

The Dynamics of Atrial Fibrillation in a Cellular Automaton Model

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Atrial fibrillation (AF) is the most common abnormal heart rhythm and the single biggest cause of stroke. AF occurs as self-terminating episodes which can increase in duration to become persistent. The progression to persistent AF differs significantly between patients and is poorly understood. Current clinical success rates in curing persistent AF are poor ($< 50\%$) [1] and could be improved through enhanced mechanistic understanding of the disease process. We show using a cellular automaton model of heart muscle tissue [2] that patient variability arises naturally from the interaction between the underlying tissue structure and the dynamics of activation wavefronts. As the cell-cell coupling is reduced, as occurs with age, we observe spontaneous episodes of self-terminating AF which progress to become persistent. The accumulation of critical sub-regions of tissue causes the transition from self-terminating to persistent AF. We show that the destruction of such sub-regions, as done clinically, can terminate or delay progression to persistent AF. Thus, the simple model reproduces the dynamics of AF and provides a novel therapeutic target: structurally critical sub-regions of tissue.

[1] A.N. Ganesan et al. J Am Heart Assoc 2.2 e004549 (2013).

[2] K. Christensen, K.A. Manani, N.S. Peters, Phys. Rev. Lett. **E99**, 99998 (2015).