

©Roger G. Mark, 2003 (adapted from original notes by Prof. Roger Kamm)

MASSACHUSETTS INSTITUTE OF TECHNOLOGY

Departments of Electrical Engineering, Mechanical Engineering, and the Harvard-MIT Division
of Health Sciences and Technology

6.022J/2.792J/HST542J: Quantitative Physiology: Organ Transport Systems

PHYSIOLOGICAL FLUID MECHANICS

PHYSIOLOGICAL FLUID MECHANICS

As a preliminary to the discussion of hemodynamics, we need to develop some basic fluid dynamic concepts. The discussions of this section pertain to fluid flow in general and are therefore useful in a much broader context than the more specific topics to be discussed in subsequent sections. For the sake of simplicity, however, some subtleties of fluid dynamics, which are of little practical import to blood flow, will be overlooked. Those wanting a somewhat more thorough presentation but still on an elementary level, should look in any basic text in fluid dynamics.

1. Forces acting on a small element

We first need to establish a convention in referring to forces of various types which may act on a fluid or solid element. If we isolate a small cubical element of some substance (as shown in Figure 1), we can distinguish three different types of forces. The first two act on the surfaces of the element, and are by convention termed the **pressure force** (associated with the direction normal to the surface) and a **shear force** (acting parallel to the surface). It is often more convenient to speak in terms of a force per unit surface area, termed the **pressure** (p) and **shear stress** (t) for the normal and tangential directions, respectively. In three dimensions, it is necessary to identify nine stress components in order to completely define the *state-of-stress* at a point in the fluid. These nine stress components comprise the *stress tensor*. We denote the various components of the stress tensor by t_{ij} where i is the face of a small cubic fluid element on which the stress acts (the “direction” of a face is determined by a vector *normal* to the surface), and j is the direction in which the stress points. For example, t_{yx} is a shear stress acting upon the y -face in the x -direction as shown in Figure 1. The third type of force is a **body force** which acts uniformly throughout the fluid and is usually expressed in terms of a force per unit volume. Gravity is the only body force we will consider.

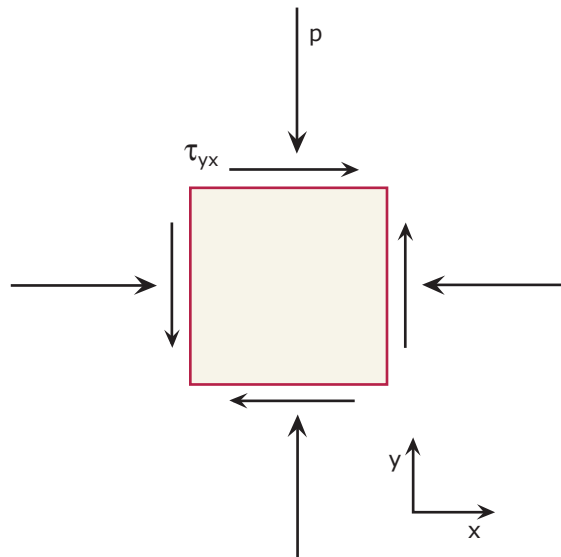


Figure by MIT OCW.

Figure 1: Forces acting on a small element. Pressure is normal to the surface, while shear stress is tangential.

2. The Properties of a Fluid

We can distinguish between a solid and fluid according to the response of a small element to a shear force. A solid will deform as the force is applied and maintain that deformation until the force is released, at which time it will return to its original configuration. (See Figure 2.) This is termed “elastic deformation”. For an ideal elastic solid (a Hookean solid) the shear deformation or “strain” is proportional to the shear stress, τ . For small strains,

$$\tau = G \frac{l}{h} = G d\gamma$$

The constant of proportionality, G , is the shear modulus of elasticity.

In contrast, if a stress is applied to a *fluid* surface, the fluid will continue to deform at a rate which is proportional to the applied stress. Here the appropriate constitutive relation is between the shear stress and the *rate* of deformation. Taking the one-dimensional case of Figure 3, the rate of deformation, or the “shear rate”, is simply the velocity gradient ($\frac{du}{dy} = \frac{U}{h}$). The constant of proportionality relating shear stress to the rate of deformation is the **coefficient of viscosity**, μ . In the one-dimensional case again, we can write:

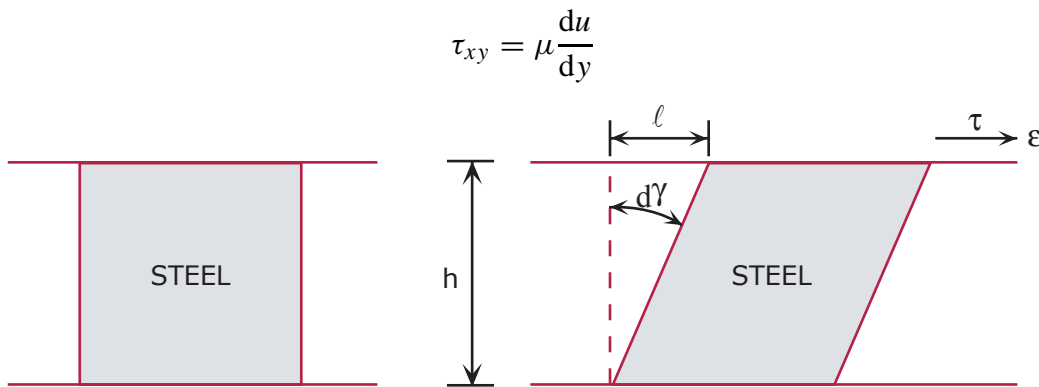


Figure by MIT OCW.

Figure 2: An elastic solid deforms with shear stress but will return to its original configuration.

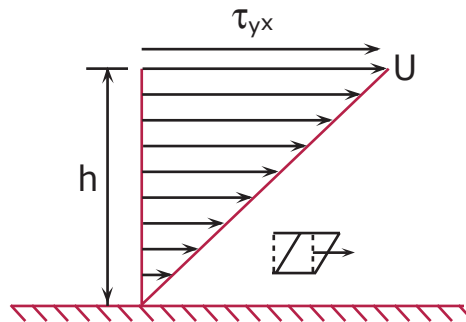


Figure by MIT OCW.

Figure 3: A fluid will deform at a rate proportional to the shear stress. Here the shear rate is the velocity gradient U/h .

In a so-called Newtonian fluid, μ is independent of shear rate (but can depend upon such parameters as temperature, and liquid or gas composition). Many fluids, however, possess the characteristic that μ changes as the shear rate changes and are therefore termed “non-Newtonian” fluids.

A plot of shear stress, τ , versus shear rate is called a *flow curve*. Examples are shown in Figure 4. Curve 1 is for a Newtonian fluid in which viscosity is independent of shear rate. The slope of the curve is the coefficient of viscosity, μ . It has units of dyne-sec/cm² or *poise*. Water has a viscosity of .01 poise or 1 centipoise. Plasma’s viscosity is 1.5 centipoise, and that of whole blood is about 4 centipoise. Curve 2 shows a behavior seen in solutions of high polymers in which the viscosity is a function of shear rate. In the curve shown, the viscosity decreases with increasing shear rates (a “shear-thinning” fluid). The fluid shown in curve 3 behaves as a solid at low levels of shear stress, but begins to flow when some critical “yield stress”, τ_y , is exceeded. If the curve were linear for $\tau > \tau_y$, the material would be known as a “Bingham plastic”.

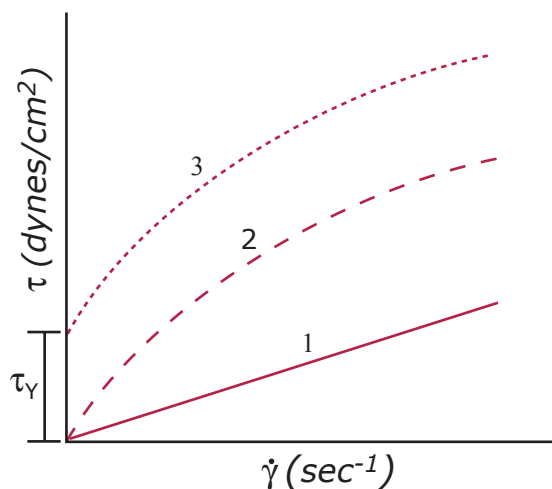


Figure by MIT OCW. After Middleman.

Figure 4: Flow curve of simple types of fluids.

Note that viscosity is usually associated with the “thickness” of a fluid. Motor oil, for example, is considered much “thicker” than water, because it is highly viscous and doesn’t flow easily. The property of “thickness” is quite different from fluid density: both water and mercury are considerably *denser* than oil, but both behave as “thinner” fluids.

An effect associated with fluid viscosity is the *no-slip condition*. It is experimentally observed that whenever a fluid is in contact with a solid surface, the fluid moves at exactly the same velocity as the surface. There is no slippage between the solid surface and the adjacent fluid. (This explains why moving fan blades can accumulate dust.) Ludwig Prandtl, in a classic 1904 paper, proposed this.

2.1 Blood as a Fluid

Blood is a suspension of cells in a solution of electrolytes and proteins. Red blood cells (RBCs) account for most of the cells, and occupy about 50% of the blood volume. (The percent of volume taken up by RBCs is the *hematocrit*.) The non-cellular fluid, separable by centrifugation, is *plasma*.

If blood is allowed to clot, the remaining fluid is called *serum*, which is similar to plasma, but is missing the protein fibrinogen. Red blood cells number about 5×10^6 per mm^3 . They are disk-shaped, measuring about $7.6 \mu\text{m}$ in diameter and $2.8 \mu\text{m}$ in thickness.

Plasma behaves as a Newtonian fluid. Whole blood, however, is non-Newtonian in its properties. Figure 5 shows a flow curve for whole blood. It is a “shear-thinning” curve of the type shown in curve 2 of Figure 4. Two phenomena explain the non-Newtonian flow curve of blood: cell aggregation at low shear rates, and RBC deformation at high shear rates.

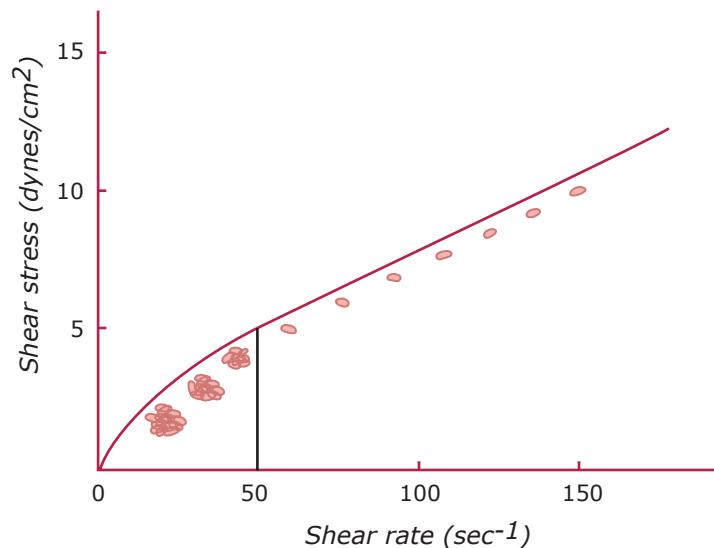


Figure by OCW. After Charm.

Figure 5: Dynamic equilibrium between cell aggregate size and shear stress applied.

In the presence of fibrinogen and globulin RBCs tend to form aggregates known as rouleaux. (In the absence of fibrinogen and globulin such clumping does not occur.) As blood flows, the rouleaux are broken up at increasing shear rates, resulting in smaller particle sizes and lower effective viscosity. An experiment done by Chien illustrates the mechanisms underlying the non-Newtonian behavior of blood. Figure 6 shows the relationship between the viscosity of several fluids and the shear rate. All of the fluids were suspensions containing 45% RBCs by volume. Curve NP is for RBCs suspended in normal plasma, in which aggregates may form at low shear rates. Curve NA is for RBCs suspended in 11% albumin (no aggregates can form). Curve HA is for acetaldehyde-hardened RBCs in albumen (no membrane deformation possible). The data show that the fixed RBC suspension has a constant viscosity at all shear rates tested.

The separation of curves NP and NA suggest that RBC aggregates are broken up to single-cell size at shear rates of about 10 sec^{-1} . The continuing decrease in viscosity as shear rates increase to several hundred suggest that the RBCs become elongated in the presence of high shear, thus decreasing viscosity slightly. (See Figure 7.)

Whole blood may be considered reasonably Newtonian for shear rates greater than 100 sec^{-1} . Estimates of the wall shear rates in various blood vessels are shown in Table 1 below. In all cases wall shear rates are above 100 sec^{-1} .

