7

Fluid Pressure, Fluid Flow in the Body, and Motion in Fluids

In the following two chapters we will examine the flow of two fluids in the body: blood in the heart and circulatory system, and air in the lungs and respiratory system. Flow of fluids elsewhere in the body is also important, such as in the urinary system (urine, liquids through the kidneys, etc.). Such directed flow of material in the body occurs predominantly by fluid flow in systems of vessels. Directed transport also occurs on microtubules in the body by motor proteins, such as kinesin and dynein, as described in Chap. 5. Undirected motion occurs by diffusion. Over "longer" distances such direct transport is preferred to diffusion because it provides a directed motion and a motion that is faster than diffusion. Diffusion is important in the body only over very short distances, up to $\sim 100\,\mu\text{m}$.

In this chapter we will discuss the concept of pressure as it relates to fluids in the body. For example, the pressure of the vitreous humor in the eyeball serves several functions, including maintaining the shape of the eyeball. This pressure is similar to the stress we examined in Chap. 4, such as that in our long bones when we walk. They both describe a force per unit area. The pressure in the fluid is hydrostatic, i.e., the force per unit area is the same stress in all directions. In solids the stress is often anisotropic. We will review the basic physics of pressure and fluid flow, including the relationship of pressure and fluid flow, and diffusion [353]. We will also examine the flow of humans in fluids, i.e., swimming, along with the possibility of human flight.

7.1 Characteristic Pressures in the Body

7.1.1 Definition and Units

The pressure of a fluid column is given by (2.48), $P = \rho g h$, where ρ is the fluid density, g is the gravitational constant, and h is the height of the column.

For mercury ρ is $13.6\,\mathrm{g/cm^3}$. For water $\rho = 1.00\,\mathrm{g/cm^3}$ at 4°C. The density of whole blood is a bit higher, $1.06\,\mathrm{g/cm^3}$ at 37°C. The units of pressure are presented in Table 2.6.

So far we have been discussing *absolute pressure*, P_{abs}, which is the total force per unit area. In discussions concerning the body it is very common to cite the *gauge pressure*, P_{gauge}, which is the pressure relative to a standard, which is usually atmospheric pressure, and so $P_{\mathrm{gauge}} = P_{\mathrm{abs}} - 1\,\mathrm{atm}$. This is helpful because it is the difference in pressure that is the net force that acts on a unit area. In discussing blood pressure and the pressure of air in the lungs, it is assumed that the term pressure P refers to the gauge pressure relative to the local atmospheric pressure. During breathing in (which is called inspiration), the pressure in the lungs is lower than that outside the body and so the internal (gauge) pressure is <0. Table 7.1 gives typical pressures in the body.

Table 7.1. Typical (gauge) pressures in the body (in mmHg). (Using data from [345])

arterial blood pressure	
maximum (systolic)	100–140
minimum (diastolic)	60–90
capillary blood pressure	
arterial end	30
venous end	10
venous blood pressure	
typical	3–7
great veins	<1
middle ear pressure	
typical	<1
eardrum rupture threshold	120
eye pressure	
humors	20 (12–23)
glaucoma threshold range	∼21–30
cerebrospinal fluid pressure	
in brain – lying down	5–12
gastrointestinal	10–12
skeleton	
long leg bones, standing	∼7,600 (10 atm.)
urinary bladder pressure	
voiding pressure	15–30 (20–40 $\mathrm{cmH_2O}$)
momentary, up to	120 (150 $\mathrm{cmH_2O}$)
intrathoracic	
between lung and chest wall	−10

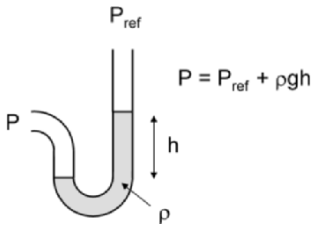

Fig. 7.1. Manometer

7.1.2 Measuring Pressure

One way of directly measuring pressure is with a manometer (Fig. 7.1). The measured pressure is that corresponding to the height of the fluid column plus the reference pressure, so

$$P = P_{\text{ref}} + \rho g h. \tag{7.1}$$

The most common way to measure blood pressure is with a *sphygmomanometer* (sfig-muh-ma-nah'-mee-ter), which consists of a cuff, a squeeze bulb, and a meter that measures the pressure in the cuff (Fig. 7.2). The cuff is the balloon-like jacket placed about the upper arm above the elbow; this

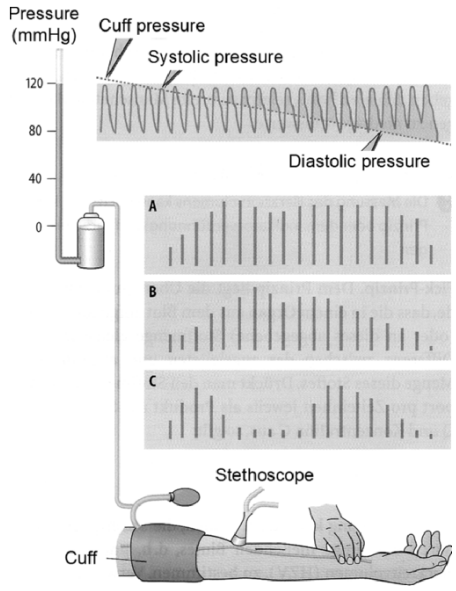

Fig. 7.2. Measuring blood pressure with a sphygmomanometer, listening to Korotkoff sounds (of varying levels during the turbulent flow shown in **A**–**C**). (Listening to sounds is called *auscultation*). (From [364])

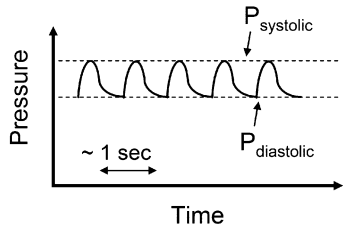

Fig. 7.3. Variation of blood pressure with time, for blood leaving the left heart for the systemic system, with the systolic and diastolic pressures shown

encircles the brachial artery. The cup of a stethoscope is placed on the lower arm, just below the elbow, to listen for the flow of blood. With no pressure in the cuff, there is normal blood flow and sounds are heard through the stethoscope. Gurgling sounds are heard after the cuff is pressurized with the squeeze bulb and then depressurized by releasing this pressure with a release valve in this bulb.

To understand when these sounds occur and their significance, we need to understand how the pressure in the main arteries varies with time. (This will be detailed in Chap. 8.) In every heart beat cycle (roughly 1/s), the blood pressure in the major arteries, such as the brachial artery, varies between the systolic pressure (∼120 mmHg) and the diastolic pressure (∼80 mmHg), as is depicted in Fig. 7.3. (The units of these cited gauge pressures are in mmHg – see (7.1) and Chap. 2.) When the pressure in the cuff exceeds the systolic pressure, there is no blood flow to the lower arm and consequently there are no sounds. When the pressure in the cuff is lowered with the release bulb to just below the systolic pressure, there is intermittent flow. During the part of the cycle when the arterial blood pressure is lower than the cuff pressure there is no flow; when it is greater, there is flow. This intermittent flow is turbulent and produces gurgling sounds. These sounds, the *Korotkoff* or K *sounds*, are heard by the stethoscope. As the cuff pressure is lowered further, the K sounds get louder and then lower, and are heard until the cuff pressure decreases to the diastolic pressure. Blood flow is not interrupted when the cuff pressure is less than the diastolic pressure and the K sounds cease because the blood flow is no longer turbulent. Therefore, the onset and end of the K sounds, respectively, denote the systolic and diastolic blood pressures. (This auscultatory method of Korotkoff was introduced by Russian army physician Korotkoff [362] who discovered a century ago that sound can be heard distally from a partially occluded limb [349].)

7.2 Basic Physics of Pressure and Flow of Fluids

In this section we overview the basics of fluids. Some of this will be a review for most. Some of the more advanced results are derived, while others are merely presented. These basics will be used in subsequent chapters.

Both gas and liquid fluids are important in the body. Gases will be treated by the ideal gas law

$$P = nRT, \tag{7.2}$$

where P is the pressure, n is the gas density, R is the gas constant ($= 8.31\,\mathrm{J/mol\text{-}K}$), and T is the temperature (in K). The gas density $n = N/V$, where N is the total number of molecules in a volume V. The gas constant $R = N_\mathrm{A} k_\mathrm{B}$, where N_A is Avogadro's number, 6.02×10^{23}, and k_B is Boltzmann's constant, $1.381 \times 10^{-23}\,\mathrm{J/K}$.

One guiding principle is *Pascal's Principle*: the pressure applied to a confined fluid increases the pressure throughout by the same amount. Also quite important is *Archimedes' Principle*: the buoyant force on a body immersed in a fluid is equal to the weight of the fluid displaced by that object. Another important relation is the *Law of Laplace*, which relates the difference of pressures inside and outside a thin-walled object – of a given shape – to the tension in the walls of the object. We will also need to understand the properties of flowing fluids to be able to analyze the physics of the circulatory system.

7.2.1 Law of Laplace

The pressure inside blood vessel walls, P, exceeds that outside, P_ext, by $\Delta P = P - P_\mathrm{ext}$. *How large of a tension should the vessel walls be able to withstand to support this positive pressure difference in equilibrium?* The answer is provided by the *Law of Laplace* for hollow cylinders. It is derived here and then used in Chap. 8.

Consider a tube of radius R and length L. Figure 7.4a shows a section of this tube with angle $\theta \ll 1$. The outward force (upward in the diagram) on this

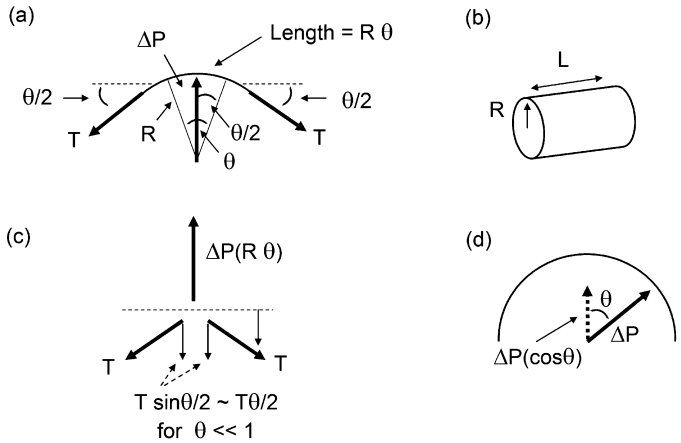

Fig. 7.4. Derivation of the Law of Laplace for the cylinder in (**a**), with the force diagram for a section of a cylinder in (**b**), leading to the force diagram in (**c**), and the resolution of pressures for analysis of a half cylinder in (**d**)

area is the pressure difference, ΔP, times the area, $(R\theta)L$. The circumferential tension T is the force per unit length (along the tube length). (Note that this use of the word "tension" has a different meaning than in earlier chapters, where it meant a force, often used to pull things apart.) This film tension has units of force/length or energy/area. It is equal to a circumferential stress $\sigma = T/w$, where w is the cylinder thickness (with $w \ll R$). These forces can be those within the blood vessel walls (Chap. 8). The horizontal components of the film tension to the left and right cancel. The vertical components are inward and each equal to $T\sin(\theta/2) \simeq T(\theta/2)$ for small angles. With both of these tension components multiplied by L, in static equilibrium force balance gives

$$\Delta P(R\theta)L = 2\left(T\frac{\theta}{2}\right)L. \tag{7.3}$$

This means

$$\Delta P = \frac{T}{R} \qquad \text{or} \qquad T = R(\Delta P). \tag{7.4}$$

This is a differential method. Alternatively we could integrate the forces over a half cylinder, as shown in Fig. 7.4d. The total downward force is the area of the walls, $2wL$, times the stress, σ, or $2wL\sigma$. The total upward force is the cross-sectional area, $2RL$, times the pressure difference, ΔP, or $2RL(\Delta P)$. In equilibrium

$$2RL(\Delta P) = 2wL\sigma \tag{7.5}$$

$$\Delta P = \frac{w\sigma}{R} = \frac{T}{R}, \tag{7.6}$$

which is the same as (7.4). (Figure 7.4d shows that the total upward force is really the integral of the upward force component, $\Delta P\cos\theta$, times the area element, $RL\mathrm{d}\theta$, integrated from -90° to 90° or

$$(\Delta P)RL\int_{-90^\circ}^{90^\circ}\cos\theta\mathrm{d}\theta = (\Delta P)RL(\sin(90^\circ) - \sin(-90^\circ)) = 2(\Delta P)RL, \tag{7.7}$$

which turns out to be the same as $(\Delta P)2RL$.)

The Law of Laplace is also important in spheres, such as soap bubbles and the alveoli in the lungs. For a sphere of radius R and wall thickness w, we can balance the forces in the half sphere. The total downward force is the area of the walls, $2\pi Rw$, times the stress, σ, or $2\pi Rw\sigma$. The total upward force is the cross-sectional area, πR^2, times the pressure difference, ΔP, or $\pi R^2(\Delta P)$. In equilibrium

$$\pi R^2(\Delta P) = 2\pi Rw\sigma \tag{7.8}$$

$$\Delta P = \frac{2w\sigma}{R} = \frac{2T}{R}. \tag{7.9}$$

Table 7.2. Surface tension (γ) for several liquids. (Using data from [351, 358, 363])

liquid	T (°C)	γ (10^{-4} N/m)
water	0	7.56
	20	7.28
	60	6.62
	100	5.89
whole blood	20	5.5–6.1
blood plasma	20	5.0–5.6
lung surfactant	20	0.1
cerebrospinal fluid	20	6.0–6.3
saliva	20	1.5–2.1
benzene	20	2.89
mercury	20	46.4

This is the Law of Laplace for a sphere. We will use it in Chap. 9. (It is derived in more detail in Problem 7.12.)

For a spheroid with different radii of curvature, R_1 and R_2, (7.4) and (7.9) generalize to

$$\Delta P = \frac{T}{R_1} + \frac{T}{R_2}. \tag{7.10}$$

For a cylinder, $R_1 = R$ and $R_2 = \infty$ and this reduces to (7.4). For a sphere, $R_1 = R$ and $R_2 = R$ and it reduces to (7.9).

Our force balance arguments have made a direct connection between this tension, or really surface tension, and its units of force/length. Surface tension also has the same units as energy/area. This is reasonable because it is also the energy "cost" of making a unit area of a surface (or interface). Representative values of surface tension are given in Table 7.2.

7.2.2 Fluids in Motion

There are five attributes of the flow of fluids:

1. Flow can be *laminar/streamline/steady* or *turbulent/unsteady*. In laminar flow, a particle in the flow moves in a smooth manner along well-defined streamlines. In contrast, the motion is very random locally in turbulent flow. The Reynolds number Re is a dimensionless figure of merit that crudely divides the regimes of laminar and turbulent flow. It is the ratio between inertial force ($\rho u^2/2$; ρu^2 is used here) and viscous force ($\rho \eta u/d$) per unit volume on the fluid, where ρ is the fluid density, u is the average speed of flow, d is the tube diameter, and η is the fluid *coefficient of viscosity* or the *dynamic or absolute viscosity*, which is defined later. This

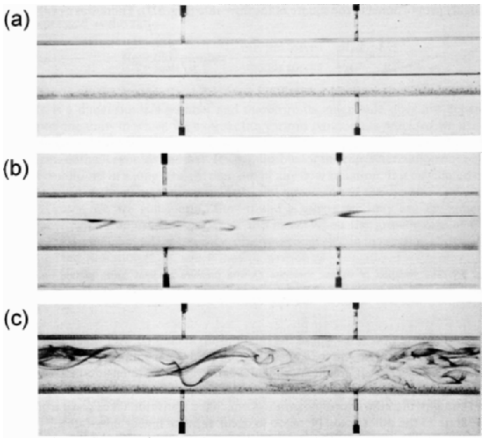

Fig. 7.5. Motion of a filament of dye in a straight pipe, showing (**a**) steady, laminar flow at low *Re*, (**b**) short bursts of turbulence for *Re* above the critical value, and (**c**) fully turbulent flow with random motion of the dye streak for higher *Re*. (From [346]. Used with permission of Oxford University Press)

gives

$$Re = \frac{\rho u^2}{\rho \eta u/d} = \frac{\rho u d}{\eta} = \frac{ud}{\upsilon}, \tag{7.11}$$

where $\upsilon = \eta/\rho$ is the coefficient of *kinematic viscosity*.
Although this dividing line is not hard and fast, generally, flow in a rigid tube with $Re < 2{,}000$ is laminar and that with $Re > 2{,}000$ is turbulent. This dividing region is often cited as being between 1,200–2,500, and in the higher range for smoother-walled tubes. Figure 7.5 shows flow in the laminar and turbulent regimes, and in the transition region between them.

2. Flow can be *compressible* or *incompressible*. Gases, such as air, are very compressible. Liquids are less compressible, and are often approximated as being incompressible.
3. Flow can be *viscous* or *nonviscous*. Fluids (other than superfluids) always have some viscosity, but in some cases it can be ignored totally, or first ignored and then considered as a perturbation.
4. Flow can be *rotational* or *irrotational*. In the cases we will consider there is no local rotation (such as vortices), so the flow will be irrotational.
5. Flow can be *steady* (constant in time) or *pulsatile* (with pulsing changes). Blood flow in the body is pulsatile, but is commonly treated as being in steady state in simple models. We will use both steady and pulsatile models in Chap. 8.

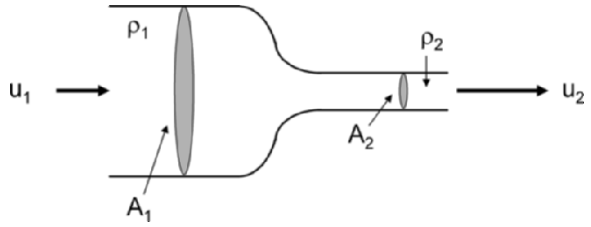

Fig. 7.6. Continuity of flow when the tube cross-sectional area changes

7.2.3 Equation of Continuity

The equation of continuity is a statement of the conservation of mass during flow. As seen in Fig. 7.6, when a fluid of a given mass density ρ moves with average speed u in a tube of cross-sectional area A, the product ρAu is constant (i.e., it is conserved). Because the speed is a longitudinal distance per unit time, Au is the volume flow per unit time (because $A \times$ distance = volume). Consequently, ρAu is the mass per unit time. In steady state, the same mass flows into a volume and leaves it. For the regions marked 1 and 2 in Fig. 7.6, this means that

$$\rho_1 A_1 u_1 = \rho_2 A_2 u_2. \tag{7.12}$$

If the fluid is incompressible, the density in $\rho_1 A_1 u_1 = \rho_2 A_2 u_2$ does not change with pressure and is the same everywhere. With $\rho_1 = \rho_2$, we follow the volume or volumetric flow rate Q, which is now a constant. This means $Q_1 = A_1 u_1$ and $Q_2 = A_2 u_2$, and so the continuity equation becomes $Q = Q_1 = Q_2$ with

$$Q = A_1 u_1 = A_2 u_2. \tag{7.13}$$

7.2.4 Bernoulli's Equation

Bernoulli's Principle (or equation) relates the average flow speed u, pressure P, and height y of an incompressible, nonviscous fluid in laminar, irrotational flow (Fig. 7.7). At any two points

$$P_1 + \frac{1}{2}\rho u_1^2 + \rho g y_1 = P_2 + \frac{1}{2}\rho u_2^2 + \rho g y_2. \tag{7.14}$$

The densities $\rho_1 = \rho_2 = \rho$ for this incompressible fluid. (Bernoulli's equation actually applies to any two points along a streamline.)

There are three special cases of Bernoulli flow. (1) For static fluids ($u = 0$), and Bernoulli equation's reduces to $P_1 + \rho g y_1 = P_2 + \rho g y_2$. (2) It reduces to Torricelli's theorem when $P_1 = P_2$, namely $\rho u_1^2/2 + \rho g y_1 = \rho u_2^2/2 + \rho g y_2$.

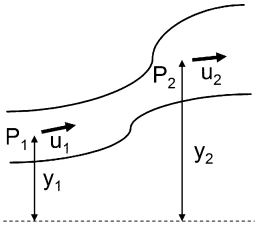

Fig. 7.7. For irrotational and nonviscous flow, the pressure, flow speed, and height are related by Bernoulli's equation along any streamline

(3) It reduces to the Venturi flow regime when $y_1 = y_2$ (Fig. 7.8), so

$$P_1 + \frac{1}{2}\rho u_1^2 = P_2 + \frac{1}{2}\rho u_2^2. \tag{7.15}$$

Because the continuity of flow in such a Venturi tube is $A_1 u_1 = A_2 u_2$

$$u_2 = \frac{A_1}{A_2} u_1. \tag{7.16}$$

Therefore we find

$$P_1 + \frac{1}{2}\rho u_1^2 = P_2 + \frac{1}{2}\rho \left(\frac{A_1}{A_2} u_1\right)^2 \tag{7.17}$$

and

$$P_2 - P_1 = \frac{1}{2}\rho u_1^2 \left(1 - \left(\frac{A_1}{A_2}\right)^2\right). \tag{7.18}$$

With $A_2 < A_1$, we see that $u_2 > u_1$ and $P_2 < P_1$. This shows that the flow becomes faster and the pressure becomes lower in clogged blood vessels.

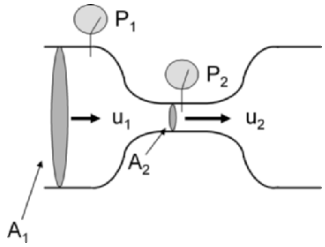

Fig. 7.8. Flow in a tube when the tube cross-sectional area changes. This is a Venturi tube, for which pressure and flow speed are related by Bernoulli's equation in the limit of constant height

7.2.5 Interactions among the Flow Parameters

Pressure P, volume V, and flow rate Q are all interrelated in flow through vessels, be it blood flow in the circulatory system or air flow in breathing. *Resistance* R_{flow} is the pressure difference ΔP needed to cause a given flow rate Q

$$R_{\text{flow}} = \frac{\Delta P}{Q}. \tag{7.19}$$

Compliance C_{flow} is the change in volume caused by a change in pressure in a vessel

$$C_{\text{flow}} = \frac{\Delta V}{\Delta P}. \tag{7.20}$$

Occasionally, the *inertance* L_{flow} is also defined. It is the change in pressure caused by a change in flow rate

$$L_{\text{flow}} = \frac{\Delta P}{\Delta Q}. \tag{7.21}$$

See Appendix D for an analog to electrical circuits.

7.2.6 Viscous Flow and Poiseuille's Law

Bernoulli's equation would predict that the pressure does not change during flow if the tube cross-section and height do not change. This is true for an ideal, nonviscous fluid. Viscosity is the friction during flow. It is always present and causes the pressure to drop during flow.

The coefficient of (dynamic or absolute) viscosity η is formally defined in (7.22), which gives the tangential or shear force F required to move a fluid layer of area A at a constant speed v, in the x direction, when that layer is a distance y from a stationary plate (Fig. 7.9) [350, 354]

$$F = \eta \frac{A}{y} v. \tag{7.22}$$

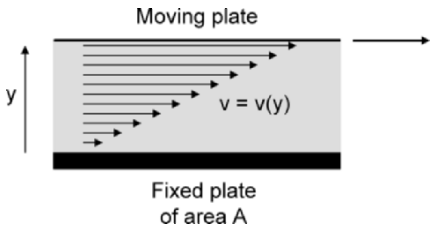

Fig. 7.9. Viscous fluid flow, with a linear gradient of fluid speed with position between a fixed and moving plate. This is shown for Newtonian flow

This equation is also written as

$$\tau = \eta \frac{\mathrm{d}v}{\mathrm{d}y}, \tag{7.23}$$

where $\tau = F/A$ is the shear stress, as in (4.5) and Figs. 4.10 and 4.11, and $\mathrm{d}v/\mathrm{d}y$ is called the *shear rate*. (Check that the units of the shear rate are those that a rate should have, 1/s.) Fluids that are characterized by (7.22) and (7.23) are called "Newtonian fluids" and are said to undergo "Newtonian flow."

The SI units of η are $(\mathrm{N/m^2})\mathrm{s}$, which is equal to kg/m-s and Pa-s; this is called a Poiseuille (PI), but this unit is not often used. More commonly used than this last unit is the poise (P) which is $10\times$ smaller. It is a natural unit in the CGS units system with 1 poise = 1 g/cm-s = 0.1 $(\mathrm{N/m^2})\mathrm{s}$ = 0.1 kg/m-s = 0.1 Pa-s. Also common is the centipoise (cP), with 1 cP = 0.01 poise = 0.001 Pa-s, because the viscosity of water at 20°C is almost equal to 1 cP (and is actually 1.002 cP). We will usually use the units of Pa-s. Also, this viscosity coefficient is often called η by physicists (and is used as such here), whereas it is often called μ by biomedical engineers. It is also related to, but different from the viscosity damping constant for the dashpot c in (4.48).

Because of this drag, there must be a pressure difference (gradient) to maintain fluid flow in a tube. The relation between this pressure drop and the volumetric flow rate Q is given by Poiseuille's Law (or Hagen-Poiseuille's Law)

$$Q = \frac{\pi R^4}{8\eta L}(P_1 - P_2), \tag{7.24}$$

where R is the radius of the tube and L is its length (Fig. 7.10). This relation can be viewed as the flow rate for a given pressure drop. Alternatively, it can be viewed as the pressure drop when there is a flow Q in the tube

$$P_1 - P_2 = \Delta P = \frac{8\eta L}{\pi R^4}\, Q. \tag{7.25}$$

We will use this expression in Chap. 8 to determine the pressure drops in blood vessels during circulation. It is derived later as an advanced topic.

Equation (7.25) is formally analogous to Ohm's Law for resistors, $V = IR_{\mathrm{elect}}$ (or in a manner more parallel to this equation, $V = R_{\mathrm{elect}}I$), where V is the voltage or potential difference across the resistor and is the driving term (which is analogous to ΔP), R_{elect} is the electrical resistance (analogous to the resistance of flow $8\eta L/\pi R^4$ here, which we will call R_{flow}), and I is the electrical current, which is the flow resulting from the driving term (analogous to the volumetric flow Q here).

Consider a tube with cross-sectional area A. The net force on the fluid in it is $(\Delta P)A$. If this force moves the fluid a distance L, the work done on it is $FL = (\Delta P)AL$. If this volume AL is moved in a given time, the work needed

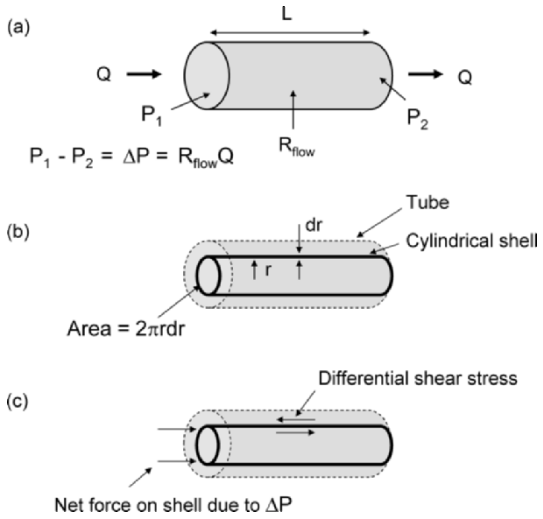

Fig. 7.10. Calculation of Poiseuille's Law for a tube in (**a**), using the cylindrical shell in (**b**), and balancing forces between the hydrostatic flow pressure force and the differential shear stress on the shell in (**c**)

to do this in this given time – the power – is

$$P_{\text{power, flow}} = (\Delta P)Q, \tag{7.26}$$

or $P_{\text{power, flow}} = Q^2 R_{\text{flow}} = (\Delta P)^2 / R_{\text{flow}}$. These expressions are analogous to those for the power dissipated by an electrical resistor: $P_{\text{power, elect}} = VI = I^2 R_{\text{elect}} = V^2 / R_{\text{elect}}$.

The coefficient of viscosity for water is 1.78×10^{-3} Pa-s at 0°C and it decreases with temperature, dropping to 1.00×10^{-3} Pa-s at 20°C and 0.65×10^{-3} Pa-s at 40°C. At 37°C, η is 1.5×10^{-3} Pa-s for blood plasma and 4.0×10^{-3} Pa-s for whole blood, which are both higher than that for water at the same temperature. (Blood is really thicker than water.) The coefficients of viscosity of common human body fluids and other materials are listed in Table 7.3. As is clear from the table, the viscosity of liquids decreases with increasing temperature T, because the kinetic energy of molecules increases with T and this can overcome intermolecular forces that slow down motion between the dense, adjacent layers. In contrast, viscosity increases with temperature for gases, as $T(\text{in K})^{1/2}$, because diffusion between adjacent layers increases with T.

Derivation of Poiseuille's Law (Advanced Topic)

Now consider flow in a tube of radius R (Fig. 7.10). The distance radially from the center line of the tube is r. Using (7.22) and (7.23), the shear force and

Table 7.3. Coefficient of viscosity η of common materials, in Pa-s (1 poise = 0.1 Pa-s). (Using data from [351, 358, 363])

material	T (°C)	η
water	0	1.78×10^{-3}
	20	1.00×10^{-3}
	37	0.69×10^{-3}
	50	0.55×10^{-3}
	100	0.28×10^{-3}
blood plasma	37	1.5×10^{-3}
whole blood[a]	37	$\sim 4.0 \times 10^{-3}$
low shear rate, Hct = 45%		$\sim 100 \times 10^{-3}$
low shear rate, Hct = 90%		$\sim 1{,}000 \times 10^{-3}$
high shear rate, Hct = 45%		$\sim 10 \times 10^{-3}$
low shear rate, Hct = 90%		$\sim 100 \times 10^{-3}$
cerebrospinal fluid	20	1.02×10^{-3}
interstitial fluid	37	$1.0\text{–}1.1 \times 10^{-3}$
human tears	37	$0.73\text{–}0.97 \times 10^{-3}$
synovial fluid[b]	20	>0.3
castor oil	20	1
motor oil, SAE 10	20	0.065
motor oil, SAE 50	20	0.54
machine oil, heavy	37	0.13
machine oil, light	37	0.035
ethylene glycol	37	0.011
mercury, liquid	37	1.465×10^{-3}
methanol	37	0.47×10^{-3}
ketchup	20	50
peanut butter	20	250
glass (anneal)	720–920 K	2.5×10^{12}
(blowing)	∼1,300 K	$\sim 1 \times 10^{6}$
(furnace)	1,500–1,700 K	$\sim 1 \times 10^{2}$
air	20	1.8×10^{-5}
	100	2.1×10^{-5}

Hct is the hematocrit, which is the volume fraction of red blood cells in blood.
[a] See Figs. 8.10 and 8.11.
[b] See Fig. 7.14.

stress are

$$F = \eta A \frac{\mathrm{d}v}{\mathrm{d}r} \tag{7.27}$$

$$\tau = \eta \frac{\mathrm{d}v}{\mathrm{d}r} \, . \tag{7.28}$$

Imagine a series of concentric cylinders within this tube of thickness dr and length L (centered about the center symmetry axis, Fig. 7.10b), with a pressure drop ΔP along L. The force pushing one of these cylindrical shells forward

is this pressure drop, ΔP, times the area of the front (and back) cylinder face, $2\pi r\mathrm{d}r$, or $(\Delta P)2\pi r\mathrm{d}r$. The viscous drag force that the cylindrical shell feels from the other shells (i.e., from the liquid) is the difference between the shear stress felt on its inner and outer surfaces $\times$ its surface area, $2\pi rL$. Using (7.28), this difference is

$$\frac{\mathrm{d}(2\pi rL\tau)}{\mathrm{d}r}\,\mathrm{d}r = \frac{\mathrm{d}\left(2\pi rL\eta\frac{\mathrm{d}v}{\mathrm{d}r}\right)}{\mathrm{d}r}\,\mathrm{d}r = 2\pi L\eta\frac{\mathrm{d}\left(r\frac{\mathrm{d}v}{\mathrm{d}r}\right)}{\mathrm{d}r}\,\mathrm{d}r \tag{7.29}$$

assuming the viscosity does not depend on r.

In steady state, the force due to the pressure drop plus the drag force equals zero, so

$$(\Delta P)2\pi r\mathrm{d}r + 2\pi L\eta\frac{\mathrm{d}\left(r\frac{\mathrm{d}v}{\mathrm{d}r}\right)}{\mathrm{d}r}\,\mathrm{d}r = 0 \tag{7.30}$$

or

$$\frac{\mathrm{d}\left(r\frac{\mathrm{d}v}{\mathrm{d}r}\right)}{\mathrm{d}r} = -\frac{\Delta P}{\eta L}\,r. \tag{7.31}$$

Integrating gives

$$r\frac{\mathrm{d}v}{\mathrm{d}r} = -\frac{\Delta P}{2\eta L}\,r^2 + C \tag{7.32}$$

$$\frac{\mathrm{d}v}{\mathrm{d}r} = -\frac{\Delta P}{2\eta L}\,r + \frac{C}{r}. \tag{7.33}$$

The constant C must equal zero, because otherwise the second term would be infinite at the center.

Integrating again gives

$$v(r) = -\frac{\Delta P}{4\eta L}\,r^2 + D. \tag{7.34}$$

(see Appendix C). Because at the tube radius the velocity is zero ($v(R) = 0$), D is determined and this gives

$$v(r) = -\frac{\Delta P}{4\eta L}\,r^2 + \frac{\Delta P}{4\eta L}\,R^2, \tag{7.35}$$

so

$$v(r) = \frac{\Delta P}{4\eta L}\left(R^2 - r^2\right) = \frac{R^2\Delta P}{4\eta L}\left(1 - \frac{r^2}{R^2}\right). \tag{7.36}$$

This speed is seen to be maximum in the center where $r = 0$. This maximum value of $R^2\Delta P/(4\eta L)$ decreases to 0 as r increases from 0 to R.

The flow rate in the tube Q equals uA when the speed is uniform across the area A. When it is not, as here, Q is obtained by integrating $v(r)$ across

the cross-sectional area. This is done by multiplying $v(r)$ by the area element $2\pi r\mathrm{d}r$ (the circumference $\times$ the differential in r) and integrating r from 0 to R, which gives

$$Q = \int_0^R \frac{\Delta P}{4\eta L}\left(R^2 - r^2\right) 2\pi r\mathrm{d}r \tag{7.37}$$

$$Q = \int_0^R \frac{\pi\Delta P}{2\eta L}\left(rR^2 - r^3\right)\mathrm{d}r = \frac{\pi\Delta P}{2\eta L}\left(\frac{R^4}{2} - \frac{R^4}{4}\right) = \frac{\pi R^4\Delta P}{8\eta L}. \tag{7.38}$$

This is Poiseuille's Law $\Delta P = \left(8\eta L/\pi R^4\right) Q$ ((7.24) and (7.25)). Because Q is also equal to the area $\times$ the average speed, this average speed is

$$u = \frac{\pi R^4\Delta P/8\eta L}{\pi R^2} = \frac{R^2\Delta P}{8\eta L} \tag{7.39}$$

and

$$v(r) = 2u\left(1 - \frac{r^2}{R^2}\right). \tag{7.40}$$

This is depicted in the rightmost profile shown in Fig. 7.11.

Many fluids are non-Newtonian fluids (Fig. 7.12), which means they are not characterized by (7.22) and (7.23), but by other relations. We assumed earlier that a fluid could generate no shear stress at any shear or strain rate; this is a frictionless or nonviscous fluid, which is unrealistic except for superfluids. (Note that such shear or strain "rates" are really gradients with respect to the direction normal to flow, i.e., y, and not with respect to time t. However, this terminology is reasonable because they have the same units as strain rates and because of the scaling argument given in Problem 7.23.) In some real non-Newtonian fluids, the shear stress is $F/A = \eta(\mathrm{d}v/\mathrm{d}y)^n$, where n could be greater or less than 1, as in Fig. 7.12. This is sometimes phrased as $F/A = \eta'(\mathrm{d}v/\mathrm{d}y)$ where the effective viscosity $\eta' = \eta(\mathrm{d}v/\mathrm{d}y)^{n-1}$ depends on the strain rate; as such a Newtonian fluid

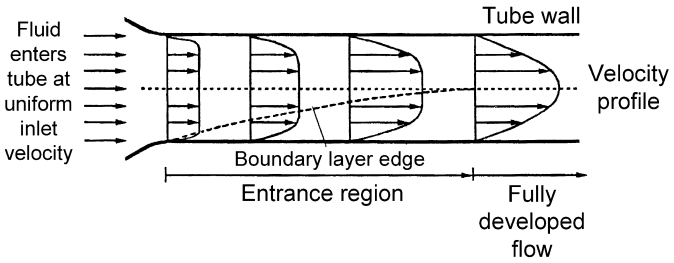

Fig. 7.11. Establishment of steady-state Newtonian flow into the parabolic velocity profile (in the fully developed flow). (From [351], based on [355]. Courtesy of Robert A. Freitas Jr., Nanomedicine, Vol. 1 (1999), http://www.nanomedicine.com)

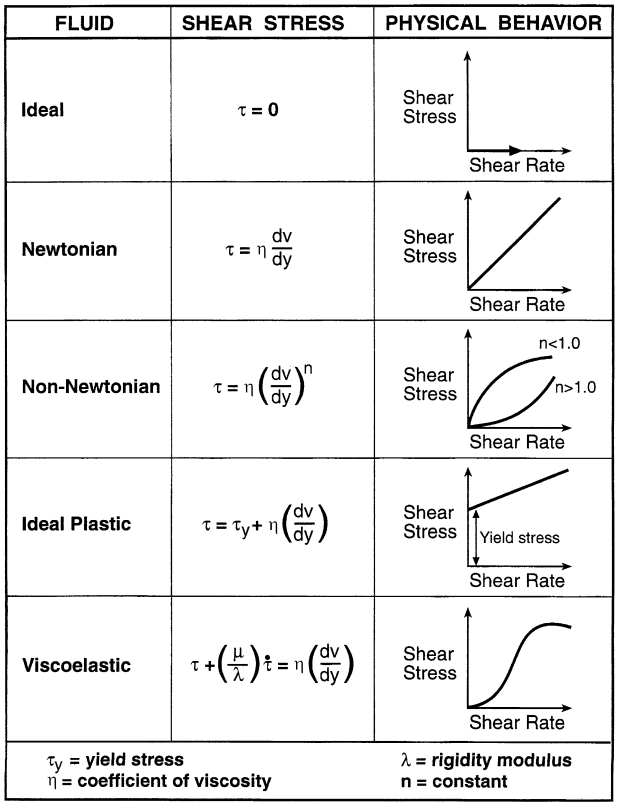

Fig. 7.12. Newtonian and non-Newtonian fluid flow. (From [357])

would have an effective viscosity that is independent of the strain rate. A dilatant or shear-thickening fluid has an effective viscosity that increases with increasing stress. A plastic or shear-thinning fluid has an effective viscosity that decreases with increasing stress. A Bingham plastic, such as toothpaste, has a finite yield stress even for $\mathrm{d}v/\mathrm{d}y = 0$, and above the yield stress it has a linear relationship with strain rate, $F/A = \alpha + \eta(\mathrm{d}v/\mathrm{d}y)$. The composition of blood makes it a non-Newtonian fluid; this is discussed in Chap. 8. Consequently, the flow pattern of blood is decidedly nonparabolic (Fig. 7.13).

Synovial fluid is one example of a non-Newtonian fluid. Figure 7.14 shows that its coefficient of friction is high at low shear rates and much lower at high shear rates. Figure 8.11 shows that whole blood is also a non-Newtonian fluid.

The dependence of flow on pressure drop within the laminar, intermediate, and turbulent regimes is shown in Fig. 7.15.

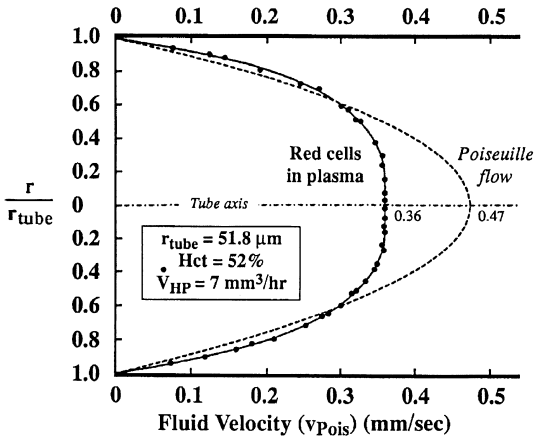

Fig. 7.13. Velocity flow profile of whole blood is blunted relative to the ideal parabolic flow of a Newtonian fluid. (From [351], based on [355]. Courtesy of Robert A. Freitas Jr., Nanomedicine, Vol. 1 (1999), http://www.nanomedicine.com)

Approach to Steady Flow

The results of Sect. 7.2.6 apply to steady, laminar flow. If a tube bifurcates – such as in branching arteries, the velocity profile we derived with its *boundary layer* at the tube circumference (where the flow velocity decreases to zero), will not represent the flow distribution immediately after the bifurcation. It will be valid only after a distance past the bifurcation called the entrance length, X [346]. Experimentally

$$X = 0.03d(Re) \tag{7.41}$$

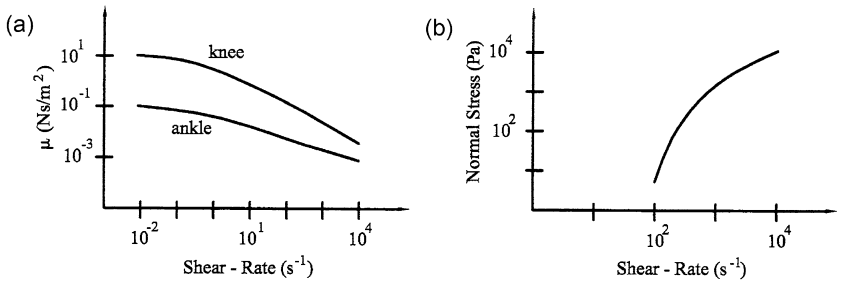

Fig. 7.14. (**a**) Synovial fluid is a non-Newtonian fluid, with a coefficient of friction that decreases with shear rate. (**b**) Another property of such a non-Newtonian fluid is that it can create a normal stress that depends on shear rate. (From [361])

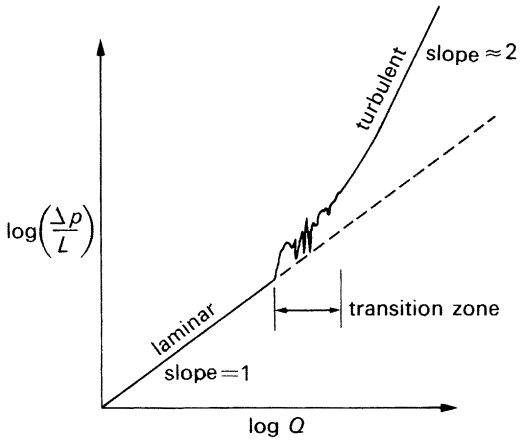

Fig. 7.15. Pressure drop per unit length vs. log of the flow rate for a long tube, showing a transition region between laminar and turbulent flow. (From [346]. Used with permission of Oxford University Press)

for a straight pipe, where d is the diameter ($d = 2R$). For the laminar flow regime with $Re < 10$, this is not valid and the entrance length is smaller; when $Re \ll 1$ and inertial forces can be ignored

$$X \sim d. \tag{7.42}$$

For $Re > 2{,}500$, the flow is likely turbulent and the entrance length (for steady state turbulent flow) is shorter than that for fast laminar flow

$$X = 0.693d(Re)^{1/4}. \tag{7.43}$$

The development of parabolic flow for a Newtonian fluid in the laminar flow region is illustrated in Fig. 7.11.

Flow in Curving Tubes such as Arteries

When you hold a hose with flowing water and try to change its direction you feel a resistance. This resistance is the force you need to apply to change the direction of the momentum of the water flow. This centripetal force becomes larger with faster flow rates (i.e., for larger hose areas and faster water flow speeds), as is well known to all firepersons. Curving arterial walls, such as the aorta, feel a pressure due to the difference in hydrostatic pressure inside and outside the vessel that arises from this force. This pressure is felt equally around the wall.

Consider a tube or artery of inner radius R that is turning with a radius $\mathcal{R}$. Figure 7.16 shows an arc of angle θ (in radians) $\ll 1$ of such a vessel. The average speed of flow is u and the mass density of blood is ρ. The magnitude

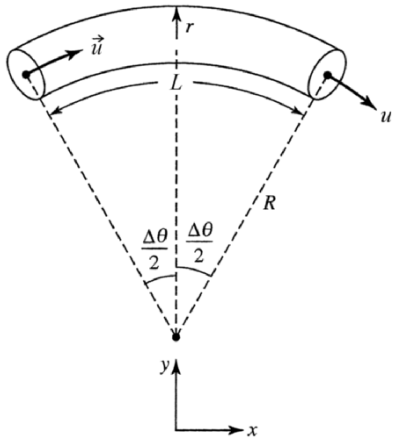

Fig. 7.16. Flow in a curved tube. (From [344])

of the momentum in the vessel per unit volume is $\rho u(AL)$, for a vessel with cross-sectional area $A = \pi R^2$ for a length L of blood flow. In traversing an angle θ, the momentum vector changes by $\sim(\rho u)(AL)\theta$. This occurs when the blood moves a distance $\mathcal{R}\theta$, given by the arc length, with a speed u, so this occurs in a time $\mathcal{R}\theta/u$. Consequently, the force needed to do this is the change of momentum per unit time, which is $(\rho uAL\theta)/(\mathcal{R}\theta/u) = \rho u^2 AL/\mathcal{R}$. Because the mass of this volume of blood is $m = \rho AL$, this looks like the centripetal force $mu^2/\mathcal{R}$. (It looks like it, because that is what it is.)

This force is distributed across the outer half of the inner arterial wall, which has a cross-sectional area πRL. Because the force is outward, there is a larger load on the outermost portions shown in Fig. 7.16 and a smaller load on the outer upper and lower regions. Therefore, the peak force per unit area is more accurate when you use a smaller effective area, say $\pi RL/2$. Consequently, the peak pressure is the force per unit area $\rho u^2 AL/\mathcal{R}$, with $A = \pi R^2$, divided by this area $\pi RL/2$, or

$$P_{\text{cent}} = \frac{\rho u^2(\pi R^2)L/\mathcal{R}}{\pi RL/2} = 2\rho u^2 \frac{R}{\mathcal{R}}. \tag{7.44}$$

Flow of Objects in Fluids: Drag and Lift

The viscosity of a fluid also creates a drag force on objects that move in the fluid [343]. The reason for this is clear from (7.22); such objects are just like the plate in Fig. 7.9 in this functional definition of viscosity. Viscosity causes the boundary layer of the fluid near the ball (or plate) to move with it. If the object is moving at a speed u relative to the fluid, this drag force on the object is given by *Stokes Law*

$$F_{\text{drag,Stokes}} = 6\pi R\eta u, \tag{7.45}$$

where R is the hydrodynamic radius of the object, which is about half the typical lateral dimension D. This expression for Stokes friction is valid when the flow speed is slow enough that the streamlines about it are laminar. Here this means the Reynolds number $Re = \rho Du/\eta = 2\rho Ru/\eta$ is smaller than $\sim$100.

