Finite element analysis of embedded blood vessel mechanics

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Finite Element Analysis
of Embedded Blood Vessel Mechanics

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A Thesis Submitted in Partial Fulfillment
of the
Requirements for the Degree of

MASTER OF SCIENCE
in
Mechanical Engineering
Rochester Institute of Technology
Rochester, New York
April 1989

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Acknowledgments

Completing the masters thesis has been the most interesting and challenging task of my academic career. It was not an undertaking I took on alone. I was fortunate enough to have the support and guidance of Dr. M.H. Kempski who I know went far beyond what is normally expected of a thesis adviser in assisting me to complete this piece of work. His patience and willingness to teach, will always be remembered.

I would like to express my thanks to the individuals who enhanced this research: Dr. H. Ghoneim, for his guidance and suggestions; Dr. J. S. Torok for the time and effort they put into reading and evaluating my work.

I wish to express my deepest gratitude to my parents Lin Che-Chih, Chen Pi-Ying for the invaluable guidance and moral support they have given through my education. They are truly the finest of parents.

Finally, I am indebted to brothers and sisters in Rochester Chinese Christian Church, U of R, Eastman school of music, and Campus Bible Study Fellowship. Their love and supervision made me grow up in spiritual life. I also have great opportunities getting close to God and servicing Him in my two-year oversea study.
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<th>Description</th>
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<tr>
<td>$Ai(A'),Aio(A)$</td>
<td>Deformed and undeformed area with normal vector in &quot;i&quot; direction</td>
</tr>
<tr>
<td>$a_1,a_2,a_3$</td>
<td>Orthogonal coordinate system</td>
</tr>
<tr>
<td>$B$</td>
<td>Displacement-strain transformation matrix</td>
</tr>
<tr>
<td>$BVP$</td>
<td>Blood vessel pressure</td>
</tr>
<tr>
<td>$C_1,C_2,C_3,C_4$</td>
<td>Mooney-Rivlin constitutive coefficients</td>
</tr>
<tr>
<td>$CBF$</td>
<td>Coronary blood flow</td>
</tr>
<tr>
<td>$\varepsilon_{ij}$</td>
<td>Green's strain tensor</td>
</tr>
<tr>
<td>$F$</td>
<td>Axial force exerted on the materials</td>
</tr>
<tr>
<td>$Fi$</td>
<td>Strain gradients</td>
</tr>
<tr>
<td>$fi$</td>
<td>Body forces</td>
</tr>
<tr>
<td>$g_i$</td>
<td>Covariant base vector of the strained body</td>
</tr>
<tr>
<td>$I_1,I_2,I_3$</td>
<td>Strain invariants</td>
</tr>
<tr>
<td>$K$</td>
<td>Stress concentration factor</td>
</tr>
<tr>
<td>$LVP$</td>
<td>Left ventricle pressure</td>
</tr>
<tr>
<td>$P$</td>
<td>Axial stresses</td>
</tr>
<tr>
<td>$p$</td>
<td>Driving pressure</td>
</tr>
<tr>
<td>$P, HP$</td>
<td>Hydrostatic pressure</td>
</tr>
<tr>
<td>$Pi$</td>
<td>Inflow pressure</td>
</tr>
<tr>
<td>$Po$</td>
<td>Outflow pressure</td>
</tr>
<tr>
<td>$Pc$</td>
<td>External pressure</td>
</tr>
<tr>
<td>$Q$</td>
<td>Coronary flow rate</td>
</tr>
<tr>
<td>$R$</td>
<td>Coronary resistance</td>
</tr>
<tr>
<td>$r$</td>
<td>Radius of the blood vessel</td>
</tr>
<tr>
<td>$S_{1,ij}$</td>
<td>The 1st Piola-Kirchhoff stress tensor</td>
</tr>
<tr>
<td>$S_{2,ij}$</td>
<td>The 2nd Piola-Kirchhoff stress tensor</td>
</tr>
<tr>
<td>$SRR$</td>
<td>Radial stresses</td>
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<tr>
<td>$STT$</td>
<td>Tangential stresses</td>
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<tr>
<td>$SZZ$</td>
<td>Axial stresses</td>
</tr>
<tr>
<td>$TRT$</td>
<td>In plane shear stresses</td>
</tr>
<tr>
<td>$TRZ,TTZ$</td>
<td>Out of plane shear stresses</td>
</tr>
<tr>
<td>$T_{ij}$</td>
<td>Engineering stress or the 1st Piola-Kirchhoff stress</td>
</tr>
<tr>
<td>$V_i,V_o$</td>
<td>Deformed and undeformed volume</td>
</tr>
<tr>
<td>$W$</td>
<td>Strain energy density function</td>
</tr>
<tr>
<td>$Y$</td>
<td>Young's Modulus</td>
</tr>
<tr>
<td>$\sigma_{ij}$</td>
<td>Cauchy stress tensor</td>
</tr>
<tr>
<td>$\lambda_i$</td>
<td>Stretch ratios</td>
</tr>
<tr>
<td>$\delta_{ij}$</td>
<td>Kronecker delta</td>
</tr>
<tr>
<td>$\nu_2$</td>
<td>The volume fraction of rubber</td>
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Abstract

Cardiac pump function is closely linked to myocardial oxygen supply during the cardiac cycle. Variations in cardiac output must be met with concurrent shifts in tissue blood flow if changing myocardial oxygen requirements are to be satisfied. Of particular importance is the adequate perfusion of the left ventricular myocardium. Experimental evidence indicates that some ninety percent of left ventricular oxygen demand is generated during systolic contraction of the myocardium. Paradoxically, blood flow measurements in the left coronary artery suggest minimal systolic perfusion. The asynchronous, phasic character of left coronary blood flow and myocardial oxygen demand has prompted much research into the time dependent origins of coronary flow impedance. Various (sometimes conflicting) theories have been proposed, all of which suggest tissue blood delivery is significantly influenced by mechanical interactions between the myocardium and its embedded vascular network. Such interactions become particularly acute during systole, where myocardial contraction produces deformations and stress contractions which influence embedded vessel patency, coronary flow impedance, and effective driving pressure.

The present study attempts to quantify the impact of various mechanical attributes on embedded blood vessel mechanics during the cardiac cycle. The influence of tissue nonlinear constitutive behavior, inhomogeneity, and vascular orientation and tethering within the myocardium are assessed numerically through the use of nonlinear finite element analysis. Particular emphasis is placed on the latter category, where small caliber microvessels may be uniformly attached or loosely tethered to the surrounding muscle tissue. The myocardium and vascular wall are modeled as Mooney-Rivlin materials as a first approximation to their nonlinear anisotropic constitutive behavior.

The results indicate that blood vessel lumenal distortion and tissue stress contours are significantly influenced by myocardial-vascular wall inhomogeneity and nonlinear constitutive behavior under physiologic loading conditions. Embedded vessel orientation and tethering within the myocardium significantly biases these data. The existence of "gel-like" interface strongly affects axial stress contours when vessels oriented along the muscle fibers.
1.1 Physiological Basis for Present Study

Overall human health depends in large part upon cardiac efficiency. All bodily functions are ultimately controlled by the supply of oxygen and nutrients from the circulatory system. With the modern incidence of heart disease, and rate of death due to cardiac insufficiency, attention must be given to the affect of disease states on cardiac performance.

The cardiovascular system includes the heart and blood vessels. The system functions to move blood between the body cells and organs of the integumentary, digestive, respiratory, and urinary systems, which communicate with the external environment. In performing this function, the heart acts as a pump that forces blood through the attached vascular network. The blood vessels, in turn, form a closed network system of ducts that transports blood and allows the exchange of gases, nutrients, and wastes between the blood and the interstitial fluid surrounding body cells (Figure 1.1.1) [8,9,73,74].

The heart is divided into four hollow chambers, two atria and two ventricles. It is functionally separable into right and left halves. The wall of the heart is composed of at least three functional layers: the outer layer or epicardium (protective outer covering), the middle layer or
myocardium (responsible for muscular contractions), and the inner layer or endocardium (protective inner lining of the chambers and valves)[8]. The connective tissue within the endocardium contains blood vessels and some specialized cardiac muscle fibers called Purkinje fibers. The myocardial muscle fibers are arranged in whorl-like patterns, so that when they are stimulated by Purkinje fibers, the ventricular walls contract with a twisting motion. This action squeezes or wrings the blood from the ventricular chambers and forces it into the arteries. The right ventricle pumps blood into the pulmonary artery and the left ventricle pumps blood into the aorta.

During a cardiac contraction-relaxation cycle, the pressure within the chambers rises and falls. The time course of coronary blood flow, for example, (as measured in the large coronary arteries) undergoes phasic shifts directly attributable to ventricular contraction. Figure 1.1.2 illustrates typical time histories of coronary flow and aortic pressure for a single cardiac cycle. Figure 1.1.3 depicts pressure changes in the left atrium and left ventricle during a cardiac cycle [8].

It is evident from Figure 1.1.2 that flow in the left coronary circulation is greatly reduced during systolic contraction of the heart. The majority of left coronary blood flow thus occurs during diastole. In contrast, phasic shifts in right coronary blood flow are less pronounced [11,101,121]; a consequence of the fact that peak right ventricular pressure is about one-sixth that occurring in the left ventricle (i.e.
typical peak pressure in the right and left ventricles are 20 and 120 mm Hg, respectively). From Figure 1.1.2, we also note that reversal of right coronary blood flow does not occur in early systole, and because of the lower systolic pressure in the right ventricle, systolic blood flow constitutes a greater proportion of total right coronary inflow. Recent research [121] also suggests that the systolic impediment to coronary flow is related to tissue pressure and is largely independent of fiber shortening. As a result, the present study were focus its attention on the study of left ventricular contraction and its influence on left coronary blood flow (CBF).

During systole, blood flow to the left myocardial wall is poorest due to extravascular compressive forces generated by the contracting muscle fibers. This action serves to distort embedded blood vessels of varying calibers thereby influency intramyocardial blood flow. Conversely, during ventricular relaxation (i.e., diastole), intramyocardial vessels are no longer compressed, and blood flow increases (Figure 1.1.2). Hence, approximately 70-90 present of left ventricular blood flow occurs during diastole [11,34,35,85,101,121].

The time dependent pressure and volume relations for the left ventricle can be divided into four phases: diastolic filling, isovolumic contraction, systolic ejection, and isovolumic relaxation [31,120]. During systolic ejection (i.e., the time during which the aortic valve is open), ventricular cavity pressure reaches its ultimate value. The distortional
affect of contracting myocardium and ventricular pressurization on embedded vessels, therefore, is greatest during this portion of the cardiac cycle.

We may assume that the left ventricle (LV) is a truncated ellipsoid of revolution in terms of simple model geometries [19,25,29,31,32,62,63,65,66]. Muscle fibers are oriented predominantly in the circumferential direction in the midwall and in the longitudinal direction in the subepicardium and subendocardium [51,76]. During myocardial contraction, the LV is often described functionally in terms of circumferential or longitudinal fiber shortening. Due to the force equilibrium on one slice of the wall, the pressure in the radial direction will induce hollow chamber compression [76]. A radial stress gradient, therefore, is created in the myocardium. The influence of extravascular compressive and tensile stress components may therefore impede blood flow in distensible intramyocardial blood vessels.

1.2 Myocardial Oxygen Demand

The heart is a complex structure preforming vital, and complex functions as mentioned above. Precise timing by the cardiac valves and tissue contraction allows the efficient coordination of these functions. The most important function, pumping oxygenated blood to the periferal
organs, is performed by the left ventricle of the heart. Not surprisingly, the LV has the thickest wall and highest oxygen consumption of any cardiac chambers [15,34]. Blood is supplied to the tissue of the LV by the left coronary artery, which branches to provide global distribution of blood to this portion of the heart [9].

Since the LV must beat continually to supply blood to the body, the myocardial cells require a constant supply of oxygenated blood. Cardiac efficiency may be measured by the oxygen supply and demand of the heart [9,15]. The volume of oxygen consumed by the heart is determined by the amount and the type of activity the heart performs. It can be increased with exercise, excessive calcium ions (hypercalcemia), and be decreased moderately under conditions such as hypotension and hypothermia (i.e. it provides a method to reduce oxygen consumption during heart surgery). Wall stress is more fundamentally related to oxygen consumption than is cavity pressure per se [35,p.27]. Since a Laplace relation (i.e., in the Laplace spherical model $S=Pr/(2t)$, where $S$ is the true wall stress, $r$ is the deformed inner radius and $t$ is the deformed thickness) [35,75] indicates that wall stress varies with the size of the chamber(radius of curvature) and, hence, myocardial oxygen consumption increases with ventricular volume. The transmural oxygen demand was found to be a function of the structure of the LV fibers, the electrical activation velocity, and the degree of the twist [34]. Oxygen consumption per beat is also a function of the reciprocal of the heart rate [33,35 p.29,37].
In the heart, the subendocardial layers consume more oxygen than the subepicardial layers at the beginning of contraction [34,80,81,118,119,121], and 91 percent of total myocardial oxygen consumption occurs during systole [34,35, p27]. Gamble's research [80] states that coronary venous oxygen saturation of blood in the subendocardial veins of the LV was significantly lower than in the subepicardial veins (33.8% vs. 51.7%). The global oxygen demand can be determined from pressure-volume-area (PVA) relationships which represent the total energy generated by the left ventricle (i.e., the time integral of systolic pressure)[34]. The peak axial (along-fiber) stress ratio between systole and diastole is about 200, and maximum circumferential stress at the equator is obtained in midwall rather than near the endocardium or epicardium during systole (i.e., load bearing fibers are oriented in the circumferential direction at midwall) [34,p.670].

Intramyocardial blood vessels which are fed by branches of the coronary arteries consist of many small arteries, arterioles, and a vast capillary bed embedded within myocardial tissues. Therefore, it is highly probable that these distensible blood vessels are deformed by extravascular compression during systole, possibly influencing systolic blood delivery to the tissue. Adequate mean coronary flow is preserved by elevated diastolic flow valves which serve to compensate for systolic flow deficiencies [9,33,34,10]. If, however, a branch of the coronary arterial network becomes abnormally constricted (stenosis) or obstructed by a thrombus or embolus, the myocardial cells it supplies may
experience a blood deficiency, (ischemia) resulting in a degraded contractile performance of these cells. Sometimes a portion of the heart dies because of severe ischemia, resulting in myocardial infarction, myocardial fibrillation (small regions of the myocardium contract and relax independently of all other areas), and a severely compromised cardiac performance.

 Increased oxygen consumption must be balanced by an increased blood flow to maintain adequate performance. A number of drugs [100] are available that enhance coronary perfusion, and they are used in patients with coronary artery disease to relieve angina pectoris, the chest pain associated with myocardial ischemia. The heart influences its blood supply by the squeezing effect of the contracting myocardium on the blood vessels. The complex interactions between LV mechanics and the oxygen demand are therefore studied by relating the left ventricular transmural oxygen demand to the myocardial structural and stress characteristics.

 Guyton [100] revealed that the rate of myocardial oxygen consumption is the major factor that determines coronary blood flow; where oxygen consumption is proportional to peak myocardial muscle tension. Strong mechanical stress gradients (and resultant coronary perfusion pressure gradients) generated in the LV wall may play an important role in the development of heart disease, specifically when combined with other (pathologic) factors which influence mean coronary
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blood flow[78,81,100]. In addition, localized mechanical stress concentrations near embedded blood vessels may augment local oxygen consumption due to localized areas of increased stress, thereby affecting cardiac performance even further.

1.3 Myocardial Oxygen Supply

Supplying oxygenated blood to the myocardium is accomplished via a specialized portion of the systemic circulatory system, as mentioned above, called the coronary circulation. Cardiac muscle is metabolically active tissue, whose capillary density is very high. Blood flow in the capillaries depends on various factors dictated by physical, intrinsic, and extrinsic physiologic mechanisms. Physical factors that affect coronary blood flow (CBF) include effective perfusion pressure, length and cross-sectional area of the vascular bed, extravascular pressure, and blood viscosity [37,11,81]. The other two factors relate to rhythmic oscillatory behavior which are caused by local blood vessel contraction and relaxation (vasoconstriction and vasodilation respectively) [35]. Coronary flow rate can be expressed as the following:
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\[ Q = \frac{\Delta P}{R} \quad (1.3.1) \]

Where

- \( Q \) = coronary flow rate
- \( \Delta P \) = driving pressure across the coronary vascular bed (arterial pressure, arterial minus venous)
- \( R \) = total coronary resistance
  \[ = R_1 + R_2 + R_3 \]
- \( R_1 \) = basal viscous resistance
- \( R_2 \) = autoregulatory resistance
- \( R_3 \) = compressive resistance

Here, \( R_1 \) is a relatively static factor and is ordinarily negligible in comparison to \( R_2 \) and \( R_3 \). Autoregulatory resistance, \( R_2 \) is normally 4 to 5 times larger than \( R_1 \) but requires several cardiac cycles to adjust. The magnitude of \( R_3 \) is greater in the subendocardium than in the subepicardium during systole[35,p.4]. If the extravascular compressive component is included, we may conclude that vasodilatory reserve is less in the subendocardium than in the subepicardium [81,p.41]. Vasodilatory reserve means the additional blood flow, above usual conditions, that can be obtained by adding a vasodilating drug[81,p.41] which effectively increases pre-capillary vessel caliber and decreases \( R_2 \).

