INCIDENCE OF CARDIAC ARRHYTHMIAS IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION AFTER DISCHARGE FROM CORONARY CARE UNIT

Thesis

Submitted in Partial Fulfilment of
The Master Degree in *Cardiology*

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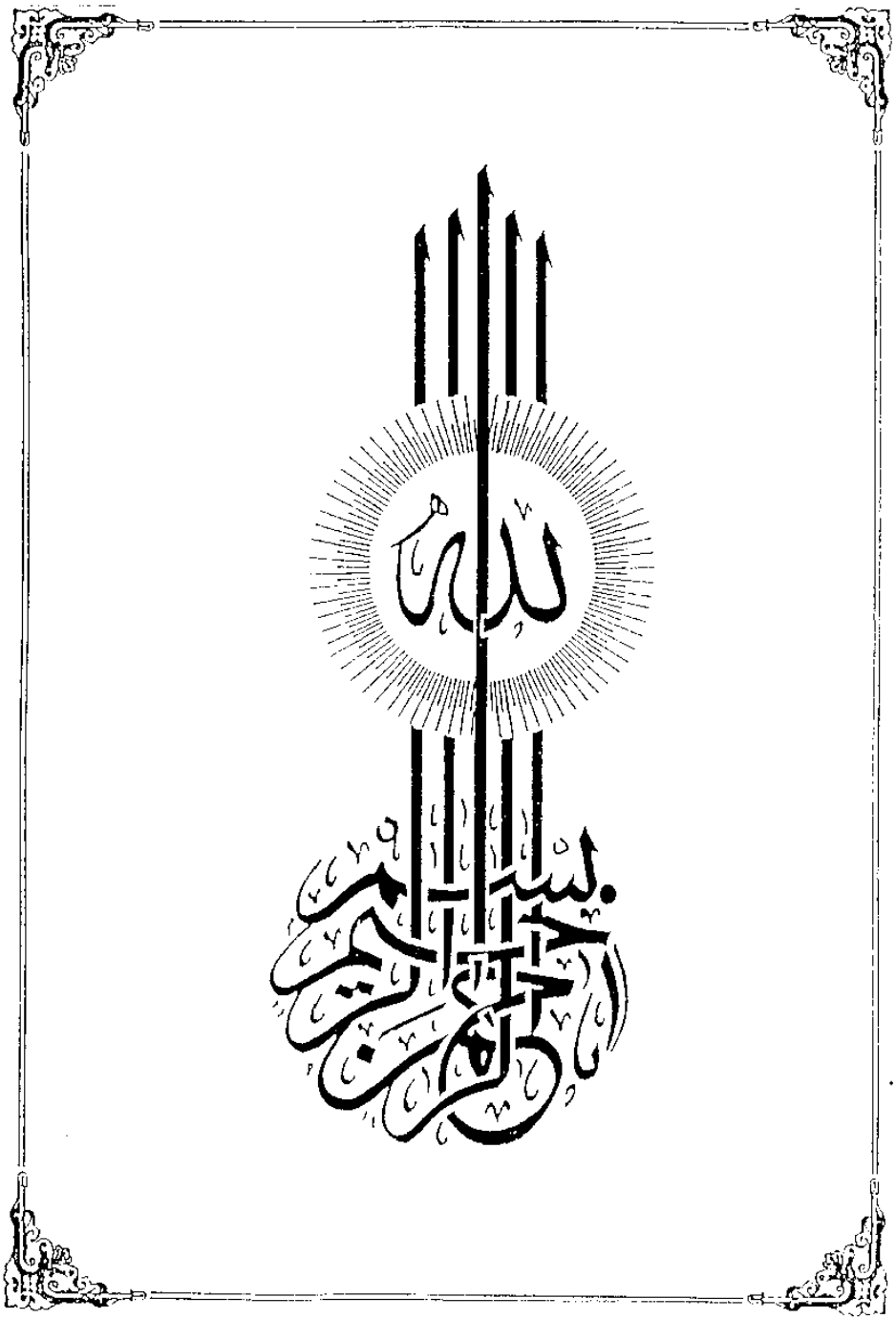
1987

TO MY FAMILY



ACKNOWLEDGEMENT

With my great pleasure I wish to acknowledge my indebtedness to Prof. Dr. ALI RAMZI, he had spared a lot of his valuable time to help and direct me by useful instructions. I have been fortunate to be under his supervision.

I wish to express my sincere gratitude to Prof. Dr. MAMDOUH EL–ASHRY, who suggested the idea of this work and who had so kindly and so carefully assisted and collaborated with me. To him I am so greatful.

I owe a great debt to Prof. Dr. RAMEZ GUINDY for his perfect and close supervision and for his unlimited cooperation throughout this work giving me his intelligent instructions kindly and friendly.

I wish to express my sincere gratitude to the Professor, who realy taught and still teaching me a lot in this work and in Cardiology, my beloved father Prof. Dr. EMIL SADEK, Head of Cardiology Department, Maadi Military Hospital. Thank you father for all you have done to me.