For Reynolds numbers much larger than 100, viscosity is no longer totally dominant and the main drag force is due to the formation of vortices that appear and trail the object, particularly as turbulent flow becomes important. This *hydrodynamic drag force* is

$$F_{\text{drag,hydrodyamnic}} = \frac{1}{2} C_{\text{D}} A \rho u^2, \tag{7.46}$$

where A is the frontal surface area and C_{D} is the drag coefficient. For $100 < Re < 2 \times 10^5$, $C_{\text{D}} \simeq 1.0$ for circular cylinders. For spheres, C_{D} decreases from 1.0 to $\simeq$0.5 as Re increases from 100 to 1,000 and it remains about 0.5 for $1,000 < Re < 2 \times 10^5$. For both cylinders and spheres, C_{D} becomes smaller at somewhat higher Re. These vortices or eddies are produced at the *Strouhal frequency*

$$f_{St} = \frac{(St)u}{D}, \tag{7.47}$$

where St is the Strouhal number. St depends on C_{D} and Re, and is typically between 0.12 and 0.23.

Problem 7.39 examines which drag regime dominates for human motion in fluids: walking and running in air and swimming in water.

Another source of drag that is present at all speeds is *skin friction*, which is due to the acceleration of the initially still fluid to the object speed u, because fluid in the boundary layer near the object sticks to it. This is different from Stokes drag, which is due to frictional losses in the fluid. This skin friction is

$$F_{\text{drag,skinfriction}} = \frac{1}{2} C_{\text{sf}} S \rho u^2, \tag{7.48}$$

where C_{sf} is the skin friction coefficient, which depends on the details of the flow, and S is the wetted surface area. When you swim at or near the surface, fluid builds up to a higher than ambient level in front of your head (as you push the water forward). The water is depressed to a level lower than ambient after your head, as it "ventilates." This *ventilation drag* force varies as u^4.

The power consumed by each of these drag forces is

$$P = F_{\text{drag}} u. \tag{7.49}$$

When a foil that is tilted up at an angle β moves in a fluid, an upward force is generated on it called *lift*, which is

$$F_{\text{lift}} = \frac{1}{2} C_{\text{lift}} S \rho u^2. \tag{7.50}$$

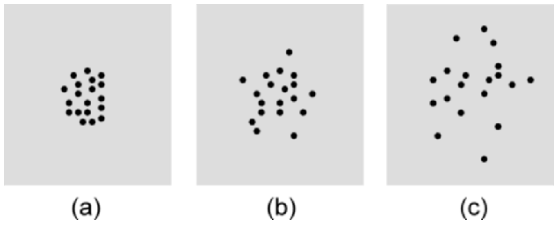

Fig. 7.17. Schematic of how the locations of particles vary at successively later times, from (**a**) to (**c**), as a result of diffusion

The lift coefficient, C_{lift}, varies linearly with this angle of attack. For small angles, it varies linearly from -0.4 to 1.2 for β varying from -4° to 12° (for $Re = 1.7 \times 10^6$). Of course, $C_{\text{lift}} = 0$ for $\beta = 0^\circ$. For β much larger than 12°, the flow separates from the upper edge of the wing and there is stalling of the lift.

Chapter 3 discussed the lift force on spinning objects, such as thrown baseballs and such, which is commonly called the Magnus force.

7.3 Diffusion (Advanced Topic)

When the concentration of particles (or molecules) is not uniform, the random particle thermal motion leads to a net movement (or diffusion) of particles from regions of higher concentration to regions of lower concentration. The net effect is to make the concentration more uniform (Fig. 7.17). This diffusion flow rate increases with the nonuniformity or gradient of the concentration, which is *Fick's First Law of Diffusion*

$$J = -D_{\text{diff}} \frac{\partial n}{\partial x} \tag{7.51}$$

for flow in one-dimension, where J is the flux of particles (particle flow per unit area per unit time), D_{diff} is the diffusion coefficient, and $n(x,t)$ is the concentration of particles. (We must use partial derivatives here because everything depends on x and t.) The diffusion coefficient depends on the background medium, and is on the order of $\sim 10^{-1}\,\text{cm}^2/\text{s}$ in gas, $\sim 10^{-5}\,\text{cm}^2/\text{s}$ in liquid, and $\sim 10^{-9}\,\text{cm}^2/\text{s}$ in solid backgrounds.

During this flow the total number of particles must be conserved. Consider the cylindrical volume construct in Fig. 7.18, with its axis along the x-axis, and of length $\mathrm{d}x$ and cross-sectional area A. The total number of particles entering from the left in a unit time $\mathrm{d}t$ is $J(x)A(\mathrm{d}t)$ and the number leaving from the right in this same time is $J(x + \mathrm{d}x)A(\mathrm{d}t) \simeq (J(x) + (\partial J/\partial x)\mathrm{d}x)\, A(\mathrm{d}t)$. Therefore the net increase in the number of particles in the cylinder is the difference $-(\partial J/\partial x)(\mathrm{d}x)A(\mathrm{d}t)$. This must be accounted for by the change in

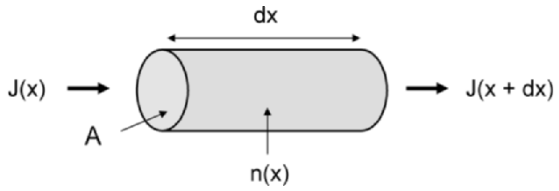

Fig. 7.18. Particles are conserved for any flow in and out of the cylinder through a change in concentration inside of it, for any flow process and for diffusion

density in this time in the volume, which is $[(\partial n/\partial t)\mathrm{d}t][A(\mathrm{d}x)]$. This gives the conservation of the number of particles

$$\frac{\partial n}{\partial t} = -\frac{\partial J}{\partial x}. \tag{7.52}$$

These two equations can be combined by differentiating (7.51) in space to get $\partial J/\partial x = -D_{\mathrm{diff}}\ \partial^2 n/\partial x^2$ (assuming that D_{diff} does not depend on x) and replacing $\partial J/\partial x$ from (7.52). This gives the *Diffusion equation* (or *Fick's Second Law of Diffusion*)

$$D_{\mathrm{diff}}\frac{\partial^2 n}{\partial x^2} = \frac{\partial n}{\partial t}. \tag{7.53}$$

Such diffusion leads to a slow gaussian-like, undirected spreading of the species over a distance $x \sim \sqrt{2D_{\mathrm{diff}}t}$ in a time t. (A gaussian profile is of the general form $\exp\left(-x^2/a^2\right)$.) For a total number of particles N initially at $x = 0$ at $t = 0$, the concentration is approximately

$$n(x,t) \sim \frac{N}{\sqrt{2\pi D_{\mathrm{diff}}t}}\exp\left(-x^2/2D_{\mathrm{diff}}t\right). \tag{7.54}$$

As presented, this solution is not valid for small times. The exact solution is slightly more complicated in other ways as well, but it is essentially the same result when the initial spread of particles is very small (Fig. 7.19) (see Appendix C). If the initial distribution is gaussian, (7.54) becomes

$$n(x,t) = \frac{N}{\sqrt{2\pi\sigma^2(t)}}\exp\left(-x^2/2\sigma^2(t)\right), \tag{7.55}$$

where

$$\sigma^2(t) = \sigma^2(0) + 2D_{\mathrm{diff}}t \tag{7.56}$$

and $\sigma(0)$ is the initial spread.

In three-dimensions, the spreading of particles by diffusion is described by

$$n(x,t) = \frac{N}{\left(2\pi\sigma^2(t)\right)^{3/2}}\exp\left(-r^2/2\sigma^2(t)\right), \tag{7.57}$$

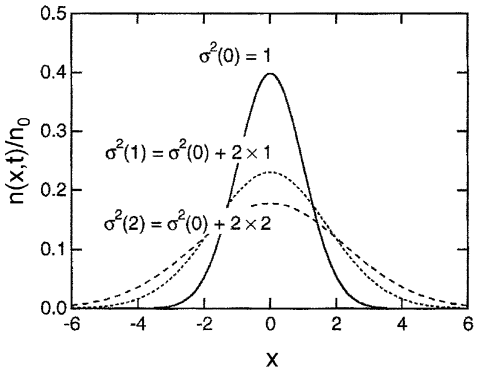

Fig. 7.19. Diffusion in one dimension, with gaussian spreading, with the initial distribution ($\sigma(0)$), one at time t during which σ^2 has tripled (in this particular example) ($\sigma(t)$), and one at time $2t$ ($\sigma(2t)$). (From [360])

with

$$\sigma^2(t) = \sigma^2(0) + 6D_{\text{diff}}t, \tag{7.58}$$

where $r^2 = x^2 + y^2 + z^2$.

Diffusion can be very important in the body over very small distances, on the order of 1–100 μm (∼100 μm for oxygen diffusion), but is not very useful over much longer distances. The amount of material that can be transported from one place to another is limited by the lack of directionality of diffusion (Problem 7.26). It leads to an increase in disorder, whereas a functioning organism requires careful control and regulation within characteristic time frames. (The level or disorder is known as *entropy*, which is discussed in more detail in discussions of thermodynamics, statistical mechanics, and in several areas of biophysics.) Smelling object depends on the diffusion of molecules to your nose (Problem 7.30).

Diffusion is also important in flowing systems. This is illustrated in Fig. 7.20 for an artery.

7.4 Pressure and Flow in the Body

Table 7.1 gives characteristic pressures in the body. The blood pressure ranges from ∼1–140 mmHg in different vessels and the speed of blood flow in these vessels ranges from ∼0.05–50 cm/s. The overall volumetric flow rate is ∼5 L/min. The relationship between pressure and flow in the circulatory system is detailed in Chap. 8. The characteristic pressure difference between the lungs and surrounding media is several mmHg and the volumetric flow rate of air into the lungs is ∼6 L/min; this is discussed further in Chap. 9.

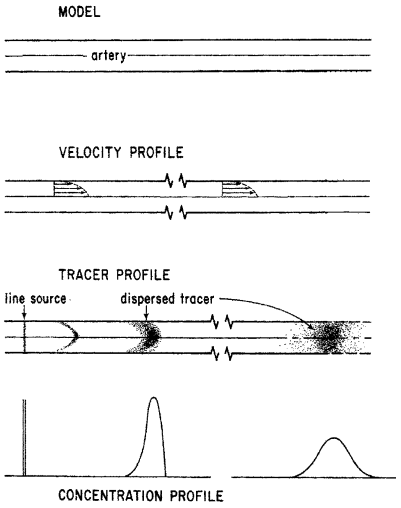

Fig. 7.20. Diffusion of an injected impulse, such as a dye, in an artery, with the shown line source initial distribution. The profile of the injection distorts as it adopts the velocity profile of the flow and it also diffuses. (From [353])

The flow rates in much of the human alimentary (digestive) system are quite slow (Table 7.4). Propulsive movements in this system are due to peristaltic action, with muscular contraction of the contractile ring around the gut sliding food forward, as diagrammed in Fig. 7.21. When there is a large amount of food in it, the gut stretches or distends and through sensors and feedback this stimulates a contractile ring 2–3 cm upstream. Mixing movements in the gut are caused by these peristaltic actions and by local constrictive contractions that occur every few cm in the gut and last for several seconds.

Table 7.4. Approximate flow rates and other properties of the human alimentary system, estimated for a 70 kg male. (Using data from [351])

component	length (cm)	external dimension or width (cm)	internal volume (cm^3)	luminal area (cm^2)	contents passage time	contents speed (cm/s)
mouth and pharynx	8	2–5	∼50	∼80	1–10 s	1–8
esophagus	25	1.3–2.5	∼100	∼200	5–20 s	3–5
stomach	12	8	230–1,000	∼600	2–6 h	∼0.001
small intestine	400	3–6	1,100	∼3,500	3–5 h	0.03
large intestine	∼150	5.0–7.5	300	∼2,000	10–20 h	0.004–0.008
rectum	16–20	2.5–3.8	40	∼100	∼1 h	0.006
total, average, or range	∼600	∼3.5	1,800–2,600	∼6,500	16–32 h	∼0.01

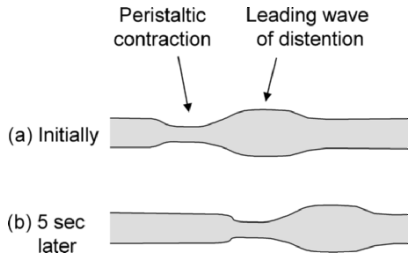

Fig. 7.21. Peristaltic action in the gut. (See Fig. 8.14 for peristaltic assistance in the return of venous blood to the heart). (Based on [356])

The relationship between volume and pressure is important in this digestive system. Pressure (tension) in the walls of the stomach increases during eating. The volume of the stomach of radius R increases as R^3. (This models the stomach as a sphere of volume V and ignores its finite radius with no food contents.) From (7.9), $\sigma = R(\Delta P)/2w = ((\Delta P)/2w)(3V/4\pi)^{1/3}$, so the tension in the stomach walls should increase, much slower, as R. Pressure in the stomach can also increase because of air swallowed during eating, which can lead to burping or belching. Bacterial action produces gas in the gut; at high enough pressure this causes flatulence.

As with the stomach, the pressure within the bladder increases slower than its volume, and this is seen in Fig. 7.22. The pressure rises to 5–10 cmH_2O when it is filled by 30–50 mL of urine. (The units of cmH_2O are commonly used in this area, with 1 cmH_2O = 0.738 mmHg.) Much additional urine can collect, 200–300 mL, with only a small rise in pressure. Above 300–400 mL the pressure increases rapidly. At ∼30 cmH_2O (3 kPa), there is an urge to urinate. Muscle contraction in the bladder (micturition reflexes) momentarily

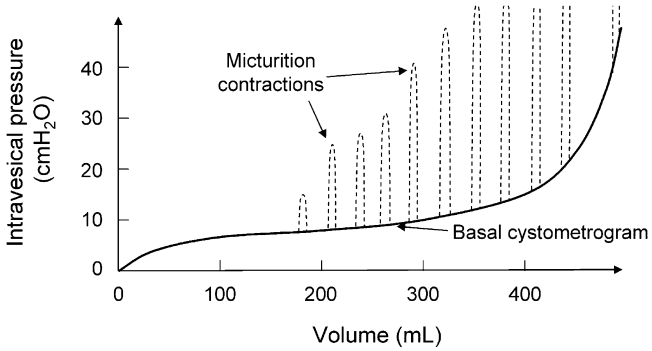

Fig. 7.22. Normal pressure–volume in the human urinary bladder (cystometrogram), also showing acute pressure waves (*dashed spikes*) caused by micturition reflexes. (Based on [356])

increases this pressure to 150 cmH_2O (15 kPa), with the normal voiding pressure being 20–40 cmH_2O (2–4 kPa). The wall tension increases with the volume of the bladder $V_{\text{bladder}}^{1/3}$, as seen from the Law of Laplace assuming a constant wall thickness. Therefore, the sensors to signal the urge to urinate would seem to be in the wall, sensing wall stress, and not sensing the pressure inside the bladder, because the pressure is fairly constant.

7.5 Motion of Humans in Fluids

We have already encountered several examples of humans in fluids. One is the loss of heat by thermal conduction and convection to the surrounding air in Chap. 6. Drag is also important in walking, running, cycling, and so forth, as is clear from how wind increases the metabolic needs during walking and running (Table 6.25). Locomotion in water, i.e., swimming, and potential human flight are examples in which the effects of the fluid are paramount [384].

7.5.1 Swimming

We are fairly buoyant, but not all can float. To float we must have an average density less than that of water (1.0 g/cm^3). (Equivalently, we must have a specific gravity (= density/water density) <1.) Those with relatively more fat (with an endomorph body shape) can float, with face, chest, and toes above the surface, because fat ($\simeq$0.8 g/cm^3) has a density lower than water. Those who are relatively muscular or big-boned (a mesomorph) cannot float because the densities of muscle ($\simeq$1.0 g/cm^3) and bone $\simeq$1.5–2.0 g/cm^3 are, respectively, roughly equal to and larger that of water. People with an average density a bit higher than that of water may be able to float after taking in a deep breath because of the low density of air (0.0012 g/cm^3). Most men and women will float after taking in a deep breath, but most men will sink with just residual air in their lungs (after an normal exhalation, see Chap. 9). Very young and very old people are more likely to float because they have more fat, less muscle mass, and (for old people) lower long bone density. (Measuring body density and fat percentage is described in Problem 1.40.)

When floating (or almost floating) people push water parallel to the surface, in the "backwards" direction, they are propelled forward by the reaction force (Newton's Third Law). In other words, they swim. Because the arm and leg strokes are periodic, the forward propulsion is really periodic in theory, much like the periodic nature of blood rhythmically pumped by the heart. The net forward acceleration of the swimmer is due to the sum of this forward reaction response of the backward pushing of water and drag. (We are ignoring other lift forces [343, 347, 359].) For a person swimming with speed u

$$m_{\text{b}} \frac{\text{d}u}{\text{d}t} = F_{\text{forward propulsion}} - F_{\text{drag}} \tag{7.59}$$

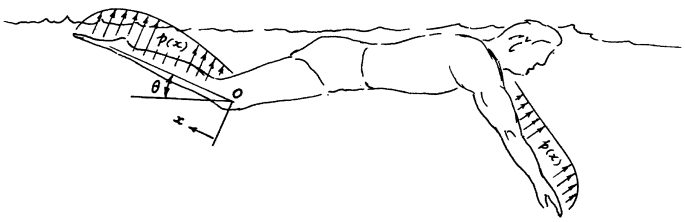

Fig. 7.23. Arm and leg motion during freestyle swimming (the crawl). Also see Problem 7.36. (From [353])

(More rigorously, $-F_{\text{drag}}$ is really $-\mid F_{\text{drag}} \mid$.) As is proved in Problem 7.39, the main source of drag is hydrodynamic, which scales as u^2, and not viscous Stokes-type drag, which scales as u; consequently, $F_{\text{drag,hydrodynamic}} = \frac{1}{2} C_D A \varrho_{\text{water}} u^2$ (7.46). Actually, there are three identifiable sources of drag that scale as u^2: that due to frontal resistance, eddy resistance (due to water not filling in the body's wake and forcing the body to drag along these eddies), and surface drag. The first two types are sometimes collectively called hydrodynamic drag. Assuming now that this propulsion is continuous (as opposed to cyclic), the left side of (7.59) is zero and the steady state speed is

$$u^2 = \frac{2F_{\text{forward propulsion}}}{C_D A \varrho_{\text{water}}}. \tag{7.60}$$

For freestyle swimming (which is technically called the "crawl," Fig. 7.23), the propulsion force during a stroke can be estimated as the momentum gained by the pushed water during the duration of the stroke T_{stroke}, divided by that stroke time. (Remember, $F = ma$ can be expressed in terms of the momentum $p = mv$, as $F = \mathrm{d}p/\mathrm{d}t$ or $\Delta p/\Delta t$.) The momentum of the water is the mass of water displaced, $\varrho_{\text{water}} V_{\text{water}}$, times the final water speed, v_{water} (relative to the swimmer), or $\varrho_{\text{water}} V_{\text{water}} v_{\text{water}}$. Therefore we find

$$F_{\text{forward propulsion, stroke}} \simeq \frac{\varrho_{\text{water}} V_{\text{water}} v_{\text{water}}}{T_{\text{stroke}}}. \tag{7.61}$$

Let us consider forward propulsion due to motion of the hands and arms only and ignore leg motion. Let us also assume that the swimmer's arm is straight during the stroke and rotates about the shoulder in a cylindrical sweeping motion with a radius of the arm length l_{arm} and a width equal to the hand width w_{hand} (which we will say is also roughly equal to the arm width, w_{arm}). Therefore, the volume of displaced water is $V_{\text{water}} = \pi l_{\text{arm}}^2 w_{\text{hand}}/2$. (The factor of two accounts for the half of the cylindrical volume that is in the water.) The speed of the end of the arm is roughly $l_{\text{arm}}/T_{\text{stroke}}$ and near the shoulder it approaches zero. Because the water is moved at the arm speed, the average speed of the water is $v_{\text{water}} \simeq l_{\text{arm}}/2T_{\text{stroke}}$. (Part of the water is pushed downward during an ideal circular motion of the arm and this

does not contribute to this forward propulsion; we will ignore this because the stroke motion is not really circular.) Therefore, (7.61) becomes

$$F_{\text{forward propulsion,stroke}} \simeq \frac{\varrho_{\text{water}} \pi l_{\text{arm}}^3 w_{\text{hand}}}{4T_{\text{stroke}}^2}. \tag{7.62}$$

We can estimate the arm length as the sum of the lengths of the upper and lower arms and half the length of the hand (because it is cupped), and so using Table 1.6 we see that $l_{\text{arm}} = 0.386H$, where H is the body height. We estimate that $w_{\text{hand}} = 0.07H$. Excellent swimmers make about 60 strokes a minute, so $T_{\text{stroke}} = 1\,\text{s}$. Using $H = 1.8$ m, we find that $F_{\text{forward propulsion,stroke}} \simeq 27\,\text{N}$. This is what we would expect for an effective force from a muscle with a cross-section of $1.3\,\text{cm}^2$ going into this motion, which seems a bit low. The steady state speed u is obtained from (7.60), using $C_{\text{D}} = 1.0$ and the transverse area $A \sim 0.076\,\text{m}^2$ (from the shoulder width, $0.259H$, times the chest depth, $0.09H$, using $H = 1.8\,\text{m}$). The average speed during a stroke is then $u \simeq 0.8\,\text{m/s}$.

The next stroke, with the other arm, starts when the previous one has stopped. The arm of this previous stroke "recovers" to the forward position above the water line and so it does not provide propulsion in reverse. Therefore, the forward propulsion is really continuous and this average speed seems reasonable. This speed of 0.7 m/s is not that different from typical swimming speeds and is not that far from the speeds of world-class freestyle swimmers. (The average speed for world-record men's freestyle swimming (in 2006) is $\sim$2 m/s, decreasing from 2.3 m/s for 50 m distances to 1.8 m/s for 400 m.) Drag may be less than estimated here – in particular C_{D} and A may be smaller – and more water is likely being pushed per stroke by good swimmers than we estimated here. Remember that we totally ignored propulsion by the kick of the feet and legs and any propulsion by the rest of the body. Also, our analysis has ignored the complication of the initial dive into the pool and of reversing directions at the ends of the pool, etc.

The stroke is not exactly as described here. Actually, the arm does not pull straight in any stroke (freestyle (crawl), butterfly, breaststroke, and backstroke); after starting straight, it bends midway through and then (except for the breaststroke) straightens again for the crawl. This suggests that good swimmers use their hands more like propellers than paddles and that this type of motion can make lift significant, which we have ignored here.

The allometric relation for the swimming speed u of aquatic animals is

$$u \simeq 0.5 m_{\text{b}}^{0.19}, \tag{7.63}$$

where u is in m/s and m_{b} is in kg. This suggests that a 70 kg aquatic animal, such as a common dolphin, would swim at about 1 m/s, which is not far off from typical human performance. (Bottle nose dolphins have the same mass, but swim several times faster.)

What happens if a swimmer stops stroking and just glides? With no forward propulsion, (7.46) and (7.59) combine to give

$$m_\mathrm{b}\frac{\mathrm{d}u}{\mathrm{d}t} = -\frac{1}{2}C_\mathrm{D}A\varrho_\mathrm{water}u^2. \tag{7.64}$$

Bringing the velocity terms to the left gives

$$\frac{\mathrm{d}u}{u^2} = -\frac{C_\mathrm{D}A\varrho_\mathrm{water}}{2m_\mathrm{b}}\mathrm{d}t \tag{7.65}$$

and integrating from the initial speed u_i at $t = 0$ to the speed at time t gives

$$-\frac{1}{u(t)} + \frac{1}{u_\mathrm{i}} = -\frac{C_\mathrm{D}A\varrho_\mathrm{water}}{2m_\mathrm{b}}t. \tag{7.66}$$

Therefore the swimmer's speed approaches zero as

$$u(t) = \frac{u_\mathrm{i}}{1 + \frac{C_\mathrm{D}A\varrho_\mathrm{water}u_\mathrm{i}}{2m_\mathrm{b}}t} \tag{7.67}$$

with a characteristic time of say $18m_\mathrm{b}/C_\mathrm{D}A\varrho_\mathrm{water}u_\mathrm{i}$, at which time $u = 0.1u_\mathrm{i}$ (see Appendix C).

Because $u = \mathrm{d}x/\mathrm{d}t$, we find

$$\mathrm{d}x = \frac{u_\mathrm{i}}{1 + \frac{C_\mathrm{D}A\varrho_\mathrm{water}u_\mathrm{i}}{2m_\mathrm{b}}t}\mathrm{d}t \tag{7.68}$$

Integrating from position $x = 0$ at $t = 0$ gives

$$x(t) = \frac{2m_\mathrm{b}}{C_\mathrm{D}A\varrho_\mathrm{water}}\ln\left(1 + \frac{C_\mathrm{D}A\varrho_\mathrm{water}u_\mathrm{i}}{2m_\mathrm{b}}t\right). \tag{7.69}$$

7.5.2 Human Flight

Why cannot we fly? (That is, why cannot we fly without the assistance of a jet or helicopter, or propulsion devices on our backs?) The answer is easy. We cannot generate enough vertical force to counter our weight to enable us to hover or fly. In principle, we could do this by pushing air down fast enough or by generating a vertical force by aerodynamic lift – which could be possible if we could propel ourselves forward fast enough.

What happens if we try to fly by pushing air down by flapping our arms up and down? The volume of air we could push down per arm flap is the arm area, which is length × width, times the distance pushed, which is approximately the arm length. This is roughly 0.7 m × 0.1 m × 0.7 m per arm or $\sim$0.1 m^3 for both arms. The mass density of air is 10^{-3} g/cm^3 = 1 kg/m^3, so the mass displaced per flap is $\sim$0.1 kg. If the ends of our arms attained a speed of

80 mph $\simeq$ 40 m/s (which is the speed of a fair major league fastball, and is clearly an overestimate), our average arm speed would be about 20 m/s. If we flapped our arms 3 times a second (which is also faster than expected), the change in momentum in the moved air per unit time would be $(0.1\,\mathrm{kg})(20\,\mathrm{m/s})(3/\mathrm{s}) = 6\,\mathrm{N}$ (assuming no air is moved when our arms return to their initial positions at the end of each flap). This is much less than the weight of a 70 kg person, which is 700 N. Let us say we wear lightweight wings that would increase the effective flapping area to $2\,\mathrm{m}^2$ ($1\,\mathrm{m}^2$ per wing) and the volume of the air we would move increases to $2\,\mathrm{m}^3$. We would then generate an upward force of 120 N from this downward draft, and so even with our wildly high estimates of wing speed and flapping rate, we could not even approach developing enough vertical force to counter gravity and fly (or at least hover). (Because water has a density that is 1,000× that of air, we can easily keep ourselves afloat by pushing water down, and this is also assisted by buoyancy.) Such hovering, by the reaction force to the down draft in air, is more difficult than flying because there is no upward lift. Perhaps we could flap and propel ourselves forward and develop some lift.

Could we at least "takeoff" after running fast with our artificial wings in place? Assume that a person accelerated to the world record speed of about 10 m/s and suddenly spread his or her $2\,\mathrm{m}^2$ area wings. Using (7.50) under optimal conditions, we find $F_{\mathrm{lift}} = \frac{1}{2}C_{\mathrm{lift}}S\rho u^2 = (0.5)(1.2)(2\,\mathrm{m}^2)(1\,\mathrm{kg/m}^3)(100\,\mathrm{m}^2/\mathrm{s}^2)$, or 120 N of lift, which is still not enough. (Of course, even if the lift were enough and the person became airborne, forward deceleration due to drag would lead to a landing (or a crash).)

Clearly, any combination of wing flapping, for forward and some upward propulsion, and wing gliding for lift will also not lead to flight. The old saying, "If man (or woman) were meant to fly, he (or she) would have wings." is not true, because we could not fly even if we had wings. Of course people can hang glide with artificial wings; such gliding involves lift, drag, wind, and gravity.

Human-powered flight has indeed been demonstrated in the bicycle-powered aircraft built by the Paul MacCready team and cycled/flown by Bryan Allen, a champion bicyclist. In this aircraft the pedaling pilot propelled the propeller at the rear of the craft, which is connected to the cycle by a series of gears. Consequently thrust is created in this craft, which was optimized for lift, with minimal drag and weight. The "Gossamer Condor" flew for 7 min, 2.7 s in a closed course, and then on June 12, 1979 the "Gossamer Albatross" (with 30 m wingspan and 30 kg mass without the pilot) flew the first completely human-powered flight across the English Channel. It covered 35.6 km in 2 h 49 min, and thereby won the Kremer Prize established in 1959. This world-class cyclist provided 125 W of mechanical power, flew very close to the surface to take advantage of the "ground effect" – which is a temperature inversion near the surface – and was completely exhausted at the end of the flight.

7.6 Summary

The Law of Laplace, the equation of continuity and Bernoulli's equation for nonviscous flow, and Poiseuille's Law of viscous flow can be used to model the flows of fluids in the body, such as blood and air – which are described in Chaps. 8 and 9, and the movement of the body in fluids, such as swimming and flight, which is described in this chapter. The physics of pressure in fluids and diffusion are also used in these models.

Problems

Basic Fluidics and Pressure

7.1. Your blood pressure is measured with a sphygmomanometer, however with your upper arm pointed upward instead of downward. If your blood pressure is really 120 mmHg/80 mmHg, approximately what pressure would be measured?

7.2. The water level in a 4 m wide and 20 m long pool rises 0.75 mm when a person enters it and floats. What is the mass of that person?

7.3. You want to measure the volume of your whole arm by sticking it in an upright, long cylindrical tube with internal diameter of 15 cm, which is partially filled with water. The water level rises by 12.7 cm when a 50 kg female makes this measurement? What are the mass, weight, and volume of her arm? (See Chap. 1.)

7.4. Who is more buoyant and consequently floats higher: a large-boned, heavy muscled person with little body fat or a small-boned, lightly muscled person with more body fat?

7.5. Will retaining water affect a person's ability to float?

7.6. A 50 kg woman has a density of 1.01 g/cm^3 after normal exhalation. Does she float? Will she float after she inhales 2 L of air?

7.7. A 70 kg man with a density of 1.03 g/cm^3 ages. He gains 5 kg of fat. Will he float?

7.8. Will a person with an ectomorph shape float?

7.9. Why can all people float in the Dead Sea? (It has a specific gravity of 1.2–1.3. We have been assuming floating in water with no salt. Ocean water has a density of 1.027 g/cm^3.)

7.10. Three 50 kg women are airborne in a balloon filled with He. What is the minimum diameter of the balloon? (What assumptions are you making about the mass of the basket in which they are riding and the balloon itself?)

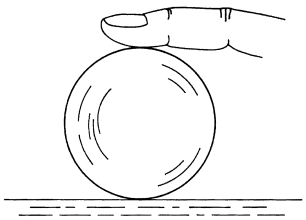

Fig. 7.24. Palpation of a blood vessel. (From [352].) For Problem 7.14

7.11. The gauge pressure inside a cylindrical tube is 100 mmHg and its radius is 1 mm, what is the tension in the tube wall at equilibrium (in SI units)?

7.12. (advanced problem) Derive the Law of Laplace for a sphere (7.9) by careful integration of the normal force on a hemisphere, in a manner analogous to the integration in (7.7) for a half-cylinder.

7.13. Over a large range of volumes, the pressure in the bladder is at a fairly constant value near 8 mmHg. If the thickness of the bladder is 5 mm, show that the wall tension is $\sigma = 600\,\text{Pa/cm}\ V_{\text{bladder}}^{1/3}$ where the bladder volume is in cm^3.

7.14. The internal pressure of an elastic vessel, such as an artery, vein, eyeball, aneurysm, or balloon, can be estimated by pushing down on it with your finger; this method is called *palpation* (Fig. 7.24):
(a) Show that the pressure felt by the finger is affected by the tension in the vessel wall.
(b) Show that the pressure you feel equals the pressure internal to the vessel when you push down on it so that the vessel wall is flat.

7.15. Assuming no viscosity and no changes in height, determine how the flow speed in a vessel changes if its diameter decreases by a factor of 4.

Viscous Flow

7.16. Compare the SI units of dynamic viscosity, η in (7.23), with those of the viscosity damping constant of the dashpot, c in (4.48).

7.17. One wants to use oil in car engines so the oil viscosity is a specific, optimized value – especially when the engine is started cold. Usually a heavyweight oil is used in very hot weather, such as SAE 50, and a lightweight oil in very cold weather, such as SAE 10. Using Table 7.3, estimate the viscosity needed at moderate temperature. Also estimate how the motor oil viscosity changes with temperature. (Nowadays, multiviscosity oils, such as SAE 10W/40 are used, which are suitable over a wide range of temperatures.)

7.18. Viscous flow with flow rate Q in a big tube of diameter D and length L, subdivides into N identical small tubes of length L with equal flow rates:
(a) What is the flow rate in each small tube?
(b) You are told that the pressure drops across the big tube and across the small tubes are the same (and both equal to ΔP). Find the diameter of the small tubes and determine if this is possible.
(c) If instead, the diameters and lengths of the small tubes are $\alpha\times$ and $\beta\times$ that of the big tube, what is the resistance across each small tube and across the whole small tube system in terms of the resistance across the big tube?

7.19. We are very sensitive to even small changes in core body temperature. Let us examine what happens when the viscosity of blood changes because of such temperature changes. It is known that the dynamic viscosity of whole blood decreases by 30% when temperature increases from 25°C to 37°C. What is the increase in systolic blood pressure, from its normal value of 120 mmHg, needed to pump blood throughout the body at the same rate if the core body temperature decreased to 25°C, with everything else being the same? (This temperature change will affect the body in many other ways even more dramatically; see Chap. 13.)

7.20. (a) How much force F (in N and lb) must be applied to a plunger to inject $1.0 \times 10^{-6}\,\mathrm{m}^3$ of the solution in 3.0 s with a hypodermic syringe? Apply Poiseuille's Law for the pressure drop across the needle, as in Fig. 7.25 [348]. The needle is injected into a vein with a (gauge) pressure of 14 mmHg (1,900 Pa). Assume the plunger has an area of $8.0 \times 10^{-5}\,\mathrm{m}^2$ and the syringe is filled with a solution with viscosity of 1.5×10^{-3} Pa-s. The needle has an internal radius of 4.0×10^{-4} m and a length of 0.025 m. Remember that you want to apply a (gauge) pressure in excess of the venous pressure to achieve the desired flow rate Q.
(b) Why are such injections performed intravenously and not intra-arterially?

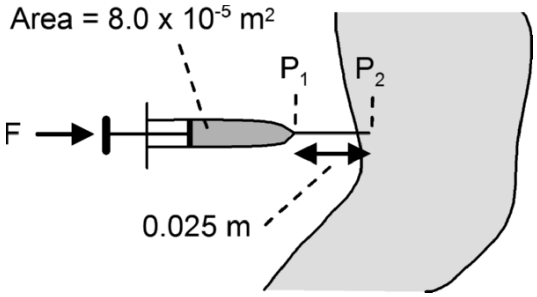

Fig. 7.25. Intravenous injection by a hypodermic syringe. (Based on [348].) For Problem 7.20

7.21. Calculate the Reynolds number for each component of the alimentary system. Assume the coefficient of viscosity is 1 N-m/s to the stomach and 10 N-m/s after the stomach. Is the flow streamline or turbulent?

7.22. (a) For Newtonian flow, calculate the shear stress on the wall of a tube of radius R, for an average fluid speed u and fluid viscosity η.
(b) Estimate this (in SI units) for a typical human artery.

7.23. (advanced problem) Show that the strain rate $\mathrm{d}v/\mathrm{d}y$ used in flow is related to the time rate of change of strain $\mathrm{d}\epsilon/\mathrm{d}t$. (Hint: Express strain as the partial derivative of a deformation u, $\epsilon = \partial u/\partial y$ and speed as $v = \partial u/\partial t$. Then evaluate $\partial\epsilon/\partial t$, and switch the order of the y and t derivatives.)

Diffusion

7.24. Important molecules are formed in the middle of a 2 μm-diameter cell. How long does it take for them to diffuse throughout the cell? (Assume the cell contents are liquids and that the diffusion coefficient $D_{\text{diff}} = 10^{-5}\,\text{cm}^2/\text{s}$.) Is this fast enough to achieve normal metabolic activity rates?

7.25. In one-dimension, estimate the characteristic distances for diffusion in 1 s in a gas, liquid, and solid.

7.26. A $1\,\text{mm}^3$ volume of biological material must be transported 2 cm away to another $1\,\text{mm}^3$ region:
(a) If it flows in a vessel at a speed of 10 cm/s, how fast does it get there and what fraction of it arrives there?
(b) If it diffuses in a liquid with $D_{\text{diff}} = 10^{-5}\,\text{cm}^2/\text{s}$, approximately when will the maximum amount of it arrive and approximately what fraction of it will arrive?
(c) Which mode of transport is preferred and why?

7.27. Refer to Fig. 7.19. If σ and the abscissa are in cm and t is in s, what is D_{diff}?

7.28. Use substitution to confirm that (7.55) and (7.56) are the solution to the one-dimensional diffusion equation, (7.53).

7.29. (advanced problem) Use substitution to confirm that (7.57) and (7.58) are the solution to the three-dimensional diffusion equation

$$D_{\text{diff}}\frac{1}{r^2}\frac{\partial}{\partial r}\left(r^2\frac{\partial n}{\partial r}\right) = \frac{\partial n}{\partial t}. \tag{7.70}$$

7.30. (a) You can detect 4×10^8 molecules of ethyl mercaptan (which causes the rotten fish smell) per cm^3, which corresponds to one molecule per 10^{11} molecules in air (because the air density is $5\times10^{19}/\,\text{cm}^3$). If $1\,\text{mm}^3$ of this

liquid is released 10 m away, how long will it take to notice this release? (Ethyl mercaptan, C_2H_5SH has 62.1 g/mole and is a liquid with a density of 1.01 g/cm^3. It has an odor resembling that of rotten eggs, and is added to natural gas and propane to give those normally odorless fuels a distinctive smell.)
(b) If your dog's nose is a thousand times more sensitive, when will she or he smell it?

Swimming, Flying, and Drag Forces

7.31. Repeat the analysis that determines the speed of a swimmer, but now assume that the effective force of 2 in diameter muscles is providing 405 N continuously. (Why is this force reasonable?) Does your answer make sense? Why?

7.32. Repeat the analysis of the speed of a swimmer, but now assume that Stokes friction is the only dominant drag force. Does your answer make sense? Why?

7.33. Go through all the steps in determining the position during gliding in swimming, from (7.68) to (7.69).

7.34. Using the parameters in the text, estimate the characteristic time needed for a world class freestyle swimmer who stops stroking and glides to slow down. Also estimate the distance she travels in that time.

7.35. Repeat the analysis of gliding, (7.64)–(7.69), assuming only Stokes drag.

7.36. In Fig. 7.23, the lower leg of a swimmer is hinged at the knee (at $x = 0$) and is acted on by forces that are normal to its axis with force per unit length of $p(x)$. Show that the work done by the leg, of length L, as is rotates by $\mathrm{d}\theta$ is [353]

$$\mathrm{d}W = \left(\int_0^L p(x) x \mathrm{d}x \right) \mathrm{d}\theta. \tag{7.71}$$

7.37. Could people fly on another planet using artificial wings? How would g and the mass density of the atmosphere ρ have to change? (Does a change in g imply the same or an oppositely signed change in ρ?) Ignore the impact of spacesuits, differences in temperature, changes in metabolism, muscle atrophy, and so on.

7.38. What is the Reynolds number of a piece of matter 1 μm in diameter, such as a cell in water or particulate in blood? Assume a density of 1 g/cm^3, a speed of 4 mm/s, and the viscosity of blood. What type of drag dominates?

7.39. (a) Is Stokes friction or hydrodynamic drag dominant for people walking and running in air?
(b) Which is dominant for people swimming in water?
(Make sure you calculate the Reynolds numbers in each case.)

7.40. (a) Estimate the hydrodynamic drag force on a very fast runner.
(b) How much power is lost to drag?
(c) How does this compare to the metabolic power needed for running?

7.41. Speed skaters often adopt a position with a nearly horizontal trunk and downhill skiers adopt the "egg" position with a hunched-down body and skis pointed backward when they are not maneuvering. Why?

7.42. A person without a parachute is dropped from a plane at an altitude of 1,000 m. Determine the "terminal" speed of the person by equating the forces of gravity and drag. (Which drag limit is appropriate? Is there enough time for a constant final speed to be attained?)

7.43. (a) A person with a parachute is dropped from a plane at an altitude of 1,000 m. Determine the final steady state speed of the person by equating the forces of gravity and drag. (Which drag limit is appropriate? Is there enough time for a constant final speed to be attained?) Assume the person has a mass of 70 kg and the parachute has a negligible mass and is 7 m across when it is open.
(b) What is the minimum height above ground that the parachute should be opened so the person lands with a speed no greater than 1.5 m/s? Assume it takes 2.5 s for the parachute to deploy fully.

8

Cardiovascular System

There are three components of the cardiovascular system. (a) Blood is the vehicle for transport. It transports fuel from the digested food to the cells, transports oxygen from the air in the lungs so it can combine with fuel to release energy, and it disposes of waste products – such as carbon dioxide from the fuel engine and other metabolic wastes. (b) The circulatory system is the distribution system, and consists of a series of branched blood vessels. (c) The heart is the four-chambered pump composed mostly of cardiac muscle that enables this circulatory flow. General descriptions of the cardiovascular system can be found in [368, 369, 372, 373, 376, 378, 384, 385, 388, 390, 395, 396, 402, 410, 417].

8.1 Overview of the Circulatory System and Cardiac Cycle

8.1.1 Circulation

Blood flow from the heart branches into two separate systems (Fig. 8.1). In the *pulmonary circulation* system, the right side of the heart pumps oxygen-poor ("blue") blood to the lungs to be oxygenated; oxygen-rich ("red") blood then returns to the left side of the heart. In the *systemic circulation* system, the left side of the heart pumps this oxygen-rich ("red") blood to the rest of the body where it is used; oxygen-poor ("blue") blood then returns to the right side of the heart. This occurs in a system of arteries that conducts the blood from the heart to the lungs and other organs and components, and a system of veins that returns the blood to the heart.

In the pulmonary system (Fig. 8.1), blood enters the *right atrium* (RA) of the heart (Fig. 8.2) through the *inferior and superior vena cava(e)* (vee'-na cae'vuh). The blood passes through the *right atrioventricular (or tricuspid) valve* to enter the *right ventricle* (RV). Blood is first pumped through the *pulmonary semilunar valve* to the pulmonary arteries, which branch out into a series of more minor arteries and arterioles, and then into capillaries in the

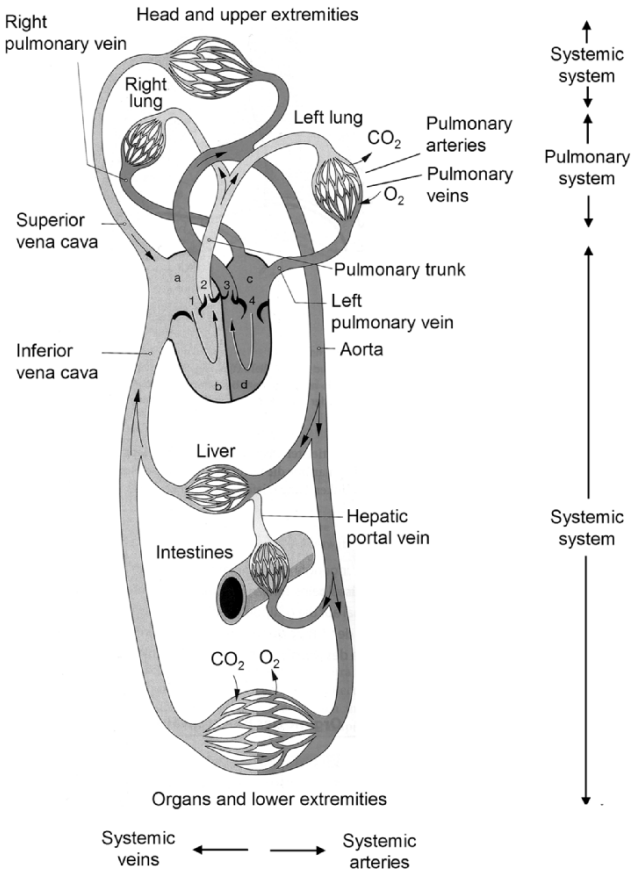

Fig. 8.1. Blood circulation system, and labeled within the heart: the (a) right atrium, (b) right ventricle, (c) left atrium, (d) left ventricle, (1) right atrioventricular (tricuspid) valve, (2) pulmonary semilunar valve, (3) aortic semilunar valve, (4) left atrioventricular (bicuspid, mitral) valve. (From [416])

lungs. These pulmonary capillaries combine into venules (veen'-yools), then into more major veins, and finally into the pulmonary veins.

In the systemic system (Fig. 8.1), blood enters the *left atrium* (LA) of the heart through the pulmonary veins. The blood passes through the *left atrioventricular (or bicuspid or mitral) valve* to enter the *left ventricle* (LV). Blood is pumped through the *aortic semilunar valve* to the *aorta*, which first branches out into a series of major and then minor arteries (with smaller diameters, the arterioles), and finally into a series of capillaries in the systems where gas exchange and diffusion occur. These systemic capillaries combine into venules, then more major veins, and finally into the superior (from above the heart) and inferior (from below the heart) vena cavae.

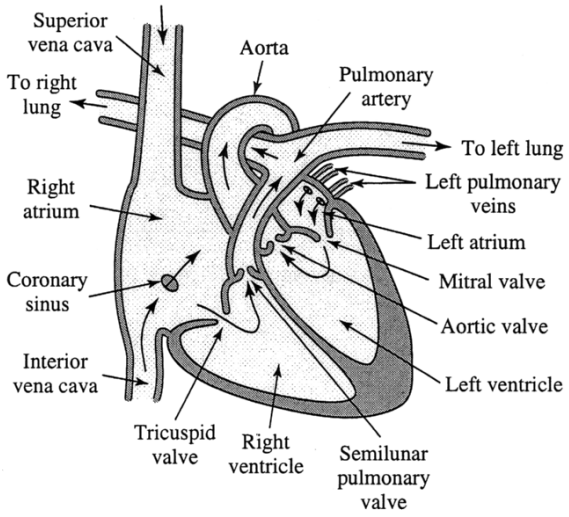

Fig. 8.2. Diagram of the heart, with its principle chambers, valves, and vessels. (From [367])

(A useful mnemonic for the flow of blood in the heart comes from knowing that the author once lived on Rahlves Drive in Castro Valley, California – a town approximately 20 miles south of Berkeley. The whole heart cycle starts with blood flowing into the right atrium (RA) and then getting oxygenated in the lungs, returning to the heart (H), and then continuing with the blood leaving the left ventricle (LV) and exiting (E) for the systems (S). Put together this spells RAHLVES. The most important concept here is that deoxygenated blood enters the heart through the right atrium (RA) and eventually oxygenated blood leaves through the left ventricle (LV) of the heart to be used by the body for metabolism. For some, it may be easier to remember that an American Daron Rahlves was the winner of the super-G downhill skiing competition in the 2001 World Championships.)