The control of \( R_2 \) is of pivotal importance in the regulation of coronary flow and several feedback mechanisms exist to provide self control (or autoregulation) by the tissue itself. These include neurohumoral, myogenic, and metabolic feedback mechanisms.
Regarding neurohumoral mechanisms, vascular smooth muscle receives direct autonomic innervation, but the quantity of neural mechanisms in normal physiological states has been difficult to define. Most workers have considered them less important than metabolic mechanisms [11,35,37,81,101]. One of the possible mechanisms for regulating blood flow that has received considerable attention is the direct response of vascular smooth muscle to changes in transmural pressure. This myogenic hypothesis originated with Bayliss in 1902 [77] and states that decreases in transmural pressure (intravascular minus extravascular pressure) are considered to result in vasodilation and vice versa. This idea presupposes some type of tension-sensing mechanism which affects the caliber of the vessel lumen. Direct evidence in support of the myogenic hypothesis has been available only in certain tissues and in isolated arterioles [101].

The myogenic hypothesis has been criticized, however, on the basis that if steady flow is to exist when perfusion pressure is increased, it is necessary for the caliber of the vessel lumen to be less than it was prior to the elevation of pressure [79]. If, on the other hand, stretch is the stimulus which evokes the smooth muscle contraction then once the vessel contracts to its original size (following the initial stretch) the stimulus for further contraction is gone[37,101]. Driscoll et.al.[78] state that if autoregulation were myogenically determined, different ratios of pressure changes might produce different rates and degrees of autoregulation. Such a response was not found experimentally.
Of all factors considered to be involved in the control of $R_2$, metabolic factors play the largest role because there is strong local control of the coronary circulation with changes in myocardial metabolism [37,38,81,101]. Here biochemical compounds, such as lactic acid, CO$_2$ potassium, inorganic phosphate, osmolarity, low oxygen tension, prostaglanins, adenosine, and hydrogen ions influence vascular caliber[35,38]. When the metabolic rate of the tissue increases or when the local blood flow is inadequate to meet oxygen demand, vasodilator substances (noted above) accumulate and elicit a decrease in vascular smooth muscle tone which results in vascular dilatation. Conversely, if metabolic activity decreases or the local blood flow is above that metabolically required, a decrease in vasodilator compound concentration results, allowing vasoconstriction and a reduction in local blood flow. The concept of dilatory reserve is therefore elucidated. It means the additional blood flow above usual conditions, that can be obtained by the addition of a vasodilatory agent like adenosine.

Metabolic control of vascular resistance is predicated on basal vessel tone which is independent of central nervous system activity (i.e., the state of tension in an arterial wall)[11,p.119]. Burton[36] suggests that the high intrinsic tone of arterioles makes this level of the coronary vascular tree the most susceptible to collapse under the influence of myocardial forces.
In recent research, Feigl [81] has classified control of CBF into four categories: 1) aortic pressure influence, 2) myocardial extravascular compression, 3) myocardial metabolism, and 4) neural control. They are similar to the concept of total coronary resistance. In the second item, intramyocardial tissue stresses may be greater in the subendocardium than in the sub-epicardium during systole due to transmural gradients in myocardial stresses (see Section 2.4). Hence, vasodilator reserve in the subendocardium may be less than that found in the subepicardium [34,121]. Beyar's analysis [34] indicated that endocardial muscle fibers develop high stress at the beginning of systole which decreases toward end systole. Feigl [81] also suggested that the major extravascular component of coronary resistance may be the result of myocardial compression or squeeze as related to intra-myocardial pressure (i.e., the radial stress component in the ventricular wall).

Another concept that we need to introduce is the vascular waterfall mechanism whereby flow through a collapsible tube is regulated by the pressure surrounding the tube rather than outflow pressure [39,40]. The resistance of arterioles, capillaries and venules, may be influenced by such phenomena thereby affecting tissue blood delivery.

If external pressure (i.e., Pc) equals zero, and inflow pressure (i.e., Pi) is greater than outflow pressure (i.e., Po), fluid flow will be governed by the difference in inflow and outflow pressure (i.e., Pi-Po) [11,100,101]. However, if we have a collapsible tube subjected to a surrounding
pressure, Pc, positioned somewhere between inflow and outflow (Figure 1.3.1) locations, flow through the tube may be governed by the difference in inflow and surrounding pressure if the tube is partially collapsed (i.e., Pi-Pc). If Pc>Pi then flow rate diminishes to zero [41]. For coronary circulation, we may assume that Pi= aortic pressure, Po= right arterial pressure, and Pc= effective intramyocardial compressive stresses. If Pi>Pc, the vascular bed will remain open.

Extending the vascular waterfall concept to the study of coronary blood flow, we note that the transmural intramyocardial pressure could be used to predict the degree of systolic coronary blood flow [41]. A number of authors have also shown that subendocardial intramyocardial pressures may exceed intraventricular pressure [53,54,55,56]. Armour and Randall [42] also stated that the intramyocardial pressure in the sub-endocardium is about two times as that in the sub-epicardium. Further research [39,40] has suggested that once intramyocardial vessels begin to collapse, blood flow becomes dependent on extravascular pressure instead of venous pressure in accord with the vascular waterfall hypothesis (or an equivalent three dimensional intramyocardial stress field). The determination of an intramyocardial pressure, therefore, plays a very important role in determining local coronary blood flow during the cardiac cycle.
1.4 Summary

Cardiac pump function is closely linked to myocardial oxygen requirements during the cardiac cycle. In the normal heart it is clear that the coronary vascular bed has sufficient reserve in general to supply enough oxygen to meet even extraordinary demands made by the organism. During the cardiac cycle, coronary blood vessels within the myocardium experience a wide range of external forces due to the effects of cardiac muscle contraction. The strong stress gradients generated in LV wall may play an important role in the development of heart disease, such as the reduction of coronary blood flow rate. Lesions (i.e., ischemia, myocardial infarction, necrosis) caused by deficiencies in local coronary blood flow have a marked effect on LV performance and tissue viability.

In the following chapters, we shall elaborate on the physical and the mechanical properties of cardiac muscle and blood vessels. Emphasis shall be placed on the prediction or measurement of intramyocardial stresses which mainly affect intramyocardial blood flow during the cardiac cycle. A selection of materials and a finite element model of embedded blood vessel mechanics are subsequently introduced.
The right ventricle forces blood to the lungs, while the left ventricle forces blood to all other body parts. (Top: courtesy American Heart Association. Bottom: from Hole, 1983)
Figure 1.1.2 Phasic blood flow in the left and right coronary arteries. (From Guyton, 1981). Upper trace: left CBF. Lower trace: right CBF.

Figure 1.1.3 Pressure changes in the left atrium and left ventricle during a cardiac cycle. The peak pressure is between 100-120 mm Hg (From Hole, 1983)
Figure 1.2.1 Anterior and posterior surface of the heart, illustrating the location and distribution of the principal coronary vessels. (From Berne and Levy, 1972)
Figure 1.2.2  
(a) An angiogram (X ray) of the coronary arteries.  
(b) A cast of the coronary arteries and their major branches.  
(From Hole, 1983)
Figure 1.3.1 The waterfall model. A collapsible tube surrounded a pressure $P_c$ and inflow pressure $P_i$. A region of partial collapse occurs at the outflow end (see text).
CHAPTER 2 MECHANICAL ASPECTS OF CARDIAC CONTRACTION

CHAPTER 2
Mechanical Aspects of Cardiac Contraction

This section specifically addresses the physical structures and mechanical properties of the myocardium and microvasculature. The myocardium and vascular wall are composed of discrete tissue substructures which are nonlinear viscoelastic, inhomogeneous, and anisotropic. Some mechanical models will also be introduced in this section.

2.1 Physical Structure

2.1.1 Myocardium

In simulation experiments, the LV is often likened to a thick-walled ellipsoidal shell with nonuniform wall thickness (Figure 2.1.1.1). Here cardiac muscle fibers are aligned primarily in the circumferential-meridional plane of the ellipsoid. The left ventricle is formed from a syncytium of muscle fibers wound in a spiral about the cavity [51]. The directions of these fibers projected on the equatorial tangential plane have been shown to vary continuously through myocardium (Figure 2.1.1.2). Fiber angle is +60 degrees at the endocardium with respect to equatorial plane, running through 0 degrees at the midwall to -60 degrees at the epicardium [11,51,100]. Significant changes in fiber angle between diastole and systole were not observed [51].
Cardiac muscle is a fibrous tissue and is composed of striated myocytes that are joined end to end by intercalated disc (Figure 2.1.1.3). These fibers are interconnected in a branching, three dimensional network [9]. There are some differences between cardiac and skeletal muscle, for example, the abundance of mitochondria (which provides the biochemical energy needed by muscle) and capillary blood vessels in the myocardium as compared with their relatively sparse distribution in skeletal muscle [28,44,82,121]. Mitochondria have a role in oxidation of the foodstuffs and resynthesis of ATP (i.e., adenosine triphosphate), the fuel with the contractile proteins use directly [29,85]. A schematic representation of the structure of ventricle cardiac muscle are shown in Figure 2.1.1.4. Myofibrils are aligned parallel to the cellular axis and are embedded in a "matrix" of supportive tissue. Such material organization represents a transversely isotropic material symmetry[85]. Figure 2.1.1.5 depicts the hierarchy of a skeletal muscle organization. During muscle contraction, the cross bridges which are arranged in a two-stranded helical pattern along the myosin filament make contact with the action filaments and, in a "ratchet-like" manner, produce sarcomere shortening and tension development.

The myocardium is a fibrous tissue, in which the myocytes are the prominent type of fibers, occupying about 70 percent of the volume [83]. The remaining 30 percent consists of various interstitial components [83,92]. About 5 percent of the interstitial volume is occupied by collagen fibers (collagenous connective tissue fibers) [84] (Figure 2.1.1.6).
Cardiac muscle are embedded in a complex weave of heavily cross-linked collagen arranged in a spatial network which forms lateral connections between adjacent muscle fibers [125]. This weave network certainly contributes to the passive viscoelastic properties and also the phenomenon of creep of unstimulated heart muscle [124]. However, the collagen concentration of the myocardium is a function of age and also depends on cardiac geometry [97,124].

Owing to the aqueous nature of blood, interstitial fluid, and intracellular fluid, cardiac muscle is approximately 70-85% water by weight in composition [122,123]. It is generally treated as an incompressible material from a solid mechanics perspective [18,122,123].

2.1.2 Blood Vessel

The vessels of the cardiovascular system form a closed circuit of tubes that carry blood from the heart to the body cells and back again. These tubes include arteries, arterioles, capillaries, venules and veins. Arteries, arterioles, venules and veins have similar wall compositions: the innermost layer (tunica intima), the middle layer (tunica media), and the outer layer (tunica adventitia) (Figure 2.1.2.1) [8,73,74,82,99]. The collagen fiber structure in blood vessels is three-dimensional and is intimately integrated with elastin fibers and
smooth muscle cells [82]. The stiffness of blood vessels at large extensions is associated with the progressive loading of elastin and of collagen fibers. Anatomically, the walls of the artery and vein also exhibit concentric stratification of constituents and cylindrical anisotropic material symmetry due to their wall composition [8,9]. Due to technical difficulties in evaluation of anisotropic parameters of vessels, isotropy is assumed by some authors [28,48,49]. Carew [50] also revealed the validity of the incompressibility assumption in the arterial wall, where water constitutes about 70% of the wall on average [98].

The wall of a capillary consists of endothelium ---- a single layer of squamous, and epithelial cells. These thin walls form the semipermeable membranes through which substances in the blood are exchanged for substances in the interstitial fluid surrounding body cells.

For vessels of smaller caliber however, electronmicrographs reveal that gel-like interstitial fluid clearly separates the vessel wall and the surrounding tissue. With a characteristic of the interstitial thickness dimension dependent on blood vessel caliber, we may estimate the thickness of this interstitial fluid is about 5-10% of the vessel wall in radius (Figure 2.1.2.2)(less than 1 um, [82]). The interstitial fluid is aqueous in composition with a random network of interspersed collagen fibers which tether the blood vessel to the surrounding tissue. The boundary condition thus created therefore lies somewhere between that of uniform attachment and free-slip. Fung [16] suggested that capillaries
in the mesentery behave like a rigid tunnel in a gel and that capillary behavior cannot be treated independently of this surrounding gel [82]. Therefore, the tunnel-in-gel concept of the capillary is incorporated in this report.

2.2 Mechanical Properties

2.2.1 Myocardium

The mechanical properties in the cardiac muscle can be classified as stimulated (active) state and unstimulted (passive) state [19,29,30,31,32,43,83,87,93,127]. Eventhough muscles are classified into three major types: skeletal, smooth, and cardiac, continuum mechanics principles may be applied with some modifications [28,85].

Passive Muscle Properties

Heart muscle in the resting state is an inhomogeneous, anisotropic, nonlinear, viscoelastic, incompressible, material subject to large deformations [86,89,92,127]. For relaxation under constant strain, stress variances are less than 30% over 1000 seconds and is independent of temperature (5-37 C) [28,82,91]. Quasi-linear viscoelasticity theory can be applied to heart muscle during the resting state [16,82,127]. Hysteresis phenomenon, which is one manifestation of viscoelastic
behavior, is found in cyclic loading and unloading of the passive tissue [82,89].

Biaxial loading studies performed on sheets of passive canine myocardium indicate that tissue under biaxial loading is stiffer than under uniaxial loading [88,89,127]. Hence ventricular wall stress calculations should be performed using multiaxial rather than uniaxial material data [88,89,127]. Uniaxial tests are relatively easy to perform and interpret but cannot provide a unique description of the tissue's properties [89,92]. Therefore, detailed understanding of cardiac mechanics of normal and diseased myocardium can only be obtained through multiaxial tests [87,88,89,90,92].

**Active Muscle Properties**

The behavior of active myocardium is dictated by crossbridge kinetics [28] and a detailed discussion of such phenomena is beyond the scope of this work. The macromolecular mechanics of heart muscle contraction have been classified according to isometric contraction (i.e., muscle contracting under constant length after stimulation) and isotonic contraction (i.e., muscle shortening under constant load after stimulation) [90,94,95,96]. According to Hill [94], muscle contraction can be consider in terms of two components in series: an active contractile element (CE) and an undamped passive series elastic element (SE) (Figure 2.2.1.1) which possesses negligible resting tension. To include
passive mechanical behavior a three-elements model is postulated which contains the aforementioned CE, SE, and a parallel elastic (PE) element which supports resting tension in passive muscle (Figure 2.2.1.1). The process of contraction is represented in Figure 2.2.1.1.

Global Mechanical Aspects

A full description of cardiac muscle based on three-dimensional, time-dependent constitutive considerations has not been fully established at present. And a three-dimensional heart muscle law is expected to be strongly influenced by geometrical, structural, and material complexities. Additional aspects, such as nonuniform organ contraction, interaction between heart chambers, tethering influence of blood vessel, nonlinear properties in biological tissue need to be considered. The superposition does not apply in stress-strain relationship for nonlinear system, and the analysis of additional stresses due to external loading must take the residual stresses (i.e., exist in organs during unloaded state) into account [28,108]. Considering the large thickness-to-radius ratio of left ventricle and its complex fiber-winding pattern, thick-shell effect and anisotropy are expected.

The viscoelastic aspect of the tissue response is not considered in the present study. Due to the relatively short duration of the cardiac cycle
compared with the characteristic relaxation time of the myocardium, we can make this assumption [44]. The permeability of the tissue is low (about $10^{-15} \text{m}^4/\text{N s}$), and therefore the fluid flow within the tissue is negligible for the pertinent time intervals [18]. Consequently we will assume vascular and cardiac tissue is incompressible.

2.2.2 Blood Vessel

The mechanical properties of blood vessels depend not only on material composition, but also on wall structure and untrastructure [82]. Collagen and elastin are key structural materials in arterial and venous walls. Harkness et al. [98] measured the quantity of elastin between the intrathoracic aorta and all other arteries in dogs by means of hydroxyproline content and got a sharp distinction. Collagen has a high tensile strength but low extensibility, that is, it has a high modulus of elasticity. Elastin, on the other hand, has a low modulus of elasticity and is more easily stretched and has relatively little tensile strength. Canfield and Dobrin [28] reported that the values of the modulus of elasticity are of the order of $1E6$ kpa for collagen and $400$ kpa for elastin. Veins contain a relatively high amount of collagen with the collagen-elastin ratio being about 3:1 [82]. The elasticity of capillary blood vessels remains to be verified. Creep, relaxation, and length-force or pressure-diameter hysteresis, are observed in blood vessels [28,82].
Vascular smooth muscle also contributes to arterial and venous elasticity in the circumferential direction. Smooth muscle has lower stress response and large deformation, and exhibits a tremendous hysteresis loop [28,82]. Some of smooth muscle cells form rings, being joined end to end, and are located between concentric elastic lamellae (Figure 2.1.2.1). Vascular smooth muscle may be activated to produce vasoconstriction and axial isometric contraction phenomena. Vasoconstriction is associated with shortening of the vascular muscle cells, and a concurrent reduction in vessel lumen caliber, thus vasoconstriction allows a blood vessel to withstand increased transmural pressure and distending force at the same diameter (i.e., by the Laplace law) [28]. Through isometric contraction, a blood vessel can increase transmural pressure but keep a constant diameter. Both of these contractile phenomena can increase the effective mechanical stiffness of the vascular wall (Figure 2.2.2.1) [28].

