Dephasing and Bursting in Coupled Neural Oscillators

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(Received 28 February 1995)

Usually, diffusive coupling of nonlinear oscillators in one dynamical variable leads to synchronization of oscillators. We study a model of coupled neural oscillators in which simple diffusive coupling in voltage, counterintuitively, leads to dephasing of oscillators. We examine the general conditions under which dephasing through diffusive interaction will occur. We show that such systems with dephasing limit cycles lead to a new burstinglike behavior: oscillators switch between high and low oscillation amplitude. This occurs because the interaction is such that oscillators tend to synchronize for sufficiently small oscillation amplitude, while they tend to desynchronize once their oscillation amplitude has become large.

PACS numbers: 87.10.+e, 05.45.+b, 82.40.Bj

The study of coupled oscillators is one of the fundamental problems in theoretical physics with applications in many different fields [1]. Recently, this field has seen renewed interest due to the possible role of coupled oscillator systems in neural processing [2]. Neurophysiological measurements in a variety of settings have shown stimulus-dependent synchronization and desynchronization of oscillatory activity in various cortical systems [3–5]. In theoretical studies of the dynamics of coupled oscillator systems, it is rather straightforward to obtain synchronization of oscillations through excitatory couplings. There is considerably more debate as to what causes such oscillator systems to desynchronize again. The mechanisms proposed and studied include time delayed interactions [6,7], dephasing through chaotic rather than periodical oscillators [8], fast-changing synaptic coupling [9], noise [10–12], and changes of excitability [13].

Recently, Sherman and Rinzel [14] observed dephasing and antiphase phase locking in simulations of two diffusively coupled neurons, but did not provide a satisfactory explanation for this effect. In this paper, we discuss the general conditions under which diffusive coupling in *one* dynamical variable can lead to dephasing of limit cycle oscillators. (The word *diffusive* is taken from the language used in the study of coupled chemical oscillators. In this context, it just refers to the fact that interacting oscillators adjust their interacting dynamical variables toward each other.) For a model system of coupled neurons, we show that simple transmembrane voltage coupling between large numbers of simple two-dimensional limit cycle oscillators may alone suffice to periodically generate synchronous oscillations, and subsequently destroy the coherency again.

Our studies are based on the Morris-Lecar system [15]. The Morris-Lecar system is a simplified version of the Hodgkin-Huxley system [16] using only two dynamical variables v and w instead of four dynamical variables in the Hodgkin-Huxley model. Nonetheless, it exhibits most of the dynamical features of the Hodgkin-Huxley model, including stimulus-dependent excitability and oscillatory behavior. Compared to the Bonhoeffer–van der Pol model [17], it has more parameters which allows reproduction of a greater variety of dynamical modes of the Hodgkin-Huxley model, while still offering the simplicity of a two-dimensional system. The Morris-Lecar system is written down as

$$\frac{dv}{dt} = -\bar{g}_{Ca} m_\infty(v)(v - 1) - \bar{g}_K w(v - v_K) - \bar{g}_L(v - v_L) + I, \tag{1}$$

$$\frac{dw}{dt} = f\,\frac{[w_\infty(v) - w]}{\tau_w(v)}, \tag{2}$$

with

$$m_\infty(v) = 0.5[1 + \tanh\{(v - v_1)/v_2\}], \tag{3}$$

$$w_\infty(v) = 0.5[1 + \tanh\{(v - v_3)/v_4\}], \tag{4}$$

$$\tau_w(v) = 1/\cosh\{(v - v_3)/(2v_4)\}. \tag{5}$$

v can be thought of as the transmembrane voltage of the neuron, while w represents its recovery variable and I is the excitation current. When not specified otherwise, we use $v_1 = -0.01$, $v_2 = 0.15$, $v_3 = 0.1$, $v_4 = 0.145$, $\bar{g}_{Ca} = 1.0$, $\bar{g}_K = 2.0$, $\bar{g}_L = 0.5$, $v_K = -0.7$, $v_L = -0.5$, and $f = 1.15$ throughout this study.

This system has been extensively studied and characterized by Rinzel and Ermentrout [18]. At very low or very high values of the stimulation current I, it has a single stable fixed point. At intermediate values of the stimulation current, a stable limit cycle can appear through a Hopf bifurcation, a heteroclinic connection, or a homoclinic connection. For the above-indicated parameter values, the limit cycle of the system arises at $I = 0.0730$ through a homoclinic connection, as can be seen in Fig. 1.

