CHAPTER 1

INTRODUCTION

1.1 Importance of Studies on the Mechanics of Blood Vessels

It is now a well-established fact that cardiovascular diseases account for more than 50% of the total number of deaths. A medical statistics shows that in over 80% of such deaths, diseases of blood vessels e.g.atherosclerosis, atherogenesis, atheroma etc. are the leading causes. In order to have a fuller understanding of the development of these diseases, an accurate knowledge of the mechanical properties of blood vessels is indispensable. The relevant informations are very much helpful to medical persons for the treatment of vascular diseases as also to the bioengineers who are engaged in the design and construction of improved artificial organs.

One of the objectives of this domain of mechanics is to develop certain mathematical laws based on which the constitutive equations for various bio-materials are derived. These constitutive relations play a very important role as in the case of engineering materials, in studying the mechanical behaviour of all biological tissues in general and vascular tissues, in particular.

For the sake of a systematic discussion on the mechanics of blood and that of the vessel walls, a brief description of the circulation of blood, the constituents of blood and those of the vessel material structures and functions of the vessel wall and those of the constituents of blood as also the mechanical properties of blood and the vessel walls, is presented in the following sections.

1.2 Structure and Function of Blood Vessels

Blood vessels are nothing but a system of pipes conveying blood to all parts of the body. There are several types of blood vessels viz. arteries, arterioles, veins, capillaries, etc. The arteries are those blood vessels which carry blood away from the heart. They consist mainly of three layers (cf. Fig.1.1, P.9) - i) the innermost layer called the tunica intima consists of a thin layer (of thickness 0.5 - 1 μm) of endothelial cells typically separated from their neighbours by narrow oblique clefts (10 - 20 μm in continuous capillaries). ii) The boundary of the middle layer, the tunica media begins from a thin layer of connective tissues which is lying below the endothelium. Tunica media is separated from the intima by a prominent layer of elastic tissues. This contains a high amount of collagen, smooth muscle fibres and elastin. iii) The outermost layer, called the adventitia, is made of fibrous tissues and contains a high proportion of collagen. Large arteries are more elastic because of the presence of relatively large amount of elastic tissues in their walls. For smaller arteries e.g. the arterioles, smooth muscle fibres are more prominent.

Veins are the blood vessels which carry blood to the heart. The venous cross-sectional area at any point is larger than that of arteries, and the velocity of blood flow is correspondingly lower. The tunica media of vein is relatively thin and weak containing less muscular and elastic tissues. When the arterioles break up into minute vessels, they are termed as capillaries. Capillaries have very thin layers of endothelial cells through which only water and substances of small molecular size can pass.

Since collagen, elastin and smooth muscles are the principal constituents of vascular walls a brief discussion on these is given below.

Collagen Collagen is the most important structural element of animals. Tendons and ligaments contain 75% collagen. Also there is a high amount of collagen present in the bone materials. Collagen is a relatively inextensible fibrous protein. These fibres always appear as a tightly bound triple polypeptide chain. They can only be identified by light or electron microscopy. Collagen is deposited in large amounts during growth. Its turnover is very slow for nongrowing tissues. Uptil now very little is known about the anatomy of collagen.

Elastin Unlike collagen, elastin is an extensible fibrous protein present in large amount in skin, blood vessels, bladder, lung etc. The elastic behaviour of these structures is solely due to the presence of elastin. Elastin has polypeptide chain but they are tangled and elongated. It can be stained for visualization by light microscopy. The fact that elastin never appears without collagen, leads us to think that there must be resemblance in structures of both. The Young's modulus of elastin was calculated by Burton (1954) to be about 3 to 8 x 10^6 dynes/cm^2.

<u>Smooth Muscle</u> Muscles consist of many fibres held together by connective tissues. Their structure and function vary widely in different organs and animals. On the basis of structure they are divided into smooth and striated muscle. One type of smooth muscle has spindle-shaped fibres which are 4 to 7 μ (micron) thick and rarely longer than than 0.2 mm. Microscopically they are nearly homogeneous except for a single nucleus but electron microscope has shown that they are filled with longitudinal filaments. These types of muscles are found in the walls of blood vessels of vertebrates. Another type of smooth muscle, observed only in invertebrates, contains large fibres with microscopically visible fibrils, often located near the surface of the fibres.

1.3 Mechanical Properties of Blood Vessels

To study the mechanics of blood vessels, one must have a thorough knowledge of the mechanical properties of the vascular walls. A number of theoretical and experimental investigations in the relevant field have established that the vascular walls are nonhomogeneous, anisotropic, visco-elastic and non-linear [cf. Gow and Taylor (1968), Anliker et al. (1968), Patel et al. (1969), Fung et al. (1979), Vaishnav et al. (1978), Young et al. (1977), Hardung (1964)].

Inhomogeneity As discussed above, the walls of blood vessels are inhomogeneous. But experimental investigations made by Simon et al. (1971) showed that the outermost layer adventitia has a very loose network and merges externally with the surrounding tissues and so a large portion of it is removed for the experiments. The innermost layer tunica intima is very thin (almost one or two cells thick) and can easily be neglected. The remaining media, the tunica media is considered homogeneous containing a matrix of smooth muscle elastin and collagen fibres.

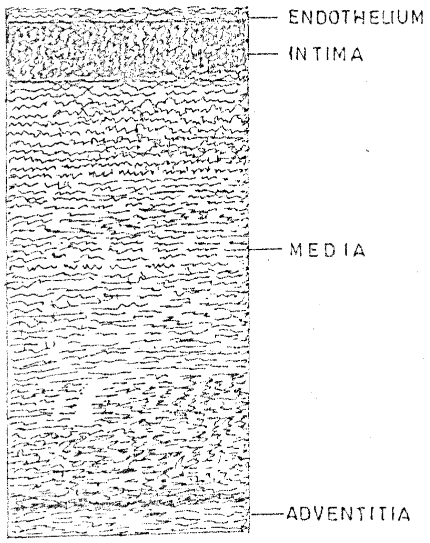
Compressibility of Vascular Tissues A material is said to be compressible if it changes its volume appreciably when subjected to stress; it is said to be incompressible if the change in volume is ignorable. Harkness et al.(1957), observing the high water content of the vessel wall, suggested that the assumption of incompressibility can safely be considered for wall tissues. His theory was further confirmed by Carew, Vaishnav and Patel (1968) who experimentally determined the bulk modulus and found that the bulk modulus of the vascular tissue is orders of magnitude higher than the shear modulus, under physiological state of strain. However, Tickner and Sacks (1967) in their experimental studies on the blood vessel walls observed 20-40% changes in volume. In any case, for practical purposes, the compressibility of vascular tissues can be considered to be small.

