The Construction of Three-dimensional Composite Finite Element Mechanical Model of Human Left Ventricle*

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Cardiomechanics deals with the motion and functioning of the heart. How the heart performs as a pump depends largely on the contractile activity of its wall muscle, the myocardium. The mechanical properties of myocardial fibers depend highly on their fibrous structures and activity status. Considered the myocardial fiber orientation, a three-dimensional finite element mechanical model of human left ventricle is constructed based on composite theory and an electrical model of heart, and the computing issues of the active force and after-load are discussed. Finally the mechanical response of the left ventricle subjected to the normal active force is simulated, and the results are compared with solutions obtained from MR tagging images and those from other models. It proves that the given model can be used to study the mechanical function of the left ventricle, and it lays a good foundation for construction of virtual heart with electro-physiological and mechanical properties.

Key Words: Biomechanics, Human Left Ventricle, Mechanical Model, Electrical Model, FEM Analysis, Composite, Active Force

1. Introduction

For a long time, many researchers have devoted themselves to the biomechanical study of the strongest cardiac pump of human heart-left ventricle (LV), hoping to better understand its dynamic properties and kinetic laws. In the absence of reliable techniques to measure force in the ventricular wall, much work has been done on modeling LV particularly for stress and strain analysis of the myocardium. Initially, the classical mathematical models of LV are analytical ones with some simplified assumption; Gradually, with the rapid development of computer technology and medical image technology, finite element method (FEM) has become one of the main tools to study cardiovascular mechanics⁽¹⁾⁻⁽³⁾. The objectives of finite element (FE) cardiac analyses are to quantitate regional myocardial stresses, to examine regional myocardial mechanical properties for both normal and diseased states, and ultimately to provide useful

information in diagnosis and procedure for surgical and medical intervention of patients with heart diseases.

In the literature, different FE heart models described important different features of the mechanics of the heart muscle. In paper⁽⁴⁾, an axisymmetric porous medium FE model was constructed to describe the mechanics of the LV. It took into account time dependent effects as redistribution of intracoronary blood during the cardiac cycle and viscoelastic behavior of the myocardial tissue resulting in hysteresis. While Paper⁽⁵⁾ proposed a 3-D FE model of the beating LV to investigate the dependence of local mechanics on myocardial muscle fiber orientations. This model account for the dependence of active stress on time, strain and strain rate, activation sequence of the LV wall and aortic after-load. It was proved that the distributions of the active muscle fiber stress and muscle fiber strain across the LV wall are sensitive to the fiber orientation. In paper⁽⁶⁾, a 3-D nonlinear FE model of the canine LV with a long-axis geometry and muscle fiber distribution based on published measurements for the anterior free wall was used to compute regional distributions of muscle fiber

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stress at the end-diastole and end-systole. The results indicate that the regional heterogeneity of the fiber stress and strain in the LV. Some other models are used to describe the material properties of the ventricular myocardium⁽⁷⁾. Paper⁽⁸⁾ provided a numerical simulation system based on FEM to study the mechanical properties of LV, it was integrated by four fundamental models: (1) a mechanical model (M-model) of myocardial muscle fiber which produced the active force, (2) a M-model of the LV which was composed of the model of the muscle fiber, (3) a transmission model of electric stimulus, (4) a circulatory system model which gave the after- and pre-loads to the LV model. This system was simple but practical, and some valuable results were obtained from it.

All the models mentioned above could describe different features of ventricular mechanics, while almost no one pay attention to the electric stimulus transmission which affect the myocardial mechanics comprehensively. E.g., the assumption in some models^{(5),(8)} that the electric stimulus transmits uniformly from the endocardium to the epicardium is simple, and it may hardly provide reasonable results on the computation of the active loads on the LV wall. Moreover, some models deals with the structure of LV simply, such as two dimensional regular cylindrical model and plane strain condition in model⁽⁸⁾, which probably confined at best to describing the mechanics of narrow equatorial cross-section of the LV wall.

In this paper, based on myocardial fiber structure and our heart electric model (E-model)⁽⁹⁾⁻⁽¹¹⁾ which can reproduce the transmission procedure of heart electric stimulus, we constructed 3-D LV composite FE M-model, and gave some preliminary simulation results.

