CHAPTER II

LITERATURE REVIEW

2.1 INTRODUCTION

The heart [10] is the centre of the cardiovascular system. Where as the term cardio refers to the heart, the term vascular refers to blood vessels (or an abundant blood supply). The heart propels blood through thousands of miles of blood vessels, and it is magnificently designed for this task. Although we ignore its activity most of the time, the heart's capacity work is remarkable. Even at rest, the heart pumps 30 times its own weight each minute, about 5 liters to the lungs and the same volume to the rest of the body. At this rate the heart would pump more than 7000 liters of blood in a day and 5 million liters in a year. Since we don't spend all our time “resting” and since our heart pumps more vigorously when we are active, the actual flow is much larger.

The cardiovascular system provides the “pump” for circulating constantly refreshed blood through an estimated 100,000 Km of blood vessels. As blood flows through body tissues, nutrients and oxygen move from the blood into the interstitial fluid and then into cells. At the same time the blood picks up wastes, carbon dioxide, and heat.

Cardiology is the branch of medicine dealing with disorders of the heart and blood vessels. To simulate an ECG/arrhythmia patterns, the literature on heart, about the structure (anatomy) of the heart which is the source of ECG, Conduction System of the heart i.e. how the impulse is generated and conducted to various parts of the heart and body and how the electrocardiogram (ECG) is generated is needed to understand first. This also describes about the details of the ECG waves, their amplitude, timings and abnormalities, and also gives the information about the arrhythmias, What are these, their classification, description, features, symptoms and causes about major life threatening arrhythmias which is of clinical significance to the cardiologist. This is discussed in the following sections.

2.2 THE HEART

The heart is a four chambered pump with two atria for collection of blood and two ventricles for pumping out of blood. Coordinated electrical events and a specialized
and attaches to the diaphragm with its open end fused to the connective tissues of the blood vessels entering and leaving the heart. Its lateral surfaces lie against the parietal pleurae, the outer coverings of the lungs. The fibrous pericardium prevents overstretching of the heart, provides protection and anchors the heart in the mediastinum.

The inner serous pericardium is a thinner, more delicate membrane that forms a double layer around the heart. The outer parietal layer of the serous pericardium is fused to the fibrous pericardium. The inner visceral layer of the serous pericardium, also called the epicardium adheres tightly to the muscle of the heart. Between the parietal and visceral layers of the serous pericardium is a thin film of serous fluid known as pericardial fluid. It is a slippery secretion of the pericardial cells and reduces friction between the membranes as the heart moves. The space that houses the pericardial fluid is called the pericardial cavity [10].

**Heart Wall**

Three layers from the wall of the heart: the epicardium (external layer), myocardium (middle layer), and endocardium (inner layer). The outermost epicardium
also called the visceral layer of the serous pericardium is the thin, transparent outer layer of the wall. It is composed of mesothelium and delicate connective tissue that imparts a smooth, slippery texture to the outermost surface of the heart.

The middle myocardium [10], which is cardiac muscle tissue, makes up the bulk of the heart and is responsible for its pumping action. Cardiac muscle fibers (cells) are involuntary, striated and branched. They swirl diagonally around the heart in interlacing bundles and form two large networks— one atrial and one ventricular. Each fiber physically contacts neighbouring fibers in its network by transverse thickenings of the sarcolemma called intercalated discs. Within the discs are gap junctions (electrical synapses) that allow muscle action potentials to spread from one fiber to another. As a result, the whole atrial network contracts as another. The intercalated discs also contain desmosomes, which act as reinforcing spot welds. They prevent adjacent cardiac fibers pulling apart during their vigorous contractions.

The innermost endocardium [10] is a thin layer of endothelium overlying a thin layer of connective tissue. It provides a smooth lining for the inside of the heart and covers the valves of the heart. The endocardium is continuous with the endothelial lining of the large blood vessels associated with the heart and the rest of the cardiovascular system.

Chambers of the heart

The interior of the heart is divided into four compartments called chambers [1, 10] that receive the circulating blood. The two superior chambers are called the right atrium and left atrium. Each atrium has an appendage called an auricle [10], so named because its shape resembles a dog’s ear, The auricles increase the volume of the atria. The two inferior chambers are the right ventricle and left ventricle.

Connective tissue separates the muscle tissue of the atria from that of the ventricles and effectively divides the myocardium into separate atrial and ventricular muscle masses. Externally, a groove known as the coronary sulcus and posterior interventricular sulcus separate the right and left ventricles externally. Sulci contain coronary blood vessels and a variable amount of fat.
A partition called the interatrial septum separates the atria. A prominent feature of this septum is an oval depression, the fossa ovalis. This was the site of the foramen ovale, an opening in the interatrial septum of the fetal heart. The irregular surface of ridges and folds of the myocardium covered by endocardium in the ventricles is known as the trabeculae carneae. A wall known as the interventricular septum [10] separates the two ventricles. The thickness of the walls of the four chambers varies according to their functions. The atria are thin-walled [10, 11] because they only have to deliver blood into the ventricles. Although the right and left sides of the heart act as two separate pumps, the left side has much larger workload. Whereas the right ventricle pumps blood only to the lungs (pulmonary circulation), the left ventricle pumps blood to all other parts of the body (systemic circulation). Thus the left ventricle must work harder than the right ventricle to maintain the same rate of blood flow. The anatomy of the two ventricles confirms this functional difference: the muscular wall of the left ventricle is two to four times as thick as the wall of the right ventricle.

**Blood flow through the heart**

The right atrium receives deoxygenated blood (blood that has given up some of its oxygen to cells) from various parts of the body through three veins. In general the superior vena cava (SVC) brings blood from most parts of the body superior to the heart, the inferior vena cava (IVC) brings blood from all parts of the body inferior to the diaphragm, and the coronary sinus drains blood from most of the vessels supplying the wall of the heart [10].

From the right atrium blood flows into the right ventricle, which pumps it to the lungs, starting in the pulmonary trunk. The pulmonary trunk divides into a right and left pulmonary artery, each of which carries blood to one lung. In the lungs, the blood releases carbon dioxide and takes on oxygen. This blood, called oxygenated blood, returns to the heart via four pulmonary veins that empty into the left atrium. The blood then passes into the left ventricle, which pumps the blood into the ascending aorta. From here the blood flows into the coronary arteries, which carry the blood to the heart, arch of the aorta, thoracic aorta and abdominal aorta [10,12]. The aorta and its branches carry the blood throughout the systemic circulation.
Valves of the heart

As each chamber of the heart contracts, it pushes a portion of blood into a ventricle or out of the heart through an artery. To prevent backflow of blood [10, 12], the heart has valves. These structures are composed of dense connective tissue covered by endocardium. Valves open and close in response to pressure changes as the heart contracts and relaxes [10, 12, 13].

Atrioventricular Valves

Atrioventricular (AV) valves [10] lie between the atria and ventricles. The right AV valve between the right atrium and right ventricle is also called the tricuspid valve because it consists of three cups (flaps). The left AV valve between the left atrium and left ventricle has two cups and is called the bicuspid [10].

Semilunar Valves

Both arteries that emerge from the heart have a valve that prevents blood from Hawing backward into the heart. These are the semilunar (SL) valves [10]. The pulmonary semilunar valve lies in the opening where the pulmonary trunk leaves the right ventricle, The aortic semilunar valve is situated at the opening between the left ventricle and the aorta.

Both valves consist of three semilunar (half-moon, or crescent-shaped) cusps, Each cusp is attached by its convex margin to the artery wall, The free borders of the cusps curve outward and project into the opening inside the blood vessel. Like the atrioventricular valves, the semilunar valves permit blood to flow in one direction only in this case, the flow is from the ventricles into the arteries.

Heart Blood Supply

The wall of the heart has its own blood vessels. Nutrients could not possibly diffuse through all the layers of cells that make up the heart tissue. The flow of blood through the many vessels that pierce the myocardium is called the coronary (cardiac) circulation [10]. The arteries of the heart encircle it like a crown encircles the head (corona = crown).
Coronary Arteries

Two coronary arteries as the right and left coronary arteries, branch from the ascending aorta. The left coronary artery courses under the left auricle and divides into the anterior interventricular and circumflex branches. The anterior interventricular branch or left anterior descending (LAD) artery is in the anterior interventricular and supply oxygenated blood to the walls of both ventricles. The circumflex branch lies in the coronary sulcus and distributes oxygenated blood to the walls of the left ventricles and left atrium.

The right coronary artery supplies small branches to the right atrium. It continues under the right auricle and divides into the posterior interventricular and marginal branches. The posterior interventricular branch follows the posterior interventricular sulcus and supplies the walls of the two ventricles with oxygenated blood. The marginal branch in the coronary sulcus transports oxygenated blood to the myocardium of the right ventricle. The left ventricle receives the most abundant blood supply because of the enormous work it must do.

Most parts of the body receive branches from more than one artery, and where two or more arteries supply the same region, they usually connect. The connections, called anastomoses provide alternate routes for blood to reach a particular organ or tissue. The myocardium contains many anastomoses, connecting branches of one coronary artery or extending between branches of different coronary arteries. Heart muscle can remain alive if it receives as little as 10 to 15% of its normal blood supply.

Coronary Veins

As blood passes through the coronary circulation, it delivers oxygen and nutrients and collects carbon dioxide and wastes. It then drains into a large vein on the posterior surface of the heart called the coronary sinus, which empties into the right atrium. A vascular sinus is a vein with a thin wall that has no smooth muscle to alter its diameter. The principal tributaries carrying blood into the coronary sinus are the great cardiac vein, which drains the anterior aspect of the heart, and the middle cardiac vein, which drains the posterior aspect of the heart.
2.3 CONDUCTION SYSTEM

An inherent and rhythmical electrical activity [13] is the force behind the heart's continuous beating. Certain cardiac muscles [13, 14, 15] cells repeatedly fire spontaneous impulses (action potentials) that then trigger heart contractions. Therefore a heart that has been completely removed from the body, for example, to be transplanted into another person will continue to beat even though all its nerves have been cut. Signals from the autonomic nervous system and hormones, such as epinephrine, in the blood do modify the heartbeat [16, 17], but they do not establish the fundamental rhythm.

2.3.1 AUTORHYTHMIC CELLS

During embryonic development, a small fraction (about 1%) of the cardiac muscle fibers become autorhythmic (self-excitable), that is, able to repeatedly and rhythmically generate impulses [15, 18, 19]. The autorhythmic fibers have two important functions. They act as a pacemaker [18] setting the rhythm [16] for the entire heart, and they form the conduction system, the route for conducting impulses throughout the heart muscles. The conduction system ensures that cardiac chambers contract in a coordinated manner, which makes the heart an effective pump. The components [14] of the conduction system are: the sinoatrial (SA) node, the atrioventricular (AV) node. The atrioventricular (AV) bundle (bundle of His), the right and left bundle branches, and the conduction myofibers (Purkinje fibers).

Normally, cardiac excitation begins in the sinoatrial (SA) node located in the right atrial wall just below the opening of the superior vena cava. Each SA node impulse travels throughout the heart via the conduction system and the gap junctions in the intercalated discs, in the wake of the impulse first the atria contract and then the ventricles contract.

