5.9 Vascular Function: Hemodynamics

THE VASCULAR SYSTEM DISTRIBUTES CARDIAC OUTPUT TO THE TISSUES

As described in Chapter 5.1, the vascular system serves four distinct functions:

1. Transforms the pulsatile flow from the heart to more continuous flow (**arteries**).
2. Distributes the cardiac output to the tissues (**arterioles**).
3. Exchanges materials with the tissue (**capillaries**).
4. Provides a volume reservoir (**veins**).

THE CIRCULATORY SYSTEM USES FOUR MAJOR PHYSICAL PRINCIPLES

The physical principles of flow through the circulatory system are incorporated into four statements:

[5.9.1]

$$(1) \quad Q_V = \frac{\Delta P}{R}$$

$$(2) \quad \Delta P = \frac{\Delta V}{C}$$

$$(3) \quad <V> = \frac{Q_V}{A}$$

$$(4) \quad R = \frac{8\eta I}{\pi a^4}$$

The first principle listed above describes steady-state flow in terms of an Ohm's law analogue. Here Q_V is the volume flow in L min^{-1}, ΔP is the difference in pressure that drives flow, and R is the resistance. We will discuss further the validity of this law and the meaning of ΔP.

The second principle is a statement of the elasticity of the vessels, where ΔP is the pressure increment produced when a volume ΔV is inserted into the vessel. The pressure increment is related to the volume increment through the **compliance** of the vessel, C.

The third principle describes the relation between average velocity in the vessels, $<v>$, the flow, and the cross-sectional area, A. As we will see, in laminar flow the velocity across the vessel is not uniform.

The fourth principle is Poiseuille's law, which describes steady-state flow in tubes. We have seen this equation before (Chapter 1.2). Here we write the resistance of a long tube under laminar flow conditions where R is the resistance, η is the viscosity of the flowing fluid, l is the length of the tube, and a is its radius.

FLOW IS DRIVEN BY A PRESSURE DIFFERENCE

It is not just the static pressure that drives flow. Flowing fluids have a kinetic energy that can be converted to pressure, and vice versa, and they also possess gravitational potential energy. The total mechanical energy was given in Chapter 5.8 as

$$[5.9.2] \quad E = E_P + E_K + E_G = PV + 1/2\ \rho V v^2 + \rho g h V$$

where E, the total mechanical energy, is the sum of the pressure−volume energy, the kinetic energy, and the gravitational energy. Here ρ is the density of the blood, v is its velocity, g is the acceleration due to gravity, and h is the height of the fluid above a reference height.
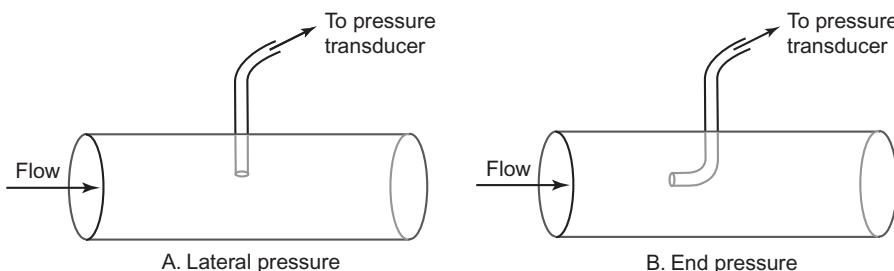
FIGURE 5.9.1 "Lateral pressure", A, and "end pressure", B, measured in a tube filled with a flowing fluid. Blood that flows into the end of the catheter loses some or all of its kinetic energy, which is converted to pressure. This measures the end pressure, which is slightly higher than the lateral pressure.

Dividing by the volume, we derive the total equivalent pressure, P', as

[5.9.3] $$P' = P + 1/2 \ \rho v^2 + \rho gh$$

This equivalent pressure drives steady-state flow through the circulatory system. This is the basis of Bernoulli's law and also allows us to determine the difference between **lateral pressure** and **end pressure**.

In a static fluid the pressure is the same in all directions. In a moving fluid the kinetic energy contributes to the pressure only in the direction of its velocity. Thus, a catheter inserted into a blood vessel will record slightly different pressures if the tip of the catheter is pointing perpendicular to the fluid flow, into the fluid flow, or away from the fluid flow, as illustrated in Figure 5.9.1. Similarly, the pressure measured in a tube when the fluid is flowing depends on the velocity of the flow.

EXAMPLE 5.9.1 Calculate the difference in lateral and end pressure in the aorta

Assume that the thoracic aorta in man has a radius of 1.1 cm. Assume a steady flow of 5 L min^{-1}. What is the difference between lateral and end pressure?

We estimate the velocity of blood from $<v> = Q_v/A$ where $<v>$ is the average velocity, Q_v is the volume flow, and A is the cross-sectional area. The area is $\pi(d/2)^2 = 3.8$ cm^2 and the flow is

$$<v> = [5000 \text{ cm}^3/60 \text{ s}]/3.8 \text{ cm}^2 = 22 \text{ cm s}^{-1}$$

The pressure corresponding to this velocity is $1/2 \ \rho v^2$. The density of blood is about 1.05 g cm^{-3} (Chapter 5.1). This is

$1/2 \times 1.05$ g cm$^{-3} \times [22$ cm s$^{-1}]^2 = 254$ g cm s^{-2} cm^{-2} or 254 dyn cm^{-2}. We convert to Pa by 254 dyn $\times 10^{-5}$ N dyn^{-1} cm$^{-2} \times 10^4$ cm^2 m$^{-2} = $ **25.4 Pa**. Since 1 mmHg = 133.3 Pa (Chapter 1.2), this is 25.4/133.3 = **0.2 mmHg**.

This calculation depends on the velocity of the blood. In the aorta, the flow is pulsatile and not constant. If flow were 100 cm s^{-1}, the end pressure would be 4 mmHg higher than lateral pressure.

