PARENTAL KNOWLEDGE, ATTITUDES AND PRACTICE TOWARDS THEIR CHILDREN’S CONGENITAL HEART DISEASE AND ITS IMPACT ON THEIR GROWTH IN SUDAN HEART CENTRE

By

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Supervisor

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FRCPCH, FRCP, FRCPI, DTCH, DCH
۹۸۷۳ ۱۰۹
DEDICATION

TO THE SOULS

OF MY

FATHER AND MOTHER
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ABSTRACT

The knowledge of one's own disease is an important determinant of health related behaviour. This hospital based cross sectional study was done in Sudan Heart Centre, during the period from April to July 2005. It involved 100 children with congenital heart disease and their parents. The children’s age ranged from 6 months to 15 years. This study was designed to assess parents' knowledge, attitudes and practice towards their children's congenital heart disease, its management and prevention of its complications. It also meant to identify determinants of parental knowledge and to assess the impact of parental knowledge on the growth of their children.

Although (30%) of the parents correctly mentioned their children's congenital heart disease, yet only (21%) of them correctly indicated the heart lesion or lesions diagrammatically. However, more than (77.3%) of the parents were aware of the indications and aims of previous surgical and catheter interventions. Seventy three percent of the parents correctly indicated that the heart defect was congenital without knowing the possible aetiological causes. Ninety two percent of the parents were aware of the symptoms attributable to the underlying heart disease. Unfortunately, of the 57 parents whose children were taking cardiac medications only (43.9%) and (15.8%) knew correctly the functions and important side effects of the medications respectively. With regard to exercise capacity, (56.9%) of the parents indicated its level appropriate for heart lesions. While (27%) of the parents had heard of the term infective endocarditis, only (09.4%) of the parents were aware of the need for antibiotics before dental procedures.

Significant determinants of parents’ knowledge of the nature of their children’s heart disease were cardiac diagnosis, occupation of parents and their educational level. No significant determinants of knowledge regarding symptoms, the impact of heart disease on exercise capacity and infective endocarditis were
identified. Parental negative attitudes and high concern were prevalent. Nutritional practice was inefficient in promoting growth. Ultimately, no significant correlation was found between parental knowledge and growth of their children.

The parents' knowledge about their children congenital heart disease is generally poor but did not correlate with their growth. Better educational programs and nutritional re-evaluation are recommended, along with providing better health care facilities and developing the concept of team management.
بلاسم(%21) وتمكنت 41% من الأباء أن يجعلوا أطفالهم حديثي الولادة. في عام 2005، تم التحكم في وظائف الأداء وأدارها للفحص في 15% من الأطفال الأباء الذين ذُكرا. كان هذا قد يشعر الأطفال والأمهات بشكل غريب. 

