CRABS by Fung Chung-Kwang (1896–1952). Ink on rice paper.

CHAPTER 5

Blood Flow in Heart, Lung, Arteries, and Veins

5.1 Introduction

The next five chapters are concerned with flow inside the bodies of man and animals. By internal flow of blood, water, and gases, the cells of the body obtain water, oxygen, and nutrients. To understand the health and disease of these organisms, it is necessary to know the mechanics of internal flow.

A great variety of things happen in the circulatory and respiratory systems. To observe them we use a variety of tools. Most important are our eyes. To help our eyes we use x-rays, cinematography, CAT scan, NMR, ultrasound imaging, etc. For smaller things, we use optical microscopes, x-rays, electron microscopes, scanning tunneling microscopes. At different levels of scale, different objects come into view. Together they reveal the phenomenon of living. It is the function of mechanics to analyze and integrate the phenomena at different scales. If the mechanics of a phenomenon at one level of scale is called macroscopic, and that at a smaller level of scale is called microscopic, then in biomechanics one often attempts to connect the microscopic to macroscopic mechanics. For example, the dimension of an endothelial cell is much smaller than the diameter of the aorta. The mechanics of the endothelial cells is microscopic relative to the mechanics of the aorta. But they are connected when atherosclerosis is concerned. Again, the diameters of the collagen and elastin fibers are much smaller than the diameter of the arteries, but fiber mechanics and arterial mechanics are connected.

In the following, we shall first study blood flow at the scale of the heart and large arteries. Then (in Chapter 6) we focus on small blood vessels and attempt to clarify the connection between the micro- and macrohemodynamics. In Chapter 7 we do the same for the phenomena of respiration. In Chapters 8 and 9, the flow of water and other constituents from blood vessels to extra-

vascular space, the fluid movement in the tissue space, and the fluid exchange between interstitium and cells are discussed. The presentations in Chapters 5 and 6 are rather condensed. Interested readers are referred to the author's book *Biodynamics: Circulation* (Springer-Verlag, 1984) for a more thorough treatment. The material in Chapters 7, 8 and 9 is presented at a more leisurely pace.

5.2 The Geometry of the Circulation System

Every animal's circulation system is special, but we shall consider typical features of man, dog, and cat. In these animals, blood flows from the superior and inferior vena cava into the right atrium, then through the tricuspid valve into the right ventricle, then through the semilunar valves into the pulmonary artery, the lung, the pulmonary veins, the left atrium, the mitral valve, the left ventricle, and finally through the aortic valve into the aorta. The peripheral circulation begins with the aorta, perfuses various organs, and returns to the right atrium. In each organ, flow begins in the arteries, perfuses the microcirculatory bed, then drains into veins. The vena cava collect blood from various organs, and send it to the heart.

Figure 5.2:1 shows the heart in greater detail. The two thin-walled atria are separated from each other by an interatrial septum. The two ventricles are separated by an interventricular septum. The left ventricle is thick-walled. In systolic condition, the pressure of blood in the left ventricle is higher than that in the right ventricle; hence the interventricular septum bulges out toward the right ventricle. The left ventricle can be represented as an ellipsoid; the right ventricle can be represented as a bellow. The four valves are seated in a plane and their bases are connected into an integrated structure, so that the enlargement of two opening valves is accompanied with the reduction in size of the other two closing valves. The mitral and tricuspid valves are attached to papillary muscles, which contract in systole, pulling down the valves to generate systolic pressure rapidly, and prevent the valves from inversion into the atrium.

The lung consists of three trees; see Fig. 5.2:2. The airway tree is for ventilation. The trachea is divided into bronchi which enter the lung, subdivided repeatedly into smaller and smaller branches called bronchioles, respiratory bronchioles, alveolar ducts, and alveoli. The alveoli are the smallest units of the airway. The walls of the alveoli are capillary blood vessels. Every wall of an alveolus is exposed to gas on both sides, so each wall is called an interalveolar septum. The entire lung is wrapped in a pleural membrane like a balloon. The chest wall also has a pleura. The pulmonary pleura and the visceral pleura are apposed to each other with a very small gap (a few μm thick) between them. The sealed compartment of space between the pleura is called the intrapleural space. The pressure in the intrapleural space is ordinarily lower than atmospheric. The chest wall and the transpulmonary

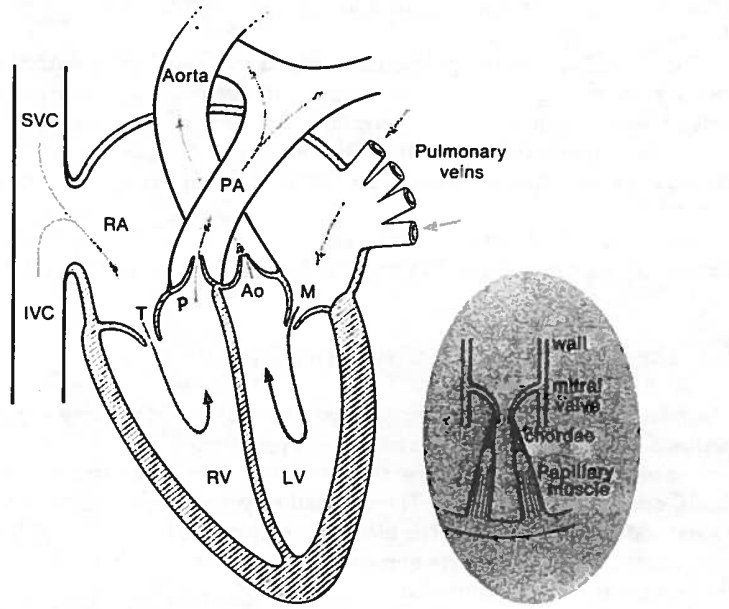


FIGURE 5.2:1 Blood flow through the heart. The arrows show the direction of blood flow. The symbols are: SVC, superior vena cava; IVC, inferior vena cava; RA, right atrium; RV, right ventricles; PA, pulmonary artery; LV, left ventricle. The valves are T, tricuspid, P, pulmonary, AO, aortic, M, mitral. From Folkow and Neil (1971) Circulation, Oxford Univ. Press, New York, p. 153, by permission.

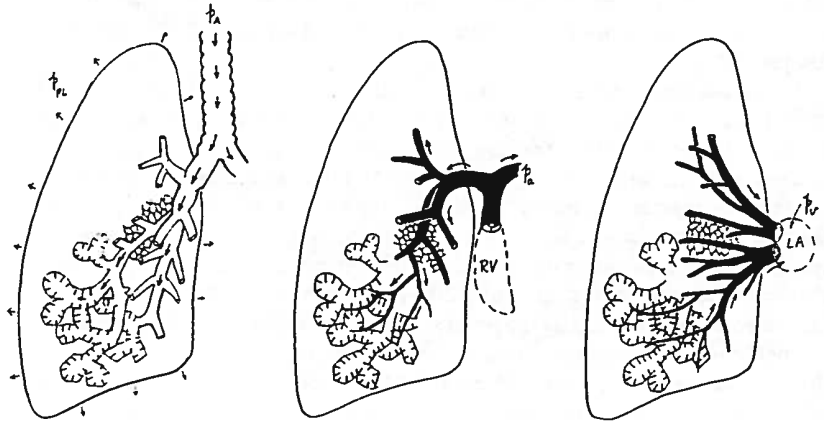


FIGURE 5.2:2 The three "trees" of the lung.

pressure (= the difference of the alveolar gas pressure and the pleural pressure) distends the lung.

The second tree is the pulmonary arterial tree. Beginning with the pulmonary artery, the tree bifurcates again and again until it forms capillary blood vessels which separate the alveoli.

The third tree is the venous tree. Beginning with the capillaries, the blood vessels converge repeatedly until they form pulmonary veins which enter the left atrium.

Such a sketch of the circulation system cannot give the needed details. Greater details can be found in Fung (1984) and other references listed therein.

5.3 The Materials of the Circulation System

The heart is a muscle. The lung is blood vessels and airways. All organs are perfused by blood via blood vessels. Blood vessels consist of smooth muscles, endothelial cells, and connective tissues. External to blood vessels are body fluids, cells, and interstitium. The circulation system also includes the lymphatic and nervous systems. The blood is a multiphase fluid composed of cells and plasma. Thus, the variety and complexity of the materials of the circulation system is truly monumental.

A detailed discussion of the chemical composition, molecular and higher structures (biochemistry, histochemistry), quantitative determination of the geometrical features of the internal structure of the tissue (morphometry, stereology, histology, anatomy), and the mechanical properties of the tissue (biomechanics) and its components (micromechanics) of any of the tissues and organs of the circulatory system would require much space, and is beyond the scope of this book. The reader is referred to the literature listed in the Bibliography at the end of this chapter. The author's book *Biomechanics: Mechanical Properties of Living Tissues* is a convenient reference. In the following sections, only the essential data required for immediate discussion are presented.

It is important to realize that the mechanical properties of many biological materials are very different from those of familiar engineering materials. For example, the incremental Young's modulus of the blood vessel wall or the relaxed muscles vary with the stress acting in the tissue; they do not remain constant as engineering materials do. For the heart, it is important to know that the maximum active tensile stress which can be generated in an isometric contraction of a cardiac muscle varies with the length of the sarcomere. See the length-tension curve in Fig. 5.3:1. If a heart normally operates at a sarcomere length marked by the point A in the figure, then when the sarcomere is lengthened, the maximum muscle tension will increase, and consequently, the systolic pressure, p_i, will increase. Since the number of sarcomeres in a heart muscle is fixed, the sarcomere length is proportional to the muscle length, and by implication, to the radius of the heart. Thus, if the radius of the

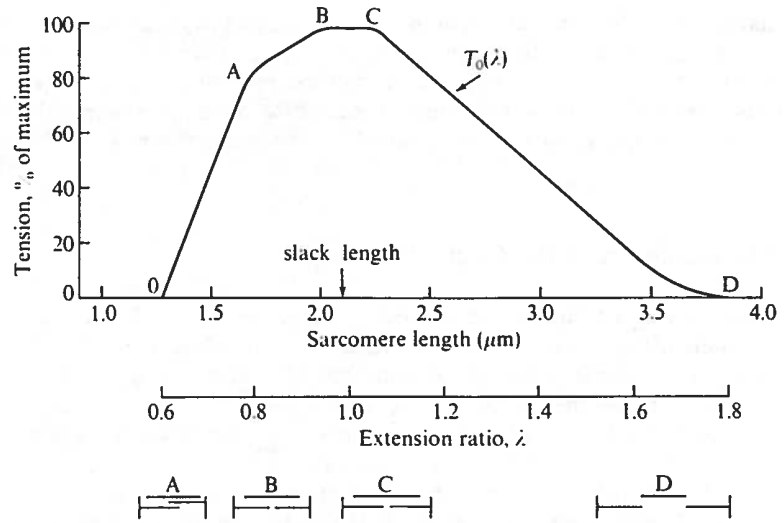


FIGURE 5.3:1 The "length-tension" curve of a skeletal muscle. The sarcomere length is plotted on the abscissa. The maximum tension achieved in isometric contraction at the length specified is plotted on the ordinate.

heart is increased, the muscle tension will increase, and so will be the systolic blood pressure. This is known as *Starling's law of the heart*. This law ceases to be valid when A moves off the upward-sloping leg of the curve shown in Fig. 5.3:1.

A similar length-tension curve exists for the vascular smooth muscle. Since the length of a muscle cell in a tissue depends on the strain at the place where the muscle cell is located, it becomes clear that the mechanical properties of the tissues of the circulatory system depend on the strain in the organ.

Another remarkable property of the blood vessels and the heart is the existence of large residual strains in these organs. Residual strains remain in an organ when all the external loads are removed; e.g., when the transmural pressure in a blood vessel is reduced to zero. This is discussed in Chapters 11 and 13. See Secs. 11.2 and 13.8.

5.4 Field Equations and Boundary Conditions

The basic equations of biomechanics are the equation of conservation of mass, the equation of motion, the constitutive equations specifying the mechanical properties of the materials, and, if heat and transfer and chemical reactions are involved, the energy equation and reaction rate equation. These and the

equations describing the boundary conditions are all that are allowed in a theoretical analysis of circulation.