I owe a great debt to Prof. Dr. AHMED HELAL, Head manager of Kobri El–Kobba Hospital, Prof. Dr. ISMAIL SABRY, Head of Cardiology department, Kobri El–Kobba Hospital, Dr. M. WAHEEB, Lecturer of Cardiology, Military Medical Academy and to Dr. SAYED ABD EL–HAFIZ, Lecturer of Cardiology, Military Medical Academy, for their continuous advices. Without their help this work could hardly be accomplished.

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INTRODUCTION & AIM OF WORK

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Cardiac arrhythmias are considered one of the serious complications of acute myocardial infarction. So, patients with acute myocardial infarction are admitted to coronary care units, as a major contribution of coronary care unit in the management of acute myocardial infarction has been the early detection and prompt suppression of life threatening arrhythmias *(Shah et al., 1977)*. Continuous monitoring and aggressive management of rhythm disturbances during the initial period in hospital have significantly reduced mortality in these patients *(Vismara, et al., 1975)*.

After discharge from the coronary care unit this monitoring is somewhat deficient. Patients who appear to be making a satisfactory recovery after myocardial infarction are still at risk of cardiac events, especially in the first 12 months. *(Bourke and Cowan, 1986)*.

Sudden unexpected deaths occurring in hospital after discharge from coronary care unit have suggested that arrhythmias may be a continuing problem. The development of portable electrocardiographic monitoring has provided a means of continuous recording and assessment of cardiac rhythm in ambulatory patients *(Vismara, et al., 1975)*.

Portable electrocardiographic monitoring of post acute myocardial infarction patients, revealed arrhythmias in high percentage of patients. Thus, during the early post coronary

care unit period following acute myocardial infarction, as in the coronary care unit, serious cardiac arrhythmias occur frequently and may be undetected by routine methods. These data indicate the need for more intensive arrhythmia monitoring in patients with acute myocardial infarction during the hospital period after discharge from the coronary care unit *(Vismara, et al., 1972)*.

The principal purpose of this work is to evaluate arrhythmias in the late hospital period of acute myocardial infarction, to assess incidence, types and possible etiology of these arrhythmias.

Diagnosis of myocardial infarction will be made on clinical findings, resting E.C.G. and cardiac enzymes.

Detection of arrhythmias will be done using continuous ambulatory 24 hours electrocardiographic recordings "Holter monitor", the obtained results will be discussed in comparison with similar studies.

REVIEW OF LITERATURE

ELECTROPHYSIOLOGIC MECHANISMS FOR CARDIAC ARRHYTHMIAS IN ACUTE MYOCARDIAL INFARCTION

As described by Hoffman and Rosen the mechanisms for arrhythmias are classified into 3 main classes namely arrhythmias caused by abnormal impulse generation, arrhythmias caused by abnormal impulse conduction and lastly arrhythmias caused by simultaneous abnormalities of impulse generation and conduction (Table, 1) *(Hoffman and Rosen, 1981).*

In studying arrhythmias of myocardial infarction, two distinct phases of ventricular arrhythmias following acute myocardial infarction are recognized. These phases are the early phase and the late phase of ventricular arrhythmias, each of which has different mechanisms *(El–Sherif, Scherlag, et al., 1977).*

The early phase of arrhythmias after myocardial infarction is subdivided further into two stages, the first stage begins almost immediately after infarction and lasts up to a few hours. This stage could be called the "pre-hospital" phase. The second stage that begins 12 to 24 hours after infarction and lasts about 24 hours. This stage is related to the "in-hospital" arrhythmias *(Wit and Bigger, 1975).*

Coronary artery occlusion does not produce the same effects on metabolism and ultrastructure of ventricular muscle cells and Purkinje fibers. The different time course for the biochemical and ultrastructural changes in

Table (1): Mechanisms of Arrhythmias. *(Hoffman and Rosen, 1981).*

I Abnormal Impulse Generation	II Abnormal Impulse Conduction	III Simultaneous abnormalities of Impulse Generation and Conduction
A. Normal automatic mechanism 1. Abnormal rate a. Tachycardia b. Bradycardia 2. Abnormal rhythm a. Premature Impulses b. Delayed Impulses c. Absent impulses B. Abnormal automatic mechanism. 1. Phase 4 depolarization at low membrane potential 2. Oscillatory depolarization at low membrane potential preceding upstrokes C. Triggered activity 1. Early after-depolarizations 2. Delayed after-depolarization. 3. Oscillatory depolarization at low membrane potentials following action potential upstrokes.	A. Slowing and block 1. Sinoatrial bock 2. Atrioventricular block. 3. His bundle block 4. Bundle branch block B. Unidirectional block & re-entry 1. Random re-entry a. Atrial muscle b. Ventricular muscle 2. Ordered re-entry a. Sinoatrial node and junction b. AV node and junction c. His-Purkinje system. d. Purkinje fiber muscle junction e. Abnormal AV connection. 3. Summation and Inhibition. C. Conduction block and reflection	A. Phase 4 depolarization & impaired conduction 1. Specialized fibers. B. Parasystole.

ventricular muscle and in subendocardial Purkinje fibers following complete coronary artery occlusion must be responsible for these two stages of ventricular arrhythmias. Arrhythmias that occur within minutes of occlusion and that may last for several hours, appear to be primarily dependant on the immediate effects of ischaemia on ventricular muscle, while arrhythmias occurring after that may be a result of the delayed effect of ischaemia on the Purkinje system *(Wit and Bigger, 1975)*.