Global Mechanical Aspects

Vascular walls are not isotropic in their material symmetry, with their departure from isotropy depending upon the vessel considered [28,128]. Cross sections of vascular walls typically exhibit layered structure of elastic laminae with intervening smooth muscle cells in the media (or middle transmural layer) [82, 28]. In addition, the vessel wall is clearly not homogeneous, being composed of elastin, collagen, ground
substance, and smooth muscle cells arranged in a highly complex architecture [28]. Several approximation for vascular walls are applicable, such that they are cylindrical [129], and incompressible [130]. Experimental studies [24] have demonstrated that arteries deform orthotropically, that is, the deformations need be examined in only three orthogonal directions. The vessel has elastic properties that are nearly symmetric about planes perpendicular to the principal stresses under physiologic loading [28]. The residual stress in the unloaded artery has been demonstrated to be significant in reducing the high stress concentrations and high stress gradient at the vessel wall [108,109,127].

2.3 Analogy to Hyperelastic Materials

There are many different elastic characteristics which related the various functions and interactions of the cardiac and vascular tissues. Burton [36] revealed that the molecular organization and fiber arrangement in the vascular wall are analogous to that of rubber. Horowitz [18] and Huisman [63] suggested that myocardium may be treated as a hyperelastic material, which means that we can use a strain energy function to describe the stress-strain relation. Other authors [16,28,48] have used elastomers, rubbers or materials with equivalent values of Young's Moduli in their small strain analyses of cardiac and vascular mechanics. According to Roy[16], a piece of artery is more like
a rubber band than a metal strip. Further scrutiny allows us to draw analogies between cardiac muscle tissue and swollen rubber (i.e., rubbers with high water content).

In determining the nonlinear elasticity of soft biological tissues, two analytic methods have been employed. One of the approaches is the use of finite deformation theory (if one assumes that a strain energy function exists); a method originally developed to study the behavior of elastomeric material such as rubber [28,48]. The corresponding strain energy function obtained from incremental experimental stress-strain curves is called a pseudo strain energy function [82,86]. If we assume the material is perfectly elastic and ignore the strain-rate effect, we may consider the bio-viscoelastic solids, as a pseudo-elastic materials.

Various strain energy functions describe the constitutive relation of rubber-like materials. These include the Mooney-Rivlin [5,7,12,28,48], Ogden [2], Valanis-Landel [2,5], and Rivlin-Saunders formulations [7]; some modified exponential forms are also available [1,2,28,69,70]. A strain energy density function of particular interest to this report is the two-term Mooney-Rivlin formulation.

\[ W = C_1(I_1-3) + C_2(I_2-3) \]  

(2.3.1)

Where \( C_1 \) and \( C_2 \) are material coefficients, with \( I_1 \) and \( I_2 \) representing the first and second strain invariants, respectively. As a
first approximation to the more general anisotropic, nonlinear constitutive formulations for cardiac fibers and blood vessels noted above. Here different coefficients values (i.e., C1 and C2) will be employed to simulate the various elastic characteristics of both myocardium and the embedded blood vessel. Such an approximation was required due to computational deficiencies associated with ANSYS algorithm, and appears justified based on the previous biomechanical work noted above.

2.4 Intramyocardial Stresses Profile In 3-D

Differential systolic intramyocardial pressures are one of mechanisms which cause differential systolic flow across the LV wall [121]. The mechanical performance of the myocardium affects its metabolism, which also has a major impact on myocardial blood flow [121]. Since transmural gradients in intramyocardial stress and vascular tone may significantly impact the stress field near an embedded blood vessel, quantifying the magnitude and direction of ventricular wall stress plays an important role in assessing microvascular blood flow.
Various analytical and numerical models of the LV have been postulated. These include spherical and cylindrical models [15,46], or truncated ellipsoids of revolution [19,25,29,31,32,62,63,65,66], which models can be further associated with thin-walled (i.e., we neglect radial, transverse shear and bending stresses)[58,62], or thick-walled (where radial, transverse shear, and bending stresses are included) [65,66] elastic shell theory. Finite element models have also been presented [67]. Experimental investigations have also sought to qualify intramyocardial stress components by using various techniques [10,35].

Generally speaking, the three myocardial stress components which are discussed in the aforementioned models or experimental protocols are radial stress (transmural stress, S1), longitudinal stress (meridional stress, S2) and circumferential stress, S3, as depicted in Figure 2.1.1.1. The nonconcentric geometry of overlying fibers and the interaction between "layers" due to tethering may result in three-dimensional stress patterns [30]. Shear stresses in differential coordinate planes of endomyocardium are found more significant than those of epicardium [30,63,127].

Experimental investigations have sought to quantify intramyocardial stresses by using a variety of techniques [30,42,53,54, 55,56,57,58]. Figure 2.4.1 is a graphic representation of intramyocardial pressure (i.e., radial stress component in the ventricular wall) (ordinate) recorded at varying depths of the myocardial wall (abscissa). Zero on the abscissa stands for the endocardial surface whereas 100% stands for epicardium.
The intramyocardial pressure exhibits a markedly high value near the endocardium.

Strong stress gradients depicted in Figures 2.4.1, 2.4.2, and 2.4.3, can be explained as follows: (1) The radial stress is determined not only by ventricular pressure but also by wall thickness and the principal radii of curvature [25,32]. (2) Direct measurement of radial stress in the LV wall through invasive techniques may distort the myocardium and muscle fiber geometry and thereby induce measurement artifacts [10,57]. (3) Intramyocardial stresses increase from base to apex in the LV [25,66]. (4) Residual stresses exist in living organs and tissues.

The second point mentioned above, on experimental measurement artifacts, can be explained by Laplace's law [62,75]. The Law of Laplace states that transmural pressure, across a cylindrical segment of the ventricular wall is directly proportional to the tension in the wall, and inversely proportional to the radius of curvature R, so that \( P = \frac{T}{R} \). Here, \( T \) is the sum of the force of active tension developed by cardiac muscle contraction plus the passive tension exerted by the elastic properties of the material [59,60,61] (Figure 2.4.4). If a pressure measurement device is inserted into the myocardium, we envision the distorted configuration depicted in Figure 2.4.5. The effective radius of curvature of the embedded device is on the order of millimeters, and is expressed as \( r \). Note that \( r \ll R \) and let the transmural pressure sensed by the implanted device be given as \( p \). Since muscle tension would generally not depend
on device implantation, the effective change in fiber radius of curvature from $R$ to $r$ augments the transmural pressure sensed by the implanted device by a factor $R/r$ times the real transmural pressure $P$ [10].

Transmural circumferential and meridional hoop stresses are plotted in Figure 2.4.2 and 2.4.3. Sandler and Dodge [62] calculated average tensile stresses for an ellipsoidal thin-walled model with finite wall thickness, but, as in their discussion, the value underestimates the actual peak stress presented in the wall by 30-40%. Wong and Rautaharju [66] calculated the stress distribution in a thick-walled ellipsoid of revolution, assuming an isotropic, linear elastic and homogeneous material. They assumed that the heart is acted upon only by radial stresses and longitudinal stresses and neglected shear and bending moments in the wall. Gould [53] applied the finite element method which is able to delineate the effect of varying chamber wall curvature for stress analysis of the left ventricle.

Misky [65] has extended the Wong and Rautaharju analysis to include the effects of shear and bending moments, but neglected the fiber orientation. His results differed from that of Wong and Rautaharju in that the meridional stress at the equator increases rather than decreases from endocardium to epicardium. Streeter [32] revealed that the circumferential and longitudinal stress distribution are very sensitive to fiber orientation. His model accounts for fiber geometry and a quasi-static systolic activation (i.e., instantaneous measurement of
geometry and left ventricular pressure are employed in a static analysis for evaluation of instantaneous stress throughout the cardiac cycle). Again the results show that LV stress distribution depends strongly on the fiber angle distribution and the relative thickness of the ventricle as the ventricle accommodates volume.

Streeter measured both fiber curvature and orientation in the LV wall in an attempt to calculate myocardial stress distributions. The fiber curvature in systole is lower than that in diastole near the endocardial surface, greater in the midwall region (maximum), and almost the same near the epicardial surface. Since the fibers perform most of the load bearing during systole [10,32] it is intuitive that we use data measured during systole.

Due to the lack of experimental verification and the influence of measurement artifact, the discrepancy between these results (i.e., Figures 2.4.1, 2.4.2, 2.4.3) make it difficult to know which data set is most accurate. Yin[29] revealed that using an averaged wall stress or a midwall stress would be most appropriate for modeling applications.
2.5 Summary

Phasic transmural blood flow during the cardiac cycle is strongly affected by mechanical interaction between the myocardium and its perfusion system. Myocardial stresses generated during systole are likely to constrict intramyocardial blood vessel particularly at the microvascular level. Global stresses in LV can be influenced by the following factors,

1. Geometric and material nonlinearity.
2. Thick-shell effect.
3. Anisotropy (fiber orientation).
4. Inhomogeneity (lamination and constituent material diversity).
5. Muscle activation.

The transmural stress profile calculated by Streeter and colleagues [32] will be applied as a first approximation. Microvascular blood vessels are assumed to be isotropic. Both the myocardium and vascular wall will be considered incompressible due to their aqueous composition. Small caliber microvessels (capillaries) are lossely tethered to the surrounding muscle tissue due to the existence of gel-like interface. Large caliber blood vessels (veins, venules, arteries) are uniformly attached to the myocardial tissue. Isotropic hyperelastic material with nonlinear stress-strain relationship, such as rubbers, will be used in a finite element model as a first approximation to their more general nonlinear, anisotropic constitutive behavior.
Figure 2.1.1.1 Three-dimensional representation of left ventricle. (From Yin, 1981)

Figure 2.1.1.2 The fiber angles through the wall thickness at the apex (△), the region midway between apex and base (▽), and at base (●). These fiber angles were considered in Streeter et al. (1969).
Figure 2.1.1.3  Diagram of an electron micrograph of cardiac muscle showing large numbers of mitochondria. (From Berne and Levy, 1988)
Figure 2.1.4  A schematic representation of the 3-D cardiac muscle structure (From Eckert and Randall, 1983)

Figure 2.1.5  Organization of striated muscle structure (From Eckert and Randall, 1983)
Figure 2.1.1.6 Cross-section of the wall of the heart. From the epicardium to the myocardium. KEY: END: Endocardium. MYO: Myocardium. EPI: Epicardium. CT: Connective tissues. BV: Blood vessels. SM: Smooth muscle fibers (From Reith & Ross, 1977).
Figure 2.1.2.1 Three-dimensional representation of blood vessel wall Top to bottom: artery, vein, capillary (From Lesson, 1976).

- Tunica intima (inner coat)
- Tunica media (middle coat)
- Tunica adventitia (outer coat)
- Endothelial cells
- Connective tissue
- Elastic tissue
- Arteriole
- Smooth muscle cell
- Endothelium
- Capillary
Figure 2.2.1.1  Mechanics of muscle contractions. (B): The beginning of contraction. (C): Tension building up with progressive stretching of SE. (D): The load is lifted and the contraction becomes isotonic. (From Eckert and Randall, 1983)

Figure 2.2.2.1  A lumen diameter of vessel with normal tone; (b) diameter as a result of vasoconstriction; (c) diameter as a result of vasodilation. (From Lesson, 1976).
Figure 2.4.1  Experimental (top) and analytical (bottom) determinations of radial stress across the left ventricular wall. Stress values normalized by left ventricular pressure. (From Kempski, 1987)
Figure 2.4.2  Analytical determinations of circumferential stress components across the left ventricular wall. Stress values normalized by left ventricular pressure. (From Kempski, 1987)
Figure 2.4.3  Analytical determinations of meridional stress components across the left ventricular wall. Stress values normalized by left ventricular pressure. (From Kempski, 1987)
Figure 2.4.4  Equilibrium force balance in a layer of cardiac muscle fibers, under the assumption of a cylindrical ventricular geometry.

Figure 2.4.5  Schematic of hypothetical changes to the force balance depicted in Figure 2.2.2 upon insertion of an intramyocardial pressure measuring device.
CHAPTER 3 METHODOLOGY

The preceding chapters have detailed various aspects of cardiovascular mechanics and constituent material properties, and underscored the need for a thorough understanding of events which lead to the occlusion of embedded blood vessels. A mathematical model for embedded blood vessel mechanics needs to be justified in this chapter. Analytical simplification in simulating the gel-like interstitial interface also needs to be considered. A brief description of nonlinear finite element methodology is presented in this chapter.

3.1 General Hyperelastic Constitutive Considerations

The continuum mechanical derivation of the constitutive equation for elastomeric materials is based on the concept of a strain energy function or elastic potential "W" which is a function of the strain invariants. Biosolid materials are also assumed to possess a strain energy density function function "W". Under the action of pure homogeneous deformations (i.e., the deformation is varied in such a manner that one of the invariants of the strain ,I1 or I2 , is maintained constant [2,4,6,7,69,70]), we have
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\[ I_1 = \lambda_1^2 + \lambda_2^2 + \lambda_3^2 \]  
\[ I_2 = \lambda_1^2 \lambda_2^2 + \lambda_2^2 \lambda_3^2 + \lambda_3^2 \lambda_1^2 \]  
\[ I_3 = \lambda_1^2 \lambda_2^2 \lambda_3^2 \]  
(3.1.1)  
(3.1.2)  
(3.1.3)

Where \( \lambda_i = L/L_i \) represents the stretch ratio of deformed to undeformed length (i.e., \( L \) and \( L_i \), respectively) along a given material direction.

For an incompressible, isotropic material, the basic assumptions of the Mooney-Rivlin constitutive relation take the form

\[ W = \sum_{i=0}^{\infty} C_{ij} (I_1 - 3)^i (I_2 - 3)^j \]  
\[ W = C_{10} (I_1 - 3) + C_{01} (I_2 - 3) \]  
(3.1.4)  
(3.1.5)

Where \( C_{10} \) and \( C_{01} \) represent material coefficients. Equation (3.1.4) is the most general form which can be valid for small and large deformations [7]. In general \( W = W(I_1, I_2, I_3) \) but incompressibility yields \( I_3 = 1 \) (see below), hence \( W = W(I_1, I_2) \).

If the \( \lambda_i \) (i=1,2,3) represent the principal stretch ratios in three directions (i.e., without shear forces), from basic geometric relations, we can state the relative area change and relative volume change in the material as, respectively
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\[ \frac{A_i}{A_b} = \lambda_j \lambda_k \quad \frac{V}{V_0} = \lambda_1 \lambda_2 \lambda_3 \]

where subscript "0" represents undeformed state and "i" represent normal vector in "i" direction. For a general deformation we can show that:

\[ \frac{V}{V_0} = I^{1/2} = \det \begin{bmatrix} \lambda_1^2 & 0 & 0 \\ 0 & \lambda_2^2 & 0 \\ 0 & 0 & \lambda_3^2 \end{bmatrix}^{1/2} \]

If the continuum is incompressible, volume elements are conserved during deformation so that \( I_3 = 1 \).

We will consider a body of perfectly elastic material to undergo a deformation in which a point \( P_0 \), initially at \( R = (a_1, a_2, a_3) \), deforms after time \( t \) to the deformed body point \( P \) at \( r = (X_1, X_2, X_3) \). Here \( R \) and \( r \) are the position vectors and \( u = r - R \) is a displacement vector. From kinematics of a deformable solid [3,12,110, 111] and the deformation gradients of the Lagrangian strain tensor (i.e., based on undeformed state), we specify:

\[ g_i = \frac{\partial r_k}{\partial a_i} e_k = [\delta_{ki} + U_{ki}] e_k \]

\[ g_{ij} = g_i \quad g_j = \delta_{im} \frac{\partial X_i}{\partial a_m} \frac{\partial X_j}{\partial a_j} \quad (3.1.6) \]

where \( g_i \) = Covariant base vector of the strained body.

From continuum mechanics we know strain can be expressed as

\[ \varepsilon_{ij} = \frac{1}{2} \left[ \delta_{im} \frac{\partial X_i}{\partial a_j} \frac{\partial X_m}{\partial a_j} - \delta_{ij} \right] \quad (3.1.7) \]
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Substituting (3.1.6) into (3.1.7), we get $\varepsilon_{ij} = 1/2 (g_{ij} - \delta_{ij})$. With the aid of $u=r-R$, we obtain the strain-displacement relations:

$$
\varepsilon_{ij} = \frac{1}{2} \left[ \frac{\partial u_i}{\partial a_j} + \frac{\partial u_j}{\partial a_i} + \frac{\partial u_i}{\partial a_j} + \frac{\partial u_j}{\partial a_i} \right]
$$

(3.1.8)

linear terms \hspace{1cm} \text{nonlinear terms}

where $g_{ij} = C_{ij} =$ the right Cauchy-Green tensor $\varepsilon_{ij} =$ the Green-Lagrange strain tensor $\delta_{ij} =$ Kronecker delta $a_1,a_2,a_3 =$ orthogonal coordinate system

The alternate strain invariants for hyperelastic materials are [110, 111],

$$
I_1 = 3+ 2 \varepsilon_{ij}
$$

(3.1.9)

$$
I_2 = 3+4 \varepsilon_{ij} + 2(\varepsilon_{ij} \varepsilon_{ij} - \varepsilon_{i} \varepsilon_{j})
$$

(3.1.10)

$$
I_3 = \det (\delta_{ij} + 2 \varepsilon_{ij})
$$

(3.1.11)

The stress may be expressed as (i.e., the 1st Piola-Kirchhoff stress tensor) [112]

$$
T_{ij} = \frac{2}{\sqrt{I_3}} \left[ (I_2 \frac{\partial W}{\partial I_2} + I_3 \frac{\partial W}{\partial I_3}) \delta_{ij} - I_3 \frac{\partial W}{\partial I_2} \varepsilon_{ij}^{-1} + \frac{\partial W}{\partial I_1} \varepsilon_{ij} \right]
$$

(3.1.13)

For $I_3=1$, (3.1.13) can be simplified to

$$
T_{ij} = -P_{ij} \delta_{ij} + 2 \frac{\partial W}{\partial I_1} \varepsilon_{ij} - 2 \frac{\partial W}{\partial I_2} \left[ \varepsilon_{ij} \right]^{-1}
$$

(3.1.14)

Where $T_{ij} =$ Engineering or Lagrangian stress components,

(force/undeformed area).

