SECTION TWO

Cardiac structure and function

This section introduces the structure and function of the cardiovascular system. Altered physiology remains a hallmark of many cardiac conditions, which will be described in detail within this text. This section therefore lays the foundation for understanding the disease processes which patients experience, and where nursing can ultimately impact on quality of life.
CHAPTER 4

The applied anatomy and physiology of the cardiovascular system

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The applied anatomy and physiology of the cardiovascular system

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As health-care services advance and change, so does the increasing relevance of the biological sciences in providing the underpinning for cardiac nursing care. This can be clearly seen in practice, with the growth of autonomous nursing roles in primary and secondary care, through to developments in nurse-led clinics and specialist roles to manage acute and chronic cardiac conditions. All cardiac nurses are expected to have a sound underpinning of how the cardiac system functions to allow them to understand and evaluate the application of nursing interventions. This could be with drug administration, the effects of intravenous (IV) fluids and inotropes to augment the cardiac output, through to explaining the variety of congenital heart defects. This latter adult population, who are younger than the traditional cardiac patient, are a growing group within the cardiac specialty due to the success of surgical and interventional procedures.

Coronary heart disease (CHD) itself remains a major cause of mortality and morbidity within the United Kingdom (UK). As it progresses, the failing heart has a unique ability to initiate a variety of compensatory mechanisms to assist and maintain tissue perfusion. However, it is frequently such mechanisms that result in a cycle of worsening heart failure for the patient. Much of non-surgical treatment consists of breaking and manipulating these biological and essentially physiological mechanisms.

To understand the variety of treatments and associated nursing care currently implemented, there is a clear need for a knowledge of the heart’s structure, function and its ability to adapt to failure. In addition to this, in an era of chronic heart disease, patients themselves are being empowered to understand their own illness and aim for greater medication compliance and lifestyle changes. This requires a nursing team with a confident and competent knowledge base regarding the anatomy and physiology of the cardiovascular system.

This chapter explores in detail the structure of the heart and its normal function. Subsequent authors within this text will utilize this knowledge base to explain the process of cardiac assessment, altered physiology and how medical and surgical interventions can assist cardiac function. The reader is encouraged to review all areas of cardiac anatomy and physiology relevant to their practice, but this chapter provides an overview of the major areas related to nursing care.

The cardiovascular system is a sophisticated transport mechanism capable of delivering the required metabolic substrates to tissue and cells. This is in addition to facilitating the removal of undesirable by-products from various organs, for elimination from the body. The blood vessels in this dynamic circulatory network provide the pathways for distributing and collecting the blood-borne substrates. However, the blood can only perform
its many complex roles if it circulates continually and in a pulsatile fashion through the pulmonary and systemic vascular compartments linking the external environment to the tissues and cells. The heart itself generates the required energy for moving the blood through the closed, but selectively permeable, circulatory network.

The shaping of the cardiovascular system in preparation for its complex function takes place in early embryonic life. To this end the formation of the cardiovascular system begins in the first 3 weeks of pregnancy with the establishment of a single heart tube, which gradually evolves into the four-chambered heart. As part of its gradual development and shaping, the heart is connected to the systemic and pulmonary vessels. This makes the cardiovascular system fit for its future dual function, in terms of maintenance of the pulmonary and systemic circulations.

**The anatomy of the adult heart**

**The normal position and shape of the heart**

The normal human heart is cone-shaped and located in the mediastinum, one-third to the right of the sternum and the remaining two-thirds to the left. This is clearly apparent on the normal anteroposterior (AP) chest X-ray. Unusually, the top of the heart is referred to as the base and the pointed lower section as the apex. The base of the heart lies in an oblique position behind the sternum and its apex lies to the left, in the fifth intercostal space in the mid-clavicular line. The normal position of the heart ensures maximal protection from the thoracic cavity and particularly the lungs and diaphragm.

The adult heart is about the size of the person’s fist and varies in weight from 230 to 340 g, a weight which is achieved between the ages of 17 and 20 years of age (Gabella 1995). Considering that mammalian life depends on the continual pumping action of this organ, it is somewhat awesome to realize that it weighs less than 1 lb. Functionally, the heart may be considered as a hollow fibromuscular organ consisting of two highly specific pumps (a left and a right), each of which includes a set of valves. These guide the continuous flow of blood to the pulmonary and the systemic circulations (Fig. 4.1).

**The pathway of blood through the heart**

Deoxygenated blood returns to the right side of the heart from the body via the inferior and superior vena cavae. The blood enters the right atrium, then passes through the tricuspid valve to the right ventricle. From here it passes through the pulmonary artery to the lungs. In the lungs, oxygenation occurs and the blood flows back to the left side of the heart to be pumped around the body. The circuit to and from the lungs is referred to as the pulmonary circuit.

Oxygenated blood returns to the left side of the heart via the pulmonary veins into the left atrium. From there it passes into the left ventricle via the mitral (bicuspid) valve. The term ‘mitral’ is used clinically, whilst the term ‘bicuspid’ is seen in physiology texts. The oxygenated blood then passes from the left ventricle to the body via the aorta. The circuit supplying blood to the body (other than the lungs) is referred to as the systemic circuit.

Each side of the heart consists of an upper chamber, the atrium, and a lower chamber, the ventricle. Thus the heart consists of four chambers (see Fig. 4.2). The right and left sides of the heart are divided by a partition known as the septum. Although the fibromuscular framework and conduction tissue of these pumps are structurally interwoven, each pump is physiologically independent, but always capable of contributing to the pulmonary and systemic circulations. The division of the heart into four chambers produces characteristic external boundaries, which frequently present as deep grooves containing prominent structures such as coronary vessels. An example of such anatomical cardiac demarcations is the atrioventricular groove, which separates the atria from the ventricles and embodies the main trunks of the coronary arteries. Similarly, the interventricular groove corresponds to the interventricular septum, which divides the two ventricular chambers.

**The chambers and valves of the heart**

**The atria**

The atria are thin-walled muscular chambers that form the most anterior aspect of the heart (Drake et al 2005). A fibromuscular septum separates the two atria, forming the distinctive right and left atrium. This septum features a fairly central fibrous oval depression known as the fossa ovalis. According to Gabella (1995), approximately 33% of normal adult hearts have a small slit in the upper margins of the fossa ovalis, a phenomenon which is generally asymptomatic, but may be detected in expectant mothers and in later life.

The atrial myocardium divides into a superficial and a deep layer. The superficial layer spans over and encircles both atria, inserting into the annulus fibrosus. By contrast, the deep layer is independent for each atrial chamber, its fibres run at right angles to the superficial muscle fibres and make up the major muscle mass of the atria. However, the arrangement of both fibres is such that they form distinctive muscular loops, which encircle the vessel inlets to the atria.

The function of the atria is to serve as storage reservoirs for blood returning to the heart from the systemic and pulmonary circulations. This function is particularly significant during ventricular systole, when the forward flow of blood is stopped by the competent...
closure of the atrioventricular valves. In these circumstances the returning blood collects in and distends the atrial cavities. However, the atria can also be compared to the venous network in that they are collapsible when partially filled. This phenomenon may be attributed to the relative thinness of the atrial myocardium. The advantage of this relative compliance is that both chambers will adapt to the volume they hold, distending when they are full or overfilled and reducing in size when venous return is compromised. In addition, there are flap-like protrusions, attached to each atrial chamber. These are known as auricles and contribute to the expelled atrial volume. As the left atrium, in addition to being smaller, is less distensible, the venous return from the lungs will invariably maintain a slightly higher left atrial pressure than that of the right. Finally, the insides of the atrial walls are not smooth, but have ridges of muscle called pectinate muscles.

Atrioventricular (AV) valves

The tricuspid valve is the largest valvular orifice in the adult heart and is situated between the right atrium and the right ventricle. A ring of collagen and fibrous tissue acts as the base for the attachments of the three cusps or leaflets (flaps), which make up this valve. The three cusps are separated by commissures, which are tethered by fan-shaped chordae tendinae of varying length and
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The mitral (bicuspid) valve guards the inlet to the left ventricle from the left atrium. This valve is fixed to an annulus, or a ring of fibrocollagenous tissue. The consistency of this tissue seems important because it allows for major changes in the shape of the annulus and valve during the different stages of the cardiac cycle. In other words, the compliance of this tissue ensures optimal efficiency in valve function. The valve itself is formed by two extensive leaflets, which are joined by commissures. These leaflets are also tethered by chordae tendineae, attached to papillary muscles emerging from the ventricular walls.

