Influence of atherosclerotic plaque on stress distribution of layer-specific coronary arteries in the presence of residual stress

MPhil Degree

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The effort made for this thesis, is dedicated to the spirit of my father to honor his short, super prolific life that allowed me to study at Imperial College London.
This work is my own and all else have been appropriately referenced.

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This work has been carried out in the Department of Biomedical Engineering at Imperial College London. There are some people that I would like to acknowledge for supporting me through these years. First, my supervisor Prof. Rob Krams (Imperial College) for giving me the opportunity to work at his lab and proposing this field of research for me. I also want to thank Dr. Ryan Pedrigi for the excellent review and comments he provided throughout my research.

My experience at Imperial College London gave me the chance to work with interesting problems for understanding the physics behind arterial mechanics, and in particular in the field of residual stress in tissues.

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Negin Pormehr
Abstract

Atherosclerosis is defined as the thickening of an artery due to the accumulation of fatty acids, cholesterol, and cellular debris within the blood vessel lining. This research aims to understand the influence of atherosclerotic plaque on residual stress within the arterial layers. Three layers of human coronary artery were studied under the influence of blood loading. Residual stress due to the opening angle and longitudinal stretch was included in the numerical model of the artery. A new method is used for modeling the effect of the opening angle on the distribution of stress. The finite element method was used for the numerical modeling of the artery in both healthy and atherosclerotic conditions. Under the blood loading condition, it was found that the presence of plaque produces higher stress in the arterial wall. This study allows us to investigate how the presence of the plaque affects residual stress and the overall stress distribution in the artery wall.
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Chapter 1. Introduction

1.1. Atherosclerosis as a Multi-Factor Inflammatory Disease

Atherosclerosis is defined as endothelial dysfunction and vascular inflammation, which is caused by the accumulation of fatty acids, cholesterol, and other cellular debris within the intima of large, and medium sized arteries (Wentzel et al, 2012). This leads to the formation of atherosclerotic plaques, especially in regions where arteries are branched or curved. Atherosclerosis is marked by chronic inflammation, lesions in the vessel walls, crystallized lipids and calcified cholesterol regions of necrotic tissue and accumulated leukocytes (Bonow et al., 2011). Contributing factors include both the morphology and physiology of the artery and the developing plaque itself. These can be confounded by the presence of genetic and environmentally associated risk factors such as the host immune response, the conditions present within the body, the person’s lifestyle, and the treatment response to either the disease itself or with the aim of reducing the risk from associated factors.

1.2. Stages of plaque development

Atherosclerotic plaques are morphologically classified based on their size and composition. Traditionally, plaques are identified by histology through the collection of biopsy samples, in-vivo imaging (Du et al, 2011, Lindsay et al, 2010) or post mortem (Burke et al., 2004). In recent times, less invasive imaging procedures have replaced the need to collect physical samples. The American Heart Association have suggested that there are six medically recognized categories of plaque.
• Phase I consists of the initial lesion, which contains isolated macrophages and macrophage foam in addition to minor accumulations of cholesterol.

• Phase II consists of an arterial thickening due to the sequestering of low-density lipoproteins. Phase II is called the ‘fatty streak’ phase, and also poses no specific danger to the patient (Maton et al., 1993).

• Phase III is called the Intermediate Lesion stage, consisting of further thickening of the intracellular lipid layer and the beginning of extracellular lipid pool accumulation as opposed to the isolated pockets of the previous phase. This phase is generally detectable with imaging technology, although clinical manifestations result in, at most, a negligible increase in blood pressure that is treatable should it become a health problem.

• Phase IV, however, moves past clinical silence and into the point where symptoms may or may not begin to show.

• Phase V, smooth muscle growth and the increase of collagen and fibrinogen (components of the extracellular matrix, or ECM) through protein secretion from afflicted smooth muscle endothelium cells bring the lesion into Phase V. Up until this phase, the plaque grows mainly via continuous lipid accumulation and the continual monocyte and macrophage recruitment response.

• Reaching Phase VI, the final phase, the plaque becomes known as a complicated lesion, and it is considered a serious health risk. While most plaques do not reach this phase, the number of plaques within a single individual in a developed country makes it statistically probable that at least
one of the plaques will reach this stage given a long enough lifespan and the presence of other risk factors (Bonow et al., 2011).

This schema has been used by many studies although, as others have pointed out, plaque progression may not be linear and sudden death can occur in patients where plaque pathology does not follow this progression (Virmani et al., 2011).

Atherosclerotic plaque, which is surrounded by endothelium, is a focal lesion formed by a lipid core covered in a fibrous cap.

Based on morphology of plaque, it’s characterized as stable or vulnerable. If there is a small amount of lipids and a large quantity of smooth muscle cells and collaged the lesion is “stable”. Stable plaques are protected from disturbance by its own structure, fibrous and homogeneous.

The critical cellular components of a plaque that will eventually characterize the atherosclerotic lesion involve interactions of endothelial cells, smooth muscle cells, platelets, and leukocytes such as macrophages, monocytes, neutrophils, and Natural Killer (NK) cells that are incompletely understood. Research into these interactions examines the traditional progression models of the disease, as will be discussed further.

However, some of the most cutting-edge research examines how to add molecular data to classical methods of clinical diagnosis based on numerical technique of likelihood, such as the Finite Elements Model (FEM). To analyze complicated problems with complicated domains (like biological environments) or when the domains have different material properties, FEM is a good choice (Zienkiewicz et al., 2005).
1.3. Relevant historical work on atherosclerosis models

Plaque formation is not uniform in all locations within the arterial vessel. Some locations in the arterial landscape are statistically more likely to form unstable plaques (Nazakawa et al, 2010). Once the vascular endothelium is activated, hemodynamic factors such as fluid shear stress play a key role in determining where plaques will form (Warboys et al., 2011). Fluid shear stress modulates gene expression levels at the sites of node formation and regulates levels of flow-sensitive proteins (Boudi et al., 2011). Image-based computational models in 3D, such as in vivo MRI, are critical mechanical indicators for determining risk of rupture (Teng et al., 2010). Areas with an increased incidence of plaque formation are usually within proximal or mid-proximal portions of a given arterial bed ((DeBakey et al., 1985). Areas with lower shear stress, such as parts of the carotid and outer lining of the coronary arteries are considered more vulnerable to unstable plaques (Helderman et al., 2007). Other identified common areas of atherosclerosis are the branching points of arteries, areas of marked curvature and irregular geometry, and those areas where blood flow changes or reverses direction or flow velocity (Boudi et al., 2011). Several research studies have examined the effect of low shear stress on the development of atherosclerotic lesions. Cheng et al., (Cheng et al., 2006) induced regions of lowered shear stress in mouse carotid arteries. They discovered that the expression of proatherogenic inflammatory mediators was higher in curved arteries near side branches where shear stress is lower. In addition, Cunningham and Gotlieb (Cunnigham and Gotlieb, 2005) and Slager et al., (Slager et al., 2005) have also reviewed the literature and discovered that vascular flow is atherprotective. Lowered flow leading to decreased shear stress alters the behaviour of the vascular endothelium, especially at arterial bifurcations (Cunningham and Gotlieb, 2005).
Lowering of shear stress aggravates and facilitates eccentric plaque growth (Slager et al., 2005).

Evidence has linked plaque rupture to plaque wall stress (PWS). Fluid structure interaction (FSI) models based on 3D MRI images, predicted that sites of ruptured plaques in the carotid artery (termed “ulcer nodes”) had 86% higher mean PWS than non-ulcer nodes (Tang et al., 2009). For these reasons, identifying susceptible arterial architecture has been a key focus for modern atherosclerosis treatment.

Vulnerable plaques that are at risk of rupture are treated by resection, balloon expansion, or stent angioplasty, all of which are high-risk procedures requiring quality surgical attention. Modern clinicians rely primarily on lumen X-ray angiography to determine the extent to which an artery is narrowing around a plaque in the coronary artery (Speelman et al., 2011). Older, less expensive techniques which included ultrasound plaque imaging and Doppler velocity measurements (Taylor and Strandness, 1987), have been replaced by more detailed 3D imaging techniques such as CT and MRI (Gao et al., 2011), which are also more expensive and time consuming. Lumen narrowing may not be the definitive parameter for diagnosing risk of cap rupture due to the level of vessel remodelling that is known to occur (Glagov et al., 1987). Therefore lumen stenosis is not significant enough to be a definitive characteristic of the potential of plaque rupture, thereby adding emphasis to the need for a correct diagnosis before invasive intervention.
1.4. Properties of vulnerable plaques

Plaques that match certain compositional criteria are termed “vulnerable”. These criteria include a thin fibrous cap, intraplaque hemorrhage, tissue inflammation, leukocyte infiltration (particularly macrophages), and few smooth muscle cells (Teng et al., 2010). Recently, the necrotic core has been added to the definition of a necrotic plaque based on recent studies that show a link between necrosis and rupture, pressurized geometries, and myocardial infarction (Speelman et al., 2011).

Vulnerable plaques also have altered responses to normal paracrine stimulants, autacoids, and vasodilators (Kuo, Davis, and Chilian, 1995), complicating the effects of early and initial treatments that are aimed at helping to initiate the body’s natural defenses. About 60% of all cerebral infarctions are the direct result of the rupturing of a vulnerable plaque (Casscells, Naghavi, and Willerson, 2003), and rupture-prone vulnerable caps, most often fibroatheromas, are the most common cause of arterial thrombosis (Slager et al., 2005).

