Physics Letters A 378 (2014) 2108–2112

Contents lists available at ScienceDirect

Physics Letters A

www.elsevier.com/locate/pla

Synchronization of map-based neurons with memory and synaptic delay

J.M. Sausedo-Solorio [a], A.N. Pisarchik [b,c,*]

[a] Universidad Autónoma del Estado de Hidalgo, Carretera Pachuca-Tulancingo Km. 4.5, 42074 Pachuca, Hidalgo, Mexico
[b] Centro de Investigaciones en Optica, Loma del Bosque 115, Lomas del Campestre, 37150 Leon, Guanajuato, Mexico
[c] Centre for Biomedical Technology, Technical University of Madrid, Campus Montegancedo, 28223 Pozuelo de Alarcon, Madrid, Spain

ARTICLE INFO

Article history:
Received 25 February 2014
Received in revised form 29 April 2014
Accepted 2 May 2014
Available online 10 June 2014
Communicated by C.R. Doering

ABSTRACT

Synchronization of two synaptically coupled neurons with memory and synaptic delay is studied using the Rulkov map, one of the simplest neuron models which displays specific features inherent to bursting dynamics. We demonstrate a transition from lag to anticipated synchronization as the relationship between the memory duration and the synaptic delay time changes. The neuron maps synchronize either with anticipation, if the memory is longer than the synaptic delay time, or with lag otherwise. The mean anticipation time is equal to the difference between the memory and synaptic delay independently of the coupling strength. Frequency entrainment and phase-locking phenomena as well as a transition from regular spikes to chaos are demonstrated with respect to the coupling strength.

1. Introduction

Biological neural networks consist of a large number of individual neurons interconnected in a complex manner usually via synapses through dendrodendritic microcircuits. The information processing tasks of neural networks are performed on the individual neuron level by generation of temporal sequences of action potentials, and then elaborated at mesoscales and macroscales by means of a network of neuron–neuron interaction. On the level of a single neuron, mechanisms and the nature of the neuron activity have been extensively investigated over the past decade; and the available literature is already redundant of rigorous and important results concerning the neuron ability to process and compute [1].

Synchronization of coupled neurons is relevant for coding and signal transmission allowing better understanding of the brain functionality and revealing distinctive features of some brain diseases. The interest in mathematical modeling of neuron synchronous behavior has significantly increased after real neurobiological experiments with two electrically coupled neurons [2,3], where various synchronization types have been identified. To simulate cooperative neuron dynamics, different models of coupled neurons based on either iterative maps [4–16] or differential equations [2,17–23] in various coupling configurations have been developed. Depending on both the coupling strength and the delay time, coupled neurons can be matched either in timings of their bursts (burst synchronization) [24], in phase [21], with lag, or anticipated [9,15,25,26]. Neuron dynamics were studied regarding intrinsic and external parameters including time constants, e.g., the influence of the rate of synaptic activation and deactivation on synchronization of bursting biological neurons. Furthermore, under specific conditions, intermittency between synchronized states was found when the time constant increased [3].

One of the important neuron functions is information transmission through a neuron network. This process is characterized by a certain delay time due to a finite velocity of the action potential propagation along the neuron axon and time lapses in dendritic and synaptic processes [27]. The delay in synaptic connections [28] is required for a neurotransmitter to be released from a presynaptic membrane, diffuse across the synaptic cleft, and bind to a receptor site on the postsynaptic membrane. On the other hand, feedback loops involving one or more neurons are ubiquitous in a nervous system [29]. The brain-stem feedback loops are thought to be responsible for short-term memory [30] that was predicted by Hermann Ebbinghaus in 1885 [31].

Since there are two different delay times, the interesting question arises: How do these time delays affect synchronization of synaptically coupled neurons? To answer this question, we explore one of the simplest neuron models, the Rulkov map [32,33]. Although this map is not explicitly referred to physiological processes in the membrane, it is capable of extraordinary complexity and quite specific neuron dynamics (silence, periodic spiking, and chaotic bursting), thus replicating a great deal of experimentally observed regimes [2,3,9], e.g., spike adaptation [34], routes from

* Corresponding author.
E-mail address: apisarch@cio.mx (A.N. Pisarchik).

