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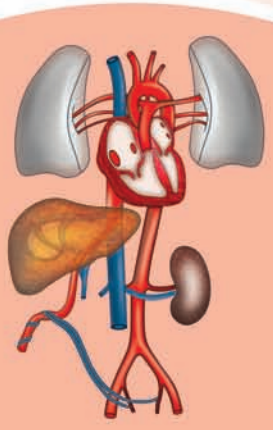

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Introduction to Cardiovascular System

Chapter 89

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CARDIOVASCULAR SYSTEM

Cardiovascular system includes **heart** and **blood vessels.** Heart pumps blood into the blood vessels. Blood vessels circulate the blood throughout the body. Blood transports nutrients and oxygen to the tissues and removes carbon dioxide and waste products from the tissues.

HEART

Heart is a muscular organ that pumps blood throughout the circulatory system. It is situated in between two lungs in the mediastinum. It is made up of four chambers, two atria and two ventricles. The musculature of ventricles is thicker than that of atria. Force of contraction of heart depends upon the muscles.

RIGHT SIDE OF THE HEART

Right side of the heart has two chambers, **right atrium** and **right ventricle.** Right atrium is a thin walled and low pressure chamber. It has got the pacemaker known as sinoatrial node that produces cardiac impulses and atrioventricular node that conducts the impulses to the ventricles.

Right atrium receives venous (deoxygenated) blood via two large veins:

1. **Superior vena cava** that returns venous blood from the head, neck and upper limbs

2. **Inferior vena cava** that returns venous blood from lower parts of the body (Fig. 89.1).

Right atrium communicates with right ventricle through tricuspid valve. Wall of right ventricle is thick. Venous blood from the right atrium enters the right ventricle through this valve.

From the right ventricle, pulmonary artery arises. It carries the venous blood from right ventricle to lungs. In the lungs, the deoxygenated blood is oxygenated.

■ LEFT SIDE OF THE HEART

Left side of the heart has two chambers, **left atrium** and **left ventricle.** Left atrium is a thin walled and low pressure chamber. It receives oxygenated blood from the lungs through pulmonary veins. This is the only exception in the body, where an artery carries venous blood and vein carries the arterial blood.

Blood from left atrium enters the left ventricle through mitral valve (bicuspid valve). Wall of the left ventricle is very thick. Left ventricle pumps the arterial blood to different parts of the body through **systemic aorta.**

■ SEPTA OF THE HEART

Right and left atria are separated from one another by a fibrous septum called **interatrial septum.** Right and left ventricles are separated from one another by **interventricular septum.** The upper part of this septum is a membranous structure, whereas the lower part of it is muscular in nature.

■ LAYERS OF WALL OF THE HEART

Heart is made up of three layers of tissues:

1. Outer pericardium
2. Middle myocardium
3. Inner endocardium.

■ PERICARDIUM

Pericardium is the outer covering of the heart. It is made up of two layers:

i. Outer parietal pericardium
ii. Inner visceral pericardium.

The space between the two layers is called **pericardial cavity** or **pericardial space** and it contains a thin film of fluid.

i. *Outer Parietal Pericardium*

Parietal pericardium forms a strong protective sac for the heart. It helps also to anchor the heart within the mediastinum.

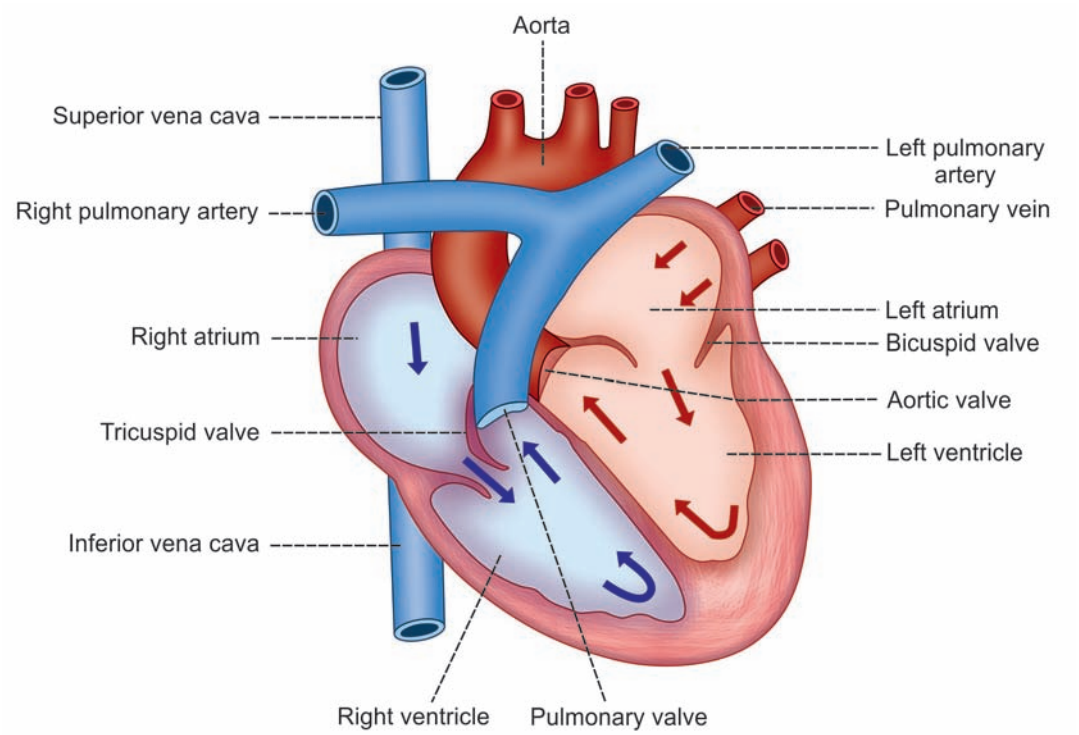

FIGURE 89.1: Section of the heart

Parietal pericardium is made up two layers:

a. Outer fibrous layer
b. Inner serous layer.

Fibrous layer

Fibrous layer of the parietal pericardium is formed by thick fibrous connective tissue. It is attached to the diaphragm and it is continuous with tunica adventitia (outer wall) of the blood vessels, entering and leaving the heart. It is attached with diaphragm below. Because of the fibrous nature, it protects the heart from over stretching.

Serous layer

Serous layer is formed by mesothelium, together with a small amount of connective tissue. Mesothelium contains squamous epithelial cells which secrete a small amount of fluid, which lines the pericardial space. This fluid prevents friction and allows free movement of heart within pericardium, when it contracts and relaxes. The total volume of this fluid is only about 25 to 35 mL.

ii. *Inner Visceral Pericardium*

Inner visceral pericardium lines the surface of myocardium. It is made up of flattened epithelial cells. This layer is also known as **epicardium.**

■ MYOCARDIUM

Myocardium is the middle layer of wall of the heart and it is formed by cardiac muscle fibers or cardiac myocytes. Myocardium forms the bulk of the heart and it is responsible for pumping action of the heart. Unlike skeletal muscle fibers, the cardiac muscle fibers are involuntary in nature.

Refer Chapter 28 for features of cardiac muscles.

Myocardium has three types of muscle fibers:

i. Muscle fibers which form contractile unit of heart
ii. Muscle fibers which form pacemaker
iii. Muscle fibers which form conductive system.

i. *Muscle Fibers which Form Contractile Unit of Heart*

These cardiac muscle fibers are striated and resemble the skeletal muscle fibers in structure. Cardiac muscle fiber is bound by **sarcolemma.** It has a centrally placed nucleus. **Myofibrils** are embedded in the sarcoplasm. **Sarcomere** of the cardiac muscle has all the contractile proteins, namely actin, myosin, troponin and tropomyosin. **Sarcotubular system** in cardiac muscle is similar to that of skeletal muscle.

Important difference between skeletal muscle and cardiac muscle is that the cardiac muscle fiber is branched and the skeletal muscle is not branched.

Intercalated disk

Intercalated disk is a tough double membranous structure, situated at the junction between the branches of neighboring cardiac muscle fibers. It is formed by the fusion of the membrane of the cardiac muscle branches (Fig. 89.2).

Intercalated disks form **adherens junctions,** which play an important role in the contraction of cardiac muscle as a single unit (Chapter 2).

Syncytium

Syncytium means tissue with cytoplasmic continuity between adjacent cells. However, cardiac muscle is like a **physiological syncytium,** since there is no continuity of the cytoplasm and the muscle fibers are separated from each other by cell membrane. At the sides, the membranes of the adjacent muscle fibers fuse together to form **gap junctions.** Gap junction is permeable to ions and it facilitates the rapid conduction of action potential from one fiber to another. Because of this, all the cardiac muscle fibers act like a single unit, which is referred as syncytium.

Syncytium in human heart has two portions, syncytium of atria and the syncytium of ventricles. Both the portions of syncytium are connected by a thick non-conducting fibrous ring called the **atrioventricular ring.**

ii. *Muscle Fibers which Form the Pacemaker*

Some of the muscle fibers of heart are modified into a specialized structure known as pacemaker. These muscle fibers forming the pacemaker have less striation.

Pacemaker

Pacemaker is structure in the heart that generates the impulses for heart beat. It is formed by **pacemaker cells**

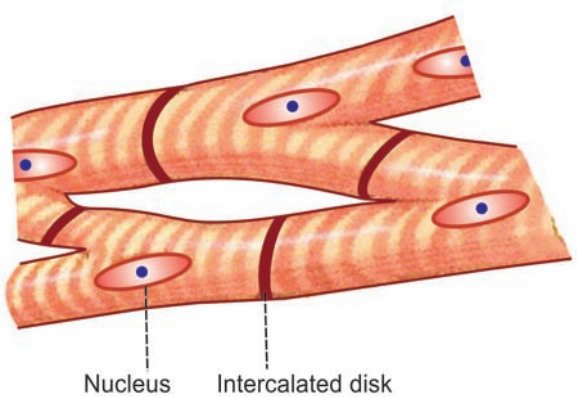

FIGURE 89.2: Cardiac muscle fibers

called **P cells.** Sinoatrial (SA) node forms the pacemaker in human heart. Details of pacemaker are given in next chapter.

iii. *Muscle Fibers which Form Conductive System*

Conductive system of the heart is formed by modified cardiac muscle fibers. Impulses from SA node are transmitted to the atria directly. However, the impulses are transmitted to ventricles through various components of conducting system, which are explained in the next chapter.

■ ENDOCARDIUM

Endocardium is the inner most layer of heart wall. It is a thin, smooth and glistening membrane. It is formed by a single layer of endothelial cells, lining the inner surface of the heart. Endocardium continues as endothelium of the blood vessels.

■ VALVES OF THE HEART

There are four valves in human heart. Two valves are in between atria and the ventricles called atrioventricular valves. Other two are the semilunar valves, placed at the opening of blood vessels arising from ventricles, namely systemic aorta and pulmonary artery. Valves of the heart permit the flow of blood through heart in only one direction.

Atrioventricular Valves

Left atrioventricular valve is otherwise known as **mitral valve** or **bicuspid valve.** It is formed by two valvular **cusps** or flaps (Fig. 89.3). Right atrioventricular valve is known as **tricuspid valve** and it is formed by three cusps.

Brim of the atrioventricular valves is attached to atrioventricular ring, which is the fibrous connection between the atria and ventricles. Cusps of the valves are attached to **papillary muscles** by means of **chordae tendineae.** Papillary muscles arise from inner surface of the ventricles. Papillary muscles play an important role in closure of the cusps and in preventing the back flow of blood from ventricle to atria during ventricular contraction.

Atrioventricular valves open only towards ventricles and prevent the backflow of blood into atria.

Semilunar Valves

Semilunar valves are present at the openings of systemic aorta and pulmonary artery and are known as **aortic valve** and **pulmonary valve** respectively. Because of the half moon shape, these two valves are called semilunar valves. Semilunar valves are made up of three flaps.

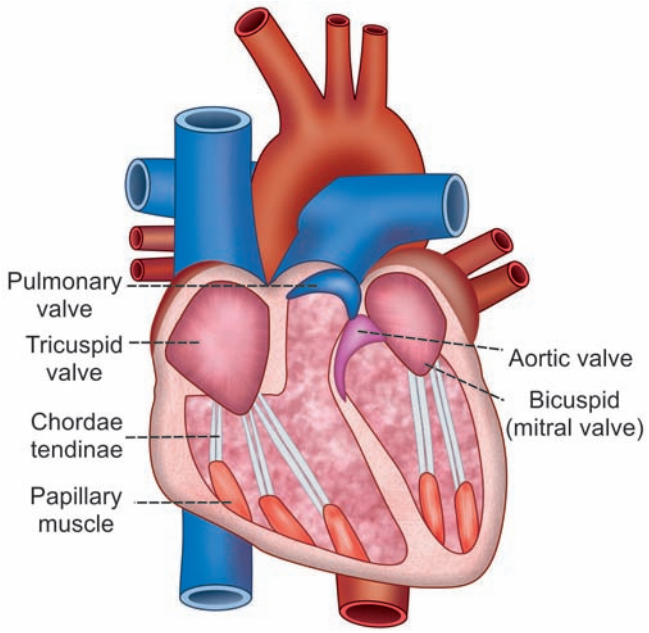

FIGURE 89.3: Valves of the heart

Semilular valves open only towards the aorta and pulmonary artery and prevent the backflow of blood into the ventricles.

■ ACTIONS OF THE HEART

Actions of the heart are classified into four types:

1. Chronotropic action
2. Inotropic action
3. Dromotropic action
4. Bathmotropic action.

■ CHRONOTROPIC ACTION

Chronotropic action is the frequency of heartbeat or heart rate. It is of two types:

i. **Tachycardia** or increase in heart rate
ii. **Bradycardia** or decrease in heart rate.

■ INOTROPIC ACTION

Force of contraction of heart is called inotropic action. It is of two types:

i. Positive inotropic action or increase in the force of contraction
ii. Negative inotropic action or decrease in the force of contraction.

■ DROMOTROPIC ACTION

Dromotropic action is the conduction of impulse through heart. It is of two types:

i. Positive dromotropic action or increase in the velocity of conduction
ii. Negative dromotropic action or decrease in the velocity of conduction.

■ BATHMOTROPIC ACTION

Bathmotropic action is the excitability of cardiac muscle. It is also of two types:

i. Positive bathmotropic action or increase in the excitability of cardiac muscle
ii. Negative bathmotropic action or decrease in the excitability of cardiac muscle.

Regulation of Actions of Heart

All the actions of heart are continuously regulated. It is essential for the heart to cope up with the needs of the body. All the actions are altered by stimulation of nerves supplying the heart or some hormones or hormonal substances secreted in the body.

■ BLOOD VESSELS

Vessels of circulatory system are the aorta, arteries, arterioles, capillaries, venules, veins and venae cavae. Structural differences between different blood vessels are given in Table 89.1.

■ ARTERIAL SYSTEM

Arterial system comprises the aorta, arteries and arterioles. Walls of the aorta and arteries are formed by three layers:

1. Outer **tunica adventitia,** which is made up of connective tissue layer. It is the continuation of fibrous layer of parietal pericardium.
2. Middle **tunica media,** which is formed by smooth muscles
3. Inner **tunica intima,** which is made up of endothelium. It is the continuation of endocardium.

Aorta, arteries and arterioles have two laminae of elastic tissues:

i. **External elastic lamina** between tunica adventitia and tunica media
ii. **Internal elastic lamina** between tunica media and tunica intima.

Aorta and arteries have more elastic tissues and the arterioles have more smooth muscles.

Arterial branches become narrower and their walls become thinner while reaching the periphery. Aorta has got the maximum diameter of about 25 mm. Diameter of the arteries is gradually decreased and at the end arteries, it is about 4 mm. It further decreases to 30 µ in the arterioles and ends up with 10 µ in the terminal arterioles. **Resistance** (peripheral resistance) is offered to blood flow in the arterioles and so these vessels are called **resistant vessels.**

Arterioles are continued as capillaries, which are small, thin walled vessels having a diameter of about 5 to 8 µ. Capillaries are functionally very important because, the exchange of materials between the blood and the tissues occurs through these vessels.

■ VENOUS SYSTEM

From the capillaries, venous system starts and it includes venules, veins and venae cavae. Capillaries end in venules, which are the smaller vessels with thin muscular wall than the arterioles. Diameter of the venules is about 20 µ. At a time, a large quantity of blood is held in venules and hence the venules are called **capacitance vessels.** Venules are continued as veins, which have the diameter of 5 mm. Veins form superior and inferior venae cavae, which have a diameter of about 30 mm.

TABLE 89.1: Structural and dimensional differences between different blood vessel walls

Blood vessel	Diameter	Thickness of the wall	Elastic tissue	Smooth muscle fibers	Fibrous tissue
Aorta	25 mm	2 mm	More	Less	More
Artery	4 mm	1 mm	More	More	Moderate
Arteriole	30 µ	6 µ	Moderate	More	Moderate
Terminal arteriole	10 µ	2 µ	Less	More	Moderate
Capillary	8 µ	0.5 µ	Absent	Absent	Moderate
Venule	20 µ	1 µ	Absent	Absent	Less
Vein	5 mm	0.5 mm	Less	More	Moderate
Vena cava	30 mm	1.5 mm	Less	More	More

Walls of the veins and venae cavae are made up of inner endothelium, elastic tissues, smooth muscles and outer connective tissue layer. In the veins and venae cavae, the elastic tissue is less but the smooth muscle fibers are more.

■ COMPLICATIONS IN BLOOD VESSELS

Aorta and Arteries

Arterial blood vessels are highly susceptible for arteriosclerosis and atherosclerosis. **Arteriosclerosis** is the disease of the arteries, associated with hardening, thickening and loss of elasticity in the wall of the vessels. **Atherosclerosis** is the disease marked by the narrowing of lumen of arterial vessel due to deposition of cholesterol.

Arterioles

When the tone of the smooth muscles in the arterioles increases, hypertension occurs.

Capillaries

Permeability of the capillary membrane may increase resulting in shock or edema due to leakage of fluid, proteins and other substances from blood.

Veins

Inflammation of the wall of veins leads to the formation of intravascular clot called **thrombosis.** The clot gets dislodged, as **thrombus.** The thrombus travels through blood and causes **embolism.** Embolism obstructs the blood flow to vital organs such as brain, heart and lungs, leading to many complications.

■ DIVISIONS OF CIRCULATION

Blood flows through two divisions of circulatory system:

1. Systemic circulation
2. Pulmonary circulation.

■ SYSTEMIC CIRCULATION

Systemic circulation is otherwise known as **greater circulation** (Fig. 89.4). Blood pumped from left ventricle passes through a series of blood vessels, arterial system

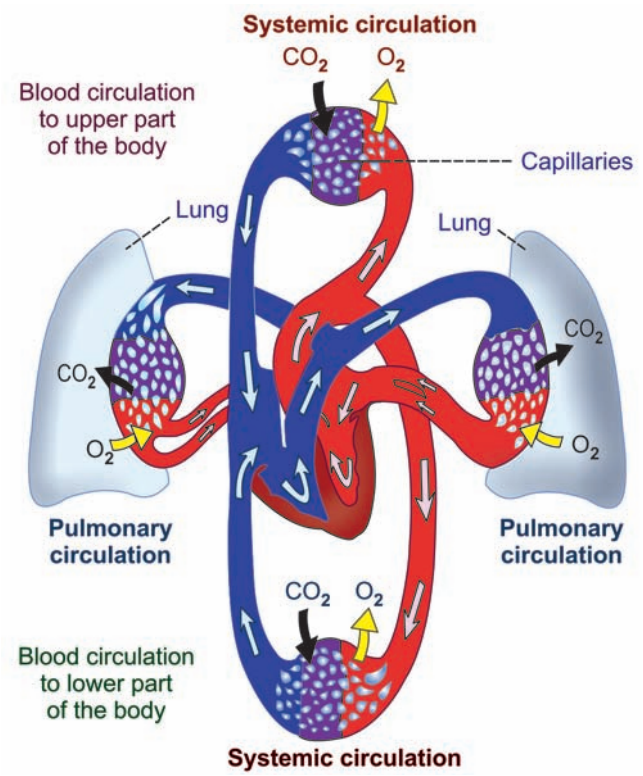

FIGURE 89.4: Systemic and pulmonary circulation

and reaches the tissues. Exchange of various substances between blood and the tissues occurs at the capillaries.

After exchange of materials, blood enters the venous system and returns to right atrium of the heart. From right atrium, blood enters the right ventricle.

Thus, through systemic circulation, oxygenated blood is supplied from heart to the tissues and venous blood returns to the heart from tissues.

■ PULMONARY CIRCULATION

Pulmonary circulation is otherwise called **lesser circulation.** Blood is pumped from right ventricle to lungs through pulmonary artery. Exchange of gases occurs between blood and alveoli of the lungs at pulmonary capillaries. Oxygenated blood returns to left atrium through the pulmonary veins.

Thus, left side of the heart contains oxygenated or arterial blood and the right side of the heart contains deoxygenated or venous blood.

Properties of Cardiac Muscle

Chapter 90

- EXCITABILITY
  - DEFINITION
  - ELECTRICAL POTENTIALS IN CARDIAC MUSCLE
  - IONIC BASIS OF ACTION POTENTIAL
  - SPREAD OF ACTION POTENTIAL THROUGH CARDIAC MUSCLE
- RHYTHMICITY
  - DEFINITION
  - PACEMAKER
  - ELECTRICAL POTENTIAL IN SINOATRIAL NODE
- CONDUCTIVITY
  - CONDUCTIVE SYSTEM IN HUMAN HEART
  - VELOCITY OF IMPULSES AT DIFFERENT PARTS OF CONDUCTIVE SYSTEM
- CONTRACTILITY
  - ALL-OR-NONE LAW
  - STAIRCASE PHENOMENON
  - SUMMATION OF SUBLIMINAL STIMULI
  - REFRACTORY PERIOD

EXCITABILITY

DEFINITION

Excitability is defined as the ability of a living tissue to give response to a stimulus. In all the tissues, initial response to a stimulus is electrical activity in the form of action potential. It is followed by mechanical activity in the form of contraction, secretion, etc.

ELECTRICAL POTENTIALS IN CARDIAC MUSCLE

Refer Chapter 31 for basics of electrical potentials in the muscle.

Resting Membrane Potential

Resting membrane potential in:

Single cardiac muscle fiber	:	– 85 to – 95 mV
Sinoatrial (SA) node	:	– 55 to – 60 mV
Purkinje fibers	:	– 90 to – 100 mV.

Action Potential

Action potential in cardiac muscle is different from that of other tissues such as skeletal muscle, smooth muscle and nervous tissue. Duration of the action potential in cardiac muscle is 250 to 350 msec (0.25 to 0.35 sec).

Phases of action potential

Action potential in a single cardiac muscle fiber occurs in four phases:

1. Initial depolarization
2. Initial repolarization
3. A plateau or final depolarization
4. Final repolarization.

1. *Initial Depolarization*

Initial depolarization is very rapid and it lasts for about 2 msec (0.002 sec). Amplitude of depolarization is about + 20 mV (Fig. 90.1).

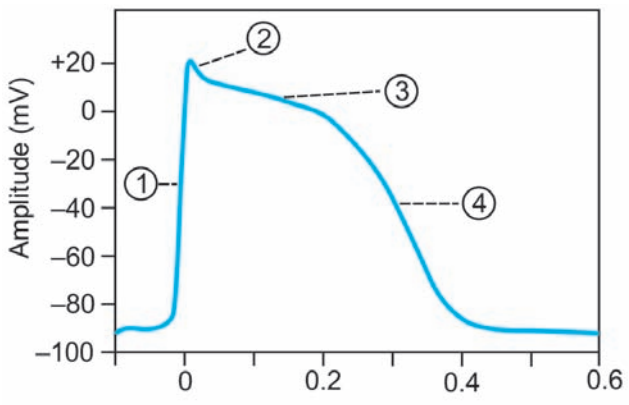

FIGURE 90.1: Action potential in ventricular muscle. 1 = Depolarization, 2 = Initial rapid repolarization, 3 = Plateau, 4 = Final repolarization.

2. *Initial Repolarization*

Immediately after depolarization, there is an initial rapid repolarization for a short period of about 2 msec. The end of rapid repolarization is represented by a notch.

3. *Plateau or Final Depolarization*

Afterwards, the muscle fiber remains in depolarized state for sometime before further repolarization. It forms the plateau (stable period) in action potential curve. The plateau lasts for about 200 msec in atrial muscle fibers and for about 300 msec in ventricular muscle fibers. Due to long plateau in action potential, the contraction time is also longer in cardiac muscle by 5 to 15 times than in skeletal muscle.

4. *Final Repolarization*

Final repolarization occurs after the plateau. It is a slow process and it lasts for about 50 to 80 msec before the re-establishment of resting membrane potential.

■ IONIC BASIS OF ACTION POTENTIAL

1. *Initial Depolarization*

Initial depolarization (first phase) is because of rapid opening of fast **sodium channels** and the **rapid influx of sodium ions,** as in the case of skeletal muscle fiber.

2. *Initial Repolarization*

Initial repolarization is due to the transient (short duration) opening of **potassium channels** and **efflux** of a small quantity of **potassium ions** from the muscle fiber. Simultaneously, the fast sodium channels close suddenly and slow sodium channels open, resulting in **slow influx** of low quantity of **sodium ions.**

3. *Plateau or Final Depolarization*

Plateau is due to the slow opening of **calcium channels.** These channels are kept open for a longer period and cause influx of large number of **calcium ions.** Already the slow sodium channels are opened, through which slow influx of sodium ions continues. Because of the entry of calcium and sodium ions into the muscle fiber, positivity is maintained inside the muscle fiber, producing prolonged depolarization, i.e. plateau. Calcium ions entering the muscle fiber play an important role in the contractile process.

4. *Final Repolarization*

Final repolarization is due to **efflux of potassium ions.** Number of potassium ions moving out of the muscle fiber exceeds the number of calcium ions moving in. It makes negativity inside, resulting in final repolarization. Potassium efflux continues until the end of repolarization.

Restoration of Resting Membrane Potential

At the end of final repolarization, all sodium ions, which had entered the cell throughout the process of action potential move out of the cell and potassium ions move into the cell, by activation of **sodium-potassium pump.** Simultaneously, excess of calcium ions, which had entered the muscle fiber also move out through sodium-calcium pump. Thus, the resting membrane potential is restored.

■ SPREAD OF ACTION POTENTIAL THROUGH CARDIAC MUSCLE

Action potential spreads through cardiac muscle very rapidly because of the presence of gap junctions between the cardiac muscle fibers. Gap junctions are permeable junctions and allow free movement of ions and so the action potential spreads rapidly from one muscle fiber to another fiber.

Action potential is transmitted from atria to ventricles through the fibers of specialized conductive system, which is explained later in this chapter.

■ RHYTHMICITY

■ DEFINITION

Rhythmicity is the ability of a tissue to produce its own impulses regularly. It is also called **autorhythmicity** or

self-excitation. Property of rhythmicity is present in all the tissues of heart. However, heart has a specialized excitatory structure, from which the discharge of impulses is rapid. This specialized structure is called pacemaker. From here, the impulses spread to other parts through the specialized conductive system.

■ PACEMAKER

Pacemaker is the structure of heart from which the impulses for heartbeat are produced. It is formed by the **pacemaker cells** called **P cells.** In mammalian heart, the pacemaker is sinoatrial node (SA node). It was Lewis Sir Thomas, who named SA node as pacemaker of heart, in 1918.

Sinoatrial Node

Sinoatrial (SA) node is a small strip of modified cardiac muscle, situated in the superior part of lateral wall of right atrium, just below the opening of superior vena cava. The fibers of this node do not have contractile elements. These fibers are continuous with fibers of atrial muscle, so that the impulses from the SA node spread rapidly through atria.

Other parts of heart such as atrioventricular (AV) node, atria and ventricle also can produce the impulses and function as pacemakers. Still, SA node is called the pacemaker because the rate of production of impulse (rhythmicity) is more in SA node than in other parts. It is about 70 to 80/minute.

Experimental Evidences

Experimental evidences to prove that SA node is the pacemaker in **mammalian heart:**

1. Stimulation of SA node accelerates the heart rate
2. Destruction of SA node causes immediate stoppage of the heartbeat. After sometime, atrioventricular node becomes the pacemaker and starts generating the impulses. So the heart starts beating, but the rate is slow.
3. Local cooling of SA node decreases the heart rate
4. Local warming of SA node increases the heart rate
5. Electrical activity starts first in SA node.

Spread of Impulses from SA Node

Mammalian heart has got a specialized conductive system, by which the impulses from SA node spreads to other parts of the heart (see below).

Rhythmicity of Different Parts of Human Heart

1. SA node : 70 to 80/minute
2. AV node : 40 to 60/minute
3. Atrial muscle : 40 to 60/minute
4. Purkinje fibers : 35 to 40/minute
5. Ventricular muscle : 20 to 40/minute.

Pacemaker in Amphibian Heart

Sinus venosus is the pacemaker in amphibian heart. It is experimentally proved by:

1. Applying Stannius ligatures
2. When sinus venosus is warmed by warm Ringer solution, heart rate increases
3. When sinus venosus is cooled by cold Ringer solution, heart rate decreases
4. Electrical activity starts first in sinus venosus.

Stannius ligature experiment

Stannius ligature experiment was demonstrated by German biologist **Stannius** in a **pithed frog.** Ligature means tying. **Pithing** is a process by which the brain and spinal cord are severed by using a needle, to abolish all the reflex activities during the experiment. Pithed frog is technically dead but some of its organs such as heart, continue to function for some time.

Chest wall of a pithed frog is opened and heart is exposed. A bent pin is fixed at the tip of ventricle and attached to a recording device by means of a thread. After recording the normal heartbeats (**normal cardiogram** or **sinus rhythm**), a ligature is applied between the sinus venosus and right auricle. It is called first Stannius ligature.

When this ligature is applied, the heart stops beating immediately. It is because the impulses produced by sinus venosus cannot be conducted to the other chambers of the heart. However, the sinus contractions are continued. After sometime, auricular muscle becomes the pacemaker and starts producing the impulses for heartbeat, but at a slower rate. Auricular contraction occurs first, followed by ventricular contraction. This rhythm of the heart is called **auriculoventricular rhythm** (Fig. 90.2).

When a second ligature is applied between auricles and ventricle, the heart stops beating again, because impulses from auricles cannot reach the ventricle. After few minutes, the ventricle produces its **own impulses** and starts beating but at a much slower rate. The slow independent ventricular rhythm is called **idioventricular rhythm.** Thus, all the three parts of the heart, sinus venosus, auricular musculature and ventricular musculature have the property of rhythmicity. However, sinus venosus is the pacemaker because it produces the impulses at a faster rate.

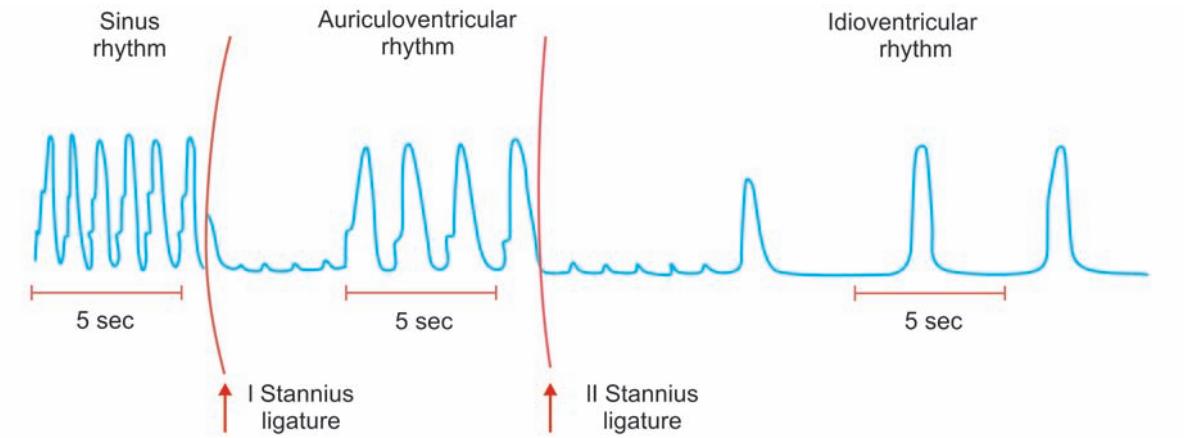

FIGURE 90.2: Effect of Stannius ligatures on frog's heart

Spread of Impulses from Sinus Venosus

Amphibian heart does not have any specialized conductive system. Pacemaker in amphibian heart is the sinus venosus and impulses from **sinus venosus** spreads through the muscles of auricles and ventricle.

Rhythmicity of Different Parts of Amphibian Heart

1. Sinus venosus : 40 to 60/minute
2. Auricular muscle : 20 to 40/minute
3. Ventricular muscle : 15 to 20/minute.

■ ELECTRICAL POTENTIAL IN SA NODE

Resting Membrane Potential – Pacemaker Potential

Pacemaker potential is the unstable resting membrane potential in SA node. It is also called prepotential.

Electrical potential in SA node is different from that of other cardiac muscle fibers. In SA node, each impulse triggers the next impulse. It is mainly due to the unstable resting membrane potential.

Resting membrane potential in SA node has a negativity of –55 to –60 mV. It is different from the negativity of –85 to –95 mV in other cardiac muscle fibers.

Action Potential

Depolarization starts very slowly and the threshold level of –40 mV is reached very slowly. After the threshold level, rapid depolarization occurs up to +5 mV. It is followed by rapid repolarization. Once again, the resting membrane potential becomes unstable and reaches the threshold level slowly (Fig. 90.3).

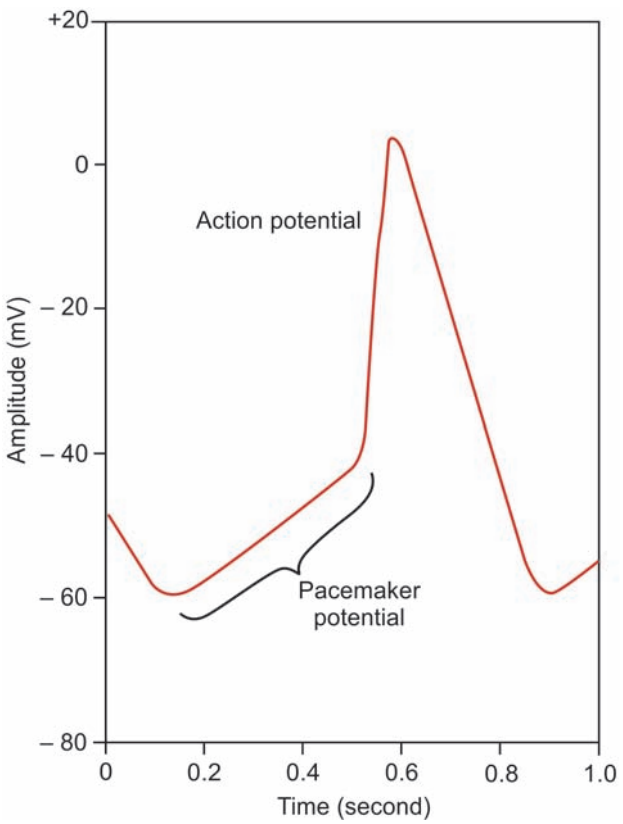

FIGURE 90.3: Pacemaker potential

Ionic Basis of Electrical Activity in Pacemaker

Pacemaker potential or resting membrane potential

Resting membrane potential is not stable in the SA node. To start with, the sodium ions leak into the pacemaker fibers and cause slow depolarization. This

slow depolarization forms the initial part of pacemaker potential. Then, the **calcium channels** start opening. At the beginning, there is a slow **influx of calcium ions** causing further depolarization in the same slower rate. It forms the later part of the pacemaker potential.

Thus, the initial part of pacemaker potential is due to slow **influx of sodium ions** and the later part is due to the slow influx of calcium ions.

Depolarization

When the negativity is decreased to –40 mV, which is the **threshold level,** the action potential starts with rapid depolarization. The depolarization occurs because of influx of more calcium ions. Unlike in other tissues, the depolarization in SA node is mainly due to the influx of calcium ions, rather than sodium ions.

Repolarization

After rapid depolarization, repolarization starts. It is due to the efflux of potassium ions from pacemaker fibers. Potassium channels remain open for a longer time, causing efflux of **more potassium ions.** It leads to the development of more negativity, beyond the level of resting membrane potential. It exists only for a short period. Then, the slow depolarization starts once again, leading to the development of **pacemaker potential,** which triggers the next action potential.

■ CONDUCTIVITY

Human heart has a specialized conductive system, through which impulses from SA node are transmitted to all other parts of the heart (Fig. 90.4).

■ CONDUCTIVE SYSTEM IN HUMAN HEART

Conductive system of the heart is formed by the modified cardiac muscle fibers. These fibers are the specialized cells, which conduct the impulses rapidly from SA node to the ventricles. Conductive tissues of the heart are also called the junctional tissues.

Components of Conductive System in Human Heart

1. AV node
2. Bundle of His
3. Right and left bundle branches
4. Purkinje fibers.

SA node is situated in right atrium, just below the opening of superior vena cava. AV node is situated in right posterior portion of intra-atrial septum. Impulses from SA node are conducted throughout right and left atria. Impulses also reach the AV node via some specialized fibers called internodal fibers.

There are three types of internodal fibers:

1. Anterior internodal fibers of Bachman
2. Middle internodal fibers of Wenckebach
3. Posterior internodal fibers of Thorel.

All these fibers from SA node converge on AV node and interdigitate with fibers of AV node. From AV node, the **bundle of His** arises. It divides into right and left branches, which run on either side of the interventricular septum. From each branch of bundle of His, many **Purkinje fibers** arise and spread all over the ventricular myocardium.

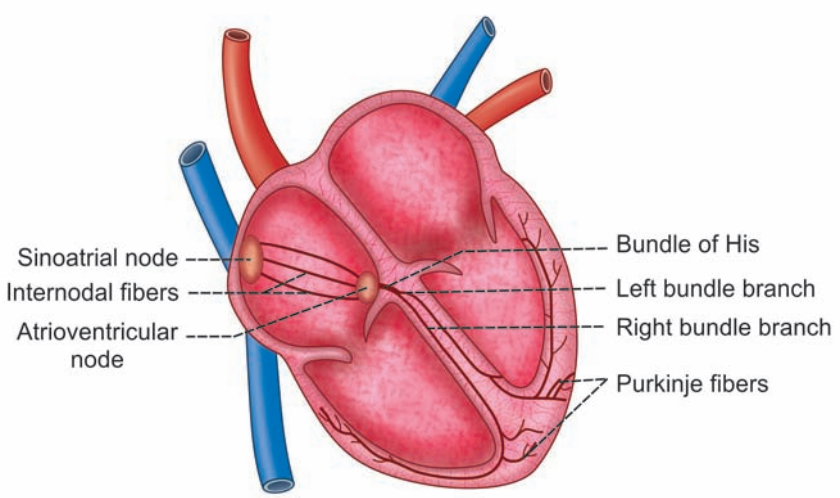

FIGURE 90.4: Sinoatrial node and conductive system of the heart

VELOCITY OF IMPULSES AT DIFFERENT PARTS OF CONDUCTIVE SYSTEM

1. Atrial muscle fibers : 0.3 meter/second
2. Internodal fibers : 1.0 meter/second
3. AV node : 0.05 meter/second
4. Bundle of His : 0.12 meter/second
5. Purkinje fibers : 4.0 meter/second
6. Ventricular muscle fibers : 0.5 meter/second.

Thus, the velocity of impulses is maximum in Purkinje fibers and minimum at AV node.

CONTRACTILITY

Contractility is ability of the tissue to shorten in length (contraction) after receiving a stimulus. Various factors affect the contractile properties of the cardiac muscle. Following are the contractile properties:

ALL-OR-NONE LAW

According to all-or-none law, when a stimulus is applied, whatever may be the strength, the whole cardiac muscle gives maximum response or it does not give any response at all. Below the threshold level, i.e. if the strength of stimulus is not adequate, the muscle does not give response.

All-or-none law is demonstrated in the **quiescent (quiet) heart** of frog. Heart is made quiescent by applying the first Stannius ligature in between the sinus venosus and right auricle.

Ventricle is stimulated by placing the electrode at the base of ventricle.

First, one stimulus is applied with a minimum strength of 1 volt at the base of ventricle and the contraction is recorded. Then, after 20 seconds, the strength of stimulus is increased to 2 volt and the stimulus is applied. The curve is recorded. The procedure is repeated by increasing the strength every time and applying the stimulus with an interval of 20 seconds (Fig. 90.5).

Amplitude of all contractions remains the same, irrespective of increasing the strength of stimulus. This shows that cardiac muscle obeys all-or-none law.

Cause for All-or-none law

All-or-none law is applicable to whole cardiac muscle. It is because of **syncytial** arrangement of cardiac muscle. In the case of skeletal muscle, all-or-none law is applicable only to a single muscle fiber.

STAIRCASE PHENOMENON

When the ventricle of a quiescent heart of frog is stimulated at a short interval of 2 seconds, without changing the strength, the force of contraction increases gradually for the first few contractions and then it remains same. Gradual increase in the force of contraction is called staircase phenomenon.

Cause for Staircase Phenomenon

Staircase phenomenon occurs because of **beneficial effect** (Chapter 30), which facilitates the force of successive contraction. So, there is a gradual increase in force of contraction (Fig. 90.5).

SUMMATION OF SUBLIMINAL STIMULI

When a stimulus with a subliminal strength is applied, the quiescent heart does not show any response. When few stimuli with same subliminal strength are applied in succession, the heart shows response by contraction, due to the summation of stimuli.

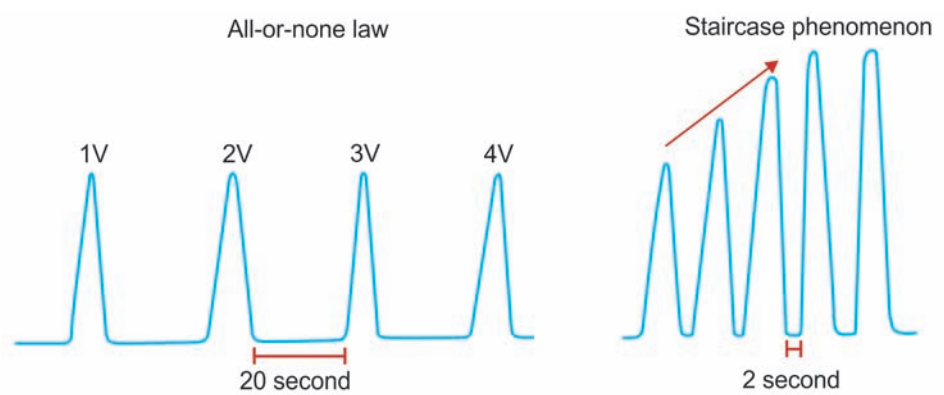

FIGURE 90.5: All-or-none law and staircase phenomenon in cardiac muscle

■ REFRACTORY PERIOD

Refractory period is the period in which the muscle does not show any response to a stimulus. It is of two types:

1. Absolute refractory period
2. Relative refractory period.

Absolute Refractory Period

Absolute refractory period is the period during which the muscle does not show any response at all, whatever may be the strength of the stimulus. It is because, the depolarization occurs during this period. So, a second depolarization is not possible.

Relative Refractory Period

Relative refractory period is the period during which the muscle shows response if the strength of stimulus is increased to maximum. It is the stage at which the muscle is in repolarizing state.

Refractory Period in Skeletal Muscle

In skeletal muscle, the refractory period is short. Absolute refractory period extends during the first half of latent period, measuring about 0.005 sec. Relative refractory period extends during the second half of latent period measuring 0.005 sec. So, the total refractory period is 0.01 sec.

Refractory Period in Cardiac Muscle

Cardiac muscle has a **long refractory period** compared to skeletal muscle. Absolute refractory period extends throughout the contraction period of cardiac muscle and its duration is 0.27 sec. Relative refractory period extends during first half of relaxation period, which is about 0.26 sec. So, the total refractory period is 0.53 sec.

Significance of Long Refractory Period in Cardiac Muscle

Long refractory period in cardiac muscle has three advantages:

1. Summation of contractions does not occur
2. Fatigue does not occur
3. Tetanus does not occur.

Demonstration of Refractory Period in Heart

Refractory period is demonstrated in the heart of a pithed frog. Refractory period can be recorded in beating heart as well as the quiescent heart.

Refractory period in beating heart

First, normal cardiogram is recorded with the heart of a pithed frog. The impulses for heartbeat arise from the sinus venosus. An electrical (external) stimulus is applied by keeping the electrode at the base of the ventricle. When the stimulus is applied during systole, the heart does not show any response. It is because the absolute refractory period extends throughout systole (Fig. 90.6).

When a stimulus is applied during diastole, the heart contracts because, diastole is the relative refractory period. This contraction of the heart is called **extrasystole** or **premature contraction.** Etrasystole is followed by the stoppage of heart in diastole for a while. This diastole is longer than the diastole after regular

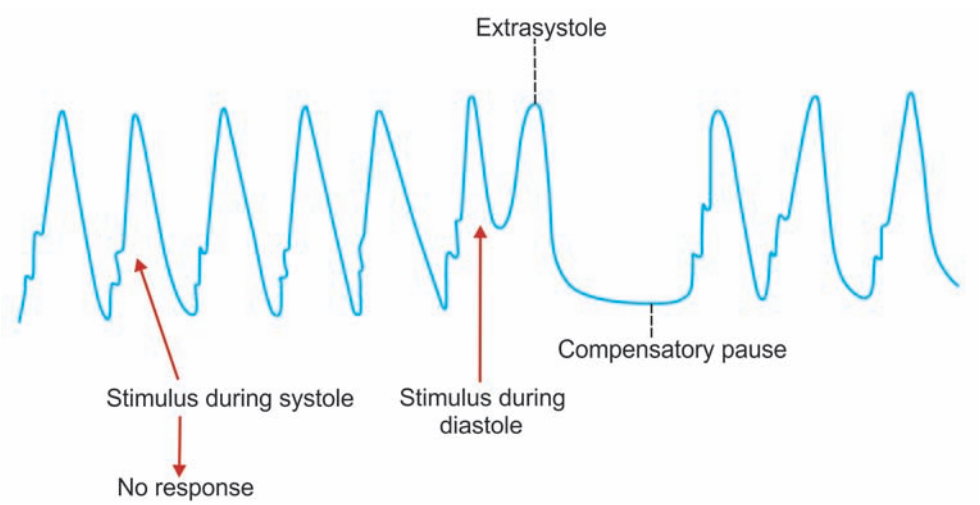

FIGURE 90.6: Refractory period in beating heart of frog

contraction. Temporary stoppage of the heart before it starts contracting is called **compensatory pause.** Duration of extrasystole and compensatory pause is equivalent to the duration of two cardiac cycles.

Cause for compensatory pause

A natural impulse from sinus venosus arrives at the time of contraction period of extrasystole. As this period is absolute refractory period, the natural impulse cannot cause contraction of heart. And the heart has to wait for the arrival of next natural impulse from sinus venosus. Till the arrival of next impulse, the heart stops in diastole.

Refractory period in quiescent heart

Frog's heart is made quiescent by applying the first Stannius ligature. Electrode is placed over the base of ventricle. When two stimuli are applied successively in such a way that the second stimulus falls during contraction period, the heart contracts only once. It is because of the first stimulus. There is no response to second stimulus because systole is the absolute refractory period. However, when a second stimulus is applied during diastole, the heart contracts again and second contraction superimposes over the first one. This shows that the relative refractory period extends during diastole (Fig. 90.7).

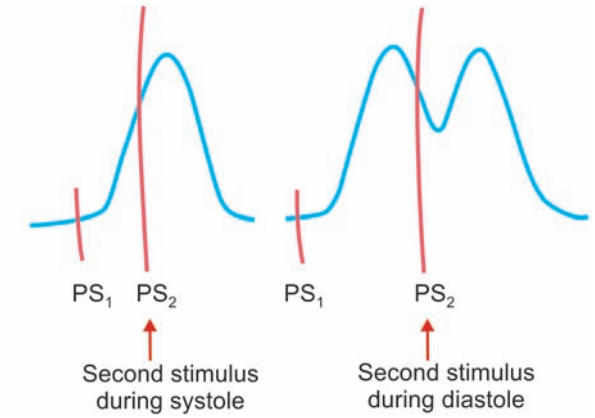

FIGURE 90.7: Refractory period in quiescent heart of a frog. PS_1 = Point of first stimulus, PS_2 = Point of second stimulus.

Cardiac Cycle

Chapter 91

- DEFINITION
- EVENTS
- DIVISIONS AND DURATION
  - ATRIAL EVENTS
  - VENTRICULAR EVENTS
- DESCRIPTION OF ATRIAL EVENTS
  - ATRIAL SYSTOLE
  - ATRIAL DIASTOLE
- DESCRIPTION OF VENTRICULAR EVENTS
  - ISOMETRIC CONTRACTION PERIOD
  - EJECTION PERIOD
  - PROTODIASTOLE
  - ISOMETRIC RELAXATION PERIOD
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  - VOLUME OF BLOOD IN RIGHT AND LEFT VENTRICLES
  - VENTRICULAR VOLUME CURVE

DEFINITION

Cardiac cycle is defined as the succession of (sequence of) **coordinated events** taking place in the heart during each beat. Each heartbeat consists of two major periods called systole and diastole. During systole, heart contracts and pumps the blood through arteries. During diastole, heart relaxes and blood is filled in the heart. All these changes are repeated during every heartbeat, in a cyclic manner.

EVENTS OF CARDIAC CYCLE

Events of cardiac cycle are classified into two:

1. Atrial events
2. Ventricular events.

DIVISIONS AND DURATION OF CARDIAC CYCLE

When the heart beats at a normal rate of 72/minute, duration of each cardiac cycle is about 0.8 second.

ATRIAL EVENTS

Atrial events are divided into two divisions:

1. Atrial systole = 0.11 (0.1) sec
2. Atrial diastole = 0.69 (0.7) sec.

VENTRICULAR EVENTS

Ventricular events are divided into two divisions:

1. Ventricular systole = 0.27 (0.3) sec
2. Ventricular diastole = 0.53 (0.5) sec.

In clinical practice, the term 'systole' refers to ventricular systole and 'diastole' refers to ventricular diastole. Ventricular systole is divided into two subdivisions and ventricular diastole is divided into five subdivisions.

Ventricular Systole

	Time (second)
1. Isometric contraction	= 0.05
2. Ejection period	= 0.22
	0.27

Ventricular Diastole

1. Protodiastole	= 0.04
2. Isometric relaxation	= 0.08
3. Rapid filling	= 0.11
4. Slow filling	= 0.19
5. Last rapid filling	= 0.11
	0.53

Among the atrial events, atrial systole occurs during the last phase of ventricular diastole. Atrial diastole is not considered as a separate phase, since it coincides with the whole of ventricular systole and earlier part of ventricular diastole.

DESCRIPTION OF ATRIAL EVENTS

ATRIAL SYSTOLE

Atrial systole is also known as **last rapid filling phase** or **presystole.** It is usually considered as the last phase of ventricular diastole. Its duration is 0.11 second.

During this period, only a small amount, i.e. 10% of blood is forced from atria into ventricles. Atrial systole is not essential for the maintenance of circulation. Many persons with atrial fibrillation survive for years, without suffering from circulatory insufficiency. However, such persons feel difficult to cope up with physical stress like exercise.

Pressure and Volume Changes

During atrial systole, the intra-atrial pressure increases. Intraventricular pressure and ventricular volume also increase but slightly.

Fourth Heart Sound

Contraction of atrial musculature causes the production of fourth heart sound.

ATRIAL DIASTOLE

After atrial systole, the atrial diastole starts. Simultaneously, ventricular systole also starts. Atrial diastole lasts for about 0.7 sec (accurate duration is 0.69 sec). This long atrial diastole is necessary because, this is the period during which atrial filling takes place. Right atrium receives deoxygenated blood from all over the body through superior and inferior venae cavae. Left atrium receives oxygenated blood from lungs through pulmonary veins.

Atrial Events Vs Ventricular Events

Out of 0.7 sec of atrial diastole, first 0.3 sec (0.27 sec accurately) coincides with ventricular systole. Then, ventricular diastole starts and it lasts for about 0.5 sec (0.53 sec accurately). Later part of atrial diastole coincides with ventricular diastole for about 0.4 sec. So, the heart relaxes as a whole for 0.4 sec. Figure 91.1 shows the correlation between atrial and ventricular events of cardiac cycle.

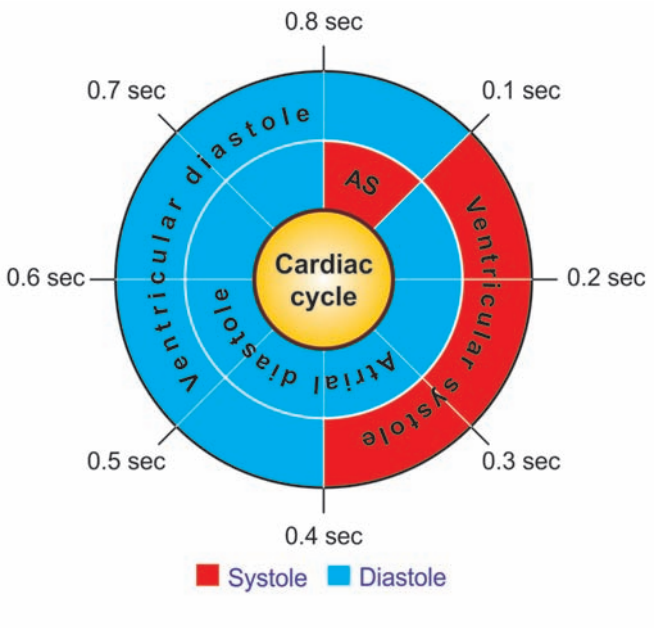

FIGURE 91.1: Atrial and ventricular events of cardiac cycle

■ DESCRIPTION OF VENTRICULAR EVENTS

■ ISOMETRIC CONTRACTION PERIOD

Isometric contraction period in cardiac cycle is the first phase of ventricular systole. It lasts for 0.05 second. Isometric contraction is the type of muscular contraction characterized by increase in tension, without any change in the length of muscle fibers. Isometric contraction of ventricular muscle is also called **isovolumetric contraction.**

Immediately after atrial systole, the atrioventricular valves are closed due to increase in ventricular pressure. Semilunar valves are already closed. Now, ventricles contract as closed cavities, in such a way that there is no change in the volume of ventricular chambers or in the length of muscle fibers. Only the tension increases in ventricular musculature.

Because of increased tension in ventricular musculature during isometric contraction, the pressure increases sharply inside the ventricles.

First Heart Sound

Closure of atrioventricular valves at the beginning of this phase produces first heart sound.

Significance of Isometric Contraction

During isometric contraction period, the ventricular pressure increases greatly. When this pressure increases above the pressure in the aorta and pulmonary artery, the semilunar valves open. Thus, the pressure rise in ventricle, caused by isometric contraction is responsible for the **opening of semilunar valves,** leading to ejection of blood from the ventricles into aorta and pulmonary artery.

■ EJECTION PERIOD

Due to the opening of semilunar valves and isotonic contraction of ventricles, blood is ejected out of both the ventricles. Hence, this period is called ejection period. Duration of this period is 0.22 second. Ejection period is of two stages:

1. *First Stage or Rapid Ejection Period*

First stage starts immediately after the opening of semilunar valves. During this stage, a large amount of blood is rapidly ejected from both the ventricles. It lasts for 0.13 second.

2. *Second Stage or Slow Ejection Period*

During this stage, the blood is ejected slowly with much less force. Duration of this period is 0.09 second.

End-systolic Volume

Ventricles are not emptied at the end of ejection period and some amount of blood remains in each ventricle. Amount of blood remaining in ventricles at the end of ejection period (i.e. at the end of systole) is called end-systolic volume. It is 60 to 80 mL per ventricle.

Measurement of end-diastolic volume

End-systolic volume is measured by **radionuclide angiocardiography** (multigated acquisition – **MUGA scan**) and **echocardiography.** It is also measured by cardiac **catheterization,** computed tomography **(CT) scan** and magnetic resonance imaging **(MRI)** (Chapter 109).

Ejection Fraction

Ejection fraction refers to the fraction (or portion) of end-diastolic volume (see below) that is ejected out by each ventricle per beat. From 130 to 150 mL of end-diastolic volume, 70 mL is ejected out by each ventricle (stroke volume). Normal ejection fraction is 60% to 65%.

Determination of ejection fraction

Ejection fraction (E_f) is the stroke volume divided by end-diastolic volume expressed in percentage. Stroke volume (SV) is, end-diastolic volume (EDV) minus end-systolic volume (ESV).

Ejection fraction is calculated as:

$$E_f = \frac{SV}{EDV} = \frac{EDV - ESV}{EDV}$$

Where,
E_f = Ejection fraction
SV = Stroke volume
EDV = End-diastolic volume
ESV = End-systolic volume.

Significance of determining ejection fraction

Ejection fraction is the measure of left ventricular function. Clinically, it is considered as an important index for assessing the ventricular contractility. Ejection fraction decreases in **myocardial infarction** and **cardiomyopathy.**

■ PROTODIASTOLE

Protodiastole is the first stage of ventricular diastole, hence the name protodiastole. Duration of this period is 0.04 second. Due to the ejection of blood, the pressure in aorta and pulmonary artery increases and pressure in ventricles drops.

When intraventricular pressure becomes less than the pressure in aorta and pulmonary artery, the semilunar valves close. Atrioventricular valves are already closed (see above). No other change occurs in the heart during this period. Thus, protodiastole indicates only the end of systole and beginning of diastole.

Second Heart Sound

Closure of semilunar valves during this phase produces second heart sound.

■ ISOMETRIC RELAXATION PERIOD

Isometric relaxation is the type of muscular relaxation, characterized by decrease in tension without any change in the length of muscle fibers. Isometric relaxation of ventricular muscle is also called **isovolumetric relaxation.**

During isometric relaxation period, once again all the valves of the heart are closed (Fig. 91.2). Now, both the ventricles relax as closed cavities without any change in volume or length of the muscle fiber. Intraventricular pressure decreases during this period. Duration of isometric relaxation period is 0.08 second.

Significance of Isometric Relaxation

During isometric relaxation period, the ventricular pressure decreases greatly. When the ventricular pressure becomes less than the pressure in the atria, the atrioventricular valves open. Thus, the fall in pressure in the ventricles, caused by isometric relaxation is responsible for the **opening of atrioventricular valves,** resulting in filling of ventricles.

■ RAPID FILLING PHASE

When atrionventricular valves are opened, there is a sudden rush of blood (which is accumulated in atria during atrial diastole) from atria into ventricles. So, this period is called the first rapid filling period. Ventricles also relax isotonically. About 70% of filling takes place during this phase, which lasts for 0.11 second.

Third Heart Sound

Rushing of blood into ventricles during this phase causes production of third heart sound.

■ SLOW FILLING PHASE

After the sudden rush of blood, the ventricular filling becomes slow. Now, it is called the slow filling. It is also called **diastasis.** About 20% of filling occurs in this phase. Duration of slow filling phase is 0.19 second.

■ LAST RAPID FILLING PHASE

Last rapid filling phase occurs because of atrial systole. After slow filling period, the atria contract and push a small amount of blood into ventricles. About 10% of ventricular filling takes place during this period. Flow of additional amount of blood into ventricle due to atrial systole is called **atrial kick.**

End-diastolic Volume

End-diastolic volume is the amount of blood remaining in each ventricle at the end of diastole. It is about 130 to 150 mL per ventricle.

Measurement of end-diastolic volume

End-diastolic volume is measured by the same methods, which are used to measure end-systolic volume (see above).