The cusps or leaflets of the AV valves hang loosely into the ventricles when the valves are in their open state. This allows blood to flow from the atria to the ventricles. The increase in pressure that occurs when the ventricles contract causes the blood to push up against the cusps, allowing them to come together and close. This prevents the flow of blood travelling back from the ventricles to the atria in a retrograde fashion.

Valvular disease and incompetence is not an uncommon occurrence seen in cardiac nursing care. The details and specific management are explored in detail in Chapter 15.

The ventricles

The function of the ventricles is to maintain the circulation via their pumping action. The two chambers are divided by the interventricular septum. An intact septum ensures that the two separate, but integrated circulations, are maintained. The right ventricle pumps deoxygenated blood through the pulmonary circulation, whilst the left ventricle pumps oxygenated blood through the systemic circulation. If the septum is damaged, either congenitally or as the result of an infarct, there is a mixing of the blood between the two ventricles. If the shunt is predominantly from the higher pressure left ventricle through to the right ventricle, there will be inadequate oxygenation of the blood. Shunts can also flow in the other direction. More detail regarding these defects is given in Chapter 14.

The right ventricle extends from the tricuspid valve almost to the apex itself. In comparison to the left ventricle, it is much smaller and with thinner walls, reflecting its role of pumping blood to the pulmonary
circulation, as opposed to the entire body. The left ventricle is larger, more powerful and has additional electrical pathways (fascicles). This highlights its role as a powerful systemic pump, maintaining tissue oxygenation. In both ventricles, the muscle fibres follow a distinctive spiral path. The fibres sweep from the base of the heart to the apex, forming a 360° clockwise rotation. This spiral arrangement ensures that when the ventricles contract, blood is propelled into the respective outflow tracts, the aorta and the pulmonary artery.

The semilunar valves (Fig. 4.3)

The pulmonary valve lies between the right ventricle and the pulmonary artery. This is generally considered as a semilunar valve, which consists of three cusps that are joined to each other by commissures. The aortic valve guards the left ventricular outflow tract. This is also a three-cusp, semilunar valve, with a similar structure to the pulmonary valve. However, it is stronger in construction due to the higher pressure generated by the left side of the heart. The leaflets of the aortic valve are attached to the annulus, which forms part of the aortic sinuses of Valsalva. This structure creates a well-defined complete circumferential tubular area, consisting largely of non-contractile collagenous tissue. This creates an ideal and structurally stable arrangement for the coronary arteries that open near the upper part of the sinus.

The semilunar valves open and close in a particular fashion. As the ventricles contract, intraventricular pressure rises above the pressure in the aorta and the pulmonary trunk. This pumps blood out of the ventricles into the blood vessels, pushing the valves open and forcing the cusps against the walls. When the pressure in the ventricles falls below that of the major vessels, blood will begin to flow backwards, fill the cusps and shut the valve.

The layers of the heart

The walls of the heart consist of three anatomically distinct layers: the outer protective pericardium, a middle layer known as the myocardium, or the muscle of the heart, and the inner layer known as the endocardium which lines the chambers of the heart (Fig. 4.4).

The pericardium

The pericardium is the outermost layer of the heart. It forms a firm fibrous sac within which the heart is suspended by its attachments to the aorta and the pulmonary artery. This mechanism seems a physiological necessity that fixes the base, but leaves the apex of the heart relatively free. This phenomenon seems important to ventricular function, especially as the dimensions of the ventricles change during contraction (see Box 4.1), moving the apex forward and thus allowing it to strike against the left side of the chest wall in the area of the fifth intercostal space.

The role of the pericardium is to provide physical protection for the heart against mechanical force. It consists of two components, the fibrous and the serous pericardium. The fibrous pericardium consists of mesothelium, a compact collagenous fibrous tissue, which encloses the heart and merges with the tunic adventitia of the major vessels. The serous pericardium, on the other hand,
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is a double-layered membrane consisting of flat secretory epithelium, connective tissue and some adipose tissue. The quantities of the adipose tissue are related to the general body fat. The innermost layer of the serous pericardium, the **epicardium**, is fused to the surface of the myocardium, thereby forming the **visceral** pericardium.

Conversely, the outer layer of the serous pericardium is fused with the inner aspect of the fibrous pericardium and now forms the **parietal** pericardium. The two layers of the serous pericardium are joined at the juncture of the great vessels with the heart, forming a small fluid-filled cavity – the pericardial cavity (see Fig. 4.4).

**Figure 4.4:** The layers of the heart (after Marieb and Hoehn 2007)

The pericardium receives its blood supply from arteries derived from the internal thoracic and musculophrenic arterial network and the descending thoracic aorta. The pericardial venous drainage is returned to the internal thoracic veins. The pericardial nerve supply is derived from branches of the vagus nerve, the phrenic nerves and the sympathetic trunk. Disruptions to the pericardium can cause medical emergencies such as cardiac tamponade which occurs when fluid collects, through trauma, malignancy or following surgery, within the pericardial cavity. This can constrict the heart’s pumping action. Conversely, pericarditis can occur due to an alteration in the serous fluid. This causes a roughening of the membranous surfaces and results in the classic ‘pericardial rub’ heard on auscultation (see Chapter 18).

**The myocardium**

The myocardium, the middle layer, forms the bulk of the heart. It is composed primarily of cardiac muscle cells (**myocytes**). Others include the autorhythmic cells with their propensity to generate electrical impulses devoid of external stimuli. It is the layer that generates the cardiac pumping action. When relating the anatomical features of the heart to its function as a pump, it is helpful to focus first upon the fibrous zone. This forms the cardiac skeleton into which insert myocardial fibres and the annulus of the heart valves. The interposition of the fibrous skeleton between the atria and the ventricles also prevents myocardial continuity between these two chambers. Consequently, the atrial myocardium is confined to the atrial chambers and the ventricular myocardium is confined to ventricular chambers. The myocardium is important within cardiac nursing care, primarily due to the serious conditions of myocardial

**Box 4.1**

Summary of the events of contraction

The action potential is transmitted speedily to the myofibrils via the T-tubules. There is an influx of calcium into the myocardial cytoplasm and also liberation of calcium from the sarcoplasmic reticulum (the intracellular calcium stores). The calcium ions bind to troponin, causing a change in conformation and freeing tropomyosin from its position, which is blocking the binding sites on actin. Contraction can then occur because the myosin can form crossbridges with the actin, pulling the actin filament inwards. Actin–myosin detachment is dependent on the binding of magnesium and adenosine triphosphate (ATP) to the ATPase site of the myosin head region. The ATP is hydrolysed, leaving the myosin head energized. During repolarization, calcium ions are removed; some are pumped into the sarcoplasmic reticulum and some become extracellular. This then re-instantes the tropomyosin blockage and relaxation of the muscle occurs.
ischaemia and infarction (see Chapter 11). More detail regarding this area and the cardiac myocytes themselves will be discussed later.

The endocardium

This is the inner layer lining the heart and is composed of squamous epithelium. Along with the vascular endothelium, it is regarded as one continuous sheet, however recent evidence suggests that endocardial endothelial cells are actually distinct from those endothelial cells in the vascular system (Brutsaert 2003). Significantly, the vascular and cardiac endothelium are known to be the source of numerous chemical mediators, including nitric oxide (NO), previously identified as endothelium-derived relaxing factor, and endothelin, that are involved in vasoregulation. Nitric oxide is a vasodilator, while endothelin is a powerful vasoconstrictor.

However endothelin also plays an important role in cardiovascular physiology, from cardiac development, through growth and remodelling to the control of cardiac contractility and rhythmicity (Brutsaert 2003). There is a high density of endothelin receptors within cardiac tissue, including cardiomyocytes and coronary vasculature. There are two types of receptors presently identified as ET\textsubscript{A} and ET\textsubscript{B}. There are now drugs being developed that work at these receptor sites.