We are interested in the behavior of large coupled ensembles of such oscillatory systems. In order to describe the effect of coupling between one oscillator i and all N other oscillators, we modify Eq. (1) by adding a linear voltage-coupling term $\frac{1}{N}\sum_{j=1}^{N} k(v_j - v_i)$.

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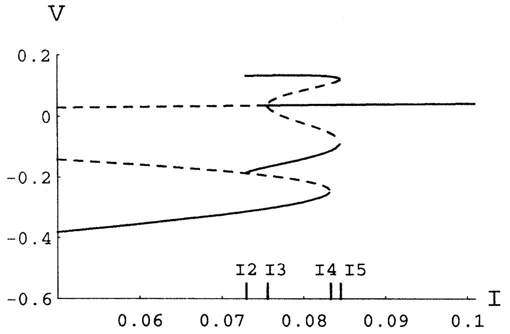

FIG. 1. Bifurcation diagram showing the voltage as a function of the excitation current I. For $I_1 < I < I_2$, a stable stationary point, a saddle point, and an unstable stationary point coexist. At I_2, a stable limit cycle appears through a homoclinic connection at the saddle point. At I_3 an unstable limit cycle bifurcates out of the unstable stationary point, and disappears together with the stable limit cycle at I_5. At I_4, the lower stationary point and the saddle point disappear via a saddle-node connection. $I_1 = -0.0207$, $I_2 = 0.0730$, $I_3 = 0.0756$, $I_4 = 0.0833$, and $I_5 = 0.0845$.

For the homoclinic limit cycle obtained for our standard parameters, we find analogous to Sherman and Rinzel's results [14] that diffusive coupling in v space between two oscillators *on the limit cycle* will actually lead to a relative dephasing of their oscillations.

Following [1], we can express the motion of a phase-space vector $X = (v, w)$ of a system along a limit cycle by a phase variable ϕ which can be extended to any other phase-space point X using the concept of isochrones. $X_0(\phi)$ then denotes the point on the limit cycle with phase ϕ. The interaction of two oscillators, ϕ_1 and ϕ_2, can then be quantified by considering the evolution of their phase difference $\delta\phi = (\phi_1 - \phi_2)$. In the limit of weak interactions, the average relative phase shift can be expressed as

$$\frac{\partial}{\partial t}\delta\phi = \Gamma(\delta\phi) = \frac{1}{2\pi}\int_{\phi'=0}^{2\pi} Z(\phi')p(\phi',\delta\phi)d\phi', \quad (6)$$

where $p(\phi',\delta\phi) = p(X_0(\phi'), X_0(\phi' + \delta\phi))$ describes the rate of change of the state vector X of an oscillator due to interaction with another oscillator ahead by a phase difference of $\delta\phi$, and the sensitivity function $Z(\phi) = (\mathrm{grad}_X \phi)_{X=X_0(\phi)}$ gives the change of phase along the limit cycle caused by the change of X.

In Fig. 2(a), we plotted the value of the integrand of Eq. (6) as a function of $t = \phi'$ for $\delta\phi = 0.001\pi$ and $k = 1$. During a limit cycle period, the interaction alternately causes the phase difference $\delta\phi$ to increase or decrease, where the increase dominates. The phases of increasing and decreasing phase difference are identified on the limit cycle in Fig. 2(b) by the four points A, B, C, and D. The dephasing is especially strong on the segment from C to D, where the oscillator approaches the saddle point along its stable manifold. In Fig. 2(c) we show the total $\Gamma(\delta\phi)$ as a function of the phase difference $\delta\phi$. From this figure, it follows that diffusive interaction will dephase interacting oscillators and stabilize them at a phase difference of π.

