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Dynamics of Blood Flow

The blood vessels are a closed system of conduits that carry blood from the heart to the tissues and back to the heart. Some of the interstitial fluid enters the lymphatics and passes via these vessels to the vascular system. Blood flows through the vessels primarily because of the forward motion imparted to it by the pumping of the heart, although, in the case of the systemic circulation, diastolic recoil of the walls of the arteries, compression of the veins by skeletal muscles during exercise, and the negative pressure in the thorax during inspiration also move the blood forward. The resistance to flow depends to a minor degree on the viscosity of the blood but mostly upon the diameter of, principally the arterioles. The blood flow to each tissue is regulated by local chemical and general neural mechanisms that dilate or constrict the vessels of the tissue. All of the blood flows through the lungs, but the systemic circulation is made up of numerous different channels in parallel, an arrangement that permits wide variations in regional blood flow without changing total systemic flow.

1. ANATOMIC CONSIDERATIONS

Arteries and Arterioles

The characteristics of various types of blood vessels are shown in Table 1. The walls of the aorta and other arteries of large diameter contain a relatively large amount of elastic tissue. They are stretched during systole and recoil during diastole. The walls of the arterioles contain less elastic tissue but much more smooth muscle. The muscle is innervated by noradrenergic

| | Lumen Diameter | Wall Thickness |
|-----------|-----------------------|-----------------------|
| Aorta | 2.5 cm | 2 mm |
| Artery | 0.4 cm | 1 mm |
| Arteriole | 30 μ m | 20 μ m |
| Capillary | 6 μ m | 1 μ m |
| Venule | 20 μ m | 2 μ m |
| Vein | 0.5 cm | 0.5 mm |
| Vena Cava | 3 cm | 1.5 mm |

nerve fibers, which are constrictor in function, and in some instances, by cholinergic fibers, which dilate the vessels. The arterioles are the major site of the resistance to blood flow, and small changes in their caliber cause large changes in total peripheral resistance.

Capillaries

The arterioles divide into smaller muscle-walled vessels, sometimes called metarterioles, and these in turn feed into capillaries. In some of the vascular beds that have been studied in detail, a metarteriole is connected directly with a venule by a capillary thoroughfare vessel, and the true capillaries are an anastomosing network of side branches of this

thoroughfare vessel. The openings of the true capillaries are surrounded on the upstream by minute small smooth muscle precapillary sphincters. The vasoconstrictor fibers to the arterioles also innervate the metarterioles and precapillary sphincters. When the sphincters are dilated, the diameter of the capillaries is normally about 6 μm , just sufficient to permit red blood cells to squeeze through in single file. As they pass through the capillaries, the red cells become thimble or parachute shaped, with the concavity pointing in the direction of flow. This configuration appears to be due simply to the pressure in the center of the vessel whether or not the edges of the red blood cell are in contact with the capillary walls.

The total area of all the capillary walls in the body exceeds 6300 m^2 in the adult. The walls, which are about 1 μm thick, are made up of a single layer of endothelial cells. The structure of the walls varies from organ to organ. In many beds, including those in skeletal, cardiac, and smooth muscle, the junctions between the endothelial cells permit the passage of molecules up to 10 nm in diameter. It also appears that plasma and its dissolved proteins are taken up by endocytosis, transported across the endothelial cells, and discharged by exocytosis. However, this process can account for only a small portion of the transport across the endothelium. In the brain, the capillaries resemble the capillaries in muscle, but the junctions between endothelial cells are tighter; they only permit the passage of small molecules. In most endocrine glands, the intestinal villi, and parts of the kidney, the cytoplasm of the endothelial cells is attenuated to form gaps called fenestrations. These fenestrations, which are closed by a thin membrane, are 20–100 nm in diameter. They permit the passage of relatively large molecules and make the capillaries porous. In the liver, where the sinusoidal capillaries are extremely porous, the endothelium is discontinuous and there are large gaps between endothelial cells.

Principles of Arterial Flow Dynamics

Laminar Flow and Turbulence

The flow of blood in the blood vessels, like the flow of liquids in narrow rigid tubes, is normally laminar (streamline). Within the blood vessels, an infinitely thin layer of blood in contact with the wall of the vessel does not move. The next layer within the vessel has a small velocity, the next a higher velocity, and so forth, velocity being greatest in the center of the stream (Fig. 1). Laminar flow occurs at velocities up to a certain critical velocity. At or above this velocity, flow is turbulent. Streamline flow is silent but turbulent flow creates sound, frequently presenting in clinical practice as a *bruit*.

