MODELING THE BRUGADA SYNDROME: A SIMPLE MODEL FOR CARDIAC DYNAMICS

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The Brugada syndrome [1] is associated with an abnormal electrocardiogram (ECG), characterized by an elevation of the ST-segment. Since its discovery in 1992, it has gained increasing recognition, and today is believed to be responsible for 4% to 12% of all sudden deaths and around 20% of deaths in patients with structurally normal heart. Recently it has been associated with a mutation in a specific gene (SCN5A), resulting in dysfunction of the sodium channel in the membrane of cardiac cells. However, the link between this channelopathy and the occurrence of arrhythmias or ventricular fibrillation is, as yet, not completely understood. We have constructed a simplified ionic model to study the propagation of the cardiac stimulus through ventricular tissue. The parameters in the model are chosen as to reproduce the action potential obtained with a realistic model [2] of the different myocardium cells (epicardium, M-cells and endocardium), as well as its action potential duration and conduction velocity restitution curves.
Decreasing the strength of the sodium current, the simplified model reproduces well the action potential observed in the Brugada syndrome.
We will discuss under which conditions the propagation of this action potential in tissue can generate reentrant waves producing arrhythmias.

[1] P. Brugada, Brugada J. J. Am Coll Cardiol **E99**, 99998 (1999).

[2] K. H. W. J. ten Tusscher et al. Am. J. Physiol.**286**, H1573 (2004).