$P =$ an arbitrary hydrostatic pressure [2,103].

Superscript "-1" represents the inverse of a matrix.
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Under the pure homogeneous definition of simple extension, biaxial extension, and pure shear, the deformations and stress boundary conditions exist as shown below,

for simple extension  \[ \lambda_2 = \lambda_3 = \lambda^{-1/2} \quad T_{22} = T_{33} = 0 \]
for Equi-biaxial extension  \[ \lambda_1 = \lambda_2 = \lambda_3^{-2} \quad T_{22} = T_{33}, T_{11} = 0 \]
for pure shear  \[ \lambda_3 = \lambda_1^{-1}, \lambda_2 = 1 \quad T_{33} = 0 \]

Experimentally \( T_{11} \) and \( T_{22} \) can be calculated from measurement of the test specimen thickness, \( \lambda_1 \) and \( \lambda_2 \), and the applied tensile forces. The strain energy function with \( C_{10}, C_{01}, \) etc. as given by (3.1.4) can be determined by curve fitting of the solution (3.1.13 or 3.1.14) with the measures \( T_{11} \) and \( T_{22} \), etc. [103,131].

3.2 Mathematical Model of Embedded Blood Vessel Mechanics

The basic construction of the model is based on microscopic physical behavior of the embedded vessel. This work is a continuation of the investigations of Kempski [10]. His study assessed the influence of cardiac muscle transverse isotropy and material differences between cardiac fibers and embedded blood vessels, in terms of finite strain,
linear elastic finite element analysis. The author of present work hopes to build on the previous linear elastic results by incorporating nonlinear constitutive behavior into the finite element analysis. Consideration of the impact the gel-like interface between myocardium and embedded microvessels may have on coronary flow impedance is a specific aspect of this study.

The present study of embedded blood vessel mechanics focuses on a single blood vessel embedded in a segment of myocardium which is located near the midwall of the LV. The ventricle is considered as a thick-walled truncated ellipsoid of revolution, with systolic cavity pressure developing in response to wall tension changes due to cardiac fiber contraction. Horowitz [14] compared strain differences between the circumferential and the longitudinal directions in a wide range of fiber variation angles of his myocardial model (0-90 degrees). He found that strain differences decrease slowly between +/- 50 to +/- 70 degrees. Since the fiber ranges of distribution in the LV wall are contained between +/- 60 degrees, the small variation in the strain difference may indicate that the ratio of circumferential-to-longitudinal stiffness is relatively uniform over the LV wall. A representative shell element of myocardium cut from the ventricular wall is shown in Figure 3.2.1. A typical myocardial-blood vessel segment for analysis is depicted in Figure 3.2.2. The inhomogeneous structure of the myocardium-interface-blood vessel as noted in Chapter 2 will be taken into account in this model by way of three different isotropic hyperelastic materials with different
constitutive coefficients. Three myocardial stress components (i.e., S1, S2, and S3) are assumed to load the tissue segment in response to ventricular cavity pressurization. A representative loading schematic is shown in the lower panel of Figure 3.2.2. The compressive sense of S1 indicated in this model stands for the radial ventricular stress. Due to symmetries in loading, geometry, and material properties, a simple quarter slice of the cube is modeled to obtain a complete representation of the entire model.

The external and internal radius of the embedded blood vessel model in this report are assumed equal to 0.1mm and 0.05mm, respectively. The vessel caliber is assumed small when compared to the thickness of a muscle shell (layer) [18,20,21]. A finite microvascular length is assumed equal to the internal diameter based on photomicrographs of unbranched arterioles and venules [28]. Burton [36] also suggests that arteriole/venule wall thickness is approximately half the vessel diameter, which is incorporated in the present model.

The muscle segment dimensions of the model used in this study are 0.5mm *0.5mm*0.1mm (L*W*D). This template is also the same dimension as a shell element done in Streeter's model [32]. The finite element discretization of the embedded vessel cross-section is depicted in Figure 3.2.3. The mesh shown was chosen because it yields an adequate balance between CPU time and result accuracy based on test case comparative evaluations (See Section 4.1). The mesh refinement near the material interface (thin band of elements located between the 5th and
7th radial elements, Figure 3.2.3) was done to better represent any stress concentrations which might arise due to material inhomogeneity between embedded vessel, interstitium, and surrounding muscle tissue.

In general, the embedded microvessel may posses an arbitrary orientation with respect to the local cardiac fiber direction (in a shell element). However, we postulate that lumenal constriction is primarily affected when the vessel axis is parallel or perpendicular to the local muscle fiber direction (i.e., where both configurations requires the vessel to lie within the plane of the cardiac fibers). Here ventricular radial stresses would compress the embedded microvessel cross-section whether the muscle fibers and blood vessel axes were parallel or perpendicular. The parallel or perpendicular fiber-vessel orientations will represent extremes in tissue circumferential-meridional plane loading. We also assume that embedded vessel constriction can only occur under the influence of extravascular forces.

At midwall, meridional stresses vanish due to the fact that the fiber angles are zero at this location (Figures 2.1.1.2 and 2.4.3). Cardiac fiber stresses and ventricular circumferential stresses are therefore identical in this model. For the purposes of this report we utilize the transmural stress profiles calculated by Streeter et al [32]. The values of radial stress distributions are monotonically decreased from endocardium to epicardium (Figure 2.4.1). From Figures 2.4.4 and 2.4.5, we note that the circumferential stress is maximal near the midwall; the longitudinal
stress is maximal near the endocardial and epicardial surfaces, and the radial stress is maximal at the endocardial surface, whether at end diastole or at end systole.

When the fiber orientation angle is parallel to the axis of the blood vessel, the fiber stress influences vessel lumenal deformation indirectly through the Poisson's effect. Therefore, for a midwall tissue segment, the stress components in this model become \( S_1 = -0.42 \times LVP \), \( S_2 = 0 \) and \( S_3 = 1.38 \times LVP \) (referring to Figure 2.4.1, 2.4.2, 2.4.3), where \( LVP \) represents left ventricular cavity pressure. In contrast, when vessel-fiber orientation is perpendicular, the fiber stress acts directly to influence lumenal distortion, such that \( S_1 = -0.42 \times LVP \), \( S_2 = 1.38 \times LVP \) and \( S_3 = 0 \).

The physiological loadings noted above are approximated by two loading scenarios in the current finite element study. LVP acts on the model in parallel fiber direction as well as perpendicular fiber direction. Each condition, therefore, is performed by biaxial loadings instead of triaxial loadings which is not suitable under incompressibility assumption (i.e., due to \( I_3 = 1 \), constant).

The study of left ventricle pressure-volume relations of has prompted some authors [32, 63] to idealize the ejection of blood from the LV as occurring at constant pressure (=100 mm Hg). It will be reasonable, therefore, for us to assume peak systolic ventricular cavity pressure
equals 13.3 kpa (100 mm Hg) in determining specific value for S1, S2 and S3.

3.3 Justification For Use of Mooney-Rivlin Constitutive Formulation

The use of the Mooney-Rivlin constitutive formulation in the current study is clearly a first approximation to the more general transverse isotropy and cylindrical orthotropy exhibited by cardiac muscle and blood vessels, respectively. For anisotropic materials, $W$ is a polynomial in the strain components $\varepsilon_{ij}$ [48,102]. Each of the strain invariants quantities $I_1, I_2, I_3, \ldots$ (say) is a polynomial in $\varepsilon_{ij}$ which is form-invariant under the group of transformations associated with the material symmetry considered. The strain energy function, $W$, is therefore expressible as a polynomial in $I_1, I_2, I_3, \ldots$ [48,102] (where $I_1, I_2, I_3$, are defined in Section 3.1). The present simplification to the isotropic condition was necessary due to restrictions imposed by the computational algorithm (ANSYS 4.3A, see appendix A).

Previous studies [10] have indicated that axial cardiac muscle elastic stiffness at physiologic stress levels comparable to those used in this report will be 700 kpa. Comparing this to analogous vascular elastic
stiffness data (i.e., 200 kpa [10]), we have the ratio of cardiac muscle axial elastic modulus to the vascular elastic modulus, of 3.5. This ratio is made under the assumption that small segments of the nonlinear strain-stress curve may be approximated as linear, hence the tangent modulus is taken as "the elastic stiffness". Under the aforementioned nonlinear hyperelastic, and isotropic assumptions for both myocardium and the vascular wall, such information allows us to specify the coefficients C1 and C2 in the respective Mooney-Rivlin strain energy formulations.

The modulus of elasticity that is of application to the biophysics of the blood vessel wall can be expressed as follows, under the assumption that the mechanical analog will be the uniaxial stretching of a rubber strap

\[ F = \left[ Y \frac{\Delta L}{L_0} \right] A \quad \text{or} \quad P = Y \lambda \]  
(3.3.1)

Here \( F \) is the axial force exerted on the material, \( P \) is the axial stress, \( \frac{\Delta L}{L_0} = \lambda \) is the axial stretch ratio, and \( A \) is the cross-sectional area. The elastic modulus is denoted as \( Y \). Since \( Y_{\text{muscle}} = 3.5 \times Y_{\text{vessel}} \) (i.e., at LVP=13.3 kpa, BVP=4kpa), The slope ratio of muscle fiber and vessel wall stiffnesses will be about 3.5.

If we postulate the myocardial constitutive form of

\[ W_{\text{myo}} = C_3(I_1^{-3}) + C_4(I_2^{-3}) \]  
(3.3.2)

and blood vessel constitutive form

\[ W_{\text{Bv}} = C_1(I_1^{-3}) + C_2(I_2^{-3}) \]  
(3.3.3)
Then, the experimental data noted above implies that both C3 and C4 are greater than C1 and C2, (i.e., C1 and C2 , or C3 and C4, control the slope of the stress-strain curves and influence the effective elastic stiffness as will be discussed later), however, the ratio will not be very large. The ratio of myocardial elastic stiffness in transverse direction will not be the same as the ratio of axial stiffness values. Here we assume that the change of fiber transverse modulus is small during systole comparing to that along the fibers [10].

For a given rubber-like material, the c_{10} coefficient (i.e., equation 3.1.5) varies widely according to the degree of vulcanization and also is a function of the network structure [2,113]. In the unswollen state, c_{10} cannot be described in terms of a single elastic constant. Organic tissue with aqueous composition can be treated as a swollen rubber where generally C01/C10=0.1 [2]. Therefore, a constant c_{10} value can be used in this embedded blood vessel model (i.e., C1 and C3 will be assumed constant in equations 3.3.2, and 3.3.3, respectively). c_{10} can also be analogous to1/2 G in the Gaussian statistical theory (Neo-Hookean, see Section 4.1, where G stands for shear modulus [2,82,113]).

According to the author's test cases (Section 4.1), in which a cube of rubber is deformed biaxially, increasing c_{10} also increases the initial slope of the force-stretch ratio curve (near the region where stretch ratio equals one). On the other hand, c_{01} controls the slope at the end of this
curve. The increased ratio of the slope at the end of the curve is proportional to the change in $C_{01}$.

A number of alternative forms of the strain-energy function with higher-order terms in $(I_1-3)$ and $(I_2-3)$ have been developed to cover a wide range of deformations [2,4,6,7,69,70,103,112]. Comparisons between these formulas in equi-biaxial tension and uniaxial tension are shown in Figures 3.3.1 and 3.3.2. When $\lambda$ exceeded 2, at moderate strains, there is a tendency for experimental stress to exceed the theoretical values, however, at very large strains, theoretical predictions diverge dramatically from experimental stresses.

These comparisons imply that Mooney-Rivlin equation does not provide a satisfactory and self-consistent basis for the representation of the whole properties of a rubber. Nevertheless, more reasonable agreement still can be achieved with a Mooney-Rivlin formulation where the stretch is small to moderate ($1<\lambda<2$) (Figure 3.3.1, 3.3.2) [131]. The justification of small $\lambda$ values is documented in Appendix C. We will employ the basic Moony-Rivlin strain energy formulation of "W" in the present study subject to such restriction.
3.4 Modeling The Vessel-Myocardial Interface

Our particular model of intramyocardial blood vessel mechanics includes cardiac muscle, vascular tissue and the gel-like interface which separates them (the interstitium). Unfortunately incorporation of an ANSYS 3-D fluid element to simulate the gel-like interface is possible only under the restriction to small deflection [1]. Computational errors therefore arise when large-deflection hyperelastic solid elements adjoin these fluid elements. To circumvent these computational difficulties, we note the following.

Gumbrell, Mullins, and Rivlin[2] derived a formula which relates $C_1$, $C_2$ to $1/V_2$, the swelling ratio,

$$\frac{f^1v^{1/3}}{2(\lambda-1/\lambda^2)} = C_1 + \frac{C_2}{\lambda} \quad (3.4.1)$$

Where $f^1$ is the force referred to the unstrained unswollen area, $V_2$ is the volume fraction of rubber, and $\lambda$ is simple extension stretch ratio. In setting $C_1$ constant (i.e., $C_1$ is independent of degree of swelling), $C_2$ falls progressively with increasing degree of swelling so that, at the highest degree of swelling, the value of $C_2$ tends to zero (Figure 3.4.1). This phenomenon implies that one may simulate a gel-like material by using a Mooney-Rivlin formulation where $C_2$ tends to zero. Since $C_2$ is dictated by the volume fraction of rubber (see equation 3.4.1), this approach seems
plausible for use in describing the constitutive behavior of the interstitium which possesses small volume fraction of collagen fibers dispersed in aqueous media.

An alternative approach for incorporating a fluid interface with large-deflection characteristics is, therefore, through the use of a hyperelastic material in which $C_{01}=0$. Fluid elements have well-known small slope in tension-displacement relation, therefore, a very small "$C_{10}$" value is expected in this approach.

3.5 Finite Element Implementation

Initially developed for structural analysis of aircraft, the Finite Element Method (FEM) can be used to solve a wide range of complex field problems including electrical, acoustic, heat transfer, structural, and fluid problems. It is a powerful tool in analyzing any complex structure that would be difficult to describe theoretically. The mechanics of the individual elements can be described in terms of particular points, called nodes. Local variations in geometry, different materials, loading and boundary constraints may be easily incorporated by varying element parameters. Classical derivation of FEM may be found in any FEM reference [3,71,72]. A brief summary of important theoretical considerations is discussed presently.
A displacement field for the nodes is assumed and the potential energy of the individual elements calculated. The total structural potential will be the sum of the element potentials. The structure is in equilibrium when the total potential is minimized. "W", as we mentioned in the previous section, will be the potential strain energy of one element. A stress measure that we use abundantly is the 2nd Piola-Kirchhoff stress tensor [110]. The 1st Piola-Kirchhoff stress tensor (i.e., the engineering stress) only considers area transformation (deformed area to undeformed area) and keeps the actual force the same. The 1st Piola-Kirchhoff is not symmetric. The 2nd Piola-Kirchhoff stress tensor includes not only area transformation but also acted forces transformation; it is symmetric [110].

The detailed derivation of strain-energy function is shown in Appendix B. Eventually, we have,

$$ S_{ij} = \frac{\partial W}{\partial \epsilon_{ij}} = 2 \frac{\partial W}{\partial C_{ij}} \quad (3.5.1) $$

The basic finite element method consists of discretizing the domain, selecting the shape function, developing the element constitutive equations, assembling the global governing equation, and solving the global equations for nodal displacements. Backward substitution of displacement data then allows the calculation of additional results such as elemental strains and stresses. Since the derivation of the basic equations used in the finite element method is rather extensive and not the
purpose of this report, only the major highlights are listed here. The reader is directed to [1] and the other references [3,5,71,72] for further detail.

