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ASSESSMENT AND DESIGN OF SLEEVES FOR CORONARY ARTERY BYPASS GRAFTING

KOH AI LEEN

SCHOOL OF MECHANICAL AND AEROSPACE ENGINEERING
NANYANG TECHNOLOGICAL UNIVERSITY

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**Assessment and Design of Sleeves
for
Coronary Artery Bypass Grafting**

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School of Mechanical and Aerospace Engineering

A thesis submitted to the Nanyang Technological University
in fulfilment of the requirement for the degree of
Master of Engineering

2005

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SUMMARY

Heart disease encompasses many heart problems, but the most common is coronary artery disease. Coronary artery bypass surgery is sometimes recommended to correct the problem, by creating an alternate route of blood supply and providing new pathways to supply blood to the heart.

This Project examines the current Coronary Artery Bypass Grafting (CABG) techniques, develops models of two-dimensional end-to-end anastomoses and three-dimensional end-to-side anastomoses using ANSYS 5.4, a commercially-available finite element software package, analyses stresses and highlights key observations arising from the solutions of these models.

The results of the two-dimensional modelling of an end-to-end anastomosis suggest that the thickness of the graft is an important factor of consideration as it could potentially affect the stresses experienced. The thicker the graft thickness, the lower the magnitude of the Von Mises stress experienced and the more rigid the overall anastomosis would be.

The results of the three-dimensional modelling of an end-to-side anastomosis show that, from a mechanical stress analysis point of view, a larger anastomotic angle is desirable because it would result in lower stresses when measured in the radial, transverse and longitudinal directions. The resultant principal stresses experienced by the host and the graft also indicate a larger anastomotic angle as the preferred model because of lower stresses generated. Thus, analyses of these two modes of stresses recommend the 60-degree model as the preferred choice. When consideration is given to the wall shear stresses, the 30-degree model would be preferred as the chances of developing intimal hyperplasia is the lowest amongst the three models considered.

Based on the results of the study, the authoress recommends having an angle of 60 degrees as the anastomotic angle. This is because the 60-degree

model would result in minimal deformations and stresses and would minimise implications when blood is restored into the vessels after the bypass grafting. Though still in rudimentary form, it is intended that the analyses performed in this study will provide useful information to the cardiac surgeons and other researchers in this same area, in helping them identify the most optimal geometries and dimensions that will result in minimal stresses on the blood vessels during the suturing process.

CHAPTER 1 INTRODUCTION

1.1 Background

The heart's primary function is to pump blood to all parts of the body, bringing nutrients and oxygen to the tissues and removing waste products. When the body is at rest, it needs a certain amount of blood to achieve this function. During exercise or times when greater demands are placed on the body, more blood is required. To meet these variable demands, the heartbeat increases or decreases, and blood vessels dilate to deliver more blood or constrict during times when less blood is required.

Just like all tissue in the body, the heart requires oxygenated blood in order to function properly. Blood nourishes the heart through the left and right coronary arteries. Coronary artery disease is always due to thrombosis, which occurs when the arteries are narrowed by fatty lesions in the arterial walls, a process known as arteriosclerosis. Once atherosclerosis has developed, there may be further narrowing of the coronary arteries by the formation of blood clots. This process is known as thrombosis.

Whatever the reasons for reduced blood flow (fatty acid build-up, blood clot or spasm), the result is that there is not enough blood and oxygen reaching the heart muscle. Without enough blood and oxygen, the heart muscle begins to fail and subsequently dies. When the blood supply to the heart muscle is not sufficient for 15 to 30 minutes, the heart muscle will be impaired. This results in a heart attack or myocardial infarction. Reducing blood supply also causes electrical instability to the heart, making it impossible to pump blood again. When the heart stops pumping, the supply of blood to the brain will be cut off. If the blood is not restored within five minutes, the result can be permanent damage to the heart and brain or even death.

Neither the heart nor the brain damage can be repaired; therefore the injury leaves behind a permanent disability. There are an estimated 2 million people in the United States with heart failure. This refers to the inability of the heart to keep up with its workload. The term “failure” indicates the heart is not pumping effectively enough to meet the body’s needs for oxygen-rich blood, either during exercise or at rest. The term congestive heart failure (CHF) is often synonymous with heart failure but also refers to the state in which decreased heart function is accompanied by a build-up of body fluid in the lungs and elsewhere. Heart failure may be reversible, and people may live for many years after the diagnosis is made.

Heart disease encompasses many heart problems, but the most common is coronary artery disease – narrowing of the arteries supplying blood to the heart muscle. Plaque builds up in the arteries and blocks blood from getting to the heart, causing pain, heart attacks and even death. Well-known risk factors of coronary artery disease are family history, high blood pressure, smoking, diabetes, excess body fat and physical inactivity. Therefore, a healthy diet and appropriate medication that lower blood cholesterol can help to slow down the process that clogs arteries. Exercise and losing weight (under the doctor’s supervision) is also helpful for the strengthening of the heart. However, if a heart attack has already occurred, medications that control blood pressure and heart rate can help in reducing the chances of another heart attack. Balloon angioplasty, a non-surgical procedure, has been employed to treat patients with the disease. In the procedure, a catheter with a balloon on its tips is inserted into an artery in the leg. It is then guided into the narrowed coronary artery. Once there, the balloon is inflated, widening the narrowed area and increasing the blood flow to the heart. To increase the long-term success of the angioplasty, a stent is usually implanted during the process (Fabregues et. al., 1998).

In some cases, coronary artery bypass surgery is recommended to correct the problem. This is usually for cases of serious blockages in the coronary

arteries. Coronary artery bypass surgery creates an alternate route of blood supply, and provides new pathways to supply blood to the heart.

During the surgery, a connection is made between the major blood vessel of the body – the aorta (the left main, the right main, the circumflex and some of their subsidiary branches) – and the blocked coronary artery, beyond the area of obstruction, using a variety of conduits. In this way, even though nothing is done about the blockage itself, blood is provided to the heart via the ‘Bypass’. Hence the term coronary bypass surgery is derived (Hochman, 1982).

The most commonly used conduits are saphenous veins, which are stripped from a patient’s legs to correct varicosity, and are removed at the same time, when the bypass operation is performed.

Beyond this, there are also some suitable choices for the graft, such as the internal mammary artery, gastroepiploic artery, radial artery, inferior epigastric artery, subscapular artery, splenic artery and intercostals artery. Meanwhile, synthetic veins such as Dacron, Teflon and Polytetrafluoroethylene (PTFE) veins are also available in the commercial market and have been used as substitutes of the graft. These man-made synthetic veins are used only if the extracted autogenous vein (from the patient’s body) is not suitable for the bypass operation.

In this anastomotic operation, one end of the vein graft is sewn to the aorta, the main artery (known as proximal anastomosis), and the other end is attached to the coronary artery below the area of blockage (known as distal anastomosis). In this way, the oxygen-rich blood is taken directly from the aorta, bypasses the obstruction and flows through the graft to nourish the heart muscle.

Although the most widely used and reliable treatment option for coronary artery disease is coronary bypass surgery, the vascular grafting is not without complications (Vorp, 1997). Various failure modes have frustrated investigators for years. These include thrombosis – the primary cause of early (less than 30 days) graft failure, which is related to the interaction of

platelets with the graft and/or vessel wall, intimal hyperplasia – the major cause of mid-term (from one month to one year) graft failure, which is associated with the hyperplastic growth of smooth muscle cells and fibroblasts near the anastomoses of the graft to the host artery and graft atherosclerosis – susceptible cause for late failure (beyond one year).

According to the statistics of American Heart Association, 571 000 bypass procedures were performed on 355 000 patients in 1999 (Note that these numbers only represent the code and vessel data). Thus, it is impossible to determine the average number of vessels per patient. The trends in cardiovascular surgery indicate that there has been an increase in the number of bypass operations over the years. The saphenous vein in the leg is the most commonly used vessel for grafting. It has a one-year patency rate of 80% to 90%, and a ten-year patency rate of 50%; the internal mammary artery is also very commonly used – it has a higher one-year patency rate of 98%, and a ten-year patency rate of 90%; the radial artery in the arm is also quite popular among surgeons, its average one-year patency rate is 93.5% and five-year rate is 83%. The average patency of the bypass graft for a ten-year period is around 91%, before it becomes partially blocked or totally occluded, and before another operation has to be carried out.

It is therefore expected that numerous research works were carried out intensively to investigate the occlusion symptom or commonly known as stenosis. Previous studies have shown that both biological and mechanical factors are involved in the formation of stenosis. These include mitogenic factors, platelet activation and chronic endothelial injury (injury induced during injury), hemodynamic factors, compliance mismatch between graft and host vessels, and interactions between blood and graft materials (Nerem, 1992; White et. al., 1993; Keynon et. al. 2001).

Among these, hemodynamic factors were thought to be the most important factor (Friedman et. al., 1992; Stewart and Lyman, 1992; Hofer et. al., 1996, Vorp, 1997). Until now, a few hemodynamic factors have been considered, including low wall shear stress (Rittgers et. al., 1978; Binns et. al., 1989), high

or unidirectional shears (Fry, 1969; Friedman, 1992), wall shear rate gradient (Henry et. al., 1996, Kleinstreuer et. al., 1996) and flow separation (Hughes and How, 1995 and 1996). Furthermore, geometric factors such as anastomosis angle (Pietrabissa et. al., 1990; Fei et. al.; 1994, Hughes and How, 1995 and 1996; Henry et. al., 1996; Inzoli et. al., 1996; Loth et. al., 1997), wall curvature (Lei et. al., 1995; Rowe et. al., 1999; How et. al., 2000), flow rate ratio (Pietrabissa et. al., 1990; Fei et. al.; 1994, Hughes and How, 1995 and 1996; Henry et. al., 1996; Inzoli et. al., 1996), wave form (White et. al., 1993; Kleinstreuer, 1996; Ethier et. al., 1998) are also thought to influence the development of stenosis.

To determine the influence of these factors, several experimental techniques have been utilised to study the flow patterns of vascular anastomoses, including flow visualisation using dye-injection (Crawshaw et. al., 1980), laser illumination of particles suspended in the fluid (White et. al., 1993; Hughes and How, 1996) and the hydrogen bubble technique (Keynton et. al., 1991). Velocity profiles and wall shear stress have also been determined using the photochromic tracer techniques (Ohja, 1993; Ohja et. al., 1993) and laser Doppler anemometer (Keynton et. al., 1991; Loth et. al., 1997). Recently, numerical simulation has become an extremely valuable tool for performing a systematic study of complex flow patterns in anastomosis (Perktold et. al., 1991; Xu et. al., 1992; Steinman et. al., 1993; Lei et. al., 1996; Hofer et. al., 1996; Kleinstreuer et. al., 1996; Lei et. al., 1997).

Based on the numerous research works conducted thus far, one can see that focus on the studies of the region of anastomoses thus far have been largely confined to studying the fluid flow patterns and the connecting geometries. The nature of such studies conducted thus provided room for analyses of the mechanical parameters – such as magnitudes of stresses and stress patterns – to be incorporated to provide more thorough insights into the problem.

1.2 Objectives

The objectives of this project are to:

1. Assess current CABG techniques;
2. Develop finite element models for two-dimensional end-to-end anastomoses and three-dimensional end-to-side anastomoses using ANSYS 5.4, a commercially-available finite element software package;
3. Analyse stresses and trends in the solutions of the finite element models; and
4. Highlight key findings to aid the remaining project team members in their research work to design an interposition sleeve for joining the aorta to vein graft and vein graft to the coronary artery in a bypass operation;

The abovementioned objectives form the scope of work undertaken by the author during her tenure of the part-time Masters in Engineering programme in Nanyang Technological University, from November 2002 to June 2004.

1.3 Outline of Report

The breakdown of the report by chapters is as follows:

The findings of the literature review conducted are documented in Chapter 2. The literature review is based on past studies conducted in trying to establish the relationship between intimal hyperplasia to factors such as flow rate, wall shear stress, wall shear rate and other geometries. Studies done in relation to establishing the mechanical properties of the arteries and veins, and the effects of factors such as age, pre-stress and localisation is also presented. Studies relating the homogeneity and incompressibility of the blood vessel walls are also documented. Finally, there is a section on the current Coronary Artery Bypass Grafting techniques.

Chapter 3 described the theories that were relevant to this Project. These included the theory of the cardiovascular system, blood vessels and blood flow. Theories on mechanical failure were also described and were subsequently used for the Chapter on Discussion

Chapter 4 describes the methodology for finite element modelling and the assumptions used in deriving the results. The finite element modelling can be classified into two major categories, namely: two dimensional finite element modelling of end-to-end anastomoses and three-dimensional finite element modelling of end-to-side anastomoses. Within these two major headings, variations in the models were made.

Chapter 5 presents the results and discussions arising from the development and solution of two-dimensional end-to-end anastomotic models. Chapter 6 presents the results and discussions arising from the development and solution of three-dimensional end-to-side anastomotic models.

Chapter 7 concludes the study and Chapter 8 provides recommendations for future work that may be undertaken.

CHAPTER 2 LITERATURE REVIEW

The literature review conducted can be classified into three parts: (1) Studies done in relation to intimal hyperplasia; (2) Mechanical properties of blood vessels and (3) Current techniques for performing Coronary Artery Bypass Grafting (CABG).

Intimal hyperplasia refers to the condition where there is an abnormal multiplication or increase in the number of cells in the inner layer of the blood vessel. A number of studies have attributed the onset of intimal hyperplasia to certain factors. Literature review done in relation to intimal hyperplasia involved studying the relationship between intimal hyperplasia and:

- Flow rate
- Wall shear stress
- Wall shear rate
- Effect of other geometries

2.1 Relationship between Flow Rate and Intimal Hyperplasia

A number of studies had focused on establishing a relationship between the flow rate and the presence of intimal hyperplasia. These studies have concluded that lower flow rates have a direct impact on the formation of intimal hyperplasia.

In an experimental study conducted by Berguer et. al. (1980), the vein graft carrying low flow rates appeared to develop more intimal hyperplasia than those carrying high flow rates when using an end-to-end anastomosis in dogs. LoGerfo et. al. (1983) investigated the precise location and progression of anastomotic hyperplasia and its possible relationship to flow conditions by studying the Dacron grafts in 28 dogs. Transmission electron microscopy showed that the hyperplasia consisted of collagen-producing smooth muscle

cells. Anastomotic hyperplasia was significantly greater at the downstream anastomosis, which was progressive with time and was the primary cause of the failure of Dacron arterial grafts in this study.

2.2 Relationship between Wall Shear Stress and Intimal Hyperplasia

Other studies had established a direct correlation between low wall shear stress and the formation of intimal hyperplasia. Morinaga et. al. (1985) investigated the effect of wall shear stress in the intimal thickening of the arterially transplanted autogenous veins in dogs. There were two Groups of Graft. In Group One, the grafts were implanted at high flow rate of 79.7 ± 3.2 ml/min and low wall shear stress of 33.1 ± 1.9 dynes/cm². In Group Two, grafts were implanted at low flow rate of 2.9 ± 1.8 ml/min and high wall shear stress of 178.8 ± 11.0 dynes/cm². The results showed that the intimal thickness after implantation in Group One was statistically significant compared to Group Two, and it is the wall shear stress, and not the rate of blood flow, that was the essential hemodynamic factor related to intimal hyperplasia.

Binns et. al. (1989) studied forty polytetrafluoroethylene grafts with varying internal diameters. The grafts were inserted end-to-end in the femoral and carotid arteries of 10 mongrel dogs. Total flow and diameter were measured, and grafts were stained and analyzed by computer for anastomotic neointimal thickening. Low shear stresses produced greater amounts of pseudointimal thickening within polytetrafluoroethylene grafts and neointimal thickening at their anastomoses.

Bassiouny et. al. (1992) studied the localization of experimental anastomotic intimal thickening in relation to known biomechanical and hemodynamic factors. Bilateral iliofemoral saphenous veins (extracted from the animals) and synthetic PTFE grafts were implanted in 13 mongrel dogs. Two separate and distinct regions of intimal hyperplasia formation were identified: The first

region of intimal hyperplasia formation was along the suture line. The second distinct type of intimal hyperplasia was developed on the arterial floor and was the same in both PTFE and vein anastomoses. Intimal thickening was absent along the graft hood where flow was laminar and high shear with short particle residence time. Arterial floor intimal thickening developed in a region corresponding to the stagnation zone where low and oscillatory shear prevailed. Flow patterns associated with relatively low shear and long particle residence time were also found along the heel and lateral walls of sinus where suture line intimal thickness were present. Studies concluded that the suture line intimal thickening, which represents vascular healing, is more prominent with the PTFE anastomosis and may be related to compliance mismatch.

Keynton et. al. (2001) investigated the potential interaction between local hemodynamics and vascular wall response and ascertained a positive correlation between low wall shear stress and intimal hyperplasia.

2.3 Relationship between Wall Shear Rate and Intimal Hyperplasia

Kenneth et. al. (1991) investigated the effect of varying blood flow rates on the accumulation of thrombotic material within arteries, with the use of the shear rate as an index of flow across the inner wall surface. The formation of thrombus on altered arterial surfaces is highly dependent on the flow characteristics at the blood-intimal interface. Regions of high shear rate appeared to be most prone to thrombus growth, with increasing platelet and fibrin deposition.

Friedman et. al. (1992) investigated the effect of arterial compliance and non-Newtonian rheology on correlations between intimal thickness and wall shear. A minimally diseased human aortic bifurcation was used to replicate flow-through casts. Results showed that the intima was thicker at sites exposed to higher shear rates. There was no significant effect of either model

compliance or fluid rheology on the slopes of the correlations between the intimal thickness and any normalised shear rate measure.

Keynton et. al. (1999) used a specially designed pulse ultrasonic Doppler wall shear rate (PUDSWR) measuring device to evaluate the effect of graft calibre, a surgically controllable variable, upon local hemodynamics, which, in turn, plays an important role in the eventual development of anastomotic hyperplasia. In the study, tapered (4 to 7 mm I.D) 6cm long grafts were implanted bilaterally in an end-to-side fashion with 30 degree proximal and distal anastomosis to bypass occluded common carotid arteries of 16 canines. The results showed that the wall shear rate (WSR) varies widely within end-to-side distal graft anastomoses, particularly along the artery floor, and may play a role in the development of intimal hyperplasia through local alteration of mass transport and mechano-signal transduction of the endothelium.

2.4 Effects of other geometries on the formation of intimal hyperplasia

The magnitude of wall shear is in turn affected by variations in anastomotic angle and flow rate. Keynton et. al. (1991) performed a steady in-vitro study on the distal arterial bypass junctions, with the purpose of examining the effects of junction angle and flow rate on the local velocity field. Three anastomotic angles with 30, 45 and 60 degree junction angles were fabricated using Plexiglas tubing of 25.4mm in diameter.

Both flow visualisation and (using Hydrogen bubbles) and Laser Doppler anemometry (LDA) measurements were performed and had revealed skewed velocity profiles towards the outer wall with a flow split around a clear stagnation point along the outer wall. Axial velocities and shear rates along the outer wall were higher than those along the inner wall and occurred in the order of 45, 60 and 30 degrees junction angles. This study identified changes in wall shear, which varied with the anastomotic angle and flow rate.

2.5 Sites of Occurrence of Intimal Hyperplasia

A number of studies also focused on possible sites in which intimal hyperplasia has the tendency to occur. Shu et. al. (1989) concluded that the occurrence of intimal hyperplasia increases around the joint of anastomoses, where there is more variation in wall shear stress. Dye injection technique was used to study the flow patterns. Detailed flow measurements were carried out using laser Doppler anemometry (LDA) and the wall shear stress were calculated from the velocity profile gradients near the wall during each phase. The results showed that more variations of wall shear stress were limited around the joint of anastomosis, where it is prone to intimal hyperplasia.

Staalsen et. al. (1995) implanted polyurethane grafts of diameter 8mm were implanted into 10 pigs in order to study the effect of the anastomosis angle (15 Deg, 45 deg and 90 Deg) on the flow fields at the end-to-side anastomoses. Results suggested that the smallest anastomosis (15 degrees) is associated with the least flow disturbances at the toe and one diameter downstream. The study confirms the in-vivo existence of regions with low and reverse velocities at the preferential sites in vascular end-to-side anastomoses where neointimal hyperplasia tends to form.

Hughes and How (1995 and 1996) studied the effects of geometry and flow division in models of the proximal and distal end-to-side anastomoses. Flow models made of polyurethane elastomer were designed at three anastomotic angles of 15, 30 and 45 degrees. Flow visualization was conducted under steady and pulsatile flow conditions using planar illumination of suspended tracer particles. The presence of a patent distal artery had a significant effect on the overall flow pattern and led to the formation of a large recirculation region at the toe of the anastomoses. The secondary flow components in the graft were more pronounced in pulsatile flow particularly during deceleration of the flow waveform. The results showed that intimal hyperplasia occurred in

regions of flow separation at the toe and the heel, and flow stagnation on the floor of the anastomoses

2.6 Mechanical Properties of the artery and vein

In this section, some of the mechanical properties of selected tissues of the human circulatory system are briefly introduced. Characteristically, a wide range of results is obtained when examining these properties. The experimental data depend on numerous factors of both experimental and biological nature. The biological aspects include the age, sex, medical condition (e.g. Atherosclerosis), physiological activity and type of sample. The experimental aspects include factors such as the condition of the sample (tissue water content), sample orientation, experimental temperature and loading rate. To compare the different results, accurate knowledge of these conditions is necessary. Soft biological tissues manifest hysteresis in the course of mechanical strain and the stress-strain dependence fails to follow Hooke's law. This was demonstrated for arteries as early as the last century by Wertheim. Moreover, Purinya and Kasyanov (1980) claimed that, at the initial phase of the experiment, sufficient accuracy of the measurement is not guaranteed with sample slippage potentially occurring at high loads. Apparently, these factors must be taken into account of. An illustration of the results of the experiments, as presented for the aorta, some arteries and vena saphena by Ceders et. al. (1975) is presented in Table 2.1 below. These measurements were carried out for a relatively narrow age group (39 to 49) so as to eliminate the age effect. The experiments have shown the differences in the tangent modulus of elasticity in the longitudinal direction significant between the different arteries (with the exception of vena saphena magna) to be only minor at low levels of stress. However, their values change substantially with the stress level. Apparently, without knowing the specific stress level and the moment, when the different moduli of elasticity were

measured during the experiment (the effect of hysteresis), the results are meaningful only in comparison within one experiment.