The viscosity of blood is a strong function of the hematocrit. Figure 8 illustrates the relative viscosity of blood as a function of hematocrit. The figure also illustrates the changes in viscosity

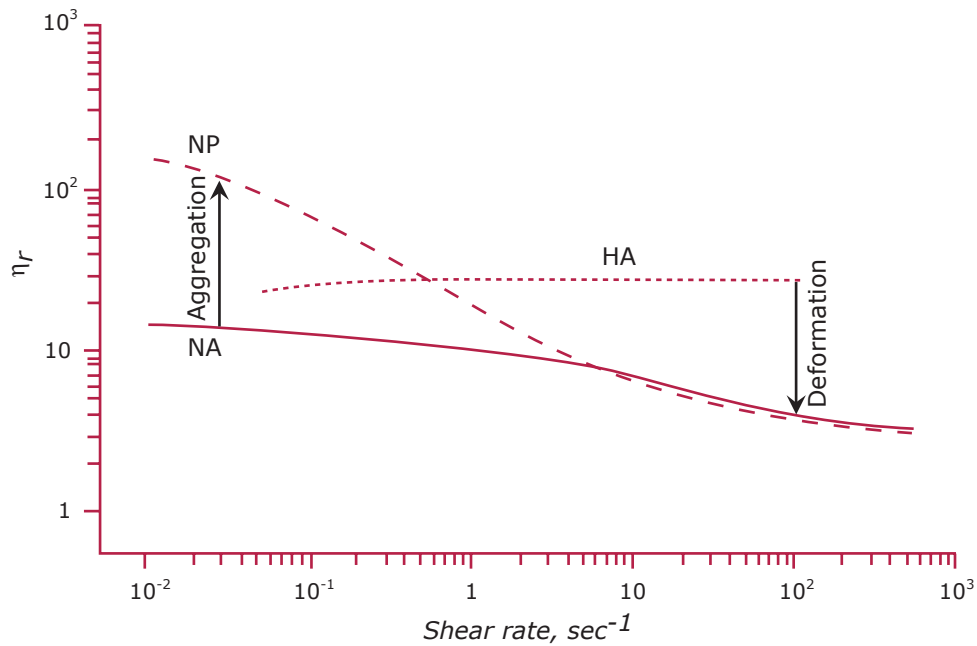


Figure by MIT OCW. After Fig. 3.4-2 in Fung, 1981.

Figure 6: Blood is not Newtonian. At low shear rates, aggregates of red cells form, increasing viscosity. At high shear rates, red cells elongate, decreasing viscosity further.

Table 1: Estimates of wall shear rate in various vessels in man $\dot{\gamma}_w = 8\bar{V}/D$

Vessel	Average velocity* (cm s ⁻¹)	Diameter† (cm)	$\dot{\gamma}_w$ (s ⁻¹)
Aorta	48	2.5	155
Artery	45	0.4	900
Arteriole	5	0.005	8000
Capillary	0.1	0.0008	1000
Venule	0.2	0.002	800
Vein	10	0.5	160
Vena cava	38	3.0	100

* Taken from Berne and Levy, 1967.

† Taken from Burton, 1965.

of suspensions of rigid spheres and discs, liquid droplets, and sickled RBCs. Note the much more dramatic increase in viscosity of suspensions of rigid particles as their volume fraction increases. At 50% volume fraction, the rigid sphere suspension cannot flow, while blood is fluid even to hematocrits above 90%. Note also the dramatic increase in viscosity of sickled cells at normal hematocrits! (What clinical implications follow?)

Figure 9 also shows the increase in relative viscosity of blood as a function of hematocrit. It makes the point that at any given hematocrit the apparent viscosity of blood is *less* when measured in the vascular system than when measured in capillary tube viscometers. Why should this be so?

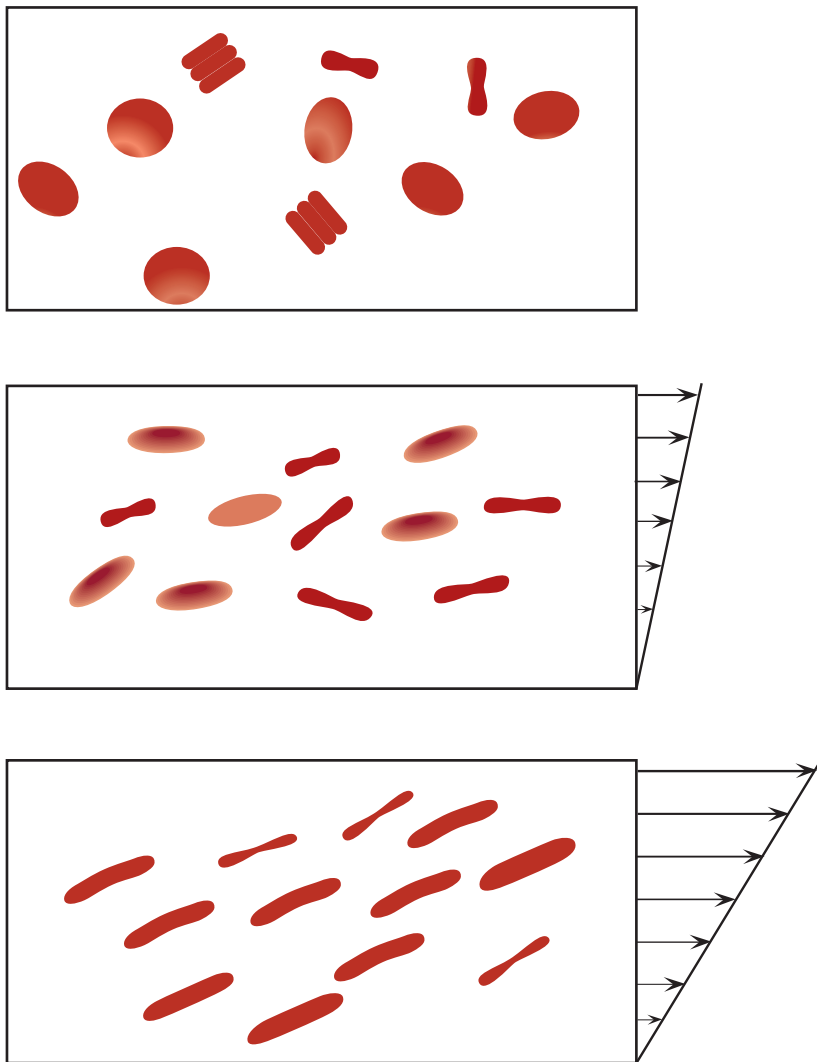


Figure by MIT OCW.

Figure 7: Non-Newtonian characteristics of blood.

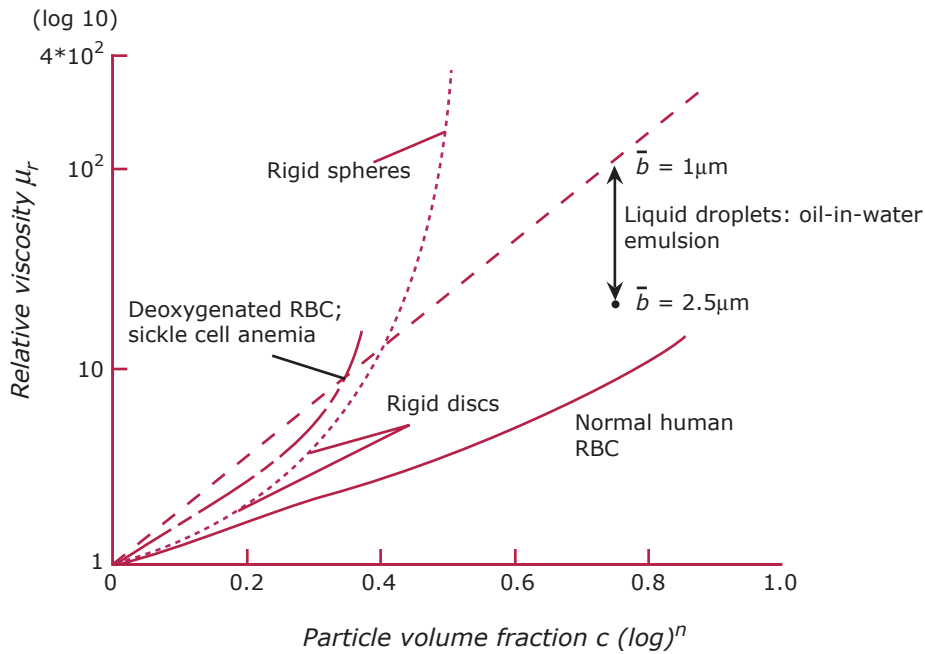


Figure by MIT OCW. After Figure 3.4-3 in Fung, 1981, based on Goldsmith, 1972.

Figure 8: Relative viscosity of human blood at 25°C as a function of red cell volume fraction, compared to that of suspensions of rigid latex spheres, rigid discs, droplets, and sickled erythrocytes, which are virtually nondeformable. From Goldsmith (1972b).

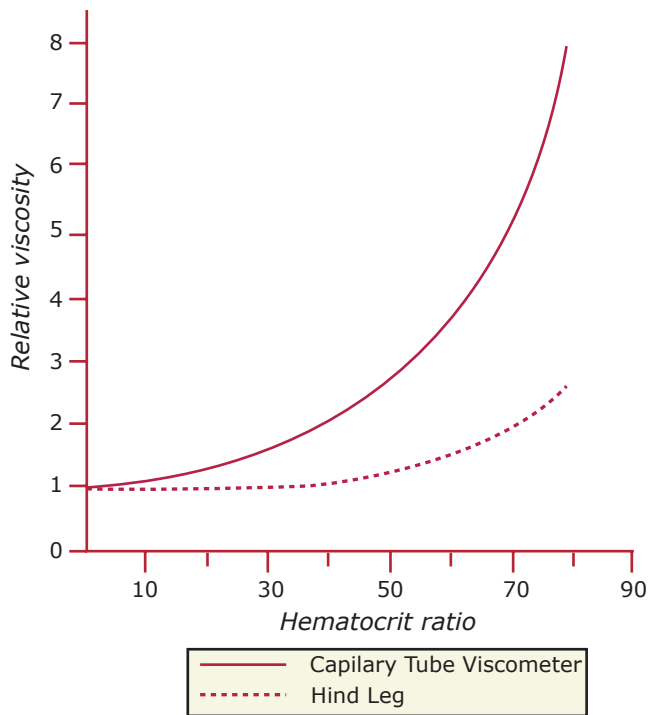


Figure by MIT OCW. After Levy and Share. *Circ Res.* 1: 247, 1953.

Figure 9: Viscosity of whole blood, relative to that of plasma, increases at a progressively greater rate as hematocrit ratio increases. for any given hematocrit ratio the apparent viscosity of blood is less when measured in a biological viscometer (such as the hind leg of a dog) than in a conventional capillary tube viscometer. (Redrawn from Levy, M. N., and Share, L.: *Circ. Res.* 1:247, 1953.)

In 1931 Fahraeus and Lindqvist measured the viscosity of blood, relative to that of water, in thin glass tubes at high shear rates. They observed a decrease in relative viscosity when tube diameter fell below 0.3 mm (Figure 10). The explanation of this effect is the fact that RBCs tend to migrate toward the center of the tube, leaving a cell-poor layer of plasma (of lower viscosity) near the walls.

It is also true that the effective hematocrit in the thin tube is *lower* than that of the reservoir feeding the tube. (The RBCs at the center of the tube move faster than the plasma at the walls; thus, to transport the same ratio of cells to plasma through the tube, the concentration of RBCs must be lower than at the feeding reservoir.) (See Figure 11.)

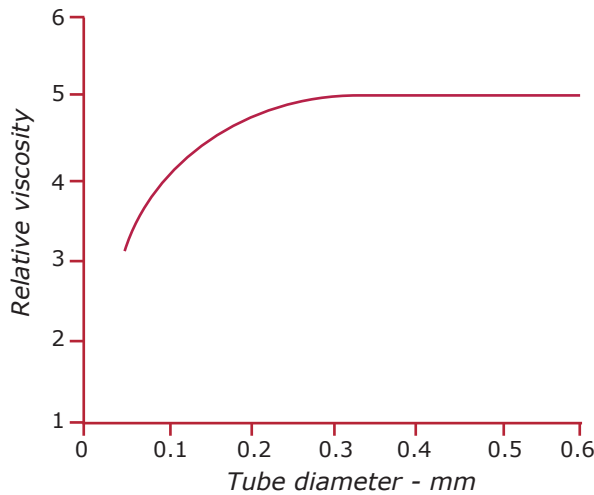


Figure by MIT OCW. After Fahraeus and Lindqvist. *Am J Physiol.* 96: 562, 1931.

Figure 10: Viscosity of blood, relative to that of water, increases as a function of tube diameter up to a diameter of about 0.3 mm. (Redrawn from Fåhræus, R., and Lindqvist, T.: *Am. J. Physiol.* 96:562, 1931.)

