Chapter

Physiology and Pathology of the Cardiovascular System

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Abstract

The cardiovascular system (CVS) is made up of the heart, blood vessels, and blood. The fundamental function of CVS is to transport substances to and from all parts of the body. The heart is the major pumping organ, pressurizing blood for circulation through the blood vessels; blood is propelled away from the heart in the arteries and returns to the heart through the veins. Cardiovascular disease (CVD) is an umbrella term for a number of inter-linked diseases, generally defined as coronary artery disease, cerebrovascular disease, high blood pressure, peripheral arterial disease, rheumatic and congenital heart diseases, arrhythmia, etc. Globally, CVDs are the leading cause of deaths, and according to the estimation of the World Health Organization (WHO), about 17.9 million people died from CVDs in 2019, accounting for 32% of all global deaths. About 75% of CVD deaths occur in low- and middle-income countries. This burden of CVDs can be decreased by careful risk reduction (such as lifestyle modification, smoking and alcohol cessation, weight optimization, physical exercise), and proper medical treatments, including herbal components. The prevention of CVDs can reduce the occurrence of major cardiovascular events, thereby reducing premature disability, morbidity, and mortality, while prolonging survival and quality of life.

Keywords: cardiovascular system (CVS), heart, coronary artery disease (CAD), physiology, pathology, cardiovascular drugs, herbal components

1. Introduction

The cardiovascular system acts as the engine that drives the human body. It is responsible for transporting oxygen, nutrients, hormones, and enzymes throughout the body, as well as removing carbon dioxide and other waste products from it [1]. This cardiovascular system is mainly separated into two parts—(i) the pulmonary circulation and (ii) the systemic circulation, which are supplied by the right and left ventricles of the heart, respectively [2]. Each of these circulations is constituted of the respective heart pump, the microcirculation, the arteries, and the veins. Basically, the cardiovascular system is a well-regulated carrier syntax of the body that allows the circulation of blood throughout an intact system under varying pressure gradients, generated by the pumping mechanism, with the heart serving as the core pumping unit [3]. This heart is a super sophisticated and highly developed organ that integrates

a diverse range of anatomical and functional features to fulfill its fundamental pumping function. It is not just a sophisticated information processing and encoding center [4], but it also functions as an endocrine gland that is capable of generating and releasing its hormones and neurotransmitters [5–8]. The heart is positioned in the center of the chest, between the lungs in humans. An average human heart measures around the size of a clenched fist and its mass falls within the range of 250–350 grams, with a typical beating of around 100,000 times per day (approximately 72 beats per minute (bpm)) [9]. The interior anatomy of the heart exposes four myocardial chambers—two atria and two ventricles. The two atria are the upper chambers that primarily serve as the collecting chambers; whereas the two ventricles are the lower chambers that primarily function as the blood-pumping chambers [10]. A healthy heart has a set of four valves that keep blood flowing in one direction to prevent backflow. The rate and strength of the heart's contractions dictate cardiac functioning [4]. The cardiovascular system entails the blood vessels as well, which circulate the blood pumped by the heart throughout the body. Since the heart and blood vessels are integral parts of the cardiovascular system, any damage or dysfunction of the heart or blood vessels can have catastrophic repercussions, leading to severe cardiovascular diseases (CVDs) and even death [11].

CVDs encompass a wide spectrum of disorders, including diseases of the heart muscle and the vascular system that supplies the brain, heart, and other vital organs with blood and oxygen. CVDs are the preeminent cause of death worldwide, claiming the lives of an estimated 17.9 million people annually [12], with the majority (80%) of these deaths occurring in developing nations [13]. The prevalence of CVDs is projected to increase as their risk factors become more pervasive in formerly low-risk countries. The death toll from CVD is currently three times higher in developing countries than that in developed ones [14]. According to the estimation of the World Health Organization (WHO), more than 75% of premature CVDs are avertible and the mitigation of the risk factors can assist to deal with the rising burden of CVDs [15]. Moreover, WHO has emphasized the importance of lifestyle factors such as unhealthy diet habits, tobacco use, psychological stress, and physical inactivity contributing to the rise of CVD, and according to the estimation of WHO, three-quarters of deaths caused by CVDs might be avoided with united efforts [16]. Furthermore, an early diagnosis of CVDs is pivotal for reorienting the focus of therapy toward prevention rather than treatment [17]. However, in recent years rapid progress is being made in the treatment of heart diseases. Several therapeutic choices are constantly being presented to cardiologists caring for patients with CVDs . The most frequently prescribed drugs for CVDs include beta-blockers, angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARB), alpha-adrenoceptor blockers, adrenergic receptor blockers, antihypertensive drugs, vasodilators, nitrates, calcium channel blockers, potassium channel activators, diuretics, positive inotropic drugs, antiarrhythmic drugs, sympathomimetic drugs, anticoagulants and protamine sulfate, antiplatelet drugs, fibrinolytic drugs and lipid-lowering agents etc. [18]. According to the Bangladesh Unani, Ayurvedic and Herbal pharmacopeia, a lot of plant-derived medications are also applied for the treatment of these ailments.

This book chapter aims to present a succinct and simplistic review of the basic physiological and anatomical aspects as well as the pathological information regarding the cardiovascular system. Additionally, this chapter includes information on the diagnosis, treatment, and prevention of CVDs.

2. Cardiovascular system

Every living body relies on a functioning cardiovascular system, which is a complex and multifaceted physiological system with numerous regulatory sub-systems controlled by the central and peripheral autonomic nervous systems as well as humoral factors [19]. The cardiovascular system is primarily responsible for supplying the body's cells with the materials they require to function properly and for removing the waste products that they make as a result of their metabolic processes. It is responsible for transporting blood throughout the body. It is controlled by numerous stimuli, including sympathetic and parasympathetic nervous systems, changes in blood volume, electrolytes, hormones, osmolarity, adrenal glands, kidneys, medications etc. [20–23].

The fundamental role of the cardiovascular system is to meet the metabolic requirements of the body as well as transporting carbon dioxide and other wastes out of the body. This function is accomplished in two ways—by maintaining a healthy circulatory system and by keeping blood pressure at an optimum level.

2.1 Divisions of the cardiovascular system

The circulatory system has two functionally opposite divisions through which blood flows—systemic circulation and pulmonary circulation as shown in **Figure 1**.

2.1.1 Systemic circulation

This circulation is more generally referred to as greater or superior circulation with a highly elevated resistance circuit [3]. It commences at the left ventricle and terminates in the right atrium [3]. The left ventricle pumps blood which is traveled through a set of blood vessels known as the arterial system [24]. At the capillaries, blood and tissue exchange a variety of substances. Following such exchange, blood turns back to the right atrium of the heart via the venous system. After that, the right ventricle receives blood from the right atrium. As a consequence of this systemic circulation, the oxygenated blood from the heart travels to the tissues, while venous blood from the tissues returns to the heart [3].

2.1.2 Pulmonary circulation

This circulatory system is known as the lesser circulatory system with a lower resistance circuit [3]. Such circulation begins at the right ventricle and terminates in the left atrium [3]. At first, the right ventricular blood flows to the lungs via the pulmonary artery [24]. By means of the pulmonary capillaries, the exchange of gases takes place between the circulating blood and the lungs' alveoli [24]. Once the blood has been oxygenated, it returns to the left atrium via the pulmonary veins.

2.2 Components of the cardiovascular system

The cardiovascular system is primarily composed of—(i) heart and (ii) blood vessels (i.e. capillaries, arteries, and veins).