The conservation of mass is expressed by the equation of *continuity* (Sec. 1.7). For a segment of a blood vessel, it says that

$$\text{The difference of inflow and outflow} + \text{the rate of change of the volume of the segment} = 0. \tag{1}$$

The equation of motion is a statement of Newton's law, which takes the following form when applied to a fluid or solid:

$$\text{Density} \times (\text{transient acceleration} + \text{convective acceleration}) = -\text{pressure gradient} + \text{force due to stress tensor} + \text{body force per unit volume}. \tag{2}$$

Here *density* refers to the density of the fluid or solid, the *transient acceleration* refers to the rate of change of the local velocity with respect to time, and the *convective acceleration* refers to the rate of change of velocity of a material particle caused by the motion of the particle from one place to another in a nonuniform velocity field. *Pressure gradient* is the rate of change of pressure versus distance. The force due to stress tensor refers to the force per unit volume due to the rate of change of stresses, taken their directions and areas into account in a proper way. See Sec. 1.7.

For the blood, the body force consists of inertial forces due to gravitational acceleration, Coriolis acceleration, and the acceleration of the body due to walking, jumping, swimming, flying, or other motion; the stress tensor consists of shear stresses caused by the viscosity of the fluid. For the blood vessel, the body forces are similar, the stress tensor is mainly caused by distension of the vessel due to blood pressure.

The constitutive equation of the blood describes the law of viscosity of the blood, which is, in fact, quite complex (see Fung, 1981). Whole blood is non-Newtonian, whose viscosity changes with the strain rate.

The constitutive equation of the blood vessel is the stress-strain relationship of the vessel wall material. It is also quite complex because it does not obey Hooke's law (see Fung, 1981, and Chapters 10 and 11 of this book).

In special situations, it is permissible to use approximate constitutive equations to simplify the analysis. For example, in large animals such as cat and man, the shear strain rate of the blood at the walls of the heart and the pulmonary and systemic arteries and veins exceeds 100 sec^{-1} so that in that region the coefficient of viscosity of blood may be regarded as a constant. The non-Newtonian feature is important only in a region near the centerline of the blood vessel. The integrated effect of the non-Newtonian feature is quite negligible so that flowing blood in these vessels may be treated as Newtonian.

For the blood vessel wall, the stress-strain relationship can be linearized (into an incremental Hooke's law) if the amplitude of deformation is very small. For pulmonary arteries and veins, the pressure-diameter relationship has been found to be linear because these vessels are embedded in an elastic medium—the lung parenchyma (see Fung, 1981, 1984, and Sec. 5.15, Figs. 5.15:4 and 5).

The boundary condition between a *viscous fluid* and a *solid* is the *no-slip condition*: there is no relative movement of material particles of the fluid at the boundary and the material particles of the solid at the same interface.

For an *ideal fluid* whose viscosity is zero, slip must be permitted, the boundary condition is then reduced to the condition that the materials on the two sides of an interface must remain contiguous: they must have the same velocity normal to the interface.

Across any surface on the boundary, the stress vectors on the two sides of the boundary surface must be equal and opposite by the condition of equilibrium.

In the analysis of blood flow in any particular blood vessel, one must not forget the two ends of the vessel. The *entry* and *exit* conditions with regard to pressure and velocity distributions at the ends must be specified.

These are the basic equations and principles. Some special problems are formulated, solved, and their physiological meaning discussed in the following sections.

5.5 Blood Flow in Heart and Through Heart Valves

The direction of blood flow in the heart is shown schematically in Fig. 5.2:1, the venous blood flows into the right atrium, through the tricuspid valve into the right ventricle, and then is pumped into the pulmonary artery and the lung, where the blood is oxygenated. The oxygenated blood then flows from the pulmonary veins into the left atrium, and through the mitral valve into the left ventricle, whose contraction pumps the blood into the aorta, and then to the arteries, aterioles, capillaries, venules, veins, and back to the right atrium.

An aortic valve with the sinus of Valsalva behind it is sketched in Fig. 5.5:1. According to model experiments by Bellhouse and Bellhouse (1969, 1972) and Lee and Talbot (1979), the flow issuing from the ventricle, immediately upon opening of the valve during systole, is split into two streams at each valve cusp, as shown in the figure. One part of the flow is directed into the sinus behind the valve cusp, where it forms a vortical flow before reemerging out of the plane of the figure, to rejoin the main stream in the ascending aorta.

When the aortic pressure rises sufficiently so that deceleration of the flow occurs, an adverse pressure gradient is produced, p_2 at the valve tip exceeds the pressure p_1 at a station upstream. The higher pressure p_2 causes a greater flow into the sinus which carries the cusp toward apposition. The peak deceleration occurs just before the valve closure. The vortical motion established earlier upon the opening of the valve has the merit of preventing the

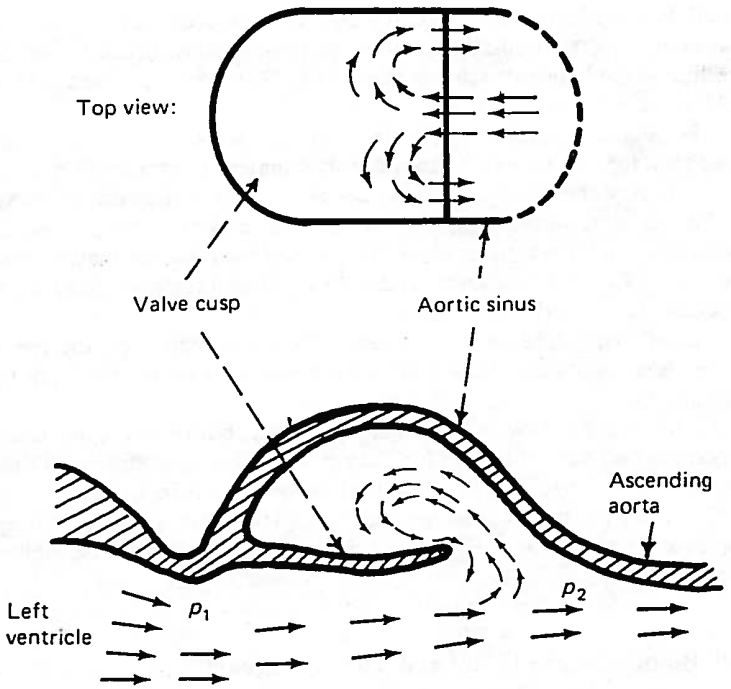


FIGURE 5.5:1 Flow pattern within the sinus of Valsalva.

valve cusp from bulging outward to contact the walls of the sinuses. The open sinus chamber thus can be supplied with fluid to fill the increasing volume behind the valve cusps as they move toward closure.

Other heart valves and the valves of the veins and lymphatics are operated by hydrodynamic forces in a similar way, although they do not have sinuses. In closing these valves, deceleration of the fluid is the essence, not backward flow.

5.6 Coupling of Left Ventricle to Aorta and Right Ventricle to Pulmonary Artery

As the heart muscle contracts periodically, blood is pumped from the left ventricle into the aorta through the aortic valve, and simultaneously from the right ventricle into the pulmonary artery through the pulmonary valve. The aorta and the pulmonary artery, being elastic, expand when they receive blood at a rate faster than the rate at which they send blood away into the peripheral organs and the lung, respectively. Expanding an elastic vessel causes an increase of the circumferential strain and stress in the vessel wall. A blood

vessel with an increased circumferential stress in its wall will press harder on the blood it contains. As a result the blood pressure is increased. The increased blood pressure in the aorta acts on the aortic side of the aortic valve, tending to close it. An additional tendency to close the valves comes from the deceleration of the blood in the aorta. The deceleration occurs when the inflow exceeds the outflow. A consequence of the deceleration is the creation of a longitudinal pressure gradient through the aortic valve, again tending to close the valve. Eventually the valve is closed, blood continues to flow from the aorta into the periphery. By this mechanism the blood flow in the aorta does not have large swing of pressure as it has in the left ventricle. Similar events occur in the lung.

The process described above can be presented mathematically in various levels of generality. To be rigorous, it seems evident that the heart, aorta, arteries, and veins should be represented by three-dimensional network, and the special geometry and materials of construction of various organs must be described and incorporated in the mathematical model. In practice it is useful to consider simplified, crude models first, learn the general features, identify the important parameters, and then add details when needed. Accordingly, we shall consider the Windkessel model in this section, and the long wave, small amplitude pulse waves in the next section. Other features are added in following sections.

The *Windkessel theory* is Otto Frank's (1899) interpretation of Stephan Hale's (1733) explanation of why the pressure fluctuation in the aorta has a much smaller amplitude than that in the left ventricle. In this theory, the aorta is represented by an elastic chamber and the peripheral blood vessels are replaced by a rigid tube of constant resistance. See Fig. 5.6:1. Let $\dot{Q}$ be the inflow (cm^3/sec) into this system from the left ventricle. Part of this inflow is sent to the peripheral vessels and part of it is used to distend the elastic chamber. If p is the blood pressure in the elastic chamber (aorta), then the flow in the peripheral vessel is assumed to be equal to p/R, where R is a constant called *peripheral resistance.* For the elastic chamber, its change of volume is assumed to be proportional to the pressure. The rate of change of the volume of the elastic chamber with respect to time, t, is therefore proportional to dp/dt. Let the constant of proportionality be written as C and called *compliance.* Then, on equating the inflow to the sum of the rate of change of volume of the elastic chamber and the outflow p/R, the differential equation governing the pressure p is

$$\dot{Q} = C\frac{dp}{dt} + p/R. \tag{1}$$

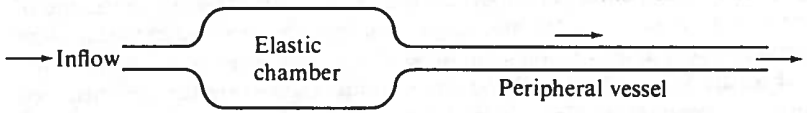


FIGURE 5.6:1 The "windkessel" model of the aorta and peripheral circulation.

To solve this equation, we can use the method of integration factor. Dividing Eq. (1) by C and multiplying it by $e^{t/RC}$, we obtain

$$\frac{1}{C}\dot{Q}(t)e^{t/RC} = \frac{dp}{dt}e^{t/RC} + \frac{1}{RC}pe^{t/RC} = \frac{d}{dt}(pe^{t/RC}). \tag{2}$$

Integrating both sides from $t = 0$ to t and writing the dummy variable as τ, we have

$$p(t)e^{t/RC} = \int_0^t \frac{1}{C}\dot{Q}(\tau)e^{\tau/RC}\,d\tau + p_0, \tag{3}$$

where p_0 is the value of p at $t = 0$. If we take the instant of time when the valve opens as $t = 0$, then p_0 is the systolic pressure in the ventricle at the instant when the valve opens. Multiplying both sides with $e^{-t/RC}$, we get

$$p(t) = e^{-t/(RC)}\int_0^t \frac{1}{C}\dot{Q}(\tau)e^{\tau/(RC)}\,d\tau + p_0e^{-t/(RC)} \tag{4}$$

which gives the pressure in the aorta as a function of the left ventricular ejection history $\dot{Q}(t)$.

An analog electric circuit can be formulated to represent the differential equation (Eq. 1). When this electric model is driven by a current $I = \dot{Q}(t)$ of the shape of an experimentally determined flow through the aortic value at the ascending aorta, the voltage V obtained is the analog of the blood pressure in the aortic arch. On comparing the analog results with an experimentally determined blood pressure curve, it is found that the actual pressure pulse deviates from the calculated results in several details: the experimental curve has a superimposed 3–6 cps oscillation apparent from midsystole throughout diastole, and a more prominent "incisura" marking aortic valve closure and a more abrupt rise, often with an "anacrotic" wave. In addition, the Windkessel model fails to explain the changes of the form of pressure wave occurring along the arterial network. These limitations of the Windkessel theory can be alleviated by an improved model such as the one presented in the next section.

The analysis also applies to the coupling of the right ventricle and pulmonary artery. The pulmonary circulation, however, is a lower pressure system. The wall of the right ventricle is thinner than that of the left ventricle; its systolic pressure is lower. The systolic and diastolic pressures in the pulmonary artery are much lower than those in the aorta. Since the flows in the aorta and pulmonary artery are about the same, the shape of $p(t)$ given by the first term on the right-hand side of Eq. (4) can be similar (except for the amplitude) only if the values of RC are approximately the same in both circuits. Hence the low pressure in pulmonary circulation must be achieved by a lower right ventricular pressure p_0, a lower resistance R, and a higher compliance C of the pulmonary circuit.

The right ventricle and the left ventricle are two pumps working in series. The flow in them must be matched perfectly, otherwise all the blood would

eventually be accumulated either in the lung or in the periphery. The matching is stabilized by Starling's law of the heart (Sec. 5.3), namely, if the diastolic volume is increased, the contracting force of the muscle will increase to pump harder.