http://dx.doi.org/10.1016/j.physleta.2014.05.003
0375-9601/

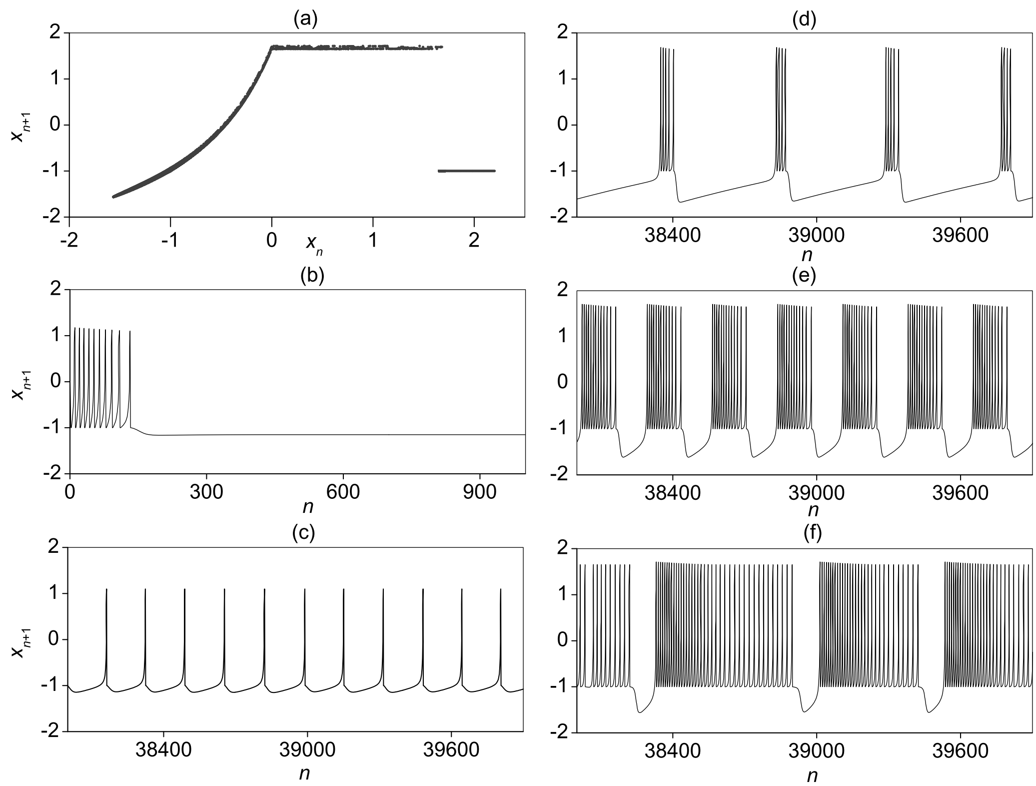

Fig. 1. (a) Function $f(x_n, y_n)$ for $\sigma = 0.3$ and (b–f) time series showing different dynamical regimes: (b) Silence after transients for $\sigma = -0.15$, (c) tonic spikes for $\sigma = -0.025$, and (d–f) bursts for (d) $\sigma = -0.30$, (e) 0.15, and (f) 0.30. $\alpha = 5.3$, $\mu = 0.001$.

silence to bursting mediated by subthreshold oscillations [35], emergent bursting [32], phase and antiphase synchronization with chaos regularization [9,33], as well as complete and burst synchronization [36–38].

Recently, Matias et al. [22,23] demonstrated a smooth transition from lag to anticipated synchronization of coupled Hodgkin–Huxley neurons [39] when the inhibitory synaptic conductance was increased. In the map-based models the time delay is independent of the coupling strength and only determined by the difference between the delay in coupling and neuron memory. In this work we will study the transition from lag to anticipated synchronization as a direct function of this difference. Being computationally more efficient than complex phenomenological models [39,40], the map-based models can improve qualitative understanding of the synchronous neuron behavior.

The paper is organized as follows. In Section 2 we review the theoretical framework of the Rulkov neuron and describe parameters explored in the model. Section 3 is devoted to synchronization of two coupled neurons; we show how two delay times affect synchronization. Finally, in Section 4 we conclude our results and outline a possible extension of this work.