The systemic and pulmonary systems have similarities and differences. They have the same volumetric flow rate Q. (If they were not equal, blood would have to pile up somewhere.) In the systemic system the blood disposes of oxygen and receives carbon dioxide, while in the pulmonary system the blood disposes of carbon dioxide and receives oxygen. Table 8.1 shows that

Table 8.1. Normal resting values of blood pressure, with system volumes

	P (mmHg)	V (L)
systemic arteries	100	1.0
systemic veins	2	3.5
pulmonary arteries	15	0.1
pulmonary veins	5	0.4

the systemic system has higher pressures (in the arteries) and larger volumes than the pulmonary system (even with the same Q). This difference in pressure makes sense because the blood vessels need to be longer to get to more distant regions in the body in the systemic system. The left heart (LA + LV) is bigger (and is a larger pump) than the right heart (RA + RV) because of this need to generate higher pressure for systemic circulation. The heart walls consist mostly of the thick middle muscle layer, the *myocardium*, which is lined internally by a thin layer of tissue, the *endocardium*, and externally by a membrane, the *epicardium*. The two sides of the heart are separated by a wall called a *septum*. The difference in volume is due to the longer distance of travel and the much higher number of systems that receive blood in the systemic system. Table 8.1 also shows that arteries have higher pressure than the corresponding veins, whereas the veins have larger volumes. The total volume of blood is $\simeq 5\,\mathrm{L}$.

For a person at rest, 12% of the blood is in the heart chambers, 2% in the aorta, 8% in the arteries, 1% in the arterioles, 5% in the capillaries, 50% in the systemic veins, and 18% in the pulmonary circulation.

Major arteries and veins are shown in Figs. 8.3 and 8.4. Tables 8.2 and 8.3 provide a very approximate quantification of the vessels in the circulatory system.

8.1.2 Cardiac Cycle

There is a highly controlled timing cycle in well-functioning hearts, the cardiac cycle, which lasts a time τ (Fig. 8.5). In the first stage of *diastole* (die-as'-toe-lee), the veins fill up both the right and left atria, while the right and left ventricles are relaxed. In the second stage, the cardiac muscle (myocardium) of the right and left atria contract and pump blood through the atrioventricular valves, into the right and left ventricles, respectively, at the same time $t = 0$. (This is actually a gross simplification of ventricular filling, because $\sim$75% of this blood flows into the ventricles from the atria before atrial contraction.) In the first step of *systole* (sis'-toe-lee, which has the same cadence as Sicily), both ventricles contract (isovolumetrically) at the same time Δ. In the second stage, they eject blood through the respective semilunar valves: the right ventricle into the pulmonary arteries and the left ventricle into the aorta. The *systolic* (sis-stah'-lic) *blood pressure* occurs in this second stage of systole, while the *diastolic* (die-uh-stah'-lic) *pressure* is that during diastole. (One way to measure the flow of blood ejected by the left ventricle is *ballistocardiography*, which is described in Problem 8.47. Another method is Doppler ultrasonography echocardiography, which is used more often clinically; it is described in Problem 10.23.)

The right and left hearts must work at exactly the same time to keep the flow rate Q the same in both systems. There is a timing mechanism in place to do this and to set the contraction times 0 and Δ for one beat, followed by τ and $\tau + \Delta$ for the next, 2τ and $2\tau + \Delta$ for the next,

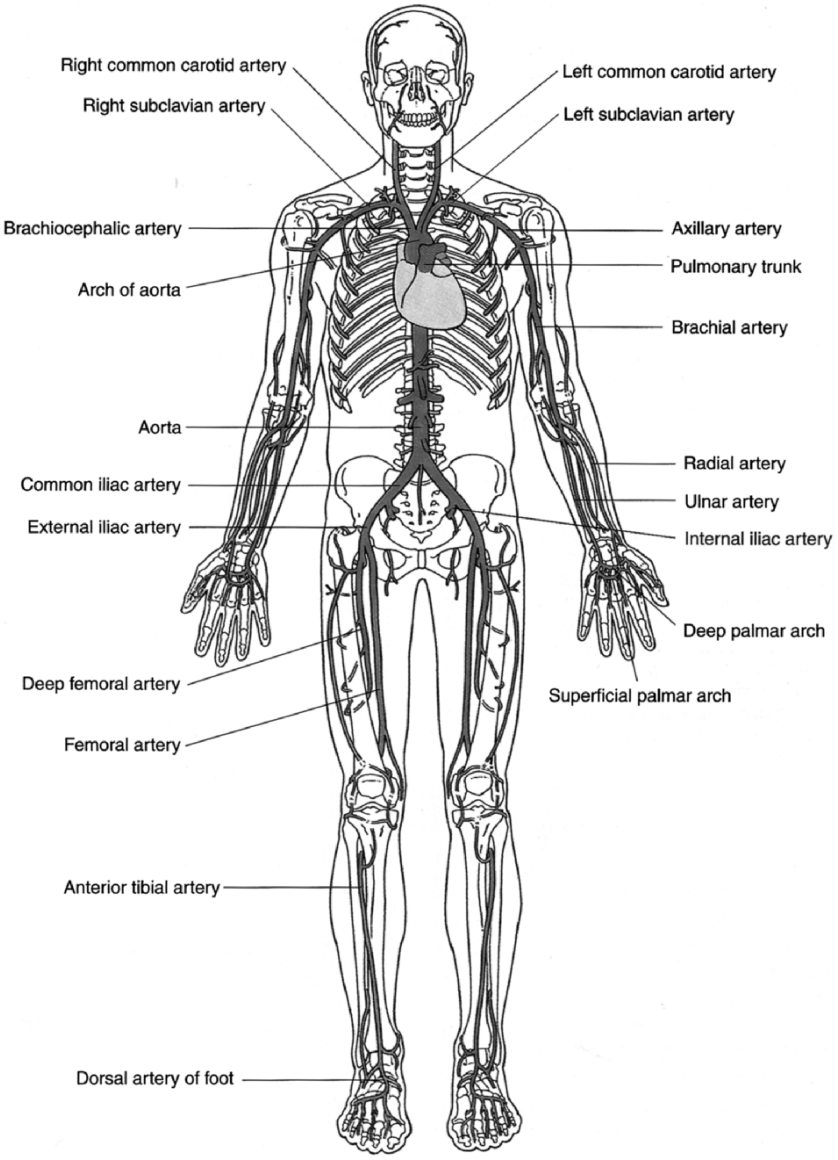

Fig. 8.3. Major arteries in the body. Arteries carry blood away from the heart in the systemic and pulmonary system. Many come in pairs, such as the right and left radial arteries. (From [408]. Used with permission)

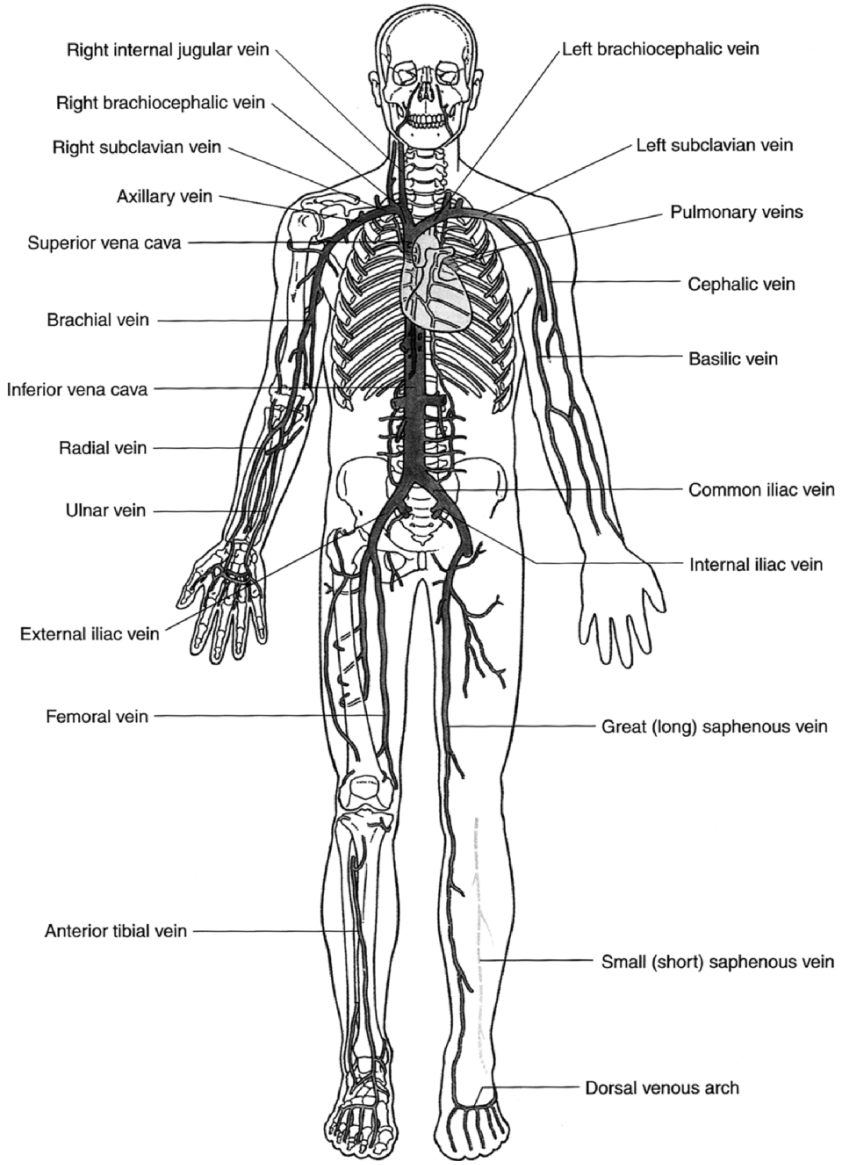

Fig. 8.4. Major veins in the body. Only the superficial veins are shown in the left limbs and only the deep veins are shown in the right limbs. Veins carry blood back to the heart in the systemic and pulmonary system. Many come in pairs, such as the right and left radial veins. (From [408]. Used with permission)

Table 8.2. Approximate quantification of individual vessels in the human circulatory system. (Using data from [382])

vessel	diameter (mm)	length (mm)	wall thickness (μm)	pressure (mmHg)
aorta	25.0	400	1,500	100
large arteries	6.5	200	1,000	100
main artery branches	2.4	100	800	95
terminal artery branches	1.2	10	125	90
arterioles	0.1	2	20	60
capillaries	0.008	1	1	30
venules	0.15	2	2	20
terminal venules	1.5	10	40	15
main venous branches	5.0	100	500	15
large veins	14.0	200	800	10
vena cava[a]	30.0	400	1,200	5
heart chambers	–	–	–	120

This is for a 30-yr-old male, with mass 70 kg and 5.4 L blood volume.
[a]There are really two vena cavae.

and so on. There is a heart pacemaker at the sinoatrial or sinus node (see the conducting system in Fig. 8.6), which sends an electrical signal to the atrial cardiac muscle of both atria for simultaneous atrial contraction. This electrical signal then travels to the atrioventricular or AV node,

Table 8.3. Approximate quantification of total vessel systems in the human circulatory system. (Using data from [382])

vessel	number	total length (mm)	total surface area (mm^2)	total blood volume (mm^3)
aorta	1	400	31,400	200,000
large arteries	40	8,000	163,000	260,000
main artery branches	500	50,000	377,000	220,000
terminal artery branches	11,000	110,000	415,000	120,000
arterioles	4,500,000	9,000,000	2,800,000	70,000
capillaries	19,000,000,000	19,000,000,000	298,000,000	375,000
venules	10,000,000	20,000,000	9,400,000	355,000
terminal venules	11,000	110,000	518,000	190,000
main venous branches	500	50,000	785,000	1,590,000
large veins	40	8,000	352,000	1,290,000
vena cava[a]	1[a]	400	37,700	280,000
heart chambers	–			450,000
Total		∼19,000 km	312,900,000	5,400,000

This is for a 30-yr-old male, with mass 70 kg and 5.4 L blood volume.
[a]There are really two vena cavae.

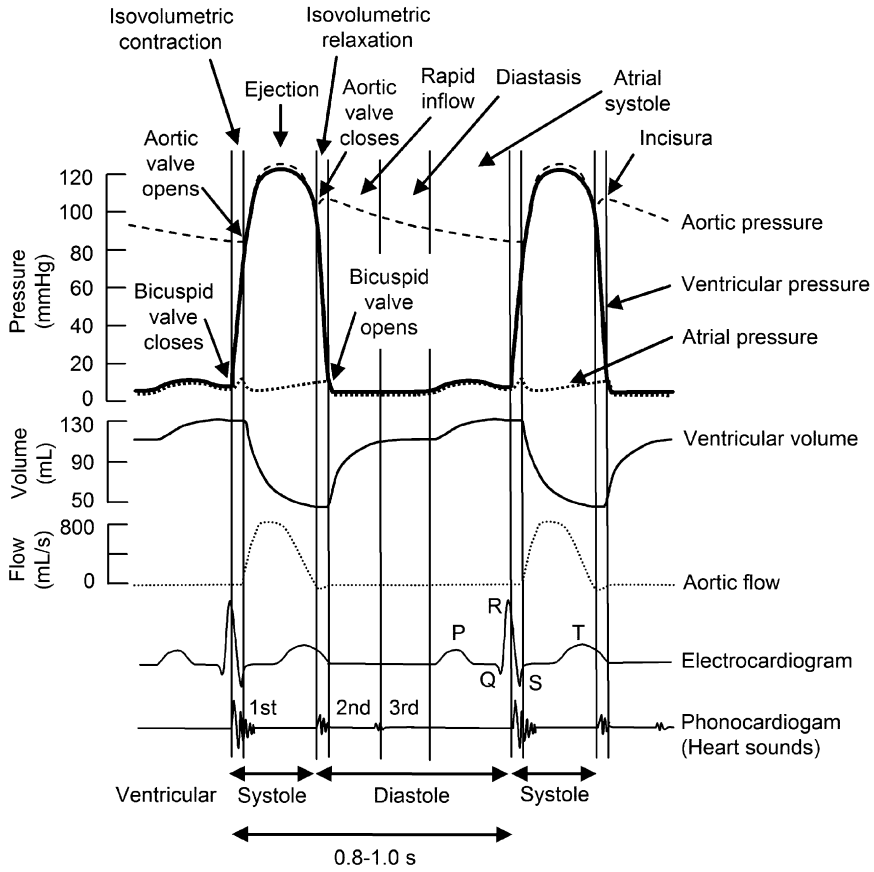

Fig. 8.5. The left ventricular and atrial pressures are plotted along with the left ventricular volume, aortic pressure and flow rate, the electrocardiogram and the phonocardiogram (which is the signal from heart sounds) in this Wiggers diagram. The opening and closing times of the aortic semilunar and bicuspid (mitral) valves are also shown. (Based on [390], [414], and [417])

is delayed there for a time Δ, and then the node sends a signal to the ventricular cardiac muscle of both ventricles for simultaneous ventricular contraction.

The electrocardiogram (EKG or ECG) is a measurement of these electrical signals, and their timing, as measured by probes on the body [379, 386, 401]. Figure 8.6 shows the EKG during one $\sim$1 s long heart beat (also see Fig. 12.28). The P wave is due to atrial depolarization (which is atrial contraction). The QRS complex is due to ventricular depolarization (contraction). The T wave is due to ventricular repolarization (relaxation). The atrial repolarization (relaxation) signal is masked by the larger QRS complex. Depolarization and

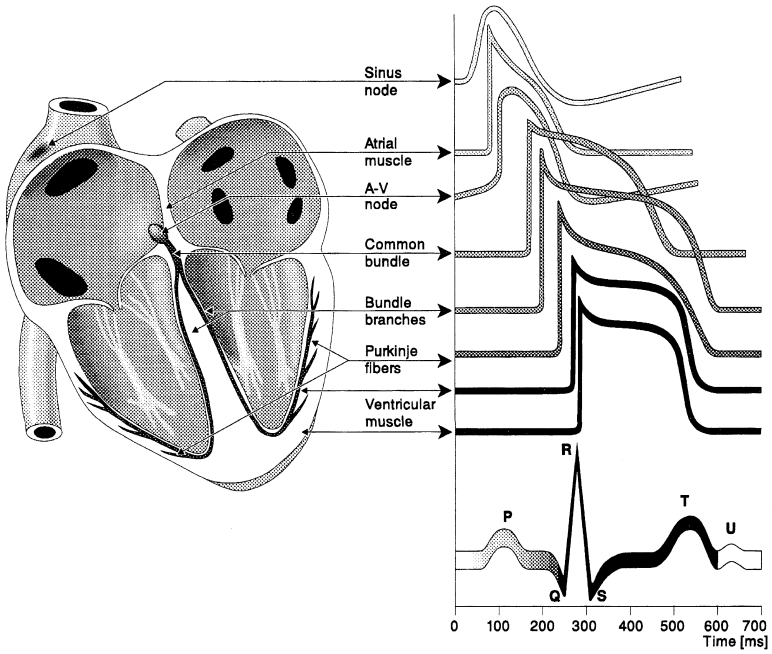

Fig. 8.6. Conducting system of heart with the sinoatrial/sinus and atriventricular/AV nodes, along with the electrical waveforms of the activity of each – including the EKG on the bottom. Also see Fig. 12.28. (From [404])

repolarization, and the electrical properties of the heart and the use of the EKG are discussed more in Chap. 12.

There are several cardiac arrhythmias (i.e., timing irregularities) of varying degrees of concern that affect this cardiac timing mechanism. In an *atrioventricular block*, there is injury to the atrioventricular (AV) fibers from the AV node to the ventricle or to the AV node itself (Fig. 8.6). In an incomplete AV block, the conduction time through the AV junction increases from the normal 0.16 s to 0.25–0.50 s and there are dropped ventricular beats; these sometimes lead to 2:1, 3:2, or 3:1 rhythms of atrial to ventricular beats. In a complete AV block, a person may faint until ventricular beats develop (with 40/min, compared to 100 beats/min in the atria). There can also be *premature contractions* of the atria or ventricles. In *paroxysmal tachycardia*, there are sudden increases in the heart rate, say from 95 to 150 beats/min in the atria or ventricles, which can cause serious ventricular (not atrial) damage. *Ventricular fibrillation* is the most serious arrhythmia and is fatal if not treated immediately. It can be caused by 60-cycle AC. There is uncoordinated muscle contraction of the ventricles, and so parts of them contract while other parts relax; this leads to little or no pumping of blood. Unconsciousness occurs in 4–5 s and the death of tissues begins in a few minutes. *Atrial fibrillation*

involves similar uncoordinated muscle contraction, but it is less serious because most blood flows passively from the atria to ventricles. Blood flow decreases by only ∼20–30%. Resuscitation after *cardiac arrest* can occur in many cases by cardiac pulmonary resuscitation (CPR) [390].

Cardiac muscle is similar to the skeletal muscle described in Chap. 5 (Fig. 5.1b). In particular, the basic building block is the sarcomere with its sliding actin and myosin filaments. There are some differences, however. At the resting muscle length, the maximum tension for skeletal muscle is $\sim 20\,\mathrm{N/cm^2}$ or more, while it is only $\sim 7\,\mathrm{N/cm^2}$ for cardiac muscle. Also, the resting, passive tension is fairly large at the length of peak tension in cardiac muscle (as is depicted in Fig. 5.25a). Both of these differences can be attributed in part to the greater fraction of noncontractile tissue in heart muscle, which contains collagen and other fibrotic tissue. The first difference also arises from the nonparallel nature of cardiac muscle fibers. Another difference between skeletal and cardiac muscle is that it is usually not possible to tetanize cardiac muscle. The twitches merge only partially at very high stimulation frequency.

Echocardiography is the use of ultrasound to diagnose heart disorders and blood flows. It and related methods are described briefly in Chap. 10 (and in Problems 10.22 and 10.23).

8.1.3 Valves

There are four major valves in the heart (Fig. 8.7). The right atrioventricular valve controls flow between the right atrium and right ventricle. It has three flaps (or cusps) and is therefore also called the tricuspid valve. The pulmonary semilunar valve controls blood flow from the right ventricle to the left and right pulmonary arteries. The left atrioventricular valve controls flow from the left atrium to the left ventricle. It has two flaps and is therefore also called the

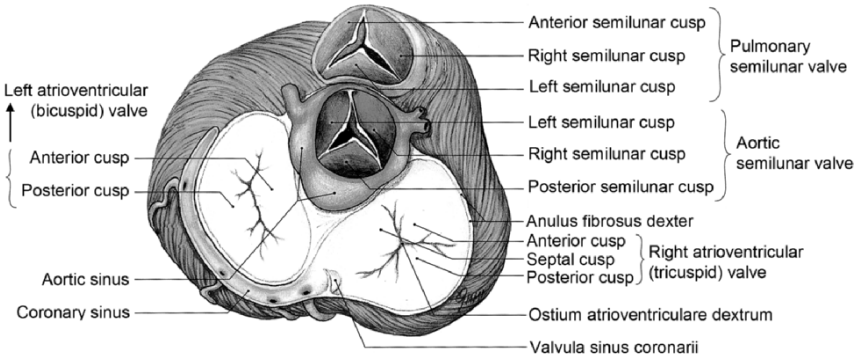

Fig. 8.7. Drawing of the four major heart valves, showing the cusps (flaps). (From [418])

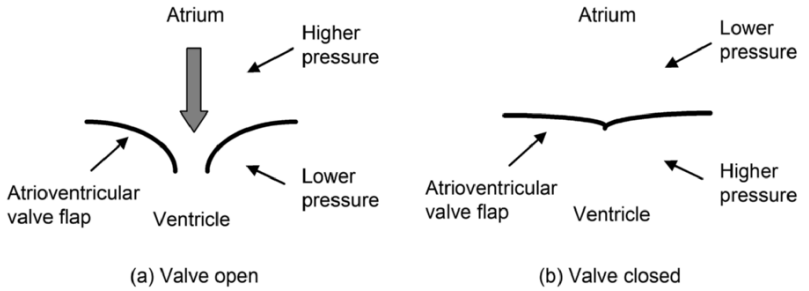

Fig. 8.8. Schematic of the unidirectional flow of an atrioventricular heart valve. (**a**) The pressure in the atrium exceeds that in the corresponding ventricle and the valve opens, with a jet of blood rushing in. Toward the end of diastole, the jet is broken. The deceleration of the blood creates a pressure, which tends to close the valve. (**b**) The valve is normally closed. (Based on [417])

bicuspid valve. Another name for this valve is the mitral valve, because it looks like a miter. The aortic semilunar valve controls flow from the left ventricle to the aorta.

These four valves share some common traits. They are one-way valves (Fig. 8.8) that allow blood flow in the described direction under some conditions, but never in the opposite direction (unless they are defective). We can imagine a flapped unidirectional valve that will not allow any back flow. With this type of valve we can see how the flaps will open, allowing this unidirectional flow, when the pressure in front of the valve exceeds that on the other side. In this way, the valve is closed until the pressure in the chamber increases due to contraction to a value greater than that after the valve. However, such a valve could not withstand very much back pressure. Backward opening of the atrioventricular valves is also prevented by the papillary muscles on the ventricular side that contract when the valve is closed, making the chordae tendineae that are attached to the flaps taut (Fig. 8.9). This prevents the flaps from bending backward, so there is no backward flow of blood.

Let us consider the cycle for the aortic semilunar valve. During ventricular relaxation the pressure in the left ventricle is ∼0 mmHg. In the aorta the pressure is ∼120 mmHg during systole and then decreases to ∼80 mmHg during diastole, just before ventricular contraction. The valve is still closed. During ventricular contraction the pressure in the LV increases to 80 mmHg, continuing up to ∼120 mmHg. Because the pressure in the aorta is ∼80 mmHg, the aortic semilunar valve opens once the pressure in the LV exceeds 80 mmHg and then remains open. During this flow, the pressure in the LV and aorta become equal, ∼120 mmHg, and then the valve closes as the flow cycle comes to an end.

The measurement of systolic and diastolic pressure by listening to Korotkoff sounds is described in Chap. 7. This method is the standard way

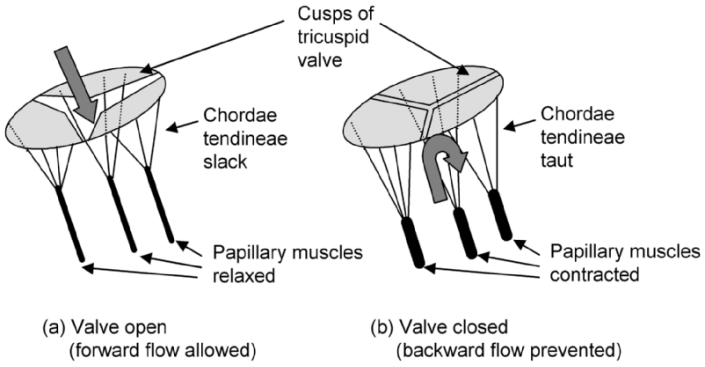

Fig. 8.9. The papillary muscles on the ventricular side of the atrioventricular valves contract when the values are closed, making the chordae tendineae taut, as in (**b**). The muscles and the chordae tendineae are relaxed when there is forward blood flow, as in (**a**). (Based on [417])

of determining blood pressure even though it routinely underestimates systolic pressure by 5–20 mmHg and overestimates diastolic pressure by 12–20 mmHg [377].

Cardiac valve openings and closings and the flow of blood in the heart create sounds that can be heard with a stethoscope, and are described in Chap. 10. (Also see Fig. 8.5.)

8.2 Physics of the Circulation System

We now examine the circulation system in more detail. First, we will examine how the blood pressure varies with distance along the arteries and veins, including within the capillaries. We then investigate the consequences of nonuniformities in arteries, such as clogged arteries and aneurysms – in the context of the strength of the artery walls. We next calculate the work done by the heart, to see how this contributes to the metabolic needs of the body. In the last section of this chapter we will develop a model of the entire circulatory system and the heart.

8.2.1 Properties of Blood

Blood is a non-Newtonian fluid, in part because of its complex, inhomogeneous composition. The blood solution consists of plasma, red blood cells (erythrocytes; 5 million/mm^3; 45% of total blood volume), white blood cells (leukocytes; 0.3%), and platelets (0.15%). (The red blood cell volume fraction is called the hematocrit.) The red blood cells are biconcave disks that are toroidal in shape with the center partially filled in, and have a diameter of

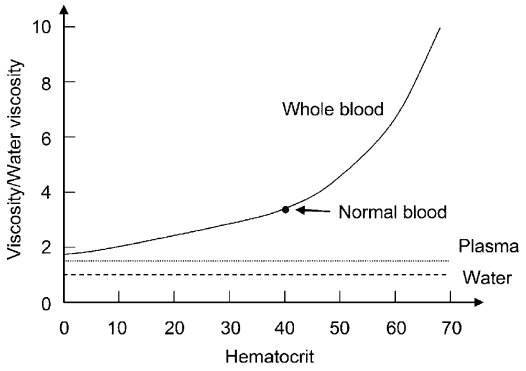

Fig. 8.10. Blood viscosity vs. hematocrit. (Based on [390])

7.5 μm and maximum thickness of 2 μm. Their diameter is about the same as the inner diameter of capillaries, but they can deform and flow in even smaller tubes. White blood cells are spherical, with a diameter of 7 μm, while the platelets are much smaller. The blood plasma is 90% water and behaves like a Newtonian fluid with a viscosity of 0.0012 Pa-s. The blood rheology is greatly altered by the red blood cells, and not much by the white blood cells or platelets because they comprise very small fractions of the blood volume. (Rheology is the study of the deformation and flow of materials, particularly unusual materials.) The blood viscosity increases with the hematocrit, as seen in Fig. 8.10.

The effective viscosity of blood decreases as the shear rate increases (Fig. 8.11). For very slow shear rates, this viscosity is more than 100× that of water, while at the high shear rates characteristic of flow in larger vessels it is about 4× that of water, with a value of 0.004–0.005 Pa-s.

The viscosity of some fluids changes even while the strain rate is constant. Blood is a thixotropic fluid, for which the shear stress decreases while the strain rate is constant. Still, for our purposes it will be adequate to treat blood as a Newtonian fluid, even though the velocity flow profile is not the ideal parabolic form for a Newtonian fluid (Fig. 7.13).

8.2.2 Blood Pressure and Flow in Vessels

Structure of Blood Vessels

Arteries contain inner layers that are 1–2 endothelial (lining) cells thick – along with elastic issue (composed of collagen and elastic proteins). This innermost region surrounding the opening – the lumen – is known as the tunica intima. Next in the wall comes a layer of circular, smooth muscle fibers interspersed with elastic tissue (the tunica media) and finally connective tissue (the tunica adventitia) (Fig. 8.12, also see Fig. 8.44). The walls of veins have a thickness

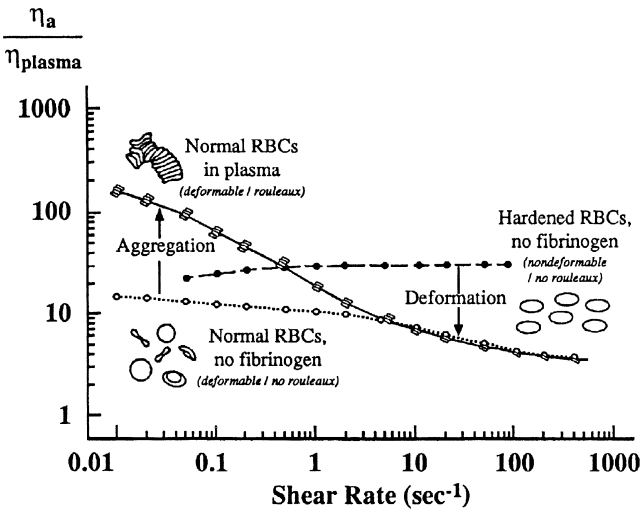

Fig. 8.11. Blood viscosity vs. shear rate for a hematocrit of 45%, at 310 K. (From [382]. Courtesy of Robert A. Freitas Jr., Nanomedicine, Vol. 1 (1999), http://www.nanomedicine.com, based on [375])

w that is typically $\sim d/10$, where d is the lumen diameter; they are thinner than the walls in arteries of corresponding diameters, for which $w \sim d/5$. The aorta and other large arteries contain much elastic tissue and stretch during systole and recoil during diastole. The walls of the arterioles contain

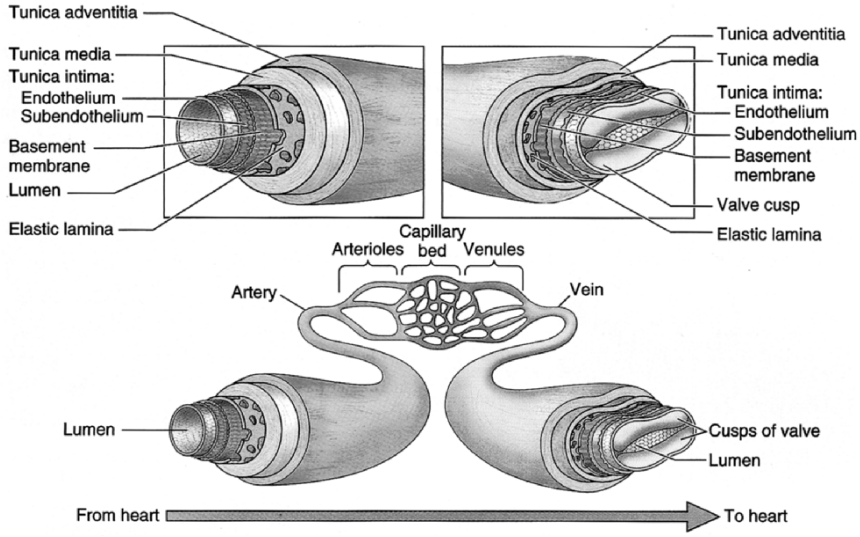

Fig. 8.12. Schematic of the walls of arteries and veins. (From [408]. Used with permission)

less elastic tissue and more smooth muscles, and stretch relatively little. The walls of the capillaries are composed of a single layer of endothelial cells. The diameters of the capillaries are so small that red blood cells can barely pass through them.

Approximately 70% of the walls of arteries and veins is composed of water, which is not elastic, except in how it withstands compression. The other 30% consists of the dry mass: elastin, collagen, and smooth muscle fibers, each having different materials properties. As discussed in Chap. 4, elastin is rubber-like and has a Young's modulus of $\sim 3 \times 10^5$ Pa; it can be stretched to twice its relaxed length. Collagen is much stiffer, with a Young's modulus of $\sim 1 \times 10^8$ Pa. Elastin has an ultimate tensile stress (UTS) less than 5% of that of collagen. Smooth muscle has a Young's modulus more like that of elastin, with $Y \sim 1 \times 10^5$ Pa when relaxed and $\sim 2 \times 10^6$ Pa when active. About half the dry mass in vessels is elastin and collagen, with more elastin than collagen in the aorta ($\sim 1.5\times$) and relatively less elastin in other arteries ($\sim 0.5\times$) and veins ($\sim 0.3\times$). Veins contain less elastin than arteries. The fraction of smooth muscle in the dry mass averages to $\sim 50\%$, and is $\sim 25\%$ in the aorta, and increases to $\sim 60\%$ more peripherally in the arteries and arterioles. The mechanical properties of these vessels (Chap. 4) also depend on the tissue to which they are attached. This is particularly significant for capillaries, because the vessel walls are essentially a single layer of endothelial cells.

Blood Pressure

Blood pressure is needed to push blood flow. Figure 8.13 is a schematic of the mean arterial and venous blood pressure at different positions in the

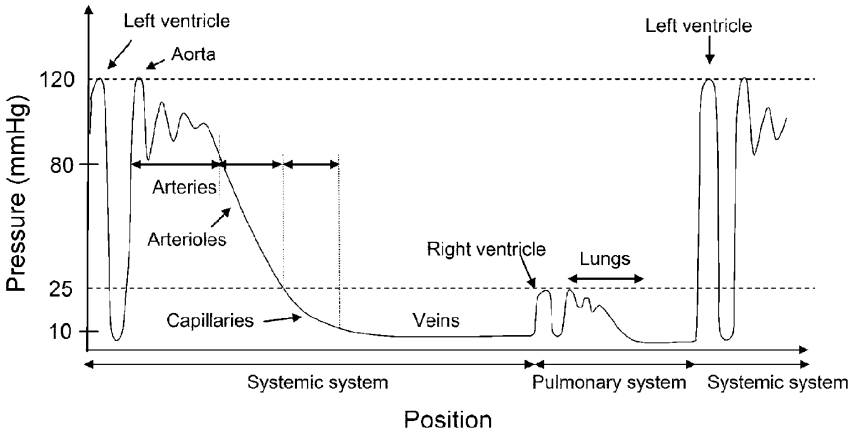

Fig. 8.13. Blood pressure along the circulatory system for a person lying horizontally. (Based on [371])

circulation cycle, for a person lying horizontally. The oscillations in blood pressure along the major arteries in systemic circulation reflect the oscillating pressure of this blood when it was leaving the aorta, at pressures between ∼80 mmHg (P_{diastole}, at diastole) and ∼120 mmHg (P_{systole}, at systole) (Fig. 8.13). Because systole lasts for about 1/3 of the cycle and diastole for about 2/3, the *mean blood pressure* is a weighted sum,

$$P_{\text{mean}} = \frac{P_{\text{systole}} + 2P_{\text{diastole}}}{3}, \tag{8.1}$$

or $(1/3)120\,\text{mmHg} + (2/3)80\,\text{mmHg} \sim 94\,\text{mmHg}$ in this example. This difference in pressure of 40 mmHg between systole and diastole is the *arterial pulse pressure* P_{pulse}. We will see that blood flows at a speed of ∼20 cm/s in these systemic arteries, so with a heart rate of about 1 Hz = 1 cycle/s it is reasonable that there are quasiperiodic variations every 20 cm or so. Much of the pressure drop in the arterial system is in the arterioles (small arteries) and the capillaries. We will see that this can be attributed to viscous flow. There is very low pressure in the veins. It is too little pressure to pump the blood back to the heart – even with the large diameters of the veins and consequently low resistance to flow (7.24). There is a peristaltic pumping mechanism by muscles surrounding the large veins that assists the return of venous blood to the heart, with one-way valves to prevent backflow (Fig. 8.14). (Similarly, blood flow in the capillaries is usually not continuous, but is turned on and off every few seconds or minutes, due to sphincter muscles that can contract the feeding arterioles (*vasomotion*).) The pulmonary system mirrors this systemic circulation, except the pressures are all lower. Figures 8.13, 8.15, and 8.16 show the blood pressure and flow speed at different points in the arterial tree.

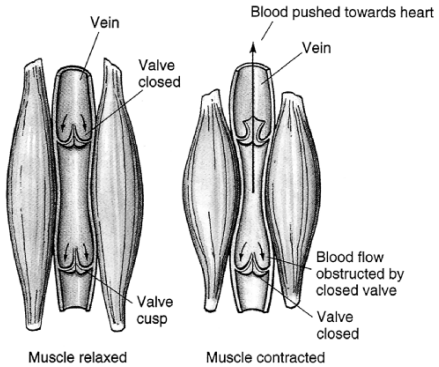

Fig. 8.14. Musculovenous pump of veins, with outward expansion of the bellies of contracting muscles pumping the blood back to the heart against gravity and distal valves closing to prevent backflow. (From [408]. Used with permission)

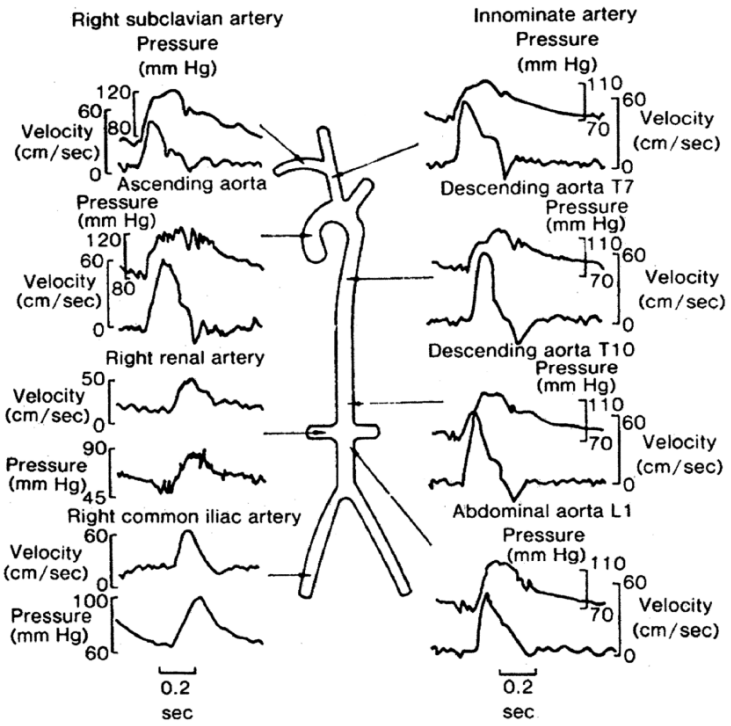

Fig. 8.15. Simultaneous pressure and flow velocity at different points in the human arterial tree for a person lying horizontally. All data were taken from one patient except for the right renal artery and the right common iliac artery. (From [391]. Adapted from [407])

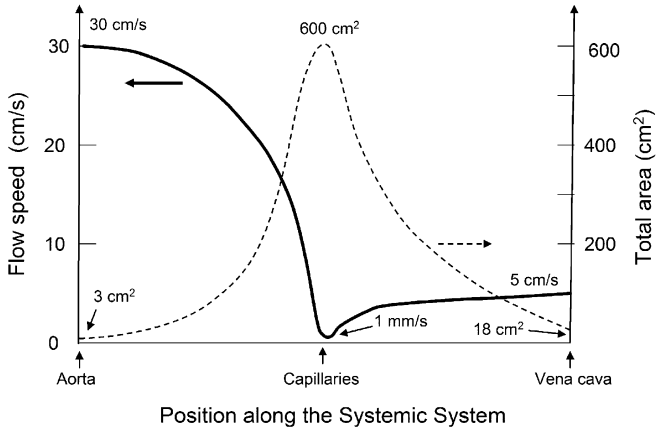

Fig. 8.16. Flow speed (*solid curve*) and total area (*dashed curve*) in the systemic circulation system. (Based on [371])

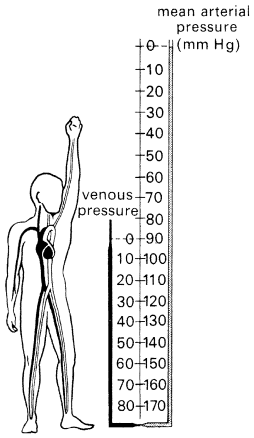

Fig. 8.17. Mean arterial and venous (gauge) pressures for a vertical person. (From [372], after [412]. Used with permission of Oxford University Press)

The pumping cycle sets up a pressure pulse wave in addition to the hydrostatic pressure variation. This pulse wave is independent of the speed of blood flow – and is faster than this blood flow speed: 4 m/s in the aorta, 8 m/s in the large arteries, and 16 m/s in the small arteries of young adults.

When you stand upright vertically (Fig. 8.17) there is an additional pressure $\rho g h$, where h is the height relative to the heart. This is approximately the height in the upper arm where blood pressure measurements are made. For $\rho = 1.06\,\mathrm{g/cm^3} = 1{,}060\,\mathrm{kg/m^3}$, $g = 9.8\,\mathrm{m/s^2}$ and $h = 1\,\mathrm{m}$, this pressure is $10{,}400\,\mathrm{N/m^2} = 10{,}400\,\mathrm{Pa} = 79\,\mathrm{mmHg}$ (with $1\,\mathrm{MPa} = 7{,}600\,\mathrm{mmHg}$). At any given height, the driving pressure difference from the arteries to the veins is unchanged. Also, this pressure change is not important when considering pressure changes between the inside and outside of a vessel because $\rho g h$ is added both inside and outside the vessel.

Still, this effect of gravity can be significant. The blood pressure at the aorta has to be high enough to pump the blood to the top of your brain. This distance is about $h = 40\,\mathrm{cm}$, so the pressure drop is about 30 mmHg (compared to the diastolic pressure of ∼80 mmHg). Problems 8.1 and 8.2 explore what happens to cranial blood circulation in humans in rapidly climbing jets and on more massive planets with higher g, and also in giraffes. One manifestation of this effect of gravity is potential fainting when you stand. When you stand up, the volume of blood in the leg veins increases and the pressure in the veins pumping blood back to the heart decreases. This can decrease the cardiac output and the flow of blood to the brain. This rarely happens because there is a reflex constriction of the veins in the legs (due to a contraction of the skeletal muscle surrounding the veins, Fig. 8.14) that limits the blood pool and an arteriolar constriction that increases flow resistance and lessens the decrease in arterial blood pressure. The effect of gravity on humans – who

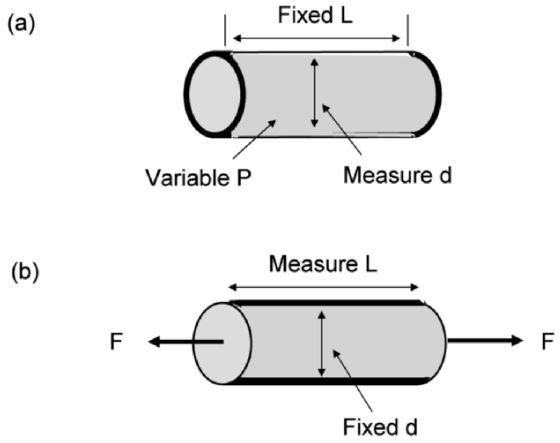

Fig. 8.18. Measuring the mechanical properties of blood vessels by fixing the vessel (**a**) length (which is called *inflation*) or (**b**) diameter (which is called *extension*). Some investigators do combined loading, with combinations of inflation, extension, and torsion

normally stand upright – also explains why standing on your head for long periods is not advisable (Problem 8.3). The veins in your head are not designed to pump blood back to the heart (as are those in the lower body). Also, your feet would stop getting blood. It also explains why varicose veins are worse when you stand upright, because blood then needs to be pumped up.

Body control of blood pressure is briefly described in Chap. 13.

Measuring Flow in Blood Vessels

The mechanical properties of blood vessels can be measured under two types of conditions (1) The length of a given vessel can be kept constant, while its diameter is measured as a function of the *distending pressure*. This leads to a tensile stress on the wall, directed around the circumference, which is called the circumferential or hoop stress. (2) The diameter of a vessel can be kept constant, while its length is measured as it is stretched longitudinally. Examples of both are shown in Fig. 8.18.

The flow of blood in arteries is affected by changes in the heart beat rate and stroke volume (which is the volume pumped per beat), and also by changes in the arteries themselves that control their diameters by chemical and neural mechanisms.

Modeling Flow in Blood Vessels

Figure 8.19 is a schematic describing the flow in a vessel of length L, with a volumetric flow rate Q_1 in and Q_2 out. In steady state $Q_1 = Q_2 = Q$. At the

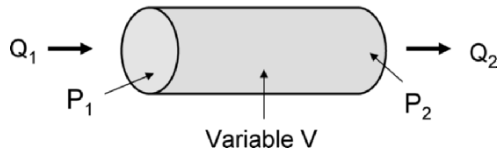

Fig. 8.19. Blood flow: general vessel

beginning of the vessel the pressure is P_1 and at the end it is P_2. The pressure outside the vessel is P_{ext}, which can be taken to be 0 when considering gauge pressure. The volume of the vessel is V. The relation between these various parameters depends on the properties of the vessels.

There are two physical attributes of blood vessels. (a) They have a *resistance* to flow, and so they need a pressure difference along the length of the vessel to drive the blood flow. (b) They have a *compliance* in response to a distending pressure. This is much like a balloon expanding when the pressure inside increases much above that outside.

One special case is a rigid vessel with constant volume V, which is called a *resistance vessel* (Fig. 8.20). Equation (7.25) applies to this vessel, so

$$P_1 - P_2 = R_{\text{flow}} Q \tag{8.2}$$

or

$$Q = \frac{1}{R_{\text{flow}}}(P_1 - P_2), \tag{8.3}$$

where the vascular resistance is $R_{\text{flow}} = 8\eta L/\pi r^4$ for a tube with radius r. The former equation has the same form as Ohm's Law $V_{\text{elect},1} - V_{\text{elect},2} = R_{\text{elect}} I$, which relates the drop in voltage, V_{elect}, when a current of charges I traverses a structure with electrical resistance R_{elect}.

