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بالرسالة صفحات

لم ترد بالأصل

The Relation between Heart Rate Variability Parameters and Left Ventricular Systolic Function in Prediction of Prognosis after Acute Myocardial Infarction

B 11 22.

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Submitted by
Fouad Ali Ibrahim El-Alfy
M.B., B.Ch., Diploma of Cardiology

Supervisors

**Prof. Dr.
Fathi Maklady**
Professor of Cardiology
Faculty of Medicine
Suez Canal University

**Prof. Dr.
Ahmed El-Hawary**
Professor of Cardiology
Faculty of Medicine
Suez Canal University

***Faculty of Medicine
Suez Canal University
2000***



وَعَلَّمَكَ مَا لَمْ تَكُنْ تَعْلَمُ وَكَانَ
فَضْلُ اللَّهِ عَلَيْكَ عَظِيمًا

(النساء - الآية ١١٣)

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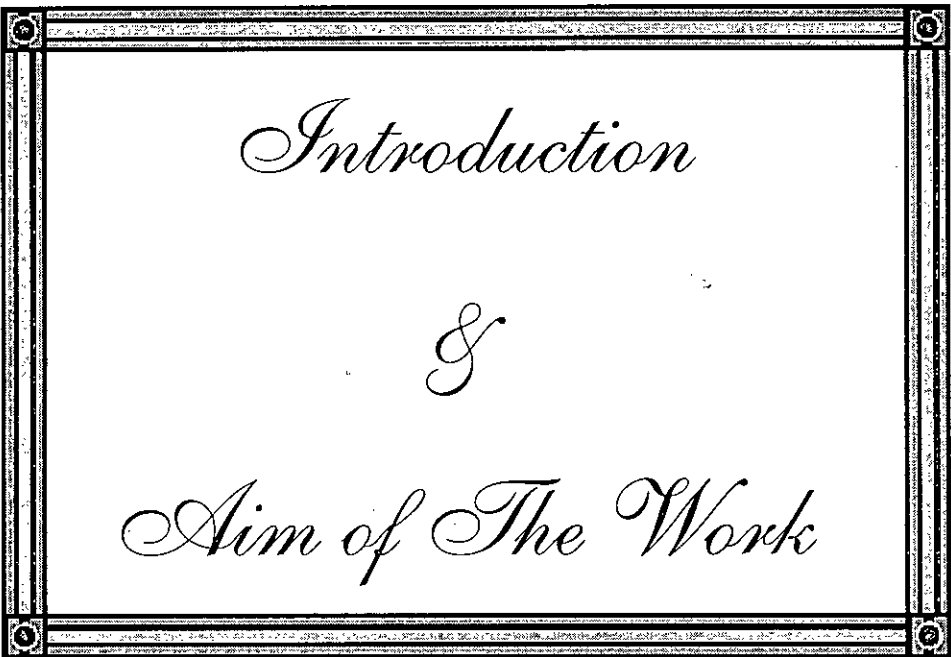
Lastly, I would like to offer my warm thanks to all those who helped me to prepare this work.

LIST OF ABBREVIATIONS

AMI	Acute myocardial infarction
AMP	Adenosine monophosphate
AV	Atrioventricular
BMI	Body mass index
CFM	Colour flow mapping
CPL	Creatine phosphokinase
ECG	Electrocardiogram
EDV	End diastolic volume
EF	Ejection fraction
ESV	End systolic volume
HF	High frequency
HRV	Heart rate variability
LDH	Lactic dehydrogenase
LF	Low frequency
LV	Left ventricle
LVEF	Left ventricular ejection fraction
MI	Myocardial infarction
RMSSD	The square root of the mean squared differences of successive NN intervals
SDANN	Standard deviation of the average NN interval
SDNN	Standard deviation of all normal R-R interval
SDNNI	Mean of standard deviation of NN interval for 5 min segments
ULF	Ultra low frequency
VLF	Very low frequency

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Introduction

&

Aim of The Work

INTRODUCTION

The last two decades have witnessed the recognition of significant relationship between the autonomic nervous system and cardiovascular mortality including sudden death. Experimental evidence for the association between a propensity for lethal arrhythmias and signs of either increased sympathetic or reduced vagal activity has encouraged the development of quantitative markers of autonomic activity. Heart rate variability (HRV) represents one of the most promising markers (*Levy et al.*, 1994).

A reduced heart rate variability has been shown to be a powerful predictor of subsequent mortality in patients surviving an acute myocardial infarction (*Zaunetti et al.*, 1996).

The mechanism by which HRV is transiently reduced after MI and by which a depressed HRV is predictive of neural response to acute MI is not yet defined, but it is likely to involve dearrangement in the neural activity of cardiac origin. One hypothesis involves cardio-cardiac, sympatho-sympathetic and sympath-vagal reflexes (*Schwartz et al.*, 1988), which suggest that the change in the geometry of a beating heart due to

necrotic and non-contracting segments may abnormally increase the firing of sympathetic afferent fibers by mechanical distortion of sensory ending (*Brown and Malliani, 1971*). This sympathetic excitation attenuates the activity of vagal fibers directed to the sinus node.

Another explanation applicable to marked reduction of HRV is the reduced responsiveness of sinus nodal cells to neural modulation (*Malliani et al., 1994*).

Most studies of HRV have used 24-hour electrocardiograph recording (Holter recording) are often not feasible for wide-scaled epidemiological studies and may be unnecessary. In men after acute MI, the HRV measures calculated from 2-15 minutes segments were remarkably similar to those calculated over 24 hours, and provided predictive information similar in strength to the entire record (*Bigger et al., 1993*).

Traditionally, HRV used for risk stratification after MI has been assessed from 24-hour recordings. HRV measured from short-term electrocardiogram recordings also provides prognostic information for risk stratification following MI but whether it is as powerful as that from 24-h recordings is uncertain. But HRV

measured from short-term recordings is depressed in patients at high risk (*Bigger et al.*, 1993).

Also it was noted that the mildest disturbance of ventricular performance in ischemic heart disease consists of focal abnormalities of contraction and relaxation present only when ischemia is induced by a stress such as exercise, but with maintenance of global ventricular function, the latter is reflected in normal end diastolic volume, end systolic volume, stroke volume and EF, as already noted. This discrepancy between regional wall motion and ventricular function may result from compensatory hyperfunction of normal segment of the ventricle. As ischemic heart disease progress particularly if myocardial infarction occurs, wall motion abnormalities at rest develop. A more severe disturbance is characterized by a normal EF at rest which fails to rise during exercise, as the fraction of the ventricle which is ischemic increase further, EF decline and filling pressure rises, at first only during stress and even at rest (*Sheehan et al.*, 1982).

Also it was shown that cardiac enlargement with or without heart failure is associated with marked disturbances of parasympathetic as well as sympathetic function. The

parasympathetic effect on sinoatrial node automaticity is markedly reduced in patients with heart disease who also exhibit less heart rate slowing for any given elevation of systemic arterial blood pressure than do normal subjects (*Higgins et al.*, 1972).

The ratio of systolic volume to end diastolic volume i.e. the EF is a global index of the extent of ventricular fiber shortening. Based on a number of empirical studies, EF is sought to provide a useful measure of overall left ventricular pump function (*Marving et al.*, 1985).