

## Phase Synchronization in Ensembles of Bursting Oscillators

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We study the effects of mutual and external chaotic phase synchronization in ensembles of bursting oscillators. These oscillators (used for modeling neuronal dynamics) are essentially multiple time scale systems. We show that a transition to mutual phase synchronization takes place on the bursting time scale of globally coupled oscillators, while on the spiking time scale, they behave asynchronously. We also demonstrate the effect of the onset of external chaotic phase synchronization of the bursting behavior in the studied ensemble by a periodic driving applied to one arbitrarily taken neuron. We also propose an explanation of the mechanism behind this effect. We infer that the demonstrated phenomenon can be used efficiently for controlling bursting activity in neural ensembles.

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At present our knowledge of the phenomenon of phase synchronization of chaotic oscillators is experiencing rapid growth (for a review see [1]). The concept of chaotic phase synchronization (CPS) lies in the coincidence of *characteristic time scales* of coupled systems, while the amplitudes stay uncorrelated [2]. Although CPS in ensembles of systems with one pronounced time scale has been studied quite scrupulously, much less is known on the coherent behavior of systems with multiple time scale dynamics. Such systems, typically combining fast and slow dynamics, are frequently encountered in physics, chemistry, and biology. One of the most challenging and inspiring problems here is an explanation of synchronization and revealing its role in neurobiological systems, where multiple time scale (spiking and bursting) chaotic behavior is often observed. Typical examples are: (i) the Central Pattern Generator [3], which is known to produce common rhythmic bursting, while its individual neurons, if isolated, would show irregular bursts, (ii) thalamic neurons during periods of drowsiness, inattentiveness, and sleep [4], and (iii) midbrain dopaminergic neurons, which exhibit spiking and bursting [5]. Coherent oscillations in such systems are thought to play a substantial role in information processing [6]. During the last decade mutual synchronization in small and large neural ensembles has been studied in much detail. It has been observed in numerical [7,8], electronic [8], and real neurobiological experiments [9]. Finally, very recently a mechanism behind regularization and synchronization of chaotic bursts in neural ensembles has been given an explanation [10]. However, the role of CPS in these processes still remains unclear.

Effects of an imposed periodic signal on the dynamics of an isolated neuron or a small group of neurons have also been studied theoretically and experimentally. They show an impressive variety, ranging from giving rise to either quasiperiodic or chaotic temporal patterns to external phase locking phenomenon [11]. At the same time,

the problem of controlling the dynamics of medium- and large- size neural ensembles by means of external synchronization has been analyzed far less. Based on the known possibility of synchronizing an ensemble of first order phase oscillators by a common external periodic force [12], one may conjecture that if each bursting oscillator in a large ensemble is forced by a common periodic signal, a global phase locking should occur. However, arranging such common driving may turn out to be quite problematic in physiological systems. Thus, developing the technique of controlling the dynamics of a neural ensemble by means of a *locally imposed* signal, i.e. by a signal applied to a single element, looks far more advantageous. Another way of controlling neural ensembles, namely, enhancing or suppressing of synchronization, can be realized by means of a time-delayed feedback [13].

In this Letter we first investigate the effect of the onset of mutual CPS in an ensemble of chaotically bursting oscillators. Then we demonstrate that an external periodic driving applied to an arbitrarily taken oscillator can lock the phases and the frequencies of all elements in the ensemble. The autonomous ensemble is modeled by a set of the mean field coupled two-dimensional maps (constructed to mimic neuronal dynamics), proposed, and studied in [10]:

$$\begin{cases} x(i, n+1) = \frac{\alpha_i}{1+x(i, n)^2} + y(i, n) + \frac{\varepsilon}{N} \sum_{j=1}^N x(j, n), \\ y(i, n+1) = y(i, n) - \sigma_i x(i, n) - \beta_i, \end{cases} \quad (1)$$

