Bursting oscillations from a homoclinic tangency in a time delay system

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We describe a type of bursting oscillations arising in a model of reciprocally connected neurons, where a time delay has been introduced to account for synaptic and propagation delays. We show that in this system bursting oscillations appear at an infinite period bifurcation characterized by a homoclinic tangency to a limit cycle. Such homoclinic bursting phenomena are characterized by a logarithmic lengthening of the period, which could be measured from experimental time series.

The bursting oscillations are among the different types of oscillating behaviors observed in biological systems, such as in the R15 neuron of the Aplysia [1], or in insulin secreting cells [2]. In chemical systems, such as the Belousov–Zhabotinskii reaction [3], this type of oscillations have also been observed. A wide variety of nonlinear systems of first order differential equations also show similar types of oscillations [4–6].

It allows the system to alternate between active and silent phases.

In many cases of bursting, the treatment of the slow variables as parameters allows one to understand qualitatively the dynamical mechanism underlying the bursting oscillations [6]. According to this singular perturbation approach, the bifurcation diagram of the fast subsystem is studied as a function of the parameters corresponding to the subsystem. In this regime, a period of the bursting oscillations cor
\[
C_m \frac{dX}{dt} = -g_L (X-V_L) - g_{\text{EE}} (X-V_E) F(X(t-\tau)) \\
- g_{\text{IE}} (X-V_I) F(Y(t-\tau)) ,
\]
\[
C_m \frac{dY}{dt} = -g_L (Y-V_L) - g_{\text{EI}} (Y-V_E) F(X(t-\tau)) \\
- g_{\text{II}} (Y-V_I) F(Y(t-\tau)) ,
\]
where \(X\) and \(Y\) represent the membrane potential of an excitatory and of an inhibitory neuron respectively, \(C_m = 1 \mu F/cm^2\) is the specific membrane capacitance, \(g_L = 0.25 \text{ mS/cm}^2\) is the leakage conductance and \(V_L = -60 \text{ mV}\) is the leakage potential. The values chosen for this model are in the range of values measured experimentally in a neuronal membrane (cf., for example, ref. [7]). \(g_{\text{EE}}, g_{\text{IE}}, g_{\text{EI}}\) and \(g_{\text{II}}\) are respectively the synaptic conductances for excitatory-to-excitatory (EE), inhibitory-to-excitatory (IE), excitatory-to-inhibitory (EI) and inhibitory-to-inhibitory (II) interactions. \(V_E = 50 \text{ mV}\) and \(V_I = -80 \text{ mV}\) are the equilibrium potentials for synaptic excitation and inhibition. Synaptic interaction is taken into account by the use of the transfer function

\[F(V) = \frac{1}{1 + \exp\left[-\frac{1}{5} (V+25)\right]}.\]

fraction of excitatory or inhibitory cells active per unit of time.

Renormalizing the conductances by \(C_m\) leads to the following set of parameters (in ms\(^{-1}\)):
\(\gamma = g_L / C_m\), 
\(\Omega_1 = g_{\text{EE}} / C_m\), 
\(\Omega_2 = g_{\text{IE}} / C_m\), 
\(\Omega_3 = g_{\text{EI}} / C_m\) and 
\(\Omega_4 = g_{\text{II}} / C_m\). The values used in this paper are \(\Omega_2 = \Omega_3 = 5 \text{ ms}^{-1}\) and \(\Omega_4 = 0\). \(\Omega_1\) is the main parameter of the model. It is important to note that similar behavior is observed for a very wide range of these parameters and the values given here are therefore representative of the system.

As indicated by fig. 1, this model exhibits multiple steady states. The lower steady branch corresponds to the resting membrane potential and is around the value of \(V_L\). For higher values of \(\Omega_1\), other fixed points appear. In this case the upper steady branch is stable whereas the intermediate branches are unstable (cf. fig. 1). It is to be noticed that the upper branch corresponds to a stable state where both neurons stay permanently above the threshold.

For the values of \([\Omega_2, ..., \Omega_4]\) considered, the lower steady branch gives rise to limit cycle oscillations via a supercritical Hopf bifurcation (\(\Omega_1^H\) in fig. 1). We focus here on the complex oscillatory phenomena.
which appear from an infinite period bifurcation ($\Omega^p_2$ in fig. 1). As shown in fig. 1, a very similar diagram is seen for different values of the time delay.

Figure 2 shows the oscillating patterns seen at the approach of the point $\Omega^p_2$. For increasing values of $\Omega_1$, the limit cycle oscillations turn into bursting behavior.

We emphasize that the presence of the delay is necessary to observe this type of oscillations. Very similar behavior is observed for a very wide range of the parameters provided the delay is not too small, and the synaptic coefficients $\Omega_2$ and $\Omega_3$ are large enough. However, bursting oscillations disappear for time delays close to zero.

Figure 2 also shows that a bursting oscillation period is composed of an intermediate segment (the silent phase) and a burst of faster oscillatory behavior (the active phase). Very similar bursting oscillations have been observed in chemical systems [13].

