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A QUICK REVIEW OF CENTRAL NERVOUS SYSTEM

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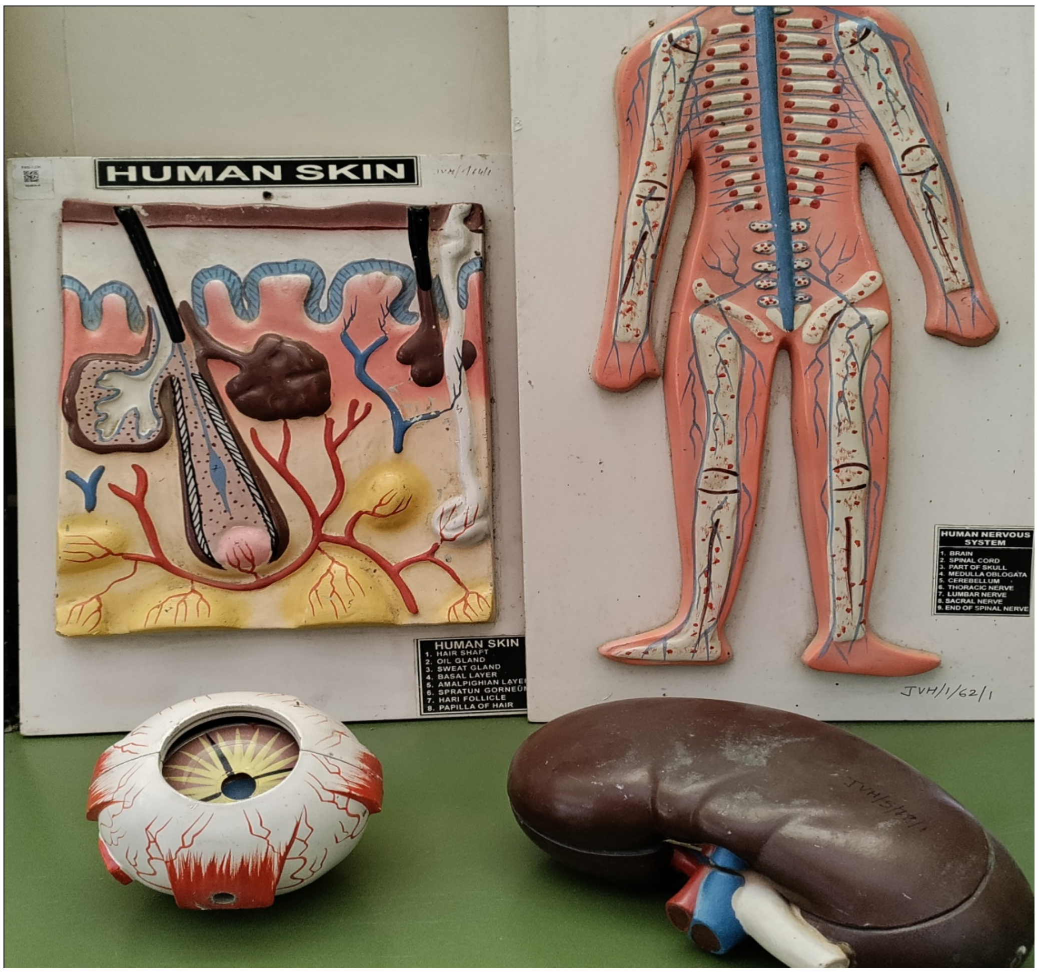

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CHAPTER – 1

INTRODUCTION OF CARDIOVASCULAR SYSTEM

Heart and **blood Vessels** are included in cardiovascular system**.** Blood is pumped by heart into the blood vessels which 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 is situated in between two lungs in the mediastinum. Heart is a four chamber structure of 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 limbs2. **Inferior vena cava** that returns venous blood from lower parts of the body .Right atrium communicates with right ventricle through tricuspid valve. Wall of right ventricle is thick. Venous blood from the right atrium enters the right

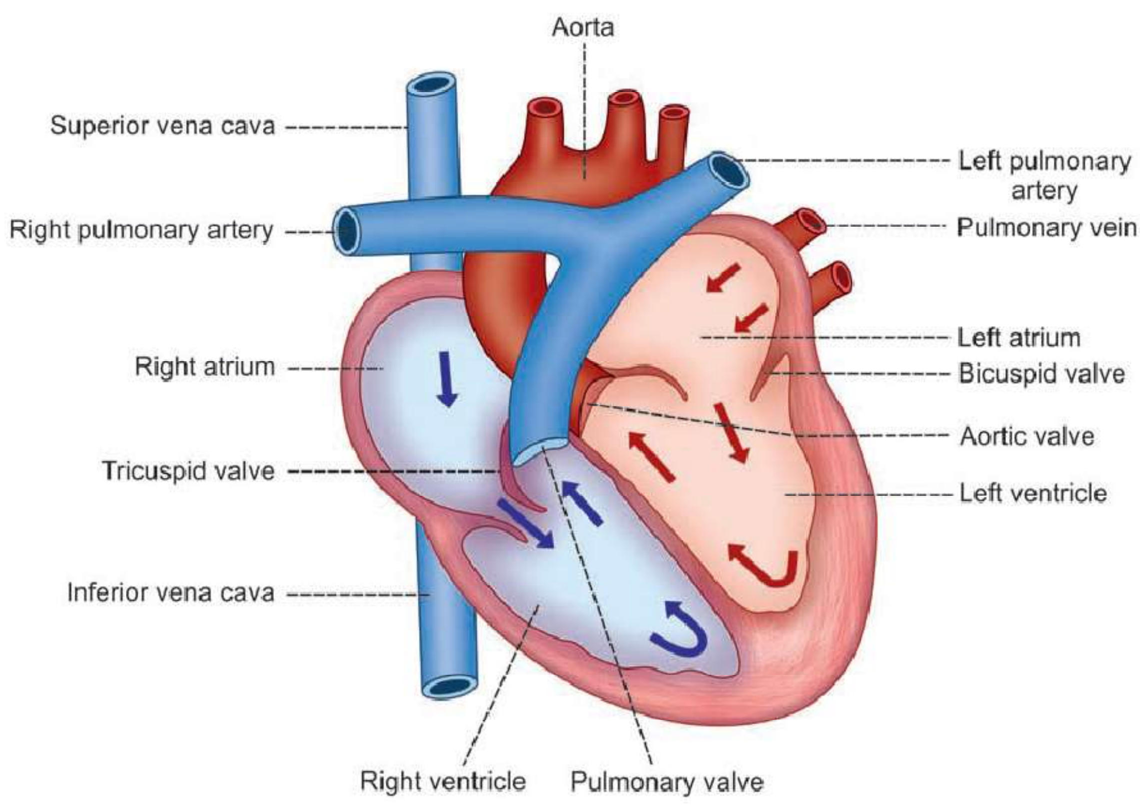
ventricle through this valve. From the right ventricle, pulmonary artery arises. It carries the venous blood from right ventricle to lungs. In the lungs, the deoxygenated blood is oxygenated.

