Synchronization in a chaotic neural network with time delay depending on the spatial distance between neurons

Guoning Tang,* Kesheng Xu, and Luoluo Jiang
College of Physics and Technology, Guangxi Normal University, Guilin 541004, China
(Received 19 March 2011; revised manuscript received 11 June 2011; published 14 October 2011)

The synchronization is investigated in a two-dimensional Hindmarsh-Rose neuronal network by introducing a global coupling scheme with time delay, where the length of time delay is proportional to the spatial distance between neurons. We find that the time delay always disturbs synchronization of the neuronal network. When both the coupling strength and length of time delay per unit distance (i.e., enlargement factor) are large enough, the time delay induces the abnormal membrane potential oscillations in neurons. Specifically, the abnormal membrane potential oscillations for the symmetrically placed neurons form an antiphase, so that the large coupling strength and enlargement factor lead to the desynchronization of the neuronal network. The complete and intermittently complete synchronization of the neuronal network are observed for the right choice of parameters. The physical mechanism underlying these phenomena is analyzed.

DOI: 10.1103/PhysRevE.84.046207 PACS number(s): 05.45.Xt, 87.19.lm, 87.19.lj

I. INTRODUCTION

Synchronization of coupled nonlinear oscillators is a widespread phenomenon occurring in physical [1], chemical [2], engineering [3,4], biological [5], and medical [6] systems, e.g., between cardiac and respiratory systems or between interacting neurons or coupled laser systems with feedback. However, synchronization is not always desirable. For example, synchronization of individual neurons leads to the emergence of pathological rhythmic brain activity in Parkinson's disease, essential tremor, and epilepsies [7,8]. Therefore it is interesting to find various methods to suppress or facilitate the synchronization of coupled nonlinear oscillators.

In recent years, the synchronization of time-delay systems has attracted a lot of attention [9–24]. Anticipating synchronization [9,10], generalized synchronization [14], phase synchronization [15–17], complete synchronization [18,19], exponential synchronization [20], chaotic burst synchronization [21], globally clustered chimera [22], and zero lag synchronization [23] are observed in time-delay coupled systems. It is found that the delayed interaction between two or among many oscillators can suppress or facilitate synchronization [12,13], and result in oscillation quenching as well [24]. Although the synchronization in a coupled neuronal network with time-varying delay [25–27], distributed time delays [16,28,29], or mixed time-varying delays [30,31] has been extensively studied, how the length of time delay depending on the spatial distance between neurons affects the synchronization of chaotic neuronal networks with time-delayed coupling has not yet been explored.

Signal transmission time delays are unavoidable in spatially distributed coupled oscillator systems. A careful measurement of axonal conduction delays in the mammalian neocortex showed [32–34] that they could be as small as 0.1 ms and as large as 44 ms, depending on the type and location of the neurons. Thus the time delays are spatially distributed due to different distances and finite signal transmission speeds between different pairs of coupled neurons in the brain. In this paper we investigate the synchronization of a two-dimensional (2D) neuronal network with global time-delayed coupling. The network is made of identical Hindmarsh-Rose (HR) neurons placed randomly inside the chaotic regime. Considering the fact that the distance between coupled neurons may delay the receiving of signals, we take the length of time delay between two neurons as $\tau \propto d$, where d denotes the spatial distance between them. We mainly focus on how the length of time delay per unit distance affects the synchronization of the neuronal network. It is found that the time delay always impedes the synchronization of the neuronal network. This results in the abnormal membrane potential oscillations (AMPOs) in neurons when the coupling strength and length of time delay per unit distance are large enough. Specifically, the AMPOs for the symmetrically placed neurons are in antiphase. The birth of these AMPOs induces desynchronization of the network. The complete synchronization (CS) and intermittently complete synchronization (ICS) are observed if the related parameters are properly chosen. It has been generally accepted that the large coupling strength can easily achieve CS in a globally coupled neuronal network. However, in our work we interestingly find that large coupling strength always induces the desynchronization of the neuronal network for large length of time delay per unit distance.

The paper is organized as follows: In Sec. II, we introduce a 2D neuronal network model with global time-delayed coupling. Section III is devoted to investigate the synchronization of the neuronal network, and the detailed numerical simulation results are given. The physical mechanism underlying desynchronization is analyzed in Sec. IV. A brief conclusion is presented in the last section.

