v(t))$ for some function h.

and $r \in \mathbb{R}$ is a new bifurcation parameter. Particulars of the function F in (1) do not affect the form of the canonical model (2), but affect only the value of the parameter r. The advantage of the canonical model (2) for neuroscience applications is that its studying sheds some light on *all* neuron models, even those that have not been invented yet. The disadvantage of the canonical model (2) is that the change of variables, h, is guaranteed to exist when (1) is near the saddle-node on limit cycle bifurcation; that is, when $|\lambda - \lambda_0|$ is small. Since the period of spiking is proportional to $1/\sqrt{|\lambda - \lambda_0|}$ in this case ([3], see also [6, Proposition 8.4]), proximity to the bifurcation implies that *the neuron under consideration fires with a very large interspike period.*

The transformation h that maps solutions of (1) to those of (2) blows up a small neighborhood of the saddle-node bifurcation point and compresses the entire limit cycle to an open set around $\pi \in \mathbb{S}^1$; see Fig. 5. Thus, when X makes a rotation around the limit cycle (generates a spike), the canonical variable φ crosses a tiny open set around π. Since the points π and $-\pi$ are equivalent on the unit circle $\mathbb{S}^1$, every time φ crosses π, it is reset to $-\pi$. In this case the activity $\varphi(t)$ treated as a variable from $\mathbb{R}$ has discontinuities that may look like spikes too; see Fig. 6.

The canonical model (2) has the following property: If $r > 0$, the neuron fires repeatedly with the period $\pi/\sqrt{r}$. If $r < 0$, then there is a rest state (stable equilibrium) φ^- and a threshold state (unstable equilibrium) φ^+ given by

$$\varphi^{\pm} = \pm\cos^{-1}\frac{1+r}{1-r}.$$

If $\varphi(0)$ is near the rest state φ^-, it converges to the rest state. If $\varphi(0)$ is perturbed so that it crosses the threshold value φ^+, it makes a rotation (fires a spike) and returns to the rest state φ^-; see Fig. 7.

The parameter r in the canonical model is a bifurcation parameter. When r crosses the bifurcation value $r = 0$, the behavior of the canonical model, and hence the original system (1), changes from excitability to periodicity. When $r \neq 0$, we

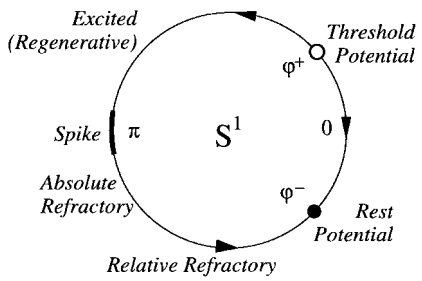

Fig. 7. Physiological state diagram of a Class 1 neuron (from [6]).

can use the change of variables

$$\vartheta = 2\text{atan}\frac{1}{\sqrt{|r|}}\tan\frac{\varphi}{2} \tag{3}$$

to transform the canonical model into one of the following simple forms:

$$\vartheta' = -\omega\cos\vartheta \quad \text{(excitable activity; } r<0\text{)}$$
$$\vartheta' = \omega \quad \text{(periodic activity; } r>0\text{)}$$

where $\omega = 2\sqrt{|r|}$ is a positive parameter. The transformation (3) justifies the empirical observation that the behavior of the Ermentrout–Kopell canonical model (2) for negative r is equivalent to that for $r=-1$; and for positive r is equivalent to that for $r=+1$.

The canonical model (2) is probably the simplest excitable system known to mathematical neuroscience: It is one-dimensional; it has Class 1 neural excitability or periodic activity; and it is biologically plausible in the sense that any other Class 1 excitable neuro-system can be converted to the form (2) by an appropriate change of variables. Many other examples of canonical models can be found in the book by Hoppensteadt and Izhikevich [6].

