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ON THE DYNAMICS OF THE LEFT VENTRICLE

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Abstract This paper is applying the cnoidal method to study the dynamics of the left ventricle. The left ventricle is a mixture of muscle and collagen fibers, coronary vessels, coronary blood and the interstitial fluid, so that its behavior results from a contractile motion of the muscle cells. The cnoidal method makes possible to use the theta function to describe the left ventricle motion as a superposition of cnoidal pulses.

Key words: Left ventricle, cnoidal method.

1. INTRODUCTION

The heart consists of the right and the left pumps which are connected in series to pump blood through the circulatory system. The left ventricle generates a high pressure (about 16 kPa) which is four times the pressure developed by the right ventricle [1]. The left ventricle has a thick-wall of myocardium between the epicardium and the endocardium.

The left ventricle exhibits a contractile motion of the muscle in the left ventricular wall due to the fibers located around the ventricle and their orientation relative to the circumferential direction. The orientation changes from about 60 at the endocardium to –60 at the epicardium.

The heart muscle is an anisotropic mixture of muscle and collagen fibers, coronary vessels, coronary blood and the interstitial fluid. This anisotropy influences the distribution of stresses in the wall [1-4]. The tissue is modeled as two-phase mixture, i.e. the solid and the fluid phases representing the different coronary microcirculatory components [5]. The parameters which define the constitutive equations are defined taking account of the nonlinear interaction between the responses to different loading schemes, the influence of the myofiber sheet architecture, the effects of transverse stresses in the myocytes and the relationship between the collagen fiber and the mechanical properties of tissues after myocardial infarction. The constitutive equations of the myocardial tissue are elaborated in [6]. In this paper, the cardiac tissue is modeled as a mixture of an incompressible solid and an incompressible fluid. The constitutive laws are specified within the framework of the intrinsic assumptions of the theory. The solutions are describable as a linear and nonlinear superposition of cnoidal pulses. The experimental results of Fung are used to determine the unknown parameters of the model by a genetic algorithm [7, 8, 11-13].

The dilaton concept is used to understand the mechanism of the strength of solids [14]. Theoretical investigations of residual strain in left ventricle are discussed in [15].

3. THEORY

In this section, the heart is modeled as a super ellipsoid surface defined by equations [16]

$$\left[\left(\frac{x}{a_1}\right)^{\frac{2}{c_2}}+\left(\frac{y}{a_2}\right)^{\frac{2}{c_2}}\right]^{\frac{c_2}{c_1}}+\left(\frac{z}{a_3}\right)^{\frac{2}{c_1}}=1, \qquad (1)$$

where $a_i, i=1,2,3$ and $c_i, i=1,2$ are known constants, ϕ_{helix} is the angle between the muscle fibre direction and the local circumferential direction, varying from 60^0 at the endocardium through 0^0 in the midwall layers to -60^0 at the epicardium, while ϕ_{trans} is kept zero, and ϕ_{trans} is the angle between the local circumferential direction and the projection of the fibre on the plane perpendicular to the local longitudinal direction, varying from 13.5^0 at the base through 0^0 at the equator to -13.5^0 at the apex (Fig. 1). The cylindrical coordinates are also represented in Fig. 1. The z-axis corresponds to the z-axis of inertia of the super ellipsoid model.
The muscle fibres in the ventricular wall are assumed to be parallel to the endocardial and epicardial surfaces. The cardiac muscle is considered to be a mixture of two phases, a solid phase and a fluid phase. The equations are derived from the general equations of the continuum theory of mixtures.

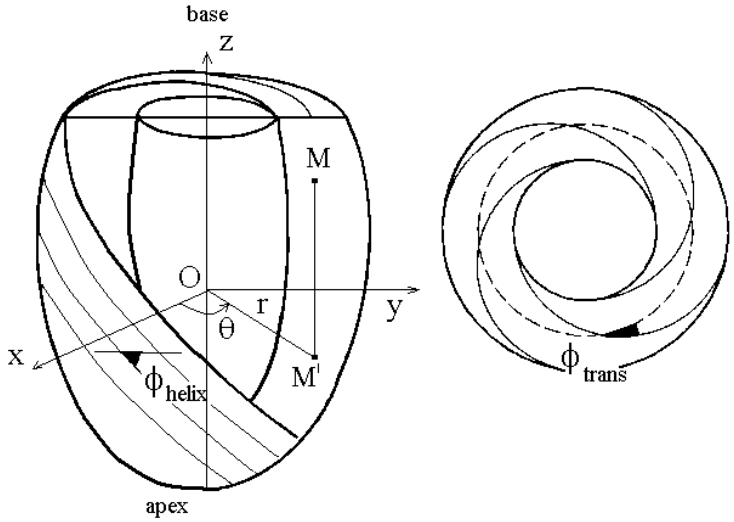

Fig.1. Cylindrical coordinates and definition of the angle ϕ_{helix} , ϕ_{trans} .

