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Phase synchronization of coupled bursting neurons and the generalized Kuramoto model

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ABSTRACT

Bursting neurons fire rapid sequences of action potential spikes followed by a quiescent period. The basic dynamical mechanism of bursting is the slow currents that modulate a fast spiking activity caused by rapid ionic currents. Minimal models of bursting neurons must include both effects. We considered one of these models and its relation with a generalized Kuramoto model, thanks to the definition of a geometrical phase for bursting and a corresponding frequency. We considered neuronal networks with different connection topologies and investigated the transition from a non-synchronized to a partially phase-synchronized state as the coupling strength is varied. The numerically determined critical coupling strength value for this transition to occur is compared with theoretical results valid for the generalized Kuramoto model.

1. Introduction

Neurons are known to exhibit a plethora of dynamical behaviors, represented by the generation of action potential patterns. One of such patterns is bursting, defined by the repeated firing of action potentials followed by quiescent periods. Hence the dynamics of bursting neurons has two timescales: a fast scale related to spiking and a slow scale of bursting itself. These timescales are related to different biophysical mechanisms occurring at the level of neuron membrane: there are fast ionic currents (chiefly Na^+ and K^+) responsible for spiking activity and slower Ca^{++} currents that modulate this activity.

Most neurons exhibit bursting behavior if conveniently stimulated. For example, in the neocortical layer 5 pyramidal neurons, when stimulated with DC current pulses, fire an initial burst of spikes followed by shorter bursts (Blank & Stoop, 1999; Connors & Gutnick, 1990). In layers 2, 3, and 4 chattering neurons fire high-frequency bursts of 3–5 spikes with a short interburst period (Gray & McCormick, 1996; Stoop et al., 2002). Cortical interneurons have been found to exhibit bursting as a response to DC pulses (Markram et al., 2004). Pyramidal neurons in the *CA*1 region of hippocampus produce high-frequency bursts after current injection (Su, Alroy, Kirson, & Yaari, 2001). Thalamocortical neurons and reticular thalamic nucleus inhibitory neurons exhibit bursting as well (Ramcharan, Gnadt, & Murray Sherman, 2000). Purkinje cells in cerebellum can burst when their synaptic input is blocked (Womack & Khodakhah, 2002). Bursting is also an important feature of sensory systems, because bursts can increase the reliability of synaptic transmission (Krahe & Gabbiani, 2004). In some systems, bursts improve the signal-to-noise ratio of sensory responses and might be involved in the detection of specific stimulus features (Metzner, Koch, Wessel, & Gabbiani, 1998).

Due to both synaptic coupling and common inputs among neurons there are many types of synchronization, which can be generally regarded as the presence of a consistent temporal relationship between their activity patterns (Elson et al., 1998; Makarenko & Llinás, 1998; Varona, Torres, Abarbanel, Rabinovich, & Elson, 2001). A strong form of the latter relationship is complete synchronization, where neurons spike at the same time, i.e. a precise temporal coincidence of events. A weaker relationship is bursting synchronization, in which only the beginning of bursting is required to occur at the same time, even though the repeated spiking may not occur synchronously.

There has been observed bursting synchronization in cell cultures of cortical neurons, where uncorrelated firing appeared within the first three days and transformed progressively into synchronized bursting within a week (Kamioka, Maeda, Jimbo, Robinson, & Kawana, 1996). Large-scale bursting synchronization in the 7–14 Hz range has been found in the thalamus during slow-wave

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sleep, partially originated in the thalamus and gated by modulatory input from the brainstem (Steriade, McCormick, & Sejnowski, 1993). Various areas of the basal ganglia have been found to exhibit bursting synchronization related to Parkinson's disease and resting tremor (Bevan, Magill, Terman, Bolam, & Wilson, 2002).

There exists sound neurophysiological evidence that hypokinetic motor symptoms of Parkinson's disease such as slowness and rigidity of voluntary movements are closely related to synchronized bursting in the 10–30 Hz range (Brown, 2007; Hutchison et al., 2004; Park, Worth, & Rubchinsky, 2010; Uhlhaas & Singer, 2006). The connection between bursting synchronization and pathological conditions like Parkinson's disease, essential tremor and epilepsy has led to the proposal of many control strategies aiming to suppress or mitigate bursting synchronization (Hammond, Bergman, & Brown, 2007).

One of such strategies is deep-brain stimulation (DBS), which consists of the application of an external high-frequency (>100 Hz) electrical signal by depth electrodes implanted in target areas of the brain like the thalamic ventralis intermedius nucleus or the subthalamic nucleus (Benabid et al., 1991). The effect of DBS would be similar to that produced by tissue lesioning and has proved to be effective in suppression of the activity of the pacemaker-like cluster of synchronously firing neurons, and achieving a suppression of the peripheral tremor (Blond et al., 1992). There is strong clinical evidence that DBS is a highly effective technique for treatment of patients with Parkinson's disease (Albanese & Romito, 2011; Rodriguez-Oroz et al., 2005).

In spite of these results, DBS is yet far from being completely understood. Many results in this field have been obtained from empirical observations made during stereotaxic neurosurgery, but further progress can be obtained with proper mathematical modeling of DBS (Hauptmann, Popovych, & Tass, 2005; Pfister & Tass, 2010; Tass, 2003). The effects of DBS in networks of bursting neurons have been investigated when DBS is implemented through an harmonic external current (Batista, Batista, de Pontes, Viana, & Lopes, 2007) and a delayed feedback signal (Batista, Lopes, Viana, & Batista, 2010).