B. Weakly Connected Neural Networks

Little is known about dynamics of biological neurons, even less about networks of such neurons. A promising approach is to take advantage of the fact that neurons are weakly connected. Such neural networks can be written in the form

$$\dot{X}_i = F_i(X_i, \lambda) + \varepsilon G_i(X_1, \cdots, X_n, \lambda, \varepsilon) \tag{4}$$

where each X_i describes activity of the ith neuron, the function G_i describes how the ith neuron is affected by the other neurons, and $\varepsilon \ll 1$ is a small dimensionless parameter that denotes weakness of connections. Neurophysiological justification of the assumption of weakness of connections is based on the *in vitro* observation that amplitudes of postsynaptic potentials (PSP's) are around 0.1 mV, which is small in comparison with the amplitude of an action potential (around 100 mV) and the amplitude of the mean EPSP necessary to discharge a quiescent cell (around 20 mV); see detailed discussion by Hoppensteadt and Izhikevich [6, Sec. 1.3] who obtained an estimate

$$0.004 < \varepsilon < 0.008$$

for a model of hippocampal granule cells using *in vitro* data [8].

C. Rate of Synaptic Transmission

Sometimes it is convenient to distinguish relatively fast processes related to the generation of an action potential, and relatively slow processes related to the synaptic transmission. For this, we can write a weakly connected neural network in the form

$$\dot{x}_i = f_i(x_i, \lambda) + \varepsilon g_i(c_1, \cdots, c_n, \lambda, \varepsilon)$$
$$\dot{c}_i = p_i(x_i, c_i, \lambda)$$

where each x_i describes generation of action potential by the ith neuron, and each vector c_i describes the processes taking place at its synaptic terminals. When x_i is quiescent, c_i converges to a stable equilibrium corresponding to the "no transmission" state. The rate of convergence γ_i is the absolute value of the largest real part of the eigenvalues of the Jacobian matrix $J = D_{c_i}p_i$ at the equilibrium. (If c_i were a scalar, then $\gamma_i = -\partial p_i/\partial c_i$.) The smaller the rate γ_i, the slower the synaptic transmission is. If $\gamma_i = \mathcal{O}(\sqrt{|\lambda-\lambda_0|})$, as was implicitly assumed in [3], then the synaptic transmission may take as much time as an interspike interval.

In this paper we assume that γ_i is a relatively small constant that is independent of $|\lambda-\lambda_0|$. This results in synaptic transmission having an intermediate rate: It is slower than the duration of a spike, but faster than the interspike interval; see the upper part of Fig. 11 for an illustration.

Let us denote $X_i = (x_i, c_i)$ and rewrite the system above in the form (4), where $F_i = (f_i, p_i)$ and $G_i = (g_i, 0)$. When each γ_i is a constant, the system $\dot{x}_i = f_i(x_i, \lambda)$ is at a saddle-node bifurcation on a limit cycle if and only if the system $\dot{X}_i = F_i(X_i, \lambda)$ is (this is not valid when $\gamma_i = \mathcal{O}(\sqrt{|\lambda-\lambda_0|})$). Thus, without loss of generality, we may consider weakly connected neural networks of the form (4) in our analysis below.

D. Conventional Synaptic Connections

It is a great challenge to study weakly connected neural networks of the form (4) since we have no information about the functions G_i. A plausible auxiliary assumption is that the connection functions G_i have the pairwise coupled form

$$G_i(X_1, \cdots, X_n, \lambda, \varepsilon) = \sum_{j=1}^{n} G_{ij}(X_i, X_j, \lambda) + \mathcal{O}(\varepsilon)$$

which is equivalent to the requirement that the synaptic connections between neurons be conventional [12]; see Fig. 8. Obviously, if this requirement is violated, then synaptic transmission between two neurons cannot be considered as a process that is independent from activities of the other neurons. For example, transmission via inhibitory axo-dendritic synapse in Fig. 9 can be shut down by the inhibitory axo-axonic synapse.

E. Spontaneous Transmitter Release

Let $X_i = 0$ denote the state when the neuron membrane potential is at rest. The behavior of the network depends crucially on the value $G_{ij}(0, 0)$. The case $G_{ij}(0, 0) \neq 0$ corresponds to the spontaneous release of a neurotransmitter even when the

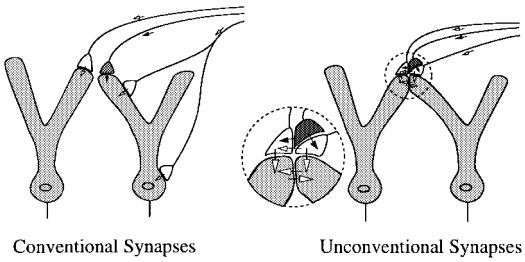

Fig. 8. Axo-dendritic and axo-somatic synapses are conventional. Axo-axonic and dendro-dendritic synapses are unconventional (from [6]).