Anisotropy Although the compressibility of blood vessel walls is small, the vessel walls elongate when they are inflated. This phenomenon was studied by Fenn (1957) and was further supported by McDonald (1974), Tucker et al. (1969) who made direct experiments on vascular tissues. This particular behaviour of the vascular wall confirms the idea that they are anisotropic. Patel et al. (1969) in his experimental investigations showed that simultaneous axial tension and pressurization of arteries result in negligible shearing strains. This indicates that the arterial walls have elastic symmetry with respect to cylindrical co-ordinates i.e. orthotropy. The transverse isotropy nature of the vascular walls was not ruled out by Patel et al. (1969). For a general anisotropic material, the number of material constants is quite large. But the assumption of orthotropicity togetherwith incompressibility brings the problem in a tractable form, since the assumptions reduce the number of the material constants to a considerable extent. Various studies with those assumptions have been made in the past by many investigators e.g. Carew et al. (1968), Patel et al. (1969), Vaishnav et al. (1972), Young et al. (1977) and others.

Viscoelasticity For a perfectly elastic body there must be a single valued relationship between the applied strain and resulting stress. But when an artery is subjected to a cyclically varying strain the stress response exhibits a hysteresis loop for each cycle (Fig.1.2). The rate of decrease is very rapid in the beginning, but a steady state is observed after a number of cycles. This phenomenon was observed by Fung et al (1979). Moreover two main characteristics of a visco-elastic material e.g. creep and stress relaxation were also observed in vascular tissues by many investigators. Stacy (1957) found that after a sudden stretch, tension of a vascular wall rises and decays towards some final value, stress relaxation. Wiederhielm (1965) observed that there is a continuous deformation (creep) after a sudden change in tension. These observations lead one to conclude that the walls of blood vessels are visco-elastic.

1.4 Circulation of Blood

The circulatory system (Fig.1.3) of the body is mainly dependent on two organs. One is the heart which acts as a pump and the other is the system of blood vessels through



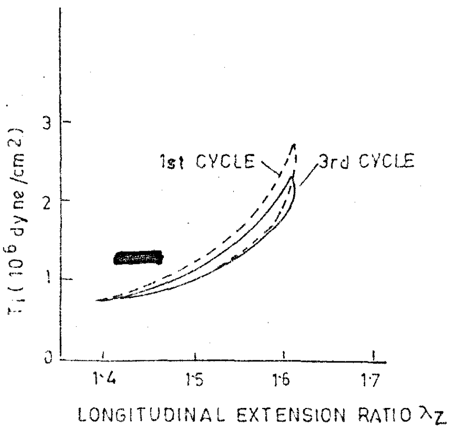


FIG. 1·1. STRUCTURE OF A BLOOD VESSEL WALL. (CF. W. Bloom and D.W. Fawcett, 1968)

FIG. 1·2. TYPICAL LOADING - UNLOADING CURVES OF ARTERIAL WALLS

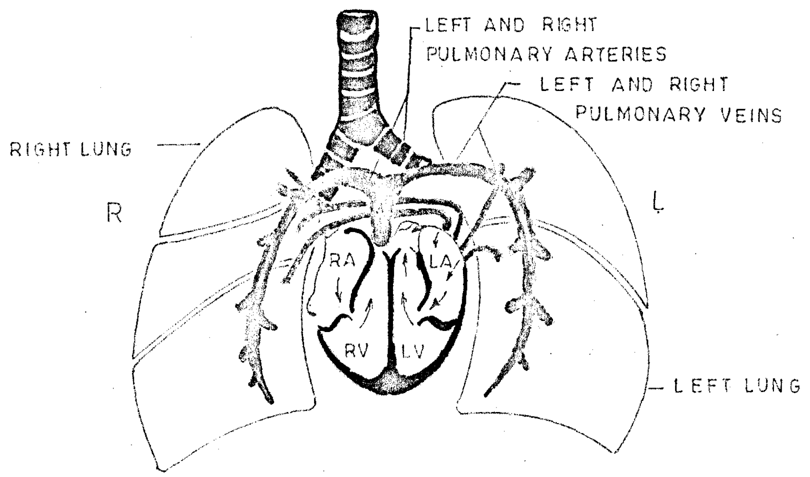


FIG. 1·3. SCHEMATIC DIAGRAM OF PULMONARY CIRCULATION which the blood is circulated throughout the body. From the right ventricle of the heart, the deoxygenated blood is carried by pulmonary artery and is passed upwards through the walls of the heart. The pulmonary artery has two branches, viz the left pulmonary artery and the right pulmonary artery. The left one with deoxygenated blood is connected to the left lung while the right one is connected to the right lung. Within the lung they divide and subdivide forming smaller arteries, capillaries etc. After some interchange of gases in the lung tissue and the capillaries the later containing oxygenated blood ultimately join up and form two veins. From each lung altogether four pulmonary veins carrying oxygenated blood, come out and enter the left atrium of the heart. During arterial systole or contraction of atria, the blood goes to the left ventricle and during ventricular systole or contraction of ventricle, the blood is pumped out and passed through the aorta together with its branches to all parts of the body.

1.5 Constituents of Blood

The composition of blood is very closely related to the functions and activities of the organs it is supplying. In a body the cells are always adding to and subtracting from the constituents of blood, but there is certain limit within which the changes can take place.

The blood is believed to be composed of a complex aequous continuous phase with numerous cells or corpuseles of different kinds suspended in it. This continuous phase is called plasma which is about 55 to 60% of the total volume of blood. The suspended particles consist of a variety of cells, the red cells or erythrocytes, the white cells or leucocytes and platelates or thrombocytes.