2. Model equations

2.1. Dynamics of a single Rulkov neuron

The Rulkov map is defined by the following equations [9]

$$x_{n+1} = f(x_n, y_n), \tag{1}$$

$$y_{n+1} = y_n - \mu(x_n + 1) + \mu\sigma, \tag{2}$$

$$f(x_n, y_n) = \begin{cases} \alpha/(1 - x_n) + y_n & \text{for } x_n \le 0, \\ \alpha + y_n & \text{for } 0 < x_n < \alpha + y_n \text{ and } x_{n-1} \le 0, \\ -1 & \text{for } x_n \ge \alpha + y_n \text{ or } x_{n-1} > 0, \end{cases} \tag{3}$$

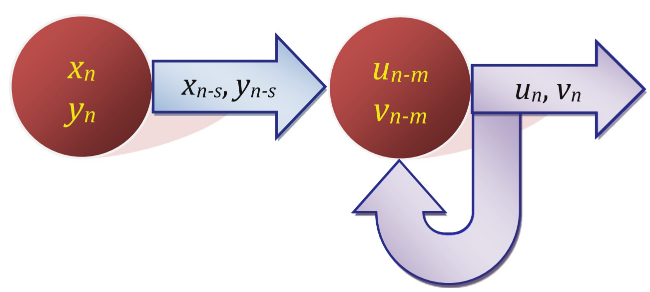

Fig. 2. Coupling scheme of two Rulkov neurons with synaptic delay s and memory m.

where x_n and y_n are the fast and slow variables and α, μ, and σ are intrinsic parameters. The map dynamics depends mostly on α and σ as shown in Fig. 1, where we plot the map function Eq. (3) [Fig. 1(a)] and typical times series illustrating different dynamical regimes [Figs. 1(b–f)]. The parameter σ regulates the neuron response under the action of the external dc bias current and synaptic inputs and therefore it is used as a control parameter to select a desired dynamical regime. For $\sigma < -0.3$ the neuron is in a silent state (subthreshold oscillations). For larger σ, the neuron generates repetitive spike bursts; the number of spikes in a burst train increases with σ, as seen from Figs. 1(c–f). Such a behavior of the Rulkov map mimics real neuron dynamics.

2.2. Two coupled Rulkov neurons

Now, we consider the scheme of two coupled Rulkov neurons shown in Fig. 2, where a memorized state of a postsynaptic neuron is coupled with a delayed state of a presynaptic neuron. While the presynaptic neuron is described by Eqs. (1)–(3), the postsynaptic neuron is modeled by the following equations

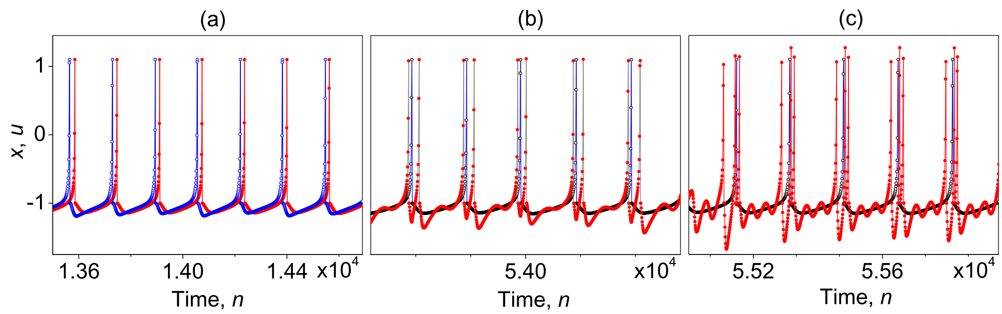

Fig. 3. Time series of fast variables of two coupled Rulkov maps demonstrating frequency entrainment to (a) 1:1 for $m = 16$, $s = 4$, and $\eta = 0.04$, (b) 2:1 for $m = 1$, $s = 0$, and $\eta = 0.009$, and (c) 3:1 for $m = 1$, $s = 0$, and $\eta = 0.02101$. $\alpha = 4.2$, $\mu = 0.001$, $\sigma = -0.025$. The open and closed dots connected by the blue and red lines correspond to the presynaptic and postsynaptic neurons, respectively. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

$$u_{n+1} = f(u_n, v_n + \beta_n), \tag{4}$$

$$v_{n+1} = v_n - \mu(u_n + 1) + \mu\sigma + \mu\sigma_n, \tag{5}$$

where u_n and v_n are the fast and slow variables of the postsynaptic neuron, the function f is defined by Eq. (3), and β_n and σ_n are related to an external stimulus under the action of the dc bias current and the synaptic input.

