NON INVASIVE INVESTIGATIONS AND CINE ANGIOGRAPHIC FEATURES OF THE CORONARY ARTERIES IN ISOLATED RIGHT VENTRICULAR HYPERTROPHY WITH CHEST PAIN

THESIS

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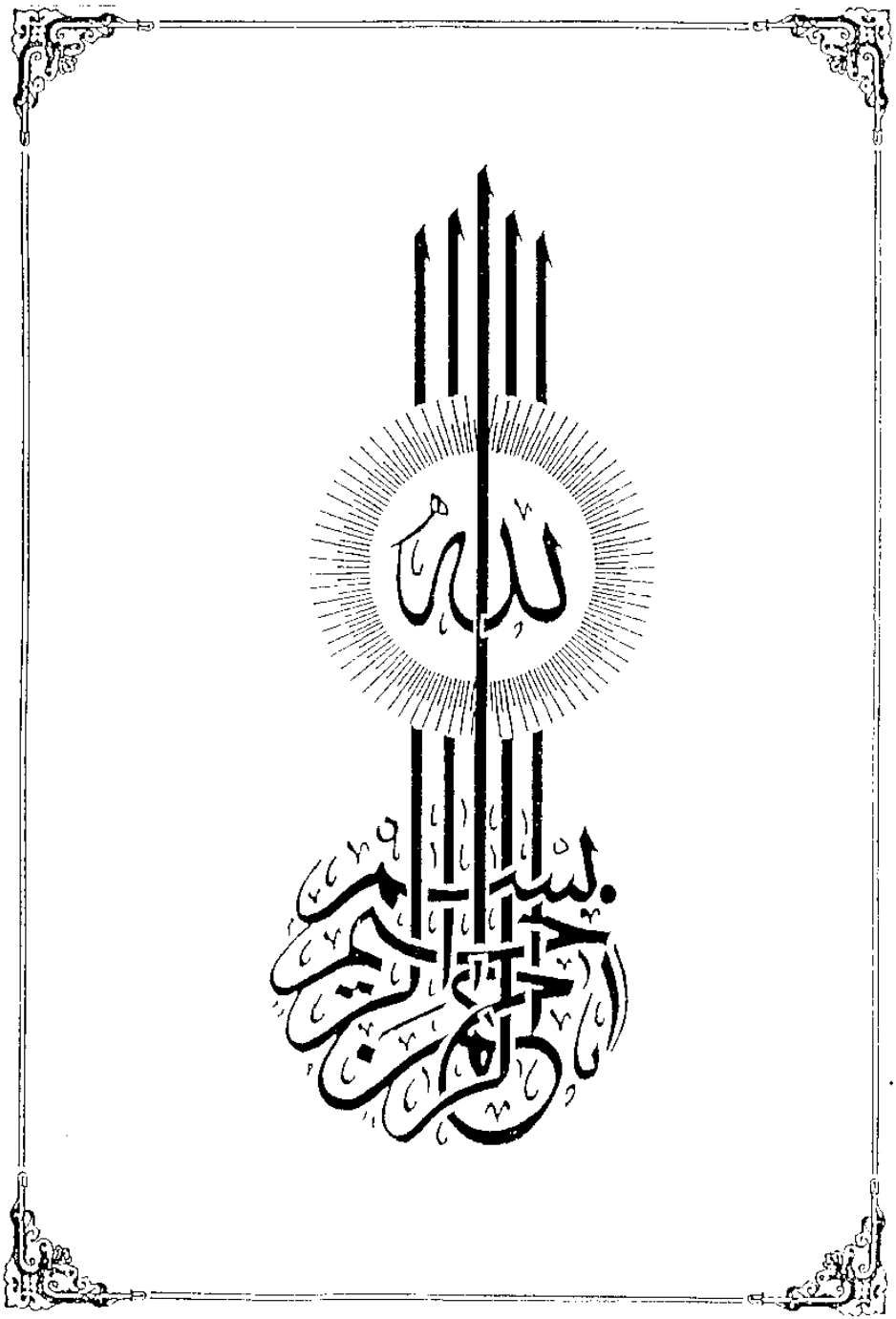
To my Father who taught me hard work