The plasma contains water (90-92%), proteins, nutrient-materials, organic waste products, hormones, enzymes etc. The red cells occupy 97% of the total cell volume. They consist of thin flexible but unstretchable membranes with a complex aequous solution inside. This complex solution is called haemoglobin solution.

1.6 Structure and Function of the Constituents of Blood

The erythrocytes are described to be circular, biconcave discs with diameter of about 7 microns and thickness of about 2 microns. The central parts of these cells are much thinner than the circumference. Haemoglobin is a complex protein, one of its main constituents is iron. Leucocytes or white cells are very fewer in number but larger in size. Their sizes vary from 8 to 15 microns. There are 6,000 to 10,000 white cells per cubic millimeter of blood. Platelates are very minute and much smaller than all the remaining cells in the blood. There are about 300,000 platelates in a cubic millimeter of blood.

The oxygenated blood is transported from the lungs to different parts of the body. This transportation is carried out by haemoglobin present inside the erythrocytes. Many nutrient materials e.g. glucose, vitamins etc. dissolved in plasma are carried by the blood to the blood cells for maintaining the constant functioning of the body cells. The plasma proteins give viscosity to the blood and thus help to maintain the blood pressure. When blood comes out of the body due to some rupture in the blood vessels, the body reacts to prevent further blood loss. This is a very complicated process and is known as clotting of blood. The platelates suspended in plasma, as well as the plasma proteins play an important role in the clotting of blood.

1.7 Mechanical Properties of Blood

Human blood is generally believed to possess the behaviour of a non-Newtonian fluid. Also the rheological properties of blood seem to be history-and time-dependent. Experimental evidences show that blood has a yield stress and it is independent of temperature. The viscosity at the finite rates of deformation decreases with the increase in temperature. The temperature-dependency of the apparent viscosity, defined as the ratio of shear stress to shear rate approaches that of water with the increase in shear rates. When the square root of shear stress is plotted against the square root of shear rate for a normal red cells in plasma for various hematocrits+ at 250°C, it is found that the H = 8.25% suspension appears Newtonian i.e. constant viscosity over the entire shear rate range (cf. Cokelet,1972).

1.8 Equations of Motion Applicable to Blood Flow

In many of the previous theoretical studies, blood has been considered as a Newtonian and incompressible fluid. The assumption of incompressibility is quite valid considering the small fluid velocities (order of 25 cms/sec) with respect to acoustic velocity in blood (approximately 10^5 cms/sec). Assuming rotational symmetry (and hence using cylindrical co-ordinates designated as 'z' for the axial and 'r' for the radial co-ordinate), Witzig (1914) considered the following forms of the Navier-Stokes equations for a Newtonian and incompressible fluid free of rotational flow

$$\frac{\partial v_z}{\partial t} + v_r\frac{\partial v_z}{\partial r} + v_z\frac{\partial v_z}{\partial z} = -\frac{1}{\rho}\frac{\partial p}{\partial z} + \nu\left(\frac{\partial^2 v_z}{\partial r^2} + \frac{1}{r}\frac{\partial v_z}{\partial r} + \frac{\partial^2 v_z}{\partial z^2}\right)$$

+ The concentration of most red cells in a suspension is generally reported as the suspension volume fraction occupied by the red cells, which is called hematocrit(H).

$$\frac{\partial v_r}{\partial t} + v_r \frac{\partial v_r}{\partial r} + v_z \frac{\partial v_r}{\partial z} = -\frac{1}{\rho}\frac{\partial p}{\partial r} + \nu\left(\frac{\partial^2 v_r}{\partial r^2} + \frac{1}{r}\frac{\partial v_r}{\partial r} + \frac{\partial^2 v_r}{\partial z^2} - \frac{v_r}{r^2}\right). \qquad (1.8.1)$$

The equation of continuity reads

$$\frac{1}{r}\frac{\partial}{\partial r}(r v_r) = -\frac{\partial v_z}{\partial z}$$

where v_z, v_r are respectively the longitudinal and radial components of the velocity, t the time, p the pressure, μ the viscosity of blood, ρ the density, and $\nu(=\frac{\mu}{\rho})$ the kinematic viscosity. The magnitudes of the velocity components and their derivatives are assumed to be so small that their products can be neglected. Under these assumptions, the above set of equations become linear and are given by

$$\frac{\partial v_z}{\partial t} = -\frac{1}{\rho}\frac{\partial p}{\partial z} + \nu\left(\frac{\partial^2 v_z}{\partial r^2} + \frac{1}{r}\frac{\partial v_z}{\partial r} + \frac{\partial^2 v_z}{\partial z^2}\right)$$

$$\frac{\partial v_r}{\partial t} = -\frac{1}{\rho}\frac{\partial p}{\partial r} + \nu\left(\frac{\partial^2 v_r}{\partial r^2} + \frac{1}{r}\frac{\partial v_r}{\partial r} + \frac{\partial^2 v_r}{\partial z^2} - \frac{v_r}{r^2}\right) \qquad (1.8.2)$$

together with the continuity equation

$$\frac{1}{r}\frac{\partial}{\partial r}(r v_r) = -\frac{\partial v_z}{\partial z} \qquad (1.8.3)$$

These fluid equations have been considered by several authors to study the wave propagation through arteries, e.g. Womersley (1957), Atabek (1968), Mirsky (1967), Bulanowsky (1971) etc.

1.9 Large Deformation

Classical theory of elasticity is based fundamentally on the idea of infinitesimal strain. This theory is mainly developed from the study of deformations of hard materials. In the case of finite or large strains, this theory is no longer applicable. A historical and critical account of large elastic deformation theory was given by Truesdell et al. (1965). Soft biological tissues are capable of undergoing large deformations even in the normal physiological state. Thus vascular walls are non-Hookean [cf. Fung (1967)]. They usually deform by relatively enormous amounts, strains of 2-4 as compared to .002 or less for metals [cf. Bergel (1972)]. Some investigators e.g. Vaishnav et al. (1972,1978), Young et al. (1977) etc. studied the elastic as well as visco-elastic properties of blood vessels by using the large deformation theory.

