Published in final edited form as:

J Biomech Eng. 2011 April ; 133(4): 044501. doi:10.1115/1.4003438.

Regional Left Ventricular Myocardial Contractility and Stress in a Finite Element Model of Posterobasal Myocardial Infarction

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Abstract

Recently, a non-invasive method for determining regional myocardial contractility, using an animal-specific finite element (FE) model-based optimization, was developed to study sheep with anteroapical infarction [1]. Using the methodology developed in the previous study [1], which incorporates tagged magnetic resonance images (MRI), three-dimensional (3D) myocardial strains, left ventricular (LV) volumes, and LV cardiac catheterization pressures, the regional myocardial contractility and stress distribution of a sheep with posterobasal infarction was investigated. Active material parameters in the noninfarcted borderzone (BZ) myocardium adjacent to the infarct (T_{max_B}), in the myocardium remote from the infarct (T_{max_R}), and in the infarct (T_{max_I}) were estimated by minimizing the errors between FE model-predicted and experimentally measured systolic strains and LV volumes using the previously developed optimization scheme. The optimized T_{max_B} was found to be significantly depressed relative to T_{max_R} , while T_{max_I} was found to be zero. The myofiber stress in the BZ was found to be elevated, relative to the remote region. This could cause further damage to the contracting myocytes, leading to heart failure.

Keywords

finite element modeling; numerical optimization; cardiac mechanics; tagged magnetic resonance imaging

Introduction

Myocardial contractility plays a central role in the heart's ability to pump blood efficiently into the body. When myocardial tissue becomes damaged, due to infarction, it is the job of clinicians to determine to what degree the heart is impaired. A non-invasive method for estimating regional myocardial contractility would be a beneficial tool for cardiologists to assess viable and nonviable tissue. This tool could also be used to evaluate the efficacy of surgical procedures and medical devices for treating infarction-induced heart failure. The

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most important benefit would be to help surgeons decide where to incise and how much of the left ventricular (LV) infarct region is impaired.

Several studies have been conducted on left ventricles with anteroapical aneurysms that employ finite element (FE) simulations, in conjunction with a manually directed pseudo-optimization, to assess the contractile function of myocardium [2, 3]. However, these studies required long computation times for each case that was evaluated. In order to overcome this deficiency, a fully automated nonlinear optimization was developed for estimating regional myocardial contractility that was at least 24 times faster than those previous studies [1, 4].

The goal of the current study is to extend the application of this formal optimization technique to examine myocardial infarction in a different region of the left ventricle. Although the previous study [1] looked at anteroapical LV aneurysm, it was found that this method is robust enough to be applied to other types of infarcts. Of particular interest is the degradation of the posterobasal region of the left ventricle, which is linked to mitral regurgitation and the advancement of heart failure [5]. The methodology described in Sun et al. [1] provided the framework for the work presented here.

Methods

The details of the experimental measurements, model generation, and optimization scheme are presented in Sun et al. [1]. A brief overview of the methodological approach, along with several key differences to the previous work, is presented in the following sections.

Experimental Measurements

A single male adult Dorsett sheep underwent posterobasal myocardial infarction as previously described [6, 7]. At 8 weeks post-myocardial infarction, a series of orthogonal short- and long-axis tagged magnetic resonance (MR) images were acquired, while ventricular pressure was continuously measured [8]. The endocardial and epicardial LV surfaces were contoured from the images, and the systolic tags were segmented for each image slice in order to compute the systolic myocardial strain at mid-wall of the LV (Figs. 1a and 1b) [9, 10]. Unlike the previous application [1], where the aneurysm wall was very thin, strain components were calculated within the infarct region in the current application. This data was used to confirm the location of the infarct due to the appearance of positive strain, which indicates that the myocardium is stretching rather than shortening during systole. This phenomenon can be seen in Fig. 1c, where the infarct strain is positive and borderzone (BZ) strain is elevated, relative to the remote region.

FE Model

An FE model was created based on the LV geometry at early diastole (Fig. 2a). This configuration was selected because the stress is at a minimum in the LV. The remote, BZ, and infarct regions were determined based on several factors. First, the infarct region was determined using the circumferential component of strain calculated from the MR images. In this case, positive circumferential strain was indicative of the infarct region. The geometric dimensions of this region were then confirmed with measurements of the infarct region from the excised heart of the animal. The BZ region was approximated as the transition in wall thickness between remote and infarct regions (Fig. 2b) [11]. Histological studies are currently being conducted on sheep with posterobasal infarcts, which will help characterize the size of the BZ. However, in a clinical setting histological studies will not be possible and thus other non-invasive factors will need to be assessed.

