Circulation System

We now examine how the blood pressure varies with distance along the arteries and veins, including within the capillaries. We then investigate the consequences of non-uniformities 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.

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/mm3; 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 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 0.0012Pa-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.

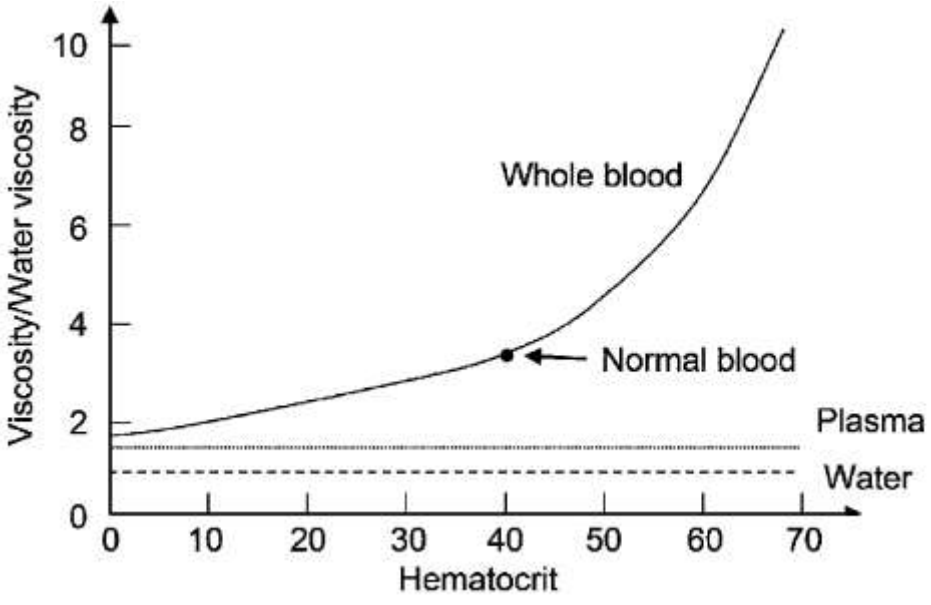

Fig. 8.10. Blood viscosity vs. hematocrit.

The effective viscosity of blood decreases as the shear rate increases. For very slow shear rates, this viscosity is more than $100\times$ that of water, while at the high shear rates characteristic of flow in larger vessels it is about $4\times$ 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.

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 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 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 $\sim 3\times 105$ Pa; it can be stretched to twice its relaxed length. Collagen is much stiffer, with a Young′s modulus of $\sim 1\times 108$ 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 $Y \sim 1 \times 105$ Pa

when relaxed and ~2 × 106 Pa when active. About half the dry mass in vessels is elastin and collagen, with more elastin than collagen in the aorta (~1.5×) and relatively less elastin in other arteries (~0.5×) and veins (~0.3×). Veins contain less elastin than arteries. The fraction of smooth muscle in the dry mass averages to ~50%, and is ~25% in the aorta, and increases to ~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.

