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Synchronization in networks of excitatory and inhibitory neurons with sparse, random connectivity

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Abstract. In model networks of E-cells and I-cells (excitatory and inhibitory neurons), synchronous rhythmic spiking often comes about from the interplay between the two cell groups: the E-cells synchronize the I-cells and vice versa. Under ideal conditions — homogeneity in relevant network parameters, and all-to-all connectivity for instance — this mechanism can yield perfect synchronization. What happens under less ideal conditions? We find that approximate, imperfect synchronization is possible even with very sparse, random connectivity. The crucial quantity is the expected number of inputs per cell. As long as it is large enough (more precisely, as long as the variance of the total number of synaptic inputs per cell is small enough), tight synchronization is possible. The desynchronizing effect of random connectivity can be reduced by strengthening the E→I synapses. More surprisingly, it *cannot* be reduced by strengthening the I→E synapses. However, the decay time constant of inhibition plays an important role: Faster decay yields tighter synchrony. In particular, in models in which the inhibitory synapses are assumed to be instantaneous, the effects of sparse, random connectivity cannot be seen.

1 Introduction

In networks of E-cells (excitatory neurons) and I-cells (inhibitory neurons), synchronous, rhythmic spiking often results from the interplay between the two cell groups, with the E-cells synchronizing the I-cells and vice versa. This mechanism, called PING (Pyramidal Interneuronal Network Gamma) (Whittington et al., 2000) or γ-II (Tiesinga et al., 2001), has been observed in modeling studies, and there are reasons to believe that some experimentally observed gamma rhythms are in fact based on this mechanism (Traub et al., 1999; Whittington et al., 2000; Tiesinga et al., 2001).

The conditions under which PING occurs are not completely understood, though we discuss them heuristically in Section 6. We focus here on the effects of random connectivity on PING. This effect is illustrated by Fig. 1. Details about this figure will be given later. For now, it suffices to say that Fig. 1A shows the emergence of PING in a model E/I network with all-to-all connectivity, and without any kind of heterogeneity in network parameters. The figure indicates spike times. Both cell groups (E and I) synchronize tightly. If the connectivity is made sparse and random, Fig. 1A turns into Fig. 1B. The two cell groups now fire spike volleys of brief but positive durations. (The spike volleys of the I-cells are so brief that they appear to have zero duration in the plot.) The main goal of this paper is to analyze the durations and shapes of these volleys, and their dependence on network parameters.

Synchronization in the presence of random connectivity has been studied previously for excitatory networks (Barkai et al., 1990), for inhibitory networks (Brunel and Hakim, 1999; Wang and Buzsáki, 1996), and for E/I networks (Brunel, 2000; Bush and Sejnowski, 1996; Golomb and Hansel, 2000; Hansel and Mato, 2001; van Vreeswijk and Sompolinsky, 1996; van Vreeswijk and Sompolinsky, 1998; Wang et al., 1995). This literature is aimed at understanding either the stability of the asynchronous state, or transitions from asynchrony to rhythms and vice versa. We do not consider these issues here, but focus instead on a detailed understanding of the near-synchronous state.

We briefly outline the paper. In Section 2, we review the theta model (Ermentrout and Kopell, 1986; Gutkin and Ermentrout, 1998; Hoppensteadt and Izhikevich, 1997), an idealization of a large class of conductance based neuronal models. Our arguments and simulations in this paper are based on this model. We also introduce our model of synapses in Section 2, and describe the connectivity of our model networks. In Section 3, we present numerical experiments demonstrating that the desynchronizing effect of sparseness and randomness in the connectivity primarily originates from the variance in the number of inputs per cell, not from the randomness *per se*. The synchronization of a population of cells by an inhibitory input pulse is analyzed in Section 4, first assuming that all cells receive the same input pulse (Section 4.1), and then, motivated by the result of Section 3, assuming that different cells receive input pulses of different strengths (Section 4.2). Similarly, the synchronization of a population of cells by an excitatory input pulse is analyzed in Section 5, first assuming that all cells receive the same input pulse (Section 5.1), then assuming that different cells receive input pulses of different strengths (Section 5.2). The results of Sections 4 and 5 are combined in Section 6 to analyze PING in E/I networks. In Section 7, we summarize our results and put them into the context of other recent work on the same subject.

2 Review of theta neurons

2.1 Equation of a single theta neuron

In the Hodgkin-Huxley model, a periodically spiking space-clamped neuron is represented by a point moving on a limit cycle in a four-dimensional phase space. Analogously, in the theta model (Ermentrout and Kopell, 1986; Gutkin and Ermentrout, 1998; Hoppensteadt and Izhikevich, 1997), a neuron is represented by a point $P = (\cos\theta, \sin\theta)$ moving on the unit circle S^1. In the absence of synaptic coupling, the

differential equation governing the motion is

$$\frac{d\theta}{dt} = \frac{1}{\tau}\,(1-\cos\theta) + I\,(1+\cos\theta)\ . \tag{1}$$

Here I should be thought of as an input "current", measured in radians per unit time. The time constant $\tau > 0$ is needed to make Eq. (1) dimensionally correct; it is analogous to a membrane time constant.

When $I < 0$, Eq. (1) has the two fixed points

$$\theta_0^{\pm} = \pm 2 \arccos \frac{1}{\sqrt{1-\tau I}}\ .$$

θ_0^- is stable, and θ_0^+ is unstable. For $I < 0$, the vector field on the circle is shown in the left panel of Fig. 2A. If θ is perturbed slightly from θ_0^-, it returns to θ_0^-. However, if θ is raised beyond θ_0^+, a large excursion occurs, with the point P moving around the entire circle while θ increases to $\theta_0^- + 2\pi$. The stable fixed point θ_0^- is the analog of the stable equilibrium of a neuron. The unstable fixed point θ_0^+ is the analog of a spiking threshold.

As I approaches 0, the fixed points approach each other. A saddle-node bifurcation occurs when $I = 0$. The two fixed points come together at $\theta = 0$; see the center panel of Fig. 2A. When $I > 0$, $d\theta/dt > 0$ for all t, so there is no fixed point; see the right panel of Fig. 2A.

The transition from $I < 0$ to $I > 0$ is the analog of the transition from excitability to spiking in a neuron. Neuronal models are called of *type I* if this transition involves a saddle-node bifurcation on a limit cycle, of *type II* if it involves a subcritical Hopf bifurcation (Ermentrout, 1996; Gutkin and Ermentrout, 1998; Rinzel and Ermentrout, 1998). This classification goes back to Hodgkin (1948). Thus the theta model is a type I neuronal model. It has been shown to be *canonical*, in the sense that other type I models can be reduced to it by coordinate transformations (Ermentrout and Kopell, 1986; Hoppensteadt and Izhikevich, 1997).

If $0 < I \ll 1/\tau$, the motion is much slower near $\theta = 0$, i.e., near the "ghost" of the fixed point annihilated in the saddle-node bifurcation, than elswhere. This is illustrated in Fig. 2B, which shows $\sin\theta$ as a function of t for $\tau = 1$, $I = 0.02$. As the point $P = (\cos\theta, \sin\theta)$ moves slowly past $(1,0)$, $\sin\theta$ changes slowly. As P moves rapidly around the circle, $\sin\theta$ rapidly rises to 1, then falls to -1, then returns to values slightly below 0. The graph of $\sin\theta$ resembles, to some extent, the voltage trace of a spiking neuron.[1] When θ crosses $(2l-1)\pi$, l integer, with $d\theta/dt > 0$, we say that the neuron *spikes*.

