

Original Paper

## Estimation of Cardiac Functions by Numerical Method

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### Abstract

A numerical simulation system using the three-dimensional finite element method (3D-FEM) is established to reproduce the performance of the left ventricle during one cardiac cycle, which may ultimately provide useful information for medical diagnoses. The simulation system consists of a 3D-FEM mechanical model of the left ventricle based on four fundamental models, that is, (1) a mechanical model of myocardial muscle fiber which produces the active force, (2) a mechanical model of the left ventricle which is composed of the myocardial muscle fiber, (3) a transmission model of electric stimulus, and (4) a circulatory system model which gives the pre- and after-loads to the left ventricular model. In this paper, the fundamental system of the simulator is explained, and some typical examples of computational results obtained by this system are shown and discussed. The reliability of the simulator is examined by comparing some numerical results with the corresponding results obtained by medical imaging technique.

Key words :Biomechanics, Finite element method, Stress and strain analysis, Left ventricle, Hypertrophic cardiomyopathy, Pressure-volume relationship, MRtagging technique

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## 1. Introduction

It is extremely important to estimate quantitatively the mechanical functions of the left ventricle from a viewpoint of the medical diagnosis of the heart diseases. The global functions of the left ventricle are mostly estimated based on its pressure-volume relationship, but which is not always an appropriate index of its performance because the pressure-volume relationship is affected by a shape and a size of a heart. On the other hand, the wall stress and strain of the left ventricle can be a general contractility index independent of the ventricular shape and size. In order to estimate the above-mentioned ventricular functions, the authors have constructed a fundamental numerical simulation system based on the finite element ventricular model connected with both a blood circulation system model and a transmission system model of electric stimulus [1], [2], [3].

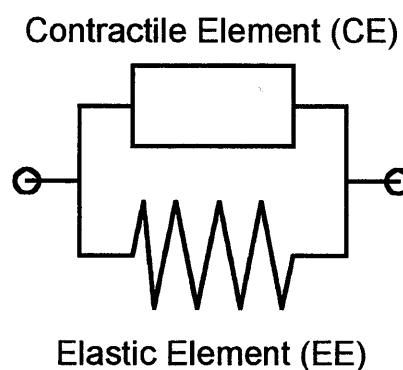
In this study, the stress and strain distributions and their time variations (in one cardiac cycle) in a human ventricular wall for both a normal heart and a heart with hypertrophic cardiomyopathy are analyzed by using the proposed numerical simulation system. The numerical results are compared with those obtained by medical imaging technique [4], [5].

## 2. Mathematical model of left ventricle

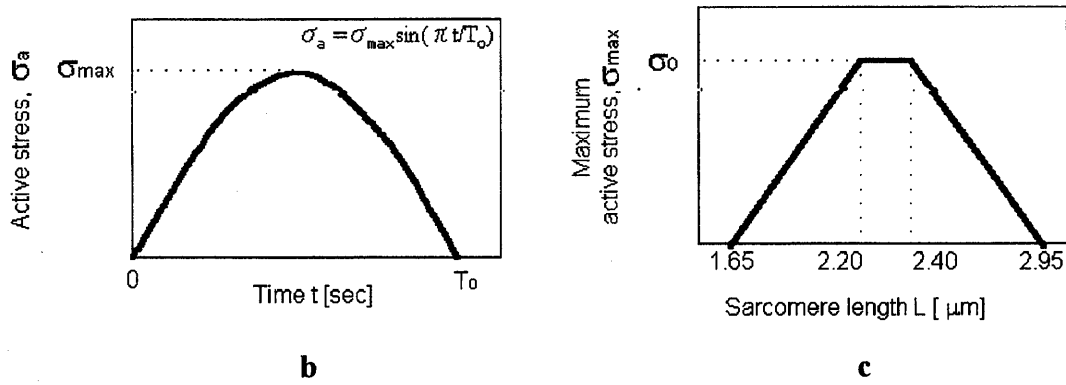
The mathematical models of left ventricle are composed of the mechanical model of muscle fiber, the electric stimulus transmission model and the circulation system model of blood. The installation of the fiber orientation into the finite element model is realized by transforming the standard element in the local coordinate system to the real element in the global coordinate system.

