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# UNIT 4    **CARDIOVASCULAR SYSTEM**

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## **4.1    INTRODUCTION**

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In the first three units we have studied about the cell – the basic unit of life, blood – the elixir of life and about the immune system. In this unit, we shall unfold the mysteries of our heart and its connecting blood vessels. We shall learn about the structure and functioning of the heart and more about the commonly used terms – blood pressure and heart attack.

### **Objectives**

After studying this unit, you will be able to:

- explain and illustrate the structure of the heart,
- describe the various functions of the heart,
- discuss the common terminologies of blood pressure and heart attack, and
- explain the role of exercise and diet in keeping the heart toned and body fit.

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## **4.2    DESIGN OF CARDIOVASCULAR SYSTEM**

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The cardiovascular system consists of a pump - the heart - and a network of pipelines--the blood vessels. Let us begin our study of the cardiovascular system, with the pump.

### **4.2.1    Heart: the Pump**

We all are familiar with the heart. The heart and its major components are shown in the Figure 4.1. As you can see, the heart has four chambers. Can you list the names of the four chambers of the heart? Yes, the four chambers are the *right atrium*, *right ventricle*,

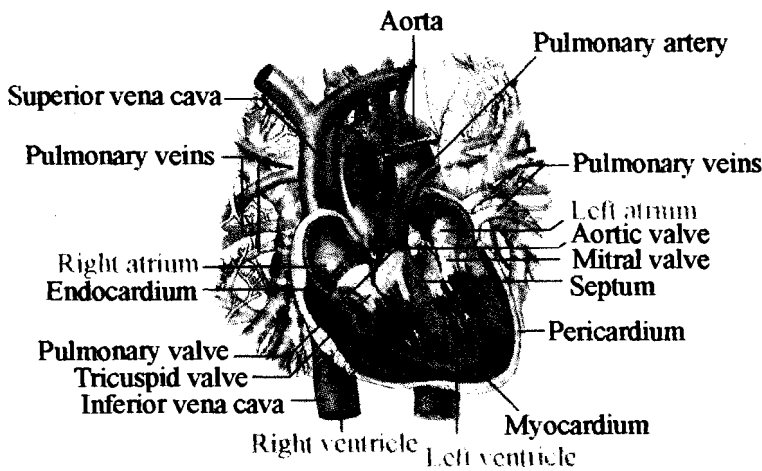


Figure 4.1: Heart and its components

Now how does the blood circulate in our body? This is explained diagrammatically in Figure 4.2, showing the major arteries and veins and the organs to which they supply blood. Let us now focus on the route, which the blood takes within the chambers of the heart. The right atrium receives blood from the body through two large veins – *the superior and the inferior vena cava* (as can be seen on the left hand side of the heart in Figure 4.1). It empties blood into another chamber of the heart, the right ventricle as shown in Figure 4.2. The right ventricle pumps the deoxygenated blood to the lungs, through the pulmonary artery.

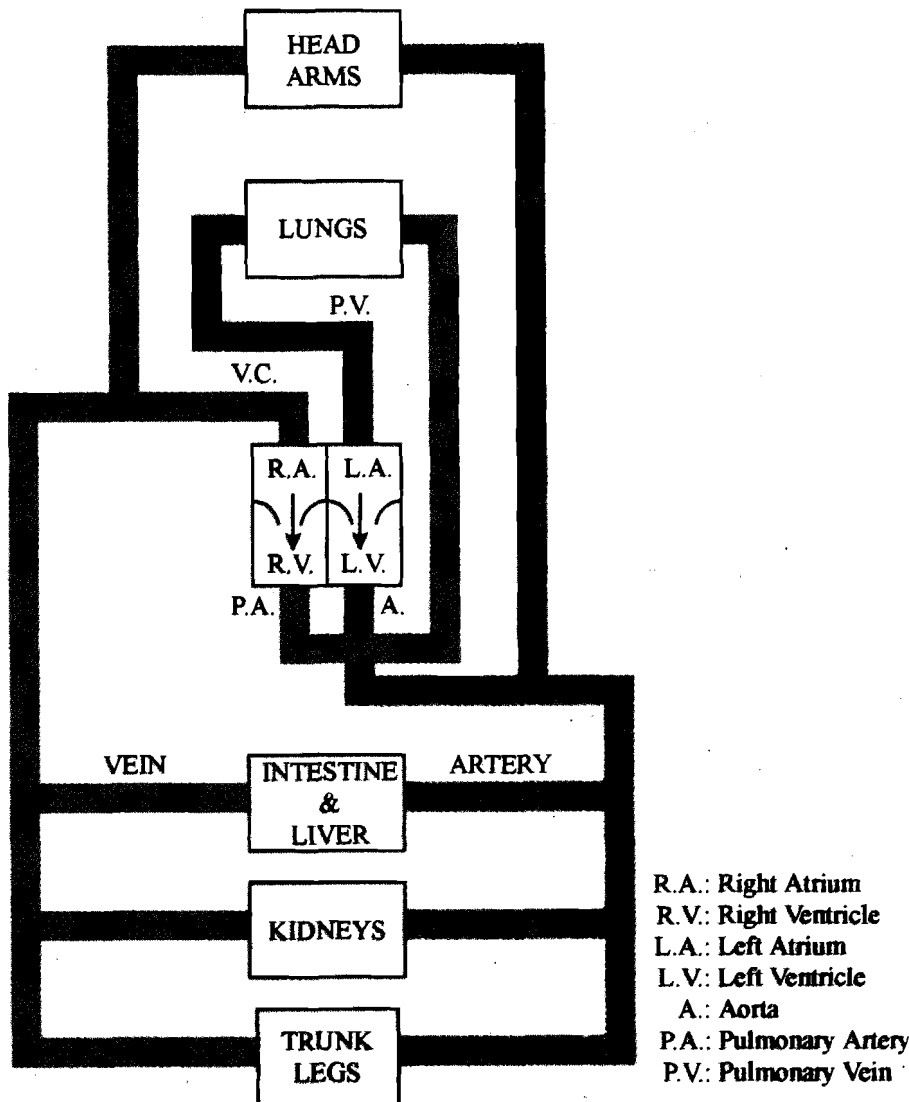


Figure 4.2: Circulation of blood in our body

Figure 4.3 (a) shows the inside of the right ventricle, which pumps blood into the pulmonary trunk and divides into right and left pulmonary arteries. A semi-lunar valve called the *pulmonary valve*, which prevents the flow of blood back into the right ventricle, guards the opening of the right ventricle into the pulmonary trunk.

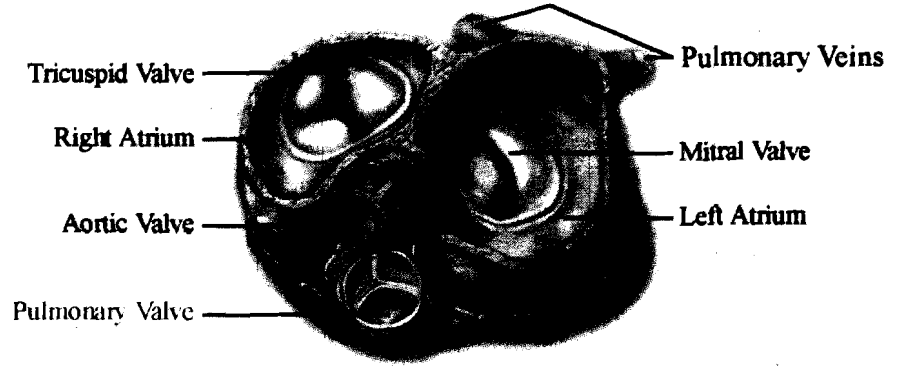


Figure 4.3(a): Right ventricle

Blood from the pulmonary trunk enters the pulmonary circulation and receives oxygen from the lungs as highlighted in Figure 4.2. This oxygenated blood flows through pulmonary veins to the left atrium as can be seen in Figure 4.3(b). From the left atrium, the oxygenated blood enters the left ventricle. The left ventricle is the main pumping chamber sending the blood through the aorta to all of the body parts except lungs.

There is a valve at the junction of the right atrium and right ventricle. The tricuspid valve, as shown in Figure 4.3(a), ensures one-way flow i.e., it prevents the blood from flowing back from the right ventricle to the right atrium. Similarly, there is a valve called *bicuspid, mitral or left atrio-ventricular valve* at the junction of the left atrium and left ventricle. Figure 4.3(a) highlights the valve. The mnemonic to remember the names of the valve is *Banwari Lal Tota Ram* i.e. **B**icuspid **L**eft heart **T**ricuspid **R**ight.