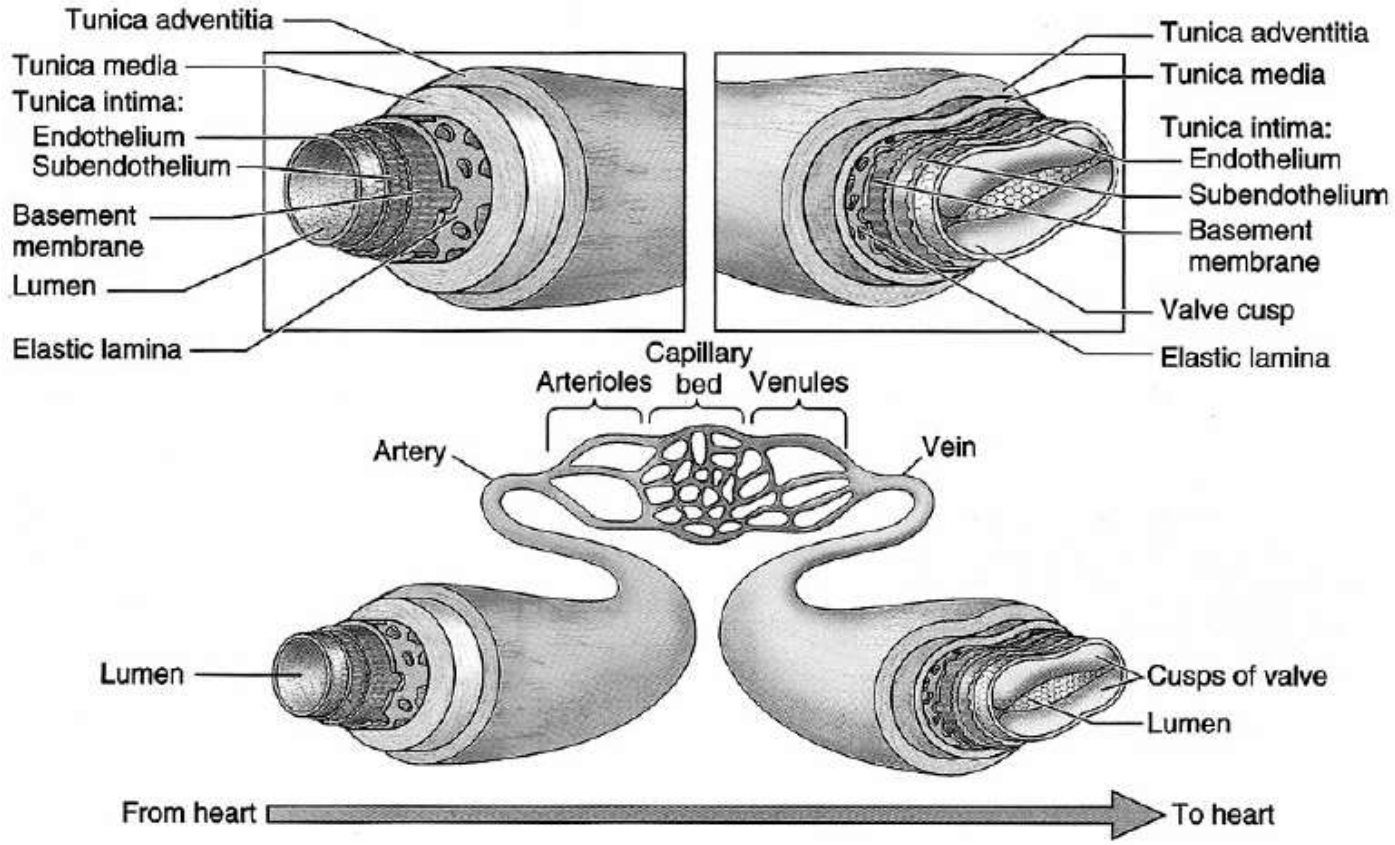

Fig. 8.12. Schematic of the walls of arteries and veins.

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 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 ~80mmHg (P_{diastole}, at diastole) and ~120mmHg (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,

$$P_{mean} = \frac{P_{systole} + 2P_{dystole}}{3} \qquad (8.1)$$

or (1/3)120mmHg + (2/3)80mmHg $\sim$94mmHg in this example. This difference in pressure of 40mmHg between systole and diastole is the *arterial pulse pressure* P_{pulse}. We will see that blood flows at a speed of $\sim$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.

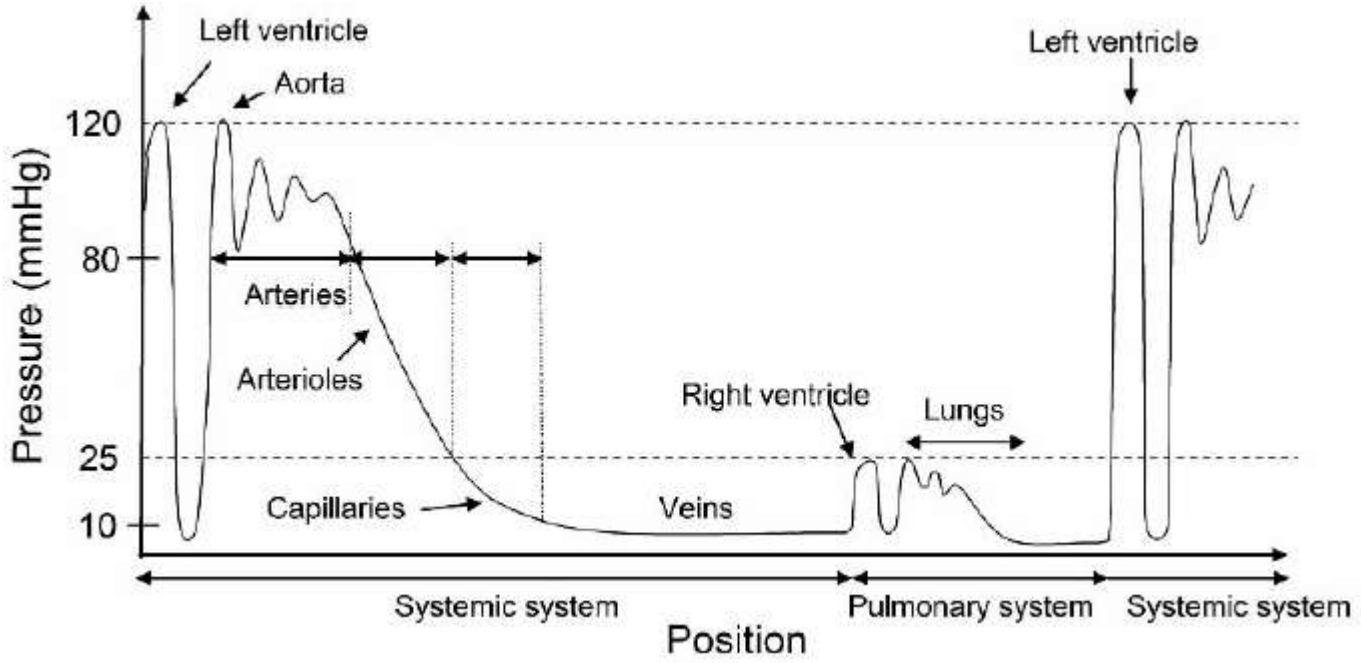

Fig. 8.13. Blood pressure along the circulatory system for a person lying horizontally.