Starting with the variational principle, we have (i.e., dot points above alphabets represent increments after deformation)

\[
\int \delta W + \dot{\delta W} \, dV = \int (P_n + \dot{P}_n) \, \delta u \, ds + (P + \dot{P}) \, \delta u
\]

work of surface loads work of point loads

TOTAL INTERNAL WORK = TOTAL WORK OF EXTERNAL LOADS

From Equation 3.1.4, 3.1.5 we note that \( W=W(I_1,I_2,I_3)=W(C_{ij})=W(g) \), where \( g \) is metric tensor of the current configuration. We note from the calculus that

\[
\delta W = \frac{\partial W}{\partial g} \, \delta g
\]

(3.5.3)

\[
\delta \dot{W} = \frac{\partial W}{\partial g} \, \delta g + \frac{\partial^2 W}{\partial g \, \partial g} \, \delta g \, \delta g
\]

(3.5.4)

Let \( F \) represent deformation gradients \( F_i = \frac{\partial X_i}{\partial a} \). Then \( g_{ij} = F^T F \), and substitution of this quantity into equation substituting to 3.5.4 and 3.5.2 yields (see derivation detail in [1])

\[
\int_{\Omega} \delta F^T \left[ \begin{array}{c}
2 \frac{\partial W}{\partial g} \, F \\
\frac{\partial^2 W}{\partial g \, \partial g} \, \delta g \, \delta g
\end{array} \right] \, dV + \int_{\Omega} \delta F^T \left[ \begin{array}{c}
2 \frac{\partial W}{\partial g} \, \dot{F} + 4 \, F \, \frac{\partial^2 W}{\partial g \, \partial g} \, F^T \, \dot{F}
\end{array} \right] \, dV = \int_{s} (P_n + \dot{P}_n) \, \delta u \, ds + (P + \dot{P}) \delta u
\]

Q Load Correction Vector
K Current Stiffness Matrix
P Total External Work

(3.5.5)
Strain-displacement functions are established to relate the strain quantities to the degrees of freedom, according to

\[ \delta g = B \delta u \quad \delta F^T = \delta u^T B^T \quad \dot{F} = B \dot{u} \] (3.5.6)

Substitution of 3.5.6 into 3.5.5 yields

\[ \int_\Omega \delta u^T B^T Q \, dV + \int_\Omega \delta u^T B^T K B \dot{u} \, dV = \delta u^T P \] (3.5.7)

\[ \int_\Omega B^T K B \dot{u} \, dV = P \int_\Omega B^T Q \, dV \] (3.5.8)

Where expressions for Q, K, and P in terms of the strain invariants \( I_1, I_2, I_3 \) are shown in reference [1]. The following assumptions are made in developing a model for the present study:

1. 3-D, Mooney-Rivlin hyperelastic material.
2. 8-node isoparameter F.E.
3. Non-linear strain-displacement relation
4. Non-Hookian material properties
5. F.E. model --- 432 nodes, 240 elements
6. Stress-strain relationships are identified under the assumption that the vessel wall and muscle tissue are stress-free at this unloaded state. (i.e., residual stresses will be neglected in this report)
Analysis will be performed to assess the influence of intramyocardial forces on embedded vessel deformation and stress contours only. Vessel pressurization will not be discussed in this report.

Because detailed constitutive information on cardiac muscle and vascular tissue is only partially available in the literature [14,18,29], we should assume an embedded blood vessel and its surrounding cardiac muscle behaves isotropically.

Data for the model described in the section 3.2 may now be generated in a standard PATRAN Plus modeling code (Release 2.2)[115]. ANSYS modeling codes are provided in Appendix A.

3.6 Summary

The influence of material differences between myocardium and embedded blood vessels will be assessed in terms of a parametric finite element analysis of different coefficients in the strain energy function. The Taylor series expansion of strain energy function in the linear form of two coefficients will be used in present approximation, where the coefficients $C_1, C_2$ are affected by the swelling condition.
The model considers a single, small caliber, blood vessel located midway across the ventricular wall. Vessel orientation is assumed parallel to the plane of cardiac muscle fibers and, with respect to local fiber orientation, the vessel long axis is oriented either perpendicular or parallel to the fiber. The ventricular stress field of Streeter et. al. [32] will be employed to determine the tissue stresses acting on the embedded blood vessel. Both the myocardium and vascular wall may be considered as swollen rubber due to their aqueous composition. Isotropy will be assumed as a first approximation to the more general transverse and cylindrical anisotropy exhibited by the myocardium and vascular wall, respectively.

Nonlinear finite element analysis will be used to determine stress and deformation contours in the model. A commercially available finite element code ANSYS Revision 4.3A/PATRAN Release 2.2 was used in this regard and numerical computations were performed on a VAX/VMS computer. Separate analysis will be performed to assess the influence of the vessel orientation, gel-like interface, and also intramyocardial forces on embedded vessel deformation and stress contours.
Figure 3.2.1  Top: the left ventricular wall is approximated by a truncated ellipsoid of revolution. Bottom: an arbitrary fiber in the ith layer, the principal radii of curvature of the layer, and various stress components of layers.
Figure 3.2.2 Top: segment of the myocardium containing a small caliber blood vessel which is acted by external loads. Bottom: directions of ventricular wall stresses in a model.
Figure 3.2.3  Finite element discretization scheme in the plane of the vessel cross-section; "gel-like" materials are considered between the vascular wall and the muscle fiber. The lower panel is one of the deformed types in chapter 4.
Statistical Theory

\[ W = C_{10} (I_1 - 3) \]

Mooney Theory

\[ W = C_{10} (I_1 - 3) + C_{01} (I_2 - 3) \]

3-Term Theory

\[ W = C_{10} (I_1 - 3) + C_{20} (I_1 - 3)^2 + C_{01} (I_2 - 3) \]

Hart-Smith Theory

\[ W = G \left[ \int e^{k_1 (I_1 - 3)^2} \, dI_1 + K_2 \ln \left( \frac{I_2}{3} \right) \right] \]

---

Figure 3.3.1  Equi-biaxial tension of a thin sheet of 8% sulphur rubber (From Alexander, 1968).
Figure 3.3.2 Uniaxial tension of a thin strip of 8% sulphur rubber (From Alexander, 1968).
Figure 3.4.1 Dependence of constant C2 on $v_2$ for various rubbers (From Treloar, 1975).
4.1 TEST CASES

The test cases described presently were conducted to validate the accuracy of the Mooney-Rivlin constitutive equation compared with closed-form results. Two test examples have been performed after the work of Argyris[132] and Oden[5]. The accuracy of standard ANSYS/PATRAN finite element code and model mesh configuration is inferred from the test cases.

Four Elements Test Cases

The first test represents a simple model using 4 elements to assess finite element accuracy when subject to uniaxial and biaxial loading. The model examined is shown in Figure 4.1.1 where a quarter sheet (i.e., due to symmetry considerations) model of 2*2*1 inch dimension is loaded equally in the X and/or Y directions by varying the imposed displacement from -0.5 to 2 inches. Under uniaxial tension/compression (T/C) and equibiaxial tension/compression (i.e., both are displacement control methods) we obtained reaction force-strain relationships as shown in Figure 4.1.2. The ordinate in Figure 4.1.2 represents values of elemental strain components (Exx, Eyy, Ezz) and stretch ratio (λ) whereas the abscissa depicts reaction forces.
The incremental method application of applied loads is a powerful method used to solve nonlinear systems of equations [5]. By sequential application of small load increments, the complete nonlinear response of the body is generated as a sequence of piecewise-linear steps. An alternative approach would be the incremental application of displacements which was used in this test case scenario. Uniaxial T/C was performed by applying 18 displacement increments of 0.1 inch in the X direction. At the final displacement increment reaction force equaled 120 lbs and the resulting strain in the Y-direction (Eyy) was 1.75. Equibiaxial T/C was carried out by simultaneously applying the same displacements as in the uniaxial case in the X as well as the Y direction. In a range of reaction forces comparable to the uniaxial case (0-120 lbs), the strain values of equibiaxial T/C manifest Exx=Eyy=0.8 (i.e. same stretch ratio in either direction) and Ezz=-0.7 (Figure 4.1.2). We note that for either uniaxial or biaxial loading protocols, the thickness and planar strain measures Ezz and Eyy respectively are equal (i.e., -0.3); while the planar shear strain Exy is identically zero.

If we assume the aforementioned test cases represent a planar LV wall segment (i.e., where X is the circumferential direction and Y is the longitudinal direction), with fiber orientation at +/-90 degrees from the circumferential equator of the LV, the characteristics of the force-strain curves from the two cases have qualitatively good agreements with physiological test cases [14, Figures 7.8].
Note On Hydrostatic Pressure

Horowitz [14] revealed that magnitude of tissue hydrostatic pressure (HP) under tension is small compared to the total tensile stress. However, when analyzing the coupling between the stress state in the ventricular wall and myocardial perfusion, the 3-D stress state in the solid tissue should be considered, in addition to consideration of HP. The HP may become a prominent factor affecting myocardial perfusion probably when considerable compressive loads are applied on the wall, such as in systole. Although HP is incorporated in the potential equation of hyperelastic materials, ANSYS does not provide plot capability for HP values. Data obtained from passive myocardial models, however, indicates that HP accounts for 8% and 13% of total stress in the fiber direction and cross fiber direction, respectively [18].

Influence of Mooney-Rivlin Constitutive Coefficients

We note that the control of the load-stretch ratio curve is totally dependent on $C_{10}$ and $C_{01}$ (referring to eqn. 3.1.4), the two Mooney-Rivlin constitutive parameters described in Sections 3.1 and 3.3. Additional test case evaluations were performed in biaxial loading where the Mooney-Rivlin coefficients were varied. In general, the material is softer in tension than in compression with respect to observed slope [1]. Figures 4.1.3 and 4.1.4 show load-stretch ratio curves under biaxial loading when "B" (i.e., the $C_{01}$ coefficient in eqn.3.1.4) equals a constant value of 6.96 psi and "A" (i.e., the
$C_{10}$ coefficient in eqn.3.1.4) is altered from 43.5 psi to 130.5 psi in three steps. The curves are qualitatively similar in Figures 4.1.3, 4.1.4, where it is seen that as "A" increases (by 100%, 150%, 200%, respectively), proportional increase in slope (100%, 150%, 200%, under the assumption of a straight line) of the tensile zone (i.e., $1<\lambda$) is also manifest. Similar phenomena are observed in the compression region (i.e., $0.5<\lambda<1.0$) of these figures. Hence, the initial slope of the load-stretch ratio relation (near $\lambda=1$) is proportional to the magnitude of "A". (i.e., in the segment of fairly linear slope)

Figures 4.1.5, 4.1.6 depict the 3-D equibiaxial T/C test case. Holding parameter "A" constant while proportionally increasing values of "B" (i.e. $0.2*A, 0.5*A, 1*A, 2*A$) yielded two changes to the load deflection curve (Figures 4.1.5, 4.1.6). The first is an increase in slope or stiffening of the material. The second is movement of the point of inflection (transition from decreasing to increasing slope) to lower tensile strain value [1]. Here the slope of the load stretch ratio curve is large in the compressive region and gradually decreases as the response shifts to tension. Once in tension the slope gradually increases.

The stiffness of the material varies directly with the parameter "A" throughout a typical T/C test. The material constant "B" is responsible for the gradual increase in slope that occurs in the tensile region. If "B"=0 the slope remains constant in the tensile region at strains exceeding 100 percent and reaction forces become very low compared to the aforementioned "A","B" values. This is referred to as "Neo-Hookean" behavior.
CHAPTER 4 COMPARISON & ANALYSIS OF RESULTS

Plate With A Hole Test Case

This second example demonstrates the numerical accuracy of the code by comparing ANSYS computations with the results of Oden's book [5]. We consider the problem of uniform stretching of a thin, initially square, homogeneous sheet with a hole at the center. The undeformed shape of this specimen is a 6.5 inch square planform which is 0.079 inch thick. The circular hole has a diameter of 0.5 inches. Segal and Klosner solved this problem by using a four-term strain-energy function—\( C_{10} = 20.28\) psi, \( C_{01} = 5.808\) psi, \( C_{02} = -0.72\) psi, \( C_{03} = 0.04596\) psi (referring to equation 3.1.4). These coefficients were determined from experiments on samples of a natural rubber. Figure 4.1.7 shows a finite element model which consists of 117 nodes and 96 elements. Approximate values of the first two constants \( C_{10} = 20.28\) and \( C_{01} = 5.808\) had been chosen in our 2-term Mooney-Rivlin constitutive equation.

In Figure 4.1.8 shows that agreement between ANSYS results and Mooney-Rivlin curves is excellent. A comparison of results from various constitutive forms of the strain-energy function (i.e., higher order polynomials or exponential-hyperbolic forms [5]) is also depicted in Figure 4.1.8. We note that the uniaxial response depicted in Figure 4.1.8 (also Figure 3.3.2) is relatively insensitive to the form of strain-energy density function. Consequently, uniaxial experiments of the type described here would likely be inadequate for characterizing a given material as we
mentioned in Section 3.2. Complete understanding of the constitutive coefficients needs to be found via simple elongation, pure shear, and equi-biaxial extension tests [131].

Figure 4.1.9 shows a typical stress contour $S_{XX}$ (i.e., element stress component in X direction) in a deformed elastic sheet together with a plot of deformed and undeformed plate geometry. The results are very similar to the classical linear elasticity — "the plate with hole problem". For the nonlinear problem, it is interesting to note that the stress concentration factor "$K$" (i.e., $K = \sigma_{\text{max}} / \sigma_{\text{nom}}$, where $\sigma_{\text{max}}$ is maximum stress, $\sigma_{\text{nom}}$ is the normal stress determined from the simple strength of material equations) is dependent on the deformation (i.e., $\lambda$) and the character of the material (i.e., $A, B$ values)[5]. In general, $K$ is proportional to the increase of stretch ratios. In this particular test specimen, $K$ equals to 5.0 at load step No.14. For linear theory, this factor is a constant (i.e., 3) and independent of stretch ratio but does vary with the plate thickness [116,133].

4.2 Analysis

The nonlinear finite element (NLFE) analysis of embedded blood vessel mechanics is discussed presently. Various material considerations and loading conditions will be addressed in the context of the physiological
situation being modeled. A three-dimensional finite element idealization of the embedded blood vessel was prepared specifically for analysis by the ANSYS computer algorithm. Computer generated graphical output was produced on an available Tektronix 4114 hardcopy device. The elements utilized were of the 8-node, isoparametric type subject to hyperelastic constitutive formulation. The constitutive coefficients (equation 3.1.4) used in present model are mainly from the experimental data of Canfield and Dobrin [28]. Blood vessels with homogeneous, isotropic, cylindrical, incompressible properties have been incorporated in their approach, therefore, the data are plausible at present study. The static response calculations required $2^{4}$ iterations and used $127\times150$ CPU minutes on a VAX 8700, under the VMS operating system. The load increments are 0.1 kpa to 0.2 kpa with 25 load steps.

The computer simulations produced output in the form of printed contours of radial stresses (SRR), tangential stresses (STT), axial stresses (SZZ), inplane shear stresses (TRT), out-of-plane shear stresses (TRZ, TTZ), as well as the deformed shape (Figures 4.2.1:1 through 4.2.3:7). These stresses are based on the cylindrical polar coordinate system (R, $\theta$, and Z) whose origin is at the intersection of three symmetry planes depicted in Figure 3.1.3. Polar coordinates, with initial $\theta=0$ at X axis, will be used in data presentation.

Intramyocardial stresses resulting from myocardial contraction and elevation of ventricular cavity pressure will be simulated in two loading
scenarios as we mentioned in Section 3.2. Intramyocardial stresses act on the embedded blood vessel model in perpendicular vessel orientation and in parallel vessel orientation. The model under the parallel loading scenarios is carried out by simultaneously applying the S1 and S3 loads (Figure 3.2.2) in the Y as well as Z directions. On the contrary, the perpendicular loading scenarios is performed by simultaneously applying the S1 and S2 loads in the Y and X directions respectively. "Parallel" vs. "Perpendicular" results are depicted in the upper panels (a) and the lower panels (b), respectively in all figures. In the following sections, the embedded blood vessel model will be classified as a uniform material, two materials without interstium (i.e., the gel-like interface), and two materials with interstium configurations.

4.2.1 NLFE of A Uniform Rubber Material Without Interface Under Physiological Loadings

Cross-sectional deformations of the uniform material under the influence of physiological loadings are depicted in Figure 4.2.1:1. The deformation scale is true size. The elliptical deformed lumen cross-section, with major axis parallel to the X direction, has a major to minor lumen axis ratio of 1.15 (i.e., 4.997/4.33) as shown in the upper panel, and 1.62 (i.e., 7.028/4.335) in the lower panel. A comparison of deformed area to its circular undeformed value (i.e., area ratio) gives a value of 0.865 in parallel vessel orientation and 1.218 in perpendicular vessel orientation. An area ratio less-than-one is indicative of a partially collapsed blood vessel.
Stress distributions in the plane of the vessel cross-section are depicted in Figures 4.2.1:2 through 4.2.1:7. In Figure 4.2.1:2(a) the maximum radial stress occurs at $\theta=90$ degrees with a value of 3.8 kpa at the vessel lumen. Stresses increase from -3.6 kpa to 3.8 kpa as $\theta$ increases from 0 to 90 degrees. Radial stress contours in the perpendicular fiber direction (Figure 4.2.1:2 (b)) are obviously under tensile forces with the maximum value 76 kpa at the vessel lumen, $\theta=90$ degrees. The tangential stress profiles of the two geometry configurations (Figure 4.2.1:3) are similar and also under compression. Peak compressive stress value is at the vessel lumen when $\theta=90$ degrees. The peak tangential stress concentrations are observed to exist in both cases when $\theta=90$ degrees, at the lumen. The axial stresses depicted in Figure 4.2.1:4 are moderately dependent on cross-sectional angular location. Note that the perpendicular vessel direction has a higher value in tension (i.e., 22kpa). In-plane shear stress contours (Figure 4.2.1:5) have maximal stress magnitudes occur at the lumen in both cases, with $\theta=22.5$ degrees and $\theta=67.5$ degrees in (a), (b) respectively. Shear stress contours exhibit higher density in the blood vessel wall than in muscle tissues. Out-of-plane shear stress quantities (TRZ, TTZ) have small values (less than 1 kpa) and have no influence on the vascular wall and its surrounding tissue (Figures 4.2.1:6, 4.2.1:7).
4.2.2 NLFE of Two Rubber Materials Without Interface Under Physiological Loadings

Cross-sectional distributions of radial, tangential, axial and shear stress components when the embedded blood vessel is considered as a different material form its surrounding tissue, without the existence of interface elements are shown in Figures 4.2.2:1 through 4.2.2:8. This model resembles embedded small arteries, arterioles, or other blood vessels which do not possess a more uniform attachment between the vessel and the surrounding tissue. Cross-sectional deformations resulting from the parallel and perpendicular geometric cases where undergoing physiological loadings are shown in Figure 4.2.2:1. The ventricular load scheme distorts the vessel lumen into an ellipse with a major to minor axis ratio of 1.17 (i.e., $4.911/4.19$) in (a), 1.62 (i.e., $7.4/4.545$) in (b). Lumen area ratio equals 0.823 in (a) and 1.345 in (b).

