CHAPTER 5

The cardiovascular system

ANIMATIONS

The cardiovascular (cardio – heart, vascular – blood vessels) system is divided for descriptive purposes into two main parts:

- the *heart*, whose pumping action ensures constant circulation of the blood
- the *blood vessels,* which form a lengthy network through which the blood flows.

The *lymphatic system* is closely connected, both structurally and functionally, with the cardiovascular system and is discussed in Chapter 6.

The heart pumps blood into two anatomically separate systems of blood vessels (Fig. 5.1):

- the pulmonary circulation
- the systemic circulation.

The right side of the heart pumps blood to the lungs (the pulmonary circulation) where gas exchange occurs, i.e. the blood collects oxygen from the airsacs and excess carbon dioxide diffuses into the airsacs for exhalation. The left side of the heart pumps blood into the systemic circulation, which supplies the rest of the body. Here, tissue wastes are passed into the blood for excretion, and body cells extract nutrients and oxygen.

The cardiovascular system ensures a continuous flow of blood to all body cells, and its function is subject to continual physiological adjustments to maintain an adequate blood supply. Should the supply of oxygen and nutrients to body cells become inadequate, tissue damage occurs and cell death may follow.

Cardiovascular function normally declines with age, which is discussed on p. 117. Disease of the cardiovascular system is likely to have significant consequences, not only for the heart and blood vessels, but also for other body systems, which is discussed from page 118.

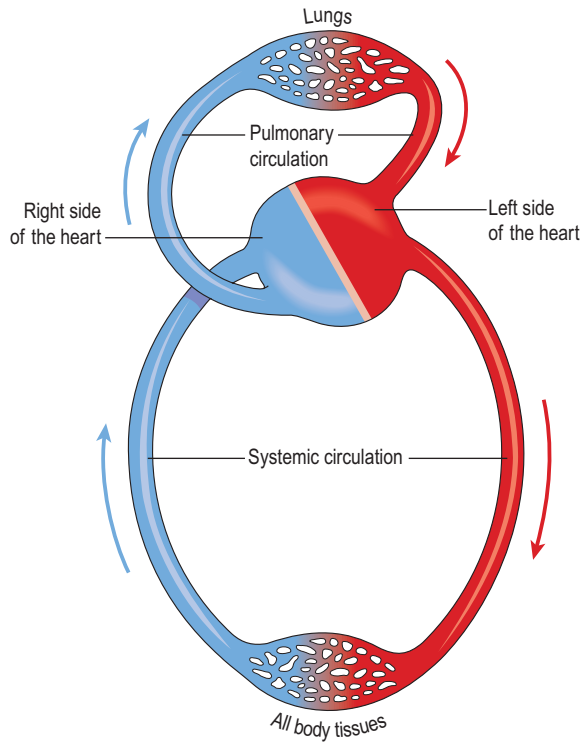


Figure 5.1 The relationship between the pulmonary and the systemic circulations.

Blood vessels

Learning outcomes

After studying this section, you should be able to:

- describe the structures and functions of arteries, veins and capillaries
- explain the relationship between the different types of blood vessel
- indicate the main factors controlling blood vessel diameter
- explain the mechanisms by which exchange of nutrients, gases and wastes occurs between the blood and the tissues.

Blood vessels vary in structure, size and function, and there are several types: arteries, arterioles, capillaries, venules and veins (Fig. 5.2). **5.1**

Arteries and arterioles

These blood vessels transport blood away from the heart. They vary considerably in size and their walls consist of three layers of tissue (Fig. 5.3):

- *tunica adventitia* or outer layer of fibrous tissue
- *tunica media* or middle layer of smooth muscle and elastic tissue
- *tunica intima* or inner lining of squamous epithelium called *endothelium*.

The amount of muscular and elastic tissue varies in the arteries depending upon their size and function. In the large arteries, including the aorta, sometimes called elastic arteries, the tunica media contains more elastic tissue and less smooth muscle. This allows the vessel wall to stretch, absorbing the pressure wave generated by the heart as it beats. These proportions gradually change as the arteries branch many times and become smaller until in the *arterioles* (the smallest arteries) the tunica media consists almost entirely of smooth muscle. This enables their diameter to be precisely controlled, which regulates the pressure within them. Systemic blood pressure is mainly determined by the resistance these tiny arteries offer to blood flow, and for this reason they are called *resistance vessels*.

Arteries have thicker walls than veins to withstand the high pressure of arterial blood.

Anastomoses and end-arteries

Anastomoses are arteries that form a link between main arteries supplying an area, e.g. the arterial supply to the palms of the hand (p. 107) and soles of the feet, the brain, the joints and, to a limited extent, the heart muscle. If one artery supplying the area is occluded, anastomotic arteries provide a *collateral circulation*. This is most likely to provide an adequate blood supply when the occlusion occurs gradually, giving the anastomotic arteries time to dilate.

An *end-artery* is an artery that is the sole source of blood to a tissue, e.g. the branches from the circulus arteriosus (circle of Willis) in the brain or the central artery to the retina of the eye. When an end-artery is occluded the tissues it supplies die because there is no alternative blood supply.

Capillaries and sinusoids

The smallest arterioles break up into a number of minute vessels called *capillaries*. Capillary walls consist of a single layer of endothelial cells sitting on a very thin basement membrane, through which water and other small molecules can pass. Blood cells and large molecules such as plasma proteins do not normally pass through capillary walls. The capillaries form a vast network of tiny vessels that link the smallest arterioles to the smallest venules. Their diameter is approximately that of an erythrocyte (7 μm). The capillary bed is the site of exchange of substances between the blood and the tissue fluid, which

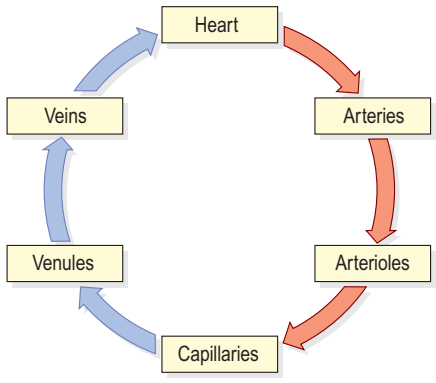


Figure 5.2 The relationship between the heart and the different types of blood vessel.

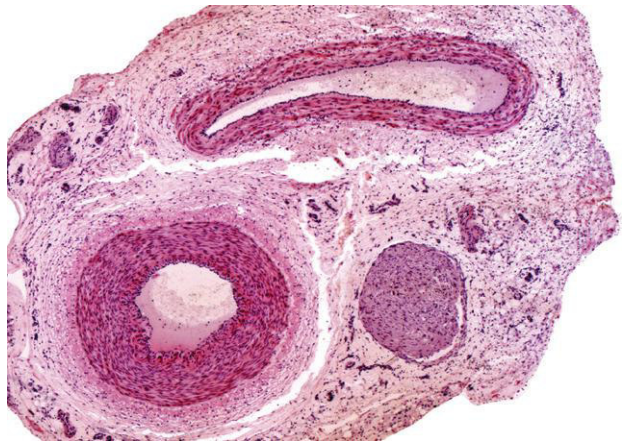


Figure 5.3 Light micrograph of an artery, a vein and an associated nerve.

bathes the body cells and, with the exception of those on the skin surface and in the cornea of the eye, every body cell lies close to a capillary.

Entry to capillary beds is guarded by rings of smooth muscle (*precapillary sphincters*) that direct blood flow. Hypoxia (low levels of oxygen in the tissues), or high levels of tissue wastes, indicating high levels of activity, dilate the sphincters and increase blood flow through the affected beds.

In certain places, including the liver (p. 309) and bone marrow, the capillaries are significantly wider and leakier than normal. These capillaries are called *sinusoids* and because their walls are incomplete and their lumen is much larger than usual, blood flows through them more slowly under less pressure and can come directly into contact with the cells outside the sinusoid wall. This allows much faster exchange of substances between the blood and the tissues, useful, for example, in the liver, which regulates the composition of blood arriving from the gastrointestinal tract.

Capillary refill time

When an area of skin is pressed firmly with a finger, it turns white (blanches) because the blood in the capillaries under the finger has been squeezed out. Normally it should take less than two seconds for the capillaries to refill once the finger is removed, and for the skin to turn pink again. Although the test may produce unreliable results, particularly in adults, its use in children can be useful and a prolonged capillary refill time suggests poor perfusion or dehydration.

Veins and venules

Veins return blood at low pressure to the heart. The walls of the veins are thinner than arteries but have the same three layers of tissue (Fig. 5.3). They are thinner because there is less muscle and elastic tissue in the tunica media, as veins carry blood at a lower pressure than arteries. When cut, the veins collapse while the thicker-walled arteries remain open. When an artery is cut blood spurts at high pressure while a slower, steady flow of blood escapes from a vein.

Some veins possess *valves*, which prevent backflow of blood, ensuring that it flows towards the heart (Fig. 5.4). They are formed by a fold of tunica intima and strengthened by connective tissue. The cusps are *semilunar* in shape with the concavity towards the heart. Valves are abundant in the veins of the limbs, especially the lower limbs where blood must travel a considerable distance against gravity when the individual is standing. They are absent in very small and very large veins in the thorax and abdomen. Valves are assisted in maintaining one-way flow by skeletal muscles surrounding the veins (p. 95).

The smallest veins are called *venules*.

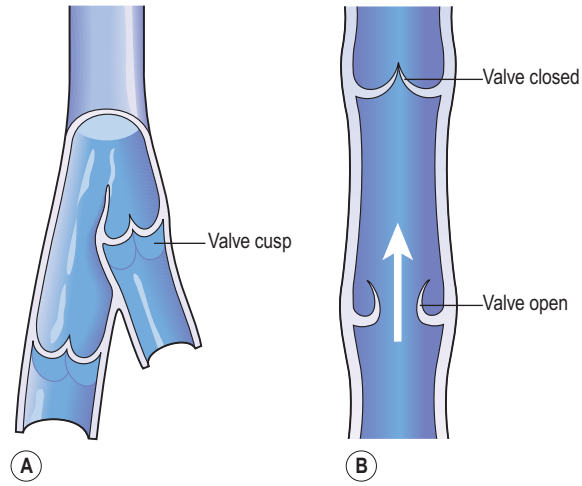


Figure 5.4 Interior of a vein. A. The valves and cusps. **B.** The direction of blood flow through a valve.

Veins are called *capacitance vessels* because they are distensible, and therefore have the capacity to hold a large proportion of the body's blood. At any one time, about two-thirds of the body's blood is in the venous system. This allows the vascular system to absorb (to an extent) sudden changes in blood volume, such as in haemorrhage; the veins can constrict, helping to prevent a sudden fall in blood pressure.

Blood supply

The outer layers of tissue of thick-walled blood vessels receive their blood supply via a network of blood vessels called the *vasa vasorum*. Thin-walled vessels and the endothelium of the others receive oxygen and nutrients by diffusion from the blood passing through them.

Control of blood vessel diameter

The smooth muscle in the tunica media of veins and arteries is supplied by nerves of the *autonomic nervous system*. These nerves arise from the *vasomotor centre* in the *medulla oblongata* and they change the diameter of blood vessels, controlling the volume of blood they contain.

The blood vessels most closely regulated by this nervous mechanism are the arterioles, since they contain proportionately more smooth muscle in their walls than any other blood vessel. The walls of large arteries such as the aorta contain mainly elastic tissue and so they tend to passively expand and recoil, depending on how much blood is passing through them. Veins also respond to nerve stimulation, although they have only a little smooth muscle in their tunica media.

Blood vessel diameter and blood flow

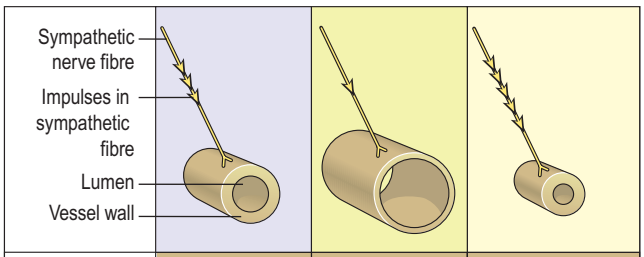
Resistance to flow of fluids along a tube is determined by three factors: the diameter of the tube; the length of the

tube; and the viscosity of the fluid. The most important factor determining how easily the blood flows through blood vessels is the first of these variables, that is, the diameter of the resistance vessels (the *peripheral resistance*). The length of the vessels and viscosity of blood also contribute to peripheral resistance, but in health these are constant and are therefore not significant determinants of changes in blood flow. Peripheral resistance is a major factor in blood pressure regulation, which is further discussed on page 96.

Blood vessel diameter is regulated by the smooth muscle of the tunica media, which is supplied by sympathetic nerves of the autonomic nervous system (p. 173). There is no parasympathetic nerve supply to most blood vessels and therefore the diameter of the vessel lumen and the tone of the smooth muscle are determined by the degree of sympathetic activity. Sympathetic activity generally constricts blood vessel smooth muscle and therefore narrows the vessel (*vasoconstriction*), increasing the pressure inside. A degree of resting sympathetic activity maintains a constant baseline tone in the vessel wall and prevents pressure falling too low (Fig. 5.5). Decreased nerve stimulation relaxes the smooth muscle, thinning the vessel wall and enlarging the lumen (*vasodilation*). This results in increased blood flow under less pressure.

Constant adjustment of blood vessel diameter helps to regulate peripheral resistance and systemic blood pressure.

Although most arterioles respond to sympathetic stimulation with vasoconstriction, the response is much less marked in some arteriolar beds, e.g. in skeletal muscle and the brain. This is important so that in a stress response, such as the flight or fight response (p. 176), when sympathetic activity is very high, these essential tissues receive the extra oxygen and nutrients they need.



	Baseline (resting)	Vasodilation	Vasoconstriction
Sympathetic stimulation	Moderate	Decreased	Increased
Smooth muscle	Moderate tone	Relaxed	Contracted
Thickness of vessel wall	Moderate	Thinner	Thicker
Diameter of lumen	Moderate	Increased	Decreased
Peripheral resistance in arterioles	Moderate	Decreased	Increased

Figure 5.5 The relationship between sympathetic stimulation and blood vessel diameter.

Local regulation of blood flow

Tissues' oxygen and nutrient requirements vary depending on their activities, so it is important that blood flow is regulated locally to ensure that blood flow matches tissue needs. The ability of an organ to control its own blood flow according to need is called *autoregulation*. Some organs, including the central nervous system, liver and kidneys receive proportionately higher blood flow as a matter of course. Other tissues, such as resting skeletal muscle, receive much less, but their blood supply can increase by as much as 20-fold during heavy exercise. Other examples include blood flow through the gastrointestinal tract increasing after a meal to allow for increased activity in the tract, and adjustments to blood flow through the skin in the control of body temperature (p. 367). Blood flow through individual organs is increased by vasodilation of the vessels supplying it, and decreased through vasoconstriction. The main mechanisms associated with this local control of blood flow include:

- release of metabolic waste products, e.g. CO_2 and lactic acid. Active tissues release more wastes than resting tissues, and increased levels of waste increase blood flow into the area
- tissue temperature: a rise in metabolic activity increases tissue temperature, which in turn causes vasodilation
- hypoxia, or lack of oxygen, stimulates vasodilation and a rise in blood flow through the affected tissue
- release of vasodilator chemicals. Inflamed and metabolically active tissues produce a number of vasodilators, which increase blood supply to the area. One important vasodilator is *nitric oxide*, which is very short lived, but which is important in opening up the larger arteries supplying an organ. Other agents include substances released in the inflammatory response, such as histamine and bradykinin (p. 378)
- action of vasoconstrictors. The sympathetic hormone adrenaline (epinephrine), released from the adrenal medulla, is a powerful vasoconstrictor. Others include angiotensin 2 (p. 343).

Capillary exchange

Exchange of gases

Internal respiration (Fig. 5.6) is the process by which gases are exchanged between capillary blood and local body cells.

Oxygen is carried from the lungs to the tissues in combination with haemoglobin (p. 66) as *oxyhaemoglobin*.

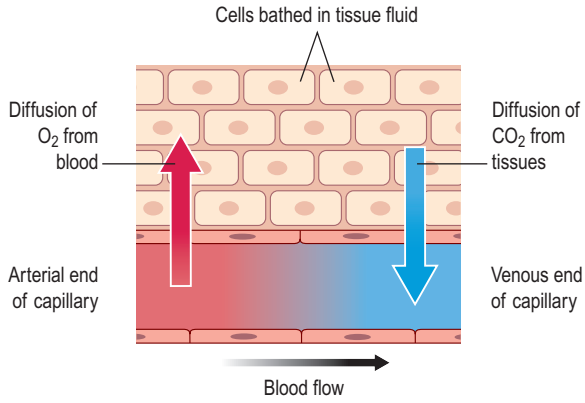


Figure 5.6 The exchange of gases in internal respiration.

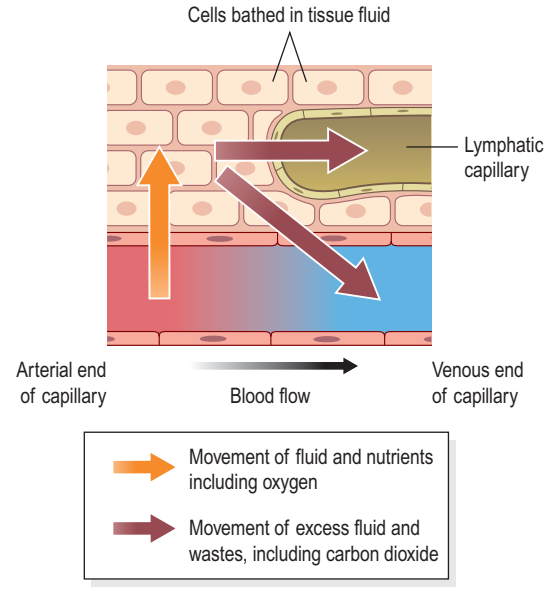


Figure 5.7 Diffusion of fluid, nutrients and waste products between capillaries and cells.

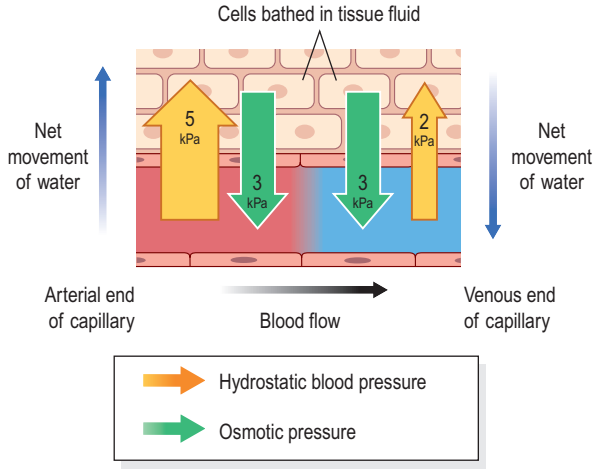


Figure 5.8 Effect of capillary pressures on water movement between capillaries and cells.

Exchange in the tissues takes place between blood at the arterial end of the capillaries and the tissue fluid and then between the tissue fluid and the cells. Oxygen diffuses down its concentration gradient, from the oxygen-rich arterial blood, into the tissues, where oxygen levels are lower because of constant tissue consumption.

Oxyhaemoglobin is an unstable compound and breaks up (dissociates) easily to liberate oxygen. Factors that increase dissociation are discussed on page 66.

Carbon dioxide is one of the waste products of cell metabolism and, towards the venous end of the capillary, it diffuses into the blood down the concentration gradient. Blood transports carbon dioxide to the lungs for excretion by three different mechanisms:

- dissolved in the water of the blood plasma – 7%
- in chemical combination with sodium in the form of sodium bicarbonate – 70%
- remainder in combination with haemoglobin – 23%.

Exchange of other substances

The nutrients, including glucose, amino acids, fatty acids, vitamins and mineral salts required by all body cells are transported round the body in the blood plasma. They diffuse through the semipermeable capillary walls into the tissues (Fig. 5.7). Water exchanges freely between the plasma and tissue fluid by osmosis. Diffusion and osmosis are described on p. 29.

Capillary fluid dynamics

The two main forces determining overall fluid movement across the capillary wall are the *hydrostatic pressure* (blood pressure), which tends to push fluid out of the bloodstream, and the *osmotic pressure* of the blood, which tends to pull it back in, and is due mainly to the presence of plasma proteins, especially albumin (Fig. 5.8).

At *the arterial end*, the hydrostatic pressure is about 5 kPa (35 mmHg), and the opposing osmotic pressure of the blood is only 3 kPa (25 mmHg). The overall force at the arterial end of the capillary therefore drives fluid out of the capillary and into the tissue spaces. This net loss of fluid from the bloodstream must be reclaimed in some way.

At *the venous end* of the capillary, the situation is reversed. Blood flow is slower than at the arterial end because the hydrostatic pressure drops along the capillary to only 2 kPa (15 mmHg). The osmotic pressure remains unchanged at 3 kPa (25 mmHg) and, because this now exceeds hydrostatic pressure, fluid moves back into the capillary.

This transfer of substances, including water, to the tissue spaces is a dynamic process. As blood flows slowly through the large network of capillaries from the arterial to the venous end, there is constant change. Not all the water and cell waste products return to the blood capillaries. Of

the 24 litres or so of fluid that moves out of the blood across capillary walls every day, only about 21 litres returns to the bloodstream at the venous end of the capillary bed. The excess is drained away from the tissue spaces in the minute lymph capillaries which originate as blind-end tubes with walls similar to, but more permeable than, those of the blood capillaries (Fig. 5.7). Extra tissue fluid and some cell waste materials enter the lymph capillaries and are eventually returned to the bloodstream (Ch. 6).

Heart

Learning outcomes

After studying this section, you should be able to:

- describe the structure of the heart and its position within the thorax
- trace the circulation of the blood through the heart and the blood vessels of the body
- outline the conducting system of the heart
- relate the electrical activity of the cardiac conduction system to the cardiac cycle
- describe the main factors determining heart rate and cardiac output.

The heart is a roughly cone-shaped hollow muscular organ. It is about 10 cm long and is about the size of the owner's fist. It weighs about 225 g in women and is heavier in men (about 310 g).

Position 5.2

The heart lies in the thoracic cavity (Fig. 5.9) in the mediastinum (the space between the lungs). It lies obliquely, a little more to the left than the right, and presents a base above, and an *apex* below. The apex is about 9 cm to the left of the midline at the level of the 5th intercostal space, i.e. a little below the nipple and slightly nearer the midline. The base extends to the level of the 2nd rib.

Organs associated with the heart (Fig. 5.10)

Inferiorly – the apex rests on the central tendon of the diaphragm

Superiorly – the great blood vessels, i.e. the aorta, superior vena cava, pulmonary artery and pulmonary veins

Posteriorly – the oesophagus, trachea, left and right bronchus, descending aorta, inferior vena cava and thoracic vertebrae

Laterally – the lungs – the left lung overlaps the left side of the heart

Anteriorly – the sternum, ribs and intercostal muscles.

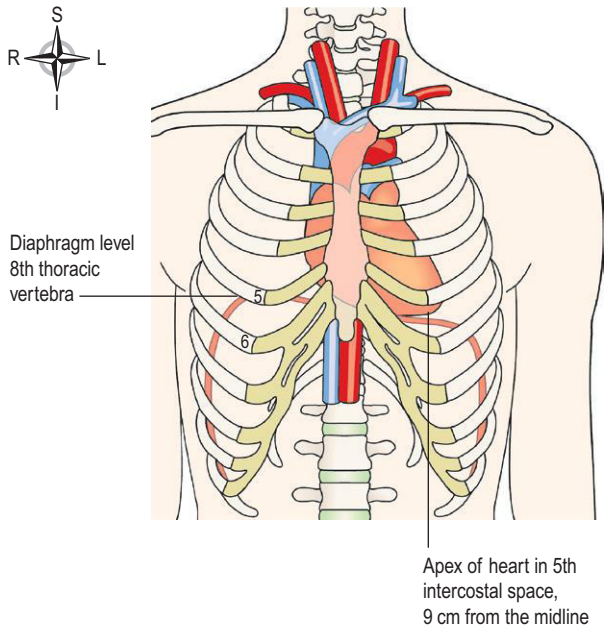


Figure 5.9 Position of the heart in the thorax.

Structure

The heart wall

The heart wall is composed of three layers of tissue (Fig. 5.11A): pericardium, myocardium and endocardium.

Pericardium

The pericardium is the outermost layer and is made up of two sacs. The outer sac (the fibrous pericardium) consists of fibrous tissue and the inner (the serous pericardium) of a continuous double layer of serous membrane.

The fibrous pericardium is continuous with the tunica adventitia of the great blood vessels above and is adherent to the diaphragm below. Its inelastic, fibrous nature prevents overdistension of the heart.

The outer layer of the serous pericardium, the *parietal pericardium*, lines the fibrous pericardium. The inner layer, the *visceral pericardium*, which is continuous with the parietal pericardium, is adherent to the heart muscle. A similar arrangement of a double membrane forming a closed space is seen also with the pleura, the membrane enclosing the lungs (see Fig. 10.15, p. 250).

The serous membrane consists of flattened epithelial cells. It secretes serous fluid, called *pericardial fluid*, into the space between the visceral and parietal layers, which allows smooth movement between them when the heart beats. The space between the parietal and visceral pericardium is only a *potential space*. In health the two layers lie closely together, with only the thin film of pericardial fluid between them.

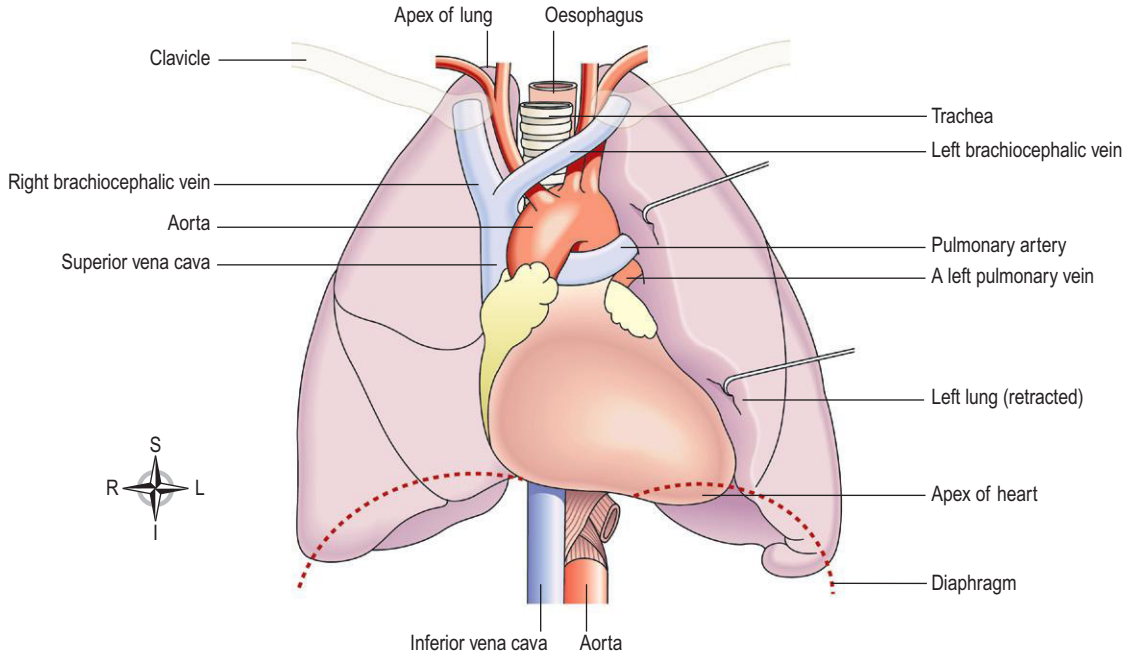


Figure 5.10 Organs associated with the heart.

Myocardium

The myocardium is composed of specialised cardiac muscle found only in the heart (Fig. 5.11B and C). It is striated, like skeletal muscle, but is not under voluntary control. Each fibre (cell) has a nucleus and one or more branches. The ends of the cells and their branches are in very close contact with the ends and branches of adjacent cells. Microscopically these 'joints', or *intercalated discs*, are thicker, darker lines than the striations. This arrangement gives cardiac muscle the appearance of being a sheet of muscle rather than a very large number of individual cells. Because of the end-to-end continuity of the fibres, each one does not need to have a separate nerve supply. When an impulse is initiated it spreads from cell to cell via the branches and intercalated discs over the whole 'sheet' of muscle, causing contraction. The 'sheet' arrangement of the myocardium enables the atria and ventricles to contract in a coordinated and efficient manner.

Running through the myocardium is also the network of specialised conducting fibres responsible for transmitting the heart's electrical signals. The myocardium is thickest at the apex and thins out towards the base (Fig. 5.12). This reflects the amount of work each chamber contributes to the pumping of blood. It is thickest in the left ventricle, which has the greatest workload.