■ INTRA-ATRIAL PRESSURE CHANGES DURING CARDIAC CYCLE

■ SIGNIFICANCE

Pressure in the atria is called the intra-atrial pressure. Intra-atrial pressure is responsible for opening of the atrioventricular valves and ventricular filling. It is also the main factor for the development of venous pulse.

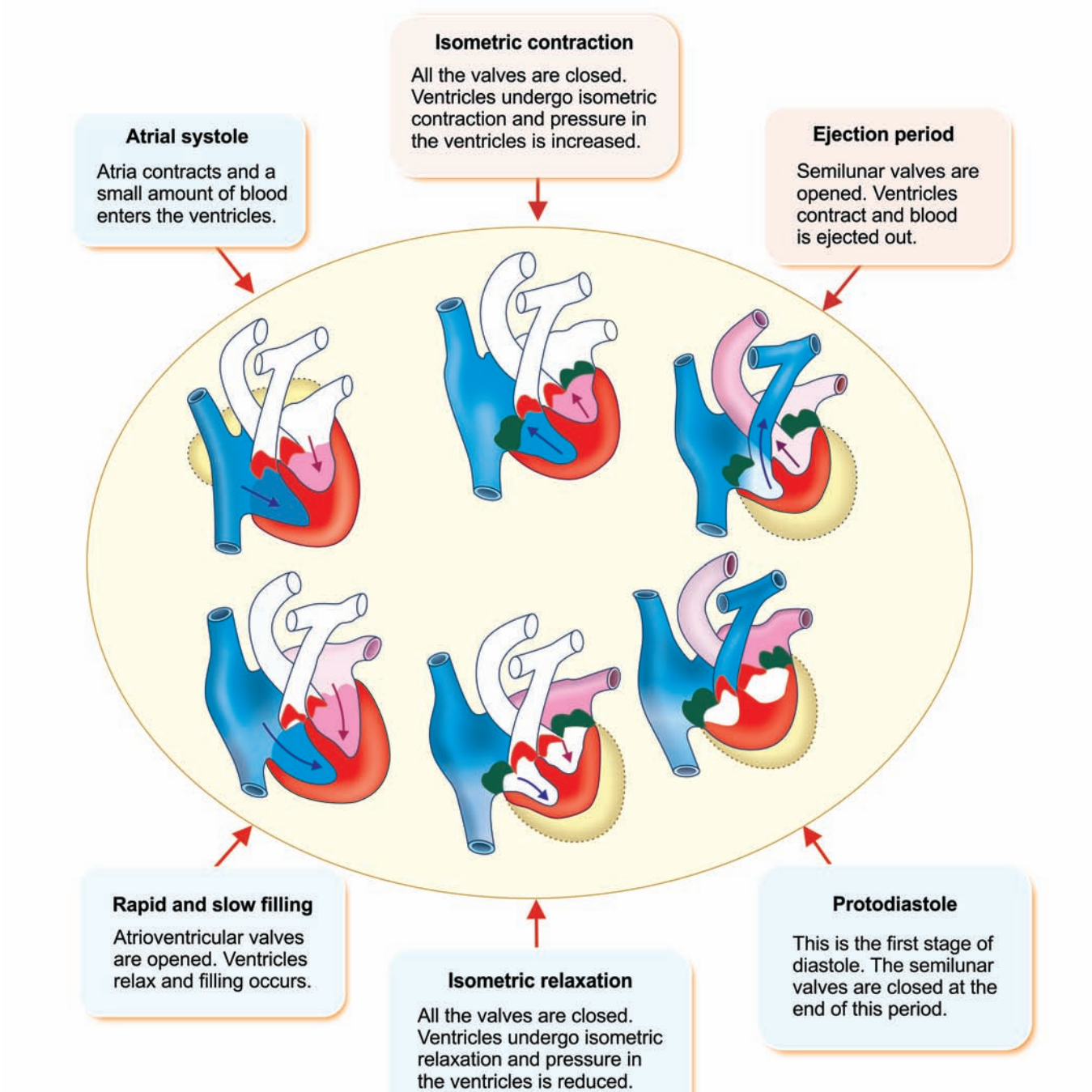

FIGURE 91.2: Events of cardiac cycle

■ METHODS OF STUDY

Right atrial pressure is recorded directly by cardiac **catheterization** (Chapter 98). Left atrial pressure is determined indirectly by measuring pulmonary capillary wedge pressure, which reflects the left atrial pressure accurately.

Pulmonary Capillary Wedge Pressure

Pulmonary capillary wedge pressure is the pressure exerted in the pulmonary capillary bed after obstructing the proximal part of pulmonary artery.

Pulmonary capillary wedge pressure is measured by using a **balloon-tipped multilumen cardiac catheter**

(Swan-Ganz catheter). Tip of the catheter is not open but a pressure transducer is attached to it.

By means of venous puncture, the catheter is guided through right atrium into right ventricle. From the right ventricle, it is advanced towards the proximal portion of pulmonary artery and the balloon is inflated with air by using a syringe. This occludes the pulmonary artery. Then, the catheter alone is advanced further into distal portion of pulmonary artery, leaving the inflated balloon at the proximal portion. It allows the catheter to float in a wedge position. Now the pressure existing in the pulmonary capillary bed ahead of catheter is called pulmonary capillary wedge pressure (the word wedge refers to being obstructed).

When the proximal part of pulmonary artery is obstructed, pressure in the distal part falls rapidly and after about 10 seconds, it becomes equal to left atrial pressure. It is because of the absence of any valve between pulmonary capillary bed and left atrium. So, the left atrial pressure can be determined by measuring pulmonary capillary wedge pressure.

■ MAXIMUM AND MINIMUM PRESSURE IN ATRIA

Maximum and minimum pressures in the left and right atria are given in Table 91.1.

TABLE 91.1: Pressure changes during cardiac cycle

Area	Maximum pressure	Minimum pressure
Left atrium	7 to 8 mm Hg	0 to 2 mm Hg
Right atrium	5 to 6 mm Hg	0 to 2 mm Hg
Left ventricle	120 mm Hg	5 mm Hg
Right ventricle	25 mm Hg	2 to 3 mm Hg
Systemic aorta	120 mm Hg	80 mm Hg
Pulmonary artery	25 mm Hg	7 to 8 mm Hg

■ INTRA-ATRIAL PRESSURE CURVE

Intra-atrial pressure curve is similar to the tracing of jugular venous pulse, which is known as **phlebogram.** It has three positive waves, a, c and v and three negative waves, x, x_1 and y (Fig. 91.3).

'a' Wave

'a' wave is the first positive wave and occurs during **atrial systole.** The pressure rises sharply up to 5 mm Hg in right atrium and 7 mm Hg in left atrium. After reaching the peak, the pressure starts decreasing.

'x' Wave

'x' wave is the first negative wave and appears during the onset of **atrial diastole.** Because of relaxation of atria, the pressure falls. Atrioventricular valves close at the end of this wave.

'c' Wave

'c' wave is the second positive wave and this appears during **isometric contraction.** Rise in pressure is due to

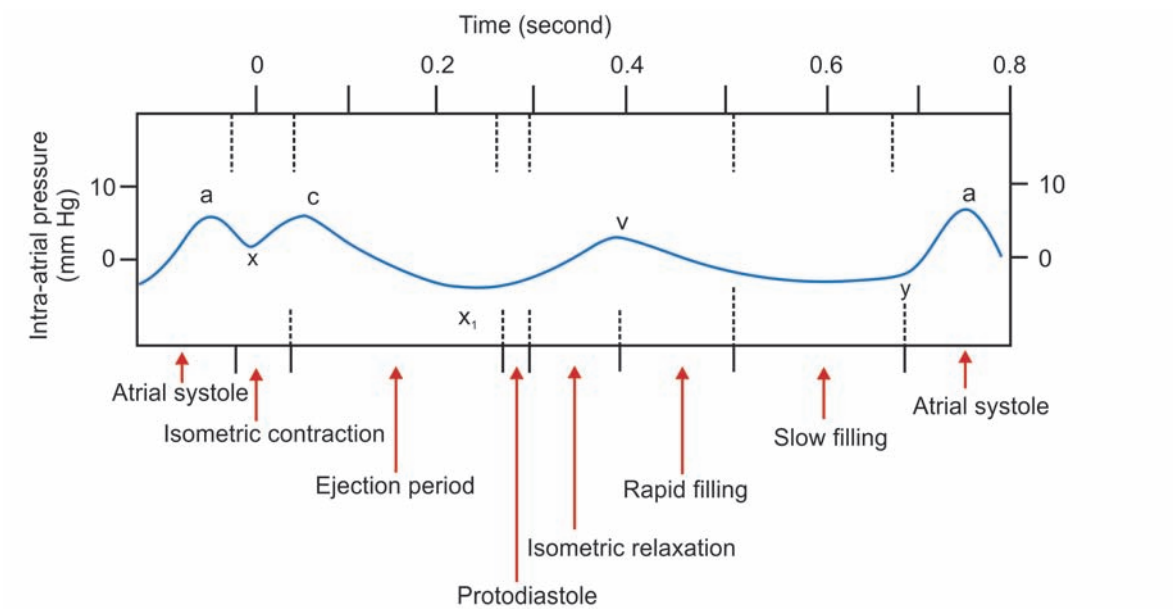

FIGURE 91.3: Intra-arterial pressure changes during cardiac cycle.
a, c, v = Positive waves. x, x_1, y = Negative waves.

the **closure of atrioventricular valves** and the increased intraventricular pressure.

When atrioventricular valves close, there is a little back flow of blood towards atria. When the intraventricular pressure increases, there is bulging of AV valves into the atria. Because of these two factors, the atrial pressure rises.

'x_1' Wave

'x_1' wave is the second negative wave and appears during **ejection period.** During ejection period, the contraction of ventricular musculature pulls the atrioventricular ring towards the ventricles. This causes fall in atrial pressure.

'v' Wave

'v' wave is the third positive wave, which is obtained during **atrial diastole.** It shows a gradual increase in atrial pressure due to filling of blood in atria (venous return).

'y' Wave

'y' wave is the third negative wave and appears after the **opening of AV valves** when the blood rushes from atria into ventricles. So, the pressure in the atria falls.

■ INTRAVENTRICULAR PRESSURE CHANGES DURING CARDIAC CYCLE

■ SIGNIFICANCE

Intraventricular pressure is the pressure developed inside the ventricles of the heart. It is essential for the circulation of blood, because the flow of blood through systemic and pulmonary circulation depends upon the pressure at which the blood is pumped out of ventricles. Thus, intraventricular pressure is essential for the circulation of blood.

■ METHODS OF STUDY

Intraventricular pressure is measured by cardiac catheterization.

■ MAXIMUM AND MINIMUM PRESSURE IN VENTRICLES

There is some difference in the pressure in right ventricle and left ventricle. The pressure is always more in left ventricle than in the right ventricle. Maximum and minimum pressures in the ventricles are given in Table 91.1.

■ INTRAVENTRICULAR PRESSURE CURVE

Intraventricular pressure curve has seven segments (Fig. 91.4).

'A-B' Segment

'A-B' segment is a positive wave and appears during **atrial systole.** Rise in pressure during this period is due to the entry of a small amount of blood into the ventricles because of atrial systole. The pressure rises to about 6 to 7 mm Hg in the right ventricle and to about 7 to 8 mm Hg in the left ventricle.

'B' indicates the **closure of atrioventricular valves.**

'B-C' Segment

'B-C' segment appears during **isometric contraction.** During isometric contraction period, there is a sharp rise in the intraventricular pressure.

'C' denotes the **opening of semilunar valves.**

'C-D' Segment

'C-D' segment appears during **ejection period.** During ejection period, the pressure in the ventricles rises to the peak and then falls down. First part of the curve indicates the maximum ejection and the pressure increases to the maximum. Second part of the curve represents the slow ejection phase when the pressure decreases.

Maximum pressure rise in right ventricle is about 25 mm Hg and the maximum pressure rise in left ventricle is about 120 mm Hg, during the peak of this wave. Maximum pressure in the left ventricle is 4 to 5 times more than that in the right ventricle, because of the thick wall of the left ventricle.

'D-E' Segment

'D-E' segment appears during **protodiastole.** Pressure decreases slightly due to the starting of ventricular relaxation.

'E' indicates the **closure of semilunar valves.**

'E-F' Segment

'E-F' segment is obtained during **isometric relaxation.** There is a sharp fall in the intraventricular pressure during this phase. Pressure in the ventricle falls below the pressure in the atria and this causes the opening of atrioventricular valves.

'F' represents the **opening of atrioventricular valves.**

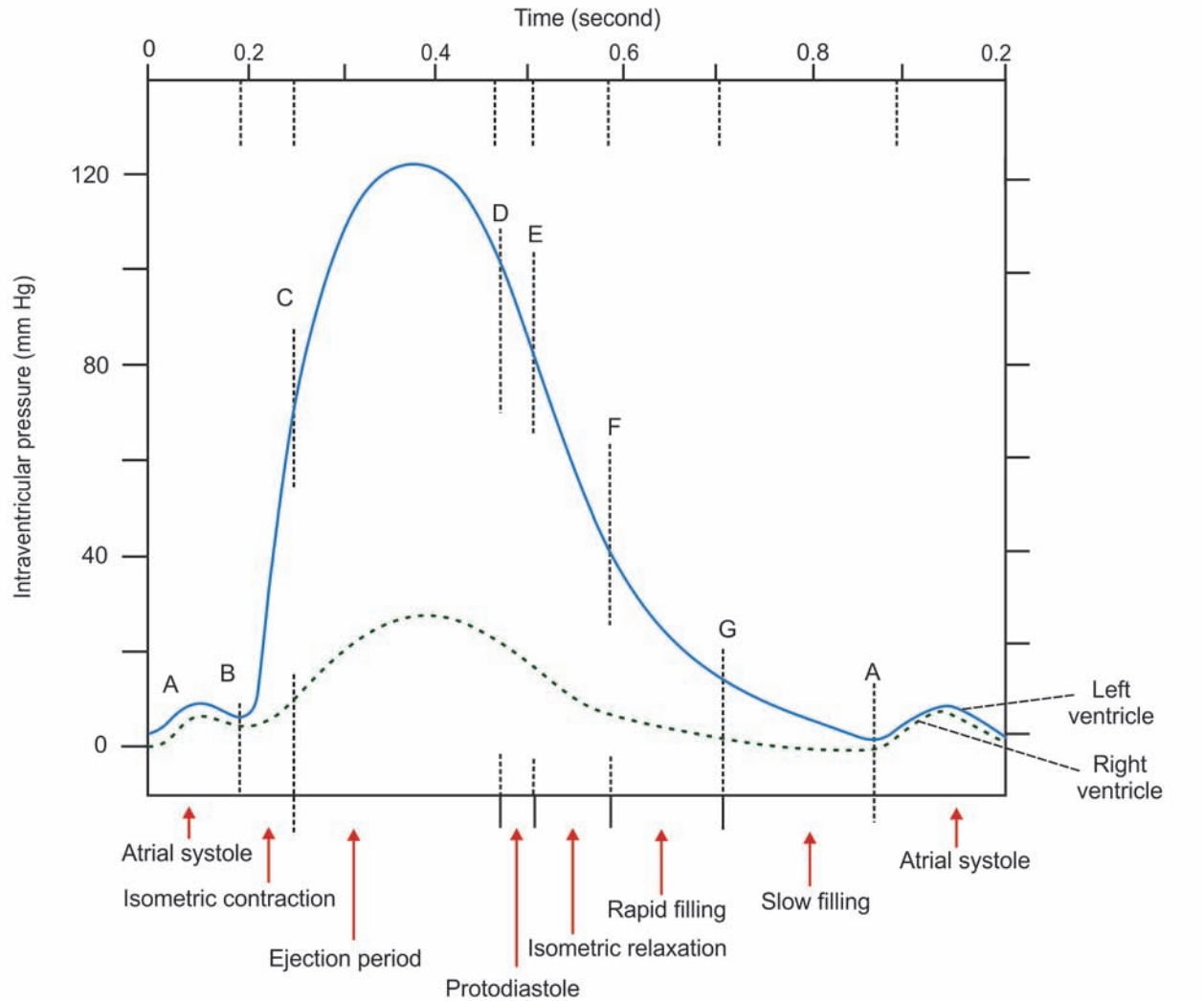

FIGURE 91.4: Intraventricular pressure changes during cardiac cycle

'F-G' Segment

'F-G' segment appears during **rapid filling phase.** In spite of filling of blood, pressure decreases in the ventricles. It is because of the relaxation of the ventricles.

'G-A' Segment

'G-A' segment is the last part of intraventricular pressure curve. It is obtained during **slow filling phase.** Because of continuous relaxation of ventricles during slow filling period, the ventricular pressure decreases further.

■ AORTIC PRESSURE CHANGES DURING CARDIAC CYCLE

■ SIGNIFICANCE

Aortic pressure is the pressure developed in the aorta. It is necessary to maintain the blood flow through the circulatory system.

■ METHOD OF STUDY

Changes in aortic pressure during the cardiac cycle are recorded by using **catheter.**

■ MAXIMUM AND MINIMUM PRESSURE IN AORTA

Pressure in systemic aorta is always higher than that of pulmonary artery. It is because of the higher pressure in left ventricle than in the right ventricle. Maximum and minimum pressures in aorta are given in Table 91.1. Minimum pressure in systemic aorta is much greater than the minimum pressure in the left ventricle. It is due to the presence of elastic tissues in the aorta, which enable the aorta to recoil and maintain the minimum pressure at a higher level.

■ AORTIC PRESSURE CURVE

During the ejection period of the cardiac cycle, the pressure in the aorta increases and reaches the peak.

During diastole, it reduces gradually and reaches the minimum level. At the time of closure of semilunar valves, an incisura occurs due to back flow of some blood towards the ventricles (Fig. 91.5).

VENTRICULAR VOLUME CHANGES DURING CARDIAC CYCLE

SIGNIFICANCE

Volume of blood in the ventricles is an important factor to maintain cardiac output and blood circulation.

METHODS OF STUDY

1. *By using Henderson Cardiometer*

This study is done only in animals. Cardiometer is a cup-shaped device with an outlet. At the top, it is closed by means of a rubber diaphragm. A small hole is made in the diaphragm, through which the ventricles of the animal are pushed. Cardiometer is connected to a recording device like **Marey tambour** (a small stainless steel capsule covered by rubber membrane) or **polygraph,** to record the volume changes (Fig. 99.1).

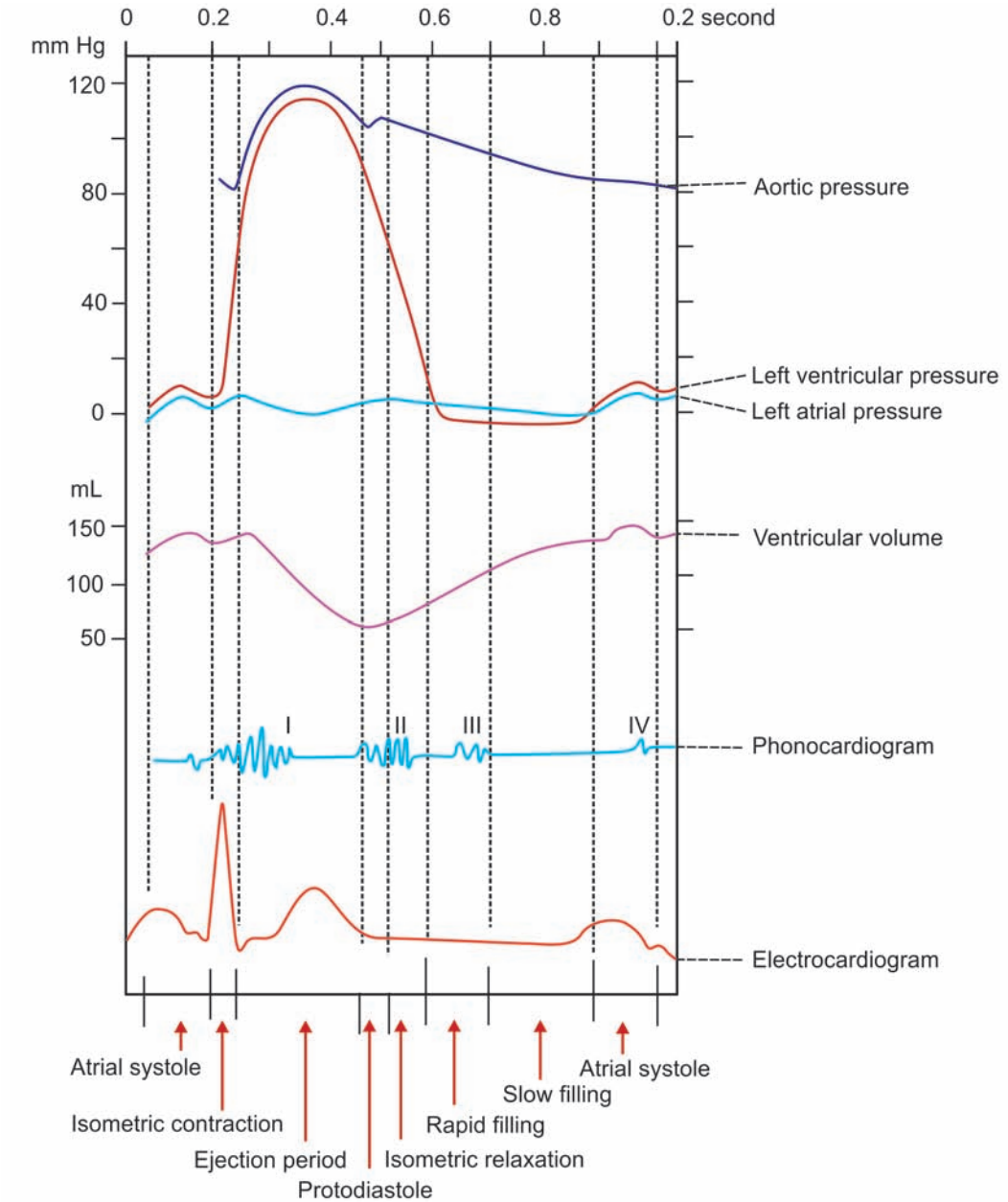

FIGURE 91.5: Comprehensive diagram showing ECG, phonocardiogram, pressure changes and volume changes during cardiac cycle

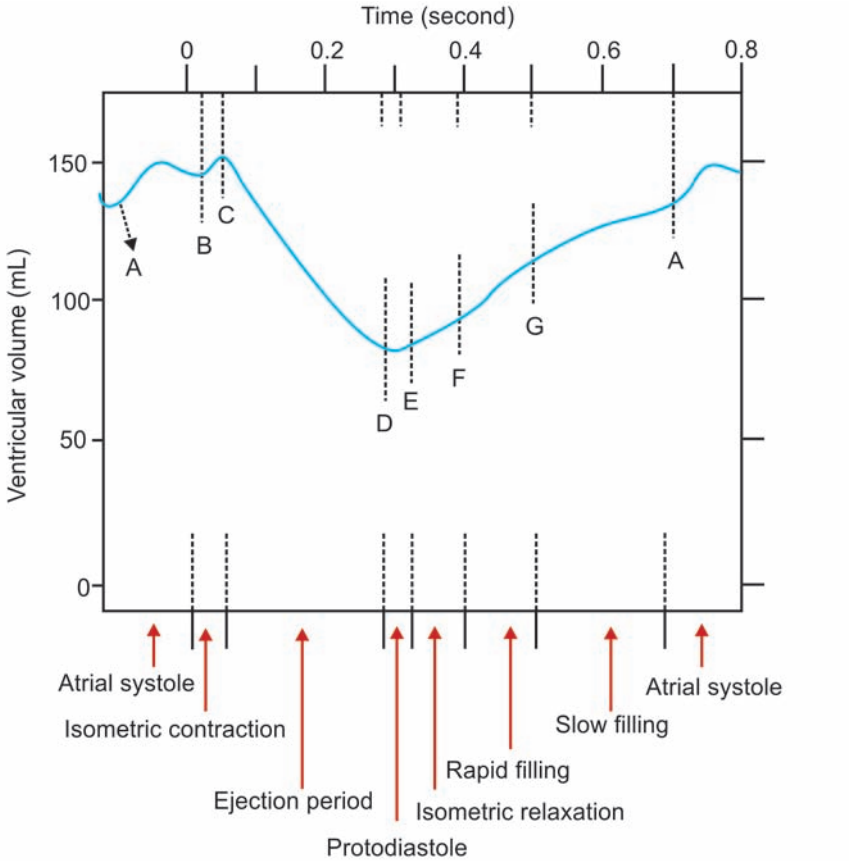

FIGURE 91.6: Ventricular volume changes during cardiac cycle

2. *By Angiography*

Angiography is the **radiographic study** of heart and blood vessels using a **radiopaque contrast** medium. During angiography, it is possible to measure the ventricular dimensional area and thickness of ventricular wall. From the values obtained, the ventricular volume is calculated.

■ VOLUME OF BLOOD IN RIGHT AND LEFT VENTRICLES

End-diastolic Volume and End-systolic Volume

Amount of blood is the same in both right and left ventricles. Maximum volume of blood in each ventricle after filling (end-diastolic volume) is 130 to 150 mL. Minimum volume of blood left in the ventricles at the end of ejection period (end of systolic volume) is 60 to 80 mL.

See above for measurement of end-diastolic volume and end-systolic volume.

Ejection Fraction

Ejection fraction (E_f) is the stroke volume divided by end-diastolic volume, expressed in percentage. See above for determination and significance of determining ejection period.

■ VENTRICULAR VOLUME CURVE

Ventricular volume curve recorded by using **Henderson cardiometer** has seven segments (Fig. 91.6).

'A-B' Segment

'A-B' segment wave is because of **atrial systole** or last filling phase of ventricles, during which a small amount of blood enters the ventricles from the atria. It increases the ventricular volume slightly.

'B' indicates the **closure of atrioventricular valves.**

'B-C' Segment

'B-C' segment is a positive wave, which is obtained during **isometric contraction.** Actually, the ventricular

volume is not altered during isometric contraction. However, the slight upward deflection of this wave is an artifact. It is because the heart thrusts itself into the cardiometer during isometric contraction.

'C' represents the **opening of semilunar valves.**

'C-D' Segment

'C-D' segment occurs during **ejection period.** Initially, there is a sharp fall in the ventricular volume. This occurs during rapid ejection. Later, during slow ejection period, the blood leaves the ventricles slowly. So the ventricular volume decreases slowly.

'D-E' Segment

'D-E' segment part of the ventricular volume curve is recorded during **protodiastole.** There is no change in the ventricular volume during protodiastole.

'E' denotes the closure of semilunar valves.

'E-F' Segment

'E-F' segment appears during **isometric relaxation period** of the cardiac cycle. Actually, the ventricular volume is not altered during isometric relaxation. However, there is a slight upward deflection in the curve due to artifact. It is because of the entrance of blood into coronary artery from aorta during this period. It increases the pressure within the cardiometer.

'F' indicates the **opening of atrioventricular valves.**

'F-G' Segment

'F-G' segment appears during **rapid filling phase.** Rapid rise in ventricular volume is due to sudden rush of blood after the opening of atrioventricular valves.

'G-A' Segment

'G-A' segment is recorded during **slow filling phase.** Ventricular volume increases slowly because of slow filling.

Heart Sounds

Chapter 92

- INTRODUCTION
  - DIFFERENT HEART SOUNDS
  - IMPORTANCE OF HEART SOUNDS
- DESCRIPTION OF DIFFERENT HEART SOUNDS
  - FIRST HEART SOUND
  - SECOND HEART SOUND
  - THIRD HEART SOUND
  - FOURTH HEART SOUND
- TRIPLE AND QUADRUPLE HEART SOUNDS
  - TRIPLE HEART SOUND OR GALLOP RHYTHM
  - QUADRUPLE HEART SOUND
- METHODS OF STUDY OF HEART SOUNDS
  - BY STETHOSCOPE
  - BY MICROPHONE
  - BY PHONOCARDIOGRAM

INTRODUCTION

Heart sounds are the sounds produced by mechanical activities of heart during each cardiac cycle.

Heart sounds are produced by:

1. Flow of blood through cardiac chambers
2. Contraction of cardiac muscle
3. Closure of valves of the heart.

Heart sounds are heard by placing the ear over the chest or by using a stethoscope or microphone. These sounds are also recorded graphically.

DIFFERENT HEART SOUNDS

Four heart sounds are produced during each cardiac cycle:

1. First heart sound
2. Second heart sound
3. Third heart sound
4. Fourth heart sound.

First and second heart sounds are called **classical heart sounds** and are heard by using the stethoscope. These two sounds are more prominent and resemble the spoken words 'LUB, (or LUBB) and 'DUBB' (or DUP), respectively.

Third heart sound is a mild sound and it is not heard by using stethoscope in normal conditions. But it can be heard by using a microphone. Fourth heart sound is an inaudible sound. It becomes audible in pathological conditions only. This sound is studied only by graphic registration, i.e. the phonocardiogram.

IMPORTANCE OF HEART SOUNDS

Study of heart sounds has important diagnostic value in clinical practice because alteration in the heart sounds indicates cardiac diseases involving valves of the heart.

DESCRIPTION OF HEART SOUNDS

FIRST HEART SOUND

First heart sound is produced during **isometric contraction** period and earlier part of **ejection period** (Table 92.1).

TABLE 92.1: Heart sounds

Features	First heart sound	Second heart sound	Third heart sound	Fourth heart sound
Occurs during	Isometric contraction period and part of ejection period	Protodiastole and part of isometric relaxation	Rapid filling phase	Atrial systole
Characteristics	Long, soft and low pitched Resembles the word 'LUBB'	Short, sharp and high pitched Resembles the word 'DUP'	Low pitched	Inaudible sound
Cause	Closure of atrioventricular valves	Closure of semilunar valves	Rushing of blood into ventricle	Contraction of atrial musculature
Duration (sec)	0.10 to 0.17	0.10 to 0.14	0.07 to 0.10	0.02 to 0.04
Frequency (cycles per sec)	25 to 45	50	1 to 6	1 to 4
Relation with ECG	Coincides with peak of 'R' wave	Precedes or appears 0.09 second after peak of 'T' wave	Between 'T' wave and 'P' wave	Between 'P' wave and 'Q' wave
Number of vibrations in phonocardiogram	9 to 13	4 to 6	1 to 4	1 to 2

Causes

Major cause for first heart sound is the sudden and synchronous (simultaneous) closure of **atrioventricular valves.** However, some other factors are also involved. Four types of factors are responsible for the production of the first heart sound.

1. *Valvular factor*

Synchronous closure of atrioventricular valves set up the vibrations in the **valvular leaflets** and **chordae tendineae.** These vibrations are mainly responsible for the production of the first heart sound.

2. *Vascular factor*

Rush of blood from the ventricles into aorta and pulmonary artery during ejection period is also responsible for the production of the first heart sound.

3. *Muscular factor*

Myocardial tension and the contraction of ventricular muscle during isometric contraction and the ejection periods also add to the production of the first heart sound.

4. *Atrial factor*

Vibrations produced by the atrial systole also play a role in the production of the first heart sound.

Characteristics

First heart sound is a long, soft and low-pitched sound. It resembles the spoken word **'LUBB'.** The duration of this sound is 0.10 to 0.17 second. Its frequency is 25 to 45 cycles/second.

Applied Physiology

1. *Reduplication of first heart sound*

Reduplication means splitting of the heart sound. First heart sound is split when the atrioventricular valves do not close simultaneously **(asynchronous closure).** Splitting of first heart sound in normal conditions **(physiological splitting)** is rare. **Pathological splitting** of first heart sound occurs in stenosis of atrioventricular valves and atrial septal defect.

2. *Soft first heart sound*

Heart sound becomes soft when the intensity of sound decreases. A soft first heart sound is heard in low blood pressure, severe heart failure, myocardial infarction and myxedema.

3. *Loud or accentuated first heart sound*

First heart sound becomes louder or accentuated (becoming prominent) in conditions like mitral stenosis, **Wolff-Parkinson-White syndrome** and acute rheumatic fever. It is loud in patients with thin chest wall also.

4. *Cannon sound*

Cannon sound refers to the loud first heart sound that is heard intermittently. It is heard in ventricular tachycardia and complete atrioventricular block.

First Heart Sound and ECG

First heart sound coincides with peak of 'R' wave in ECG.

■ SECOND HEART SOUND

Second heart sound is produced at the end of **protodiastolic period.**

Cause

Second heart sound is produced due to the sudden and synchronous closure of the **semilunar valves.**

Characteristics

Second heart sound is a short, sharp and high-pitched sound. It resembles the spoken word **'DUBB'** (or DUP). Duration of second heart sound is 0.10 to 0.14 second. Its frequency is 50 cycles/second.

Applied Physiology

1. Reduplication of second heart sound

Splitting of second heart sound occurs due to asynchronous closure of semilunar valves. It may occur both in physiological and pathological conditions.

Physiological splitting: It occurs during deep inspiration. Normally, aortic valve closes prior to the closure of pulmonary valve. Interval between the two valves widens during inspiration and narrows during expiration.

Increased negative intrathoracic pressure during deep inspiration increases lung expansion and venous return into right atrium. However, the venous return from lungs to left atrium is reduced during this condition. Because of increased venous return in right atrium and subsequent increase in blood volume in right ventricle, pulmonary valve is kept open for slightly longer time than the aortic valve. So, the pulmonary valve closes little later than the aortic valve causing splitting of second heart sound.

Pathological splitting: The splitting of second heart sound occurs during pulmonary stenosis, right bundle branch block and right ventricular hypertrophy.

Reverse splitting: It is the splitting of second heart sound, in which aortic valve closes after the closure of pulmonary valve. It is due to the delay in emptying of left ventricle. It is also called **paradoxical splitting** (paradoxical = contradictory or opposite). Reverse splitting is common in left bundle-branch block, aortic stenosis and left ventricular hypertrophy.

2. *Loud or accentuated second heart sound*

Loud or accentuated second heart sound is produced by the closure of either aortic valve or pulmonary valve. Aortic valve produces loud sound during systemic hypertension and **coarctation** (narrowing) of aorta. Pulmonary valve produces loud sound during pulmonary hypertension.

3. *Soft second heart sound*

Second heart sound becomes soft in heart failure.

Second Heart Sound and ECG

Second heart sound coincides with the 'T' wave in ECG. Sometimes, it may precede the 'T' wave or it may commence after the peak of 'T' wave.

■ THIRD HEART SOUND

Third heart sound is a low-pitched sound that is produced during **rapid filling period** of the cardiac cycle. It is also called ventricular gallop or protodiastolic gallop, as it is produced during earlier part of diastole.

Usually, the third heart sound is **inaudible** by stethoscope and it can be heard only by using microphone.

Causes

Third heart sound is produced by the **rushing of blood** into ventricles and vibrations set up in the ventricular wall during rapid filling phase. It may also be due to vibrations set up in chordae tendineae.

Characteristics

Third heart sound is a short and low-pitched sound. Duration of this sound is 0.07 to 0.10 second. Its frequency is 1 to 6 cycles/second.

Conditions when Third Heart Sound becomes Audible by Stethoscope

Third heart sound can be heard by stethoscope in children and athletes. Pathological conditions when third heart sound becomes loud and audible by stethoscope are aortic regurgitation, cardiac failure and cardiomyopathy with dilated ventricles.

When third heart sound is heard by stethoscope, the condition is called **triple heart sound** (see below). Third heart sound is usually heard best with the bell of stethoscope placed at the apex beat area, when the patient is in left **lateral decubitus** (lying on left side) position.

Third Heart Sound and ECG

Third heart sound appears between 'T' and 'P' waves of ECG.

■ FOURTH HEART SOUND

Normally, the fourth heart sound is an **inaudible** sound. It becomes audible only in pathological conditions. It is studied only by graphical recording, i.e. by phonocardiography. This sound is produced during **atrial systole** (late diastole) and it is considered as the physiologic atrial sound. It is also called **atrial gallop** or **presystolic gallop.**

Causes

Fourth heart sound is produced by contraction of **atrial musculature** and vibrations are set up in atrial musculature, flaps of the atrioventricular valves during systole. It is also due to the vibrations set up in the ventricular myocardium because of ventricular distention during atrial systole.

Characteristics

Fourth heart sound is a short and low-pitched sound. Duration of this sound is 0.02 to 0.04 second. Its frequency is 1 to 4 cycles/second.

Conditions when Fourth Heart Sound becomes Audible

Fourth heart sound becomes audible by stethoscope when the ventricles become stiff. Ventricular stiffness occurs in conditions like ventricular hypertrophy, long standing hypertension and aortic stenosis. To overcome the ventricular stiffness, the atria contract forcefully, producing audible fourth heart sound.

When fourth heart sound is heard by stethoscope, the condition is called **triple heart sound** (see below). It is usually heard best with the bell of stethoscope placed at the apex beat area, when the patient is in supine or left semilateral position.

Fourth Heart Sound and ECG

Fourth heart sound coincides with the interval between the end of 'P' wave and the onset of 'Q' wave.

■ TRIPLE AND QUADRUPLE HEART SOUNDS

■ TRIPLE HEART SOUND OR GALLOP RHYTHM

Triple heart sound or **triple rhythm** is an **abnormal rhythm** of heart, characterized by three clear heart sounds during each heart beat. It is due to an abnormal third or fourth heart sound that is heard besides first and second heart sounds. It is also called **gallop rhythm,** since it resembles the sound of a horse's gallop. Usually, it is indicative of serious cardiovascular disease.

Conditions when Triple Heart Sound is Produced

Triple heart sound is produced in conditions like myocardial infarction and severe hypertension.

■ QUADRUPLE HEART SOUND

Quadruple heart sound is an abnormal rhythm of heart, characterized by four clear heart sounds during each heart beat. It is also called **quadruple rhythm.** It is due to third and fourth heart sounds that are heard besides first and second heart sounds. It is also called **quadruple gallop.**

Quadruple heart sound is also indicative of serious cardiovascular disease.

Conditions when Quadruple Heart Sound is Produced

Quadruple heart sound is produced in patients with congestive heart failure.

Summation Gallop

Whenever there is tachycardia in patients with quadruple heart sound, the third and fourth heart sounds merge together and give rise to a single sound. This sound is called **summation gallop** and it resembles gallop rhythm.

■ METHODS OF STUDY OF HEART SOUNDS

Heart sounds are studied by three methods:

1. By using stethoscope
2. By using microphone
3. By using phonocardiogram.

■ BY STETHOSCOPE

First and second heart sounds are heard on the auscultation areas, by using the stethoscope. The chest piece of the stethoscope is placed over four areas on the chest, which are called auscultation areas.

Auscultation Areas

i. Mitral area (Bicuspid area)

Mitral area is in the left 5th intercostal space, about 10 cm away from the midline (midclavicular line). Sound produced by the closure of mitral valve (first heart

sound) is transmitted well into this area. It is also called **apex beat area** because apex beat is felt in this area.

Apex beat

Apex beat is the thrust of the apex of ventricles, against the chest wall during systole.

ii. *Tricuspid area*

Tricuspid area is on the xiphoid process. Sound produced by the closure of tricuspid valve (first heart sound) is transmitted well into this area.

iii. *Pulmonary area*

Pulmonary area is on the left 2nd intercostal space, close to sternum. Sound produced by the closure of pulmonary valve (second heart sound) is heard well on this area.

iv. *Aortic area*

Aortic area is over the right 2nd intercostal space, close to the sternum. On this area, the sound produced by the closure of aortic valve (second heart sound) is heard well.

First heart sound is best heard in mitral and tricuspid areas. However, it is heard in other areas also but the intensity is less. Similarly, the second heart sound is best heard in pulmonary and aortic areas. It is also heard in other areas with less intensity.

■ BY MICROPHONE

A highly sensitive microphone is placed over the chest. The heart sounds are amplified by means of an **amplifier** and heard by using a **loudspeaker.** First, second and third heart sounds are heard by this method.

■ BY PHONOCARDIOGRAM

Phonocardiography is the technique used to record the heart sounds. Phonocardiogram is the graphical record of heart sounds. It is done by placing an electronic **sound transducer** over the chest. This transducer is connected to a recording device like polygraph. All the four heart sounds can be recorded in phonocardiogram. It helps to analyze the frequency of the sound waves.

Appearance of Heart Sounds in Phonocardiogram

In phonocardiogram, the heart sounds are recorded in the following manner (Fig. 91.6).

First heart sound

First heart sound is recorded as single group of waves. The waves are of small amplitude to start with. Later, the amplitude rapidly rises and falls to form **crescendo** and **diminuendo** series of waves. About 9 to 13 waves appear.

Second heart sound

Second heart sound appears as single group of waves, which have same amplitude. About 4 to 6 waves are recorded.

Third heart sound

Third heart sound is found in phonocardiogram with only 1 to 4 waves grouped together.

Fourth heart sound

Mostly, the fourth heart sound merges with first heart sound. If it appears as separate form, it has 1 to 2 waves with very low amplitude.

Cardiac Murmur

Chapter 93

- INTRODUCTION
  - CAUSES OF MURMUR
- CLASSIFICATION OF MURMUR
  - SYSTOLIC MURMUR
  - DIASTOLIC MURMUR
  - CONTINUOUS MURMUR

INTRODUCTION

Cardiac murmur is the abnormal or unusual heart sound. It is also called **abnormal heart sound** or **cardiac bruit.** Cardiac murmur is heard by stethoscope, along with normal heart sounds.

Cardiac murmur is heard by placing chest piece of stethoscope over the auscultatory areas. Murmur due to disease of a particular valve is heard well over the auscultatory area of that valve. Sometimes, the murmur is felt by palpation as **'thrills'.** In some patients, murmur is heard without any aid, even at a distance of few feet away from the patient.

CAUSES OF MURMUR

Cardiac murmur is produced because of change in the pattern of blood flow. Normally, blood flows in **streamline** through the heart and blood vessels. However, during abnormal conditions like valvular diseases, the blood flow becomes **turbulent.** It produces the cardiac murmur.

Murmur is produced because of valvular diseases, septal defects and vascular defects (Table 93.1).

Valvular Diseases

Valvular diseases are of two types:

1. Stenosis
2. Incompetence.

1. *Stenosis*

Stenosis means narrowing of heart valve. Blood flows rapidly with turbulence through the narrow orifice of the valve, resulting in murmur.

TABLE 93.1: Causes for cardiac murmur

Type of murmur	Causes
Systolic murmur	1. Incompetence of atrioventricular valves 2. Stenosis of semilunar valves 3. Anemia 4. Septal defect 5. Coarctation of aorta
Diastolic murmur	1. Stenosis of atrioventricular valves 2. Incompetence of semilunar valves
Continuous murmur	1. Patent ductus arteriosus

2. *Incompetence*

Incompetence refers to weakening of the heart valve. When the valve becomes weak, it cannot close properly. It causes back flow of blood, resulting in **turbulence.** This disease is also called **regurgitation** or **valvular insufficiency.**

■ CLASSIFICATION OF MURMUR

Cardiac murmur is classified into three types:

A. Systolic murmur
B. Diastolic murmur
C. Continuous murmur.

■ SYSTOLIC MURMUR

Systolic murmur is the murmur which is produced during systole. It is produced in the following conditions:

1. *Incompetence of Atrioventricular Valves*

When the atrioventricular valves become weak, these valves cannot close completely. It causes **regurgitation** of blood from ventricles to the atria during ventricular systole, producing the murmur. It is a **harsh blowing sound** with high frequency.

2. *Stenosis of Semilunar Valves*

During stenosis of aortic valve, the left ventricular pressure raises up to 300 mm Hg during systole. It causes a greater turbulence in the blood flow. The vibrations of this sound can be felt as **'thrills'** by palpation over lower neck region and upper chest. In severe conditions, the sound is heard even a few feet away from the affected person. It is a **harsh** and a **loud sound.**

3. *Murmur due to Anemia*

A systolic murmur is heard in severe anemia because of reduced viscosity and accelerated flow of blood.

4. *Septal Defect*

During interventricular septal defect, blood flows from left ventricle to right ventricle during systole. It produces a systolic murmur. Septal defect is a rare disorder.

5. *Coarctation of Aorta*

Coarctation of aorta is a congenital disorder, characterized by the narrowing of a part of systemic aorta. A loud murmur is produced during systole and it is heard in the earlier part of diastole also.

■ DIASTOLIC MURMUR

Diastolic murmur is the murmur that is produced during diastole. It is produced in the following conditions:

1. *Stenosis of Atrioventricular Valves*

When the atrioventricular valves become narrow, the turbulence of blood flow occurs during diastole, i.e. when blood enters the ventricles from atria. Murmur due to stenosis of mitral valve is heard better at mitral area. Murmur due to stenosis of tricuspid valve is heard best at tricuspid area. It is a **weak sound** with low frequency.

Sometimes, murmur due to mitral stenosis cannot be heard by stethoscope, due to low frequency. But it can be felt as a mild thrill over mitral area of the chest.

2. *Incompetence of Semilunar Valves*

Murmur is produced during the regurgitation of blood from aorta into the ventricle, through incompetent semilunar valve during diastole. It is like a **blowing sound** with low frequency.

■ CONTINUOUS MURMUR

Continuous murmur is the murmur that is heard in conditions such as patent ductus arteriosus.

Patent Ductus Arteriosus

Intact ductus arteriosus is called patent ductus arteriosus (Chapter 114). A continuous murmur is heard in this condition. However, intensity of the sound is more during systole and less during diastole. Because of this, it is also called **machinery murmur.**

It is a **harsh blowing sound** and is heard best in the pulmonary area. The murmur is heard 1 year after birth.

Electrocardiogram (ECG)

Chapter 94

- DEFINITIONS
- USES OF ECG
- ELECTROCARDIOGRAPHIC GRID
  - DURATION
  - AMPLITUDE
  - SPEED OF THE PAPER
- ECG LEADS
  - BIPOLAR LEADS
  - UNIPOLAR LEADS
- WAVES OF NORMAL ECG
  - 'P' WAVE
  - 'QRS' COMPLEX
  - 'T' WAVE
  - 'U' WAVE
- INTERVALS AND SEGMENTS OF ECG
  - 'P-R' INTERVAL
  - 'Q-T' INTERVAL
  - 'S-T' SEGMENT
  - 'R-R' INTERVAL

DEFINITIONS

Electrocardiography

Electrocardiography is the **technique** by which electrical activities of the heart are studied. The spread of excitation through myocardium produces local electrical potential. This low-intensity current flows through the body, which acts as a **volume conductor.** This current can be picked up from surface of the body by using suitable electrodes and recorded in the form of electrocardiogram. This technique was discovered by Dutch physiologist, **Einthoven Willem,** who is considered the father of electrocardiogram (ECG).

Electrocardiograph

Electrocardiograph is the **instrument** (machine) by which electrical activities of the heart are recorded.

Electrocardiogram

Electrocardiogram (ECG or EKG from electrokardiogram in Dutch) is the record or **graphical registration** of electrical activities of the heart, which occur prior to the onset of mechanical activities. It is the **summed electrical activity** of all cardiac muscle fibers recorded from surface of the body.

USES OF ECG

Electrocardiogram is useful in determining and diagnosing the following:

1. Heart rate
2. Heart rhythm
3. Abnormal electrical conduction
4. Poor blood flow to heart muscle (ischemia)
5. Heart attack

6. Coronary artery disease
7. Hypertrophy of heart chambers.

■ ELECTROCARDIOGRAPHIC GRID

The paper that is used for recording ECG is called ECG paper. ECG machine amplifies the electrical signals produced from the heart and records these signals on a moving ECG paper.

Electrocardiographic grid refers to the markings (lines) on ECG paper. ECG paper has horizontal and vertical lines at regular intervals of 1 mm. Every 5th line (5 mm) is thickened.

■ DURATION

Time duration of different ECG waves is plotted horizontally on X-axis.

On X-axis

1 mm = 0.04 second
5 mm = 0.20 second

■ AMPLITUDE

Amplitude of ECG waves is plotted vertically on Y-axis.

On Y-axis

1 mm = 0.1 mV
5 mm = 0.5 mV

■ SPEED OF THE PAPER

Movement of paper through the machine can be adjusted by two speeds, 25 mm/second and 50 mm/second. Usually, speed of the paper during recording is fixed at 25 mm/second. If heart rate is very high, speed of the paper is changed to 50 mm/second.

■ ECG LEADS

ECG is recorded by placing series of electrodes on the surface of the body. These electrodes are called ECG leads and are connected to the ECG machine.

Electrodes are fixed on the limbs. Usually, right arm, left arm and left leg are chosen. Heart is said to be in the center of an **imaginary equilateral triangle** drawn by connecting the roots of these three limbs. This triangle is called Einthoven triangle.

Einthoven Triangle and Einthoven Law

Einthoven triangle is defined as an equilateral triangle that is used as a model of standard limb leads used to record electrocardiogram. Heart is presumed to lie in the center of Einthoven triangle.

Electrical potential generated from the heart appears simultaneously on the roots of the three limbs, namely the left arm, right arm and the left leg.

Refer next Chapter for Einthoven law.

ECG is recorded in 12 leads, which are generally classified into two categories.

I. Bipolar leads
II. Unipolar leads.

■ BIPOLAR LIMB LEADS

Bipolar limb leads are otherwise known as **standard limb leads.** Two limbs are connected to obtain these leads and both the electrodes are **active recording electrodes,** i.e. one electrode is positive and the other one is negative (Fig. 94.1).

Standard limb leads are of three types:

a. Limb lead I
b. Limb lead II
c. Limb lead III.

Lead I

Lead I is obtained by connecting right arm and left arm. Right arm is connected to the negative terminal of the instrument and the left arm is connected to the positive terminal.

Lead II

Lead II is obtained by connecting right arm and left leg. Right arm is connected to the negative terminal of the instrument and the left leg is connected to the positive terminal.

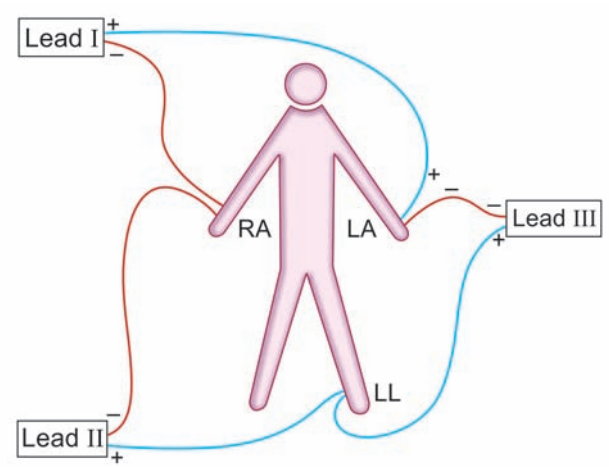

FIGURE 94.1: Position of electrodes for standard limb leads
RA = Right arm, LA = Left arm, LL=Left leg.

Lead III

Lead III is obtained by connecting left arm and left leg. Left arm is connected to the negative terminal of the instrument and the left leg is connected to the positive terminal.

■ UNIPOLAR LEADS

Here, one electrode is **active electrode** and the other one is an **indifferent electrode.** Active electrode is positive and the indifferent electrode is serving as a **composite negative electrode.**

Unipolar leads are of two types:

1. Unipolar limb leads
2. Unipolar chest leads.

1. *Unipolar Limb Leads*

Unipolar limb leads are also called **augmented limb leads** or **augmented voltage leads.** Active electrode is connected to one of the limbs. Indifferent electrode is obtained by connecting the other two limbs through a resistance.

Unipolar limb leads are of three types:

i. aVR lead
ii. aVL lead
iii. aVF lead.

i. *aVR lead*

Active electrode is from right arm. Indifferent electrode is obtained by connecting left arm and left leg.

ii. *aVL lead*

Active electrode is from left arm. Indifferent electrode is obtained by connecting right arm and left leg.

iii. *aVF lead*

Active electrode is from left leg (foot). Indifferent electrode is obtained by connecting the two upper limbs.

2. *Unipolar Chest Leads*

Chest leads are also called **'V' leads** or **precardial chest leads.** Indifferent electrode is obtained by connecting the three limbs, viz. left arm, left leg and right arm, through a **resistance** of 5000 ohms. Active electrode is placed on six points over the chest (Fig. 94.2). This electrode is known as the chest electrode and the six points over the chest are called V_1, V_2, V_3, V_4, V_5 and V_6. V indicates vector, which shows the direction of current flow.

Position of chest leads:

V_1 : Over 4th intercostal space near right sternal margin
V_2 : Over 4th intercostal space near left sternal margin

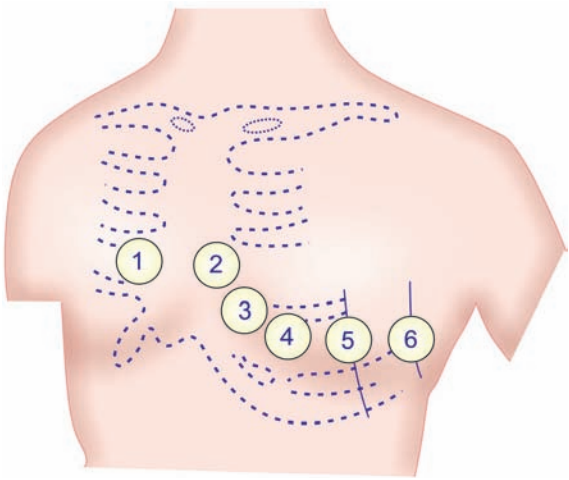

FIGURE 94.2: Position of electrodes for chest leads (V_1 to V_6)

V_3 : In between V_2 and V_4
V_4 : Over left 5th intercostal space on the mid clavicular line
V_5 : Over left 5th intercostal space on the anterior axillary line
V_6 : Over left 5th intercostal space on the mid axillary line.

■ WAVES OF NORMAL ECG

Normal ECG consists of waves, complexes, intervals and segments. Waves of ECG recorded by limb lead II are considered as the typical waves. Normal electrocardiogram has the following waves, namely P, Q, R, S and T (Table 94.1 and Fig. 94.3). Einthoven had named the waves of ECG starting from the middle of the English alphabets (P) instead of starting from the beginning (A).

Major Complexes in ECG

1. 'P' wave, the atrial complex
2. 'QRS' complex, the initial ventricular complex
3. 'T' wave, the final ventricular complex
4. 'QRST', the ventricular complex.

■ 'P' WAVE

'P' wave is a positive wave and the first wave in ECG. It is also called **atrial complex.**

Cause

'P' wave is produced due to the **depolarization** of **atrial musculature.** Depolarization spreads from SA node to all parts of atrial musculature. **Atrial repolarization** is not

TABLE 94.1: Waves of normal ECG

Wave/Segment	From – To	Cause	Duration (second)	Amplitude (mV)
P wave	–	Atrial depolarization	0.1	0.1 to 0.12
QRS complex	Onset of Q wave to the end of S wave	Ventricular depolarization and atrial repolarization	0.08 to 0.10	Q = 0.1 to 0.2 R = I S = 0.4
T wave	–	Ventricular repolarization	0.2	0.3
P-R interval	Onset of P wave to onset of Q wave	Atrial depolarization and conduction through AV node	0.18 (0.12 to 0.2)	–
Q-T interval	Onset of Q wave and end of T wave	Ventricular depolarization and ventricular repolarization	0.4 to 0.42	–
S-T segment	End of S wave and onset of T wave	Isoelectric	0.08	–

recorded as a separate wave in ECG because it merges with ventricular repolarization (QRS complex).

Duration

Normal duration of 'P' wave is 0.1 second.

Amplitude

Normal amplitude of 'P' wave is 0.1 to 0.12 mV.

Morphology

'P' wave is normally positive (upright) in leads I, II, aVF, V_4, V_5 and V_6. It is normally negative (inverted) in aVR. It is variable in the remaining leads, i.e. it may be positive, negative, biphasic or flat (Fig. 94.4).

Clinical Significance

Variation in the duration, amplitude and morphology of 'P' wave helps in the diagnosis of several cardiac problems such as:

1. *Right atrial hypertrophy:* 'P' wave is tall (more than 2.5 mm) in lead II. It is usually pointed
2. *Left atrial dilatation or hypertrophy:* It is tall and broad based or M shaped
3. *Atrial extrasystole:* Small and shapeless 'P' wave, followed by a small compensatory pause
4. *Hyperkalemia:* 'P' wave is absent or small
5. *Atrial fibrillation:* 'P' wave is absent
6. *Middle AV nodal rhythm:* 'P' wave is absent
7. *Sinoatrial block:* 'P' wave is inverted or absent
8. *Atrial paroxysmal tachycardia:* 'P' wave is inverted
9. *Lower AV nodal rhythm:* 'P' wave appears after QRS complex.

■ 'QRS' COMPLEX

'QRS' complex is also called the **initial ventricular complex.** 'Q' wave is a small negative wave. It is continued as the tall 'R' wave, which is a positive wave. 'R' wave is followed by a small negative wave, the 'S' wave.

Cause

'QRS' complex is due to **depolarization** of **ventricular musculature.** 'Q' wave is due to the depolarization of basal portion of interventricular septum. 'R' wave is due to the depolarization of apical portion of interventricular septum and apical portion of ventricular muscle. 'S' wave is due to the depolarization of basal portion of ventricular muscle near the atrioventricular ring.

Duration

Normal duration of 'QRS' complex is between 0.08 and 0.10 second.

Amplitude

Amplitude of 'Q' wave = 0.1 to 0.2 mV.
Amplitude of 'R' wave = 1 mV.
Amplitude of 'S' wave = 0.4 mV.

Morphology

'Q' wave is normally small with amplitude of 4 mm or less. It is less than 25% of amplitude of 'R' wave in leads I, II, aVL, V_5 and V_6. In the remaining leads, its amplitude is < 0.2 mm.

From chest leads V_1 to V_6, 'R' wave becomes gradually larger. It is smaller in V_6 than V_5. 'S' wave is large in V_1 and larger in V_2. It gradually becomes smaller from V_3 to V_6.

