DIAGNOSIS AND MANAGEMENT OF INTRAOPERATIVE DYSRHYTHMIAS OCCURING DURING NON-CARDIAC SURGERY

ESSAY

SUBMITTED TO PARTIAL FULFILMENT OF MASTER DEGREE IN (ANESTHESIOLOGY AND INTENSIVE CARE)

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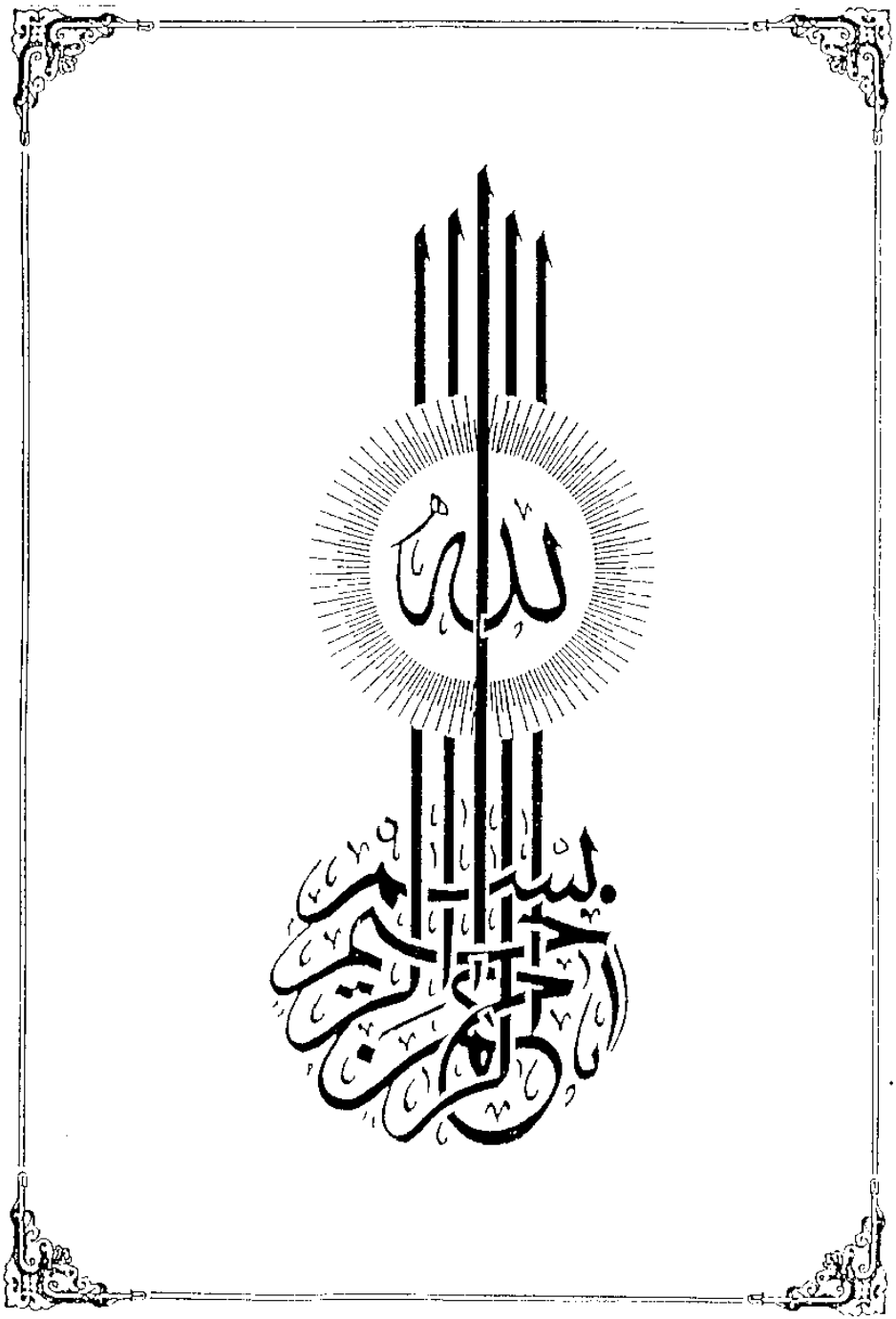
TO MY PARENTS

"To whom I owe so much;
To whom I have offered so little."

