Numerical Investigation of effect of Myocardium Visco-elasticity and Inertia on Left Ventricle Cardiac Cycle

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Computational models of cardiac function are gradually translating towards clinical use as a noninvasive mean of cardiac treatment using cellular, tissue and organ level multiscale simulations. Recent experimental findings suggest that myocardium tissue of human ventricles behaves as an orthotropic viscoelastic material. Besides dependence on material properties, the strains occurring during heart's mechanical cycle are likely to have dependence on the inertial effects as well. Considering the effect of mechanical tissue behavior over heart's electrical activity and vice versa, we performed fully coupled simulations of complete cardiac cycle for quasistatic, dynamic elastic and viscoelastic formulations using a near physiological finite element model of left ventricle (LV) with Holzapfel-Ogden orthotropic constitutive law fitted to human LV movement (See Figure 1). The numerical results obtained from these simulations are used to quantify effect of inertial and viscoelastic terms over electrophysiology through mechanoelectrical feedback (MEF) and stretch activated currents (SAC). Figure 2 shows a comparison of activation times for elastic and viscoelastic models with benchmark electrophysiology results. These results show dependence of conduction velocity as well as action potential duration (APD) on tissue viscoelasticity and inertial effects. Our dynamic elastic LV model (with inertial effects) exhibits 8% increase in conduction velocity as compared to the dynamic viscoelastic LV formulation and 2.5% increase as compared to the quasistatic formulation. APD duration is found to be reduced causing an increase in refractory period depending on the magnitude and timing of stretch which is also related to tissue viscoelastic properties. These results can help to quantify effect of tissue viscoelastic properties and inertia on mechanoelectric currents, cardiac electrophysiology and left ventricle function thereby contribute towards translation of cardiac computational models to clinical use.

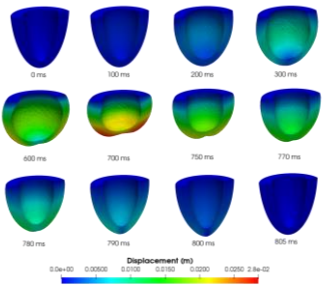

Fig 1. Cardiac cycle simulation using fully coupled viscoelastic model

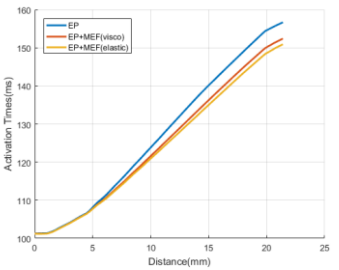

Fig 2. Comparison of activation times