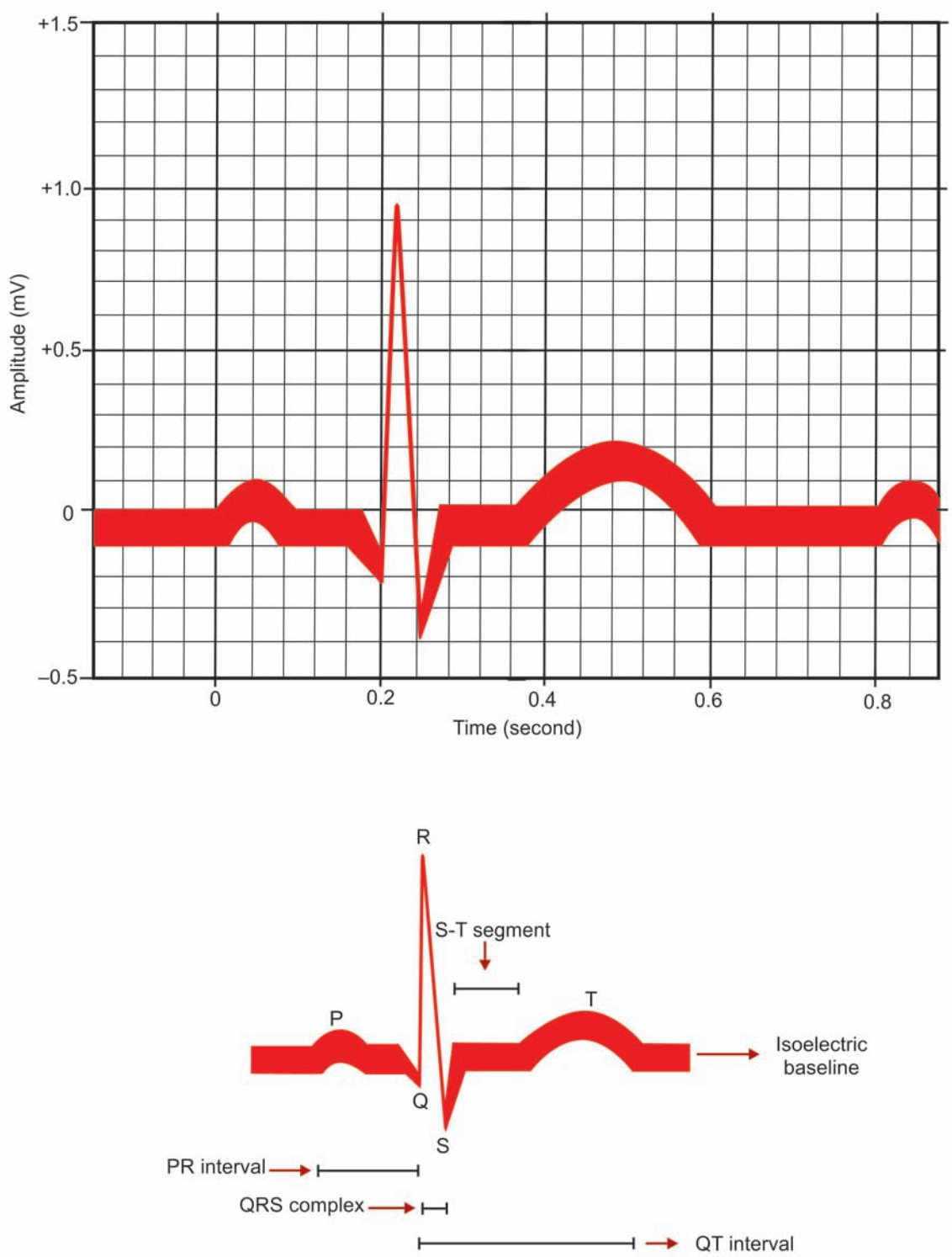

FIGURE 94.3: Waves of normal ECG

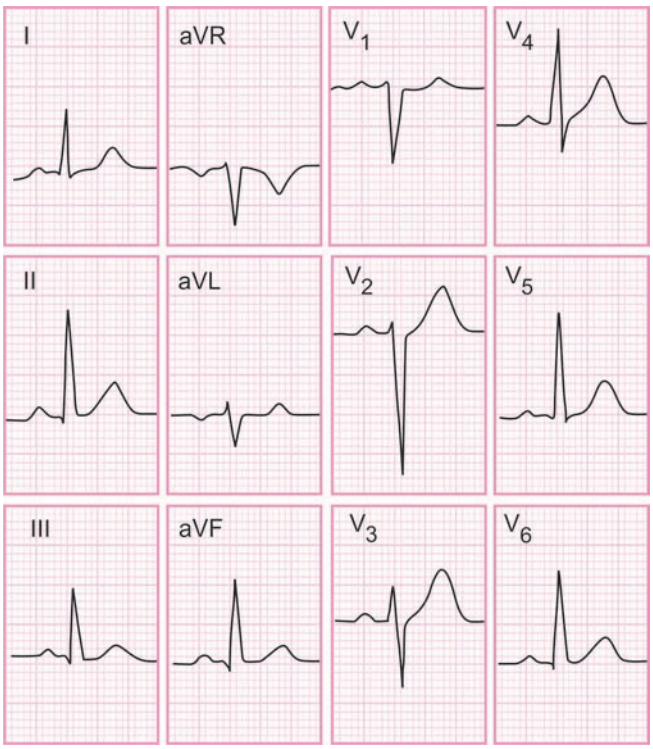

FIGURE 94.4: 12-lead ECG
(*Courtesy:* Dr Atul Ruthra)

Clinical Significance

Variation in the duration, amplitude and morphology of 'QRS' complex helps in the diagnosis of several cardiac problems such as:

1. *Bundle branch block:* QRS is prolonged or deformed
2. *Hyperkalemia:* QRS is prolonged.

■ 'T' WAVE

'T' wave is the **final ventricular complex** and is a positive wave.

Cause

'T' wave is due to the **repolarization** of **ventricular musculature.**

Duration

Normal duration of 'T' wave is 0.2 second.

Amplitude

Normal amplitude of 'T' wave is 0.3 mV.

Morphology

'T' wave is normally positive in leads I, II and V_5 and V_6. It is normally inverted in lead aVR. It is variable in the other leads, i.e. it is positive, negative or flat.

Clinical Significance

Variation in duration, amplitude and morphology of 'T' wave helps in the diagnosis of several cardiac problems such as:

1. *Acute myocardial ischemia:* Hyperacute 'T' wave develops. Hyperacute 'T' wave refers to a tall and broad-based 'T' wave, with slight asymmetry.
2. *Old age, hyperventilation, anxiety, myocardial infarction, left ventricular hypertrophy and pericarditis:* 'T' wave is small, flat or inverted
3. *Hypokalemia:* 'T' wave is small, flat or inverted
4. *Hyperkalemia:* 'T' wave is tall and tented.

■ 'U' WAVE

'U' wave is not always seen. It is also an insignificant wave in ECG. It is supposed to be due to **repolarization** of **papillary muscle.**

Clinical Significance

Appearance of 'U' wave in ECG indicates some clinical conditions such as:

1. *Hypercalcemia, thyrotoxicosis and hypokalemia:* 'U' wave appears. It is very prominent in hypokalemia.
2. *Myocardial ischemia:* Inverted 'U' wave appears.

■ INTERVALS AND SEGMENTS OF ECG

■ 'P-R' INTERVAL

'P-R' interval is the interval between the onset of 'P' wave and onset of 'Q' wave.

'P-R' interval signifies the atrial depolarization and conduction of impulses through AV node. It shows the duration of conduction of the impulses from the SA node to ventricles through atrial muscle and AV node.

'P' wave represents the atrial depolarization. Short **isoelectric** (zero voltage) period after the end of 'P' wave represents the time taken for the passage of depolarization within AV node.

Duration

Normal duration of 'P-R interval' is 0.18 second and varies between 0.12 and 0.2 second. If it is more than 0.2 second, it signifies the delay in the conduction of impulse from SA node to the ventricles. Usually, the delay occurs in the AV node. So it is called the **AV nodal delay.**

Clinical Significance

Variation in the duration of 'P-R' intervals helps in the diagnosis of several cardiac problems such as:

1. It is prolonged in bradycardia and first degree heart block

2. It is shortened in tachycardia, Wolf-Parkinson-White syndrome, Lown-Ganong-Levine syndrome, Duchenne muscular dystrophy and type II glycogen storage disease.

■ 'Q-T' INTERVAL

'Q-T' interval is the interval between the onset of 'Q' wave and the end of 'T' wave.

'Q-T' interval indicates the ventricular depolarization and ventricular repolarization, i.e. it signifies the electrical activity in ventricles.

Duration

Normal duration of Q-T interval is between 0.4 and 0.42 second.

Clinical Significance

1. 'Q-T' interval is prolonged in long 'Q-T' syndrome, myocardial infarction, myocarditis, hypocalcemia and hypothyroidism
2. 'Q-T' interval is shortened in short 'Q-T' syndrome and hypercalcemia.

■ 'S-T' SEGMENT

'S-T' segment is the time interval between the end of 'S' wave and the onset of 'T' wave. It is an isoelectric period.

J Point

The point where 'S-T' segment starts is called 'J' point. It is the junction between the QRS complex and 'S-T' segment.

Duration of 'S-T' Segment

Normal duration of 'S-T' segment is 0.08 second.

Clinical Significance

Variation in the duration of 'S-T' segment and its deviation from isoelectric base indicates the pathological conditions such as:

1. Elevation of 'S-T' segment occurs in anterior or inferior myocardial infarction, left bundle branch block and acute pericarditis. In athletes, 'S-T' segment is usually elevated
2. Depression of 'S-T' segment occurs in acute myocardial ischemia, posterior myocardial infarction, ventricular hypertrophy and hypokalemia
3. 'S-T' segment is prolonged in hypocalcemia
4. 'S-T' segment is shortened in hypercalcemia.

■ 'R-R' INTERVAL

'R-R' interval is the time interval between two consecutive 'R' waves.

Significance

'R-R' interval signifies the duration of one cardiac cycle.

Duration

Normal duration of 'R-R' interval is 0.8 second.

Significance of Measuring 'R-R' Interval

Measurement of 'R-R' interval helps to calculate:

1. Heart rate
2. Heart rate variability.

1. Heart Rate

Heart rate is calculated by measuring the number of 'R' waves per unit time.

Calculation of heart rate

Time is plotted horizontally (X-axis). On X-axis, interval between two thick lines is 0.2 sec (see above). Time duration for 30 thick lines is 6 seconds. Number of 'R' waves (QRS complexes) in 6 seconds (30 thick lines) is counted and multiplied by 10 to obtain heart rate. For the sake of convenience, the ECG paper has special time marking at every 3 seconds. So it is easy to find the time duration of 6 seconds.

2. Heart Rate Variability

Heart rate variability (HRV) refers to the beat-to-beat variations. Under resting conditions, the ECG of healthy individuals exhibits some periodic variation in 'R-R' intervals. This rhythmic phenomenon is known as **respiratory sinus arrhythmia** (RSA), since it fluctuates with the phases of respiration. 'R-R' interval decreases during inspiration and increases during expiration (Chapter 96).

Significance of Heart Rate Variability

HRV decreases in many clinical conditions like:

1. Cardiovascular dysfunctions such as hypertension
2. Diabetes mellitus
3. Psychiatric problems such as panic and anxiety.

Vector

Chapter 95

- INTRODUCTION
- INSTANTANEOUS MEAN VECTOR
- DEGREE OF INSTANTANEOUS MEAN VECTOR
  - DEGREE OF INSTANTANEOUS MEAN VECTOR AT DIFFERENT LIMB LEADS
- CALCULATED VECTOR OR MEAN QRS VECTOR
  - CALCULATION OF MEAN QRS VECTOR
- VECTORAL ANALYSIS
- VECTOR CARDIOGRAM

INTRODUCTION

Cardiac vector is the direction at which electrical potential generated in the heart travels at an instant. It is also called **cardiac axis.**

Vector is represented by an arrow. Arrowhead shows the **direction** of electrical potential. Length of the arrow represents the **amplitude** (magnitude or voltage) of the potential.

INSTANTANEOUS MEAN VECTOR

Current flows in all directions. Mean direction of flow of electrical potential at one instance is known as instantaneous mean vector or instantaneous summated vector (Fig. 95.1).

For example, when current flows through interventricular septum from the base of ventricles towards apex, the electrical potential generated by flow of current travels in different directions as follows:

1. Electrical potential travels downwards through the interventricular septum, towards the apical part, i.e. from depolarized part of septum towards non-depolarized (polarized) part of septum. This potential is strong.
2. Through the inner surface of ventricles, the potential travels upwards from apical part towards the base. Magnitude of this potential is very weak.

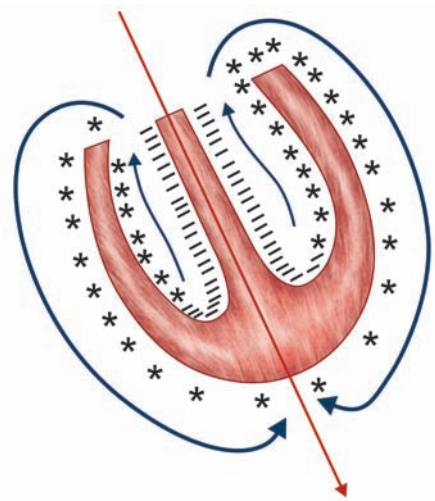

FIGURE 95.1: Instantaneous mean vector when current flows through interventricular septum of the heart

3. Through the outer surface of heart, the electrical potential travels downwards. It has a higher magnitude.

Though the potential travels in all directions in this instance, the potential flowing downwards (from base to apex of the heart) is much greater in magnitude than the potential flowing in other directions. Thus, the mean direction of flow of electrical potential in this instance is downwards. This downward vector is called

instantaneous mean vector or instantaneous summated vector at this instance.

■ DEGREE OF INSTANTANEOUS MEAN VECTOR

While recording electrocardiogram (ECG) in different limb leads, the degree of vector is altered. Direction of current flow is always from negative point towards the positive point. When the electrical potential flows in a horizontal plane from right side towards left side of the heart, the degree of vector is zero (Fig. 95.2).

■ DEGREE OF INSTANTANEOUS MEAN VECTOR AT DIFFERENT LIMB LEADS

Standard Limb Lead I (Right Arm and Left Arm)

In this instance, the electrical potential travels from right side (negative point) of the heart towards the left side (positive point) in the horizontal plane. So, the degree of vector is considered as zero.

Standard Limb Lead II (Right Arm and Left Leg)

Vector is from above downwards and slightly towards left, i.e. at 60°.

Standard Limb Lead III (Left Arm and Left Leg)

Here, vector is from above downwards and slightly towards right at 120°.

Lead Augmented Vector Right (aVR)

Vector is from below towards upper part of the heart and slightly towards right at 210°.

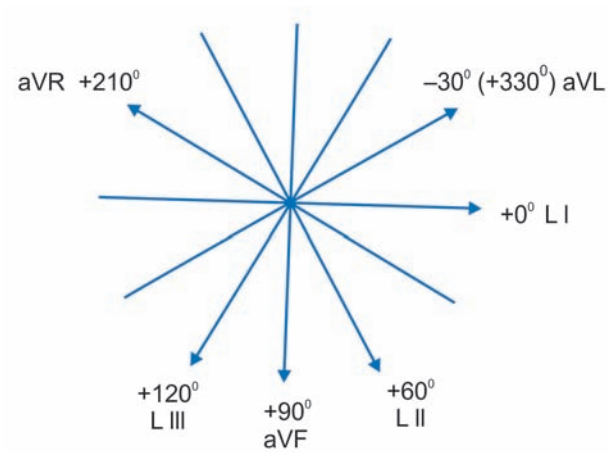

FIGURE 95.2: Degree of instantaneous vector at different leads

Lead Augmented Vector Front (aVF)

Vector is from above downwards at 90°.

Lead Augmented Vector Left (aVL)

In this, the vector is from below, towards upper part of the heart and slightly towards left, at –30° or at +330°.

■ CALCULATED VECTOR OR MEAN QRS VECTOR

Instantaneous mean vector cannot be determined by the recording of ECG. But, another vector can be calculated by measuring the amplitude of QRS complex from the ECG, recorded in standard limb leads. It is called the calculated vector or mean QRS vector.

It is also called the electrical axis of the heart or cardiac vector.

Calculated cardiac vector is useful in the diagnosis of heart diseases.

■ CALCULATION OF MEAN QRS VECTOR

Calculation of mean QRS vector depends upon the fact that if the amplitude of QRS complex is determined from ECG recorded at any two standard limb leads, the amplitude of QRS complex in the remaining lead can be known from the calculation.

Amplitude is measured in mm. For determining the amplitude of QRS complex, first the height of R wave is measured. From this value, height of negative wave Q or S (whichever is more) is deducted. The calculation is based on Einthoven triangle.

Einthoven Triangle

Refer previous Chapter for Einthoven triangle.

Steps for Calculation of Mean QRS Vector

1. An equilateral triangle is drawn on a plain paper. This triangle represents Einthoven triangle. Each side of this triangle represents one standard limb lead.
2. From the midpoint of each side, a perpendicular line is drawn towards the center. Meeting point of the perpendicular lines represents center of electrical activity in the heart (Fig. 95.3).
3. On each side of triangle, the amplitude of QRS complex is plotted from midpoint towards the positive point of the lead. For example, the amplitude of QRS complex in lead I is 10 mm and in lead II, it is 16 mm (Fig. 95.4).
4. In the triangle, upper side represents lead I and in this lead, the left is positive. So, a 10 mm line is drawn

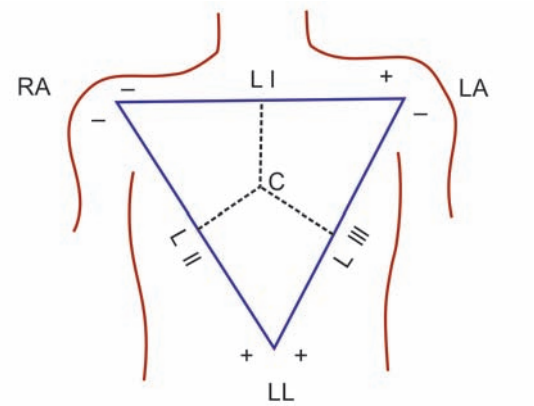

FIGURE 95.3: Einthoven triangle. C = Center of electrical activity, RA = Right arm, LA = Left arm, LL = Left leg, LI, LII and LIII = Standard limb leads.

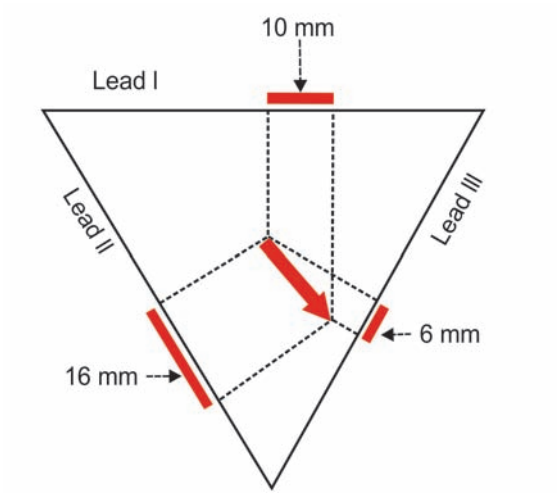

FIGURE 95.4: Calculation of cardiac vector. Arrow in the center indicates the cardiac vector.

on upper side from the midpoint, towards left (positive). This 10 mm distance along the axis of lead I is called **projected vector** for lead I.

5. In the same way, the projected vector for Lead II is drawn on the right side of the triangle
6. From the positive end of each projected vector another perpendicular line is drawn towards interior of the triangle
7. Now an arrow is drawn between center of electrical activity and the meeting point of perpendicular lines from positive end of projected vectors (Fig. 95.5).

   This arrow shows the vector. Arrowhead is drawn towards positive end, i.e. downwards.
8. Degree and the length of the arrow are measured. Degree denotes the direction of vector and length denotes the magnitude.

Amplitude of QRS Complex in Lead III

Amplitude (electrical potential) of QRS complex in lead III can be calculated by applying Einthoven law.

Einthoven law

Einthoven law states that potential differences between the bipolar leads measured simultaneously will, at any given moment, have the values II=I+III. That is, the potential of any wave or complex in lead II of ECG is equal to the sum of potentials in lead I and lead III. Einthoven law is the modification of Kirchhoff's law of voltage.

Kirchhoff's law of voltage

According to Kirchhoff law, the algebraic sum of voltage rise in a closed circuit is equal to the algebraic sum of voltage drops.

Application of Einthoven law in calculating QRS complex

By applying Einthoven law, amplitude (electrical potential) of QRS complex in one lead can be mathematically calculated, by summing up or subtracting the amplitude in other two leads, depending upon the potentials of these leads.

For example, amplitude of QRS in lead II = I + III and the amplitude of QRS in lead III = II – I. Thus, in this case of Einthoven triangle mentioned above, amplitude of QRS in lead I is 1 mV and lead II is 1.6 mV. Thus, the amplitude of QRS in lead III is 0.6 mV. It can also be measured from the triangle drawn to calculate the vector (Fig. 95.5).

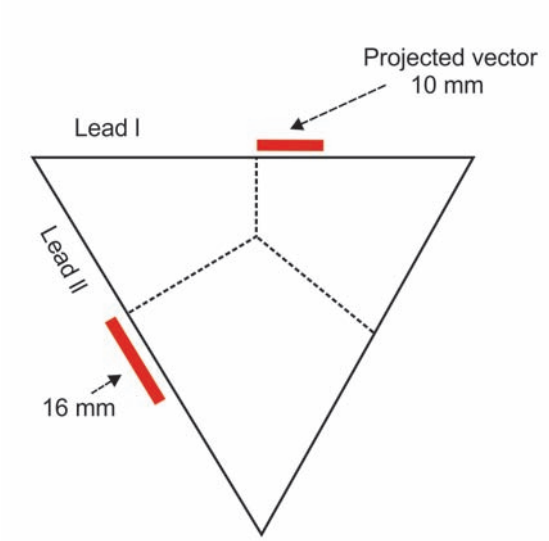

FIGURE 95.5: Determination of vector in Lead I and Lead II

■ VECTORAL ANALYSIS

Mean QRS vector (cardiac axis) in normal conditions is at about +59°. It varies between –30° and +110°.

When the axis deviates towards the left, i.e. in anti-clockwise direction, away from –30°, it is called left axis deviation. When the axis deviates towards the right (clockwise direction), away from +110°, it is known as right axis deviation.

Left axis deviation

Left axis deviation occurs in left ventricular hypertrophy, left bundle-branch block and posterior wall infarction.

Right axis deviation

Right axis deviation occurs due to right ventricular hypertrophy, right bundle-branch block and anterior wall infarction.

■ VECTOR CARDIOGRAM

From the recording of the electrocardiogram, only the calculated vector, i.e. cardiac axis is determined. Instantaneous mean vector cannot be determined by the electrocardiogram, but it can be determined by means of vector cardiogram.

Vector cardiogram is the simultaneous recording of electrical potential in different axis across the heart above, downward and sideward. It is obtained by using a cathode-ray oscilloscope. The technique is equal to connecting the tops of all instantaneous mean vectors in the series of 3 loops. It is done by means of a sophisticated electronic device along with oscilloscope.

Each loop of electronic connection is used to record different vector cardiogram called P vector cardiogram, QRS vector cardiogram and T vector cardiogram.

Arrhythmia

Chapter 96

- DEFINITION
- CLASSIFICATION
- NORMOTOPIC ARRHYTHMIA
  - SINUS ARRHYTHMIA
  - SINUS TACHYCARDIA
  - SINUS BRADYCARDIA
- ECTOPIC ARRHYTHMIA
  - HEART BLOCK
  - EXTRASYSTOLE
  - PAROXYSMAL TACHYCARDIA
  - ATRIAL FLUTTER
  - ATRIAL FIBRILLATION
  - VENTRICULAR FIBRILLATION
- ABNORMAL PACEMAKER
- ARTIFICIAL PACEMAKER
- CURRENT OF INJURY

DEFINITION

Arrhythmia refers to **irregular heartbeat** or disturbance in the rhythm of heart. In arrhythmia, heartbeat may be fast or slow or there may be an extra beat or a missed beat. It occurs in physiological and pathological conditions.

CLASSIFICATION

In arrhythmia, SA node may or may not be the pacemaker. If SA node is not the pacemaker, any other part of the heart such as atrial muscle, AV node and ventricular muscle becomes the pacemaker.

Accordingly, arrhythmia is classified into two types:

A. Normotopic arrhythmia
B. Ectopic arrhythmia.

NORMOTOPIC ARRHYTHMIA

Normotopic arrhythmia is the irregular heartbeat, in which SA node is the pacemaker.

Normotopic arrhythmia is of three types:

1. Sinus arrhythmia
2. Sinus tachycardia
3. Sinus bradycardia.

SINUS ARRHYTHMIA

Sinus arrhythmia is a normal rhythmical increase and decrease in heart rate, in relation to respiration. It is also called **respiratory sinus arrhythmia** (RSA). Normal sinus rhythm means the normal heartbeat with SA node as the pacemaker.

Normal heart rate is 72 per minute. However, under physiological conditions, in a normal healthy person, heart rate varies according to the phases of respiratory cycle. Heart rate increases during inspiration and decreases during expiration.

ECG Changes

ECG is normal during sinus arrhythmia. Only the duration of R-R interval varies rhythmically according to phases

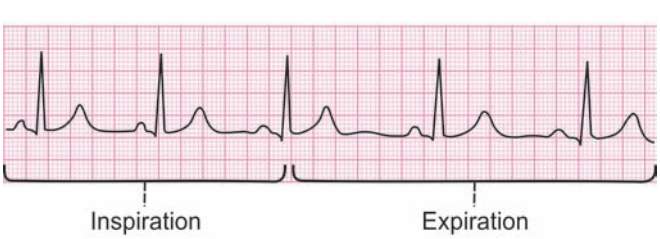

FIGURE 96.1: ECG in sinus arrhythmia. Normal P-QRS-T. R-R interval is shortened during inspiration and prolonged during expiration (*Courtesy:* Dr Atul Ruthra).

of respiration (Fig. 96.1). It is shortened during inspiration and prolonged during expiration (Chapter 94).

Cause

Sinus arrhythmia is due to fluctuation in the discharge of impulses from SA node (Fig. 96.2). During inspiration, the lungs are inflated and the intrathoracic pressure decreases. This increases the venous return. Inflation of lungs stimulates the stretch receptors of lungs, which send impulses to vasodilator area (cardioinhibitory center) through afferent fibers of vagus. It leads to reflex inhibition of vasodilator area and reduction in vagal tone. Because of these two factors, heart rate increases. Simultaneously, increased venous return initiates **Bainbridge reflex** that causes increase in heart rate (Chapter 101).

During expiration, the lungs are deflated and intrathoracic pressure increases. This decreases the venous return. During deflation of lungs, the stretch receptors are not stimulated and vasodilator area is not inhibited. So, vagal tone increases, resulting in decreased heart rate. Simultaneously, decreased venous return abolishes Bainbridge reflex. It also decreases the heart rate.

■ SINUS TACHYCARDIA

Sinus tachycardia is the increase in discharge of impulses from SA node, resulting in increase in heart rate. Discharge of impulses from SA node is very rapid and the heart rate increases up to 100/minute and sometimes up to 150/minute.

ECG Changes

ECG is normal in sinus tachycardia, except for short R-R intervals because of increased heart rate (Fig. 96.3).

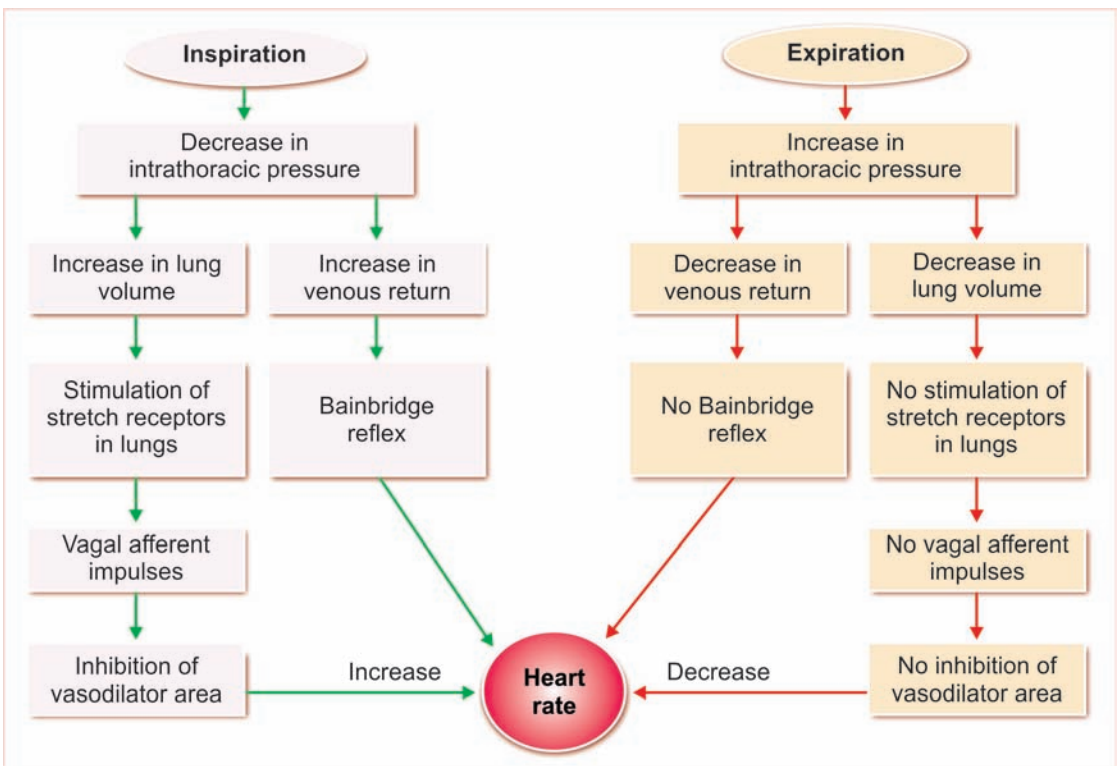

FIGURE 96.2: Sinus arrhythmia

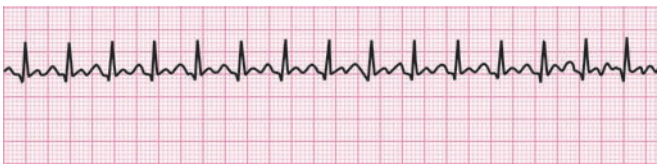

FIGURE 96.3: ECG in sinus tachycardia. Normal P-QRS-T. R-R interval is shortened. Heart rate is more than 100/min (*Courtesy:* Dr Atul Ruthra).

Conditions when Sinus Tachycardia Occurs

Sinus tachycardia occurs in physiological as well as pathological conditions.

Physiological conditions when tachycardia occurs

1. Exercise
2. Emotion
3. High altitude
4. Pregnancy.

Pathological conditions when tachycardia occurs

1. Fever
2. Anemia
3. Hyperthyroidism
4. Hypersecretion of catecholamines
5. Cardiomyopathy
6. Valvular heart disease
7. Hemorrhagic shock.

Features of Sinus Tachycardia

1. Palpitations (sensation of feeling the heartbeat)
2. Dizziness
3. Fainting
4. Shortness of breath
5. Chest discomfort (angina).

■ SINUS BRADYCARDIA

Sinus bradycardia is the reduction in discharge of impulses from SA node resulting in decrease in heart rate. Heart rate is less than 60/minute.

ECG Changes

ECG shows prolonged waves and prolonged R-R interval (Fig. 96.4).

Conditions when Sinus Bradycardia Occurs

Sinus bradycardia occurs in both physiological and pathological conditions. It occurs during sleep. It is common in athletes due to the cardiovascular reflexes, in response to increased force of contraction of heart.

Physiological conditions when sinus bradycardia occurs

1. Sleep
2. Athletic heart.

Pathological conditions when sinus bradycardia occurs

1. Disease of SA node
2. Hypothermia
3. Hypothyroidism
4. Heart attack
5. Congenital heart disease
6. Degenerative process of aging
7. Obstructive jaundice
8. Increased intracranial pressure
9. Use of certain drugs like beta blockers, channel blockers, digitalis and other **antiarrhythmic drugs**
10. Atherosclerosis. Bradycardia due to **atherosclerosis** of carotid artery, at the region of carotid sinus is called **carotid sinus syndrome.**

Features of Sinus Bradycardia

1. Sick sinus syndrome
2. Fatigue
3. Weakness
4. Shortness of breath
5. Lack of concentration
6. Difficulty in exercising.

Sick sinus syndrome

Sick sinus syndrome is the common feature of sinus bradycardia. It is the condition characterized by dizziness and unconsciousness.

■ ECTOPIC ARRHYTHMIA

Ectopic arrhythmia is the abnormal heartbeat, in which one of the structures of heart other than SA node becomes the pacemaker. Impulses produced by these structures are called **ectopic foci.**

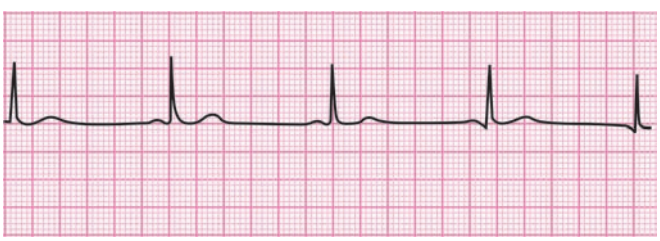

FIGURE 96.4: ECG in sinus bradycardia. Normal P-QRS-T. R-R interval is prolonged. Heart rate is less than 60/min. (*Courtesy:* Dr Atul Ruthra).

Subtypes of Ectopic Arrhythmia

Ectopic arrhythmia is further divided into two subtypes:

1. Homotopic arrhythmia, in which the impulses for heartbeat arise from any part of conductive system
2. Heterotopic arrhythmia, in which the impulses arise from the musculature of heart other than conductive system.

Different Ectopic Arrhythmia

1. Heart block
2. Extrasystole
3. Paroxysmal tachycardia
4. Atrial flutter
5. Atrial fibrillation
6. Ventricular fibrillation.

■ HEART BLOCK

Heart block is the blockage of impulses generated by SA node in the conductive system. Because of the blockage, the impulses cannot reach the cardiac musculature, resulting in ectopic arrhythmia. Based on the area affected, the heart block is classified into two types (Fig. 96.5):

1. Sinoatrial block
2. Atrioventricular block.

Sinoatrial Block – AV Nodal Rhythm

Sinoatrial block is the failure of impulse transmission from SA node to AV node. It is also called **sinus block.** During sinoatrial block, heart stops beating. Immediately, AV node takes over the pacemaker function and produces the impulses. This leads to AV nodal (atrioventricular) rhythm.

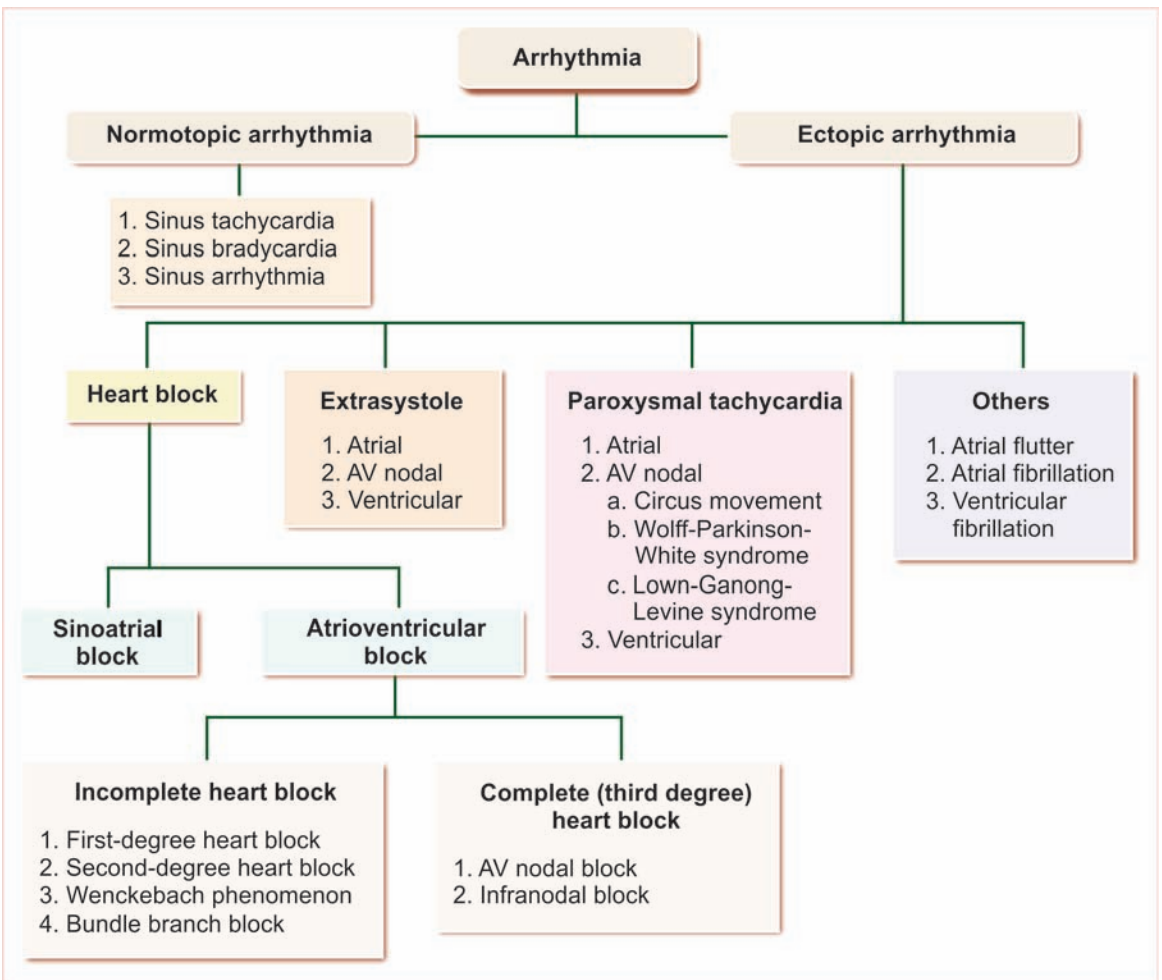

FIGURE 96.5: Classification of arrhythmia. AV = Atrioventricular.

Sinoatrial block is due to the defect in internodal fibers and it occurs suddenly. Initially, the heart stops for a while. Then after few seconds, the AV node becomes the pacemaker and the heart starts beating with decreased rate of 40 to 60/minute.

Impulses may be discharged from any part of AV node, viz.

1. In upper nodal rhythm, the impulses are discharged from the upper part of AV node. In this rhythm, the P wave of ECG is inverted. QRS complex and T wave are normal
2. In middle nodal rhythm, the impulses are by the middle part of AV node. Here, all the chambers of the heart contract simultaneously. P wave of ECG is absent as it merges with QRS complex
3. In lower nodal rhythm, the impulses are produced by the lower part of AV node. In this condition, ventricular contraction occurs prior to atrial contraction as the impulses reach the ventricles prior to the atria. In ECG, QRS complex appears prior to P wave and R-P interval is obtained instead of P-R interval. It is called **reversed heart block.**

Atrioventricular Block

Atrioventricular block is the heart block in which the impulses are not transmitted from atria (from AV node) to ventricles because of defective conductive system.

Atrioventricular block is of two categories:

1. Incomplete heart block
2. Complete heart block.

1. *Incomplete Heart Block*

Incomplete heart block is the condition in which the transmission of impulses from atria to ventricles is slowed down and not blocked completely. Impulses reach ventricles late.

Incomplete heart block is of four types:

i. First degree heart block
ii. Second degree heart block
iii. Wenckebach phenomenon
iv. Bundle branch block.

i. *First degree heart block*

First degree heart block is the heart block in which the conduction of impulses through AV node is very slow, i.e. the AV nodal delay is longer. It is also called **delayed conduction.** In ECG, the P-R interval is very much prolonged and is more than 0.2 second.

First degree heart block is common in young adults and trained athletes. It is also caused by rheumatic fever and some drugs. It does not produce any symptom.

ii. *Second degree heart block*

Second degree heart block is the type of heart block in which some of the impulses produced by SA node fail to reach the ventricles. It is also called the **partial heart block.** When some of the impulses from SA node fail to reach the ventricles, one ventricular contraction occurs for every 2, 3 or 4 atrial contractions, i.e. 2 : 1, 3 : 1 or 4 : 1. In ECG, the ventricular complex (QRST) is missing accordingly.

During frequent development of second degree heart block, bradycardia occurs.

iii. *Wenckebach phenomenon or syndrome*

Wenckebach phenomenon is a type of heart block characterized by progressive increase in AV nodal delay, resulting in missing of one beat. Afterwards, the conduction of impulse is normal or slightly delayed. In ECG, the progressive lengthening of P-R interval is noticed till QRST complex disappears.

iv. *Bundle branch block*

Bundle branch block (BBB) is the heart block that occurs during dysfunction of right or left branch of bundle of His. During this type of block, the impulse from atria reaches unaffected ventricle first. Then, from here, the impulse travels to the affected side. So, ECG shows normal ventricular rate, but the QRS complex is prolonged or deformed.

2. *Complete Heart Block (Third degree heart block)*

Complete heart block is the condition in which the impulses produced by SA node cannot reach the ventricles. It is also called **complete atrioventricular block** or third degree heart block. Because of this, the ventricles beat in their own rhythm, independent of atrial beat. It is called **idioventricular rhythm.**

Complete heart block occurs due to any one of the following causes:

i. Disease of AV node, which leads to AV nodal block
ii. Defective conductive system below the level of AV node, causing infranodal block.

i. *AV nodal block*

In this type of block, a part of AV node is defective and the unaffected part becomes the pacemaker. Rhythmicity of AV node is about 45 to 60/minute.

ii. *Infranodal block*

Infranodal block is the heart block in which the impulses from SA node are blocked in the branches of bundle of His (below the level of AV node). In this condition, the

distal part of the conductive system (i.e. the Purkinje fibers) becomes the pacemaker. The rhythmicity of Purkinje fibers is about 35/minute. Sometimes, a part of ventricular musculature becomes the pacemaker and the ventricular rate in such conditions is about 20/minute.

Third degree heart block is the serious one since it decreases the pumping action of the heart. Very often, it results in Stokes-Adams syndrome. It may also cause heart failure.

Stokes-Adams syndrome

Stokes-Adams syndrome is the sudden attack of dizziness and unconsciousness caused by heart block. It may be accompanied by convulsions also. In many patients suffering from heart block, the complete heart block occurs intermittently. When the block occurs, the ventricles stop beating immediately. **Ectopic pacemaker** (AV node, Purkinje fiber or ventricular muscle) starts functioning only after 5 to 30 seconds.

During this time, the blood circulation is affected because of lack of ventricular output. Brain cannot withstand the stoppage of blood supply and oxygen supply even for 5 seconds. Before the onset of discharge from ectopic pacemaker, dizziness and fainting occurs. If the discharge of impulses from ectopic pacemaker is delayed beyond 30 seconds, death occurs.

■ EXTRASYSTOLE

Extrasystole and Compensatory Pause

Extrasystole is the premature contraction of the heart before its normal contraction. It is caused by an ectopic focus (discharge of an impulse from any part of the heart other than the SA node). The ectopic focus produces an extra beat of the heart that is always followed by a compensatory pause. Compensatory pause is the period during which the heart stops in relaxed state.

Cause for the compensatory pause

In the cardiac muscle, absolute refractory period extends throughout contraction period. When the heart is in extrasystole (because of ectopic focus), an impulse is discharged from natural pacemaker, SA node. As this natural impulse reaches the myocardium during the contraction period of extrasystole, the myocardium does not give response, because it is refractory now. For the next beat, the heart has to wait till the discharge of next natural impulse from SA node. During this time, the heart stops in diastole. It is the cause for compensatory pause (Chapter 90).

Parts of the heart which give origin for ectopic foci are AV node, bundle of His, atrial musculature and ventricular musculature.

Accordingly, extrasystole is divided into three types:

1. Atrial extrasystole
2. Nodal extrasystole
3. Ventricular extrasystole.

1. *Atrial Extrasystole*

Atrial extrasystole is the premature contraction produced by a stimulus arising from atrial muscle. In this condition, an extra P wave appears immediately after the regular T wave. P wave is small and shapeless. The P-R interval of this beat is short.

2. *Nodal Extrasystole*

Nodal extrasystole is caused by stimulus arising from AV node. P wave is merged with QRS complex and all the chambers of the heart contract together.

3. *Ventricular Extrasystole*

Ventricular extrasystole is the extrasystole that is caused by stimulus from ventricular muscle. In this condition, an extra QRS complex follows the regular T wave. This QRS complex is prolonged as the impulse is conducted through ventricular muscle and not through the conductive system. This QRS complex also has a high voltage. T wave of this beat is inverted.

Conditions when Extrasystole Occurs

Extrasystole is associated with organic diseases of the heart. Particularly, any ischemic area of ventricular musculature can produce an ectopic focus.

Other conditions which produce extrasystole:

i. Emotions
ii. Severe exhaustion
iii. Excessive ingestion of coffee or alcohol
iv. Excessive smoking
v. Hyperthyroidism
vi. Reflexes elicited from abnormal viscera.

■ PAROXYSMAL TACHYCARDIA

Paroxysmal tachycardia is the sudden attack of increased heart rate due to ectopic foci arising from atria, AV node or ventricle. It is also called **Bouveret-Hoffmann syndrome.**

Increase in heart rate due to ectopic foci arising from either atria or AV node is called **supraventricular**

tachycardia (SVT). It differs from ventricular tachycardia, which does not depend upon atria or AV node. The attack lasts for a period of few seconds to few hours. It also stops suddenly. After the attack, heart functions normally. Symptoms include palpitations, chest pain, rapid breathing and dizziness.

Paroxysmal tachycardia is of three types:

1. Atrial paroxysmal tachycardia
2. AV nodal paroxysmal tachycardia
3. Ventricular paroxysmal tachycardia.

1. *Atrial Paroxysmal Tachycardia*

Atrial paroxysmal tachycardia is the sudden increase in heart rate caused by ectopic impulses discharged from atrial musculature. Heart rate is 150 to 220/minute. P wave in ECG is inverted, with normal QRST.

2. *AV Nodal Paroxysmal Tachycardia – Bundle of Kent*

AV nodal paroxysmal tachycardia is the sudden increase in heart rate caused by ectopic foci arising from AV node due to a temporary block in the conductive system. It also involves circus movement. This type of tachycardia is very common in some healthy persons who have got an additional conductive system. This system is formed by some abnormal junctional tissues constituting a structure called bundle of Kent. Bundle of Kent connects the atria and ventricles directly, so the conduction is very rapid than through the regular conductive system.

Circus movement – Re-entry and atrial echo beat

Circus movement is defined as circuitous propagation of impulses around a structural or functional obstruction, resulting in re-entry of the impulse and **re-excitation** of heart. When there is a sudden and temporary block in normal conductive system, the impulses from SA node reach the ventricle through bundle of Kent. By this time, the blockage in normal conductive system disappears. Now, the impulse, which passes through bundle of Kent, after exciting the ventricular muscle, travels in the opposite direction through the normal conductive system and finally, it re-enters the AV node. Re-entered impulse activates the AV node and depolarizes the atria, resulting in atrial contraction. It is called **atrial echo beat.**

Re-entered nodal impulse simultaneously spreads to ventricle through normal conductive system, completing the circus movement. This circus movement is repeated producing tachycardia called AV nodal paroxysmal tachycardia. ECG shows normal QRST complex. But P wave is mostly absent.

Wolff-Parkinson-White syndrome

Wolff-Parkinson-White syndrome is the condition characterized by repeated attacks of AV nodal paroxysmal tachycardia in persons with bundle of Kent. ECG shows short P-R interval with normal QRS complex and T wave.

Lown-Ganong-Levin syndrome

Lown-Ganong-Levin syndrome is another condition characterized by AV nodal paroxysmal tachycardia. This occurs in persons who have got another type of abnormal conductive fibers like bundle of Kent. These fibers also connect atria and distal part of conductive system directly bypassing the AV node. So the impulse from SA node reaches ventricle through the abnormal conductive fibers. After exciting the ventricular muscle, the impulse travels in opposite direction through normal conductive system and finally, it re-enters the AV node. The re-entered impulse activates the AV node causing atrial contraction. ECG shows short P-R interval with normal QRS complex and T wave.

3. *Ventricular Paroxysmal Tachycardia*

Ventricular paroxysmal tachycardia is the sudden increase in heart rate caused by ectopic foci arising from ventricular musculature. Sometimes, a part of ventricular muscle, particularly an ischemic area is excited abnormally, followed by a series of extrasystole. This condition is dangerous as the circus movement is developed within ventricular muscle. This circus movement leads to ventricular fibrillation, which is fatal.

■ ATRIAL FLUTTER

Atrial flutter is an arrhythmia characterized by rapid ineffective atrial contractions, caused by ectopic foci originating from atrial musculature. It is often associated with atrial paroxysmal tachycardia. Both the atria beat rapidly like the wings of a bird, hence the name atrial flutter.

Atrial rate is about 250 to 350/minute. Maximum number of impulses conducted by AV node is about 230 to 240 /minute. So, during atrial flutter, the second degree of heart block occurs. The ratio between atrial beats and ventricular beats is 2 : 1 or sometimes 3 : 1.

Atrial flutter is common in patients suffering from cardiovascular diseases such as hypertension and coronary artery disease. Initially, it is marked by palpitations that are unnoticed. However, prolonged atrial flutter may lead to atrial fibrillation or heart failure.

■ ATRIAL FIBRILLATION

Atrial fibrillation is the type of arrhythmia characterized by rapid and irregular atrial contractions at the rate of 300 to 400 beats/minute. It is mostly due to circus movement of impulses within atrial musculature. P wave is absent in ECG.

Atrial fibrillation is common in old people and patients with heart diseases. Though it is not life-threatening, it may cause complications. If it continues for long time, it may cause blood clot and blockage of blood flow to vital organs.

■ VENTRICULAR FIBRILLATION

Ventricular fibrillation is the dangerous cardiac arrhythmia, characterized by rapid and irregular twitching of ventricles. Ventricles beat very rapidly and irregularly due to the circus movement of impulses within ventricular muscle. The rate reaches 400 to 500/minute. This is triggered by ventricular extrasystole. This type of arrhythmia is serious as it leads to death, since the ventricles cannot pump blood.

Ventricular fibrillation is very common during electric shock and during ischemia of conductive system. It also occurs in other conditions like coronary occlusion, chloroform anesthesia, cyclopropane anesthesia, trauma of heart and disturbances of heart (due to improper handling) during cardiac surgery.

■ ABNORMAL PACEMAKER

Abnormal pacemaker is the part of the heart other than SA node that becomes the pacemaker and discharges ectopic foci. Various types of arrhythmia develop when an abnormal pacemaker is activated. These arrhythmias are already described in this Chapter.

Common abnormal pacemakers:

1. Atrioventricular node
2. Atrial musculature
3. Ventricular musculature.

1. *AV Node as Pacemaker*

When AV node becomes the pacemaker, the following arrhythmias occur:

i. AV nodal rhythm
ii. AV nodal extrasystole
iii. AV nodal paroxysmal tachycardia.

2. *Atrial Musculature as Pacemaker*

Following arrhythmias occur if atrial musculature becomes the pacemaker:

i. Atrial extrasystole
ii. Atrial paroxysmal tachycardia
iii. Wolff-Parkinson-White syndrome
iv. Lown-Ganong-Levine syndrome
v. Atrial flutter
vi. Atrial fibrillation.

3. *Ventricular Musculature as Pacemaker*

If ventricular muscle becomes the pacemaker, following arrhythmias are developed:

i. Ventricular extrasystole
ii. Ventricular paroxysmal tachycardia
iii. Ventricular fibrillation.

■ ARTIFICIAL PACEMAKER

Artificial pacemaker is a small **electronic device** that is surgically implanted to regulate abnormal heartbeat. It contains a battery powered **pulse generator,** that produces electrical impulses capable of stimulating the heart. This pacemaker is implanted under the skin over the chest of the patient. Pulses generated by this device are transmitted to the heart through electrodes. Electrodes connected to the device are inserted and passed through a vein and positioned in the heart chambers. The device has a **lithium battery** that may last for 10 to 15 years. The outer casing of the pacemaker is usually made of titanium, which is rarely rejected by body's immune system.

Pulse generator of the pacemaker has multiple functions. It is programmed to cope up with the needs of the individual patient.

■ CURRENT OF INJURY

Current of injury means flow of current from an injured region of heart to the unaffected part. When ischemia occurs in any part of the ventricular musculature due to coronary occlusion, that part of ventricle becomes depolarized either partially or completely and the repolarization does not occur. It causes flow of current from affected (depolarized) part to unaffected part of the ventricular muscle.

Current of injury in myocardial infarction affects the ECG pattern and cardiac vector. In ECG, the J point and ST segments are displaced (Chapter 94). Deviation of cardiac axis is also common during the current of injury.

Cardiac Axis

In the infarction of anterior wall of the ventricle, the cardiac axis (vector) is deviated to right up to +150° due to current of injury and in the posterior wall infarction, there is left axis deviation up to –95°.

Effect of Changes in Electrolyte Concentration on Heart

Chapter 97

- INTRODUCTION
- EFFECT OF CHANGES IN SODIUM ION CONCENTRATION
- EFFECT OF CHANGES IN POTASSIUM ION CONCENTRATION
  - EFFECT OF HYPERKALEMIA
  - EFFECT OF HYPOKALEMIA
- EFFECT OF CHANGES IN CALCIUM ION CONCENTRATION
  - EFFECT OF HYPERCALCEMIA
  - EFFECT OF HYPOCALCEMIA
- EXPERIMENTAL EVIDENCES

INTRODUCTION

Distribution of electrolytes in extracellular fluid and intracellular fluid is responsible for the electrical activity of the tissues including myocardium. Thus, any change in the concentration of any electrolyte will definitely alter the electrical activity of cardiac muscle.

EFFECT OF CHANGES IN SODIUM ION CONCENTRATION

Normal sodium ion concentration in blood is 135 to 145 mEq/L. Change in concentration of sodium ion does not alter the electrical activity of heart severely. Only the low level of sodium ion in body fluids reduces the electrical activity of cardiac muscle and electrocardiogram (ECG) shows low-voltage waves.

Changes in the concentration of potassium and calcium ions have significant effects on heart.

EFFECT OF CHANGES IN POTASSIUM ION CONCENTRATION

Normal potassium ion concentration in blood is about 3.5 to 5 mEq/L. Changes in ECG appear when the potassium level increases to 6 mEq/L (hyperkalemia) or when it decreases to 2 mEq/L (hypokalemia).

EFFECT OF HYPERKALEMIA

Hyperkalemia decreases:

1. Resting membrane potential, leading to hyperpolarization
2. Excitability of the muscle.

Effects of hyperkalemia on the excitability of cardiac muscle, depend upon the severity of hyperkalemia.

Changes in ECG When Potassium Level Increases to 6 or 7 mEq/L

T wave is tall and tented. P-R interval and QRS complex are normal.

Changes in ECG When Potassium Level Increases to 8 mEq/L

P-R interval and the duration of QRS complex are prolonged because, hyperkalemia decreases the rate of conduction. P wave may be small.

Changes in ECG When Potassium Level Increases beyond 9 mEq/L

Severe hyperkalemia makes the atrial muscle unexcitable. So, P wave is absent in ECG. QRS complex merges with T wave. This condition is fatal because, it

leads to ventricular fibrillation or stoppage of heart in diastole, due to the lack of excitability.

■ EFFECT OF HYPOKALEMIA

Hypokalemia decreases the sensitivity of heart muscle.

Changes in ECG When Potassium Level Falls to 2 mEq/L

1. S-T segment is depressed
2. T wave is small, flat or inverted
3. U wave appears. Sometimes, the U wave merges with T wave. Because of this, the Q-T interval is mistaken for being prolonged.

Changes in ECG When Potassium Level Falls below 2 mEq/L

1. Depression of S-T segment below the isoelectric baseline
2. Inversion of T wave
3. Appearance of prominent U wave
4. Prolongation of P-R interval.

■ EFFECT OF CHANGES IN CALCIUM ION CONCENTRATION

Normal concentration of calcium ion in blood is 9 to 11 mg/dL (4.5 to 5.5 mEq/L). Mostly, hypocalcemia affects the heart, rather than hypercalcemia.

■ EFFECT OF HYPERCALCEMIA

Hypercalcemia is the elevation in blood calcium level. It increases the excitability and contractility of the heart muscle. In clinical conditions, the effect of hypercalcemia is very rare.

Changes in ECG

1. Shortening of duration of S-T segment
2. Shortening of QT interval
3. Appearance of U wave.

Calcium Rigor

Stoppage of the heart in systole, due to hypercalcemia is called the calcium rigor. It can be demonstrated in experimental animals by infusing large quantity of calcium. Calcium rigor is a reversible phenomenon and the heart starts functioning normally, when the calcium ions are washed.

■ EFFECT OF HYPOCALCEMIA

Hypocalcemia is the reduction in blood calcium level. It reduces the excitability of the cardiac muscle.

Changes in ECG

1. Prolongation of S-T segment
2. Prolongation of Q-T interval
3. Appearance of a prominent U wave.

■ EXPERIMENTAL EVIDENCES

Effects of ions on heart are demonstrated experimentally by perfusion of heart from animals such as frog and rabbit.

Cardiac Output

Chapter 98

- INTRODUCTION
- DEFINITIONS AND NORMAL VALUES
  - STROKE VOLUME
  - MINUTE VOLUME
  - CARDIAC INDEX
- EJECTION FRACTION
- CARDIAC RESERVE
- VARIATIONS IN CARDIAC OUTPUT
  - PHYSIOLOGICAL VARIATIONS
  - PATHOLOGICAL VARIATIONS
- DISTRIBUTION OF CARDIAC OUTPUT
- FACTORS MAINTAINING CARDIAC OUTPUT
  - VENOUS RETURN
  - FORCE OF CONTRACTION
  - HEART RATE
  - PERIPHERAL RESISTANCE
- MEASUREMENT OF CARDIAC OUTPUT
  - DIRECT METHODS
  - INDIRECT METHODS
- CARDIAC CATHETERIZATION
  - DEFINITION
  - CONDITIONS WHEN CARDIAC CATHETERIZATION IS PERFORMED
  - PROCEDURE
  - USES OF CARDIAC CATHETERIZATION

INTRODUCTION

Cardiac output is the amount of blood pumped from each ventricle. Usually, it refers to left ventricular output through aorta. Cardiac output is the most important factor in cardiovascular system, because rate of blood flow through different parts of the body depends upon cardiac output.

DEFINITIONS AND NORMAL VALUES

Usually, cardiac output is expressed in three ways:

1. Stroke volume
2. Minute volume
3. Cardiac index.

However, in routine clinical practice, cardiac output refers to minute volume.

STROKE VOLUME

Stroke volume is the amount of blood pumped out by each ventricle during each beat.

Normal value: 70 mL (60 to 80 mL) when the heart rate is normal (72/minute).

MINUTE VOLUME

Minute volume is the amount of blood pumped out by each ventricle in one minute. It is the product of stroke volume and heart rate:

Minute volume = Stroke volume × Heart rate

Normal value: 5 L/ventricle/minute.

CARDIAC INDEX

Cardiac index is the minute volume expressed in relation to square meter of body surface area. It is defined as the amount of blood pumped out per ventricle/minute/square meter of the body surface area.

Normal value: 2.8 ± 0.3 L/square meter of body surface area/minute (in an adult with average body surface area of 1.734 square meter and normal minute volume of 5 L/minute).

EJECTION FRACTION

Ejection fraction is the fraction of end diastolic volume that is ejected out by each ventricle. Normal ejection fraction is 60% to 65%. Refer Chapter 91 for details.

CARDIAC RESERVE

Cardiac reserve is the maximum amount of blood that can be pumped out by heart above the normal value. Cardiac reserve plays an important role in increasing the cardiac output during the conditions like exercise. It is essential to withstand the stress of exercise.

Cardiac reserve is usually expressed in percentage. In a normal young healthy adult, the cardiac reserve is 300% to 400%. In old age, it is about 200% to 250%. It increases to 500% to 600% in athletes. In cardiac diseases, the cardiac reserve is minimum or nil.

VARIATIONS IN CARDIAC OUTPUT

PHYSIOLOGICAL VARIATIONS

1. *Age:* In children, cardiac output is less because of less blood volume. Cardiac index is more than that in adults because of less body surface area.
2. *Sex:* In females, cardiac output is less than in males because of less blood volume. Cardiac index is more than in males, because of less body surface area.
3. *Body build:* Greater the body build, more is the cardiac output.
4. *Diurnal variation:* Cardiac output is low in early morning and increases in day time. It depends upon the basal conditions of the individuals.
5. *Environmental temperature:* Moderate change in temperature does not affect cardiac output. Increase in temperature above 30°C raises cardiac output.
6. *Emotional conditions:* Anxiety, apprehension and excitement increases cardiac output about 50% to 100% through the release of catecholamines, which increase the heart rate and force of contraction.
7. *After meals:* During the first one hour after taking meals, cardiac output increases.
8. *Exercise:* Cardiac output increases during exercise because of increase in heart rate and force of contraction.
9. *High altitude:* In high altitude, the cardiac output increases because of increase in secretion of adrenaline. Adrenaline secretion is stimulated by hypoxia (lack of oxygen).
10. *Posture:* While changing from recumbent to upright position, the cardiac output decreases.
11. *Pregnancy:* During the later months of pregnancy, cardiac output increases by 40%.
12. *Sleep:* Cardiac output is slightly decreased or it is unaltered during sleep.