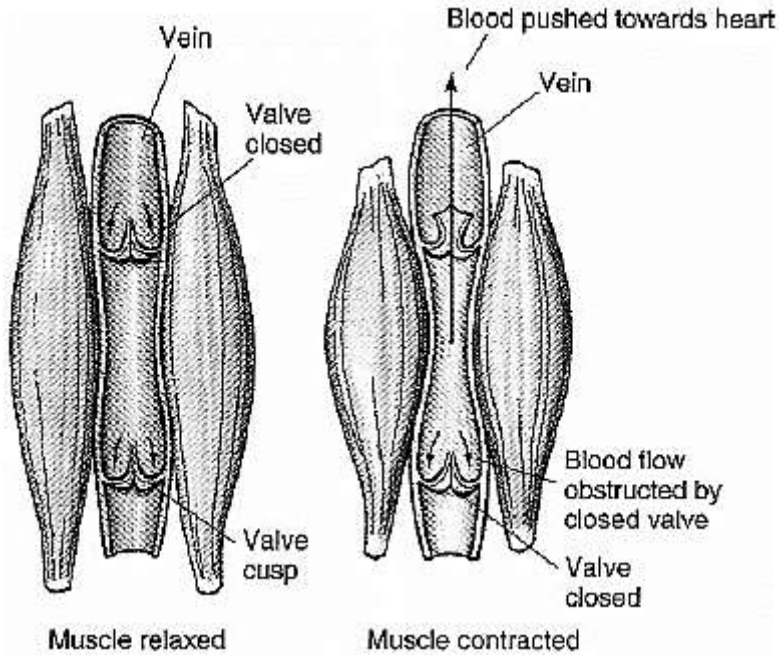

Fig. 8.14. Musculovenous pump of veins, with outward expansion of the bellies of contracting muscles pumping the blood back to the heart against gravity and distal valves closing to prevent backflow.