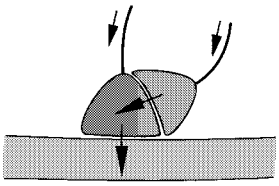

Fig. 9. Transmission of the conventional inhibitory synapse can be shut down by an unconventional axo-axonic inhibitory synapse (from [6]).

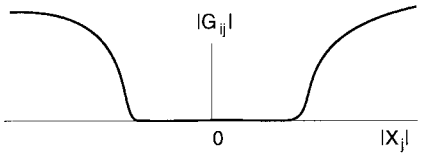

Fig. 10. The function $G_{ij}(X_i, X_j) = 0$ for all X_j near zero.

presynaptic neuron is quiescent. Such a release always exists in biological neurons [12] due to its the stochastic nature.

In this paper we assume that $G_{ij}(X_i, X_j) = 0$ for all X_j from a small neighborhood of the origin; see Fig. 10. We interpret this as the following: The spontaneous release of neurotransmitter is negligible when the neurons are silent. Moreover, small wobbling of the membrane potential of the presynaptic neuron does not affect dynamics of the postsynaptic one. Thus, to evoke any postsynaptic response, the presynaptic neuron must generate a spike.

III. The Canonical Model

The following theorem follows from Theorem 8.11 and Proposition 8.12 by Hoppensteadt and Izhikevich [6]. Its proof involves invariant manifold reduction and a number of singular transformations.

Theorem 1: Consider an arbitrary weakly connected neural network of the form

$$\dot{X}_i = F_i(X_i, \lambda) + \varepsilon G_i(X_1, \cdots, X_n, \lambda, \varepsilon) \tag{5}$$

satisfying the assumptions discussed in Section II. That is, each equation $\dot{X}_i = F_i(X_i, \lambda)$ undergoes a saddle-node bifurcation on a limit cycle for some $\lambda = \lambda_0$. Each function G_i has the pair-wise connected form

$$G_i(X_1, \cdots, X_n, \lambda_0, 0) = \sum_{j=1}^{n} G_{ij}(X_i, X_j)$$

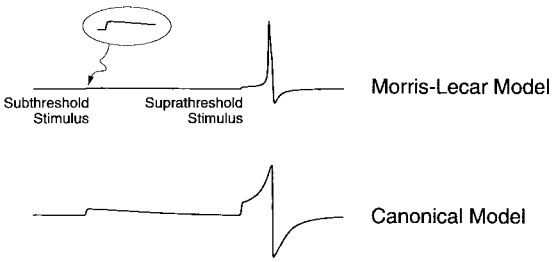

Fig. 11. Response of a postsynaptic neuron to spiking of presynaptic neurons. First spike produces a subthreshold EPSP, second spike produces a suprathreshold EPSP. Simulations are performed for $\varepsilon = 0.1$.

and each $G_{ij}(X_i, X_j) = 0$ for X_j from some open neighborhood of the saddle-node bifurcation point. Then, there is $\varepsilon_0 > 0$ such that for all $\varepsilon < \varepsilon_0$ and all $\lambda = \lambda_0 + \mathcal{O}(\varepsilon^2)$ there is a piece-wise continuous transformation that maps solutions of (5) to those of the canonical model of the form

$$\varphi_i' = (1 - \cos\varphi_i) + (1 + \cos\varphi_i) r_i + \sum_{j=1}^{n} w_{ij}(\varphi_i)\delta(\varphi_j - \pi) + \mathcal{O}(\sqrt{\varepsilon}\,\ln\,\varepsilon)$$

where $' = d/d\tau$, $\tau = \varepsilon t$ is the slow time, δ is the Dirac delta function satisfying $\delta(y) = 0$ if $y \neq 0$, $\delta(0) = \infty$, and $\int \delta = 1$. Each function w_{ij} has the form

$$w_{ij}(\varphi_i) = 2\mathrm{atan}\Big(\tan\frac{\varphi_i}{2} + s_{ij}\Big) - \varphi_i \tag{6}$$

and each s_{ij} is a constant, which is proportional to $|G_{ij}|$.

It should be stressed that quantitative behavior of (5) and the canonical model above may differ (as in Fig. 11), but qualitatively they are the same in the sense that the latter is obtained from the former by a piece-wise continuous change of variables. The particulars of the functions F_i and G_{ij} do not affect the form of the canonical model, but affect only the values of the parameters r_i and s_{ij}.

