Pattern of Synchrony in Inhomogeneous Networks of Oscillators with Pulse Interactions

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Systems of globally coupled oscillators often display states of full synchrony in which all oscillators are phase locked. It is shown that for globally coupled oscillators with neuronlike pulse interactions, the phase-locked state is unstable to inhomogeneity in the local frequency. For weak inhomogeneity the system breaks into two subpopulations: one that is phase locked and another one that consists of aperiodic oscillators. The fraction of the unlocked population remains finite in the limit of vanishing inhomogeneity.

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Mutual synchronization of oscillators is ubiquitous in nature. Examples include synchronization of cardiac pacemaker cells, flashing fireflies, and charge density waves [1,2]. Recently, considerable attention has been drawn to the various forms of coherent oscillatory activity discovered in the central nervous system (see, e.g., [3]). In general, synchrony in a system of coupled oscillators is characterized by nonzero correlations between the oscillators' phases. A strong form of synchrony is a phase-locked state where all the oscillators maintain fixed phase relationships among themselves. Of particular interest is the question whether a phase-locked state is stable to inhomogeneity or noise in the local intrinsic frequencies. This question has been studied mostly in models in which the interaction between oscillators was taken to depend continuously on their state variables (see, e.g., [2,4]). In these models it was found that the phase-locked state is stable to weak disorder or noise. When the width of the distribution of the intrinsic frequencies is small compared to the coupling strength, almost all oscillators are phase locked. In this paper we examine this issue in a system of globally coupled oscillators with pulse interactions, i.e., interactions that depend discontinuously on the state variables of the interacting oscillators. Such interactions are of special interest for neural systems, where often synaptic potentials are generated by the spiking of the presynaptic neurons. We show that in such a system the completely phase-locked state is unstable to weak disorder. For a small degree of inhomogeneity, the oscillators are divided into two populations, one of which exhibits phase-locked periodic behavior, whereas the other consists of unlocked oscillators which exhibit aperiodic patterns that are only partially synchronized. The fraction of unlocked population remains finite in the limit of weak disorder, and grows as the inverse logarithm of the width of disorder signaling the rapid destruction of synchronization by the inhomogeneity. These results are derived analytically, by solving a set of self-consistent mean-field equations in the limit of weak disorder. Numerical results supporting the analytical prediction are presented. The implications for other dynamical systems are discussed at the end of the paper.

We consider a network of fully connected oscillators modeled as *integrate-and-fire neurons*. The dynamics is described by

$$\frac{dV_i}{dt} = -V_i + I + I_i^0 , \tag{1}$$

$$\frac{dI(t)}{dt} = -\frac{I(t)}{\tau_0} + K\rho(t) , \tag{2}$$

where

$$\rho(t) = \frac{1}{N}\sum_j \delta(t - t_j) . \tag{3}$$

Here $V_i(t)$ represents the membrane potential of the ith neuron; I_i is an external, time-independent input to the neuron i. The current $I(t)$ is the mean-field synaptic current generated by the spikes of all the neurons in the system, and K is the normalized strength of the interaction. The normalized density of spikes is $\rho(t)$. The times t_j are the spiking times of the neuron j. These equations are supplemented by the condition that each time the potential V_i equals a threshold value, the neuron i emits a spike, and the value of its potential is set instantaneously to zero. The threshold value is taken here to equal unity. Finally, τ_0 is the integration time constant of the synaptic current relative to that of V_i.

We first discuss the state of a spatially homogeneous network, i.e., $I_i^0 = I_0$. In the case of $I_0 > 1$, the network dynamics possesses a fully synchronized solution where all the neurons are phase locked [5,6]. This state is stable and is asymptotically reached from random initial conditions. In the case of $I_0 < 1$, the neurons are not excited without synaptic input (since the threshold is 1). This leads to the emergence of other states. In particular, in this regime the quiescent state $V_i = V < 1$, $\rho = 0$ is stable (see also [7–9]). Here we limit ourselves to the phase-locked state for $I_0 > 1$. In the phase-locked state all the neurons fire simultaneously. Equations (1) and (2) with $V_i = V$ describe a single neuron coupled to itself with an excitatory connection of strength K.