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

Septa is present in between right and left atria are called **interatrial septum.** Right and left ventricles are separated from one another by **interventricular septum.** The upper part 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. called **P cells.** Sinoatrial (SA) node forms the pacemaker in human 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 made up of 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.

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.

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

ENDOCARDIUM

Inner most layer of heart wall is called endocardium. 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

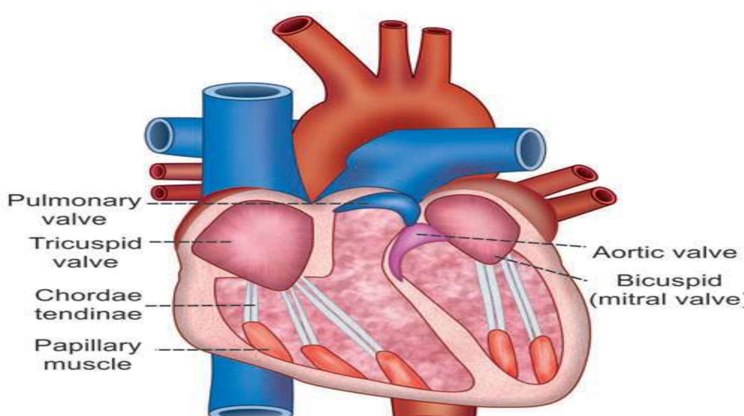
There are four valves in human heart. Two valves are in between atria and the ventricles called atrioventricular valves. Other two are the semilunar valves, placed at the opening of blood vessels arising from ventricles, namely systemic aorta and pulmonary artery. Valves of the heart permit the flow 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 . 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 **chordate 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. 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 divided into four types:

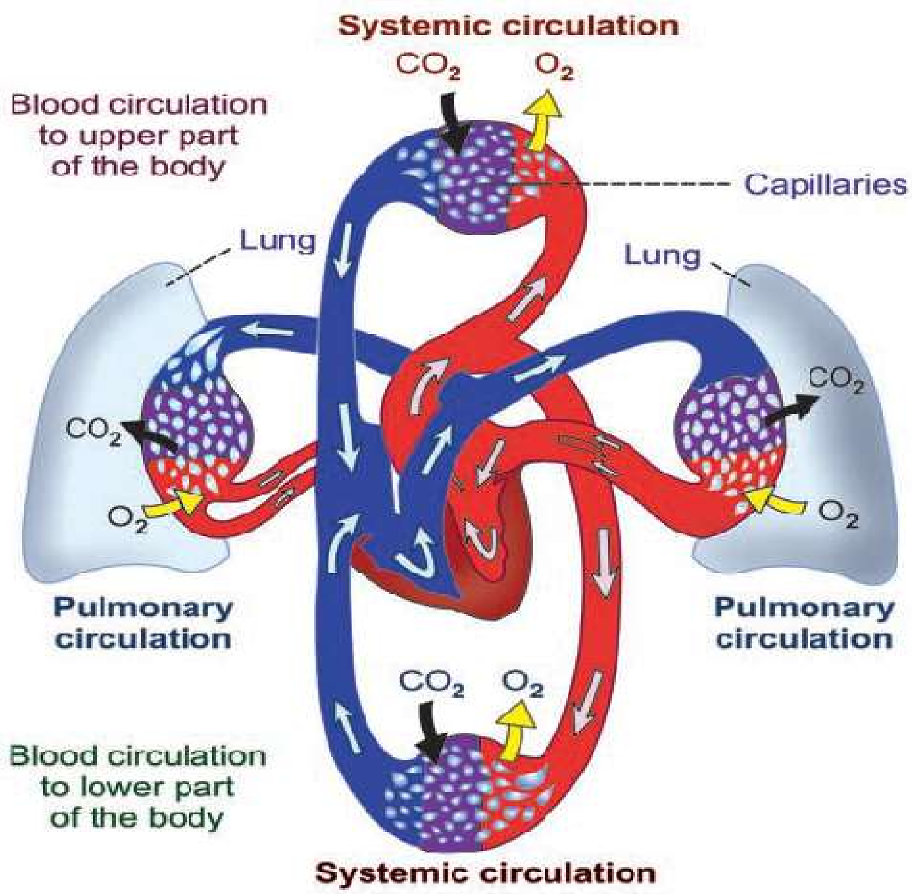
1. Chronotropic action
2. Inotropic action
3. Dromotropic action
4. Bathmotropic action.

Regulation of Actions of Heart

Heart are continuously regulated by nerves and hormones. 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.



