Hemodynamic Effects of Infarct Location in Left Ventricular Wall Based on an Integrated Finite Element and Circulatory Model

SA Chiaramida[1], R Dong[2], FJ Vetter[2], Y Sun[2]

[1]Cardiology, Medical University of South Carolina, Charleston, SC, USA
[2]Biomedical Engineering, University of Rhode Island, Kingston, RI, USA

Abstract

An integrated finite element and circulatory model was developed and used to assess the differential impact of infarct location on left ventricular hemodynamics. In this study a finite element model of the left ventricle (LV) was fully integrated with an electrical analog model of the circulatory system. The model was capable of generating pressure and volume waveforms that are consistent with the clinical observations. Simulations based on this model determined the hemodynamic effects of an infarction located near the base, mid-wall, or apex of the LV. Infarction near the apex showed lesser effects than infarctions near the base or the mid-wall. Infarctions near the base and mid-wall showed similar effects. The decrease in ejection fraction was most sensitive to infarction near the base, while the increase in end-systolic volume and end-diastolic volume was most sensitive to infarction near the mid-wall.

1. Introduction

The differential impact of infarct size and location on left ventricular systolic and diastolic functions has not been well defined in the medical literature. In this paper, a finite element and circulatory model was used to assess the hemodynamic effects of different infarct size and location. The model developed for this study is capable of relating regional myocardial impairment to its effect on global hemodynamics.

In this study a finite element model (FEM) of the left ventricle was fully integrated with an electrical analog model of the circulatory system so that global LV contractility and regional LV wall motion abnormalities can be related to hemodynamics. Finite element methods based on the potential energy approach were applied to two two-dimensional (2D) long-axis cross-sectional slices: one for normal myocardium and one containing an infarct zone. The three dimensional (3D) LV model was reconstructed from the two slices based on axial symmetry by circumferential interpolation. A time-varying Young's modulus curve over a cardiac cycle was assigned to the finite elements for normal myocardium. A reduced Young's modulus curve was assigned to the infarct finite elements [1]. The circulatory model was represented by an electrical analog circuit governed by 28 nonlinear differential equations [2]. Simulations based on this integrated model were conducted to predict the hemodynamic effects of varying size and location of the infarction. The model was capable of generating pressure and volume waveforms that are consistent with the clinical observations [3]. The hemodynamic effects were assessed by LV ejection fraction (EF), end-systolic volume (ESV), and end-diastolic volume (EDV) as functions of the percent infarct size. The model was intended to predict 1) the direct hemodynamic consequence of reduced LV contractility due to the presence of infarction for a given size and 2) a secondary effect of preload elevation induced by compensatory mechanisms built into the circulatory model.

By carefully defining the grids of the finite element model, an infarct zone of an exact size can be positioned near the base, mid-wall, or apex of the LV. The resulting differences in hemodynamics should reveal the significance of the infarct location. Such a study would be difficult to conduct with animal or human models, thereby justifying the use of a computer model.

2. Methods

The 3D LV in this study was based on a 2D axisymmetric model with triangular elements. The LV wall was modelled as a thick-walled truncated ellipsoid. The unloaded LV mesh was generated by a set of 2D elliptic curves rotated around the LV long axis. The LV geometry parameters were user-defined. The input load to LV was the left ventricular pressure (Plv). The boundary conditions were set such that nodes at the apex could displace along the long-axis direction but not along the short-axis direction. Conversely, the nodes at the base could displace along the short-axis direction but not along the long-axis direction. The material properties of each element were assumed to be isotropic, homogeneous, and linearly elastic that can be completely characterized by Young's modulus (Y) and Poisson's ratio (ν). The

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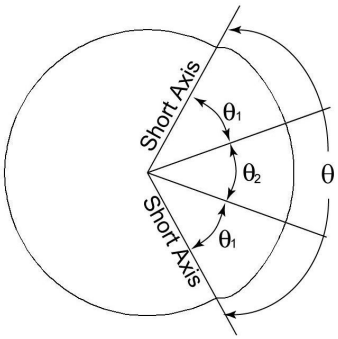

Figure 1. The infarct zone was defined by angle θ (θ =θ_1+ θ_2+ θ_1), where θ_1 was the angle for the akinetic regions and θ_2 was the angle for the dyskinetic region. In this study, we set $\theta_1 = \theta_2$. The sector outside of θ was defined as zone of normal myocardium.

