Chapter 4
Physiology and functioning of human heart

4.1 Functional description and related anatomy.

The heart, shown in Figure 4-1, can be simplified functionally into two separate pumps- a right heart that pumps blood through the lungs, and a left heart that pumps blood through the peripheral organs. In turn, each of these hearts is a pulsatile, two-chamber pump composed of an atrium and a ventricle. Each atrium is a weak primer pump for the ventricle, helping to move blood into the ventricle. The ventricles then supply the main pumping force that propels the blood either

(1) Through the pulmonary circulation by the right ventricle or
(2) Through the peripheral circulation by the left ventricle

Special mechanisms in the heart cause a continuing succession of heart contractions called cardiac rhythmicity, transmitting action potentials throughout the heart muscle to cause the heart’s rhythmical beat. [38]

4.2 Types of Cardiac Muscle
The heart is composed of three major types of cardiac muscles atrial muscle and ventricular muscle, special excitatory muscle and conductive fiber muscles. The
atrial and ventricular type of muscles contract for large intensity and duration. The excitatory and conductive fibers contract with less contractile force because they contain few contractile fibrils. They exhibit either automatic rhythmical electrical discharge in the form of action potentials or conduction of the action potentials through the heart, providing an excitatory system that controls the rhythmical beating of the heart. [38]

### 4.3 Action Potentials in Cardiac Muscle

The action potential recorded in a ventricular muscle fiber, shown in Figure 8-2. Average pressure is about 105 millivolts, which indicated that the intracellular potential changes from -85 millivolts, about +20 millivolts, during each beat. After the initial spike, the membrane remains depolarized for about 0.2 second, exhibiting a plateau as shown followed at the end of the plateau by abrupt repolarization. The presence of this plateau in the action potential causes ventricular contraction.

![Figure 4.2 Action potential of ventricles. Figure adapted from [38]](image)

In cardiac muscle, the action potential is caused by opening of two types of channels. The fast sodium channels and slow calcium channels, which are also called calcium-sodium channels. The Sodium channels are faster to open and close. The Calcium channels are slower to open and remain open for several tenths of a second. During this time, a large quantity of both calcium and sodium ions flows through these channels to the interior of the cardiac muscle fiber, and this maintains a prolonged period of depolarization, causing the plateau in the action potential. The calcium ions that enter during this plateau phase activate the muscle contractile process. Immediately after the onset of the action potential, the permeability of the cardiac muscle membrane for potassium ions decreases about fivefold. As a result, the decreased potassium permeability decreases the outflux of positively charged potassium ions during the action potential plateau and thereby prevents early return of the action potential voltage to its resting level. When the
slow calcium-sodium channels close at the end of 0.2 to 0.3 second and the influx of calcium and sodium ions ceases, the membrane permeability for potassium ions also increases rapidly. This rapid loss of potassium from the fiber immediately returns the membrane potential to its resting level, thus ending the action potential. [38]

### 4.4 Velocity of Signal Conduction in Cardiac Muscle

The velocity of conduction of the excitatory action potential signal along both atrial and ventricular muscle fibers is about 0.3 to 0.5 m/sec. The velocity of conduction in the specialized heart conductive system i.e. in the Purkinje fibers is as great as 4 m/sec. allowing reasonably rapid conduction of the excitatory signal to the different parts of the heart. [38]

#### Duration of Contraction

Cardiac muscle begins to contract a few milliseconds after the action potential begins and continues to contract until a few milliseconds after the action potential ends. Therefore, the duration of contraction of cardiac muscle is mainly a function of the duration of the action potential, including the plateau about 0.2 second in atrial muscle and 0.3 second in ventricular muscle. [38]

#### The Cardiac Cycle

Each cycle is initiated by spontaneous generation of an action potential in the sinus node located in the superior lateral wall of the right atrium near the opening of the superior vena cava. The action potential travels from here rapidly through both atria and then through the A-V bundle into the ventricles. Because of this special arrangement of the conducting system from the atria into the ventricles, there is a delay of more than 0.1 second during passage of the cardiac impulse from the atria into the ventricles. This allows the atria to contract ahead of ventricular contraction, thereby pumping blood into the ventricles before the strong ventricular contraction begins. Thus, the atria act as primer pumps for the ventricles, and the ventricles in turn provide the major source of power for moving blood through the body’s vascular system. The cardiac cycle starting from action potential and resulting into ECG signal is shown in Figure 4.2.