PATHOLOGICAL VARIATIONS

Increase in Cardiac Output

Cardiac output increases in the following conditions:

1. *Fever:* Due to increased oxidative processes
2. *Anemia:* Due to hypoxia
3. *Hyperthyroidism:* Due to increased basal metabolic rate.

Decrease in Cardiac Output

Cardiac output decreases in the following conditions:

1. *Hypothyroidism:* Due to decreased basal metabolic rate
2. *Atrial fibrillation:* Because of incomplete filling of ventricles
3. *Incomplete heart block with coronary sclerosis or myocardial degeneration:* Due to defective pumping action of the heart
4. *Congestive cardiac failure:* Because of weak contractions of heart
5. *Shock:* Due to poor pumping and circulation
6. *Hemorrhage:* Because of decreased blood volume.

DISTRIBUTION OF CARDIAC OUTPUT

The whole amount of blood pumped out by the right ventricle goes to lungs. But, the blood pumped by the left ventricle is distributed to different parts of the body.

Fraction of cardiac output distributed to a particular region or organ depends upon the metabolic activities of that region or organ.

Distribution of Blood Pumped out of Left Ventricle

Distribution of blood pumped out of left ventricle to different organs and the percentage of cardiac output are given in Table. 98.1. Heart, which pumps the blood to all other organs, receives the least amount of blood. Liver receives maximum amount of blood.

■ FACTORS MAINTAINING CARDIAC OUTPUT

Cardiac output is maintained (determined) by four factors:

1. Venous return
2. Force of contraction
3. Heart rate
4. Peripheral resistance.

■ 1. VENOUS RETURN

Venous return is the amount of blood which is returned to heart from different parts of the body. When it increases, the ventricular filling and cardiac output are increased. Thus, cardiac output is **directly proportional** to venous return, provided the other factors (force of contraction, heart rate and peripheral resistance) remain constant.

Venous return in turn, depends upon five factors:

i. Respiratory pump
ii. Muscle pump
iii. Gravity
iv. Venous pressure
v. Sympathetic tone.

i. *Respiratory Pump*

Respiratory pump is the respiratory activity that helps the return of blood, to heart during inspiration. It is also called **abdominothoracic pump.** During inspiration, thoracic cavity expands and makes the intrathoracic pressure more negative. It increases the diameter of inferior vena cava, resulting in increased venous return. At the same time, descent of diaphragm increases the intra-abdominal pressure, which compresses abdominal veins and pushes the blood upward towards the heart and thereby the venous return is increased (Fig. 98.1).

Respiratory pump is much stronger in forced respiration and in severe muscular exercise.

TABLE 98.1: Distribution of blood pumped out of left ventricle

Organ	Amount of blood (mL/ minute)	Percentage
Liver	1,500	30
Kidney	1,300	26
Skeletal muscles	900	18
Brain	800	16
Skin, bone and GI tract	300	6
Heart	200	4
Total	5,000	100

ii. *Muscle Pump*

Muscle pump is the muscular activity that helps in return of the blood to heart. During muscular activities, the veins are compressed or squeezed. Due to the presence of valves in veins, during compression the blood is moved towards the heart (Fig. 98.2). When muscular activity increases, the venous return is more.

When the skeletal muscles contract, the vein located in between the muscles is compressed. Valve of the vein

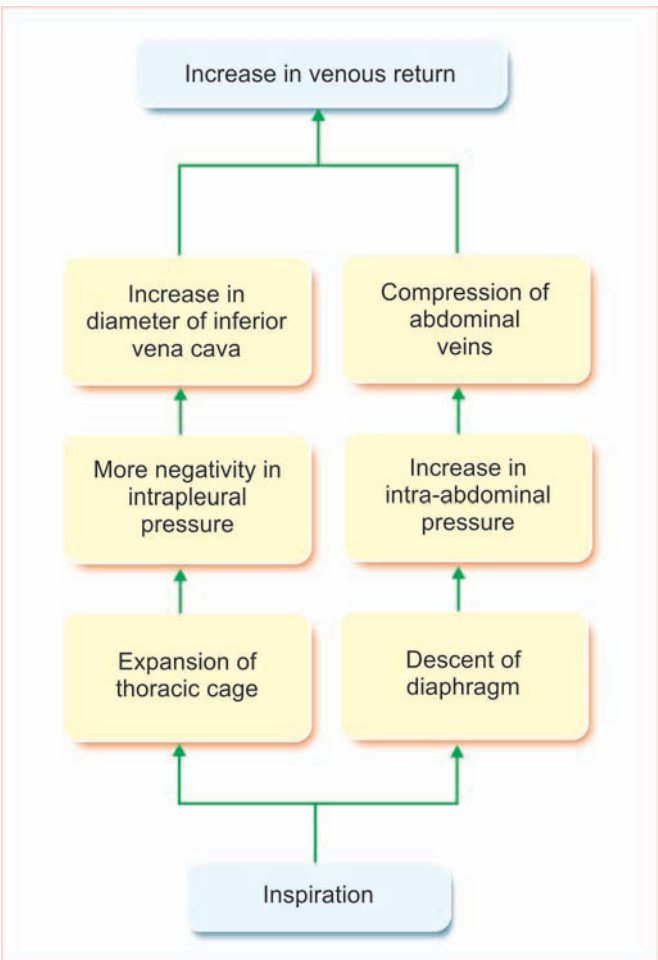

FIGURE 98.1: Effect of respiratory pump on venous return

proximal to the contracting muscles (Fig. 98.2 A) is opened and the blood is propelled towards the heart. Valve of the vein distal to the muscles is closed by the back flow of blood.

During relaxation of the muscles (Fig. 98.2 B), the valve proximal to muscles closes and prevents the back flow of blood. The valve distal to the muscles opens and allows the blood to flow upwards.

iii. *Gravity*

Gravitational force reduces the venous return. When a person stands for a long period, gravity causes pooling of blood in the legs, which is called **venous pooling.** Because of venous pooling, the amount of blood returning to heart decreases.

iv. *Venous Pressure*

Venous pressure also affects the venous return. Pressure in the venules is 12 to 18 mm Hg. In the smaller and larger veins, the pressure gradually decreases. In the great veins, i.e. inferior vena cava and superior vena cava, the pressure falls to about 5.5 mm Hg. At the junction of venae cavae and right atrium, it is about 4.6 mm Hg. Pressure in the right atrium is still low and it alters during cardiac action. It falls to zero during atrial diastole. This pressure gradient at every part of venous tree helps as a driving force for venous return.

v. *Sympathetic Tone*

Venous return is aided by sympathetic or vasomotor tone (Chapter 103), which causes constriction of venules. Venoconstriction pushes the blood towards heart.

■ 2. FORCE OF CONTRACTION

Cardiac output is **directly proportional** to the force of contraction, provided the other three factors remain constant. According to **Frank-Starling law,** force of contraction of heart is directly proportional to the initial length of muscle fibers, before the onset of contraction.

Force of contraction depends upon preload and afterload.

Preload

Preload is the stretching of the cardiac muscle fibers at the end of diastole, just before contraction. It is due to increase in ventricular pressure caused by filling of blood during diastole. Stretching of muscle fibers increases their length, which increases the force of contraction and cardiac output.

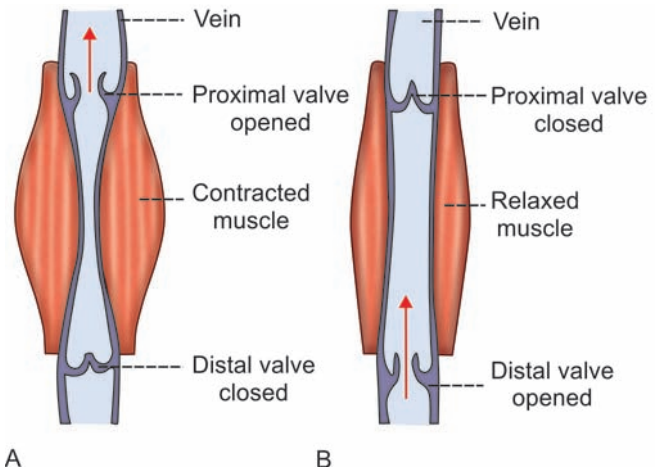

FIGURE 98.2: Mechanism of muscle pump. A. During contraction of the muscle; B. During relaxation of the muscle.

Thus, force of contraction of heart and cardiac output are **directly proportional** to preload.

Afterload

Afterload is the force against which ventricles must contract and eject the blood. Force is determined by the arterial pressure. At the end of isometric contraction period, semilunar valves are opened and blood is ejected into the aorta and pulmonary artery. So, the pressure increases in these two vessels. Now, the ventricles have to work against this pressure for further ejection. Thus, the afterload for left ventricle is determined by aortic pressure and afterload for right ventricular pressure is determined by pressure in pulmonary artery.

Force of contraction of heart and cardiac output are **inversely proportional** to afterload.

■ 3. HEART RATE

Cardiac output is **directly proportional** to heart rate provided, the other three factors remain constant. Moderate change in heart rate does not alter the cardiac output. If there is a marked increase in heart rate, cardiac output is increased.

If there is marked decrease in heart rate, cardiac output is decreased.

■ 4. PERIPHERAL RESISTANCE

Peripheral resistance is the resistance offered to blood flow at the peripheral blood vessels. Peripheral resistance is the resistance or load against which the heart has to pump the blood. So, the cardiac output is **inversely proportional** to peripheral resistance.

Resistance is offered at **arterioles** so, the arterioles are called **resistant vessels.** In the body, maximum peripheral resistance is offered at the **splanchnic region.** Other details of peripheral resistance are given in Chapter 102.

MEASUREMENT OF CARDIAC OUTPUT

Cardiac output is measured by direct methods and indirect methods. Direct methods are used only in animals. Indirect methods are used both in animals and human beings.

MEASUREMENT OF CARDIAC OUTPUT BY DIRECT METHODS

Direct methods used to measure cardiac output in animals:

1. By using cardiometer
2. By using flowmeter.

1. *By Using Cardiometer*

This is described in Chapter 91.

2. *By Using Flowmeter*

Mechanical flowmeter

Mechanical flowmeter is used to measure cardiac output or the amount of blood flow to any organ. It is used only in animals. It has an inlet, a measuring device in the middle and an outlet. Aorta or the artery entering any organ is cut. Inlet and outlet of the flowmeter are inserted into cut ends of the blood vessel. When the blood passes through the flowmeter, the measuring device determines the amount of blood flow (Fig. 99.1).

Electromagnetic flowmeter

Principle: Principle of this flowmeter is to develop an electromagnetic field by means of two coils of wire. If the coils are placed on either side of a blood vessel, the electromagnetic field is produced around the vessel. When blood flows through the vessel, there is an alteration in the electromagnetic field. By using appropriate electrodes, the changes in the magnetic field can be detected. By connecting electrodes to an electronic device, velocity of blood flow is determined on the basis of changes in the magnetic field. From the velocity of blood flow, the volume of blood flow is calculated.

Instrument: An **electromagnetic probe** is devised with the electromagnetic coils and the electrodes. The probe has a cleft and it is fixed in such a way that the intact blood vessel passes through the cleft. The probe almost encircles the blood vessel. The probe is connected to the electronic device to measure the volume of blood flow.

Advantage of this flowmeter is that the blood vessel need not be cut open.

Ultrasonic Doppler flowmeter

Principle: Ultrasound is the sound with very high frequency. It is very much beyond the audible range of human ears. The waves of the **ultrasound** are transmitted through a blood vessel. These sound waves are called **transmitted waves.** While passing through the blood vessels, the sound waves hit against the blood cells, particularly the red blood cells and are reflected back. Frequency of the **reflected waves** is different from that of the transmitted waves. This effect is called the **Doppler effect** (named after the discoverer **Johann Christian Doppler**). Alteration in the frequency of reflected waves depends upon the velocity of blood flowing through the blood vessel. By detecting the differences between frequencies of transmitted and reflected sound waves, the velocity of blood flow and then the volume of blood flow are determined.

Instrument: Ultrasonic device has piezoelectric crystals, which produce the **ultrasonic waves** and act as sensors to receive the reflected waves. This device is connected to an electronic equipment, which detects the difference between the frequencies of transmitted and reflected waves and thereby, determines the velocity of blood flow and the volume of blood flow.

Disadvantages of Direct Methods

i. Direct methods to measure cardiac output can be used only in animals
ii. Blood vessel has to be cut open at the risk of animal's life
iii. While using cardiometer, the size of the cardiometer must be suitable for the size of the heart
iv. While using mechanical flowmeter, diameter of inlet and the outlet of the flowmeter must be equivalent to the diameter of the blood vessel.

MEASUREMENT OF CARDIAC OUTPUT BY INDIRECT METHODS

Several methods are available to measure cardiac output. Each method has got its own advantages and disadvantages. Generally, the safe and accurate method is preferred. In view of safety, always non-invasive methods are preferred. The invasive method

is also accepted provided, it gives accurate results. In addition to providing measurement of cardiac output, nowadays the methods are expected to provide other hemodynamic data and some useful information about the structure and movements of valves and chambers of the heart.

Invasive and Non-invasive Methods

Invasive method refers to a procedure which involves invasion or penetration of healthy tissues, organs or parts of the body, by means of perforation, puncture, incision, injection or catheterization. Non-invasive method means the procedure that does not involve invasion or penetration of tissues, organs or parts of the body.

Different Indirect Methods

Indirect methods used to measure cardiac output:

1. By using Fick principle
2. Indicator (dye) dilution technique
3. Thermodilution technique
4. Ultrasonic Doppler transducer technique
5. Doppler echocardiography
6. Ballistocardiography.

1. By Using Fick Principle

Adolph Fick described Fick principle in 1870. According to this principle, the amount of a substance taken up by an organ (or by the whole body) or given out in a unit of time is the product of amount of blood flowing through the organ and the arteriovenous difference of the substance across the organ.

Amount of substance taken or given = Amount of blood flow/minute × Arteriovenous difference

For example,

Amount of blood flowing through lungs is 5,000 mL/minute
O_2 content in arterial blood = 20 mL/100 mL of blood
O_2 content in venous blood = 15 mL/100 mL of blood

Amount of oxygen moved from lungs to blood = Amount of blood flow/minute × Arteriovenous difference of O_2

$$= 5{,}000 \times \frac{20 - 15}{100}$$

$$= 5{,}000 \times \frac{5}{100} = 250$$

Amount of oxygen moved from lungs to blood = 250 mL/minute

Modification of Fick principle to measure cardiac output

Fick principle is modified to measure the cardiac output or a part of cardiac output (amount of blood to an organ). Thus, cardiac output or the amount of blood flowing through an organ in a given unit of time is determined by the formula:

$$\text{Cardiac output} = \frac{\text{Amount of substance taken or given by the organ/minute}}{\text{Arteriovenous difference of the substance across the organ}}$$

By modifying Fick principle, cardiac output is measured in two ways:

i. By using oxygen consumption
ii. By using carbon dioxide given out.

Measurement of Cardiac Output by Using Oxygen Consumption

Fick principle is used to measure the cardiac output by determining the amount of oxygen consumed in the body in a given period of time and dividing this value by the arteriovenous difference across the lungs.

$$\text{Cardiac output} = \frac{O_2 \text{ consumed (in mL/minute)}}{\text{Arteriovenous } O_2 \text{ difference}}$$

Oxygen consumption: Amount of oxygen consumed is measured by using a **respirometer** or **BMR apparatus (Benedict Roth apparatus).**

Oxygen content in arterial blood: Blood is collected from any artery to determine the oxygen content in arterial blood. Oxygen content is determined by blood gas analysis.

Oxygen content in venous blood: Only mixed venous blood is used to determine the oxygen content of venous blood, since oxygen content is different in different veins. Mixed venous blood is collected from right atrium or pulmonary artery. It is done by introducing a **catheter** through basilar vein of forearm. Oxygen is determined from this blood by **blood gas analysis** (Fig. 98.3).

Calculation

For example, in a subject, the following data are obtained:

O_2 consumed (by lungs) = 250 mL/minute
O_2 content in arterial blood = 20 mL/100 mL of blood
O_2 content in venous blood = 15 mL/100 mL of blood

$$\text{Cardiac output} = \frac{O_2 \text{ consumed (in mL/minute)}}{\text{Arteriovenous } O_2 \text{ difference}}$$

$$= \frac{250}{5/100} = \frac{250 \times 100}{5}$$

$$= 5{,}000 \text{ mL/minute}$$

5 mL of oxygen is taken by 100 mL of blood while passing through the lungs. Thus, 250 mL of oxygen is taken by 5,000 mL of blood. Since cardiac output is equivalent to the amount of blood passing through pulmonary circulation, the cardiac output = 5 L/minute.

Measurement of Cardiac Output by Using Carbon Dioxide

Cardiac output is also measured by knowing the arteriovenous difference of carbon dioxide and amount of carbon dioxide given out (removed) by lungs (Fig. 98.4). Thus:

$$\text{Cardiac output} = \frac{CO_2 \text{ evolved (in mL/minute)}}{\text{Arteriovenous } CO_2 \text{ difference}}$$

Calculation

For example, in a subject
CO_2 removed by lungs = 200 mL/minute
CO_2 content in arterial blood = 56 mL/100 mL of blood
CO_2 content in venous blood = 60 mL/100 mL of blood

$$\text{Cardiac output} = \frac{200}{60 - 56 \text{ mL}/100 \text{ mL}}$$

$$= \frac{200 \times 100}{4}$$

$$= 5{,}000 \text{ mL} = 5 \text{ L/minute}$$

Since cardiac output is equal to the amount of blood passing through lungs (pulmonary circulation), the cardiac output = 5 L/minute

Nitrous oxide is also used to measure cardiac output by applying Fick principle.

Advantage of measurement of cardiac output by Fick principle

The results are accurate.

Disadvantage

Fick principle is an invasive method and involves the insertion of catheter through subject vein.

2. Indicator (Dye) Dilution Method

Indicator dilution technique is described in detail in Chapter 6. Marker substance used to measure cardiac output is lithium chloride.

Advantage

The results are accurate.

Disadvantage

Indicator dilution method is an invasive method and involves injection of marker substance.

3. Thermodilution Technique

Cardiac output can also be measured by thermodilution technique or **thermal indicator method.** This method is the modified indicator dilution method. It is the popular method to measure cardiac output.

In this method, a known volume of cold sterile solution is injected into the right atrium via inferior vena

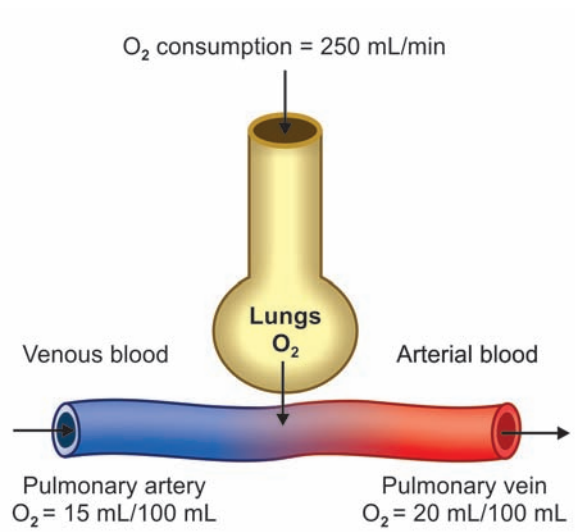

FIGURE 98.3: Oxygen consumption

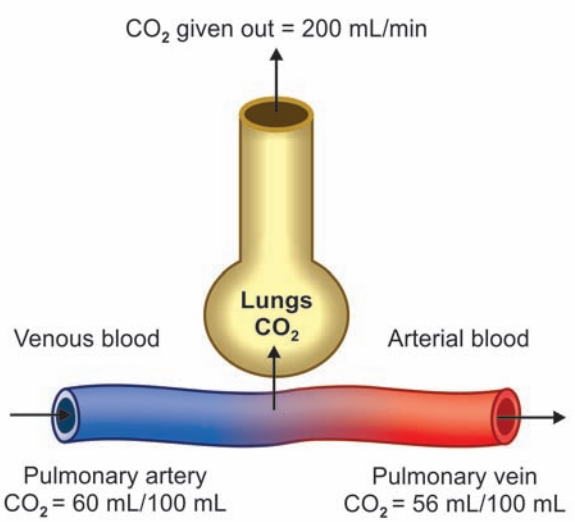

FIGURE 98.4: Carbon dioxide given out

cava by using a catheter. Cardiac output is measured by determining the resultant change in the blood temperature in pulmonary artery. For this purpose, two **thermistors (temperature transducers)** are used. One of them is placed in the inferior vena cava and the second one is placed in pulmonary artery. A pulmonary artery catheter is used to place the thermistors in their positions.

A known quantity of cold saline or cold dextrose solution is injected into inferior vena cava. Thermistors determine the temperature of blood entering the heart via inferior vena cava and temperature of blood leaving the heart via pulmonary artery. From the values of temperature, cardiac output is measured by applying indicator dilution technique.

Advantages

Results are accurate in this method. Even low cardiac output can be measured. Saline is also harmless. Catheter is also used to determine hemodynamic pressures and to collect mixed venous blood.

Disadvantage

Thermodilution technique is an invasive method and it requires catheterization.

Continuous cardiac output measurement catheter

Cardiac output can be measured continuously by using a modified pulmonary artery catheter called **continuous cardiac output measurement catheter** (CCO catheter). CCO catheter works on thermodilution principle. Instead of injecting cold saline, a heating filament which delivers heat directly to blood is used. The heating filament is fitted to the ventricular portion of the catheter. Cardiac output is measured as done in thermodilution technique. This method is commonly used in intensive care unit (ICU).

4. *Esophageal Ultrasonic Doppler Transducer Technique*

Esophageal ultrasonic doppler transducer technique involves insertion of a flexible probe into midthoracic part of esophagus. A pulse wave ultrasonic **Doppler transducer** is fixed at the tip of the probe. This transducer calculates the velocity of blood flow in descending aorta (refer ultrasonic Doppler flow meter for details). The diameter of aorta is determined by echocardiography (see below). Cardiac output is calculated by using the values of velocity of blood flow and diameter of aorta.

Advantages

The cardiac output can be measured continuously. This can be used during cardiac surgery.

Disadvantages

Esophageal ultrasonic doppler transducer is an invasive method and results are less accurate.

5. *Doppler Echocardiography*

Doppler echocardiography is a method for detecting the direction and velocity of moving blood within the heart. This is also a popular method to measure cardiac output.

Echocardiography is a **diagnostic procedure,** which uses the ultrasound waves (more than 20,000 Hz) to produce the image of the heart muscle. Ultrasound waves which reflect or echo off the heart can determine the size, shape, movement of the valves and chambers and the flow of blood through the heart.

During echocardiographic examination, the patient lies bare-chested on the examination table. A special gel is spread over the chest to help the transducer make good contact and slide smoothly over the skin. The transducer is a small hand operated device, which is attached to machine by a flexible cable. The transducer is placed against the chest. The transducer produces and directs ultrasound waves into the chest. Some of the waves get reflected (or echoed) back to the transducer. The reflection of sound waves depends upon the type of tissues and blood. The reflected sound waves are received by the transducer and translated into an image of the heart and displayed on a monitor or recorded on paper or tape.

Echocardiography may also show the abnormalities in functioning of heart valves or damage to the myocardium from an earlier heart attack.

When **Doppler principle** is applied in echocardiography, it enables the determination of direction, rate and other characteristics of blood flow. Doppler echocardiography is based upon the changes in frequency of the reflected sound waves from red blood cells (refer ultrasonic Doppler flow meter for details).

By Doppler echocardiography, the velocity of blood flow through aortic valve is determined. The diameter of the aorta is determined by simple echocardiography. From these values, cardiac output is calculated.

Advantage

Doppler echocardiography is a non-invasive technique. It also provides other useful information about the structures and movements of valves and chambers of heart.

Disadvantage

Doppler echocardiography method provides less accurate results. It requires well trained operator.

6. *Ballistocardiographic Method*

Ballistocardiography is the technique to record the movements of the body caused by **ballistic recoil,** associated with contraction of heart and ejection of blood. It is based on **Newton's third law of motion** (for every action there is an equal and opposite reaction). When heart pumps blood into aorta and pulmonary artery, a recoiling force is exerted against heart and the body. It is similar to that of ballistic recoil when a bullet is fired from a riffle.

Pulsations due to this ballistic recoil can be recorded graphically by making the subject to lie on a suspended bed, movable in the long axis of the body. The cardiac output is determined by analyzing the graph obtained.

Advantage

The only advantage of ballistocardiography is that it is a non-invasive method.

Disadvantage

Ballistocardiography is not a commonly used technique because it involves cumbersome procedures for calibrating the equipment and analyzing the graph. It also does not provide accurate results.

■ CARDIAC CATHETERIZATION

■ DEFINITION

Catheter is a thin radiopaque tube, made up of elastic web, rubber, plastic, glass or metal. Cardiac catheterization is an invasive procedure in which a catheter is inserted **intravascularly** into any chamber of the heart or a blood vessel.

Cardiac catheterization is helpful to study the different variables of hemodynamics, both in normal and diseased states. Cardiac catheterization was discovered by a German medical student Werner Forsmann, who practiced this technique first on himself.

■ CONDITIONS WHEN CARDIAC CATHETERIZATION IS PERFORMED

Cardiac catheterization is generally performed:

1. When clinical assessments indicate rapid deterioration of patient's health and immediate treatment. This is the most common condition when cardiac catheterization is needed.
2. Whenever there is a need to confirm the suspected cardiac disease of a patient
3. Whenever there is need to determine anatomical and physiological status of heart and blood vessels.

■ PROCEDURE

Cardiac catheterization is performed by insertion of catheter into the peripheral blood vessel through skin, by needle puncture. This procedure is called **percutaneous insertion** of catheter.

Left Heart Catheterization

Left heart catheterization is done by passing a catheter through femoral artery, brachial artery or axillary artery. Catheter is guided into left ventricle under fluoroscopic observation via aorta. From left ventricle, the catheter is advanced into left atrium.

In patients with aortic stenosis or **prosthetic** (artificial) **valve,** the direct left ventricular puncture is performed. Under local anesthesia, a needle with a catheter is inserted through the thoracic wall at the level of apex beat. When the needle enters left ventricle, the catheter is advanced through the needle into left ventricle and later the needle is removed.

Latest technology includes catheterization through radial artery, which is called **transradial catheterization.**

Right Heart Catheterization

Right heart catheterization is usually performed by venous puncture via femoral vein. Catheter can also be introduced via internal jugular vein, subclavian vein or medial vein. Under **fluoroscopic observation,** the catheter is advanced into right atrium. From right atrium, it can be guided into right ventricle and also into pulmonary artery.

■ USES OF CARDIAC CATHETERIZATION

Cardiac catheterization is useful for both diagnostic and therapeutic purposes. It gives crucial information about the need for cardiac surgery, coronary angioplasty and other **therapeutic procedures.** It also gives information about anticipated risks and reversibility in the patient's condition during **cardiac surgery** or other **therapeutic interventions.**

Diagnostic Uses of Cardiac Catheterization

1. Blood samples are collected during cardiac catheterization to measure oxygen saturation and the concentration of ischemic metabolites like lactate
2. Cardiac output is measured by using Fick principle, indicator dilution technique or thermodilution technique during cardiac catheterization

3. Angiography is done with the help of catheterization. Angiography or **arteriography** is the diagnostic or **therapeutic radiography (imaging technique),** in which the fluoroscopic picture is used to visualize the blood filled structures like cardiac chambers, arteries and veins of heart and other blood vessels, by using a **radiopaque contrast** medium. It is used to determine the obstruction or occlusion of coronary blood vessels or other blood vessels. It is also used to determine the anomalies of coronary blood vessels.
4. Various pressures are determined by attaching a pressure transducer to the cardiac catheter.

   Right heart catheterization is used to measure:
   i. Right atrial pressure
   ii. Right ventricular pressure
   iii. Pulmonary arterial pressure
   iv. Pulmonary capillary wedge pressure.

   Left heart catheterization is used to measure:
   i. Aortic pressure
   ii. Left ventricular pressure
   iii. Left atrial pressure.

Therapeutic Uses of Cardiac Catheterization – Interventional Cardiology

Cardiac catheterization is performed for various therapeutic procedures. Interventional cardiology is a branch of cardiology that deals with performance of traditional surgical procedures by cardiac catheterization. It helps in:

1. Thrombolysis
2. Percutaneous transluminal coronary angioplasty
3. Laser coronary angioplasty
4. Catheter ablation.

1. *Thrombolysis*

Thrombolysis **(reperfusion therapy)** is the procedure used to break up and dissolve a **thrombus** (clot) in the coronary artery of patient affected by acute myocardial infarction due to coronary thrombus. Cardiac catheterization is used for intracoronary administration of **thrombolytic agents** which cause thrombolysis.

Thrombolytic agents:
i. Tissue plasminogen activator
ii. Streptokinase
iii. Urokinase.

All these thrombolytic agents convert plaminogen into plasmin, which degrades fibrin in clot and restore normal blood flow.

2. *Percutaneous transluminal coronary angioplasty*

Coronary angioplasty means the correction of narrowed or totally obstructed lumen of blood vessels by mechanical methods. In percutaneous transluminal coronary angioplasty (PTCA), a narrowed coronary artery is dilated by inflating a balloon attached to the tip of catheter that is introduced into the blood vessel. Sometimes, a **stent** (expandable wire mesh) is introduced into the corrected blood vessel by the catheter to keep the vessel in dilated state.

3. *Laser coronary angioplasty*

Catheter is also used to emit laser (Light amplification by stimulated emission of radiation) energy. Laser energy which is emitted into the occluded coronary artery vaporizes the atherosclerotic plaque in the diseased vessel. This technique is called laser coronary angioplasty.

4. *Catheter ablation*

Catheter ablation is the procedure to destroy (ablate) an area of cardiac tissue that blocks the electrical pathway or produces abnormal electrical impulses, resulting in cardiac arrhythmia such as supraventricular tachycardia (SVT) or Wolff-Parkinson-White syndrome (Chapter 96).

It involves advancing a catheter (with electrodes attached to its tip) towards the heart via either femoral vein or subclavian vein. When the catheter enters right atrium, arrhythmia is induced. Then the electrodes at the tip of catheter record the electrical potentials. By using these recordings, the area of faulty electrical site is pinpointed. This procedure is called **electrical mapping.**

Once the damaged site is confirmed, **radiofrequency energy** is used to destroy the small amount of tissue that disturbs the electrical flow through the heart. Thus, the healthy heart rhythm is restored. Tissue is also destroyed by freezing with intense cold **(cryoablation).**

Heart-lung Preparation

Chapter 99

- INTRODUCTION
- PROCEDURE
- USES OF HEART-LUNG PREPARATION

INTRODUCTION

Heart-lung preparation is an experimental set up, devised by **Starling.** It is used to demonstrate the effects of various factors on the activities of heart, particularly heart rate and cardiac output. This preparation is also used to record the cardiac function curves.

PROCEDURE

Heart-lung preparation is usually done in dogs. After giving **anesthesia,** neck of the dog is opened and a **tracheal cannula** is inserted into the trachea. Tracheal cannula is connected to a **respiratory pump,** so that respiration in the animal is controlled artificially, to avoid any disturbance during the experimental procedure (Fig. 99.1).

Then, chest is opened and an **arterial cannula** is inserted into one of the branches of aorta. All the other branches from arch of aorta and descending aorta are ligated. Arterial cannula is connected to two instruments:

1. **Mercury manometer** to measure the arterial blood pressure
2. **Air bottle,** which provides elasticity artificially (as in the case of arterial wall).

Thus, the blood ejected from left ventricle passes into air bottle through the arterial cannula and rubber tubes. From the air bottle, the blood is diverted through a tube which provides **artificial resistance.** Air bottle is also connected to a **pressure bottle.** Pressure bottle is attached to a pressure pump. This pump is used to maintain the pressure within the set up.

Artificial resistance is offered by applying pressure surrounding the **resistance tube.** Resistance tube is also connected to a manometer.

After passing through the resistance tube, blood is allowed to flow through a **warming glass coil,** which is kept inside a water bath with a heater. Temperature of water bath is controlled, so that the temperature of blood could be maintained.

Warming coil is connected to a **venous reservoir** through a flowmeter, which determines the amount of blood flow (cardiac output). Venous reservoir is connected to superior vena cava by a rubber tube. A screw type clamp is fitted to the rubber tube. This clamp is used to adjust the amount of blood returning to heart (venous return). A **thermometer** is also fitted to the tube to note the temperature of blood.

A third mercury manometer is connected to the inferior vena cava. It is used to determine the venous pressure. A cardiometer is fitted to the ventricle. This cardiometer is connected to a recording device like **Marey tambour** or **polygraph,** to record the ventricular volume changes.

Pulmonary circulation is kept intact for continuous oxygenation of blood.

USES OF HEART-LUNG PREPARATION

Thus, in this set up, the heart works as an isolated organ. So, the effects of various factors can be demonstrated on the activities of heart, like heart rate, ventricular volume and cardiac output.

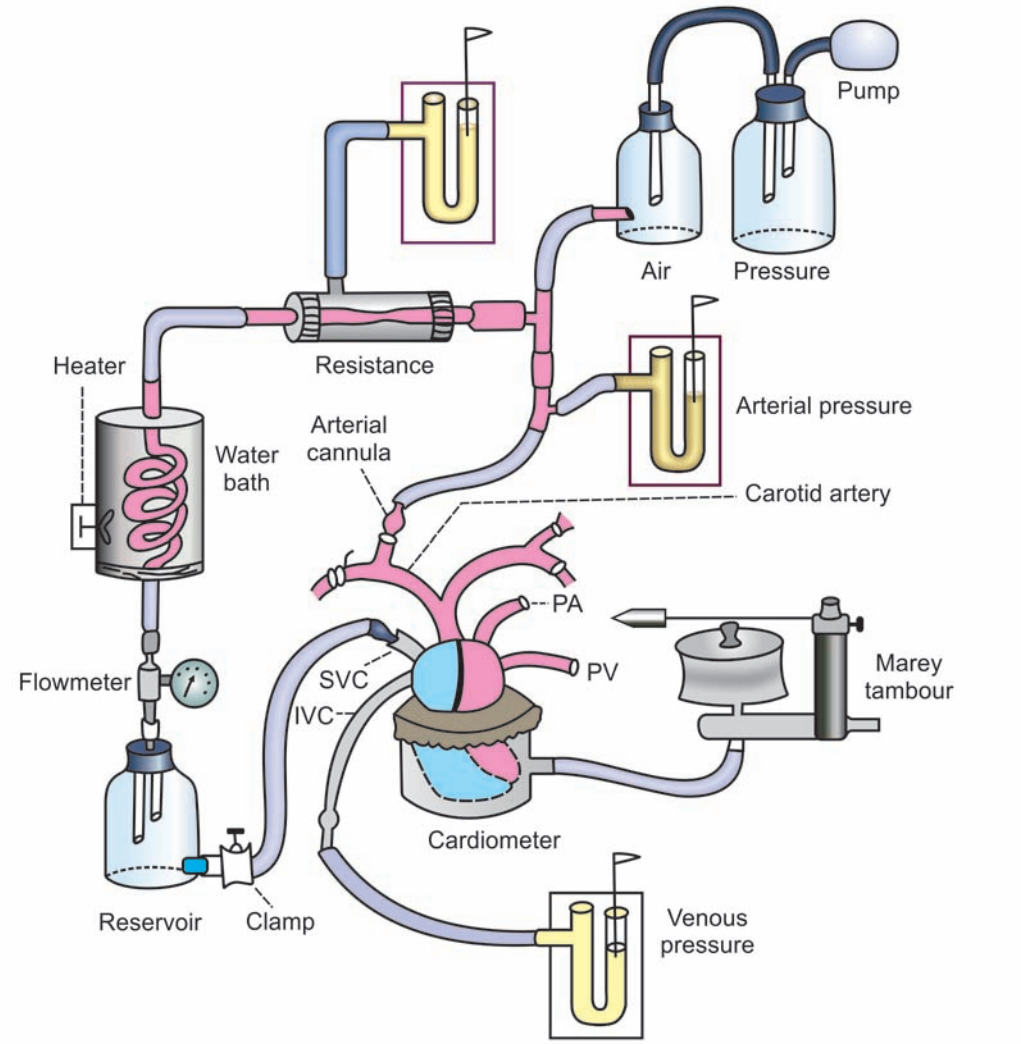

FIGURE 99.1: Heart-lung preparation.
SVC = Superior vena cava, IVC = Inferior vena cava, PA = Pulmonary artery, PV = Pulmonary vein.

Examples

1. When venous return decreases, stroke volume decreases
2. When venous return increases, stroke volume increases
3. When resistance increases, cardiac output decreases
4. When resistance decreases, cardiac output increases
5. Heart-lung preparation is also used to record two types of cardiac function curves:
   i. Cardiac output curves
   ii. Venous return curves.

Though the cardiac function curves are obtained in experiments using the animals, these curves represent the functions of the ventricles in human heart also (Chapter 100).

Cardiac Function Curves

Chapter 100

- INTRODUCTION
- CARDIAC OUTPUT CURVES
  - NORMAL CARDIAC OUTPUT CURVES
  - FACTORS AFFECTING CARDIAC OUTPUT CURVES
  - EFFECT OF EXTRACARDIAC PRESSURE ON CARDIAC OUTPUT CURVE
- VENOUS RETURN CURVES
- ANALYSIS OF CARDIAC FUNCTION CURVES
  - COUPLING OF CARDIAC AND VASCULAR FUNCTIONS

INTRODUCTION

Cardiac function curves are **Frank-Starling curves,** which demonstrate the capacity of ventricles to pump blood and to maintain blood circulation throughout the body. Most of the cardiac function curves are obtained from animal experiments, by using heart-lung preparation. However, these curves are considered to represent the functions of ventricles in human heart.

Cardiac function curves are of two types:

1. Cardiac output curves
2. Venous return curves.

CARDIAC OUTPUT CURVES

Cardiac output curves are the curves that show the relationship between cardiac output and right atrial pressure. Right atrial pressure, in turn, depends upon venous return.

NORMAL CARDIAC OUTPUT CURVES

Normally, left ventricular output is 5 L/minute, when the pressure in right atrium is 2 mm Hg. When the atrial pressure rises between 4 and 8 mm Hg, the left ventricular output also increases. It increases to about two and a half times of normal (basal) output, i.e. the output increases to about 13 to 14 L/minute. This is the maximum limit for increase in cardiac output. Further increase in right atrial pressure does not increase the ventricular output and the curve shows a **plateau** (Fig. 100.1).

Right ventricular output is 5 L/minute, when the right atrial pressure is zero. This reaches the maximum, i.e. 13 to 14 L/minute when the atrial pressure increases between 2 and 4 mm Hg (Fig. 100.1).

Thus, the cardiac output curves demonstrate that cardiac output is directly proportional to atrial pressure up to a certain extent (as explained above).

Plateau of the curve shows that the heart can control the output by itself if the atrial pressure rises beyond +8 mm Hg. It is due to the fact that in normal conditions, venous return is decreased when atrial pressure raises above +8 mm Hg.

FACTORS AFFECTING CARDIAC OUTPUT CURVES

Shifting of cardiac output curve to left indicates increase in cardiac output and shifting to right indicates decrease in cardiac output. The conditions which shift the cardiac output curve to left or right are discussed below:

Shift to Left

When there is an abnormal increase in the functioning of the heart **(hypereffective heart),** the cardiac output curve is shifted to left, indicating **increase in cardiac output.**

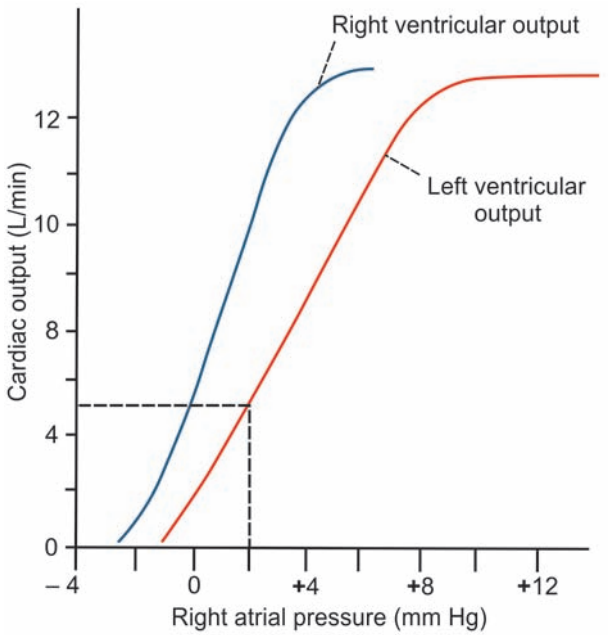

FIGURE 100.1: Normal cardiac output curves

Conditions when shift to left occurs

1. Combined stimulation of sympathetic and the parasympathetic nerves supplying the heart: It causes hyperexcitation of the heart, resulting in increased rate and force of contraction. The cardiac output increases up to 25 L/minute (i.e. the plateau is shifted to left). Increase in output is about twice the maximum output in normal conditions (13 to 14 L/minute).
2. Hypertrophy of heart: It increases cardiac output up to 10 to 19 L/minute. It is because of increase in force of contraction.
3. Excitation (by cardiac nerves) of the heart along with hypertrophy of the ventricles: In this condition, the cardiac output is elevated above 35 L/minute. It occurs in Marathon runners. Increase in cardiac output is an important factor for prolonged running time of Marathon runners.

Shift to Right

When the functioning of heart decreases **(hypoeffective heart),** cardiac output curve is shifted to right, indicating **decrease in cardiac output.**

Conditions when shift to right occurs

1. Stimulation of parasympathetic nerve fibers of the heart
2. Inhibition of sympathetic nerves to heart
3. Myocardial infarction
4. Diseases of the valves in the heart
5. Congenital heart diseases.

■ EFFECT OF EXTRACARDIAC PRESSURE ON CARDIAC OUTPUT CURVE

Extracardiac pressure is the pressure outside the heart. Intrapleural pressure is the major extracardiac pressure. When it increases above the normal level, i.e. from –6 to –2 mm Hg or becomes positive, the venous return decreases, resulting in decrease in cardiac output. Cardiac output curve is shifted to right. It happens in opening of thoracic cage and in positive pressure breathing.

When the intrapleural pressure decreases, i.e. when it becomes more negative, the venous return increases and the cardiac output also increases. The curve is shifted towards left. It is common in **negative pressure breathing.**

Cardiac Tamponade

Cardiac tamponade is the mechanical compression of heart due to accumulation of fluid in **pericardial space.** In addition to intrapleural pressure, accumulation of fluid in pericardial space also increases the extracardiac pressure and compresses the heart. In cardiac tamponade, the cardiac output decreases and output curve is shifted to right.

■ VENOUS RETURN CURVES

Venous return curves are the curves which demonstrate the relationship between venous return (blood flow in vascular system) and right atrial pressure. Venous return curves are also called **systemic vascular function curves.**

Normally, 5 L of blood returns to heart every minute. When right atrial pressure increases, venous return decreases due to **backpressure.** When venous return decreases, the cardiac output also decreases (Fig. 100.2).

■ ANALYSIS OF CARDIAC FUNCTION CURVES

Relation of cardiac output and venous return with right atrial pressure is determined when cardiac output curves and venous return curves are merged together (Fig. 100.3).

■ COUPLING OF CARDIAC AND VASCULAR FUNCTIONS

Cardiac output represents cardiac function and venous return represents vascular function. Coupling or merging of cardiac output (cardiac function) curves and venous return (vascular function) curves shows that when venous return is normal (5 L/minute), the cardiac output as well as the right atrial pressure are normal.

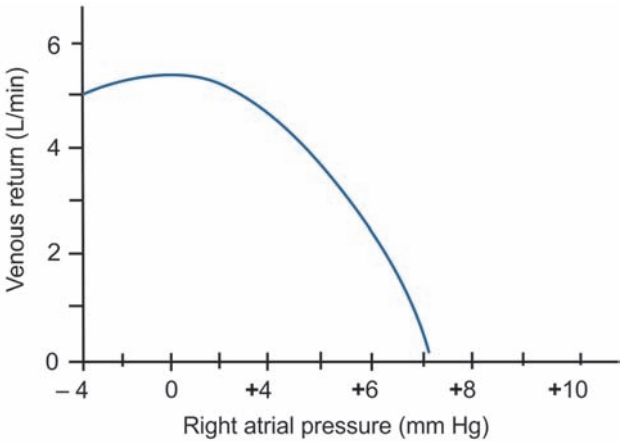

FIGURE 100.2: Venous return curve

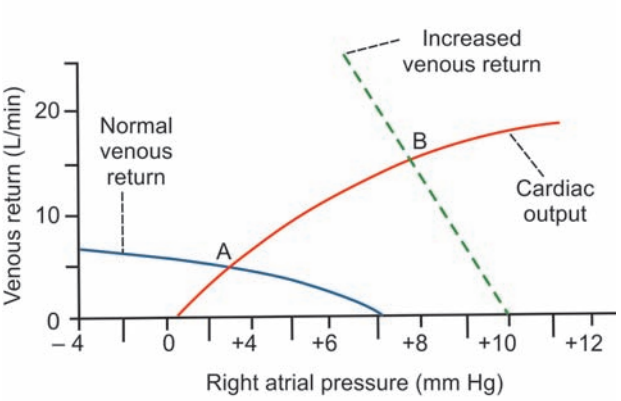

FIGURE 100.3: Analysis of cardiac function curves

Relation between cardiac output and venous return under normal conditions is represented by (A). When the venous return increases (B), the cardiac output also increases along with increase in right atrial pressure. Thus, any factor that alters venous return alters the cardiac output also.

Heart Rate

Chapter 101

- HEART RATE
  - NORMAL HEART RATE
  - TACHYCARDIA
  - BRADYCARDIA
- REGULATION OF HEART RATE
- VASOMOTOR CENTER – CARDIAC CENTER
  - VASOCONSTRICTOR AREA
  - VASODILATOR AREA
  - SENSORY AREA
- MOTOR (EFFERENT) NERVE FIBERS TO HEART
  - PARASYMPATHETIC NERVE FIBERS
  - SYMPATHETIC NERVE FIBERS
- SENSORY (AFFERENT) NERVE FIBERS FROM HEART
- FACTORS AFFECTING VASOMOTOR CENTER – REGULATION OF VAGAL TONE
  - IMPULSES FROM HIGHER CENTERS
  - IMPULSES FROM RESPIRATORY CENTERS
  - IMPULSES FROM BARORECEPTORS
  - IMPULSES FROM CHEMORECEPTORS
  - IMPULSES FROM RIGHT ATRIUM
  - IMPULSES FROM OTHER AFFERENT NERVES
  - BEZOLD-JARISCH REFLEX

HEART RATE

NORMAL HEART RATE

Normal heart rate is 72/minute. It ranges between 60 and 80 per minute.

TACHYCARDIA

Tachycardia is the increase in heart rate above 100/minute.

Physiological Conditions when Tachycardia Occurs

1. Childhood
2. Exercise
3. Pregnancy
4. Emotional conditions such as anxiety.

Pathological Conditions when Tachycardia Occurs

1. Fever
2. Anemia
3. Hypoxia
4. Hyperthyroidism
5. Hypersecretion of catecholamines
6. Cardiomyopathy
7. Diseases of heart valves.

■ BRADYCARDIA

Bradycardia is the decrease in heart rate below 60/minute.

Physiological Conditions when Bradycardia Occurs

1. Sleep
2. Athletes.

Pathological Conditions when Bradycardia Occurs

1. Hypothermia
2. Hypothyroidism
3. Heart attack
4. Congenital heart disease
5. Degenerative process of aging
6. Obstructive jaundice
7. Increased intracranial pressure.

Drugs which Induce Bradycardia

1. Beta blockers
2. Channel blockers
3. Digitalis and other antiarrhythmic drugs.

■ REGULATION OF HEART RATE

Heart rate is maintained within normal range constantly. It is subjected for variation during normal physiological conditions such as exercise, emotion, etc. However, under physiological conditions, the altered heart rate is quickly brought back to normal. It is because of the perfectly tuned regulatory mechanism in the body.

Heart rate is regulated by the nervous mechanism, which consists of three components:

A. Vasomotor center
B. Motor (efferent) nerve fibers to the heart
C. Sensory (afferent) nerve fibers from the heart.

■ VASOMOTOR CENTER – CARDIAC CENTER

Vasomotor center is the nervous center that regulates the heart rate. It is the same center in brain, which regulates the blood pressure. It is also called the cardiac center.

Vasomotor center is bilaterally situated in the **reticular formation** of medulla oblongata and lower part of pons.

Areas of Vasomotor Center

Vasomotor center is formed by three areas:

1. Vasoconstrictor area
2. Vasodilator area
3. Sensory area.

■ VASOCONSTRICTOR AREA – CARDIOACCELERATOR CENTER

Situation

Vasoconstrictor area is situated in the reticular formation of medulla in floor of IV ventricle and it forms the lateral portion of vasomotor center. It is otherwise known as **pressor area** or cardioaccelerator center.

Function

Vasoconstrictor area increases the heart rate by sending accelerator impulses to heart, through **sympathetic nerves.** It also causes constriction of blood vessels. Stimulation of this center in animals increases the heart rate and its removal or destruction decreases the heart rate.

Control

Vasoconstrictor area is under the control of **hypothalamus** and **cerebral cortex.**

■ VASODILATOR AREA – CARDIOINHIBITORY CENTER

Situation

Vasodilator area is also situated in the reticular formation of medulla oblongata in the floor of IV ventricle. It forms the medial portion of vasomotor center. It is also called **depressor area** or cardioinhibitory center.

Function

Vasodilator area decreases the heart rate by sending inhibitory impulses to heart through vagus nerve. It also causes dilatation of blood vessels. Stimulation of this area in animals with weak electric stimulus decreases the heart rate and stimulation with a strong stimulus stops the heartbeat. When this area is removed or destroyed, heart rate increases.

Control

Vasodilator area is under the control of **cerebral cortex** and **hypothalamus.** It is also controlled by the impulses from baroreceptors, chemoreceptors and other sensory impulses via afferent nerves.

■ SENSORY AREA

Situation

Sensory area is in the posterior part of vasomotor center, which lies in **nucleus of tractus solitarius** in medulla and pons.

Function

Sensory area receives sensory impulse via glossopharyngeal nerve and vagus nerve from periphery, particularly, from the baroreceptors. In turn, this area controls the vasoconstrictor and vasodilator areas.

■ MOTOR (EFFERENT) NERVE FIBERS TO HEART

Heart receives efferent nerves from both the divisions of autonomic nervous system. Parasympathetic fibers arise from the medulla oblongata and pass through vagus nerve. Sympathetic fibers arise from upper thoracic (T1 to T4) segments of spinal cord (Fig. 101.1).

■ PARASYMPATHETIC NERVE FIBERS

Parasympathetic nerve fibers are the cardioinhibitory nerve fibers. These nerve fibers reach the heart through the cardiac branch of vagus nerve.

Origin

Parasympathetic nerve fibers supplying heart arise from the **dorsal nucleus of vagus.** This nucleus is situated in the floor of fourth ventricle in medulla oblongata and is in close contact with vasodilator area.

Distribution

Preganglionic parasympathetic nerve fibers from dorsal nucleus of vagus reach the heart by passing through the main trunk of vagus and **cardiac branch of vagus.** After reaching the heart, preganglionic fibers terminate on postganglionic neurons. Postganglionic fibers from these neurons innervate heart muscle.

Most of the fibers from right vagus terminate in sinoatrial (SA) node. Remaining fibers supply the atrial muscles and atrioventricular (AV) node. Most of the fibers from left vagus supply AV node and some fibers supply the atrial muscle and SA node.

Ventricles do not receive the vagus nerve supply. Few fibers are located in the bases of ventricles, but the functions of these nerve fibers are not known.

Function

Vagus nerve is cardioinhibitory in function and carries inhibitory impulses from vasodilator area to the heart.

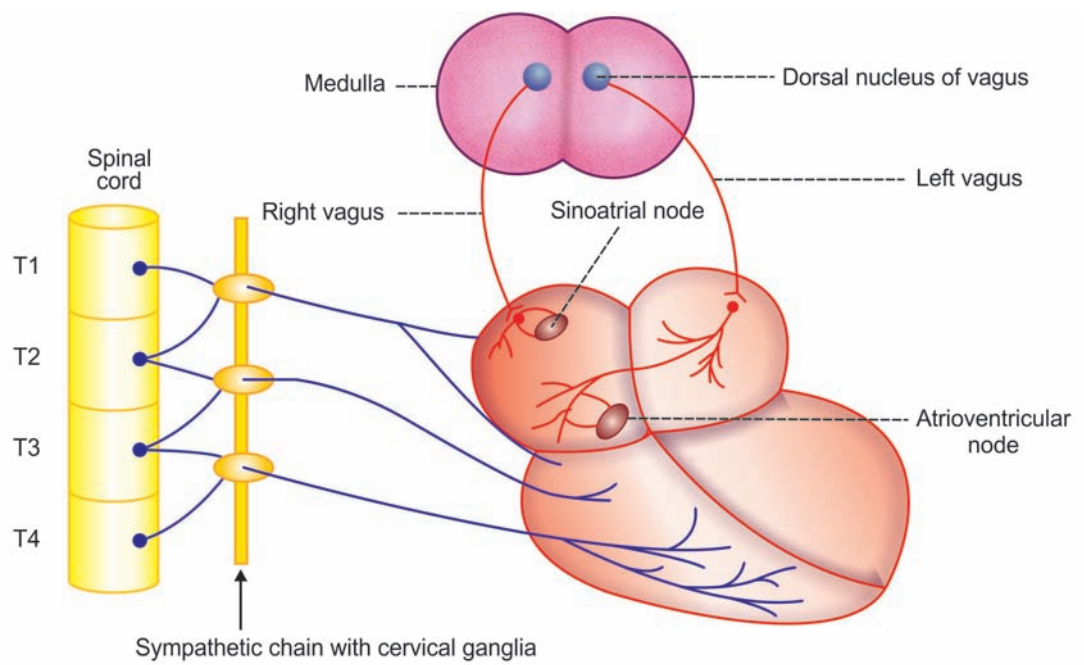

FIGURE 101.1: Nerve supply to heart

Vagal Tone

Vagal tone is the continuous stream of inhibitory impulses from vasodilator area to heart via vagus nerve. Heart rate is kept under control because of vagal tone.

These impulses reach the heart and exert inhibitory effect on heart. Heart rate is inversely proportional to vagal tone. In experimental animals (dog), removal of vagal input (by sectioning vagus) increases the heart rate. This proves the existence of vagal tone. Under resting conditions, vagal tone dominates sympathetic tone (see below).

Impulses from different parts of the body regulate the heart rate through vasomotor center, by altering the vagal tone. Vagal tone is also called **cardioinhibitory tone** or **parasympathetic tone.**

Effect of Stimulation of Vagus Nerve

Effect of stimulation of right vagus nerve – Vagal escape

Right vagus supplies mainly SA node. Stimulation of right vagus in experimental animals such as dog, with a weak stimulus causes reduction in heart rate and force of contraction. Stimulation with strong stimulus causes stoppage of heart due to inhibition of SA node. If the stimulus is continued for some time, the ventricle starts beating; but the rate of contraction is slower than before. This is because of vagal escape.

Vagal escape refers to escape of ventricle from inhibitory effect of vagal stimulation. If stimulation of vagus nerve is stopped, heart starts beating normally (Fig. 101.2).

Cause for vagal escape

Stimulation of right vagus stops the heartbeat due to inhibition of SA node and atria. However, ventricles are not supplied by vagus. So, the ventricles are not inhibited by vagal stimulation. Because of this, when stoppage of heart beat is continued for some time (by vagal stimulation), a part of ventricular musculature becomes pacemaker and starts producing impulses. It results in contraction of ventricles, which is called vagal escape.

Thus, vagal escape includes only ventricular contractions. However, the rhythmicity of ventricular muscle is less and it is about 20/minute.

Effect of stimulation of left vagus nerve – heart block

Left vagus supplies mainly the AV node. Stimulation of left vagus in dog with a weak stimulus causes a slight reduction in rate of ventricular contraction. Stimulation of left vagus causes inhibition of AV node. Because of inhibition of AV node, some of the impulses from SA node are not conducted to ventricles. This is called the **partial heart block.** The ratio between atrial contraction and ventricular contraction is 2 : 1, 3 : 1 or 4 : 1, depending upon the strength of stimulus.

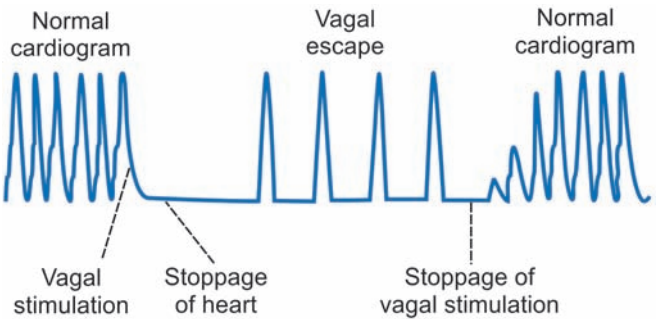

FIGURE 101.2: Effect of vagal stimulation on frog heart

Stimulation of left vagus with strong stimulus causes stoppage of ventricular contraction, which is called **complete heart block.** This is because of the complete inhibition of AV node. The prolongation of stimulation causes **idioventricular rhythm,** which is different from the rhythm of atrial contraction.

Mode of Action of Vagus Nerve

Vagus nerve inhibits the heart by secreting the neurotransmitter substance known as **acetylcholine.**

■ SYMPATHETIC NERVE FIBERS

Sympathetic nerve fibers supplying the heart have cardioacceleratory function.

Origin

Preganglionic fibers of the sympathetic nerves to heart arise from lateral gray horns of the first 4 thoracic (T1 to T4) segments of the spinal cord. These segments of the spinal cord receive fibers from vasoconstrictor area of vasomotor center.

Course and Distribution

Preganglionic fibers reach the superior, middle and inferior **cervical sympathetic ganglia** situated in the sympathetic chain. Inferior cervical sympathetic ganglion fuses with first thoracic sympathetic ganglion, forming **stellate ganglion.** From these ganglia, the postganglionic fibers arise.

Postganglionic fibers form three nerves:

1. Superior cervical sympathetic nerve, which innervates larger arteries and base of the heart
2. Middle cervical sympathetic nerve, which supplies the rest of the heart
3. Inferior cervical sympathetic nerve, which serves as **sensory (afferent) nerve** from the heart.

Function

Sympathetic nerves are cardioaccelerators in function and carry cardioaccelerator impulses from vasoconstrictor area to the heart.

Sympathetic Tone

Sympathetic tone or **cardioaccelerator tone** is the continuous stream of impulses produced by the vasoconstrictor area. Impulses pass through sympathetic nerves and accelerate the heart rate.

Under normal conditions, the vagal tone is dominant over sympathetic tone. Whenever vagal tone is reduced or abolished, the sympathetic tone becomes powerful. It is generally believed that the sympathetic tone does not play an important role in the regulation of cardiac function under resting physiological conditions. However, it plays a definite role in increasing the heart rate during emergency conditions.

Rate of contraction of a completely denervated heart of dog is higher than the rate of an innervated heart in resting conditions. This shows that under resting conditions, the vagal tone is **dominant** over sympathetic tone.

Effect of Stimulation of Sympathetic Nerves

Stimulation of sympathetic nerves increases the rate and force of contraction of heart. The effect depends upon the strength of stimulus.

Mode of Action of Sympathetic Nerves

Cardioacceleration by sympathetic stimulation is due to the release of neurotransmitter substance, **noradrenaline.**

■ SENSORY (AFFERENT) NERVE FIBERS FROM HEART

Afferent (sensory) nerve fibers from the heart pass through **inferior cervical sympathetic nerve.** These nerve fibers carry sensations of stretch and pain from the heart to brain via spinal cord.