2. 3-D Finite Element Model of Human Left Ventricle

2.1 Finite element discretization based on myofiber structure

The myocardium of the LV is a fibrous structure, the fibers being wound helically anticlockwise on the epicardium and changing progressively through the wall thickness to being wound helically clockwise on the endocardium. Then the muscle fiber direction may be quantified by the helix angle α_{helix} (Fig. 1), defined as the angle between the fiber direction and the local circumferential direction^{(5),(7),(12)}. The myocardium is a kind of optimal laminated composite material, and it has been evolved for thousands of years. It has complicated micromechanism and compound mechanism, and possesses total merits of the composite. It's difficult to analyze ventricular mechanics using general FEM⁽¹³⁾, so we refer ourselves to the

Fig. 1 The fibrous architecture of the ventricular myocardium

Fig. 2 The finite element mesh of the left ventricle

theory of laminated composite to study LV.

According to reference^{(7),(12)}, we can assume that the fiber layout varies linearly through the wall thickness from +60 degrees on the inner wall (epicardium), through zero, to -60 degrees on the outer wall (endocardium). The myocardium is considered as a composite composed of a number of layers of different directional, fiber-reinforced laminas. This assumption can well deal with the dependence of local LV wall mechanics on myocardial fiber orientation.

In this paper, according to CT cross-sections of LV along its long axis, we reconstruct the geometric shape of LV and constructed 3-D FE model based on theory of composite. We selected 3-D 8 nodes isoparametric element to be the basic element. There are totally 3-D 760 brick elements, 1 005 nodes and 3 015 degrees of freedom in the model. The FE meshes are shown in Fig. 2. We radially divide the wall of LV into 4 sections (from the endocardium to the epicardium), each section independently covers the scope of fiber angles of $[-60^\circ, -30^\circ]$, $[-30^\circ, 0^\circ]$, $[0^\circ, 30^\circ]$, $[30^\circ, 60^\circ]$.

It is known that the stiffness in the fiber direction is much greater than that of in the transverse direction, the along-fiber speed of electric stimulus transmission is also greater than that of other directions.

As far as mechanical properties are concerned, at any point in the myocardium the material may be considered as transversely isotropic. The transmural distribution of α_{helix} is an important determinant of the transmural distribution of ventricular wall stress. The amount of anisotropy in the fiber architecture can have a significant effect on the myocardial behavior.

Because LV is a composite structure with a very thick cross section, 2-D plate analyses are inadequate. 3-D FE analysis is necessary in order to calculate the stresses and strains accurately. Since material properties vary from layer to layer due to the change of fiber orientation, FE modeling for LV becomes extremely difficult and expensive. The normal 3-D FEMs (based on one layer per element) are not efficient enough for the analysis⁽¹³⁾. Hence, a simple but sufficiently accurate FE analysis is needed for the analysis of LV.

Based on paper⁽¹³⁾, we proposed an isoparametric method to discrete the LV. The major feature of the proposed analysis is that each element in the analysis can accommodate several layers having different ply orientations. A 3-D FE program was developed during the investigation for calculating the stresses, strains, and deformations of LV. When discretizing the LV into finite elements in radial direction, we group 10 layers per element, and it may be legitimate to assume that all the fibers within a layer are in the same orientation. This method can provide relatively accurate solutions using less degrees of freedom than conventional FEMs one layer for each element, and it saves in computer memory and computational time. Such computational efficiencies become significant for large-scale problems such as modeling the heart.

2.2 The load of active force

The heart is a neuroelectrically actuated mechanical pump. The electrical potential generation and conduction properties govern the myocardial mechanical constitutive properties and the chamber wall motion. The regular movement of LV is initiated by electric stimulus from the pacemaker (sinoatrial node). Before mechanical contraction, the electric excitation is generated and transmitted throughout the heart wall via special transmission system. When to study contraction force caused by electric stimulus, researchers generally simplified the transmission pattern of depolarization wave and considered it traveled uniformly from endocardium to epicardium^{(5),(6),(8)}. But according to heart electrophysiology, the electrical transmission progress or pattern is heterogeneous, which means that such approximation above may be simple.