The probability of turbulence is also related to the diameter of the vessel and the viscosity of the blood. This probability can be expressed by the ratio of inertial to viscous forces. The mathematical relationship of these forces yields a unitless number called the Reynolds number, named for the man who described the relationship. Basically, the higher the value of the Reynolds, the greater the probability of turbulence.

In humans, critical velocity is sometimes exceeded in the

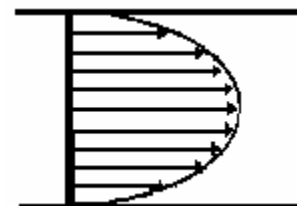


Figure 1. Velocity concentrations in a laminar flow pattern.

ascending aorta at the peak of systolic ejection, but it is usually exceeded only when an artery is constricted. Turbulence occurs more frequently in anemia because the viscosity of the blood is lower. This may be the explanation of the systolic murmurs that are common in anemia.

Average Velocity

When considering flow in a system of tubes, it is important to distinguish between velocity, which is displacement per unit time (e.g., cm/s), and flow, which is volume per unit time (e.g., cm³/s). Velocity (V) is proportionate to flow (Q) divided by the area of the conduit (A): $V = Q/A$.

The average velocity of fluid movement at any point in a system of tubes is inversely proportionate to the *total* cross-sectional area at that point. Therefore, the average velocity of the blood is rapid in the aorta, declines steadily in the smaller vessels, and is slowest in the capillaries, which have 1000 times the *total* cross-sectional area of the aorta. The average velocity of blood flow increases again as the blood enters the veins and is relatively rapid in the vena cava, although not so rapid as in the aorta.

Poiseuille-Hagen Formula

The relation between the flow volume in a long narrow tube, the viscosity of the fluid, and the radius of the tube is expressed mathematically in the Poiseuille-Hagen formula. Since flow volume varies directly and resistance inversely with the fourth power of the radius, blood flow and resistance *in vivo* are markedly affected by small changes in the caliber of the vessels. Thus, for example, flow through a vessel is doubled by an increase of only 19% in its radius; and when the radius is doubled, resistance is reduced to 6% of its previous value. This is why organ blood flow is so effectively regulated by small changes in the caliber of the arterioles and why variations in arteriolar diameter have such a pronounced effect on systemic arterial pressure

Viscosity and Resistance

Blood flow varies inversely and resistance directly with the viscosity of the blood *in vivo*, but the relationship deviates from that predicted by the Poiseuille-Hagen formula. Viscosity depends for the most part on hematocrit, i.e. the percentage of blood volume occupied by red blood cells. In large vessels, increases in hematocrit cause appreciable increase in viscosity. However, in vessels smaller than 100 μm in diameter, i.e., in arterioles, capillaries, and venules, the viscosity change per unit change in hematocrit is much less than it is in large-bore vessels. This is due to a difference in the nature of flow through the small vessels. Therefore, the net change in viscosity per unit change in hematocrit is considerably less in the body than it is *in vitro*. This is why hematocrit changes have relatively little effect on the peripheral resistance when the changes are large. In severe polycythemia, the increase in resistance does increase the work of the heart. Conversely, in anemia, the peripheral resistance is decreased, though the decreased resistance is only partly due the decrease in viscosity. Cardiac output is increased and as a result, the work of the heart is also increased in anemia.

Viscosity is also affected by the composition of the plasma and the resistance of the cells to deformation. Clinically significant increases in viscosity are seen in diseases in which plasma proteins such as the immunoglobulins are markedly elevated and in

diseases such as hereditary spherocytosis, in which the red blood cells are abnormally rigid.

In the vessels, red cells tend to accumulate in the center of the flowing stream. Consequently, the blood along the side of the vessels has a low hematocrit, and branches leaving a large vessel at right angles may receive a disproportionate amount of this red cell-poor blood. This phenomenon, which has been called plasma skimming, may be the reason the hematocrit of capillary blood is regularly about 25% lower than the whole body hematocrit.