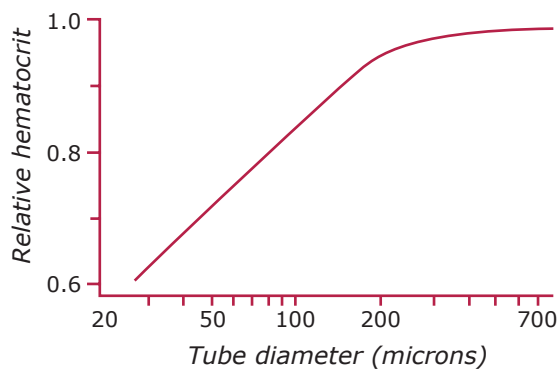


Figure by MIT OCW. After Barbee and Cokelet. *Microvasc. Res.* 3: 6, 1971.

Figure 11: The “relative hematocrit” of blood flowing from a feed reservoir through capillary tubes of various calibers, as a function of the tube diameter. The relative hematocrit is the ratio of the hematocrit of the blood in the tubes to that of the blood in the feed reservoir. (Redrawn from Barbee, J. H., and Cokelet, G. R.: *Microvasc. Res.* 3:6, 1971.)

It is the Fahraeus-Lindqvist effect which explains the lower apparent viscosity of blood in the microcirculation as compared to the viscometer (Figure 9).

From all of the above, we may conclude that to a good approximation blood may be considered as a Newtonian fluid provided the diameter of the vessel is greater than about 0.3 mm., and the shear rate exceeds 100 sec.⁻¹.

3. Conservation of Mass

In addition to the constitutive relation just discussed (Eq. 2.), two other concepts are commonly used in fluid dynamics; *conservation of mass* and *conservation of momentum*. We sometimes also need to invoke the equation for conservation of energy. In the context of flow through vessels of varying cross-sectional area, the principle of mass conservation can be simply visualized. Take, for example, the tube shown in Figure 12.

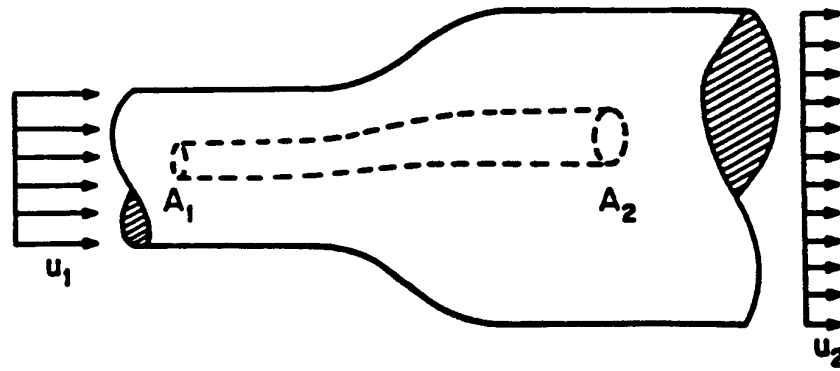


Figure 12: Conservation of Mass

A fluid region has been denoted by dashed lines having two ends which are perpendicular to the tube axis, and a curved lateral surface which we define as being parallel to the fluid velocity vector at every point on the surface. This is called a stream tube.¹ In steady flow the stream tube can be thought of as an imaginary vessel, completely contained by the actual vessel. No fluid passes into or out of the stream tube through the lateral surface. Furthermore, if we choose the stream tube to be very small, we can ascribe a single inlet velocity (u_1) and a single outlet velocity (u_2) to all the fluid particles passing through areas A_1 and A_2 , respectively.

Conservation of mass then dictates that the mass flow rate through area A_1 ($\rho u_1 A_1$) must equal the mass flow rate through area A_2 ($\rho u_2 A_2$) where ρ is the fluid density. In blood flow, the density can be assumed constant, giving

$$u_1 A_1 = u_2 A_2 \quad (1)$$

Considering the entire vessel as the stream tube, the same equation can be written, except that u_1 and u_2 must then be thought of as average velocities over the entire cross-sectional area.

¹Another useful concept is that of the “streamline”, which is an imaginary line that is everywhere tangent to the fluid velocity vector.

Having derived an equation for mass conservation for a simple, very specific case, consider now a more general formulation.

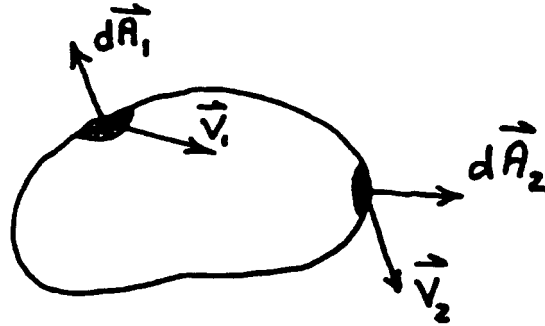


Figure 13: Mass Flux through an arbitrary control volume

Consider an arbitrary control volume in a fluid with a velocity field $\vec{v}(x, y, z, t)$ as shown in Figure 13. The mass contained within the control volume is

$$m = \int_{cv} \rho dV$$

where ρ is the density of the fluid and dV is a volume element. The net flow of matter through a differential surface area dA_i , into the control volume is:

$$\frac{dm_i}{dt} = -\rho \vec{v}_i \cdot d\vec{A}_i \quad (2)$$

where \vec{v}_i is the fluid velocity vector at point i , and $d\vec{A}_i$ is a vector differential area pointing in the inward direction. By integrating over the entire closed surface of the control volume we have:

$$\frac{dm}{dt} = -\oint_{cs} \rho \vec{v} \cdot d\vec{A} \quad (3)$$

Conservation of mass states that the time rate of change of the mass inside the control volume must equal the rate of mass transfer across the surface into the control volume. Hence,

$$\frac{\partial}{\partial t} \int_{cv} \rho dV = -\oint_{cs} \rho \vec{v} \cdot d\vec{A} \quad (4)$$

or

$$\frac{\partial}{\partial t} \int_{cv} \rho dV + \oint_{cs} \rho \vec{v} \cdot d\vec{A} = 0$$

If we deal with *incompressible fluids* (such as blood) ($\rho = \text{const.}$) then the left-hand side of eq. (4) is zero, and we have,

$$\oint_{cs} \vec{v} \cdot d\vec{A} = 0 \quad (5)$$

An alternative formulation, useful when the velocity field varies in both magnitude and direction, can be derived from equation 5 using Green's theorem, and states that for an incompressible fluid,

$$\vec{\nabla} \bullet \vec{v} = 0 \quad (6)$$

This is called the *continuity equation*.

The evaluation of the integral in equation 5 to the tube depicted in Figure 12 is quite simple if we assume that the velocities are constant across each section:

$$A_1 u_1 - A_2 u_2 = 0$$

or

$$A_1 u_1 = A_2 u_2 = Q \quad (7)$$

as in equation 1.

A more general statement for this situation is:

$$\int_A 2\pi r \vec{u}(r) \bullet d\vec{r} = \text{constant}$$

Equation 7 states that in a conduit of varying cross-sectional areas, the flow velocity must vary inversely with the area of the tube. This principle may be applied to the circulation as a whole in order to estimate blood velocity at various points. The table below documents the geometry of the mesenteric vascular bed of the dog. Note that the total cross-sectional area of the vascular bed increases almost three orders of magnitude from aorta to capillaries. As shown diagrammatically in Figure 14, the mean velocity in the vascular tree varies inversely with total area. Thus, if the mean velocity in the aorta is about 30 cm/sec., it would be in the order of 0.3 mm/sec. in the capillaries.

Table 2: Geometry of Mesenteric Vascular Bed of the Dog*

Kind of Vessel	Diameter (mm)	Number	Total Cross-sectional Area (cm ²)	Length (cm)	Total Volume (cm ³)
Aorta	10	1	0.8	40	30
Large arteries	3	40	3.0	20	60
Main artery branches	1	600	5.0	10	50
Terminal branches	0.6	1,800	5.0	1	25
Arterioles	0.02	40,000,000	125	0.2	25
Capillaries	0.008	1,200,000,000	600	0.1	60
Venules	0.03	80,000,000	570	0.2	110
Terminal veins	1	1,800	30	1	30
Main venous branches	2	600	27	10	270
Large veins	6	40	11	20	220
Vena cava	12	1	1.2	40	50
					930

* Data of F. Mall.

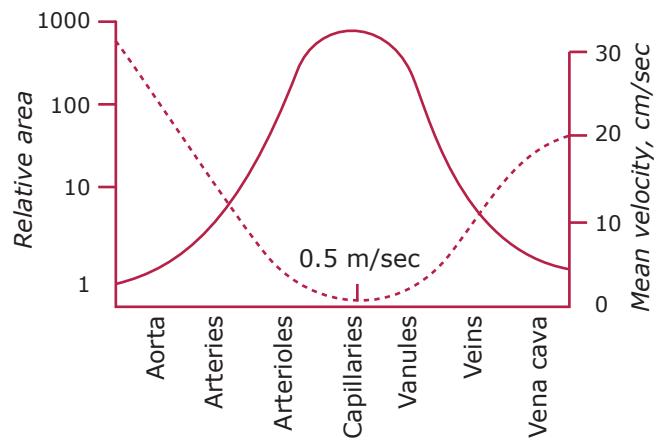


Figure by MIT OCW. After Burton.

Figure 14: Schematic graph showing: *broken line*, the changes in relative total cross-sectional area (on a logarithmic scale) of the vascular bed; *solid line*, the mean velocity in the different categories of vessel.

4. Conservation of Momentum

4.1 Differential Approach

The principle of conservation of momentum is no more than a statement of Newton's second law written for a particle of mass, m .

$$\vec{F} = m\vec{a} \quad (8)$$

where \vec{a} is the particle acceleration and \vec{F} is the *total* force acting on the particle. However, we will find it more convenient to re-write the right-hand side of equation 8 in terms of the rate of change of momentum, where the momentum of a particle is equal to its mass times its velocity. For fluids, we prefer to use the mass per unit volume or density, ρ , and also the momentum per unit volume, $\rho\vec{v}$. Hence, equation 8 can be re-written as

$$\vec{F} = \frac{d}{dt}(\rho\vec{v}) \quad (9)$$

where \vec{F} is the net force per unit volume and the right-hand side is the rate of change of momentum with time.

The components of the velocity, u , v , and w are each functions of both time and position. Hence,

$$\begin{aligned} \frac{du(x, y, z, t)}{dt} &= \frac{\partial u}{\partial t} + \frac{\partial u}{\partial x} \frac{\partial x}{\partial t} + \frac{\partial u}{\partial y} \frac{\partial y}{\partial t} + \frac{\partial u}{\partial z} \frac{\partial z}{\partial t} \\ &= \frac{\partial u}{\partial t} + u \frac{\partial u}{\partial x} + v \frac{\partial u}{\partial y} + w \frac{\partial u}{\partial z} \end{aligned} \quad (10)$$

with similar expressions for v and w . Equation 10 may be rewritten with the help of the vector notation $\vec{\nabla}$:

$$\frac{du}{dt} = \frac{\partial u}{\partial t} + (\vec{v} \cdot \vec{\nabla}) u \quad (11)$$

Note that the operator $(\vec{v} \cdot \vec{\nabla})(\)$ is a scalar, and is given in equation 12.

$$(\vec{v} \cdot \vec{\nabla})(\) = u \frac{\partial(\)}{\partial x} + v \frac{\partial(\)}{\partial y} + w \frac{\partial(\)}{\partial z} \quad (12)$$

This operator is applied to each velocity vector component, and the total rate of change of momentum may be written as:

$$\rho \frac{\partial \vec{v}}{\partial t} + \rho (\vec{v} \cdot \vec{\nabla}) \vec{v} = \sum (\text{forces/volume}) \quad (13)$$

The first term is called the *unsteady* or *temporal acceleration*, and vanishes if the flow is steady as viewed by a stationary observer. The second term is called the *convective acceleration*.

To complete the description of fluid motion, we must specify the forces which are acting on the fluid. For blood and other "simple" fluids, there are only three forces of interest.

- Gravitational force, which is simply ρg per unit volume acting in the vertical direction.
- Pressure forces.
- Viscous forces arising from friction between the flowing fluid element and its surroundings.

4.1.1 Pressure Forces

Since equation 13 is written in differential form, we are looking for the local forces on a small element of fluid. The local *net pressure force* per unit volume which the fluid element experiences is simply the local pressure gradient. Consider first the x -direction.