Specialised muscle cells in the walls of the atria secrete atrial natriuretic peptide (ANP, p. 228).

Fibrous tissue in the heart. The myocardium is supported by a network of fine fibres that run through all the heart muscle. This is called the *fibrous skeleton* of the heart. In addition, the atria and the ventricles are separated by a ring of fibrous tissue, which does not conduct electrical impulses. Consequently, when a wave of electrical activity passes over the atrial muscle, it can only spread to the ventricles through the conducting system that bridges the fibrous ring from atria to ventricles (p. 90).

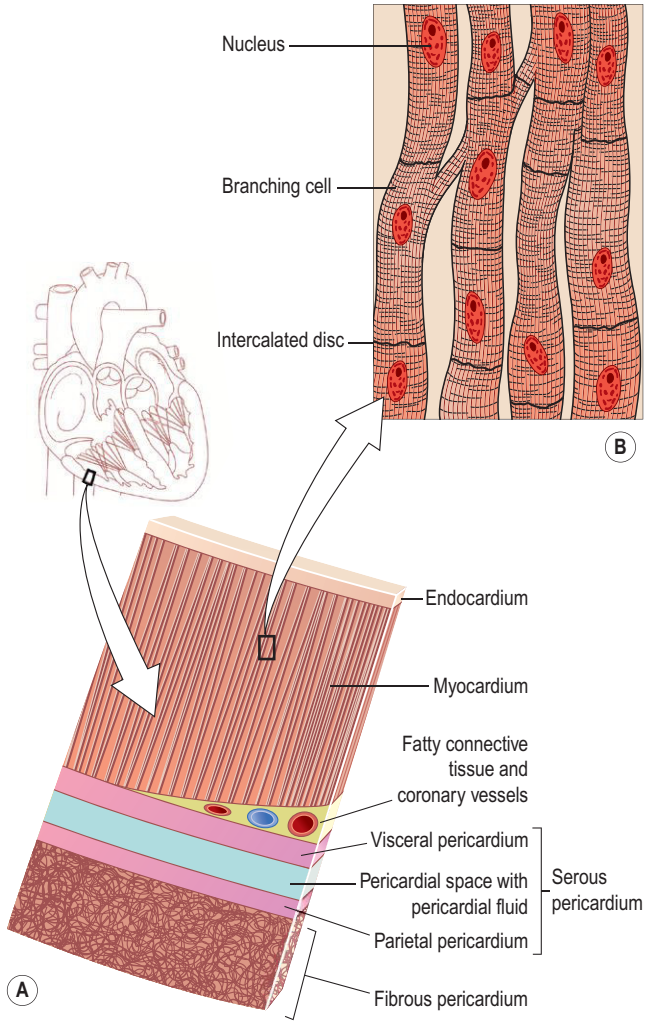
Endocardium

This lines the chambers and valves of the heart. It is a thin, smooth membrane to ensure smooth flow of blood through the heart. It consists of flattened epithelial cells, and it is continuous with the endothelium lining the blood vessels.

Interior of the heart 5.3, 5.4

The heart is divided into a right and left side by the *septum* (Fig. 5.12), a partition consisting of myocardium covered by endocardium. After birth, blood cannot cross the septum from one side to the other. Each side is divided by an *atrioventricular valve* into the upper atrium and the ventricle below. The atrioventricular valves are formed by double folds of endocardium strengthened by a little fibrous tissue. The right atrioventricular valve (tricuspid valve) has three flaps or cusps and the left atrioventricular valve (mitral valve, Fig. 5.13) has two cusps. Flow of blood in the heart is one way; blood enters the heart via the atria and passes into the ventricles below.

The valves between the atria and ventricles open and close passively according to changes in pressure in the chambers (Fig. 5.13A and B). They open when the pressure in the atria is greater than that in the ventricles. During *ventricular systole* (contraction) the pressure in the ventricles rises above that in the atria and the valves snap shut, preventing backward flow of blood. The valves are



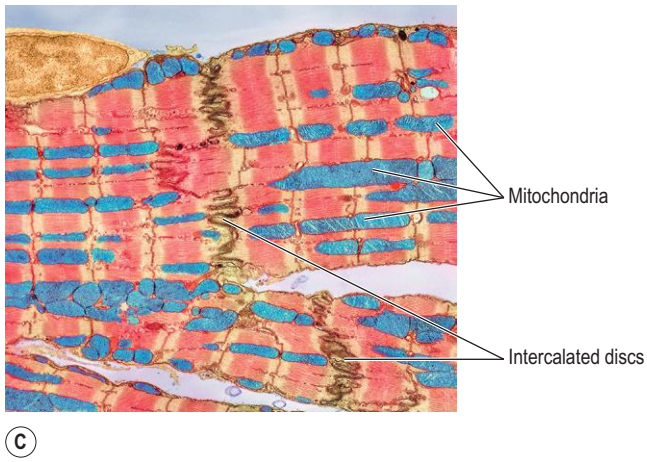


Figure 5.11 Tissues of the heart wall. A. Layers of the heart wall: endocardium, myocardium and pericardium. **B.** Cardiac muscle tissue. **C.** Coloured electron micrograph of cardiac muscle tissue.

prevented from opening upwards into the atria by tendinous cords, called *chordae tendineae* (Fig. 5.13C), which extend from the inferior surface of the cusps to little projections of myocardium into the ventricles, covered with endothelium, called *papillary muscles* (Fig. 5.13).

Flow of blood through the heart

(Fig. 5.14) 5.5

The two largest veins of the body, the *superior* and *inferior venae cavae*, empty their contents into the right atrium. This blood passes via the right atrioventricular valve into the right ventricle, and from there is pumped into the *pulmonary artery* or *trunk* (the only artery in the body which carries deoxygenated blood). The opening of the pulmonary artery is guarded by the *pulmonary valve*, formed by three *semilunar cusps*. This valve prevents the backflow of blood into the right ventricle when the ventricular muscle relaxes. After leaving the heart the pulmonary artery divides into *left* and *right pulmonary arteries*, which carry the venous blood to the lungs where exchange of gases takes place: carbon dioxide is excreted and oxygen is absorbed.

Two *pulmonary veins* from each lung carry *oxygenated blood* back to the *left atrium*. Blood then passes through the left atrioventricular valve into the left ventricle, and from there it is pumped into the aorta, the first artery of the general circulation. The opening of the aorta is guarded by the *aortic valve*, formed by three *semilunar cusps* (Fig. 5.15).

From this sequence of events it can be seen that the blood passes from the right to the left side of the heart via the lungs, or pulmonary circulation (Fig. 5.16). However, it should be noted that both atria contract at the same time and this is followed by the simultaneous contraction of both ventricles.

The muscle layer of the walls of the atria is thinner than that of the ventricles (Fig. 5.12). This is consistent with the amount of work they do. The atria, usually assisted by gravity, pump the blood only through the atrioventricular valves into the ventricles, whereas the more powerful ventricles pump the blood to the lungs and round the whole body.

The pulmonary trunk leaves the heart from the upper part of the right ventricle, and the aorta leaves from the upper part of the left ventricle.

Blood supply to the heart (the coronary circulation) 5.6

Arterial supply (Fig. 5.17)**.** The heart is supplied with arterial blood by the *right* and *left coronary arteries*, which branch from the aorta immediately distal to the aortic valve (Figs 5.15 and 5.17). The coronary arteries receive about 5% of the blood pumped from the heart, although the heart comprises a small proportion of body weight.

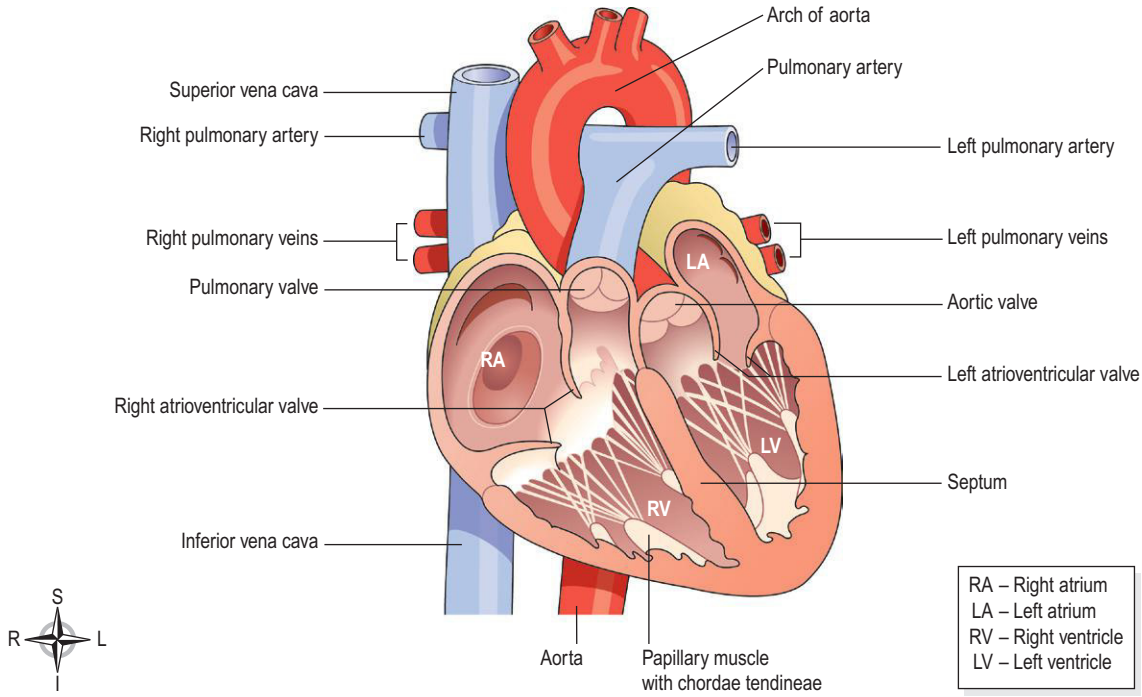


Figure 5.12 Interior of the heart.

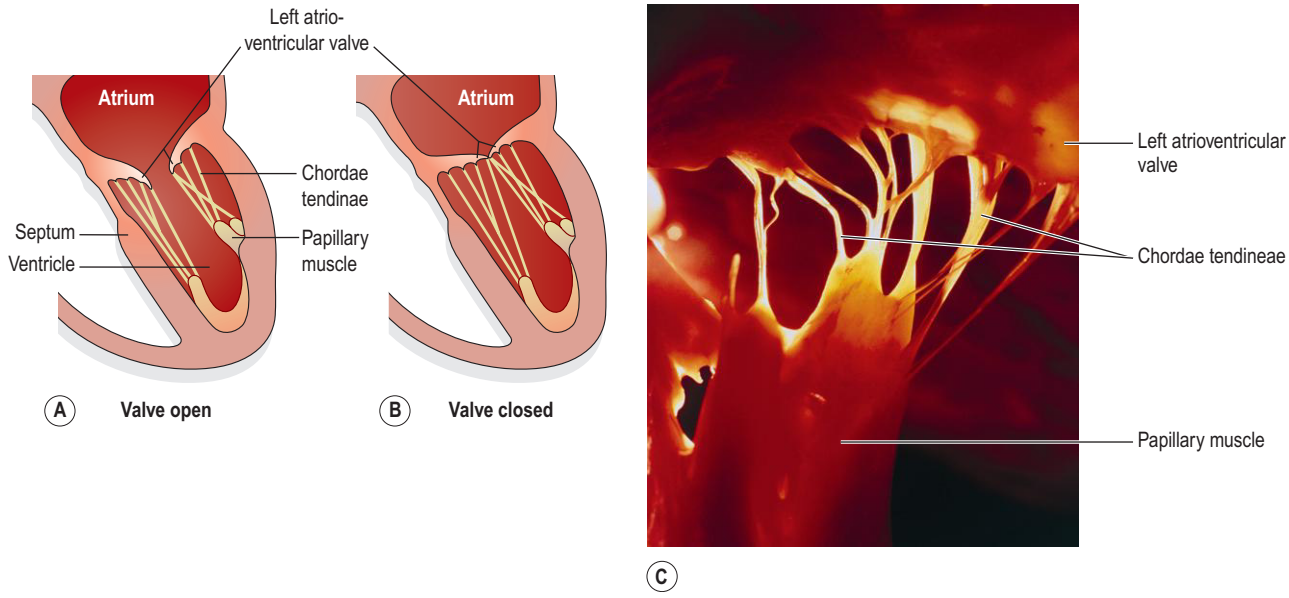


Figure 5.13 The left atrioventricular (mitral) valve. A. Valve open. **B.** Valve closed. **C.** Photograph of the chordae tendinae.

This large blood supply, of which a large proportion goes to the left ventricle, highlights the importance of the heart to body function. The coronary arteries traverse the heart, eventually forming a vast network of capillaries.

Venous drainage. Most of the venous blood is collected into a number of *cardiac veins* that join to form the *coronary sinus*, which opens into the right atrium. The remainder passes directly into the heart chambers through little venous channels.

Conducting system of the heart

(Fig. 5.18) **5.7**

The heart possesses the property of *autorhythmicity*, which means it generates its own electrical impulses and beats independently of nervous or hormonal control, i.e. it is not reliant on external mechanisms to initiate each heartbeat. However, it is supplied with both sympathetic and parasympathetic nerve fibres, which increase and

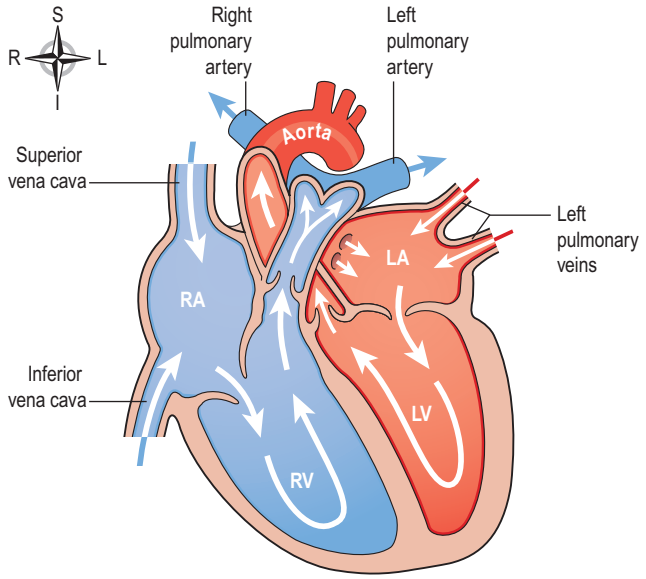


Figure 5.14 Direction of blood flow through the heart.

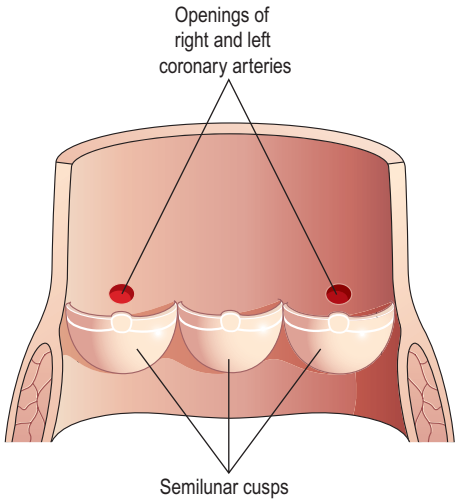


Figure 5.15 The aorta cut open to show the semilunar cusps of the aortic valve.

decrease respectively the intrinsic heart rate. In addition, the heart responds to a number of circulating hormones, including adrenaline (epinephrine) and thyroxine.

Small groups of specialised neuromuscular cells in the myocardium initiate and conduct impulses, causing coordinated and synchronised contraction of the heart muscle.

Sinoatrial node (SA node)

This small mass of specialised cells lies in the wall of the right atrium near the opening of the superior vena cava.

The sinoatrial cells generate these regular impulses because they are electrically unstable. This instability leads them to discharge (*depolarise*) regularly, usually between 60 and 80 times a minute. This depolarisation is

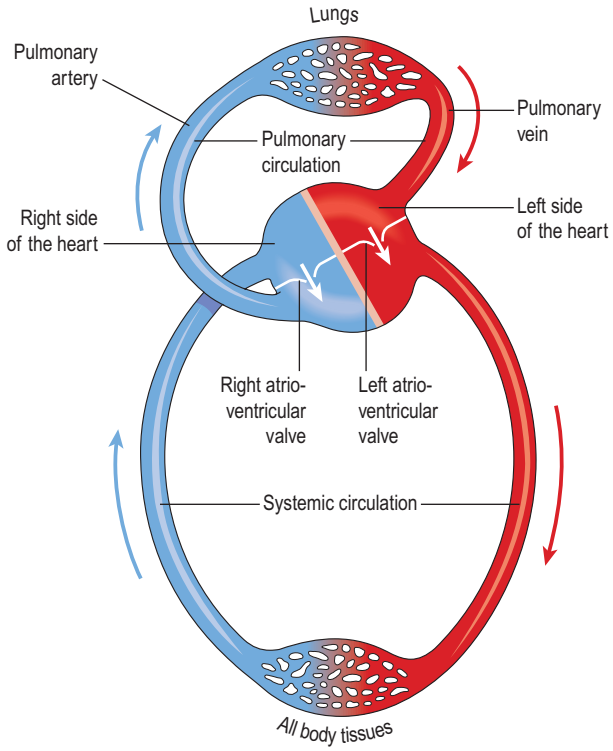


Figure 5.16 Circulation of blood through the heart and the pulmonary and systemic circulations.

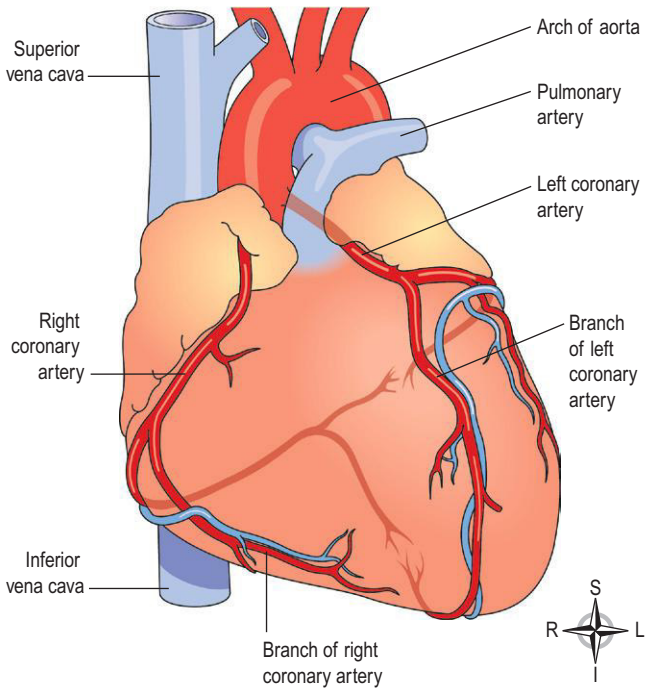


Figure 5.17 The coronary arteries.

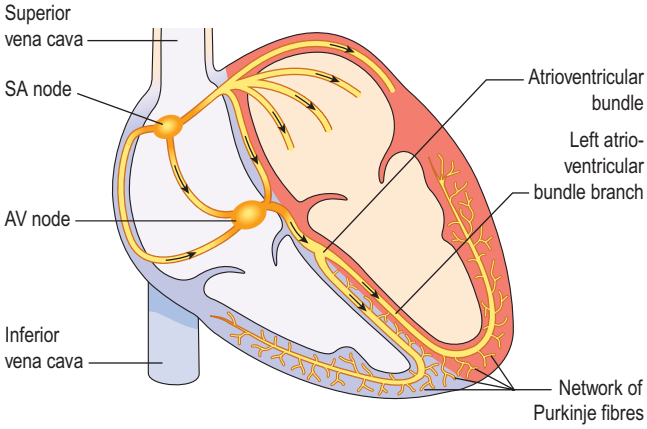


Figure 5.18 The conducting system of the heart.

followed by recovery (*repolarisation*), but almost immediately their instability leads them to discharge again, setting the heart rate. Because the SA node discharges faster than any other part of the heart, it normally sets the heart rate and is called the *pacemaker* of the heart. Firing of the SA node triggers atrial contraction.

Atrioventricular node (AV node)

This small mass of neuromuscular tissue is situated in the wall of the atrial septum near the atrioventricular valves. Normally, the AV node merely transmits the electrical signals from the atria into the ventricles. There is a delay here; the electrical signal takes 0.1 of a second to pass through into the ventricles. This allows the atria to finish contracting before the ventricles start.

The AV node also has a secondary pacemaker function and takes over this role if there is a problem with the SA node itself, or with the transmission of impulses from the atria. Its intrinsic firing rate, however, is slower than that set by the SA node (40–60 beats per minute).

Atrioventricular bundle (AV bundle or bundle of His)

This mass of specialised fibres originates from the AV node. The AV bundle crosses the fibrous ring that separates atria and ventricles then, at the upper end of the ventricular septum, it divides into *right* and *left bundle branches*. Within the ventricular myocardium the branches break up into fine fibres, called the *Purkinje fibres*. The AV bundle, bundle branches and Purkinje fibres transmit electrical impulses from the AV node to the apex of the myocardium where the wave of ventricular contraction begins, then sweeps upwards and outwards, pumping blood into the pulmonary artery and the aorta.

Nerve supply to the heart

As mentioned earlier, the heart is influenced by autonomic (sympathetic and parasympathetic) nerves originating in the *cardiovascular centre* in the *medulla oblongata*.

The *vagus nerve* (parasympathetic) supplies mainly the SA and AV nodes and atrial muscle. Vagal stimulation reduces the rate at which impulses are produced, decreasing the rate and force of the heartbeat.

Sympathetic nerves supply the SA and AV nodes and the myocardium of atria and ventricles, and stimulation increases the rate and force of the heartbeat.

Box 5.1 The main factors affecting heart rate

- Gender
- Autonomic activity
- Age
- Circulating hormones
- Activity and exercise
- Temperature
- The baroreceptor reflex
- Emotional states

Factors affecting heart rate

The most important ones are listed in Box 5.1, and explained in more detail on page 95.

The cardiac cycle

At rest, the healthy adult heart is likely to beat at a rate of 60–80 beats per minute (b.p.m.). During each heartbeat, or *cardiac cycle* (Fig. 5.19), the heart contracts (systole) and then relaxes (diastole).

Stages of the cardiac cycle

Taking 74 b.p.m. as an example, each cycle lasts about 0.8 of a second and consists of:

- *atrial systole* – contraction of the atria
- *ventricular systole* – contraction of the ventricles
- *complete cardiac diastole* – relaxation of the atria and ventricles.

It does not matter at which stage of the cardiac cycle a description starts. For convenience the period when the atria are filling has been chosen.

The superior vena cava and the inferior vena cava transport deoxygenated blood into the right atrium *at the same time* as the four pulmonary veins bring oxygenated blood into the left atrium. The atrioventricular valves are open and blood flows passively through to the ventricles. The SA node triggers a wave of contraction that spreads over the myocardium of both atria, emptying the atria and completing ventricular filling (atrial systole 0.1 s; Fig. 5.19A). When the electrical impulse reaches the AV node it is slowed down, delaying atrioventricular transmission. This delay means that the mechanical result of atrial stimulation, atrial contraction, lags behind the electrical activity by a fraction of a second. This allows the atria to finish emptying into the ventricles before the

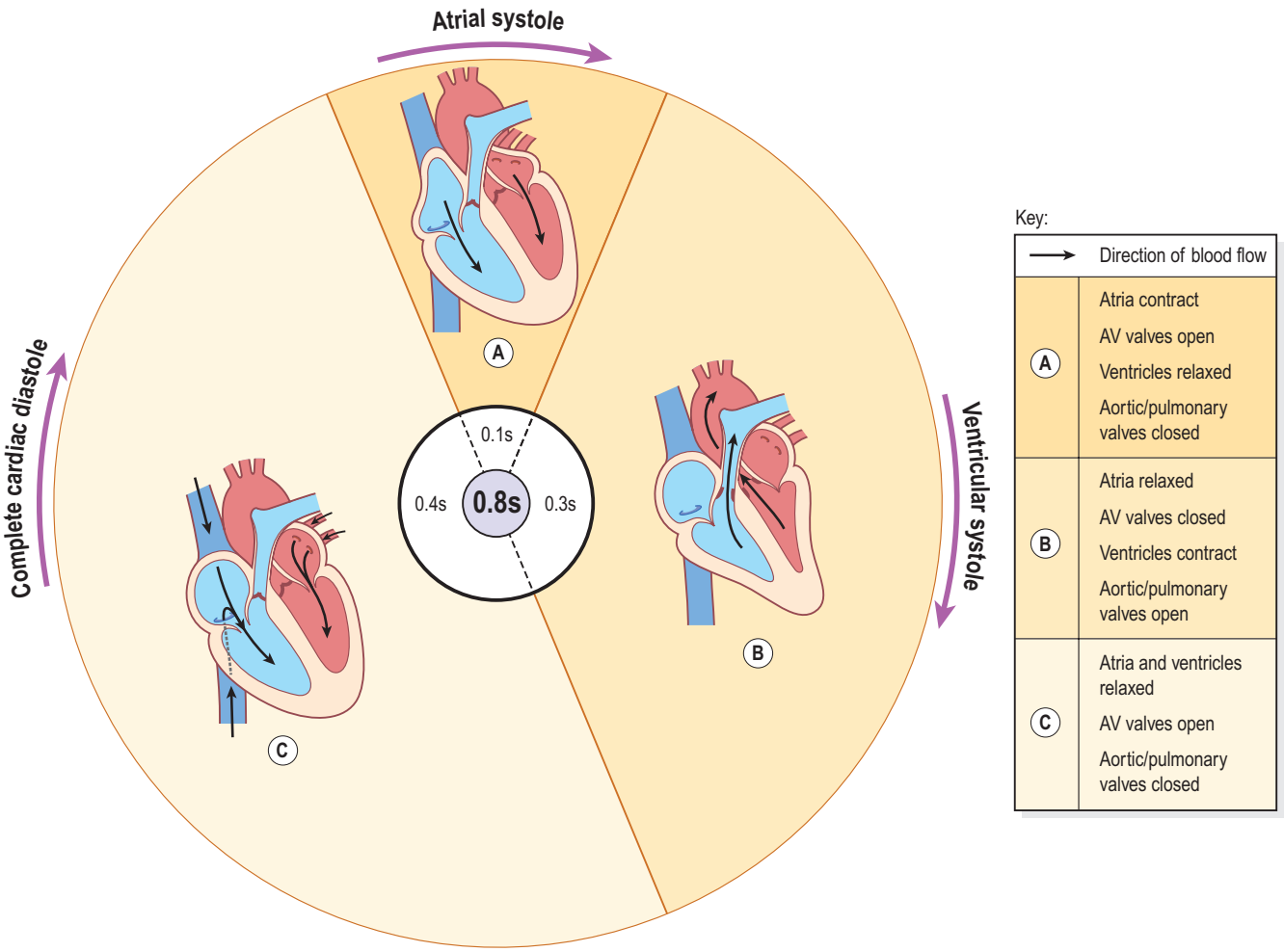


Figure 5.19 The cardiac cycle.

ventricles begin to contract. After this brief delay, the AV node triggers its own electrical impulse, which quickly spreads to the ventricular muscle via the AV bundle, the bundle branches and Purkinje fibres. This results in a wave of contraction which sweeps upwards from the apex of the heart and across the walls of both ventricles pumping the blood into the pulmonary artery and the aorta (ventricular systole 0.3 s; Fig. 5.19B). The high pressure generated during ventricular contraction forces the atrioventricular valves to close, preventing backflow of blood into the atria.

After contraction of the ventricles there is *complete cardiac diastole*, a period of *0.4 seconds*, when atria and ventricles are relaxed. During this time the myocardium recovers ready for the next heartbeat, and the atria refill ready for the next cycle (Fig. 5.19C).

The valves of the heart and of the great vessels open and close according to the pressure within the chambers of the heart. The AV valves are open while the ventricular muscle is relaxed during atrial filling and systole. When the ventricles contract there is a rapid increase in the pressure in these chambers, and when it rises above atrial pressure the atrioventricular valves close. When the ventricular pressure rises above that in the pulmonary artery and in the aorta, the pulmonary and aortic valves open and blood flows into these vessels. When the ventricles relax and the pressure within them falls, the reverse process occurs. First the pulmonary and aortic valves close, then the atrioventricular valves open and the cycle begins again. This sequence of opening and closing valves ensures that the blood flows in only one direction.

Heart sounds

The individual is not usually conscious of their heartbeat, but if the ear, or the diaphragm of a stethoscope, is placed on the chest wall a little below the left nipple and slightly nearer the midline the heartbeat can be heard (Fig. 5.9).

There are four heart sounds, each corresponding to a particular event in the cardiac cycle. The first two are most easily distinguished, and sound through the stethoscope like 'lub dup'. The first sound, '*lub*', is fairly loud and is due to the closure of the atrioventricular valves.

