

BISTABILITY IN HINDMARSH-ROSE NEURAL OSCILLATORS INDUCED BY ASYMMETRIC ELECTRICAL COUPLING

Rider Jaimes-Reátegui

Centro Universitario de los Lagos
Universidad de Guadalajara
Mexico
rjaimes@culagos.udg.mx

Mariano Alberto García-Vellisca

Center for Biomedical Technology
Technical University of Madrid
Spain
alberto.garcia@ctb.upm.es

Alexander N. Pisarchik

Center for Biomedical Technology
Technical University of Madrid
Spain
Yuri Gagarin Saratov State University
Russia
alexander.pisarchik@ctb.upm.es

Francisco del Pozo-Guerrero

Center for Biomedical Technology
Technical University of Madrid
Spain
CIBER-BBN
Centro de Investigación Biomédica en Red
Bioingeniería, Biomateriales y Nanomedicina
francisco.delpozo@ctb.upm.es

Abstract

In this paper, we are interested in the question of how bistability can appear in coupled neurons. Concrete motivation for such a general problem is the search for a way to destroy an organism having a stable dynamics by destabilizing its metabolism. To address this issue, we consider the model of a pair of neuron cells coupled via an electrical synapse. We focus on the Hindmarsh-Rose model which provides a simple description of the patterned activity observed in molluscan neurons. The results of numerical simulations show that asymmetric electrical coupling between periodically spiking neural oscillators results in bistability in this system. One of the coexisting attractors is a limit cycle similar to the attractor of the uncoupled neuron, while the other one can be either a chaotic or a periodic orbit depending on the coupling strengths. Bistability is only observed for relatively small couplings. When the coupling is sufficiently strong, the neurons are in a monostable periodic regime, similar to the spiking regime observed in the uncoupled neurons.

Key words

Neuron, nonlinear dynamics, bistability, chaos.

1 Introduction

Synchronous firing of cortical inhibitory neurons plays an important role in encoding of information in the cortex [Singer and Gra, 1995; Ritz and Sejnowski, 1997]. Certain neurons in the mammalian brain have long been known to be joined by electrical synapses implicated in several physiological aspects of brain function and in anomalous population activity characteristics of epilepsy. An electrical synapse is a conductive link between two neurons in the form of a narrow gap known as a gap junction which allows various molecules and ions to pass directly from one cell to another. Gap junction intercellular channels are the structural basis of synapses that provide a high electric conductance for direct exchange of ions, metabolites, secondary messengers, and small molecules between neighboring cells [Laird, Castillo, and Kasprzak, 1995; Evans and Martin, 2002]. Depending on the gap junction type, the molecules can pass either in both directions or asymmetrically, thus providing electrical communication between the neighboring cells. Gap junctions can synchronize electrical activity and may subserve metabolic coupling and chemical communication as well [Bennett and Zukin, 2004]. Since a gap junction creates a bridge connecting two cells, when current flows from a more positive cell to depolarize a more negative cell, the transjunction current makes the first cell less depolarized; the coupling excites one cell

while inhibiting the other. Therefore, one can characterize these synapses as synchronizing rather than excitatory or inhibitory. Although most of electrical synapses are bidirectional, some gap junctions restrict communication to only one direction or represent an asymmetry in coupling [Bukauskas et al., 2002], i.e. the efficacy of transmission in one direction is greater than in the other, as is the case at the giant motor synapse of the crayfish [Furshpan and Potter, 1959].

In spite of a great interest in understanding cooperative behavior of coupled neurons, where identification and characterization of neuron dynamics are essential, and a large number of papers on synchronization of coupled neural oscillators [Belykh, de Lange, and Hasler, 2005; Abarbanel, Huerta, and Rabinovich, 1996; Bazhenov et al., 1998; Baptista, Moukam Kakmeni, and Grebogi, 2010; Liang et al., 2009], the problem of how interaction among the cells promotes synchrony is not yet well understood [Whittington, Traub, and Jefferys, 1995; Bennett and Zuckin, 2004]. In this work, we are interested in the question of how bistability can appear in a given neuron model. Concrete motivation of such a general problem is the search for a way to destroy an organism having a stable dynamics by destabilizing its metabolism. To address this issue theoretically, we study the model of a pair of neuron cells coupled via an electrical synapse. Here, we focus on the Hindmarsh-Rose (HR) model [Hindmarsh and Rose, 1984] which provides a simple description of the patterned activity observed in molluscan neurons. Even though this model is not wholly based on physiology as the accurate Hodgkin-Huxley model [Hodgkin and Huxley, 1952], it allows basic phenomenological description of neuron dynamics, such as quiescence, spiking, irregular spiking and chaotic bursting [Rabinovich and Abarbanel, 1998; Belykh, de Lange, and Hasler, 2005], and reveals nonlinear dynamical mechanisms underlying many biological processes.

