

## Assessment of Left Atrial Volume in Cases of Ventricular Septal Defects in Childern

AThesis Submitted in partial fullfillment for the requirements of The Master Degree in pediatrics  $oldsymbol{BY}$ 

Ibrahim Abdel-Rhman Ibrahim Abo El Soud (M.B.B.CH) 2005 Al-Azhar University

# **Supervisors**

### Dr. Abdel-Aziz Mohamed Soliman

Professor of Pediatrics
Faculty of medicine AL -Azhar University

## Dr .Sherif Mostafa Reda

Professor of Pediatrics
Faculty of medicine AL -Azhar University

## Dr .Hassan Saad AbuSaif

Professor of Pediatrics. ped.cardiology unit Faculty of medicine Al-Azhar University

## Dr .Ahmed Yosef Al Sawah

Professor of Pediatrics
Faculty of medicine Al-Azhar University

## Dr .Mohamed Mostafa Ahmed Shaban

Assistant.Professor of Pediatrics ped.cardiology unit Faculty of medicine Al-Azhar University

2014

### LIST OF CONTENTS

Title	Page
List of Abbreviation	ii
List of Tables	iii
List of figures	iv-v
Introduction	
Aim of the work	
Definition and prevalence of vsd	3
Anatomy of vsd	4
Anatomy of left atrium	
Types of vsd	
Embryology in vsd	
Associated anomalies in vsd	
Pathophysiology of vsd	
Clinical manifestations of vsd	
Invesigations of vsd	
Echocardiography in vsd	30
Visulization of left atrium by echocardiography	
Estimation of left atrial volume in vsd	
Complications of vsd	68
Natural history and treatment in vsd	
Patients and methods	77
Results	
Discussion	118
Conclusion	128
Recommendations	129
Summary	130
References	
Arabic summary	• • • • • • • • • • • • • • • • • • • •

## List of Abbreviation

AR:	Aortic regurge
ΑO	Aortic root
BSA	Body surface area
BW	Body weight
CHD	Congenital heart disease
CHF	Congestive heart failure
ECG	Electrocardiography
ECHO	Echocardiography
EF	Ejection fraction
FS	Fractional shortening
FTT	Failure to thrive
ISWT	Interventricular septal wall thickness
I V C	Inferior vena cava
IVS	Interventricular septum
LA	Left atrium
LA D	Left axis deviation
LAD(cm)	left atrial dimension in cm
L/AO	Left atrial aortic root ratio
LV	Left ventricle
LVDD	Left ventricular diastolic dimension
LVH	Left ventricular hypertrophy L
LV S D	Left ventricular systolic dimension
M R	Mitral regurge
Pa	Pulmonary artery
PΑ	Pulmonary atresia
PAP	Pulmonary artery pressure
PG	Pressure gradient
P S	Pulmonary stenosis
PVR	Pulmonary vascular resistance
R A	Right atrium
R A D	Right axis deviation
R V	Right ventricle
RVH	Right ventricular hypertrophy
TAR	Thrombocytopenia absent radius
2 D ECHO	Two dimensional echocardiography
TR	Tricuspid regurge
V.S.D	Ventricular septal defect
V. S. D PG	Pressure gradient across vsd defect

# **LIST OF TABLES**

Table.No	Subject	Page	
(1)	Associated anomalies with V.S.D	13	
(2)	Continue of syndromes associated with VSD	15	
(3)	Normal m-mode echo values (mm) by weight (lb):		
	mean (range)		
(4)	Normal Doppler velocities in children and adults:	43	
	mean (range) (m/sec)		
	Tables of results		
(1)	Demographic clinical data of different groups	82	
(2)	Clinical data of different groups	84	
(3)	M mode data for the different groups	85	
(4)	Doppler echo findings in different groups	86	
(5)	2 dimension echo in different groups	86	
(6)	Left atrial measurements in different groups	88	
(7-8-9)	Correlation between BSA &LAV	90	
(10-11-12)	Correlation between BW&LAV	92	
(13-14-15)	Correlation between LAD & LAV	94	
(16-17-18)	(16-17-18) Correlation between Age & LAV		
(19-20-21)	(19-20-21) Correlation between EF & LAV		
(22-23-24)	Correlation between Fs & LAV	99	
(25-26-27)	Correlation between VSD & LAV	101	
(28-29-30)	Correlation between PG & LAV	103	
(31-32-33)	Correlation between LVSD & LAV	105	
(34-35-36)	Correlation between LVDD& LAV	107	
(37-38-39)	Correlation between LA/ AO & LAV	109	

