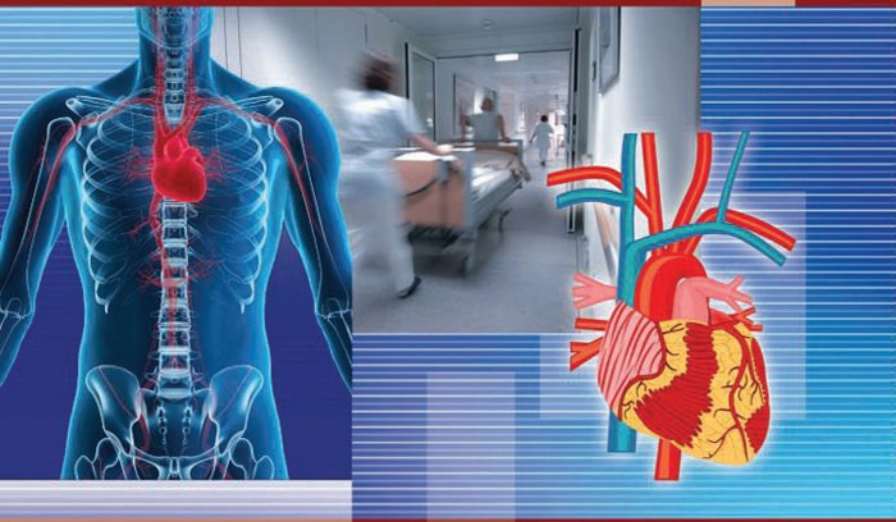
Acute Cardiac Care

a practical guide for nurses

edited by Angela M. Kucia & Tom Quinn



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A Practical Guide for Nurses

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Foreword

As the editors of this book cogently remind us, cardiovascular disease touches the lives of virtually everyone. Nurses are invariably at the forefront, working in collaboration with doctors and other health professionals, in providing acute cardiac care, including prevention and rehabilitation, to patients and their families. They have a professional duty to ensure that the care they give is safe and of a high quality and is informed by the best evidence. This requires them keeping up to date with the rapid developments in science and technology, changes in health policy and planning and increased expectations of the profession and the public whom they serve: a major challenge to busy nurses working in cardiac care settings.

Acute Cardiac Care, edited by two authorities in the field, with contributions from recognised experts (nurses, doctors and a paramedic) from both sides of the world, is therefore a welcome resource that will help meet this challenge. It is certainly a practical guide for nurses, presenting in a highly readable way, the essential topics that pertain to acute cardiac care. Each chapter begins with an overview, learning objectives and key concepts, is interspersed with key points and concludes with learning activities, pertinent references and resources and suggested further reading. It deserves to be in the library of every clinical setting where nurses care for patients with acute cardiac conditions.

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July 2009

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Preface

Cardiovascular diseases touch the lives of millions of people – patients, their families and friends, together with those who provide and plan care, and those responsible for planning and funding care: in essence, all members of society.

Great advances in scientific knowledge have accumulated since the advent of the cardiac care unit (CCU) in the 1960s, stimulated by the work of a British cardiologist, Professor Desmond Julian, who undertook pioneering work in the UK and Australia that changed the paradigm of care for patients with acute myocardial infarction. Much literature has accumulated on the key role nurses played in the development of the CCU in its formative years, and continue to do so in the present day.

But cardiac nursing is not solely about what happens on the CCU. We believe that nurses are crucial to improved prevention, care and rehabilitation of cardiovascular disease. Whether in the emergency department, cardiac care unit, catheter laboratory, cardiac surgical ward, or in the community setting, or as researchers, managers or policy makers, nurses have opportunities to make a real difference.

As two cardiac nurses with a combined total of more than half a century of experience in acute care, research and policy, we have worked with colleagues with a wide range of experience and knowledge from across our two countries to produce what we hope will be a key resource for nurses embarking on studies of this exciting and constantly evolving arena of practice, and serve as a stimulating source of continuing professional development for more experienced colleagues.

We are grateful to all our contributors for their expertise and commitment, and to Magenta Lampson, Senior Commissioning Editor and Rachel Coombs, Development Editor, Nursing, for their invaluable assistance in bringing this project to fruition.

We dedicate this book to our partners, with thanks for their love and support, and look forward to spending more time with them than we have had in the past 2 years while we've been nursing this book!

Angela Kucia
Adelaide, South Australia

Tom Quinn
Surrey, UK.

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1 Mechanics of the Cardiovascular System

B. Greaney & A.M. Kucia

Overview

The cardiovascular system consists of two primary components: the heart and blood vessels. The lymphatic system also has a cardiovascular exchange function but does not contain blood. This chapter will highlight the mechanics of the cardiovascular system and present an overview of the essential elements and structures involved in the flow of blood through the venous and arterial systems. It will also highlight how abnormalities in the mechanics of the cardiovascular system can result in degrees of cardiac disease states.

