INTRODUCTION

The human heart beats 2.5 billion times during a normal lifespan.This is accomplished by cells of the cardiac conduction system (CCS). The functional components of the CCS can be broadly divided into the impulse-generating nodes and the impulse-propagating His-Purkinje system ***(Benson et al., 2003)***.

Human diseases of the conduction system have been identified that alter impulse generation, impulse propagation, or both. CCS dysfunction is primarily due to acquired conditions such as myocardial ischemia/infarct, age-related degeneration, procedural complications, and drug toxicity***(Benson et al., 2003).***

Battery-operated pacing devices were introduced by C.W.Lillehei and Earl Bakken in 1958.The natural progression of pacemaker (PM) developments led to invention of the implantable cardioverter defibrillator (ICD) around 1980 by Michael Morchower.A pacemaking system consists of an impulse generator and lead or leads to carry the electrical impulse to the patient's heart. Leads can be unipolar, bipolar or multipolar ***(Rozner et al., 2009***).

Cardiovascular implantable electronic device (CIED) is a term that encompasses pacemakers for bradyarrhythmia treatment, implantable cardioverter defibrillators (ICDs) for

tachyarrhythmia management, and cardiac resynchronization therapy (CRT) devices for systolic dysfunction with conduction delays. As the indications for device placement continue to expand and with data supportive of device placement compared to medical therapy well established, CIEDs are becoming common in our patient population ***(Roger, 2011).***

A focused preoperative evaluation should include establishing whether a patient has a CIED, defining the type of the device, determining whether a patient is CIED dependant for pacemaking function and determining CIED function. Preoperative CIED function is ideally assessed by a comprehensive evaluation of the device. Consultation with a cardiologist or CIED service may be necessary ***(Javieret al., 2012).***

Implantable cardioverter-defibrillators should have their tachyarrhythmia treatment algorithms programmed off before surgery and turned on after surgery to prevent unwanted shocks due to spurious signals that the device might interpret as ventricular tachycardia or fibrillation. During the period of time when device therapy has been inactivated, the patient should be monitored continuously for a lifethreatening arrhythmia ***(Fleisher et al., 2007).***

All patients with implanted devices should have both continuous ECG monitoring and continuous pulse monitoring during surgery.This reflects the fact that electrocautery may

interfere with ECG monitoring and make it difficult or impossible to determine the patient's rhythm. Efforts should be made to minimize the chance for interactions by careful management of potential sources of EMI ***(Fleisher et al., 2007).***

Most patients with a CIED can be managed safely with the use of a magnet in the perioperative setting.Therefore, it is important to understand the effect of placing a magnet over a pulse generator ***(Javier et al., 2012).***

Aim of the Work

The aim of the work is to review the current medical literature as regards the anesthetic management of patients with cardiac implantable electronic devices undergoing non cardiac surgeries.

ELECTROPHYSIOLOGY CONSIDERATIONS OF THE HEART

The heart essentially is a conical structure composed of layers of myocardium enclosing the atrial and ventricular chambers. The atrial and ventricular walls are anchored to the fibrous atrioventricular valve annuli. The aorta and main pulmonary artery arise from their respective fibrous valve rings, and these four fibrous rings together are termed the fibrous skeleton of the heart**.** Located in the central chest, the heart within the pericardial sac resides in the middle mediastinum, with two-thirds of its volume to the left and one-third to the right of center ***(Virmani et al., 1987).***

