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First Evidence of Depressed Contractility in the Borderzone of a Human Myocardial Infarction

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Abstract

Background—The temporal progression in extent and severity of regional myofiber contractile dysfunction in normally perfused borderzone (BZ) myocardium adjacent to a myocardial infarction (MI) has been shown to be an important pathophysiologic feature of the adverse remodeling process in large animal models. We sought, for the first time, to document the presence of impaired contractility of the myofibers in the human BZ myocardium.

Methods and Results—A 62 year-old man who suffered a MI in 1985 and had recently had complete revascularization was studied. Myofiber systolic contractile stress developed in the normally perfused borderzone adjacent to the MI ($T_{\max\_B}$) and that developed in regions remote from the MI ($T_{\max\_R}$) were quantified using cardiac catheterization, MRI and mathematical modeling. The resulting finite element model of the patient's beating left ventricle was able to simulate the reduced systolic strains measured using MRI at matching left ventricular pressures and volumes. $T_{\max\_B}$ (73.1 kPa) was found to be greatly reduced relative to $T_{\max\_R}$ (109.5 kPa). These results were found to be independent of assumptions relating to BZ myofiber orientation.

Conclusions—The results of this study document the presence of impaired contractility of the myofibers in the BZ myocardium and support its role in the post MI remodeling process in patients. To fully establish this important conclusion serial evaluations beginning at the time of the index MI will need to be performed in a cohort of patients. The current study supports the importance and demonstrates the feasibility of larger and longer-term studies.

Keywords

Modeling; Magnetic Resonance Imaging; Cardiac Function; Heart Failure; Bioengineering

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Disclosures None.

Introduction

Adverse left ventricular (LV) remodeling after myocardial infarction (MI) is responsible for nearly 70% of heart failure cases. Previous studies in clinically relevant large animal preparations using state-of-the-art MRI tissue tagging and finite element modeling algorithms have demonstrated that a spatially progressive loss of contractile function in perfused myocardium outside the infarct zone is central to the mechanism by which an initially well-tolerated acute myocardial loss progressively leads to chronic symptomatic heart failure [1–4]. These animal studies have established that loss of contractile function occurs initially and is most severe in the perfused borderzone (BZ) adjacent to the infarct. In this report we present the first use of a fully validated tissue tagging and analytic modeling technique to assess regional contractile function in the remodeled human heart.

An important feature of this investigation is the combined use of three-dimensional complimentary spatial modulation of magnetization (3D CSPAMM) for measuring regional myocardial deformation and a realistic mathematical (finite element) model of the infarcted LV for computing regional myocardial force development. The technique has been previously validated by direct *ex-vivo* active force measurements in skinned fiber preparations [5]. Direct measurement of such forces in an intact LV has been unreliable in animals and is not possible in patients.

Materials and Methods

This study was approved by the Committee on Human Research at the University of California at San Francisco Medical Center and the Institutional Review Board of the San Francisco VA Medical Center. Informed consent for magnetic resonance imaging was obtained from the patient who had no contraindication to the procedure.

Experimental Measurements

A 62 year old male suffered MI in 1985. In 2010, he underwent coronary artery bypass grafting (CABG). Prior to CABG persantine thallium stress testing demonstrated reversible defects that were all amenable to surgical revascularization. The patient underwent 3D CSPAMM (Figure 1a) six weeks after CABG at which time there was no evidence of ongoing ischemia. In addition, the patient underwent MR delayed hyperenhancement, in order to precisely delineate the infarct region. Left ventricular pressure was continuously measured via cardiac catheterization. The endocardial and epicardial LV surfaces were contoured from the MR images, and the systolic tags were segmented in order to compute the systolic myocardial strain at mid-wall of the LV [5, 6].

Finite Element Model

The images used to build the model were from the last MRI time step before mitral valve opening. This time point was during the latter part of isovolumic relaxation, and was selected because the stress is at a minimum in the LV. A customized version of the MRI post-processing software, FindTags (Laboratory of Cardiac Energetics, National Institutes of Health, Bethesda, MD) was used to contour the endocardial and epicardial LV surfaces. A finite element model was then projected to the LV wall surfaces (Truegrid, XYZ Scientific Applications, Inc., Livermore, CA). The geometry was meshed with 8-noded trilinear brick elements, for a total of 2400 elements, using a single integration point for computational efficiency. The transmural mesh density was adjusted until the ventricular volume changed by less than 5% for a given load. It was found that 3 elements are sufficient for accurate ventricular volume calculations [5]. Each region (remote, BZ and infarct) was assigned different material properties [5]. Cardiac myofiber angles were assigned to vary

transmurally from -60° to 60° (epicardium to endocardium), relative to the circumferential direction, in all regions [7].