Learning objectives

After reading this chapter, you should be able to:

- Identify the anatomical location of the heart and its basic function.
- Identify the key structures within the heart, which are involved in the flow of blood through the heart and identify their specific function.
- Define the term 'cardiac cycle' and explain the key physiological changes that occur in the heart during this process.
- Define the terms 'cardiac output' (CO) and 'stroke volume' (SV), and explain their physiological significance in relation to the cardiac cycle.
- Define the terms 'preload', 'afterload' and 'contractility', and explain their physiological impact upon myocardial contraction.

Key concepts

Cardiac cycle; cardiac output; cardiac chambers; cardiac valves; layers of the heart

Basic heart anatomy

The human heart is essentially a muscular pump which delivers blood containing oxygen, nutrients and other vital elements to the body tissues and major organs. The structure and location of the heart was described by Henry Gray in 1918. It is conical in shape, about the size of a human fist and weighs between 230 and 340 g in an adult. The heart is located in the mediastinum, with one-third lying to the right of the sternum and two-thirds to the left. The top of the heart is known as the base, and this is located behind the sternum; the bottom of the heart, known as the apex, is located

in the fifth intercostal space in the mid-clavicular line. The heart is a four-chambered structure – the upper chambers known as the right and left atria, the lower two chambers known as the right and left ventricles, with right and left-sided chambers divided by the septum.

The bulk of the heart's wall is the myocardium, which is a thick contractile mass of cardiac muscle cells. It is the myocardium that provides the force of contraction to move blood out of the ventricles at the end of each cardiac cycle. The heart is surrounded by the pericardium, which is comprised of two principal layers that surround and protect the heart. The outer layer is known as the fibrous pericardium, which is made up of tough and fibrous connective tissue. This layer provides both protection and anchorage for the heart. The second layer, the serous pericardium, is a thinner, more delicate layer and forms two distinct layers around the heart. The outer parietal layer is adhered to the inner side of the fibrous pericardium, whilst the inner visceral layer, also known as the epicardium, is adhered tightly to the myocardium. Between these two layers there exists a potential space termed the pericardial cavity. Within this cavity is a very thin film of serous fluid known as pericardial fluid, which is normally between 15 and 35 mL in volume (Spodick 1997). The key function of this fluid is to reduce friction between the pericardial layers as the heart contracts. The inner layer lining the heart is a continuous sheet of squamous epithelium, continuing into the tunica intima of blood vessels, and is known as the endocardium.

The heart is divided into four chambers: two upper atria and two lower ventricles. These chambers are separated by a set of heart valves termed the atrioventricular (AV) valves; the tricuspid valve separates the right atrium (RA) and right ventricle (RV) and the bicuspid valve or mitral valve separates the left atrium (LA) and left ventricle (LV) (Figure 1.1a). Attached to each AV valve are two structures: the chordae tendinae and the papillary muscles. These two structures are adhered to the walls of each ventricle (Figure 1.1a). Their function is to prevent the valve cusps inverting or swinging upward into the atria during ventricular systole. The key function of the heart valves is to permit the flow of blood in one direction only as it flows through the heart.

The heart can be viewed functionally as two pumps serving the pulmonary and systemic circulations. The pulmonary circulation refers to the flow of blood within the lungs that is involved in the exchange of gases between the blood and the alveoli. Deoxygenated blood returns to the RA via the inferior and superior vena cavae. It then passes through the tricuspid valve to the RV before entering the pulmonary circulation via the pulmonary artery, where gases are exchanged. The pulmonary artery has a pulmonary valve or semilunar valve which opens and closes during contraction and relaxation of the heart, again having a similar function to the AV valves, allowing the flow of blood in one direction only (Figure 1.1). The systemic circulation consists of all the blood vessels within and outside of all organs excluding the lungs. Once oxygenated, the blood returns to the LA via the pulmonary veins and then passes through the mitral valve into the thicker-walled left ventricle, which ejects the oxygenated blood through the aortic valve into the aorta and into the systemic circulation. The aorta also has a valve, the aortic valve, which prevents the backflow of blood during myocardial contraction (Figure 1.1a).

The cardiac cycle

In simple terms, the heart is a pump that receives blood from the venous system at low pressure and generates pressure through contraction to eject the blood into the arterial system. The mechanical action of the heart is created by a synchronised contraction and relaxation of the cardiac muscle, referred to as systole and diastole. The actual mechanical function of the heart is influenced by pressure, volume and flow changes that occur within the heart during one single cardiac cycle.

When the heart muscle contracts (systole) and relaxes (diastole), sequential changes in pressure are produced in the heart chambers and blood vessels, which result in blood flowing from areas of high pressure to areas of lower pressure. The valves prevent backflow of blood. Under normal conditions, this cycle will take place in the human heart between 60 and 100 times per minute.

Figure 1.2a demonstrates the seven phases of the cardiac cycle.