Events of the cardiac cycle for left ventricular function, showing changes in left atrial pressure, left ventricular pressure, aortic pressure are shown the figure- 4.3. The pressure variations in ventricles atria and aorta are plotted for one cardiac cycle. [38]
4.5 Relationship of the Electrocardiogram to the Cardiac Cycle

The electrocardiogram in Figure- 4.4 shows the P, Q, R, S, and T waves, which are electrical voltages generated by the heart and recorded by the electrocardiograph from the surface of the body. The P wave is caused by spread of depolarization through the atria, and this is followed by atrial contraction, that causes a slight rise in the atrial pressure curve immediately after the electrocardiographic P wave. About 0.16 second after the onset of the P wave, the QRS waves appear as a result of electrical depolarization of the ventricles, which initiates contraction of the ventricles and causes the ventricular pressure to increase, as also shown in the Figure 4.4. Therefore, the QRS complex begins slightly before the onset of ventricular systole. Finally, one observes the ventricular T wave in the electrocardiogram. This represents the stage of repolarization of the ventricles when the ventricular muscle fibers begin to relax. Therefore, the T wave occurs slightly before the end of ventricular contraction. [38]
4.5.1 Hormonal Control on SA node

Cardiac impulse through the heart originates from the sinus node. The rate at which the cardiac impulse is generated depends upon many different parameters. Among them major hormonal stimulations are from sympathetic and parasympathetic sites.

4.5.2 Effects of Parasympathetic Stimulation

Hormone acetylcholine released at the nerve endings causes parasympathetic nerve stimulation. This hormone has two major effects on the heart. It decreases the rate of rhythm of the sinus node and it decreases the excitability of the A-V junction fibers between the atrial muscles and the A-V node, thereby slowing transmission of the cardiac impulse into the ventricles. Weak to moderate vagal stimulation slows the rate of heart pumping, often to as little as one half normal. Strong stimulation of the vagal input can stop completely the rhythmical excitation by the sinus node or block completely transmission of the cardiac impulse from the atria into the ventricles through the A-V node.

In either case, rhythmical excitatory signals are no longer transmitted into the ventricles. The ventricles stop beating for 5 to 20 seconds, but then some point in the Purkinje fibers, usually in the ventricular septal portion of the A-V bundle, develops a rhythm of its own and causes ventricular contraction at a rate of 15 to 40 beats per minute. This phenomenon is called ventricular escape. [11]

4.5.3 Mechanism of the Parasympathetic Effects

The acetylcholine released at the vagal nerve endings greatly increases the permeability of the fiber membranes to potassium ions, which allows rapid leakage of potassium out of the conductive fibers. This causes increased negativity inside the fibers, an effect called hyperpolarization, which makes this excitable tissue much less excitable. In the sinus node, the state of hyperpolarization decreases the resting potential of the sinus nodal fibers to a level considerably more negative than usual, to -65 to -75 millivolts rather than the normal level of -55 to -60 millivolts. Therefore, the initial rise of the sinus nodal membrane potential caused by inward sodium and calcium leakage requires much longer duration to reach the threshold potential for excitation. This slows down the rate of rhythmicity of these nodal fibers by a great extent. If the vagal stimulation is strong enough, it is possible to stop entirely the rhythmical self-excitation of this node. In the A-V
node, a state of hyperpolarization caused by vagal stimulation makes it difficult for the small atrial fibers entering the node to generate enough electricity to excite the nodal fibers. Therefore, the safety factor for transmission of the cardiac impulse through the transitional fibers into the A-V nodal fibers decreases. A moderate decrease simply delays conduction of the impulse, but a large decrease blocks conduction entirely.