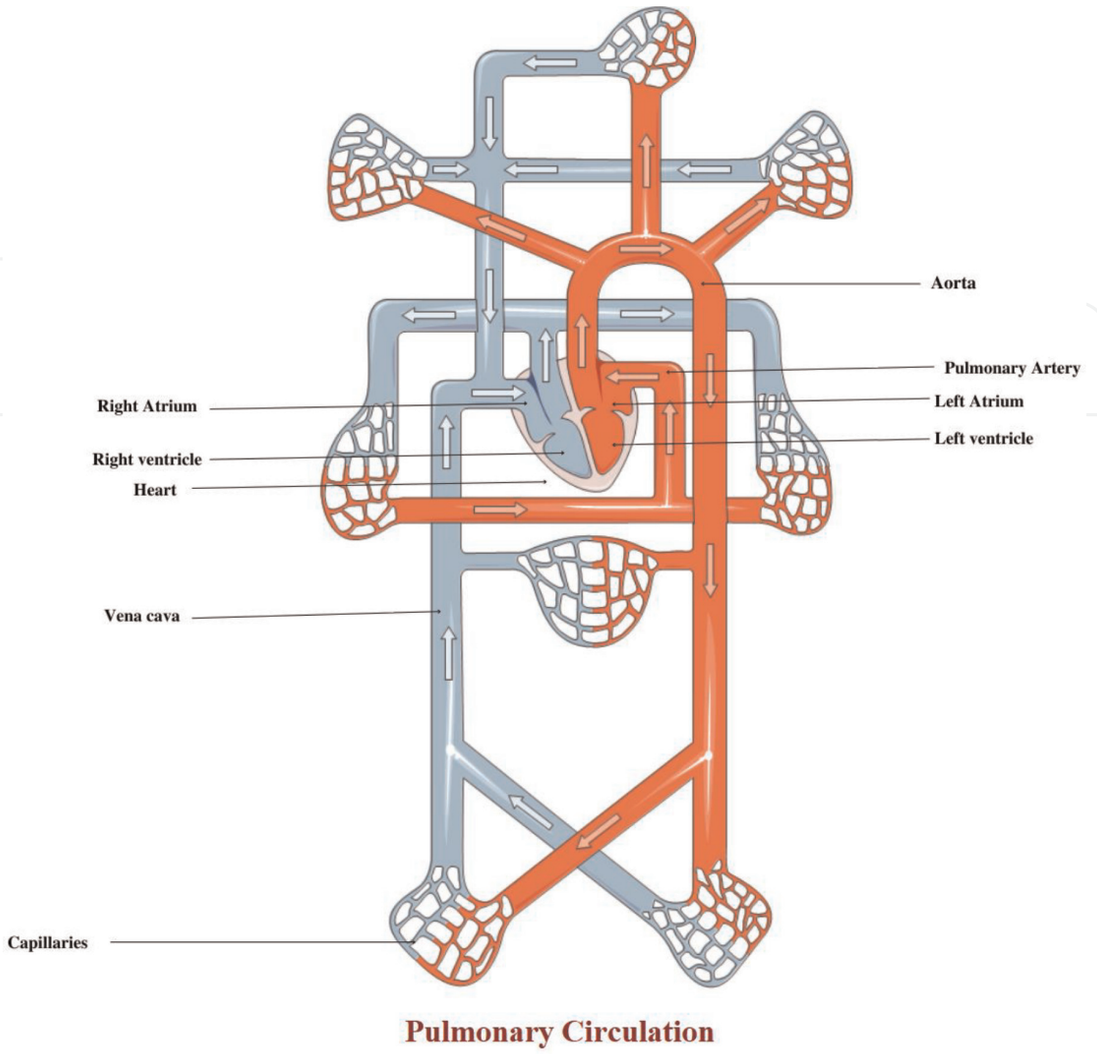


Figure 1.
Circulatory system of a heathy heart (designed with biorender).

3. Healthy heart

A healthy heart serves as the chief pumping unit of the cardiovascular system. In a healthy state, the muscular heart performs two key functions. Firstly, a healthy heart takes oxygen-depleted blood from the tissues and pumps it to the lungs, where the lungs pick up oxygen and discharge carbon dioxide. The second function of a healthy heart is to draw oxygen-rich blood from the lungs and deliver it throughout the body. The heart also removes interstitial fluid from the bloodstream and transports it to the extracellular space through systemic circulation.

4. Anatomical and physiological aspects of a healthy human heart

4.1 Structural features and anatomical position of healthy heart

The structure of a healthy human heart roughly resembles the shape of the heart on a playing card (**Figure 2A**) [3], with around two-thirds of its mass located to the left of the midline [10]. The human heart lies obliquely in the thorax which shields the

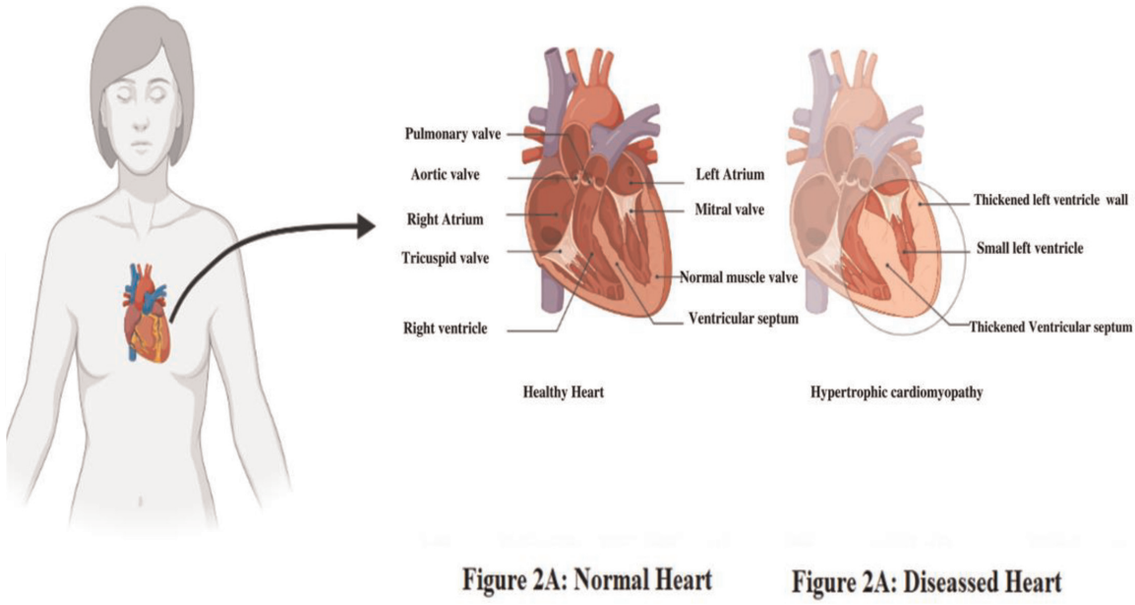


Figure 2.
Healthy heart (A) vs. diseased heart (B) (designed with biorender).

delicate anatomical structure of the heart. It is positioned roughly on a plane that spans from the right shoulder to the left nipple of the body [10]. Its anterior surface confronts the sternum, whereas its posterior surface confronts the vertebral column. However, the inferior surface of the heart is supported by the superior surface of the diaphragm. The human heart is housed in an enclosed region within the pleural cavities known as the middle mediastinum, which refers to the inner space of the pericardium, the protective sac that covers and cushions the heart as well as keeps the heart separated from other parts of the chest, including the lungs [9]. This pericardium is a serous membrane, which consists of a fibrous thick outer layer (the parietal pericardium) and an inner layer (visceral pericardium) separated by a lubricating substance known as the "serous fluid" (~25–35 ml), that aids to "glide" the inner visceral pericardium against the outer parietal one [2, 10]. This pericardium serves to limit the heart's ability to expand excessively.

4.2 Layers of the heart wall

There are three distinct layers that make up the walls of a healthy human heart (as illustrated in **Figure 3**). These are—(i) Superficial Epicardium (ii) Middle Myocardium, and (iii) Inner Endocardium.

i. Superficial Epicardium: This layer is the most external to the heart. It is the visceral layer of the pericardial sac, which constitutes the innermost layer of the serous pericardium. An exterior layer of flat mesothelial cells forms this epicardium, with a layer of adipose and connective tissue lying beneath [25]. This inner layer shields the heart and it directly connects the epicardium to the muscular myocardium. This epicardium houses the blood vessels and nerves that furnish the heart [25]. At the base of the great vessels, the epicardium extends as the pericardial sac, composing an enclosed pericardial cavity.

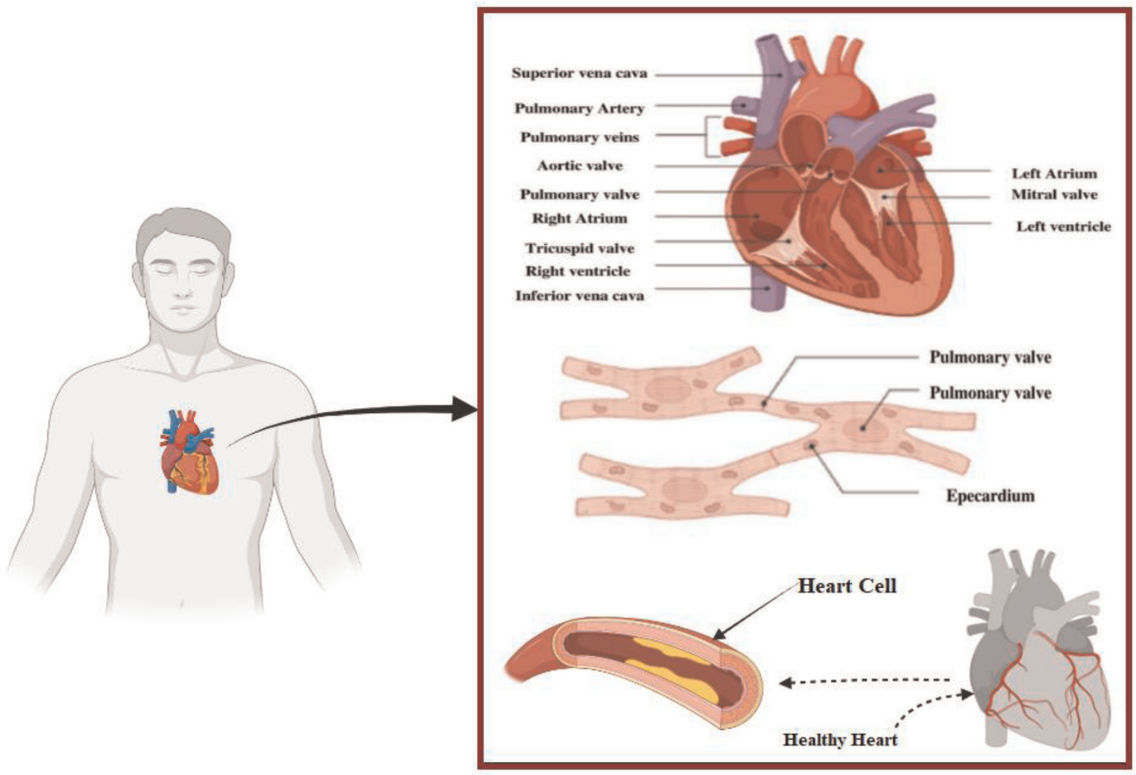


Figure 3.
Physiology of healthy human heart (designed with biorender).

ii. Middle Myocardium: The myocardium is the thickest of the three heart layers and it is the primary functioning component of the heart. The pumping action of the heart is made possible by this layer, which allows the heart to contract. This layer is primarily made up of capillaries, collagen fibers, and cardiomyocytes. The cardiomyocytes are arranged in a spiral pattern in the myocardium to press the blood into an appropriate trajectory throughout the heart [10]. These cardiomyocytes possess a high concentration of mitochondria and glycogen deposits, which has a tremendous functional eminence since this layer is contracting persistently, requiring a lot of energy all the time.

iii. Inner Endocardium: It is the inner layer of the heart wall. This layer is a smooth, thin gleaming membrane. This endocardium consists of an endothelium that is connected to the endothelium of blood vessels and connective tissue that fuses with the muscular myocardium [10, 24]. This layer forms the heart valves and serves as the lining of heart chambers.