1.10 Various Forms of Strain Energy Functions

To describe the elastic properties of a vascular wall one must know the stress-strain relation for that particular wall material. The best approach by Green and Zerna (1968) everknown for an elastic body, having been deformed finitely is to predict a suitable form of elastic potential function. For an isotropic and homogeneous body, the strain energy function is only a function of the strain-invariants. Various types of strain energy functions for isotropic materials were derived experimentally as well as theoretically by Mooney (1940), Rivlin (1948), Rivlin and Saunders (1951) and others. The strain energy function given by Mooney is

$W = C_1(I_1 - 3) + C_2(I_2 - 3)$, C_1 and C_2 are the elastic constants. Valanis and Landel (1967) presented the form

$W = \sum_{i=1}^{3} f(\ln\lambda_i)$, where λ_i are the stretch ratios. The stretch ratio is defined as the ratio of the deformed length and the original length of a body. Blatz and Chu as well as Wayland (1969) used this form of W for their study of soft tissues and predicted the form of the function as

$$f(\ln\lambda_i) = C(\lambda_i^{\alpha} - 1).$$

For a skeletal muscle the form is slightly modified as

$$f(\ln\lambda_i) = C\left[e^{\alpha(\lambda_i^2 - 1)} - 1\right],$$ where C and α are experimentally determined constants. Veranda and

Westmann (1970) proposed the following form of strain-potential

$$W = C_1\left[e^{\beta(I_1-3)} - 1\right] + C_2(I_2 - 3) + g(I_3), \quad \text{where}$$

I_1, I_2 and I_3 are strain invariants, in which C_1, C_2 and β are the constants, determined experimentally.

The strain energy function for the anisotropic vascular wall of a anesthetized dog was derived by Hayashi et al. (1975) as

$$\lambda_\theta \frac{\partial W}{\partial \lambda_\theta} = \left[A(\lambda_z^2 - 1) + 1\right] \exp B(\lambda_\theta^2 - 1) - 1$$

$$\lambda_z \frac{\partial W}{\partial \lambda_z} = \left[C(\lambda_\theta^2 - 1) + 1\right] \exp D(\lambda_z^2 - 1) - 1 \tag{1.10.1}$$

A, B, C, D are the material constants, and λ_θ , λ_z are the stretch ratios in the circumferential and longitudinal directions respectively.

The most convenient and widely used form of strain energy function for a canine aorta has been presented by Vaishnav et al. (1972). They expressed the strain energy function W as a polynomial. Imposing the condition of incompressibility together with the orthotropicity, they reduced the degree of the polynomial. The derived expressions read

$$W = A_1 a^2 + B_1 ab + C_1 b^2$$
$$W = A_2 a^2 + B_2 ab + C_2 b^2 + D_2 a^3 + E_2 a^2 b + F_2 ab^2 + G_2 b^3$$
$$W = A_3 a^2 + B_3 ab + C_3 b^2 + D_3 a^3 + E_3 a^2 b + F_3 ab^2 + G_3 b^3 + H_3 a^4 + I_3 a^3 b + J_3 a^2 b^2 \qquad (1.10.2)$$

where A_1, A_2 etc. are material constants, the values of which were experimentally determined for a canine middle discending thoracic aorta by Vaishnav and his associates (1972); in the above expressions (1.10.2), $a = E_{\theta\theta}$ and $b = E_{zz}$ are the components of strain in the circumferential and longitudinal directions respectively. Fung et al.(1979) showed that the following exponential pseudo-strain-energy function is a very convenient practical approximation to describe the inelastic behaviour of arterial walls subjected to internal pressures and longitudinal stretching in the physiologic range

$$\rho_o W^{(2)} = \frac{C}{2} \exp\left[a_1(E_{\theta\theta}^2 - E_{\theta\theta}^{*2}) + a_2(E_{zz}^2 - E_{zz}^{*2}) + 2a_4(E_{\theta\theta}E_{zz} - E_{\theta\theta}^* E_{zz}^*) \right]$$

where W is the strain energy per unit mass of the tissue, ρ_o is the mass density of the wall, C, a_1, a_2, a_4 are material constants of the arterial wall and $E_{\theta\theta}^*$, E_{zz}^* are strains corresponding to a standard pair of stresses $S_{\theta\theta}^*, S_{zz}^*$.

In the following two sections some fundamental mathematical concepts which are extensively used in the large deformation analysis, are briefly discussed. It is believed that the introduction of the following sections will be helpful for an easy access to the notations and the methods of solution of the large deformation problems presented in the subsequent chapters of the thesis.

1.11 The Base Vectors and Metric Tensors

Let us suppose that the cartesian system of coordinates x_i defines a three dimensional Eucledian space. We introduce curvilinear co-ordinates $\theta_i (i=1,2,3)$ such that

$\theta_i = \theta_i (x_1, x_2, x_3)$, in which θ_i are single valued function of x_i. Since the transformation is reversible, one can as well write

$$x_i = x_i(\theta_1, \theta_2, \theta_3).$$

Let $\bar{R}$ be the position vector of a point P(cf. Fig.1.4) with co-ordinates x_i. We consider a neighbouring point Q with co-ordinates $x_i + dx_i$. $d\bar{R}$ denotes the infinitesimal vector $\overline{PQ}$

$$d\bar{R} = \frac{\partial \bar{R}}{\partial x^r} dx^r = dx_r \hat{i}_r , \quad \text{where} \quad \hat{i} = \frac{\partial \bar{R}}{\partial x^r} .$$

Since $\bar{R}$ is also a function of $(\theta_1, \theta_2, \theta_3)$

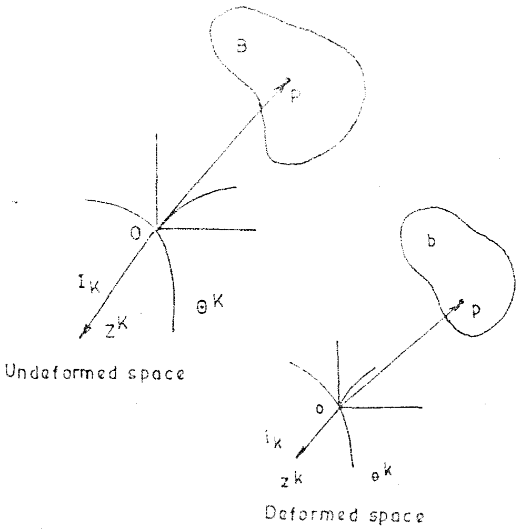


FIG. 1·5. THE DEFORMED AND UNDEFORMED MEDIA i_k UNIT BASE VECTOR ASSOCIATED WITH z^k.

