Anesthetic Management of Patient with Pacemaker undergoing surgery using Electrocautery

Assay

Submitted for partial fulfillment of Master Degree in Anaesthesia

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Introduction

Artificial pacing is indicated for treatment of persistent bradycardia of any origin if it compromises hemodynamics. The science of pacing is a relatively new development in the treatment of cardiac patients *(Atlee, Bernstein, 2001)*.

Since large numbers of patients have pacemakers 1: 460 in the general population. It is likely that most anesthesiologists will eventually care for these patients during surgery and diagnostic procedures, as well as during pacemaker implantation and battery replacement *(Parsonnet and Bernstein, 1983)*.

The purpose of this essay is to provide a basic knowledge about pacemakers and anesthetic management of the pacemaker patient so as to minimize complications and malfunction.

PHYSIOLOGICAL CONSIDERATIONS

Cardiac Impulse Conductive System:

Cardiac electrical impulses originate in the sinus node (SA node), a spindle shaped structure 10-20 m.m long located near the junction of the superior vena cava and the right atrium *(Hoffman, 1979)*.

Even though various specialized tissue have been postulated to conduct the electrical impulses from the sinus node (SA node) to the atrio-ventricular node (AV node), electrical transmission is probably cell to cell via working atrial muscle.

The AV node provides the only normal conduction pathway between the atria and ventricles. It is situated just beneath the right atrial endocardium above the insertion of the septal leaflet of the tricusped valve and anterior to the ostium of the coronary sinus *(Emily, 2005)*.

After conduction delay in the AV node, the electrical impulse travels to the His-bundle, which descends posteriorly along the membranous interventricular septum to the top of the muscular septum. The His-bundle gives rise to the right and left bundle branches. The right bundle branch is a single group of fibers that travels down the right ventricular side of the muscular interventricular septum. The left bundle branch is a larger, less discrete array of

conducting fibers located on the left side of the interventricular septum. The left and right bundle branches progressively divide into tiny purkinje fibers that arborize and finally make intimate contact with ventricular muscle tissue *(Suma, Sunao, 2001)*.

Like the SA node, the AV node and His bundle regions are innervated with a rich supply of cholinergic and adrenergic fibers *(Emily, 2005).*

Spread of Cardiac Excitation:

Depolarization initiated in the SA node spreads rapidly through the atria, converging on the AV node. Atrial depolarization is complete in about 0.1 second. Because conduction in the AV node is slow, there is a delay of about 0.1 second (AV node delay) before excitation spreads to the ventricles.

From the top of the septum, the wave of depolarization spreads in the rapidly conducting purkinje fibers to all parts of the ventricles in about 0.08-0.1 sec. In humans depolarization of the ventricular muscle starts at the left side of the interventricular septum and moves first to the right across the midportion of the septum then down the septum to the apex of the heart. It returns along the ventricular walls to the AV groove, proceeding from the endocardial to the epicardial surface. The last parts of the heart to be depolarized are the posteriobasal portion of the left ventricle, the pulmonary conus, and

the upper most portion of the septum *(Edward, Maged and Michael, 2002)*.

Electrophysiology of Cardiac Stimulation:

If a microelectrode is introduced into a single myocardial cell, an action potential can be recorded by measuring the potential difference between the inside and the outside of the cell, (inside negative).

The resting membrane potential of a normal purkinje cell is approximately -90 mv with respect to the outside of the cell. When the membrane potential is depolarized to a certain threshold level, an action potential occurs with a rapid up stroke (phase 0), a return toward zero from the up stroke or early rapid repolarization (phase 1), a plateau (phase 2), finally rapid repolarization (phase 3), and resting membrane potential and diastolic depolarization (phase4) Fig. (1) *(Edward, Maged and Michael, 2002)*.

The normal resting potential is maintained by the active (i.e, energy requiring) exclusion of sodium and accumulation of potassium inside the cell (Fig 2). (Phase 0) or rapid depolarization is due to the opening of the sarcolemmal channels to sodium entrance in atrial muscle, ventricular muscle and cells in the His-purkinje system *(Klein, Reek, 2000)*. Calcium is important in the maintenance of the action potential plateau of fast sodium channel- dependent cells and in generation of the action potential up stroke in slow

calcium channel-dependent cells such as the SA&AV nodes Fig. (1) *(Hoffman, 1979)*.

