MECHANISM OF LEFT VENTRICULAR PRESSURE INCREASE DURING ISOVOLUMIC CONTRACTION, AND DETERMINATION OF ITS EQUIVALENT MYOCARDIAL FIBERS ORIENTATION

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The left ventricle (LV) is modelled as a fluid-filled, thick-walled finite-elasticity cylindrical shell, subject to internal pressure increase during isovolumic contraction. Our objective is to elucidate that the tremendous internal pressure build-up during isovolumic contraction is caused by stress development in the spirally-wound myocardial fibers due to their contraction. The LV model data consists of LV chamber pressure, LV dynamic geometry and LV twist angle. In our analysis, the LV chamber pressure increase and LV (radial, longitudinal, and twist) deformations are formatted to be caused by the contractile stresses in the LV myocardial fibers (based on the hyper-elastic constitutive property of the LV myocardial wall, expressed in terms of the strain energy density function). The LV wall stresses are expressed in terms of the strain energy density function, and hence in terms of the measured LV wall strains and the material parameters. Then, by satisfying the stress boundary conditions, from the measured data on LV deformation state and LV pressure, we first determine the LV wall’s constitutive properties, and then the instantaneous stress state in the LV.

The stress generated in the LV cylindrical model is equivalent to the development of active compression force and torsion within the model, as a mechanism for the high intra-LV cavity pressure build-up during isovolumic contraction. We in turn adopt (i) the principal compressive stress to be the stresses developed in the myocardial fibers (by

*Corresponding author.
their contraction), and (ii) the principal stress trajectory to correspond to the orientation of the myocardial fibers. The results show that the myocardial fiber orientation changes during the isovolumic phase, as the LV contracts.

Hence, an important determinant of our analysis is the orientation of the myocardial fibers. Conversely, it can be said that the fibers are so optimally oriented, that their contraction causes LV deformation to in turn cause the appropriate increase in intra-LV pressure. Another important outcome of our analysis is the determination of the “LV torque vs twist angle” relationship, which has the potential to be employed as an index of contractility.

**Keywords:** Left ventricular thick-walled finite-elasticity cylindrical model; isovolumic contraction; contractile wall stresses and strains; principal stresses; LV myocardial fibers’ orientation; LV torsion vs twist-angle contractile property.

1. Introduction

The left ventricular (LV) myocardial fibers are adroitly oriented such that their contraction causes compression and twisting, resulting in intra-LV pressure increase during isovolumic contraction. For many years, researchers have employed the famous law of Laplace for the determination of left ventricular (LV) wall stress. In particular, Sandler and Dodge approximated the left ventricle (LV) as an axisymmetric ellipsoidal shell and used the Laplace law to determine the stress and tension in the wall. However, the Laplace law is not suitable for determining the wall stress variation in thick-walled structures such as the LV. The main limitation of these models was their thin-walled representation that assumed the ventricular wall thickness to be small relative to the radii of curvature. With this constraint, variations of material properties across the wall thickness were ignored and the stresses were assumed to be constant across the wall thickness.

Wong and Rautaharju were among the first to come up with a thick-walled ventricular model. The wall stress was formulated with ellipsoidal LV shells, assumed to be isotropic, linearly elastic and compressible. Despite further simplification of the analysis using small-strain theory, they discovered nonlinear stress distributions through the wall thickness, an outcome that cannot be predicted by the Laplace law. It was Mirsky who first applied large deformation elasticity for the evaluation of LV stress. He discovered that the stresses at the endocardium using large strain theory were about ten times as high as the values predicted by linear small strain models.

Several researchers including Arts et al., Tözeren, and Guccione et al. have developed various cylindrical models to investigate the mechanics and material properties of the Azhari et al., recognizing that a cylindrical model is not adequate to describe the LV geometry and the non-uniformity of LV wall motion, constructed a conical LV model. Chaudhry et al. formulated a mathematical model based on a thick-walled truncated conical shell and incorporated the theory of large deformation elasticity. It was found that conical models lead to more realistic results than the spherical models, and can better evaluate stresses and strains from base to apex.

