The Simulation Study of the Influence of Electrical Asynchrony on Regional Mechanics of the Ischemic Ventricle Using Electromechanical Heart Models

L Xia, M Huo, F Liu

Department of Biomedical Engineering, Zhejiang University, Hangzhou 310027, PR China

Abstract

Using mathematical modeling method, this paper investigates the interactions between electrical conduction and mechanical function in the ischemia or infarcted ventricle. The influence of electrical asynchrony on regional mechanics of the ventricle is simulated based on electromechanical heart models. Regional deformation, strain and stress are calculated during systole phase. The preliminary results show that considerable disruption of the contraction pattern occurs in the near ischemic zone, the minimum principal strains in local infarction regions were significantly smaller than those in corresponding regions of the normal heart, while the stresses are larger than those of normal tissues. The simulated results are compared with dog experiments and solutions obtained in the literature. This simulation suggests that such coupled heart models can be used to assess the mechanical function of the ventricle with diseases such as myocardial ischemic or infarction.

1. Introduction

The human heart is a neuroelectrically actuated mechanical pump. Before mechanical contraction, the electric excitation is generated and transmitted throughout the heart wall via special transmission system. When myocardial ischemia or infarction (MI) occurs, the electrical transmission progress or pattern is much more heterogeneous and asynchronous [1-3], which causes various abnormalities in left ventricle (LV) function [4]. For example, the influence of the electrical asynchrony on regional mechanics of LV is obvious in such pathological condition, but the knowledge of the excitation-contraction coupling mechanism in three-dimensional infarcted ventricular wall is limited [5]. The purpose of this paper is to investigate the interactions between electrical conduction and mechanical function in the infarcted LV using mathematical modeling method.

Information about LV mechanical performance is of critical importance in understanding the etiology of heart disease such as MI [6]. Regional wall stress and strain are often used to assess LV function of ischemia and MI. Myocardial strain distributions can be measured experimentally and clinically [7], while regional wall stresses must be computed from mathematical models because reliable measurement of ventricular wall stress is practically impossible. A number of mechanical heart models have been developed to approximate wall stress and some of them were presented for the ischemic ventricle [8-13]. Although these models are reasonable enough to reproduce some complex features of regional myocardial mechanics, the influence of the asynchronous electrical activation after MI is usually simplified or neglected. In this investigation, we attempt to evaluate the influence of the electrical asynchrony on cardiac contractility of the infracted LV during systole phase. The analysis is based on an earlier developed electrical heart model [14] (E-model) and a mechanical heart model [15] (M-model). At first, the activation series of the infarcted LV are simulated by the E-model, and then the active force is calculated based on the results from the E-model, finally the cardiac mechanics were calculated by the M-model and verified by other mathematical model results and imaging solutions.

2. Method

2.1. Model description

Based on the image data of the human body and heart sections, the E-model was reconstructed as a 3-D array of approximately 65000 cell units spaced 1.5mm apart. An action potential waveform with variable in duration is assigned to each unit. The excitation propagation algorithm is a hybrid of the rule-based algorithm and the wavefront-based algorithm. It was proved that this propagation algorithm is efficient with reasonable accuracy [14]. Considered the myocardial fiber orientation, a 3-D finite element mechanical model of human left ventricle is also constructed based on composite theory and an electrical model of heart, and the computing issues of the active force and after-load are discussed [15]. In the M-model, 3-D nodes isoparametric element was the basic element, and there were total 760 brick elements, 1005 nodes and 3015 degrees of freedom. We developed a special isoparametric method to discretize the LV, whose major feature was that each element can accommodate several layers having different

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Computers in Cardiology 2002;29:505-508.

ply orientations. It could provide accurate solutions using less degrees of freedom than conventional finite element methods, and it saved both in computer memory and computational time. According to the finite element discretization, the active force vectors are unidirectional in a layer. After the activation sequences were determined by E-model, the active forces per element could be determined. Using this M-model, the regional strain, stress and deformation of LV could be calculated.