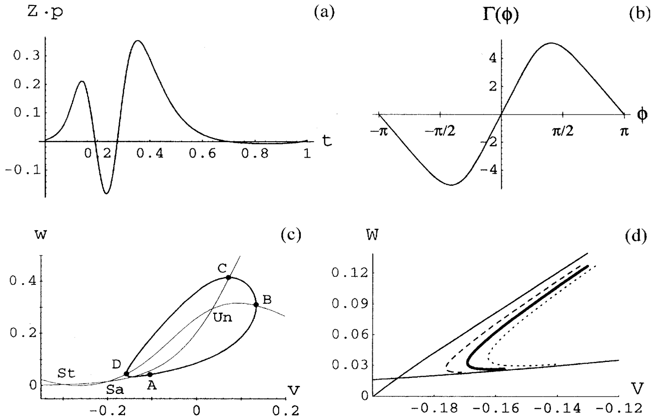

FIG. 2. (a) Phase shift induced by diffusive coupling as a function of time during one limit cycle oscillation, normalized for $k = 1$. $\delta\phi = 0.01\pi$ and $I = 0.075$. (b) Average relative phase shift [antisymmetric part of $\Gamma(\phi)$] as a function of the phase difference ϕ of two coupled oscillators. $k = 1$, $I = 0.075$. (c) Picture of null clines and limit cycle. The points A, B, C, D indicate where the interaction shown in (a) changes between dephasing and synchronizing. The points St, Sa, and Un are the stable fixed point, saddle point, and unstable fixed point, respectively. (d) Enlargement of (c), with the saddle point as well as its stable and unstable manifolds. Three trajectories are shown, integrated each for $dt = 3$: the unperturbed trajectory along the limit cycle, and trajectories following perturbations to the inside (dotted line) or outside (broken line) of the limit cycle.

These results counter the widespread intuitive assumption that diffusive-type coupling in one of the variables, in this case the voltage v, should lead to gradual synchronization of the phases along the limit cycle. Closer scrutiny, however, shows that this does not necessarily have to be the case: At any given moment, two interacting systems typically differ not only in their v variable, but also in their w variable. When they adjust their voltages v toward each other, they will also depart from the limit cycle. On a linear stretch of a small part of the limit cycle as shown in Fig. 3, this will mean that one system will be pushed inside the limit cycle and the other one outside the limit cycle. The effect of being pushed away from the limit cycle can have a much larger effect on the phase than the fact that they approach each other in the v variable [19]. If, as in Fig. 3(c), the lagging system is pushed into a region with slower parallel velocity, it can actually be delayed relative to the advanced system.

If, on the other hand, the systems differ only in the v variable, as in Fig. 3(a), or if the lagging system is pushed away from the limit cycle into a region of faster phase velocity, then the overall effect of the interaction will be to synchronize the oscillators. In actual limit cycle systems, it is not immediately obvious whether the net result of diffusive coupling will lead to synchronization or desynchronization. However, for most limit cycle systems, the synchronizing action will prevail. In order for the desynchronizing action to prevail, some rather strong deformations of the phase flow in the vicinity of the limit cycle are required. In the present case, the presence of a saddle point just outside the homoclinic limit cycle means that systems lagging behind the other oscillators on the stretch *CD* of the limit cycle will be pushed toward the outside of the limit cycle. They then follow a trajectory that passes much closer to and more slowly by the saddle point.

While the saddle point causes the deformations of the phase flow and thus leads to desynchronizing interaction, the main features of the phase flow in the vicinity of the limit cycle continue to remain unchanged even after the saddle point disappears in a saddle-node connection at $I_4 = 0.0833$. We therefore find that oscillators on the limit cycle desynchronize for any value of I for which the homoclinic limit cycle exists.

If we change the standard parameter values such that the limit cycle appears through a heteroclinic connection ($f \rightarrow 1/3$) or a Hopf bifurcation ($f \rightarrow 0.2, v_3 \rightarrow 0, v_4 \rightarrow 0.3, \bar{g}_{Ca} \rightarrow 1.1$) [18], we find that diffusive interaction in v synchronizes oscillators for all values of $I_2 < I < I_5$ for which those limit cycles exist.

It has to be pointed out that for the considered Morris-Lecar system with our standard parameter values, the v coupling becomes desynchronizing only when the orbits of the oscillators are on or close to the limit cycle. If the oscillators are placed close to the unstable stationary point, they will initially synchronize their oscillations while spiraling away from the stationary point. Only when they start approaching the limit cycle will the v interaction be dephasing.

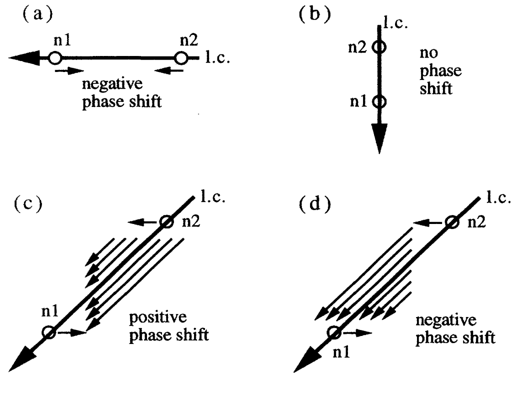

FIG. 3. Schematic illustrations of how attractive interaction in one phase variable (horizontal direction) can lead to synchronizations or dephasing of interacting oscillators: (a) The interaction in v leads to synchronization when $\Delta w = 0$. (b) The interaction does not act when $\Delta v = 0$. (c) and (d) The interaction can lead to a dephasing or faster synchronization on stretches of the limit cycle where $\Delta v \neq 0 \neq \Delta w$. In (c), the lagging oscillator is pushed into a region of slower phase velocity, leading to dephasing, while in (d), the lagging oscillator is pushed into a region of faster phase velocity, leading to enhanced synchronization.