Noting the irregular LV geometry, Gould et al. formulated one of the first LV finite-element (FE) models, with axisymmetric geometry based on the longitudinal cross-section of the LV. In this analysis, they found that the irregularity
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of the LV geometry had considerable effects on wall stresses. Following this, many FE models of the LV have been constructed for the three-dimensional analysis of stress and strain distribution in the LV wall. In addition, the effects of anisotropy due to myofiber orientation on the ventricular wall stresses have also been examined. Several other studies have also shown that the architecture and orientation of myocardial fiber have significant effects on the mechanical properties of myocardium. Recently, Nash and Hunter formulated one of the finest FE models of the LV based on finite deformation elasticity. This anatomically representative model took into account the effects of muscle fiber orientation, to analyze the deformations of the heart during different phases of the cardiac cycle.

While all of this work has been oriented to LV wall stress and deformation, we believe that the mechanism of LV pressure generation during isovolumic contraction is of great significance to LV systolic function, LV contractility and LV ejection. It is this phase of early systole that sees the fastest rise in LV pressure. However, very few studies are dedicated to the analysis of mechanism of LV pressure rise and longitudinal and transverse changes in LV endocardium during isovolumic contraction and relaxation.

In this paper, we develop the analysis to determine the stresses developed in the LV myocardial fibers during isovolumic contraction from LV deformation, using large deformation theory. Although simple geometries cannot accurately represent the LV shape, we have employed a thick-walled cylindrical LV model, to reduce the mathematical intricacy and focus mainly on the effects of myofiber stresses on LV pressure development. In the process, we also obtain the constitutive properties of the myocardium and the relevant LV wall stresses, and most importantly the equivalent myofiber orientation responsible for the LV deformation.

Our model can lend insight into how the myofiber orientation and the stress developed in the myofibers can cause the observed rapid LV pressure rise and the LV deformation during isovolumic contraction. At this stage, this is intended to be a theoretical mechanistic model. Later on, when we acquire the technological capability to monitor the complex LV deformation (radii, axial and twist in the cylindrical coordinate frame) along with LV pressure, this model can be employed to determine the equivalent myofiber orientation (EMFO), along with the LV myofiber stress state. We deem this LV EMFO to be an intrinsic property of the LV, which governs how effectively the LV can develop pressure to effect adequate stroke volume ejection. In this regard, EMFO could well come to be regarded as a LV diagnostic property characterizing LV contraction.

The left ventricular (LV) myocardial wall is made up of helically oriented fibers. As the bioelectrical wave propagates along these fibers, it causes concomitant contraction wave propagation. The contraction of the helical oriented myocardial fibers causes compression and twisting of the LV, resulting in increase of its internal pressure. Herein, we simulate this phenomenon of LV isovolumic contraction (which causes the intra-LV pressure to rise so fast during 0.04–0.06 seconds of isovolumic contraction) by means of a finite-elasticity analysis of the thick-walled LV cylindrical shell under incremental pressure increase. We have determined that the LV
pressure generated during isovolumic contraction as well as the LV torsion and twist-angle are caused by the stress state in the LV wall (equivalent to LV twisting and compression), due to contractile stress in the helically wound myocardial fibers (depicted in Fig. 1).

Based on experimental studies, myocardial fibers are found to be helically wound, at angle varying from $-60^\circ$ to $60^\circ$ across the LV wall.\textsuperscript{4–7} However, there is no way that this fiber orientation can be measured; nor can the LV deformation (for a cylindrical model of the LV) be precisely measured. Hence, in this paper, we determine the equivalent myofiber orientation averaged across the LV wall as well as the stress in the LV wall, from LV deformation and pressure increase. For this purpose, the stresses in the myocardial fibers are adopted to be the principal stresses in LV wall. Then from the orientation of the principal compressive stresses, we determine the myocardial fibers orientation during the isovolumic contraction phase. Conversely, it is intended to thereby indicate that the contractile stress developed in the myocardial fibers (of the specific equivalent orientation, averaged across the wall) causes LV pressure increase and LV deformation (including LV twist).

Although there has been some work on mechanics of active contraction of the heart muscle,\textsuperscript{8,9} it has not been hitherto shown that the development of principal stresses and the principal directions causes adroit compression and twisting of the LV, which in turn cause internal pressure increase. In this context, this work is pioneering in providing analytical demonstration of LV twisting and shortening (by the contraction of the helically-wound myocardial fibers) being associated with and causing the rise of LV pressure during isovolumic contraction. It has also been shown that the fiber angle changes during isovolumic contraction.

2. Methodology

We approximate the LV during isovolumic contraction as a fluid-filled, thick-walled cylindrical shell that is closed at both ends and constrained in the longitudinal
direction at the base (depicted in Fig. 2). We further consider that the LV wall behaves as an incompressible, transversely isotropic, hyperelastic material.