Infective endocarditis (IE) occurs when microorganisms colonize the endocardial surface of the heart and valves, causing local tissue destruction. It remains a debilitating and in many cases a life-threatening disease. Chapter 16 explores the condition in depth.

The coronary blood vessels

Figure 4.5 provides a useful diagrammatic representation of the coronary circulation. The development of specialist roles within cardiac nursing, such as the angiography nurse, has led to the need for a clear overview of the coronary circulation and, importantly, which areas of the myocardium the various blood vessels serve.

The right and left coronary arteries are the first vessels to arise from the anterior and left posterior sinuses of the ascending aorta. The opening orifices of these arteries are generally found above the aortic cusp margins. The two arteries form an oblique crown by way of anastomoses in the atrioventricular sulcus. In encircling the heart along the atrioventricular groove, the coronary arteries form a distinctive ring that gives off branches supplying the atria and ventricles.

The right half of this ring, which is formed by the right coronary artery, gives off a short penetrating artery, which supplies the atrioventricular node. Correspondingly, the left half of the ring is formed by the left coronary artery, which divides to deliver oxygenated blood to both ventricles. However, according to West (1990), in 90% of human subjects the right coronary artery is the dominant larger vessel that traverses down the groove between the right atrium and the right ventricle, giving rise to branches that supply both chambers. This artery then turns toward the apex, as it reaches the posterior aspect of the heart. At this point, the artery becomes the posterior descending branch, which supplies the lower aspect of the left ventricle.

Figure 4.5: The adult coronary artery circulation. A: Anterior view. B: Inferior-posterior view
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The left coronary artery is a short vessel, which divides into an anterior descending branch (LAD) and a circumflex branch (CX). The LAD branch supplies the interventricular septum and the anterior surface of the left ventricle. The circumflex artery supplies the lateral aspect and part of the posterior aspect of the left ventricle. Whilst this is a common distribution, there are many variations in coronary artery anatomy and in some subjects the left coronary artery may be the dominant vessel. In such circumstances, the right coronary artery (RCA) is very small and the circumflex supplies the inferior wall of the left ventricle. The pattern of coronary artery distribution is of course critically important, as any alteration in the coronary blood flow may result in changes in the blood supply to specific areas of the heart.

As the heart is an aerobic (requiring oxygen), constantly active organ, an efficient coronary circulation must provide a rich blood supply to the myocardium. For this reason, a rich network of capillaries passes through the myocardium, creating a relatively short diffusion distance between the myocytes and their energy-producing mitochondria. Mitochondria, which occupy a large proportion of each myocyte, are often described as the powerhouse of the cell, producing the energy (such as adenosine triphosphate; ATP) that the cells need to survive and function. It is for this reason that some degree of arterial anastomosis may occur in the mature heart in conditions of myocardial hypoxia and coronary artery disease. These may be anastomoses between two branches of the same coronary artery or connections of branches of the RCA with branches of the left. The functional importance of this collateral circulation has more recently been seen as a method of protecting the heart (McCance 2006).

The cardiac veins, which collect the venous blood returning from the heart itself, consist of numerous venous tributaries and a coronary sinus. The anterior cardiac veins drain the anterior region of the right ventricle. The large majority of cardiac veins drain into the wide coronary sinus, which is about 2 or 3 cm long, lying posterior to the coronary sulcus between the left atrium and the left ventricle. The coronary sinus opens into the right atrium between the orifice of the inferior vena cava and the atrioventricular annulus. An endocardial fold, known as the semilunar valve of the coronary sinus, guards its opening.

The great cardiac vein begins at the apex of the heart and ascends in the anterior interventricular sulcus en route to the coronary sulcus, turning to the left as it eventually enters the coronary sinus. A range of smaller cardiac veins, which receive deoxygenated blood from specific regions of the heart, thus support the work of the great cardiac vein. These smaller vessels can be identified in Figure 4.6.

Figure 4.6: The coronary venous circulation (after Marieb and Hoehn 2007)

Lymph drainage of the heart

A substantial lymphatic network supports the heart. In this context, fine thin-walled lymphatic vessels are distributed throughout the myocardium, forming a plexus immediately below the endocardial surface. The lymph channels follow the path of the conductive tissue. The bundle branches and the atrial surfaces of the tricuspid and mitral valves have a particularly extensive lymph vessel supply. A single large channel carries the lymph to a pretracheal node near the arch of the aorta.

The innervation of the heart

The heart receives its nerve supply from the sympathetic and parasympathetic branches of the autonomic nervous system. Branches of the afferent and efferent fibres of the vagus and phrenic nerves and the thoracic and cervical cardiac nerves convey impulses to the cardiac plexus. The cardiac plexus is a group of nerve ganglia positioned between the aortic arch and the bifurcation of the trachea. Further networks of nerve ganglia are found in the subendocardium of the right atrium, the interatrial septum and the epicardium near the roots of the aorta and the pulmonary artery. Branches of the sympathetic and the parasympathetic nervous system also supply the sino-atrial (SA) and AV nodes. However, the sympathetic and parasympathetic nerves of the cardiac plexus only serve to modulate myocardial performance, they do not participate in the propagation of the action potentials through the myocardium.

The myocardial architecture

The myocardium is predominantly composed of cardiac muscle, which makes up the greater proportion of the
The anatomy of the adult heart

The myocardial cells can be classified into two main types. The most important of these are the myocytes, which are elongated force-generating cells (Fig. 4.7). The second type, which are in the minority, are those with autorhythmic properties. These form a specialized electrical pathway (the conduction pathway) that will be discussed later in this chapter.

The myocytes are embedded in a matrix of connective tissue and surrounded by a rich network of blood vessels, capillaries, lymph vessels and nerves. They are cylindrical in shape, but branch freely. The myocytes vary in size, depending on their atrial or ventricular origin. However, they are small. Atrial myocytes are less than 10µm in diameter and about 20µm in length, whilst ventricular myocytes are 10–20µm in diameter and 50–100µm long.

The branching myocytes are attached to each other at junctions known as intercalated discs (Fig. 4.8). These discs contain desmosomes and gap junctions. The function of desmosomes is to rivet the adjacent cells together, to prevent them separating during muscle contraction and consist of plaques of cadherin molecules. Cadherin is a transmembrane glycoprotein that spans the 25 nm wide space between the adjacent myocyte membranes (Levick 2003). Gap junctions (or nexus) allow the movement of ions from one cell to another via connexons, which are protein particles with a central channel that spans the gap. This direct movement of ions from one cell to another ensures that the entire myocardium behaves as one unit and this is referred to as a functional syncytium. When coronary heart disease causes myocardial ischaemia, the rise in intracellular acidity and Ca²⁺ causes closure of some of the connexons, leading to poor electrical coupling (Levick 2003).

The myocytes contain mitochondria, whose function is to produce energy for the cells, in the form of ATP. ATP is an organic molecule that stores and releases chemical energy for use within cells. Therefore cardiac muscle cells that are constantly working need a large amount of energy and thus large mitochondria. The myocytes also contain organelles called sarcoplasmic reticulum whose function is to store calcium ions. The surface of the cell consists of a membranous structure known as the sarcolemma (cell membrane). The cell membrane protrudes down into the cell, forming a set of transverse tubules (T-tubules) whose function is to rapidly transmit the external electrical stimulus inside the cell.

The cardiac muscle cells are filled with cross-striated myofibrils, which are similar to those in skeletal muscle. They are composed of smaller units known as sarcomeres (the contractile units).

Within the sarcomere are two types of protein filament (myofilaments) that are arranged in such a way that when there is a muscle contraction, they partially slide over each other to cause shortening, known as the slidding filament theory (see Fig. 4.7). The two types of protein filament are known as the thick and the thin filaments. The former is composed of the protein myosin. The myosin molecules are made up of two globular heads and a rod-like region, consisting of a neck and tail, which provide stability to the molecule. The globular head contains active sites that hydrolyse ATP and interact, forming bridges, with designated sites of the actin molecule.