A second special case is an elastic vessel that has no noticeable resistance, which is called a *compliance vessel*. There is no pressure drop, so $P_1 = P_2 = P$. One model (Fig. 8.21) of the properties of such a vessel is

$$V(P) = C_{\text{flow}}(P - P_{\text{ext}}) = C_{\text{flow}} P, \tag{8.4}$$

where C_{flow} is the compliance and P_{ext} is taken to be 0. Because the vessel usually has a volume with no pressure, called the dead volume V_{d}, a better

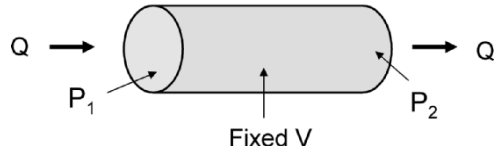

Fig. 8.20. Blood flow: ideal resistance vessel

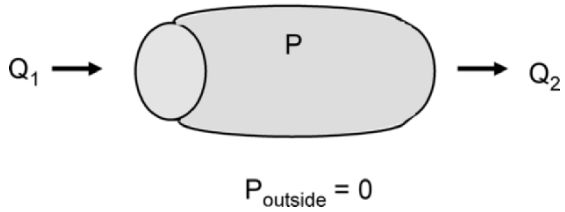

Fig. 8.21. Blood flow: ideal compliance vessel

relation is

$$V(P) = V_{\mathrm{d}} + C_{\mathrm{flow}} P. \tag{8.5}$$

Note that a property of the resistance vessel, namely the flow rate, is affected by the pressure drop along (and inside the vessel), while a property of the compliance vessel, its volume, is affected by the pressure difference between the inside and outside of the vessel. We can also describe compliance by changes in the radius r

$$r(P) = r_{\mathrm{d}} + \frac{C'_{\mathrm{flow}}}{2} P, \tag{8.6}$$

where r_{d} is the radius with no pressure difference. Therefore

$$\frac{\mathrm{d}r}{\mathrm{d}P} = \frac{C'_{\mathrm{flow}}}{2}. \tag{8.7}$$

Because $V = \pi r^2$ and $V_{\mathrm{d}} = \pi r_{\mathrm{d}}^2$, these two formulations can be interrelated.

Real vessels have some attributes of both types of vessels. Still, the aorta, large arteries and large veins are much like compliance vessels. We will see that the pressure drops along them are relatively small. Arterioles, capillaries, and venules act like resistance vessels. We will see that they, and in particular the arterioles, are the main sites of the pressure drop, and this is the reason why the heart needs to pump blood to such high pressures.

Pressure Drops in Arteries and Resistive Vessels

We will use Poiseuille's Law $\Delta P = \left(8\eta L/\pi r^4\right) Q$ (7.25) to estimate the pressure drop $\Delta P = P_1 - P_2$ across the aorta, large arteries, arterioles, and capillaries, and compare these results to the plot in Fig. 8.13 (also see Fig. 8.15). First we calculate the resistance $R_{\mathrm{flow},0} = 8\eta L_0/\pi r_0^4$ for a standard radius $r_0 = 1\,\mathrm{cm}$ and standard length $L_0 = 1\,\mathrm{cm}$ and scale the results for each specific case. With the viscosity $\eta = 4.0 \times 10^{-3}\,\mathrm{Pa\text{-}s} = 4.0 \times 10^{-3}\,(\mathrm{N\text{-}s/m^2}) = 4.0 \times 10^{-2}$

poise for whole blood at 37°C

$$R_{\text{flow},0} = \frac{8 \times (4.0 \times 10^{-3}\,\text{N-s/m}^2)(1\,\text{cm})}{\pi(1\,\text{cm})^4} = \frac{1.02 \times 10^{-2}\,\text{N/m}^2}{\text{cm}^3/\text{s}} \tag{8.8}$$

$$= \frac{1.02 \times 10^{-8}\,\text{N/mm}^2}{\text{cm}^3/\text{s}} = \frac{7.7 \times 10^{-5}\,\text{mmHg}}{\text{cm}^3/\text{s}}, \tag{8.9}$$

where we have used $0.1\,\text{N/mm}^2 = 1\,\text{atm.} = 760\,\text{mmHg}$. The units in (8.9) are mmHg-s/cm^3. This is the resistance when the pressure difference is 1 mmHg and the flow rate is 1 mL/s, and is also known as a PRU, a peripheral resistance unit. This unit is commonly used in physiology.

The resistance of a vessel of an arbitrary length and radius is

$$R_{\text{flow}} = R_{\text{flow},0}\frac{L/L_0}{(r/r_0)^4} = R_{\text{flow},0}\frac{L(\text{in cm})}{r(\text{in cm})^4} \tag{8.10}$$

and so

$$\Delta P = R_{\text{flow}}Q = 7.7 \times 10^{-5}\,\text{mmHg}\,\frac{L(\text{in cm})}{r(\text{in cm})^4}\,Q\ (\text{in cm}^3/\text{s}). \tag{8.11}$$

The total flow rate, Q_t, from the aorta, enters the large arteries, and the whole flow from the large arteries enters the arterioles, and finally this whole flow enters the capillaries. In each level of flow we will model the arteries as n parallel vessels of roughly equivalent length and diameter carrying roughly the same flow (Fig. 8.22), where n increases for each successive level of flow. So in a given level of flow with n vessels, the flow rate in each vessel is $\sim Q_\text{t}/n$.

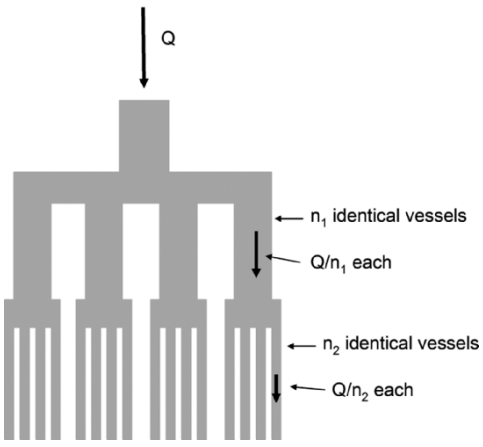

Fig. 8.22. Schematic of blood flow in idealized branching vessels

With $Q_t = 80\,\text{cm}^3/\text{s}$ and $Q = Q_t/n$, we get

$$\Delta P = 7.7 \times 10^{-5}\,\text{mmHg}\ \frac{L(\text{in cm})}{r(\text{in cm})^4}\ \frac{80}{n} \tag{8.12}$$

$$= \frac{0.0062\,\text{mmHg}}{n}\ \frac{L(\text{in cm})}{r(\text{in cm})^4}. \tag{8.13}$$

This is the pressure drop across any vessel in a given level of flow and, because they are in parallel, it is the pressure drop across the entire given level of arterial flow. We now determine this for the various levels of arteries.

Aorta. There is one aorta ($n = 1$) with $r \sim 1.25\,\text{cm}$ and $L \sim 10\,\text{cm}$, and so ΔP across the aorta is 0.025 mmHg, which is insignificant.

Largest arteries. There are about 200 large arteries with $r \sim 0.2\,\text{cm}$ and $L \sim 75\,\text{cm}$, and so ΔP is 1.4 mmHg, which is pretty insignificant.

Smallest arteries and arterioles. There are about 5×10^5 arterioles with $r \sim 30\,\mu\text{m}$ and $L \sim 0.6\,\text{cm} = 6\,\text{mm}$, and so ΔP is 91 mmHg, which is very significant.

Capillaries. There are about 10^{10} capillaries with $r \sim 3.5\,\mu\text{m}$ and $L \sim 0.2\,\text{cm} = 2\,\text{mm}$, and so ΔP is 8.2 mmHg, which is fairly significant.

We could have just plugged the parameters for each vessel directly into (7.25), without calculating $R_{\text{flow},0}$, but our scaling approach does give some new insight.

These estimates agree with what we would expect from Fig. 8.13. Also, arterioles and capillaries are seen to be well modeled as resistance vessels. The aorta and large arteries have very small pressure drops across them, and behave more like compliance vessels. Veins have larger diameters than the corresponding arteries, and consequently much lower resistances and pressure drops across them.

Along any vessel there is obviously a linear pressure drop with distance x along the vessel. This is seen from Poiseuille's Law, (7.25), $(\Delta P/L) = \left(8\eta/\pi r^4\right) Q$ or, recognizing that this change in pressure is negative,

$$\frac{\mathrm{d}P}{\mathrm{d}x} = -\frac{8\eta}{\pi r^4} Q. \tag{8.14}$$

This represents a "distributed" or "transmission-line" view of blood flow, in which flow is analyzed per unit length along the vessel, whereas in (8.2) and (8.11) flow was analyzed with the vessel as a "lumped" parameter (see Appendix D).

We can study the pressure drop in clogged arteries. There could be a larger pressure drop for the same Q or a smaller Q for the same pressure drop if r decreases, as occurs with clogged arteries, or with fewer vessels. This can stimulate an increase in blood pressure to maintain the flow rate or lead to a reduction in flow at a given inlet pressure, which is what actually happens in coronary artery disease.

How can we "optimize" the design of resistive vessels and how such vessels bifurcate and otherwise branch into smaller vessels (and what does it really mean to optimize the design)? See Problems 8.28–8.31.

Radial Profile of Blood Flow

Blood flow in vessels is not uniform. We have implicitly been assuming that the blood flow is parabolic (Fig. 7.11) because we are modeling blood as a Newtonian fluid. However, the parabolic profile of blood flow speed in a resistive vessel from (7.40) is not quite accurate. This assumes steady-state flow, which begins only a certain distance from a bifurcation ((7.41)–(7.43)). This approach to steady-state flow is depicted for a different initial condition in Fig. 7.11. Furthermore, because whole blood is not a Newtonian fluid, the steady-state profile is not parabolic, as is seen in Fig. 7.13.

Properties of a Compliance Vessel

We can show that (8.4) and (8.5) are reasonable models of an elastic compliance vessel and determine the compliance C_{flow} by examining a thin-walled cylindrical tube of inner radius r, thickness w, and length L, with a pressure difference P between the inside and outside of the vessel (Fig. 8.23). The Law of Laplace for cylinders (7.4) shows that the tension T in the walls of a cylinder in equilibrium is rP. We can conceptually slit the vessel along its length and see that this tension (force per unit length along L) corresponds to a force per unit area of T/w on the rectangular face with dimensions w and L. The length of the rectangular solid is $2\pi r$. Let us consider the stress–strain relation $\sigma = Y\epsilon$ in the context of this unfolded vessel. The stress is $\sigma = T/w$.

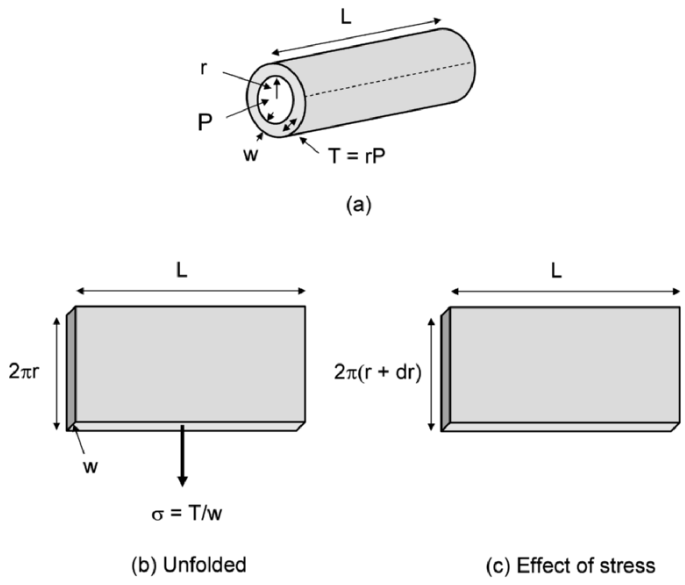

Fig. 8.23. Compliance vessels: unfolding the vessel

The strain is $\epsilon = \mathrm{d}(2\pi r)/2\pi r = \mathrm{d}r/r$ or really $\mathrm{d}r/r_0$, where r_0 is the radius with no pressure. The stress–strain relation is

$$\frac{T}{w} = Y\frac{\mathrm{d}r}{r_0} \tag{8.15}$$

or with $T = rP$

$$\frac{\mathrm{d}r}{r_0} = \frac{T}{wY} = \frac{rP}{wY} = \frac{r_0}{w}\frac{P}{Y}. \tag{8.16}$$

The internal volume of the vessel is $V = \pi r^2 L$. Therefore we see that $\mathrm{d}V = 2\pi r(\mathrm{d}r)L$ and $\mathrm{d}V/V = 2\mathrm{d}r/r = 2(r_0/w)(P/Y)$. For small changes in volume

$$V(P) = V_\mathrm{d}\left(1 + \frac{\mathrm{d}V}{V_\mathrm{d}}\right) = V_\mathrm{d}\left(1 + 2\frac{r_0}{w}\frac{P}{Y}\right) \tag{8.17}$$

$$= V_\mathrm{d} + 2V_\mathrm{d}\frac{r_0}{w}\frac{P}{Y} = V_\mathrm{d} + 2(\pi r_0^2 L)\frac{r_0}{wY}P, \tag{8.18}$$

with $V_\mathrm{d} = \pi r_0^2 L$. Using (8.5) the compliance is

$$C_\mathrm{flow} = \frac{2\pi r_0^3 L}{wY}. \tag{8.19}$$

How large is this expansion? The pressure in the aorta and large arteries is 120 mmHg = 0.0158 MPa during systole. The value of Y for such vessels is about 1 MPa (Table 4.2) and so $P/Y = 0.0158 \sim 1.6\%$. The thickness of arterial walls is typically 1/5 of the radius, so $r_0/w = 5$. This means that the fractional increase in radius of these vessels due to this internal pressure is 8% and the fractional increase in volume is 16% – both sizeable fractions. Also, this predicts that the radius changes by ∼3% during each heart beat during the changes between systolic (120 mmHg) and diastolic (80 mmHg) pressure. Veins are also compliance vessels.

Distensibility

Such compliance changes are equally well described in terms of the distensibility D_flow of the tube. The cross-sectional area A of a tube increases by ΔA when the pressure difference between the inside and outside of the tube increases by ΔP. The distensibility is defined as the fractional change in area for a change in pressure:

$$D_\mathrm{flow} = \frac{\Delta A/A}{\Delta P}. \tag{8.20}$$

With $A = \pi r^2$ and $\Delta A = 2\pi r\,\Delta r$, we see that $\Delta A/A = 2\,\Delta r/r$ and using (8.16),

$$D_\mathrm{flow} = \frac{2\,\Delta r/r}{\Delta P} = \frac{2(r/w)(\Delta P/Y)}{\Delta P} = \frac{2r}{wY} = \frac{1}{Y(w/d)}, \tag{8.21}$$

where $d = 2r$ is the diameter, w is the wall thickness, and w/d ($\ll$1).

A more exact analysis relates the Young's modulus for circumferential stretch, which we still call Y, to the external and internal diameters d_e and d_i, the change in external diameter Δd_e occurring with this change in pressure difference, and Poisson's ratio υ [372]. This gives

$$Y = \frac{\Delta P}{\Delta d_\mathrm{e}} \frac{2 d_\mathrm{e} d_\mathrm{i}^2}{d_\mathrm{e}^2 - d_\mathrm{i}^2} (1 - \upsilon^2). \tag{8.22}$$

For a thin-walled tube with wall thickness $w = (d_\mathrm{e} - d_\mathrm{i})/2 \ll d_\mathrm{i}$, and with $d_\mathrm{e} \sim d$ and $d_\mathrm{i} \sim d - 2w$, we find

$$Y = \frac{\Delta P}{\Delta d} \frac{d^2}{2w} (1 - \upsilon^2). \tag{8.23}$$

With $\Delta A/A = 2\,\Delta d/d$

$$D_\mathrm{flow} = \frac{\Delta A}{A} \frac{1}{\Delta P} = \frac{2\,\Delta d}{d} \frac{1}{\Delta P} = \frac{(1 - \upsilon^2)}{Y(w/d)}. \tag{8.24}$$

This reduces to (8.21) for small Poisson's ratios.

Flow with Resistance and Compliance

If a vessel is resistive and compliant [405], the change in pressure with distance is

$$\frac{\mathrm{d}P}{\mathrm{d}x} = \frac{\mathrm{d}P}{\mathrm{d}r} \frac{\mathrm{d}r}{\mathrm{d}x} = \frac{2}{C'_\mathrm{flow}} \frac{\mathrm{d}r}{\mathrm{d}x}, \tag{8.25}$$

using $\mathrm{d}P/\mathrm{d}r = 2/C'_\mathrm{flow}$ from (8.7). Setting this equal to $\mathrm{d}P/\mathrm{d}x$ from (8.14) and bringing the r terms to the left and the x terms to the right, gives

$$r^4 \mathrm{d}r = -\frac{4 C'_\mathrm{flow} \eta}{\pi} Q\,\mathrm{d}x. \tag{8.26}$$

After integrating over a vessel length from $x = 0$ to $x = L$, we get

$$(r(x=0))^5 - (r(x=L))^5 = \frac{20 C'_\mathrm{flow} \eta}{\pi}\, QL \tag{8.27}$$

and after using (8.6)

$$\left(r_\mathrm{d} + \frac{C'_\mathrm{flow}}{2} P(x=0)\right)^5 - \left(r_\mathrm{d} + \frac{C'_\mathrm{flow}}{2} P(x=L)\right)^5 = \frac{20 C'_\mathrm{flow} \eta}{\pi}\, QL. \tag{8.28}$$

(See Appendix C.)

Both terms on the left side can be expanded to five terms. The first terms are both r_d^5, which cancel, and for relatively small compliance

($C'_{\text{flow}}P/2r_{\text{d}} \ll 1$) only the next two of the remaining four terms in each need to be retained, giving

$$Q = \frac{\pi r_{\text{d}}^4}{8\eta L}\left(P(0) - P(L)\right)\left(1 + \frac{C'_{\text{flow}}}{r_{\text{d}}}\left(P(0) - P(L)\right)\right). \tag{8.29}$$

This is Poiseuille's Law (7.24) with a correction for compliance. So, for a rigid wall vessel ($C'_{\text{flow}} = 0$) the flow rate Q is linear with the pressure drop, but when compliance is included, the variation with pressure drop is between linear and quadratic. This relation says that for a given pressure drop, the flow rate is increased due to the compliant nature of the vessel.

The electrical analog of blood flow is described in Appendix D.

The Strength of Blood Vessel Walls

The pressure inside blood vessel walls P exceeds that outside P_{ext}, by $\Delta P = P - P_{\text{ext}}$. *How large of a tension should the vessel walls be able to withstand to support this positive pressure differential?* Chapter 7 showed the answer is provided by the Law of Laplace for hollow cylinders (7.4). For a cylinder of radius of curvature R, this tension T is

$$\Delta P = \frac{T}{R}. \tag{8.30}$$

Table 8.4 shows that the tension capillaries need to withstand is very small because of their small radius. This circumferential stress, the tension (force

Table 8.4. Calculated tension in blood vessel walls. (Using data from [382] and [391])

vessel	diameter (mm)	wall thickness, w (mm)	internal pressure, ΔP (mmHg)	wall tension, T (dyne/cm)	T/w (kPa)
aorta	24.0	3.0	100	160,000	53
large artery	8.0	1.0	97	52,000	52
medium artery	4.0	0.8	90	24,000	30
small artery	2.0	0.5	75	10,000	20
arteriole	0.3	0.02	60	1,200	60
capillary	0.008	0.001	30	16	16
venule	0.02	0.002	20	27	13
small vein	3.0	0.2	18	3,600	18
medium vein	5.0	0.5	15	5,000	10
large vein	15.0	0.8	10	10,000	12
vena cava	30.0	1.5	10	20,000	13

The wall thickness w is $R/5$ for arteries and $R/10$ for veins (where R is the vessel radius) and 1 µm for capillaries. Also see Table 8.2.

per tube length) divided by the vessel wall thickness w, is surprisingly similar for these very different vessels. T/w can be compared to the UTS of such vessels.

Flow in Curving Arteries

In Chap. 7 we showed that the arterial walls feel a pressure due to the difference in hydrostatic pressure inside and outside the vessel. This pressure is felt equally around the wall. When blood flows in an artery that curves, a force equal to the centripetal force is felt on the arterial wall on the outer surface of the curve to change the direction (but not the magnitude) of the momentum vector of blood flow. *How large is this force? Is it comparable to the uniform hydrostatic pressure? Does it constitute a significant extra load on the arterial wall?*

For an artery of internal radius R that is turning with a radius $\mathcal{R}$ (Fig. 7.16), with blood of density ρ and average flow speed u, (7.44), this peak pressure is

$$P_{\text{cent}} = 2\rho u^2 \frac{R}{\mathcal{R}}. \tag{8.31}$$

This is largest for the fastest blood flow, which is in the aorta. Using $\rho = 1\,\text{g/cm}^3$, $R = 1.25\,\text{cm}$, $\mathcal{R} = 2\,\text{cm}$, and $u = 100\,\text{cm/s}$, we find $P_{\text{cent}} = 4.7\,\text{mmHg}$. This $\sim$5 mmHg is the extra pressure that must be supplied by the outer aorta wall to turn the blood around the aortic arch. This is small compared to the typical average aorta pressure of 100 mmHg, and does not likely promote pathological conditions such as aneurysms.

8.2.3 Capillaries and Osmotic Pressure

The purpose of systemic circulation is to supply blood to the capillary bed. We have seen that pressure is needed to bring the blood to the capillaries. There is diffusion and bulk flow between the blood in the capillaries and the interstitial fluid. Diffusion across the capillary wall transports oxygen, which is carried in red blood cells, and carbon dioxide, which is dissolved in the blood. (Not enough oxygen can be directly dissolved in the blood for our metabolic needs.) In the systemic capillaries there is net diffusion of oxygen out of the capillaries and carbon dioxide into them. In the pulmonary capillaries there is net diffusion of oxygen into the capillaries and carbon dioxide out of them. There is also bulk flow of fluid across the capillary walls due to the net pressure across the walls.

There are two forces driving this bulk transport: the force/area mechanical pressure P we have been discussing, which we will call *hydrostatic pressure* in this section, and a chemical driving force, called *osmotic pressure*, Π. The osmotic pressure characterizes the flow across a semipermeable membrane that occurs to equalize the concentrations of solutes on either side of

the wall. This drives species that can permeate across the membrane, such as water, across it from the side of low concentration of solute to the side of high concentration. For low concentrations, the osmotic pressure is given by the van't Hoff equation

$$\Pi = n_{\mathrm{s}} RT, \tag{8.32}$$

where n_{s} is the density (or concentration) of the solute in solution in moles per unit volume. This looks deceptively similar to the ideal gas law (7.2). It is the difference in the sum of these on either side of the capillary walls, $P + \Pi$, that drives the net transport across these walls.

As seen in Fig. 8.24, the hydrostatic force in the capillary is always greater inside the vessel than outside, and it decreases from about 36 mmHg at the arteriole side to 15 mmHg at the venule end because of viscosity. If this were

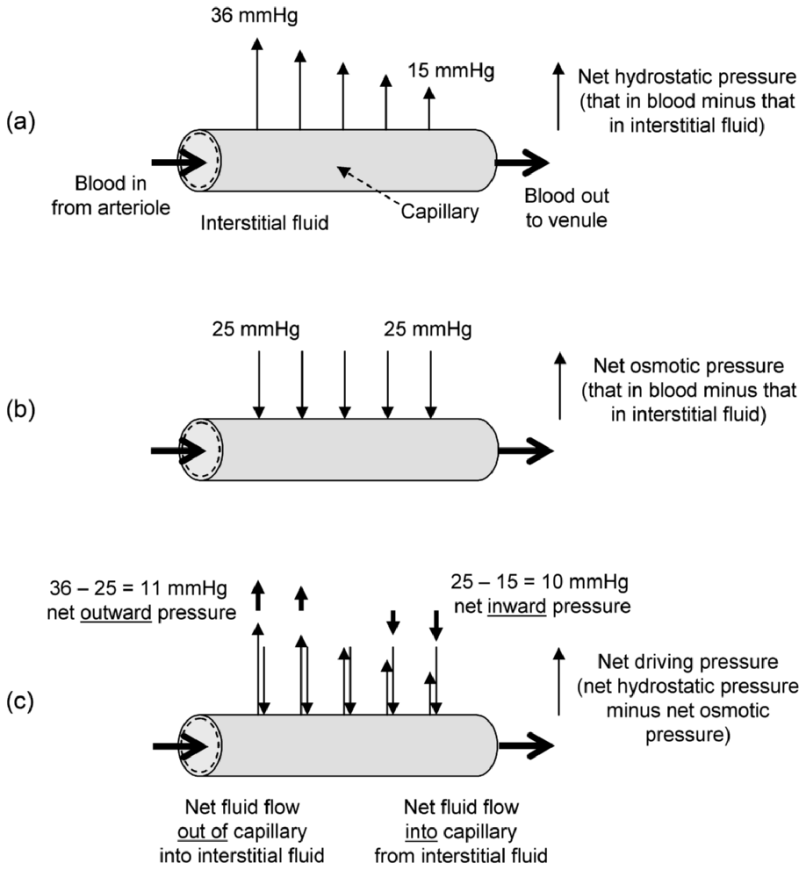

Fig. 8.24. Osmotic pressure in a capillary. (Based on [417])

the only driving force, there would be a large net flow of fluid from inside the capillary to the interstitial fluid! Small molecules, such as oxygen and carbon dioxide, are able to diffuse across the capillary wall, but larger molecules, such as proteins, cannot. Because there is a much higher density of proteins in the blood than in the interstitial fluid, there is a chemical driving force of fluid into the capillary to try to equalize these densities. This leads to a net osmotic pressure of about 25 mmHg into the capillary. In Fig. 8.24, the net pressure is 36 mmHg − 25 mmHg = 11 mmHg outward at the arteriole end and 15 mmHg − 25 mmHg = −10 mmHg inward at the venule end. Therefore, there is net flow out of the capillary in the arteriole end and net flow into the capillary in the venule end. There is a small imbalance in this and a small net bulk flow out of the capillary.

We have assumed that the flow in all blood vessels, including the capillaries, is laminar. This cannot be really true for capillaries because many capillaries have an inner diameter of 5 or 6 μm and the red blood cells have a diameter of about 7.5 μm. The red blood cells deform to pass through the capillary and the resulting flow is called *bolus flow*. The red blood cells form plugs and the blood plasma is trapped in the regions between these plugs and moves in streamlines. Nowhere else in the body is the multicomponent nature of blood more apparent.

One major function of this capillary blood flow is the transfer of oxygen to the cells, leaving oxygen-depleted blood in the veins. As in (6.18), the rate of body consumption of O_2, $\mathrm{d}V_{O_2}/\mathrm{d}t$ equals the product of the cardiac output Q_t (see below) and the difference in the oxygen partial pressure in the arteries and veins, $p_a - p_v$

$$\frac{\mathrm{d}V_{O_2}}{\mathrm{d}t} = Q_t(p_a - p_v). \tag{8.33}$$

If the lungs are bringing in air fast enough, then $p_a - p_v$ is fixed, and during aerobic exercise $\mathrm{d}V_{O_2}/\mathrm{d}t$ increases linearly with Q_t. For a person with average fitness, the maximum blood flow rate is ≈19 L/min, for a highly fit person it is ≈25 L/min, and for an elite athlete it can be 35 L/min.

Oxygen combines with hemoglobin in the red blood cells in the lungs where the partial pressure of oxygen is high, about 100 mmHg. It is transported in the arteries to the tissues where it is released because the partial pressure of oxygen is low – and it is then used. The blood in the veins is then depleted in oxygen. Figure 8.25a shows the hemoglobin–oxygen dissociation curve. Clearly, hemoglobin is over 90% saturated with O_2 for partial pressures above 60 mmHg O_2. Increased CO_2 levels, increased temperature, and decreased pH all shift this curve to the right (Fig. 8.25b), which improves body performance. In Fig. 8.25a the dissociation curve for the lung is seen to be to the left of that in the tissues because the pH is higher and the CO_2 level is lower in the lung, increasing oxygen binding in the lungs relative to that in the tissues. During exercise, the muscle tissue pH falls and the local partial pressure of CO_2 and the local temperature increase. All of these changes move the curve to the right and this leads to more oxygen release (Fig. 8.25b).

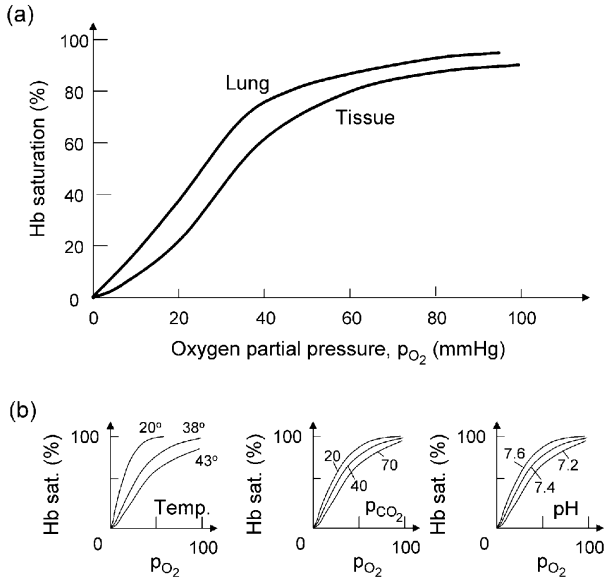

Fig. 8.25. (**a**) Hemoglobin–oxygen equilibrium in the lungs and tissue. During exercise oxygen intake is improved by the lung curve moving to the left and the tissue curve (exercising muscle) moving to the right due to increasing temperature, CO_2 partial pressure, and 2,3-diphosphoglycerate (DPG) (an end-product of red blood cell metabolism), and decreasing pH. (**b**) The hemoglobin–oxygen equilibrium shifts to the right with increasing temperature, increasing CO_2 partial pressure, and increasing DPG (not shown), and decreasing pH. (Based on [411] and [419])

During exercise $p_a - p_v$ increases from the resting value of about 50 mL of oxygen per L of blood to 150 mL/L in normal people at $(dV_{O_2}/dt)_{max}$ (and to 160–170 mL/L in very fit people), in part because blood flow is being diverted from the organs to the muscles, where oxygen extraction is higher because of the exercise (see Fig. 8.25).

8.2.4 Blood Flow Rates and Speeds

The heart pumps about 80 mL ($= 80\,cm^3$) of blood per contraction; this quantity is called the *stroke volume* V_{stroke}. The pump rate is the *heart beat rate* F of about 60/min or 1/s = 1 Hz. The *cardiac output* or total volumetric flow rate Q_t is the product of these two

$$Q_t = FV_{stroke} \tag{8.34}$$

or about $80\,cm^3/s = 4.8$ L/min. The total volume of blood is about 4.5–5.0 L, so all the blood is pumped throughout the body every minute. The flow rate in the arteries, arterioles, capillaries, venules, and veins are all the same because

of the continuity of flow. (Q_t is actually a little less in the venules and veins because of the net fluid loss in the capillaries.)

The total flow in each of these vessel systems, Q_t, equals the total cross-sectional area A times the blood speed u, $Q_t = Au$. The parameters A and u are plotted in Fig. 8.16, which shows this inverse relationship for a flow rate of $90\,\mathrm{cm}^3/\mathrm{s}$. The cross-sectional area of the aorta is $3\,\mathrm{cm}^2$, so in the aorta $u = (90\,\mathrm{cm}^3/\mathrm{s})/3\,\mathrm{cm}^2 = 30\,\mathrm{cm/s}$. In the capillaries the flow speed is much slower, $(90\,\mathrm{cm}^3/\mathrm{s})/4{,}000\,\mathrm{cm}^2 = 0.02\,\mathrm{cm/s} = 0.2\,\mathrm{mm/s}$. The net cross-sectional area in the capillaries is larger ($\sim$4,000 cm^2) even though they are very small ($\sim 3.5\,\mu\mathrm{m}$ in radius) because there are so many of them ($\sim 10^{10}$). In the vena cava the flow speed is relatively fast $(90\,\mathrm{cm}^3/\mathrm{s})/18\,\mathrm{cm}^2 = 5\,\mathrm{cm/s}$. In the arterial and venous systems, the smaller the vessel radius, the larger the total cross-section of all vessels in that order and the slower the blood speed. These are actually average blood flows during each cycle.

The maximum Reynolds number ($Re = \rho u d/\eta$, (7.11)) over a cardiac cycle ranges from $\sim$6,000 in the heart and aorta to $<10^{-3}$ in the capillaries. The nominal lower threshold for turbulent flow is $Re \sim 2,000$, so it is possible that flow in the aorta is turbulent.

The overall flow in the systemic arterial system can be described by relating the total cardiac output Q_t to the systemic arterial pressure P_{sa}, by

$$P_{sa} = (\mathrm{TPVR})Q_t, \tag{8.35}$$

where TPVR is the total peripheral vascular resistance – which is due to the combined effect of all the organ beds of systemic circulation (mostly arterioles and capillaries). (P_{sa} should really be replaced by the pressure drop in the system. See Problem 8.20.) Normal values for the systemic system range from 700 to 1,600 dyne-s/cm^5, and analogous normal values for the pulmonary system range from 20 to 130 dyne-s/cm^5. Equation (8.2) applies to an individual vessel, while this describes the entire systemic system. The body regulates P_{sa} by controlling the cardiac output and this peripheral resistance. When we lie down, a large volume of blood is transiently stored in the lower extremities and abdomen, and so when we stand there is initially less flow of blood to the heart and a drop in blood pressure, which can make you faint. Even though Q_t decreases, P_{sa} drops only mildly because there is a prompt reflex that increases the TPVR (vasoconstriction). In contrast, when blood pressure rises suddenly, feedback tends to decrease the overall vascular resistance, to restore a lower blood pressure. These are two examples of body feedback and control, as described in Chap. 13.

We can also evaluate the overall compliances of the vascular systems, such as those of the systemic arterial and venous systems. The compliance is the reciprocal of the slope of a pressure–volume curve in Fig. 8.26. The smooth muscles surrounding a large vessel can change the volume of the vessel at a given pressure, either decreasing it (by stimulating the muscles) or increasing it (by inhibiting the muscles).

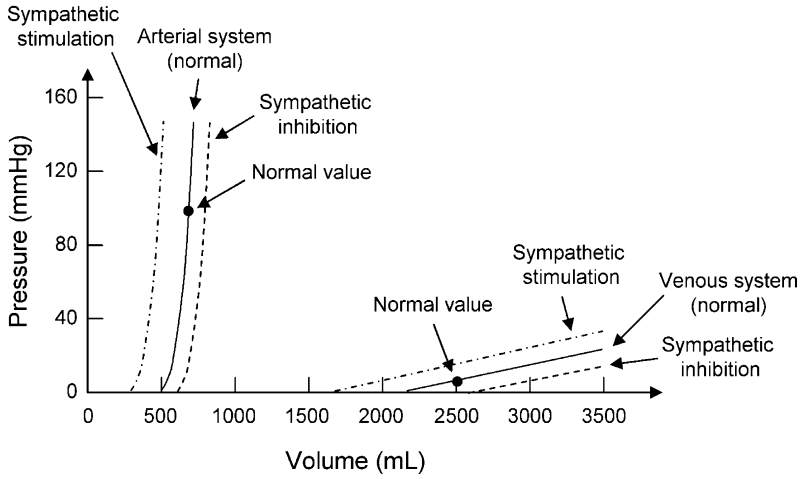

Fig. 8.26. Volume–pressure curves for the systemic arterial and venous systems, for normal conditions and for sympathetic stimulation and inhibition. (Based on [390])

During even moderate exercise the blood flow rate increases substantially, as seen in Fig. 8.27, and the absolute and relative distribution of blood to different parts of the body also changes radically. Figure 8.28 shows an example in which the flow rate increases from 5 to 12.5 L/min during exercise. There are extremely large increases of blood flowing to the skeletal muscle to supply oxygen for aerobic metabolism (up 1,066%), to the heart so it can pump faster (up 367%), and to the skin (up 370%) to assist cooling (which increases

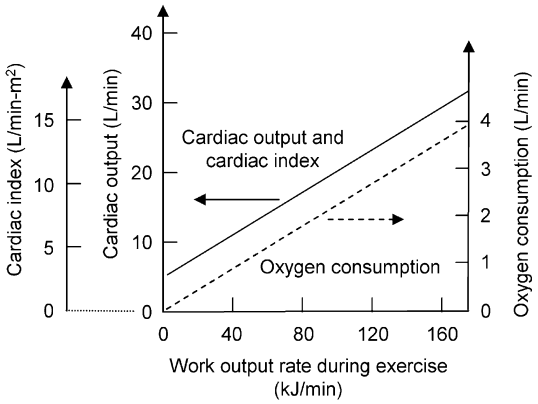

Fig. 8.27. Variation in cardiac output (and cardiac index) and oxygen consumption needed during varying levels of exercise with work output. (The cardiac index is the cardiac output divided by the person's surface area.) (Based on [389] and [390])

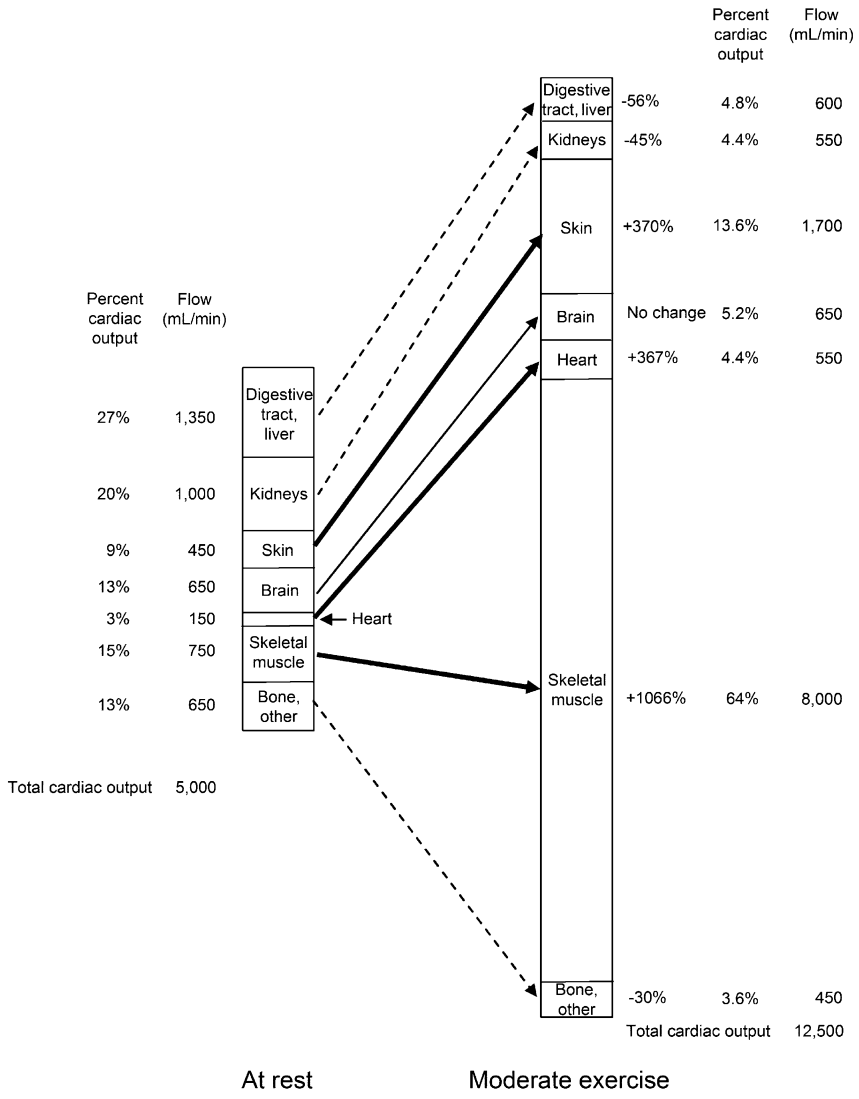

Fig. 8.28. Blood flow to different organs at rest and during moderate exercise, showing no change to the brain, increases to the skin, heart, and skeletal muscles (*thick arrows*), and less blood flow elsewhere (*dashed arrows*). (Based on [417])

the skin temperature, thus accelerating radiative and convection conduction from the body). Blood flow to the brain is unchanged. In contrast, blood flow to the digestive track, liver, and kidneys decrease by a factor of ∼2. These changes are also seen in Tables 8.5 and 8.6. Figure 8.29 shows that the blood

Table 8.5. Total cardiac flow (mL/min) for organs during exercise, including percentage of total flow. (Using data from [381])

organ	rest	light exercise	heavy exercise	maximal exercise
brain	750 (13%)	750 (8%)	750 (4%)	750 (3%)
heart	250 (4%)	350 (3.5%)	750 (4%)	1,000 (4%)
muscle	1,200 (21%)	4,500 (47%)	12,500 (72%)	22,000 (88%)
skin	500 (8.5%)	1,500 (16%)	1,900 (11%)	600 (2.5%)
kidney	1,100 (19%)	900 (9.5%)	600 (3.5%)	250 (1%)
abdomen	1,400 (24%)	1,100 (11.5%)	600 (3.5%)	300 (1.2%)
other	600 (10.5%)	400 (4%)	400 (2%)	100 (0.4%)
Total	5,800 (100%)	9,500 (100%)	17,500 (100%)	25,000 (100%)

flow to the calf during rhythmic exercise is higher than normal and it varies with time.

This increase in cardiac output occurs because of increases in both the heart rate and the stroke volume; the blood speed also increases because $Q_t = Au$. The faster the heart rate, the shorter is diastole, while the duration of systole does not change. For short term (5–10 min) submaximal exercise, the cardiac output increases from 5 L/min to a new steady-state value in about 2 min. For a steady-state cardiac output of 18 L/min, the stroke volume increases from about 70 to 120 mL/beat and the heart rate F from about

Table 8.6. Approximate blood flow (perfusion) for tissues and organs, per gram. (Using data from [382])

tissue type	location or organ	specific blood flow rate (mm^3/s-g)
adipose tissue	abdomen, ∼20 mm thick	0.51
	abdomen, >40 mm thick	0.31
	thigh, ∼20 mm thick	0.33
bone	humerus, marrow flow only	0.055
connective tissue	typical basal (max)	0.50 (2.5)
muscle	typical basal (max)	0.50 (10)
organ	brain, basal (max)	9.1 (18.3)
	gastrointestinal track, basal (max)	6.7 (26.7)
	heart, basal (max)	13.7 (64.0)
	kidney, basal (max)	68 (100)
	liver, basal (max)	12 (54)
	lung, basal (max)	90 (490)
skin	typical resting flow (max)	1.7 (25.0)

In some cases the basal rate is given, along with the maximum rate in parentheses.

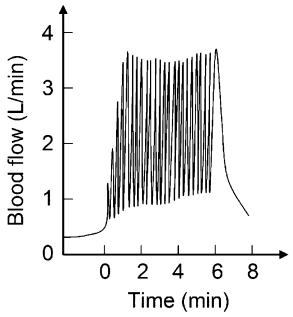

Fig. 8.29. Blood flow to the calf during rhythmic contraction exercises, showing less blood flow during contractions than between them. (Based on [366] and [390])

70 to 150 beats/min. The cardiac output returns to the resting value in 5–10 min after exercise.

For a longer submaximal workout (30–60 min), the new steady-state cardiac output is maintained, but the stroke volume slowly decreases and the heart rate gradually increases with time, particularly in warmer environments. This *cardiovascular drift* is caused by a decrease in the venous return of blood to the heart, which decreases the stroke volume and so the heart rate must increase to maintain the same cardiac output. This decrease in venous return is caused by two factors. (1) During such exercise more blood flows under the skin to help lower the increase in body core temperature caused by the increased metabolic activity (Chap. 6), and this lowers the steady-state flow of blood back. (2) During exercise water flows from the blood to the surrounding cells and tissues because of increased arterial pressure and the compression of venules due to muscle action. This produces a steady-state decrease in blood plasma and blood volume, and a steady-state decrease in blood returning to the heart and the stroke volume.

Stroke volume and cardiac output are determined by the preload and afterload conditions (see later), contractility (ability to contract), and heart rate. The cardiac output is not directly regulated, but there is a feedback and control system that regulates arterial pressure that affects the heart rate and contractility, as well as afterload and other factors that control the preload [415].

The Frank–Starling mechanism (or Starling's Law of the heart) states that the larger the end-of-diastole volume or pressure (the *preload*), the larger the stroke volume, as is seen in Fig. 8.30. Furthermore, the larger the aortic pressure, the less blood can be ejected by the left ventricle (the *afterload*), as is seen in Fig. 8.31. An increase in the heart rate also increases cardiac output, however, the increase is sublinear because the stroke volume decreases (Fig. 8.32) due to the above preload and postload factors. With greater cardiac output there is less blood in the veins to return to the heart for diastole (lower preload) and the arterial pressure is higher so the heart

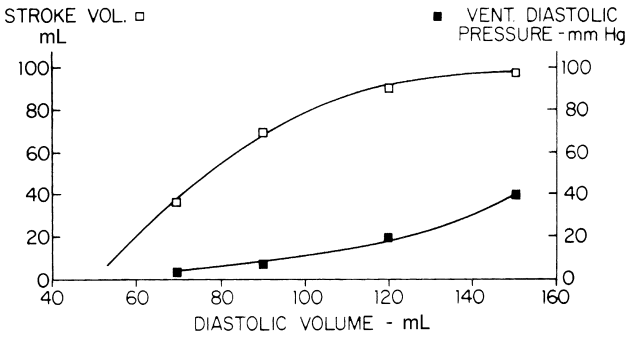

Fig. 8.30. The stroke volume increases with diastolic volume and pressure, as seen with data for four heart beats. This dependence, along with the explanation of it, is known as the Frank–Starling mechanism. (Reprinted from [415]. Used with permission of Elsevier)

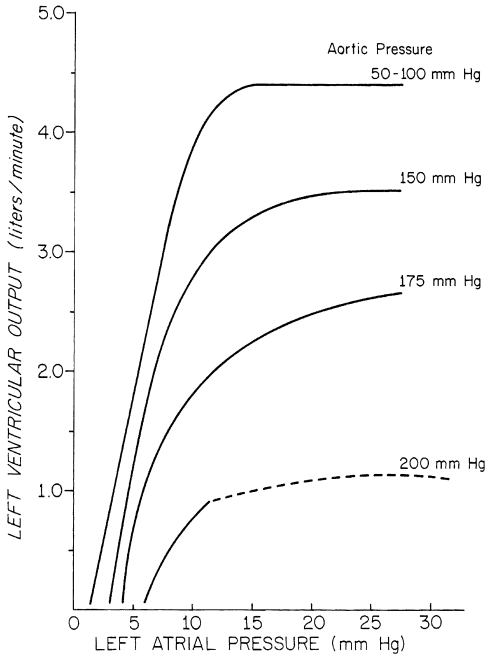

Fig. 8.31. The left ventricular output (the cardiac output) vs. left atrial pressure for different aortic pressures. It increases with this atrial pressure and then levels off, and decreases with increasing aortic pressure. (Reprinted from [415]. Used with permission of Elsevier; adapted from [413])

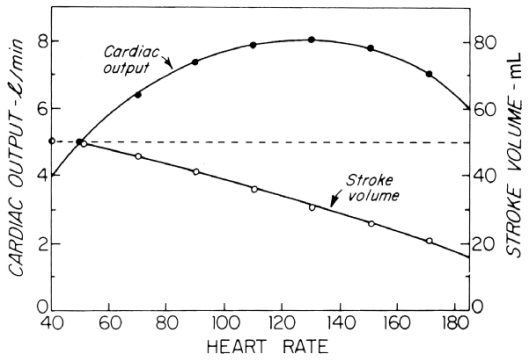

Fig. 8.32. Typical dependence of cardiac output and stroke volume on heart rate. (Reprinted from [415]. Used with permission of Elsevier)

can eject less blood (higher afterload), assuming the peripheral resistance is constant.

