CARDIAC PACING
3 YEARS EXPERIENCE IN AIN SHAMS C.C.U.

THESIS

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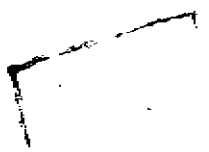

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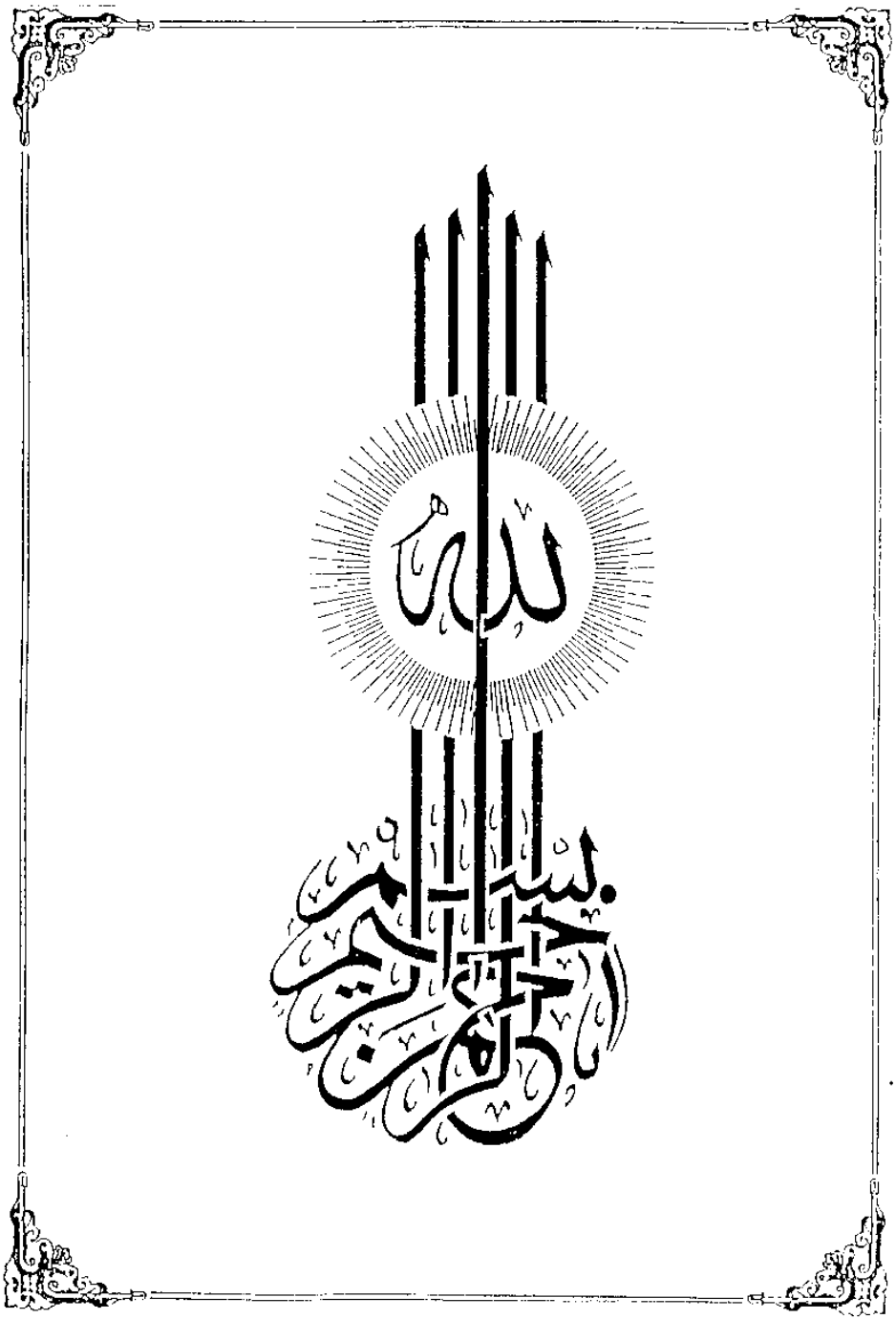

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INTRODUCTION

HISTORY OF PACING

The fundemental principles for utilization of artificial pacing were established as early as 1932 by Hymen(1). He suggested that the effect of intracardiac epinepharine injection was nonspecific, and that the effect was actually due to the injecting needle creating an irritable focus which produces a premature beat and stimulates beat formation. To test this theory, he constructed an electrical needle which he inserted into the right atrial wall of animals to act as a pacemaker, he used this needle with some apparent success in Guina Pigs during cardiac arrest by suffocation.