If $I > 0$ and $-\pi \le \theta_1 \le \theta_2 \le \pi$, the time it takes for θ to rise from θ_1 to θ_2 equals

$$\int_{\theta_1}^{\theta_2} \frac{d\theta}{(1-\cos\theta)/\tau + I(1+\cos\theta)} = \sqrt{\frac{\tau}{I}} \left[\arctan \frac{\tan(\theta/2)}{\sqrt{\tau I}} \right]_{\theta_1}^{\theta_2} . \tag{2}$$

Setting $\theta_1 = -\pi$ and $\theta_2 = \pi$ in this formula, we find that the period equals

$$P = \pi \sqrt{\frac{\tau}{I}} . \tag{3}$$

We denote the time it takes for θ to rise from $\pi/2$ to $3\pi/2$ by W, and call it the *spike width*. Applying formula (2) with $(\theta_1, \theta_2) = (\pi/2, \pi)$ and $(\theta_1, \theta_2) = (-\pi, -\pi/2)$ and adding the results, we find

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[1] Some authors think of $-\cos\theta$, not $\sin\theta$, as the "voltage-like" quantity in the theta model. Which of the two we call "voltage-like" is of no consequence for this paper, and neither is "voltage-like" in any precise sense. However, we prefer to think of $\sin\theta$ as the "voltage-like" quantity for the following aesthetic reason. Consider a theta neuron driven slightly above threshold. Near the "ghost" of the equilibrium point ($\theta = 0$), $\sin\theta$ is slowly increasing, while $-\cos\theta$ has a local minimum. On the other hand, for a Hodgkin-Huxley-type neuron of type I driven slightly above threshold, the membrane potential V is slowly increasing near the "ghost" of the equilibrium point. In this sense, V is more similar to $\sin\theta$ than to $-\cos\theta$.

$$W = \left[\pi - 2\arctan\frac{1}{\sqrt{\tau I}}\right]\sqrt{\frac{\tau}{I}}\,. \tag{4}$$

For physiological realism, we wish to ensure $W/P \ll 1$. By Eqs. (3) and (4),

$$\frac{W}{P} = 1 - \frac{2}{\pi}\arctan\frac{1}{\sqrt{\tau I}}\,.$$

Therefore $W/P \ll 1$ means the same as $\tau I \ll 1$. Since $\arctan(1/\varepsilon) = \pi/2 - \varepsilon + O(\varepsilon^2)$ as $\varepsilon \to 0$, Eq. (4) implies $W \approx 2\tau$ when $\tau I \ll 1$. Thus in the parameter regime of interest to us, τ is approximately half the spike width. Motivated by this discussion, and by the fact that spike widths in real neurons are on the order of milliseconds, we set

$$\tau = 1$$

for the remainder of this paper, think of time as measured in milliseconds, and always consider input currents $I \ll 1$.

2.2 Synapses between theta neurons

We model synapses by adding time dependent input currents to Eq. (1). When the pre-synaptic neuron spikes, the post-synaptic neuron receives an input current (positive or negative, depending on whether the synapse is excitatory or inhibitory) which jumps to its maximum value instantaneously, then decays exponentially. This is the model of Sec. 2.1.5 of Izhikevich (2000); see also Ermentrout (1996) for a discussion of synapses between theta neurons.

Thus a network of N coupled theta neurons is described by a system of differential equations of the form

$$\frac{d\theta_j}{dt} = 1 - \cos\theta_j + \left(I_j + \sum_{i=1}^{N} \alpha_i g_{ij} s_{ij}\right)\left(1 + \cos\theta_j\right)\,, \quad 1 \le j \le N\,.$$

I_j denotes the external input to the j-th neuron, which can be positive or negative. The constant α_i equals $+1$ or -1, depending on whether neuron i is *excitatory* (E) or *inhibitory* (I). The constant $g_{ij} \geq 0$ measures the strength of the synapse from neuron i to neuron j, and $s_{ij} = s_{ij}(t)$ is the *synaptic gating variable* associated with this synapse. The value of s_{ij} always lies between 0 and 1. It jumps to 1 when neuron i spikes. Between spikes of neuron i, it decays exponentially, following the differential equation

$$\frac{ds_{ij}}{dt} = -\frac{s_{ij}}{\tau_{ij}}.$$

The *decay time constants* τ_{ij} are positive.

The jumps of s_{ij} occurring when neuron i spikes cause difficulties in the numerical simulation of the network, and are, of course, not physiologically realistic. In our simulations, we therefore replace the jumps by rapid but smooth rises, letting s_{ij} be governed by a differential equation of the form

$$\frac{ds_{ij}}{dt} = -\frac{s_{ij}}{\tau_{ij}} + e^{-\eta(1+\cos\theta_i)}\,\frac{1-s_{ij}}{\tau_R}$$

with $\tau_R = 0.1$ and $\eta = 5$. The term $e^{-\eta(1+\cos\theta_i)}\,(1-s_{ij})/\tau_R$ is very close to zero unless $\theta_i \approx (2l-1)\pi$, l integer, and drives s_{ij} towards 1 rapidly when $\theta_i \approx (2l-1)\pi$. The parameter τ_R is reminiscent of a synaptic rise time. Fig. 3 shows s_{ij} for $\tau_{ij} = 2$ and 10, with the input of the pre-synaptic neuron chosen so that its period equals 25.

2.3 Sparse, random E/I networks

Throughout this paper, the decay time constants τ_{ij} are assumed to depend on the type of i only (excitatory or inhibitory). So there are two distinct values of τ_{ij}, denoted τ_E and τ_I. We also assume that all excitatory neurons receive the same constant external input drive I_E, and all inhibitory neurons receive the same constant external drive I_I.

In later sections, we will consider networks of coupled theta neurons including $N_E = 4N/5$ excitatory and $N_I = N/5$ inhibitory neurons. These proportions are moti-

vated by the fact that in large portions of the cortex, there are about four times more excitatory than inhibitory neurons (Braitenberg and Schüz, 1998).

The strengths g_{ij} of the synapses are chosen at random. For a given network, they are chosen once and for all, i.e., they do not depend on time. To describe the choice of the g_{ij}, the following notation is useful. Let $\mathcal{E} \subseteq \{1,\ldots,N\}$ denote the set of all indices of excitatory neurons, and similarly I the set of all indices of inhibitory neurons. For $i \in \mathcal{E}$ and $j \in I$, we define

$$g_{ij} = \frac{g_{EI}}{p_{EI} N_E} w_{ij} \ , \tag{5}$$

with

$$w_{ij} = \begin{cases} 1 & \text{with probability } p_{EI}, \\ 0 & \text{otherwise,} \end{cases} \tag{6}$$

where $g_{EI} \geq 0$ and $p_{EI} \in (0,1]$ are constants.

For $j \in I$, we define

$$w_{Ej} = \sum_{i \in \mathcal{E}} w_{ij} \ . \tag{7}$$

Note that w_{Ej} is a binomially distributed random variable. We also define

$$g_{Ej} = \sum_{i \in \mathcal{E}} g_{ij} = \frac{g_{EI}}{p_{EI} N_E} w_{Ej} \ . \tag{8}$$

From the formulas for the mean and standard deviation of binomially distributed random variables, we see that g_{Ej} has mean g_{EI} and standard deviation

$$\sigma_{EI} = g_{EI} \sqrt{\frac{1 - p_{EI}}{p_{EI} N_E}} \ . \tag{9}$$

By the Central Limit Theorem, g_{Ej} is approximately normally distributed if $p_{EI} N_E$ is large. Assuming $p_{EI} \ll 1$, as is physiologically realistic (Braitenberg and Schüz, 1998), Eq. (9) shows that

$$\frac{\sigma_{EI}}{g_{EI}} \approx \frac{1}{\sqrt{p_{EI} N_E}} \ . \tag{10}$$

The left-hand side of (10) is the *coefficient of variation* (the standard deviation divided by the mean) of g_{Ej}, $j \in I$. The expression $p_{EI}N_E$ appearing on the right-hand side is the expected number of excitatory inputs per I-cell.

Formulas analogous to (5)–(10) apply to the I→E, E→E, and I→I synapses. In particular,

$$\sigma_{IE} = g_{IE}\sqrt{\frac{1-p_{IE}}{p_{IE}N_I}}\,, \tag{11}$$

and therefore

$$\frac{\sigma_{IE}}{g_{IE}} \approx \frac{1}{\sqrt{p_{IE}N_I}} \tag{12}$$

for $p_{IE} \ll 1$. The left-hand side of (12) is the coefficient of variation of g_{Ij}, $j \in \mathcal{E}$. The expression $p_{IE}N_I$ appearing on the right-hand side is the expected number of inhibitory inputs per E-cell.

Several authors have pointed out that $p_{EI}N_E$ and $p_{IE}N_I$ are much more important than p_{EI}, N_E, p_{IE}, and N_I in isolation (Golomb and Hansel, 2000; Tiesinga et al., 2002; Wang et al., 1995).

3 Loss of tight synchrony is attributable to variance in the number of inputs per cell

Before considering networks with sparse, random connectivity, we consider one with all-to-all connectivity ($p_{EI} = p_{IE} = 1.0$). Fig. 1A shows an example of a simulation with

$$I_E = 0.1,\ \ I_I = 0,\ \ g_{EI} = g_{IE} = 0.25,\ \ g_{EE} = g_{II} = 0,\ \ \tau_E = 2,\ \ \tau_I = 10\,. \tag{13}$$

Fig. 1A shows spike times, with the horizontal axis indicating time, and the vertical axis cell index. Each of the two cell groups (E and I) synchronizes very rapidly, with

the synchronous population spikes of the I-cells slightly lagging behind those of the E-cells. The synchronization mechanism seen in Fig. 1A is PING, briefly described in the Introduction, and discussed in more detail in Section 6.1.

