# AN ECHO-DOPPLER EVALUATION OF MYOCARDIAL HYPERTROPHY AND LEFT VENTRICULAR DIASTOLIC FUNCTIONS IN HYPERTENSIVE PATIENTS

## THESIS

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## PRSENTED BY

AMR ALY GAMAL EL-DIN
M.B.,B.CH.
FACULTY OF MEDICINE
AIN-SHAMS UNIVERSITY



## SUPERVISORS

PROF.DR. MOHAMED SAMY EL BEBLAWY
PROFESSOR OF RADIODIAGNOSIS
FACULTY OF MEDICINE
AIN-SHAMS UNIVERSITY

DR.WAHID H. TANTAWY
LECTURER OF RADIODIAGNOSIS
FACULTY OF MEDICINE
AIN-SHAMS UNIVERSITY

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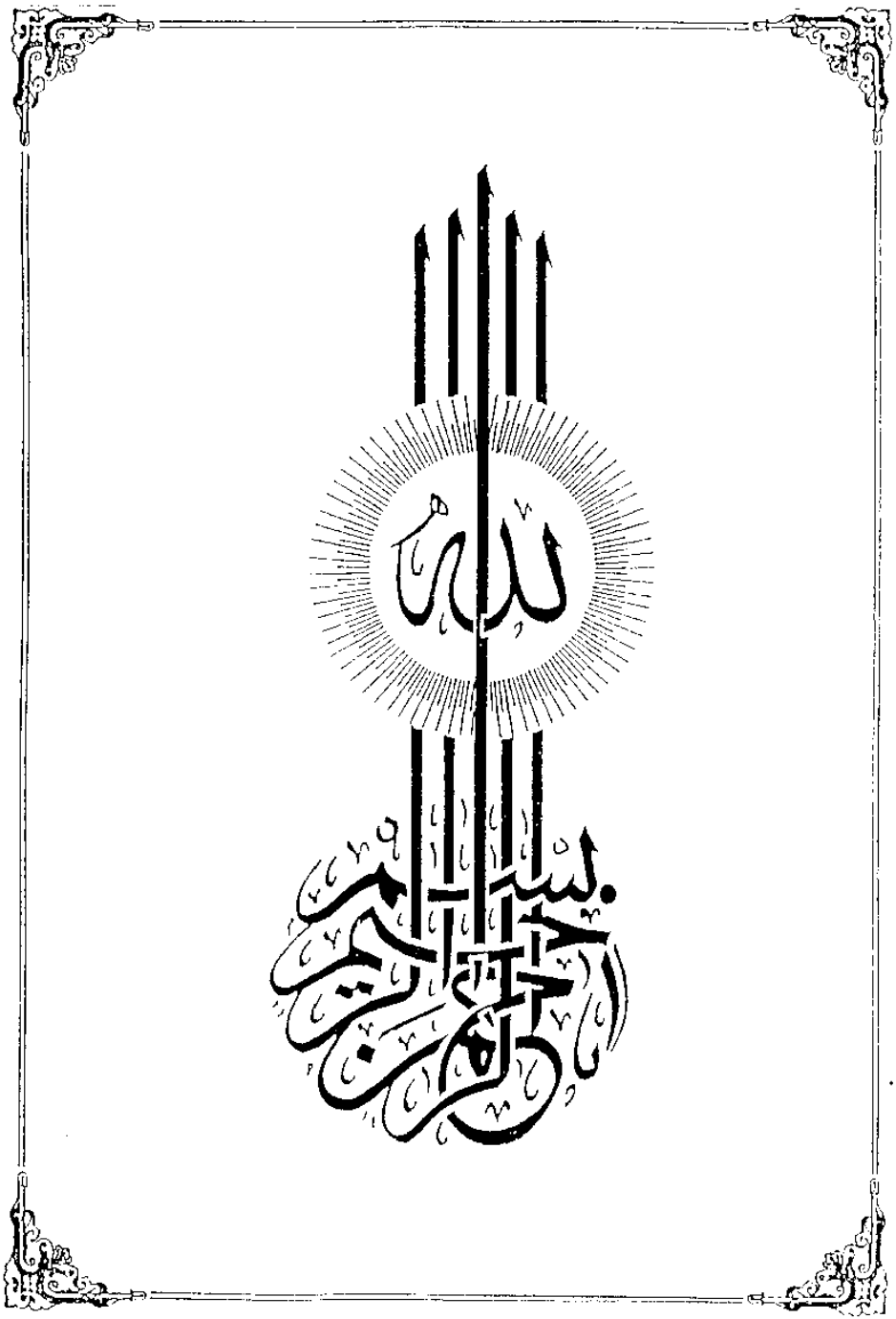
*TO MY MOTHER*