The cardiac impulse [18] spreads from the SA node throughout the atrial fibers and down to the atrioventricular (AV) node located in the septum between the two atria. From the AV node the impulse enters the atrioventricular (AV) bundle (bundle of His), the only electrical connection between the atria and the ventricles. (Elsewhere, fibrous rings and sheets of connective tissue act as electrical insulation between the atria and ventricles.) After traveling along the AV bundle the impulse then enters both the right, and left bundle branches, that course through the interventricular septum toward the apex of the heart. Finally, large-diameter conduction myofibers (Purkinje fibers) rapidly conduct the impulse into the mass of ventricular muscle tissue.
Autorhythmic fibers in the SA node spontaneously initiate action potentials [10, 14, 18] 60 to 100 times per minute, faster than any other region. As a result, action potentials from the SA node spread to other areas of the conduction system, stimulating them before they are able to generate an impulse at their own slower rate. Thus the normal pacemaker of the heart is the SA node.

Sometimes a site other than the SA node becomes the pacemaker because it develops abnormal self-excitability. Such a site is called an ectopic pacemaker or ectopic focus. The ectopic focus may operate only occasionally, producing extra beats, or it may pace the heart for some period of time. Triggers of ectopic activity include caffeine and nicotine, electrolyte imbalances, hypoxia, and toxic reactions to drugs such as digitalis [1].

2.3.2 TIMING OF ATRIAL AND VENTRICULAR EXCITATION

From the SA node the cardiac impulse travels (throughout the atrial muscle and down to the AV node in about 0.05 sec (50 milliseconds or msec). The impulse slows considerably at the AV node because the fibers there have much smaller diameters. The resulting 0.1 sec (100 msec) delay has an advantage. It gives the atria, time to complete their contraction and add to the volume of blood in the ventricles before ventricular contraction begins. After the cardiac impulse enters the AV bundle conduction again is rapid. The entire ventricular myocardium undergoes depolarization [10] (loss of and then reversal of polarization) about 0.15 to 0.2 sec (150 to 200 msec) after the impulse arises in the SA node.

If the SA node becomes diseased or damaged, the slower AV node fibers can pick up the pacemaking chores. With pacing by the AV node, heart rate ranges between 40 and 50 beats/min. If the activity of both nodes is suppressed, the heartbeat may still be maintained by autorhythmic fibres in the ventricles-the AV bundle, a bundle branch, or conduction myofibers. However, these fibers fire impulses very slowly, only about 20 to 40 times per minute. At such an abnormally low heart rate blood flow to the brain is inadequate. In patients with such a conditions normal heart rhythm can be restored and maintained with an artificial pacemaker, a device that sends out small electrical currents that stimulate the heart. Many of the newer pacemakers, called activity-adjusted pacemakers automatically speed up the heartbeat during exercise.
2.3.3 PHYSIOLOGY OF CARDIAC MUSCLE CONTRACTION

The impulse initiated by the SA node travels along the conduction system [14, 15] and spreads out to excite the ‘working’ atrial and ventricular muscle fibers, which are called contractile fibers. The contractile fibers have a resting membrane potential [10, 16] close to -90 mV, when they are brought to threshold by excitation in neighbouring fibers. Certain sodium ion channels opens very rapidly; these are called voltage-gated fast Na\(^+\) channels. This increase in membrane permeability allows an inflow of Na\(^+\) down its concentration gradient and produces a rapid depolarization. Refer Figure 2.2 for impulse (Action Potential) in a ventricular Contractile Fiber.

![Diagram of Action Potential in Cardiac Muscle Fiber](image)

**Figure 2.2 : Impulse (Action Potential) in a ventricular Contractile Fiber.**

During the next phase, called the plateau, voltage-gated slow Ca\(^{2+}\) channels open, allowing calcium ions enter the cytosol. Some Ca\(^{2+}\) passes through the sarcolemma (plasma membrane) from the extra cellular fluid (which has a higher Ca\(^{2+}\) concentration) while other calcium pours out of the sarcoplasmic reticulum within the fiber. The combined buildup of Na\(^+\) and Ca\(^{2+}\) in the cytosol maintains the depolarization for about 0.25 sec (250 msec). By comparison, depolarization in a neuron or skeletal muscles fiber lasts about 1 msec.

The next steps are similar in skeletal and cardiac muscle fibers. Ca\(^{2+}\) binds to troponin, which allows the actin and myosin filaments to begin sliding past one another,
and tension starts to develop. Substances that alter the movement of $\text{Ca}^{2+}$ through slow $\text{Ca}^{2+}$ channels influence the strength of heart contractions. Epinephrine, for e.g., increases contraction force by enhancing $\text{Ca}^{2+}$ inflow. Certain drugs, appropriately called calcium channel blockers such as verapamil reduce $\text{Ca}^{2+}$ inflow and diminish the strength of the heartbeat.

The repolarization (recovery of resting membrane potential) phase of the impulse in a cardiac muscle fiber resembles repolarization in other excitable tissues after a delay (which is particularly prolonged in cardiac Muscle), voltage-gated $\text{K}^+$ channels open, and potassium ions diffuse out along their concentration gradient. At the same time, the $\text{Na}^+$ and $\text{Ca}^{2+}$ channels are closing which slows and then almost stops further inflow of these two ions. As more $\text{K}^+$ leaves the fiber and fewer $\text{Na}^+$ and $\text{Ca}^{2+}$ enter the negative resting membrane potential (-90 mV) is restored and the muscle fiber relaxes.

In muscle the refractory period is the time interval when a second contraction cannot be triggered. The refractory period of a cardiac fiber is longer than the contraction itself. As a result, another contraction cannot begin until relaxation is well underway and tetanus (maintained contraction) cannot occur. The pumping function of ventricles depends on alternating contraction, when they eject blood and relaxation, when they refill. If tetanus could occur blood flow would stop.

2.3.4 CARDIAC ACTION POTENTIAL

The cardiac action potential [1, 10] is the electrical activity of the individual cells of the electrical conduction system of the heart.

The cardiac action potential differs significantly in different portions of the heart. This differentiation of the action potentials allows the different electrical characteristics of the different portions of the heart. For instance, the specialized conduction tissue of the heart has the special property of depolarizing without any external influence. This is known as automaticity.

The electrical activity of the specialized conduction tissues are not apparent on the surface electrocardiogram (ECG) [13]. This is due to the relatively small mass of these tissues compared to the myocardium (muscle of the heart).
Resting membrane potential

The resting membrane potential [10] is the difference in ionic charge across the membrane of the cell during phase 4 of the action potential. Table 2.1 shows intracellular and extracellular ion concentrations.

**Table 2.1 Intracellular and extracellular ion concentrations**

<table>
<thead>
<tr>
<th>Ion</th>
<th>Extracellular concentration (mM)</th>
<th>Intracellular concentration</th>
<th>Ratio of extracellular to intracellular concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na⁺</td>
<td>145</td>
<td>15 mmol/L</td>
<td>9.7</td>
</tr>
<tr>
<td>K⁺</td>
<td>4</td>
<td>150 mmol/L</td>
<td>0.027</td>
</tr>
<tr>
<td>Cl⁻</td>
<td>120</td>
<td>5-30 mmol/L</td>
<td>4-24</td>
</tr>
<tr>
<td>Ca²⁺</td>
<td>2</td>
<td>10-7 mmol/L</td>
<td>2x10⁴</td>
</tr>
</tbody>
</table>

Although Intracellular Ca2+ content is about 2 mM, most of this is bound or sequestered in intracellular organelles (mitochondria and sarcoplasmic reticulum).

The normal resting membrane potential in the ventricular myocardium is about -85 to -95 mV. This potential is determined by the selective permeability of the cell membrane to various ions. The resting membrane potential is permeable to K⁺, and is relatively impermeable to other ions. The resting membrane potential is therefore determined by the K⁺ gradient across the cell membrane (the reversal potential for K⁺). The maintenance of this electrical gradient is due to various ion pumps and exchange mechanisms, including the Na⁺ - K⁺ ion exchange pump and the Na⁺ - Ca²⁺ exchange mechanism.

Intracellularly (within the cell), K⁺ is the principle cation, and phosphate and the conjugate bases of organic acids are the dominant anions. Extracellularly (outside the cell), Na⁺ and Cl⁻ predominate.

**Phases of the cardiac action potential**

The standard model used to understand the cardiac action potential is the action potential of the ventricular myocyte. The action potential has 5 phases (numbered 0 - 4).
Phase 4 is the resting membrane potential, and describes the membrane potential when the cell is not being stimulated.

Once the cell is electrically stimulated (typically by an electric current from an adjacent cell), it begins a sequence of actions involving the influx and efflux of multiple cations and anions that together produce the action potential of the cell, propagating the electrical stimulation to the cells that lie adjacent to it. In this fashion, an electrical stimulation is conducted from one cell to all the cells that are adjacent to it, to all the cells of the heart. Refer figure 2.3 for Cardiac Action Potential Phases.

![Figure 2.3: Cardiac Action Potential Phases](image)

**Phase 4**

Phase 4 is the resting membrane potential. This is the period that the cell remains in until it is stimulated by an external electrical stimulus (typically an adjacent cell). This phase of the action potential is associated with diastole [12, 13] of the chamber of the heart.

Certain cells of the heart have the ability to undergo spontaneous depolarization, in which an action potential is generated without any influence from nearby cells. This is also known as automaticity. The cells that can undergo spontaneous depolarization the fastest are the primary pacemaker cells of the heart, and set the heart rate [16]. Usually, these are cells in the SA node of the heart. Electrical activity that originates from the SA node is propagated to the rest of the heart. The fastest conduction of the electrical activity is via the electrical conduction system [14] of the heart.

In cases of heart block [20, 21, 22], in which the activity of the primary
pacemaker does not propagate to the rest of the heart, a latent pacemaker (also known as an escape pacemaker) will undergo spontaneous depolarization and create an action potential.

The mechanism of automaticity is still unclear. Depolarization of SA and AV nodal cells largely depend on a net increase in intracellular positive charge [23]. Mechanisms include a decrease in the net K⁺ outward flow, and a time-dependent increase in flow of Na⁺ and Ca²⁺ ions.

**Phase 0**

Phase 0 is known as the rapid depolarization phase. The slope of phase 0 is determined by the maximum rate of depolarization of the cell and is known as Vmax. This phase is due to opening of the fast Na⁺ channels and the subsequent rapid increase in the membrane conductance to Na⁺ (gNa) and a rapid influx of ionic current in the form of Na⁺ ions (INa) into the cell.)

The ability of the cell to open the fast Na⁺ channels during phase 0 is related to the membrane potential at the moment of excitation. If the membrane potential is at its baseline (about -85 mV), all the fast Na⁺ channels are closed, and excitation will open them all, causing a large influx of Na⁺ ions. If, however, the membrane potential is less negative, some of the fast Na⁺ channels will be opened earlier, causing a lesser response to excitation of the cell membrane and a lower Vmax.

The maximal fast inward Na⁺ current is generated when the membrane potential is at the normal resting potential (-85 to -95 mV). If the resting membrane potential is reduced to a low enough level, the increase in fast inward Na⁺ current may be inadequate to produce a response, making the fiber unexcitable

**The fast Na⁺ channel**

The fast sodium channel is made up of two gates, the m gate and the h gate. It is the interaction of these two gates that allows Na⁺ to enter the cell through this channel. In the resting state, the m gate is closed and the h gate is open. Upon electrical stimulation of the cell, the m gate opens quickly while simultaneously the h gate closes slowly. For a brief period of time, both gates are open and Na⁺ can enter the cell across the electrochemical gradient.
Phase 1

Phase 1 of the action potential is due to closure of the fast Na\(^+\) channels. The transient net outward current is due to the movement of K\(^+\) and Cl\(^-\) ions.

Phase 0 and 1 together correspond to the R and S waves of the EGG.

Phase 2 of the action potential corresponds to the ST segment of the EGG.