CHAPTER- 2

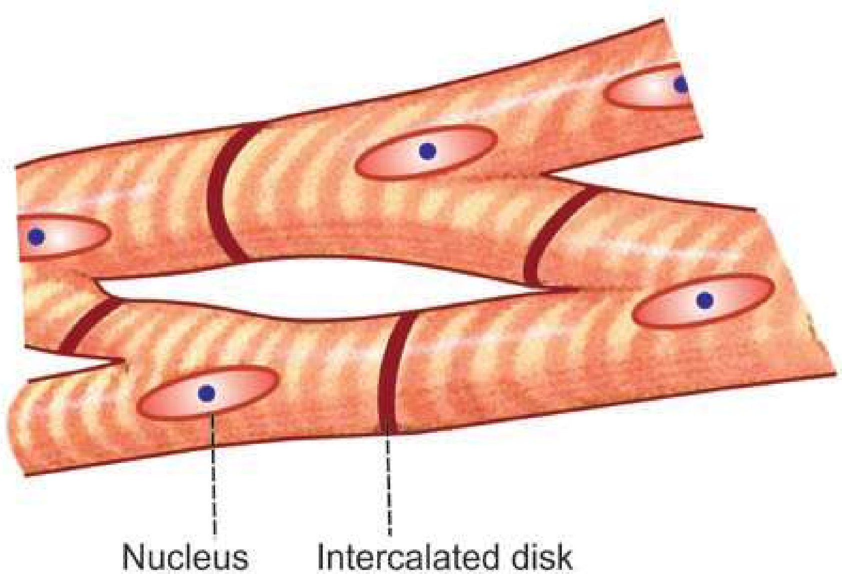
PHYSIOLOGY OF CARDIAC MUSCLE

The heart is composed of three major types of cardiac muscle: *atrial muscle, ventricular muscle,* and specialized *excitatory* and *conductive muscle* fibers. The atrial and ventricular types of muscle contract in much the same way as skeletal muscle, except that the duration of contraction is much longer. The specialized excitatory and conductive fibers of the heart, however, contract only feebly because they contain few contractile fibrils; instead, they exhibit either automatic rhythmical electrical discharge in the form of action potentials or conduction of the action potentials through the heart, providing an excitatory system that controls the rhythmical beating of the heart.

PHYSIOLOGIC ANATOMY OF CARDIAC MUSCLE

cardiac muscle is *striated* in the same manner as in skeletal muscle. Further, cardiac muscle has typical myofibrils that contain *actin* and *myosin filaments* almost identical to those found in skeletal muscle; these filaments lie side by side and slide during contraction in the same manner as occurs in skeletal muscle.

In other ways, however, cardiac muscle is quite different from skeletal muscle.



Cardiac Muscle Is a Syncytium.

The dark areas crossing the cardiac muscle fibers are called *intercalated discs;* they are actually cell membranes that separate individual cardiac muscle cells from one another. cardiac muscle fibers are made up of many individual cells connected in series and in parallel with one another. At each intercalated disc the cell membranes fuse with one another to form permeable “communicating” junctions (gap junctions) that allow rapid diffusion of ions.

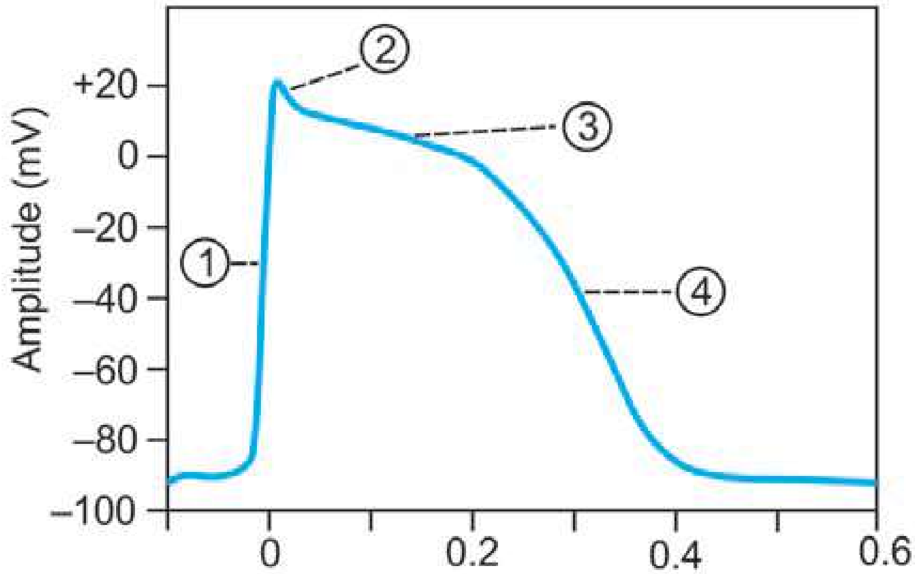
Therefore, from a functional point of view, ions move with ease in the intracellular fluid along the longitudinal axes of the cardiac muscle fibers so that action potentials travel easily from one cardiac muscle cell to the next, past the intercalated discs. Thus, cardiac muscle is a *syncytium* of many heart muscle cells in which the cardiac cells are so interconnected that when one cell becomes excited, the action potential rapidly spreads to all of them.

The heart actually is composed of two syncytiums: the *atrial syncytium,* which constitutes the walls of the two atria, and the *ventricular syncytium,* which constitutes the walls of the two ventricles. The atria are separated from the ventricles by fibrous tissue that surrounds the atrioventricular (A-V) valvular openings between the atria and ventricles. Normally, potentials are not conducted from the atrial syncytium into the ventricular syncytium directly through this fibrous tissue. Instead, they are conducted only by way of a specialized conductive system called the *A-V bundle,* a bundle of conductive fibers several millimeters in diameter that is discussed in

This division of the muscle of the heart into two functional syncytiums allows the atria to contract a short time ahead of ventricular contraction, which is important for effectiveness of heart pumping.

ACTION POTENTIALS IN CARDIAC MUSCLE

The *action potential* recorded in a ventricular muscle fiber, averages about 105 millivolts, which means that the intracellular potential rises from a very negative value, about −85 millivolts, between beats to a slightly positive value, about +20 millivolts, during each beat. After the initial *spike,* the membrane remains depolarized for about 0.2 second, exhibiting a *plateau*, followed at the end of the plateau by abrupt repolarization. The presence of this plateau in the action potential causes ventricular contraction to last as much as 15 times as long in cardiac muscle as in skeletal muscle.