وحينها أن عادة الأداء، (35) 40% من الأباء الذين وجدوا بالعافية، أو الأصوات المثبتة (17) (17) 57.3% أظهرت أنهم كانوا يشعرون بالسعادة بالكامل. في عام 2005، كان ذلك في 27% من الأطفال الأباء الذين وجدوا بالعافية، أو الأصوات المثبتة (17) (17) 57.3% أظهرت أنهم كانوا يشعرون بالسعادة بالكامل. في عام 2005، كان ذلك في 27% من الأطفال الأباء الذين وجدوا بالعافية، أو الأصوات المثبتة (17) (17) 57.3% أظهرت أنهم كانوا يشعرون بالسعادة بالكامل.
تتضمن دراسة الأباء معرفة وامتيازات الأمهات، وبالتالي، تكون معناها في توفير التغذية، وصحة القلب للأولاد، وإكمال النمو والتطور. تستعرض هذه النماذج التي تشير إلى التحسينات، وتعزز الأباء، إضافة إلى ذلك، تعزز التعليم، ويحدث تغييرات جذابة في الأداء، وتعزز الصحة والرعاية، مما يؤدي إلى التحسينات في الأداء.
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>ACE</td>
<td>Angiotensin converting enzyme</td>
</tr>
<tr>
<td>AR</td>
<td>Aortic regurgitation</td>
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<td>AS</td>
<td>Aortic stenosis</td>
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<tr>
<td>ASD</td>
<td>Atrial septal defect</td>
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<tr>
<td>AVSD</td>
<td>Atrioventricular septal defect</td>
</tr>
<tr>
<td>AV</td>
<td>Atrioventricular valve</td>
</tr>
<tr>
<td>CPB</td>
<td>Cardiopulmonary bypass</td>
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<tr>
<td>CHD</td>
<td>Congenital heart disease</td>
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<tr>
<td>CHF</td>
<td>Congestive heart failure</td>
</tr>
<tr>
<td>COA</td>
<td>Coarctation of the aorta</td>
</tr>
<tr>
<td>CO</td>
<td>Cardiac output</td>
</tr>
<tr>
<td>CXR</td>
<td>Chest X-ray</td>
</tr>
<tr>
<td>DORV</td>
<td>Double outlet right ventricle</td>
</tr>
<tr>
<td>ECD</td>
<td>Endocardial cushion defect</td>
</tr>
<tr>
<td>ECG</td>
<td>Electrocardiogram</td>
</tr>
<tr>
<td>ECHO</td>
<td>Echocardiography</td>
</tr>
<tr>
<td>FO</td>
<td>Foramen ovale</td>
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<tr>
<td>FTT</td>
<td>Failure to thrive</td>
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<tr>
<td>GH</td>
<td>Growth hormone</td>
</tr>
<tr>
<td>IBW</td>
<td>Ideal body weight</td>
</tr>
<tr>
<td>IE</td>
<td>Infective endocarditis</td>
</tr>
<tr>
<td>IVC</td>
<td>Inferior vena cava</td>
</tr>
<tr>
<td>IV</td>
<td>Intravenous</td>
</tr>
<tr>
<td>Kcal</td>
<td>Kilo-calorie</td>
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<tr>
<td>Kcl</td>
<td>Potassium chloride</td>
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<td>Abbreviation</td>
<td>Full Form</td>
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<td>--------------</td>
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<tr>
<td>LA</td>
<td>Left atrium</td>
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<tr>
<td>L-R shunt</td>
<td>Left-to-right shunt</td>
</tr>
<tr>
<td>LV</td>
<td>Left ventricle</td>
</tr>
<tr>
<td>MPA</td>
<td>Main pulmonary artery</td>
</tr>
<tr>
<td>MR</td>
<td>Mitral regurgitation</td>
</tr>
<tr>
<td>MS</td>
<td>Mitral stenosis</td>
</tr>
<tr>
<td>MVP</td>
<td>Mitral valve prolapse</td>
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<tr>
<td>PA</td>
<td>Pulmonary artery</td>
</tr>
<tr>
<td>PAPVR</td>
<td>Partial anomalous pulmonary venous return</td>
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<tr>
<td>PBE</td>
<td>Prophylaxis bacterial endocarditis</td>
</tr>
<tr>
<td>PBF</td>
<td>Pulmonary blood flow</td>
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<tr>
<td>PDA</td>
<td>Patent ductus arteriosus</td>
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<td>PGE</td>
<td>Prostaglandin</td>
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<tr>
<td>PS</td>
<td>Pulmonary stenosis</td>
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<tr>
<td>PVM</td>
<td>Pulmonary vascular marking</td>
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<td>PVOD</td>
<td>Pulmonary vascular obstructive disease</td>
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<tr>
<td>PVR</td>
<td>Pulmonary vascular resistance</td>
</tr>
<tr>
<td>RA</td>
<td>Right atrium</td>
</tr>
<tr>
<td>R-L shunt</td>
<td>Right-to-left shunt</td>
</tr>
<tr>
<td>RV</td>
<td>Right ventricle</td>
</tr>
<tr>
<td>RVH</td>
<td>Right ventricular hypertrophy</td>
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<tr>
<td>RVOT</td>
<td>Right ventricular outflow tract</td>
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<tr>
<td>SVC</td>
<td>Superior vena cava</td>
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<tr>
<td>SVR</td>
<td>Systemic vascular resistance</td>
</tr>
<tr>
<td>SVT</td>
<td>Supra ventricular tachycardia</td>
</tr>
<tr>
<td>TA</td>
<td>Tricuspid atresia</td>
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<tr>
<td>TAPVR</td>
<td>Total anomalous pulmonary venous return</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Description</td>
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<td>--------------</td>
<td>--------------------------------------------------</td>
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<tr>
<td>TGA</td>
<td>Transposition of great artery</td>
</tr>
<tr>
<td>TOF</td>
<td>Tetralogy of Fallot</td>
</tr>
<tr>
<td>TR</td>
<td>Tricuspid regurgitation</td>
</tr>
<tr>
<td>TS</td>
<td>Tricuspid stenosis</td>
</tr>
<tr>
<td>VSD</td>
<td>Ventricular septal defect</td>
</tr>
<tr>
<td>WPW</td>
<td>Wolff-Parkinson-White (syndrome)</td>
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</tbody>
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CHAPTER ONE

