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1. Introduction

Hemodynamics is the study of the relationship among physical factors affecting blood flow through the vessels. In this chapter these factors and their relationship were discussed.

2. Blood flow is a function of pressure difference and resistance (Darcy’s law)

Blood flow (F) through a blood vessel is determined by two main factors: (1) pressure difference (ΔP) between the two ends of the vessel and (2) the resistance (R) to blood flow through the vessel (Fig. 1).

![Fig. 1. Blood flow through a blood vessel.](image)

The equation relating these parameters is:

\[ F = \frac{\Delta P}{R} \]  

(1)

This equation is called Darcy’s law or Ohm’s law.

Flow (F) is defined as the volume of blood passing each point of the vessel in one unit time. Usually, blood flow is expressed in milliliters per minute or liters per minute, but it is also expressed in milliliters per second.

Pressure which is the force that pushes the blood through the vessel is defined as the force exerted on a unit surface of the wall of the tube perpendicular to flow. Pressure is expressed as millimeters of mercury (mmHg). Since the pressure is changing over the course of the blood vessel, there is no single pressure to use; therefore the pressure parameter used is pressure difference (ΔP), also called pressure gradient, which is the difference between the pressure at the beginning of the vessel (P₁) and the pressure at the end of the vessel (P₂), i.e.
$\Delta P = P_1 - P_2$. As seen in the Darcy’s law, $\Delta P$ is the cause of the flow; with no pressure difference there would be no flow. The pressure energy is produced by the ventricle and it drops throughout the vessel due to resistance. In other words, resistance is the cause of the pressure drop over the course of a vessel.

Resistance is how difficult it is for blood to flow from point 1 to point 2. Resistance impedes flow and it is a measure of interactions between flowing particles (including molecules and ions) themselves and interactions between flowing particles and the wall of the vessel. As seen Darcy’s law, resistance is the impeding cause of the flow; the bigger the resistance the lesser the flow. If the resistance is $\infty$ (complete closure of the vessel) there will be no flow. The resistance equation is:

$$ R = \frac{8 \eta L}{\pi r^4} \quad (2) $$

Where $\eta$ = fluid viscosity

$L$ = vessel length

$r$ = inside radius of the vessel

Viscosity represents the interactions between flowing particles themselves and radius represents the interactions between flowing particles and the wall of the vessel. The units of viscosity are Pa⋅s = Ns/m², or Poise (dynes⋅s/cm²), with 1 Pa⋅s = 10 Poise.

The red blood cells, erythrocytes (RBCs), constitute 99% of the suspending particle volume of blood. Therefore viscosity of blood depends on the concentration of various constituents of plasma and volume % of red blood cells (hematocrit), as well as size, shape and deformability of RBCs. In a healthy individual all these parameters are constant, therefore blood viscosity is constant and viscosity is not a mean of control (regulation) of the resistance. In abnormal situations viscosity abnormally affects the total resistance. Low hematocrit, as in anemia, decreases viscosity of blood. Inversely, polycythemia increases viscosity and lowers blood flow. In sickle cell anemia the erythrocytes are misshapen and inflexible causing serious disturbances of regional blood flow.

In the resistance equation, $L$ represents the vessel length. Since the length of the vessels of the body is constant, $L$ could not be used for control of the resistance.

Resistance has an inverse relation with the 4th power of $r$ (inside radius of the vessel); therefore radius of the vessel has the most powerful effect on the resistance, so that with small changes in radius, resistance will change dramatically. Radius is the main factor for control of the resistance by the cardiovascular system. Radius of the vessels of the body is controlled by the sympathetic system.

Resistance is mainly located in the arterioles. Assuming an aortic radius of 15 mm and an (arbitrary) length of 50 cm and an arteriole with a radius of 7.5 $\mu$m and a length of 1 mm. The ratio of the radius is 2000 and the length ratio is ~500, therefore the resistance ratio would be $(2000)^4/500$, i.e., $\sim 3 \times 10^{10}$. It means that the resistance of a single arteriole is $3 \times 10^{10}$ as large as that of a 50 cm long aorta. Since there are $3 \times 10^8$ parallel arterioles, their total resistance is about $3 \times 10^{10}/3 \times 10^8 = 100$ times as large as the resistance of the aorta (Westerhof et al. 2010).
Even though all vessels except metarterioles and capillaries are innervated by the sympathetic system, the arterioles receive the most profound innervations and play the main role in the control of the total peripheral resistance by the sympathetic system.