Phase 3

During phase 3 of the action potential, the K\(^+\) channel is still open, allowing more K\(^+\) to leave the cell and accumulate in the extracellular space. This net loss of positive charge causes the cell to repolarize. The k\(^+\) channels close when the membrane potential is restored to about -40 to -45 mV.

Phase 3 of the action potential corresponds to the T wave [24] on the EGG.

2.3.5 PACEMAKER TISSUES

Certain tissues in the heart [10, 12, 13], concerned with the initiation (generation of impulses) and propagation (conduction) of the heart beat, are called pacemaker tissues. They include:

1. Sinu or Sino Atrial Node (SAN)
2. Atrio-Ventricular Node (AVN)
3. Atrio-Ventricular Bundle or Bundle of His
4. Purkinje fibers that is ramification of Bundle of His

Sinu Atrial Node (SAN)

I. Location: on the posterior aspect of heart [10, 12] at the junction of the superior venacava (SVC) with right atrium (RA)(free b order of the RA appendix).

II. Dimensions: Length-15mm; Width-2mm and Thickness-1mm.

III. Structure: more embryonal in character i.e. cell outline ill defined; highly vascular; consists of thin, elongated muscle fibers (approx. 1/3\(^{rd}\) the size of heart muscle fibers); rich in glycogen and mitochondria, fusiform in shape with longitudinal striations. These are called P-cells or pacemaker cells. These fibers normally can generate and discharge impulses more rapidly than any other
pacemaker tissue and their rate of discharge determines the rate at which the heart beats. Because cardiac myocytes, like all nerve cells, have refractory periods following contraction during which additional contractions cannot be triggered, their pacemaker potential is overridden by the sinoatrial node. Cells in the SA node will naturally discharge (create action potentials) at about 70-80 times/minute. Because the sinoatrial node is responsible for the rest of the heart's electrical activity, SAN is called the Cardiac Pacemaker [10, 12, 13].

IV. Innervation: It develops from structures on the right side of the embryo. That is why, in adults, SAN is innervated by right vagus nerve. It also receives sympathetic nerve fibers predominantly of right side from the cervical sympathetic ganglia via the cardiac nerves. This makes the SA node susceptible to autonomic influences. Stimulation of the vagus nerve causes decrease in the SA node rate (thereby causing decrease in the heart rate). Stimulation via sympathetic fibers causes increase in the SA node rate (thereby increasing the heart rate).

In the majority of patients, the SA node receives blood from the right coronary artery, meaning that a myocardial infarction occluding it will cause ischaemia in the SA node unless there is a sufficiently good anastomosis from the left coronary artery. If not, death of the affected cells will stop the SA node from triggering the heartbeat.

Atrio ventricular node (AVN)

If the SA node doesn't function, or the impulse generated in the SA node is blocked before it travels down the electrical conduction system, a group of cells further down the heart will become the heart's pacemaker. These cells form the atrioventricular node (AV node), which is an area between the atria and ventricles, within the atrial septum.

I. Location: posteriorly on right side of the interatrial septum near the opening of coronary sinus.

II. Structure: same as that of SAN.
III. Innervation: It is a left sided structure of the embryo. Therefore, in adults, it is innervated by left vagus nerve; also receives sympathetic nerve supply simply from left side.

**Atrio-ventricular bundle or the bundle of His**

I. It takes origin from AVN and then runs upwards to the posterior margin of the membranous inter-ventricular septum and then forwards below it, ensheathed and isolated in the canal. At the anterior part of the membranous inter-ventricular septum the bundle divides into a left and right branch.

II. The left branch pierces the membrane and then lies on the upper border of the muscular septum to divide into an anterior fascicle and a posterior fascicle. The right branch passes down the right side of the septum.

III. Both branches divide repeatedly to form a network of fibers lying subendocardially in the ventricles.

**Purkinje fibers**

I. Takes origin from terminal divisions of right and left branch of the bundle of His to penetrate the ventricular wall.

II. These fibers [13] are somewhat larger and thicker than the cardiac muscle fibers; length: 10-46μm and diameter 70-80 μm with indistinct cell outlines and granular central cytoplasm containing several nuclei; the peripheral cytoplasm [17] contains myofibrillae and is rich in glycogen content.

III. Because of the large diameter they transmit the impulse at a fast velocity of 4mts/sec as compared to other conducting tissue. This allows almost immediate transmission of the cardiac impulse throughout the entire ventricular system. Table 2.2 gives conduction speed in Cardiac Tissue.
Table 2.2: Conduction speed in Cardiac Tissue.

<table>
<thead>
<tr>
<th>Cardiac Tissue</th>
<th>Conduction Speed (mts/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Sino Atrial Node</td>
<td>0.05</td>
</tr>
<tr>
<td>II Atrial Pathways</td>
<td>1.0</td>
</tr>
<tr>
<td>III Atrio-ventricular node</td>
<td>0.05</td>
</tr>
<tr>
<td>IV Bundle of His</td>
<td>1.0</td>
</tr>
<tr>
<td>V Purkinje system</td>
<td>4.0</td>
</tr>
<tr>
<td>VI Ventricular muscles</td>
<td>1.0</td>
</tr>
<tr>
<td>VII Atrial muscles</td>
<td>1.0</td>
</tr>
</tbody>
</table>

Refer Figure 2.4 for Conduction System of the Heart. Pacemaker activity at the SA node reaches the AV node by cell-to-cell atrial conduction. The conduction system carries the impulse from the AV node to the working ventricular muscle by traversing the common bundle of His, the right or left bundle branch, and the Purkinje network. RA, right atria; LA, left atria; RV, right ventricle; LV, left ventricle.

Figure 2.4: Conduction System of the Heart

Pacemaker cells are located in the region of the sinoatrial (SA) node, which is a bit of tissue the size of a pencil tip located at the site of entry of the descending vena cava.
Repetitive activity is initiated at this point and propagates to adjoining atrial tissue by means of the local-circuit (action) currents. The flow of this current from active to inactive neighboring cells is facilitated by the presence of low-resistance intercellular structures. Activation proceeds in this way from cell to cell until the entire right and then left atria are activated. (The nature of the intercellular structures in both atrial and ventricular tissue will be described subsequently in this section.) Because the atria and ventricles are separated by fibrous tissue, direct propagation from the atria to ventricles cannot occur. Instead activation must follow a path that starts in the atria at the atrioventricular (AV) node and proceeds through the common and then right and left bundles of His, to the terminal Purkinje fibers which arborize and invaginate the endocardial ventricular tissue. The initial part of this path involves slow conduction in the AV junction. Since electrical activation of cardiac muscle initiates the successive mechanical contraction, this results in a delay in ventricular activation and contraction that is beneficial. Since it allows for completion of atrial contraction. Once the electrical impulse reaches the bundles of His, conduction is very rapid, resulting in the initiation of ventricular activation over a wide region. The subsequent cell-to-cell propagation is consequently sequenced and coordinated, resulting in a mechanical contraction that is similarly synchronized and efficient.

Activation of Ventricular musculature, initiated at many endocardial sites by the conduction system, proceeds generally from endocardium to epicardium through cell-to-cell conduction. Such conduction is made possible by low-resistance intercellular junctions, which permit the flow of a sufficient fraction of prejunctional action current to the post junctional cell to result in activation of the latter. Specific low-resistance hexagonal structures called connexons have been identified through freeze fracture electron microscopy; the connexons are organized in Hexagonal arrays that comprise the gap junctions. It is a central tube of the connexon that is believed to constitute an intercellular channel for the movement of ions and small molecules. The connexons span a gap between adjoining cells that is narrowed to 30 μm from a normal 200 μm intercellular space. If one thinks of two end-to-end cells in the shape of successive tank cars, the connexons represent a number of interconnecting pipes providing for interunit cytoplasmic continuity. In this model one can understand how action currents arising in a prejunctional cell can flow, relatively easily, into the post junctional cell and then
outward across the membrane. In so doing, a depolarizing transmembrane potential is produced in the post junctional cell, which will eventually reach threshold in much the same way as this occurs at a downstream site in a uniform continuous (single) cylindrical cell.

Cardiac ventricular tissue when viewed on a larger scale is seen to be composed of bands of fibers that spiral around the central cavity. If passive gross electrical conductivity measurements are made at a specific site, conductivity along the fiber axis will be found to exceed that transverse to the fibers by a factor of 4 or so. One consequence is that propagation of an impulse in the fiber direction will proceed at a velocity that is around two times greater than that in the direction normal to fiber. These anisotropies play an important role in determining activation patterns and source strength and configuration. On the other hand for the most part, principal effects can be discerned if one assume isotropic behaviour. Such an assumption implies that all cells connect equally well to all neighbors and that electrical activity initiated at a point would propagate outward as a spherical wave. The heart muscle [15] is referred to as syncytial reflecting an electrically excitable continuum.

2.3.6 (SINGLE-FIBER) ELECTROPHYSIOLOGICAL PRINCIPLES

Activation

Nerves and muscles have the properties of electrical excitability. When stimulated the result is a cyclic movement of ions into and out of each activated cell, and this is accompanied by changes in transmembrane potential. The latter constitutes an electrical impulse that is seen to propagate from the site of excitation to adjoining regions.

This behaviour arises basically from the nature of the excitable membrane that bounds each cell. This membrane contains active elements (pumps), which establish and maintain an ionic intracellular composition that is different from the extracellular milieu. The intracellular space is high in potassium and low in sodium and chloride while the extracellular medium, conversely, has a low potassium concentration but high sodium and chloride concentrations. The additional membrane property that accounts for excitability is its selective permeability. At rest it is high for potassium so that the outward diffusion of this ion (alone) causes the membrane capacitance to develop a negative intracellular
charge (positive extracellularly). The consequence is a resting intracellular electrical potential that is negative relative to the extracellular space (taken as a reference), and this is in the order of 0.1 V.

If a stimulating current is applied such that a ‘patch’ of membrane is caused to have an increased transmembrane potential by a threshold amount (around 10-15 mV), the membranes permeabilities are caused to change through a cyclic process. The change is characterized by an initial rapid rise in sodium permeability to a dominant value and an equally rapid fall in this value toward its resting level; this is accompanied by a slower rise and fall in potassium permeability in the case of nerve and muscle (for cardiac muscle a series of permeability changes results in delayed recovery.

The concomitant transmembrane potential [12, 15] undergoes the phasic changes as described, and this constitutes an action potential. As noted previously the resting potential arises from potassium efflux due to diffusion and this ceases (i.e., a steady state is reached) when the inward electric field from the charge displacement equilibrates the outward diffusional force. This equilibrium can be evaluated through the application of electrical field theory (10) and diffusion theory, giving rise to the Nernst equation, which (for potassium) is

\[ V_m = 25 \ln\left(\frac{[K]_e}{[K]_i}\right) \]  

where \( V_m \) is the transmembrane potential in millivolts (the Nernst potential) \([K]_e\) is the extracellular potassium concentration, and \([K]_i\) is the intracellular potassium concentration. For typical values of \([K]_e = 4\mu \text{mol/cm}^3\) and \([K]_i = 155 \mu \text{mol/cm}^3\), \( V_m = -91.4 \text{ mV} \) is determined. The steady resting potential of a cardiac cell is approximated by the potassium Nernst potential. Activation (depolarization) arises when the transmembrane potential is increased say to -75 mV by abruptly passing a current that flows outward across the membrane. A consequence is a rapid increase in sodium permeability. The result is an incremental sodium influx (due to both the inward electric field and the inward sodium concentration gradient and this results in a further increase (algebraically) in transmembrane potential, and so on. The process is regenerative producing further increases in sodium permeability and transmembrane potential, and ends essentially when
the transmembrane potential reaches the sodium equilibrium (Nernst) potential. Typical values of intracellular and extracellular sodium concentrations in cardiac muscle are $[\text{Na}]_e = 145\mu\text{mol/cm}^3$ and $[\text{Na}]_i = 12\mu\text{mol/cm}^3$, so that

$$V_m = 25\ln \left( \frac{145}{12} \right) = 62.3\text{mV}$$  \hspace{1cm} (2)

is the potential at the action potential peak; one notes a reversal in polarity at this time. Recovery consists mainly in the sodium and potassium permeabilities returning to their resting values and, consequently, a return of the transmembrane potential to its resting value. During the early period of recovery, the membrane is inexcitable (absolutely refractory) and, toward the close of recovery, can be excited, but requires an abnormally high stimulus (relatively refractory).