In 1951 Callaghan and Bigelaw(2) stimulated the canine sinoatrial node with transvenously introduced unipolar and bipolar catheters during hypothermic cardiac arrest, to increase the rate of the heart with intact atrioventricular conduction.

In 1952, Paul Zoll(3) succeeded for the first time in reviving an arrested human heart by the application of an external, transthoracic electric shock. He used pulses of 2 msec. duration and 75 to 150 volts in strength repeated 40 to 90 times per minute to produce cardiac systole. This technique proved to be life saving, and opened the era of wide spread

electrical stimulation of the heart.

In 1957, spurred by the impetus of surgically produced A-V dissociation, Weirich(4) and associates demonstrated the clinical feasibility of direct electrical stimulation of the myocardium. They inserted a stimulating wire to the myocardium through the closed chest and connected it to an external pulse generator to produce sustained repititive ventricular contractions.

In 1958, Furman(5) introduced a catheter electrode into the right ventricle and thus paved the way for transvenous endocardial electrostimulation of the heart.

In 1959, Furman and Schwedel(6) reported the acute and chronic therapeutic application of external transvenous cardiac stimulation in human beings when they used this technique on a large scale.

The development of transistor in 1959, enabled Elmquist and Senning (7) in sweden to implant a pacemaker subcutaneously, utilizing radio-frequency induction of pacemaker stimuli into an implanted recieving unit, which deliver stimuli to the myocardium via two stainless steel wires inserted to the myocardium after thoracotomy.

Mercury Zinc Cells enabeld Chardack and Greatbatch(8) in april 1960 to construct a completely implantable pacemaker unit independent of any external power source. The electrode was fixed to the myocardium via thoracotomy.

With demonestration that transvenous electrodes could be left in place indefinitely with continued stimulation, and that infection and dislocation of electrodes placed by thoracotomy could occur, with the high incidence of secondry thoracotomy for repair or replacement of leads, the first pulse generator and electrode for total transvenous implant were made available in 1965(9).

Initially, all pulse generator produced regularly timed stimuli yeilding a fixed cardiac rate, usually set between 60 and 70 beat per minute. In 1963 Nathan(10) and associates produced a pacemaker which stimulate the ventricle in response to atrial systole, with a cardiac rate sensitive to those physiologic (and sometimes pathologic) stimuli which control atrial rate.

The development of non-competitive (Demand) pacemakers about 1965(11) resolved the problem of whether heart block is fixed or intermittent. The demand pacemakers allow sinus rhythm to exist, and stimulate the heart when the cardiac rate fall below a predetermined rate.

REVIEW OF LITERATURE

I. INDICATIONS OF CARDIC PACING

The artificial cardiac pacemaker has been an important advance in the application of modern electronics to human biology and medicine.

Until about 1970 most physicians and surgeons would have considered carefully whether to implant a pacemaker in the absence of demonstrated Adams-Stokes attacks. Gradually a variety of minor neurologic lapses and prophylactic indications have become dominant. For purposes of clarity and utility in comprehensing Adams-Stokes Seizures, we should know that any condition which lead to diminution of left ventricular output, will produce episodic cerebrol ischemia, and any disturbance in the action of the heart that begins and ends abruptly will also cause interruption of cardiac output and hence cerebral ischemia will result, so symptoms may include dizziness, light headedness, brief lapses of consciousness, and fainting, with or without convulsions-all based on interruption of cardiac output. These definitions are useful as they direct modern therapy to the circulatory arrest and its consequences, with only secondery attention to the underlying cardiac rhythm.

Frequently the determination of whether a patient had any Adams-Stokes seizure, especially where it entails less neurolgic manifestation, could

proof most difficult. The diagnosis of aquired intermittent or partial complete heart block, 2:1 or Mobitz II block can be made electrocardiographically. Once made there are few who would dispute the need for cardiac pacing. Unfortunately, capture of the moment of A-V dissociation may not be easy and a careful neurologic evaluation may still be required. The presence of bifascicular block while strongly suggestive of a cardiac basis for accompanying syncope, does not preclude the possibility of neurologic disease. The problem becomes even more complex when dealing with bradycardia unrelated to heart block. For example, how often does atrial fibrillation with a ventricular rate below 50 in a patient 70 years of age or older cause symptoms of any sort and in the specific instance, is pacing required to prevent further bradycardia? The answer is probably that the relatively asymptomatic patient with that complex should be paced and medication such as digitalis and diuretics should be continued as needed(12).

