Study Of The Incidence Of Vasospastic Angina In Patients With Chest Pain & Normal Coronaries By Means Of Holter Monitor Recording

THESIS

Submitted for the Partial Fulfilment of

The Master Degree in Cardiology

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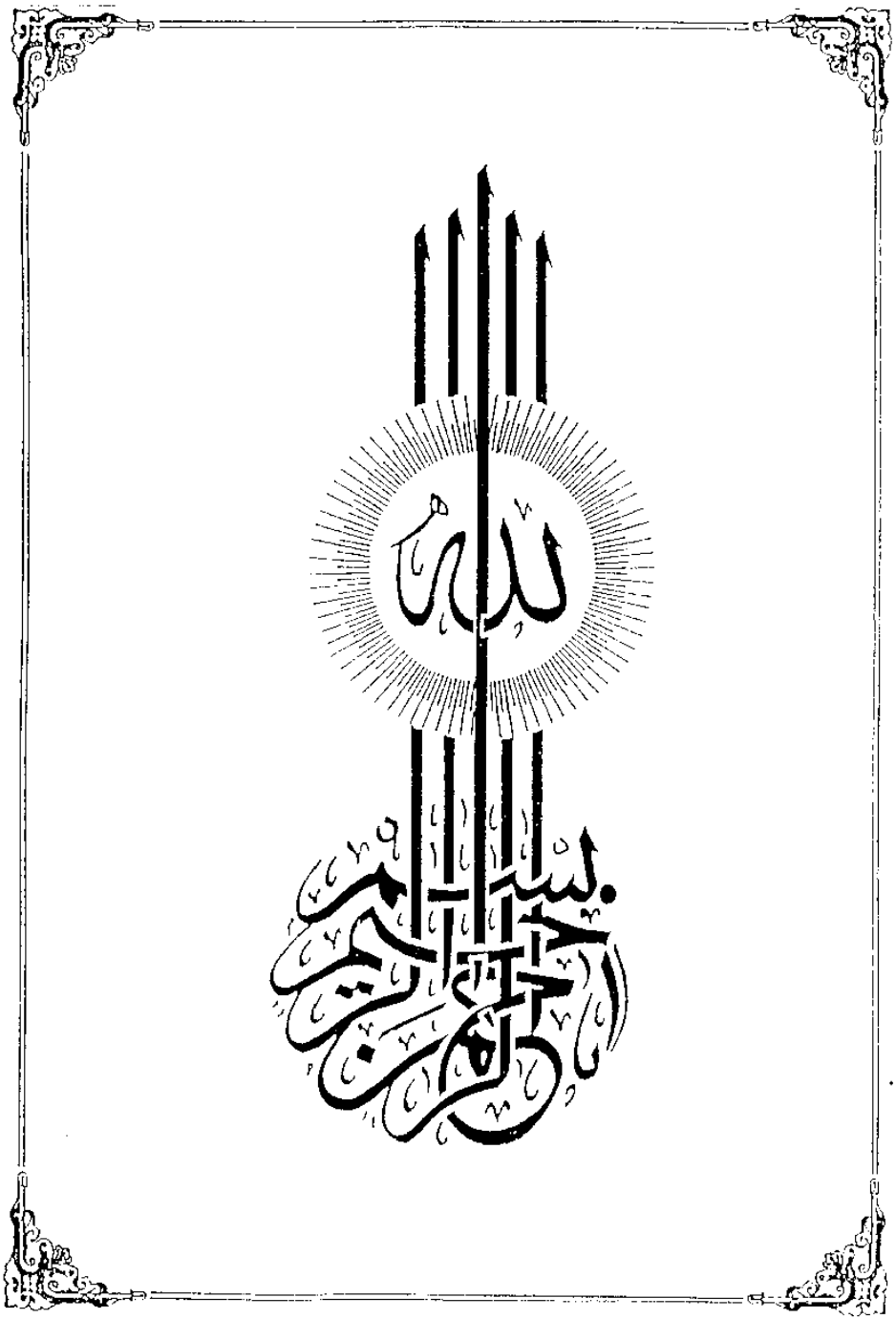
1984

بسم الله الرحمن الرحيم

«وقل رب زدني علما»

صدق الله العظيم



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ACKNOWLEDGEMENT

I would like to express my profound gratitude and sincere appreciation to professor Dr. HAMDI DEMERDASH, Chairman and Head of Cardiology Department, Ain Shams University for his advise and kindness.

