When Heart Fibrosis Becomes a Disease

Beata Jackowska-Zduniak[1]

[1] *Gdańsk University of Technology*

Fibrosis is a hallmark of cardiac aging and is traditionally viewed as a physiological adaptation. In the sinoatrial node (SAN) and atrioventricular node (AVN), moderate fibrotic remodeling may stabilize tissue architecture. However, with progressive aging, the accumulation of fibrotic tissue and the loss of functional cardiomyocytes disrupt impulse generation and conduction.

Using a cellular automaton model, this study simulates age-related changes in the heart's conduction system. The results show that excessive fibrosis within the SAN impairs pacemaker activity, while fibrotic barriers in atrial tissue and around the AVN lead to conduction delays or blocks. These structural alterations contribute to the development of arrhythmias, particularly sinus bradycardia and atrioventricular block, both commonly observed in elderly individuals.

Where, then, lies the boundary between physiological remodeling and disease? Recognizing this transition is essential to understanding age-related cardiac dysfunction and may guide future therapeutic strategies focused on preserving conduction system integrity and cardiomyocyte viability.

[1] Beata Jackowska-Zduniak. A Simplified Heart Age Model Based on Cellular Automata. Nunes-Gonzales J. David, Grana Manuel (red.): ***Modelling and Simulation'2024. The European Simulation and Modelling Conference*** (2024), 114–121.