where  $x(i, n)$  and  $y(i, n)$  are, respectively, the fast and slow dynamical variables of the  $i$ th oscillator,  $N$  is the number of oscillators in the ensemble, and  $\varepsilon$  is the strength of global coupling. The slow evolution of  $y(i, n)$  is due to the small values of the positive parameters  $\beta_i$  and  $\sigma_i$  (each one of the order 0.001). The parameters  $\alpha_i$  define the dynamics of the fast variables  $x(i, n)$  of the individual maps, which we interpret as amplitudes. In [10]  $\alpha_i$  were taken random and uniformly distributed in [4.1, 4.9] so

that each uncoupled map produces either chaotic bursts or continuous chaotic oscillations. In [10] it was shown that if the mean field coupling is quite strong, initially uncorrelated, time scale different neuronal bursts become synchronized. A common rhythm of bursting also appears in the elements, which produces continuous chaotic spiking when isolated. We would like to stress that in this kind of ensemble, the onset of coherent bursting cannot be described in terms of CPS because no characteristic time scale of bursting can be found in a continuously spiking neuron. In contrast, we set  $\alpha_i$  random and uniform in [4.1, 4.4],  $\beta_i = \sigma_i = 0.001$  obtaining thus bursting oscillators (which we also call neurons following [10]). In Fig. 1(a), typical realizations of the fast dynamics (the evolution of the  $x$  variable) of isolated oscillators are shown. The existence of a characteristic time scale allows us to introduce the phase and the frequency of bursting in each oscillator. The phase of bursting oscillations in the  $i$ th oscillator  $\varphi(i, n)$  increases linearly between the moments  $n_k$  at which bursts start ( $k$  being a number of a burst) and gains a  $2\pi$  growth over each time interval  $n_{k+1} - n_k$ .  $\varphi(i, n) = 2\pi k + 2\pi(n - n_k)/(n_{k+1} - n_k)$ . The frequency of bursts is an average speed of the phase increase:  $\Omega_i = \lim_{n \rightarrow \infty} [\varphi(i, n) - \varphi(i, 0)]/n$ .

**Mutual synchronization.**—First we show that the transition to mutual phase synchronization takes place on the bursting time scale of globally coupled oscillators, while on the spiking time scale they behave asynchronously. The onset of mutually synchronized bursting in the studied ensemble has much in common with the classical example of global phase entrainment of phase oscillators [14,15]. The transition to synchronized bursting is observed as the coupling between the oscillators is in-

creased [Fig. 1(b),  $N = 1000$  here). A nonzero mean field is formed and its oscillations make neurons develop a common rhythm. Remarkably, only the slow time scale (i.e. bursting) dynamics becomes coherent. Spikes remain uncorrelated and do not substantially contribute to the mean field (close to periodic) dynamics. The frequencies  $\Omega_i$  of the bursting oscillators calculated for different coupling illustrate the appearance of a synchronized cluster and its gradual increase in size (Fig. 2). The asymptotic (very large  $n$ ) behavior of the order parameter  $r = |\sum_{k=1}^N e^{i\varphi(k,n)}|/N$  indicates a second-order phase transition to coherence (Fig. 3), typical for mean field coupled phase oscillators [15].

**External synchronization.**—Now we demonstrate the effect of the onset of external chaotic phase synchronization of the bursting behavior in the studied ensemble by the periodic driving applied to an arbitrarily taken neuron. Once all the neurons have developed a common time scale of bursting behavior, synchronizing this rhythm by applying a local periodic driving may be attempted. We fix the mean field coupling coefficient at  $\varepsilon = 0.1$ , so that neurons stay mutually phase synchronized. Then we subject one arbitrarily taken neuron  $i^*$  to a harmonic signal so that its equation for the  $x$  variable reads as

$$x(i^*, n + 1) = \frac{\alpha_{i^*}}{1 + x(i^*, n)^2} + y(i^*, n) + \frac{\varepsilon}{N} \sum_{j=1}^N x(j, n) + d \sin \omega n. \tag{2}$$

The equations for the  $y$  variable of this neuron and those for the other neurons remain unchanged. The number of bursting oscillators in the ensemble was  $N = 50$ . Having varied the frequency of the driving signal  $\omega$  and taken three values of its amplitudes  $d$ , we observe the effect of