For the most part, these are changes in the systemic system. Similar increases in cardiac output have to occur in the pulmonary system. (Why?) This occurs by an increase in the number of open capillaries in the lung, by up to a factor of three, and by a distending of all the pulmonary capillaries, which increases the flow in each capillary by up to a factor of two, with very little change in the pulmonary arterial pressure (Fig. 8.33).

The maximum heart rate $F_{\max}$ (in beats/min) depends on age Y (in years) as

$$F_{\max} = 220 - Y. \tag{8.36}$$

The standard error in this relation is ± 10 beats/min, which means 67% of people have a maximum rate ± 10 beats/min within the value predicted by

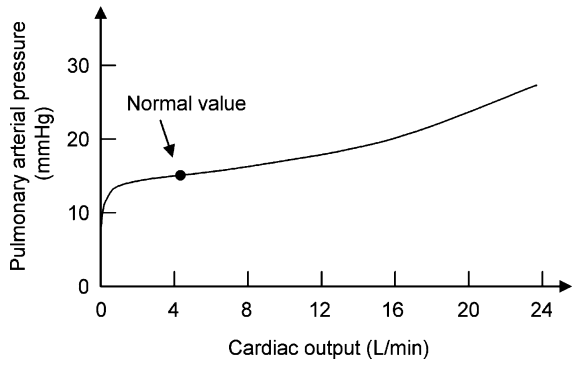

Fig. 8.33. The pulmonary arterial pressure vs. cardiac pressure, showing it does not change much during exercise. (Based on [390])

this relation and 95% of all people have a rate that is ±20 beats/min within the predicted value. Because F does not depend on the level of fitness, athletes increase their maximum cardiac output by increasing their stroke volumes. Stroke volumes in untrained athletes are 50–70 mL at rest and reach 80–110 mL during heavy activity. For trained and highly trained athletes these stroke volumes increase to 70–90 mL and 90–110 mL at rest and 110–150 mL and 150–220 mL during heavy activity.

In steady state, the cardiac output Q_t must equal FV_{stroke} (8.34), as well as P_{sa}/TPVR (8.35). During heavy exercise, Q_t increases from 5 to 20 L/min, so not only must F and V_{stroke} increase, as we have described, but P_{sa}/TPVR must increase accordingly. Diastolic pressure changes little during exercise, remaining within ±10 mmHg of the resting value. The systolic pressure increases to about 200 mmHg for men and 180 mmHg for women. Using (8.1), the mean arterial pressure then increases only to 140–150 mmHg, which cannot account for most of the increase in blood flow rate. During heavy exercise the systemic vascular resistance TPVR decreases to about 40% of its resting value because of the widening of muscular vascular beds that are normally constricted at low levels of activity.

Figure 8.34 show that blood pressure typically increases with age. Hypertension begins with systolic pressure ≥140 mmHg or diastolic pressure ≥90 mmHg. In essential hypertension this blood pressure is heightened for no obvious reason. The average blood pressure is the product of the total peripheral vascular resistance and the cardiac output, $P_{sa} = (\text{TPVR})Q_t$ (8.35). In people under 40 years of age, hypertension is driven by increased cardiac output, with normal TPVR. In older people, the cardiac output is normal or reduced, but the TPVR is high.

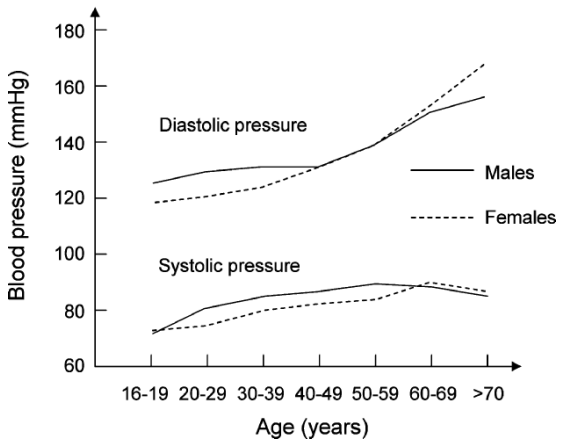

Fig. 8.34. Systolic and diastolic blood pressure is shown for males and females, averaged over age groups. The trend is to increased blood pressure with age. (Based on [400] and [403])

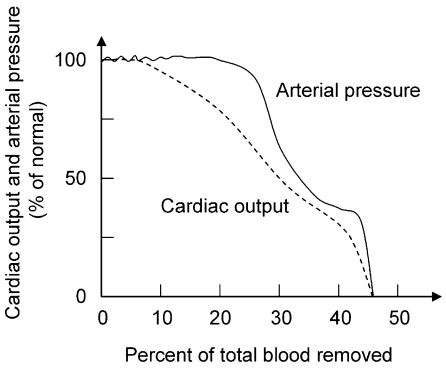

Fig. 8.35. Cardiac output and arterial pressure with decreased blood volume from hemorrhaging. (Based on [390])

Circulatory shock occurs when there is too little blood flowing generally in the body, and this results in tissue damaged from the inadequate delivery of oxygen and nutrients to the cells and the inadequate removal of waste products. Such shock can occur from inadequate pumping of blood by the heart or by inadequate venous return of blood to the heart, such as due to diminished blood volume (*hypovolemia*), decreased capillary vasomotion, or obstructed circulation. *Hemorrhage* is often the cause of the diminished blood volume. Figure 8.35 shows that cardiac output and arterial pressure can withstand a ∼10% blood loss – if this were not so you would not be able to donate blood – but decrease for larger losses and approach zero with 35–45% blood loss. The localized loss of blood flow to the brain, *strokes*, is discussed later. This occurs due to clogged arteries and hemorrhaging. The localized loss of blood flow to the heart results in a *myocardial infarction* (heart attack).

8.2.5 Consequences of Clogged Arteries

Atherosclerosis (a-thear-oh'-scler-oh-sis) occurs when a deposit or atheroma (a-thear-oh'-ma) (or plaque) forms on an arterial wall (Fig. 8.36). The smaller

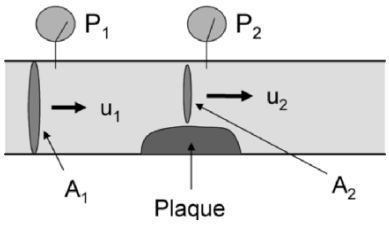

Fig. 8.36. Sketch of flow in a clogged artery

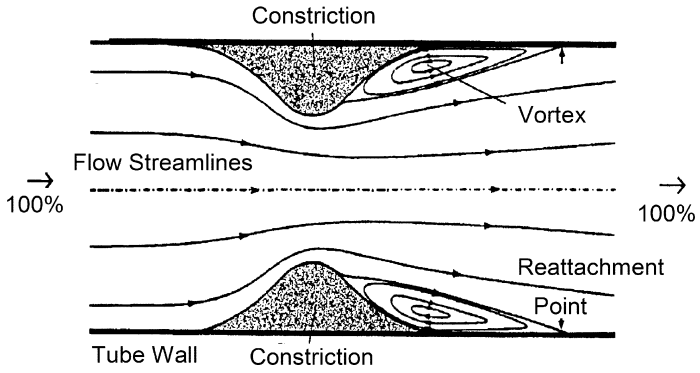

Fig. 8.37. The flow is partially turbulent in clogged arteries. (From [382]. Adapted from [398]. Courtesy of Robert A. Freitas Jr., Nanomedicine, Vol. 1 (1999), http://www.nanomedicine.com)

cross-sectional area at this site, because $A_2/A_1 < 1$, leads to a faster flow speed due to continuity of flow, with

$$u_2 = \frac{A_1}{A_2} u_1 \tag{8.37}$$

from (7.16). For $A_2/A_1 = 1/3$, we find that $u_2 = 3u_1$. Also from Bernoulli's equation (7.18) we find that

$$P_2 - P_1 = \frac{1}{2}\rho u_1^2 \left(1 - \left(\frac{A_1}{A_2}\right)^2\right) \tag{8.38}$$

and so $P_2 < P_1$ and for $A_2/A_1 = 1/3$ we see that $P_2 - P_1 = -4\rho u_1^2$. This pressure drop increases with blood speed and so it is expected to increase with increased physical activity. This flow is not necessary laminar in the occluded region, as is seen in Fig. 8.37.

In 1954 Arturo Toscanini was conducting the NBC Symphony Orchestra. (This orchestra was pretty prestigious then, performing on radio and the then-new television, but it does not exist now.) He was vigorously waving his arms, as conductors often do, and he fainted. Why? Equation (8.38) contains the answer and Fig. 8.38 illustrates why. He suffered a *transient ischemic attack*, or TIA [387, 399]. *Ischemia* (iss-kee'-mee-uh) is the local decrease in blood flow. TIA is the temporary loss of blood to the brain by the "subclavian steal syndrome." It results in temporary dizziness, double vision, headache, and weakness in the limbs. By its nature it is only temporary, but it indicates a more severe problem.

The left and right carotid arteries are two major arteries supplying blood to the anterior brain. The left and right vertebral arteries supply blood to the posterior part of the brain. They branch off from the subclavian arteries that

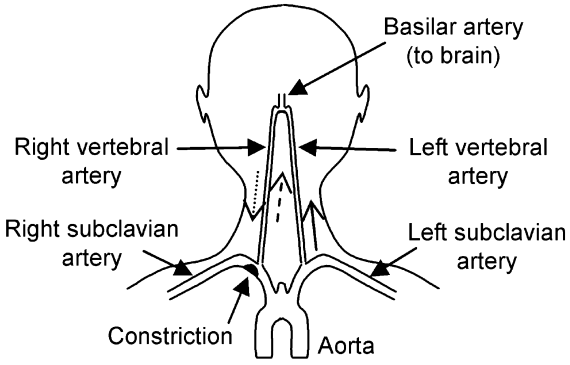

Fig. 8.38. Anterior view of the blood flow to the brain with the subclavian steal syndrome, resulting in a transient ischemic attack. Blood flow in the left vertebral artery is shown by the *unbroken line arrow*. Without the constriction, the blood flow in the right vertebral artery is normal, as shown by the *dashed arrow*, so there is normal blood flow to the basilar artery. With the constriction, there can be blood flow from the left vertebral artery to the right vertebral artery (*dotted arrow*), and there is no blood flow into the basilar artery. (Based on [387])

also supply blood to the arms. The internal carotid and vertebral systems join with each other at the base of the brain, forming the circle of Willis (Fig. 8.39, also see Fig. 8.43). Posteriorly, the flow in the left and right vertebral arteries merge to form a single basilar artery to the brain.

Say there is a constriction in the right subclavian artery near where the vertebral artery branches off (Fig. 8.38). The pressure before the constriction

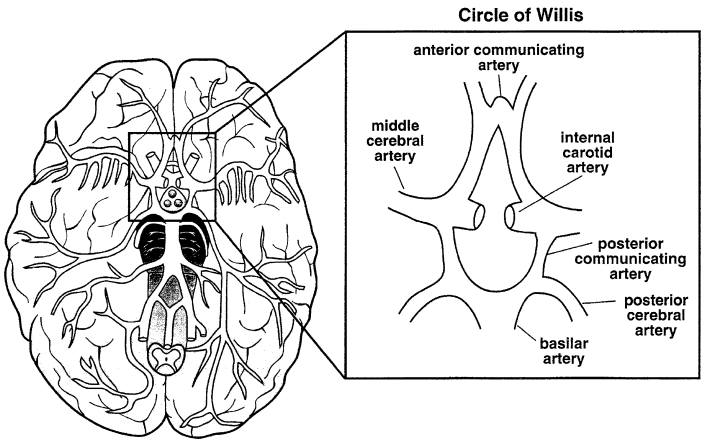

Fig. 8.39. Circle of Willis in the brain. (From [391])

and in the same region in the normal right side is P_1. Because of the constriction, the pressure on the left side is $P_2 < P_1$. This difference increases with faster blood flow. With vigorous motion of the arms, there is a need for greater blood flow in the subclavian arteries to supply more oxygen to the skeletal muscle in the shoulder and arms, and the blood speed in these arteries u_1 increases. If u_1 increases enough, P_2 becomes so much smaller than P_1 that flow in the left vertebral artery is diverted to the right vertebral artery – and does not flow to the basilar artery to the brain. When this happened to Toscanini, he fainted. He stopped waving his hands, of course. Blood flow to his arms then slowed down. The difference in P_1 and P_2 decreased to its usual smaller value (even with this constriction). Blood from both vertebral arteries then flowed to his basilar artery. His brain started receiving a normal flow of blood again, and he regained consciousness. All was fine – but this constriction had to be removed. (Toscanini never conducted again. The underlying reason for his fainting spell was not known in 1954; TIA was first explained in 1961.)

More examples of the effects of obstructions in arteries will be examined later in this chapter.

8.2.6 Work Done by the Heart and the Metabolic Needs of the Heart

How much work is done by the heart? Consider the left ventricle, which is a pump during systole, as diagrammed in Fig. 8.40.

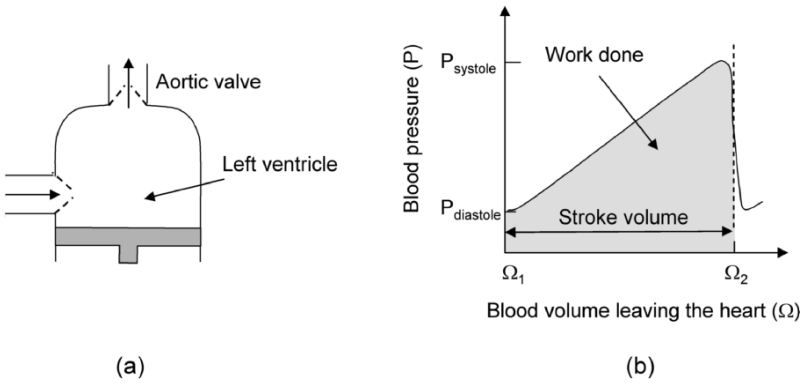

Fig. 8.40. (**a**) Schematic of the heart left ventricle as a pump, (**b**) and the pressure and volume of the left ventricle during systolic contraction during systole (from t_1 to t_2), showing the work done by the heart (*shaded area*). (Based on [367])

Work is done by this pump, with a force pushing the piston from the initial to final positions L_i to L_f to give

$$W = \int_{L_\mathrm{i}}^{L_\mathrm{f}} F\,\mathrm{d}L = \int_{L_\mathrm{i}}^{L_\mathrm{f}} (F/A)(A\,\mathrm{d}L) = \int_{V_\mathrm{i}}^{V_\mathrm{f}} P\,\mathrm{d}V, \tag{8.39}$$

where the force/area is the pressure, P, and the distance times the area is the ventricular volume, V. During systole the ventricular pressure increases from a very low value (that in the left atrium), to $P_\mathrm{diastole} \sim 80\,\mathrm{mmHg}$ (at time t_1), and then up to $P_\mathrm{systole} \sim 120\,\mathrm{mmHg}$, and it stays at this value until the end of systole (at time t_2). The aortic valve first opens when this pressure rises above P_diastole (at t_1), and blood is pumped out until systole is over (at t_2). At t_1, the volume of pumped blood is $\Omega_\mathrm{i} = 0$ and at t_2 it is $\Omega_\mathrm{f} = V_\mathrm{i} - V_\mathrm{f}$. Figure 8.40 shows this evolution of the ventricular pressure and pumped volume from t_1 to t_2.

The area under this curve is the work done and so $W = P_\mathrm{av} V_\mathrm{stroke}$, where P_av is average pressure during this cycle (averaged over the volume displaced and which does not necessarily scale linearly with time), and $V_\mathrm{stroke} = \Omega_\mathrm{f}$, the stroke volume. Clearly, $P_\mathrm{diastole} < P_\mathrm{av} < P_\mathrm{systole}$. For a linear variation, we see that $P_\mathrm{av} = (P_\mathrm{diastole} + P_\mathrm{systole})/2 \sim 100\,\mathrm{mmHg} = 1.3 \times 10^4\,\mathrm{N/m^2}$. (This averaging is different from that in (8.1) because of the simplicity of this model. This leads to an ∼20% uncertainty.) We take $V_\mathrm{stroke} = 80\,\mathrm{cm^3} = 8 \times 10^{-5}\,\mathrm{m^3}$, so $W = (1.3 \times 10^4\,\mathrm{N/m^2})(8 \times 10^{-5}\,\mathrm{m^3}) = 1.04\,\mathrm{J}$ per cycle. With a heart rate of 60/min = 1/s, the rate the left ventricle does work is $P_\mathrm{power,mech,av} = (1.04\,\mathrm{J}$ per cycle$)(1\,\mathrm{cycle/s}) = 1.04\,\mathrm{W}$.

The efficiency, ϵ, of converting metabolic energy into this mechanical work is approximately 20% (and sometimes this range is given as 12–30%), and so the metabolic power needed to run the left ventricle is $P_\mathrm{power,metab,av} = P_\mathrm{power,mech,av}/\epsilon = 5$ W.

The heart pumps for about 1/3 of the cardiac cycle and rests for the other 2/3 of the time. Therefore the peak powers are higher than these average values by a factor of 3, with $P_\mathrm{power,mech,peak} = 1.5\,\mathrm{W}$ and $P_\mathrm{power,metab,peak} = 15\,\mathrm{W}$.

The energy consumed to run the left ventricle is $(86{,}400\,\mathrm{s/day})(5\,\mathrm{W}) = 4.32 \times 10^5\,\mathrm{J/day} = 104$ kcal/day. So far we examined the work done by only the left ventricle. The right ventricle pumps the same volume per cardiac cycle (to maintain the steady-state flow throughout), but at a pressure 1/5 times that of left ventricle, so the work and all of these powers are smaller by a factor of five. This increases the required metabolic power by 20%. Similarly, the pressures for the two atria are also relatively very small. Overall, with 20% muscle efficiency we expect to need ∼125 kcal/day to run the heart; with 10% muscle efficiency it would be ∼250 kcal/day.

The experimental value for the BMR contribution for the entire heart is ∼117 kcal/day (Table 6.17), which is close to our estimate. The biggest uncertainty here is the efficiency of the cardiac muscle.

What happens with strenuous exercise? The blood pressure can increase by 50% and the blood flow rate can increase by a factor of 5. Therefore, the mechanical power exerted by the heart and the associated metabolic requirements can increase by a factor of 7.5.

8.3 Strokes and Aneurysms

Cerebral blood flows from the internal carotid and vertebral arteries and through the circle of Willis at the base of the brain (Fig. 8.39), and then permeates the brain through a complex series of capillaries.

Any severe restriction of blood to the brain is called a *stroke* [391, 393]. *Ischemia* means there is a lack of blood flow. The nearby tissue becomes deficient in oxygen and metabolites, and has excessive metabolic waste products. (We saw an example of ischemia earlier this chapter with the Toscanini TIA. Because TIA is transient, there is an oxygen deficiency due to the stroke but the patient can still recover with little or no brain damage.) *Hypoxia* means a lack of oxygen, and it can result from ischemia or other causes, such as high altitude (see Chap. 9) or CO poisoning. *Infarction* means that the stroke causes permanent brain damage. (The terms *ischemia* and *infarction* are actually more general and also apply to tissues outside the brain, with a myocardial infarction in the heart as one example.) The transition from the reversible event to the irreversible infarct with the formation of necrotic tissue occurs when the stroke is particularly long or of particularly large magnitude. (Necrotic tissue is dead tissue that did not die in a manner programmed by the body, which is in contrast to apoptotic tissue which the body kills as part of the life cycle.) The occurrence of a transient ischemic attack, or ministroke, sometimes means a more damaging stroke is imminent.

Hemorrhagic strokes are due to a ruptured vasculature (blood vessels) within the brain, attributed to an aneurysm or weakened blood vessel (Fig. 8.41). An *aneurysm* is an enlarged blood vessel. In addition to the loss

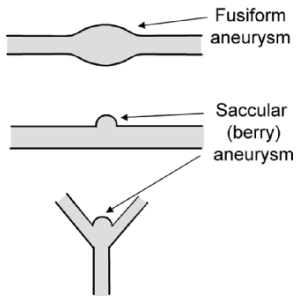

Fig. 8.41. Fusiform and saccular/berry aneurysms, the latter in a vessel and for bifurcated flow

of blood to the targeted regions, during hemorrhages blood fills the surrounding spaces and compresses the surrounding tissues. This accumulating blood compresses other blood vessels, decreasing their diameters and reducing the flow of blood to other parts of the brain, and increases the intracranial pressure, which leads to neurological complications. About 20% of all strokes are hemorrhagic; they occur mostly in the young and middle-aged, due to vascular *lesions* such as arteriovenous malformations and aneurysms. (Lesions are entities of diseased or abnormal tissue.) In the elderly, blood vessels are brittle and less distensible due to atherosclerotic deposits, and this can lead to possible spontaneous rupture of these vessels in the brain and hemorrhagic stroke. In *atherosclerosis* ("hardening of the arteries") lipid or fatty deposits in the blood accumulate on the inner vessel wall and eventually form hard arterial plaques.

During an *ischemic stroke* there is cessation of blood flow in arteries transporting blood to the brain due to a luminal obstruction or clogging. (As alluded to earlier, the *lumen* is the opening of a blood vessel.) About 80% of all strokes are ischemic. An *embolus* is a gaseous (air bubble), particulate matter, or blood clot that travels within a blood vessel and causes the obstruction of blood flow. For example, artherosclerotic lesions (in the brain and elsewhere in the body) cause an irregular inner vessel surface and blood platelet aggregation due to turbulence, that can produce emboli that are platelet aggregates. Such emboli can be formed outside the brain, in the heart, lungs, and systemic circulation, and travel to the brain until they reach vessels too small for further travel; this prevents blood flow to more distal (downstream) regions in the brain. A *thrombus* is blood coagulation that can produce a local fibrin clot; this can also cause an ischemic stroke. (If the thrombus forms and moves elsewhere, such as to a smaller diameter vessel or a partially occluded vessel, it is an embolus.)

The majority of cerebral aneurysms are *saccular (or berry) aneurysms* that most often occur where large cerebral arteries bifurcate (Figs. 8.42 and 8.43). In *fusiform aneurysms* there is uniform ballooning of the circumference of the vessel walls, instead of in localized regions of the vessels as in saccular aneurysms; this leads to ellipsoidal or football shaped aneurysms. (Fusiform means tapering at each end. Here it indicates a cylindrically symmetric aneurysm that tapers to the normal vessel at either end, while for the fusiform muscles in Chap. 5 it indicates a cylindrically symmetric muscle that tapers to tendons on each end.) Fusiform aneurysms are less common in the brain than are saccular aneurysms, but are common elsewhere in the body. The abdominal aortic aneurysm, which develops along the aorta in the abdominal or gut region, is the most common aneurysm found in the body and is a fusiform aneurysm (Fig. 8.44).

Healthy arteries contain the structural proteins elastin and collagen. Collagen has the larger Young's modulus (Table 4.2) and is expected to dominate the elastic properties of arteries. Still, the elastin contributes to the distensibility of the artery. The resistance to stretching at low pressures seems to be due to the elastin fibers, at normal physiological pressures it is due to elastin

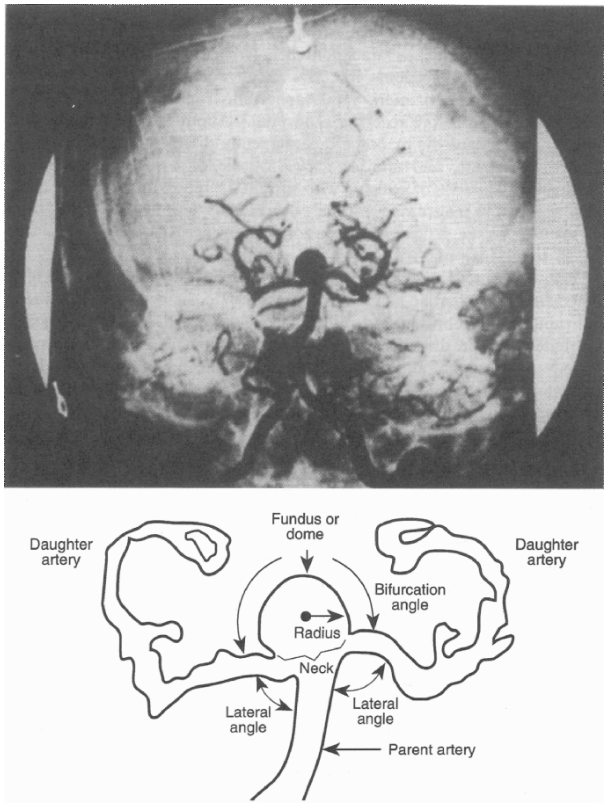

Fig. 8.42. Saccular (berry) aneurysm at an apex of a branching vessel, showing an angiographic projectional image (*top*) and a model (*bottom*). (From [391])

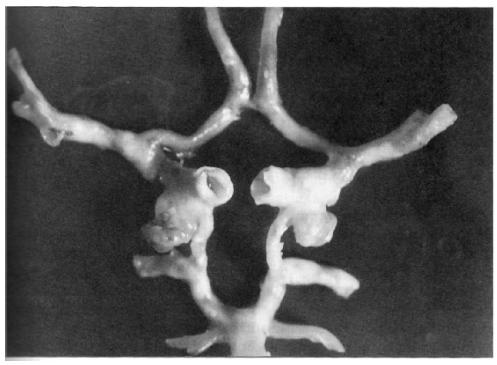

Fig. 8.43. Photograph of an inferior view of an excised human circle of Willis. Bilateral (i.e., on both sides) saccular aneurysms are seen near the junction between the internal carotid artery and the circle; the larger lesion (the one on the *left side* of the photograph) had ruptured. (From [395])

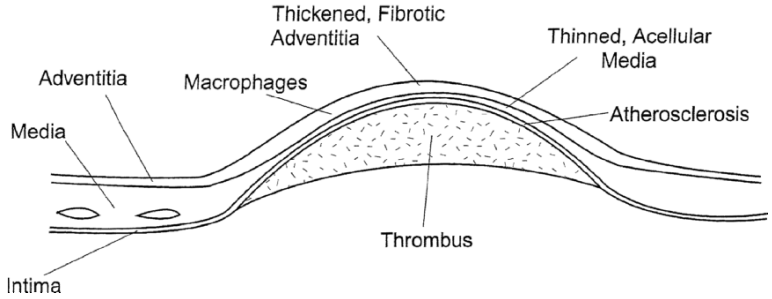

Fig. 8.44. Schematic of an abdominal aortic aneurysm, showing an attenuated media and an intraluminal thrombus. (From [395])

and collagen fibers, and at even higher pressures it is due to collagen. When the artery wall balloons or sacculates (i.e., it balloons in one circumferential part of the wall) as the aneurysm develops, the elastin becomes less effective in maintaining structural integrity of the artery and the collagen takes on most of the load. (The artery becomes less distensible and this translates to greater stress for the same strain, thereby accelerating structural fatigue.) This process accelerates the load on the arterial wall, and leads to rupture. A possible scenario for the formation and rupture of a saccular aneurysm is shown in Fig. 8.45. Figure 8.46 shows the equilibrium circumferential tension for a vessel assuming the Law of Laplace, for a normal artery, and one with an aneurysm. Figure 7.11 shows that the maximum blood flow velocity is in the center, where the wall shear stress is minimum. The minimum blood flow velocity is near the wall, where the wall shear stress is maximum.

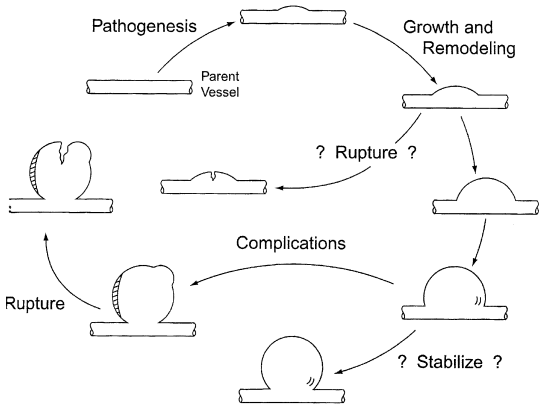

Fig. 8.45. A possible natural history for the development of a saccular aneurysm. A local weakening of the vessel wall, leading to a mild dilatation, can be caused by an initial "insult" from one of several causes. (From [395])

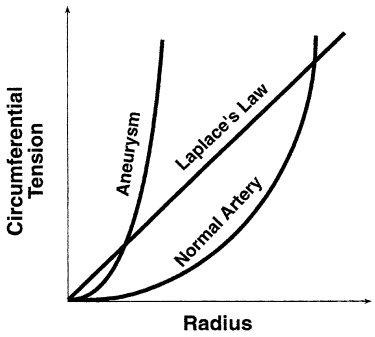

Fig. 8.46. Circumferential tension needed for equilibrium as a function of vessel radius, assuming the Law of Laplace, a normal artery, and an artery with an aneurysm. (From [391, 392])

There are (at least) four physical reasons why the larger radius of a fusiform aneurysm can lead to rupture. All are related to the stress relation $T/w = r(\Delta P)/w$, where w = wall thickness and r is local vessel radius. (1) r is larger so more tension is needed to withstand even an unchanged pressure difference (because $T = r(\Delta P)$). (2) This increase in vessel radius can be accompanied by thinning walls (if the volume of vessel wall per unit length, $\sim 2\pi rw$, is relatively unchanged by the aneurysm), so the stress T/w increases even more. (3) With this wall thinning there may be damage that lowers the UTS locally, and the UTS needs to be $\gg T/w$ to avoid rupture. (4) A larger r, and the concomitant larger cross-sectional area A, leads to a slower blood speed u, through volumetric continuity (7.16). This in turn leads to a larger pressure P, through Bernoulli's equation (7.18). This increases the tension that the vessel must withstand. The magnitudes of several of these effects are evaluated in Problem 8.32.

8.3.1 Arterial Bifurcations and Saccular Aneurysms

Two of the reasons for the formation of saccular aneurysms are the forces on the arterial walls caused by the change of momentum (like the fire hose effect in curving arteries) and shear stress. Figure 8.47 depicts a "parent" artery with cross-sectional area A_1 in which blood flows at an average speed u_1, which divides into two "daughter" vessels at an angle θ to the parent (and 2θ to each other). (The half-angle is shown as θ_2 in the figure.) Each daughter vessel has cross-sectional area A_2 and blood speed u_2. The angle 2θ usually ranges from 30 to 120° (also see Problems 8.28–8.31). The apex of the bifurcation (Fig. 8.47) is the site of maximum stress due to the impact, deflection, and separation of the flow, and possible turbulence and vortex formation (Fig. 8.48). Conservation of flow rate Q (volume flow/time)

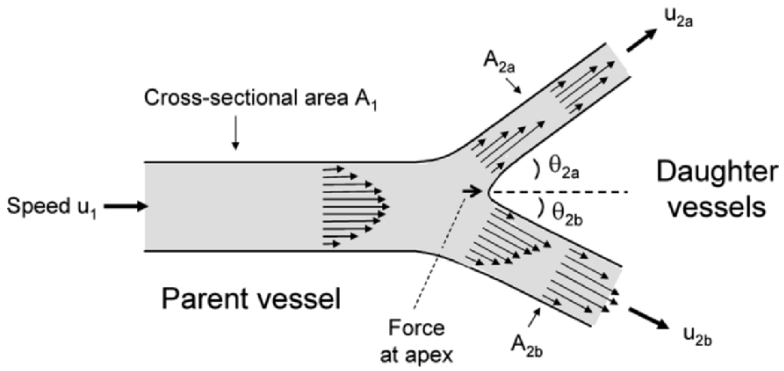

Fig. 8.47. Diagram of asymmetric bifurcation in vessel, with bifurcation angle $\theta_{2a} + \theta_{2b}$. For the symmetric bifurcation described in the text the "a" and "b" labels can be ignored, and the half-angle is called θ. (See Problems 8.28–8.31)

means

$$Q = u_1 A_1 = 2u_2 A_2. \tag{8.40}$$

In these vessels, usually $2A_2 > A_1$, so $u_2 < u_1$.

How large is the force on the arterial wall at the apex? The linear momentum per unit volume of blood in the parent artery (which we will say is in the x direction) is ρu_1, where ρ is the blood mass density. The momentum per unit volume carried in each daughter artery is ρu_2, of which $\rho u_2 \cos\theta$ is along the x direction. This change in momentum causes a force on the arterial wall. The force this flow exerts on an imaginary screen across the vessel in the parent artery is the change of this momentum per unit time, which equals this linear momentum per unit volume $\times$ the flow rate

$$F_{z,\text{parent}} = \rho u_1 Q. \tag{8.41}$$

Because the flow rate in each vessel is $Q/2$ and there are two of them, the force of the flow in the daughter arteries is

$$F_{z,\text{daughters}} = 2\,\frac{\rho u_2 \cos\theta\, Q}{2} = \rho u_2 \cos\theta\, Q. \tag{8.42}$$

The difference of these forces is

$$\begin{aligned} F_{\text{arterial wall}} &= F_{z,\text{daughters}} - F_{z,\text{parent}} \\ &= \rho u_2 \cos\theta\, Q - \rho u_1 Q = \rho Q(u_2 \cos\theta - u_1) \end{aligned} \tag{8.43}$$

or with (8.40)

$$F_{\text{arterial wall}} = \rho Q u_1 \left(\frac{A_1}{2A_2}\cos\theta - 1\right) = \rho A_1 u_1^2 \left(\frac{A_1}{2A_2}\cos\theta - 1\right). \tag{8.44}$$

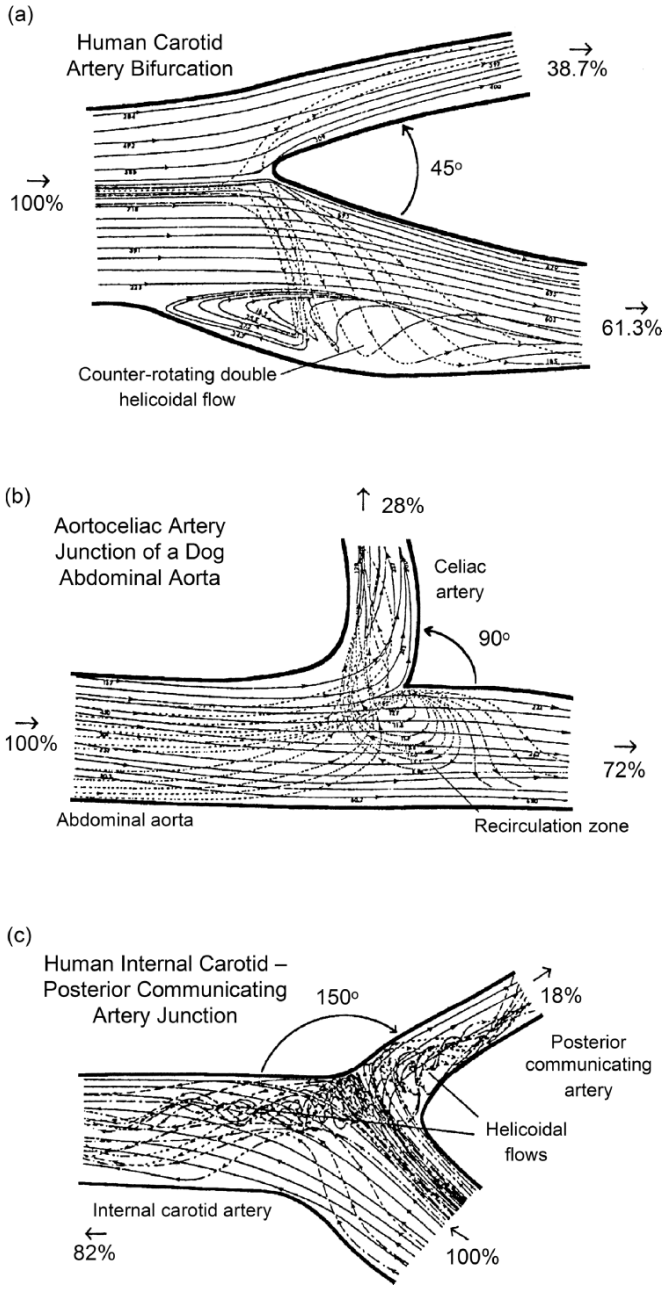

Fig. 8.48. Disturbed flow streamlines for progressively large angle bifurcations at bifurcations, and approach to steady flow afterward. (From [382], based on (**a**) [409], (**b**) [397], and (**c**) [398]. Courtesy of Robert A. Freitas Jr., Nanomedicine, Vol. 1 (1999), http://www.nanomedicine.com)

This has a maximum negative value of $-\rho Q u_1 = -\rho A_1 u_2^2$ when $\theta = 90°$ (which is a bit larger than the typical maximum angles). Because pressure is force/area, if this force is exerted on the vessel wall of cross-sectional area A_1, as in Fig. 8.42, there is a pressure on the arterial wall of

$$P_{\text{arterial wall}} = \frac{\rho Q u_1}{A_1} = \frac{\rho A_1 u_1^2}{A_1} = \rho u_1^2 \tag{8.45}$$

(which is also the kinetic energy per unit volume). This can lead to the formation of an aneurysm there. This mechanism of momentum change is the same as that causing a force on an arterial wall during flow in a curving artery. The saccular aneurysm gets larger and larger until it ruptures.

The shear stress near the bifurcation apex due to viscosity is another factor in the formation of a saccular aneurysm. Figure 8.47 shows that in steady flow the velocity is small near the walls and the velocity gradient and consequently the shear stress is large near the walls. After bifurcation and momentum transfer to the apex, the blood velocity, velocity gradient, and shear are larger near the vessel walls after the apex. The shear stress is large, until the flow pattern rearranges to give (7.40) (Fig. 7.11). This is also seen in Fig. 8.48 for bifurcations at increasingly large angles. Equation (7.41) can be used to estimate the distance from the apex for steady-state flow. Note that the Law of Laplace for spheres applies to saccular aneurysms.

8.3.2 Stenosis and Ischemic Strokes

Strokes can also occur by *stenosis* (narrowing or closure of lumens) or *occlusions* (closures or obstructions) (Fig. 8.49). They are most commonly due to artherosclerotic lesions. Such lesions are irregularly distributed masses of calcified fatty deposits that narrow the arterial lumen. If the normal inner diameter of the vessel is d_{norm} and the minimum diameter due to the stenotic lesion is d_{sten}, then the % stenosis is defined as: $((d_{\text{norm}} - d_{\text{sten}})/d_{\text{norm}}) \times 100\%$. They are characterized as being mild (1–39%), moderate (40–59%), severe (60–79%), critical (80–99%), and occluded (100%). Poiseuille's Law (7.24) shows that the flow decreases with decreasing lumen diameter for a given initial pressure. Flow decreases dramatically above the onset of critical stenosis, and the pressure drop across the stenosis increases, resulting in a need for a greater blood pressure to maintain the same flow rate.

This calcified lesion stiffens the vessel and abruptly changes the flow pattern as the blood flows from an elastic, distensible region of a vessel to this rigid and narrower region and back to a distensible vessel. Overall blood flow is slower because of the stenosis (unless the pressure increases), which can lead to clotting. The resulting clot (or thrombus) does not adhere well to the vessel wall and can move to elsewhere in the flow stream and this embolus can lead to a stroke. Within the stenosis itself, the blood flow is faster than just before it, from Bernoulli's Principle. This increased blood flow has

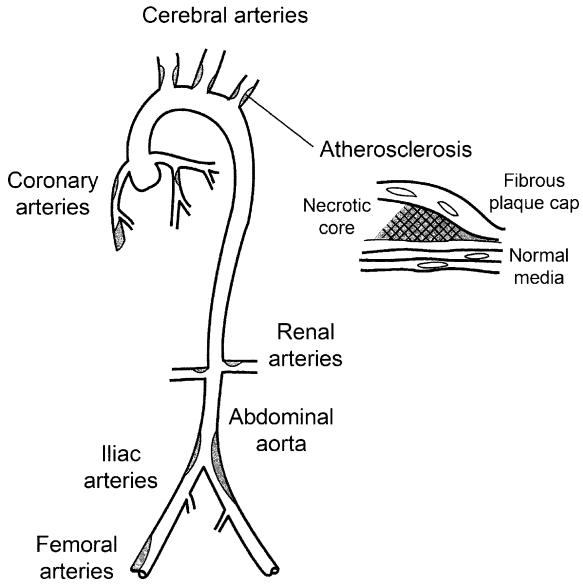

Fig. 8.49. Clogging of arteries by plaque. A schematic of the atherosclerosis is shown, along with some of the preferred sites of atherosclerosis in the vasculature (blood vessels). (From [395])

several negative consequences. As this increased kinetic energy (per unit volume) in the stenotic region decreases in the poststenotic normal vessel (due to Bernoulli's Principle), there can be structural fatigue in this latter region leading to distention and this possible dilatation can lead to a fusiform aneurysm. The increased stenotic flow speed and irregular geometry can increase the Reynolds number, resulting in turbulent flow and the eventual disengaging of arterial plaque, which then becomes a particulate embolus. The increased speed also leads to higher shear stresses on the lesion.

8.3.3 Equation of Motion of Arteries and Aneurysms during Pulsatile Flow (Advanced Topic)

For steady flow, the tension in the arterial wall balances the pressure difference inside and outside the artery, as described by the Law of Laplace. Because the pressure inside the artery really varies within every cycle (during the arterial pulse), this balance needs to be evaluated to account for these cyclic pressure variations. Let us model a saccular aneurysm as a sphere of radius R, with a thin wall of thickness w. (We could similarly model a fusiform aneurysm as a cylinder of radius R.)

If the change in radius due to this time-varying pressure is r, then the circumference of the sphere changes from $2\pi R$ to $2\pi(R+r)$. The circumferential

strain ϵ is $(2\pi(R+r)-2\pi R)/2\pi R = r/R$, and the circumferential stress σ is related to this strain by

$$\sigma = Y\epsilon = Y\frac{r(t)}{R}. \tag{8.46}$$

The response of the arterial wall has a component that balances the average pressure $P_{\rm av}$ – which is a time-weighted average of the systolic and diastolic pressures for larger arteries – and a time varying component – due to the pulse $P_{\rm pulse}(t) = P_{\rm p}\cos\omega_{\rm p}t$, where $P_{\rm p}$ is a time-weighted difference of the systolic and diastolic pressures for larger arteries and $\omega_{\rm p}$ is the pulse (radial) frequency. This time-varying pressure is $P(t) = P_{\rm ss} + P_{\rm p}\cos\omega_{\rm p}t$.

The inertial response force ma, per unit area on the sphere is the mass per unit area of the spherical shell, ρw, where ρ is the mass density, times the radial acceleration, $\mathrm{d}^2r/\mathrm{d}t^2$, or $\rho w\,\mathrm{d}^2r/\mathrm{d}t^2$. The hydrostatic pressure term $P(t)$ tends to increase the radius, while the stress due to the wall resists this change, so the inertial response is

$$\rho w\frac{\mathrm{d}^2r_{\rm p}(t)}{\mathrm{d}t^2} = P_{\rm av} + P_{\rm p}\cos\omega_{\rm p}t - Y\frac{r(t)}{R} \tag{8.47}$$

because $\mathrm{d}^2r_{\rm av}/\mathrm{d}t^2 = 0$.

The perturbation $r(t)$ has a component in response to the average pressure $P_{\rm av}$ and a time-varying part in response to the pulse pressure $P_{\rm p}\cos\omega_{\rm p}t$. Substituting $r(t) = r_{\rm av} + r_{\rm p}(t)$ into (8.47), we get

$$\rho w\frac{\mathrm{d}^2r_{\rm p}(t)}{\mathrm{d}t^2} = P_{\rm av} + P_{\rm p}\cos\omega_{\rm p}t - Y\frac{r_{\rm av}+r_{\rm p}(t)}{R}. \tag{8.48}$$

Equating the time-varying terms to give one equation and the average terms to give another leads to

$$\rho w\frac{\mathrm{d}^2r_{\rm p}(t)}{\mathrm{d}t^2} = P_{\rm p}\cos\omega_{\rm p}t - Y\frac{r_{\rm p}(t)}{R} \tag{8.49}$$

and

$$0 = P_{\rm av} - Y\frac{r_{\rm av}}{R}. \tag{8.50}$$

Equation (8.50) is related to the Law of Laplace for a sphere (7.9) ($\Delta P = 2T/R$). Equation (8.49) can be rewritten as

$$\frac{\mathrm{d}^2r_{\rm p}(t)}{\mathrm{d}t^2} + \omega_0^2 r_{\rm p}(t) = \frac{P_{\rm p}}{\rho w}\cos\omega_{\rm p}t, \tag{8.51}$$

where $\omega_0^2 = Y/R\rho w$. This looks like the equation of motion for a simple harmonic oscillator of frequency ω_0 plus an extra term (the last one), which is due to the pulse driving force at frequency $\omega_{\rm p}$. The steady-state solution to

this equation is

$$r_{\mathrm{p}}(t) = \frac{P_{\mathrm{p}}/\rho w}{\omega_0^2 - \omega_{\mathrm{p}}^2} \cos(\omega_{\mathrm{p}} t), \tag{8.52}$$

which can be verified by substitution (see Appendix C). If r_{p} is large, it could lead to rupture. In principle, r_{p} can become larger as the aneurysm develops because Y, R, ρ, or w change (in $\omega_0^2 = Y/R\rho w$). Without the driving term ($P_{\mathrm{p}} = 0$), the solution is the usual harmonic solution: $r_{\mathrm{p}}(t) = A\cos(\omega_0 t + \phi)$.

If ω_0 were to approach ω_{p}, (8.52) indicates that the change in radius would become very large because of this resonance. If this were to occur (and it does in some examples of driven oscillators but it really does not for aneurysms), the viscous (or damping) properties of the vessel wall would have to be included through a term $-\gamma\, \mathrm{d}r_{\mathrm{p}}/\mathrm{d}t$, leading to the new equation of motion

$$\frac{\mathrm{d}^2 r_{\mathrm{p}}(t)}{\mathrm{d}t^2} + \gamma \frac{\mathrm{d}r_{\mathrm{p}}(t)}{\mathrm{d}t} + \omega_0^2 r_{\mathrm{p}}(t) = \frac{P_{\mathrm{p}}}{\rho w} \cos \omega_{\mathrm{p}} t \tag{8.53}$$

with steady-state solution

$$r_{\mathrm{p}}(t) = \frac{\left(\omega_0^2 - \omega_{\mathrm{p}}^2\right) P_{\mathrm{p}}/\rho w}{\left(\omega_0^2 - \omega_{\mathrm{p}}^2\right)^2 + (\gamma\omega_{\mathrm{p}})^2} \cos(\omega_{\mathrm{p}} t). \tag{8.54}$$

(See Appendix C.) This now includes the viscoelastic properties of the arterial wall, which dampens the resonance a bit.

Far above the $\omega_0 \simeq \omega_{\mathrm{p}}$ resonance (with $\omega_0 \gg \omega_{\mathrm{p}}$), (8.52) and (8.54) give

$$r_{\mathrm{p}}(t) \simeq \frac{P_{\mathrm{p}}}{\rho w \omega_0^2} \cos(\omega_{\mathrm{p}} t). \tag{8.55}$$

Without the driving term ($P_{\mathrm{p}} = 0$), the solution is that of a damped harmonic oscillator: $r_{\mathrm{p}}(t) = A\exp(-\gamma t/2)\cos(\omega_0 t + \phi)$, for $\omega_0 \gg \gamma$. This harmonic oscillation damps in a time $\sim 1/\gamma$, which corresponds to about $\omega_0/(2\pi\gamma)$ cycles; this last number is often called the quality factor Q of the system, as is discussed in the Chap. 10 discussion of acoustic resonances and in Appendices C and D.