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.

RHYTHMICITY

autorhythmicity or **self-excitation** is the another name of rhythmicity which has the ability to produce regular impulses and its Property is present in all the tissues of heart. However, heart has a specialized contractile structure known as pacemaker from which the discharge of impulses is rapid. From here, the impulses spread to other parts through the specialized conductive system.

Sinoatrial Node

Sinoatrial (SA) node is a small modified cardiac muscle, present in the superior part of lateral wall of right atrium, just below the opening of superior vena cava. It does 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 is more in SA node than in other parts. It is about 70 to 80/minute.

CONDUCTIVITY s

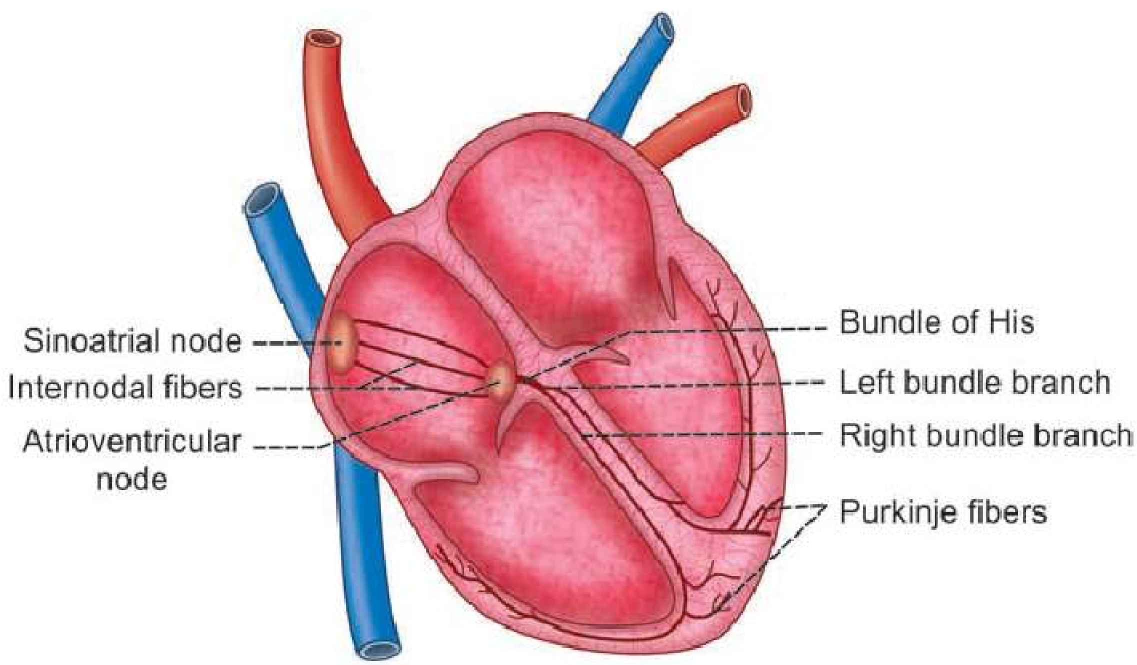
Specialized conductive system of heart passes impulses from SA node which are transmitted to other parts of the heart.

CONDUCTIVE SYSTEM IN HUMAN HEART

Junctional tissues is also known as Conductive tissues of the heart which is formed by the modified cardiac muscle fibers, which conduct the impulses very fast from SA node to the ventricles.

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.

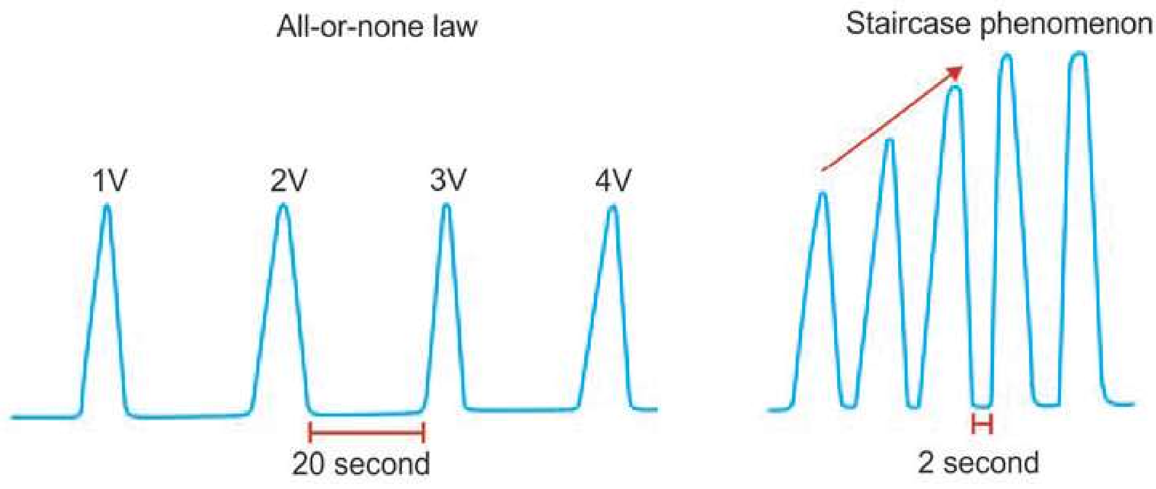
All these fibers from SA node converge on AV node and interdigitate with fibers of AV node. From AV node, the **bundle of His** arises. It divides into right and left branches, which run on either side of the interventricular septum. From each branch of bundle of His, many **Purkinje fibers** arise and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

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



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.

CHAPTER- 3
CARDIAC CYCLE

Cardiac cycle is defined as the succession of (sequence of) **coordinated events** taking place in the heart during each beat. Each heartbeat has two major periods.