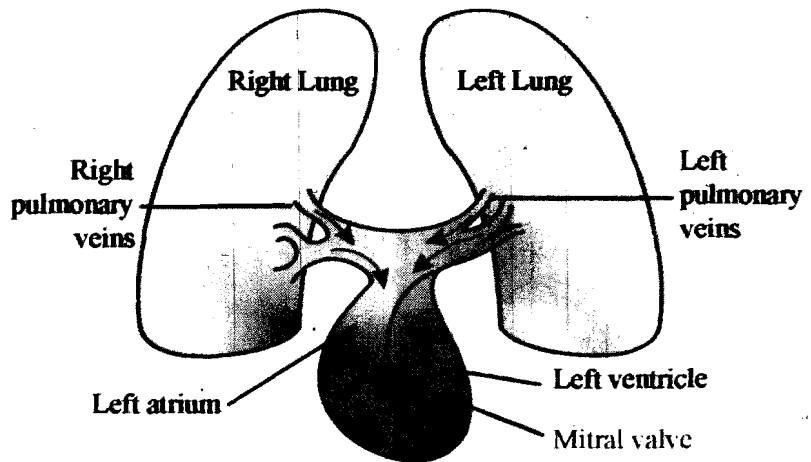


Figure 4.3 (b): Left side

Figure 4.3(c) summarizes the blood circulation within the chambers of the heart. The left ventricle pumps blood into the aorta as can be seen in Figure 4.3 (c). Here you would realize that the aorta supplies blood rich in oxygen and nutrients to the entire body system. The branches of the aorta are the arteries and are called *systemic arteries*. Since the systemic arterial network is much more extensive than the pulmonary arterial network, the left ventricle has to pump blood at a much higher pressure than the right ventricle – this is made possible by a much thicker wall (musculature) than that of the right ventricle.

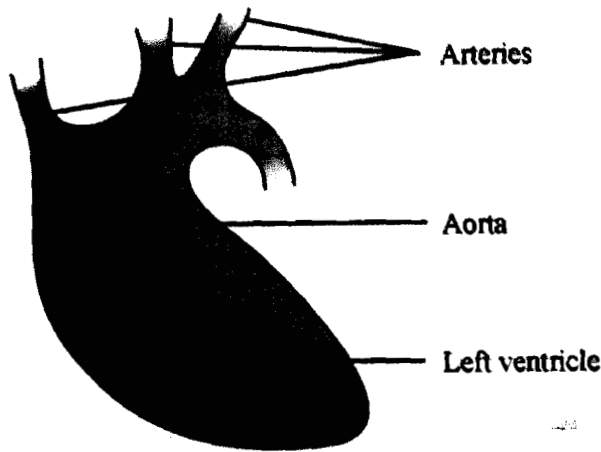


Figure 4.3 (c): Blood distribution within the chambers of the heart

In this section we studied the heart i.e. the pump. Next, let us learn about the blood vessels.

### 4.2.2 Blood Vessels: the Pipelines

Blood vessels are the pipelines of the heart. Can you suggest which are the blood vessels of the heart? Yes, *aorta*, *arteries*, *arterioles*, *capillaries* and *veins* are the blood vessels of the cardiovascular system. The branches of the cardiovascular system may be likened to a tree, which divides itself into smaller thinner divisions. Aorta divides to arteries, arteries into arterioles and arterioles into capillaries and capillaries join to form veins. Let us now study about the functions of each of these.

Starting with the *aorta*, the aorta we learnt above supplies oxygenated blood to the entire body. It is distensible. The elastic tissue present in the aortic wall absorbs the shocks of blood striking against it from the heart. This along with decrease in diameter of blood vessels away from aorta changes the spurty pulsatile blood flow into a continuous flow. This is termed as the *windkessel effect*, after the scientist who showed this effect.

*Arterioles* function as 'resistance vessels'. When their diameter is reduced, the blood flows with a much greater resistance and vice-versa. This is the manner in which the blood flow is redistributed during aerobic exercise to other parts of the body. Arterioles ensure healthy blood supply to the vital organs of the body – the brain and the heart during exercise, decreasing blood supply to the intestines. That is why there is wisdom in the saying that *one should not exercise after meals*, as food may not be digested properly because of the decreased blood supply to the intestines after exercise.

Continuous blood flow is essential for exchange of gases ( $O_2$  and  $CO_2$ ), food and water across vessels called *capillaries*. The *capillaries* are thin-walled narrow tubes without any muscular coat. The capillaries come in intimate contact with the cells while picking carbon dioxide and waste products from them. That is why they are termed as the *exchange vessels*.

*Veins* are the vessels that are extremely thin-walled. They lack the strength of the solid arterial wall but are extremely flexible. Their main function is to store blood in large capacities. They are called as *capacitance vessels*. The veins of the calf muscle in the lower leg are termed as *peripheral hearts* as they store a lot of blood that is pumped back by muscle action during running to the heart. Do you recall how the blood would flow from the legs to the heart? Yes, it is via the inferior vena cava to the right atrium.

Having understood the heart and its blood vessels, it is time for us to learn about the nerves that control the heart functioning.

### 4.2.3 Control of Our Heart Through Nerves

When we exercise in the mornings, the heart beats faster and when we sleep at night, it slows down. Have you ever wondered how does the heart tick? Our old Rishis and Saints have spent years answering this question. Some have managed to even stop their heart from beating while others have made it beat at will at a rate so fast that it would kill a normal human being. How do they manage to do that? They, over the years, have simply gained control over their autonomic nerves that supply the heart and blood vessels. What are these nerves? Let us get to know them.

The nerves are divided into two systems: *sympathetic* and *parasympathetic*. They constitute the *autonomic system* (auto: independent from the rest, a system that is different and unique). How is this system unique? It's quite simple.

*Sympathetic*, as the word suggests, means to sympathize. The body sympathizes during fight, flight and fright reaction. For example, have you ever felt your heart beat faster when in the dark or when frightened? This increase in the heart rate is termed as '*positive chronotropic effect*' (Chrono: from chronological meaning related to time or rate). The heart also beats with an increased force as you may have experienced your heart pounding against a heaving chest after a sprint. This is called as '*positive inotropic effect*', as the increase in force is brought about by the changes in ion concentration across the cell membrane. Calcium within the cell makes the heart beat stronger. (To remember, milk gives strength and milk has calcium). Have you ever wondered about the mechanism that makes the beat faster? Nature has designed a beautiful mechanism which symbioses the electrical impulse and the solid heart. A flame is ignited in the heart, which then travels to the cardiac muscle, imparting it energy to contract. The rate of transmission of this flame or electrical impulse increases during sympathetic situations and is termed as *positive dromotropic*. An increase in the excitability of the cardiac tissue is termed as *positive bathmotropic*.

Another system that runs parallel to the sympathetic system is termed as the *parasympathetic system*. This system has an effect just opposite to the sympathetic system. It relaxes the heart, decreases the heart rate (negative chronotropic), the force of contraction (negative inotropic), the conduction rate (negative dromotropic) and the excitability of tissue (negative bathmotropic).

The nerves that supply the heart's atria are termed as *vagi* (singular *vagus*). As the name suggests, *vagus* is a vagabond, a wanderer charting an unfamiliar, vague course supplying the digestive tract, heart, the voice box etc. The heart is supplied by the right and left *vagi*. The peculiarity is that they do not supply the ventricles. The sympathetic system only supplies the ventricles.

Overall, we have seen that the heart hangs in a balance of positive and negative forces: the sympathetic and parasympathetic system. Now you know the mechanism that may be operating when our yogis increase their heart rate or stop it!

The sympathetic nerves have a chemical neurotransmitter called *nor-adrenaline*, whereas, the parasympathetic nerves release *acetylcholine* at the postganglionic nerve endings.

Next, like the heart, let us see how the nerves control the blood vessels.

### 4.2.4 Control of Our Blood Vessels Through Nerves

The sympathetic system supplying the blood vessels constricts the lumen, redistributing blood flow to the regions of the body. Parasympathetic nerves usually do not supply blood vessels. If you recall, you have just read that these nerves do not supply even the ventricles. The exceptional blood vessels receiving parasympathetic innervation are

those of the salivary glands, pancreas and genital organs. Stimulation of these nerves results in increasing the luminal diameter or vasodilatation (dilation of vessels). They play a role in increasing salivary and pancreatic secretion after meals and in erection of the penis (male sex organ). The neurotransmitter of sympathetic nerves is nor-adrenaline.

There exists another specialized sympathetic nerve called *sympathetic cholinergic nerves*. Do you know how these neurons differ from sympathetic nerves? Just before the exercise, the very thought of exercise may increase the heart rate or some students may faint during the stress of an examination. These fibers produce vasodilatation of blood vessels of skeletal muscles, unlike the sympathetic nerves that cause vasoconstriction. How these fibers help the body to prepare for stressful situations is debatable. The neurotransmitter is acetylcholine, hence the name cholinergic.

**Check Your Progress Exercise 1**

1) Name the four chambers of the heart.

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2) Briefly describe the blood flow in the heart.

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3) What is the difference between aorta and arterioles?

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4) How the capillaries are designed for exchange of nutrients at tissue level?