Table 2.1 Selected mechanical properties of different blood vessels of man in the longitudinal direction (Adapted from Purinya et. al.)

Vessel	h_o (mm)	R_m (MPa)	ϵ_{max}	E_t for various stresses (MPa)			
				0.05	0.10	0.20	0.40
Aorta – anterior wall	2.35	1.11	0.46	0.84	1.36	2.52	3.89
	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$
	0.03	0.11	0.03	0.07	0.08	0.22	0.20
Aorta – posterior wall	2.26	0.71	0.42	0.10	1.72	2.44	3.45
	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$
	0.15	0.09	0.04	0.14	0.18	0.23	0.29
Arteria – carotis communis	1.66	1.99	0.61	0.78	1.25	2.49	4.24
	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$
	0.04	0.27	0.04	0.10	0.07	0.22	0.32
Arteria – carotis interna	1.59	1.56	0.52	0.82	1.31	2.51	4.22
	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$
	0.10	0.20	0.12	0.16	0.19	1.15	1.44
Arteria – iliaca communis	1.63	1.86	0.39	0.97	1.70	3.11	5.65
	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$
	0.12	0.39	0.04	0.19	0.23	0.52	0.78
Arteria – femoralis	1.89	1.32	0.40	1.01	1.68	3.14	5.36
	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$
	0.11	0.18	0.03	0.08	0.15	0.30	0.50
Vena saphena magna	1.52	3.92	0.37	2.13	3.46	5.30	8.50
	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$	$\pm$
	0.12	1.09	0.06	0.44	0.71	1.00	1.32

Note:

h_o – initial wall thickness

R_m – tensile strength in the longitudinal direction

ϵ_{max} – limit strain in the longitudinal direction

E_t – tangent modulus of elasticity in the longitudinal direction

Arteries, to which the heart pumps the blood, form an integral part of the system of circulation. Their wall, as well as that of veins, is comprised of three layers: tunica intima (interna), tunica media and tunica adventitia (externa) (Klika et. al., 1974; Canfield et. al., 1987). According to the vessel type, the internal structures of the individual layers may differ significantly.

Tunica intima (the internal layer) is formed by a layer of endothelium cells, which line all the vessels. Under it, there is a thin sub-endothelium fibrous layer and then a marked elastic membrane (the membrane elastica interna), which is formed by elastic and collagenous fibres. With an adult, for example, tunica intima of aorta reaches 100 μ m. Tunica media (the middle layer) is a thick, prevailing muscle layer. Its skeleton is formed by 40 to 60 elastic membranes (Klika et. al., 1974, Bergel et. al., 1961). Running among these membranes, at an angle of 30° to 50° relatively to the vascular longitudinal axis, are smooth muscle fibres, which are wrapped in collagenous fibres. This basically spring-like arrangement is advantageous to the periodic loading of the vascular wall since it enables vessel contraction to the original shape after the pulsatile wave passage. At the border of the tunica media and tunica adventitia (the outer layer) there is a membrane consisting of thick longitudinally arranged elastic fibres and spiral bundles of collagenous fibrils (Fung, 1972). The outer layer is formed mostly by collagenous fibres.

Patel and Vaishnav (1972) claimed that elastin is the only component engaged in the first phase of the vascular wall deformation (its extensibility being as many as 150% of the initial length). Under physiological conditions, both elastin and collagen fibres are engaged. With large deformations only the effect of collagenous fibres is manifested. Rachev (1977) claimed that elastin and collagenous fibres are responsible only for the so-called passive properties of the vascular wall and that their behaviour could be described by constitutive equations. However, the ultimate behaviour of the vascular wall is determined by the smooth muscles and the residual stress. Due to its high extensibility, no marked effects are apparent during the so-called passive deformation (for example, pulsatile wave propagation). However, smooth muscle activity may be invoked by the nervous system resulting in significant effect for the vascular wall behaviour. Evidently, the respective contents of elastin, collagen and smooth muscles in the vascular wall will affect its mechanical behaviour.

By the composition and scope of the individual layers of the artery wall, arteries are sub-divided into the following three basic types:

- (i) Arterioles – these are arteries of the smallest diameters (about 100µm) responsible for the primary blood flow control in the respective area. Smooth muscles prevail in their walls.
- (ii) Arteries of small and medium diameter – these are also classified as arteries of the muscular type, as their walls of considerable thickness are formed by smooth muscles to a large proportion.
- (iii) Arteries of large diameter – these are the so-called elastic type arteries. The walls of these arteries contain a relatively high proportion of elastin causing the characteristic yellow colouring of the wall. The wall thickness of these arteries is small relatively to their large lumen.

Blood vessels with inner diameter over 1mm have their own nourishing vessels, the so-called “vessel of vessels” (vasa vasorum). In arteries, they supply the outer layer, where they ramify. The inner layer and a part of the middle layer are nourished from the blood flow through diffusion and effusion. In veins, these nourishing vessels penetrate deeper and they intervene as far as to the middle and internal layers due to a low content of nutriment in venous blood.

Mechanical properties and the effect of different factors on blood vessels are dealt with below. For basic orientation, the diameters of individual arteries, as measured by Gyurko and Szabo (1968) are presented in Table 2.2 below:

Table 2.2 Outer diameters of selected arteries in adult humans. Source: Gyurko and Szabo (1968).

Artery	Minimum diameter (mm)	Maximum diameter (mm)	Mean diameter (mm)
Aorta	16.00	30.30	24.14
Arteria coronaria R	1.59	4.15	2.71
L	1.91	3.80	2.68
Truncus brachiocephalicus	7.00	15.30	10.41
Aorta descendens	13.40	22.00	18.21
Aorta abdominalis	9.55	16.60	13.40
Truncus coeliacus	4.15	7.64	5.32

Artery	Minimum diameter (mm)	Maximum diameter (mm)	Mean diameter (mm)
a. mesenterica inferior	2.23	3.50	2.74
Arteria renalis R	2.87	5.73	4.20
L	3.18	5.73	4.23
a. iliaca communis R	6.09	11.46	7.99
L	4.46	12.10	8.05
1. iliaca externa	4.77	9.87	7.39
1. iliaca interna R	2.87	8.91	5.09
L	1.91	8.28	4.97
Arteria femoralis R	2.87	8.91	5.09
L	1.91	8.28	4.97
Arteria poplitea	3.18	5.73	4.11
a. carotis communis	4.77	8.28	6.27
a. carotis externa R	2.87	5.73	3.82
L	2.55	5.41	3.85
a. carotis interna R	3.80	7.00	5.09
L	3.50	6.68	5.00
Arteria vertebralis R	3.18	4.15	3.69
L	3.18	4.77	3.85

Note: R = Right, L = Left

2.6.1 The effect of pre-stress

In the physiological state, the arteries in the human body are pre-stressed (Patel, 1972). For example, according to Fung, the natural length of vessel is one of the more elusive quantities to measure. Kasianenko (1983) claimed that the degree of contraction of vessels samples after they are taken out of the organism is given directly by the type of vessel. With experiments on animals they ascertained that this shortening is highest in vessels, in the wall of which smooth muscles prevail (47% to 58%). With vessels of elastic type, the shortening is substantially smaller (29% to 31%).

Bergel (1972) stated that contraction of vessels upon removal from the body depends on age. For example, arteria femoralis contracts by 40% on average, and with older persons, 25% on average. Aorta abdominalis shortens by 30% and 15% with young and older persons respectively. Jartsev (1974) indicated that the shortening of various parts of the aorta, after it is taken out of organism, changes with age. With persons under 30, this shortening was found to range from 14% to 20% on average. With persons

over 50, this shortening is more than ten times smaller (1% to 1.5%). In the case of atherosclerosis, no shortening of samples is observed.

2.6.2 The effect of localization

Recognizing the fact that the wall composition of different arteries differs and that the functional conditions of one artery may change with the distance from the heart, a number of mechanical properties depend on the sample localization. Kasianenko et. al. (1983) gave, for different arteries, the following average maximum elongations, in percentage, in longitudinal direction:

- Aorta abdominalis – 148%
- Aorta thoracica – 145%
- Arteria femoralis – 124%
- Arteria carotis communis – 124%

By comparing individual vascular wall thickness, Purinya et. al. (1980) stated that the highest values are with abdominal aorta and in 64% the anterior wall is thicker than the posterior wall. As far as the strength is concerned, on average it is 1.5 times higher with the anterior wall in 73% in comparison with the values with the posterior wall.

Figure 2.1 shows the stress-strain relationship (longitudinal direction) for the iliac artery (arteria iliaca) (a), femoral artery (b), the posterior (c) and anterior (d) parts of the abdominal aorta, internal carotid artery (arteria carotis interna) (e) and the common carotid artery (arteria carotis communis) (f). The figure clearly shows the highest tensile strength and the maximum strain are with the common carotid artery.

Figure 2.1 Stress-strain relationship in simple tension tests of different arteries in the longitudinal direction – a – iliac artery, b – femoral artery, c, d – the posterior and anterior parts of abdominal aorta, e, f – internal, resp. common carotid artery, R_m – tensile strength, T_R – Lagrange stress. Adapted from Purinya et. al. (1980)

2.6.3 The effect of sample orientation

As with the effect of localization, also in this case, the specific mechanical properties depending on the sample orientation reflect the functionality of the vascular wall. For example, Zavalishin et. al. (1983) studied in detail the mechanical properties of the pulmonary artery and its valve leaflets, demonstrating clearly that the stress-strain relationship depends not only on the position, but also on the orientation of the sample. Table 2.3 below shows the tensile strength in the longitudinal direction R_{m1} and circumferential direction R_{m2} of the iliac artery wall in dependence on age, as presented by Chamin (1978). The experimental findings indicate that the strength in the circumferential direction is higher than that in the longitudinal direction.

Table 2.3 The tensile strength of the human iliac artery wall in dependence on age for longitudinal (R_{m1}) and circumferential directions (R_{m2}). (Adapted from Chamin).

Age (years)	R_{m1} (MPa)	R_{m2} (MPa)
11 – 20	4.14 ± 0.19	4.63 ± 0.36
21 – 30	2.20 ± 0.11	3.07 ± 0.17
31 – 40	1.55 ± 0.13	1.76 ± 0.19
41 – 50	1.03 ± 0.08	1.32 ± 0.08
51 – 60	0.79 ± 0.14	1.21 ± 0.14
61 – 70	0.79 ± 0.11	0.93 ± 0.07
Over 71	-	0.88 ± 0.07

According to some U.S authors, 16% of all deaths in the U.S are caused by transverse rupture of the aorta. An explanation of the condition was presented by Purinya and Kasyanov (1980). It was claimed that the mechanical properties of the aorta were more favourable in the circumferential direction causing a transverse disruption to originate in certain situations with the chest colliding with the steering wheel. However, the primary cause of this rupture may be the weakening of the aortic arch wall at the point where ligamentation arteriosum is attached. This ligament connects the aorta with the pulmonary artery (less often with the pulmonary trunk (truncus pulmonalis). At the embryonal stage, this link is formed by the arterial duct (ductus arteriosus), through which most of the blood from the pulmonary branch flowed to the aorta. After birth, this flow is closed and the link changes into a ligament. The weakening of the aortic wall occurring at the point of the attachment can result in ruption developing in the circumferential direction due to the mechanical properties of the wall.

2.6.4 The effect of age

Age and medical condition are two essential aspects to be taken into account when assessing experimental results involving mechanical properties of arterial walls. As early as the 1960s, Yosimatsu (1958) noticed the dependence of tensile mechanical properties of coronary arteries on age, namely for the longitudinal direction. In Table 2.4 below, it can be observed that the value of the force (per unit of thickness) by which arterial wall is failed; tensile strength and maximum elongation in percent are higher in the 10 – 19 age group. The highest decrease with age is observed for the maximum elongation (a decrease of over 50%). Yamada (1970) dealt with the issue in great detail.

Table 2.4 Coronary Artery tensile mechanical properties.

Age (years)	10 - 19	20 - 39	40 - 59	60 – 79
Tensile force up to failure per unit of thickness (Nmm^{-1})	0.85 ± 0.03	0.82 ± 0.02	0.82 ± 0.02	0.79 ± 0.03
Tensile Strength (MPa)	1.40 ± 0.03	1.14 ± 0.09	1.04 ± 0.05	1.04 ± 0.05
Maximum elongation (%)	99 ± 2	78 ± 2	68 ± 4	45 ± 4

Table 2.5 below illustrates the tensile mechanical properties of arteries, as predicted by Japanese researchers. Again the highest decrease with age is that observed for the maximum elongation. The highest tensile force to failure, related to unit thickness, was observed in the ascending part of the aorta in the circumferential direction, the highest tensile strength being observed in the carotid artery in the circumferential direction. The highest elongation was also observed in the carotid artery, this time in the longitudinal direction. The highest values were mostly found in the 10 to 19 age group. In keeping with the above rationale, the detailed analysis of the Table indicates the mechanical properties in the circumferential direction to be mostly more favourable than those in the circumferential direction.

In the opposite case, the measured values in the circumferential direction decrease more slowly than those in the longitudinal direction (such as the maximum tensile force and tensile strength of the abdominal aorta, the maximum elongation of the carotid artery). Only with carotid artery, the tensile strength decreases more quickly in the circumferential direction than in the longitudinal direction.

Table 2.5 Tensile mechanical properties of some human arteries.

age (years)		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79
		Tensile force up to failure per unit of thickness (Nmm ⁻¹)							
ascending part of a.	L	1.11 ±0.14	1.48 ±0.07	1.48 ±0.07	1.46 ±0.15	1.39 ±0.09	1.31 ±0.09	1.27 ±0.09	1.27 ±0.09
	C	1.67 ±0.19	2.45 ±0.15	2.45 ±0.15	2.22 ±0.17	2.14 ±0.20	2.01 ±0.16	1.94 ±0.13	1.94 ±0.13
thoracic part of a.	L	1.22 ±0.10	1.26 ±0.04	1.45 ±0.06	1.36 ±0.05	1.36 ±0.05	1.17 ±0.07	1.08 ±0.04	1.08 ±0.04
	C	1.47 ±0.05	1.63 ±0.01	1.81 ±0.06	1.69 ±0.08	1.64 ±0.07	1.49 ±0.09	1.46 ±0.08	1.46 ±0.08
abdominal part of a.	L	1.63	1.63	1.32	1.12	1.12	1.05	1.05	—
	C	1.54	1.54	1.54	1.68	1.68	1.35	1.35	—
pulmonary artery	L	0.62	0.88	0.85	0.82	0.74	0.70	0.69	0.69
	C	1.06	1.42	1.21	1.21	1.21	1.21	1.09	1.09
carotid artery	L	0.95	0.95	1.19	1.06	1.06	1.06	1.06	—
	C	1.16	1.16	1.36	1.00	1.00	0.86	0.86	—
		Tensile strength (MPa)							
ascending part of a.	L	0.86 ±0.06	0.86 ±0.06	0.76 ±0.06	0.73 ±0.07	0.68 ±0.02	0.68 ±0.02	0.68 ±0.02	0.68 ±0.02
	C	1.36 ±0.10	1.36 ±0.10	1.18 ±0.05	1.18 ±0.05	1.04 ±0.05	1.04 ±0.05	1.04 ±0.05	1.04 ±0.05
thoracic part of a.	L	1.22 ±0.08	1.02 ±0.03	1.02 ±0.03	1.02 ±0.03	0.89 ±0.04	0.64 ±0.04	0.53 ±0.08	0.53 ±0.08
	C	1.49 ±0.07	1.31 ±0.03	1.31 ±0.03	1.06 ±0.04	1.06 ±0.04	0.74 ±0.03	0.74 ±0.03	0.74 ±0.03
abdominal part of a.	L	1.62	1.62	1.12	0.84	0.84	0.70	0.70	—
	C	1.52	1.52	1.22	1.22	1.22	0.90	0.90	—
pulmonary artery	L	0.70 ±0.04	0.70 ±0.04	0.64 ±0.04	0.64 ±0.04	0.54 ±0.02	0.54 ±0.02	0.54 ±0.02	0.54 ±0.02
	C	1.16 ±0.09	1.16 ±0.09	0.93 ±0.04	0.93 ±0.04	0.93 ±0.04	0.93 ±0.04	0.84 ±0.05	—
carotid artery	L	1.38	1.38	1.38	1.19	1.19	1.02	1.02	—
	C	1.63	1.63	1.63	1.14	1.14	0.80	0.80	—
		Maximum elongation (%)							
ascending part of a.	L	60±4	111±5	106±8	91±11	79±7	74±5	68±8	68±8
	C	56±5	111±8	104±8	84±12	79±8	70±5	62±5	62±5
thoracic part of a.	L	85±3	85±3	74±2	74±2	62±3	56±3	43±3	43±3
	C	83±4	98±2	98±2	85±3	74±4	74±4	60±2	34±9
abdominal part of a.	L	102	102	94	69	69	56	56	—
	C	103	103	114	87	79	62	62	—
pulmonary artery	L	72±6	141±13	132±9	108±7	108±7	100±8	93±3	93±3
	C	64±4	134±10	126±8	94±6	94±6	87±5	87±5	87±5
carotid artery	L	145	145	100	100	87	60	60	—
	C	113	113	98	98	88	63	63	—

L – longitudinal direction, C – circumferential direction

Kasyanov and Kregers (1975) dealt with the properties of the abdominal aorta and the femoral artery. They studied individuals of average health subdivided into 20 to 40 and 41 to 60 age groups. They studied the dependence of the stress-strain relationship on age, type of vessel and direction of load. The dependence of the tensile strength on age can be expressed, according to these authors, by a simple equation of the form

$$R_m^i = a^i + b^i T \quad (2.1)$$

where T is the age and a^i and b^i are the correlation coefficients obtained from experimental data by the least squares method. Using the notation $i=1$ for longitudinal and $i=2$ for circumferential direction, the following constants were obtained:

Table 2.6 Constants obtained for abdominal part of aorta and femoral aorta.

Abdominal part of aorta	$a^1 = 0.2956 \text{ MPa}$	$b^1 = -0.0042 \text{ MPa.yr}^{-1}$
	$a^2 = 0.3395 \text{ MPa}$	$B^2 = -0.0047 \text{ MPa.yr}^{-1}$
Femoral aorta	$a^1 = 0.3734 \text{ MPa}$	$b^1 = -0.0063 \text{ MPa.yr}^{-1}$

Ozola et. al. (1983) carried out a detailed analysis of mechanical properties of the coronary arteries. The samples were taken from 102 male bodies of the age from 4 days to 85 years. They found out that the strain energy density of the right coronary artery achieves its maximum values at the age of 20 to decrease non-linearly thereafter. The tangent modulus of elasticity of the coronary arteries reaches its minimum value also at around the age of 20 to increase thereafter. The authors claim the collagenous fibres content to be the main factor determining the stiffness characteristics of the vascular wall. The higher the collagenous content, the stiffer is the wall. The opposite situation was observed for elastic fibres. It can therefore be inferred that the ratio of content of the collagenous and elastic fibres of coronary arteries grows with age. Since this assessment is only a phenomenological observation, it requires closer specification of the state of the particular individuals.

Ozolanta and Purinya (1985) undertook a similar analysis. They examined the coronary arteries of 71 men and 50 women. The samples were divided into six age groups: I – 0 to 1 year (9 samples), II – from 1 to 7 years (15 samples), III – from 8 to 19 years (15 samples), IV – from 20 to 39 years (34 samples), V – from 40 to 59 years (27 samples), VI – from 60 to 80 years (21 samples). The samples were taken from the right coronary artery at its ostium, and from the left one behind ramus circumflexus. Beginning from the III age group sample already, symptoms of atherosclerosis were observed in the sample walls. The samples with major sclerotic changes were not included in the experiments. Figure 2.2 shows the stress-strain relationships

for the circumferential direction of the right and left coronary artery of men. It is evident that this dependence changes the most with age with the left coronary artery. The deformability decreases significantly for the age group IV already. With the right coronary artery, this decrease is evident with the age group V only.

Figure 2.3 presents similar dependencies for the right and left coronary arteries of women. Here the maximum loss of deformability with age is comparable for both arteries. With the right artery, however, the largest change was observed in age group IV already, with the left one in the age group V. The authors also studied the change of the wall thickness and the outer diameter in dependence on age. The results of measurements are presented in Figures 2.4 to 2.7. The results indicate that with men, the thickness of the coronary arteries grows with age (with the exception of a moderate decrease of thickness in age group V observed for the left coronary artery) from $0.20 \pm 0.03\text{mm}$ to $0.85 \pm 0.21\text{mm}$ with the left artery and from $0.15 \pm 0.02\text{mm}$ to $0.78 \pm 0.10\text{mm}$ with the right artery. With women, the thickness of the coronary artery also grows with age, but only until age group IV to decrease thereafter. The dependence of the outer diameter on age can be described analogously. The measurements showed that, for example, the coronary arteries outer diameter decreased with men with growing age from $1.26 \pm 0.15\text{mm}$ to $3.48 \pm 0.90\text{mm}$ with the right coronary artery and from $1.51 \pm 0.12\text{mm}$ to $4.16 \pm 0.40\text{mm}$ with the left coronary artery. Important conclusions proceed from the above dependencies. The optimum deformation and tensile strength parameters in the circumferential direction of the coronary arteries are observed in men approximately up to 40 years and with women up to 50 years. Thereafter, a significant decrease in the tangent modulus of elasticity occurs, accompanied by a decrease in the deformability and an increase of the inner diameter of the measured sections (Zhou et. al., 1988). The changes in women are observed to proceed more evenly than those in men.

Figure 2.2 Stress-strain relationships for the right (a) and left (b) coronary artery of healthy men (in circumferential direction). T_R – Lagrange stress: I – from the birth to 1 year; II – 1 to 7 years; III – 8 to 19 years; IV – 20 to 30 years; V – 40 to 59 years; VI – 60 to 80 years. Adapted from Ozolanta and Purinya (1985).

