DY 10: Focus session: Nonlinear Dynamics of the Heart I (organized by Markus Bär, Stefan Luther and Ulrich Parlitz)

Time: Wednesday 15:00–16:30 Location: H6

Invited Talk DY 10.1 Wed 15:00 H6

Effect of fibrosis on propagation on non-linear waves and onset of arrhythmias in cardiac tissue — •Alexander Panfilov[1,2], Timur Nezlobinsky[1,2], and Farhad Pashakhanloo[3] — [1]Department of Physics and Astronomy, Ghent Unibversity, Belgium — [2]Ural Federal University, Ekaterinburg, Russia — [3]Cardiovascular Division, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, Massachusetts, 02115, USA

Cardiac fibrosis is a well-known arrhythmogenic condition which can lead to sudden cardiac death. Physically, fibrosis can be viewed as a large number of small obstacles in an excitable medium, which may create nonlinear wave turbulence or reentry. The relation between the specific texture of fibrosis and the onset of reentry is of great theoretical and practical importance. In my talk I present results of several recent studies which show how basic properties of wave propagation are affected by fibrosis. We also characterize properties of fibrotic texture which led to cardiac arrhythmias and propose a concept of minimal functional cluster which allows quantitatively predict the arrhythmia probability for different fibrosis densities and tissue excitabilities.

DY 10.2 Wed 15:30 H6

The mechanism of defibrillation of cardiac tissue by time-periodic low-energy shocks I: Refractory boundary length is key for prediction of success probabilities — •Markus Bär, Pavel Buran, and Thomas Niedermayer — Physikalisch-Technische Bundesanstalt, Abbestr. 2 - 12, 10587 Berlin

Rotating excitation waves and electrical turbulence in cardiac tissue are associated with arrhythmias such as life-threatening ventricular fibrillation. Experimental studies have shown that a periodic sequence of four or more electrical far-field pulses is able to terminate fibrillation with less energy than a single shock protocol. During this so-called periodic low-energy anti-fibrillatory pacing (LEAP), only tissue near sufficiently large conduction heterogeneities, such as large coronary arteries, is activated. By means of simulation of the impact of periodic pacing on fibrillation in a two-dimensional electrophysiological model exhibiting multiple stable spirals (vortices) with a representative array of heterogeneities, we show that the success probability for defibrillation depends exponentially on the length of the refractory boundary, i. e. the total length of the borders between refractory and excitable parts of the tissue. This exponential dependency is also derived analytically from simple arguments assuming that successful defibrillation by a low energy shock requires not only to annihilate all vortices, but also needs to prevent initiation of new vortices in the vulnerable excitable region near the refractory boundary.

DY 10.3 Wed 15:45 H6

The mechanism of defibrillation of cardiac tissue by time-periodic low-energy shocks II: Subsequent shortening of refractory boundary length enables low energy antifibrillatory pacing (LEAP) — •Pavel Buran, Thomas Niedermayer, and Markus Bär — Physikalisch-Technische Bundesanstalt, Abbestr. 2 - 12, 10587 Berlin

We present a generic mechanism for the success of LEAP protocols, which covers termination of multiple stable rotors as well as of states of spatiotemporal chaos. Previously, we found that knowledge of the refractory boundary length is sufficient to estimate the success probability of an individual LEAP pulse which is found to decay exponentially with this length in a medium with stable spirals. This result is also found in simulations of cardiac models exhibiting spatiotemporal chaos. Whereas single shock defibrillation requires instantaneous annihilation of all existing vortices, during LEAP the defibrillation process is more gradual and is based on a subsequent shortening of the total refractory boundary length. The average shortening factor, i. e. the ratio between the refractory boundary lengths just before subsequent pulses during periodic pacing can be determined numerically both for media with spatiotemporal chaos and multiple stable spirals and provides a good indicator for the efficiency of a given LEAP protocol.

Invited Talk DY 10.4 Wed 16:00 H6

Chaos and nonlinear dynamics in the heart: Experiments and simulations of arrhythmias and defibrillation — •Flavio fenton — School of Physics, Georgia Institute of Technology, Atlanta, GA

In this talk we present experimental examples of chaotic dynamics including unstable periodic orbits (period 3 and higher orders) in the heart. As a nonlinear system we further demonstrate a universal mechanism for terminating spiral waves in generic excitable media using an established topological framework. Under this mechanism it is possible to explain when defibrillation shocks, by high- or low-energy methods, succeed or fail. Furthermore, it is also possible to design a single minimal stimulus capable to defibrillate, at any time, any turbulent state driven by multiple spiral waves. We demonstrate this in a variety of cardiac tissue models. The theory described here shows how this mechanism underlies all successful defibrillation and can be used to further develop existing and future low-energy defibrillation strategies.