This corresponds with the start of ventricular systole. The second sound, '*dup*', is softer and is due to the closure of the aortic and pulmonary valves. This corresponds with ventricular diastole.

Electrical changes in the heart 5.8

The body tissues and fluids conduct electricity well, so the electrical activity in the heart can be recorded on the skin surface using electrodes positioned on the limbs and/or the chest. This recording, called an electrocardiogram (ECG) shows the spread of the electrical signal generated by the SA node as it travels through the atria, the AV node and the ventricles. The normal ECG tracing shows five waves which, by convention, have been named P, Q, R, S and T (Fig. 5.20).

The P wave arises when the impulse from the SA node sweeps over the atria (atrial depolarisation).

The QRS complex represents the very rapid spread of the impulse from the AV node through the AV bundle and the Purkinje fibres and the electrical activity of the ventricular muscle (ventricular depolarisation). Note the delay between the completion of the P wave and the onset of the QRS complex. This represents the conduction of the impulse through the AV node (p. 92), which is much slower than conduction elsewhere in the heart, and allows atrial contraction to finish completely before ventricular contraction starts.

The T wave represents the relaxation of the ventricular muscle (ventricular repolarisation). Atrial repolarisation occurs during ventricular contraction, and so is not seen because of the larger QRS complex.

The ECG described above originates from the SA node and is called *sinus rhythm*. The rate of sinus rhythm is 60–100 b.p.m. A faster heart rate is called *tachycardia* and a slower heart rate, *bradycardia*.

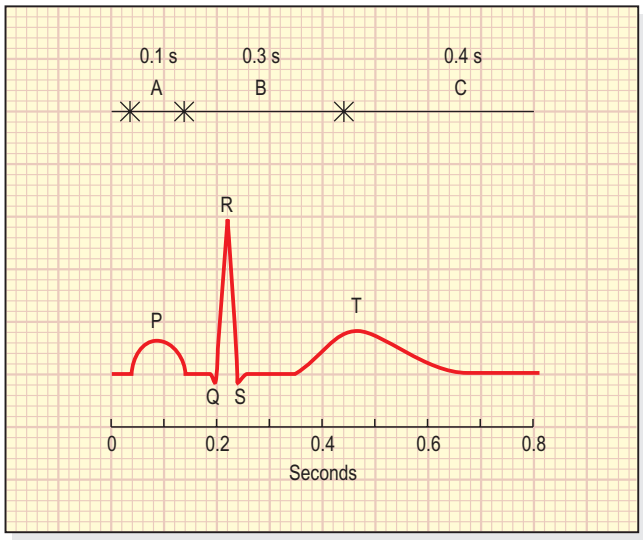


Figure 5.20 Electrocardiogram of one cardiac cycle. A, B and **C** correspond to the phases of the cardiac cycle shown in Figure 5.19.

By examining the pattern of waves and the time interval between cycles and parts of cycles, information about the state of the myocardium and the cardiac conduction system is obtained.

Cardiac output

The cardiac output is the amount of blood ejected from each ventricle every minute. The amount expelled by each contraction of each ventricle is the *stroke volume*. Cardiac output is expressed in litres per minute (L/min) and is calculated by multiplying the stroke volume by the heart rate (measured in beats per minute):

Cardiac output = Stroke volume × Heart rate.

In a healthy adult at rest, the stroke volume is approximately 70 mL and if the heart rate is 72 per minute, the cardiac output is 5 L/minute. This can be greatly increased to meet the demands of exercise to around 25 L/minute, and in athletes up to 35 L/minute. This increase during exercise is called the *cardiac reserve*.

When increased blood supply is needed to meet increased tissue requirements of oxygen and nutrients, heart rate and/or stroke volume can be increased (see Box 5.2).

Stroke volume

The stroke volume is determined by the volume of blood in the ventricles immediately before they contract, i.e. the *ventricular end-diastolic volume* (VEDV), sometimes called *preload*. In turn, preload depends on the amount of blood returning to the heart through the superior and inferior venae cavae (the *venous return*). Increased preload leads to stronger myocardial contraction, and more blood is expelled. In turn the stroke volume and cardiac output rise. In this way, the heart, within physiological limits, always pumps out all the blood that it receives, allowing it to adjust cardiac output to match body needs. This capacity to increase the stroke volume with increasing preload is finite, and when the limit is reached, i.e. venous return to the heart exceeds cardiac

Box 5.2 Summary of factors affecting cardiac output

Cardiac output = Stroke volume × Heart rate

Factors affecting stroke volume:

- VEDV (ventricular end-diastolic volume – preload)
- Venous return
  - position of the body
  - skeletal muscle pump
  - respiratory pump
- Strength of myocardial contraction
- Blood volume

output (i.e. more blood is arriving in the atria than the ventricles can pump out), cardiac output decreases and the heart begins to fail (p. 126). Other factors that increase the force and rate of myocardial contraction include increased sympathetic nerve activity and circulating hormones, e.g. adrenaline (epinephrine), noradrenaline (norepinephrine) and thyroxine.

Arterial blood pressure. This affects the stroke volume as it creates resistance to blood being pumped from the ventricles into the great arteries. This resistance (sometimes called *afterload*) is determined by the distensibility, or *elasticity*, of the large arteries and the peripheral resistance of arterioles (p. 85). Increasing afterload increases the workload of the ventricles, because it increases the pressure against which they have to pump. This may actually reduce stroke volume if systemic blood pressure becomes significantly higher than normal.

Blood volume. This is normally kept constant by the kidneys. Should blood volume fall, e.g. through sudden haemorrhage, this can cause stroke volume, cardiac output and venous return to fall. However, the body's compensatory mechanisms (p. 96) will tend to return these values towards normal, unless the blood loss is too sudden or severe for compensation (see Shock, p. 118).

Venous return

Venous return is the major determinant of cardiac output and, normally, the heart pumps out all blood returned to it. The force of contraction of the left ventricle ejecting blood into the aorta is not sufficient to push the blood through the arterial and venous circulation and back to the heart. Other factors are involved.

The position of the body. Gravity assists venous return from the head and neck when standing or sitting and offers less resistance to venous return from the lower parts of the body when lying flat.

Muscular contraction. Backflow of blood in veins of the limbs, especially when standing, is prevented by valves (Fig. 5.4). The contraction of skeletal muscles surrounding the deep veins compresses them, pushing blood towards the heart (Fig. 5.21). In the lower limbs, this is called the *skeletal muscle pump*.

The respiratory pump. During inspiration, the expansion of the chest creates a negative pressure within the thorax, assisting flow of blood towards the heart. In addition, when the diaphragm descends during inspiration, the increased intra-abdominal pressure pushes blood towards the heart.

Heart rate

The heart rate is a major determinant of cardiac output. If heart rate rises, cardiac output increases, and if it falls, cardiac output falls too. The main factors determining heart rate are outlined below.

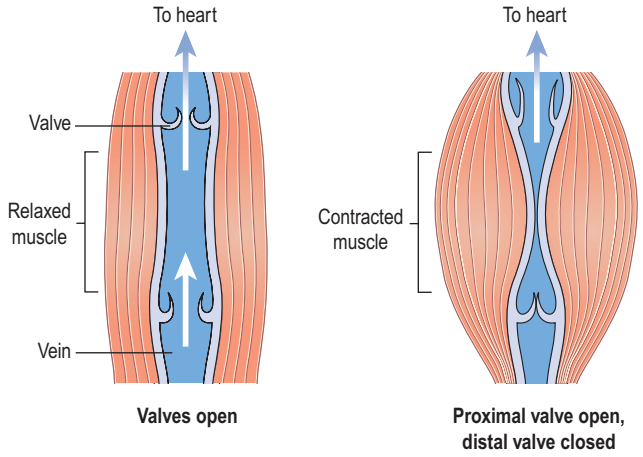


Figure 5.21 The skeletal muscle pump.

Autonomic nervous system. The intrinsic rate at which the heart beats is a balance between sympathetic and parasympathetic activity and this is the most important factor in determining heart rate.

Circulating chemicals. The hormones adrenaline (epinephrine) and noradrenaline (norepinephrine), secreted by the adrenal medulla, have the same effect as sympathetic stimulation, i.e. they increase the heart rate. Other hormones, including thyroxine, increase heart rate. Hypoxia and elevated carbon dioxide levels stimulate heart rate. Electrolyte imbalances may affect it, e.g. hyperkalaemia depresses cardiac function and leads to bradycardia (slow heart rate). Some drugs, such as β-receptor antagonists (e.g. atenolol) used in hypertension, can also cause bradycardia.

Position. When upright, the heart rate is usually faster than when lying down.

Exercise. Active muscles need more blood than resting muscles and this is achieved by an increased heart rate and local vasodilation.

Emotional states. During excitement, fear or anxiety the heart rate is increased. Other effects mediated by the sympathetic nervous system may be present (see Fig. 7.43, p. 174).

Gender. The heart rate is faster in women than men.

Age. In babies and small children the heart rate is more rapid than in older children and adults.

Temperature. The heart rate rises and falls with body temperature.

Baroreceptor reflex. See page 97.

A summary of the factors that alter CO is shown in Box 5.2.

Blood pressure

Learning outcomes

After studying this section, you should be able to:

- define the term blood pressure
- describe the factors that influence blood pressure
- explain the two main mechanisms that control blood pressure.

Blood pressure is the force or pressure that the blood exerts on the walls of blood vessels. Systemic arterial blood pressure maintains the essential flow of blood into and out of the organs of the body. Keeping blood pressure within normal limits is very important. If it becomes too high, blood vessels can be damaged, causing clots or bleeding from sites of blood vessel rupture. If it falls too low, then blood flow through tissue beds may be inadequate. This is particularly dangerous for essential organs such as the heart, brain or kidneys.

The systemic arterial blood pressure, usually called simply arterial blood pressure, is the result of the discharge of blood from the left ventricle into the already full aorta.

Blood pressure varies according to the time of day, the posture, gender and age of the individual. Blood pressure falls at rest and during sleep. It increases with age and is usually higher in women than in men.

Systolic and diastolic pressures. When the left ventricle contracts and pushes blood into the aorta, the pressure produced within the arterial system is called the *systolic blood pressure*. In adults it is about 120 mmHg or 16 kPa.

In complete cardiac diastole when the heart is resting following the ejection of blood, the pressure within the arteries is much lower and is called *diastolic blood pressure*. In an adult this is about 80 mmHg or 11 kPa. The difference between systolic and diastolic blood pressures is the *pulse pressure*.

Arterial blood pressure (BP) is measured with a *sphygmomanometer* and is usually expressed with the systolic pressure written above the diastolic pressure:

$$BP = \frac{120}{80}\ \text{mmHg} \quad \text{or} \quad BP = \frac{16}{11}\ \text{kPa}$$

Elasticity of arterial walls. There is a considerable amount of elastic tissue in the arterial walls, especially in large arteries. Therefore, when the left ventricle ejects blood into the already full aorta, the aorta expands to accommodate it, and then recoils because of the elastic tissue in the wall. This pushes the blood forwards, into the systemic circulation. This distension and recoil occurs throughout the arterial system. During cardiac diastole the elastic recoil of the arteries maintains the diastolic pressure.

Factors determining blood pressure

Blood pressure is determined by *cardiac output* and *peripheral resistance*. Change in either of these parameters tends to alter systemic blood pressure, although the body's compensatory mechanisms usually adjust for any significant change.

$$\text{Blood pressure} = \text{Cardiac output} \times \text{Peripheral resistance}$$

Cardiac output

Cardiac output is determined by the stroke volume and the heart rate (p. 94). Factors that affect the heart rate and stroke volume are described above, and they may increase or decrease cardiac output and, in turn, blood pressure. An increase in cardiac output raises both systolic and diastolic pressures. An increase in stroke volume increases systolic pressure more than diastolic pressure.

Peripheral or arteriolar resistance

Arterioles, the smallest arteries, have a tunica media composed almost entirely of smooth muscle, which responds to nerve and chemical stimulation. Constriction and dilation of the arterioles are the main determinants of peripheral resistance (p. 85). Vasoconstriction causes blood pressure to rise and vasodilation causes it to fall.

When elastic tissue in the tunica media is replaced by inelastic fibrous tissue as part of the ageing process, blood pressure rises.

Autoregulation

Systemic blood pressure continually rises and falls, according to levels of activity, body position, etc. However, the body organs are capable of adjusting blood flow and blood pressure in their own local vessels independently of systemic blood pressure. This property is called *autoregulation*, and protects the tissues against swings in systemic pressures. It is especially important in the kidneys, which can be damaged by increased pressure in their delicate glomerular capillary beds (p. 339), and in the brain, which is very sensitive to even slight increases in levels of cellular waste.

Control of blood pressure (BP)

Blood pressure is controlled in two ways:

- short-term control, on a moment-to-moment basis, which mainly involves the baroreceptor reflex, discussed below, and also chemoreceptors and circulating hormones
- long-term control, which involves regulation of blood volume by the kidneys and the renin–angiotensin–aldosterone system (p. 343).

Table 5.1 The effects of the autonomic nervous system on the heart and blood vessels

	Sympathetic stimulation	Parasympathetic stimulation
Heart	↑rate ↑strength of contraction	↓rate ↓strength of contraction
Blood vessels	Most constrict, but arteries supplying skeletal muscle and brain dilate	Most blood vessels do not have a parasympathetic blood supply

Short-term blood pressure regulation

The cardiovascular centre (CVC) is a collection of interconnected neurones in the medulla and pons of the brain stem. The CVC receives, integrates and coordinates inputs from:

- baroreceptors (pressure receptors)
- chemoreceptors
- higher centres in the brain.

The CVC sends autonomic nerves (both sympathetic and parasympathetic [see Ch. 7]) to the heart and blood vessels (Table 5.1). It controls BP by slowing down or speeding up the heart rate and by dilating or constricting blood vessels. Activity in these fibres is essential for control of blood pressure (Fig. 5.22).

Baroreceptors

Within the wall of the aortic and carotid sinuses are *baroreceptors*, nerve endings sensitive to stretch (pressure) (Fig. 5.23), which are the body's principal moment-to-moment regulatory mechanism for controlling blood pressure. A rise in blood pressure in these arteries stimulates the baroreceptors, increasing their input to the CVC. The CVC responds by increasing parasympathetic nerve activity to the heart; this slows the heart down. At the same time, sympathetic stimulation to the blood vessels is inhibited, causing vasodilation. The net result is a fall in systemic blood pressure.

Conversely, if pressure within the aortic arch and carotid sinuses falls, the rate of baroreceptor discharge also falls. The CVC responds by increasing sympathetic drive to the heart to speed it up. Sympathetic activity in blood vessels is also increased, leading to vasoconstriction. Both these measures counteract the falling blood pressure. Baroreceptor control of blood pressure is also called the *baroreceptor reflex* (Fig. 5.23).

Chemoreceptors

These are nerve endings situated in the carotid and aortic bodies, and are primarily involved in control of respiration (p. 260). They are sensitive to changes in the levels of carbon dioxide, oxygen and the acidity of the blood (pH) (Fig. 5.24). Rising blood CO_2, falling blood O_2 levels and/or falling arterial blood pH all indicate failing tissue perfusion. When these changes are detected by the chemoreceptors, they send signals to the CVC, which then increases sympathetic drive to the heart and blood vessels, pushing blood pressure up to improve tissue blood supply. Because respiratory effort is also stimulated, blood oxygen levels rise as well.

Chemoreceptor input to the CVC influences its output only when severe disruption of respiratory function occurs or when arterial BP falls to less than 80 mmHg. Similar chemoreceptors are found on the brain surface in the medulla oblongata, and they measure carbon dioxide/oxygen levels and pH of the surrounding cerebrospinal fluid. Changes from normal activate responses similar to those described above for the aortic/carotid receptors.

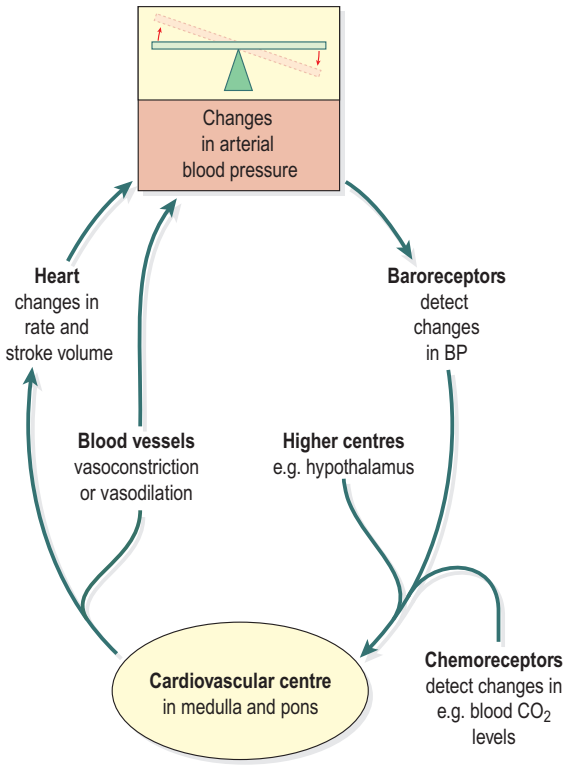


Figure 5.22 Summary of the main mechanisms in blood pressure control.

Higher centres in the brain

Input to the CVC from the higher centres is influenced by emotional states such as fear, anxiety, pain and anger that may stimulate changes in blood pressure.

The hypothalamus in the brain controls body temperature and influences the CVC, which responds by adjusting the diameter of blood vessels in the skin. This important mechanism regulates conservation and loss of heat so that core body temperature remains in the normal range (p. 366).

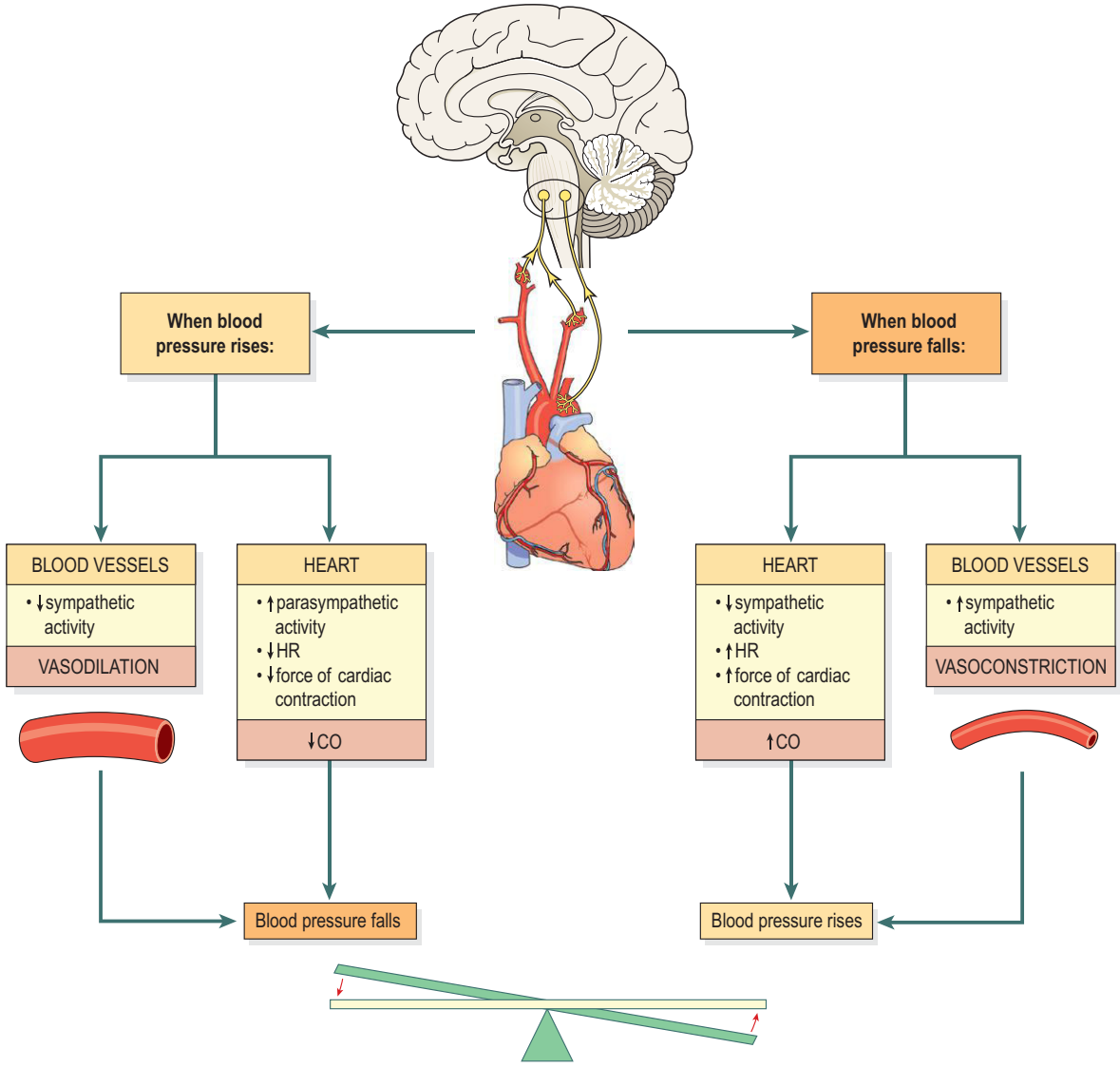


Figure 5.23 The baroreceptor reflex.

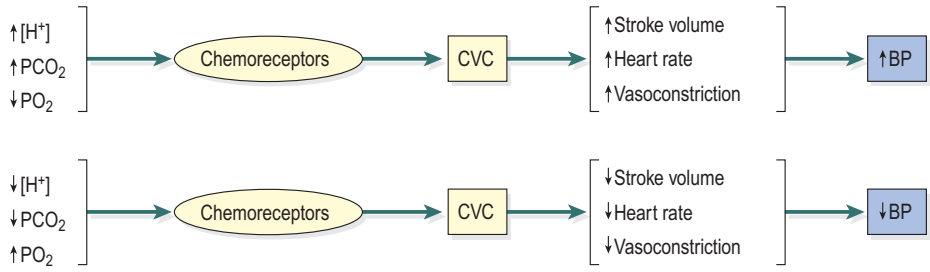


Figure 5.24 The relationship between stimulation of chemoreceptors and arterial blood pressure.

Long-term blood pressure regulation

Slower, longer lasting changes in blood pressure are effected by the *renin–angiotensin–aldosterone* system (RAAS, see p. 343) and the action of *antidiuretic hormone* (ADH, see p. 221). Both of these systems regulate blood volume, thus influencing blood pressure. In addition, *atrial natriuretic peptide* (ANP, p. 228), a hormone released by the heart itself, causes sodium and water loss from the kidney and reduces blood pressure, opposing the activities of both ADH and the RAAS.

Pressure in the pulmonary circulation

Pulmonary blood pressure is much lower than in the systemic circulation. This is because although the lungs receive the same amount of blood from the right ventricle as the rest of the body receives from the left ventricle, there are so many capillaries in the lungs that pressure is kept low. If pulmonary capillary pressure exceeds 25 mmHg, fluid is forced out of the bloodstream and into the airsacs (*pulmonary oedema*, p. 125), with very serious consequences. Autoregulation in the pulmonary circulation makes sure that blood flow through the vast network of capillaries is directed through well-oxygenated airsacs (p. 260).

Pulse

Learning outcomes

After studying this section, you should be able to:

- define the term pulse
- list the main sites on the body surface where the pulse is detected
- describe the main factors affecting the pulse.

The pulse can be felt with gentle finger pressure in a superficial artery when its wall is distended by blood pumped from the left ventricle during contraction (systole). The wave passes quickly as the arterial wall recoils. Each contraction of the left ventricle forces about 60–80 millilitres of blood through the already full aorta and into the arterial system. The aortic pressure wave is transmitted through the arterial system and can be felt at any point where a superficial artery can be pressed firmly but gently against a bone (Fig. 5.25). The number of pulse b.p.m. normally represents the heart rate and varies considerably in different people and in the same person at different times. An average of 60–80 is common at rest. Information that may be obtained from the pulse includes:

- the rate at which the heart is beating
- the regularity of the heartbeat – the intervals between beats should be equal

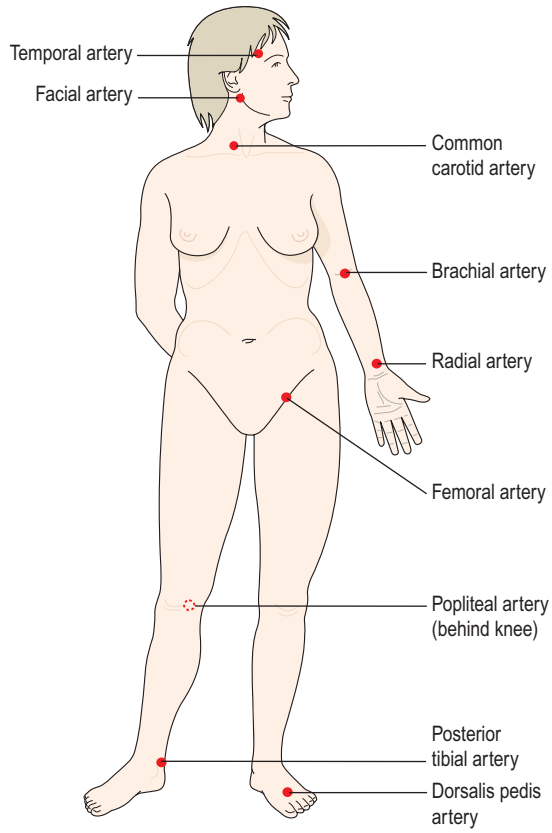


Figure 5.25 The main pulse points.

- the volume or strength of the beat – it should be possible to compress the artery with moderate pressure, stopping the flow of blood; the compressibility of the blood vessel gives some indication of the blood pressure and the state of the blood vessel wall
- the tension – the artery wall should feel soft and pliant under the fingers.

Factors affecting the pulse 5.9

In health, the pulse rate and the heart rate are identical. Factors influencing heart rate are summarised on page 95. In certain circumstances, the pulse may be less than the heart rate. This may occur, for example, if:

- the arteries supplying the peripheral tissues are narrowed or blocked and the blood therefore is not pumped through them with each heartbeat. Provided enough blood is reaching an extremity to nourish it, it will remain pink in colour and warm to touch, even if the pulse cannot be felt
- there is some disorder of cardiac contraction, e.g. atrial fibrillation (p. 129) and the heart is unable to generate enough force, with each contraction, to circulate blood to the peripheral arteries.

Circulation of the blood

Learning outcomes

After studying this section, you should be able to:

- describe the circulation of the blood through the lungs, naming the main vessels involved
- list the arteries supplying blood to all major body structures
- describe the venous drainage involved in returning blood to the heart from the body
- describe the arrangement of blood vessels relating to the portal circulation.

Although circulation of blood round the body is continuous (Fig. 5.16) it is convenient to describe it in two parts:

- pulmonary circulation
- systemic or general circulation (Figs 5.26 and 5.27).

Pulmonary circulation 5.10

This is the circulation of blood from the right ventricle of the heart to the lungs and back to the left atrium. In the lungs, carbon dioxide is excreted and oxygen is absorbed.

The pulmonary artery or trunk, carrying *deoxygenated blood*, leaves the upper part of the right ventricle of the heart. It passes upwards and divides into left and right pulmonary arteries at the level of the 5th thoracic vertebra.

The left pulmonary artery runs to the root of the left lung (p. 251) where it divides into two branches, one passing into each lobe.

The right pulmonary artery passes to the root of the right lung (p. 251) and divides into two branches. The larger branch carries blood to the middle and lower lobes, and the smaller branch to the upper lobe.

Within the lung these arteries divide and subdivide into smaller arteries, arterioles and capillaries. The exchange of gases takes place between capillary blood and air in the alveoli of the lungs (p. 259). In each lung the capillaries containing oxygenated blood merge into progressively larger venules, and eventually form two pulmonary veins.

Two pulmonary veins leave each lung, returning oxygenated blood to the left atrium of the heart. During atrial systole this blood is pumped into the left ventricle, and during ventricular systole it is forced into the aorta, the first artery of the general circulation.

Systemic or general circulation

The blood pumped out from the left ventricle is carried by the branches of the *aorta* around the body and returns to the right atrium of the heart by the *superior* and *inferior venae cavae*. Figure 5.26 shows the general positions of the aorta and the main arteries of the limbs. Figure 5.27 provides an overview of the venae cavae and the veins of the limbs.

The circulation of blood to the different parts of the body will be described in the order in which their arteries branch off the aorta.

Major blood vessels

The aorta is the largest artery of the body. The two largest veins, the superior and inferior venae cavae, return blood from all body parts to the heart.