5.7 Pulsatile Flow in Arteries

The weakness of the Windkessel theory is that it allows only one degree of freedom. The pressures in the aorta and arteries are represented by a single number. It ignores the change of pressure along the vascular tree. To improve the understanding of events occurring in the arteries, we go to the next simplest model: *considering each artery as a long, isolated, circular cylindrical elastic tube, allowing an infinite number of degrees of freedom, approximating the flow to be one dimensional, and blood as a homogeneous, nonviscous, incompressible fluid.* The flow in each tube is excited at one end by the heart. The excitation is propagated in the form of elastic waves, much as an earthquake generates seismic waves. At the distal end each tube bifurcates, and the waves are partly transmitted to the daughter branches and partly reflected. This theory was originated by Euler (1775) and Young (1808, 1809), and developed by many others. It explains many things, but must be supplemented by three-dimensional theories when one wants to know the velocity profile, flow separation, stenosis, microcirculation, etc., which are important to the understanding of atherosclerosis, hypertension, etc.

To present this theory in the simplest form, *it is further assumed that the wave amplitude is small and the wave length is long* compared with the tube radius, so that the radial and circumferential velocity components are negligibly small compared with the longitudinal velocity component $u(x, t)$, which is a function of the axial coordinate x and time t only. Then the basic field equations (Sec. 3.2) are: the equation of motion,

$$\frac{\partial u}{\partial t} + u\frac{\partial u}{\partial x} + \frac{1}{\rho}\frac{\partial p_i}{\partial x} = 0 \tag{1}$$

and the equation of continuity,

$$\frac{\partial A}{\partial t} + \frac{\partial}{\partial x}(uA) = 0. \tag{2}$$

Here $A(x, t)$ is the cross-sectional area of the tube and $p_i(x, t)$ is the pressure in the tube. The relationship between p_i and A may be quite complex. For simplicity we introduce another hypothesis, that A *depends on the transmural pressure,* $p_i - p_e$, *alone*:

$$p_i - p_e = P(A), \tag{3}$$

where p_e is the pressure acting on the outside of the tube. Equation (3) is a gross simplification. In the theory of elastic shells we know that the tube

deformation is related to the applied load by a set of partial differential equations and that the external load includes the inertial force of the tube wall. Hence Eq. (3) implies that the mass of the tube is ignored, and that the partial differential equations are replaced by an algebraic equation. The viscoelasticity of tube wall is ignored also.

Equation (1) is the one-dimensional case of the Eulerian equation of motion (Eq. (1.7:1)). Equation (2) can be obtained by integrating Eq. (1.7:5) over a tube. A special example of Eq. (3) is the pressure–diameter relationship of the pulmonary artery or vein (Yen et al., 1980, 1981):

$$2a_i = 2a_{i0} + \alpha p_i. \tag{4}$$

Here $2a_i$ is the vessel diameter, p_i is the blood pressure, a_{i0} and α are constants which depend on the pleural pressure p_{PL} and airway pressure p_A, but are independent of blood pressure p_i. α is the *compliance constant* of the vessel, and a_{i0} is the radius when $p_i = 0$.

Let us solve a linearized version of these equations. Consider small disturbances in an initially stationary liquid-filled circular cylindrical tube. In this case u is small and the second term in Eq. (1) can be neglected. Hence

$$\frac{\partial u}{\partial t} + \frac{1}{\rho}\frac{\partial p_i}{\partial x} = 0. \tag{5}$$

The area A is equal to πa_i^2. Substituting πa_i^2 for A in Eq. (2), remembering the hypothesis that the wave amplitude is much smaller than the wave length, so that $\partial a_i/\partial x \ll 1$, then, on neglecting small quantities of the second order, we can reduce Eq. (2) to the form

$$\frac{\partial u}{\partial x} + \frac{2}{a_i}\frac{\partial a_i}{\partial t} = 0. \tag{6}$$

Combining Eqs. (4) and (6), we obtain

$$\frac{\partial u}{\partial x} + \frac{\alpha}{a_i}\frac{\partial p_i}{\partial t} = 0. \tag{7}$$

Differentiating Eq. (5) with respect to x and Eq. (7) with respect to t, subtracting the resulting equations, and neglecting the second order term (α/a_i^2) $(\partial a_i/\partial t)$ $(\partial p_i/\partial t)$, we obtain

$$\frac{\partial^2 p_i}{\partial x^2} - \frac{1}{c^2}\frac{\partial^2 p_i}{\partial t^2} = 0, \tag{8}$$

where

$$c^2 = \frac{a_i}{\rho\alpha}. \tag{9}$$

Equation (8) is the famous *wave equation*. The quantity c is the wave speed.

By direct substitution, one can verify that Eq. (8) is satisfied by the solution

$$p_i = f(x - ct) + g(x + ct), \tag{10}$$

where f and g are arbitrary functions of the variables $x - ct$ and $x + ct$. The function $f(x - ct)$ represents a wave propagating to the right (increasing x) whereas $g(x + ct)$ represents a wave propagating to the left.

Velocity, Pressure, and Wall Displacement Waves

The velocity u is linearly related to p through Eqs. (5) and (7), and small change of the radius a is linearly related to changes in p through Eq. (4). Hence by eliminating p, it is seen that u and a are governed by the same wave equation with the same wave speed. If we write

$$\begin{aligned} p &= p_0 f(x - ct) + p_0' g(x + ct), \\ u &= u_0 f(x - ct) + u_0' g(x + ct), \end{aligned} \tag{11}$$

then on substituting Eqs. (11) into Eqs. (5) and (7), one sees that the amplitude p_0 and u_0 are related by the simple relationship

$$p_0 = \rho c u_0 \tag{12}$$

for a wave that is moving in the positive x direction, and

$$p_0' = -\rho c u_0' \tag{13}$$

for a wave which moves in the negative x direction.

Equations (12) and (13) show that *the amplitude of pressure wave is proportional to the product of wave speed and velocity disturbance and the fluid density. The pressure and velocity are in phase in an advancing progressive wave; they are out of phase in the reflected wave.*

5.8 Progressive Waves Superposed on a Steady Flow

It can be shown that the equations of Sec. 5.7 are applicable to tubes carrying a steady flow, provided that we adopt a coordinate system that moves with the undisturbed flow, and interpret u as the perturbation velocity superposed on the steady flow and c as the speed of perturbation wave relative to the undisturbed flow. The proof is as follows.

Let U be the velocity of the undisturbed flow, and u the small perturbation superposed on it. Treating u as an infinitesimal quantity of the first order, we see that the equation of motion, Eq. (5.7:1), can be linearized into

$$\frac{\partial u}{\partial t} + U\frac{\partial u}{\partial x} = -\frac{1}{\rho}\frac{\partial p_i}{\partial x}. \tag{1}$$

This can be reduced to Eq. (5.7:6) by introducing a transformation of variables

from x, t to x', t':

$$x' = x - Ut, \qquad t' = t. \tag{2}$$

From Eq. (2) we have

$$\frac{\partial}{\partial t} = \frac{\partial}{\partial t'}\frac{\partial t'}{\partial t} + \frac{\partial}{\partial x'}\frac{\partial x'}{\partial t} = \frac{\partial}{\partial t'} - U\frac{\partial}{\partial x'},$$
$$\frac{\partial}{\partial x} = \frac{\partial}{\partial t'}\frac{\partial t'}{\partial x} + \frac{\partial}{\partial x'}\frac{\partial x'}{\partial x} = \frac{\partial}{\partial x'}. \tag{3}$$

Hence, a substitution into Eq. (1) reduces it to

$$\frac{\partial u}{\partial t'} = -\frac{1}{\rho}\frac{\partial p}{\partial x'}, \tag{4}$$

which is exactly Eq. (5.7:5) in the new coordinates.

The equation of continuity, Eq. (5.7:2), now becomes

$$\frac{\partial a_i}{\partial t} + U\frac{\partial a_i}{\partial x} + \frac{a_i}{2}\frac{\partial u}{\partial x} = 0 \tag{5}$$

when πa_i^2 is substituted for A, $U + u$ is substituted for u, and the equation is linearized for small perturbations. Under the transformation Eq. (2), and using Eq. (3), Eq. (5) becomes

$$\frac{\partial a_i}{\partial t'} + \frac{a_i}{2}\frac{\partial u}{\partial x'} = 0, \tag{6}$$

which is exactly Eq. (5.7:7).

The pressure–radius relationship, Eq. (5.7:4), is independent of reference coordinates. Thus all the basic equations are unchanged. But x' and t' are the distance and time measured in the moving coordinates which translate with the undisturbed flow. Hence what we set out to prove is done.

5.9 Reflection and Transmission of Waves at Junctions

An arterial tree is composed of segments of cylindrical tubes. Consider a single junction as shown in Fig. 5.9:1 in which a tube branches into two daughters. A wave traveling down the parent artery will be partially reflected at the junction and partially transmitted down the daughters. At the junction, the conditions are: the pressure is a single-valued function and the flow must be continuous. Expressing this mathematically: p_I denote the oscillatory pressure associated with the incident wave, p_R that associated with the reflected wave, and p_{T_1} and p_{T_2} those associated with the transmitted waves in the two daughter tubes; then the single-valuedness of pressure means

$$p_I + p_R = p_{T_1} = p_{T_2}. \tag{1}$$

Similarly, let $\dot{Q}$ denote the volume–flow rate, and let the subscripts, I, R, T_1, T_2 refer to the various waves as before; then the continuity condition means

$$\dot{Q}_I - \dot{Q}_R = \dot{Q}_{T_1} + \dot{Q}_{T_2}. \tag{2}$$

But $\dot{Q}$ is the product of the cross-sectional area A and the mean velocity u, which is related to p by Eqs. (12) and (13) of Sec. 5.7. Thus, the flow–pressure relationship is:

$$\dot{Q} = Au = \pm\frac{A}{\rho c}p. \tag{3}$$

Here ρ is the density of the blood and c is the wave speed. The + sign applies if the wave goes in the direction of flow; the − sign applies if the wave goes the other way.

The quantity $\rho c/A$ is an important characteristic of the artery, and is called the *characteristic impedance* of the tube. It is denoted by the symbol Z:

$$Z = \frac{\rho c}{A}. \tag{4}$$

Equation (3) shows that Z is the ratio of oscillatory pressure to oscillatory flow when the wave goes in the direction of flow:

$$Z = \frac{p}{\dot{Q}}, \qquad Z\dot{Q} = p, \tag{5}$$

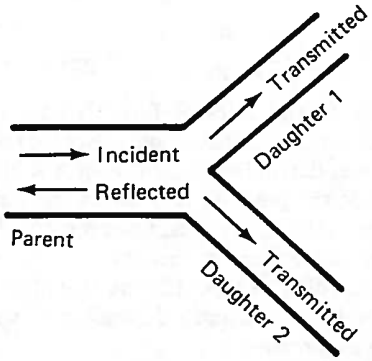
Z has the physical dimensions $[ML^{-4}T^{-1}]$, and can be measured in units of kg m^{-4} sec^{-1}. With the Z notation, Eq. (2) can be written as

$$\frac{p_I - p_R}{Z_0} = \frac{p_{T_1}}{Z_1} + \frac{p_{T_2}}{Z_2}. \tag{6}$$

Solving Eqs. (1) and (6) for the p's, we obtain

$$\frac{p_R}{p_I} = \frac{Z_0^{-1} - (Z_1^{-1} + Z_2^{-1})}{Z_0^{-1} + (Z_1^{-1} + Z_2^{-1})} = \mathscr{R} \tag{7}$$

FIGURE 5.9:1 A bifurcating artery.



and

$$\frac{p_{T_1}}{p_I} = \frac{p_{T_2}}{p_I} = \frac{2Z_0^{-1}}{Z_0^{-1} + (Z_1^{-1} + Z_2^{-1})} = \mathscr{J}. \tag{8}$$

The right-hand sides of Eqs. (7) and (8) shall be denoted by $\mathscr{R}$ and $\mathscr{J}$, respectively. Hence the amplitude of the reflected pressure wave at the junction is $\mathscr{R}$ times that of the incident wave, the amplitude of the transmitted pressure waves at the junction is $\mathscr{J}$ times the incident wave. The amplitude of the reflected velocity wave is, however, equal to $-\mathscr{R}$ times that of the incident velocity wave, because the wave now moves in the negative x-axis direction, and according to Eqs. (12) and (13) of Sec. 5.7, there is a sign change in the relation between u and p depending on whether the waves move in the + or − x-axis direction.