A closer scrutiny of fig. 2 reveals that, during the approach to the critical point $\Omega^p_1$, the period continuously increases together with the number of pseudo-cycles of the fast oscillation, while the intermediate segment remains essentially unchanged. At the critical point $\Omega^p_1$, the oscillatory active phase lasts an infinite time, which leads us to assume the existence of a second limit cycle LC2 corresponding to the active phase fast oscillations. Since this limit cycle LC2 does not exist alone without the intermediate segment of the silent phase, we conclude that LC2 must be an unstable limit cycle of saddle type because trajectories like LC1 can enter the vicinity of LC2 before escaping from it. We now show that this behavior can be accounted for by the presence of a homoclinic tangency to the limit cycle LC2 as schematically illustrated in fig. 3.

As $\Omega_1$ increases, fig. 2 shows that the amplitude of the limit cycle LC1 increases with its period. Bursting oscillations appear when the limit cycle LC1 approaches the region of phase space where the unstable limit cycle LC2 exists (cf. fig. 3). A homoclinic tangency to LC2 can explain the properties of these oscillations. When the trajectory of LC1 is forced to pass in the vicinity of LC2, the limit cycle is distorted and fast oscillations appear transiently. In fig. 3a, a Poincaré section illustrates how the successive iterates of LC1 approach LC2. At the critical point, the trajectory of LC1 coalesces with LC2. The resulting homoclinic orbit is represented schematically in fig. 3b.

In such a homoclinic tangency to an unstable cycle,
least squares fitting is $\lambda = 0.1299 \pm 0.0014 \, \text{ms}^{-1}$. (other parameters are given in the caption of fig. 4).

If we refer to the scheme of the Poincaré section of the system (fig. 3), then one can deduce that, close to the critical point, the successive iterates follow the unstable direction of the limit cycle (indicated by U in fig. 3c). These particular iterates correspond to the escape from the unstable limit cycle. The distance between each iterate and the unstable cycle should evolve exponentially, with an argument approaching $\lambda t$. Therefore, sufficiently close to the critical point, studying the successive iterates of LC1 on a Poincaré section should allow one to estimate the positive eigenvalue $\lambda$ of LC2. This value must be compared with that obtained from relation (2).

We realized a first return map of the system by considering the successive maxima $X_m(t)$ of the variable $X$ (fig. 5). This quantity seems to obey the following relation,

$$X_m(t) \approx X_{\text{max}} - \exp[\lambda(t-t_0)], \quad (3)$$

where $X_{\text{max}}$ and $t_0$ are constants. If we assume that $X_{\text{max}}$ represents the amplitude of LC2, then the argument of the exponential constitutes an estimation of the eigenvalue corresponding to the unstable direction of LC2. The value obtained from least squares fitting is $\lambda = 0.1299 \pm 0.0014 \, \text{ms}^{-1}$.
of the system is remarkably close to that obtained from period measurements using relation (2). The coincidence of these two values confirms that the bursting oscillations are based here on a homoclinic tangency to an unstable limit cycle.

As a conclusion, we have described a novel type of bursting oscillations which appear in a model of coupled neurons with time delay. Neuronal bursting, such as in the R15 neuron of the Aplysia [1], usually results from the combination of several active intrinsic currents, and therefore is typically a property of the single cell. On the other hand, the oscillations presented here are due to the interaction between several cells. The combination of leakage current, synaptic currents and time delays are the basis of the type of bursting oscillations described here.

We show that the mechanism underlying this type of oscillations is a homoclinic tangency to an unstable limit cycle. Similar types of bifurcation have been described previously in nonlinear systems with time delays (see for example ref. [20]). Let us mention also a recent work [21] on the transition between chaotic bursting and continuous spiking in a class of three-dimensional ODE models of excitable membranes. However, the model we describe here is a two-dimensional delay differential equation system without chaotic behavior in the studied region of parameters. Nevertheless, both the present paper as well as ref. [21] use homoclinic orbits but at different levels of the analysis. In the present model, we assume a unique and global homoclinic orbit to a limit cycle in the full time delay system (1) although, in ref. [21], a homoclinic orbit to a fixed point is assumed in a 2D fast subsystem at the singular limit (ε=0) of the 3D ODE system of ref. [21]. To be complete, let us add here that global homoclinic orbits also exist in the full 3D ODE system of ref. [21] by a general mechanism similar to the homoclinic tangency to a Smale horseshoe in ODEs of ref. [16]. For further discussion, see ref. [22].

If this system is generalized to a network of excitatory and inhibitory neurons, oscillatory behavior can turn into spatiotemporal chaos if Ωj is increased (cf. refs. [9–11]). Preliminary results [10,11] indicate that in this case, the apparition of spatiotemporal chaos also seems to be related to homoclinic phenomena.

Comparing this type of bursting oscillations to previously described mechanisms [6] shows that, here also, a “slow” oscillatory process is associated to the stable limit cycle LC1, whereas the unstable limit cycle LC2 is characterized by a faster time scale. On the other hand, in this system, the “fast” oscillation characterizing the active phase of the bursting oscillation is not associated to a stable oscillating branch in the fast subsystem. It rather corresponds to an approach to an unstable oscillating state.

The characteristic logarithmic scaling of the period near the critical point allows an identification of homoclinic phenomena from the analysis of experimental time series [17]. In the case described here, the lengthening of the period also follows such a logarithmic law. Therefore, if such bursting phenomena can be observed experimentally, the measure of the scaling of the period near the critical point should allow the identification of this type of bifurcation.

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References