The resistance of any vessel can be calculated by having $\Delta P$ and $F$. For systemic circulation, if mean aortic pressure ($P_1$) is taken to be 100 mmHg and mean right atrial pressure ($P_2$) is 0 mmHg, the pressure difference ($\Delta P$) is 100 mmHg. With a cardiac output of 6 l/min (100 ml/s), the total resistance is $100/100 = 1$ mmHg/ml/s. This unit is called peripheral resistance unit (PRU). Other physical units are used in the clinic and resistance is expressed in dyn$\cdot$s$\cdot$cm$^{-5}$ or Pa$\cdot$s/m$^3$. The total peripheral resistance of the systemic circulation may change from 4 PRU in very strong constriction to 0.2 PRU in great dilation of the vessels. In the pulmonary system, the mean pulmonary arterial pressure is 16 mm Hg and the mean left atrial pressure is 2 mm Hg, giving a $\Delta P$ of 14 mm. With a cardiac output of 100 ml/sec, the total pulmonary vascular resistance is 0.14 PRU, about one seventh of that in the systemic circulation.

In the body, blood vessels arranged in series and in parallel. The arteries, arterioles, capillaries, venules and veins are arranged in series. The total resistance of a series of vessels is equal to the sum of the resistances of each vessel:

$$R_{total} = R_1 + R_2 + R_3 + \ldots$$  \hspace{1cm} (3)

Blood vessels branch extensively to form parallel circuits in all organs and tissues of the body. The total resistance of parallel vessels is calculated by:

$$\frac{1}{R_{total}} = \frac{1}{R_1} + \frac{1}{R_2} + \frac{1}{R_3} + \ldots$$  \hspace{1cm} (4)

As a result, adding a parallel vessel to a circuit will reduce the total resistance. This is the reason that the resistance of each organ alone is far greater than the total peripheral resistance. For example in renal circulation, if blood pressure in the renal artery is taken to be 100 mmHg and that of the renal vein be 10 mmHg and renal flow is taken to be 20 ml/s (1200 ml/min), then $R = 90/20 = 4.5$ PRU, 4.5 times as much as the total resistance of the systemic circulation.

### 2.1 Poiseuille’s law

In equation $F = \Delta P/R$ if we substitute $R$ with its equation results in:

$$F = \frac{\pi \Delta P L}{8 \eta L}$$  \hspace{1cm} (5)

This is called Poiseuille’s law. As seen, flow is proportional to $\Delta P$ which is the main cause of flow. Flow is also proportional to the $4^{th}$ power of internal radius of the vessel indicating the great importance of the radius for flow.

### 2.2 Physiological and clinical applications of Darcy’s law

In equation $F = \Delta P/R$ if $P_1$ is increased, since $\Delta P = P_1 - P_2$, $\Delta P$ will increase which results in an increase in blood flow ($F$) and $P_2$. For example, during exercise, contractility of the left ventricle
increases and produces more pressure energy which results in the increase of aortic pressure 
\( (P_1) \), causing blood flow to various organs and capillary pressure \( (P_2) \) to increase. On the 
contrary, a decrease of \( P_1 \) results in a decrease of flow and capillary pressure.

An increase in \( P_2 \) results in a decrease of \( \Delta P \) and blood flow. For example if the venous 
resistance increases or atrial pressure \( (P_2) \) increases, such as in heart failure, \( \Delta P \) will be lower 
than that of the normal and blood flow will decrease. This subsequently results in a small 
increase in the arterial pressure \( (P_1) \). Therefore a change in either one of the \( P_1 \) or \( P_2 \) causes a 
similar change in the corresponding \( P \) which is smaller than the first one. It is smaller, since 
resistance always causes a pressure drop between the two points of the vessel. For example, 
\( \uparrow P_1 \rightarrow \uparrow \Delta P \) (1st) \( \rightarrow \uparrow F \rightarrow \uparrow P_2 \rightarrow \downarrow \Delta P \) (2nd), but flow is still higher, since due to resistance, 
the magnitude of the increase of \( P_2 \) is smaller than the increase of \( P_1 \), therefore the 
magnitude of the second change (decrease) of \( \Delta P \) is smaller than the first change (increase) 
of \( \Delta P \).