■ FACTORS AFFECTING VASOMOTOR CENTER – REGULATION OF VAGAL TONE

Vasomotor center regulates the cardiac activity by receiving impulses from different sources in the body. After receiving the impulses from different sources, the vasodilator area alters the vagal tone and modulates the activities of the heart.

Various sources from which the impulses reach the vasomotor center:

■ 1. IMPULSES FROM HIGHER CENTERS

Vasomotor center is mainly controlled by the impulses from higher centers in cerebral cortex and hypothalamus.

Cerebral Cortex

Area 13 in cerebral cortex is concerned with emotional reactions of the body. During emotional conditions, this area sends inhibitory impulses to the vasodilator area. This causes reduction in vagal tone, leading to increase in heart rate.

Hypothalamus

Hypothalamus influences the heart rate via vasomotor center. Stimulation of posterior and lateral hypothalamic nuclei causes **tachycardia.** Stimulation of preoptic and anterior nuclei causes **bradycardia.**

■ 2. IMPULSES FROM RESPIRATORY CENTERS

In forced breathing, heart rate increases during inspiration and decreases during expiration. This variation is called **respiratory sinus arrhythmia.** This is common in some children and in some adults even during quiet breathing.

Sinus arrhythmia is due to the alteration of vagal tone because of impulses arising from respiratory centers during inspiration. These impulses inhibit the vasodilator area, resulting in decreased vagal tone and increased heart rate. During expiration, the respiratory center stops sending impulses to vasodilator center. Now, vagal tone increases, leading to decrease in heart rate.

■ 3. IMPULSES FROM BARORECEPTORS – MAREY REFLEX

Baroreceptors

Baroreceptors are the receptors which give response to change in blood pressure. These receptors are also called **pressoreceptors.**

Situation

Depending upon the situation, baroreceptors are divided into two types:

1. Carotid baroreceptors, situated in carotid sinus, which is present in the wall of internal carotid artery near the bifurcation of common carotid artery.
2. Aortic baroreceptors, situated in the wall of arch of aorta.

Nerve Supply

Carotid baroreceptors are supplied by **Hering nerve,** which is the branch of glossopharyngeal (IX cranial) nerve. Aortic baroreceptors are supplied by **aortic nerve,** which is a branch of vagus (X cranial) nerve (Fig. 101.3).

Nerve fibers from the baroreceptors reach the nucleus of tractus solitarius, which is situated adjacent to vasomotor center in medulla oblongata.

Function – Marey Reflex

Baroreceptors regulate the heart rate through Marey reflex. Stimulus for this reflex is increase in blood pressure.

Marey reflex is a **cardioinhibitory reflex** that decreases heart rate when blood pressure increases. Whenever blood pressure increases, the aortic and carotid baroreceptors are stimulated and stimulatory impulses are sent to nucleus of tractus solitarius via Hering nerve and aortic nerve (afferent nerves). Now, the nucleus of tractus solitarius stimulates vasodilator area, which in turn increases the vagal tone, leading to decrease in heart rate (Fig. 101.4). Marey reflex includes **aortic reflex** and **carotid sinus reflex.**

When pressure is less, the baroreceptors are not stimulated. So, no impulses go to nucleus of tractus solitarius. There are no inhibitory impulses to the heart and heart rate is not decreased. Thus, the heart rate is inversely proportional to blood pressure.

Marey law

According to Marey law, the pulse rate (which represents heart rate) is inversely proportional to blood pressure.

Baroreceptors induce the Marey reflex only during resting conditions. So, in many conditions such as exercise, there is an increase in both blood pressure and heart rate.

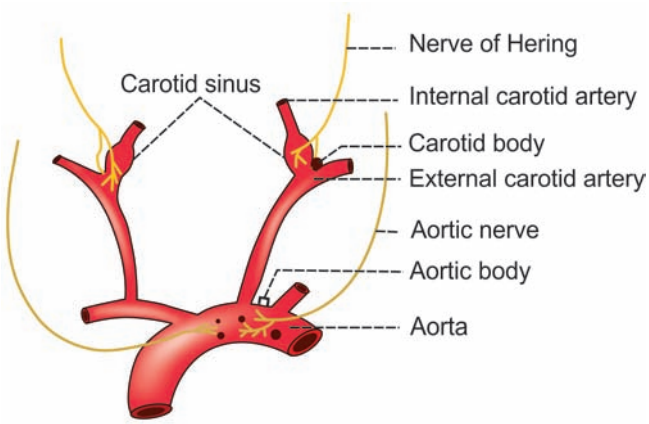

FIGURE 101.3: Nerve supply to baroreceptors and chemoreceptors

■ 4. IMPULSES FROM CHEMORECEPTORS

Chemoreceptors

Chemoreceptors are receptors giving response to change in chemical constituents of blood, particularly oxygen, carbon dioxide and hydrogen ion concentration.

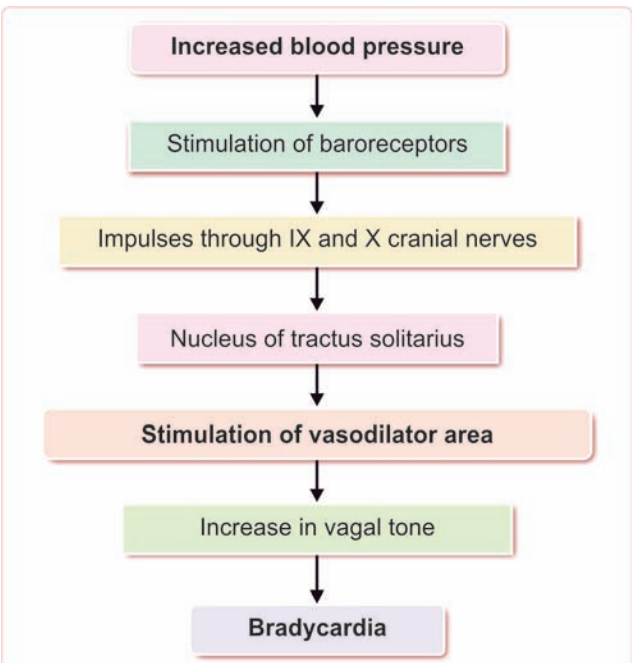

FIGURE 101.4: Marey (cardioinhibitory) reflex

Situation

Peripheral chemoreceptors are situated in the carotid body and aortic body, adjacent to baroreceptors.

Structure

Chemoreceptors are made up of two types of cells, type I or **glomus cells** and type II or **sustentacular cells.** Glomus cells have afferent nerve endings, which are stimulated by hypoxia. Type II cells are **glial cells** and provide support for type I cells.

Nerve Supply

Chemoreceptors in the carotid body are supplied by **Hering nerve,** which is the branch of glossopharyngeal nerve. Chemoreceptors in the aortic body are supplied by **aortic nerve** which is the branch of vagus nerve (Fig. 101.3).

Function

Whenever there is hypoxia, hypercapnea and increased hydrogen ions concentration in the blood, the chemoreceptors are stimulated and inhibitory impulses are sent to vasodilator area. Vagal tone decreases and heart rate increases. Chemoreceptors play a major role in maintaining respiration than the heart rate.

Sinoaortic Mechanism and Buffer Nerves

Sinoaortic mechanism is the mechanism of baro-receptors and chemoreceptors in carotid and aortic regions, that regulates heart rate, blood pressure and respiration. The nerves supplying these receptors are called **buffer nerves.**

■ 5. IMPULSES FROM RIGHT ATRIUM – BAINBRIDGE REFLEX

Bainbridge reflex is a **cardioaccelerator reflex** that increases the heart rate when venous return is increased. Since this reflex arises from right atrium, it is also called **right atrial reflex.**

Increase in venous return causes distention of right atrium and stimulation of **stretch receptors,** situated in the wall of right atrium. Stretch receptors, in turn, send inhibitory impulses through **inferior cervical sympathetic nerve** to vasodilator area of vasomotor center. Vasodilator area is inhibited, resulting in decrease in vagal tone and increase in heart rate (Fig. 101.5).

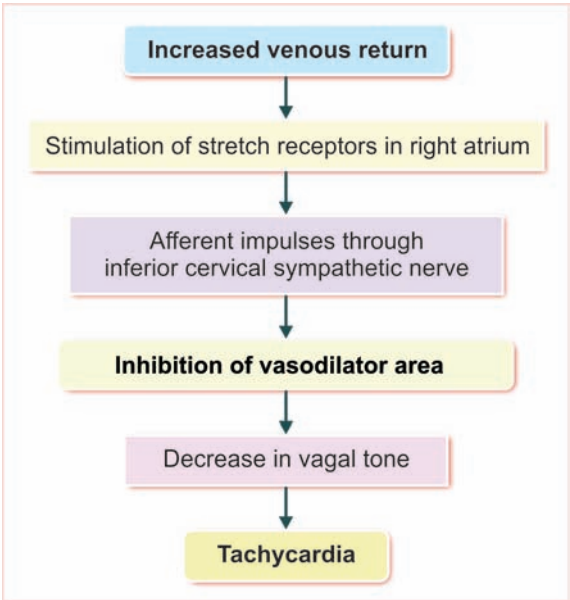

FIGURE 101.5: Bainbridge (cardioaccelerator) reflex

■ 6. IMPULSES FROM OTHER AFFERENT NERVES

Stimulation of sensory nerves produces varying effects.

Examples:

i. Stimulation of receptors in nasal mucous membrane causes **bradycardia.** Impulses from nasal mucous membrane pass via the branches of V cranial nerve and decrease the heart rate.
ii. Most of the painful stimuli cause tachycardia and some cause **bradycardia.** Impulses are transmitted via pain nerve fibers (Fig. 101.6).

■ 7. BEZOLD-JARISCH REFLEX

Bezold-Jarisch reflex is the reflex characterized by bradycardia and hypotension, caused by stimulation of **chemoreceptors** present in the wall of left ventricles by substances such as alkaloids. It is also called **coronary**

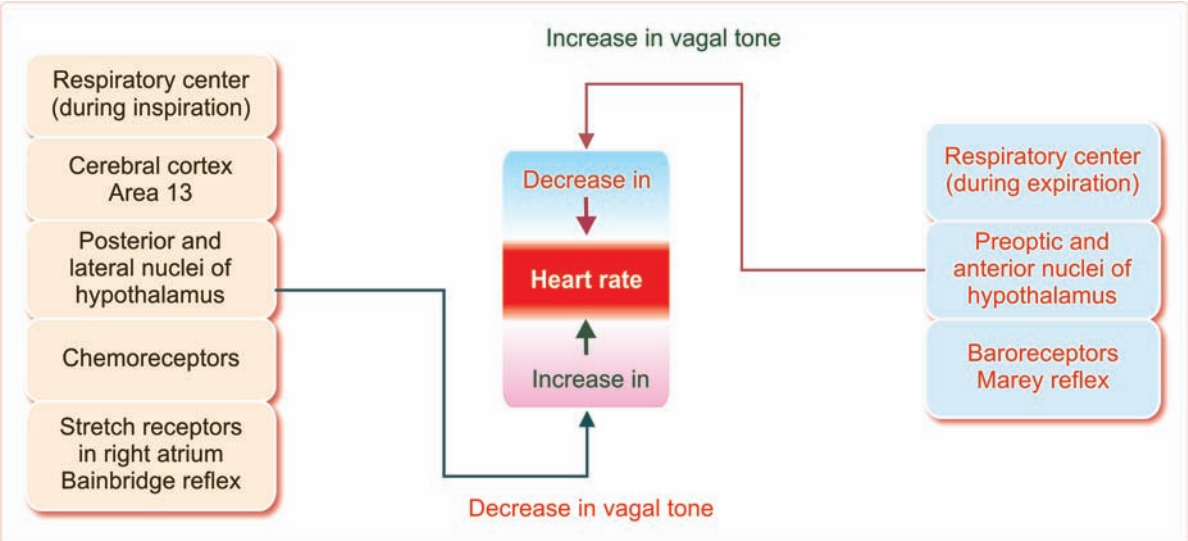

FIGURE 101.6: Factors regulating vagal tone and heart rate

chemoreflex. Vagal fibers form the afferent and efferent pathways of this reflex.

Conditions when Bezold-Jarisch Reflex Occurs

Bezold-Jarisch reflex is a pathological reflex and it does not occur in physiological conditions.

Conditions when this reflex occurs:

1. Myocardial infarction
2. Administration of thrombolytic agents
3. Hemorrhage
4. Aortic stenosis
5. Syncope.

Hemodynamics

Chapter 102

- INTRODUCTION
- MEAN VOLUME OF BLOOD FLOW
  - DEFINITION AND FORMULA
  - IMPORTANCE
  - METHODS OF STUDY
  - TYPES OF BLOOD FLOW
  - FACTORS DETERMINING VOLUME OF BLOOD FLOW
- HAGEN-POISEUILLE EQUATION
- WINDKESSEL EFFECT
- VELOCITY OF BLOOD FLOW
  - DEFINITION
  - MEAN VELOCITY OF BLOOD FLOW
  - METHODS OF STUDY
  - FACTORS MAINTAINING VELOCITY
  - PHASIC CHANGES IN THE VELOCITY OF BLOOD FLOW
- CIRCULATION TIME
  - DEFINITION
  - MEASUREMENT OF CIRCULATION TIME
  - TYPICAL CIRCULATION TIMES
  - TOTAL CIRCULATION TIME AND HEARTBEAT
  - CONDITIONS ALTERING CIRCULATION TIME
- LOCAL REGULATION OF BLOOD FLOW – AUTOREGULATION
  - INTRODUCTION
  - ROLE OF PRESSURES IN AUTOREGULATION
  - THEORIES OF AUTOREGULATION
  - AUTOREGULATION IN SOME VITAL ORGANS

INTRODUCTION

Dynamics means study of motion. **Hemodynamics** refers to the study of movement of blood through circulatory system.

Major function of cardiovascular system is to pump the blood and to circulate it through different parts of the body. It is essential for the maintenance of pressure and other physical factors within the blood vessels, so that the volume of blood supplied to different parts of the body is adequate. Circulatory system is designed for carrying out all these actions.

MEAN VOLUME OF BLOOD FLOW

DEFINITION AND FORMULA

Mean volume of blood flow is the volume of blood which flows into the region of circulatory system in a given unit of time. It is the product of mean velocity and the cross-

sectional area of the vascular bed.

$$Q = V \times A$$

Where,
Q = Quantity of blood
V = Velocity of blood flow
A = Cross-sectional area of the blood vessel.

■ IMPORTANCE

In terms of transport of foodstuffs and oxygen to the tissues and waste products away from the tissues, mean volume of blood flow is of greater physiological importance than linear velocity.

■ METHODS OF STUDY

1. *By Using Flowmeters*

Different types of flowmeters are described in Chapter 98.

2. *By Using Plethysmograph*

Plethysmograph is an instrument used for measuring the volume of an enclosed organ.

3. *By Venous Occlusion Plethysmography*

In this, the venous outflow from an organ is stopped by clamping the vein, without disturbing the artery. Blood flow into the organ causes a corresponding increase in its volume for the first few seconds. This increase in volume is recorded graphically. Amount of blood flow is determined by proper calibration of the graph.

4. *By Fick Principle*

Fick principle is explained in the measurement of cardiac output in Chapter 98.

■ TYPES OF BLOOD FLOW

Blood flow through a blood vessel is of two types:
1. Streamline or laminar flow
2. Turbulent flow.

1. *Streamline Flow*

Streamline flow is a **silent flow.** Within the blood vessel, a very thin layer of blood is in contact with the vessel wall. It does not move or moves very slowly. Next layer within the vessel has a low momentum. Next layer of blood has a slightly higher momentum. Gradually, the momentum increases in the inner layers, so that the momentum is greatest in the center of the stream. This type of flow is known as streamline flow and it does not produce any sound within the vessel (Fig. 102.1). Streamline flow occurs only at velocities up to a critical level.

2. *Turbulent Flow*

Turbulent flow is the **noisy flow.** When the velocity of blood flow increases above critical level, the flow becomes turbulent. Turbulent flow creates sounds.

Reynolds number

Critical velocity at which the flow becomes turbulent is known as Reynolds number.

Formula to determine Reynolds number:

$$N_R = \frac{PDV}{\eta}$$

N_R = Reynolds number
P = Density of the blood
D = Diameter of the vessel
V = Velocity of the flow
η = Viscosity of the blood

■ FACTORS DETERMINING VOLUME OF BLOOD FLOW

Volume of blood flow is determined by five factors:
1. Pressure gradient
2. Resistance to blood flow
3. Viscosity of blood
4. Diameter of blood vessels
5. Velocity of blood flow.

1. *Pressure Gradient*

Volume of blood flowing through any blood vessel is **directly proportional** to the pressure gradient. Pressure gradient is the pressure difference between the two ends of the blood vessel.

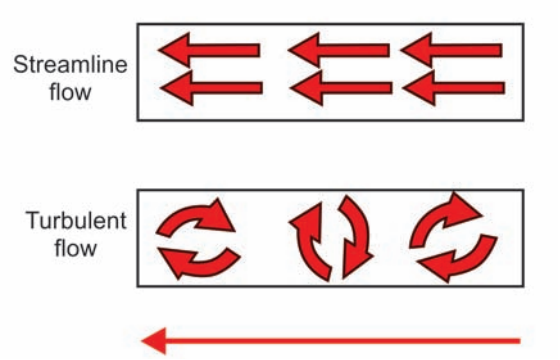

FIGURE 102.1: Streamline flow and turbulent flow

Formula to determine pressure gradient

Pressure gradient = $P_1 - P_2$

Where,

P_1 = Pressure at proximal end of the vessel

P_2 = Pressure at distal end of the vessel.

Maximum pressure gradient exists between the aorta and the inferior vena cava. The pressure in aorta is 120 mm Hg and the pressure in inferior vena cava is 0 mm Hg. So, the pressure gradient is 120 – 0 = 120 mm Hg. Pressure gradient in different areas of vascular bed is given in Table 102.1.

2. Resistance to Blood Flow (Peripheral Resistance)

Volume of blood flow is **inversely proportional** to the resistance. Resistance is the friction, tension or hindrance, against which the blood has to flow. Peripheral resistance means the resistance offered to blood flow in peripheral blood vessels. Though resistance exists in all the blood vessels to some extent, it is remarkable in the peripheral vessels, particularly the arterioles.

Determinants of peripheral resistance

i. Radius of blood vessels
ii. Pressure gradient
iii. Viscosity of blood.

Peripheral resistance is inversely related to radius of the blood vessel, i.e. lesser the radius, more will be the resistance. Radius of the arterioles is very less. It is because the arterioles remain partially constricted all the time due to sympathetic tone. So, the resistance is more. Hence, the **arterioles** are called **resistant vessels.**

Formula to determine resistance

$$\text{Resistance} = \frac{\text{Pressure gradient}}{\text{Volume of blood flow}}$$

$$= \frac{P_1 - P_2}{Q}$$

3. Viscosity of Blood

Volume of blood flow is inversely proportional to the viscosity of blood. Viscosity is the friction of blood against the wall of the blood vessel. Isaac Newton described viscosity as the internal friction or lack of slipperiness. Viscosity influences the blood flow through resistance.

Factors determining viscosity

RBC count is the main factor which determines the viscosity of the blood. Another factor determining viscosity is plasma protein, mainly albumin.

When hemoconcentration occurs as in case of burns or in polycythemia, the viscosity increases and the velocity of blood flow decreases, so the volume of blood reaching the organ is decreased.

4. Diameter of Blood Vessels

Volume of blood flow is directly proportional to the diameter of the blood vessels. When the diameter of a segment of blood vessel is considered, the aorta has the maximum diameter and capillary has got the minimum diameter. But, in circulation, the diameter of the vessel is considered in relation to the cross-sectional area through which the blood flows.

TABLE 102.1: Pressure gradient in different areas of vascular bed

Blood vessels	P_1 (mm Hg)	P_2 (mm Hg)	Pressure gradient (mm Hg)
Between aorta and vena cava	120	0	120
Between two ends of aorta	120	100	20
Between beginning of arteries and end of arterioles	100	30	70
Between arterial and venous ends of capillaries	30	15	15
Between two ends of venules	15	10	5
Between two ends of veins	10	0	10
Between two ends of vena cava	0	– 2	– 2

P_1 = Pressure at proximal end of the blood vessel, P_2 = Pressure at distal end of the blood vessel, Pressure gradient = $P_1 - P_2$.

Cross-sectional area is progressively increased as the arteries ramify and as the distance from the heart is increased. Cross-sectional area of each branch is smaller, but the sum of the cross-sectional areas of all the branches is always greater than that of the parent vessel. In this way, the aorta has got less cross-sectional area of 4 cm^2, compared to that of capillaries, which is 2,500 cm^2.

But, the cross-sectional area is subjected to variations under physiological and pathological conditions. Diameter of the aorta depends upon the elasticity of the wall and its recoiling tendency helps in maintaining the flow and pressure. Diameter of the arterioles depends upon the **sympathetic tone.**

5. *Velocity of Blood Flow*

Volume of blood flow is **directly proportional** to the velocity of blood flow. Velocity of blood flow is the rate at which blood flows through a particular region. It is described later in this chapter.

■ HAGEN-POISEUILLE EQUATION

Hagen and Poiseuille have worked on dynamics extensively. Equation which explains the relationship between different variables of dynamics, is named after them. Variables of dynamics are applied to hemodynamics also.

According to Hagen-Poiseuille equation, volume (Q) of any fluid flowing through a rigid tube is:

1. Directly proportional to pressure gradient ($P_1 - P_2$)
2. Directly proportional to the fourth power of radius (r^4)
3. Inversely proportional to the length of the tube (L).

$$\text{Thus, } Q = K \frac{(P_1 - P_2) \times r^4}{L}$$

K is the constant for fluid flowing at a temperature. It is directly proportional to temperature of the fluid. Viscosity of the fluid is also affected by the temperature. Viscosity is inversely proportional to temperature of the fluid. Therefore in the equation, the constant 'K' is expressed as the reciprocal of viscosity (η).

$$\text{So, } Q = \frac{(P_1 - P_2) \times r^4}{L \times \eta}$$

Volume of flow of fluid is always expressed in a given unit of time. π/8 is the arithmetic value derived while determining volume of fluid flowing in a given unit of time. So, the equation has to be rewritten as:

$$Q = \frac{(P_1 - P_2) \times r^4}{L \times \eta \times \pi/8}$$

$$= \frac{(P_1 - P_2) \times \pi r^4}{8 (L \times \eta)}$$

$$\text{Thus, } Q = \frac{(P_1 - P_2)\, \pi r^4}{8 (L \times \eta)}$$

■ WINDKESSEL EFFECT

Windkessel effect is the recoiling effect of blood vessels that converts the **pulsatile flow** of blood into a **continuous flow.** Blood vessels showing the windkessel effect are known as **windkessel vessels.**

Mean velocity of the blood that flows through the aorta is more than 50 cm/second, but it is not constant. During systole, it increases up to 120 cm/second and during diastole, it becomes almost negative. This variation is noticed even in the larger arteries.

During systole, velocity of blood flow reaches maximum, because of the force created by contraction of the heart. Therefore, the maximum volume of blood is pumped into the aorta. During diastole, this force is absent and the volume of blood entering the aorta is zero. Thus, the flow of blood into the aorta is not continuous. This type of flow is called pulsatile flow.

However, the flow of blood through other blood vessels is continuous. It is because of the behavioral pattern of aorta and to a little extent, the behavioral pattern of larger arteries. During systole, the aorta is completely dilated and during diastole, it recoils. The elastic recoiling of this vessel creates the continuous momentum of blood. So, the pulsatile flow of blood is converted into a continuous flow.

This effect was named as windkessel effect by **Otto Frank,** in 1899. Windkessel is a German word used for an **'elastic reservoir'.**

Thus, the windkessel vessels play an important role in maintaining the continuous flow of blood through the circulatory tree by acting as a **second pump,** the **first pump** being the heart.

■ VELOCITY OF BLOOD FLOW

■ DEFINITION

Velocity of blood flow is the rate at which blood flows through a particular region of the body. It mainly depends upon the diameter or cross-sectional area of blood vessel.

■ MEAN VELOCITY OF BLOOD FLOW IN DIFFERENT VESSELS

Mean velocity (cm/second) of blood flow in different blood vessels:

Large arteries	:	50.00
Small arteries	:	5.00
Arterioles	:	0.50
Capillaries	:	0.05
Venules	:	0.10
Small veins	:	1.00
Large veins	:	2.00

■ METHODS OF STUDY

1. *By Using Flowmeters*

Flowmeters are described in Chapter 98.

2. *By Hemodromography*

Hemodromography is a technique by which the velocity of blood is continuously recorded.

■ FACTORS MAINTAINING VELOCITY

Three factors are responsible for the maintenance of the velocity of blood flow:

1. Cardiac output
2. Cross-sectional area of the blood vessel
3. Viscosity of the blood.

1. *Cardiac Output*

Velocity of blood flow is **directly proportional** to cardiac output. Increase in cardiac output leads to increase in the velocity of blood flow in all parts of the circulation.

2. *Cross-sectional Area of Blood Vessels*

Velocity of blood flow is **inversely proportional** to the total cross-sectional area of the vascular bed, through which the blood circulates. Cross-sectional area increases progressively as the arteries ramify. Cross-sectional area of each branch is smaller, but the sum of the cross-sectional areas of all the branches is always greater than that of the parent vessel. So, velocity of blood flow is decreased as the distance from the heart is increased.

3. *Viscosity of Blood*

Velocity of blood flow is **inversely proportional** to the viscosity of blood. If viscosity is more, the velocity of blood flow is reduced (See in Factors maintaining volume of blood flow). It is because of the friction of blood against arterial wall, which is more when viscosity of blood is increased.

■ PHASIC CHANGES IN THE VELOCITY OF BLOOD FLOW

Velocity of blood flow is altered according to the phases of cardiac cycle. Blood flows in the large arteries at a greater speed during systole than during diastole. In common carotid artery, the velocity reaches 50 cm/sec during systole and it is only 30 cm/sec during diastole.

■ CIRCULATION TIME

■ DEFINITION

Circulation time is the time taken by blood to travel through a part or whole of the circulatory system.

If a substance is injected into a vein, the time taken by it to appear in the blood of the same vein or in the corresponding vein on the opposite side shows the total circulation time.

Similarly, if the transit is from vein to the lungs, it shows the circulation time through pulmonary circuit and if it is from vein to capillaries, it shows the time for flow through pulmonary circuit, left heart and arteries to capillaries, i.e. the total circulation time minus the time for venous return.

■ MEASUREMENT OF CIRCULATION TIME

Circulation time is measured by introducing some easily recognized substance into bloodstream and determining the time when the substance appears at a given point **(end point)** in the circulation.

The injected substance must produce some characteristic response at its end point, so that its appearance could be easily recognized. Introduction of the substance into circulation is done by injecting through median cubital vein or directly into the heart.

Substances used for Measuring Circulation Time

1. Histamine: Causes flushing of face due to vasodilatation
2. Dehydrocholine (20%): Gives a bitter taste when it reaches the tongue
3. Ether or acetone: Detectable in breath by smell
4. Sodium cyanide (small dose): Causes hyperpnea when it reaches the carotid artery (by acting on baroreceptors)
5. Dye fluorescein: Identified at the end point by yellow color; it is used for total circulation time
6. Radioactive substances: Detected at various points of the body by using an ionization chamber.

TYPICAL CIRCULATION TIMES

1. Arm vein to arm vein (total circulation time): 25 seconds (22 to 28), determined by using dye fluorescein
2. Arm vein to face: 24 seconds, determined by using histamine
3. Arm vein to tongue: 11 seconds (8 to 16), determined by using dehydrocholine
4. Arm vein to lung (pulmonary circulatory time): 6 seconds (4 to 6), determined by using ether or acetone
5. Arm vein to heart (shortest circulation time): 4 seconds, determined by using radioactive substances
6. Arm vein to carotid artery: 14 seconds (12 to 15), determined by using sodium cyanide.

TOTAL CIRCULATION TIME AND HEARTBEAT

Number of heartbeat/total circulation time, however, remains the same for human beings and all the animals, i.e. about 30/total circulation time.

CONDITIONS ALTERING CIRCULATION TIME

Circulation time is decreased when the velocity of blood flow is increased and the circulation time is more when the velocity is less.

Conditions when Circulation Time is Prolonged (Sluggish Blood Flow)

1. Myxedema: Due to decreased metabolic activity
2. Polycythemia: Due to increased viscosity of blood
3. Cardiac failure: Due to inability of the heart to pump blood.

Conditions when Circulation Time is Shortened (Rapid Blood Flow)

1. Exercise: Due to increased cardiac activity and vasodilatation
2. Adrenaline administration: Due to increased cardiac activity
3. Hyperthyroidism: Due to increased metabolic activity
4. Anemia: Due to decreased blood volume and less viscosity
5. Decrease in peripheral resistance: Due to vasodilatation.

LOCAL REGULATION OF BLOOD FLOW – AUTOREGULATION

INTRODUCTION

Autoregulation means the regulation of blood flow to an organ by the organ itself. It is defined as the **intrinsic ability** of an organ to regulate a constant blood flow, in spite of changes in the perfusion pressure (arterial pressure – venous pressure).

Normally, a sudden increase or decrease in arterial blood pressure momentarily increases or decreases the blood flow. Local mechanisms start functioning and the blood flow is brought to relatively normal level within few minutes.

Autoregulatory response is independent of neural and hormonal influences. So, it is the **intrinsic capacity** of the organ.

ROLE OF PRESSURES IN AUTOREGULATION

Perfusion Pressure and Effective Perfusion Pressure

Generally, the term perfusion pressure refers to balance between the pressure in blood vessels on either side of the organ, i.e. arterial pressure minus venous pressure ($P_A - P_V$) across the organ.

However, the blood flow to any organ or region of the body depends up on the effective perfusion pressure. Effective perfusion pressure is the perfusion pressure divided by resistance in the blood vessels.

Formula to determine effective perfusion pressure

$$EFP = \frac{P_A - P_V}{R}$$

EFP = Effective perfusion pressure
P_A = Arterial pressure
P_V = Venous pressure
R = Resistance

But basically, the major factor that determines the perfusion pressure and effective perfusion pressure is the **mean arterial pressure.** The normal mean arterial blood pressure is about 93 mm Hg. Usually, blood flow through an organ is kept constant when the mean arterial pressure increases up to 170 mm Hg or when it falls till 60 mm Hg (the range varies slightly in different organs). However, beyond this range, the autoregulation fails and the blood flow is altered in relation to rise or fall in pressure.

■ THEORIES OF AUTOREGULATION

Autoregulation is explained by two theories:

1. Myogenic theory
2. Metabolic theory.

1. *Myogenic Theory*

According to this theory, the intrinsic contractile property of the smooth muscle fibers present in the blood vessels is responsible for autoregulation. It is known that the sudden stretching of blood vessels causes contraction of smooth muscle fibers present in the wall of the vessels, particularly small arteries and arterioles. So, when the arterial blood pressure increases suddenly, the stretching of the blood vessels immediately causes vasoconstriction and thereby, the blood flow is controlled.

Stretching of blood vessels due to increased blood pressure increases the flow of calcium ions into the cells from ECF. Calcium influx causes contraction of smooth muscles in the blood vessels, leading to vasoconstriction.

On the other hand, when the blood pressure is less, the stretching of blood vessels is less causing vasodilatation and increase in blood flow.

2. *Metabolic Theory*

According to metabolic theory the normal blood flow is maintained by the metabolic end products. Normally, the flow of blood washes away the metabolic end products. When the blood flow is reduced, there is accumulation of metabolites. These metabolites dilate the blood vessels and bring the blood flow back to normal. Conversely, when blood flow increases, the vasodilator metabolites are washed out of the tissues quickly. It leads to vasoconstriction and the volume of blood flow becomes normal.

Common vasodilators of metabolic origin:

i. Adenosine
ii. Carbon dioxide
iii. Lactate
iv. Hydrogen.

■ AUTOREGULATION IN SOME VITAL ORGANS

Volume of blood flow is regulated by local mechanisms in almost all the tissues of the body. However, autoregulation is more effective in some of the **vital organs** like **kidney** (Chapter 51), **heart** (Chapter 108) and **brain** (Chapter 109). Mechanism of autoregulation also varies slightly in these organs.

Arterial Blood Pressure

Chapter 103

- DEFINITIONS AND NORMAL VALUES
  - SYSTOLIC BLOOD PRESSURE
  - DIASTOLIC BLOOD PRESSURE
  - PULSE PRESSURE
  - MEAN ARTERIAL PRESSURE
- VARIATIONS
  - PHYSIOLOGICAL VARIATIONS
  - PATHOLOGICAL VARIATIONS
- DETERMINANTS OF ARTERIAL BLOOD PRESSURE
  - CENTRAL FACTORS
  - PERIPHERAL FACTORS
- REGULATION OF ARTERIAL BLOOD PRESSURE
- NERVOUS MECHANISM
  - VASOMOTOR CENTER
  - VASOCONSTRICTOR FIBERS
  - VASODILATOR FIBERS
  - MECHANISM OF ACTION OF VASOMOTOR CENTER
- RENAL MECHANISM
  - BY REGULATION OF EXTRACELLULAR FLUID VOLUME
  - THROUGH RENIN-ANGIOTENSIN MECHANISM
- HORMONAL MECHANISM
  - HORMONES WHICH INCREASE BLOOD PRESSURE
  - HORMONES WHICH DECREASE BLOOD PRESSURE
- LOCAL MECHANISM
  - LOCAL VASOCONSTRICTORS
  - LOCAL VASODILATORS
- MEASUREMENT OF ARTERIAL BLOOD PRESSURE
  - DIRECT METHOD
  - INDIRECT METHOD
- APPLIED PHYSIOLOGY
  - HYPERTENSION
  - HYPOTENSION

DEFINITIONS AND NORMAL VALUES

Arterial blood pressure is defined as the lateral pressure exerted by the column of blood on wall of arteries. The pressure is exerted when blood flows through the arteries. Generally, the term 'blood pressure' refers to arterial blood pressure.

Arterial blood pressure is expressed in four different terms:

1. Systolic blood pressure
2. Diastolic blood pressure
3. Pulse pressure
4. Mean arterial blood pressure.

SYSTOLIC BLOOD PRESSURE

Systolic blood pressure (systolic pressure) is defined as the **maximum pressure** exerted in the arteries **during systole** of heart.

Normal systolic pressure: 120 mm Hg (110 mm Hg to 140 mm Hg).

DIASTOLIC BLOOD PRESSURE

Diastolic blood pressure (diastolic pressure) is defined as the **minimum pressure** exerted in the arteries **during diastole** of heart.

Normal diastolic pressure: 80 mm Hg (60 mm Hg to 80 mm Hg).

PULSE PRESSURE

Pulse pressure is the difference between the systolic pressure and diastolic pressure.

Normal pulse pressure: 40 mm Hg (120 – 80 = 40).

MEAN ARTERIAL BLOOD PRESSURE

Mean arterial blood pressure is the average pressure existing in the arteries. It is not the arithmetic mean of systolic and diastolic pressures. It is the diastolic pressure plus one third of pulse pressure. To determine the mean pressure, diastolic pressure is considered than the systolic pressure. It is because, the diastolic period of cardiac cycle is longer (0.53 second) than the systolic period (0.27 second).

Normal mean arterial pressure: 93 mm Hg (80 + 13 = 93).

Formula to calculate mean arterial blood pressure:

Mean arterial blood pressure
= Diastolic pressure + 1/3 of pulse pressure

$$= 80 + \frac{40}{3} = 93.3 \text{ mm Hg}$$

VARIATIONS

Blood pressure is altered in physiological and pathological conditions. Systolic pressure is subjected for variations easily and quickly and its variation occurs in a wider range. Diastolic pressure is not subjected for easy and quick variations and its variation occurs in a narrow range.

PHYSIOLOGICAL VARIATIONS

1. *Age*

Arterial blood pressure increases as age advances.

Systolic pressure in different age

Newborn	:	70 mm Hg
After 1 month	:	85 mm Hg
After 6 month	:	90 mm Hg
After 1 year	:	95 mm Hg
At puberty	:	120 mm Hg
At 50 years	:	140 mm Hg
At 70 years	:	160 mm Hg
At 80 years	:	180 mm Hg

Diastolic pressure in different age

Newborn	:	40 mm Hg
After 1 month	:	45 mm Hg
After 6 month	:	50 mm Hg
After 1 year	:	55 mm Hg
At puberty	:	80 mm Hg
At 50 years	:	85 mm Hg
At 70 years	:	90 mm Hg
At 80 years	:	95 mm Hg

2. *Sex*

In females, up to the period of menopause, arterial pressure is 5 mm Hg, less than in males of same age. After menopause, the pressure in females becomes equal to that in males of same age.

3. *Body Built*

Pressure is more in obese persons than in lean persons.

4. *Diurnal Variation*

In early morning, the pressure is slightly low. It gradually increases and reaches the maximum at noon. It becomes low in evening.

5. *After Meals*

Arterial blood pressure is increased for few hours after meals due to increase in cardiac output.

6. *During Sleep*

Usually, the pressure is reduced up to 15 to 20 mm Hg during deep sleep. However, it increases slightly during sleep associated with dreams.

7. *Emotional Conditions*

During excitement or anxiety, the blood pressure is increased due to release of adrenaline.

8. *After Exercise*

After moderate exercise, systolic pressure increases by 20 to 30 mm Hg above the basal level due to increase in rate and force of contraction and stroke volume. Normally, diastolic pressure is not affected by moderate exercise. It is because, the diastolic pressure depends upon peripheral resistance, which is not altered by moderate exercise.

After severe muscular exercise, systolic pressure rises by 40 to 50 mm Hg above the basal level. But, the diastolic pressure reduces because the peripheral resistance decreases in severe muscular exercise. More details are given in Chapter 117.

■ PATHOLOGICAL VARIATIONS

Pathological variations of arterial blood pressure are hypertension and hypotension. Refer applied physiology of this chapter for details.

■ DETERMINANTS OF ARTERIAL BLOOD PRESSURE – FACTORS MAINTAINING ARTERIAL BLOOD PRESSURE

Some factors are necessary to maintain normal blood pressure. These factors are called **local factors, mechanical factors** or determinants of blood pressure (Table 103.1).

Types of Local Factors

Local factors are divided into two types:

A. Central factors, which are pertaining to the heart:
   1. Cardiac output
   2. Heart rate

B. Peripheral factors, which are pertaining to blood and blood vessels:
   3. Peripheral resistance
   4. Blood volume
   5. Venous return
   6. Elasticity of blood vessels
   7. Velocity of blood flow
   8. Diameter of blood vessels
   9. Viscosity of blood.

■ CENTRAL FACTORS

1. *Cardiac Output*

Systolic pressure is **directly proportional** to cardiac output. Whenever the cardiac output increases, the systolic pressure is increased and when cardiac output is less, the systolic pressure is reduced. Cardiac output increases in muscular exercise, emotional conditions, etc. So in these conditions, the systolic pressure is increased. In conditions like myocardial infarction, the cardiac output decreases, resulting in fall in systolic pressure.

2. *Heart Rate*

Moderate changes in heart rate do not affect arterial blood pressure much. However, marked alteration in the heart rate affects the blood pressure by altering cardiac output (Chapter 98).

■ PERIPHERAL FACTORS

3. *Peripheral Resistance*

Peripheral resistance is the important factor, which maintains diastolic pressure. Diastolic pressure is **directly proportional** to peripheral resistance. Peripheral resistance is the resistance offered to the blood flow at the periphery. Resistance is offered at arterioles, which are called the resistant vessels. When peripheral resistance increases, diastolic pressure is increased and when peripheral resistance decreases, the diastolic pressure is decreased.

TABLE 103.1: Local factors determining arterial blood pressure

Arterial blood pressure	Factors
Arterial blood pressure is directly proportional to	1. Cardiac output 2. Heart rate 3. Peripheral resistance 4. Blood volume 5. Venous return 6. Velocity of blood flow 7. Viscosity of blood
Arterial blood pressure is inversely proportional to	1. Elasticity of blood vessel 2. Diameter of blood vessel

4. *Blood Volume*

Blood pressure is **directly proportional** to blood volume. Blood volume maintains the blood pressure through the venous return and cardiac output. If the blood volume increases, there is an increase in venous return and cardiac output, resulting in elevation of blood pressure (Fig. 103.1).

5. *Venous Return*

Blood pressure is **directly proportional** to venous return. When venous return increases, there is an increase in ventricular filling and cardiac output, resulting in elevation of arterial blood pressure.

6. *Elasticity of Blood Vessels*

Blood pressure is **inversely proportional** to the elasticity of blood vessels. Due to elastic property, the blood vessels are distensible and are able to maintain the pressure. When the elastic property is lost, the blood vessels become rigid **(arteriosclerosis)** and pressure increases as in old age. Deposition of cholesterol, fatty acids and calcium ions produce rigidity of blood vessels and **atherosclerosis,** leading to increased blood pressure.

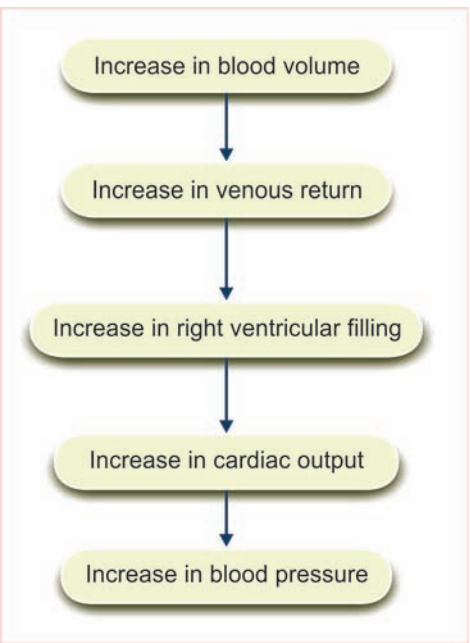

FIGURE 103.1: Effect of blood volume and venous return on arterial blood pressure

7. *Velocity of Blood Flow*

Pressure in a blood vessel is **directly proportional** to the velocity of blood flow. If the velocity of blood flow increases, the resistance is increased. So, the pressure is increased.

8. *Diameter of Blood Vessels*

Arterial blood pressure is **inversely proportional** to the diameter of blood vessel. If the diameter decreases, the peripheral resistance increases, leading to increase in the pressure.

9. *Viscosity of Blood*

Arterial blood pressure is **directly proportional** to the viscosity of blood. When viscosity of blood increases, the frictional resistance is increased and this increases the pressure.

■ REGULATION OF ARTERIAL BLOOD PRESSURE

Arterial blood pressure varies even under physiological conditions. However, immediately it is brought back to normal level because of the presence of well organized regulatory mechanisms in the body. Body has four such regulatory mechanisms to maintain the blood pressure within normal limits (Fig. 103.2):

A. Nervous mechanism or short-term regulatory mechanism
B. Renal mechanism or long-term regulatory mechanism
C. Hormonal mechanism
D. Local mechanism.

■ NERVOUS MECHANISM FOR REGULATION OF BLOOD PRESSURE – SHORT-TERM REGULATION

Nervous regulation is rapid among all the mechanisms involved in the regulation of arterial blood pressure. When the pressure is altered, nervous system brings the pressure back to normal within few minutes. Although nervous mechanism is **quick in action,** it operates only for a short period and then it adapts to the new pressure. Hence, it is called short-term regulation. The nervous mechanism regulating the arterial blood pressure operates through the vasomotor system.

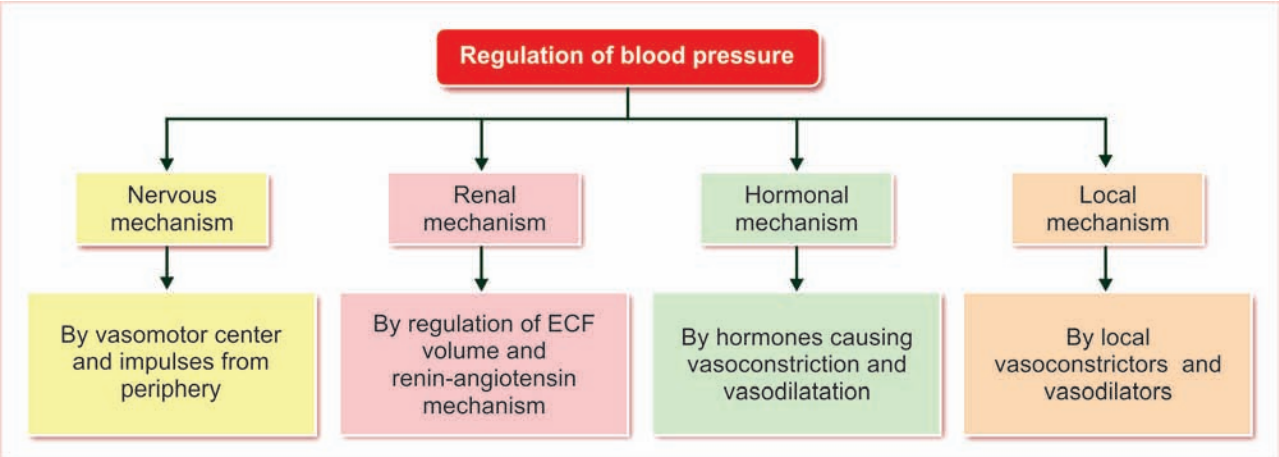

FIGURE 103.2: Regulation of blood pressure. ECF = Extracellular fluid.

Vasomotor System

Vasomotor system includes three components:
1. Vasomotor center
2. Vasoconstrictor fibers
3. Vasodilator fibers.

■ VASOMOTOR CENTER

Vasomotor center is bilaterally situated in the reticular formation of medulla oblongata and the lower part of the pons.

Vasomotor center consists of three areas:
- i. Vasoconstrictor area
- ii. Vasodilator area
- iii. Sensory area.

i. *Vasoconstrictor Area*

Vasoconstrictor area is also called the **pressor area.** It forms the lateral portion of vasomotor center. Vasoconstrictor area sends impulses to blood vessels through sympathetic vasoconstrictor fibers. So, the stimulation of this area causes vasoconstriction and rise in arterial blood pressure. This area is also concerned with acceleration of heart rate (Chapter 101).

ii. *Vasodilator Area*

Vasodilator area is otherwise called **depressor area.** It forms the medial portion of vasomotor center. This area suppresses the vasoconstrictor area and causes vasodilatation. It is also concerned with cardioinhibition (Chapter 101).

iii. *Sensory Area*

Sensory area is in the nucleus of tractus solitarius, which is situated in posterolateral part of medulla and pons. This area receives sensory impulses via glossopharyngeal and vagal nerves from the periphery, particularly from the baroreceptors. Sensory area in turn, controls the vasoconstrictor and vasodilator areas.

■ VASOCONSTRICTOR FIBERS

Vasoconstrictor fibers belong to the sympathetic division of autonomic nervous system. These fibers cause vasoconstriction by the release of neurotransmitter substance, **noradrenaline.** Noradrenaline acts through alpha receptors of smooth muscle fibers in blood vessels.

Vasoconstrictor fibers play major role than the vasodilator fibers in the regulation of blood pressure.

Vasomotor Tone

Vasomotor tone is the continuous discharge of impulses from vasoconstrictor center through the vasoconstrictor fibers. Vasomotor tone plays an important role in regulating the pressure by producing a constant partial state of constriction of the blood vessels. Thus, the arterial blood pressure is directly proportional to the vasomotor tone. Vasomotor tone is also called **sympathetic vasoconstrictor tone** or **sympathetic tone.**

■ VASODILATOR FIBERS

Vasodilator fibers are of three types:
- i. Parasympathetic vasodilator fibers
- ii. Sympathetic vasodilator fibers
- iii. Antidromic vasodilator fibers.

i. *Parasympathetic Vasodilator Fibers*

Parasympathetic vasodilator fibers cause dilatation of blood vessels by releasing **acetylcholine.**

ii. *Sympathetic Vasodilator Fibers*

Some of the sympathetic fibers cause vasodilatation in certain areas, by secreting **acetylcholine.** Such fibers are called **sympathetic vasodilator** or **sympathetic cholinergic fibers.** Sympathetic cholinergic fibers, which supply the blood vessels of skeletal muscles, are important in increasing the blood flow to muscles by vasodilatation, during conditions like exercise.

Sympathetic cholinergic vasodilator fibers form the important part of vasomotor system. Signals for the vasodilator fibers are generated in cerebral cortex. Signals are relayed through the fibers from cerebral cortex to lateral gray horn of the spinal cord via hypothalamus, midbrain and medulla. In the spinal cord, these impulses activate the preganglionic sympathetic fibers. These fibers in turn, activate the postganglionic fibers. Postganglionic fibers cause dilatation of blood vessels by secreting acetylcholine.

iii. *Antidromic Vasodilator Fibers*

Normally, the impulses produced by a cutaneous receptor (like pain receptor) pass through sensory nerve fibers. But, some of these impulses pass through the other branches of the axon in the opposite direction and reach the blood vessels supplied by these branches. These impulses now dilate the blood vessels. It is called the **antidromic** or **axon reflex** and the nerve fibers are called antidromic vasodilator fibers (see Fig. 113.1, Chapter 113).

■ MECHANISM OF ACTION OF VASOMOTOR CENTER IN THE REGULATION OF BLOOD PRESSURE

Vasomotor center regulates the arterial blood pressure by causing vasoconstriction or vasodilatation. However, its actions depend upon the impulses it receives from other structures such as baroreceptors, chemoreceptors, higher centers and respiratory centers. Among these structures, baroreceptors and chemoreceptors play a major role in the short-term regulation of blood pressure.

1. *Baroreceptor Mechanism*

Baroreceptors are the receptors, which give response to change in blood pressure. Baroreceptors are also called **pressoreceptors.**

Situation

Baroreceptors are situated in the **carotid sinus** and wall of the **aorta** (Refer Chapter 101).

Nerve supply

Refer Chapter 101 and Figure 101.3.

Functions

Role of baroreceptors when blood pressure increases

When arterial blood pressure rises rapidly, baroreceptors are activated and send stimulatory impulses to **nucleus of tractus solitarius** through glassopharyngeal and vagus nerves. Now, the nucleus of tractus solitarius acts on both vasoconstrictor area and vasodilator areas of vasomotor center. It inhibits the vasoconstrictor area and excites the vasodilator area.

Inhibition of vasoconstrictor area reduces vasomotor tone. Reduction in vasomotor tone causes vasodilatation, resulting in decreased peripheral resistance. Simultaneous excitation of vasodilator center increases vagal tone (Chapter 101). This decreases the rate and force of contraction of heart, leading to reduction in cardiac output. These two factors, i.e. decreased peripheral resistance and reduced cardiac output bring the arterial blood pressure back to normal level (Fig. 103.3).

Role of baroreceptors when blood pressure decreases

The fall in arterial blood pressure or the occlusion of common carotid arteries decreases the pressure in carotid sinus. This causes inactivation of baroreceptors. Now, there is no inhibition of vasoconstrictor center or excitation of vasodilator center. Therefore, the blood pressure rises.

Information regarding blood pressure within the range of 50 to 200 mm Hg (mean arterial pressure) reaches the vasomotor center through the carotid baroreceptors. Information about the blood pressure range of 100 to 200 mm Hg goes through aortic baroreceptors.

Both carotid and aortic baroreceptors are stimulated by the rising pressure than the steady pressure and their response depends upon the rate of increase in the blood pressure.

Since the baroreceptor mechanism acts against the rise in arterial blood pressure, it is called **pressure buffer mechanism** or system and the nerves from baroreceptors are called the **buffer nerves.**

2. *Chemoreceptor Mechanism*

Chemoreceptors are the receptors giving response to change in chemical constituents of blood. Peripheral chemoreceptors influence the vasomotor center.

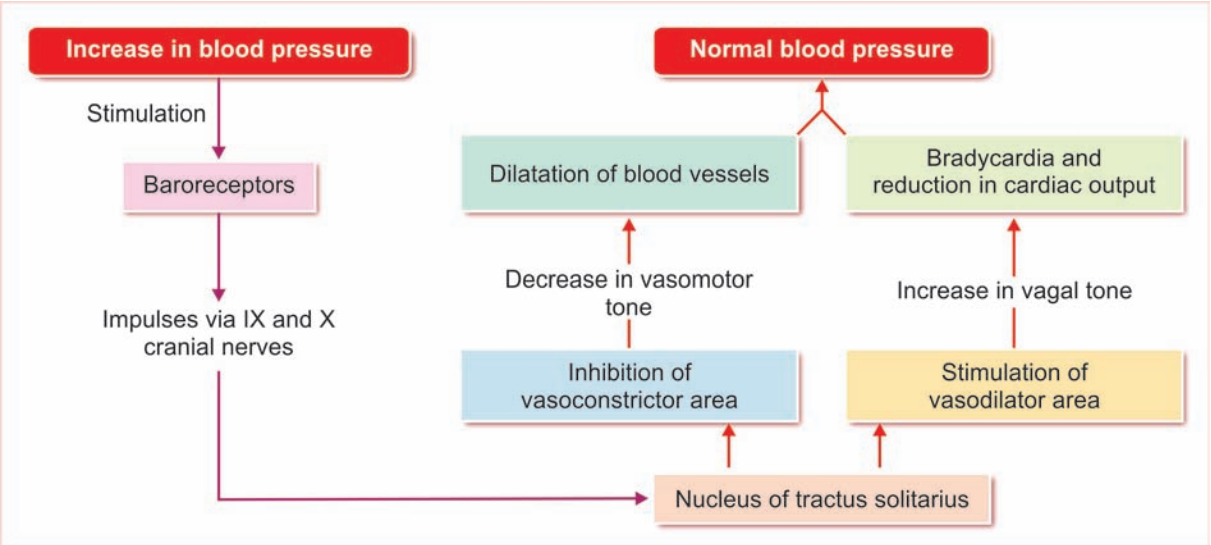

FIGURE 103.3: Regulation of blood pressure by baroreceptor mechanism

Situation

Peripheral chemoreceptors are situated in the carotid body and aortic body (Chapter 101).

Nerve supply

Refer Chapter 101 and Figure 101.3.

Function

Peripheral chemoreceptors are sensitive to lack of oxygen, excess of carbon dioxide and hydrogen ion concentration in blood. Whenever blood pressure decreases, blood flow to chemoreceptors decreases, resulting in decreased oxygen content and excess of carbon dioxide and hydrogen ion. These factors excite the chemoreceptors, which send impulses to stimulate vasoconstrictor center. Blood pressure rises and blood flow increases. Chemoreceptors play a major role in maintaining respiration rather than blood pressure (Chapter 126).

Sinoaortic mechanism

Mechanism of action of baroreceptors and chemoreceptors in carotid and aortic region constitute sinoaortic mechanism. Nerves supplying the baroreceptors and chemoreceptors are called **buffer nerves** because these nerves regulate the heart rate (Chapter 101), blood pressure and respiration (Chapter 126).

3. *Higher Centers*

Vasomotor center is also controlled by the impulses from the two higher centers in the brain.

i. *Cerebral cortex*

Area 13 in cerebral cortex is concerned with emotional reactions. During emotional conditions, this area sends impulses to vasomotor center. Vasomotor center is activated, the vasomotor tone is increased and the pressure rises.

ii. *Hypothalamus*

Stimulation of posterior and lateral nuclei of hypothalamus causes vasoconstriction and increase in blood pressure. Stimulation of preoptic area causes vasodilatation and decrease in blood pressure. Impulses from hypothalamus are mediated via vasomotor center.

4. *Respiratory Centers*

During the beginning of expiration, arterial blood pressure increases slightly, i.e. by 4 to 6 mm Hg. It decreases during later part of expiration and during inspiration because of two factors:

i. Radiation of impulses from respiratory centers towards vasomotor center at different phases of respiratory cycle
ii. Pressure changes in thoracic cavity, leading to alteration of venous return and cardiac output.

■ RENAL MECHANISM FOR REGULATION OF BLOOD PRESSURE – LONG-TERM REGULATION

Kidneys play an important role in the long-term regulation of arterial blood pressure. When blood pressure alters slowly in several days/months/years, the nervous

mechanism adapts to the altered pressure and looses the sensitivity for the changes. It cannot regulate the pressure any more. In such conditions, the renal mechanism operates efficiently to regulate the blood pressure. Therefore, it is called long-term regulation.

Kidneys regulate arterial blood pressure by two ways:

1. By regulation of ECF volume
2. Through renin-angiotensin mechanism.

■ BY REGULATION OF EXTRACELLULAR FLUID VOLUME

When the blood pressure increases, kidneys excrete large amounts of water and salt, particularly sodium, by means of **pressure diuresis** and pressure natriuresis. Pressure diuresis is the excretion of large quantity of water in urine because of increased blood pressure. Even a slight increase in blood pressure doubles the water excretion. Pressure natriuresis is the excretion of large quantity of sodium in urine.

Because of **diuresis** and **natriuresis,** there is a decrease in ECF volume and blood volume, which in turn brings the arterial blood pressure back to normal level.

When blood pressure decreases, the reabsorption of water from renal tubules is increased. This in turn, increases ECF volume, blood volume and cardiac output, resulting in restoration of blood pressure.

■ THROUGH RENIN-ANGIOTENSIN MECHANISM

Source of renin secretion, formation of angiotensin and conditions when renin is secreted are described in Chapter 50.

Actions of Angiotensin II

When blood pressure and ECF volume decrease, renin secretion from kidneys is increased. It converts angiotensinogen into angiotensin I. This is converted into angiotensin II by ACE (angiotensin-converting enzyme).

Angiotensin II acts in two ways to restore the blood pressure:

i. It causes constriction of arterioles in the body so that the peripheral resistance is increased and blood pressure rises. In addition, angiotensin II causes constriction of afferent arterioles in kidneys, so that glomerular filtration reduces. This results in retention of water and salts, increases ECF volume to normal level. This in turn increases the blood pressure to normal level.

ii. Simultaneously, angiotensin II stimulates the adrenal cortex to secrete aldosterone. This hormone increases reabsorption of sodium from renal tubules. Sodium reabsorption is followed by water reabsorption, resulting in increased

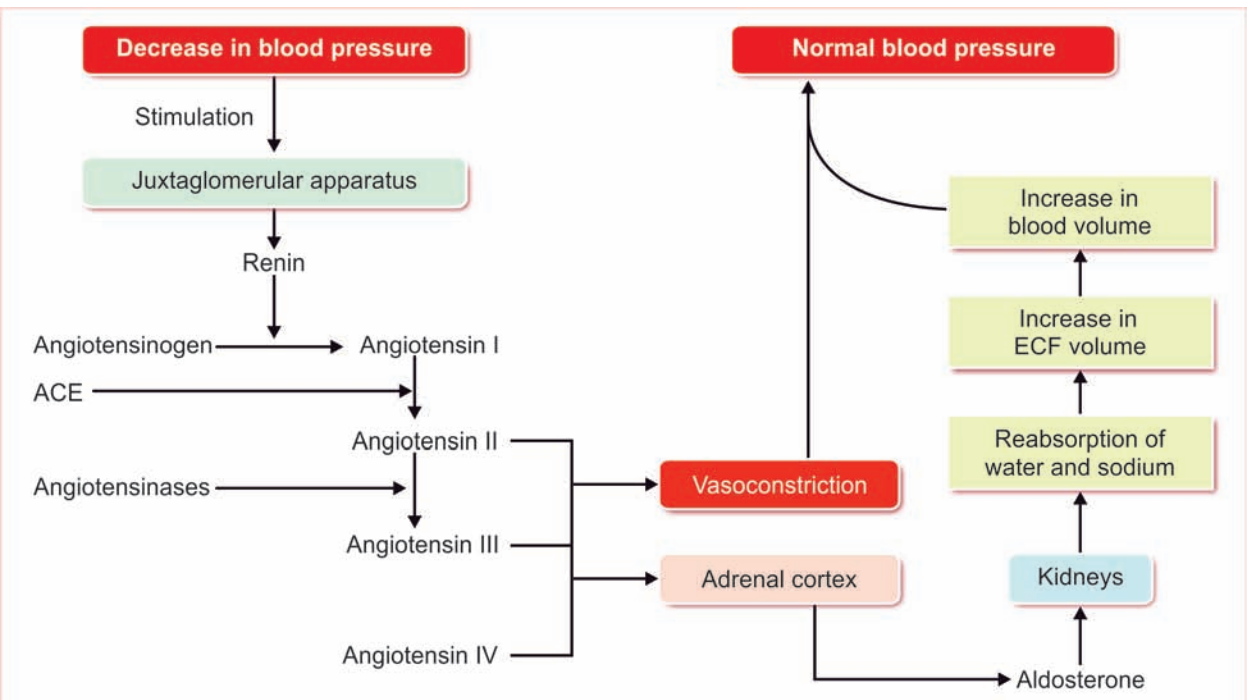

FIGURE 103.4: Regulation of blood pressure by renin-angiotensin mechanism. ACE = Angiotensin-converting enzyme.