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5) How sympathetic cholinergic neurons differ from sympathetic nerves?

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## 4.3 WHAT IS THE HEART MADE UP OF?

Earlier in this unit, we have learnt about the design of our cardiovascular system. Here in this section, we shall study what the heart – as a pump, is made up of i.e. the tissues and the fibers and their functions.

### 4.3.1 Pacemaker and Conduction Tissues

Heart has a well-developed pacemaker tissue, which can generate rhythmic impulses. As the name suggests, the tissue sets the pace at which the heart beats. Figure 4.4 illustrates the heart and its tissues namely the sinoatrial (SA) node, atrioventricular (AV) node, atrioventricular bundle (Bundle of His). Pacemaker and conduction tissues are made up of modified cardiac muscles, which are not well striated. They conduct electrical impulses to the cardiac muscle. Let us get to know them.

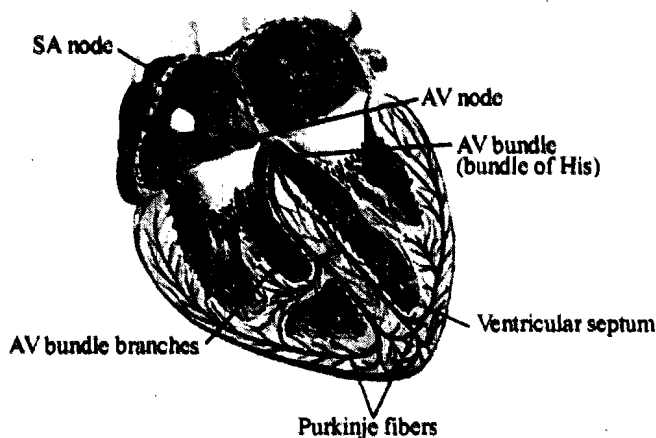


Figure 4.4: Heart and major tissues

The pacemaker of heart is the *sinoatrial node (SA node)*, a section of the nodal tissue, that is situated in the right atrium near the opening of the superior vena cava as can be seen in Figure 4.4. This is the node from where the electrical impulses originate. Its functions are to set the rate of contraction for the heart and spontaneously generate nerve impulses that travel throughout the heart wall causing both atria to contract.

The other pacemaker tissue is the *atrioventricular node (AV node)*. Notice in Figure 4.4 that this lies between the atrium and ventricle situated on right side of the *interatrial septum* (the wall between the two atria), near the opening of the *coronary sinuses* (the vessels that carry venous blood to the heart). The AV node controls the transmission of the electrical impulse from the atria to ventricles. Hence, its functions are to delay cardiac impulses from the sinoatrial node to allow the atria to contract and empty their contents first, and relay cardiac impulses to the atrioventricular bundle, as illustrated in Figure 4.4.

AV node is bulbous in appearance as can be seen in Figure 4.4 and gives rise to a bundle of conduction tissues called as *Bundle of His*. It is a bundle of heart tissues that transmit the electrical impulses from the AV node to the ventricles causing cardiac muscles in the ventricles to contract. Next, the bundle passes along the posterior and inferior margin of the pars membranacea septi to reach its junction with the superior margin of the muscular interventricular septum. At this point, the main AV bundle divides into two bundle branches to the respective ventricles.

The Bundle of His divides into a network of *purkinje fibers* (refer to Figure 4.4) as they enter the ventricles. The microscopic structure of purkinje fibers reveals that these are not fibers but are actually modified strands of cardiac muscles as shown in Figure 4.5. These help to spread the impulses throughout the heart in a specific sequence

at appropriate velocities to help make the heart contract and relax at the right time. The Purkinje fibers are very large and conduct the action potential at about six times the velocity of ordinary cardiac muscle (i.e., 1.5 to 4.0 meters per second). Thus, the Purkinje fibers permit a very rapid and simultaneous distribution of the impulse throughout the muscular walls of both ventricles. Did you also know that the cardiac muscle has a latent or sleeping pacemaker activity, which may be manifested if the normal pacemakers fail?

The cardiac impulse originates in the SA node. The impulse spreads to the atria like 'ripples in a pond'. Three specific conduction pathways have also been described which help spread through the atria faster. After spreading to the atria, the impulses reach the AV node. AV node has very slow conducting fibers. Therefore, the impulse is delayed by about 0.1 sec at the AV node, after which it spreads rapidly to the ventricles.

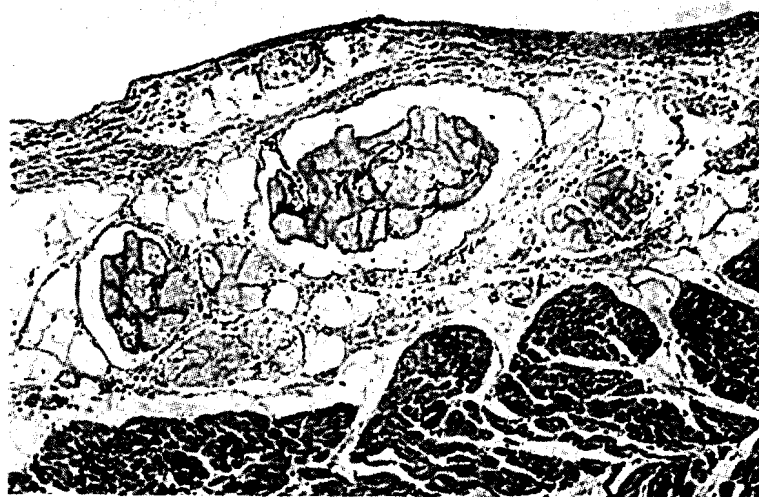


Figure 4.5: Purkinje fibers

After the cardiac tissue, it is the turn of cardiac muscle. Let's get to know it better.

### 4.3.2 The Cardiac Muscle

We have seen that our heart is an amazing structure. It pumps out blood about 70 times a minute for 70 years without a break. This herculean task is performed by unique musculature of the heart called *cardiac muscle*. What makes this muscle go on and on forever? Let's find out.

The cardiac muscle is not under voluntary control, unless you are a *Rishi*! This muscle is a branched one. Under the microscope, the muscle shows cross striations as shown in Figure 4.6.

Here, the longitudinal cardiac muscle can be identified by centrally placed round to oblong nuclei, striations, branching and intercalated discs. Each fiber is 100 microns long and about 15 microns wide. At the junction between two adjacent fibers are specialized areas called *intercalated discs*, which have a low electrical resistance. They enable faster transmission of impulse. Although the cardiac muscle fibers are separated from each other, they act as a functional syncytium or one unit because cells are electrically coupled. This means that an impulse generated anywhere in the cardiac muscle spreads throughout the musculature. The heart contracts as one system in a coordinated manner.

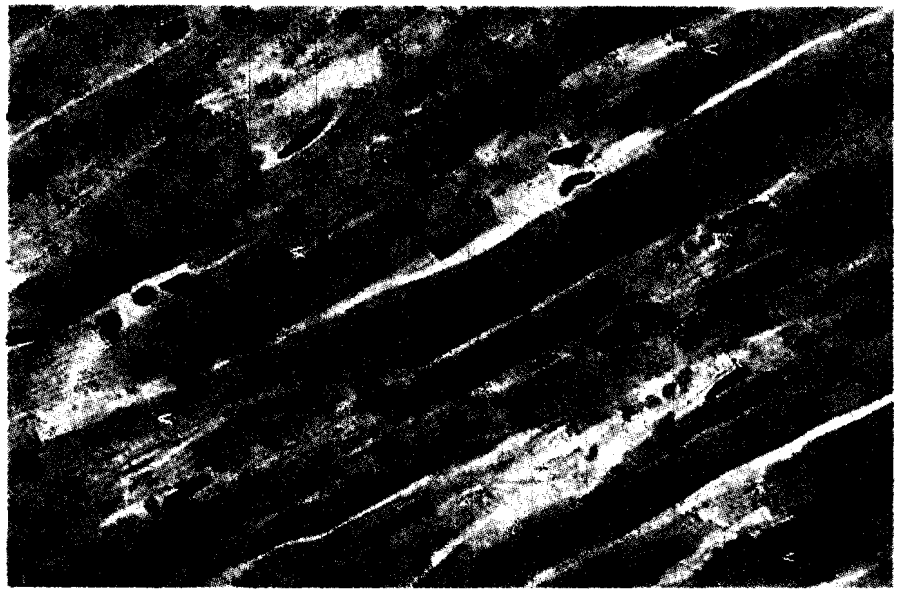


Figure 4.6: Cardiac muscle

So you can see our heart is unique. Let's see how unique our heart is, in the next section.

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## 4.4 THE UNIQUENESS OF OUR HEART

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Cardiac muscle has some unique properties that make it ideally suited for the function it performs. These properties can be easily understood in the light of the structural features as we have just read. These may be studied further as "Properties of a Beating Heart". Let us have a look at what these properties are:

### *Properties of a Beating Heart*

Let us study a few interesting properties of a beating heart which make it such a unique organ.

