Anesthetic Management of Patients with Cardiac Pacemakers Undergoing Noncardiac Surgery

An Essay
Submitted For Partial Fulfillment of Master
Degree in **Anesthesiology**

Presented By
Ahmed Monier Ahmed Eldemerdash
M.B., B.Ch, 2005
Faculty of Medicine, Ain Shams University

Under supervision of
Prof. Dr. Laila Ali Al-Sayed Al-Kafrawy

Professor of Anesthesiology and Intensive Care
Ain Shams University - Faculty of Medicine

Dr. Hany Abd El-Fattah Sayed Ahmed

Assistant Professor of Anesthesiology and Intensive Care
Faculty of Medicine - Ain Shams University

Dr. Magdy Shehata Metias

Lecturer of anesthesia and Intensive Care
Ain Shams University - Faculty of Medicine

Faculty of Medicine
Ain Shams University
2010

Introduction and Aim of The Work

Patients with cardiac disease presenting for noncardiac surgery pose a considerable challenge to the anesthesiologists. With the availability of better medical facility and sophisticated diagnostic methods, many patients especially of the elderly age group, are detected to have electrophysiological disorders. Pacemakers are being used with greater frequency for both conduction and arrhythmia problems in such patients ***(Atlee and Bernstein, 2001).***

These patients may require one or more surgical procedures after receiving the pacemaker ***(Levine et al., 1986).***

The initial pacing system consisted of a single lead asynchronous pacemaker, which paced the heart at a fixed rate ***(Mehta et al., 1998).***

Care of the pacemaker during surgery as well as understanding its anesthetic implications is crucial in the management of these patients. The perioperative management of patients with permanent pacemaker undergoing noncardiac surgery will be discussed in this essay ***(Hayes and Zipes, 2001).***

Anatomy of the Heart

The heart is a cone-shaped organ that lies obliquely in the mediastinum within the pericardial sac, suspended by the large vessels. Its four chambers, the left & right atria, and the left and right ventricles are demarcated on its surface by coronary and interventricular sulci. Its square base lies posteriorly and is formed largely of the left atrium, which receives the four pulmonary veins at each corner. The inferior vena cava enters the right atrium at its right postero-inferior angle, which rests on the central tendon of the diaphragm, the superior vena cava enters the upper part of the atrium **(**Fig. 1**)** ***(Craven J., 2003).***

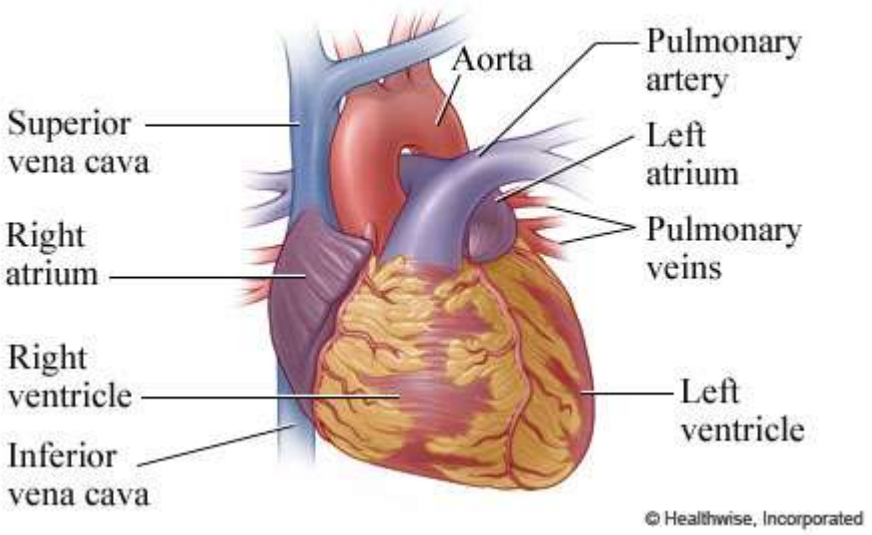

Figure 1: External Anatomy of the Heart ***(Craven J., 2003)***

The right atrium is a thin-walled chamber. It forms the right border of the heart and has a small projection, the right auricle, overlapping the origin of the ascending aorta. Its posterior wall behind the caval openings is smooth and marked by a shallow depression, the fossa ovalis, which is the remnant of the fetal opening **(**Fig. 2**).**

