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## Title: Model Validation of Ventricular Repolarization Dispersion Assessment by QRST Integral Maps

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### Abstract

Elevated Repolarization Dispersion (RD) of the ventricular myocardium is the substrate of malignant arrhythmias. The actiology of pathological RD can be traced back to the deterioration of gap-junction connection of cells. Pathological RD is causally reflected in the body surface QRST integral maps, yielding non-dipolar body surface QRST integral distributions. In our application non-dipolarity was expressed both by QRST integral maps and non-dipolarity indices (NDIs). In our simulation a five-layer numerical ventricular model was used consisting of elementary cubic volumes with programmable action potential durations (APDs) and conduction velocities (CVs). In the beat-to-beat simulations normal or pathological transmural gradients (TGs) were characterized by their  $APD_{epi} - APD_{endo}$  values. Similarly, CV values were expressed regionally by the propagation distance in a modelling time unit (mdu/mtu). Finally, a realistically shaped inhomogeneous torso model was used to compute the QRST integral maps and NDIs beat-to-beat from the source terms. Our simulations imitated the optically measured APD and CV data distributions of normal and arrhythmogenic human hearts. We concluded that NDI was primarily affected by the change of TG values. In normal hearts TG was -15 mtu, throughout the left ventricle yielding an NDI of 0.16. By systematically increasing TG (i.e. changing APD profile) in the apical part of the ventricular model from -15 mtu up to 0 mtu in five steps, NDI increased from 0.16 up to 0.76. The impact of CV modulation was minor. The simulation justified the suitability and noise immunity of beat-to-beat QRST integrals and NDI plots for temporal RD characterization.

Key Words: Repolarization dispersion, QRST integral maps, Non-dipolarity index, Action potential duration, Conduction velocity

### Biography

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