A comparison of radial, tangential, axial, and inplane shear stress contours under parallel and perpendicular loading condition are depicted in Figures 4.2.2:2 to 4.2.2:8. Radial stress contour at the vessel-cardiac muscle interface exhibit significant slope discontinuities (Figure 4.2.2:2). Such changes were observed whether parallel or perpendicular load schemes were employed. In contrast to the uniform rubber material data noted in the last Section, peak compressive and tensile stress locations in the two materials have a tendency to move at the material interface, however, they still occur in the same angular locations, $\theta=0$ degrees and $\theta=90$ degrees. Radial stress contours
in the vascular wall are markedly perturbed with respect to changes in material coefficients. This phenomenon was significant when the embedded blood vessel was orientated parallel to the local cardiac fiber direction.

Tangential stress contours are depicted in Figure 4.2.2:3. Both panels exhibit stress concentrations from approximately $\theta=0$ degrees to $\theta=40$ degrees, at the lumen as well as at the material interface. All stress values were attenuated when compared to those of a uniform rubber material (Figure 4.2.2:3). Radial and tangential stress through the tissue segment are largely compressive for vessels oriented along the muscle fibers (i.e., parallel) (Figures 4.2.2:2(a) and 4.2.2:3(b)). Such predominance of compressive stress was found earlier in the uniform material configuration (Figures 4.2.1:2 and 4.2.1:3). Radial stresses are largely tensile whereas tangential stresses are predominantly compressive for vessels orientated perpendicular to muscle fibers (Figures 4.2.2:2(b) and 4.2.2:3(b), respectively).

Axial stress contours (Figure 4.2.2:4) vary smoothly within the radial location $R<0.04$mm, however, the marked slope discontinuities near the vessel-myocardial interface were observed in the vascular wall. The width of disturbed-contour zone is about 0.01 mm in radius and the stresses within this zone are all under tension. The jagged stress profile is very apparent in the vessels oriented along muscle fibers. A noticeable change in the stress gradient occurs at the angular locations of $\theta=0-30$ degrees, $\theta=60-90$ degrees.
near the material interface when the vessel-fiber orientation is perpendicular. The stress magnitudes in the two rubber-material model are smaller than those in a uniform rubber material under myocardial loads.

Finite inplane shear stress contours are shown in Figure 4.2.2:5. Sharp discontinuities are observed in material interface when compared with Figure 4.2.1:5. Out-of-plane shear stress quantities TRZ and TTZ were also computed and depicted in Figures 4.2.2:6,4.2.2:7. Maximal stress value in TTZ has a tendency to increase and reach 2kpa when the vessel-fiber orientation is perpendicular.

4.2.3 NLFE of Two Rubber Materials With Interface Under Physiological Loadings

The model simulates the small caliber microvessels, such as capillaries which are attached to the surrounding muscle tissue through a gel-like medium. A comparison of radial, tangential, axial, and inplane shear stress contours under fiber and cross-fiber load conditions are depicted in Figures 4.2.3:1 to 4.2.3:7. The myocardial loading distorts the vessel lumen into an ellipse with a major to minor axis ratio of 1.26 (i.e., 5.1766/4.0892) and area ratio is 0.8467 which represents lumen constriction. In cross fiber direction, major to minor axis ratio is 2. The area ratio is equal to 1.17. The aforementioned data suggest that parallel vessels are partially collapsed while
perpendicular vessels exhibit initial lumen dilatation during myocardial loading. In Figures 4.2.2.1 and 4.2.3.1, the distortion comparisons of vessel lumen areas due to the existence of interstium, we note that the "gel-like" interface may alleviate the lumen area ratio in perpendicular vessel orientation (i.e., 1.345 to 1.17, respectively).

If we compare the results in Figures 4.2.2:2 and 4.2.3:2, the existence of the " gel-like" interface will substantially elevate stress gradients in the vascular wall and also elevate the stress values in the myocardium for either parallel or perpendicular geometric schemes. The maximal tensile radial stress occurs in $\theta=90$ degrees, $R=0.06\text{mm}$ in both loading conditions. The same angular locations are observed in Figure 4.2.3:2 with the exception of $R=0.05\text{mm}$ and smaller values. This phenomenon implies that the existence of the material interface amplifies the stress values and enlarges the disturbed-contour zone as well. Tangential stress contours as depicted in Figure 4.2.3:3 suggest compressive stress concentration at $\theta=0$ degrees, $R=0.06\text{ mm}$ in both load cases. Axial stress contours (Figure 4.2.3:4) exhibit strong tension arc with width from $R=0.04\text{ mm}$ to $0.06\text{ mm}$ and form a stress barrier across the vascular wall and the muscle fiber for vessels oriented along the muscle fibers. Such phenomenon is not obvious in cross-fiber direction. Inplane shear force reach 2.6 kpa near the material interace in (a) and -6kpa within vascular wall in (b). These peak values were observed in the vascular wall as well as in its surrounding tissue.
Figure 4.1.1 Equi-biaxial stretching of a 3-D sheet of Mooney-Rivlin material, comparison of undeformed type (upper panel) and deformed type (lower panel).
Figure 4.1.2  Load/strain responses of biaxial loading (upper panel) and uniaxial loading (lower panel).
Figure 4.1.3  Load/stretch ratio responses of holding B constant, increasing while proportionally increasing the values of A (see text).
Figure 4.1.4  Load/stretch ratio responses of holding B constant, increasing while proportionally increasing the values of A (see text).
Figure 4.1.5  Load/stretch ratio responses of holding A constant, increasing while proportionally increasing the values of B (see text).
Figure 4.6 Load/stretch ratio responses of holding A constant, increasing while proportionally increasing the values of B (see text).
Figure 4.1.7  
(a) Finite element representations of a quarter of a square sheet containing a circular hole. (from ANSYS 4.3A).  
(b) Undeformed and deformed finite element model (from Oden).
Figure 4.1.8  
(a) Total edge force in X direction versus stretch ratio (from ANSYS).  
(b) Force-stretch ratio relation with various forms of the strain energy function (from Oden's textbook).
Figure 4.1.9
(a) Stress contours in X direction (SXX) under the load step No. 14
(b) Displacement configurations under the load step No. 14
The deformed configurations for a uniform hyperelastic material subject to physiological loadings. Constitutive coefficients $C_1=15$ kpa, $C_2=1.5$ kpa were used as the first approximation. (a) parallel vessel orientation; (b) perpendicular vessel orientation.
Figure 4.2.1:2 Comparison of radial stress contours (SRR) generated by a uniform hyperelastic material subject to physiological loadings. (a) parallel vessel orientation; (b) perpendicular vessel orientation.
Figure 4.2.1: Comparison of tangential stress contours (STT) generated by a uniform hyperelastic material subject to physiological loadings. (a) parallel vessel orientation; (b) perpendicular vessel orientation.
Figure 4.2.1:4  Comparison of axial stress contours (SZZ) generated by a uniform hyperelastic material subject to physiological loadings. (a) parallel vessel orientation; (b) perpendicular vessel orientation.
Figure 4.2.1:5 Comparison of inplane shear stress contours (TRT) generated by a uniform hyperelastic material subject to physiological loadings. (a) parallel vessel orientation; (b) perpendicular vessel orientation.
Figure 4.2.1:6 Comparison of out-of-plane shear stress contours (TRZ) generated by a uniform hyperelastic material subject to physiological loadings. (a) parallel vessel orientation; (b) perpendicular vessel orientation.
Figure 4.2.1: Comparison of out-of-plane shear stress contours (TTZ) generated by a uniform hyperelastic material subject to physiological loadings. (a) parallel vessel orientation; (b) perpendicular vessel orientation.
The deformed configurations for uniform hyperelastic materials subject to physiological loadings. Constitutive coefficients $C_1=15 \text{kpa}$, $C_2=1.5 \text{kpa}$, $C_3=30 \text{kpa}$, $C_4=3 \text{kpa}$ were used as the first approximation. (a) parallel vessel orientation; (b) perpendicular vessel orientation.
Figure 4.2.2:2 Comparison of radial stress contours (SRR) generated by uniform hyperelastic materials subject to physiological loadings. (a) parallel vessel orientation; (b) perpendicular vessel orientation.
Figure 4.2.2:3  Comparison of tangential stress contours (STT) generated by uniform hyperelastic materials subject to physiological loadings. (a) parallel vessel orientation; (b) perpendicular vessel orientation.
Figure 4.2.2: Comparison of axial stress contours $$(SZZ)$$ generated by uniform hyperelastic materials subject to physiological loadings. (a) parallel vessel orientation; (b) perpendicular vessel orientation.
Figure 4.2.2:5 Comparison of inplane shear stress contours (TRT) generated by uniform hyperelastic materials subject to physiological loadings. (a) parallel vessel orientation; (b) perpendicular vessel orientation.
Figure 4.2.6: Comparison of out-of-plane shear stress contours (TRZ) generated by uniform hyperelastic materials subject to physiological loadings. (a) parallel vessel orientation; (b) perpendicular vessel orientation.
Figure 4.2.2:7  Comparison of out-of-plane shear stress contours (TTZ) generated by uniform hyperelastic materials subject to physiological loadings. (a) parallel vessel orientation; (b) perpendicular vessel orientation.
The deformed configurations for uniform hyperelastic materials subject to physiological loadings. Constitutive coefficients $C_1=15$ kpa, $C_2=1.5$ kpa, $A=20E-3$ kpa, $B=0$ kpa, $C_3=30$ kpa, $C_4=3$ kpa were used as the first approximation. (a) parallel vessel orientation; (b) perpendicular vessel orientation.
Figure 4.2.3:2 Comparison of radial stress contours (SRR) generated by uniform hyperelastic materials subject to physiological loadings. (a) parallel vessel orientation; (b) perpendicular vessel orientation.
Figure 4.2.3:3  Comparison of tangential stress contours (STT) generated by uniform hyperelastic materials subject to physiological loadings. (a) parallel vessel orientation; (b) perpendicular vessel orientation.
Figure 4.2.3: Comparison of axial stress contours (SZZ) generated by uniform hyperelastic materials subject to physiological loadings. (a) parallel vessel orientation; (b) perpendicular vessel orientation.
Comparison of in-plane shear stress contours (TRT) generated by uniform hyperelastic materials subject to physiological loadings. (a) parallel vessel orientation; (b) perpendicular vessel orientation.
Figure 4.2.3:6 Comparison of out-of-plane shear stress contours (TRZ) generated by uniform hyperelastic materials subject to physiological loadings. (a) parallel vessel orientation; (b) perpendicular vessel orientation.
Figure 4.2.3:7 Comparison of out-of-plane shear stress contours (TTZ) generated by uniform hyperelastic materials subject to physiological loadings. (a) parallel vessel orientation; (b) perpendicular vessel orientation.
5.1 Mechanical Influence of Interstitium

Finite element results in the two-material model using ventricular load schemes indicate radial and tangential stresses throughout the tissue segment are largely compressive for vessels orientated along the muscle fibers. One obvious location for a stress concentration is in the vessel lumen near the gel-like interface (Figures 4.2.3:2, 4.2.3:3, 4.2.3:4). The radial stresses are predominantly tensile when the vessel-fiber orientation is perpendicular (Figures 4.2.1:2 (b), 4.2.2:2 (b), 4.2.3:2 (b)). With the existence of the material interface, axial stresses in the three-material model exhibit strong tensile stress concentrations for vessels oriented along the muscle fibers (Figure 4.2.3:4). Radial and tangential stresses are dependent on angular location in the vessel cross-sectional plane, and vary partially with stress concentration in the myocardium close to the interface.

Axial stress contours depicted in Figures 4.2.2:4(a) and 4.2.3:4(a) have been generated under the assumption that axial fiber stress occurs in the cardiac muscle as well as on the embedded blood vessel. An alternative load scheme would apply this axial load only to the myocardium. Under this consideration, finite element data runs on parallel vessel orientations were performed in two models (i.e., Sections 4.2.2 and 4.2.3) and significant "stress jumps" between myocardium and the vessel wall does
exist as might be expected. However, stress patterns (i.e., SRR, STT, SRT, etc.) are similar and lumen deformations possess less than 3% error when compared to the data presented in Chapter 4. Axial stress contours, however, do exhibit differences as might be expected, and are depicted in Figure 5.1.1 for the three-material model.

Myocardial loads brought out by ventricular pressurization are observed to deform the embedded blood vessel such that its cross-section becomes elliptical. Comparison of deformed to undeformed lumen area indicates a net lumen constriction in the parallel-vessel direction and with a net dilation in the perpendicular-vessel direction (Figures 4.2.1:1, 4.2.2:1, 4.2.3:1). These data compare favorably with the baseline results of Kempski [10]. Perusal of the area ratio data in Section 4.2 indicates that the cross-sectional area of the model without interstium is large compared with that of the model with interstium in either parallel or perpendicular direction during systole.

5.2 Implication of Stress Concentrations on Local Oxygen Demand

As we mentioned in Chapter 1, Guyton [100] has shown that the resistance of an isolated perfused artery is directly dependent on oxygen tension (Po2) of the perfusion blood. The observations from Detar [116] also supported the possibility that Po2 plays a role in autoregulation of
local blood flow. His findings show that in the physiological range low oxygen tension is associated with diminished contractility and that contractility returns to normal when control levels of oxygen tension are reestablished. The relation of Po2 to contractile tension developed by rabbit aortic strips in response to epinephrine demonstrates the dependency of tension development upon Po2, as well as the complete reversibility of the contractility after the diminished response during hypoxia [116].

Possible explanations to account for the dependence of tension development on Po2 may involve nonmetabolic mechanism. In this category, one might consider mechanisms which associate oxygen tension with diminished activity of membrane excitation, of excitation-contraction coupling, or of the contractile machinery itself. The cardiac muscle cells of the vascular wall which have been observed in our studies (Figure 4.2.3:4, note that arteries convey blood only whereas capillaries have responsibility for oxygen exchange) in parallel direction are exposed to oxygen tensions below that in perpendicular direction, impeding the perfusion of oxygen. Under normal conditions, the Po2 of cardiac muscle cells of the vascular wall are normally below that of blood (i.e., capillaries blood). This will allow for both increase and decrease of vascular tone dependent on local changes in Po2. The regulatory system under Guyton's hypothesis might be initiated by a fall in perfusion pressure which would cause a reduced blood flow, and a lowering of Po2 in the vicinity of the smooth muscle cells. The observation will be more obvious and true.
through the presence of interface elements in longitudinal stress gradients (i.e., stress contours). The stress contours in Figure 4.2.3:4 provide a precise explanation in longitudinal gradients which mainly influence oxygen tension [117].

5.3 Additional Considerations and Suggestions for Further Research

Medical Aspects

The single most common cause of death is ischemic heart disease, which results from insufficient coronary blood flow. Approximately 35% of all deaths reported in the U.S. are due to this cause [100]. Some deaths occur suddenly as a result of an acute coronary occlusion (myocardial infarction) or of cardiac fibrillation, whereas others occur slowly as a result of chronic weakening of heart pumping efficiency (congestive heart failure). When the area of ischemia is large, some of the muscle fibers in the very center may die. Immediately around the dead area is a nonfunctional border zone of ischemic tissue. This phenomenon will decrease cardiac pumping efficiency even further degrading cardiac output (i.e., cardiac shock or cardiogenic shock). Coronary by-pass surgery has been used in the treatment of acute ischemia brought on by coronary artery stenoses. Complex tissue material properties may result in nonuniform heart contraction and hence influence intramyocardial tissue deformations [15].
Microvessels which transverse these regions of nonuniform contraction may experience tremendous stress gradients and/or "jump" discontinuities in stress level [100,117].

Scanning electron microscopy[99] reveals a "spiral configuration" of capillaries in the myocardium. This condition may give an added degree of freedom to capillaries undergoing axial loading, as the spiral configuration allows for modest deformation without appreciable stress development. An abnormal configuration such as ballooning of blood vessels (i.e., aneurysms) due to the fact that the collagen sheath breaks down, will cause a new boundary condition of vessel wall and also disturb CBF. These factors need to be incorporated in further research. An improved understanding of stress development in the LV wall incorporating the various complexities noted previously (Chapter 2, Summary) is paramount to determining the true nature of extravascular forces. Suitable verification between analytical and experimental results on embedded vessel patency needs to be conducted.

**Mechanical Aspects**

We presently assumed vascular and cardiac tissue to be isotropic, incompressible, and possessing nonlinear stress-strain relations. The hydrostatic pressure was incorporated in the ANSYS algorithm as a Lagrange multiplier of the incompressibility constraint on the strain field.
CHAPTER 5 DISCUSSIONS

The quantification of large caliber vessel nonlinear stiffness properties under the assumption of cylindrical orthotropy has been investigated [28] whereas limited information exists on microvessel and active cardiac fiber nonlinear stiffness properties.