II. TWO-DIMENSIONAL NEURONAL NETWORK MODEL

We consider identical HR neurons placed on a two-dimensional square lattice with global time-delayed coupling, which is the self-time-delay coupling used by Dhamala *et al.* in Ref. [12]. The neuronal network is composed of $N = n' \times n'$ neurons. The time evolution of the neuron labeled (i,j) is

*tangguoning@sohu.com

described by the set of equations [35]

$$\dot{u}_{i,j} = v_{i,j} - au_{i,j}^3 + bu_{i,j}^2 - w_{i,j} + I_{\text{ext}} + F_{i,j}(t), \quad (1a)$$

$$\dot{v}_{i,j} = c - du_{i,j}^2 - v_{i,j}, \quad (1b)$$

$$\dot{w}_{i,j} = r[s(u_{i,j} + \chi) - w_{i,j}], \quad (1c)$$

$$F_{i,j}(t) = k \sum_{l,m=1}^{n'} [u_{l,m}(t - \tau_{lm,ij}) - u_{i,j}(t - \tau_{lm,ij})], \quad (1d)$$

$$i,j,l,m = 1,2,\ldots,n',$$

where variable $u_{i,j}$ represents the membrane potential, variables $v_{i,j}$ and $w_{i,j}$ represent fast and slow ion currents in the neuronal dynamics, respectively, $F_{i,j}(t)$ is global coupling term, k is coupling strength, and $\tau_{lm,ij}$ is the length of time delay. The parameters are defined as $a = 1, b = 3, c = 1, d = 5, r = 0.006, s = 4, \chi = 1.56$, and $I_{\text{ext}} = 3.0$ where an isolated neuron exhibits a chaotic behavior. In this model, the neuron (i,j) receives the signal from the neuron (l,m) after a time delay $\tau_{lm,ij}$. On the other hand, the membrane potential, generated in the soma, spreads forward to the axon and back into a dendritic terminal. This is referred to as a back-propagating membrane potential. There is a corresponding back-propagating membrane potential for different received signals. The back-propagating membrane potential $u_{i,j}(t - \tau_{lm,ij})$ means the state of the neuron (i,j) delayed by $\tau_{lm,ij}$. In the global coupling term, the corresponding feedback signal is generated from the difference of received signal and the corresponding back-propagating signal.

In our numerical simulation, the model equations are integrated by using the fourth-order Runge-Kutta algorithm with time step $\Delta t = 0.001$. The total integration time length of each run of simulation is 5000. The synchronization of the neuronal network with time delay that is taken to be the same for all neurons had been investigated in Ref. [12]. We now consider the time delay that only depends on the spatial distance between neurons. The length of time delay between a pair of neurons is given as

$$\tau_{lm,ij} = \text{int}[pd_{lm,ij}]\Delta t = \text{int}[p\sqrt{(l-i)^2 + (m-j)^2}]\Delta t. \quad (2)$$

Where int[$\cdot$] denotes the integer part of [$\cdot$], $d_{lm,ij}$ is the distance between neurons (i,j) and (l,m), and p is an enlargement factor which takes an integer value for simplicity. The dynamics of an isolated HR neuron and the initial state of the neuronal network are shown in Fig. 1.

In order to study the synchronization degree of the neuronal network, we define a synchronization parameter $\delta(t)$ and its average value as

$$\delta(t) = \frac{1}{N-1} \sum_{i,j=1}^{n'} |u_{i,j} - u_{1,1}|, \quad (3)$$

$$\delta_0 = \frac{1}{1000} \int_{4000}^{5000} \delta(t)dt, \quad (4)$$

where $\delta(t) = 0$ indicates the precisely complete synchronization. But, $\delta(t)$ is generally not equal to zero. The more synchronous the neuronal network, the smaller is the synchronization parameter $\delta(t)$. Hence, in this situation, the neuronal network is considered to be CS when the network fulfills $\delta_0 < 10^{-3}$.

