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

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\begin{align*}
\frac{dX}{dt} &= -g_L(X-V_L) - g_{EE}(X-V_E)F(X(1-\tau)) \\
&\quad - g_{EE}(X-V_I)F(Y(t-\tau)) , \\
\frac{dY}{dt} &= -g_L(Y-V_L) - g_{IE}(Y-V_E)F(X(t-\tau)) \\
&\quad - g_{II}(Y-V_I)F(Y(t-\tau)) ,
\end{align*}

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 $V_i = -60 \text{ mV}$ 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]). $g_{EE}$, $g_{IE}$, $g_{II}$ and $g_{II}$ 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

\[ F(V) = \frac{1}{1+\exp\left(-\frac{1}{2}(V+25)\right)} . \]

This sigmoidal function gives the output activity of the neuron as a function of its potential $V$. For the most negative values of the potential, the neuron is silent and $F \approx 0$. Above the threshold value of $V \approx -50 \text{ mV}$, the output activity of the neuron increases and saturates to a maximum value of $F \approx 1$ for higher potentials. The time delay due to signal propagation and synaptic transfer is $\tau = 4 \text{ ms}$.

These equations can be derived from models describing the dynamics of the membrane potential of a network of excitatory and inhibitory neurons [8,9]. If one restricts the network to have uniform solutions, then eqs. (1) are obtained (cf. refs. [10,11]). Thus, this system can also be viewed as describing the uniform solutions of a network of excitatory and inhibitory neurons.

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$^{-1}$): $\gamma = g_L/C_m$, $\Omega_1 = g_{EE}/C_m$, $\Omega_2 = g_{IE}/C_m$, $\Omega_3 = g_{EI}/C_m$ and $\Omega_4 = g_{II}/C_m$. 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 $V_{1}$. 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 $\{\Omega_1, ..., \Omega_4\}$ 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 that can appear.

![Fig. 1. Stability diagram of the system of interconnected neurons. The fixed points of the variable X are shown here as a function of the parameter $\Omega_1$. They were obtained numerically using a Newton-Raphson algorithm [14]. Linear stability analysis of these fixed points shows that limit cycle oscillations occur in the range of values between $\Omega_1^H$ (Hopf bifurcation) and $\Omega_1^F$ (infinite period bifurcation). The legends are: SFP=stable fixed point, UFP=unstable fixed point, I=intermediate branch, H=Hopf fixed point, L=lower branch, U=upper branch, LFP=lower fixed point, UFP=upper fixed point, E=excitatory, I=inhibitory.](image)
which appear from an infinite period bifurcation ($\Omega^\infty$ in fig. 1). As shown in fig. 1, a very similar diagram is seen for different values of the time delay. A closer scrutiny of fig. 2 reveals that, during the approach to the critical point $\Omega^c$, the period continuously increases together with the number of pseudo...
least squares fitting is $\lambda = 0.1299 \pm 0.0014$ 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 $\alpha$-
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 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 characteristics appear in cells...