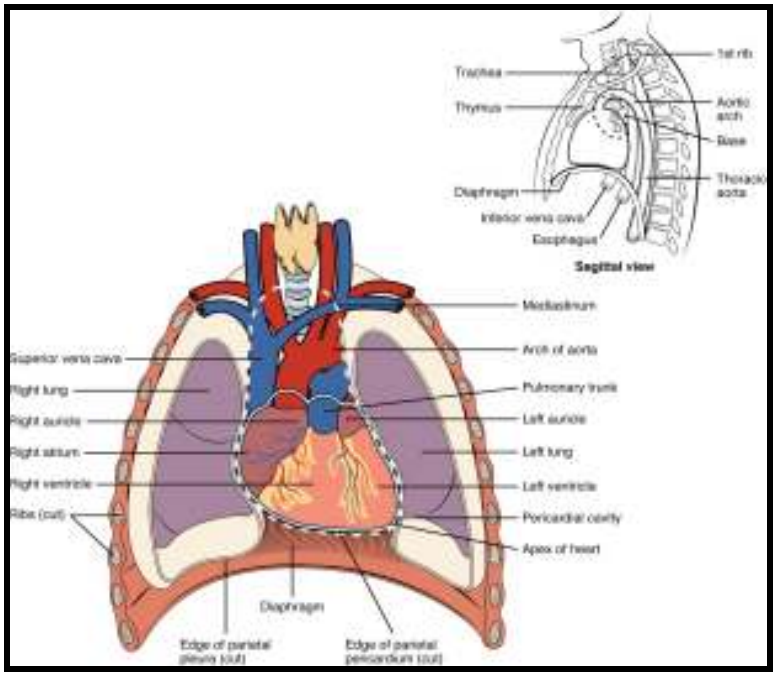

Figure (1): Location of the heart ***(Virmani et al., 1987).***

Anatomy of the heart

The heart has four chambers, two upper atria, the receiving chambers, and two lower ventricles, the discharging chambers. The atria are connected to the ventricles by the atrioventricular valves and separated from the ventricles by the coronary sulcus ***(Virmani et al., 1987).***

The conductive system of the heart.

A-Anatomy of the conductive system

Although the work of the heart is dependent on myocardial contractility, the mechanism that initiates and provides order to the phasic contraction and relaxation of the cardiac muscle is dependent on the other two fundamental properties of cardiac muscle, automaticity and conductivity. The conduction system including nodes, bundle branches and Purkinje fibers—consists of modified myocardial cells that are positioned to either facilitate or slow impulse conduction. Both the sinoatrial node and the atrioventricular node are comprised of specialized myocardial cells with highly developed automaticity, and the myocardial cells of the His bundle, bundle branches and Purkinje fibers have the specialized property of rapid conductivity ***(Mommersteeg et al., 2007).***

The normal rhythmical heart beat, called sinus rhythm, is established by the sinoatrial node, the heart's pacemaker. Here an

electrical signal is created that travels through the heart, causing the heart muscle to contract. The **sinoatrial node** is found in the coronary sinus of the right atrium. The electrical signal generated by the sinoatrial node travels through the right atrium in a radial way. It travels to the left atrium via **Bachmann's bundle**, such that both left and right atrium contract together. The signal then travels to the atrioventricular node. This is found at the bottom of the right atrium in the atrioventricular septum-the boundary between the right atrium and the left ventricle. The septum is part of the cardiac skeleton, tissue within the heart that the electrical signal cannot pass through, which forces the signal to pass through the atrioventricular node only. The signal then travels along the Bundle of His to left and right bundle branches to the ventricles of the heart. In the ventricles the signal is carried by specialized tissue called the Purkinje fibers which then transmit the electric charge to the cardiac muscle ***(De Ponti & Roberto, 2002).***