Remark 2—B. Ermentrout, Personal Communication: Let us drop the assumption that the synaptic transmission rate is intermediate, and assume that it is as slow as the interspike period. For example, we may take $p_i(x_i, c_i, \lambda) = q(x_i) - \varepsilon c_i$ (see Section II-C), which corresponds to fast rise but slow decay of synaptic transmission. Then the canonical model would have the form

$$\varphi_i' = (1 - \cos\varphi_i) + (1 + \cos\varphi_i)(r_i + c_i)$$
$$c_i' = \sum_{j=1}^{n} s_{ij}\delta(\varphi_j - \pi) - c_i$$

which is very close to what is studied in [3].

A. The Pulse-Coupled Model

The canonical model in Theorem 1 has a small remainder $\mathcal{O}(\sqrt{\varepsilon}\ln\varepsilon)$ that smooths the pulses; see Fig. 12. Since $\varepsilon \ll 1$, we drop the remainder in what follows. Thus, we study a pulse-coupled neural network of the form

$$\varphi_i' = (1 - \cos\varphi_i) + (1 + \cos\varphi_i) r_i + \sum_{j=1}^{n} w_{ij}(\varphi_i)\delta(\varphi_j - \pi). \tag{7}$$

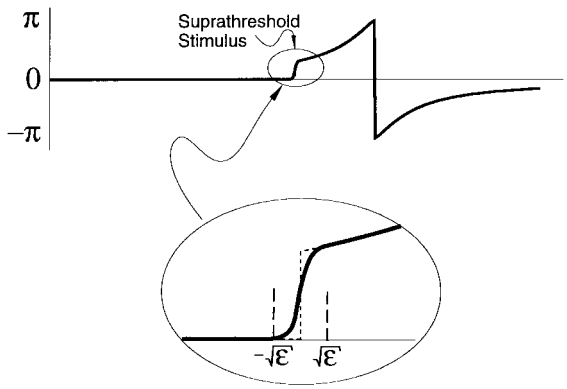

Fig. 12. Solutions of the pulse-coupled model with (continuous curve) and without (dotted curve) the small remainder $\mathcal{O}(\sqrt{\varepsilon}\ln\varepsilon)$. Simulations are performed for $\varepsilon = 0.1$.

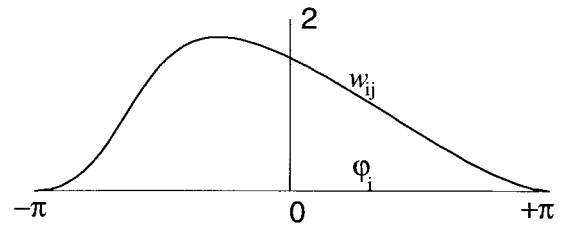

Fig. 13. Graph of the function $w_{ij}(\varphi_i) = 2a\tan(\tan\varphi_i/2 + s_{ij}) - \varphi_i$ for $s_{ij} = 1$.

We see that each neuron is governed by the Ermentrout–Kopell canonical model

$$\varphi_i' = (1-\cos\varphi_i) + (1+\cos\varphi_i)r_i.$$

When $\varphi_j \in \mathbb{S}^1$ crosses π (fires a spike), the value of φ_i is incremented by $w_{ij}(\varphi_i)$, which depends on the state of the ith neuron. A typical shape of w_{ij} is depicted in Fig. 13 (for positive s_{ij}). The neurons interact by a simple form of pulse coupling: When φ_j fires, it resets φ_i to the new value

$$\varphi_i^{\text{new}} = 2\text{atan}\left(\tan\frac{\varphi_i^{\text{old}}}{2} + s_{ij}\right) \tag{8}$$

which depends on the current activity φ_i^{old}. This might be easier to analyze when we rewrite (8) in the form

$$\tan\frac{\varphi_i^{\text{new}}}{2} = \tan\frac{\varphi_i^{\text{old}}}{2} + s_{ij}. \tag{9}$$

The variable φ_i integrates many such inputs from other neurons, and φ_i fires when it crosses π, hence the name *integrate-and-fire*. One can treat $\varphi_i \in \mathbb{S}^1$ as being the phase of the ith oscillator. In this case the function w_{ij} is the *phase resetting curve*. When connections between neurons are excitatory, i.e., when $s_{ij} > 0$, then w_{ij} is nonnegative, and firing of the jth neuron could only advance that of the ith one. Similarly, when the connections are inhibitory, $s_{ij} < 0$, then $w_{ij} \le 0$, and firing of the jth neuron can never advance that of the ith one.