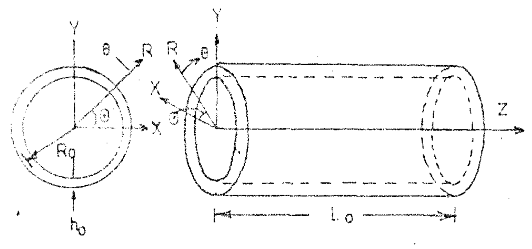


FIG. 1·6. A CYLINDRICAL BLOOD VESSEL SEGMENT IN THE UNDEFORMED STATE.

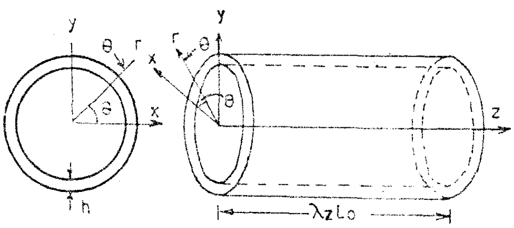


FIG. 1·7 THE BLOOD VESSEL SEGMENT IN ITS DEFORMED STATE

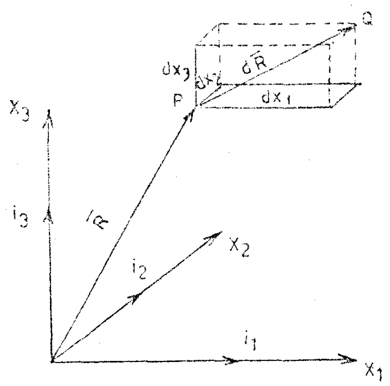


FIG. 1·4. POSITION VECTORS AND ITS DIFFERENTIAL i.e $\bar{R} = \bar{R}(\theta_1, \theta_2, \theta_3)$,

we have $d\bar{R} = \bar{g}_r \, d\theta^r = \bar{g}^r \, d\theta_r$

where $\bar{g}_r = \frac{\partial x^s}{\partial \theta^r} \hat{i}_s$, and $\bar{g}_r = \frac{\partial \theta^r}{\partial x^s} \hat{i}^s$

The inverse transformation gives

$$\hat{i}_r = \hat{i}^r = \frac{\partial x^r}{\partial \theta^s} \bar{g}^s = \frac{\partial \theta^s}{\partial x^r} \bar{g}_s .$$

The vectors $\bar{g}_r$ and $\bar{g}_s$ are called base vectors. The metric tensors g_{rs} and g^{rs} are defined as

$$g_{rs} = \bar{g}_r . \bar{g}_s , \quad g^{rs} = \bar{g}^r . \bar{g}^s .$$

1.12 Green's Deformation and Lagrangian Strain Tensors

If the rectangular and curvilinear co-ordinate systems of a representative point P (Fig. 1.5) in a body B be represented as Z_K and Θ_K respectively and those of p at time t (which was P at t = 0) be z_k and θ_k , the motion of the entire body can be specified by considering z_k as a function of Z_K and θ_k as a function of Θ_K i.e.

$$z^k = z^k(Z^K , t)$$

and

$$\theta^k = \theta^k(\Theta^K , t).$$

The square of the length of the line segment joining the points Θ_K and $\Theta^K + d\Theta^K$ is given by

$$dS^2 = G_{KL}\, d\Theta^K\, d\Theta^L$$

, where G_{KL} is the metric tensor corresponding to the co-ordinate system Θ^K and is given as

$$G_{KL} = \frac{\partial Z_N}{\partial \Theta^K} \frac{\partial Z_N}{\partial \Theta^L} .$$

The co-ordinate system θ^k is related to Θ^K by the following relation

$$d\theta^k = \frac{\partial \theta^k}{\partial \Theta^K} d\Theta^K .$$

The square of the same length referred to above, corresponding to the co-ordinate system θ^k may further be written as

$$ds^2 = g_{kl}\, dx^k\, dx^l,$$

g_{kl} being the metric tensor for the θ^k co-ordinate system.

The total strain is given as

$$ds^2 - dS^2 = (C_{KL} - G_{KL})d\Theta^K\, d\Theta^L = 2E_{KL}\, d\Theta^K d\Theta^L,$$

where

$$C_{KL} = g_{kl} \frac{\partial \theta^k}{\partial \Theta^K} \frac{\partial \theta^l}{\partial \Theta^L} .$$

C_{KL} is called the Green's strain tensor and E_{KL}, the Lagrangian strain tensor or Green-St.Venant's strain tensor.

1.13 Cylindrical Model

In most of the analytical studies on blood vessels, the vascular walls are assumed to be cylindrical. Of course, the vessel walls are not perfectly circular cylindrical segments of constant thickness, but the reduction of the mean diameter and the wall thickness vary directly with the distance from the centre in such a way that their ratio remains almost constant. Since this ratio is dominant everywhere in the formulae for the average circumferential, longitudinal and radial stresses, the assumption that the walls are cylindrical seems to be quite reasonable.

1.14 Strains for cylindrical Symmetry

Patel and Fry (1969) showed that for an orthotropic vascular wall under the physiologic state of loading, no shearing strain develops. The three normal components of strain can be obtained by considering the geometry of the underformed as well as deformed states.

