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ARTICLE | VOLUME 120, ISSUE 2, P352-369, JANUARY 19, 2021

Mechanisms of Premature Ventricular Complexes Caused by QT Prolongation

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Published: December 14, 2020 • DOI: https://doi.org/10.1016/j.bpj.2020.12.001 • Check for updates

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Abstract

QT prolongation, due to lengthening of the action potential duration in the ventricles, is a major risk factor of lethal ventricular arrhythmias. A widely known consequence of QT prolongation is the genesis of early afterdepolarizations (EADs), which are associated with arrhythmias through the generation of premature ventricular complexes (PVCs). However, the vast majority of the EADs observed experimentally in isolated ventricular myocytes are phase-2 EADs, and whether phase-2 EADs are mechanistically linked to PVCs in cardiac tissue remains an unanswered question. In this study, we investigate the genesis of PVCs using computer simulations with eight different ventricular action potential models of various species. Based on our results, we classify PVCs as arising from two distinct mechanisms: repolarization gradient (RG)-induced PVCs and phase-2 EAD-induced PVCs. The RG-induced PVCs are promoted by increasing RG and L-type calcium current and are insensitive to gap junction coupling. EADs are not required for this PVC mechanism. In a paced beat, a single or multiple PVCs can occur depending on the properties of the RG. In contrast, phase-2 EAD-induced PVCs occur only when the RG is small and are suppressed by increasing RG and more sensitive to gap junction coupling. Unlike with RG-induced PVCs, in each paced beat, only a single EAD-induced PVC can occur no matter how many EADs in an action potential. In the wide parameter ranges we explore, RG-induced PVCs can be observed in all models, but the EAD-induced PVCs can only be observed in five of the eight models. The links between these two distinct PVC mechanisms and arrhythmogenesis in animal experiments and clinical settings are discussed.

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Article Info

Publication History

Editor: Eric Sobie.
Accepted: December 8, 2020
Received: July 29, 2020

Identification

DOI: https://doi.org/10.1016/j.bpj.2020.12.001

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