ASSESSMENT OF LEFT VENTRICULAR FUNCTION IN VENTRICULAR SEPTAL DEFECT BY ECHOCARDIOGRAPHY

Thesis

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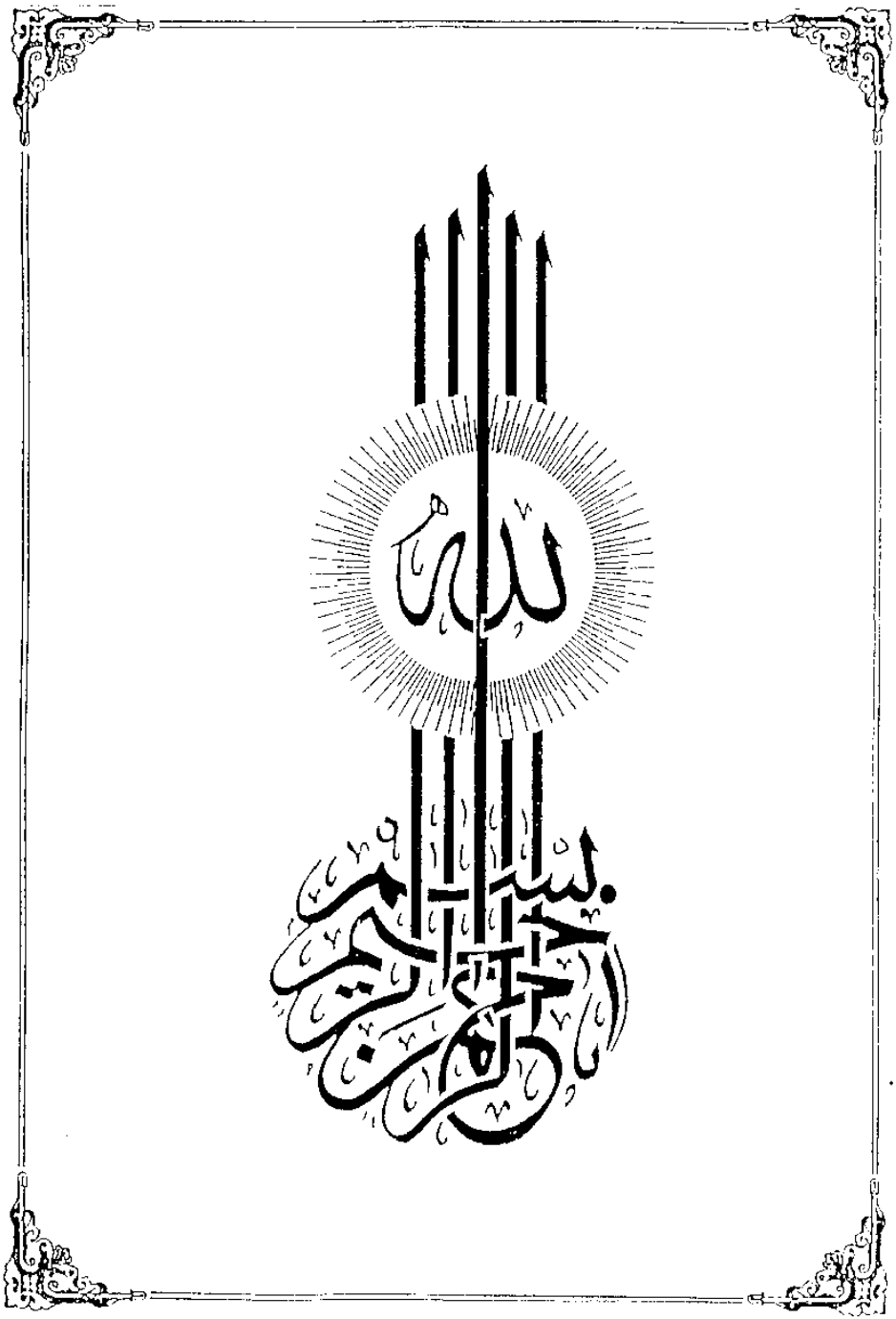

