Coupled maps analysis of cardiac wave instabilities due to tissue contraction

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Several life-threateaning arrhythmias are related to electrical wave instabilities in the heart. At rest, there is a large gradient of ionic concentration across the myocytes' cell membrane. Given an external stimulus, ion channels at the membrane open, allowing the flux of ions, that results in an increase in transmembrane electrical potential - the depolarization of the cell. This front then propagates along tissue, triggering the contraction of the cardiac cells in a coordinated manner. Instabilities of propagation give rise to wavebreak, the formation of reentry (i.e., tachycardia) and, eventually, a disordered electrical wave pattern (i.e., fibrillation) in which the heart is not able to pump blood and death intervenes in a matter of minutes.

In all the former mechanism, tissue contraction is usually thought to be of no relevance, being just a consecuence of electrical depolarization. However, several studies suggest that it can play an important role in sustaining and/or inducing the instability. In this contribution we will show that a small amount of contraction can in fact give rise to a wave instability, termed alternans, followed by wave blocks and the initiation of reentry.

At a cellular level, an alternans rhythm is characterized by a beat-to-beat change in the duration of the depolarized phase, or action potential (AP). In tissue, this may result in spatially homogeneous patterns of oscillations (concordant alternans, CA), or in domains of out-of-phase oscillations (discordant alternans, DA)[1]. Remarkably, besides all the complex microscopic details neccessary to properly characterize the dynamics of the transmembrane potential, the main characteristics of this instability can be captured considering a mesoscopic approach[2]. In this, a description in terms of coupled maps, relating the action potential duration (APD) and the conduction velocity (CV) of the AP, at a given point, with the local time lapse between the end of an excitation and the beginning of the following one (diastolic interval, DI), reproduces the main characteristic observed during cardiac alternans. Furthermore, close to the transition to alternans, the small oscillations in the APD have been shown generically to obey a Ginzburg-Landau type equation with an additional nonlocal term that causes spontaneous nucleations of domains giving rise to DA[3].

We have recently used a simplified model of cardiac excitation-contraction coupling to study the effect of tissue deformation on the dynamics of alternans[4]. We showed that small stretch-activated currents produce large effects, causing a transition from in-phase to off-phase alternations (i.e. from concordant to discordant alternans) and to conduction blocks. This effect is the result of a generic change in the slope of the CV-restitution curve due to electromechanical coupling. Thus, excitation-contraction coupling plays a relevant role in the transition to reentry and fibrillation. This effect can be traced to a change in the spatial and functional dependence of the CV, that modifies the solutions of the coupled maps. In this contribution, we elaborate further on the details of this transition, analysing the solutions of the coupled maps equations close to the onset of alternans. We find a good agreement between the full ionic equations, the coupled map equations and the reduced description in terms of Ginzburg-Landau amplitude equations, therefore stressing the usefulness of studying simplified models for the undertanding of complex spatio-temporal phenomena in cardiac tissue.

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