1.5. Using Finite Elements for arterial modeling

Modern technology and research findings have uncovered patient-specific estimators of rupture risk, taking into account CT-data, MRI data, shear stress and total artery geometry, which are used to calculates arterial stress and strain. The computed stresses/strains are used to infer risk of plaque rupture (Speelman et al., 2009). Current methods using MRI can pinpoint critical mechanical indicators of the risk of plaque rupture, such as critical plaque wall stress and 3D fluid structure interaction that make an even more sensitive diagnosis possible (Teng et al., 2010). Chu et al. (Chu et al., 2006) were the first to add precision and accuracy to this analysis through their breakthrough technique of high-spatial-resolution multi-
contrast MRI of ruptured plaques at ulcer nodes and its correlation with diagnosis and patient outcome. All of this specific and statistically relevant information is taken into account in the FEM, as well as other tools such as the multi-layer isotropic model and plaque geometry correlations with PWS. For this reason, a computation using the FEM is a highly sensitive, patient-specific analysis of clinical risk (Trujiers et al., 2007). The FEM uses

1. Complete plaque geometry to model plaque development,
2. The blood pressure of the patient as an indicator of risk, and
3. The mechanical properties of the plaque constituents to be the so-called finite element determinants of the level of stress placed on the plaque cap (Speelman et al., 2011).

1.6. Effect of stress distribution

Researchers first quantitatively identified mechanical stress and residual strain in the arteries in the late 1980s. The details of such work were compiled and examined in a lecture by Y.C. Fung (Fung, 1991). The work of his group challenged the long-standing hypothesis that without any external traction on a tissue such as an arterial vessel, the tissue would be stress-free.

Cheruvu et al. (Cheruvu et al., 2007) made new headway with the FEM by examining the frequency and distribution of ruptured plaques in coronary arteries. Examining coronary arteries at post-mortem, they found 20 of 50 (40%) to contain at least one plaque that was ruptured and that for each patient there were about two plaques present in each heart. The plaques themselves accounted for <3% of the total coronary area, and were infrequent and focally distributed within individuals. The
data mirrored statistical findings within the population at large, demonstrating that the lesions themselves are relatively common in Western populations, although those that lead to death are relatively rare. The study presented a landmark case for distinguishing plaque type based on geometry and other characteristics.

Early in 2011, Tracqui et al. studied plaque development using FEM (Tracqui et al. 2011). The concept of the experiment comes from the hypothesis that the evolution of mechanical stress on the atherosclerotic plaque determines its morphological development and ultimately its vulnerability to rupture. Plaque remodeling at the intima, media, and/or adventitia layers, according to previous data, was related to the plaque’s developing vulnerability.

1.7. Description of current work using the FEM

This research continues the line of investigation on mechanical stresses and aims to describe the effect of the existence of a plaque on the arterial stresses with special reference to the different arterial layers. A novel method was used to consider the effect of residual stress. With further examination of the existing stresses the FEM could provide one of the most accurate and reproducible means of measuring these differences in order to aid clinicians in making a diagnosis.

One of the keys to the hypothesis is the opening angle to the artery. The opening angle (OA) is a geometric term that refers to the angle at the vertex of an arc if it were cut in cross section. In the case of the arteries under examination, the opening angle is a precise parameter that directly indicates the residual stress in the vessel (Han et al, 2006). The open angle of the artery is typically measured by taking short segments of artery and making radial cuts in the artery wall such that the rings ‘pop’ open into a stress-free stable structure. The angle subtended by the medial wall
and the two cut pieces is the opening angle (Han et al, 2006). Research has also shown how important arterial openings are to the FEM, including research by Saini et al. (Saini et al., 1995). In that study of patients with ages ranging from 3 months to 87 years, Saini et al. concluded that the opening angle of the aorta increased with age, and that patients with atherosclerosis had a significantly larger opening angle. The study also found that the significantly larger opening angles were at all ages larger in males than in females, consistent with concurrent findings that the opening angle is an associated factor in age- and sex-related differences in the incidence of atheroscleroma.
Table 1.1. demonstrates a list of selected previous studies for analysis of arterial residual stress. Experiment-based methods are studies that are based on laboratory experiments on in-vitro samples. This table includes a summary of each method, advantages, disadvantages, effective parameters and limitations of each study.

### Table 1.1. List of literatures of experiment-based methods for measurement of residual stress in the artery.

<table>
<thead>
<tr>
<th>References</th>
<th>Method</th>
<th>Effective parameters</th>
<th>Advantages</th>
<th>Disadvantages</th>
<th>Limitations</th>
<th>Conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chuong and Fung (1986)</td>
<td>First estimation of RS with OA</td>
<td>Geometry, opening angle</td>
<td>First study to report a 3D non-linear anisotropic relation based on residual stress</td>
<td>Cut specimens rarely take the form of circular segments.</td>
<td>The ring is suspended</td>
<td>Residual strain and stress calculated</td>
</tr>
<tr>
<td>Rehal, Guo, Lu &amp; Kassab (2005)</td>
<td>Measuring viscoelastic properties.</td>
<td>Opening angle</td>
<td>Variability in opening angles</td>
<td>The parameters are valid for viscoelastic models.</td>
<td>Duration of no-load state changes opening angle</td>
<td></td>
</tr>
<tr>
<td>Li and Hayashi (1996)</td>
<td>added the edge angle to the formulation</td>
<td>Opening angle</td>
<td>Two stress components are calculated.</td>
<td>More precise measurement</td>
<td>the OA may be in error if the edge angle is not small</td>
<td>Important in thick walled arteries</td>
</tr>
<tr>
<td>Han and Fung, (1996)</td>
<td>measure residual strains using black ink micro-dots</td>
<td>Opening angle, Micro-dot ink</td>
<td>Higher resolution of strain.</td>
<td>Still invasive method</td>
<td></td>
<td>Residual strain is negative in inner layer</td>
</tr>
<tr>
<td>Okamoto et al (2002)</td>
<td>Measured with hyperelastic property</td>
<td>Opening angle</td>
<td>Including hyperelastic property</td>
<td>Only two components of residual stress calculated</td>
<td>Increase number of aortas studies across a variety of ages</td>
<td>OA was greater in patients older than 50 years</td>
</tr>
<tr>
<td>Holzapfel (2007)</td>
<td>Layer specific model is developed</td>
<td>Opening angle, Modified to different layers</td>
<td>Analyzed in different layers over time</td>
<td>Study is done in large artery (aorta). Only strain</td>
<td></td>
<td>Three dimensional OA</td>
</tr>
<tr>
<td>Bustamante &amp; Holzapfel (2010)</td>
<td>Two model to reconstruct 3D residual stress</td>
<td>Opening angle Modified to 3D multi layers</td>
<td>Stress components analyzed</td>
<td>Complicated partial differential equations difficult to solve</td>
<td>Complexity of mechanical equations</td>
<td>Residual stresses influence arterial develop</td>
</tr>
</tbody>
</table>
In 1986, Chuong and Fung (Chuong et al 1986) introduced residual stress as an important factor that affects the stress distribution within an artery. After that, different methods have been proposed to estimate the arterial residual stress. Some of the methods to estimate the arterial residual stress are based on laboratory experiments, which are listed in Table 1.1. Some other studies are based on physical rules that are listed in Table 1.2. Also, simulation and computational methods provide tools to estimate the residual stress within an artery.

**Table 1.2. List of literatures of rule-based methods for measurement of residual stress in the artery.**

<table>
<thead>
<tr>
<th>References</th>
<th>Method</th>
<th>Assumptions</th>
<th>Effective parameters</th>
<th>Advantages</th>
<th>Dis-advantages</th>
<th>Limitations</th>
<th>Conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Takamizawa and Hayashi (1987)</td>
<td>First study to propose using physical rules to estimate arterial residual stress</td>
<td>Model based calculation</td>
<td>Physical rule</td>
<td>Is not invasive. 3d distribution estimated.</td>
<td>Based on hypothesis is valid only in circular conducts.</td>
<td>From in vitro measurements.</td>
<td>Residual stresses and strains exist within the wall without external loads.</td>
</tr>
<tr>
<td>Guo, Kassab (2004)</td>
<td>studied both physical rule and opening angle with opening higher than 180 degree</td>
<td>Validated with the opening angle method based on thin wall tubes</td>
<td>Physical rule Opening angle</td>
<td>Improve the physical rule for opening higher than 180 degree.</td>
<td>Assumes a thin wall vessel. Same as the Takamizawa et al (1987)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Labrosse, Beller, Mesana &amp; Veinot (2009)</td>
<td>Analytical, experimental and 3D FEM of human aortas conducted to include residual stress and accompanying material constants</td>
<td>Analytical models as pressurization axisymmetric, thick walled, closed cylinder homogenous hyperelastic anisotropic incompressible material</td>
<td>Physical rule Opening angle</td>
<td>Shed light onto the mechanical properties of the aorta.</td>
<td>Results only meaningful for closed-end physiologic pressures. Small sample size, age restricted.</td>
<td>Studying the consequences of negative opening angles. Increase number of aortas studies across a variety of ages.</td>
<td>Close match between experimental, analytical and finite element analysis.</td>
</tr>
</tbody>
</table>
This research aims to further the understanding of atherosclerosis through perfecting the FEM. Specifically, the focus will be on how residual stress affects the stress distribution from loadings from blood pressure in the presence of plaque. This will be accomplished by implementing both aspects of residual strain and external loads independently and in the integrated form. A realistic material constitutive model employing, a layered geometry of the arteries is used. The next step is to build two different FEMs. In one of them the material of different arterial layers will be considered. In the second model, the atherosclerotic plaque will be added to the arterial model.
Chapter 2. Materials and Methods

Biomechanical tools were used to develop a model to study the effect of the residual stress distribution within the artery. Healthy and atherosclerotic coronary arteries were studied within the mid-LAD coronary artery system. This section of the coronary system has a relatively straighter cylindrical shape and is especially prone to atherosclerotic disease because of its proximity to an arterial branch. This coronary artery has been studied under two opening angles and longitudinal residual strain conditions. The effect of blood flow load was also integrated in two healthy and diseased coronary artery models, which consisted of straight tubes of 8mm lengths.