For simplicity, we consider only an electrical synaptic connection, i.e., the coupling causes an immediate physiological response of the postsynaptic neuron that depends linearly on the difference between membrane potentials of presynaptic and postsynaptic neurons [41,42]. Therefore, the coupling between the cells can be defined as

$$\beta_n = \sigma_n = \eta(x_{n-s} - u_{n-m}), \tag{6}$$

where s and m are, respectively, synaptic and memory delay times (in units of the number of iterations) and η is the synaptic coupling strength.

3. Synchronization

3.1. Frequency entrainment

We study synchronization of the two Rulkov neurons in the regime of tonic spikes shown in Fig. 1(c). To obtain this regime, we fix the parameters of the presynaptic neuron at $\sigma = -0.025$, $\alpha = 5.3$, and $\mu = 0.001$. Since for any random initial condition the maximum transient duration does not exceed 5000 iterations, we neglect the first 10 000 iterations in all simulations. Our results show that synchronization of the neurons with synaptic delay and memory is determined by both the relation between two delay times and the coupling strength η. When η is too weak, the neurons fire asynchronously. For a strong enough coupling, spikes of the postsynaptic neuron are entrained by the presynaptic neuron as a rotation number $\omega = p : q$, where p and q are the numbers of spikes of the postsynaptic and presynaptic neuron, respectively. Fig. 3 illustrates typical examples of the frequency entrained states with $\omega = 1:1$ [Fig. 3(a)], 2:1 [Fig. 3(b)], and 3:1 [Fig. 3(c)].

3.2. Phase locking

While the frequency is entrained, the phase of the postsynaptic neuron can drift over time, so that the difference $\Delta\varphi = \varphi_{post} - \varphi_{pre}$ between instantaneous phases of the postsynaptic and presynaptic neuron spikes (φ_{post} and φ_{pre}) fluctuates around its mean value equal to $\theta = m - s$. This is the case of imperfect phase locking. For certain η the phase locking becomes perfect. Fig. 4 shows the alternation between imperfect and perfect phase locking when η is changed.

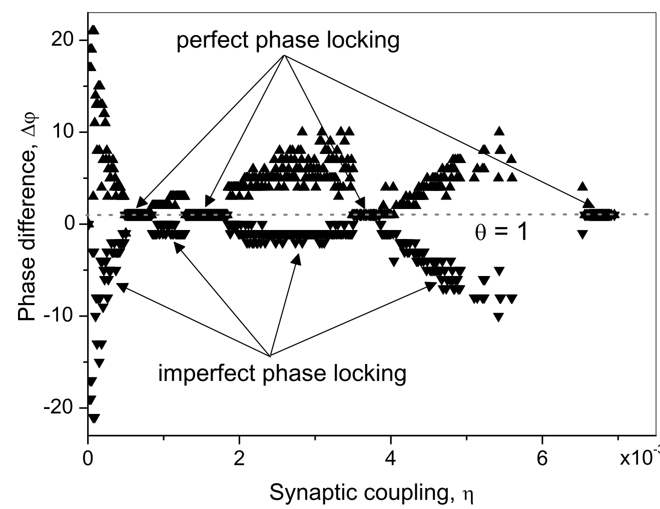

Fig. 4. Phase difference (in units of number of iterations) versus coupling strength for $\theta = 1$. While the postsynaptic neuron frequency is entrained to 1:1, the phase difference drifts in time (imperfect phase synchronization) around its mean value $\theta = 1$ shown by the dotted line. Only for certain η, the phase is locked with anticipation time θ.

We find that the relation between memory m and synaptic delay s is crucial for neuron synchronization. As seen from Fig. 5, the type of synchronization is determined by a sign of the difference $\theta = m - s$. When $\theta < 0$ the neurons synchronize with lag, otherwise they synchronize either with anticipation (for $\theta > 0$) or with zero lag (for $\theta = 0$). Fig. 5 shows the location of the 1:1 frequency entrained states in the (θ, η)-parameter space. One can see that inside of this triangle region, lag and anticipated synchronization occur, respectively, in neurons with short ($m < s$) and long memory ($m > s$).