Law of Laplace

It is perhaps surprising that structures as thin-walled and delicate as the capillaries are not more prone to rupture. The principal reason for their relative invulnerability is their small diameter. The protective effect of small size in this case is an example of the operation of the law of Laplace, an important physical principle with several other applications in physiology. This law states that the distending pressure (P) in a distensible hollow object is equal at equilibrium to the tension in the wall (T) divided by the 2 principal radii of curvature of the object (R_1 and R_2):

$$P = T(1/R_1 + 1/R_2)$$

The relation between distending pressure and tension is shown diagrammatically in Figure 2.

In the equation, P is actually the transmural pressure, the pressure on one side of the wall minus that on the other. T is expressed in dynes/cm and R_1 and R_2 in cm, so P is expressed in dynes/cm². Consequently, the smaller the radius of a blood vessel, the less the tension in the wall necessary to balance the distending pressure. In the human aorta, for example, the tension at normal pressures is about 170,000 dynes/cm, and in the vena cava it is about 21,000 dynes/cm; but in the capillaries, it is approximately 16 dynes/cm.

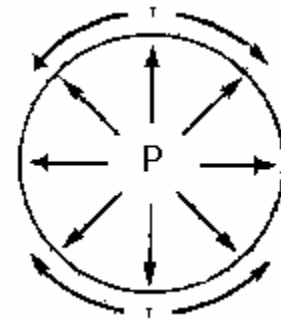


Figure 2. Relation between distending pressure (P) and wall tension (T)

Velocity and Flow of Blood

Although the mean velocity of the blood in the proximal portion of the aorta is 40cm/s, the flow is phasic and velocity ranges from 120cm/s during systole to a negative value at the time of the transient backflow before the aortic valves close in diastole. This short-lived reduction in pressure produces a typical negative deflection of in a Doppler waveform immediately after end systole and just before early diastole called a **dichrotic notch**. In the distal portion of the aorta and in the arteries, velocity is also greater in systole than in diastole, but forward flow is continuous because of the recoil during diastole of the vessel walls that have been stretched during systole. An exception of this continuous forward flow may be found in the peripheral arteries in extremities, where a normal flow reversal is seen in early diastole. Pulsatile flow appears in some poorly understood way to maintain optimal function of the tissues. If an organ is perfused with a pump that delivers a non-pulsatile flow, there is a gradual rise in vascular resistance, and

tissue perfusion fails.

Arterial Pressure

The pressure in the aorta and in the **brachial** and other large arteries in a young adult human rises to a peak value (systolic pressure) of about 120 mm Hg during each heart cycle and falls to a minimum value (diastolic pressure) of about 70-mmHg. The arterial pressure is conventionally written as systolic pressure over diastolic pressure, e.g., 120/70 mm Hg. The **pulse pressure**, the difference between the systolic and diastolic pressure, is normally about 50 mmHg. The **mean pressure** is the average pressure throughout the cardiac cycle. Because systole is shorter than diastole, mean pressure is slightly less than the value halfway between systolic and diastolic pressure. It can actually be determined only by integrating the area of the pressure curve; however, as an approximation, the diastolic pressure plus one-third of the pulse pressure is reasonably accurate.

The pressure falls very slightly in the large and medium sized arteries because their resistance to flow is small, but it falls rapidly in the small arteries and arterioles, which are the main sites of the peripheral resistance against which the heart pumps. The mean pressure at the end of the arterioles is 30–38 mm Hg. Pulse pressure also declines rapidly to about 5 mm Hg at the ends of the arterioles. The magnitude of the pressure drop along the arterioles varies considerably depending upon whether they are constricted or dilated.

Effect of Gravity

The pressure in any vessel below heart level is increased and that in any vessel above heart level is decreased by the effect of gravity. The magnitude of the gravitational effect—the product of the density of the blood, the acceleration due to gravity (980 cm/s/s), and the vertical distance above or below the heart is 0.77 mm Hg/cm at the density of normal blood. Thus, in the upright position, when the mean arterial pressure at heart level is 100 mm Hg, the mean pressure in a large artery in the head (50 cm above the heart) is 62 mm Hg ($100 - [0.77 \times 50]$) and the pressure in a large artery in the foot (105 cm below the heart) is 180 mm Hg ($100 + [0.77 \times 105]$).

