

Wavefront-Obstacle and Wavefront-Wavefront Interactions as Mechanisms for Atrial Fibrillation: A Study Based on the FitzHugh-Nagumo Equations

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Atrial fibrillation (AF) is the most common arrhythmia of the heart in the industrialized countries. Understanding the mechanisms that lead to AF is at the root for improving existing or developing new therapies. In this work we investigate, based on the FitzHugh-Nagumo (FHN) equations for excitable media, certain generating mechanisms for AF that were suggested in the literature. In particular, we study the influence of locally modified cell properties (obstacles) on the propagation of initially planar excitation waves. In contrast to earlier studies, which considered passive or insulating obstacles, we consider areas where the cell properties vary gradually over some finite correlation length. We find that a transition from functional to anatomical reentry occurs in dependence of both the reduction strength of excitability and the size of the correlation length. This behaviour is characterized in terms of dynamical phase diagrams.

In order to tackle the question how regular self-excitatory sources as stable spiral waves in the left atrium can possibly lead to irregular, fibrillatory excitation patterns in the right atrium, we furthermore investigate the perturbation of regularly paced waves, representing, for example, those emanating from the sinus node, by regular waves emanating from another pacemaker, as, for example, a spiral wave. Both sources of the excitation waves are located in two distinct regions of the simulation area and are connected by a small bridge only. Here we find from the solutions of the FHN equations that the perturbation of the regularly paced waves by the waves emanating from the self-excitatory pacemaker can lead to fibrillatory states, thus confirming corresponding conjectures discussed in the literature. The extent of irregularity of these fibrillatory states is quantified in terms of an entropic measure in dependence of the frequency of the perturbing self-excitatory pacemaker.