An activity-induced transition in single-cell spiking dynamics

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In the majority of conductance-based single-neuron studies, the reversal potentials for ionic species are kept fixed. During extended activation of a neuron over several seconds, however, ionic concentrations across the membrane can be expected to vary [1]. Here, we demonstrate that these changes can induce a codimension-two bifurcation between different spike-onset mechanism with consequences for a neuron's encoding properties and spiking statistics. We show that while $Na^+ - K^+-$ ATPases contribute to reversing this trend, their action takes time and a neuron is transiently pushed into a different dynamical regime.

Specifically, we combine a conductance-based neuronal point model with previously described dynamics of the $Na^+ - K^+-$ATPase and allow intracellular and extracellular ionic concentrations to vary. At first glance, the net current produced by the pump reduces a cell's excitability comparable to the adaptation mediated by slow potassium channels. In addition, the transiently unbalanced changes in ionic concentrations also result in the less predictable, yet computationally relevant transition in neuronal dynamics. In particular, for neurons with an initial spike generation via a saddle-node-on-an-invariant-cycle (SNIC) bifurcation, ion flow eventually shifts spike generation into a homoclinic regime (via the so-called saddle-node-loop bifurcation) [2]. Interestingly, prolonged activity also induces bistable firing: the cell translates into a mode of intermittently interrupted firing (stochastic bursting). We hence predict that such a firing mode could be a prominent property of strongly stimulated neurons. Its functional relevance for computation in local networks remains to be explored.

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References

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