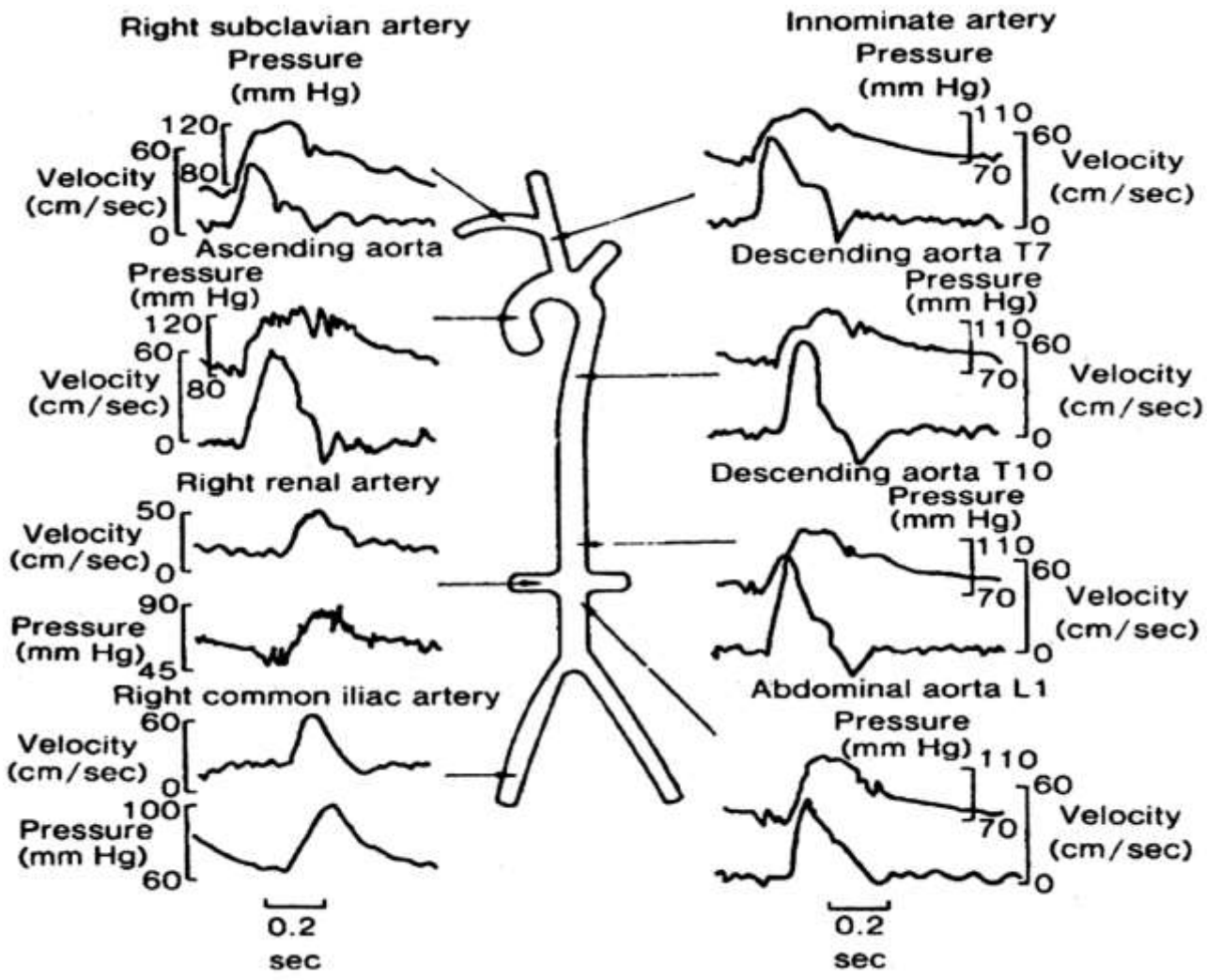

Fig. 8.15. Simultaneous pressure and flow velocity at different points in the human arterial tree for a person lying horizontally. All data were taken from one patient except for the right renal artery and the right common iliac artery.

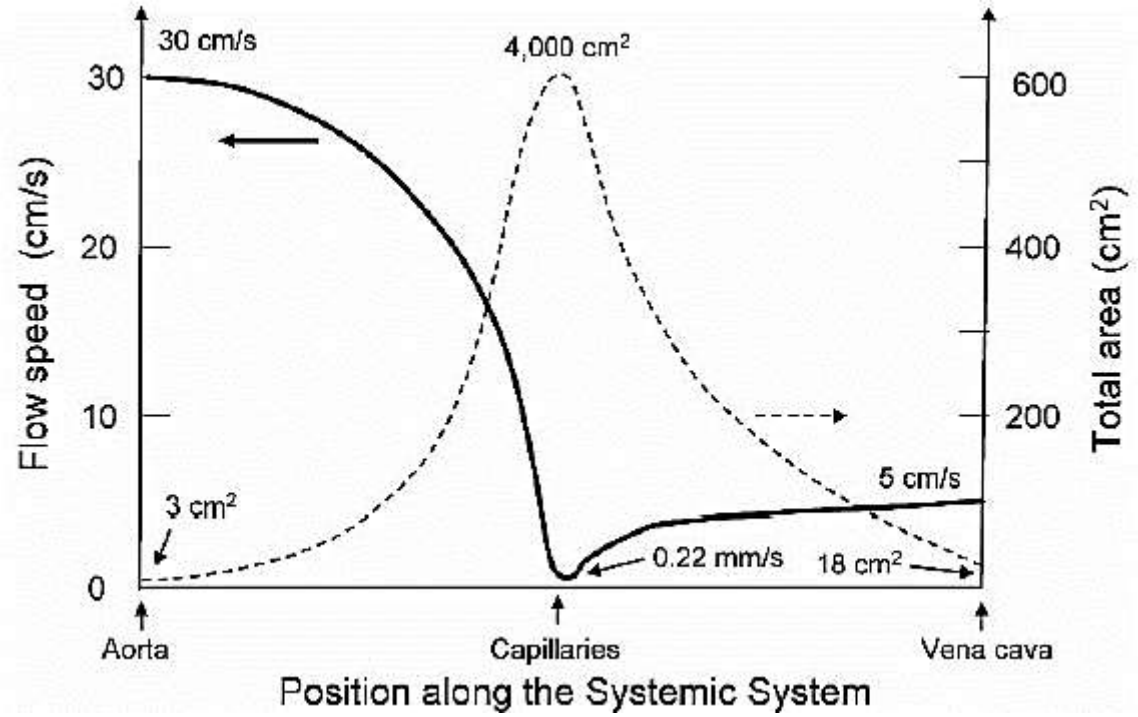

Fig. 8.16. Flow speed (*solid curve*) and total area (*dashed curve*) in the systemic circulation system.

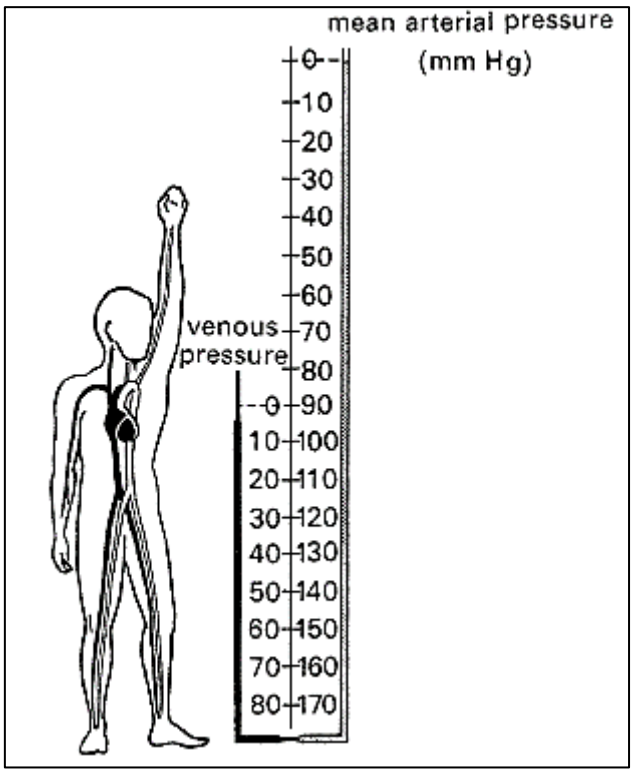

Fig. 8.17. Mean arterial and venous (gauge) pressures for a vertical person.