2.2. Myocardial ischemia simulation

Myocardial ischemia or MI is an imbalance between the supply of oxygenated blood and the oxygen requirements of the myocardium. According to the degree of myocardial disease, the condition can be identified as myocardial ischemia, injury, or necrosis. The asynchrony of electrical activation on the infarcted LV is simulated by the E-model first and then the abnormal mechanical behavior of LV is simulated by the M-model.

The ischemia or MI can change the myocardial action potentials. The principal changes are decreases in the magnitude of the resting potential and in the action potential duration. Action potentials with prolonged duration have been observed in the infracted regions of experimental animals after the development of inverted T waves in the surface electrocardiogram (ECG) [1-2]. Such abnormal action potentials are used in the E-model to study the effects of acute myocardial ischemia and infarction (Fig. 1).

When the active sequences are determined based on the electrical simulation, the stress, strain and displacement during systole could be calculated. The simulation and calculation are realized by adjusting the model parameters according to some data or ideas from the references [8-9, 11]. The model editor can deal with three kinds of parameters: myocardial fiber type (normal and diseased muscle) and position, material parameters (elastic modulus, Poisson's ratio, etc.) and external loads (active force, cardiovascular loads, etc.). Once model parameters are determined, the simulation can be carried out.

In the fiber-coordinate system (one axis is chosen to coincide with the local muscle fiber direction, another one is determined by the epicardium surface normal vector.), the expression of the active force along the fiber is:

$$\sigma_e^{'} = \begin{Bmatrix} \sigma_o^{'} \sin(\frac{t-\tau}{T_e}\pi), & 0 \le t \le T_e \\ 0, & t > T_e \end{Bmatrix} \quad (1)$$

where t is the time; τ is the time lag of active stress, T_e is the activation period. $\sigma_o^{'}$ is the active force of a myocardial fiber and which is a function of fiber length, time t, fiber directions, etc.

In the calculation, the stress and strain vector is expressed with respect to two coordinate frame of reference: global coordinate system X-Y-Z and local coordinate system ξ-η-ζ. The nodal force equivalent to the active contractile force is calculated based on equation (1) and the isoparametric transformation:

$$\{F_f\}^e = -w\sum_{l=1}^{L_e}\int_{\zeta_{l-1}}^{\zeta_l}\int_{-1}^{1}\int_{-1}^{1}[B]^T T\{0,0,\sigma_e^{'},0,0,0\}_l^T |J| d\xi d\eta d\zeta \quad (2)$$

where [B] is geometric matrix of element, l, L_e stand for the number and total number of layers in an element respectively, T is the transformation matrix between the fiber coordinate and global coordinate. $\{0,0,\sigma_e^{'},0,0,0\}_l^T$ is the active force row vector in the fiber-coordinate system, $|J|$ is the determinant of the Jacobian matrix. ξ, η, ζ are the local coordinate system with the magnitudes ranging from -1 to 1; ζ^l and ζ^{l-1} represents individually the coordinate values along ξ direction on the front-side and back-side of layer l in an element. When l=1, ζ^{l-1}=-1, when l=L_e, ζ^l= 1. w is a MI factor. It represents the ability of the tissue to generate active force and varies between 0 (completely ischemic tissue) and 1 (normally contracting tissue). The velocity-stress relation is neglected here.

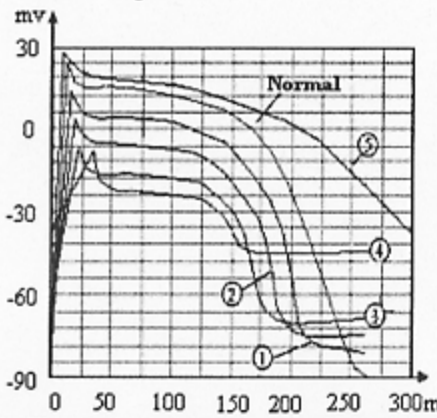

Fig. 1 The action potential waveforms of ischemia myocardium used in the E-model. The injured degree of the muscle become more severe from the action potential curve (1) to (4), and curve (5) is the prolonged action potential waveform caused by ischemia.

