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Impact of limits in pathways between sinoatrial node and atrium on heart rhythm by timed automata model.

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There are evidences that the human right atrium and sinoatrial node (SAN) are functionally separated except at discrete SAN-atrial electrical junctions, called SAN-exit-pathways. We hypothesize that this type anatomy is a source of re-entry around the SAN. A computationally efficient model will be presented which reconstructs human right atrium electrophysiology. Activity of a myocyte is simulated by a timed automaton with continuous and discrete transitions reproducing stages of cellular membrane. A stochastic 2D-network of timed automata is designed to model the right atrium architecture: SAN, atria-ventricular node (AVN), SAN-exit-pathways and heterogeneous atrial tissue.

Simulations were performed to measure effects of quantity of SAN-exit-pathways: all-, half-, few-cells connections, on development and propagation of normal versus arrhythmic excitations: SAN-re-entry or fibrillation. Additionally, two parameters were controlled to measure an influence of (1) atrial tissue fibrosis: p_{trans} - probability for transversal intercellular network connections, and (2) impairment of individual cells: p_{refuse} - probability of a cell to refuse to excite. Simulations provided a critical relationship between atrial anatomy and rhythm of heart excitations. A discrete model of cells and intercellular connections was found as efficient method for quantifying this relationship.