If the incident wave is

$$p_I = p_0 f(t - x/c_0) \tag{9}$$

and the junction is located at $x = 0$, so that x is negative in the parent tube and positive in the daughter tubes, then at the junction $x = 0$, the pressure is

$$p_I = p_0 f(t). \tag{10}$$

The reflected and transmitted waves are, therefore,

$$\begin{aligned} p_R &= \mathscr{R} p_0 f(t + x/c_0), \\ p_{T_1} &= \mathscr{J} p_0 f(t - x/c_1), \\ p_{T_2} &= \mathscr{J} p_0 f(t - x/c_2). \end{aligned} \tag{11}$$

Here, c_0, c_1, c_2 are the wave speeds in the respective tubes. The wave in the parent tube is

$$p = p_I + p_R = p_0 f(t - x/c_0) + \mathscr{R} p_0 f(t + x/c_0). \tag{12}$$

$$\dot{Q} = \frac{A p_0}{\rho c_0} f(t - x/c_0) - \mathscr{R} \frac{A p_0}{\rho c_0} f(t + x/c_0). \tag{13}$$

Equations (12) and (13) show that with a reflection, the pressure and flow wave forms are no longer equal.

5.10 Velocity Profile of a Steady Flow in a Tube

Having analyzed the aortic blood flow by lumped parameter method (Sec. 5.6), and pulse wave in arteries as one-dimensional nonstationary flow of a nonviscous fluid in an elastic tube (Secs. 5.7–5.9), we shall now consider the effect of viscosity of blood on the flow. We shall first consider blood as a Newtonian fluid.

Consider first a steady flow of an incompressible Newtonian fluid in a rigid, horizontal channel of width $2h$ between two parallel planes as shown in Fig.

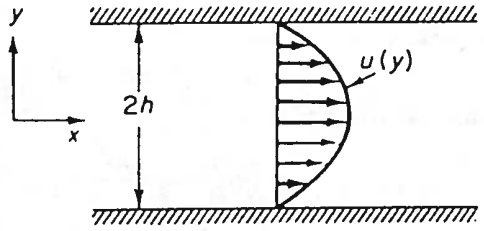


FIGURE 5.10:1 Laminar flow in a channel.

5.10:1. The channel is assumed horizontal so that the gravitational effect (a body force) may be ignored.

We search for a flow,

$$u = u(y), \qquad v = 0, \qquad w = 0, \tag{1}$$

which satisfies the no-slip conditions on the boundaries $y = \pm h$:

$$u(h) = 0, \qquad u(-h) = 0. \tag{2}$$

The function u must satisfy the Navier–Stokes equation and the equation of continuity (Sec. 1.7). It is seen that the equation of continuity is satisfied exactly. The Navier–Stokes equation is simplified to:

$$0 = -\frac{\partial p}{\partial x} + \mu \frac{d^2 u}{dy^2}, \tag{3a}$$

$$0 = \frac{\partial p}{\partial y}, \tag{3b}$$

$$0 = \frac{\partial p}{\partial z}. \tag{3c}$$

Equations (3b) and (3c) show that p is a function of x only. If we differentiate Eq. (3a) with respect to x and use Eq. (1), we obtain $\partial^2 p/\partial x^2 = 0$. Hence $\partial p/\partial x$ must be a constant. Equation (3a) then becomes

$$\frac{d^2 u}{dy^2} = \frac{1}{\mu} \frac{dp}{dx}, \tag{4}$$

which has a solution

$$u = A + By + \frac{1}{\mu} \frac{y^2}{2} \frac{dp}{dx}. \tag{5}$$

The two constants A and B can be determined by the boundary conditions, Eq. (2), to yield the final solution

$$u = -\frac{1}{2\mu}(h^2 - y^2) \frac{dp}{dx}. \tag{6}$$

Thus, the velocity profile is a parabola.

Next consider a flow through a horizontal circular cylindrical tube of radius a. Using polar coordinates, it is easy to show that the solution is [see Fung (1984), p. 83]

$$u = -\frac{1}{4\mu}(a^2 - r^2)\frac{dp}{dx}. \tag{7}$$

This is the famous parabolic velocity profile of the *Hagen–Poiseuille flow.*

From the solution (7) we obtain the *rate of flow* through the tube by integration:

$$\dot{Q} = 2\pi \int_0^a ur\,dr = -\frac{\pi a^4}{8\mu}\frac{dp}{dx}. \tag{8}$$

This classical solution has been subjected to innumerable experimental validation. It was found to be invalid near the entrance to a tube. It is satisfactory at a sufficiently large distance from the entrance but is again invalid if the tube is too large or too long if the velocity is too high. The difficulty at the entry region is due to the transitional nature of the flow in that region so that our assumption $v = 0$, $w = 0$, is not valid. The difficulty with too large a Reynolds number, however, is of a different kind: the flow becomes turbulent.

Osborne Reynolds demonstrated the transition to turbulent flow in a classical experiment in which he examined an outlet from a large water tank through a small tube. At the end of the tube there was a stopcock used to vary the speed of water through the tube. The junction of the tube with the tank was nicely rounded, and a filament of colored fluid was introduced at the mouth. When the speed of water was slow, the filament remained distinct through the entire length of the tube. When the speed was increased, the filament broke up at a given point and diffused throughout the cross-section. Reynolds identified the governing parameter $u_m d/\nu$—the Reynolds number—where u_m is the mean velocity, d is the diameter, and ν is the kinematic viscosity. The point at which the color diffuses throughout the tube is the transition point from laminar to turbulent flow in the tube. Reynolds found that transition occurred at Reynolds numbers between 2,000 and 13,000, depending on the smoothness of the entry conditions. When extreme care is taken, the transition can be delayed to Reynolds numbers as high as 40,000. On the other hand, a value of 2,000 appears to be about the lowest value obtainable on a rough entrance. Turbulence is one of the most important and difficult problems in fluid mechanics.

5.11 Steady Laminar Flow in an Elastic Tube

If the tube is elastic (Fig. 5.11:1), then the high-pressure end would distend more than the low-pressure end. The diameter of the tube is, therefore, nonuniform (if it were uniform originally) and the degree of nonuniformity depends on the flow rate.

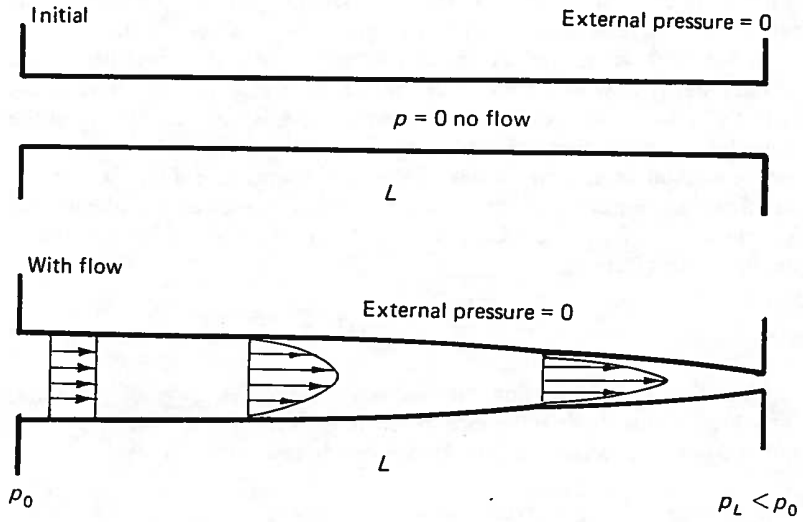


FIGURE 5.11:1 Flow in an elastic tube of length L.

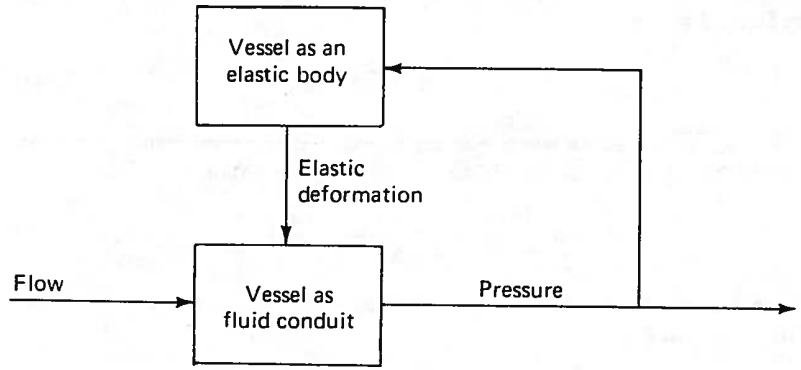


FIGURE 5.11:2 A hemoelastic system analyzed as a feedback system of two functional units: an elastic body, and a fluid mechanism.

If we wish to determine the pressure–flow relationship for such a system, we may break down the problem into two familiar components. This is illustrated in Fig. 5.11:2. In the lower block, we regard the vessel as a rigid conduit with a specified wall shape. For a given flow, we compute the pressure distribution. This pressure distribution is then applied as loading on the elastic tube, represented by the upper block. We then analyze the deformation of the elastic tube in the usual manner of the theory of elasticity. The result of the calculation is then used to determine the boundary shape of the hydrodynamic

problem of the lower block. When a consistent solution is obtained, the pressure distribution corresponding to a given flow is determined.

In Sec. 5.10 we derived Poiseuille's formula under the assumption of a laminar steady flow in a circular cylindrical tube of constant radius. If the radius $a(x)$, as a function of the axial coordinate x, is not a constant, but the slope da/dx is small, then the fluid dynamic problem can be solved by perturbation method as a power series of the small parameter da/dx. Under an additional assumption that the Reynolds number is so small that the inertial force term $\rho u\,\partial u/\partial x$ is negligible in the zeroth order equations, the solution is the Poiseuille's formula

$$\frac{dp}{dx} = -\frac{8\mu}{\pi a^4}\dot{Q} \tag{1}$$

in which $\dot{Q}$ is the volume–flow rate and is a constant for the whole tube, $a(x)$ is the local radius, dp/dx is the local pressure gradient. For an elastic tube, the radius a is a function of pressure. Hence we can rewrite Eq. (1) as

$$a^4(p)\frac{dp}{dx} = -\frac{8\mu}{\pi}\dot{Q} = \text{const.} \tag{2}$$

This is very easy to integrate if the function $a(p)$ is known. For the pulmonary arteries and veins it is known that the pressure–radius relationship is given by Eq. (5.7:4)

$$a = a_0 + \frac{\alpha}{2}p, \tag{3}$$

where a_0 is the radius when p is zero, and α is the compliance constant. Substituting Eq. (3) into Eq. (2), and integrating, we obtain

$$a^4\frac{dp}{da}\frac{da}{dx} = \frac{2}{\alpha}a^4\frac{da}{dx} = -\frac{8\mu}{\pi}\dot{Q}. \tag{4}$$

Since the right-hand side term is a constant independent of x, we obtain the integrated result

$$[a(x)]^5 = -\frac{20\mu\alpha}{\pi}\dot{Q}x + \text{const.} \tag{5}$$

The integration constant can be determined by the boundary condition that when $x = 0$, $a(x) = a(0)$. Hence the constant $= [a(0)]^5$. Then by putting $x = L$, we obtain from Eqs. (5) and (3) the elegant result (Fung, 1984)

$$\begin{aligned}\dot{Q} &= \frac{\pi}{20\mu\alpha L}\{[a(0)]^5 - [a(L)]^5\} \\ &= \frac{\pi}{20\mu\alpha L}\left[\left(a_0 + \frac{\alpha}{2}p_0\right)^5 - \left(a_0 + \frac{\alpha}{2}p_L\right)^5\right].\end{aligned} \tag{6}$$

Thus the flow varies with the difference of the fifth power of the tube radius at the entry section ($x = 0$) minus that at the exit section ($x = L$). If the ratio $a(L)/a(0)$ is $\frac{1}{2}$, then $[a(L)]^5$ is only about 3% of $[a(0)]^5$ and is negligible by comparison. Hence when $a(L)$ is one-half of $a(0)$ or smaller, the flow varies directly with the fifth power of the tube radius at the entry, whereas the radius (and the pressure) at the exit section has little effect on the flow.

This analysis applies very well to the lung, in which the phenomenon just described has an important effect. See Sec. 6.8 infra.