3.3. Anticipated synchronization

Both lag and anticipated synchronization can be quantitatively characterized by similarity function $S^2(\phi)$ [43] that can be defined as the normalized averaged-in-time difference between the fast variables x_n and u_n of the presynaptic and postsynaptic neurons:

$$S^2(\phi) = \frac{\langle u_n - x_{n+\phi}\rangle^2}{[\langle x_n^2\rangle\langle u_n^2\rangle]^{1/2}}, \tag{7}$$

where ϕ is a delay time used to compare waveforms of coupled oscillators. If the variables x and u are uncorrelated, $S^2(\phi) \approx 1$ for all ϕ, otherwise $S^2(\phi)$ reaches its minimum at a certain value $\phi = \theta$ which indicates either lag (if $\phi < 0$) or anticipation (if $\phi > 0$) time. In particular, the similarity function in Fig. 6 reveals

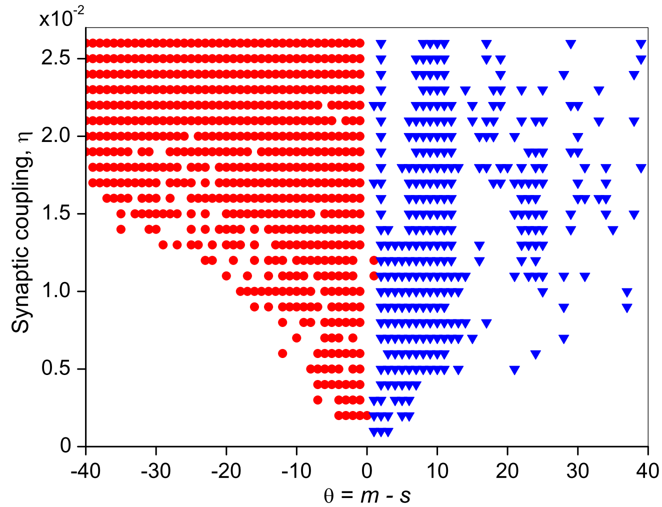

Fig. 5. Synchronization state diagram in 1:1 frequency entrained region in parameter space of difference θ between memory m and synaptic delay time s, and coupling strength η. The red dots and blue triangles indicate, respectively, lag and anticipated synchronization. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

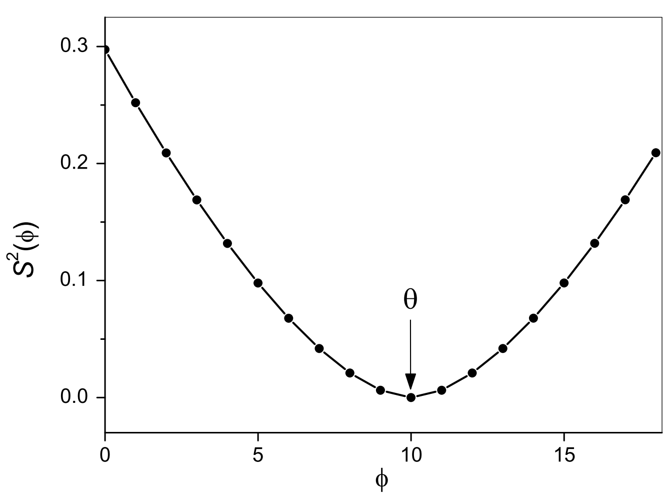

Fig. 6. Similarity function of two Rulkov neurons synchronized with anticipation time $\theta = 10$.

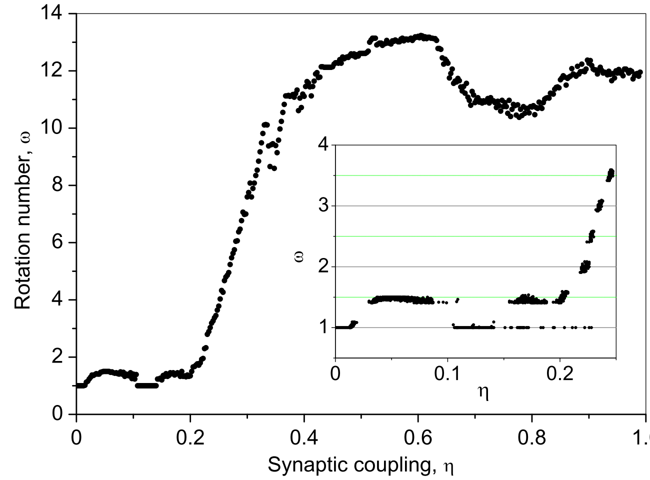

Fig. 7. Rotation number ω of frequency entrained states versus coupling strength η for $m = 3$ and $s = 2$. The inset is the enlarged part of the main graph.

synchronization with anticipation time $\theta = 10$, meaning that the postsynaptic neuron is activated before the postsynaptic neuron. Such a contradictory behavior of coupled oscillators discovered in 2000 [44] has attracted much attention because it gives possibility to predict the future of the system state. Later, this type of synchronization has been found in many systems [45–47], including neuron models based on difference [9,10,13–16] and differential equations [2,19,21].

