Evaluation of a Method for Quantification of Restitution Dispersion from the Surface ECG

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Dynamic action potential duration (APD) restitution (APDR), expressing the relationship between APD and heart rate (HR) at different steady-state HR levels, presents spatial variations due to electrophysiological heterogeneities in the heart. Clinical studies have suggested that dispersion of APDR in the ventricle may act as a potent arrhythmogenic substrate. In this study, we evaluated a method aimed at quantifying APDR slope dispersion from the surface electrocardiogram (ECG) by computing rate-normalized differences in the T peak to T end (Tpe) interval: \( d_{\text{APDR}} = \Delta \text{Tpe}/\Delta \text{RR} \).

Cardiac propagation was simulated in a 2D tissue preparation representing a ventricular slice of 7.5 cm from base to apex and 1 cm from endocardium to epicardium. The ten Tusscher 2006 model was used to represent the human ventricular cell membrane. APDRs were derived from the 2D simulation to eventually compute the APDR slope dispersion, and pseudo-ECGs were calculated to compute the simulated \( d_{\text{APDR}} \) indices. These simulated \( d_{\text{APDR}} \) values were compared with values obtained from recordings of a set of healthy subjects in which pronounced HR changes were induced by a tilt-test protocol.

We found that \( d_{\text{APDR}} \) values in our simulations were in all cases within the range of clinical values measured from tilt-test recordings (mean ± std of the differences being: -0.0074 ± 0.0141 ms/ms). We then used the validated tissue model to relate the \( d_{\text{APDR}} \) index from the derived pseudo-ECG to measurements of APDR slope dispersion, and we found that the mean error relative to the slope range is 3%, being 8% the maximum and 0.02% the minimum.

We conclude that the proposed \( d_{\text{APDR}} \) index provides valuable estimates of APDR dispersion with the advantage of being able to be measured non-invasively from the surface ECG.