3. Results

3.1. Simulated ECG based on E-model

Fig. 2 (a) shows the simulated Lead-III ECG of the normal human heart (left) and of the abnormal heart with ischemia on left anterior ventricle (right). To verify our E-model, we did some dog experiments. First, a suture snare was fitted to the equatorial region of the left anterior descending coronary artery of the dogs. Then, we recorded the Lead-III ECG using the MadLab-U biological signal collect and analysis system. Fig. 2 (b)

shows the normal ECG of one dog (left) and the ischemia ECG of the same dog (right). From Fig. 2, we could see that our E-model can simulate ECG of ischemia with reasonable accuracy.

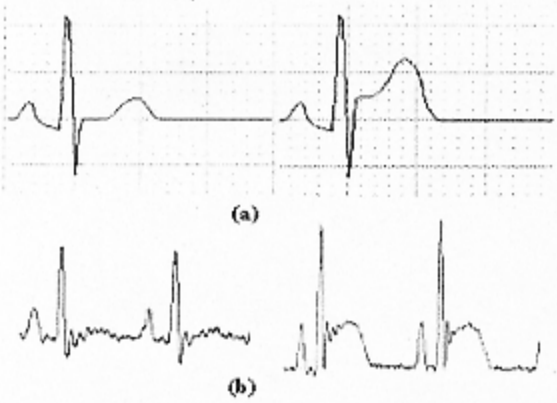

Fig. 2 Simulation and experiment results of ECG (see the text description).

3.2. Deformation

Fig. 3 shows a movie sequence of the infarcted LV during systole. The deformation is taken over three time sequences (T1<T2<T3, which comes near end-diastole, middle-systole and end-systole, respectively). It shows that the contraction procedure of LV is complicated, including axial shortening, radial contraction and twisting at different degrees. The deformation in the healthy zone is relatively homogeneous, while near the infarcted zone is irregular. Suffered high pressure, the diseased zone bulges outside of the LV wall. This typical deformation, showing a bulging of the ischemic region, has also been found in experiments [9].

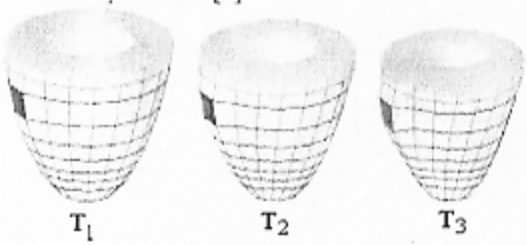

Fig. 3 The deformation of the infarcted LV during systole.

3.3. Stress fields

The stress distribution relates with the activation sequences. When the MI disturbs the electrical excitation regionally in the ventricular wall, the time lag of active force is changed compared with the normal one. From Fig. 4, it is easy to find that the stress near the diseased tissue is larger than that of normal tissue, in the normal zone the stress on the inner wall is larger than that on the outer wall. The predicted peak stress in the infracted LV is compressive and has a magnitude about eight times that in the normal LV. These results are the same as the conclusion of paper [12].

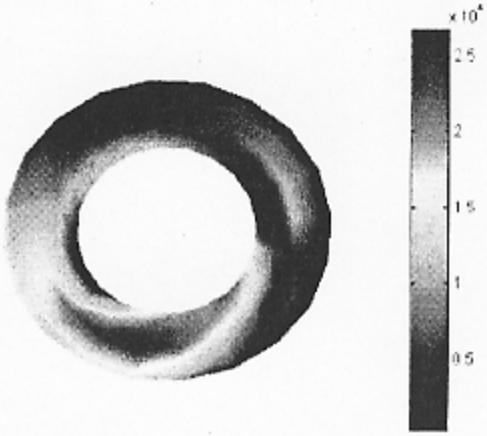

Fig. 4 The stress distribution on the cross-section of LV with MI.