In the undeformed configuration, let the arterial segment be referred to a Cartesian co-ordinate system Z_K, denoted by (X,Y,Z) and also to polar co-ordinate system of co-ordinates Θ^K, denoted by (R, Θ , Z). The two co-ordinate systems are related by (cf. Fig. 1.6)

$$
\begin{aligned}
X &= Z_1 = \Theta^1 \cos \Theta^2 = R \cos \Theta \\
Y &= Z_2 = \Theta^1 \sin \Theta^2 = R \sin \Theta \qquad (1.14.1) \\
Z &= Z_3 = \Theta^3 = Z
\end{aligned}
$$

Let the deformed configuration after a time t be referred to a cartesian system z_k, denoted by (x,y,z) and to a polar co-ordinate system θ^K, denoted by (r,θ,z). Then z^k and θ^k are related in the following manner (cf. Fig. 1.7)

$$
\begin{aligned}
x &= z_1 = \theta^1 \cos \theta^2 = r \cos \theta \\
y &= z_2 = \theta^1 \sin \theta^2 = r \sin \theta \qquad (1.14.2) \\
z &= z_3 = \theta^3 = z.
\end{aligned}
$$

The deformed segment is obtained by a uniform stretch of the undeformed segment longitudinally with a simultaneous displacement of all points radially maintaining the material continuity everywhere. For the deformed segment to remain cylindrical, the following relations must be satisfied.

$$
\begin{aligned}
r &= \theta^1 = \theta^1(\Theta^1, t) = r(R, t) \\
\theta &= \theta^2 = \Theta^2 = \Theta \qquad (1.14.3) \\
z &= \theta^3 = \lambda_z(t)\, \Theta^3 = \lambda_z(t) Z
\end{aligned}
$$

$\lambda_z(t)$ is the stretch ratio along the longitudinal direction. The deformation gradient matrix is given by

$$\frac{\partial \theta^k}{\partial \Theta^K} = \text{diag}\left[\frac{\partial r}{\partial R}, 1, \lambda_z\right]. \tag{1.14.4}$$

The formulae for G_{KL}, g_{kl}, C_{KL} and E_{KL} are respectively

$$\begin{aligned} G_{KL} &= \text{diag}\left[1, R^2, 1\right] \\ g_{kl} &= \text{diag}\left[1, r^2, 1\right] \\ C_{KL} &= \text{diag}\left[\left(\frac{\partial r}{\partial R}\right)^2, r^2, \lambda_z^2\right] \\ E_{(K)(L)} &= \text{diag}\left[\frac{1}{2}\left[\left(\frac{\partial r}{\partial R}\right)^2 - 1\right], \frac{1}{2}\left[\frac{r^2}{R^2} - 1\right], \frac{1}{2}\left[\lambda_z^2 - 1\right]\right] \end{aligned} \tag{1.14.5}$$

where $E_{(K)(L)}$ is the physical component of E_{KL} and is given by

$$E_{(K)(L)} = \sqrt{G^{KK} G^{LL}}\, E_{KL},$$ where G^{KL} is the associated metric tensor and its matrix is the reciprocal of that for G_{KL}.

1.15 Constitutive Equations

Constitutive equations are nothing but the relations between the stress and strain for a particular (elastic) body.

better: and their corresponding relationships

There are mainly two objectives for studying the constitutive equations for a biological system, viz. i) to obtain a general law for the mathematical analysis of the boundary value problem and 2) to know the specific details about the living tissues.

For Hookean materials, the stress is related to strain as $\tau_{ij} = c_{ijkl}\, e_{kl}(i,j,k,l = 1,2,3)$, τ_{ij} and e_{ij} being the stress and strain tensors and c_{ijkl} the elastic constants. For an orthotropic material, the number of constants is more but for isotropic case they reduce to only two. For a material undergoing finite deformation, the stress-strain relations may be described in terms of strain energy functions as

$$\tau^{ij} = \frac{1}{2\sqrt{I_3}} \left(\frac{\partial W}{\partial e_{ij}} + \frac{\partial W}{\partial e_{ji}} \right),$$

for a compressible body, whereas for an incompressible material, the relation assumes the form

$$\tau^{ij} = p\, G^{ij} + \frac{1}{2} \left(\frac{\partial W}{\partial e_{ij}} + \frac{\partial W}{\partial e_{ji}} \right),$$ p is the hydrostatic pressure and G^{ij} is the contravariant metric tensor.

It is well-known from the visco-elastic behaviour of the soft biological tissues that they are highly time dependent. The stress at a given point does not only depend upon the strain but also on the strain-history. For a linear visco-elastic body the one-dimensional constitutive equation may be described as

$$\sigma(t) = \int_0^t K(t - \xi) \frac{\partial e(\xi)}{\partial \xi} d\xi$$

where $K(t - \xi)$ is the kernel function, determined experimentally and σ , e are the stress and the strain. The non-linear visco-elastic constitutive law was used by Vaishnav et al. (1978) as

$$T^{kl}(t) = \frac{\partial \theta^k(t)}{\partial \Theta^K} \frac{\partial \theta^l(t)}{\partial \Theta^L} \underset{t-\tau=\infty}{\overset{o}{\underline{G}}{}^{KL}} \left[E_{MN}(t-\tau) \right],$$

where $\overset{o}{\underline{G}}{}^{KL}$ is a functional and E_{MN} is the Lagrangian strain tensor. $t - \tau$ represents the elapsed time between the present and the generic time. For an incompressible material, the strain history determines the stress only to within an arbitrary hydrostatic pressure P.

Thus

$$T^{kl}(t) = - Pg^{kl} + \frac{\partial \theta^k(t)}{\partial \Theta^K} \frac{\partial \theta^l(t)}{\partial \Theta^L} \underset{t-\tau=\infty}{\overset{o}{\underline{G}}{}^{KL}} \left[E_{MN}(t-\tau) \right] \tag{1.15.1}$$

where g^{kl} is the metric tensor for the θ^k co-ordinate system. After expanding the functional $\overset{o}{\underline{G}}{}^{KL}$, the above relation (1.15.1) takes the form

$$\tau^{kl}(t) = -pg^{kl} + \frac{\partial\theta^{k}(t)}{\partial\Theta^{K}} \frac{\partial\theta^{l}(t)}{\partial\Theta^{L}} \Big[\int_{-\infty}^{t} K^{KLMN}(t-\tau)\dot{E}_{MN}(\tau)\,d\tau$$

$$+ \int_{-\infty}^{t}\int_{-\infty}^{t} K^{KLM_1N_1M_2N_2}(t-\tau_1, t-\tau_2)\dot{E}_{M_1N_1}(\tau_1)\dot{E}_{M_2N_2}(\tau_2)$$