As an example, consider that a blood vessel has a partial stenosis—a narrowing of its caliber—for part of its length. The steady-state flow through all sections of the tube must be the same because at steady state there is no net storage or loss of volume. Thus Q_v is the same through every cross-section of the vessel. According to principle (3), this means that the average velocity in the narrow portions must increase inversely with the cross-sectional area. The fluid accelerates, converting pressure energy into kinetic energy. On the other side of the stenosis the fluid slows again. The lateral pressure varies inversely with the fluid velocity, which is Bernoulli's law. The lateral pressure, equivalent pressure due to kinetic energy, and total pressure for the vessel with a partial stenosis is shown in Figure 5.9.2.

COMPLIANCE DESCRIBES THE RELATION BETWEEN PRESSURE AND VOLUME IN THE VESSELS

The elasticity of the vessels is described empirically by the compliance, as described in Eqn [5.9.1] and reproduced here:

[5.9.4] $$\Delta P = \frac{\Delta V}{C}$$

We may rearrange this to define compliance as

[5.9.5] $$C = \frac{dV}{dP}$$

This definition fits our subjective sense of what we mean by compliance. A compliant system is one that stretches. According to Eqn [5.9.5], a high compliance means that you can fit in more volume per unit change in pressure—it stretches easily. Conversely, a low compliance means that adding small volumes markedly increases the pressure. The compliance depends on the makeup of the vessel and varies according to location within the circulatory system. The large arteries have thick walls and are not as compliant as the more distensible veins. In particular, in humans

[5.9.6] $$\frac{C_V}{C_A} \approx 19$$

where C_V is the compliance of the veins and C_A is the compliance of the arteries. The compliance makes the ejected blood functionally compressible even though the fluid itself is not compressible—the expansion of

the vessels in response to the ejected blood achieves the same result as if the blood were compressible. After the heart stops ejecting blood, the elastic arteries recoil and continue to push the blood forward. The fluid movement is driven by the blood pressure, whose time dependence results from the size and time course of the ejected volume of blood by the heart, the viscosity of the blood, and the size and compliance of the vascular tree leading away from the heart. The pressure pulse that results propagates down the arterial tree at some $5-8$ m s^{-1}. The pressure pulse is transmitted faster than fluid flow, which is only about 0.2 m s^{-1}.

THE HEART'S EJECTION OF BLOOD INTO THE ARTERIAL TREE CAUSES THE ARTERIAL PRESSURE PULSE

The pressure pulse obtained from a catheter in the subclavian artery is shown in Figure 5.9.3. The lowest pressure, the **diastolic pressure**, about **80 mmHg** in healthy adults, occurs immediately prior to the opening of the aortic valve. The heart ejects blood faster than it can run off through the arteries, so part of the ejected volume ($67-80\%$) distends the elastic arteries and raises the pressure toward its maximum value, the **systolic pressure**, typically about **120 mmHg**. When ejection slows, runoff of blood catches up and exceeds ejection. The volume in the elastic arteries falls and pressure falls with it. Intraventricular pressure falls precipitously, causing the aortic valve to snap shut. This causes a brief interruption in the fall of pressure, called the **incisura** or **dicrotic notch**.

THE PULSE PRESSURE DEPENDS ON THE STROKE VOLUME AND COMPLIANCE OF THE ARTERIES

The rise of pressure from diastolic to systolic levels is called the **pulse pressure**, defined as

[5.9.7] $$\Delta P_{\text{pulse}} = P_{\text{systolic}} - P_{\text{diastolic}}$$
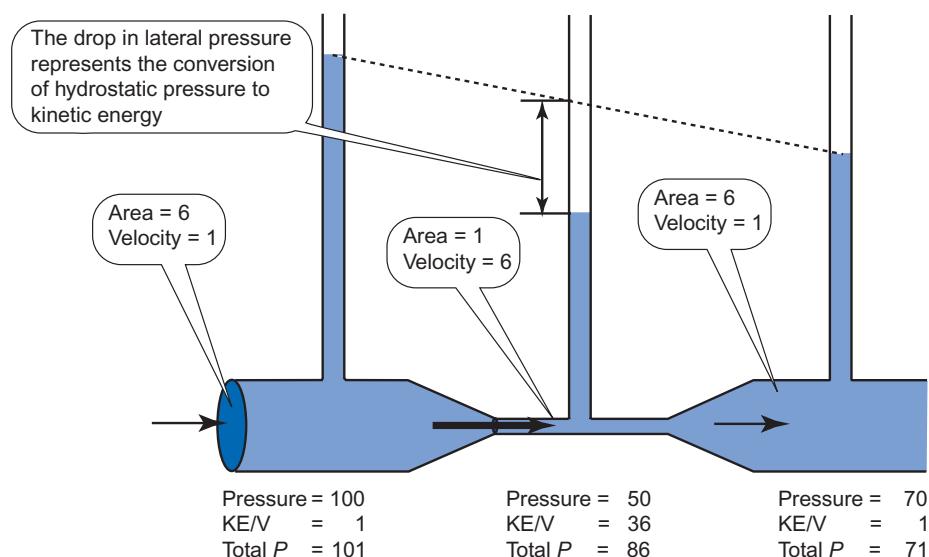
FIGURE 5.9.2 The effect of tube caliber on steady-state pressures. A single tube is shown with a narrow portion in its middle. During steady-state flow, the fluid in the narrow portion flows more quickly, and in this section the lateral pressure is reduced because the pressure−volume energy is converted to kinetic energy. When the tube widens again, the kinetic energy is converted back to pressure−volume energy. Fluid flows from the narrow part to the wide part in opposition to the pressure gradient but in accord with the total mechanical energy of the fluid. (Source: *Adapted from J.R. Levick, Cardiovascular Physiology*, Arnold, New York, NY, 2003.)
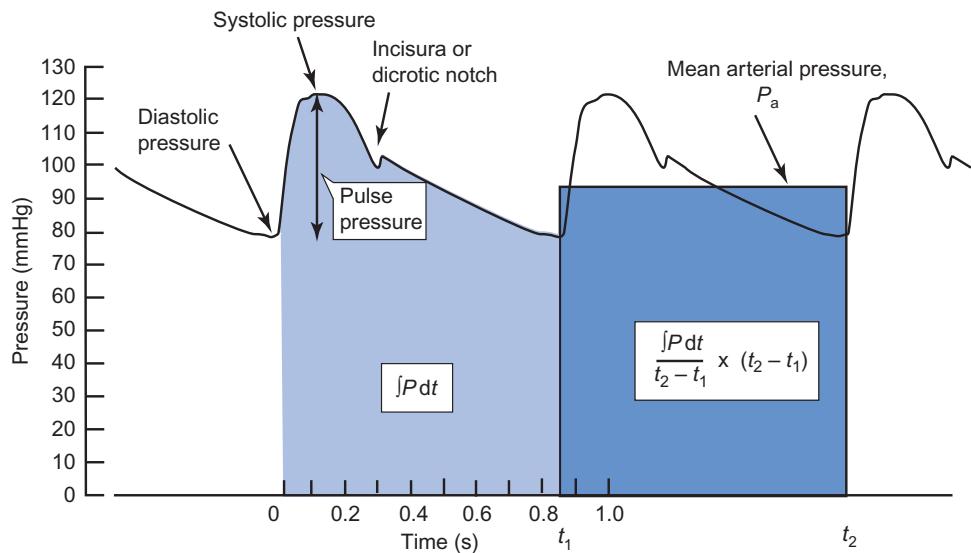
FIGURE 5.9.3 Arterial pressure pulse measured in the subclavian artery. The pressure rises upon ejection of blood during systole, peaking at the systolic pressure. When ejection slows, blood flows down the artery, volume within the elastic artery decreases, and pressure falls. The incisura or dicrotic notch occurs when the aortic valve closes. Pressure then falls to the diastolic pressure. The mean arterial pressure is the pressure that, when multiplied by the period, equals the area under the arterial pressure trace.