We comment briefly on our parameter choices. A neuron driven with $I_E = 0.1$ spikes periodically with an interspike interval equal to $\pi/\sqrt{I} \approx 9.93$. Since we think of time as measured in milliseconds, this corresponds to a frequency of $(1000/9.93)$Hz $\approx$ 100Hz. Thus the E-cells are driven so hard that they would spike above gamma frequency if they were not subject to any inhibition. The I-cells are driven at threshold, i.e., they do not spike without additional excitatory input, but any excitatory input, regardless how weak, will make them spike. The values of g_{EI} and g_{IE} can be varied considerably without any qualitative change in Fig. 1A. However, for small values of g_{EI} (roughly < 0.1), two or more population spikes of the E-cells occur before the I-cells respond. For large values of g_{EI} (roughly > 0.7), two or more population spikes of the I-cells occur in response to a population spike of the E-cells. For small values of g_{IE} (roughly < 0.1), the E-cells are not synchronized. For large values of g_{IE}, the rhythm is slow, but not qualitatively different from that in Fig. 1A. For simplicity, we assume here that there are no E$\rightarrow$E or I$\rightarrow$I synapses, i.e., $g_{EE} = g_{II} = 0$. In our experience, E$\rightarrow$E synapses (with a brief synaptic decay time such as $\tau_E = 2$) do not affect PING rhythms much. However, I$\rightarrow$I synapses are crucial in some parameter regimes; this will be discussed in Section 6.1. Our choices of $\tau_E = 2$ and $\tau_I = 10$ are motivated by the decay time constants of excitatory synapses involving AMPA receptors (approximately 2 ms) and inhibitory synapses involving $GABA_A$ receptors (approximately 10 ms); recall that we think of time as measured in milliseconds.

When p_{EI} and p_{IE} are reduced from 1.0 to 0.5, Fig. 1A turns into Fig. 1B. The two cell groups now fire spike volleys of positive durations. As stated in the Introduction, the main goal of this paper is to understand the durations and shapes of these volleys.

In the network underlying Fig. 1B, each E-cell receives input from a random number of I-cells. The expected value of this number is 50. Similarly, each I-cell receives input

from a random number of E-cells, with expectation 200. We now repeat the simulation of Fig. 1B with a network in which the connectivity is still sparse and random, but the variance in the number of inputs per cell has been eliminated. That is, each E-cell receives input from a random set of exactly 50 I-cells, and each I-cell receives input from a random set of exactly 200 E-cells. The network is like that of Fig. 1B in all other regards. The result is shown in Fig. 4A — tight synchrony is restored.

When the number of inputs per cell is fixed, tight synchronization is possible even for much sparser networks. Fig. 4B shows a simulation in which each E-cell receives input from one I-cell, and each I-cell receives input from four E-cells. Although synchronization is achieved a little less rapidly than in Fig. 4A, it becomes tight within a few oscillation periods. A four times larger network, again with one inhibitory input into each E-cell and four excitatory inputs into each I-cell, shows similar behavior; see Fig. 4C.

If the number of excitatory inputs per I-cell is reduced again, from four to one, there appears to be no synchronization any more; see Fig. 4D. Thus there appears to be a minimum number of inputs per cell needed for synchronization, a "percolation threshold". However, this number is very small in our simulations. In previous work on different models, similar thresholds have been found (Golomb and Hansel, 2000; Wang and Buzsáki, 1996; Wang et al., 1995). In all three of these references, asynchrony was found to become unstable when the number of inputs per cell exceeded a threshold value independent of network size.

A cell in a human brain typically receives input from thousands of other cells (Braitenberg and Schüz, 1998). Our numerical experiments suggest that with so many synapses, the impact of sparse, random connectivity on synchronization is attributable to the variance in the number of inputs per cell, not to the percolation threshold. We will assume this to be the case from now on.

4 Synchronization of a population of theta neurons by a single strong inhibitory pulse

4.1 Synchronization by an inhibitory pulse of uniform strength

We consider a population of N identical, uncoupled neurons with common constant external drive above threshold, receiving a common inhibitory synaptic input pulse at time 0. Following Section 2, we model this population by the equations

$$\frac{d\theta_j}{dt} = (1-\cos\theta_j) + (I - gs(t))\,(1+\cos\theta_j)\,, \qquad 1 \le j \le N\,, \tag{14}$$

with $I > 0$, $g > 0$, and

$$s(t) = \begin{cases} e^{-t/\tau_I} & \text{if } t > 0 \\ 0 & \text{if } t \le 0 \end{cases}$$

with $\tau_I > 0$. If the inhibitory pulse is strong, it brings the population close to synchrony. An example is shown in Fig. 5A, which displays results of a simulation with

$$N = 100,\;\; I = 0.05,\;\; g = 0.25,\;\; \tau_I = 10\,. \tag{15}$$

The synchronization brought about by the inhibitory pulse at time 0 is immediate and nearly perfect.

To understand the synchronization shown in Fig. 5A, consider the initial value problem

$$\frac{d\theta}{dt} = 1-\cos\theta + \left(I - ge^{-t/\tau_I}\right)(1+\cos\theta)\,, \qquad t > 0\,, \tag{16}$$

$$\theta(0) = \theta_0\,, \tag{17}$$

with $-\pi < \theta_0 < \pi$. We define

$$J(t) = I - ge^{-t/\tau_I}\,.$$

Eqs. (16) and (17) can then be re-written in the form

$$\frac{d\theta}{dt} = 1-\cos\theta+J(1+\cos\theta) \tag{18}$$

$$\frac{dJ}{dt} = -\frac{J-I}{\tau_I} \tag{19}$$

$$\theta(0) = \theta_0 \tag{20}$$

$$J(0) = I-g \tag{21}$$

The phase portrait for the two-dimensional dynamical system (18), (19) is shown in Fig. 5C for $I = 0.05$ and $\tau_I = 10$. The dashed line in Fig. 5C is the nullcline $d\theta/dt = 0$. The nullcline $dJ/dt = 0$ is the horizontal line $J = I$, the upper edge of the window shown in Fig. 5C. The figure should be extended periodically in θ with period 2π. The flow is upward, in the direction of increasing J.

The most striking feature of Fig. 5C is the existence of strongly attracting and strongly repelling trajectories. Trajectories of this kind exist in many systems of ordinary differential equations, and are called "rivers" (Diener, 1985a; Diener, 1985b). The figure reveals a "stable river", i.e., a trajectory (θ_s, J_s) that is attracting in forward time, indicated as a bold line in Fig. 5C, with $(\theta_s, J_s) \to (-\pi, -\infty)$ as $t \to -\infty$, $d\theta_s/dt > 0$, and

$$J_s(t) = I - ge^{-t/\tau_I} \tag{22}$$

for all t. We denote by T the time when $\theta_s(T) = \pi$, and define

$$J^* = J_s(T) \in (0,I)\,. \tag{23}$$

Eqs. (22) and (23) imply

$$T = \tau_I \ln g - \tau_I \ln(I-J^*)\,. \tag{24}$$

Note that the phase portrait depends on the parameters I and τ_I, but not on g. Therefore the value of J^* depends on I and τ_I, but not on g.

The synchronization seen in Fig. 5A can be understood from Fig. 5C in the following way. For g sufficiently large (that is, $J(0)$ sufficiently negative), and for θ_0 sufficiently far from π, $(\theta(t), J(t))$ is rapidly attracted to $(\theta_s(t), J_s(t))$. At the time when $\theta = \pi$, we therefore have $J \approx J^*$, or $t \approx T$. Thus the first spike after time zero occurs approximately at time T. For θ_0 sufficiently close to π, $\theta(t)$ quickly passes through π, and is then rapidly attracted to $(\theta_s(t) + 2\pi, J_s(t))$. When $\theta(t)$ reaches 3π, $J \approx J^*$ and therefore $t \approx T$. Thus a spike occurs soon after time zero, followed by a spike approximately at time T. Only for values of θ_0 in a narrow transition regime is $(\theta(t), J(t))$ attracted neither to $(\theta_s(t), J_s(t))$, nor to $(\theta_s(t) + 2\pi, J_s(t))$.