Aorta (Fig. 5.28)

The aorta begins at the upper part of the left ventricle and, after passing upwards for a short way, it arches backwards and to the left. It then descends behind the heart through the thoracic cavity a little to the left of the thoracic vertebrae. At the level of the 12th thoracic vertebra it passes behind the diaphragm then downwards in the abdominal cavity to the level of the 4th lumbar vertebra, where it divides into the *right* and *left common iliac arteries*.

Throughout its length the aorta gives off numerous branches. Some of the branches are *paired*, i.e. there is a right and left branch of the same name, for instance, the right and left renal arteries supplying the kidneys, and some are single or *unpaired*, e.g. the coeliac artery.

The aorta will be described here according to its location:

- thoracic aorta (see below)
- abdominal aorta (p. 103).

Thoracic aorta (Fig. 5.28)

This part of the aorta lies above the diaphragm and is described in three parts:

- ascending aorta
- arch of the aorta
- descending aorta in the thorax (p. 103).

Ascending aorta. This is the short section of the aorta that rises from the heart. It is about 5 cm long and lies well protected behind the sternum.

The right and left coronary arteries are its only branches. They arise from the aorta just above the level of the aortic valve (Fig. 5.15) and supply the myocardium.

Arch of the aorta. This is a continuation of the ascending aorta. It begins behind the manubrium of the sternum

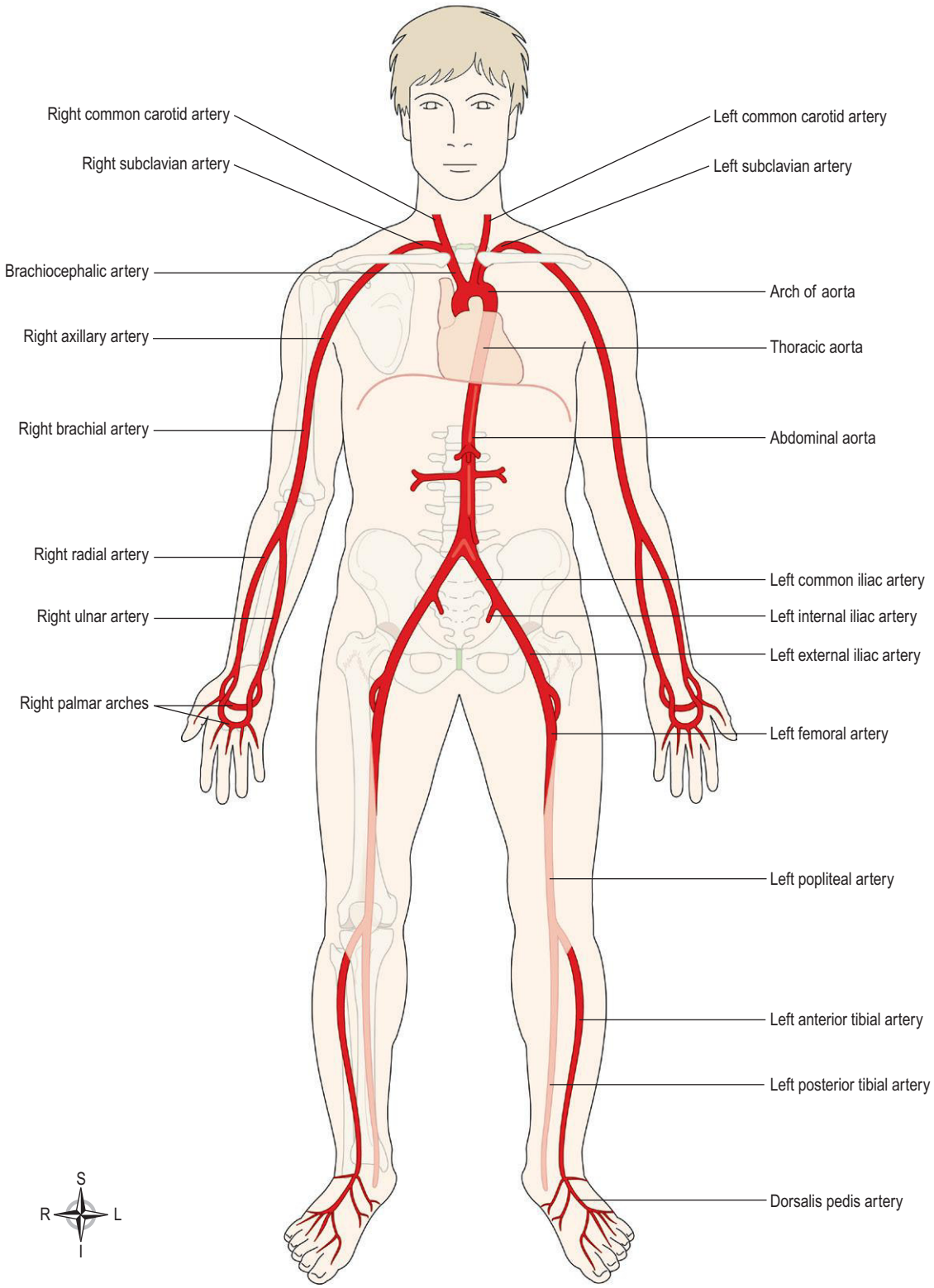


Figure 5.26 The aorta and the main arteries of the limbs.

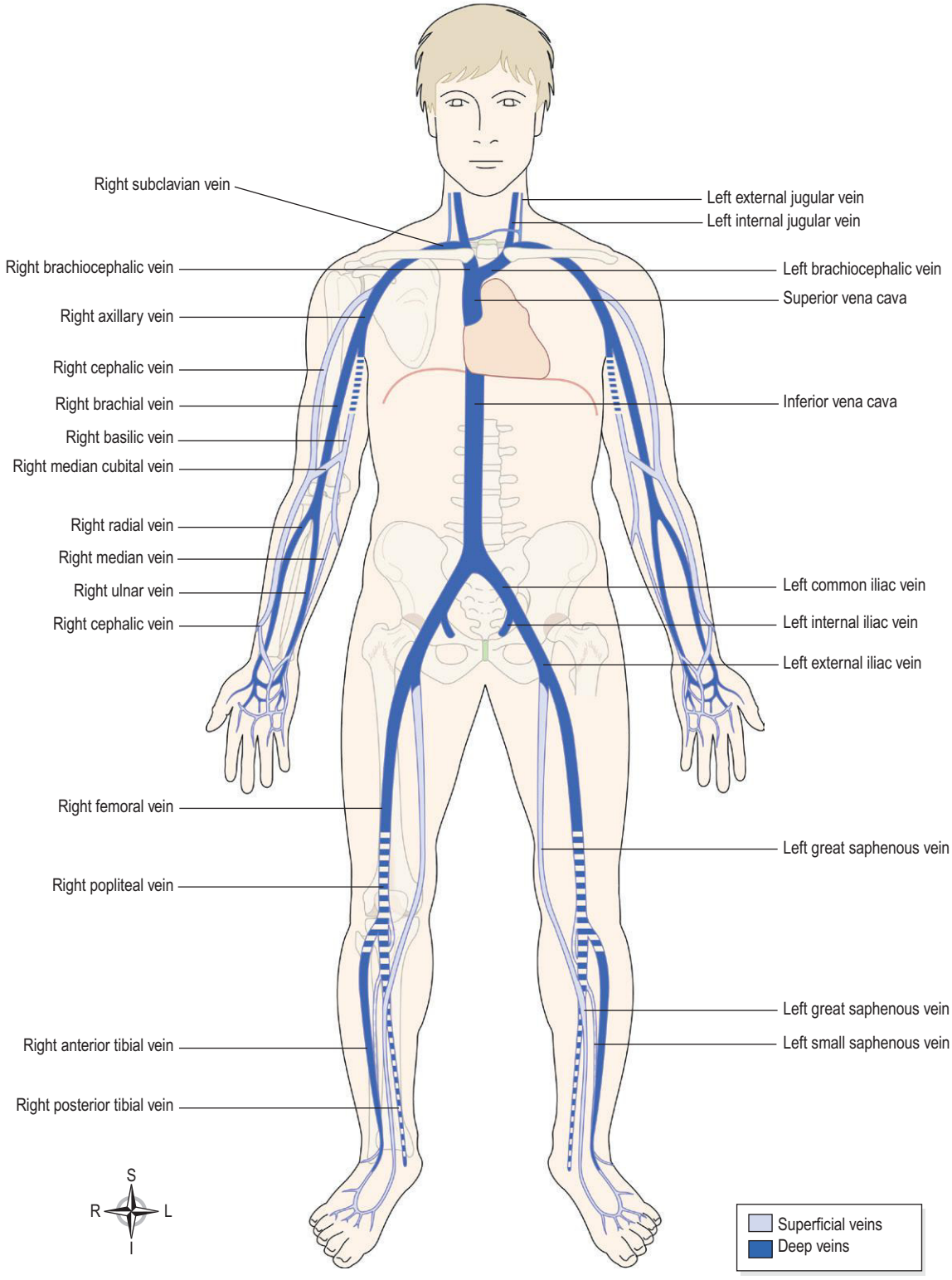


Figure 5.27 The venae cavae and the main veins of the limbs.

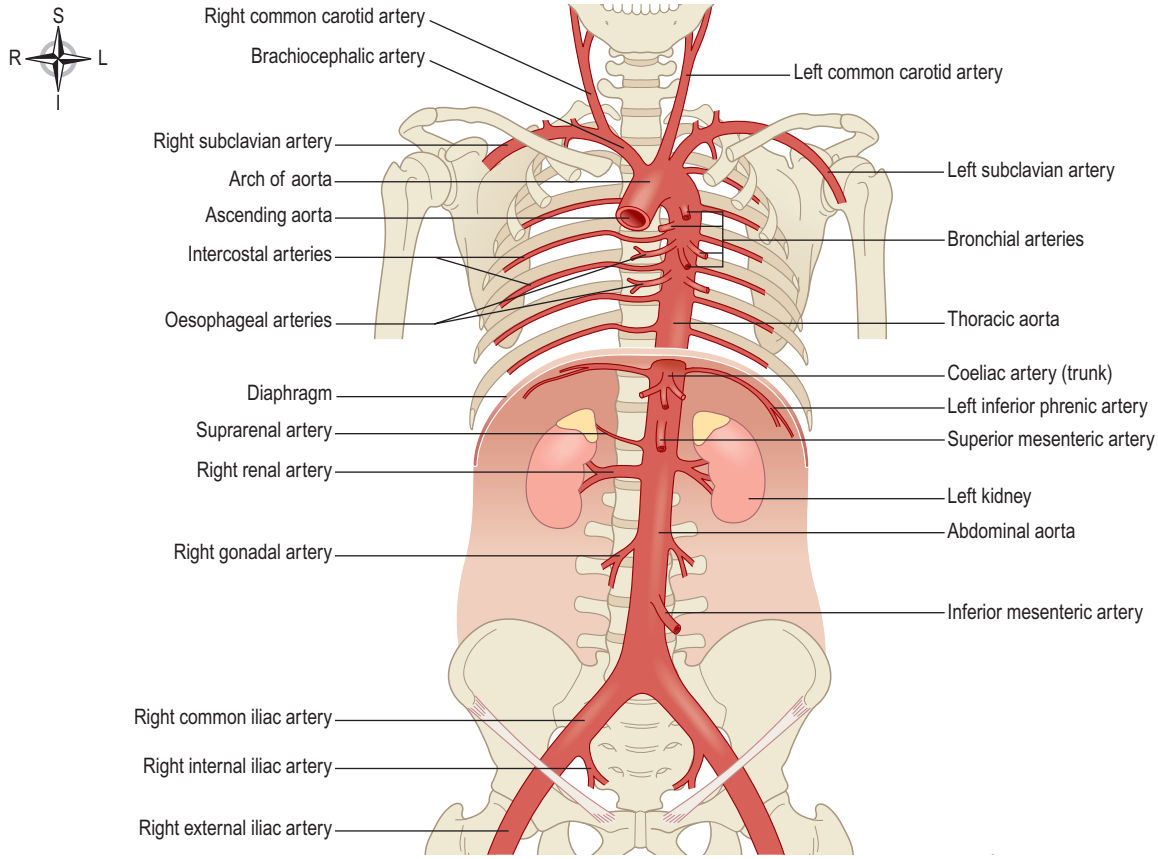


Figure 5.28 The aorta and its main branches.

and runs upwards, backwards and to the left in front of the trachea. It then passes downwards to the left of the trachea and is continuous with the descending aorta.

Three branches arise from its upper aspect:

- brachiocephalic artery or trunk
- left common carotid artery
- left subclavian artery.

The *brachiocephalic artery* is about 4 to 5 cm long and passes obliquely upwards, backwards and to the right. At the level of the sternoclavicular joint it divides into the *right common carotid artery* and the *right subclavian artery*.

Descending aorta in the thorax. This part is continuous with the arch of the aorta and begins at the level of the 4th thoracic vertebra. It extends downwards on the anterior surface of the bodies of the thoracic vertebrae to the level of the 12th thoracic vertebra, where it passes behind the diaphragm to become the abdominal aorta.

The descending aorta in the thorax gives off many paired branches which supply the walls and organs of the thoracic cavity (p. 108).

Abdominal aorta (Fig. 5.28)

The abdominal aorta is a continuation of the thoracic aorta. The name changes when the aorta enters the abdominal cavity by passing behind the diaphragm at the level of the 12th thoracic vertebra. It descends in front of the vertebral column to the level of the 4th lumbar vertebra, where it divides into the *right* and *left common iliac arteries*.

Many branches arise from the abdominal aorta, some paired and some unpaired, supplying the abdominal structures and organs (p. 108).

Venae cavae (Fig. 5.29)

The superior and inferior venae cavae are the largest veins in the body and empty blood directly into the right atrium of the heart (Fig. 5.14). The superior vena cava drains all body structures lying above the diaphragm and the inferior vena cava drains blood from all structures below the diaphragm.

Superior vena cava

This is about 7 cm long and is formed by the union of the left and right brachiocephalic veins.

Inferior vena cava

This is formed at the level of the 5th lumbar vertebra by the union of the right and left common iliac veins, and

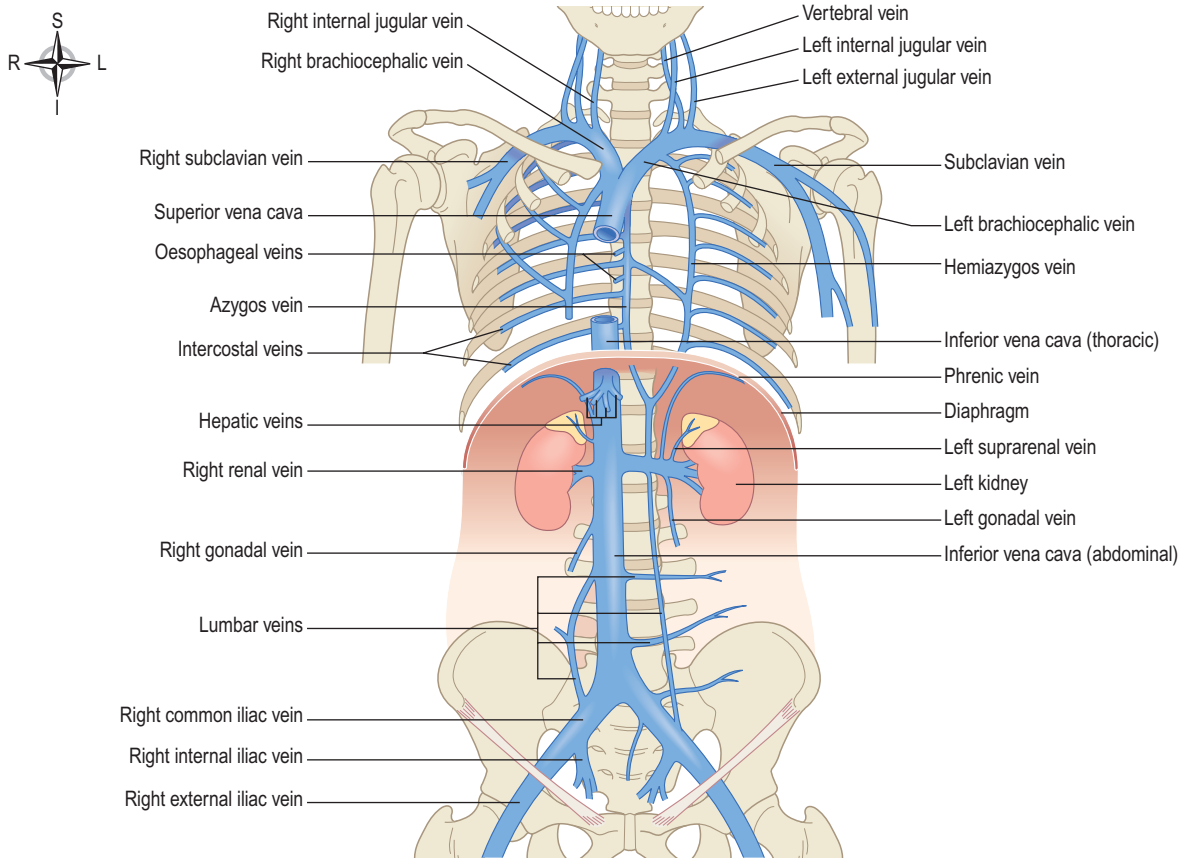


Figure 5.29 The venae cava and the main veins that form them.

ascends through the abdomen, lying close against the vertebral column and parallel to and just to the right of the descending abdominal aorta. It passes through the tendinous portion of the diaphragm into the thorax at the level of the 8th thoracic vertebra. As the inferior vena cava ascends through the abdomen, veins draining pelvic and abdominal organs empty into it (p. 110).

Circulation in the head and neck

Arterial supply

The paired arteries supplying the head and neck are the common carotid arteries and the vertebral arteries (Figs 5.30 and 5.32).

Carotid arteries. The *right common carotid artery* is a branch of the brachiocephalic artery. The *left common carotid artery* arises directly from the arch of the aorta. They pass upwards on either side of the neck and have the same distribution on each side. The common carotid arteries are embedded in fascia, called the *carotid sheath*. At the level of the upper border of the thyroid cartilage each divides into an *internal carotid artery* and an *external carotid artery*.

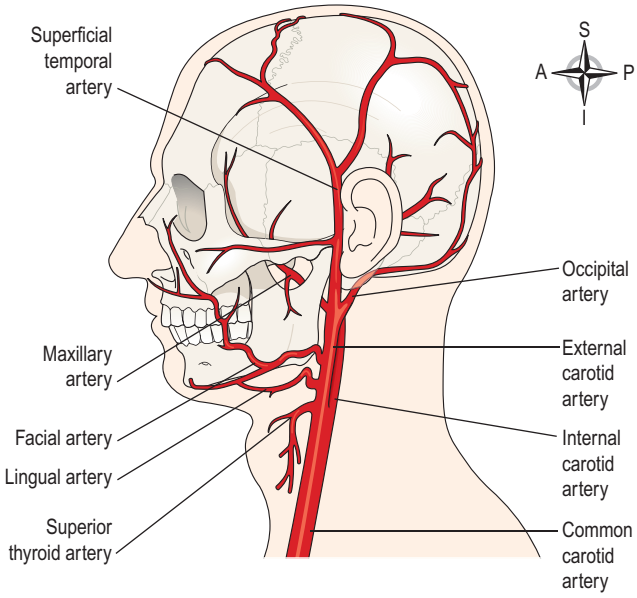


Figure 5.30 Main arteries of the left side of the head and neck.

The *carotid sinuses* are slight dilations at the point of division (bifurcation) of the common carotid arteries into their internal and external branches. The walls of the sinuses are thin and contain numerous nerve endings of the glossopharyngeal nerves. These nerve endings, or *baroreceptors*, are stimulated by changes in blood pressure in the carotid sinuses. The resultant nerve impulses initiate reflex adjustments of blood pressure through the vasomotor centre in the medulla oblongata (p. 159).

The carotid bodies are two small groups of *chemoreceptors*, one lying in close association with each common carotid artery at its bifurcation. They are supplied by the glossopharyngeal nerves and are stimulated by changes in the carbon dioxide and oxygen content of blood. The resultant nerve impulses initiate reflex adjustments of respiration through the respiratory centre in the medulla oblongata (p. 159).

External carotid artery (Fig. 5.30)**.** This artery supplies the superficial tissues of the head and neck, via a number of branches:

- The *superior thyroid artery* supplies the thyroid gland and adjacent muscles.
- The *lingual artery* supplies the tongue, the membrane that lines the mouth, the structures in the floor of the mouth, the tonsil and the epiglottis.
- The *facial artery* passes outwards over the mandible just in front of the angle of the jaw and supplies the muscles of facial expression (p. 423) and structures in the mouth. A pulse can be felt where the artery crosses the jaw bone.
- The *occipital artery* supplies the posterior part of the scalp.
- The *temporal artery* passes upwards over the zygomatic process in front of the ear and supplies the frontal, temporal and parietal parts of the scalp. The temporal pulse can be felt in front of the upper part of the ear.
- The *maxillary artery* supplies the muscles of mastication and a branch of this artery, the *middle meningeal artery*, runs deeply to supply structures in the interior of the skull.

Internal carotid artery. This is a major contributor to the circulus arteriosus (circle of Willis) (Fig. 5.31), which supplies the greater part of the brain. It also has branches that supply the eyes, forehead and nose. It ascends to the base of the skull and passes through the carotid foramen in the temporal bone.

Circulus arteriosus (circle of Willis [Fig. 5.31]**).** The greater part of the brain is supplied with arterial blood by an arrangement of arteries called the *circulus arteriosus* or the *circle of Willis*. Four large arteries contribute to its formation: the two *internal carotid arteries* and the two *vertebral arteries* (Fig. 5.32). The vertebral arteries arise from the subclavian arteries, pass upwards through the foramina in the transverse processes of the cervical vertebrae, enter the skull through the foramen magnum, then join to form the *basilar artery*. The arrangement in the circulus arteriosus is such that the brain as a whole receives an adequate blood supply even when a contributing artery is damaged and during extreme movements of the head and neck.

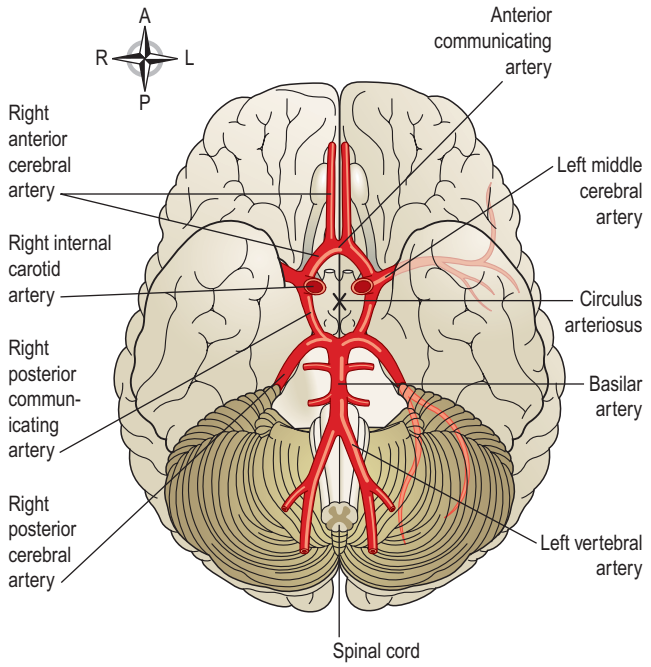


Figure 5.31 Arteries forming the circulus arteriosus (circle of Willis) and its main branches to the brain. Viewed from below.

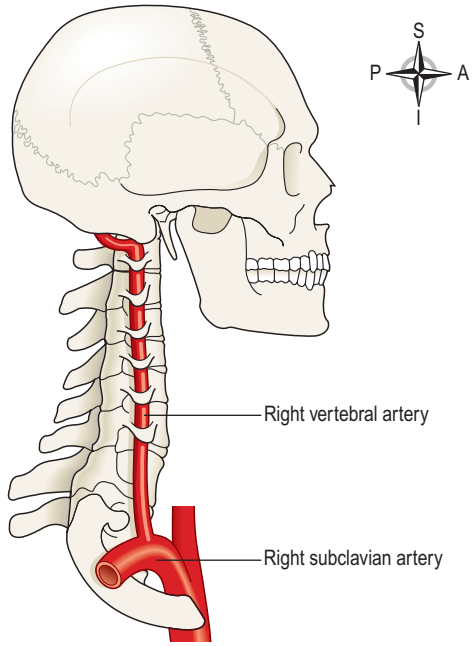


Figure 5.32 The right vertebral artery.

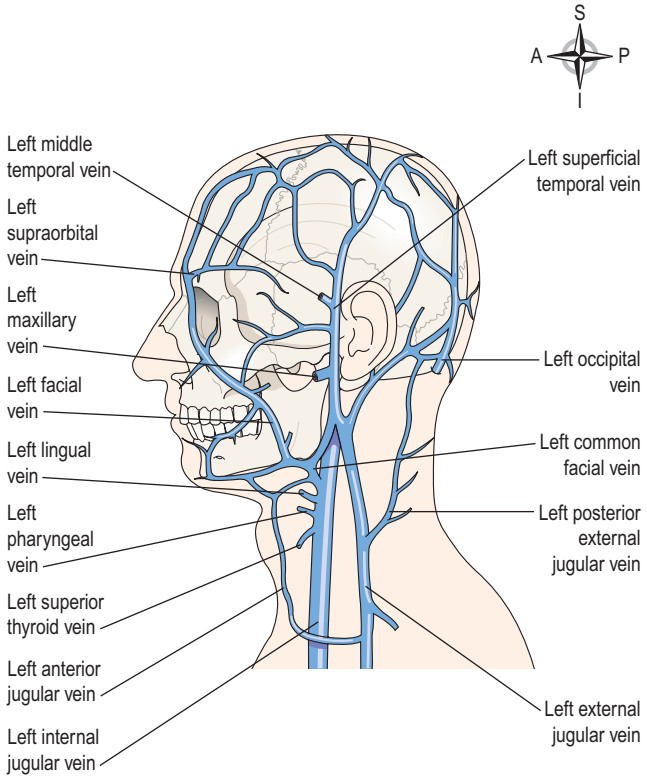


Figure 5.33 Veins of the left side of the head and neck.

Anteriorly, the two *anterior cerebral arteries* arise from the internal carotid arteries and are joined by the anterior *communicating artery*.

Posteriorly, the two *vertebral arteries* join to form the basilar artery. After travelling for a short distance the *basilar artery* divides to form two *posterior cerebral arteries*, each of which is joined to the corresponding internal carotid artery by a *posterior communicating artery*, completing the circle. The circulus arteriosus is therefore formed by:

- 2 anterior cerebral arteries
- 2 internal carotid arteries
- 1 anterior communicating artery
- 2 posterior communicating arteries
- 2 posterior cerebral arteries
- 1 basilar artery.

From this circle, the *anterior cerebral arteries* pass forward to supply the anterior part of the brain, the *middle cerebral arteries* pass laterally to supply the sides of the brain, and the *posterior cerebral arteries* supply the posterior part of the brain.

Branches of the basilar artery supply parts of the brain stem.

Venous return

Venous blood from the head and neck is returned by deep and superficial veins.

Superficial veins with the same names as the branches of the external carotid artery return venous blood from the superficial structures of the face and scalp and unite to form the external jugular vein (Fig. 5.33).

The external jugular vein begins in the neck at the level of the angle of the jaw. It passes downwards in front of the sternocleidomastoid muscle, then behind the clavicle before entering the subclavian vein.

Venous blood from the deep areas of the brain is collected into channels called the *dural venous sinuses* (Figs 5.34 and 5.35), which are formed by layers of dura mater lined with endothelium. The dura mater is the outer protective covering of the brain (p. 152). The main venous sinuses are listed below:

- The *superior sagittal sinus* carries the venous blood from the superior part of the brain. It begins in the

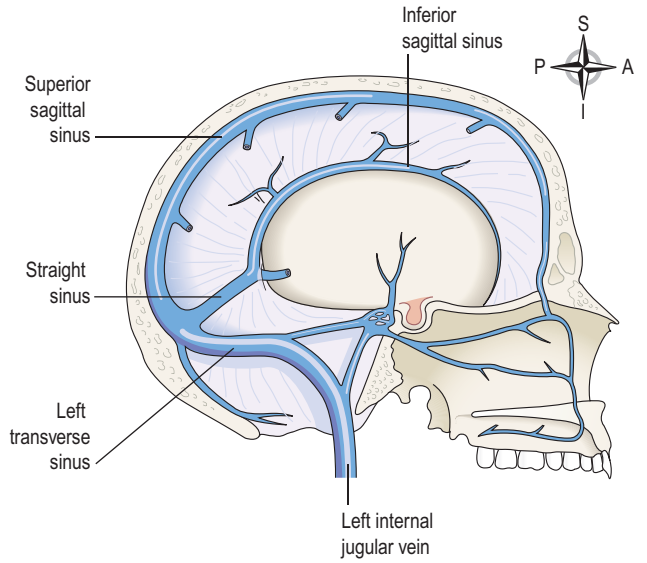


Figure 5.34 Venous sinuses of the brain viewed from the right.