Based on the electrophysiological property and anatomical structure of the heart, we construct heart

E-model⁽⁹⁾⁻⁽¹¹⁾ which can really reflects the transmission mechanics of bioelectricity. The excitement series of heart is simulated using a heart-torso model with realistic geometry. Using computer tomography (CT) pictures of the human body and heart sections, the heart E-model was reconstructed as a 3-D array of approximately 65 000 cell units spaced 1.5 mm apart. An action potential waveform with variable in duration is assigned to each unit. One layer of Purkinje fibers was sprinkled in the innermost endocardial surfaces of the ventricles, and four simultaneous starting units (the earliest excited units in the ventricles, which assumed to be the junctions at the terminations of the left and right bundles, one in the right ventricle and three in the left) were set in the endocardial surfaces. The right ventricle starting unit was set at the right low septum, and the LV starting units were set in the right low septum, and the LV starting units were set at the left midseptum, the left low anterior, and the left posterior, respectively. The distribution of the purkinje fibers and the four starting units were adjusted repeatedly until the excitation sequence of the E-model resembles the pattern reported by Durrer⁽¹¹⁾. The conducting velocity of the Purkinje fiber was assumed to be five times faster than that of the normal ventricular cells. In the simulation, the action potential waveforms with different duration can be set arbitrarily to each unit.

Cardiac field simulation is one of the frontier subjects in the electrocardiogram study, and the simulation of excitation propagation is the key to the simulation of the cardiac field. The excitation propagation algorithm we used is a hybrid of the rule-based algorithm and the wavefront-based algorithm, and has been described in detail in published papers^{(9),(11)}. In this propagation algorithm the initial excitation time (i.e. the time when a unit is excited by the adjacent excited unit) of all myocardium units is determined first. For each unit, the time elapsed since its excitation is obtained by subtracting the initial excitation time of the unit from the current simulation time. The excitation wavefront of a simulation step is a set of myocardium units that are excited in a certain time interval. The simulation step time is set at the smallest excitation propagation time between an adjacent node pair. It is proved that this algorithm is more accurate and efficient compared with either the rule-based algorithm or the wavefront-based algorithm⁽¹¹⁾.

By means of simulation above, we reproduce the cardioelectrical excitation series (that is τ in Eq. (1)) on LV and right ventricle (RV) (see Fig. 3).

In Fig. 3, different color represents depolarization wave at different time, it describes cardioelectrical

Fig. 3 Transmission sequence of electric stimulus

excitation series (time lag) at different part of LV. Activated by different excitation series at different parts of LV, local myofibers will contract. According to the FE discretization, three degrees a layer in an element, and the active force vectors are in unidirection. After the excitation series determined, we can calculate the active forces per element at different time. 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.) in Fig. 1, the expression of the active force along the fiber^{(8),(14)} is:

$$\sigma'_e = \begin{cases} \sigma'_0 \sin\left(\frac{t-\tau}{T_e} \pi\right), & 0 \leq t \leq T_e \\ 0, & t > T_e \end{cases} \quad (1)$$

Here, t is the time of excitation; τ is the time lag of active stress, T_e is the period of cardioelectrical excitation. σ'_0 is active force of sarcomere which is the function of sarcomere length l_f , time t and contraction speed, fiber directions, etc.

In the model, load of cardiovascular system is simulated by Windkessel Model^{(8),(14)}.

2.3 Coordinate transformation and integral conversion

Because of the different directions of myocardial fibers, we have to transform coordinates from fiber coordinate system to global rectangular Cartesian coordinate system⁽¹⁵⁾. At first, we convert fiber coordinate system $N-C_0-F$ to surface-tangent-coordinate $N-C-L$ determined by circumferential (surface-tangent) vector and the surface normal vector, the transformation matrix T_1 is determined by the direction angle of myocardial fiber on every layer. Then, we convert $N-C-L$ to global coordinate system $X-Y-Z$ (the center is located in the centroid of the base of LV with the X axis pointing toward RV, and Z axis along the long axis (from the base to the apex of LV), the transformation matrix T_2 is determined by the direction cosine of these two coordinate systems. Then, the material property, stress, strain, loads and other variables in the fiber coordinate system can be transform-

Fig. 4 The coordinate transformation

ed into the global coordinate system.