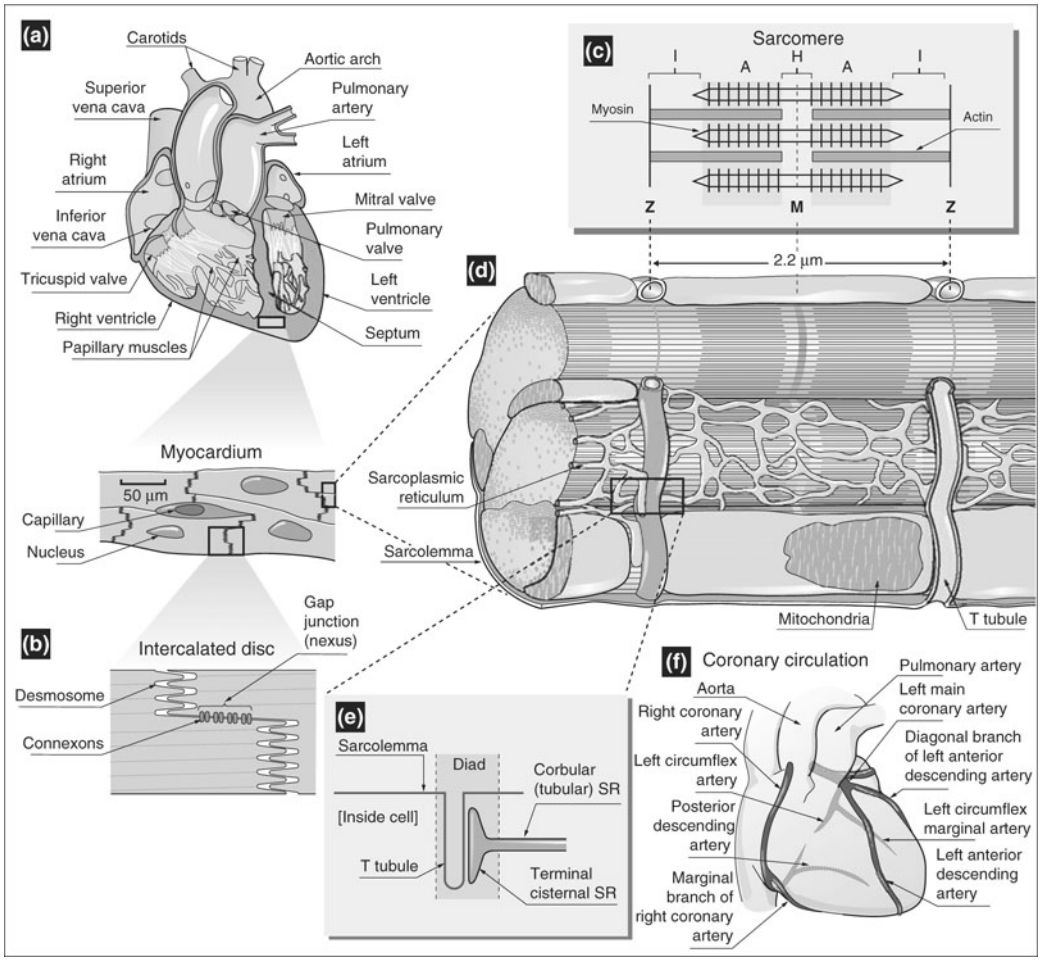

Figure 1.1 Gross anatomy of the heart.
Source: From Aaronson and Ward (2007).

Phase 1: Atrial systole

Atrial systole begins after a wave of depolarisation passes over the atrial muscle. Atrial depolarisation is represented by the P wave on the electrocardiograph (ECG). As the atria contract, pressure builds up inside the atria forcing blood through the tricuspid and mitral valves into the ventricles. Atrial contraction causes a small increase in proximal venous pressure (in the pulmonary veins and vena cavae). This is represented by the 'a' wave of the jugular venous pulse, which is used to measure jugular venous pressure (JVP) (Klabunde 2005).

- Blood flows from the RA across the tricuspid valve into the RV.
- Blood flows from the LA through the mitral valve into the LV.

Pressure in the atria falls and the AV valves float upward. Ventricular volumes are now at their maximum (around 120 mL) and this is known as end diastolic volume (EDV). Left ventricular end diastolic pressure (LVEDP) is approximately 8–12 mmHg; right ventricular end diastolic pressure (RVEDP) is usually around 3–6 mmHg. A fourth heart sound (S4) may be heard in this phase if ventricular compliance is reduced, such