The compliance of the arteries is nonlinear, being stiffer at higher volumes. Thus, the pulse pressure for a given stroke volume is greater at higher pressures (see Figure 5.9.4). When the compliance of the arteries is decreased, as occurs in general in older individuals due to **arteriosclerosis** (hardening of the arteries) and **atherosclerosis** (build up of fatty deposits in the arterial walls), the pulse pressure also increases (see Figure 5.9.5).
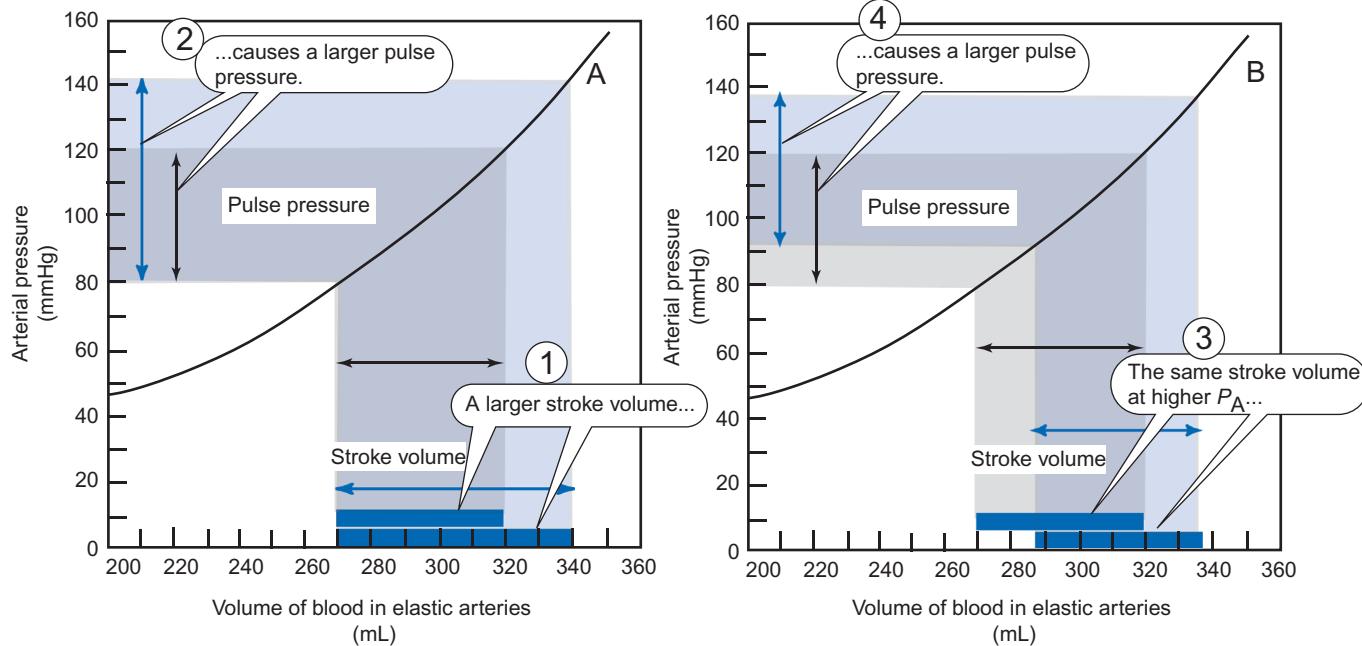

FIGURE 5.9.4 Relation between stroke volume and pulse pressure. Part A shows that increasing the stroke volume increases the pulse pressure. Because the compliance is nonlinear, the increase in pulse pressure is even greater than the increase in stroke volume. Part B shows that the same stroke volume at a higher mean blood pressure results in a larger pulse pressure. Note that the elevation of the pulse pressure is due to a greater increase in the systolic pressure than in the diastolic pressure.
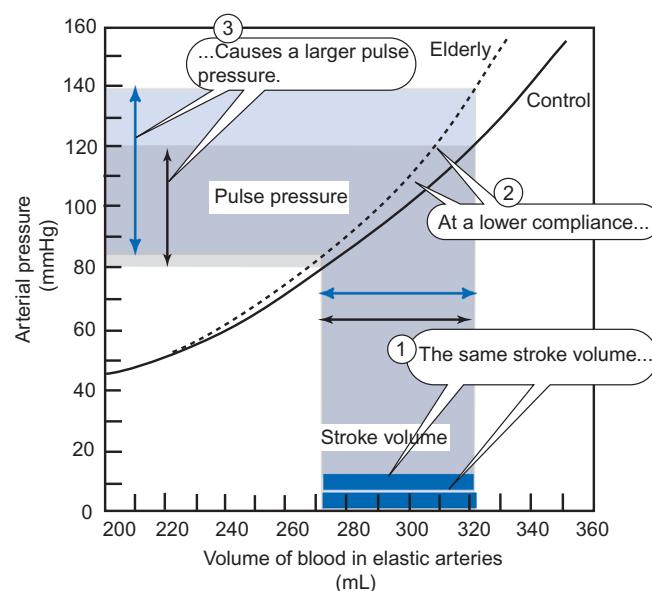