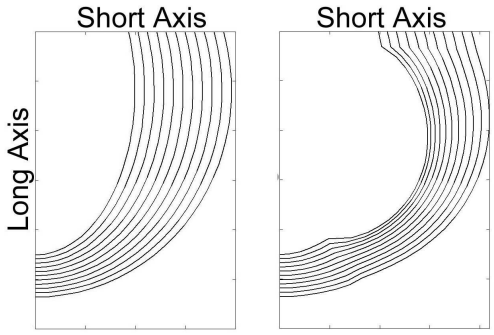

Figure 2. For the purpose of computational efficiency, only two 2D slices, one normal slice (left panel) and one infarct slice (right panel) were calculated at each time step. In order to keep the clarity of the figure, layers instead of elements are shown.

contractile and elastic properties of the element were represented by Young's modulus that varied over a cardiac cycle. A time-varying Young's modulus curve was chosen empirically and assigned for all the elements for normal myocardium, and a reduced time-varying Young's modulus curve was assigned to the elements for impaired myocardium. Thus the normal and impaired elements showed different contractile properties within a cardiac cycle.

The infarct zone was defined within a sector on the short-axis plane, with the user-defined top and bottom borders generally parallel to the short axis. The sector angle θ was user-defined. It includes all the tissue within the sector from the epicardium to endocardium, from the top border to bottom border. An infarct zone was composed of one dyskinetic region and two akinetic regions. Figure 1 shows the cross-section of an infarct zone in the short-axis plane.

For computational efficiency, the 3D LV geometry was reconstructed from 2D slices. Only two 2D slices of the LV wall were computed. Each slice represented a cross section of a quadrant of the LV wall parallel with the long axis. For the normal slice a normal Young's modulus was assigned to all finite elements within the slice. For the infarct slice a reduced Young's modulus was assigned to the finite elements within the infarct zone. Figure 2 shows a normal slice (left) and an infarct slice (right) at early systole in a cardiac cycle. At each time step, the 3D LV geometry was reconstructed by assuming that the normal sector and dyskinetic sector were axisymmetric (see Figure 1). The infarct slice from the 2D model was assigned to the dyskinetic sector. The normal slice was assigned to the normal sector, so each akinetic sector was located between the normal slice and one infarct slice. Circumferential interpolation was applied to the akinetic sectors. This was accomplished by use of the square root of sine function from 0 to 90 degrees.

With the 3D LV geometry generated by the 2D model, left ventricular volume (Vlv) at each time step was calculated by numerical integration.

As shown in Figure 3, the FEM of the LV interacted with the analog model of circulatory system. At each time step, the inputs to the FEM were the unloaded LV mesh and the Plv generated by the electrical analog model of circulatory system. With two 2D slices calculated by the FEM, a 3D LV geometry was reconstructed. Then Vlv was calculated, and the instantaneous LV elastance (Elv) is determined by Elv = Plv / Vlv. This elastance was used to propagate the state of the electrical analog model to the next time step.

The new ventricular pressure was further used to drive the FEM and the iteration continued. This interaction between the FEM and the electrical analog model occurs at a time step of 5 ms for the entire cardiac cycle, and continued over 10-25 cardiac cycles (depending on the infarct size and initial conditions) to reveal the transient and steady state responses.

Software for the integrated FEM and the electrical analog model was developed on an open source C++

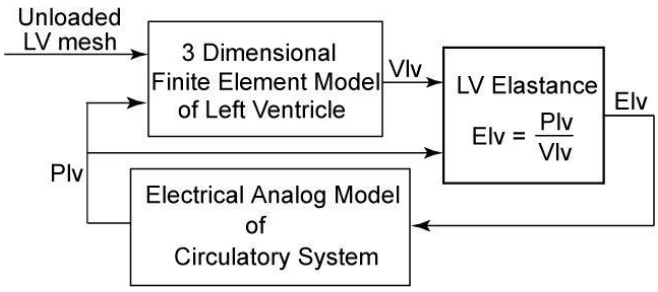

Figure 3. Information flow between the FEM of the LV and the electrical analog mode of the circulatory system.

platform. The 2D slice of the LV wall was represented with 10 layers transmurally, and 60 elements on each layer.

