Method for Incorporating Changes in Extracellular Volume and Myocyte Size into Cardiac Bidomain Equations

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Aim: Extracellular volume (ECV) determined by magnetic resonance imaging (MRI) is used in clinical practice for describing changes in the extracellular matrix caused by diseases and surgical treatments, and is linked to cell size. The objective of this study is to develop a robust approach for integrating changes in ECV and cell size into the cardiac bidomain equations.

Methods: We developed a simple method for scaling conduction velocity (CV) in cardiac tissue as a function of myocyte radius (r) and ECV. Simulations of apical pacing were performed in a computational model of the epicardial surface from the human ventricles under control (r=10.6 μm) and hypertrophic conditions (r=15.45 μm), and for three different ECV levels corresponding to the range of values reported for patients with healthy and diseased hearts: 21.5, 25 and 30%. Ventricular activation time was calculated as the difference between the earliest (apex) and latest (base) activation times.

Results: The results from the apical pacing simulations show that increases in ECV shortened total activation time, under both control and hypertrophic conditions (control: 200, 189 and 178 ms; hypertrophy: 162, 154 and 154 ms; values for ECV of 21.5, 25 and 30, respectively), indicating faster CV with increasing ECV. An increase in r from the control to hypertrophic condition also shortened total activation time on average by 23%.

Conclusion: These findings indicate that changes in both ECV and cell radius noticeably alter ventricular conduction. Future work will expand this method to include diffuse fibrosis for matching QRS durations in patient-specific hearts with normal and hypertrophic geometries.