Regional Delayed Afterdepolarizations Induced by Short-Coupled Premature Ventricular Contractions

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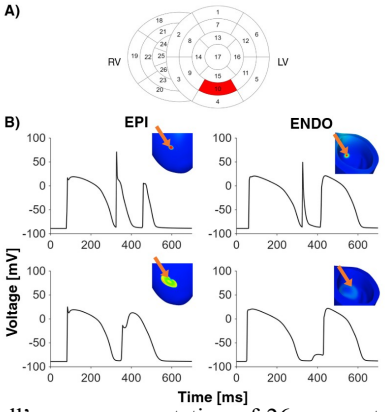
Premature ventricular contractions (PVCs) with short coupling intervals (CI) can induce spatially heterogeneous electrophysiological responses across the ventricles. In this work, we analyzed how short-coupled PVCs alter action potential (AP) morphology across ventricular segments, with particular attention to regional abnormalities in repolarization associated with premature activation.

A patient-specific heart–torso model reconstructed from clinical CT data was used to simulate ventricular electrophysiology. Cellular electrophysiology was described using the human ventricular model of Tomek et al., while organ-scale simulations were performed with a pseudo-bidomain formulation in openCARP. Sinus activation followed by PVCs initiated from 26 standardized ventricular segments based on an extended AHA model, on both endocardial and epicardial surfaces. CIs ranged from 350 to 550 ms. In total, 260 simulations were performed, and AP morphology and repolarization dynamics were analyzed to assess spatial heterogeneity of electrophysiological responses.



Bull's eye representation of 26 segment AHA model (A). Effects of short CI on ventricular Aps (B).

CI effects were spatially heterogeneous, with the largest changes in ECG morphology, local activation time, and AP waveforms occurring in left ventricular (LV) segments for both endocardial and epicardial origins. Segment 10 showed the strongest alterations and was used as a representative example. At short CI (350 ms), APs near the stimulation site displayed prolonged repolarization, and nearby tissue exhibited delayed secondary depolarization consistent with delayed afterdepolarizations (DAD), as shown in the figure. Similar DADs were observed in other LV regions, particularly near the end of sinus depolarization.

These results suggest that short-coupled PVCs interact with spatially heterogeneous refractoriness, promoting localized electrical heterogeneity and conditions associated with DAD emergence, which may contribute to triggered activity and arrhythmogenic substrates.