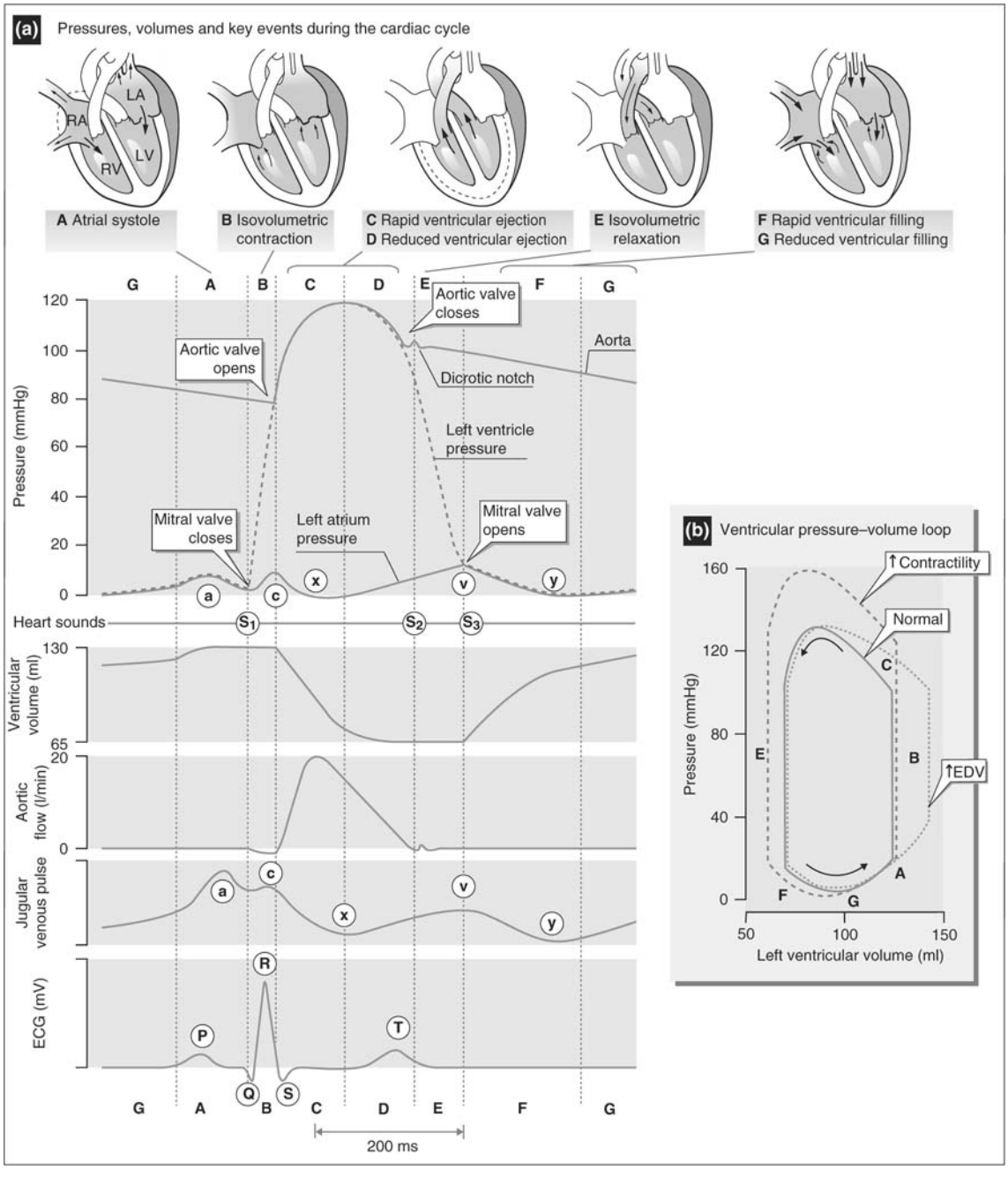

Figure 1.2 Cardiac cycle.
Source: From Aaronson and Ward (2007).

as happens with ventricular hypertrophy, ischaemia or as a common finding in older individuals.

Key point

Ventricular filling occurs passively before the atria contract and depends upon venous return. Atrial contraction normally accounts for only around 10% of ventricular filling, when the body is at rest. However, at high heart rates (such as during exercise), there is a shortened period of diastole where passive filling normally occurs. Under these conditions, atrial contraction is more important and can contribute up to 40% of ventricular filling. Enhanced ventricular filling due to atrial contraction is sometimes referred to as the 'atrial kick' (Klabunde 2005).

Phase 2: Isovolumetric contraction

This phase is represented by the QRS complex on the ECG. The ventricle depolarises and initiates contraction of the myocytes, resulting in a rapid increase in ventricular pressure. This rise in pressure causes the AV valves to close. Closure of the AV valves generates the first heart sound (S1). A split S1 may be heard as mitral valve closure precedes tricuspid valve closure by around 0.04 of a second, although usually only one sound can be heard through a stethoscope. The time between closure of the AV valves and opening of the semilunar valves is known as isovolumetric contraction because there is no change in the volume of blood in the ventricle at this stage, although the ventricle contracts and becomes more spheroid in shape. The pressure in the LV becomes maximal at this stage and is termed dp/dt (maximal slope of the ventricular pressure tracing/time) (Klabunde 2005).

Phase 3: Rapid ventricular ejection

When the ventricular pressure exceeds that of the aorta (around 80 mmHg) and pulmonary arteries (around 10 mmHg) the aortic and pulmonary valves open and blood is ejected out of the ventricles. The LV has a thick muscular wall that allows it to generate high pressures during ventricular contraction. Maximal outflow velocity occurs early in the ejection phase, so the highest aortic and pulmonary artery pressures are reached at this time (Klabunde 2005).

- Blood is ejected from the RV across the pulmonic valve and into the pulmonary artery to the pulmonary circulation.
- Blood is ejected from the LV across the aortic valve and into the aorta to the systemic circulation.