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 ρgh, 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 ρ = 1.06 g/cm3 = 1,060 kg/m3, g = 9.8 m/s2 and h = 1 m, this pressure is 10,400N/m2 = 10,400Pa = 79mmHg (with 1MPa = 7,600 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 ρgh 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 cm, so the pressure drop is about 30mmHg (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 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.

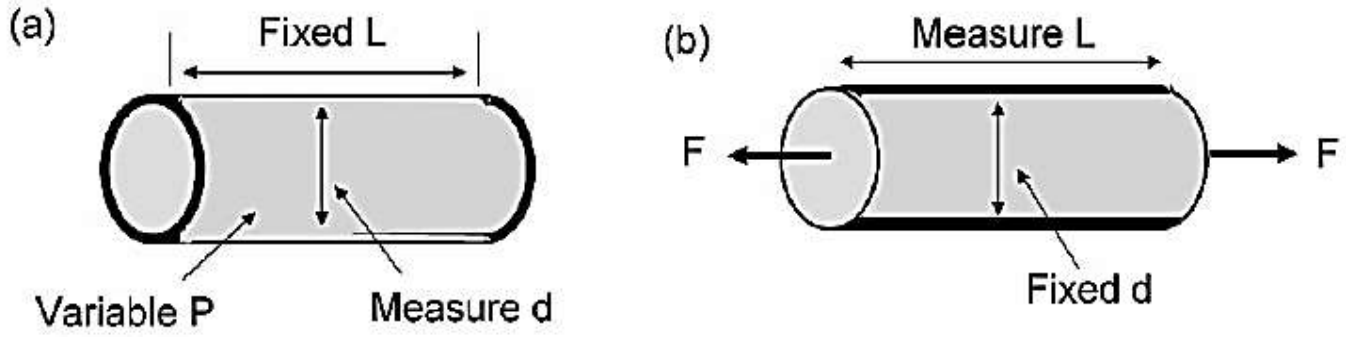

Fig. 8.18. Measuring the mechanical properties of blood vessels by fixing the vessel (**a**) length (which is called *inflation*) or (**b**) diameter (which is called *extension*). Some investigators do combined loading, with combinations of inflation, extension, and torsion

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 beginning of the vessel, the pressure is $P1$ 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.

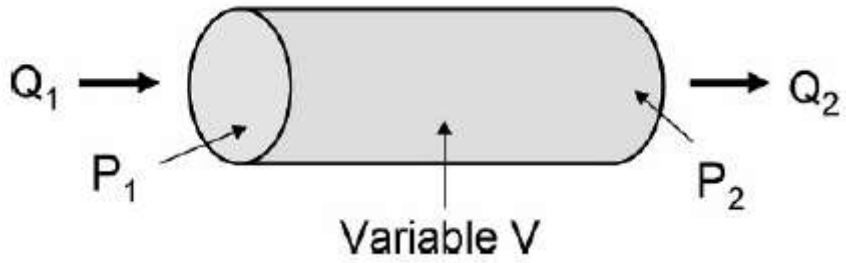

Fig. 8.19. Blood flow: general vessel

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

$$P_1 - P_2 = R_{flow} Q \qquad (8.2)$$

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

where the vascular resistance is $R_{flow} = 8\mu L/\pi r^4$ for a tube with radius r. The former equation has the same form as Ohm′s Law $V_{elect,1} - V_{elect,2} = R_{elect}I$, 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

$$V(P) = C_{flow}(P - P_{exit}) = C_{flow}P \qquad (8.4)$$

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 relation is

$$V(P) = V_d + C_{flow}P \qquad (8.5)$$

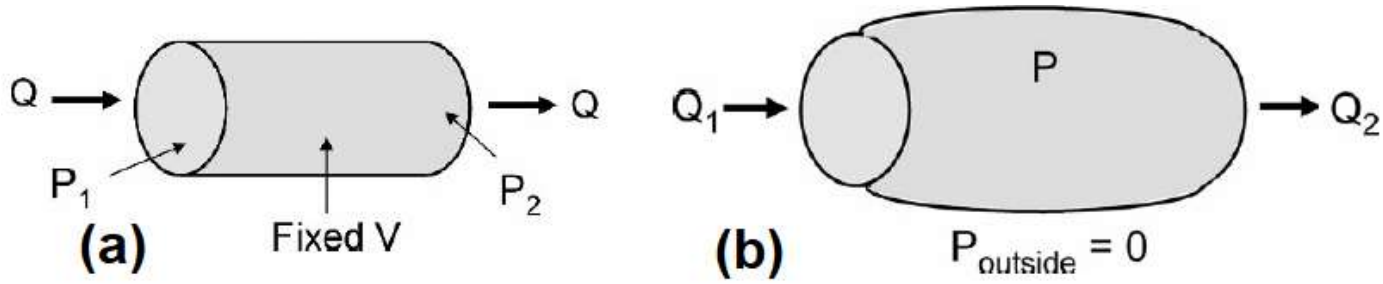

Fig. 8.20. (a) Blood flow: ideal resistance vessel, (b) Blood flow: ideal compliance vessel