Choosing the parameters so that the uncoupled neurons are in a periodic spiking regime, we study how the system dynamics depends on the coupling strengths by analyzing bifurcation diagrams of interspike intervals (ISI) and Lyapunov exponents.

2 Model

The system of two coupled HR neurons can be described by the following nonlinear differential equations:

$$\begin{aligned} \dot{x}_1 &= y_1 - ax_1^3 + bx_1^2 - z_1 + I_{\text{ext}1} + \sigma_1(x_2 - x_1), \\ \dot{y}_1 &= c - dx_1^2 - y_1, \\ \dot{z}_1 &= r[s(x_1 - x_0) - z_1], \\ \dot{x}_2 &= y_2 - ax_2^3 + bx_2^2 - z_2 + I_{\text{ext}2} + \sigma_2(x_1 - x_2), \\ \dot{y}_2 &= c - dx_2^2 - y_2, \\ \dot{z}_2 &= r[s(x_2 - x_0) - z_2], \end{aligned} \quad (1)$$

where $x_{1,2}$ are membrane potentials, $y_{1,2}$ are recovery variables associated with a fast current of Na^+ or K^+ ions, $z_{1,2}$ are adaptation currents associated with a slow current of Ca_2^+ ions, $I_{\text{ext}1,2}$ are external input currents, $x_0 = -1.6$ is the x -component of the stable equilibrium point without input ($I_{\text{ext}} = 0$), and $0 < \sigma_{1,2} < 1$ are electrical coupling strengths used as control parameters. The following parameters are used in numerical simulations: $a = 1$, $b = 3$, $c = 1$, $d = 5$, $s = 4$, and $r = 0.006$.

The dynamics of the membrane potential x is determined by the value of I_{ext} . For small and large I_{ext} , the solitary neuron generates tonic spikes, whereas for intermediate current its dynamics is chaotic. In this work we fix the currents for both neurons at $I_{\text{ext}1} = I_{\text{ext}2} = I_{\text{ext}} = 1.4$, where the uncoupled neurons oscillate in a periodic spiking regime.

2.1 Bistability

In the majority of papers a bidirectional coupling is considered as symmetrical. It is well known that two identical neurons with symmetric coupling represent similar dynamics, as shown in Fig. 1. No bistability was found in symmetrically coupled neurons.

Significantly less attention was paid to a study of asymmetrically coupled neurons [Kim and Jones, 2011]. Here, we will show that the asymmetry leads to bistability in the neural system, that was never observed in symmetrically coupled neurons.

The time series of two coexisting regimes, periodic and chaotic, in asymmetrically coupled neurons are shown, respectively, in Figs. 2 and 3. These states were found by taking different initial conditions, when all system parameters were fixed.

One can see that one of the coexisting states is periodic (Fig. 2), whereas the other one is chaotic (Fig. 3).

Bistability can also be revealed with bifurcation diagrams calculated by taking random initial conditions. Such bifurcation diagrams of inter-spike intervals (ISI) for two coupled neurons are shown in Fig. 4.

One can see that the bifurcation diagram contains several branches. One of them (P1) corresponds to tonic spikes, similar to the uncoupled neuron, and the other ones to the coexisting chaotic (for $0.11 < \sigma_2 < 0.16$ and $0.20 < \sigma_2 < 0.24$) or periodic attractor.

2.2 Lyapunov Exponent

Another measure which can reveal the coexistence of attractors is the Lyapunov exponent. In Fig. 5 we plot the largest Lyapunov exponent for the coupled neural system in the space of two coupling strengths, σ_1 and σ_2 , calculated for fixed initial conditions. One can see that the exponent values are asymmetric with respect to the diagonal, although the system Eq. (1) is symmetric. This means that the system is bistable for certain values of the coupling parameters.