**Step 1:** Starting with monitored LV dynamic geometry and LV pressure increase, we determine the corresponding LV internal volume change, based on the bulk modulus of blood. From this volume change, we characterize the radial and longitudinal deformations of the LV cylindrical model. We also adopt a reasonable value of LV twist deformation (because the current technology does not permit its determination and also of the LV deformation due to change in its internal volume) during the isovolumic contraction phase.

**Step 2:** From these deformations, we determine the corresponding stretches, and therefore the strains in the LV wall, using finite-elasticity formulations.

**Step 3:** The LV myocardial constitutive property is expressed as the strain energy density function, in terms of the LV wall strains.

**Step 4:** Next, we express the LV wall stresses in terms of the strain energy density function, and hence in terms of the LV wall strains.

**Step 5:** We now impose the boundary conditions involving equilibrating the LV stress in the inner wall to the LV internal blood pressure. In these equations, the LV constitutive property parameters are unknown. By computationally solving these equations, we determine the best values of these myocardial constitutive property parameters.

**Step 6:** Once the LV constitutive property parameters are determined, we can go back to the expressions for the LV wall stresses and evaluate the LV wall stress state. We then continue on, to determine the principal stresses from the above-obtained stress state.
Step 7: We designate the relevant principal-stresses and accompanying principal stress directions to correspond to the LV myocardial fiber stress and orientations, respectively. By so doing, we can estimate the equivalent myocardial fiber-angle. The estimated fiber angle values correspond to the value obtained from experimental studies. Also, the computed principal stress magnitude is found to be in the same range as a typical isometric tension value, which lends credibility to our analysis.

Step 8: From the LV wall stress state, we can also determine the equivalent torsion \((T)\) and compression \((F)\) applied at the apical end of the cylindrical model (which is taken to be fixed at the base of upper end) by the active contraction of the myocardium. We can hence obtain the relationship between LV torsion and twist angle, which is a key determinant of the paper.

Although the paper presents a theoretical model for the mechanism of development of LV pressure, we have employed some relevant clinical data of a subject, in order to elucidate the model. The subject in this study was studied in a resting recumbent (baseline) state, after pre-medication with 100–500mg of sodium pentobarbital by retrograde aortic catheterization. Left ventricular chamber pressure was measured by a pigtail catheter and Statham P23Eb pressure transducer; the pressure was recorded during ventriculography. Angiography was performed by injecting 30–36ml of 75% sodium diatrizoate into the LV at 10 to 12ml/s. These findings are used to justify the use of single-plane cine-techniques, which allow for beat-to-beat analysis of the chamber dimensions.

For our study, monoplane cineangiocardiograms were recorded in a RAO 30° projection from a 9 inch image intensifier using 35 mm film at 50 frames/s using INTEGRIS Allura 9 system at the National Heart Centre (NHC), Singapore. Automated LV analysis was carried out to calculate LV volume and myocardial wall thickness. The LV data, derived from the cineangiographic films consists of measured volume and myocardial thickness of the chamber as well as the corresponding pressure. All measurements are corrected for geometric distortion due to the respective recording systems.

We model the LV as an incompressible thick-walled cylindrical shell, which is constrained in the long-axis direction at one end to represent the suspension of the LV by the aorta at its base. Considering the LV at end-diastole (ED) to be in the unloaded reference configuration, the cylindrical model in its undeformed state is represented geometrically in terms of cylindrical coordinates \((R, \Theta, Z)\) by

\[
R_i \leq R \leq R_o, \quad 0 \leq \Theta \leq 2\pi, \quad 0 \leq Z \leq L
\]  

(1)

where \(R_i, R_o, \) and \(L\) denote the inner and outer radii, and length of the undeformed LV model respectively. The LV model in its deformed state can then be defined in terms of cylindrical coordinates \((r, \theta, z)\) as

\[
r_i \leq r \leq r_o, \quad 0 \leq \theta \leq 2\pi, \quad 0 \leq z \leq l
\]  

(2)
where \( r_i, r_o \) and \( l \) denote the inner and outer radii, and length of the deformed cylindrical model respectively.

