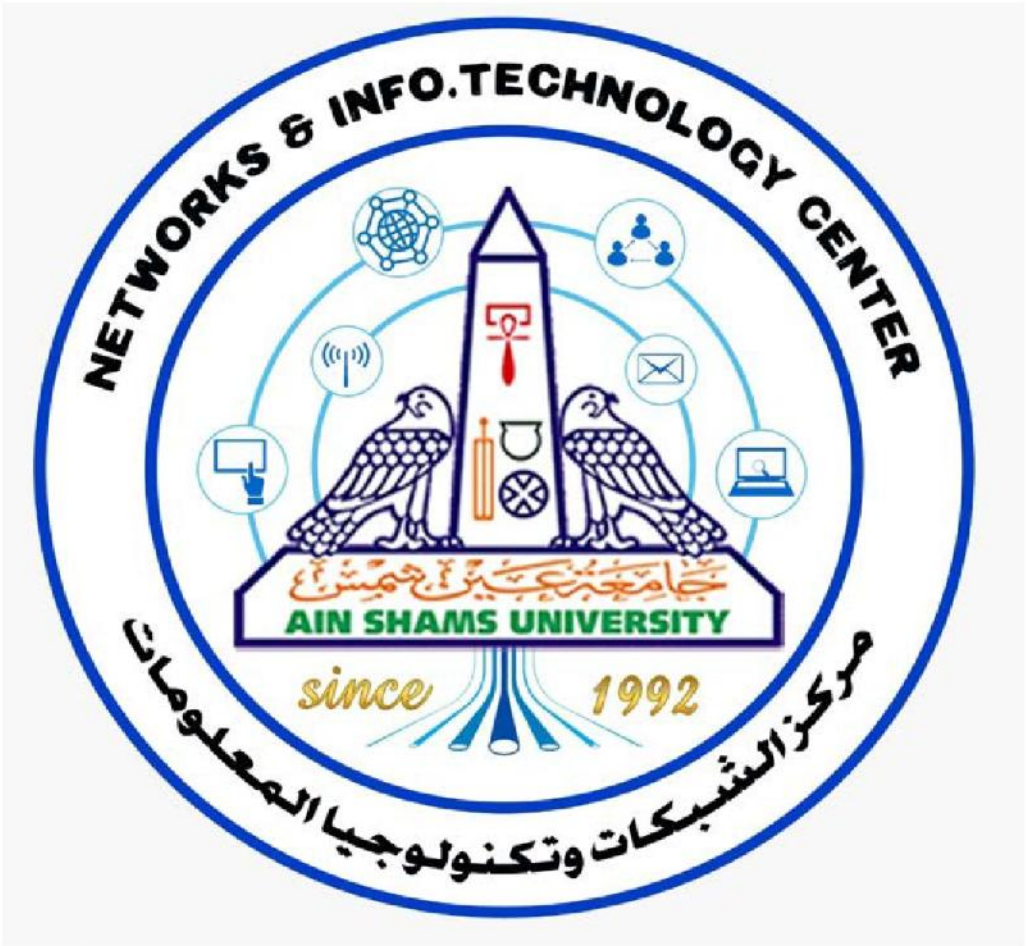
hossam maghraby

بسم الله الرحمن الرحيم

مركز الشبكات وتكنولوجيا المعلومات

قسم التوثيق الإلكتروني



hossam maghraby

جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها

علي هذه الأقراص المدمجة قد أعدت دون أية تغيرات

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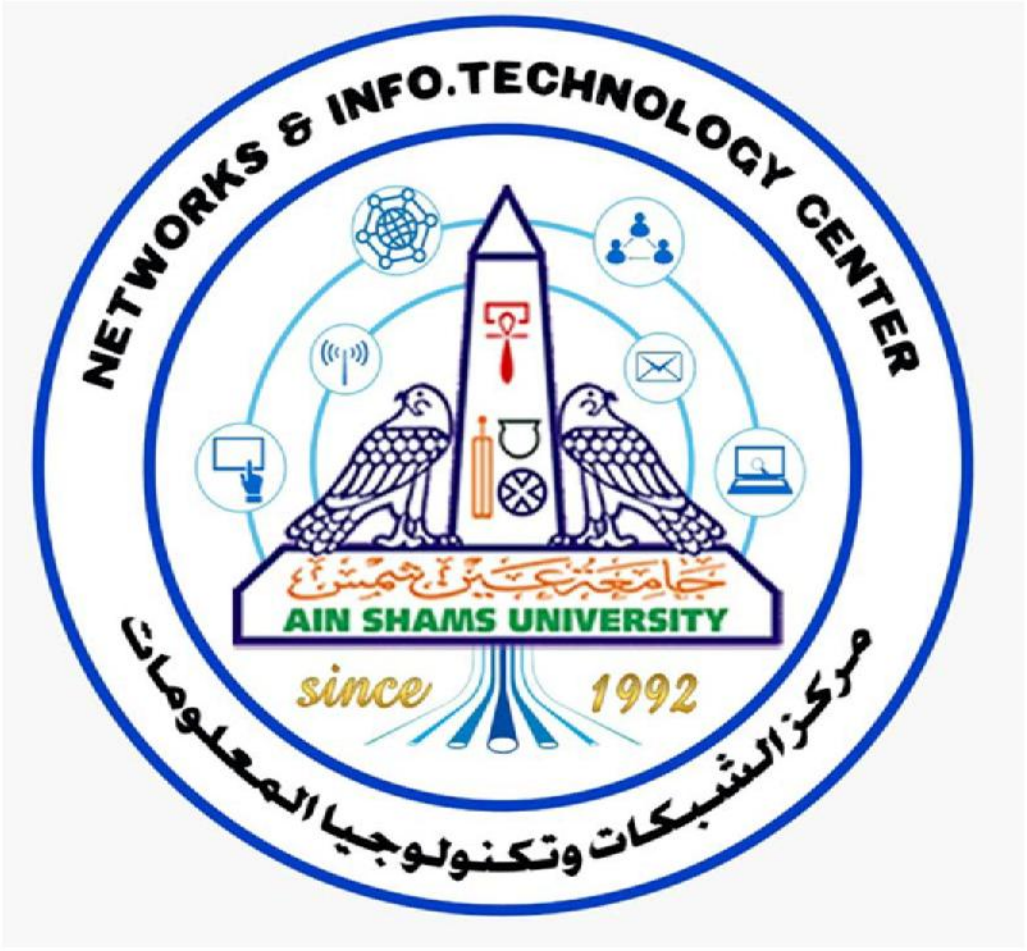

بعض الوثائق الأصلية تالفة
وبالرسالة صفحات لم ترد بالأصل

بسم الله الرحمن الرحيم

GROWTH PARAMETERS IN RELATION TO SEVERITY OF VSD IN CHILDREN WITH COMPENSATED CONGENITAL HEART DISEASE.

THESIS

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CONTENTS

ABBREVIATIONS

A-V canal	= Atrioventricular canal
AS	= Aortic stenosis
ASD	= Atrial septal defect
BMI	= Body mass index
CFI	= Colour flow imaging
CHD	= Congenital heart disease
CHF	= Congestive heart failure
EF	= Ejection fraction
FS	= Fraction shortening
GH	= Growth hormone
Gr. Vel	= Growth velocity
HtSDS	= Height standard deviation score
IGF-I	= Insulin like growth factor I
IUGR	= Intrauterine growth retardation
IVG	= Inter - ventricular gradient
MAC	= Mid arm circumference
PDA	= Patent ductus arteriosus
PVR	= Pulmonary vascular resistance
Qp	= Pulmonary stroke volume
Qp/Qs	= Shunt ratio
Qs	= Systemic stroke volume
TOF	= Tetralogy of Fallot
VSD	= Ventricular septal defect

Introduction

INTRODUCTION

Congenital heart disease (CHD) refers to structural or functional heart disease that is present at birth, even if it is discovered later.[1] The modern studies yielded an incidence of CHD in live born infants of 4.05 to 10.2 per 1000. For lesions assumed to be due to flow disturbances, recurrence risks were often high. It was 5.8% for perimembranous ventricular septal defect (VSD).[2, 3]

