BSICoS
Biomedical Signal Interpretation & Computational Simulation

Computer Modeling to Understand the Failing Heart

Dear Colleagues,

It is a great pleasure to invite you to attend the "Computer Modeling to Understand the Failing Heart" seminar by

Prof. MARK POTSE

Date: Tuesday, June 24, 2014

Time: 09:30 am

Location:

Campus Río Ebro, Edificio I+D+i
2ª planta, bloque 5, Seminario I3A
C/Mariano Esquillor, s/n
Zaragoza

The seminar is free and open to the public.

Instituto Universitario de Investigación en Ingeniería de Aragón
Universidad Zaragoza

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Centro Investigación Biomédica en Red
Bioingeniería, Biomateriales y Nanomedicina

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PROF. MARK POTSE

Prof. Mark Potse is a scientist affiliated with the Institute of Computational Science, University of Lugano (Switzerland) and with LIRYC, the Electrophysiology and Heart Modeling Institute, Bordeaux, France.

His research concentrates on 3 topics: myocardial ischemia, ventricular repolarisation heterogeneity , and Development of realistic computer models of the human heart electrophysiology.

He obtain PhD degree in physics, from the University of Amsterdam. He has been active in cardiac electrophysiology since 1994. He worked on computer analysis of multichannel ECG data. Later he joined simulation teachings and today, he is trying to do merge both, in order to better understand the heart behavior and its diseases.

http://www.potse.nl/index.html

Computer Modeling to Understand the Failing Heart

The best way to study a complex system is not by taking it apart and studying its components, but by attempting to put it together from components that are well understood. We are using this approach to improve our understanding of conduction disturbances in the heart-failure patients. Heart failure is a chronic and ultimately fatal condition in which the heart does not pump enough blood to meet the demands of the body. It is often accompanied by disruptions in the electrical activation mechanism which, in normal hearts, synchronizes force development in the cardiac muscle and so ensures an effective contraction of the heart. The precise nature of these conduction disturbances, clinicaly often referred to as as Left Bundle Branch Block, differs between patients and is not well understood in most of them. Better knowledge of the problems in individual patients can help to decide on the best treatment options such as implantation of a pacemaker for cardiac resynchronization therapy.

With ECG analysis alone, and even with electroanatomical mapping alone, it is not possible to characterize the problems in individual patients precisely. Therefore we are investigating the opposite approach. We use detailed, patient-tailored numerical models of individual patients with which we predict, based on hypothesized pathologies, the ECG, ventricular activation order, and morphologies of endocardial catheter electrograms. These predictions are compared with measured results to determine which hypothesis is the most likely in a given patient. Simulations are performed with a large-scale monodomain reaction-diffusion model of the heart, coupled to a bidomain torso model for simulation of ECGs and local electrograms. Simulations typically run on 2048 cores of a Cray XE6 system at the Swiss National Supercomputing Center CSCS.

Results in the first two patients show a correlation of 91% and 86%, respectively, between measured and simulated activation times. Visually matching ECG waveforms and a matching QRS duration were obtained. The results of parameter optimization suggest a wide variety of mechanisms, including early left-ventricular breakthroughs, scar-related block, and slow conduction suggestive of cardiomyopathy. In both patients, assuming re-entry in the left-ventricular Purkinje network worsened the match. We conclude from these pilot results that it is feasible, though presently still very time-consuming to parameterize a heart-torso model with patient-tailored geometry to the ECG and left-ventricular activation sequence of an individual patient. Initial results suggest that abnormal conductivity in the left-ventricular working myocardium plays a role in these patients.

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