Between 70 and 90 mL of blood is ejected with each stroke (stroke volume), but about 50 mL remains in each ventricle. The residual amount of blood left in the ventricle is known as the end-systolic volume (ESV). Stroke volume thus is the difference between EDV and ESV. Around 60% of the total volume of the ventricle is ejected in each cycle. To work out the ejection fraction of the ventricle, divide the stroke volume by the EDV. The normal left ventricular ejection fraction (LVEF) is above 55% (Klabunde 2005).

Key point

In the healthy heart, no heart sounds should be heard during the ejection phase of the cardiac cycle. The presence of sounds during ejection indicates valvular disease or intracardiac shunts (Klabunde 2005).

Phase 4: Reduced ventricular ejection

The ventricle relaxes and the rate of ejection begins to fall, although kinetic or inertial energy continues to propel the blood forward into the aorta. This phase coincides with ventricular repolarisation, which occurs approximately 150–200 ms after the QRS complex and appears as the T wave on the ECG. Atrial pressure starts to rise during this phase due to venous return (Klabunde 2005).

- The RA receives blood from the systemic circulation via the inferior and superior vena cavae at a low pressure (approximately 0–4 mmHg).
- After circulating through the lungs, blood returns to the heart via the four pulmonary veins into the LA. The pressure in the LA is usually between 8–12 mmHg.

Phase 5: Isovolumetric relaxation

In this phase, the pressure in the ventricles continues to fall and when the point is reached where the pressure is less in the ventricles than that in the outflow tracts (aorta and pulmonary veins), the aortic and pulmonary valves close abruptly, causing a second heart sound (S2). Aortic and pulmonary artery pressures fall slowly due to a combination of stored energy in the elastic walls of these vessels which controls pressure and flow, and because forward flow is impeded by systemic and pulmonic vascular resistance as blood is distributed through the systemic and pulmonary circulations (Klabunde 2005).

Key point

As the aortic valve closes before the pulmonic valve, there is a physiological splitting of the S2 sound and this may be heard with a stethoscope. Closure of the aortic and pulmonary valves result in a characteristic notch in aortic and pulmonary artery pressure tracings (Figure 1.2a). The aortic notch is important in setting timing for intra-aortic balloon counterpulsation.

Phase 6: Rapid ventricular filling

Low pressures in the heart allow blood to passively return to the atria. When the ventricular pressure falls below the atrial pressure, the AV valves open and the ventricles fill quickly. Blood flows into the atria and ventricles throughout diastole with the rate of filling decreasing as the amount of blood in the chambers distends the walls. About 70% of ventricular filling occurs passively at this time.

Key point

No prominent heart sounds should be heard at this time. If a third heart sound (S3) is heard during ventricular filling in adults, it may indicate tensing of the chordae tendinae and AV ring, often associated with ventricular dilation. It is a normal finding in children.

Phase 7: Reduced ventricular filling

There is no clear demarcation as to when this phase begins, but this is a stage during diastole when passive ventricular filling is near completion. As the ventricles fill, they become less compliant, causing intraventricular pressure to rise and the rate of ventricular filling starts to fall. Immediately following this phase, atrial systole occurs following firing of the sino-atrial node.

Key point

At slow heart rates, diastole is lengthened, resulting in increased filling time. In rapid heart rates, there is less filling time. This would compromise CO, if not for compensatory mechanisms.

Cardiac output

CO is an important index of cardiac function, and refers to the amount of blood that is ejected with each contraction (stroke volume) multiplied by heart rate (HR):

$$CO = SV \times HR$$

At typical resting values, if the heart rate is 75 beats/min and the stroke volume is 70 mL/beat, the CO should equal 5.25 L/min. Therefore the body's total volume of blood (4–6 L/min) passes through the body each minute (Saladin 2001).

CO never remains at a constant rate: any factor that alters stroke volume or heart rate will alter CO and it can vary significantly according to normal physical exercise as well as impaired cardiac function. Other factors such as preload, afterload and contractility (inotropy) will indirectly affect CO.

Preload is defined as the actual stretch or tension on the ventricular myocardium prior to contraction (Totora & Gabowski 2002). The greater the preload on the myocardium (the larger the amount of blood that has filled the heart during diastole), the greater the contraction will be. A simple analogy to explain this concept is that the further you stretch an elastic band prior to releasing it, the further it will recoil. The same principle applies here: the greater the stretch or tension on the myocardium, the greater the force of contraction. When venous return to the heart increases,

ventricular filling and preload also increase. The Frank Starling Law of the Heart (Starling's Law) asserts that the more the ventricle is filled with blood during diastole (EDV), the greater the volume of blood that will be ejected (stroke volume) during the ensuing systolic contraction. Thus, altered preload is a mechanism by which the force of contractility can be affected (Klabunde 2005).

Contractility, also known as inotropy, is the ability of a cardiac myocyte to alter its tension development independently of preload changes (Klabunde 2005). Contractility is affected by autonomic innervation and circulating catecholamines (adrenaline, noradrenaline), and additionally changes in afterload and heart rate can augment contractility. A number of pharmacological agents positively or negatively affect contractility. Agents that affect contractility are called positive or negative inotropes, depending upon whether they increase or decrease contractility. Loss of myocardial contractility results in heart failure.