Boundary and Loading Conditions

The boundary conditions of the LV were assigned to fully constrain the displacement at the epicardial-basal edge, while allowing the remaining nodes at the base to move in the circumferential-radial plane. The inner endocardial wall of the LV was loaded with the clinically measured pressures, in order to simulate the end-diastolic (ED) and end-systolic (ES) states. It should be noted that the pressure boundary conditions were applied by quickly ramping the load to the measured value, and then holding the load constant as the simulation reached a steady state solution at ED and ES.

Material Properties of the Heart

Nearly incompressible, transversely isotropic, hyperelastic constitutive laws for passive [8] and active myocardium [9] were modeled with a user-defined material subroutine in the explicit FE solver, LS-DYNA (Livermore Software Technology Corporation, Livermore, CA). Passive material properties were represented by the strain energy function:

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

where E_{11} is fiber strain, E_{22} is cross-fiber strain, E_{33} is radial strain, E_{23} is shear strain in the transverse plane, and E_{12} and E_{13} are shear strains in the fiber-cross fiber and fiber-radial planes, respectively. Values for the material constants b_f , b_t , and b_{fs} were chosen as 24.63, 9.63, and 8.92, respectively, which were scaled (i.e., a value of 6 was added to each exponential material constant) from previous studies of canine myocardium [8]. In order to validate the models, the material constant C was adjusted until the LV enddiastolic volumes matched the experimentally measured values. As was the case in our previous finite element models of the infarcted ovine LV [2, 5, 6], we assume that C has the same value in the borderzone and remote myocardium but is 10 times stiffer in the infarct.

Active contraction was modeled by defining total stress as the sum of the passive stress derived from the strain energy function and an active fiber directional component, $\mathbf{T}_0$, 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, $T_{\max}$ [5],

$$\mathbf{S} = p \mathbf{J} \mathbf{C}^{-1} + 2 \mathbf{J}^{-2/3} \text{Dev} \left(\frac{\partial \tilde{W}}{\partial \mathbf{C}} \right) + \mathbf{T}_0 \{t, Ca_0, l, T_{\max}\} \quad (2)$$

where $\mathbf{S}$ is the second Piola-Kirchoff stress tensor, p is the hydrostatic pressure introduced as the Lagrange multiplier needed to ensure incompressibility, $\mathbf{J}$ is the Jacobian of the deformation gradient tensor, $\mathbf{C}$ is the right Cauchy-Green deformation tensor, Dev is the deviatoric projection operator,

$$\text{Dev}(\bullet) = (\bullet) - \frac{1}{3} ([\bullet] : \mathbf{C}) \mathbf{C}^{-1} \quad (3)$$

and $\tilde{W}$ is the deviatoric contribution of the strain energy function, W (Eq. 1). The assumption of near incompressibility of the myocardium requires the decoupling of the strain energy function into dilational and deviatoric components,

$$W = U(J) + \tilde{W}(\tilde{C}) \quad (4)$$

where U is the dilatational (volumetric) contribution [10].

The active fiber-directed stress component is defined by a time-varying elastance model, which at end-systole, is reduced to [11],

$$T_0 = \frac{1}{2} T_{\max} \frac{Ca_0^2}{Ca_0^2 + ECa_{50}^2} \left(1 - \cos \left(\frac{0.25}{m l_R \sqrt{2 E_{11} + 1} + b} + 1 \right) \pi \right) \quad (5)$$

with m and b as constants, and the length-dependent calcium sensitivity, ECa_{50} , is given by,

$$ECa_{50} = \frac{(Ca_0)_{\max}}{\sqrt{\exp \left[B \left(l_R \sqrt{2 E_{11} + 1} - l_0 \right) \right]} - 1} \quad (6)$$

where B is a constant, $(Ca_0)_{\max}$ is the maximum peak intracellular calcium concentration, l_0 is the sarcomere length at which no active tension develops and l_R is the stress-free sarcomere length. The material constants for active contraction were taken to be [12]: $Ca_0 = 4.35 \mu\text{mol/L}$, $(Ca_0)_{\max} = 4.35 \mu\text{mol/L}$, $B = 4.75 \mu\text{m}^{-1}$, $l_0 = 1.58 \mu\text{m}$, $m = 1.0489 \text{ sec } \mu\text{m}^{-1}$, $b = -1.429 \text{ sec}$, and l_R was set at $1.85 \mu\text{m}$, the sarcomere length in the unloaded reference configuration. Based on biaxial stretching experiments [13] and FE analyses [14, 15], cross-fiber, in-plane stress equivalent to 40% of that along the myocardial fiber direction was added. In order to further validate the models, the parameter $T_{\max}$ was formally optimized.