3. Results

The unstressed LV geometry was defined such that the total LV mass was about 151g. This value was set according to clinic data reported by Grothues et al. [4]: LV mass = 148 ± 36g. The myocardium density was assumed to be 1.05g/cm^3. On a 3.0 GHz desktop computer with 1GB RAM, the computation time for a cardiac cycle with heart rate of 71 bpm was about 9 minutes. Depending on the infarct size, the model required 10-25 cardiac cycles to reach steady-state.

Simulations based on this integrated model were carried out to assess the hemodynamic effects of varying infarction. The reconstructed 3D LV geometry showed a reasonable shape of LV remodelling after a myocardial infarction. The result also showed that this model was capable of generating pressure and volume waveforms that are consistent with the clinical observations, as shown in Figure 4.

This model was used to assess the hemodynamic effects of an infarction located near the base, mid-wall or apex. For each location, infarctions of different percentage infarct sizes were simulated. The hemodynamic effects were assessed by LV EF, ESV and EDV as functions of the percent infarct size.

As shown in Figure 5, when infarct size increased, reduced contractility caused EF to decrease. The model also correctly represented the preload elevation secondary to LV diastolic dysfunction, indicated by progressive increases in ESV and EDV, as demonstrated in Figures 6 and 7.

These results suggest that infarctions near the apex showed lesser effects than infarctions near the base or the

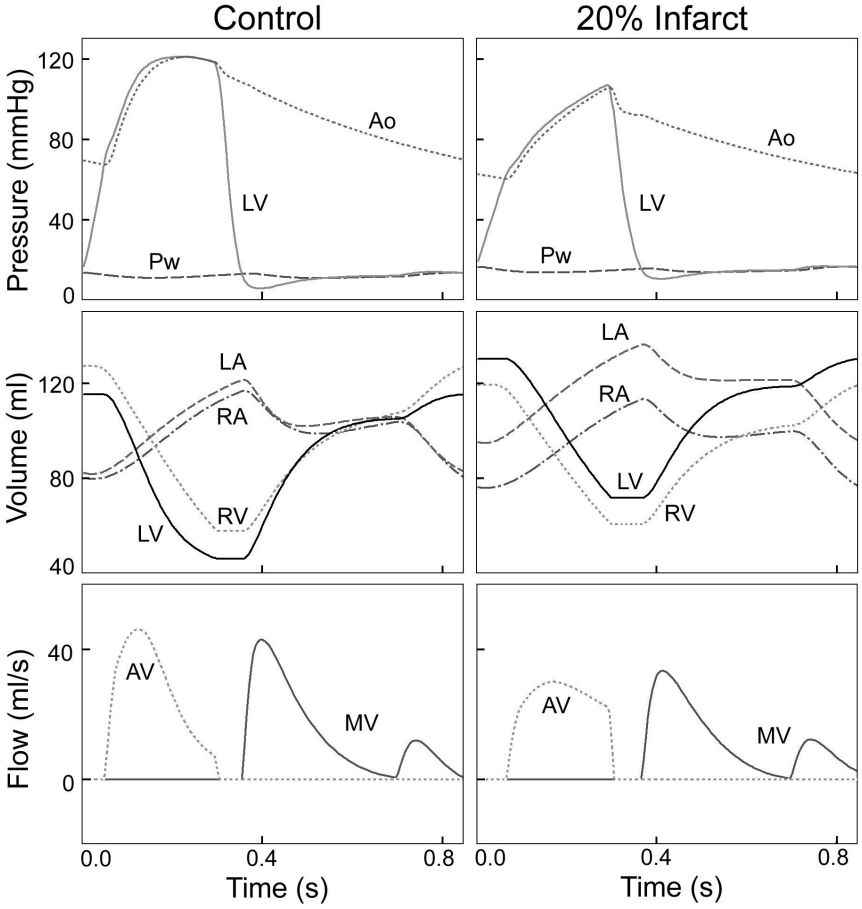

Figure 4. Hemodynamics generated by the integrated finite element and circulatory model. The left column shows the curves of a normal LV (Control), the right column shows curves of a LV with a 20% infarct. For pressure, Ao, aortic pressure; LV, left ventricular pressure; Pw, pulmonary capillary wedge pressure; For volume, LA, left atrial volume; RA, right atrial volume; LV, left ventricular volume; RV, right ventricular volume; For flow, AV, flow through aortic valve; MV, flow through mitral valve.