Let us to introduce the notations :

V actual volume of the heart,

$x=(r,\theta,z)$ spatial cylindrical (Eulerian) coordinates, centred in O , $\begin{pmatrix}x\\y\\z\end{pmatrix}\rightarrow\begin{pmatrix}r\\\theta\\z\end{pmatrix}$,

t time coordinate,

σ^s effective Cauchy stress in the solid representing the stress induced by the deformation in the absence of fluid and measured per unit bulk surface,

p intra-myocardial pressure representing the stress in the liquid component of the bi-phasic mixture,

$\sigma = \sigma^s - pI$ total Cauchy stress tensor in the mixture, $\sigma = \sigma^T$,

u displacement vector, $\begin{pmatrix} u_1 \\ u_2 \\ u_3 \end{pmatrix} \rightarrow \begin{pmatrix} u_r \\ u_\theta \\ u_z \end{pmatrix}$,

q Eulerian spatial fluid flow vector,

K^0 permeability tensor of the underformed tissue,

N^b averaged porosity of the underformed tissue,

c^c volumetric modulus of the empty solid matrix,

$H = \nabla u$ displacement gradient,

F deformation gradient tensor, $F = I + H$,

$J = \det \mathrm{F} > 0$ Jacobean of the deformation,

C isotropic energy function, $C = \frac{c^c}{2}(J-1)^2$, zero in the underformed state and positive elsewhere,

W strain energy function, zero in the underformed state and positive elsewhere,

K permeability tensor, $K = (\frac{J-1}{N^b} + 1)^2 K^0$,

E Green-Lagrange strain tensor, $E = \frac{1}{2}(F^T F - I)$,

$S(E,t)$ effective second Piola-Kirchhoff stress tensor, $S = JF^{-1}\sigma^s(F^{-1})^T$, $S = S^T$, split into two components $S = S^a + S^p$,

$S^a(E,t)$ active stress tensor,

$S^p(E,t)$ passive stress tensor, split into two components $S^p = S^c + S^s$,

$S^c(E)$ component of the passive stress tensor resulting from elastic volume change of the myocardial tissue, zero in the underformed state,

$S^s(E,t)$ component of the passive stress tensor resulting from viscoelastic shape change of the myocardial tissue,

$S^e(E)$ anisotropic (orthotropic) elastic response of the tissue. S^e is zero in the underformed state,

$G(t)$ a scalar relaxation function,

T^a first Piola-Kirchhoff active stress (not symmetric) related to the second Piola-Kirchhoff active stress by $S^a = F^{-1}T^a$,

l current sarcomere length,

v velocity of shortening of the sarcomeres $v = \frac{dl}{dt}$,

$\nabla = (\frac{\partial}{\partial r}, \frac{1}{r}\frac{\partial}{\partial \theta}, \frac{\partial}{\partial z})$ gradient operator with respect to the current configuration.

With this, the equilibrium equation of the deformed myocardicum is written for neglecting the inertia forces

$$\nabla\sigma^s - \nabla p = 0 \ . \tag{2}$$

The Darcy's law in Eulerian form (by neglecting the transmural pressure differences across blood vessel walls) is

$$q = -K\nabla p \,, \tag{3}$$

with $K = (\frac{J-1}{N^b}+1)^2 K^0$, the parameters K^0 and N^b being specified.

The continuity equation (conservation of mass) is written as

$$\nabla\dot{u} + \nabla q = 0 \,, \tag{4}$$

where dot means the material time derivative.