2.1. Analytical solution for residual stress in normal artery

The cylindrical shape of a healthy artery makes it easy to study the stress distribution within the cylindrical coordinate system. Residual stress due to the opening angle is studied in this coordinate system. In the cross section of the healthy artery, the stress tensor will be presented by three components, $\sigma_r$, $\sigma_\theta$ and $\tau_{r\theta}$. $\sigma_r$ is the radial component of the stress tensor, which is perpendicular to the symmetry axis, $\sigma_\theta$ is the circumferential component of the stress tensor which represents the normal stress in the tangential direction. $\tau_{r\theta}$ is the shear stress. In the unloaded condition, where there is no blood pressure on the inner wall of the artery, $\sigma_r$ and $\tau_{r\theta}$ are zero in both the inner surface and the outer surface. Since arterial thickness remains constant, it can be assumed that the variation in the layers is negligible.
Figure 2.1. Schematic of the distribution of residual stress components producing the opening angle. With a 90-degree cross-section of the artery, there are only compressive and tensile components that produce the opening angle.

In a closed artery, residual stress in the cross section of the wall has both compressive and tensile circumferential components. Figure 2.1 shows stress distribution in a cross-section of a healthy artery. In the absence of blood pressure, due to the equilibrium condition, the integral of the stress components in a cross section of the healthy artery is zero.

Based on the equilibrium, the summation of the compressive and tensile stress is zero, therefore, the integral for compressive stress will be equal to the tensile stress. This is shown as the following equation:

\[
\int_{r}^{r+h} \sigma_0^c \, ds + \int_{r}^{r+h} \sigma_0^t \, ds = 0
\]

Here \( \sigma_0^c \) and \( \sigma_0^t \) are the compressive and tensile residual stress components in the artery wall, respectively. \( ds \) is an element along the radial direction situated in the
arterial wall. This relation also implies that there is a *neutral axis* in the artery wall, which is neither compressed nor extended. Due to zero stress, if this neutral axis has a radius $\rho$ in the closed configuration of the artery, its length is equal to the stress-free state after deformation.

Due to equilibrium in geometrical symmetry in a healthy artery, stress distribution will remain constant in the circumferential direction. Since the cylindrical artery’s cross-sections remain in-plane and perpendicular to the neutral axes, after opening the vessel longitudinally the artery’s wall deforms following an arc of a circle.

This can be shown mathematically by considering the similarity in the deformed elements on the artery wall. For example, when considering a deformed artery after a longitudinal cut at time $t_0 + dt$ (Figure 2.2.b) with an opening angle $d\alpha$, the length of the neutral axis will remain constant since it is stress free. In an element of the artery wall with constant neutral segments $AB=BC$, due to the perpendicular planes, they will form a part of the arc of a circle. We need to show that the center of these two arcs will coincide after deformation. Due to the constant circumferential stress distribution, a similar distribution will be expected in the circumferential direction. Then after deformation, similarity of the strain in the wall presents $A_1B_1=B_1C_1$ and $A_2B_2=B_2C_2$. This proves the coincidence of the center in two circles. This is valid in all infinitesimal elements of the artery wall. In other words, the neutral axis after deformation will present an arc of a circle.
Figure 2.2. Neutral axis and sectional deformation in the cross-section of a cylindrical healthy artery (a) before and (b) after a longitudinal cut. (c) represents the opening angle definition in the cross section of the artery. The opening angle is due to the residual stress in the cross section of artery.

If the radius of the neutral axis in the deformed configuration is defined by a radius $\rho$, after an opening angle $\alpha$, we will have the following relation (see Figure 2.2.c):

$$2\pi \rho = \rho'(2\pi - 2\alpha)$$

(2)
\[ \rho' = \frac{\pi}{(\pi - \alpha)}\rho \]  

For a small element of artery with a constant length of L and angle of \(d\theta\) before the radial cut, as is shown in Figure 2.2.a, there will be an angle of \(d\Theta\) after the longitudinal cut. Assuming that the length L remains constant, since the length of the neural axis AB will remain constant (see Figure 2.2.b), we will have the relation below:

\[ AB = \rho d\theta = \rho' d\Theta \]  

from (3) and (4) we have

\[ d\Theta = \frac{\pi - \alpha}{\pi}d\theta \]  

Now to calculate the strain distribution in the wall we turn to the extension and contraction of a differential line element lying off the neutral axis with distance s, say the element CD in Figure 2.3. CD here is presenting the line in stress free state and C'D' in the deformed and stressed condition. The circumferential strain is defined by:
which yields:

\[
\varepsilon_\phi = \lim_{d\theta \to 0} \frac{C'D' - CD}{CD} = \lim_{d\theta \to 0} \frac{(\rho - s)d\theta}{(\rho' - s)d\theta} - 1
\]  

(6)

which yields:

\[
\varepsilon_\phi(s) = \frac{-\alpha s}{\pi(\rho - s) + \alpha s}
\]  

(7)

The strain varies non-linearly with \(s\); the elements at the inner radius of the artery are in compression, those outside the neutral axis in tension. \(s\) is defined in the radial direction toward the center of the cylinder and its value is zero at the \((0,0)\) position of the neutral axis.

Figure 2.3. An infinitesimally small element from the artery wall with small arc angle \(d\theta\) in the closed form. Part (b) represents the same part in the stress free condition when there is similar length of neutral axis AB.
For development of the analytical benchmark for this study, linear-elastic material properties have been assumed for the arterial wall, which simplifies the stress-strain relationship in the equations. This analytical benchmarking demonstrates some parameters used in the models such as the mesh size and boundary conditions. The next step is to apply non-linear material properties to the models. Circumferential stress-strain relationship in the artery wall will be as follows:

\[ \sigma_\theta = G \varepsilon_\theta \]  

(8)

Which allows us to obtain:

\[ \sigma_\theta = \frac{-G\alpha s}{\pi (\rho - s) + \alpha s} \]  

(9)

\( G \) is the Young’s modulus. In order to find the neutral axis position, the equilibrium relation in equation (1) was used. By applying \( \sigma_\theta \) from the equation (9)
into the equation (1), the integration of this equation from \( r = r \) to \( r = r + h \) will result in:

\[
h = \frac{\rho \pi}{\alpha - \pi} \left[ \ln \left( 1 + \frac{h(\alpha - \pi)}{\alpha \rho - (r + h)(\alpha - \pi)} \right) \right]
\]  

(10)

Solving this equation will result in the value of \( \rho \) that is necessary to find the distribution of circumferential stress values in equation (9).

2.2. Normal coronary artery model

The geometry of the healthy coronary artery was extracted from experimental data of the human coronary artery (Holzapfel et al. 2005). Figure 2.4 shows a schematic of the three-layer geometry configuration of this model. In those experiments, the average shape of the coronary artery was measured in 13 human cases. Given those data, an outer diameter of 4.5 ± 0.3 (SD) mm and a ratio of wall thickness to radius 0.189 ± 0.014 (mean ± SD) is used. The inner and outer radii of the model were 1.825mm and 2.25mm, respectively.

The coronary artery wall was modelled as a three-layer geometry consisting of the intima, media and adventitia. Again from measured geometrical data (Holzapfel et al. 2005) the ratio of adventitia, media and intima thickness to wall thickness were used 0.40 ± 0.03, 0.36 ± 0.03, and 0.27 ± 0.02 (mean ± SD) respectively. Mean values
were used to identify the thickness of each region in the wall. The length of 8mm was assigned for the longitudinal geometry of the artery. This length corresponds to the lengths of samples that are used for experiments with atherosclerotic arteries (Ohayon et al. 2007) and are used because this gives a relatively straight, workable length of artery, which is convenient to model. These will be used for comparison with the diseased arteries in the next section.

![Figure 2.4. Schematic of the cross-sectional geometry of the coronary artery and the related layers in the model.](image)

As mentioned before, the coronary artery in the unloaded configuration has residual stresses. This stress is usually measured as the residual strain caused by cutting the artery in its longitudinal direction. The resulting opening angle is used here to estimate the stress-free configuration in the model. A method to estimate the stress-free configuration of the coronary vessel is proposed. To estimate the stress free configuration the following assumptions were made:
1. The artery wall in its stress free configuration after opening angle has the same curvature at all points with an arc of radius R.

2. The artery wall in its stress free configuration after opening angle has the same thickness at all points.

3. Material in the tissue is incompressible. In other words, assuming constant density in the tissue a constant volume of tissue during deformation was assumed.

Figure 2.5 shows a schematic of the coronary artery wall in the stress free configuration as is modeled in this study. Thickness $h$ consists of three coronary layers as explained previously. Opening angle $\alpha$ is defined as the input data given from the experiments.