FIGURE 5.9.5 Effect of decreased compliance on the pulse pressure. Elderly persons often exhibit decreased compliance of their arteries, which manifests itself on these plots as an increased slope because $C = dV/dP$ and the plot is P against V. The decreased compliance causes an increase in pulse pressure, with a greater effect on increased systolic pressure.

DIASTOLIC PRESSURE PLUS ONE-THIRD PULSE PRESSURE ESTIMATES THE MEAN ARTERIAL PRESSURE

The arterial pressure has a complicated time course, as shown in Figure 5.9.3. The mean arterial pressure is the constant pressure that would produce the same flow as the arterial pressure. It is defined mathematically as

$$[5.9.8] \qquad P_A = \frac{\int_{t_1}^{t_2} P \, dt}{t_2 - t_1}$$

This means that the mean arterial pressure multiplied by the time of the cycle gives the integral of the pressure−time trace. Thus, the area under the pressure−time trace is equal to the area of a box one cycle long with a constant pressure of P_A. The relationship between the pressure trace and P_A is shown in Figure 5.9.3.

The mean arterial pressure can be approximated by a rule-of-thumb calculation:

$$[5.9.9] \qquad \begin{aligned} P_A &= P_{\text{diastolic}} + \frac{\Delta P_{\text{pulse}}}{3} \\[6pt] &= P_{\text{diastolic}} + \frac{(P_{\text{pulse}} - P_{\text{diastolic}})}{3} \end{aligned}$$

Thus, for a person with a brachial artery systolic pressure of 120 mmHg and diastolic pressure of 80 mmHg, the mean arterial pressure is approximately 93 mmHg.

PRESSURE AND FLOW WAVES PROPAGATE DOWN THE ARTERIAL TREE

The ejection of blood by the heart generates pressure waves in the aorta and pulmonary artery that propagate down the arterial tree at some $5-8 \text{ m s}^{-1}$, whereas the flow is only about 0.2 m s^{-1}. This should present no conceptual difficulties because pressure waves in air (sound) propagate in dry air at about 331 m s^{-1} in the absence of any flow whatever! Blood that is ejected by the heart is not compressible, and therefore it has to make room for itself in the aorta. It does this partly by distending the aorta (and thereby increasing the pressure there) and partly by pushing the blood already in the aorta into the next section of artery. This displaced blood moves forward and distends the artery in the next section of the circulation, increasing the pressure there, and also pushing some of the blood forward. This sequence of events is repeated for each segment of the arterial tree causing the pressure wave to propagate down the arterial tree. Because the vessels change size and compliance as one progresses from aorta forward, the shape of the pressure wave also changes. Figure 5.9.6 shows representative pressure pulses obtained by a catheter as it was gradually withdrawn from the subclavian artery to the radial artery. Several changes occur in the waveform as it travels peripherally:

- The dicrotic notch is damped out and eventually disappears.
- The mean pressure falls by about 2 mmHg from the subclavian artery to the radial artery.
- The systolic wave steepens and increases in magnitude.
- A diastolic wave appears due to reflection of the pressure pulse from the periphery.

CLINICIANS USE A *SPHYGMOMANOMETER* TO MEASURE BLOOD PRESSURE

In 1773, Stephen Hales made the first measurement of blood pressure when he connected a 3-m glass tube to the carotid artery of a horse via a goose trachea, and he noted the height to which the blood rose in the tube. Noninterventional clinical measurements were made possible by the invention of the mercury manometer by Poiseuille, which has evolved into the **sphygmomanometer**.

In this procedure, an inflatable cuff is placed around the upper arm. The lower end of the cuff should be at heart level to prevent gravitational pressures being added or subtracted from the measured pressure. The cuff is inflated to stop all flow through the brachial artery. The clinician places a stethoscope over the antecubital space (the inner surface of the elbow joint) over the brachial artery. Slow release of the pressure cuff will lessen the occlusion until some blood squirts through
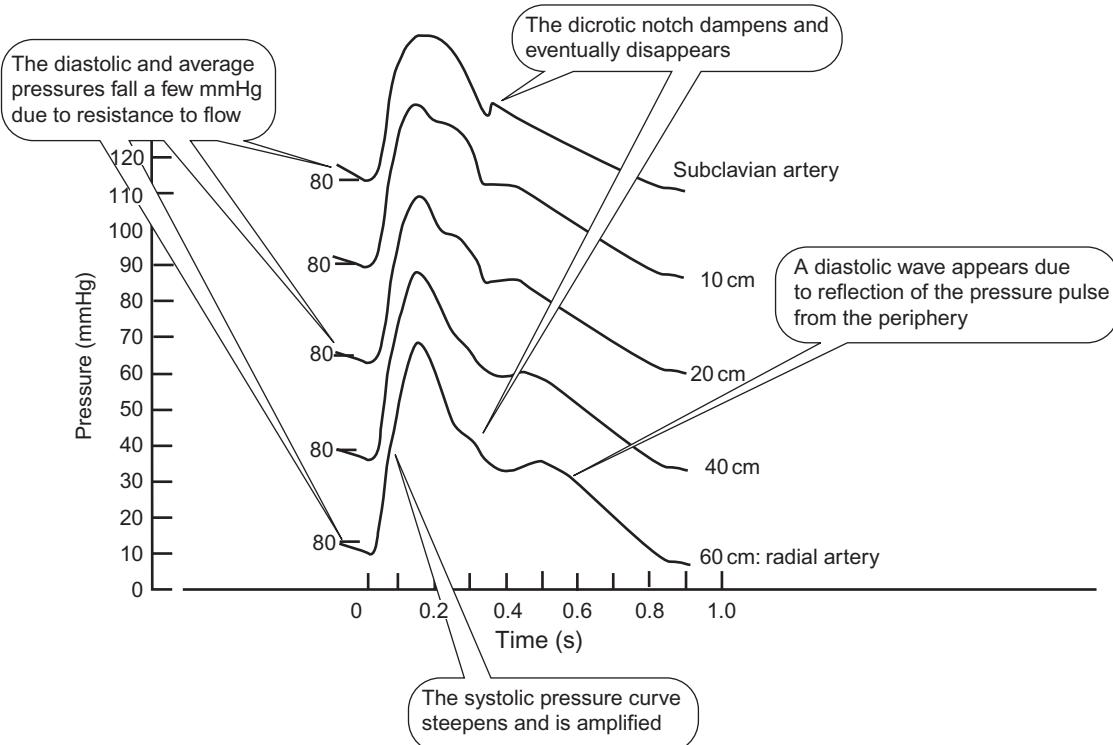