Figure 2.3 Stress-strain relationship for the right (R) and left (L) coronary artery of women (in circumferential direction). Adapted from Ozolanta and Purinya (1985).

Figure 2.4 Dependence of thickness h_0 of the left (L) and right (R) coronary artery of men on age. Adapted from Ozolanta and Purinya (1985).

Figure 2.5 Dependence of thickness h_0 of the left (L) and right (R) coronary artery of women on age. Adapted from Ozolanta and Purinya (1985).

Figure 2.6 Dependence of outer diameter D_0 on the left (L) and right (R) pulmonary coronary artery of men on age. Adapted from Ozolanta and Purinya (1985).

Figure 2.7 Dependence of outer diameter D_0 on the left (L) and right (R) pulmonary coronary artery of women on age. Adapted from Ozolanta and Purinya (1985).

Table 2.7 shows the composition of the coronary arteries walls, in grams per 100 grams of dry tissue. It is evident from the presented summary that the contents of collagen and elastin decrease with age, an observation out of keeping with the conclusion presented by Ozola (1983). A much higher content of collagen with birth coronary arteries was observed in newborn infants. Considering the changes of hexurone acid and hexozamine content with age the collagen-elastin model is probably not sufficient for describing the influence of age on the change in mechanical properties of the coronary arteries.

Table 2.7 Individual components contained in coronary artery wall in g/100g of dry tissue in dependence on age.

component	coronary artery	age (years)					
		1	1-7	8-19	20-29	40-59	60-80
collagen	L	40.70 ±1.65	25.28 ±1.26	25.00 ±2.59	27.67 ±2.94	27.69 ±1.55	26.51 ±0.43
	R	44.10 ±0.94	24.69 ±1.01	29.10 ±1.25	27.40 ±0.86	25.90 ±1.67	20.99 ±1.29
elastin	L	7.50 ±0.65	10.00 ±0.65	4.88 ±0.81	6.66 ±0.71	8.52 ±0.61	10.32 ±1.73
	R	11.60 ±0.53	9.28 ±0.36	3.75 ±0.48	6.74 ±0.68	9.19 ±0.29	10.32 ±1.73
collagen/ elastin	L	5.43	2.53	5.10	4.13	3.26	2.57
	R	3.80	2.15	7.76	4.08	2.81	1.72
hexurone acids	L	0.41 ±0.04	0.60 ±0.04	0.63 ±0.07	0.38 ±0.07	0.49 ±0.06	0.50 ±0.05
	R	0.31 ±0.03	0.70 ±0.06	0.83 ±0.02	0.41 ±0.03	0.53 ±0.01	0.54 ±0.07
hexozamine	L	1.00 ±0.19	0.62 ±0.07	1.07 ±0.16	0.81 ±0.13	1.16 ±0.23	0.97 ±0.05
	R	1.03 ±0.11	0.45 ±0.09	0.60 ±0.11	1.03 ±0.06	1.08 ±0.13	0.83 ±0.09

L - left, R - right

Ceders et. al. (1975) studied the mechanical properties of the abdominal aorta close above its bifurcation into the common iliac arteries. The experiments were carried out for two age groups from 30 to 39 years (Group A) and from 40 to 57 years (Group B), and uniaxial and biaxial tensional experiments were carried out with the samples. The results are presented in Table 2.8.

Table 2.8 Tensile mechanical properties of human abdominal aorta.

		30-39 years	40-57 years
tensile strength (MPa)	UA	L 1.88±0.29	0.87±0.11
		C 2.10±0.14	1.30±0.26
maximum elongation	UA	L 2.08±0.07	1.58±0.02
		C 2.28±0.11	1.89±0.05
limit stress intensity (MPa)		1.06±0.19	0.42±0.04
maximum elongation	BA	L 1.495±0.077	1.348±0.040
		C 1.658±0.002	1.498±0.002
initial modulus of elasticity in the longitudinal direction (MPa) with σ_i (MPa):	BA	0.075±0.001	0.075±0.001
		0.150±0.004	0.170±0.020
		0.215±0.006	0.225±0.004
initial modulus of elasticity in the circumferential direction (MPa) with σ_i (MPa):	BA	0.088±0.011	0.077±0.001
		0.141±0.002	0.155±0.016
		0.200±0.003	0.210±0.004

L – longitudinal direction, C – circumferential direction,
 UA – uniaxial loading, BA – biaxial loading.

It is evident from the Table that the tensile strength, the limit stress intensity and the elongation decrease with age for both types of experiments. The moduli of elasticity, on the other hand, increase with age. The thickness of the aorta wall also increases with age, from $1.59 \pm 0.03\text{mm}$ for the age group A to $2.27 \pm 0.10\text{mm}$ for the age group B. The authors attributed this increase to the influence of atherosclerosis. Given in Table 2.9 is a representation of the individual components in the abdominal aorta wall for both age groups. The survey proved again the inadequacy of the collagen-elastin model if solely used for modelling the mechanical properties and their dependence on age.

Table 2.9 Individual components contained in the wall of the abdominal aorta in grams per 100 grams of dry tissue in dependence of age.

Age (years)	Elastin	Collagen	Thryosin	Hexozamin	Hexurone acids
30 – 39	19.32 ± 0.98	36.83 ± 5.78	3.25 ± 0.15	1.06 ± 0.04	0.46 ± 0.10
40 – 57	19.83 ± 1.53	30.78 ± 2.68	3.03 ± 0.03	1.13 ± 0.03	0.56 ± 0.10

Jartsev (1974) studied the mechanical properties of aorta for longitudinal direction together with their change in dependence on the distance from the heart. The experimental data presented in Table 2.10 indicates that the tensile strength of the vascular wall increases with the distance from the heart. The strength is the highest in the abdominal aorta. However, with age

the strength decreases by 2 to 2.5 times. The maximum relative extension is observed in the ascending aorta, to decrease by the arch and the thoracic aorta increase again in the abdominal aorta. With age, the strength decreases by 3 to 4 times. The largest decrease of the maximum elongation was observed in the 35 to 44 age groups and between 45 to 59 years. The above literature study demonstrates significant changes to proceed in the vessel wall with aging influencing their mechanical properties.

Table 2.10 Dependence of selected mechanical properties of aortic wall in longitudinal direction on age and sample localization.

Aorta	Age (years)	Limit force (N)	Maximum elongation (%)	Tensile strength (MPa)	Elongation (%) with force	
					10 N	20 N
ascending	15-19	80.4	184	1.06	57	80
	20-34	74.0	155	0.97	35	54
	35-44	97.8	142	0.87	31	52
	45-59	95.0	98	0.79	14	26
	60-74	92.9	56	0.65	10	16
	75-89	97.6	46	0.57	6	10
arch	15-19	60.4	145	1.66	45	62
	20-34	77.9	141	1.61	35	51
	35-44	73.2	140	1.32	33	51
	45-59	90.3	83	1.10	11	16
	60-74	87.8	58	0.89	9	14
	75-89	105.9	43	0.76	5	8
thoracic	15-19	73.6	130	2.22	41	56
	20-34	75.4	124	1.96	28	43
	35-44	77.3	121	1.34	25	42
	45-59	93.2	70	1.25	13	23
	60-74	83.3	50	0.95	9	15
	75-89	100.3	44	0.78	7	10
abdominal	15-19	59.1	165	2.36	55	80
	20-34	79.5	134	2.62	41	58
	35-44	83.5	124	1.99	30	51
	45-59	91.9	99	1.49	17	25
	60-74	94.4	61	1.24	8	12
	75-89	82.0	42	0.80	8	11

2.6.5 The effect of temperature and strain rate

The effect of temperature on vessels is manifested in two ways. First, thermo-receptors are excited and thermoregulation is accomplished by the nervous system, as an active reaction of the organism to a change in the external conditions. Secondly, the effect of temperature causes the mechanical properties of the individual components forming the vascular wall to change. The latter case is a passive reaction. Considering vascular wall

structure, it is evident that smooth muscle fibres are the only element able to respond actively to changes in temperature (Zatzman et. al., 1954). Green and Jackman (1979) studied the dependence of the modulus of elasticity of a dog's aorta on temperature. They found out that the modulus of elasticity decreases by 4% with the increase of temperature by 1°C. This property corresponds to visco-elastic material (Cohen et. al., 1976). It can be inferred from this that, with decreasing temperature the modulus of elasticity of aortic wall increases. Moreover, this increase of the modulus of elasticity reduces the wall deformability as confirmed also by Vilks et. al. 1975), where it was demonstrated that the vascular walls became brittle with low temperatures. Figure 2.8 shows the dependence (average values) of stress increments on temperature for the femoral artery for various values of pre-stress. The presented dependence was obtained by measuring isolated samples which were loaded at the defined pre-stress to be subsequently heated, while maintaining constant length, from 10°C to 45°C. Since samples from the femoral artery contract when heated, increments of the axial stress in dependence on temperature were measured. A similar dependence of axial stress increment on temperature is given in Figure 2.9 for two different age groups. The Figure shows the dependence to be linear in young men (15 years). In older persons (60 years), the dependence of stress increments on temperature is non-linear. The authors also studied the growth of femoral artery outer diameters in dependence on increasing temperature for different values of transmural pressure. They found out that, with pressures of 3.94×10^{-2} MPa and 0.9×10^{-2} MPa, the diameter grew by 2% and 3% respectively due to the increase of temperature to 45°C.

Figure 2.8 Dependence of stress increments on temperature for the femoral artery at different pre-stress values. a: 38.5kPa; b: 25.6kPa; c: 13.6kPa; d: 9.0kPa; e: 6.0kPa (a rectangular sample which was cut out of the wall and exposed to pre-stress). Adapted from Vilks et. al. (1975).

Figure 2.9 Dependence of stress increments on temperature for the femoral artery at pre-stress of 1.35MPa. Notations are a: 15 years; b: 60 years. Adapted from Vilks et. al. (1975).

Fung (1972) considered contractility with increasing temperature to be a common property of soft biological tissues. Vilks et. al. (1975) confirmed that samples from femoral artery manifest this dependence. On the other hand, samples from veins, tendons and muscles were found to extend with growing temperature. These different properties were explained by the change in elastin and collagen contents in the particular tissue.

Figure 2.10 presents the dependence of axial stress on strain for the aorta with the strain rate of $(1 \text{ to } 3.5)\text{s}^{-1}$ and 0.005s^{-1} . It is evident from this Figure that, with higher strain rate, the stress-strain dependence is deeper.

Figure 2.10 Stress-strain relationship for aorta with different strain rate. Note that strain rate = 1 to 3.5 s⁻¹ for the dashed line and 0.005 s⁻¹ for the solid line. Adapted from Collins and Hu (1972).

2.6.6 The Effect of Atherosclerosis

A number of authors studied the effect of atherosclerosis on mechanical properties of vessels. Loshtchilov and Savrasov (1977) observed the effect of atherosclerosis on the mechanical properties of the human abdominal aorta. The results for two age groups [A – (33 to 55) years and B – (56 to 77) years] are recorded in Table 2.11. The first column (I) presents the mechanical properties of the affected layer which was cut off from the wall. The second column (II) presents the mechanical properties of the rest of the wall. The experiments were carried out for the longitudinal section.

Table 2.11 Some mechanical properties of the wall of human abdominal aorta (Adapted from Loshtchilov and Savrasov (1977).

	Group A		Group B	
	I	II	I	II
Initial thickness (mm)	0.467±0.124	1.335±0.110	0.580±0.120	1.521±0.156
Tensile strength (MPa)	0.955±0.117	0.633±0.093	0.831±0.124	0.497±0.089
Maximum strain	0.380±0.094	0.661±0.989	0.279±0.025	0.450±0.079
Tangent modulus of elasticity (MPa)	2.771±0.612	1.028±0.186	3.069±0.558	1.251±0.198

It is evident from the results that the thickness of both the affected layer and the rest of the wall increase with age. The tensile strength (at the equipment jaws displacement rate of 24mm/min) is higher with the layer affected by atherosclerosis and it decreases with age. The tangent modulus of elasticity is also higher in the affected layer but, in this case, it grows with age. Purinya

and Kasyanov (1975) performed a similar analysis by studying 19 samples of the abdominal aorta in biaxial experiments (at the speed of loading of 2N/min) for two age groups A – from 43 to 45 years old and B – from 51 to 61 years old. The results are shown in Table 2.12. The parameters for the wall affected by atherosclerosis are presented in column I, column II showing the parameters for the wall from which the affected layer was cut off at the level of elastic membrane. This intervention is also used, in some cases, in clinical practice. It is evident from the findings that the thickness of the aorta, as well as that of the non-affected layer, grew with age. However, it is interesting to note that, on the contrary, the thickness of the removed layer decreased with age from 0.46mm to 0.38mm (on the average). After removing the sclerotic layer, the maximum stress intensity σ_i^* obtained as

$$\sigma_i^* = \sqrt{\sigma_1^2 + \sigma_2^2 - \sigma_1 \sigma_2} \quad (2.1)$$

increases. The relevant tensile strengths are substituted for circumferential σ_1 and longitudinal σ_2 stresses. The quantity σ_i is only of informative nature without any physiological interpretation. The effect of re-section of the affected layer on the maximum strain in both the longitudinal and circumferential directions has not actually been classified. Interestingly, this intervention results in no change of the secant modulus of elasticity.

Table 2.12 The influence of atherosclerosis on some mechanical properties of abdominal aorta wall. Adapted from Purinya and Kasyanov (1975).

	A		B	
	I	II	I	II
initial thickness (mm)	2.20±0.19	1.74±0.28	2.31±0.33	1.93±0.17
maximum stress intensity σ_i^* (MPa)	0.74±0.10	0.91±0.14	0.59±0.14	0.86±0.28
limit stretch ratio in longitudinal direction	1.329 ±0.041	1.404 ±0.066	1.346 ±0.131	1.310 ±0.048
limit stretch ratio in circumferential direction	1.570 ±0.052	1.502 ±0.114	1.375 ±0.022	1.468 ±0.053
secant modulus of elasticity E_{s1} (MPa) with σ_i (MPa)				
0.1	0.072 ±0.008	0.085 ±0.005	0.081 ±0.005	0.084 ±0.002
0.2	0.162 ±0.003	0.160 ±0.002	0.158 ±0.001	0.162 ±0.006
0.3	0.232 ±0.003	0.232 ±0.003	0.240 ±0.006	0.240 ±0.011
secant modulus of elasticity E_{s2} (MPa) with σ_i (MPa)				
0.1	0.077 ±0.002	0.079 ±0.004	0.082 ±0.004	0.077 ±0.002
0.2	0.130 ±0.007	0.147 ±0.009	0.155 ±0.006	0.148 ±0.003
0.3	0.201 ±0.007	0.211 ±0.012	0.226 ±0.007	0.216 ±0.005

Figure 2.11 shows the dependence of stress intensity on the stretch ratio λ for the abdominal aorta wall and for the vessel wall, from which the affected layer was removed at the inside elastic membrane level. The dependencies for both the longitudinal and circumferential directions are shown. Apparently, the removal of the layer has no marked effect in the circumferential direction. In the longitudinal direction, the stress/stretch ratio dependence after removing the insider layer becomes steeper.

Figure 2.11 Dependence of stress intensity σ_i on stretch ratio for the abdominal aorta in the longitudinal (a) and circumferential (b) directions. The accents (a', b') mark the state when internal part of the wall was removed at the level of elastic membrane (Adapted from Vossoughi et. al. (1985)).

By comparing the two works mentioned above, contradictory experimental findings are obtained in some cases by examining the different layers separately. Loshtchilov and Savrasov found a higher modulus of elasticity to apply for the layer affected by atherosclerosis, than the case is for the non-affected layer. Purinya and Kasyanov, on the other hand, found no substantial difference in the moduli of elasticity for the intact wall, and for that from which the inside layer was removed (the same was found to apply for both the tangent and secant moduli). It is hence probable that with the said intervention the re-section of the affected layer at the inside elastic membrane level) all the affected area failed to be removed. The tissues apparently require additional consideration, especially in relation to passage of pulsatile waves.

2.6.7 The Effect of Body Weight

Since most of the experiments may not be carried out with man, different experimental animals are used. The results so obtained are extrapolated to man. A number of animals have been used for the purpose and the experimental data tend to differ considerably. This is why the consideration of the experiment must always be defined exactly, so that procedures can be found by comparing the different results. The body weight of the experimental animal or person is considered by the authors to be one of the very important quantities to be determined exactly. In the following, the dependencies of selected mechanical quantities on body weight will be shown.

Vossoughi et. al. (1985) carried out experiments in the longitudinal and circumferential directions with 222 samples from the aorta of 7 pigs, 8 rabbits and 13 rats. Some of the results so obtained are given in Figure 2.12.

Figure 2.12 The dependence of the secant modulus of elasticity on strain for various kinds of animals. The full line marks the longitudinal direction whereas the dashed line marks the circumferential direction (Adapted from Vossoughi et. al. (1985)).

Several conclusions can be made from the Figure.

- (i) The modulus of elasticity as obtained from samples from the pig's aorta is higher both in the longitudinal and circumferential directions than with a rabbit and a rat;
- (ii) The moduli of elasticity of the rabbit and rat aorta are approximately the same;

- (iii) The stress-strain relationship is steeper with the pig's aorta for the circumferential strain than for longitudinal direction. The curve shows a hardening characteristics as the case is for similar dependencies obtained in man;
- (iv) In rabbit and rat, the stress for the same levels of strain was found to be higher for the circumferential direction, the dependence being mostly linear.

Weizsacker et. al. (1985) investigated the wall thicknesses, aorta diameters and circumferential stresses under the physiological pressure for various experimental animals and man.

The stress was determined from the elementary relation $\sigma = pr/h$ (where p is the internal pressure, r is the mean radius of the aorta and h is the wall thickness). The authors claim this dependence to be a constant. A relatively simple equation, this so-called Huxley's equation, was proposed to bind the aorta radius (r), wall thickness (h), tensile strength (R) in the circumferential direction and body weight.

$$y \approx am^b \quad y \equiv r, h, R \quad (2.2)$$

where m is the body weight and a , b are constants to be determined by experiment. These constants are given for information in Table 2.13 for the thoracic and abdominal aortas. The constants a and b from the Table served for determining the theoretical dependence of the radius r , wall thickness h and circumferential stresses on body weight. Figure 2.13 shows the dependence of abdominal aorta circumferential stress on body weight.

Table 2.13 The constants a , b for determining radius r , wall thickness h and tensile strength R_m in circumferential direction for abdominal and thoracic aortas. Adapted from Weizsacker et. al. (1985).

	abdominal aorta		thoracic aorta	
	a	b	a	b
$2r$ (m)	$1.66 \cdot 10^{-2}$	0.318	$1.93 \cdot 10^{-2}$	0.353
h (m)	$1.25 \cdot 10^{-3}$	0.344	$1.60 \cdot 10^{-3}$	0.396
R_m (Pa)	$1.79 \cdot 10^6$	-0.027	$1.87 \cdot 10^6$	-0.051
m (kg) average	0.975		0.920

Figure 2.13 The dependence of the abdominal aorta circumferential stress on body weight with average physiological transmural pressure. 1 – horse; 2 – bull; 3 – pig; 4 – man; 5 – sheep; 6 – dog; 7 – rabbit; 8 – cat; 9 – rat; 10 – mouse (Weizsacker et. al., 1985)

Figure 2.14 shows another stress-strain relationship for the coronary artery in the longitudinal direction in dependence on age as obtained from experimental data, published by Yoshimatsu (1958). These data are used to determine the individual constants in the stochastic model. In this model the biosystem life span was included. Since the initial stress equals zero, the Equation is of the following form:

$$\varepsilon_s = \frac{T_R}{E} \exp(aT_R^n) \quad (2.3)$$

where

$$\begin{aligned} E &= a_1 + a_2 \tanh\left(\frac{t - a_3}{a_4}\right) \\ a &= a_5 + a_6 \tanh\left(\frac{t - a_7}{a_8}\right) \\ n &= 0.18 \end{aligned} \quad (2.4)$$

The constants a_1 through a_8 have the following values:

a_1	2.51×10^{-4} MPa
a_2	1.46×10^{-4} MPa
a_3	63.46 years
a_4	13.62 years
a_5	$- 4.53 \text{ MPa}^{-n}$
a_6	0.31 MPa^{-n}
a_7	56.90 years
a_8	10.91 years

t is the biosystem lifespan in years. The points shown in Figure 2.14 indicate the theoretical values, calculated according to this model.

Figure 2.14 The tensile stress-strain relationship for the longitudinal direction of vena cava inferior (solid line). The isolated points indicate the theoretical value. Published by Yoshimatsu (1958)

Figure 2.15 shows the theoretical dependence of the coronary artery modulus of elasticity on age (longitudinal direction). This dependence may be divided into three basic sections. In the first section, approximately up to 30 years of age, the value of the modulus of elasticity is almost independent of age and it is also the lowest. Between 30 and 90 years of age, a steep increase occurs, the rate of change observed around 60 years being the highest. Over 90, the modulus is again almost independent of age and it is the highest. Since the stochastic model fails to include the period of growth, the above dependence does not correspond in reality in this area. As shown above, the arteries lose elasticity with growing age and their modulus of elasticity increases markedly.

Figure 2.15 The theoretical dependence of the modulus of elasticity of the coronary artery on age (longitudinal direction).

2.6.8 The effect of rheological properties of blood

The effect of rheological properties of blood and the resulting mechanical stresses in inducing thrombosis and atherosclerosis has been the subject of many investigations. Dintenfass (1964) has shown that parameters such as blood viscosity, velocity gradient, turbulence, smoothness and roughness of the endothelial of the blood vessels and viscoelasticity of the vessel wall are relevant factors to the stability of circulation and the formation of thrombosis and atherosclerosis. Stein and Sabbah (1974) induced turbulence through an atriovenous shunt in dogs and showed that a linear relationship existed between the intensity of turbulence and the weight of the resulting thrombi whereas in a laminar shunt, the thrombi were significantly less. They suggest that the mechanism of thrombus formation by turbulence is related to the effects of turbulence on the formed elements in blood. Such effects can include shear stress, collision of formed elements with the tubing and prolonged contact with foreign surfaces.