ECF volume and blood volume. It increases the blood pressure to normal level (Fig. 103.4).

Actions of Angiotensin III and Angiotensin IV

Like angiotensin II, the angiotensins III and IV also increase the blood pressure and stimulate adrenal cortex to secrete aldosterone (Chapter 50).

■ HORMONAL MECHANISM FOR REGULATION OF BLOOD PRESSURE

Many hormones are involved in the regulation of blood pressure. Hormones, which increase or decrease the arterial blood pressure are listed in Table 103.2.

■ HORMONES WHICH INCREASE BLOOD PRESSURE

Hormones, which increase the arterial blood pressure have different mechanism of action.

1. *Adrenaline*

Adrenaline is secreted by the adrenal medulla. It is also released by sympathetic postganglionic nerve endings. Adrenaline regulates the blood pressure by acting through heart and blood vessels. It increases systolic pressure by increasing the force of contraction of the heart and cardiac output. It decreases diastolic pressure by reducing the total peripheral resistance.

Adrenaline causes constriction of blood vessels through alpha receptors. It also causes dilatation of blood vessels through β_2-receptors in some areas of the body like skeletal muscle, liver and heart. So, the total peripheral resistance is reduced leading to decrease in diastolic pressure (Chapter 71).

2. *Noradrenaline*

Noradrenaline is secreted by the adrenal medulla. It is also released by sympathetic postganglionic nerve endings. Noradrenaline increases diastolic pressure due to its general vasoconstrictor effect (Chapter 71). It has stronger effects on blood vessels than on the heart. It causes constriction of all blood vessels throughout the body via alpha receptors. So it is called **'general vasoconstrictor'.** The action of noradrenaline is to increase the total peripheral resistance and diastolic pressure.

It also increases the systolic pressure slightly, by increasing the force of contraction of heart.

3. *Thyroxine*

Thyroxine secreted form thyroid gland increases systolic pressure but decreases the diastolic pressure. It increases the systolic pressure by increasing cardiac output. The cardiac output is increased because of increase in the blood volume and force of contraction of the heart (Chapter 67).

Thyroxine has indirect action on diastolic pressure. Large quantities of metabolites are produced during increased metabolic activity induced by thyroxine. These metabolites cause vasodilatation, leading to decrease in peripheral resistance. It causes decrease in diastolic pressure.

Generally, mean arterial pressure is not altered by the activity of thyroxine. Systolic pressure is increased and the diastolic pressure is decreased. So, only the pulse pressure increases.

4. *Aldosterone*

Aldosterone is secreted from adrenal cortex. It causes retention of sodium and water and thereby, increases the ECF fluid volume and blood volume, leading to increase in blood pressure. Thus, an increase in the secretion of aldosterone increases the blood pressure by increasing the blood volume (Chapter 70).

5. *Vasopressin*

Vasopressin or ADH, which is secreted by posterior pituitary has a potent action on the blood vessels, particularly the arteries. It causes constriction of the arteries in all parts of the body. Due to the vasoconstriction, the blood pressure is increased. However, the amount of this hormone required to cause the **vasopressor effect** is very much high than the amount required to cause the **antidiuretic effect** (Chapter 66).

6. *Angiotensins*

Angiotensin II, III and IV, which are obtained from angiotensinogen cause constriction of systemic arterioles and elevate blood pressure (Chapter 50).

7. *Serotonin*

Serotonin is otherwise known as **5-hydroxytryptamine.** Serotonin is secreted from many sources (refer Chapter 73 for details). It increases the blood pressure by vasoconstriction.

TABLE 103.2: Hormones involved in regulation of arterial blood pressure

Hormones which increase arterial blood pressure	Hormones which decrease arterial blood pressure
1. Adrenaline*	1. Vasoactive intestinal polypeptide (VIP)
2. Noradrenaline	2. Bradykinin
3. Thyroxine*	3. Prostaglandin
4. Aldosterone	4. Histamine
5. Vasopressin	5. Acetylcholine
6. Angiotensin	6. Atrial natriuretic peptide
7. Serotonin	7. Brain natriuretic peptide
	8. C-type natriuretic peptide

*Adrenaline and thyroxine increase systolic pressure but decrease diastolic pressure.

■ HORMONES WHICH DECREASE BLOOD PRESSURE

Following hormones decrease the arterial blood pressure by causing vasodilatation:

1. *Vasoactive Intestinal Polypeptide*

Vasoactive intestinal polypeptide (VIP) is secreted in the stomach and small intestine. A small amount of this hormone is also secreted in large intestine. VIP is a vasodilator and causes dilatation of peripheral blood vessels and decrease in blood pressure.

2. *Bradykinin*

Bradykinin is produced in blood during the conditions like inflammation. During such conditions, the enzyme in the blood called kallikrein is activated. It acts on α_2-globulin to form kallidin, which is converted into bradykinin (Chapter 73).

Bradykinin is a vasodilator substance and causes reduction in blood pressure.

3. *Prostaglandins*

Prostaglandin PGE_2 is a vasodilator substance. It is secreted from almost all tissues of the body (Chapter 73). It decreases blood pressure.

4. *Histamine*

Histamine is secreted in nerve endings of hypothalamus, limbic cortex and other parts of cerebral cortex. Histamine is also released from tissues during allergic conditions, inflammation or damage (Chapter 73).

Histamine causes vasodilatation and decreases the blood pressure.

5. *Acetylcholine*

Acetylcholine is the cholinergic neurotransmitter released from many sources (Chapter 73). Acetylcholine causes vasodilatation and decreases the blood pressure.

6. *Atrial Natriuretic Peptide*

Atrial natriuretic peptide (ANP) is a hormone secreted by the atrial musculature of heart. It causes dilatation of blood vessels and decreases the blood pressure (Chapter 72).

7. *Brain Natriuretic Peptide*

Brain natriuretic peptide (BNP) is a hormone secreted by the atrial musculature of heart. Like ANP, this hormone also causes dilatation of blood vessels and decreases the blood pressure (Chapter 72).

8. *C-type Natriuretic Peptide*

C-type natriuretic peptide (CNP) is secreted by several tissues including myocardium and vascular endothelium (Chapter 71). CNP decreases blood pressure by vasodilatation.

■ LOCAL MECHANISM FOR REGULATION OF BLOOD PRESSURE

In addition to nervous, renal and hormonal mechanisms, some local substances also regulate the blood pressure. The local substances regulate the blood pressure by vasoconstriction or vasodilatation.

■ LOCAL VASOCONSTRICTORS

Local vasoconstrictor substances are derived from vascular endothelium. These substances are called **endothelium-derived constricting factors** (EDCF). Common EDCF are endothelins (ET), which are peptides with 21 amino acids. Three types of endothelins ET1, ET2 and ET3 are identified so far.

Endothelins are produced by stretching of blood vessels. These peptides act by activating phospholipase, which in turn activates prostacyclin and thromboxane A_2. These two substances cause constriction of blood vessels and increase the blood pressure.

■ LOCAL VASODILATORS

Local vasodilators are of two types:

1. Vasodilators of metabolic origin
2. Vasodilators of endothelial origin.

Vasodilators of Metabolic Origin

Vasodilators of metabolic origin are carbon dioxide, lactate, hydrogen ions and adenosine (Table 103.3).

Vasodilators of Endothelial Origin

Nitric oxide (NO) is an endothelium-derived relaxing factor (EDRF). It is synthesized from arginine. Nitric oxide synthesis is stimulated by acetylcholine, bradykinin, VIP, substance P and platelet breakdown products. As nitric oxide is a vasodilator, deficiency of this leads to constant vasoconstriction and hypertension.

Other functions of nitric oxide are penile erection with vasodilatation and engorgement of corpora cavernosa, activation of macrophages in brain, destruction of cancer cells and relaxation of smooth muscles of gastrointestinal tract.

Types of nitric oxide

i. NO_3 (nitrate)
ii. NO^+ (nitrosonium cation)
iii. NO^- (nitroxyl anion).

■ MEASUREMENT OF ARTERIAL BLOOD PRESSURE

Blood pressure was first measured in horse in 1733, by **Stephen Hales,** with a long tube of about 9 feet length. Later, **Poiseuille** reduced the length of the tube to one foot and used mercury to balance the column of blood.

In 1847, **Ludwig** placed a float on the top of **mercury column** and made continuous recording possible. Introduction of rubber tubing, anesthesia and **manometer** enabled the accurate measurement of blood pressure.

Blood pressure is measured by two methods:

A. Direct method
B. Indirect method.

■ DIRECT METHOD

Direct method to measure arterial blood pressure is employed only in animals. Animal is given suitable anesthesia, then the neck is opened and a tracheal cannula is inserted into the trachea. This tracheal cannula is connected to a respiratory pump, so that the respiration in the animal is controlled artificially to avoid any disturbance during the experimental procedure. A venous cannula is inserted through the femoral vein. It is used to infuse saline to compensate blood loss during experimental procedure.

TABLE 103.3: Local substances involved in the regulation of arterial blood pressure

Local vasoconstrictors (Endothelins)	Local vasodilators	
	Metabolic products	Endothelins (ET)
EDCF: 1. ET1 2. ET2 3. ET3	1. Carbon dioxide 2. Lactate 3. Hydrogen 4. Adenosine	EDRF: 1. Nitric oxide

Carotid artery is cannulated and connected to a **mercury manometer.** By using a **kymograph,** the blood pressure can be recorded continuously in the form of graph. The **cannula** inserted into carotid artery can also be connected to an electronic pressure transducer, which in turn is connected to a recording device like **polygraph** to obtain the recordings.

■ INDIRECT METHOD

Indirect method is used to measure arterial blood pressure in man as well as in animals.

Apparatus

Apparatus used to measure blood pressure in human beings is called **sphygmomanometer.** Along with sphygmomanometer, **stethoscope** is also necessary to measure blood pressure.

Principle

When an external pressure is applied over the artery, the blood flow through it is obstructed. And the pressure required to cause occlusion of blood flow indicates the pressure inside the vessel.

Procedure

Brachial artery is usually chosen because of convenience. The arm cuff of sphygmomanometer is tied around upper arm, above the cubital fossa. Cuff should not be too tight or too loose. It is connected to sphygmomanometer. Now, blood pressure can be measured by three methods.

1. Palpatory method
2. Auscultatory method
3. Oscillatory method.

1. *Palpatory method*

First, the **radial pulse** is felt. While feeling the pulse, pressure is increased in the cuff by inflating air into it, with the help of a hand pump. While doing this, mercury column in the sphygmomanometer shows the pressure in the cuff.

When pressure is increased in the arm cuff, brachial artery is compressed and blood flow is obstructed. So,

radial pulse disappears. When radial pulse disappears, the pressure is further increased by about 20 mm Hg. Then, the pressure in the cuff is slowly reduced by releasing the valve of the hand pump, i.e. the cuff is deflated slowly. This is done by feeling the pulse and simultaneously watching the mercury column in the apparatus. Pressure is noted when the pulse reappears. This pressure indicates the systolic pressure.

Disadvantage of palpatory method is that the diastolic pressure cannot be measured.

2. *Auscultatory method*

Auscultatory method is the most accurate method to determine arterial blood pressure. After determining the systolic pressure in palpatory method, the pressure in the cuff is raised by about 20 mm Hg above that level, so that the brachial artery is occluded due to compression. Now, the chest piece of the stethoscope is placed over the antecubital fossa and the arm cuff is slowly deflated. While doing so, series of sounds are heard through the stethoscope. These sounds are known as **Korotkoff sounds,** named after the discoverer Korotkoff (1905). While reducing the pressure, Korotkoff sounds have five phases:

First phase – appearance of tapping sound

While decreasing the pressure from arm cuff, the occlusion of the artery is relieved and when blood starts flowing through the artery, first sound appears suddenly. In a normal person, it appears, when the pressure is reduced to 120 mm Hg. It is a clear **tapping sound.**

Appearance of tapping sound indicates **systolic pressure.** When the pressure is reduced further by 10 mm Hg from the initial level, this sound slowly becomes louder.

Second phase – appearance of murmuring sound

Following the clear taping sound, a murmuring sound is heard when the pressure is reduced further by about 15 mm Hg.

Third phase – appearance of gong sound

After the murmuring sound, a very clear and louder sound is heard. It is of **gong type.** It is heard while reducing the pressure by another 15 mm Hg.

Fourth phase – appearance of muffled sound

Next to the gong type sound, a mild and muffled sound is heard when the pressure is decreased further by 5 mm Hg.

Fifth phase – disappearance of muffled sound

Muffling sound disappears. **Disappearance** of this sound indicates **diastolic pressure.**

Thus, in auscultatory method, the appearance of clear tapping sound during first phase indicates the systolic pressure and disappearance of the muffling sound in fifth phase shows diastolic pressure.

3. *Oscillatory method*

When pressure in the arm cuff is increased above the level of systolic pressure, the artery is occluded due to compression. At this stage, the mercury column in the manometer remains static. When the pressure is gradually reduced, some oscillations occur at the top of the mercury column. While deflating the cuff further, the amplitude and duration of oscillations increase suddenly. It denotes systolic pressure. When the cuff pressure is reduced further, the amplitude and duration of oscillations is reduced. It reflects the diastolic pressure.

Because of its inaccuracy, this method is not followed in routine clinical practice. By connecting the manometer to an appropriate recording device, the oscillations of mercury column can be recorded graphically.

Automatic Blood Pressure Instrument

Nowadays automatic blood pressure instrument is widely used. The instrument has a **microprocessor-driven air pump,** which automatically inflates the arm cuff at a fixed pressure value. Then, it records the pressure oscillation pattern during a stepwise deflation. The principle of measuring pressure depends up on the non-linear properties of brachial arterial wall, which induce non-constant oscillations of the cuff pressure during deflation. The sensors in the instrument detect the oscillometric waves and determine the systolic pressure, diastolic pressure, pulse pressure and mean arterial pressure. The instrument determines the pulse rate also.

Automatic instrument does not need expert personnel to measure the blood pressure since it has the self-measuring facilities. However, the accuracy of oscillometric method is still controversial.

Microprocessor controlled blood pressure **monitors** that are fixed around wrist or finger are also available.

■ APPLIED PHYSIOLOGY

Pathological variations of arterial blood pressure:

A. Hypertension
B. Hypotension.

■ HYPERTENSION

Definition

Hypertension is defined as the persistent high blood pressure. Clinically, when the systolic pressure remains

elevated above 150 mm Hg and diastolic pressure remains elevated above 90 mm Hg, it is considered as hypertension. If there is increase only in systolic pressure, it is called **systolic hypertension.**

Types of Hypertension

Hypertension is divided into two types:
1. Primary hypertension or essential hypertension
2. Secondary hypertension.

1. Primary Hypertension or Essential Hypertension

Primary hypertension is the elevated blood pressure in the absence of any underlying disease. It is also called essential hypertension. Arterial blood pressure is increased because of increased peripheral resistance, which occurs due to some unknown cause.

Primary hypertension is of two types:
i. Benign hypertension
ii. Malignant hypertension.

i. *Benign hypertension*

Benign hypertension is the high blood pressure that does not cause any problem. It is defined as the essential hypertension that runs a relatively long and symptomless course. In early stages of this condition, there is moderate increase in blood pressure, with systolic pressure of 200 mm Hg and the diastolic pressure of about 100 mm Hg. However, in resting conditions and sleep, the blood pressure returns to normal level. Later, there is a further increase in blood pressure and it does not come back to normal level in resting conditions. Persistent increase in pressure over the years causes development of vascular, cardiac or renal diseases.

ii. *Malignant hypertension*

Malignant hypertension is a severe form of hypertension with a rapid course leading to progressive cardiac and renal diseases. It is also called **accelerated hypertension.** In this case, the blood pressure is elevated to a great extent. Systolic pressure rises to about 250 mm Hg and diastolic pressure rises to 150 mm Hg. It is always developed due to the combined effects of primary and secondary hypertension. Malignant hypertension cause severe damage of tunica intima of small blood vessels and organs like eye (retina), heart, brain and kidneys. It is a **fatal disease,** since it causes death within few years.

2. Secondary Hypertension

Secondary hypertension is the high blood pressure due to some underlying disorders. The different forms of secondary hypertension are:

i. *Cardiovascular hypertension*

Cardiovascular hypertension is produced due to the cardiovascular disorders such as:
a. Atherosclerosis: Hardening of blood vessels due to fat deposition
b. Coarctation of aorta: Narrowing of aorta.

ii. *Endocrine hypertension*

Endocrine hypertension is developed because of hyperactivity of some endocrine glands:
a. Pheochromocytoma: Tumor in adrenal medulla, resulting in excess secretion of catecholamines
b. Hyperaldosteronism: Excess secretion of aldosterone from adrenal cortex
c. Cushing syndrome: Excess secretion of glucocorticoids from adrenal cortex.

iii. *Renal hypertension*

Renal diseases causing hypertension:
a. Stenosis of renal arteries
b. Tumor of juxtaglomerular cells, leading to excess production of angiotensin II
c. Glomerulonephritis.

iv. *Neurogenic hypertension*

Nervous disorders producing hypertension:
a. Increased intracranial pressure
b. Lesion in tractus solitarius
c. Sectioning of nerve fibers from carotid sinus.

v. *Hypertension during pregnancy*

Some pregnant women develop hypertension because of **toxemia of pregnancy.** Arterial blood pressure is elevated by the low glomerular filtration rate and retention of sodium and water. It may be because of some autoimmune processes during pregnancy or release of some vasoconstrictor agents from placenta or due to the excessive secretion of hormones causing rise in blood pressure. Hypertension is associated with **convulsions** in **eclampsia** (Chapter 84).

Experimental Hypertension

Hypertension can be produced in experimental animals by various methods. These methods correlate with the causes of hypertension in human beings.

Experimental hypertension is produced by the following methods:
1. Clamping the renal artery
2. Denervation of baroreceptors in carotid sinus and aortic arch

3. Injections of corticosteroids
4. Infusion of salt with aldosterone.

Goldblatt hypertension

Goldblatt hypertension is one of the experimental hypertension produced in dogs by Goldblatt and it is named after him. He removed one kidney of the dog and clamped the artery of other kidney. It produced slow and steady increase in arterial pressure. The elevation of blood pressure was due to excessive secretion of renin from intact kidney, leading to the formation of a large quantity of angiotensin II. It is known as 'one kidney Goldblatt hypertension'. Hypertension is also developed when the artery of one kidney is clamped without doing anything with the kidney of the other side. It is called 'two kidney Goldblatt hypertension'. It is due to the renin-angiotensin mechanism and retention of salts.

Manifestations of Hypertension

Severe manifestations of primary hypertension:

1. Renal failure
2. Left ventricular failure
3. Myocardial infarction
4. Cerebral hemorrhage
5. Retinal hemorrhage.

Treatment of Hypertension

Secondary hypertension is cured by treating the disease causing hypertension. Primary hypertension can be controlled but cannot be cured.

Following are the **antihypertensive drugs** to control primary hypertension:

1. *Beta adrenoceptor blockers*

Beta adrenoceptor blockers or **beta antagonists (adrenergic beta blockers** or beta blockers) block the effect of sympathetic nerves on heart and blood vessels by binding with beta adrenoceptors, so that there is reduction in cardiac output and inhibition of vasoconstriction, leading to fall in blood pressure.

2. *Alpha adrenoceptor blockers*

Alpha adrenoceptor blockers or alpha **antagonists (adrenergic alpha blockers** or alpha blockers) block the effect of sympathetic nerves on blood vessels by binding with alpha adrenoceptors, leading to vasodilatation and fall in blood pressure.

3. *Calcium channel blockers*

Calcium channel blockers are drugs, which block the calcium channels in myocardium and thereby, reduce the contractility of myocardium. It causes decrease in cardiac output and fall in blood pressure.

4. *Vasodilators*

Vasodilator agents reduce blood pressure by vasodilatation.

5. *Diuretics*

Diuretics cause diuresis and reduce the ECF volume and blood volume. So, blood pressure is decreased.

6. *Inhibitors of angiotensin-converting enzyme (ACE inhibitors)*

ACE inhibitors reduce the blood pressure by blocking the formation of angiotensin.

7. *Depressors of vasomotor center*

Depressor drugs act on vasomotor center and reduce the vasomotor tone. So, vasoconstriction is prevented.

8. *Angiotensin II receptor blockers*

Angiotensin II receptor blockers or antagonists are the antihypertensive drugs that decrease the blood pressure by blocking the effect of angiotensin II (vasoconstriction and secretion of aldosterone).

■ HYPOTENSION

Definition

Hypotension is the low blood pressure. When the systolic pressure is less than 90 mm Hg, it is considered as hypotension.

Types

1. Primary hypotension
2. Secondary hypotension.

1. *Primary hypotension*

Primary hypotension is the low blood pressure that develops in the absence of any underlying disease and develops due to some unknown cause. It is also called **essential hypotension.** Frequent fatigue and weakness are the common symptoms of this condition. However, the persons with primary hypotension are not easily susceptible to heart or renal disorders.

2. *Secondary hypotension*

Secondary hypotension is the hypotension that occurs due to some underlying diseases. Diseases, which cause hypotension are:

i. Myocardial infarction
ii. Hypoactivity of pituitary gland
iii. Hypoactivity of adrenal glands

iv. Tuberculosis
v. Nervous disorders.

Orthostatic hypotension

Orthostatic hypotension is the sudden fall in blood pressure while standing for some time. It is due to the effect of gravity. It develops in persons affected by myasthenia gravis or some nervous disorders like tabes dorsalis, syringomyelia and diabetic neuropathy. Common symptom of this condition is **orthostatic syncope.** Syncope is described in detail in Chapter 116.

Venous Pressure

Chapter 104

- DEFINITION AND NORMAL VALUES
  - VENOUS PRESSURE IN EXTREMITIES OF THE BODY
  - VENOUS PRESSURE IN CENTRAL AND PERIPHERAL VEINS
- VARIATIONS OF VENOUS PRESSURE
  - PHYSIOLOGICAL VARIATIONS
  - PATHOLOGICAL VARIATIONS
- MEASUREMENT
  - DIRECT METHOD
  - INDIRECT METHOD
- FACTORS REGULATING VENOUS PRESSURE
  - LEFT VENTRICULAR CONTRACTION OR *VIS A TERGO*
  - RIGHT ATRIAL PRESSURE OR *VIS A FRONTE*
  - RESISTANCE OR *VIS A LATRE*
  - VOLUME OF VENOUS BLOOD
  - PERIPHERAL RESISTANCE
  - GRAVITY AND POSTURE
- EFFECT OF RESPIRATION ON VENOUS PRESSURE
  - VALSALVA MANEUVER
  - MÜELLER MANEUVER

DEFINITION AND NORMAL VALUES

Venous pressure is the pressure exerted by the contained blood in the veins. The pressure in vena cava and right atrium is called **central venous pressure.** The pressure in peripheral veins is called **peripheral venous pressure.**

Pressure is not same in all the veins. It varies in different veins in the extremities of the body and also varies from central veins to peripheral veins.

VENOUS PRESSURE IN EXTREMITIES OF THE BODY

Venous pressure is less in the parts of the body above the level of the heart and it is more in parts below the level of the heart. Pressure in:

Jugular vein: 5.1 mm Hg (6.9 cm H_2O)
Dorsal venous arch of foot: 13.2 mm Hg (17.9 cm H_2O).
(1 mm Hg pressure = 1.359 cm H_2O pressure)

VENOUS PRESSURE IN CENTRAL AND PERIPHERAL VEINS

Pressure is greater in peripheral veins than in central veins. Pressure in:

Antecubital vein: 7.1 mm Hg (9.6 cm H_2O)
Superior vena cava: 4.6 mm Hg (6.2 cm H_2O).

VARIATIONS OF VENOUS PRESSURE

Venous pressure is altered both in physiological and pathological conditions.

■ PHYSIOLOGICAL VARIATIONS

Venous pressure increases in:

1. Changing from standing to supine position
2. Tilting the body
3. Forced expiration (Valsalva maneuver)
4. Contraction of abdominal and limb muscles
5. Effect of gravity during prolonged travelling or standing
6. Excitement.

■ PATHOLOGICAL VARIATIONS

Venous pressure increases in:

1. Low cardiac output
2. Congestive heart failure
3. Venous obstruction
4. Failure of valves in veins
5. Paralysis of muscles
6. Immobilization of parts of body
7. Renal failure.

Venous pressure decreases in:

1. Severe hemorrhage
2. Surgical shock.

■ MEASUREMENT OF VENOUS PRESSURE

■ DIRECT METHOD

Central venous pressure is measured by a catheter introduced through median cubital vein of forearm. Position of tip of the catheter is checked by fluoroscopy. Other end of catheter is connected to a manometer, which measures the pressure. Peripheral venous pressure is measured by using a needle connected to a manometer.

■ INDIRECT METHOD

Measurement of venous pressure is done by using an apparatus designed by Ranger. By this apparatus, collapse of the vein is noticed by the reflection of light through a transparent device. Pressure required to cause the collapse of peripheral vein denotes the pressure in the particular vein.

■ FACTORS REGULATING VENOUS PRESSURE

■ 1. LEFT VENTRICULAR CONTRACTION OR *VIS A TERGO*

Left ventricular contraction is also called *vis a tergo* or force from behind. It forces the blood through the arteries, arterioles, capillaries and veins to the right atrium. Venous pressure is **directly proportional** to left ventricular pressure. By the time blood passes through capillaries and reaches the venules, the pressure becomes less than 8 mm Hg and when it reaches right atrium, the pressure may be less than 1 mm Hg.

■ 2. RIGHT ATRIAL PRESSURE OR *VIS A FRONTE*

Right atrial pressure is also called *vis a fronte* or force from front. It determines the venous return. It is also called central venous pressure, which in turn regulates the peripheral venous pressure. Normal right atrial pressure is 0 mm Hg.

■ 3. RESISTANCE OR *VIS A LATRE*

Resistance offered to blood flow through the veins is also called *vis a latre* or force from side. Venous pressure is **directly proportional** to the resistance, which is due to venous tone and extravascular factors. Because of the thin-walled nature, veins and venules are compressed by the extravascular factors such as:

i. Compression of arm vein while passing over first rib
ii. Compression of neck veins in erect posture due to fall in pressure and by atmospheric pressure
iii. Compression of abdominal veins by increased intra-abdominal pressure
iv. Compression of veins while passing in between the muscles.

■ 4. VOLUME OF VENOUS BLOOD

Venous pressure is **directly proportional** to the volume of blood in the venous system.

■ 5. PERIPHERAL RESISTANCE

Venous pressure is **inversely proportional** to peripheral resistance. When peripheral resistance is more, arterioles constrict and the veins are filled with less blood. Hence, the pressure decreases. When peripheral resistance is less, the veins are filled with more blood and venous pressure increases.

■ 6. GRAVITY AND POSTURE

Pressure is more in the veins below the level of heart and the pressure is less in veins above the level of heart.

Weight of the column of blood in veins influences the venous pressure. During prolonged standing, the pressure in lower extremities is more (90 cm H_2O). It is

TABLE 104.1: Valsalva maneuver Vs Müeller maneuver

Features	Valsalva maneuver	Müeller maneuver
1. Intrathoracic pressure	Increases up to +50 mm Hg	Decreases up to –70 mm Hg
2. Central vein in thorax	Compressed	Dilated and blood rushes
3. Venous return to right atrium	Decreases	Increases
4. Peripheral venous pressure	Increases to 30 cm H_2O	Decreases to 3 cm H_2O
5. Central venous pressure	Decreases	Increases

because of pooling of blood in the legs due to gravity. It increases the weight of the column of blood, leading to increase in pressure. During the movement, the venous pressure in foot decreases.

In head region, the venous pressure is –10 cm H_2O because of the hydrostatic suction below the skull. So, there is always a negative venous pressure in the head.

■ EFFECT OF RESPIRATION ON VENOUS PRESSURE

During normal quiet breathing, the central venous pressure is altered in accordance with intrathoracic pressure. Thus, during inspiration, the central venous pressure decreases because of decreased intrathoracic pressure. During expiration, it increases because of increased intrathoracic pressure.

The effect of respiration on venous pressure is demonstrated by some procedures which exaggerate these effects on venous pressure. Such procedures are Valsalva maneuver and Mueller maneuver.

■ VALSALVA MANEUVER OR VALSALVA EXPERIMENT

Valsalva maneuver is the forced expiratory effort with closed glottis. It is performed by attempting to exhale forcibly, while keeping the mouth and nose closed.

Effects of Valsalva Maneuver

During this maneuver, the intrathoracic pressure becomes positive and increases greatly. It may reach +50 mm Hg. High intrathoracic pressure produces the following effects (Table 104.1):

1. Compression of central vein in thorax
2. Decrease in venous return to right atrium
3. Increase in peripheral venous pressure to about 30 cm H_2O, due to accumulation of blood in peripheral veins such as veins of neck, face and limbs
4. Decrease in central venous pressure.

Uses of Valsalva Maneuver

1. Valsalva maneuver is used as a diagnostic tool to evaluate the cardiovascular disorders. Best example is the 30 minutes endurance test.
2. Valsalva maneuver is practiced to relieve chest pain
3. It is used to correct the abnormal heart rhythms.

30 seconds endurance test

The subject is asked to blow against sphygmomanometer, in which the pressure is maintained at 40 mm Hg for 30 seconds. Then the changes in heart rate, blood pressure or murmurs are observed to evaluate the cardiovascular disorders.

■ MÜELLER MANEUVER OR MÜELLER EXPERIMENT

Müeller maneuver or experiment is the forced inspiratory effort with closed glottis. It is performed by attempting to inhale forcibly, while keeping the mouth and nose closed. It is also called reverse Valsalva maneuver.

Effects of Müeller Maneuver

During this maneuver, the intrathoracic pressure decreases greatly (becomes more negative). It is about –70 mm Hg. This pressure produces the following effects (Table 104.1):

1. Dilatation of right atrium and central vein because of increase in negative intrathoracic pressure
2. Rapid emptying of blood from peripheral veins into the central veins and increase in venous return to right atrium
3. Decrease in peripheral venous pressure to less than 3 to 4 cm H_2O
4. Increase in central venous pressure.

Uses of Müeller Maneuver

Müeller maneuver is used to evaluate:

1. Upper respiratory tract problems
2. Sleep apnea syndrome.

Capillary Pressure

Chapter 105

- INTRODUCTION
- REGIONAL VARIATIONS
- MEASUREMENT
- REGULATION
- CAPILLARY ONCOTIC PRESSURE

INTRODUCTION

Definition

Capillary pressure is the pressure exerted by the blood contained in capillary. It is also called **capillary hydrostatic pressure.**

Significance

Capillary pressure is responsible for the exchange of various substances between blood and interstitial fluid through capillary wall.

Normal Values

Generally, the pressure in the arterial end of the capillary is about 30 to 32 mm Hg and in venous end it is 15 mm Hg. However, capillary pressure varies depending upon the function of the organ or region of the body.

REGIONAL VARIATIONS

Regional variation in capillary pressure is in relation to the physiological activities of the particular region. So, it has some functional significance. Capillary pressure remarkably varies in kidneys and lungs.

Capillary Pressure in Kidneys

In kidneys, the glomerular capillary pressure is high. It is about 60 mm Hg. This high capillary pressure is responsible for glomerular filtration.

Capillary Pressure in Lungs

In lungs, the pulmonary capillary pressure is low and it is about 7 mm Hg. It favors exchange of gases between blood and alveoli.

MEASUREMENT

Direct Method

Capillary pressure was first measured by **EM Landis,** when he was a medical student. Minute vessels in the web of foot in a frog were cannulated by using micropipette, with a diameter of 5 µ at the tip with the aid of microscope. The cannula was connected to a manometer.

This method was later followed to measure capillary pressure in other organs.

Indirect Method

Indirect method is based upon the principle of exerting an external pressure necessary to obstruct the flow of blood in capillaries. The capillaries are observed under microscope.

REGULATION

Arterioles play an important role in regulating the capillary pressure and the pressure in capillaries is considered as a function of arteriolar resistance.

When the arterioles constrict, resistance increases in arterioles, which raises the arterial blood pressure. At

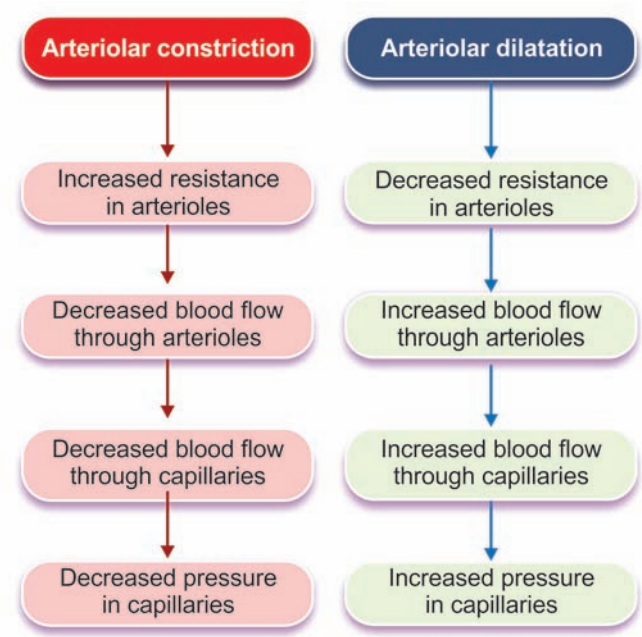

FIGURE 105.1: Regulation of capillary pressure

the same time, the volume of blood flowing into capillaries decreases, leading to fall in capillary pressure.

On the other hand, during dilatation of arterioles, the resistance decreases and arterial blood pressure decreases. But the capillary pressure increases because of increase in volume of blood flowing into capillaries (Fig. 105.1).

■ CAPILLARY ONCOTIC PRESSURE

Capillary membrane is permeable to all substances except plasma proteins. So, the plasma proteins stay within the capillaries and exert some pressure which is called oncotic pressure or colloidal osmotic pressure. Normal oncotic pressure is about 25 mm Hg. Among the plasma proteins, albumin exerts 70% of oncotic pressure.

Oncotic pressure plays an important role in filtration across capillary membrane, particularly in renal glomerular capillaries.

Arterial Pulse

Chapter 106

- INTRODUCTION
- TRANSMISSION OF PULSE
  - VELOCITY OF TRANSMISSION OF PULSE
  - DELAY IN TRANSMISSION OF PULSE
- METHODS OF RECORDING ARTERIAL PULSE
  - BY MANOMETER
  - BY DUDGEON SPHYGMOGRAPH
  - BY ELECTRONIC PULSE TRANSDUCER
- INTERPRETATION OF ARTERIAL PULSE TRACING
- PULSE POINTS
- EXAMINATION OF RADIAL PULSE
  - RATE
  - RHYTHM
  - CHARACTER
  - VOLUME
  - CONDITION OF THE BLOOD VESSEL WALL
  - DELAYED PULSE
- APPLIED PHYSIOLOGY – ABNORMAL PULSE
  - PULSUS DEFICIT
  - PULSUS ALTERNANS
  - ANACROTIC PULSE
  - THREADY PULSE OR WEAK PULSE
  - PULSUS PARADOXUS
  - WATER HAMMER PULSE
  - ABNORMAL PULSE IN PATENT DUCTUS ARTERIOSUS
  - ABNORMAL PULSE IN AORTIC REGURGITATION

INTRODUCTION

Arterial pulse is defined as the pressure changes transmitted in the form of waves through arterial wall and blood column from heart to periphery.

When heart contracts, the blood is ejected into aorta with great force. It causes distension of this blood vessel and a rise in pressure. A pressure wave is produced on the elastic wall of the aorta. It travels rapidly from the heart and can be felt after a brief interval, at any superficial peripheral artery like radial artery at wrist.

Pulse rate is the accurate measure of heart rate, except in conditions like pulses deficit (see below).

TRANSMISSION OF PULSE

Central arterial pulse is transmitted to the peripheral arteries as **peripheral arterial pulse.** Formation and transmission of pulse wave depends upon the elasticity of blood vessels. Thus, when the walls of the arteries are more distensible, the pressure rise is less and so the transmission of pulse is less. When the arterial wall

loses its elastic property and becomes rigid as in old age, the pressure rise is more and the transmission of pulse is also more.

Pulse is not transmitted to capillaries because capillaries are devoid of elastic tissues.

■ VELOCITY OF TRANSMISSION OF PULSE

Average velocity at which the pulse wave is transmitted varies between 7 and 9 meter/second. Pulse travels faster than the blood. Maximum velocity of blood flow in the body (in larger arteries) is only 50 cm/second.

■ DELAY IN TRANSMISSION OF PULSE

At the arteries, pulse is felt after a short interval from the beginning of ventricular systole. This delay is very small and it can be measured only by accurate recording. The delay is directly proportional to the distance from heart.

Delay of pulse at:

1. Common carotid artery: 0.01 to 0.02 second
2. Radial artery: About 0.08 second.

■ METHODS OF RECORDING ARTERIAL PULSE

■ BY MANOMETER

In animals, pulse is recorded by inserting a cannula into the dissected artery. This cannula is connected to a manometer or any recording device.

■ BY DUDGEON SPHYGMOGRAPH

Dudgeon sphygmograph is tied to the wrist in such a way that, a small plate rests on the skin over radial artery. Movements of arterial wall are magnified by a series of levers and are recorded on a moving strip of smoked paper. This instrument is outdated and it is replaced by electronic pulse transducers.

■ BY ELECTRONIC PULSE TRANSDUCER

Pulse transducer is placed over the finger and tied. This device throws light on the blood vessel through skin. Sensor of the transducer detects the light rays reflected from the flowing blood. Alteration in frequency of the reflected light rays is amplified and recorded by connecting the transducer to a recording device like polygraph. The record shows finger pulse volume, which represents the arterial pulse tracing.

■ INTERPRETATION OF ARTERIAL PULSE TRACING

Pulse recorded in **radial artery** or **femoral artery** is the **typical peripheral pulse** (Fig. 106.1). Peripheral pulse tracing has three main features:

1. *Anacrotic Limb*

Anacrotic limb or primary wave is the ascending limb or upstroke. It is due to the rise in pressure during systole.

2. *Catacrotic Limb*

Catacrotic limb is the descending limb or downstroke. It is due to the fall in pressure during diastole.

3. *Catacrotic Notch*

In the upper part of the catacrotic limb of pulse tracing, a small notch appears. It is known as catacrotic notch or incisura. This notch is produced by the backflow of blood during the closure of semilunar valves at the beginning of diastolic period, which produces slight increase in the pressure.

4. *Precatacrotic and Postcatacrotic Waves*

The wave appearing before the catacrotic notch is called precatacrotic wave. The wave appearing after the notch is called postcatacrotic wave.

■ PULSE POINTS

Usually, pulse is palpated on the radial artery because it is easily approachable and placed superficially. However, arterial pulse can be felt in different areas on the body. These areas are called pulse points. Pulse points and the area of palpation are given in Table 106.1.

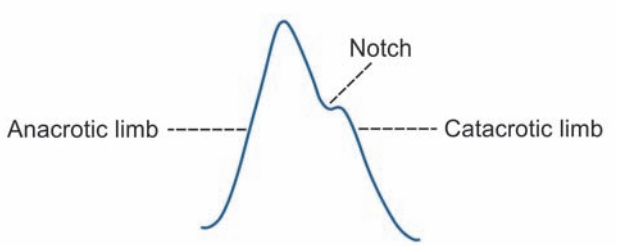

FIGURE 106.1: Radial pulse tracing

TABLE 106.1: Pulse points

Pulse point	Area of palpation
1. Temporal pulse	Over the temple, in front of ear on superficial temporal artery
2. Facial pulse	On facial artery at the angle of jaw
3. Carotid pulse	In the neck along anterior border of sternocleidomastoid muscle on common carotid artery
4. Axillary pulse	In axilla on axillary artery
5. Brachial pulse	In cubital fossa along medial border of biceps muscle on brachial artery
6. Radial pulse	Over the thumbside of wrist between tendons of brachioradialis and flexor carpi radialis muscles on radial artery
7. Ulnar pulse	Over the little fingerside of wrist on ulnar artery
8. Femoral pulse	In the groin on femoral artery
9. Popliteal pulse	Behind knee, in the popliteal fossa on popliteal artery
10. Dorsalis pedis pulse	Over the dorsum of foot on dorsalis pedis artery
11. Tibial pulse	Over the back of the ankle, behind medial malleolus on posterior tibial artery

■ EXAMINATION OF RADIAL PULSE

Examination of pulse is a valuable clinical procedure. Pulse represents the heartbeat. By examining pulse, important information regarding cardiac function such as rate of contraction, rhythmicity, etc. can be obtained. In addition, an experienced physician can determine the mean arterial pressure by hardness of pulse and its amplitude.

Method of Examining Radial Pulse

Subject is made to sit comfortably with forearm placed in mid or semi prone position, with wrist slightly flexed. The observer must stand by the right side of the subject. Tips of the middle three fingers (index finger, middle finger and ring finger) are placed over the radial artery below the wrist at the base of thumb. Light pressure is applied by the fingers until the pulse is felt. If necessary, the fingers are moved around till the pulse is felt.

Index finger is used to occlude blood flow from radial artery. **Ring finger** is used to occlude retrograde flow of blood from ulnar artery through palmar arch. **Middle finger** is used to assess the pulse.

Observations during Examination of Pulse

1. Rate
2. Rhythm
3. Character
4. Volume
5. Condition of blood vessel wall
6. Delayed pulse.

■ 1. RATE

Pulse rate is the number of pulse per minute. It has to be counted at least for 30 seconds. Pulse rate in adults is 72/minute.

Pulse Rate at Different Age

In fetus	:	150 to 180/minute
At birth	:	130 to 140/minute
At 10 years of age	:	90/minute
After puberty	:	72/minute.

Variations

Conditions that alter the heart rate alter pulse rate also.

Pulse rate increases during:

i. Exercise
ii. Pregnancy
iii. Emotional conditions
iv. Fever
v. Anemia
vi. Hypersecretion of catecholamines
vii. Hyperthyroidism.

Pulse rate decreases during:

i. Sleep
ii. Hypothermia
iii. Hypothyroidism
iv. Incomplete heart block.

■ 2. RHYTHM

Rhythm is the regularity of pulse. It refers to interval between beats. Under normal conditions, the pulse appears at regular intervals. Rhythm of the pulse becomes irregular in conditions like atrial fibrillation, extrasystole and other types of arrhythmia (Chapter 96).

Pulse with irregular rhythm is of two types:

i. Regularly irregular pulse
ii. Irregularly irregular pulse.

■ 3. CHARACTER

Character denotes the tension on the vessel wall produced by the waves of pulse. It is usually evaluated at right carotid artery. Normally, it is not possible to detect the different waves of the pulse or slight variations in the character or form of the pulse. However, it becomes more prominent in some abnormal conditions such as anacrotic pulse, water hammer pulse, pulsus paradoxus, etc. which are explained later in this chapter.

■ 4. VOLUME

Volume is the determination of movement of the vessel wall, produced by the transmission of pulse wave. It is also a measure of pulse pressure. It depends upon the condition of the blood vessel.

■ 5. CONDITION OF THE BLOOD VESSEL WALL

Condition of wall of the blood vessel is assessed by feeling the radial artery and rolling it against the underlying bones. Normally, the wall of the vessel is not palpable in children and young adults. However, in old age the wall of the vessel becomes rigid and palpable. In abnormal conditions like arteriosclerosis, it is felt as a hard rope.

■ 6. DELAYED PULSE

Sometimes, the arrival of pulse in certain peripheral arteries is delayed. It is an important feature to be noted because it is useful in diagnosis of certain diseases.

Types of delayed pulse:

i. Femoral delay
ii. Radial-radial delay.

i. *Femoral Delay*

While palpating radial pulse and femoral pulse simultaneously, there is a short delay in the arrival of femoral pulse wave. Normally, it is negligible and unnoticed. However, the prolonged or noticeable delay in the arrival of femoral pulse indicates coarctation (narrowing) of aorta. This delay is called femoral delay, **radial femoral delay** or **radiofemoral delay.**

ii. *Radial-radial Delay*

When both the radial pulses are examined simultaneously, sometimes the arrival of pulse is delayed on one side. It is called **radio-radial delay** or radial-radial delay or **radial-radial inequality.** This indicates the narrowing of large artery due to atherosclerosis.

■ APPLIED PHYSIOLOGY – ABNORMAL PULSE

■ 1. PULSUS DEFICIT

Pulsus deficit is the abnormal condition in which the pulse rate is less than the heart rate. It occurs in atrial fibrillation, when the stroke volume is reduced. Because of reduced stroke volume, some of the pulse waves become weak and disappear before reaching the peripheral arteries. Pulsus deficit is the only condition in which pulse rate is less than the heart rate.

■ 2. PULSUS ALTERNANS

Pulsus alternans is the abnormal condition in which the amplitude of every second wave in pulse tracing is relatively smaller. It is because of the alternate variation in the force of ventricular contraction. However, the rhythm of the pulse is not altered. It is common in severe myocardial diseases, paroxysmal tachycardia and atrial fibrillation.

■ 3. ANACROTIC PULSE

Anacrotic pulse is the abnormal pulse, characterized by a slow ascending limb which has a notch called anacrotic notch. It is produced in aortic stenosis, when ejection is slow.

■ 4. THREADY PULSE OR WEAK PULSE

Thready or weak pulse is the abnormal pulse in which the volume of pulse becomes very feeble and is hardly felt at the arteries. It usually occurs whenever the stroke volume decreases or when there is severe vasoconstriction, as in the case of severe hemorrhage or severe chills. In these conditions, the sympathetic activity increases enormously, leading to generalized vasoconstriction.

■ 5. PULSUS PARADOXUS

Pulsus paradoxus is the condition when the pulse becomes very strong and very weak alternately, in

relation to respiratory cycle. Normally, there is a slight increase in volume of pulse during inspiration and slight decrease in volume during expiration. But, it is hardly noticed. However, when it becomes very prominent, it is pathological. This type of pulse is noticed in cardiac tamponade (Chapter 100). It is also noticed in physiological conditions such as deep breathing.

■ 6. WATER HAMMER PULSE

Water hammer pulse is the abnormal pulse, characterized by a rapid upstroke and an equally rapid downstroke. It is also called **collapsing** or **Corrigan pulse.** It is seen in conditions like aortic regurgitation, patent ductus arteriosus and arteriovenous fistula. It is best felt by raising the arm of the subject and holding it by grasping the wrist with palm of the observer.

■ 7. ABNORMAL PULSE IN PATENT DUCTUS ARTERIOSUS

Patent ductus arteriosus is the permanent existence of ductus arteriosus. In fetus, the lungs are nonfunctioning. So, the blood which is pumped by right ventricle into the pulmonary artery, is diverted to systemic aorta through ductus arteriosus. Ductus arteriosus closes after birth. However, in some cases, it exists without closing (Fig. 106.2).

Pulse pressure wave is very much altered in this condition. Since, the blood flows from systemic aorta to pulmonary artery, after every ventricular systole, the blood flows out of aorta quickly. It decreases the diastolic pressure and the catacrotic limb of the pulse tracing falls below the level of 80 mm Hg.

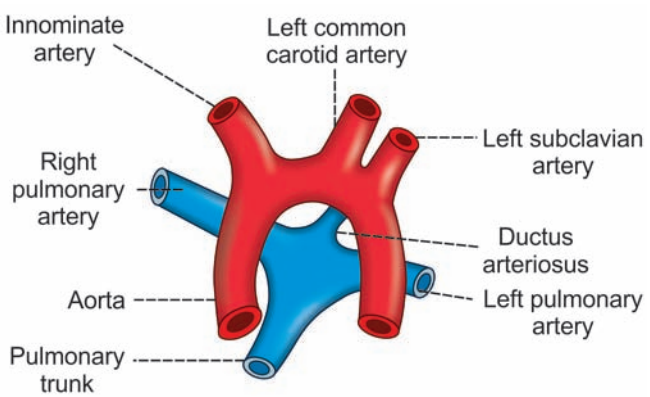

FIGURE 106.2: Diagram showing ductus arteriosus

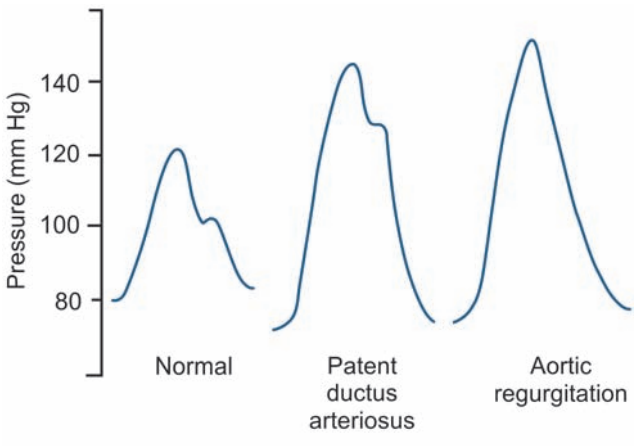

FIGURE 106.3: Radial pulse tracing in patent ductus arteriosus and aortic regurgitation

Flow of blood from aorta to pulmonary artery increases the venous return to left side of the heart. So, left ventricular output increases, which in turn elevates the systolic pressure in arteries. Thus, in pulse tracing, the peak of the pulse wave is elevated above the level of 120 mm Hg. So, the pulse tracing in this condition reveals the increased pulse pressure (Fig. 106.3).

■ 8. ABNORMAL PULSE IN AORTIC REGURGITATION

Aortic regurgitation is the backflow of blood from aorta into left ventricle. It is common during incompetence of semilunar valve in aorta. It decreases the diastolic pressure. Because of backflow of blood, the left ventricular filling increases greatly, leading to increase in output and systolic pressure. Thus, the pulse tracing in aortic regurgitation is more or less similar to that in patent ductus arteriosus. Only difference is that in the tracing during aortic regurgitation, the incisura is very mild. And in severe conditions, when the aortic valve does not close, the incisura is absent (Fig. 106.3).

Venous Pulse

Chapter 107

- INTRODUCTION
- SIGNIFICANCE
- EXAMINATION OF VENOUS PULSE
- METHODS TO RECORD VENOUS PULSE
- RECORDING OF VENOUS PULSE – JUGULAR VENOUS PULSE TRACING
- APPLIED PHYSIOLOGY – ABNORMAL VENOUS PULSE
  - ELEVATED JUGULAR VENOUS PULSE
  - KUSSMAUL SIGN
  - ABNORMALITIES OF WAVES IN JUGULAR PULSE TRACING

INTRODUCTION

Venous pulse is defined as the pressure changes transmitted in the form of waves from right atrium to veins near heart. Venous pulse is observed only in larger veins near the heart such as jugular vein.

Observation of venous pulse is an integral part of the physical examination because it reflects right atrial pressure and the hemodynamic events in right atrium.

SIGNIFICANCE

1. Venous pulse recording is used to determine the rate of atrial contraction, just as the record of arterial pulse is used to determine the rate of ventricular contraction
2. Many phases of cardiac cycle can be recognized by means of venous pulse tracing
3. Venous pulse tracing is the simple and accurate method to measure the duration of different phases in diastole
4. Venous pulse also represents the atrial pressure changes taking place during cardiac cycle.

EXAMINATION OF VENOUS PULSE

Inspection of jugular vein pulsations is routinely done by bedside examination of neck veins. It provides valuable information about the cardiac function.

To observe the pulsation of internal jugular vein, head of the subject is tilted upwards at 45°. However, in patients with increased venous pressure, the head should be tilted as much as 90°. Pulsations of jugular vein can be noticed when light is passed across the skin overlying internal jugular vein with relaxed neck muscles. Simultaneous palpation of the left carotid artery helps the examiner confirm the venous pulsations.

METHODS TO RECORD VENOUS PULSE

A small **funnel** covered by thin **rubber membrane** is placed over the skin at the level of external jugular vein, in the **supraclavicular fossa.** Slight pressure is exerted to provide perfect contact between edge of the funnel and skin.

Pressure changes in the vein cause some oscillations in rubber membrane through the skin. The oscillations are transmitted through rubber tube to a recording device like **Marey tambour.** Nowadays, **electronic transducer** is used for this purpose.

The subject should be in such a position so as to avoid the effect of gravity, which tends to empty veins and reduce the amplitude of the venous pulse.

RECORDING OF VENOUS PULSE – JUGULAR VENOUS PULSE TRACING

Recording of jugular venous pulse is called **phlebogram.** It is similar to intra-atrial pressure curve (Fig. 107.1).

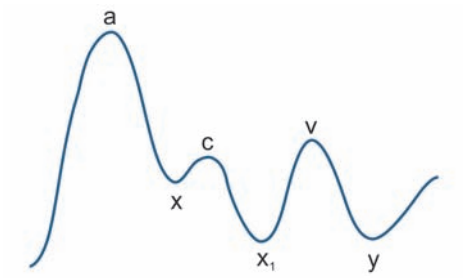

FIGURE 107.1: Phlebogram

Like intra-atrial pressure curve, phlebogram also has three positive waves, namely a, c, v and three negative waves namely x, x_1, y.

'a' Wave

'a' wave is the first positive wave. It is due to rise in atrial pressure during **atrial systole.** It precedes ventricular systole.

'x' Wave

'x' wave is a negative wave due to fall in atrial pressure. It coincides with **atrial diastole** and beginning of **ventricular systole.**

'c' Wave

'c' wave is a positive wave due to rise in atrial pressure during **isometric contraction period.** During this period, the atrioventricular valves bulge into the atria and increase the pressure in the atria slightly.

Earlier, it was thought that this wave was due to transmission of pulse from neighboring carotid artery. Hence, it was called 'c' wave.

'x_1' Wave

'x_1' wave is a negative wave due to fall in atrial pressure during **ejection period.** During ejection period, the atrioventricular ring is pulled towards ventricles causing distention of atria. So, the atrial pressure falls.

'v' Wave

'v' wave is a positive wave due to rise in atrial pressure. The pressure increases because of filling of atria (venous return). It is obtained during **isometric relaxation period** or during atrial diastole.

'y' Wave

'y' wave is a negative wave which denotes fall in atrial pressure. Pressure falls due to the opening of atrioventricular valve and emptying of blood into the ventricle. This wave appears during **rapid and slow filling periods.** 'y' wave is followed by 'a' wave and the cycle is repeated.

■ APPLIED PHYSIOLOGY – ABNORMAL VENOUS PULSE

■ ELEVATED JUGULAR VENOUS PULSE

Elevated jugular venous pulse indicates the rise in right ventricular pressure.

It occurs in:

1. Bradycardia
2. Pericardial effusion
3. Constrictive pericarditis
4. Tricuspid stenosis
5. Pulmonary hypertension.

■ KUSSMAUL SIGN

Kussmaul sign is the increase in venous distention and venous pressure. Normally, it occurs during inspiration.

Pathological Conditions when Kussmaul Sign occurs

1. Cardiac tamponade
2. Constrictive pericarditis
3. Restrictive cardiomyopathy
4. Right ventricular infarction.

■ ABNORMALITIES OF WAVES IN JUGULAR PULSE TRACING

1. *Elevation of 'a' Wave*

Elevation of 'a' wave occurs in:

i. Tricuspid stenosis
ii. Pulmonary hypertension.

2. *Cannon 'a' Wave*

Giant 'a' wave with abrupt fall (downward deflection) is called Cannon 'a' wave. It appears in:

i. Complete heart block
ii. Paroxysmal atrioventricular nodal tachycardia
iii. Ventricular tachycardia.

3. *Abnormal 'v' Wave*

'v' wave becomes abnormal in tricuspid incompetence.

4. *Abnormal 'x' Wave*

Abnormal 'x' wave appears in:

i. Atrial fibrillation
ii. Cardiac temponade
iii. Constrictive pericarditis.

5. *Abnormal 'y' Wave*

'y' wave becomes abnormal in:

i. Tricuspid regurgitation
ii. Constrictive pericarditis.

Coronary Circulation

Chapter 108

- DISTRIBUTION OF CORONARY BLOOD VESSELS
  - CORONARY ARTERIES
  - VENOUS DRAINAGE
  - PHYSIOLOGICAL SHUNT
- CORONARY BLOOD FLOW AND ITS MEASUREMENT
  - NORMAL CORONARY BLOOD FLOW
  - MEASUREMENT OF CORONARY BLOOD FLOW
- PHASIC CHANGES IN CORONARY BLOOD FLOW
  - PHASIC CHANGES IN LEFT VENTRICLE
  - PHASIC CHANGES IN RIGHT VENTRICLE
- FACTORS REGULATING CORONARY BLOOD FLOW
  - NEED FOR OXYGEN
  - METABOLIC FACTORS
  - CORONARY PERFUSION PRESSURE
  - NERVOUS FACTORS
- APPLIED PHYSIOLOGY – CORONARY ARTERY DISEASE
  - CORONARY OCCLUSION
  - MYOCARDIAL ISCHEMIA AND NECROSIS
  - MYOCARDIAL INFARCTION – HEART ATTACK
  - CARDIAC PAIN – ANGINA PECTORIS

DISTRIBUTION OF CORONARY BLOOD VESSELS

CORONARY ARTERIES

Heart muscle is supplied by two coronary arteries, namely right and left coronary arteries, which are the first branches of aorta. Arteries encircle the heart in the manner of a **crown,** hence the name coronary arteries (Latin word corona = crown).

Right and Left Coronary Arteries

Right coronary artery supplies whole of the right ventricle and posterior portion of left ventricle. Left coronary artery supplies mainly the anterior and lateral parts of left ventricle. There are many variations in diameter of coronary arteries.

Variations in Coronary Arteries

1. In 50% to 60% of human beings, the right coronary artery is larger (right dominant) and supplies more blood to heart than left coronary artery
2. In 15% to 20% of human beings, the left coronary artery is larger (left dominant)
3. In 20% to 30% of human beings, both arteries supply almost equal amount of blood.

Branches of Coronary Arteries

Coronary arteries divide and subdivide into smaller branches, which run all along the surface of the heart. Smaller branches are called **epicardiac arteries** and give rise to further smaller branches known as **final arteries** or **intramural vessels.** Final arteries run at right

angles through the heart muscle, near the inner aspect of wall of the heart.

■ VENOUS DRAINAGE

Venous drainage from heart muscle is by three types of vessels.

1. *Coronary Sinus*

Coronary sinus is the larger vein draining 75% of total coronary flow. It drains blood from left side of the heart and opens into right atrium near tricuspid valve.

2. *Anterior Coronary Veins*

Anterior coronary veins drain blood from right side of the heart and open directly into right atrium.

3. *Thebesian Veins*

Thebesian veins drain deoxygenated blood from myocardium, directly into the concerned chamber of the heart.

■ PHYSIOLOGICAL SHUNT

Physiological shunt is the diverted route (diversion), through which the venous (deoxygenated) blood is mixed with arterial blood. Deoxygenated blood flowing from thebesian veins into cardiac chambers makes up the part of normal physiological shunt.