- *Automaticity*

Take out my heart from my body and it will still go on and on beating. How true! Even a completely denervated heart from the body continues to beat. The capability to contract even in the absence of neural control is called "*automaticity*".

- *Rhythmicity*

Not only does the denervated heart continues to beat, it does so remarkably rhythmically. What it means is that the heartbeats are extremely regular. It also means that the time interval between any two consecutive beats is exactly the same. Rhythmicity is conferred on the heart by the conduction system of the heart. The SA node discharges impulses at the fastest rate, the AV node next and the rest of the conduction system does so with the least frequency. This situation may be understood by imagining a train with more than one engine. If each of the engines is set for running at different speeds, the fastest engine will determine the speed of the train. That is why SA node is the pacemaker of the heart because it determines the pace of the heart. If the fastest engine fails, the second fastest engine will take over and will determine the speed of the train. In the same way, if the SA node fails, the AV node dictates the heartbeats at the slower rate. This orchestra goes on and on rhythmically and coordinated for life.

- *Long refractory period*

Refractory period is *the period during which a stimulus fails to evoke a response*. In a beating heart, if an external stimulus is applied during contraction, there is no response irrespective of how strong the stimulus is. This is because the cardiac muscle is refractory

throughout the contraction. The reason why cardiac muscle has a long refractory period is that the duration of its action potential is almost as long as that of its mechanical activity. The fact that the heart sleeps and does not react when exposed to day-to-day exciting and stressful situations without missing a heartbeat every now and then ensures its smooth functioning hundred long years.

● *Extrasystole and Compensatory Pause*

During the relaxation period, cardiac muscle is in the relative refractory period i.e. sleeping but only just. If a sufficiently strong electrical stimulus is applied during this period, it leads to a contractile response. Since this contraction appears earlier than the normally expected contraction, it may be considered an 'extra' contraction. Hence, it is called *extrasystole*.

The next natural impulse then arrives during the refractory period corresponding to the extrasystole. Hence, the natural impulse is ineffective and a normally expected contraction is missed. As they say, "I missed a beat". The pause in cardiac activity is called *compensatory pause*.

In real life, if we drink too much coffee, the heart fires leading to premature contraction of the ventricles and extrasystole may be produced.

Next let us look at the properties of a quiescent heart.

*Length-Tension Relationship:* The force of contraction of cardiac muscle is directly proportional to the initial length of the muscle fibers. This is known as the *Starling's Law of the Heart* after the scientist *Frank Starling*. In practice, the length of the cardiac muscle increases when the venous return increases. Hence, an increase in venous return increases the force of contraction of the ventricle. This property has several implications for cardiovascular function and would be discussed in greater detail as we read along.

**Check Your Progress Exercise 2**

1) What do you understand by the term 'pacemaker'? Give the names of the pacemakers (tissues) of our heart?

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2) Enlist the unique properties of our heart.

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3) What is an extrasystole and compensatory pause?

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So far we have learnt about the heart, its components, functions and the properties. Next, we shall get to know about how the heart function is expressed or measured.™

## 4.5 CARDIAC OUTPUT

Let us start by understanding what is meant by *cardiac output*. To put in the simple terms, cardiac output is *the output of the heart per minute*. We already know that the function of the heart is to pump blood. Hence, cardiac function is best expressed in quantitative terms as *the amount of blood pumped by each ventricle per minute*. The amount of blood pumped by either ventricle during every beat (called stroke volume) is about 70 ml at rest, and the heart beats about 70 times per minute at rest.

Hence, cardiac output is expressed as under:

$$\begin{aligned}\text{Cardiac output} &= \text{Stroke Volume} \times \text{Heart Rate} \\ &= 70 \text{ ml} \times 70/\text{min} \\ &= 4900 \text{ ml/min} \\ &= 5 \text{ L / min (approx.)}\end{aligned}$$

The cardiac output per unit body surface area is relatively constant from one individual to another and is called *cardiac index*, which is expressed as under.

$$\begin{aligned}\text{Cardiac index} &= \frac{\text{Cardiac output}}{\text{Body Surface Area}} \\ \text{Surface Area} &= \frac{5 \text{ L/min}}{1.7 \text{ m}^2} \\ &= 3 \text{ L/min/ m}^2 \text{ (approx.)}\end{aligned}$$

Body surface area (BSA) may be *calculated* from height and weight using *dubois formula*, which is as stated herewith:

$$\text{BSA (m}^2\text{)} = \text{Weight (kg)}^{0.425} \times \text{Height (cm)}^{0.725} \times 0.007184$$

Alternatively, BSA may be read of from a nomogram.

Cardiac output varies in day-to-day situations. One everyday example of such a situation is exercise, which is associated with an increase in blood flow to the working muscles of the legs, hands and heart. During exercise, the cardiac output may increase up to about five-fold even in untrained persons and up to ten-folds in the trained athletes.

Having studied about the measure of heart functioning, next we shall get to know about the sequence of events that occur every time our heart beats, which is referred to as *cardiac cycle*.

## 4.6 THE CARDIAC CYCLE

The cardiac cycle is *the sequence of events that occur when the heart beats*. You may have heard of the terms *diastole* and *systole*, particularly with reference to blood pressure. Well, these are the two phases of the cardiac cycle, as highlighted herewith:

- 1) *Diastole* - Ventricles are relaxed.
- 2) *Systole* - Ventricles contract.

A simplified representation of these phases is shown in Figure 4.7.

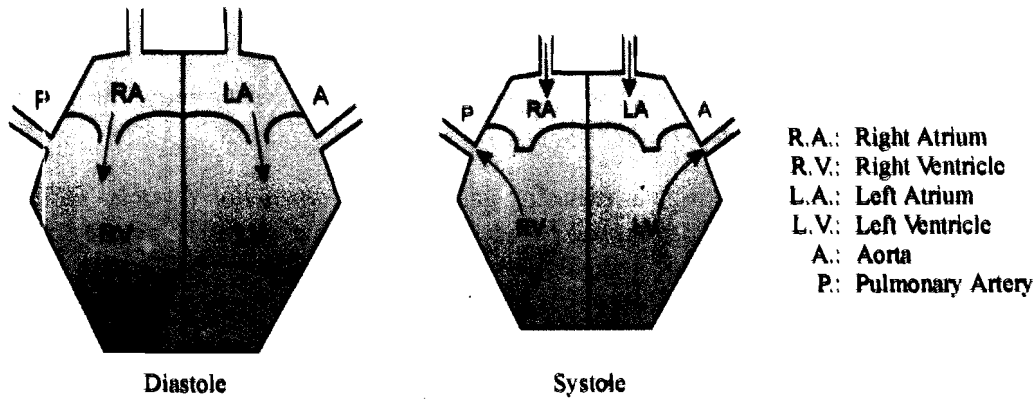


Figure 4.7: Phases of cardiac cycle

Let us now study about both of these phases in the right heart (right atrium and ventricle).

- 1) *The diastole phase* - During this phase, the atria and ventricles are relaxed and the atrioventricular valves are open. De-oxygenated blood from the superior and inferior vena cava flows into the right atrium. The open atrioventricular valves allow the blood to pass through to the ventricles, as you can see in the Figure 4.7. The SA node contracts, triggering the atria to contract. The right atrium empties its contents into the right ventricle. The tricuspid valve prevents the blood from flowing back into the right atrium.
- 2) *The systole phase* - During this phase, the right ventricle receives impulses from the purkinje fibers and contracts. The atrioventricular valves close and the semi-lunar valves open. The de-oxygenated blood is pumped into the pulmonary artery.

The pulmonary valve prevents the blood from flowing back into the right ventricle. The pulmonary artery carries the blood to the lungs. There, the blood picks up oxygen and is returned to the left atrium of the heart by the pulmonary veins. This is more clearly depicted in the Figure 4.8.