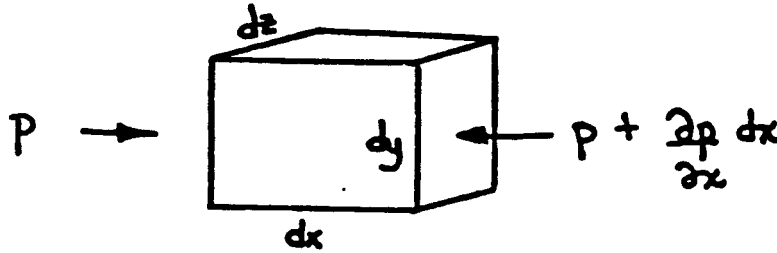


Figure 15: Pressure body force on a fluid element

The force per unit volume in the x -direction will be given by:

$$f_{x,p} = \frac{\left[p - \left(p + \frac{\partial p}{\partial x} dx \right) \right] dy dz}{dx dy dz} = -\frac{\partial p}{\partial x} \quad (14)$$

In more general terms:

$$\vec{f}_p = -\vec{\nabla} p \quad (\text{in three dimensions})$$

The *net viscous force* per unit volume acting on the same fluid particle in one-dimensional flow results from the difference in shear stress between the upper and lower surfaces.

The force per unit volume in the x -direction due to viscosity can then be written:

$$\begin{aligned} f_{x,v} &= \frac{\left(\tau_{yx} + \frac{\partial \tau_{yx}}{\partial y} \cdot dy - \tau_{yx} \right) dx dz}{dx dy dz} \\ &= \frac{\partial \tau_y}{\partial y} = \mu \frac{\partial^2 u(y)}{\partial y^2} \end{aligned} \quad (15)$$

Neglecting some subtleties in the derivation, the vector force per unit volume due to viscosity in a three-dimensional, incompressible flow can be written:

$$\vec{f}_v = \mu \nabla^2 \vec{v} \quad \text{where} \quad \nabla^2 \equiv \frac{\partial}{\partial x^2} + \frac{\partial}{\partial y^2} + \frac{\partial}{\partial z^2} \quad (16)$$

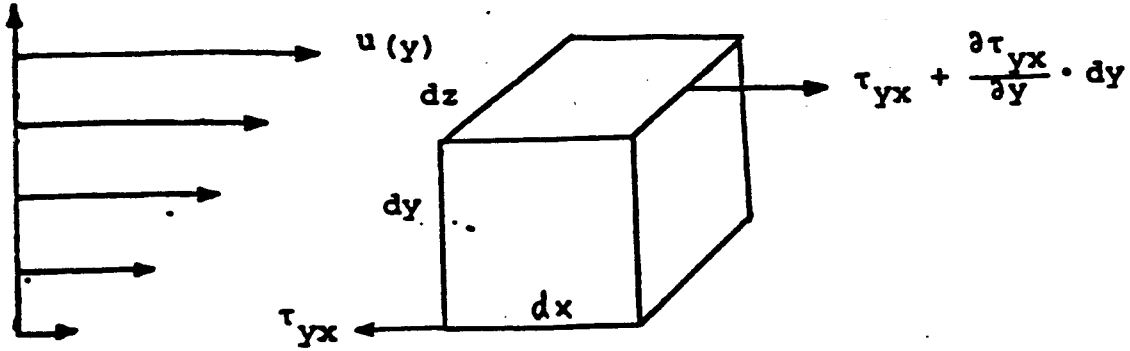


Figure 16: Viscous force on an element

Combining equations 9 - 16 we obtain the vector differential equation for conservation of momentum, also known as the *Navier-Stokes equation*:

$$\rho \frac{\partial \vec{v}}{\partial t} + \rho (\vec{v} \cdot \vec{\nabla}) \vec{v} = -\vec{\nabla} p + \mu \nabla^2 \vec{v} + \rho \vec{g} \quad (17)$$

We rarely use this equation in its complete form, however, since very few exact solutions have been found. Instead, we make various simplifications, appropriate for the problem at hand, to reduce this equation to a more tractable form. For example, in cases of steady one-dimensional flow in the x-y plane we can write (neglecting gravity):

$$-\frac{\partial p}{\partial x} + \mu \frac{\partial^2 u}{\partial y^2} = 0 \quad (18)$$

and

$$\frac{\partial p}{\partial y} = 0 \quad (19)$$

The velocity profile can therefore be obtained simply by integrating equation 18 over y and applying the appropriate boundary conditions.

4.1.2 Laminar Viscous Flow in Rigid Tubes

As a specific example, we will consider the problem of steady laminar viscous flow in rigid tubes. One solution, following directly from equation 17 written in cylindrical coordinates is given in Appendix A. A simpler solution based on a direct application of equation 8 is given here.

Consider a tube of radius a and length Δl , and a fluid of viscosity μ . (See Figure 17.) The pressure at $x = 0$ is p_a , and at $x = \Delta l$ the pressure is p_b . Consider the cylindrical shell control volume of radius r , thickness dr , and length Δl . Since the flow is *steady*, there is no acceleration, so the total force acting on the control volume must be zero. Two forces must be considered: the force due to the pressure gradient, and viscous forces.

The net force acting on the control volume due to the pressure gradient is:

$$f_p = (p_a - p_b)dA = (p_a - p_b)2\pi r dr$$

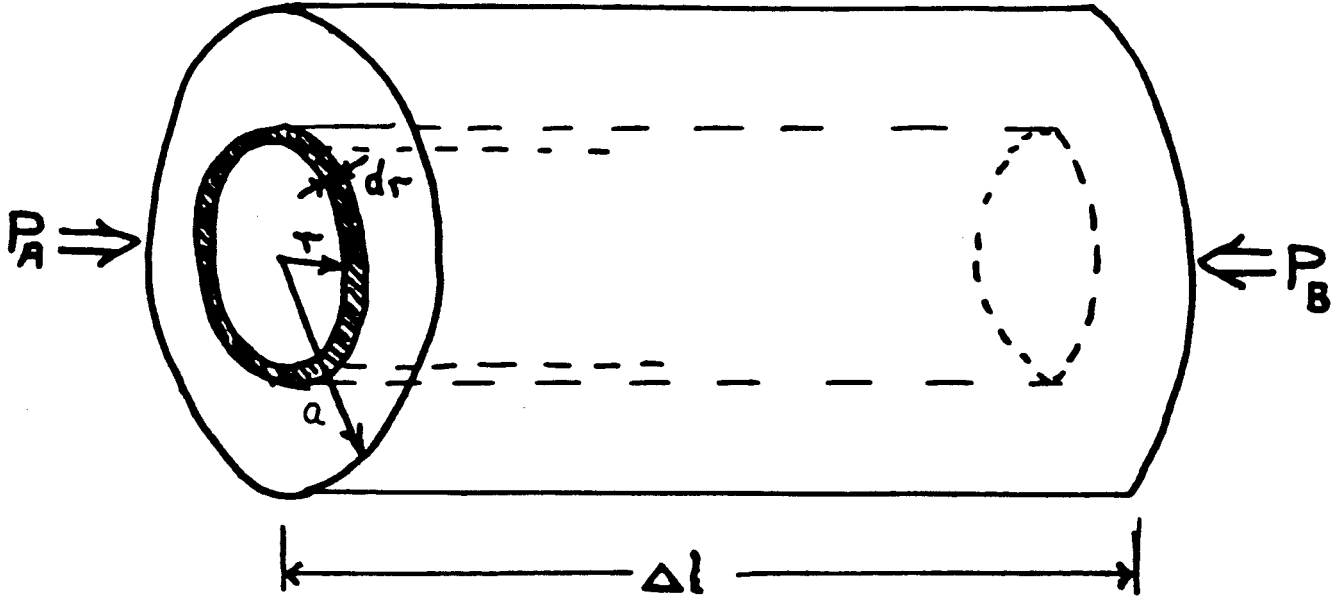


Figure 17: Steady flow through a straight cylinder, known as Poiseuille Flow

The net viscous force acting on the control volume would be given by:

$$\begin{aligned}
 f_v(r) &= f_v(r + dr) - f_v(r) \\
 &= \frac{\partial}{\partial r} [f_v(r)] dr \\
 &= \frac{\partial}{\partial r} [2\pi r \Delta l \tau(r)] dr
 \end{aligned}$$

Since the flow is steady:

$$\begin{aligned}
 f_p + f_v &= 0 \\
 (p_a - p_b)2\pi r dr + \frac{\partial}{\partial r} (2\pi r \Delta l \tau(r)) dr &= 0
 \end{aligned}$$

Integrating once, we have:

$$2\pi r \Delta l \tau(r) = -\pi(p_a - p_b)r^2 + k_1$$

Since $\tau(r) = \mu \frac{\partial u}{\partial r}$, we have

$$\frac{\partial u}{\partial r} = -\frac{r(p_a - p_b)}{2\mu \Delta l} + \frac{k_1}{2\pi \mu \Delta l r}$$

When $r = 0$, $\frac{\partial u}{\partial r}$ must be zero by symmetry, so that k_1 must be zero. Rearranging, we have:

$$\frac{\partial u}{\partial r} = \frac{-(p_a - p_b)}{2\mu\Delta l}r$$

Integrating again,

$$u(r) = \frac{-(p_a - p_b)}{4\mu\Delta l}r^2 + k_2$$

The second boundary condition is the no-slip condition, namely, $u(a) = 0$. Hence, we have

$$u(r) = \frac{1}{4\mu} \frac{(p_a - p_b)}{\Delta l} a^2 \left[1 - \left(\frac{r}{a} \right)^2 \right] \quad (20)$$

This is the equation of a parabola, and the velocity distribution is shown below:

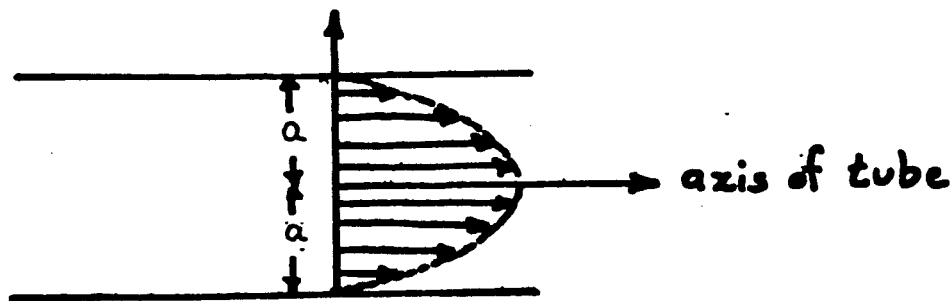


Figure 18: Parabolic Flow Profile of Poiseuille Flow (also called Hagen-Poiseuille Flow)

We may now calculate the total flow, Q , through the tube.

$$\begin{aligned} Q &= \int_0^a u(r) \cdot 2\pi r dr \\ &= \frac{\pi(p_a - p_b)a^2}{2\mu\Delta l} \int_0^a r \left(1 - \frac{r^2}{a^2} \right) dr \\ &= \frac{\pi}{8} \cdot \frac{1}{\mu} \cdot \frac{a^4}{\Delta l} (p_a - p_b) \end{aligned} \quad (21)$$

Equation 21 is the well-known **Poiseuille's law** which relates flow to pressure drop for rigid tubes. Note the division of the terms into a constant, a viscosity term, a geometric term, and a pressure term. Note particularly the strong dependence of flow on tube radius, a . Equation 21 may be written in the form

$$(p_a - p_b) = QR \quad \text{where} \quad R = \frac{8}{\pi} \cdot \mu \cdot \frac{\Delta l}{a^4}$$

Here R is the “resistance” of the tube and is directly proportional to length and viscosity, and inversely proportional to the fourth power of the radius. Thus, one would expect that the major

contribution to vascular resistance would be made by the small vessels. This is born out by consideration of the following table. Note also that the strong dependence of resistance upon vessel radius implies sensitive regulation of flow is possible by changing vessel diameter through action of vascular smooth muscle.

Table 3: Relative Resistance to Flow in the Vascular Bed: Calculated from Table 2 and Poiseuille's Law ($R \propto \frac{1}{r^4}$)

Aorta	4%	Venules	4%
Large arteries	5%	Terminal veins	0.3%
Main arterial branches	10%	Main venous branches	0.7%
Terminal branches	6%	Large veins	0.5%
Arterioles	41%	Vena cava	1.5%
Capillaries	27%		
Total arterial + capillary:		Total venous:	
93%		7%	

Another common simplification of the Navier-Stokes equation involves the neglect of all terms involving viscosity. This leads to:

$$\rho \frac{\partial \vec{v}}{\partial t} + \rho (\vec{v} \cdot \vec{\nabla}) \vec{v} = -\vec{\nabla} p + \rho \vec{g} \quad (22)$$

commonly referred to as *Euler's equation*. When using Euler's equation we make the assumption that the flow can be treated locally as *inviscid*. In this context the inviscid assumption simply means that the pressure gradient is due primarily to the effects of *inertia* rather than viscous stresses.

