

PACEMAKER RHYTHM BY CELLULAR AUTOMATA

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ABSTRACT

A short time of insomnia is enough to trigger our awareness and reflection on mechanisms driving the heart. The sinoatrial node (SAN)—a small structure in the right atrium, is the main pacemaker for the heart. The question arises how this node can deliver more than 90 thousand beats per day, 365 days a year for many years?

Basic mechanisms of self-stimulation of individual nodal cells are well grounded and accepted. However how nodal cells contribute to overall pacemaker function is still unclear. Paradigms such as reaction-diffusion equations or coupled lattice maps are commonly used in modeling excitable systems. But complexity of these models increases dramatically as molecular details are added. Therefore the expectation is growing to shift the model perspective from the component-level to the system-level. Such perspective can be achieved by cellular automata approach because cellular automata modeling is pragmatic.

Cellular automata have been successfully used in explaining collective phenomena like continuous phase transition or self–organized criticality. In biological systems the self–organization manifests as wide–spread oscillatory–type dynamics of coupled oscillating units. In the lecture, I will describe the system of cellular automata, tightly motivated by experimental data, which is able to address the question on the role of heterogeneity in intercellular connections. Moreover, the model reproduces many aspects of the electrical properties of the SAN tissue. First of all, it explains emergence of the leading pacemaker site as the result of self–organization process for which the structure of inter–cellular connections is crucial. Furthermore, effects which are known to be related to the biological aging of SAN, such as structural changes in cell–to–cell interactions due to collagen deposition and/or impairment of the expression of genes responsible for gap junctions, could be demonstrated within the model. All these phenomena can be efficiently simulated on commercial computers since cellular automata approach reduces the computational burden.

REFERENCES

D. Makowiec: Modeling heart pacemaker tissue by a network of stochastic oscillatory cellular automata, LNCS 7956 (2013), 138–149.