Stein and Sabbah (1976) made point velocity measurements in the ascending human aorta with normal and diseased aortic valves. They showed that the blood flow was highly disturbed during peak ejection in subjects with normal aortic valves whereas in patients with diseased valves, the flow was turbulent during most period of ejection. Stein and Sabbah (1980) discussed the effect of turbulent blood flow in human circulation. They suggested that disturbed or turbulent flow occurs in the human circulation in normal and diseased states. Under normal physiological conditions, they suggest that turbulence is present in the vicinity of the cardiac valves. In diseased states, turbulent flow may be present also in peripheral vessels distal to obstructions. They suggest that turbulent flow may be detrimental to erythrocytes and platelets and may play a role in causing poststenotic dilatations, intimal injury as well as atherosclerosis. Stein et. al. (1970) also demonstrated that turbulent flow may augment the process of sickling of red blood cells.

Rapid advancements in technology have resulted in the emergence of extracorporeal circulatory assist devices such as artificial heart valves, arterial grafts and total artificial heart devices. In such devices, the blood comes into

contact with foreign surfaces and the effect of mechanical stresses developed during the laminar or turbulent flow through such devices in the presence of foreign surfaces has been of interest to several investigators. Such studies have undertaken to investigate the effect of shear stresses or bulk turbulent stresses on the formed elements in blood such as the red blood cells or platelets.

Blackshear et. al. (1966) have shown that blood can withstand higher shear stresses with hemolyzing than those to be expected in extracorporeal circulation and hence the hemolysis that occur in tube flow is related to the interaction of the red blood cells with the tube wall. Nevarail et. al. (1969) subjected human, bovine and rabbit blood to high pressures without shear as well as to high shear flows. Their results showed that red blood cells subjected to high rates of changes of pressure of up to 165×105 mmHg/s for periods of up to 1 hour, did not reveal significant hemolysis and there was also no observed changes in the morphology of the cells. On the other hand, when blood was subjected to a shearing flow in a concentric cylinder viscometer, significant hemolysis was observed with shear stresses above 3000dynes/cm². Moreover, they also demonstrated a decrease in red blood cell survival after being subjected to high shear stresses. Shapiro and Williams (1970) indicated that laminar shear flows of up to 600dynes/cm² were not the cause for red blood cell hemolysis and they also suggested the importance of interaction between the cells and a foreign surface.

Sutera et. al. (1972) investigated the shear-induced hemolysis in laminar and turbulent flows and their results showed that hemolysis and subhemolytic damage are found to be dominated by the surface interaction. Blackshear (1972) has discussed the in-vivo and in-vitro tests on blood changes due to prosthetic valves and has discussed the mechanical factors in flow that induce hemolysis. Sutra and Mehrjardi (1975) subjected gluteraldehyde fixed human red blood cells to turbulent shear flow and observed that at shear stresses above 2500dynes/cm², fragmentation of the cells occur with the rupture occurring in tension in the bulk flow. Brown et. al. (1975) investigated the

response of human platelets subjected to shear stress. Their results indicated that platelets are sensitive to shear stresses in the same order of magnitude found in the normal circulation. With magnitudes of shear stress of about 50 to 100 dynes/cm², morphological, biochemical and functional changes in the platelets were observed which could result in thrombus formation.

Hung et. al. (1976) also showed that the threshold for lysis of platelets is of the order of about 100 dynes/cm². With increase in exposure time to the shear stresses, the threshold stresses for the destruction of elements are lower. At lower shear stresses, the interaction between the formed elements in blood and the surface dominates.

2.7 Homogeneity of the Vessel Wall

On visual inspection, blood vessels appear to be fairly homogeneous and distinct from surrounding connective tissue. The non-homogeneity of the vascular wall is realized when one examines the tissue under a low-power microscope, where one can easily identify two distinct structures: the media and adventitia.

For this reason the assumption of vessel wall homogeneity is applied cautiously. Such an assumption may be valid only within distinct macroscopic structures. However, few investigators have incorporated macroscopic non-homogeneity into studies of vascular mechanics (Von Maltzahn et. al., 1981).

2.8 Incompressibility of the Vessel Wall

Experimental measurement of wall compressibility of 0.06% at 270 cm of H₂O indicates that the vessel can be considered incompressible when subjected to physiologic pressure and load (Carew et. al. 1968). In terms of the mechanical

behaviour of blood vessels, this is small relative to the large magnitude of the distortional strains that occur when blood vessels are deformed under the same conditions. Therefore, vascular compressibility may be important to understanding other physiologic processes related to blood vessels, such as the transport of interstitial fluid.

2.9 Inelasticity of the Vessel Wall

The notion that blood vessel walls exhibit inelastic behaviour such as length-tension and pressure-diameter hysteresis, stress relaxation, and creep has been reported extensively (Bergel, 1961; Fung et. al., 1979). However, blood vessels are able to maintain stability and contain the pressure and flow of blood under a variety of physiologic conditions.

These conditions are dynamic but slowly varying with a large static component.

2.10 Residual Stress and Strain

Blood vessels are known to retract both longitudinally and circumferentially. This retraction is caused by the relief of distending forces resulting from internal pressure and longitudinal tractions. The magnitude of retraction is influenced by several factors. Among these factors are growth, aging, and hypertension. Circumferential retraction of medium-caliber blood vessels, such as the carotid, iliac, and bracheal arteries, can exceed 70% following reduction of internal blood pressure to zero. In the case of the carotid artery, the amount of longitudinal retraction tends to increase during growth and to decrease in subsequent aging (Dobrin, 1978). It would seem reasonable to assume that blood vessels are in a nearly stress-free state when they are fully retracted and free of external loads. This configuration also seems to be a

reasonable choice for the reference configuration. However, this ignores residual stress and strain effects that have been the subject of current research (Chuong and Fung, 1989; Vaishnav and Vassoughi, 1983).

Blood vessels are formed in a dynamic environment which gives rise to imbalances between the forces that tend to extend the diameter and length and the internal forces that tend to resist the extension. This imbalance is thought to stimulate the growth of elastin and collagen and to effectively reduce the stresses in the underlying tissue. Under these conditions it is not surprising that a residual stress state exists when the vessel is fully retracted and free of external tractions. This process has been called remodelling (Fung et. al., 1993). Striking evidence of this remodelling is found when a cylindrical slice of the fully retracted blood vessel is cut longitudinally through the wall. The cylinder springs open, releasing bending stresses kept in balance by the cylindrical geometry (Vaishnav and Vassoughi, 1983).

2.11 Current Coronary Artery Bypass Grafting Techniques

Treatments are available for patients diagnosed with Coronary Artery Disease. Today's standards of care are typically broken down into three categories:

- Medical Therapy, including pharmacological (drug) treatment
- Percutaneous transluminal coronary angioplasty, or PTCA
- Surgical intervention, including CABG or Coronary Artery Bypass Graft surgery

Medical Therapy

Typically the first line treatment for coronary artery disease is medical therapy. Medical therapy involves mostly pharmacological treatment strategies using drug's including vasodilators, beta-blockers and calcium channel blockers. Medical therapy is primarily palliative as its main goal is the reduction of the

symptoms not necessarily fixing the underlining problem causing the ischemic conditions.

Percutaneous Transluminal Coronary Angioplasty (PTCA)

Patients who are not candidates for medical therapy or not responding to medical therapy are usually referred to less invasive treatment such as PTCA or more commonly referred to as "balloon angioplasty". In these cases the patients generally receive only a local anesthetic and/or sedatives. The procedure involves accessing the patient's coronary arteries through the femoral artery via a small incision near the patients groin area. The physician will then introduce a guiding catheter to the targeted coronary artery and attempt to open the narrowing in the vessel using a number of devices including:

Balloon Angioplasty

In balloon angioplasty, a tiny balloon is introduced into the coronary artery and then is inflated and stretches the artery to allow for more blood flow through the blockage. This procedure is used to compress obstructing plaque in a clogged artery against the arterial wall so that blood can flow freely again. In a balloon angioplasty procedure, the doctor, with the help of a guide wire and a guide catheter, inserts a thin, flexible balloon angioplasty catheter into the patient's arm or leg and threads it through the blood vessels. The doctor positions the balloon of the angioplasty catheter at the site of blockage and gently expands it to compress the plaque and create a wider opening in the artery. The doctor may need to inflate the balloon several times to achieve an adequate opening. Sometimes, depending on the size of the obstruction, successively larger balloons are needed. During balloon angioplasty, the doctor monitors the progress of the procedure on a special X-ray machine called a fluoroscope. When the artery appears clear, the doctor removes the catheters and guidewire.

Figure 2.16 Balloon Angioplasty. Figure extracted from <http://heartdisease.about.com/library/weekly/aa122897.htm>

Coronary Stents

A coronary stent is a tiny expandable mesh tube made of medical grade stainless steel. A stent is delivered on a balloon catheter and implanted in the coronary artery after balloon angioplasty to help keep the artery open. After the plaque is compressed against the arterial wall, the stent is fully expanded into position, thereby acting as a miniature “scaffolding” for the artery.

Unlike balloon angioplasty, in which the balloon is removed after the procedure, coronary stents are permanent implants, which are left behind the patients’ artery. While stents have generally improved patient outcomes they too are susceptible to narrowing over time, a process called in-stent restenosis.

Figure 2.17 Insertion of coronary stent into the artery. Figure extracted from <http://heartdisease.about.com/library/weekly/aa122897.htm>

Atherectomy

Atherectomy is a procedure to remove plaque from arteries. It uses a laser catheter, or a rotating shaver ("burr" device on the end of a catheter), to remove plaque from the arteries supplying blood to the heart muscle. The catheter is inserted into the body and advanced through an artery to the area of narrowing. Other devices are dissectional catheterectomy, catheters that shave off the plaque, or laser catheters that vaporize the plaque. Balloon angioplasty or stenting may be used after an atherectomy.

In some cases where the artery is more severely diseased or calcified the physician may deploy an atherectomy device, such as a laser, that will ablate or remove the diseased tissue from the inside of the artery. The physician may decide to place a stent or use a balloon to increase the vessel diameter and further improve blood flow.

Figure 2.18 Removal of plaque from the arteries. Figure extracted from <http://heartdisease.about.com/library/weekly/aa122897.htm>

Surgical Intervention

The next and most invasive of treatment strategies for more severe coronary artery disease is surgical intervention. The most common surgical procedure is Coronary Artery Bypass Graft or CABG.

Coronary Artery Bypass Grafting (CABG)

Coronary Artery Bypass Grafting (CABG) is a surgical procedure that restores blood flow to the heart beyond a blockage in a coronary artery. It is an open-heart surgery in which the rib cage is opened and a section of a blood vessel is grafted from the aorta to the coronary artery to bypass the blocked section of the coronary artery.

Coronary artery bypass graft surgery builds a detour around one or more blocked coronary arteries with a graft from a healthy vein or artery. The graft goes around the clogged artery (or arteries) to create new pathways for oxygen-rich blood to flow to the heart.

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Coronary artery bypass graft surgery is widely performed in the United States. The American Heart Association estimates that 573,000 coronary artery bypass graft surgeries were performed on 363,000 patients in 1995. Seventy four percent of these procedures were performed on men and 44% on men and women under the age of 65 (1995 data). The estimated average cost of this procedure in 1995 was \$44,820.

Figure 2.19 Coronary Bypass Using suitable arteries and veins. Figure extracted from <http://www.howstuffworks.com>

Conduits Used in CABG

Grafts are created by using portions of another artery or vein from the patient's body. The most commonly used vessels are the internal mammary artery, which is inside the chest wall, or the greater saphenous vein, which is in the leg. The surgeon may choose to use other conduits. Vein grafts are typically connected to the aorta and then attached beyond the blockage to re-establish blood flow.

▪ **Saphenous Vein Grafts**

The first CABG operation was performed in 1962 at Johns Hopkins, Baltimore, by David C. Sabiston Jr. He used a segment of vein from the leg (called the long saphenous vein) of the patient to fashion a pathway between the aorta and diseased coronary arteries. This is one of the commonly used conduits even today.

Its main drawback is the possibility of atherosclerotic plaque-formation in the vein conduit itself over a period of five to ten years, leading to graft occlusion. This has similar consequences to the original disease.

- **Arterial Grafts**

The arterial grafts used include the Internal Thoracic Artery (ITA) or Internal Mammary Artery (IMA). A little known Russian, Kolessof, performed the first CABG operation using another artery as the conduit.

On the inside of the chest wall is a large arterial branch called the Internal Thoracic Artery (ITA). This artery is more or less similar in size to a coronary artery, and by its close proximity to the coronaries, is easy to use as a bypass graft to diseased arteries. It wasn't until much later that the significance of using ITAs for grafting was understood. After 10 years, 95 percent of these conduits remained free of plaque and repeat obstruction, and this instantly made it the preferred conduit over saphenous vein. Today, this is the conduit of choice to perform bypass surgery, and significantly improves long term survival after performance of this operation.

- **Other Arteries that may be useful as conduits**

When the advantages of using arteries to bypass the coronaries were recognized, many different surgeons used their imagination to expand the choice of arteries to use as conduits. Today, the possible targets are:

- Gastroepiploic artery - which supplies the stomach and intestines
- Radial artery - which is the artery that nourishes the forearm and hand
- Inferior Epigastric Artery - which runs in the wall of the lower abdomen
- Subscapular artery - on the back of the chest
- Splenic artery - supplies the spleen

- Intercostal artery - which runs just inside the rib cage

Of these, only the radial artery and gastro-epiploic arteries are commonly used. Though none has been proven better than ITAs, expectations are that they will be at least as good, and that the benefits of a bypass operation will then be more long lasting.

CABG Techniques

The following are the techniques of CABG that are available:

(i) Conventional CABG Surgery (C-CABG):

This requires that a patient's heart be stopped during the surgical process. A cardiopulmonary bypass (heart-lung) machine supports blood flow throughout the body.

A conventional CABG procedure is typically performed through an incision (called a median sternotomy) in the middle of the chest. Some surgeons may prefer to perform a smaller mini-sternotomy. The patient's heart is stopped, or "arrested" and she or he is placed on circulatory support. The circulatory support system (called cardiopulmonary bypass, or CPB for short) works in place of the patient's heart and lungs, providing blood flow to the whole body. As blood enters the CPB system, it exchanges carbon dioxide for oxygen (just as the lungs would), with the tubing functioning as the patient's veins and arteries. The blood is filtered, and cooled or heated to keep the patient at a proper temperature.

CPB has been recognized as an important medical invention — it has allowed surgeons to safely operate on the heart. However, CPB may have some negative effects on some patients' organs and tissues.

(ii) Off-Pump CABG

The surgeon opens the chest by a vertical midline incision splitting the breast bone (sternum). Some surgeons place sutures in preparation for connection to the heart-lung machine, to allow immediate establishment of an artificial circulation in case of accidents during surgery. A few others do not make any such preparation in advance.

CABG is performed to the diseased coronary arteries in the conventional manner, using either vein or artery (ITA) grafts. To make the process easier, the heart may be transiently slowed down using drugs like Esmolol or Adenosine. The surgeon takes care to avoid excessive manipulation or compression of the heart during the operation. Most diseased coronary arteries can be safely accessed and grafted using this method.

The off-pump CABG is similar to conventional CABG. The difference lies in the fact that the patient is *not* hooked on to the heart-lung machine. The absence of the heart-lung machine means that:

- the heart-beat cannot be stopped
- the aorta cannot be clamped
- cardioplegia cannot be injected
- surgical connection of the graft to coronary artery is slightly more difficult, since the heart is constantly moving

The harmful effects of using a heart-lung machine are avoided by off-pump CABG. If there is one major drawback to using the heart-lung machine, it is this - the artificial circulation causes injury to the blood cells and all organ systems of the body. The damage occurs because

- the "non-biological" surface of the tubes that lead from the patient to the machine can injure blood cells
- blood circulating inside these tubes is subject to considerable external stress forces
- air, plastic particles and tiny blood clots might get mixed in the blood stream and cause damage to different organs

The effect on the patient is a "whole body inflammation" response that can be minimized but not totally eliminated by current perfusion techniques.

(ii) Beating Heart CABG Surgery (BH-CABG):

Recent advances in surgical technique and equipment allow the surgeon to perform CABG surgery while the heart continues to beat, thus the name "Beating Heart CABG." In the great majority of cases, this technique avoids placing the patient on a cardiopulmonary bypass system used in conventional CABG procedures.

Recent clinical studies suggest that there may be benefits to beating heart surgery, such as:

- Less blood trauma
- Decreased risk of adverse events (stroke, etc.)
- A more rapid return to normal activities

One of the greatest challenges in beating heart CABG surgery is the difficulty of suturing on a beating heart. The surgeon must use a stabilisation system to steady the portion of the heart where the suturing takes place, so that the surgeon can perform the delicate cutting and sewing needed during bypass surgery.

Figure 2.20 One example of the Stabilisation System – The Medtronic Octopus®. This device limits the motion of only a small area of the heart while the rest of the heart continues to beat normally. This allows the surgeon to perform the CABG surgery without having to completely stop the heart and place the patient on cardiopulmonary bypass (heart-lung machine). Figure extracted from:
http://www.medtronic.com/cardsurgery/products/octo_pos.html

Complications following CABG

Under the best circumstances, bypass surgery carries about a 1% operative mortality rate and the average is 3%. Complications include the following:

- **Atrial Fibrillation.**

Atrial fibrillation (very fast and irregular heart beats) occurs after CABG.

- **Blood Clots.**

Blood clots may form in the new graft, closing it up or narrowing the treated vessel over time. Therapy with aspirin and other anti-clotting drugs help keep the graft open and working properly. The anti-platelet agent clopidogrel is proving to be particularly effective. For long-term prevention of closure as well as slowing progression of

atherosclerosis, aggressive use of cholesterol-lowering drugs may be more beneficial than the standard anti-clotting drugs.

- **Decline of Mental Function**

There were studies reporting a decline in mental function five years after bypass surgery. It is not known, however, if patients with bypass procedures tend to have other higher risk factors for mental decline (being older or sicker than those who choose angioplasty). Long-term studies are underway.

Minimally Invasive Bypass

Minimally invasive bypass (also called buttonhole or keyhole bypass) surgeries are exciting advances in basic bypass surgery that are currently being tested with good success for patients with disease in single vessels.

One uses a four-inch incision, and the surgeon works on the front of the heart while it is beating slowly. It is used just for bypasses with one or two arteries.

In another, the heart is stopped, and the patient is put on a machine that reroutes the blood through a device that keeps it oxygenated. Fiberoptic scopes and instruments are passed through a number of finger-sized incisions and the surgeon works on all sides of the heart guided by a video image from a tiny camera inserted through a four-inch incision.

Some advanced heart centres now employ robotic systems, which allow the surgeon to perform extremely delicate manoeuvres on tiny vessels through pencil-size incisions. They are not yet used for the whole bypass process.

Early results show that minimally invasive bypass procedures will be less expensive, require a shorter hospital stay, and be a significant

improvement over conventional coronary artery bypass surgery. To date, they are performed only in a few medical centers for select candidates.

Table 2.14 Comparison of Advantages and Disadvantages of the Various Surgical Approaches.

Approach	Advantages	Disadvantages
Conventional CABG	Excellent, proven results. Not as technically demanding as other methods; no need for cardiac surgeon with specialty experience. Usable for multiple bypasses.	Large incision may lead to more pain, longer recovery. CPB ¹ leads to higher complication rate, longer recovery. More stress on heart. May be too stressful for some patients.
Off-Pump CABG	Less technically demanding than MIDCAB. Like conventional CABG, usable for multiple bypass. Avoids the complications and longer recovery of CPB.	Large incision may result in more pain, longer recovery. Technique is more difficult than conventional CABG, requiring special expertise.
Minimally Invasive Direct Coronary Artery Bypass (MIDCAB)	Avoids the pain and longer recovery caused by long incision. Avoids the complications and longer recovery of CPB. Surgery is a short procedure (90 minutes). Short hospital stay (48 hours).	Small incision requires high degree of technical skill. Surgeon can operate only on limited parts of the heart (the anterolateral artery).

CHAPTER 3 THEORY: CARDIOVASCULAR SYSTEM

3.1 The Cardiovascular System

The cardiovascular system includes the heart and blood vessels of the systemic and pulmonary circulation. Two fundamental requirements of the circulatory system are to provide circulation to the body without interruption and to adjust blood flow according to the various demands of the body (Rushmer, 1976). The energy required to maintain the blood flow through the vessels is supplied to the contracting heart. The pressure gradient developed between the arterial and the venous end of the circulation is the driving force causing blood flow. The energy is dissipated in the form of heat due to the frictional resistance due to blood flow in the vessels.

3.1.1 The Heart

The heart consists of four chambers whose function is to pump blood throughout the body. A schematic view of the heart is shown in Figure 3.1. The heart is a hollow, cone-shaped muscle located between the lungs and behind the sternum (breastbone). Two-thirds of the heart is located to the left of the midline of the body and 1/3 is to the right.

Figure 3.1 Schematic view of the heart.

[Note that Medical illustrations assume that the patient is facing you so that the right and left correspond to the patient's right and left. That's why the left and right labels here seem backwards.]. Figure extracted from <http://www.howstuffworks.com>

The apex (pointed end) points down and to the left. It is 5 inches (12 cm) long, 3.5 inches (8-9 cm) wide and 2.5 inches (6 cm) from front to back, and is roughly the size of your fist. The average weight of the heart in a female is 9 ounces and in the male is 10.5 ounces. The heart comprises less than 0.5% of the total body weight.

The heart has 3 layers. The smooth inside lining of the heart is called the endocardium. The middle layer of heart muscle is called the myocardium. It is surrounded by a fluid filled sac called the pericardium.

The heart is divided into 4 chambers (see Figure 3.2), namely:

1. Right Atrium (RA)
2. Right Ventricle (RV)
3. Left Atrium (LA)
4. Left Ventricle (LV)

Each chamber has a sort of one-way valve at its exit that prevents blood from flowing backwards. When each chamber contracts the valve at its exit opens. When it is finished contracting the valve closes so that blood does not flow backwards. The main valves are:

5. Tricuspid valve- is at the exit of the Right Atrium.

6. Pulmonary valve - is at the exit of the Right Ventricle.
7. Mitral valve - is at the exit of the Left atrium.
8. Aortic valve - is at the exit of the Left Ventricle.