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The solution of these equations is a periodic state with $\rho(t) = \sum_n \delta(t - nT)$ and a mean-field current $I(t)$ that is given by the expression

$$I(t) = \tilde{K} \exp(-t/\tau_0), \quad 0 \le t < T \,, \tag{4}$$

where $\tilde{K} \equiv K/[1-\exp(-T/\tau_0)]$ and $I(t) = I(t+T)$. Note that I has a discontinuity at $t = nT$. The period of oscillations T is determined from a simple self-consistency equation requiring that $V(t \to T^-) = 1$. An example is shown in Fig. 1 for parameters $\tau_0 = 0.5$, $K = 0.1$, and $I_0 = 1.5$.

We focus on the effect of nonuniform local external currents on the phase-locked state. Let us assume that

$$I_i^0 = I_0 + \delta_i, \quad -\Delta < \delta_i < \Delta \,, \tag{5}$$

where Δ is the width of inhomogeneity, and take for simplicity a uniform distribution of δ_i. The results of numerical simulation [10] of such a network with $\Delta = 10^{-3}$ are shown in Fig. 2. The displayed spike density shows a substantial departure from full phase locking even for such a small dispersion in local frequencies. Furthermore, the results indicate that the spike density consists of two contributions with substantially different widths. Further analysis shows that the two components originate from two distinct subpopulations. The narrow component represents neurons that are frequency locked and phase locked to the current I with fixed small phase shifts which depend on the local value of I_i^0. The rest of the neurons are not locked with $I(t)$ and exhibit aperiodic patterns of firing. Nevertheless, as is evident from the form of ρ, most of them do retain a substantial degree of synchrony with I. In fact, they fire for a long time in close synchrony with $I(t)$ but undergo phase slips which occur on a time scale that is long relative to the period of I. The separation of the network into these two populations is illuminated in Fig. 2 (inset), where the mean frequency of firing of the different neurons is displayed versus their local external currents. The plateau corresponds to the frequency-locked population, which consists of the neurons with slow intrinsic frequency, i.e., $I_i^0 < I_c$. In contrast, the neurons with $I_i^0 > I_c$ are not frequency locked. These surprising results are the main subject of the paper.

To understand the behavior of the system, we first study the behavior of a single neuron, Eq. (1), under the influence of a fixed periodic current $I(t)$ with period T. We will then close the solution by demanding that I be consistent with the spike density ρ, as required by Eq. (2). The behavior of the neurons is best described by computing their phase, which is the time of their firing relative to the periodicity of the external current. These times are governed by a map $t_n \to t_{n+1} = \phi(t_n)$, where t_n is the phase of the neuron at the nth period of $I(t)$ [11], defined as its time of spiking relative to the nth peak of $I(t)$. The map $t' = \phi(t)$ can be easily computed from Eq. (1) which yields

$$e^{t'} = e^{-T} \int_t^{t'+T} d\tau \, e^{\tau} (I(\tau) + I^0) \,. \tag{6}$$

The map depends, of course, on the value of I^0 and therefore differs for different neurons. We will denote this dependence by writing $\phi = \phi(t; I^0)$.

For small values of I^0, ϕ has a stable fixed point $t^0 = \phi(t^0; I^0)$, which is given by

$$I^0 = \frac{1 - e^{-T} \int_0^T e^{\tau} I(t' + t^0) d\tau}{1 - e^{-T}} \,. \tag{7}$$

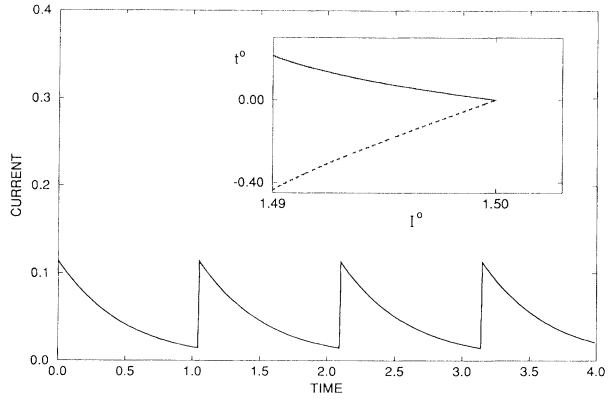

FIG. 1. The mean-field current $I(t)$ in the fully synchronized state of the homogeneous network, given by Eq. (4). Inset: the function $t_0(I_0)$ (see text). Solid line, stable branch; dashed line, unstable branch. Parameters are $\tau_0 = 0.5$, $K = 0.1$, and $I_0 = 1.5$.

