

Biophysically based multicompartmental models [2,3] have been successful in reproducing these observed bursts, and with the aid of experimental results [10,14], the mechanism involved in bursting has been understood. In summary, the dendrite and soma of the neuron are both capable of producing action potentials, but the refractory period of the dendrite is longer than that of the soma. During a burst, the dendritic action potentials (which follow the somatic ones via backpropagation) effectively provide a weak positive feedback to the soma which results in depolarizing afterpotentials (DAPs) at the soma, and the sizes of these DAPs increase on a slow time scale, due to slow inactivation of dendritic K^+ channels—this is responsible for the increasing instantaneous frequency at the soma. The burst terminates when a somatic ISI is smaller than the refractory period of the dendrite, so the dendrite no longer fires an action potential in response to one at the soma, and the effect of the positive feedback is rapidly removed, producing a long ISI that groups spike clusters into bursts. The burst mechanism (labeled “ghostbursting”) is explained in greater detail in Refs. [2,3,10,14].

One consequence of this form of bursting is that if the magnitude of a DC current injected to a pyramidal cell is slowly increased, the cell changes from quiescent to tonic (periodic) firing of action potentials to bursting. The two bifurcations separating the three types of behavior were determined in Ref. [2] to be a saddle-node bifurcation of fixed points on a circle, and a saddle-node bifurcation of periodic orbits, respectively. This sequence is in contrast to many other burst mechanisms, where the sequence is quiescent \rightarrow bursting \rightarrow tonic firing, as applied current is increased. The “burst threshold” in the ghostburster is demonstrated experimentally in Ref. [10] and discussed further in Ref. [2]. This threshold is very important if these neurons are involved in feature detection [6] and information processing [11], since information from other cells will be manifested as a change in input current to a pyramidal cell, which may then cause a change from periodic firing to bursting or vice versa.

In Ref. [3], a multicompartment model of an ELL pyramidal cell was presented, and in Ref. [2] a simplified version of this model was analyzed. Bifurcation analysis was done using the injected current to the soma and the dendritic potassium conductance as parameters. In this paper, we extend the bifurcation analysis using (a) the soma to dendrite coupling conductance, and (b) the ratio of the somatic area to the area of the whole cell, as parameters.

2. Model

The model consists of two isopotential compartments, representing the soma and dendrite of the neuron. They are diffusively coupled through voltage, following Refs. [12,13]. The equations, previously presented in Ref. [2], are

$$C \frac{dV_s}{dt} = I - g_{Na,s} [m_{\infty,s}(V_s)]^2 (h_0 - n_s)(V_s - V_{Na}) - g_{K,s} n_s^2 (V_s - V_K) - g_L (V_s - V_L)$$

$$\frac{dn_s}{dt} = \frac{n_{\infty,s}(V_s) - n_s}{\tau}$$

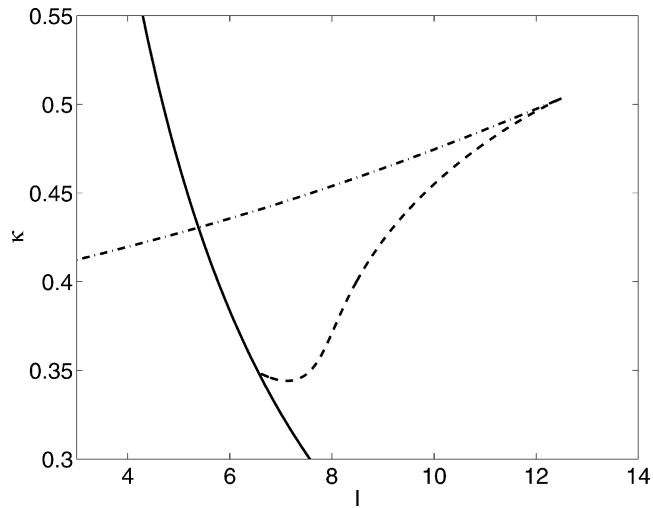


Fig. 1. Bifurcation set using I and κ as bifurcation parameters. The solid curve indicates a saddle-node bifurcation of fixed points on a circle, dashed is a saddle-node bifurcation of periodic orbits (one of which is stable) marking the tonic to burst transition, and the dashed-dotted is a saddle-node bifurcation of periodic orbits, both of which are unstable. $g_c = 1$.

The behavior in Fig. 1 can be understood as follows: decreasing κ increases the effect of the last term in Eq. (1), leading to a bigger DAP at the soma, and is qualitatively the same as decreasing $g_{K,d}$. Decreasing $g_{K,d}$ was found in Ref. [2] to move the curve of saddle-node bifurcations of periodic orbits closer to the curve of saddle-node bifurcations of fixed points on a circle, such that they eventually meet at a codimension-two point. For $\kappa < \sim 0.35$, the neuron switches from quiescence to doublets as I is increased,

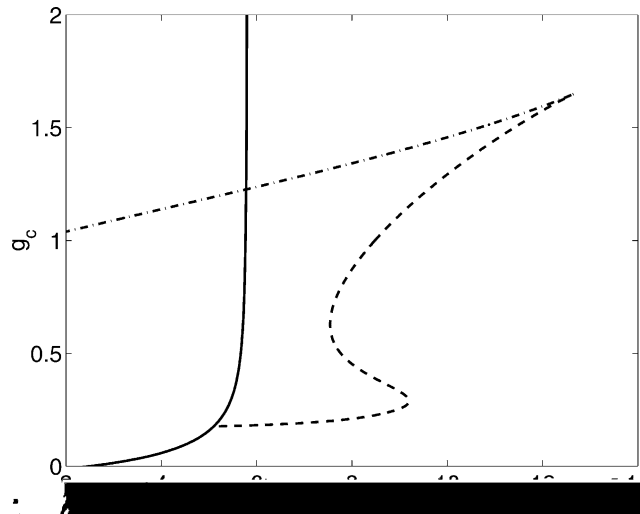


Fig. 2. Bifurcation set using I and g_c as bifurcation parameters. The solid curve indicates a saddle-node bifurcation of fixed points on a circle, dashed is a saddle-node bifurcation of periodic orbits (one of which is stable), and the dashed-dotted is a saddle-node bifurcation of periodic orbits, both of which are unstable. $\kappa = 0.4$.

For large g_c , the voltages in the soma and dendrite track one another very closely. An essential ingredient for bursting is the dendritic to somatic current that causes the DAP, and specifically, the slow growth of the DAP. In this model, this current is purely a result of the different halfwidths of the somatic and dendritic action potentials—the somatic is normally shorter than the dendritic. If the somatic and dendritic voltages track one another closely, the effect of these different halfwidths, and thus the DAP, is removed. Hence there is no bursting for large g_c . However, when g_c is small, the effective coupling between the soma and dendrite is also small. The soma is not capable of bursting by itself, so for small g_c , the neuron moves from quiescence to tonic firing as I is increased, in the same way as a single-compartment type I neuron [4].

4

We have investigated the effects of varying parameters related to the coupling between the soma and dendrite of a two-compartment model of a pyramidal cell that undergoes “ghostbursting” [2]. We have found that both parameters must have moderate values for bursting to occur. This can be explained in terms of the effects of changing these parameters on the known mechanism involved in bursting [2,3]. It is of interest to vary κ because pyramidal cells in the ELL have been measured to have a wide range of κ values, and furthermore, cells with smaller κ are more likely to burst via soma–dendrite interactions [1], in agreement with our results.

- [1] J. Bastian, J. Nguyenkim, Dendritic modulation of burst-like firing in sensory neurons, *J. Neurophysiol.* 85 (2001) 10–22.