Figure 3.2 Chambers of the Heart. Figure extracted from <http://www.howstuffworks.com>

When the heart muscle contracts or beats (called systole) it pumps blood out of the heart. The heart contracts in two stages. In the first stage the Right and Left Atria contract at the same time, pumping blood to the Right and Left Ventricles. Then, the Ventricles contract together to propel blood out of the heart. The heart muscle relaxes (called diastole) before the next heartbeat. This allows blood to fill up the heart again.

The right and left sides of the heart have separate functions. The right side of the heart collects oxygen-poor blood from the body and pumps it to the lungs where it picks up oxygen and releases carbon dioxide. The left side of the heart then collects oxygen rich blood from the lungs and pumps it to the body so that the cells throughout your body have the oxygen they need to function properly.

Flow of Blood

All blood enters the right side of the heart through two veins:

1. The Superior Vena Cava (SVC) and
2. Inferior Vena Cava (IVC) (see Figure 3.3).

The SVC collects blood from the upper half of the body. The IVC collects blood from the lower half of the body. Blood leaves the SVC and the IVC and enters the Right Atrium (RA) (3).

When the RA contracts, the blood goes through the Tricuspid Valve (4) and into the Right Ventricle (RV) (5). When the RV contracts blood is pumped through the Pulmonary Valve (6), into the Pulmonary Artery (PA) (7) and into the lungs where it picks up oxygen.

Figure 3.3 Flow of blood. Figure extracted from <http://www.howstuffworks.com>

The flow of blood occurs this way because blood returning from the body is relatively poor in oxygen. It needs to be full of oxygen before being returned to the body. So the right side of the heart pumps blood to the lungs first to pick

up oxygen before going to the left side of the heart where it is returned to the body full of oxygen. Blood now returns to the heart from the lungs by way of the Pulmonary Veins (8) and goes into the Left Atrium (LA) (9). When the LA contracts, blood travels through the Mitral Valve (10) and into the Left Ventricle (LV) (11). The LV is a very important chamber that pumps blood through the Aortic Valve (12) and into the Aorta (13) to the rest of the body. The Aorta is the main artery of the body. It receives all the blood that the heart has pumped out and distributes it to the rest of the body. The LV has a thicker muscle than any other heart chamber because it must pump blood to the rest of the body against much higher pressure in the general circulation (blood pressure).

Heart failure may occur suddenly, or it may develop gradually. When heart function deteriorates over years, one or more conditions may exist. The strength of muscle contractions may be reduced, and the ability of the heart chambers to fill with blood may be limited by mechanical problems, resulting in less blood to pump out to tissues in the body. Conversely, the pumping chambers may enlarge and fill with too much blood when the heart muscle is not strong enough to pump out all the blood it receives. In addition, as the architecture of the heart changes as it enlarges, regurgitation of the mitral valve may develop, making the heart failure even worse. The arrows indicate the direction of blood flow through the heart. The heart consists of the right and left atria (atrium), and the right and left ventricles (ventriculus cordis). The right atrium and right ventricle are divided by the septum from the left atrium and the left ventricle.

3.1.2 Blood Circulation in the Body

Venous blood from the whole organism (with the exception of the lungs) enters, through veins, the right atrium and from there, through the right ostium, the right ventricle. From the right ventricle, the blood enters the pulmonary artery ramifying to the right and left lung. Here, the branches of

the pulmonary artery ramify down to the smallest vessels, the so-called capillaries. In the lungs, the exchange of gases takes place between the alveolar air and the blood, namely the decrease of carbon dioxide content in the blood and its oxidation. The oxidised blood returns, through four pulmonary veins to the left atrium. From there, the blood flows, through the left atrioventricular ostium to the left ventricle. From the left ventricle, the blood flows into the largest arterial tree, called the aorta. By its branches ramifying in the organism down to the capillary level, the blood is distributed throughout the body. The blood transfers oxygen to the tissues, nutrients metabolites and its mediates the transport of metabolism products to the kidneys and the transport of hormones to the cells. It participates also in the body temperature control by increase transfer of heat to the surroundings or by conserving the heat in the organism. After transferring oxygen to the tissues, the blood receives carbon dioxide from them, thereby becoming the so-called venous blood. The capillaries join up to form larger vessels (veins).

All the veins in the body converge into two large venous trees: the vane cava superior and vena cava inferior. Vena cave superior collects the blood from the area of the organs of the head and the neck, the upper limbs and from some areas of the trunk wall. Vena cave inferior brings the blood from the lower limbs, from the pelvic walls and the abdominal organs. Both the cavus veins bring the blood to the right atrium, where also the venous blood from the heart walls is brought. This circuit is called the total blood circulation. Hence the small blood circulation and the large system blood circulation are distinguished between.

In the vascular system, we distinguish the arteries, veins and the smallest vessels connecting them, the capillaries. By the inner diameter, we classify the arteries as the large arteries with the diameter of about 8mm or more, including the aorta (of diameter approximately 26mm), arteries of small and medium diameters (up to 3mm) and the arterioles (of diameter approximately 100 μ m). In the direction from aorta, both the blood flow pressure and the

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speed decrease because, due to the gradual ramification, the resulting inner diameter, given by the sum of these vessels individual diameters increases.

Please refer to Appendix I for detailed write-ups and formulations on the mechanics of blood vessels.

CHAPTER 4 METHOD

This Chapter describes the development and construction of the finite element models and the analyses that were conducted after obtaining a solution for each of the models constructed. The models that were developed and analysed can be categorised into two main groups, namely: (i) two-dimensional axisymmetric modelling of an end-to-end anastomosis; and (ii) three-dimensional modelling of a symmetric end-to-side anastomosis. Within each of these two categories, variations were further applied to the models. These are described in further details as follows.

4.1 Two-Dimensional Modelling of End-to-End Anastomoses

The assumptions adopted for all the two-dimensional finite element models constructed were:

1. Only two-dimensional, end-to-end anastomoses are considered.
2. Both the host artery and the graft are assumed to be perfectly cylindrical.
3. The models are assumed to be axisymmetric. This means that there is no radial variation in the loads and stresses experienced by the host and the graft vessels.
4. For all analyses, the host artery is assumed to have a Young's Modulus of 2MPa. The Young's Modulus for the graft is assumed to be 1.5MPa. The assumed values are based on approximations from literature (Please refer to Table 2.1 of the report).
5. The Poisson's ratio for the host artery is assumed to be 0.35 and that of the graft is assumed to be 0.3.
6. The properties of the vessels are assumed to be isotropic and linearly elastic.

The models constructed were further sub-divided into: (1) Anastomoses modelled with a region of overlap between the host artery and graft, and (2) Anastomoses without region of overlap.

4.1.1 Models with Region of Overlap

Figure 4.1 Schematic View of the model constructed (End-to-end anastomosis with overlap between host and graft).

Figure 4.1 above shows a schematic view of the end-to-end anastomotic models that were constructed and analysed. The methodology for performing the analyses was as follows:

Axisymmetric models of the host and graft were first constructed in ANSYS 5.4 with an overlap of 0.5mm in length between them. The model was meshed using Solid45 elements, with the corresponding material properties applied onto the host and the graft. Contact elements were added on the right

face of the host and the left face of the graft (see Figure 4.1) to detect contact between the two vessels in the solution phase of the programme.

From Figure 4.1, it can also be seen that a displacement boundary constraint of $U_x = 0$ was set for the ends of the host and graft, to simulate the two ends being held in-place by sutures. 100 sub-steps were set for the solution phase to solve this initial model to obtain the deformed “sutured” state. After it had been established that contact was made between the host and the graft vessels, the UPCOORD (Update coordinate) feature was applied to update the model with the resultant (deformed) coordinates. Following that, a pressure of 8,000 Pa (equivalent to 60mmHg) was applied to the inside of the vessels to simulate the return of blood pressure into the sutured region after the completion of the suturing process.

In the analyses, the dimensions and properties of the host vessel were fixed. The host vessel was assumed to have an external diameter of 2.5mm and a thickness of 0.3mm.

The external diameter of the graft vessel was fixed at 4.0mm, but its thickness was varied from 0.5mm to 0.7mm, in steps of 0.1mm, to analyse trends and make observations arising from the variation of graft thicknesses.

4.1.2 Models without region of overlap

The same methodology that was described in Section 4.1 above was also employed in the second phase of the two-dimensional finite element models of the end-to-end anastomosis. A schematic diagram of the model created was illustrated in Figure 4.2 below. From the Figure, the gap between the host and graft vessels represented the space between them as a result of their differences in diameters. The objective of this exercise was to determine the suture force needed to close the gap between the host and graft vessels.

When compared against the first phase of the study conducted, a few differences were noted:

1. There is no region of overlap between the lengths of the host and graft vessels.
2. Contact elements were added onto the lower edge of the host artery and the upper edge of the graft. The reason for this difference in location of the contact elements was that without a region of overlap, contact might be made on the ends of the host and graft instead of at the sides.

The suturing process was simulated by means of a force application in the Fx direction. A trial and error method was employed to ascertain the force needed to close the gap between the host and graft vessels to ensure that there was continuity in geometry in the resultant sutured model

The external diameter of the host vessel was assumed to be 2.5mm and its thickness 0.1mm. The graft was assumed to have an external diameter of 3mm and a thickness of 0.15mm.

A schematic view of the model constructed is illustrated in Figure 4.2 below:

Figure 4.2 Schematic View of the model constructed (End-to-end anastomosis without overlap between host and graft).

4.2 Three-Dimensional Modelling of End-to-Side Anastomoses

With the completion of the two-dimensional finite element models of an end-to-end anastomosis, the next phase of the Project focused on the development and analyses of three-dimensional finite element models for an end-to-side anastomosis. The results of end-to-side anastomotic models would be of greater significance to the cardiologist because most of the bypass operations carried out involved end-to-side anastomoses. Furthermore, the results and observations from these models would better complement the research work of the remaining team members.

The three-dimensional end-to-side models created can be categorised into two main groups. For ease of reference, they are referred to as (1) initial models created and (2) models with additional constraints applied. Within the group, variations were made in the angles at which the host and graft vessels meet. The angle variations considered were in accordance with those in the coronary artery bypass group, to complement their research work to design an interposition sleeve for joining the aorta to vein graft and vein graft to the coronary artery in a bypass operation.

For each of the finite element models created, stress analyses were performed along the lengths of the inner and outer walls for the host and the graft to study the effects (in terms of stresses, deformations) as a result of pressure application. Observations were made by considering the effects of varying the angles, and this were discussed in Chapter 6 of this report.

4.2.1 Initial Models

For three-dimensional modelling of end-to-side anastomoses, three finite element models of the comprising the host and graft were created. The angles at which the host and graft were subtended were varied at 30, 45 and 60 degrees, which were the same angle variations considered by the team members in the bypass group for the design of the interposition sleeve. Figure 4.3 below shows the 45-degree finite element model that was

developed. Since the model exhibits reflective symmetry and loading is symmetric, a one-half symmetry model is only required for the solution. As illustrated from the Figure, only half-cylinders – representing the host and graft vessels – were created as a result of the assumption of symmetric conditions.

The following assumptions were made in the development of the finite element models:

1. The host and graft vessels are assumed to be perfectly cylindrical in shape.
2. The models exhibit reflective symmetry and loading is symmetric – this allows only half the cylinders to be modelled.
3. The length of the host was assumed to be 30mm.
4. The Young's Modulus of the host is 1MPa, and its Poisson's ratio is 0.45.
5. The length of the graft was assumed to be 16mm.
6. The Young's Modulus of the graft is 1.5MPa, and its Poisson's ratio is 0.45.
7. The properties of host and graft vessels are assumed to be isotropic and linearly elastic.

The boundary conditions that were applied onto the model were:

1. $U_x=0$ along the lengthwise edges of both host and graft, to represent symmetric boundary conditions
2. $U_x=0$ and $U_y=0$ at the ends of the host and graft vessels
3. Pressure application of 8 KPa (equivalent to 60mmHg) to simulate the pressure experienced by the vessels with the return of blood flow and the pumping action of the heart.

The critical regions identified in the models were the region of interface between the host and graft vessels and their immediate vicinity. It was anticipated that greater distortions might be experienced in this region, as a result of differences in material properties between the host and graft. Hence, when the model was meshed, more degrees of freedom were placed in these

critical regions through the use of smaller elements. The entire model was meshed using SOLID45 elements. Areas away from the critical region were meshed with coarser element. This also allowed the model to be more optimally meshed, with regards to computer efficiency.

Identical boundary conditions were also applied to the 30-degree and 60-degree models that were developed.

Figure 4.3 Initial finite element model created for an end-to-side anastomosis.

After the models were developed and the boundary conditions applied, the finite element model was solved. The post-processing phase involved defining paths for both the host and graft and extracting data from these paths to obtain values of stresses and displacements.

Figure 4.4 shows the path that was defined for the host. Using the area of intersection between the host and graft as the origin, Direction 1 was defined as the downward direction (towards the reader) and Direction 2 the upward

direction (away from the reader). For each of these two directions, paths were defined for both the inner and outer walls of the host vessel.

Figure 4.4 Path Definitions for Host

A similar method of path definition was also adopted for the graft. Using the area of intersection between the host and graft as the origin and taking the isometric view of the graft as the basis, Direction 1 was defined as the downward direction of the left edge of the graft and Direction 2 was defined as the downward direction of the right edge of the graft. Paths were identified for both inner and outer walls of the graft and the relevant data were extracted for analyses.

Figure 4.5 Path Definitions for Graft.

4.2.2 Models with additional displacement constraints

The final phase of the three-dimensional finite element modelling of an end-to-side anastomosis, with additional conditions imposed. These conditions are incorporated after subsequent meetings and discussions with the heart surgeon – such as support to the artery from the muscle layer – are made.

Figure 4.6 Schematic diagram of artery with epicardium and muscle layer.

Figure 4.6 illustrates that the artery is covered by an outer layer known as the epicardium. The other side of the artery rests on the muscle layer. The thickness of the epicardium is about 1mm. Coronary artery bypass grafting is carried out by first making an incision on the epicardium, and then joining the artery to the graft.

The following assumptions were made in the development of the finite element models:

1. The host and graft vessels are assumed to be perfectly cylindrical in shape.
2. The models exhibit reflective symmetry and loading is symmetric – this allows only half the cylinders to be modelled.
3. The length of the host was assumed to be 30mm.
4. The Young's Modulus of the host is 1MPa, and its Poisson's ratio is 0.45.
5. The length of the graft was increased to 26mm. The graft length was extended so as to extract data along the length but ignoring the edge, as the displacement boundary conditions imposed on the edge of the graft could affect the values of the stresses and displacements extracted.
6. The Young's Modulus of the graft is 1.5MPa, and its Poisson's ratio is 0.45.
7. The properties of host and graft vessels are assumed to be isotropic and linearly elastic.
8. The entire model is meshed using SOLID45 elements.

The boundary conditions that were imposed for this set of finite element models were as follows:

1. $U_x=0$ along the lengthwise edges of both host and graft, to represent symmetric boundary conditions
2. $U_x=0$ and $U_y=0$ at the ends of the host and graft vessels
3. $U_y=0$ on the 15-degree portion of the host, to simulate the effect of the host resting on the muscle layer

4. Pressure application of 8 kPa (equivalent to 60mmHg) to simulate the pressure experienced by the vessels with the return of blood flow and the pumping action of the heart.

As with the previous set of models, angle variations of 30, 45 and 60 degrees between the graft and the artery were considered. The paths defined on the host and graft for the extraction of data of stresses and displacements were exactly the same as that described in the preceding section.

Figure 4.7 Finite element model of an end-to-side anastomosis with additional constraints imposed.

CHAPTER 5 RESULTS AND DISCUSSION (2-D MODELLING)

5.1 Verification of Tangential and Radial Stresses Using Finite Element Software

Before beginning the analysis of the problem with ANSYS, a few simple tests were performed to gain confidence on the correctness of the programme and learn how to use it. This verification exercise was also conducted to acquire basic finite element modelling techniques and compare the results derived from the analyses against those readily-available data based on the theory of thick-walled cylindrical pressure vessels.

In this exercise, a simple model of the artery was constructed using ANSYS 5.4, a commercially-available Finite Element software package, by assuming the artery to resemble a thick-walled cylindrical pressure vessel. An arbitrary internal radial pressure of 100Pa is applied onto the inner wall of the cylinder and the solution from the finite element model is compared against the results computed for a thick-walled cylindrical pressure vessel (Using equations 5.1 and 5.2).

The formula for radial stress for a thick-walled cylindrical pressure is:

$$\sigma_r = \frac{P_i r_i^2}{r_o^2 - r_i^2} \left(1 - \frac{r_o^2}{r^2}\right) \quad (5.1)$$

and the corresponding tangential stress is:

CHAPTER 5: RESULTS AND DISCUSSION (2-D MODELLING)

$$\sigma_t = \frac{P r_i^2}{r_o^2 - r_i^2} \left(1 + \frac{r_o^2}{r^2} \right) \quad (5.2)$$

Figures 5.1 and 5.2 below show the first model ("Model 1") that was constructed. This cylinder is assumed to have an internal radius of 4mm and an external radius of 5mm.

Figure 5.1 Model 1 of Thick-walled cylindrical pressure vessel constructed.

Figure 5.2 Cross Section of Model 1.

The magnitudes for tangential and radial stresses for Model 1 were extracted directly from the finite element model, and were compared against those obtained from theory by plotting Figures 5.3 and 5.4 below.

A comparison of the two plots shows that there are discrepancies in the two sets of results. One of the possible reasons for this was the number of elements used in the modelling. A coarser mesh may result in a less accurate solution obtained.

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Figure 5.3 Comparison of theoretical tangential stress against results from ANSYS (Model 1).

Figure 5.4 Comparison of theoretical radial stress against results from ANSYS (Model 1).

Based on the results above, mesh refinement was carried out for the existing model. In the revised model, only one-quarter of the original model was considered as a result of symmetric conditions. This also allows the elements

within the quadrant to be refined. Figure 5.5 shows the revised finite element model (Model 2) that was constructed.

Figure 5.5 Model 2 of the thick-walled cylindrical pressure vessel constructed.

A comparison of Figure 5.5 with Figure 5.2 shows that the radial stresses experienced by Model 2 as a result of pressure application vary with the radius. A comparison of the tangential and radial stresses between the theoretical and experimental values also shows an exact correspondence in the stress magnitudes whether the problem is solved using Equations (5.1) and (5.2) or obtained by ANSYS.

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Figure 5.6 Comparison of theoretical tangential stress against results from ANSYS (Model 2).

Figure 5.7 Comparison of theoretical radial stress against results from ANSYS (Model 2).

5.2 Two-Dimensional Modelling of End-to-End Anastomoses

5.2.1 Models with Region of Overlap

The two-dimensional modelling of the end-to-end anastomoses with overlap involved determining the profile of the sutured anastomosis by applying displacement conditions to simulate the effect of suturing. This was followed by pressure application to the area of “suture” to analyse the final deformed shape and stresses. Grafts of thicknesses of 0.5mm, 0.6mm and 0.7mm were considered in the modelling.

Figure 5.8 Stress Distribution of “Sutured” Model incorporating the effects of displacement in the Ux direction (For Graft thickness 0.5mm).

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Figure 5.9 Stress Distribution of Host and Graft, after pressure application (For Graft thickness 0.5mm).

Figure 5.10 Stress Distribution of "Sutured" Model incorporating the effects of displacement in the Ux direction. (For Graft thickness 0.6mm).

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Figure 5.11 Stress Distribution of Host and Graft, after pressure application (for Graft thickness 0.6mm).

Figure 5.12 Stress Distribution of "Sutured" Model incorporating the effects of displacement in the Ux direction. (for Graft thickness 0.7mm)

Figure 5.13 Stress Distribution of Host and Graft (for Graft thickness 0.7mm)

Figures 5.8, 5.10 and 5.12 the stress distributions of the sutured models incorporating the effects of displacement in the U_x direction, for graft thicknesses of 0.5mm to 0.7mm, in steps of 0.1mm. Figures 5.9, 5.11 and 5.13 show the stress distributions on the host and graft after pressure is applied onto the three “sutured” models.

From Figures 5.9, 5.11 and 5.13, it could be observed that with pressure application, the largest Von Mises stresses recorded decreased when graft thickness increased. In all three models, the location of maximum stress was at the inner wall of the host.

Figure 5.14 below shows the longitudinal stresses experienced by the various grafts of different thicknesses, after pressure was applied to the sutured model. The largest magnitude of longitudinal stress occurred at the region of interface between the host and the graft. The thinner the graft, the larger the magnitude of longitudinal stresses recorded.

Figure 5.14 Longitudinal Stresses Experienced by Grafts of Different Thicknesses.

Figure 5.15 shows the longitudinal stress experienced by the host due to the effects of varying grafts thicknesses. The origin is taken to be the top of the model. The longitudinal stresses of the host increased along its length, but there are fluctuations in the stress magnitudes towards the region of host-graft interface.

Figure 5.15 Longitudinal Stress of Host Artery, for different graft thicknesses. (Note that the Origin is taken from the top of the model).

Figure 5.16 shows the radial stresses experienced by the grafts. The point of origin on the x-axis is taken at the centre of the region of interface between the host artery and the graft, and it extends for the combined thickness of the anastomosis investigated.

Figure 5.16 Magnitudes of Radial Stress experienced, for grafts of different thicknesses.

Figure 5.17 indicates the horizontal displacement experienced by the host when pressure is applied back to the end-to-end anastomosis. It could be observed that as a result of the pressure application, all elements in the host have undergone a horizontal (outward) displacement of about 2×10^{-2} mm. The direction of horizontal translation is in alignment with the outward direction of pressure applied. More literature searches will need to be conducted to ascertain if the magnitude of such a displacement is in accordance to observations recorded during surgery. At the point of compilation of this report, it is not certain if such a displacement is significant.

Figure 5.17 Displacement Ux for Host.