FIGURE 5.9.6 The pressure pulse obtained sequentially by withdrawal of a catheter from the subclavian artery to the radial artery. The distances by each trace refer to the distance withdrawn from its central location in the subclavian artery. Each trace is offset vertically and realigned so that the times of initiation of the pressure pulses coincide.

when the cuff pressure is just below systolic pressure, creating the **first Korotkoff sound**. The cuff pressure at the first Korotkoff sound is the **systolic pressure**. As the cuff pressure is further lowered, more blood spurts through the partially occluded brachial artery and the Korotkoff sounds become louder. When the cuff pressure is further lowered, near the diastolic pressure, the artery remains open almost all of the time. The Korotkoff sounds suddenly muffle and then, as cuff pressure decreases further, the sounds disappear. The pressure at which the sound first disappears is the diastolic pressure. Figure 5.9.7 illustrates this method of obtaining blood pressure.

BLOOD VESSELS BRANCH EXTENSIVELY, REDUCING THEIR DIAMETER BUT INCREASING THE OVERALL AREA

A single ascending blood vessel, the aorta, leaves the left ventricle, and it branches immediately to form the coronary arteries at the base of the aorta. The aorta branches to form the major arteries, which branch again to form small arteries and then smaller arteries. At each branching, the subsequent vessels become smaller in diameter but larger in number. The number of branches increases
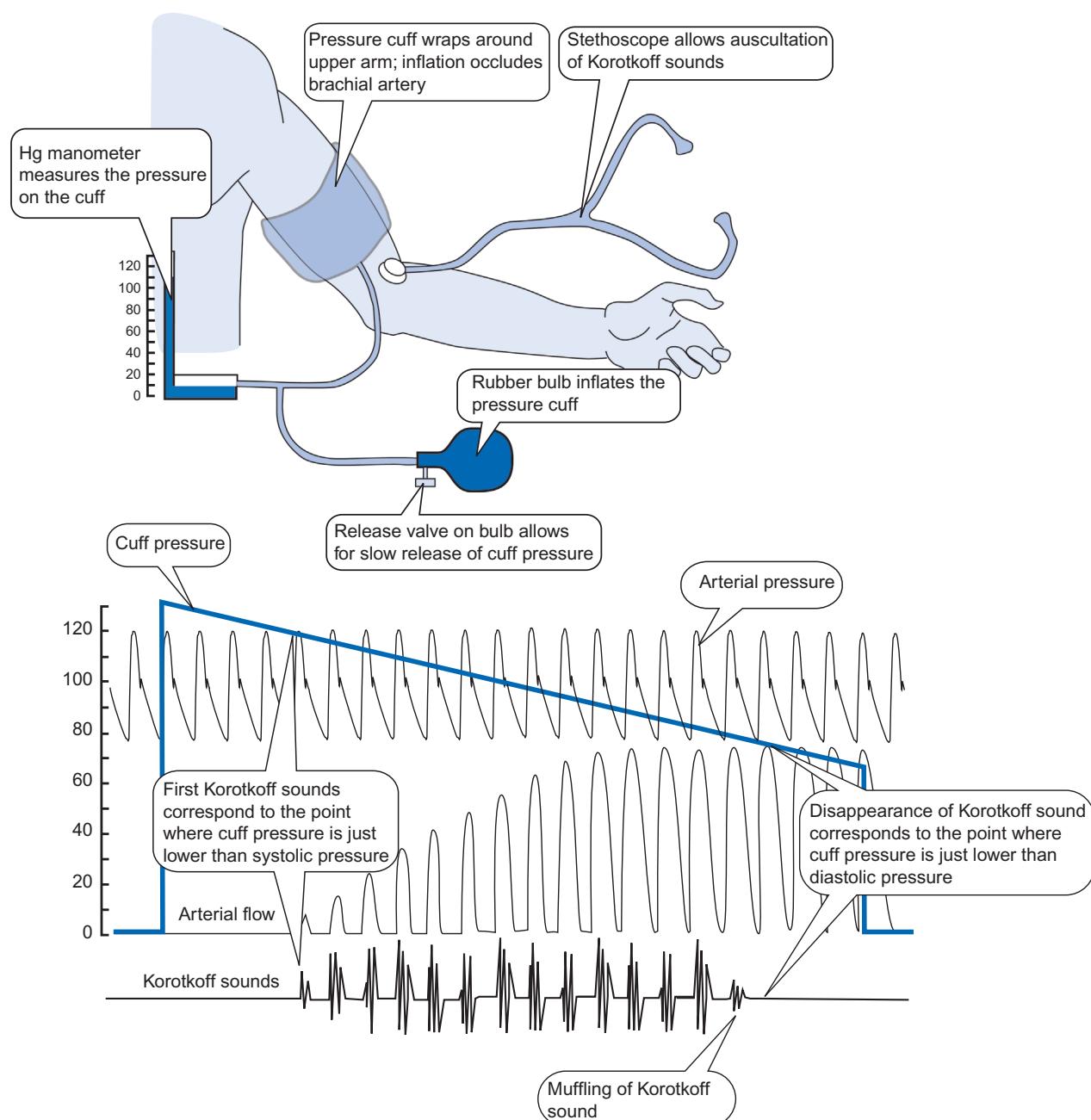