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LIST OF ABBREVIATIONS

AS	:	Aortic stenosis
ASD	:	Atrial septal defect
A-V	:	Atrio-ventricular
BAV	:	Bicuspid aortic valve
CA	:	Coarcitation of aorta
CHD	:	Congenital heart disease
CHF	:	Congestive heart failure
C.O.P.	:	Cardiac output
C/T ratio	:	Cardiothoracic ratio
Dd	:	Diastolic diameter
Ds	:	Systolic diameter
E/A	:	"E" velocity / "A" velocity
ECD	:	Endocardial cushion defect
ECG	:	Electrocardiogram
EDD	:	End diastolic diameter
EDV	:	End diastolic volume
EF%	:	Percentage ejection fraction
ESD	:	End systolic diameter
ESV	:	End systolic volume
ET	:	Ejection time
FS%	:	Percentage fraction shortening

LAD	:	Left atrial diameter
LV	:	Left ventricle
LVD	:	Left ventricular dilatation
LVH	:	Left ventricular hypertrophy
LVIV	:	Left ventricular inflow volume
LVOV	:	Left ventricular outflow volume
MR	:	Mitral regurgitation
MRI	:	Magnetic resonance imaging
MVP	:	Mitral valve prolapse
PDA	:	Patent ductus artriosus
PS	:	Pulmonary stenosis
PVOD	:	Pulmonary valve obstructive disease
QP/QS	:	Pulmonary to systemic pressure
RVD	:	Right ventricular dilatation
RVH	:	Right ventricular hypertrophy
SV	:	Stroke volume
VSD	:	Ventricular septal defect

Introduction & Aim of Work

INTRODUCTION & AIM OF THE WORK

Ventricular septal defect is a congenital defect of the interventricular septum allowing free communication between the ventricular chambers (Soto and Pacifico, 1990).

It is one of the most common congenital cardiac malformations accounting for approximately 30-40% of all children with CHD (Harrigan and Lee, 1985). It can occur either alone or in association with other cardiovascular anomalies (Canale et al., 1981).

The size of the defect and the relationship between systemic and pulmonary vascular resistance determine the degree and direction of shunting (Hamigan and Lee, 1985). Ventricular septal defect leads to increase pulmonary blood flow and this leads to increase left atrial diameter and left ventricular end diastolic volume, which will lead to increase left ventricular work and left ventricular dilatation. Very large pulmonary blood flow will result in additional right ventricular hypertrophy (Anderson et al., 1987). The assessment of left ventricular function has become progressively very important in the diagnosis and management of children with congenital and acquired heart disease since the

preservation of myocardium is now the standard by which therapy should be measured (Wilse, 1990).

The question of when to recommend operation in a patient with heart disease represent a difficult problem that is still not fully solved, early surgical treatment aims at avoiding irreversible left ventricular damage which eventually leads to postoperative cardiac failure and even death but premature operation is undesirable because of the problems of valvular prosthesis and other types of operative and post operative morbidities (Ross, 1981). Although many recent techniques for evaluating left ventricular performance have been established, echocardiography has gained great popularity as a safe, inexpensive and non-invasive technique (Kraus, 1985). Doppler signal with the two-dimensional echocardiographic instrument may give information in both a qualitative and quantitative sense (Goldberg et al., 1988).

The aim of this work is to study VSD as regard site, size and pressure gradient across it in order to find the relation of symptoms to the size of VSD and to asses the effect of VSD on left ventricular function and dimensions.

Review of Literature

Chapter I
Ventricular Septal Defect

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VENTRICULAR SEPTAL DEFECT

Congenital heart disease refers to structural or functional heart disease that is present at birth, even if it is discovered much later (Hoffman, 1990). VSD is a congenital defect of the interventricular septum allowing free communication between the ventricular chambers (Soto and Pacifico, 1990). VSD occurs either as an isolated malformation as an essential part of more complex malformations (Rao et al., 1971), or in association with unrelated cardiac anomalies of which the VSD does not form an essential part (Girod et al., 1966). A major portion of this chapter will be concerned with isolated VSD.

* *Development of the Interventricular Septum*

Formation of the heart tube takes place in the visceral mesoderm between the yolk sac and the intraembryonic coelam which will later become the pericardium.

The embryonic heart soon acquires several curvatures and becomes an S-shaped organ. This transformation process is called "looping" (Wenink, 1987). The major septa of the heart are formed between the 27th and the 37th day of development as the embryo grows in length. In the first mechanism of septation, masses of actively growing tissue fuse together to bridge the cardiac lumen.