EVALUATION OF ST SHIFT IN MEN WITH SYSTEMIC HYPERTENSION AND WITHOUT CLINICAL EVIDENCE OF CORONARY ARTERY DISEASE BY 24-HOUR HOLTER MONITORING

THESIS

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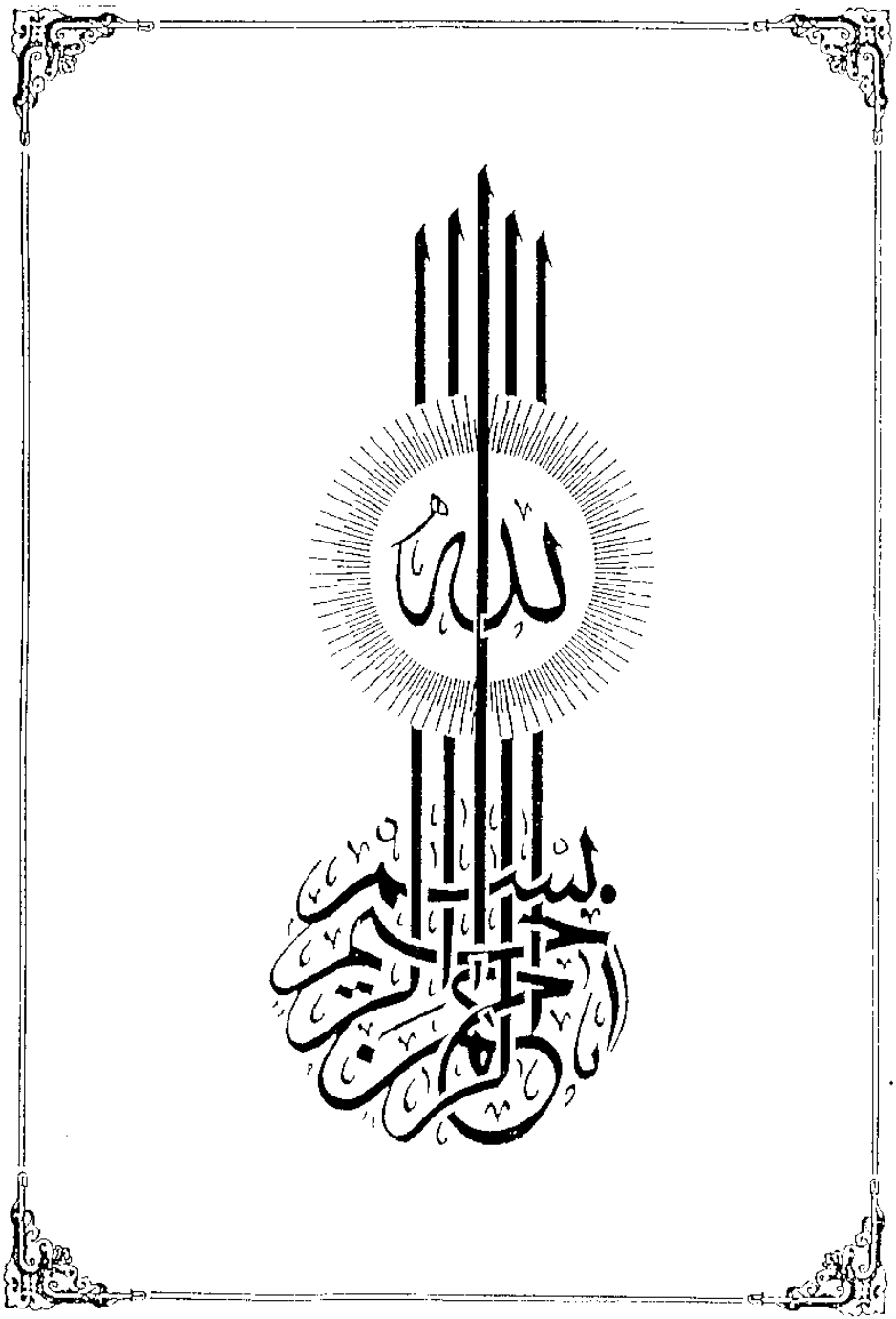

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CONTENTS

INTRODUCTION

AND AIM OF THE WORK

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Hypertensive heart disease is a major risk factor for the development of coronary artery disease and sudden cardiac death **(Koren et al., 1991)**.

Recent interest has focused on the presence of potentially malignant ventricular arrhythmias in patients with systemic hypertensive disease because sudden death is caused by ventricular fibrillation or rapid tachycardia in almost all monitored cases **(Loaldl et al., 1983)**.

In the presence of left ventricular hypertrophy ventricular arrhythmias are common in treated and temporarily untreated patients **(Levy et al., 1987)**.

Recently, **James et al., (1989)** described a 26 % prevalence of repetitive arrhythmias in untreated hypertensives; hypokalemia instead of left ventricular hypertrophy was predictive for presence of ventricular arrhythmia.

An association between hypertensive disease and fatal arrhythmias has also been gathered from victimes of sudden cardiac death. In these patients, hypertensive disease or necropsy findings of left ventricular hypertrophy were common **(McLaran et al., 1987; Reichenbach et la., 1977)**.