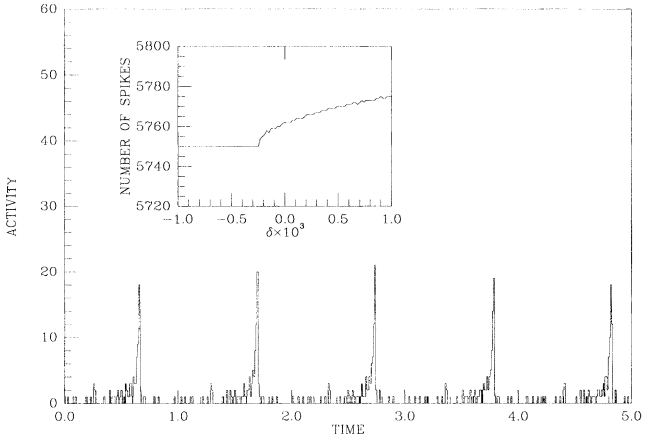

FIG. 2. Simulated activity of an inhomogeneous network of 100 neurons, with $\Delta = 10^{-3}$. Other parameters are the same as in Fig. 1. The number of spikes emitted by the network in the time interval $dt = 0.01$ is plotted vs time. The transient up to $t = 5000$ was discarded. Inset: the number of spikes emitted by a neuron in a time window of length 6000, against the deviation of its local external current I^0 from the mean I_0.

The stable fixed phase t^0 is monotonically decreasing with I^0. It has a minimum value, denoted as $t_{\min}$, corresponding to a maximal value of I^0, denoted as I_c, for which there is a fixed point. The function $t^0(I^0)$ for $I(t)$ of Eq. (4) is depicted in the inset of Fig. 1. Note that there are two branches: an unstable and a stable branch. The two branches meet at $t_{\min}$. As can be seen, in this case $t_{\min} = 0; I_c = I_0$. It should be noted that the two branches of t^0 depicted in the inset meet with finite slopes. This is the consequence of the discontinuity of the current at $t = t_{\min}$. In the case of a continuous $I(t)$ the two branches meet smoothly, implying that in this case

$$\frac{dI^0}{dt^0} = 0, \quad t^0 = t_{\min} . \tag{8}$$

Neurons with $I_i^0 > I_c$ are not phase locked and have a spiking rate bigger than $1/T$. Thus, if $I_0 - \Delta < I_c < I_0 + \Delta$, the system is divided into two populations, phase locked and unlocked, and the spike density is given by the sum of two corresponding densities: $\rho(t) = \rho^l(t) + \rho^u(t)$. The fraction of phase-locked population, N_s, is related to I_c via

$$I_c = I_0 + (2N_s - 1)\Delta . \tag{9}$$

The behavior of the unlocked neurons with small values of $\delta I^0 \equiv I^0 - I_c > 0$ can be understood by linearizing $\phi(t^0; I^0)$ about the point $t^0 = t_{\min} = 0; I^0 = I_c$. Performing this expansion, using Eq. (6), one obtains for $I(t)$ of Eq. (4),

$$\begin{aligned} t_{n+1} &= a_+ t_n - b_+ \delta I^0, \quad 0 < t << 1 , \\ t_{n+1} &= a_- t_n - b_- \delta I^0, \quad 0 < -t << 1 , \end{aligned} \tag{10}$$