Many different constitutive formulations for various tissues have been proposed [17,18,69,86]. One approach is to formulate constitutive relations, independent of structural considerations to fit experimental data reasonably well (i.e., Phenomenological Approach). Alternatively, one can formulate constitutive relations for tissue by accurately modeling their microstructure (i.e., Microstructural Approach). The results from such models can be compared with idealized behavior of tissues through a pseudostrain-energy function [86,103,107]. Here the pseudostrain-energy function can be explicitly determined from empirical data or can be "build-up" on the basis of limited structural information and qualitative experimental data [131]. Future advances in the area of cardiovascular biomechanics will allow further investigation of the true nature of myocardium-embedded vessel mechanics through finite element analysis.

In addition, a modified strain energy density function which incorporates anisotropic and compressible material properties is necessary to adequately mimic tissue properties. The anisotropic effects are illustrated in Kempski [10]. General considerations of anisotropy and compressibility on compliant material deformation have been documented by Peng[112], Green and Zerna [111], and Smith and Rivlin [102].
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APPENDIX A

ANSYS is a general-purpose finite element computer program for engineering analysis. It is developed, supported, and maintained by Swanson Analysis Systems, Inc. PATRAN Plus is an open-ended, general purpose, 3-D Mechanical Computer-Aided Engineering software package (MCAE) that uses interactive graphs to link engineering design, analysis and results evaluation function. The PAT/ANSYS interface provides a communication link between PATRAN and ANSYS. The PATRAN neutral file is the key element which simplifies the data exchange process between the two applications.

Appendix A consists of three programs which are relative to Chapter 4 data presentations. The data in the third program need to be modified in order to fit various test cases and boundary conditions. Interactive PATRAN codes and the translator file will be eliminated in appendix A due to their non-organized format. Revision 4.3 of the ANSYS program introduces two new elements that can be used to model rubber-like materials. These new elements are called hyperelastic elements. STIF84 is a two dimensional element that may have two representations: 1) plane strain and 2) axisymmetric. STIF 86 is a three dimensional solid element. These elements can be used to represent hyperelastic materials where solutions involve large deformations and large strains. Two strain energy density functions are provided to simulate the material behavior (i.e., only in Rev.4.3). The first, Mooney-Rivlin, is used to simulate nearly
incompressible behavior. The second, Blatz-Ko, is used for compressible foam type polyurethane rubbers.

These elements may be employed in static analysis (KAN=0) and nonlinear transient dynamic analysis (KAN=4) and require an iterative solution. The large deflection key must be activated (KAY(6)=1) to update coordinates during the solution phase. Temperatures may be applied but they are only used for material property evaluation. No thermal strains are computed. Precise summaries are shown in the beginning of each program.
/INTER
/PREP7
/TITLE, 3-D BIAXIAL C/E TEST A=43.5 B=87
C*******************************************************************************
C** EQUIBIAXIAL E/C TEST CASE
C** DISPLACEMENT CONTROL, DATE: 1988/9/19
C** FILE NAME: FILE29.ARGY
C** PROGRAMMER: SAMUEL I-EN LIN
C** 18-26 LOAD STEPS, CPUTIME = 93-160 SECOND
C*******************************************************************************
ET, 1, 86
KAY, 6, 1
EX, 1, 1
NUXY, 1, 0.49
NL, 1, 1, , 10 $, 1, 7, 43.5, 43.5 $, 1, 13, 87, 87
K, 1 $, 2, 1 $, 3, 1, 1 $, 4, , 1
KGEN, 2, ALL, , , , 0.5
V, 1, 2, 3, 4, 5, 6, 7, 8
LSSE, LINE, 9
LSAS, LINE, 5
LSAS, LINE, 7
LSAS, LINE, 11
LMOD, ALL, , , 1
LSALL
ELSI, , 2
VMESH, 1
SYMBC, , 1 $, 2 $, 3
NSEL, X, 1
CP, 1, UX, ALL
NSEL, Y, 1
CP, 2, UY, ALL
NSEL, Z, 0.5
CP, 3, UZ, ALL
NALL
/COM APPLY DISPLACEMENTS TO NODE WHICH BELONG TO ALL 3 COUPLED NODE SETS
NSEL, X, 1 $NRSE, Y, 1 $NRSE, Z, 0.5
*GET, NODE, NMAX
ITER, -15
NALL
D, NODE, UX, -0.5, , , UY
LWRITE
D, NODE, UX, -0.4, , , UY
LWRITE
D, NODE, UX, -0.35, , , UY
LWRITE
D, NODE, UX, -0.3, , , UY
LWRITE
D, NODE, UX, -0.2, , , UY
LWRITE
D, NODE, UX, -0.15, , , UY
LWRITE
D, NODE, UX, -0.1, , , UY
LWRITE
D, NODE, UX, -0.05, , , UY
LWRITE
D, NODE, UX, , , , UY
LWRITE
D, NODE, UX, 0.10, , , UY
LWRITE
D, NODE, UX, 0.25, , , UY
LWRITE D, NODE, UX, 0.5, ..., UY
LWRITE D, NODE, UX, 0.75, ..., UY
LWRITE D, NODE, UX, 1, ..., UY
LWRITE D, NODE, UX, 1.25, ..., UY
LWRITE D, NODE, UX, 1.5, ..., UY
LWRITE D, NODE, UX, 1.75, ..., UY
LWRITE D, NODE, UX, 2, ..., UY
LWRITE AWRITE, ..., 1
FINI
/EXEC
/INPUT, 27
FINI

/POST1
CSYS, 1
/VIEW, 1, 1, 0.3, 0.5
STRESS, STR, 86, 1  ---- STORE STRESS
SET,
/TYPE, 1, _
/DSCA, 1, _
/DIST, 1, _
PLDI, 2
/CLABEL, 1, 1
PLNSTR, SY

/POST26
TVAR, 1
DISP, 2, 13, UX, DISP
RFORCE, 3, 13, FX, RFOR
FILL, 4, ..., 1
ADD, 5, 2, 4, , LAMB, DA
PROD, 3, 3, , REACTION, , 1
GRID, 1
XLAB, _
YLAB, _
XVAR, 5  ---- LAMBDA
PLVA, 3  ---- REACTION
/GOPR
PRVAR, 2, 3, 5  ---- LIST DATA
FINI

/POST26  C** THE PROPERTIES OF BLOCK NO. 4
TVAR, 1
ESTR, 2, 4, 60, EPXX
, 3, 4, 61, EPXY
, 4, 4, 62, EPXY
, 5, 4, 63, EPZZ
RFORCE, 6, 13, FX, RFOR
DISP, 7, 13, UY $XVAR, 6 $YLAB, STRN $PLVA, 2, 3, 4 $PRVAR, 2, 3, 4, 5, 6

A-4
INTER, NO

[ 3-D ] TEST EXAMPLE SHOWN IN ODEN’S BOOK PAGE 313

DIMENSION 3.25*3.25*0.079

DATE: 1/17/89

ODEN’S MODEL ASSUMPTION SUMMARY

FILE NAME : FILE29.ODEN

REvised NUMBER: 4

PROGRAMMER: SAMUEL I-EN LIN

1. STATIC ANALYSIS, LARGE DEFLECTION ANALYSIS
2. 3-D HYPERELASTIC SOLID, ALL ELEMENTS HAVE EIGHT NODES
MATERIAL PROPERTIES ARE EVALUATED AT THE AVERAGE OF THE NODAL TEMPERATURES (IN THIS CASE 60F)
3. MOONEY-RIVLIN NEARLY INCOMPRESSIBLE MATERIAL
4. BASIC ELEMENT PRINTOUT
5. FULL INTEGRATION OF SHEAR TERMS AND REDUCED INTEGRATION FOR VOLUMETRIC TERMS OF STRAIN ENERGY.
6. THIS METHOD IS COMPUTATIONALLY MORE EXPENSIVE THAN THE FULL INTEGRATION, BUT MAY BE NECESSARY FOR PROBLEMS WHERE THE POSSION RATIO IS GREATER THAN 0.48 AND WHERE SIGNIFICANT HYDROSTATIC STRESSES ARE LIKELY TO DEVELOP IN ANY PORTION OF THE MODEL
7. DUMMY YOUNG’S VALUE = 1
8. POSSION’S RATIO = 0.49
9. 432 NODES, 240 ELEMENTS
10. 33000 BLOCKS, 14 LOAD STEPS WERE LIMITED, CPUTIME=70 MIN.
11. COMPATIBLE WITH ANSYS REV. 4.3 AND REV. 4.3A

*/PREP7

/TITLE, 3-D ODEN-313 C1=20.28psi C2=5.808psi

/HOMOGENEOUS MATERIAL IN THIS MODEL

KAY, 6, 1

ET, 1, 86, 0, 0, 0, 0, 0, 1
EX, 1, 1
NUXY, 1, 0.49
NL, 1, 1, 60 S, 1, 7, 20.28, 20.28 S, 1, 13, 5.808, 5.808

*/UNIAXIAL STRETCHING OF A SHEET WITH A CIRCULAR HOLE

*/LARGE DEFLECTION

TYPE, 1
MAT, 1
CSYS, 1
K, 1, 0.25, 0, 0 S, 2, 1, 0, 0
K, 3, 1, 90, 0 S, 4, 0.25, 90, 0
KGEN, 2, ALL,, , , , , , 0.079
V, 1, 2, 3, 4, 5, 6, 7, 8
LSSE, LINE, 9
LSAS, LINE, 5
LSAS, LINE, 7
LSAS, LINE, 11
LMOD, ALL,, , , 2
LSALL
LSSE, LINE, 3
LSAS, LINE, 10

A-5
LSAS, LINE, 6
LSAS, LINE, 1
LMOD, ALL, 5
LSALL
ELSI, 8
VMESH, 1
C***
TYPE, 1 $MAT, 1
KAY, 6, 1
K, 9, 1.225, 0, 0 $, 10, 1.225, 90, 0
K, 11, 1.225, 0, 0.079 $, 12, 1.225, 90, 0.079
V, 2, 9, 10, 3, 6, 11, 12, 7
LSSE, LINE, 15
LSAS, LINE, 20
LSAS, LINE, 13
LSAS, LINE, 17
LMOD, ALL, 1
LSALL
LSSE, LINE, 16
LSAS, LINE, 19
LMOD, ALL, 2
LSALL
ELSI, 8
VMESH, 2
C***
TYPE, 1 $MAT, 1
K, 13, 2.575, 0, 0 $, 14, 2.575, 0, 0.079
K, 15, 2.575, 90, 0 $, 16, 2.575, 90, 0.079
V, 9, 13, 15, 10, 11, 14, 16, 12
LSSE, LINE, 22
LSAS, LINE, 26
LMOD, ALL, 8
LSALL
LSSE, LINE, 24
LSAS, LINE, 27
LMOD, ALL, 2
LSALL
LSSE, LINE, 21
LSAS, LINE, 25
LSAS, LINE, 28
LSAS, LINE, 23
LMOD, ALL, 6
LSALL
VMESH, 3
C***
TYPE, 1 $MAT, 1
K, 17, 3.025, 0, 0 $, 18, 3.025, 90, 0
K, 19, 3.025, 0, 0.079 $, 20, 3.025, 90, 0.079
V, 13, 17, 18, 15, 14, 19, 20, 16
LSSE, LINE, 30
LSAS, LINE, 34
LMOD, ALL, 8
LSALL
LSSE, LINE, 32
LSAS, LINE, 35
LMOD, ALL, 2
LSALL
LSSE, LINE, 29
LSAS, LINE, 33

*LARGE DEFLECTION
**LARGE DEFLECTION**

**TYPE, 1**

**MAT, 1**

**CSYS, 0**

N, 406, 3.25, 0, 0
N, 407, 3.25, 0.6465, 0
N, 408, 3.25, 1.3462, 0
N, 409, 3.25, 2.1716, 0
N, 410, 3.25, 3.25, 0
N, 411, 2.1716, 3.25, 0
N, 412, 1.3462, 3.25, 0
N, 413, 0.6465, 3.25, 0
N, 414, 0, 3.25, 0

**NGEN, 3, 9, 406, 414, 1, 0, 0, 0.0395**
E, 406, 407, 355, 352, 415, 416, 383, 371
E, 415, 416, 383, 371, 424, 425, 376, 370
E, 407, 408, 356, 355, 416, 417, 384, 383
E, 416, 417, 384, 383, 425, 426, 377, 376
E, 408, 409, 357, 356, 417, 418, 385, 384
E, 417, 418, 385, 384, 426, 427, 378, 377
E, 409, 410, 358, 357, 418, 419, 386, 385
E, 418, 419, 386, 385, 427, 428, 379, 378
E, 410, 411, 359, 358, 419, 420, 387, 386
E, 419, 420, 387, 386, 428, 429, 380, 379
E, 411, 412, 360, 359, 420, 421, 388, 387
E, 420, 421, 388, 387, 429, 430, 381, 380
E, 412, 413, 361, 360, 421, 422, 389, 388
E, 421, 422, 389, 388, 430, 431, 382, 381
E, 413, 414, 354, 361, 422, 423, 375, 389
E, 422, 423, 375, 389, 431, 432, 374, 382

**BOUNDARY CONDITION**

**CSYS, 0**
SYMBC, 0, 1, 0
SYMBC, 0, 2, 0
SYMBC, 0, 3, 0
CPSIZE, 320
NSEL, X, 3.25
CP, 1, UX, ALL
NSEL, Y, 3.25
CP, 2, UY, ALL
NSEL, Z, 0.079
CP, 3, UZ, ALL

**COUPLED NODE SETS**

**NSEL, X, 3.25 SNSEL, Y, 3.25 $NRSEL, Z, 0.079**

**GET, NODE, NMAX**

**ITER, -15**

**NALL**

**D, NODE, UX, 0.2**

**LWRITE**
D,NODE,UX,0.4
LWRITE
D,NODE,UX,0.8
LWRITE
D,NODE,UX,1.0
LWRITE
D,NODE,UX,1.5
LWRITE
D,NODE,UX,2
LWRITE
D,NODE,UX,2.5
LWRITE
D,NODE,UX,3
LWRITE
D,NODE,UX,3.25
LWRITE
D,NODE,UX,3.5
LWRITE
D,NODE,UX,4
LWRITE
D,NODE,UX,4.5
LWRITE
D,NODE,UX,5
LWRITE
D,NODE,UX,5.5
LWRITE
D,NODE,UX,6
LWRITE
AFWRITE,,,,1
FINI
/EXEC
/INPUT,27
FINI

-----------------------------------------------

/POST1
/SHOW,9600,VT240 for ANSYS 4.3A /SHOW,240,,1,4
CSYS,0
STRESS,STR,86,1(OR 2,3)
SET,
/DSCA,1,1
/TYPE,1,2(OR /EDGE,1,1) FOR ANSYS 4.3A /TYPE,1,3 OR /TYPE,1,0
CSYS,0
/VIEW,1,1,1,1(CHANGE VIEWPOINTS)
PLDI,2 FOR ANSYS 4.3A PLDI,1 OR PLDI,3
/VIEW,1,0,0,0
/DIST,1,3
/TYPE,1,0
/DIST,1,?(ZOOM IN)
PLNSTR,SX

PLNSTR,SY,(SZ,SXY,SYZ,SXZ ETC.) USING /CONTOUR,1,10 IN SXZ AND SYZ
C***/CONTOUR,1,10(SET UP CONTOUR LINE NUMBER--->10)
C***FOR ANSYS 4.3A /CLAB,WN,-1, TURN OFF CONTOUR LABELS
C***
CSYS,1 (FOLLOW ABOVE STEPS,GET SRR,STT,STC...)