The number of spikes in a burst train of the postsynaptic neuron depends on the coupling strength η. Fig. 7 shows that the rotation number ω of the frequency entrained states is a non-monotonous piece-wise function of η. The number of spikes in the train grows as η is increased and saturates to 13 at $\eta = 0.6$. When η is further increased, ω goes down and reaches its local minimum at $\eta \approx 0.8$. Such a nontrivial behavior is explained by the fact that at $\eta = 0.6$ the train duration becomes equal to the inter-train interval [Fig. 8(a)] and a further increase in η gives rise to an irregular spiking behavior shown in Fig. 8(b).

3.4. Intermittency and chaos

For intermediate values of the coupling, where the spike frequency is not entrained, intermittent switches between states with different ω occur. One such regime with intermittent switches between 1:1 and 2:1 entrained states is shown in Fig. 9.

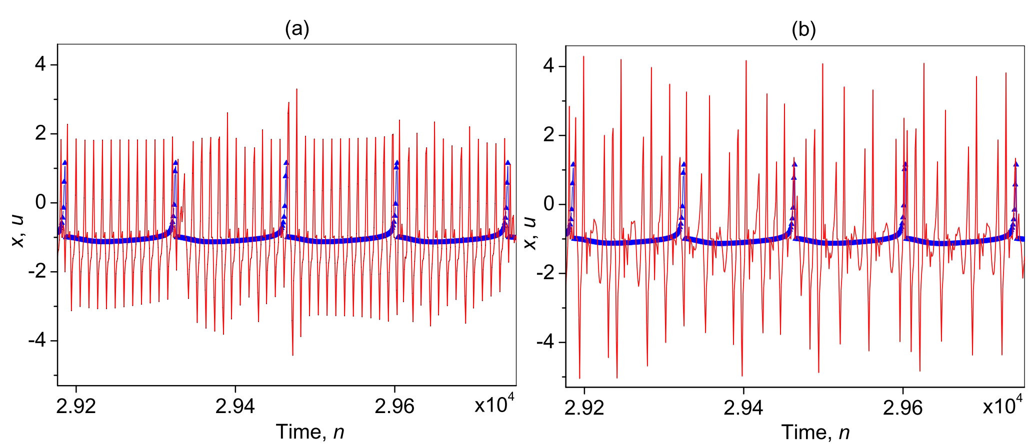

Fig. 8. Time series for strong coupling (a) $\eta = 0.6$ and (b) $\eta = 0.8$. The blue triangles and red traces show respectively the time series of the presynaptic and postsynaptic neurons. The large number of spikes in the burst train results in irregularity in the spiking sequence of the postsynaptic neuron. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

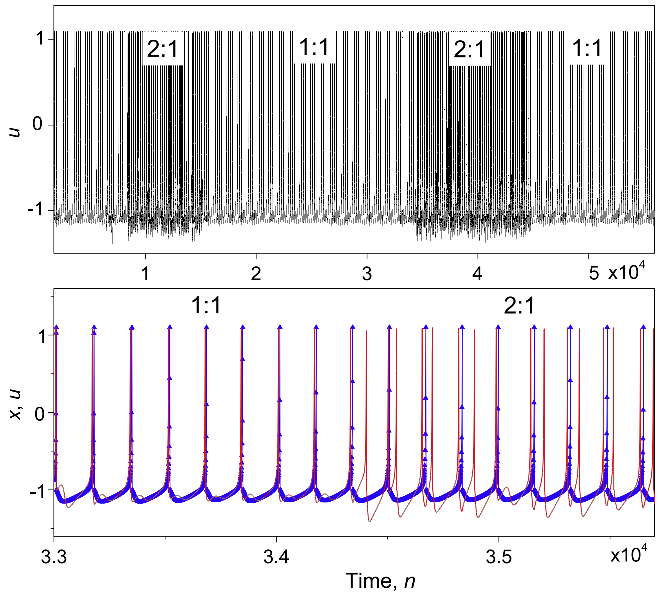

Fig. 9. Intermittent switches between 1:1 and 2:1 frequency entrained states for $\eta = 0.00401$, $m = 1$, and $s = 0$. The lower frame is the enlarged part of the upper frame.

