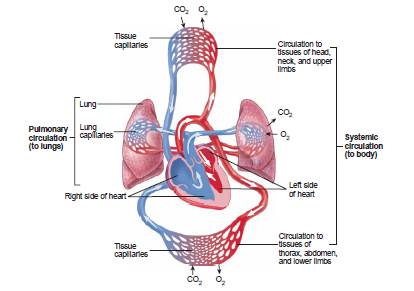
**CARDIOVASCULAR SYSTEM :-THE HEART:**

Approximately 370 years ago, it was established that the heart’s pumping action is essential to maintain the continuous circulation of blood throughout the body. The current understanding of the detailed function of this amazing pump, its regulation, and modern treatments for heart disease is, in comparison, very recent. The heart is actually two pumps in one. The right side of the heart receives blood from the body and pumps blood through the **pulmonary** **circulation,** which carries blood to the lungs and returns it to the left side of the heart. In the lungs, carbon dioxide diffuses from the blood into the lungs, and oxygen diffuses from the lungs into the blood. The left side of the heart pumps blood through the **systemic circulation,** which delivers oxygen and nutrients to all the remaining tissues of the body. From those tissues, carbon dioxide and other waste products are carried back to the right side of the heart.

The heart of a healthy 70 kg person pumps approximately 7200 L (approximately 1900 gallons) of blood each day at a rate of 5 L/min. For most people, the heart continues to pump for more than 75 years. During periods of vigorous exercise, the amount of blood pumped per minute increases several fold, but the life of the individual is in danger if the heart loses its ability to pump blood for even a few minutes. **Cardiology** is the medical specialty concerned with the diagnosis and treatment of heart disease.



Systemic and Pulmonary Circulation

The circulatory system consists of the pulmonary and systemic circulations. The right side of the heart pumps blood through vessels to the lungs and back to the left

side of the heart through the pulmonary circulation. The left side of the heart pumps blood through vessels to the tissues of the body and back to the right side of the

heart through the systemic circulation.

**FUNCTIONS OF THE HEART :-**

The functions of the heart include:-

1. Generating blood pressure. Contractions of the heart generate blood pressure, which is responsible for blood movement through the blood vessels.

2. Routing blood. The heart separates the pulmonary and systemic circulations and ensures better oxygenation of blood flowing to tissues.

3. Ensuring one-way blood flow. The valves of the heart ensure a one-way flow of blood through the heart and blood vessels.

4. Regulating blood supply. Changes in the rate and force of contraction match blood delivery to the changing metabolic needs of the tissues, such as during rest, exercise, and changes in body position.

**SIZE, SHAPE, AND LOCATION OF THE HEART :-**

The adult heart is shaped like a blunt cone and is approximately the size of a closed fist. It is larger in physically active adults than in other healthy adults, and it generally decreases in size after approximately age 65, especially in those who are not physically active. The blunt, rounded point of the cone is the apex ; the larger, flat part at the opposite end of the cone is the base. The heart is located in the thoracic cavity between the lungs. The heart, trachea, esophagus, and associated structures form a midline partition, the mediastinum. It is important for clinical reasons to know the location of the

heart in the thoracic cavity. Positioning a stethoscope to hear the heart sounds and positioning electrodes to record an electrocardiogram from chest leads depend on this knowledge. Effective cardiopulmonary resuscitation also depends on a reasonable knowledge of the position and shape of the heart. The heart lies obliquely in the mediastinum, with its base directed posteriorly and slightly superiorly and the apex directed anteriorly and slightly inferiorly. The apex is also directed to the left so that approximately two-thirds of the heart’s mass lies to the left of the midline of the sternum. The base of the heart is located deep to the sternum and extends to the second intercostal space. The apex is located deep to the fifth intercostal space, approximately 7–9 centimeters (cm) to the left of the sternum and medial to the midclavicular line, which is a perpendicular line that extends down from the middle of the clavicle.

**ANATOMY OF THE HEART :-**

**Pericardium** :- The pericardium, or pericardial sac , is a double-layered, closed sac that surrounds the heart. It consists of a tough, fibrous connective tissue outer layer called the fibrous pericardium and a thin, transparent inner layer of simple squamous epithelium called the serous pericardium. The fibrous pericardium prevents overdistention of the heart and anchors it within the mediastinum. Superiorly, the fibrous pericardium is continuous with the connective tissue coverings of the great vessels, and inferiorly it is attached to the surface of the diaphragm. The part of the serous pericardium lining the fibrous pericardium is the parietal pericardium, and the part covering the heart surface is the visceral pericardium, or epicardium. The parietal and visceral portions of the serous pericardium are continuous with each other where the great vessels enter or leave the heart. The pericardial cavity, between the visceral and parietal pericardia, is filled with a thin layer of serous pericardial fluid, which helps reduce friction as the heart moves within the pericardial sac. Even though the pericardium contains fibrous connective tissue, it can accommodate changes in heart size by gradually enlarging. The pericardial cavity can also increase in volume to hold a significant volume of pericardial fluid.

**Heart Wall** :- The heart wall is composed of three layers of tissue: the epicardium, myocardium, and endocardium. The epicardium, or visceral pericardium, is a thin serous membrane that constitutes the smooth outer surface of the heart. The serous pericardium is called the epicardium when considered a part of the heart and the visceral pericardium when considered a part of the pericardium. The thick middle layer of the heart, the myocardium, is composed of cardiac muscle cells and is responsible for the heart’s ability to contract. The smooth inner surface of the heart chambers is the endocardium, which consists of simple squamous epithelium over a layer of connective tissue. The smooth inner surface allows blood to move easily through the heart. The heart valves are modified folds of the endocardium that consist of a double layer of endocardium with connective tissue in between. The interior surfaces of the atria are mainly flat, but the interior of both auricles and a part of the right atrial wall contain muscular ridges called pectinate muscles. The pectinate muscles of the right atrium are separated from the larger, smooth portions of the atrial wall by a ridge called the crista terminalis. The interior walls of the ventricles contain larger, muscular ridges and columns called trabeculae carneae.