For a system of not-too-weakly coupled oscillators, we obtain a scenario as depicted in a series of snapshots of the phase space shown in Fig. 4: After a distribution of synchronized oscillators has reached the limit cycle, it will start to dephase. When the oscillators have spread out along the limit cycle, they will start to pull each other towards the center of the limit cycle. As the distribution contracts toward the stationary point, the oscillators will synchronize and start again to spiral out toward the limit cycle.

This sequence of sychronization and desynchronization can lead to a variety of complex long-time behavior patterns, as illustrated in Fig. 5, which shows the average voltage ($1/N \sum v_i$) of a population of interacting neurons as a function of time. It should serve only as an indication of the variety of temporal patterns that can be obtained; a more exhaustive overview over the modes of dynamical behavior will be presented elsewhere.

Two-dimensional dynamical systems usually do not show complicated dynamical behavior beyond simple limit cycle oscillations. The reason for the complicated behavior seen in Fig. 5 for the mean-field-type treatment of populations of coupled limit cycle oscillators is that coupling of the oscillator v to the average voltage $\bar{v}$ introduces *one additional variable* into the dynamical system. Dephasing interaction between oscillators ensures that the average voltage $\bar{v}$ will deviate from the voltage of the individual oscillators, making the mean-field system of the oscillators effectively three dimensional.

Similar complex time behavior has been found for abstract systems of coupled two-dimensional Ginzburg-Landau oscillators [20]. It has been speculated before [1] that chemical turbulence might arise in systems where diffusive coupling leads to dephasing, because the effective phase diffusion constant is proportional to $\Gamma'(0)$. In the present work, we obtain this condition

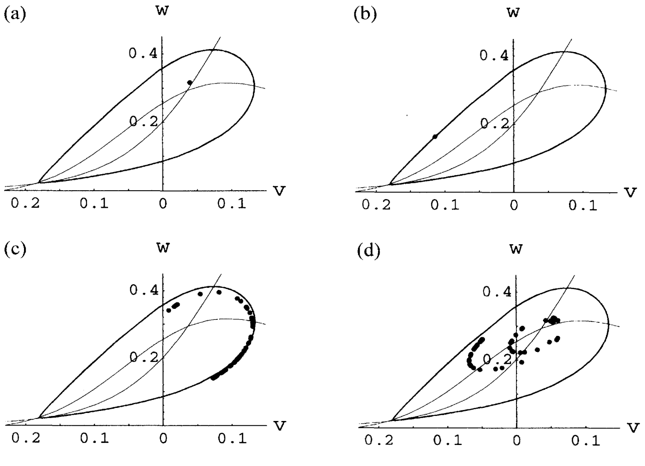

FIG. 4. Typical distribution of oscillators in phase space ($I = 0.075$, $k = 0.2$, and $N = 100$): (a) synchronized cluster spirals toward limit cycle; (b) synchronized cluster spirals on limit cycle; (c) cluster desynchronize on limit cycle; and (d) desynchronized cluster spirals toward stationary point.

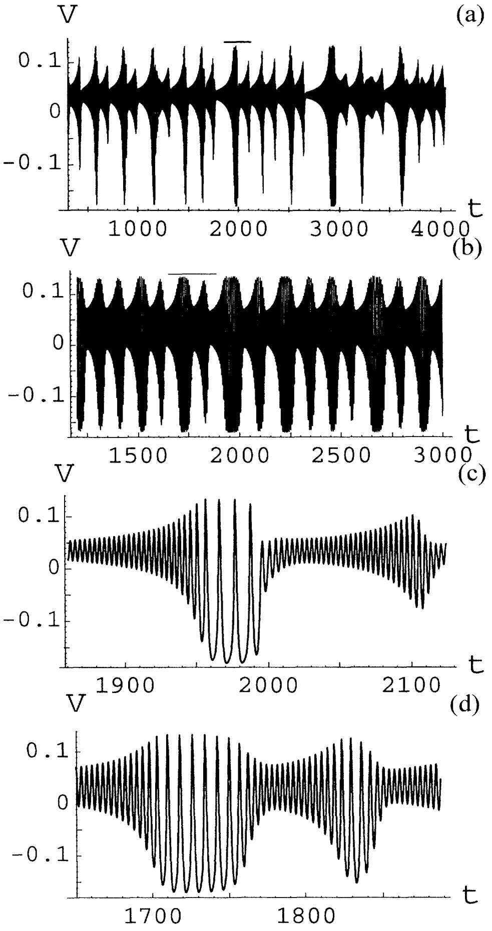

FIG. 5. Voltage traces (average voltage) for large systems of coupled Morris-Lecar neuron. (a) $I = 0.0735$, $k = 0.2$, (b) $I = 0.075$, $k = 0.4$. (c) and (d) show enlargements from (a) and (b), respectively. $N = 200$.

in a model with physiologically motivated dynamical variables, which should provide guidance toward finding chemical turbulence in biochemical systems.