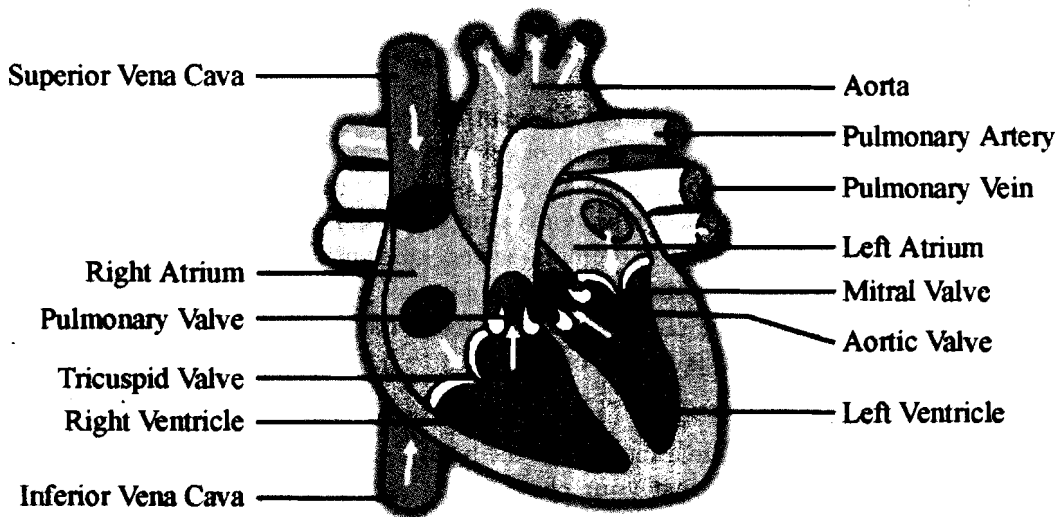


Figure 4.8: The cardiac cycle

Simultaneously in the left heart during diastole period, the semilunar valves close and the atrioventricular valves open. Blood from the pulmonary veins fills the left atrium. Blood from the vena cava is also filling the right atrium. The SA node contracts again, triggering the atria to contract. The left atrium empties its contents into the left ventricle. The mitral valve prevents the oxygenated blood from flowing back into the left atrium.

During the systole phase, the atrioventricular valves close and the semilunar valves open. The left ventricle receives impulses from the Purkinje fibers and contracts. Oxygenated blood is pumped into the aorta. The aortic valve prevents the oxygenated blood from flowing back into the left ventricle.

The aorta branches out to provide oxygenated blood to all parts of the body. The oxygen-depleted blood is returned to the heart via the vena cava.

The heartbeat follows a regular recurring pattern. *One contraction (systole) followed by relaxation (diastole) of the heart is known as cardiac cycle.* There are several mechanical, physical and electrical events associated with the cardiac cycle. Understanding the time course of these events is interesting and also useful in management of cardiovascular diseases.

Suppose the heart rate is 75 per minute,

If 75 beats take 1 minute, then, 1 beat will take  $1/75 \text{ min} = 1 \times 60 \text{ sec}/75 = 0.8 \text{ sec}$

Thus the duration of the cardiac cycle is 0.8 sec. Out of this, ventricular systole (generally called “the systole”) lasts 0.3 sec and ventricular diastole (generally called “the diastole”) lasts 0.5 sec. When the heart rate increases, the duration of cardiac cycle decreases. In such a situation, the reduction in duration of diastole is greater than that of systole.

Next, let us learn about the heart sound.

### *Heart Sounds*

Have you ever heard your heart go lub...dub...? What are these sounds? “Lub” is the closing of the atrioventricular valves while “dub” is the closing of the pulmonary and aortic valves. Place a hand against your chest, preferably on the left side. Why left side? You know now that the heart is placed towards the left like a pyramid of Egypt only inverted with its apex placed on the left side.... What do you feel? Do you feel the heart beating? Yes, that is the thumping motion of the left ventricle against the chest. Then, why is there a gap in between the two thumps? It’s simply because during this time, the heart is resting. Do you remember what this period is called? Yes, it is the *diastole*...and the importance of this resting phase is that the heart receives its nutrition and air supply during this period. It’s a well-deserved breather for an organ that goes on and on for a hundred odd years without stopping.

Now that you have palpated or felt the heart, let us hear or auscultate the chest. Place your ear on the chest of some friend or relative. What do you hear? Doctors use an instrument called as *stethoscope* to listen to your heart beat. It is an instrument used for examining the heart and lungs by conveying to the ear of the examiner, the sounds produced in the thorax. This is built on the very same principle of talking to someone 100 m away with a cord of string attached or a hollow bamboo conducting sound better when placed against the chest. The stethoscope has a bell and a conducting tube and earpieces. The bell is placed on the chest to hear the wonders the heart unveils before us.

The tap of the music is like the beat of the heart. Snap your fingers, tap your feet or make a baby sleep against your chest, what are the common factors? It’s the synchronization with your heartbeat. The baby has a bonding and recognizes the mother from the heartbeat when placed on her chest from the womb memory. The song troupes synchronize their rhythmic dance routine by placing a hand on the heart. That’s our fascinating four chambered heart beating as one, rhythmically that gets the coordination of the choreographic team right.

Thus we reach where we started from and the cardiac cycle starts all over again.

**Check Your Progress Exercise 3**

1) What is cardiac output? How is it calculated?

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2) What do you understand by the term 'cardiac cycle'? Discuss its phases.

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3) What are the two heart sounds and their significance?

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Next, let us get to know about blood pressure.

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## 4.7 BLOOD PRESSURE

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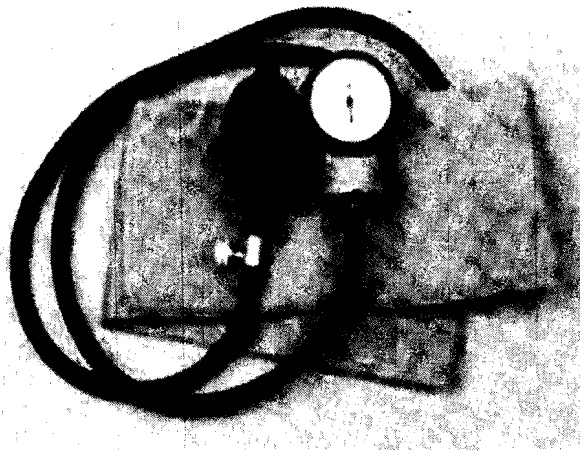
You may have seen someone record the blood pressure or someone may have taken your blood pressure.

Blood pressure is a very common term used casually in everyday conversation. People often advise others to check anger in order to prevent the blood pressure from going up, but forget it when they themselves are angry. Any declaration of high blood pressure by a physician immediately brings a lot of worries to the patient. However, blood pressure is most important for maintaining blood flow through tissues. Thus, blood pressure is a necessary evil for survival. As long as it is within the normal limits, it does not bother us. In fact, it keeps us alive. So then what is blood pressure?

### 4.7.1 What is Blood Pressure?

Blood pressure, without qualification, refers to *the arterial blood pressure*. Hence, it is the lateral pressure exerted by the column of blood on the walls of the arteries. The pressure is not steady, it fluctuates during the cardiac cycle. During ventricular systole, arterial blood pressure is higher than that during diastole. The normal range of systolic blood pressure is 100 to 140 mm Hg and that of diastolic pressure is 60 to 90 mm of Hg. Blood pressure is commonly measured using a mercury manometer and is commonly expressed in millimeters of mercury (mm Hg).

Blood pressure may be measured fairly accurately by *sphygmomanometer* – this is a noninvasive method. The sphygmomanometer consists of an inelastic cuff, which contains an inflatable rubber bag. As you can see from the Figure 4.9, the rubber bag is connected to two tubes. One tube connects the bag to a manometer and the other tube is connected to a bulb fitted with a valve, which may be used for inflating air and deflating the rubber bag.



**Figure 4.9: Sphygmomanometer - BP apparatus**

The cuff is snugly placed around the arm, 3-4 cm above the elbow joint. It is inflated till the cuff pressure stops all the blood flow through the brachial artery. If kept for too long, the patient's hand feels numb and may pain. The cuff pressure is gradually (@1mm/sec) brought down. Then blood starts flowing into the brachial artery, as soon as the cuff pressure falls just below the systolic arterial pressure. As you are deflating cuff, place bell of stethoscope over brachial artery at the elbow and hear the loud tapping sounds getting muffled and finally disappear. The pressure at which the sounds just appear is known as *systolic pressure*. The pressure at which the sounds just disappear is taken as the *diastolic pressure*.

#### *Mean blood pressure*

Mean blood pressure is not the arithmetic mean or average of the systolic and diastolic blood pressure. This is because systole is much shorter than diastole. Therefore, arterial pressure is near the diastolic pressure for a longer part of the cardiac cycle than it is nearer the systolic pressure. A mathematical pressure method for finding the true mean pressure is when one-third of the pulse pressure is added to the diastolic pressure, we get a value which is very close to the mean pressure determined by the graphic method. That is, in the simpler terms, multiplying the diastolic pressure by two, adding the systolic pressure, and then dividing this sum by three estimates it. Therefore, if systolic pressure is 110 mm Hg and the diastolic pressure is 80 mm Hg, then

$$\begin{aligned} \text{Mean blood pressure} &= 80 + \frac{1}{3}(110 - 80) \text{ mm Hg or } 80 \times 2 + 110/3 \text{ mm Hg} \\ &= 90 \text{ mm Hg} \end{aligned}$$

Mean blood pressure is the major determinant of blood flow through the tissues.

#### *How to record the blood pressure?*

Blood pressure is recorded as two numbers—the systolic pressure (as the heart beats) over the diastolic pressure (as the heart relaxes between beats). The measurement is written one above or before the other, with the systolic number on top and the diastolic number on the bottom. For example, a blood pressure measurement of 120/80 mm Hg (millimeters of mercury) is expressed verbally as “120 over 80.”