8.4 Modeling the Circulatory System and the Heart

The branching in the circulatory system is very complex. Still, there is an orderly transition from larger to smaller arteries and then from smaller to larger veins, and so we can imagine an overall model of the circulation with all arteries or veins of a given diameter combining to form a subsystem. We have seen that larger vessels can be modeled quite well as ideal compliance vessels, while smaller vessels can be modeled as resistance vessels. We will now use these models to develop a comprehensive model of circulation, which can

handle steady-state flow and changes that depend on time, like the arterial pulse. It can be used to understand the control of circulation. For a complete circulation model, we need to include the action of the heart, which is really two separate pumps: the right heart for the pulmonary system and the left heart for the systemic system. We first develop static and dynamic models of the left and right hearts.

8.4.1 Model of the Heart

Let us consider the left ventricle, which is the major pump in the left heart; the treatment of the right heart is analogous. During systole, the mitral valve is closed and the aortic valve is open. The pressure that develops is essentially that in the systemic arteries (sa) P_{sa} because of the very small pressure drop. During diastole the aortic valve is closed and the mitral valve is open. The left ventricle receives blood from the left atrium at a pressure that is pretty low, and is essentially equal to that in the pulmonary veins (pv), $P_{\mathrm{pv}} \sim 5\,\mathrm{mmHg}$, that feeds the left atrium.

Static Model of the Ventricles

We will model the left (or right) ventricle as a hemispherical shell, with an inner radius r_{i} and outer radius r_{o}, with a very thick wall of thickness $r_{\mathrm{o}} - r_{\mathrm{i}}$. Assume the open side is facing upward, as in Fig. 8.50. The (gauge) pressure inside P_{i} pushes the ventricle down and reaches a maximum during systole of 120 mmHg. The pressure outside (acting on the round bottom surface) P_{o} is from the pericardium and pushes the ventricle up and is approximately the pleural pressure, which can be negative (i.e., less than an atmosphere). The circumferential wall stress σ acts vertically and pushes the ventricle up. These arguments are the same as those for the Law of Laplace for a sphere (7.9), except we are now assuming the wall has finite thickness.

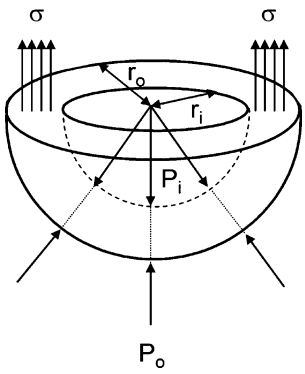

Fig. 8.50. Model of the left ventricle. (Based on [405])

Force balance in the vertical direction gives

$$(\pi r_{\mathrm{i}}^2)P_{\mathrm{i}} = (\pi r_{\mathrm{o}}^2)P_{\mathrm{o}} + \left(\pi(r_{\mathrm{o}}^2 - r_{\mathrm{i}}^2)\right)\sigma \tag{8.56}$$

and so the circumferential wall stress is

$$\sigma = \frac{P_{\mathrm{i}} r_{\mathrm{i}}^2 - P_{\mathrm{o}} r_{\mathrm{o}}^2}{r_{\mathrm{o}}^2 - r_{\mathrm{i}}^2}. \tag{8.57}$$

The first two terms in (8.56) are not just the surface areas of the respective areas, $2\pi r_{\mathrm{i,o}}^2$, times the hydrostatic pressure, $P_{\mathrm{i,o}}$, but half of that, because only part of the force due to the pressure is in the vertical direction (as in the Law or Laplace for spheres and Problem 7.12). Because $P_{\mathrm{i}} \gg P_{\mathrm{o}}$

$$\sigma \simeq \frac{P_{\mathrm{i}}}{(r_{\mathrm{o}}/r_{\mathrm{i}})^2 - 1}. \tag{8.58}$$

Because the material in the heart wall is incompressible, the volume of the heart wall V_{wall} does not change with pressure. From the difference of the volumes of the outer and inner hemispherical shells, we know that $V_{\mathrm{wall}} = 2\pi(r_{\mathrm{o}}^3 - r_{\mathrm{i}}^3)/3$ and so $(r_{\mathrm{o}}/r_{\mathrm{i}})^2 = (1 + V_{\mathrm{wall}}/V_{\mathrm{i}})^{2/3}$, where the inner volume of the left ventricle is $V_{\mathrm{i}} = 2\pi r_{\mathrm{i}}^3/3$. Therefore we see

$$\sigma \simeq \frac{P_{\mathrm{i}}}{(1 + V_{\mathrm{wall}}/V_{\mathrm{i}})^{2/3} - 1}. \tag{8.59}$$

Using the expansion $(1 + x)^n \simeq 1 + nx$ for $\mid x \mid \ll 1$ and the fact that the internal volume of the left ventricle is much larger than the volume of the heart wall, we find

$$\sigma \simeq \frac{3V_{\mathrm{i}}}{2V_{\mathrm{wall}}}\, P_{\mathrm{i}}. \tag{8.60}$$

This shows how excessive systolic pressure or the enlargement of the left ventricle (and of the heart) will lead to excessive cardiac wall stress (for a constant wall thickness).

Dynamic Model of the Ventricles

We will now model a ventricle as a compliance vessel with a compliance that changes with time [394], so we use (8.5)

$$V(t) = V_{\mathrm{d}} + C(t)P(t). \tag{8.61}$$

(We will simply call the flow resistance R_{flow} and compliance C_{flow}, respectively R and C in this section.) Figure 8.51 shows how the compliance of the ventricle changes during a cardiac cycle. During systole the compliance becomes low, which causes a high pressure to develop because the volume

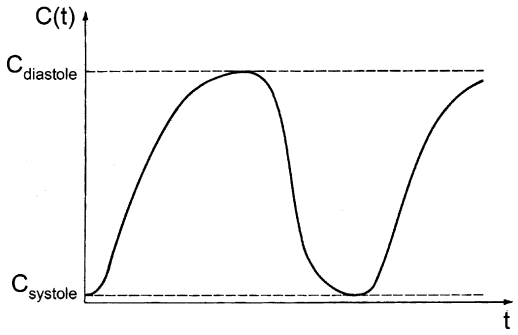

Fig. 8.51. Changes in ventricle compliance during the cardiac cycle, from a small value while it is contracting during systole to a large value when it is relaxing during diastole. (From [394])

remains pretty constant. In diastole the ventricle relaxes, which is associated with a large compliance that induces a low pressure.

The changes of volume and pressure in the ventricle during a cardiac cycle are modeled in Fig. 8.52. Stage A is when the inflow valve (which is the mitral valve for the LV) closes, which marks the end of diastole (ED) and the beginning of systole. The ventricle volume is a maximum

$$V_{\mathrm{ED}} = V_{\mathrm{d}} + C_{\mathrm{diastole}} P_{\mathrm{pv}}. \tag{8.62}$$

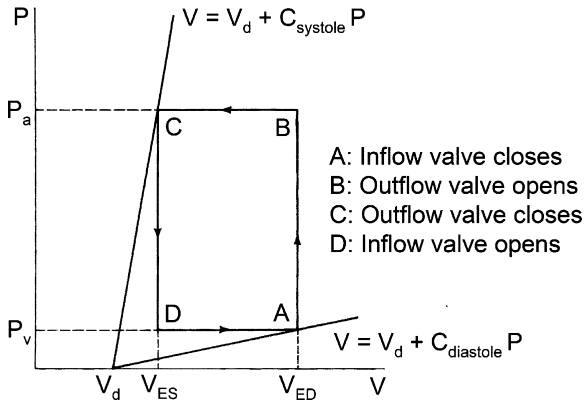

Fig. 8.52. Cycle of pressure and volume in either ventricle is given by the ABCD rectangle in this simplified model. The slanting lines radiating from the dead volume are the ventricle pressure–volume relationships at the end of systole and diastole. (Similar slanting lines with different slopes (not shown) characterize the ventricle at other times.) The venous pressure is the same as the inflow atrial and venous pressures during diastole. The arterial pressure is the outflow ventricle pressure during systole. (From [394])

During systole, the compliance decreases isovolumetrically, so the pressure increases. At Stage B, the outflow valve (which is the aortic valve for the LV) opens; the pressure remains constant as blood leaves the ventricle and concomitantly the volume of the ventricle decreases. Stage C is when the outflow valve closes, which marks the end of systole (ES) and the beginning of diastole. The ventricle volume is a minimum:

$$V_{\mathrm{ES}} = V_{\mathrm{d}} + C_{\mathrm{systole}} P_{\mathrm{sa}}. \tag{8.63}$$

During diastole, the compliance increases isovolumetrically, so the pressure decreases. At Stage D, the inflow valve opens; the pressure then remains constant as blood enters the ventricle and concomitantly the volume of the ventricle increases – until the end of diastole is reached, Stage A again.

The stroke volume is

$$V_{\mathrm{stroke}} = V_{\mathrm{ED}} - V_{\mathrm{ES}} = C_{\mathrm{diastole}} P_{\mathrm{pv}} - C_{\mathrm{systole}} P_{\mathrm{sa}}. \tag{8.64}$$

Because $C_{\mathrm{systole}} \sim 0$, we can take

$$V_{\mathrm{stroke}} = C_{\mathrm{diastole}} P_{\mathrm{pv}}. \tag{8.65}$$

With a heart rate F, the volumetric flow is

$$Q = F V_{\mathrm{stroke}} = F C_{\mathrm{diastole}} P_{\mathrm{pv}}. \tag{8.66}$$

Calling $K = F C_{\mathrm{diastole}}$ the pump coefficient, we can model the left ventricle by

$$Q_{\mathrm{L}} = K_{\mathrm{L}} P_{\mathrm{pv}}. \tag{8.67}$$

Analogously for the right heart, the right ventricle, which is fed by the systemic veins (sv), is modeled by

$$Q_{\mathrm{R}} = K_{\mathrm{R}} P_{\mathrm{sv}}. \tag{8.68}$$

8.4.2 Model of the Overall Flow in the Circulatory System

We will model the eight subsystems shown in Fig. 8.53, two hearts (L and R), large arteries in the systemic and pulmonary systems (sa and pa), large veins in the systemic and pulmonary systems (sv and pv), and the small vessels (arterioles/capillaries/venules) in the systemic and pulmonary systems [394]. Blood flows from the left ventricle successively through the systemic large arteries, small vessels, and large veins, and to the right ventricle. Then blood goes from the right ventricle successively through the pulmonary large arteries, small vessels, and large veins, and to the left ventricle.

The flows through the ventricles are determined by the pressures in the veins feeding them (through the atria)

$$Q_{\mathrm{L}} = K_{\mathrm{L}} P_{\mathrm{pv}} \qquad \text{and} \qquad Q_{\mathrm{R}} = K_{\mathrm{R}} P_{\mathrm{sv}}. \tag{8.69}$$

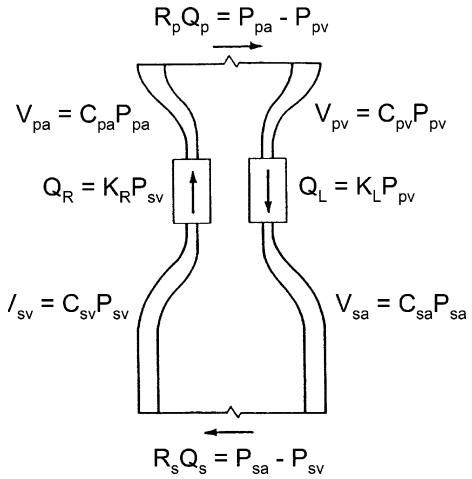

Fig. 8.53. The eight subsystems in systemic and pulmonary flow, with their model equations. (From [394])

The volumes in the large arteries are determined by their compliances and the pressures within them

$$V_{\mathrm{sa}} = C_{\mathrm{sa}} P_{\mathrm{sa}} \qquad \text{and} \qquad V_{\mathrm{pa}} = C_{\mathrm{pa}} P_{\mathrm{pa}}, \tag{8.70}$$

as are the volumes of the veins

$$V_{\mathrm{sv}} = C_{\mathrm{sv}} P_{\mathrm{sv}} \qquad \text{and} \qquad V_{\mathrm{pv}} = C_{\mathrm{pv}} P_{\mathrm{pv}}. \tag{8.71}$$

(A more refined model would include the dead volume in these large vessels.) The pressure drop across the small vessels is the difference in pressure between the large arteries and large veins. For the respective systemic and pulmonary systems, the pressure drop is determined by their resistances (R_{s} and R_{p}) and the flows through them (Q_{s} and Q_{p})

$$P_{\mathrm{sa}} - P_{\mathrm{sv}} = R_{\mathrm{s}} Q_{\mathrm{s}} \qquad \text{and} \qquad P_{\mathrm{pa}} - P_{\mathrm{pv}} = R_{\mathrm{p}} Q_{\mathrm{p}}. \tag{8.72}$$

We see a symmetry here. There are three types of variables, the flow Q, volume V, and pressure P. Each subsystem depends on relations between two of them: Q and P for the ventricles, V and P for the large vessels, and P and Q for the small vessels.

There are 12 unknowns: four flows: Q_{R}, Q_{L}, Q_{s}, Q_{p}; four pressures: P_{sa}, P_{sv}, P_{pa}, P_{pv}; and four volumes: V_{sa}, V_{sv}, V_{pa}, V_{pv}. We need 12 equations to solve for these 12 unknowns, but have only eight here and need four more. However, for steady-state flow, the flow in each region is the same, so $Q_{\mathrm{R}} = Q_{\mathrm{L}} = Q_{\mathrm{s}} = Q_{\mathrm{p}}$. These are really the three independent equations

$$Q_{\mathrm{R}} = Q_{\mathrm{L}}, \qquad Q_{\mathrm{R}} = Q_{\mathrm{s}}, \qquad \text{and} \qquad Q_{\mathrm{R}} = Q_{\mathrm{p}}, \tag{8.73}$$

with each flow rate equal to Q. The fourth equation describes the constant total volume and is

$$V_0 = V_{\rm sa} + V_{\rm sv} + V_{\rm pa} + V_{\rm pv}. \tag{8.74}$$

We can now solve these equations. From the pump (8.67) and (8.68), the venous pressures are

$$P_{\rm sv} = \frac{Q}{K_{\rm R}} \qquad \text{and} \qquad P_{\rm pv} = \frac{Q}{K_{\rm L}}. \tag{8.75}$$

Inserting these venous pressures into the resistance (8.72) gives

$$P_{\rm sa} = \frac{Q}{K_{\rm R}} + R_{\rm s}Q \qquad \text{and} \qquad P_{\rm pa} = \frac{Q}{K_{\rm L}} + R_{\rm p}Q. \tag{8.76}$$

Inserting these venous and arterial pressures into the compliance (8.70) and (8.71) gives

$$V_{\rm sa} = QC_{\rm sa}\left(\frac{1}{K_{\rm R}} + R_{\rm s}\right) \qquad \text{and} \qquad V_{\rm pa} = QC_{\rm pa}\left(\frac{1}{K_{\rm L}} + R_{\rm p}\right) \tag{8.77}$$

$$V_{\rm sv} = QC_{\rm sv}\left(\frac{1}{K_{\rm R}}\right) \qquad \text{and} \qquad V_{\rm pv} = QC_{\rm pv}\left(\frac{1}{K_{\rm L}}\right). \tag{8.78}$$

These can be expressed as

$$V_{\rm i} = T_{\rm i}Q \qquad \text{for i} = \text{sv, pv, sa, pa}, \tag{8.79}$$

with

$$T_{\rm sa} = C_{\rm sa}\left(\frac{1}{K_{\rm R}} + R_{\rm s}\right) \qquad \text{and} \qquad T_{\rm pa} = C_{\rm pa}\left(\frac{1}{K_{\rm L}} + R_{\rm p}\right) \tag{8.80}$$

$$T_{\rm sv} = C_{\rm sv}\left(\frac{1}{K_{\rm R}}\right) \qquad \text{and} \qquad T_{\rm pv} = C_{\rm pv}\left(\frac{1}{K_{\rm L}}\right). \tag{8.81}$$

Inserting these equations in (8.74) for the total blood volume gives

$$V_0 = V_{\rm sa} + V_{\rm sv} + V_{\rm pa} + V_{\rm pv} \tag{8.82}$$

$$= Q(T_{\rm sa} + T_{\rm sv} + T_{\rm pa} + T_{\rm pv}) \tag{8.83}$$

or

$$Q = \frac{V_0}{T_{\rm sa} + T_{\rm sv} + T_{\rm pa} + T_{\rm pv}}. \tag{8.84}$$

All the volumes are obtained from this and (8.79)

$$V_{\rm i} = \frac{T_{\rm i}V_0}{T_{\rm sa} + T_{\rm sv} + T_{\rm pa} + T_{\rm pv}} \tag{8.85}$$

and all of the pressures from $P_{\rm i} = V_{\rm i}/C_{\rm i}$ (from (8.70) and (8.71))

$$P_{\rm i} = \frac{1}{C_{\rm i}}\,\frac{T_{\rm i}V_0}{T_{\rm sa} + T_{\rm sv} + T_{\rm pa} + T_{\rm pv}}. \tag{8.86}$$

With known values of the flow rate and the total volume, the model parameters can be determined – see Table 8.7.

Table 8.7. Normal resting parameters of the circulation model. (Using data from [394])

	systemic system	pulmonary system
resistance, mmHg/(L/min)	$R_{\mathrm{s}} = 17.5$	$R_{\mathrm{p}} = 1.79$
compliance, L/mmHg		
arterial	$C_{\mathrm{sa}} = 0.01$	$C_{\mathrm{pa}} = 0.00667$
venous	$C_{\mathrm{sv}} = 1.75$	$C_{\mathrm{pv}} = 0.08$
heart	$K_{\mathrm{L}} = 1.12$	$K_{\mathrm{R}} = 2.8$
total volume: $V_0 = 5.0$ L		

8.4.3 The Arterial Pulse

This model is capable of analyzing much more than just this idealized average flow. We can use it to understand the arterial pulse, which is the periodic deviation of the systemic arterial pressure from the diastolic value due to systole (Fig. 8.54). This pulse pressure is

$$P_{\mathrm{pulse}} = P_{\mathrm{systole}} - P_{\mathrm{diastole}}, \tag{8.87}$$

which is ∼40 mmHg (= 120 mmHg − 80 mmHg). These steady-state assumptions we just made are no longer appropriate here for this *pulsatile* flow.

Consider the systemic artery system, which has volume V_{sa} and pressure P_{sa}. As a compliance vessel its volume can change, and it will change when the flow into it does not equal to the flow leaving it (Fig. 8.55). The flow into it is that from the left heart Q_{L} and the flow out of it goes into the (noncompliant) small vessel system, which has flow Q_{s}. Conservation of volume gives

$$\frac{\mathrm{d}V_{\mathrm{sa}}(t)}{\mathrm{d}t} = Q_{\mathrm{L}}(t) - Q_{\mathrm{s}}(t). \tag{8.88}$$

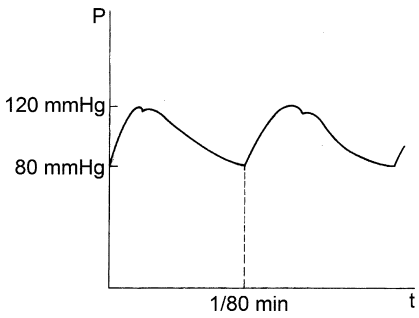

Fig. 8.54. The systemic arterial pulse. Also see the aortic pressure in Fig. 8.5. (From [394])

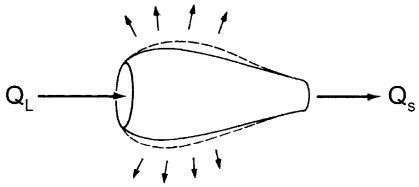

Fig. 8.55. During pulsatile flow, the change of arterial volume is equal to the volumetric inflow minus the outflow, as required by the conservation of volume (mass). (From [394])

For this compliance vessel we know that

$$V_{\mathrm{sa}}(t) = V_{\mathrm{sa,d}} + C_{\mathrm{sa}} P_{\mathrm{sa}}(t), \tag{8.89}$$

where we have now included the dead volume. Differentiating this equation with respect to time and combining it with the previous equation gives

$$\frac{\mathrm{d}V_{\mathrm{sa}}(t)}{\mathrm{d}t} = C_{\mathrm{sa}} \frac{\mathrm{d}P_{\mathrm{sa}}(t)}{\mathrm{d}t} = Q_{\mathrm{L}}(t) - Q_{\mathrm{s}}(t). \tag{8.90}$$

Using (8.72) gives

$$R_{\mathrm{s}} Q_{\mathrm{s}} = P_{\mathrm{sa}} - P_{\mathrm{sv}} \sim P_{\mathrm{sa}} \tag{8.91}$$

since $P_{\mathrm{sv}} \ll P_{\mathrm{sa}}$. Therefore we arrive at

$$C_{\mathrm{sa}} \frac{\mathrm{d}P_{\mathrm{sa}}(t)}{\mathrm{d}t} = Q_{\mathrm{L}}(t) - \frac{P_{\mathrm{sa}}}{R_{\mathrm{s}}}. \tag{8.92}$$

This determines the time dependence of the systemic artery pressure, and consequently the arterial pulse, if the flow rate out of the left ventricle is known.

We will assume that systole occurs very fast (for a very small fraction of the cardiac cycle of duration T, with $T = 1/F$) with a very large $Q_{\mathrm{L}}(t)$ for $t \sim 0$, and that it is zero for the rest of the cardiac cycle. This idealized model is amenable to simple analysis. Although it is not very accurate, some of the features it predicts are accurate.

When $Q_{\mathrm{L}}(t) = 0$, (8.92) is

$$C_{\mathrm{sa}} \frac{\mathrm{d}P_{\mathrm{sa}}(t)}{\mathrm{d}t} = -\frac{P_{\mathrm{sa}}}{R_{\mathrm{s}}} \tag{8.93}$$

or

$$\frac{\mathrm{d}P_{\mathrm{sa}}(t)}{\mathrm{d}t} = -\frac{P_{\mathrm{sa}}}{R_{\mathrm{s}} C_{\mathrm{sa}}} = -\frac{P_{\mathrm{sa}}}{\tau}, \tag{8.94}$$

where $\tau = R_{\mathrm{s}} C_{\mathrm{sa}}$. Therefore

$$P_{\mathrm{sa}}(t) = P_{\mathrm{sa}}(0) \exp(-t/\tau), \tag{8.95}$$

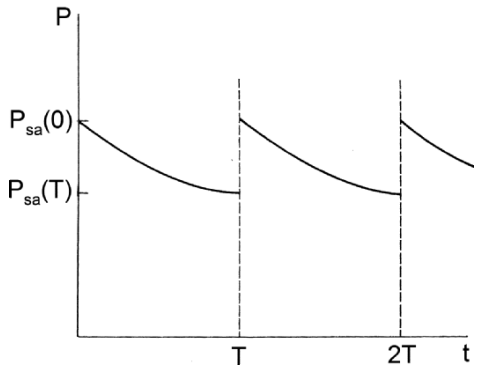

Fig. 8.56. Systemic arterial pulse from idealized model. (From [394])

as is depicted in Fig. 8.56 (see Appendix C). In this model, at the start of the cardiac cycle $P_{\rm sa}(0) = P_{\rm systole}$ and at the end of the cardiac cycle $P_{\rm sa}(T) = P_{\rm diastole} = P_{\rm systole}\exp(-T/\tau)$. The pressure of the arterial pulse is $P_{\rm pulse} = P_{\rm systole} - P_{\rm diastole}$.

We can determine these pressures in terms of the stroke volume using (8.62)–(8.64) and (8.70)

$$V_{\rm sa}(0) = V_{\rm sa,d} + C_{\rm sa}P_{\rm sa}(0) \tag{8.96}$$

$$V_{\rm sa}(T) = V_{\rm sa,d} + C_{\rm sa}P_{\rm sa}(T). \tag{8.97}$$

The difference is the stroke volume

$$\begin{aligned} V_{\rm stroke} &= V_{\rm sa}(0) - V_{\rm sa}(T) = C_{\rm sa}(P_{\rm sa}(0) - P_{\rm sa}(T)) \\ &= C_{\rm sa}P_{\rm sa}(0)[1 - \exp(-T/\tau)]. \end{aligned} \tag{8.98}$$

Consequently, we find

$$P_{\rm systole} = P_{\rm sa}(0) = \frac{V_{\rm stroke}}{C_{\rm sa}[1 - \exp(-T/\tau)]} \tag{8.99}$$

$$P_{\rm diastole} = P_{\rm sa}(T) = \frac{V_{\rm stroke}\exp(-T/\tau)}{C_{\rm sa}[1 - \exp(-T/\tau)]}. \tag{8.100}$$

With (8.87) we see

$$P_{\rm pulse} = P_{\rm systole} - P_{\rm diastole} = \frac{V_{\rm stroke}}{C_{\rm sa}} \tag{8.101}$$

and so $P_{\rm systole}$, $P_{\rm diastole}$, $V_{\rm stroke}$, the heart rate $1/T$, $C_{\rm sa}$, and $R_{\rm s}$ $(= \tau/C_{\rm sa})$ are all interrelated.

Let us consider a numerical example. For $V_{\rm stroke} = 70\,{\rm cm}^3 = 0.070\,{\rm L}$ and $P_{\rm pulse} = 40\,{\rm mmHg}$, (8.98) gives $C_{\rm sa} = V_{\rm stroke}/P_{\rm pulse} = 0.00175\,{\rm L/mmHg}$.

Using R_s from Table 8.7 of 17.5 mmHg/(L/min), we know that $\tau = R_s C_{sa} = 0.0306$ min. With $T = 1/80$ min $= 0.0125$ min, (8.99) and (8.100) give the last remaining parameter, which could be either $P_{systole}$ or $P_{diastole}$, because they are related by $P_{pulse} = P_{systole} - P_{diastole}$. From (8.101), we get $P_{systole} = 120$ mmHg. As expected from self-consistency, $P_{diastole} = P_{systole} \exp(-T/\tau) = 120\,\text{mmHg} \times \exp(-0.0125\,\text{min}/(-0.0306\,\text{min})) = 80$ mmHg.

A better model for the arterial pulse is described in Problem 8.49, in which the flow of blood from the left ventricle to the systemic arteries occurs with a finite (nonzero) duration in the cardiac cycle. It gives predictions that agree with Fig. 8.54 better than do those in Fig. 8.56. Even this improved model does not explain the small increase in pressure just after systole that is seen in Figs. 8.5 and 8.54, which is known as the "incisura" or "dicrotic notch." When the semilunar valve closes, some backward flowing blood bounces off the elastic aorta walls, setting up a pressure wave in the aorta; this pressure blip can be included with even more refinement in the model.

8.4.4 Windkessel Model

This simplified model of the arterial pulse is also known as the Windkessel Model [370], which was the first real model of blood flow. In this model blood flows from the left ventricle at a rate $Q_{in}(t)$ into an elastic chamber of compliance C (i.e., the larger arteries) of volume $V(t)$ and leaves it at a rate $Q_{out}(t)$ to enter a resistive element of resistance R_p (peripheral resistance of the arterioles and capillaries). It assumes that all pressure changes in the arteries occur at the same time. Because conservation of volume for an incompressible fluid gives

$$Q_{in}(t) = \frac{dV(t)}{dt} + Q_{out}(t), \tag{8.102}$$

with $C = dV(t)/dP(t)$ (8.61) and $Q_{out}(t) = P(t)/R_p$ (8.72), (8.102) becomes

$$Q_{in}(t) = C\frac{dP(t)}{dt} + \frac{P(t)}{R_p}. \tag{8.103}$$

The formal solution to this is

$$P(t) = \exp(-(t/\tau))\left(P(0) + \frac{1}{C}\int_0^t \exp(t'/\tau) Q_{in}(t')dt'\right), \tag{8.104}$$

where $\tau = R_p C$. This can be shown to be the solution by substitution and by the method shown in Appendix C. Still it is simple and instructive to examine what happens after ventricular ejection, starting at $t = 0$ when $Q_{in}(t) = 0$ and $P = P(0)$, until the next cycle starts at $t = T$. Then

$$\frac{dP(t)}{dt} + \frac{P(t)}{R_p C} = 0 \qquad \text{for } 0 < t < T. \tag{8.105}$$

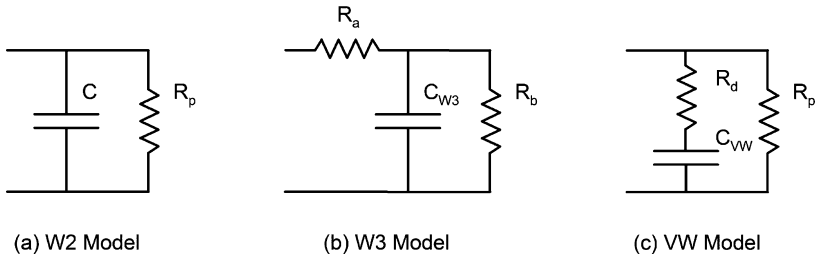

Fig. 8.57. Electrical analogs of the (**a**) classic or elastic Windkessel Model (W2), (**b**) three-element (or improved) Windkessel Model (W3), and (**c**) viscoelastic Windkessel Model (VW). The electrical resistances represent flow resistances and the electrical capacitances represent flow compliances. (See Appendix D)

The solution is

$$P(t) = P(0)\exp(-(t/\tau)) \qquad \text{for } 0 < t < T, \tag{8.106}$$

which is the same as (8.95) (see Appendix C).

This two-element classic Windkessel Model is formally equivalent to the electrical circuit, where the electrical resistance R_{elect} maps into the flow resistance in the capillaries and the electrical capacitance C_{elect} maps into the flow compliance (Table D.1, Appendix D). (This is how the model is expressed in Fig. 8.57a.) The voltage V_{elect} in the electrical model corresponds to the pressure P in the flow model and the current I corresponds to the volumetric flow rate Q. Ohm's Law relates V_{elect} and I across the resistor by $V_{\text{elect}} = IR_{\text{elect}}$. The voltage across the capacitor is $V_{\text{elect}} = q/C_{\text{elect}}$, where the charge on the capacitor is q. The voltages across both elements sum to zero (Kirchhoff's 2nd Law, Chap. 12), so $IR_{\text{elect}} + q/C_{\text{elect}} = 0$. Because $I = \mathrm{d}q/\mathrm{d}t$, we have $(\mathrm{d}q/\mathrm{d}t)R_{\text{elect}} + q/C_{\text{elect}} = 0$ or with $V_{\text{elect}} = q/C_{\text{elect}}$

$$\frac{\mathrm{d}V_{\text{elect}}}{\mathrm{d}t} + \frac{V_{\text{elect}}}{R_{\text{elect}}C_{\text{elect}}} = 0. \tag{8.107}$$

The two-element Windkessel Model can be refined by adding more elements to the electrical analog. The three-element Windkessel Model shown in Fig. 8.57b predicts a more realistic arterial pulse. The vascular resistance R_{a} of the aorta is added in series, and the value of the resistance in parallel is now R_{b}, which equals $R_{\text{p}} - R_{\text{a}}$. The viscoelastic Windkessel Model (Fig. 8.57c) represents a different type of improvement of the two-element Windkessel Model. The compliant arterial systems are represented by a capacitance (in the electrical analog of flow compliance) that is in series with a resistor R_{d}, which represents the viscous wall motion to more fully represent the viscoelasticity of the aorta.

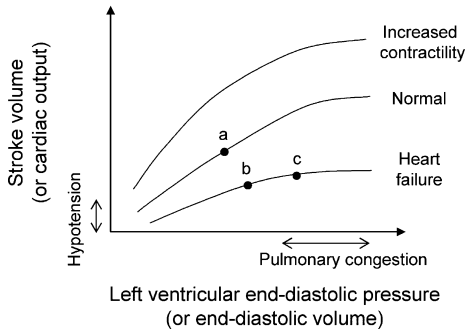

Fig. 8.58. Left ventricular performance (Frank–Starling) curves relate cardiac performance to preconditions (preloading), such as the pressure or volume of the left ventricle after diastole. (Based on [380])

8.4.5 Modeling the Malfunctioning Heart

There are many ways a heart can malfunction [402]. These conditions can be characterized quantitatively and, in principle, can be included in our models of the heart and circulation. Cardiac performance can be characterized by several parameters, such as the stroke volume V_{stroke} and cardiac output $Q_{\text{t}} = FV_{\text{stroke}}$, described earlier.

Figure 8.58 shows cardiac performance as a function of the conditions at the end of diastole in the left ventricle. The middle curve describes the operation of that person's heart, and point a represents that normal person at rest. After heart failure, such as after a myocardial infarction, the curve shifts down due to lessened left ventricle contractility. Point b represents the person described by point a after heart failure. Increased circulatory volume in this person is represented by point c. The stroke volume is increased due to increased contractility in the uppermost curve, perhaps caused by the infusion of the drug norepinephrine.

The solid line pressure–volume loops in Fig. 8.59 represent normal heart function, and are more accurate representations than the simplified rectangular one depicted in our model in Fig. 8.52. In systolic dysfunction, the systolic curve shifts from 1 to 2 in (a) due to decreased cardiac contractility. As seen in the dashed loop, the volume at the end of systole increases. In diastolic dysfunction, the diastolic curve shifts from 1 to 2 in (b) due to increased stiffness (decreased compliance). As seen by the dashed loop, the ventricular pressure is higher than normal at any diastolic volume.

Several series of problems occur when there is leakage between heart chambers. In valvular *regurgitation* there is backflow through one of the heart valves, such as mitral regurgitation and aortic regurgitation in the left heart valves. The regurgitant fraction for mitral regurgitation is the volume of blood flowing back through the mitral valve divided by the left ventricle stroke

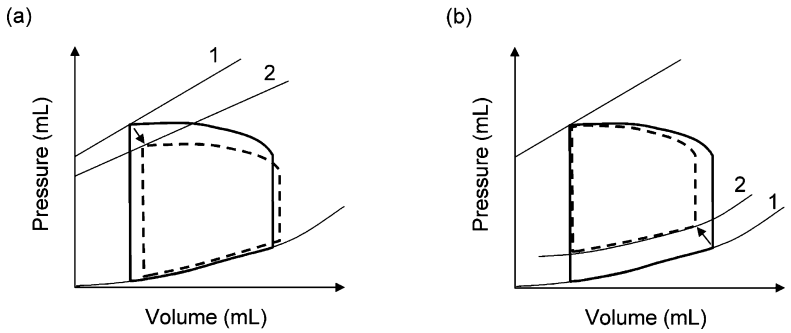

Fig. 8.59. Pressure–volume curves for the left ventricle for normal performance (*solid loop*), systolic dysfunction (*dashed loop* in (**a**)), and diastolic dysfunction (*dashed loop* in (**b**)). (Based on [380])

volume. There can also be openings in the septum between the atria or the ventricles, called a septal defect [374]. For example, a ventricular septal defect is a congenital condition in which there is an opening between the left and right ventricles (Fig. 8.60). During systole blood flows from the left to right ventricle because of the higher pressure in the former. This leads to increased blood return to the left side of the heart which causes the left atrium and ventricle to enlarge. The ejection fraction (EF) is the stroke volume divided by the ventricular volume at the end of diastole (when the ventricle is full). It is normally 55–75%, but can be less with a septal defect.

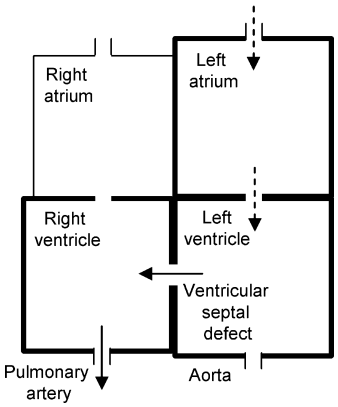

Fig. 8.60. With a ventricular septal valve defect blood flows from the left to right ventricle. (Based on [374])

8.5 Summary

The flow of blood in the vessels of the circulation system can be modeled using the resistive and compliant nature of the flow. This can be combined with a model of the heart to understand the flow in the entire circulation system. These models can also be used to understand the physical consequences of a malfunctioning heart and problems in the circulation system, such as clogged and weakened arteries, that can lead to strokes and aneurysms. The energy needed to operate the heart can also be modeled.

Problems

Blood Pressure

8.1. (a) The brain in a human is 55 cm above the heart. If the average blood pressure in the major arteries near the heart is 100 mmHg, what is the blood pressure in major arteries in the brain (in both mmHg and cmH_2O) when a person is either lying down or standing up.
(b) Repeat part (a) for an erect human on the moon ($g = g_{\text{Earth}}/6$) and on Jupiter ($g = 2.34 g_{\text{Earth}}$).
(c) A pilot coming out of a dive experiences an upward centripetal acceleration a of magnitude v^2/r, where v is the speed of the jet and r is the radius of curvature of the trajectory, that adds to gravity (effectively increasing g to $g + a$). What is the arterial pressure in the pilot's brain for $v = 200\,\text{m/s}$ and $r = 2\,\text{km}$? What could happen to the pilot during this recovery from the dive? Would you expect dizziness because of a lack of blood to the head? (See Fig. 8.61.)
(d) What must the pressure in the aorta in a giraffe be (on Earth) for its brain to receive blood? (How can you estimate the elevation of its brain above its aorta?)

8.2. In Problem 8.1d we saw that the pressure of the blood leaving the heart of a giraffe and entering its systemic system must be much larger than that for humans because of its long neck.

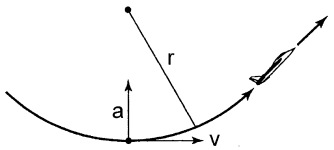

Fig. 8.61. Trajectory of a pilot coming out of a dive, with the acceleration and velocity vectors shown for the low point of the dive. For Problem 8.1. (From [367])

(a) Would a blood pressure of 280 mmHg/180 mmHg account for pumping the blood up this long neck in large arteries and then for the pressure drop that occurs in the very small arteries in the brain?
(b) The giraffe has this relatively high blood pressure because of this long neck, which is usually nearly vertical. However, we could expect that there would be a rush of blood to the brain because of this high pressure when the giraffe lowers its head by almost 7 m when it bends down to drink water, and that this could lead to rupture of the arteries in the brain. Why?
(c) This artery rupturing does not occur because of compensating effects. Explain this by considering the following (i) The elastic walls of the long giraffe carotid artery help force blood upward (which is a peristaltic action), and this also means that this artery can swell to absorb excess blood when the head is lowered because it is very compliant. (ii) The giraffe jugular vein contains a series of one-way valves that prevent back-flow of the blood when the giraffe's head is down.

8.3. When you stand on your head, why does your head become red and why do your legs become pale?

8.4. Why is blood pressure measured using major arteries in the upper arm, rather than those in the lower arm or leg?

8.5. You are told that your blood pressure is 880 mmHg/840 mmHg. You are quite understandably concerned because these values are astronomically high, but you are told not to worry because your blood pressure is normal. Should you be concerned?

8.6. (a) An intravenous infusion is made under gravity. If the fluid to be delivered has a density of 1.0 g/cm^3, at what height above the vein, h, should the top surface of the fluid in the bottle be positioned so the fluid just barely enters the vein? The gauge pressure in the vein is 18 mmHg. (Assume the needle entering the vein has a "large" inside diameter.) (See Fig. 8.62.)
(b) If this needle has a "very small" inside diameter, should the bottle be placed higher, lower, or at the same height? Why?
(c) Why are such infusions performed intravenously and not intra-arterially?

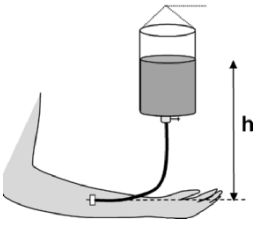

Fig. 8.62. Intravenous infusion under gravity. (Based on [387].) For Problem 8.6

8.7. You are lying down and are injured in such a way that blood from a major artery squirts upward. How high can it spurt?

8.8. Twirl one arm around as fast as you can many times until you see your fingers on that arm turn red. (Continue even if you do not see them getting redder.) [365]
(a) Estimate the centrifugal acceleration at the end of your finger tips, $v_{\text{radial}}^2/r = v_{\text{radial}}^2/l_{\text{arm}}$, where l_{arm} is your arm length (to your fingertips) and v_{radial} is the radial speed of your finger tips. (Why is $v_{\text{radial}} = 2\pi l_{\text{arm}}/T$, where T is the period for a complete cycle of this motion?)
(b) Express this acceleration in units of g.
(c) Calculate the effective pressure pushing your blood to your fingers by this motion. This is the apparent outward force divided by the cross-sectional area of your fingers, A_{fingers}, or $(m_{\text{fingers}} v_{\text{radial}}^2/l_{\text{arm}})/A_{\text{fingers}}$. Because $m_{\text{fingers}} = \rho_{\text{fingers}} l_{\text{fingers}} A_{\text{fingers}}$, this pressure is $\rho_{\text{fingers}} v_{\text{radial}}^2 (l_{\text{fingers}}/l_{\text{arm}})$.
(d) Compare this to the systolic pressure 120 mmHg and explain why your fingers (could have or should have) turned red.

Blood Pressure Drop During Flow

8.9. Calculate the pressure drop (in mmHg) across the following arterial systems using Poiseuille's Law with $\eta_{\text{blood}} = 4 \times 10^{-3}$ Pa-s, for a total flow of $80\,\text{cm}^3/\text{s}$ across each system:
(a) aorta (internal radius $r = 1.25$ cm, length $L = 10$ cm, all of the flow in this one aorta)
(b) large arteries ($r = 0.2$ cm, $L = 75$ cm, $n = 200$ of them, each with equal flow and the same dimensions)
(c) arterioles ($r = 30\,\mu$m, $L = 0.6$ cm, $n = 5 \times 10^5$)
(d) capillaries ($r = 3.5\,\mu$m, $L = 2$ mm, $n = 10^{10}$).

8.10. In estimating pressure drops across the different arterial branches we assumed specific numbers of arteries of given diameters and lengths. There is really a wide range of arterial diameters and lengths. How does this affect the pressure drops in the systemic arterial system?

8.11. In estimating pressure drops across the arterioles we assumed a specific number of arterioles with the same diameter and length.
(a) Let us say that all arterioles have the same radius, but their lengths (instead of all being L) range between $0.8L$ and $1.2L$ (with equal probability throughout). How does this change the overall resistance of the arteriole system?
(b) Let us say that all arterioles have the same length, but their radii (instead of all being r) range between $0.8r$ and $1.2r$ (with equal probability throughout). How does this change the overall resistance of the arteriole system?
(c) The pressure drop across each arteriole in the system must be the same because each is fed by the large arteries, whose pressure is set by the left

ventricle, and by the arterial side of the capillaries, whose pressure is also set. If the overall pressure drop across the arterioles is unchanged (by the changes in (a) or (b)), how is the overall flow rate in the arteriole system changed and what is the flow in each arteriole, for the situations alternately described in (a) and (b).
(d) If you wanted the flow rate to stay the same in each arteriole in (a) and (b) for the given distributions of lengths and radii, how would you have to change the distributions of radii and lengths in each, respectively?

8.12. Find the pressure drop across the arterioles in Problem 8.9c, if – with the same total flow in both cases – and either
(a) all the arterioles become clogged in such a way that their radii decrease to 28 μm or
(b) the number of the arterioles decreases to 4×10^5.
(c) By how much would the pressure in the main arteries need to change if the body responded to either change by maintaining the same flow rate?

8.13. Assume that the diameter of each blood vessel in a person is doubled and the total volumetric flow rate is not changed.
(a) What is the new total volume of blood? (Assume the base line parameters in the chapter.)
(b) What is the new circulation time for blood (total blood volume/total volumetric flow rate)?
(c) How do the resistances of the arterioles and capillaries change?
(d) How does the pressure drops across the arterioles and capillaries change?
(e) How does the work done by the heart change?

8.14. Repeat Problem 8.13 if instead the length of each blood vessel is doubled.

8.15. The length of a blood vessel is doubled and its diameter is doubled.
(a) How does the flow resistance change?
(b) If the flow through it is unchanged, how does the pressure drop change?
(c) How does the flow through it change if instead the pressure drop is unchanged?

8.16. Your internal body temperature increases from 37 to 40°C. Assuming that the only thing that changes is the viscosity of blood, how must the blood pressure change to ensure the flow rate remains unchanged?

8.17. Use (8.29) to relate $P(L)$ to $P(0)$ and other flow terms.

8.18. (a) Estimate how much the flow is changed in small arteries by including the influence of compliance by using (8.29) and assuming the same pressure drop.
(b) Repeat this estimate for how much the pressure drop changes with this analysis assuming the same flow rate.

8.19. Express the flow resistance units of N-s/m^5 in terms of (N/m^2)/(cm^3/s), dyne-s/cm^5, and PRU (with 1 PRU = 1 mmHg-s/mL).

8.20. (a) Calculate the total peripheral vascular resistance in the systemic and pulmonary systems for someone with a steady-state blood flow rate of 5 L/min, and with 120 mmHg/80 mmHg blood pressure in the systemic system and 25 mmHg/8 mmHg pressure in the pulmonary system. Express your answer in the units of dyne-s/cm^5, which are CGS units and those that are often used by cardiologists.
(b) The expression given in the text for this vascular flow resistance should be corrected because it uses the average pressure at the beginning of the system instead of the pressure drop across the system. For the systemic system the final pressure is that at the right atrium (2 mmHg) and for the pulmonary system it is that at the left atrium (5 mmHg). How does using the actual pressure drop affect your calculations in part (a) (both qualitatively and quantitatively)?

8.21. (a) The pulmonary vascular resistance changes with lung volume. Figure 8.63 shows that it increases much with larger lung volumes, in part because the larger alveoli stretch the pulmonary capillaries. It also increases at very small lung volumes because these capillaries surrounding the alveoli become narrow. Calculate the range of pulmonary blood flow rates (in L/min), assuming this range of resistances and assuming that the pulmonary pressures are the same as in Problem 8.20.
(b) The pulmonary pressures actually change with lung volume. If they changed in a manner to keep the average flow the same as it is for a 110 mL lung volume, determine this change. (For the purpose of this calculation,

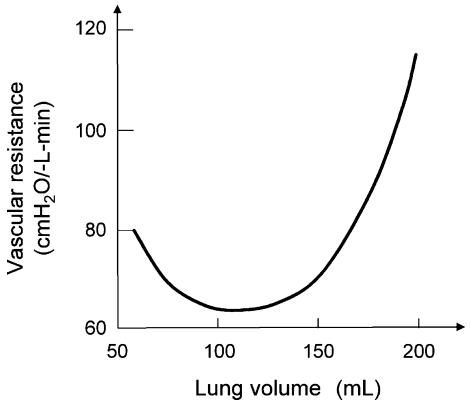

Fig. 8.63. Effect of lung volume on pulmonary vascular (blood flow) resistance (from an animal lobe preparation). (Based on [419].) For Problem 8.21

assume that the ratio of systolic and diastolic pressures remains a constant and that the left atrium pressure remains the same.)

8.22. (a) Determine the overall compliances of the systemic arterial and venous systems by using Fig. 8.26.
(b) Is the ratio of these two compliances reasonable, given our model for compliance and the data for the vessels in both groups? (Consider only the large vessels in both groups.)