We further consider the incompressible LV model in its reference state to be subjected to twisting, radial and axial deformations in the radial and long-axis directions during isovolumic contraction, such that based on incompressibility criterion of \( \pi r(R^2 - R_i^2) L = \pi (r^2 - r_i^2) \lambda_z \cdot l \)

\[
r = \sqrt{\frac{R^2 - R_i^2}{\lambda_z} + r_i^2}, \quad \theta = \Theta + Z \frac{\Phi}{L}, \quad z = \lambda_z Z
\]

where \( \lambda_z \) is an axial stretch per unit unloaded length, and \( \phi \) is the angle of twist measured at the apex (relative to the base).

For the myocardium material, we assume a Fung-type exponential strain energy density function of the form

\[
\Psi = C[\exp(Q) - 1]/2
\]

where \( Q \) characterizes the material's transverse isotropy in the cylindrical polar coordinate system, and is given by

\[
Q = b_1 E_{\theta\theta}^2 + b_2 E_{ZZ}^2 + b_3 E_{RR}^2 + b_4 E_{\theta\theta} E_{ZZ} + b_5 E_{RR} E_{ZZ} + b_6 E_{RR} E_{ZZ} + b_7 E_{RZ}^2 + b_8 E_{\theta R}^2 + b_9 E_{\theta R}^2
\]

Herein, \( b_i \) \((i = 1, 2, \ldots, 9)\) are non-dimensional material parameters, and \( E_{i,j}, \) \((I,J = R, \Theta, Z)\) are components of the modified Green–Lagrange strain tensor referred to cylindrical polar coordinates \((R, \Theta, Z)\).

To reduce the mathematical complexity of the problem, we assume negligible transverse shear during isovolumic contraction. Thus \( E_{RZ} \) and \( E_{\theta R} \) in Eq. (5) and their corresponding stress components \((i.e. \sigma_{RZ} \text{ and } \sigma_{\theta R})\) are neglected in the subsequent analysis. The stress equilibrium relation (in the cylindrical coordinate system) is:

\[
\frac{d\sigma_{rr}}{dr} + \frac{(\sigma_{rr} - \sigma_{\theta \theta})}{r} = 0
\]

where \( \sigma_{rr} \) and \( \sigma_{\theta \theta} \) denote the radial and circumferential stresses respectively. The other two equilibrium equations (in the cylindrical coordinate system) are satisfied trivially, because the transverse shear stresses \((\sigma_{RZ} \text{ and } \sigma_{\theta R})\) are treated as zero.

We further consider the epicardial surface to be free of external pressure, while the endocardial is subject to the intra-cavity blood pressure. Therefore the boundary conditions on the outer and inner surfaces of the LV cylindrical model can be designated to be:

\[
\sigma_{rr}(r = r_o) = 0; \quad \sigma_{rr}(r = r_i) = -p
\]

where \( p_i \) denotes the internal or cavity blood pressure acting on the inner surface of the LV model.
By integrating Eq. (6), we obtain the Cauchy radial stress $\sigma_{rr}$ as
\[
\sigma_{rr}(\xi) = \int_{\xi_i}^{\xi_o} (\sigma_{rr} - \sigma_{\theta\theta}) \frac{dr}{r}, \quad r_i \leq \xi \leq r_o
\] (8)

Substituting this into the boundary condition equation (7), the internal pressure $p_i$, can be denoted as
\[
p_i = -\int_{r_i}^{r_o} (\sigma_{rr} - \sigma_{\theta\theta}) \frac{dr}{r}
\] (9)

Since the valves are closed during isovolumic contraction, we impose a set of boundary conditions at both the top and bottom of the internal LV surface, giving
\[
\sigma_{zz}\pi(r_o^2 - r_i^2) = p(\pi r_i^2)
\] (10)

where $\sigma_{zz}$ denotes the axial component of the Cauchy stresses.

The geometrical parameters of the LV cylindrical model are defined in Fig. 1. The volumes of myocardial wall ($V_M$) and of the LV are given as:
\[
V_M = \pi(R_o^2 - R_i^2)L = \pi(2R_i + h)hL
\] (11)
\[
V = \pi R_i^2L
\] (12)

wherein $V_M$ is myocardial volume, and $V$ is ventricular chamber volume, $R_o$ is outer radius and $R_i$ is inner radius, $h$ is the wall-thickness and $L$ is the length. Herein, the LV volume ($V$), wall thickness ($h$) and myocardial volume ($V_M$) are obtained by cineventriculography.

Using Eqs. (1) and (2), we can calculate the instantaneous radii $R_i(t)$ and length $L(t)$ (or any time instant $t$) in terms of the measured $V_M$, $V$ and $h$, as
\[
R_i = \frac{2Vh/V_M + \sqrt{(2Vh/V_M)^2 + 4Vh^2/V_M}}{2}
\] (13)
\[
L = V/\pi R_i^2
\] (14)

Then $R_o = R_i + h$.