1. systole
2. diastole

During systole, heart contracts and pumps the blood through arteries and During diastole, heart relaxes and blood is filled in the heart. All these changes are repeated in every heartbeat which is known as cardiac cycle.

EVENTS OF CARDIAC CYCLE

Events of cardiac cycle are classified into two:

1. Atrial events
2. Ventricular events.

DIVISIONS AND DURATION OF CARDIAC CYCLE

heart beats at a normal rate of 72/minuteand 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

Total-=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

Total=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 intraatrial 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

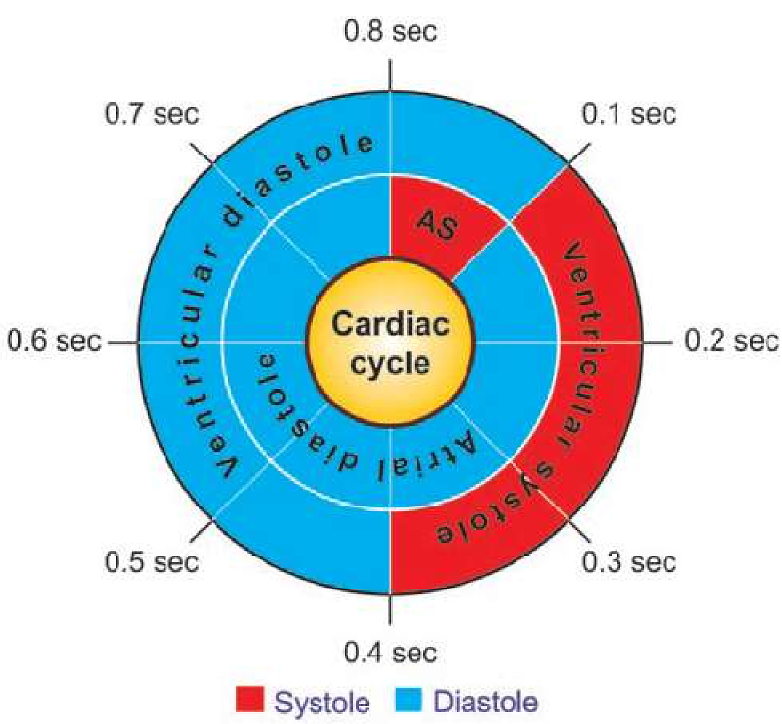
lasts for about 0.7 sec (accurate duration is 0.69 sec). This long atrial diastole is necessary because, this is the period during which atrial filling takes place. Right atrium receives deoxygenated blood from all over the body through superior and inferior venae cavae. Left atrium receives oxygenated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



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.

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 endsystolic volume. It is 60 to 80 mL per ventricle.

Measurement of end-diastolic volume Endsystolic 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)** .

Ejection Fraction

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

Determination of ejection fraction

- Ejection fraction (Ef) is the stroke volume divided byenddiastolic volume expressed in percentage.
- Stroke volume (SV) is, enddiastolic volume (EDV) minus endsystolicvolume (ESV).

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.

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 **is volumetric relaxation.**

During isometric relaxation period, once again all the valves of the heart are closed . 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

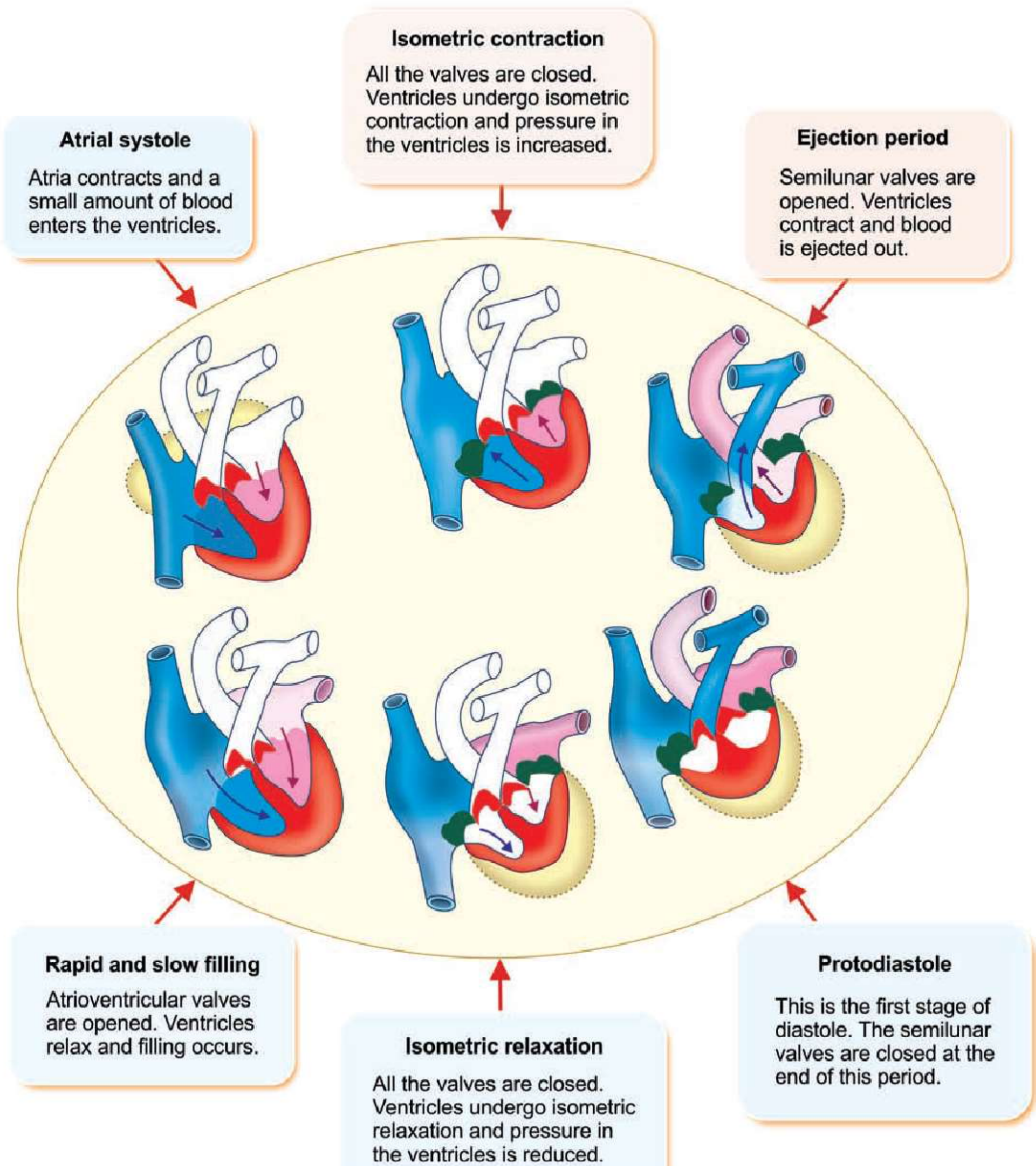
Last rapid filling phase occurs because of atrial systole. After slow filling period, the atria contract and push a small amount of blood into ventricles. About 10% of ventricular filling takes place during this period. Flow of additional amount of blood into ventricle due to atrial systole is called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