Other component of physiological shunt is the drainage of deoxygenated blood from bronchial circulation into pulmonary vein, without being oxygenated. Refer Chapter 119 for more details about physiological shunt.

■ CORONARY BLOOD FLOW AND ITS MEASUREMENT

■ NORMAL CORONARY BLOOD FLOW

Normal blood flow through coronary circulation is about 200 mL/minute. It forms 4% of cardiac output. It is about 65 to 70 mL/minute/100 g of cardiac muscle.

■ MEASUREMENT OF CORONARY BLOOD FLOW

Direct Method

Coronary blood flow is measured by using an **electromagnetic flowmeter.** It is directly placed around any coronary artery (refer Chapter 98 for details of electromagnetic flowmeter).

Indirect Method

1. *By Fick principle*

Coronary blood flow is measured by applying Fick principle (Chapter 98) using **nitrous oxide** (N_2O). The subject is asked to inhale a known quantity of the gas with atmospheric air. Then, blood samples are collected from an artery and from coronary sinus, by using a catheter. The blood flow is determined by using the formula:

$$\text{Blood flow} = \frac{\text{Amount of } N_2O \text{ taken up/minute}}{\text{Arteriovenous difference of } N_2O \text{ content}}$$

2. *By using Doppler flowmeter*

Piezoelectric crystals are used in the Doppler flowmeter probe, to transmit and receive the pulses of high frequency sound waves (Chapter 98). The Doppler flowmeter probe is mounted to a catheter and positioned at the ostium of right or left coronary artery to measure the velocity of phasic flow of blood. The cross-sectional area of the artery is determined by angiography. From velocity of blood flow and cross-sectional area, the volume of blood flow is calculated.

3. *By videodensitometry*

Videodensitometry is the technique used to measure both velocity of blood flow and the cross-sectional area of coronary arteries, simultaneously. From these two values, the coronary blood flow can be calculated.

■ PHASIC CHANGES IN CORONARY BLOOD FLOW

Blood flow through coronary arteries is not constant. It decreases during systole and increases during diastole (Fig. 108.1).

Intramural vessels or final arteries supplying myocardium are perpendicular to the cardiac muscles. So, during systole, the intramural vessels are compressed and blood flow is reduced. During diastole, the compression is released and the blood vessels are distended. So, the blood flow increases.

■ PHASIC CHANGES IN LEFT VENTRICLE

In left ventricle, during the onset of isometric contraction, blood flow declines sharply due to two reasons, namely increase in myocardial tissue pressure and decrease in aortic pressure.

During ejection period, rise in aortic pressure causes a sharp rise in flow into left coronary artery. However,

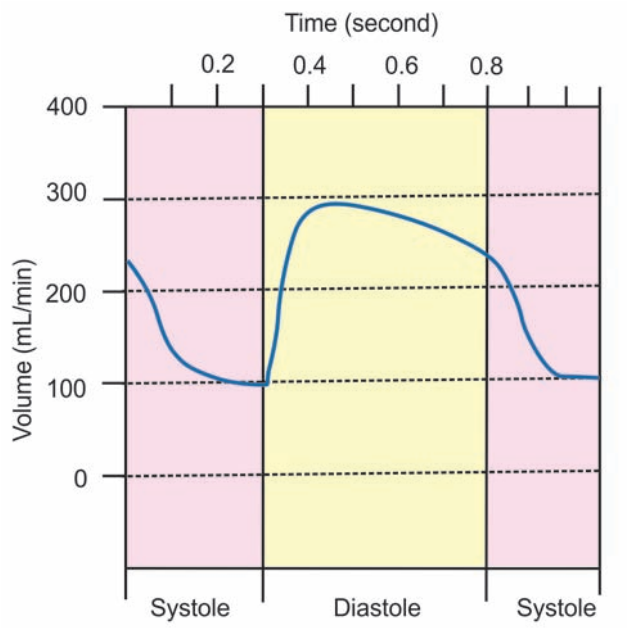

FIGURE 108.1: Phasic changes in coronary blood flow

the flow of blood through coronary capillaries is less. It is due to the high intramural myocardial pressure in the contracting ventricle. Decreased blood flow is maintained until the closure of aortic valve, i.e. till the end of systole.

During the onset of diastole, blood flow rises and it reaches the peak sharply. During the later part of diastole, the flow is reduced slightly along with decreasing aortic pressure. Once again, there is a sharp fall in flow during the onset of systole.

■ PHASIC CHANGES IN RIGHT VENTRICLE

A small amount of blood flows into right ventricle during systole. It is because the force of contraction is not as severe as in the case of left ventricle. Still, the amount of blood flowing is very much less than that during diastole.

■ FACTORS REGULATING CORONARY BLOOD FLOW

Autoregulation

Like any other organ, heart also has the capacity to regulate its own blood flow by autoregulation (Chapter 102). Coronary blood flow is not affected when mean arterial pressure varies between 60 and 150 mm Hg. Several factors are involved in the autoregulation mechanism.

Coronary blood flow is regulated mainly by local vascular response to the needs of cardiac muscle.

Factors regulating coronary blood flow:

1. Need for oxygen
2. Metabolic factors
3. Coronary perfusion pressure
4. Nervous factors.

■ 1. NEED FOR OXYGEN

Oxygen is the most important factor maintaining blood flow through the coronary blood vessels. Amount of blood passing through coronary circulation is directly proportional to the consumption of oxygen by cardiac muscle.

Even in resting condition, a large amount of oxygen, i.e. 70% to 80% is consumed from the blood by heart muscle than by any other tissues. In conditions associated with increased cardiac activity, the need for oxygen increases enormously.

Thus, the need for oxygen, i.e. hypoxia immediately causes coronary vasodilatation and increases the blood flow to heart.

■ 2. METABOLIC FACTORS

Coronary vasodilatation during hypoxic conditions occurs because of some metabolic products, which increase the coronary blood flow by vasodilatation.

Reactive Hyperemia

Reactive hyperemia is the increase in blood flow due to the vasodilator effects of metabolites.

Metabolic Products which Increase the Coronary Blood Flow

Adenosine

Adenosine is a potent vasodilator and it increases the blood flow to cardiac muscle. During hypoxia, ATP in the muscle is degraded in large amount, forming ADP. Some ADP molecules are further degraded into adenosine, which is released into tissue fluids of heart muscle.

Other substances

Other substances which increase the coronary blood flow by vasodilatation are:

i. Potassium
ii. Hydrogen
iii. Carbon dioxide
iv. Adenosine phosphate compounds.

3. CORONARY PERFUSION PRESSURE

Perfusion pressure is the balance between mean arterial pressure and venous pressure (Chapter 102). Thus, coronary perfusion pressure is the balance between mean arterial pressure in aorta and the right atrial pressure. Since right aterial pressure is low, the mean arterial pressure becomes the major factor that maintains the coronary blood flow. Range of mean arterial pressure at which the coronary blood flow can be maintained is given above.

4. NERVOUS FACTORS

Coronary blood vessels are innervated both by parasympathetic and sympathetic divisions of autonomic nervous system. It is not known whether the autonomic nerves have direct effect on blood flow in various conditions. However, these nerves influence the coronary blood flow indirectly by acting on the musculature of heart.

For example, stimulation of sympathetic nerves increases the rate and force of contraction of heart. This in turn, causes liberation of more metabolites which dilate the blood vessels and increase the coronary blood flow. Similarly, when parasympathetic nerves are stimulated, the cardiac functions are inhibited and the production of metabolites is less. Coronary blood flow decreases.

APPLIED PHYSIOLOGY – CORONARY ARTERY DISEASE

Coronary artery disease (CAD) is the heart disease that is caused by inadequate blood supply to cardiac muscle due to occlusion of coronary artery. It is also called coronary heart disease.

CORONARY OCCLUSION

Definition

Coronary occlusion is the partial or complete obstruction of the coronary artery.

Cause

Coronary occlusion is caused by atherosclerosis, a condition associated with deposition of cholesterol on the walls of the artery. In due course, this part of the arterial wall becomes fibrotic and it is called **atherosclerotic plaque.** The plaque is made up of cholesterol, calcium and other substances from blood. Because of the atherosclerotic plaque, the lumen of the coronary artery becomes narrow. In severe conditions, the artery is completely occluded.

Development of atherosclerotic plaque is common in coronary arteries near the origin from aorta. This plaque activates platelets, resulting in **thrombosis** and the blood clot is called **thrombus.** When three fourth of the lumen of the coronary artery is obstructed either by atherosclerotic plaque or thrombus, the blood flow to myocardium is reduced. It results in **ischemia** of myocardium. Coronary thrombosis is associated with **spasm** of coronary artery.

Smaller blood vessels are occluded by the thrombus or part of atherosclerotic plaque, detached from coronary artery. This thrombus or part of the plaque is called **embolus.**

MYOCARDIAL ISCHEMIA AND NECROSIS

Myocardial Ischemia

Myocardial ischemia is the reaction of a part of myocardium in response to hypoxia. Hypoxia develops when blood flow to a part of myocardium decreases severely due to occlusion of a coronary artery.

Blood flow is usually restored if a small quantum of myocardium is affected by ischemia due to obstruction of smaller blood vessels. It is due to rapid development of **coronary collateral arteries.**

Necrosis

Necrosis refers to death of cells or tissues by injury or disease in a localized area. Ischemia leads to necrosis of myocardium if a large part of myocardium is involved or the occlusion is severe involving larger blood vessels. Necrosis is irreversible.

MYOCARDIAL INFARCTION – HEART ATTACK

Myocardial infarction is the necrosis of myocardium caused by insufficient blood flow due to embolus, thrombus or vascular spasm. It is also called heart attack. In myocardial infarction, death occurs rapidly due to ventricular fibrillation.

Myocardial Stunning

Myocardial stunning is a type of transient mechanical dysfunction of heart, caused by a mild reduction in blood flow. A substantial reduction in coronary blood flow causes ischemia followed by necrosis. A mild reduction in blood flow causes only ischemia and it may not be sufficient to cause necrosis of myocardium. However,

it produces some transient (short lived) mechanical disturbances or dysfunction of the heart. Since it is short lived, heart recovers completely from this.

Symptoms of Myocardial Infarction

Common symptoms of myocardial infarction:

1. Cardiac pain
2. Nausea
3. Vomiting
4. Palpitations
5. Difficulty in breathing
6. Extreme weakness
7. Sweating
8. Anxiety.

■ CARDIAC PAIN – ANGINA PECTORIS

Cardiac pain is the **chest pain** that is caused by myocardial ischemia. It is also called angina pectoris. It is the common manifestation of coronary artery disease. Pain starts beneath the sternum and radiates to the surface of left arm and left shoulder. Cardiac pain is a referred pain and it is felt over the body, away from heart. It is because, heart and left arm develop from the same dermatomal segment in embryo.

Cause for Cardiac Pain

Ischemia is mainly due to hypoxia. During myocardial ischemia, there is accumulation of anaerobic metabolic end products such as uric acid. Metabolites and other pain producing substances like substance P, histamine and kinin stimulate the sensory nerve endings, leading to pain.

Sensory Pathway

Sensory pathway from the heart is as follows:

1. Inferior cervical sympathetic nerve fibers (Chapter 101) carrying the sensations of pain (or stretch) from the heart reach the posterior gray horn of first 4 thoracic segments of spinal cord
2. Here, these fibers synapse with second order neurons (substantial gelatinosa of Rolando) of lateral spinothalamic tract
3. Fibers from substantial gelatinosa of Rolando form lateral spinothalamic tract and reach the sensory cortex via thalamus.

If hypoxia in myocardium is relieved by coronary collateral circulation or by treatment, the pain producing substances are washed away by blood flow.

Chronic Angina Pectoris

In chronic angina pectoris, the patient does not feel the pain normally. The pain is felt only when the workload of heart increases. The workload of the heart increases in conditions like exercise and emotional outburst.

When the frequency of angina attack increases, the patient is prone to develop acute myocardial infarction.

Treatment for Angina Pectoris

1. *By using drugs*

   i. *Vasodilator drugs:* Vasodilator drugs like glycerol trinitrate or sodium nitrite relieve the pain by dilating coronary arteries. However, the main therapeutic effect of such drugs is to dilate splanchnic blood vessels, which cause reduction in venous return, cardiac output, workload of the heart and oxygen consumption in myocardium so that, release of pain promoting substances is inhibited.

   ii. *Calcium **channel blockers:*** These drugs block the influx of calcium into the cells. When calcium influx is blocked, the myocardial contractility and workload of the heart are decreased.

   iii. *Sympathetic blocking agents:* Sympathetic blocking agents like propranolol **(beta blockers)** block the beta-adrenergic receptors and inhibit the cardiac activity. This decreases heart rate, stroke volume, workload on heart and oxygen consumption. It also stops the production of nociceptive substances in myocardium.

2. *By thrombolysis*

Refer Chapter 98.

3. *By surgical methods*

   i. *Aortic-coronary artery bypass graft:* Part of myocardium affected by coronary occlusion is detected by **angiography.** Then, the anastomosis is made between aorta and the coronary artery beyond occlusion, by a technique called aortic-coronary artery bypass graft. Mostly, a small vein from lower limb is used for anastomosis. Though this method can relieve the pain, it is not useful if the myocardium is damaged extensively.

   ii. *Percutaneous transluminal coronary angioplasty (PTCA):* Refer Chapter 98.

   iii. *Laser coronary angioplasty:* Refer Chapter 98.

Cerebral Circulation

Chapter 109

- INTRODUCTION
- CEREBRAL VESSELS AND NORMAL CEREBRAL BLOOD FLOW
- MEASUREMENT OF CEREBRAL BLOOD FLOW
  - KETY AND SCHMIDT NITROUS OXIDE METHOD
  - BY USING RADIOACTIVE SUBSTANCES
  - BY COMPUTERIZED AXIAL TOMOGRAPHY (CAT)
  - BY POSITRON EMISSION TOMOGRAPHY (PET)
  - BY MAGNETIC RESONANCE IMAGING (MRI)
- REGULATION OF CEREBRAL BLOOD FLOW
  - AUTOREGULATION
  - CHEMICAL FACTORS
  - NERVOUS FACTORS
- APPLIED PHYSIOLOGY – STROKE

INTRODUCTION

Brain tissues need adequate blood supply continuously. Stoppage of blood flow to brain for 5 seconds leads to unconsciousness and for 5 minutes leads to irreparable damage to the brain cells.

CEREBRAL VESSELS AND NORMAL CEREBRAL BLOOD FLOW

Brain receives blood from the **basilar artery** and **internal carotid artery.** Branches of these arteries form **circle of Willis.** Venous drainage is by sinuses, which open into **internal jugular vein.**

Normally, brain receives 750 to 800 mL of blood per minute. It is about 15% to 16% of total cardiac output and about 50 to 55 mL/100 g of brain tissue per minute.

MEASUREMENT OF CEREBRAL BLOOD FLOW

1. KETY AND SCHMIDT NITROUS OXIDE METHOD

Nitrous oxide method is an indirect method to measure the blood flow to the brain. It is based on **Fick principle** (Chapter 98). **Nitrous oxide** is used as an indicator substance in this method.

The subject is asked to inhale nitrous oxide at a low concentration, which is less than the amount required for anesthesia. After inhalation of the gas for about 10 minutes, the amount of nitrous oxide retained in the brain tissues becomes equal to the amount of nitrous oxide present in cerebral venous blood. Now, the concentration of nitrous oxide is determined in the arterial blood and

cerebral venous blood and the cerebral blood flow is calculated by the formula:

$$\text{Cerebral blood flow} = \frac{\text{Amount of } N_2O \text{ taken by brain}}{\text{Arteriovenous difference of } N_2O}$$

■ 2. BY USING RADIOACTIVE SUBSTANCES

Radioactive substances method is used to determine the amount of blood flow to different regions of the cerebral cortex. Radioactive substance is injected into the carotid artery. By measuring the radioactivity in the brain tissues using **radioactive detectors (scintillation counter),** the blood flowing through each area of brain is determined. Advantage of this method is that the blood flow to about 250 areas of cerebral cortex can be measured by using many radioactive detectors. Radioactive xenon and 2-deoxyglucose are the commonly used radioactive substances to measure the cerebral blood flow.

■ 3. BY COMPUTERIZED AXIAL TOMOGRAPHY

Computerized axial tomography (CT or CAT) scanning was introduced in 1970s. Tomography scanning is a process which combines many two dimensional X-ray images to generate cross sectional pictures of different organs or regions of the body. Advancement of technology resulted in combination of many three dimensional X-ray images of body structures and organs including brain. CT scan of brain is useful to determine brain damage and local changes in cerebral blood flow, while the subject performs a task.

■ 4. BY POSITRON EMISSION TOMOGRAPHY

Positron emission tomography (PET) scanner is a type of computerized tomography machine. A short-lived radioactive substance called radionuclide combined with sugar is injected into the patient. Radionuclide emits positrons (antiparticle or antimatter counterpart of electron). Positron emissions from radionuclide are detected by rotating the PET scanner around patient's head. PET is used to study blood volume, oxygen consumption, pH, glucose utilization, blood flow and the activity of receptors in brain cells.

■ 5. BY MAGNETIC RESONANCE IMAGING

Magnetic resonance imaging (MRI) is a different type of imaging technique. It involves polarization of hydrogen atoms in the soft tissues by using a large magnet and detecting the resonant signals (summation of the spinning energies within the living cells) from the tissues. Since the images are very clear, this technique is useful for scanning soft tissues, brain, spinal cord, abdomen, joints and malignant tissues. MRI is also used to measure blood flow to the organs such as brain. Measurement of blood flow to a part or area of the organ is called functional magnetic resonance imaging (fMRI).

■ REGULATION OF CEREBRAL BLOOD FLOW

Cerebral circulation is regulated by three factors:

1. Autoregulation
2. Chemical factors
3. Neural factors.

■ AUTOREGULATION

Like any other vital organ, brain also regulates its own blood flow by means of autoregulation (Chapter 102). However, the autoregulation in brain has got its own limitations. It depends upon:

i. Effective perfusion pressure
ii. Cerebral vascular resistance.

Cerebral blood flow is directly proportional to the balance between effective perfusion pressure and the vascular resistance in brain.

i. *Effective Perfusion Pressure*

Effective perfusion pressure is the balance between the mean arterial blood pressure and venous pressure across the organ, divided by resistance (Chapter 102). Since venous pressure is zero in brain, mean arterial blood pressure plays an important role in regulating cerebral blood flow. Autoregulation is possible in brain if the mean arterial pressure is within the range of 60 mm Hg and 140 mm Hg. Autoregulation fails beyond this range on either side.

ii. *Cerebral Vascular Resistance*

When the vascular resistance is more, the blood flow to the brain is less. Resistance to blood flow in brain is offered by intracranial pressure, cerebrospinal fluid pressure and viscosity of blood.

Intracranial pressure and cerebrospinal fluid pressure

Increase in the intracranial pressure or the pressure exerted by the cerebrospinal fluid (CSF) compresses the cerebral blood vessels and decreases blood flow. These pressures are elevated in conditions like head

injury. However, severe ischemic effects are avoided by some protective reflexes such as Cushing reflex.

Cushing reflex

Cushing reflex is a protective reflex that helps save the brain tissues from ischemic effects during the periods of reduced cerebral blood flow. It is also called **Cushing reaction,** response or phenomenon.

Increase in intracranial pressure or increase in CSF pressure compresses the cerebral blood vessels and decreases the blood flow. However, blood flow is decreased only for a short period. It is restored immediately by means of Cushing reflex. When cerebral blood flow decreases by the compression of cerebral arteries, the cerebral ischemia develops. Compression of blood vessels decreases the blood flow to vasomotor center also. Local hypoxia and hypercapnea activate vasomotor center, resulting in peripheral vasoconstriction and rise in the arterial pressure. The increased arterial pressure helps to restore the cerebral blood flow. Thus, Cushing reflex plays the most important role in maintaining the cerebral blood flow (Fig. 109.1).

Cushing reflex operates only when the rise in arterial blood pressure is proportional to increase in intracranial pressure. When the increase in intracranial pressure is very high and if it exceeds the arterial blood pressure, this protective mechanism fails. And the cerebral ischemia becomes severe, leading to irreversible damage of the brain tissues.

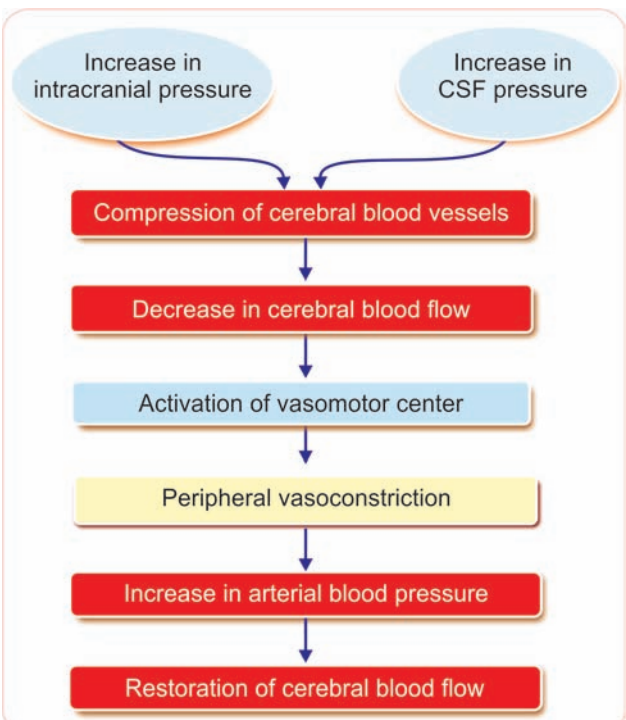

FIGURE 109.1: Schematic representation of Cushing reflex. CSF = Cerebrospinal fluid.

Monro-Kellie doctrine

According to Monro-Kellie doctrine or principle, though the cerebral arteries are compressed by increased intracranial pressure or cerebrospinal fluid pressure, the volume of brain tissue is not affected. It is because the brain tissue is not compressible.

Viscosity

Increase in the viscosity of blood as in polycythemia, increases the cerebral vascular resistance and blood flow decreases. When viscosity decreases as in the case of anemia, the resistance is decreased and blood flow increases. Thus, the cerebral blood flow is inversely proportional to the viscosity of blood.

■ CHEMICAL FACTORS

Chemical factors which increase the cerebral blood flow:

i. Decreased oxygen tension
ii. Increased carbon dioxide tension
iii. Increased hydrogen ion concentration.

Carbon dioxide is the most important factor, as it causes dilatation of cerebral blood vessels, leading to increase in blood flow. A moderate increase in carbon dioxide tension does not alter the blood flow due to autoregulation. When arterial partial pressure of carbon dioxide rises above 45 mm Hg, the cerebral blood flow increases.

Carbon dioxide combines with water to form carbonic acid, which dissociates into bicarbonate ions and hydrogen ion. The hydrogen ion causes dilatation of blood vessels in brain.

Hypoxia increases cerebral blood flow by vasodilatation.

■ NERVOUS FACTORS

Cerebral blood vessels are supplied by sympathetic vasoconstrictor fibers. But, these fibers do not play any role in regulating cerebral blood flow under normal conditions. In pathological conditions like hypertension, the sympathetic nerves cause constriction of cerebral blood vessels, leading to reduction in blood flow. It prevents cerebral vascular hemorrhage and cerebral stroke.

■ APPLIED PHYSIOLOGY – STROKE

Definition

Stroke is the sudden death of neurons in localized area of brain due to inadequate blood supply. It is characterized by reversible or irreversible paralysis with other symptoms. Stroke is also called **cardiovascular accident** (CVA) or **brain attack.**

Types

Stroke is classified into two types:

1. **Ischemic stroke,** which occurs due to interruption of blood flow to a part of brain by thrombus or atherosclerotic embolus
2. **Hemorrhagic stroke,** which develops by the rupture of a blood vessel in the brain and spilling of blood into the surrounding areas.

Causes

Most common factors (risk factors) which causes stroke are:

1. Heart disease
2. Hypertension
3. High cholesterol in blood
4. High blood sugar (diabetes mellitus)
5. Heavy smoking
6. Heavy alcohol consumption.

Symptoms

Symptoms of stroke depend upon the area of brain that is damaged. Generally, stroke causes dizziness, loss of consciousness, coma or death.

Other features of stroke:

1. Weakness
2. Numbness or paralysis, particularly on one side of the body
3. Impairment of speech
4. Emotional disturbances
5. Loss of coordination
6. Loss of memory.

Splanchnic Circulation

Chapter 110

- INTRODUCTION
- MESENTERIC CIRCULATION
  - DISTRIBUTION OF BLOOD FLOW
  - REGULATION OF MESENTERIC BLOOD FLOW
- SPLENIC CIRCULATION
  - IMPORTANCE OF SPLENIC CIRCULATION
  - STORAGE OF BLOOD
  - REGULATION OF BLOOD FLOW TO SPLEEN
- HEPATIC CIRCULATION
  - BLOOD VESSELS
  - NORMAL BLOOD FLOW
  - REGULATION OF BLOOD FLOW TO LIVER

INTRODUCTION

Splanchnic or visceral circulation constitutes three portions:

1. Mesenteric circulation supplying blood to GI tract
2. Splenic circulation supplying blood to spleen
3. Hepatic circulation supplying blood to liver.

Unique feature of splanchnic circulation is that the blood from mesenteric bed and spleen forms a major amount of blood flowing to liver. Blood flows to liver from GI tract and spleen through portal system.

MESENTERIC CIRCULATION

DISTRIBUTION OF BLOOD FLOW

Stomach : 35 mL/100 g/minute
Intestine : 50 mL/100 g/minute
Pancreas : 80 mL/100 g/minute.

REGULATION OF MESENTERIC BLOOD FLOW

Mesenteric blood flow is regulated by the following factors:

1. *Local Autoregulation*

Local autoregulation is the primary factor regulating blood flow through mesenteric bed (Chapter 102).

2. *Activity of Gastrointestinal Tract*

Contraction of the wall of the GI tract reduces blood flow due to compression of blood vessels. And relaxation of wall of GI tract increases the blood flow due to removal of compression on the vessel wall.

3. *Nervous Factor*

Mesenteric blood flow is regulated by sympathetic nerve fibers. Increase in sympathetic activity as in the case of emotional conditions or **'fight and flight reactions'** constrict the mesenteric blood vessels. So, more blood is diverted to organs like skeletal muscles, heart and brain, which need more blood during these conditions. Parasympathetic nerves do not have any direct action on the mesenteric blood vessels. But these nerves increase the contraction of GI tract which compresses the blood vessels, resulting in reduction in blood flow.

4. *Chemical Factors – Functional Hyperemia*

Functional hyperemia is the increase in mesenteric blood flow immediately after food intake. It is mainly because of **gastrin** and **cholecystokinin** secreted after food intake. In addition to these two GI hormones, digestive products of food substances such as glucose and fatty acids also cause vasodilatation and increase the mesenteric blood flow.

SPLENIC CIRCULATION

IMPORTANCE OF SPLENIC CIRCULATION

Spleen is the main reservoir for blood. Due to the dilatation of blood vessels, a large amount of blood is stored in spleen. And the constriction of blood vessels by sympathetic stimulation releases blood into circulation.

STORAGE OF BLOOD

In spleen, two structures are involved in storage of blood, namely **splenic venous sinuses** and **splenic pulp** (Chapter 25).

Small arteries and arterioles open directly into the venous sinuses. When spleen distends, sinuses swell and large quantity of blood is stored. Capillaries of splenic pulp are highly permeable. So, most of the blood cells pass through capillary membrane and are stored in the pulp.

Venous sinuses and the pulp are lined with **reticuloendothelial cells.**

REGULATION OF BLOOD FLOW TO SPLEEN

Blood flow to spleen is regulated by sympathetic nerve fibers.

HEPATIC CIRCULATION

BLOOD VESSELS

Liver receives blood from two sources:

1. Hepatic artery
2. Portal vein.

More details are given in Chapter 40.

NORMAL BLOOD FLOW

Liver receives maximum amount of blood as compared to any other organ in the body since, most of the metabolic activities are carried out in the liver. Blood flow to liver is 1,500 mL/minute, which forms 30% of the cardiac output. It is about 100 mL/100 g of tissue/minute.

Normally, about 1,100 mL of blood flows through portal vein and remaining 400 mL of blood flows through hepatic artery. However, portal vein carries only about 25% of oxygen to liver. It is because it carries the blood, which has already passed through the blood vessels of GI tract, where oxygen might have been used. Hepatic artery transports 75% of oxygen to the liver.

REGULATION OF BLOOD FLOW TO LIVER

Blood flow to liver is regulated by the following factors:

1. *Systemic Blood Pressure*

Systemic blood pressure is the important factor responsible for blood flow to liver and hepatic blood flow is directly proportional to systemic blood pressure.

2. *Splenic Contraction*

During splenic contraction, blood flow to liver increases.

3. *Movements of Intestine*

Motility of intestine increases hepatic blood flow.

4. *Chemical Factors*

Chemical factors which increase the blood flow to liver by vasodilatation are:

i. Excess carbon dioxide
ii. Lack of oxygen
iii. Increase in hydrogen ion concentration.

5. *Nervous Factors*

Sympathetic fibers to liver cause vasoconstriction in liver and decrease the blood flow.

Sympathetic fibers to liver and other portions of splanchnic circulation pass through splanchnic nerve. Role of parasympathetic fibers in hepatic circulation is not known.

Capillary Circulation

Chapter 111

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INTRODUCTION

MICROCIRCULATION

Microcirculation refers to flow of blood through the minute blood vessels such as arterioles, capillaries and venules. Capillary circulation forms the major part of microcirculation. Human body contains about 10 billion capillaries.

Study of Capillary Circulation

Blood flow through capillaries is studied by focusing the capillaries under dissecting microscope. Frog's web, mesentery of mammals and fingernail bed of humans can be observed by using microscope.

FEATURES OF CAPILLARIES

1. Capillaries arise from arterioles and form the actual functional area of circulatory system, i.e. exchange of materials between blood and tissues
2. Structurally, capillaries are very narrow and short. However, quantitatively, these vessels outnumber the other blood vessels. About 10 billion capillaries are present in the body.
3. Each capillary lies in a very close proximity to the cells of the tissues at a distance of about 20 to 30 mm. This enables easy and rapid exchange of substances between blood and the tissues through interstitial fluid.

DIMENSIONS OF CAPILLARIES

Dimensions of capillaries are given in Table 111.1.

TABLE 111.1: Dimensions and details of capillaries

Dimensions/Details	Normal value
Total number of capillaries	10 billion
Surface area of all capillaries	500 to 700 sq m
Average length	0.5 to 1 mm
Average diameter	8 μ
Pressure at arterial end	30 to 32 mm Hg
Pressure at venous end	14 to 16 mm Hg
Velocity of blood flow	0.05 cm/second

VELOCITY AND VOLUME OF BLOOD FLOW

Average velocity of blood flow through capillaries is 0.05 cm/second. About 5% of total blood is present in capillaries.

STRUCTURE OF CAPILLARIES

Capillaries are formed by single layer of endothelial cells, which are wrapped around by pericytes.

ENDOTHELIAL CELLS

Endothelial cells of the capillaries are thin, flattened, nucleated polygonal cells joined together by a cement substance.

Capillaries do not have muscular coat. Yet, these blood vessels actively modify their own diameter in response to nervous, hormonal, chemical and physical stimuli. Endothelial cells themselves alter the diameter of capillaries by swelling or shrinking.

In most of the capillaries, adjacent endothelial cells leave a cleft called fenestra through which several substances may traverse the endothelium by means of transcytosis (Fig. 111.1). However, in cerebral capillaries the fenestra are absent because the endothelial cells fuse to each other by tight junctions (Chapter 163).

PERICYTES

Pericyte is a perivascular mesenchymal like cell associated with walls of small blood vessels such as capillaries and postcapillary vessels. It is similar to renal mesangial cell. It is also known as mural cell or Rouget cell (named after the discoverer Charles Rouget).

Pericytes extend long cytoplasmic processes, which wrap around the endothelial cells. Pericytes play important role in remodeling and maintenance of capillary system. These cells are contractile in nature and secrete several vasoactive agents, growth factors, extracellular matrix and components of basement membrane. Pericytes are also involved in regulation of blood flow through endothelial junctions particularly in conditions such as inflammation.

PATTERN OF CAPILLARY SYSTEM

Capillaries are disposed between arterioles and venules. From the arterioles, the meta-arterioles take origin (Fig. 111.2). From meta-arterioles, two types of capillaries arise:

1. Preferential channels
2. True capillaries.

1. PREFERENTIAL CHANNELS

Preferential channels are also called continuous capillaries. After arising from meta-arterioles, these capillaries form a network and finally join the venules. Preferential channels or continuous capillaries have same diameter as meta-arterioles.

2. TRUE CAPILLARIES

True capillaries also form a network and join the venules. Diameter of the true capillaries is less than that of the meta-arterioles.

Precapillary Sphincter

Beginning of true capillaries is encircled by smooth muscle fibers. It functions as a sphincter; so it is known as precapillary sphincter. It controls the blood flow through true capillaries.

ANATOMICAL AND PHYSIOLOGICAL SHUNTS

Anatomical Shunt

Anatomical shunt is the direct link between arterioles and venules. It is also called arteriovenous shunt. Flow

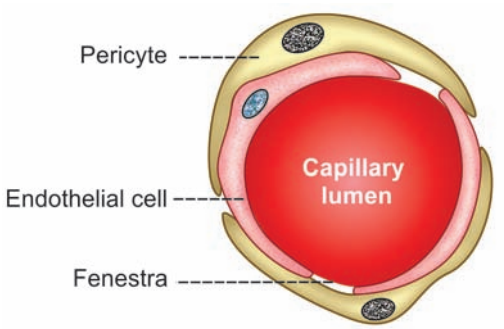

FIGURE 111.1: Cross section of capillary

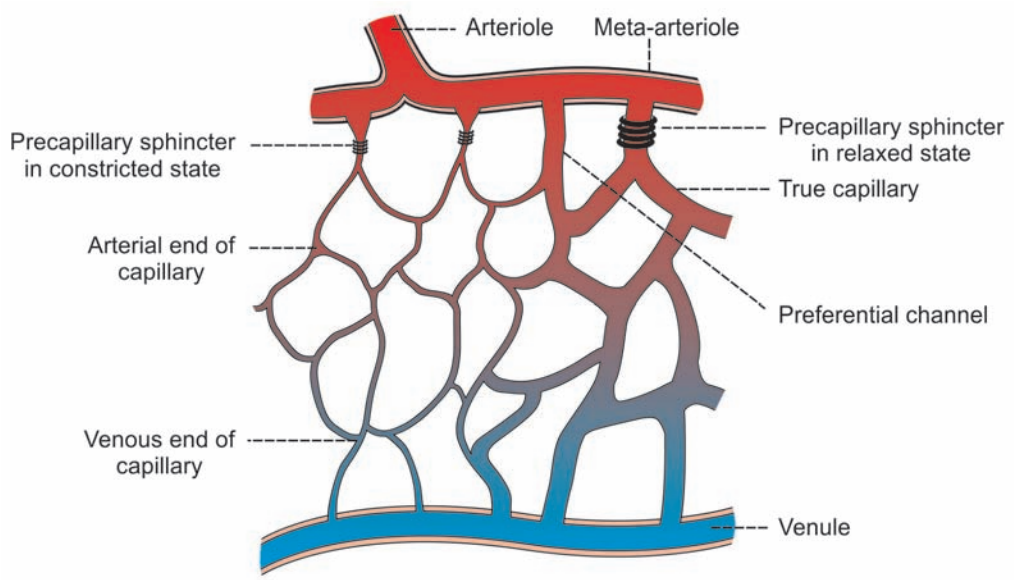

FIGURE 111.2: Capillary bed

of blood through the capillaries where exchange of nutrients, gases and other substances takes place is called nutritional flow. Blood flow through anatomical shunt is called non-nutritional flow. Non-nutritional blood flow occurs in many tissues of the body particularly during resting conditions when metabolic activities are low.

Physiological Shunt

Physiological shunt is the link between arterial and venous side of circulation provided by meta-arteriole. Many tissues of the body such as muscles do not have anatomical shunts. However, the meta-arteriole in these tissues acts as the physiological shunt between arterial and venous sides of the circulation. Non-nutritional blood flow occurs through physiological shunt under resting conditions.

Shunt in Capillaries Vs Shunt in Heart

Physiological shunt in capillaries is different from physiological shunt in heart. In capillaries, the oxygenated blood flows towards deoxygenated blood. But in heart, the deoxygenated blood flows towards the oxygenated blood (Chapter 108).

■ PECULIARITIES OF CAPILLARY BLOOD FLOW

1. Blood does not pass through capillary system continuously. It is because of the alternate constriction and dilatation of meta-arterioles and the alternate opening and closure of precapillary sphincters.
2. Direction of blood flow through capillaries is not fixed as in the case of other blood vessels. Blood may flow in opposite direction in two adjacent capillaries.
3. In capillaries, blood flows as a single pile or single row of blood cells. In other blood vessels, the blood flows in either axial stream containing mainly blood cells or peripheral stream containing plasma.
4. Under resting conditions, most of the capillaries lie in collapsed state. Only during activity, all the capillaries open up and increase the vascularity.
5. Amount of blood flowing through the capillary system throughout the body is very low. It is only about 150 mL/minute.
6. Velocity of blood flow is least in capillaries. It is only about 0.05 cm/second. It facilitates exchange of substances between capillaries and tissues.

■ FUNCTIONS OF CAPILLARIES

Most important function of capillaries is the exchange of substances between blood and tissues. Oxygen, nutrients and other essential substances enter the tissues from capillary blood; carbon dioxide, metabolites and other unwanted substances are removed from the tissues by capillary blood.

Exchange of materials across the capillary endothelium occurs by the following processes:

1. Diffusion
2. Filtration
3. Pinocytosis.

■ DIFFUSION

Diffusion is the main process for exchange of gases, water, glucose, sodium, urea and many other substances. These substances diffuse through the intercellular clefts present in the endothelial wall of the capillaries. Diffusion occurs because of concentration gradient across the capillary wall.

■ FILTRATION

Site of filtration of substances through capillary membrane varies in different organs. In skeletal muscles, cardiac muscles, kidneys and intestine, filtration occurs through the slit pores present in capillary endothelium. Capillaries in other organs have discontinued endothelium through which filtration occurs.

Filtration of substances through capillary endothelium depends upon the net filtration pressure. Net filtration pressure is the balance between the driving pressures and the opposing pressures. It is well explained by Starling hypothesis (Chapter 52). Process of filtration is explained in Chapter 27.

■ PINOCYTOSIS

Larger molecules are transported across the capillary endothelium in the form of vesicles. Large molecules are packed as vesicles in the capillary endothelial cells. These vesicles are transported across the endothelial membrane by the process called pinocytosis (Chapter 3).

■ FACTORS CONTROLLING CAPILLARY CIRCULATION

Capillary blood flow is controlled by the nervous and chemical factors.

■ NERVOUS FACTORS

Capillaries are mainly supplied by the sympathetic vasoconstrictor fibers.

■ CHEMICAL FACTORS

Many chemical factors such as excess of carbon dioxide, increased hydrogen ion concentration, lack of oxygen, histamine and metabolites like lactic acid cause dilatation of capillaries. Serotonin causes constriction of capillaries.

Circulation through Skeletal Muscle

Chapter 112

- INTRODUCTION
- FACTORS REGULATING BLOOD FLOW TO SKELETAL MUSCLE
  - MECHANICAL FACTORS
  - CHEMICAL FACTORS
  - NERVOUS FACTORS
- APPLIED PHYSIOLOGY – VARICOSE VEINS

INTRODUCTION

During resting condition, blood flow to skeletal muscle is 4 to 7 mL/100 g/minute. During exercise, it increases to about 100 mL/100 g/minute.

FACTORS REGULATING BLOOD FLOW TO SKELETAL MUSCLE

Blood flow through skeletal muscle is regulated by three factors:

1. Mechanical factors
2. Chemical factors
3. Nervous factors.

MECHANICAL FACTORS

During contraction of the muscle, blood vessels are compressed and the blood flow decreases. And during relaxation of the muscle, compression of blood vessels is relieved and the blood flow increases.

In severe muscular exercise, blood flow increases in between the muscular contractions.

CHEMICAL FACTORS

Important chemical factors, which regulate the blood flow through skeletal muscles, are lack of oxygen, excess of carbon dioxide and increased hydrogen ion concentration. All these chemical factors increase the blood flow to muscle by causing vasodilatation.

NERVOUS FACTORS

Blood vessels of the skeletal muscles are mostly innervated by sympathetic nerve fibers and few para-sympathetic nerve fibers are also seen. Special feature of sympathetic nerve fibers supplying the skeletal muscles is that these nerve fibers are **vasodilators** and not constrictors. Since the sympathetic nerve fibers cause dilatation of blood vessels in muscle by secreting acetylcholine, these nerve fibers are called **sympathetic vasodilator fibers** or **sympathetic cholinergic fibers.**

APPLIED PHYSIOLOGY – VARICOSE VEINS

Varicose vein is the vein that becomes irregularly swollen (twisted or tortuous) and enlarged. Superficial veins of the leg are mostly affected.

Causes for Varicose Vein

1. Permanent dilatation of veins due to **incompetence** of the valves of the veins or absence of muscular activity for long periods. So, varicose veins are common in the individuals with occupations, which require standing for long periods.
2. Thrombophlebitis (inflammation of vein associated with formation of thrombus).

Varicose veins may also develop in obese persons and pregnant women.

Varicose Vein in Obesity

Obesity is the major factor for varicose veins. Excess fat increases the pressure on veins of legs and aggravate the condition.

Varicose Vein in Pregnancy

During pregnancy, varicose veins develop because of two reasons:

1. Increased blood level of progesterone, which dilates the blood vessel
2. Enlarged uterus, which compresses the major veins in pelvic region leading to increase in venous pressure.

Cutaneous Circulation

Chapter 113

- ARCHITECTURE OF CUTANEOUS BLOOD VESSELS
- FUNCTIONS OF CUTANEOUS CIRCULATION
- NORMAL BLOOD FLOW TO SKIN
- REGULATION OF CUTANEOUS BLOOD FLOW
- APPLIED PHYSIOLOGY – VASCULAR RESPONSES OF SKIN TO MECHANICAL STIMULI
  - WHITE REACTION
  - LEWIS TRIPLE RESPONSE

ARCHITECTURE OF CUTANEOUS BLOOD VESSELS

Architecture of cutaneous blood vessels is formed in the following manner:

1. **Arterioles** arising from the smaller arteries reach the base of **papillae of dermis** (Chapter 60)
2. Then, these arterioles turn horizontally and give rise to **meta-arterioles**
3. From meta-arterioles, hairpin-shaped **capillary loops** arise. Arterial limb of the loop ascends vertically in the papillae and turns to form a venous limb, which descends down.
4. After reaching the base of papillae, few venous limbs of neighboring papillae unite to form the **collecting venule**
5. Collecting venules anastomose with one another to form the **subpapillary venous plexus**
6. Subpapillary plexus runs horizontally beneath the bases of papillae and drain into **deeper veins.**

FUNCTIONS OF CUTANEOUS CIRCULATION

Cutaneous blood flow performs two functions:

1. Supply of nutrition to skin
2. Regulation of body temperature by heat loss.

NORMAL BLOOD FLOW TO SKIN

Under normal conditions, the blood flow to skin is about 250 mL/square meter/minute. When the body temperature increases, cutaneous blood flow increases up to 2,800 mL/square meter/minute because of cutaneous vasodilatation.

REGULATION OF CUTANEOUS BLOOD FLOW

Cutaneous blood flow is regulated mainly by body temperature. Hypothalamus plays an important role in regulating cutaneous blood flow.

When body temperature increases, the hypothalamus is activated. Hypothalamus in turn causes cutaneous vasodilatation by acting through medullary vasomotor center. Now, blood flow increases in skin. Increase in cutaneous blood flow causes the loss of heat from the body through sweat. When body temperature is low, vasoconstriction occurs in the skin. Therefore, the blood flow to skin decreases and prevents the heat loss from skin.

APPLIED PHYSIOLOGY – VASCULAR RESPONSES OF SKIN TO MECHANICAL STIMULI

Vascular responses of skin are the reactions developed in blood vessels of skin when some mechanical stimuli are applied over the surface of it.

Vascular responses of skin are of two types:

A. White reaction
B. Lewis triple response.

■ WHITE REACTION

White reaction is the response of the blood vessels in skin to a mechanical stimulus. When the surface of skin is stroked lightly with a pointed object, a pale line appears within 20 seconds. This line takes the path of the stroke. This response in skin is known as white reaction. Maximum intensity of the line is obtained in 1 minute and it fades away after 5 minutes.

White reaction is due to the **constriction of cutaneous capillaries.** Capillaries constrict because of the local stimulation of capillary wall and exertion of tension upon capillary wall. No nervous factor is involved in this process.

■ LEWIS TRIPLE RESPONSE

Lewis triple response is the vascular response of skin that includes three consecutive reactions of blood vessels of skin to a mechanical stimulus. It was discovered by **Lewis Sir Thomas** in 1927. He noticed that the vascular reactions of skin to various injuries occur in three stages and named these reactions as triple response.

Three reactions of this response:

1. Red reaction
2. Flare
3. Wheal.

1. *Red Reaction*

Red reaction is the appearance of a **red line** when a pointed instrument is drawn firmly over the surface of the skin. This reaction occurs over the line of the stroke. Red reaction appears within 15 seconds after the stroke. It obtains the maximum intensity at the end of 1 minute and disappears later gradually.

Red reaction is because of **dilatation of capillaries** due to mechanical stimulus. This reaction is purely a local response. It occurs due to the release of histamine-like substance from the tissues damaged by the stimulus. Lewis called it **'H' substance.**

Red reaction does not depend upon nervous factors. It occurs even after the sectioning or degeneration of nerves of skin.

2. *Flare*

If the stroke is applied with little more force or if the stroke is repeated on the same line, the red reaction spreads around the line of stroke. It spreads for about 10 cm from the line of stroke, depending upon the force applied. This is called flare or **spreading flush.** Flare appears within 30 seconds after appearance of red line. It also disappears later. Flare is due to **dilatation of arterioles.** It depends upon nervous mechanism and is due to **axon reflex.**

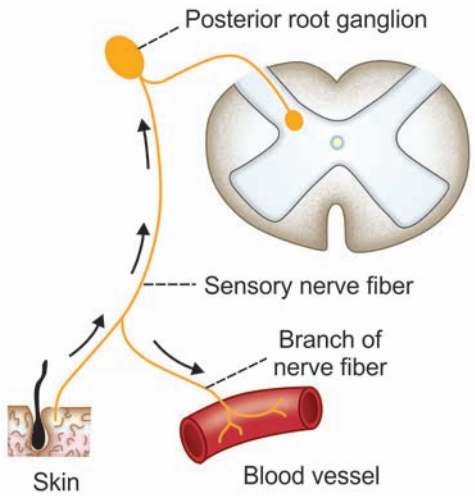

FIGURE 113.1: Axon reflex during flare

Axon Reflex

Axon reflex or antidromic reflex is the process by which the impulses are conducted in a direction opposite to the normal direction. Normally, the impulses produced by a cutaneous pain receptor pass through sensory nerve fiber towards the nerve cell body in posterior nerve root ganglion. Some of these impulses pass through the other branches of the same fiber in the opposite direction and reach the blood vessels supplied by these branches. Impulses now dilate the blood vessels. This is called the **antidromic** or axon reflex (Fig. 113.1). Nerve fibers transmitting the impulses in the opposite direction are called **antidromic vasodilator fibers.**

Flare occurs if the main trunk of nerves is cut. It does not occur when the nerves degenerate.

3. *Wheal*

When intensity of stimulus is severe, the surface of skin on the line of stroke is interrupted. A small elevation or swelling is seen in the surrounding area up to a height of 2 mm. It is called wheal or local edema.

Wheal appears within 3 minutes after the stimulus and it replaces the red line. Maximum height is obtained within 5 minutes and it disappears after several hours.

Wheal appears due to the leakage of fluid from capillaries. The permeability of capillary membrane is increased. Wheal does not depend upon nervous mechanism.

Dermographism

The process of embossing signs over skin is called dermographism. It is also called **writing on skin.** Some letters or designs can be embossed upon the skin over back or in the forearm in the same manner by which the wheal is produced.

Fetal Circulation and Respiration

Chapter 114

- INTRODUCTION
- BLOOD VESSELS IN FETUS
- FETAL LUNGS
- CHANGES IN CIRCULATION AND RESPIRATION AFTER BIRTH – NEONATAL CIRCULATION AND RESPIRATION
  - FIRST BREATH OF THE CHILD
  - FLOW OF BLOOD TO LUNGS
  - CLOSURE OF FORAMEN OVALE
  - REVERSAL OF BLOOD FLOW IN DUCTUS ARTERIOSUS
  - CLOSURE OF DUCTUS VENOSUS
  - CLOSURE OF DUCTUS ARTERIOSUS

INTRODUCTION

Fetal circulation is different from that of adults because of the presence of **placenta.** Since fetal lungs are non-functioning, placenta is responsible for exchange of gases between fetal blood and mother's blood. So, the blood from right ventricle is diverted to placenta.

Development of heart is completed at 4th week of intrauterine life and it starts beating at the rate of 65 per minute. Along with heart, the blood vessels also develop. Heart rate gradually increases and reaches the maximum rate of about 140 beats per minute just before birth.

Fetus is connected with the mother through placenta. Fetal blood passes to placenta through umbilical vessels and the maternal blood runs through uterine vessels. These two sets of blood vessels lie in close proximity in the placenta through which exchange of substances takes place between mother's blood and fetal blood. However, there is no direct admixture of maternal and fetal blood (Fig. 114.1).

BLOOD VESSELS IN FETUS

As **fetal lungs** are **non-functioning,** there is no necessity of large amount of blood to be pumped into lungs. Instead, the fetal heart pumps large quantity of blood into the placenta for exchange of substances. From placenta, the **umbilical veins** collect the blood, which has more oxygen and nutrients. Umbilical vein passes through liver. Some amount of blood is supplied to liver from umbilical vein. However, a large quantity of blood is diverted from umbilical vein into the **inferior vena cava** through **ductus venosus.** Liver receives blood from portal vein also.

In liver, the oxygenated blood mixes slightly with deoxygenated blood and enters the right atrium via inferior vena cava. From right atrium, major portion of blood is diverted into left atrium via **foramen ovale.** Foramen ovale is an opening in intra-atrial septum.

Blood from upper part of the body enters the right atrium through superior vena cava. From right atrium, blood enters right ventricle. From here, blood is pumped into pulmonary artery. From pulmonary artery, blood enters the systemic aorta through **ductus arteriosus.** Only a small quantity of blood is supplied to fetal lungs. Blood from left ventricle is pumped into aorta. Fifty percent of blood from aorta reaches the placenta through **umbilical arteries.**

FETAL LUNGS

Pulmonary vascular resistance is the resistance offered to blood flow through pulmonary vascular bed. This resistance is very high in fetus because of the non-

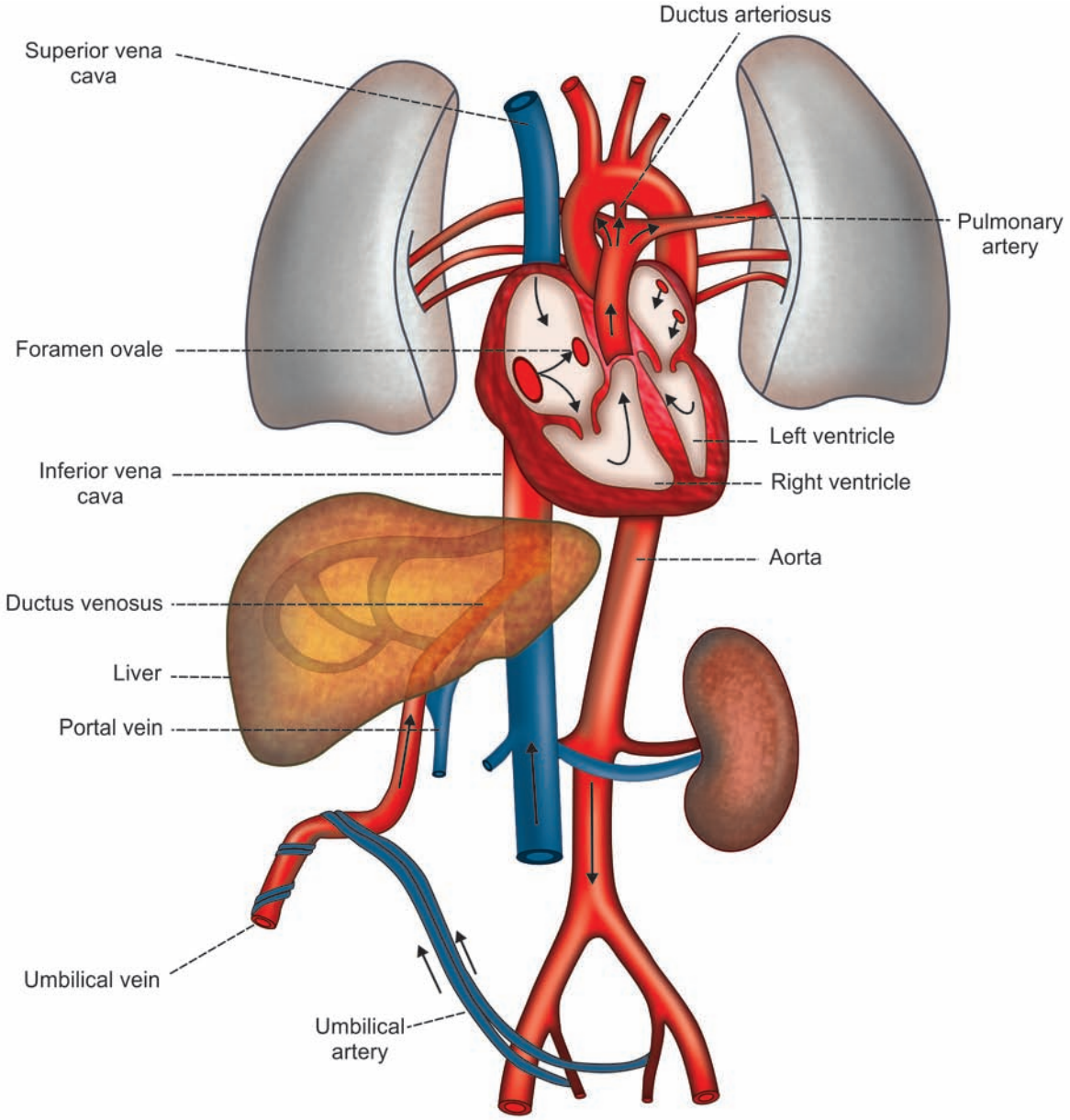

FIGURE 114.1: Fetal circulation

functioning of fetal lungs. The high resistance in fetal lungs increases the pressure in the blood vessels of lungs. Because of the high pressure, the blood is diverted from pulmonary artery into aorta via ductus arteriosus.

CHANGES IN CIRCULATION AND RESPIRATION AFTER BIRTH – NEONATAL CIRCULATION AND RESPIRATION

1. FIRST BREATH OF THE CHILD

When fetus is delivered and umbilical cord is cut and tied, the lungs start functioning. When placental blood flow is cut off, there is sudden **hypoxia** and **hypercapnia.** Now, the respiratory center is strongly stimulated by these two factors and the respiration starts. Initially, there is **gasping,** which is followed by normal respiration.

2. FLOW OF BLOOD TO LUNGS

Lungs expand during the first breath of the infant. Expansion of lungs causes immediate reduction in the pulmonary vascular resistance and a sudden fall in pressure in the blood vessels of lungs. Therefore, the blood flow from pulmonary artery to lungs increases.

3. CLOSURE OF FORAMEN OVALE

When blood starts flowing through the pulmonary circulation, the oxygenated blood from the lungs returns to

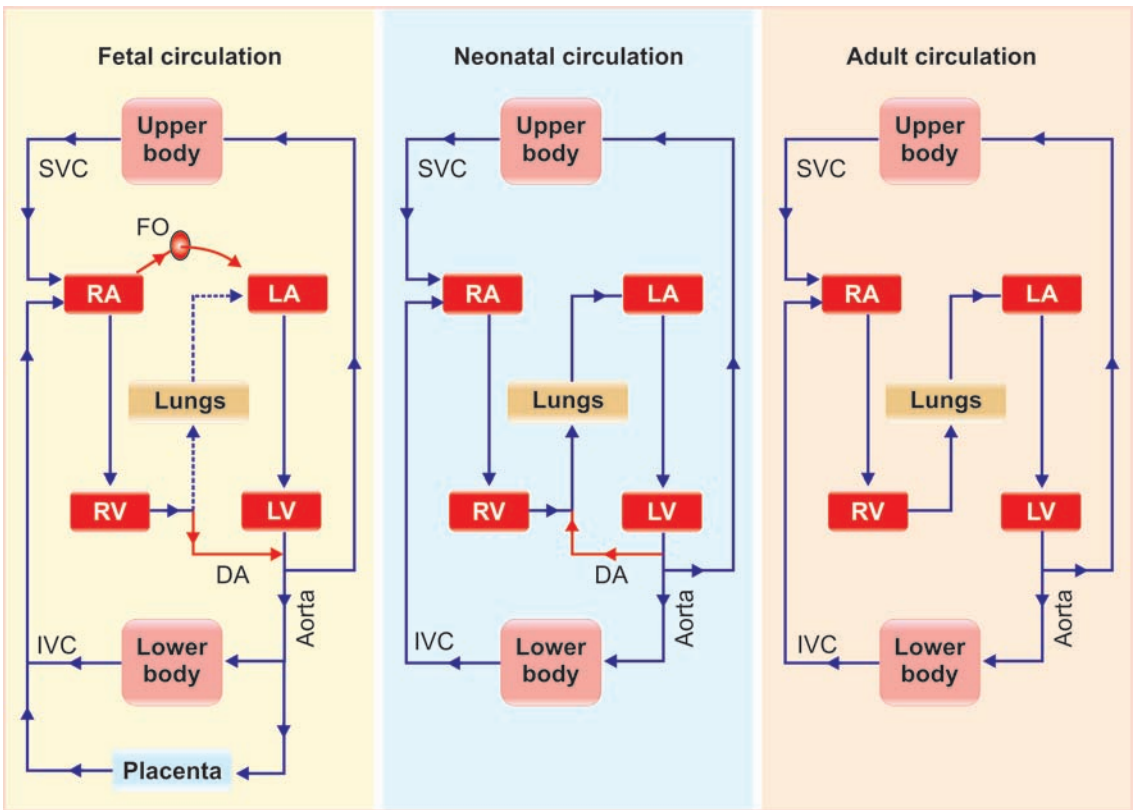

FIGURE 114.2: Fetal, neonatal and adult circulation. RA = Right atrium, LA = Left atrium, RV = Right ventricle, LV = Left ventricle, FO = Foramen ovale, DA = Ductus arteriosus, SVC = Superior vena cava, IVC = Inferior vena cava, Dashed blue line (Fetal circulation) indicates flow of very less quantity of blood.

left atrium. It causes increase in the left atrial pressure. Simultaneously, due to stoppage of blood from placenta, pressure in inferior vena cava is decreased. It leads to fall in right atrial pressure. Thus, the pressure in right atrium is less and the pressure in left atrium is already high. This causes the closure of foramen ovale. Within few days after birth, the foramen ovale closes completely and fuses with the atrial wall.