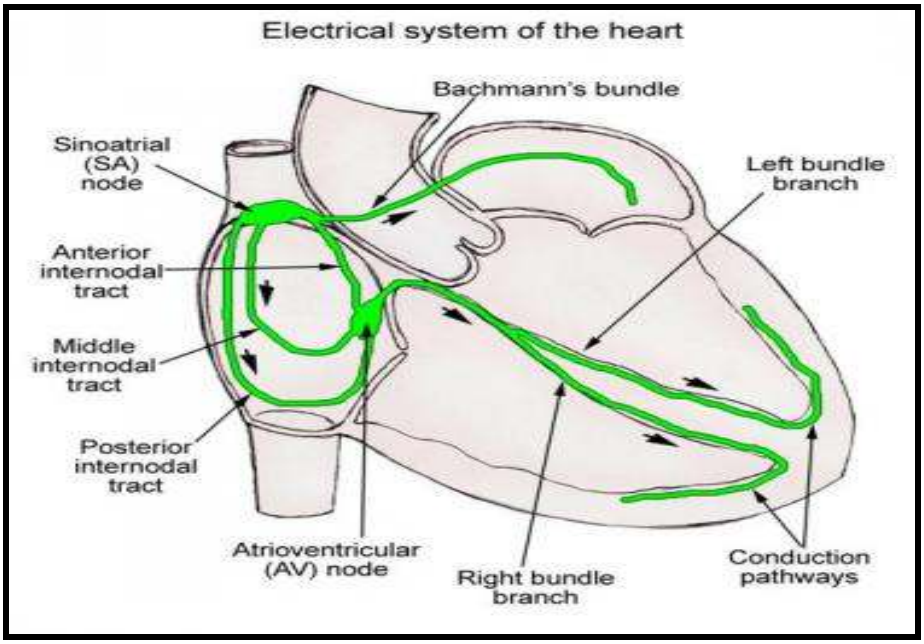

Figure (2): Anatomy of the conductive system ***(De Ponti & Roberto, 2002).***

In some hearts there are accessory muscle bundles that span the fibrous atrioventricular rings, connecting atrial to ventricular muscle directly and thus bypassing the AV node. Called **bundles of Kent**, these muscle bridges offer low-resistance pathways for the impulse to travel from atrium to ventricle, and they form the anatomic basis for the Wolff-Parkinson-White syndrome or accelerated atrioventricular conduction. Kent bundles can be situated anywhere along the atrioventricular annuli ***(Kent, 1913).***

All living cells, including cardiomyocytes, maintain a difference in the concentration of ions across their membranes.

There is a slight excess of positive ions on the outside of the membrane and a slight excess of negative ions on the inside of the membrane, resulting in a difference in the electrical charge (i.e., voltage difference) across the cell membrane, called the membrane potential (Em). A membrane that exhibits an Em is said to be polarized ***(Zaza, 2010).***

In non-excitable cells, and in excitable cells in their baseline states (i.e., not conducting electrical signals), the Em is held at a relatively stable value, called the resting potential. All cells have a negative resting Em (i.e., the cytoplasm is electrically negative relative to the extracellular fluid), which arises from the interaction of ion channels and ion pumps embedded in the membrane that maintain different ion concentrations on the intracellular and extracellular sides of the membrane ***(Zaza, 2010).***

The most important ion fluxes that depolarize or repolarize the membrane are passive (i.e., the ions move down their electrochemical gradient without requiring the expenditure of energy), occurring through transmembrane ion channels. In excitable cells, a sufficiently large depolarization can evoke a short-lasting all-or-none event called an action potential, in which the Em very rapidly undergoes specific and large dynamic voltage changes ***(Zaza, 2010).***

Both resting Em and dynamic voltage changes such as the action potential are caused by specific changes in

membrane permeabilities for Na^+, K^+, Ca^{+2}, and Cl^-, which, in turn, result from concerted changes in functional activity of various ion channels, ion transporters, and exchangers (***Grant, 2009).***

B-The cardiac action potenial

The cardiac action potential reflects a balance between inward and outward currents. When a depolarizing stimulus (typically from an electric current from an adjacent cell) abruptly changes the Em of a resting cardiomyocyte to a critical value (the threshold level), the properties of the cell membrane and ion conductances change dramatically, precipitating a sequence of events involving the influx and efflux of multiple ions that together produce the action potential of the cell. In this fashion, an electrical stimulus is conducted from one cell to all the cells that are adjacent to it ***(Zaza, 2010).***

Unlike skeletal muscle, cardiac muscle is electrically coupled so that the wave of depolarization propagates from one cell to the next, independent of neuronal input. The heart is activated by capacitive currents generated when a wave of depolarization approaches a region of the heart that is at its resting potential.

Unlike ionic currents, which are generated by the flux of charged ions across the cell membrane, capacitive currents are

generated by the movement of electrons toward and away from the surfaces of the membrane ***(Zaza, 2010).***

When an excitatory stimulus causes the Em to become less negative and beyond a threshold level (approximately −65 mV for working atrial and ventricular cardiomyocytes), Na^+ channels activate (open) and permit an inward Na^+ current (I_{Na}), resulting in a rapid shift of the Em to a positive voltage range. This event triggers a series of successive opening and closure of selectively permeable ion channels (***Grant, 2009).***

Electrical changes in the action potential follow a relatively fixed time and voltage relationship that differs according to specific cell types. Whereas the entire action potential takes several milliseconds in nerve cells, the cardiac action potential lasts several hundred milliseconds. The course of the action potential can be divided into five phases (numbered 0 to 4). Phase 4 is the resting Em, and it describes the Em when the cell is not being stimulated ***(Bodi et al., 2005).***

