شبكة المعلومات الجامعية

بسم الله الرحمن الرحيم

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
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بعض الوثائـــــــق الاصليـــــة تالفـــة

بالرسالة صفحات لم ترد بالاصل

TIME COURSE & PROGNOSTIC VALUE OF LATE POTENTIALS AFTER THEOMBOLYTIC THERAPY IN ACUTE MYOCARDIAL INFARCTION

Thesis

Submitted in partial Fulfillment for the requirement of
(M.D.) Degree in Cardiology

By

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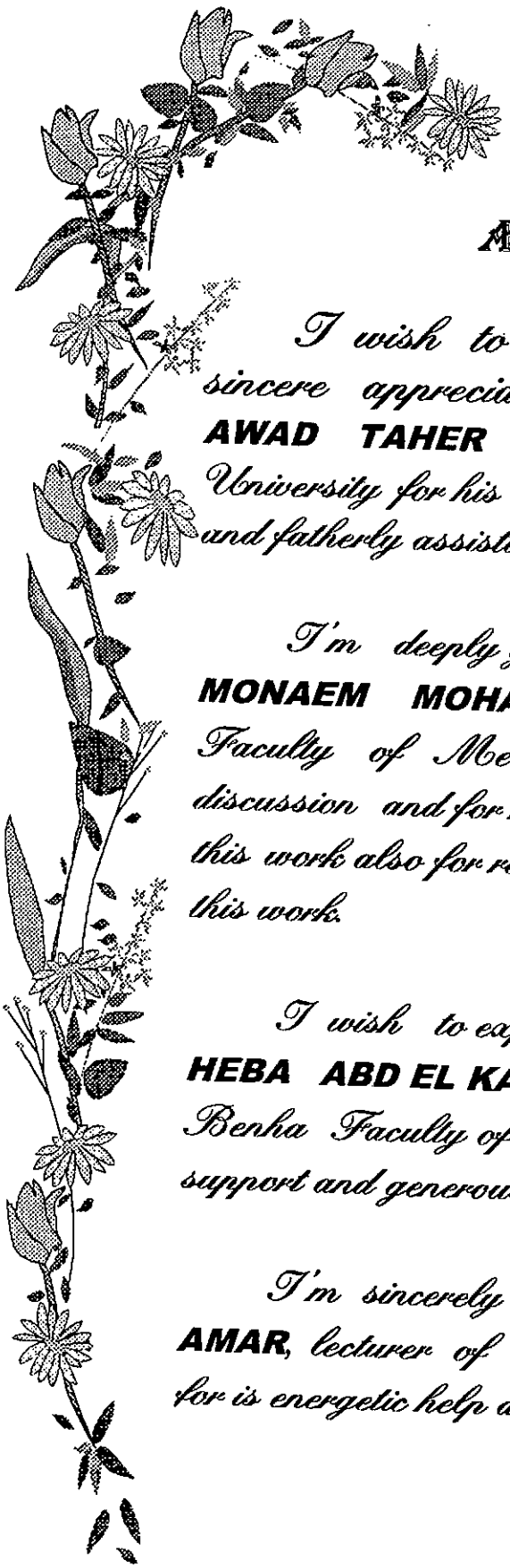
CONTENT