Changing the resistance by adjusting the radius of the vessels is the main mechanism of 
controlling blood flow to each tissue and organ, called local control of blood flow. It is also 
one of the two major mechanisms (control of the heart and the resistance) to control arterial 
blood pressure.

Darcy’s equation \( (F = \Delta P/R) \) could be rewritten as: \( \Delta P = P_1 - P_2 = FR \). If \( R \) is increased by 
decreasing the radius, other three parameters of the equation will change. The first thing 
that happens is the reduction of flow, exactly as expected from the Darcy’s equation. 
Reduction of flow then causes a pile up of flowing materials (such as blood) before 
resistance and a decrease in blood volume after the resistance; thus \( P_1 \) will increase and \( P_2 \) 
will decrease and subsequently \( \Delta P \) will get bigger, exactly as expected from the 2nd form of 
Darcy’s equation. The resultant increase in \( \Delta P \) is always quantitatively smaller than the 
primary increase of \( R \); therefore \( F \) is always less than before. The steps could be summarized 
as follows:

\[
R \uparrow \Rightarrow F \downarrow = \frac{\Delta P}{R} \uparrow \Rightarrow P_1 \uparrow \& P_2 \downarrow \Rightarrow \Delta P \uparrow \Rightarrow F \downarrow = \frac{\Delta P}{R} \uparrow
\]

Exactly opposite will happen if \( R \) is decreased. As seen, any change in \( R \) will change both \( P_1 \) 
and \( P_2 \) in opposite to each other. For example when some one turns the valve of a tap clock 
wise, the radius of the outlet is decreased, flow decreases, output pressure \( (P_2) \) decreases 
and the pressure of pre-valve water \( (P_1) \) increases.

Based on Darcy’s law, if the cardiovascular system is to increase blood flow, it could either 
increase \( \Delta P \) by increasing the heart work, or decrease \( R \) by decreasing sympathetic outflow 
to the vessels, especially to arterioles, resulting in a decrease of resistance. Also it could do 
both \( (\Delta P \uparrow \& R \downarrow) \). In local control mechanism, only local resistance is adjusted to control 
blood flow to a tissue. High metabolism of a tissue changes the concentration of some 
chemical factors including oxygen. These factors make metarterioles and precapillary 
sphincters dilate. Based on Darcy’s law, blood flow to that tissue increases so that the blood 
supply to that tissue will be proportional to its metabolism.

When arterial pressure is low, baroreflex stimulates the heart by increasing contractility and 
heart rate which results in higher \( P_1 \). Baroreflex also increases the total peripheral resistance 
which results in higher \( P_1 \).
Resistance causes pressure drop across the vascular system (Fig. 2). In the large arteries, resistance is relatively small and pressure drop is small. The small arteries have moderate resistance to blood flow. Resistance is highest in the arterioles, which are sometimes referred to as the stopcocks of the vascular system. Therefore, the pressure drop is greatest across the terminal part of the small arteries and the arterioles (Fig. 2).

Resistance vessels make pressure drop from ~100 to 30 mmHg. Based on Darcy’s law, high resistance of these vessels increases the $P_1$ (arterial pressure) and decreases the $P_2$ (capillary pressure). Both effects are absolutely necessary for survival. High arterial pressure makes it possible for blood to reach all parts of the body especially to the head which is at a higher level than the heart. The second effect, low capillary pressure, is also very useful because it prevents the capillaries from being damaged and it is necessary for stable transport between the capillaries and the interstitial fluid. High capillary pressure makes capillaries too permeable so that proteins can cross the endothelium which results in edema.

Fig. 2. Pressure drop across the vascular system in the hamster cheek pouch. AP, mean arterial pressure; VP, venous pressure (From Bern et al., Physiology, 2007, with permission).