■ 4. REVERSAL OF BLOOD FLOW IN DUCTUS ARTERIOSUS

In fetus, since pulmonary arterial pressure is very high, the blood passes from pulmonary artery into aorta via ductus arteriosus. However, in neonatal life, since the systemic arterial pressure is more than pulmonary arterial pressure, the blood passes in **opposite direction** in ductus arteriosus, i.e. from systemic aorta into pulmonary aorta (Fig. 114.2). The **reversed flow** in ductus arteriosus is heard as **continuous murmur** in infants.

■ 5. CLOSURE OF DUCTUS VENOSUS

Due to the contraction of smooth muscle near junction between umbilical vein and ductus venosus, the constriction and closure of ductus venosus occurs. Later, the ductus venosus becomes fibrous band.

■ 6. CLOSURE OF DUCTUS ARTERIOSUS

Ductus arteriosus starts closing due to narrowing. It closes completely after 2 days and the adult type of circulation starts. In some rare cases, the ductus arteriosus does not close. It remains intact producing a continuous murmur. This condition with intact ductus arteriosus is known as **patent ductus arteriosus** (Refer to Chapter 106).

Hemorrhage

Chapter 115

- DEFINITION
- TYPES AND CAUSES OF HEMORRHAGE
  - ACCIDENTAL HEMORRHAGE
  - CAPILLARY HEMORRHAGE
  - INTERNAL HEMORRHAGE
  - POSTPARTUM HEMORRHAGE
  - HEMORRHAGE DUE TO PREMATURE DETACHMENT OF PLACENTA
- COMPENSATORY EFFECTS OF HEMORRHAGE
  - IMMEDIATE COMPENSATORY EFFECTS OF HEMORRHAGE
  - DELAYED COMPENSATORY EFFECTS OF HEMORRHAGE

DEFINITION

Hemorrhage is defined as the excess loss of blood due to rupture of blood vessels.

TYPES AND CAUSES OF HEMORRHAGE

Hemorrhage occurs due to various reasons. Based on the cause, hemorrhage is classified into five categories:

1. ACCIDENTAL HEMORRHAGE

Accidental hemorrhage occurs in road accidents and industrial accidents, which are very common in the developed and developing countries.

Accidental hemorrhage is of two types:

i. Primary hemorrhage, which occurs immediately after the accident
ii. Secondary hemorrhage, which takes place sometime (about few hours) after the accident.

2. CAPILLARY HEMORRHAGE

Capillary hemorrhage is the bleeding due to the rupture of blood vessels, particularly capillaries. It is very common in brain **(cerebral hemorrhage)** and heart during cardiovascular diseases. The rupture of the capillary is followed by spilling of blood into the surrounding areas.

3. INTERNAL HEMORRHAGE

Internal hemorrhage is the bleeding in viscera. It is caused by rupture of blood vessels in the viscera. The blood accumulates in viscera.

4. POSTPARTUM HEMORRHAGE

Excess bleeding that occurs immediately after labor (delivery of the baby) is called postpartum hemorrhage. In some cases, it is very severe and leads to major complications.

5. HEMORRHAGE DUE TO PREMATURE DETACHMENT OF PLACENTA

In some cases, the placenta is detached from the uterus of mother before the due date of delivery causing severe hemorrhage.

COMPENSATORY EFFECTS OF HEMORRHAGE

Many effects are observed during and after hemorrhage. Effects are different in acute hemorrhage and chronic hemorrhage.

Acute Hemorrhage

Acute hemorrhage is the sudden loss of large quantity of blood. It occurs in conditions like accidents. Decreased blood volume in acute hemorrhage causes **hypovolemic shock** (Chapter 116).

Chronic Hemorrhage

Chronic hemorrhage is the loss of blood either by internal or by external bleeding over a long period of time. Internal bleeding occurs in conditions like ulcer. External bleeding occurs in conditions like hemophilia and excess vaginal bleeding (menorrhagia). Chronic hemorrhage produces different types of effects such as anemia.

Compensatory Effects

After hemorrhage, series of compensatory reactions develop in the body to cope up with the blood loss.

Compensatory effects of hemorrhage are of two types.

A. Immediate compensatory effects
B. Delayed compensatory effects.

■ IMMEDIATE COMPENSATORY EFFECTS OF HEMORRHAGE

1. *On Cardiovascular System*

Reduced blood volume after hemorrhage decreases venous return, ventricular filling and cardiac output. In severe hemorrhage, there is fall in blood pressure also. However, when blood loss is slow or less, the arterial blood pressure is not affected much. If it is affected it is restored quickly.

During mild hemorrhage

During slow or mild hemorrhage when there is loss of a small amount of blood up to 350 to 500 mL the blood pressure decreases slightly and soon it returns back to normal.

Mechanism involved in maintenance of blood pressure:

i. Usually when arterial blood pressure increases, the carotid and aortic baroreceptors are stimulated and send impulses to brain resulting in decrease in blood pressure (Chapter 103). During hemorrhage when the arterial blood pressure falls, baroreceptors become inactivated and stop discharging impulses.
ii. This increases the vasomotor tone leading to vasoconstriction. This type of reflex vasoconstriction occurs in all regions of the body except brain and heart.
iii. Vasoconstriction results in increase in the peripheral resistance
iv. Loss of blood also causes reflex constriction of veins
v. Venoconstriction enhances the venous return, ventricular filling and stroke volume
vi. Thus, because of increased peripheral resistance and stroke volume the arterial blood pressure is restored
vii. One more factor is involved in this mechanism. Vasoconstriction occurs in the organs having reservoir function such as skin, liver and spleen. Blood from these reservoir organs is directed into systemic circulation. This may compensate the volume of blood that is lost during hemorrhage.

During severe hemorrhage

When hemorrhage is severe with loss of about 1,500 to 2,000 mL of blood, the arterial blood pressure falls to a great extend. It is because of decreased venous return and stroke volume.

In the heart, the reflex tachycardia increases the quantity of metabolic products in myocardium. These metabolic products cause coronary vasodilatation.

2. *On Skin*

Vasoconstriction in skin, which occurs after hemorrhage decreases the cutaneous blood flow. It increases the deoxygenation of blood and large quantity of reduced hemoglobin is accumulated in cutaneous blood vessels. It results in greyish pallor color of skin.

Sometimes cyanosis develops in certain areas of the body. Skin also becomes cold due to less blood flow. Sweating is decreased.

3. *On Tissue Fluid*

Arteriolar constriction decreases the capillary pressure. Therefore, tissue fluid enters capillaries. It helps to compensate the blood loss. It also causes **hemodilution.**

4. *On Kidneys*

Constriction of afferent and efferent arterioles of kidneys after hemorrhage decreases the glomerular filtration rate (GFR) very much. Therefore, the urinary output decreases. The blood level of nitrogenous substances, particularly urea, increases resulting in uremia.

Severe hemorrhage leads to fall in arterial blood pressure and damage of renal tubules resulting in acute **renal failure.**

5. *On Renin Secretion*

Hypoxia produced after blood loss increases secretion of renin from kidney and the subsequent formation of

angiotensin II. Angiotensin II helps in restoring blood pressure by producing generalized vasoconstriction. It also increases release of aldosterone from adrenal cortex. Aldosterone causes retention of sodium and this helps increasing the blood pressure. Angiotensins III and IV are also involved in restoring the blood pressure (Chapter 50).

6. *On Secretion of Antidiuretic Hormone*

Antidiuretic hormone (ADH) is released in large quantities immediately after the hemorrhage. It is probably due to increased osmolality of body fluid by aldosterone induced sodium retention. ADH promotes water retention and helps in restoring osmolality and volume of ECF.

7. *On Secretion of Catecholamines*

Sympathetic activity increases due to blood loss. It causes secretion of large quantities of catecholamines, which are also involved in restoring blood pressure by the vasoconstrictor effect.

8. *On Respiration*

Hemorrhage causes stagnant hypoxia because of decrease in venous return, cardiac output and velocity of blood flow. Hypoxia stimulates the chemoreceptors leading to increase in respiratory rate. The catecholamines, which are secreted in large quantities due to hemorrhage, increase the respiratory movements through **reticular activating system** (RAS).

9. *On Nervous System*

i. *On brain*

Though hemorrhage causes vasoconstriction in many organs of the body, it causes vasodilatation in brain. It is because of increased **sympathetic activity.** However, the blood flow to brain is not affected very much after hemorrhage because of **autoregulation.**

ii. *On reticular formation*

Catecholamines stimulate the RAS. It causes restlessness, anxiety and increased motor activity after hemorrhage. The respiratory movements are also accelerated due to stimulation of RAS.

iii. *Fainting*

When hemorrhage is severe, cardiac output decreases and blood pressure falls. The autoregulation in brain fails to cope up with the hypotension. So, the blood flow to brain decreases resulting in **fainting** (refer Chapter 116 for details).

iv. *Cerebral ischemia*

When the blood flow to brain is severely affected due to hypoxia, ischemia of the brain tissues develops within 5 minutes. It causes irreversible damage to brain tissues.

■ DELAYED COMPENSATORY EFFECTS OF HEMORRHAGE

If hemorrhage is not severe, some delayed compensatory reactions occur. These reactions help to restore blood volume, blood pressure and blood flow to different regions of the body.

Delayed reactions are:

1. Restoration of plasma volume
2. Restoration of plasma proteins
3. Restoration of red blood cell count and hemoglobin content.

1. *Restoration of Plasma Volume*

During the period of hemorrhage itself, tissue fluid starts entering the blood because of low capillary pressure. So, the plasma volume increases.

Because of increase in plasma volume, hemodilution occurs. So, the concentration of plasma proteins and hemoglobin is low. Transport of fluid from tissues is continued for long time after hemorrhage.

2. *Restoration of Plasma Proteins*

The reserve proteins stored in liver start mobilizing within few hours after hemorrhage. Liver also starts synthesizing the plasma proteins. Restoration of plasma proteins occurs within 3 to 4 days. Plasma proteins help to retain the fluid transported from tissues to blood.

3. *Restoration of Red Blood Cell Count and Hemoglobin Content*

Hypoxia that is developed after hemorrhage stimulates the secretion of erythropoietin from kidney. Erythropoietin in turn stimulates red bone marrow causing erythropoiesis. However, restoration of RBC count is a slow process. It takes about 4 to 6 weeks. Reticulocyte count increases in blood.

Hemoglobin content also comes back to normal level along with RBC count, if the diet contains adequate quantity of iron and proteins.

Circulatory Shock and Heart Failure

Chapter 116

- DEFINITION
- MANIFESTATIONS OF CIRCULATORY SHOCK
- STAGES OF CIRCULATORY SHOCK
  - FIRST STAGE OR COMPENSATED STAGE
  - SECOND STAGE OR PROGRESSIVE STAGE
  - THIRD STAGE OR IRREVERSIBLE STAGE
- TYPES AND CAUSES OF CIRCULATORY SHOCK
  - SHOCK DUE TO DECREASED BLOOD VOLUME
  - SHOCK DUE TO INCREASED VASCULAR CAPACITY
  - SHOCK DUE TO CARDIAC DISEASES
  - SHOCK DUE TO OBSTRUCTION OF BLOOD FLOW
- TREATMENT FOR CIRCULATORY SHOCK
  - BLOOD TRANSFUSION
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  - ADMINISTRATION OF PLASMA SUBSTITUTES
  - ADMINISTRATION OF SYMPATHOMIMETIC DRUGS
  - ADMINISTRATION OF GLUCOCORTICOIDS
  - OXYGEN THERAPY
  - BY CHANGING THE POSTURE
- HEART FAILURE
  - INTRODUCTION
  - CAUSES
  - SIGNS AND SYMPTOMS
  - TYPES
  - COMPENSATED VERSUS DECOMPENSATED HEART FAILURE

DEFINITION

Shock is a general term that refers to the depression or suppression of body functions produced by any disorder. **Circulatory shock** refers to the shock developed by inadequate blood flow throughout the body. It is a **life-threatening** condition and it may result in death if the affected person is not treated immediately.

MANIFESTATIONS OF CIRCULATORY SHOCK

Characteristic feature of all types of circulatory shock is the insufficient blood flow to the tissues particularly the brain. Major cause of decreased blood flow is the reduction in cardiac output.

Following are the manifestations of circulatory shock:

1. Whenever cardiac output is decreased, arterial blood pressure drops down
2. Low blood pressure produces reflex tachycardia and reflex vasoconstriction
3. Tachycardia decreases the diastolic period. So, filling of the heart reduces leading to decrease in stroke volume and systolic pressure. This decreases the pulse pressure below 20 mm Hg. Pulse also becomes feeble.

4. Stagnant hypoxia develops because of decreased velocity of blood flow
5. Skin becomes pale and cold due to the vasoconstriction
6. Along with hypoxia, cyanosis also develops in many parts of the body, particularly ear lobes and fingertips
7. Glomerular filtration rate (GFR) and urinary output are reduced due to fall in blood pressure and constriction of renal blood vessels
8. Metabolic activities of myocardium are accelerated because of reduced blood flow and increased heart rate. A large amount of lactic acid is produced, resulting in acidosis.
9. Acidosis decreases myocardial efficiency and pumping action of the heart leading to further reduction in cardiac output
10. So, the blood flow to vital organs is severely affected
11. Lack of blood flow to brain tissues produces ischemia resulting in fainting and irreparable damage of brain tissues
12. Finally the damage of brain tissues and cardiac arrest kill the victim.

■ STAGES OF CIRCULATORY SHOCK

Circulatory shock occurs in three stages:

1. First stage or compensated stage
2. Second stage or progressive stage
3. Third stage or irreversible stage.

■ FIRST STAGE OR COMPENSATED STAGE

First stage is also called **non-progressive stage.** When blood loss is less than 10% of total volume, the blood pressure decreases only moderately. And the regulatory mechanisms in the body operate successfully to re-establish normal blood pressure and normal blood flow throughout the body. Thus the shock becomes non-progressive and the person recovers. Regulatory mechanisms **involve negative feedback control.**

Regulatory mechanisms are:

i. Baroreceptor mechanism
ii. Renal mechanism
iii. ADH mechanism.

i. *Baroreceptor Mechanism*

Ischemic response by baroreceptors initiates strong sympathetic stimulation, which causes vasoconstriction and tachycardia (Fig. 116.1).

ii. *Renal Mechanism*

Kidneys release large amount of renin that increases the angiotensin II formation. Angiotensin II produces intense vasoconstriction and increases release of aldosterone from adrenal cortex. Aldosterone in turn promotes retention of water and salts by kidneys. This helps in restoration of blood volume.

iii. *ADH Mechanism*

Antidiuretic hormone (ADH) released from posterior pituitary increases retention of water by kidneys. ADH also enhances vasoconstriction.

Because of severe vasoconstriction caused by the regulatory mechanisms, normal blood pressure is re-established. Retention of water by kidneys and the consequent fluid shift mechanism that moves water from interstitial space and intestinal lumen restores the blood volume. And the person recovers if shock is not severe enough to progress further. With proper treatment, the progression can be arrested completely.

■ SECOND STAGE OR PROGRESSIVE STAGE

Second stage is also called **decompensated stage.** When the shock is severe, positive feedback system develops so that regulatory mechanisms become inadequate to compensate. And the shock enters progressive stage. With immediate and appropriate treatment, this stage of shock can be reversed.

During this stage, blood pressure falls to a low level, which is not adequate to maintain the blood flow to cardiac muscle. So the myocardium starts deteriorating because of lack of nutrition and oxygen. **Toxic substances** released from tissues also suppress the myocardium. Particularly, the bacterial toxin called **endotoxin affects** the myocardium severely (Fig. 116.2).

Loss of blood flow also causes suppression of vasomotor system and the sympathetic system. This causes further fall in blood pressure. Due to low pressure, **thrombosis** starts in small blood vessels like capillaries. Now the capillary permeability increases allowing passage of fluid from blood vessels into interstitial space. Finally because of tissue deterioration severe symptoms start appearing. And the shock progresses to irreversible stage.

■ THIRD STAGE OR IRREVERSIBLE STAGE

Third stage is the last stage prior to the collapse. It is also called **refractory stage.** Irreversible stage leads to death regardless of type of treatment offered to the patient. It is because the brain fails to function due to severe **cerebral ischemia.** The blood pressure falls drastically. Even the infusion of blood fails to restore blood pressure. Finally, cardiac failure occurs due to decrease in the myocardial activity and reduced arteriolar tone resulting in **death** of the affected person. Details of this stage are given in Figure 116.3.

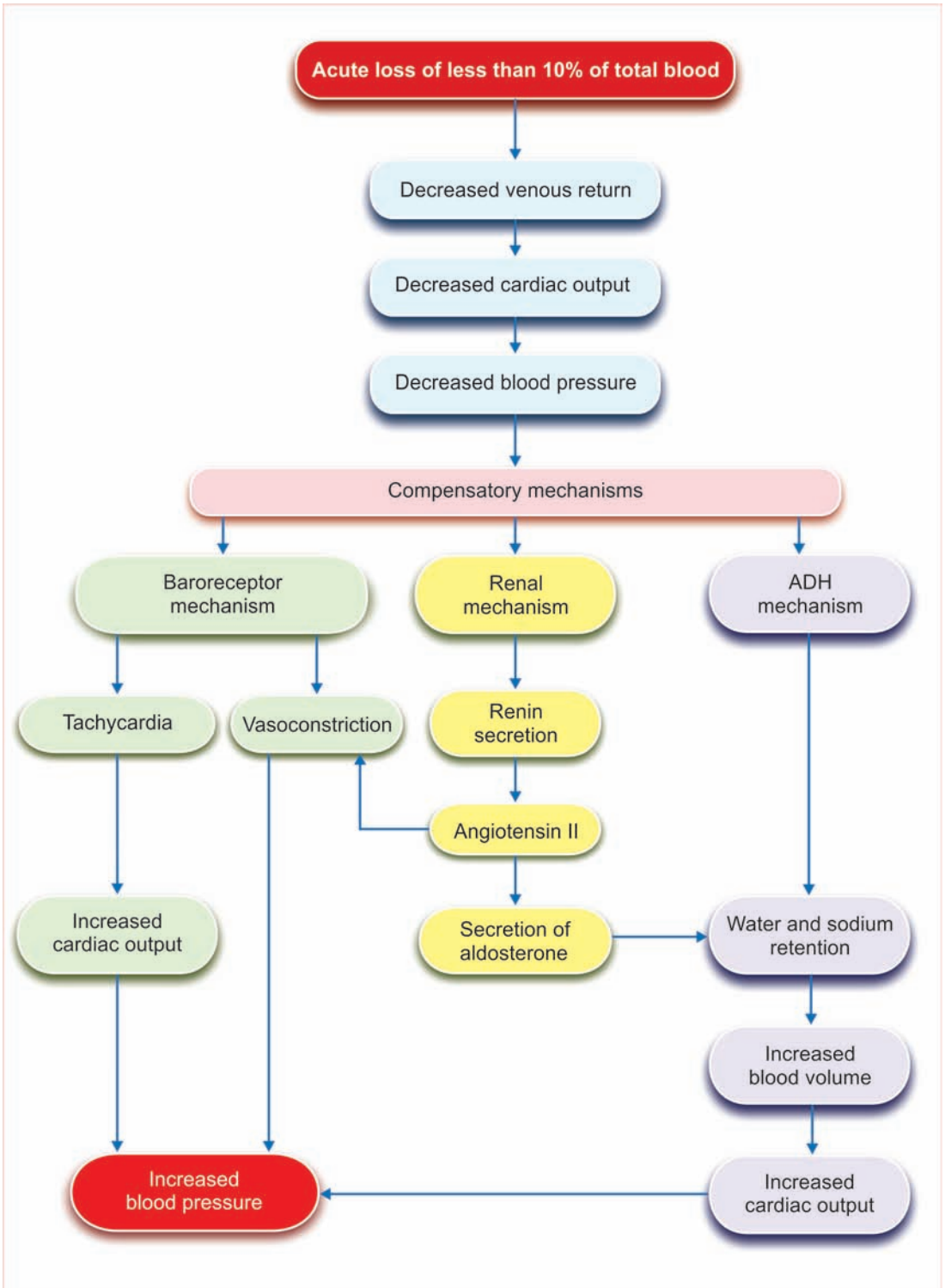

FIGURE 116.1: Compensated stage of circulatory shock. ADH = Antidiuretic hormone.

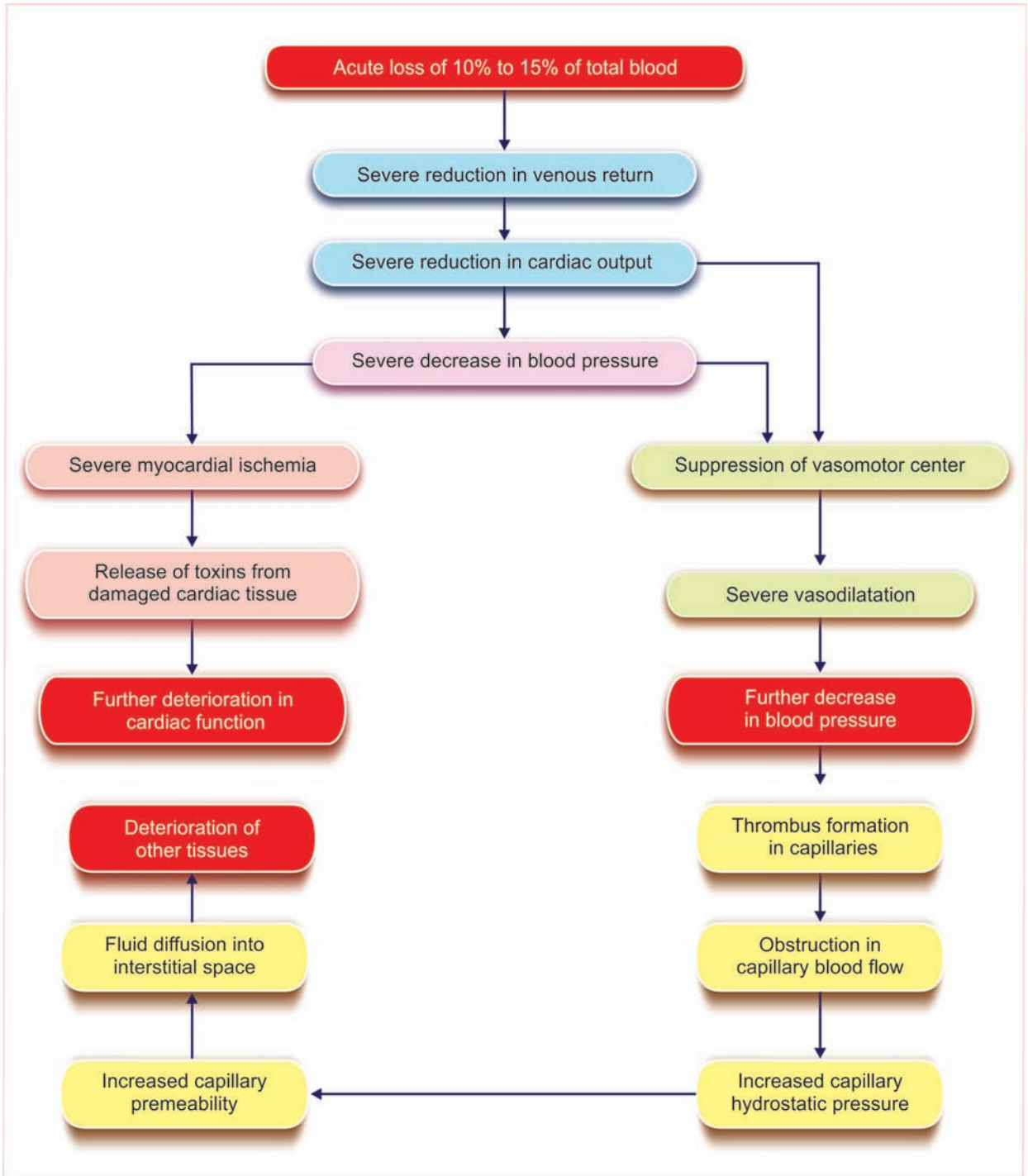

FIGURE 116.2: Progressive stage of circulatory shock

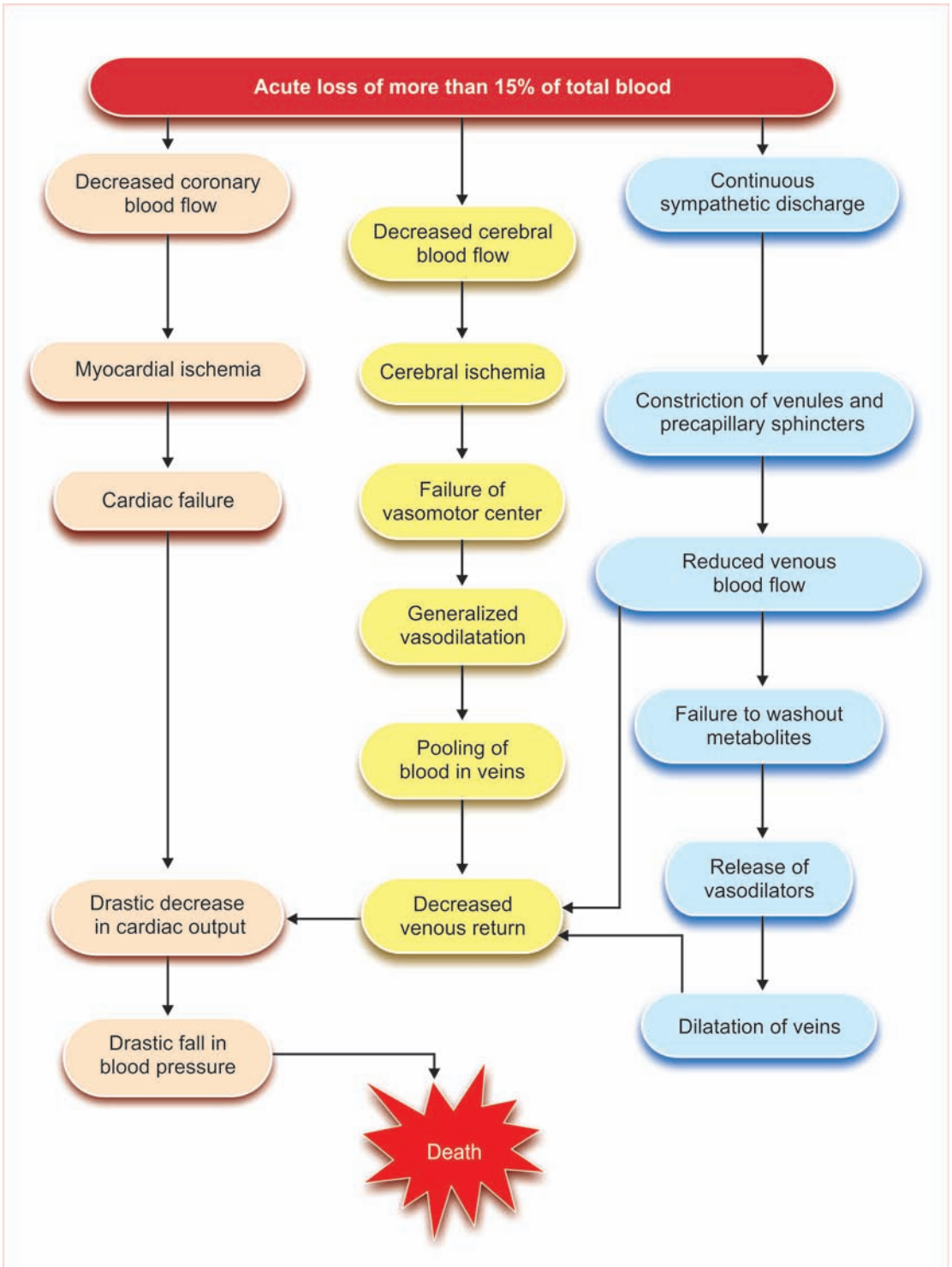

FIGURE 116.3: Irreversible stage of circulatory shock

TYPES AND CAUSES OF CIRCULATORY SHOCK

Circulatory shock is primarily classified into four types (Fig. 116.4).

A. Shock due to decreased blood volume
B. Shock due to increased vascular capacity
C. Shock due to cardiac disease
D. Shock due to obstruction of blood flow.

SHOCK DUE TO DECREASED BLOOD VOLUME – HYPOVOLEMIC SHOCK

Shock due to decreased blood volume is called hypovolemic shock or cold shock. It occurs when there is acute loss of at least 10% to 15% of blood. Loss of blood less than 10% may not produce any significant effect because of immediate compensatory mechanism.

Important Manifestations of Hypovolemic Shock

1. Decrease in cardiac output
2. Low blood pressure
3. Thin thready pulse
4. Pale and cold skin
5. Increase in respiratory rate
6. Restlessness or lethargy.

Pathological Conditions when Hypovolemic Shock Occurs

1. Hemorrhage: Hemorrhagic shock
2. Trauma: Traumatic shock
3. Surgery: Surgical shock
4. Burns: Burn shock
5. Dehydration: Dehydration shock.

1. *Hemorrhagic Shock*

Hemorrhagic shock is the shock due to hemorrhage. **Acute hemorrhage** as in the case of accident causes shock. **Chronic hemorrhage** as in ulcers does not produce shock. Details of effects of hemorrhage are given in the Chapter 115.

2. *Traumatic Shock*

Trauma means serious injury or wound caused by some external force. Shock caused by trauma is called traumatic shock. Shock occurs due to the damage of muscles and bones, which is common in battlefields and road accidents. Apart from loss of blood, the plasma escapes to the tissue spaces.

Following are the common symptoms of traumatic shock:

Crush syndrome

Crush syndrome is the condition characterized by renal failure when the limb of a person is crushed or compressed in traumatic condition. **Myoglobin** and some **toxic substances** released from affected muscles damage the renal tubular cells leading to **degeneration** of renal tubules. Stimulation of somatic afferents from the damaged muscles causes constriction of renal blood vessels. All these factors result in **renal failure.**

Reperfusion injury

Reperfusion injury refers to dysfunction of myocardium, blood vessels or any other tissue, which is induced by restoration of blood flow to previously ischemic tissue. It is also called **injury by reperfusion.**

Due to compression or damage during traumatic conditions, the ischemic tissues release some toxic substances. Later, when blood supply is restored to the

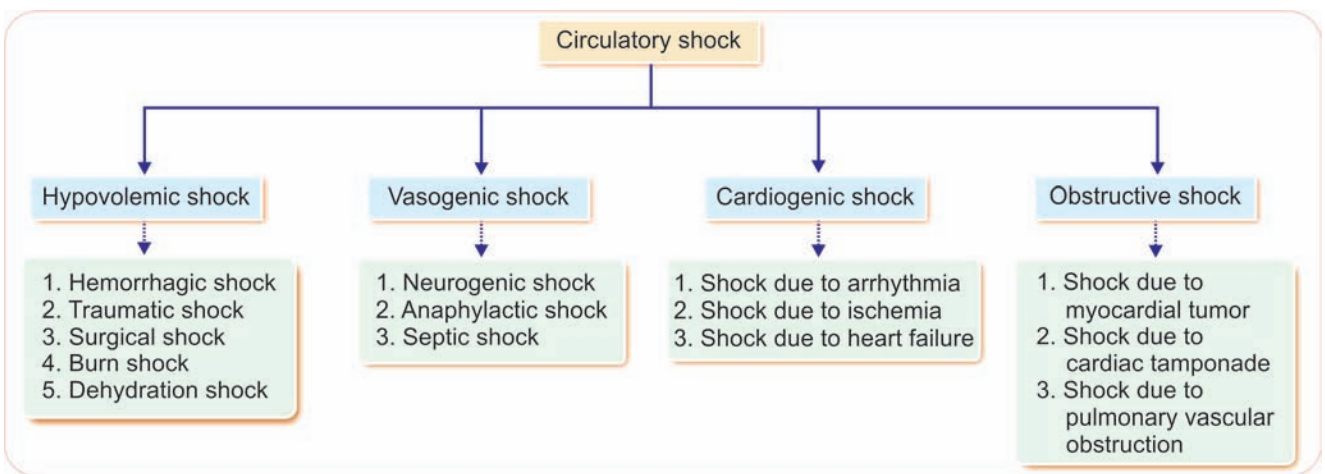

FIGURE 116.4: Different types of circulatory shock

tissues again, the **toxic substances** enter the tissues and cause further damage of the tissues. Common instance is myocardial reperfusion injury.

3. *Surgical Shock*

Surgical shock is the shock developed by surgical procedures. Surgical shock develops due to some reasons like internal hemorrhage, external hemorrhage and **dehydration** that occur during or after surgical procedures.

4. *Burn Shock*

Burn shock is the shock produced by the effects of burn. In burns, loss of plasma through the burnt surface is more than the loss of whole blood. It decreases the ECF volume and plasma volume, resulting in **hemoconcentration.** This leads to sluggish blood flow, which decreases the cerebral blood flow causing shock.

5. *Dehydration Shock*

Shock due to dehydration is called dehydration shock. Dehydration means decrease in water content of the body. It decreases the blood volume resulting in shock. Refer Chapter 6 for the causes of dehydration.

■ SHOCK DUE TO INCREASED VASCULAR CAPACITY – VASOGENIC SHOCK

In this case, the blood volume is normal. Shock occurs because of inadequate blood supply to the tissues due to increased vascular capacity. Capacity of the vascular system increases by the extensive dilatation of blood vessels. It is also known as vasogenic or **low resistance** or **distributive shock.**

Causes and Types of Vasogenic Shock

1. Sudden loss of vasomotor tone: Neurogenic shock
2. Anaphylaxis: Anaphylactic shock
3. Sepsis: Septic shock.

1. *Neurogenic Shock*

Neurogenic shock is the type of shock characterized by sudden depression of nervous system due to extensive vasodilatation caused by loss of vasomotor tone.

Conditions when neurogenic shock develops

i. Ischemia of brain: Severe ischemia in medulla depresses the activity of vasomotor center
ii. General anesthesia
iii. Spinal anesthesia
iv. Emotional conditions: Extreme emotions cause sudden and exaggerated activity of autonomic nervous system, the subject faints because of neurogenic shock.

Syncope (Fainting)

Syncope or fainting is the sudden and transient (short-time) loss of consciousness and postural tone with spontaneous recovery. It occurs due to temporary inadequate cerebral blood flow.

Types of syncope:

i. *Vasovagal syncope or emotional fainting:* Fainting is caused by sudden stimulation of vagus nerve. It is also called **neurocardiogenic syncope.** It is due to extreme activation of parasympathetic division of autonomic nervous system. There is sudden decrease in heart rate (bradycardia) because of inhibition of myocardium by vagus. At the same time, the blood pressure also decreases (hypotension) due to severe vasodilatation by the parasympathetic nerve fibers (Fig. 116.5).

Simultaneously, sympathetic tone is decreased and it also causes vasodilatation leading to hypotension.

Because of bradycardia and hypotension, the cerebral blood flow decreases. This results in fainting. Vasovagal syncope is common in conditions like severe emotional distress and exertion.

ii. *Postural syncope:* Loss of consciousness because of **prolonged standing.** It is due to pooling of blood in lower limbs during prolonged standing resulting in decreased blood supply to the brain.

iii. *Micturition syncope:* Fainting during micturition. It is common in the patients who suffer from **orthostatic hypotension.** Fall in blood pressure while standing is called orthostatic hypotension (Chapter 103).

iv. *Effort syncope:* Fainting caused during exercise or any other strain. It is the common symptom in the patients with **stenosis of semilunar valves.** These patients faint during exercise or any other physical strain. It is due to the failure of the heart to increase the cardiac output, when the tissues need more blood flow.

v. *Cough syncope:* Fainting while coughing. Sometimes, severe cough increases intrathoracic pressure, which reduces the venous return and cardiac output leading to fainting.

vi. *Carotid sinus syncope:* Fainting in persons wearing dress with **tight collar.** Tight collar of the dress exerts pressure over the region of carotid sinus. This leads to reduction in heart rate, vasodilatation and fainting.

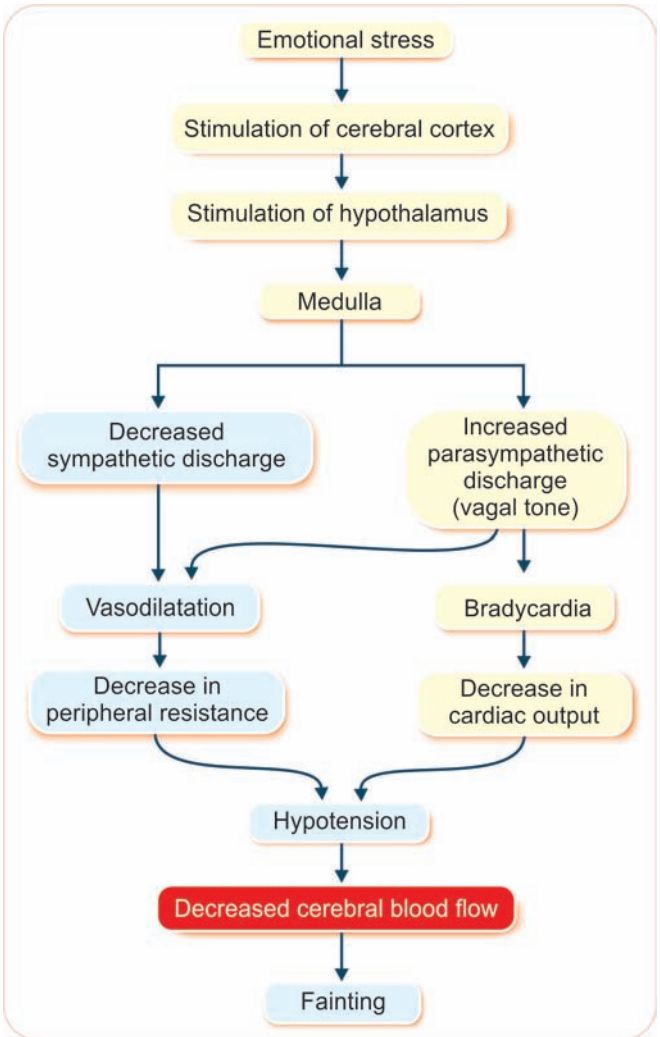

FIGURE 116.5: Schematic representation of vasovagal syncope

2. *Anaphylactic Shock*

Anaphylaxis means **exaggerated allergic reaction** to a foreign protein or antigen or any other substance to which the person has been previously sensitized (Chapter 17). Shock that develops during **anaphylactic reactions** is called anaphylactic shock. Shock occurs because of vasodilatation and sudden fall in blood pressure. It is caused by the chemical mediators such as histamine that are secreted during anaphylactic reaction.

3. *Septic Shock*

Sepsis is the pathological condition characterized by the presence of pathogenic organisms or their toxins in blood or tissues. Shock developed during sepsis is known as septic shock or **blood poisoning.**

Conditions when septic shock occurs

i. Infection of the uterus and fallopian tube, commonly occurring in abortion by instrumentation
ii. Infection of peritoneum
iii. Spreading of skin infection due to bacteria like streptococci or staphylococci
iv. Spread of infection from any other part of the body.

Septic shock develops due to the depression of myocardium, dilatation of blood vessels and increased permeability of capillary membrane. All these effects occur due to the toxic substances released by bacteria. Septic shock is also called as **vasogenic, cardiogenic** or **hypovolemic shock.**

Endotoxin shock

Endotoxin shock is the shock developed by a bacterial toxin called endotoxin. Endotoxin is a lipopolysaccharide. It causes vasodilatation and depresses myocardial activity. It also activates the macrophages to release cytokines. Endotoxin shock is very common during the infection of alimentary tract by **gram-negative bacteria** like colon bacilli. It is actually released from dead bacteria. Endotoxin shock can also occur in urinary tract infection.

■ SHOCK DUE TO CARDIAC DISEASES – CARDIOGENIC SHOCK

Shock due to cardiac disease is also called cardiogenic shock.

Conditions when Cardiogenic Shock Occurs

1. Arrhythmia, particularly those which lead to reduced cardiac output
2. Depressed activity of myocardium due to ischemia
3. Congestive cardiac disease.

■ SHOCK DUE TO OBSTRUCTION OF BLOOD FLOW – OBSTRUCTIVE SHOCK

Shock developed due to the obstruction of blood flow through circulatory system is called obstructive shock.

Conditions when Obstructive Shock Occurs

1. Tumor in myocardium
2. Cardiac tamponade (Chapter 100)
3. Obstruction of blood vessels in lungs due to embolism.

TREATMENT FOR CIRCULATORY SHOCK

Treatment for shock is based on the cause of the shock. Following are the various measures taken during the treatment of shock.

BLOOD TRANSFUSION

Transfusion of whole blood is done in hypovolemic shock except burn shock.

PLASMA TRANSFUSION

Plasma transfusion is very useful in burns or other shocks in which there is loss of more plasma.

ADMINISTRATION OF PLASMA SUBSTITUTES

Plasma substitute is a solution of a substance that is used for transfusion instead of plasma. Plasma substitutes are used when plasma is not available.

Commonly used Plasma Substitutes

i. Plasma expanders (solutions of sugar with high molecular weight such as dextran); such substances do not escape through capillary membrane
ii. Concentrated human serum albumin
iii. Hypertonic solutions, which cause drawing of fluid into blood from interstitial space.

ADMINISTRATION OF SYMPATHOMIMETIC DRUGS

Sympathomimetic drugs like epinephrine and norepinephrine are useful in neurogenic and anaphylactic shocks, which occur due to vasodilatation. These two drugs restore the blood pressure by vasoconstriction. However, the sympathomimetic drugs should not be used for longer period since, these drugs induce severe myocardial activity. In traumatic and cardiogenic shocks, dopamine is used.

ADMINISTRATION OF GLUCOCORTICOIDS

Glucocorticoids are administered in serious conditions. Glucocorticoids increase the glucose metabolism in damaged tissues, prevent further damage of tissues and increase the myocardial activity.

OXYGEN THERAPY

Oxygen therapy is given only in severe conditions involving reduced oxygenation of tissues.

BY CHANGING THE POSTURE

This is the first measure to be taken in cases of hemorrhagic and neurogenic shock. The head down position (by raising the bed at the foot end) increases venous return, cardiac output and cerebral blood flow. However, this should not be used for longer periods because prolonged head down position might affect the ventilation. It is because of effect of the increased pressure exerted by abdominal viscera on diaphragm.

HEART FAILURE

INTRODUCTION

Heart failure or **cardiac failure** is the condition in which the heart looses the ability to pump sufficient amount of blood to all parts of the body. Heart failure may involve left ventricle or right ventricle or both. It may be acute or chronic.

Acute Heart Failure

Acute heart failure refers to sudden and rapid onset of signs and symptoms of abnormal heart functions. Its symptoms are severe initially. However, the symptoms last for a very short time and the condition improves rapidly. Usually it requires treatment.

Chronic Heart Failure

Chronic heart failure is the heart failure that is characterized by the symptoms that appear slowly over a period of time and become worst gradually.

Congestive Heart Failure

Congestive heart failure is a general term used to describe the heart failure resulting in accumulation of fluid in lungs and other tissues. When heart is not able to pump blood through aorta, the blood remains in heart. It results in dilatation of the chambers and accumulation of blood in veins **(vascular congestion). Fluid retention** and **pulmonary edema** also occur in this condition.

CAUSES OF HEART FAILURE

Common causes of heart failure are:

1. Coronary artery disease
2. Defective heart valves
3. Arrhythmia
4. Cardiac muscle disease such as cardiomyopathy
5. Hypertension
6. Congenital heart disease

7. Diabetes
8. Hyperthyroidism
9. Anemia
10. Lung disorders
11. Inflammation of cardiac muscle **(myocarditis)** due to viral infection, drugs, alcohol, etc.

■ SIGNS AND SYMPTOMS OF HEART FAILURE

Signs and Symptoms of Chronic Heart Failure

1. Fatigue and weakness
2. Rapid and irregular heartbeat
3. Shortness of breathing
4. Fluid retention and weight gain
5. Loss of appetite
6. Nausea and vomiting
7. Cough
8. Chest pain, if developed by myocardial infarction.

Signs and Symptoms of Acute Heart Failure

Signs and symptoms of acute heart failure may be same as chronic heart failure. But the signs and symptoms appear suddenly and severely. When heart starts to fail suddenly, the fluid accumulates in lungs causing pulmonary edema. It results in sudden and severe shortness of breath, cough with pink, foamy mucus and heart palpitations. It may lead to sudden death, if not attended immediately.

■ TYPES OF HEART FAILURE

1. *Systolic Heart Failure*

Systolic heart failure is the heart failure due to the decreased ability of heart to contract. It may involve right heart or left heart or both. It is caused either by muscular weakness or valvular defect. Ventricles may be filled with blood but cannot pump it out with sufficient force. Ejection fraction decreases to about 20%. So the amount of blood pumped to the body and to the lungs is decreased. As a result, more amount of blood remains in ventricle. Later the blood starts accumulating in lungs or systemic veins or both. Usually the ventricle enlarges in systolic heart failure.

2. *Diastolic Heart Failure*

Diastolic heart failure is the heart failure that occurs when the ventricles cannot relax properly due to the stiffening of cardiac muscle. So, there is reduction in ventricular filling and cardiac output.

3. *Right Sided Heart Failure*

Right sided heart failure occurs due to loss of pumping action of the right side of the heart. Because of loss of pumping action of right ventricle, blood accumulates in right atrium and blood vessels. It causes edema in the feet, ankles, legs and abdomen.

4. *Left Sided Heart Failure*

Left sided heart failure is due to the loss of pumping action of the left side of the heart. It causes congestion of lungs.

■ COMPENSATED VERSUS DECOMPENSATED HEART FAILURE

Chronic heart failure may be compensated or de-compensated.

Compensated Heart Failure

Compensated heart failure is the heart failure with adequate cardiac output. Heart tries to maintain cardiac output by normal compensatory mechanisms such as increase in heart rate, increase in force of ventricular contraction and ventricular hypertrophy. In compensated heart failure, the symptoms are stable and features of fluid retention and pulmonary edema are absent. Eventually, in most of the patients the heart can no longer meet the demand even by compensatory mechanisms and this condition leads to decompensated heart failure.

Decompensated Heart Failure

Decompensated heart failure is the heart failure with inadequate cardiac output. It is characterized by deterioration and sudden and drastic worsening of cardiac function, resulting in death.

Cardiovascular Adjustments during Exercise

Chapter 117

- INTRODUCTION
- TYPES OF EXERCISE
  - DYNAMIC EXERCISE
  - STATIC EXERCISE
- AEROBIC AND ANAEROBIC EXERCISES
  - AEROBIC EXERCISE
  - ANAEROBIC EXERCISE
  - METABOLISM IN AEROBIC AND ANAEROBIC EXERCISES
- SEVERITY OF EXERCISE
  - MILD EXERCISE
  - MODERATE EXERCISE
  - SEVERE EXERCISE
- EFFECTS OF EXERCISE
  - ON BLOOD
  - ON BLOOD VOLUME
  - ON HEART RATE
  - ON CARDIAC OUTPUT
  - ON VENOUS RETURN
  - ON BLOOD FLOW TO SKELETAL MUSCLES
  - ON BLOOD PRESSURE

INTRODUCTION

During exercise, there is an increase in metabolic needs of body tissues, particularly the muscles.

Various adjustments in the body during exercise are aimed at:

1. Supply of various metabolic requisites like nutrients and oxygen to muscles and other tissues involved in exercise
2. Prevention of increase in body temperature.

TYPES OF EXERCISE

Exercise is generally classified into two types depending upon the type of muscular contraction:

1. Dynamic exercise
2. Static exercise.

Cardiovascular changes are slightly different in these two types of exercise.

DYNAMIC EXERCISE

Dynamic exercise primarily involves the **isotonic muscular contraction.** It keeps the joints and muscles moving. Examples are swimming, bicycling, walking, etc. Dynamic exercise involves **external work,** which is the shortening of muscle fibers against load.

In this type of exercise, the heart rate, force of contraction, cardiac output and systolic blood pressure increase. However, the diastolic blood pressure is unaltered or decreased. It is because, during dynamic exercise, peripheral resistance is unaltered or decreased depending upon the severity of exercise.

■ STATIC EXERCISE

Static exercise involves **isometric muscular contraction** without movement of joints. Example is pushing heavy object. Static exercise does not involve **external work.**

During this exercise, apart from increase in heart rate, force of contraction, cardiac output and systolic blood pressure, the diastolic blood pressure also increases. It is because of increase in peripheral resistance during static exercise.

■ AEROBIC AND ANAEROBIC EXERCISES

Based on the type of metabolism involved, exercise is classified into two types:

1. Aerobic exercise
2. Anaerobic exercise.

The terms aerobic and anaerobic refer to the energy producing process during exercise. Aerobic means **'with air'** or **'with oxygen'.** Anaerobic means **'without air'** or **'without oxygen'.** Both aerobic and anaerobic exercises are required to maintain physical fitness.

■ AEROBIC EXERCISE

Aerobic exercise involves activities with lower intensity, which is performed for longer period. The energy is obtained by utilizing nutrients in the presence of oxygen and hence it is called aerobic exercise. At the beginning, the body obtains energy by burning glycogen stored in liver. After about 20 minutes, when stored glycogen is exhausted the body starts burning fat. Body fat is converted into glucose, which is utilized for energy.

Aerobic exercise requires large amount of oxygen to obtain the energy needed for prolonged exercise.

Examples of aerobic exercise:

1. Fast walking
2. Jogging
3. Running
4. Bicycling
5. Skiing
6. Skating
7. Hockey
8. Soccer
9. Tennis
10. Badminton
11. Swimming
12. Rowing.

■ ANAEROBIC EXERCISE

Anaerobic exercise involves exertion for short periods followed by periods of rest. It uses the muscles at high intensity and a high rate of work for a short period. Body obtains energy by burning glycogen stored in the muscles without oxygen hence it is called anaerobic exercise.

Burning glycogen without oxygen liberates lactic acid. Accumulation of lactic acid leads to fatigue. Therefore, this type of exercise cannot be performed for longer period. And a recovery period is essential before going for another burst of anaerobic exercise. Anaerobic exercise helps to increase the muscle strength.

Examples of anaerobic exercise:

1. Pull-ups
2. Push-ups
3. Weightlifting
4. Sprinting
5. Any other rapid burst of strenuous exercise.

■ METABOLISM IN AEROBIC AND ANAEROBIC EXERCISES

When a person starts doing some exercise like jogging, bicycling or swimming, the muscles start utilizing energy. In order to have quick energy during the first few minutes, the muscles burn glycogen stored in them. During this period, fat is not burnt. Only glycogen is burnt and it is burnt without using oxygen. This is called **anaerobic metabolism.** Lactic acid is produced during this period. Presence of lactic acid causes some sort of burning sensation in the muscles particularly the muscles of arms, legs and back.

Muscles burn all the muscle glycogen within 3 to 5 minutes. If the person continues the exercise beyond this, glycogen stored in liver is converted into glucose, which is transported to muscles through blood. Now the body moves into **aerobic metabolism.** The glucose obtained from liver is burnt in the presence of oxygen. No more lactic acid is produced. So the burning sensation in the muscles disappears. Proper breathing is essential during this period so that adequate oxygen is supplied to the muscles to extract the energy from glucose. The supply of glucose from liver in combination with adequate availability of oxygen allows the person to continue the exercise.

Utilization of all the glycogen stored in liver is completed by about 20 minutes. If the exercise is continued beyond this, the body starts utilizing the fat. The stored fat called body fat is converted into carbohydrate, which is utilized by the muscles. This allows the person to do the exercise for a longer period.

■ SEVERITY OF EXERCISE

Cardiovascular and other changes in the body depend upon the severity of exercise also. Based on severity, the exercise is classified into three types.

1. MILD EXERCISE

Mild exercise is the very simple form of exercise like slow walking. Little or no change occurs in cardiovascular system during mild exercise.

2. MODERATE EXERCISE

Moderate exercise does not involve strenuous muscular activity. So, this type of exercise can be performed for a longer period. Exhaustion does not occur at the end of moderate exercise. The examples of this type of exercise are fast walking and slow running.

3. SEVERE EXERCISE

Severe exercise involves strenuous muscular activity. The severity can be maintained only for short duration. Fast running for a distance of 100 or 400 meters is the best example of this type of exercise. Complete exhaustion occurs at the end of severe exercise.

EFFECTS OF EXERCISE ON CARDIOVASCULAR SYSTEM

1. ON BLOOD

Mild hypoxia developed during exercise stimulates the juxtaglomerular apparatus to secrete erythropoietin. It stimulates the bone marrow and causes release of red blood cells. Increased carbon dioxide content in blood decreases the pH of blood.

2. ON BLOOD VOLUME

More heat is produced during exercise and the thermoregulatory system is activated. This in turn, causes secretion of large amount of sweat leading to:

i. Fluid loss
ii. Reduced blood volume
iii. Hemoconcentration
iv. Sometimes, severe exercise leads to even dehydration.

3. ON HEART RATE

Heart rate increases during exercise. Even the thought of exercise or preparation for exercise increases the heart rate. It is because of impulses from cerebral cortex to medullary centers, which reduces vagal tone.

In moderate exercise, the heart rate increases to 180 beats/minute. In severe muscular exercise, it reaches 240 to 260 beats/minute. Increased heart rate during exercise is mainly because of **vagal withdrawal.** Increase in sympathetic tone also plays some role.

Increased heart rate during exercise is due to four factors:

i. Impulses from proprioceptors, which are present in the exercising muscles; these impulses act through higher centers and increase the heart rate
ii. Increased carbon dioxide tension, which acts through medullary centers
iii. Rise in body temperature, which acts on cardiac centers via hypothalamus, increased temperature also stimulates SA node directly
iv. Circulating catecholamines, which are secreted in large quantities during exercise.

4. ON CARDIAC OUTPUT

Cardiac output increases up to 20 L/minute in moderate exercise and up to 35 L/minute during severe exercise. Increase in cardiac output is directly proportional to the increase in the amount of oxygen consumed during exercise.

During exercise, the cardiac output increases because of increase in heart rate and stroke volume. Heart rate increases because of **vagal withdrawal.** Stroke volume increases due to increased force of contraction. Because of vagal withdrawal, sympathetic activity increases leading to increase in rate and force of contraction.

5. ON VENOUS RETURN

Venous return increases remarkably during exercise because of muscle pump, respiratory pump and splanchnic vasoconstriction (Chapter 98).

6. ON BLOOD FLOW TO SKELETAL MUSCLES

There is a great increase in the amount of blood flowing to skeletal muscles during exercise. In resting condition, the blood supply to the skeletal muscles is 3 to 4 mL/100 g of the muscle/minute. It increases up to 60 to 80 mL in moderate exercise and up to 90 to 120 mL in severe exercise.

During the muscular activity, stoppage of blood flow occurs when the muscles contract. It is because of compression of blood vessels during contraction. And in between the contractions, the blood flow increases.

Sometimes the blood supply to muscles starts increasing even during the preparation for exercise. It is due to the sympathetic activity. Sympathetic nerves cause vasodilatation in muscles. The sympathetic nerve fibers causing vasodilatation in skeletal muscle are called **sympathetic cholinergic fibers** since these fibers secrete **acetylcholine** instead of noradrenaline.

Several other factors also are responsible for the increase in blood flow to muscles during exercise. All such factors increase the amount of blood flow to muscles by means of dilatation of blood vessels of the muscles. Such factors are:

i. Hypercapnea
ii. Hypoxia
iii. Potassium ions
iv. Metabolites like lactic acid
v. Rise in temperature
vi. Adrenaline secreted from adrenal medulla
vii. Increased sympathetic cholinergic activity.

■ 7. ON BLOOD PRESSURE

During moderate isotonic exercise, the systolic pressure is increased. It is due to increase in heart rate and stroke volume. Diastolic pressure is not altered because peripheral resistance is not affected during moderate isotonic exercise.

In severe exercise involving isotonic muscular contraction, the systolic pressure enormously increases but the diastolic pressure decreases. Decrease in diastolic pressure is because of the decrease in peripheral resistance. Decrease in peripheral resistance is due to vasodilatation caused by metabolites.

During exercise involving isometric contraction, the peripheral resistance increases. So, the diastolic pressure also increases along with systolic pressure.

Blood Pressure after Exercise

Large quantities of metabolic end products are produced during exercise. These substances accumulate in the tissues, particularly the skeletal muscle. Metabolic end products cause vasodilatation. So, the blood pressure falls slightly below the resting level after the exercise. However, the pressure returns to resting level quickly as soon as the metabolic end products are removed from muscles.

QUESTIONS IN CARDIOVASCULAR SYSTEM

LONG QUESTIONS

1. Define cardiac cycle. Describe various events of cardiac cycle with pressure and volume changes.
2. Define electrocardiogram. Describe the waves, segments and intervals of normal ECG. Add a note on ECG leads.
3. Give the definitions, normal values and variations of cardiac output. Explain the factors regulating cardiac output.
4. What is cardiac output? Enumerate the various methods to measure cardiac output and explain the measurement of cardiac output by applying Fick principle.
5. Describe the innervation of heart and the regulation of heart rate.
6. Define arterial blood pressure. Describe the nervous regulation of arterial blood pressure.
7. Describe renal mechanism of (long term) regulation of arterial blood pressure.
8. What is the normal blood flow through coronary circulation? Explain the phasic changes, measurement and regulation of coronary blood blow.
9. Give an account of cerebral circulation.
10. Define hemorrhage. Explain various effects of hemorrhage.
11. Describe the cardiovascular and respiratory changes during exercise.

SHORT QUESTIONS

1. Action potential in cardiac muscle.
2. Pacemaker.
3. Pacemaker potential.
4. Conductive system in heart.
5. All-or-none law.
6. Refractory period in cardiac muscle.
7. Isometric contraction period.
8. Atrial pressure changes during cardiac cycle.
9. Ventricular pressure changes during cardiac cycle.
10. Ventricular volume changes during cardiac cycle.
11. Ejection fraction.
12. Heart sounds.
13. First and second heart sounds.
14. Phonocardiogram.
15. Cardiac murmurs.
16. Waves of normal ECG.
17. ECG leads.
18. Mean QRS vector.
19. Vectorcardiogram.
20. Sinus arrhythmia.
21. Heart block.
22. Extrasystole.
23. Stokes-Adams syndrome.
24. Abnormal pacemaker.
25. Current of injury.
26. Effect of electrolyte changes on heart.
27. Venous return.
28. Peripheral resistance.
29. Fick principle.
30. Cardiac catheterization.
31. Cardiac function curves.
32. Cardiac centers.
33. Nerve supply to heart.
34. Vagal tone.
35. Marey reflex.
36. Sinoaortic mechanism.
37. Buffer nerves.
38. Baroreceptors.
39. Chemoreceptors.
40. Bainbridge reflex.
41. Streamline and turbulent flow of blood.
42. Windkessel effect.
43. Mean volume of blood flow.
44. Velocity of blood flow.
45. Circulation time.
46. Autoregulation.
47. Determinants of arterial blood pressure.
48. Vasomotor center.
49. Vasomotor tone.
50. Nerve supply to blood vessels.
51. Renal regulation of blood pressure.
52. Vasoconstrictor substances.
53. Vasodilator substances.
54. Renin-angiotensin mechanism.
55. Hypertension.
56. Venous pressure.
57. Capillary pressure.
58. Arterial pulse.
59. Phlebogram.
60. Phasic changes in coronary blood flow.
61. Regulation of coronary circulation.
62. Coronary occlusion.
63. Myocardial infarction.
64. Angina pectoris.
65. Physiological shunt in heart.
66. Measurement of cerebral blood flow.

67. Regulation of cerebral blood flow.
68. Cushing reflex.
69. Stroke or cardiovascular accident.
70. Capillary circulation (microcirculation).
71. Shunt in capillaries.
72. Cutaneous circulation.
73. Vascular responses of skin.
74. Lewis triple response.
75. Fetal circulation.
76. Neonatal circulation.
77. Hemorrhage.
78. Manifestations of circulatory shock.
79. Syncope or fainting.
80. Vasovagal syncope.
81. Cardiovascular changes in moderate exercise.
82. Effect of exercise on blood pressure.