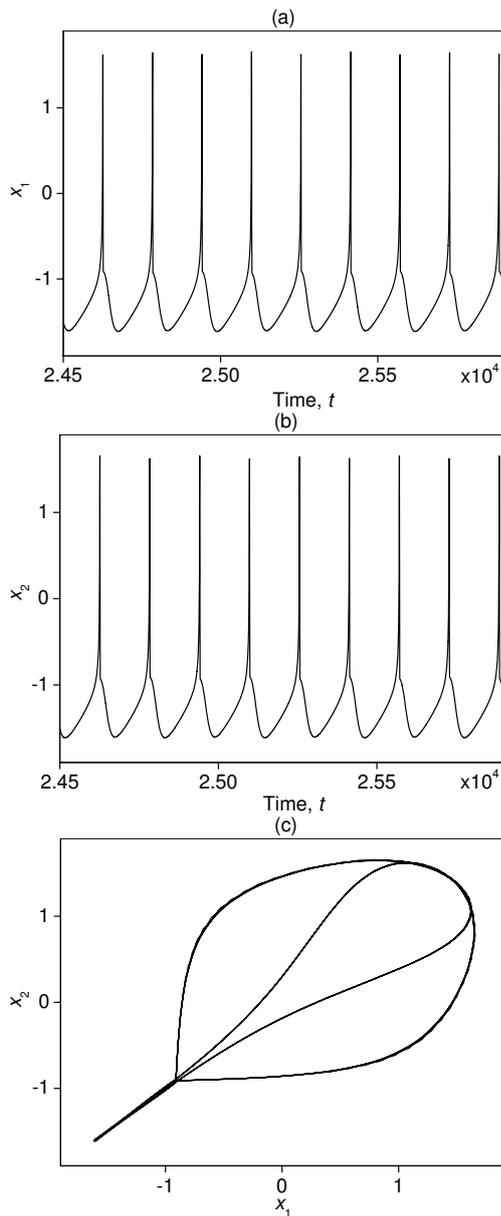


Figure 1. (a,b) Time series of neural membrane potentials and (c) synchronization phase portrait for symmetrically coupled neurons with $\sigma_1 = \sigma_2 = 0.051$. Starting from different initial conditions, the neurons generate asynchronous tonic spikes.

3 Conclusion

We have shown that bistability can appear in asymmetrically coupled spiking neurons for certain values of the coupling strengths. While one of the coexisting attractors is periodic, the other one can be either periodic or chaotic. This effect has been demonstrated in the Hindmarsh-Rose neural model with time series, phase-space portraits, bifurcation diagrams, and Lyapunov exponents.

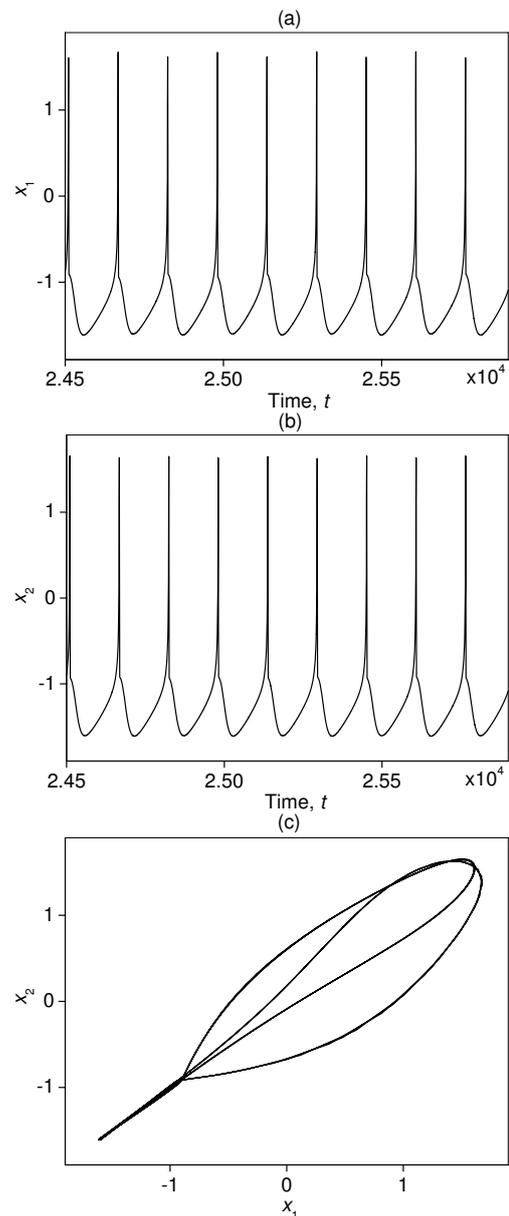


Figure 2. Coexisting periodic regime in asymmetrically coupled neurons. (a,b) Tonic spikes of (a) neuron 1 and (b) neuron 2, and (c) synchronization phase portrait for $\sigma_1 = 0.05$ and $\sigma_2 = 0.2$.