Eq. (24) gives the time of the first approximately synchronous population spike. Since we have no explicit formula for J^*, Eq. (24) is not an explicit formula for T. However, since J^* does not depend on g, Eq. (24) does give the precise dependence of T on g. This dependence will be of primary interest to us in the remainder of Section 4. For later reference, we note how (24) is modified when the synchronizing inhibitory pulse arrives not at time 0, but at some time T_0:

$$T = T_0 + \tau_I \ln g - \tau_I \ln(I - J^*) \; . \tag{25}$$

4.2 Approximate synchronization by an inhibitory pulse of non-uniform strength

Motivated by Section 3, we are interested in the effects of variable synaptic strengths. We therefore let the constant g in Eq. (14) depend on j:

$$\frac{d\theta_j}{dt} = (1 - \cos\theta_j) + \big(I - g_j s(t)\big)\,(1 + \cos\theta_j)\,, \qquad 1 \le j \le N\,.$$

That is, different neurons receive inhibitory pulses of different strengths. We assume that the g_j are independent, normally distributed random variables, with mean $\overline{g} > 0$ and standard deviation $\sigma_g > 0$. If $\overline{g}$ is large enough, and σ_g is small enough, the population

is still synchronized approximately. This is illustrated in Fig. 5B, which shows results of a simulation similar to that of Fig. 5A, with

$$N = 100,\ \ I = 0.05,\ \ \overline{g} = 0.25,\ \ \sigma_g = 0.025,\ \ \tau_I = 10\,. \tag{26}$$

(Compare these parameters with those in Eqs. (15).) Instead of the nearly synchronous population spikes of Fig. 5A, we now see spike volleys of brief but positive durations.

To analyze the durations of these spike volleys, let us consider the initial value problem (16), (17) with a random $g > 0$. Let $\rho_g = \rho_g(\gamma)$, $\gamma > 0$, be the probability density of g, and let $\overline{g} > 0$ and $\sigma_g > 0$ be its mean and standard deviation. Let

$$X = \ln g\ . \tag{27}$$

Combining Eqs. (24) and (27),

$$T = \tau_I X - \tau_I \ln(I - J^*)\ . \tag{28}$$

The only random quantity on the right-hand side of Eq. (28) is X. We will discuss its distribution first. Let $\rho_X = \rho_X(\xi)$, $-\infty < \xi < \infty$, be the probability density of X. For $-\infty < a < b < \infty$,

$$\begin{aligned}\int_a^b \rho_X(\xi)\,d\xi &= P(X \in (a,b)) = P(\ln g \in (a,b)) = P(g \in (e^a, e^b)) \\ &= \int_{e^a}^{e^b} \rho_g(\gamma)\,d\gamma = \int_a^b e^{\xi}\rho_g(e^{\xi}))\,d\xi\,.\end{aligned}$$

Therefore

$$\rho_X(\xi) = e^{\xi}\rho_g(e^{\xi}) \tag{29}$$

for all ξ. For small σ_g, the standard deviation of X is

$$\sigma_X \approx \ln'(\overline{g})\sigma_g = \frac{\sigma_g}{\overline{g}}\ . \tag{30}$$

From Eqs. (28) and (29), we see that the probability density function of T is

$$\rho_T(t) = \frac{1}{\tau_I}\, e^{(t+\tau_I \ln(I-J^*))/\tau_I} \rho_g\left(e^{(t+\tau_I \ln(I-J^*))/\tau_I}\right) . \tag{31}$$

For later reference, we note how formula (31) changes if the approximately synchronizing inhibitory pulse arrives not at time 0, but at some time T_0:

$$\rho_T(t) = \frac{1}{\tau_I}\, e^{(t-T_0+\tau_I \ln(I-J^*))/\tau_I} \rho_g\left(e^{(t-T_0+\tau_I \ln(I-J^*))/\tau_I}\right) . \tag{32}$$

Using Eqs. (28) and (30), we see that the standard deviation of T is

$$\sigma_T = \tau_I\, \sigma_X \approx \tau_I\, \frac{\sigma_g}{\overline{g}} \tag{33}$$

for small σ_g. We think of σ_T as a measure of the duration of the spike volleys. Thus the duration of the spike volleys is proportional to the product of τ_I, the decay time constant of inhibition, and the coefficient of variation $\sigma_g/\overline{g}$ of g.

To verify these results computationally, we return to the example of Fig. 5B. Strictly speaking, the preceding discussion does not apply to this example, since g, which is assumed to be normally distributed, is not guaranteed to be positive. However, formulas (29), (31), and (33) are well-defined if ρ_g is a normal density. Since $\overline{g} = 0.25$ and $\sigma_g = 0.025$, the probability of $g \leq 0$ is extremely small. We therefore expect (29), (31), and (33) to hold with good accuracy.

We define $T^{(j)}$ to be the time of the spike of the j-th neuron within the first nearly synchronous spike volley. We set

$$\hat{\overline{T}} = \frac{\sum_{j=1}^{N} T^{(j)}}{N} \quad \text{and} \quad \hat{\sigma}_T = \sqrt{\frac{\sum_{j=1}^{N} \left(T^{(j)} - \hat{\overline{T}}\right)^2}{N-1}} . \tag{34}$$

Here and for the remainder of this paper, hats indicate results obtained from numerical simulations. In the example of Fig. 5B, we find

$$\hat{\sigma}_T \approx 1.02$$

We see that $\hat{\sigma}_T$ is indeed close to $\tau_I \sigma_g / \overline{g}$, which equals 1.0 in this example. If we double τ_I in this experiment, $\hat{\sigma}_T$ rises from 1.02 to 2.04, in agreement with Eq. (33).

To verify Eq. (31) numerically, we must know the value of $\tau_I \ln(I - J^*)$. Taking expectations on both sides of Eq. (28), we find

$$\tau_I \ln(I - J^*) = \tau_I E(X) - E(T) = \tau_I E(\ln g) - E(T) \; .$$

For sufficiently small σ_g, this implies

$$\tau_I \ln(I - J^*) \approx \tau_I \ln \overline{g} - E(T) \; ,$$

suggesting the approximation

$$\tau_I \ln(I - J^*) \approx \tau_I \ln \overline{g} - \hat{\overline{T}} \; . \tag{35}$$

Fig. 5D shows the density ρ_T, as defined in Eq. (31), using the approximation (35), with $\tau_I = 10$, and assuming that ρ_g is a normal density with $\overline{g} = 0.25$ and $\sigma_g = 0.025$. The histogram in Fig. 5D indicates the actual spike time density, determined from the numerical simulation. The agreement between the theoretical prediction and the actual spike time distribution is excellent.

For later reference, we note how Eq. (35) changes when the inhibitory pulse arrives not at time 0, but at some time T_0. From Eq. (25), we then obtain

$$\tau_I \ln(I - J^*) \approx \tau_I \ln \overline{g} + T_0 - \hat{\overline{T}} \; . \tag{36}$$

5 Synchronization of a population of theta neurons by a single strong excitatory pulse

5.1 The synchronous population spike triggered by an excitatory pulse of uniform strength

We next consider a population of N identical, uncoupled neurons with common constant external drive below or at threshold, receiving a common excitatory synaptic input pulse at time 0. We model this situation by the equations

$$\frac{d\theta_j}{dt} = (1-\cos\theta_j) + (I+gs(t))\,(1+\cos\theta_j)\,, \qquad 1 \le j \le N\,, \tag{37}$$

with $I \le 0$, $g > 0$, and

$$s(t) = \begin{cases} e^{-t/\tau_E} & \text{if } t > 0 \\ 0 & \text{if } t \le 0 \end{cases}$$

with $\tau_E > 0$. If the excitatory pulse is strong, it triggers a nearly synchronous population spike soon after time 0.

To analyze this in more detail, we consider the initial value problem

$$\frac{d\theta}{dt} = 1-\cos\theta + \left(I+ge^{-t/\tau_E}\right)(1+\cos\theta), \qquad t > 0\,, \tag{38}$$

$$\theta(0) = -2\arccos\frac{1}{\sqrt{1-I}}\,. \tag{39}$$

Recall from Section 2 that for $I < 0$, the right-hand side of Eq. (39) represents the stable fixed point of the equation $d\theta/dt = 1-\cos\theta + I(1+\cos\theta)$. Thus we are considering the *response of a neuron at rest to an excitatory synaptic pulse*. We denote by T the first time at which the neuron spikes, with $T = \infty$ if there is no spike at all.

We have no general analytic expression for T as a function of I, τ_E, and g. However, it is easy to see that for fixed I and τ_E, T is a strictly decreasing function of g, with

$\lim_{g\to\infty} T = 0$ and $\lim_{g\to g_c+} T = \infty$ for some $g_c \geq 0$; see Fig. 6B. For $\tau_E = \infty$, T can be computed using formula (2), with I replaced by $I+g$ and $\tau = 1$. The formula becomes particularly simple for $I = 0$; in that case,

$$T = \frac{\pi}{2}\frac{1}{\sqrt{g}}\,. \tag{40}$$

The approximation $\tau_E = \infty$ is accurate as long as $e^{-T/\tau_E} \approx 1$, since then the exponential decay in Eq. (38) can be neglected over one period. Since $T \to 0$ as $g \to \infty$, this means that the assumption $\tau_E = \infty$ is accurate for sufficiently large g. The assumption $I = 0$ is accurate when $|I|/g$ is sufficiently small. So this assumption, too, is accurate for sufficiently large g.