Due to the continuity of the mass, with a homogenous density, the volume of the artery wall will be equal to the configuration before the cut. Then:
This relationship will result in:

\[
\pi((r + h)^2 - r^2) = \frac{\beta}{2}((R + h)^2 - R^2)
\]  \hspace{1cm} (11)

\[
\pi(2r + h) = \frac{\beta}{2}(2R + h)
\]  \hspace{1cm} (12)

From relations for a circle and its arc angles one can show that in the configuration shown in Figure 2, the following relation is valid between opening angle \( \alpha \) and arc angle \( \beta \):

\[
\beta = 2(\pi - \alpha)
\]  \hspace{1cm} (13)

(12) and (13) results in

\[
R = \frac{\pi(2r + h) - h}{2(\pi - \alpha) - \frac{h}{2}}
\]  \hspace{1cm} (14)
This equation estimates the radius $R$ in the stress-free configuration. Here, $h$ is wall thickness, $r$ is inner radius for the closed artery, and $\alpha$ is opening angle. Here the stress-free state of the arterial lumen is modelled with an opening angle of $\alpha = 118$ degrees which is taken from the literature (Ohayon et al. 2007). This opening angle is a mean value of opening angles of six human vulnerable coronary samples. The ex-vivo experiments on these six human coronary samples resulted in an opening angle of $118 \pm 34.76$ degrees (mean ± SD).

2.3. Atherosclerotic coronary artery model

In order to construct the atherosclerotic coronary artery model, the parameters from the mean lumen area (LA) and external elastic membrane area (EEMA) of six human vulnerable coronary plaque samples were taken from Ohayon et al. (Ohayon et al., 2007). Here LA is the lumen area and the EEMA or external elastic membrane area resulted from the measurement of the vulnerable coronary plaque. The radius of the lumen area, $r_{LA}$, is calculated as below;
The data from (Ohayon et al., 2007) demonstrates that LA and EEMA parameters are $4.82\pm1.80\text{mm}^2$ and $19.73\pm6.59\text{mm}^2$ (mean ± SD) respectively. Using the mean value of the parameters, the lumen area radius $r_{LA}$ can be calculated as $1.125\text{mm}$ (15). Assuming that the plaque is restricted to the lumen area to one side of the artery (similar to the cases reported in (Ohayon et al., 2007), an eccentric parameter $d$ resulted from the equation below:

\[
d = r - r_{LA}
\]  

(16)

Which results in $d=0.7\text{mm}$. 
Figure 2.6. Schematic of the geometry for the coronary artery including the plaque model. Three layers of the artery and a plaque is modeled. Lumen area with radius $r_{LA}$ demonstrates the part of the artery in which the blood flows.

The stress free configuration of the atherosclerotic artery is modeled with a similar approach as for the normal coronary artery. Figure 2.7 shows the configuration of the model in its stress free state. Here $D$, the maximum thickness of the plaque region in the vessel is calculated from the relationship below:

$$D = r - (r_{LA} - d)$$  \hspace{1cm} (17)
2.4. Material properties

The stress analysis in the coronary artery model was studied by assigning hyperelastic material properties for the tissue. The parameters for the hyperelastic model are obtained from the data provided by Holzapfel et al. (Holzapfel et al. 2005) that are used for the geometrical modelling. The Ogden-type hyperelastic material is used to model the nonlinear material properties in the wall (Zahedmanesh 2009). These are shown in Table 2.1.
Table 2.1. Properties of different layers with Ogden type hyperelastic material properties.

<table>
<thead>
<tr>
<th>Ogden hyperelastic model constants</th>
<th>Intima</th>
<th>Media</th>
<th>Adventitia</th>
<th>Plaque</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \mu_1 )</td>
<td>-7,037,592.77</td>
<td>-1,231,144.96</td>
<td>-1,276,307.99</td>
<td>93,726.43</td>
</tr>
<tr>
<td>( \mu_2 )</td>
<td>4,228,836.05</td>
<td>785,118.59</td>
<td>846,408.08</td>
<td></td>
</tr>
<tr>
<td>( \mu_3 )</td>
<td>2,853,76</td>
<td>453,616.46</td>
<td>438,514.84</td>
<td></td>
</tr>
<tr>
<td>( \alpha_1 )</td>
<td>24.48</td>
<td>16.59</td>
<td>24.63</td>
<td>8.17</td>
</tr>
<tr>
<td>( \alpha_2 )</td>
<td>25</td>
<td>16.65</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>( \alpha_3 )</td>
<td>23.54</td>
<td>16.5</td>
<td>23.74</td>
<td></td>
</tr>
<tr>
<td>( D_1 )</td>
<td>( 8.95 \times 10^{-7} )</td>
<td>( 5.31 \times 10^{-6} )</td>
<td>( 4.67 \times 10^{-6} )</td>
<td>( 4.30 \times 10^{-7} )</td>
</tr>
</tbody>
</table>

2.5. Mesh generation, boundary condition and numerical solution

First the geometry of the three layers of the artery was modelled in its stress-free state using the approach mentioned in section 2.2. The arterial geometries were identical to those obtained by experimental data used by Holzapfel et al. (Holzapfel et al. 2005).

2.5.1. Healthy artery model

For the healthy artery, the model had an inner radius \( r \) of 5.703mm and the outer radius \( r_0 \) of 6.128mm and an opening angle \( \alpha \) of 118°. Thus the wall thickness \( h \) was 0.425mm. The model consisted of straight tube of 8mm lengths. The FEM was used for numerical simulation of stress distribution due to the opening angle in the coronary artery. Finite Element analyses were performed with ABAQUS (Version 6.9.1, Dassault Systemes Simulia Corp., Providence, RI, USA).
Large deformation formulations and plane strain assumptions, allowing for out of plane stress build up, were used for all computational models. Appropriate boundary conditions were used to suppress rigid body motion. All nodes along the cut edges were displaced to meet at their center using a rotation-displacement boundary condition. A steady wall thickness is assumed for before and after of closing the opening angle. While allowing for the cut edges to displace freely in the axial direction, one node was pinned from displacement in the central section to eliminate any rigid body motion.

A static intraluminal pressure of 13,300 Pa (~100mmHg) was applied as the loading condition. For each model, it was checked that there was independency of the simulation results from mesh sizes. Figure 2.8 shows the computational model of the healthy artery in the stress-free condition.
Figure 2.8. Computational model of healthy coronary artery. Hexahedral mesh was used for this model. The model has an inner radius ($r$) of 5.703mm and the outer radius ($r_0$) of 6.128mm and an opening angle ($\alpha$) of 118°. Thus the wall thickness ($h$) was 0.425mm. The model consisted of a straight tube of 8mm length.
2.5.2. Atherosclerotic artery model

For the diseased artery, the model had a lumen area radius \((r_{LA})\) of 1.125mm and the outer radius \((r_0)\) of 6.128mm and an opening angle \((\alpha)\) of 118°. The model consists of a straight tube of 8mm in length. Finite element analysis was performed using ABAQUS (Version 6.9.1, Dassault Systemes Simulia Corp., Providence, RI, USA). Due to the irregular geometry in the diseased condition, the wall was meshed with tetrahedral elements. A convergence test was performed to find the optimal mesh density. Optimal mesh was used for arterial models for the independency of the results from the computational mesh.

Large deformation formulation and plane strain assumptions with a steady state formulation were used for all computations. Appropriate boundary conditions were used to suppress rigid body motion. Similarly a static intraluminal pressure of 13,300Pa (~100 mmHg) was applied as the loading condition.
Figure 2.9. Computational model of an atherosclerotic coronary artery. The model had a lumen area radius ($r_{LA}$) of 1.125mm and the outer radius ($r0$) of 6.128mm and an opening angle of 118°.

The model consisted of a straight tube of 8mm length.

2.6. Data analysis

Given the simulation of the FEM analysis, some post-processing of the data was performed to demonstrate the results of the study more efficiently. Since the aim is to study the alteration of stress distribution and its effect in yield/rupture of the arterial wall, two different approaches were used to demonstrate the final stress distribution.
2.6.1 Von Mises stress distribution

The von Mises stress distribution was used as the yield/rupture criterion in the study. This criterion suggests that a material starts yielding when its von Mises stress reaches a critical value known as the yield strength. In materials science and engineering the von Mises yield criterion can be formulated with a scalar stress value that can be computed from the stress tensor.

\[
\sigma_v = \sqrt{\frac{1}{2}[(\sigma_{11} - \sigma_{22})^2 + (\sigma_{22} - \sigma_{33})^2 + (\sigma_{11} - \sigma_{33})^2 + 6(\sigma_{23}^2 + \sigma_{31}^2 + \sigma_{12}^2)]} \quad (18)
\]

Where \(\sigma_{ij}\) are different components of the Cauchy stress tensor. The above equation can be reduced and reorganized for practical use in different loading scenarios. In terms of the principal stress components von Mises stress can be presented as the equation below:

\[
\sigma_v = \sqrt{\frac{1}{2}[(\sigma_1 - \sigma_2)^2 + (\sigma_2 - \sigma_3)^2 + (\sigma_1 - \sigma_3)^2]} \quad (19)
\]
The resulting yielding stress from models was compared with experimental data present in the literature (Holzapfel et al. 2005). The ultimate tensile stress for all the arterial layers has been studied and results show that the intima and media layers show very similar ultimate stress of $391.0 \pm 144.0\text{kPa}$ and $419.0 \pm 188.0\text{kPa}$ respectively in the circumferential direction. The ultimate stress for the adventitia layer is $1300.0 \pm 692.0\text{kPa}$ (Holzapfel et al. 2005).

