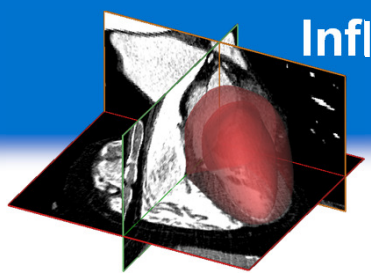

Influence of including infarcted material to a left ventricular contraction simulation

STUDENT PROJECT
Computational Bioengineering laboratory SS2013

Severin Hämmerl, Markus Horvath, Dionis Minev, Christoph Moosbauer

Institute for Computational Mechanics, Technische Universität München, Germany

Introduction

Infarction of the heart is the most leading cause of death in the developed world. Infarction of muscular tissue develops, if the coronary artery does not provide enough blood. This ischemia occurs mainly in the endocardial part of the muscle and can locally lead to muscular decease. Then these parts are not able to support the ventricular contraction any longer. For the global heart system this could lead to reduced blood volume output or ventricular fibrillation. For this project we want to focus on the effects on the dynamic of the contraction.

Model generation

To evaluate these effects, 3 different ischemic models are compared to a healthy reference:

- P1R10: Small infarcted area on side wall, position 1, radius=10mm
- P1R20: Large infarcted area on side wall, position 1, radius=20mm
- P2R20: Large infarcted area on apex, position 2, radius=20mm

Fig. 1 depict a characteristic step in the model generation process. The workflow will be described more precisely in the following.

Geometry

In order to obtain a three-dimensional model for Finite Element (FE) analysis, following steps have been conducted:

- Segmentation of the geometry from patient-specific CT-data using Mimics ®
- Reasonable smoothening of the geometry with 3-matics ®
- Generation of a pseudo valve

Remark: The applied pseudo valve allows effective monitoring of volume changes in the lumen of the heart by applying the Gaussian divergence theorem, by which an evaluation of the effect of an infarct on the cardiac output is enabled

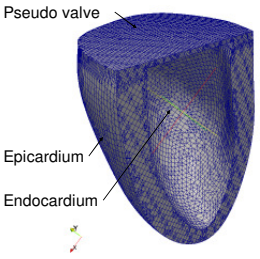

Figure 1 Meshing of a cut through the non-infarcted reference model of the left ventricle along the long axis.

Discretization and volume classification

The preparation of the geometric model for a FE analysis contains following steps:

- Mesh generation in Harpoon ® (unstructured hex-dominant elements with approximately $5.0 \cdot 10^5$ degrees of freedom)
- Classification of different volumes (myocardium, spherical infarcted regions, pseudo valve)
- Infarcted material is represented as a sphere around a center point on the endocardium at one of the main bifurcations of the coronary arteries (see fig. 2)

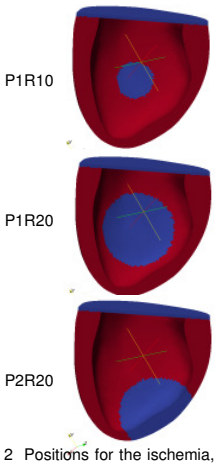

Figure 2 Positions for the ischemia, cut through left ventricle

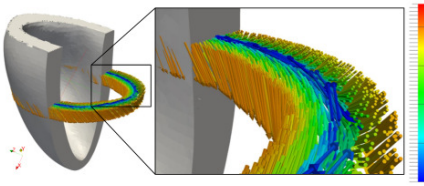

Figure 3 Distribution and directions of fibers

Fibers in the tissue

The dynamics of the cardiovascular tissue is dominated by a fiber structure, which has to be taken into account. This was done by solving a scalar transport equation, following a rule-based method according to Streeter[1], see fig. 3.

Computational setup

- Dynamic computation with Rayleigh damping at a timestep size $\Delta t = 0.002s$ in BACI
- Load: cardiac pulse (see fig. 4) at a frequency of $75Hz$ (in total $T = 0.8s$)
- Apical activation: propagation from apex to atrial area
- Hyperelastic material behaviour, see Table 1

Table 1 Selected material model parameters

Region	Material model	Active	Characteristic parameters	
Contracting	CoupAnisoNeoHooke_ActiveStress	Yes	$\sigma = 2.0 \cdot 10^5$	
Myocardium	IsoMooneyRivlin	No	$C_1 = 2.0 \cdot 10^4$	$C_2 = 40$
	VolPenalty	No	$\varepsilon = 1.0 \cdot 10^7$	$\gamma = 1.0$
Ischemic	IsoMooneyRivlin	No	$C_1 = 2.0 \cdot 10^4$	$C_2 = 40$
Myocardium	VolPenalty	No	$\varepsilon = 1.0 \cdot 10^7$	$\gamma = 1.0$
Pseudo valve	IsoMooneyRivlin	No	$C_1 = 2.0 \cdot 10^6$	$C_2 = 0.0$
	VolPenalty	No	$\varepsilon = 1.0 \cdot 10^{-13}$	$\gamma = 1.0$