**External Anatomy and Coronary Circulation:-**

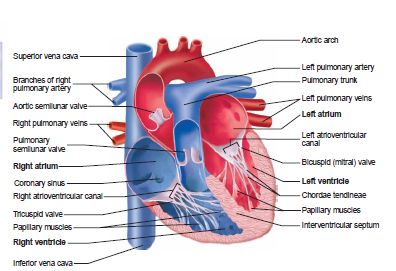
The heart consists of four chambers: two atria and two ventricles. The thin-walled atria form the superior and posterior parts of the heart, and the thick-walled ventricles form the anterior and inferior portions. Flap-like auricles are extensions of the atria that can be seen anteriorly between each atrium and ventricle. The entire atrium used to be called the auricle, and some medical personnel still refer to it as such. Several large veins carry blood to the heart. The superior vena cava and the inferior vena cava carry blood from the body to the right atrium, and four pulmonary veins carry blood from the lungs to the left atrium. In addition, the smaller coronary sinus carries blood from the walls of the heart to the right atrium. Two arteries, the aorta and the pulmonary trunk, exit the heart. The aorta carries blood from the left ventricle to the body, and the pulmonary trunk carries blood from the right ventricle to the lungs. A large coronary sulcus runs obliquely around the heart, separating the atria from the ventricles. Two more sulci extend inferiorly from the coronary sulcus, indicating the division between the right and left ventricles. The anterior interventricular sulcus, or groove, is on the anterior surface of the heart, and the posterior interventricular sulcus, or groove, is on the posterior surface of the heart. In a healthy, intact heart, the sulci are covered by fat, and only after this fat is removed can the sulci be seen. The major arteries supplying blood to the tissue of the heart lie within the coronary sulcus and interventricular sulci on the surface of the heart. The right and left coronary arteries exit the aorta just above the point where the aorta leaves the heart and lie within the coronary sulcus. The right coronary artery is usually smaller in diameter than the left one, and it does not carry as much blood as the left coronary artery. A major branch of the left coronary artery, called the anterior interventricular artery, or the left anterior descending artery, extends inferiorly in the anterior interventricular sulcus and supplies blood to most of the anterior part of the heart. The left marginal artery branches from the left coronary artery to supply blood to the lateral wall of the left ventricle. The circumflex artery branches from the left coronary artery and extends around to the posterior side of the heart in the coronary sulcus. Its branches supply blood to much of the posterior wall of the heart. The right coronary artery lies within the coronary sulcus and extends from the aorta around to the posterior part of the heart. A larger branch of the right coronary artery, called the right marginal artery, and other branches supply blood to the lateral wall of the right ventricle. A branch of the right coronary artery, called the posterior interventricular artery, lies in the posterior interventricular sulcus and supplies blood to the posterior and inferior part of the heart. Most of the myocardium receives blood from more than one arterial branch. Furthermore, there are many anastamoses, or direct connections, between the arterial branches. The anastomoses are either between branches of a given artery or between branches of different arteries. In the event that one artery is blocked, the areas primarily supplied by that artery may still receive some blood through other arterial branches and anastamoses. Aerobic exercise tends to increase the density of blood vessels supplying blood to the myocardium and the number and extent of the anastomoses increase. Consequently, aerobic exercise increases the chance that a person will survive the blockage of a small coronary artery. The blockage of larger coronary blood vessels still has the potential to permanently damage large areas of the heart wall. The major vein draining the tissue on the left side of the heart is the great cardiac vein, and a small cardiac vein drains the right margin of the heart. These veins converge toward the posterior part of the coronary sulcus and empty into a large venous cavity called the coronary sinus , which in turn empties into the right atrium. A number of smaller veins empty into the cardiac veins, into the coronary sinus, or directly into the right atrium. Blood flow through the coronary blood vessels is not continuous. When the cardiac muscle contracts, blood vessels in the wall of the heart are compressed and blood does not readily flow through them. When the cardiac muscle is relaxing, the blood vessels are not compressed and blood flow through the coronary blood vessels resumes. In a resting person, blood flowing through the coronary arteries of the heart gives up approximately 70% of its oxygen. In comparison, blood flowing through arteries to skeletal muscle gives up only about 25% of its oxygen. The percentage of oxygen the blood releases to skeletal muscle can increase to 70% or more during exercise. Because the percentage of oxygen the blood releases to cardiac muscle is near its maximum at rest, it cannot increase substantially during exercise. Cardiac muscle is therefore very dependent on an increased rate of blood flow through the coronary arteries above its resting level to provide an adequate oxygen supply during exercise.

**Heart Chambers and Valves:**-

**Right and Left Atria:-** The right atrium has three major openings: The openings from the superior vena cava and the inferior vena cava receive blood from the body, and the opening of the coronary sinus receives blood from the heart itself. The left atrium has four relatively uniform openings that receive blood from the four pulmonary veins from the lungs. The two atria are separated from each other by the interatrial septum. A slight, oval depression, the fossa ovalis, on the right side of the septum marks the former location of the foramen ovale, an opening between the right and left atria in the embryo and the fetus. This opening allows blood to flow from the right to the left atrium in the fetus to bypass the pulmonary circulation.

**Right and Left Ventricles :-** The atria open into the ventricles through atrioventricular canals. Each ventricle has one large, superiorly placed outflow route near the midline of the heart. The right ventricle opens into the pulmonary trunk, and the left ventricle opens into the aorta. The two ventricles are separated from each other by the interventricular septum, which has a thick, muscular part toward the apex and a thin, membranous part toward the atria.

**Atrioventricular Valves :-** An atrioventricular valve is in each atrioventricular canal and is composed of cusps, or flaps. These valves allow blood to flow from the atria into the ventricles but prevent blood from flowing back into the atria. The atrioventricular valve between the right atrium and the right ventricle has three cusps and is therefore called the tricuspid valve . The atrioventricular valve between the left atrium and left ventricle has two cusps and is therefore called the bicuspid, or mitral valve. Each ventricle contains cone-shaped, muscular pillars called papillary muscles. These muscles are attached by thin, strong connective tissue strings called chordae tendineae to the cusps of the atrioventricular valves. The papillary muscles contract when the ventricles contract and prevent the valves from opening into the atria by pulling on the chordae tendineae attached to the valve cusps. Blood flowing from the atrium into the ventricle pushes the valve open into the ventricle, but, when the ventricle contracts, blood pushes the valve back toward the atrium. The atrioventricular canal is closed as the valve cusps meet.



**ROUTE OF BLOOD FLOW THROUGH THE HEART:-**

Even though it is more convenient to discuss blood flow through the heart one side at a time, it is important to understand that both atria contract at about the same time and both ventricles contract at about the same time. This concept is particularly important when considering electrical activity, pressure changes, and heart sounds. Blood enters the right atrium from the systemic circulation, which returns blood from all the tissues of the body. Blood flows from an area of higher pressure in the systemic circulation to the right atrium, which has a lower pressure. Most of the blood in the right atrium then passes into the right ventricle as the ventricle relaxes following the previous contraction. The right atrium then contracts, and most of the blood remaining in the atrium is pushed into the ventricle to complete right ventricular filling. Contraction of the right ventricle pushes blood against the tricuspid valve, forcing it closed, and against the pulmonary semilunar valve, forcing it open, thus allowing blood to enter the pulmonary trunk. The pulmonary trunk branches to form the pulmonary arteries, which carry blood to the lungs, where carbon dioxide is released and oxygen is picked up. Blood returning from the lungs enters the left atrium through the four pulmonary veins. The blood passing from the left atrium to the left ventricle opens the bicuspid valve, and contraction of the left atrium completes left ventricular filling. Contraction of the left ventricle pushes blood against the bicuspid valve, closing it, and against the aortic semilunar valve, opening it and allowing blood to enter the aorta. Blood flowing through the aorta is distributed to all parts of the body, except to the parts of the lungs supplied by the pulmonary blood vessels.

**HISTOLOGY :-**

**Heart Skeleton :-** The heart skeleton consists of a plate of fibrous connective tissuebetween the atria and ventricles. This connective tissue plate formsfibrous rings around the atrioventricular and semilunar valvesand provides a solid support for them. The fibrousconnective tissue plate also serves as electrical insulation betweenthe atria and the ventricles and provides a rigid site for attachmentof the cardiac muscles.