8.23. Does Fig. 8.26 suggest that sympathetic stimulation and inhibition change the vessels' compliances, dead volumes, or both?

Flow and Pressure

8.24. What is the average time blood spends in a capillary?

8.25. The *cardiac index* is the cardiac output divided by the person's surface area. It normally ranges from 2.6 to 4.2 $(\mathrm{L/min})/\mathrm{m}^2$. Use this to determine the cardiac output of a standard human. How does this value compare to the normal cardiac output we have assumed?

8.26. An artery with radius r_1 and blood speed u_1 divides into n arteries of equal radius. Find the radius r_2 and blood speed u_2 in these daughter arteries assuming that the pressure drop per unit distance $\mathrm{d}P/\mathrm{d}x$ is the same in the initial artery and each daughter artery.

8.27. Four veins with radius r_1 and flow speed u_1 combine to form one vein with radius $r_2 = 4r_1$. Find the flow speed in the larger vein.

8.28. The design of blood vessels is sometimes optimized by minimizing a "cost function," F, which is the sum of the rate work is done on the blood in the vessel and the rate that energy is used by metabolism through the blood in the vessel [385]. The first term is $Q(\Delta P)$, for flow rate Q and pressure drop ΔP, and the second term is assumed to be proportional to the volume of the vessel of radius r and length L, $K\pi r^2 L$, where K is a constant. Consequently, the cost function can be written as

$$F = Q(\Delta P) + K\pi r^2 L. \tag{8.108}$$

(a) For a resistive vessel, show that this becomes

$$F = \frac{8\eta L}{\pi r^4}\, Q^2 + K\pi r^2 L. \tag{8.109}$$

(b) For a fixed vessel length and flow rate, show the optimal radius is

$$r_{\mathrm{opt}} = \left(\frac{16\eta}{\pi^2 K}\right)^{1/6} Q^{1/3} \tag{8.110}$$

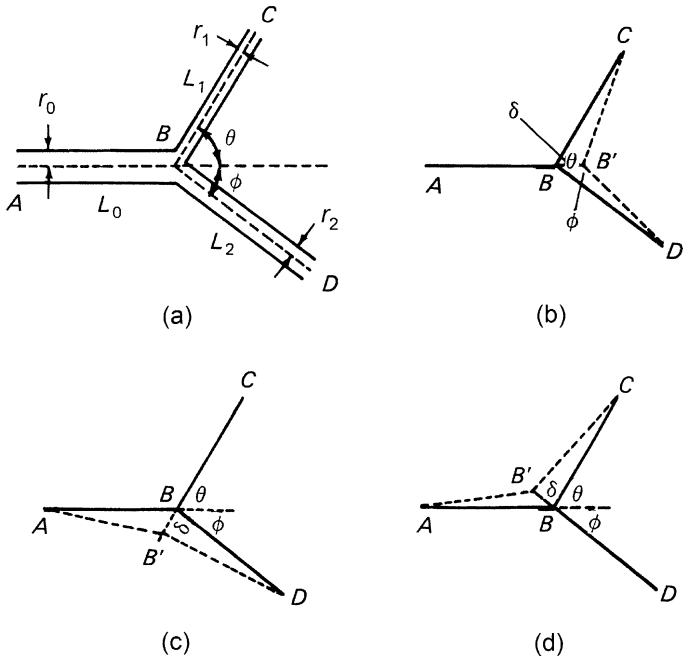

Fig. 8.64. (**a**) A planar, bifurcating vessel. Determining variations in the length of each vessel for small planar displacements of B to B' in the (**b**) AB, (**c**) AC, and (**d**) DB directions. (Based on [385].) For Problem 8.29

and the minimum cost function is

$$F_{\min} = \frac{3\pi}{2} K L r_{\text{opt}}^2. \tag{8.111}$$

8.29. (advanced problem) We will use the cost function defined in Problem 8.28 to optimize the flow in a vessel of radius r_0 and length L_0 with flow rate Q_0, that bifurcates into a vessel of radius r_1 and length L_1 with flow rate Q_1 at an angle θ to the first vessel and one with radius r_2 and length L_2 with flow rate Q_2 at an angle ϕ to the first vessel, as seen in Fig. 8.64a [385]. Having straight, coplanar vessels minimizes the vessel lengths.
(a) Show that the total cost function is

$$F_{\min} = \frac{3\pi K}{2}(r_0^2 L_0 + r_1^2 L_1 + r_2^2 L_2). \tag{8.112}$$

(b) We can optimize the lengths and angles of the vessels by considering how the displacements of point B in Figs. 8.64b–d change the cost function. Show that any such movement of point B causes length changes δL_0, δL_1, and δL_2

that lead to a change of the cost function of

$$\delta F_{\min} = \frac{3\pi K}{2}(r_0^2(\delta L_0) + r_1^2(\delta L_1) + r_2^2(\delta L_2)). \tag{8.113}$$

This is optimized by setting $\delta F_{\min,\text{opt}} = 0$.
(c) Show that moving point B along AB to B' by a distance δ as shown in Fig. 8.64b gives $\delta L_0 = \delta$, $\delta L_1 = -\delta\cos\theta$, and $\delta L_2 = -\delta\cos\phi$, and $\delta F_{\min,\text{opt}} = (3\pi K\delta/2)(r_0^2 - r_1^2\cos\theta - r_2^2\cos\phi) = 0$, and so it is optimized by

$$r_0^2 = r_1^2\cos\theta + r_2^2\cos\phi. \tag{8.114}$$

(d) Show that moving point B along CB to B' by a distance δ as shown in Fig. 8.64c gives $\delta L_0 = -\delta\cos\theta$, $\delta L_1 = \delta$, and $\delta L_2 = \delta\cos(\theta+\phi)$, and $\delta F_{\min,\text{opt}} = (3\pi K\delta/2)(-r_0^2\cos\theta + r_1^2 + r_2^2\cos(\theta+\phi)) = 0$, and so it is optimized by

$$-r_0^2\cos\theta + r_1^2 + r_2^2\cos(\theta+\phi) = 0. \tag{8.115}$$

(e) Show that moving point B along DB to B' by a distance δ as shown in Fig. 8.64d gives the optimization condition

$$-r_0^2\cos\phi + r_1^2\cos(\theta+\phi) + r_2^2 = 0. \tag{8.116}$$

(Note the symmetry in the last two equations.)
(f) Show that (8.114)–(8.116) can be solved to give

$$\cos\theta = \frac{r_0^4 + r_1^4 - r_2^4}{2r_0^2r_1^2}, \tag{8.117}$$

$$\cos\phi = \frac{r_0^4 - r_1^4 + r_2^4}{2r_0^2r_2^2}, \tag{8.118}$$

and

$$\cos(\theta+\phi) = \frac{r_0^4 - r_1^4 - r_2^4}{2r_1^2r_2^2}. \tag{8.119}$$

(g) Use continuity of flow and (8.110) to show that

$$r_0^3 = r_1^3 + r_2^3. \tag{8.120}$$

(h) Show that (8.117) then becomes

$$\cos\theta = \frac{r_0^4 + r_1^4 - (r_0^3 - r_1^3)^{4/3}}{2r_0^2r_1^2} \tag{8.121}$$

and find the analogous relations for (8.118) and (8.119).

8.30. Use Problem 8.29 to show that for optimized bifurcating vessels if [385]
(a) $r_1 = r_2$, then $\theta = \phi$,
(b) $r_2 > r_1$, then $\theta > \phi$,
(c) r_2 is much greater than r_1, then r_2 approaches r_0 and ϕ approaches $\pi/2$,
(d) $r_1 = r_2$, then $r_1/r_0 = 2^{-1/3} = 0.794 = \cos\theta$ and so $\theta = 37.5°$.
These results generally agree with observations.

8.31. Use Problem 8.30d to show that it would take ∼30 generations of symmetric bifurcations starting with a vessel with the aorta radius of 1.5 cm to arrive at a vessel with the capillary radius of 5×10^{-4} cm [385]. (Note, however, that such arterial divisions are usually not simple symmetric bifurcations.)

8.32. There is a fusiform aneurysm in an aorta where the internal radius increases from $r_1 (= 1.25\,\text{cm})$ in the normal section to $r_2 = 1.3r_1$ in the diseased section, while staying at the same vertical height. The speed of blood flow is $v_1 = 0.4\,\text{m/s}$ in the normal section and the (gauge) pressure P_1 is 100 mmHg. The blood density is $1{,}060\,\text{kg/m}^3$.
(a) Find the speed of blood flow v_2 in the aneurysm.
(b) Find the pressure P_2 in the aneurysm.
(c) Use the Law of Laplace to find the tensions required in the normal part of the aorta and in the aneurysm to maintain the pressure difference (from inside to outside the vessel). Compare these values.
(d) Describe how this increase in the tension needed in the aneurysm wall and the decreased strength of the wall (due to the thinner aorta wall in the aneurysm) can lead to an unstable situation.

8.33. The normal inner radius of a large artery is 2 mm. It is 75 cm long, and the flow through it is 1/200 of the total blood flow. How would the pressure drop across it change if the flow through it were unchanged and there were severe stenosis in the artery
(a) across its entire length or
(b) across 5 cm of it?
(c) In each case, if the pressure at the beginning of the artery were 75 mmHg, would the pressure drop be severe enough to affect flow in the arterioles and capillaries?
(d) In each case, what added pressure would be needed at the beginning of the artery to maintain an unchanged flow in these arterioles and capillaries?

8.34. Arteriosclerotic plaque narrows down a section of an artery to 20% of its normal cross-sectional area. What is the pressure in that section if immediately before it the pressure is 100 mmHg and the flow speed is 0.12 m/s?

8.35. The osmotic pressure of blood is 25 mmHg higher than that of interstitial fluid because it has a higher density of proteins. What is the difference in their densities of proteins that accounts for this?

8.36. (more advanced) The blood hematocrit is usually higher nearer the center of a blood vessel than at the blood vessel wall and has a distribution that we will take as $h(r) = H[1 - (r/R)^2]$ from $r = 0$ to R [406]. (The reason why flowing suspended particles, such as red blood cells, have higher concentrations near the center, called the Fahraeus–Lindquist effect, is not obvious.)
(a) The volume flow of a cylindrical shell in the vessel is $2\pi r v(r)\mathrm{d}r$, where $v(r) = 2u\left(1 - r^2/R^2\right)$ from (7.40), so this flow weighted for the hematocrit is $2\pi r h(r) v(r)\mathrm{d}r$. Therefore the average hematocrit in the transported blood is $h_{\mathrm{av}} = \int_0^R 2\pi r h(r) v(r)\mathrm{d}r / \int_0^R 2\pi r v(r)\mathrm{d}r$. Show that $H = 3h_{\mathrm{av}}/2$.
(b) Now find the average value of the hematocrit at any given time in the blood vessel by calculating $\int_0^R 2\pi r h(r)\mathrm{d}r / \int_0^R 2\pi r\,\mathrm{d}r$. Show that for the parabolic distribution of hematocrit this volume-averaged hematocrit is $3h_{\mathrm{av}}/4$.
(c) The result in (b) states that the average hematocrit of the blood in the vessel at any given time is less than that in the blood that is being transported. Is this a contradiction?

8.37. (more advanced) Repeat parts (a) and (b) in Problem 8.36 assuming $h(r) = H\exp(-r/R)$ and show the volume-averaged hematocrit is $0.88h_{\mathrm{av}}$ [406].

8.38. (more advanced) The analysis in Problems 8.36 and 8.37 assumed that the parabolic $v(r)$ we derived earlier assuming a constant viscosity is still valid when the hematocrit – and consequently the viscosity – decreases with radius. This should not be true. Qualitatively, how would you expect the spatially varying hematocrit and viscosity to affect the parabolic flow rate?

The Heart and Circulation

8.39. Would you expect cardiac muscle to be most similar to Type I, IIA, or IIB skeletal muscle? Why?

8.40. Compare the total mechanical and metabolic powers needed by the left heart and the right heart to pump blood.

8.41. Determine all the pressures, volumes, and flow rates in the overall body circulation model using the data provided in the Table 8.7. Do your answers agree with your expectations, such as the values in Table 8.1?

8.42. The volumetric flow rate out of a ventricle has been described in terms of the heart rate F and stroke volume V_{stroke} as $Q = FV_{\mathrm{stroke}}$, while flow rates have also been expressed in terms of the vessel cross section A and flow speed u as $Q = Au$. Explain why these two characterizations are either consistent or inconsistent.

8.43. If the cardiac output is 5 L/min and heart rate is 1 Hz, determine the volume of the left ventricle at its peak if the ejection fraction is 65%.

8.44. There is a hole in the septum that separates the left ventricle and right ventricle (Fig. 8.60).
(a) Explain why you would expect the pressure in the left ventricle to decrease and that in the right ventricle to increase.
(b) Explain why you would expect the stroke volume from the left ventricle to decrease because of this.
(c) Explain why the oxygenation of the blood in the left ventricle would decrease and that in the right ventricle would increase.
(d) If during systole the (gauge) pressure, stroke volume, and oxygenation levels (relative to that in the vena cavae) in the left ventricle each decreases by 10% as a result of this, explain quantitatively how the body could try to compensate for this?

8.45. (a) If the inner volume of the left ventricle is $100\,\mathrm{cm}^3$ and the wall volume is $30\,\mathrm{cm}^3$, find the inner radius, outer radius, and wall thickness for the ventricle modeled as a hemispherical shell.
(b) Find the wall stress during systole.

8.46. The cardiac output of a woman remains at 5 L as she ages from 25 to 65 years of age, while her blood pressure increases in the average way.
(a) How does her total vascular resistance change?
(b) What fractional changes in vessel radius do this correspond to? (Assume conditions for arterioles.)

8.47. When blood is pumped out of the left ventricle, it travels "upward" a distance of about 10 cm in the aorta during the ∼0.2 s duration of the peak of systole, stretching the walls of this very compliant vessel. There are no external forces on the body during this time, so the center of mass of the body does not move. Consequently, when the stroke volume of blood (of mass $m_{\mathrm{blood}} \simeq 70\,\mathrm{g}$) is ejected upward, the rest of the body (of mass $m_{\mathrm{rest}} \simeq 70\,\mathrm{kg}$) moves "downward" (ignoring gravity and frictional forces). This is the basis of the diagnostic method called *ballistocardiography*, in which a person rests horizontally on a light, very low friction horizontal suspension [367]. (Such devices have been used to develop methods that assess heart function, but are not in clinical use.) Assume the person is lying along the x direction on this "couch" – with his head pointing in the positive direction – and the center of mass of ejected blood is at x_{blood}, that of the rest of the body is at x_{rest}, and that of the entire body is x_{body}.
(a) Show that $x_{\mathrm{rest}} = (x_{\mathrm{blood}} m_{\mathrm{blood}} + m_{\mathrm{rest}} x_{\mathrm{rest}})/(m_{\mathrm{blood}} + m_{\mathrm{rest}})$ always.
(b) Now let us call the positions in (a) those before systole. At the end of the main part of systole, the blood and rest of the body have moved by

Δx_{blood} and Δx_{rest}, respectively. Show that the body has moved by $\Delta x_{\text{rest}} = -(m_{\text{blood}}/m_{\text{rest}})\Delta x_{\text{blood}}$ and that this is -0.1 mm.
(c) Because the blood moves with a constant velocity in this motion in the aorta, show that the velocity of the body during systole is -0.5 mm/s in the x direction.
(d) What is the average of Δx_{rest} during a full cardiac cycle? Why?

8.48. Someone wants to donate two pints of blood instead of the usual (and allowed maximum of) one. What consequences could this have?

8.49. (advanced problem) (a) Solve (8.92) assuming that the flow $Q_{\text{L}}(t)$ is a constant a from $t = 0$ to αT, and 0 from $t = \alpha T$ to T, where $0 \leq \alpha \leq 1$. (This repeats for every heart beat.) Note that the pressure at the beginning and end of each cardiac cycle is P_{diastole} and it becomes P_{systole} at $t = \alpha T$. (Hint: The analysis is similar to that for exciting an isometric muscle in Chap. 5 (see (5.11)–(5.13)) and temperature regulation in Chap. 13 (see (13.18)); also see Appendix C.)
(b) Show that $a = V_{\text{stroke}}/\alpha T C_{\text{sa}}$.
(c) Show the solutions from (a) lead to the relations

$$P_{\text{diastole}} = P_{\text{systole}} \exp(-(1-\alpha)T/\tau) \qquad \text{and} \tag{8.122}$$

$$P_{\text{systole}} = V_{\text{stroke}}\tau/\alpha T C_{\text{sa}} + (P_{\text{diastole}} - V_{\text{stroke}}\tau/\alpha T C_{\text{sa}}) \exp(-\alpha T/\tau). \tag{8.123}$$

(d) Sketch $P_{\text{sa}}(t)$ for several heart beats for $\alpha = 1/3$. Compare this sketch with those from the simple model in Fig. 8.56 and the real pulse in Fig. 8.54. Is this model better? Why?
(e) Show that when $\alpha = 0$ the solutions in (a) and (c) give the results presented in the text for the simpler model.

8.50. The solution to the classic Windkessel Model for steady-state flow that is suddenly turned off is exponential decay of the flow, as we saw in the simple model of the arterial pulse. In the electrical analog in Fig. 8.57 this corresponds to tracking the current when a constant voltage is initially applied and is suddenly turned off. Analyze this electrical problem analog and show that it has the same solution as the flow problem.

8.51. (advanced problem) Solve the electrical analog in Problem 8.50 for the three-element Windkessel Model.

8.52. (advanced problem) Solve the electrical analog in Problem 8.50 for the viscoelastic Windkessel Model.

Scaling

8.53. Calculate the heart mass and heart beat rate (in beats per minute) for a man (70 kg), woman (50 kg), and an infant (5 kg) using the allometric relation parameters in Table 1.13.

8.54. The heart rate of mammals F is known to decrease with body mass as $m_{\mathrm{b}}^{-1/3}$. This seems to be true interspecies and also within a species. The human heart rate is known to decrease from infancy, through childhood and to maturity in a manner described better by body mass than age. Derive this relation using the dimensional analysis methods presented in Chap. 1. Assume that the stroke volume scales as body mass. Assume that a primary function of circulation is to bring warm blood from the core to the body surface to keep it warm. This means that the total blood flow rate scales as the rate of heat loss from the body.

9

Lungs and Breathing

Our lungs serve several important functions. They interact with blood by exchanging carbon dioxide for oxygen (Chap. 8) and they maintain the blood pH. The lungs are involved in heat exchange and fluid balance in the body, because relatively dry and usually cooler air is inhaled and air at the body temperature saturated with water vapor is exhaled (Chap. 6). They are also a key element in voice production (Chap. 10). We will highlight the mechanics of breathing [423, 424, 428, 429, 430, 432, 434, 435, 439, 443, 444].

We typically breathe in 6 L/min of air. (This compares to the ∼5–6 L of blood pumped per min in the pulmonary circulation through the lungs.) Because air is ∼20% oxygen, we inhale (inspire) 1.2 L oxygen/min. The breathing rate is typically 12/min for men, 20/min for women, and 60/min for infants. The air we inhale has 80% N_2/20% O_2 (or more precisely 78.084% N_2/20.947% O_2/0.934% Ar/0.035% CO_2), and the air we exhale (expire) has 80% N_2/16% O_2/4% CO_2. (If the air we exhaled had little or no oxygen, we could not use it for mouth-to-mouth resuscitation.) We breathe in roughly 10 kg air/day, with ∼2 kg O_2/day. The lungs absorb about 0.5 kg O_2/day (400 L). We exhale air with ∼0.5 kg water vapor/day.

Because we inspire and expire air at the same rate (if not where would the difference go?), it is clear the body uses ∼0.3 L oxygen/min during usual sedentary activity, delivered by the ∼5–6 L of blood pumped per min. We have called this rate of oxygen consumption in the body dV_{O_2}/dt in Chaps. 6 and 8. During aerobic exercise dV_{O_2}/dt increases linearly with cardiac output Q_t (see Fig. 8.27, and (6.18) and (8.33)). The maximum rate of oxygen usage is ≈2.8 L/min for a person of average fitness and ≈4 L/min for a highly fit person. This assumes the lungs bring in air at a rate fast enough to maintain the needed oxygenation of arterial blood.

Gauge pressures, relative to atmosphere, are usually used in discussing breathing. Two roughly equal types of units are commonly used, mmHg and cmH_2O, with 1 mmHg = 1.36 cmH_2O.

9.1 Structure of the Lungs

Air is inhaled through the nose or mouth and then through the pharynx, larynx, and the trachea (windpipe) (Fig. 9.1). The trachea divides into the right and left bronchus (Fig. 9.2), each of which continues to bifurcate into smaller and smaller bronchi and bronchioles over 23 levels of bifurcation ($2^{24} = 1.6 \times 10^8$) (Table 9.1, Figs. 9.2 and 9.3) until they form alveoli (which is the plural of alveolus) (al-vee-oh'-lie (lus)), which are the actual operating units of the lungs. The average diameter of the airways decreases with generation z, as $d(z) = 2^{-z/3}d(0)$ until generation 16. This relation is the optimal design of a branched system of tubes in hydrodynamics. There are about 3×10^8 alveoli, each $\sim$0.2–0.3 mm in diameter, with walls that are

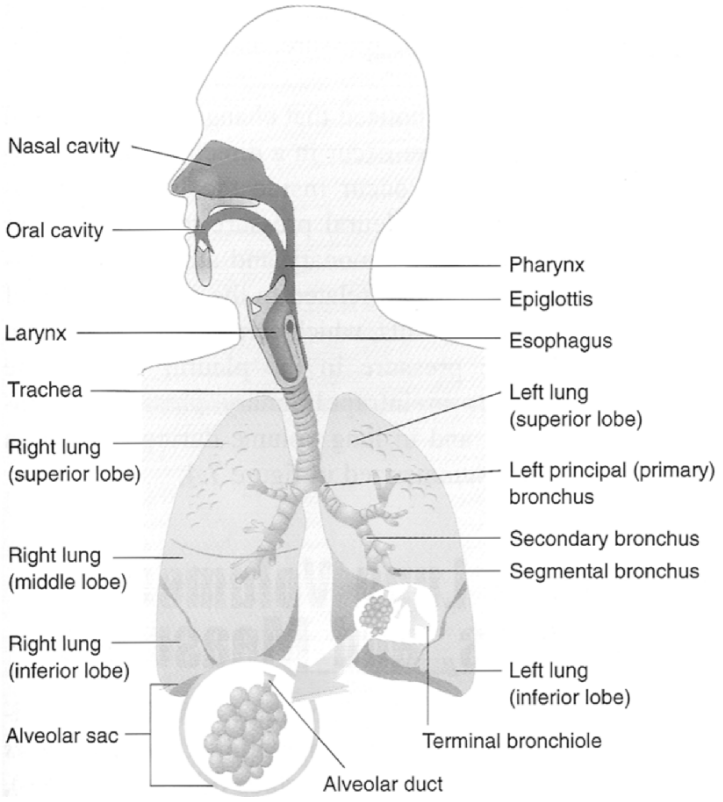

Fig. 9.1. Diagram of parts of the respiratory system. (These components are also important in voice production (Chap. 10). The vocal cords (or vocal folds) used in speaking are in the larynx.) (From [425]. Used with permission)

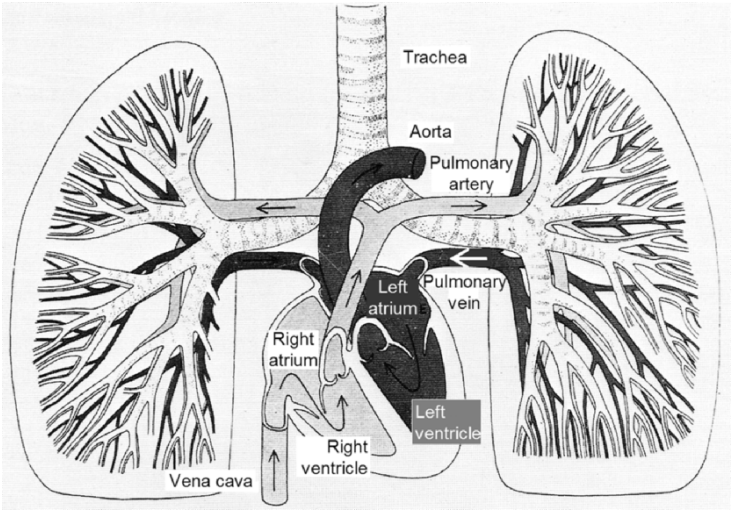

Fig. 9.2. The relationship between the lung and heart is shown. The first few generations of the branching of the air vessels in the lungs, pulmonary arteries, and the pulmonary veins are shown. These three systems can be called the three "trees" of the lung. Note that the pulmonary arteries are close to the bronchi, while the pulmonary veins stand alone. (From [427])

$\sim$0.4 μm thick. They are in contact with blood in the pulmonary capillaries (Fig. 9.4), which themselves form after subdividing in 17 branches (Table 9.2, Figs. 9.2 and 9.5). Oxygen diffuses from the alveoli to the red blood cells, while carbon dioxide diffuses from the blood into the air in the alveoli. The total surface area of the alveoli is $\sim$80 m^2 (ranging from 50–100 m^2). The total external surface area of the lungs is only $\sim$0.1 m^2, so subdividing into alveoli results in a tremendous increase in the surface area in contact with the blood, by a factor of almost 1,000. This is also the factor by which the oxygen intake increases. Without this, we would never even come close to meeting our metabolic needs for oxygen. Our chests expand when we breathe because incoming air filling the alveoli makes each one bigger, just as with ordinary bubbles.

The circulatory system is the conduit for the transfer of O_2 and CO_2 between the alveoli and tissues, and so we should track the partial pressure in each system. Within the alveoli the partial pressure of O_2 is $\simeq$105 mmHg, which is smaller than that in the atmosphere (159 mmHg = 21% of 760 mmHg) because of the dead volume in the respiratory system. The partial pressure of O_2 blood in the pulmonary capillaries increases from 40 to $\simeq$100 mmHg after O_2 is transferred from the alveoli, and this is the partial pressure in the pulmonary veins and systemic arteries. The partial pressure of O_2 in tissue is 40 mmHg, so that after transfer of O_2 from the capillaries to surrounding

Table 9.1. Approximate quantification of the bronchial system. (Using data from [426, 440]. Also see [441, 442])

pulmonary branch	generation z	branch diameter (mm)	branch length (mm)	total cross-sectional area (cm^2)	volume (cm^3)	air speed (cm/s)
trachea	0	18.0	120.0	2.5	31	393
main bronchus	1	12.2	47.6	2.3	11	427
lobar bronchus	2	8.3	19.0	2.1	4.0	462
	3	5.6	7.6	2.0	1.5	507
segmental bronchus	4	4.5	12.7	2.5	3.5	392
	5	3.5	10.7	3.1	3.3	325
bronchi	6	2.8	9.0	4.0	3.5	254
w/cartilage in wall	7	2.3	7.6	5.1	3.8	188
	8	1.86	6.4	7.0	4.4	144
	9	1.54	5.4	9.6	5.2	105
	10	1.30	4.6	13	6.2	73.6
terminal bronchus	11	1.09	3.9	20	7.6	52.3
	12	0.95	3.3	29	9.8	34.4
bronchioles	13	0.82	2.7	44	12	23.1
w/muscle in wall	14	0.74	2.3	69	16	14.1
	15	0.66	2.0	113	22	8.92
terminal bronchiole	16	0.60	1.65	180	30	5.40
respiratory bronchiole	17	0.54	1.41	300	42	3.33
respiratory bronchiole	18	0.50	1.17	534	61	1.94
respiratory bronchiole	19	0.47	0.99	944	93	1.10
alveolar duct	20	0.45	0.83	1,600	139	0.60
alveolar duct	21	0.43	0.70	3,200	224	0.32
alveolar duct	22	0.41	0.59	5,900	350	0.18
alveolar sac	23	0.41	0.50	12,000	591	0.09
alveoli, 21 per duct		0.28	0.23		3,200	

The air speed is assumed to be 1 L/s. The data include that for both lungs. The number in each generation is 2^z (for generations $z = 0$–23), and 300×10^6 for the alveoli.

tissues, the partial pressure in the systemic veins and pulmonary arteries is also $\simeq$40 mmHg – and then it is again increased to 100 mmHg in the lungs.

Similarly, within the alveoli the partial pressure of CO_2 is $\simeq$40 mmHg; this is much larger than that in the atmosphere ($\sim$0.25 mmHg), again because of the dead volume. The partial pressure of CO_2 blood in the pulmonary capillaries decreases from 46 to $\simeq$40 mmHg after CO_2 is transferred to the alveoli, and this is the partial pressure in the pulmonary veins and systemic arteries. The partial pressure of CO_2 in tissue is 46 mmHg, so that after transfer of CO_2 into the capillaries from the tissues, the partial pressure in the systemic veins and pulmonary arteries is also $\simeq$46 mmHg – and then it is again decreased to 40 mmHg in the lungs.

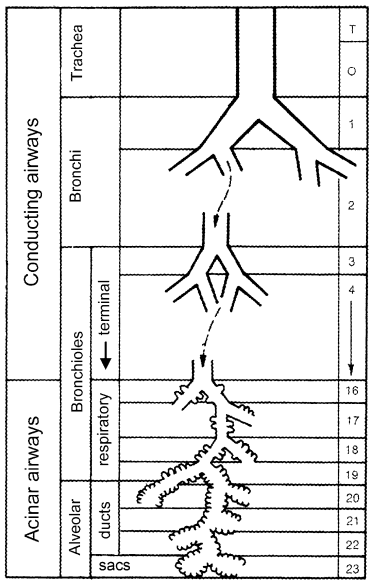

Fig. 9.3. Bifurcations of lung airways, showing generation number z. (From [436])

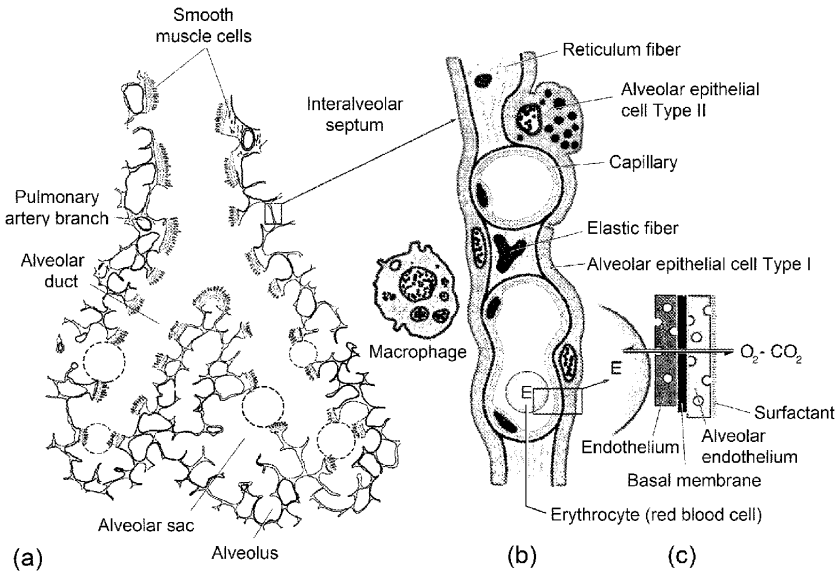

Fig. 9.4. The details of the alveolar bifurcation are shown in (**a**). These alveoli are sacs imbedded in capillary beds. The details of the interaction between the alveoli and capillaries are depicted in (**b**) and (**c**). (From [436])

Table 9.2. Branching structure of the pulmonary arterial network. (Using data from [426, 438])

pulmonary branching order	number of branches of each order	vessel length (mm)	vessel diameter (mm)
1	1	90.5	30.0
2	3	32.0	14.83
3	8	10.9	8.06
4	20	20.7	5.82
5	66	17.9	3.65
6	203	10.5	2.09
7	675	6.6	1.33
8	2,290	4.69	0.85
9	5,861	3.16	0.525
10	17,560	2.10	0.351
11	52,550	1.38	0.224
12	157,400	0.91	0.138
13	471,300	0.65	0.086
14	1,411,000	0.44	0.054
15	4,226,000	0.29	0.034
16	12,660,000	0.20	0.021
17	300,000,000	0.13	0.013

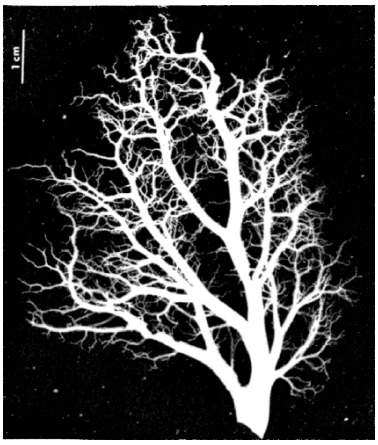

Fig. 9.5. A silicone elastomer cast of the venous tree of the lung of a cat. The venous pressure was $-7\,\mathrm{cmH_2O}$ ($= -5\,\mathrm{mmHg}$), the airway pressure was $10\,\mathrm{cmH_2O}$ ($= 7\,\mathrm{mmHg}$), and the pleural pressure was $0\,\mathrm{cmH_2O}$. (From [427])

9.2 The Physics of the Alveoli

The alveoli are similar to interconnected bubbles. Inside them the pressure is P_{in} and outside the pressure is P_{out}, with $\Delta P = P_{\text{in}} - P_{\text{out}}$, and they have a radius R. The Law of Laplace for a sphere (7.9) is

$$\Delta P = \frac{2T}{R}, \tag{9.1}$$

where T is the tension in the sphere walls. The main source of this tension in the alveoli is not within the walls but on the surfaces. This contribution is called the surface tension γ, which has the same units as T – of force/length or energy/area. In typical bubbles, such as soap bubbles, both surfaces contribute the same surface tension and so T is replaced by 2γ. Therefore we find

$$\Delta P = P_{\text{in}} - P_{\text{out}} = \frac{4\gamma}{R}. \tag{9.2}$$

For the water/air interface $\gamma \simeq 7.2\times10^{-4}\,\text{N/m}$ (Table 7.2). In alveoli, however, only the surface tension of the inner surface is really important because it is a fluid/air interface with larger surface tension than the fluid/fluid interface of the outer surface, and so

$$\Delta P_{\text{alveoli}} = P_{\text{in}} - P_{\text{out}} = \frac{2\gamma}{R}. \tag{9.3}$$

There is an apparent instability that seemingly leads to an unreasonable situation in interconnected bubbles or alveoli. Consider two bubbles that are initially not interconnected, as in Fig. 9.6, because there is a plug between them. Bubble #1 has an internal pressure P_1 and radius R_1, and Bubble #2 has an internal pressure P_2 and radius R_2. (Because the difference between the pressure inside and outside the bubble is what is significant, the external

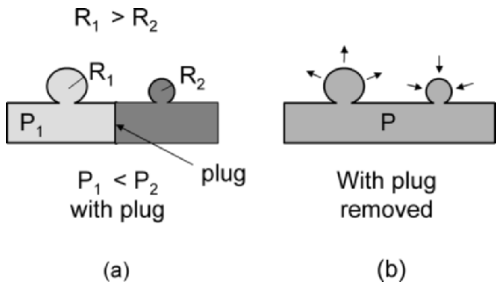

Fig. 9.6. Instability of bubbles, according to the Law of Laplace. This assumes that the surface tension does not change with bubble (or alveolus) radius. The external pressure is 0

pressure is equal to zero.) In equilibrium, the internal pressure $P_{\text{internal}} = 4\gamma/R$ for each bubble. (Whether this factor is 4 or 2 is not significant here.) Say Bubble #2 is the smaller bubble. Because $R_2 < R_1$, in equilibrium $P_2 > P_1$; the smaller bubble has the higher internal pressure. If the plug is opened, air will flow from higher pressure to lower pressure, and therefore from the smaller bubble to the larger bubble. The loss of air in Bubble #2 makes it smaller. With this smaller radius, the equilibrium internal pressure increases. Because this pressure is still higher than in Bubble #1, air continues to flow from the smaller bubble to the larger bubble, until it collapses.

This implies that the largest of the hundreds of millions of alveoli would get ever larger at the expense of all of the smaller ones and the system of alveoli we have described for the lungs could not be stable. What is wrong? There is no error in our reasoning; however, we have made one assumption that is not accurate for alveoli. We implicitly assumed that the surface tension is not a function of radius R. There is a surfactant on the surfaces of the alveoli of healthy people, containing dipalmitoyl phosphatidycholine or DPPC, that causes $\gamma(R)$ to decrease for decreasing R. With $\Delta P = 2\gamma(R)/R$, as R of the smaller bubble or alveolus becomes smaller in Fig. 9.6, eventually $\gamma(R)$ decreases with smaller R faster than R does itself, as in Fig. 9.7, so ΔP begins to decrease with smaller R. Such a system of interconnected alveoli is stable.

We can see how such a dependence of $\gamma(R)$ can occur with the following model. The surface of an alveolus can be covered either with a lipoprotein or by water; the surface tension of the lipoprotein is much lower ($\gamma_{\text{lung}} = 1 \times 10^{-3}\,\text{N/m}$) than that of water ($\gamma_{\text{water}} = 7.2 \times 10^{-2}\,\text{N/m}$ (= 72 dynes/cm)). Assuming the alveolus is spherical, for one particular radius R_0 there is exactly one monolayer of lipoprotein on the whole surface and at that radius the surface tension is γ_{lung} over the $4\pi R_0^2$ surface area. If this alveolus becomes smaller, so $R < R_0$, it has several monolayers of lipoprotein on its surface and

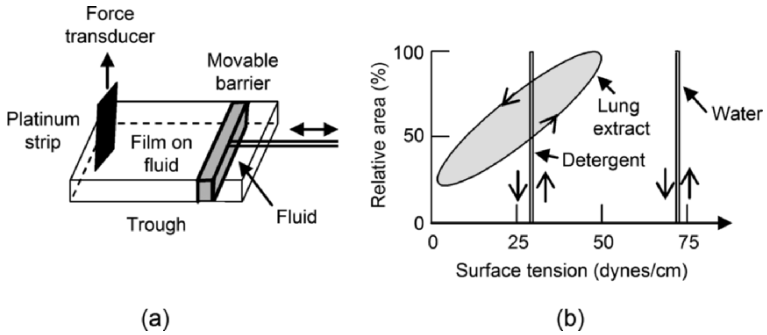

Fig. 9.7. Surface tension on alveoli walls (lung extract) in (**b**), as measured by the surface balance in (**a**) which measures surface tension vs. area. Similar measurements for detergent and water are also shown. (Based on [430, 443])

its surface tension is still γ_{lung}, and so

$$\gamma(R) = \gamma_{\text{lung}} \quad \text{for } R \leq R_0. \tag{9.4}$$

If this same alveolus instead becomes larger, so $R > R_0$, it has a monolayer of lipoprotein over only a portion of its surface (of surface area $4\pi R_0^2$ because the layer cannot become smaller than a monolayer) and water over the rest of the surface (of area $4\pi R^2 - 4\pi R_0^2$). So the average surface tension is

$$\gamma(R) = \frac{4\pi R_0^2 \gamma_{\text{lung}} + (4\pi R^2 - 4\pi R_0^2)\gamma_{\text{water}}}{4\pi R^2} \quad \text{for } R > R_0 \tag{9.5}$$

or

$$\gamma(R) = \gamma_{\text{water}} - \frac{R_0^2}{R^2}(\gamma_{\text{water}} - \gamma_{\text{lung}}) \quad \text{for } R > R_0. \tag{9.6}$$

This approaches the much smaller γ_{water} for $R \gg R_0$ at a rate that is faster than $1/R$, so the alveoli will be stable.

Because this lipoprotein is only on one of the surfaces, the stability condition is $\Delta P = 2\gamma/R$. For $R > R_0$, there is a stable equilibrium when $\mathrm{d}(\Delta P)/\mathrm{d}R = \mathrm{d}[2\gamma_{\text{water}}/R - 2(R_0^2/R^3)(\gamma_{\text{water}} - \gamma_{\text{lung}})]/\mathrm{d}R = 0$ or

$$\frac{\mathrm{d}(\Delta P)}{\mathrm{d}R} = -\frac{2\gamma_{\text{water}}}{R^2} + 6\frac{R_0^2}{R^4}(\gamma_{\text{water}} - \gamma_{\text{lung}}) = 0 \tag{9.7}$$

or

$$R_{\text{eq}} = \sqrt{3\frac{\gamma_{\text{water}} - \gamma_{\text{lung}}}{\gamma_{\text{water}}}}\, R_0. \tag{9.8}$$

Because $\gamma_{\text{water}} \gg \gamma_{\text{lung}}$, the equilibrium radius $R_{\text{eq}} \simeq \sqrt{3}R_0$.

Figure 9.7 shows that this surface tension of the surfactant in the lung decreases from $5 \times 10^{-2}\,\text{N/m}$ (50 dynes/cm) to zero as the area of the film gets smaller. Alveoli are typically stable at approximately 1/4 of their maximum size.

One function of the surfactant is to provide alveolus stability. Another function is to lower the amount of force needed to be supplied by the diaphragm to inflate the alveoli. With $\gamma_{\text{water}} = 7.2 \times 10^{-2}\,\text{N/m}$ and $R = 0.05\,\text{mm}$ of the alveoli when they are collapsed (and need to be inflated), (9.3) gives $\Delta P_{\text{alveoli}} = 2.9 \times 10^3\,\text{N/m}^2 = 22\,\text{mmHg}$. The area of an adult diaphragm muscle is about $500\,\text{cm}^2$, so the force it needs to exert to expand the alveoli for breathing is $\sim$150 N – which corresponds to a weight of 15 kg. With the lower surface tension of the lung surfactant, this force is over an order of magnitude smaller and breathing is easier, especially for infants. This explains why people with insufficient surfactant – with hyaline membrane disease – have difficulty breathing.

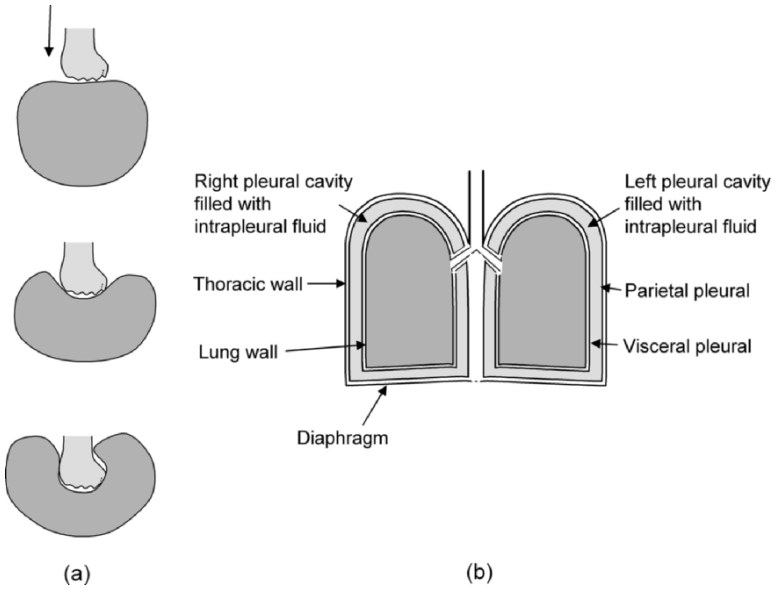

Fig. 9.8. (**a**) Pushing a fist into a balloon is analogous to the lungs in the pleural cavity. (**b**) Schematic of the lungs in the pleural cavities. (Based on [439])

9.3 Physics of Breathing

Each lung is surrounded by a sac membrane within the thoracic cavity. We can picture the pleural sac as a balloon, as in Fig. 9.8, filled with intrapleural fluid. The inside wall of this sac, the visceral pleura (membrane), attaches to the outer lung wall. The outside wall of this sac, the parietal pleura (membrane), attaches to the thoracic wall. It is the springiness of the lung that pulls the two pleural membranes apart, and this causes a slight decrease of pressure of the pleural sac relative to atmospheric pressure of $-4\,\mathrm{mmHg}$ to $-6\,\mathrm{mmHg}$. This pressure difference is what keeps the lungs expanded, and keeps them from collapsing. The mechanical "driving force" in controlling lung volume is the *transpulmonary pressure*, which is the difference in pressure in the alveoli in the lungs and that around the lung in the pleural sac, which is called the *intrapleural (or pleural) pressure*. (The alveolar and pleural pressures are gauge pressures, referenced to atmospheric pressure.)

The lungs are expanded and contracted by the motion of structures surrounding them by way of inspiratory and expiratory muscles. This occurs in two ways (Fig. 9.9), of which only the first is used during quiet breathing (1) The diaphragm moves downward to lengthen the chest cavity (by pulling the bottom of the lungs downward) during inspiration. During quiet breathing, the lungs contract by the natural elastic recoil of the lungs and chest wall, with the diaphragm relaxed, while in heaving breathing this contraction is

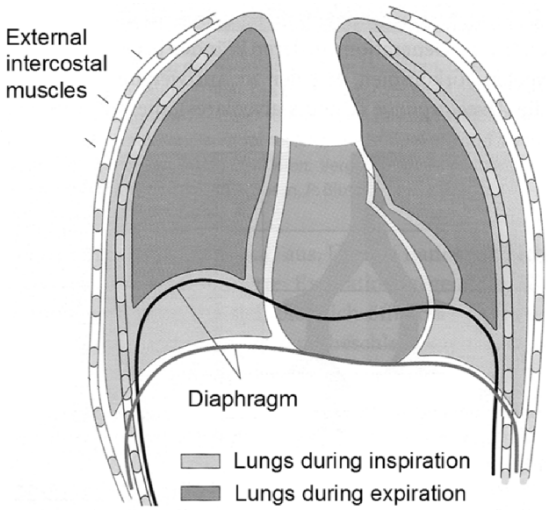

Fig. 9.9. Expansion and contraction of the thoracic cage during expiration and inspiration, showing the ribs, lungs and heart, the external intercostal muscles (that contract during inspiration to elevate the rib cage and widen it laterally so the cage increases in all three dimensions), and the diaphragm (that contracts to increase the vertical dimension of the cage during inspiration). (From [437])

accelerated by the contraction of the abdominal muscles that push the abdominal contents and then the diaphragm upward to shorten the chest cavity. (2) The ribs are elevated by the neck muscles to increase the anteroposterior (front-to-back) diameter of the chest cavity and are depressed (lowered) by the abdominal recti to decrease it. This causes chest cavity expansion and contraction, respectively, because the ribs slant outward and have larger transverse cross-sectional areas in the lower sections; this can increase the anterior–posterior chest thickness by about 20% during inspiration.

How does this help bring air into the lungs? Before inspiration, there is atmospheric pressure in the lungs. The attractive force of the visceral pleura for the parietal pleura and the outward force of the outer lung wall due to the lower-than-atmospheric pressure in the pleural sac ($\sim -4\,\mathrm{mmHg}$) cause each lung to expand. In equilibrium their sum is balanced by the tendency of the lungs to contract due to their springiness. This preinspiration force balance is shown in Fig. 9.10. They are no longer in balance during inspiration.

