A two–variable model of somatic–dendritic interactions in a bursting neuron

Carlo R Laing and André Longtin,
Department of Physics,
University of Ottawa
claing@science.uottawa.ca
alongtin@physics.uottawa.ca

We present a two–variable delay–differential–equation model of a pyramidal cell from a weakly electric fish that is capable of burst discharge. It is a simplification of a 6-dimensional ODE model for such a cell (B. Doiron et al., J. Comput. Neurosci., **12**(1), 2002). We have modeled the effects of back–propagating action potentials by a delay, and use an integrate–and–fire mechanism for action potential generation. The simplicity of the model allows us to derive a map for successive interspike intervals, and to analytically investigate the effects of time–dependent forcing on such a model.

A number of mathematical models of bursting cells have been developed, but they are generally difficult to treat analytically in any detail. Much past analysis of bursting cells has been influenced by the "slow–fast" separation of timescales in bursting systems where it is assumed that fast, spiking variables act on a much shorter timescale than the slow variable(s) that are responsible for the shifts between spiking and quiescent behavior.

Recently, a new mechanism for burst discharge in pyramidal cells of the weakly electric fish *Apteronotus leptorhyncus* was investigated [1]. These cells receive input directly from electroreceptor cells on the fish's skin, and are thought to play a significant role in the processing of electrosensory information. The model presented in [1] was a set of six coupled nonlinear first–order ordinary differential equations, which was a reduction from the multicompartment model involving over 1500 variables presented in [2]. This reduction was obtained by lumping the many compartments into two, representing the soma and the dendrite, and by ignoring the dynamics of the channels not thought to be important in the mechanism for bursting. That the model in [1] reproduced both the bursting behavior observed in the model of [2] and experimentally observed bursts [3] indicates that this process was successful.

The model analyzed in [1] was studied using the "slow–fast" approach of others but it differed from all previous bursting models in that it had only one slow variable; when that variable was held constant, the remaining "fast" system did not show bistability. The bifurcation in the fast system that ended a burst was found to be a transition from period–one to period–two behavior associated with the failure of a somatic action potential to induce a dendritic one, and the interburst interval was found to involve the passage in phase space near a fixed point. Several aspects of the timing of bursts were found to be related to the distance in parameter space from a saddle–node bifurcation, hence the name "ghostbursting".

In this work we further reduce the model in [1] to a set of two discontinuous delay differential equations, from which a two–dimensional map can be derived (assuming constant input current). This gives us analytical insight into complex soma–dendrite interactions. This reduced model can be constructed because, as a result of the work in [1], we understand the essential ingredients of this type of bursting. A short time after most somatic spikes, current flows from the dendrite to the soma, producing a depolarizing afterpotential (DAP) at the soma. For large enough current injected to the soma, the sizes of these DAPs slowly increase due to a slow inactivation of the dendritic potassium that is responsible for the repolarization of dendritic action potentials. This results in progressively smaller inter–spike intervals (ISIs), and this process continues until an ISI is smaller than the refractory period of the dendrite. Once this happens, there is dendritic spike failure, which removes the normal current flow to the soma, and a DAP does not appear. This results in a long ISI, during which the variable controlling inactivation of dendritic potassium increases, and the sequence starts again. The spike patterning is similar to that seen in the multicompartment model in [2] and in experimental recordings [3].

If we consider gradually increasing the DC current injected into the soma of such an actual pyramidal cell, it is observed that the cell's behavior changes from quiescent to periodic firing and then to bursting [3], and this behavior is seen in the pyramidal cell models [1, 2]. The model presented here also shows these transitions.

In this work we also consider periodically modulating the current applied to the model neuron, and for the case of sinusoidal modulation we obtain a three–dimensional map for successive spike times. This map can be used to determine the boundaries in parameter space of resonance tongues, in which the neuron's firing frequency is locked to that of the forcing.

The two–dimensional model that we have developed would be very useful for large–scale simulations of networks of such neurons as it has fewer variables than common neuron models that involve ionic channels [1], the differential equations involved are not stiff, and its piecewise linear nature aids its analysis. The way we have modeled backpropagation of an action potential along a dendrite and the resulting "ping–pong" effect by a discrete delay in a low–dimensional system is also novel. This type of modeling could possibly be applied to other systems in which backpropagation in active dendrites (or more generally, having different voltages in different parts of a cell at the same time) is thought to be important.

References

[1] Doiron, B., C. R. Laing, A. Longtin and L. Maler (2002). "Ghostbursting": a novel bursting mechanism in pyramidal cells. *J. Comput. Neurosci.* **12**(1) (to appear).

[2] Doiron, B., A. Longtin, R. W. Turner and L. Maler (2001). Model of gamma frequency burst discharge generated by conditional backpropagation. *J. Neurophysiol.* **86** 1523-1545.

[3] Lemon, N. and R. W. Turner (2000). Conditional spike backpropagation generates burst discharge in a sensory neuron. *J. Neurophysiol.* **84** 1519-1530.