$$d\tau_1\, d\tau_2 \Big] \qquad (1.15.2)$$

K^{KLMN}'s are the relaxation functions. Young et al. (1977) by experimenting on the vascular segment of canine middle discending thoracic aorta, determined the values of those relaxation functions. They also modified the above relation (1.15.2) for an incompressible anisotropic artery under a physiologic state of strain to the form

$$\tau^{(k)(l)}(t) = -p\delta^{k}_{l} + \frac{\partial\theta^{k}(t)}{\partial\Theta^{K}} \frac{\partial\theta^{l}(t)}{\partial\Theta^{L}} \Big[\int_{-\infty}^{t} K^{KL22}(t-\tau)\dot{E}_{22}(\tau)\,d\tau$$

$$+ \int_{-\infty}^{t}\int_{-\infty}^{t} K^{KL2222}(t-\tau_1, t-\tau_2)\dot{E}_{22}(\tau_1)\dot{E}_{22}(\tau_2)$$

$$d\tau_1\, d\tau_2 + \cdots\cdots \Big], \qquad (1.15.3)$$

$\tau^{(k)(l)}$ is the physical component of τ^{kl}.

1.16 Equations of Motion

For the small deformations, the equations of motion of a body subjected to a body force are given by

$$\tau_{ij,j} + \rho X_i = \rho \ddot{u}_i \quad (i,\ j = 1,2,3\ ) \qquad (1.16.1)$$

where τ_{ij} are the components of the stress-tensor and X_i those of the body force, u_i are the infinitesimal displacements of the body and ρ is the density of the body; dots denote the differentiation with respect to time.

In the case of finite or large deformations, the equations of motion without body force are given by

$$\tau^{ij}\|_j = \rho f^i \quad (i,j = 1,2,3) \qquad (1.16.2)$$

where ρ is the density of the undeformed body, [redacted] f^i are the components of the accelaration vector and

$$\tau^{ij}\|_j = \tau^{ij}_{,j} + \Gamma^j_{j\lambda}\,\tau^{\lambda i} + \Gamma^i_{j\lambda}\,\tau^{\lambda j} \qquad (1.16.3)$$

comma denotes simple differentiation and Γ^i_{jk} called the christoffel symbol of 2nd kind is defined as

$$\Gamma^i_{jk} = \frac{1}{2} g^{i\lambda}\left[g_{\lambda k,j} + g_{\lambda j,k} - g_{jk,\lambda} \right], \qquad (1.16.4)$$

where g_{ij} are the metric tensors corresponding to the deformed configuration.

1.17 A Brief Account of Relevant Previous Studies

Studies on the mechanical properties of the blood vessels have become a subject of intensive research for more than a century. In this section, an attempt is made to briefly review only some of the previous experimental as well as analytical studies which are somewhat relevant to the problems considered in the present thesis.

As early as 1847, Wertheim showed that the stress-strain relations deviate from Hook's law. Charles Roy (1880) studied the elastic properties of the arterial wall. It was he who first observed that an artery expands on warming, like other materials. Experimental studies by Gow and Taylor (1968), Anliker et al. (1968b) show that vascular walls are inelastic, nonlinear and anisotropic. The investigations of Hardung (1953), Bergel (1964), Peterson et al. (1960) and Learoyd and Taylor (1966) indicate that the Young's modulus of vascular material increases as we go away from the heart and also that it is frequency-dependent. A mathematical model was proposed by Cox (1972) to represent the frequency-dependence of the mechanical properties of the artery. Roach and Burton (1957), Bergel (1961a, 1961b) considered the vascular walls to be thin walled cylinders in incrementally linear elastic models. But using such a model one cannot quantify the large strains developed in blood vessels. Some recent workers e.g. Simon et al. (1971), Vaishnav et al. (1972, 1973), Young et al. (1977), Fung et al. (1979), Wiederhielm (1965) and some others used the finite deformation theory in course of various studies on arterial walls. Vaishnav et al. (1972) developed a non-linear theory suitable for the study of vascular mechanics. They put forward three expressions, second, third and fourth degree polynomials for strain energy function on the assumption that aortic segments are anisotropic and incompressible. They experimentally determined the material constants involved in the analysis. The third degree expression containing seven material constants was recommended for general use. Fung et al. (1979) in their recent investigation, considered only the seven-constant form of the strain energy function. They postulated a new expression for the strain energy function which they consider to be the most practical approximation for an artery under a physiological state of strain. They further examined the validity of their strain energy function by performing experiments on rabbit arteries. Polynomial expressions for the strain energy function was also used by Wealey et al. (1978) in order to explain the static linear and non-linear elastic properties of human and canine venous segments. Young et al (1977) as an extension of the works (1972, 1973) presented a non-linear visco-elastic constitutive relations for studying the mechanical behaviour of blood vessel walls. They expressed the strain-rates and stresses in the circumferential and longitudinal directions in terms of integrals involving four and ten relaxation functions. On the basis of experimental findings, it was concluded that while the 10-function theory should be used for critical applications, the 4-function theory is adequate for general use. Expressions for storage and loss moduli were also obtained in terms of the relaxation functions by Vaishnav et al. (1978), Greenfield and Patel (1962) investigated the relation between the pressure and diameter for human ascending aorta. Hudetz (1979) found an expression for the incremental modulus, which in itself is sufficient to characterize the elastic stiffness of an axisymmetric blood vessel under the physiologic state of strain. The response of living small blood vessels to an applied pressure was discussed on the basis of experimental findings in the monograph of Copley (1960). Burton (1951) and Nichol (1951) studied the phenomenon of instability of the vessel walls and found some correlation between the phenomenon of stability and the critical closure of small blood vessels in the microcirculatory system. Burton (1962) by employing Laplace's law discussed the stability of equilibrium of blood vessels. It was pointed out that if the active tension was held constant and the transmural pressure was gradually reduced, the vascular walls cannot be in a state of equilibrium until and unless the transmural pressure exceeded a critical value. Azuma and Oka (1971) remarked that the law of Laplace should not be applied for discussing the mechanical equilibrium of vascular walls. They derived a more general equation and Oka et al. (1970) concluded that this general equation can be applied to almost all blood vessels, irrespective of the material or the architecture of the wall. Sharma (1974) made a study of viscoelastic behaviour of aortas in vitro through stress relaxation experiments and stress-strain experiments under various strain-rates. The properties of rabbit aorta were studied by Sharma and Hollis (1976), under control and hypertensive conditions. It was reported that below a particular strain (which certainly depends on the location of the segment within the aorta), the arterial tissue indicated pure elastic response and the material constants for hypertensive aortas differed significantly from the normotensive ones. An empirical representation of the frequency-dependent effective viscoelastic modulus of arterial walls were recently put forward by Goodman and Imaeda (1977). Taira et al. (1973) studied the stress distribution in a vascular wall by making use of the finite element technique. The phenomenon of instability of carotid and femoral arterial walls was discussed by Taira et al. (1975). Hayashi et al. (1974) examined the static mechanical behaviour of the isotropic arterial wall through the change in the external radius due to distending pressure. They further extended their study for an anisotropic model (1975). The axial mechanical properties of arterial walls and their anisotropy were studied experimentally using abdominal aortas, common carotid arteries and femoral arteries of mongrel dogs by Sato et al. (1978). An in-vitro study on the deformation of soft biological tissues was made by Hartung (1973). Further discussions on the mechanical properties of soft biological tissues with particular emphasis on the material behaviour of blood vessel walls can be found in Hartung (1975, 1976).