Material Parameter Optimization

The objective function for the optimization was taken to be the mean squared error (MSE) [1]. The passive material parameters were determined such that the FE model-predicted end-diastolic LV volume matched the patient-specific *in-vivo* measured value. The systolic material parameters in the remote ($T_{\max\_R}$) and borderzone ($T_{\max\_B}$) regions were estimated by minimizing the errors between FE model-predicted and *in-vivo* measured systolic strains and end-systolic LV volume. The goal of the optimization is to minimize the MSE,

$$\text{MSE} = \sum_{n=1}^N \sum_{i=1,2,3; j=1,2,3; i \neq j} w_n \left(E_{ij,n} - \bar{E}_{ij,n} \right)^2 + w_{VED} \left(\frac{V_{ED} - \bar{V}_{ED}}{\bar{V}_{ED}} \right)^2 + w_{VES} \left(\frac{V_{ES} - \bar{V}_{ES}}{\bar{V}_{ES}} \right)^2 \quad (7)$$

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 the experimental *in-vivo* measurements.

Results

The end-diastolic and end-systolic LV pressures were measured with cardiac catheterization, and found to be 10 mmHg and 107 mmHg, respectively. These values were applied to the endocardial wall of the FE model as a patient-specific boundary condition. The measured end-diastolic and end-systolic volumes were found to be 60.3 mL and 35.3 mL, respectively. The computed end-diastolic and end-systolic volumes from the optimization were 60.3 mL and 37.05 mL, respectively, which are within 5% of the measured values.

The optimization displayed good convergence, with a 90% confidence interval of 18%, which only took 10 iterations to reach (Figure 2). However, the optimization procedure was allowed to run 5 extra iterations to ensure convergence. The final MSE value of 8.53 was calculated using 816 strain components and two volumes. The MSE value indicates generally good agreement between the FE model-predicted systolic strains and the patient-specific *in-vivo* measured strains, and is similar in magnitude to our previous studies, which were validated with infarcted ovine hearts [5, 6].

The passive material parameter was found to be $C = 0.195$ kPa, which allowed the FE model LV volume to match the experimental value within 2%. The optimized contractility parameters for this patient are given in Table 1, and were found to be $T_{\max\_R} = 109.5$ kPa and $T_{\max\_B} = 73.1$ kPa. This shows a 33% decrease in contractile function in the borderzone compared to the remote region. In order to assess the influence of varying borderzone helix fiber angles, a sensitivity study was conducted in which the BZ fiber angles were rotated. Table 1 shows the effect of helix angles in remote (HA_R), borderzone (HA_B) and infarct (HA_I) regions on the optimized parameters $T_{\max\_R}$ and $T_{\max\_B}$. A helix angle is the angle (in degrees) that the local myofiber direction makes with the circumferential direction (e.g., a helix angle of zero means the myofiber direction is circumferential, whereas a helix angle of 90 degrees means the myofiber direction is longitudinal). A twenty-degree “leftward” shift in HA_B and HA_I (Case 2 vs 1) results in only a 2.1% decrease and 9.5% increase in $T_{\max\_R}$ and $T_{\max\_B}$, respectively.

It can be seen in Figure 3 that the systolic myofiber strain in the remote region of the FE model is negative, which implies the myofibers are shortening during contraction. In addition, the strain in the BZ is elevated (i.e. less negative) relative to the remote region, indicating that the myofibers are being stretched during systole. The infarct region demonstrates positive strain indicating that this region is non-contractile and dyskinetic. It can be seen in the CSPAMM image (Figure 1a) that the posterior wall has virtually no contractility during the systolic phase (region in the blue circle). This confirms the presence of the MI, which was assumed to have $T_{\max\_I} = 0$ kPa.

Comment

These studies provide the first evidence that myocardial contractility in the BZ region of a human MI is substantially less (25%–33% depending on regional myofiber orientation) than that in regions remote from the MI. The temporal progression in extent and severity of regional contractile dysfunction in normally perfused myocardium adjacent to the infarct has been shown to be an important pathophysiologic feature of the adverse remodeling process in large animal models [16]. The results reported here support that the process demonstrated in large animal models likely occurs in patients

Myocardial tissue tagging using complimentary spatial modulation of magnetization (CSPAMM) allows detailed assessment of myocardial motion. To capture the complex 3D cardiac motion pattern, multiple 2D tagged slices are usually acquired in different orientations. These approaches are prone to slice misregistration and associated with long acquisition times. In this work, we applied the accelerated 3D tagging acquisition method of Rutz et al. [17], which enabled assessment of 3D motion information with whole heart coverage in three short breath-holds. They found hypokinetic regions in patients with an MI corresponded well with regions exhibiting hyperenhancement after contrast injection. However, Rutz et al. did not attempt to quantify the forces or stresses responsible for that hypokinesis.