The right ventricle is a thick-walled chamber that projects to the left of the right atrium forming part of the heart's anterior and inferior surfaces. The interventricular septum separates the right from the left ventricle and bulges into the right cavity, making it crescentic in cross-section. The atrioventricular (AV) orifice lies postero-inferiorly guarded by the tricuspid valve, comprising three cusps each consisting of thin fibrous tissue covered on both sides by endocardium. The atrial surfaces of the cusps are smooth and the ventricular surfaces are rough and anchored to the ventricular walls by tendinous cords arising directly from the septum or from two papillary muscles. The muscles tighten the cords during ventricular contraction and thereby prevent eversion of the cusps into the atrial cavity. The pulmonary orifice is a fibrous ring lying at the upper end of the ventricle, the infundibulum. A valve, comprising three semilunar valvules lies at the entrance to the orifice ***(Craven J., 2003).***

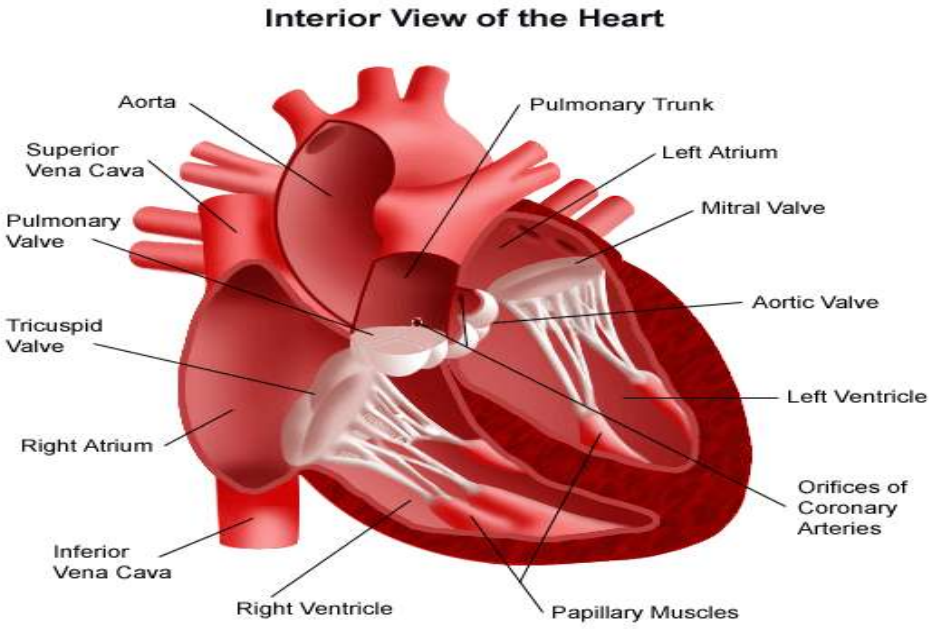

Figure 2: Interior view of the Heart ***(Craven J., 2003)***

The left atrium is a thin-walled chamber that lies behind the right ventricle and forms most of the base of the heart. Superiorly, the left auricle, a small projection, overlies the origin of the pulmonary trunk. The four pulmonary veins enter each corner of the posterior wall. Their orifices possess no valves. The left AV orifice lies in the anterior wall. The posterior wall of the left atrium is separated from the oesophagus and the left bronchus by the pericardial sac.

The left ventricle extends forwards and to the left from the left atrium, and lies mainly behind the right ventricle. It forms the apex, the left border and surface, and part of the anterior and inferior surfaces of the heart. Its walls are thick and surround a conical cavity with two orifices, the left AV orifice posteriorly and the aortic orifice superiorly.

The left AV orifice is guarded by the mitral (bicuspid) valve, the two cusps of which are attached to a fibrous ring surrounding the orifice. The free margins of the cusp are anchored to papillary muscles on the ventricular wall by tendinous cords. The aortic orifice is a fibrous ring guarded by a valve of three semilunar valvules similar to the pulmonary valve, The thick interventricular septum is marked on the surface by anterior and posterior interventricular sulci; the right ventricle lies anterior to the left ventricle ***(Craven J., 2003).***

Blood supply

Arteries

The right coronary artery arises from the anterior aortic sinus just above the aortic valve, and descends in the right coronary sulcus on the anterior surface and crosses the posterior surface of the heart to anastomose with the left coronary artery, it supplies atrial and ventricular branches, and a larger posterior interventricular artery, which anastomoses with the anterior interventricular artery.