We add the pasive constitutive laws,as

$$S^c = \frac{\partial C}{\partial E} \,, \tag{5}$$

where

$$S = S^a + S^p \quad , \ S^p = S^c + S^s \ , \tag{6}$$

$$C = \frac{c^c}{2}(J-1)^2 \ , \ J = \det \mathrm{F} > 0 \,, \tag{7}$$

$$S(E,t) = JF^{-1}\sigma^s(F^{-1})^T \,, \ S = S^T \,, \tag{8}$$

$$E = \frac{1}{2}(F^T F - I) \,, \ F = I + H \,, \ H = \nabla u \ . \tag{9}$$

In (1-9), C is the isotropic energy function, E is the Green-Lagrange strain tensor, and $S(E,t)$ the effective second Piola-Kirchhoff stress tensor, split it into an active stress $S^a(E,t)$ and a passive stress $S^p(E,t)$. The passive stress tensor is split into a component resulting from elastic volume change of the myocardial tissue $S^c(E)$, and a component resulting from viscoelastic shape $S^s(E,t)$ described in the form of quasi-linear viscoelasticity [7, 8] as

$$S^s = \int_{-\infty}^{t} G(t-\tau)\frac{\mathrm{d}}{\mathrm{d}\tau}S^e \mathrm{d}\tau \ , \tag{10}$$

$$S^e = \frac{\partial W}{\partial E} \,, \tag{11}$$

where S^e is the anisotropic elastic response of the material, $G(t)$ is a scalar function (reduced relaxation function) derived from a continuous relaxation spectrum [2, 3], W is the potential energy of deformation per unit volume (or elastic potential). The full orthotropic behaviour is described in [3] while the transversely isotropic behaviour with respect to the fibre orientation is described in[6]. The expression of W is exponential depending of E_{ij} [7, 8].

In this paper, the pseudopotential energy approach is adopted. The energy W is given by taking into account of the ion-core (Born-Mayer) repulsive energy [9, 10]

$$W = \frac{0.5}{V}\int_V \alpha(\varphi)\exp[-\beta(\varphi)R]\mathrm{d}V\,, \tag{12}$$

where $\alpha(\varphi(x))$ is the repulsive energy function, $\beta(\varphi(x))$ the repulsive range function and V is the heart volume, and $R=\sqrt{(x-x_0)^2+(y-y_0)^2+(z-z_0)^2}$ with (x_0,y_0,z_0) an arbitrary point. We suppose that $\alpha(\varphi)$ and $\beta(\varphi)$ depend on the angles angle φ_{helix} φ_{trans} in the form

$$\alpha(\varphi)=\alpha_1(\varphi_{helix})\alpha_2(\varphi_{trans})\,,\ \beta(\varphi)=\beta_1(\varphi_{helix})\beta_2(\varphi_{trans})\ . \tag{13}$$

The active constitutive laws is written as [12]

$$T^a = T^{a0}A(t,l,v)\,, \tag{14}$$

where T^a is the first order Piola-Kirchhoff non-symmetric active stress tensor, related to the second Piola-Kirchhoff active stress by $S^a = F^{-1}T^a$, and T^{a0} is a constant associated with the load of maximum isometric stress.

The stress tensor T^a is convenient for some purposes; it is measured relative to the initial underformed configuration and can be determined experimentally. The cardiac muscle is striated across the fibre direction. The sarcomere length l (the distance between the striations) is used as a measure of fibre length. The experiments show that the active stress generated by cardiac muscle depends on time t, sarcomere length l and velocity of shortening of the sarcomeres $v=\frac{dl}{dt}$ [4]. The active stress generated by the sarcomeres is directed parallel to the fibre orientation. The function $A(t,l,v)$ represent the dependency on t, l and $v=\frac{dl}{dt}$. We suppose that $A(t,l,v)$ has the form

$$A(t,l,v) = f(t)g(l)h(v)\,. \tag{15}$$

The functions $A(t,l,v)$, $\alpha(\phi)$ and $\beta(\phi)$ are evaluated from experimental data [5,6] by using a genetic algorithm. The equations (1)-(14) represent a coupled set of four equations in $u_k(x,t)$, $k=1,2,3$ and $p(x,t)$ that can be written as

$$\nabla\dot{u}+\nabla(-K\nabla p)=0\,,$$
$$\nabla(J^{-1}F[S^a+S^c+\mathrm{S}^{\mathrm{s}}]F^T)-\nabla p=0\ , \tag{16}$$

with

$$K=(\frac{J-1}{N^b}+1)^2K^0\,,\ F=1+\nabla u\,,\ J=det\,F\,,$$
$$S^a=F^{-1}T^{a0}A(t,l^s,v^s)\,,\ S^c=\frac{\partial C}{\partial E}\,,\ S^s=\int_{-\infty}^{t}G(t-\tau)\frac{d}{d\tau}\frac{\partial W}{\partial E}d\tau\ , \tag{17}$$
$$C=\frac{c^c}{2}(J-1)^2\ ,\ E=\frac{1}{2}(F^TF-I)\,,\ W=\frac{0.5}{V}\int_V\alpha(\phi)\exp[-\beta(\phi)R]dV\ .$$