**Propagation**

If one end of a long cylindrical fiber [25] is excited by the application of a transthoracic stimulus, propagation of the action potential impulse to the opposite end is seen to occur. The extracellular pair of electrodes A, B is connected so that A is the cathode and B the anode of a stimulus current. The flow of current from B to A, which passes across the initially resting membrane in an inward direction at B and outward at A. The resting membrane can be characterized by a passive resistance and capacitance in parallel and the effect of the stimulating current will be an exponential change in transmembrane potential. From application of elementary electrical circuit concepts. This stimulating current is seen to depolarize the membrane at A and hyperpolarize it at B. Consider the IR drop in potential in the membrane resistance. If the stimulus current is large enough to exceed threshold at A, an action potential will be elicited at that site. Refer Figure 2. 5 for Stimulating Current flow following closure of switch corresponds to the passive circuit shown. Depolarisation takes place under the cathode A.
Figure 2.5 (a) Stimulating current flow following closure of switch corresponds to the passive circuit shown. Depolarization takes place under the cathode. A. (b) At a short time following (a), there is a rapid rise in sodium permeability at A, permitting a sodium influx and rise in transmembrane potential. The currents shown respond to the resulting differences in potential.

The current flow pattern arising from initiation of excitation at A is depicted. The pattern results from the following considerations. Activation at A is associated with the rapid rise in sodium permeability at this site and results in the transmembrane potential approaching the sodium Nernst potential. Since the transmembrane potential is at rest in an adjoining region, such as C, currents must flow in the direction, as a result of the differences in potential. Since charge must be conserved, these comprise the closed current loops (called local-circuit currents) of particular importance is the outward current at site C. Since this region is subthreshold it behaves electrically as a resistance and capacitance in parallel as noted earlier. The transmembrane current, consequently, has two components- one a capacitive charging current and the second a resistive current (the latter being actually an ionic current). The transmembrane potential at C is depolarizing (since the intracellular potential is being made more positive). In real and normal physiological [12] preparations it turns out that the strength of the depolarization is always more than adequate to achieve activation. The result is that the pattern shifts to the right (C is now the activation site). The continued (sequential) operation of this mechanism accounts for propagation of the impulse along the fiber. The propagation rapidly reaches a uniform velocity so that if \( V(t) \) is the temporal action potential, \( V(\theta - t) \) is its spacio temporal behavior, where \( \theta \) is the velocity of propagation along the z axis. The field of electric currents associated with the propagating action potential is
referred to as action currents (or local-circuit currents), particularly when their role in supporting propagation is emphasized.

For a single fiber lying in an extensive volume conductor [25] and supporting a propagating action potential, the potential field in the extracellular medium is found to be small compared with intracellular values. An equivalent electrical network can be assigned this preparation reflecting the small extracellular potentials is the assignment of zero resistance to this region. For the intracellular space, its long and narrow character ensures axial current (only), and accordingly an axial ohmic resistance is assigned. The transmembrane path is shown nonspecifically. Since this may involve active as well as passive elements. Refer figure 2.6 for Linear Core conductor model [25] of an excitable fiber in an extensive extracellular space. The open boxes represent a parallel $C_m \Delta z$ and $r_m/\Delta z$ if voltages are subthreshold or a more complex non linear representation if transthreshold. The intracellular potential is designated $\Phi_1$

![Diagram](image)

**Figure 2.6 : Linear Core conductor model**

**Source/Field Relationship (Single Fibers)**

Though the extracellular field is assumed to be small, it is not negligible. The transmembrane current enters the extracellular space and constitutes, thereby a potential field source. The strength of this current can be evaluated by applying Kirchhoff's circuit laws. Therefore

$$ r_i i_i = -\Phi_1 / \Delta z $$

(3)
The above equation equates the intracellular potential [26] change in the z direction to the axial current $i_t$ times the axial resistance per unit length $r_1$, an application of Ohm's law. The transmembrane current per unit length $i_m$ is related to the rate of decrease in intracellular axial current per unit length, since current must be conserved. Accordingly,

$$i_m = -\varphi_i \partial z = (1/r_1)(\partial^2 \varphi_i / \partial z^2)$$  \hspace{1cm} (4)

By defining the transmembrane potential $V_m = \Phi_i - \Phi_e$ and since $\Phi_e = 0$, $\partial \Phi_i / \partial z$ and

$$i_m = (1/r_1)(\partial^2 V_m / \partial z^2)$$  \hspace{1cm} (5)

Now an element of transmembrane current $(i_m \partial z)$ constitutes a point current source within the extracellular volume conductor. If $\sigma_e$ is the extracellular conductivity (assumed uniform) and the region is assumed to extend indefinitely, then the extracellular field of a point current source of strength $I_0 = i_m dz$ is

$$\Phi_e = I_0/(4\pi \sigma R)$$  \hspace{1cm} (6)

where $R$ is the distance from $I_0$ to an arbitrary field point. This formula can be verified calculating the extracellular electrical field $E = -\partial \Phi_e / \partial R = I_0 / (4\pi \sigma_e R^2)$ and the extracellular current destiny $J = \sigma_e E = I_0 / (4 \pi R^2)$, a value that conforms to the uniform radial outflow of current from a point source of magnitude $I_0$. The extracellular potential field from the entire fiber is found by superposition of fields due to all point source elements $(i_m \partial z)$ and applying in equation 6, this leads to

$$\Phi_e = (1/(4\pi \sigma_e)) \int (i_m \partial z)/R$$  \hspace{1cm} (7)

$$\Phi_e = (1/(4\pi \sigma_e r_t)) \int (\partial^2 V_m / \partial z^2)/R$$  \hspace{1cm} (8)

In equation 8, $\partial^2 V_m / \partial z^2$ corresponds to an axial source density [26] so that $\partial^2 V_m / \partial z^2$ behaves like a point source for extracellular fields. By applying this expression to active cardiac muscle which can be thought of as a collection of single fibers, which introduces the specific nature of such muscle.

2.3.7 CARDIAC SOURCES

Because of the syncytial nature of cardiac tissue one can consider the muscle cells as if they were arranged in an endocardial-epicardial direction (even though in fact they
lie transverse to this direction). But such an idealization permits to take into account its application to a multicellular structure to be applied for the evaluation of the cardiac sources of the electrocardiographic field. This is graphically illustrated in Figure 2.7. Refer Figure 2.7 for Evaluation of cardiac sources [15]. Phase 0 is shown spatially having been converted from its temporal waveform based on a rise time of around 1ms and a propagation velocity of 50 cm/s. Assuming an idealized plateau period following depolarization leads to the described source description of a leading positive source and trailing negative source [15]. Since it can be shown that the net positive and negative sources are equal in magnitude, the source arrangement constitute a double (or dipole) [24, 25] layer. It is clear infact that such a dipole layer arises along all isochrones (i.e., along all surfaces defining phase 0 cells) that exist at a particular time instant. As propagation proceeds in the usual endocardial-epicardial direction, double-layer sources accompany them and account for the space-time changing electrocardiographic field.

![Diagram of cardiac sources](image)

**Figure 2.7: Evaluation of cardiac sources**

The strength of the dipole-layer source can be evaluated by the application. Apart from the factor 1/ri, the total positive source is found by integrating \(\frac{dV_m}{az^2}\) from resting \(V_m\) to where \(dV_m/az\) peaks. This integration yields the value \((dV_m/az)_{peak}\) since
\( \partial V_m / \partial z = 0 \), where \( V_m \) is resting. A similar magnitude is found for the total negative source. These magnitudes can be approximated as \((V_{m_{\text{peak}}} - V_{m_{\text{rest}}}) / D\), where \( D \) is the separation of the positive and negative source. The dipole strength \([25]\) involves multiplication of the single source magnitude by the source separation \( D \). As a result the double-layer density \( \tau \), is proportional to \((V_{m_{\text{peak}}} - V_{m_{\text{rest}}}) / r_i\).

The preceding results, since they depend on what was derived for a single fiber source \([15]\) in an unbounded volume conductor, must be modified to some extent to take into account the real (multicellular) cardiac tissue. The modifications affect the quantitative results, but leave the qualitative picture outlined here unchanged.

According to this (approximate) model the double layer source magnitude will be the same everywhere (a uniform, density) since it depends on \( V_{m_{\text{peak}}} - V_{m_{\text{rest}}} \) which appears to be unvarying in experimental studies. The waveform of the electrocardiogram associated with ventricular depolarization is thus thought to arise from the changing shapes of the isochrones, hence double layers \([24, 25]\), from the earliest moment of Purkinje activation to the time when the last bit of tissue has been reached and is activated by the advancing depolarization wave.

The field generated by a double layer can be thought of as the superposition of two component fields, one arising from the layer of positive sources and one from the layer of negative sources.

Denoting the sources layer density of each layer as \( W \) and their separation as \( D \), then, as noted the dipole density strength \( \tau = WD \) (the bold type denotes a vector quantity and \( D \) is the displacement from negative to positive source along the normal to the isochrone). Keeping in mind that \( WdS \) behaves like a point source \((10,11)\). Consequently,

\[
\Phi = \frac{W}{4\pi\sigma} \int \left( \frac{1}{R^+} - \frac{1}{R^-} \right) dS \quad (9)
\]

where \( R^+ \) represents the source-field distance from elements, on the positive layer and \( R^- \) represents that from the negative layer. Since \( D \) is small compared with typical values of \( R^+ \) or \( R^- \) then Eq. can be rewritten as
\[
\Phi = \frac{\tau}{4\pi \sigma} \int \nabla \left( \frac{1}{R} \right) \cdot dS
\]  \hfill (10)

This result arises only because the double layer is assumed to be uniform and consequently its strength (density) can be removed from the integration. Although the sources actually occupy a region around 0.25 mm thick, the procedure has reduced this to an equivalent infinitesimal thickness and the double layer may be thought of as lying at some median position.

The preceding result can be expressed in terms of the net subtended solid angle, since an element of solid angle \(d\Omega = \nabla (1/R) \cdot dS\). Consequently

\[
\Phi = \frac{\tau}{4\pi \sigma} \int d\Omega
\]  \hfill (11)

where \(\Omega\) is the solid angle subtended at the field point by the associated double layer. (If there is more than one double layer, as, e.g., one each may arise due to activation of the right ventricle, left ventricle, and septum, then it must be applied to each and the results superimposed). Since every activation wave with the same periphery has the same solid angle the field of each will be identical. Conversely, given a particular measured field there is no way to distinguish the responsible double-layer-source configuration beyond some particular solid angle, Finally, since a closed (uniform) double layer has zero solid angle, it thereby generates zero potential.