4.3 Chambers of the heart

A healthy human heart is composed of four chambers—right atrium, right ventricle, left atrium, and left ventricle (**Figure 3**). Therefore, humans have two sides of their hearts—the right and left parts.

4.3.1 The right part of the heart

This section is made up of the right atrium and the right ventricle. The wall of the right atrium is quite thin, and this chamber remains under low pressure [24]. The

right atrium carries the pacemaker called the SA (sinoatrial) node, which generates cardiac impulses, as well as the AV (atrioventricular) node, which transmits electrical signals to the ventricles [22]. This right atrium collects the deoxygenated (venous) blood from the whole body via the superior and inferior vena cava and the coronary sinus [3]. This right atrium is connected to the right ventricle via the tricuspid valve, through which deoxygenated blood enters the right ventricle from the right atrium. The pulmonary trunk, which originates in the right ventricle, transports the deoxygenated blood from the right ventricle to the lungs, where it is oxygenated [24]. The right ventricle has a thicker wall than that of the right atrium, but it's less muscular compared to the left ventricular wall.

4.3.2 Left part of the heart

This part is made up of the left atrium and the left ventricle. The left atrium is a low-pressure and thin-walled chamber [24]. Pulmonary veins deliver the oxygenated blood from the lungs to the left atrium, from where the blood moves to the left ventricle via the bicuspid or mitral valve [24]. The arterial blood is pumped by the left ventricle throughout the body via the systemic aorta.

However, the two right chambers are detached from the left ones by a constant divider, the ventricular section of which is known as the interventricular septum whereas the atrial portion is called the interatrial septum [3]. The interatrial septum is fibrous in nature, but the interventricular septum possesses dual structural characteristics, with the upper one-fourth segment being fibrous and the lower three-fourth section being muscular.

4.4 Valves of the heart

The human heart possesses four valves, each with a distinct function. Two of them are situated within the junction of the atria and the ventricles. These two valves are termed as the atrioventricular valves. On the other hand, the remaining two valves are located at the aperture of blood vessels originating from the ventricles, i.e., the pulmonary trunk and systemic aorta. These two valves are termed as semilunar valves. Four of these heart valves ensure the unidirectional flow of blood across the heart [26].

4.4.1 Atrioventricular valves

The right atrioventricular orifice is shielded by the tricuspid valve, which is composed of three cusps or flaps, i.e., infundibular or anterior cusp, medial or septal cusp, and marginal or posterior cusp [3]. Conversely, the left atrioventricular orifice is protected by the bicuspid valve, which is composed of two valvular cusps, namely, the anterior cusp and the posterior cusp [3]. This bicuspid valve is also known as the mitral valve because of its similarity to the miter of a bishop. The cusps of both of these valves are triangular in appearance and are linked to the edges of the fibrous or dense connective tissue surrounding the atrioventricular openings.

4.4.2 Semilunar valves

The aperture within the pulmonary artery and the right ventricle is guarded by the pulmonic semilunar valve, while the opening between the systemic aorta and the left

ventricle is guarded by the aortic semilunar valve that is stronger and larger compared to the other one [3]. Both of these valves have a shape that resembles a half moon, because of which these are referred to as semilunar valves. These valves are composed of three cusps. The pulmonary semilunar valve is composed of the left anterior cusp, right posterior, and left posterior cusp; whereas the aortic semilunar valve is made up of the right anterior cusp, right posterior, and left posterior cusps, respectively [3].

4.5 Special junctional tissues of the healthy heart

A healthy heart muscle is primarily made up of some specialized structures that play critical roles in commencing and transmitting impulses at a rate that is significantly faster than that of the remaining muscles. These structures are inclusively termed as the "Junctional Tissues of the Heart". They consist of the following structures—(i) S.A. (sino-atrial) node, (ii) A.V. (atrioventricular) node, (iii) Bundle of His, (iv) Bundle branch, (v) Purkinje fibers. A brief description is given below:

i. S.A. node: It is located in the right atrium of the heart at the intersection of the right auricular appendage and superior vena cava [3]. It is wider at the top and tapers toward the bottom, measuring approximately 5 × 20 mm. It functions as the heart's natural pacemaker and produces impulses at a rate of 70–80 bpm in adults [3, 24, 26]. The rhythm initiated from the S.A. node is commonly referred to as the sinus rhythm.

ii. A.V. node: It is located in the right atrium, near the opening of the coronary sinus, in the posterior portion of the interatrial septum [3]. It has a measurement of approximately 2 × 5 mm. It serves as the reserve pacemaker of the heart. It accepts the impulses generated by the SA node and conducts it to the ventricles at a rate of 40–60 bpm via the bundle of His [24, 26]. The rhythm emerging from this node is termed as the nodal rhythm.

iii. Bundle of His: The main trunk of this bundle is continuous with the A.V. node and passes upwards until it reaches the posterior margin of the membranous part of the interventricular septum and then forwards below it [3]. It is approximately 20 mm in length.

iv. Purkinje fibers: The branches of the bundle of His give rise to these fibers, which travel through the papillary muscle and lateral ventricular walls, eventually terminating in the subendocardial network of the heart [3]. The primary function of these fibers is to instantly transmit impulses to all parts of the ventricular muscle fiber. Atrioventricular dissociation can cause these fibers to fire at a rate of 30–35 bpm [3, 24, 27].

v. Bundle branch: Immediately above the muscular portion of the septum, two branches (right and left) of the bundle are visible. Both of these branches remain just below the endocardium [3]. The right branch of the bundle travels along the right side of the septum and is comparatively longer than its left counterpart. On the contrary, the left bundle branch travels along the left side of the septum, bifurcating into inferior and superior sections, and culminates in the Purkinje system, which is located within the ventricular subendocardial tissue [3]. The atrial impulse is normally carried to the

ventricles by these bundle branches. These branches can generate cardiac impulses at a rate of 36 beats/min in the event of failure of the S.A. and A.V. nodes.

5. Physiological properties of heart muscle

Heart muscle possesses certain special features. These include:

i. Rhythmicity

ii. Conductivity

iii. Contractility

iv. Excitability

v. All or None Law

vi. The Staircase Phenomenon

vii. Refractory Period

viii. Tone

A summary of all these properties is given below:

i. Rhythmicity: It is the capacity of a cardiac tissue to generate its own impulses regularly. This property is also known as self-excitation or autorhythmicity. All of the tissues of the heart own this feature. However, in the human heart, there is an exclusive excitatory structure that produces rapid electrical impulses. This exclusive structure is termed as the "pacemaker of the heart". The sinoatrial node (SA node) serves the purpose of the pacemaker in the mammalian heart. From this node, the impulses propagate to other portions of the heart through a specific conductive system. Moreover, the AV node, the atria, and the ventricles of the heart are also capable of generating impulses and can perform as pacemakers. In spite of this, SA node is referred to be the pacemaker because of its high rate of impulse generation capacity compared to others.

   The rhythmicity of different portions of a healthy human heart is shown in **Table 1**.

ii. Conductivity: The human heart possesses a unique conducting system, which is constructed by specialized cardiac muscle fibers known as internodal fibers. These fibers are responsible for the quick transmission of impulses from the SA node to other parts of the heart. The fundamental elements of the conductive system in the heart include—the AV node, Purkinje fibers, right and left bundle branches, and bundle of His. These conductive tissues are also referred to as the junctional tissues of the human heart. The conductivity of the heart muscle is maintained in the following way:

Portions of human heart	Rhythmicity
SA node	70–80 bpm
AV node	40–60 bpm
Purkinje fibers	30–35 bpm
Atrial muscle	40–60 bpm
Ventricular muscle	20–40 bpm

Table 1.
Rhythmicity of different portions of a healthy human heart.

Components of conductive system	Velocities of impulses
SA node	0.05 m/s
AV node	0.1 m/s
Purkinje fibers	1 m/s
Bundle of His	1 m/s
Ventricular muscle fibers	0.4 m/s

Table 2.
The velocities of impulses at various portions of the conductive system.

At first, the AV node receives the impulses from the SA node through the internodal fibers. The AV node then sends these impulses to the ventricles via the bundle of His and its branches. The Purkinje fibers further carry these impulses from the top of the heart down to the base.

The velocities of impulses at various portions of the conductive system of the heart are given in **Table 2**.

iii. Contractility: Contractility of the heart muscle refers to its ability to shorten or contract in length in response to a stimulus. Myofibril is the core contractile unit of the heart muscle, which is made up of actin and myosin. These two units are linked together under the presence of ATP during contraction, which causes the fiber to be shortened. However, at the time of rest, these two units become dissociated as the ATP is resynthesized.