End-diastolic Volume

Enddiastolic 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

Enddiastolic volume is measured by the same methods, which are used to measure endsystolic
volume.



CHAPTER- 4
HEART SOUND

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.**

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.

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.

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.

CHAPTER-5
ELECTROCARDIOGRAM

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.

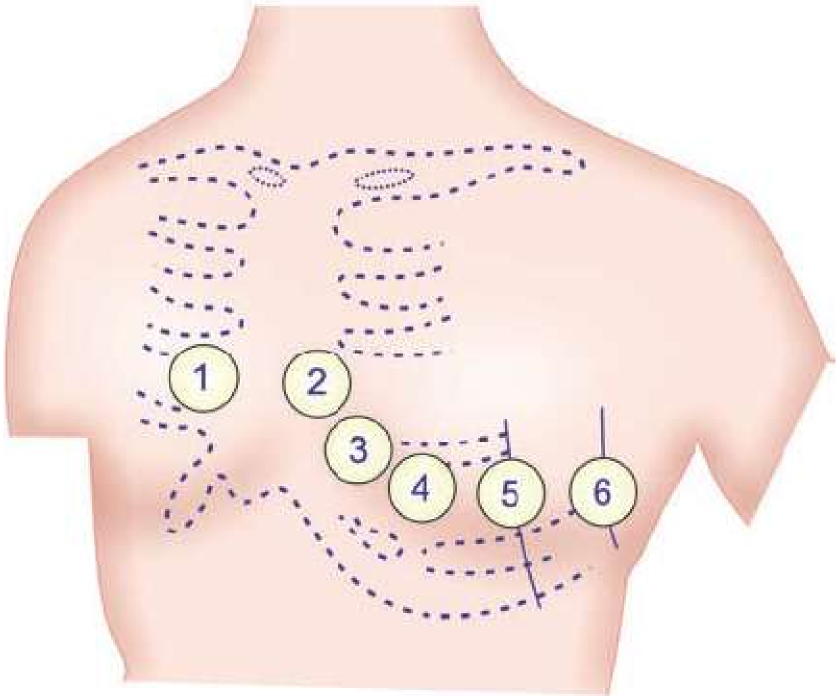
Electrocardiogram

Electrocardiogram (ECG or EKG from electrocardiogram 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.



Position of electrodes for chest leads (V1 to V6)

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 recorded as a separate wave in ECG because it merges with ventricular repolarization

‘QRS’ COMPLEX

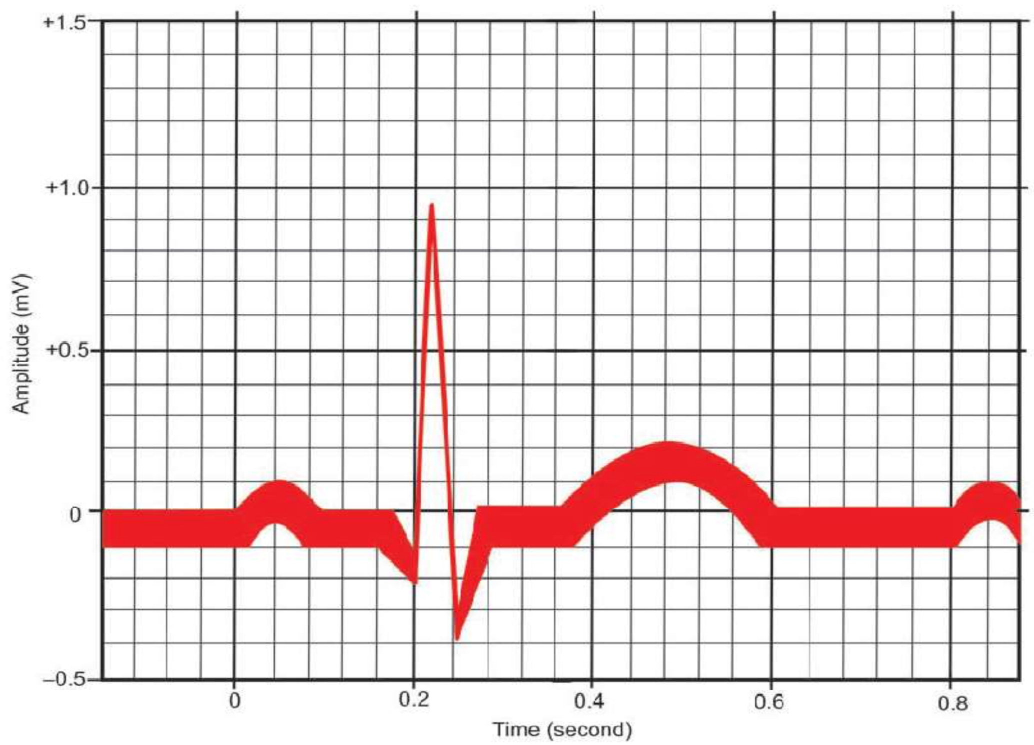
‘QRS’ complex is also called the **initial ventricular complex.** ‘Q’ wave is a small negative wave. It is continued as the tall ‘R’ wave, which is a positive wave. ‘R’ wave is followed by a small negative wave, the ‘S’ wave.