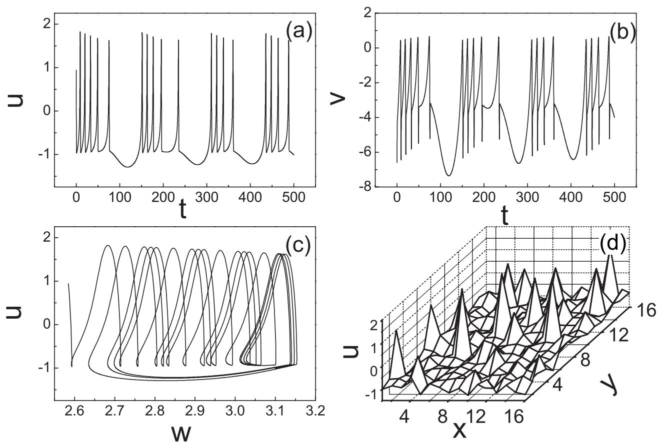

FIG. 1. (a)–(c) Chaotic bursting of a single HR neuron. (a), (b) Chaotic time series of the variables u and v, respectively. (c) Phase trajectory on the $u - w$ phase plane. (d) Initial pattern of the neuronal network.

III. NUMERICAL STUDIES

Generally speaking, $u_{l,m}(t - \tau_{lm,ij})$ and $u_{l,m}(t - \tau_{lm,i'j'})$ are different due to time delay, so $\sum_{l,m=1}^{n'}[u_{l,m}(t - \tau_{lm,ij})]$ in Eq. (1d) is different for major neurons. The computation load thus increases sharply as the size of the network increases. In order to reduce the computation load, we apply a small network with $N = 16 \times 16$ neurons to investigate the synchronization of the neuronal network. Coupling strength k and enlargement factor p are treated as adjustable parameters. We vary p in [0,50] and k in [0,0.3]. The numerical results show that the CS of the neuronal network can be achieved at $k \geqslant 0.004$ for $p = 0$ (i.e., $\tau = 0$). The increase of k only facilitates synchronization. However, we note that large k and p always lead to the desynchronization of the neuronal network with the time delay.

Figure 2 shows the time evolution of the synchronization parameter $\delta(t)$ for $p = 13$ and different coupling strengths. Figures 2(a) and 2(f) indicate that the neuronal network is chaotic. Figures 2(c) and 2(d) show that the network has reached the CS state. Obviously, the $\delta(t)$ of the latter

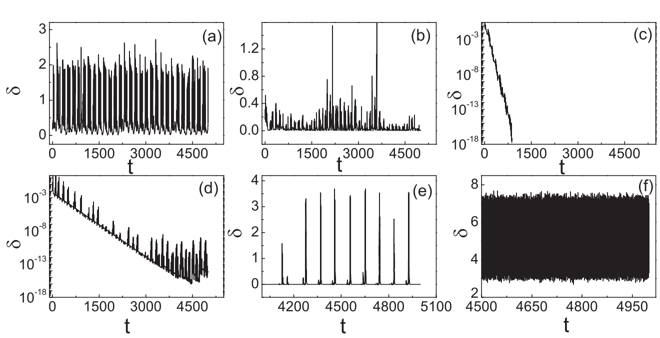

FIG. 2. Time evolution of synchronization parameter δ for $p = 13$ and different values of k. (a) $k = 0.0005$, (b) $k = 0.0044$, (c) $k = 0.0075$, (d) $k = 0.069$, (e) $k = 0.072$, (f) $k = 0.20$.

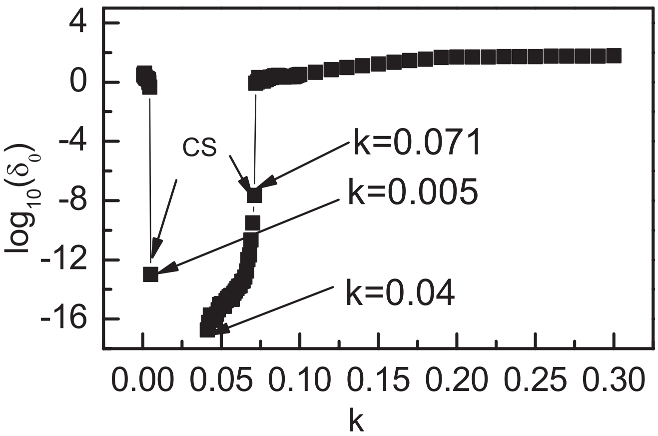

FIG. 3. $\log_{10}(\delta_0)$ vs k for $p = 13$.