Because isoparametric element is selected in the model, we have to convert global coordinate system $X-Y-Z$ to local coordinate system $\xi-\eta-\zeta$. We calculate nodal force equivalent to the active contractile force based on Eq. (1), and then come into isoparametric transformation:

$$\{F_f\}^e = - \sum_{l=1}^{L_e} \int_{\xi_{l-1}}^{\xi_l} \int_{-1}^1 \int_{-1}^1 [B]^T \times ((T_2 T_1) \{0, 0, \sigma'_e, 0, 0, 0\}^T) |J| d\xi d\eta d\zeta \quad (2)$$

In Eq. (2), $[B]$ is the matrix associated with the derivatives of the shape functions, l, L_e stand for the number and total number of layers in an element respectively, $T_2 T_1 \{0, 0, \sigma'_e, 0, 0, 0\}^T$ is the active force row vector that has been transformed into global 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 ; ξ^l and ξ^{l-1} represents individually the coordinate values along ξ direction on the front-side and back-side of layer l in an element. When $l=1$, $\xi^{l-1}=-1$, and when $l=L_e$, $\xi^l=1$ (see Fig. 5).

Moreover, the cardiovascular load, blood pressure is distributed load which is perpendicular to the endocardium, and its equivalent nodal force can be expressed as integral form with intensity of pressure perpendicular to ξ in the local coordinate system.

When Eq. (2) is integrated using Gaussian quadrature, the new transformation⁽¹⁵⁾ (see Fig. 5) is needed:

$$\begin{cases} \xi = -1 + \frac{1}{H} \left[-h_l(1-\xi_l) + 2 \sum_{i=1}^l h_i \right] \\ d\xi = \frac{h_l}{H} d\xi_l \end{cases} \quad (3)$$

here, H is the total thickness of the element, h_l is the thickness of layer l in an element. When the variable ξ is replaced by ξ_l , the value of ξ_l in a layer varies from -1 to 1 , so the upper and lower limit of integral is 1 and -1 individually, then we can process Gauss quadrature numerical integration as below:

Fig. 5 Transformation of coordinates from an element system to a layer system

$$\{F_f\}^e = - \sum_{l=1}^{L_e} \int_{-1}^1 \int_{-1}^1 \int_{-1}^1 [B]^T \times ((T_2 T_1) \{0, 0, \sigma'_e, 0, 0, 0\} \bar{T}) |J| \frac{h_l}{H} d\xi_l d\eta d\zeta \quad (4)$$

Myocardial diseases such as myocardial infarction can change the stiffness of myocardium, activation sequences and contraction forces. Since we could not obtain myocardial material properties from the in vivo heart exactly, if we want to simulate the myocardial diseases, perhaps we could divide each layer into several segments in the circumferential or long-axis direction in the same way of layer-division, so the gradual differences of material properties and active forces in different sites of the ventricular wall may be simulated by Eq.(5).

According to the principle of virtual work and isoparametric integration transformation, the stiffness matrix of the element is:

$$[k]^e = \sum_{l=1}^{L_e} \int_{\xi_{l-1}}^{\xi_l} \left(\sum_{m=1}^{M_e} \int_{\eta_{m-1}}^{\eta_m} \int_{-1}^1 [B]^T [C]_{lm} [B] |J| d\xi_l d\eta \right) d\zeta \quad (5)$$

here, $[C]_{lm}$ is elasticity matrix of layer l and segment m , m/M_e stand for the number/total numbers of segments in layer l in circumferential direction respectively. Here we consider normal LV, so we only deal with layer-transformation.