### 2.1 Mechanical model of myocardium

The myocardium consists of numerous contractile muscle fiber elements called "sarcomere", which produce an active tensile force and contract by themselves after receiving the electric stimulus sent from the so-called pacemaker of the heart [6]. The mechanical model of myocardium consists of both a contractile element and an elastic element (Fig.1a). The magnitude of active tensile force produced by the sarcomeres may be approximated through a set of simple relations proposed by Beyar and Sideman [7]. The relationships of the magnitude of active tensile force against time and sarcomere length are indicated in Figs. 1b and 1c, respectively.



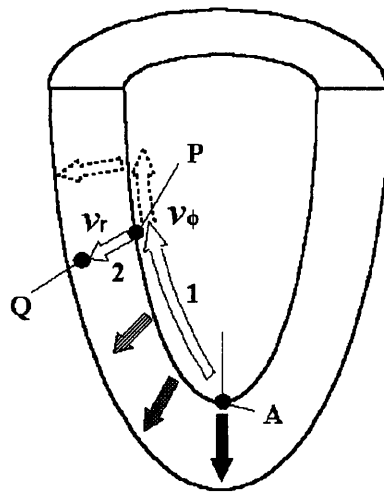
**Fig. 1 a** Muscle fiber model



**Fig. 1 b**  $\sigma_a$  is a nominal active tensile stress, i.e., the active force divided by the original cross-sectional area of the myocardium,  $T_0$  is the activation time of the myocardium and  $t$  is the time measured from the instance at which the electric stimulus arrives at the myocardium. **c** The maximum active stress  $\sigma_{\max}$  induced in the myocardium depends on the length of the sarcomere fiber  $L$ .  $\sigma_0$  is the maximum active stress

## 2.2 Electric stimulus model

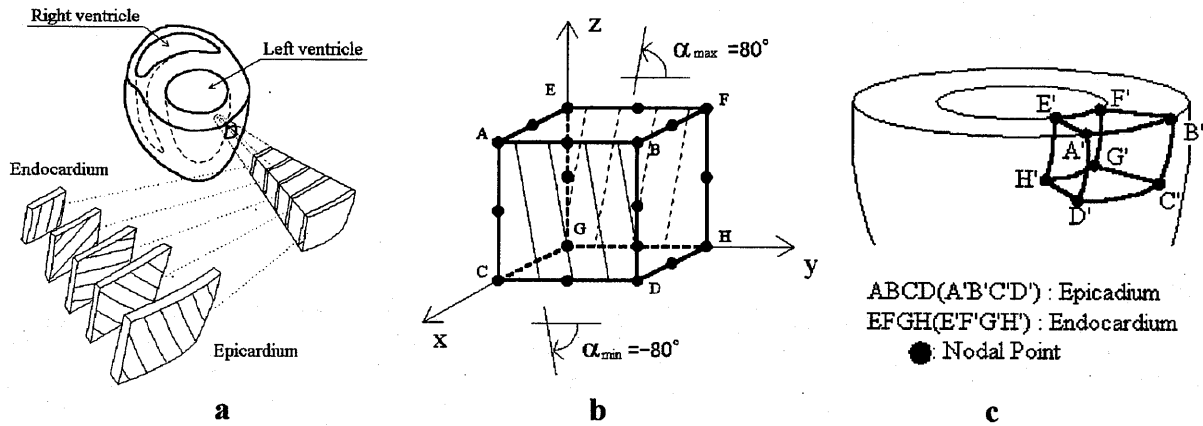
The electric stimulus launched from the pacemaker (sinoatrial node) transmits firstly to the apex through a ventricular septum and secondly to the basis through the Purkinje's fibers. Considering the characteristics of Purkinje's fiber, in the present mathematical model, it is appropriate to assume that the electric stimuli launched from the apex in the endocardium (a point A in Fig. 2) transmit firstly along the meridian of the endocardium (Arrow 1 in Fig. 2) and secondly in the thickness direction (Arrow 2 in Fig. 2) from an activated portion (for example, a point P in Fig. 2) to the epicardium.



**Fig. 2** Pathway of electric stimulus transmission, first along the meridian of the endocardium (arrow1, velocity  $v_\phi$ ) and then across the ventricular wall (arrow 2, velocity  $v_r$ )

### 2.3 Muscle fiber orientation

The ventricular wall is assumed to be an isotropically elastic material with myocardial muscle fibers whose orientations alter continuously from endocardium to epicardium as shown in Fig. 3a. The myocardial fiber angle is installed to the finite element model by the following manner. First, taking a standard cubic finite element A~H under the local coordinate system  $xyz$  as shown in Fig. 3b, the fiber angle in the element is assumed to be  $\alpha_{\max} = +80^\circ$  on the endocardium surface and  $\alpha_{\min} = -80^\circ$  on the epicardium surface, and interpolated linearly in the thickness direction. As a 4-points Gauss quadrature is employed in the present numerical analysis, there exist 64 Gaussian points in a finite element. The fiber angle is assigned at each Gaussian point according to the fiber orientation scheme described above. The final fiber orientation in the left ventricular wall is realized by transforming the standard element A~H in the local coordinate system to the element A'~H' in the global coordinate system (Fig. 3c).