Fig. 6B shows T as a function of g, for various values of I and τ_E, demonstrating that Eq. (40) approximates T reasonably well over a large range of parameter values.

5.2 The approximately synchronous population spike triggered by an excitatory pulse of non-uniform strength

If the synaptic strength in Eq. (37) depends on j, i.e., if different neurons receive excitatory pulses of different strengths, the equations are:

$$\frac{d\theta_j}{dt} = (1-\cos\theta_j) + \big(I + g_j s(t)\big)\,(1+\cos\theta_j)\,, \qquad 1 \leq j \leq N\,.$$

Fig. 6A shows that the resulting population spike is not perfectly synchronous. (But notice that Fig. 6A shows a brief time window only; the synchronization is not perfect, but fairly tight.) In the simulation underlying this figure,

$$N = 100,\ \ I = 0,\ \ \overline{g} = 0.25,\ \ \sigma_g = 0.025,\ \ \tau_E = 2\,. \tag{41}$$

To analyze the duration of the spike volley triggered by an excitatory pulse of non-uniform strength, we consider the initial value problem (38), (39) with a random $g > g_c$.

If σ_g is small, the standard deviation of T is

$$\sigma_T \approx \left| \frac{\partial T}{\partial g} \right| \sigma_g \, . \tag{42}$$

For $\tau_E = \infty$ and $I = 0$,

$$\frac{\partial T}{\partial g} = \frac{\pi}{4} g^{-3/2} \tag{43}$$

by Eq. (40). Fig. 6C shows $\partial T / \partial g$ as a function of g, for various values of I and τ_E. The figure confirms that the right-hand side of Eq. (43) approximates $\partial T / \partial g$ reasonably well for large values of g. Combining Eqs. (42) and (43) yields

$$\sigma_T \approx \frac{\pi}{4} \frac{1}{\sqrt{\overline{g}}} \frac{\sigma_g}{\overline{g}} \, . \tag{44}$$

For illustration, we return to the example of Fig. 6A ($I = 0$, $\tau_E = 2$, $\overline{g} = 0.25$, $\sigma_g = 0.025$). We define $T^{(j)}$ to be the time of the spike of the j-th neuron, and define $\hat{\overline{T}}$ and $\hat{\sigma}_T$ as in (34). Numerically, we find

$$\hat{\sigma}_T \approx 0.270 \, .$$

To evaluate the right-hand side of Eq. (42), we approximate $\partial T / \partial g$ numerically. We find $\partial T / \partial g \approx -10.30$ for the parameter values of Fig. 6A. The approximation of Eq. (42), based solely on the assumption that σ_g is so small that the relation between σ_T and σ_g is approximately linear, proves fairly accurate here; it yields

$$\sigma_T \approx 0.256 \, .$$

The assumption $\tau_E = \infty$, which underlies Eq. (44), degrades the accuracy, but by less than a factor of two:

$$\sigma_T \approx 0.157 \, .$$

6 The PING synchronization mechanism

6.1 PING in fully connected E-I networks

We return to the example of Fig. 1A. Each of the cell groups (E and I) synchronizes rapidly, with the population spikes of the inhibitory neurons slightly lagging those of the excitatory ones. We state, in a non-rigorous way, based on numerical experience and heuristics, conditions that are sufficient to induce firing patterns as in Fig. 1A:

(1) The E-cells receive external input significantly above their spiking threshold.

(2) The E→I synapses are so strong, and have so short a rise time that a surge in spiking of the E-cells quickly triggers a surge in spiking of the I-cells.

(3) The I-cells spike only in response to the E-cells.

(4) The I→E synapses are so strong that a population spike of the I-cells approximately synchronizes the E-cells.

If conditions (1)–(4) are satisfied, synchronous rhythmic spiking develops as follows (Whittington et al., 2000; Tiesinga et al., 2001). Initial activity in the E-cells triggers activity in the I-cells. This inhibits activity in the E-cells, thereby removing the drive to the I-cells. A period of low activity in both E- and I-cells results. When the inhibition wears off and the E-cells spike again, they are closer to synchrony than previously because of the mechanism described in Section 4.1. The spiking of the E-cells causes spiking of the I-cells, closer to synchrony than previously because of the mechanism of Section 5.1. The cycle now repeats.

Condition (1) is evidently needed to drive activity. We discuss conditions (2)–(4) in some more detail, and present numerical results illustrating what happens when they

are violated.

When condition (2) is violated, i.e., when the E→I synapses are weak, a pattern such as the one shown in Fig. 7A often develops: The E-cells and the I-cells still synchronize, but the E-cells spike several times between population spikes of the I-cells. The parameters in Fig. 7A are as in Fig. 1A, except that g_{EI} has been reduced from 0.25 to 0.05.

Condition (3) is violated if the drive to the I-cells becomes too strong, but can be restored by introducing I→I-synapses. We illustrate this with the following numerical experiment. In Fig. 1A, $I_I = 0$. If we raise I_I to 0.05, the figure changes dramatically, as shown in Fig. 7B. Here I_I is strong enough to drive asynchronous activity in the I-cells that suppresses the E-cells altogether. Condition (3), and with it the rhythm, is restored by setting $g_{II} = 0.25$; see Fig. 7C.

We note that in the example of Fig. 7C, the I-cells would spike synchronously even without the E-cells. Thus the role of the I-I synapses is to synchronize the I-cells, replacing nearly constant inhibition by phasic inhibition, which allows the E-cells to fire. One might therefore consider the rhythm in Fig. 7C an "ING" or "γ-I" rhythm (see Section 7). The parameter regime investigated here is similar to that of Fig. 7d of Tiesinga et al. (2001). There as here, asynchronous activity of the I-cells suppresses activity in the E-cells altogether, while synchronous activity of the I-cells permits firing of the E-cells. In our Fig. 7C, the E-cells do play an important role in setting the frequency of the rhythm. In the absence of the E-cells, the rhythm would be much slower. Thus the firing of the I-cells in Fig. 7C does come in response to the firing of the E-cells, as in PING.

Fig. 7D shows what may happen when condition (4) is violated, i.e., when the I→E synapses are weak. The parameters in Fig. 7D are as in Fig. 1A, except that g_{IE} has been reduced from 0.25 to 0.05. The inhibitory synapses no longer suffice to synchronize the E-cells. As a result, the I-cells, which were synchronized by the E-cells in Fig. 1A, are no longer synchronized either.

A mathematical examination of conditions (1)–(4) will be the subject of future publications.

6.2 PING in sparsely, randomly connected E-I networks

When p_{EI} and p_{IE} are reduced from 1.0 to 0.5, Fig. 1A turns into Fig. 1B. In analyzing the population spikes of the E-cells in Fig. 1B, we make the simplifying assumption that the population spikes of the I-cells are perfectly synchronous. Since the I-cells are in fact fairly tightly synchronized in Fig. 1B, this is a good approximation at least for the parameters used in Fig. 1B. When the I-cells spike, all E-cells receive inhibitory pulses. However, different neurons receive inputs of different strengths because of the random connectivity. The resulting approximately synchronous spike volley of the E-cells can be analyzed using Section 4.2.

We focus on one particular spike volley of the E-cells, say the first one following $t = 100$ in Fig. 1B. We define $T_E^{(j)}$ to be the time of the spike of the j-th E-cell during this volley. We define

$$\hat{\overline{T}}_E = \frac{\sum_j T_E^{(j)}}{N_E} \quad \text{and} \quad \hat{\sigma}_E = \sqrt{\frac{\sum_j \left(T_E^{(j)} - \hat{\overline{T}}_E\right)^2}{N_E - 1}} \,.$$

From Eqs. (11) and (33), we find the prediction

$$\hat{\sigma}_E \approx \tau_I \sqrt{\frac{1 - p_{IE}}{p_{IE} N_I}} \,. \tag{45}$$

For the first spike volley of the E-cells following $t = 100$ in Fig. 1B, we find numerically

$$\hat{\sigma}_E = 1.18 \,.$$

The prediction of Eq. (45) is remarkably accurate:

$$\tau_I \sqrt{\frac{1 - p_{IE}}{p_{IE} N_I}} \approx 1.22 \,.$$

We expect the shape of the spike volley to be approximately described by Eq. (32). To evaluate Eq. (32), we must evaluate $T_0 - \tau_I \ln(I - J^*)$. (Recall that T_0 is the time at which the synchronizing inhibitory pulse arrives — here the time of the inhibitory population spike immediately preceding the excitatory population spike under consideration.) Following Eq. (36), we use the approximation

$$T_0 - \tau_I \ln(I - J^*) \approx \hat{\overline{T}}_E - \tau_I \ln g_{IE} \ .$$

The predicted and actual spike time distributions are shown in Fig. 8. The agreement is good.