As it pertains to contractility, Starling's Law of the heart or Frank-Starling Law has been codified. This law states that the heart has the ability to modify its stroke volume and force of contraction in response to changes in venous return.

iv. Excitability: It is the capacity of the cardiac muscle to generate a propagated action potential in response to a stimulation that is sufficiently strong.

v. All or None Law: The cardiac muscle strictly obeys this law. In accordance with this law, whenever a stimulus is provided, regardless of its strength, the entire heart muscle either responds to it at its maximum capacity or there is no response at all.

vi. The Staircase Phenomenon: When the heart muscle is excited with sequential maximal stimuli, the initial few contractions display a progressive increase in magnitude, which is referred to as a staircase. Following that, the strength of contraction stabilizes at its regular level.

Refractory Period: The refractory period of the heart is the duration of time during which a typical cardiac impulse cannot re-excite a section of cardiac muscle that has already been stimulated or excited. It is of two types: absolute refractory period and relative refractory period.

Absolute Refractory Period: In the absolute refractory period, the muscle does not respond at all, no matter how strong the stimulus is. The reason is that depolarization is taking place at the time. As a result, there cannot be a second depolarization.

Relative Refractory Period: During this period, the muscle responds if the power of stimulus is maximum. At this point, the muscle is in a repolarizing state.

The comparative refractory periods of different portions of the heart muscle are given in **Table 3**.

vii. Tone: The heart muscle of humans has tone. This tone is nerve-independent and adjustable. This allows the heart to keep reasonably constant tension on its different contents.

6. Cardiac cycle of healthy heart

A single cardiac cycle consists of two primary phases- diastole and systole. The term "diastole" denotes relaxation, and the term "systole" refers to contraction [3].

6.1 Diastole

During the diastolic phase, blood flows into the right atrium from the superior and inferior vena cava, which elevates the internal pressure of the right atrium. When the right atrial pressure surpasses the right ventricular pressure, the tricuspid valve passively opens, enabling the blood to move toward the right ventricle. Simultaneously, the oxygen-rich blood from the lungs returns to the left atrium, resulting in an

Portions of heart muscle	Refractory period
SA node	Highest
AV node	Lower than S.A node
Ventricular muscle fibers	Lower than nodal
Atrial muscle fibers	Lower than ventricular
Purkinje fibers	Lower than ventricular

Table 3.
The comparative refractory periods of different portions of the heart muscle.

escalation in left atrial pressure. This leads to the opening of the bicuspid valve, which allows the passage of blood from the left atrium to the left ventricle.

6.2 Systole

During systole, the right and left ventricles contract and discharge blood into the pulmonary trunk and the aorta respectively. At this time the pulmonic and aortic heart valves open to allow the passage of blood into the pulmonary artery and aorta. However, the bicuspid and tricuspid atrioventricular valves remain closed during this period. But during the closure of these two valves, the first cardiac sound, i.e. the "lub" sound is generated at the beginning of the ventricular systole; and at the end of the ventricular systole, the second heart sound, i.e. the "dub" sound is generated due to the closure of the pulmonic and aortic valves.

So, each cardiac cycle comprises the full contraction and relaxation of both the atria and ventricles and lasts around 0.8 s.

The time periods of the atrial and ventricular events of the cardiac cycle are given below:

Atrial events	• Atrial systole	0.1 s
	• Atrial diastole	0.7 s
Ventricular events	• Ventricular systole	0.3 s
	• Ventricular diastole	0.5 s

7. Cardiac output of heart

The volume of blood that a healthy heart pumps in 1 min is termed as the cardiac output of the heart. Logically, the cardiac output (CO) is equal to the product of the stroke volume (SV) and heart rate (HR). It is expressed in l/min.

$$\mathrm{CO}\ (\mathrm{ml}/\mathrm{min}) = \mathrm{SV}\ (\mathrm{ml}/\mathrm{beat}) \times \mathrm{HR}\ (\mathrm{beats}/\mathrm{min})$$

8. Pathology of cardiovascular system

8.1 Diagnosis of cardiovascular diseases

Diagnosis of CVDs is often conducted by the following approach

i. Assessment of risk factors, medical, and family history of the patient.

ii. Physical Examination: Physical examinations can assist in evaluating an individual's risk of developing heart disease. Complete cholesterol testing is the most generally recommended physical examination to determine susceptibility of CVDs.

iii. Electrocardiogram (ECG): ECG helps to monitor the electrical activity of the heart painlessly. The activities of the heart are recorded on graph paper via a portable small machine. ECG aids in detecting the incidence of arrhythmias, angina, and heart attacks.

iv. Echocardiogram: It is a type of ultrasonography of the heart. It creates an image of the heart using sound waves. It may be used by doctors to examine the conditions of the heart muscles and heart valves of the patient.

v. Chest X-ray: In this test small doses of radiation are used to provide high-resolution pictures of the chest and heart. The causes of chest discomfort can be identified with the help of this test.

vi. Magnetic Resonance Imaging (MRI) of the Heart: An MRI uses radio waves and large magnets to produce internal images of the body. During this test, a technician generates images of the heart and blood vessels as it beats. The captured pictures can help doctors in diagnosing diseases of the coronary artery and heart muscle.

8.2 Risk factors of cardiovascular diseases

The risk factors of CVDs can be categorized into two classes: non-modifiable and modifiable risk factors (**Figure 4**). Non-modifiable risk factors are those that cannot be altered or controlled. These include- ethnicity, race, age, gender, and genetic factors of an individual. On the contrary, modifiable risk factors are those that can be altered or controlled by modifying the lifestyle of an individual. For example—

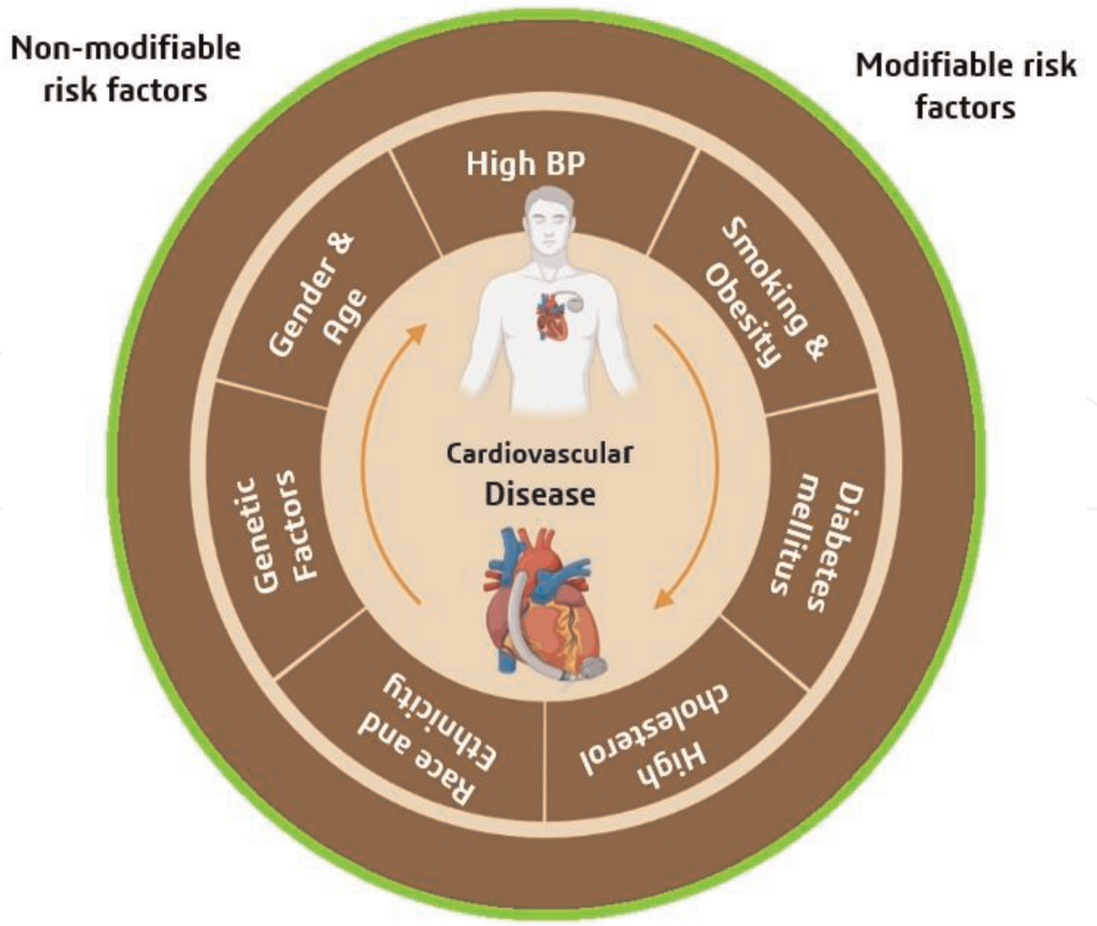


Figure 4.
Risk factors of cardiovascular diseases (designed with biorender).

smoking cessation, weight control, proper maintenance, and control of lifestyle diseases (e.g. diabetes, hypertension) can significantly reduce the risk of CVDs.