We consider the blood in the LV cavity to be slightly compressible, based on the value of its bulk modulus ($K = 2.0 \times 10^9$ Pa). By doing so, we are allowing a small change in cavity volume as a result of the substantial pressure change during isovolumic contraction. The instantaneous change in cavity volume ($\Delta V$) can thus be expressed in terms of the instantaneous pressure change ($\Delta p$) and the bulk modulus ($K$) of blood, as
\[
\Delta V/V = \Delta p/K
\] (15)

From Eq. (15), we can calculate the sequence $\Delta V$, and hence $V (= V_j - \Delta V_j)$. For this change $\Delta V_j$ in the LV volume, by assuming the ratios of our LV cylindrical
model length and radius changes ($\Delta r_j$ and $\Delta l_j$) to be equal during isovolumic contraction, we obtain their expressions as:

$$
\Delta l = (1 - \sqrt{1 - \Delta p/K})L, \quad \Delta r_i = (1 - \sqrt{1 - \Delta p/K})R \tag{16}
$$

From Eq. (16), the incremental quantities $\Delta l_j$ and $\Delta r_{ij}$ can be calculated, and hence

$$
l_j = l'_j - \Delta l_j, \quad r_{ij} = r'_{ij} - \Delta r_{ij} \tag{17}
$$

where $l'_j$ and $r'_{ij}$ are the model length and radius of the previous step.

So the wall-thickness $h_j$ can be obtained from:

$$
h_j = \sqrt{\frac{V_M}{l_j} + \pi r_{ij}^2} - r_{ij} \tag{18}
$$

Let $\Delta \phi$ denote the relative angle of twist measured at the apex, at each of the 3 stages of isovolumic contraction phase, obtained by magnetic resonance imaging (MRI). Because at this stage, we are unable to simultaneously measure pressure and twist angle at the different stages of isovolumic contraction phase, we will adopt reasonable values of $\Delta \phi$ from literature $^{27,28}$ to be the twist angle.

Table 1 shows a sample data including pressure, volume and model parameters for the cylindrical LV model. The pressure was obtained by catheterization for different instants. The volume ($V$), myocardial volume ($V_M$) and wall thickness ($h$) was measured by ventriculography for the beginning of the isovolumic contraction ($t = 0$).

The cylindrical model radius $R_i$, $R_o$ and $L$ are calculated using Eqs. (13) and (14). Then the $\Delta V_j$, $\Delta r_{ij}$ and $\Delta l_j$ are calculated using Eqs. (15) and (16) for the instants ($t = 0.02, 0.04, 0.06$), hence the $V$, $r_{ij}$ and $l_j$ are obtained. The wall-thickness for the cylindrical model is calculated using Eq. (18). The outer radius $r_{oj}$ is then obtained ($= r_{ij} + h_j$).

We can now determine the stretches in the different directions as follows $^{29}$

$$
\lambda_z(R) = \frac{l}{L}, \quad \lambda_r(R) = \frac{\partial r}{\partial R} = \frac{r}{R}, \quad \lambda_\theta(R) = \frac{r \partial \theta}{\partial \Theta} = \frac{r}{R} \tag{19}
$$

where $l = L - \Delta l$ and $r_i = R - \Delta r_i$, noting that $\lambda_2 \lambda_1 \lambda_\theta = 1$.

On the other hand, the twist stretch due to torsion can be expressed as

$$
\lambda_\phi(r) = \frac{r \partial \theta}{\partial z} = \frac{r \Delta \phi}{l} \tag{20}
$$

The components of the Lagrange Green’s strain tensor ($E_{ij}$) can thus be expressed in terms of the stretches and deformations obtained from Eqs. (13) and (14) as $^{29}$:

$$
E_{rr} = \frac{1}{2}(\lambda_r^2 - 1), \quad E_{\theta \theta} = \frac{1}{2}(\lambda_\theta^2 - 1), \quad E_{zz} = \frac{1}{2}[(\lambda_z^2(1 + \gamma^2) - 1)], \quad E_{\theta z} = \frac{\gamma \lambda_z \lambda_\theta}{2} \tag{21}
$$
Table 1. Pressure-volume and model parameters for a sample subject with $V_M = 185$ ml.