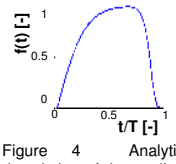

Figure 4 Analytic description of the applied cardiac contraction

Simulation results

All postprocessing was done in Paraview

Von Mises stresses

Von Mises stress distributions were calculated using main stresses $\sigma_{I,II,III}$ (see fig. 5):

$$\sigma_v = \sqrt{\frac{1}{2}[(\sigma_I - \sigma_{II})^2 + (\sigma_{II} - \sigma_{III})^2 + (\sigma_{III} - \sigma_I)^2]}$$

- The contraction of the muscle tissue starts from the apex towards the pseudo valve
- It becomes clear how the ischemic areas of all sizes, in each location do not contract and show no contribution to the change of volume

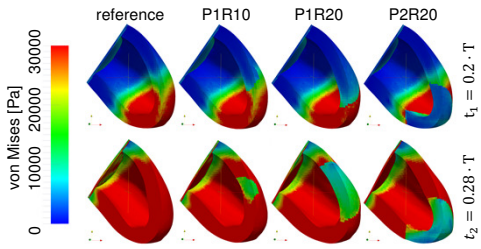

Figure 5 Simulated von Mises stress distributions of the left ventrical at two time steps during contraction. Three different setups of ischemic tissue due to infarcted areas are compared.

Displacements

Fig. 6 shows magnitudes of displacements:

- Almost no displacements of infarcted material (column 2 to 4)
- Rotation at $t = t_2$ (reference) does not occur when an ischemia is assumed
- Small influence of size of infarct on deformation (column 2, 3), but magnitudes decrease
- Deformation starts delayed and maximum displace-ment is near the pseudo valve when infarct is located at apex (column 4)

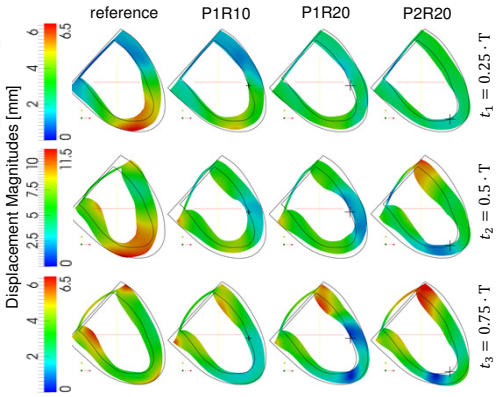

Figure 6 Comparison of simulated magnitudes of displacements for the different ischemic setups at t_i (contracting phase), t_2 (maximum contraction), t_3 (partially relaxed muscle state). Outline and crosses: contour at $t = 0.0s$ and positions of the ischemic centerpoint

Volume change

- Volumes of all four heart models decrease during the first half of a cycle which physiologically were to cause an outflow of blood equal to the volume change (fig. 7)
- Minimal volume (and thus the integrated maximum outflow) is reached at 0.4 sec
- Infarcted heart models' volumes change less compared to the reference model
- The bigger the radius of the infarcted area, the greater is the lack of volume change
- The infarct at the apex influences the volume contraction less, but earlier than the infarct closer to the valves

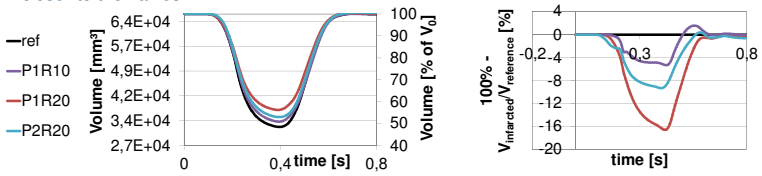

Figure 7 Volume in left ventricle over one cycle and relative developing of the infarcted hearts' volume changes compared to the reference model

Conclusion

All four simulated models show realistic behaviour for a left ventricular contraction cycle with and without an infarcted area. Models P1R10, P1R20 and P2R20 include the infarcted material and show less displacements and lower von Mises stress distributions in the ischemic tissue compared to the reference model. Subsequently, the volume change during the contraction cycle is reduced with increasing amount of infarcted material.

The behaviour of the ischemic material can be observed for each time step. Variations of the infarcted area (from R=10 to R=20) and center point position (P1 compared to P2) influence the contraction cycle more severely, whereby an infarct located in the apex region seems to have relatively less effect on the output and is therefore less critical than a sidewall affection. This might be due to the fact that cross-sectional areas at position 1 are greater than at the apex region (position 2).

Apart from that, the following simplifications limit the validity of the investigations:

A so-called Purkinje fiber system has to be considered for exact representation of the physiological activation process rather than a strongly simplifying apical activation, but its application is a field of ongoing research and therefore beyond the scope of this study. The pseudo valve was added only for the sake of simplified volume monitoring and does not represent the real atrial structure.

Nevertheless, the simulations clearly represent the reduction of cardiac output in the case of infarcted tissue compared to a healthy reference model and the harmful effects of infarcted muscular tissue on the dynamics of a human heart.

[1]Streeter, Daniel D., "Gross morphology and fiber geometry of the heart." *Handbook of physiology* (1979): 61-112