4.5.4 Effect of Sympathetic Stimulation

Sympathetic stimulation causes essentially the opposite effects on the heart to that caused by vagal stimulation. It increases the rate of sinus nodal discharge and it increases the rate of conduction as well as the level of excitability. It also increases the force of atrial and ventricular contraction of the cardiac musculature. Sympathetic stimulation increases the overall activity of the heart. Maximal stimulation can almost triple the frequency of heartbeat and can increase the strength of heart contraction as much as two fold.

4.5.5 Mechanism of the Sympathetic Effect

Stimulation of the sympathetic nerves releases the hormone norepinephrine at the sympathetic nerve endings. The precise mechanism by which this hormone acts on cardiac muscle fibers is not very clear, but the belief is that it increases the permeability of the fiber membrane to sodium and calcium ions. In the sinus node, an increase of sodium-calcium permeability causes a more positive resting potential and also causes increased rate of upward drift of the diastolic membrane potential toward the threshold level for self excitation, thus accelerating self excitation and therefore, increasing the heart rate. In the A-V node and A-V bundles, increased sodium-calcium permeability makes it easier for the action potential to excite each succeeding portion of the conducting fiber bundles, thereby decreasing the conduction time from the atria to the ventricles. The increase in permeability to calcium ions is at least partially responsible for the increase in contractile strength of the cardiac muscle under the influence of sympathetic stimulation, because calcium ions play a powerful role in exciting the contractile process of the myofibrils.

Inhibition of the sympathetic nerves to the heart can decrease cardiac pumping to a moderate extent. Under normal conditions, the sympathetic nerve fibers to the heart discharge continuously at a slow rate that maintains pumping at about 30 per cent above when there is no sympathetic stimulation. Therefore, when the activity of the sympathetic nervous system is depressed below normal, this decreases both
heart rate and strength of ventricular muscle contraction, thereby decreasing the level of cardiac pumping as much as 30 per cent below normal. [38], [69] and [72]

4.5.6 Sympathetic and parasympathetic Stimulation and it’s effect on heart

1. Strong stimulation of the parasympathetic nerve fibers in the vagal nerves to the heart can stop the heartbeat for a few seconds, but then the heart usually “escapes” and beats at a rate of 20 to 40 beats per minute as long as the parasympathetic stimulation continues.
2. Strong vagal stimulation can decrease the strength of heart muscle contraction by 20 to 30 per cent.
3. The vagal fibers are distributed mainly to the atria and not much to the ventricles, where the power contraction of the heart occurs. This explains the effect of vagal stimulation mainly to decrease heart rate rather than to decrease greatly the strength of heart contraction.
4. The decrease in heart rate combined with a slight decrease in heart contraction strength can decrease ventricular pumping 50 per cent or more.

4.5.7 Effect of ions on Heart Function

Potassium ions:
Excess potassium in the extracellular fluids causes the heart to become dilated and flaccid and also slows the heart rate. Large quantities also can block conduction of the cardiac impulse from the atria to the ventricles through the A-V bundle. Elevation of potassium concentration to only 8 to 12 m Eq/L two to three times the normal value can cause weakness of the heart and abnormal rhythm that this can cause death. These effects result partially from the fact that a high potassium concentration in the extracellular fluids decreases the resting membrane potential in the cardiac muscle fibers. As the membrane potential decreases, the intensity of the action potential also decreases, which makes contraction of the heart progressively weaker. [38]

Calcium Ions:
An excess of calcium ions causes effects almost exactly opposite to those of potassium ions, causing the heart to go toward spastic contraction. This is caused by a direct effect of calcium ions to initiate the cardiac contractile process, as explained earlier in the chapter. Conversely, deficiency of calcium ions causes cardiac flaccidity, similar to the effect of high potassium. Fortunately, however, calcium ion levels in the blood normally are regulated within a very narrow range. Therefore, cardiac effects of abnormal calcium concentrations are seldom of clinical concern. [38]