In the end, assembling the stiffness matrixes and load matrixes of all elements, neglecting the inertial effect⁽¹⁶⁾, and then we can get quasi-static equilibrium equation of overall structure at time t :

$$[K]\{\delta\} = \{F_f\} + \{F_p\} \quad (6)$$

Here, $[K]$ is the total stiffness matrix, $\{\delta\}$ is the volume vector of nodal displacement, $\{F_f\}$, $\{F_p\}$ are the total vectors of the active forces and distributed loads respectively.

The boundary conditions: Axial displacement of nodes in the basal plane and circumferential displacement of the nodes in the subendocardial basal ring are suppressed. The endocardial surface is loaded by the

intraventricular pressure. External loading on the epicardial surface is assumed to be zero during the whole cardiac cycle. According to the feature of forces and restraints applied on the LV, we select 'boundary spring constraint' on the suppressed nodes to simulate the in vivo muscle restraints. This boundary constraint can avoid the phenomena of stress concentration caused by the oversize restraint stiffness. According to boundary condition of displacement, we can get nodal displacements after solving the Eq.(6), then we can get the strain and stress of layer l . Using general method and basing on physical equation of laminated composite, we can get local strain and stress values on LV wall⁽¹⁷⁾.

3. Results and Discussion

Based on fundamental M-model mentioned above, we simulated the stress, strain and displacement responses under the action of active force at different time during systole. At first, on the Basis of myocardial physiological property as well as some references^{(8),(14)}, the material constants are employed as follows:

Young's modulus E_L : 50 kpa; E_T : 11 kpa; shear modulus G_{LT} : 10 kpa; Poisson's ratio: $\mu_{LT} = \mu_{TT} = 0.49$; Uniform thickness of single layer in an element: $h_l = H/L_e$; The initial value of $\sigma'_0 = 53$ kpa;

The heart muscles have mechanical or physiological properties such passive anisotropic, nonlinear viscoelastic properties, excitation-contraction coupling mechanism, etc. is obvious now^{(18),(19)}, but to simplify the FE analysis and focus on the relationship between the mechanical properties and the myofiber structure especially the active force, here we neglect some mechanical properties of the muscle and just introduce passive linear elastic (transversely isotropic).

In the simulation, the time incremental step of simulation is 7 ms; Activation period $T_e = 353$ ms.

The active force and material constants are adjusted slightly in order to get proper results, and the pre- and after-loads are also considered.

Figure 6(a) shows two different views of the LV deformation during systole, it is taken over three time sequences ($T_1 < T_2 < T_3$, which comes near end-diastole, middle-systole and end-systole, respectively). The upper is front view, the lower is the view from apex of LV. We can easily observe that the procedure of LV contraction is very complicated, it includes axial shortening, radial contraction, basal contraction and twisting at different degrees, among these deformations, the apical contraction is the strongest. Figure 7(a) depicts the twisting angles of the endocardium from apex to base during systole, as a whole we can learn that the torsion at apex is the greatest. The deformation of LV does not like that of thick-walled cylindrical tube that other model assumed⁽⁸⁾.

From Fig. 6(a) we can know that the volume of ventricle will be reduced due to the movement of heart wall, and the length of myocardium fiber will be reduced too. Figure 8 shows the maximum and minimum eigenvectors of the strain tensor for the epicardial nodes, the length and color of the vector encode the associated eigenvalue.

The stress distribution within cross section of LV

Fig. 6(a) The simulated deformation of LV during systole (model)

Fig. 6(b) The imaged deformation of LV during systole (tagged MR⁽²⁰⁾)

at time T_1 , T_2 , T_3 during systole is shown in Fig. 9. The color encodes stress value. From Fig. 9, we can also observe the situation of radial contraction of the ventricular wall. Figure 10 is the first main stress distribution near the end of systole. According to these panels, we can know that the stress distribution on the LV wall isn't uniform, it reduces from the endocardium to the epicardium, the stress on the apex and base of LV is much greater, and the greatest exists on the apex of endocardium.