Afterload is defined as the force or pressure against which the ventricular myocardium must push prior to contraction (Totora & Grabowski 2003). This force or pressure is constantly present in the arteries as arterial blood pressure. Therefore, any increase in systemic blood pressure will result in the left ventricular myocardium having to contract more forcefully to eject its volume of blood. Any increase in the pressure of the pulmonary circulation, such as pulmonary oedema, or the presence of any physical obstruction to the pulmonary circulation, such as lung scar tissue, will result in the right ventricular myocardium having to contract more forcefully. In the long term, this increased workload for the myocardium will eventually result in the abnormal enlargement of the myocardium (hypertrophy), which may in turn lead to heart failure.

Key point

The myocardium requires oxygen to regenerate adenosine triphosphate (ATP) that is hydrolysed to produce energy during contraction and relaxation. Any change to the force or frequency of contraction will have an effect on myocardial oxygen consumption (MVO_2). Imbalances in the supply and demand of oxygen to the myocardium may result in myocardial ischaemia or infarction.

Conclusion

This chapter has provided you with an overview of anatomical and physiological underpinnings underlying much of the assessment and nursing care of the patient with a cardiovascular disorder. When next you check a patient's heart rate or blood pressure, or listen to their heart sounds, consider in detail the anatomical and physiological determinants of those measures.

Learning activities

There are a number of interactive online websites where you can test your knowledge of cardiac anatomy and physiology. The Columbia University Medical Center Department of Surgery in New York has some great heart animations and information at http://www.columbiasurgery.org/pat/cardiac/anatomy.html

The Texas Heart Institute at St Luke's Episcopal Hospital Heart Information Center likewise has some good cardiovascular information and animations at http://texasheart.org/HIC/Anatomy/index.cfm

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2 Regulation of Cardiac and Vascular Function

B. Greaney & A.M. Kucia

Overview

Regulation of cardiac and vascular function is somewhat complex and involves autonomic nerves and circulating hormones. You will hear this referred to as 'neurohumoral control of the cardiovascular system'. These mechanisms control cardiac output, blood pressure and local control of blood flow in response to physiological requirements and in the setting of an adverse clinical event such as trauma, disease or stress. In turn, neurohumoral control is influenced by sensors that monitor blood pressure (baroreceptors), blood volume (volume receptors), blood chemistry (chemoreceptors) and plasma osmolarity (osmoreceptors). These sensors work together to maintain arterial pressure at a level that is adequate for organ perfusion (Klabunde 2005). This chapter reviews the mechanisms involved in neurohumoral controls of the cardiovascular system.

Learning objectives

After reading this chapter, you should be able to:

- Describe the components of the autonomic nervous system that relate to cardiac function.
- Describe the effects of sympathetic and parasympathetic stimulation on the cardiovascular system.
- Discuss the function of baroreceptors in the regulation of arterial pressure.
- Discuss the function of chemoreceptors in the regulation of respiratory activity and arterial pressure.
- List the chemicals that can stimulate the heart and cardiovascular system and describe their negative and positive effects.

Key concepts

Neurohumoral control; sympathetic and parasympathetic nervous system; baroreceptors; chemoreceptors; blood pressure regulation

Central nervous system regulation of the cardiovascular system

The central nervous system (CNS) controls the autonomic regulation of cardiovascular function. Autonomic refers to functions of the nervous system that are not under voluntary control (such as regulation of heart rate). The heart is innervated by both parasympathetic and sympathetic nerve fibres. These fibres together play a vital role in the control of heart rate and contractility, as well as

regulation of blood pressure. These nerve fibres are conveyed directly to the heart from the cardiovascular centre located in the medulla oblongata of the brain, which is the main region for nervous system regulation of the heart and blood vessels (Totora & Grabowski 2003). Parasympathetic innervation is associated with the cardioinhibitory centre of the cardiovascular centre, and sympathetic innervation is associated with the cardioacceleratory centre (also known as cardiostimulatory centre) of the cardiovascular centre.

The cardioinhibitory centre sends signals via parasympathetic fibres in the vagus nerve to the sino-atrial (SA) and atrio-ventricular (AV) nodes, conduction pathways, myocytes and coronary vasculature. The right vagus nerve predominantly innervates the SA node, and the left vagus nerve innervates the AV node and ventricular conduction system. Nerve fibres in the parasympathetic nervous system are cholinergic, which means they release acetylcholine. Acetylcholine binds to muscarinic receptors which are specifically associated with vagal nerve endings in the heart, resulting in negative chronotropy (decreased heart rate); negative inotropy (decreased contractility, more so in the atria than the ventricles) and negative dromotropy (decreased conduction velocity).