On modeling the response of a neuronal network to an external perturbation like DBS it is of paramount importance to keep the model simple enough such that large-scale simulations (using a large number of neurons) can be performed in a reasonable computer time. In such reductionist point of view a minimal model could be one in which we can assign a geometrical phase to the bursting activity. The bursting neuron is thus regarded as a phase oscillator undergoing spontaneous oscillations with a given frequency (Rabinovich, Varona, Torres, Huerta, & Abarbanel, 1999). Thus bursting synchronization becomes a special case of phase synchronization, a phenomenon well understood for coupled oscillators with and without external excitation (Pikovsky, Rosenblum, Osipov, & Kurths, 1997).

A simple model for the dynamics of nonlinearly coupled phase oscillators is the Kuramoto model, which in its original version considers a global (all-to-all) coupling (Kuramoto, 1984). It can be generalized by considering an arbitrary coupling architecture (generalized Kuramoto model) (Acebrón, Bonilla, Vicente, Ritort, & Spigler, 2005). The particular interest in such models is that many analytical and numerical results are known for them, specially the global case for which a mean-field theory exists for the transition between a non-synchronized to a (phase-)synchronized behavior (Strogatz, 2000). For generalized Kuramoto models it is possible to derive analytical expressions for the critical value of the coupling strength for which the above mentioned transition occurs (Restrepo, Ott, & Hunt, 2005). Hence such a body of knowledge can be applied to networks of bursting neurons, helping to design strategies of synchronization control and/or suppression like DBS.

The main goal of this paper is to show, using analytical and numerical arguments, that a system of coupled bursting neurons described by Rulkov's model can be reduced to a generalized Kuramoto model. This reduction is valid as long as phase synchronization is concerned, since for frequency synchronization the behaviors can be quite different. We consider, in particular, some widely used connection topologies, like random (Erdös–Rényi), small-world, and scale-free networks. We show that the analytical results for the critical coupling strength to synchronized behavior, originally derived for the generalized Kuramoto model, can be used to describe the synchronization transition also for networks of bursting neurons.

As a matter of fact, since bursting activity presents two timescales it can be also approached from the point of view of a relaxation oscillator (Wang, 2005). In our work, however, we describe bursting using a single phase. This simplification is justified since phase synchronization of bursting is chiefly related to the slow timescale. In other words, the fast spikes can be nonsynchronized even though the slow dynamics is synchronized.

This paper is organized as follows: in Section 2 we describe the model we used to describe bursting neurons. Section 3 considers networks of coupled bursting neurons and introduces quantifiers to characterize phase synchronization. Section 4 discusses different connection architectures like global, random, small-world, and scale-free networks. In Section 5 we review some results on the generalized Kuramoto model, and Section 6 contains a mathematical discussion on the phase reduction near global phase synchronization of a network of coupled neurons. Section 7 includes the comparisons we made between Kuramoto model and the network of bursting neurons. Our Conclusions are left to the final Section.

2. Models of bursting neurons

The choice of a suitable model describing the dynamics of biological neurons is dictated by some requirements. First the model must take into account the kind of dynamics one wishes to describe (Ibarz, Casado, & Sanjuán, 2011). For example, if all one needs is to describe a spiking neuron, for the sake of neural coding simulations for example, a simple leaky integrate-and-fire (LIF) model would be enough (Koch & Segev, 1999). However if one needs to describe the interplay between different ionic currents flowing through the neuron membrane, the Hodgkin–Huxley (HH) model would be a natural choice (Hodgkin & Huxley, 1952). On the other hand, the HH model would require far more computational power than the LIF since the former involves four complicated first-order differential equations whereas the latter just one simple equation.

With bursting neurons this criterion also holds. Given that bursting results from the interplay between fast and slow ionic currents, Hodgkin–Huxley-type models would need at least one more equation to describe slow Ca modulation (Plant & Kim, 1976; Shorten & Wall, 2000). A model of thermally sensitive neurons exhibiting bursting has been proposed by Huber and Braun (Braun, Eckhardt, Braun, & Huber, 2000; Braun et al., 2001; Braun, Huber, Dewald, Schäfer, & Voigt, 1998), which describes spike train patterns experimentally observed in facial cold receptors and hypothalamic neurons of the rat (Braun et al., 1999), electro-receptor organs of freshwater catfish (Schäfer, Braun, Peters, & Bretschneider, 1995), and caudal photo-receptor of the crayfish (Feudel et al., 2000). However, the Huber–Braun model has 5 differential equations for each neuron, and computational limitations impose restrictions to its use for large networks (Prado, Lopes, Batista, Kurths, & Viana, 2014).

If numerical simulations do not need to take into account the effect of system parameters and only the phenomenological aspects of bursting are relevant, then a good choice is the two-dimensional mapping equations proposed by (Rulkov, 2001)

$$x(n+1) = \frac{\alpha}{1+[x(n)]^2} + y(n), \tag{1}$$

$$y(n+1) = y(n) - \sigma x(n) - \beta, \tag{2}$$