Comparing our simulation results with the deformation results of LV obtained from MR tagging method⁽²⁰⁾, we find that the displacement field distribution based on our model is similar with the result of medical image analysis. E.g., if we compare Fig. 6(b) (adapted from Fig. 4 in Ref. (20), and time 2, time 3, time 4 are corresponding to T_1 , T_2 , T_3 respectively.) with Fig. 6(a) in this paper, we can see that the deformations of LV are similar, the amplitude and direction of major axis contraction, minor axis contraction, heart wall distortion are nearly in agreement. Having no real displacements of LV, we could not compare the simulated displacement with the true displacement quantitatively, the comparison here is just qualitative one. The simulated result should be

Fig. 7(a) The simulated twisting degrees of the endocardium during systole (model)

Fig. 7(b) The imaged twisting degrees of the endocardium during systole (tagged MRI⁽²⁰⁾)

Fig. 8 The distribution of strain on the epicardium during systole

Fig. 9 The first main stress distribution within cross section of LV during systole

verified further. Though such comparison is limited, it reflects some common points above between the simulated displacements and imaged ones. From the comparison between Fig. 7(b) (adapted from Fig. 7 in Ref.(20)) and Fig. 7(a), we can also get that the variation tendency of anti-clock twisting angle is nearly congruent, too. While we can see that the model can not depict the twisting condition on the base of LV now. The reason that our model can get reasonable displacement field is probably that we consider the spatial direction of myocardial fiber and the real geometric shape of LV. If we select 2-D model having regular shape such as cylinder and sphere, the difference of the result will be great. The active force, after-load and other forces bring about displacement field directly, so the active stimulus series is one of the guarantee of the displacement. On the other hand, this model may confirms the active stimulus transmission mechanics of the heart E-model. The M-model can provide much more information than the model in Ref.(20), we can get the data of stress, strain field of LV wall, and these data depict the local physiopathological situation of LV.

The model results show that the stress on the

Fig. 10 The first main stress distribution on the ventricular wall during systole (T_3)

apex is much more concentrated, and the stress on the endocardium is greater than that on the epicardium, these results are similar to those of many paper^{(3),(6),(20)}. The fact that the stress distribution is non-uniform may be caused by the complicated structure of the myocardium. Figures 9 and 10 demonstrate that the stress distributions are sensitive to the activation

series, the different part of LV is stimulated at different time, which causes different power of contraction, and the stress and strain field will be developed differently.

Although many researchers put forward the theory of uniform stress distribution on heart wall based on optimization theory^{(21),(22)}, our results indicate that, unlike in the midventricle region that has been studied most fully, there may be significant regional non-homogeneity of stress and strain in the normal LV associated with regional variations in shape and fiber angle. The distribution of ventricular wall stress is a principal factor governing myocardial energetics⁽³⁾, and there exists difference of structure and energy demand between the endocardium and the epicardium, the heterogeneity maybe the result of mechanical adaptation of LV. Hence, the hypothesis of uniform stress may need to be modified, the heterogeneity may confer some functional advantage on LV performance. In reality, the presence of residual stress has been demonstrated in the heart⁽³⁾, and is thought to play an important functional role by reducing endocardial stress concentrations. The real value of stress is not what we need now, we focus on the stress comparison relationship between different parts of LV. So the authenticity of the material constants given here may not be so important.

Now, in the image analysis of myocardial wall motion, strain tensor is an important index to myocardial function. In MRI (tagged)⁽²⁰⁾, if an adequate sampling of the myocardial displacements is obtained with tagged images, the full 3-D displacement field of the heart can be reconstructed from these images. From this displacement field, the strain tensor field over the myocardium can be calculated. Strain is quantity of interest because it can be used to quantify the amount of tissue contraction: e.g., one can compute the amount of "shortening" the muscle achieves along the muscle fiber direction, and the amount of "thickening" the muscle achieves in the direction perpendicular to the wall. Our simulation indicates that the maximum strain is the radial thickening direction (positive, red vectors), and the minimum one is roughly in the fiber direction (negative, blue vectors), these provide a precise metric for quantifying the magnitude and direction of strain in the heart muscle. These simulation results are similar to those from tagged MRI, and it coincide with the LV physiological phenomena of radial thickening and shortening of fibers during the systole. The verification of the simulated strain results is qualitative, it should be studied further, too.