**Cardiac Muscle:-** Cardiac muscle cells are elongated, branching cells that containone, or occasionally two, centrally located nuclei. Cardiac musclecells contain actin and myosin myofilaments organized toform sarcomeres, which join end-to-end to form myofibrils. The actin and myosin myofilaments are responsiblefor muscle contraction, and their organization gives cardiacmuscle a striated (banded) appearance. The striations are less regularly arranged and less numerous than in skeletal muscle.Cardiac muscle has a smooth sarcoplasmic reticulum, but it isnot as regularly arranged as it is in skeletal muscle fibers, and thereare no dilated cisternae, as in skeletal muscle. The sarcoplasmicreticulum comes into close association at various points with membranes of transverse (T) tubules . The T tubules of cardiacmuscle are found near the Z disks of the sarcomeres instead of wherethe actin and myosin overlap as in skeletal muscle. The T tubules incardiac muscle are larger in diameter than in skeletal muscle, andthere are extensions of T tubules that are parallel with the sarcoplasmicreticulum. The loose association between the sarcoplasmicreticulum and the T tubules is partly responsible for the slow onsetof contraction and the prolonged contraction phase in cardiac muscle.Depolarizations of the cardiac muscle plasma membrane are notcarried from the surface of the cell to the sarcoplasmic reticulumas efficiently as they are in skeletal muscles, and calcium must diffusea greater distance from the sarcoplasmic reticulum to the actinmyofilaments. In addition, some Ca 2 enter the cardiac muscle cellsfrom the extracellular fluid and from the T tubules.Adenosine triphosphate (ATP) provides the energy for cardiacmuscle contraction, and, as in other tissues, ATP productiondepends on oxygen availability. Cardiac muscle, however, cannotdevelop a large oxygen debt, a characteristic that is consistent withthe function of the heart. Development of a large oxygen debt wouldresult in muscular fatigue and cessation of cardiac muscle contraction.Cardiac muscle cells are rich in mitochondria, which performoxidative metabolism at a rate rapid enough to sustain normalmyocardial energy requirements. The extensive capillary networkprovides an adequate oxygen supply to the cardiac muscle cells. Cardiac muscle cells are organized in spiral bundles or sheets.The cells are bound end-to-end and laterally to adjacent cells byspecialized cell–cell contacts called intercalateddisks. The membranes of the intercalated diskshave folds, and the adjacent cells fit together, thus greatly increasingcontact between them. Specialized plasma membrane structurescalled desmosomes hold the cells together,and gap junctions function as areas of low electric resistancebetween the cells, allowing action potentials to pass from one cellto adjacent cells. Electrically, the cardiac musclecells behave as a single unit, and the highly coordinated contractionsof the heart depend on this functional characteristic.

**Conducting System :-**  The conducting system of the heart, which relays action potentials through the heart, consists of modified cardiac muscle cells that form two nodes (knots or lumps) and a conducting bundle. The two nodes are contained within the walls of the right atrium and are named according to their position in the atrium. The sinoatrial (SA) node is medial to the opening of the superior vena cava, and the atrioventricular (AV) node is medial to the right atrioventricular valve. The AV node gives rise to a conducting bundle of the heart, the atrioventricular (AV) bundle (bundle of His). This bundle passes through a small opening in the fibrous skeleton to reach the interventricular septum, where it divides to form the right and left bundle branches, which extend beneath the endocardium on each side of the interventricular septum to the apices of the right and left ventricles, respectively. The inferior terminal branches of the bundle branches are called Purkinje fibers, which are large-diameter cardiac muscle fibers. They have fewer myofibrils than most cardiac muscle cells and do not contract as forcefully. Intercalated disks are well developed between the Purkinje fibers and contain numerous gap junctions. As a result of these structural modifications, action potentials travel along the Purkinje fibers much more rapidly than through other cardiac muscle tissue. Cardiac muscle cells have the capacity to generate spontaneous action potentials, but cells of the SA node do so at a greater frequency. As a result, the SA node is called the pacemaker of the heart. The SA node is made up of specialized, small-diameter cardiac muscle cells that merge with the other cardiac muscle cells of the right atrium. Thus, the heart contracts spontaneously and rhythmically. Once action potentials are produced, they spread from the SA node to adjacent cardiac muscle fibers of the atrium. Preferential pathways conduct action potentials from the SA node to the AV node at a greater velocity than they are transmitted in the remainder of the atrial muscle fibers, although such pathways cannot be distinguished structurally from the remainder of the atrium. When the heart beats under resting conditions, approximately 0.04 second is required for action potentials to travel from the SA node to the AV node. Within the AV node, action potentials are propagated slowly, compared with the remainder of the conducting system. The slow rate of action potential conduction in the AV node is due, in part, to the smaller-diameter muscle fibers and fewer gap junctions in their intercalated disks. Like the other specialized conducting fibers in the heart, they have fewer myofibrils than most cardiac muscle cells. As a consequence, a delay occurs of 0.11 second from the time action potentials reach the AV node until they pass to the AV bundle. The total delay of 0.15 second allows completion of the atrial contraction before ventricular contraction begins. After action potentials pass from the AV node to the highly specialized conducting bundles, the velocity of conduction increases dramatically. The action potentials pass through the left and right bundle branches and through the individual Purkinje fibers that penetrate into the myocardium of the ventricles. Because of the arrangement of the conducting system, the first part of the myocardium that is stimulated is the inner wall of the ventricles near the apex. Thus, ventricular contraction begins at the apex and progresses throughout the ventricles. The spiral arrangement of muscle layers in the wall of the heart results in a wringing action, which proceeds from the apex toward the base of the heart as contraction proceeds. During the process, the distance between the apex and the base of the heart decreases.

**ELECTRICAL PROPERTIES :-** Cardiac muscle cells—like other electrically excitable cells, suchas neurons and skeletal muscle fibers—have a resting membranepotential (RMP). The RMP depends on a low permeability of theplasma membrane to Na and Ca 2 and a higher permeability toK . When neurons, skeletal muscle cells, and cardiac muscle cellsare depolarized to their threshold level, action potentials result.