### 2.3.8 EQUIVALENT SINGLE DIPOLE

The cardiac activation [24, 25] sources can be approximated as uniform double layers associated, at each instant of time, with the existing isochrones. A further simplification is possible if each elementary dipole is added to every other, vectorially, to form a single net dipole. Such an approximation, in effect, ignores the spatial distribution of the dipole elements. One could justify this simplification if the distance from source to field (i.e., from points in the heart to point on the torso) is large compared with the extent of the sources, which at most, is a linear heart dimension). Since precordial torso sites are as close or closer to the heart as the extent of the heart this is a questionable approximation. Nevertheless, experimental studies show that the procedure reflects first-order affects reasonably well.
The total dipole vector is assumed to have a fixed origin though a moving origin model has been explored and could be expected to reflect source behavior more accurately). Since activation double layers undergo a continuous smooth set of changes the net dipole can also be expected to change smoothly and continuously. The locus of the tip of this vector forms a closed loop in space and is called the vector loop; in clinical vectorcardiography it is specifically sought as a measure of the heart's electrical nature.

The representation of the electrical activity of the heart as a dipole fixed in location but free to change orientation and magnitude is obviously a great simplification in estimating the source generated by the heart. It also provides a great simplification in estimating the cardiac sources from measurements at the torso surface. (In principle, only three independent electrocardiographic potentials are needed to reconstruct the vector loop. Although the biophysical problem has been simplified, it retains features where one can often make a judicious guess from the behavior of the heart dipole of what may be happening at underlying isochronal double layers. The latter, in turn, can be directly related to the clinical status of the tissue.

Recovery

To this point almost all attention has been placed on the activation process in the heart. Cellular recovery depends on the time of activation, recovery of neighbours, and intrinsic membrane properties (temperature, metabolic activity, etc.). Recovery sources are generated, just as are activation sources, by the presence of spatial gradients of transmembrane potential. But while these could be inferred from the propagating activation wave (given the phase 0 morphology and propagation velocity) the many additional factors in recovery prevent an easy evaluation of these sources (recovery cannot be characterized as a propagating process). There is in fact no existing applicable model for recovery. On the other hand, most of cardiac diagnosis that is based on ECG waveform morphology utilizes only the activation period.

Although recovery sources are more difficult to evaluate than activation sources, it is clear that during recovery cardiac sources arise throughout the entire heart. Their source density magnitude is, however, small. Nevertheless, in the same way as described for activation, each dipole source element can be added to each other to obtain a net recovery heart vector. It, too, is an approximation since the spatial distribution of dipole
elements is ignored.) Since the underlying physical processes are uniform and continuous, 
the net recovery dipole has a continuous locus and defines a recovery (T-wave) vector 
loop. Even though the behavior of this vector loop is poorly understood on theoretical 
grounds, it has been found useful in empirical (statistical) cardiac diagnosis.

**Potential Field Theory**

The equation provides an explicit relationship between a double-layer source and 
the field it generates in a uniform homogeneous conducting medium. It is sometimes 
useful to regard the cardiac electrical sources as a volume distribution of dipoles 
described by a continuous function of position $J_i$. Obviously a system of double-layer 
sources congruent with the instantaneous set of isochrones is a special case of this more 
general formulation. The electrical potential field can be found from equation by 
recognizing that a dipole element $\tau$ dS and a dipole element $J_i$dV must yield identical 
potential field expressions. Accordingly, for a uniform homogeneous conducting medium 
with conductivity $\sigma$,

\[ \Phi = \frac{1}{(4\pi \sigma)} \oint J_i \nabla(1/R)dV \]  \hspace{1cm} (12)

If the Laplacian of both sides of equation 12 is taken, then it can be shown that

\[ \nabla^2 \Phi = \nabla J_i/\sigma \]  \hspace{1cm} (13)

which is a form of Poisson's equation. This result may be reached in a somewhat different 
way if we assume the presence of a continuous volume source density of current, $I_v$. ($I_v$ is 
analogous to charge density as a source of electrostatic flux.) The presence of the source 
$I_v$ means that there must be a net outflow of current from any region containing $I_v$. More 
specifically, for a volume $V$ bounded by a surface $S$ the net outflow of current can be 
found by a surface integral of current density $J$ and this must be equal to the net source 
(volume integral of source density $I_v$); thus

\[ \oint J_i dS = \int I_v dV \]  \hspace{1cm} (14)

The outflow of current from a differential volume (outflow per unit volume) is 
evaluated by the divergence function, namely $\nabla J$, and this must equal $I_v$, according to 
equation 14 that is

\[ \nabla J = I_v \]  \hspace{1cm} (15)
Now, within the conducting medium. Ohm's law applies, namely,

\[ J = \sigma E \]  \hspace{1cm} (16)

where \( E \) is the electric field. Although all fields are time varying, at each instant of time they satisfy static equations to a condition described as quasi-static. In electrostatics, the electrical field may be derived as the (negative) gradient of a scalar potential function \( \Phi \). Consequently, we can rewrite equation 16 as

\[ J = -\sigma \nabla \Phi \]  \hspace{1cm} (17)

Now taking the divergence of eq 17. and substituting eq 15. Gives

\[ \nabla^2 \Phi = -I_v / \sigma \]  \hspace{1cm} (18)

which is an alternative expression of Poisson's equation for \( \Phi \). In fact, comparing the equations 13 and 18, it shows that

\[ I_v = \nabla \cdot J_I \]  \hspace{1cm} (19)

which identifies a source description volume current (outflow) density with a source described as a volume dipole density. By duality with electrostatics, the solution of equation in integral form is

\[ \Phi = \left(1/(4\pi\sigma)\right) \int \left(I_v/R\right) dV \]  \hspace{1cm} (20)

2.4. GENERATION OF ECG AND ECG WAVES

Impulse conduction through the heart [10, 12, 13] generates electrical currents that can be detected at the surface of the body. A recording of the electrical changes that accompany each cardiac cycle (heartbeat) is called an electrocardiogram [43], abbreviated either ECG or EKG. The ECG is a composite of action potentials produced by all the heart muscle fibers during each heartbeat [16]. The instrument used to record the changes is an electrocardiograph. In clinical practice, the ECG [27] is recorded by placing electrodes on the arms and legs (the limb leads) and at six positions on the chest. As the person lies still the electrocardiograph amplifies the heart's electrical activity and produces 12 different tracings from different combinations of limb and chest leads. This takes about a minute. Each limb and chest electrode records slightly different electrical activity because it is in a different position relative to the heart. By comparing these
records [28] with one another and with normal records it is possible to determine if the conduction pathway is normal, if the heart is enlarged, and if certain regions are damaged. Refer Figure 2.8 for Normal ECG Waveform (Lead II).

In a typical Lead II record, three clearly recognizable waves accompany each heartbeat. The first called the P wave is a small upward wave. It represents atrial depolarization, which spreads from the SA node through both atria. About 0.1 sec after the P wave begins the atria contracts. The second wave, called the QRS complex begins as a downward deflection, continues as a large upright, triangular wave, and ends as a downward wave. The QRS complex represents the onset of ventricular depolarization [29, 30] the spread of the wave of electrical excitation through the ventricles. Shortly after the QRS complex begins the ventricles start to contract. The third wave is a dome shaped upward deflection called the T wave. It indicates ventricular repolarization and occurs just before the ventricles start to relax. The T wave is smaller and more spread out than the QRS complex because repolarization occurs more slowly than depolarization. Usually repolarization of the atria is not evident in an ECG because it is buried in the larger QRS complex.

![Normal ECG Waveform (Lead II)](image_url)

**Figure 2.8** Normal ECG Waveform (Lead II).
In reading an electrocardiogram [31], it is important to note the size and timing of the waves. Larger P waves [32] for example, indicate enlargement of an atrium, as may occur in mitral stenosis. In this condition, the mitral valve narrows, blood backs up into the left atrium, and there is expansion of the atrial wall. An enlarged Q wave may indicate a myocardial infarction (heart attack). An enlarged R wave generally indicates enlarged ventricles. The P-Q (PR) interval is measured from the beginning of the ORS complex. It represents the conduction time from the beginning of atrial excitation to the beginning of ventricular excitation. The P-Q interval is the time required for an impulse to travel through the atria, atroventricular node and the remaining fibers of the conduction system. In coronary artery disease and rheumatic fever, scar tissue may form in the heart. As the impulse detours around scar tissue, the P-Q interval lengthens.

The S.T segment begins at the end of the S wave and ends at the beginning of the T wave. It represents the time when the ventricular contractile fibers are fully depolarized, during the plateau phase of the impulse. The ST segment is elevated (above the baseline) in acute myocardial infarction and depressed (below the baseline) when the heart muscle receives insufficient oxygen.

The T wave represents [33] ventricular repolarization. It is flatter than normal when the heart muscle is receiving insufficient oxygen, for example, in coronary artery disease. It may be elevated in hyperkalemia (increased blood K⁺ level).

Sometimes it is necessary to evaluate the heart's response to the stress of physical exercise. Such a test is called a stress electrocardiogram or stress test. It is based on the principle that narrowed coronary arteries may carry adequate oxygenated blood while a person is at rest, but during exercise will be unable to meet the heart's increased need for oxygen, creating changes that can be noted on an electrocardiogram.

2.4.1 THE NORMAL ECG WAVE

A typical EGG [10, 13, 28] tracing of a normal heartbeat consists of a P wave, a QRS complex and a T wave. A small U wave is not normally visible. Body is a volume conductor i.e. body fluids are good conductors of electricity; therefore, electrical changes occurring in the heart with each heart beat are conducted all over the body and can be picked up from the body surface. The record of the electrical fluctuations during cardiac cycle is called as
Electrocardiogram (ECG)

Thus, the ECG recorded at the surface of the body represents the resultant activity in the individual myocardial fiber.

The waves associated with electrical activity of the heart during each cardiac cycle are represented by letter P, Q, R, S and T.

I. 'P’ wave [32] is due to atrial depolarization and precedes atrial systole.

II. ‘Q’, ’R’ and ‘S’ waves together constitute the QRS complex and are due to ventricular depolarization. It precedes ventricular systole.

III. ‘T’ wave [33] is due to ventricular repolarisation. It coincides with closure of semilunar valves.

2.4.2 ECG RECORDING CONVENTIONS

1. ECG is recorded on mm square graph paper moving at a speed of 25 mm/sec. ‘X-axis’ represents the time therefore 1mm=0.04 sec (along the X-axis) ‘Y-axis’ represents the voltage therefore 1mm=0.1Mv (along the Y-axis).

2. Any deflection of the record above the baseline is regarded as positive deflection and any deflection below the baseline is regarded as negative deflection. No deflection from the baseline means the isoelectric line or the isoelectric segment.

3. Spread of the excitation wave i.e. depolarization process towards the electrode gives an upward deflection (positive deflection); and spread of excitation wave away from it causes a downward (negative) deflection.

2.4.3 WAVES ASSOCIATED WITH ECG

The waves associated with the electrical activity of the various parts of the heart tissue during each cardiac cycle are represented by letters P, Q, R, S, T and U.

Axis

The axis is the general direction of the electrical impulse through the heart. It is usually directed to the bottom left, although it can deviate to the right in very tall people and to the left in obesity. Extreme deviation is abnormal and indicates a bundle branch block, ventricular hypertrophy or (if to the right) pulmonary embolism. It also can
diagnose dextrocardia or a reversal of the direction in which the heart faces, but this condition is very rare and often has already been diagnosed by something else (such as a chest x-ray).