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# INTRODUCTION AND AIM OF WORK

# CHAPTER I

## INTRODUCTION AND AIM OF WORK

A major advance in diagnostic applications of ultrasound in heart disease is the development and refinement of Doppler echocardiography. Although the Doppler principle has been used to measure arterial flow, its use to assess blood flow within cardiac chambers and the great vessels is relatively recent.

M-mode and two dimensional echocardiography are useful method for visualizing intracardiac structures, quantitating chamber dimensions, wall thickness and assessing segmental wall motion. For direct evaluation of intracardiac hemodynamics, cardiac catheterization and angiography have been required. These invasive procedures are neither practical for general screening nor well suited for serial studies to assess therapeutic interventions.

The introduction of the Doppler technique into clinical practice add a new dimension in imaging by providing direct hemodynamic data that are often complementary to those demonstrated by M-mode and two dimensional echocardiography.

Rokey et al. (1985) used the left ventricular diastolic flow velocity waveform obtained with Doppler recording, in combination with a 2-dimensional echocardiographic assessment of mitral valve area and left ventricular volume, to estimate instantaneous diastolic flow.

Indices of left ventricular filling rate calculated with this method showed a good correlation with those obtained with contrast angiography ( Rokey et al., 1985 ) and radionuclide angiography ( Spirito and Maron, 1988 ). These findings convincingly demonstrate that Doppler diastolic flow velocity waveform may provide measurement of left ventricular diastolic function. This technique have allowed evaluation of the diastolic function of the heart in asymptomatic patients.

A slow left ventricular filling rate was found in hypertensive patients ( Guazzi, 1979 ) before alteration in systolic function. However, these early studies of left ventricular filling in hypertension did not report on the simultaneous structural changes in the left ventricle, particularly the presence or absence of left ventricular hypertrophy.

It is uncertain whether these changes are inherent consequence of the hypertrophic process or represent additional pathologic factors. To investigate this issue, echocardiographic indices of left ventricular diastolic function have been performed for hypertensive patients and compared with those in age matched normal control subjects.

# PATHOPHYSIOLOGY OF SYSTEMIC HYPERTENSION

# CHAPTER II

# PATHOPHYSIOLOGY OF SYSTMIC HYPERTENSION

## 2.1. Basic Hemodynamic Principles.

Under ordinary circumstances, pressure in the arterial system is maintained with fairly narrow limits in normotensive individuals. The exceptions to this generalization occur during sleep and excercise. During sleep arterial pressure may fall as low as 60/40 mmHg, and during excercise there are marked rises, particularly in systolic pressure. To understand the hemodynamics of hypertension, it may be helpful to review briefly some basic principles of hemodynamics as they relate to the flow of blood throughout the vascular system.

The flow of liquid in a tube is related to the gradient of pressure along the tube and the resistance the liquid meets as it flows. Resistance R cannot be measured directly, so it is expressed as the ratio of the pressure gradient $\Delta P$ to the flow rate F . This ratio can be translated into clinical terms in the following equation:

$$\text{Total Peripheral Resistance} = \frac{\text{Mean Arterial Pressure}}{\text{Cardiac Output.}}$$

The mean arterial pressure can be obtained by damping pulsatile pressure or by calculating it as diastolic pressure plus one-third pulse pressure. The total peripheral resistance is the sum of the resistances in all the vascular beds of the body.

2.1.1. Systems controlling areterial pressure.

Arterial pressure is determined by cardiac output and total peripheral resistance. These can be called direct determinants, and there are two others, aortic impedance and diastolic arterial volume. Impedance is a term used to describe resistence to phasic flow ( McDonald, 1974 ). For the aorta this is particularly apparant because it receives the systolic ejection volume from the left ventricle.