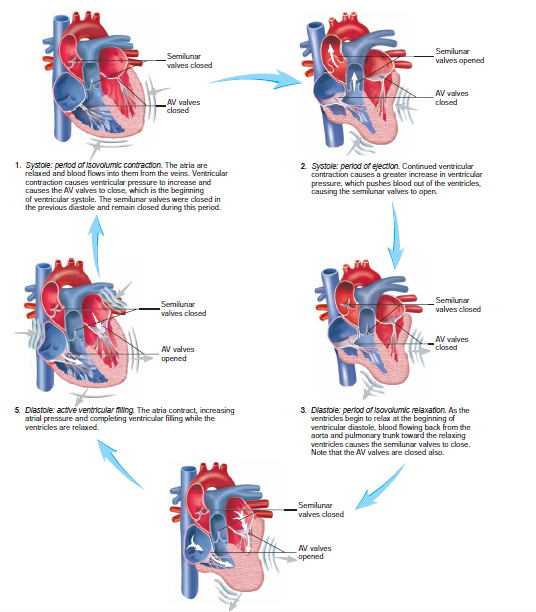
**Action Potentials:-** Like action potentials in skeletal muscle, those in cardiac muscleexhibit depolarization followed by repolarization of the RMP.Alterations in membrane channels are responsible for the changesin the permeability of the plasma membrane that produce theaction potentials. Action potentials in cardiac muscle last longerthan those in skeletal muscle, and the membrane channels differsomewhat from those in skeletal muscle. In contrast to action potentialsin skeletal muscle, which take less than 2 milliseconds (ms) tocomplete, action potentials in cardiac muscle take approximately200–500 ms to complete.In cardiac muscle, the action potential consists of a rapiddepolarization phase, followed by a rapid but partial early repolarizationphase. Then a prolonged period of slow repolarizationoccurs, called the plateau phase. At the end of the plateau phase,a more rapid final repolarization phase takes place, duringwhich the membrane potential returns to its resting level .Depolarization is the result of changes in membrane permeabilityto Na , K , and Ca 2. Membrane channels, called voltage gatedNa channels, open, bringing about the depolarizationphase of the action potential. As the voltage-gated Na channelsopen, Na diffuse into the cell, causing rapid depolarization untilthe cell is depolarized to approximately 20 millivolts (mV).The voltage change occurring during depolarization affectsother ion channels in the plasma membrane. Several types ofvoltage-gated K channels exist, each of which opens and closesat different membrane potentials, causing changes in membranepermeability to K. For example, at rest, the movement of Kthrough open voltage-gated K channels is primarily responsiblefor establishing the resting membrane potential in cardiacmuscle cells. Depolarization causes these voltage-gated K channelsto close, thereby decreasing membrane permeability to K.Depolarization also causes voltage-gated Ca 2 channels to begin toopen. These changes contribute to depolarization. Compared withsodium channels, the calcium channels open and close slowly.Repolarization is also the result of changes in membrane permeabilityto Na, K, and Ca 2. Early repolarization occurs whenthe voltage-gated Na channels and some voltage-gated Ca 2channels close, and a small number of voltage-gated K channelsopen. Sodium ion movement into the cell slows, and some Kmove out of the cell. The plateau phase occurs as voltage-gatedCa 2 channels remain open, and the movement of Ca 2 and someNa through the voltage-gated Ca 2 channels into the cell counteractsthe potential change produced by the movement of K outof the cell. The plateau phase ends and final repolarization beginsas the voltage-gated Ca 2 channels close and many more voltage gatedK channels open. Thus, Ca 2 and Na stop diffusing intothe cell, and the tendency for K to diffuse out of the cell increases.These permeability changes cause the membrane potential toreturn to its resting level.Action potentials in cardiac muscle are conducted from cellto cell, whereas action potentials in skeletal muscle fibers are conductedalong the length of a single muscle fiber, but not from fiberto fiber. Also, the rate of action potential propagation is slower in cardiac muscle than in skeletal muscle because cardiac musclecells are smaller in diameter and much shorter than skeletal musclefibers. Although the gap junctions of intercalated disks allow thetransfer of action potentials between cardiac muscle cells, they doslow the rate of action potential conduction between the cardiacmuscle cells.The movement of Ca 2 through the plasma membrane,including the membranes of the T tubules, into cardiac musclecells stimulates the release of Ca 2 from the sarcoplasmic reticulum,a process called calcium-induced calcium release (CICR).When an action potential occurs in a cardiac muscle cell, Ca 2 enter the cell and bind to receptors in the membranes of sarcoplasmicreticulum, resulting in the opening of Ca 2 channels. Calciumions then move out of the sarcoplasmic reticulum and activate theinteraction between actin and myosin to produce contraction ofthe cardiac muscle cells.

**Autorhythmicity of Cardiac Muscle :-** The heart is said to be auto-rhythmic because itstimulates itself ( auto ) to contract at regular intervals ( rhythmic ).If the heart is removed from the body and maintained underphysiologic conditions with the proper nutrients and temperature,it will continue to beat autorhythmically for a long time.In the SA node, pacemaker cells generate action potentials spontaneouslyand at regular intervals. These action potentials spreadthrough the conducting system of the heart to other cardiac musclecells, causing voltage-gated Na channels to open. As a result, actionpotentials are produced and the cardiac muscle cells contract.The generation of action potentials in the SA node resultswhen a spontaneously developing local potential, called the pre-potential,reaches threshold. Changes in ion movementinto and out of the pacemaker cells cause the pre-potential. Sodium ions cause depolarization by moving into the cells through specializednon-gated Na channels. A decreasing permeability to Kalso causes depolarization as fewer K move out of the cells. Thedecreasing K permeability occurs due to the voltage changes at theend of the previous action potential. As a result of the depolarization,voltage-gated Ca 2 channels open, and the movement of Ca 2into the pacemaker cells causes further depolarization. When thepre-potential reaches threshold, many voltage-gated Ca 2 channelsopen. Unlike other cardiac muscle cells, the movement of Ca 2 intothe pacemaker cells is primarily responsible for the depolarizationphase of the action potential. Repolarization occurs, as in other cardiacmuscle cells, when the voltage-gated Ca 2 channels close andthe voltage-gated K channels open. After the RMP is reestablished,production of another pre-potential starts the generation of the nextaction potential. Although most cardiac muscle cells respond to action potentialsproduced by the SA node, some cardiac muscle cells in theconducting system can also generate spontaneous action potentials.Normally, the SA node controls the rhythm of the heartbecause its pacemaker cells generate action potentials at a fasterrate than other potential pacemaker cells to produce a heart rateof 70–80 beats per minute (bpm). An ectopic focus is any part of the heart other than the SAnode that generates a heartbeat. For example, if the SA node doesnot function properly, the part of the heart to produce actionpotentials at the next highest frequency is the AV node, whichproduces a heart rate of 40–60 bpm. Another cause of an ectopicfocus is blockage of the conducting pathways between the SA nodeand other parts of the heart. For example, if action potentials donot pass through the AV node, an ectopic focus can develop in anAV bundle, resulting in a heart rate of 30 bpm.Ectopic foci can also appear when the rate of action potentialgeneration in cardiac muscle cells outside of the SA node becomesenhanced. For example, when cells are injured their plasmamembranes become more permeable, resulting in depolarization.Inflammation or lack of adequate blood flow to cardiac muscletissue can injure cardiac muscle cells. These injured cells can bethe source of ectopic action potentials. Also, alterations in bloodlevels of K and Ca 2 can change the cardiac muscle membranepotential, and certain drugs, such as those that mimic the effect ofepinephrine on the heart, can alter cardiac muscle membrane permeability.Changes in cardiac muscle cells’ membrane potentials orpermeability can produce ectopic foci.

**Refractory Periods of Cardiac Muscle :-** Cardiac muscle, like skeletal muscle, has refractoryperiods associated with its action potentials. During the absoluterefractory period, the cardiac muscle cell is completelyinsensitive to further stimulation. During the relative refractoryperiod, the cell is sensitive to stimulation, but a greaterstimulation than normal is required to cause an action potential.Because the plateau phase of the action potential in cardiacmuscle delays repolarization to the RMP, the refractory periodis prolonged. The long refractory period ensures that contractionand most of relaxation are complete before another actionpotential can be initiated. This prevents tetanic contractionsfrom occurring in cardiac muscle and is responsible for rhythmiccontractions.

**Electrocardiogram :-** The conduction of action potentials through the myocardiumduring the cardiac cycle produces electric currents that can bemeasured at the surface of the body. Electrodes placed on thesurface of the body and attached to an appropriate recordingdevice can detect small voltage changes resulting fromaction potentials in the cardiac muscle. The electrodes detecta summation of all the action potentials that are transmittedby the cardiac muscle cells through the heart at a given time.Electrodes do not detect individual action potentials. The summatedrecord of the cardiac action potentials is an electrocardiogram(ECG or EKG).The ECG is not a direct measurement of mechanical eventsin the heart, and neither the force of contraction nor bloodpressure can be determined from it. Each deflection in the ECGrecord, however, indicates an electrical event within the heart thatis correlated with a subsequent mechanical event. Consequently,it is an extremely valuable diagnostic tool in identifying a numberof abnormal cardiac rhythms andother abnormalities, particularly because it is painless, easy torecord, and noninvasive (it does not require surgical procedures).Abnormal heart rates or rhythms, abnormal conduction pathways,hypertrophy or atrophy of portions of the heart, and the approximatelocation of damaged cardiac muscle can be determined fromanalysis of an ECG.The normal ECG consists of a P wave, a QRS complex, anda T wave. The P wave, which is the result of actionpotentials that cause depolarization of the atrial myocardium, signalsthe onset of atrial contraction. The QRS complex is composedof three individual waves: the Q, R, and S waves. The QRS complexresults from ventricular depolarization and signals the onset ofventricular contraction. The T wave represents repolarization ofthe ventricles and precedes ventricular relaxation. A wave representingrepolarization of the atria cannot be seen because it occursduring the QRS complex. The time between the beginning of the P wave and thebeginning of the QRS complex is the PQ interval, commonlycalled the PR interval because the Q wave is often very small.During the PR interval, which lasts approximately 0.16 second,the atria contract and begin to relax. The ventricles begin todepolarize at the end of the PR interval. The QT interval extendsfrom the beginning of the QRS complex to the end of the T wave,lasts approximately 0.36 second, and represents the approximatelength of time required for the ventricles to contract and beginto relax.