3. Methods of Measuring Blood Pressure

Auscultatory Method

The arterial blood pressure in humans is routinely measured by the auscultatory method. An inflatable (sphygmomanometer) is wrapped around the arm and a stethoscope is placed over the brachial artery at the elbow. The cuff is rapidly inflated until the pressure in it is well above the expected systolic pressure in the brachial artery. The artery is occluded by the cuff, and no sound is heard with stethoscope. The pressure in the cuff is lowered slowly. At the point at which systolic pressure in artery just exceeds the cuff pressure, a spurt of blood passes through with each heartbeat and, synchronously with each beat, a tapping sound is heard below cuff. The cuff pressure at which the sounds are first heard is the systolic pressure. As the cuff lowered further, the sounds become louder, then dull and muffled, and finally, in most individuals, they disappear. These are the sounds of Korotkoff. When direct and indirect blood pressure are made

simultaneously, the diastolic pressure correlates better with the pressure at which the sounds muffled than with the pressure at which they disappear.

The sounds of Korotkow are produced by turbulent flow in the brachial artery. The streamline flow in the unstricted artery is silent., but when the artery is narrowed the velocity through the constriction exceeds the critical velocity, and turbulent flow results. AT cuff pressures just below the systolic pressure, flow through the artery occurs only at the peak of systole, and the intermittent turbulence produces a tapping sound. As long as the pressure in the cuff is above the diastolic pressure in the artery, flow is interrupted at least during part of diastole, and the intermittent sounds have a staccato quality. When the cuff pressure s just below the arterial diastolic pressure, the vessel is still constricted, but the turbulent flow is continuous. Continuous sounds have a muffled rather than a staccato quality.

The auscultatory method is accurate when used properly, but a number of precautions must be observed. The cuff must be at heart level to obtain a pressure that is uninfluenced by gravity. The blood pressure in the thighs can be measured with the cuff around the thigh and the stethoscope over the popliteal artery, but there is more tissue between the cuff and the artery in the leg than there is in the arm, and some of the cuff pressure is dissipated. Therefore, pressures obtained using the standard arm cuff are falsely high. The same thing is true when brachial arterial pressures are measured in individuals with obese arms, because the blanket of fat dissipates some of the cuff pressure. In both situations, accurate pressures can be obtained by using a cuff that is wider than the standard arm cuff. If the cuff is left inflated for sometime, the discomfort may cause generalized reflex vasoconstriction, raising the blood pressure. It is always wise to compare the blood pressure in both arms when examining an individual for the first time. Persistent major differences between the pressure on the two sides indicate the presence of vascular obstruction, usually in the subclavian artery on the side with the diminished pressure.

Palpation Method

The systolic pressure can be determined by inflating an arm cuff and then letting the pressure fall and determining the pressure at which the radial pulse first becomes palpable. Because of the difficulty in determining exactly when the first beat is felt, pressures obtained by this palpation method are usually 2—5 mm Hg lower than those measured by the auscultatory method.

It is wise to form a habit of palpating the radial pulse while inflating the blood pressure cuff during measurement of the blood pressure by the auscultatory method. When the cuff pressure is lowered, the sounds of Korotkow sometimes disappear at pressures well above diastolic pressure, then reappear at lower presauscultatory gap. If the cuff is initially inflated until the radial pulse disappears, the examiner be sure that the cuff pressure is above systolic and falsely low pressure values will be avoided.

Normal Arterial Blood Pressure

The blood pressure in the brachial artery in young adults in the sitting or lying position at rest is approximately 120/70 mm Hg. Since the arterial pressure is the product of the cardiac output and the peripheral resistance, it is affected by conditions that affect either or both of these factors. Emotion, for example, increases the cardiac output, and it may be

difficult to obtain a truly resting blood pressure in an excited or tense individual. In general, increases in cardiac output increase the systolic pressure, whereas, increases in peripheral resistance increase the diastolic pressure. There is a good deal of controversy about where to draw the line between normal and elevated blood pressure levels (hypertension) particularly in older patients. However, the evidence seems incontrovertible that in apparently healthy humans both the systolic and the diastolic pressure rise with age. The systolic pressure increase is greater than the diastolic. An important cause of the rise in systolic pressure is decreased distensibility of the arteries as their walls become increasingly more rigid. At the same level of cardiac output, the systolic pressure is higher in older subjects than in young ones because there is less increase in the volume of the arterial system to accommodate the same amount of blood.