where $a_\pm = \partial\phi(0^\pm; I_c)/\partial t$ and $b_\pm = \partial\phi(0^\pm; I_c)/\partial I^0$. These coefficients can be easily evaluated using Eq. (6). They obey $0 < a_+ < 1$ and $a_- > 1$, $b_\pm > 0$. For $\delta I^0 < 0$, the map has a fixed point, representing phase-locked behavior. For $\delta I^0 > 0$ the phase t does not converge to a fixed point. Starting from an initial phase t_0, $\delta << t_0$, it will approach the origin in time $n \propto \ln(t_0/\delta)$. It spends time of order $|\ln\delta|$ near the origin, and then slips. The probability of spikes at a phase t_0 is roughly proportional to dn/dt_0; hence the spike density of the unlocked neurons has the shape $\rho_\pm^u(t) \propto |t|^{-1}$, $\Delta << |t| << 1$. A more precise derivation of this result is as follows. In the regime $\Delta << t$ we can neglect the additive δI^0 terms in Eq. (10) and the form of the map $\phi(t)$ is therefore the same for all neurons. The only spike density that is invariant to simultaneous linear transformations of all t_i is of the form $\rho(t) \propto |t|^{-1}$. Thus we obtain

$$\rho_\pm^u(t) = \left(\frac{(1 - N_s)C_\pm}{|\ln\Delta|}\right) \frac{1}{|t|}, \quad \Delta << |t| < 1 . \tag{11}$$

Here C_+ and $C_- \equiv 1 - C_+$ are the relative fractions of spikes of unlocked neurons to the right and to the left of the point $t = 0$, respectively. It follows that $\rho(t) \sim 1/|\ln(\Delta)|$ for $t \sim 1$, so that the spikes of the unlocked neurons are mostly concentrated around $t = nT$. The coefficients $C_\pm$ can be found from the condition that the number of spikes of the unlocked neurons appearing in the peak of the spike distribution (11) after the transformation (10) equals the number of spikes leaving this peak. This requirement leads to the relation

$$C_+ \ln a_+ = -C_- \ln a_- . \tag{12}$$

Up until now we have computed the distribution of phases of a population of neurons with a spread of local external currents, responding to a periodic, discontinuous current. We now turn to consider the self-consistency of $I(t)$ and $\rho(t)$ for the case of $|\Delta| << 1$. Our basic assumption is that in the limit of $N \to \infty$ the population of unlocked neurons produces at long time a spike density $\rho^u(t)$, which is periodic in t. The limit $\Delta \to 0$ presents two simplifications: First, all neurons have external currents I_i^0 close to $I_c \approx I_0$. Second, the self-consistent form of the mean-field current $I(t)$ is close to that of Eq. (4), except in the immediate vicinity of $t = 0$ where the discontinuity is smoothed by the spread of activity of the population. More detailed analysis shows that the most important deviation in the forms of $\rho(t)$ and $I(t)$ occurs in the regime of phases $0 < |t| \leq \Delta$. In particular, this is the scale at which the self-consistent current is smooth. On the other hand, most of the weight under $\rho^u(t)$ lies in the regime $\Delta << |t| << 1$. In this regime $\phi(t)$ remains the same as in the uniform case, Eq. (10), and therefore, Eq. (11) applies as well. The only difference is the value of N_s. In the case of the unperturbed current, the maximum value of local external current for which the phase is locked is $I_c = I_0$, implying that half the population is locked, i.e., $N_s = 1/2$.

The relative fraction of phase-locked neurons N_s in the case of the inhomogeneous network can be obtained from the self-consistency equation (9), if I_c is expressed in terms of $\rho(t)$ with the help of Eqs. (8) and (7):

$$I_c = \frac{1 - e^{-T} K\tau_0 \int_0^T e^{t'} \rho(t')dt'}{1 - e^{-T}} . \tag{13}$$

In the limit of $\Delta \to 0$ ρ collapses to

$$\rho(t) = [N_s + (1 - N_s)C_+]\delta(t - 0^+) + (1 - N_s)C_-\delta(t - T^-) .$$

Substituting this form in Eq. (13) yields

$$N_s = \frac{C_- + C_+ \exp(-T)}{C_-[1 - \exp(-T)]} - \frac{1}{K\tau_0 C_-[1 - \exp(-T)]} + \frac{I_0}{K\tau_0 C_-} . \tag{14}$$

This result implies that even in the limit $\Delta \to 0$, a finite fraction of neurons remains unlocked. However, as $\Delta \to$