INTRODUCTION AND LITERATURE REVIEW
CHAPTER ONE

1. INTRODUCTION AND LITERATURE REVIEW

Congenital abnormalities have become the main cause of heart disease in children especially in developed countries where incidence of rheumatic fever has declined\(^{(1)}\).

1.1. DEFINITION.

Congenital heart diseases (CHD) are abnormalities or defects of the heart and its major blood vessels that develop before birth. These heart defects result from failure of the heart or the blood vessels near the heart to develop normally and ranges in severity from simple defects to complex abnormalities\(^{(2)}\).

1.2. HISTORICAL BACKGROUND.

Post mortem examinations held in the early sixteenth century disclosed many abnormalities of the heart and blood vessels. Credit for the first clinical description of ventricular septal defect (VSD) is generally given to Roger's article published in 1879. In 1898, Eisenmenger described a patient with (VSD), cyanosis and pulmonary hypertension\(^{(3)}\). In the 1930s with the development of cardiac catheterization many congenital anomalies became diagnosable. Before 1938, surgical treatment was non-existent and few lived beyond adolescence\(^{(4)}\). The first surgical cure of a patent ductus arteriosus (PDA) was achieved by Robert Gross in 1938\(^{(5)}\). The past 40 years have witnessed major changes in surgical treatment and
early corrective surgery becomes feasible and many children have an excellent outlook for long-term survival and quality of life\textsuperscript{(4,6)}.

1.3. EPIDEMIOLOGY.

The reported incidence of (CHD) is 0.5-0.8 % of live births\textsuperscript{(7)}. The incidence is higher in stillborns (3-4%), abortuses (10-25%) and premature infants (2%) excluding patent ductus arteriosus\textsuperscript{(8)}. The relative frequency of the most common congenital cardiac lesions is as follows: VSD (25-30 %), atrial septal defect (ASD) 6-8 %, PDA (6-8 %), coarctation of the aorta (COA) 5-7 %, tetralogy of Fallot (TOF) 5-7 %, pulmonary stenosis (PS) 5-7%, aortic stenosis (AS) 4-7 % and transposition of the great arteries (TGA) 3-5 %.

1.3.1. Epidemiology of CHD in Developed Countries.

The reported prevalence of (CHD) in Washington was 3-5 and 4-5/1000 live births in the years 1981 and 1984, respectively\textsuperscript{(9)}. In Canada, the prevalence of (CHD) was found to be 5.5/1000 live births\textsuperscript{(10)}. In Italy, the prevalence of (CHD) was 4.6\%\textsuperscript{(11)}. In Australia, the incidence of (CHD) was 17.5/1000 live birth\textsuperscript{(12)}.

1.3.2. Epidemiology of CHD in Developing Countries.

There was no evidence of major variation in incidence of heart defects, based on racial, geographical, socioeconomical or secular factors\textsuperscript{(13,14)}. The frequency of the different types of (CHD) showed good general agreement with developed countries\textsuperscript{(15)}. However, over the last two decades, there has been a growing
awareness and recognition of heart disease in Africa, in particular (CHD) have now been recognized as an important cause of morbidity and mortality\(^{(16)}\). An incidence rate of (CHD) of 7/1000 hospital admission was reported in Tanzanian children\(^{(17)}\). An incidence rate of 3.9/1000 was reported in nursery and school children in Soweto, South Africa\(^{(18)}\). In Nigeria a study carried out by Jaiyesimi F and Antia AU, revealed that VSD was the commonest lesion (35%), PDA (22%), TOF (10%), PS (09%) and (7.5%) was the ASD\(^{(19)}\). In Kenya, the prevalence of (CHD) found to be 1.7/1000, and the frequency of cardiac lesions was: VSD (30.1%), PDA (27.9%), TOF (5.9%) and (4.4%) was the ASD\(^{(20)}\). In Saudi Arabia, in the south western region Fuad Abbag studied the pattern of (CHD), 335 patients were found to have (CHD), VSD represented (32.5%), PDA (15.8%) and (10.4%) was the ASD\(^{(21)}\).