**P wave**

I. 1st wave of ECG of duration 0.1 sec; directed upwards, rounded or pointed.

II. It is due to atrial depolarization and represents the spread of impulses from ‘SA node’ to atrial muscles.

III. Its peak represents invasion of ‘AV node’ by excitation process.

IV. It occurs just before the ‘C’ wave of atrial pressure changes during cardiac cycle.

V. Its height is 0.5mV which represents the functional activity of atrial muscles.

VI. If bifurcated or absent, it is regarded as abnormal.

VII. The P wave is the electrical signature of the current that causes atrial contraction. Both the left and right atria contract simultaneously. Irregular or absent P waves may indicate arrhythmia [32]. Its relationship to QRS complexes determines the presence of a heart block.

**P-R segment**

Following the ‘P’ wave there is a brief isoelectric period of 0.04sec, called P-R segment.

**QRS Complex**

I. It is due to the ventricular depolarization.

II. It is completed just before the opening of the semilunar valves.

III. Atrial repolarisation activity merges with the QRS complex.

IV. QRS complex corresponds to the current that causes contraction of the left and right ventricles, which is much more forceful than that of the atria and involves more muscle mass, thus resulting in a greater ECG deflection.

Abnormalities in the QRS complex may indicate bundle branch block [34] (when wide), ventricular origin of tachycardia, ventricular hypertrophy or other ventricular
abnormalities. The complexes are often small in pericarditis.

‘Q’ wave

I. It is small negative deflection of height less than 0.2mV and duration less than 0.04 sec.

II. Beginning of ‘Q’ wave represents invasion of mid-portion of the interventricular septum by excitation process.

III. The Q wave, when present, represents the small horizontal (left to right) current as the action potential travels through the interventricular septum. Very wide and deep Q waves do not have a septal origin, but indicate myocardial infarction.

‘R’ wave

I. Prominent, positive wave.

II. Its upstroke coincides with the onset of ventricular systole.

III. It represents excitation process suddenly invadig both ventricles i.e. interventricular apex and major portion of both ventricles.

IV. Its height is directly proportional to the functional activity of ventricles.

‘S’ wave

I. Negative deflection which follows the ‘R’ wave.

II. It represents excitation of more basal part of ventricles.

III. The Rand S waves indicate contraction of the myocardium.

Thus the QRS complex extends from the beginning of ‘Q’ wave to end of ‘S’ wave with 0.08 to 0.12 sec duration and height 1.5 to 2mV duration

If its duration is more than 0.12 sec, it indicates heart block i.e. conduction in both or one of the branches of the bundle of His.

S-T segment

Following QRS complex there is a long isoelectric period which extends from the end of ‘S’ wave to the beginning of ‘T’ wave called S-T segment. Its duration is 0.04 to 0.08 sec. The ST segment connects the QRS complex and the T wave. It can be depressed in ischemia and elevated in myocardial infarction, and downslopes in digoxin use.
‘T’ wave

I. Rounded positive deflection of duration 0.27 sec and 0.5 mV height.

II. It represents ventricular repolarisation.

III. End of T-wave coincides with the closure of the semilunar valves.

IV. In most leads, the T wave is positive. Negative T waves can be signs of disease, although an inverted T wave is normal in V1 (and V2-3 in black people).

V. T wave abnormalities [35, 36, 37, 38, 39] may indicate electrolyte disturbance, such as hyperkalemia.

Iso-electric period

Following T-wave is a brief isoelectric period of 0.04 sec.

‘U’ wave

I. Rarely seen, as positive small [40] round wave of 0.08sec duration and 0.2 mV height.

II. It is due to slow repolarisation of papillary muscles.

PR interval

I. Interval from the beginning of ‘P’ wave to the beginning of Q or R wave (if Q is absent).

II. It represents atrial depolarization plus conduction time of bundle of His.

III. Normal duration 0.13 to 0.16 sec at a heart rate (HR) of 72/min, duration decreases with increase in HR.

IV. If duration is more than 0.2 sec indicates delayed conduction in bundle of His.

V. Duration of less than 0.13 sec indicates impulse has probably arisen in the AVN.

QT interval

I. Interval [41, 42, 43]from the beginning of ‘q’ wave to the end of T-wave, normal duration [44] of 0.40 to 0.43 sec.
II. It represents ventricular depolarization and repolarisation.

**ST interval**

I. (QT-QRS complex) i.e. end of ‘S’ wave to end of ‘T’ wave, normal duration 0.32 sec.

II. It represents ventricular repolarisation.

**TP segment**

I. Period from the end of ‘T’ wave to the beginning of ‘P’ wave of next cardiac cycles.

II. It represents polarized state of whole heart.

III. Its duration is inversely related to H.R. Normal is 0.2sec @ H.R 75/min.

**J point**

I. Point between ‘S’ wave and ST segment.

II. It is point of ‘no’ electrical activity.

### 2.5 ARRHYTHMIA

Arrhythmia (a-RITH-me-a) is a general term that refers to an abnormality or irregularity in the heart rhythm. Some physicians use the term dysrhythmia since that implies an abnormal rhythm, whereas arrhythmia implies no rhythm. An arrhythmia results when there is a disturbance in the conduction system of the heart. It may be due to either faulty production of electrical impulses as they pass through the system.

#### 2.5.1 MECHANISMS THAT CAUSE ABNORMAL IMPULSES

**Automaticity**

Automaticity refers to a cardiac muscle cell firing off an impulse on its own. Every cardiac cell has this potential: if it does not receive any impulses from elsewhere, its internal pacemaker" will fire off an impulse after a certain amount of time. A single specialized location in the atria, the sinoatrial node, has a higher automaticity (a faster pacemaker) than the rest of the heart, and therefore is usually the one to start the heartbeat.
Any part of the heart that initiates an impulse without waiting for the sinoatrial node is called an ectopic focus, and is by definition a pathological phenomenon. This may cause a single premature beat now and then, or, if the ectopic focus fires more often than the sinoatrial node, it can produce a sustained abnormal rhythm. Rhythms produced by an ectopic focus in the atria [45], or by the atrioventricular node, are the least dangerous [46] arrhythmias; but they can still produce a decrease in the heart's pumping efficiency [47], because the signal reaches the various parts of the heart muscle with slightly different timing than usual and causes a poorly coordinated contraction. Conditions that increase automaticity include sympathetic nervous system stimulation and hypoxia. The resulting heart rhythm depends on where the first signal begins: if it is the sinoatrial node, the rhythm remains normal but rapid; if it is an ectopic focus, many types of arrhythmia [48] can result.

Reentry

Reentrant arrhythmias occur when an electrical impulse travels in a circle within the heart, rather than moving outward and then stopping. Every cardiac cell is able to transmit impulses in every direction, but will only do so once within a short period of time. Normally the impulse spreads through the heart quickly enough that each cell will only respond once, but if conduction is abnormally slow in some areas, part of the impulse will arrive late and will be treated as a new impulse, which can then spread backward. Depending on the timing, this can produce a sustained abnormal rhythm [49], such as atrial flutter, a self-limiting burst of supraventricular tachycardia, or the dangerous ventricular tachycardia [50].

2.5.2. CAUSES OF ARRHYTHMIAS

* Factors [51] such as caffeine, nicotine, alcohol, anxiety, certain drugs, hyperthyroidism, potassium deficiency and certain heart diseases [52].

* When the heart’s natural pacemaker develops an abnormal rate or rhythm.

* When the normal conduction pathway is interrupted.

* When another part of the heart takes over as pacemaker.
* Coronary artery disease, high blood pressure, diabetes, smoking, excessive use of alcohol, drug abuse and stress.

* Certain substances, including prescription medications, dietary supplements and herbal remedies are known to cause arrhythmias in some people.

2.5.3 **CLASSIFICATION OF ARRHYTHMIAS**

Abnormal rhythms [1] occur as primary and secondary disorders. Primary disorders of rhythm reflect a basic, essential abnormality. Secondary disorders of rhythm only occur as a result of, and secondary to, a primary disorder.

The primary disorders of rhythm (33) may, in simplified form, be classified into two major categories:

1. Disturbances of impulse formation
2. Disturbances of impulse conduction.

Disturbances of impulse formation generate following arrhythmias:

**Sinus Rhythms**
- Sinus arrhythmia
- Sinus tachycardia
- Sinus bradycardia
- Ectopic atrial rhythms
- Atrial Extrasystoles
- Paroxysmal atrial tachycardia
- Atrial Fibrillation
- Atrial flutter
- AV nodal rhythms
- AV nodal extrasystoles
- Extrasystolic-paroxysmal- AV nodal tachycardia
- Idionodal tachycardia
Ventricular rhythms

Ventricular Extrasystoles

Extrasystolic ventricular tachycardia

Idioventricular Tachycardia

Ventricular flutter

Ventricular fibrillation

Ventricular parasystole

Disturbances of impulse conduction generates following arrhythmias

SA block

AV block

The Wolff- Parkinson white syndrome

Reciprocal rhythms

The secondary disorders of rhythm

Escape Rhythms

Atrial Escape

AV nodal Escape

Ventricular Escape

AV dissociation

Phasic aberrant ventricular conduction

2.5.4 FUNDAMENTAL DESCRIPTIVE PROPERTIES OF CARDIAC RHYTHMS

1 the rhythm [1] has a anatomical origin. The impulse may rise in the SA node, the atria, the AV node and ventricles.
The rhythm has a discharge sequence: normal inherent discharge, (as would occur normal sinus rhythm or an idioventricular escape rhythm), tachycardia, bradicardia extrasystole, parasystole, flutter or fibrillation.

The rhythm has a conduction sequence: for example, 2:1 AV block, complete AV block, 2:1 SA block.

**DUAL RHYTHMS**

A dual rhythm is a rhythm wherein two pacemakers concomitantly contribute to the rhythm of the heart. A dual rhythm is present in every form of AV dissociation – one pacemaker activating the atria and the other the ventricles. When this occurs, the descriptive properties of both the dissociated rhythms must be stated.

**Example**

1. Sinus rhythm with complete AV block and an idioventricular escape rhythm.
2. Ventricular tachycardia with AV nodal interference dissociation from normal sinus rhythm.

**ANALYSIS OF CARDIAC RHYTHMS**

On the basis of the aforementioned principles cardiac rhythm [53, 54] may be fundamentally analysed as follows:

1. The atrial deflexion is defined and analysed to determine whether it represents;
   
   (a) a normal P wave,
   
   (b) an ectopic or P deflexion,
   
   (c) a flutter –F-wave,
   
   (d) a chaotic fibrillation –f-wave.

2. The atrial rate is determined.

3. The regularity of the atrial rhythm is determined.

4. The relationship of the atrial deflexions to the QRS complexes is determined.

5. The QRS configuration is analysed.
THE GRAPHIC REPRESENTATION OF THE INTRACARDIAC CONDUCTION

Intracardiac conduction [1] may be conveniently and conventionally represented by means of a ‘ladder’ diagram. Refer figure 2.9. This is a graphic representation:

![Ladder Diagram](image)

**Figure 2.9 : Graphic representation of the use of the ‘ladder diagram in depicting arrhythmias’**.

The ordinate represents the anatomic levels of SA node, atria, AV node and ventricles; the abscissa represents time.

A illustrates normal conduction of a sinus impulse – black dot – arise in the SA node and is conducted relatively quickly through the atria (A), as reflected by the relatively steep slope. The impulse delayed within the AV node or junction (A-V), as reflected by the gradual slope, and is finally conducted relatively steep slope.

First degree AV block – prolonged P-R interval –a delay in conduction through the AV junction. This reflected by an even shallower slope.