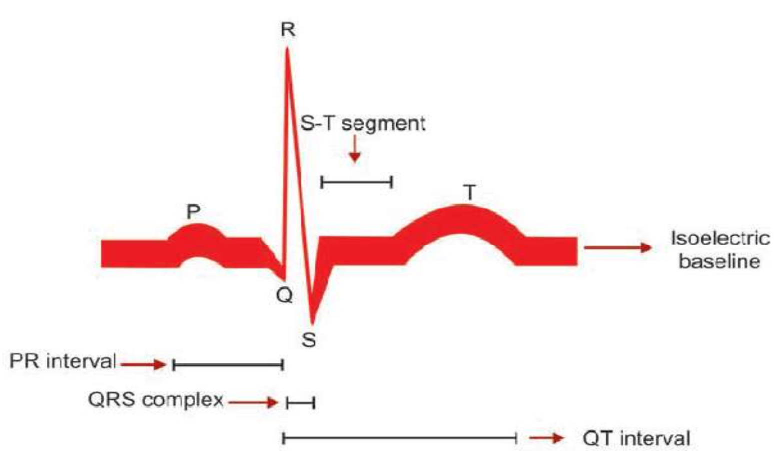
Cause

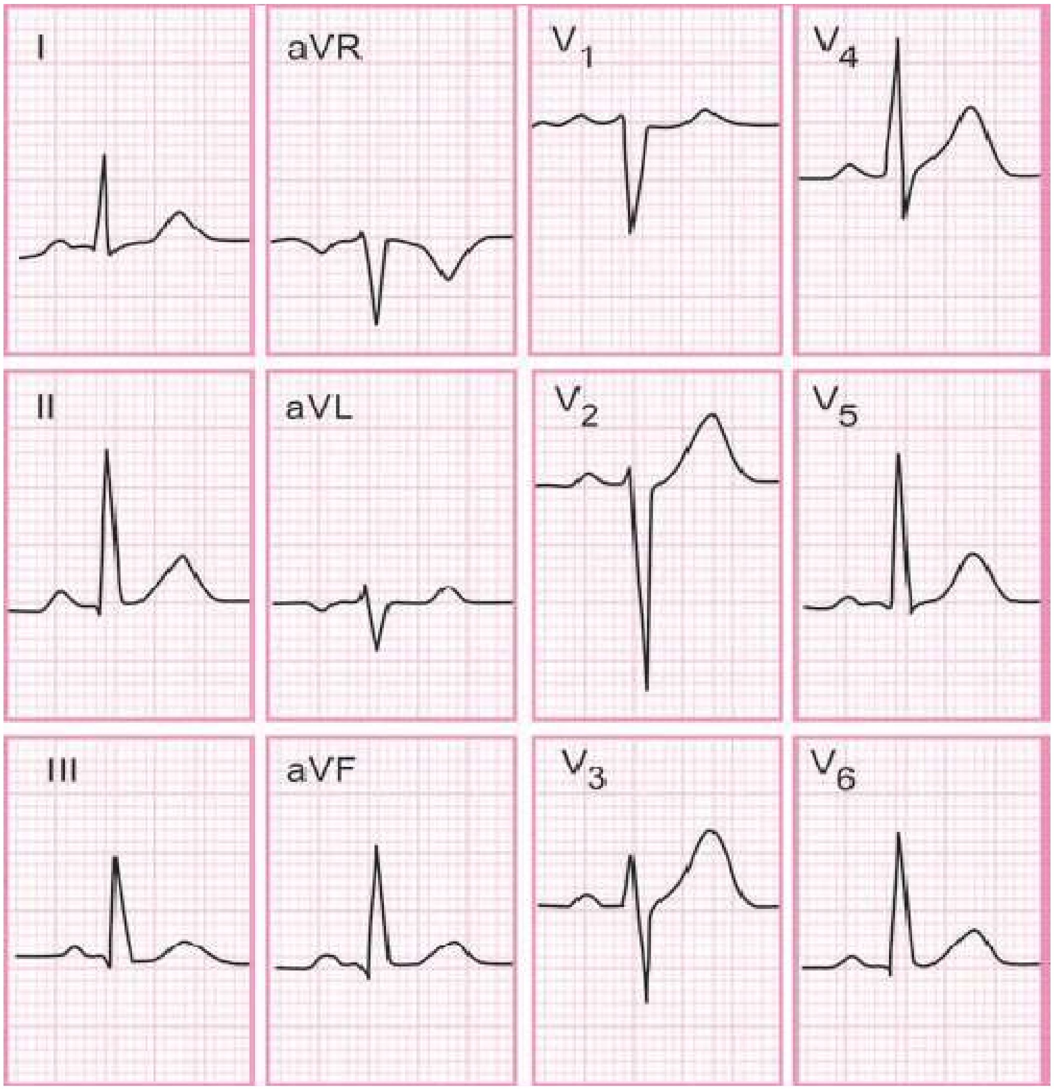
'QRS' complex is due to **depolarization** of **ventricular musculature.** 'Q' wave is due to the depolarization of basal portion of interventricular septum. 'R' wave is due to the depolarization of apical portion of interventricular septum and apical portion of ventricular muscle. 'S' wave is due to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







‘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.

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.

2. ***Heart Rate Variability***

Heart rate variability (HRV) refers to the beat-tobeat 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-6
CARDIAC OUTPUT

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.