2.6.2 Circumferential, radial and axial stress distribution

The von Mises stress distribution was used as a strength criterion of the material. However in order to study the variations of the stress distribution in different situations of the artery in detail, the stress distribution in three main directions that fits well with the geometry of the arteries was used. Figure 2.10 demonstrates the schematic of the artery and directions in which the stress values are presented in all studies for comparison.

![Figure 2.10. Schematic representation of axial, circumferential and radial stress components that are represented in all models in this study.](image)
Chapter 3. Results

This study focused on the mechanical stress distribution within the mid-LAD simulation of the human coronary artery model. The results section is divided into two main subsections. In the first section, the stress distribution due to the residual stress and blood pressure in the healthy/normal human coronary artery model is presented. The second section describes the stress distribution in atherosclerotic human coronary arteries. In the healthy arterial models, the stress distribution results are obtained from a path on the inner arterial wall starting from the intima toward the adventitia layer. In the atherosclerotic arterial models, the stress distribution results are obtained by defining a path starting from the maximum intimal thickening atherosclerotic artery toward the adventitia layer.

The study on residual strain includes two different configurations: residual strain measured as the opening angle of the artery and residual strain measured as the longitudinal stretch. The stress distributions in axial, circumferential and radial directions were studied for these cases. For the first step, a benchmarking test of our simulations with the analytical solutions was conducted.

3.1. Benchmarking the results

In order to benchmark the results from Finite Element (FE) analysis, the residual stress resulting from an opening angle in a one layer healthy coronary geometry was calculated, using the analytical solution described in equation (9). The geometry is composed of a one-layer arterial wall with the material properties of the
adventitia layer. For this test experiment an opening angle of 80 degrees was performed on the analytical and numerical models. Linear material properties were used for this model to validate the FE results. After FE results are validated, hyperelastic material properties were used to build the FE arterial models.

Stress distribution in the cross section of the wall is extracted from both analytical and the numerical solutions. The computed von Mises stress distribution is presented in Figure 3.1. The color bar here demonstrates the distribution of the computed stress in the wall. From both models the maximum von Mises stress is in the range of 100kPa.

![Figure 3.1. Stress distribution from the (a) analytical and (b) numerical solution in this study.](image)

In order to show more quantitative distribution of stress in the wall thickness, the von mises stress value in the radial direction was presented as the curves in Figure 3.2. The stress distribution here is presented as a function of the distance from the inner wall. Computations of the analytical and numerical solution have been demonstrated with the blue and red lines respectively. There is a good agreement
between the results of the analytical and numerical solutions (Matlab code is included in the appendix section).

As shown in Figure 3.2, the stress in the inner part of the wall is negative which corresponds to a compressive stress. This negative stress is changing to positive in the outer part of the wall with a linear function.

![Figure 3.2. Stress distribution in the thickness of the one layer elastic vessel obtained from analytical and numerical methods. There is a good fit between the results of two methods.](image-url)
3.2. Normal Coronary Artery

3.2.1. Closed arterial model

3.2.1.1 Axial stretch (stretched without pressure)

To study the effect of the axial residual stress, human coronary artery with the length of 8mm was simulated. For the healthy/normal model, a three-layered coronary artery model was studied using FE analysis. On the basis of published literature (Holzapfel et al. 2005) the ratio of adventitia, media and intima thickness to total wall thickness was used as $0.40 \pm 0.03\text{mm}$, $0.36 \pm 0.03\text{mm}$, and $0.27 \pm 0.02\text{mm}$ (mean $\pm$ SD), respectively. The inner and outer diameters were 1.825mm and 2.25mm and the ratio of wall thickness to its radius was $0.189 \pm 0.014$ (mean $\pm$ SD) (Ohayon et al., 2005).

An axial in situ stretch of 5% with respect to the load-free length was applied to the models as it has been reported in previous studies (Holzapfel et al. 2005). The appropriate boundary conditions were applied to the model. The arterial model is fixed in the longitudinal direction in one side. The axial stretch has been applied to the other side of the arterial model below by stretching the unloaded configuration of the arterial model in the longitudinal direction. Figure 3.3 illustrates the von Mises stress distribution in the arterial model.
Figure 3.3. Axial stretch was applied to the arterial model by stretching the artery in its longitudinal direction. In the unloaded configuration of the artery, the color bar demonstrates residual stress induced in the arterial wall thickness starting from the inner layer of the intima layer toward the adventitia layer.

Different arterial layers show different mechanical behavior. The color code illustrates von Mises stress through the wall thickness. The artery has its highest residual stress value of 6,230 Pa in the intima and decreases toward media layer where it reaches its minimum value of 1,116 Pa in the adventitial layer.

Finite element solver generates Cauchy stress components as outputs. Different Cauchy stress components have been analyzed to show their evolution in the vessel wall as a function of wall thickness (Figure 3.4). In this figure, radial stress demonstrates the residual stress induced in the radial direction of the arterial model, axial stress illustrates the axial stress component and circumferential demonstrates the stress component in the circumferential direction.
Results demonstrate a stress jumps at the interfaces between the layers. These jumps are explained due to shear stress and the change of material properties in the layers. A smaller jump was observed in the media-adventitia interface, as we have similar material properties in these two layers. However this jump is relatively significant at the interface between intima-media, where the mechanical and compositional properties are changing.

The residual stress has positive values in the longitudinal direction of the arterial model.
3.2.1.2 Circumferential stress (pressure without residual stress)

A three-layered coronary artery model was simulated including blood pressure load in the arterial geometry. The boundary conditions are applied to the model. The inner wall of the artery was supplemented by 100mmHg (13.33kPa) pressure loading to simulate the loaded physiological configuration. The arterial model is fixed in its longitudinal direction. The inhomogeneous nature of the cylindrical wall affects the circumferential stress distribution. Figure 3.5 shows a snapshot of the color-coded visualization of von Mises stress distribution in the model with this configuration. As a result higher stress distribution was observed in the intima.

Figure 3.5. Von-Mises stress distribution in the arterial model with blood pressure load 100mmHg that is applied to the endothelial surface (inner surface) of the arterial model.

A more quantitative study on the stress distribution was conducted by extracting the radial, axial and circumferential stress in the model. The stress distribution results along the radial direction in the arterial wall were extracted from
the model and their variation was presented. These stress curves were obtained by defining a path starting from the inner radius of the artery in the intima layer toward the outer radius of the artery in the adventitia layer. Von-Mises stress was calculated from the represented Cauchy stress tensor within the path starting from the inner radius of the artery in the intima layer toward the outer radius of artery. Figure 3.6 represents these stress components in the coronary wall. The results were obtained from the loaded state of the arterial model when the effect of residual stress was ignored.

![Figure 3.6. Cauchy stress components vs. normalized wall thickness in loaded physiological state without the effect of axial residual stress.](image)

In the loaded state of the arterial model, the most nonlinearity of circumferential stress is observed in the intima layer where the tensile stress and its value decrease toward the media and adventitia layers. Applying blood pressure to the arterial model, induces a small compressive stress is observed in longitudinal direction. The maximum axial stress is observed in the intima with the value of -8kPa.
3.2.1.3 Axial and circumferential stress (stretched with pressure)

The arterial model was analysed when the axial stretch was applied to the arterial model and the blood pressure of 100mmHg was exerted to obtain the loaded physiological state. Boundary conditions were applied to the model. The arterial model is fixed in the longitudinal direction in one side and stretched in the other side. Von-Mises stress distribution is demonstrated in the wall in Figure 3.7.

In the model below, the axial component of the stress is negligible. Figure 3.6 represents that applying blood pressure induces compressive axial stress in the artery. Therefore, in the model below when the combined effect of axial stretch and blood pressure is combined, the negative value of axial stress is compensated by the tensile axial stress induces by the axial stretch.
Figure 3.7. Result from axial in situ stretch in the loaded state of the arterial model. A 100mmHg blood pressure was applied to the endothelial surface in the presence of a uniform axial stretch in the model.

Similar to the previous cases, the intima layer exhibited the most stress nonlinearity in the circumferential direction. The stress results showed a large distribution in the intima layer and small dispersion in the media and adventitia layers. Figure 3.8 illustrates the radial, axial and circumferential stress distributions in a normalized thickness of the arterial wall.

![Figure 3.8](image)

Figure 3.8. Cauchy stress tensor components vs. normalized wall thickness in stretched, loaded arterial model. The arrows show the stress jumps between the layers.

The stress curve for the intima exhibits the most nonlinearity of circumferential stress. In a path from the intima towards the media, the circumferential stress dramatically decreased towards the media where its value
decreases slightly towards the outer layer. The longitudinal stress is much smaller than the circumferential stress for the intima layer. Relatively small compression was found in the radial direction for the intima layer. Its value is larger in the media and adventitia.

3.2.2. Open angle arterial model

3.2.2.1. Residual stress without blood pressure (closing the opening angle/ unstretched with pressure)

This section represents the results obtained from the experimental data achieved from the opening angle simulation by closing the normal human coronary artery from its “zero-stress state” to the closed position in the unloaded state. The opening angle method, which has been defined previously by other authors (Ohayon et al., 2007) was applied to the arterial segment by making a radial cut to obtain a thin ring-like slice of the artery, which resulted in the two cut edges opening up into an arc with a certain opening angle. This releases the circumferential stress and then the arterial ring is assumed to be totally stress-free in the zero-stress configuration.

