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CORRELATION BETWEEN LEFT VENTRICULAR HYPERTROPHY AND CAROTID ATHEROSCLEROSIS

Thesis

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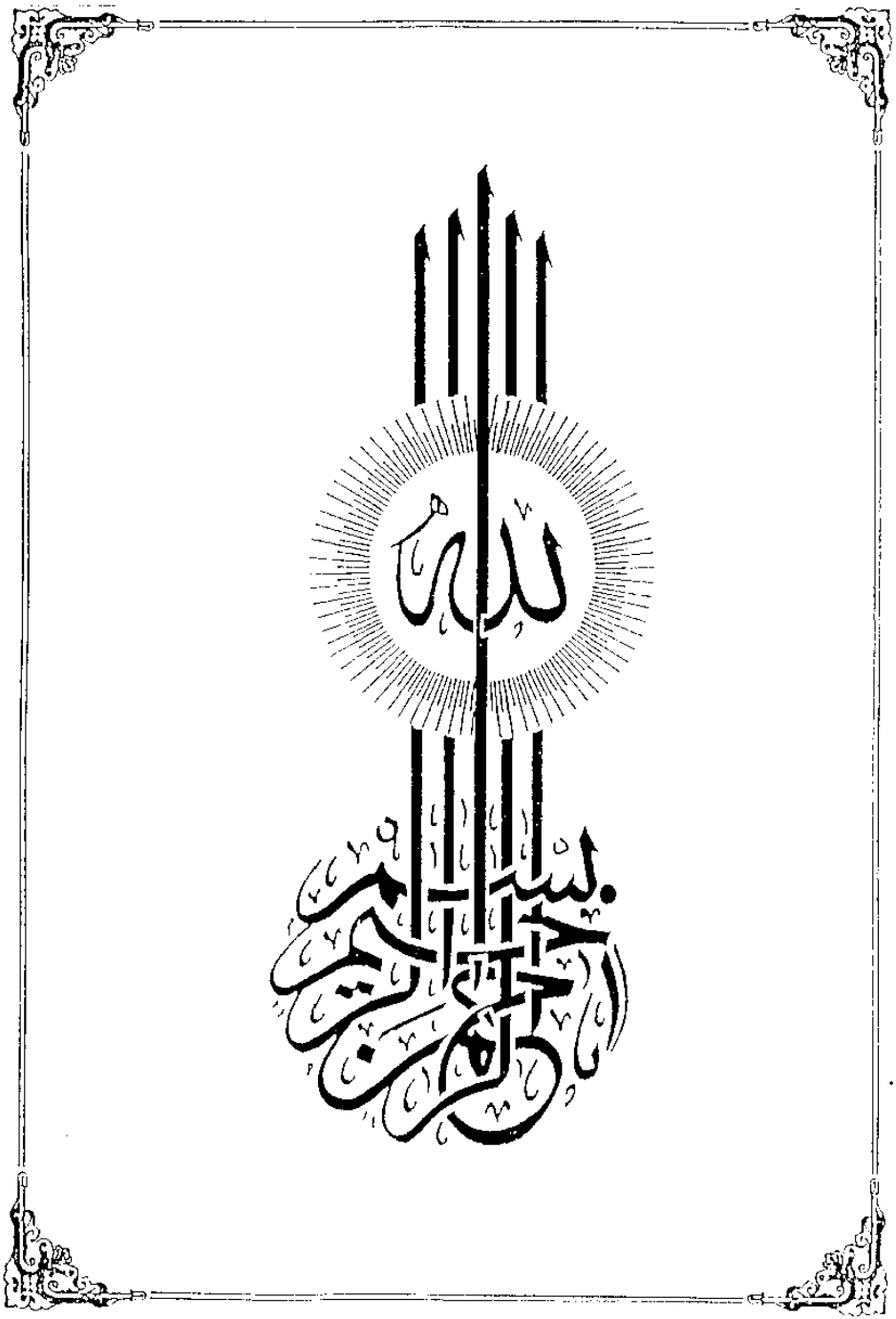

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INTRODUCTION AND AIM OF THE WORK

INTRODUCTION

The predictive value of both electrocardiographic and, as a more sensitive measure, echocardiographic evidence of left ventricular hypertrophy for subsequent cardiovascular morbidity and mortality is well established in the general population (*Levy et al., 1990*).

Progress has been made in elucidating mechanisms underlying the relation between left ventricular hypertrophy and complications from ischemic heart disease, including impairment of conorary vascular reserve, increased risk of ventricular arrhythmias and greater susceptibility of hypertrophied myocardium to ischemic damage (*Ghali et al., 1991*).

Additional observations have documented a relation between electrocardiographic or echocardiographic left ventricular hypertrophy and the subsequent risk of stroke (*Kannel, 1992*).

Recent longitudinal data from the Framingham study demonstrates a stepwise increase in the risk of transient ischemic attack and stroke by quartile of left ventricular mass indexed by height independent of blood pressure, serum lipids and smoking history in both men and women (*Bikkina et al., 1994*).

Although the mechanism of this association is unknown, indirect ECG evidence of left ventricular hypertrophy was found to be associated with increased carotid wall thickness and more prevalent carotid stenosis among elderly subjects participating in the Cardiovascular Health Study (*O' Leary et al., 1992*).

Available studies have not evaluated the possible association between the more sensitive echocardiographic measurement of left ventricular mass and the presence of atherosclerosis in the carotid circulation, a topic which is now amenable to non-invasive studies.

AIM OF THE WORK

The aim of this study is to determine the prevalence of carotid atherosclerosis in asymptomatic hypertensive compared to normotensive adults and to examine its relation to left ventricular hypertrophy.

REVIEW OF LITERATURE

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PATHOLOGY AND PATHOGENESIS OF LEFT VENTRICULAR HYPERTROPHY

Pathology of Left Ventricular Hypertrophy :

In compensated pressure overload hypertrophy, the left ventricular cavity elongates and becomes narrower. And, as the mitral ring does not enlarge, the increase in wall thickness leads to myocardial enchroachment upon the cavity. The cells may be qualitatively normal except for their diameter that can increase from a normal value of 5 - 7 μm^2 to 20 - 30 μm^2. Cell length increases by addition of sarcomeres, initiated by the proliferation of intercalated discs. There is an increased number of myofibrils which eventually leads to a significant elevation of the ratio of myofibrils to mitochondria.

Degenerative changes are frequently present in chronic left ventricular hypertrophy especially when the pressure overload is severe or prolonged. There are foci of interstitial fibrosis particulary in proximity to atrophic cells and the surrounding blood vessels and areas of myocardium may be replaced by fibrous tissue (*Morre et al., 1980*).

Pathogenesis of Left Ventricular Hypertrophy :

Adaptive or compensatory cardiac hypertrophy can be defined as the increase in heart size occuring in response to a physiological or pathological increase in the work load of the heart. This compensatory growth may be either generalized or may involve one chamber only, depending on the cause.

Two possible Mechanisms have been proposed :

First, it has been suggested that an increase in the wall stress of the heart through the increased stretch of muscle fibres could activate the genetic apparatus and lead to enhanced protein synthesis.

Second, it has been suggested that relative hypoxia in the myocardial fibre, exposed to increased tension, would cause a feedback activation of protein synthesis through the accumulation of metabolites. Against this hypothesis is another hypothesis claiming that regional ischemia did not cause changes in lactate dehydrogenase isoenzyme pattern, seen in cardiac hypertrophy (*Ostman - Smith, 1981*).