The initial conditions are given by

$$p(x,0)=p_0(x)\,,\ x\in\Sigma_1,\ k=1,2,3\,,\ t\in[0,T]\,,$$
$$u_k(x,0)=u_k^0\ ,\ x\in\Sigma_1,\ k=1,2,3\,,\ t\in[0,T]\,, \tag{18}$$

$$u_k(x,0) = 0 \ , \ x \in \Sigma_3 \subset \Sigma_1, \ k = 2,3, \ t \in [0,T],$$

where Σ_1 is the epicardial surface, and $[0,T]$ is the time interval during a cardiac cycle composed from a systole phase and a diastole phase.

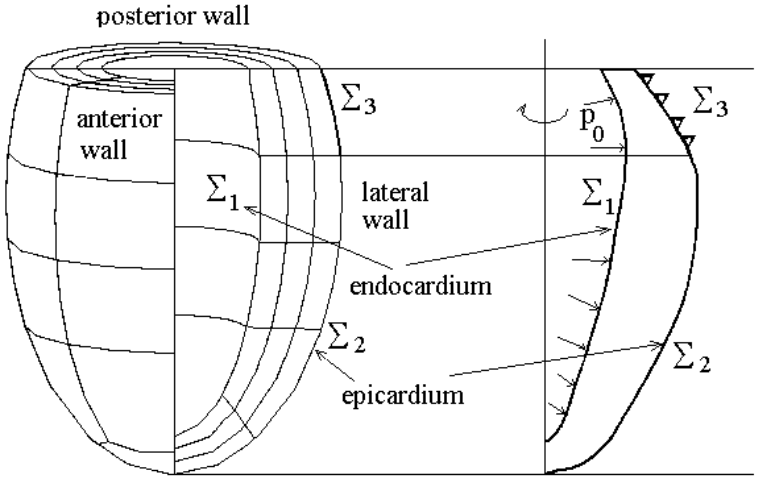

Fig. 2. Representations of endocardium, epicardium and the surface where only radial displacement is allowed.

The initial conditions are given by

$$p(x,0) = p_0(x), \ x \in \Sigma_1, \ k = 1,2,3, \ t \in [0,T],$$
$$u_k(x,0) = u_k^0 \ , \ x \in \Sigma_1, \ k = 1,2,3, \ t \in [0,T],$$
$$u_k(x,0) = 0 \ , \ x \in \Sigma_3 \subset \Sigma_1, \ k = 2,3, \ t \in [0,T],$$

where Σ_1 is the epicardial surface, and $[0,T]$ is the time interval during a cardiac cycle composed from a systole phase and a diastole phase. The systole is the contraction of the ventricle and the diastole is the relaxation of the ventricle. We have supposed that at the endocardial surface Σ_1 a uniform intraventricular pressure p_0 is applied as an external load on Σ_1(Fig.2). At the upper surface $\Sigma_3 \subset \Sigma_1$ of the heart only radial displacement u_1 is allowed.

The expression $\dfrac{\partial W}{\partial E}$ is computed from [9]

$$\frac{\partial}{\partial E_{ij}} = \frac{1}{2}(\mathrm{x}_i \frac{\partial}{\partial x_j} + x_j \frac{\partial}{\partial x_i}), \tag{19}$$

where x_i are the Eulerian coordinates ($x_1 \equiv x, x_2 \equiv y, x_3 \equiv z$). For specified form for $A(t,l,v)$, $\alpha(\phi)$ and $\beta(\phi)$ the analytical solutions of (14)-(16) are determined next. Equations (11)-(14) are solved analytically by the cnoidal method [16, 17]. For specified values for controlling parameters $P = \{ \alpha, \beta, a_r^f, a_r^g, a_r^h, \$ the analytical solutions $z_i(x,t)$, $i = 1,2,3,4$ are given by

$$z_i(x,t) = 2\sum_{m=1}^{2} \alpha_{mi} cn^2\{k_m(x - C_m t)\} + \frac{1 + \sum_{m=1}^{2} \gamma_{mi} cn^2\{k_m(x - C_m t)\}}{\sum_{m=1}^{2} \lambda_{mi} cn^2\{k_m(x - C_m t)\}} \ , \tag{20}$$

where α_{mi}, k_m, γ_{mi}, λ_{mi} and C_m, $i = 1,2,3,4$, $m = 1,2$ are 28 unknown constants evaluated by a genetic algorithm.