B. Simplification of the Pulse-Coupled Model

The pulse-coupled model can be simplified when the parameters $|s_{ij}|$ are small. Indeed, the Taylor series of each w_{ij} as a function of s_{ij} has the initial portion

$$w_{ij}(\varphi_i) = 0 + s_{ij}(1+\cos\varphi_i) + \mathcal{O}(s_{ij}^2).$$

Therefore, the pulse-coupled model can be written in the form

$$\varphi_i' = (1-\cos\varphi_i) + (1+\cos\varphi_i)\left(r_i + \sum_{j=1}^{n} s_{ij}\delta(\varphi_j - \pi)\right) \tag{10}$$

plus small terms of order $\mathcal{O}(s^2)$. This system is easier to analyze and faster to simulate, but caution should be used when s_{ij} have intermediate values, since the terms $\mathcal{O}(s_{ij}^2)$ may not be negligible in this case. To summarize, one should use the original pulse-coupled model (7) whenever possible, and simplified model (10) only when there is a need to speed up simulations.

If the bifurcation parameters r_i are all fixed and nonzero, we may use the transformation (3) to simplify the pulse-coupled system above even further. When all r_i are negative, then we have the following canonical pulse-coupled network of excitable neurons:

$$\vartheta_i' = -\omega_i\cos\vartheta_i + (1+\cos\vartheta_i)\sum_{j=1}^{n} c_{ij}\delta(\vartheta_j - \pi) \tag{11}$$

where $\omega_i = 2\sqrt{|r_i|}$ and $c_{ij} = \sqrt{|r_i|}s_{ij}$. If the bifurcation parameters r_i are all positive, then we have the following canonical network of pulse-coupled oscillators:

$$\vartheta_i' = \omega_i + (1+\cos\vartheta_i)\sum_{j=1}^{n} c_{ij}\delta(\vartheta_j - \pi). \tag{12}$$

Obviously, if r_i had different signs, we would have a mixture of the canonical models above.

Remark 3: Notice that the standard assumption of integrate-and-fire models, viz., that the postsynaptic membrane potential is incremented by a constant value due to each firing of a presynaptic neuron, is not valid for Class 1 excitable neurons due to the term $(1+\cos\vartheta_i)$. The term takes into account the absolute and relative refractory periods. Indeed, after the neuron fires a spike; that is, ϑ_i crosses π, the term $(1+\cos\vartheta_i)$ is small meaning that the neuron is not sensitive to the spikes converging from the other neurons at that time.

Let us find out when the synaptic parameters $|s_{ij}|$ in (7) are small. Surprisingly, this is related to the question: "Why do we require that the distance to the saddle-node bifurcation, $|\lambda - \lambda_0|$, in the Theorem 1 be of order ε^2?" Suppose it is not, then we rewrite the weakly connected network (5) in the form

$$\dot{X}_i = F_i(X_i, \lambda) + \sqrt{|\lambda-\lambda_0|}\tilde{G}_i(X_1, \cdots, X_n, \lambda, \varepsilon)$$

where $\tilde{G}_i = \varepsilon/\sqrt{|\lambda-\lambda_0|}G_i$. Formal application of Theorem 1 yields the pulse-coupled canonical model in which s_{ij} are proportional to $\varepsilon/\sqrt{|\lambda-\lambda_0|}$. Therefore, s_{ij} is small when $|\lambda - \lambda_0| \gg \varepsilon^2$.

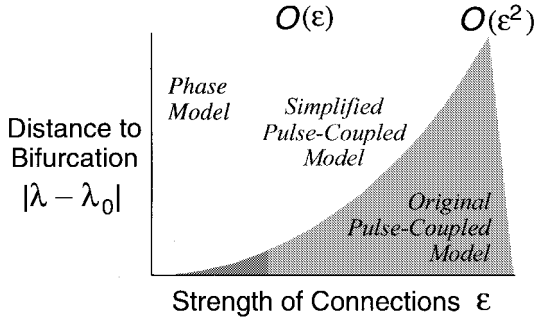

Fig. 14. A weakly connected network of Class 1 excitable neurons can be converted into (original) pulse-coupled model (7) when $|\lambda-\lambda_0| = \mathcal{O}(\varepsilon^2)$, into (simplified) model (10) when $\varepsilon^2 \ll |\lambda-\lambda_0| \ll \varepsilon$, or into the phase model (13) when $|\lambda-\lambda_0| \gg \varepsilon/(\ln\varepsilon)^2$.