B, Represents AV block – an interruption of conduction within the AV node.

A, B and C together would represent sinus rhythm complicated by 3:2 second degree AV block of the wenckebach type.

D, Represents phasic aberrant ventricular conduction –the abnormal intermittent intraventricular conduction of a supraventricular impulse.

E represents a ventricular impulse with retrograde AV conduction to the atria.

F represents a ventricular impulse, which is dissociated from a near-synchronous sinus impulse. Interference with consequent AV dissociation occurs within the AV node.

### 2.5.8 SOME IMPORTANT ARRHYTHMIAS

The major life threatening arrhythmias are explain as follows
Normal Sinus Rhythm

Normal sinus rhythm is reflected by the inscription of normal P waves at a rate [55] which ranges between 60 and 100 per minute. Normal sinus rhythm is usually associated with normal intraventricular conduction, and is thus reflected by the sequential inscription of P-QRS-T complexes.

Sinus Arrhythmia

Sinus arrhythmia is characterized by alternating periods of slow and rapid rates; [1, 55] it is due to an irregular fluctuating discharge of the SA node. The condition is most commonly associated with the phases of respiration – respiratory sinus arrhythmia. The period of faster rate occur towards the end of inspiration and the periods of slower rate towards the end of expiration. The mechanism is mediated by reflex stimulation of the vagus nerve from receptors in the lungs.

Diagnosis

The impulse rise from the SA node and the P waves are therefore normal; the subsequent course of the sinus impulse, on the absence of a conduction disturbance, is also normal, resulting in a normal P-R interval and QRS-T complex. The arrhythmias is thus characterized by normal P-QRS-T complexes, with alternating periods of gradually lengthening and gradually shortening P-P intervals [1, 55].

Sinus arrhythmia is accentuated by vagotonic procedures, such as digitalis administration and carotid sinus compression. It is abolished by vagolytic procedures, namely exercise, atropine and amyl nitrate.

Significance

Respiratory sinus arrhythmia [56] is a normal physiological; phenomena and is most marked in young persons. It may cause considerable irregularity of the pulse in childhood [57].

Sinus Tachycardia

Sinus tachycardia [58] occurs when the SA node discharges at a rate faster than 100 per minute in the adult. The normal resting rate in infant’s averages 120-130 beats per minute. Slowing gradually to reach the adult rate at puberty.
Diagnosis

Sinus tachycardia is, in the absence of a complicating conduction disturbance, characterized by normal P-QRS-T complexes, which are recorded in rapid succession. It varies with emotion, respiration and exercise. Vagotonic procedures, e.g. carotid sinus compression, result in slight but gradual slowing.

Significance

Sinus tachycardia [59] is a normal physiological response to exercise and emotion. A sinus tachycardia that persists at rest usually and expression of some underlying disorder. It occurs in anxiety states, thyrotoxicosis, toxemia, cardiac failure (as a result of an increased Bainbridge reflex) and acute carditis. It is a normal accompaniment of fever. The sinus rate will increase by 8 beats per minute for every one-degree increase in temperature. A diminution in oxygen saturation, as occurs at high altitudes or in association with congenital heart disease, will also cause a sinus tachycardia. Failure to develop sinus tachycardia with exercise or fever may be an expression of structural nodal disease – the so-called sick sinus syndrome. It may be caused by the administration of adrenaline, atropine, caffeine and amyl nitrate.

4 Sinus bradycardia

Sinus bradycardia, occurs when the SA node discharges at a rate slower than 60 per minute.

Diagnosis

Sinus bradycardia [60], in the absence of a complicating conduction distribution, is characterised by normal P-QRS-T complexes, which are recorded in slow succession. It is commonly associated with respiratory sinus arrhythmias.

Significance

Sinus bradycardia occurs as normal phenomena in athletes. Slowing of the sinus rate – at times to bradycardia levels – is the physiological response to sleep. Sinus bradycardia is accentuated by digitalis and Vagotonic procedures, such as carotid sinus compression. The rate quickens gradually with exercise, emotions and amyl nitrate.
Sinus bradycardia is associated with myxoedema [1], obstructive jaundice (the effect of direct action of the bile salts on the SA node) uraemia, (increased and persistence oculocardiac reflexes)

A common present-day cause of sinus bradycardia is the administration of beta-blocking agents [1].

5 Atrial extrasystoles

An atrial extrasystole [2] is due to the premature discharge of an ectopic atrial focus. It has the following characteristics.

Bizarre P wave

The discharge arises from an ectopic atrial focus, i.e. from a point other than the SA node. The activation front thus travels across the atria by unusual pathways, resulting in an abnormal or bizarre P’ wave – a P’ wave that is different from the sinus P wave and which may be pointed, notched, biphasic or inverted.

The ectopic impulse arises prematurely, i.e. in the diastolic period of the preceding sinus beat, and is thus recorded earlier than the next anticipated sinus P wave.

6 Heart Block

One serious arrhythmia [1] is called a heart block [61, 62]. Perhaps the most common blockage is in the atrioventricular (AV) node, which is the only path for impulses in the atria to reach the ventricles. This disturbance is called atrioventricular (AV) block. In first-degree AV block, P-Q (PR) interval [63]is prolonged, usually because conduction through the AV node is slower than normal. In second-degree AV block, some of the SA node impulses are not conducted through the AV node. This results in “dropped” beats, since excitation doesn’t reach the ventricles. In third-degree (complete) AV block, none of the SA node impulses get through the AV node. Autorhythmic cells in the atria and ventricles pace the upper and lower chambers independently. With complete AV block [21, 22], the ventricular contraction rate is less than 40 beats/min. Due to decreased cardiac output and diminished brain blood flow, patients may experience dizziness, unconsciousness or convulsions.
Ventricular Arrhythmias

When pacemakers in the SA node and in the AV junction both fail then the ventricular pacemakers, which have the lowest intrinsic rate, are able to take over. This usually occurs at a rate of 20-40 beats per minute. These pacemakers may also be manifest on occasions when the normal beat fails to reach the ventricles (e.g., in complete heart blocks); the result may be a ventricular escape [23] beat or a series of beats at the escape rhythm.

Premature ventricular complex (PVC) beats are very common in both health and disease. Activation tends to spread outward from the Ventricular [64, 65]ectopic site of activation mainly by cell-to-cell conduction. Consequently, the QRS duration is abnormally long (greater than 0.12s) and the QRS contour is abnormal [66]. There may also be lack of a discernible P wave preceding the QRS. Following the PVC is a compensatory pause. This simply refer to the fact that the basic sinus rate is unaffected by the PVC; consequently since the PVC prevents one sinus beat from reaching the ventricle when the next arrives a greater than PR interval will have elapsed.

Ventricular tachycardia

Ventricular tachycardia is a very serious arrhythmia [58, 67] arising most frequently in association with the occlusion of a coronary artery and/or electrolyte disturbance. It usually lies in the range 130 to 180 beats per minute. This arrhythmia arises from an enhanced automaticity of focal ventricular tissue or by reentry, which involves slow conduction and/or block that enables activation to return to an normal site beyond the refractory period permitting subsequent loop circuits to be made.

Ventricular Fibrillation and flutter

Definition

Ventricular fibrillation [65, 68] is a disorganized and chaotic activity of the heart, which results in irregular and deformed deflections of varying height, width and shape. This condition is terminal unless cardioverted. Ventricular flutter is a very rapid and regular ectopic ventricular discharge with an abnormal intraventricular conduction resulting in a wide, bizarre and sine like QRS complex fused with The T wave.
Causes

1. Coronary heart disease.
2. Drugs: Digitalis, adrenaline and anesthetics (79).
3. During cardiac surgery, due to hypoxia.
4. Hypothermia
5. Electric Shock

Diagnosis

Bizarre ventricular pattern of different shapes and sizes.

10 Ventricular Premature Contraction (VPC)

Another form of arrhythmia arises when a small region of the heart outside the pacemaker (an ectopic focus) becomes more excitable than normal, causing an occasional abnormal impulse to rise between normal impulses. As a wave of depolarization spreads outward from the ectopic focus, it causes a ventricular premature contraction (VPC). The contraction occurs early in diastole before the SA node is normally scheduled to discharge its impulse. VPCs may be relatively benign and may be caused by emotional stress, excessive intake of stimulants such as caffeine or nicotine and lack of speed. In other cases, the contractions may indicate an underlying pathology.

11 Supraventricular Premature Beats Or Extrasystoles (SVPB)

Definition

SVPB [21, 22] occurs due to a premature discharge of an ectopic focus, situated above the ventricles, either in the atrium or the AV node.

Characteristics

1. An atrial beat occurs prematurely so that a P wave is recorded earlier than the anticipated P wave.
2. Premature atrial excitation leads to an alternation in the P wave and the PR interval as the impulse travels along unusual pathways. The P wave may be upright, inverted or diphasic.
3. The premature beat usually initiates a ventricular complex which resembles the normal beat; hence the QRST complex of the premature beat resembles the QRST complex of the normal beat.

4. The compensatory pause is incomplete i.e. the sum total of the R-R intervals of the normal beat preceding and following a premature beat is not double the normal R-R interval.

Causes

1. Idiopathic
2. Heart diseases: coronary, rheumatic, thyrotoxicosis, diphtheria and hypertension.
3. Excessive use of tea, coffee, tobacco and alcohol.
4. Drugs: digitalis, amphetamine, adrenaline, thyroxine and emetine [49].
7. Manipulation of intrathoracic organs during surgical procedures on the heart or thoracic organs.

12 PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA (SVT)

Paroxysmal supraventricular tachycardia [1] is characterized by normal QRS but inverted P waves in leads II, III, and aVp along with a heart rate most frequently in the range 170-220 beats per minute. The most common mechanism involves patients with dual AV nodal pathways in which one has fast conduction and long refractory time while the other has slow conduction and rapid recovery. A premature atrial impulse will result in blocked conduction on the fast pathway and consequently a lengthened PR interval due to conduction on the, slow pathway. The impulse may then enter the fast pathway, conducting retrograde, and ultimately reexcite the slow link to establish a closed reentry loop.

Under unusual circumstances the normal rate may become so slow that a site in the AV junction may become an active source of a cardiac beat. This phenomenon is described as junctional escape.

Junctional escape is easy to recognize from its physiological properties. These include normal QRS complexes, a long pause preceding the escape arising from the
underlying bradycardia and retrograde activation of the atria resulting in an inverted P wave.

A situation sometimes arises when the atria are depolarized by impulse initiation from the SA node in the normal way but the ventricles are depolarized from a junctional pacemaker at a somewhat higher rate. Under these conditions retrograde conduction in the AV tissue is inhibited because of the preceding sinus impulses. The result is that the atria and ventricles beat completely independently.

Definition

SVT is a series of three or more SVPBs which may occur for a few beats or continuously for several hours or days. The last beat of the series is followed by a compensatory pause that is incomplete. Usually the rhythm is regular and at a rate of 160-220/min.

Mechanism

The exact mechanism of SVT is not known but two theories have been proposed:

1. Ectopic Mechanism: An ectopic focus, in either of the atria discharges regularly at the rate of 160-220/min. At this rate each atrial stimulus activated the ventricular muscle resulting in a regular ventricular rhythm.

2. Re-entry Mechanism: The tachycardia is initiated by an extra systole with a prolonged PR interval but there is normal conduction in the AV node and the ventricles. The stimulus reenters the bundle of his in a retrograde direction and stimulated non-refractory sited in the bundle. It is then propagated in a normal anterograde direction which results in a second ventricular capture. Perpetuation of the stimulus along this pathway causes SVT.