Further related studies of neuronal oscillator synchronization and dephasing were reported in [21,22]. This work is not concerned with detailed modeling of cortical architecture, as is Hansel and Sompolinsky [23]. Our aim was only to show that complex time behavior can naturally arise in simple networks of simple oscillators, if one takes into account that simple diffusive coupling can lead to alternating phases of synchronization and desynchronization of coupled neuronal oscillators. We emphasize that we obtain bursting in coupled two-dimensional oscillator systems *without* any need to add a third slow variable to the oscillator dynamics.

S. K. H. wants to express thanks for the hospitality of the Physics Department of Kyoto University during the conduct of the present investigation. C. K. was supported by a postdoctoral fellowship from the European Union Science and Technology Fellowship Programme in Japan (STF8). The authors would like to thank S. Shinomoto, H. Kook, S. Kim, N. Nakagawa, S. Kitsunesaki, and K. Watanabe for useful discussions. Numerical computation in this work was supported by the Yukawa Institute for Theoretical Physics.

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[1] Y. Kuramoto, *Chemical Oscillations, Waves, and Turbulence* (Springer-Verlag, Berlin, 1984).

[2] C. von der Malsburg, Report No. 81-2, Max-Planck-Institute for Biophysical Chemistry, Göttingen, Germany, 1981 (unpublished).

[3] C. M. Gray, P. König, A. K. Engel, and W. Singer, Nature (London) **338**, 334 (1989).

[4] R. Eckhorn *et al.,* Biol. Cybernet. **60**, 121 (1988).

[5] C. S. Skarda and W. Freeman, Behavioural and Brain Sciences **10**, 161 (1987).

[6] T. B. Schillen and P. König, in *Nonlinear Dynamics and Neuronal Networks,* edited by H. G. Schuster (VCH Publishers, New York, 1991).

[7] Y. Kuramoto, Physica (Amsterdam) **50D**, 15 (1991).

[8] D. Hansel and H. Sompolinsky, Phys. Rev. Lett. **68**, 718 (1992).

[9] C. von der Malsburg and W. Schneider, Biol. Cybernet. **54**, 29 (1986).

[10] H. G. Schuster and P. Wagner, Biol. Cybernet. **64**, 77 (1990); **64**, 83 (1990).

[11] O. Sporns, G. Tononi, and G. M. Edelman, Proc. Natl. Acad. Sci. U.S.A. **88**, 129 (1991).

[12] H. Sompolinsky, D. Golomb, and D. Kleinfeld, Proc. Natl. Acad. Sci. U.S.A. **87**, 7200 (1990); Phys. Rev. A **43**, 6990 (1991).

[13] C. Kurrer and K. Schulten, Phys. Rev. E. **51**, 6213 (1995).

[14] A. Sherman and J. Rinzel, Proc. Natl. Acad. Sci. U.S.A. **89**, 2471 (1992).

[15] C. Morris and H. Lecar, Biophys. J. **35**, 193 (1981).

[16] A. L. Hodgkin and A. F. Huxley, J. Physiol. London **117**, 117 (1952).

[17] R. Fitzhugh, Biophys. J. **1**, 445 (1961).

[18] J. Rinzel and G. B. Ermentrout, in *Methods in Neuronal Modeling,* edited by C. Koch and I. Segev (MIT Press, Cambridge, 1989).

[19] C. Kurrer and K. Schulten, Physica (Amsterdam) **50D**, 311 (1991).

[20] N. Nakagawa and Y. Kuramoto, Prog. Theor. Phys. **89**, 313 (1993); Physica (Amsterdam) **80D**, 307 (1995).

[21] D. Golomb and J. Rinzel Physica (Amsterdam) **72D**, 259 (1994); X.-J. Wang and J. Rinzel, Neural Comp. **4**, 84 (1992).

[22] D. Hansel, G. Mato, and C. Meunier, Europhys. Lett. **23** (5), 367 (1993).

[23] D. Hansel and H. Sompolinsky (to be published).