The left coronary artery arises from the left posterior aortic sinus and, in the left coronary sulcus supplies atrial, ventricular branches, and the important anastomotic vessels, the anterior interventricular artery and the circumflex artery. The latter anastomoses with the right coronary artery. Generally, the right ventricle is supplied by the right coronary artery, the left ventricle by the left coronary artery and the interventricular septum by both **(**Fig. 3**)** ***(Craven J., 2003).***

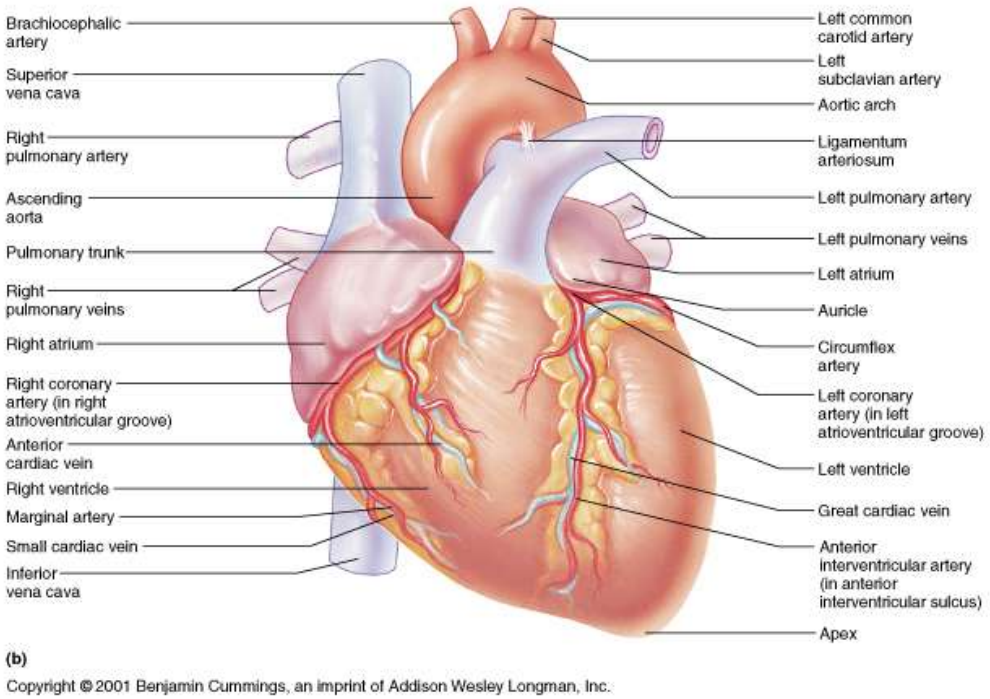

Figure 3: Blood supply of the Heart ***(Craven J., 2003)***

Veins

Veins accompany the major arteries and most drain via the coronary sinus, which is formed at the left border of the heart as a continuation of the great cardiac vein. It lies in the posterior coronary sulcus and enters the right atrium above and posterior to the orifice of the inferior vena cava **(**Fig. 3**)** ***(Craven J., 2003).***

Nerve supply

The nerve supply is by vagus and sympathetic fibers through the cardiac plexus. The fibers are distributed with the coronary vessels. Parasympathetic ganglion cells are found in the heart walls. Sensory fibers subserving reflex activity pass in the vagus and pain fibers in the spinal nerves (T1 - T3) ***(Craven J., 2003).***

Cardiac conducting system

The conducting system of the heart is formed of specialized heart muscle cells. It comprises the sinoatrial node, the AV node, the AV bundle (of His), its right and left branches, and a terminal subendocardial plexus of Purkinje fibers. The conducting system initiates the muscle contractions of the cardiac cycle and controls its regularity **(**Fig. 4**)** ***(Craven J., 2003).***