We now consider the first spike volley of the I-cells following $t = 100$ in Fig. 1B. We define $T_I^{(j)}$ to be the time of the spike of the j-th I-cell during this volley, and

$$\hat{\overline{T}}_I = \frac{\sum_j T_I^{(j)}}{N_I} \quad \text{and} \quad \hat{\sigma}_I = \sqrt{\frac{\sum_j \left(T_I^{(j)} - \hat{\overline{T}}_I \right)^2}{N_I - 1}} \ .$$

From Eqs. (9) and (44), we find the prediction

$$\hat{\sigma}_I \approx \frac{\pi}{4} \frac{1}{\sqrt{g_{EI}}} \sqrt{\frac{1 - p_{EI}}{p_{EI} N_E}} \, . \tag{46}$$

For the first spike volley of the I-cells following $t = 100$ in Fig. 1B, we find numerically

$$\hat{\sigma}_I = 0.151 \, .$$

The prediction of Eq. (46) is somewhat inaccurate, as was to be expected because it is based on three rather substantial idealizations: $\tau_E = \infty$, perfect synchrony of the E-cells, and the assumption that the I-cells return to rest between the spike volleys of the E-cells; see Eq. (39). However, the discrepancy is still not greater than a factor of two:

$$\frac{\pi}{4} \frac{1}{\sqrt{g_{EI}}} \sqrt{\frac{1 - p_{EI}}{p_{EI} N_E}} \approx 0.0785 \, .$$

We note that it would be possible to relax the assumption of perfect synchrony in the E-cells, since we have fairly precise information about the durations and even the shapes of the spike volleys of the E-cells. We don't pursue this here, since our goal here is qualitative insight, not precise quantitative information.

7 Discussion

We have analyzed the effects of sparse, random connectivity on the PING synchronization mechanism. In particular, we have derived approximate formulas for the durations of the spike volleys, Eqs. (45) and (46). To make these formulas as transparent as possible, let us use the approximations $1 - p_{IE} \approx 1$ and $1 - p_{EI} \approx 1$, and write

$$M_{EI} = p_{EI} N_E$$

for the expected number of excitatory inputs per inhibitory cell, and

$$M_{IE} = p_{IE} N_I$$

for the expected number of inhibitory inputs per excitatory cell. Eqs. (45) and (46) then become

$$\hat{\sigma}_E \approx \tau_I \frac{1}{\sqrt{M_{IE}}} \tag{47}$$

and

$$\hat{\sigma}_I \approx \frac{\pi}{4\sqrt{g_{EI}}} \frac{1}{\sqrt{M_{EI}}} \,. \tag{48}$$

An interesting feature of these formulas is their lack of symmetry. The time constant τ_I appears in (47), but the time constant τ_E does not appear in (48). Similarly, g_{EI} appears in (48), but g_{IE} does not appear in (47).

If a theta neuron were driven with a constant drive equal to g_{EI}, it would spike periodically with a period which we call P_{EI}. From Eq. (3),

$$P_{EI} = \frac{\pi}{\sqrt{g_{EI}}} \,.$$

Using this in Eq. (48), we find

$$\hat{\sigma}_I \approx \frac{1}{4} \frac{P_{EI}}{\sqrt{M_{EI}}} \,. \tag{49}$$

Let P denote the period of the PING rhythm. In the simulations of this paper,

$$P_{EI} \ll \tau_I < P, \quad M_{EI} > M_{IE} \gg 1 \,. \tag{50}$$

The inequality $M_{IE} \gg 1$ is certainly realistic (Braitenberg and Schüz, 1998). The inequality $\tau_I < P$ holds for a gamma rhythm if the inhibitory synapses are mediated by $GABA_A$, since the typical period of gamma oscillations is about 25 ms, and the decay time constant of $GABA_A$ synapses is about 10 ms. It appears that PING without $P_{EI} \ll \tau_I$ is not possible with realistic values of τ_I; more will be said on this point below. Combining (47), (49), and (50), we find

$$\hat{\sigma}_I \ll \hat{\sigma}_E \ll P \,.$$

Thus our theory predicts that for PING in realistic parameter regimes, sparse and random connectivity will not prevent either E- or I-cells from synchronizing fairly tightly, but the I-cells will synchronize much more tightly than the E-cells. Our computational results are in agreement with these conclusions; see Fig. 1B.

It is possible to obtain PING without the condition $P_{EI} \ll \tau_I$ if τ_I is made unrealistically small and g_{IE} is made unrealistically large. In fact, in many modeling studies, synapses have been assumed to act instantaneously, i.e., with zero rise and decay times (Brunel, 2000; Brunel and Hakim, 1999; Izhikevich, 1999; Mirollo and Strogatz, 1990; Tiesinga and Sejnowski, 2001); this amounts to taking a limit in which simultaneously $\tau_I \to 0$ and $g_{IE} \to \infty$. (In Brunel (2000) and Brunel and Hakim (1999), there is a delay

between the spiking of the pre-synaptic neuron and its effect on the post-synaptic neuron; however, this delay is not relevant for the present discussion.) In some regards, the behavior of instantaneous synapses is indeed not very different from that of synapses with more realistic time courses. However, Eqs. (47) and (48) predict that the effects of random connectivity will be reduced dramatically by making the synapses instantaneous. To verify this numerically, we repeat the simulation of Fig. 1B, with τ_I reduced by a factor of 50, and g_{IE} raised by a factor of 50. This closely mimics instantaneous synapses, but is computationally simpler. The result is shown in Fig. 9. As predicted, synchronization is much tighter than in Fig. 1B, even though the randomness in the connectivity is the same in Fig. 9 as in Fig. 1B. The rhythm is also accelerated; this is a result of the reduction in τ_I.

Our theory for the spike volleys of the E-cells is precise enough to allow accurate predictions not just of the durations of the volleys, but even of their shapes, as shown in Fig. 8. A refinement of the theory could be obtained by taking into account the positive durations of the spike volleys of the I-cells, perhaps using ideas similar to those of Section 3.2 of Tiesinga and Sejnowski (2001). (Recall that in Section 4, the duration of the spike volleys of the I-cells was assumed to be negligible.) However, our numerical results indicate that, in our parameter regime, neglecting the durations of the spike volleys of the I-cells gives an excellent approximation. Tiesinga et al. (2002) discuss a related problem, using numerical simulation primarily, and with an emphasis on information theoretic ideas. They also present experimental results. For instance, Fig. 8b of their paper illustrates how the jitter in the spike times of a rat hippocampal neuron decreases when the quantity "n_{pre}" (our "$p_{IE}N_I$") increases, in rough qualitative agreement with Eq. (45). (Rough qualitative agreement is the best that can be expected here, in view of the idealized nature of our theory.)

Our theory for the spike volleys of the I-cells is less accurate than that for the excitatory ones. However, since the synchronization of the I-cells is typically quite tight, and brought about by a crude mechanism (a burst of excitation triggers an almost im-

mediate, and therefore almost synchronous response of the I-cells), a precise theory for the spike volleys of the I-cells is of less interest here. To create such a theory, we would need to study how the duration of an excitatory input spike volley is related to the duration of an output spike volley triggered by it. (Recall that in Section 5, the duration of the spike volleys of the E-cells was assumed to be negligible.) This issue is centrally important in the study of synfire chains (Diesmann et al., 1999). Fig. 3c of Diesmann et al. (1999) shows that the *strength* of the input spike volley is crucial. Strong, loosely synchronous input spike volleys can trigger tightly synchronous output spike volleys. Using a different way of measuring synchrony, the relation between input synchronization and output synchronization was also studied by Burkitt and Clark (2001).

Combining ideas from the preceding two paragraphs, a more accurate overall theory of PING may be created as follows. First, the approximate spike time distribution within the spike volleys of the E-cells, computed based on Section 4, is taken into account in approximating the spike time distribution within the spike volleys of the I-cells. This yields a refinement of Section 5, which in turn can be taken into account in approximating the spike time distribution within the spike volleys of the E-cells, leading to a refinement of Section 4. Iterating this process, one may obtain increasingly accurate approximations to the spike time distributions within spike volleys. However, such an improved theory of PING is beyond the scope of the current paper.