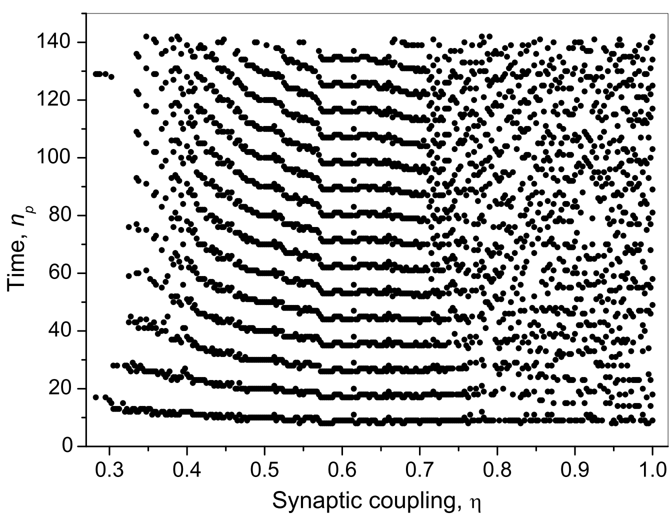

Fig. 10. Poincaré map at $u = 0$ of postsynaptic neuron versus coupling strength for $m = 3$ and $s = 2$.

As we already mentioned above, a very strong coupling leads to chaotic oscillations when the burst train of the postsynaptic neuron becomes longer than the distance between the trains. Such a transition from a regular motion to chaos is illustrated in Fig. 10, where we show the times of intersection of the postsynaptic neuron trajectory with the Poincaré section at $u = 0$. The periodic structure represents the frequency-locked spikes which are converted to chaotic unlocked spikes at $\eta = 0.71$.

4. Conclusions

The type of synchronization of synaptically coupled neurons is determined by the difference between synaptic delay time s and memory m. While for $s > m$ lag synchronization takes place, for $s < m$ anticipated synchronization occurs. Depending on the coupling strength, the neuron spikes of the postsynaptic neuron can either be locked to a rotation number from 1 to 13 or vary in time. Such a behavior has been demonstrated in coupled Rulkov maps whose dynamics represents main features inherent to a real neuron. Future research should be directed towards understanding the mechanisms behind learning and memory, that may lie in synchronization of neurons coupled in complex networks with memory loops.

Acknowledgement

The authors acknowledge support from CONACYT (Mexico), grant No. 203779.