To my Wife who stands behind me

To my Daughter the smile of my life



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I N D E X

ABBREVIATIONS

A br	:	Atrial branch.
A cx	:	Atrial Circumflex
A M	:	Acute Marginal
A T P	:	Adenosine Triphosphate
Cx	:	Circumflex
E.T.	:	Ejection Time
E j	:	Ejection
I.C.S.	:	Intercostal space
I.F.S.	:	Intensity of function of structures
I.V.S.	:	Inter ventricular septum
L A D	:	Left anterior descending
L.A.I.D.	:	Left atrial internal dimension
L.A.O.	:	Left Anterior oblique
L C A	:	Left Coronary artery
L M	:	Left Main
L.S.E	:	Left sternal edge
L.s.V	:	Left sinus of valsalva
L V br	:	Left ventricular branch.
L V E D P	:	Left ventricular en-diastolic pressure.
L V I D	:	Left ventricular internal dimension.
L V P W	:	Left ventricular posterior wall
M C L	:	Mid clavicular Line.

O M : Obtuse marginal.

P : Pulmonary.

P D : Posterior descending .

PL br : Postero Lateral branch

R A D : Right axis deviation.

R A O : Right anterior oblique.

R C A : Right coronary artery.

R S V : Right sinus of vatsalva.

R V : Right ventricle.

R V E D P : Right ventricular end-diastolic pressure.

R V H : Right ventricular hypertrophy.

R V I D : Right ventricular internal dimension.

R V S P : Right ventricular systolic pressure.

R V W : Right ventricular wall.

S_3 : Third Heart Sound.

S_4 : Fourth Heart Sound.

S N B : Sinus Node Branch.

T : Tricuspid.

V.C.F : velocity of circumferential fibre shortening.

RV inf. : Right ventricular infundibulum.

INTRODUCTION AND AIM OF WORK

Chest pain simulating angina pectoris had been encountered in some of the individuals suffering from right ventricular hypertrophy. This pain may occur with lesions such as mitral stenosis, Eisenmenger's syndrome, Primary pulmonary hypertension, cor pulmonale and pulmonary stenosis. This type of chest pain is believed to be due to inadequate myocardial perfusion as a result of decreased stroke volume, reduced coronary flow, especially during systole, and to an increase in right ventricular oxygen demand (1). Therefore this chest pain is most probably secondary to myocardial ischaemia. It is not clear whether the ischaemia is confined to the right or left ventricle, but logic dictates that the right ventricle may be more affected in most instances.

The Association of angina pectoris with pulmonary hypertension was first described by Vaquez and Giroux (190 8) (2), Who coined the phrase "angina pulmonaire hypercyanotique" to describe a clinical syndrome associated with pulmonary vascular abnormalities and lung disease. Brenner (19 3 5)(3), described a boy with anginal pain, in whom necropsy revea led primary pulmonary atherosclerosis and gross hypertorophy of the right ventricle. He suggested that pain was due to myocardial anoxia associated with arterial hypoxaemia. Brill and Krygier (19 41)(4), found that chest pain of anginal type had been reported in cases of pulmonary hypertension and suggested that it might be due to coronary insufficiency associated with diminished systemic blood flow through an obstructed pulmonary circulation. Viar and Harrison (19 5 2)(5), Reported that chest pain in pulmonary hypertension may resemble angina pectoris not

only as regards location, radiation, intensity and quality of discomfort, but also as regards the tendency to be initiated by physical exertion. In some cases the pain may be associated with manifest electrocardiographic changes.

Livene and Kauvar (19 4 2) [6] reported that significant disease of the coronary arteries was found post-mortem in 5 percent of 314 cases of mitral stenosis. They concluded that in the majority of such patients mitral stenosis and coronary artery disease merely co- exist.