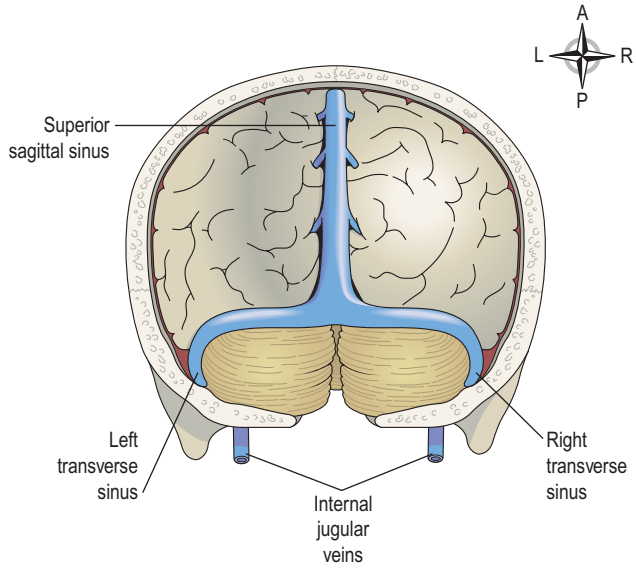


Figure 5.35 Venous sinuses of the brain viewed from above.

frontal region and passes directly backwards in the midline of the skull to the occipital region where it turns to the right side and continues as the *right transverse sinus*.

- The *inferior sagittal sinus* lies deep within the brain and passes backwards to form the *straight sinus*.
- The *straight sinus* runs backwards and downwards to become the *left transverse sinus*.
- The *transverse sinuses* begin in the occipital region. They run forward and medially in a curved groove of the skull, to become continuous with the *sigmoid sinuses*.
- The *sigmoid sinuses* are a continuation of the transverse sinuses. Each curves downwards and medially and lies in a groove in the mastoid process of the temporal bone. Anteriorly only a thin plate of bone separates the sinus from the air cells in the mastoid process of the temporal bone. Inferiorly it continues as the internal jugular vein.

The *internal jugular veins* begin at the jugular foramina in the middle cranial fossa and each is the continuation of a sigmoid sinus. They run downwards in the neck behind the sternocleidomastoid muscles. Behind the clavicle they unite with the *subclavian veins*, carrying blood from the upper limbs, to form the *brachiocephalic veins*.

The brachiocephalic veins are situated one on each side in the root of the neck. Each is formed by the union of the internal jugular and the subclavian veins. The left brachiocephalic vein is longer than the right and passes obliquely behind the manubrium of the sternum, where it joins the right brachiocephalic vein to form the superior vena cava (Fig. 5.29).

The *superior vena cava*, which drains all the venous blood from the head, neck and upper limbs, is about 7 cm long. It passes downwards along the right border of the sternum and ends in the right atrium of the heart.

Circulation in the upper limb

Arterial supply

The subclavian arteries. The right subclavian artery arises from the brachiocephalic artery; the left branches from the arch of the aorta. They are slightly arched and pass behind the clavicles and over the first ribs before entering the axillae, where they continue as the *axillary arteries* (Fig. 5.36).

Before entering the axilla, each subclavian artery gives off two branches: the *vertebral artery*, which passes upwards to supply the brain (Fig. 5.32), and the *internal thoracic artery*, which supplies the breast and a number of structures in the thoracic cavity.

The *axillary artery* is a continuation of the subclavian artery and lies in the axilla. The first part lies deeply; then it runs more superficially to become the *brachial artery*.

The *brachial artery* is a continuation of the axillary artery. It runs down the medial aspect of the upper arm,

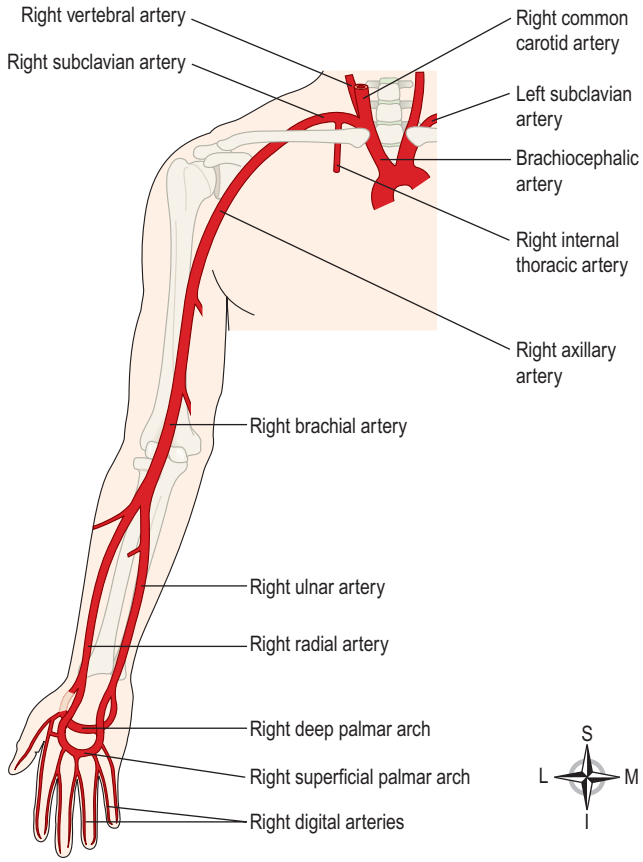


Figure 5.36 The main arteries of the right arm.

passes to the front of the elbow and extends to about 1 cm below the joint, where it divides into the *radial* and *ulnar arteries*.

The *radial artery* passes down the radial or lateral side of the forearm to the wrist. Just above the wrist it lies superficially and can be felt in front of the radius, as the radial pulse. The artery then passes between the first and second metacarpal bones and enters the palm of the hand.

The *ulnar artery* runs downwards on the ulnar or medial aspect of the forearm to cross the wrist and pass into the hand.

There are anastomoses between the radial and ulnar arteries, called the *deep* and *superficial palmar arches*, from which *palmar metacarpal* and *palmar digital arteries* arise to supply the structures in the hand and fingers.

Venous return

The upper limb is drained by both deep and superficial veins (Fig. 5.37). The deep veins follow the course of the arteries and have the same names:

- palmar metacarpal veins
- deep palmar venous arch
- ulnar and radial veins
- brachial vein
- axillary vein
- subclavian vein.

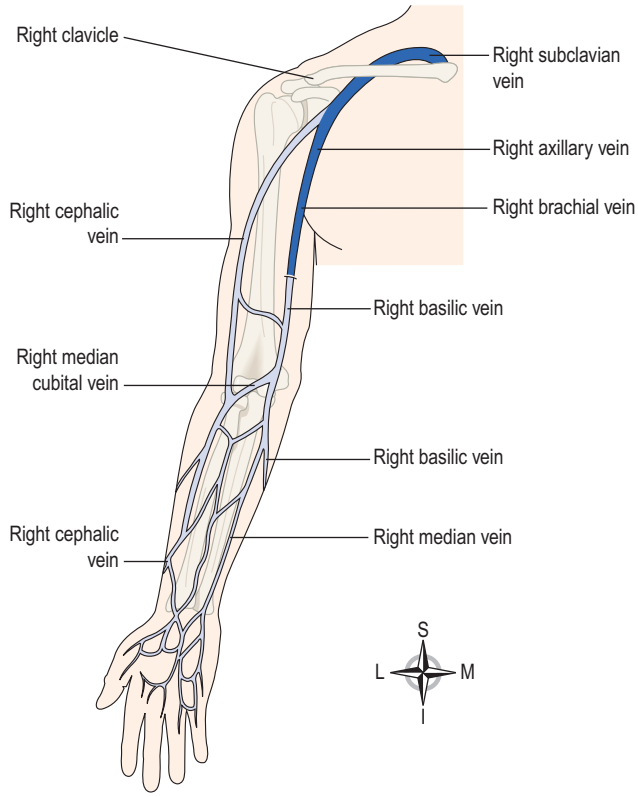


Figure 5.37 The main veins of the right arm. Dark blue indicates deep veins.

The superficial veins begin in the hand and consist of the following:

- cephalic vein
- basilic vein
- median vein
- median cubital vein.

The *cephalic vein* begins at the back of the hand where it collects blood from a complex of superficial veins, many of which can be easily seen. It then winds round the radial side to the anterior aspect of the forearm. In front of the elbow it gives off a large branch, the *median cubital vein*, which slants upwards and medially to join the *basilic vein*. After crossing the elbow joint the cephalic vein passes up the lateral aspect of the arm and in front of the shoulder joint to end in the axillary vein. Throughout its length it receives blood from the superficial tissues on the lateral aspects of the hand, forearm and arm.

The *basilic vein* begins at the back of the hand on the ulnar aspect. It ascends on the medial side of the forearm and upper arm then joins the axillary vein. It receives blood from the medial aspect of the hand, forearm and arm. There are many small veins which link the cephalic and basilic veins.

The *median vein* is a small vein that is not always present. It begins at the palmar surface of the hand, ascends on the front of the forearm and ends in the basilic vein or the median cubital vein.

The brachiocephalic vein is formed when the subclavian and internal jugular veins unite. There is one on each side.

The *superior vena cava* is formed when the two brachiocephalic veins unite. It drains all the venous blood from the head, neck and upper limbs and terminates in the right atrium. It is about 7 cm long and passes downwards along the right border of the sternum.

Circulation in the thorax

Arterial supply

Branches of the thoracic aorta (Fig. 5.28) supply structures in the chest, including:

- *bronchial arteries*, which supply lung tissues not directly involved in gas exchange
- *oesophageal arteries*, which supply the oesophagus
- *intercostal arteries*, which run along the inferior border of each rib and supply the intercostal muscles, some muscles of the thorax, the ribs, skin and its underlying connective tissues.

Venous return

Most of the venous blood from the organs in the thoracic cavity is drained into the *azygos vein* and the *hemiazygos* vein (Fig. 5.29). Some of the main veins that join them are the *bronchial, oesophageal* and *intercostal veins*. The azygos vein joins the superior vena cava and the hemiazygos vein joins the left brachiocephalic vein. At the distal end of the oesophagus, some oesophageal veins join the azygos vein, and others the left gastric vein. A venous plexus is formed by anastomoses between the veins joining the azygos vein and those joining the left gastric veins, linking the general and portal circulations (see Fig. 12.46, p. 322).

Circulation in the abdomen

Arterial supply

Branches of the abdominal aorta (Fig. 5.28) supply structures in the abdomen.

Paired branches. These include:

- *phrenic arteries*, supplying the diaphragm
- *renal arteries*, which supply the kidneys
- *suprarenal arteries*, supplying the adrenal glands
- *gonadal arteries*, supplying the ovaries (female) and testes (male). These arteries are much longer than the other paired branches, because the gonads begin their development high in the abdominal cavity. As fetal development proceeds, they descend into the pelvis and their supplying arteries become correspondingly longer to maintain supply.

Unpaired branches. These include the:

- *coeliac artery* (sometimes called the *coeliac trunk*, Fig. 5.38), a short thick artery about 1.25 cm long.

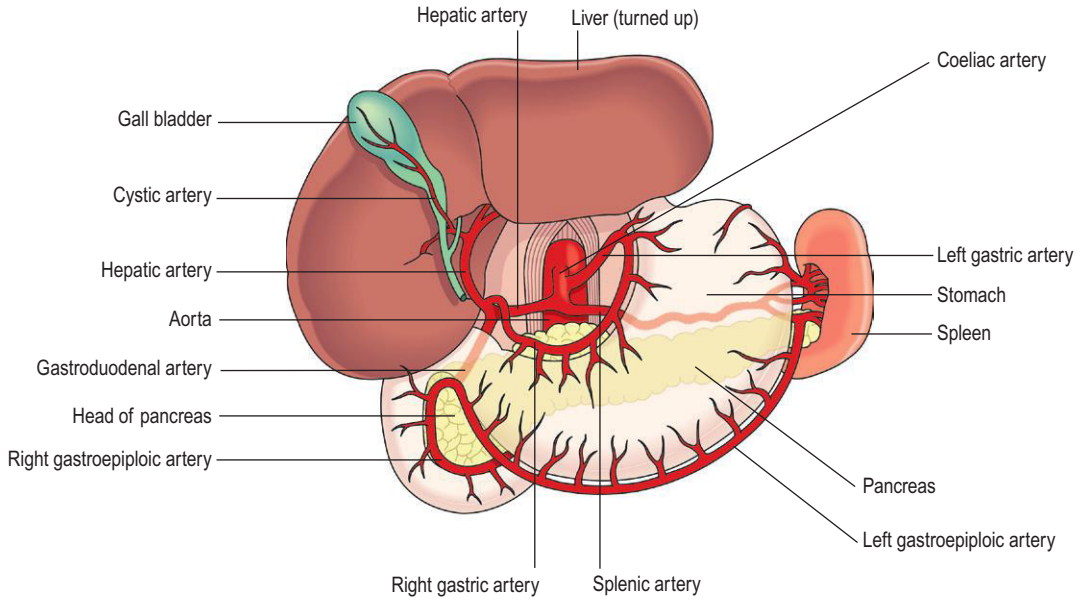


Figure 5.38 The coeliac artery and its branches, and the inferior phrenic arteries.

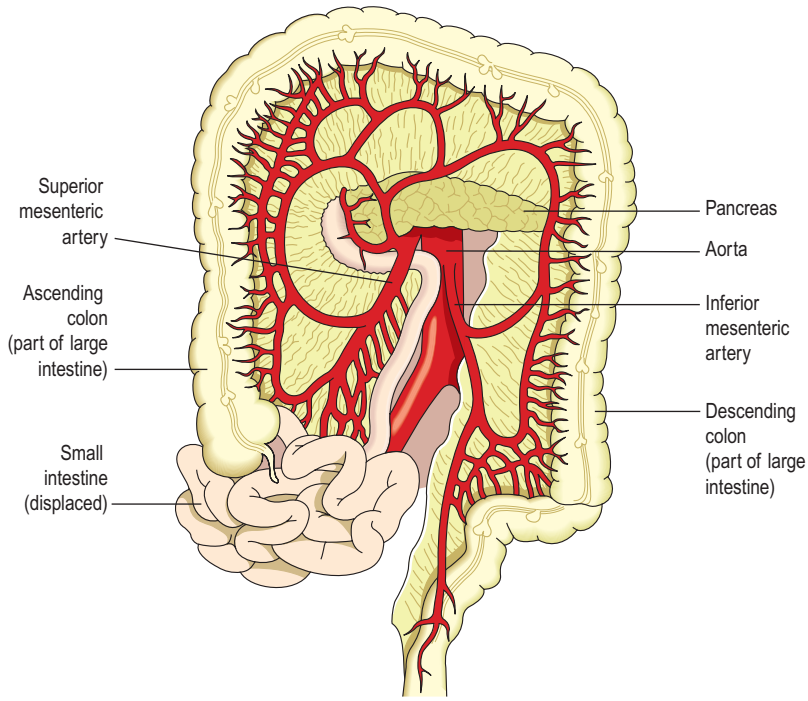


Figure 5.39 The superior and inferior mesenteric arteries and their branches.

It arises immediately below the diaphragm and divides into three branches:

- the *left gastric artery* supplying the stomach
- the *splenic artery* supplying the spleen and pancreas
- the *hepatic artery* supplying the liver, gall bladder and parts of the stomach, duodenum and pancreas

- *superior mesenteric artery* (Fig. 5.39), which branches from the aorta between the coeliac artery and the renal arteries. It supplies the entire small intestine and about half the proximal large intestine
- *inferior mesenteric artery* (Fig. 5.39), which arises from the aorta about 4 cm above its division into the common iliac arteries. It supplies the distal half of the large intestine and part of the rectum.

Venous return

Blood drains from some abdominal organs directly into the inferior vena cava via veins named as the corresponding arteries (Fig. 5.29). *Hepatic* veins drain the liver, *renal veins* drain the kidneys, *suprarenal veins* drain the adrenal glands, *lumbar veins* drain lower abdominal structures and *gonadal veins* drain the ovaries (female) and testes (male). However, most blood from the digestive organs in the abdomen is drained into the *hepatic portal vein* and passes through the liver before being emptied into the inferior vena cava (the *portal circulation*, see below).

Portal circulation 5.11

As a general rule, venous blood passes from the tissues to the heart by the most direct route through only one capillary bed. In the portal circulation, venous blood from the capillary beds of the abdominal part of the digestive system, the spleen and pancreas travels first to the liver. In the liver, it passes through a second capillary bed, the hepatic sinusoids, before entering the general circulation via the inferior vena cava. In this way, blood with a high concentration of nutrients, absorbed from the stomach and intestines, goes to the liver first. This supplies the liver with a rich source of nutrients for its extensive metabolic activities and ensures that the composition of blood leaving the alimentary tract can be appropriately regulated. It also ensures that unwanted and/or potentially toxic materials such as drugs are eliminated before the blood is returned into general circulation.

Portal vein. This is formed by the union of several veins (Figs 5.40 and 5.41), each of which drains blood from the area supplied by the corresponding artery:

- the *splenic vein* drains blood from the spleen, the pancreas and part of the stomach
- the *inferior mesenteric vein* returns the venous blood from the rectum, pelvic and descending colon of the large intestine. It joins the splenic vein
- the *superior mesenteric vein* returns venous blood from the small intestine and the proximal parts of the large intestine, i.e. the caecum, ascending and transverse colon. It unites with the splenic vein to form the portal vein
- the *gastric veins* drain blood from the stomach and the distal end of the oesophagus, then join the portal vein
- the *cystic vein,* which drains venous blood from the gall bladder, joins the portal vein.

After blood has passed through the hepatic portal circulation, it is then returned directly to the inferior vena cava through the hepatic veins.

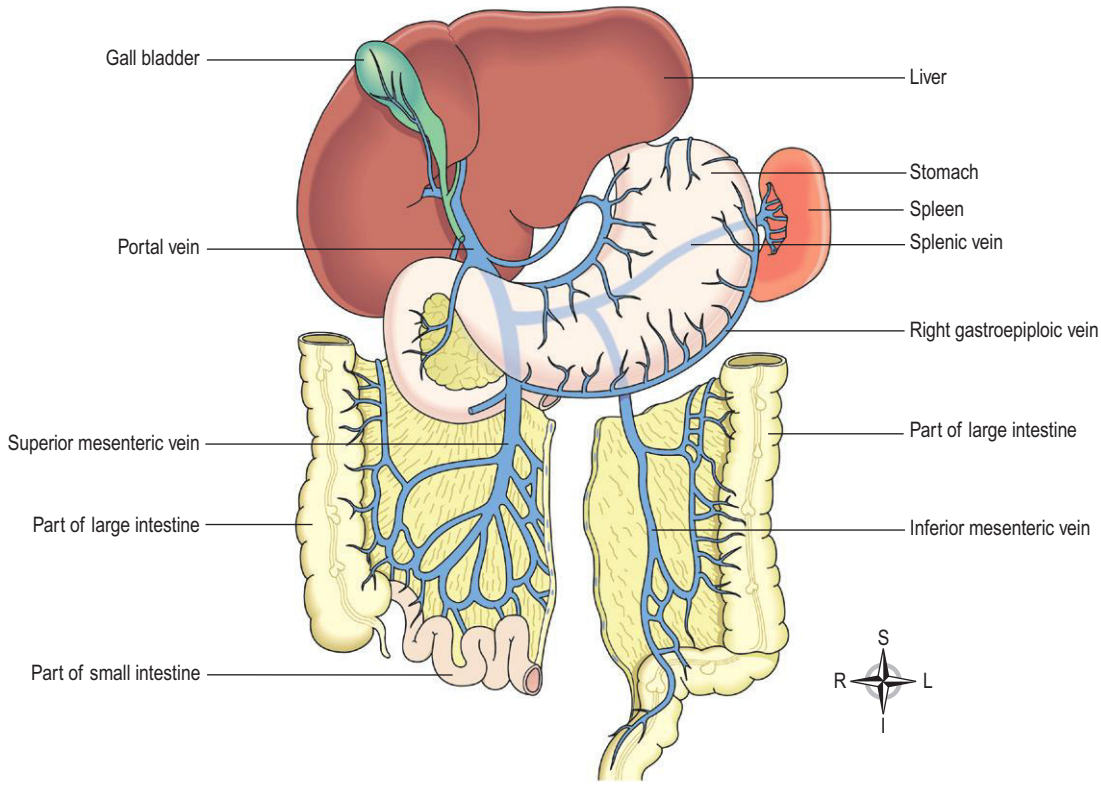


Figure 5.40 Venous drainage from the abdominal organs, and the formation of the portal vein.

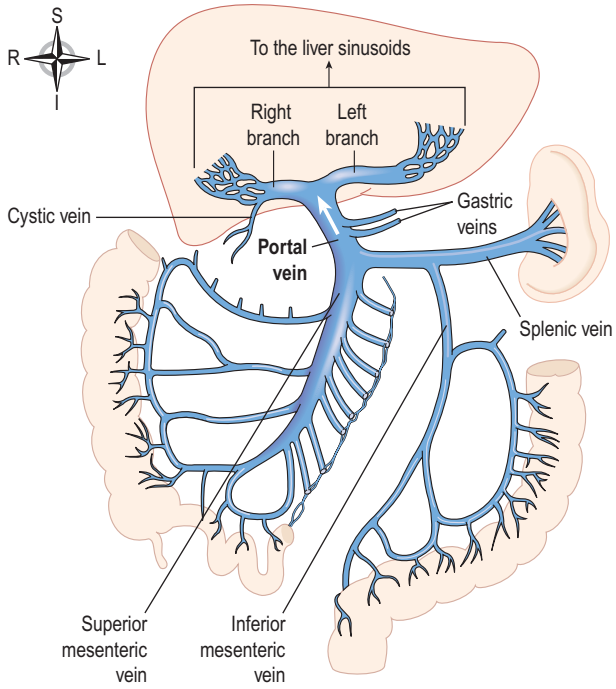


Figure 5.41 The portal vein: origin and termination.

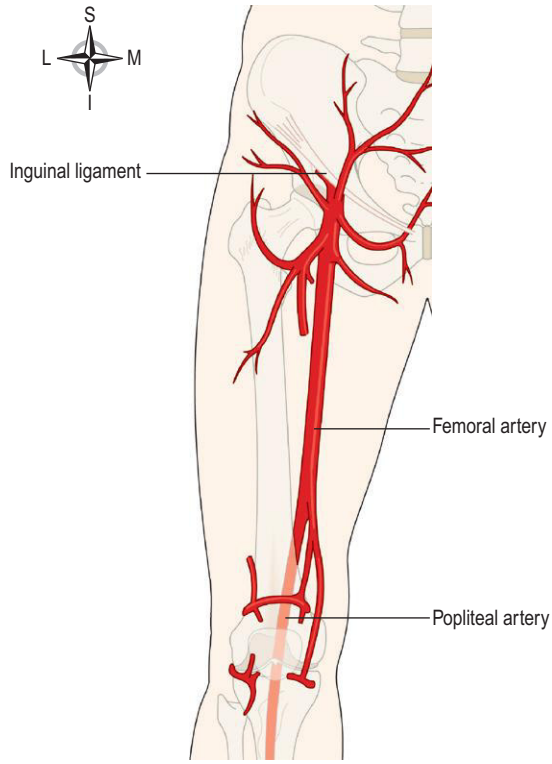


Figure 5.42 The femoral artery and its main branches.

Circulation in the pelvis and lower limb

Arterial supply

Common iliac arteries. The right and left common iliac arteries are formed when the abdominal aorta divides at the level of the 4th lumbar vertebra (Fig. 5.26). In front of the sacroiliac joint each divides into the internal and the external iliac arteries.

The *internal iliac artery* runs medially to supply the organs within the pelvic cavity. In the female, one of the largest branches is the uterine artery, which provides the main arterial blood supply to the reproductive organs.

The external iliac artery runs obliquely downwards and passes behind the inguinal ligament into the thigh where it becomes the femoral artery.

The *femoral artery* (Fig. 5.42) begins at the midpoint of the inguinal ligament and extends downwards in front of the thigh. The femoral pulse can be felt at the origin of the femoral artery. It then turns medially and eventually passes round the medial aspect of the femur to enter the popliteal space where it becomes the popliteal artery. It supplies blood to the structures of the thigh and some superficial pelvic and inguinal structures.

The *popliteal artery* (Fig. 5.43) passes through the popliteal fossa behind the knee, where the popliteal pulse can be felt. It supplies the structures in this area, including the knee joint. At the lower border of the popliteal fossa it divides into the anterior and posterior tibial arteries.

The *anterior tibial artery* (Fig. 5.43) passes forwards between the tibia and fibula and supplies the structures in the front of the leg. It lies on the tibia, runs in front of the ankle joint and continues over the dorsum (top) of the foot as the *dorsalis pedis artery*.

The *dorsalis pedis artery* is a continuation of the anterior tibial artery and passes over the dorsum of the foot, where the pulse can be felt, supplying arterial blood to the structures in this area. It ends by passing between the first and second metatarsal bones into the sole of the foot where it contributes to the formation of the plantar arch.

The *posterior tibial artery* (Fig. 5.43) runs downwards and medially on the back of the leg. Near its origin it gives off a large branch called the *peroneal artery*, which supplies the lateral aspect of the leg. In the lower part it becomes superficial and passes medial to the ankle joint to reach the sole of the foot, where it continues as the *plantar artery*.

The *plantar artery* supplies the structures in the sole of the foot. This artery, its branches and the dorsalis pedis artery form the *plantar arch* from which the digital branches arise to supply the toes.

Venous return

There are both deep and superficial veins in the lower limb (Fig. 5.27). Blood entering the superficial veins passes to the deep veins through *communicating veins*. Movement of blood towards the heart is partly dependent on contraction of skeletal muscles. Backward flow is prevented by a large number of valves. Superficial veins receive less support from surrounding tissues than deep veins.

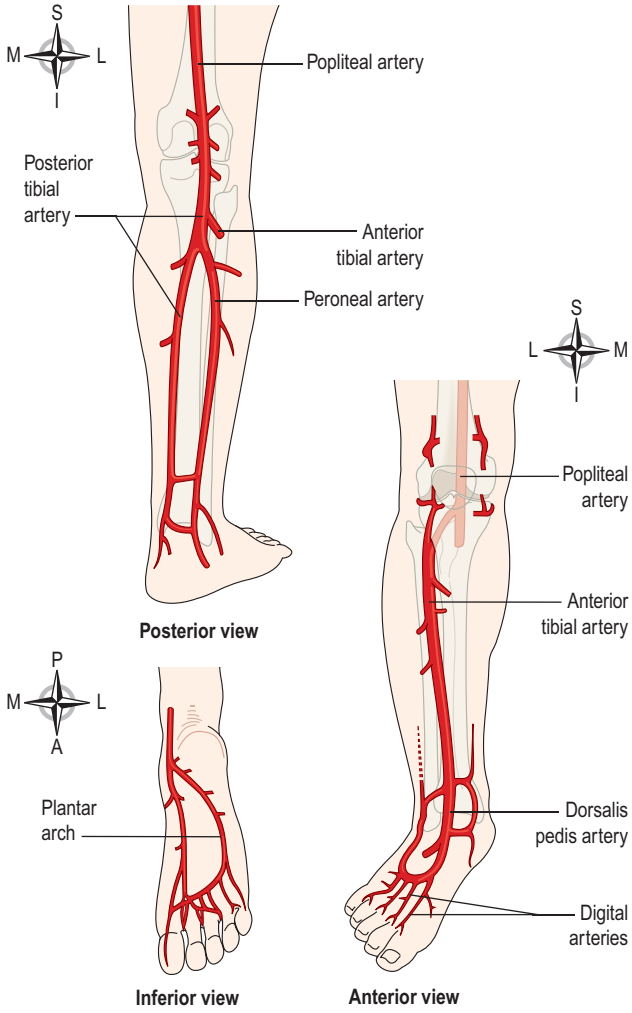


Figure 5.43 The right popliteal artery and its main branches.