Acknowledgements

The authors acknowledge support from the Ministry of Economy and Competitiveness (Spain) (project SAF2016-80240) for the work on numerical simulations and analysis. The model development has been supported by the Russian Science Foundation (grant 17-72-30003).

References

- Singer, W and Gra, C. M. (1995). Visual feature integration and the temporal correlation hypothesis. *Ann. Rev. Neurosci.*, **18**, pp. 555–586.
- Ritz, R. and Sejnowski, T. J. (1997). Synchronous os-

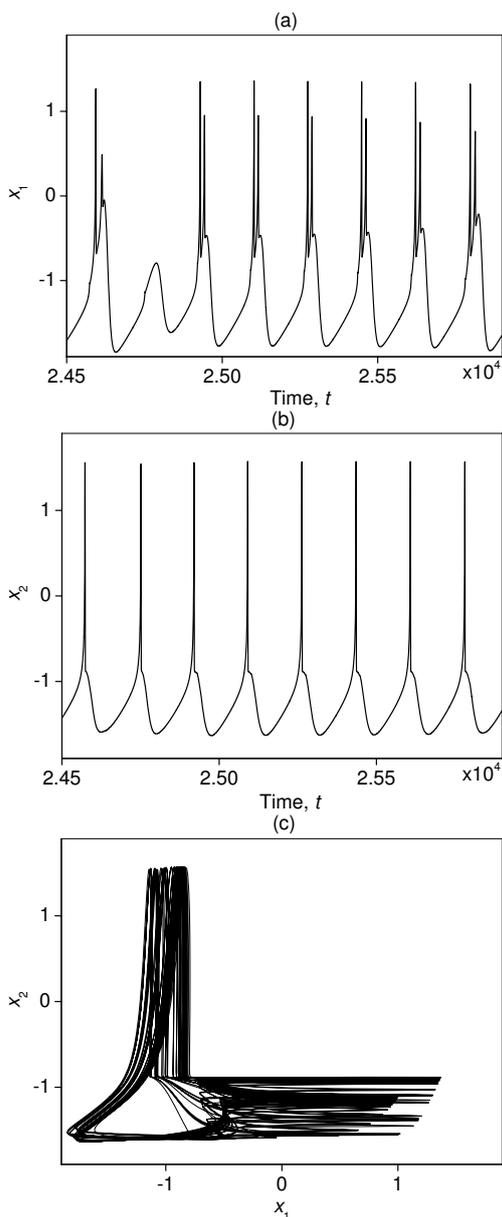


Figure 3. Coexisting chaotic regime in asymmetrically coupled neurons. (a) Chaotic and (b) periodic spiking regimes of neuron 1 and neuron 2, respectively, and (c) synchronization phase portrait for $\sigma_1 = 0.05$ and $\sigma_2 = 0.2$.

cillatory activity in sensory systems: new vistas on mechanisms. *Curr. Opin. Neurobiol.*, **7**, pp. 536–546.

Laird, D. W., Castillo, M., and Kasprzak, L. (1995). Gap junction turnover, intracellular trafficking, and phosphorylation of connexin43 in brefeldin A-treated rat mammary tumor cells. *J. Cell Biol.*, **131**, pp. 1193–1203.

Evans, W. H. and Martin, P. E. (2002). Gap junctions: structure and function (Review). *Mol. Membr. Biol.*, **19**, pp. 121–136.

Bennett, M. V. L. and Zukin, R. S. (2004). Electrical coupling and review neuronal synchronization in the mammalian brain. *Neuron*, **41**, pp. 495–511.