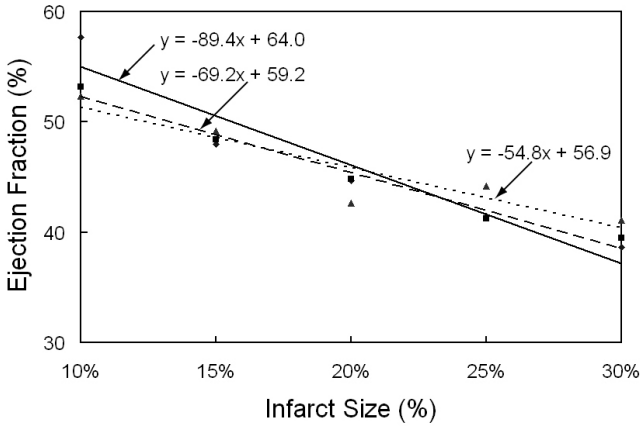

Figure 5. The model predicted that EF decreased as infarct size increased: linear regression fit (lines) to model data (dots). Solid line: the infarction located near the base. Dash line: the infarction located near the mid-wall. Dotted line: the infarction located near the apex.

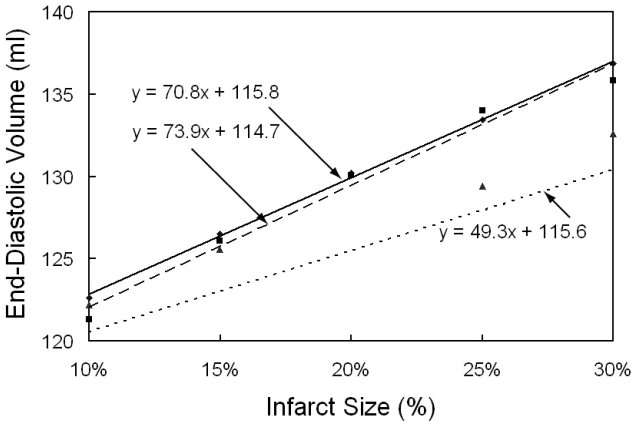

Figure 6. The model predicted that EDV increased as infarct size increased: linear regression fit (lines) to model data (dots). Solid line: the infarction located near the base. Dash line: the infarction located near the mid-wall. Dotted line: the infarction located near the apex.

mid-wall. Infarctions near the base and mid-wall showed similar effects. The decrease in EF was most sensitive to infarctions near the base, while the decrease in ESV and EDV was most sensitive to infarction near the mid-wall.

4. Discussion and conclusions

This study demonstrated the utility of a computational model that combines a finite element model with an electrical analog model. The integrated model proposed in this study was capable of relating a regional myocardial impairment of specific size and location to its effects on global hemodynamics. Since the 3D LV geometry was actually reconstructed by two 2D slices, this model is computationally efficient.

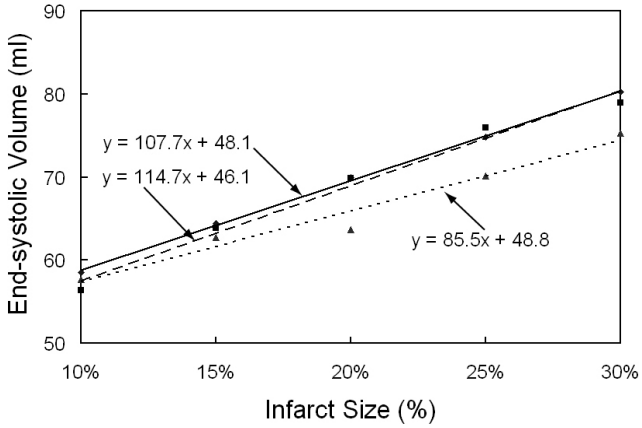

Figure 7. The model predicted that ESV increased as infarct size increased: linear regression fit (lines) to model data (dots). Solid line: the infarction located near the base. Dash line: the infarction located near the mid-wall. Dotted line: the infarction located near the apex.

Acknowledgements

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Address for correspondence

Salvatore A. Chiaramida, MD, Professor of Medicine
Cardiology, Medical University of South Carolina
135 Rutledge Avenue
Charleston, SC 29425
USA
E-mail: chiara@musc.edu