In normal atrial and ventricular myocytes and in His-Purkinje fibers, action potentials have very rapid upstrokes, mediated by the fast inward I_{Na}. These potentials are called fast response potentials. In contrast, action potentials in the normal sinus and atrioventricular (AV) nodal cells and many types of diseased tissues have very slow upstrokes, mediated by a slow inward, predominantly L-type voltage-gated Ca^{2+} current (I_{CaL}),

rather than by the fast inward I_{Na} These potentials have been termed slow response potentials ***(Bodi et al., 2005).***

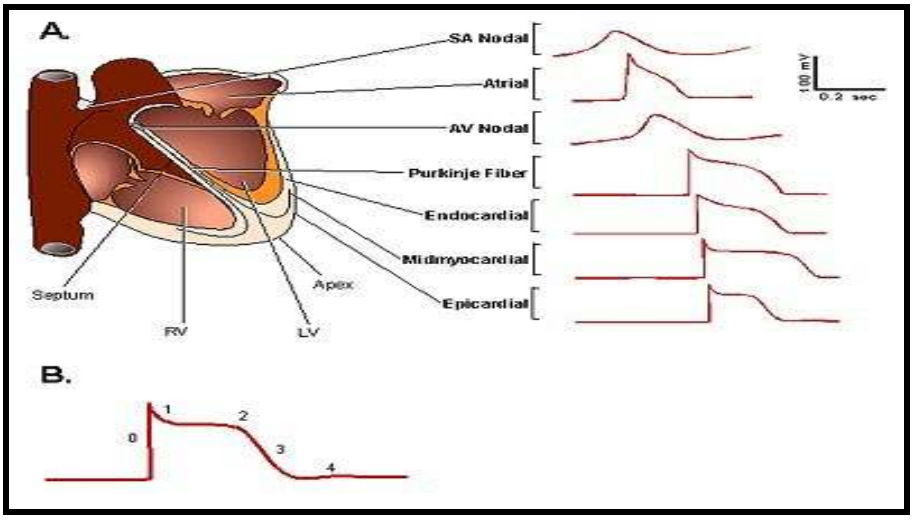

Figure (3): Different Action potenials of the heart ***(Zaza, 2010).***

There are two types of action potential:

1- The Fast Response Action Potential

- PHASE 4: The resting membrane potenial

The Em of resting atrial and ventricular cardiomyocytes remains steady throughout diastole. The resting Em is caused by the differences in ionic concentrations across the membrane and the selective membrane permeability (conductance) to various ions. Large concentration gradients of Na^+, K^+, $Ca2^+$, and Cl^- across the cell membrane are maintained by the ion pumps and exchangers ***(Zaza, 2010).***