8.3 Classification of cardiovascular diseases

Heart disease is of basically two types

i. Coronary Artery Diseases (CAD) and

ii. Arrhythmia.

iii. Brief descriptions of both of these diseases are given below:

8.3.1 Coronary artery diseases (CADs)

Coronary artery disease (CAD) or coronary heart disease (CHD) is one of the most pervasive forms of CVD which is considered to be the preeminent cause of mortality in both first-world and third-world countries [28]. According to the estimation from a study, CAD accounts for 32.7% of overall CVDs and 2.2% of the total burden of maladies worldwide [29]. CAD is an inflammatory atherosclerotic disease [27]. It is a multifarious human disorder in which there is an insufficient transmission of oxygen and blood to the cardiac muscle, resulting from a blockage of the coronary arteries. It is often characterized by plaque deposition within the lumens of coronary arteries, which obstruct blood flow [30]. Hence CADs are linked to impaired circulation to the heart via the coronary artery. There are various forms of CAD. Among these are:

a. Myocardial Ischemia and Reperfusion: Ischemia and reperfusion is a diseased state marked by a temporary reduction in blood flow to an organ, followed by a successive restoration of perfusion and accompanying reoxygenation [31]. Myocardial ischemia develops when the oxygen requirements of the heart muscle are greater than the amount of oxygen that is available to the heart. Unless this situation is remedied, cell damage is likely to occur. Ischemic myocardial cells can have their oxygen and energy substances restored when the ischemic myocardium is repercussed [32] during procedures such as coronary artery bypass surgery, thrombolysis, or angioplasty. However, this process may create another form of myocardial damage, which is referred to as "reperfusion injury".

b. Angina Pectoris: Angina pectoris is caused by a lack of oxygen supply in the myocardium. There are three clinically distinct types of angina pectoris, each with its own pathogenesis:

- Stable or typical angina
- Prinzmetal's variant angina and
- Unstable or crescendo angina.

A brief description of each type is given below:

- Stable or typical angina: It is the most typical form of angina. It is marked by the sudden attack of chest pain or bouts of discomfort after emotional stimulation or physical activity, which are assuaged by rest. The pathogenesis of such a situation lies in atherosclerosis which is chronically stenosing the coronary arteries and thereby preventing the heart muscle from receiving adequate blood supply when the heart's workload rises [33].

- Prinzmetal's variant angina: Pain at rest characterizes this type of angina, which is unrelated to physical exertion. The specific cause of prinzmetal's angina is still a mystery to scientists. Atherosclerosis-induced sudden coronary vasospasm or mast cell-stimulated release of humoral vasoconstrictors in the coronary adventitia may be to blame for this phenomenon [33].

- Unstable or crescendo angina: It is the most severe form of angina. It is marked by a greater recurrence of pain onsets that last longer and occur more frequently during rest [33]. Therefore, it may be a warning sign of an oncoming myocardial attack and should be handled seriously.

c. Myocardial infarction (MI): It refers to the occurrence of a heart attack. MI takes place when the blood ceases to flow adequately to a segment of the heart, causing injury to the heart muscle due to a deficit of oxygen delivery, when one of the coronary vessels that supply the heart with blood becomes blocked due to an unstable deposition of cholesterol, white blood cells, plaques, and fat [34]. When the situation becomes more critical, it is referred to as acute myocardial infarction (AMI).

d. Coronary Sclerosis or Atherosclerosis: Accumulation of plaque in the inner lining of artery results in a condition known as arterial hardening or thickening. When fatty materials, calcium, and fibrous elements build up in the intima of an artery, it is known as atherosclerosis or coronary sclerosis.

8.3.2 Arrhythmia

Cardiac arrhythmias are a form of irregular heartbeats that are either too slow (i.e. bradycardia) or too fast (i.e. tachycardia). Arrhythmia attacks can be triggered by even a slight shift in the morphology or dynamics of the electrocardiogram (ECG), resulting in shortness of breath, chest pain, exhaustion, and even unconsciousness due to reduced heart pumping capacity [35].

8.3.2.1 Mechanisms of cardiac arrhythmias

Cardiac arrhythmias are caused by three basic mechanisms:

- Abnormal Automaticity
- Triggered Electrical Activity
- Reentry

Brief descriptions of these mechanisms are given below:

- Abnormal Automaticity: Premature heartbeats are caused by abnormal automaticity, which happens when non-pacemaker cells initiate spontaneous firing. Ventricular tachycardia, atrial tachycardia, accelerated idioventricular rhythm, and premature beats are examples of arrhythmias caused by abnormal autorhythmicity.

- Triggered Electrical Activity: Although activated once, cardiac cells contract twice in response to triggered activity. This is frequently brought on by events known as early after-depolarizations (EADs) or delayed after-depolarizations (DADs), which are attributable to electrical instability in the cell membrane of the heart. Torsade de Pointes is a typical example of this phenomenon.

- Reentry: Following normal activation of the heart, when a propagating impulse does not die out, reentry occurs, leading to re-excitation of the heart and causing it to beat faster after the refractory period has expired. Many forms of arrhythmias are brought about by this mechanism. Wolff-Parkinson-White syndrome, atrial flutter, atrioventricular nodal reentry, and bundle branch reentry are some examples of reentry-based cardiac arrhythmias.

8.3.2.2 Types of cardiac arrhythmias

Arrhythmias come in many varieties, which are associated to origination of impulses in the S.A. node and their consequent distribution to every portion of the heart. Some of the most notable forms of arrhythmias are mentioned in **Table 4** along with their morphology and characteristic features.

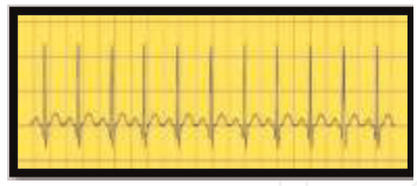
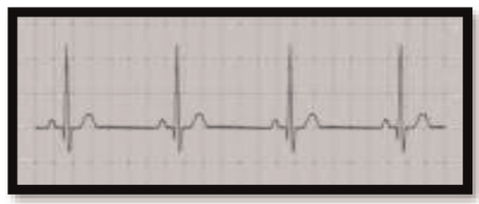
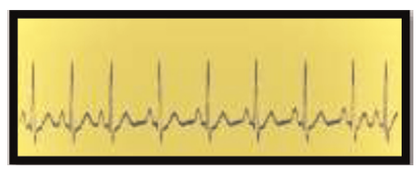
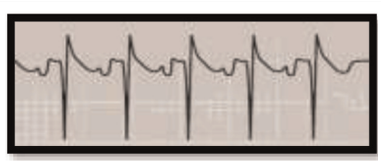
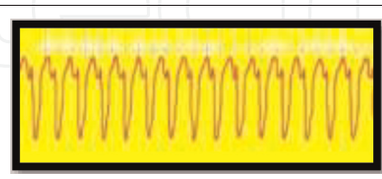
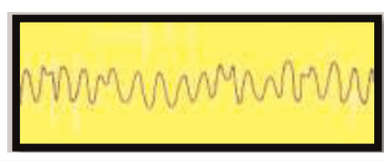
Besides these two major cardiovascular problems, there are a number of other critical heart ailments. These include:

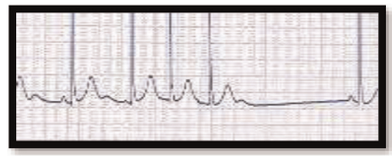
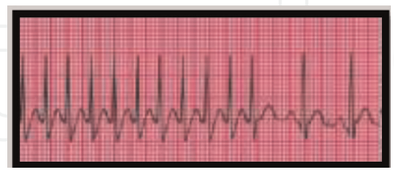
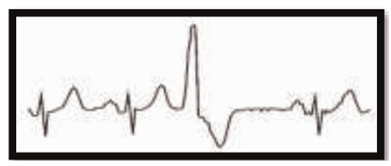
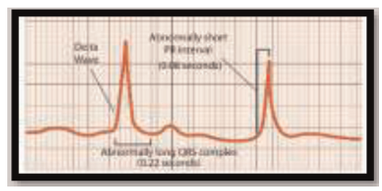
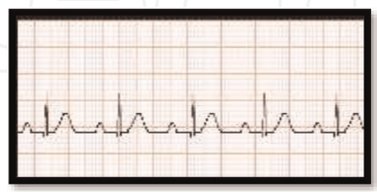
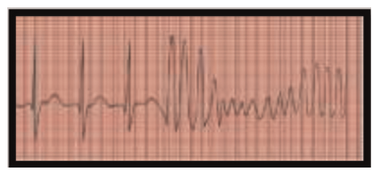
8.3.3 Hypertension

Hypertension or high blood pressure occurs when the blood exerts an excessive force against the walls of the blood vessels. In other terms, a persistently elevated blood pressure of more than 140 mmHg over 90 mmHg (i.e. a systolic pressure greater than 140 or diastolic pressure greater than 90) is considered to be hypertension. The most severe form of hypertension is chronic hypertension, which is an asymptomatic "silent" illness. It can lead to alterations in the retinal blood vessels, brain damage, kidney failure, and atypical thickening of the cardiac muscle.

8.3.4 Cor pulmonale

Cor pulmonale is the medical term for cardiac disease affecting the right side of the heart as a result of respiratory issues [33]. More specifically, cor pulmonale is defined as an anomaly in the structure and performance of the right ventricle of the heart owing to an ailment in the primary respiratory system that results in pulmonary hypertension. Hypertrophy, right ventricular dilatation or both are hallmarks of this condition [33].