The thin filament, although predominantly made of actin, contains two other types of protein, tropomyosin and troponin. These filaments are functionally involved in tension generation and muscle contraction. The tropomyosin filaments are positioned in the grooves of the actin helix. Attached to the tropomyosin, at intervals of 40 nm, are the tropinin complexes, consisting of three distinguishable proteins known as troponin-T, troponin-C and troponin-I. Collectively the troponin tropomyosin molecules are responsible for the regulation of actin-myosin function.

The above principles offer some clarification of the sliding filament hypothesis, which is a major contributor
to the current understanding of muscle contraction. The theory implies that although a muscle shortens during contraction, the length of both the thick and thin filaments remains constant and unchanged. Indeed, the shortening events are best described in terms of the thin filaments being pulled into the lattice of the thick filaments. Each contraction therefore depends on the thin filaments being pulled towards the centre of the sarcomere. Thus the degree of contraction is limited by the length of the sarcomere. The Frank–Starling mechanism (Starling’s Law) of the heart states that, within physiological limits, the greater the degree of stretch, the greater the force of contraction. The term ‘physiological limit’ refers to the sarcomere; if stretched too far then some of the bridges between the myosin and actin become detached and will reduce the force of contraction.

The cardiac nurse needs to have an increased knowledge of the myocardial architecture to fully understand advances made in cardiology. Examples include the discovery that some forms of hypertrophic cardiomyopathy are due to new mutations in parts of myosin filaments (Watkins et al 1992) and the identification of cardiac troponins as highly sensitive and specific biochemical markers for diagnosing myocardial infarction (MI) (Wu 2003).

**The conduction pathway**

A knowledge of the conduction system, together with the associated recordings of the electrocardiogram (ECG), is essential for the cardiac nurse. Nurses working within acute units need to understand how conduction abnormalities will manifest and the subsequent appropriate treatments. The management of cardiopulmonary resuscitation by nursing and medical staff against printed algorithms is one example.

The autorhythmic cells within the myocardium, in health, function in a specific order, known as the conduction pathway: first the sino-atrial (SA) node, then the atrioventricular (AV) node or junction, then the bundle of His, left and right bundle branches and the Purkinje fibres (Fig. 4.9).

The cardiac ‘pacemaker’ or SA node initiates each cardiac cycle. Its anatomic location at the junction of the embryonic sinus venosus and the right atrium proper, appears to be ideally suited to its function, ensuring that the atria are depolarized first. A plaque of subepicardial fat frequently covers this node, which makes it visible to the naked eye. The node has its own large central artery, which takes its origin from either the right or the circumflex coronary artery. The structural features
of this node include some slender cells, which are confined to the nodal centre surrounding the nodal artery. These cells are arranged somewhat more irregularly on the external surfaces and are considered as the specialized pacemakers of the heart. They initiate the wave of electrical depolarization that is eventually propagated through the entire myocardium. They make functional contact with each other and the adjacent transitional myocytes, which are smaller than the contractile myocytes. These transitional cells establish a zone, which forms a junction between the SA node and the surrounding myocardium.

The AV node is located in the muscular part of the AV septum. Its inferior aspect enters a central fibrous body forming the ativoventricular bundle, also known as the bundle of His. This node consists of irregular collagenous reticulum, which enmeshes the myocytes, forming a compact and a transitional zone. The transitional zone is thought to be responsible for the delays in the cardiac impulse. However, the myocardial discontinuity must permit the bundle of His to penetrate the central fibrous body and establish a bridge of excitable tissue between the atria and the ventricles. This anatomical feature is of considerable physiological significance because it provides the only acceptable pathway for the conducting system between atria and ventricles. Generally, the AV node receives its blood supply from a tributary of the RCA, although in a small percentage of people this may be from the left circumflex artery. The blood supply to the SA and AV nodes and other components of the conduction pathway is of great relevance to the cardiac nurse. This knowledge will assist in predicting conduction problems in association with blockages to the coronary arteries (see Chapters 7 and 23).

The bundle of His is the direct extension of the AV node. It branches on the upper surface of the muscular interventricular septum, forming a right and a left bundle branch. The blood supply to the bundle of His and the first few millimetres of the bundle branches is via the tributary of the RCA that supplies the AV node and also from branches of the left anterior descending artery.

The right and left bundle branches are similar in structure. However, the left bundle branch consists of two sets of fibres, termed anterior and posterior fascicles, whilst the right bundle branch is a single cord-like structure. The right bundle branch is embedded in the myocardium and subendocardium, radiating towards the apex of the right ventricle and reaching the anterior papillary muscles. It then forms fine subendocardial conduction pathways, which supply the remainder of the right ventricular wall. The left bundle branch forms a framework down the smooth left ventricular septum, which eventually give rise to a rich subendocardial network, which first surrounds the papillary muscles and then the remaining part of the left ventricle. The bundle branches are supplied by branches of the left anterior descending artery.

The smallest structures of the conduction system are the Purkinje fibres, which are large pale cells containing more glycogen and fewer contractile filaments than the cells of the contractile myocardium. Contact between the Purkinje fibres and the contractile myocardium is best established in the subendocardial regions. This arrangement permits the papillary muscles to contract first.
This is followed by a wave of excitation and the ensuing contraction then travels from the apex to the ventricular outflow tract. As the Purkinje network is subendocardial, myocardial excitation and contraction proceed from the endocardium to the epicardium. The spread of the excitation is rapid, but not instantaneous. A short delay allows different parts of the right and left ventricles to receive their impulse at slightly different times.

The functional relationship of the conduction pathway to the heart as a whole allows us to appreciate how failure in the conduction system may not necessarily block myocardial contractility, but the function of the heart as a pump will become poorly co-ordinated. Importantly, as slower conduction and alterations in rhythm occur, a ‘new’ dominant pacemaker may arise from focal spontaneous myogenic activity in the contractile myocytes or distal part of the conduction system. Arrhythmias, particularly tachycardias, occur when an irritable focus predominates, becoming, for that period of time, the pacemaker of the heart.

Although the autonomic nervous system influences various functions of the heart, including the frequency and vigour of each contraction, cardiac function is not entirely dependent on neural stimulation and control. This is because automaticity and rhythmicity are physiological phenomena intrinsic to the myocardium and its conduction system. The heart will continue to beat even when denervated. Therefore, providing the coronary blood vessels are perfused, rhythmic myocardial contraction will continue. The transplanted heart is a clear example of this.

**Electrophysiology**

- **Action potential.** Changes that occur in the cell membrane, allowing the inward and outward flow of ions during depolarization and repolarization
- **Depolarization.** The moment when the interior of the cardiac cell is maximally charged with positive ions
- **Repolarization.** The process of restoration of a cell to its normal resting membrane polarity following depolarization.

As highlighted previously, autorhythmicity of the heart has been described as spontaneous diastolic depolarization and is initiated in the SA node. Each action potential thus generated by the SA node will set up the propagated wave of depolarization that initiates the systole in all regions of the heart. However, the action potentials of the SA node are small with a slow upstroke, which suggests that the sodium ion channels in these cells may not be fully functioning. It could also be argued that the absence of an inward sodium current is partly due to the high resting potential enjoyed by the SA node. As a high resting potential will inactivate sodium ion channels, it must be assumed that the upstroke of the action potentials generated within the SA node is largely due to an influx of calcium ions.

Spontaneous pacemaker activity exists in all regions of the AV node. However, this node appears to be under the control of the autonomic nervous system. As in the SA node, the characteristic slow AV node depolarization reflects the absence of functional sodium ion channels. A slow inward calcium current primarily causes the spontaneous diastolic depolarization. Control of this current

**Overview of the action potential**

The action potential of the autorhythmic cells (Fig. 4.10A)

The autorhythmic cells do not depend on an extrinsic (external) nerve supply as the stimulus because they have an intrinsic ability to depolarize. The precise mechanism of how this occurs is unclear. However, the belief is that the autorhythmic cells have a reduced membrane permeability to potassium (K⁺). Yet, sodium (Na⁺) permeability is unchanged and it continues to diffuse into the cell at a slow rate. As the interior of the cell becomes less negative, gates along the cell open and allow sodium in at a rapid rate. The membrane interior thus becomes less and less negative and slowly more positive. Ultimately, when a certain threshold is reached, fast calcium channels open, allowing an explosive and fast entry (influx) of calcium into the cell from the extracellular space. Therefore, in autorhythmic cells, an influx of calcium, rather than sodium, produces the rising phase in the action potential and rapidly reverses the membrane potential, creating an electrical impulse

(Marieb and Hoehn 2007).