1.3.3. Previous Studies in Sudan.

The situation in Sudan is similar to other African countries. In 1988, a study was done by Amal Mohamed El Nour, to determine the pattern of echocardiography in Sudanese children with heart disease. She came to a conclusion that (60 %) of the children had (CHD). VSD and TOF were the commonest lesions\(^{(22)}\).

Regarding the pattern of (CHD) in Sudan, a study conducted over 2 years period (1991-1993) by A. I. El Hag, concluded that almost all types of defect were represented, the commonest being VSD, TOF, PS, PDA, atrioventricular septal defect (AVSD) and ASD\(^{(23)}\).
In 1996 a study was conducted by Ibtisam Ibrahim Abdulla, to determine the prevalence of rheumatic and congenital heart disease in a group of school children and to outline the methods used for diagnosis. In this study the prevalence of (CHD) was 2.3 per 1000 while VSD was the commonest lesion comprising sixty percent\(^{(24)}\). In 1996 a study was carried by Awatif Khogali reported that (CHD) caused (27.5 %) of congestive heart failure\(^{(25)}\).

The most recent study in 2004 was conducted by Intisar Sid Ahmed concluded that the pattern of (CHD) in Sudan is comparable to the reported pattern in the other parts of the world\(^{(26)}\).

### 1.4. AETIOLOGY.

As with most congenital defects, the precise aetiology is unknown but both genetic and environmental factors have been identified. Inheritance is mostly polygenic but occasionally a single gene mutation occurs. Incidence of (CHD) is high in children with chromosomal abnormalities. In particular, (CHD) is found in (90\%) of patients with trisomy 18, (50\%) of patients with trisomy 21 and (40\%) of patients with Turner syndrome. Other genetic factors were suspected to play a role in (CHD); certain types of VSD (supracristal) are more common in Asian children. The risk of recurrence of (CHD) increases when there is positive family history\(^{(27)}\).

A well characterized genetic cause of CHD is the deletion of a large region of chromosome 22q11, known as the DiGeorge critical region. This deletion is most
often seen in association with either the DiGeorge syndrome or the Shprintzen (velocardiofacial) syndrome. The specific cardiac anomalies in these patients are conotruncal defects (TOF, truncus arteriosus, double-outlet right ventricle, subarterial VSD) and branchial arch defects (COA, interrupted aortic arch, right aortic arch). Although the risk of recurrence is extremely low in the absence of a parental 22q11 deletion, it is (50%) if one of the parents carries the deletion(8).

The hypertrophic cardiomyopathy is linked to missense mutations in the beta-myosin heavy chain gene on chromosome 14, the cardiac troponin T gene on chromosome 1q3, alpha-tropomyosin on chromosome 15q2, and cardiac myosin-binding protein C on chromosome 11q11. X-linked cardiomyopathies have been linked to dystrophin gene on Xp21. The heritable arrhythmia, most notably the long Q-T syndromes, has been linked to mutations of genes coding for subunits of cardiac potassium and sodium channels(28,29).

Cardiac defects occur in a large number of syndromes and non cardiac malformations were found in identifiable syndromes in about (25%) of patients with CHD. These syndromes include De Lange syndrome (VSD), Holt-Oram syndrome (ASD), William syndrome (supra valvular AS).

Maternal diabetes mellitus, phenylketonuria, systemic lupus erythematosus, first trimester rubella infection and exposure to drugs such as lithium, indomethacin,
ethanol, warfarin, thalidomide, antimetabolites and anticonvulsants agents are environmental factors associated with increased incidence of CHD\(^8\).

1.5. CLASSIFICATION.

Congenital heart defects are classified into two broad categories: acyanotic and cyanotic lesions:

1.5.1. Acyanotic congenital heart lesions.

1.5.1.1. Acyanotic lesions resulting in increased volume load.

The most common lesions in this group are those that cause left-to-right shunting: ASD, VSD, AVSD, and PDA. Additional lesions that impose a volume load on the heart include regurgitant lesions and cardiomyopathies\(^8\).