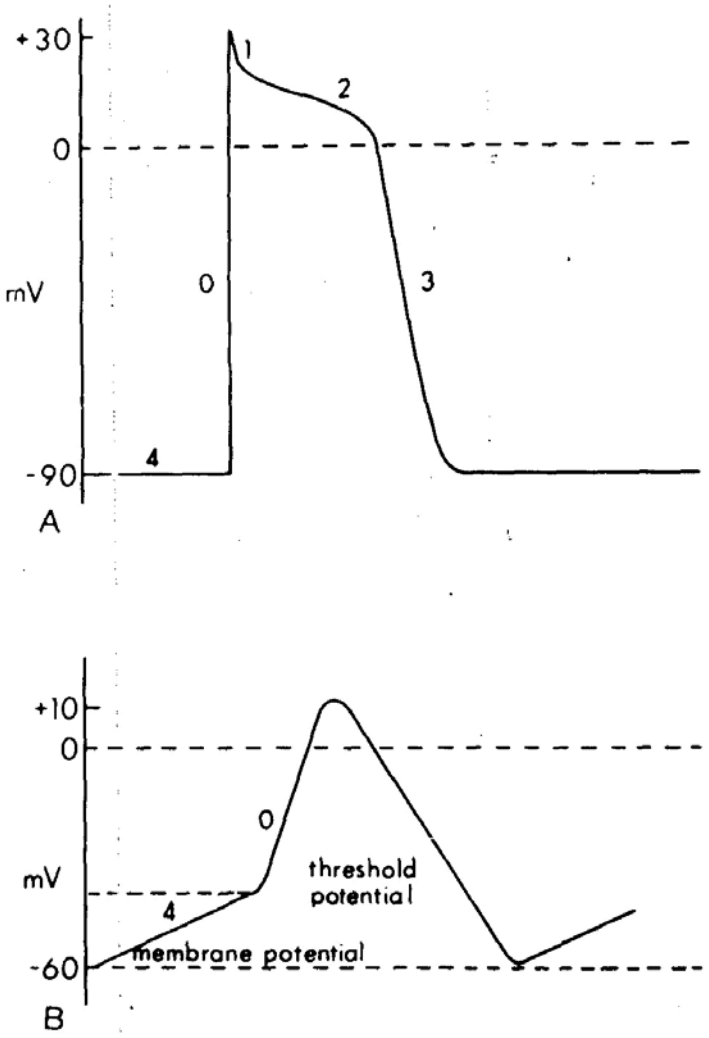

Fig. (1): A, An action potential typical of a Purkinje fiber of ventricular myocardial cell. The resting membrane potential is – 90 mV, and there is no spontaneous phase 4 depolarization. The upstroke (phase 0) is rapid. B, An action potential recorded from a cell displaying automaticity such as found in the sinoatrial (SA) and atrioventricular (AV) nodes. Spontaneous phase IV depolarization is present. The resting membrane potential is lower (-60 mV), and the upstroke is slower.

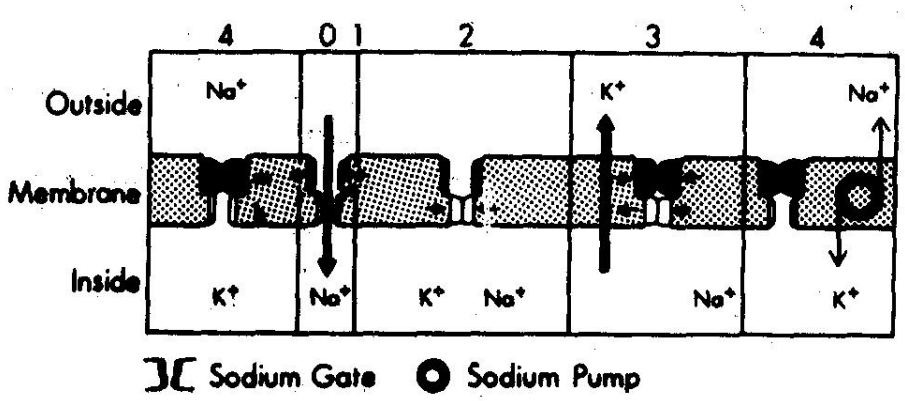

Fig. (2): Above are shown the cardiac membrane, the gates which open and close to allow Na and K ions to move across the membrane, causing depolarization and then repolarization. Finally, the activity of the pump which restores Na and K during phase 4 is illustrated. *(Hoffman, 1979).*

Phase (3) is mediated by an outward potassium current, and the membrane returns to its negative resting potential during electrical diastole.

Automaticity : is a property of some cardiac tissues to undergo gradual (phase 4) depolarization spontaneously until threshold potential is reached and the cell initiates an action potential that is propagated from one cell to another. Normal automaticity is present in sinus nodal tissue, some atrial and junctional tissues, the bundle branches, and purkinje fibers.

The sinus node discharges more rapidly than the other cells and is the normal pacemaker of the heart *(Surawicz, 1980).*

Conduction : is the propagation of a cardiac impulse and is most closely influenced by the amplitude and upstroke velocity of [phase 0] of the action potential *(Klein, Reek, 2000)*.