4. Principles of Venous Flow Dynamics

Blood flows through the blood vessels, including the veins, primarily because of the pumping action of the heart, although venous flow is aided by the heartbeat, the increase in the negative intrathoracic pressure during each inspiration, and contractions of skeletal muscles that compress the veins (**muscle pump**).

Venous Pressures & Flow

The pressure in the venules is 12-18 mm Hg. It falls steadily in the larger veins to about 5.5 mm Hg in the great veins outside the thorax. The pressure in the great veins at their entrance into the right atrium (central venous pressure) averages 4.6 mm Hg but fluctuates with respiration and heart action.

Peripheral venous pressure, like arterial pressure, is affected by gravity. It is increased by 0.77 mm Hg for each cm below the right atrium and decreased alike amount for each cm above the right atrium the pressure is measured.

When blood flows from the venules to the large veins, its average velocity increases as the total cross-sectional area of the vessels decreases. In the great veins, the velocity of blood is about one-fourth as great as that in the aorta, averaging about 10 cm/s.

Thoracic Pump

During inspiration, the intrapleural pressure falls from -2.5 mm Hg to -6 mm Hg. This negative pressure is transmitted to the great veins and, to a lesser extent, the atria, so that central venous pressure fluctuates from about 6 mm Hg during expiration to approximately 2 mm Hg during quiet inspiration. The drop in venous pressure during inspiration aids venous return. When the diaphragm descends during inspiration, intra-abdominal pressure rises, and this also squeezes blood toward the heart because backflow into the leg veins is prevented by the venous valves.

Effects of Heartbeat

The variations in atrial pressure are transmitted to the great veins to produce various wave of a venous pressure-pulse curve. Atrial pressure drops sharply during the ejection phase of ventricular systole because the atrioventricular valves are pulled downward, increasing the capacity of atria. This action sucks blood into the atria from great veins.

The sucking of the blood into the atria during systole contributes appreciably to the venous return, especially at rapid heart rates.

Close to the heart, venous flow becomes pulsatile. When the heart rate is slow, 2 periods of flow are detectable, one during ventricular systole, due to pulling down of the atrioventricular valves, and in early diastole, during the period of rapid ventricular filling.

Muscle Pump

In the limbs, the veins are surrounded by skeletal muscles, and contraction of these muscles during activity compresses the veins. Pulsations of nearby arteries may also compress veins. Since the venous valves prevent reverse flow, the blood moves toward the heart. During quiet standing, when the full effect of gravity is manifest, venous pressure at the ankle is 85—90mm Hg. Pooling of blood in the leg veins reduces venous return, with the result that cardiac output is reduced, sometimes to the point where fainting occurs. Rhythmic contractions of the leg muscles while the person is standing serve to lower the venous pressure in the legs to less than 30 mm Hg by propelling blood toward the heart. This heartward movement of the blood is decreased in patients with varicose veins, whose valves are incompetent, and such patients may have venous stasis and ankle edema. However, even when the valves are incompetent, muscle contractions will continue to produce a basic heartward movement of the blood because the resistance of the larger veins in the direction of the heart is less than the resistance of the small vessels away from the heart.

Measuring Central Venous Pressure

Central venous pressure can be measured directly by inserting a catheter into the thoracic great veins. Peripheral venous pressure correlates well with central venous pressure in most conditions. To measure peripheral venous pressure, a needle attached to a manometer containing sterile saline is inserted into an arm vein. The peripheral vein should be at the level of the right atrium (a point 10 cm or half the chest diameter from the back in the supine position). The values obtained in mm of saline can be converted into mm Hg by dividing by 13.6 (the density of mercury). The amount by which peripheral venous pressure exceeds central venous pressure increases with the distance from the heart along the veins. The mean pressure in the antecubital vein is normally 7.1 mm Hg, compared with a mean pressure of 4.6mm Hg in the central veins.

A fairly accurate estimate of central venous pressure can be made without any equipment by simply noting the height to which the external jugular veins are distended when the subject lies with the head slightly above the heart. The vertical distance between the right atrium and the place the vein collapses (the place where the pressure in it is zero) is the venous pressure in mm of blood.

Central venous pressure is decreased during negative pressure breathing and shock. It is increased by positive pressure breathing, straining, expansion of the blood volume, and heart failure. In advanced congestive heart failure or obstruction of the superior vena cava, the pressure in the antecubital vein may reach values of 20 mm Hg or more.