Anaphylaxis is an allergic condition in which the cardiac output and arterial pressure often decrease drastically. It results from an antigen-antibody reaction after an antigen to which the person is sensitive enters the circulation, causing secretion of histamine by basophile and mast cells. Histamine dilates the arterioles, resulting in greatly reduced arterial pressure that could result in coma and death. Too much vasodilator drugs also could produce similar effect.

In aneurysm disease, part of an artery dilates abnormally. Based on Darcy’s law, blood flow to the zone perfused by that artery is increased ($F_\uparrow$) which results in a higher pressure in microcirculation of that zone ($P_2\uparrow$), which may produce pain and damage.
In coarctation of descending aorta, a local malformation marked by deformed aortic media, causes narrowing of the lumen. As expected from Darcy’s law, blood flow to the lower parts of the body is seriously decreased ($F \downarrow$). As a consequence, the arterial pressure in the lower part of the aorta decreases ($P_2 \downarrow$) and of the upper part of the aorta may be 40-50 per cent higher ($P_1 \uparrow$) than the lower aorta. Due to the low renal blood pressure, water and salt retention occurs that eventually returns the blood pressure of the lower part of the body to normal and produces hypertension in the upper part of the body.

In heart ischemic diseases, narrowing or obstruction of one or more coronary arteries, decreases or ceases blood flow to the regions supplied by the affected arteries.

In aortic stenosis, the diameter of the aortic valve opening is reduced significantly, and the aortic pulse pressure (difference between systolic and diastolic pressure) is decreased significantly because of great decrease of systolic pressure. Based on Darcy’s law, due to high resistance of aortic valve, $P_1$ (ventricular pressure) increases and $P_2$ (aortic systolic pressure) decreases. This is exactly what we see in the disease. Due to high ventricular pressure, ventricle hypertrophy may occur.

Migraine, is a symptom complex of periodic headaches, often with irritability, nausea, vomiting, constipation or diarrhea and photophobia. It is preceded by constriction of some cranial arteries, which results in low blood flow to the affected regions and consequently results in prodromal sensory, especially ocular symptoms. Then remarkable vasodilation of those cranial arteries occurs resulting in overperfusion of the affected regions which produces other symptoms, especially headache.

3. Laminar or turbulent flow

Blood flow in the straight vessels, is normally laminar. Blood moves in smooth parallel concentric layers. As flow increases, the fluid motion becomes wavy, leading to vortices in different seemingly random directions. This irregular fluid motion is called turbulence (Fig. 3).

![Fig. 3. Laminar and turbulent flow](https://www.intechopen.com)
In turbulent flow the resistance to flow is higher and energetically is more costly than laminar flow, since part of the mechanical energy is lost in the erratic motion between the fluid particles. The probability of turbulence is related to blood density, velocity, the diameter of the vessel and the viscosity of the blood. To judge whether a fluid flow is laminar or turbulent, the Reynolds number (Re, a dimensionless parameter: has no unit) is often used. Re is defined as:

$$Re = \frac{\rho v D}{\eta}$$

\(\rho\) is the fluid density, \(v\) is the mean fluid velocity; \(D\) is the tube inner diameter and \(\eta\) is the fluid viscosity. The Reynolds number reflects the ratio of inertia and viscous effects. The critical Reynolds number is 2200. For low Reynolds numbers (<2200) the viscous effects are dominant and flow is laminar, but for high Reynolds numbers (>2200), flow is turbulent. For transitional numbers around the critical Reynolds number of 2200 flow is neither strictly laminar nor strictly turbulent (Westerhof et al. 2010, p- 22).

At normal resting conditions, arterial flows are laminar. But in heavy exercise, where flow may increase as much as five-folds, the Reynolds number may get higher than the critical value and turbulence occurs.

Laminar flow can be disturbed at the branching points of arteries resulting in turbulence which may deposit the atherosclerotic plaques.