# **LIST OF FIGURES**

Fig. No	Title	Page
(1)	Picture showing VSD	3
(2)	Picture showing location of VSD	5
(3)	Membranous septum	6
(4)	Types of VSD	11
(5)	Embyology in VSD	12
(6)	Syndromes associated with VSD	14
(7)	Illustrating figures of the three main defects of VSD	19
(8)	X-Ray showing cardiomegally in VSD	24
(9)	Normal ECG in VSD	25
(10)	RVH in ECG of VSD	25
(11)	LVH in ECG of VSD	25
(12)	Left atrial enlargement in VSD	26
(13)	M mode ,parasternal long axis show	30
(14)	Prasternal long axis show perimembranous VSD and color jet	34
(15)	through defect Parasternal short axis show subpumonary VSD	36
(16)	Apical 4 chamber show inlet VSD	38
(17)	2D coupled with Doppler echo and color flow mapping	46
(18)	Trans thoracic perimembranous VSD	47
(19)	Parasternal long axis show supracristal VSD with buckling and prolapsed right coronary leaflet of aortic valve	51
(20)	Malalignment of VSD	52
(21)	Color flow Doppler on 4 types muscular VSD	54
(22)	Doppler image show spectral recording of VSD	55
(23)	Two dimentions of left atrium	58
(24)	Lt atrial calculation measures	60

Fig. No	Title	Page
(25)	Different measurement methods of left	62
	atrium	
(26)	Measurement of left atrium from	63
	simpson method	
(27)	Measurment of lt atrium from prollate	63
	ellipse method	
(28)	Difference of means of indexed left	64
	atrial volum measured from area length	
	in relation to prolate ellpse	
(29)	Difference of means of indexed left	64
	atrial volum measured from simpson in	
	relation to prolate ellpse	
(30)	Difference of means of indexed left	65
	atrial volum measured from area length	
	in relation to bi plane modified simpson	
(31)	Difference of means of indexed left	65
	atrial volum measured from area length	
	in relation to bi plane modified simpson	
	LVEDD (left ventricular diastolic	
	dimention )	
(32)	Images of perimembranous VSD	72
	closure withAmplatzer membranous	
	device	
(33)	Amplatzer membranous device	75
(34)	Figures of results	86-118

### **INTRODUCTION**

Vetricular septal defects are the most common congenital cardiac anomalies. They are found in 30-60% of all newborns with congenital heart defect.or about 2-6 per1000 births. During heart formation when the heart begins as ahollow tube, it begins to partition forming a septa. If this does not occur properly it can lead to an opening being left within the ventricular septum. it is debatable whether all these defects are true heart defects, or if some of them are normal phenomena, since most of the trabecular VSDs close spontaneously (Roguin et al,1995).

Two-dimentional echocadiography, along with Doppler echocardiography and colour flow imaging can assess the size and location of virtually all ventricular septal defects (VSDs). Doppler echocardiography also provides physiological information including right ventricular pressure, pulmonary artery pressure and the difference in pressure between the ventricles. Measurment of left atrial and left ventricular diameter provides semi-quantitative information about shunt volume. Defect size is often given in terms of the size of the aortic root. defects that are about the size of the aortic root are classified as large, those one third to two thirds of the diameter of the aorta are moderate, and those less than one third of the aortic root diameter are small (Arora et al, 2003).

Left atrial volume has been showen to reflect diastolic function and is powerfull predector of cardiac morbidity and mortality ,normative left atrial volume values in childern with congenital heart diseases and ventricular septal defects are lacking (Taggart Nw. et al,2010).

Increase left atrial volume is an indicator of diastolic dysfunction and asurrogate marker of significant left to right shunts (**Bhtala,etal,2012**).

Increased left atrial dimension easured by M-mode echocardiography is arisk factor for atrial fibrillation, stroke, and death. (**Pritchett, et al, 2003**).