1.5.1.2. Acyanotic lesions resulting in increased pressure load.

The pathophysiology of these lesions is an obstruction to normal blood flow:

- Obstruction to ventricular outflow: PS, AS, COA.
- Obstruction to ventricular inflow: tricuspid stenosis (TS), mitral stenosis(MS), and cor triatriatum.

1.5.2. Cyanotic congenital heart lesions.

This group of lesions can be further subdivided according to pathophysiology and with the help of chest radiogram into:
1.5.2.1. Cyanotic lesions with decreased pulmonary blood flow (PBF).

These lesions include both an obstruction to PBF, at tricuspid valve or RV or pulmonary valve level, and a R-L shunt. Common lesions in this group include: TA, TOF, and various form of single ventricle with PS.

1.5.2.2. Cyanotic lesions with increased pulmonary blood flow.

Cyanosis is caused by either abnormal ventricular-arterial connection such as TGA, or it may be due to total mixing of systemic and pulmonary venous blood within the heart such as cardiac defects with a common atrium or ventricle, total anomalous pulmonary venous return (TAPVR), and truncus arteriosus.

The nine lesions which together make up (90 %) of all cases are: VSD, ASD, AVSD, PS, PDA, AS, TOF, and TGA the remaining (10 %) are rare complex anomalies\(^{(1,8)}\).

1.6. PATHOPHYSIOLOGY.

The anatomic and physiologic changes in the heart and circulation due to any specific congenital cardiocirculatory lesion are not static but rather progressive from prenatal life to adulthood. Thus, malformations that are benign or escape detection in childhood may become clinically significant in the adult\(^{(30)}\).
1.6.1. Pathophysiology of acyanotic defects.

1.6.1.1. Left-to-right shunt lesions.

These lesions when large cause volume overload of the LA, LV and RV (VSD) or RA and RV (ASD). PBF is increased to varying degrees, and pulmonary hypertension may result. With a long-standing large VSD (and much later for ASD), pulmonary vascular obstructive disease (PVOD) develops, with severe pulmonary hypertension and cyanosis from a R-L shunt\(^{(31)}\).

1.6.1.2. Obstructive and valvular regurgitant lesions.

- **Obstruction to ventricular outflow.**

  1. **Coarctation of the Aorta (COA).**

     There is narrowing of the aorta, most commonly in the upper thoracic aorta. More than (50%) of the patients with COA have bicuspid aortic valve. Preductal COA is frequently associated with other cardiac defects (40%), such as VSD, PDA, or TGA. Collateral circulation is poorly developed. These patients become symptomatic very early in life. Postductal COA is less frequently associated with other cardiac defects and usually does not produce symptoms in infancy.

  2. **Aortic Stenosis (AS).**

     The stenosis may be valvular, subvalvular, or supravalvular. Valvular stenosis most commonly due to a bicuspid valve. Supravalvular stenosis is often associated with William’s syndrome (mental subnormality, characteristic facies, and
PA stenosis). Subvalvular stenosis may be due to a simple diaphragm (discrete) or a long, tunnellike narrowing of the LV outflow tract.

3. Pulmonary Stenosis (PS).

Pulmonary stenosis may be valvular (90%), subvalvular (infundibular), or supravalvular. Dysplasia of the pulmonary valve is frequently seen with Noonan’s syndrome. Isolated infundibular PS is uncommon, usually associated with a large VSD (TOF). Depending on the severity of PS; a varying degree of RVH is present\(^{(31)}\).

- Valvular regurgitant lesions.

Valvular regurgitant lesions such as mitral regurgitation (MR) and aortic regurgitation (AR) are rather rare. Severe pulmonary valve regurgitation is extremely rare, except in a postoperative state. Although tricuspid valve regurgitation is rare it had been observed that Ebstein’s malformation of the tricuspid valve has a high incidence (2% vs 0.5%) in Sudan than that reported in the Western literatures\(^{(32)}\). In general, when regurgitation is severe, the chambers both proximal and distal to a regurgitant valve become dilated with volume overload of these chambers.
1.6.2. Pathophysiology of Cyanotic Congenital Heart Defects.