Euler's equation may be integrated along a streamline to yield

$$p + \rho gh + \frac{1}{2} \rho v^2 = \text{constant along a streamline} \quad (23)$$

where ρ is the density of the fluid in grams/cc, v the velocity in cm/sec, p the pressure in dynes/cm², g the acceleration of gravity (980 cm/sec²), and h the height of the fluid above some arbitrary reference in cm. Instead of gram-centimeter-second units, we may use millimeters of mercury for all terms (1 mmHg = 1,330 dynes/cm²). In the above expression $\frac{1}{2} \rho v^2$ is the **kinetic energy** per unit volume along the stream line, while p and ρgh appear as the **potential energy** per unit volume due to pressure and the earth's gravitational field respectively. It should be noted that the relation expressed in equation 23 assumes no energy losses due to friction—it does not apply to viscous flows. It is known as *Bernoulli's equation*, and is an expression of *conservation of energy*. Its application in situations where dissipation of energy is negligible is extremely helpful. However, you should note that the absence of viscosity and thermodynamic terms indicates that this is not a complete energy balance equation. Several illustrative examples follow:

1. Vascular Constrictions:

Consider the simple example shown in Figure 18. A tube of varying cross-sectional area carries fluid in a horizontal direction. We wish to determine the relation between p_1 and p_2 .

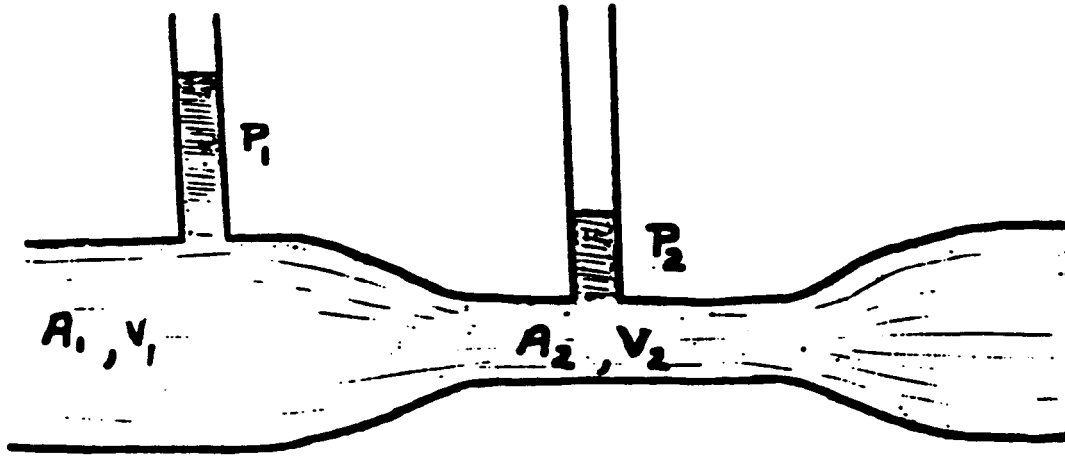


Figure 19: Manometry: flow through a constriction

Since the tube is horizontal, we may neglect the gravitational term in equation 23. The equation of continuity permits us to relate the cross-sectional areas and velocities:

$$A_2 v_2 = A_1 v_1$$

$$v_2 = \frac{A_1}{A_2} v_1$$

Bernoulli's principle states:

$$\frac{1}{2} \rho v_1^2 + p_1 = \frac{1}{2} \rho v_2^2 + p_2$$

$$p_2 v_1^2 = p_1 - \frac{1}{2} \rho (v_2^2 - v_1^2)$$

$$p_2 = p_1 - \frac{1}{2} \rho v_1^2 \left[\left(\frac{A_1}{A_2} \right)^2 - 1 \right]$$

Thus, in the narrowed portions of the tube, the pressure drops as the velocity increases. One interesting physiologic application of this finding is discussed by Burton (Chap. 10) in connection with the arterial narrowing due to atherosclerotic plaques. Consider the vessel shown in Figure 20.

Assume the vessel's normal area to be A_1 , with a mean pressure of 100 mmHg and a velocity of 30 cm/sec ($\rho \approx 1$). If the vessel is narrowed by the plaque to an effective area only one-ninth as large as normal, what will the transmural pressure be at the point of narrowing? Consider also the possible disastrous results of capillary ingrowth or cracks into plaques as illustrated in Figure 21. How might such geometry lead to rupture of the capillaries into the plaque?

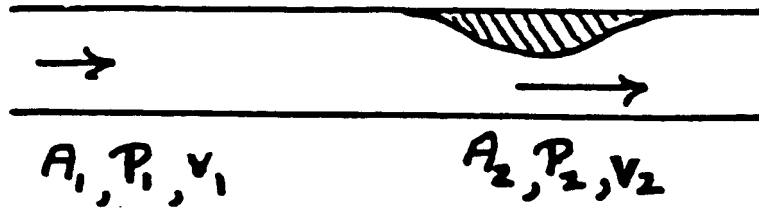


Figure 20: Flow past an atherosclerotic blockage.

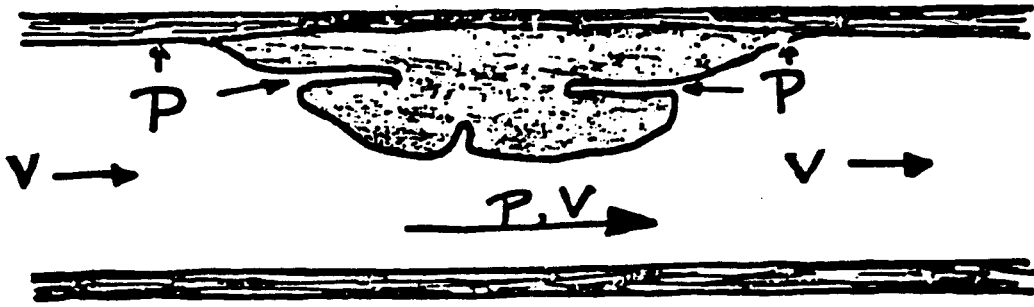


Figure 21: Pressure differential across an atherosclerotic plaque.

2. Pressure Measurement

If an obstacle is placed in a liquid flowing with velocity, v_0 , the liquid must come to rest just before the obstacle, and the streaming is divided into two branches one on each side of the obstacle. The original parallel streamlines are deformed as shown in Figure 22.

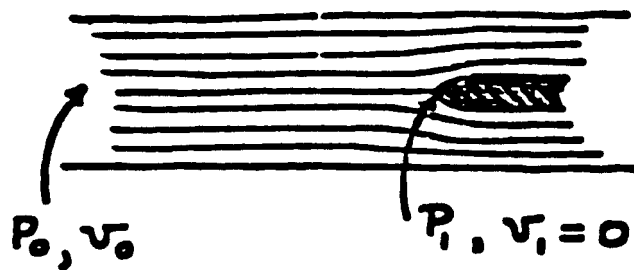


Figure 22: Stagnation point of a catheter in flow.

There is a stagnation point at the tip of the obstacle ($v_1 = 0$) and by the Bernoulli theorem,

$$p_1 = p_0 + \frac{1}{2}\rho v_0^2$$

where p_0 and v_0 are measured well upstream from the obstacle.

Question: Extend this reasoning to the problem of measuring pressures in the arterial system with catheters. Compare the pressure readings which would be obtained from a catheter with the opening facing “upstream” (end pressure) versus one with laterally oriented openings

(side pressure). Consider Table 4 below, which shows the amount and relative importance of kinetic energy at different cardiac outputs. Note the rather significant effect in the pulmonary artery. (Here, however, catheter opening usually points “downstream”, and would come very close to measuring true pressure.)

Table 4: Amount and Relative Importance of Kinetic Energy in Different Parts of the Circulation*

Vessel	Resting Cardiac Output				Cardiac Output Increased 3 Times		
	Velocity (cm/sec)	Kinetic Energy (mmHg)	Pressure (mmHg)	Kinetic Energy as % of Total	Kinetic Energy (mmHg)	Pressure (mmHg)	Kinetic Energy as % of Total
Aorta, systolic	100	4	120	3%	36	180	17%
Mean	30	0.4	100	0.4%	3.8	140	2.6%
Arteries, systolic	30	0.35	110	0.3%	3.8	120	3%
Mean	10	0.04	95	Neg.		100	Neg.
Capillaries	0.1	0.000004	25	Neg.	Neg.	25	Neg.
Venæ cavæ and atria	30	0.35	2	12%	3.2	3	52%
Pulmonary artery, systolic	90	3	20	13%	27	25	52%
Mean	25	0.23	12	2%	2.1	14	13%

* The cases where kinetic energy should not be neglected—that is, where it is more than 5% of the total fluid energy—are indicated by italic figures. When an artery is narrowed by disease processes, the kinetic energy becomes very important. Note: Neg. = Negligible.

3. Calculation of Valve Areas

The Bernoulli principle provides an approach for estimating the size of the valve areas using data obtained via cardiac catheterization. In Figure 23 the chamber represents the ventricle during ejection.

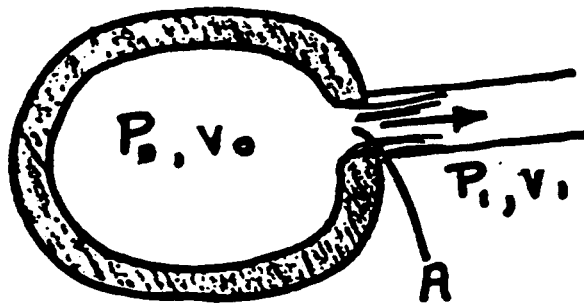


Figure 23: Blood ejected during systolic out of the left ventricle through the aortic valve (A)

Fluid is ejected through the orifice, and the cross-section area of the jet is A . The pressure inside the chamber is p_o , and the velocity is v_o . The velocity and pressure of the fluid in the jet are v_1 , p_1 . Bernoulli’s equation states:

$$p_0 + \frac{1}{2}\rho v_0^2 = p_1 + \frac{1}{2}\rho v_1^2$$

$$\frac{1}{2}\rho (v_1^2 - v_0^2) = p_0 - p_1$$

If we assume that $v_0 \ll v_1$, this equation becomes:

$$\frac{1}{2}\rho v_0^2 = p_0 - p_1$$

$$v_1 = \sqrt{\frac{2}{\rho}(p_0 - p_1)}$$

If the flow rate, Q , is known, we have

$$Q = Av_1$$

$$A = \frac{Q}{v_1}$$

Substituting from above, we have

$$A = \frac{Q}{k\sqrt{p_0 - p_1}} \quad (24)$$

In our derivation $k = \sqrt{2/\rho}$. The form of equation 4.1.2 has been verified experimentally for diseased human heart valves, but the value of the constant, k , differs depending on the valve involved. Q is measured in cc/sec; $p_0 - p_1$ is the pressure gradient across the valve in mmHg; k is 44.5 for the aortic valve and 37 for the mitral valve; and A is the valve area in cm^2 .

Note: Q is not cardiac output, but the instantaneous flow rate through the valve at the time the pressures are being measured.

5. Reynolds Number and Turbulent Flow

A dimensionless parameter called the **Reynolds number** is used as a measure of the relative importance of inertial effects (related to fluid momentum) and viscous effects. From equation 23 it can be seen that changes in pressure due to changes in fluid velocity scale with ρv^2 . Viscous stresses in a two-dimensional flow, as noted earlier, are equal to the coefficient of viscosity, times the shear rate, $\mu du/dy$, which can be roughly approximated for flow through a tube by $\mu v/d$. We can form a ratio, which we call the Reynolds number, between inertia-induced stress and viscous shear stress of the form

$$Re_d \equiv \frac{\rho v^2}{\mu v/d} = \frac{\rho v d}{\mu} = \frac{v d}{\nu} \quad (25)$$

where re_d is the Reynolds number using tube diameter, d , as the characteristic length, and where ν is the **kinematic viscosity** ($\nu \equiv \mu/\rho$).

High Reynolds number flow ($Re_d \gg 1$) is dominated by inertial effects, and viscous effects may be neglected as a first approximation. Bernoulli's equation (eq. 23), for example, would be valid when the Reynolds number is high. When the Reynolds number is low ($\ll 1$) viscous effects dominate.

In steady laminar flow, fluid particles move along well-defined trajectories or streamlines and no mixing occurs between adjacent streamlines. A different kind of flow results, however, when the Reynolds number is sufficiently large; a flow characterized by apparently random velocity fluctuations superimposed (see Figure 24).