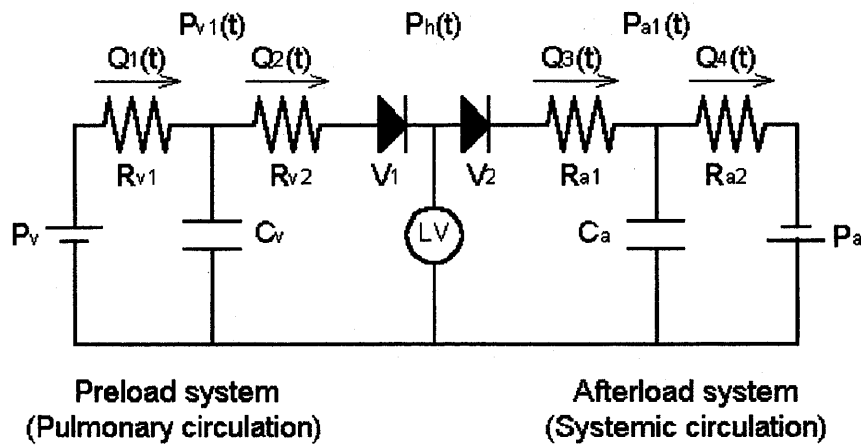
**Fig. 3** Modeling of myocardial muscle fiber orientation

### 2.4 Circulation system model of blood

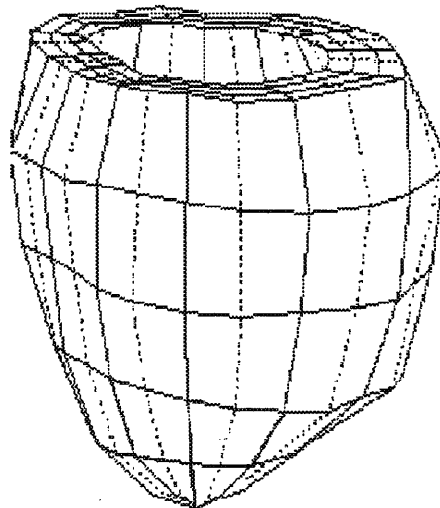
In order to concentrate on the estimation of mechanical properties and functions of the left ventricle, we employ the simplified electric circuit analogy model, which provides the proper pre- and after-loads to the left ventricle. Among several proposed models, we select here the electric circuit model constructed by Sekioka [8], shown in Fig. 4. The left-hand side of the circuit corresponds to the circulatory system model which produces the pre-load at the input side, including the effects of the left pulmonary vein, the left atrium and so on. The right-hand side corresponds to the circulatory system, which produces the after-load at the output side, including the effects of the aortic compliance, the peripheral vasculature and so on. In this electric circuit analogy model, the voltage and electric current correspond to the blood pressure and the blood flux, respectively, the resistances correspond to the vascular and valvular resistances for the blood flow, and the condensers correspond to the elastic properties of the vasculature. The battery indicates the averaged blood pressure to which the pre- and after-loads are subjected stationarily. In this circuit, the left ventricle is regarded as a kind of generator producing the fluctuating voltage (blood pressure) or fluctuating electric current (the blood flux).

### 3. Three dimensional construction of human left ventricle FEM model and numerical calculations

The three-dimensional geometry of the left ventricle is constructed from the cross sectional images acquired by the magnetic resonance imaging (MRI) technique. The finite element meshes of left ventricle are formed based on the above three-dimensional geometry. The isoparametric parallelepiped finite elements (198 elements in total) are arranged along the ventricular wall as shown in Fig. 5. The mechanical properties of myocardial muscle fiber and the other various material constants are not determined for a specific individual, but chosen on the basis of studies up to the present as well as the experience and knowledge of medical doctors. The initially employed material constants of myocardial muscle are corrected slightly by the trial-and-error technique. Various numerical parameters employed in the numerical calculations are indicated in table 1.