Ayman



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CONTENTS

INTRODUCTION AND AIM OF THE WORK

INTRODUCTION

The art of palpating the pulse and its relationship to the heart dates to antiquity. The Egyptians during the Pyramid Age (3000 to 2500 BC) recognized and counted the pulse and this was employed to evaluate the status of the heart. *(Springman and Atlee, 1989).*

Among the most common vexing and perplexing problems encountered in the operating suite is the patient with an abnormal cardiac rhythm which occurs in 60% or more of the anesthetized patients. *(Atlee, 1986).*

Certainly, one must first preceive the existence of a rhythm disorder before a diagnosis and subsequent corrective

measures are taken. In the operating suite, one must interpret the implications of dysrhythmias differently according to the type, acuteness and duration, clinical setting, the patient's general condition and finally the hemodynamic consequences. *(Royster, 1989).*

It is worth to regard the abrupt onset of dysrhythmias as a sign of an underlying problem rather than a final diagnosis. *(Rosen, 1988).*

In this essay the subject of intraoperative dysrhythmia, which may occur during non cardiac surgery and that faces the anesthesiologist with a problem that may dangerously affect the hemodynamics of his patient, will be discussed.

The pathophysiology and the etiology of intraoperative dysrhythmia will be presented. Also, the pharmacology of different drugs and electrical methods that can be used to control intraoperative dysrhythmia together with the art of managing different types of dysrhythmia, will be discussed.

PATHOPHYSIOLOGY OF CARDIAC DYSRHYTHMIAS

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* Anatomical consideration:-

The sinoatrial node (SAN) is located at the junction of superior vena cava and the right atrium.(Fig. 1) Impulse arising in the SAN spreads throughout the atria but may be conducted throngh the anterior, middle, and posterior internodal tracts, and interatrial tract *(Bachmann's bundle). (Becker, et al., 1981).*

The impulse is conducted from atria to ventricles via the atrioventricular node (AVN), which is located in the right atrial endocardium. The AVN contains slowly conducting cells that significantly delay transmission of electrical impulse. *(Ganong, 1989).*

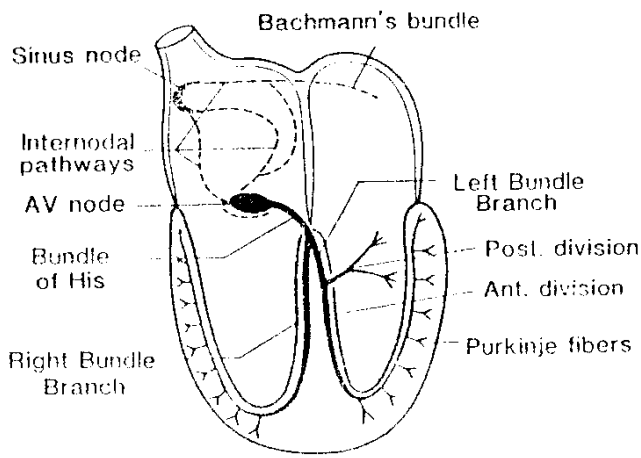


Fig 1– Diagrammatic representation of cardiac conduction system. (Routed from Ganong, 1989)

From the AVN the impulse is conducted by the His-Purkinje system to ventricular myocardium. The His bundle arises from AVN and divides at the muscular interventricular septum into right and left bundle branches (RBB, LBB). The LBB usually divides into anterior and posterior divisions. The terminal Purkinje fiber network extends from the bundle branches to the ventricular subendocardium delivering electrical impulse to both ventricules nearly Simultaneously. *(Becker, et al., 1981).*