The biochemical and ultrastructural changes that accompany the initial phase of myocardial infarction have a wide range of effects on the electrophysiological properties of ventricular muscle cells. Action potentials generated in the presence of severe hypoxia, increased lactate, decreased intracellular pH, or partial depolarization resulting from changes in the properties of the cell membrane may be slow response action potentials *(Wit and Bigger, 1975)*.

These conditions which lead to marked reduction in resting membrane potential, partly or completely abolish or inactivate the normal fast response action potential, however the slow action potential is not inactivated.

The very slow rate of depolarization of the slow response is associated with very slow propagation. The slow response also has a low safety factor for conduction and is prone to block at branch points in the cardiac syncytium where it encounters impediments to forward conduction. Unidirectional conduction block is often associated with the slow response. These

properties allow reentry to occur in very short path lengths even though cardiac muscle is refractory for such a long period after excitation. So, the most significant cause of early or immediate ventricular arrhythmias is the occurrence of slow response action potentials or severely depressed fast response action potentials in ischaemic myocardium *(Wit and Bigger, 1975).*

Infarcting ventricular muscles may demonstrate an abnormal type of automaticity when their resting membrane potential is low. In addition to spontaneous depolarization during phase 4 resulting in automatic activity, there is membrane oscillations or after-potentials occurring during phase 2 of the action potential of ventricular muscle fibers in infarcted ventricle. These oscillations occur at membrane potentials of -50 mV or less and may result from slow inward current. If they could propagate to surrounding myocardium, an impulse propagating in the infarct would be necessary to initiate continuous spontaneous activity of this type *(Wit and Bigger, 1975).*

Between 6 and 10 hours after coronary artery occlusion, lipid droplets begin to accumulate in the cytoplasm of the Purkinje fibers, suggesting that changes in metabolism are taking place. After 12 to 24 hours, numerous aggregates of lipid droplets are present in the cytoplasm and the electrical activity of these Purkinje fibers is abnormal *(Friedman et al., 1975).*

The marked change in the subendocardial Purkinje fiber action potential duration in the infarct and the disparity in repolarization time between these action potentials and the action potentials of Purkinje fibers in the non-infarcted areas may also be a direct cause of cardiac arrhythmias since this disparity facilitates the occurrence of reentry in the subendocardial Purkinje network which survives in the regions of extensive infarction *(Friedman et al., 1973).*

Boineau and Cox noted local areas of sustained excitation after the onset of ventricular activation which act as a source of re-entrant activity and were associated with premature ventricular complexes after coronary artery occlusion *(Boineau and Cox, 1973).*

Abnormal automaticity occurs in Purkinje fibers that survive on the subendocardial surface of myocardial infarction *(Wit and Rosen, 1983).*

There is no doubt that with infarction, some fibers loose maximum diastolic potential and show automatic firing due to phase 4 depolarization at a low level of membrane potential *(Spear et al., 1979).*

Early after depolarizations leading to triggered activity may be present in an infarcted region of the ventricles.

Delayed after depolarizations may also occur in Purkinje fibers surviving on the subendocardial surface of infarcts *(Wit and Rosen, 1983).*

The emergence of after depolarizations may precipitate ventricular arrhythmias *(Cranefield, 1977).*

Parasystolic foci initiating premature ventricular complexes must be considered as a possible important mechanism for arrhythmias in myocardial infarction *(Tye et al., 1979).*

The late myocardial infarction period as described in electrophysiologic studies begins from the third day of the onset of myocardial infarction *(El Sherif, Scherlag et al., 1977 – Miller and Josephson, 1985).*

Electrophysiologic mechanisms for arrhythmias during this period are enhanced automaticity of the Purkinje cell which may result in automatic tachycardia or reentrant arrhythmias if impaired conduction and heterogenicity of refractoriness are present *(Marchlinski et al., 1983).*

In a long series of experiments, *El–Sherif* et al, studied reentrant ventricular arrhythmias in late myocardial infarction period 3-7 days after the onset of the infarction and they found that after the subsidence of the late stage of enhanced automaticity, still there is a high propensity for reentrant ventricular arrhythmias *(El–Sherif, Scherlag et al., 1977). Hope et al., 1980* reported that these reentrant ventricular arrhythmias arise in epicardium overlying the transmural infarction zone 4-5 days after myocardial infraction. After acute occlusion of the coronary artery, blood flow is reduced more in the subendocardium and resistance to flow in the