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SIMULATION OF SHORT-TERM ADAPTATION PROCESSES IN THE INFARCTED HEART

P. Saez[1], J.F. Rodriguez[1], E. Kuhl[2]

[1] Department of Mechanical engineering, University of Zaragoza. psaez@unizar.es
[2] Department of Mechanical Engineering, Stanford University. ekuhl@stanford.edu

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Myocardial Infarction (MI) is the event at which heart tissue does not received the needed blood flow [1]. This is usually caused by the occlusion of a coronary artery. Depriving the tissue from blood flow cause chemical and mechanical effects in the heart. Cardiomyocyte death, inflammation, thinning, and ventricular expansion of the infarct zone (IZ) are the main consequences in the early stages, from hours to a week. Hypertrophy of the non infarcted zone (NIZ) and collagen scar formation in the infarcted zone (IZ) are the main auto-regulatory mechanisms in the late stages. These adaptive processes lead to the modification of the pressure and volume curves which are important clinical indicators for diagnosis.

The heart is composed of two major component in terms of its mechanical behavior, cardiomyocytes and collagen. Cardiomyocytes exert tension by contraction, which causes the heart to pump out blood. Collagen types I and III are the most important types of collagen in the myocardium. Their degradation and deposition is mainly influenced by growth factors like TGF-β and the MMP/TIMP ratio.

Here we model the short-term events in the ischemic heart. We first define the ischemic process by imposing the decrease of oxygen supply and compute the diffusion of cell death in cardiac tissue (Fig. 1a). Cell death induces a thinning in the ventricle, which we model by a multiplicative decomposition [2, 3] of the deformation gradient into an elastic and a growth part. The growth part is defined as $\mathbf{F}_{\text{g}} = \mathbf{I} + [\,\vartheta - 1\,]\,\mathbf{n}_{\text{r}} \otimes \mathbf{n}_{\text{r}}$, where ϑ is the scalar-valued growth multiplier that defines the level of growth and $\mathbf{n}_{\text{r}}$ characterizes the radial direction [3]. After the ischemic process, the damaged cells undergo mechanotransduction pathways that eventually lead to the formation of collagen fibers. This induces a variation of the collagen density ρ_{col}, which we define as follows,

$$\dot{\rho}_{\text{col}} = \mathcal{R} \qquad \text{with} \qquad \mathcal{R} = \left[\frac{\rho_{\text{col}}}{\rho^*_{\text{col}}}\right]^{-m} \rho_{\text{MMP}} - \rho^*_{\text{MMP}}. \tag{1}$$

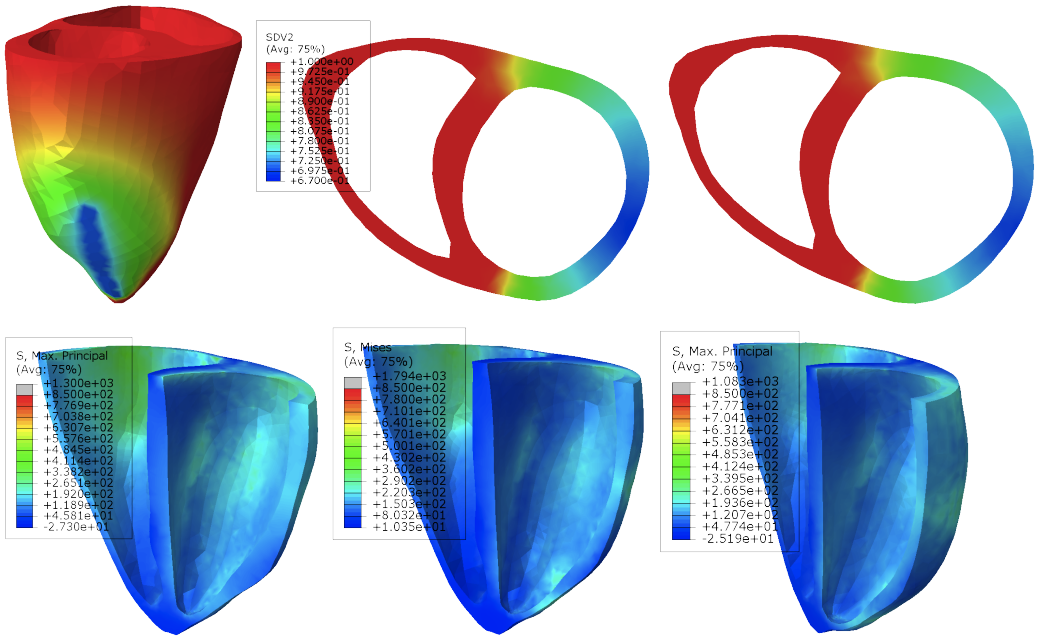

Figure 1: Ischemic condition (a) and the subsequent thinning (b) and expansion of the infarct heart. Mechanical behavior of the healthy (d), thinned (e) and expanded heart (f).

Here $\dot{\rho}_{col}$ denotes the material time derivative of ρ_{col}, ρ^*_{col} is the initial density, and the exponent m typically varies between two and three.

The result of this collagen degradation is the expansion of the ventricle that we simulate here as $\mathbf{F}_{\rm g} = \mathbf{I} + [\,\vartheta - 1\,]\,\mathbf{n}_\theta \otimes \mathbf{n}_\theta$, which models the expansion of the ventricular wall in the circumferential direction $\mathbf{n}_\theta$. The results show a decrease of the maximum principal stress due to the decrease in collagen content, initiating the expansion of the left ventricle. This is in accordance with experimental results found in literature [4].

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