For flow in large blood vessels with Reynolds number much greater than 1, the inertial force terms must be added. Let us consider the case of a steady flow of Newtonian fluid in an elastic tube whi which is initially a circular cylinder. Assume that the flow is predominantly one-dimensional. Let q represent the average velocity in the tube. The convective inertial force is $\rho q(\partial q/\partial x)$. The pressure drop due to blood viscosity is given by Eq. (1) even if the flow is turbulent, provided that the coefficient of viscosity μ is reinterpreted as the "*apparent*" *coefficient of viscosity* which is a function of the Reynolds number, see Sec. 5.13 infra. Then the equation of motion is

$$\rho q\frac{dq}{dx} = -\frac{dp}{dx} - \frac{8\mu}{\pi a^4}\dot{Q}. \tag{7}$$

Here ρ is the density of the blood, x is the axial coordinate, p is the pressure, $\dot{Q}$ is the volume flow rate, and μ is the apparent coefficient of viscosity of the blood corrected for turbulence, secondary flow, or entrance effect, i.e. it is a function of Reynolds number. Finally, a is the radius of the tube, which is a linear function of pressure as given by Eq. (3). When the transmural pressure is zero the tube is assumed to be cylindrical, $a_0 = \text{const}$. The equation of continuity is

$$\pi a^2 q = \text{const} = \dot{Q}. \tag{8}$$

By differentiation, one obtains

$$a^2 dq + 2qada = 0. \tag{9}$$

On solving Eq. (8) for q, and substituting into Eq. (9) multiplied by q/a^2, we have

$$q\,dq = -\frac{2\dot{Q}^2}{\pi^2 a^5}da. \tag{10}$$

Substituting Eq. (10) into Eq. (7) and reducing, one obtains

$$\left(a^4 - \frac{\rho\alpha\dot{Q}^2}{\pi^2 a}\right)\frac{da}{dx} = -\frac{4\mu\alpha}{\pi}\dot{Q}. \tag{11}$$

Integration yields

$$a^5 - \frac{5\rho\alpha\dot{Q}^2}{\pi^2}\ln a = -\frac{20\mu\alpha}{\pi}\dot{Q}x + \text{const.} \tag{12}$$

The boundary condition $a = a(0)$ when $x = 0$ yields the integration constant. On putting this constant into Eq. (12), and then letting $x = L$ where $a = a(L)$, we obtain

$$\dot{Q} - \left[\frac{\rho}{4\mu\pi L}\ln\frac{a(L)}{a(0)}\right]\dot{Q}^2 = \frac{\pi}{20\mu\alpha L}\{[a(0)]^5 - [a(L)]^5\}. \tag{13}$$

This is a modification of Eq. (6). The effect of inertial force is embodied in the second term. If the elastic deformation is small, $a(L) \doteq a(0)$, then the second term tends to zero, and the solution is the same as Eq. (6) except that the apparent viscosity μ must now be considered as a function of the Reynolds number. If the elastic deformation is significant, in that $a(L)$ differs considerably from $a(0)$, then the second term must be considered. For a given set of values $a(L)$, $a(0)$, we now have two solutions of $\dot{Q}$. Conversely, for a given $\dot{Q}$ we now have multiple solutions of $a(L)$, $a(0)$. This is possible at high Reynolds number, because the inertial force and the viscous dissipation influence the pressure gradient in opposite ways.

5.12 Velocity Profile of Pulsatile Flow

To obtain the velocity profile of nonstationary flow in a blood vessel, one must solve the equations of motion and continuity of both the blood and the blood vessel wall, and boundary conditions that match the displacements, velocities, and stresses. The calculation is usually lengthy. References to the literature can be found in Fung (1984), McDonald (1974), Patel and Vaishnav (1980), Pedley (1980). In the following, a simple example is given.

Assume that the fluid is homogeneous, incompressible, and Newtonian; the vessel wall is rigid, circular, and cylindrical; the motion is laminar, axisymmetric, and parallel to the longitudinal axis of the tube. A pressure gradient drives the flow, the vessel is horizontal, and gravitation has no effect on the flow. Then the field equations are the Navier–Stokes equations, and the equation of continuity. They are simplified to the following under the conditions named above:

$$0 = -\frac{\partial p}{\partial r}, \tag{1}$$

$$0 = -\frac{\partial p}{\partial \Theta}, \tag{2}$$

$$\rho\frac{\partial u}{\partial t} = -\frac{\partial p}{\partial x} + \mu\left(\frac{\partial^2 u}{\partial r^2} + \frac{1}{r}\frac{\partial u}{\partial r}\right), \tag{3}$$

$$\frac{\partial u}{\partial x} = 0. \tag{4}$$

The boundary conditions are the axisymmetry condition at the center and no-slip on the wall, at radius a:

$$\frac{\partial u}{\partial r} = 0 \quad \text{when} \quad r = 0, \tag{5}$$

$$u = 0 \quad \text{when} \quad r = a. \tag{6}$$

Here p stands for pressure; (x, r, Θ) are cylindrical polar coordinates with x in the axial direction; u is the velocity component in the direction of x; t is time. According to Eqs. (1) and (2), p is a function of x and t only. According to (4), u is a function of r and t. On differentiating Eq. (3) with respect to x, one obtains

$$\frac{\partial}{\partial x}\left(\frac{\partial p}{\partial x}\right) = 0. \tag{7}$$

This shows that the pressure gradient must not vary with x. It can be a function of t. For a general periodic motion, one can write

$$\frac{\partial p}{\partial x} = \sum_{n=0}^{N} C_n e^{in\omega t}. \tag{8}$$

On substituting into Eq. (3), one obtains

$$\rho\frac{\partial u}{\partial t} = -\sum_{n=0}^{N} C_n e^{in\omega t} + \mu\left(\frac{\partial^2 u}{\partial r^2} + \frac{1}{r}\frac{\partial u}{\partial r}\right). \tag{9}$$

The term $n = 0$ corresponds to a steady pressure gradient investigated in Sec. 5.10; the solution is given by Eq. (5.10:7). To the other terms in (7), we can try $u(r, t)$ in the form

$$u = \sum_{n=0}^{N} v_n(r) e^{in\omega t} \tag{10}$$

which is periodic. Substituting Eq. (10) into Eq. (9) we see that the resulting equation is satisfied if we set

$$i\rho n\omega v_n = -C_n + \mu\left(\frac{d^2 v_n}{dr^2} + \frac{1}{r}\frac{dv_n}{dr}\right). \tag{11}$$

The boundary condition, Eq. (6), is satisfied if

$$\begin{aligned} v_n &= 0 \quad \text{when} \quad r = a, \\ \frac{\partial v_n}{\partial r} &= 0 \quad \text{when} \quad r = 0. \end{aligned} \tag{12}$$

The general solution of Eq. (11) is

$$v_n(r) = A_n J_0\left(\alpha\frac{r}{a}n^{1/2}i^{3/2}\right) + B_n Y_0\left(\alpha\frac{r}{a}n^{1/2}i^{3/2}\right) + \frac{iC_n}{\rho n\omega}. \tag{13}$$

Here $J_0(kr)$ is the Bessel function of the first kind of order zero of kr, $Y_0(kr)$ is the Bessel function of the second kind of order zero of kr, k being a constant. A_n, B_n are arbitrary constants, and α is a dimensionless quantity known as the Womersley number (Sec. 5.13):

$$\alpha = a\sqrt{\frac{\omega}{\nu}}. \tag{14}$$

To determine A_n, B_n, the boundary conditions, Eq. (12), are used. As r approaches zero, the derivative J_0' approaches zero and Y_0' approaches infinity. Hence B_n must vanish, and the first of Eq. (12) requires

$$A_n J_0(\alpha n^{1/2} i^{3/2}) + \frac{iC_n}{\rho n\omega} = 0. \tag{15}$$

Solving this equation for A_n and substituting into Eq. (13) together with $B_n = 0$, we obtain

$$v_n(r) = \frac{iC_n}{\rho n\omega}\left[1 - \frac{J_0(\alpha \frac{r}{a} n^{1/2} i^{3/2})}{J_0(\alpha n^{1/2} i^{3/2})}\right]. \tag{16}$$

The problem is solved by substituting Eq. (16) into Eq. (10). The velocity profile depends on Womersley number α. An illustration is given in Fig. 5.12:1.

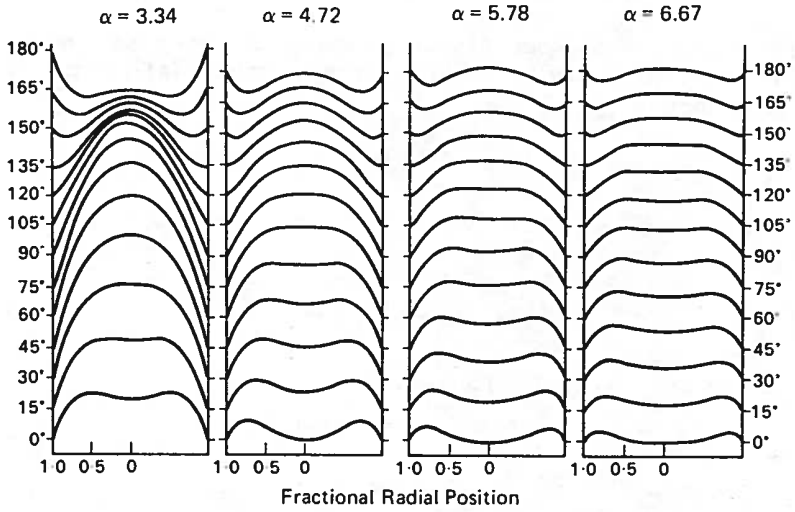


FIGURE 5.12:1 Theoretical velocity profiles of a sinusoidally oscillating flow in a pipe, with pressure gradient varying like $\cos \omega t$. α is the Womersley number. Profiles are plotted for phase angle steps of $\Delta\omega t = 15°$. For $\omega t > 180°$, the velocity profiles are of the same form but opposite in sign. Reproduced with permission from D.A. McDonald, *Blood Flow in Arteries*, copyright © 1974, the Williams & Wilkins Co., Baltimore.

5.13 The Reynolds Number, Stokes Number, and Womersley Number

The general equations of hemodynamics appear formidable. Some essential features can be identified when different terms are compared. The Navier–Stokes equation

$$\rho\frac{\partial u_i}{\partial t} + \rho\left(u_1\frac{\partial u_i}{\partial x_1} + u_2\frac{\partial u_i}{\partial x_2} + u_3\frac{\partial u_i}{\partial x_3}\right) = X_i - \frac{\partial p}{\partial x_i} + \mu\left(\frac{\partial^2}{\partial x_1^2} + \frac{\partial^2}{\partial x_2^2} + \frac{\partial^2}{\partial z x_3^2}\right)u_i, \tag{1}$$

represents the balance of four kinds of forces. Term by term, they are

$$\begin{matrix}\text{transient}\\ \text{inertial}\\ \text{force}\end{matrix} + \begin{matrix}\text{convective}\\ \text{inertial}\\ \text{force}\end{matrix} = \begin{matrix}\text{body}\\ \text{force}\end{matrix} + \begin{matrix}\text{pressure}\\ \text{force}\end{matrix} + \begin{matrix}\text{viscous}\\ \text{force}\end{matrix}.$$

Not all the forces are important all the time. In a steady flow the transient inertial force vanishes. In an ideal fluid the viscous force vanishes. In hydrostatic equilibrium all but the body and pressure forces vanish. Simplifications are recognized for these cases.

Compare the transient inertial force term with the viscous force term. To make an estimate, let U be a characteristic velocity, ω a characteristic frequency, and L a characteristic length. Then the first term in Eq. (1) is of the order of magnitude $\rho\omega U$, and the last term is of the order of magnitude $\mu U L^{-2}$. The ratio is

$$\frac{\text{transient inertial force}}{\text{viscous force}} = \frac{\rho\omega U}{\mu U L^{-2}} = \frac{\rho\omega L^2}{\mu} = \frac{\omega L^2}{\nu}. \tag{2}$$

This is a dimensionless number. If it is large, the transient inertial force dominates. If it is small, the viscous force dominates.

The dimensionless number $\omega L^2/\nu$ is a *frequency parameter*, and is called the Stokes' number because its significance was pointed out by George Stokes in 1840. It is better known by its square root,

$$N_W = L\sqrt{\left(\frac{\omega}{\nu}\right)}, \tag{3}$$

which is called Womersley number in honor of J.R. Womersley, who made extensive calculations on pulsatile blood flow in the 1950's. If L is taken to be the radius of the blood vessel, then Womersley's number is often written as α:

$$\alpha = N_W = \frac{D}{2}\sqrt{\left(\frac{\omega}{\nu}\right)}, \tag{4}$$

D being the blood vessel diameter. In large arteries of all but the smallest mammals, the value of α, calculated from the circular frequency of the heart-

beat in rad/sec, is considerably larger than 1. For example, a typical value of α in the aorta of man is 20, in a dog it is 14, in a cat 8, and in a rat 3. Hence in these aortas the inertial force dominates in pulsatile flow.