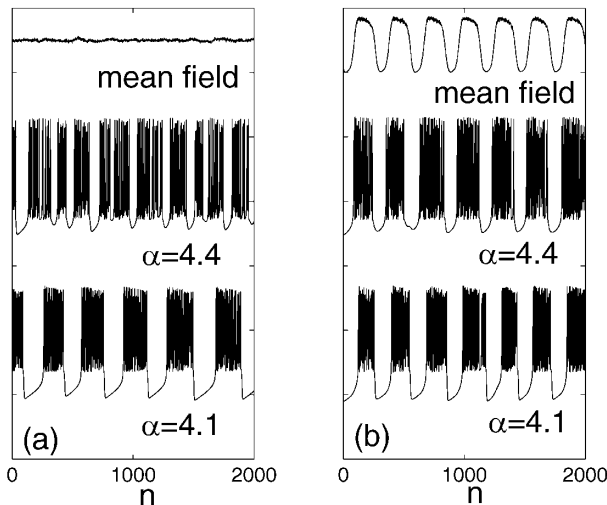


FIG. 1. Realizations of  $x(i, n)$  of two neurons and the mean field from (a) an uncoupled ensemble ( $\varepsilon = 0$ ) and (b) a coupled ensemble ( $\varepsilon = 0.04$ , synchronization of bursts is achieved), in the absence of external signal ( $d = 0$ ). Different values of  $\alpha_i$  are implemented,  $\sigma = \beta = 0.001$ . Here  $N = 1000$ .

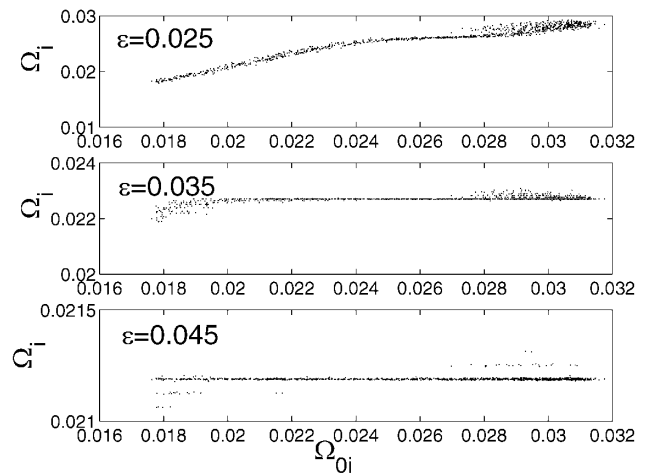


FIG. 2. Frequencies of bursting in the mean field coupled ensemble vs those at the zero mean field coupling show a growth of a synchronization cluster as the coupling coefficient  $\varepsilon$  is gradually increased. The external signal is absent ( $d = 0$ ),  $N = 1000$ .

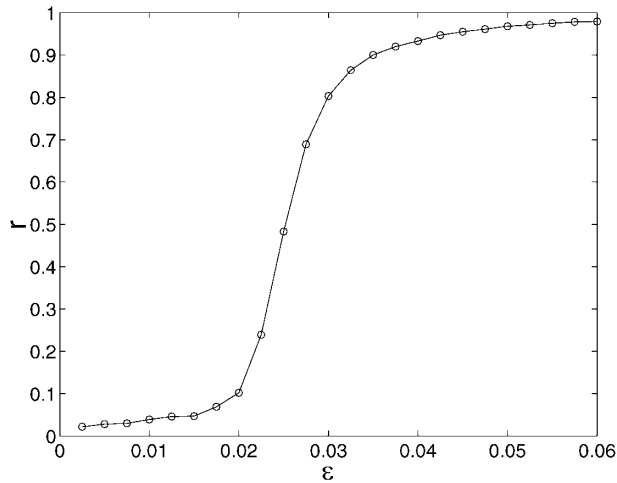


FIG. 3. The order parameter  $r$  vs mean field coupling coefficient  $\varepsilon$  indicates a second-order phase transition to CPS of bursting ( $N = 1000$ ).