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

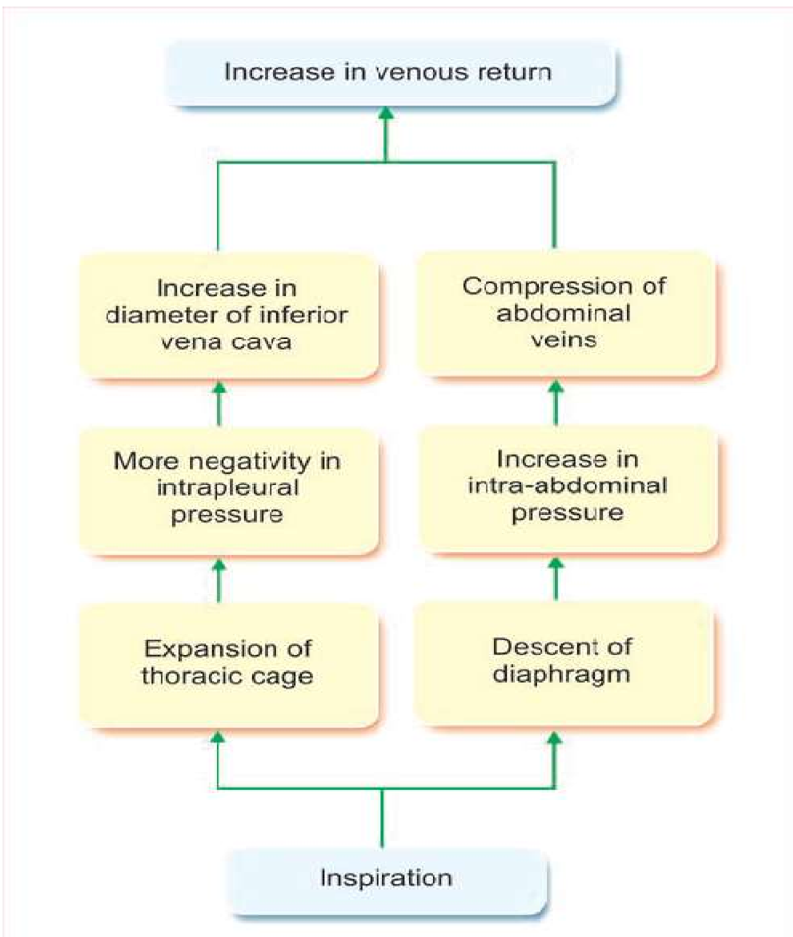
5. *Shock:* Due to poor pumping and circulation

6. *Hemorrhage:* Because of decreased blood volume.

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.



CHAPTER-7
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.**

MOTOR (EFFERENT) NERVE FIBERS TO HEART

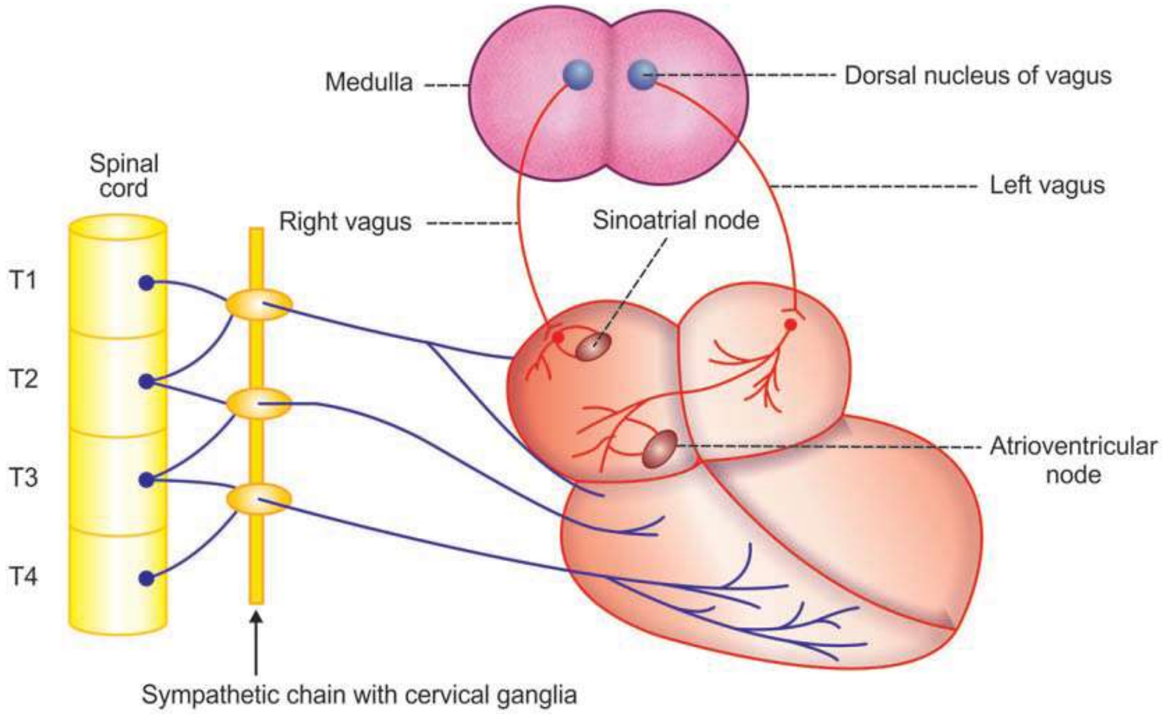
Heart receives efferent nerves from both the divisions of autonomic nervous system. Parasympathetic fibers arise from the medulla oblongata and pass through vagus nerve. Sympathetic fibers arise from upper thoracic (T1 to T4) segments of spinal cord.

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.


Medulla
Dorsal nucleus of vagus
Spinal cord
Left vagus
Right vagus
Sinoatrial node
T1
T2
T3
T4
Atrioventricular node
Sympathetic chain with cervical ganglia

CHAPTER-8
HEMORRHAGE

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 some ti m e (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** .

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.

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