References

[1] M.V. Good, J.G. Zalatan, W.A. Lim, Science 332 (2011) 680.
[2] R.C. Elson, A.I. Selverston, R. Huerta, N.F. Rulkov, M.I. Rabinovich, H.D.I. Abarbanel, Phys. Rev. Lett. 81 (1998) 5692.
[3] R.C. Elson, A.I. Selverston, H.D.I. Abarbanel, M.I. Rabinovich, J. Neurophysiol. 88 (2002) 1166.
[4] D.R. Chialvo, Chaos Solitons Fractals 5 (1995) 461.
[5] O. Kinouchi, M.H.R. Tragtenberg, Int. J. Bifurc. Chaos 6 (1996) 2343.
[6] S.M. Kuva, G.F. Lima, O. Kinouchi, M.H.R. Tragtenberg, A.C. Roque, Neurocomputing 38–40 (2001) 255.
[7] B. Cazelles, M. Courbage, M. Rabinovich, Europhys. Lett. 56 (2001) 504.
[8] C.R. Laing, A. Longtin, Bull. Math. Biol. 64 (2002) 829.
[9] N.F. Rulkov, Phys. Rev. E 65 (2002) 041922.
[10] A.L. Shilnikov, N.F. Rulkov, Int. J. Bifurc. Chaos 13 (2003) 3325.
[11] M. Copelli, M.H.R. Tragtenberg, O. Kinouchi, Physica A 342 (2004) 263.
[12] E.M. Izhikevich, F. Hoppensteadt, Int. J. Bifurc. Chaos Appl. Sci. Eng. 14 (2004) 3847.
[13] J.C.A. de Pontes, R.L. Viana, S.R. Lopes, C.A.S. Batista, A.M. Batista, Physica A 387 (2008) 4417.
[14] X. Sun, Q. Lu, J. Kurths, Q. Wang, Int. J. Bifurc. Chaos 19 (2009) 737.
[15] Y. Uwate, Y. Nishio, R. Stoop, in: Proceedings of RISP International Workshop on Nonlinear Circuits and Signal Processing, 2009, p. 197.
[16] H. Cao, M.A.F. Sanjuan, Cogn. Process. 10 (2009) S23.
[17] A. Hodgkin, A. Huxley, J. Physiol. 117 (1952) 500.
[18] J.L. Hindmarsh, R.M. Rose, Proc. R. Soc. Lond. B, Biol. Sci. 221 (1984) 87.
[19] M.V. Ivanchenko, T. Nowotny, A.I. Selverston, M.I. Rabinovich, J. Theor. Biol. 253 (2008) 452.
[20] M.I. Rabinovich, H.D.I. Abarbanel, Neuroscience 87 (1999) 5.
[21] X. Lang, Q. Lu, J. Kurths, Phys. Rev. E 82 (2010) 021909.
[22] F.S. Matias, P.V. Carelli, C.R. Mirasso, M. Copelli, Phys. Rev. E 84 (2011) 021922.
[23] F.S. Matias, L.L. Gollo, P.V. Carelli, M. Copelli, C.R. Mirasso, BMC Neurosci. 14 (Suppl. 1) (2013) P275.
[24] M.V. Ivanchenko, G.V. Osipov, V.D. Shalfeev, J. Kurths, Phys. Rev. Lett. 92 (2004) 134101.
[25] I. Franović, V. Miljković, Eur. Biophys. J. 76 (2010) 613.
[26] C. Mayol, C.R. Mirasso, R. Toral, Phys. Rev. E 85 (2012) 056216.
[27] E.R. Kandel, J.H. Schwartz, T.M. Jessell, Principles of Neural Science, Appleton & Lange, Prentice Hall, 2000.
[28] B. Katz, R. Miledi, Proc. R. Soc. Lond. B 161 (1965) 483.
[29] J. Maa, J. Wu, Math. Model. Nat. Phenom. 5 (2010) 67.
[30] D.J. Amit, Behav. Brain Sci. 18 (1995) 617.
[31] H. Ebbinghaus, Memory: A Contribution to Experimental Psychology, Teachers College, Columbia Univ., 1913.
[32] G. de Vries, Phys. Rev. E 64 (2001) 051914.
[33] N.F. Rulkov, Phys. Rev. Lett. 86 (2001) 183.
[34] N.F. Rulkov, I. Timofeev, M. Bazhenov, J. Comput. Neurosci. 17 (2004) 203.
[35] A. Shilnikov, N.F. Rulkov, Phys. Lett. A 328 (2004) 177.
[36] A. Shilnikov, R. Gordon, I. Belykh, Chaos 18 (2008) 037120.
[37] I. Belykh, E. de Lange, M. Hasler, Phys. Rev. Lett. 94 (2005) 188101.
[38] H. Wang, Q. Lu, Q. Wang, Commun. Nonlinear Sci. Numer. Simul. 13 (2008) 1668.
[39] A.L. Hodgkin, A.F. Huxley, J. Physiol. 117 (1952) 500.
[40] J.L. Hindmarsh, R.M. Rose, Proc. R. Soc. Lond. B 221 (1984) 87.
[41] M.V. Bennett, Brains Res. Rev. 32 (2000) 16.
[42] P. Greengard, Science 294 (2001) 1024.
[43] M.G. Rosenblum, A.S. Pikovsky, J. Kurths, Phys. Rev. Lett. 78 (1997) 4193.
[44] H.U. Voss, Phys. Rev. E 61 (2000) 5115.
[45] A.N. Pisarchik, R. Jaimes-Reátegui, J.R. Villalobos-Salazar, J.H. García-López, S. Boccaletti, Phys. Rev. Lett. 96 (2006) 244102.
[46] M. Ciszak, R. Toral, C. Mirasso, Mod. Phys. Lett. B 18 (2004) 1135.
[47] T. Pyragienė, K. Pyragas, Nonlinear Dyn. 47 (2013) 297.