Sympathetic innervation of heart arises from the thoracic segments T_1 to T_4. Catecholamines increase pacemaker discharge rate, shorten AVN conduction time, but donot affect normal His-bundle conduction. *(Ganong, 1989).*

Parasympathetic innervation via vagi nerves provide an opposite effects to those of sympathetic stimulation and are mediated by acetylcholine (ACh). It is currently known that vagal fibers are probably distributed only to the nodal and atrial tissue. In fact, at least two types of ACh receptors are found in the ventricles: Type I receptors are vagal, while type II receptors are not blocked by atropine *(Rogers, 1986).*

* Basic Electrophysiology:-

The inside of the myocardial cell is negatively charged in respect to the outside, with transmembrane potential of (-60) to (-90) mV. This is known as resting membrane potential (RMP). This RMP results from unequal distribution of ions across cell memberane. The chief intracellular ion is potassium (K^+), while the chief extracellular one is sodium (Na^+) *(Katz, et al., 1982).*

Cardiac cell membranes possess ion channels (protein molecules), that permit ions to move across the sarcolemma. These ionic movements give rise to depolarizing and repolarizing currents that are responsible for electrical activity of the heart. *(Katz, et al., 1982).*

The action potential (AP) is a self propagating, all-or-none sequence of transmembrane potential changes caused by changes in memberane permeability to various inos, and accompanied by transmembrane ion currents. *(Rosen, 1988).*

The cardiac action potential has five distinct phases:- (Fig.2)

(1) The initial depolarization (phase o), due to a rapid increase in Na^+conduction.

(2) The initial rapid repolarization (phase 1), due to closure of Na^{+} channels and chloride (Cl^{-}) influx.

(3) Plateau (phase 2), due to a slower but prolonged opening of Ca^{++} channels.

(4) Final repolarization (phase 3), due to closure of Ca^{++} channels and prolonged opening of K^{+} channels. This restors the resting potential.

(5) Spontaneous diastolic depolarization (phase 4), mainly due to slow Ca^{++} rather than Na^{+} current. In general, there are cells that typically display spontaneous depolarization (Slow response type) or pacemakers, and those that usually do not (fast response type) or Purkinje cells. *(Springman and Atlee, 1989).*

* **Fast Response cells:- (purkinje cells)**

They have the following characteristics:-

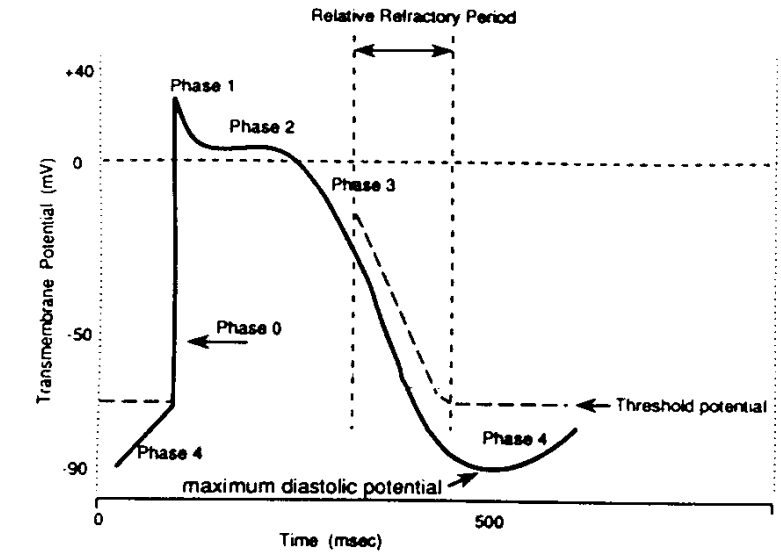
(1) Maximum negative diastolic potential is about -80 to - 90 mV.