The difference between systolic and diastolic pressure is known as *pulse pressure*. Therefore, the normal pulse pressure is approximately  $(120 - 80) = 40$  mm Hg.

Let us now study about the factors affecting the blood pressure.

### **4.7.2 Factors Affecting Blood Pressure**

From Ohm's Law, it is easy to deduce that the blood pressure would roughly equal to the product of cardiac output (CO) and peripheral resistance (PR).

$$\text{Hence, BP} = \text{CO} \times \text{PR}$$

Hence, the factors which alter the cardiac output or peripheral resistance would also affect the arterial blood pressure.

Let us learn about these factors next.

### *Factors Affecting Cardiac Output*

Changes in the cardiac output affect mainly the systolic pressure while changes in the peripheral resistance affect mainly the diastolic pressure. Let us now study what are these changes. We shall first have a look at the consequences of increased cardiac output.

#### ● *Increased Cardiac Output*

- i) An increase in blood volume increases the venous return and leads to an increased stroke volume. The increase in the blood volume may be due to water retention, which may follow sodium retention, due to increased aldosterone secretion or some other factors.
- ii) During exercise, the cardiac output increases due to an increase in both stroke volume and heart rate. Hence, systolic pressure generally increases during exercise.
- iii) An increase in heart rate does not always alter the blood pressure because if stroke volume decreases simultaneously, cardiac output does not change significantly.
- iv) Emotional excitement increases systolic, as well as, diastolic pressure because of increased sympathetic adrenal activity.

Now let us see what happens when cardiac output decreases.

#### ● *Decreased Cardiac Output*

- i) *Change of posture:* When a person stands up from lying down posture, there is an immediate but transient fall in systolic blood pressure. This is because, upon standing, there is a pooling of venous blood in the lower limbs due to gravity. This leads to a decreased venous return, which in turn decreases the stroke volume and cardiac output causing a fall in the systolic pressure. However, within fifteen seconds, the baroreceptor reflexes bring about a cardio acceleration and vasoconstriction. As a result, the blood pressure soon returns to normal or may rise above value due to overcompensation. You may work out the changes on standing.
- ii) *Hypovolemia or reduction in blood volume:* Hypovolemia leads to a decrease in cardiac output, which causes a fall in the systemic blood pressure. Hypovolemia may be produced by hemorrhage. However, the baroreceptor reflex corrects the fall in blood pressure due to moderate hemorrhage. If the loss of blood is less than 10% of the total blood volume, the compensatory mechanisms succeed in stabilizing the blood pressure at a level below the normal pressure. But if the loss is more than 30% of the blood volume, the compensatory mechanisms may fail leading to an irreversible and fatal fall in blood pressure.
- iii) *Cardiac compression:* Rapid accumulation of fluid in the pericardial sac decreases ventricular distensibility leading to a decrease in cardiac output.
- iv) *Myocardial ischemia and infarction:* Myocardial ischemia (insufficient blood flow) or myocardial infarction (death of heart cells) reduces myocardial contractile force and thereby reduces the cardiac output. Further, there is also a reflex fall in systemic blood pressure by liberation of certain metabolites which stimulate the ventricular unmyelinated afferent fibers.
- v) *Trauma:* Any injury which causes intense pain produces a fall in blood pressure.

Next, let us look at the factors affecting peripheral resistance which ultimately influences the blood pressure.

#### *Factors Affecting Peripheral Resistance*

Resistance offered by arterioles or resistance vessels, as you have read above, is termed as *peripheral resistance*. Changes in peripheral resistance mainly affect diastolic pressure. The factors that affect peripheral resistance, include:

- i) If more than 30% of the blood volume of a person is lost through bleeding (haemorrhage), the compensatory mechanism may fail. In that case, there may be a reduction in the vasomotor center activity and a fall in peripheral resistance, which may turn out to be irreversible. The perfusion to the tissues is decreased. This condition is known as *shock*.
- ii) Some emotionally disturbing events, such as sight of blood or frightening objects may lead to fainting due to generalized vasodilation. Although this is called *vaso-vagal attack*, it seems to be mediated by sympathetic cholinergic vasodilatation fibers, which supply skeletal muscles. It is generally short-lasting. The treatment is to lie down so that the blood flow, especially to the brain, is maintained. If this treatment is not promptly instituted, the person may faint and fall due to a reduction in blood flow to the brain – a condition known as *syncope*. Syncope is actually *a spontaneous loss of consciousness caused by insufficient blood to the brain*. It may be considered nature's way of instituting the treatment. Although rude and potentially traumatic, the fall can be life-saving.
- iii) Anaphylactic shock, trauma, peritonitis (inflammation of the peritoneum), crush syndrome or an allergy following a bee bite. In all these conditions, there is production of some toxic substances, which lead to systemic vasodilation and increased capillary permeability. Increased vascular capacity due to vasodilation and reduced volume due to increased capillary permeability is a dangerous combination. It frequently leads to circulatory shock.
- iv) Stimulation of myelinated pain fibers may produce generalized vasodilation.

Besides, the factors discussed above, there are a few other factors affecting the viscosity of blood which also impacts on blood pressure. These are discussed next.

#### *Factors Affecting Viscosity of Blood*

Viscosity of blood affects the systemic blood pressure in the same way as changes in the peripheral resistance. In anaemia, viscosity of blood is low and a fall in blood pressure may occur. In polycythemia (increase in the production of red blood cells or erythrocytes) viscosity is high and blood pressure may rise.

Next, we shall read about the factors regulating blood pressure.

### **4.7.3 Factors Regulating Blood Pressure**

Blood pressure regulating mechanisms may be classified into two categories:

- a) *Short term regulating mechanisms*, which regulate and maintain the normal blood pressure inspite of factors which tends to disturb it every minute. These regulators are mainly neural and includes baroreceptors, chemoreceptors etc.
- b) *Long term regulating mechanisms*, which adjust the body fluid volume. These regulations are mainly through hormones. The hormonal mechanisms include the catecholamine release from adrenal medulla, renin-angiotensin mechanism of kidney etc.

**Check Your Progress Exercise 4**

1) What is Blood Pressure? What is the normal range?

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2) What is Mean Blood Pressure? How is it calculated?

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3) Enumerate the factors increasing and decreasing cardiac output.

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4) What is peripheral resistance? Which factors change peripheral resistance?

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5) Enumerate the mechanisms that regulate blood pressure.

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## 4.8 PATHOPHYSIOLOGY OF HYPERTENSION

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In spite of the regulatory mechanisms discussed above, they have their own limitations and the blood pressure frequently shows persistent elevation. Some rise in blood pressure with age is accepted as physiological. The rule of thumb is to consider the systolic blood pressure as normal if it is less than  $(100 + \text{age in years})$  mm Hg. However, many specialists have an evidence to believe that a rise in blood pressure with age is a process we have to pay for our lifestyle, especially due to the high salt content of our diet and sedentary habits.

Some experts assert that if no additional salt is added to food throughout life, the blood pressure will stay constant throughout life. Since this hypothesis cannot be widely tested on human beings at the present stage of our civilization, we have to accept some rise in blood pressure with age as a part of the aging process. Although the change is gradual, and there is not a sharp dividing line between normal and high blood pressure, an arbitrary upper limit is 140 and 90 mm Hg for systolic and diastolic blood pressure, respectively. Out of the two, the diastolic pressure is more reliable for determining whether a person is hypertensive because the systolic pressure shows wider fluctuations from time to time in the same individuals. In any case, a single casual reading is not enough to label a person hypertensive, especially in the borderline cases. But if the diastolic pressure is

repeatedly found to be 90 mm Hg or above, on at least three occasions, the individual may be considered hypertensive, irrespective of age.

The classification of hypertension is presented next.

### *Classification of Hypertension*

In most cases of hypertension, the cause is only vaguely known in terms of old age, familial tendency, emotional stress, sedentary life, overeating etc. In these cases, where the basic cause of hypertension cannot be pin-pointed, the hypertension is said to be *primary or essential*.

In the few cases where the underlying cause is known, the hypertension is said to be *secondary*. Another way to classify hypertension is in the terms of its prognosis. In cases where it progresses slowly, hypertension is called *benign*. In contrast, *malignant hypertension* progresses rapidly, is severe and often leads to death within two years if not treated. Some of the common causes of secondary hypertension are listed in Table 4.1.

**Table 4.1: Common causes of secondary hypertension**

Cause	Mechanism
Renal disease	Excess renin production leading to high level of angiotensin II.
Hyperfunctioning of adrenal cortex	Excess aldosterone, a mineralocorticoid secretion leading to salt and water retention
a) Conn's disease	Excess glucocorticoid secretion.
b) Cushing's syndrome	Glucocorticoids also have weak mineralocorticoid activity (i.e. salt and water retention).
Long term oral contraceptive use	Mineralocorticoid activity of estrogen and progesterone.
Phaeochromocytoma	Tumor of adrenal medulla leading to excess secretion of adrenaline and nor-adrenaline.
Polycythemia	Increased viscosity of blood leading to higher peripheral resistance.

