BP 12: Focus: Nonlinear Dynamics of the Heart (with DY)

Time: Wednesday 9:30–12:00 Location: MA 001

Invited Talk BP 12.1 Wed 9:30 MA 001

Modelling Excitation Contraction Coupling — •Martin Falcke — Max Delbrück Centrum für Molekulare Medizin Berlin

We present an efficient but detailed approach to modelling Ca^{2+}-induced Ca^{2+} release in the diadic cleft of cardiac ventricular myocytes. We developed a spatial resolved Ca^{2+} release unit (CaRU), consisting of the junctional sarcoplasmic reticulum and the diadic cleft. Individaul channels are modelled by Markov chains. By taking advantage of time scale seperation, the model could be finally reduced to only one ordinary differential equation for describing Ca^{2+} fluxes and diffusion. Additionally the channel gating is described in a stochastic way. The resulting model is able to reproduce experimental findings like the gradedness of SR release, the voltage dependence of ECC gain and typical spark life time.

Invited Talk BP 12.2 Wed 10:00 MA 001

Modeling of electrical and mechanical function of the heart — •Alexander Panfilov — Department of Physics and Astronomy, Gent University, Krijgslaan 281, S9, 9000 Gent, Belgium

Cardiac arrhythmias and sudden cardiac death is the leading cause of death accounting for about 1 death in 10 in industrialized countries. Although cardiac arrhythmias has been studied for well over a century, their underlying mechanisms remain largely unknown. One of the main problems in studies of cardiac arrhythmias is that they occur at the level of the whole organ only, while in most of the cases only single cell experiments can be performed. Due to these limitations alternative approaches such as mathematical modeling are of great interest. From mathematical point of view excitation of the heart is described by a system of non-linear parabolic PDEs of the reaction diffusion type with anisotropic diffusion operator. Cardiac arrhythmias correspond to the solutions of these equations in form of 2D or 3D vortices characterized by their filaments. In my talk I will briefly report on main directions of our research, such as development of virtual human heart model, and study organization of ventricular fibrillation due to dynamical instabilities in cardiac tissue and due to tissue heterogeneity. I will also report on modeling mechano-electric feedback in the heart using reaction-diffusion mechanics systems and ventricular fibrillation mechanisms due to deformation of cardiac tissue.

Invited Talk BP 12.3 Wed 10:30 MA 001

Mechanisms for calcium alternans — •Blas Echebarria, Enric Alvarez-Lacalle, Carlos Lugo, Angelina Peñaranda, and Inma R. Cantalapiedra — Departament de Física Aplicada, Universitat Politècnica de Catalunya, 44-50 Av. Dr. Marañón, 08028 Barcelona, Spain

Alternans is a well-known cardiac pathology, in which the duration of the action potential (AP) alternates at consecutive beats. Due to its proarrhythmic effects it is important to understand the mechanisms underlying its genesis. It has been amply studied the case where alternans appears due to a steep relationship between the duration of an action potential and the time elapsed since the end of the previous AP. However, now it is widely accepted that alternans often appears due to instabilities in the dynamics of intracellular calcium cycling (itself an important messenger for the contraction of the cell). This instability can be due to a steep relationship between the amount of calcium released to the cytosol, and the calcium loading of the sarcoplasmic reticulum (SR), but also due to a slow recovery of the channels that regulate the release from the SR.

Invited Talk BP 12.4 Wed 11:00 MA 001

Synchronization as a mechanism of chaos control; Applications to cardiac arrhythmias. — •Flavio H. Fenton[1], Stefan Luther[1,2], Philip Bittihn[2], Daniel Hornung[2], Eberhard Bodenschatz[2], and Robert F. Gilmour Jr[1] — [1]2Department of Biomedical Sciences, Cornell University, Ithaca, New York, USA — [2]1Max Planck Institute for Dynamics and Self-Organization, Goettingen, Germany

The heart is an excitable system, with electrical waves propagating in a coordinated manner to initiate a mechanical contraction. In pathologic states, normal electrical wave propagation can be disrupted, resulting in the development of spiral and scroll waves that repetitively excite the tissue. These waves are often unstable and break into multiple waves, a chaotic state that underlies cardiac fibrillation.

In this talk, we will discuss experimental and theoretical approaches for the control and termination of arrhythmias using low energy pulses. We will show how naturally occurring discontinuities in cardiac tissue conductivity can produce internal electrical activations following an electric field and how this *virtual electrode activations* can be used to synchronize and terminate arrhythmias with just 10% the energy of a standard defibrillation shock. Numerical simulations as well as experimental data from in vivo experiments will be presented along with a theory for the mechanism.

Invited Talk BP 12.5 Wed 11:30 MA 001

Cardiac dynamics from a nonlinear system's perspective - from basic science to applications — •Stefan Luther — Max Planck Institute for Dynamics and Self-Organization, Goettingen, Germany — Department of Biomedical Sciences, Cornell University, Ithaca, NY

Self-organized complex spatial-temporal dynamics underlies cardiac arrhythmias, a significant cause of mortality and morbidity worldwide. The term dynamical disease was coined, suggesting that they can be best understood from a dynamical system's perspective. The systematic integration experimental data from sub-cellular, cellular, tissue, and organ level to the in-vivo organism into mathematical models is key to the understanding of this complex biological system. The talk will provide an introduction to the biophysics and nonlinear dynamics of the heart, and discuss mechanisms that induce, sustain, and control life-threatening cardiac arrhythmias.