How slow a bradycardia is pathologic in an elderly person with a sinus mechanism and A-V conduction? How slow is sinus bradycardia that requires treatment and what complex of symptoms should be considered as indicating the need for pacing? Clearly elderly patients can tolerate a sinus rate of 40-50 per minute apparently without deleterious effects. An "asymptomatic" sinus rate of 40 per minute or above in the older patients argues aganist pacemaker implant. Again if so, what duration of sinus arrest and asystole

become pathologic? We would probably all agree that a sinus arrest of 3.0sec, demands treatment, but does an episode of 1.5 to 1,8 seconds during an otherwise more rapid rate and without symptoms require pacing?

The decision concerning pacemaker implantation is often difficult and careful evaluation is required. Holter monitoring, carried out around the clock(13), observation with continuous monitoring in a coronary care unit. and provocative tests such as bundle of His studies(14,15) and sinus node recovery time after rapid atrial pacing(16) and programmed cardiac stimulation may be required. Even with all these techniques a definitive answer concerning whether to pace or not to pace may not be available. If necessary temporary cardiac pacing under observation, may be required to resolve the problem of whether an increase in a sinus bradycardia of 40 per minute to 60 or 70 changes a patient's mentation, cardiac compensation, or feeling of well being. Often only such an approach, with the assessment by other colleagues, the patient himself and family will enable a decision to be made.

In general the current concepts for cardiac pacing today, put forwards the followings as indications for pacing: (12, 17, 18, 44, 62, 66, 72).

A- Temporary pacing.

1- Symptomatic second degree, trifascicular or complete A-V block especially

during acute myocardial infarction requires temporary pacing. It should be noted that A-V block per se does not require pacing.

2- Symptomatic and drug resistant, sinus arrhythmia including sinus bradycardia, sinus arrest and sino-atrial block.

3- Left bundle branch block, bifascicular block and trifascicular block with acute anterior wall infarction usually require a short term pacing because the above findings are often forerunners of complete heart block.

4- Emergency treatment for Adams-Stokes syndrome and symptomatic bilateral bundle branch block.

5- Symptomatic digitalis induced trifascicular or complete AV block or SA block.

6- Before or during implantation of permenant pacemaker.

7- Therapeutic trial for intractable congestive heart failure, cardiogenic shock and cerebral or renal insufficiency.

8- Prophylactic pacing during major surgery when the Adams-Stokes syndrome is anticipated.

9- Drug resistant and refractory tachycardias.

B. Permenant Pacing.

1- Symptomatic (Mobitz II) second degree and complete AV block.

2- Symptomatic chronic and drug resistant sinus arrhythmia including sinus bradycardia and sinus arrest.

3- Complete AV block in acute myocardial infarction lasting more than 2-3 weeks.

4- Symptomatic bilateral bundle branch block.

5- Sick sinus syndrome.

6- Hypersensitivity carotid sinus syndrome.

7- Recurrent Adams-Stokes attacks.

8- Recurrent drug resistant tachyarrhythmia.

Sick Sinus Syndrome

Dysfunction of the sino-atrial node like sino-atrial block and paroxysmal atrial fibrillation had been described in 1916 by Levine(19), whereas Short(20) described the alternating sinus bradycardia-tachycardia phenomenon in 1954. Lown in 1967,(21) first used the term sick sinus syndrome to describe certain arrhythmias following direct current cardioversion where a chaotic atrial rhythm, changing P wave contour, bradycardia, multiple recurrent ectopic beats with runs of atrial and nodal tachycardia occured. Ferrer(22) in 1968 studied the sinus nodal dysfunctions more closely and reported that these dysfunctions, referred to as the sick sinus syndrome, were not rare, but difficult to diagnose and defined it as including the following: sinus bradycardia, sinus arrest leading to either asystole or ectopic rhythm, chronic atrial fibrillation with slow ventricular response and junctional bradycardia. After Ferrer's description many reports followed and

various terms were used to describe this phenomenon like, inadequate sinus mechanism(23), sluggish sinus node syndrome(24), sinoatrial syncope(25),and bradycardia-tachycardia syndrome(26).

In the last few years, sick sinus syndrome as an indication for cardiac pacing make about 20-40 percent of pacemaker indications(12,27).

The arrhythmias of the sick sinus syndrome can be classified in:

A- Generator failure of the sinus node, with:

1- Intermittent or permenant sinus bradycardia;

2- Intermittent or permenant cessation of sinus rhythm(sinus arrest) with or without atrial (Flutter or fibrillation), junctional or ventricular escape rhythms,

3- Combination of the above in the bradycardia-tachycardia syndrome;

4- Permenant cessation of the sinus rhythm and replacement by atrial fibrillation often associated with a slow ventricular rate due to accompanying atrio-ventricular nodal disease; with

5- No or very slow sinus rhythm after cardioversion.

B- Sino-atrial exit block, with or without atrial, junctional or ventricular escape rhythms.

Sick sinus syndrome is not a disease of the sinus node but also a diffuse