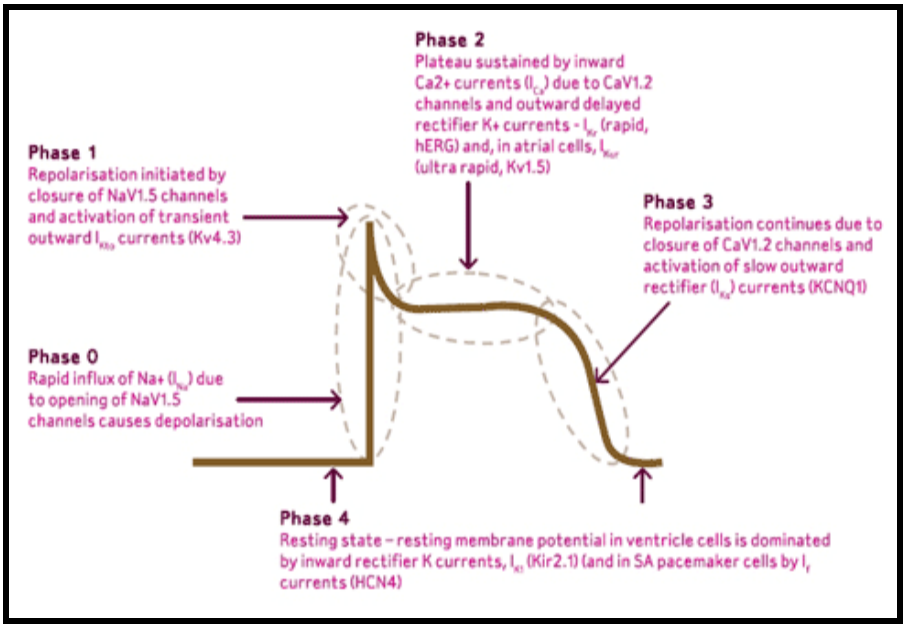

Figure (4): Stages of fast response action potential ***(Grant, 2009).***

- Phase 0: The upstroke-Rapid depolarization

On excitation of the cardiomyocyte by electrical stimuli from adjacent cells, its resting Em (approximately −85 mV) depolarizes, leading to opening (activation) of Na^+ channels from its resting (closed) state and enabling a large and rapid influx of Na^+ ions (inward I_{Na}) into the cell down their electrochemical gradient. As a consequence of increased Na^+ conductance, the excited membrane no longer behaves like a K^+ electrode (i.e., exclusively permeable to K^+), but more closely approximates a Na^+ electrode ***(Andavan et al., 2011).***

- Phase 1: Early repolarization

Phase 0 is followed by phase 1 (early repolarization) during which the membrane repolarizes rapidly and transiently to almost 0 mV (early notch), partly because of the inactivation of I_{Na} and concomitant activation of several outward currents. The transient outward K^+ current is mainly responsible for phase 1 of the action potential ***(Niwa & Nerbonne, 2010).***

- Phase 2: The plateau

Importantly, during the plateau phase, membrane conductance to all ions falls to rather low values. Thus, less change in current is required near plateau levels than near resting potential levels to produce the same changes in Em ***(Tamargo et al., 2004)***

- Phase 3: Final Rapid Repolarization

Phase 3 is the phase of rapid repolarization that restores the Em to its resting value. Final repolarization during phase 3 results from K^+ efflux through the I_K channels, which open at potentials negative to-20 mV ***(Tamargo et al., 2004).***

- Phase 4: Restoration of resting membrane potenial

During the action potential, Na^+ and $Ca2^+$ ions enter the cell and depolarize the Em. Although the Em is quickly repolarized by the efflux of K^+ ions, restoration of transmembrane ionic concentration gradients to the baseline

resting state is necessary. This is achieved by the Na^+-K^+ ATPase (Na^+-K^+ pump, which exchanges two K^+ ions inside and three Na+ ions outside) and by the Na^+-$Ca2^+$ exchanger (I_{Na-Ca}, which exchanges three Na^+ ions for one $Ca2^+$ ion) ***(Bodi et al., 2005).***

2- The Slow Response Action Potential

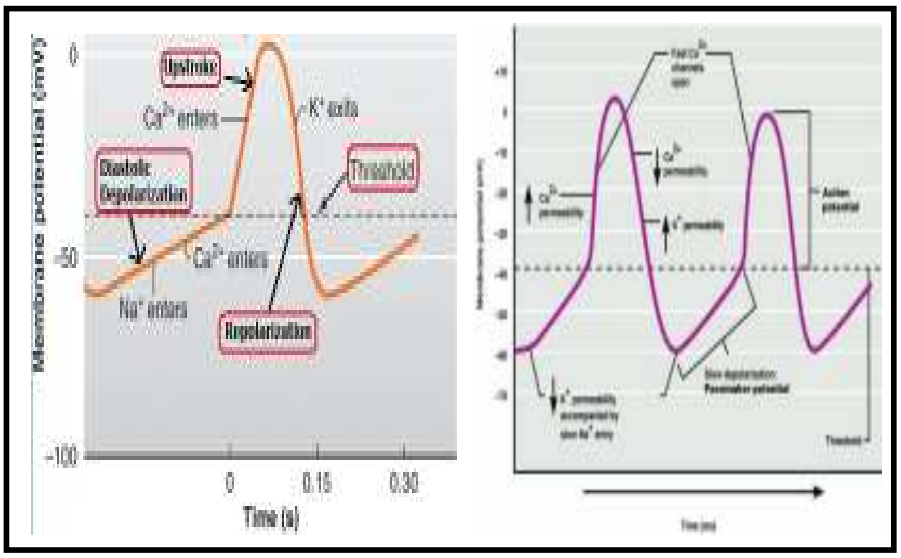

Figure (5): Stages of slow response action potential ***(Bodi et al., 2005).***

- PHASE 4: Diastolic Depolarization

In contrast to working atrial and ventricular myocytes and fibers in the His-Purkinje system, which maintain a steady diastolic Em level of approximately –85 mV, sinus and AV nodal excitable cells exhibit a spontaneous, slow, progressive decline in the Em during diastole (spontaneous diastolic depolarization or phase 4 depolarization) that underlies normal