The treatment of hypertension depends on its severity and cause. In essential hypertension, relaxation techniques, tranquilizers, vasodilators, diuretics,  $\beta$ -blockers or some other drugs may be useful in different combinations. In case of secondary hypertension, it is best to treat the basic causes.

Other than hypertension, there are a few other conditions related to the heart functioning. We shall get to know them next.

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## **4.9 MYOCARDIAL ISCHEMIA AND INFARCTION**

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A patient experiencing a heart attack suffers from severe pain on the left side of chest that radiates to the left arm. He may sweat a lot, vomit and may not be able to even move because of the pain. The cause of heart attack is eating faulty diet. An old saying in the Gita goes: *your mind and body is what you eat*.

A person over the years may eat fatty diet such as butter, meat and eggs rich in cholesterol. These may accumulate on the coronary vessels and obliterate it. When coronary blood flow decreases due to *atherosclerosis*, it leads to *myocardial ischemia*. If myocardial ischemia is very severe, or a coronary artery is blocked due to thrombosis, embolism or spasm, it causes death of the myocardium supplied by the blocked artery.

The condition is known as *myocardial infarction*, and is a serious condition, which may lead to death. The doctors dissolve the clot or remove the obstruction in the artery to restore blood supply to the cardiac muscle.

Earlier we referred to atherosclerosis. What is *atherosclerosis*? Atherosclerosis is *the hardening of the walls of the arteries caused by fatty deposits that build on the inner walls of the arteries which interfere with blood flow* as shown in Figure 4.10.

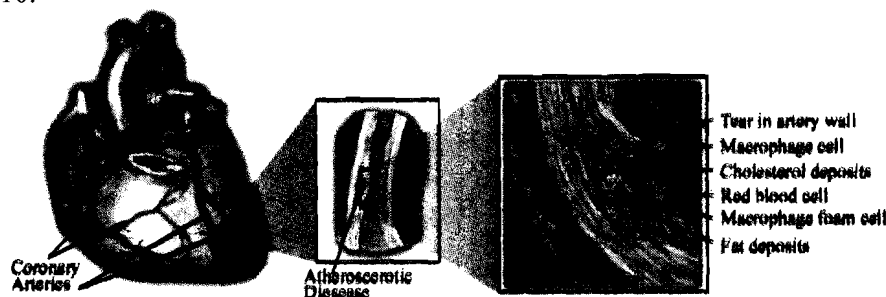


Figure 4.10: Atherosclerosis

Insufficient blood flow to the heart muscles from narrowing of coronary artery can cause chest pain referred to as *angina pectoris*.

So what do we do keep our heart healthy? Read the next section and find out. Do practise what you learn next.

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## 4.10 AEROBICS EXERCISE AND DIET: HOW TO KEEP YOUR HEART HEALTHY

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Aerobics or exercise keeps the muscles of the body toned. The heart receives blood supply, rich in oxygen and nutrients. It pumps in more amount of blood over the years. Research has shown that the heart is larger in trained athletes. The person keeps happy because of the happy polypeptides, such as 'endorphins', that are released. The exerciser delays retirement from the job as studies have shown. The morning is the best time to exercise when the sun rises and the air is fresh. The chirping of the birds has a positive therapeutic effect on the health.

Supplementing exercise with balanced diet is a must. Salt needs to be restricted in hypertensive as salt causes water retention and fluid overload, aggravating blood pressure. Less of fats and eating lots of fruits, green vegetables, and milk and cereals constitute a healthy nutritious diet.

We shall end our study of coronary vascular system with a discussion on ECG.

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## 4.11 ECG — WHAT IT IS AND WHY DO WE NEED IT?

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You may have seen a flat line on the monitor screen in the movies or in the hospital when the heart stops beating and the doctor declares that the patient is clinically dead. The flat line recording is an *electrocardiogram* or an *ECG*. It is an *electrical recording of the heart, made by electrodes, picking up cardiac activity from the human body surface*.

The heart may be considered a small generator. This generator is situated in the human body, which is a good volume conductor. Thus, it is possible to record the electrical activity of the heart from the surface of the body even at a considerable distance from the heart. It is the insignia or language of the beating heart. It's a simple but commonly used procedure by the doctors to know about the patient's heart. It tells us about the heart rate, rhythm, regularity and state of every heart chamber.

Changes in hemodynamics, damage to the muscle fibers or a change in the ionic environment of the heart affects the ECG. The doctor knows if the heart is diseased or normal. It thus serves as an important diagnostic and prognostic tool for the assessment of cardiovascular function. It has to be combined, however, with clinical judgment and other laboratory investigation for greater reliability. The doctor decides on what medication or action he may have to prescribe by just looking at an ECG and by correlating with other findings. As a student of dietetics we need not ponder much on this, however, we must learn about a few important diseased states diagnosed by an ECG. These are:

- *Sinus Bradycardia*

If the SA node discharge rate is slow, the heart rate is slow. ECG is normal in every respect except that the heart rate at rest is below 60 per minute. This is known as *sinus bradycardia*. It may be normally seen in athletes or abnormality in patients having an increased intracranial pressure or hypothyroidism.

- *Sinus Tachycardia*

When the only abnormality is that the heart rate at rest is more than 100 per minute, the condition is known as *sinus tachycardia*. It may be normally seen in emotional stress or exercise or abnormality in fever, hyperthyroidism or anaemia.

- *Sinus Arrhythmia*

In some adults and most children, there is an increase in heart rate during inspiration and a decrease during expiration. This rhythm is most commonly seen with breathing due to fluctuations in parasympathetic vagal tone. During inspiration, the chest expands creating a suction force that results in greater backflow of blood into the atria from the veins. Greater filling of right atrium results in higher heart rate, possibly by stretching the SA node.

- *Ventricular Fibrillation*

This is a fatal condition in which the heart beats like a bag of worms and the patient may die in a couple of hours, if the action is not taken immediately. You are already aware that the heart beats when electrical signals move through it. In case of ventricular fibrillation, the heart's electrical activity becomes disordered. When this happens, the heart's lower (pumping) chambers contract in a rapid, unsynchronized way. The ventricles "flutter" rather than beat. The heart does not pump sufficient blood to the entire body. The patient has no pulse and the heart is quivering. It may be necessary to give a high voltage DC shock to revive the patient. You may have seen this being done when the doctor places electrodes on the chest to provide electrical shock to jump-start the dying heart.

The action of the defibrillator is based on the principle that a high voltage current throws the entire ventricular musculature into contraction miraculously and simultaneously. Ventricular fibrillation may be seen in the following two conditions:

- 1) when a person is electrocuted, and
- 2) as a consequence of heart attack, myocardial infarction.

There are certain abnormalities caused due to changes in serum electrolyte that influence ECG. We shall learn about these changes next.

*Abnormalities due to Changes in Serum Electrolytes*

The intracellular and extracellular electrolyte concentration difference is responsible for the resting membrane potential and the electrical activity of all excitable tissues of the body.

Since heart is a large organ made up of excitable tissues, serum electrolyte changes alter its electrical activity and hence the ECG. Serum electrolyte changes may affect life and can be suspected from ECG at an early stage so that the fatal impairment of cardiac function can be avoided. The serum electrolyte changes are discussed next.

- *Sodium*

A decrease in sodium ion concentration decreases the voltage in ECG. ECG changes are similar to pericarditis or inflammation of the pericardium.

- *Potassium*

An increase or a decrease in potassium ion concentration changes ECG considerably. It affects the depolarization or repolarization of the cardiac muscle. The heart becomes floppy in cases of hyperkalemia (increase in potassium ion concentration).

- *Calcium*

Hypercalcemia may lead to an increased force of contraction of the heart.

**Check Your Progress Exercise 5**

1) Explain the following terms:

a) Sinus bradycardia

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b) Sinus tachycardia

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c) Sinus arrhythmia

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d) Ventricular fibrillation

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2) Define hypertension. How do you classify hypertension?

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3) What are the possible causes and associated mechanisms of hypertension of unknown origin?

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4) What is heart attack? What may cause a heart attack?

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5) Prescribe a healthy diet for a patient with heart disease.

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**4.12 LET US SUM UP**

In this unit, we have learnt about the heart and its connecting blood vessels, its structure and functioning of the heart. We have also studied about blood pressure and heart attack and understood the role of exercise and diet in keeping the heart toned and body fit.