**Fig. 4** Circulatory system model. *LV*: left ventricle, *P<sub>v</sub>*: mean pulmonary arterial pressure, *R<sub>v1</sub>*: pulmonary venous vascular resistance, *R<sub>v2</sub>*: mitral valvular resistance, *C<sub>v</sub>*: pulmonary vein and left atrial compliances, *P<sub>a</sub>*: mean peripheral arterial pressure, *R<sub>a1</sub>*: characteristic impedance, *R<sub>a2</sub>*: peripheral vascular resistance, *C<sub>a</sub>*: aortic compliance



**Fig. 5** Finite element mesh for left ventricle

**Table 1** Cardiovascular parameters

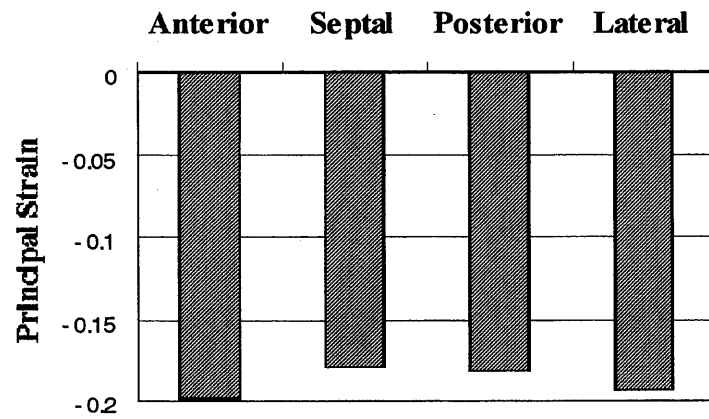
Mechanical properties of myocardial muscle	
Young's modulus	400mmHg
Poisson's ratio	0.49
Maximum active stress	800mmHg
Activation time	0.40sec
Initial sarcomere length	2.10 $\mu$ m
Transmission velocity of electric stimulus	
Meridian direction	5.00m/sec
Thickness direction	0.50m/sec
Parameters for circulatory system	
Mean pulmonary arterial pressure	5.00mmHg
Pulmonary venous vascular resistance	0.01 mmHg $\cdot$ sec/ml
Mitral valvular resistance	0.01 mmHg $\cdot$ sec/ml
Pulmonary vein and left atrial compliances	2.00ml/mmHg
Characteristic impedance	0.02 mmHg $\cdot$ sec/ml
Peripheral vascular resistance	1.20 mmHg $\cdot$ sec/ml
Aortic compliance	1.20ml/mmHg

#### 4. Numerical results and discussions

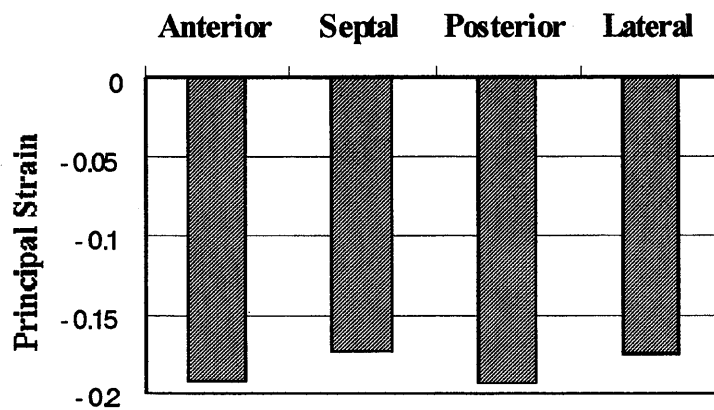
The dynamic changes and biomechanical properties are analyzed through a complete cardiac cycle. The reliability of the obtained numerical results, e.g., strain distributions, is verified through the comparison with the measurement obtained from the corresponding MR tagging [4], [5].

##### 4.1 Strain analysis in ventricular wall and pressure-volume relationship

The magnitudes of averaged minimum principal strains in the anterior, the posterior and the lateral walls for a normal heart are depicted in Figs. 6a and 6b. As recognized from these figures, the simulated results (Fig. 6b) may reproduce well the corresponding results obtained by tagged MR images (Fig. 6a).