3.4. Strain distribution

Strain is a quantity of interest because it can be used to quantify the amount of tissue contraction. Our simulation indicates that, in the normal zone, the maximum strain is in the radial thickening direction, and the minimum one is roughly in the fiber direction. These results are similar to those from tagged MRI [16], and it coincides with the LV physiological phenomena of radial thickening and shortening of fibers during the systole. In the abnormal region, however, the minimum value is in the radial direction, indicating that the heart wall is being compressed rather than thickened.

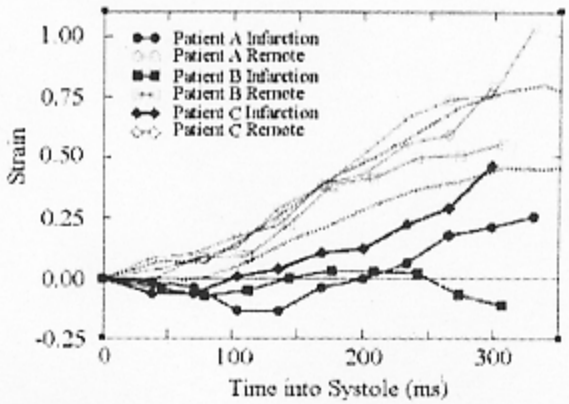

Fig. 5 The evolution of strain of the infarcted LV during systole phase.

Fig. 5 shows the evolution of strain in the ischemic zone for three patients with inferior ischemia, the data was obtained by tagged MRI [17]. Fig. 6 is the strain-time curve from our simulation. From these two figures, it is obvious that the model could predict the evolution of strain throughout the diseased heart during systole. The

discrepancy may be due to model parameters such as the position, size of the ischemic region. These influences of the asynchrony on the strain were also found in some other experiments [2].

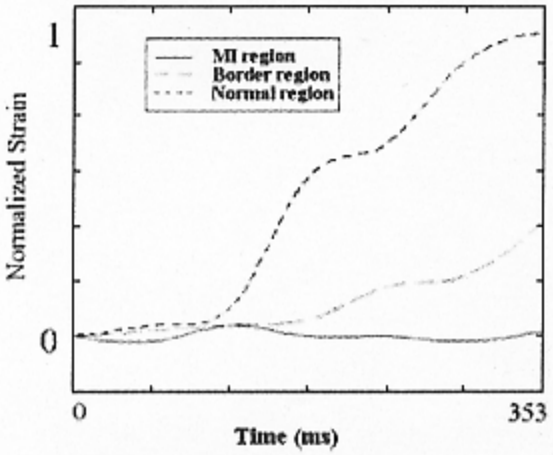

Fig. 6 Simulated and normalized strain-time curve of the infarcted LV during systole phase.

4. Conclusions

The regional mechanics of the infarcted LV during systole phase is studied using a mechanical heart model and an electrical heart model. The investigation focuses on the influence of electrical asynchrony on ventricular wall stress, strain and deformation. The preliminary simulation results show considerable disruption of the contraction pattern near the diseased zone, the minimum principal strains in local MI regions were significantly smaller than those in corresponding regions of the normal hearts, while the stress is larger than normal tissues. So asynchronous electrical activation after MI deteriorates the regional mechanical performance of LV. This simulation suggests that such electromechanical model provides a way to investigate the biomechanical function of the LV with diseases such as MI, and more realistic models of cardiac function are useful for clinical evaluation of heart disease.

Acknowledgements

This project is supported by National Natural Science Foundation of China (30170243) and the Special Grant for the Authors of Excellent Doctoral Dissertation of Ministry of Education (199941).

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Address for correspondence.

Ling Xia
Department of Biomedical Engineering
Zhejiang University, Hangzhou 310027, PR China
E-mail:xialing@hzcnc.com.