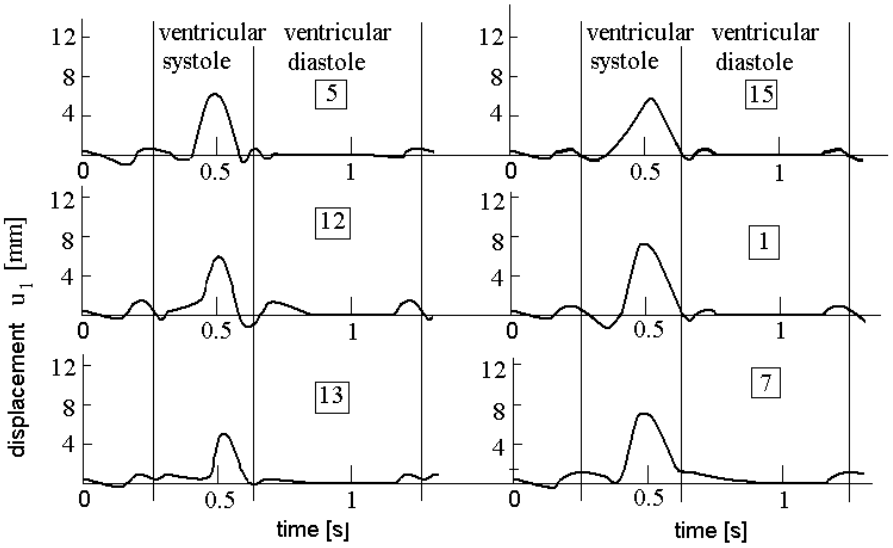

Fig. 3. Variation of the displacement u_1 during a cardiac cycle in selected points.

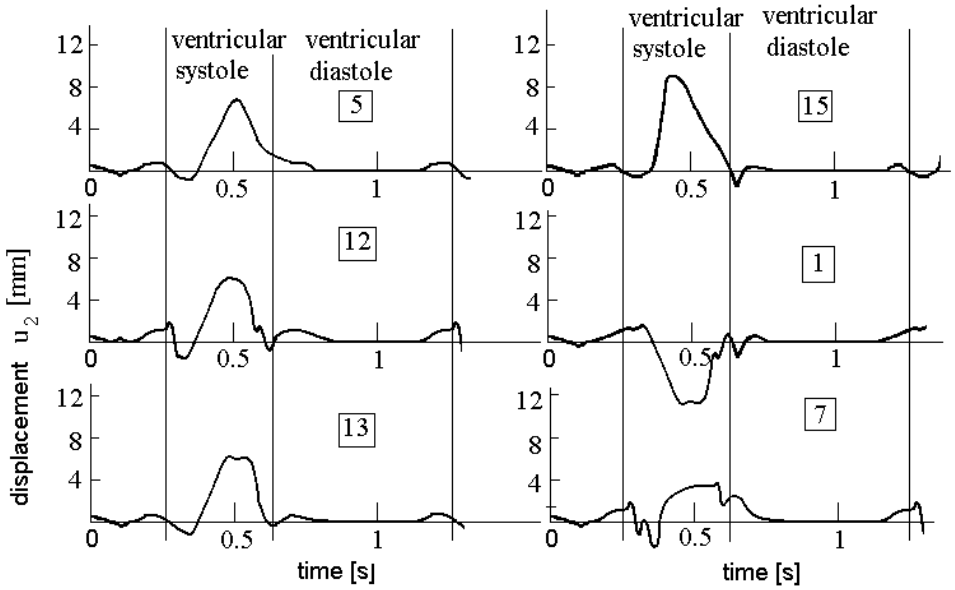

Fig. 4. Variation of displacement u_2 during a cardiac cycle in selected points.

The $\frac{\partial W}{\partial E}$ is computed for $p = 90$ points inside the volume V. In fig. 3 and fig. 4, the variation of displacements $u_k(x,t)$, $k = 1,2,3$ during a cardiac cycle calculated in selected points are presented.

3. CONCLUSION

In this paper, the cnoidal method is applied to describe the behavior of the left ventricle. The left ventricle is modeled as a mixture of muscle and collagen fibers, coronary vessels, coronary blood and the interstitial fluid, so that its behavior results from a contractile motion of the muscle cells. The cnoidal method makes possible to describe the left ventricle motion as a superposition of cnoidal pulses.

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