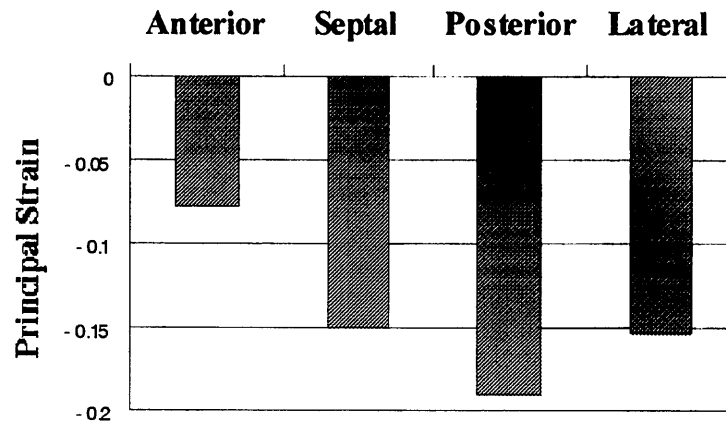
a



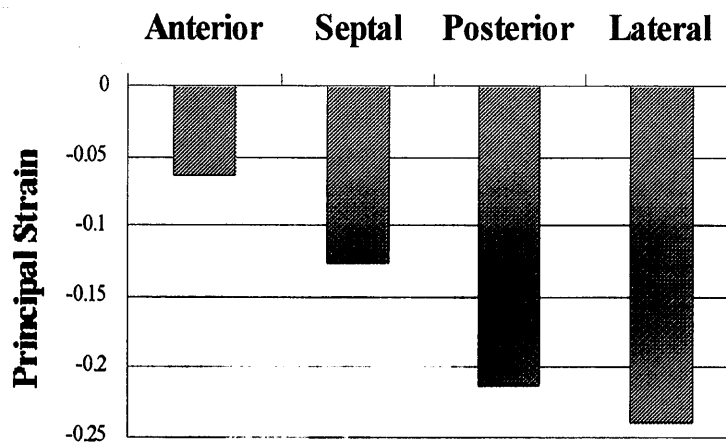
b

**Fig. 6** Magnitude of minimum principal strain for normal heart obtained by tagged MR images **a** and by FEM simulation **b**

Figures 7a and 7b show the magnitudes of averaged minimum principal strains in the anterior, the posterior and the lateral walls for a heart with hypertrophic cardiomyopathy (HCM). In this case, the diseased part exists in the anterior wall. From these figures, it can be seen that the behavior of the left ventricular wall obtained MR images (Fig. 7a) are reproduced by the numerical results obtained by the present simulation system (Fig. 7b). In this simulation, the magnitude of active force of myocardium is reduced by 50%. The pressure-volume relationships for a normal and a diseased heart are shown in Fig.8. It is recognized from the figure that the ejection fraction in a diseased heart is decreased compared with the one in a normal heart.

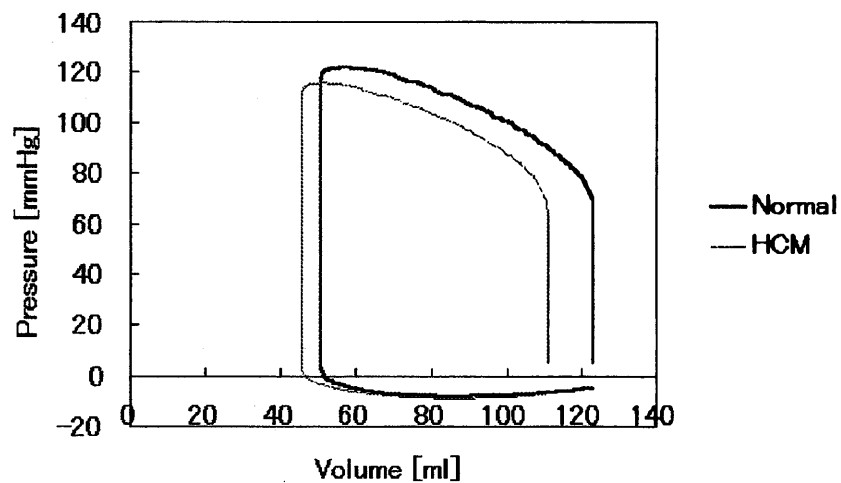


a



b

**Fig. 7** Magnitude of minimum principal strain for heart with HCM obtained by tagged MR images **a** and by FEM simulation **b**