The studies on wave propagation through blood vessels were made by many investigators. Morgan and Kiely (1954) made an analysis of the oscillatory flow problem considering both the fluid viscosity and the viscous drag on the vascular wall. The effect of the longitudinal component of the force exerted by the surrounding tissues of the vascular wall on the wave propagation was first considered by Womersley (1957a, 1957b). Anliker et al. (1968) observed that at normal blood pressure levels the surgically exposed carotid artery of anesthetized dogs exhibits mild dispersion and strong attenuation with respect to artificial axial waves with frequencies between 25 and 150 Hz. Various possible modes of propagation of waves in blood vessels were discussed by Anliker et al. (1966) and Anliker (1972), by considering the wall material to be elastic and isotropic, Maxwell et al. (1968) extended their work (1966) for a viscoelastic arterial wall by making use of the correspondence principle. Recently some investigators considered mathematical models which are suitable for taking into account the effect of the initial stress together with the effect of surrounding tissues on the pulse wave propagation in arteries (cf. Atabek et al. (1966, 1968), Mirsky (1967)). Rachev (1978) studied an analytical model by taking into consideration the effect of muscle contraction on pulse wave propagation for canine middle descending thoracic aorta and also for humans of different age groups (1980).

1.18 Problems Considered in the Thesis

The thesis contains a few analytical studies on the mechanical behaviour of arterial walls and the flow properties of blood inside the wall.

The second chapter deals with a study on the finite deformations and stresses in vascular walls. Nonlinear viscoelastic constitutive relations are used in the analysis and they are suitable for taking into account the structural anisotropy of arterial walls. The pressures exerted by the surrounding connective tissues are also considered. Using approximations for short time ranges, a quantitative analysis is made in order to illustrate the applicability of the analytical study. Variations of the radial and tangential stresses versus time at a given point, are exhibited. Also the effect of the pressure due to tissues around the wall is quantitatively studied., The effect of nonlinearility is found to be significant.

The subject of discussion in the third chapter is a problem of wave propagation through a blood vessel. The effect of initial stress of the vessel wall on the propagating waves is of particular concern in the study. The analysis is carried out under the purview of the membrane theory. Nonlinearity and viscoelasticity of the wall are also considered here, as in the problems studied in the preceding chapter. Blood is considered as an incompressible Newtonian viscous fluid. It may be mentioned in this connection that the assumption of incompressibility can be justified by considering the small fluid velocities (order of 25 cms/sec) with respect to the acoustic velocity in blood (approx. 10^5 cms/sec); although experimental measurements of blood viscosity indicate that blood is non-Newtonian, previous experimenters also reported that as the shear stress increases, blood viscosity approaches a constant value. The motion of the fluid as also of the wall is considered axisymmetric. By employing the equations of motion of the fluid and those for the wall, together with the equation of continuity, a frequency equation is derived by exploiting the conditions of continuity of the velocities of the fluid and the vessel wall on the interface. The phase velocities are found for different frequencies. Also the variations of the phase velocity and the transmission coefficients are shown for different transmural pressures.

In the fourth chapter, attention has been paid to study the effect of tethering of the vascular wall, on the flow of blood. Both the radial and longitudinal tetherings are accounted for. The applicability of the analysis is illustrated by the numerical computations of the derived analytical expressions through the use of experimentally determined values of the material parameters and the effect of tethering is quantified thereby.

The response of a cylindrical arterial segment of a finite length in a state of finite strain, is investigated in the fifth chapter, when a small additional sinusoidally varying strain is superimposed upon the arterial segment, one end of which is held fixed while the other end is subjected to a given load in the longitudinal direction. The analysis pertains to a typically large artery for which the radial stress is negligibly small compared to the circumferential and longitudinal stresses.

The sixth chapter deals with a study on the loss of stability of arterial walls. The theory of a small deformation superposed on a known finite deformation, has been the basis of the analysis. The consideration is restricted to static stability, as per the following definition: 'A body is said to be statically stable for a given finite deformation and certain constraints if no non-trivial equilibrium configurations exist neighbouring to the finite deformation state and satisfying the constraints; it is said to be statically unstable if such configurations do occur'. The essence of a study on the static instability lies in the fact that one can discuss the stability of a system without investigating the reality. Thus only the existence of non-trivial equilibrium configurations (latent instabilities) of arterial walls are examined. On the basis of the analytically derived criterion for determining the stable region, the instabilities of canine middle descending thoracic aorta are studied by using suitable material functions determined empirically by previous investigators.

The effects of inertia and the compressibility of vascular tissues are studied through an analysis presented in the seventh and the concluding chapter. For this study a long cylindrical artery is considered. The outer surface of the arterial wall is taken to be traction-free while the inner surface is subjected to a pressure exerted by blood contained in it. It is assumed (as per a previous experimental study) that during finite deformation, the dilatational change in any element of vascular tissues is small (but finite). The effects of the inertial force and the low compressibility are quantified by making use of a particular set of relaxation functions.