



Life Sciences Seminar

Cardiac fibrillation : an interdisciplinary puzzle

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Cardiac fibrillation is a rapid arrhythmia that affects the atria and ventricles, still posing major challenges to public health management. Atrial fibrillation is the predominant cause of embolic stroke while ventricular fibrillation leads to sudden cardiac death. The consensus pictures cardiac fibrillation as being triggered by a source of abnormal activity and maintained by multiple reentries of the electric pulses. The myocardium is seen basically as a syncytium of connected cells. Therefore, a relatively safe combined therapy of anti-arrhythmic drug delivery with catheter ablation has been implemented aiming at knocking off those two factors. After two decades of this strategy, it has been shown to be suboptimal with no foreseeable cure. Recently (10.3389/fphys.2017.01139 and 10.3389/fphys.2019.00480), we have provided evidence and modeling which clash with the accepted wisdom. Incompatibility is uncovered in recordings of the electrical activity of fibrillating human hearts, because multifractal scaling is found, with which the model shows excellent quantitative agreement. The model focuses on a kinetic instability of junctional channels generating a chaotic modulation of conductance. The important multifractal parameter is related to the variance of the strong capacitive electric field, building up across cell junctions. If this is true, during fibrillation, electric recordings have been mistakenly taken for cardiac action potentials instead of a mixture with capacitive currents, invalidating in practice a host of methods for signal visualization and interpretation. A puzzle, common to cardiac recordings and time series in the model, which comes as a surprise, is the absence of log-correlations. Beside being mathematically challenging to understand, this trait imposes strict constraints about the possible underlying mechanism. In this respect, the model also puts under scrutiny unknown mechanisms of the biology of connexins, the ubiquitous proteins which make up gap junction channels.

Wednesday, November 20, 2019 03:00pm - 04:00pm

Mondi Seminar Room 3, Central Building



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