oscillates irregularly around a small value and does not vanish. Figure 2(b) corresponds to a transition state between spatiotemporal chaos and CS. The spike and burst synchronization between neurons occur sporadically in the transition state. Figure 2(e) shows that $\delta(t)$ approaches intermittently zero. This means that the network reaches ICS state. In Fig. 3, $\log_{10}(\delta_0)$ is plotted against k for $p = 13$. Figure 3 shows that when k is small the neuronal network is chaotic. In the range of value of k [0.0055,0.039], $\delta_0 = 0$ is achieved. For further increased k, δ_0 would increase gradually. Finally, this results in the desynchronization of the neuronal network, generating ICS or spatiotemporal chaos. The CS region is clearly observed from Fig. 3. However, the ICS region is not quite clear.

To get more insight into the ICS state, we plot in Fig. 4 the three-dimensional patterns of variable u at different time moments for $k = 0.072$ and $p = 13$. The corresponding time series of variables u of specially chosen two neurons are shown in Fig. 5. It is observed that the abnormal high frequency oscillation of membrane potential arises during the after-spiking stage of neuron. Moreover, the AMPOs of symmetrically placed neurons have inverse phase. The AMPOs of neurons closely resembles the early after-depolarizations in cardiac myocytes, which are a type of triggered activity that arises before action potential repolarization is completed. In contrast to the former, the latter involves dynamic changes in intracellular Ca^{2+}, such as spontaneous Ca^{2+} release from the sarcoplasmic reticulum and activation of membrane currents

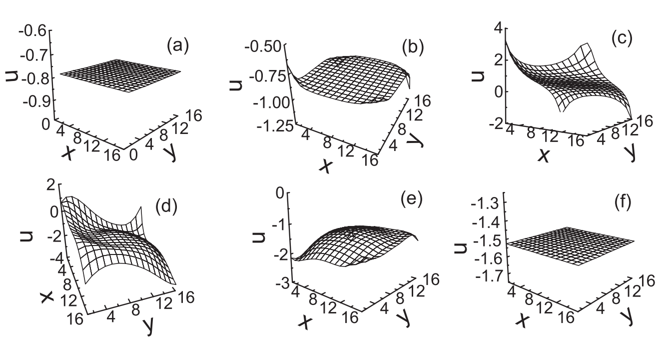

FIG. 4. Three-dimensional patterns of variable u at different time moments for $k = 0.072$ and $p = 13$. (a) $t = 535$, (b) $t = 545$, (c) $t = 553$, (d) $t = 554.9$, (e) $t = 555.5$, (f) $t = 565$.

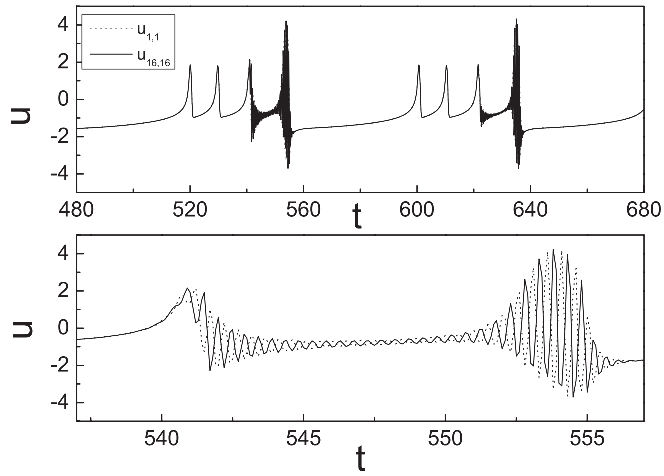

FIG. 5. Time series of variables u of neurons (1,1) and (16,16) for $k = 0.072$ and $p = 13$. Solid and dotted lines correspond to neurons (16,16) and (1,1), respectively. Bottom diagram is the local blowup of top diagram.

through a Ca^{2+}-activated process [36]. The desynchronization of the neuronal network begins at the corners of the network with the square boundary and is induced by the AMPOs of neurons. The AMPOs in neurons vanish when the membrane potential goes back to its resting potential. Thus the CS of the network is restored. We find that both amplitude and oscillation duration of AMPO increase as coupling strength increases. The AMPOs would become very strong if coupling strength is large, so that the neuronal network cannot restore synchronization after membrane potential returns to the resting potential. The corresponding neuronal network is chaotic.