FIGURE 5.9.7 Estimation of arterial blood pressure using a sphygmomanometer. The clinician inflates the pressure cuff to a pressure higher than the anticipated systolic pressure and slowly releases the pressure while watching the manometer and listening for Korotkoff sounds. (See text for details).

faster than the decrease in area, so that the aggregate area increases with each branching. Because the flow through each level of the circulatory system must be the same, the velocity of flow becomes slower with each branching because $<v> = Q_v/A$, where A is the total area. Arteries with radius $a < 50\ \mu m$ are called **arterioles**. These branch several times, forming smaller arterioles until at last the vessels form the **capillaries**, small structures with radii between 1 and 3 μm. Exchange of nutrients in the tissues occurs at the small arterioles, capillaries, and small venules (see Chapter 5.10). The blood from the capillaries then collects into venules, small veins, and the larger veins, in which the cross-sectional area of the vessels progressively increases and their number decreases, the opposite of what occurs in the arteries. The key parameters that vary with branching are given in Table 5.9.1.

THE MAJOR PRESSURE DROP IN THE ARTERIAL CIRCULATION OCCURS IN THE ARTERIOLES

The pressure drops in the circulatory system indicate hydrodynamic resistances, just like voltage drops in a network of electrical resistances indicate the magnitude of the electrical resistances. There is little change in the average pressure going from the subclavian artery to the radial artery (Figure 5.9.6) indicating little resistance to flow along that length of artery. Figure 5.9.8 shows how the pressure drops along the systemic and pulmonary circulations, showing that the major pressure drop occurs in the arterioles.

POISEUILLE'S LAW ONLY APPROXIMATELY DESCRIBES FLOW IN THE VASCULATURE

In 1817, Jean Leonard Marie Poiseuille (1799−1869) introduced the mercury manometer to measure blood pressure. His early investigation using this device led him to believe that the blood pressure remained constant as blood traveled through the large arteries because the actual small decrease in the average pressure was not resolved with his method. He knew that the blood pressure within the veins was low and that therefore there must be a drop in pressure in the small vessels <2 mm in diameter. This led him to study flow in very small tubes. He discovered that the relation between flow, Q_v, and the drop in pressure, ΔP, along a tube of length l and inner diameter D was

$$[5.9.10] \qquad Q_v = \frac{KD^4 \Delta P}{l}$$

where K is an empirical coefficient that Poiseuille found was independent of tube caliber, length or flow, but fell with decreasing temperature. Wiedemann in 1856 and Hagenbach in 1860 independently derived a theoretical solution to this problem:

$$[5.9.11] \qquad Q_v = \frac{\pi a^4 \Delta P}{8 \eta l}$$

where a is the radius and η is the viscosity. Thus, Poiseuille's experimental result identifies his coefficient as $K = \pi/128\ \eta$. The derivation of Poiseuille's law is presented in Chapter 1.2.A1. The theoretical derivation of Poiseuille's law requires several assumptions:

- The fluid is a Newtonian fluid (viscosity is independent of the shear rate, dv/dr).
- Flow is laminar.
- There is no "slippage" at the vascular wall (zero velocity of the fluid in contact with the wall).
- Flow is steady.
- The tube is cylindrical with circular cross section and parallel walls.
- The walls of the tube are rigid.

TABLE 5.9.1 Key Parameters Dealing with the Branching of the Systemic Circulatory System[a]

Vessel	Number	Radius (cm)	Area per Unit: $A_i = \pi a^2$ (cm²)	Aggregate Cross-sectional Area $A_{total} = n\ \pi a^2$ (cm²)	$<v> = Q_v/A_{total}$ (cm s⁻¹)	Single Unit Flow $q_v = Q_v/n$ (cm³ s⁻¹)
Aorta	1	1.1	4	4	21	83
Arteries[b]	8000	5×10^{-2}	8×10^{-3}	64	1.3	1×10^{-2}
Arterioles[b]	1×10^7	25×10^{-4}	2×10^{-5}	200	0.4	8.3×10^{-6}
Capillaries[b]	1×10^{10}	4×10^{-4}	5.0×10^{-7}	5000	1.7×10^{-2}	8.3×10^{-9}
Venules[b]	4×10^7	5×10^{-3}	7.9×10^{-5}	3160	2.6×10^{-2}	2.1×10^{-6}
Veins[b]	8000	0.09	1×10^{-1}	800	0.1	1×10^{-2}
Vena Cava	2	1.4	6	12	6.9	41

[a]The area per unit is the cross-sectional area calculated from the radius as $A_i = \pi a^2$ where a is the radius. The aggregate cross-sectional area was calculated by multiplying the cross-sectional area per vessel times the number of vessels of that generation or order: $A_{total} = n\ \pi a^2$ where n is the number of vessels at that generation. The average velocity of blood flow within the vessel was calculated as $<v> = Q_v/A_{total}$ where A_{total} is the aggregate cross-sectional area. The single unit flow is the average blood flow through a single vessel of the size described.

[b]For each category of arteries, arterioles, capillaries, venules, and veins there are several orders of vessels. The calculations shown are for a representative generation of the approximate size indicated.

The assumption of Newtonian behavior is reasonably good. This is surprising, given that blood is not even a fluid but a suspension of cells. Deviations from Newtonian behavior occur in vessels with radius less than 0.025 cm.

