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AN EXAMINATION OF STRESS CONCENTRATIONS DUE TO MYOCARDIAL INFARCTION IN THE WALL OF THE HUMAN LEFT VENTRICLE

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INTRODUCTION

The American Heart Association estimates that about 790,000 people suffer from heart attack annually in the United States alone. During a heart attack or myocardial infarction, blood supply is cut off to portions of the heart, starving the tissue of oxygen, and often resulting in cell necrosis. Myocardiocytes, the primary cell type in heart muscle, have limited regenerative potential. Although resident stem cell populations exist in the heart, ischemia/reperfusion injuries sustained from an infarction limit regrowth of functional cardiac muscle. Instead, remodeling of damaged heart muscle often results in the formation of scar tissue by fibroblasts. These fibroblasts not only fail to function as contractile elements but also secrete fibrous matrices that possess very different material properties than the surrounding healthy muscle tissue. Both the loss of function and the differing material properties may have a significant impact on the heart as time passes, influencing remolding, function, and cell growth.

One procedure proposed to reduce the adverse effects of heart attack is cell therapy. In this procedure, stem or progenitor cells are introduced directly into the damaged tissue either through the bloodstream or by direct injection. The newly introduced cells should assist in regrowth/repair of the damaged myocardium; however, recent studies suggest that the microenvironment of damaged myocardium is not conducive to stem/progenitor repair. To adequately promote repair through cell therapy, the damaged tissue should be similar in nature to a healthy cardiac environment - supplying adequate mechanical loading, electrical stimulation, and other "environmental" factors.

In this study, we investigate the material properties of the infarcted zone. To date, very little research has focused on understanding how these material properties affect stress in the wall of the heart. The purpose of this research is to determine how different values of the elastic modulus within the infarcted zone affect the stress levels within and around the infarcted zone. This research will enable us to better understand how abnormal stresses may affect the heart during recovery, or influence stem or progenitor cell-mediated repair.

METHODS

In order to determine how stresses in the damaged heart are distributed, a three-dimensional (3D) model of the human left ventricle was created using the modeling software CREO 2.0. For this model, the ventricle was assumed to be a truncated ellipsoid with a wall thickness of 10mm, a total length of 70mm, and a major diameter of 50mm. The infarcted zone was defined as a half circle section of the ventricle with a radius of 5mm located 40mm from the bottom of the ventricle. To reduce solve time, symmetry was used by defining only a quarter section of the geometry (figure 1).

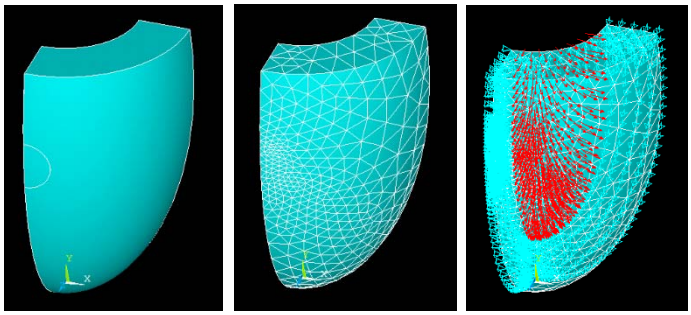

Figure 1: Showing volumes (left), elements (center), and pressure and displacement loads (right).

The 3D model was then imported, meshed, and solved in the ANSYS 17.2 ADPL environment for finite element analysis. The apex

of the section was constrained in the Y direction to represent how the ventricle would naturally be constrained by the attached atrium. To implement boundary conditions resulting from symmetry, the X=0 wall was constrained in the Z direction and the Z=0 wall was constrained in the X direction. A Tet-10 element was chosen for meshing. This is a tetrahedral element with 10 nodes, each node has three degrees-of-freedom. The ventricular pressure was set at 140 mm Hg and the pericardial pressure was considered to be negligible.

The elastic modulus of both the infarcted zone and the surrounding healthy tissue was assumed to be linear, homogeneous, and isotropic. The elastic modulus of the healthy heart tissue was set at 5.88 kPa [1]. Several different elastic moduli for the infarcted zone were evaluated (160 kPa, 40 kPa, 10 kPa, 2 kPa, 0.5 kPa, and 0.125 kPa). Both tissue types were considered incompressible and assigned a Poisson's ratio of 0.49. Convergence analysis was performed to check the accuracy of the results of the finite element model.