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

$$r(P) = r_d + \frac{C'_{flow}}{2}P \qquad (8.6)$$

where r_d is the radius with no pressure difference. Therefore

$$\frac{dr}{dP} = \frac{C'_{flow}}{2} \qquad (8.7)$$

Because $V = \pi r^2$ and $V_d = \pi r^2 d$, 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 $\Delta P = (8\mu L/\pi r^4)Q$ 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 $R_{\text{flow}},0 = 8\mu L0/\pi r^4{}_0$ for a standard radius r_0 = 1cm and standard length L_0 = 1 cm and scale the results for each specific case. With the viscosity μ = 4.0 $\times 10^{-3}$ Pa-s = 4.0 $\times 10^{-3}$ (N-s/m2) = 4.0 $\times 10^{-2}$ poise for whole blood at 37 °C.

$$R_{flow,0} = \frac{8*(4*10^{-3}N.\frac{s}{m^2})(1cm)}{\pi(1cm)^4} = \frac{1.02*10^{-2}N/m^2}{cm^3/s} = \frac{7.7*10^{-5}mmHg}{cm^3/s} \qquad (8.8)$$

where we have used 0.1N/mm^2 = 1 atm. = 760 mmHg. The units in (8.8) are mmHg-s/cm^3. This is the resistance when the pressure difference is 1mmHg 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

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

and so

$$\Delta P = R_{flow}Q = 7.7 * 10^{-5}mmHg\frac{L(in\ cm)}{(r(in\ cm))^4}Q(in\frac{cm^3}{s}) \qquad (8.11)$$

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 $\sim Q_t/n$.

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

$$\Delta P = 7.7 * 10^{-5}mmHg\frac{L(in\ cm)}{(r(in\ cm))^4}\left(\frac{80}{n}\frac{cm^3}{s}\right)$$

$$= \frac{0.0062 mmHg}{n} \frac{L(in\ cm)}{\left(r(in\ cm)\right)^4} \qquad (8.12)$$

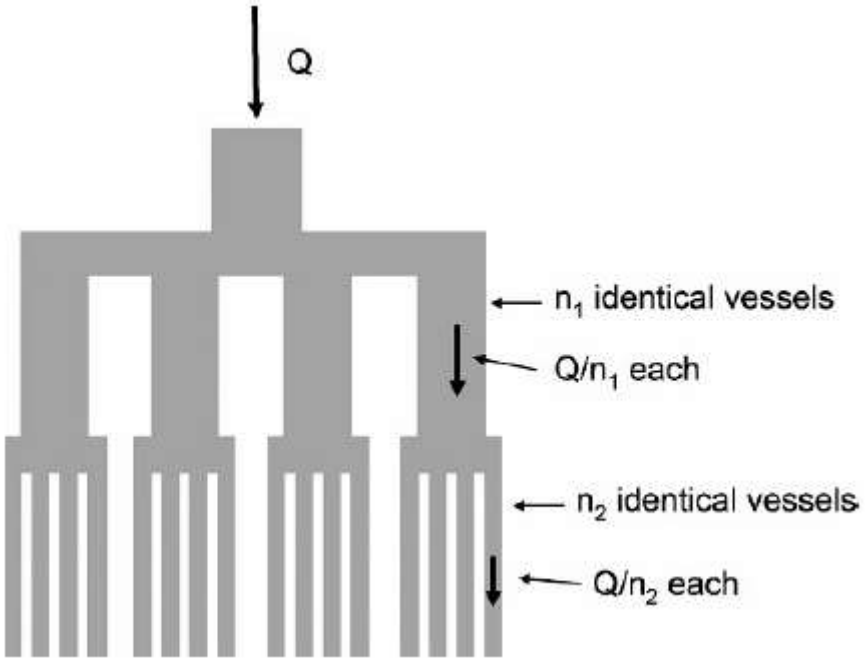

Fig. 8.22. Schematic of blood flow in idealized branching vessels

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 $r \sim 1.25$ cm and $L \sim 10$ cm, and so ΔP across the aorta is 0.025 mmHg, which is insignificant.

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

Smallest arteries and arterioles. There are about 5×105 arterioles with $r \sim 30$ μm and $L \sim 0.6$ cm = 6 mm, and so ΔP is 91 mmHg, which is very significant.

Capillaries. There are about 1010 capillaries with $r \sim 3.5$ μm and $L \sim 0.2$ cm = 2 mm, and so ΔP is 8.2 mmHg, which is fairly significant.