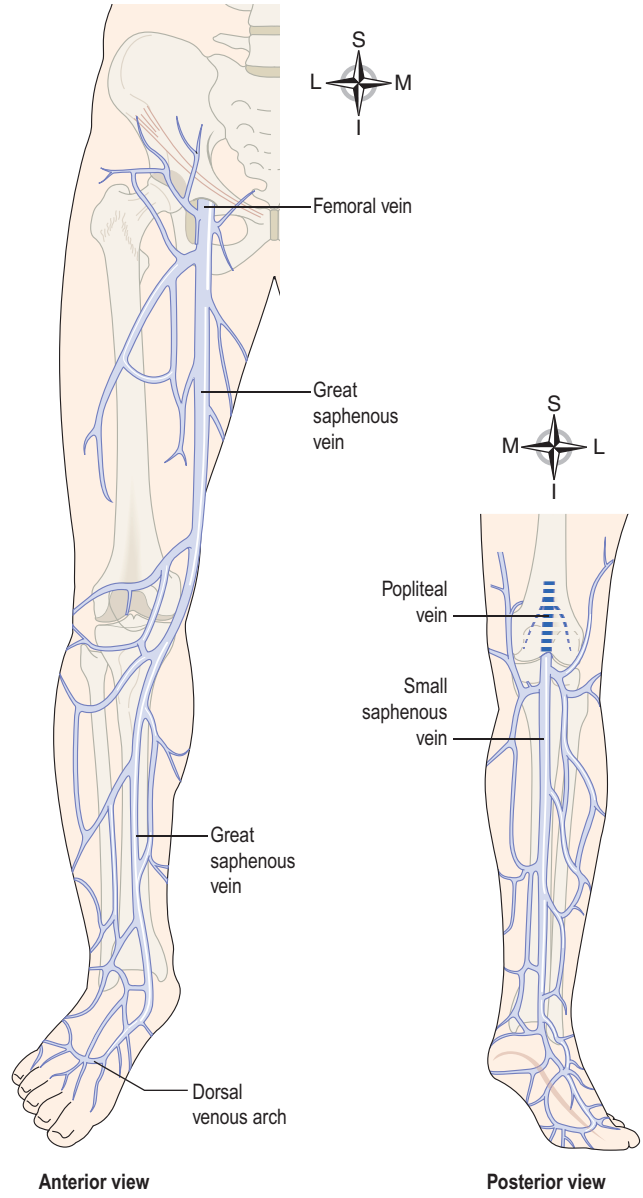


Figure 5.44 Superficial veins of the leg.

Deep veins. The deep veins accompany the arteries and their branches and have the same names. They are the:

- *femoral vein*, which ascends in the thigh to the level of the inguinal ligament, where it becomes the external iliac vein
- *external iliac vein*, the continuation of the femoral vein where it enters the pelvis lying close to the femoral artery. It passes along the brim of the pelvis, and at the level of the sacroiliac joint it is joined by the *internal iliac vein* to form the *common iliac vein*
- *internal iliac vein*, which receives tributaries from several veins draining the organs of the pelvic cavity
- *two common iliac veins*, which begin at the level of the sacroiliac joints. They ascend obliquely and end a little to the right of the body of the 5th lumbar vertebra by uniting to form the *inferior vena cava*.

Superficial veins (Fig. 5.44). The two main superficial veins draining blood from the lower limbs are the small and the great saphenous veins.

The *small saphenous vein* begins behind the ankle joint where many small veins which drain the dorsum of the foot join together. It ascends superficially along the back of the leg and in the popliteal space it joins the *popliteal vein* – a deep vein.

The *great saphenous vein* is the longest vein in the body. It begins at the medial half of the dorsum of the foot and runs upwards, crossing the medial aspect of the tibia and up the inner side of the thigh. Just below the inguinal ligament it joins the *femoral vein*.

Many communicating veins join the superficial veins, and the superficial and deep veins of the lower limb.

Summary of the main blood vessels (Fig. 5.45)

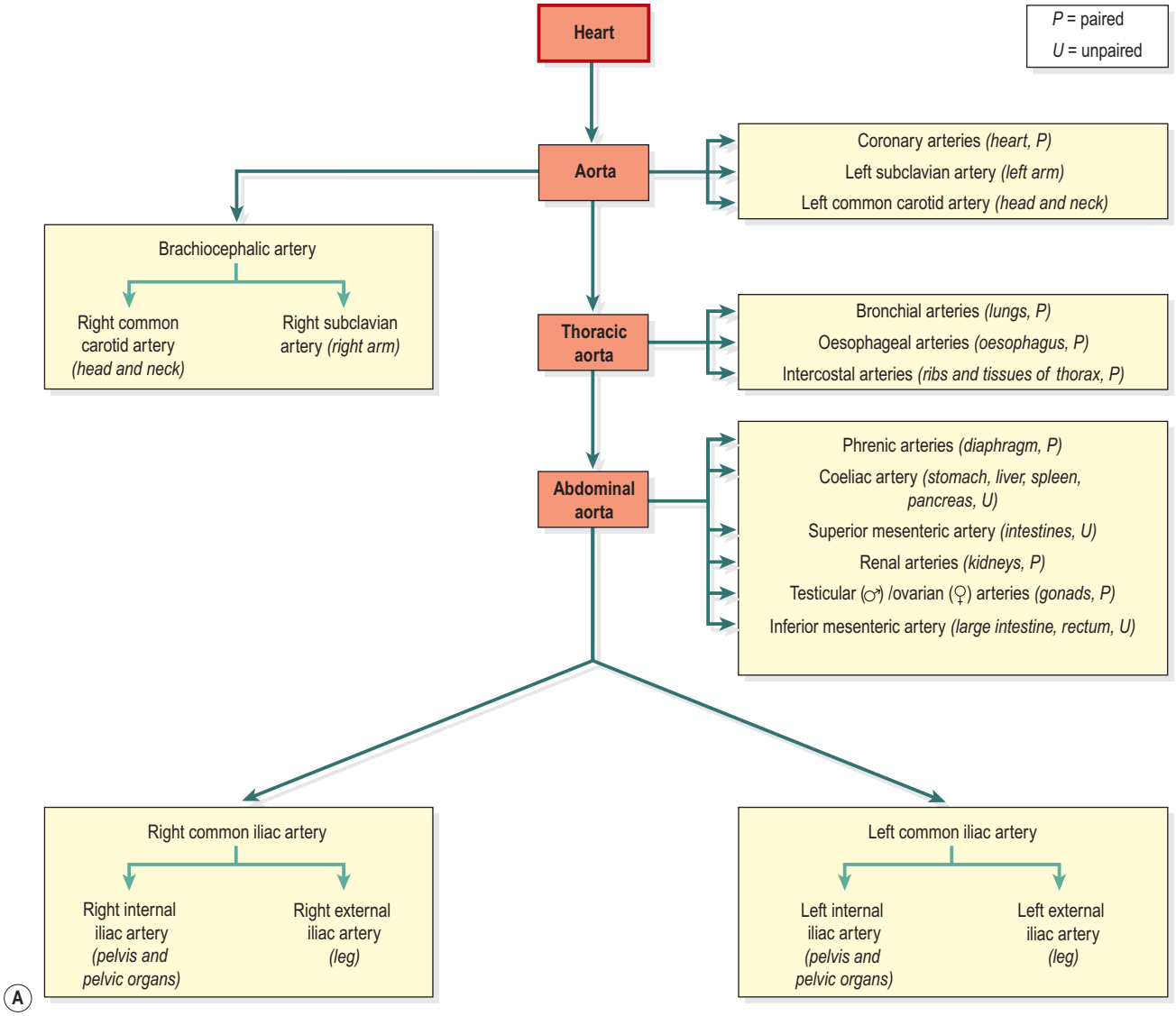


Figure 5.45 A. The aorta and main arteries of the body.

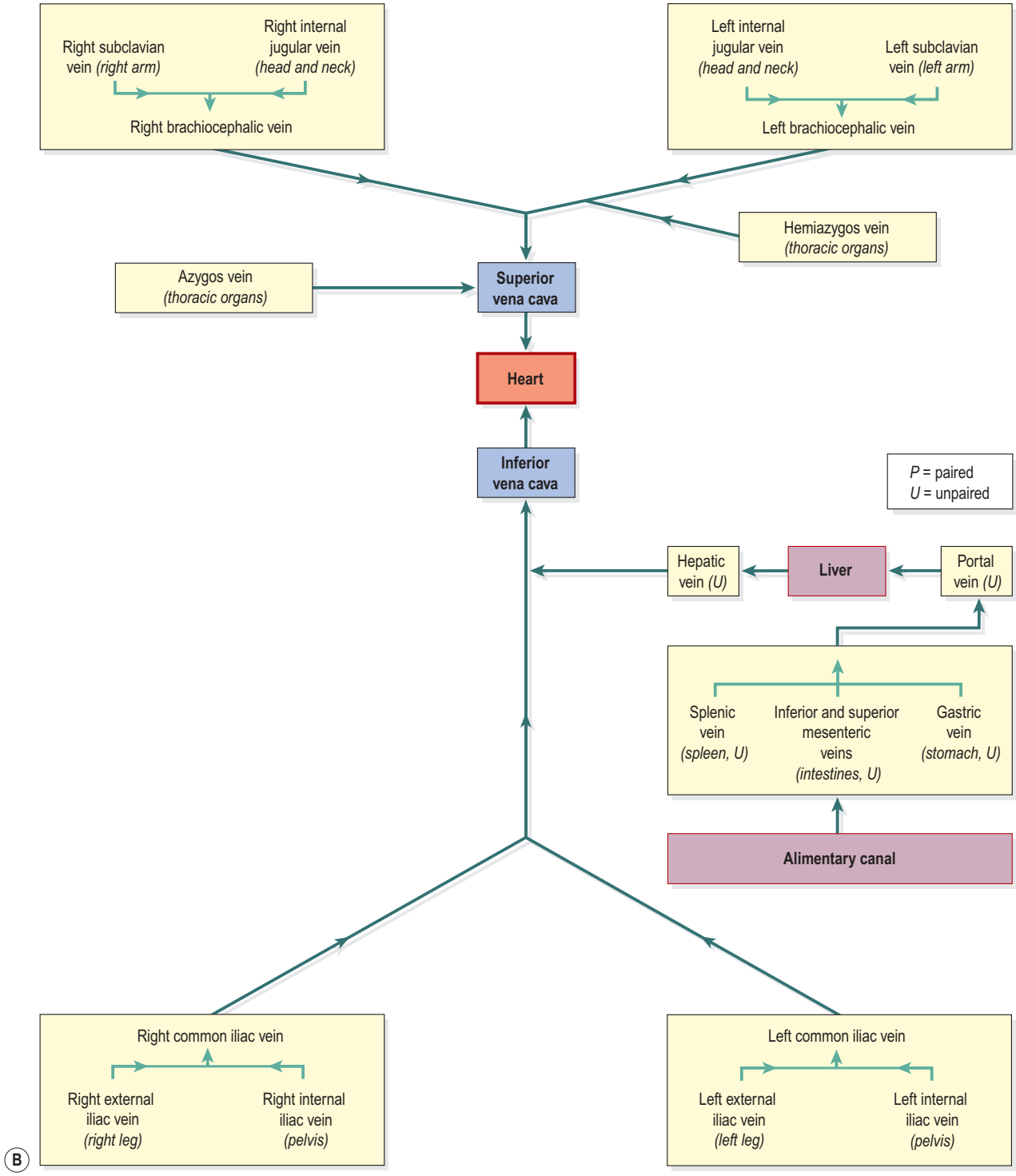


Figure 5.45 Continued B. The venae cavae and main veins of the body.

Fetal circulation

Learning outcomes

After studying this section, you should be able to:

- outline the functions of the placenta
- describe the fetal circulation
- compare blood flow through the heart, lungs and liver before and shortly after birth.

Features of the fetal circulation 5.12

The developing fetus obtains its oxygen and nutrients, and excretes its waste, via the mother's circulation. To this end, both maternal and fetal circulations develop specific adaptations unique to pregnancy. Because the lungs, gastrointestinal system and kidneys do not begin to function till after birth, certain modifications in the fetal circulation divert blood flow to meet pre-natal requirements.

Placenta

This is a temporary structure that provides an interface between the mother and fetus, and allows exchange of substances between their circulatory systems. It develops from the surface of the fertilized ovum embedded into the maternal uterine endometrium (Fig. 5.46). It is expelled from the uterus during the final stage of labour soon after birth, when it is no longer needed.

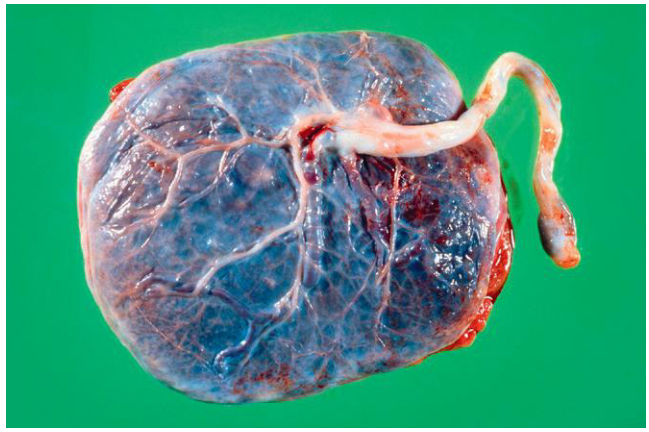
Structure

The mature placenta (Fig. 5.46A) is pancake-shaped, weighs around 500 g, has a diameter of 20 cm and is about 2.5 cm thick, although wide individual variations occur. The placenta is firmly attached to the uterine wall and consists of an extensive network of fetal capillaries bathed in maternal blood. Whilst the fetal capillaries are in very close proximity to the maternal blood supply, the two circulations are completely separate. The placenta is attached to the fetus by a cord (the *umbilical cord*), which is usually about 50 cm long and contains two *umbilical arteries* and one *umbilical vein* wrapped in a soft connective tissue coat (Fig. 5.46B). The cord enters the fetus at a spot on the abdomen called the *umbilicus*.

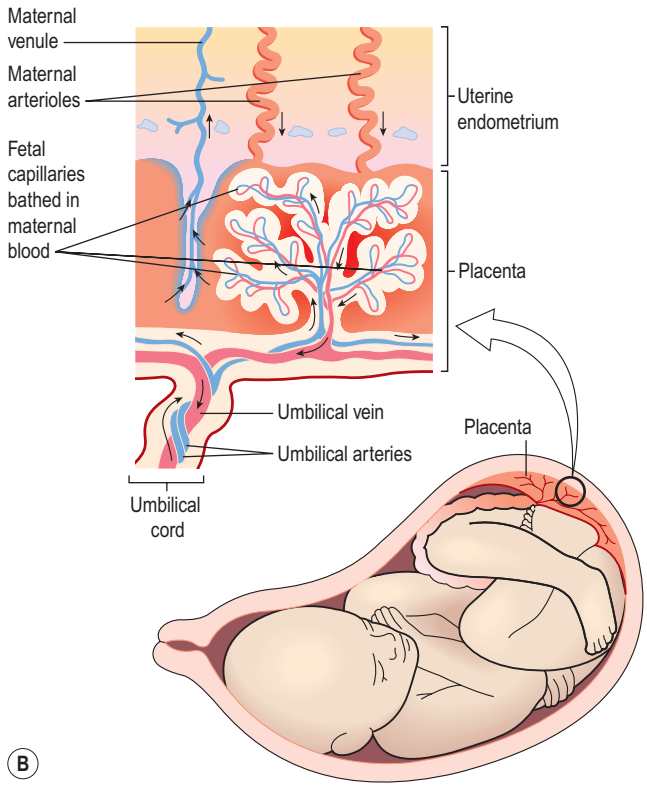
Functions

Placental functions include exchange of substances, protection of the fetus and maintenance of pregnancy.

Exchange of nutrients and wastes. Deoxygenated blood flows from the fetus into the placenta through the umbilical arteries, and travels through the network of fetal capillaries in the placenta. Because these capillaries are bathed in maternal blood, exchange of nutrients and gases takes place here and the blood that returns to the fetus in the umbilical vein has collected oxygen and nutrients and lost excess carbon dioxide and other wastes (Fig. 5.46).



A



B

Figure 5.46 The placenta. A. The mature placenta. **B.** The relationship between the uterine wall and the placenta.

Protection of the fetus. Temporary passive immunity (p. 383) lasting for a few months is provided by *maternal antibodies* that cross the placenta before birth.

Indirect exchange between the fetal and maternal circulations provides a 'barrier' to potentially harmful substances, including bacteria and drugs, although some may cross into the fetus, causing abnormal development.

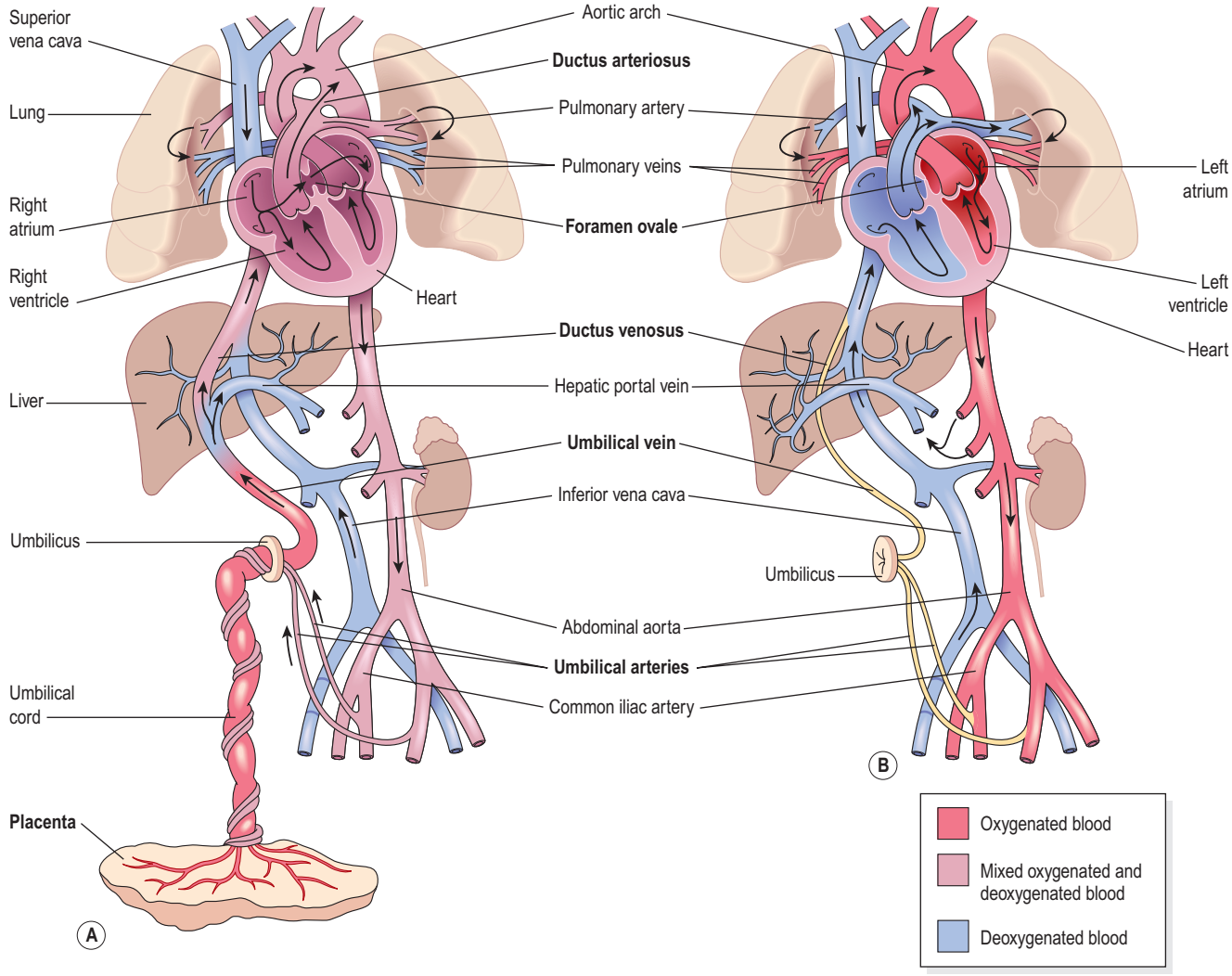


Figure 5.47 A. Fetal circulation before birth. **B.** Changes to the fetal circulation at birth.

Any substance causing abnormal fetal development is called a *teratogen*. Important teratogens include alcohol, certain drugs including some antibiotics and anticancer agents, ionising radiation and some infections, including the rubella (German measles) virus, cytomegalovirus and syphilis.

Maintenance of pregnancy. The placenta has an essential endocrine function and secretes the hormones that maintain pregnancy.

Human chorionic gonadotrophin (hCG). This hormone is secreted in early pregnancy, peaking at around 8 or 9 weeks and thereafter in smaller amounts. hCG stimulates the *corpus luteum* (Ch. 18) to continue secreting progesterone and oestrogen which prevent menstruation and maintain the uterine endometrium, sustaining pregnancy in the early weeks (see Fig. 18.10, p. 457).

Progesterone and oestrogen. As pregnancy progresses, the placenta takes over secretion of these hormones from the corpus luteum, which degenerates after about 12 weeks. From 12 weeks until delivery, the placenta secretes increasing levels of oestrogen and progesterone. These hormones are essential for maintenance of pregnancy.

Fetal adaptations (Fig. 5.47A)

Ductus venosus. This is a continuation of the umbilical vein that returns blood directly into the fetal inferior vena cava, and most blood, therefore, bypasses the non-functional fetal liver.

Ductus arteriosus. This small vessel connects the pulmonary artery to the descending thoracic aorta and diverts more blood into the systemic circulation, meaning that very little blood passes through the fetal lungs (see Fig. 5.59).

Foramen ovale. This forms a valve-like opening (see Fig. 5.60) allowing blood to flow between the right and

left atria, so that most blood bypasses the non-functional fetal lungs.

Changes at birth (Fig. 5.47B)

When the baby takes its first breath the lungs inflate for the first time, increasing pulmonary blood flow. Blood returning from the lungs increases the pressure in the left atrium, closing the flap over the foramen ovale and preventing blood flow between the atria. Blood entering the right atrium is therefore diverted into the right ventricle and into the pulmonary circulation through the pulmonary veins. As the pulmonary circulation is established (see Fig. 5.1) blood oxygen levels increase, causing constriction and closure of the ductus arteriosus. If these adaptations do not take place after birth, they become evident as congenital abnormalities (see Figs 5.59 and 5.60). When the placental circulation ceases, soon after birth, the umbilical vein, ductus venosus and umbilical arteries collapse, as they are no longer required.

Ageing and the cardiovascular system

Learning outcome

After studying this section, you should be able to:

- Describe the effects of ageing on the cardiovascular system.

Ageing and the heart

As the heart gets older, its function generally declines; cardiac output falls and the conduction pathways become less efficient. Cardiac muscle cell numbers steadily reduce with age, but hypertrophy (cell enlargement) generally balances this and the ventricles of the heart in older adults are actually slightly larger than in younger people. The compliance (stretchability) of the heart falls with age, mainly because the fibrous skeleton (p. 88) of the heart stiffens, increasing the heart's workload. The ability of the heart muscle to respond to adrenaline and noradrenaline lessens, and the contractile strength of the heart and cardiac reserve are reduced. The older heart is therefore more prone to heart failure (p. 126).

These changes occur in the healthy ageing heart, and are not consequences of disease. It is notable that age-related decline in cardiovascular function is greatly slowed in individuals who take regular exercise, even in old age.

Ageing and blood vessels

Vasoconstriction and vasodilation responses are less efficient in ageing blood vessels, so regulation of blood flow to the tissues is less well controlled. Arterial and arteriolar walls become stiffer and less compliant, which raises blood pressure and increases the work of the left ventricle. Blood pressure tends to rise with age, even in the absence of any overt cardiovascular disease. The amount of smooth muscle in the walls of most arteries, including those of the heart, kidneys and brain, rises with age, which contributes to their stiffening. This means that the blood supply to most body organs tends to fall, but in healthy old age it does not cause problems because it is matched by a general reduction in metabolic rate.

The baroreceptor reflex (p. 97) becomes less brisk with age, not only because the heart and blood vessels are slower to respond, but also because of neuronal ageing. This may lead to postural hypotension (p. 132).

Shock

Learning outcomes

After studying this section, you should be able to:

- define the term shock
- describe the main physiological changes that occur during shock
- explain the underlying pathophysiology of the main causes of shock.

Shock (circulatory failure) occurs when the metabolic needs of cells are not being met because of inadequate blood flow. In effect, there is a reduction in circulating blood volume, in blood pressure and in cardiac output. This causes tissue hypoxia, an inadequate supply of nutrients and the accumulation of waste products. A number of different types of shock are described.

Hypovolaemic shock

This occurs when the blood volume is reduced by 15–25%. Cardiac output may fall because of low blood volume and hence low venous return, as a result of different situations:

- severe haemorrhage – whole blood is lost
- extensive burns – serum is lost
- severe vomiting and diarrhoea – water and electrolytes are lost.

Cardiogenic shock

This occurs in acute heart disease when damaged heart muscle cannot maintain an adequate cardiac output, e.g. in myocardial infarction.

Septic shock (bacteraemic, endotoxic)

This is caused by severe infections in which bacterial toxins are released into the circulation. These toxins trigger a massive inflammatory and immune response, and many powerful mediators are released. Because the response is not controlled, it can cause multiple organ damage, depression of myocardial contractility, poor tissue perfusion and tissue death (necrosis). Profound hypotension occurs because the inflammatory mediators cause profound vasodilation.

Neurogenic shock

The causes include sudden acute pain, severe emotional experience, spinal anaesthesia and spinal cord damage. These interfere with normal nervous control of blood vessel diameter, leading to hypotension.

Anaphylactic shock

Anaphylaxis (p. 385) is a severe allergic response that may be triggered in sensitive individuals by substances like penicillin, peanuts or latex rubber. Vasodilation, provoked by systemic release of inflammatory mediators, e.g. histamine and bradykinin, causes venous pooling and hypotension. Severe bronchoconstriction leads to respiratory difficulty and hypoxia. Onset is usually sudden, and in severe cases can cause death in a matter of minutes if untreated.

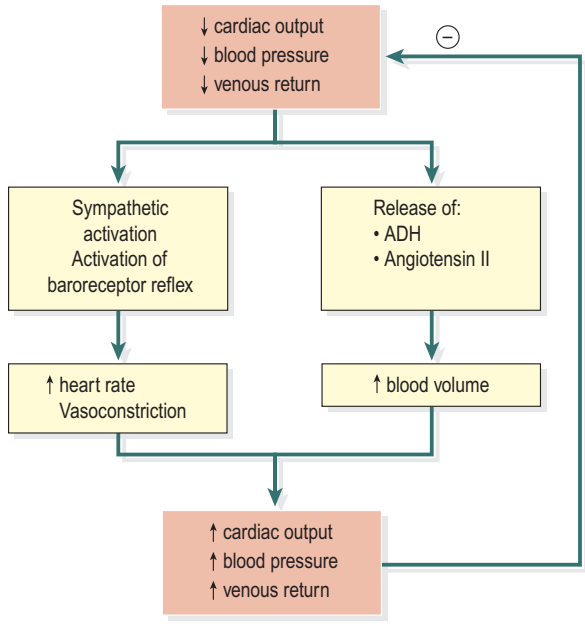


Figure 5.48 Compensatory mechanisms in shock.

Physiological changes during shock

In the short term, changes are associated with physiological attempts to restore an adequate blood circulation – *compensated shock* (Fig. 5.48). If the state of shock persists, the longer-term changes may be irreversible.

Compensated shock

As the blood pressure falls, a number of reflexes are stimulated and hormone secretions increased in an attempt to restore it. These raise blood pressure by increasing peripheral resistance, blood volume and cardiac output (Fig. 5.48).

Increased sympathetic stimulation increases heart rate and cardiac output, and also causes vasoconstriction, all of which increase blood pressure. Low blood volume and increased osmolarity of the blood cause secretion of ADH (p. 221) and activation of the renin–angiotensin–aldosterone system (p. 225). Consequent release of aldosterone reduces water and sodium excretion and promotes vasoconstriction. The veins also constrict, helping to reduce venous pooling and support venous return.

If these compensatory mechanisms, plus any medical interventions available, are sufficient then perfusion of the heart and brain can be maintained and the patient's condition may be stabilised.