Types of arrhythmia	Morphology	Characteristic features	References
Sinus Tachycardia		• Increased impulse release from the SA node causes an abnormal elevation in heart rate which may rises up to 150 bpm; • ECG exhibits short R-R interval due to elevated heart rate	[24, 36]
Sinus Bradycardia		• Decrease in heart rate which is less than 60 bpm; • Extended R-R intervals on ECG.	[24, 37]
Sinus Disrhythmia		• Periodic rise (during inspiration) and fall (during expiration) in heart rate associated with respiration; • Shortened R-R intervals during inspiration and prolonged R-R intervals during expiration.	[19, 36, 37]
Atrial Tachycardia		• Atria beat at a rate of 300 bpm; • Characteristic sawtooth pattern observed in the intervals between the QRS complexes on ECG.	[38]
Ventricular Tachycardia		• Three or more consecutive abnormal heartbeats in a row beating faster than 100 bpm.	[38]
Atrial Fibrillation		• Irregular and rapid contractions of atria at a rate of 300–400 bpm; • ECG shows no P wave.	[24, 39]
Ventricular Fibrillation		• Irregular and rapid ventricular twitching at a rate of 400–500 bpm.	[24, 40]

Types of arrhythmia	Morphology	Characteristic features	References
Premature Supraventricular Contractions		• Premature actuation of the atria originating from a site except for the S. A. node.	[41]
Paroxysmal supraventricular tachycardia (PSVT)		• Events of rapid heart rate (150–250 bpm) originating in a portion of the heart above the ventricles; • ECG shows a narrowed QRS complex with regular rhythm.	[42]
Premature Ventricular Contractions (PVCs)		• The heartbeat is generated by the purkinje fibers instead of the S.A. node; • With each PVC, there is an additional pause in the heart's regular rhythm; • PVCs may appear singly or repeatedly in a pattern.	[43]
Wolff-Parkinson-White Syndrome		• Marked by frequent attacks of AV nodal paroxysmal tachycardia in those with bundle of Kent; • ECG exhibits shortened P-R interval with normal T wave and QRS complex.	[24, 44]
Heart Block		• Partial or complete blockade of electrical signals controlling heartbeat, leading to an obstruction in transmission of impulses from atria to ventricles	[45]
Torsade de Pointes		• Each subsequent QRS complex has a different shape than the preceding one; • Twisted QRS complex is observed around the baseline on ECG surface;	[46]

Types of arrhythmia	Morphology	Characteristic features	References
		• Multiform and queer shaped QRS complexes with unidirectional sharp points for a short duration; • Extremely rare arrhythmia that may be caused by prolonged QT complexes.	

Table 4.
Different forms of arrhythmias with their morphology and characteristic features.

8.3.5 Heart failure

Heart failure is characterized as a pathophysiologic condition in which the defective heart function is incapable of maintaining optimal circulation for the metabolic demands of the body's tissues [33]. Heart failure may be chronic or acute.

8.3.6 Valvular heart disease

It develops when the valves of the heart become impaired or defective. Valvular heart diseases are of two types:

- Stenosis and
- Regurgitation.
- Stenosis: Stenosis refers to the inability of the heart valve to completely open during diastole, which obstructs the blood flowing in a forward direction [33]. Aortic and mitral stenosis are the most prevalent types of valvular stenosis.
- Regurgitation: Regurgitation of blood occurs when a valve fails to close perfectly during systole, causing regurgitation or backflow of blood [33]. Aortic and mitral regurgitation are the two most common types of valvular regurgitation.

8.3.7 Inflammatory heart disease

Inflammatory heart disease (IHD) refers to a set of conditions that include myocarditis, pericarditis, and endocarditis [47].

- Myocarditis: It is an inflammation or infection that develops inside the heart muscle prompted by viruses like specific immunological conditions and sarcoidosis [48, 49].
- Pericarditis: It refers to the inflammation or infection of the pericardium [48].

- Endocarditis: This condition is brought on by an infection of the inner lining of the heart (i.e. endocardium), which causes severe inflammation [48].

8.3.8 Rheumatic heart disease

It is a nonsuppurative, post-streptococcal, systemic, inflammatory disorder that mostly affects the central nervous system, heart, skin, joints, and subcutaneous tissues. The chronic episode of RF affects all layers of the heart (pancarditis), resulting in significant cardiac consequences known as rheumatic heart disease (RHD).

8.3.9 Stroke

According to the definition of World Health Organization (WHO), stroke can be defined as- "rapidly developing clinical signs of focal (or global) disturbance of cerebral function, with symptoms lasting 24 h or longer or leading to death, with no apparent cause other than of vascular origin [50].

8.3.10 Peripheral arterial disease (PAD)

It is a chronic atherosclerotic condition that leads to the narrowing of the peripheral artery vasculature, primarily in the lower extremities [50]. This usually restricts blood supply to the extremities, resulting in calf or thigh pain while exertion or walking. It has an estimated global incidence of up to 10%, rising to approximately 30% in patients older than 50 years [51].

8.3.11 Cardiomyopathy

It is a pathological and anatomic condition related to electrical or muscular malfunction of the heart [49]. In other terms, cardiomyopathies constitute a varied category of disorders that frequently result in progressive heart failure and substantial morbidity and mortality. Cardiomyopathies can be either primary (i.e., inherited, acquired, or mixed) or secondary (e.g., infammatory, toxic, infltrative) [52]. Hypertrophic cardiomyopathy, dilated cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, and restrictive cardiomyopathy are the most prevalent forms of cardiomyopathy.

8.3.12 Congenital heart disease

The malformation of the heart that is apparent at birth is referred to as congenital heart disease [30]. The condition affects roughly 0.5% of all newborns and is the most common kind of congenital cardiac disease. Premature newborns have a higher risk of congenital heart disease. Some notable forms of congenital heart diseases include—congenital long QT syndrome (LQTS), congenital short QT syndrome (SQTS) etc.

- Congenital LQTS: An inherited heart condition known as congenital long QT syndrome (LQTS) is attributable to a prolonged QT interval at rest and a significant risk of life-threatening arrhythmias [53]. Nearly one in every 2500 live newborns is estimated to be affected by the disease.

- Congenital SQTS: This condition is characterized by an unusually short QT interval, potentially fatal ventricular arrhythmias, and paroxysmal atrial fibrillation. Children and young adults can be affected by this autosomal dominant condition; often a family history of cardiac abrupt death is revealed [54].

- Andersen-Tawil Syndrome: It is a congenital heart disorder in which irregular heartbeats (arrhythmia), muscle weakness (periodic paralysis), and developmental anomalies are common occurrences. The paralysis episodes generally begin in early infancy and last from a few hours to several days.

9. Management of cardiovascular diseases

CVDs can be managed by following two approaches

- Non-pharmacological approach

- Pharmacological approach

Brief descriptions of both of these approaches are presented below:

9.1 Non-pharmacological management of cardiovascular diseases

The recommendations for the management of heart failure developed by the European Society of Cardiology (ESC) contain a number of tips and advice that should be included in patient education. These tips and advice are intended for patients who have chronic heart diseases [55]. In patients suffering from heart disease, the following are some of the most important aspects of non-pharmacological therapy and treatment plans -.

9.1.1 Diet modification

Diet modification is of utmost significance for persons suffering from chronic heart diseases to maintain their disease conditions [55, 56]. These may include:

i. Control of Salt Intake: Chronic cardiovascular patients should intake less than 2000 mg salts per day [57]. It is important for patients with advanced heart failure to keep their daily salt intake under 2000 mg, and these patients should also be encouraged to limit their fluid intake between 1500 and 2000 ml [58]. It should also be pointed out that salt replacements need to be used with extreme care since potassium might be included in them. They have the potential to cause hyperkalemia when consumed in significant doses in conjunction with medications that inhibit angiotensin-converting enzymes (ACEs) [59].

ii. Inhibition of Alcohol Consumption: In patients with a diagnosis alcoholic cardiomyopathy, drinking alcohol is strictly restricted; nevertheless, in other situations, drinking alcohol in moderation is acceptable [60].

iii. Smoking Cessation: For smoker cardiac patients, quitting smoking is the most successful course of therapy. It greatly curtails the fatality rates of cardiac patients compared to any other form of intervention or treatment [61–63]. Cardiac patients who cease the habit of smoking minimize their possibilities for prospective diseases and fatality by one-third two years later [62, 64].

iv. Intake of Calorie Restricted Diets: Calorie-restricted diet consumption may lead to modest loss of weight and reduction in blood pressure in overweight hypertensive individuals.

9.1.2 Rest and exercise

Patients diagnosed with heart failure were traditionally counseled for engaging in physical activity in the hopes of preventing their condition from deteriorating. Numerous investigations have reported that physical rest has come to be recommended only in cases of acute heart failure or instability in chronic heart failure [58]. Physical exercise is critical for reducing obesity, overweight and is also useful for chronic cardiovascular suffering. Even if no weight is lost, exercise can help reduce the risk factors for CVDs and assist weight loss efforts for those who are overweight and have type 2 diabetes.

9.1.3 Ventilatory support: oxygen and non-invasive ventilation

Oxygen has been utilized extensively outside of hospitals as well as in emergency rooms due to the widespread belief that it may alleviate breathlessness and increase myocardial oxygenation, regardless of the fact that oxygen saturation levels should be maintained and available for heart patients. On the other hand, supplementary oxygen and supported breathing such as ventilation should be stored or reserved for cardiac patients who are experiencing hypoxemia. Several research findings have comprehensively evaluated the effects of elevating fraction of inspired oxygen (FiO2), oxygen deficiency produces a decline in cardiac output as well as enhances systemic vascular resistance (SVR), and ventricular filling pressures [65].

9.2 Pharmacological management of cardiovascular diseases

Pharmacological management involves two approaches –

i. Treatment with Allopathic Medicines

ii. Treatment with Herbal Medicines

9.2.1 Treatment with allopathic medicines

Different types of medications are recommended for cardiac patients depending on their disease conditions. In **Table 5**, major classes of cardiovascular drugs are presented along with their mechanisms of action.