Turbulence is delayed in accelerating flow whereas occurs faster in decelerating flows. For example turbulence occurs distal to a stenosis. Fluid particles accelerate through the narrow part of the stenosis and decelerate fast in the distal expanding part resulting in turbulence. Turbulence in severe stenosis can be initiated for Reynolds numbers as low as 50 (Westerhof et al. 2010, p- 23). This turbulence also widens the vessel after the stenotic part. Constriction of an artery likewise produces turbulence and sound beyond the constriction. This is the reason that murmurs are heard over arteries constricted by atherosclerotic plaques and the sounds of Korotkoff heard when measuring blood pressure (Barrett et al. 2010, p- 540). In severe anemia, because of low viscosity, functional cardiac murmurs are often heard.

### 4. Bernoulli’s principle

Regarding Darcy’s law alone, some aspects of hemodynamics seem puzzling. For example, mean arterial pressure of aorta is about 100 mmHg while it is 180 mmHg in the foot arteries during standing. The very high arterial pressure of the foot is due to gravitational force, as a column of blood with an altitude of ~ 130 cm produces a high pressure in the foot’s arteries. Based on Darcy’s law, since the pressure in the foot’s arteries is higher than aorta, blood should move upward from the foot arteries to aorta, which is not the case. Also blood pressure in the venous sinuses of the brain is highly negative, while the right atrial pressure is ~0. Again based on Darcy’s law, blood should move upward from right atrium to venous system of brain, which is not the case. Such problems are solved by Bernoulli’s principle. Bernoulli’s theory states that flow between point A and point B is dependent on the total mechanical energy difference between A and B, not on pressure difference alone. Total mechanical energy consisted of pressure energy, potential energy and kinetic energy. The pressure energy equals pressure \(\times\) volume \((P \times V)\). The potential energy equals fluid mass...
(m) \times \text{gravitational force (g)} \times \text{height (h)}. \text{Kinetic energy equals mass (m) \times velocity squared (v^2) divided by 2 (m \times v^2/2). Thus:}

\text{Total mechanical energy} = PV + mgh + \frac{1}{2}mv^2 \quad (7)

Based on the conditions, these pressures could easily convert to each other. For example consider the model presented in figure 4 (Burton 1972). This figure demonstrates an experiment showing some basic hydraulic points. In this experiment, flow in the tube is constant. As seen in figure 4, flow is driven by the gradient of total mechanical energy. At the first part, cross-sectional area (A) is 6 and velocity (v) is 1. Based on the equation \( V = \frac{F}{A} \) (V: velocity, F: flow, A: cross-sectional area), as at the middle of the tube cross-sectional area gets smaller, velocity increases with the same ratio.

Fig. 4. Flow is driven by the total mechanical energy difference. In the middle of the tube, the cross-sectional area (A) gets smaller resulting in an increase of velocity (v). In other words, pressure energy is converted to kinetic energy. In the third part of the tube, opposite will happen. Pr.E., pressure energy; K.E., kinetic energy. (Data from Burton 1972).

It means that pressure energy is converted to kinetic energy. This is shown by the numbers at the bottom of the figure and is displayed on the middle vertical tube. Since there are both pressure and total mechanical gradients, flow from the first part to the second part of the tube is consistent with both Darcy’s and Bernoulli’s equations. The third part of the tube gets wider again resulting in an increase of pressure energy and a decrease of kinetic energy. Here kinetic energy is converted to pressure energy. This shows that these three mechanical energies can readily convert to each other. Flow from the middle part to the third part is not expected from Darcy’s law, but is consistent with Bernoulli’s principle. Another important point shown in this experiment is that due to resistance, total mechanical energy is decreasing over the course of the tube.
Now we can explain the puzzling examples mentioned above. In the upright posture, the aortic blood possesses much more gravitational potential energy than the foot arteries, so that the total mechanical energy in the aorta is higher than the foot arteries and makes blood flow from aorta to the foot.

When someone lies down, Darcy’s law is sufficient for explaining blood flow, but in sitting or standing positions, the gravitational potential energy gets quite large and Bernoulli’s law should be applied for more accurate explanation of the blood flow. For example in the upright posture, blood flow to the lung could not be explained well without using Bernoulli’s principle. Since the pressure in the pulmonary arteries is low, the gravitational energy is comparatively large and greatly affects the pulmonary blood flow, so that during diastole, blood does not reach the apex of the lung.