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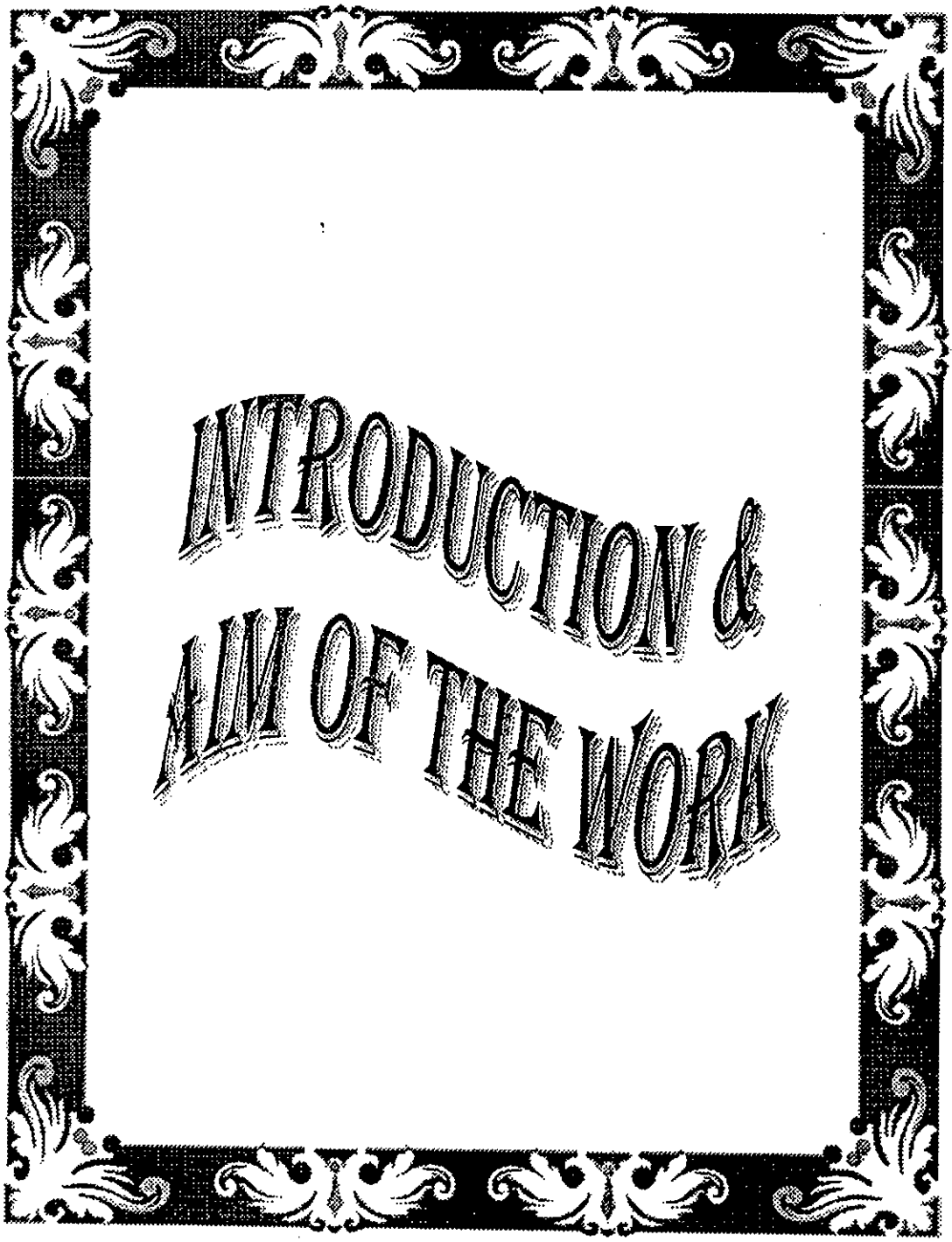

INTRODUCTION &
AIM OF THE WORK

INTRODUCTION

Acute myocardial infarction is a fatal disease and is responsible for high percent of deaths along its short and long terms.

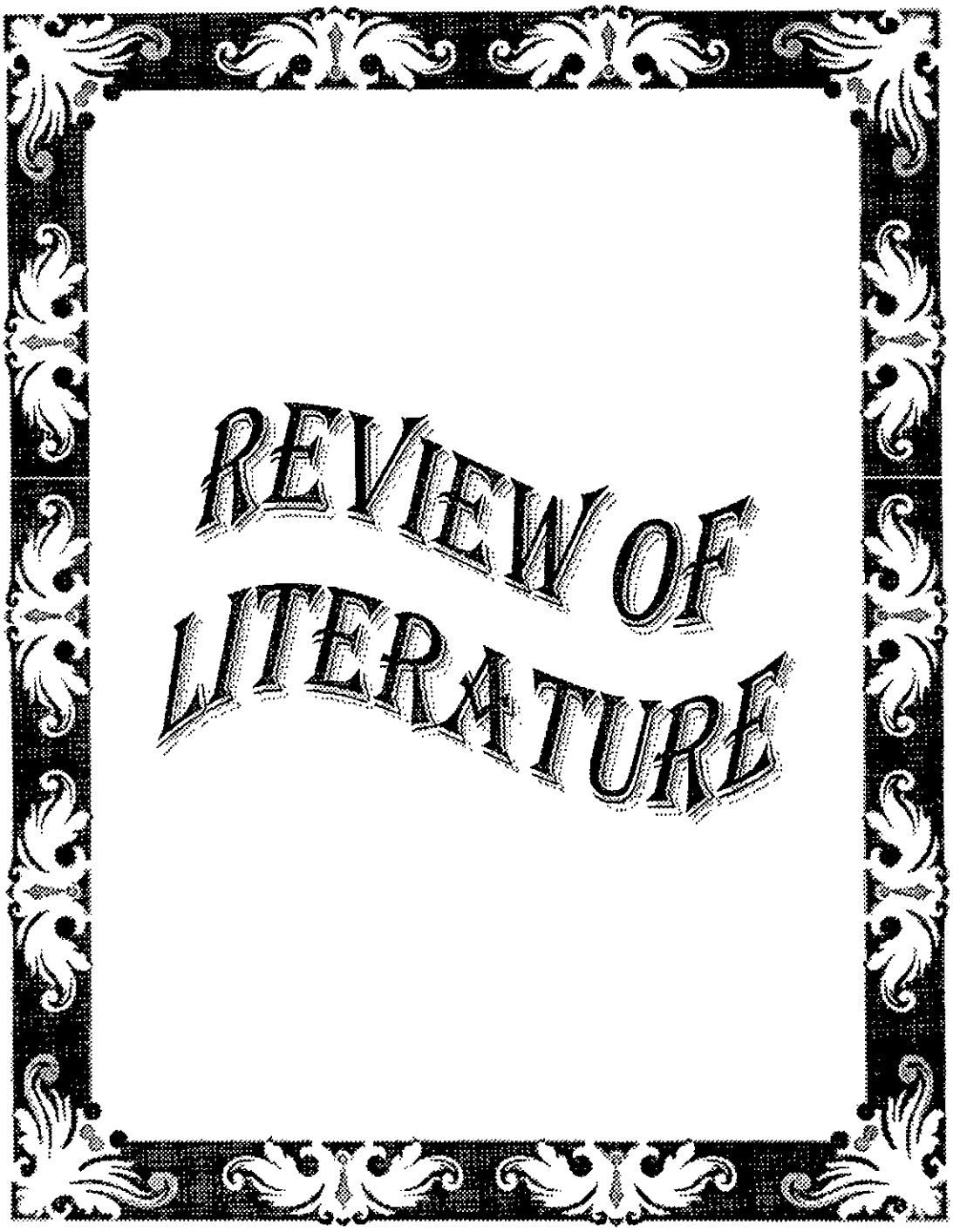
The overall incidence of rhythm disturbance in myocardial infarction may actually be as high as 100 percent and it is affected mostly by the extent of underling infarction size ***(Hinkel et al., 1989).***