**CARDIAC CYCLE :-** The heart is actually two separate pumps that work together, one inthe right half and the other in the left half of the heart. Each pumpconsists of a primer pump—the atrium—and a power pump—theventricle. Both atrial primer pumps complete the filling of theventricles with blood, and both ventricular power pumps producethe major force that causes blood to flow through the pulmonaryand systemic arteries. The term cardiac cycle refers to the repetitivepumping process that begins with the onset of cardiac muscle contraction and ends with the beginning of the next contraction. Pressure changes producedwithin the heart chambers as a result of cardiac muscle contractionare responsible for blood movement because blood moves fromareas of higher pressure to areas of lower pressure.The duration of the cardiac cycle varies considerably amonghumans and during an individual’s lifetime. It can be as short as0.25–0.3 second in a newborn or as long as 1 or more seconds ina well-trained athlete. The normal cardiac cycle of 0.7–0.8 seconddepends on the capability of cardiac muscle to contract and on thefunctional integrity of the conducting system.The term systole means to contract, and diastole means to dilate. Atrial systole is contraction of theatrial myocardium, and atrial diastole is relaxation of the atrialmyocardium. Similarly, ventricular systole is contraction of theventricular myocardium, and ventricular diastole is relaxation ofthe ventricular myocardium. When the terms systole and diastoleare used without reference to specific chambers, however, theymean ventricular systole or diastole.Just before systole begins, the atria and ventricles are relaxed,the ventricles are filled with blood, the semilunar valves are closed,and the AV valves are open. As systole begins, contraction of theventricles increases ventricular pressures, causing blood to flowtoward the atria and close the AV valves. As contraction proceeds,ventricular pressures continue to rise, but no blood flows fromthe ventricles because all the valves are closed. This brief intervalis called the period of isovolumic contractionbecause the volume of blood in the ventricles does not change, even though the ventricles are contracting. As the ventricles continue to contract, ventricular pressuresbecome greater than the pressures in the pulmonary trunkand aorta. As a result, during the period of ejection, the semilunarvalves are pushed open and blood flows from the ventricles intothose arteries.As diastole begins, the ventricles relax and ventricular pressuresdecrease below the pressures in the pulmonary trunk and aorta. Consequently, blood begins to flow back toward the ventricles,causing the semilunar valves to close.With closure of the semilunar valves, all the heart valves are closedand no blood flows into the relaxing ventricles during the periodof isovolumic relaxation.Throughout ventricular systole and the period of isovolumicrelaxation, the atria relax and blood flows into them from the veins.As the ventricles continue to relax, ventricular pressures becomelower than atrial pressures, the AV valves open, and blood flows fromthe atria into the relaxed ventricles. At rest,most ventricular filling is a passive process resulting from the greaterpressure of blood in the veins and atria than in the completely relaxedventricles. Completion of ventricular filling is an active processresulting from increased atrial pressure produced by contraction ofthe atria. During exercise, atrial contractionis more important for ventricular filling because, as heart rateincreases, less time is available for passive ventricular filling.



CARDIAC CYCLE.

**Events Occurring During Ventricular Systole :-** An ECG indicates the electrical events that cause contraction andrelaxation of the atria and ventricles.The pressure graph shows the pressure changes within the left atrium, left ventricle, and aorta resulting from atrial and ventricularcontraction and relaxation. Although pressure changes in the rightside of the heart are not shown, they are similar to those in the leftside, only lower. The volume graph presents the changes in left ventricularvolume as blood flows into and out of the left ventricle as aresult of the pressure changes. The sound graph records the closing ofvalves caused by blood flow. See also figure 20.18 for illustrations ofthe valves and blood flow and table 20.2 for a summary of the eventsoccurring during each period.

Period of Isovolumic Contraction :- Completion of the QRS complex initiates contraction of the ventricles. Ventricular pressure rapidly increases, resulting in closure of the AV valves. During the previous ventricular diastole, the ventricles were filled with 120–130 mL of blood, which is called the end-diastolic volume. Ventricular volume does not change during the period of isovolumic contraction because all the heart valves are closed.

Period of Ejection :- As soon as ventricular pressures exceed the pressures in the aorta and pulmonary trunk, the semilunar valves open. The aortic semilunar valve opens at approximately 80 mm Hg ventricular pressure, whereas the pulmonary semilunar valve opens at approximately 8 mm Hg. Although the pressures are different, both valves open at nearly the same time. As blood flows from the ventricles during the period of ejection, the left ventricular pressure continues to climb to approximately 120 mm Hg, and the right ventricular pressure increases to approximately 25 mm Hg. The larger left ventricular pressure causes blood to flow throughout the body (systemic circulation), whereas the lower right ventricle pressure causes blood to flow through the lungs (pulmonary circuit). Even though the pressure generated by the left ventricle is much higher than that of the right ventricle, the amount of blood pumped by each is almost the same.

**Events Occurring During Ventricular Diastole :-**

Period of Isovolumic Relaxation :- Completion of the T wave results in ventricular repolarization and relaxation. The already decreasing ventricular pressure falls very rapidly as the ventricles suddenly relax. When the ventricular pressures fall below the pressures in the aorta and pulmonary trunk, the recoil of the elastic arterial walls, which were stretched during the period of ejection, forces the blood to flow back toward the ventricles, thereby closing the semilunar valves. Ventricular volume does not change during the period of isovolumic relaxation because all the heart valves are closed at this time.

Passive Ventricular Filling :- During ventricular systole and the period of isovolumic relaxation, the relaxed atria fill with blood. As ventricular pressure drops below atrial pressure, the atrioventricular valves open and allow blood to flow from the atria into the ventricles. Blood flows from the area of higher pressure in the veins and atria toward the area of lower pressure in the relaxed ventricles. Most ventricular filling occurs during the first one-third of ventricular diastole. At the end of passive ventricular filling, the ventricles are approximately 70% filled.

Active Ventricular Filling :- Depolarization of the SA node generates action potentials that spread over the atria, producing the P wave and stimulating both atria to contract (atrial systole). The atria contract during the last one-third of ventricular diastole and complete ventricular filling. Under most conditions, the atria function primarily as reservoirs, and the ventricles can pump sufficient blood to maintain homeostasis even if the atria do not contract at all. During exercise, however, the heart pumps 300%–400% more blood than during resting condition. It is under these conditions that the pumping action of the atria becomes important in maintaining the pumping efficiency of the heart.

**Heart Sounds :-** Distinct sounds are heard when a stethoscope is used to listen tothe heart. The first heart sound is alow-pitched sound, often described as a “lubb” sound. It is caused byvibration of the atrioventricular valves and surrounding fluid as thevalves close at the beginning of ventricular systole. The second heartsound is a higher-pitched sound often described as a “dupp” sound.It results from closure of the aortic and pulmonary semilunar valves,at the beginning of ventricular diastole. Systole is, therefore, approximatelythe time between the first and second heart sounds. Diastole,which lasts somewhat longer, is approximately the time between thesecond heart sound and the next first heart sound.Occasionally, a faint third heart sound can be heard in somenormal people, particularly in those who are thin and young. It is caused by blood flowing in a turbulent fashion into the ventricles,and it can be detected near the end of the first one-third of diastole.