Figure 5.18 Displacement Ux for Graft.

Figure 5.18 shows the displacement in the Ux direction for the grafts of the three thicknesses investigated as the distance of the graft (measured from the top of the graft) increases. It can be observed that the graft was displaced by a larger amount towards its lower end. One of the possible reasons for this is that, since the origin of the x-axis is at the host-graft interface, the effective thickness of the anastomosis is largest at the origin and decreased as we progress along the x-axis. The relatively thicker region of interface may have resulted in decreased flexibility and hence decrease in displacement value recorded.

Figure 5.19 shows the displacement in the y-direction recorded by the host, for grafts of different thicknesses. A positive U_y displacement value represents an elongation in the model. As observed from the Figure, points at a greater distance from the top of the artery experience a greater elongation. However, for grafts of thicknesses 0.5mm and 0.6mm, there are some points within the artery where a contraction is recorded.

Figure 5.19 Displacement U_y for the Host, for different graft thicknesses.

Figure 5.20 shows the corresponding U_y displacement for the grafts.

Figure 5.20 Displacement U_y for Graft of different thicknesses.

Based on the observations recorded, the following conclusions can be made about the two-dimensional finite element modelling of an end-to-end anastomosis:

- The thicker the graft, the lower the magnitude of Von Mises stresses recorded. This may suggest that an end-to-end anastomosis may be less susceptible to mechanical failure when the thickness of the graft is increased;
- The overall outward displacement experienced by an end-to-end anastomosis is reduced when a thicker graft is utilised, possibly because of increased rigidity in the overall structure.

5.2.2 Models without Region of Overlap

In this section, the suture force needed to close the gap between the host artery and the graft was estimated. Contact elements were created at the bottom edge of the host artery and at the top edge of the host artery to ensure that the force applied could indeed result in contact made between these two vessels.

Figure 5.21 Model with Force and Displacement constraints applied

Using the process of trial and error, it was found that a force application of magnitude of 1.3×10^{-2} N (in the Fx direction) at each of the two ends of the host and graft vessels is sufficient to result in contact between the edges of the host and graft vessels.

The stress distribution of the resultant model as a result of the force application is illustrated in Figure 5.22 below.

Figure 5.22 Stress Distribution of the "Sutured" Model, after application of suture force.

CHAPTER 6 RESULTS AND DISCUSSION (3-D MODELLING)

After the development of the two-dimensional end-to-end anastomotic models, three-dimensional finite element models of end-to-side anastomosis were developed and the effects of pressure application on stress distributions along the host and graft were analysed. Studies done in relation to end-to-side anastomoses are thought to have a greater impact to the surgeons, as end-to-side anastomoses are usually performed for patients requiring coronary artery bypass grafting.

6.1 Element Convergence Tests

Before the finite element models were developed and formal analyses conducted, element convergence tests were performed to determine the appropriate number of elements for the models so that the results will be of reasonable accuracy and cost-effective to run at the same time.

The element convergence test was carried out using the 45-degree finite element model. Five variations were made in the total number of elements used in the entire model, and their corresponding maximum Von Mises stresses were recorded. The element density (expressed in terms of the total number of elements used in the model) and the magnitude of the stress recorded are presented in Figure 6.1. Based on the Figure, it could be observed that the rate of increase of the maximum-recorded Von Mises stress was more prominent when fewer element were used, but it gradually tapered off from about 8000 elements. Based on the results of the element convergence test, one could see that the results indeed converge to a value

beyond a certain element density. The 8000-element model was selected because the deviation in results is less than 10% from that of the 22,000-element model.

Figure 6.1 Results of element convergence test.

6.2 Results of Initial Models

6.2.1 Deformed Shapes

Figures 6.2 to 6.4 show the deformed shapes of the 30-, 45- and 60-degree models, after displacement constraints and pressure were applied. It could be observed from the three figures that the severity of deformation decreased when the angle between the host and graft increased.

CHAPTER 6: RESULTS AND DISCUSSION (3-D MODELLING)

Figure 6.2 Deformed shape of the 30-degree model.

Figure 6.3 Deformed shape of the 45-degree model.

Figure 6.4 Deformed shape of the 60-degree model.

6.2.2 Normalised Stress Plots in x-, y- and z-directions

Based on the path definitions for the host and graft (Refer to Section 4.2.1), the magnitude of stresses in the x- (radial), y- (transverse) and z- (longitudinal) directions were extracted and normalised against the applied pressure of 8,000 Pa. As defined by the paths, stress values of both the inner and outer walls of host and graft were extracted for analyses.

Normalised Stress Plots for the Host

The plots of normalised stresses against normalised length of the host, for both inner and outer walls, are presented in Figures 6.5 to 6.7. Two observations could be made from Figure 6.5: Firstly, for both inner and outer walls, the smaller the angle, the higher the normalised stresses recorded. Secondly, taking into account the path definitions for the host, higher magnitudes of normalised radial stresses were registered for Direction One than Direction Two.

Even though there were differences in the highest magnitude of stresses recorded, all three models had nominal stress ratio S_x/P_o of about 30, which occurred from $x/L = 0.25$. This translates to a nominal radial stress value of about 240kPa.

CHAPTER 6: RESULTS AND DISCUSSION (3-D MODELLING)

Figure 6.6 shows the graph of normalised stress for the transverse direction against normalised length for the host, for inner and outer walls. The registered stresses appeared more erratic when compared to the normalised

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stresses in the x-direction. There was no obvious trend. The normalised transverse stress ratio recorded was zero at $x/L \approx 0.5$.

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Figure 6.7 shows the normalised stresses in the z- (longitudinal) direction for inner and outer walls of the host. For all the angles considered, the stress value recorded was highest at the origin of the x-axis. This coincides with the region of interface between the host and the graft. The stresses then decrease, and for each of the angles and paths, the lowest value occurred at about 50% of the total length of the path considered.

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The nodal stress plots (in three orthogonal views) for a typical host vessel – in the radial, transverse and longitudinal directions – are shown in Figures 6.8 to 6.10 below.

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Figure 6.8 Nodal stress plot of a host vessel in the radial direction.

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Figure 6.9 Nodal stress plot of a host vessel in the transverse direction.

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Figure 6.10 Nodal stress plot of a host vessel in the longitudinal direction.

Normalised Stress Plots for the Graft

Figures 6.11 to 6.13 shows the normalised stresses versus length plots for the inner and outer walls of the grafts, in the x-, y- and z-directions. The normalised S_x value for the graft was higher for path Direction Two than Direction One. The results of Figure 6.11 were consistent with those of Figure 6.5, since path Direction Two defined for the graft falls on the same side as path Direction One defined for the host. For the same path considered, the normalised stress value was higher when the angle between the host and the graft was smaller. For the inner wall of the graft, the nominal stress value was close to zero, and this occurred at approximately 50% of the length of the graft segment defined for analysis.

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Figures 6.12 and 6.13 show the normalised stresses of the graft, in the transverse and longitudinal directions. High peaks in stress values are recorded for the transverse and longitudinal stresses in Direction One of the outer wall of the graft, for the 30-degree model. The results indicate that higher stress values are usually found near to the origin of the x-axis, thus

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suggesting that the region of interface between the host and the graft is a critical region.

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The nodal stress plots of the models – extracted from ANSYS – can be found from Figures 6.14 to 6.16.

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Figure 6.14 Nodal stress plot of a graft vessel in the radial direction.

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Figure 6.15 Nodal stress plot of a graft vessel in the transverse direction.

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Figure 6.16 Nodal stress plot of a graft vessel in the longitudinal direction.

6.2.3 Normalised Principal Stresses Plots

Normalised Principal Stress Plots for Host

Besides analysing the stresses in the radial, transverse and longitudinal directions, values of the three principal stresses were also extracted from the defined paths for analyses. The results are presented in Figures 6.17 to 6.22 below.

For the three principal stresses considered for the host, highest values were recorded for the first principal stress. For the same angle for the host, Direction One of the path defined registered higher stress values compared to Direction Two. The 30 degree model had the largest recorded stress. However, regardless of the angle and path direction, the nominal normalised ratio is about 35 for the inner wall of the host, and this occurred from about 30% of the total length host path definition in both directions. This translates to a stress value of about 280kPa.

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As compared to the first principal stress, there was no apparent trend for the second principal stress S_2 for all three models, except that the stresses in Direction One of the defined path for the host decreased along the length of the host, whereas the magnitude of S_2 -stresses in Direction Two under the

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defined path initially decreased along the length of the host, but started to increase from $x/L \approx 0.4$. The plots for the normalised third principal stress of the graft (Figure 6.22) also revealed fluctuations at the beginning of the defined path, before the nominal value close to zero was attained at about $x/L \approx 0.4$.

Figures 6.20 to 6.22 show the nodal principal stress plots for a typical host vessel.

Figure 6.20 Principal nodal stress plot (S1) for a host vessel.

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Figure 6.21 Principal nodal stress plot (S2 direction) for a host vessel.

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Figure 6.22 Principal nodal stress plot (S3 direction) for a host vessel.

Normalised Principal Stress Plots for Grafts

For the grafts, the largest magnitudes for the first principal stresses were recorded for Direction Two of the graft, which is of the same side as the host with the highest recorded stresses. Nominal stresses for the inner wall of the host are close to zero, whereas that for the outer wall has an average value of about 320kPa. For the same model considered, the highest principal stress values for the graft were higher than those of the host.

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Figures 6.17 to 6.19 as well as 6.23 to 6.25 show that nominal principal values were attained by both the host and the graft beyond a certain ratio of the total length of the paths considered. However, there appears to be fluctuations in the principal stress values, towards the end of the paths (i.e. where $x/L \approx 1$). One of the possible reasons for this is that the lengths of the host and graft in the finite element models that were constructed were not long enough to discard the effects of boundary condition applications at the ends of the host and graft. It was decided that the lengths of the host and graft be lengthened for the next set of results generated (see Section 6.3).

Figures 6.26 to 6.28 show the nodal stress plots for a typical graft for the three principal stresses.

Figure 6.26 Principal nodal stress plot (S1) for a graft vessel.

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Figure 6.27 Principal nodal stress plot (S2) for a graft vessel.

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Figure 6.28 Principal nodal stress plot (S3) for a graft vessel.

6.2.4 Normalised Shear Stresses Plots (Inner Wall)

Normalised Wall Shear Stress Plots for the Host

Figures 6.29 to 6.31 show the normalised inner wall shear stress plots for the host, in the xy-, yz- and xz-directions. The normalised inner wall shear stress ratios for these three directions are -5, 0 and 0 respectively. A comparison between the three angles investigated show that the 30-degree model experienced more fluctuations in shear stress values as compared to the other two models. It also recorded the highest magnitude of shear stresses.

Figure 6.29 Normalised wall shear stress (XY-direction) plot for the inner wall of the host.

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Figure 6.30 Normalised wall shear stress (YZ-direction) plot for the inner wall of the host.

Figure 6.31 Normalised wall shear stress (XZ-direction) plot for the inner wall of the host.

Normalised Wall Shear Stress Plots for the Graft

Figures 6.32 to 6.34 show the corresponding inner wall shear stress plots for the graft. Likewise, the 30-degree models of the graft demonstrated greater fluctuations and magnitudes of shear stress values.

Figure 6.32 Normalised wall shear stress (XY-direction) plot for the inner wall of the graft.

Figure 6.33 Normalised wall shear stress (YZ-direction) plot for the inner wall of the graft.

Figure 6.34 Normalised wall shear stress (XZ-direction) plot for the inner wall of the graft.

6.2.5 Normalised Displacement Plots (Outer Wall)

Normalised Displacement Plots for Host

Figures 6.35 and 6.36 show the graphs of normalised displacement (in the y- and z-directions) against the normalised lengths for the host. From Figure 6.35, it was observed that for each of the six paths examined, the maximum displacement in the y-direction was recorded at around $x/L \approx 0$, which corresponded to the region of anastomosis.

Figure 6.35 Graph of normalised displacement (UY) against normalised length for the outer wall of the Host.

Figure 6.36 shows that the displacement of the host in the z- (longitudinal) direction is dependent on the path defined for the host. For the three paths in Direction One, there was an increase in the normalised UZ-displacement along the length of the paths defined. For each of the three models, maximum magnitudes of longitudinal displacements were recorded at the end of Path Direction One. The three paths that were in Direction Two of the path definition for the host recorded a decrease in displacement along the path length.

Figure 6.36 Graph of normalised displacement (UZ) against normalised length for the outer wall of the Host.

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Figure 6.38 Nodal displacement plots (UZ direction) for a host vessel.

Normalised Displacement Plots for Graft

Figures 6.39 and 6.40 show the graphs of normalised displacements (in the y- and z-directions) against the normalised lengths for the outer wall of the graft. The maximum displacements in the y-direction were recorded at around $x/L \approx 0$. This was identical to the observation made for the host vessel. There was, however, a slightly different trend observed for the UZ-displacements of the graft. The three paths in Direction One of the graft experienced an increasing trend in the magnitude of longitudinal displacements as they moved further away from the region of anastomosis. For grafts of Path Direction Two, however, the magnitudes of longitudinal displacements decreased from the region of anastomosis till about $x/L \approx 0.3$, following which there was an increase in the displacements recorded.

Figure 6.39 Graph of normalised Displacement (UY) against normalised length for the outer wall of the Graft.

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Figure 6.40 Graph of normalised displacement (U_z) against normalised length for the outer wall of the Graft.

Figures 6.41 and 6.42 show the nodal displacements in the y- and z-directions for a typical graft.

Figure 6.41 Nodal displacement plots (UY direction) for a graft vessel.

Figure 6.42 Nodal displacement plots (UZ direction) for a graft vessel.

6.3 Results of Models with Additional Displacement Constraints

Modifications were made to the original three finite element models developed by introducing additional displacement constraints, to simulate the effect of the host resting against the heart muscle. In addition, the lengths of the host and graft were increased and it was decided that the portion of results to be analysed should end just before $x/L \approx 1$, in order to assume that the host and graft modelled are infinitely long and the conditions at the boundary lengths can be ignored. The three modified models were then solved to obtain the following set of results.

6.3.1 Deformed Shapes

Figures 6.43 to 6.45 show the deformed shapes of the models that were derived as a result of the inclusion of additional displacement constraints. As observed from these three figures, the top segment of the host now appeared rigid as a result of the displacement constraints imposed on the 15-degree of its radial segment, to simulate the effect of it resting and being supported by the heart muscle. Of the three models, the 30-degree model experienced the most severe deformations, especially at the region of interface between the host and the graft. On the contrary, the 60-degree model showed the least deformations.

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Figure 6.43 Deformed shape of 30-degree model with additional displacement constraints.

Figure 6.44 Deformed shape of 45-degree model with additional displacement constraints.

Figure 6.45 Deformed shape of 60-degree model with additional displacement constraints.

6.3.2 Normalised Stress Plots in x-, y- and z-directions

Normalised Stress Plots for Host

Figures 6.46 to 6.48 show the normalised stress plots of the host in the x- (radial), y- (transverse) and z- (longitudinal) directions. In all the three figures, for both inner and outer walls, the highest stress ratio was registered by path Direction One of the 30-degree model.

The nominal stress ratio in the x-direction, for all the three models and the six paths considered, is approximately 35. This occurs at about $x/L = 0.3$. The results suggest that the variation in anastomotic angle is crucial only at the region of interface between the host and the graft, but beyond a certain point, the stresses experienced are independent of anastomotic angles.

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As compared to the normalised stresses in the x-direction, there are greater fluctuations of stresses experienced by the host in the y-direction. The nominal stress value is zero, occurring at about $x/L = 0.5$.

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The graph of normalised stresses versus length in the z-direction show a decreasing trend in the stresses experienced by the host in the z-direction, as one progresses along the paths defined for the host – which happen to be away from the interface between the host and the graft. The nominal stress magnitude attained by the host was close to zero, away from the vicinity of the host-graft interface.

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The nodal stress plots in the radial, transverse and longitudinal directions for a host vessel are illustrated in Figures 6.49 to 6.51 below.

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Figure 6.49 Nodal stress plots in the radial direction for a host vessel (additional boundary conditions imposed).

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Figure 6.50 Nodal stress plots in the transverse direction for a host vessel (additional boundary conditions imposed).

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Figure 6.51 Nodal stress plots in the longitudinal direction for a host vessel (additional boundary conditions imposed).

Normalised Stress Plots for the Graft

Figures 6.52 to 6.54 show the normalised stress plots of the graft in the x- (radial), y- (transverse) and z- (longitudinal) directions. In all the three figures for both inner and outer walls, the highest stress ratio was registered by path Direction Two of the 30-degree model, which is at the same side as the host.

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Figures 6.55 to 6.57 show the nodal stress plots (extracted from ANSYS) of the graft, in the x-, y- and z-directions. The highest magnitude of stress recorded by the model decreased as the anastomotic angle increased. This observation is similar to that for the initial models developed (i.e. without additional boundary conditions imposed).

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Figure 6.55 Nodal stress plots in the radial direction for a graft vessel (additional boundary conditions imposed).

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Figure 6.56 Nodal stress plots in transverse direction for a graft vessel (additional boundary conditions imposed).

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Figure 6.57 Nodal stress plots longitudinal direction for a graft vessel (additional boundary conditions imposed).

6.3.3 Normalised Principal Stresses Plots

Normalised Principal Stress Plots for Host

Figures 6.58 to 6.60 show the graphs of the normalised principal stresses plotted against normalised length of the host, for both the inner and outer walls.

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The nodal principal stress plots for a host vessel are illustrated from Figures 6.61 to 6.63 below:

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Figure 6.62 Nodal principal stress plots (S2) for a host vessel (with additional boundary conditions imposed).

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Normalised Principal Stress Plots for Graft

Figures 6.64 to 6.66 show the graphs of the normalised principal stresses against normalised length of the graft, for both the inner and outer walls.

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A comparison of the principal stress plots for this modified set of models generated with the previous set of models (without additional boundary conditions) show that with the imposition of additional boundary conditions, the principal stress values obtained for both host and graft appear to converge to a nominal value, regardless of the anastomotic angle considered. These results suggest that beyond a certain ratio of the length, the nominal principal

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stress values are independent of the angle of anastomosis between the host and the graft.

Figures 6.67 to 6.69 show the principal stresses nodal plots (extracted from ANSYS) for a typical graft, based on the modified displacement constraints.

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Figure 6.67 Nodal principal stress plots (S1) for a graft vessel (with additional boundary conditions imposed).

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Figure 6.68 Nodal principal stress plots (S2) for a graft vessel (with additional boundary conditions imposed).

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6.3.4 Normalised Shear Stresses Plots (Inner Wall)

Normalised Shear Stress Plots for Host

Figures 6.70 to 6.72 show the graphs of the normalised shear stresses – S_{xy} , S_{yz} and S_{xz} – plotted against the normalised length, for the inner wall of the host.

Figure 6.70 Graph of normalised shear stress S_{xy} against normalised length for inner wall of host.

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Figure 6.71 Graph of normalised shear stress S_{yz} against normalised length for inner wall of host.

Figure 6.72 Graph of normalised shear stress S_{xz} against normalised length for inner wall of host.

Normalised Shear Stress Plots for Graft

Figures 6.73 to 6.75 show the graphs of the normalised shear stresses – S_{xy} , S_{yz} and S_{xz} – plotted against the normalised length, for the inner wall of the graft. For both the host and the graft, the highest magnitudes of wall shear stresses are experienced by the 30-degree model.

Figure 6.73 Graph of normalised shear stress S_{xy} against normalised length for inner wall of graft.

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Figure 6.74 Graph of normalised shear stress S_{yz} against normalised length for inner wall of graft.

Figure 6.75 Graph of normalised shear stress S_{xz} against normalised length for inner wall of graft.

6.3.5 Normalised Displacement Plots (Outer Wall)

Normalised Displacement Plots for Host

Figures 6.76 and 6.77 show the graphs of normalised displacement in the y- and z-directions against normalised lengths for the outer wall of the host. From Figure 6.76, it was observed that the largest absolute magnitudes of displacements were recorded at around $x/L \approx 1$, towards the end of the path segment considered. This was in direct contrast to the results that were derived for the models without the additional displacement constraints, where the largest displacements were recorded in the immediate vicinity of the anastomosis.

From the Figure, it could be seen that Path Direction Two for the host recorded negative normalised displacements in the region of anastomosis ($x/L \approx 0$). The largest magnitude of displacement recorded in the y-direction is negative, meaning that the graft is contracting in length, the results show that for Path Direction Two, there was an increase in transverse displacement of the host vessel before it started to decrease from about $x/L \approx 0.1/0.2$, depending on the angle of anastomosis considered.

Figure 6.76 Graph of normalised displacement (U_y) against normalised length for the outer wall of the host.

Figure 6.77 Graph of normalised displacement (U_z) against normalised length for the outer wall of the host.

Figure 6.77 shows the normalised longitudinal displacement of the host, for the paths defined. The results indicate that the two paths for the 45- and 60-degree models experienced different signs in the normalised displacements. This means that from the anastomosis, one of the segments experiencing an increase in length whereas the other would be experiencing a decrease in length.

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Normalised Displacement Plots for Graft

Figures 6.80 and 6.81 show the graphs of normalised displacement in the y- and z-directions against normalised lengths for the outer wall of the graft. All three models recorded zero y-displacement towards the end of their paths defined. However, the patterns of “damping” or “dying-out effects” were significantly different for the six paths registered. Of significant interest is that of the 45-degree model. For this model, path Direction One showed erratic fluctuations in the positive and negative normalised displacement regions. This implied that across the length of the path considered, there were segments which were experiencing transverse expansions and contractions simultaneously. However, across the other side of the anastomosis, the pattern of expansion / contraction was more gradual.

Figure 6.80 Graph of normalised displacement (U_y) against normalised length for the outer wall of the graft.

For the longitudinal (z-) displacements, it was also observed that Path Direction One of the 45-degree model experienced fluctuations in expansion and contraction across the length of the path considered. However, in this

case, the highest magnitude of longitudinal displacement was recorded in the region of anastomosis of Path Direction Two.

Figure 6.81 Graph of normalised displacement (U_z) against normalised length for the outer wall of the graft.