While working on this project, we carried out far more simulations than have been presented here. Our conclusions hold over a wide range of parameters. For instance, E$\rightarrow$E synapses with $g_{EE} = 0.25$ have little effect on Fig. 1B. Weak I$\rightarrow$I synapses have little effect, except for the point made at the end of Section 6.1 — such synapses can make gamma rhythms possible in cases when the external drive to the I-cells is fairly strong. Strengthening I$\rightarrow$I synapses often leads to a transition to a different synchronization mechanism, called ING by Whittington et al. (2000) and γ-I by Tiesinga et al. (2001). In ING, the I-cells are driven externally, not by the E-cells, and synchronize

not only the E-cells, but also themselves. For ING, the widths and shapes of the spike volleys of both E- and I-cells can be approximated using the ideas of Section 4.1. Fig. 10 shows a simulation with

$$g_{IE} = g_{II} = 0.25,\ \ g_{EI} = g_{EE} = 0,\ \ \tau_I = 10,\ \ I_E = I_I = 0.1,\ \ p_{IE} = p_{II} = 0.5$$

As our theory would predict, the volleys of the E- and I-cells are now of equal durations. In future work, we will investigate ING in sparse, random networks in more detail, including in particular the effects of E→I-synapses.

Throughout this paper, we have used the theta model. As remarked in Section 2.1, the theta model is *canonical* for type I neuronal models, in the sense that other type I models can be reduced to it by coordinate transformations (Ermentrout and Kopell, 1986; Hoppensteadt and Izhikevich, 1997). We therefore expect the picture to be qualitatively similar for all type I models, even though the details of our calculations, and in particular the details of the centrally important Fig. 5C, do depend on our choice of model. Whether and how our results generalize to type II models remains to be explored.

It would also be interesting to explore the effects of spike adaptation on our analysis. Synchronization in sparsely, randomly connected networks with spike adapation has been studied previously by van Vreeswijk and Hansel (2001). Van Vreeswijk and Hansel discuss synchronization of bursts (not individual spikes) via adaptation (not inhibition). They observe that strong I→E synapses desynchronize bursts, in contrast with our regime, in which strong I→E synapses synchronize spikes. The model and analysis are so different from ours that a detailed comparison would be a major endeavor, but the paper certainly suggests studying effects of adaptation in our model in the future.

We have analyzed synchronization by common input for the purpose of better understanding PING and ING. We remark, however, that synchronization by common input is also of neurobiological interest by itself (Usrey and Reid, 1999). We have shown

that even very sparse input can synchronize, and have analyzed the desynchronizing effect of heterogeneity in input strength.

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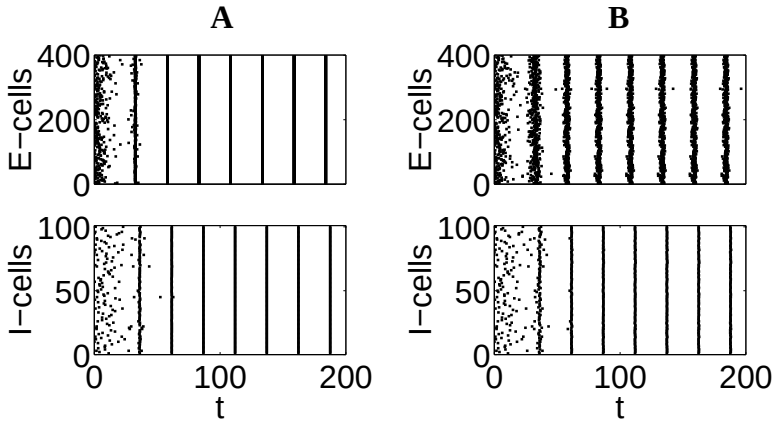

Figure 1: PING in E-I networks with A: all-to-all connectivity and B: sparse connectivity.

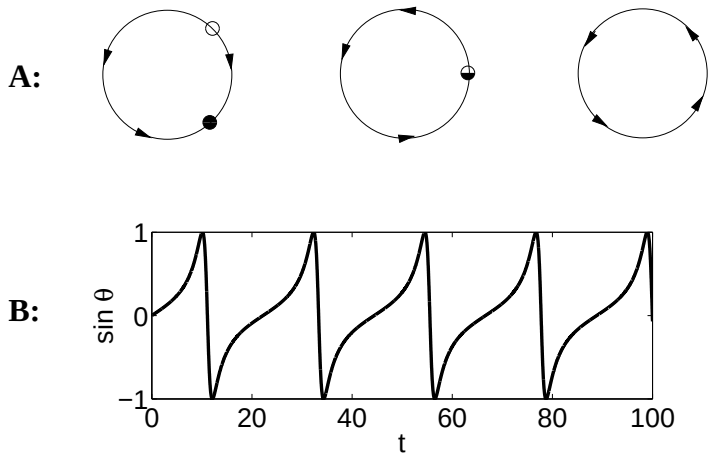

Figure 2: Theta model. A: The vector field on the circle for $I < 0$, $I = 0$, and $I > 0$. B: $\sin\theta$ as a function of time.

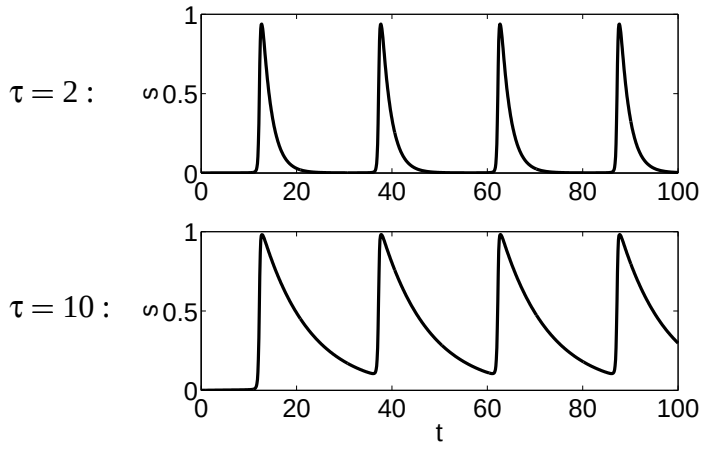

Figure 3: Synaptic gating variable s as a function of time, with decay time constants $\tau = 2$ and $\tau = 10$.

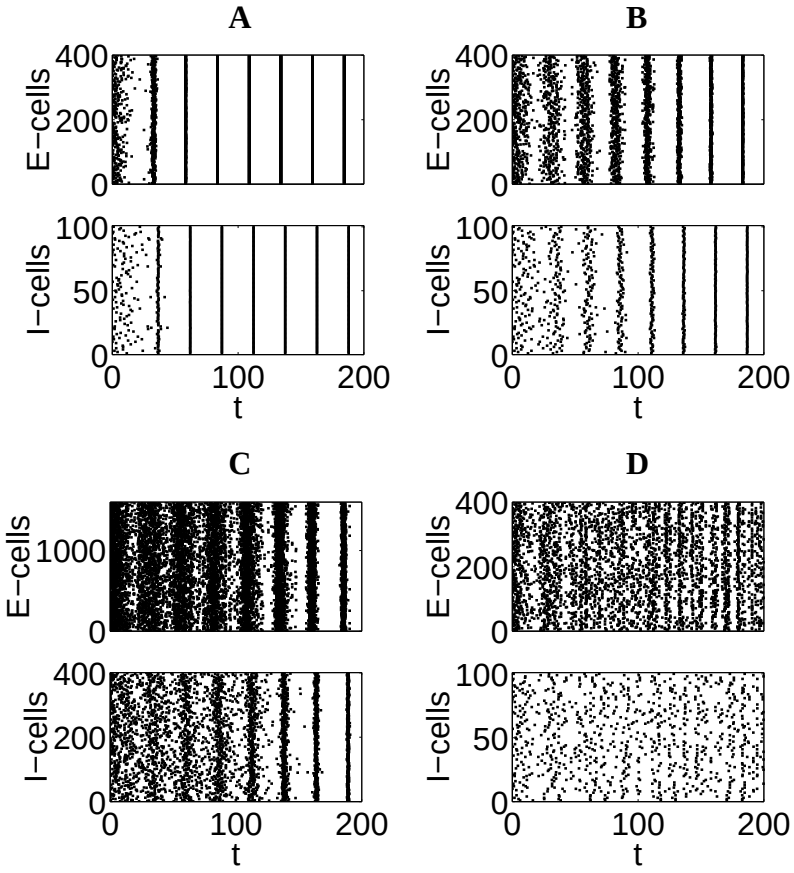

Figure 4: Simulation of E-I networks with sparse, random connectivity, but without variance in the numbers of inputs per cell. A: Parameters as in Fig. 1B, but with variance in numbers of inputs per cell eliminated. B: Only four excitatory inputs per I-cell, and one inhibitory input per E-cell. C: Same as B in a larger network. D: Same as B, but with only one excitatory input per I-cell.