"Laminar" flow refers to streamlined flow. Its counterpart is turbulent flow. The point at which turbulence begins is estimated from the **Reynolds number**, a dimensionless constant that is the ratio of the inertial forces to the viscous forces:

[5.9.12]
$$Re = \frac{2a <V> \rho}{\eta}$$

where Re is the Reynolds number, a is the radius of the tube, $<V>$ is the average velocity, ρ is the density, and η is the viscosity. Flow becomes turbulent at $Re \sim 2000$, but in the circulatory system Re is significantly lower than 2000. Turbulent flow sometimes occurs in the aortic root and around irregularities in the arterial surface such as atheromatous plaques. Turbulence produces a **bruit** ("noise") that is audible through a stethoscope.

The assumption of no slippage forms part of the boundary conditions that allows us to solve the differential equations leading to Poiseuille's law. Hydrodynamic experiments in general confirm it.

The assumption of steady flow is not met in the circulation because flow and pressure are pulsatile until the blood meets very small vessels. The derivation of Poiseuille's law shows that the velocity profile within steady-state flow is parabolic:

[5.9.13]
$$V = V_{max} \left[1 - \frac{r_2}{a_2} \right]$$

where r is the distance from the center of the vessel and a is its radius. Thus the velocity is maximal in the center and zero when $r = a$, and it varies parabolically (see Appendix 1.2.A1 for the derivation of this velocity profile). This velocity profile develops as fluid flows down a tube. At the entrance, the velocity profile is blunt and slowly develops as the viscous drag on the vessel wall and subsequent layers of the fluid gradually communicates through the entire moving fluid. This is shown schematically in Figure 5.9.9. The distance necessary to establish the parabolic profile is called the **entrance length**. This idea pertains only to vessels where $a > 50\ \mu m$. Thus Poiseuille's law is valid only for tubes that are long compared to the entrance length.

Arteries typically have circular cross-sections, while veins typically have elliptical cross-sections. Further, arteries typically taper toward their periphery so that their walls are not parallel. The arteries also branch extensively and typically branches occur every 3−4 cm. These branches alter the geometry of the tubes and introduce complexities into the analysis of the relation between flow and pressure.

Blood vessels are distensible and therefore their diameter depends on the transmural pressure (the pressure difference between the inside and outside of the tube). Because pressure falls along the length of the vessel, tubes of constant dimensions and composition must taper along their length. Thus, even under conditions of constant initial pressure the vessels would not meet the
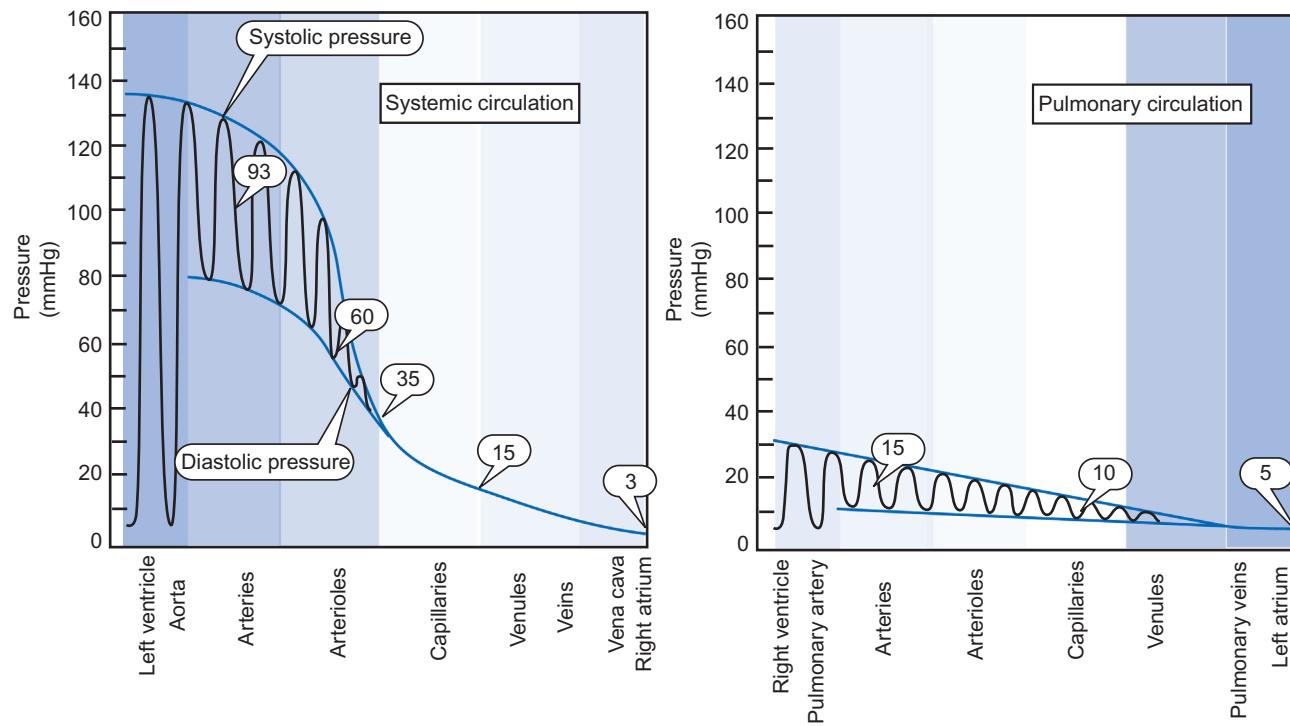