5. Law of Laplace

The law of Laplace gives the relation between transmural pressure, wall tension, radius and wall thickness in a vessel (Fig. 5) as:

$$T = \frac{P \cdot r}{w}$$  \hspace{1cm} (8)

Where $T$ is the force per unit length tangential to the vessel wall called wall tension (dynes/cm), $P$ is transmural pressure, intravascular pressure minus extravascular pressure, in dynes/cm$^2$, $r$ is radius of the vessel in cm, and $w$ is thickness of the vessel in cm. Distending force ($P \cdot r$) tends to pull apart a theoretical slit in the vessel, while the wall tension ($T$) will keep the parts together.

![Fig. 5. Transmural pressure (P) and wall tension (T) in a vessel.](www.intechopen.com)
distending force is higher and the ventricle must work harder to pump the normal stroke volume and it will deteriorates the already diseased ventricle.

6. **Velocity is inversely related to cross-sectional area**

Velocity \( V \) is related to flow \( F \) and inversely related to cross-sectional area \( A \) of a vessel as follows:

\[
V = \frac{F}{A} \quad (9)
\]

As blood vessels branch extensively from aorta to capillaries, cross-sectional area of each vessel decreases while the total cross-sectional area increases.

![Diagram showing velocity and total cross-sectional area in the systemic circulation](https://www.intechopen.com)

Fig. 6. Velocity and total cross-sectional area in the systemic circulation. There is the maximal cross-sectional area and minimal velocity in the capillaries. AO, Aorta; LA, large arteries; SA, small arteries; ART, arterioles; CAP, capillaries; VEN, venules; SV, small veins; LV, large veins; VC, venae cavae. (Bern et al. 2007, p-267, with permission)

As seen in figure 6, capillaries have the maximal total cross-sectional area resulting in the lowest blood velocity. This low velocity provides ample time for exchange between blood and interstitial fluid.

7. **Elasticity and compliance**

When a strip of material with cross-sectional area \( A \), and length \( l_0 \), is subjected to a force \( F \) it will lengthen by \( \Delta l \) (Fig. 7). For a specimen with a larger cross-sectional area the same force will produce a smaller change of the length. Also if the starting length \( l_0 \) is longer, the same force causes a larger length change. To have a unique characterization of the material, independent of the sample primary length and thickness, force is normalized by starting
cross-sectional area, \( \sigma = F/A \) called stress, and length is normalized by starting length \( \varepsilon = \Delta l/l_0 \) called strain. Elasticity is defined as \( E = \sigma / \varepsilon \) (Westerhof et al. 2010, p-49).

The relation between stress and strain for biological material is given in the right part of the figure 7. As seen the relationship between stress and strain for biological material almost always is nonlinear. This nonlinearity implies that a biological material cannot be characterized by a single \( E \). Therefore we should get the local slope of the stress-strain relation for the desired point. This point elasticity is called incremental elasticity (\( E_{\text{inc}} \)). \( E_{\text{inc}} \) increases with strain, i.e., the biological materials become stiffer with increasing stress and strain.

![Fig. 7. Stress-strain relationship for biological materials (taken from snapshot p-49 with permission)](https://www.intechopen.com)

For a vessel or a heart, increasing its blood volume results in an increase in the internal pressure and increasing the internal pressure results in an increase in the volume. Pressure is comparable to stress and volume is comparable to strain. Therefore in cardiovascular physiology, pressure-volume relation (Fig. 8) is normally used instead of stress-strain relation. An advantage of pressure-volume relation is that it can be measured in vivo. It is important to note that pressure-volume relation does not characterize the material alone but includes the structure of the organ as a whole ((Westerhof et al. 2010, p-58). The change of volume per one unit change of pressure is called compliance (\( C = \Delta V/\Delta P \)). The change of pressure per one unit change of volume is called elastance (\( E = \Delta P/\Delta V \)). For biological organs like vessels and heart, the pressure-volume relation is curved toward volume axis indicating that by increasing volume or pressure stiffness increases (Fig. 8). Therefore there is not a single compliance or elastance and for a working point, the tangent of the pressure-volume curve is used. Thus, when comparing compliance or elastance the chosen working point, the pressure at which compliance or elastance was determined, should be reported.
Compliance and elastance depend on the original volume \( V_0 \) of the organ under study. To compare properties of different blood vessels, or hearts, compliance and elastance should be normalized with respect to the original volume of the organ. Normalized compliance is called distensibility \([\text{distensibility} = C/V_0 = \Delta V/(\Delta P \cdot V_0)\] \). Normalized elastance is called volume elasticity \([\text{volume elasticity} = E \cdot V_0 = (\Delta P \cdot V_0) / \Delta V\] \).