-----------------------------------------------

/POST26
C** THIS PART IS FOR DISPLACEMENT CONTROL
C** NOW THIS IS X DIRECTION "OK"
TVAR,1
DISP,2,428,UX,DISP
RFORCE,3,428,FX,RFOR
FILL,6,,3.25
ADD,7,2,6,,LAMB,DA,(DISP*1+50*1=7)
ADD,7,7,,LAMB,DA,,0.30769("7"=7*0.30769,LAMBDA VALUE)
PROD,3,,REAC,X-DI,,2
GRID,1
XLABEL,LAMB
XVAR,7 *(LAMBDA VALUE)
PLVA,3 *(REACTION FORCE VALUE)
PRVAR,2,3,7
FINI
C*** 1. STATIC ANALYSIS, LARGE DEFLECTION ANALYSIS
C*** 2. 3-D HYPERELASTIC SOLID, ALL ELEMENTS HAVE EIGHT NODES
C*** MATERIAL PROPERTIES ARE EVALUATED AT THE AVERAGE OF THE NODAL
C*** TEMPERATURES (IN THIS CASE 60°F)
C*** 3. MOONEY-RIVLIN NEARLY INCOMPRESSIBLE MATERIAL
C*** 4. ELEMENT COORDINATE SYSTEM IS PARALLEL TO THE GLOBAL
C*** COORDINATE SYSTEM
C*** 5. BASIC ELEMENT PRINTOUT
C*** 6. FULL INTEGRATION OF SHEAR TERMS AND REDUCED INTEGRATION FOR
C*** VOLUMETRIC TERMS OF STRAIN ENERGY.
C*** THIS METHOD IS COMPUTATIONALLY MORE EXPENSIVE THAN THE FULL
C*** INTEGRATION, BUT MAY BE NECESSARY FOR PROBLEMS WHERE THE POSITION
C*** RATIO IS GREATER THAN 0.48 AND WHERE SIGNIFICANT HYDROSTATIC
C*** STRESSES ARE LIKELY TO DEVELOP IN ANY PORTION OF THE MODEL
C*** 7. DUMMY YOUNG'S VALUE = 1
C*** 8. POSVION'S RATIO = 0.49
C*** 9. 432 NODES, 240 ELEMENTS
C*** 10. 43000 BLOCKS, 25 LOAD STEPS WERE LIMITED, CPUTIME = 140 MIN.
C*** 11. DISPLACEMENT CONTROL
C*** 12. USING TECH. "V" + "E" COMMANDS
C*** 13. COMPATIBLE WITH ANSYS REV. 4.3 AND REV. 4.3A
C***
C************************************************************
/PREP7
/TITLE, THREE MATERIALS C1=15E3 C2=1.5E3 A=20 B=0 C3=30E3 C4=3E3
/COM
KAY, 6, 1
C*** VESEL TISSUE
ET, 1, 86, 0, 0, 0, 0, 0, 0, 1
EX, 1, 1
NUXY, 1, 0.49
NL, 1, 1, 60 $, 1, 7, 15E3, 15E3 $, 1, 1, 3, 1.5E3, 1.5E3
C***
ET, 2, 86, 0, 0, 0, 0, 0, 0, 1
EX, 2, 1
NUXY, 2, 0.49
NL, 2, 1, 60 $, 2, 7, 30E3, 30E3 $, 2, 13, 3E3, 3E3
C***
ET, 3, 86, 0, 0, 0, 0, 0, 0, 1
EX, 3, 1
NUXY, 3, 0.49
NL, 3, 1, 60 $, 3, 7, 20, 20 $, 3, 13, 0, 0
C*** VESSEL MODEL
TYPE, 1
MAT. 1
CSYS, 1
K, 1, 5E-4, 0, 0 $, 2, 10E-4, 0, 0
K, 3, 10E-4, 90, 0 $, 4, 5E-4, 90, 0

A-10
KGEN, 2, ALL, 10E-4
V, 1, 2, 3, 4, 5, 6, 7, 8
LSSE, LINE, 9
LSAS, LINE, 5
LSAS, LINE, 7
LSAS, LINE, 11
LMOD, ALL, 2
LSALL
LSSE, LINE, 3
LSAS, LINE, 10
LSAS, LINE, 6
LSAS, LINE, 1
LMOD, ALL, 5
LSALL
ELSI, 8
VMESH, 1
C***
  TYPE, 3 $MAT, 3
  KAY, 6, 1
  K, 9.10, 25E-4, 4, 0, 0, 0, 10, 10, 25E-4, 90, 0
  K, 11.10, 25E-4, 4, 0, 10E-4, 0, 12, 10, 25E-4, 90, 10E-4
  V, 2, 9, 10, 3, 6, 11, 12, 7
  LSSE, LINE, 15
  LSAS, LINE, 20
  LSAS, LINE, 13
  LSAS, LINE, 17
  LMOD, ALL, 1
  LSALL
  LSSE, LINE, 16
  LSAS, LINE, 19
  LMOD, ALL, 2
  LSALL
  ELSI, 8
  VMESH, 2
C***
  TYPE, 2 $MAT, 2
  SKAY, 6, 1
  K, 13.20E-4, 0, 0, 0, 14, 20E-4, 4, 0, 10E-4
  K, 15.20E-4, 4, 90, 0, 0, 16, 20E-4, 4, 90, 10E-4
  V, 9, 13, 15, 10, 11, 14, 16, 12
  LSSE, LINE, 22
  LSAS, LINE, 26
  LMOD, ALL, 8
  LSALL
  LSSE, LINE, 24
  LSAS, LINE, 27
  LMOD, ALL, 2
  LSALL
  LSSE, LINE, 21
  LSAS, LINE, 25
  LSAS, LINE, 28
  LSAS, LINE, 23
  LMOD, ALL, 6
  LSALL
  VMESH, 3
C***
  TYPE, 2 $MAT, 2
  K, 17.40E-4, 0, 0, 0, 18, 40E-4, 4, 0, 90, 0
  K, 19.40E-4, 4, 0, 10E-4, 0, 20, 40E-4, 4, 90, 10E-4
  V, 13, 17, 18, 15, 14, 19, 20, 16

LARGE DEFLECTION
MUSCLE MODEL 1

LARGE DEFLECTION
MUSCLE MODEL 2
OUTER MUSCLE TISSUE

MUSCLE MODEL 3
*LARGE DEFLECTION

LSSE, LINE, 30
LSAS, LINE, 34
LMOD, ALL, , 8
LSALL
LSSE, LINE, 32
LSAS, LINE, 35
LMOD, ALL, , 2
LSALL
LSSE, LINE, 29
LSAS, LINE, 33
LSAS, LINE, 36
LSAS, LINE, 31
LMOD, ALL, , 2
LSALL
VMESH, 4

C***
TYPE, 2
MAT, 2
CSYS, 0

N, 406, 50E-4, 0, 0
N, 407, 50E-4, 9.9456E-4, 0
N, 408, 50E-4, 20.71067E-4, 0
N, 409, 50E-4, 33.40893E-4, 0
N, 410, 50E-4, 50E-4, 0
N, 411, 33.40893E-4, 50E-4, 0
N, 412, 20.71067E-4, 50E-4, 0
N, 413, 9.9456E-4, 50E-4, 0
N, 414, 0, 50E-4, 0

NGEN, 3, 9, 406, 414, 1, 0, 0, 10E-4

E, 406, 407, 355, 352, 415, 416, 383, 371
E, 415, 416, 383, 371, 424, 425, 376, 370
E, 407, 408, 356, 355, 416, 417, 384, 383
E, 416, 417, 384, 383, 425, 426, 377, 376
E, 408, 409, 357, 356, 417, 418, 385, 384
E, 417, 418, 385, 384, 426, 427, 378, 377
E, 409, 410, 358, 357, 418, 419, 386, 385
E, 418, 419, 386, 385, 427, 428, 379, 378
E, 410, 411, 359, 358, 419, 420, 387, 386
E, 419, 420, 387, 386, 428, 429, 380, 379
E, 411, 412, 360, 359, 420, 421, 388, 387
E, 420, 421, 388, 387, 429, 430, 381, 380
E, 412, 413, 361, 360, 421, 422, 389, 388
E, 421, 422, 389, 388, 430, 431, 382, 381
E, 413, 414, 354, 361, 422, 423, 375, 389
E, 422, 423, 375, 389, 431, 432, 374, 382

CSYS, 0
SYMBC, 0, 1, 0
SYMBC, 0, 2, 0
SYMBC, 0, 3, 0
CPSIZE, 320
NSEL, X, 50E-4
CP, 1, UX, ALL
NSEL, Y, 50E-4
CP, 2, UY, ALL
NSEL, Z, 10E-4
CP, 3, UZ, ALL
NALL

A-12
C***WFRONT,1
ITER,-50
CNVR,,1E-7,,1
C********************************************************************************
C*** LOAD STEPS
C*** THIS PART VARIES WITH DIFFERENT LOAD TYPES AND DEPEND ON
C*** DISPLACEMENT CONTROL, LOAD CONTROL, LOAD DIRECTIONS
C********************************************************************************
*CREATE,LOAD
PSF,0,2,50E-4,ARG1,,1E-4
PSF,0,3,10E-4,ARG2,,1E-4
LWRITE
*END
*USE,LOAD,0.1E3,-0.8E3
RP2,,0.1E3,-1E3
*USE,LOAD,0.5E3,-3E3
RP10,,0.2E3,-1E3
*USE,LOAD,2.586E3,-13.354E3
RP8,,0.25E3,-0.5E3
*USE,LOAD,4.586E3,-17.354E3
RP5,,0.25E3,-0.25E3
AFWRITE,,1
FINI
/EXEC
/INPUT,27
FINI

/SHOW1
/SHOW,9600,VT240 for ANSYS 4.3A /SHOW,240,,1,4
CSYS,0
STRESS,STR,86,1 (OR 2,3)
SET,__,
/DSCA,1,1
/TYPE,1,2(OR /EDGE,1,1) FOR ANSYS 4.3A /TYPE,1,3 OR /TYPE,1,0
CSYS,0
/VIEW,1,1,1,1(CHANGE VIEWPOINTS)
PLDI,2 FOR ANSYS 4.3A PLDI,2
/VIEW,1,0,0,0
/DIST,1,0,0.003
/TYPE,1,0
/EDGE,1,1(EDGE PLOT,AT THIS TIME MODEL IN FORCED CONDITION)
/DIST,1,?(ZOOM IN)
PLNSTR,SX
C***
PLNSTR,SY,(SZ,SXY,SYZ,SXZ ETC.)! USING /CONTOUR,1,10 IN SXZ AND SYZ
C*** /CONTOUR,1,10(SET UP CONTOUR LINE NUMBER-->10)
C*** FOR ANSYS 4.3A /CLAB,WN,-1, TURN OFF CONTOUR LABELS
C***
CSYS,1 (FOLLOW ABOVE STEPS,GET SRR,STT,STC...)
C**
C** SMALL REGION APPROACH ************
/DIST,1,10
(/CLABEL,1,1)
NRSEL,X,9E-4,11E-4
NRSEL,Y,0,90
ENODE
PLNSTR,SX
PLNSTR,SY
CSYS,0
PLNSTR,SX
PLNSTR, SY
NSEL, X, __,
(SELECT ANOTHER REGION ETC.)
FINI
FOR ANSYS 4.3A /ZOOM (DO ANY ZOOM IN OR ZOOM OUT WORK)
/ZOOM, 1, OFF OR (/AUTO)
-----------------------------------------------
/POST26
C** THIS PART IS FOR DISPLACEMENT CONTROLLED
C** NOW THIS IS Y DIRECTION "OK"
TVAR, 1
DISP, 2, 428, UY, DISP
RFORCE, 3, 428, FY, RFOR
FILL, 6, ..., 50E-4
ADD, 7, 2, 6, , LAMB, DA (DISP*1+50*1=7)
ADD, 7, 7, ..., LAMB, DA, , 0.02E4 ("7"=7*0.02, LAMBDA VALUE)
PROD, 3, 3, ..., REAC, X-DI, , 1
GRID, 1
XLABEL, LAMB
XVAR, 7 *(LAMBDA VALUE)
PLVA, 3 *(REACTION FORCE VALUE)
PRVAR, 2, 3, 7
C**
C** SEE WHAT HAPPEN IN Z-DIRECTION
C** USE THE SAME NODE-->428 THEN
C** REPEAT ABOVE UY-->428, FY-->FZ, 50E-4-->10E-4
C** 0.02E4-->0.1E4 EVERYTHING WILL BE "OK"
FINI
-----------------------------------------------
/POST26
C** THIS PART IS FOR PRESSURE CONTROLLED
C**
TVAR, 1
ESTR, 6, 240, 79, PRES C*** PRESSURE IN Y-DIR
ESTR, 8, 225, 79, PRES C*** PRESSURE IN X-DIR
ESTR, 9, , 79, PRES C*** PRESSURE IN Z-DIR
ESTR, 2, 8, 50, EPXX C*** ELEMENT #8 DEMO.
ESTR, 3, 8, 61, EPYY
, 4, 8, 61, EPZZ
, 5, 8, 63, EPXY
DISP, 7, 15, UY C*** NODE #15 TYPICITLY
GRID, 1
XVAR, 6
YLAB, STRN
PLVA, 2, 3, 4, 5
YLAB, DISP
PLVA, 7
PRVAR, 2, 3, 4, 5, 7
FINI
-----------------------------------------------
Constitutive Equation of Biomechanics (Elasticity)

The elastic model which has been studied in the report contains no mechanism for the dissipation of energy. Thus all work done in deforming an elastic body is recoverable. This work is said to be stored in the body as strain energy. Using the concept of strain energy, the entire theory of elasticity can be derived with the aid of work-energy principles. These principles are expressed in scalar form, and therefore they can provide us with a convenient tool for deriving the equations of elasticity in various curvilinear coordinate system. One can easily verify that the stress-strain relation through the study of an elementary, one dimensional example. Here I provide a general concept of verification.

* Special Coordinate

\[ T_{ij} = \sigma_{ij} \ n_j \]

Cauchy Stress \( \rightarrow \sigma_{ij} \) Actual force \( dP \) acts on the undeformed A

* Reference Coordinate (Pseudo Stress)

1st Piola-Kirchhoff Stress \( \rightarrow S_{ij}^1 \)

\[ T_{ij}^1 = S_{ij}^1 n_j \]

Actual force \( dP \) acts on the deformed \( A' \)

2nd Piola-Kirchhoff Stress \( \rightarrow S_{ij}^2 \)

\[ T_{ij}^2 = S_{ij}^2 n_j \]

Pseudo force \( dP^2 \) acts on the deformed \( A' \)

\( F \) represents deformation gradient

\[ F_i = \frac{\partial X_i}{\partial a_i} \] (i.e. \( F = \delta_{ki} + u_{ki} \)) curvilinear coordinate \( a_i \)
APPENDIX B

\[ dA = n_j dS \] undeformed area
\[ dA' = n' j dS' \] deformed area
\[ dP^2 = F^{-1} dP \]
\[ dA'_j = J^{-1} F^* A_j \]
\[ n_j dS' = J^{-1} F^* n_j dS \] \( \text{\textsuperscript{1}} \) see proof in [110]

1st Piola-Kirchhoff Stress

\[ T^1 dS' = dP = T dS \]
\[ (S'^1 n dS'^1) = dP = (\sigma n) dS \]
\[ S^1 n dS' = \sigma J F^{-1} n dS' \]
\[ S^1 = J F^{-1} \sigma \] or \[ S'^1_{ij} = J \partial a_i / \partial x_k \sigma_{kj} \]

2nd Piola-Kirchhoff Stress

\[ T^2 dS' = dP F^{-1} dP = F^{-1} T dS \]
\[ S^2 n dS' = F^{-1} (\sigma n) dS \]
\[ S^2 n dS' = F^{-1} \sigma J [F^{-1}]^T n dS' \]
\[ S^2 = J F^{-1} [F^{-1}]^T \sigma \]

or \[ S^2_{ij} = J \frac{\partial a_i}{\partial x_k} \frac{\partial a_j}{\partial x_l} \sigma_{kl} \]

The variation of the strain due to \( \delta u_i \) is \( \delta e_{ij} = 1/2(\delta u_{i,j} + \delta u_{j,i}) \) (i.e., under small deformation theory, we can drop nonlinear terms in equation 3.1.8).

Under the adiabatic condition, 1st law of thermodynamics says: during the variation \( du_j \), the variation of the work of the external force, \( \delta W_e \), is equal to the variation of internal energy, \( \delta U \), i.e., \( \delta W_e = \delta U \).
The same work can be done by using the first Piola-Kirchhoff stress tensor, with a subsequent mapping of stress from the 1st Piola-Kirchhoff stress tensor to the 2nd Piola-Kirchhoff stress tensor for reference.

\[
\text{Wext} = J_T \sum_{ij} \mathbf{a}_i \mathbf{a}_j + \sum_j \mathbf{f}_j \cdot \mathbf{u}_j \, \text{d}V
\]

\[
= \int \left( \mathbf{S}_{ij} \mathbf{a}_i + \mathbf{f}_j \mathbf{u}_j \right) \, \text{d}V
\]

\[
= \int \left( \mathbf{S}_{ij} \mathbf{a}_i + \mathbf{f}_j \mathbf{u}_j \right) \, \text{d}V
\]

\[
= \int \mathbf{S}_{ij} \mathbf{a}_i \, \text{d}V
\]

\[
= \int \mathbf{S}_{ij} \mathbf{a}_i \, \text{d}V
\]

\[
= \frac{1}{2} \int \det \mathbf{S} \, \text{d}V
\]

\[
= \frac{1}{2} \int \det \mathbf{S} \, \text{d}V
\]

\[
= 1/2 \int \mathbf{C}_{ij} \, \text{d}V
\]

\[
= 1/2 \int \mathbf{C}_{ij} \, \text{d}V
\]
The Justification Of Small Stretch-Ratio Value in Mooney-Rivlin Formulation

With the aid of $\lambda_1^{-1}=\lambda_2^2=\lambda_3^2$ (simple tensile deformation) and equation 3.1.13, we obtain,

$$T_{11} = 2\left(\lambda_1^2 - \lambda_2^{-2}\lambda_2^2\right)\frac{\partial W}{\partial I_1} - 2\left(\lambda_1^{-2} - \lambda_2^2\lambda_2^{-2}\right)\frac{\partial W}{\partial I_2}$$

$$T_{22} = 2\left(\lambda_2^2 - \lambda_1^{-2}\lambda_1^2\right)\frac{\partial W}{\partial I_1} - 2\left(\lambda_2^{-2} - \lambda_1^2\lambda_1^{-2}\right)\frac{\partial W}{\partial I_2}$$

We have $T_{11}=2(\lambda_1^2-\lambda_1^{-1})\left(\partial W/\partial I_1 + \lambda_1^{-1} \partial W/\partial I_2\right)$ and $T_{22}=0$

Let $\sigma^T = \frac{T_{11}}{2(\lambda_1^2-\lambda_1^{-1})}$ and $W=C_1(I_1^{-3}) + C_2(I_2^{-3})$

we have $\sigma^T=C_1 + \lambda_1^{-1}C_2$ at $\lambda_1=1$, $\sigma^T=C_1+C_2$

$$C_2 = \frac{\sigma^T}{\lambda_1^{-1}}$$

Thus $C_2$ is the slope of $s^T$ against $I_1^{-1}$, with a local minimum at $I_1=2$

(see Figure 1 in [103], data from simple tensile and biaxial tension). The slope between $\lambda_1=1$ to 2 is nearly linear, which imply $C_2$ is a constant.

Canfield and Dobrin [28] have used $G=30$ kpa for measuring pressure-radius data in dogs' arteries and obtained good agreement with experimental data when $\lambda<1.4$. 

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