Different classes of cardiovascular drugs	Mechanism of actions	Examples of drugs	References
Beta-adrenoceptor Antagonists	Block beta-adrenoceptors in heart and inhibit the actions of epinephrine and norepinephrine, leading to a deceleration in heart beat and a reduction in blood pressure (**Figure 5**).	• Propranolol (non-selective) • Timolol (non-selective) • Atenolol (selective) • Metoprolol (selective)	[67]
Angiotensin Converting Enzyme (ACE) Inhibitors	Inhibit the conversion of angiotensin I to angiotensin II (**Figure 5**).	• Captopril • Enalapril	[68]
Angiotensin II Receptor Antagonists	Selectively block the action of angiotensin II by competitively antagonizing the angiotensin II receptors, specially AT1 receptors and aid in dilating the arteries and veins to reduce elevated blood pressure (**Figure 5).**	• Losartan • Valsartan • Candesartan	[69, 70]
Alpha-Adrenoceptor Blocking Drugs	Block alpha receptor in heart.	• Prazosin • Doxazosin	[71]
Adrenergic Receptor Blocking Drugs	Block adrenergic neurons and prevent the release of noradrenaline from postganglionic adrenergic neurons.	Guanethidine	[72]
Vasodilator Antihypertensive Drugs	Dilate the constriction or narrowing of blood vessels.	• Diazoxide • Minoxidil • Hydralazine	[73]
Centrally Acting Antihypertensive Drugs	Regulate and control impulses along certain nerve pathways.	• Clonidine • Methyldopa	[74]
Ganglionic Blocking Drugs	Inhibit transmission of impulses at both sympathetic and parasympathetic ganglia	• Pempidine • Trimetaphan • Mecamylamine • Hexamethonium	[75]
Nitrates	Coronary vasodilation (**Figure 6**)	• Nitroglycerine • Isosorbide Dinitrate	[76]
Calcium Channel Blockers	Blocks inward movement of calcium ions (**Figure 5, Figure 6**).	• Verapamil • Diltiazem • Nifedipine • Nicardipine	[77, 78]
Potassium Channel Activators	Dilates veins and arteries.	• Nicorandil	[79]
Cerebral and Peripheral Vasodilators	Dilates blood vessels	• Cilostazole • Niftidrofuryln	[80, 81]
Potassium Channel Inhibitor	Block efflux of potassium ions through the cell membranes, resulting in a prolongation in action potentials.	• Satolol • Amiodarone • Amifampridine	[82]
Potassium-sparing Diuretics	Act either by disrupting the exchange of sodium-potassium in the distal convoluted tubule or as an aldosterone receptor antagonist (**Figure 7**).	• Amiloride • Eplerenone • Triamterene • Spironolactone	[83, 84]

Different classes of cardiovascular drugs	Mechanism of actions	Examples of drugs	References
Thiazide Diuretics	Reduce sodium and fluid reabsorption (**Figure 7**).	• Metolazone • Chlorthalidone	[85]
Loop Diuretics	Inhibit the luminal Na-K-Cl cotransporter in the thick ascending limb of the loop of Henle (**Figure 7**).	• Furosemide • Bumetanide	[86]
Osmotic Diuretics	Elevate the osmolality of blood plasma (**Figure 7**).	• Mannitol • Isosorbide	[87]
Inotropic Sympathomimetic Drugs	Directly stimulate beta-1 receptors of the heart to increase myocardial contractility and stroke volume, resulting in increased cardiac output.	• Dobutamine • Dopamine • Isoprenaline	[88]
Vasoconstrictors	Indirectly stimulate the adrenergic receptor system by enhancing the activity of norepinephrine.	• Ephedrine • Methoxamine	[89]
Drugs for Cardiopulmonary Resuscitation	Relax the muscles in the airways and tightens the blood vessels.	• Epinephrine • Norepinephrine	[90]
Parenteral Anticoagulants	Produce antithrombotic effect by binding to antithrombin III.	• Heparin • Fondaparinux	[91]
Oral Anticoagulants	Block one of the enzymes (proteins) that uses vitamin K to produce clotting factors.	• Dabigatran • Rivaroxaban	[92]
Anti-heparin Agent	Form a complex by binding with high affinity heparin and rapidly reverse the anticoagulant effects.	Protamine Sulphate	[93]
Antiplatelet Drugs	Irreversibly blocks prostaglandin H synthase in platelets and prevent platelet aggregation by inhibiting the synthesis of thromboxane A2.	• Clopidogrel • Aspirin	[94]
Fibrinolytic Drugs	Act with plasminogen to produce an "activator complex" that converts plasminogen to the proteolytic enzyme plasmin.	• Anistreplase • Urokinase	[95]
Antifibrinolytic Drugs and Hemostatic	Act by inhibiting the breakdown of blood clots, which prevents bleeding.	Aminocaproic acid	[96]
Anion Exchange Resins	Reduce high cholesterol levels in the blood.	• Cholestipol • Cholestyramine	[97]
Fibrates	Decrease the levels of triglycerides and increase HDL cholesterol levels.	• Clofibrate • Fenofibrate	[98]
Statins	Block 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase (**Figure 8**).	• Atorovastatin • Fluvastatin • Simvastatin	[100]
Nicotinic Acid Derivatives	Reduce plasma viscosity and platelet aggregation.	Inositol nicotinate	[101]

Table 5.
Major classes of cardiovascular drugs and their mechanisms of actions with examples of each class.

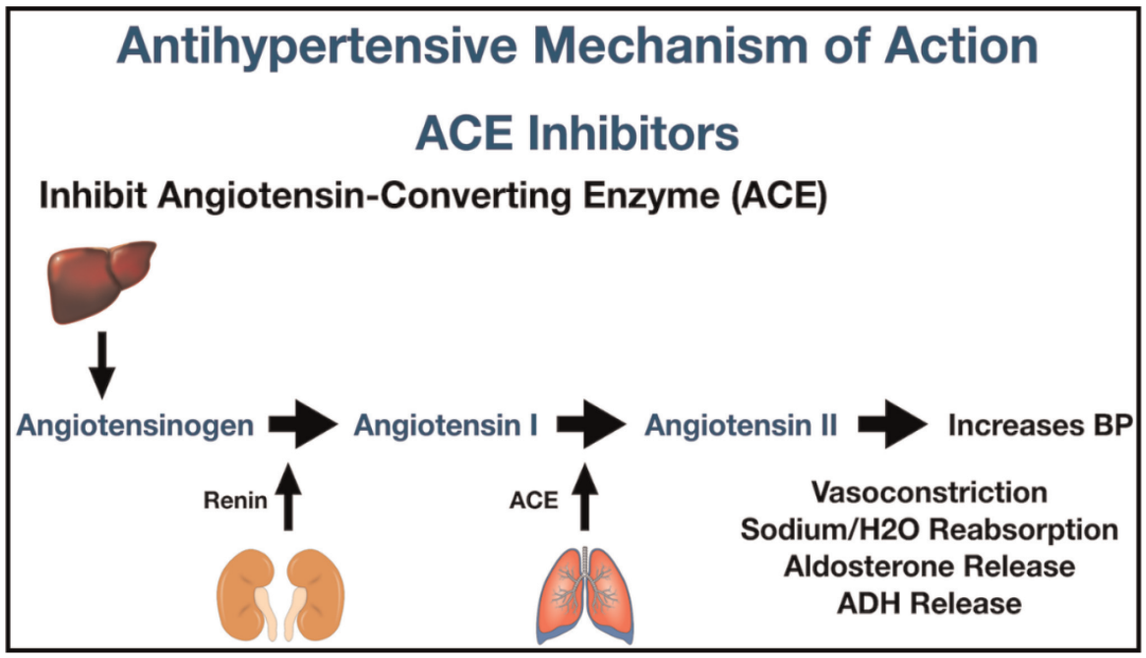


Figure 5.
General mechanism of antihypertensive drugs (significantly modified as per study design) [66].

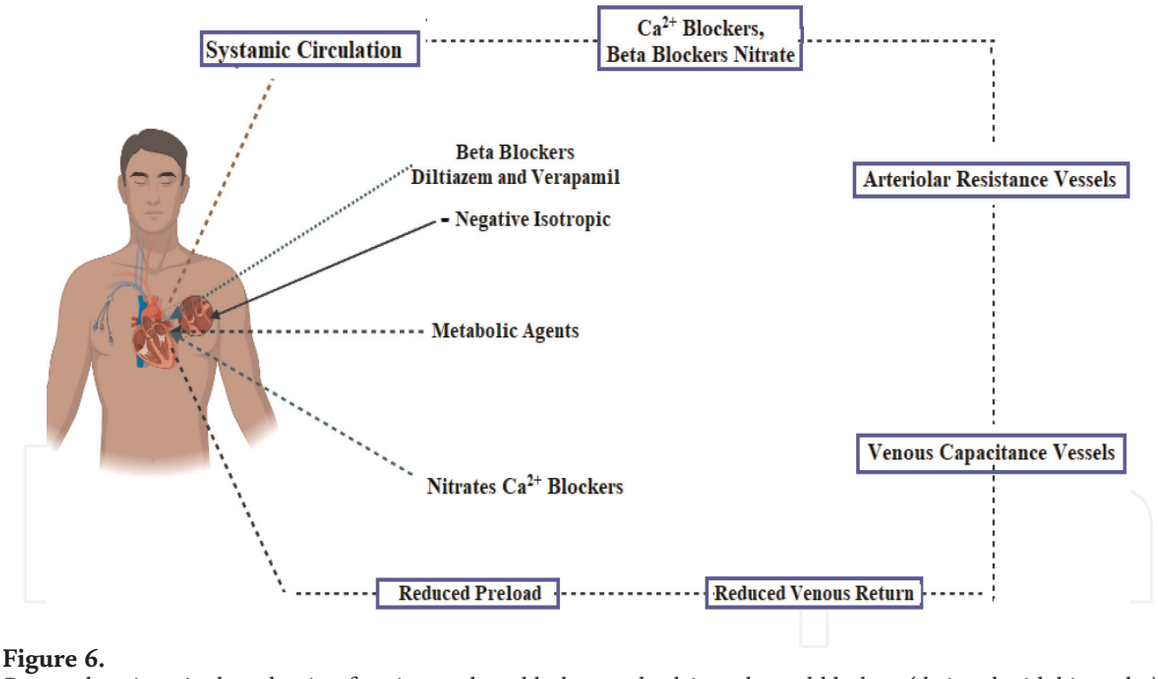


Figure 6.
Proposed anti-anginal mechanism for nitrates, beta-blockers and calcium channel blockers (designed with biorender).