## **AIM OF THE WORK**

Evaluation of percentage of different types of ventricular septal defects in children and assessment of left atrial volume in these cases.

### Ventricular septal defect (VSD)

#### **Definition**

A ventricular septal defect (VSD) is an opening in the interventricular septum resulting in direct communication between the left and right ventricles. VSD can be single or multiple. (*Nichols*, 2008)

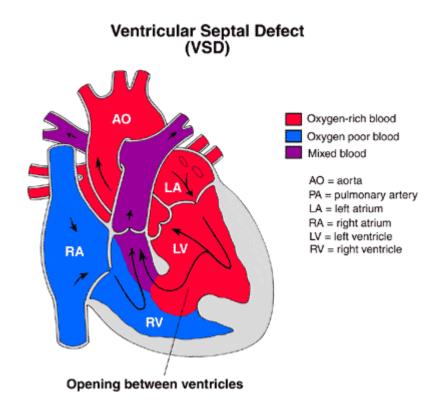


Figure (1): Showing ventricular septal defect

#### **Prevalence**

Ventricular septal defects (VSDs) are the most common form of congenital heart disease if bicuspid aortic valve is excluded. The defect can be in any portion of the ventricular septum, and the physiologic consequences can range from trivial to severe (Moss&adam,2008).

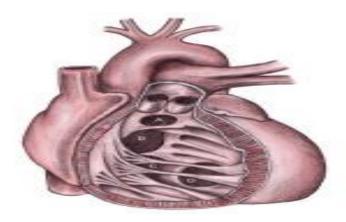
Approximately 20% of patients in congenital heart disease registries have VSD as a solitary lesion recent echocardiographic studies demonstrated an incidence of VSD in newborns to be 5 to 50 per 1,000). Outlet: 5% -7% Inlet: 5% -8%, posterior and inferior to perimembranous defect Muscular: 5%-20%. The lower prevalence in adults with congenital heart disease is due to spontaneous closure of many defects. VSDs are slightly more common in females: Approximately 56% female, 44% male. VSDs are the most common lesion in many chromosomal syndromes, including trisomy 13, trisomy 18, and trisomy 21 groups, as well as in rarer syndromes). patients with VSDs (>95%), the defects are not associated with a chromosomal abnormality. A multifactorial cause has been proposed in which interaction between hereditary predisposition and environment results in the defect) (*Moss and adam, 2008*).

#### **Anatomy of ventricular septal defect**

The ventricular septum is a complex structure that can be divided into four components. The largest component is the muscular septum. The inlet or posterior septum comprises endocardial cushion tissue. The subarterial or supracristal septum comprises conotruncal tissue. The membranous septum is below the aortic valve and is relatively small. VSDs occur when any of these components fails to develop normally perimembranous VSDs are the most common of all VSDs 67%)Although the location of the VSD is important prognostically and in approach to repair, physiologically, the amount of flow crossing a VSD depends on the size of the defect and the pulmonary vascular resistance (*Nelson ess*, 2010).

the ventricular septum is considered to have four components: 1- An inlet septum separating the mitral and tricuspid valves; 2- a trabecular septum, which extends from the attachments of the tricuspid leaflets outward to the apex and

upward to the crista supraventricularis; the smooth-walled 3-outlet or infundibular septum, which extends from the crista to the pulmonary valve; and 4- the membranous septum, which is relatively small and is usually divided into two parts by the septal leaflet of the tricuspid valve (*Moss&adam,2008*).



**Figure (2):** the location of various types of ventricular septal defects (VSDs) from the right ventricular aspect. A = Doubly committed subarterial ventricular septal defect; B = Perimembranous ventricular septal defect; C = Inlet or atrioventricular canal–type ventricular septal defect; D = Muscular ventricular septal defect.

1. The muscular septum has three components: the inlet septum, the trabecular septum, and the outlet (infundibular or conal) septum. The trabecular septum (also simply called muscular septum) is further divided into anterior, posterior, middle, and apical portions. Therefore, a VSD may be classified as a membranous, inlet, outlet (or infundibular), midtrabecular (or midmuscular), anterior trabecular (or anterior muscular), posterior trabecular (or posterior muscular), or apical muscular defect ).