RESULTS

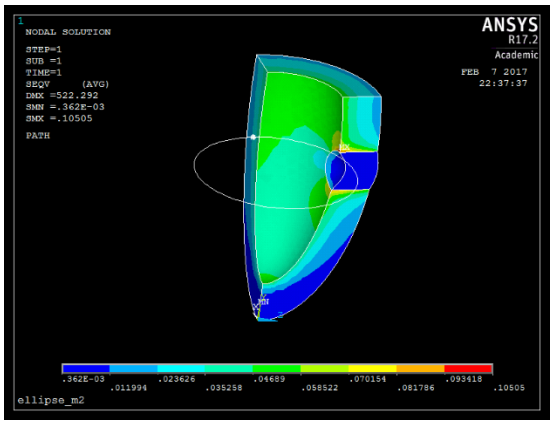

Figure 2: Element von Mises stress contour plot with circular path ($E_{infarct}$=0.125kPa).

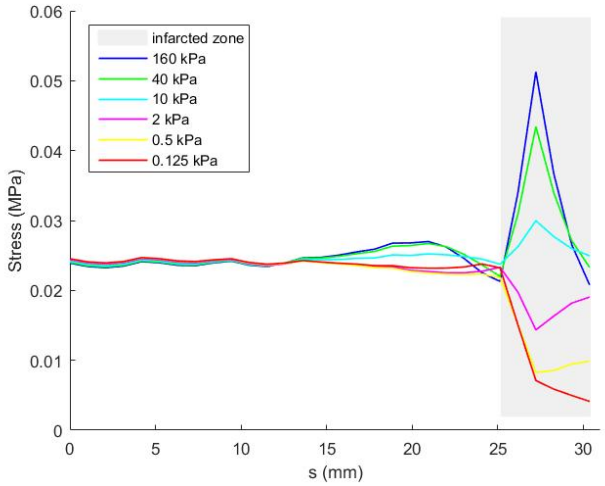

Figure 3: Von Mises stress along the circular path where s is the distance from the XY plane.

To evaluate stresses, a circular path was defined parallel to the XZ plane with the center located at Y=40mm, X=0mm, and Z=0mm and a radius of 20mm (figure 2). The model was solved multiple times, each with a different infarcted zone modulus. The von Mises stress for each different modulus was mapped onto the path and is shown in figure 3.

The results indicate that ventricular wall stress was affected by regional differences in material properties. For infarcted zones with lower elastic modulus than the surrounding tissue, lower stresses were observed within the infarcted zone. Conversely, infarcted zones with higher elastic modulus showed exacerbated stresses, especially along the border between the infarcted and healthy tissue. As the material properties move further away from the healthy tissue properties, the effect intensified. An additional observation was that elements closer to the interior surface had a higher stress than those located on the outer surface of the ventricle.

DISCUSSION

The elevated stresses seen in infarcted zones with a higher elastic modulus are likely due to added shearing, compressive, and tensile forces, which develop when the more deformable healthy tissue stretches under pressure next to the less deformable infarcted tissue. This happens all around the boundary of the infarcted zone. Towards the center of the infarcted area, these added stresses are dissipated by interfacing elements with the same material properties. In infarcted zones with reduced elastic modulus, stresses decrease through the infarcted zone for precisely the same reason. In these cases, the surrounding material is stiffer than the infarcted zone which produces a protective effect similar to stress shielding.

While many sources disagree on the magnitude of the elastic modulus of both the infarcted and non-infarcted tissues, they do agree that infarcted tissue is stiffer than healthy tissue after fibroblasts have moved in [2]. Based on our results, it is easy to see how this stiffness may affect the heart in many ways. The resulting increase in wall stresses could be a motivating factor in cardiac remolding following a myocardial infarction. Increased stresses may also affect the efficacy of cell therapy. It is known that the mechanical environment experienced by stem cells is a major determining factor in their differentiation. Increased stresses may provide a poor environment for implanted cells.

The results indicate that a softer infarcted region, achieved through early treatment or pharmacological methods, may increase the likelihood of successful cell therapy. The use of biomaterial scaffolds may also change cell therapy outcomes. Many biomaterials, including keratin, can be altered to exhibit different material properties [3]. These biomaterials may then be injected and used to reduce stresses on stem cells while also providing a scaffold on which cells can differentiate and mature.

More research is needed to fully understand how these stresses affect the heart on a long term basis. Important steps moving forward include determining an accurate range of material properties for both healthy and infarcted cardiac tissue and; taking into account the time elapsed since infarction. This may change treatment timelines and help us to understand remodeling. Future models should include various infarcted zone sizes and shapes, nonlinear and anisotropic material properties, and the inclusion of a biomaterial zone.

ACKNOWLEDGEMENTS

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