In Fig. 6 we plot the CS region in the p-k parameter plane. One can observe that the time delay does not lead to the synchronization of the neuronal network when coupling strength is outside the CS region of the neuronal network without time delay, i.e., it can not extend the CS region. On the contrary, the right border of the CS region shrinks as p increases. Moreover, δ_0 is not equal to 0 for $p > 1$ when coupling strength is close to the right border of the CS region. This implies that the time delay defined by Eq. (2) always

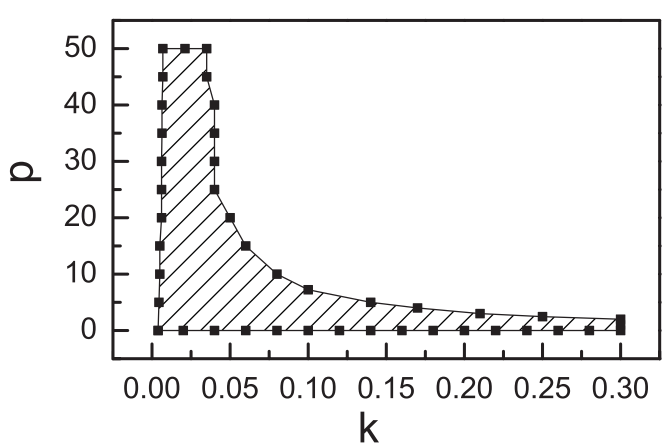

FIG. 6. The CS region in the p-k parameter plane. The shaded region represents CS while the blank region represents nonsynchronization.

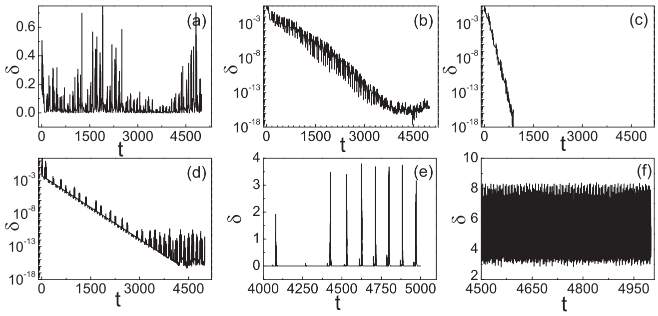

FIG. 7. Time evolution of synchronization parameter δ for $p = 13$ and different values of k. Triangular lattice with 16×16 grid points is applied. (a) $k = 0.0044$, (b) $k = 0.005$, (c) $k = 0.0075$, (d) $k = 0.069$, (e) $k = 0.073$, (f) $k = 0.20$.

suppresses the synchronization of the neuronal network, whereas the time delay that is independent of the spatial distance between neurons can either suppress or enhance the synchronization of a neuronal network, but does not induce the AMPOs in neurons [12]. The above phenomena will be explained in the next section.

So far our investigation has focused on square lattice, the square boundary of the network, and a chaotic oscillatory state. Now we investigate the effects of different factors on the synchronization of the neuronal network. When a square lattice is applied, we consider the different factors, such as the circular boundary of the network and the different chaotic or nonchaotic oscillatory states. We also apply a triangular lattice to investigate the synchronization of the neuronal network. The results obtained in all cases are similar to those shown in Fig. 2 when the number of neurons is 256 (see Fig. 7). The phenomena of CS, ICS, spatiotemporal chaos, and AMPO are also observed. The desynchronizations of the neuronal networks also begin at the corners of the network with the square boundary or at the circular boundary of the network. These factors only lead to a little change of the CS region shown in Fig. 6. However, we find that the number of neurons can significantly affect the synchronization of the neuronal network (see Fig. 8). Comparing Fig. 8 with Fig. 2, we observe that the increase of the neuronal network size causes the considerable shrinkage of the CS range.