<table>
<thead>
<tr>
<th>$t$ (second)</th>
<th>$P$ (mmHg)</th>
<th>$\Delta P$ (mmHg)</th>
<th>$V$ (ml)</th>
<th>$\Delta V$ (ml)</th>
<th>$r_{ij}$ (cm)</th>
<th>$\Delta r_{ij}$ (cm)</th>
<th>$l_j$ (cm)</th>
<th>$\Delta l_j$ (cm)</th>
<th>$h_j$ (cm)</th>
<th>$r_o j$ (cm)</th>
<th>$\Delta \Phi$ (°)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>18</td>
<td>1.36700000E</td>
<td>2.03208400E</td>
<td>1.053745000E</td>
<td>1.085247000E</td>
<td>3.117331000E</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.02</td>
<td>43</td>
<td>1.36699773E</td>
<td>2.27263750E</td>
<td>2.032083992E</td>
<td>8.46701669E</td>
<td>1.053744996E</td>
<td>4.39060418E</td>
<td>1.085246679E</td>
<td>3.117330670E</td>
<td>0.667</td>
<td></td>
</tr>
<tr>
<td>0.04</td>
<td>63</td>
<td>1.36699591E</td>
<td>4.09074750E</td>
<td>2.032083985E</td>
<td>1.52406301E</td>
<td>1.053744992E</td>
<td>7.90308757E</td>
<td>1.085246684E</td>
<td>3.117330669E</td>
<td>1.333</td>
<td></td>
</tr>
<tr>
<td>0.06</td>
<td>81</td>
<td>1.36699427E</td>
<td>5.72704650E</td>
<td>2.032083979E</td>
<td>2.13308822E</td>
<td>1.053744989E</td>
<td>1.10643226E</td>
<td>1.085246689E</td>
<td>3.117330667E</td>
<td>2.00</td>
<td></td>
</tr>
</tbody>
</table>

Table 2. The parameters of the strain energy function for the sample case shown in Table 1.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>$b_1$</th>
<th>$b_2$</th>
<th>$b_3$</th>
<th>$b_4$</th>
<th>$b_5$</th>
<th>$b_6$</th>
<th>$b_7$</th>
<th>$b_8$</th>
<th>$b_9$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Value</td>
<td>5946.2278</td>
<td>15690.58158</td>
<td>422.514993</td>
<td>16157.10454</td>
<td>16360.53744</td>
<td>33299.28998</td>
<td>680.7385218</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>
By expressing the Cauchy stress tensor in terms of the strain energy density function ($\psi$) described in Eq. (4), we get:

$$
\sigma_{\theta\theta} = \lambda_\theta^2 \frac{\partial \psi}{\partial E_{\theta\theta}} + 2\gamma \lambda_\theta \frac{\partial \psi}{\partial E_{\theta z}} + \gamma^2 \lambda_z^2 \frac{\partial \psi}{\partial E_{zz}} - \bar{p} 
$$

$$
\sigma_{rr} = \lambda_r^2 \frac{\partial \psi}{\partial E_{rr}} - \bar{p}, \quad \sigma_{zz} = \lambda_z^2 \lambda_\theta \frac{\partial \psi}{\partial E_{\theta z}} + \gamma \lambda_z^2 \frac{\partial \psi}{\partial E_{zz}}
$$

where $\bar{p}$ denotes the hydrostatic pressure, and $\psi$ is defined in Eqs. (4) and (5).

By employing these stress expressions, we are now in a position to satisfy the boundary conditions of the stress along the inner surface of the LV wall to be equal to the internal pressure in the LV. These boundaries are expressed by Eqs. (9) and (10).

By integrating Eq. (9) along with the boundary conditions described in Eq. (10), we can determine the material parameters $b_i$, ($i = 1, 2, \ldots, 9$) and $\bar{p}$, using a nonlinear least squares method. For the sample case shown in Table 1, the material parameters determined are shown later in Table 2 ($b_1 = 5946.2278$, $b_2 = 15690.58158$, $b_3 = 422.514993$, $b_4 = 16157.10454$, $b_5 = 16360.53744$, $b_6 = 33299.28998$, $b_7 = 680.7385218$, $b_8 = 0$, $b_9 = 0$).

We next determine (i) the stretches from Eqs. (21) and (22) shown in Table 3, and (ii) therefrom, the strains $E_{ij}$ from Eq. (23). By substituting the computed strain components ($E_{ij}$) and strain energy function ($\Psi$) into Eq. (22), we can then determine the LV wall stress components for the cylindrical LV model (as shown in Tables 4 and 5).