There is increasing role that late potentials after QRS complex which results form delayed excitation in marginal region of infarction on SAECG,can identify patients at high risk for sudden death in post infarction period, Now late potentials are considered as independent risk factor ***(Gomes et al., 1989).***

Also there is increasing evidence that that thrombolytic therapy may decrease the ventricular ectopy and late potentials by reducing electrical instability which may improve the overall prognosis in myocardial infarction ***(Gang et al., 1989).***

AIM OF THE WORK

To study time course of late potentials as recorded by SAECG in cases of acute myocardial infarction and to determine its relation and the effect of thrombolytic therapy & prognosis.


REVIEW OF
LITERATURE

VENTRICULAR LATE POTENTIALS

Definition:

Ventricular late potentials are microvolt signals that are part of terminal QRS complex and persist into the ST segment. They correspond to areas of delayed ventricular activation which are manifestations of slowed conduction velocity ***(Jarret and Flower's 1991).***

The standard level of amplification as used for routine electrocardiograph (ECG) reading, dose not make use of the full range of information contained in the ECG - Signal on the body surface. Under physiological and pathologic conditions ventricular late potentials or low amplitude signals can be recorded directly from the epicardium or endocardium of the heart ***(Breithardt et al., 1990).***

Due to progress of the last decade, recording of these low amplitude signals from body surface by "High resolution ECG) has been achieved. The most widely used technique is the signal averaging technique which was used as early as in 1973 by ***Stopczyk et al (1973)*** to perform surface recording of the electrical activity in man later on, the technique of signal averaging has attracted interest in detection of delayed ventricular activation on body surface ***(Breithardt et al 1960- Simson et al 1980- Simson 1981 – Breithardt et al., 1981).***

Experimental and clinical studies have provided evidence that myocardial infarction may leave a zone of electrically abnormal ventricular myocarduim that may be the site of origin of ventricular tachycardia. ***(El-Sherif et al 1977).***

This tissue is mostly located at the border zone of a previous myocardial infarction and is characterized by islands of relatively viable muscle alternating with areas of necrosis and later fibrosis. Abnormal ventricular conduction during sinus rhythm has been observed in regions bordering the infarct and appears related to the development of ventricular tachycardia ***Grander et al., (1985). Klieg et al., (1982).*** Implicate reentrant mechanisms, at least in part, in the genesis of sustained ventricular tachycardia complicating ischemic heart disease, and that areas of delayed epicardial activation, fractionation and double potentials are characteristic finding for ventricular mapping during sinus rhythm and presumably constitute the reentray substrate for development of these arrhythmias ***(Klein et al., 1982, Josephson and Wit 1984, de Bakker et al., 1988).*** Myocardial activation may be delayed because the pathway of excitation is lengthened, conduction velocity is slowed or both ***(Granders et al., 1985).***

Clinically most myocardial infarctions do not result in complete transmural necrosis ***(De - Bakker et al., 1990).*** The amount of surviving myocardium is viable and may be localized in subepicardial, subendocardial and transmural regions. Islands of fibrosis create barriers, that lengthen the excitation pathway. The increased separation of myocardial bundles and disruption of their parallel orientation by fibrosis distort ventricular activation ***(Grander et al., 1985).*** Extra cellular electrograms recorded from the endocardial surface from such bundles usually have small amplitudes because of the intervening layers of fibrous tissue and small diameter of muscle bundles. When individual bundles are separated by connective tissue septa, heterogeneous patterns of activation