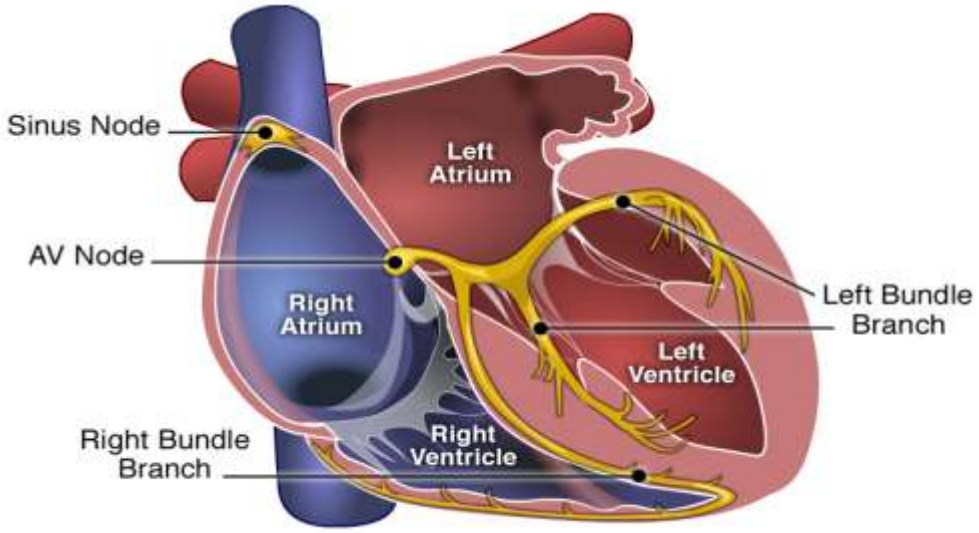

Figure 4: Conduction system of the Heart ***(Craven J., 2003)***

The sinus node (SA node) is a small, flattened, ellipsoid strip of specialized muscle about 3 millimeters wide, 15 millimeters long, and l millimeter thick, it is located in sulcus terminalis in the superior lateral wall of the right atrium immediately below and slightly lateral to the opening of the superior vena cava. The fibers of this node have almost no contractile filaments and are each 3 to 5 micrometers in diameter, in contrast to a diameter of 10 to 15 micrometers for the surrounding atrial muscle fibers. The sinus fibers connect directly with the atrial fibers, so that any action potential that begins in the sinus node spreads immediately into the atria. The Atrioventricular node (AV node) is located in the posterior septal wall of the right atrium immediately behind the tricuspid valve and adjacent to the opening of the coronary sinus ***(Woods et al., 1982).***

There are 3 bundles of atrial fibers that contain purkinje type fibers and connect the SA node to the AV node: the anterior internodal tract of Bachman, the middle internodal tract of Wenckebach, and the posterior internodal tract of Thorel. The AV node is normally the only conducting pathway between the atria and ventricles. It is continuous with the bundle of His, which gives off a left bundle branch at the top of the interventricular septum and continues as the right bundle branch. The left bundle branch divides into an anterior fascicle and a posterior fascicle. The branches and fascicles run subendocardially down either side of the septum and come into contact with the purkinje system, whose fibers spread to all parts of the ventricular myocardial ***(Woods et al., 1982).***

The atrial muscle fibers are separated from those of the ventricles by a fibrous tissue ring ***(Anderson et al., 1981).***

The SA node develops from structures on the right side of the embryo and the AV node from structures on the left. This is why in the adult the right vagus is distributed mainly to the SA node and the left vagus mainly to the AV node.

Similarly, the sympathetic innervation on the right side is distributed primarily to the SA node and the sympathetic innervation on the left side primarily to the AV node. On each side, most sympathetic fibers come from the stellate ganglion ***(Ganong, 1983).***

Cardiac Physiology

The heart is endowed with a specialized system for generating rhythmical impulses to cause rhythmical contraction of the heart muscle and conducting these impulses rapidly throughout the heart. The structures that make up the conduction system are the sinoatrial node (SA node), the internodal atrial pathways, the atrioventricular node (AV node), the bundle of his and its branches and the purkinje system. The various parts of the conduction system, under abnormal conditions and parts of the myocardium are capable of spontaneous discharge. However, the SA node normally discharges most rapidly, depolarization spreading from it to the other regions before they discharge spontaneously. The SA node is therefore the normal cardiac pacemaker, its rate of discharge determining the rate at which the heart beats ***(Berne and Levy, 1981).***

When this system functions normally, the atria contract about one sixth of a second ahead of ventricular contraction, which allows extra filling of the ventricles before they pump the blood through the lungs and the peripheral circulation. Another special importance of the system is that it allows all portions of the ventricles to contract almost simultaneous which is essential for effective pressure generation in the ventricular chamber. This rhythmical and conductive system of the heart is susceptible to damage by heart disease, especially by ischemia of the heart tissues resulting from poor coronary blood flow. The consequence is often a bizarre heart rhythm or abnormal sequence of contraction of the heart chambers, and the pumping effectiveness of the heart is often

affected severely, even to the extent of causing death ***(Berne and Levy, 1981).***

Automatic electrical rhythmicity of the sinus fibers

Many cardiac fibers have the capability of self-excitation, a process that can cause automatic rhythmical discharge and contraction. This is especially true of the fibers of the heart's specialized conducting system the portion of this system that displays self-excitation to the greatest extent is the fibers of the sinus node. For this reason, the sinus node ordinarily controls the rate of beat of the entire heart ***(Wit et al., 1974).***