We further consider that the active contraction force developed by the myocardial fibers can be approximated by an equivalent torsion $\Delta T$ and axial compression $\Delta F$ applied at the apical end, such that

$$
\Delta F = 2\pi \int_{r_i}^{r_o} \sigma_{zz} r dr
$$

$$
\Delta T = 2\pi \int_{r_i}^{r_o} \sigma_{\theta z} r^2 dr
$$

The computed values of the axial compression and torque are depicted in Table 5.

<table>
<thead>
<tr>
<th>$t$</th>
<th>$\lambda_\theta$</th>
<th>$\lambda_r$</th>
<th>$\lambda_z$</th>
<th>$\lambda_\phi$</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0.9999999958</td>
<td>1.0000000008E+00</td>
<td>9.99999996E−01</td>
<td>1.28626948E−01</td>
</tr>
<tr>
<td>0.04</td>
<td>0.9999999925</td>
<td>1.000000015E+00</td>
<td>9.99999993E−01</td>
<td>2.56482520E−01</td>
</tr>
<tr>
<td>0.06</td>
<td>0.9999999895</td>
<td>1.000000021E+00</td>
<td>9.99999990E−01</td>
<td>3.85688000E−01</td>
</tr>
</tbody>
</table>
### Table 4. Radial stresses distributions along LV wall from endocardium to epicardium.

<table>
<thead>
<tr>
<th>t (second)</th>
<th>Endocardium</th>
<th>1.06E-01</th>
<th>2.30E-01</th>
<th>4.08E-01</th>
<th>6.20E-01</th>
<th>8.16E-01</th>
<th>1.72E-00</th>
<th>1.98E-00</th>
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### Table 5. Results for the sample subject at different instants.

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<tr>
<th>t (second)</th>
<th>Circumferential stress (Pa)</th>
<th>Axial stress (Pa)</th>
<th>Radial stress (Pa)</th>
<th>Shear stress (Pa)</th>
<th>Torque (Nm)</th>
<th>Principal stress-tension (Pa)</th>
<th>Principal stress-compression (Pa)</th>
<th>Principal shear stress (Pa)</th>
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Given the stress components $\sigma_{\theta z}$, $\sigma_{\theta \theta}$, and $\sigma_{zz}$, we can then compute the principal stresses $\sigma_1$, $\sigma_2$ the principal angle $\phi$

$$\sigma_{1,2} = \frac{\sigma_{zz} + \sigma_{\theta \theta}}{2} \pm \sqrt{\left(\frac{\sigma_{zz} - \sigma_{\theta \theta}}{2}\right)^2 + \sigma_{\theta z}^2}$$  \hfill (25) \hfill (26)

The computed principal stresses are also shown in Table 5.

3. Results

Using Tables 1 and 4, the variations of the induced equivalent active torque $\Delta T$ and axial compressive force $\Delta F$ during the isovolumic phase are calculated, and shown in Fig. 3. We hence inversely demonstrate that the big increment of internal pressure in LV cavity during the isovolumic phase is caused by the corresponding torque and axial force developed within the LV, due to contraction of the LV myocardial fibers.

The variations of the LV wall radial and circumferential stresses ($\sigma_{rr}$, $\sigma_{\theta \theta}$), as well as axial and shear stresses ($\sigma_{zz}$, $\sigma_{\theta z}$) are shown in Table 5 and Fig. 4. It can be seen that axial and shear stresses are much greater than the circumferential stress and the radial stress in the LV wall.

The radial stress distribution along the thickness of LV wall at different times is shown in Table 4 and Fig. 5. It can be seen that the radial stress distribution along the LV wall becomes linear with time, during the isovolumic phase. The radial stress has maximum magnitude at the endocardium (equal to the LV internal pressure) and is zero at the epicardium.