Figures 6.82 and 6.83 show the nodal displacements in the y- and z-directions, for a graft developed.

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Figure 6.83 Nodal displacement plots in the UZ direction for a graft vessel (additional boundary constraints imposed).

6.4 Effects of Angle Variation on Displacement and Stress Magnitudes

The results of the finite element modelling studies conducted so far suggested that when considerations are made to the component and principal stresses, having a larger anastomotic angle between the host and the graft would give rise to lower magnitudes of stresses. From the mechanical stresses point of view, the sixty degree model would be the most desirable model out of the three models considered because it results in the least magnitude of principal stresses (Figures 6.84 to 6.86).

For models with and without additional displacement constraints to represent the epicardium, higher interface stresses are observed with smaller angles between the host and the graft.

Figure 6.84 Effects of angle variation on principal stresses of Host – Initial models.

Figure 6.85 Effects of angle variation on principal stresses of Graft – Initial models.

Figure 6.86 Effects of angle variation on principal stresses of Host – with additional displacement constraints.

Figure 6.87 Effects of angle variation on principal stresses of Graft – with additional displacement constraints.

Figures 6.88 and 6.89 show the maximum displacements recorded in the y- and z-directions, for the three models initially created. A negative displacement value indicates that the host/graft vessel experiences a contraction. When the absolute magnitudes of displacements are considered (i.e. ignoring the sign conventions), it could be observed that – except for the case of displacement in the z-direction for the graft – the 60-degree model registered the lowest magnitude of displacement.

(a) Maximum UY displacement of host, for different angles.

(a) Maximum UY displacement of graft, for different angles.

Figure 6.88 Effects of angle variation on maximum displacement value in the y-direction – initial models.

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(a) Maximum UZ displacement of host, for different angles.

(a) Maximum UZ displacement of graft, for different angles.

Figure 6.89 Effects of angle variation on maximum displacement value in the z-direction – initial models.

Figures 6.90 and 6.91 show the maximum displacements recorded in the y- and z-directions when additional displacement constraints were applied to the models. When the signs of the displacement values were ignored, it was observed that – except for the case of displacement in the z-direction for the host – the 60-degree model registered the lowest magnitude of displacements.

The results indicated that when the effects of deformations and displacements are considered, the 60-degree anastomosis gives the best results.

(a) Maximum UY displacement of host, for different angles.

(a) Maximum UY displacement of graft, for different angles.

Figure 6.90 Effects of angle variation on maximum displacement value in the y-direction – with additional displacement constraints.

(a) Maximum UZ displacement of host, for different angles.

(a) Maximum UZ displacement of graft, for different angles.

Figure 6.91 Effects of angle variation on maximum displacement value in the z-direction – with additional displacement constraints.

6.5 Effects of Displacement Constraints on Stress Magnitudes

For the three-dimensional end-to-side anastomoses, two categories were considered: the first set of models created involved essential boundary conditions, and the second set of models created involved the incorporation of additional boundary conditions on the host, to simulate the effect of support from the heart muscle.

When additional displacement constraints are imposed on the host, for the same angle between the host and the graft, the magnitudes of maximum principal stresses recorded for the host was lower as compared to the initial models created. However, the magnitudes of principal stresses experienced by the graft were higher.

Table 6.1 Comparison of largest principal stresses for the host, for the two sets of models generated.

	Initial Models			Modified Models (with additional displacement constraints)		
	S1 (MPa)	S2 (MPa)	S3 (MPa)	S1 (MPa)	S2 (MPa)	S3 (MPa)
30 Deg	2.92	1.38	1.19	2.47	1.42	1.00
45 Deg	1.72	0.735	0.522	1.71	0.577	0.450
60 Deg	1.35	0.497	0.323	1.17	0.465	0.303

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Table 6.2 Comparison of largest principal stresses for the graft, for the two sets of models generated.

	Initial Models			Modified Models (with additional displacement constraints)		
	S1 (MPa)	S2 (MPa)	S3 (MPa)	S1 (MPa)	S2 (MPa)	S3 (MPa)
30 Deg	5.31	2.08	1.17	5.95	2.41	1.05
45 Deg	3.12	0.727	0.548	3.10	0.808	0.300
60 Deg	2.24	0.586	0.223	2.20	0.553	0.168

To a large extent, the additional displacement constraints to simulate the effect of the host resting on the heart muscle probably define the most rigid situation that the host can possibly experience. In the more realistic situation, one would expect the largest principal stresses experienced by the host and graft to be somewhere intermediate between these two sets of results generated.

6.6 Critical Stress Regions

From Section 4.2.1 of the report, the critical regions in the three-dimensional end-to-side anastomoses were identified as the region of intersection between the host and graft vessels, due to discontinuities in material properties in this region.

For the initial set of models created, the nodes where the highest magnitude of stresses and deformations occurred and their locations are highlighted from Figures 6.92 to 6.94 below:

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(a) 30 Degree Host – Initial model

(b) 30 Degree Graft – Initial model

Figure 6.92 Location of highest stresses and deformations for (a) host and (b) graft for the 30 degree initial model.

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(a) 45 Degree Host – Initial model

(b) 45 Degree Graft – Initial model

Figure 6.93 Location of highest stresses and deformations for (a) host and (b) graft for the 45 degree initial model.

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(a) 60 Degree Host – Initial model

(b) 60 Degree Graft – Initial model

Figure 6.94 Location of highest stresses and deformations for (a) host and (b) graft for the 60 degree initial model.

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Based on Figures 6.92 to 6.94, it can be deduced that the locations of the nodes where the highest magnitude of stresses occurred are located at the region of interface between the host and graft vessels. For the same angle considered, the magnitude of principal stresses experienced by the graft is higher than that experienced by the host vessel. When the angle between the host and graft increases, the magnitude of highest stresses experienced by the vessels decrease.

For the set of models with additional constraints applied, the locations of highest stresses and deformations and their locations are illustrated in Figures 6.95 to 6.97 below:

(a) Host

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(b) Graft

Figure 6.95 Location of highest stresses and deformations for (a) host and (b) graft for the 30 degree model with modified boundary conditions.

(a) Host

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Figure 6.96 Location of highest stresses and deformations for (a) host and (b) graft for the 45 degree model with modified boundary conditions.

Figure 6.97 Location of highest stresses and deformations for (a) host and (b) graft for the 60 degree model with modified boundary conditions.

As with the initial models created, the locations of the nodes where the highest magnitude of stresses occurred are also located at the region of interface between the host and graft vessels, and the magnitudes of principal stresses experienced by the graft is higher than that experienced by the host vessel for the same angle considered.

The identification of the joint of anastomosis – or the region of interface between the host and graft – as the critical region suggests that the results obtained from this finite element study are in harmony with the literature findings conducted thus far. These results suggest that in the design of an interposition sleeve, more attention should be paid in the region of intersection between the vessels, and reinforcements may be needed in this area to minimise the probability of the host or the graft failing.

6.7 Application of Mechanical Failure Criterion to Models

This section discusses the possibility of applying mechanical failure criteria in the analysis of blood vessels to establish similar failure. Literature findings that were reported had used the ultimate tensile strength as a means of determining the failure of the blood vessels (Refer to Section 2.6.4 of report). Moreover, unlike conventional materials where the yield and ultimate tensile stresses are fixed, the tensile strength of blood vessels is a function of other factors such as pre-stress, localisation, age, medical conditions and body weight of the person considered.

The following mechanical failure theories are considered and their applicability to failure of blood vessels is examined:

- Maximum stress theory
- Equivalent Von Mises stress
- Equivalent Von Mises strain

6.7.1 Maximum Stress Theory

Failure occurs when one of the stresses in principal material direction reaches the ultimate value. For a three-dimensional case, three separate criteria are as follows:

$$\begin{aligned}\sigma_1 &\geq F_{1U} \\ \sigma_2 &\geq F_{2U} \\ \sigma_3 &\geq F_{3U}\end{aligned}\tag{6.1}$$

Equation (6.1) suggests given the physiological conditions of any person, failure of the blood vessels would occur once any of the three principal stresses experienced by the host / graft exceeds the ultimate tensile strength recorded for a particular type of blood vessel.

Consider the carotid artery of a person of age 20 – 29 years. Using Table 2.5 of the report as a guideline, the tensile strength of the carotid artery in the circumferential direction is 1.63MPa. If the carotid artery is used as the graft

vessel, it would fail if any of the recorded principal stresses exceeds this ultimate tensile strength value. Comparing this value against the values derived from the finite element modelling, one would expect the blood vessels to fail since the maximum principal stress recorded far exceeded the tensile strength of the carotid artery. However, until the actual tensile strength of the graft is established, no conclusion on the failure of the blood vessels can be made. Also, in the finite element models created, the host and the graft are assumed to be joined at perfect angles, and the properties and effects of the suture were not incorporated. In reality, the suture lines may create additional reinforcement in the critical regions where the host and the graft are joined, thus providing more support against failure.

6.7.2 Equivalent Von Mises Stress

Expressed in terms of principal stresses and strains, the equivalent Von Mises stress is:

$$\sigma_e = \frac{1}{\sqrt{2}} [(\sigma_1 - \sigma_2)^2 + (\sigma_2 - \sigma_3)^2 + (\sigma_3 - \sigma_1)^2]^{\frac{1}{2}} \quad (6.2)$$

Using Equation (6.2), the computed Von Mises stresses for host and graft for the initial models developed are as follows:

Table 6.3 Equivalent Von Mises stresses of host, for the initial models developed (computations based on maximum principal stresses).

	Max. Principal Stress σ_1 (MPa)	Max. Principal Stress σ_2 (MPa)	Max. Principal Stress σ_3 (MPa)	Equivalent Von Mises Stress σ_e (MPa)
30 Deg	2.9193	1.3768	1.192	1.643
45 Deg	1.7237	0.73532	0.52191	1.111
60 Deg	1.348	0.49683	0.32251	0.9504

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Table 6.4 Equivalent Von Mises stresses of Graft, for the initial models developed (computations based on maximum principal stresses).

	Max. Principal Stress σ_1 (MPa)	Max. Principal Stress σ_2 (MPa)	Max. Principal Stress σ_3 (MPa)	Equivalent Von Mises Stress σ_e (MPa)
30 Deg	5.3083	2.0751	1.1737	3.766
45 Deg	3.1214	0.72705	0.54758	2.489
60 Deg	2.2394	0.58634	0.22335	1.861

The calculations indicate that the equivalent Von Mises stress decreases when the angle between host and graft increases. This indicates that the both blood vessels are less likely to fail when the angle between them is larger. Until documentation on the yield strength of the blood vessels has been established, a conclusion as to whether the blood vessels will fail as a result of the displacement constraints and pressure application cannot be reached.

Tables 6.5 and 6.6 show the computed equivalent Von Mises stresses of the host and the graft when additional displacement boundary conditions are applied to simulate the effect of the host resting on the heart muscle:

Table 6.5 Equivalent Von Mises stresses of Host, with additional constraints applied (computations based on maximum principal stresses).

	Max. Principal Stress σ_1 (MPa)	Max. Principal Stress σ_2 (MPa)	Max. Principal Stress σ_3 (MPa)	Equiv. Von Mises Stress σ_e (MPa)
30 Deg	2.4739	1.4151	1.0002	1.316
45 Deg	1.7055	0.57764	0.45023	1.197
60 Deg	1.1773	0.46466	0.30274	0.8059

Table 6.6 Equivalent Von Mises stresses of Graft, with additional constraints applied (computations based on maximum principal stresses).

	Max. Principal Stress σ_1 (MPa)	Max. Principal Stress σ_2 (MPa)	Max. Principal Stress σ_3 (MPa)	Equiv. Von Mises Stress σ_e (MPa)
30 Deg	5.9502	2.4106	1.0524	4.380
45 Deg	3.1043	0.80826	0.30021	2.588
60 Deg	2.2029	0.55321	0.16784	1.872

When additional displacement constraints are imposed, it was also observed that as with the case of the initial models developed, the equivalent Von Mises

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stress also decreases when the angle between the host and graft increases. However, by comparing the results of the host and those of the graft for the two sets of boundary conditions, the magnitude of equivalent Von Mises stresses decreases for the host with the additional displacement boundary conditions. The equivalent Von Mises stresses for the graft increases for the modified set of models developed.

A comparison of Figures 6.14 to 6.19 – the normalised principal stresses plots for the initial models developed versus Figures 6.47 to 6.52 – the normalised principal stresses plots for the modified models show that when additional boundary conditions are imposed, the principal stress values obtained for both host and graft appear to converge to a nominal value, regardless of the anastomotic angle considered. These results suggest that beyond a certain ratio of the total length of the path considered, the nominal principal stress values are independent of the angle of anastomosis between the host and the graft. On this note, computations were made to calculate the nominal equivalent principal stress values that are likely to be experienced by the host and the graft, and these are presented in Tables 6.7 to 6.10 below:

Table 6.7 Equivalent Von Mises stresses of inner wall of host, with additional constraints applied (computations based on nominal principal stresses).

	Nominal Principal Stress σ_1 (MPa)	Nominal Principal Stress σ_2 (MPa)	Nominal Principal Stress σ_3 (MPa)	Equiv. Von Mises Stress σ_e (MPa)
All models	0.216	0	0	0.216

Table 6.8 Equivalent Von Mises stresses of outer wall of host, with additional constraints applied (computations based on nominal principal stresses).

	Nominal Principal Stress σ_1 (MPa)	Nominal Principal Stress σ_2 (MPa)	Nominal Principal Stress σ_3 (MPa)	Equiv. Von Mises Stress σ_e (MPa)
All models	0.306	0.0172	0	0.298

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Table 6.9 Equivalent Von Mises stresses of inner wall of graft, with additional constraints applied (computations based on nominal principal stresses).

	Nominal Principal Stress σ_1 (MPa)	Nominal Principal Stress σ_2 (MPa)	Nominal Principal Stress σ_3 (MPa)	Equiv. Von Mises Stress σ_e (MPa)
All models	0.177	0.0774	0	0.154

Table 6.10 Equivalent Von Mises stresses of outer wall of graft, with additional constraints applied (computations based on nominal principal stresses).

	Nominal Principal Stress σ_1 (MPa)	Nominal Principal Stress σ_2 (MPa)	Nominal Principal Stress σ_3 (MPa)	Equiv. Von Mises Stress σ_e (MPa)
All models	0.147	0.0708	0	0.127

Comparing the equivalent Von Mises stresses computed based on the nominal principal stress values against those computed based on the maximum principal stress values, one could see that the nominal equivalent Von Mises stress values do not exceed 300kPa. These values are also well below the values of Young's Moduli that were used in the development of the finite element models.

6.7.3 Equivalent Von Mises Strain

Based on literature, research findings on the stress-strain relationships established for blood vessels thus far had indicated strain dominance in the stress-strain relationship. The equivalent Von Mises strain should therefore be used as one of the criteria for consideration when discussing about mechanical failure.

There are six components in a strain tensor. While each component measures its own specific characteristics of deformation, it is not easy to estimate the overall effect integrating all six components. These strain components are often reduced down to three principal strains – eigenvalues of the strain tensor – where the maximum and minimum principal directions have some meaningful information related to the deformation. The strain

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components can be reduced further down to one value so that the overall effect of the deformation can be visualised. The Von Mises yield criteria is one of the most widely used concepts that have provided a practical guideline in estimating the mechanical safety of the structure in the field of computational mechanics. The Von Mises strain is defined as follows:

$$\varepsilon_{VM}^2 = \frac{1}{3}[(\varepsilon_x - \varepsilon_y)^2 + (\varepsilon_y - \varepsilon_z)^2 + (\varepsilon_z - \varepsilon_x)^2 + 2(\varepsilon_{xy}^2 + \varepsilon_{yz}^2 + \varepsilon_{xz}^2)] \quad (6.3)$$

The equivalent strain based on Von Mises is:

$$\begin{aligned} \varepsilon_e &= \beta [(\varepsilon_1 - \varepsilon_2)^2 + (\varepsilon_2 - \varepsilon_3)^2 + (\varepsilon_1 - \varepsilon_3)^2]^{\frac{1}{2}} \\ &= \frac{1}{\sqrt{2}(1+\nu)} [(\varepsilon_1 - \varepsilon_2)^2 + (\varepsilon_2 - \varepsilon_3)^2 + (\varepsilon_1 - \varepsilon_3)^2]^{\frac{1}{2}} \end{aligned} \quad (6.4)$$

where ν is the Poisson's ratio and

$$\beta = \frac{1}{\sqrt{2}(1+\nu)} \quad (6.5)$$

The values of ε_e are computed for the initial models developed can be found in Table 6.11 below:

Table 6.11 Equivalent Von Mises strain of Host, for initial models developed.

	Poisson's ratio, ν	Max. Principal strain ε_1	Max. Principal strain ε_2	Max. Principal strain ε_3	Equiv. Von Mises strain ε_e
30 Deg	0.45	1.9491	0.42043	-5.45×10^{-2}	1.251
45 Deg	0.45	1.3605	0.23781	-3.35×10^{-2}	0.8828
60 Deg	0.45	1.0863	0.17322	-2.68×10^{-2}	0.7088

Table 6.12 Equivalent Von Mises strain of Graft, for initial models developed.

	Poisson's ratio, ν	Max. Principal strain ε_1	Max. Principal strain ε_2	Max. Principal strain ε_3	Equiv. Von Mises strain ε_e
30 Deg	0.45	3.5131	0.42781	-1.02×10^{-2}	2.294
45 Deg	0.45	2.0558	0.16838	-5.91×10^{-4}	1.364
60 Deg	0.45	1.4723	0.21428	-1.63×10^{-2}	0.9571

For the models with additional displacement constraints applied to simulate the effect of the epicardium, the values of ϵ_e are illustrated in Table 6.13 below:

Table 6.13 Equivalent Von Mises strain of Host, with additional displacement boundary conditions imposed.

	Poisson's ratio, ν	Max. Principal strain ϵ_1	Max. Principal strain ϵ_2	Max. Principal strain ϵ_3	Equiv. Von Mises strain ϵ_e
30 Deg	0.45	1.9605	0.3894	-7.65×10^{-2}	1.275
45 Deg	0.45	1.2879	0.2481	-4.32×10^{-2}	0.8359
60 Deg	0.45	1.0146	0.12163	-6.16×10^{-2}	0.6878

Table 6.14 Equivalent Von Mises strain of Graft, with additional displacement boundary conditions imposed.

	Poisson's ratio, ν	Max. Principal strain ϵ_1	Max. Principal strain ϵ_2	Max. Principal strain ϵ_3	Equiv. Von Mises strain ϵ_e
30 Deg	0.45	3.8322	0.49602	-7.34×10^{-5}	2.490
45 Deg	0.45	2.0722	0.27693	-8.33×10^{-5}	1.344
60 Deg	0.45	1.4103	0.19619	-9.38×10^{-5}	0.9126

6.8 Relationship between Wall Shear Stress and Intimal Hyperplasia

Analyses that were conducted prior this section of the report had focussed on how the stresses generated in an end-to-side anastomotic joint differ according to the anastomotic angles considered. From the mechanical viewpoint, a larger anastomotic angle is desirable because of the lower resultant stresses generated, in both component and principal directions.

However, analyses of the wall shear stresses generated as a result of variances in the anastomotic angle indicate otherwise. Based on the literature findings (please refer to Section 2.2 of this report), wall shear stress is the essential hemodynamic factor related to intimal hyperplasia. Low wall shear stresses produced greater amounts of pseudointimal thickening. The lower

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the wall shear stress experienced by the wall of the vessel, the higher the chance of developing intimal hyperplasia. Based on the argument of the wall shear stresses generated, a 30-degree model would be desirable as it experienced higher wall shear stresses and hence has a lower chance of developing intimal hyperplasia. On the contrary, a 60-degree model is more likely to develop intimal hyperplasia as lowest wall shear stresses are generated from this model!

Moreover, from Section 2.2 of the report, the smaller the anastomotic angle, the smaller the flow disturbances. Based on this argument, the 30-degree would be the most optimal model from the fluid-flow point of view, since it has the largest recorded wall shear stress value, and is the smallest of the three anastomotic angles considered.

CHAPTER 7 CONCLUSION

The effects of graft thicknesses on stress distributions for an end-to-end anastomosis and that of suture angles for end-to-side anastomoses on stresses experienced by the host and graft vessels were investigated. There are several conclusions that one can draw from the studies, and these are described below:

The results of the two-dimensional modelling of an end-to-end anastomosis suggest that the thickness of the graft is an important factor of consideration as it could potentially affect the stresses experienced. The thicker the graft thickness, the lower the magnitude of the Von Mises stress experienced and the more rigid the overall anastomosis would be.

The results of the three-dimensional modelling of an end-to-side anastomosis show that, from a mechanical stress analysis point of view, a larger anastomotic angle is desirable because it would result in lower stresses when measured in the radial, transverse and longitudinal directions. The resultant principal stresses experienced by the host and the graft also indicate a larger anastomotic angle as the preferred model because of lower stresses generated. Thus, analyses of these two modes of stresses recommend the 60-degree model as the preferred choice. When consideration is given to the wall shear stresses, the 30-degree model would be preferred as the chances of developing intimal hyperplasia is the lowest amongst the three models considered.

Based on the results of the study, the authoress recommends having an angle of 60 degrees as the anastomotic angle. This is because the 60-degree model would result in minimal deformations and stresses and would minimise implications when blood is restored into the vessels after the bypass grafting.

Though still in rudimentary form, it is intended that the analyses performed in this study will provide useful information to the cardiac surgeons and other researchers in this same area, in helping them identify the most optimal geometries and dimensions that will result in minimal stresses on the blood vessels during the suturing process.