Closing the zero-stress open configuration of the normal artery generates a compression of the sub-intimal layers and an extension of the sub-adventitial layers. Figure 3.9 demonstrates snapshots of the recovery process for the artery from its opening angle state as is modeled in this study. Von-Mises stress distribution is
demonstrated for each configuration. The stress distribution in the closed configuration is the distribution that is caused by the residual stress in the artery wall.
Figure 3.9. Three snapshots show the recovery of the artery model from its opening angle state. Von-Mises stress distribution in the closed configuration demonstrates the residual stress in a healthy artery due to its opening angle.

Closing of the opening angle for the unloaded length in the arterial model induces residual stress in the artery. Figure 3.10 demonstrates the distribution of circumferential, radial and axial Cauchy stress tensor components in the wall.

Figure 3.10. Cauchy stress tensor components vs. normalized wall thickness after closing the opening angle

The results of circumferential residual stress show compressive stress in the intima and tensile stress in the media and adventitia. There are two jumps in the stress magnitude. The higher stress jump is about 1,800 Pa, which is observed on the intima-media interface and the lower stress jump of about 800 Pa is observed in the media-adventitia interface. These are due to the change of material properties between arterial layers.
Radial residual stress remains relatively uniform with a negligible value through the whole arterial wall thickness. There is a small compressive residual longitudinal stress in the outer layer of the intima towards the inner layer of media.

Axial/longitudinal stress results show nonlinear compressive stress for the intima layer. There is a stress jump from the outermost intima layer toward the innermost media layer that demonstrates tensile stress for the media layer.

3.2.2.2. Residual stress with blood pressure

In this model, the opening-angle recovery model was applied and the blood pressure load was supplemented by 100 mmHg (13.33 kPa) to the closed model of artery when residual stress is included. Figure 3.11 shows the resulting stress distribution in the wall. It shows that stress is higher in the inner and outer part of the wall and its value is reduced in the middle. Including the pressure boundary condition, the simulation shows that the stress is higher in the inner wall in this condition than the outer part of the wall with an order of 100 and 50kPa, respectively.

![Figure 3.11. Von-Mises stress distribution in the arterial model under physiological loading after closing the recovery from opening angle.](image)
Circumferential stress of tensile type in the inner radius is observed through the wall thickness of the arterial model. Figure 3.12 demonstrates the distribution of circumferential, radial and axial stress components in the thickness of the wall. The circumferential stress variations are higher in the inner radius. The distribution of circumferential stress is affected by the inhomogeneous nature of the cylinder.

![Figure 3.12. Cauchy stress tensor components vs. normalized wall thickness for loaded arterial model after closing the opening angle under physiological loading condition.](image)

More quantitative study on the stress distribution has been obtained by extracting the radial, axial and circumferential stress values in the wall. Figure 3.12 represents these stress components in the thickness of the wall. By closing the opening angle of the arterial model, under the physiological loading condition of the artery, the linear relationship exists between strain and circumferential stress. Results show tensile stress through the wall thickness. Radial stress is not linear in the intima
layer where it demonstrates a compressive stress. Its value increases towards the media and adventitia layers where it changes to tensile type stress.

Table 3.1 demonstrates the comparison of maximum stress for the healthy artery models. The models are distinguished by different conditions of axial stretch, blood pressure and circumferential stress due to the opening angle. The table below shows that the arterial stress caused by blood pressure has a greater value than the residual stress caused by the opening angle.

Table 3.1. Stress in healthy artery models including stretch, pressure and residual stress components

<table>
<thead>
<tr>
<th>Healthy artery</th>
<th>Axial stretch (%)</th>
<th>Opening angle (degrees)</th>
<th>Pressure (Pa)</th>
<th>Max von mises stress (Pa)</th>
<th>Max stress location</th>
</tr>
</thead>
<tbody>
<tr>
<td>Axial stretch</td>
<td>5</td>
<td>0</td>
<td>0</td>
<td>6,200</td>
<td>intima</td>
</tr>
<tr>
<td>Pressure</td>
<td>0</td>
<td>0</td>
<td>13,300</td>
<td>400,000</td>
<td>intima</td>
</tr>
<tr>
<td>Axial+pressure</td>
<td>5</td>
<td>0</td>
<td>13,300</td>
<td>425,000</td>
<td>intima</td>
</tr>
<tr>
<td>RS</td>
<td>0</td>
<td>118</td>
<td>0</td>
<td>7,700</td>
<td>intima</td>
</tr>
<tr>
<td>RS+OA</td>
<td>0</td>
<td>118</td>
<td>13,300</td>
<td>242,300</td>
<td>Intima-media interface</td>
</tr>
</tbody>
</table>
3.3. Atherosclerotic Coronary artery

3.3.1. Closed arterial model

3.3.1.1. Axial stress (stretched without pressure)

This section represents the axial stress distribution for the three-layer atherosclerotic human coronary artery model, which is compared to the non-atherosclerotic model of the human coronary artery to investigate the effect of plaque formation on axial stress distribution. A diseased coronary artery is modeled as a closed cylinder and the layer-specific material properties were used for each layer. The atherosclerotic plaque geometry is added to the three-layer arterial model using the hyperelastic material properties of the Ogden constitutive relation.

To simulate the in situ axial stress, certain boundary conditions were applied to stretch the artery in the longitudinal direction. Axial in situ stretch with 5% prestretch with respect to the load-free length was used to model this condition (Holzapfel et al., 2005). Different boundary conditions were applied to the model. The surface area of the artery is fixed in the longitudinal direction on one side and stretched on the other side.
Figure 3.13 shows the distribution of von-Mises stress in the wall. The maximum von-Mises stress was 1,200 Pa that is presented in the intima layer in the non-atherosclerotic part of the artery.

Figure 3.13. Von-Mises stress distributions in arterial model under axial stretch.
Different stress components have been analyzed to show their evolution in the wall thickness as presented in Figure 3.14. As expected when axial stretch was applied to the arterial model, residual axial stress was not linear in this case. Compressive stress in the innermost layer of the arterial wall was observed. Radial stress remained almost steady with relatively small value through the whole wall thickness. The radial stress curve demonstrated almost the same behaviour for both non-atherosclerotic and atherosclerotic arterial models.

3.3.1.2. Circumferential stress (pressure without residual stress)

In this section a three-layer atherosclerotic coronary artery was modelled including a blood pressure load of 100 mmHg (13,330 Pa) to the endothelium of atherosclerotic coronary artery model. Both surface areas of the model were fixed in the longitudinal direction. The inhomogeneous nature of the cylinder affected the stress distribution. Stress distribution curves were obtained by representing the Cauchy stress tensor within the arterial layers starting from the intima layer toward the adventitia layer defined in the arterial model. Figure 3.15 shows a snapshot of the Von-Mises stress distribution in the wall under this configuration. Higher von-Mises stresses were observed in the intima.

Results of von-Mises stress represent higher stress in the circumferential direction of the innermost layer. The maximum value of the stress result is 758,000 Pa in the intima layer.
Figure 3.15. Stress distribution in the arterial model with blood pressure load in its (a) cross-sectional and (b) full view display.

More quantitative study on the stress distribution was obtained by extracting the radial, axial and circumferential stress values in the wall. Figure 3.16 represents these stress components in the thickness of the wall. The results show that under the
physiological loading condition of the arterial model, when the effect of residual stress is ignored, tensile circumferential stress is distributed throughout the inner layer of the wall thickness. Compressive circumferential stress is observed for the outer layer of the artery.

![Figure 3.16. Cauchy stress components vs. normalized wall thickness in loaded physiological state without the effect of axial residual stress.](image)

3.3.1.3. Axial and circumferential stress (stretched with pressure)

The arterial model was analysed when the axial stretch and blood pressure of 100 mmHg was exerted to the model. The appropriate boundary conditions were applied to the model. The model is fixed on one side in the longitudinal direction and stretched on the other side. Von-Mises stress distribution is shown in Figure 3.17. Again intima had the highest stress distribution, but von-Mises stress was decreased
with respect to the model without residual stress (from about 750,000 Pa to 770,000 Pa). This effect was demonstrated by the fact that the high tensile circumferential stress due to external pressure in this region was moderated by the compressive stress components due to the axial residual stress.
Figure 3.17. Von-Mises stress distribution in arterial model under axial stretch in loaded physiological state with (a) cross-section and (b) full view display.
Similar to the previous cases, longitudinal stress exhibits the most fluctuation. The stress results show large distribution in the media layer and small dispersion in media and adventitia layers. Figure 3.18 illustrates the radial, axial and circumferential stress distribution in an arterial wall with normalized thickness.

![Figure 3.18. Cauchy stress tensor components vs. normalized wall thickness in stretched, loaded arterial model.](image)

Circumferential stress has the highest value compared to axial and radial stresses. Circumferential stress value is higher in the inner layer and its value decreases toward the outer layers.

### 3.3.2. Opening angle arterial model
3.3.2.1. Residual stress without blood pressure (Closing the opening angle/ unstretched with pressure)

This section represents the data obtained from the three-layer atherosclerotic human coronary artery model to investigate the effect of the opening angle on stress distribution. A human coronary artery was modelled as a three-layered open angle in its zero-stress geometrical configuration and layer-specific material properties were used for each layer.

The appropriate boundary conditions were applied to the model. The longitudinal surfaces of the arterial model are fixed in the longitudinal direction. The effective residual strain in the thin fibrous cap appeared to be non-negligible. Thus, even under no external load (i.e., blood pressure), the plaque was subjected to high stress/strain. According to the chosen stress-strain law, a non-linear relationship exists between strain and circumferential stress. Results showed some zones of high stress located on the rear side of the lipid core of the plaque and at the interface between the wall and the plaque fibrosis. Negative stresses near the inner layer and tensile stresses near the outer layer were observed. Inside an atherosclerotic artery, however, the residual stress distribution was found to be more complex than in a normal artery. The results illustrated the residual stress distribution in the closed geometry in the “unloaded physiological state” demonstrated in the graphs below.
Figure 3.19. This snapshot shows the recovery of the artery model from its opening angle state. The Von-Mises distribution in the closed configuration demonstrates the residual stress in a healthy artery due to its opening angle.