The falling phase of the action potential, repolarization, reflects an increased potassium (K⁺) permeability and its efflux (exit) from the cell. Once repolarization is complete, the potassium ion channels are inactivated and the membrane permeability to K⁺ is reduced. Then the whole process of depolarization begins again.

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Spontaneous pacemaker activity exists in all regions of the AV node. However, this node appears to be under the control of the autonomic nervous system. As in the SA node, the characteristic slow AV node depolarization reflects the absence of functional sodium ion channels. A slow inward calcium current primarily causes the spontaneous diastolic depolarization. Control of this current
by neurotransmitters such as adrenaline (epinephrine), from the sympathetic fibres and acetylcholine, from the parasympathetic, plays a critical role in regulating the slow conduction velocity. However, slow conduction in the AV node is also due to the small nodal cell size and the relatively small number of gap junctions, which are responsible for a high internal resistance. By contrast, the cells of the remaining part of the conduction system pathway have action potentials that are long and of large magnitude. This ensures that impulses do not re-enter the Purkinje system after they have activated the ventricles.

In addition to the SA and AV nodes, many other regions of the heart have the capacity to generate characteristic action potentials. These are specific for their specialized electrophysiological role. However, two dominant action potentials may be noted with respect to the heart: the fast response action potentials that occur in the atrial and ventricular myocytes and Purkinje fibres; and the slow response action potentials which occur in the SA and AV nodes.

The physiological relationship between the electrical events of the action potential and the actual mechanical contraction of the muscle is detailed later. Rapid depolarization precedes the development of muscle force and the completion of depolarization generally coincides with peak force or contraction. Of course, the duration and magnitude of the action potential will influence the duration of the muscle contraction. On the other hand, an unacceptable increase in the number of action potentials in a given time will result in a decrease in the time committed to each impulse and, of course, a corresponding decrease in the mechanical contraction of the muscle.

The action potential in the autorhythmic cells can be summarized in the following phases:
- Phase 0 representing depolarization
- Phase 3 representing repolarization
- Phase 4 representing the ‘resting potential’.
  However, as this can be regarded as unstable, it is not truly a resting potential.

The action potential of the contractile myocytes (Fig. 4.10B)

Although the ionic basis of the action potential in myocytes continues to be investigated, the assumption is that any abrupt changes in the resting membrane potential will result in a propagation of an action potential (West 1990, Katz 1992). This assumption gives rise to a belief that the rapid myocyte depolarization or

Figure 4.10: Cardiac action potential sources. A: Autorhythmic cell. B: Contractile myocyte
CHAPTER 4  The applied anatomy and physiology of the cardiovascular system

excitation (phase 0) may be almost exclusively attributed to a sudden increase in sodium influx. The influx of the sodium ions diminishes the transmembrane potential and this increases the influx of sodium ions further, causing the membrane potential to become positive. The sodium channels then close and the membrane potential begins to fall (phase 1). This is due to a short period when chloride ions re-enter the cell. However, it is at this point that there is a sustained inward current of calcium ions. This prolongs the action potential for a brief period of time (200–400 ms) and prevents the myocyte from rapidly repolarizing. This is the plateau phase (phase 2).

In phase 3, repolarization, the cell membrane returns to its resting potential of about −90 mV. The falling phase of the action potential reflects an increased potassium (K⁺) permeability and its efflux (exit) from the cell. Once repolarization is complete, the potassium channels are inactivated and phase 4 or the resting state is reached. The inside of the cell is once again negative due to the K⁺ leakage. An excess of sodium remains in the cell, because the membrane is much less permeable to this ion. The sodium–potassium pump is then activated, pumping sodium out of the cell and moving potassium inside. At phase 3 of the action potential, some of the gates of the sodium ion channels open and as the channels recover from their inactivation, sodium influx resumes and the myocyte begins to respond again, but rather weakly initially.

The action potential in the contractile myocytes can be summarized in the following phases:

- An upstroke (phase 0) representing depolarization
- A brief partial repolarization (phase 1)
- A plateau (phase 2)
- A gradual, but progressive, repolarization (phase 3)
- A resting potential (phase 4).

During the plateau period, the myocyte is said to enter its effective refractory period. The benefit of this mechanism is considerable because the refractory period prevents tetanic contraction (a sustained muscle contraction) of the myocardium.

Excitation – contraction coupling

As indicated earlier, co-ordinated myocardial contractility is initiated by the rhythmic discharge of action potentials that spread across the myocardium. However, the mechanisms involved in the excitation and contraction of the myocardium overlap considerably in time. Consequently, the ending of an action potential coincides with the beginning of myocardial relaxation. This characteristic relationship seems entirely appropriate to the pump action of the heart. An important event of the excitation–contraction coupling is the influx of calcium into the myocytes during the action potential which prolongs both the action potential and the refractory period.

The co-ordination of cardiac activity

The rhythmic action potentials that drive the heart as a pump originate in a group of pacemaker cells in the SA node and spread rapidly, but sequentially, through the atria and then the ventricles, depolarizing them to contract. This mechanism ensures that the human (adult) heart pumps ceaselessly at about 70–80 cycles per minute, maintaining a constant perfusion of the pulmonary and systemic tissue. However, as the entire cardiovascular system enjoys a certain capacity for adaptability, the heart rate and stroke volume (the amount of blood ejected in one heart beat) fluctuate in accordance with the prevailing physiological demands of the individual.

The principal events of a cardiac cycle are complex and dependent on precise timing of the many mechanical operations. Neural influences are important contributors in that they adapt the intrinsic cardiac rhythm to functional demands made on the cardiovascular system by the whole of the body.

The mechanical events of the cardiac cycle

The cardiac cycle may be defined in terms of mechanical events that occur from the beginning of one heart beat to the beginning of the next, as illustrated in Figure 4.11. As the resting human heart rate is maintained at approximately 70 beats/min, the phases of the cardiac cycle need to be accomplished in less than a second (about 0.8 seconds). Furthermore, the electrical activation of the myocardium must ensure that the four chambers of the heart contract sequentially, but not entirely, in synchrony. This allows the atria to act as primer pumps, whilst the ventricles provide the major source of power for the movement of blood through the extensive pulmonary and systemic vascular compartments.

The cardiac cycle is characterized by a series of pressure changes within the heart that result in blood flowing from high- to low-pressure areas. Healthy cardiac valves prevent blood from flowing in the wrong direction. The cardiac cycle, due to its continuous nature, can be described from different starting points. This description will start when the ventricles are relaxing, in diastole. The terms ‘diastole’ and ‘systole’, by convention, refer to the state of the ventricle. When they are used for the atria, this will be specifically stated.

Mid to late ventricular diastole. The pressures are low in the heart and the blood passively returns to the atria and to the ventricles through the open AV valves. About 70–80% of ventricular filling occurs by this route. The semilunar valves are closed because the pressure in the major vessels (the pulmonary artery and aorta) is
(systole) follows, forcing the remaining 20–30% of blood into the ventricles. Atrial contraction is normally slightly asynchronous because the right atrium contraction occurs ahead of the left atrium by approximately 0.02 seconds, but this has no significant effect on cardiac output. The atria then relax for the rest of the cycle.

**Ventricular systole.** As the atria relax, the ventricles depolarize (the QRS complex on the ECG). There is then ventricular contraction (systole), which causes an increase in the pressure within the ventricles. As the pressure starts to rise, the AV valves close. So at this stage in the cycle, all four valves are closed and the volume of blood in the ventricles is constant. This is known as isovolumetric contraction. As the ventricles continue to contract, the rapidly rising pressure exceeds the pressure in the major vessels and the semilunar valves open. This occurs when the left ventricular pressure rises slightly above 80 mmHg and the right ventricular pressure slightly above 27–30 mmHg. This marks the onset of the ejection phase.

During the ejection phase, the pressures in the left ventricle and the aorta briefly rise to a maximum of about 120 mmHg. At this point the systolic and diastolic pressures in the right ventricle and the pulmonary artery are thought to be approximately 25–28 mmHg.