If α is large, the effect of the viscosity of the fluid does not propagate very far from the wall. In the central portion of the tube the transient flow is determined by the balance of the inertial forces and pressure forces (first and fourth terms in Eq. 1), and the elastic forces in the wall (through the boundary conditions), as if the fluid were nonviscous. We, therefore, expect that when the Womersley number is large, the velocity profile in a pulsatile flow will be relatively blunt, in contrast to the parabolic profile of the Poiseuillean flow, which is determined by the balance of viscous and pressure forces. This is shown in Fig. 5.12:1.

Now compare the convective inertial force term with the viscous force term. With characteristic velocity U and characteristic length L, the order of magnitude of the inertial force is ρU^2, that of the viscous force is $\mu U/L$. The ratio is

$$\frac{\text{inertial force}}{\text{viscous force}} = \frac{\rho U^2}{\mu U/L} = \frac{\rho UL}{\mu} = \text{Reynolds number.} \qquad (5)$$

A large Reynolds number signals a preponderant inertial effect. A small Reynolds number signals a predominant viscous force effect. In aorta of man the Reynolds number based on vessel diameter can be 2,000–3,000, large enough to cause possible turbulence (Sec. 5.10). In the capillary blood vessels, the Reynolds number is in the order of 0.001 to 0.01, so small that it suggests complete insignificance of the inertial force.

The occurrence of turbulence in a pulsatile flow in the aorta could be transient. Even when the condition of flow favors the transition of a laminar flow into turbulent, the actual transition into turbulence would require a certain amount of time to develop. Hence if the flow velocity fluctuates too fast, the turbulence may not develop. Similarly, if a flow is turbulent but the condition has changed to favor a transition into laminar flow, the actual transition may lag behind for a while.

Quantitative studies of the laminar–turbulent transition may seek to express the critical Reynolds number as a function of the Womersley number. Experimental results can be plotted as shown in Fig. 5.13:1. The ordinate is the peak Reynolds number. The stippled area indicates the conditions under which the flow is stable and laminar.

In the experiments whose results are shown in Fig. 5.13:1, the wide variations of velocity and heart rate were obtained with drugs and nervous stimuli in anesthetized dogs. In normal, conscious, free-ranging dogs the peak Reynolds number usually lies in an area high above the stippled area of Fig. 5.13:1. This suggests that some turbulence is generally tolerated in deceleration of systolic flow in the dog.

Turbulence in blood flow implies fluctuating pressure acting on the arterial wall, and fluctuating, increased shear stress. These stresses are implicated in murmurs, post-stenotic dilation, and atherogenesis.

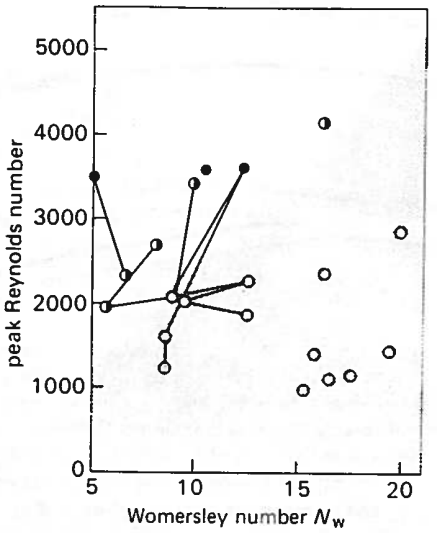


FIGURE 5.13:1 The stability of blood flow in the descending aorta of anesthetized dogs is influenced by the peak Reynolds number and the Womersley number. Points joined by the lines refer to the same animal. Open circles: laminar flow; filled circles: turbulent flow; half-filled circles: transiently turbulent flow. From Nerem, R.M., and Seed, W.A. (1972), by permission.

5.14 Equation of Balance of Energy and Work

According to the principle of conservation of energy, the rate of gain of energy of a material system (the sum of the kinetic, potential and internal energies) must be equal to the sum of the rate at which work is done on the system and heat transported in. Apply this principle to a body of blood contained in a blood vessel between two arbitrary cross sections, 1 and 2, perpendicular to the vessel axis, as illustrated in Fig. 5.14:1. Let p denote the pressure, u denote the axial component of the velocity of flow, q denote the magnitude of the velocity vector, $\dot{Q}$ denote the volume rate of flow. Let dA denote a small element of area in a cross section. At the left end, section 1, the outward normal vector of the cross section points to the left, the force acting on the area dA due to the pressure, pdA, points to the right. The positive direction of the axial velocity u point to the right. The rate of work done by the force due to pressure is $pudA$. The total work done by the force over the entire cross section is, therefore

$$\int_{A_1} pu\,dA \qquad (1)$$

where the integral is taken over the area A_1 of the cross section No. 1.

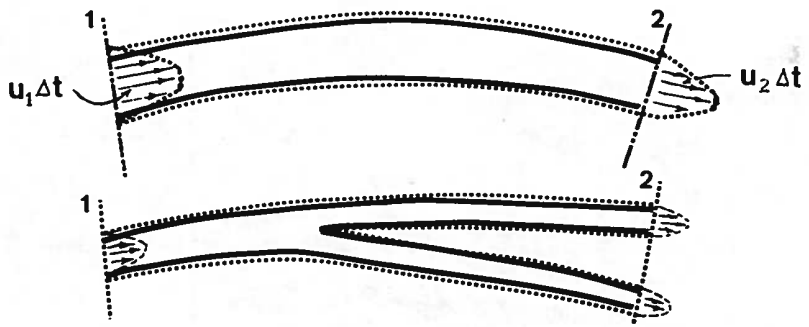


FIGURE 5.14:1 Two arbitrary cross sections, 1 and 2, of a blood vessel. At an instant of time t, a control volume of blood is bounded by the plane sections 1 and 2 and the wall of the blood vessel shown by solid lines. An infinitesimal time Δt later, the boundary of the control volume becomes that shown by the dotted line: consisting of two paraboloidal surfaces at sections 1 and 2, and a distended blood vessel wall. The equations of motion, continuity, and energy are written for the fluid in the control volume. The symbols u_1 and u_2 represent axial component of the velocity at stations 1 and 2, respectively.

The kinetic energy per unit volume of blood is $\frac{1}{2}\rho q^2$ where ρ is the density of the blood. The total kinetic energy of the blood contained in the volume between the sections 1 and 2 is, at time t,

$$\int_V \frac{1}{2}\rho q^2\, dv. \tag{2}$$

A short time Δt later, the same body of fluid would occupy a slightly different volume which is bounded by the dotted lines shown in Fig. 5.14.1. The side wall distends a little because of vessel wall elasticity. The fluid particles composing the cross section 1 are displaced by a distance $u\Delta t$ to the right. The plane cross section No. 1 becomes curved and bulges to the right. The particles at Section 2 are also displaced to the right by the amount $u\Delta t$. During the time interval Δt, therefore, the total kinetic energy of the blood is changed by the amount

$$\int_{V'} \frac{\partial}{\partial t}\left(\frac{1}{2}\rho q^2\right)\Delta t\, dV - \int_{A_1} \frac{1}{2}\rho q^2 u\Delta t\, dA + \int_{A_2} \frac{1}{2}\rho q^2 u\Delta t\, dA. \tag{3}$$

Where V' is the volume bounded by the dotted lines. The rate of change of kinetic energy is obtained by dividing the quantity above with Δt. The $-$ and $+$ signs in the expression (3) should be carefully noted.

A similar consideration should be given to the work done by force imposed on the blood by the blood vessel wall, the potential energy change due to

gravitation, the internal energy change due to temperature change, the heat transported through the boundary, and the rate of heat generation due to internal friction equal to the sum of all the products of stress components and the corresponding strain rates. The last term, the heat dissipation, is denoted by $\mathscr{D}$:

$$\mathscr{D} = \int_V \sigma_{ij} V_{ij}\, dv. \tag{4}$$

Where σ_{ij} is the stress tensor and V_{ij} is the strain rate tensor.

Now, on equating the change of energy with the work done, and dividing through by the volume flow rate $\dot{Q}$:

$$\dot{Q} = \int_A u\, dA \tag{5}$$

we obtain, if gravitational effect and heat transfer were negligible, the following equation:

$$\widehat{p_1} - \widehat{p_2} = \frac{1}{2}\rho\widehat{q_2^2} - \frac{1}{2}\rho\widehat{q_1^2} + \rho g h_2 - \rho g h_1 + \frac{\mathscr{D}}{\dot{Q}} + \frac{1}{\dot{Q}}\int_V \frac{\partial}{\partial t}\left(\frac{1}{2}\rho q^2\right) dv. \tag{6}$$

Here the velocity-weighted pressure $\widehat{p}$ and the square of velocity $\widehat{q^2}$ are defined by dividing Eqs. (1) and (2) by $\dot{Q}$:

$$\widehat{p} = \frac{1}{\dot{Q}}\int_A pu\, dA, \qquad \widehat{q^2} = \frac{1}{\dot{Q}}\int_A q^2 u\, dA. \tag{7}$$

Note that $\widehat{p}$ and $\frac{1}{2}\rho\widehat{q^2}$ have the dimensions of pressure.

The energy equation (6) was given by Pedley et al. (1977) and derived in full detail in Fung (1984), pp. 15–20. It is used frequently in this book.

5.15 Systemic Blood Pressure

If we apply the results derived in the preceding sections to a circuit of blood vessels beginning at the aortic valve and ending in the right atrium, take the average of the pressure-flow relationship of every segment over a period of time which is long compared with a single heart beat, and synthesize the segments into a whole circuit, then we obtain the result:

$$\text{Average pressure at aortic valve} - \text{average pressure at right atrium} = \text{integrated frictional loss.} \tag{1}$$

This is often written as:

$$\text{Systemic arterial pressure} = \text{flow} \times \text{resistance.} \tag{2}$$

Here the systemic arterial pressure is the difference between the pressure at the aortic valve and that at the vena cava at the right atrium, the flow is the

cardiac output, and the resistance is the *total peripheral vascular resistance*. Hence, writing in greater detail, we have

$$\text{Pressure at aortic valve} - \text{pressure at right atrium} = (\text{cardiac output}) \times (\text{total peripheral vascular resistance}), \tag{3}$$

where

$$\text{Total peripheral vascular resistance} = \frac{\text{integrated frictional loss}}{\text{cardiac output}}. \tag{4}$$

The last term in Eq. (3) represents the sum of the pressure drops due to the friction loss along all segments of blood vessels. Since there are millions of capillary blood vessels in the body, there are millions of pathways along which one can integrate the equation of motion to obtain Eq. (3), so the final result Eq. (3) is useful only if the pressures at the aortic valve and right atrium are uniform no matter which path of integration is used. Fortunately, this is the case.

The integrated frictional loss is the sum of frictional losses in all segments of vessels of the circuit. To compute the frictional loss of a segment, let us first consider a steady laminar flow (i.e., one that is not turbulent) in a long, rigid, circular, cylindrical vessel. To such a flow, Poiseuille's formula, Eq. (5.10:8) applies. Let the vessel length be L and the vessel diameter be d, then

$$\dot{Q} = -\frac{\pi d^4}{128}\frac{\Delta p}{\mu L}. \tag{5}$$

Here μ is the coefficient of viscosity of the fluid, and Δp is the pressure drop. Equation (5) can be written as

$$\Delta p = (\text{laminar resistance in a tube}) \times (\text{flow in the tube}), \tag{6}$$

from which we obtain the resistance of a steady laminar flow in a circular cylindrical vessel:

$$\text{laminar resistance in a tube} = \frac{128\mu L}{\pi d^4}. \tag{7}$$

If the nth generation of a vascular tree consists of N identical vessels in parallel, then the

$$\text{Pressure drop in the } n\text{th generation of vessels} = (\text{resistance in } N \text{ parallel tubes}) \times (\text{total flow in } N \text{ tubes}) = \frac{(\text{resistance in one tube})}{N} \times (\text{cardiac output}). \tag{8}$$

Note that according to Eq. (7) the laminar flow resistance is proportional to the coefficient of viscosity μ and the length of the vessel L, and inversely proportional to the fourth power of the diameter d. Obviously the vessel

diameter d is the most effective parameter to control the resistance. A reduction of diameter by a factor of 2 raises the resistance 16-fold, and hence leads to a 16-fold pressure loss. In peripheral circulation the arterioles are muscular and they control the blood flow distribution by changing the vessel diameters through contraction or relaxation of the vascular smooth muscles.