As mentioned above, the presence of residual stress has been demonstrated in the heart, and it has

also been shown that viscoelasticity may give rise to residual stress in a biphasic model of the LV⁽²³⁾. Myocardial tissue growth, which produces residual stress was proved in the rat heart opening-angle experiments⁽²⁴⁾. The residual stress or residual strain may affect on the relationship between the isometric maximum active force and the length in the cardiac muscle because the stress-free state changes depending on the residual stress or residual strain. While in the basic M-model here, we introduce some simplified assumptions such as (1) passive linear elasticity, (2) regular fiber orientation in the ventricular wall, (3) active force change (Eqs.(1) and (4)) neglecting viscoelastic properties (stress relaxation or creep phenomenon), (5) the assumption that all the fibers within a layer are in the same orientation which reduces an accuracy of the model. The assumptions above lead some system errors, it can not describe some important mechanical features such as residual stress. These are the disadvantages of the M-model compared with some reported delicate M-models^{(4)-(6),(19),(21)}. To analyze conveniently, we introduce them, and it should be improved in future study. While different FE heart models described important different features of the biomechanics of LV depending on different assumptions (simplifications). As the analysis above, our model has some advantage yet. The model now focuses on the dependence of local LV mechanics on myofiber structure especially active stimulus series. As we know, the mechanical properties depend on these two factors highly. E.g., if we alter the fiber angle or active series, the stress, strain field of LV and the displacement of wall will change distinctly. Lots of models in the literature didn't consider the complicated structure of the myocardium^{(3),(8)}, and almost no model considered the active stimulus in such a comprehensive way. Moreover, the simplified transmission pattern may not reflect pathological status of the diseased heart. In the literature, there are almost no work dealing with the combination of the E-model and M-model of the heart, here the work is done something about it. But the research may be too simple in some aspects, it should be improved further. From the view of this point, our model has its own features. And we have done some simulation research about heart disease such as myocardium infarction based on our E-model and M-model. The results of the abnormal simulation studies will be presented in detail in a future paper.

Another important thing should be mentioned is that, in the basic M-model, myocardial fibers are assumed to lie in the epicardial tangent plane. Although this is reasonable throughout most of the ventricular wall⁽²¹⁾, significant transverse angles have

been observed at the apex and base of the heart and at papillary muscle insertions. Reference (21) showed that while some features of systolic mechanics are sensitive to the presence of a non-zero transverse fiber angle. In further study, the transverse fiber angle should be considered in the model.

All in all, the simulation results show that the contraction of LV is complicated, the cardiomechanics is sensitive to myocardial fibrous structure, LV geometric shape and active stimulus. Though the basic M-model is simple in some aspects, it reproduces the action of LV in some degree.

4. Conclusions

The basic composite 3-D FE model to simulate the mechanical properties and functions of the human LV has been constructed in this paper by combining real geometric shape, fiber angle, cardioelectric excitation series and cardiovascular loads. The selected isoparametric-element method can divide each element into many layers and segments avoiding the oversize error of using 2-D model. It may be helpful to simulate the myocardium pathology. Our model is merely an initial one and we make many simplified assumptions about myocardial mechanics such as myofiber constitutive equation and fiber angle distribution, but it has described the mechanical property of LV properly. If more delicate models were set up, the complicated performance of the LV could be reproduced in a more precise and reliable manner and many details of cardiomechanics might be found. It is anticipated that by combining a solution of stress from the computer model of LV with that of wall motion, better estimates of in vivo strain and stress can be obtained from imaging data. This has important clinical implications because the distribution of stress affects the performance of the heart.

Constructing organ models can be expected to improve the diagnostic techniques for heart diseases from subjective, empire and qualitative ones to objective, scientific and quantitative ones⁽⁸⁾. The advantage of using the heart simulation model with electrophysiological and mechanical properties to study the cardiac biomechanics is that it can set up various physiopathological model parameters optionally. It is much more convenient than animal experiment and human experiment. Simulation can quantify the mechanical status of LV, e.g., quantifying the regional myocardial stress and strain field to look for myocardial mechanical difference to provide the information in diagnosis and procedure for surgical and medical intervention of patients with heart diseases.

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