Isolated cases have been reported of angina pectoris occurring in patients with atrial septal defect (Bedford etal. (1941)[7], the tetralogy of fallot (Blackford, 193o and Dow etal. 195o)[8],[9] pulmonary stenosis (lowance etal. 1948[10], wood, 195c[11], Abraham's and wood 1952)[12], and Eisemenger's complex or patent ductus arteriosus[5]. Coronary arteriography has been recommended as the only way to exclude coronary artery disease[1]. In the absence of factors which alter compliance such as incomplete ventricular relaxation, stress relaxation or hypertrophy the ventricular end-diastolic pressure is a direct function of the end- diastolic volume. Accordingly, ventricular end- diastolic pressure has been used as an index of end- diastolic volume. Relation of stroke volume cr work to end- diastolic pressure has been used extensively to quantify ventricular perfomance. In addition to the factors intrinsic to the left ventricle that alter compliance, the left ventricular pressure- volume relation ship is infleunced by filling of the right ventricle both ir

vivo and in the post mortem (Taylor etal. 1967)[13]. Furthermore, during experimental diastolic overloading of the right ventricle, the left ventricular function was reduced relative to LV end- diastolic pressure (Moulopoulos etal. 1965)[14].

Finally, during clinical overloading of the right ventricle, LV end- diastolic pressure was elevated in the absence of overt LV disease (Rao etal. 1968)[15].

Clinical studies suggested that left ventricular hypertophy may occur in patients with cor pulmonale, and several investigatiors have shown that when isolated right ventricular failure was experimentally produced in animals, biochemical and morpholcgic changes similar to those present in the right ventricle occurred in the unstressed left ventricle. These changes include:

A decreased concentration of norepinephrine,and abnormal histochemical appearance of the adrenergic nerve fibers, depressed myofibrillar adenosine triphosphatase activity and increased amounts of collagen (Kelly etal. 1971)[16].

An elevation of left ventricular end- diastolic pressure is generally considered to reflect left ventricular dysfunction, Herbert and Yellin, 1969[17] found that pulmonary stenosis was also accompanied by elevation of left ventricular end- diastolic pressure.

The cardiac output is low in most patients with symptomatic mitral stenosis. The traditional concept that the low cardiac output is the

direct result of narrowed mitral orifice cannot explain the failure of cardiac output to rise in many patients after adequate mitral commissurotomy. This fact has led many workers to postulate that some sort of left ventricular dysfunction due to rheumatic myocarditis exists in such patients (Heller and Carleton 1970)[18].

The electrocardiographic criteria of right ventricular hypertrophy were studied widely by different investigators. These changes have a correlation with right ventricular muscle mass. The ST segment and T wave changes may have a correlation with the right ventricular arterial supply a point to be discussed in detail later on.

Chest roentgenograms may confirm right ventricular hypertrophy, pulmonary arterial hypertension and pulmonary artery aneurysm especially with different projections. With increasing right ventricular enlargement, The left ventricle rotates further upwards and outwards but also backwards. The right ventricle may ultimately form the whole left cardiac border in front of the left ventricle. A large right ventricle may also dilates anteriorly and may be seen on the lateral view as increased heart contact with the sternum, but this sign must be interpreted with extreme caution. The basic radiological changes of chronic pulmonary hypertension are dilatation of the pulmonary trunk and proximal pulmonary arteries with attenuation of the peripheral vessels giving the pruning appearance. In pulmonary hypertension secondary to pulmonary venous hypertension, shunts, or embolism, the basic radiological appearances

are modified and frequently show the features characteristic of the underlying aetiology (Jefferson and Rees, 1980)[19].

The diagnostic value of echocardiography is unlimited especially with rapid progress and increasing accuracy of the procedure. The echocardiographic examination of the right ventricle has many limitations. These limitations originated from the facts that the right ventricle lies directly beneath the sternum, the chamber has an irregular shape, walls are trabeculated and its location within the chest may vary significantly depending on the position of the patient during examination. Despite these limitations echocardiagraphy can reveal useful information concerning the RVW thickness and RV dimensions with RVH and these were well correlated with postmortem examination (Baker etal. 1982)[20]. Echocardiographic features of right ventricular volume overload differs from those due to pressure overload and criteria of both were extensively discussed. Pulmonary valve and tricupid valve echograms have a useful correlation with the pulmonary artery and right ventricular pressures respectively. Left ventricular function could be assessed echocardiographically with results near that obtained angiographically.

The aim of this work is to study the cine- angiographic features of the coronary arteries in patients with conditions resulting in RVH and presenting with chest pain simulating angina pectoris as one of their major symptoms. The features of the coronary arteries will be compared with those obtained from patients with anginal pain and angiographically