Each time the heart contracts, it ejects a relatively small volume of blood into the arterial system, which has limited distensibility and is already partly filled. The result is a pressure pulse; its height is determined not only by the volume ejected and the residual diastolic volume but also by the elasticity of the aorta. A normally elastic aorta dampens the pressure by dilating, then it recoils during diastole, thus creating diastolic flow. When the elasticity decreases, as with aging the arterial wall does not dilate during ejection and the full force of ejection pressure is expressed as an elevated systolic pressure. Thus, the condition of the aortic wall is a determinant of arterial pressure.

Diastolic arterial volume is a theoretical function because arterial volume cannot now be measured, nor can the residual volume at the end of diastole. But since flow out the aorta and large vessels is governed in large measure by the arterioles and precapillary sphincters, it seems logical to consider diastolic arterial volume as a determinant of

pressure.

These direct determinants of arterial pressure are influenced by a variety of systems which can be called indirect determinants, these are the activity of the central and peripheral autonomic nervous systems, total body sodium stores and / or the extracellular fluid volume, renal pressor system, and salt active steroids. Although prostaglandins and the Kallikrein-Kinin system may influence arterial pressure, yet a clear picture of their roles has not emerged.

These indirect determinants affect cardiac output, vascular resistance, circulating blood volume, and probably aortic impedance. They are also intimately inter-related. Interrelationships and interdependencies are the hallmarks of these systems that basically control arterial pressure.

It is these direct and indirect deteminants of arterial pressure that are important to any consideration of the pathophysiology of hypertension.

2.1.2. Systemic hemodynamics.

The formula ( Mean Arterial Pressure = Cardiac Output X Total Peripheral Resistance ) indicates the primacy of flow and resistance in the control of arterial pressure. In the presence of an increased cardiac output, pressure is controlled within the normal range only if vascular resistance falls. The hyperdynamic heart syndrome described by Gorlin (1962) dramatizes this because the patients studied were normotensive. In contrast, most of those reported by Forhlich

et al. (1966) were hypertensive because vascular resistance was either inappropriately normal or slightly elevated.

There are two ways that cardiac output can rise (Dustan et al., 1976). One is an increase in myocardial contractility with a rise in ejection fraction and is common in borderline and mild hypertension. The other is an elevated central blood volume associated with either hypervolemia or decreased capacity of venous reservoirs. The former occurs with obesity, and the latter leads to a central translocation of blood as reflected by an increased ratio of cardiopulmonary volume to the total blood volume.

Peripheral resistance is controlled by neural, local and humoral factors. All blood vessels are richly supplied by adrenergic nerves that provide for vasoconstriction. In mild hypertension there is evidence for increased neural tone, although in severe hypertension this does not seem to be the case. Currently, there is much interest in the possibility that an abnormality of the vascular smooth-muscle contractile mechanism is the basic fault in hypertension (Report of Hypertension Task Force, 1979). Work in progress is focussed on the control of intracellular sodium, potassium and calcium because their concentrations determine membrane potential and vascular smooth muscle contractility. Local hormones, such as prostaglandins and kinins, may also play a role in peripheral resistance, but there is no clear evidence as yet that they are important in hypertension.

2.1.3. Neural mechanisms.

The autonomic nervous system, particularly its sympathetic component, plays an important role in circulatory control. The nervous system works to control arterial pressure in two ways. One is through neural reflexes, and the other is through an ongoing maintenance of sympathetic vasomotor tone (Shepherd and Vanhoutte, 1979). The Valsalva maneuver allows an excellent view of reflex mechanism. The positive intrathoracic pressure diminishes venuos return so that cardiac output and arterial pressure fall. The reflex increase in sympathetic outflow increases heart rate and constricts arterioles. When straining is suddenly stopped, blood rushes into the thorax and the rapidly increasing cardiac output is delivered into a vasoconstricted arterial bed, so pressure rises abruptly. When this happens the mechanoreceptors are stimulated and the resultant central sympathetic inhibition slows heart rate and reduce pressure. However, why the mechanism fails in hypertension is not known, but there is evidence for upward resetting of mechanoreceptor sensitivity so that pressure rises are inadequately sensed although pressure decreases are not.

Ongoing control of sympathetic vasomotor tone is not so easy to demonstrate as reflex mechanisms are. However, there is evidence that sympathetic activity may be elevated in borderline hypertension.

Sympathetic control of the circulation, both reflex and tonic is carried out through the noradrenergic neuroeffector