2-**The Membranous septum** is a relatively small area immediately beneath the aortic valve. The membranous defect involves varying amounts of muscular tissue adjacent to the membranous septum (perimembranous VSD). According to the accompanying defect in the adjacent muscular septum, called perimembranous **VSDs** have been perimembranous inlet (atrioventricular [AV] canal type), perimembranous trabecular, perimembranous outlet (tetralogy type) defects. Perimembranous defects are most common (70%) (Park,2008).

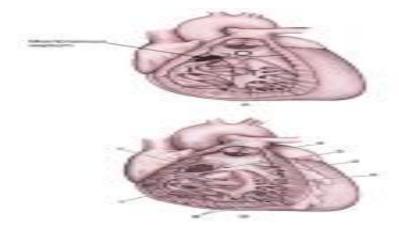


Figure (3) :show membranous septum

Membranous septum Lies between the anterior and the septal tricuspid leaflets and below the right and the noncoronary cusps of the aortic valve A: Image shows a ventricular septum viewed from the right side. It has the following 4 components: inlet septum from the tricuspid annulus to the attachments of the tricuspid valve (I); trabecular septum from inlet to apex and up to the smooth-walled outlet (T); outlet septum, which extends to the pulmonary valve (O); and membranous septum. B: Anatomic positions of the defects are as follows: outlet defect (a); papillary muscle of the conus (b);

perimembranous defect (c); marginal muscular defects (d); central muscular defects (e); inlet defect (f); and apical muscular defects (g) (**Hurset,2009**).

Perimembranous defect lies in the outflow tract of the left ventricle immediately beneath the aortic valve. Synonyms include membranous defect and infracristal defect.

When viewed from the right side of the heart, the defect is beneath the crista supraventricularis and posterior to the papillary muscle of the conus..

With the perimembranous defect, there can be a variable degree of anterior malalignment between the infundibular septum and the anterior ventricular septum such that the aortic valve appears to override the defect . Posterior or leftward malalignment also occurs, producing subaortic stenosis. . Outlet VSDs constitute approximately 5% to 7% of defects seen. Inlet defects that are posterior and inferior to the membranous defect beneath the septal leaflet of the tricuspid valve and inferior to the papillary muscle of the conus have been called atrioventricular septal defects (Moss and adam,2010 ).

<u>A)-Outlet septum</u>: 5% -7% of autopsy and surgical series (29% in the Far East), situated just beneath the pulmonary value (synonyms: supracristal, conal, infundibular, subpulmonary, doubly committed subarterial) (*Moss and adam,2008*).

In an infundibular defect, the right coronary cusp of the aortic valve may herniate through the defect. This may result in an actual reduction of the VSD shunt but may produce aortic regurgitation (AR) and cause an obstruction in the right ventricular outflow tract. A similar herniation of the right and /or noncoronary cusp occasionally occurs through perimembranous defects(*park*, 2008).

B)-Inlet septum It lies beneath the septal leaflet of the tricuspid valve) (park,2008).

The ventricular septum is curved and therefore does not lie in a single plane. Multiple views are required to examine the entire septal region, and a single imaging plane will neither interrogate the complete structure nor detect every defect, Visualization of a ventricular septal defect in more than one imaging plane is the most direct. means of diagnosis. In general, false-negative findings are more common than false-positive results. The sensitivity of two-dimensional echocardiography for diagnosis of a ventricular septal defect depends on location. Sensitivity is highest for inlet and outlet defects (approaching 100%), slightly less for perimembranous defects (80%-90%), and least for trabecular defects (as low as 50% in some earlier studies but considerably higher with modern equipment and techniques). The reasons for this low detection rate are that trabecular defects can occur anywhere within a. fairly large area (Figenbaum,2009).

#### **Anatomy** of left atrium

oval-shaped chamber with thin, muscular walls, the left atrium is easily visualized posterior to the aortic root and superior to the left ventricle. With the advent of transesophageal echocardiography, the ability to thoroughly interrogate the left atrium, including its appendage, became possible, and a thorough assessment of its structure and function is now routinely performed.

The ventricular septum may be divided into a small membranous portion and a large muscular portion (Park,2008).