CHAPTER 8 RECOMMENDATIONS FOR FUTURE WORK

The work that has been conducted marks the first step towards a more thorough study in the assessment of sleeve techniques for coronary artery bypass grafting. Future work that may be considered can include the following:

8.1 Incorporation of other variables for three-dimensional analyses of an End-to-Side Anastomosis

Currently, the variable that was considered in the three-dimensional analyses of an end-to-side anastomosis is the variation in angle subtended between the host and graft. Going forward, other variables that may be considered can include:

- Variation in the geometries of the graft – This includes variations in the graft thicknesses and material properties such as Young's Modulus and Poisson's ratio;
- Variation in the properties of different grafts – In the regular discussions with the surgeon and other members of the heart surgery bypass group, it was found that possible grafts include the saphenous vein grafts, the internal mammary artery and the radial artery. The effects of using each of these types of grafts can be studied in greater detail;
- Variations in element types – Currently, all finite element models are meshed using solid elements. As the thicknesses of the blood vessel walls investigated are relatively small, the effects of using other element types – such as shell elements – should also be studied. In addition, the properties of the blood vessels may not be isotropic in nature. It is also therefore appropriate to consider using viscoelastic elements for the analyses.

8.2 Incorporation of Suture Lines

In the analyses that were conducted so far, it is assumed that the anastomosis is “perfect”. This means that there is no irregularity in the region of suture and there is no residual stress after. If suture lines are incorporated in the models, the effects of the following can be investigated:

- Thickness of the suture material – It is thought that a thinner suture results in a smaller deformation and creates less stress in the anastomosis as compared to the thicker suture. This can be verified;
- Optimal number of sutures required for stress minimisation;
- Comparison between running sutures (i.e. continuous, using one strand of thread and one resulting knot) and interrupted sutures (one thread per stitch, and each suture results in one knot formed), in terms of potential stresses experienced in the region.

Some guidelines for the “Perfect” anastomosis (provided by the surgeon) are:

- There should not be unnecessary sutures;
- The region of suture should be sealed tight enough and yet not pucker
- The radial stress should be minimal / optimal (As suture actually “punctures” the affected artery and graft)

The surgeon routinely puts 12 sutures for a coronary artery bypass. The optimality of this procedure remains to be tested.

8.3 Incorporation of Fluid Flow Effects

The results of the three-dimensional modelling of an end-to-side anastomosis show that by comparing the component (radial, transverse and longitudinal) and principal stresses, models of larger anastomotic angle are preferred since they give rise to lower magnitudes of component and principal stresses. However, analyses of the inner wall shear stresses of the models indicate that models with smaller anastomotic angle are more desirable as it experiences

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higher wall shear stresses which will result in lower chance of developing intimal hyperplasia. A smaller anastomotic angle will also result in more optimal fluid flow being generated.

These conflicting arguments from the mechanical stress analysis and fluid flow analysis point of view suggest that in order to conclude the most optimal anastomotic angle, further investigations would need to be carried out by combining the results of the mechanical modelling and analyses with the effects of fluid flow as undertaken by the other members of the Project Group.

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GLOSSARY

Anastomosis	A surgical communication made between vessels.
Angina	Chest pain that occurs secondary to the inadequate delivery of oxygen to the heart muscle. Often described as a heavy or squeezing pain in the mid-sternal area of the chest
Atherosclerosis	Chronic disease characterised by abnormal thickening of the walls of the arteries. In atherosclerosis, fatty deposits of cholesterol form on arterial inner walls. These thicken, forming plaques that narrow the vessel channel (lumen) and impede blood flow. Scarring and calcification make the walls less elastic, raising blood pressure. Eventually, plaques may completely block a lumen, or a blood clot (thrombus) may obstruct a channelled flow
Atherogenesis	Formation of fatty deposits in the arteries, the origin of atherosclerosis
Autogenous	Originating or derived from sources within the same individual.
Coronary arteries	Blood vessels that supply oxygen-rich blood to the heart muscle.
Epicardium	The inner, serious portion of the pericardium that is in contact with the heart.
Endarteritis	Inflammation of the lining of an artery.
Endarteritis obliterans	Endarteritis, particularly of small arteries, accompanied by degeneration of the intima, leading to the occlusion of the blood vessel.
Endothelium	The epithelial layer of cells lining the heart and vessels of the circulatory system.
Femoral artery	The principal artery of the thigh; originates as a continuation of the external iliac artery.

Hemodynamics	A branch of physiology concerned with circulatory movements of the blood and the forces involved in circulation.
Hyperplasia	Increase in cell number causing an increase in the size of a tissue or organ.
Intimal Hyperplasia	Accumulation of new tissue within the artery wall. It is a response to injury and a complication of all forms of arterial reconstruction. Intimal hyperplasia followed by stenosis or restenosis is the principal cause for failure in vein and synthetic grafts and in stented atherosclerotic arteries.
Ischemia	Conditions in which there is not enough oxygen-rich blood supply to the heart muscle to meet the heart's needs.
Ligature	A cord or thread used for tying vessels.
Lumen	Cavity or channel within a tube or tubular organ
Patency	The state of being freely open or exposed
Pericardium	The membranous sac that envelopes the heart.
Popliteal artery	A continuation of the femoral artery in the posterior position of the thigh above the popliteal space and below the buttock.
Postulate	Any of the assumptions upon which a mathematical theory is based. Also known as an Axiom.
Prosthesis	An artificial substitute for a missing part of a body.
Restenosis	Recurrence of stenosis after corrective surgery of the heart valve; narrowing of a structure (usually a coronary artery) following the removal or reduction of a previous narrowing
Rheology	The study of the deformation and flow of matter
Stenosis	Constriction or narrowing, as of the heart or blood vessels.
Thrombosis	Formation of a blood clot (thrombus) in the heart or

Thrombus

a blood vessel. Contributing factors include injury to a blood vessel's lining from inflammation (thrombophlebitis) or atherosclerosis, blood flow that is turbulent (e.g. From an aneurysm) or sluggish (e.g. From a prolonged blood clot) or coagulation abnormalities (e.g. from high number of platelets or excessive fats in the blood)

A blood clot occurring on the wall of a blood vessel where the endothelium is damaged.

APPENDIX I – MECHANICS OF BLOOD VESSELS

In this section, we are concerned with the mechanical behaviour of blood vessels under static loading conditions and the methods required to analyze this behaviour. The assumptions underlying this discussion are for *ideal* blood vessels that are at least regionally homogeneous, incompressible, elastic, and cylindrically orthotropic. Although physiologic systems are *non-ideal*, much understanding of vascular mechanics has been gained through the use of methods based upon these ideal assumptions.

I.1 Vascular Anatomy

A blood vessel can be divided anatomically into three distinct cylindrical sections when viewed under the optical microscope. Starting at the inside of the vessel, they are the intima, the media, and the adventitia. These structures have distinct functions in terms of the blood vessel physiology and mechanical properties.

The intima consists of a thin monolayer of endothelial cells that line the inner surface of the blood vessel. The endothelial cells have little influence on blood vessel mechanics but do play an important role in hemodynamics and transport phenomena. Because of their anatomical location, these cells are subjected to large variations in stress and strain as a result of pulsatile changes in blood pressure and flow.

The media represents the major portion of the vessel wall and provides most of the mechanical strength necessary to sustain structural integrity. The media is organized into alternating layers of interconnected smooth muscle cells and elastic lamellae. There is evidence of collagen throughout the media. These small collagen fibers are found within the bands of smooth muscle and may participate in the transfer of forces between the smooth muscle cells and the elastic lamellae. The elastic lamellae are composed principally of the fibrous

protein elastin. The number of elastic lamellae depends upon the wall thickness and the anatomical location (Wolinsky and Glagov, 1969). In the case of the canine carotid, the elastic lamellae account for a major component of the static structural response of the blood vessel (Dobrin and Canfield, 1984). This response is modulated by the smooth-muscle cells, which have the ability to actively change the mechanical characteristics of the wall (Dobrin and Rovick, 1969). The adventitia consists of loose, more disorganized fibrous connective tissue, which may have less influence on mechanics.

Figure I.1 Cylindrical geometry of a blood vessel: *top*: stress-free reference configuration; *middle*: fully retracted vessel free of external traction; *bottom*: vessel in situ under longitudinal tether and internal pressurization.

I.2 Axisymmetric Deformation

In the following discussion we will concern ourselves with deformation of cylindrical tubes, see Figure I.1. Blood vessels tend to be nearly cylindrical in situ and tend to remain cylindrical when a cylindrical section is excised and studied in vitro. Only when the vessel is dissected further does the geometry begin to deviate from cylindrical. For this deformation there is a unique coordinate mapping

$$(R, \Theta, Z) \rightarrow (r, \theta, z) \quad (\text{I.1})$$

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where the un-deformed coordinates are given by (R, Θ, Z) and the deformed coordinates are given by (r, θ, z) . The deformation is given by a set of restricted functions

$$\begin{aligned} r &= r(R) \\ \theta &= \beta \Theta \\ z &= \mu Z + C_1 \end{aligned} \quad (I.2)$$

where the constants μ and β have been introduced to account for a uniform longitudinal strain and a symmetric residual strain that are both independent of the coordinate Θ .

If $\beta = 1$, there is no residual strain. If $\beta \neq 1$, residual stresses and strains are present. If $\beta > 1$, a longitudinal cut through the wall will cause the blood vessel to open up, and the new cross-section will form a *c*-shaped section of an annulus with larger internal and external radii. If $\beta < 1$, the cylindrical shape is unstable, but a thin section will tend to overlap itself. In Choung and Fung's (1986) formulation, $\beta = \pi / \Theta_0$, where the angle Θ_0 is half the angle spanned by the open annular section.

For cylindrical blood vessels there are two assumed constraints. The first assumption is that the longitudinal strain is uniform through the wall and therefore

$$\lambda_z = \mu = \text{a constant} \quad (I.3)$$

for any cylindrical configuration. Given this, the principal stretch ratios are computed from the above function as

$$\begin{aligned} \lambda_r &= \frac{dr}{dR} \\ \lambda_\theta &= \beta \frac{r}{R} \\ \lambda_z &= \mu \end{aligned} \quad (I.4)$$

The second assumption is wall incompressibility, which can be expressed by

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$$\lambda_r \lambda_\theta \lambda_z = 1 \quad (I.5)$$

or

$$\beta\mu \frac{r}{R} \frac{dr}{dR} = 1 \quad (I.6)$$

and therefore

$$rdr = \frac{1}{\beta\mu} R dR \quad (I.7)$$

Integration of the expression yields the solution

$$r^2 = \frac{1}{\beta\mu} R^2 + C_2 \quad (I.8)$$

where

$$C_2 = r_e^2 - \frac{1}{\beta\mu} R_e^2 \quad (I.9)$$

As a result, the principal stretch ratios can be expressed in terms of R as follows:

$$\lambda_r = \frac{1}{\sqrt{\beta\mu(R^2 + \beta\mu C_2)}} \quad (I.10)$$

$$\lambda_\theta = \sqrt{\frac{1}{\beta\mu} + \frac{C_2}{R^2}} \quad (I.11)$$

I.3 Experimental Measurements

The basic experimental setup required to measure the mechanical properties of blood vessels in vitro is described in Dobrin and Rovick (1969). It consists of a temperature-regulated bath of physiologic saline solution to maintain immersed cylindrical blood vessel segments, devices to measure diameter, an apparatus to hold the vessel at a constant longitudinal extension and to measure longitudinal distending force, and a system to deliver and control the

internal pressure of the vessel with 100% oxygen. Typical data obtained from this type of experiment are shown in Figures I.2 and I.3.

I.4 Equilibrium

When blood vessels are excised, they retract both longitudinally and circumferentially. Restoration to natural dimensions requires the application of internal pressure p_i , and a longitudinal tether force, F_T . The internal pressure and longitudinal tether are balanced by the development of forces within the vessel wall. The internal pressure is balanced in the circumferential direction by a wall tension, T . The longitudinal tether force and pressure are balanced by the retractive force of the wall, F_R .

$$T = p_i r_i \quad (I.12)$$

$$F_R = F_T + \pi p_i r_i^2 \quad (I.13)$$

Figure I.2 Pressure-radius curves for the canine carotid artery at various degrees of longitudinal extension.

Figure I.3 Longitudinal distending force as a function of radius at various degrees of longitudinal extension.

The first equation is the familiar law of Laplace for a cylindrical tube with internal radius r_i . It indicates that the force due to internal pressure, p_i , must be balanced by a tensile force (per unit length), T , within the wall. This tension is the integral of the circumferentially directed force intensity (or stress, σ_θ) across the wall:

$$T = \int_{r_i}^{r_e} \sigma_\theta dr = \overline{\sigma_\theta} h \quad (I.14)$$

where $\overline{\sigma_\theta}$ is the mean value of the circumferential stress and h is the wall thickness. Similarly, the longitudinal tether force, F_T , and extending force due to internal pressure are balanced by a retractive internal force, F_R , due to axial stress, σ_z , in the blood vessel wall:

$$F_R = 2\pi \int_{r_i}^{r_e} \sigma_z r dr = \overline{\sigma_z} \pi h (r_i + r_e) \quad (I.15)$$

where $\overline{\sigma_z}$ is the mean value of this longitudinal stress. The mean stresses are calculated from the above equation as:

$$\overline{\sigma_{\theta}} = P_i \frac{r_i}{h} \quad (I.16)$$

$$\overline{\sigma_z} = \frac{F_T}{\pi h(r_e + r_i)} + \frac{P_i r_i}{2 h} \quad (I.17)$$

The mean stresses are a fairly good approximation for thin-walled tubes where the variations through the wall are small. However, the range of applicability of the thin-wall assumption depends upon the material properties and geometry. In a linear elastic material, the variation in σ_{θ} is less than 5% for $r/h > 20$. When the material is nonlinear or the deformation is large, the variations in stress can be more severe (see Figure I.10).

The stress distribution is determined by solving the equilibrium equation,

$$\frac{1}{r} \frac{d}{dr} (r \sigma_r) - \frac{\sigma_{\theta}}{r} = 0 \quad (I.18)$$

This equation governs how the two stresses are related and must change in the cylindrical geometry. For uniform extension and internal pressurization, the stresses must be functions of a single radial coordinate, r , subject to the two boundary conditions for the radial stress:

$$\sigma_r(r_i, \mu) = -p_i \quad (I.19)$$

$$\sigma_r(r_e, \mu) = 0 \quad (I.20)$$

I.5 Strain Energy Density Functions

Blood vessels are able to maintain their structural stability and contain steady oscillating internal pressures. This property suggests a strong elastic component, which has been called the *pseudoelasticity* (Fung et. al., 1979). This elastic response can be characterized by a single potential function called the *strain energy density*. It is a scalar function of the strains that determines the amount of stored elastic energy per unit volume.

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In the case of a cylindrically orthotropic tube of incompressible material, the strain energy density can be written in the following functional form:

$$W = W^*(\lambda_r, \lambda_\theta, \lambda_z) + \lambda_r \lambda_\theta \lambda_z p \quad (I.21)$$

where p is a scalar function of position, R . The stresses are computed from the strain energy by the following:

$$\sigma_i = \lambda_i \frac{\partial W^*}{\partial \lambda_i} + p \quad (I.22)$$

We make the following transformation (Chu and Oka, 1973)

$$\lambda = \frac{\beta r}{\sqrt{\beta \mu (r^2 - C_2)}} \quad (I.23)$$

which upon differentiation gives

$$r \frac{d\lambda}{dr} = \beta^{-1} (\beta \lambda - \mu \lambda^3) \quad (I.24)$$

After these expressions and the stresses in terms of the strain energy density function are introduced into the equilibrium equation, we obtain an ordinary differential equation for p :

$$\frac{dp}{d\lambda} = \frac{\beta W_{,\lambda\theta}^* - W_{,\lambda r}^*}{\beta \lambda - \mu \lambda^3} - \frac{dW_{,\lambda r}^*}{d\lambda} \quad (I.25)$$

subject to the boundary conditions:

$$p(R_i) = p_i \quad (I.26)$$

$$p(R_e) = 0 \quad (I.27)$$

Isotropic Blood Vessels

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A blood vessel generally exhibits anisotropic behaviour when subjected to large variations in internal pressure and distending force. When the degree of anisotropy is small, the blood vessel may be treated as isotropic. For isotropic materials it is convenient to introduce the strain invariants:

$$I_1 = \lambda_r^2 + \lambda_\theta^2 + \lambda_z^2 \quad (I.28)$$

$$I_2 = \lambda_r^2 \lambda_\theta^2 + \lambda_\theta^2 \lambda_z^2 + \lambda_z^2 \lambda_r^2 \quad (I.29)$$

$$I_3 = \lambda_r^2 \lambda_\theta^2 \lambda_z^2 \quad (I.30)$$

These are measures of strain that are independent of the choice of coordinates. If the material is incompressible:

$$I_3 = 1 \quad (I.31)$$

And the strain energy density is a function of the first two invariants, then

$$W = W(I_1, I_2) \quad (I.32)$$

The least complex form for an incompressible material is the first-order polynomial, which was first proposed by Mooney to characterize rubber:

$$W^* = \frac{G}{2} [(I_1 - 3) + k(I_2 - 3)] \quad (I.33)$$

It involves only two elastic constants. A special case, where $k = 0$, is the neo-Hookean material, which can be derived from thermodynamics principles for a simple solid. Exact solutions can be obtained for the cylindrical deformation of a thick-walled tube. In the case where there is no residual strain, we have the following:

$$p = -G(1 + k\mu^2) \left[\frac{\log \lambda}{\mu} + \frac{1}{2\lambda^2 \mu^2} \right] + C_0 \quad (I.34)$$

$$\sigma_r = G \left[\frac{1}{\lambda^2 \mu^2} + k \left(\frac{1}{\mu^2} + \frac{1}{\lambda^2} \right) \right] + p \quad (I.35)$$

$$\sigma_\theta = G \left[\lambda^2 + k \left(\frac{1}{\mu^2} + \lambda^2 \mu^2 \right) \right] + p \quad (I.36)$$

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Figure I.4 Pressure-radius curves for a Mooney-Rivlin tube with the approximate dimensions of the carotid.

Figure I.5 Longitudinal distending force as a function of radius for the Mooney-Rivlin tube.

$$\sigma_z = G[\mu^2 + k(\lambda^2 \mu^2 + \frac{1}{\lambda^2})] + p \quad (I.37)$$

However, these equations predict stress softening for a vessel subjected to internal pressurization at fixed lengths, rather than the stress stiffening

observed in experimental studies on arteries and veins (see Figures I.4 and I.5).

An alternative isotropic strain energy density function which can predict the appropriate type of stress stiffening for blood vessels is an exponential where the arguments is a polynomial of the strain invariants.

The first-order form is given by:

$$W^* = \frac{G_0}{2k_1} \exp[k_1(I_1 - 3) + k_2(I_2 - 3)] \quad (I.38)$$

Figure I.6 Pressure-radius curves for tube with the approximate dimensions of the carotid calculated using an isotropic exponential strain energy density function.

Figure I.7 Longitudinal distending force as a function of radius for the isotropic tube.

This requires the determination of only two independent elastic constants. The third, G_0 , is introduced to facilitate scaling of the argument of the exponent (see Figures I.6 and I.7). This exponential form is attractive for several reasons. It is a natural extension of the observation that biologic tissue stiffness is proportional to the load in simple elongation. This stress stiffening has been attributed to a statistical recruitment and alignment of tangled and disorganized long chains of proteins. The exponential forms resemble statistical distributions derived from these same arguments.

Anisotropic Blood Vessels

Studies of the orthotropic behaviour of blood vessels may employ polynomial or exponential strain energy density functions that include all strain terms or extension ratios. In particular, the strain energy density function can be of the form:

$$W^* = q_n(\lambda_r, \lambda_\theta, \lambda_z) \quad (I.39)$$

or

$$W^* = e^{q_n(\lambda_r, \lambda_\theta, \lambda_z)} \quad (I.40)$$

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Figure I.8 Pressure-radius curves for a fully orthotropic vessel calculated with an exponential strain energy density function.

Figure I.9 Longitudinal distending force as a function of radius for the orthotropic vessel.

Where q_n is a polynomial of order n . Since the material is incompressible, the explicit dependence upon λr can be eliminated either by substituting $\lambda_r = \lambda_\theta^{-1} \lambda_z^{-1}$ or by assuming that the wall is thin and hence that the contribution of these terms is small. Figures I.8 and I.9 illustrate how well the experimental

data can be fitted to an exponential strain density function whose argument is a polynomial of order $n = 3$.

Care must be taken to formulate expressions that will lead to stresses that behave properly. For this reason it is convenient to formulate the strain energy density in terms of the Lagrangian strains

$$e_i = 1/2(\lambda_i^2 - 1) \quad (I.41)$$

and in this case we can consider polynomials of the lagrangian strains, $q_n(e_r, e_\theta, e_z)$.

Vaishnav et al. (1972) proposed using a polynomial of the form

$$W^* = \sum_{i=2}^n \sum_{j=0}^i a_{ij-i} e_\theta^{i-j} e_z^j \quad (I.42)$$

to approximate the behaviour of the canine aorta. They found better correlation with order-three polynomials over order-two, but order-four polynomials did not warrant the addition work. Later, Fung et al. (1979) found very good correlation with an expression of the form

$$W = \frac{C}{2} \exp[a_1(e_\theta^2 - e_z^{*2}) + a_2(e_z^2 - e_\theta^{*2}) + 2a_4(e_\theta e_z - e_\theta^* e_z^*)] \quad (I.43)$$

for the canine carotid artery, where $e^*\theta$ and e^*z are the strains in a reference configuration at in situ length and pressure.

Why should this work? One answer appears to be related to residual stresses and strains. When residual stresses are ignored, large-deformation analysis of thick-walled blood vessels predicts steep distributions in σ_θ and σ_z through the vessel wall, with the highest stresses at the interior. This prediction is

considered significant because high tensions in the inner wall could inhibit vascularization and oxygen transport to vascular tissue.

When residual stresses are considered, the stress distributions flatten considerably and become almost uniform at in situ length and pressure. Figure I.10 shows the radial stress distributions computed for a vessel with $\beta = 1$ and $\beta = 1.11$. Takamizawa and Hayashi (1987) have even considered the case where the strain distribution is uniform in situ. The physiologic implications are that vascular tissue is in a constant state of flux. New tissue is synthesized in a state of stress that allows it to redistribute the internal loads more uniformly. There probably is no stress-free reference state. Continuous dissection of the tissue into smaller and smaller pieces would continue to relieve residual stresses and strains (Vassoughi, 1992).

Figure I.10 Stress distributions through the wall at various pressures for the orthotropic vessel.