Notice that each s_{ij} has the lower limit $s_{ij} \gg \sqrt{\varepsilon}\ln\varepsilon$, which guarantees that the connection term in (10) has more weight than the small remainder $\mathcal{O}(\sqrt{\varepsilon}\ln\varepsilon)$ that we neglected. Therefore $|\lambda-\lambda_0| \ll \varepsilon/(\ln\varepsilon)^2$ and we obtain the range

$$\varepsilon^2 \ll |\lambda-\lambda_0| \ll \frac{\varepsilon}{(\ln\varepsilon)^2}$$

in which the usage of the simplified pulse-coupled model (10) is justified; see summary in Fig. 14.

C. The Phase Model

When $\varepsilon \ll |\lambda-\lambda_0|$; that is, when the strength of connections is much weaker than the distance to the bifurcation, the pulse-coupled model (10) becomes uncoupled (up to the next order in ε). In this case each neuron is either quiescent (when $r_i < 0$) or a pacemaker (when $r_i > 0$) regardless of activities of the other neurons [6, Corollary 8.9]. The latter case is interesting since we can study various synchronization phenomena in a network of such pacemakers. For example, if all neurons have nearly identical frequencies, then we may apply the Malkin Theorem [6, Th. 9.2] to the weakly connected oscillatory system (5) to convert it to the canonical (phase) model

$$\varphi_i' = \omega_i + \sum_{j=1}^{n} s_{ij} H(\varphi_j - \varphi_i), \quad H(\chi) = 1-\cos\chi \qquad (13)$$

plus high-order terms in $|\lambda-\lambda_0|$. The form of the connection function H was obtained numerically by Golomb and Hansel (personal communication) for a particular neural model, and it is canonical for an arbitrary weakly connected network of Class 1 neurons having periodic activity [7]. A short way to see this is to assume that all ω_i are equal and all c_{ij} are small in the system (12). In this case the phase model (13) is a direct consequence of [6, Th. 9.12].

IV. Synchronization Versus Desynchronization

Consider a network of identical pulse-coupled neurons

$$\varphi_i' = (1-\cos\varphi_i) + (1+\cos\varphi_i)r + \sum_{j=1}^{n} w_{ij}(\varphi_i)\delta(\varphi_j-\pi)$$

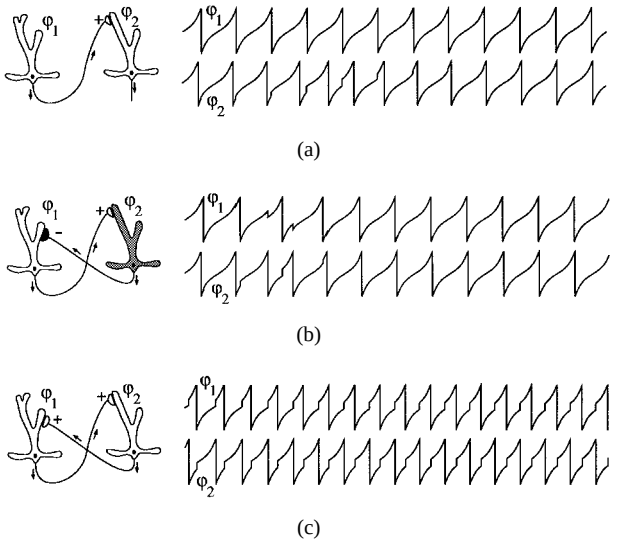

Fig. 15. Various synaptic organizations and corresponding behavior of the pulse-coupled model.

for $r > 0$. Since $w_{ij}(\pi) = 0$, it is easy to see that the network always has an in-phase synchronized solution

$$(\varphi_1(\tau), \cdots, \varphi_n(\tau)) = (\phi(\tau), \cdots, \phi(\tau))$$

where $\phi(\tau)$ is the periodic solution to the equation

$$\phi' = (1-\cos\phi) + (1+\cos\phi)r.$$

Let us study the stability of the in-phase solution for the case $n = 2$ and $n > 2$.

