The Cardiac Biomechanics Group focuses on the interactions between mechanics, function, and growth and remodeling in the heart. The mechanical properties of normal and diseased myocardium are important determinants of overall heart function. These mechanical properties change during growth, remodeling or disease, often in part as a response to changes in the mechanical environment. Our group studies this interplay between mechanical environment, tissue response, and heart function, not only to better understand the basis for heart disease but also to identify new opportunities to intervene.

“Innovation focused on better diagnosis and treatment for heart disease.”
Mechanical Properties of Healing Myocardial Infarcts
Following a myocardial infarction, dead muscle is gradually replaced by scar tissue. The mechanical properties of that scar tissue are a critical determinant of left ventricular function and subsequent remodeling. Our group studies the structure and mechanical properties of the healing scar, as well as the relationship between scar mechanics and heart function. We found that scar structure and mechanics are very different in different animal models and in different regions of the heart, and are working to understand how these differences arise. We are also designing and testing new therapies that modify mechanical properties of a healing infarct to improve ventricular function and delay or prevent heart failure.

Mechanical Environment and Wound Healing
The structure and mechanical properties that develop in a healing scar depend on the mechanical environment during healing, in the heart as well as in skin and other tissues. We use computational models and engineered tissues (collagen and fibrin gels) to probe how mechanical conditions, such as direction, magnitude, and frequency of stretch, affect fibroblast migration, alignment, and collagen deposition. In collaboration with the Saucerman lab, we are developing and validating models of the intracellular and extracellular signaling cascades that underlie these responses, in order to better design novel interventions to control collagen content and alignment in mechanically loaded tissues.

Mechanics of the Left Atrium
Atrial fibrillation (AF) is the most common heart rhythm disorder, affecting 2.5 million Americans; patients with AF have an increased risk of stroke and death. We work with cardiologists and radiologists here at UVa to understand how left atrial mechanical function changes during AF and how we can limit or reverse these changes through various treatments. We monitor atrial structure and function in patients using hemodynamic measurements and cardiac magnetic resonance imaging (CMR). We develop MATLAB-based surface-fitting algorithms to quantify regional wall motion from the CMR images. We have shown that patients with AF have increased regional heterogeneity in atrial mechanics, and are now exploring how atrial mechanical function responds to different treatments commonly used in these patients.

Mechanical Environment and Cardiac Hypertrophy
Hypertension, valve disease, myocardial infarction, and even pregnancy cause cardiac hypertrophy, defined as an increase in cardiomyocyte mass. The risk of progression to heart failure in these conditions, however, depends primarily on changes in myocyte shape. In patients with pressure overload (PO) states such as chronic hypertension, individual cardiomyocytes and the left ventricular wall become thicker, and heart failure typically develops only after decades. In volume overload (VO) states such as mitral regurgitation, myocytes become longer, the left ventricle dilates, and heart failure develops much faster. Our lab uses a combination of multiscale models and experiments to understand this regulation and develop predictive models of hypertrophy following myocardial infarction, reverse remodeling in response to cardiac resynchronization therapy (CRT), and heart growth in children with congenital heart disease.

RECENT RESEARCH DEVELOPMENTS
• Determined that anisotropic reinforcement is a promising new approach to improving left ventricular function after a large myocardial infarction.
• Showed that mechanical stretch regulates collagen fiber alignment in healing infarcts in the heart, explaining a wide range of apparently contradictory observations in different animal models.
• Developed an agent-based model of infarct healing that accounted for the combined influence of mechanical, structural and chemical guidance cues on scar formation in the heart.

RECENT GRANTS
• U.S. National Heart, Lung, and Blood Institute – Computational Modeling of Scar Formation After Myocardial Infarction
• U.S. National Heart, Lung, and Blood Institute – Multiscale Models of Cardiac Growth, Remodeling, and Myocardial Infarction