The time variations of the principal stress and the corresponding angle during the isovolumic phase are shown in Table 5 and Fig. 6. The notable result from Fig. 6 is that both the principal stresses and their orientation angle keep changing during the isovolumic phase. At the end of the isovolumic phase, the magnitude of the compression principal stress is around $1.75 \times 10^5$ Pa, which is in good agreement with the isometric tension value of $1.45 \times 10^5$ Pa achieved under maximal activation.\(^8\)

It is seen from Fig. 6, that the equivalent myocardial fiber orientation is $38^\circ$ at the start of isovolumic contraction, and becomes $33^\circ$ at the end of the isovolumic phase. In other words, the monitored internal pressure increase during isovolumic phase from 25 mmHg to 45 mmHg to 63 mmHg is attributed to the active contraction of the helically woven myocardial fibers from $38^\circ$ to $33^\circ$. It is noteworthy that the computed equivalent myocardial fiber orientation is in the range of the values determined experimentally.\(^{19-21}\)

The variations of principal stresses and the corresponding principal angle, along the LV wall thickness during isovolumic phase are shown in Fig. 7. The principal stresses (tension and compression) do not vary much from endocardium to epicardium. However, the principal angle increases slightly from endocardium to
Fig. 3. Variations of axial force and torque induced within in the LV as a function of time during isovolumic phase.
Fig. 4. Variations of stresses as a function of time during isovolumic phase.
epicardium during the isovolumic contraction phase. The relationships between the torque induced in the LV and the twist angle as well as between the principal stress angle and the twist angle are shown in Fig. 8.

4. Concluding Remarks and Discussions

This paper is meant to theoretically demonstrate that the LV pressure build-up is due to contraction of the LV spiral-wound myocardial fibers. It also enables us to provide a measure of the equivalent LV myocardial fiber orientation. In other words, what is implied is that if (for known pressure-rise and twist-angle) we can determine the LV deformation state, we can determine the LV myocardial fiber orientation and stresses that cause this deformed state. From the wall stress state, we can determine the principal stresses and their orientations. The flow chart in Fig. 9 summarizes the calculation of the principal stresses and their orientation.

We can thereafter postulate that this equivalent myocardial fiber orientation of the LV can be employed as an intrinsic property of the LV which governs its contractility of the LV. Taking into consideration that it is not possible to determine the LV wall fiber orientation, the determination of this equivalent fiber orientation is an important outcome of this work, because it can help to provide an important clue as to why some persons are not able to effectively raise their LV pressure and are more prone to impaired LV contractility.
Fig. 6. Variations of the principal stresses and the corresponding angle as a function of time during isovolumic phase.
Fig. 7. Variations of the principal stresses and the corresponding angle along wall thickness during isovolumic phase.
Mechanism of Left Ventricular Pressure Increase During Isovolumic Contraction

![Diagram](image)

Fig. 7. (Continued)

![Diagram](image)

Fig. 8. Relation between (a) torque induced in the LV and rotate twist angle, and (b) between principal stress angle and rotate twist angle during the isovolumic phase.
Fig. 8. (Continued)

Cineventriculography & Catheter pressure

End diastolic volume, Myocardial volume, wall thickness (h) & pressure during isovolumic contraction (Table 1)

Calculate volume change using Eq. (18) (Table 1)

Calculate deformation using Eqs. (19) and (20) (Table 1)

Calculate stretch using Eqs. (21) and (22) (Table 3)

Calculate strain using Eq. (23)

Calculate constitutive parameters using Eqs. (9) and (10) (Table 2)

Calculate stress using Eq. (24) (Table 4)

Calculate principle stress using Eq. (27) (Table 5)

Calculate equivalent torsion and axial compression force using Eqs. (25) and (26) (Table 5)

Calculate principle angle using Eq. (28) (Table 5)

Postulate principal stress orientation to be the equivalent myocardial fiber orientation

End diastolic volume, Myocardial volume, wall thickness (h) & pressure during isovolumic contraction (Table 1)

Boundary condition using Eqs. (9) and (10)

Express stress using Eq. (24)

Calculate stress using Eq. (24) (Table 4)

Calculate principle angle using Eq. (28) (Table 5)

Calculate equivalent torsion and axial compression force using Eqs. (25) and (26) (Table 5)

Postulate principal stress orientation to be the equivalent myocardial fiber orientation
We have shown that the incremental intra-LV pressures ($\Delta p_i$) and LV deformations during isovolumic contraction are associated with and result in the development within the LV of incremental torsion and axial compression of the LV, and incremental principal stresses. The compressive principal stress corresponds to the active contractile stress generated in the myocardial fibers, while the angle of the compressive principal stress corresponds to the myocardial fiber helical angle, which is in agreement with the experiment data on the fiber angle.

Conversely, we have been able to analytically demonstrate how active twist and shortening, caused by the contraction of the helically-wound and specifically-oriented myocardial fibers, result in the substantial increase in LV pressure during isovolumic contraction.

References