A. Two Neurons

Consider a network of two identical neurons connected in one direction, say from φ_1 to φ_2 [see Fig. 15(a)]

$$\varphi_1' = (1-\cos\varphi_1) + (1+\cos\varphi_1)r$$
$$\varphi_2' = (1-\cos\varphi_2) + (1+\cos\varphi_2)r + w(\varphi_2)\delta(\varphi_1-\pi).$$

Let us perturb the in-phase synchronized solution $\varphi_1(\tau) = \varphi_2(\tau)$ by assuming that $\varphi_2 > \varphi_1$; that is, the second oscillator is slightly ahead of the first one. Since $w \geq 0$, firings of φ_1 advance φ_2 even further, which means that the in-phase solution is unstable. After a while, $\varphi_2(\tau) \to \varphi_1(\tau) + 2\pi$, and each firing of φ_1 advances φ_2 even closer to $\varphi_1(\tau) + 2\pi \equiv \varphi_1(\tau)$, which may look like the neurons are trying to synchronize again. We see that the in-phase synchronized solution for the synaptic organization in Fig. 15(a) is stable in one direction and unstable in the other. The pulse-coupled model is at double limit cycle bifurcation [see 6, Sec. 2.7.3]. This occurs because we consider two *identical* neurons, which is not a generic situation. If we allow them to be slightly different, then there is no synchronized solution when $r_1 < r_2$ (i.e., when φ_1 is slower than φ_2), and there is a nearly in-phase synchronized solution with a small phase shift when $r_1 > r_2$. The shift increases, though, when $r_1 - r_2$ increases. Similar considerations are applicable to the case when φ_1 is an inhibitory neuron.

Now consider the synaptic organization in Fig. 15(b). Such neurons are governed by the system

$$\varphi_1' = (1-\cos\varphi_1) + (1+\cos\varphi_1)r + w_{12}(\varphi_1)\delta(\varphi_2-\pi)$$
$$\varphi_2' = (1-\cos\varphi_2) + (1+\cos\varphi_2)r + w_{21}(\varphi_2)\delta(\varphi_1-\pi)$$

where $w_{12} \le 0$ (inhibitory synapse) and $w_{21} \ge 0$ (excitatory synapse). If $\varphi_2 > \varphi_1$, then firing of φ_1 advances φ_2, and firing of φ_2 delays φ_1 so that the distance between them increases. Both synapses contribute to instability of the in-phase synchronized solution in one direction and its stability in the other. Qualitative behavior of such a network is similar to the one considered above.

So far our arguments were similar to those in [3]. They were based on the fact that the phase-resetting curve (function w_{ij}) does not change sign. This was enough for synaptic organizations in Fig. 15(a) and (b), but it is not enough for the synaptic organization in Fig. 15(c). Indeed, a firing of φ_1 pushes φ_2 and increases $\varphi_2-\varphi_1$, but a subsequent firing of φ_2 pulls φ_1 and decreases $\varphi_2-\varphi_1$. To determine stability of the in-phase solution we must take into account the relative sizes of those pushes and pulls. In the next section we show that the sizes are exactly the same if $s_{ij} = s_{ji}$. This implies that the in-phase as well as any out-of-phase synchronized solution is neutrally stable; that is, the phase difference between φ_1 and φ_2 may differ during one oscillation, but returns to the initial value at the end of the oscillation, i.e., $\varphi_1(0)-\varphi_2(0) = \varphi_1(T)-\varphi_2(T)$, where T is the period. Such a behavior is not generic in the sense that small perturbations of r_i or s_{ij} may destroy it.

In conclusion, *the in-phase synchronized solution of two identical Class 1 neurons exists, but it is not exponentially stable. Small perturbations can make it disappear or stabilize with a small phase shift. The result is valid for an arbitrary synaptic organization.*