**Early ventricular diastole.** There is then repolarization of the ventricles (T wave on the ECG) and, as a consequence, the ventricles then relax (diastole). The pressure then drops in these chambers, allowing the raised aortic and pulmonary artery pressures to push blood back towards the ventricles, a process that snaps the aortic and pulmonary valves into a closed position. The dicrotic notch on the arterial pressure trace marks this event (see Chapter 21). So once again the ventricles are closed chambers and this is known as isovolumetric relaxation. During the period of ventricular activity, the atria have been filling. Eventually, the pressure in the ventricles will be lower than that in the atria and so the AV valves will open, allowing blood to flow through into the ventricles.

Each cardiac cycle is about 0.8 seconds, although this will depend on heart rate. Atrial systole takes 0.1 seconds, ventricular systole takes 0.3 seconds, so for 0.4 seconds the heart is totally relaxed. An increase in heart rate will decrease the resting period (the diastolic time), which has implications because the coronary arteries fill during diastole and therefore any reduction in filling time may impair the blood supply to the myocardium.

**Atrial function.** The function of the atria may be normally represented by characteristic pressure curves and haemodynamic recordings identify three major pressure elevations (Fig. 4.12).

- An ‘a’ wave, which is initiated by atrial contraction.
  In the healthy adult, the right atrial pressure may rise to 5 or 6 mmHg during systole and the left
Atrial pressure may rise to approximately 7 or 8 mmHg

- A ‘c’ wave, which is initiated by the beginning of ventricular contraction. Guyton & Hall (2006) attribute this positive wave to the slight backflow of blood towards the AV valves and a corresponding backward bulging of these valves into the atria as the ventricles begin to contract and the ventricular pressure rises.

- A ‘v’ wave, which arises as a consequence of the slow return of venous blood to the atria, while the AV valves are closed during ventricular contraction. Characteristically, the ‘v’ wave appears towards the end of ventricular contraction and disappears as the ventricles relax and the AV valves open.

- This allows the accumulated atrial blood to flow rapidly into the respective ventricles, causing the ‘v’ wave to disappear again.

The significance of the end-diastolic, end-systolic volume and stroke volume

The period of diastole normally allows the ventricles to increase their volume to about 110–125 ml each and this is known as the end-diastolic (filling) volume or preload. The preload is defined as the volume of blood that stretches the muscles of the ventricular chambers prior to contraction. During systole, in a resting individual, the ventricles eject approximately 70 ml of blood; this is known as the stroke volume output. The remaining volume in each ventricle, about 40–55 ml, is known as the end-systolic volume. There is, however, considerable variation in the volume of blood ejected during each systole. This variation is largely influenced by the activity of the individual person; for example, when the heart contracts more forcefully during exercise, the end-systolic volume can fall to as little as 10–20 ml. Conversely, when large quantities of blood are returned to the ventricles during diastole, the end-diastolic volume may increase to 150–250 ml. Therefore, by increasing the end-diastolic volume and decreasing the end-systolic volume, the stroke volume output will be increased significantly and may even double at times of high metabolic demand.

Another physiological variable of considerable clinical importance is the ejection fraction, a concept which defines the ratio of stroke volume to end-diastolic volume. Essentially the ejection fraction describes the stroke volume as a percentage of the ventricular end-diastolic volume, normally approximately 50–65%. However, when evaluating ventricular function, it has to be remembered that the ejection fraction is a measure of pump function, rather than contractility, because it is influenced by a number of haemodynamic variables. These include...
the preload, afterload and the heart rate. The afterload is the resistance against which the left ventricle must eject its volume of blood during systole. The blood present in the vascular compartment and the blood vessel walls themselves generate this resistance. The ejection fraction can, however, distinguish between two major types of heart failure. For example, in circumstances of impaired myocardial contractility, the ejection fraction is low. Conversely, in circumstances where ventricular relaxation is impaired, for example in some presentations of hypertrophic cardiomyopathy, the ejection fraction may increase.

### Normal heart sounds

An understanding of the sequence of electrical and mechanical events that occur in the right and left sides of the heart is central to the appreciation of the timing of heart sounds and murmurs. Auscultation with the aid of a stethoscope is one of the best-established methods used to distinguish between the different sounds of the heart. When listening to a normal heart, one hears sounds generally described as ‘lub … dub … lub … dub’. The ‘lub’, also known as the first sound, is associated with the closure of the AV valves at the onset of systole. The ‘dub’ gives rise to the second heart sound and is associated with the closure of the semilunar valves at the end of systole.

#### The first heart sound

The first heart sound is attributed to vibrations caused by the taut AV valves immediately following their closure. These vibrations are enhanced by sounds generated by the blood, the walls of the heart and of course the major vessels around the heart. Contraction of the ventricles causes sudden backflow of blood against the closed tricuspid and mitral valves, causing them to bulge towards the atria. The elastic nature of these valves then closes the AV valves and establishes the characteristic vibrations that are then transmitted to the chest wall. The duration of the first heart sound averages 0.14 seconds.

#### The second heart sound

The second heart sound is attributed to the sudden closure of the aortic and pulmonary valves. As these valves close, they bulge towards the ventricles. However, as the elastic tissue of these valves recoils, the blood is moved back into the arteries, which causes characteristic vibrations that are then transmitted to the chest wall.

The duration of the second heart sound averages 0.11 seconds. The shorter sound is attributed to the extra tautness of the semilunar valves, which gives rise to a shorter vibration period in comparison to the vibrations of the AV valves. However, the second heart sound normally has a higher (pitch) frequency than the first heart sound. The rationale for this can again be found in the greater tautness of the semilunar valves in comparison to the AV valves and the greater elasticity of the arteries in comparison to the looser ventricular chambers.

### The third heart sound

Occasionally, a third sound is heard in middle diastole. The cause of this sound is attributed to the oscillation of blood between the walls of the ventricles, which is initiated by the entry of blood from the atria. The reason for this sound appearing in mid-diastole is presumably that in the early part of diastole the heart is not filled sufficiently to generate tension in the ventricles, which is essential for the rumbling reverberation characteristic of the third sound. The frequency (pitch) of this sound is normally very low, making it difficult to hear on auscultation.

### The fourth heart sound

This sound is frequently referred to as the atrial sound because it occurs as the atria contract. The main contributor to this sound is the flowing of blood into the ventricles, which initiates the characteristic low-frequency vibrations. Due to its low frequency, this sound can almost never be heard on auscultation.

### The regulation of the pumping heart

As indicated earlier, the heart is a highly adaptive organ. Its activities are regulated in accordance with the activities and metabolic needs of the body as a whole. The two most responsive mechanisms that regulate the ‘pumping’ of the heart are:

- the intrinsic cardiac regulation, which augments the pumping action of the heart in direct response to changes in the volume of the blood flowing into the heart
- the autonomic nervous system.

### Intrinsic regulation of the mechanical activities of the heart

The pumping activity of the heart and the amount of blood pumped each minute are determined by the rate of blood flow into the heart. This is generally known as the venous return, which may vary in volume depending on the physical demands made on the body. In the systemic circulation, the venous return consists of deoxygenated blood being returned to the right atrium and thus the right ventricle. By contrast, the pulmonary venous circulation consists of oxygenated blood being
returned through the pulmonary veins to the left atrium and left ventricle. The heart then automatically pumps the oxygenated blood into the aorta and the deoxygenated blood into the pulmonary artery (see Fig. 4.1).

The intrinsic capacity of the heart to adapt to changing volumes of returning blood is referred to as the Frank–Starling mechanism (Starling’s Law) of the heart as highlighted earlier. Starling’s Law of the heart is based simply on the notion that a greater stretch of the myocardial fibres during filling will generate a greater subsequent force of contraction, resulting in larger volumes of blood being pumped by the ventricles. The basis of this law may be outlined as follows. The additional volume of blood returning to the heart causes the myocardial fibres to stretch to a greater length. This in turn stimulates the myocardium to contract with increased force as actin and myosin filaments are brought to a nearly optimal degree of interdigitation required for force generation.