The geometry was meshed with 8-noded trilinear brick elements, for a total of 3940 elements. Each region, remote, BZ and infarct, were assigned different material properties

[1]. Cardiac myofiber angles were assigned to vary transmurally from -37° to 83° (epicardium to endocardium), relative to the circumferential direction, in the remote and BZ regions [12]. At the infarct region, fiber angles were set to 0° in order to use experimentally determined material parameters with respect to this direction [13]. Although the results from Moonly [13] were for an anteroapical infarct, it has been shown in a porcine animal model that the distribution of collagen fibers in a lateral mid-ventricular MI is primarily dominated by the circumferential direction, and is driven by the direction of highest tensile stretching [14, 15].

The constitutive laws for passive and active myocardium have been described previously [16, 17]. Briefly, the passive material response is given by the strain energy function, W , that is transversely isotropic with respect to the local fiber direction,

$$W = \frac{C}{2} \left(\exp^{b_f E_{11}^2 + b_t (E_{22}^2 + E_{33}^2 + E_{23}^2 + E_{32}^2) + b_{fs} (E_{12}^2 + E_{21}^2 + E_{13}^2 + E_{31}^2)} - 1 \right) \quad (1)$$

where C , b_f , b_t and b_{fs} are diastolic myocardial material parameters, E_{11} is strain in fiber direction, E_{22} is cross-fiber in-plane strain, E_{33} is radial strain transverse to the fiber direction, and the rest are shear strains. Systolic contraction was modeled as the sum of the passive stress derived from the strain energy function and an active fiber directional component, T_0 [17], which is a function of time, t , peak intracellular calcium concentration, Ca_0 , sarcomere length, l , and maximum isometric tension achieved at the longest sarcomere length, T_{max} [18],

$$\mathbf{S} = p \mathbf{J} \mathbf{C}^{-1} + 2 \mathbf{J}^{-2/3} \text{Dev} \left(\frac{\partial \tilde{W}}{\partial \mathbf{C}} \right) + T_0 \{t, Ca_0, l, T_{max}\} \quad (2)$$

where $\text{Dev}()$ is the deviatoric operator [1].

The tagged MR images were acquired during systole, which means that only systolic myocardial strains were determined. Therefore, the systolic material parameters in the remote (T_{max_R}), BZ (T_{max_B}), and infarct (T_{max_I}) regions were computed from the optimization. The passive parameter, C , in the remote (C_R) was determined such that the model-predicted end-diastolic LV volume matched the experimentally measured value. The initial search range in the optimization was set between 0 and 800 kPa for T_{max_R} , between 0 and 400 kPa for T_{max_B} , and between 0 and 200 kPa for T_{max_I} . Although calcium concentration can vary in the region around the infarct, the contractility parameter, T_{max} , was used as the single measure of change to contractile capability in the various myocardial regions.

Material Parameter Optimization

The objective function for the optimization was taken to be the mean squared error (MSE), which evaluates the difference between the computed FE and measured experimental results (end-diastolic and end-systolic volumes, and systolic strains). The goal of the optimization is to minimize the MSE. One primary difference between the previous application of the optimization [1] and the current, is the use of strain in the infarct as part of the MSE,

$$\text{MSE} = \sum_{n=1}^N \sum_{\substack{i=1,2,3; \\ j=1,2,3; \\ i \neq 3 \& j \neq 3}} w_n \left(E_{ij,n} - \bar{E}_{ij,n} \right)^2 + w_{VED} \left(\frac{V_{ED} - \bar{V}_{ED}}{\bar{V}_{ED}} \right)^2 + w_{VES} \left(\frac{V_{ES} - \bar{V}_{ES}}{\bar{V}_{ES}} \right)^2 \quad (3)$$

where n is the *in-vivo* strain point, N is the total number of *in-vivo* strain points, $E_{ij,n}$ is the computed FE strain at each strain point, V_{ED} and V_{ES} are the computed FE end-diastolic and end-systolic LV volumes, respectively. The overbar represents experimental *in-vivo* measurements. Although the end-diastolic volume does not affect the outcome of the active parameter optimization, it was included in the objective function to account for all of the constraints involved in matching the predicted outcomes to those that were experimentally measured.

The weights, defined as w_n , w_{VED} , and w_{VES} , were used to balance the importance of each term in the MSE. In the current case, there are 4 times as many experimental strain points in the remote region than in the BZ and infarct. Thus the weights for the BZ and infarct MSE components were set to 4, while the rest were kept equal to 1. Strain in the radial direction was excluded as it cannot be measured with sufficient accuracy in the BZ region with tagged MR images [19, 20].