I gratefully acknowledge my deep thanks and gratitude to my supervisors professor Dr. MAHMOUD EL SHERBINI Professor of Cardiology and to Dr. OMAR AWWAD lecturer of Cardiology; Ain Shams University for their kind advice, help and guidance in bringing this work forward. I would like also to express particular thanks to Dr. ADEL EL ATREBY & Dr. AHMED NASSAR ass.lecturers for their kind help, a similar note of thanks goes to every member in the Department for their help and cooperation wishing them the best of Luck.

INTRODUCTION AND AIM OF THE WORK

INTRODUCTION

In 1959 & 1960 Prinzmetal et al delineated, a variant form of angina pectoris that primarily differs from Heberden's classic angina pectoris in that the pain is frequently spontaneous, occur at rest and it is unrelated to physical activity or emotion (Braunwald 1980). The transient reversible episodes of angina pectoris at rest are characterized by ST & T wave alterations which disappear with subsidence of the pain and are not followed by permenant electrocardiographic changes or serum enzyme elevation (Maseri et al 1978).

The role of coronary arterial spasm in the production of angina pectoris has long been of interest the progress in our understanding of the phenomenon of coronary sparm seems to follow the typical evolution of the philosophical process:-

(1) Thesis (2) Antithesis (3) Synthesis.(Chahine & Luchi,1976).

The "Thesis" was developed in the 19th century & prevailed until the earlier part of the 20th century. It can be summarised by a quotation from Sir William Osler "Spasm of a coronary artery or even of one branch, may so modify the action of a section of the heart

that it works with disturbed tension, & there are stretching & strain sufficient to arouse painful sensations. I do not know of any better explanation of anginal pain (Osler 1910).

Prinzmetal's et al 1959 hypothesized. that a temporary & often cyclic increase in tonus in a narrowed vessel might be responsible for episodes of ischemic cardiac pain & sT segment elevation.

The "antithesis" followed shortly after, in the early 1950's the recognition of the association of angina with atheromatous narrowing of coronary arteries & later the failure to find coronary spasm with any significant frequency in the extensive number of coronary angiograms performed during the last 2 decades, focused attention on organic obstruction of the coronary arteries as the basic pathophysiologic mechanism underlying angina pectoris the spasm theory was dead. There was doubt that spasm may play any role in ischemic heart diseesc.(chahine,Luchi 1976).

Today we seem to be living in the era of "Synthesis". The demonstration of coronary arterial spasm during chest pain in Prinzmetal's variant angina(Dhurander,et al 1972) & in cases of withdrawal from long-term industrial

exposure to nitroglycerin (Lange et al 1972) resuscitated the interest in spasm as a possible etiologic factor in at least a subgroug of patients with ischemic heart disease,(chahine,& Luchi 1976).

During recent years the increasing interest in coronary spasm found that this phenomenon occurs in a variety of clinical situations "variant-variant angina" (Fuller et al 1980).

Vasospastic acute myocardial ischemia most likely represents a continuum that:

- Produce S-T segment elevation as well as ST segment depression. ST segment elevation represents the most striking and dramatic electrocardiographic-clinical subset,rather than the only cardiac manifestation of vasospastic angina(Boden et al 1981).

- May occur in normal coronaries & in the presence of extremely variable degrees of coronary atherosclerosis & in any phase of ischemic heart disease(Boden et al 1981).

- May evolve into acute myocardial infarction & sudden death (Waters et al 1982).

