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#001-Header-BGRS →

Cardiac Mechanics, Calcium Overload and Arrhythmogenesis

#002-Author-BGRS →

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#004-Key words-BGRS →

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Motivation and Aim: It is well-known that Ca²⁺ overload may cause cardiac arrhythmias. However, possible contribution of the mechanical factors to the arrhythmia in Ca²⁺-overloaded cardiomyocytes has been insufficiently addressed. Earlier we developed a mathematical model of cardiomyocyte electro-mechanical function that predicted a significant role of the intra- and extracellular mechanical factors in arrhythmogenesis. Model prediction was verified in experiments on papillary muscle from the right ventricle of guinea pigs overloaded with calcium [2].

Methods and Algorithms: We utilized the cellular model to study effects of electro-mechanical coupling between cardiomyocytes in a 1D heterogeneous strand formed of 90% of normal (N) cardiomyocytes and 10% of sub-cardiomyocytes with decreased Na⁺-K⁺ pump activity. Single SC-cardiomyocytes do not demonstrate spontaneous activity during isometric contractions at a reference length. Regular fiber twitches at the reference initial cell length were induced by 1 bps electrical stimulation applied at an edge of the strand. Excitation spread along the tissue via electro-diffusional cell coupling followed by cell contractions and force development in the fiber.

Results: Mechanical interactions between N- and SC-cells in the tissue resulted in the spontaneous activity emerged in the SC-zone between the regular stimuli. If the excitation wave spread from SC- to N-region, the SC-cells developed delayed after-depolarizations (DAD) that caused a slowly developing beat-to-beat decrease in the force of fiber contraction. If the excitation spread in opposite direction, DAD in the SC-cells induced reflected downward excitation waves capturing the normal region and followed by extrasystoles in the whole fiber.

Conclusion: The results obtained in the model suggest that ectopic activity may emerge in a sub-critical myocardial region, e.g. comprising cardiomyocytes with moderately depressed Na⁺-K⁺ pump, due to its mechanical interactions in the myocardial tissue. Moreover, such ectopic zone may expand by capturing normal regions in myocardium via the electro-mechanical coupling between cardiomyocytes.

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#007-List_references-BGRS →

1. Katsnelson L.B. et al. (2011) Contribution of mechanical factors to arrhythmogenesis in calcium overloaded cardiomyocytes: Model predictions and experiments. Progress in Biophysics and Molecular Biology. 107(1): 81-89.
2. Lashin S.A., Matushkin Yu.G. (2012) Haploid evolutionary constructor: new features and further challenges. In Silico. Biol. 11(3): 125-135.