Von-Mises stress results show that the region with the most plaque thickness has the maximum stress concentration. Maximum von-Mises stress was 35,500 Pa (see Figure 3.19).

A path was defined along the radial direction of the arterial model, starting from the maximum intimal thickening toward the adventitia layer. The residual stress components are illustrated in the figure below.
The stress curve in Figure 3.20 demonstrates compressive circumferential stress in the inner layer and tensile stress toward the outer layer. Radial stress is negligible. There’s a small value for axial stress due to the strain in the circumferential direction.

3.3.2.2. Residual stress with blood pressure

The final section represents the results obtained from a “loaded physiological state” after closing the opening angle model of the atherosclerotic artery to investigate the effect of blood pressure on the circumferential stress distribution. The combined effect of residual stress caused by closing the opening angle and blood pressure of 13,300 Pa (100 mmHg) as an internal load in the artery, are simulated in the model. The appropriate boundary conditions were applied to the model. The longitudinal surfaces of the arterial model are fixed in the longitudinal direction.
Applying blood pressure to the arterial model, releases the circumferential stress induced by closing the opening angle. Therefore, since the compressive stress value in the cap of the plaque had the highest stress value in the previous model (Figure 3.19), therefore the tensile circumferential stress has a greater value in the surrounding area of the plaque cap (see Figure 3.21).
Figure 3.22. Cauchy stress tensor components vs. normalized wall thickness for loaded arterial model after closing the opening angle under physiological loading condition.

Figure 3.22 shows that the circumferential stress value was considerably higher in the inner layer of the arterial model with respect to other stress components. Tensile circumferential stress was distributed through most of the whole wall thickness.

Table 3.2 demonstrates the comparison of maximum stress for the atherosclerotic artery models. The models are distinguished by different conditions by axial stretch, blood pressure and circumferential stress due to opening angle. Table below shows that the arterial stress caused by blood pressure has a greater value than the residual stress.
<table>
<thead>
<tr>
<th>Healthy artery</th>
<th>Axial stretch (%)</th>
<th>Opening angle (degrees)</th>
<th>Pressure (Pa)</th>
<th>Max von mises stress (Pa)</th>
<th>Max stress location</th>
</tr>
</thead>
<tbody>
<tr>
<td>Axial stretch</td>
<td>5</td>
<td>0</td>
<td>0</td>
<td>12,300</td>
<td>Non-atherosclerotic intima</td>
</tr>
<tr>
<td>Pressure</td>
<td>0</td>
<td>0</td>
<td>13,300</td>
<td>535,800</td>
<td>Non-atherosclerotic intima</td>
</tr>
<tr>
<td>Axial+pressure</td>
<td>5</td>
<td>0</td>
<td>13,300</td>
<td>774,600</td>
<td>Non-atherosclerotic intima</td>
</tr>
<tr>
<td>RS</td>
<td>0</td>
<td>118</td>
<td>0</td>
<td>35,500</td>
<td>Plaque cap</td>
</tr>
<tr>
<td>RS+OA</td>
<td>0</td>
<td>118</td>
<td>13,300</td>
<td>120,000</td>
<td>The interface of lipid core and media</td>
</tr>
</tbody>
</table>
Chapter 4. Discussion

This research aims to understand the influence of residual stress on the stress distribution over the cap of atherosclerotic plaque. This study is in continuation of recent work for modeling residual stress in arteries using Finite element modeling (Alastrue et al., 2007, Holzapfel et al., 2009, Raghavan et al., 2004) Longitudinal stretch and circumferential stretch (opening angle) are included in healthy and atherosclerotic arterial modeling. The circumferential and longitudinal residual stress effect on the stress distribution in an atherosclerotic artery was studied in the realistic geometry of the coronary artery.

The study focused on the mechanical stress distribution within the mid-LAD of human coronary artery. This is because this region has small curvature within its structure and is prone to plaque formation. A novel formulation was proposed to calculate residual stress at each point of a healthy artery. Stress distribution was reported for the healthy and diseased artery with and without the pressure load through blood flow in the arteries. The effect of longitudinal and circumferential residual stress was included in the simulations.

FEM was used for the estimation of the stress distribution in the artery. The method used in this study for computing residual stress related to the opening angle, was based on modeling a stress free artery when it is radially cut. Two planes were tied into cutting edges for closing the coronary artery models from its “zero-stress state” to the closed position in the unloaded state. Closing the arterial opening angle, induces the residual stress in the arterial model.

To validate the FEM results of opening angle models, the opening angle model was simplified to a one-layer model with linear-elastic material properties. The
results of this FE model was compared to the results obtained from analytical solution of the same simplified model. The geometry was composed of a one-layer arterial wall with the material properties of the adventitia layer.

The stress distribution in the cross section of the wall extracted from the analytical solution and the numerical solution is presented in Figures 3.1 and 3.2. Similar results are obtained from the analytical and numerical solutions that confirm the correct approach in using the model, meshing and boundary conditions in the numerical approach. Maximum error happened in the endothelial part of the artery with a maximum error of 5%. This error was related to the limitations in the numerical solution and the errors they provide in the boundary elements. There was close fit of data in other parts of the artery wall.

Stress distribution changed from negative compressive stress in the inner wall of the artery to positive and extensive stress in the outer wall. Maximum stress appeared in the outer wall of the artery with a tensile type stress. Here the neutral axis ρ is at a radius 2.042mm of the model. The position of the neutral axis is an important factor to demonstrate the size of the radial portion that tensile and compressive stress was applied on the cross section of the artery. This study has shown that the location of the neutral axis is changing linearly with respect to the opening angle. Figure 4.1 shows the change in the neutral axis as computed from the analytical solution in equation 10. This shows that by increasing the opening angle in the artery, the neutral axis moves closer to the outer wall of artery, meaning that the region with tensile stress gets smaller.
Figure 4.1. Variation of the neutral axis position in the wall as a function of the opening angle. With increasing the opening angle the neutral axis moves towards the outer wall of artery, meaning that the region with tensile stress gets smaller.

4.1. Stress distribution in healthy artery

A three-layered coronary artery model was studied with an axial stretch of 5% with respect to the load-free length in the longitudinal direction. Different arterial layers show different mechanical behavior. The maximum residual stress value of 6,229 Pa was demonstrated in the innermost layer of intima, which was decreasing towards the media layer where it reached the minimum value of 1,116 Pa in the adventitia layer.

There were also significant stress jumps between the layers. These jumps are explained by the material mismatch across the interfaces. The same experiments were repeated but this time only including the blood pressure load of 100 mmHg (13,330 Pa) to the artery model. In the loaded state of the arterial model, more nonlinearity of circumferential stress was observed in the intima layer with a tensile type stress. This stress was decreased towards the media and adventitia layer. Axial and radial stresses remain almost steady with a relatively small value through the whole wall thickness.
In the third part, the longitudinal residual strain was combined with the blood pressure distribution to see how residual stress affects the stress distribution. As a result the intima again had the highest stress gradient, but its axial stress was moderated with respect to the model without the effect of axial residual stress. This effect was demonstrated by the fact that the high compressive axial stress due to external pressure in this region was moderated by tensile stress components due to the axial residual stress. This observation is in accordance with the hypothesis that residual axial stress produces lower stress changes in the axial direction of the artery.

In the next part of the experiments the opening angle effect on the stress distribution in the arteries was studied. In the first experiment the residual stress distribution due to the opening angle was studied. It has been proven that this stress forms only a circumferential stress in the arterial wall. Compressive stress was observed in the intima and tensile stress was seen in the media and adventitia. Also two jumps in the stress magnitude have been observed. The higher stress jump was 1,800 Pa, which is observed on the intima-media interface. The shorter jump of 800 Pa is observed in media-adventitia interface. These jumps are explained by the material mismatch in the interfaces of the arterial layers. Results are validated by comparing with previous work of Ohayon et al., 2007 and Williamson’s group 2003.

The blood pressure in the presence of opening angle was added. The blood pressure load was set to 100mmHg (13,300 Pa). Stress concentration was higher in the inner and outer part of the wall with reduced values in the middle. Including the pressure boundary condition, releases compressive stress in the intima layer. Therefore, it induces tensile stress distribution in the intima. This finding shows that residual circumferential stress, produces lower stress changes in the circumferential direction of an artery.
4.2. Stress distribution in atherosclerotic artery

In the second part of the studies the stress distribution modeling in the atherosclerotic arteries was repeated. First a longitudinal strain with conditions similar to the healthy artery was applied. With an axial strain of 5%, a maximum stress of 12,000 Pa was observed in the intima layer of the non-atherosclerotic part of the artery, which is almost two times larger than the maximum stress in the intima layer of the healthy coronary artery. This stress was decreasing toward the media layer where it reaches its minimum value of 169 Pa in the adventitial layer.

Significant stress jumps between the layers were also observed. These jumps are explained by the change of material properties in the layers.