FIGURE 5.9.8 Pressure profiles in the systemic circulation (left) and pulmonary circulation (right). The oscillations in the profile recapitulate pressure variation in time, not distance.
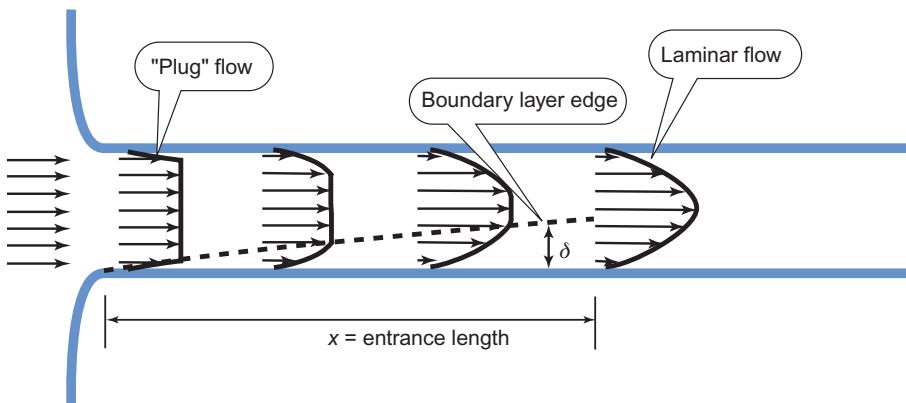
FIGURE 5.9.9 Development of the parabolic velocity profile at the entrance of a blood vessel. At the entrance of the vessel nearly all laminae, or layers, advance with a common velocity, but those laminae near the vessel edge are first slowed by viscous drag. The blunt velocity profile in the inner core becomes smaller as the fluid moves further along. The parabolic velocity profile is achieved at an entrance length, x.

assumption of Poiseuille flow. The generation of pressure pulses by the heart further complicates matters.

The circulatory system satisfactorily meets the assumptions of Newtonian fluid, laminar flow, and no slippage at the vascular wall, but it does not meet the assumptions of steady flow, cylindrical shape of the vessels, and rigid walls. Pulsatile flow, the complicated geometry of the vascular tree, and its distensibility all seriously limit the validity of Eqn [5.9.11] in quantifying blood flow.

THE RATIO OF ΔP TO Q_V DEFINES THE VASCULAR *RESISTANCE*

Despite the fact that Poiseuille's law only approximates the flow through blood vessels, flow through the vasculature is still linear with the pressure difference (actually, the total mechanical energy) between the beginning and end of the vessel. Thus we may write

$$[5.9.14] \qquad Q_v = \frac{\Delta P}{R}$$

where R is the vascular resistance. This is the same as Poiseuille's law if we identify

$$[5.9.15] \qquad R = \frac{8\eta I}{\pi a^4}$$

which is Principle (4) listed at the beginning of this chapter. Equation [5.9.14] is the hydrodynamic analogue of Ohm's law

$$[5.9.16] \qquad I = \frac{\Delta V}{R}$$

where I is the current, ΔV is the voltage drop, and R is the electrical resistance. The resistance of a network of vessels can therefore be modeled as a network of electrical resistances, and we find that vascular resistances in series add like electrical resistances, and vascular resistances in parallel add like electrical resistances in parallel. Thus for vascular resistance in series the resistances add:

$$[5.9.17] \qquad R_{total} = R_1 + R_2 + R_3 + \cdots$$

and for vascular resistances in parallel, the conductances add:

$$\frac{1}{R_{total}} = \frac{1}{R_1} + \frac{1}{R_2} + \frac{1}{R_3} + \cdots$$

$$[5.9.18] \qquad R_{total} = \frac{1}{\dfrac{1}{R_1} + \dfrac{1}{R_2} + \dfrac{1}{R_3} + \cdots}$$

SUMMARY

The total mechanical energy of blood includes its pressure energy, kinetic energy, and gravitational potential energy. Blood flows down its total mechanical energy gradient, but the most important measured variable is the pressure.

Insertion of extra volume into any part of the cardiovascular system raises the pressure. The relationship between the extra volume and the pressure increment is the compliance. The veins are some 19 times more compliant than the arteries. When the heart ejects its stroke volume into the aorta, the resulting increased volume produces a pressure pulse that propagates down the arterial tree. Its velocity is about $5-8$ m s^{-1}, but this increases as arterial compliance decreases with age. The actual flow of blood is about 0.2 m s^{-1}, so the pressure pulse travels much faster than the blood itself. The pressure pulse displays a maximum at the systolic pressure and a minimum at the diastolic pressure. Closure of the aortic valve produces a brief interruption, the dicrotic notch, in the diastolic fall in pressure. As it moves toward the periphery the pressure pulse loses its dicrotic notch but gains a diastolic wave due to pressure wave reflection as it travels down the arteries.

Blood pressure can be measured with a sphygmomanometer. The pulse pressure is the difference between systolic and diastolic pressure. The mean arterial pressure can be estimated as the diastolic pressure plus one-third of the pulse pressure. The size of the pulse pressure depends on the compliance of the arteries and the volume of blood ejected by the heart, the stroke volume. Increasing the stroke volume proportionately increases the pulse pressure. Decreasing the compliance increases the pulse pressure.

Blood pressure falls only a few mmHg over the large arteries but falls more quickly traveling over the arterioles. This indicates that most of the vascular resistance

resides in the arterioles. Poiseuille studied flow through small tubes and found that the flow is proportional to the fourth power of the radius. Although this relationship cannot be rigorously applied to the complicated geometry of the vascular system, its basic conclusion is correct: vascular resistance shows a steep dependence on vascular radius.

Flow in the vascular system is linearly related to the pressure difference that drives it. The ratio of ΔP to flow defines the vascular resistance, which acts like electrical resistances in that vascular resistance of a series arrangement is the sum of the individual resistances, and in a parallel arrangement the conductances add to give the total equivalent conductance.

REVIEW QUESTIONS

1. What is meant by the total mechanical energy of a fluid? Why does kinetic energy contribute to pressure only in the direction of velocity? Why is end pressure greater than lateral pressure? Is this a significant problem in cardiovascular physiology?
2. Where is compliance greatest in the cardiovascular system?
3. Draw an arterial pressure trace. Label its components. How does one determine systolic and diastolic pressure clinically?
4. What is meant by "average arterial pressure"? How is it typically estimated?
5. What is the pulse pressure? What produces it? How does compliance affect it? Is it generally greater or smaller in older persons? Why?
6. What happens to the pressure waveform as it travels down the arteries toward the periphery?
7. What is the typical velocity of the pressure wave? What is the typical average velocity of fluid flow?
8. Define vascular resistance. Where is most of the resistance in the circulatory system?
9. Does Poiseuille's law describe flow in the vascular system? Why or why not?