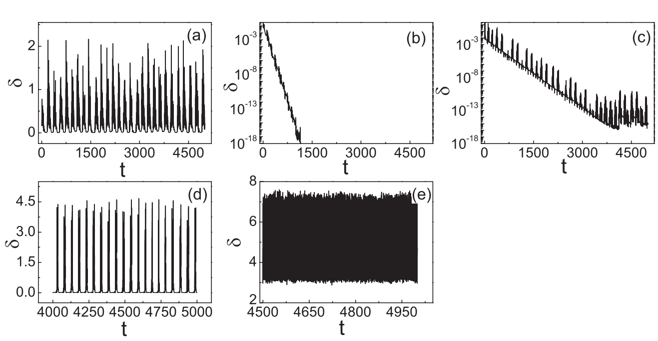

FIG. 8. Time evolution of synchronization parameter δ for $p = 13$ and different values of k. Square lattice with 32×32 grid points is applied. (a) $k = 0.001$, (b) $k = 0.002$, (c) $k = 0.012$, (d) $k = 0.015$, (e) $k = 0.05$.

IV. ANALYSIS OF DESYNCHRONIZATION

In order to understand the above results, we define the synchronization error between any two neurons in the network as

$$e^u_{ij,i'j'} = u_{i,j} - u_{i',j'}, \quad e^v_{ij,i'j'} = v_{i,j} - v_{i',j'},$$
$$e^w_{ij,i'j'} = w_{i,j} - w_{i',j'}. \tag{5}$$

Their dynamics in linear approximation can be derived from Eq. (1) as

$$\dot e^u_{ij,i'j'} = e^v_{ij,i'j'} - 3au_{i,j}u_{i',j'}e^u_{ij,i'j'} + b(u_{i,j}+u_{i',j'})e^u_{ij,i'j'} - e^w_{ij,i'j'} + \Delta F_{ij,i'j'}, \tag{6a}$$

$$\dot e^v_{ij,i'j'} = -de^u_{ij,i'j'}(u_{i,j}+u_{i',j'}) - e^v_{ij,i'j'}, \tag{6b}$$

$$\dot e^w_{ij,i'j'} = rse^u_{ij,i'j'} - re^w_{ij,i'j'}, \tag{6c}$$

$$\Delta F_{ij,i'j'} = F_{i,j} - F_{i',j'} = g_{ij,i'j'} + f_{ij,i'j'}, \tag{6d}$$

$$g_{ij,i'j'} = -Nke^u_{ij,i'j'}, \tag{6e}$$

$$f_{ij,i'j'} \approx k\Delta t \sum_{l,m=1}^{16}\sum_{n=L_{\min}+1}^{L_{\max}} \dot u_{l,m}(t-n\Delta t) + k\Delta t\sum_{n=0}^{Q}\left[N^n_{ij}\dot u_{i,j}(t-n\Delta t) - N^n_{i'j'}\dot u_{i',j'}(t-n\Delta t)\right], \tag{6f}$$

$$L_A = \mathrm{int}[p\sqrt{(l-i)^2+(m-j)^2}],$$
$$L_B = \mathrm{int}[p\sqrt{(l-i')^2+(m-j')^2}],$$
$$L_{\max} = \max(L_A,L_B), \quad L_{\min} = \min(L_A,L_B),$$
$$\text{for given } l \text{ and } m,$$
$$Q = \mathrm{int}[p\sqrt{(1-16)^2+(1-16)^2}] = \mathrm{int}[21.21p],$$
$$i,j,i',j' = 1,2,\ldots,16,$$

where N^n_{ij} is the number of $\dot u_{i,j}(t-n\Delta t)$. From Eq. (6) we draw conclusions as follows: (1) The synchronization errors $e^v_{ij,i'j'}$ and $e^w_{ij,i'j'}$ will approach zero if $e^u_{ij,i'j'}$ approaches zero. Therefore we use synchronization parameter δ defined by Eq. (3) to measure the synchronization degree of the neuronal network. (2) $\Delta F_{ij,i'j'}$ is composed of negative feedback term $g_{ij,i'j'}$ and time delay term $f_{ij,i'j'}$. $f_{ij,i'j'}$ is equal to zero when the time delay is not considered. The global coupling can easily achieve the CS of the neuronal network with $p = 0$ for large coupling strength. (3) When the time delay is considered, $f_{ij,i'j'}$ is generally not equal to zero except that the neuronal network exhibits the precisely complete synchronization. Therefore $f_{ij,i'j'}$ reduces the synchronization ability of the negative feedback term.