external phase locking (i.e., external CPS) of bursts (Fig. 4). This transition to external CPS in this ensemble is characterized by the following important properties: i) At relatively large amplitudes of driving outside the synchronization region the driven oscillator switches to a frequency different from those of the driving signal and of the other mutually synchronized oscillators. At smaller amplitudes of driving this effect is far less pronounced. ii) The synchronization plateau enlarges basically in the direction of the higher frequencies of the driving as its amplitude increases. iii) For  $d > \varepsilon$ , a further increase of the amplitude of driving does not enlarge the synchroni-

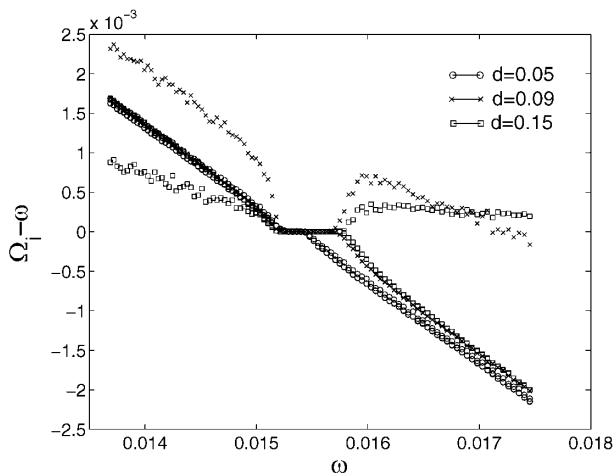


FIG. 4. The difference between frequencies of bursting in oscillators and the driving frequency vs the driving frequency for three values of the driving amplitude demonstrate external CPS in the ensemble of bursting oscillators. The fixed mean field coupling  $\varepsilon = 0.1$  ensures mutual phase synchronization between oscillators in the absence of the driving signal. Here  $N = 50$ .

zation plateau significantly. In Fig. 5 we show the dynamics of phases of the oscillators inside (the phases are locked) and outside (the phases are drifting) the synchronization region. Computations confirmed that the shown synchronization persists whatever neuron is driven. This observation makes the proposed controlling strategy very advantageous in the context of experiments and possible applications. We also obtained the described effect for all generated realizations of the randomly distributed parameters  $\alpha_i$ , for which the unforced ensemble demonstrates mutually synchronized bursting.

The observed effects can be explained as follows: The influence of the slow variable  $y$  on the fast one  $x$  results in triggering and terminating a burst in an isolated oscillator [10]. An imposed periodic signal precipitates a burst of a neuron into a quiescent regime when positive and delay it when negative, which tends to synchronize the driven neuron. The mean field coupling term reflects averaged individual dynamics of neurons. Suppose that the external frequency exceeds that of the mutually synchronized autonomous ensemble. Then the periodic signal will fasten oscillations of the driven neuron. When the whole ensemble is about to start or stop bursting, the global dynamics becomes very sensitive to changes in the amplitudes of individual oscillators. If the driven neuron starts (stops) bursting, the abrupt change of its amplitude increases (decreases) the mean field value, pushing the other neurons toward bursting (silence). Thus higher frequencies win the competition with the lower ones in the considered neural ensemble. Quite the opposite, should the frequency of the driving signal be smaller than that of the autonomous ensemble, only tiny synchronization effects can be expected. From this follows the frequency asymmetry of the synchronization plateaus. We would like to stress that a local driving can result in external CPS of the whole ensemble only when the oscillations in

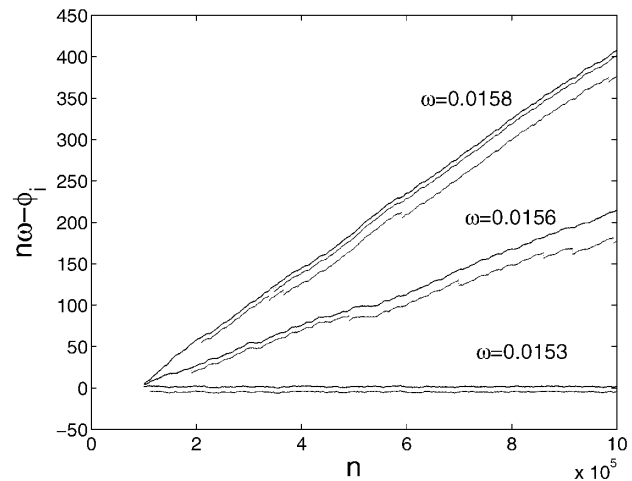


FIG. 5. Drifting ( $\omega = 0.0158$  and  $\omega = 0.0156$ ) and locked ( $\omega = 0.0153$ ) phases  $\phi_i$  of all  $N$  bursting oscillators illustrate the transition to CPS in a neural ensemble ( $N = 50$ ).