In addition, most of the studies also stressed the uncertain role of myocardial ischemia because the high prevalence of ST segment depression in sudden cardiac death patients **(Tubau et al., 1987).**

Clinically, the coincidence of coronary artery disease and hypertension is known to carry an increased risk of sudden death **(Stamler et al., 1989).**

However, myocardial ischemia as a potential link to sudden death frequently is not symptomatic in hypertensive patients **(Stamler et al., 1989) (Yurenev et al., 1990).**

Recent use of stress testing in angiographically studied hypertensive patients indicates a high prevalence of asymptomatic myocardial ischemia **(Yurenev et al., 1990).**

Recently, ambulatory monitoring of the electrocardiogram during daily life has been validated as a rapid and reliable analysis of the frequency and duration of myocardial ischemic episodes from 24-hours Holter recordings. This method enables the analysis of ST-T wave changes with a high degree of accuracy **(Nademanee et al., 1986).**

Aim of the work :

The aim of this work is to assess prevalence and interaction of ventricular arrhythmias and significant ST segment shift during 24 hours Holter recording in patients with systemic arterial hypertension.


REVIEW
OF LITERATURE

coronary arteriography. Thirty-three (2.2 percent) of the 1,390 crewmen had significant coronary artery disease, including 18 with multivessel disease. On the basis of these two studies, it would appear that 2 to 4 percent of middle-aged men have asymptomatic significant coronary artery disease **(Cohn, 1980).**

Bruce et al., (1977) estimated that 3.5 percent of an asymptomatic male population had a seven fold risk of any subsequent coronary heart disease event (angina, myocardial infarction or sudden death) on the basis of abnormal exercise test results.

Epidemilogic studies indicate that more than 25 percent of myocardial infractions may be asymptomatic **(Kannel and Abbott 1984).**

Classification of silent myocardial ischemia :

Cohn, (1985) has proposed a three-part classification for silent myocardial ischemia.

Type I silent myocardial ischemia :

This includes totally asymptomatic subjects, often detected by a screening exercise test. Indeed, some of these

patients do not even experience pain in the course of myocardial infarction.

Epidemiological studies of sudden death, clinical and postmortem studies of patients with silent myocardial infarction, and studies of patients with chronic angina pectoris suggest that many individuals with extensive coronary artery obstruction do not have angina pectoris in any of its recognized forms (stable, unstable or variant) **(Parmley, 1989)**.

These individuals included in type I silent ischemia, may be considered to have a defective anginal "warning system". Both the patient and physician may be unaware of the presence of ischemic heart disease until a fatal event ensues or an old infarction is detected on routine electrocardiogram **(Cohn, 1980)**.

Type II silent myocardial ischemia :

This type of silent ischemia occurs in patients asymptomatic after a myocardial infarction. They are usually identified by post infarction exercise testing soon after the infarction. Also, recently, Holter monitoring has identified silent ischemia in this population.

Type III silent myocardial ischemia :

This include patients with angina who have additional episodes of silent myocardial ischemia.

Such patients have been extensively studied with ambulatory electrocardiographic monitoring and it is estimated that 75 percent of all patients with angina have some degree of silent myocardial ischemia.

Pathophysiology of silent myocardial ischemia :

The mechanism of cardiac pain is still poorly understood. Three separate theories have been proposed in an effort to explain why significant myocardial ischemia may fail to produce angina pectoris.

Theory number 1: Global deficiency in pain perception:

A generalized defect in pain perception (somatic as well as visceral) would explain the total absence of symptoms in type 1 patients and the incomplete pain awarness of the type III patients. Forty two patients (22 with angina, 20 with silent myocardial ischemia) exhibited significant ST-segment depression during exercise stress testing, no significant difference were found between angiographic data or coronary

risk factors. Their somatic pain perception was studied with different types of pain-reception modalities, including electric shock, cold pressor stimulation, and tourniquet-induced forearm ischemia. Significant differences in pain perception were measurable, with the silent ischemia patients demonstrating a higher pain threshold and greater tolerance for all types of pain. This explain lack of pain in patients with asymptomatic myocardial ischemia **(Droste, 1983)**.

There is evidence that endorphins, endogenous opioid peptides, play a significant role in modifying individual pain threshold **(Buchsbaum et al., 1981)**.

This finding led to the use of naloxone a specific opiate antagonist, with the expectation that this substance could provoke the appearance of anginal pain in patients with silent myocardial ischemia. Two studies using naloxone in patients with exercise-induced ischemia brought controversial results.

Droste and Roskamm (1984) reported that the appearance of anginal pain in 2 out of 10 patients with silent ischemia. Conversely, in a study performed by **(Ellestad and Kuan 1984)** naloxone failed to induce anginal pain in 10 patients with silent ischemia.

These data support the view that endorphinic activity does not play an essential role in the Pathophysiology of silent myocardial ischemia.

Theory number 2 : Anatomic changes in pain receptors and nerves :

A specific deficiency in cardiac nociception has been proposed as an explanation of silent myocardial ischemia. **(Faerman, et al., 1977)**. The nociceptive pathways projecting from heart may be destroyed by substantial myocardial infarction, very diffuse coronary heart disease or polyneuropathy at a more central location **(Dorste 1983)**.

Central inhibition of potentially painful stimuli may explain silent myocardial ischemia. Central transmission of potentially painful may be simultaneously (1) inhibited by other afferent stimuli as described by the (Gate control theory of pain) proposed by **Melzack (1965) and wall (1978)**, or (2) modulated by descending inhibitory stimuli **(Oliveras, et al., 1974)**.