1.6.2.1. Transposition of Great Arteries.

Transposition of great arteries is the most common cyanotic (CHD) in newborns. The aorta arises anteriorly from the RV, and the PA arises posteriorly from the LV. The result is complete separation of the two circuits, with hypoxaemic blood circulating in the body and hyperoxaemic blood circulating in the pulmonary circuit. Defects that permit mixing of the circulations are necessary for survival. A VSD is present in (40%) of the cases. In neonates with poor mixing of the two circulations, progressive hypoxaemia and acidosis develop, with resulting early death. Congestive heart failure (CHF) develops in the first week of life in many patients. The RV is the systemic ventricle\(^\text{(33)}\).

1.6.2.2. Tetralogy of Fallot.

The original description of TOF includes four abnormalities: a large VSD, right ventricular outflow tract (RVOT) obstruction, RVH, and overriding of the aorta. However, only two abnormalities are important: a VSD large enough to equalize pressure in both ventricles and an RVOT obstruction. The RVOT may be in the form of infundibular stenosis (50%), pulmonary valve stenosis (10%), or a combination of the two (30%). In the most severe form of the anomaly the pulmonary valve is atretic (10%). Because of the nonrestrictive VSD, systolic pressure in the RV and the LV are identical. Depending on the degree of the RVOT
obstruction, either a L-R or R-L shunt is present. The major heart murmur audible in cyanotic TOF originates from the RVOT obstruction, rather than the VSD\(^{(31)}\).

**1.6.2.3. Tricuspid Atresia.**

The tricuspid valve is absent, and the RV and PA are hypoplastic, with decreased PBF. The great arteries are transposed in (30\%) of cases. Associated defects, such as ASD, VSD or PDA are necessary for survival. Systemic venous return is shunted from the RA to the LA, with resulting hypertrophy and enlargement of the RA. The LA and LV are large because they handle both systemic and pulmonary venous return. The degree of cyanosis is proportionally related to the amount of PBF.

**1.6.2.4. Pulmonary Atresia with Intact Interventricular Septum.**

The pulmonary valve is atretic, and the interventricular septum is intact. The RV cavity is usually hypoplastic, with a thick ventricular wall (peach pit RV, type I) occurring in about (85\%) of the cases. Occasionally the RV is of normal size with significant TR (type II). An interatrial communication is necessary for survival. Pathophysiologic findings are similar to those of tricuspid atresia. PBF depends on the patency of PDA; closure of PDA after birth results in death.

**1.6.2.5. Total anomalous pulmonary venous return.**

The pulmonary veins drain into the RA or its venous tributaries, rather than into the LA:
- Supracardiac (50%): the common pulmonary vein drains into the superior vena cava (SVC), via the left SVC (vertical vein) and the left innominate vein.

- Cardiac (20%): the common pulmonary vein drains into the coronary sinus or the pulmonary veins enter the RA separately through four openings.

- Infracardiac (subdiaphragmatic, 20%): the common pulmonary vein drains to the portal vein, hepatic vein or inferior vena cava (IVC).

- Mixed type (10%): combination of the above types.

An interatrial communication is necessary for survival. The left side of the heart is relatively small. Pulmonary venous return reaches the RA, in which systemic venous blood and pulmonary venous blood are completely mixed, before it is shunted to the LA through an ASD. Oxygen saturations in the systemic and pulmonary circulations are the same, resulting in systemic arterial desaturation. The level of systemic arterial oxygen saturation is proportional to the amount of PBF. When there is no obstruction to pulmonary venous return (seen in the supracardiac and cardiac types), pulmonary venous return is large and the systemic arterial blood is only minimally desaturated. When there is obstruction to pulmonary venous return (seen in the infracardiac type), pulmonary venous return is small and the patient is extremely cyanotic, with signs of pulmonary oedema on CXRs.
1.6.2.6. **Ebstein's anomaly.**

The leaflets of the tricuspid valve are displaced into the RV cavity; thus a portion of the RV is incorporated into the RA (atrialized RV) and functional hypoplasia of the RV results. At the same time, tricuspid valve regurgitation results. An interatrial communication is present in the majority (80%) of patients, with resulting R-L atrial shunt. The RA is massively dilated and hypertrophied. In addition, attacks of supraventricular tachycardia (SVT) are frequent, with or without associated Wolff-Parkinson-White (WPW) syndrome.

