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Heart Rate Variability Alters Cardiac Repolarization and Electromechanical Dynamics

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Abstract

The cardiac action potential depends on both heart rate and intracellular calcium signaling, and the interplay between these two is not well understood. Physiologically, heart rate continuously varies for many reasons, including autonomic regulation, circadian rhythm, body temperature, and stochasticity in cardiac pacemaking. This variability also influences calcium signaling in cardiac cells. In pathological settings, the action potential duration and intracellular calcium concentrations can experience a phenomenon termed alternans, a beat-to-beat alternation, that is arrhythmogenic. The effects of heart rate variability on the generation of such alternans and the electromechanical properties of the cardiac myocyte are not well studied. We employed a discrete-time map model that governs the action potential duration (APD), intracellular calcium levels, and their interactions in a single cardiac myocyte to understand the consequences of heart rate variability. A stochastic heat rate was introduced into the model by adding a Gaussian distributed noise term, with defined standard deviation sigma, to a baseline heart rate. We found that, for fast heart rates, alternans is present in both APD and peak calcium concentration, and that increased sigma, i.e. more heart variability, tended to suppress alternans. Further, the peak calcium alternans are suppressed for smaller values of sigma. This suggests that arrhythmogenic properties can be suppressed due to heart rate variability. Further study will include investigating the effects of sarcoplasmic calcium release and uptake variability.