Results

The measured end-diastolic pressure (6.1 mmHg) was applied to the endocardial wall of the FE model while the passive myocardial stiffness was varied, such that V_{ED} was accurately predicted at 104.9 mL with the measured value being 105 mL. The parameters C_R and C_B were calibrated to be 0.017 kPa, while C_I was defined to be 10 times stiffer at 0.17 kPa. The calibrated end-diastolic state was used as the reference state for the systolic strain calculation, which was computed using a multiplicative decomposition of the deformation gradient, as described in Sun et al. [1].

The minimum MSE of 11.13, consisting of 720 systolic strain and 2 LV volume data points, was reached in 10 iterations. The optimized contractility parameters for this sheep were found to be $T_{\max_R} = 180$ kPa, $T_{\max_B} = 115.4$ kPa, and $T_{\max_I} = 0.0$ kPa. This shows a 36% decrease in contractile function in the BZ compared to the remote region, and confirms that the infarct has no contractile function. Using the measured end-systolic pressure (91.5 mmHg), the volume V_{ES} was predicted to be 67.4 mL, which is only 4.6% higher than the measured value of 64.4 mL.

The predicted systolic strains, using the optimized material parameters, were in generally good agreement with the *in-vivo* measured strains. Of particular interest is the agreement in the circumferential component of strain, because it aligns very closely to the fiber direction at mid-wall. The mean and standard deviation of the circumferential strain, by region, is given in Table 1 and Table 2. It can be seen that the experimental and finite element strains are within one standard deviation of each other, in each region. In fact, the difference is well below one standard deviation in the remote and BZ regions.

The root mean square (RMS) error for the circumferential strain component between the 116 pairs of measured and predicted strains in the remote region was 0.673, in the BZ the RMS error was 0.249 with 18 pairs of strain points, and in the infarct region the RMS was 0.167 with 10 pairs of strain points. An example of the circumferential strain agreement at a mid-

ventricular slice is shown in Fig. 3a. The mean and standard deviation of the experimental strain in this slice is -0.112 ± 0.078 and for the FE strain is -0.118 ± 0.080 , which are in very close agreement. An example of the longitudinal strain in the same mid-ventricular slice is shown in Fig. 3b. It can be seen that the strain is also positive in the infarct region, indicating longitudinal stretching.

The total stress in the myofiber direction at end-systole, as given by Equ. (2), was elevated in the BZ region relative to the remote region.. This is indicated by the peak stresses seen in Fig. 4. The region of the BZ that is most affected corresponds to sector 11 (shown in Fig. 1b). At this sector the circumferential strain is elevated relative to the remote region, which indicates that there is less shortening going on in the muscle fibers at this point (i.e. they are being extended). This stress concentration could potentially lead to further expansion of the infarct and BZ regions, through impairment of the myocytes.

For the optimization of 3 material parameters, 7 experimental points were selected using a D-optimal method for each iteration. The entire optimization process involving 10 iterations required about 3 hours on a Dell Precision T7400 with dual 3.2 GHz Quad Core Xeon processors.

Discussion

After adapting the formal optimization method [1], it was found in this study that T_{max_B} is also substantially depressed compared to T_{max_R} in a posterobasal MI animal model. In addition, with improved computing power, which is now becoming more standard, the simulation time was improved by 50% compared to the previous optimization study [1].

A significant difference between the current study and initial application of the formal optimization method is the ability to compute strain in the infarct region. This is because posterobasal infarcts are thicker than anteroapical LV aneurysms (1-3 mm), and thus can be tagged during MRI. Posterobasal infarcts behave differently during remodeling, as they do not stretch as much as anteroapical infarcts. The optimization method was able to be applied to this type of infarct without any major changes to the methodology. The method should be robust enough to handle several different types of infarcts. An overview of several previous optimization studies is given in Sun et al. [1].

In order to assess the influence of varying BZ fiber angles, a sensitivity study was conducted in which the BZ fiber angles were rotated with a 23° leftward shift, resulting in a distribution of -60° to 60° from epicardium to endocardium. It was found that T_{max_B} decreased by 9% and T_{max_R} increased by 6%, compared to the original optimization. This result still shows a significant depression in BZ contractility relative to the remote. The angles in the sensitivity study correspond to the *in-vivo* dMRI data from Wu et al. [21, 22] concerning patients with an MI in which the percentage of left-handed helical fibers (negative subepicardial fiber angles) increased from the remote zone to the BZ, and the percentage of right-handed helical fibers (positive supendocardial fiber angles) decreased from the remote zone to the BZ. Alternatively, recent *ex-vivo* dMRI data from infarcted rat hearts indicate that BZ helix angles are preserved [23, 24]. Thus the effect of MI on BZ fiber angle change is still unresolved.