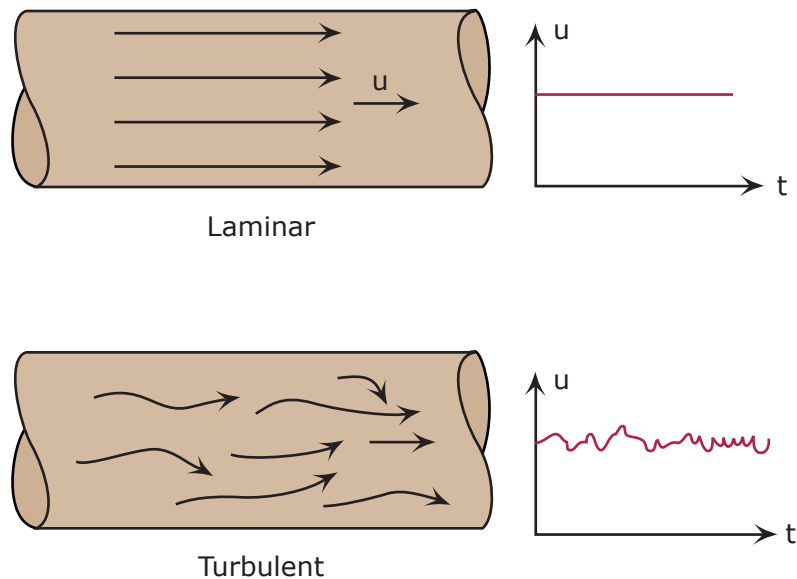


Figure by MIT OCW.

Figure 24: Reynolds number predicts development of turbulence in a given geometry.

In contrast to laminar flow, turbulence causes each fluid particle to move down the vessel in an apparently random fashion in which kinetic energy associated with the axial flow direction is continuously converted, first into a swirling or eddy motion, and finally into heat by viscous dissipation.

Turbulent flow results from an instability of laminar flow and occurs at a value of Reynolds number which depends on the local geometry. For example, flow in a tube of uniform circular cross-section typically becomes turbulent when $Re_d \gtrsim 2300$. In contrast, flow passing from a narrow orifice into a larger tube or reservoir can become turbulent at Reynolds numbers as low as 10. Judging from the range of Re_d given in Table 5 below (page 37), turbulence in fully-developed flow is not likely to occur in the venous circulation, but flow through venous valves or past vessel constrictions could become turbulent.

Without going into a detailed description of turbulent dissipation and the pressure drops associated with turbulence, one general statement, derived primarily from experimental observations, will be useful. Due to the internal dissipation of fluid energy associated with turbulence, an additional loss or pressure drop must be included in the mechanical energy equation. For a wide range of geometries, it can be shown that pressure changes due to turbulence vary as the square of the mean velocity.

Turbulent blood flow creates mechanical vibrations in the walls of vessels. The vibrations are often audible through the stethoscope (bruits or murmurs), or palpable (thrills). While turbulent flow is common in cases of narrowed heart valves or partial obstructions to arterial flow, it may also occur in normal individuals when sufficiently high Reynolds numbers are encountered. This is particularly likely during situations of high cardiac output such as exercise, fever, anemia, etc. Murmurs heard in such cases are called “flow murmurs” or “functional murmurs”, and are benign.

6. Entrance Effects

In our discussion so far we have assumed steady viscous flow, and have derived the equation for Poiseuille flow. This type of flow is sometimes called “fully developed” viscous flow in a cylindrical conduit in which the viscous drag from the walls penetrates throughout the fluid leading to a parabolic velocity profile. Clearly blood flow at the proximal aorta is not fully developed laminar flow, since it enters the aorta as a bolus with a uniform velocity distribution over the cross-section. As the blood flows down the aorta, the viscous drag from the walls has an increasing effect on the flow. In particular, velocities near the wall tend to decrease, and the penetration of the drag into the fluid increases as the fluid continues down the conduit. Eventually the “boundary layer” penetrates to the center of the conduit and steady laminar flow is established. (See Figure 24.) The distance from the entrance to the point where fully developed flow occurs is termed the **entrance length**, L_E . The ratio L_e/D , where D is the diameter of the conduit, is related to the velocity of the fluid. One might expect L_e/D to be directly proportional to v and inversely proportional to μ . In fact, a theoretical entrance length which agrees reasonably well with experiment for non-turbulent flow is

$$\frac{L_e}{D} = 0.065Re_d \quad , \text{ where } \quad Re_d = \frac{vD\rho}{\mu} \quad (\text{Reynolds number}). \quad (26)$$

For the aorta $v \approx 25$ cm/sec, $D = 2.5$ cm, $\mu = 4 \times 10^{-2}$, and $Re_d \equiv 1560$. Hence,

$$\frac{L_e}{D} = 100 \quad , \text{ or } \quad L_E = 250\text{cm}$$

This result implies that the velocity distribution in the entire aorta will tend to be uniform over the cross-section. Experimental results for dogs is shown in Figure 25 which show rather uniform velocity distributions almost all the way to the iliac bifurcation. Note that the above analysis assumes steady flow, and we have used the mean aortic flow in estimating the entrance length. The important point is that even if we neglect the pulsatile component of the flow, viscous effects are not important in the large vessels such as the aorta. They become important, however, in smaller vessels.

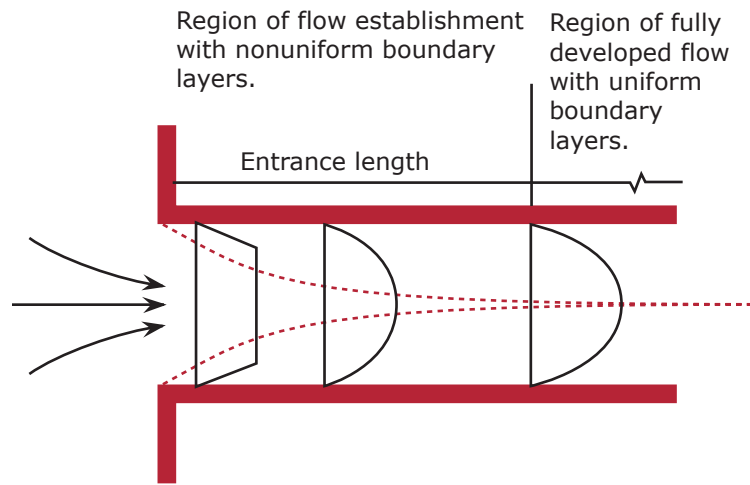


Figure by MIT OCW.

Figure 25: Entrance effects.

Image removed for copyright reasons.

Figure 26: Velocity distribution in the aorta of dogs.

7. Oscillating Viscous Flow

Flow in the aorta and large arteries is clearly not steady, but rather is pulsatile. The question is—what is the nature of the flow? How much influence upon velocity profiles is played by shear stresses at the vessel walls? Is it possible to estimate where in the arterial system we might expect Poiseuille flow, and where to expect bolus or slug flow?

As a first approach to this problem, consider the problem of a one-dimensional oscillating plate immersed in an infinite fluid. (See Figure 27.)

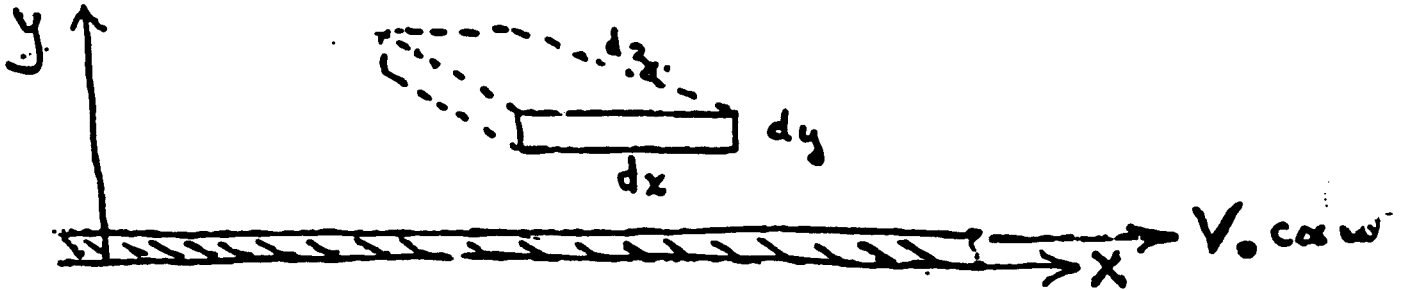


Figure 27: Oscillating plate in an infinite fluid reservoir.

The plate, assumed to be of infinite length in the x -direction, and perpendicular to the xy plane is oscillated back and forth with velocity $v_0 \cos \omega t$.

Intuition permits us to predict the behavior of the fluid motion in some limiting cases. If $\omega = 0$, and the plate moved at constant velocity v_0 , eventually all the fluid would move with the plate at a velocity, v_0 . On the other hand, it is apparent that as $\omega \rightarrow \infty$, only an infinitesimal layer of fluid which clings to the plate at $y = 0$ would tend to move with the plate, and that fluid away from the plate would tend to remain at rest. For intermediate values of ω one would expect the viscous drag of the plate to “penetrate” the liquid to various distances. It would also appear reasonable to expect the depth of penetration of the viscous effects to be greater as the fluid viscosity increased. We are interested in getting a rough estimate of this “boundary layer thickness”, and in applying the result to the vascular system.

The problem is solved as follows:

Consider the control volume $dx dy dz$ at a distance y cm from the oscillating plate.

The equations of motion need consider only the “mass \times acceleration” term and the viscous shear forces since the pressure gradient $\partial p / \partial x = 0$.

$$\begin{aligned} \text{Mass} &= \rho \, dx dy dz \\ \text{Acceleration} &= \frac{\partial u(y, t)}{\partial t} \\ \text{shear force} &= \frac{\partial}{\partial y} [\tau(y) \, dx dz] \, dy = \mu \frac{\partial^2 u}{\partial y^2} dx dy dz \end{aligned}$$

The equation of motion becomes:

$$\frac{\partial u(y, t)}{\partial t} - \nu \frac{\partial^2 u(y, t)}{\partial y^2} = 0 \quad \text{where} \quad \nu \equiv \frac{\mu}{\rho} \quad (27)$$

The boundary conditions are:

$$\begin{aligned} u(y = 0) &= v_0 \cos \omega t \\ u(y = \infty) &= 0 \end{aligned}$$

The solution to the differential equation is of the form:

$$u(y, t) = A e^{-y/\delta} \cos\left(\omega t - \frac{y}{\delta}\right) \quad (28)$$

Substituting back into the original equation, and recalling the boundary conditions we have

$$A = u_0 \quad \delta = \sqrt{\frac{2\nu}{\omega}} \quad (29)$$

When $y = \delta$, the amplitude of the fluid oscillation drops to $\frac{1}{e}$ of its maximum, and hence δ is a measure of the boundary layer thickness.

An estimate of δ may be made for the cardiovascular system. Assume a heart rate of 100 beats/min. and a kinematic viscosity of blood of $\nu = 5 \times 10^{-2}$. For H.R. = 100 beats/min. $\omega = 2\pi f \approx 10$. Substituting, we obtain

$$\delta = 0.1 \text{ cm} = 1 \text{ mm}$$

This implies that for large ($\gg 1$ mm) vessels such as the aorta the boundary layer extends only a very short distance into the fluid. Thus, in the aorta the fluid tends to behave as plug flow, with relatively uniform velocity distribution.

Using similar methods one may solve the problem of oscillating flow in a rigid tube. The mathematics is quite complex and not worth discussing here, but it does permit one to estimate boundary layer effects in tubes. The resultant expression for boundary layer thickness is identical to that of equation 29 above. It is emphasized again that fully developed laminar (Poiseuille) flow is to be expected only in vessels in the order of several mm. and less in diameter.

8. Pulse wave propagation in arteries

In this section, we wish to develop a simple model which illustrates how pulse propagation may occur in the arterial system. We will represent the artery as a thin-walled elastic tube characterized by an equation of state relating the area of the vessel, A , to the transmural pressure, p ,

$$A = A(p)$$

For simplicity we will assume a linear operating region and define a compliance per unit length C_u such that

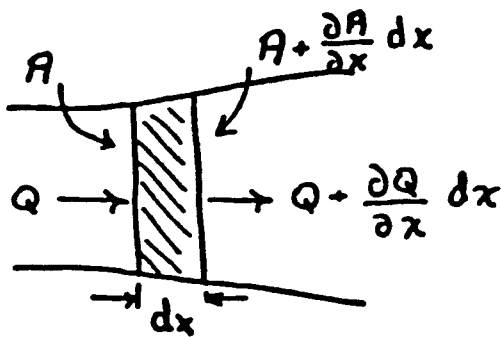
$$C_u = \frac{\partial A}{\partial p} = \text{constant}$$

This implies

$$A = A_0 + C_u p \quad (30)$$

Consider a section of vessel as shown in Figure 28.

Figure 27



$Q = \text{volume flow rate}$
 $A = \text{cross sectional area}$

Figure 28:

The equation of continuity states that the net rate of increase in mass of the control volume, $\rho \frac{\partial A}{\partial t} dx$ must equal the net inflow into the control volume which is $-\rho \frac{\partial Q}{\partial x} dx$.