The cardioacceleratory centre sends signals by way of the thoracic spinal cord and sympathetic cardiac accelerator nerves to the SA node, AV node and myocardium. These nerves secrete norepinephrine, which binds to β-adrenergic receptors in the heart. The term 'pressor' is sometimes used to describe the responses associated with sympathetic stimulation on the heart, which are positive chronotropy (increased heart rate); positive inotropy (increased contractility, more so in the atria than the ventricles) and positive dromotropy (increased conduction velocity).

Key point

It is important to note that despite this continual regulation of the heart, the SA and AV nodes are autorhythmic: they fire at their own intrinsic rate (see Chapter 3 for further detail). Therefore, if parasympathetic and sympathetic nerve fibres to these nodes were severed, the heart would continue to beat at its own intrinsic rate. Parasympathetic activity, or vagal tone, is the dominant controlling factor of heart rate and it inhibits the nodes to a normal rate of 70–80 beats per minute (bpm). Maximum vagal stimulation can reduce the heart rate to as low as 20 bpm (Saladin 2001). In clinical situations, where a patient's heart rate has become dangerously low due to myocardial infarction, ischaemia or other reasons, the drug atropine, a vagal nerve blocker, may be used to block vagal stimulation on the heart, allowing sympathetic nerve fibres to be the dominant nervous stimulus, producing an increase in the heart rate. Parasympathetic activity in the heart inhibits sympathetic activity and vice versa (Klabunde 2005).

The cardiovascular centre receives both neural and chemical input from many sources. Stimuli such as exercise, anxiety, fear, pyrexia and pain will act upon the cardiovascular centre via higher centres in the brain such as the cerebral cortex, the limbic system and the hypothalamus. A number of specific mechanisms exist at various locations in the body which control and regulate the heart and vascular system in response to such factors. Sudden fear or emotion, for example, may cause vagal stimulation resulting in bradycardia, loss of vascular tone and fainting (vasovagal syncope) (Klabunde 2005).

Vasomotor control

As described, the CNS plays an important role in regulating systemic vascular resistance (SVR) and cardiac function which in turn influence arterial blood pressure. The distribution of blood, as well as the control of arterial blood pressure, can be influenced by factors that control changes in the diameter of blood vessels. The vasomotor centre controls sympathetic activation of the vascular system and is located in the medulla of the brain. Sympathetic activation causes an impulse outflow via sympathetic fibres that terminate in the smooth muscle tissue of both resistance (arteries and arterioles) and capacitance (veins and venules) vessels, causing constriction. This increases SVR and thus arterial blood pressure.

Baroreceptors

Arterial blood pressure is regulated through a negative feedback system which uses pressure sensors, known as baroreceptors, located in the carotid sinus and aortic arch and the bifurcation of the subclavian artery (Bridges 2005). These baroreceptors are sensitive to changes in pressure or stretch in the vessels walls where they are located. They are also sensitive to the rate of pressure change and to a steady (mean) pressure.

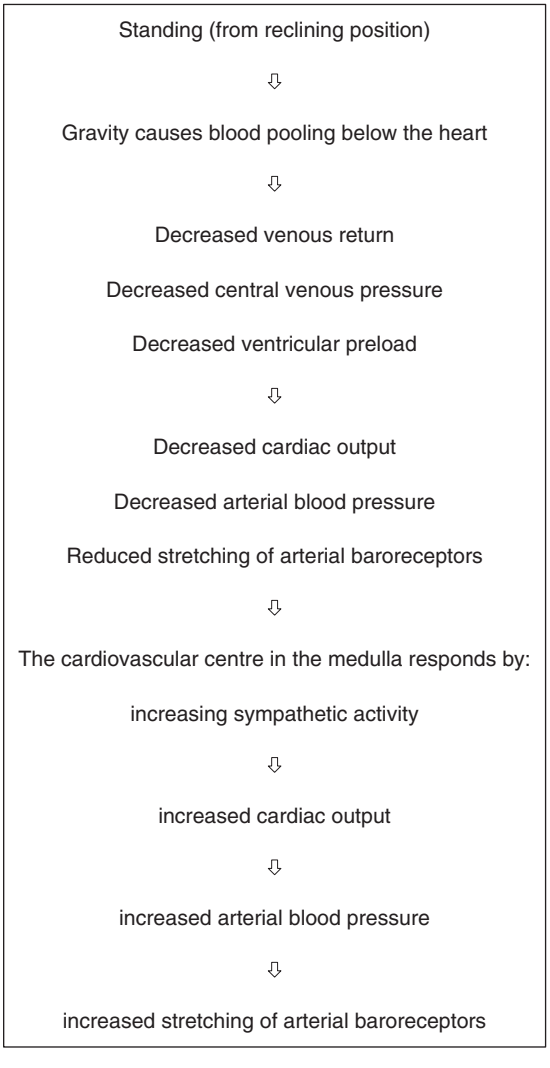

Figure 2.1 Physiological changes to cardiac output associated with body position change.

To understand how baroreceptors function, let us consider what happens in the physiologic circumstance of when a person suddenly changes from a reclining position to one of standing as in Figure 2.1.