**Aortic Pressure Curve :-** The elastic walls of the aorta are stretched as blood is ejected intothe aorta from the left ventricle. Aortic pressure remains slightlybelow ventricular pressure during this period of ejection. Asventricular pressure drops below that in the aorta, blood flowsback toward the ventricle because of the elastic recoil of the aorta.Consequently, the aortic semilunar valve closes, and pressure withinthe aorta increases slightly, producing a dicrotic notchin the aortic pressure curve. The term dicroticmeans double-beating; when increased pressure caused by recoil islarge, a double pulse can be felt. The dicrotic notch is also called anincisura. Aortic pressure then graduallyfalls throughout the rest of ventricular diastole as blood flowsthrough the peripheral vessels. When aortic pressure has fallen toapproximately 80 mm Hg, the ventricles again contract, forcingblood once more into the aorta.Blood pressure measurements performed for clinical purposesreflect the pressure changes that occur in the aorta rather than inthe left ventricle. The blood pressure in the aortafluctuates between systolic pressure, which is about 120 mm Hg,and diastolic pressure, which is about 80 mm Hg for the averageyoung adult at rest.

**MEAN ARTERIAL BLOOD PRESSURE :-** Blood pressure is necessary for blood movement and, therefore,is critical to the maintenance of homeostasis. Blood flows fromareas of higher to areas of lower pressure. For example, during onecardiac cycle, blood flows from the higher pressure in the aorta,resulting from contraction of the left ventricle, toward the lowerpressure in the relaxed right atrium.Mean arterial pressure (MAP) is slightly less than the averageof the systolic and diastolic pressure in the aorta. It is proportionalto cardiac output (CO) times peripheral resistance (PR). Cardiacoutput, or minute volume, is the amount of blood pumped by theheart per minute, and peripheral resistance is the total resistanceagainst which blood must be pumped.MAP CO PRChanges in cardiac output and peripheral resistance can alter mean arterial pressure.Cardiac output is equal to heart rate times stroke volume.Heart rate (HR) is the number of times the heart beats (contracts) per minute. Stroke volume (SV) , which is the volume of bloodpumped during each heartbeat (cardiac cycle), is equal to end diastolicvolume minus end-systolic volume. During diastole,blood flows from the atria into the ventricles, and end-diastolicvolume normally increases to approximately 125 mL. After the ventriclespartially empty during systole, end-systolic volume decreasesto approximately 55 mL. The stroke volume is therefore equal to70 mL (125 55).To better understand stroke volume, imagine that you arerinsing out a sponge under a running water faucet. As you relaxyour hand, the sponge fills with water; as your fingers contract,water is squeezed out of the sponge; and, after you have squeezedit, some water is left in the sponge. In this analogy, the amount ofwater you squeeze out of the sponge (stroke volume) is the differencebetween the amount of water in the sponge when your handis relaxed (end-diastolic volume) and the amount that is left in thesponge after you squeeze it (end-systolic volume).Stroke volume can be increased by increasing end-diastolicvolume or by decreasing end-systolic volume. During exercise, end-diastolic volume increases because of anincrease in venous return , which is the amount of blood returningto the heart from the peripheral circulation. End-systolic volumedecreases because the heart contracts more forcefully. For example,stroke volume could increase from a resting value of 70 mL to anexercising value of 115 mL by increasing end-diastolic volume to145 mL and decreasing end-systolic volume to 30 mL.Under resting conditions, the heart rate is approximately72 bpm, and the stroke volume is approximately 70 mL/beat,although these values can vary considerably from person to person.The cardiac output is thereforeCO HR SV 72 bpm 70 mL/beat5040 mL/min (approximately 5 L/min)**.** During exercise, heart rate can increase to 190 bpm, and thestroke volume can increase to 115 mL. Consequently, cardiac outputisCO 190 bpm 115 mL/beat21,850 mL/min (approximately 22 L/min)**.** The difference between cardiac output when a person is atrest and maximum cardiac output is called cardiac reserve . Thegreater a person’s cardiac reserve, the greater his or her capacityfor doing exercise. Lack of exercise and cardiovascular diseasescan reduce cardiac reserve and affect a person’s quality of life.Exercise training can greatly increase cardiac reserve by increasing cardiac output. In well-trained athletes, stroke volume duringexercise can increase to over 200 mL/beat, resulting in cardiac outputsof 40 L/min or more.

**REGULATION OF THE HEART :-** To maintain homeostasis, the amount of blood pumped by theheart must vary dramatically. For example, during exercise cardiacoutput can increase several times over resting values. Intrinsic andextrinsic regulatory mechanisms control cardiac output. Intrinsicregulation results from the normal functional characteristicsof the heart and does not depend on either neural or hormonalregulation. It functions when the heart is in place in the body oris removed and maintained outside the body under proper conditions.On the other hand, extrinsic regulation involves neuraland hormonal control. Neural regulation of the heart results fromsympathetic and parasympathetic reflexes, and the major hormonalregulation comes from epinephrine and norepinephrinesecreted from the adrenal medulla.

**Intrinsic Regulation :-** As venous return increases, end-diastolic volume increases. A greater end-diastolic volume increases the stretch of the ventricular walls. The extent to which the ventricular walls are stretched is sometimes called the preload. An increased preload causes an increase in cardiac output, and a decreased preload causes a decrease in cardiac output. Cardiac muscle exhibits a length-versus-tension relationship similar to that of skeletal muscle. Skeletal muscle, however, is normally stretched to nearly its optimal length before contraction, whereas cardiac muscle fibers are not stretched to the point at which they contract with a maximal force. An increased preload, therefore, causes the cardiac muscle fibers to contract with a greater force and produce a greater stroke volume. This relationship between preload and stroke volume is commonly referred to as Starling’s law of the heart , which describes the relationship between changes in the pumping effectiveness of the heart and changes in preload. Venous return can decrease to a value as low as 2 L/min or increase to as much as 24 L/min, which has a major effect on the preload. Afterload is the pressure the contracting ventricles must produce to overcome the pressure in the aorta and move blood into the aorta. Although the pumping effectiveness of the heart is greatly influenced by relatively small changes in the preload, it is very insensitive to large changes in afterload. Aortic blood pressure must increase to more than 170 mm Hg before it hampers the ventricles’ ability to pump blood. During physical exercise, blood vessels in exercising skeletal muscles dilate and allow an increased flow of blood through the vessels. The increased blood flow increases oxygen and nutrient delivery to the exercising muscles. In addition, skeletal muscle contractions repeatedly compress veins and cause an increased rate of blood flow from the skeletal muscles toward the heart. As blood rapidly flows through skeletal muscles and back to the heart, venous return to the heart increases, resulting in an increased preload. The increased preload causes an increased force of cardiac muscle contraction, which increases stroke volume. The increase in stroke volume results in increased cardiac output, and the volume of blood flowing to the exercising muscles increases. When a person rests, venous return to the heart decreases because arteries in the skeletal muscles constrict and because muscular contractions no longer repeatedly compress the veins. As a result, blood flow through skeletal muscles decreases, and preload and cardiac output decrease.