Using an MRI-based finite element stress analysis of a clinically relevant large animal preparation, Guccione et al. [2] suggested that the mechanism underlying mechanical dysfunction in the BZ of LV aneurysm is primarily the result of myocardial contractile dysfunction rather than increased wall stress in this region. Then, Walker and co-workers used a fixed ratio of 2:1 in remote versus BZ region contractility to compute regional myocardial material properties and stress in six animals [18] after linear repair of LV aneurysm and in five animals that underwent a sham operation [15]. More recently, Sun et al. [5] developed a computationally efficient formal optimization that allows regional myocardial contractility to be quantified without enforcing the fixed ratio mentioned above. Moreover, *in-vivo* estimates of regional myocardial contractility were validated using *ex-vivo* direct force measurements in skinned fiber preparations. Then, that formal optimization was applied in six animals two weeks before and two and six weeks after patch repair of LV aneurysm [16], as well as in an animal with posterobasal MI [6]. Most important, in every single animal and time point included in those previous studies (32 instances), myocardial contractility in the BZ of the MI was significantly less than that in regions remote from the MI.

In this paper we present two distinct values for the contractility parameters for borderzone ($T_{\max\_B}$) and remote myocardium ($T_{\max\_R}$). In a recent study of an ovine model of LV aneurysm [19], we found that our corresponding finite element model was better able to reproduce the experimental LV pressure versus myocardial strain data when we allowed $T_{\max\_B}$ to have values that vary linearly from zero at the boundary between the aneurysm and borderzone to $T_{\max\_R}$ at the boundary between the borderzone and remote myocardium. We expect a similar gradient in $T_{\max\_B}$ to exist in the borderzone of other types of MIs, including the MI of the patient in the present study.

At present, the greatest challenge in quantifying regional myocardial contractility in patients is the very sophisticated MRI hardware required to quantify regional myofiber orientation *in-vivo*. The diffusion imaging approach proposed by Gamper et al. [20] appears to be the state-of-the-art, but it requires an MRI scanner with uniquely large gradients (of at least 80 mT/m). Because we did not have access to such a scanner, we instead repeated our quantification of regional myocardial contractility in a patient with an MI using a range of myofiber angle distributions. Case 2 of the fiber angle sensitivity study corresponds to the *in-vivo* dMRI data of Wu et al. [21, 22] concerning patients with an MI in which the percentage of left-handed helical fibers (negative subepicardial helix angles) increased from the remote zone to the borderzone and infarct zone, and the percentage of right-handed helical fibers (positive subendocardial helix angles) decreased from the remote zone to the borderzone and infarct zone. The findings of Wu et al. [21, 22] are at variance with the *ex-vivo* observations of Chen et al [23]. Recent *ex-vivo* dMRI data from infarcted rat hearts indicate that borderzone helix angles are preserved [24, 25].

Conclusions and Limitations

This investigation demonstrates for the first time the substantial depression of myocardial contractility in the BZ of a human MI relative to that in regions remote from the MI. The current study evaluates one patient at one time point very remote from the index MI; however, the results of this study support the central role of the BZ myocardium in the post MI remodeling process in patients that has been demonstrated in large animal infarct models. To fully establish this important conclusion serial evaluations beginning at the time of the index MI will need to be performed in a large cohort of patients. The current study supports the importance and demonstrates the feasibility of such large and long-term studies.

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Figure 1.

(a) Short axis view from 3D CSPAMM image of patient LV, with the posterior region circled in blue, and (b) FE model that was reconstructed from the MR images, showing the remote (red), borderzone (green), and infarct (tan) regions.

Figure 2. Convergence plots of (a) $T_{\max\_R}$ and (b) $T_{\max\_B}$ during the numerical optimization process. Note that the solution was stable after roughly 10 iterations.

Figure 3.

End-systolic myofiber strain distribution. (a) View of epicardial surface with infarct and borderzone outlined, (b) interior view of posterior endocardial wall with infarct and borderzone outlined, (c) mid-ventricular slice through remote, borderzone, and infarct. Note the elevated strain in the borderzone.

Table 1

Contractility parameter, based on myofiber helix angle

Case	HA _R (deg)	HA _B (deg)	HA _J (deg)	T _{max_R} (kPa)	T _{max_B} (kPa)
1	-60 (epi) to 60 (endo)	-60 (epi) to 60 (endo)	-60 (epi) to 60 (endo)	109.5	73.1
2	-60 (epi) to 60 (endo)	-80 (epi) to 40 (endo)	-80 (epi) to 40 (endo)	107.2	80.1