VSD, being the most common cardiac anomaly, constitutes about 25-30% of congenital heart lesions. The incidence is higher among still born (2%), abortuses (10-25%), and premature infants (2%, excluding transient PDA). This overall incidence does not include atrial valve prolapse, PDA of preterm and bicuspid aortic valves.[4]

Most congenital defects are well tolerated during fetal life. It is only after birth, when the Cardiovascular System is independently sustained, that the impact of an anatomic and subsequent hemodymamic abnormality becomes apparent.[4]

The infant's circulation continues to change after birth and later changes have a hemodynamic effect on cardiac lesions. For example, as pulmonary vascular resistance falls over the 1st few weeks of life, left to right shunts become more apparent. The relative significance of various defects also changes with growth; the large VSD may become a relatively small communication later.[4]

The direction of the shunt in acyanotic VSD is left-to-right. The magnitude of the shunt is determined by the size, not the location, of the defect and the level of pulmonary vascular resistance (PVR). With a small defect, a large resistance to the left-to-right shunt is offered at the defect, and the shunt does not depend on the level of pulmonary vascular resistance. With a large VSD, the resistance offered by the defect is minimal, and the left-to-right shunt depends on the level of pulmonary vascular resistance. The lower the pulmonary vascular resistance, the greater the magnitude of the left-to-right shunt. This type of left-to-right shunt is called "dependent shunt". Even in the presence of a large VSD, the decrease in pulmonary vascular resistance to a critical level does not occur until the age of 6 to 8 weeks, thus the onset of congestive heart failure is delayed until that age.[5]

