Physiological and Pathological Cardiac Motion Generation Using a Soft Robotic Approach
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Overview
Nature has abundant examples of soft muscular systems; examples of these in the human body are the stomach, diaphragm and heart. Replication of these motions with traditional robotic systems is challenging, and involves complex mechanisms and many actuators. We took inspiration from biology to design, model and fabricate an active cardiac simulator using soft robotics (Figure 1).

![Figure 1: Inspiration, concept and realization of bioinspired soft actuated material for physiological motion generation.](image)

Method
-A finite-element (FE) approach was proposed for simulating biologically-inspired arrangements of myocardial fiber-like contractile elements in soft elastomeric matrices
-The FE approach was validated in 2D and a 3D simulation then demonstrated that ventricular “twist” could be achieved
-Based on this simulation, a soft cardiac simulator with active pneumatic air muscles (PAMs) was cast in a low durometer silicone using a multi-step molding process (Figure 2).
-Rotation of the LV was measured with electromagnetic trackers
-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.

![Figure 2: (a) 2D specimen manufacture b) Actuator characterization c) A multi-component reconfigurable mold d) Mold with Core 1 for casting outer shell e) 1st pour of Ecoflex 00-30 created outer shell with alignment tabs for PAMs f) With the outer Ecoflex shell remaining in mold, actuators were inserted and aligned with molded alignment features.](image)

Results
The FE model predicts an apical rotation of 7.78°±0.55° with LV supported at the base, with experimental measurements agreeing at 7.89°±0.59° and both are within clinical values 6.8°±2.5° (Fig 3).

PAMs could be selectively deactivated in both the simulation and our experimental model. Apical rotation decreased predictably as PAMs were sequentially de-activated (Fig 4).

![Figure 3: (a) Heart with opposing rotation at apex and base b) Sub-epicardial and sub-endocardial fibres are arranged in opposing helices c) Physical prototype d) Mesh showing deformation of corresponding pressures e) Displacement contour plot f) Apical rotation compared to clinical values g) Apical and basal rotation.](image)

![Figure 4: (a) FE model showing sequential deactivation of PAMs (all at 20ps). Displacement contour plot for each case at 20ps viewed from anterior view (b) and apex (c), respectively d) Physical prototype at 20ps with 6, 1, 2 and 3 muscles deactivated (shown in red). e) Total rotation for FE model and experimental showing a decrease in rotation of markers 1 and 2 that lie in the “akinetic region”.](image)

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. Using this simulation, an increased understanding of akinetic motion can be achieved.

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