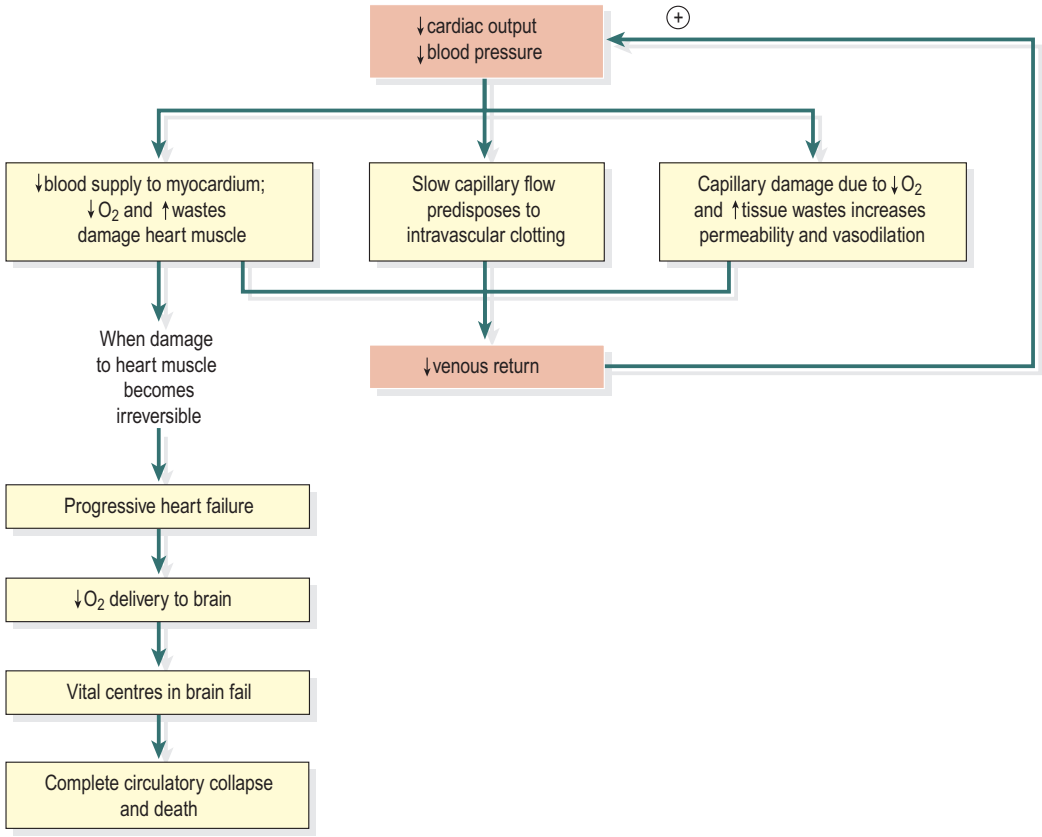


Figure 5.49 Uncompensated shock.

Uncompensated shock

If the insult is more severe, shock becomes a self-perpetuating sequence of deteriorating cardiovascular function – *uncompensated shock* (Fig. 5.49). Hypoxia causes cellular metabolism to switch to anaerobic pathways (p. 316), resulting in accumulation of lactic acid and progressive acidosis, which damages capillaries. The capillaries then become more permeable, leaking fluid from the vascular system into the tissues, further lowering blood pressure and tissue perfusion. Also, the accumulation of waste products causes vasodilation, making it harder for control mechanisms to support blood pressure. Organs, including the heart, are deprived of oxygen and may start to fail.

Eventually, the cardiovascular system reaches the stage when, although its compensatory mechanisms are running at maximum, it is unable to supply the brain's requirements. As the brain, including the cardiovascular and respiratory centres in the brain stem, becomes starved of oxygen and nutrients, it begins to fail and there is loss of central control of the body's compensatory mechanisms. Circulatory collapse follows. Finally, degenerating cardiovascular function leads to irreversible and progressive brain-stem damage, and death follows.

Thrombosis and embolism

Learning outcomes

After studying this section, you should be able to:

- define the terms thrombosis, embolism and infarction
- explain, in general terms, the effects of the above on the body
- describe three risk factors for thrombosis formation.

Thrombosis

Thrombosis is the formation of a blood clot (thrombus) inside a blood vessel, interrupting blood supply to the tissues. The risk of a thrombus developing within a blood vessel is increased by:

Slow blood flow. This may happen in immobility, e.g. prolonged sitting or in bedrest, or if a blood vessel is compressed by an adjacent structure such as a tumour or tight clothing, or if there is a sustained fall in blood pressure, as in shock.

Box 5.3 Possible embolic materials

- Fragments of atheromatous plaques (p. 121)
- Fragments of vegetations from heart valves, e.g. in infective endocarditis (p. 128)
- Tumour fragments, which may cause metastases
- Amniotic fluid, during childbirth
- Fat, from bone fractures
- Air, from a punctured blood vessel, e.g. by a broken rib or during a clinical procedure
- Nitrogen bubbles in decompression sickness (the 'bends')
- Pus from an abscess

Damage to the blood vessel intima. This is usually associated with atherosclerosis (p. 121).

Increased blood coagulability. Dehydration, pregnancy and childbirth, blood clotting disorders, some malignant disease, the presence of an intravenous cannula and oestrogen (including when used as a contraceptive) all increase the risk of blood clots forming.

Embolism

Embolism is the blocking of a blood vessel by any mass of material (an *embolus*) travelling in the blood. This is usually a thrombus or a fragment of a thrombus, but other embolic materials are shown in Box 5.3.

Emboli originating in an artery travel away from the heart until they reach an artery too narrow to let them pass, and lodge there, partly or completely blocking blood supply to distal tissues. This is a common cause of stroke (p. 181), myocardial infarction (p. 127) and gangrenous limbs (Fig. 5.50). Emboli originating in veins (DVT, p. 123) travel towards the heart, and from there to the lungs in the pulmonary artery. They then lodge in the first branch narrower than they are (pulmonary embolism).

Pulmonary embolism. Where a pulmonary artery or one of its branches is blocked causing an immediate reduction in blood flow through the lung, is one of the most serious consequences of venous embolism. Massive pulmonary embolism blocks a main pulmonary artery and usually causes sudden collapse and death.

Infarction and ischaemia

Infarction is the term given to tissue death because of interrupted blood supply. The consequences of interrupting tissue blood supply depend of the size of the artery blocked and the functions of the tissue affected. *Ischaemia* means tissue damage because of reduced blood supply (Fig. 5.50).

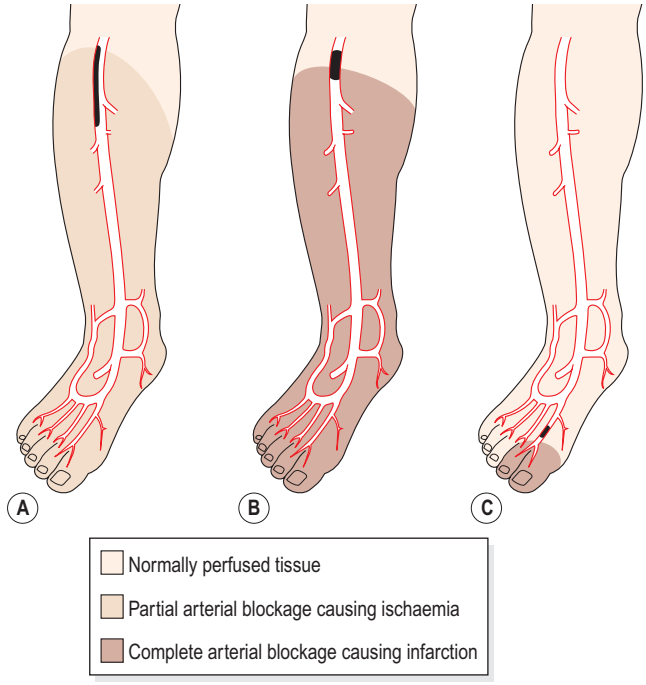


Figure 5.50 Ischaemia and infarction. A. Partial blockage but normal perfusion. **B, C.** Complete blockage causes distal tissue ischaemia and infarction, dependent on the location of the blockage.

Blood vessel pathology

Learning outcomes

After studying this section, you should be able to:

- discuss the main causes, effects and complications of arterial disease, including atheroma, arteriosclerosis and aneurysm
- discuss the underlying abnormality in varicose veins
- list the predisposing factors and the common sites of occurrence of varicose veins
- describe the main tumours that affect blood vessels.

Atheroma

Pathological changes

Atheromatous plaques are patchy changes that develop in the tunica intima of large and medium-sized arteries. Initial changes show a fatty streak in the artery wall. Mature plaques consist of accumulations of cholesterol and other lipids, excess smooth muscle and fat-filled monocytes (foam cells). The plaque is covered with a rough fibrous cap. As plaques grow and thicken they spread along the artery wall and protrude into the lumen. Eventually the whole thickness of the wall and long

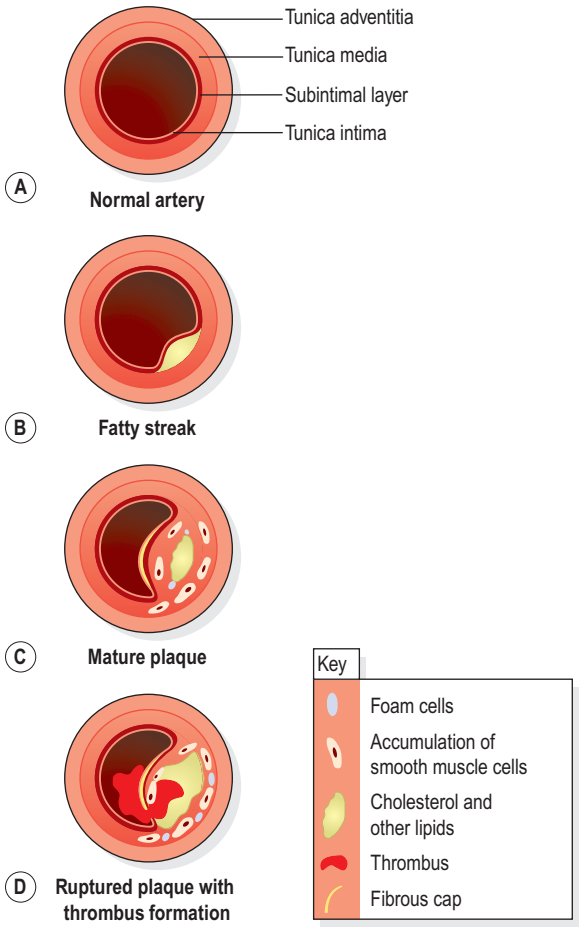


Figure 5.51 Stages in the formation of an atheromatous plaque.

sections of the vessel may be affected (Fig. 5.51). Plaques may rupture, exposing subintimal materials to the blood. This may cause thrombosis and vasospasm and will compromise blood flow.

Arteries most commonly involved are those in the heart, brain, kidneys, small intestine and lower limbs.

Causes of atheroma

The origin of atheromatous plaques is uncertain. *Fatty streaks* present in artery walls of infants are usually absorbed but their incomplete absorption may be the origin of atheromatous plaques in later life.

Atherosclerosis (the presence of plaques) is considered to be a disease of older people because it is usually in these age groups that clinical signs appear. Plaques, however, start to form in childhood in developed countries.

The incidence of atheroma is widespread in developed countries. Why atheromatous plaques develop is not clearly understood, but the predisposing factors appear to exert their effects over a long period. This may mean that the development of atheroma can be delayed or even arrested by a change in lifestyle (Box 5.4).

Box 5.4 Predisposing factors in atherosclerosis

(Modifiable factors are shown in green.)

- Heredity – family history
- Obesity
- Gender – males are more susceptible than females, until after the female menopause
- Diet – high in refined carbohydrates and/or saturated fats and cholesterol
- Increasing age
- Smoking cigarettes
- Diabetes mellitus
- Excessive emotional stress
- Hypertension
- Sedentary lifestyle
- Hyperlipidaemia, especially high levels of LDL (p. 227)
- Excessive alcohol consumption

Effects of atheroma 5.13

Atheromatous plaques may cause partial or complete obstruction of an artery (Fig. 5.50). The blockage may be complicated by clot formation. The consequences of this depend on the site and size of the artery involved and the extent of collateral circulation.

Narrowing of an artery

The tissues distal to the narrow point become ischaemic. The cells may receive enough blood to meet their minimum needs, but not enough to cope with an increase in metabolic rate, e.g. when muscle activity is increased. This causes acute cramp-like ischaemic pain, which disappears when exertion stops. Cardiac muscle and skeletal muscles of the lower limb are most commonly affected. Ischaemic pain in the heart is called *angina pectoris* (p. 127), and in the lower limbs, *intermittent claudication*.

Occlusion of an artery

When an artery is completely blocked, the tissues it supplies rapidly degenerate (ischaemia), which leads to infarction (p. 120). If a major artery supplying a large amount of tissue is affected, the consequences are likely to be more severe than if the obstruction occurs in a minor vessel. If the tissue is well provided with a collateral circulation (such as the circulus arteriosus provides in the brain), tissue damage is less than if there are few collateral vessels (which may be the case in the heart).

When a coronary artery is occluded *myocardial infarction* (p. 127) occurs. Occlusion of arteries in the brain causes cerebral ischaemia and this leads to *cerebral infarction* (stroke, p. 181).

Complications of atheroma

Thrombosis and infarction (p. 120)

If the fibrous cap overlying a plaque breaks down, platelets are activated by the damaged cells and an

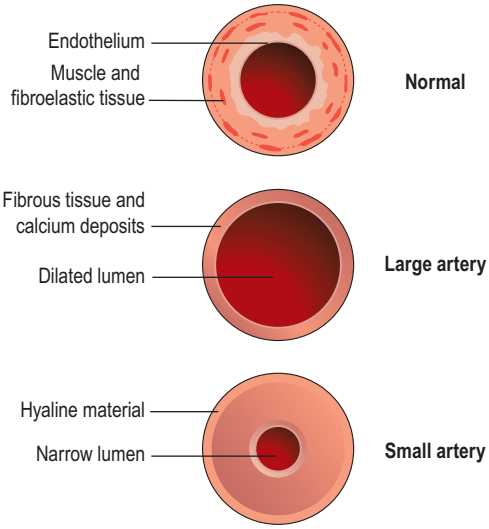


Figure 5.52 Arteriosclerotic arteries.

intravascular blood clot forms (thrombosis), blocking the artery and causing ischaemia and infarction. Emboli may break off, travel in the bloodstream and lodge in small arteries distal to the clot, causing small infarcts.

Haemorrhage

Plaques may become calcified, making the artery brittle, rigid and more prone to aneurysm formation, increasing the risk of rupture and haemorrhage.

Aneurysm

When the arterial wall is weakened by spread of the plaque between the layers of tissue, a local dilation (aneurysm) may develop (see below). This may lead to thrombosis and embolism, or the aneurysm may rupture causing severe haemorrhage. The most common sites affected by atheroma are the aorta and the abdominal and pelvic arteries.

Arteriosclerosis

This is a progressive degeneration of arterial walls, associated with ageing and accompanied by hypertension.

In large and medium-sized arteries, the tunica media is infiltrated with fibrous tissue and calcium. This causes the vessels to become dilated, inelastic and tortuous (Fig. 5.52). Loss of elasticity increases systolic blood pressure, and the *pulse pressure* (the difference between systolic and diastolic pressure).

When small arteries (arterioles) are involved, their lumen is narrowed because of a deposition of a substance called *hyaline material*, which reduces the elasticity of the vessel wall. Because arterioles control peripheral resistance (p. 84), this narrowing increases peripheral resistance and blood pressure. Damage to small vessels has a disproportionate effect on blood flow, leading to ischaemia of tissues supplied by affected arteries. In the limbs, the resultant ischaemia predisposes to gangrene, which is particularly serious in people with diabetes mellitus. If arteries supplying the brain are affected, cerebral ischaemia can result in progressive deterioration of higher order functions (p. 181).

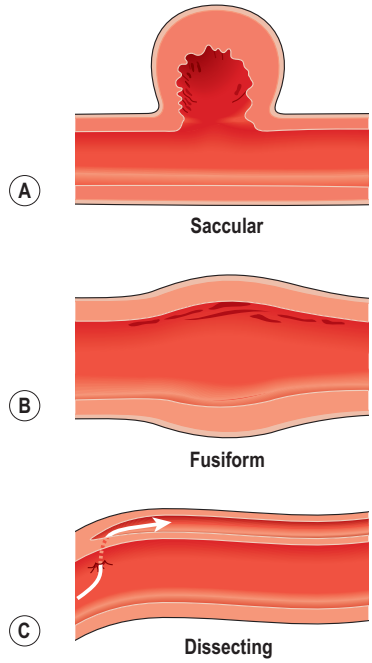


Figure 5.53 Types of aneurysm. A. Saccular. **B.** Fusiform. **C.** Dissecting.

Aneurysms

Aneurysms are abnormal local dilations of arteries, which vary considerably in size (Fig. 5.53). Predisposing factors include atheroma, hypertension and defective formation of collagen in the arterial wall.

If an aneurysm ruptures, haemorrhage follows, the consequences of which depend on the site and extent of the bleed. Rupture of the aorta is likely to be fatal, while bleeding into the subarachnoid space can also cause death, or permanent disability. Bleeding in the brain can cause symptoms of stroke. An aneurysm damages the blood vessel endothelium, making it rougher than usual, which increases the risk of clot formation. Clots may block circulation locally, or elsewhere if they travel in the bloodstream as emboli. In addition, the swelling associated with the distended artery can cause pressure on local structures such as other blood vessels, nerves or organs.

Types of aneurysm

Saccular aneurysms (Fig. 5.53A) bulge out on one side of the artery. When they occur in the relatively thin-walled arteries of the circulus arteriosus (circle of Willis, p. 105)

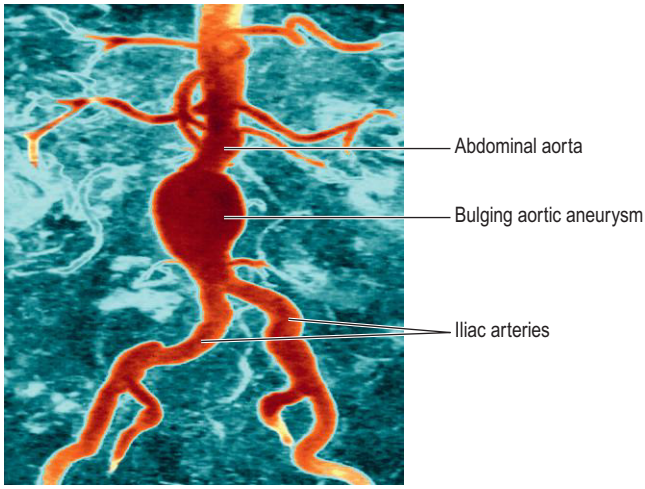


Figure 5.54 Abdominal aortic aneurysm.

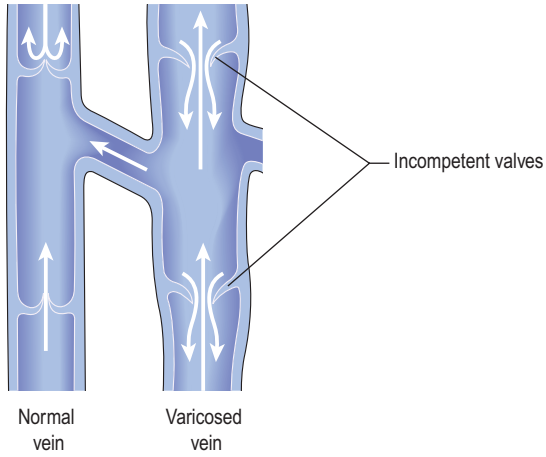


Figure 5.55 Anastomatic connection between superficial and varicosed vein (right) and deeper unaffected vein (left).

in the brain they are sometimes called 'berry' aneurysms. They may be congenital, or be associated with defective collagen production or with atheroma.

Fusiform or spindle-shaped distensions (Fig. 5.53B) occur mainly in the abdominal aorta. They are usually associated with atheroma.

Dissecting aneurysms (Fig. 5.53C) occur mainly in the arch of the aorta. They are caused by infiltration of blood between the endothelium and tunica media, beginning at a site of endothelial damage.

Figure 5.54 shows the bulging abdominal aortic wall caused by an aneurysm.

Venous thrombosis

The risk factors predisposing to a clot developing within a vein are discussed on page 119.

Venous thrombosis may be *superficial thrombophlebitis*, which usually resolves spontaneously, or *deep vein thrombosis*.

Superficial thrombophlebitis

If a thrombus forms in a superficial vein, the tissue around the affected vein becomes inflamed, red and painful. The most common causes are intravenous infusion and varicosities in the saphenous vein.

Deep vein thrombosis (DVT)

DVT usually affects the lower limb, pelvic or iliac veins, but occasionally the upper limb veins. It may be accompanied by local pain and swelling, but is often asymptomatic. Risk factors for DVT include varicose veins, surgery, pregnancy and prolonged immobility, e.g. long journeys with restricted leg room ('economy class syndrome'). It carries a significant risk of death (often from pulmonary embolism (p. 120) if a clot fragment travels to the lungs).

Varicosed veins

Blood pooling in a vein stretches and damages its soft walls and the vein becomes inelastic, dilated and coiled. Generally, superficial veins with little support are involved. The valves then cannot close properly because the vein is distended, and pooling and engorgement get worse. Venous return is maintained because superficial veins are usually connected into the network of deeper veins, which are better supported by surrounding tissues and less likely to become varicosed (Fig. 5.55).

Sites and effects of varicose veins

Varicose veins of the legs

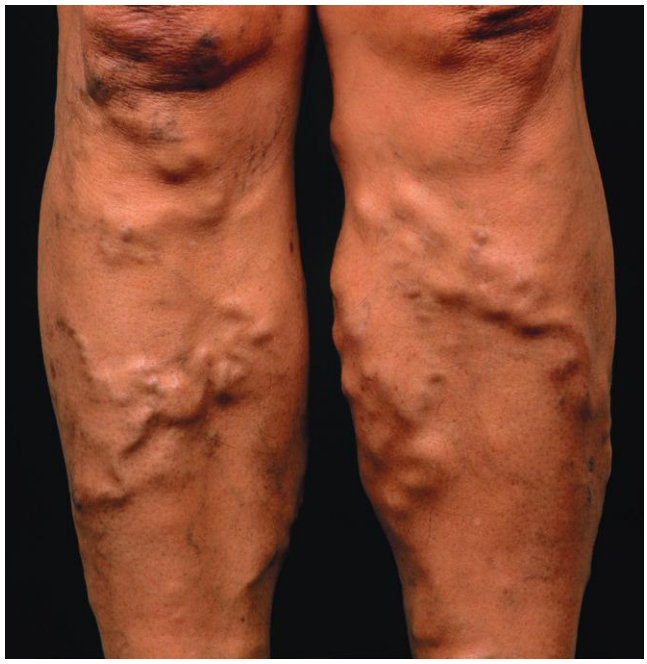
Blood in the veins of the leg is constantly subject to gravity, which can lead to sluggish venous return and accumulation of blood in these veins. If the valves become incompetent, pooling gets worse, and the leg veins become chronically dilated, twisted and lengthened. The superficial veins are more prone to this than deeper ones, because there is less support from surrounding tissues such as muscle, and the varicose veins become clearly visible (Fig. 5.56A). The great and small saphenous veins and the anterior tibial veins are most commonly affected, causing aching and fatigue of the legs, especially during long periods of standing. These dilated, inelastic veins rupture easily if injured, and haemorrhage occurs.

The skin over a varicose vein may become poorly nourished due to stasis of blood, leading to *varicose ulcer*, usually on the medial aspects of the leg just above the ankle.

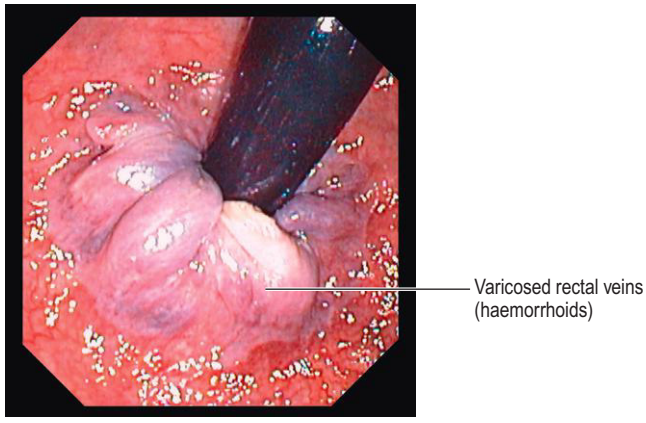
Risk factors include increasing age, obesity, pregnancy, standing for long periods, wearing constricting clothing, family history and female gender.

Haemorrhoids

Sustained pressure on distended veins at the junction of the rectum and anus leads to increased venous pressure,



A



B

Figure 5.56 Varicosed veins. A. Of the leg. **B.** In the rectum (haemorrhoids).

valvular incompetence and the development of haemorrhoids (piles; Fig. 5.56B). The most common causes are chronic constipation, and the increased pressure in the pelvis towards the end of pregnancy. Slight bleeding may occur each time stools are passed and, in time, may cause anaemia. Severe haemorrhage is rare.

Scrotal varicocele

Each spermatic cord is surrounded by a plexus of veins that may become varicosed, especially in men whose work involves standing for long periods. If the varicocele is bilateral, the increased temperature due to venous congestion may depress spermatogenesis and cause infertility.

Oesophageal varices

Raised pressure in the lower oesophageal veins can rupture them, leading to a potentially fatal haemorrhage (p. 321).

Tumours of blood and lymph vessels

Angiomas

Angiomas are benign tumours of either blood vessels (haemangiomas) or lymph vessels (lymphangiomas). The latter rarely occur, so angioma is usually taken to mean haemangioma.

Haemangiomas. These are not true tumours, but are sufficiently similar to be classified as such. They consist of an excessive growth of blood vessels arranged in an uncharacteristic manner and interspersed with collagen fibres.

Capillary haemangiomas. Excess capillary growth interspersed with collagen in a localised area makes a dense, plexus-like network of tissue. Each haemangioma is supplied by only one blood vessel and if it thromboses, the haemangioma atrophies and disappears.

They are usually present at birth and are seen as a purple or red mole or birthmark. They may be quite small at birth but grow at an alarming rate in the first few months, keeping pace with the growth of the child. After 1–3 years, atrophy may begin, and after 5 years about 80% have disappeared.

Oedema

Learning outcomes

After studying this section, you should be able to:

- define the term oedema
- describe the main causes of oedema
- relate the causes of oedema to relevant clinical problems
- explain the causes and consequences of excess fluid collecting in body cavities.

In oedema, excess tissue fluid accumulates, causing swelling. It may occur either in superficial tissues or deeper organs.

Sites of oedema

Oedema of the superficial tissues causes *pitting*, i.e. an indentation remains after firm finger pressure has been applied. Oedema develops at different sites depending on body position and gravity. When standing or sitting, the

oedema develops in the lower limbs, beginning in the feet and ankles. Patients on bedrest tend to develop oedema in the sacral area. This is called *dependent oedema*.

In *pulmonary oedema*, venous congestion in the lungs or increased pulmonary vessel permeability results in accumulation of fluid in the tissue spaces and in the alveoli. This reduces the area available for gaseous exchange and results in *dyspnoea* (breathlessness), cyanosis and coughing up (expectoration) of frothy sputum. The most common causes of pulmonary oedema are cardiac failure, inflammation or irritation of the lungs and excessive infusion of intravenous fluids.

Causes of oedema

Fluid accumulates in the tissues when some aspect of normal capillary fluid dynamics (Fig. 5.57A and see also p. 85) is deranged.

Increased venous hydrostatic (blood) pressure

Congestion of the venous circulation increases venous hydrostatic pressure, reducing the net effect of osmotic pressure that draws fluid back into the capillary at the venous end. Excess fluid then remains in the tissues. This may be caused by heart failure, kidney disease or compression of a limb due to prolonged sitting or tight clothes.

Decreased plasma osmotic pressure

When plasma protein levels fall, less fluid returns to the circulation at the venous end of the capillary (Fig. 5.57B). Causes include excessive protein loss in kidney disease (p. 351), and reduced plasma protein levels caused by, for example, liver failure or a protein-deficient diet.

Impaired lymphatic drainage

Some fluid returns to the circulation via the lymphatic system and when flow is impaired, oedema develops (Fig. 5.57C). Causes include malignancy that blocks lymph drainage, surgical removal of lymph nodes or lymph node destruction by chronic inflammation.

Increased small-vessel permeability

In inflammation (p. 377), chemical mediators increase small vessel permeability in the affected area. Plasma proteins then leave the circulation (Fig. 5.57D) and the resultant increased tissue osmotic pressure draws fluid into the area causing swelling of the affected tissue. This type of oedema also occurs in allergic reactions (p. 385), e.g. anaphylaxis, asthma or hay fever.