Control of the heart function by the autonomic nervous system

The effectiveness and efficiency of the pumping action of the heart are controlled by parts of the sympathetic and parasympathetic (vagus) nerves, which supply the heart abundantly. Under normal physiological conditions, the sympathetic nerve fibres to the heart discharge continuously at a slow rate that maintains cardiac pumping at about 30% (Katz 1992) above that which would occur with no sympathetic stimulation. Consequently, when the activity of the sympathetic nervous system is depressed, a reduction in the heart rate and force of ventricular contraction becomes evident. This eventually contributes to a significant reduction in cardiac output. Conversely, the parasympathetic (vagal) stimulation can induce significant bradycardia. This may be demonstrated clinically by any ‘vagal manoeuvre’. This is a purposeful stimulation of the vagus nerve, perhaps carotid sinus massage under the jaw or asking the patient to breathe out against a closed epiglottis (the Valsalva manoeuvre). This is used to slow an excessive tachycardia that may be compromising the haemodynamic status and, if successful, avoids the use of drug therapy. However, under physiological conditions, the heart will resume beating normally again within a short period of time.

In some circumstances, strong vagal stimulation will also decrease the force of myocardial contraction by 20–30%. However, this has a relatively small impact, which is attributed to the limitations of the vagal fibre distribution, being predominantly in the atria. As vagal fibre distribution to the ventricles is limited, any impact on the force of ventricular pumping is also limited. Nevertheless, a significant reduction in the heart rate combined with the moderate decrease in myocardial contractility will have a considerable impact on ventricular pumping and so cardiac output.

Finally, sympathetic nerve stimulation may increase cardiac output by more than 100%. Indeed, the general consequences of sympathetic stimulation are:

- raised (adult) heart rate to 200 beats per minute
- increases in the force of myocardial contractility
- raised ventricular ejection pressure
- raised cardiac output.

In view of the above, it must be argued that neural stimulation augments the heart rate and the contractile force of the myocardium. Under physiological conditions, the heart rate determines the cardiac output, although there are important limitations to this. For instance, when the heart rate rises above the critical point, the force of myocardial contractility decreases. In addition, in such circumstances, the period of diastole is significantly reduced, so that ventricular filling is compromised and stroke volume reduced. However, the reduction in the stroke volume may be just one of the physiological adaptations manifested by the heart in clinically significant tachycardia.

The cardiac output and distribution of blood

The cardiac output (CO) may be defined as the volume of blood ejected by each ventricle per unit of time. This is usually measured in litres per minute and can be calculated as follows:

\[
\text{Heart rate (HR)} \times \text{stroke volume (SV)} = \text{cardiac output (CO)}
\]

In the resting individual, the cardiac output generally amounts to approximately 51/min, e.g. normal heart rate 70 beats/min × stroke volume 70 ml = 4900 ml/min = 4.91/min. Slight normal variations do exist and this is particularly evident in the different values noted for females (4.61/min) and males (5.61/min). However, for all individuals the cardiac output can be increased to many times this value by an increased heart rate and a greater stroke volume. Conversely, bradycardia and reductions in the effective circulating blood volume and stroke volume may significantly reduce the cardiac output. It may therefore be concluded that cardiac output is the most critical factor that must be taken into consideration when evaluating a patient’s cardiovascular function. However, cardiac output is not a fixed entity, sustained only by myocardial contractility and the amount of blood volume present in the circulation. In reality, the cardiac output is determined principally by the sum of various factors that control local and regional blood flow throughout the body. The sum of this local blood flow forms the venous return to the heart.

Although the individual’s cardiac output is largely dependent on intrinsic cardiac factors, such as the heart
rate and myocardial contractility, other factors also play an important role. The two major factors identified are the venous return and the peripheral resistance.

**The venous return**

Blood returning to the heart via the systemic and pulmonary veins is generally known as the venous return: the sum of all the local blood flow from the individual segments of the peripheral circulation. It follows, therefore, that the cardiac output is the sum of all the blood flow regulations. Local and regional blood flow is almost always responsive to the variations of tissue metabolism. Consequently, when local tissue oxygen consumption increases, the volume of blood flow is augmented in proportion to that tissue metabolism. The advantage of such fine local adaptation is that blood flow increases in response to greater metabolic activities. This ensures that the appropriate substrates are delivered to the relevant area and the waste products are removed.

**Peripheral resistance**

One of the important factors that could affect the cardiac output is the physical resistance offered by the peripheral blood vessels. This phenomenon may be detailed as follows. Under physiological conditions and normal arterial pressure, the long-term cardiac output is reciprocal to the changes in the peripheral resistance. This implies that as peripheral resistance increases, the cardiac output falls. Conversely, as peripheral resistance decreases, the cardiac output rises. However, there are limitations to the amount of blood that the heart can pump. These limitations are best expressed in the form of cardiac output curves. The systemic vascular resistance (SVR) can be measured clinically with a pulmonary artery (PA) catheter. It is frequently regarded as the afterload, because it is a resistance offered to the ejecting left ventricle by the arterial system. The usual range is 770–1500 dynes/s/cm

The vascular system (Fig. 4.13)

Essential to the function of the cardiovascular system is the circulating blood volume, which is propelled in a pulsatile, continuous fashion through the body. The lumen of all blood vessels is lined by endothelium, which usually consists of a single layer of flat cells. The

**Figure 4.13:** The structure of arteries, veins and capillaries (adapted from Marieb and Hoehn 2007)
endothelium occupies an important location, being situated between blood and tissues. It has now been discovered that the vascular endothelium not only has an important role in regulating cardiovascular homeostasis in health, but also contributes to the pathophysiology of cardiovascular disease (Brutsaert 2003). The endothelium has the ability to respond to changes, whether physical, chemical or humoral, by the production of biologically active mediators that influence the control of the vascular diameter, e.g. prostacyclin, nitric oxide (NO) and endothelin. The endothelium also mediates haemostasis, cell proliferation and inflammatory mechanisms in the cell wall. Damage to the endothelium disrupts its normal functions and results in endothelial dysfunction. Endothelial dysfunction is an important area of research because it is a key feature of atherosclerosis, the most common cause of CHD, and seems to play a role in myocardial ischaemia and infarction.

In addition, all blood vessels, with the exception of the capillary network, are made up of varying amounts of elastic and collagen fibres as well as smooth muscle fibres. The elastic fibres, particularly in the tunica intima (the inner layer composed of endothelium), form a relatively dense network and can easily be stretched to many times their original length. These fibres exert a certain degree of tension, which opposes the tendency of the blood pressure to stretch the lumen of the vessels, without the use of any energy. Conversely, in the tunica media (the middle layer) and tunica adventitia (the outer layer), the collagen fibres form a network that offers a great deal more resistance to stretch than the elastic fibres. The spindle-shaped smooth muscle cells are connected to one another as well as to the elastic and collagen fibres. Their chief function is to provide active tension in the vessel wall (myogenic vascular tone) and to regulate the size of the lumen in response to the tendency of the blood pressure to stretch it. Autonomic nerve fibres innervate the smooth muscle in the blood vessels.

Although the vascular compartment is generally divided into the pulmonary and systemic units, from a functional point of view, the blood vessels can be classified into elastic vessels, resistance vessels, sphincter vessels, capillaries and capacitance vessels or veins. Whilst all these vessels are important to the efficient function of the cardiovascular system, the resistance vessels appear to make the greatest contribution in the regulation of the volume of blood flow, within each vascular bed. The aorta and its dominant arterial branches account for about 19% of the total resistance to blood flow. The contribution of the terminal arteries and arterioles amounts to just under 50%. That is, vessels only a few millimetres long, due to their small capacity, generate half of the resistance to blood flow. However, resistance in capillaries is also considerable, amounting to approximately 25% of the total resistance. The venous compartment offers a fairly small resistance to blood flow, averaging 4% for venules with the remaining larger veins contributing only about 3%.

Venules and veins largely determine the postcapillary resistance. Venules are tiny vessels formed when capillaries unite. As they return blood to the heart from the capillary beds, they join together to form larger veins. Significantly, veins also serve as the capacitance or reservoir vessels, in that their distensibility allows them to hold or pass on large quantities of blood without marked effect on other parameters of the circulation. Furthermore, some veins have anatomical characteristics that make them particularly capacious storage areas. This applies in particular to the venous network in the liver, the splanchnic region and the subpapillary plexus of the skin. Together these vessels can hold more than 1000 ml of blood for release as required. However, the pulmonary vessels too can be used for short-term storage or mobilization of fairly large amounts of blood, by altering venous return or augmenting the left ventricular volume output.