We could have just plugged the parameters for each vessel directly into Poiseuille′s Law, 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, $(\Delta P/L) = (8\mu/\pi r^4)Q$ or, recognizing that this change in pressure is negative,

$$\frac{dp}{dx} = -\frac{8\mu}{\pi r^4} Q \qquad 8.14$$

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 vessel 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 because we are modeling blood as a Newtonian fluid. However, the parabolic profile of blood flow speed in a resistive vessel is not quite accurate. This assumes steady-state flow, which begins only a certain distance from a bifurcation تشعبات. Furthermore, because whole blood is not a Newtonian fluid, the steady-state profile is not parabolic, as is seen in Fig. 7.13.

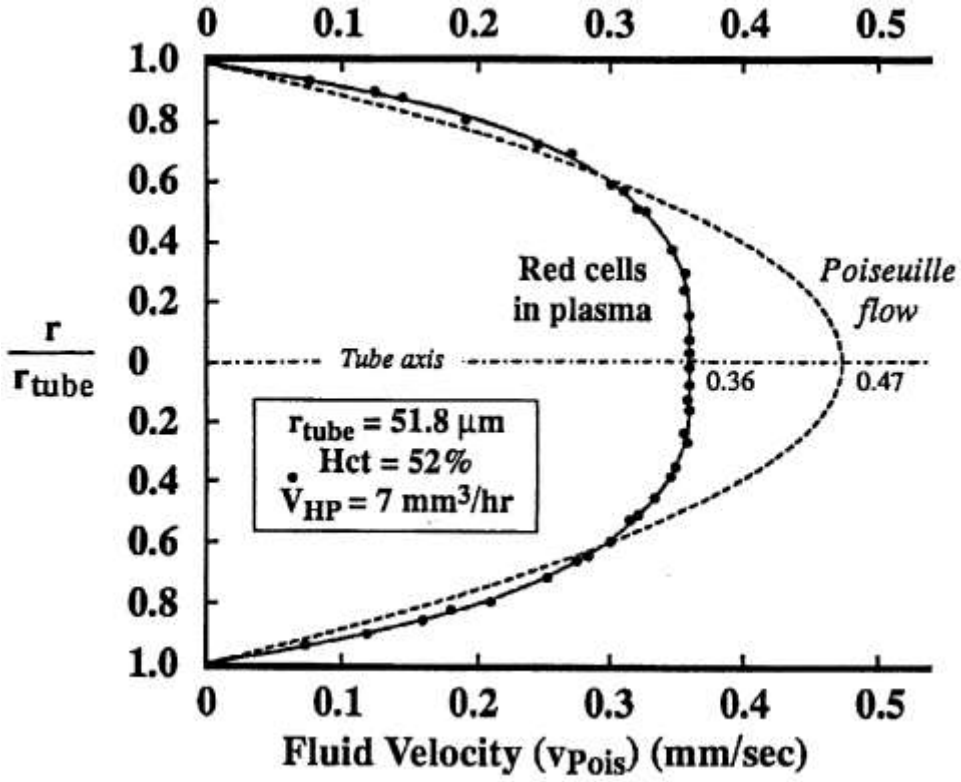

Fig. 7.13. Velocity flow profile of whole blood is blunted relative to the ideal parabolic flow of a Newtonian fluid.

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 $\sigma = T/w$.