The circumferential strain can be used as a means of non-invasively identifying the infarct region. This can be done in concert with delayed enhancement, in order to fully understand the location and depth of the infarct [25]. In many simulations, the contractility in the infarct is assumed to be zero [2, 3, 26]. In the present study, this parameter was allowed to vary during the optimization. The fact that T_{max_I} was determined to be zero confirms this

assumption. It is possible for an infarct not to be fully transmural. In this case the contractility would most likely be non-zero.

Due to the presence of strain in the infarct, the weights were adjusted in the objective function in order to balance the contribution of each region to the MSE. By shifting the weights, it was found that the agreement of the circumferential strain was improved, particularly in the BZ, when the weights were changed from 1 to 4 in the BZ and infarct region. In general, the weights can have a significant effect on the outcome of an optimization [27]. The influence of the weights can be explored through techniques such as the Pareto Frontier [28]. However, the weights were chosen manually in the present work.

There are several limitations in the present study. The interaction with the RV was neglected in the current study, which could explain the prediction of overstretching in the infarct compared to the experimental results. Future studies will include the RV geometry and pressure as part of the model. The quality and resolution of the experimental strain could be improved by utilizing a 3T scanner rather than a 1.5T scanner, which can produce a more precise gradient of strain in the LV wall [29, 30]. Additionally, it is difficult to quantify the steep transition in the wall thickness at the BZ. Thus the BZ region was approximated using a combination of circumferential strain and a clinician's best judgment. With further development, higher quality experimental strain may be able to help identify the BZ region more precisely.

In order to more accurately assess the degree of anisotropy in the passive response of each region in the infarcted LV, the optimization technique will be extended to evaluate diastolic material properties. In future work, this will require the acquisition of tagged MR images during the diastolic phase. Due to the robustness of this tool, the goal is to track changes in myocardial material properties of the same heart as it remodels over time. This will allow for a long term assessment of how various surgical procedures affect the mechanical response of the heart [3, 31-33].

Acknowledgments

This study was supported by NIH grants R01-HL-84431 (Dr. Ratcliffe), R01-HL-63348 (Dr. Ratcliffe), R01-HL-77921 (Dr. Guccione), and R01-HL-86400 (Dr. Guccione). This support is gratefully acknowledged.

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Figure 1.

3D cardiac strain analysis from *in-vivo* tagged MR images. Endocardial and epicardial contours as well as segmented tag-lines were traced from (a) short-axis MR images to create a 3D geometry. (b) Each short axis slice was divided into 12 sectors and a 4D B-spline-based motion tracking technique was applied to the tag-line (dotted lines) deformations in order to calculate the Lagrangian Green's strains in cylindrical coordinates. For each sector of each short axis slice, longitudinal, radial, (c) circumferential and shear strains throughout systole were determined.

Figure 2.

(a) Animal-specific finite element model used in the optimization method. The green elements represent the healthy remote region, the red elements are the borderzone, and the blue elements are the infarcted region. (b) Cross-section view of the animal-specific finite element model near mid-ventricle. It can be seen that the infarcted region is thinner than the surround myocardium. The red borderzone is used to approximate the transition from healthy to diseased tissue.

Figure 3. A comparison of the experimental and finite element (a) circumferential strain and (b) longitudinal strain in a slice near mid-ventricle. On average, there is good agreement between the experimental and numerical strains in the remote, borderzone, and infarct regions.

Figure 4. Stress (kPa) in the myofiber direction at end-systole. The black lines represent the boundaries of the borderzone. The stress is elevated in the borderzone region, which is indicated by the red color contour.

Table 1

Experimental circumferential strain shown by region, determined from tagged MR images.

Remote strain		BZ strain		Infarct strain	
Mean	Std dev	Mean	Std dev	Mean	Std dev
-0.14195	0.034294	-0.08443	0.054901	0.02699	0.028928

Table 2
FE calculated circumferential strain shown by region, determined from the optimization method.

Remote strain		BZ strain		Infarct strain	
Mean	Std dev	Mean	Std dev	Mean	Std dev
-0.13008	0.055723	-0.07166	0.051143	0.04701	0.04701