The aforementioned term systemic vascular resistance (SVR), is used to define the overall resistance of the systemic circulation; that is, the resistance of all the vascular beds together. Together the SVR and the cardiac output determine the blood pressure.

**Blood pressure**

The pressure differences between the various vascular regions make the flow of blood possible because in response to the pulsatile ejection of blood from the left ventricle, blood flows from high-pressure regions to lower-pressure regions. The evolving pressure gradient provides the force that overcomes the resistance to the flow of blood. Resistance to flow may vary depending on the differences in the blood vessels and the viscosity of the blood.

The pressures in the vascular compartment, that is the arterial and venous blood pressures, are equivalent to the force per unit area exerted by the blood on the walls of the vessels. Clinically, however, blood pressure implies the arterial pressure in the systemic circulation. Under normal circumstances, the blood pressure fluctuates with each heart beat between a maximum value known as the systolic pressure, which occurs during systole, and a minimum value referred to as the diastolic pressure, attributed to the diastole of the cardiac cycle. The difference between the systolic and diastolic pressure values is known as the pulse pressure. However, the clinically significant blood pressure in critical care areas is the mean arterial pressure (MAP). This is the decisive pressure that reflects the blood flow through the body (see Chapter 6). Although mean arterial pressures are not used in some cardiac areas, it is important that the nurse has some understanding of
the term because these figures appear on the automatic blood pressure recording machines.

The intrinsic regulation of the vascular system ensures that the entire body receives adequate amounts of blood at rest and during various changing physical circumstances. This must involve:

- The maintenance of a minimum blood flow to all organs
- The optimal regulation of cardiac activity and blood pressure
- The redistribution of blood to active organs when required, at the expense of the resting organs.

The regulation of blood flow, SVR and blood pressure are achieved predominantly by alterations in the diameter of the blood vessels. Local effects, neural activity and hormonal signals influence the state of tension (tone) of the smooth muscle of the blood vessels. At rest, most blood vessels are in an intermediate state of tension and this is known as the resting tone. The primary purpose of local control of circulation is to autoregulate the local blood flow and ensure its constancy in the face of constantly changing blood pressure. Furthermore, blood flow must be carefully adjusted in line with the metabolic needs of a specific organ or tissue. The autoregulation may be of myogenic origin (smooth muscle of the vessels) or of metabolic origin, such as through oxygen deficiency or a rise in local tissue metabolites. Both mechanisms will induce vasodilatation, which will contribute towards the solution of the problem. Branches of the sympathetic nervous system mediate neural control of the blood vessels, principally the arterioles. In this fashion, postganglionic transmission involves $\alpha$-1-receptors, the stimulation of which results in vasodilatation, and $\beta$-2-receptors, the stimulation of which results in vasodilatation.

The arterial blood pressure needs to be maintained within an appropriate physiological range, but be able to fluctuate to meet the demands of everyday living. There are three main regulatory mechanisms that assist in blood pressure control. The immediate or short-term mechanism involves the autonomic nervous system. The medium-term control is regulated via a hormonal mechanism, whilst the long-term control is, perhaps surprisingly, under the influence of the renal system.

**Neural control**

Pressure receptors (baroreceptors) are found in the aortic arch in the thorax and carotid sinuses which are situated in the neck. These are strategically placed to monitor any pressure changes in the blood supply to the body (via the aortic arch) and to the brain (via the carotid sinuses). They then transmit afferent impulses to the cardiovascular control centre in the medulla, part of the brainstem. The primary function in this instance is to stabilize the blood pressure. For example, an acute rise in blood pressure will initiate an increase in the firing of afferent impulses. This mechanism will in turn activate the parasympathetic nervous system, thus setting off a reflex response via the vagus nerve. This reduces cardiac activity. In addition, the sympathetic nerve pathway initiates vasodilatation and gives rise to a decrease in SVR. These two responses bring about a lowering of the elevated blood pressure.

Conversely, a drop in blood pressure will be detected by the baroreceptors and sympathetic cardiac and vasoconstrictor nerve activity will be increased, accompanied by a decreased parasympathetic activity. This will result in an increase in heart rate and stroke volume and cause arteriolar and venous vasoconstriction. This then gives rise to an increased cardiac output and SVR. This mechanism elevates the blood pressure to a physiologically desirable value.

**Hormonal control**

As the cardiac output lowers, catecholamines are released from the chromaffin cells of the adrenal medulla. Adrenaline (epinephrine) and noradrenaline (norepinephrine) are the primary catecholamines. The latter has a more specific action on the peripheral vessel $\alpha$-adrenergic receptors, raising the SVR and therefore the blood pressure. Adrenaline has a wider range of effects, including stimulating the $\beta$-1-adrenergic receptors to increase the heart rate and myocardial contractility. The general stimulatory actions will therefore raise the heart rate to increase the cardiac output and promote arterial and venous constriction. In specific areas, the constrictive action in the renal vasculature promotes excessive sodium and water retention. The arterial constriction will raise the SVR and hence the blood pressure and tissue perfusion. Venous constriction will cause a natural augmenting of Starling’s Law of the heart, by shunting blood into the right side of the heart to raise the preload and increase the cardiac output. The above responses are similar to those of the sympathetic nervous system, but they are longer lasting.

**Renal control**

The kidneys control blood pressure in two ways, directly and indirectly. Renal physiology is complex and so this section is only a brief overview, introducing the reader to the kidneys’ effect on the cardiovascular system.

The direct mechanism involves altering blood volume. When blood pressure or volume rises, the kidneys respond by producing more urine. This reduces volume and therefore blood pressure and is the principle for the administration of diuretics in hypertension. On the other hand, if blood pressure or volume drops, water is conserved. This increases volume and therefore blood pressure. Although antidiuretic hormone (ADH), which
is released from the posterior pituitary gland, but produced by the hypothalamus, stimulates the kidneys to save water, this tends to play a more prominent role in blood pressure regulation when there is a large reduction in blood volume. At this stage, ADH also causes vasoconstriction.

The indirect mechanism occurs when there is a reduction in the blood supply to the kidneys. The juxtaglomerular apparatus (JGA) is an area where the distal tubules and afferent arterioles of the kidney lie in close proximity. The afferent arterioles feed blood into the glomerular capsule, part of the nephron that ultimately creates urine. The distal convoluted tubule is the lower part of this nephron coiled back to come into contact with the afferent arteriole vessel (Fig. 4.14). The response to a reduction in blood pressure, volume or osmolarity is complex, but ultimately results in the release of renin from the juxtaglomerular cells of the JGA. This commences what is known as the renin–angiotensin–aldosterone mechanism.

Renin acts to convert circulating angiotensinogen to angiotensin I. This is then converted, notably in the lining of the lungs, to angiotensin II, which is a potent vasoconstrictor, raising the SVR, and causing the blood pressure to rise. In addition, it stimulates the adrenal cortex to release aldosterone, which causes the renal tubules to reclaim sodium from the filtrate. Osmotically, water follows sodium and hence the intravascular blood volume rises to induce an elevation in blood pressure. Additionally, the preload will rise, augmenting Starling’s Law of the heart and hence increasing the cardiac output. Treatments, such as the diuretics, reduce this excessive fluid retention, while the angiotensin-converting enzyme inhibitors are used as long-term therapy to block this chronic effect.

**Conclusion**

This chapter has discussed the anatomy and physiology of the cardiovascular system and aims to assist the reader in applying biological concepts to cardiac nursing practice. Throughout this text, the knowledge will provide a useful underpinning for the nursing practice explored. An understanding of areas such as physical assessment, however detailed this may be, and the ability to evaluate nursing interventions can only be based on a sound recognition of the normal functioning of the cardiovascular system. It will also prove invaluable to understanding the compensatory mechanisms that the cardiovascular system is capable of demonstrating in varying disease processes.

**References**

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Further reading


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