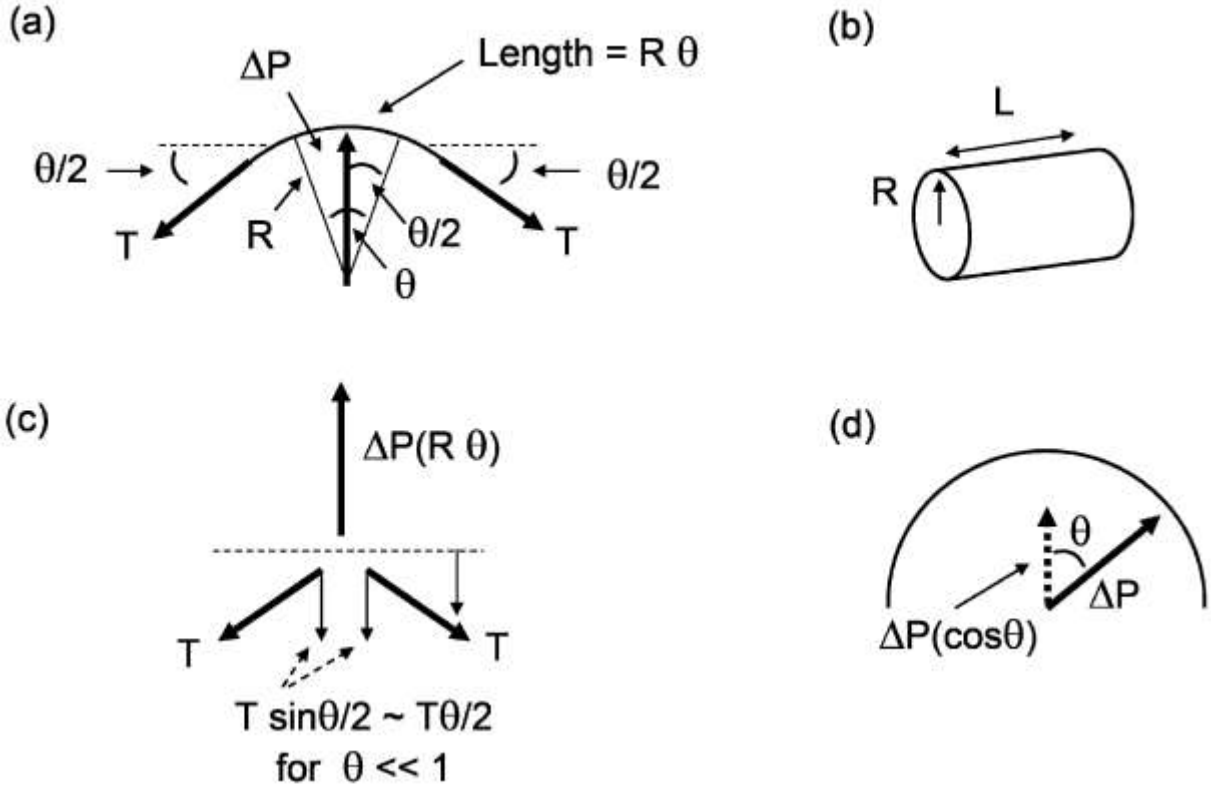

Fig. 7.4. Derivation of the Law of Laplace for the cylinder in (**a**), with the force diagram for a section of a cylinder in (**b**), leading to the force diagram in (**c**), and the resolution of pressures for analysis of a half cylinder in (**d**)

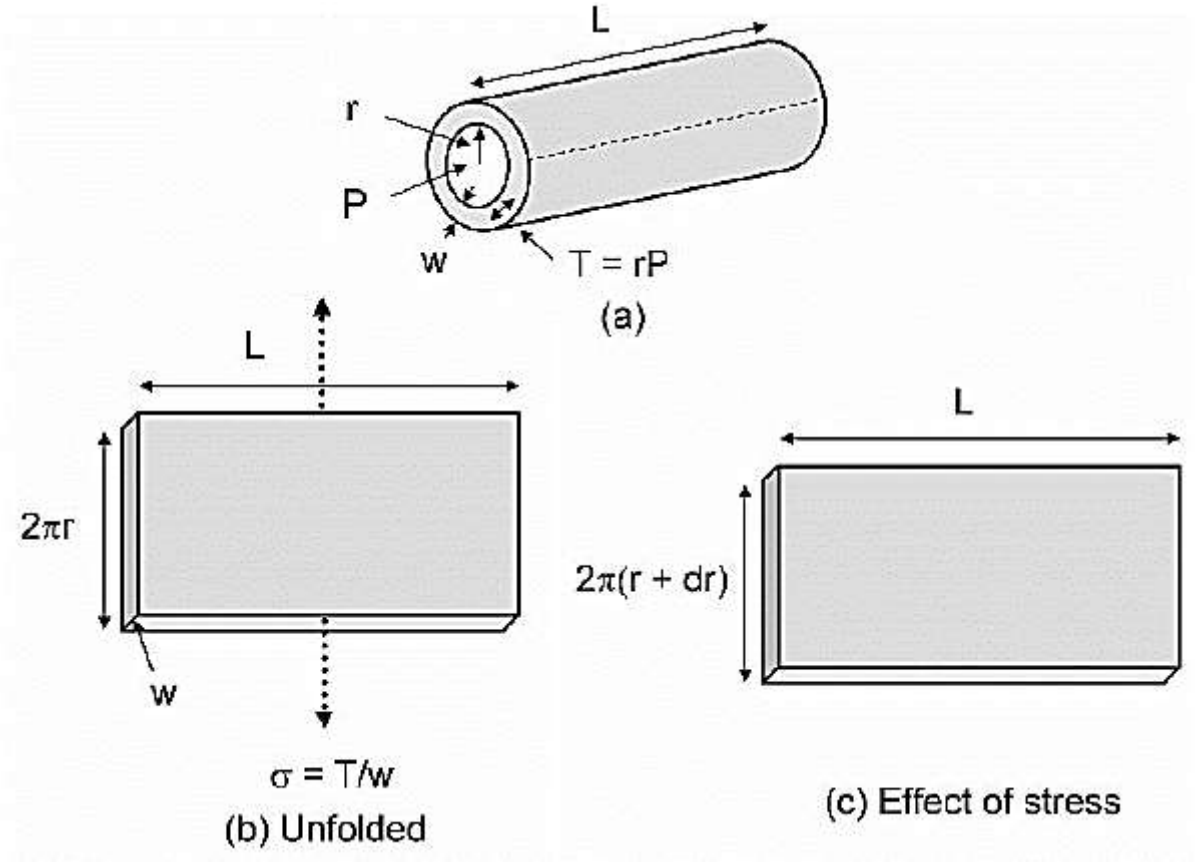

Fig. 8.23. Compliance vessels: unfolding the vessel