The number of terms in the right-hand side of Eq. (6f) is proportional to M $(=\sum_{l,m=1}^{16}\sum_{n=L_{\min}+1}^{L_{\max}} 1)$. The value of M is generally different for different neuron pairs. M reaches its maximum for the neurons at the opposite corners of the network with square boundary. The effect of the corresponding $f_{ij,i'j'}$ on $\Delta F_{ij,i'j'}$ reaches maximum value over $f_{ij,i'j'}$. The result indicates that the desynchronization of the neuronal

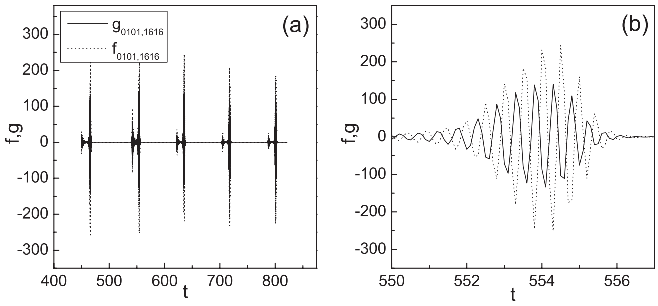

FIG. 9. (a) Time evolution of $f_{0101,1616}$ and $g_{0101,1616}$ for $k = 0.072$ and $p = 13$. (b) Local blowup of diagram (a).

network will begin at the corners of the network. This deduction is supported by the results shown in Fig. 4. On the other hand, $f_{ij,i'j'}$ depends on the time derivative of the variable u. When neuron is in the spiking stage, the time derivative of the variable u is large. Thus $f_{ij,i'j'}$ will sporadically be larger than $g_{ij,i'j'}$ if the coupling strength is large enough. In Fig. 9 we plot the time evolution of $f_{0101,1616}$ and $g_{0101,1616}$ for the specially chosen two neurons (1,1) and (16,16) at $k = 0.072$ and $p = 13$. It is observed that both $f_{0101,1616}$ and $g_{0101,1616}$ oscillate in opposite manners when the two neurons are in after-spiking stage. That is why large k and p lead to the AMPO of neuron and desynchronization of the neuronal network.

V. CONCLUSION

We have investigated the synchronization in the globally coupled square network with time delay depending on the spatial distance between two neurons. Numerical experiments demonstrate that the time delay always impedes the synchronization of the neuronal network. We have uncovered a phenomenon of the AMPO of the neuron, which is induced by time delay and emerges under the after-spiking stage of the neuron. A novel desynchronization and ICS are observed in the neuronal network since the AMPOs of the symmetrically placed neurons form an antiphase. Abnormal oscillation of membrane potential is a widespread phenomenon in biological systems. In heart diseases, cardiac myocytes can exhibit abnormal membrane potential oscillations, such as early afterdepolarizations, which are associated with lethal arrhythmias [37]. The abnormal neural oscillations in brain may lead to tics [38]. Hopefully, this study will contribute to the basic understanding of the AMPO occurring in biological systems, and can help one to achieve a good control over synchronization and desynchronization in an interacting population of neurons.

ACKNOWLEDGMENTS

This work was supported by the National Natural Science Foundation of China (Grant Nos. 11165004, 10765002, 10947011, and 11047012).