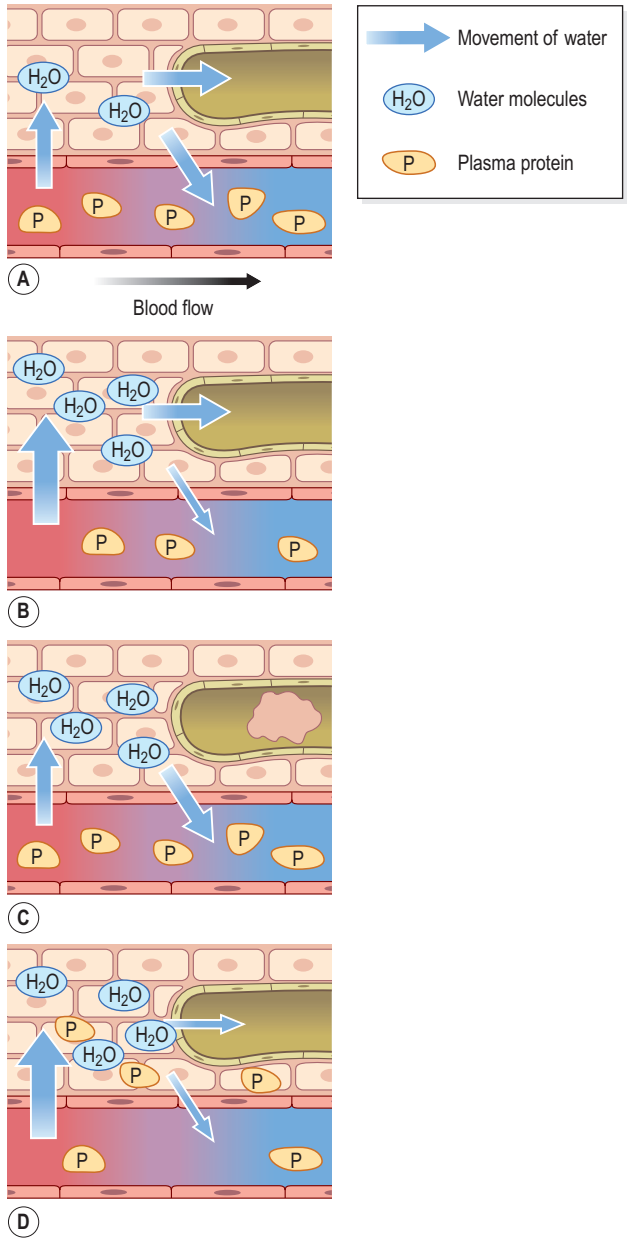


Figure 5.57 Capillary fluid dynamics. A. Normal. **B.** Effect of reduced plasma proteins. **C.** Effect of impaired lymphatic drainage. **D.** Effect of increased capillary permeability. Arrows indicate direction of movement of water.

Effusions and ascites

Abnormal accumulation of excess fluid in body spaces, e.g. the pericardial sac or a joint space, is often associated with inflammatory, infective or obstructive conditions and is generally referred to as an *effusion*.

Pleural effusion. This is excess serous fluid in the pleural cavity. This is usually due to infection or inflammation of the pleura (p. 250), or to left ventricular failure, which increases pressure in the pulmonary circulation because the left ventricle is not able to pump out all the blood returning to it from the lungs.

Ascites. This is accumulation of excess fluid in the peritoneal cavity. The most common causes include liver failure (when plasma protein synthesis is reduced), obstruction of abdominal lymph nodes draining the peritoneal cavity, or inflammatory conditions. This includes malignant disease, because many tumours release pro-inflammatory mediators.

Diseases of the heart

Learning outcomes

After studying this section, you should be able to:

- describe the consequences of failure of either or both sides of the heart
- the compensatory mechanisms that occur in heart failure
- explain the causes and consequences of faulty heart valve function
- define the term ischaemic heart disease
- discuss the main conditions associated with ischaemic heart disease
- describe rheumatic heart disease and its effects on cardiac function
- explain the underlying pathophysiology of pericarditis
- describe, with reference to standard ECG trace, the main cardiac arrhythmias
- describe the principal congenital cardiac abnormalities.

Heart (cardiac) failure

The heart is described as *failing* when the cardiac output is unable to circulate sufficient blood to meet the needs of the body. In mild cases, cardiac output is adequate at rest and becomes inadequate only when tissue needs are increased, e.g. in exercise. Heart failure may affect either side of the heart, but since both sides of the heart are part of one circuit, when one half of the pump begins to fail it frequently leads to increased strain on, and eventual failure of, the other side. The main clinical manifestations depend on which side of the heart is most affected. Left ventricular failure is more common than right, because of the greater workload of the left ventricle.

Compensatory mechanisms in heart failure

In acute heart failure, the body has little time to make compensatory changes, but if the heart fails over a period of time the following changes are likely to occur in an attempt to maintain cardiac output and tissue perfusion, especially of vital organs:

- the cardiac muscle mass increases (hypertrophy), which makes the walls of the chambers thicker
- the heart chambers enlarge
- decreased renal blood flow activates the renin–angiotensin–aldosterone system (p. 225), which leads to salt and water retention. This increases blood volume and cardiac workload. The direct vasoconstrictor action of angiotensin 2 increases peripheral resistance and puts further strain on the failing heart.

Acute heart failure

If heart failure occurs abruptly, the supply of oxygenated blood to body tissues is suddenly and catastrophically reduced and there is no time for significant compensation to take place. Death may follow if the brain's vital centres are starved of oxygen. Even if the acute phase is survived, myocardial damage may lead to chronic heart failure. Common causes include:

- myocardial infarction (p. 127)
- pulmonary embolism, blocking blood flow through the pulmonary circulation – the heart fails if it cannot pump hard enough to overcome the obstruction
- life-threatening cardiac arrhythmia, when the pumping action of the heart is badly impaired or stopped
- rupture of a heart chamber or valve cusp; both greatly increase the cardiac effort required to maintain adequate output
- severe malignant hypertension, which greatly increases resistance to blood flow.

Chronic heart failure

This develops gradually and in the early stages there may be no symptoms because compensatory changes occur as described above. When further compensation is not possible, myocardial function gradually declines. Underlying causes include degenerative heart changes with advancing age, and many chronic conditions, e.g. anaemia, lung disease, hypertension or cardiac disease.

Right-sided (congestive cardiac) failure

The right ventricle fails when the pressure developed within it by the contracting myocardium is insufficient to push blood through the lungs.

When compensation has reached its limit, and the ventricle can no longer empty completely, the right atrium and venae cavae become congested with blood and this is followed by congestion throughout the venous system. The organs affected first are the liver, spleen and kidneys. *Oedema* (p. 124) of the limbs and *ascites* (excess fluid in the peritoneal cavity) usually follow.

This problem may be caused by increased vascular resistance in the lungs or weakness of the myocardium.

Resistance to blood flow through the lungs. When this is increased the right ventricle has more work to do. The two commonest causes are pulmonary embolism and left ventricular failure, when the pulmonary circulation is congested because the left ventricle is not clearing all the blood flowing into it.

Weakness of the myocardium. This is caused by myocardial damage following ischaemia or infarction.

Left-sided (left ventricular) failure

This occurs when the pressure developed in the left ventricle by the contracting myocardium is not enough to force blood into the aorta and the ventricle cannot then pump out all the blood it receives. Causes include ischaemic heart disease, which reduces the efficiency of the myocardium, and hypertension, when the heart's workload is increased because of raised systemic resistance. Disease of the mitral (left atrioventricular) and/or aortic valves may prevent efficient emptying of the heart chambers, so that myocardial workload is increased.

Failure of the left ventricle leads to dilation of the atrium and an increase in pulmonary blood pressure. This is followed by a rise in the blood pressure in the right side of the heart and eventually systemic venous congestion.

Exercise tolerance becomes progressively reduced as the condition worsens and is accompanied by cough caused by pulmonary oedema. The sufferer is easily tired and is likely to have poorly perfused peripheral tissues and low blood pressure.

Congestion in the lungs leads to pulmonary oedema and dyspnoea, often most severe at night. This paroxysmal *nocturnal dyspnoea* may be due to raised blood volume as fluid from peripheral oedema is reabsorbed when the patient slips down in bed during sleep.

Disorders of heart valves 5.14

The heart valves prevent backflow of blood in the heart during the cardiac cycle. The mitral and aortic valves are subject to greater pressures than those on the right side and are therefore more susceptible to damage.

Distinctive heart sounds arise when the valves close during the cardiac cycle (p. 93). Damaged valves generate abnormal heart sounds called *murmurs*. A severe valve disorder causes heart failure. The most common causes of valve defects are rheumatic fever, fibrosis following inflammation and congenital abnormalities.

Stenosis

This is the narrowing of a valve opening, impeding blood flow through the valve. It occurs when inflammation and encrustations roughen the edges of the cusps so that they stick together, narrowing the valve opening. When healing occurs, fibrous tissue is formed which shrinks as it ages, increasing the stenosis and leading to incompetence.

Incompetence

Sometimes called *regurgitation*, this is a functional defect caused by failure of a valve to close completely, allowing blood to flow backwards.

Ischaemic heart disease

This is due to ischaemia, usually caused by atheromatous plaques narrowing or occluding of one or more branches of the coronary arteries. Occlusion may be by plaques alone, or plaques complicated by thrombosis. The overall effect depends on the size of the coronary artery involved and whether it is only narrowed or completely blocked. Narrowing of an artery leads to angina pectoris, and occlusion to myocardial infarction.

When atheroma develops slowly, a *collateral arterial blood supply* may have time to develop and effectively supplement or replace the original. This consists of the dilation of normally occurring anastomotic arteries joining adjacent arteries. When sudden severe narrowing or occlusion of an artery occurs, the anastomotic arteries dilate but may not be able to supply enough blood to meet myocardial needs.

Angina pectoris

This is sometimes called *angina of effort* because the increased cardiac output required during extra physical effort causes severe chest pain, which may also radiate to the arms, neck and jaw. Other precipitating factors for angina include cold weather and emotional states.

A narrowed coronary artery may supply sufficient blood to the myocardium to meet its needs during rest or moderate exercise but not when greatly increased cardiac output is needed, e.g. walking may be tolerated but not running. The thick, inflexible atheromatous artery wall is unable to dilate to allow for the increased blood flow needed by the more active myocardium, which then becomes ischaemic. In the early stages of angina, the chest pain stops when the cardiac output returns to its resting level soon after the extra effort stops.

Myocardial infarction

The myocardium may infarct (p. 120) when a branch of a coronary artery is blocked. The commonest cause is an atheromatous plaque complicated by thrombosis. The damage is permanent because cardiac muscle cannot regenerate, and the dead muscle is replaced with non-functional fibrous tissue. Speedy restoration of blood flow through the blocked artery using clot-dissolving (thrombolytic) drugs can greatly reduce the extent of the permanent damage and improve prognosis, but treatment must be started within a few hours of the infarction occurring. The effects and complications are greatest when the left ventricle is involved.

Myocardial infarction is usually accompanied by very severe crushing chest pain behind the sternum which, unlike angina pectoris, continues even when the individual is at rest. It is a significant cause of death in the developed world.

Complications

These may be fatal and include:

- severe and sometimes life-threatening arrhythmias, especially *ventricular fibrillation* (p. 129), due to disruption of the cardiac conducting system
- acute heart failure (p. 126), caused by impaired contraction of the damaged myocardium and, in severe cases, cardiogenic shock
- rupture of a ventricle wall, usually within 2 weeks of the original episode
- pulmonary or cerebral embolism originating from a mural clot within a ventricle, i.e. a clot that forms inside the heart over the infarct
- pericarditis
- angina pectoris (p. 127)
- recurrence.

Rheumatic heart disease

Rheumatic fever is an inflammatory illness that sometimes follows streptococcal throat infections, most commonly in children and young adults. It is an autoimmune disorder; the antibodies produced to combat the original infection damage connective tissues, including the heart, joints (p. 432) and skin.

Death rarely occurs in the acute phase, but after recovery there may be permanent damage to the heart valves, eventually leading to disability and possibly cardiac failure.

Acute rheumatic heart disease. In the acute stages, all layers of the heart wall are inflamed (*pancarditis*, 'pan-' meaning 'all of'). The heart valves, especially the mitral valve, are frequently affected. Fibrotic nodules develop on their cusps, which shrink as they age, distorting the cusp and causing stenosis and incompetence of the valve. The inflamed myocardium can fail, leading to signs of heart failure, including tachycardia, breathlessness and cardiac enlargement. Inflammation of the pericardium can lead to friction within the pericardial cavity as the heart beats, pain behind the sternum and interference with the pumping action of the heart. Permanent fibrotic damage may fuse the visceral and parietal layers of the serous pericardium together, restricting the heart's action.

Chronic rheumatic heart disease. Inflamed tissue becomes fibrous as it heals, and this fibrous tissue interferes with the action of the myocardium and the heart valves. At least half of acute cases develop chronic valvular incompetence following recovery. The great majority of these patients have mitral valve damage, but the aortic valve is frequently affected too. Chronic fibrotic changes in the pericardium and myocardium cause heart failure.

Sometimes rheumatic valvular disease presents with no history of acute rheumatic fever or streptococcal infection.

Infective endocarditis

Pathogenic organisms (usually bacteria or fungi) in the blood may colonise any part of the endocardium, but the most common sites are on or near the heart valves and round the margins of congenital heart defects. These areas are susceptible to infection because they are exposed to fast-flowing blood that may cause mild trauma. This illness, which may be acute or subacute, is serious and sometimes fatal without treatment.

The main predisposing factors are bacteraemia, depressed immune response and heart abnormalities.

Bacteraemia

Microbes in the bloodstream, if not destroyed by phagocytes or antibodies, tend to adhere to platelets and form tiny infected emboli. Inside the heart, the emboli are most likely to settle on already damaged endocardium. Vegetations consisting of platelets and fibrin surround the microbes and seem to protect them from normal body defences and antibiotics. Because of this, infection may be caused by a wide range of bacteria, including some that do not normally cause clinical infection. They normally originate from the skin or the mouth.

Depressed immune response

This enables low-virulence bacteria, viruses, yeasts and fungi to become established and cause infection. These are organisms always present in the body and the environment. Depression of the immune systems may be caused by HIV infection, malignant disease, cytotoxic drugs, radiotherapy or steroid therapy.

Heart abnormalities

The sites most commonly infected are already abnormal in some way. Pathogenic organisms present in the bloodstream cannot adhere to healthy endothelium, but if the endothelial lining of the cardiovascular system is damaged, infection is more likely. Often, the cardiac valves are involved, especially if damaged by rheumatic disease or congenital malformation. Other likely sites of infection include regions of cardiac abnormality, such as ventricular septal defect (p. 130) and patent ductus arteriosus (p. 130). Prosthetic (artificial) valves can also be a focus for infective growths.

Cardiac arrhythmias

The heart rate is normally determined by intrinsic impulses generated in the SA node. The rhythm is determined by the route of impulse transmission through the conducting system. The heart rate is usually measured as the pulse, but to determine the rhythm, an electrocardiogram (ECG) is required (Fig. 5.58A). A *cardiac arrhythmia* is any disorder of heart rate or rhythm, and is the result of abnormal generation or conduction of impulses. The

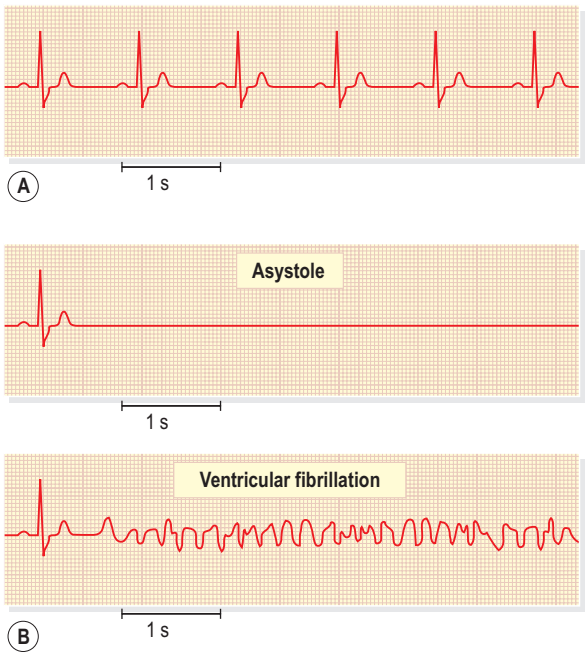


Figure 5.58 ECG traces. A. Normal sinus rhythm. **B.** Life-threatening arrhythmias.

normal cardiac cycle (p. 92) gives rise to *normal sinus rhythm*, which has a rate of between 60 and 100 b.p.m.

Sinus bradycardia. This is normal sinus rhythm below 60 b.p.m. This may occur during sleep and is common in athletes. It is an abnormality when it follows myocardial infarction or accompanies raised intracranial pressure (p. 179).

Sinus tachycardia. This is normal sinus rhythm above 100 b.p.m. when the individual is at rest. This accompanies exercise and anxiety, but is an indicator of some disorders, e.g. fever, hyperthyroidism, some cardiac conditions.

Asystole

This occurs when there is no electrical activity in the ventricles and therefore no cardiac output. The ECG shows a flat line (Fig. 5.58B). Ventricular fibrillation and asystole cause sudden and complete loss of cardiac output, i.e. *cardiac arrest* and death.

Fibrillation

This is the contraction of the cardiac muscle fibres in a disorderly sequence. The chambers do not contract as a coordinated unit and the pumping action is disrupted.

In *atrial fibrillation* (AF), contraction of the atria is uncoordinated and rapid, pumping is ineffective and stimulation of the AV node is disorderly. AF is very common, especially in older adults. It may be asymptomatic, because although atrial function is disordered, most ventricular filling happens passively and atrial contraction only tops it up, so cardiac output is maintained. However, common symptoms include unpleasant palpitations, breathlessness and fatigue. The pulse is irregular and there are no discernible P waves on the ECG. Often the cause is unknown but AF can develop as a result of many forms of heart disease, thyrotoxicosis (p. 231), alcoholism and lung disease.

Ventricular fibrillation is a medical emergency that will swiftly lead to death if untreated, because the chaotic electrical activity within the ventricular walls cannot coordinate effective pumping action (cardiac arrest).

Blood is not pumped from the heart into either the pulmonary or the systemic circulation. No pulses can be felt; consciousness is lost and breathing stops. The ECG shows an irregular chaotic trace with no recognisable wave pattern (Fig. 5.58B).

Heart block

Heart block occurs when normal impulse transmission is blocked or impaired. A common form involves obstruction of impulse transmission through the AV node, but (less commonly) conducting tissue in the atria or ventricles can also be affected. When the AV node is involved, the delay between atrial and ventricular contraction is increased. The severity depends on the extent of loss of stimulation of the AV node.

In *complete heart block*, ventricular contraction is entirely independent of impulses initiated by the SA node. Freed from the normal pacing action of the SA node, the ventricles are driven by impulses generated by the pacemaker activity of the AV node, resulting in slow, regular ventricular contractions and a heart rate of about 30 to 40 b.p.m. In this state the heart is unable to respond quickly to a sudden increase in demand by, for example, muscular exercise. The most common causes are:

- acute ischaemic heart disease
- myocardial fibrosis following repeated infarctions or myocarditis
- drugs used to treat heart disease, e.g. digitalis, propranolol.

When heart block develops gradually there is some degree of adjustment in the body to reduced cardiac output but, if progressive, it eventually leads to death from cardiac failure and cerebral anoxia.

Congenital abnormalities

Abnormalities in the heart and great vessels at birth may be due to intrauterine developmental errors or to the failure of the heart and blood vessels to adapt to extrauterine life. Sometimes, there are no symptoms in early life

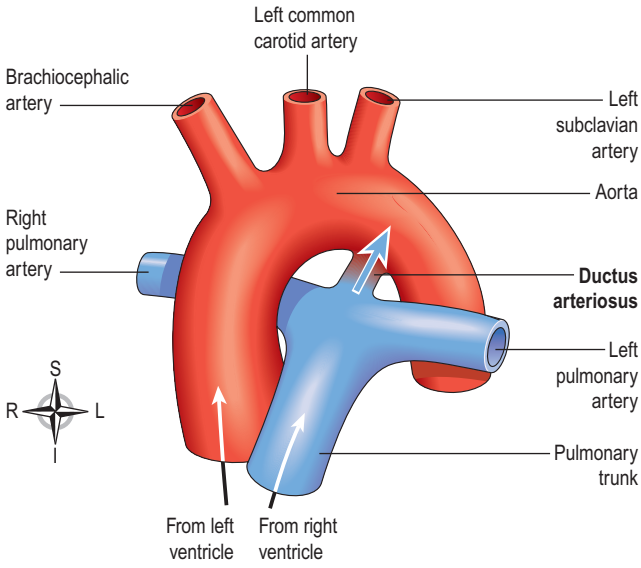


Figure 5.59 The ductus arteriosus in the fetus. The arrow indicates the direction of flow of blood from the pulmonary circulation into the aorta.

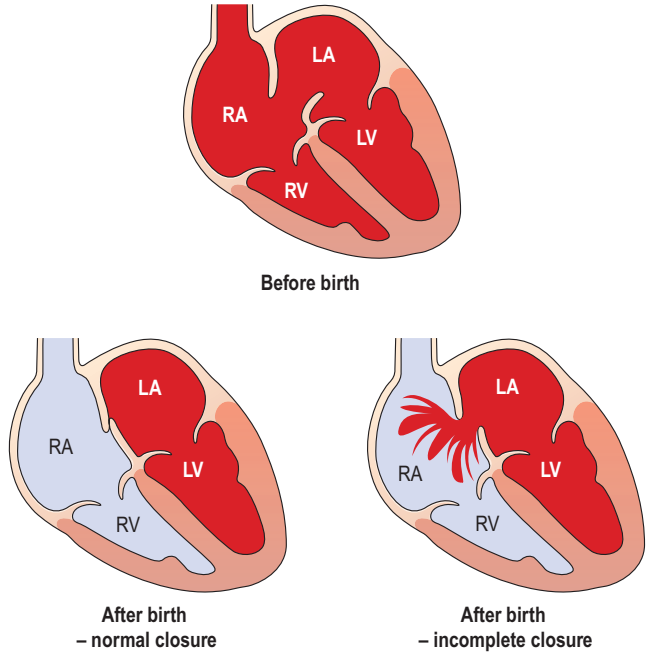


Figure 5.60 Atrioseptal valve: normal and incomplete closure after birth.

and the abnormality is recognised only when complications appear.

Patent ductus arteriosus

In the fetus, the ductus arteriosis (p. 116) bypasses the non-functional lungs (Fig. 5.59). At birth, when the pulmonary circulation is established, the ductus arteriosus should close completely. If it remains patent, blood regurgitates from the aorta to the pulmonary artery where the pressure is lower, reducing the volume entering the systemic circulation and increasing the volume of blood in the pulmonary circulation. This leads to pulmonary congestion and eventually cardiac failure.

Atrial septal defect

This is commonly known as 'hole in the heart'. After birth, when the pulmonary circulation is established and the pressure in the left atrium exceeds that in the right atrium, the atrioseptal valve closes. Later the closure becomes permanent due to fibrosis (Fig. 5.60).

When the membranes do not overlap, an opening between the atria remains patent after birth. In many cases it is too small to cause symptoms in early life but they may appear later. In severe cases blood flows back to the right atrium from the left. This increases the right ventricular and pulmonary pressure, causing hypertrophy of the myocardium and eventually cardiac failure. As pressure in the right atrium rises, blood flow through the defect may be reversed, but this is not an improvement because deoxygenated blood gains access to the general circulation.

Coarctation of the aorta

The most common site of coarctation (narrowing) of the aorta is between the left subclavian artery and ductus arteriosus. This leads to hypertension in the upper body (which is supplied by arteries arising from the aorta proximal to the narrowing) because increased force of contraction of the heart is needed to push the blood through the coarctation. There may be systemic hypotension.

Fallot's tetralogy

This is a characteristic combination of four congenital cardiac abnormalities, which causes cyanosis, growth retardation and exercise intolerance in babies and young children. The four abnormalities are:

- stenosis of the pulmonary artery at its point of origin, which increases right ventricular workload
- ventricular septal defect, i.e. an abnormal communicating hole between the two ventricles, just below the atrioventricular valves
- aortic misplacement, i.e. the origin of the aorta is displaced to the right so that it is immediately above the septal defect
- right ventricular hypertrophy to counteract the pulmonary stenosis.

Cardiac function is inadequate to meet the needs of the growing child; surgical correction carries a good prognosis.

Disorders of blood pressure

Learning outcomes

After studying this section, you should be able to:

- explain the term hypertension
- define essential and secondary hypertension and list the main causes of the latter
- discuss the effects of prolonged hypertension on the body, including elevated blood pressure in the lungs
- describe the term hypotension.

Hypertension

The term hypertension is used to describe a level of blood pressure that, taking all other cardiovascular risk factors into account, would benefit the patient if reduced. Blood pressure readings where systolic and diastolic values fall below 130/85 respectively are considered normal. Readings that indicate hypertension are listed in Table 5.2. Blood pressure tends to rise naturally with age. Arteriosclerosis (p. 122) may contribute to this, but is not the only factor.

Hypertension is classified as *essential* (primary, idiopathic) or *secondary* to other diseases. Irrespective of the cause, hypertension commonly affects the kidneys (p. 352).

Essential hypertension

Essential hypertension (hypertension of unknown cause) is very common in the Western world and accounts for 95% of all cases of hypertension. Treatment aims to prevent complications, which can be serious, primarily cardiovascular and renal disease. Sometimes complications, such as heart failure, cerebrovascular accident or myocardial infarction are the first indication of hypertension, but often the condition is symptomless and is only discovered during a routine examination.

Risk factors. Risk factors for hypertension include obesity, diabetes mellitus, family history, cigarette smoking, a sedentary lifestyle and high intakes of salt or alcohol. Stress may increase blood pressure, and there is a well-documented link between low birth weight and incidence of hypertension in later life.

Table 5.2 Hypertension: indicative blood pressure readings. British Hypertension Society/NICE guidelines, 2011

Grade	Systolic reading (mmHg)	Diastolic reading (mmHg)
1, mild	140–59	90–99
2, moderate	160–179	100–109
3, severe	≥180	≥ 110

Malignant (accelerated) hypertension

This is a rapid and aggressive acceleration of hypertensive disease. Diastolic pressure in excess of 120 mmHg is common. The effects are serious and quickly become apparent, e.g. haemorrhages into the retina, papilloedema (oedema around the optic disc), encephalopathy (cerebral oedema) and progressive renal disease, leading to cardiac failure.

Secondary hypertension

Hypertension resulting from other diseases accounts for 5% of all cases.

Some causes of secondary hypertension are listed in Box 5.5.

Effects and complications of hypertension

The effects of long-standing and progressively rising blood pressure are serious. Hypertension predisposes to atherosclerosis and has specific effects on particular organs.

Heart. The rate and force of cardiac contraction are increased to maintain the cardiac output against a sustained rise in arterial pressure. The left ventricle hypertrophies and begins to fail when compensation has reached its limit. This is followed by back pressure and accumulation of blood in the lungs (pulmonary congestion), hypertrophy of the right ventricle and eventually

Box 5.5 Some causes of secondary hypertension

Causes of secondary hypertension

- Kidney disease (p. 352)
- Adrenal gland disorders
  - excessive steroid secretion (Conn's syndrome, Cushing's syndrome, p. 233)
  - excessive adrenaline secretion, e.g. phaeochromocytoma (p. 235)
- Thyrotoxicosis (p. 231)
- Stricture of the aorta
- Alcohol
- Obesity
- Pregnancy
- Drug treatment, e.g. oral contraceptives containing oestrogen, corticosteroids

to right ventricular failure. Hypertension also predisposes to ischaemic heart disease (p. 127) and aneurysm formation (p. 122).

Brain. Stroke, caused by cerebral haemorrhage, is common, the effects depending on the position and size of the ruptured vessel. When a series of small blood vessels rupture, e.g. microaneurysms, at different times, there is progressive disability. Rupture of a large vessel causes extensive loss of function or death.

Kidneys. Hypertension causes kidney damage. If sustained for only a short time recovery may be complete. Otherwise the kidney damage causes further hypertension owing to activation of the renin–angiotensin–aldosterone system (p. 343), progressive loss of kidney function and kidney failure.

Blood vessels. High blood pressure damages blood vessels. The walls of small arteries become hardened, and in larger arteries, atheroma is accelerated. If other risk factors for vascular disease are present, such as diabetes or smoking, damage is more extensive. The vessel wall may become so badly weakened by these changes that an aneurysm develops, and as the blood vessels become progressively damaged and less elastic, hypertension worsens.

The capillaries of the retina and the kidneys are particularly susceptible to the effects of chronic hypertension, leading to retinal bleeding and reduced renal function.

Pulmonary hypertension

Normally, the pulmonary circulation is a low-pressure system, to prevent fluid being forced out of the pulmonary capillaries into the alveoli. When blood pressure rises, alveoli begin to fill with fluid, which blocks gas exchange. Rising pulmonary blood pressure may result from left-sided heart failure (p. 127), or other problems with left ventricular function, when blood accumulates in the pulmonary circulation because the left ventricle is not pumping efficiently. Lung disease can also increase in pulmonary blood pressure because of destruction of lung capillaries, e.g. in emphysema. Primary pulmonary hypertension, where there is no identifiable cause, is rare.

Hypotension

This usually occurs as a complication of other conditions, such as shock (p. 118) or Addison's disease (p. 235). Low blood pressure leads to inadequate blood supply to the brain. Depending on the cause, unconsciousness may be brief (fainting) or more prolonged, possibly causing death.

Postural hypotension is an abrupt fall in blood pressure on standing up suddenly from a sitting or lying position. It causes dizziness and occasionally syncope (fainting).

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