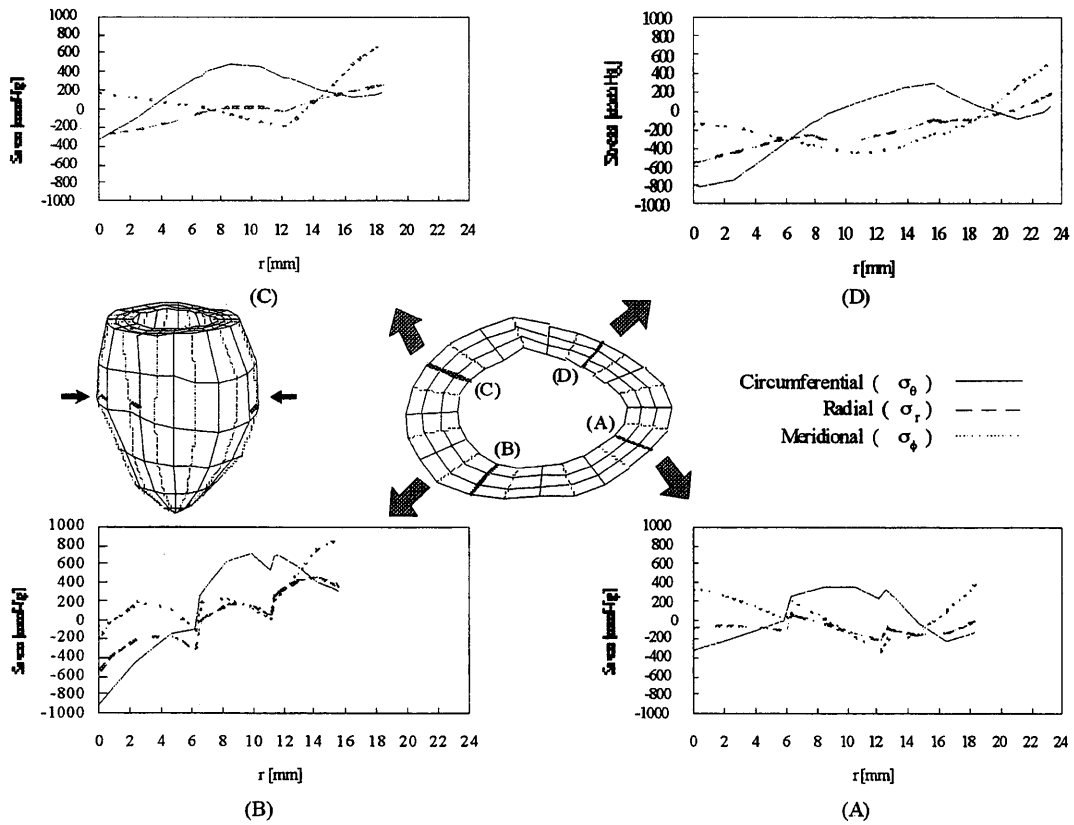


**Fig. 8** Pressure-volume relationships



## 4.2 Stress distribution in ventricular wall

The transverse wall stress distributions for a normal heart at the end-systole are depicted in Fig. 9. In this figure, the circumferential stress  $\sigma_\theta$  is extremely different from those of the thick cylinder subjected to the internal pressure. That is, the stress concentration does not occur on the inner surface and the maximum stress is observed near the middle part of ventricular wall. On the other hand, the magnitude of meridian stress  $\sigma_\phi$  becomes larger near both endocardium and epicardium unlike the distribution of circumferential stress  $\sigma_\theta$ . It is predicted that the above-mentioned complicated stress distribution in the ventricular wall arises from the complex distribution of myocardial muscle fiber orientation through the ventricular wall.



**Fig. 9** Transverse stress distribution in ventricular wall

## 5. Concluding remarks

The numerical system to analyze the mechanical properties and functions of the left ventricle has been constructed in this study by combining the mechanical model of the left ventricle with the circulatory system model. The proposed simulation system can reproduce the generally well-accepted properties and functions of the left ventricle, and also makes it possible

to estimate, for example, the stress distributions which are quite difficult to measure, while all of the basic elements of the system are very simple and fundamental. By substituting higher-grade models for the present fundamental models, the more complicated performance of the left ventricle could be reproduced in a more precise and reliable manner. Hence, in the future we can expect objective and quantitative diagnoses for heart diseases by improving the present numerical system.

There are still many problems to solve before we can realize the ideal simulation system. One of them is that there is a shortage of reliable *invivo* information about the mechanical properties of the bio-tissues and organs. And even if we could obtain such information, the characteristic complexity, the time dependence and the individual differences of organisms are turned to considerable barriers for the numerical simulation. However, it is expected that numerical simulators like those presented here will play a major role in overcoming these difficulties. In the authors' opinion, both the bio-measurement technique and the numerical simulation will be developed by compensating for each other in this research field.

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