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Effect of Heart Rate Variability on Electromechanical Dynamics in Cardiac Tissue

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Under normal physiological conditions, the heart rate varies due to autonomic regulation, circadian rhythm, and stochasticity in pacemaking, and this heart rate variability (HRV) influences the cardiac action potential duration (APD), intracellular calcium (Ca) signaling, and the relationship between APD and Ca (i.e., electromechanical dynamics). Recent work has shown that HRV also impacts the formation of cardiac alternans, a beat-to-beat alternation in APD or intracellular Ca. Clinical studies have shown that reduced HRV correlates with an increased risk of arrhythmias. We recently showed in a model of a single cardiac cell that HRV disrupts electromechanical alternans, decoupling APD and peak Ca and decreasing APD variability, suggesting that HRV may play an important role in preventing arrhythmias at the single cell level. While alternans in an individual cell indicates electrical instability, the spatial pattern of alternans in cardiac tissue is a more direct marker for arrhythmia propensity, specifically the appearance of spatially discordant alternans, characterized by a long-short and short-long APD patterns in different regions of tissue. Our work aims to investigate the effect of HRV on spatial dynamics of cardiac cells using a one-dimensional cable model governing the intracellular Ca concentrations, APD, and cell-cell coupling. HRV is modeled as the sum of a baseline pacing period and Gaussian distributed noise term with a specified standard deviation. We investigate the presence and type of alternans over a range of physiological heart rates and degrees of HRV and analyze the formation of conduction block, spatially discordant alternans, and changes in electromechanical concordance. We expect that spatially discordant alternans will transition to spatially concordant with greater HRV, mediated by decoupling of APD and Ca. Overall, we find that HRV can alter spatial patterns of electromechanical dynamics that can lead to arrhythmia formation.