Hence,

$$\frac{\partial Q}{\partial x} + \frac{\partial A}{\partial t} = 0 \quad (31)$$

The equation of motion equates the time rate of change of momentum to the pressure gradient.

$$\frac{\partial}{\partial t}(u\rho A dx) = -\frac{\partial}{\partial x}(p \cdot A) dx$$

Differentiating and suitably factoring we obtain:

$$\rho u \left(\frac{1}{u} \frac{\partial u}{\partial t} + \frac{1}{A} \frac{\partial A}{\partial t} \right) = -\rho \left(\frac{1}{A} \frac{\partial A}{\partial x} + \frac{1}{p} \frac{\partial p}{\partial x} \right)$$

$$\rho u \left(\frac{\partial}{\partial t} \ln u + \frac{\partial}{\partial t} \ln A \right) = -\rho \left(\frac{\partial}{\partial x} \ln A + \frac{\partial}{\partial x} \ln p \right)$$

small

small

We will assume a small-signal case; that is, the variation in A with respect to time and space is small compared to similar variations in velocity u and pressure, p . Hence, we may neglect the terms containing $\ln A$, and we have:

$$\frac{\partial u}{\partial t} + \frac{1}{\rho} \frac{\partial p}{\partial x} = 0 \quad (32)$$

Multiplying by A we obtain (noting that $Q = Au$),

$$\frac{\partial Q}{\partial t} + \frac{A}{\rho} \frac{\partial p}{\partial x} = 0 \quad (33)$$

Using equation 30, differentiating with respect to t , and substituting into equation 31, we obtain

$$\frac{\partial Q}{\partial x} + C_u \frac{\partial p}{\partial t} = 0 \quad (34)$$

Equations 33 and 34 are similar to the equations governing lossless transmission lines, and ρ/A may be identified as L_u , the **inertance** per unit length.

If the compliance of the vessel is assumed to be independent of pressure and location, and if the area of the vessel is assumed to be constant (no tapering and small perturbations with pressure), we may treat C_u and L_u as constants and solve equations 33 and 34.

Differentiating 33 with respect to x , and 34 with respect to t and eliminating the Q terms, we have:

$$C_u \frac{\partial^2 p}{\partial t^2} - \frac{A}{\rho} \frac{\partial^2 p}{\partial x^2} = 0$$

or

$$\frac{\partial^2 p}{\partial t^2} - \frac{A}{\rho C_u} \frac{\partial^2 p}{\partial x^2} = 0 \quad (35)$$

Solutions are of the form

$$p = p(x - ct)$$

Thus pressure waves propagate at constant velocity, c , without distortion, where

$$c = \sqrt{\frac{A}{\rho C_u}} = \sqrt{\frac{1}{L_u C_u}} \quad (36)$$

This velocity is known as the *Moens-Korteweg wave speed*. The Moens-Korteweg relationship indicates a constant wave velocity in the arterial system. In man, pulse wave velocity varies greatly—for instance it is about 3 m/sec. at the proximal aorta, and 8 or 9 m/sec. at the iliac bifurcation. Further, it is also true that the pulse wave distorts as it moves down the aorta. (See Figure 29.)

Factors of importance which influence pulse propagation in the circulatory tree include: 1) vessel stiffness increases with pressure such that the peaks of pressure move faster than the low-pressure points; 2) the arterial tree is tapered in area and wall properties; and 3) reflections may distort pressure waveforms.

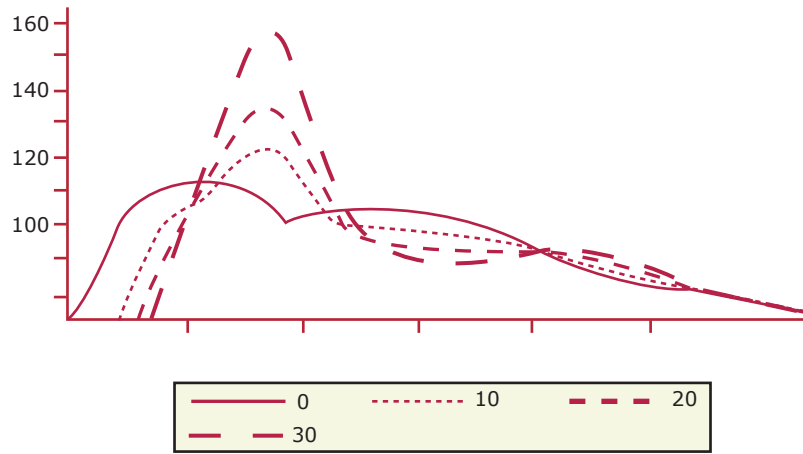
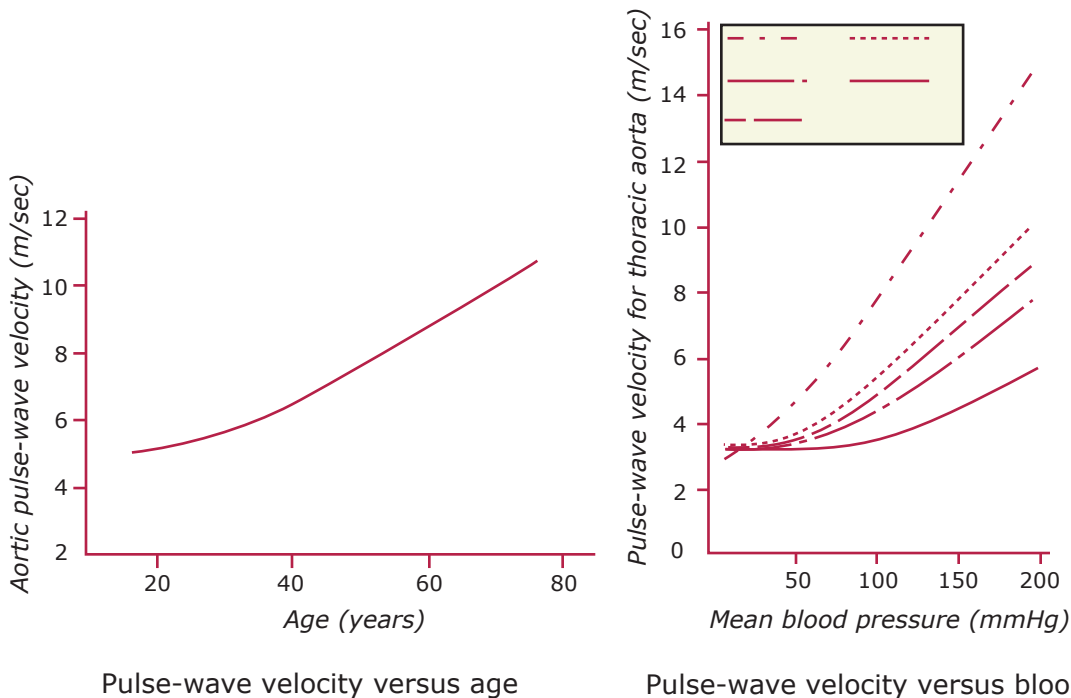


Figure by MIT OCW.

Figure 29: Contour of arterial pressure pulse in various parts of aorta. Curve 0, pulse near root of aorta; curves 10, 20, and 30, pulse at 10, 20, and 30 cm farther down aorta.

In general, vessels become stiffer with age as elastic tissue becomes replaced with collagen, and as arteriosclerosis becomes more common. As one would expect, pulse wave velocity in the aorta increases with age. Also, since vessels expand and become stiffer as pressure increases, pulse wave velocity increases with mean blood pressure (Figure 30).



Pulse-wave velocity versus age

Pulse-wave velocity versus blood pressure

Figure by MIT OCW. After King, A. L., pp. 190 in *Medical Physics*. Vol II. Chicago: Year Book Medical Publishers, 1950.

Figure 30: Changes in pulse wave velocity with age and mean blood pressure.

9. Reflection of Waves

Characteristic impedance, z_0 , is defined by analogy to transmission line theory.

$$z_0 = \frac{p}{Q} \quad (37)$$

From equation 34 above we have

$$\frac{\partial Q}{\partial x} + C_u \frac{\partial p}{\partial t} = 0$$

Using (37) we get

$$\begin{aligned} \frac{\partial}{\partial x} \left(\frac{p}{z_0} \right) + C_u \frac{\partial p}{\partial t} &= 0 \\ \frac{1}{z_0} \frac{\partial p}{\partial x} + C_u \frac{\partial p}{\partial t} &= 0 \end{aligned} \quad (38)$$

From equation 33 we have

$$\begin{aligned} \frac{\partial Q}{\partial t} + \frac{A}{\rho} \frac{\partial p}{\partial x} &= 0 \\ \frac{1}{z_0} \frac{\partial p}{\partial t} + \frac{A}{\rho} \frac{\partial p}{\partial x} &= 0 \end{aligned}$$

Substituting from (38) we have

$$\left(\frac{1}{z_0} - \frac{Az_0 C_u}{\rho} \right) \frac{\partial p}{\partial t} = 0 \quad (39)$$

Since $\frac{\partial p}{\partial t} \neq 0$, the terms in the parentheses must be zero.

$$\begin{aligned} \frac{1}{z_0} - \frac{Az_0 C_u}{\rho} &= 0 \\ z_0^2 &= \frac{\rho}{AC_u} = \frac{L_u}{C_u} \quad \text{where } L_u = \frac{\rho}{A} \\ z_0 &= \sqrt{\frac{L_u}{C_u}} \end{aligned} \quad (40)$$

The reflection coefficient is the ratio of the reflected to the incident wave.

$$\Gamma = \frac{z_L - z_0}{z_L + z_0} \quad (41)$$

Here z_L is the terminating impedance, and z_0 the characteristic impedance.

10. Fluid Mechanics of Heart Valve Action

10.1 Aortic Valve

The aortic valve consists of three thin (0.1 mm) flexible, self-supporting cusps. Corresponding to each cusp, there is a bulge in the aortic wall called a sinus (sinuses of Valsalva). The coronary arteries arise from two of these sinuses.

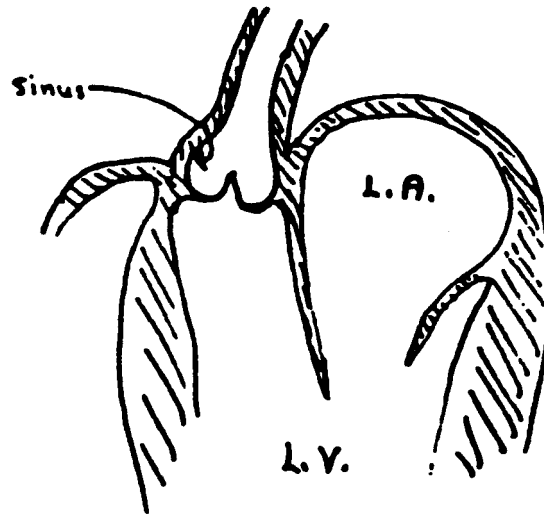


Figure 31: Sinuses of Valsalva of the Aortic Valve.

In the normal valve, blood flow is laminar, and furthermore, the reversed flow is less than 5% of the stroke volume. The fact that flow is observed to be laminar despite peak Reynolds numbers of near 10^4 suggests that the normal valve offers no obstruction to forward flow. On the other hand the very low reverse flow at the end of systole suggests the valve may be almost closed before the end of systole. It also seems important that the ostia of the coronary arteries not be occluded during the heart cycle.

The movement of the valve cusps has been studied by Bellhouse by means of a model system, and they have demonstrated the importance of the sinuses. The observations are:

At the start of systole the valve cusps open rapidly and move out toward the sinuses. Vortices formed between the cusps and the sinus walls. The cusps did not flutter, and flow entered each sinus at the ridge, curled back around the sinus wall, and then along the cusp to flow out into the main stream at the points of attachment of the cusp to the aorta. Thus, the valve leaflets are supported, during ejection, between the main stream and the trapped vortices.

After peak ejection velocity, as blood was being decelerated (but still moving out of the heart), the valve cusps move away from the sinuses, and are almost completely closed before the end of systole. Reverse flow was less than 5% of the forward S.V.

If the sinuses are occluded, the cusps open and touch the walls of the aorta during systole, and there are no trapped vortices. The valve closes by means of reversed flow above, and back flow increased to 25% of the forward flow.