Equation (7) gives the resistance to a Poiseuillean flow in a pipe and for a given flow rate $\dot{Q}$ is the minimum of resistance of all possible flows in the pipe. If the flow becomes turbulent, the resistance increases. If the blood vessel bifurcates, the local disturbance at the bifurcation region raises resistance. In these deviations from the Poiseuillean flow the governing parameter is the Reynolds number. If a flow is turbulent, then

$$\text{Resistance of a turbulent flow in a vessel} = (\text{laminar resistance}) \cdot (0.005\, N_R^{3/4}). \tag{9}$$

Thus, if the Reynolds number is 3,000, the resistance of a turbulent flow is over two times that of the laminar resistance. In the ascending and descending aorta of man and dog the peak Reynolds number does exceed 3,000. The energy loss that occurs at points of bifurcation, entry flow, flow separation, etc., are also functions of Reynolds number. In these cases one writes the pressure–flow relationship as

$$\dot{Q} = \frac{1}{Z(N_R)}\frac{\pi d^4}{128}\frac{\Delta p}{\mu L}, \tag{10}$$

where $Z(N_R)$ is a dimensionless function of the Reynolds number. Equation (9) shows that for a turbulent flow $Z(N_R)$ is equal to $0.005\, N_R^{3/4}$. Other examples are given in Sec. 7.2, especially Eqs. (7.2:4)–(7.2:6). All the results of fluid mechanics research on flow resistance in pipes can be packed into the function $Z(N_R)$.

Equation (3) or (8) shows the basic factors that control the systemic blood pressure. The resistance is proportional to the blood viscosity. Hence lowering the coefficient of viscosity will promote the flow. Hemodilution is thus a practical clinical tool. The resistance is sensitive to the diameter of the blood vessel. The diameter is controlled by the vascular smooth muscle. Hence the control of smooth muscle behavior is the key to the treatment of hypertension.

5.16 The Veins and Their Collapsibility

Veins are similar to arteries in size and construction, but veins have valves and smaller wall thickness to diameter ratio. In fact the wall thickness of veins is often quite nonuniform around the circumference. Because veins have thinner walls, they are more compliant than the arteries. Because the blood pressure is low in the veins, they are more sensitive to external pressure. If the external pressure exceeds the internal pressure by an amount known as the *critical buckling pressure*, then a vein will collapse. Normally, 80% of a man's

blood is in the veins. For this reason veins are said to be capacitance vessels. The capacitance is sensitive to internal and external pressures. Thus raising one's leg or moving the leg muscles will reduce the blood volume in the legs, pushing blood to the heart and circulating to other parts of the body.

The collapsibility of the vein gives the venous blood flow some unique features. In dynamic condition the *transmural pressure* (Δp = internal − external pressures) acting on a vein may be a) positive throughout, b) negative and exceeding the critical buckling pressure throughout, or c) positive at the entry section, but negative and exceeding the critical buckling pressure at the exit section. Then in condition a) the vein is patent, in b) it is collapsed, whereas in c) something special will happen. If in condition c) the exit end is collapsed, then the flow would stop, the pressure drop would become zero, the whole tube would have a Δp equal to that of the entry section, the condition of a) would then prevail, and flow would start again. But if flow starts, the pressure will drop along the tube, and the exit section may be choked again. This may lead to a dynamic phenomenon of "flutter,", or to a limiting steady flow controlled by a narrowed section. In the last case, the actual value of Δp at the exit section is quite immaterial as long as the cross sectional area at the exit section is much reduced. An analogy may be drawn between this and the waterfall in our landscape, or sluicing in industry or flood control: The volume flow rate in a waterfall depends on the conditions at the top of the fall, and is independent of how high the drop is. Thus, the phenomenon of flow in case c) is described as the "waterfall" phenomenon, or as sluicing.

The waterfall phenomenon occurs in a number of important organs: the lung, the vena cava, etc. It occurs in thoracic arteries during resuscitation maneuvers, and in brachial arteries while measuring blood pressure by cuff and Korotkov sound. The same phenomenon also occurs in male and female urethra in micturition, and in manmade instruments such as the blood pump and the heart–lung machine.

Since so much depends on the collapsibility, let us consider the mechanical property of blood vessels at negative transmural pressure in greater detail.

Moreno et al. (1970) measured the change of the cross-sectional area of dog's vena cava when the transmural pressure was varied. Shapiro (1977) measured the same in latex tubing. The characteristics of the vessel and tube deformations are similar. Shapiro's results are shown by the solid curve in Fig. 5.16:1. If the tube were circular cylindrical and of Hookean elastic material when the transmural pressure is zero, then the elastic stability of the tube is amenable to mathematical analysis. The theoretical results of Flaherty et al. (1972) are shown in Fig. 5.16:1 by the curve with long dashes. Theoretically, the pressure-area curve has a sudden change of slope at each critical transmural pressure. The deformation pattern changes when the transmural pressure exceeds the critical value. If one defines the dimensionless variables

$$\tilde{p} = \frac{12(1-\nu^2)R^3}{Eh^3}(p - p_e) \quad \text{and} \quad \alpha = \frac{A}{\pi R^2} \tag{1}$$

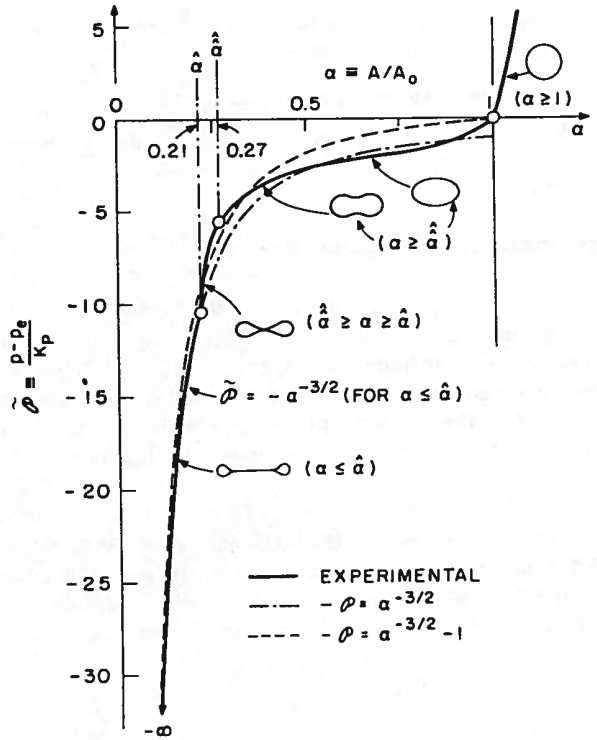


FIGURE 5.16:1 Behavior of a collapsible tube. Dimensionless transmural pressure difference, $\tilde{p}$, versus dimensionless area ratio, α. Solid curve shows a typical experimental curve for thin-walled latex tube, and adjacent to it, typical cross-sectional shapes for the different ranges of α. Dot-dash curve represents Eq. (8), coincides with solid curve for $\alpha < \hat{\alpha}$. Dashed curve represents Eq. (10). Curve with long dashes represents the theoretical result given by Flaherty et al. for cylinders whose cross sections are perfectly circular when $\tilde{p} = 0$. Point contact occurs at $\alpha = \hat{\hat{\alpha}}$, and line contact occurs at $\alpha = \hat{\alpha}$. From Shapiro (1977), by permission.

in which p represents internal pressure, p_e is the external pressure, E is the Young's modulus of the tube wall material, ν is its Poisson's ratio, R is the tube radius at midwall, h is the tube wall thickness, A is the cross-sectional area of the lumen, then Flaherty et al. showed that the buckling occurs when $\tilde{p} < -3$. When $\tilde{p} = 5.247$, the opposite walls touch each other at the midpoint. Upon further increase in external pressure, the contact area increases and the open portion of the cross section is reduced in size but remains similar in shape. For this "self-similar" type of deformation Flaherty et al. obtained the relationship

$$-\tilde{p} = \alpha^{-3/2}. \tag{2}$$

Noticing the difference between the experimental curve and the theoretical curve, Shapiro (1977) proposed an empirical formula

$$-\tilde{p} = \alpha^{-3/2} - 1. \tag{3}$$

Now let us see something different. Figure 6.6:5 on p. 209 shows the thickness of the pulmonary capillary sheet in the interalveolar septa as a function of the transmural pressure. These pulmonary capillary blood vessels form a dense network whose "thickness" varies with the blood pressure, whereas the dimension in the plane of the interalveolar septa is unaffected by

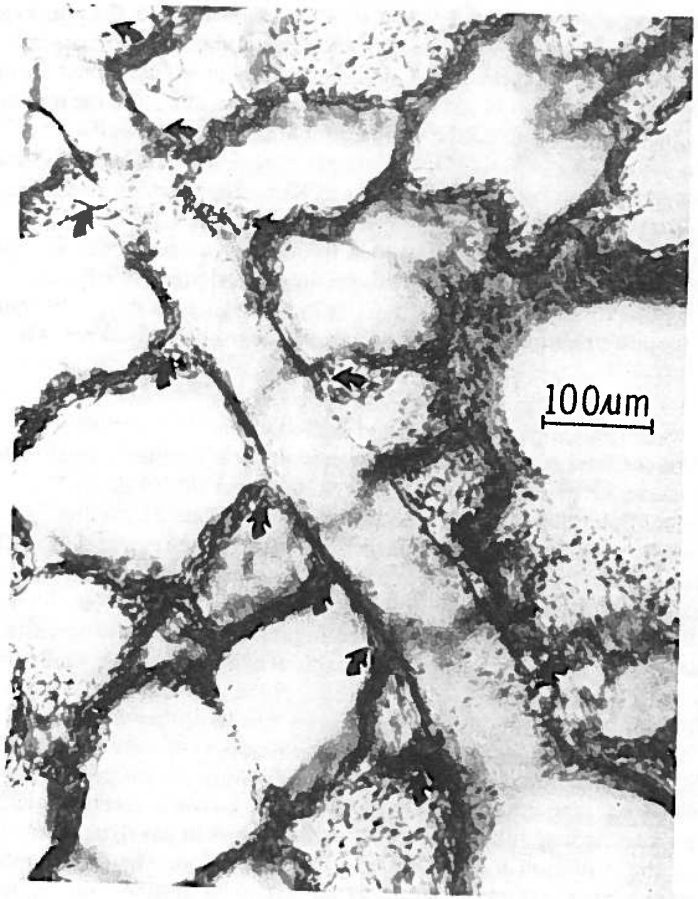


FIGURE 5.16:2 The connection between a pulmonary vein and interalveolar septa in cat's lung. Courtesy of Dr. Sidney Sobin.

the blood pressure. The difference between the blood pressure and the airway pressure is defined as the transmural pressure. Fung et al. (1972) have shown that the thickness drops very rapidly to zero when the transmural pressure changes from positive to negative values.

On the other hand, Fig. 6.6:4 on p. 208 shows the diameter versus transmural pressure relationship of pulmonary veins (Yen and Fappiano, 1981). It is seen that the relationship can be represented by straight lines. The slope of the straight line does not change when Δp changes from the positive to negative value. These veins would not collapse under negative transmural pressure in the physiological range (Fung et al., 1983).

Thus the elastic stability characteristics of the pulmonary capillaries is similar to that of the vena cava, but that of the pulmonary vein is entirely different from that of the vena cava. Not all veins are alike! The difference is actually easily explained. The vena cava was tested as an isolated tube. The pulmonary veins were, however, tested intact, embedded in the lung parenchyma which was in tension. The lung parenchyma provides an elastic support to the pulmonary veins.

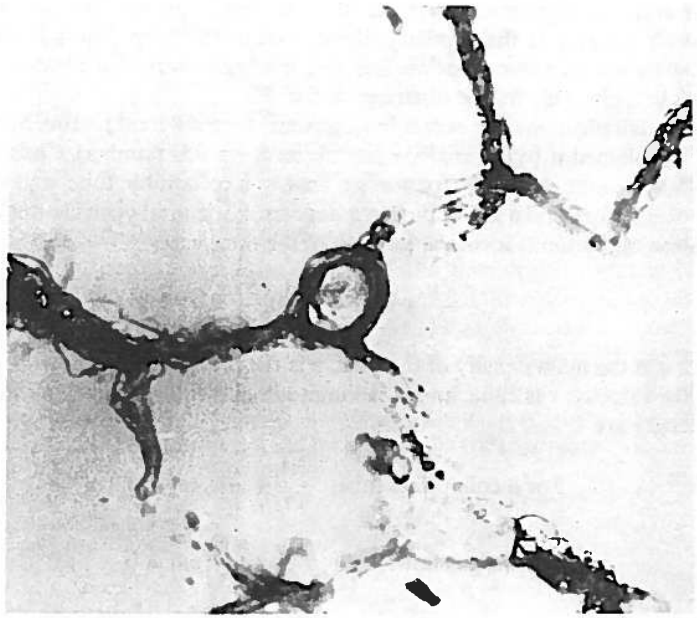


FIGURE 5.16:3 A photo micrograph of cat lung showing a venule tethered by three interalveolar septa. Vasculature perfused with a catalyzed silicone elastomer and hardened; gelatin-embedded; cresyl violet stained. From Fung et al. (1983). Reproduced by permission.