Diagnosis

SVT (60) is a continuous run of SVPBs so that each P wave is followed by a QRS complex.

The spread of the impulse through the atrial muscles occurs more slowly than the normal sinus beat or SVPB. Hence the PR interval is prolonged and the P wave may be obscured by the preceding QRS complex simulating junctional tachycardia.
Causes

Same as SVPB.

Significance

SVT may last for a few seconds to several days. It is usually benign and if without an underlying cause does not reduce life expectancy. Persistence of SVT in a patient with organic heart disease may lead to cardiac failure and coronary insufficiency. Persistence for a very long period, even in a normal individual, may cause cardiac failure.

13  Ventricular Premature beat or Extrasystole (VPB)

Definition

VPB occurs due to premature discharge [21, 22] of an ectopic focus in the ventricles.

Characteristics

1. The beat arises prematurely.

2. since the impulse originates in the ventricles and does not activate the atria, the P wave is absent.

3. The QRS complex is wide, bizarre and tall, with t waves in the opposite direction as the major deflection of the QRS complex i.e. if the R wave is prominent, the T wave is inverted and if the S wave is prominent, the T wave is upright.

4. The compensatory pause is complete because VPB does not depolarize the SA node. The impulse from the SA node following a VPB does not activate the ventricles as they will be in the refractory period. The ventricles will respond only to the next sinus impulse and hence the interval between the two sinus beats preceding and following the VPB will be exactly twice the normal interval between two sinus beats.

Types

1. Single isolated extrasystole [21].
2. Bigeminy or coupling: The extrasystole occurs regularly after every sinus beat [71, 72, 73, 74, 75]. If it occurs every second sinus beat it is called trigeminy.

3. In salvos: Two or more extrasystoles [21, 22] occur in succession. This may lead to paroxysmal tachycardia.

4. Interpolated: The extrasystole occurs in between two normal beats without any compensatory pause.

5. Multifocal: The extrasystoles arise from different foci, so that the morphology of the extrasystole varies in the same lead.

6. Reciprocal beat: The extrasystole spreads from the AV node to the ventricles and upwards into the atria. When it reaches the AV node it reverses its direction and reenters the ventricles causing a second ventricular contraction. Thus two QRS complexes separated by a nodal P waves are recorded.

7. Parasystole: The impulse simultaneously arises from the normal pacemaker and the ectopic focus. The two rhythms occur regularly but independent of each other so that the coupling distance between the ectopic focus and the previous sinus beat varies. At times, a fusion or summation beat occurs, the morphology of which is a combination of the sinus and the ectopic beat.

8. Blocked atrial premature beat: The P wave occurs during the refractory period of the AV node and the ventricles and hence these beats are blocked. However, the early P wave alters the morphology of the preceding T waves.

Significance

An extrasystole can occur in a normal person without any lesion of the heart. Usually it is benign and of no significance. However it is significant if:

1. It occurs for the first time after the age of 40
2. It is associated with a heart lesion.
3. It is multifocal.
4. It occurs more than 5 times per minute.
5. There is R on T phenomenon.
6. It occurs in salvos of 2 or more.
7. It occurs following exercise.
Causes

Same as VPB.

14 Paroxysmal Ventricular Tachycardia (VT)

Definition

VT is a series of three or more VPBs which may occur for a few beats or continuously for several hours or days. The last beat of the series is followed by a compensatory pause that is complete. Usually the rhythm is regular at 160-220/min.

Diagnosis

VT [50] is a continuous run of VPBs with QRS complexes smoothly merging with the ST segment and T waves giving an appearance of large wide undulations which are irregular.

Causes

Same as SVBP.

15 Atrial Flutter And Fibrillation

Definition

Atrial flutter [76] is a rapid and regular contraction of the heart at a rate of about 220-350/min. Varying degrees of AV block lead to a much slower ventricular rate. The P waves of the atrial flutter have a saw-tooth appearance and are called flutter waves.

Atrial fibrillation [77] is a chaotic rhythm of the atria which causes small twitches of the atrial myocardium instead of an active atrial contraction which normally aids ventricular filling phase of the ventricular diastole.

Mechanism

AV conduction of the atrial impulse depends upon the atrial rate. With atrial rates 160-220/min, (atrial tachycardia), vagal stimulation leads to asystole followed by reversion to sinus rhythm. With rates 220-350/min. (atrial flutter), varying degrees of AV block occur without a change in atrial rate [76]. With rates above 350/min. (atrial fibrillation), the atrium cannot respond completely to each stimulus and a chaotic disturbance occurs. Majority of the atrial impulses reaching the AV node are blocked. An
occasional impulse occurs during the non-refractory period and is conducted to the ventricles leading to an irregular ventricular rhythm.

With atrial rate less than 200/min. P waves of the atrial flutter resemble those of SVT. As the atrial rate increases, a more prominent T wave in a direction opposite to the P wave appears. With increasing atrial rates the T wave amplitude increases and at rate 300/min. It will be equal in amplitude to the P wave resulting in a saw-tooth appearance.

Causes

<table>
<thead>
<tr>
<th>Common</th>
<th>Uncommon</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Rheumatic fever</td>
<td>1. Constrictive pericarditis</td>
</tr>
<tr>
<td>2. Coronary heart disease</td>
<td>2. Cor-pulmonale</td>
</tr>
<tr>
<td>3. Thyrotoxicosis</td>
<td>3. Bronchogenic carcinoma</td>
</tr>
<tr>
<td>5. Drugs: digitalis, adrenaline, emetine [78]</td>
<td>5. Hypertension</td>
</tr>
</tbody>
</table>

Diagnosis

Atrial Flutter

1. Fast atrial rate of 220-350 / min. with ventricular rate half or one forth of the atrial rate

2. P waves replaced by flutter waves.

3. Ventricular rhythm usually regular, unless there is a changing AV block.

Atrial Fibrillation

1. Irregularly irregular ventricular rhythm [79].

2. P waves replaced by fibrillation waves.

At times the rhythm may alternate between flutter and fibrillation and a precise
difference cannot be discerned. This is called ‘flutter fibrillation’.

Chaotic Atrial Rhythm:

When there are more than three morphologically different P waves [80, 81] that
activate the ventricles, it is called chaotic atrial rhythm. It resembles wondering
pacemaker but unlike the latter there is no dominant P wave. This rhythm [82, 83] is seen
in elderly persons with chronic obstructive pulmonary disease or coronary artery disease.

2.5.6. SUMMARY OF COMMONLY OCCURRING LIFE THREATENING
ARRHYTHMIAS

Refer figure 2.10 for Arrhythmia Waveforms (Lead II)

The commonly occurring life threatening arrhythmias are explained below in
brief:

Asystole

It is a condition in which there is lack of conduction for an extended duration.

Fusion beat

It is a parasystolic condition in which two pacemakers in heart (Say SA and AV)
discharge at their own inherent rate, occasionally causing simultaneous invasion of
ventricular musculature, each activating part of ventricles. The resulting QRS complex
has a configuration intermediate below ‘pure’ sinus beat and ‘pure’ ventricular beat. The
resulting summation complex is known as fusion beat [84, 85].

Missed beat

In second degree AV block, transmission through the conducting system becomes
difficult until it falls completely and a beat is ‘dropped’. The condition is noted if R-R
interval is greater than 2 R-R

Atrial fibrillation

In this excitation and recovery of atria are disorganized and chaotic.

Ventricular fibrillation

Unconditional, chaotic, uncoordinated fluttering of ventricles [20, 86] is called
ventricular fibrillation. No defined P-QRS-T signal is observed.
Bigeminy

Presence of ventricular premature beat [87, 88, 89, 90] between alternate normal beat. The VPB occurs very early i.e. AV node still partially refractory. Following sinus beat occurs on time but relatively late in relationship to the extrasystole.

Multifocal ventricular extrasystoles

Extrasystole that arise from different foci and consequently give rise to different QRS complexes are termed as multifocal ventricular extrasystoles. Usually these are indication of serious myocardial disease.

R on T wave

Ventricular extrasystoles may rarely occur with very short coupling interval and will consequently coincide with and be superimposed upon or near the apex or the distal limb of preceding T wave. These are more prone to ventricular fibrillation [91] occurring in context of acute myocardial infarction.

Ventricular Tachycardia

It is due to rapid discharge of an ectopic ventricular pacemaking focus. It may be defined as series of three or more consecutive ventricular ectopic beats.

Heart Block

It is a condition in which the conduction of the pacing signal is either delayed or completely blocked resulting in inability to activate the chamber or part being paced. This leads to absence of the characteristic waveform of the chamber i.e. if there is complete AV block, R wave is missing.

VPB1

In this condition there is premature ventricular contraction arising in diastolic period of the preceding sinus beat followed by compensatory pause.

VPB2

Ventricular extrasystole arising from an ectopic focus followed by incomplete compensatory pause.
<table>
<thead>
<tr>
<th>Rhythms</th>
<th>Diagram</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. VPB 1</td>
<td><img src="image1" alt="Waveform" /></td>
</tr>
<tr>
<td>B. Multifocal Run</td>
<td><img src="image2" alt="Waveform" /></td>
</tr>
<tr>
<td>C. Missed Beat</td>
<td><img src="image3" alt="Waveform" /></td>
</tr>
<tr>
<td>D. Fusion Beat</td>
<td><img src="image4" alt="Waveform" /></td>
</tr>
<tr>
<td>E. VPB 2</td>
<td><img src="image5" alt="Waveform" /></td>
</tr>
<tr>
<td>F. Couplet (Pair)</td>
<td><img src="image6" alt="Waveform" /></td>
</tr>
<tr>
<td>G. Run</td>
<td><img src="image7" alt="Waveform" /></td>
</tr>
<tr>
<td>H. R on T wave</td>
<td><img src="image8" alt="Waveform" /></td>
</tr>
<tr>
<td>I. Asystole</td>
<td><img src="image9" alt="Waveform" /></td>
</tr>
<tr>
<td>J. Ventricular Fibrillation</td>
<td><img src="image10" alt="Waveform" /></td>
</tr>
<tr>
<td>K. Atrial Fibrillation</td>
<td><img src="image11" alt="Waveform" /></td>
</tr>
<tr>
<td>L. Heart Block</td>
<td><img src="image12" alt="Waveform" /></td>
</tr>
<tr>
<td>M. Normal Rhythm</td>
<td><img src="image13" alt="Waveform" /></td>
</tr>
<tr>
<td>N. Paced Rhythm</td>
<td><img src="image14" alt="Waveform" /></td>
</tr>
<tr>
<td>O. Tachycardia</td>
<td><img src="image15" alt="Waveform" /></td>
</tr>
<tr>
<td>P. Bradycardia</td>
<td><img src="image16" alt="Waveform" /></td>
</tr>
<tr>
<td>Q. Ventricular Tachycardia</td>
<td><img src="image17" alt="Waveform" /></td>
</tr>
<tr>
<td>R. Bigeminy</td>
<td><img src="image18" alt="Waveform" /></td>
</tr>
</tbody>
</table>

Figure 2.10: Arrhythmia Waveforms (Lead II)
2.6 CONCLUSION

A survey of various life threatening arrhythmias is done and taking the views of many cardiologists and cardiac surgeons, out of all, seventeen arrhythmias and a normal ECG waveform his selected for simulation. The literature provided in this chapter shows that there is a need of a various simulation programs for arrhythmias as the ECG signal shows changes with the age, health, drug intake by patient, and patient suffering from respiratory diseases.