Mechanism of sinus nodal rhythmicity

The potential of the sinus nodal fiber between discharges has a negativity of only -55 to -60 millivolts in comparison with -85 to -90 millivolts for the ventricular muscle fiber. The cause of this reduced negativity is that the cell membranes of the sinus fibers are naturally leaky to sodium ions ***(Levitan and Kuzcmurek, 1991).***

There are three types of membrane ion channels play important roles in causing the voltage changes of the action potential. They are fast sodium channels, slow calcium-sodium channels, and potassium channel. Opening of the fast sodium channels for a few tenths of a second is responsible for the rapid spike-like onset of the action potential observed in ventricular muscle because of rapid influx of positive sodium ions to the interior of the fiber. Then the plateau of the ventricular action potential is caused primarily by slower opening of the slow calcium-sodium channels, which lasts for few tenths of a second. Finally, increased opening of the potassium channels and diffusion of large amounts of positive potassium ions out of the fiber return the membrane potential to its resting level ***(Levitan and Kuzcmurek, 1991).***

There is a difference in the function of these channels in the sinus nodal fiber because of the much lesser negativity of the "resting" potential only -55 millivolts. At this level of negativity, the fast sodium channels have mainly become "inactivated" which means that they have become blocked. The cause of this is that any time the membrane potential remains less negative than about -60 millivolts for more than a few milliseconds. The inactivation gates on the inside of the cell membrane that close the fast sodium channels become closed and remain so. Therefore, only the slow calcium-sodium channels can open and thereby cause the action potential. As a result, the action potential is slower to develop than that of the ventricular muscle, and it also recovers with a slow decrement of the potential rather than the abrupt recovery that occurs for the ventricular fiber ***(Cohen et al., 2001).***

Self-excitation of sinus nodal fibers

Because of the high sodium ion concentration in the extracellular fluid as well as the negative electrical charge inside the resting sinus nodal fibers. The positive sodium ions outside the fibers even normally tend to leak to the inside. Furthermore, the resting nodal fibers have a moderate number of channels that are already open to the sodium ions. Therefore, influx of positively charged sodium ions causes a rising membrane potential. Thus, as shown in, the “prepotential", "pacemaker potential", or “‘resting potential" rises between each two heart beats. When it reaches a threshold voltage of about -40 millivolts. The ca1cium-sodium channels become activated, leading to rapid entry of both calcium and sodium ions, thus causing action potential. Therefore, basically, the inherent leakiness of the sinus nodal fibers to sodium ions causes their self excitation ***(Mazzanti and Defeliee, 1987).***

This leakiness to sodium ions does not cause the sinus nodal fibers to remain depolarized all the time because of two

events occurring during the course of the action potential. First, the calcium-sodium channels become inactivated within about 100 to 150 milliseconds after opening, and second, at about the same time, greatly increased numbers of potassium channels open. Therefore, the influx of calcium and sodium ions through the calcium-sodium channels ceases, while at the same time large quantities of positive potassium ions diffuse out of the fiber, thus terminating the action potential. Furthermore, the potassium channels remain open for another few tenths of a second, carrying a great excess of positive potassium charges out of the cell, which temporarily causes considerable excess negativity inside the fiber; this is called hyperpolarization ***(Zobrist et al., 1986).***

This hyperpolarization initially carries the resting membrane potential down to about -55 to -60 millivolts at the termination of the action potential. This new state of hyperpolarization is not maintained forever. The reason is that during the next few tenths of a second alter the action potential is over, progressively more and more of the potassium channels begin to close. Now the inward leaking sodium ions once again overbalance the outward flux of potassium ions, which causes the resting potential, prepotential or the pacemaker potential to drift upward once more, finally reaching the threshold level for discharge at a potential of about -40 millivolts. Then the entire process begins again: self-excitation, recovery from the action potential, hyperpolarization after the action potential is over, drift of the resting potential again to threshold, the re-excitation again to elicit another cycle. This process continues indefinitely throughout a person's life ***(Mitchell et al., 1983).***