**Extrinsic Regulation :-** The heart is innervated by both parasympathetic and sympathetic nerve fibers. They influence the pumping action of the heart by affecting both heart rate and stroke volume. The influence of parasympathetic stimulation on the heart is much less than that of sympathetic stimulation. Sympathetic stimulation can increase cardiac output by 50%–100% over resting values, whereas parasympathetic stimulation can cause only a 10%–20% decrease. Extrinsic regulation of the heart keeps blood pressure, blood oxygen levels, blood carbon dioxide levels, and blood pH within their normal ranges of values. For example, if blood pressure suddenly decreases, extrinsic mechanisms detect the decrease and initiate responses that increase cardiac output to bring blood pressure back to its normal range.

**Parasympathetic Control :-** Parasympathetic nerve fibers are carried to the heart through thevagus nerves. Preganglionic fibers of the vagus nerve extend from **t**he brainstem to terminal ganglia within the wall of the heart, andpostganglionic fibers extend from the ganglia to the SA node, AVnode, coronary vessels, and atrial myocardium.Parasympathetic stimulation has an inhibitory influence onthe heart, primarily by decreasing the heart rate. When a personis at rest, continuous parasympathetic stimulation inhibits theheart to some degree. An increase in heart rate during exerciseresults, in part, from decreased parasympathetic stimulation.Strong parasympathetic stimulation can decrease the heart ratebelow resting levels by at least 20–30 bpm, but it has little effect onstroke volume. In fact, if venous return remains constant while the heart is inhibited by parasympathetic stimulation, stroke volumeactually can increase. The longer time between heartbeats allowsthe heart to fill to a greater capacity, resulting in an increasedpreload, which increases stroke volume because of Starling’s lawof the heart. Acetylcholine , the neurotransmitter produced by postganglionicparasympathetic neurons, binds to ligand-gated channelsthat cause cardiac plasma membranes to become more permeableto K. As a consequence, the membrane hyperpolarizes. Heart ratedecreases because the hyperpolarized membrane takes longer todepolarize and cause an action potential.

**Sympathetic Control:-** Sympathetic nerve fibers originate in the thoracic region of thespinal cord as preganglionic neurons. These neurons synapse withpostganglionic neurons of the inferior cervical and upper thoracicsympathetic chain ganglia, which project to the heart as cardiacnerves. The postganglionic sympatheticnerve fibers innervate the SA and AV nodes, the coronaryvessels, and the atrial and ventricular myocardium.Sympathetic stimulation increases both the heart rateand the force of muscular contraction. In response to strongsympathetic stimulation, the heart rate can increase to 250 or,occasionally, 300 bpm. Stronger contractions also can increasestroke volume. The increased force of contraction resulting fromsympathetic stimulation causes a lower end-systolic volume inthe heart; therefore, the heart empties to a greater extent. Limitations exist, however, to the relationship betweenincreased heart rate and cardiac output. If the heart rate becomestoo fast, ventricular diastole is not long enough to allow completeventricular filling, end-diastolic volume decreases, and stroke volumeactually decreases. In addition, if heart rate increases beyond a critical level, the strength of contraction decreases, probablyas a result of the accumulation of metabolites in cardiac musclecells. The limit of the heart’s ability to increase the volume ofblood pumped is 170–250 bpm in response to intense sympatheticstimulation.Sympathetic stimulation of the ventricular myocardium playsa significant role in regulation of its contraction force when a personis at rest. Sympathetic stimulation maintains the strength ofventricular contraction at a level approximately 20% greater thanit would be with no sympathetic stimulation.Norepinephrine, the postganglionic sympathetic neurotransmitter,increases the rate and degree of cardiac muscledepolarization so that both the frequency and the amplitude ofthe action potentials are increased. The effect of norepinephrineon the heart involves the association between norepinephrineand cell surface -adrenergic receptors. This combination causesa G protein–mediated synthesis and accumulation of c AMP inthe cytoplasm of cardiac muscle cells. Cyclic AMP increases thepermeability of the plasma membrane to Ca 2 , primarily byopening calcium channels in the plasma membrane.Increased sympathetic stimulation causes coronary arteriesto constrict, to some degree. However, increased metabolism ofcardiac muscle, in response to sympathetic stimulation, resultsin the accumulation of metabolic by-products in cardiac musclethat cause dilation of coronary blood vessels. The dilation effect ofthese metabolites predominates.

**Hormonal Control:-** Epinephrine and norepinephrine released from the adrenal medullacan markedly influence the pumping effectiveness of the heart.Epinephrine has essentially the same effect on cardiac muscle asnorepinephrine and, therefore, increases the rate and force of heartcontractions.The secretion of epinephrine and norepinephrine from theadrenal medulla is controlled by sympathetic stimulation ofthe adrenal medulla and occurs in response to increased physicalactivity, emotional excitement, or stressful conditions. Manystimuli that increase sympathetic stimulation of the heart alsoincrease release of epinephrine and norepinephrine from theadrenal gland. Epinephrine and norepinephrineare transported in the blood through the vessels of the heart to thecardiac muscle cells, where they bind to -adrenergic receptorsand stimulate cAMP synthesis. Epinephrine takes a longer time toact on the heart than sympathetic stimulation does, but the effectlasts longer.

**HEART AND HOMEOSTASIS :-** The pumping efficiency of the heart plays an important role in themaintenance of homeostasis. Blood pressure in the systemic vesselsmust be maintained at a level that is high enough to achievenutrient and waste product exchange across the walls of the capillariesthat meets metabolic demands. The heart’s activity must beregulated because the metabolic activities of the tissues changeunder such conditions as exercise and rest.

**Effect of Blood Pressure :-** Baroreceptor reflexes detectchanges in blood pressure and result in changes in heart rate andin the force of contraction. The sensory receptors of the baroreceptorreflexes are stretch receptors. They are in the walls ofcertain large arteries, such as the internal carotid arteries and theaorta, and they measure blood pressure. Afferent neurons project primarily through the glossopharyngeal (cranial nerve IX) and vagus (cranial nerve X) nerves from the baroreceptors to an area in the medulla oblongata called the cardioregulatory center, where sensory action potentials are integrated. The part of the cardioregulatory center that increases heart rate is called the cardioacceleratory center, and the part that decreases heart rate is called the cardioinhibitory center. Efferent action potentials then are sent from the cardioregulatory center to the heart through both the sympathetic and the parasympathetic divisions of the autonomic nervous system. Increased blood pressure within the internal carotid arteries and aorta causes their walls to stretch, thereby stimulating an increase in action potential frequency in the baroreceptors. At normal blood pressures (80–120 mm Hg), afferent action potentials are sent from the baroreceptors to the medulla oblongata at a relatively constant frequency. When blood pressure increases, the arterial walls are stretched farther, and the afferent action potential frequency increases. When blood pressure decreases, the arterial walls are stretched to a lesser extent, and the afferent action potential frequency decreases. In response to increased blood pressure, the baroreceptor reflexes decrease sympathetic stimulation and increase parasympathetic stimulation of the heart, causing the heart rate to decrease. Decreased blood pressure causes decreased parasympathetic and increased sympathetic stimulation of the heart, resulting in an increased heart rate and force of contraction. Withdrawal of parasympathetic stimulation is primarily responsible for increases in heart rate up to approximately 100 bpm. Larger increases in heart rate, especially during exercise, result from sympathetic stimulation. The baroreceptor reflexes are homeostatic because they keep the blood pressure within a narrow range of values, which is adequate to maintain blood flow to the tissues.