The same experiments were repeated with only including blood pressure load of 100mmHg (13,330Pa) to the atherosclerotic arterial model. In the loaded state of the arterial model, more nonlinearity of circumferential stress is observed in the intima layer with a tensile type stress. This stress was decreased toward the media and adventitia layer. Axial and radial stresses show a great decrease in intima layers, which results in a decreasing graph. Axial and radial stress graphs, show stress jumps for each layer but the value remains negative, which represents compressive stress in all the layers. The maximum von Mises stress in this model is 535,000 Pa, which is 1.2 times greater than the maximum stress in the loaded state of healthy arterial model.

In the third model the longitudinal residual strain was combined with the blood pressure distribution to see how the axial residual stress affects the stress distribution. As a result it was observed that the intima again has the highest stress
distribution, but its axial stress component is slightly moderated with respect to the model without the effect of axial residual stress. This effect is demonstrated by the fact that the high tensile axial stress due to external pressure in this region is moderated by the compressive stress components due to the blood pressure (Ohayon et al. 2007). This observation is in accordance with the hypothesis that axial residual produces lower stress changes in the axial direction of an artery. The maximum stress of 774,000 Pa in this model is 1.8 larger than in the healthy model.

In the next part of the experiments the opening angle effect on the stress distribution in the arteries was studied. In the first experiment the residual stress distribution due to the opening angle was studied. The maximum stress in this model is 35,000 Pa, which is 5 times greater than in the healthy arterial model. A large compressive stress is observed in the inner layer of the artery with atherosclerotic intimal thickening. Tensile stress is observed in the outer part of the artery.

Then pressure loading from blood flow was added in the presence of opening angle residual stress. The blood pressure load was supplemented by 100 mmHg (13,300 Pa). The maximum stress distribution in this model is 2,500,000 Pa. Comparing the results of this section with the previous studies (Ohayon et al., 2007 and Williamson’s group 2003) validates that blood pressure release compressive stress in the inner layer of the artery with the intimal thickening. The tensile stress induces by blood pressure, reduces the compressive stress in the cap of the atherosclerotic plaque. This finding shows that circumferential residual stress reduces the stress changes in the circumferential direction of an atherosclerotic artery.
4.3 Effect of opening angle and longitudinal stretch on cap stress

Both healthy and atherosclerotic models were studied under the influence of 5% longitudinal stretch (Holzapfel et al., 2005), and opening angle of 118 degree (Ohayon et al., 2007). Residual stress due to the opening angle and longitudinal stretch was included in the numerical models of the artery. The von Mises stress distribution was used as the yield/rupture criterion in the study. Stress values were reported on the cap of the atherosclerotic plaque. Table 4.1 shows the stress distribution in all the models with different boundary conditions. In the cap of the plaque, the stress induced by longitudinal stretch and opening angle are 370 Pa and 5700 Pa, respectively. It can be concluded that circumferential residual stress induced by the opening angle is greater than the axial residual stress induced by longitudinal stretch. In the healthy arterial model, the value of residual stress induced by longitudinal stretch is almost the same as its value induced by the opening angle (6,229 Pa and 5,500 Pa), respectively.
Table 4.1. von Mises stress in arterial models. The table demonstrates the stress in healthy models and also in the cap of atherosclerotic plaque in diseased arterial models.

<table>
<thead>
<tr>
<th>Healthy artery</th>
<th>Axial stretch (%)</th>
<th>Opening angle (degrees)</th>
<th>Pressure (Pa)</th>
<th>Von Mises stress in intima (Pa)</th>
<th>Von Mises stress in cap (Pa)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Axial stretch</td>
<td>5</td>
<td>0</td>
<td>0</td>
<td>6,229</td>
<td>370</td>
</tr>
<tr>
<td>Pressure</td>
<td>0</td>
<td>0</td>
<td>13,300</td>
<td>410,400</td>
<td>37,000</td>
</tr>
<tr>
<td>Axial+pressure</td>
<td>5</td>
<td>0</td>
<td>13,300</td>
<td>434,600</td>
<td>40,000</td>
</tr>
<tr>
<td>RS</td>
<td>0</td>
<td>118</td>
<td>0</td>
<td>6,000</td>
<td>37,000</td>
</tr>
<tr>
<td>RS+OA</td>
<td>0</td>
<td>118</td>
<td>13,300</td>
<td>150,000</td>
<td>115,000</td>
</tr>
</tbody>
</table>

The maximum von mises stress for healthy and atherosclerotic arteries when the effect of residual stress is considered was 150 kPa and 115 kPa respectively.

Based on the results obtained from analyzing the stress distribution in the healthy arterial model and previous studies, residual stress decreases the stress changes within an artery.

By comparing the results obtained from closing the opening angle model in the healthy and atherosclerotic artery, it’s concluded that the circumferential residual stress in a diseased artery has a much higher value than in the healthy artery model.
Chapter 5. Conclusions

This study focuses on 3D residual stress distribution in arterial wall. A three-layered coronary artery has been modelled in three-dimensions, using hyperelastic material properties. The geometry of a coronary artery was extracted from the literature. The stress-free state of the artery was modelled. A new formulation was used to calculate the radius of the stress-free state of the artery. This finding proposes a method to solve the limitation of the work of Bustamante and Holzapfel., 2010 by finding the geometry of stress-free state of an artery. The opening angle method was used to estimate the residual stress when the artery is unloaded. A new analytical solution was proposed to calculate residual stress at each point of an arterial wall. In order to validate the results obtained from FEM, the residual stress estimated by FE was compared to the results obtained from the analytical solution.

Axial in situ stretch of 5% with respect to the load-free length was applied to the model in the longitudinal direction. Different arterial layers present different mechanical behavior. In this study the influence of atherosclerotic plaque on stress distribution in the arterial model has been studied. The procedure takes into account the effect of circumferential and longitudinal residual stresses separately and in combination. Arterial models were analyzed in both loaded and unloaded states. In the loaded state’ blood pressure of 100mmHg (13.3kPa) was applied to the arterial model. The models were studied to investigate 1) The effect of the residual stress on stress distribution and 2) The effect of atherosclerotic plaque on stress gradient in the artery when residual stress is considered.
Significant stress jumps between the arterial layers were observed. These jumps are explained by the change of material properties in the layers. This shows that the more the material properties of two layers are different, the bigger the jumps became.

By comparing the results of the opening angle model with and without the effect of blood pressure, it has been concluded that blood pressure increases the stress gradient in the artery. The maximum von Mises stresses in atherosclerotic models are greater than the maximum stress in the healthy arterial models. This result indicates that atherosclerotic plaque increases the stress in the artery wall.

In the arterial model, the blood pressure of 100 mmHg is applied to the model with and without the effect of residual stress. The results of these models show that axial residual stress increases the stress distribution within the artery for both healthy and atherosclerotic artery. This effect is demonstrated by the fact that the high tensile circumferential stress due to external pressure in this region is moderated by the compressive stress components due to the axial residual stress. Circumferential residual stress for atherosclerotic model has a greater value than the healthy model.

In addition to the underlying effect of residual stress within an artery, plaque formation increases the mechanical stress distribution in the arterial wall. These findings have been validated by other studies. For example Gijsen et al.,[2008] performed three-dimensional coronary artery imaging and discovered that plaque regions are exposed to higher stress. Also Slager et al.,[2005] found that coronary plaques experience greater tensile stress at their shoulders.
The limitation of the proposed modelling method presented in chapter 2 is the limitation of including fluid structure interaction of blood flow. The obtained results are based on the assumption that the opening angle remains constant in the axial length of the artery. Also, the proposed analytical solution to calculate residual stress, is based on that the material is incompressible, that after the opening angle cut, the arterial curvature remains the same at each point and form an arc of a circle.

Hence, one of the possible future steps would be to consider fluid structure interaction (FSI) within an artery and investigate the effect of FSI on stress distribution in the atherosclerotic plaque.

Residual stresses have a significant influence on stress distribution in arteries within in vivo conditions. Therefore, any additional findings with respect to modelling of arterial residual stresses would be very helpful for a better understanding of artery in normal and atherosclerotic conditions.
Appendix

Matlab code of benchmarking the results:

clear all; clc;
format long

% constants
pi=3.14159265;

% geometric data (m)
r = 0.001825; h = 0.000425;

% opening angle
phi = 300 *pi/180;

% material property (Pa)
G = 2166000;

% calculate ru
syms ru;
f = h-ru*pi/(phi-pi)*log(1+h*(phi-pi)/(ru*phi+(pi-phi)*(h+r)));
soln = solve(f,ru);
ru = double(soln)

% calculate strain distribution
n = 10;
y = ru-r:-h/n:ru-h-r;
epsilon = phi*y./(pi*ru+(phi-pi)*y);

% calculate stress distribution
sigma = -G*phi*y./(pi*ru+(phi-pi)*y);

%% report stress
x = ru - y;
xlswrite('stress', [x', sigma'])

% plot stress
plot(x, sigma)

m = 50;
tetha = pi*(0:2/m:2);
for j = 1:m+1
    for i = 1:n+1
        X(i,j) = x(i)*cos(tetha(j));
        Y(i,j) = x(i)*sin(tetha(j));
        Z(i,j) = 0;
        C(i,j) = sigma(i);
    end
end
surf(X,Y,Z,C)
colorbar
view(2)
axis off
axis equal

phi = (10:30:330)*pi/180;
syms ru;
for i=1:size(phi,2)
    f = h-ru*pi/(phi(i)-pi)*log(1+h*(phi(i)-pi)/(ru*phi(i)+(pi-phi(i))*(h+r))); 
    soln = solve(f,ru);
    rut(i) = double(soln);
end
figure;
plot(phi*180/pi,rut)
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