Refractoriness: is a property of cardiac tissue during which a stimulus occuring soon after a previous action potential fails to elicit another normal action potential. It is most closely related to the duration of (phase 3) of the cardiac action potential in most tissues *(Surawicz, 1980)*.

The genesis of the normal Electrocardiogram : is from electrical activity recorded by skin electrodes that is the sum of all the cardiac action potentials of its component cells. The "P" wave represents atrial depolarization. The "P-R" interval is a measure of the time necessary to travel from the sinus node through the atrium, AV node and His- purkinje system to activate ventricular myocardial cells. The "QRS" complex represents the sum of all ventricular muscle cell depolarization (phase 0), the "ST" segment represents the plateau phase, and the "T" wave represents the rapid repolarization (phase 3) of the heart as a whole Fig. (3) *(Suma, Sunao, 2001)*.

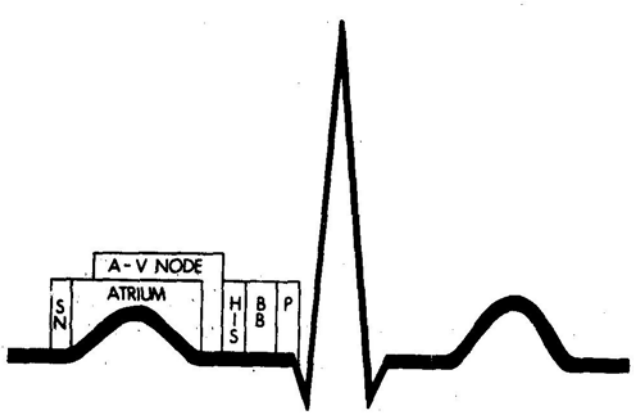

Fig. (3): Normal electrocardiographic complex. Also shown is the sequence of activity in the specialized conducting system

SN = sinus node; HIS = bundle; BB = bundle branches; P = peripheral Purkinje tissue.

Although the autonomic nervous system may affect atrial and ventricular tissue to a small extent, the most prominent autonomic effects are observed on the SA node and AV node. Sympathetic stimulation increases the rate of automaticity and increases conduction velocity, whereas parasympathetic (vagal) activation does the opposite. Baroreceptors in the carotid sinus, located at the bifurcation of the internal and external carotid arteries, activate the vagus nerve when blood pressure increases and reflexely decreases heart rate and AV nodal-conduction velocity *(Ammirati, Colvicchi, Santini, 2001).*

PATHOLOGICAL CONSIDERATIONS

I. Sinus Node Dysfunction:

Sinus node dysfunction may manifest as [sinus bradycardia, sinus pause or arrest, or sinoatrial block, with or without escape rhythms].It often occurs in association with paroxysmal supraventricular tachydysrhythmias (bradycardia-tachycardia syndrome). Sinus bradycardia due to increased vagal tone is physiologic in trained athletes, who may have sleeping heart rates as low as 30 beats/min, with sinus pauses or type I 2ºAVHB *(Lamas et al.,2002).*

Patients with sinus node dysfunction may have symptoms due to bradycardia, tachycardia, or both. Correlation of symptoms with dysrhythmias is essential and is established by ambulatory monitoring. Sinus node dysfunction may also present as a deficient rate response to stress or exercise (i.e chronotropic incompetence). *(Santini et al.,1990) .*

II- Atrioventricular Heart Block (AVHB):

AVHB is classified as first-degree (1º),second-degree (2º), or third-degree (3º;complete). Anatomically, it may occur above, within, or below the His bundle.

AVHB may be acquired (ie, disease, surgical) or congenital, and it may be temporary (ie, drugs, myocardial infarction, surgical) or permanent, which will influence the decision to institute temporary or

permanent pacing *(Atlee and Bernstein, 2001).*

Patients with AVHB may be asymptomatic or they may experience debilitating symptoms related to bradycardia and/or ventricular dysrhythmias *(Gregoratos et al.,2002)*

A-First-degree (1°AVHB):

P-R interval is greater than 0.20 s and is usually due to A-V node conduction delay (Fig. 4).

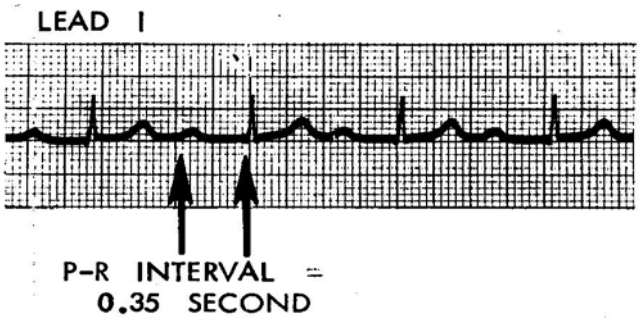

Fig. (4): First degree AV block. The PR interval is prolonged to 0.35s.