Internodal pathways and transmission of the cardiac impulse through the atria

The ends of the sinus nodal fibers fuse with the surrounding atrial muscle fibers, and action potentials originating in the sinus node travel outward into these fibers, In this way, the action potential spreads through the entire atrial muscle mass and, eventually, to the AV node. The velocity of conduction in the atrial muscle is about 0.3 m/sec. Conduction is somewhat more rapid in several small bundles of atrial muscle fibers. One of these, called the anterior interatrial band, passes through the anterior walls of the atria to the left atrium and conducts the cardiac impulse at a velocity of about l m/sec. In addition, three other small bundles curve through the atrial walls and terminate in the AV node, also conducting the cardiac impulse at this rapid velocity. These three small bundles curve are called, respectively, the anterior, middle, and posterior, internodal pathways. The cause of the more rapid velocity of conduction in these bundles is the presence of a number of specialized conduction fibers mixed with the atrial muscle. These fibers are similar to the rapidly conducting purkinje fibers of the ventricles ***(Shen and Blaustein, 1992).***

AV nodal delay in impulse conduction

The conductive system is organized so that the cardiac impulse will not travel from the atria into the ventricles too rapidly. It is primarily the AV node and its adjacent conductive fibers that delay this transmission of the cardiac impulse from the atria into the ventricles. The impulse, alter traveling through the internodal pathway, reaches the AV node about 0.03 second alter its origin in the sinus node. Then there is a further delay of 0.09 second in the AV node itself before the impulse enters the penetrating portion of the AV bundle, where is passes into the ventricles.A final delay of another

0.04 second occurs mainly in this penetrating AV bundle, which is composed of multiple small fascicles passing through the fibrous tissue separating the atria from the ventricles ***(Meijler, 1985).***

Thus, the total delay in the AV nodal and AV bundle system is about 0.13 second, in addition to the initial conduction delay of 0.03 second from the sinus node to the AV node, making a total delay of 0.16 second. The AV nodal delay is shortened by stimulation of the sympathetic nerves to the heart and lengthened by stimulation of the vagi.

Cause of the slow conduction: the extremely slow conduction in the transitional, nodal, and penetrating AV bundle fibers is partly due to their sizes which are considerably smaller than the sizes of the normal atrial muscle fibers. However, most of the slow conduction is probably caused by two other entirely different factors. First, all these fibers have resting membrane potentials that are much less negative than the normal resting potential of other cardiac muscle. Second, few gap junctions connect the successive muscle cells in the pathway, so that there is great resistance to the conduction of excitatory ions from one cell to the next ***(Fozzard et al., 1986).***

Transmission in the purkinje system

The purkinje fibers lead from the AV node through the AV bundle into the ventricles. Except for the initial portion of these fibers where they penetrate the AV fibrous barrier, they have functional characteristics that are quite the opposite of those of the AV nodal fibers. They are very large fibers, even larger than the normal ventricular muscle fibers, and they transmit action potentials at a velocity of 1.5 to 4.0 m/sec, a velocity about 6 times that in the usual cardiac muscle and 150 times that in some of the AV transitional fibers. This allows almost immediate transmission of the cardiac impulse throughout the entire ventricular system. The rapid

transmission of action potentials by purkinje fibers is believed to be caused by high level of permeability of the gap junctions at the intercalated discs between the successive cardiac cells that make up the purkinje fibers. The purkinje fibers also have few myofibrils, which means that they barely contract during the course of impulse transmission ***(Katz, 1992).***

One-way conduction through the AV bundle: A special characteristic of the AV bundle is the inability, except in abnormal states, of action potentials to travel backward in the bundle from the ventricles to the atria. This prevents re-entry of cardiac impulses by this route from the ventricles to the atrial The atrioventricular fibrous barrier normally acts as an insulator to prevent passage of the cardiac impulse between the atria and the ventricles through any other route besides forward conduction through the AV bundle itself. In rare instances, an abnormal muscle bridge penetrates the fibrous barrier elsewhere besides at the AV bundle. Under such conditions, the cardiac impulse can re-enter the atria from the ventricles and cause a serious cardiac arrhythmia ***(Gravanis, 1987).***

Distribution of the purkinje fibers in the ventricles: they lie beneath the endocardium. The terminal purkinje fibers penetrate about one third of the way into the muscle mass and then become continuous with the cardiac muscle fibers. From the time the cardiac impulse enters the bundle branches in the ventricular septum until it reaches the terminations of the purkinje fibers, the total time that elapses averages only 0.03 second. Therefore, once the cardiac impulse enters the purkinje system, it spreads almost immediately to the entire endocardial surface of the ventricular muscle. This causes all portions of the ventricular muscle in both ventricles to begin contracting at almost the same time. Effective pumping by the two ventricular chambers requires this synchronous type of contraction. ***(Guyton and Hall, 1996).***