

Physiological and Pathological Cardiac Motion Generation Using a Soft Robotic Approach

Background: Anatomically and physiologically accurate *in vitro* bench-top models of the heart are critical for rapid and effective cardiac device design. During the contraction phase of the cardiac cycle the apex of the left ventricle twists anti-clockwise while the base of the heart has a net clockwise rotation. The resultant complex motion is left ventricular (LV) twist. Cardiac wall motion in the majority of bench-top simulators is achieved through the use of pumps to passively drive flow in elastomeric cardiac models but most do not simulate LV twist. An ideal bench-top cardiac simulator would mimic the soft material properties and active contractile motion of the natural heart tissue and be capable of replicating physiological and pathological motions.

Method: A finite-element (FE) approach was proposed for simulating biologically-inspired arrangements of myocardial fiber-like contractile elements in soft elastomeric matrices using Abaqus® software. We first validated the modeling approach in 2D and then demonstrated a 3D simulation that can generate similar twisting motions to that of the heart. Based on this simulation, we fabricated a soft cardiac simulator with active pneumatic air muscles (PAMs) cast in a low durometer silicone (Ecoflex 00-30, Smooth On Inc.) with a multi-step molding process. Rotation of the LV was measured with electromagnetic trackers (3D Guidance TrakSTAR, Ascension). Subsequently, pathological motion was simulated by deactivating contractile elements in the FE and physical model in order to represent the clinical scenario of a transmural infarct in which muscles are rendered non-contractile or akinetic.

Results: The FE model predicts horizontal and vertical strain with an accuracy of 84% and 87% for single and multiple actuator test specimens respectively. The 3D FE model predicts an apical rotation of $7.78^{\circ} \pm 0.55^{\circ}$ with LV supported at the base, with experimental measurements agreeing closely at $7.89^{\circ} \pm 0.59^{\circ}$. These values are within clinical values of $6.8^{\circ} \pm 2.5^{\circ}$ as reported in the literature [1]. A key feature of the approach is the ability to selectively deactivate active elements in both the simulation and our experimental model. Apical rotation decreased predictably as PAMs were sequentially de-activated.

Conclusion: We have demonstrated a finite-element approach that predicts physiological motion for a fully soft structure with embedded contractile elements. We have also fabricated a physical model whose motion matches simulation results and clinical data with applications as an *in vitro* bench-top cardiac simulator for meaningful device evaluation. Furthermore, we have demonstrated the ability to generate pathological-like motion with our simulations and experiments by sequentially de-activating selected PAMs - a key feature not present in other silicone models. Using this simulation, an increased understanding of akinetic motion can be achieved.

[1] E. Nagel et al, *European heart journal* 2000, 21, 582–9