In a VSD of moderate size, there is enlargement of the main pulmonary artery, left atrium, and left ventricle, as well as an increase in the pulmonary vascular markings.[6] In VSD, it is the left ventricle that sustains volume overwork, not the right ventricle. This results in left ventricular enlargement; the right ventricle does not enlarge. Since the shunt of VSD mainly occurs during systole when the right ventricle also contracts, the shunted blood goes directly to the pulmonary artery rather than remains in the right ventricular cavity. Therefore, there is no significant volume overload to the right ventricle, and the right ventricle remains relatively normal in size.[7]

To understand the hemodynamics and clinical manifestations of varying sizes, we should notice that the size of a cardiac chamber directly

relates to the amount of blood handled by the chamber.[8] With a small VSD, there is only small amount of the blood coming from the left ventricle to the main pulmonary artery. In addition, the degree of pulmonary vascular congestion and the chamber enlargement is either minimal or too small to result in a significant change of the chest x-ray films. The degree of volume work imposed on the left ventricle is also too small to produce left ventricular hypertrophy on the ECG. The shunt itself produces a heart murmur (regurgitant systolic), and the intensity of the P_2 is normal because the pulmonary artery pressure is normal. With a VSD of moderate size, a significant amount of blood shunts from the left ventricle to the right ventricle, and all the chambers that are enlarged handle more amount of blood. Therefore the cardiomegaly on the x-ray film is of a significant degree. The volume overwork done by the left ventricle is significant so that the ECG produces left ventricular hypertrophy of "volume overload" type. Although the shunt is large, the right ventricle is not significantly dilated, and the pressure in this chamber is elevated only slightly; therefore ECG signs of right ventricular hypertrophy are absent. As in a small VSD, a heart murmur (regurgitant systolic type) is produced by the left-to-right shunt. The normal sized mitral valve handles a significant amount of blood. This relative mitral stenosis produces a mid diastolic rumble at the apex. The pulmonary artery pressure is mildly elevated; therefore the intensity of the P_2 may increase slightly. With a large VSD, the overall heart size is larger than that seen with a moderate VSD because there is a much greater shunt. Since there is direct transmission of the left ventricle pressure through the large defect to the right ventricle, in addition to a much greater shunt, the right ventricle becomes enlarged and hypertrophied. Therefore the x-ray film shows

biventricular enlargement, left atrial enlargement, and greatly increased pulmonary vascularity. The ECG shows combined ventricular hypertrophy and sometimes left atrial hypertrophy. A large VSD usually results in congestive heart failure.[8, 9, 10] When a large VSD is left untreated, irreversible changes take place in the pulmonary arterioles. With gradual development of the "pulmonary vascular obstructive disease" or "Eisenmenger's syndrome", which may take years to develop, striking changes occur in the heart size, ECG and clinical findings. Since the pulmonary vascular resistance is notably elevated at this stage, approaching the systemic level, the magnitude of the left-to-right shunt decreases. This results in the removal of a volume overload placed on the left ventricle. Therefore the size of the left ventricle and the overall heart size decreases, and the ECG evidence of left ventricular hypertrophy disappears, leaving right ventricular hypertrophy because of the persistence of pulmonary hypertension.[11]

In chest x-ray, the main pulmonary artery and the hilar pulmonary arteries enlarge noticeably but the peripheral lung fields are ischemic. The bidirectional shunt causes cyanosis. Since the shunt is small, the loudness of the murmur decreases or may even disappear. The S_2 is loud and single because of pulmonary hypertension.[12]

Clinically, with a small VSD, the patient is asymptomatic with normal growth and development, with a moderate to large VSD, delayed growth and development, decreased exercise tolerance, repeated pulmonary infections and congestive heart failure are relatively common

during infancy, and with long standing pulmonary hypertension, a history of cyanosis and a decreased level of activity may be present.[13]

On physical examination, systolic thrill may be present at the lower left sternal border. Pericordial bulge and hyperactivity are present with a large VSD shunt. The intensity of P_2 is normal with a small shunt and moderately increased with a large shunt. The S_2 is loud and single in patients with pulmonary vascular obstructive disease. A grade 2-5/6 regurgitant systolic murmur is present at the lower left sternal border. It may be holosystolic or early systolic. An apical diastolic rumble is present with a moderate to large shunt.[14 - 15]

NORMAL GROWTH

The term "growth" is used to describe changes in size with maturation. Normal growth can occur only if the individual is healthy. Thus, measurement of height and weight is an essential part of the physical examination to determine if the individual's health is normal. Acute illnesses do not impair growth significantly, but long standing illness of the bowel, kidney, heart, lung, may lead to marked change in growth rate. Physicians who take care of children should have measuring devices available that permit an accurate determination of "length". Children who can stand erect steadily should have their height measured by a device that is fixed to a wall or some other study support. The standing height is best measured by having the subject stand with heels, buttocks, thoracic spine, and head touching the device. It is advisable to have the subject stand as tall or erect as possible to counter the slouching that tends to become more marked as the day progresses. A sliding device that projects from the