The steps in inspiration (inhaling) are shown in Fig. 9.11. The inspiratory muscles (diaphragm and external intercostals) increase the dimensions of the rib cage (the thoracic cavity). This causes the visceral and parietal pleurae to separate. The lung volume then increases because (1) the attraction of the visceral and parietal pleurae increases as they are separated further and (2) this separation causes $P_{\mathrm{lung}} - P_{\mathrm{pleura}}$ to decrease even more, from ~ -4 to

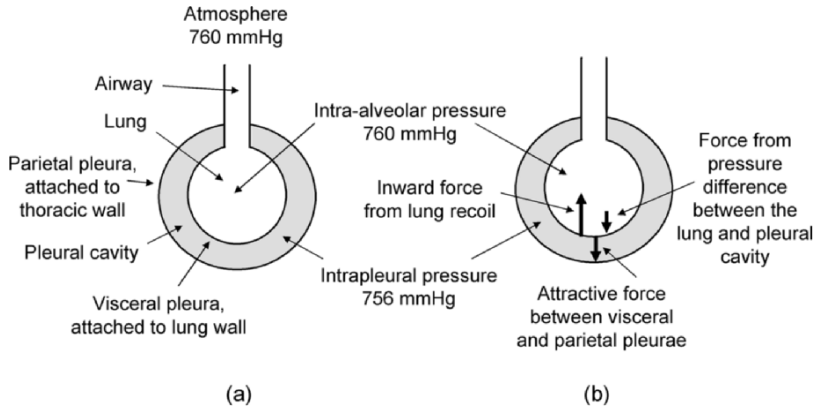

Fig. 9.10. Force balance of the visceral pleura/outer lung wall during preinspiration. Note that the forces are really normal to the wall everywhere, not just at the bottom as depicted. (Based on [439])

~ -6 mmHg (i.e., from $\sim$756 to 754 mmHg absolute pressure). Because both of these forces in the direction of lung expansion increase, they now overcome the springiness of the lungs that favors lung contraction – and the lung expands. The pressure in the lungs and alveoli decreases from $\sim$0 to ~ -1 mmHg (i.e., from $\sim$760 to 759 mmHg absolute pressure), and then air flows from the

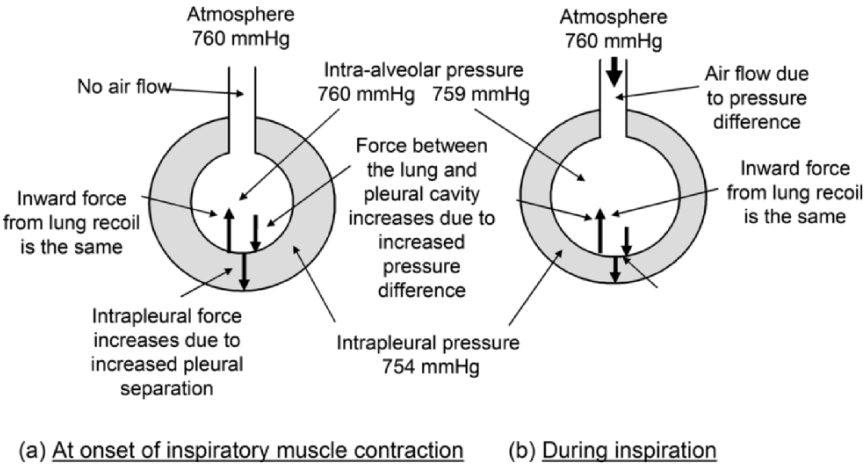

Fig. 9.11. (**a**) Force imbalance at the onset of inspiratory muscle contraction (and expansion of the thoracic wall/parietal pleura) leads to a (**b**) subatmospheric pressure in the lungs and flow of air into the lungs. (Compare this to the preinspiration force balance in Fig. 9.10). (Based on [439])

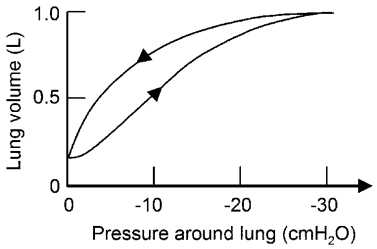

Fig. 9.12. The measurement of the pressure–volume curve of an excised lung, which shows hysteresis in inflation and deflation. (Based on [443])

mouth and nose into the lungs. During normal breathing exhaling is automatic, requiring no contraction by muscles. Muscle contraction is necessary during heavy exercise to inhale more fresh air and to actively exhale stale air.

9.4 Volume of the Lungs

The volume of the lungs depends on the transpulmonary pressure, as is seen in Fig. 9.12 for an excised lung. The inflation and deflation curves are not the same; as in Chap. 4, this is called hysteresis.

The volume of the lungs during different stages of normal and deep breathing is a good diagnostic of lung functionality. It is easily measured using a *spirometer* (Fig. 9.13). Figure 9.13 shows one such measurement during different types of breathing. In this example, during normal breathing the lung volume is seen to oscillate between 3.2 L after normal inspiration and 2.2 L after normal expiration. The difference is the tidal volume (TV) ($\sim$1 L), which is the usual lung volume used during breathing when at rest. The volume after normal expiration is the functional residual capacity (FRC). After a deep inspiration the lung volume is the total lung capacity (TLC), $\sim$6 L. This exceeds the volume after normal inspiration by the inspiratory reserve volume (IRV, which is also one of the author's nicknames). After a deep expiration, the remaining volume is the residual volume (RV), $\sim$1 L. The difference in lung volumes after deep inspiration and deep expiration is the vital capacity (VC) $\sim$5 L, which also equals the total lung capacity minus the residual volume. After deep expiration the lung volume is smaller than that after normal expiration by the expiratory reserve volume (ERV), which also equals the functional residual capacity minus the residual volume.

The vital capacity is an important measure of how well the lungs are functioning. (More importantly, you need a robust vital capacity to inflate balloons.) The functional reserve capacity is the volume of stale air that normally mixes with new air (the tidal volume). There is also dead space. Some is anatomic (0.15 L), due to the trachea and bronchii, and some is physiological alveoli dead space, where the alveoli have no access to blood.

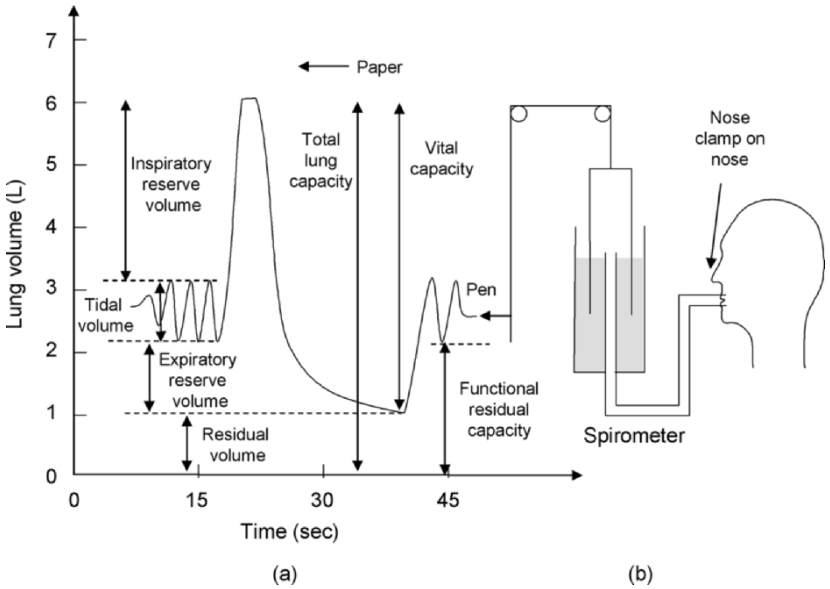

Fig. 9.13. (**a**) Lung volume changes during breathing cycles, (**b**) along with a schematic of a spirometer. (Based on [422, 443])

We know that we inhale air that is 80% N_2/20% O_2/0% CO_2, it mixes with stale air, and after diffusion across the alveoli walls we exhale air that is 80% N_2/16% O_2/4% CO_2. *What is the composition of this stale air?* Let us call its composition 80% N_2/x% O_2/y% CO_2. After inspiration the lungs have a tidal volume of 80% N_2/20% O_2/0% CO_2 air that has mixed with a functional reserve capacity of 80% N_2/x% O_2/y% CO_2 air. If the tidal volume is a fraction α of this volume, the functional reserve capacity volume fraction is $1-\alpha$. After inhalation the composition of air in the lungs is 80% $N_2/[20\alpha + (1-\alpha)x]$% $O_2/(1-\alpha)y$% CO_2. Say a fraction β of all of the inhaled air is absorbed by the lungs. This means that a fraction β of the 20α% O_2 in the lungs is absorbed, which is $20\alpha\beta$% O_2. There is an equal $20\alpha\beta$% increase in CO_2 that diffuses into the lungs. So, after the oxygen and carbon dioxide transfer, the air in the lungs has a composition of 80% $N_2/[20\alpha - 20\alpha\beta + (1-\alpha)x]$% $O_2/[20\alpha\beta + (1-\alpha)y]$% CO_2, which is exhaled. Therefore, the oxygen and carbon dioxide fractions are, respectively,

$$16\% = [20\alpha - 20\alpha\beta + (1-\alpha)x]\% \tag{9.9}$$

$$4\% = [20\alpha\beta + (1-\alpha)y]\%. \tag{9.10}$$

The tidal volume fraction is $\alpha = 1/3$ and the fraction of inhaled oxygen that is absorbed by the lungs is $\beta = 1/4$. This gives $x = 16.5$ and $y = 3.5$, so the stale air in the lungs has a composition 80% N_2/16.5% O_2/3.5% CO_2.

(This air is clearly oxygenated enough to be useful during mouth-to-mouth resuscitation.)

9.5 Breathing Under Usual and Unusual Conditions

9.5.1 Flow of Air During Breathing

During inspiration, air flows because the pressure is lower in the lungs and alveoli by a positive amount ΔP than in the atmosphere. The amount of air that flows is determined by the resistance and compliance of the respiratory system.

The compliance of the lung is $\sim 0.2\,\mathrm{L/cmH_2O}$ and it decreases for a normal person with higher expanding pressures, as is seen by the decreasing slope in Fig. 9.12. In trying to evaluate lung performance, the *specific compliance* of the lung is perhaps more meaningful, for which the lung compliance is normalized by a characteristic of the person's size, such as a characteristic lung volume (FRC, VC, TLC, etc.), the lung dry weight, or the body weight. The elasticity of the chest, as well as that of the lung, contributes to the lung compliance.

Airway resistance is dominant in the generation of the intermediate sized bronchii, as seen in Fig. 9.14a and Problem 9.21. Poiseuille's Law (7.24) can be used to calculate the airway resistance in the lungs (Problems 9.21–9.23) and other passages (Problems 9.16 and 9.18). The total airway resistance is typically $\sim 2\,\mathrm{cmH_2O/(L/s)}$ during normal breathing and it decreases with increasing lung size (Fig. 9.14b). (It is measured as in Fig. 9.17 later).

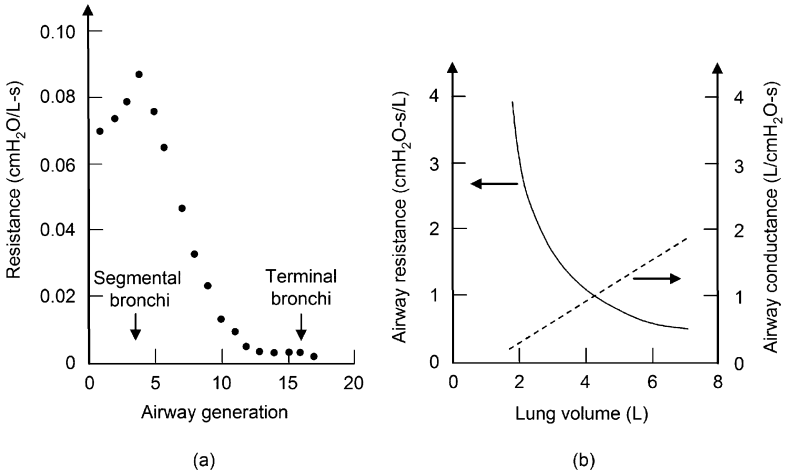

Fig. 9.14. (**a**) Airway resistance for each bronchus generation, and (**b**) total airway resistance and conductance vs. lung volume. (Based on [443], from (a) [433], (b) [421])

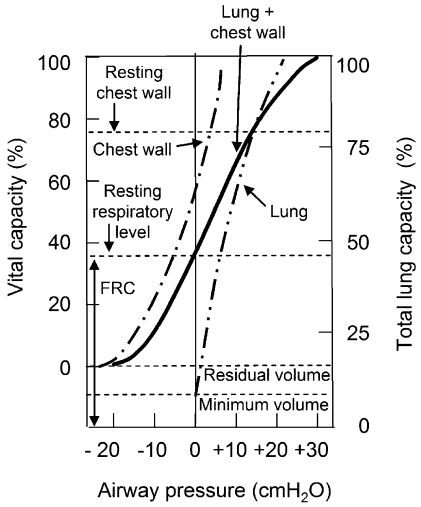

Fig. 9.15. Pressure–volume curves of the lungs, chest wall (no lungs), and chest wall with lungs, with relaxed respiratory muscles. The lung data are the same as those in Fig. 9.12, except no hysteresis is shown and the airway pressure is of the opposite sign to the pressure around the lung. FRC is the functional residual capacity. The measurement is made with a spirometer-like apparatus, similar to that in Fig. 9.13b. (Based on [443])

This airway resistance is about 80% of the total *pulmonary resistance*. The other 20% is due to viscous forces in chest and lung wall movement, and this is called *tissue resistance*.

The elastic properties of the thoracic cage (with the chest well) are important in breathing, as are those of the lungs. Figure 9.15 shows the volume of the lungs, chest walls alone, and the lungs in the chest wall as a function of pressure. This is measured after inspiration or expiration with a spirometer and subsequent relaxing of respiratory muscles. At every volume the pressure (the relaxation pressure) of the lung/chest wall combination is the sum of those for the lungs and chest walls separately.

Is the pressure difference between the alveoli and atmosphere large enough to drive the right amount of air into our lungs each breath? (Under normal conditions, it had better be.)

During each breath, this pressure difference starts at zero, increases to a maximum $\Delta P_{\max}$, and then decreases to zero again at the end of the breath. Let us determine the average pressure difference in this sequence. We will model inhalation as a half cycle of a sine wave with: $\Delta P(t) = \Delta P_{\max} \sin(2\pi f t) = \Delta P_{\max} \sin(\pi t / T_{\text{half period}})$, which lasts a half-cycle time $T_{\text{half period}} = 1/(2f)$. (The parameter f is the same as the breathing or respiratory rate only if the inhalation and exhalation times are the same.) For

$f = 0.25$ Hz, $T_{\text{half period}} = 2\,\text{s}$. The average pressure difference during this inhalation is

$$\Delta P_{\text{av}} = \frac{1}{T_{\text{half period}}} \int_0^{T_{\text{half period}}} \Delta P_{\max} \sin(\pi t / T_{\text{half period}}) \mathrm{d}t \tag{9.11}$$

$$\begin{aligned} \Delta P_{\text{av}} &= \frac{\Delta P_{\max}}{T_{\text{half period}}} \frac{T_{\text{half period}}}{\pi} \left(\cos(\pi T_{\text{half period}}/T_{\text{half period}}) - \cos(0)\right) \\ &= \frac{2}{\pi} \Delta P_{\max}, \end{aligned} \tag{9.12}$$

so for $\Delta P_{\max} = 1.1\,\text{mmHg}$, we see that $\Delta P_{\text{av}} = 0.7\,\text{mmHg}$.

Let us say that the inflow of air per breath is V_{in}. Then the average flow rate is $Q_{\text{av}} = V_{\text{in}}/T_{\text{half period}}$. For $V_{\text{in}} = 0.5$ L, this is $0.25\,\text{L/s}$. If the flow rate is proportional to the pressure drop, Q and ΔP have the same dependence on time and so $Q_{\max} = (\pi/2)Q_{\text{av}}$, which is $(\pi/2)0.25\,\text{L/s} = 0.4\,\text{L/s}$, and $Q(t) = Q_{\max}\sin(2\pi f t) = Q_{\max}\sin(\pi t/T_{\text{half period}})$. Moreover, $Q = \Delta P/R_{\text{flow}}$, where R_{flow} is the total resistance to flow in the nasal passages, trachea, and so on. The resistance to flow is $R_{\text{flow}} = \Delta P/Q$, and so using average values we see that $R_{\text{flow}} = 0.7\,\text{mmHg}/(0.25\,\text{L/s}) = 3.7 \times 10^5$ Pa-s/m^3.

Is the flow laminar or turbulent? If the trachea has a radius of 9 mm, the air flows at a maximum speed $u_{\max} = Q_{\max}/A = (400\,\text{cm}^3/\text{s})/(\pi(0.9\,\text{cm})^2) = 160\,\text{cm/s}$. The Reynolds number (7.11) is $Re = \rho u d/\eta$. Using the mass density of air at body temperature $\rho = 1.16 \times 10^{-3}\,\text{g/cm}^3$ and the air viscosity 2×10^{-5} Pa-s $= 2 \times 10^{-5}(\text{N/m}^2)\text{s}$, we find that $Re = (1.16 \times 10^{-3}\,\text{g/cm}^3)(160\,\text{cm/s})(1.8\,\text{cm})/(2 \times 10^{-4}(\text{dyne/cm}^2)\text{s}) = 1{,}600$, so the flow would generally be expected to be laminar in the trachea, as well as in the nasal passages and pharynx. However, some turbulence is expected because the walls of these passages are not smooth.

9.5.2 Mechanical Model of Breathing and Model Parameters

We have just examined only the resistance to flow during breathing. Figure 9.16b shows a more complete mechanical model of the lungs and breathing. It is a compliance vessel described by $V(t) = V_{\text{d}} + C_{\text{flow}}P(t)$, attributed to the springiness of the lungs, in series with an inertial element. They are in parallel with a resistive element, attributed to the airway resistance we just examined. These model elements are driven by a pressure determined by the inspiratory muscles.

9.5.3 Inspiration/Expiration Cycle

Figure 9.17 shows the lung pressure, rate of flow of air into the lungs, and lung volume vs. time during a cycle of inspiration and expiration. If the effect of airway resistance were neglected, the alveolar pressure would be zero and the intrapleural pressure would follow the broken curve, which is determined by the elastic recoil of the lung.

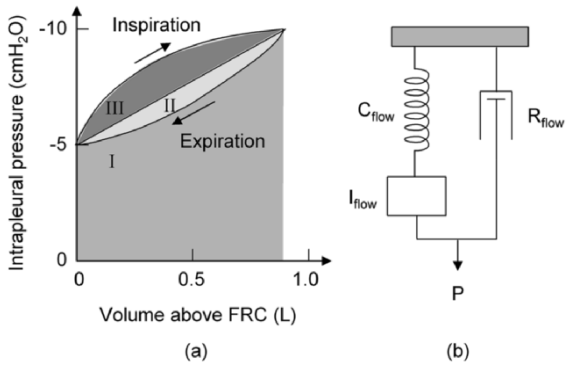

Fig. 9.16. (**a**) Work done during inspiration (areas I + II + III) and work recovered during expiration (area I). With no viscous, resistive forces, the work in inflating the lung would be areas I + II, and this is associated with the lung compliance. The extra work done overcoming respiratory flow resistance is area III. (**b**) Mechanical model of breathing has the lumped compliance (elastance) C_{flow}, resistance R_{flow}, and inertance I_{flow}, and P represents the inspiratory muscles. The inertance can be neglected except for large flows. (Based on [430, 444])

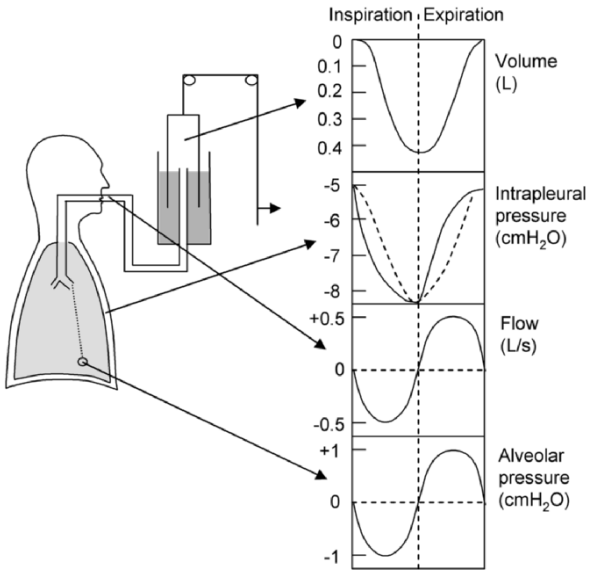

Fig. 9.17. Lung volume, intrapleural pressure, flow, and alveolar pressure vs. time during a breathing cycle. If the effect of airway resistance were neglected, the alveolar pressure would be zero and the intrapleural pressure would follow the broken curve. (Based on [443])

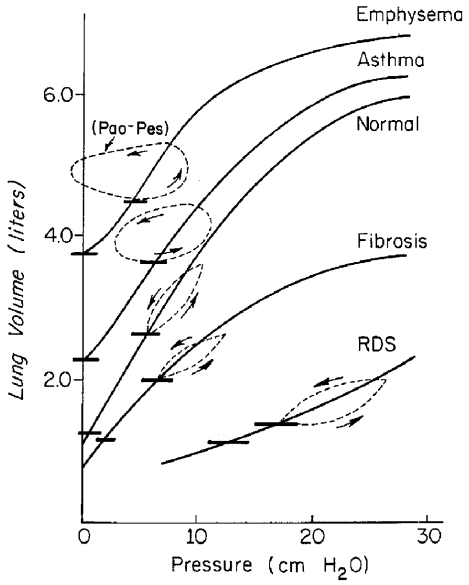

Fig. 9.18. Typical lung volume vs. pressure for patients with various respiratory conditions. The monotonically increasing curves are for static conditions, with the lower horizontal bar being the residual volume and the upper horizontal bar the functional residual capacity. Representative dynamic breathing loops (enclosed *dashed curves*) for tidal volume breathing are shown for each condition. (Reprinted from [430], with permission of Elsevier)

It is also instructive to plot these variables as functions of each other. Lung volume is plotted vs. lung pressure for all times during a breathing cycle in the dashed cycle trajectories in Fig. 9.18. Time is an implicit variable along the trajectories. Such plots are useful because the model of the lungs includes a compliance vessel in which volume and pressure are interrelated. The differences in various modes of breathing are easily seen in such plots.

9.5.4 Breathing with a Diseased Lung

The static and dynamic pressure–volume curves in Fig. 9.18 indicate how different diseases affect lung compliance, lung volume, and airway resistance. *Obstructive* disorders are due to airway obstructions, and include chronic bronchitis (excessive mucus production in the bronchial tree), emphysema (enlargement of air spaces after the terminal bronchiole, with the destruction of respiratory system walls, such as those of the alveoli), and asthma (widespread narrowing of airways, sometimes spontaneously). Such obstructions (Fig. 9.19) can be due to excessive secretions (due to chronic bronchitis), thickening of airway walls (edema or muscle hypertrophy), and outside

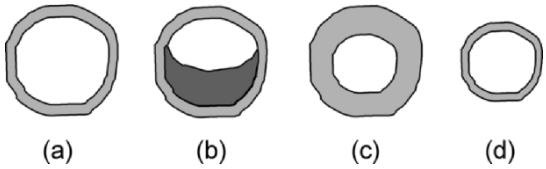

Fig. 9.19. (**a**) Normal airway and obstructed airways due (**b**) blocking, (**c**) airway wall thickening, and (**d**) outside abnormality. Also see Fig. 9.22. (Based on [444])

abnormalities (edema, enlarged lymph nodes, or destruction of lung alveoli tissue as in emphysema). (Edema is an excessive accumulation of fluid in tissue spaces or a body cavity.) These obstructions and the loss of small airways due to the destruction of lung tissue all increase airway resistance. The breakdown of elastic alveoli walls also reduces the springiness, and therefore also the compliance. *Restrictive* disorders are those in which the expansion of the lung is restricted. The decreased compliance (slope) in *pulmonary fibrosis* and idiopathic respiratory distress syndrome (RDS) is clear in Fig. 9.18. They are characterized by a lower vital capacity, but airway resistance (per lung volume) is not increased. There are also *vascular disorders*, such as pulmonary edema, which is the abnormal accumulation of fluid in the lungs.

Poor breathing due to a diseased lung is manifest in different ways. As seen in Fig. 9.20, the inspired volume is very low if the compliance is less than normal and/or the *airway resistance* is greater than normal. A spirometer with a low resistance can also be used to test for lung malfunctions in a manner that is slightly different from that used in Fig. 9.13. After a very deep inhalation, a person forces air out as fast as possible. The total volume exhaled is the forced vital capacity (FVC), which can be a bit less than the vital capacity

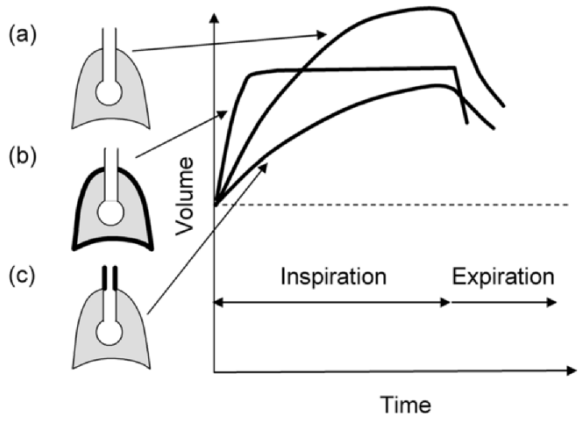

Fig. 9.20. Lung volume during inspiration for (**a**) normal conditions, (**b**) decreased compliance, and (**c**) increased airway resistance. (Based on [443])

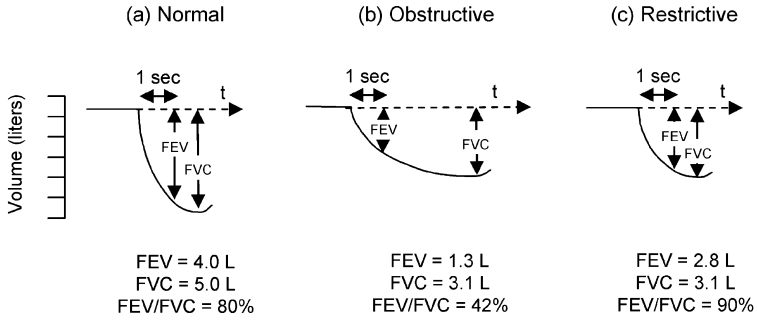

Fig. 9.21. Forced expiration for (**a**) normal, (**b**) obstructive, and (**c**) restrictive patterns. (Based on [444])

measured with slower expiration. Also of note is the volume exhaled in the first 1 s, which is the forced expiratory volume (FEV) (or FEV_1, which denotes specifically that this volume was expired in 1 s), and the ratio FEV/FVC. As seen in Fig. 9.21, for a normal lung FVC = 5.0 L and FEV/FVC = 80%. The example of an *obstructive* pattern has a lower FV, 3.1 L, and smaller FEV/FVC, 42%. The example of a *restrictive* pattern also has a lower FV, 3.1 L, but a high FEV/FVC, 90%. The flow rates for the obstructive pattern are also abnormally low (Problem 9.27).

Figure 9.18 shows cycles that typify the breathing cycles of a normal person and of people with lung disorders. The divisions between the alveoli break down in people with *emphysema*. Consequently, the lungs become less springy and more compliant, and the airway resistance contribution dominates breathing. In pulmonary fibrosis, the compliance is reduced by an increase in fibrous tissue. This condition increases in pulmonary emphysema and in normal aging, due to a change in elastic tissue in the lungs. The volume/pressure locus is also shown for idiopathic respiratory distress syndrome (RDS). It is seen to move to higher pressure due to a lack of alveoli surfactant, as occurs in some premature babies. In such infants the minimal surface tension is only 2×10^{-4} N/m, compared to $\ll 0.5 \times 10^{-4}$ N/m for normal lungs. With less surfactant, the alveolus surface tension decreases and, at the same pressure difference the alveolus is smaller. Figure 9.22 shows that the airways tend

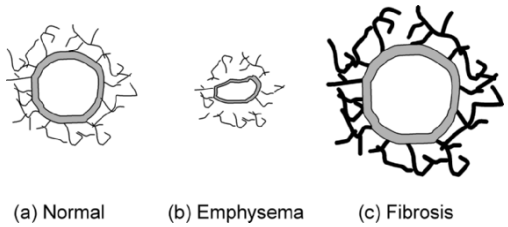

Fig. 9.22. Airways dimensions for different patients. (Based on [444])

to collapse in emphysema because of the loss of radial traction with exterior structures, while in interstitial fibrosis the airways can become large in diameter, due to excessive radial traction, making the airways large in volume relative to the lungs.

9.5.5 Breathing at Higher Elevations

The partial pressure of oxygen is 161 mmHg (21.2 kPa) at sea level (300 K). Hypoxia (which is the lack of oxygen reaching living tissues) occurs below a critical partial pressure of 57 mmHg (7.5 kPa), for a normal, relaxed breathing rate. (Another way of saying this is: Our bodies have been designed so that our rate of breathing air, rate of transferring oxygen to the blood, the capacity of the blood to hold oxygen, the rates of blood flow to tissues, and so on leads to a metabolism that functions well when the partial pressure of oxygen is above this critical value.) *At what elevation above sea level does hypoxia occur?*

The pressure of a fluid column of constant fluid density ρ and constant gravitational constant g is given by (2.48), $P = \rho g h$, where h is the height of the column. In Chap. 8 we considered a column of blood; now let us consider a column of air at a height z and above, where $z = 0$ at sea level. The change in pressure for a change in height is

$$\mathrm{d}P = -\rho g \mathrm{d}z. \tag{9.13}$$

Because we will be considering heights above sea level that are much smaller than the radius of the earth, we have ignored the dependence of the gravitational acceleration g on z. (It is considered in Problem 9.35.)

The ideal gas law (7.2) is $P = nRT$, where n is the density, R is the gas constant, and T is the temperature, or $P = \rho RT/m$

$$P = \frac{RT}{m}\,\rho, \tag{9.14}$$

where $\rho = mn$ is the mass density, with m the molecular mass. We will assume that the atmospheric temperature (300 K) does not vary with height (see Problem 9.40). Dividing (9.13) and (9.14) gives

$$\frac{\mathrm{d}P}{P} = -\frac{mg}{RT}\,\mathrm{d}z. \tag{9.15}$$

Integrating from sea level, $z = 0$ with pressure $P_{\text{sea level}}$, to a height h, with pressure $P(h)$, gives

$$P(h) = P_{\text{sea level}} \exp\left(-\frac{mg}{RT}\,h\right). \tag{9.16}$$

Using the partial pressure of oxygen at sea level and $m = 32$ g/mol for oxygen, this shows that hypoxia occurs at a height of 8.25 km (= 27,100 ft = 5.1 miles).

9.6 Work Needed to Breathe

During inspiration the thorax and abdomen do positive work to expand the lungs [420, 430, 431, 443]. The body does no work during normal expiration, but during forced breathing work is also done to contract the lungs during expiration. Because $V = V_{\rm d} + C_{\rm flow}P$, for the breathing cycle with volume changing between the functional residual capacity volume, $V_{\rm FRC}$, and that plus the tidal volume, $V_{\rm FRC} + V_{\rm t}$, we can write $V(t) = V_{\rm FRC} + C_{\rm flow}P(t)$. We see that $\Delta V(t) = V(t) - V_{\rm FRC} = C_{\rm flow}P(t)$ or $P(t) = \Delta V(t)/C_{\rm flow}$. The work done during inhaling a tidal volume $V_{\rm t}$ is

$$W = \int_0^{V_{\rm t}} P{\rm d}(\Delta V) = \int_0^{V_{\rm t}} \frac{\Delta V}{C_{\rm flow}}{\rm d}(\Delta V) = \frac{V_{\rm t}^2}{2C_{\rm flow}}. \tag{9.17}$$

In Fig. 9.16a, the work done overcoming these elastic (compliance) effects is the area defined by regions I + II, and this is what we have derived here (and will use later). The work is really larger, the area represented by regions I + II + III, because of viscous (resistive) effects. (These viscous/resistive effects also lead to the hysteresis here and in Fig. 9.12, just as in Chap. 4.)

For a breathing rate of f, the rate of doing work for inspiration is

$$\frac{{\rm d}W}{{\rm d}t} = f\frac{V_{\rm t}^2}{2C_{\rm flow}}. \tag{9.18}$$

With a breathing rate of 20/min, tidal volume of 500 $\rm cm^3$, and lung compliance of 0.1 $\rm L/cmH_2O$ = 0.1 $\rm cm^3/(dyne/cm^2)$, this is

$$\frac{{\rm d}W}{{\rm d}t} = \frac{(20/{\rm min})(500\ {\rm cm}^3)^2}{2\times 0.1\ {\rm cm}^3/({\rm dyne/cm}^2)} = 3.6\times 10^3\ {\rm J/day} = 0.86\ {\rm kcal/day}. \tag{9.19}$$

The respiratory muscle efficiency is ϵ = 5–10%, and so the metabolic need is

$$\frac{{\rm d}E}{{\rm d}t} = \frac{{\rm d}W/{\rm d}t}{\epsilon} = \frac{0.86\ {\rm kcal/day}}{0.05} = 17\ {\rm kcal/day} \tag{9.20}$$

assuming 5% efficiency; this calculation is very sensitive to the values chosen for $C_{\rm flow}$ and ϵ. This result is about 1% of the BMR; however this value is really about 2% of the BMR, so maybe ϵ is closer to 3% or other effects need to be considered, such as dissipation due to resistance of the flow and the viscous nature (of the overall viscoelasticity) of the lungs and chest wall. Airflow resistance in the nose seems to be responsible for about half of the work needed to breath.

During normal breathing there is no mechanical work done in expiration. During heavy workouts and strenuous exercise the metabolic needs increase because there is (1) also work done during expiration, (2) a faster breathing rate, and (3) a larger tidal volume. The work done to breathe can use 25%

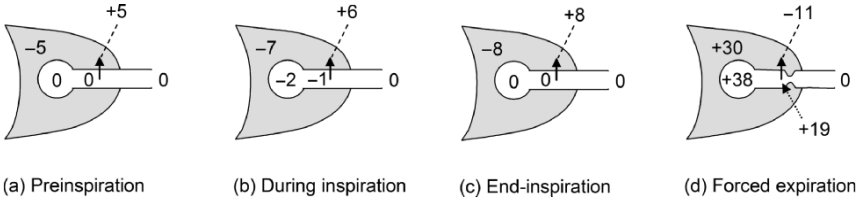

Fig. 9.23. Intrapleural, alveoli, airway, and atmospheric pressures in cmH_2O during inspiration and forced expiration. (Based on [443])

of the total body energy consumption. Such a large increase is suggested by Fig. 8.27. Rapid shallow breathing (as recommended in Lamaze training for childbirth) requires more energy than does normal breathing, to overcome the resistive nature of flow in the air passages. Similarly, slow, deep breathing requires more energy to overcome the elastic nature of the lung and chest. Problem 9.12 compares other relative advantages and disadvantages of these last two modes of breathing.

Another interesting thing can occur during forced expiration. In normal breathing the pressure in the airways always exceeds the intrapleural pressure during inspiration and expiration (as is seen in Fig. 9.23 during normal inspiration). However, during forced expiration the intrapleural and alveoli pressure both increase by the same amount ($38\,cmH_2O$) and so they are both positive relative to the atmosphere ($+30$ and $+38\,cmH_2O$ in the figure). There is now a large pressure drop from the alveoli to the lips and nostrils. At some point along this route the pressure in the airway will be lower than $30\,cmH_2O$ and this will compress the airways (shown where the pressure is $+19\,cmH_2O$ in this figure) and increase the airway resistance.

9.7 Summary

The macroscopic basis of lung function and breathing can be understood by analyzing the volume, pressure, and air flow during breathing, by using models of fluid flow and lung expansion. The physical nature of the individual operating units in the lungs, i.e., the alveoli, can also be understood this way. These models can also be used to understand the physical consequences of a diseased lung. The energy needed to operate the lungs can also be modeled.

Problems

Lungs

9.1. Calculate the effective lung volumes and breathing rates for a man (70 kg), woman (50 kg), and an infant (5 kg) using the allometric relation

parameters in Table 1.13. How do the breathing rates compare with those given above?

9.2. If there are 3×10^8 alveoli in a lung with a functional residual capacity (FRC) of 2.5 L, calculate the average volume and radius of an alveolus.

9.3. Use Table 9.1 to show that the air travels a total distance of 273 mm from the trachea to the alveoli.

9.4. What is the total volume of the lungs described in Table 9.1? Where is most of the volume?

9.5. Is continuity of flow obeyed by the data for the lungs in Table 9.1? Check this using the data for bronchial generations 0, 1, 2, 3, 4, 5, 10, 16, 20, and 23.

9.6. Calculate the Reynolds number for the bronchial generations listed in Problem 9.5. Is the flow laminar or turbulent in the respiratory system?

9.7. Calculate the pressure drop across pulmonary arterial orders 1, 4, 10, 13, 16, and 17, assuming a total blood flow of 5 L/min.

9.8. The CO_2 level in the atmosphere was ∼280 ppm (parts per million) in preindustrial times and is ∼380 ppm now. Express these levels in terms of mmHg. Would this change be expected to affect the exchange of CO_2 in the lungs in any significant manner?

Alveoli and Surface Tension

9.9. Derive (9.8) from (9.7).

9.10. Estimate the force the adult diaphragm would need to exert if there were no lung surfactant.

Breathing

9.11. Use Fig. 9.13b to explain how a spirometer works. How much should the water in the spirometer rise and fall during breathing cycles? (Assume reasonable dimensions for the instrument.)

9.12. During breathing, the pulmonary ventilation, V_p (in L/min) (the rate at which air enters the trachea), equals the respiratory rate, R (in units of per min), times the tidal volume, V_t (in L). Because of the anatomical dead space volume V_d, only $V_\mathrm{t} - V_\mathrm{d}$ enters the alveoli (and is thus of use). Therefore, a more meaningful ventilation rate is the alveolar ventilation $V_\mathrm{a} = R(V_\mathrm{t} - V_\mathrm{d})$:

Table 9.3. Examples of breathing cycles

activity		R (per min)	V_t (L)
(i)	at rest, quiet breathing	12	0.5
(ii)	at rest, with rapid, shallow breathing	24	0.25
(iii)	at rest, with very rapid, very shallow breathing	40	0.15
(iv)	at rest, with slow, deep breathing	6	1.0
(v)	exercising, with rapid, shallow breathing	24	0.5
(vi)	exercising, with slow, deep breathing	12	1.0

(a) Find V_p and V_a for the conditions in Table 9.3, assuming $V_d = 0.15$ L.
(b) Compare the pulmonary ventilation for the four breathing patterns in this table for the person at rest. (Patterns (ii)–(iv) require more metabolic power than does (i), because of increased work due to resistance to flow and resistance in the tissues for (ii) and (iii), and increased work due to compliance (elastic) forces of the lung and chest in (iv)) Which of the four are clearly inadvisable because of poor alveolar ventilation?
(c) During exercise, both the respiratory rate and tidal volume increase. Based on the results in part (a) for (i)–(iv) and for (v)–(vi), do you gain more by breathing faster or deeper for a given pulmonary ventilation?

9.13. What are the maximum and average air flows for each breathing cycle in Problem 9.12, assuming the inhalation and exhalation periods are the same?

9.14. (a) When you take in a deep breath of say 1 L, how much does your mass (in kg) and weight (in N and lb) increase?
(b) Does your average density increase, decrease, or stay the same? If there is a change, estimate it.

9.15. (a) What does Fig. 8.27 say about the amount of oxygen that can be consumed per amount of cardiac output?
(b) What does it say about how much oxygen is needed to do work? Is this consistent with what is presented in the text?
(c) How is work output defined in this figure?

9.16. Estimate the resistance of the trachea using Poiseuille's Law, assuming it has a radius of 9 mm and a length of 110 mm. How does this compare to the total resistance?

9.17. Estimate the resistance of the vocal tract using Poiseuille's Law, assuming it can be modeled as three tubes in series with respective lengths 6, 3, and 6 cm and cross-sectional areas 5, 1, and 5 cm^2. (Also sketch this model.)

9.18. Estimate the resistance of the nasal passage using Poiseuille's Law, assuming it has a radius of 4 mm and a length of 3 cm. How does this compare to the total resistance and is it a limiting factor in the resistance to flow?

9.19. If you model the breathing airway as a series of sequential passages, the nasal or mouth passage, the pharynx, larynx, and then trachea, each with a resistance to air flow, what is the total resistance to air flow in terms of these individual resistances?

9.20. In both inspiration and expiration, a pressure difference of $0.4\,\mathrm{cmH_2O}$ causes a flow of $0.15\,\mathrm{L/s}$ in the nose. Determine the flow resistance in it.

9.21. Consider the lung bifurcation generations 1–19 in Table 9.1:
(a) In which generation is the flow resistance largest? What is its value?
(b) Do your results agree with those in Fig. 9.14a?
(c) In which generation is the pressure drop greatest, and generally in what range of bifurcations is most of the pressure drop?

9.22. The total airway resistance is the sum of those in each generation. Do the resistances in Fig. 9.14a add to give you a total resistance consistent with that in Fig. 9.14b?

9.23. Calculate the resistance for generation 4 using Poiseuille's Law and compare it to the values given in the chapter.

9.24. (a) Use Fig. 9.15 to determine the compliance of the lungs ($C_{\mathrm{flow,lung}}$) and chest walls ($C_{\mathrm{flow,chest\ wall}}$) at 0, 20, 40, 60, and 80% of vital capacity.
(b) Determine the compliance of the combined lung/chest wall system ($C_{\mathrm{flow,lung/chest\ wall}}$) at these volumes, and compare these values with those from part (a) by using $1/C_{\mathrm{flow,lung/chest\ wall}} = 1/C_{\mathrm{flow,lung}} + 1/C_{\mathrm{flow,lung/chest\ wall}}$.

9.25. (a) Use Fig. 9.18 to determine the compliance of the lungs for each condition (within the lowest $5\,\mathrm{cmH_2O}$ pressure range shown for each).
(b) How does the compliance vary for each over the pressure range shown?

9.26. Compare the specific lung compliances of a 65 kg man and 20 g mouse, with respective compliances of $0.2\,\mathrm{L/cm\text{-}H_2O}$ and $0.0001\,\mathrm{L/cm\text{-}H_2O}$.

9.27. Show that the forced expiratory flow (FEF) rates for the normal, obstructive, and restrictive flows in Fig. 9.21 are 3.5, 1.4, and 3.7 L/s, respectively. Do this by determining the slopes of the three curves in this figure. (Use a straight-line fit between points that have decreased by 25% and 75% on the way to the FVC.)

9.28. Determine the air flow resistance from the flow rate and alveoli pressure in Fig. 9.17.

9.29. Consider only the compliance in the work of breathing and assume that the compliance C_{flow} for normal lungs is $0.1\,\mathrm{cm^5/dyne}$:
(a) In fibrosis of the lungs the compliance of the lungs decreases. For a given tidal volume, how does the rate of work of breathing change if the compliance decreases by x%?
(b) Compare the rate of work done in breathing (J/day) and the associated rate of metabolism (kcal/day) (if the muscles associated with breathing are 5% efficient) for cases (i) and (iv) in Problem 9.12 for normal lungs.

9.30. (advanced problem) Write down the equation of motion for the mechanical model in Figure 9.16b and solve it for inspiration.

9.31. (advanced problem) Show that the solution in Problem 9.30 qualitatively agrees with the trends seen in Fig. 9.20: with decreased compliance, the time constant decreases and the volume breathed during a cycle decreases, while with increased airway resistance, the time constant increases and the volume breathed during a cycle decreases.

9.32. Estimate the rate of energy consumed by the lungs during exercise with a breathing rate of 40/min and tidal volume of 1,000 cm^3.

9.33. Use a blood circulation rate of 5 L/min and the known change in the partial pressures of O_2 and CO_2 in the systemic capillaries to find the number of liters of O_2 consumed and CO_2 exhaled each day. How do your results change if you instead use the change in the partial pressures of O_2 and CO_2 in the pulmonary capillaries? Explain why.

9.34. If your chest wall and parietal pleura of a lung are punctured, the intrapleural pressure will increase to atmospheric pressure and that lung will collapse. Explain why. Also draw a diagram explaining this.

Breathing at High Elevation

9.35. (a) Show that the gravitational acceleration constant g varies with height z above sea level as $g(z) = g\,(R_{\mathrm{Earth}}/(R_{\mathrm{Earth}} + z))^2$, where the radius of the earth is $R_{\mathrm{Earth}} = 6,378\,\mathrm{km}$.
(b) Show that this variation does not affect the analysis of oxygen deprivation at high elevations, described in the text.

9.36. What is the atmospheric pressure in the "mile-high" city of Denver? What is the partial pressure of oxygen there?

9.37. Commercial jets typically cruise at an altitude of ∼10,700 m (∼35,000 ft). What are the total pressure and partial pressure of oxygen at that height? Why are jets pressurized? Why are oxygen masks made available just in case the cabin is depressurized?

9.38. Why do some athletes train at high elevations?

9.39. Apply (9.16) to the variation of the partial pressure of nitrogen, using $m = 28\,\mathrm{g/mol}$. Let us say here that the ratio of oxygen to nitrogen is $20.9\%/78.1\% = 0.268$ at sea level. What is this ratio at the critical height for hypoxia?

9.40. The temperature of the troposphere (the atmosphere up to roughly 11 km) decreases with height, by a bit less than 1 K per 100 m of elevation. In

Table 9.4. Total and partial pressures at different elevations. (Using data from [445])

altitude	sea level	at 2,500 m
atmospheric pressure (total)	760	560
atmospheric pressure (O_2)	159	117
in alveoli (O_2)	105	77
in arterial blood (O_2)	100	72
in venous blood (O_2)	40	40

the standard atmosphere $T(z) = T_{\text{sea level}} + \alpha z$, with $T_{\text{sea level}} = 288.19\,\text{K}$ and $\alpha = -0.00649\,\text{K/km}$. (For a dry atmosphere, $\alpha = -0.0098\,\text{K/km}$.):
(a) Use this temperature variation in (9.15) to show that

$$P(h) = P_{\text{sea level}}\,(T_{\text{sea level}}/(T_{\text{sea level}} + \alpha h))^{gm/R\alpha}\,. \tag{9.21}$$

(b) Show that hypoxia occurs at a lower elevation, 7.21 km.

9.41. Table 9.4 compares the partial pressure of oxygen (in mmHg) in the air and in the body at sea level and at an elevation of 2,500 m:
(a) Justify the values given for total pressure and O_2 partial pressure at 2,500 m.
(b) Justify the O_2 partial pressure in the alveoli at 2,500 m by using the pressure at sea level.
(c) At sea level your blood flows at a rate of 5 L/min. How fast would it have to flow at an elevation of 2,500 m to provide the same flow of oxygen to the tissues? (Assume no change in the red blood cell and hemoglobin concentrations in the blood. These increase as part of adapting to higher elevations.)
(d) How much faster would you have to breathe at this elevation (in liters of air per min) to maintain the same rate of oxygen delivery? How could this translate into changes in the breathing rate and tidal volume?