9.2.2 Treatment with herbal medicines

Various herbal drugs are also utilized for the management of cardiovascular diseases (**Table 6**).

10. Conclusion

This review shines a spotlight on the physiology, pathology, and management of the cardiovascular system. The cardiovascular system simultaneously eliminates

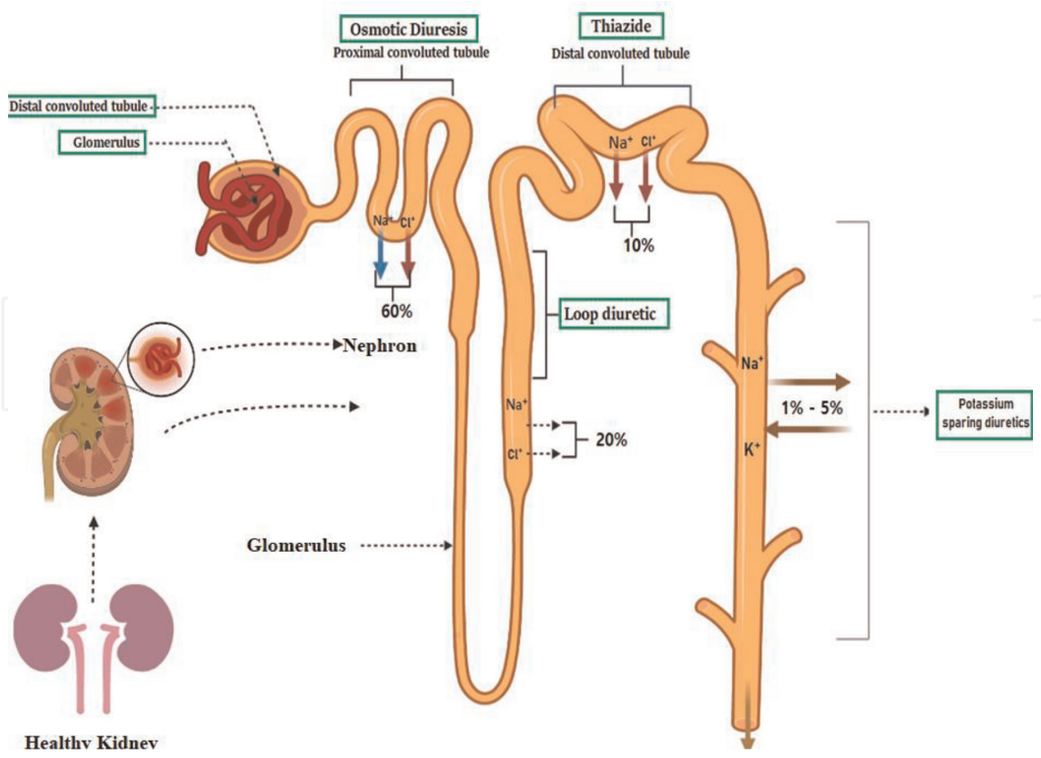


Figure 7.
General mechanism of action of diuretics (designed with biorender).

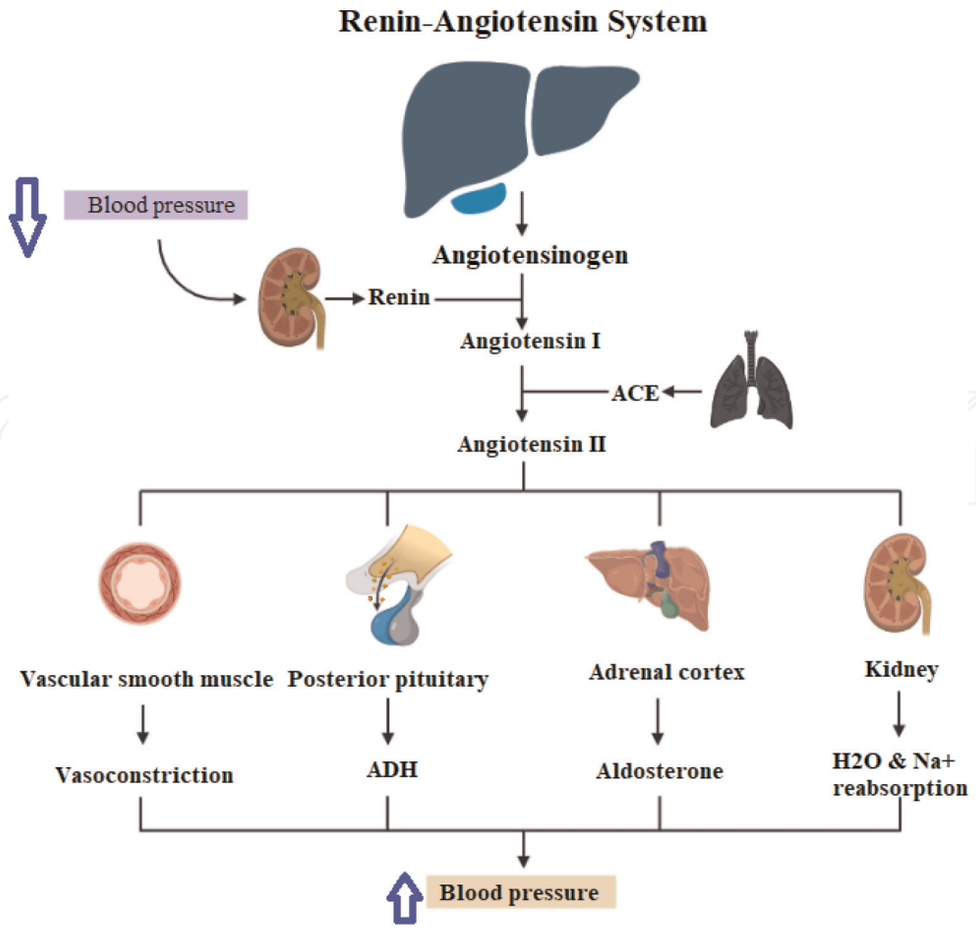


Figure 8.
Mechanism of action of statins [99].

Herbal drugs	Treated diseases	References
Anthocyanidin	Coronary heart disease	[100]
	Ischemia–reperfusion injury	
Citrus fruits	Ischemia–reperfusion	[101]
Peanuts, red wine,	Hypertension	[102]
	Ischemia–reperfusion	
Rhizoma coptidis	Hypertension	[103]
Rhizome of a turmeric plant	Hypertension	[104]
Crocus sativus L.	Heart failure	[105]
Angelica sinensis	Heart failure	[106]
Sophora flavescens	Arrhythmia	[107]
Dendrobium nobile	Acute myocardial infarction	[108]
	Ischemia–reperfusion	
Glycine max	Acute myocardial infarction	[109]
Allium in *Liliaceae*	Hypertension	[110]
Tea	Hypertension	[110]
Carthamus tinctorius L.	Acute myocardial infarction	[111]
Panax ginseng	Coronary heart disease	[112]
	Ischemia–reperfusion injury	

Table 6.
Lists of herbal drugs and treated cardiovascular disorders.

waste products from the tissues and delivers fresh oxygen and nutrients to the tissues and cells of the body. The heart and blood vessels are the fundamental components of this cardiovascular system, with the heart serving as the core pumping unit. The heart is composed of two atria and two ventricles. It has been considered that the right side of the heart receives blood that is depleted in oxygen but rich in carbon dioxide. This blood is then pushed into the pulmonary veins and finally travels back to the left side of the heart, where the blood is oxygenated by the lungs, which removes carbon dioxide from the blood. The left ventricle is responsible for ejecting blood from the heart and distributing it to the rest of the body. During each phase of the cardiac cycle, the atria compress while the ventricles remain relaxed, and then the process is reversed. However, any dysfunction of this precious organ can have disastrous consequences, leading to serious cardiovascular ailments and even death. These cardiovascular ailments are the greatest cause of morbidity and mortality in both developing and developed countries, with CADs and arrhythmias being the most prominent. These CVDs can be triggered by a variety of risk factors that can be either modifiable or nonmodifiable, including age, gender, ethnic background, smoking, physical inactivity, high cholesterol, and blood pressure etc. The treatment of these cardiovascular ailments requires the administration of certain allopathic drugs and herbal medications based on disease conditions and progression, as well as adherence to specified non-pharmacological interventions, which will significantly help in reducing the morbidity associated with severe cardiovascular events.

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Conflict of interest

The authors declare no conflict of interest.

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