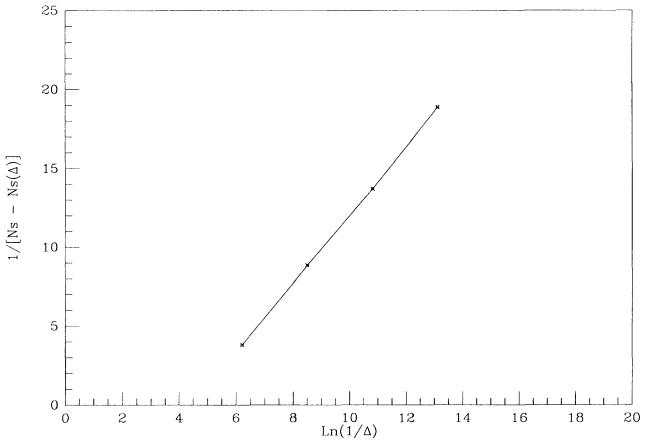

FIG. 3. Simulation results of the fraction of phase-locked neurons for various values of Δ. N_s is obtained as the relative width of the plateau in Fig. 2.

0, the time that they spend in the neighborhood of the origin grows so that the resultant distribution approaches a delta function as in the uniform system.

Further analysis shows that for small Δ the leading correction to the integral in Eq. (7) is logarithmic, because of the logarithmic dependence of ρ^u, Eq. (11). In particular, this yields

$$N_s - N_s(\Delta) \propto 1/|\ln(\Delta)| . \quad (15)$$

Figure 3 presents simulation results for N_s vs $|\ln(\Delta)|$ with the same parameters as in Fig. 2. A linear relationship is obtained for $N_s \approx 0.65$. This value is in a reasonable agreement with Eq. (14) which yields $N_s = 0.64$ for these parameters.

Our study shows that inhomogeneity in local frequency strongly affects the synchrony of the network. We expect that a similar behavior occurs for other kinds of local disorder, e.g., inhomogeneity in the time constants. The behavior of our system differs from other systems of globally coupled oscillators ([2,4]), where for weak disorder almost all the oscillators are phase locked. The origin of this difference lies in the pulselike nature of the interaction in our model, which is reflected by the discontinuity of the synaptic current (Fig. 1) in the fully synchronized state. In reality, the interaction is expected to depend continuously on the state of the coupled neurons. It is thus important to understand the extent of the validity of our results in systems where the interaction is a continuous but sharply varying function of the activity of the presynaptic neuron. A qualitative analysis shows that in this situation there is a crossover between an inhomogeneous regime with a behavior similar to that described in this paper and a homogeneous regime at

$$\Delta \approx 1/\max\{dI/dt\} . \quad (16)$$

An example of a system with continuous interactions that exhibits the above mentioned crossover is given by an integrate-and-fire model in which the synaptic current has a finite rise time. Instead of Eq. (2), $I(t)$ is given in this model by

$$I(t) = K \int_0^t d\tau \{\exp[-(t-\tau)/\tau_0] - \exp[-(t-\tau)/\tau_1]\}\rho(\tau) . \quad (17)$$

The original model is recovered in the limit where the rise time constant τ_1 vanishes. Here we consider the case $0 < \tau_1 << \tau_0$. In this case, following a spike in the network, the current rises continuously with $\max\{dI/dt\} \sim \tau_1$. Unlike our original model, this network is not phase locked in the absence of disorder, i.e., $N_s = 0$ for $\Delta = 0$. Here the effect of turning on the disorder is mixed. Above a small critical value of $\Delta \approx \tau_1$, the disorder first serves to pin the phases yielding a nonzero value of N_s. As Δ further increases, $N_s(\Delta)$ reaches a maximum and then decreases. In the regime of $\Delta >> \tau_1$, the value of N_s and the behavior of ρ are indistinguishable from that of $\tau_1 = 0$ described here. It is interesting to note that a similar nonmonotonic dependence of N_s on Δ was recently observed also in a network of globally coupled neurons modeled by the realistic Hodgkin-Huxley model, with weak disorder [12]. Finally, our analysis of Eq. (14) shows that when both τ_0 and τ_1 are zero N_s approaches 1, which is consistent with the recent results of Ref. [13] for the case of two neurons.

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