The action of the valve may be explained on the basis of local pressure gradients in the regions of the aortic root. During deceleration of the blood (latter half of systole) there must be a reverse

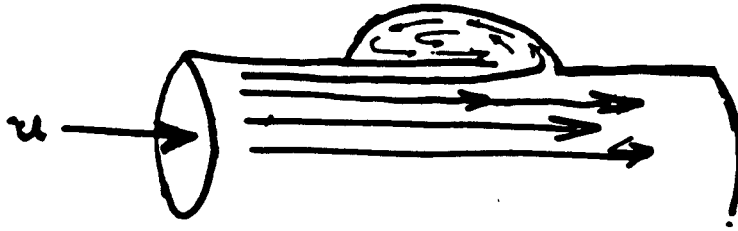


Figure 32: Vortices in the Sinuses of Valsalva in Systole.

pressure gradient in the region of the aortic outlet. In the figure below, $p_a > p_1$. If the velocities in the aorta and in the vortex are the same, then $p_c \approx p_a$. Hence:

$$\frac{p_a - p_1}{\Delta L} = -\rho \frac{\partial u}{\partial t}$$

and hence

$$p_c - p_1 = \Delta L \rho \frac{\partial u}{\partial t}$$

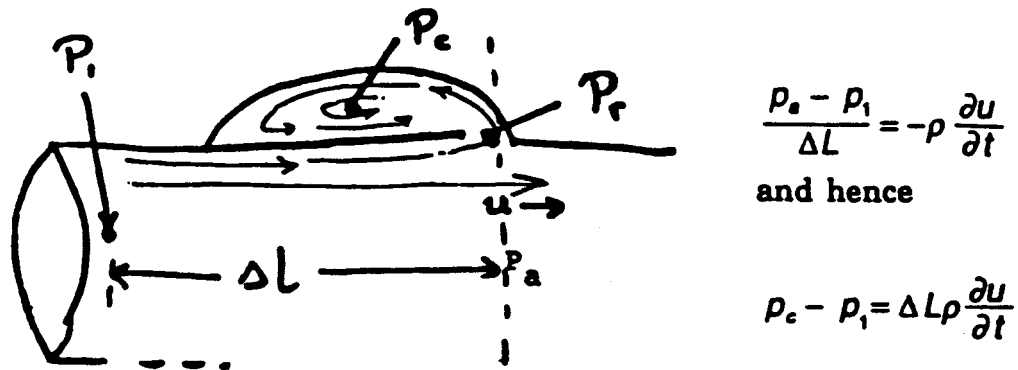


Figure 33: Closure of the aortic valve due to temporal deceleration.

Since $P_c - P_1$ is > 0 during deceleration, the valve leaflets tend to close. The figure below shows data from a simulated aortic valve system designed by Bellhouse. The measurements include fluid velocity, u , and the pressures p_1 , p_r , and p_c . Note change in direction of the pressure gradient across the valve ($p_c - P_p$) as a function of time during systole.

10.1.1 Vortex formation in the ventricle during filling.

The vortex behind the anterior leaflet was stronger than the posterior vortex, tending to close the anterior leaflet first.

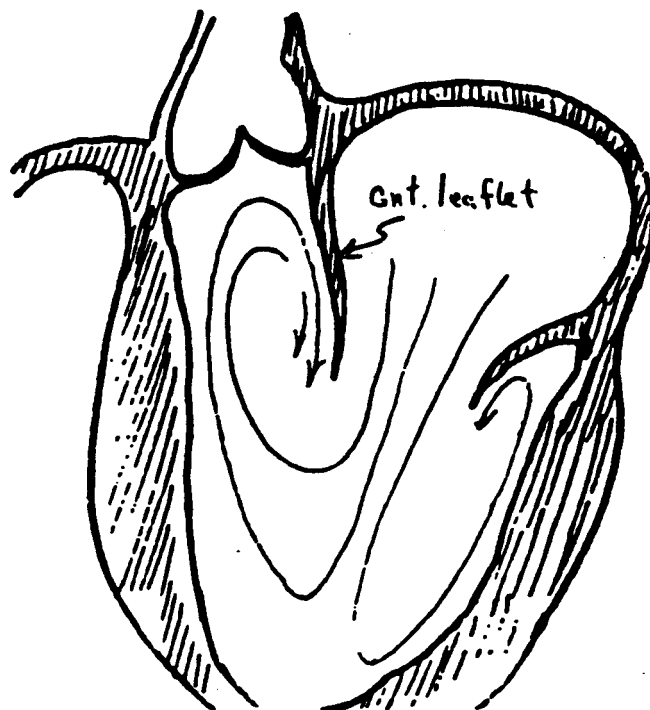


Figure 34: Closure of the mitral valve.

Table 5: Geometry of Mesenteric Vascular Bed of the Dog*

Site	Ascending aorta	Descending aorta	Abdominal aorta	Femoral artery	Carotid artery	Arteriole	Capillary	Venule	Inferior vena cava	Main pulmonary artery
Internal diameter d_i (cm)	1.5 1.0-2.4	1.3 0.8-1.8	0.9 0.5-1.2	0.4 0.2-0.8	0.5 0.2-0.8	0.005 0.001-0.008	0.0006 0.0004-0.0008	0.004 0.001-0.0075	1.0 0.6-1.5	1.7 1.0-2.0
Wall thickness h (cm)	0.065 0.05-0.08		0.05 0.04-0.06	0.04 0.02-0.06	0.03 0.02-0.04	0.002	0.0001	0.0002	0.015 0.01-0.02	0.02 0.01-0.03
h/d_i	0.07 0.055-0.84		0.06 0.04-0.09	0.07 0.055-0.11	0.08 0.053-0.095	0.4	0.17	0.05	0.015	0.01
Length (cm)	5	20	15	10	15 10-20	0.15 0.1-0.2	0.06 0.02-0.1	0.15 0.1-0.2	30 20-40	3.5 3-4
Approximate cross-sectional area (cm ²)	2	1.3	0.6	0.2	0.2	2×10^{-5}	3×10^{-7}	2×10^{-5}	0.8	2.3
Total vascular cross-sectional area at each level (cm ²)	2	2	2	3	3	125	600	570	3.0	2.3
Peak blood velocity (cm s ⁻¹)	120 40-290	105 25-250	55 50-60	100 100-120		$\left\{ \begin{array}{l} 0.75 \\ 0.5-1.0 \end{array} \right\}$	$\left\{ \begin{array}{l} 0.07 \\ 0.02-0.17 \end{array} \right\}$	$\left\{ \begin{array}{l} 0.35 \\ 0.2-0.5 \end{array} \right\}$	25 15-40	70
Mean blood velocity (cm s ⁻¹)	20 10-40	20 10-40	15 8-20	10 10-15					15 6-28	
Reynolds number (peak)	4500	3400	1250	1000		0.09	0.001	0.035	700	3000
α (heart rate 2 Hz)	13.2	11.5	8	3.5	4.4	0.04	0.005	0.035	8.8	15
Calculated wave-speed c_0 (cm s ⁻¹)	580		770	840	850				100	350
Measured wave-speed c (cm s ⁻¹)	500 400-600		700 600-750	900 800-1030	800 600-1100				400 100-700	250 200-330
Young's modulus E (Nm ⁻² $\times 10^5$)	4.8 3-6		10 9-11	10 9-12	9 7-11				0.7 0.4-1.0	6 2-10

* (From C. G. Caro, T. J. Pedley, and W. A. Seed (1974). "Mechanics of the circulation," Chapter 1 of *Cardiovascular Physiology* (ed. A. C. Guyton). Medical and Technical Publishers, London.)

Appendix A

Laminar Viscous Flow in Rigid Tubes

Poiseuille, a French Physician, was interested in estimating the pressure drop in various parts of the circulation. On a somewhat less formal basis than the Navier Stokes equation above, he reasoned that to maintain a steady flow in a tube, one had to balance the viscous forces retarding the flow with a pressure drop from inlet to outlet.

If we assume steady flow $\left[\frac{\partial \vec{v}}{\partial t} = 0\right]$ and straight parallel flow $[\vec{v} = u(x)]$, so $(\vec{v} \cdot \vec{\nabla})\vec{v} = 0$, then equation 17 simplifies to

$$-\vec{\nabla} p + \mu \nabla^2 \vec{v} = 0 \quad (42)$$

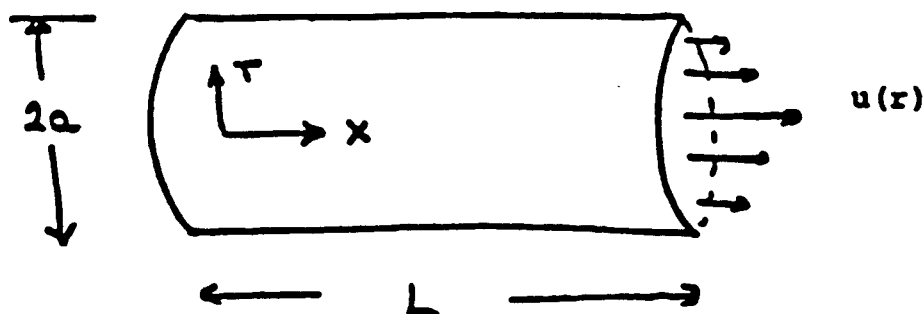


Figure 35: Poiseuille Flow

For the geometry of Figure 35, equation 42 becomes (in cylindrical coordinates):

$$\frac{dp}{dx} = \mu \frac{1}{r} \frac{d}{dr} \left(r \frac{du}{dr} \right) \quad (43)$$

This is a separable partial differential equation. If the tube is L long, then

$$\frac{dp}{dx} = \text{constant} = -\frac{p(0) - p(L)}{L} \quad (44)$$

The velocity $u(r)$ is subject to the two boundary conditions:

1. $u = 0$ at $r = a$ (no-slip condition at surface of tube)
2. $\frac{du}{dr} = 0$ at $r = 0$ (continuous velocity of at origin)

The form of $u(r)$ satisfying these boundary conditions and $\nabla^2 \vec{v} = \text{constant}$ is the parabolic profile

$$\frac{u(r)}{u(0)} = 1 - \frac{r^2}{a^2}$$

and the total flow rate, Q , through the pipe is

$$\begin{aligned} Q &= \int_0^a 2\pi r \, dr \cdot u(r) \\ &= \frac{1}{2}\pi a^2 u(0) \end{aligned} \quad (45)$$

Combining (43) and (45),

$$\mu \left[\frac{1}{r} \frac{d}{dr} \left(r \frac{du}{dr} \right) \right] = -\frac{4\mu u(0)}{a^2} = -\frac{8\mu Q}{\pi a^4}$$

and, with (44) we have the *Poiseuille flow* relation

$$\frac{p(0) - p(L)}{L} = \frac{8\mu Q}{\pi a^4} \quad (46)$$

Equation (46) may be rewritten in the form:

$$p(0) - p(L) = QR$$

where

$$R = \frac{8}{\pi} \cdot \mu \cdot \frac{L}{a^4} \quad (47)$$

Here R is termed the “resistance” of the tube and is directly proportional to length and viscosity and inversely proportional to the fourth power of the radius. Thus, one would expect that the major contribution to vascular resistance would be made by the small vessels. Note that the *mean* velocity, \bar{v} , is *half* the centerline velocity:

$$\bar{v} = \frac{Q}{\pi a^2} = \frac{1}{2}u(0) \quad (48)$$

Although Poiseuille’s law has many engineering applications, and although it gives considerable insight into flow in the circulation; nevertheless, it cannot be rigorously applied to the circulation. It requires the following assumptions:

1. The fluid is homogeneous and Newtonian. Blood may be considered as a Newtonian fluid only if the radius of the vessel exceeds 0.5 mm and if the shear rate exceeds 100 sec.⁻¹. This condition, therefore, excludes arterioles, venules, and capillaries.
2. The flow is steady and inertia-free. If the flow is pulsatile, the variable pressure gradient communicates kinetic energy to the fluid, and the flow is no longer inertia-free. This condition excludes the larger arteries.
3. The tube is rigid so that its diameter does not change with pressure. This condition is never met in the circulatory system, particularly the veins.

More precise models of blood flow in the circulation have been devised which take account of the properties of vessel walls, inertia, pulsatile flow, etc.

REFERENCES

1. Gehrhart, P.M., and Gross, R.J. *Fundamentals of Fluid Mechanics*. Addison-Wesley, Reading, Mass. 1985. This is a college-level, introductory text in fluid mechanics for engineering students. It is clearly written, and will provide a useful reference.
2. Fung, Y.C. *Biomechanics: Mechanical Properties of Living Tissues*. Springer-Verlag, New York, 1981. This book is an engineering consideration of the mechanical properties of a variety of tissues including blood (chs. 3-5).
3. Middleman, S. *Transport Phenomena in the Cardiovascular System*. Wiley & Sons, 1972. An excellent monograph by a chemical engineering who discusses quantitatively such areas as rheology of blood, capillary flow and exchange, flow in elastic arteries, etc.
4. Caro, C.G., Pedley, T.J. et al. *The Mechanics of the Circulation*. Oxford University Press, 1978. An engineering view of the circulation with chapters on background mechanics, the mechanical properties of blood, cardiac function, arterial blood flow including wave propagation, the microcirculation, flow in veins, and the mechanics of the pulmonary circulation. The book is written to be understandable.