This elastic support can be seen from the photomicrographs of the lung parenchyma. In Figure 5.16:2 is shown a histological section of a cat lung parenchyma containing a large blood vessel. The lacy tissue tethering the outer wall of the blood vessel is the alveolar structure, which is in tension in an inflated lung. Thus the blood vessel is embedded in a foam-rubber-like material. In Figure 5.16:3, at a larger magnification, is shown a histological section of cat lung parenchyma containing a pulmonary arteriole. The diameter of the arteriole is about 25 μm, which is small compared with the dimension of the alveoli of the cat, about 10 μm. See that the arteriole is pulled by three interalveolar septa. These septa are in tension in an inflated lung, they tend to distend the arteriole. When the alveolar gas pressure outside the arteriole exceeds the blood pressure in the arteriole, the tendency for the vessel to collapse is resisted by the tension in the interalveolar septa attached to the outer wall of the blood vessel.

5.17 Flow in Collapsible Tubes

In circulatory physiology, flow in blood vessels in collapsible condition may occur either in microcirculation, or in large vessels. In microcirculation, a common example is the capillary blood flow in the lung. The pulmonary capillaries are readily collapsible (see Fig. 6.6:5) and waterfall phenomenon occurs in them. This will be discussed in Sec. 6.8.

Waterfall phenomenon occurs in large veins for a different reason. Shapiro (1977) explained it by an analogy (at infinite Reynolds number). Consider a one-dimensional, unsteady, frictionless flow in a collapsible tube, a gas flow in a wind tunnel, and a liquid flow in a uniform, horizontal open channel. The equation of motion is identical for each of the three cases:

$$\frac{\partial u}{\partial t} + u\frac{\partial u}{\partial x} = -\frac{1}{\rho}\frac{\partial p}{\partial x}, \tag{1}$$

where ρ is the mass density of the fluid, p is the pressure in the flowing fluid, u is the velocity, t is time, and x is longitudinal distance. The equations of continuity are

$$\text{For a collapsible tube:} \quad \frac{\partial A}{\partial t} + \frac{\partial}{\partial x}(Au) = 0; \tag{2}$$

$$\text{For the gas flow:} \quad \frac{\partial \rho}{\partial t} + \frac{\partial}{\partial x}(\rho u) = 0; \tag{3}$$

$$\text{For the channel flow:} \quad \frac{\partial h}{\partial t} + \frac{\partial}{\partial x}(hu) = 0. \tag{4}$$

Here A is the cross-sectional area in the first case, ρ is the mass density in the second case, and h is the height of the free surface above the bottom in the

third case. The phase velocity of propagation of small perturbations, c, is, in the three cases:

$$c^2 = \frac{A}{\rho}\frac{d(p - p_e)}{dA}; \tag{5}$$

$$c^2 = \left(\frac{dp}{d\rho}\right) \quad \text{at constant entropy;} \tag{6}$$

$$c^2 = \frac{h}{\rho}\frac{dp}{dh} = gh; \tag{7}$$

where g is the gravitational acceleration. Thus the analog is seen. Those readers who are familiar with gas dynamics may recall the shock waves, the supersonic wind tunnel, the Laval nozzle for steam turbine, the convergent section to accelerate the fluid in subsonic regime, the sonic throat, and the divergent section to accelerate the fluid in supersonic regime. Those familiar with the open channel flow may recall the flow over a dam, and the hydraulic jump. One could anticipate the existence of analogous phenomena in blood flow in collapsible vessels. One anticipates also, of course, that similar phenomena occur in air flow in the airways, Korotkov sound in arteries, urine flow in urethra, etc.

For a flow from a reservoir with a fixed total pressure head p_0 into a collapsible tube, the flow rate depends on the suction pressure p downstream and the pressure outside the tube p_e. The rate of change of flow with respect to the suction pressure p is given by the equation (to be derived below):

$$\frac{d\dot{Q}}{d(p - p_e)} = \frac{A}{\rho u}\left(\frac{u^2}{c^2} - 1\right). \tag{8}$$

Here $\dot{Q}$ is the flow rate, p is the internal pressure, u is the mean speed of flow, and c is the speed of the flexural wave. Note that $d\dot{Q}/dp$ depends on the ratio u/c. If the flow speed u is smaller than c, then decreasing internal pressure increases the flow. If u is larger than c, then the reverse is true. Thus the condition $u = c$ signifies the maximum flow obtainable with decreasing internal pressure. This maximum is $Q_{max} = Ac$. At this condition, the maximum flow depends neither on the upstream pressure, nor on the downstream pressure. It is an exact analog of the sonic throat of the supersonic wind tunnel. The ratio

$$S = \frac{u}{c} \tag{9}$$

is called the *speed index* of Shapiro (1977). It plays a central role in liquid flow through a collapsible tube as the Mach number does in gas dynamics.

Thus the condition $u = c$ signifies a flow limitation. The upstream and downstream pressures matter only to the extent of getting this condition established, just as a supersonic wind tunnel has to have suitable conditions to get it started.

The derivation of the intriguing Eq. (8) is as follows. Consider laminar flow in an elastic tube at a large Reynolds number so that Bernoulli's equation holds:

$$p + \tfrac{1}{2}\rho u^2 = p_0, \tag{10}$$

where p_0 is the stagnation pressure, p is the static pressure, ρ is fluid mass density, and u is velocity. The volume flow rate, $\dot{Q}$ is

$$\dot{Q} = Au = A\sqrt{\tfrac{2}{\rho}(p_0 - p)} = A\sqrt{\tfrac{2}{\rho}[(p_0 - p_e) - (p - p_e)]}. \tag{11}$$

A is the cross-sectional area which is a function of $p - p_e$. Although p varies with distance down the tube, $\dot{Q}$ remains constant, of course. Differentiating $\dot{Q}$ with respect to $p - p_e$, we obtain, after some reduction and using Eq. (10),

$$\frac{d\dot{Q}}{d(p - p_e)} = -A\frac{du}{dp} + \frac{dA}{d(p - p_e)}u = -\frac{A}{\rho u} + \left[\frac{\rho}{A}\frac{dA}{d(p - p_e)}\right]\frac{A}{\rho}u. \tag{12}$$

The factor in the bracket of the last term is $1/c^2$. Thus Eq. (8) is obtained.

Shapiro (1977) analyzed a number of cases in which these equations apply. Experience shows that this one-dimensional analysis is adequate to deal with the flow leading to the sonic throat. But beyond the sonic throat, the recovery of flow to the subsonic condition seems to be a three-dimensional phenomenon beyond the reach of the simplifed approach.

5.18 Pulse Wave as Message Carrier for Noninvasive Diagnosis

A subsonic flow is influenced by conditions on all of its boundary. The flow field, governed by the equations of motion and continuity and the constitutive equations, is determined by the conditions on the boundary. Anything happening anywhere on the boundary will be felt everywhere in the flow field. In fluid mechanics and the theory of partial differential equations, this is a feature of potential flow or elliptic differential equations, as distinguished from supersonic flow or hyperbolic differential equations. Now, blood flow is subsonic. Therefore, if we have the full, detailed mathematical solution of the flow field, then, in principle, by examining the flow at a given region, one should be able to tell any disturbances occurring anywhere on the boundary. Extending this concept to the diseases of the blood vessels and organs, we can anticipate that the pulse waves in a given region of an artery should carry information about stenosis, aneurysm, or atherosclerosis at distant places.

The object of studying the messages carried in arterial pulse waves is similar to the use of seismic waves to detect oil reserves underground. The mathematical problem has not been solved yet, but anecdotal, empirical information exists.

In the traditional Chinese medicine, physicians use fingers to feel the pulse waves of the radial artery on the forearm at the wrist. Through empirical information accumulated over the years, they have developed an art of

diagnosis which is often marvelous but not well understood. The use of pulse waves for diagnosis was discussed extensively in one of the most ancient classics of medicine: the *Nei Jing*, i.e., the *Internal Classic of Huangti* (for Chinese references see Fung, 1984, pp. 14 and 157; and Xue and Fung, 1989) which is believed to have been written in the Warring Period (475–221 BC). In essence, the idea is that all disturbances in the function of any organ can be detected by changes in the pulse waves in the radial artery. The sensation felt by the fingers when they press on the radial artery at specified points with varying degrees of pressure is used as diagnostic criterion.

The pulse wave diagnosis method is studied intensively in China clinically, experimentally, and theoretically. In older literature the waves are treated as axisymmetric motion in circular cylindrical elastic tubes. Recent literature has included articles treating non-axisymmetric motion, including lateral oscillation of the centerline of the blood vessel. Dai et al. (1985) tested the hypothesis that a disturbance of blood flow at one place can be detected in the arterial pulse wave at a distant site. They transiently occluded blood flow in a leg and recorded the pulse waves in both radial arteries. They asked whether the right and left radial arterial waves can differentiate a disturbance in the right leg from that in the left leg. The results show that the right and left radial arterial waves do respond to the disturbances in the right and left legs differently, but the discrimination is not very strong. Xue and Fung (1989) tried to explain it on the basis of fluid mechanics. They created an unsymmetrical entry condition by blocking off one-half of the entry section of a circular cylindrical tube. As the distance from the entry section increases, the flow tends to become axisymmetric, but there is an asymmetric component which persists in propagating downstream with slowly damped amplitude. This suggests that the asymmetric flow condition from the legs may reach the arms, but whether the suggestion is quantitatively meaningful or not is entirely unknown. This remains a fascinating problem.

Problems

5.1 An energy balance equation for blood flow is desired. Consider all the arteries between two planes, for example, one plane cutting a renal artery, the other plane cut through the kidney supplied by that renal artery. Identify the rate of gain of energy of the blood in these arteries (the sum of the kinetic, potential, and internal energies) and the rate at which work is done on the blood in this system. The energy balance requires that the rate of gain of energy must be equal to the sum of the rate of work done on the system and the heat transported in. Express this energy equation in terms of pressure and velocities in the system. Cf. Fung (1984) pp. 15–20.

5.2 One of the great achievements of man in the twentieth century is the mechanical heart. What is the present status of the art in this field? What do you think needs to be done in order to make this device really available to more people at an affordable price?

5.3 What effect does a stenosis in a large artery have? To study the effect, laser-doppler velocimeter may be used. Describe the principle of this instrument. Can it be used for an unsteady flow?

5.4 Describe the theoretical criterion for the velocity distribution in the boundary layer when boundary separation from a solid wall occurs. (Cf. Yih (1977) pp. 352, 360.)

5.5 Consider the pulsatile flow in the aortic arch, part of which is highly curved as a torus. Because of the curvature of the vessel, secondary flow exists and the boundary layer thickness is a function of time and space. For the consideration of atherogenesis, we need to know the shear stress on the arterial wall. Give a qualitative discussion on the nature of variation of the flow and shear stress in the aortic arch. (Cf. Jayaraman, G., Singh, M.P., Padmanabhan, N. and Kumar, A. (1984). "Reversing flow in the aorta: a theoretical model", *J. Biomechanics*, **17**: 479–490.)

5.6 An aorta has an aneurysm, which is a sac formed by the dilatation of the wall of the artery. From the point of view of fluid mechanics, discuss the pulsatile velocity distribution and pressure in the aneurysm and its contiguous parts, and the possible sound emission (bruit, aneurysmal bruit). From the point of view of solid mechanics, discuss the stress distribution in the vessel wall. From the general biological relation between stress and growth or resorption, discuss possible reasons for the creation of the aneurysm and possible direction of its development. (Cf. Chapters 10–13 infra.)

5.7 Discuss the stress distribution in the endothelium, the intima, and the adventitia in the region of arterial bifurcation. Delve into further detail, considering the stresses acting in the endothelial cells, smooth muscle cells, collagen fibers of various kinds, elastin fibers, fibronectin, and ground substances in the vessel wall. Again, precise data are lacking. Develop a research proposal to clarify this problem. Again, cf. Chapters 10–13, and biological points of view as mentioned in Prob. 5.6.

5.8 Looking at the stenosis problem of 5.3 from the point of view of solid mechanics and biology as mentioned in Prob. 5.6, discuss the possible remodeling of the blood vessel wall when a stenosis develops.

5.9 The place where an artery branches off from the aorta is often the site of atherosclerosis. Discuss qualitatively the velocity distribution, fluid pressure, and wall shear stress on the endothelium in this region. Develop a plan of research to gain a better understanding of these features.

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