(2) The threshold potential is usually greater than - 70 mV.

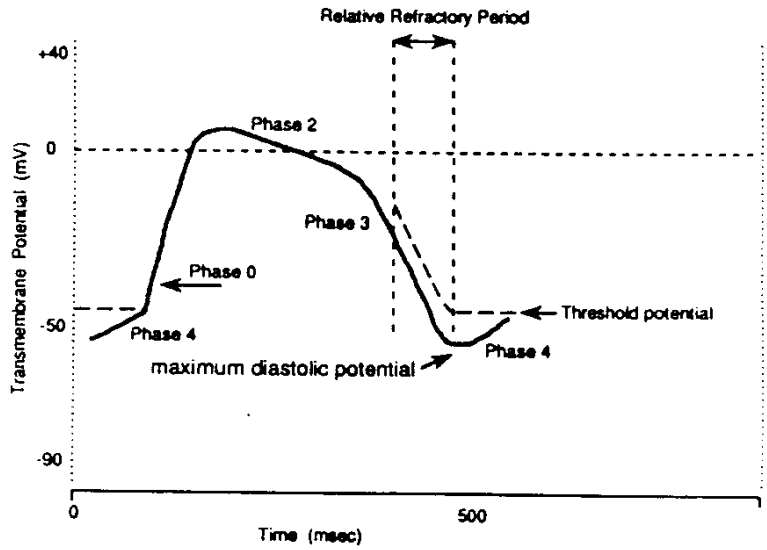
(3) Phase 0 is rapid.

(4) There is an early rapid phase 1 repolarization.

(5) The AP is completed by a phase 2 plateau and a phase 3 final repolarization.



Panel A.



Panel B.

Figure 2. Stylized cardiac action potentials from fast and slow cells. (*A*) A typical action potential from a fast response fiber displaying automaticity (Purkinje). (*B*) Action potential from a slow response fiber displaying automaticity (SA and AV nodes).

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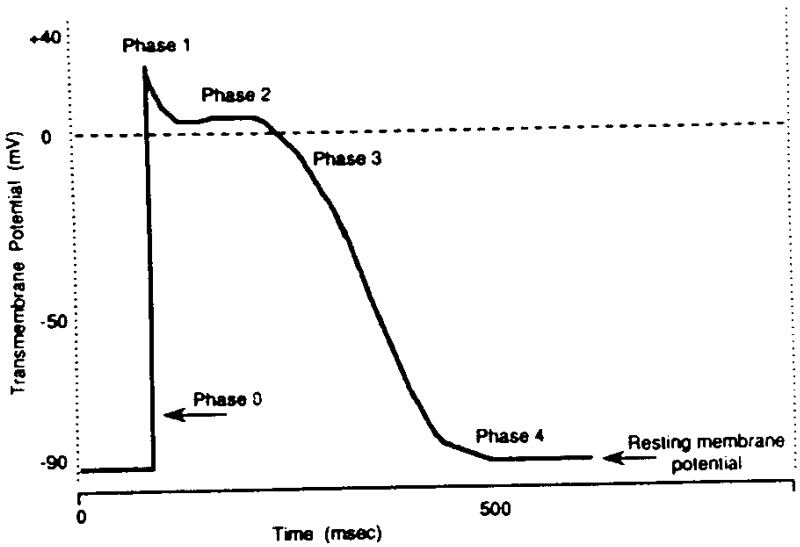

Panel C.

Figure 2. *Continued*). (C) A fast response action potential from cardiac muscle, not usually showing automaticity.

[Quoted from Springman and Attlee, 1989]

* Slow Response Cells:- (SA and AV nodal cells)

They exhibit features as follows:-

(1) The maximum diastolic potential is about - 40 to - 70 mV.

(2) Threshold potential is less than - 70 mV.

(3) Phase 0 is slower.

(4) Phase 1 is not apparent. *(Ganong, 1989).*