In addition to arterial baroreceptors, there are stretch receptors located at the veno-atrial junctions of the heart that respond to atrial filling and contraction (Klabunde 2005). Low-pressure baroreceptors are located in the atria, ventricles, pulmonary artery and veins that are sensitive to changes in transmural pressure in these chambers or vessels.

Learning activity

Carotid sinus massage is sometimes used to abort some forms of supraventricular tachycardia. Considering the action of baroreceptors, how do you think this works?

Clinical states such as hypovolaemia may result in the vascular system recruiting blood from the reservoirs found in the venous plexuses and sinuses in the skin and abdominal organs, especially the liver and spleen (Thibodeau & Patton 2007). Blood can be shifted quickly out of these reservoirs to arteries that supply heart and skeletal muscles when increased activity demands.

Key point

Stimulation of certain mechanoreceptors (sensory receptors that respond to mechanical pressure or distortion), and chemoreceptors in the heart and coronary arteries can result in a vagally mediated triad of bradycardia, apnoea and hypotension (Bridges 2005) known as the Bezold–Jarisch reflex. This happens commonly when dye is injected into the coronary arteries during coronary angiography or during ischaemia/reperfusion involving the infero-posterior wall of the left ventricle.

Chemoreceptors

Chemoreceptors are specialised cells that have a significant role in the regulation of respiratory

activity to maintain arterial blood PO_2, PCO_2 and pH within a physiologic range (Klabunde 2005). These receptors are sensitive to small changes in oxygen levels but are more sensitive to abnormal carbon dioxide and hydrogen ion levels in the blood plasma. Abnormal levels of any of these substances trigger the chemoreceptors to send impulses to the cardiovascular centre. In response, the cardiovascular centre increases sympathetic stimulation to the smooth muscle of arterioles and veins, bringing about vasoconstriction and a subsequent increase in arterial blood pressure and heart rate, thus improving tissue perfusion. Peripheral chemoreceptors are located in the aortic arch (known as the aortic bodies) and in the carotid arteries (known as the carotid bodies), and are responsive to hypoxaemia (decreased arterial PO_2), hypercapnia (increased arterial PCO_2) and hydrogen ion concentration (acidosis). Central chemoreceptors are located within the medulla of the brain (central chemoreceptors) and are responsive to hypercapnia and acidosis but not directly to hypoxia (Klabunde 2005). Stimulation of these receptors leads to hyperventilation and sympathetic activation causing vasoconstriction in most vascular beds except those of the brain and heart (Bridges 2005). Although the chemoreceptor reflex results in an increase in arterial blood pressure, this rise will be mediated by the baroreceptor response.

Key point

Central and peripheral chemoreceptor responses may be enhanced in heart failure patients, resulting in increased sympathetic activation which may contribute to sleep apnoea in those patients and is associated with a poor prognosis (Javaheri 2003; Narkiewicz & Somers 2003).

Humoral control

There are a number of naturally produced chemicals (humoral substances) in the body that significantly effect the action of the heart and vascular system. These can have both positive and negative effects. These include circulating catecholamines, the renin-angiotensin-aldosterone system (RAAS), atrial natriuretic peptide (ANP) and antidiuretic hormone (ADH) (vasopressin). Other substances such as thyroxine, oestrogen, insulin and growth hormone also have direct or indirect effects on the cardiovascular system (Klabunde 2005).

Epinephrine (adrenalin) and norepinephrine (noradrenalin) are classed as non-steroid hormones called catecholamines and are particularly potent cardiac stimulants. They are secreted by the adrenal medulla and cardiac accelerator nerves in response to arousal, stress (physical or emotional) and exercise (Saladin 2001) and are associated with the body's 'fight and flight' reflex. Epinephrine accounts for about 80% of the adrenal medullas secretion, the other 20% is norepinephrine (Thibodeau & Patton 2007). When secreted into the bloodstream, epinephrine prepares the body to respond to an acute stressor by increasing the supply of oxygen and glucose to the brain and muscles, while suppressing other non-emergency bodily processes such as digestion (fight or flight mechanism). It binds to numerous adrenergic receptors (β_1, β_2, α_1 and α_2) throughout the body, although it has a greater affinity for β-adrenoreceptors than α-adrenoreceptors. Therefore, when plasma levels of epinephrine are low, it will bind preferentially to β-adrenoreceptors. This is important to know because heart rate, inotropy and dromotropy are mainly mediated by β_1-adrenoreceptors (Klabunde 2005). Low dose epinephrine binds to β_2-adrenoreceptors in skeletal muscle and splanchnic arterioles, triggering vasodilation. However, when epinephrine binds with α-adrenergic receptors that are found in smooth muscle in the walls of blood vessels, it causes vasoconstriction. Blood pressure is increased due to the resulting increase in cardiac output and SVR.

Key point

When epinephrine is administered exogenously, its effects are dose related. Low dose epinephrine stimulates the β-adrenoreceptors resulting in vasodilation and increased heart rate and contractility. Higher doses stimulate the α-adrenoreceptors, increasing vascular resistance and blood pressure. Thus, if the intent of epinephrine administration is vasoconstriction, it is important to administer a large enough dose to achieve this effect (Bridges 2005).