B- Second-degree (2° AVHB):

1. Type I or Wenckebach 2°AVHB (I 2°AVHB):

There is gradual PR interval prolongation before dropped beats. It is usually associated with a narrow QRS complex. It almost always occurs at the A-V node. When associated with bundle-branch block, there is infra-Hisian block in up to 30% of cases Fig. (5).

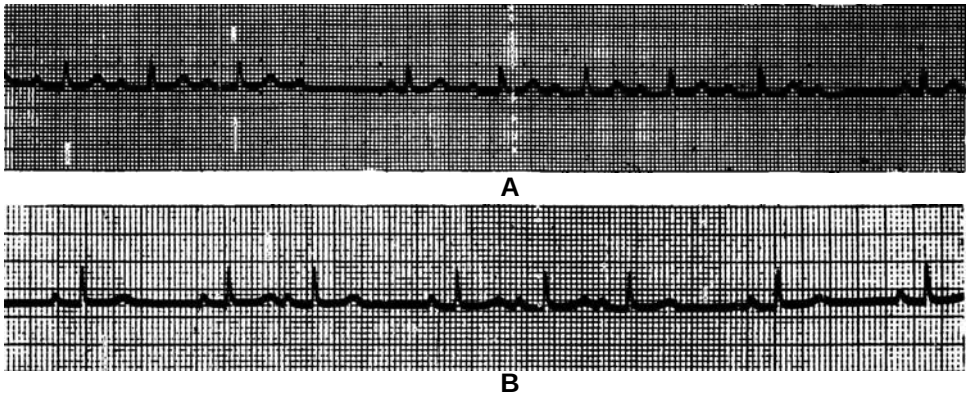

Fig. (5): Wenckebach phenomenon. A. A 4:3 and 6:5 AV Wenckebach period. Note that the PQ interval progressively lengthens, but by a decreasing increment; therefore the ventricular cycle tends to shorten (at least for the first two cycles following the dropped beat). B. A 3:3 and 4:3 sinus Wenckebach period with 2:1 SA block at beginning and end of strip *(Suma, Sunao, 2001).*

2. Type II or Mobitz 2° AVHB (II 2° AVHB):

No P-R interval prolongation. It is associated with a wide QRS complex and generally localized to within the His – Purkinje system. Advanced type II 2°AVHB refers to block of two or more consecutive P waves (fig. 6).

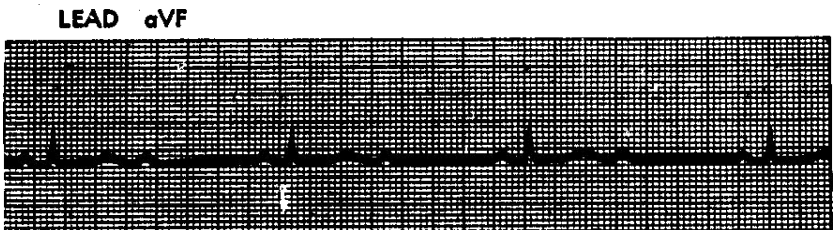

Fig. (6): Second degree AV block. There are two P waves to each QRS-2:1 AV block (every alternate sinus impulse is blocked).

C- Third-degree (3°AVHB):

There is no association between atrial and ventricular beats. This block can occur in the A-V node, in the bundle of his, or more distally if the right, left bundle branches are simultaneously blocked (fig. 7) *(Ellenbogen, 2000)*.

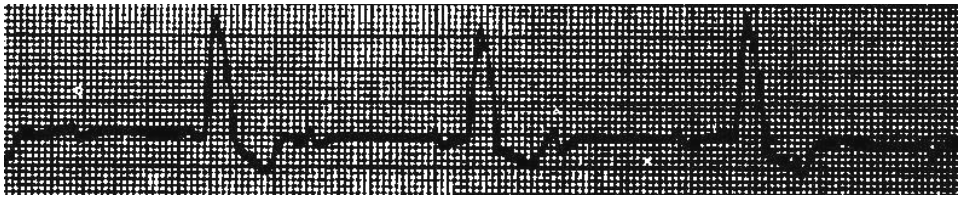

Fig. (7): Complete (third degree) AV block. There is a regular idioventricular rhythm at rate 36, and the P waves indicate their independence by changing their relation to the QRS complexes.

III Chronic Bifascicular and Trifascicular Block :

Major fascicles of the conduction system below the His bundle are the right bundle branch and the left anterior and posterior fascicles of the left bundle branch. The latter activate the left ventricular free wall. In addition, septal branches of the left bundle branch supply the middle third of the ventricular septum and provide the earliest ventricular activation *(Klein, Reek, 2000)*.

Isolated block of any one of these fascicles is unifascicular block. Left or right bundle branch block with left anterior or posterior fascicular block is bifascicular block. Block involving any three