B. Many Neurons

Let us show that the in-phase solution for

$$\varphi_i' = (1-\cos\varphi_i)+(1+\cos\varphi_i)r+\sum_{j=1}^{n} w(\varphi_i)\delta(\varphi_j-\pi) \qquad (14)$$

is neutrally stable when $n=2$ and unstable when $n>2$. For this, consider its small perturbation

$$(\varphi_1(\tau), \varphi_2(\tau), \cdots, \varphi_n(\tau)) = (\phi(\tau)+a(\tau), \phi(\tau), \cdots, \phi(\tau))$$

where $a(\tau)$ is small. To prove instability it suffices to show that $|a(\tau)|$ grows with each cycle. If parameter s in (9) is positive, we consider positive perturbation a (negative otherwise). Thus, the first neuron is ahead of the other neurons; see Fig. 16. When it fires, $\varphi_1 = \phi + a = \pi$, and, hence, $\varphi_i = \pi - a$, for $i = 2, \cdots, n$. At this moment all such φ_i are increased by $w(\pi-a)$ and acquire a new value, say, $\pi - b$, where $b > 0$ can be determined from

$$\tan\frac{\pi-b}{2} = \tan\frac{\pi-a}{2} + s \qquad (15)$$

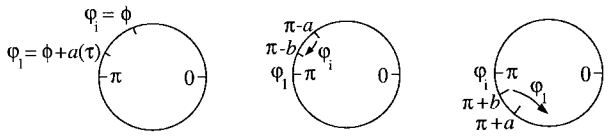

Fig. 16. Illustration to the proof that Class 1 neurons desynchronize.

see (9). We are not interested in exact value of b, we just notice that when φ_i cross π (fire a spike), the phase of the first neuron is $\varphi_1 = \pi + b$. At this moment it receives pulses from the other $n-1$ neurons, and its phase is increased by $(n-1)w(\pi+b)$, so that

$$\tan\frac{\varphi_1^{\text{new}}}{2} = \tan\frac{\pi+b}{2} + (n-1)s.$$

Since

$$\tan\frac{\pi+b}{2} = \tan\frac{-\pi+b}{2} = -\tan\frac{\pi-b}{2}$$

we can use (15) to obtain

$$\tan\frac{\pi+b}{2} = -\tan\frac{\pi-a}{2} - s = \tan\frac{\pi+a}{2} - s.$$

Therefore

$$\tan\frac{\varphi_1^{\text{new}}}{2} = \tan\frac{\pi+a}{2} + (n-2)s.$$

One can see from this equation that $\varphi_1^{\text{new}} = \pi + a$ when $n = 2$, which means that the perturbation persists after the network activity crosses an a-neighborhood of π. In this case the in-phase solution of (14) is neutrally stable and one should take into account the small remainder $\mathcal{O}(\sqrt{\varepsilon}\ln\varepsilon)$ in the pulse coupled model (see Theorem 1) to investigate the stability further. If $n > 2$, then $\varphi_1^{\text{new}} > \pi + a$, which means that the perturbation increases in size. Therefore, the in-phase solution is unstable, and neurons desynchronize. What kind of dynamical regimes the pulse-coupled model can have besides in-phase synchronization is an important, but still unsolved problem.

We see that *Class 1 neurons spontaneously desynchronize*. This fact was observed numerically [4] and proved analytically [3] for two coupled neurons. Since we use the canonical model to prove this fact for $n > 2$ neurons, we confirm and extend the Ermentrout's result [3] that difficulty to synchronize in-phase is a general property of Class 1 excitability that is independent of the equations describing the neuron dynamics.

V. Discussion

In this paper we discuss how an arbitrary weakly connected network of Class 1 neurons (5) satisfying just a few assumptions can be transformed to the pulse-coupled form (7), (10)–(12) by a suitable piece-wise continuous change of variables (see Theorem 1). Therefore, the point of view that pulse-coupled neural networks are "toy" models that were "motivated" by biological neurons is no longer appropriate: The only difference between studying pulse-coupled neural networks mentioned above and biophysically detailed and accurate Hodgkin–Huxley-type neural networks is just a matter of coordinate change.

Particulars of the dynamics of Class 1 neurons do not affect the form of the pulse-coupled models, but affect only the values of parameters r_i and s_{ij}. If we knew the exact equations that describe completely neuron dynamics, we would be able to determine exact values of r_i and s_{ij}. Thus, understanding behavior of the pulse-coupled model for arbitrary r_i and s_{ij} means understanding behavior of *all* weakly connected networks of Class 1 excitable neurons, including those that have not been invented yet, and those consisting of enormous number of equations and taking into account all possible information about the neuron dynamics.

Even though the pulse-coupled network looks simple [in comparison with the original weakly connected system (5)], its behavior is far from being understood. The fact that its activity desynchronizes suggests that the model may have rich and interesting neuro-computational dynamics even for elementary choice of the parameters r_i and s_{ij}.

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