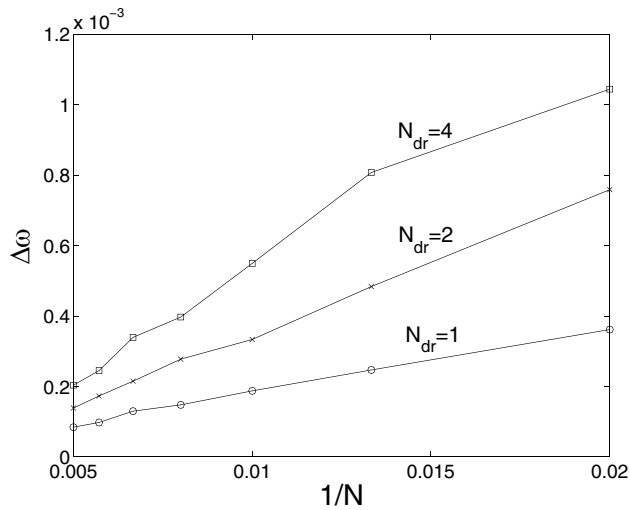


FIG. 6. The average size of synchronization plateau  $\Delta\omega$  (see details in the text) for the fixed mean field coupling  $\varepsilon = 0.2$  and the driving amplitude  $d = 0.15$  vs the inverse number of oscillators in the ensemble  $1/N$  for different number of driving neurons  $N_{dr}$ .

the autonomous ensemble are mutually synchronized. Oscillators that are not bursting coherently with the driven oscillator are not susceptible to the driving signal. The contribution of the driven oscillator to the mean field is proportional to  $\varepsilon/N$  and does not depend upon the amplitude of the driving  $d$ . That explains why the increase of the synchronization region is limited when  $d$  is increased and  $\varepsilon$  is fixed. It also follows that the synchronization region should decrease as the number of oscillators in the ensemble grows. To analyze this dependence, we calculate the size of the synchronization plateau  $\Delta\omega$  for fixed mean field coupling  $\varepsilon = 0.2$  and driving amplitude  $d = 0.15$  in ensembles with different number of oscillators  $N$ . For each size value  $N$  we generate 100 realizations of random parameters  $\alpha_i$ ,  $i = \overline{1, N}$  and average the obtained sizes of synchronization regions. In Fig. 6 we observe that the synchronization region size scales as  $\Delta\omega \propto 1/N$ . For control purposes one can overcome this problem by applying the same driving signal, not to one, but to several arbitrarily taken neurons  $N_{dr}$  (see Fig. 6 for two and four driving neurons).

In conclusion, we have analyzed the role of CPS in the onset of mutually synchronized dynamics in a globally coupled ensemble of bursting oscillators. This transition occurs on the time scale of bursting, while on the time scale of spiking, the synchrony does not appear. Features typical of the second-order phase transition to synchronization have been observed. We have also demonstrated the effect of the onset of external CPS in the ensemble of bursting oscillators by a sinusoidal driving signal applied to an arbitrarily taken oscillator. We also propose an explanation of the mechanism behind this effect. Our

results show that the studied ensemble can be effectively synchronized by the frequency of the driving signal that is higher than that of the autonomous ensemble, while lower driving frequencies are practically unable to cause this synchronization. We infer that this phenomenon can be used for efficiently controlling bursting activity in neural ensembles. The next step in the study of the found phenomenon will be its observation in electronic and biological experiments. We also anticipate that this mechanism of regulating neural behavior may be identified in natural neurobiological systems.

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