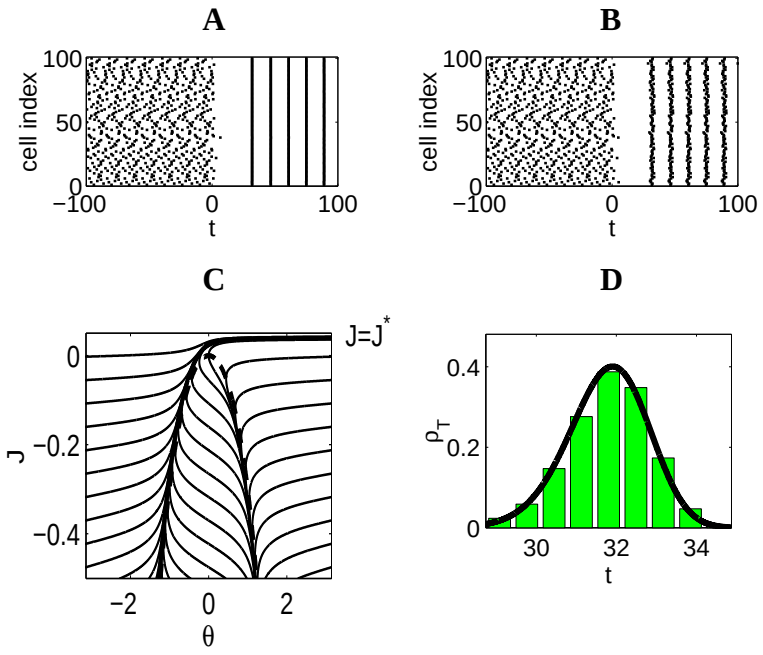

Figure 5: Synchronization by a single inhibitory pulse. A: Inhibitory pulse of uniform strength. B: Inhibitory pulse of non-uniform strength. C: Phase portrait for Eqs. (18), (19) with $I = 0.05$, $\tau_I = 10$, with the stable river indicated boldly. D: Distribution of spikes within the first volley following $t = 0$, in a simulation identical to that in B, but with 1000 neurons: predicted (solid) and actual (bars).

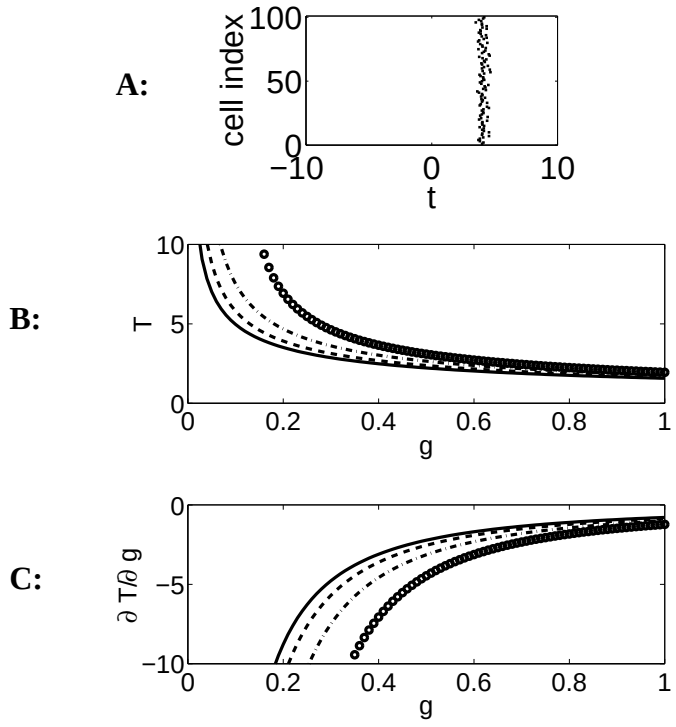

Figure 6: A: Approximately synchronous population spike triggered by a single non-uniform excitatory pulse. B: Time T between arrival of excitatory pulse and spike triggered by it, as a function of the strength g of the pulse, for $(I,\tau_E)=(0,\infty)$ (solid), $(0,5)$ (dashes), $(0,2)$ (dash-dots), $(-0.01,2)$ (circles). C: $\partial T/\partial g$ as a function of g, for the parameter values of B.

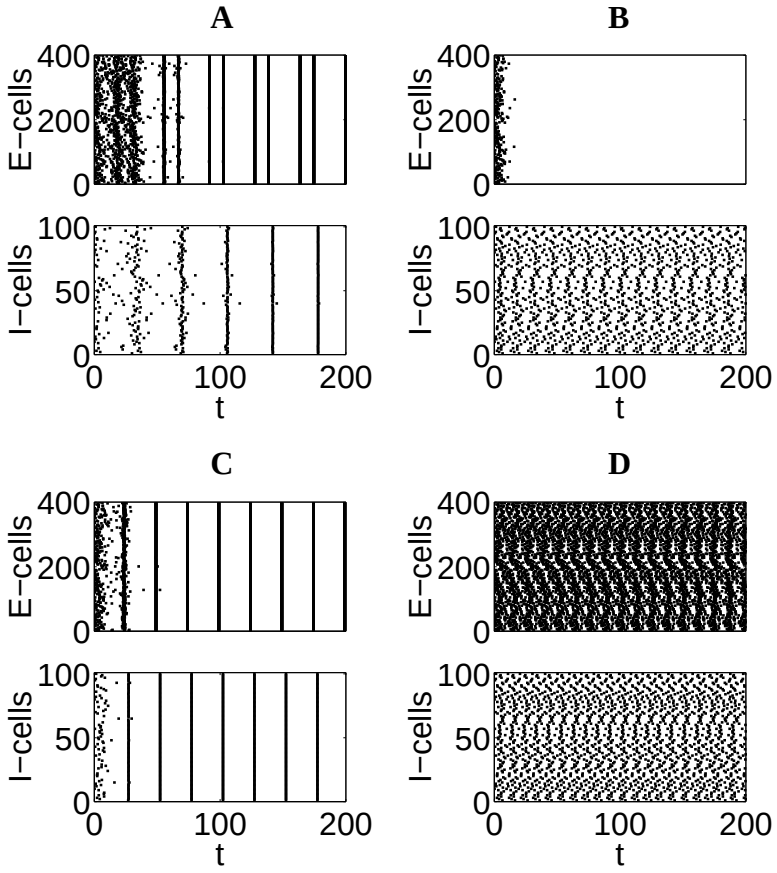

Figure 7: Illustration of conditions (2)–(4) from Section 6.1. A: PING is lost when E→I synapses become too weak. B: PING is lost as a result of too much drive to the I-cells. C: Rhythm is restored by adding I→I synapses. D: PING is lost when I→E synapses become too weak.

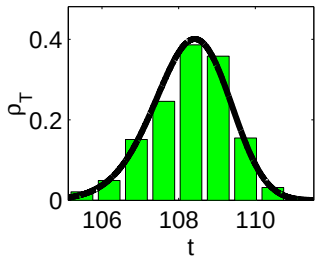

Figure 8: Distribution of spikes within the first spike volley following $t = 100$ in Fig. 1B: predicted (solid) and actual (bars).

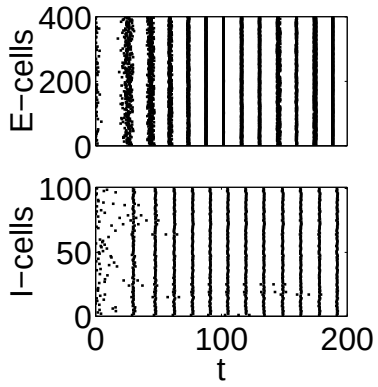

Figure 9: PING in an E-I network with sparse, random connectivity, with nearly instantaneous synapses.

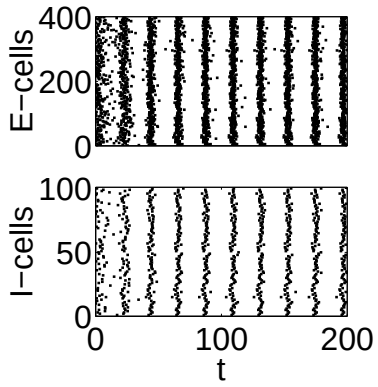

Figure 10: ING in an E-I network with sparse, random connectivity.