The incidence of variant angina depends upon the diligence with which it is looked for, An incidence of 13 percent was found by Maseri et al when they began to record the 12-lead electrocardiogram during all the episodes of chest pain at rest in all the patients.

This compares to a lesser incidence of 2 percent found prior to the institution of this policy.

Variant angina is unlikely to be searched for, for several reasons, At one extreme is the preconceived idea accepted by the vast majority of physicians, that angina is typically brought about by exertion or emotion & relieved by rest. Thus, when confranted with a history of episodic anginal chest pain at rest with a normal electrocardiogram at rest & on effort, the physician may dismiss the patient rather than refer him to a cardiac department. At the other extreme some patients who can sometimes perform heavy and prolonged exercise without symptoms can on other occasions experience angina on moderate effort or at rest, or both, if they are found to have some atherosclerotic narrowing in their coronary arteries, they are often judged to have only "Classic" angina. (Maseri et al 1978).

AIM OF THE WORK

The purpose of this study is two fold:

1) Study of the diagnosis of variant angina by means of Holter recording.

2) Study of the incidence of vasospastic angina in patients presented by chest pain and normal coronary angiogram.

REVIEW OF LITERATURE

Pathophysiology

The recognition that coronary artery spasm plays a role in ischemic heart disease has led to multiple investigations of its possible cause.

Hypothesis to explain coronary spasm include:-

I . Disorder of neural control mechanism of coronary circulation.

II . Disorder of metabolic control of coronary circulation.(Robertson et al 1983) .

III. Local abnormality of vascular smooth muscle(Ricci et al 1979) .

I) Neural disorder of coronary circulation:- Abnormality of autonomic nervous system as a whole.

The mechanism whereby spontaneous coronary arterial spasm is precipitated in variant angina is far from clear,Several studies have suggested the possiblity of cyclical increase in autonomic neural stimulation of epicardial coronary arteries (Higgins et al 1976) .

However the investigation by "Robertson et al 1979 " of autonomic function did not support the concept of a generalised increase in sympathetic outflow in variant angina. They found that urinary

excretion of catecholamine & ther metabolites was within the normal range. Baseline & stimulated plasma levels of norepinephrine & dopamine were normal. The results of physiologic maneuvers affecting cardiovascular function by way of the autonomic nervous system were normal. So a generalized abnormality of autonomic activity is an unlikely cause of vasotonic angina. Congurent with this is the observation that episodes of angina is not associated with changes in blood pressure or heart rates(Robertson et al 1979) .Also states as physical & emotional stress that result in outpouring of catecholamines rarely result in episodes of variant angina (Shubrooks 1979) .

Abnormality of alpha adrenergic receptors.

Enhanced alpha-adrenergic tone, has been proposed as an important etiologic factor for vasospastic angina."Ricci et al 1979" propose that the ability of phenoxybenzamine to abolish spasm can be taken as an evidence that a pathologic alteration in the alpha adrenergic system is responsible for the spasm.Alpha adrenergic mediation of coronary spasm is not a new idea. "Yasue et al 1974".

proposed that stimulation of alpha receptors mediated the spasm provoked by subcutaneous injection of epinephrine after beta adrenergic blocade.

(Endo et al 1976) showed that methacholine injection could precipitate coronary spasm in patients with variant angina, Methacholine stimulated the preganglionic cholinergic sympathetic fibers to excite alpha adrenergic receptors acting upon the coronary arteries.

Abnormality at hypothalamic level.

Other proposed mechanism of coronary spasm in patients with variant angina suggests a functional abnormality in the autonomic nervous system possibly at a hypothalamic level, results in a state of adrenergic imbalance (Ricci et al 1979) . Experiments in animals have shown that unbalancing the sympathatic nerve supply to the myocardium by ablation or stimulation of one stellate ganglion causes the T-waves in the electrocardiogram to move markedly anteriorly or posteriorly depending on which stellate ganglion was made dominant.Stimulation of the right ganglion produces negative T-waves,while reversing the side