

XXX KKZMBM

Wikno, 16th–20th September 2025

DYNAMICS OF INFARCTION IN THE HUMAN CARDIAC CONDUCTION SYSTEM: A CELLULAR AUTOMATA APPROACH

Beata Jackowska-Zduniak¹

¹Faculty of Applied Physics and Mathematics, Gdańsk University of Technology
ul. Narutowicza 11/12, 80-233 Gdańsk

¹beazduni@pg.edu.pl

ABSTRACT

In this contribution, we present a cellular automaton model that reproduces the microstructure and bioelectric conduction of the human cardiac conduction system, with particular emphasis on the sinoatrial node (SAN). The model accounts for cellular differentiation (myocytes, fibroblasts, collagen) as well as the presence of connexins (Cx43, Cx45, Cx40) with their assigned conduction resistance. Infarction in the SAN head region is represented as a progressive gradient affecting both cellular biophysical properties and connexin degradation. Our results demonstrate that the progression of infarction disrupts SAN conduction, interrupts the signal to the atria, and may trigger an escape rhythm from the atrioventricular node (AVN). Moreover, the model reveals electrophysiological phenomena not previously reported in the SAN context, though known from neurophysiological studies. By reproducing physiological mechanisms, the approach provides a promising tool for investigating cardiac pathology and exploring potential therapeutic strategies.

REFERENCES

- [1] A. Kalyanasundaram, N. Li, and et al.: *Canine and Human Sinoatrial Node: Differences and Similarities in Structure, Function, Molecular Profiles and Arrhythmia*, J Vet Cardiol **22** (2019), 2–19.
- [2] T. A. Csepe, A. Kalyanasundaram, and et al.: *Fibrosis: a structural modulator of sinoatrial node physiology and dysfunction*, Front. Physiol. **6** (2015), 1–8.
- [3] P. Camelliti, G. P. Devlin, K. G. Matthews, P. Kohl, and C. R. Green: *Spatially and temporally distinct expression of fibroblast connexins after sheep ventricular infarction*, Cardiovasc Res. **62** (2004), 415–425.
- [4] T. A. Csepe, J. Zhao, B. J. Hansen, and N. Li: *Human sinoatrial node structure: 3D microanatomy of sinoatrial conduction pathways*, Prog Biophys Mol Biol. **120** (2016), 164–178.
- [5] C. Campana, E. Ricci, C. Bartolucci, S. Severi, and E. A. Sobie: *Coupling and heterogeneity modulate pacemaking capability in healthy and diseased two-dimensional sinoatrial node tissue models*, PLoS Comput Biol **18** (2022), 1–21.
- [6] J. P. Ugarte, E. A. Perez Alday, and L. M. Rocha: *Modeling myocardial ischemia with cellular automata: Effects on cardiac conduction and excitability*, Applied Mathematical Modelling **105** (2022), 534–550.