[1] P. Ashwin and J. Borresen, Phys. Rev. E **70**, 026203 (2004).
[2] I. Z. Kiss, Y. Zhai, and J. L. Hudson, Phys. Rev. Lett. **88**, 238301 (2002).
[3] R. Roy and K. S. Thornburg Jr., Phys. Rev. Lett. **72**, 2009 1994).
[4] G. Kozyreff, A. G. Vladimirov, and P. Mandel, Phys. Rev. Lett. **85**, 3809 (2000).
[5] A. B. Neiman and D. F. Russell, Phys. Rev. Lett. **88**, 138103 (2002).
[6] P. A. Tass, Phys. Rev. E **66**, 036226 (2002).
[7] M. Rosenblum and A. Pikovsky, Phys. Rev. E **70**, 041904 (2004).
[8] A. Nini, A. Feingold, H. Slovin, and H. Bergman, J. Neurophysiol. **74**, 1800 (1995).
[9] H. U. Voss, Phys. Rev. E **61**, 5115 (2000).
[10] G. Ambika and R. E. Amritkar, Phys. Rev. E **79**, 056206 (2009).
[11] M. G. Rosenblum and A. S. Pikovsky, Phys. Rev. Lett. **92**, 114102 (2004).
[12] M. Dhamala, V. K. Jirsa, and M. Ding, Phys. Rev. Lett. **92**, 074104 (2004).
[13] O. V. Popovych, C. Hauptmann, and P. A. Tass, Phys. Rev. Lett. **94**, 164102 (2005).
[14] E. M. Shahverdiev and K. A. Shore, Phys. Rev. E **71**, 016201 (2005).
[15] D. V. Senthilkumar, M. Lakshmanan, and J. Kurths, Phys. Rev. E **74**, 035205(R) (2006).
[16] X. Liang, M. Tang, M. Dhamala, and Z. Liu, Phys. Rev. E **80**, 066202 (2009).
[17] R. Suresh, D. V. Senthilkumar, M. Lakshmanan, and J. Kurths, Phys. Rev. E **82**, 016215 (2010).
[18] A. S. Landsman and I. B. Schwartz, Phys. Rev. E **75**, 026201 (2007).
[19] W. Kinzel, A. Englert, G. Reents, M. Zigzag, and I. Kanter, Phys. Rev. E **79**, 056207 (2009).
[20] Y. Sun, J. Cao, and Z. Wang, Neurocomputing **70**, 2477 (2007).
[21] Y. H. Zheng and Q. S. Lu, Physica A **387**, 3719 (2008).
[22] J. H. Sheeba, V. K. Chandrasekar, and M. Lakshmanan, Phys. Rev. E **79**, 055203(R) (2009).
[23] A. Englert, W. Kinzel, Y. Aviad, M. Butkovski, I. Reidler, M. Zigzag, I. Kanter, and M. Rosenbluh, Phys. Rev. Lett. **104**, 114102 (2010).
[24] D. V. Ramana Reddy, A. Sen, and G. L. Johnston, Phys. Rev. Lett. **80**, 5109 (1998).
[25] W. Wang and J. Cao, Physica A **366**, 197 (2006).
[26] W. Yu and L. Yao, J. Comput. Appl. Math. **206**, 679 (2007).
[27] C. Huang and L. Huang, Appl. Math. Lett. **19**, 126 (2006).
[28] T. Li, A. Songa, S. Fei, and Y. Guo, Nonlinear Anal. **71**, 2372 (2009).
[29] J. Zhou and Z. Liu, Phys. Rev. E **77**, 056213 (2008).
[30] Y. Tang, R. Qiu, J. Fang, Q. Miao, and M. Xia, Phys. Lett. A **372**, 4425 (2008).
[31] L. Sheng and H. Yang, Neurocomputing **71**, 3666 (2008).
[32] H. A. Swadlow, J. Neurophysiol. **68**, 605 (1992).
[33] H. A. Swadlow, J. Neurophysiol. **59**, 1162 (1988).
[34] H. A. Swadlow, J. Neurophysiol. **54**, 1346 (1985).
[35] J. L. Hindmarsh and R. M. Rose, Proc. R. Soc. Lond. B **221**, 87 (1984).
[36] C. H. Luo and Y. Rudy, Circ. Res.**74**, 1097 (1994).
[37] D. Sato, L. H. Xie, A. A. Sovari, D. X. Tran, N. Morita, F. Xie, H. Karagueuzian, A. Garfinkel, J. N. Weiss, and Z. Qu, Proc. Natl. Acad. Sci. **106**, 2983 (2009).
[38] D. G. Sukhodolsky, J. F. Leckman, A. Rothenberger, and L. Scahill, Eur. Child Adolesc. Psychiatry **16**, 51 (2007).