Effect of pH, Carbon Dioxide, and Oxygen Chemoreceptor reflexes help regulate the heart’s activity. Chemoreceptors sensitive to changes in pH and carbon dioxide levels exist within the medulla oblongata. A drop in pH and a rise in carbon dioxide decrease parasympathetic and increase sympathetic stimulation of the heart, resulting in an increased heart rate and force of contraction. The increased cardiac output causes greater blood flow through the lungs, where carbon dioxide is eliminated from the body. This helps bring the blood carbon dioxide level down to its normal range of values and helps increase blood pH. Chemoreceptors primarily sensitive to blood oxygen levels are found in the carotid and aortic bodies. These small structures are located near large arteries close to the brain and heart, and they monitor blood flowing to the brain and to the rest of the body. A dramatic decrease in blood oxygen levels, such as during asphyxiation, activates the carotid and aortic body chemoreceptor reflexes. In carefully controlled experiments, it is possible to isolate the effects of the carotid and aortic body chemoreceptor reflexes from other reflexes, such as the medullary chemoreceptor reflexes. These experiments indicate that a decrease in blood oxygen results in a decrease in heart rate and an increase in vasoconstriction. The increased vasoconstriction causes blood pressure to rise, which promotes blood delivery despite the decrease in heart rate. The carotid and aortic body chemoreceptor reflexes may protect the heart for a short time by slowing the heart, thereby reducing its need for oxygen. The carotid and aortic body chemoreceptor reflexes normally do not function independently of other regulatory mechanisms. When all the regulatory mechanisms function together, the effect of large, prolonged decreases in blood oxygen levels is to increase the heart rate. Low blood oxygen levels also result in increased stimulation of respiratory movements. Increased inflation of the lungs stimulates stretch receptors in the lungs. Afferent action potentials from these stretch receptors influence the cardioregulatory center, which causes an increase in heart rate. The reduced oxygen levels that exist at high altitudes can cause an increase in heart rate even when blood carbon dioxide levels remain low. The carotid and aortic body chemoreceptor reflexes are more important in the regulation of respiration and blood vessel constriction than in the regulation of heart rate.

**Effect of Extracellular Ion Concentration :-** The ions that affect cardiac muscle function are the same ions(potassium, calcium, and sodium) that influence membranepotentials in other electrically excitable tissues. Some differencesexist, however, between the response of cardiac muscle and that ofnerve or muscle tissue to these ions. For example, the extracellularlevels of Na rarely deviate enough from the normal value to affectthe function of cardiac muscle significantly.Excess K in cardiac tissue cause the heart rate and stroke volume to decrease. A twofold increase in extracellular K results in heart block, which is loss of the functional conductionof action potentials through the conducting system of the heart.The excess K in the extracellular fluid cause partial depolarizationof the resting membrane potential, resulting in a decreasedamplitude of action potentials and, because of the decreasedamplitude, a decreased rate at which action potentials are conductedalong cardiac muscle fibers. As the conduction ratesdecrease, ectopic action potentials can occur. In many cases,partially depolarized cardiac muscle cells spontaneously produce 9906757543action potentials because the membrane potential reachesthreshold. Increased blood levels of K can produce enoughectopic action potentials to cause fibrillation. The reducedaction potential amplitude also results in less calcium enteringthe sarcoplasm of the cell; thus, the strength of cardiac musclecontraction decreases.Although the extracellular concentration of K normally issmall, a decrease in extracellular K results in a decrease in theheart rate because the resting membrane potential is hyperpolarized;as a consequence, it takes longer for the membrane todepolarize to threshold. The force of contraction is not affected,however.An increase in the extracellular concentration of Ca 2 producesan increase in the force of cardiac contraction because of a greater influx of Ca 2 into the sarcoplasm during action potentialgeneration. Elevated plasma Ca 2 levels have an indirect effect onheart rate because they reduce the frequency of action potentialsin nerve fibers, thus reducing sympathetic and parasympatheticstimulation of the heart. Generally, elevated bloodCa 2 levels reduce the heart rate.A low blood Ca 2 level increases the heart rate, althoughthe effect is imperceptible until blood Ca 2 levels are reducedto approximately one-tenth of their normal value. The reducedextracellular Ca 2 levels cause Na channels to open, whichallows Na to diffuse more readily into the cell, resulting in depolarization and action potential generation. Reduced Ca 2levels, however, usually cause death as a result of tetany of skeletalmuscles before they decrease enough to markedly influence theheart’s function.

**Effect of Body Temperature :-** Under resting conditions, the temperature of cardiac musclenormally does not change dramatically in humans, althoughalterations in temperature influence the heart rate. Small increasesin cardiac muscle temperature cause the heart rate to increase,and decreases in temperature cause the heart rate to decrease. Forexample, during exercise or fever, increased heart rate and force ofcontraction accompany temperature increases, but the heart ratedecreases under conditions of hypothermia. During heart surgery,body temperature sometimes is reduced dramatically to slow theheart rate and other metabolic functions.

**EFFECTS OF AGING ON THE HEART :-** Aging results in gradual changes in the function of the heart, whichare minor under resting conditions but become more significantin response to exercise and when age-related diseases develop. Themechanisms that regulate the heart compensate effectively formost of the changes under resting conditions.Hypertrophy of the left ventricle is a common age-relatedchange. This appears to result from a gradual increase in the pressurein the aorta against which the left ventricle must pump bloodand a gradual increase in the stiffness of cardiac muscle tissue. Theincreased pressure in the aorta results from a gradual decrease inarterial elasticity, resulting in an increased stiffness of the aortaand other large arteries. Myocardial cells accumulate lipid, andcollagen fibers increase in cardiac tissue. These changes make thecardiac muscle tissue stiffer and less compliant. The increased volumeof the left ventricle can sometimes result in an increase in leftatrial pressure and increased pulmonary capillary pressure. Thiscan cause pulmonary edema and a tendency for people to feel outof breath when they exercise strenuously.There is a gradual decrease in the maximum heart rate. Thiscan be roughly predicted by the following formula: Maximum heartrate 220 age of the individual. There is an increase in the rate atwhich ATP is broken down by cardiac muscle and a decrease in therate of Ca 2transport. There is a decrease in the maximum rate atwhich cardiac muscle can carry out aerobic metabolism. In addition,there is a decrease in the degree to which epinephrine and norepinephrinecan increase the heart rate. These changes are consistentwith longer contraction and relaxation times for cardiac muscle anda decrease in the maximum heart rate. Both the resting and maximumcardiac output slowly decrease as people age and, by 85 yearsof age, the cardiac output may have decreased by 30%–60%.Age-related changes in the connective tissue of the heartvalves occur. The connective tissue becomes less flexible and Ca 2 deposits increase. The result is an increased tendency for heartvalves to function abnormally. There is especially an increasedtendency for the aortic semilunar valve to become stenosed, butother heart valves, such as the bicuspid valve, may become eitherstenosed or incompetent.Atrophy and replacement of cells of the left bundle branchand a decrease in the number of SA node cells alter the electricalconduction system of the heart and lead to a higher rate of cardiacarrhythmias in elderly people.The enlarged and thickened cardiac muscle, especially in theleft ventricle, consumes more oxygen to pump the same amount ofblood pumped by a younger heart. This change is not significantunless the coronary circulation is decreased by coronary artery disease.However, the development of coronary artery disease is agerelated.Congestive heart disease is also age-related. Approximately10% of elderly people over 80 have congestive heart failure, anda major contributing factor is coronary artery disease. Becauseof the age-related changes in the heart, many elderly people arelimited in their ability to respond to emergencies, infections, bloodloss, and stress.Exercise has many beneficial effects on the heart. Regularaerobic exercise improves the functional capacity of the heart atall ages, providing no conditions develop that cause the increasedworkload of the heart to be harmful.