**4.13 GLOSSARY**

<b>Atherosclerosis</b>	: lipid and fat deposition in the walls of the arteries.
<b>Bathmotropic</b>	: excitability of the cardiac tissue.
<b>Cardiac index</b>	: the cardiac output per unit body surface area.
<b>Cardiac output</b>	: the amount of blood pumped by each ventricle per minute. Cardiac output = Stroke Volume × Heart Rate.
<b>Coronary sinuses</b>	: the vessels that carry venous blood to the heart from its own musculature
<b>Coronary arteries</b>	: two arteries arising from aorta and supplying nutrition and O <sub>2</sub> to heart.
<b>Dromotropic</b>	: rate of conduction.
<b>Embolism</b>	: undissolved matter in blood.
<b>Extrasystole</b>	: an ‘extra’ contraction due to stimulation of the heart during diastole relative refractory period.
<b>Inotropic</b>	: force of contraction.
<b>Myocardial ischemia</b>	: decreased blood supply to the heart.
<b>Parasympathetic</b>	: system of nerves that decreases the heart rate, rate of conduction and excitability of the heart tissue.
<b>Spasm</b>	: sudden contraction or narrowing.
<b>Sympathetic</b>	: system of nerves that increases the heart rate, force of contraction, rate of conduction and excitability of the heart tissue.
<b>Sympathetic cholinergic nerves</b>	: these fibers produce vasodilation of blood vessels of skeletal muscles.
<b>Thrombosis</b>	: Intravascular clotting
<b>Vagus Nerve</b>	: the X cranial nerve.

## 4.14 ANSWERS TO CHECK YOUR PROGRESS EXERCISES

### Check Your Progress Exercise 1

1) The four chambers of the heart are right ventricle, left ventricle, right atrium and left atrium.

2) The blood flow in the heart can be described as follows:

The right atrium receives the blood from the body through two large veins – the superior and the inferior vena cava. It empties blood into the right ventricle. The right ventricle pumps the deoxygenated blood to the lungs through the pulmonary artery. The pulmonary artery carries blood from the heart to the lungs.

The oxygenated blood from lungs reaches left atrium via pulmonary veins. It empties the blood into left ventricle, which on contraction pumps oxygenated blood into aorta and its branches (systemic circulation).

3) The aorta is large diameter blood vessel, with large amount of elastic tissue in the walls but arterioles have thick muscular walls. The aorta divides into arteries, arterioles.

4) Capillaries are thin walled vessels lined by only a single lining of cells called endothelium. This enables free exchange of nutrients at the tissue level.

5) Cholinergic neurons produce vasodilation of blood vessels of skeletal muscles unlike the sympathetic nerves that cause vasoconstriction.

### Check Your Progress Exercise 2

1) The pacemaker tissue sets the pace at which the heart beats. Pacemaker and conduction tissues are made up of modified cardiac muscle, which are not well striated. They conduct electrical impulses to the cardiac muscle. The pacemakers of our heart are sinoatrial node (SA node), atrioventricular node (AV node) and Bundle of His.

2) The unique properties of our heart are:

- Automaticity
- Rhythmicity
- Long Refractory Period
- Extrasystole and Compensatory Pause
- Length-Tension Relationship

3) If a sufficiently strong electrical stimulus is applied during relative refractory period, it leads to a contractile response. Since this contraction appears earlier than the normally expected contraction, it may be considered an 'extra' contraction. Hence it is called extrasystole. The pause in cardiac activity is called compensatory pause.

### Check Your Progress Exercise 3

1) Cardiac output is the amount of blood pumped by each ventricle per minute. It is calculated as:

$$\text{Cardiac output} = \text{Stroke Volume} \times \text{Heart Rate}$$

2) The sequence of events that occur when the heart beats is referred to as the cardiac cycle. Its two phases in the right heart (rt atrium and rt ventricle) are:

- Diastole phase : During this phase, the atria and ventricles relax and atrio-ventricular (AV) valves open. Deoxygenated blood from the superior and inferior vena cava flows into the right atrium. The open AV valve allows the blood to pass through to the ventricles. The SA node contracts, triggering the atria to contract. The right atrium empties its contents into the right ventricle. The tricuspid valve prevents the blood from flowing back into the right atrium.
  - Systole Phase: During this phase, the right ventricle receives impulses from the purkinje fibers and contracts. The AV valves close and the semi-lunar valves open. The deoxygenated blood is pumped into the pulmonary artery.
- 3) The 2 heart sounds stemming from cardiac contraction/relaxation are normally heard through a stethoscope placed on the chest wall. The first sound, a soft, low pitched Lub, is associated with closure of AV valves at the onset of systole. The second, a louder dup, is associated with closure of pulmonary and aortic valves at the onset of diastole.

Clinically they signify regular beatings of the heart. Various events can be timed with the help of heart sounds. Time between 1<sup>st</sup> and 2<sup>nd</sup> sound marks systole and between 2<sup>nd</sup> and 1<sup>st</sup> sound as diastole.

#### Check Your Progress Exercise 4

- 1) Blood Pressure refers to arterial blood pressure. It is the lateral pressure exerted by the column of blood on the walls of the arteries. The normal range of systolic blood pressure is 100 to 140 mm Hg, and that of diastolic pressure is 60 to 90 mm of Hg.

- 2) Mean blood pressure is the major determinant of blood flow through tissue.

Mean blood pressure = systolic pressure + 1/3(systolic pressure – diastolic pressure)

- 3) An increased cardiac output may be caused due to an increase in blood volume or during exercise cardiac output and due to emotional excitement.

Decreased cardiac output is caused by change of posture on standing, reduction in blood volume, cardiac compression and by myocardial ischemia and infarction .

- 4) Resistance offered by arterioles or resistance vessels is termed as peripheral resistance. Changes in peripheral resistance mainly affect diastolic pressure.

Factors which change peripheral resistance are:

- Loss of blood volume
  - Emotionally disturbing events
  - Anaphylactic shock, trauma, peritonitis (inflammation of the peritoneum), crush syndrome or an allergy following a bee bite.
- 5) Blood pressure regulating mechanisms may be classified into two categories as:
- a) Short term regulating mechanisms, which regulate and maintain the normal blood pressure in spite of factors which tend to disturb it every minute. There regulators are mainly neural.
  - b) Long term regulating mechanisms, which adjust the body fluid volume. These regulations are mainly hormones.

Long term regulation of blood pressure is primarily regulation of blood volume, which is a part of regulation of fluid balance. It is accomplished by humoral mechanisms involving kidneys. The principal hormone ADH, aldosterone, renin-angiotensin system and atrial naturetic peptide.

**Check Your Progress Exercise 5**

- 1) a) SA node discharge rate is slow, the heart rate is slow. The heart rate at rest is below 60 per minute.
  - b) The heart rate at rest is more than 100 per minute; it may be normally seen in emotional stress or exercise or in fever or Anaemia.
  - c) In some adults and most children, there is an increase in heart rate during inspiration and a decrease during expiration.
  - d) The heart beats like a bag of worms and the patient may die in a couple of hours if action is not taken immediately. The heart does not pump sufficient blood to the entire body. May occur as a consequence of heart attack, myocardial infarction.
- 2) Systolic and diastolic blood pressure greater than 140 and 90 mm Hg respectively at least during three separate recordings is termed as hypertension. The cases where the basic cause of hypertension cannot be pin pointed, the hypertension is said to be "primary or essential". The cases whose underlying cause is known, the hypertension is said to be secondary.

Hypertension can also be classified in terms of its prognosis. In cases where it progresses slowly, hypertension is called benign. In contrast, malignant hypertension progresses rapidly, is severe, and often leads to death within two years if no treated.

- 3) Common causes of Secondary Hypertension are:

Cause	Mechanism
Renal disease	Excess renin production leading to high level of angiotensin II.
Hyperfunctioning of adrenal cortex	Excess aldosterone, a mineralocorticoid secretion leading to salt and water retention
a) Conn's disease	Excess glucocorticoid secretion.
b) Cushing's syndrome	Glucocorticoids also have weak mineralocorticoid activity (i.e. salt and water retention).
Long term oral contraceptive use	Mineralocorticoid activity of estrogen and progesterone.
Phaeochromocytoma	Tumor of adrenal medulla leading to excess secretion of adrenaline and nor-adrenaline.
Polycythemia	Increased viscosity of blood leading to higher peripheral resistance.

- 4) A patient experiencing a heart attack suffers from severe pain on the left side of chest that radiates to the left arm. He may sweat a lot, vomit and may not be able to even move because of the pain. A person over the years may eat fatty diet such as butter, meat, and eggs rich in cholesterol. These may accumulate on the coronary vessels and obliterate it. When coronary blood flow decreases due to atherosclerosis it leads to myocardial ischemia. If myocardial ischemia is very severe or a coronary artery is blocked due to thrombosis, embolism or spasm, it causes death of the myocardium supplied by the blocked artery. The doctors dissolve the clot or remove the obstruction in the artery to restore blood supply to the cardiac muscle.
- 5) A balanced diet is a must. Salt needs to be restricted in hypertensive as salt causes water retention and fluid overload, aggravating blood pressure. Sprouted *dals* are very good for the heart as they have antioxidant properties. Less of fats and eating lots of fruits, green vegetables, and milk and cereals constitute a healthy nutritious diet.