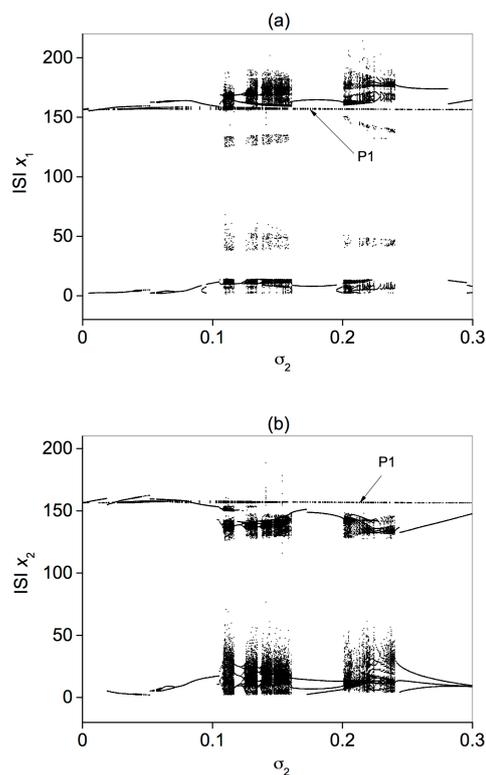


Figure 4. Bifurcation diagrams of ISI of (a) neuron 1 and (b) neuron 2 as a function of σ_2 for $\sigma_1 = 0.051$. The arrows show the branch of the period-1 attractor (P1).

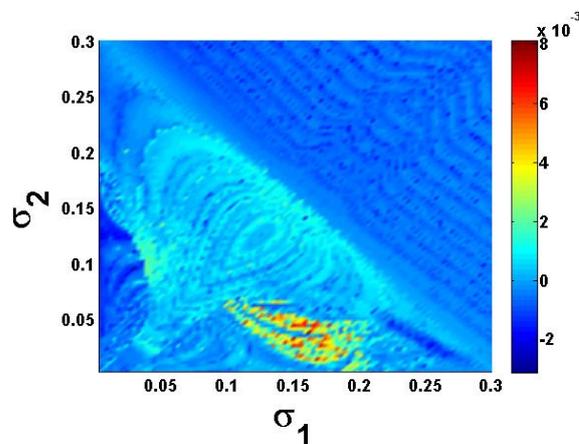


Figure 5. Largest Lyapunov exponents as a function of coupling strengths σ_1 and σ_2 . The exponent is calculated by taking fixed initial conditions.

Bukauskas, F. F., Angele, A. B., Verselis, V. K., and Bennett, M. V. L. (2002). Coupling asymmetry of heterotypic connexin 45/connexin 43-EGFP gap junctions: Properties of fast and slow gating mechanisms. *Proc. Natl. Acad. Sci USA*, **99**, pp. 7113–7118.

Furshpan, E. J. and Potter, D. D. (1959). Transmission at the giant motor synapses of the crayfish. *J. Phys-*

- iol.*, **145**, pp. 289–325.
- Whittington, M. A., Traub, R. D., and Jefferys, J. G. R. (1995). Synchronized oscillations in interneuron networks driven by metabotropic glutamate receptor activation. *Nature*, **373** 612–615.
- Hindmarsh, J. L. and Rose, R. M. (1984). A model of neuronal bursting using three coupled first order differential equations. *Proc. Roy. Soc. Lond. B*, **221** 87–102.
- Hodgkin, L. A. and Huxley, A. F. (1952). A quantitative description of membrane current and its application to conduction and excitation in nerve. *J. Physiol.*, **117**, pp. 500–544.
- Rabinovich, M. I. and Abarbanel, H. D. I. (1998). The role of chaos in neural systems. *Neuroscience*, **87**, pp. 5–14.
- Belykh, I., de Lange, E., and Hasler, M. (2005). Synchronization of bursting neurons: What matters in the network topology. *Phys. Rev. Lett.*, **94**, 188101.
- Abarbanel, H. D. I., Huerta, R., and Rabinovich, M. I. (1996). Synchronized action of synaptically coupled chaotic model neurons. *Neural Comput.*, **8**, pp. 1567–1602.
- Bazhenov, M., Huerta, R., Rabinovich, M. I., and T. Sejnowski, T. (1998). Cooperative behavior of a chain of synaptically coupled chaotic neurons. *Physica D*, **116**, pp. 392–400.
- Baptista, M. S., Moukam Kakmeni, F. M., and Grebogi, C. (2010). Combined effect of chemical and electrical synapses in Hindmarsh-Rose neural networks on synchronization and the rate of information. *Phys. Rev. E*, **82**, 036203.
- Liang, X., Tang, M., Dhamala, M., and Liu, Z. (2009). Phase synchronization of inhibitory bursting neurons induced by distributed time delays in chemical coupling. *Phys. Rev. E*, **80**, 066202.
- Kim, H. and Jones, K. E. (2011). Asymmetric electrotonic coupling between the soma and dendrites alters the bistable firing behaviour of reduced models. *J. Comput. Neurosci.*, **30**, pp. 659–674.