### 7.1 Physiological and clinical applications

Distensibility of the veins is 8 times as much as the arteries and the original volume of the veins is 3 times as much as the arteries, thus compliance of each vein is 24 times as much as its corresponding (parallel artery and vein which have the same flow) artery. It means that perfusing a vein and its corresponding artery with the same volume of blood, increases the artery’s pressure 24 times as much as the vein. Therefore veins can store large amount of blood with little increase in pressure. Veins are called capacitance vessels storing 60-70 percent of the total blood volume.

Figure 9 shows the effect of blood pressure on blood flow through an isolated vessel. As expected from Darcy’s law \((F = \Delta P / R)\) increasing pressure results in an increase of flow, but in fact, the effect of pressure on blood flow is greater than expected from Darcy’s law (Fig. 9b), as shown by the upward curving lines in Figure 9a. This is because due to vascular distensibility, increased arterial pressure not only increases the force that pushes blood through the vessels but it also distends the elastic vessels, actually decreasing vascular resistance. Therefore elasticity makes the heart work less to pump normal cardiac output, resulting in longer survival.
In arteriosclerosis, blood vessels are less distensible, therefore they extend less which results in a higher resistance causing hypertension, high pulse pressure and high work load of the heart. These symptoms have serious deteriorating effects on the cardiovascular system.

During systole, due to vascular distensibility, high blood pressure distends the arteries, i.e. some pressure energy is stored in the walls of the arteries as potential energy. During diastole the wall of the arteries return to their diastolic position releasing the stored potential energy to the blood as pressure energy. This function attenuates systolic pressure and increases diastolic pressure resulting in normal pulse pressure (difference between systolic pressure and diastolic pressure) of 40 mmHg. Keeping diastolic pressure reasonably high, keeps blood flowing during diastole. In arteriosclerosis, due to stiffness of the arteries, less pressure energy is stored in the wall of the arteries causing systolic pressure to get abnormally high, resulting in a high pulse pressure which has a deteriorating effect on the arteries.

Another physiological benefit of elasticity is damping of the pulse pressure in the smaller arteries, arterioles, and capillaries. Figure 10 shows typical changes in the pulse pressure as the pulse travels into the peripheral vessels. The intensity of pulsation becomes progressively less in the smaller arteries and eventually disappears in the capillaries. In fact, only when the aortic pulsations are extremely large or the arterioles are greatly dilated can pulsations be observed in the capillaries. Lack of pulsation in the capillaries guarantees stable pressure thus stable permeability and stable transport across the capillaries’ wall.
The cause of progressive diminution of the pulsations in the periphery is twofold: (1) resistance and (2) elasticity of the vessels. Resistance is the cause of pressure drop throughout the vessels, thus decreases the pulse pressure. Elasticity continuously decreases the systole and adds to diastole pressure bringing them closer to each other.

8. References
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The cardiovascular system includes the heart located centrally in the thorax and the vessels of the body which carry blood. The cardiovascular (or circulatory) system supplies oxygen from inspired air, via the lungs to the tissues around the body. It is also responsible for the removal of the waste product, carbon dioxide via air expired from the lungs. The cardiovascular system also transports nutrients such as electrolytes, amino acids, enzymes, hormones which are integral to cellular respiration, metabolism and immunity. This book is not meant to be an all encompassing text on cardiovascular physiology and pathology rather a selection of chapters from experts in the field who describe recent advances in basic and clinical sciences. As such, the text is divided into three main sections: Cardiovascular Physiology, Cardiovascular Diagnostics and lastly, Clinical Impact of Cardiovascular Physiology and Pathophysiology.

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