1.6.2.7. **Single Ventricle (Common ventricle, Univentricular heart).**

Both AV valves empty into a common ventricular chamber. A rudimentary infundibular chamber is usually present and communicates with the common ventricular chamber. A great artery arises from the common chamber, and the other great artery usually arises from the infundibular chamber. TGA is present in (85%) of the cases, and AS or PS is common. A high incidence of asplenia or polysplenia syndrome is found.

There is a complete mixing of systemic and pulmonary venous blood; therefore oxygen saturation in the aorta and PA is identical. The systemic oxygen saturation is proportional to the amount of PBF. With decreased PBF (seen in patients with associated PS), marked cyanosis results. In patients without PS, PBF is large and cyanosis is minimal.
1.6.2.8. Double-outlet Right Ventricle (DORV).

Both the aorta and PA arise side-by-side from the RV. The only outlet from the LV is a large VSD. DORV may be subdivided, depending on the position of the VSD (and further by the presence of PS).

- Subaortic VSD (50%-70%). PS is common (50%) in this (Fallot) type.
- Subpulmonary VSD (Taussig-Bing anomaly).
- Doubly committed VSD.
- Remote VSD.

Pathophysiology of DORV is determined primarily by the position of VSD and the presence or absence of PS. With subaortic VSD, oxygenated blood from the LV is directed to the aorta, and desaturated systemic venous blood to the pulmonary artery, producing mild or no cyanosis. The PBF is increased in the absence of PS, resulting CHF. Therefore clinical findings resemble those of a large VSD with pulmonary hypertension and CHF. With subpulmonary VSD, oxygenated blood from the LV is directed to the PA and desaturated blood from the systemic vein to the aorta, producing severe cyanosis. The PBF is increased with the decrease in pulmonary vascular resistance (PVR). Clinical findings therefore resemble those of TGA. In the presence of PS, clinical findings resemble those of TOF. With the VSD close to both semilunar valves or remotely located from these valves, cyanosis of mild degree is present and the PBF is increased\(^{31,33}\).
1.7. CLINICAL PRESENTATION.

1.7.1. Symptoms.

Children with (CHD) can either present with symptoms of congestive heart failure (breathlessness particularly after the exertion of feeding or crying, sweating, poor feeding, recurrent chest infections), failure to thrive and cyanosis or can be asymptomatic discovered during routine examination\(^1\). Suspicion of (CHD) should be raised if feeding takes more than 30 minutes. A history of feeding difficulty often precedes overt CHF, even if only by six to 12 hours\(^{34}\).

Symptoms of specific types of congenital heart diseases are as follows:

- **Patent ductus arteriosus**: quick tiring, slow growth, susceptibility to pneumonia, rapid breathing. If the ductus is small, there are no symptoms.
- **Obstruction defects**: cyanosis, chest pain, tiring easily, dizziness or fainting, congestive heart failure and high blood pressure.
- **Septal defects**: difficult breathing, stunted growth. Sometimes there are no symptoms.
- **Cyanotic defects**: cyanosis, sudden rapid breathing or unconsciousness and shortness of breath and fainting during exercise\(^{35}\).
1.7.2. Physical examination.

The presence of cyanosis, respiratory distress, stunted growth, associated malformations and chromosomal syndromes which are suspected by the presence of dysmorphic features, all are indicatives for the presence of (CHD). The presence or absence of a heart murmur is unreliable as a basis for the diagnosis of (CHD). Transient murmurs are often heard in infants without cardiac abnormalities. Furthermore, a murmur is not present in many severe forms of (CHD), such as TA, COA and TGA. The nature of the murmur (harsh, blowing or musical), along with other heart sounds, is useful in differentiating mild defects from severe abnormalities\textsuperscript{(36)}. The infant’s age at the time a murmur is first heard is also helpful in determining the chance and risk of congenital heart disease\textsuperscript{(7)}.

Femoral and brachial pulses should be palpated. In infants with some obstructive lesions of the left side of the heart, femoral pulses may be palpable, but one or both brachial pulses may not be palpable\textsuperscript{(34)}. In infants with PDA, femoral pulses may be present at birth but may become diminished or absent with closure of the ductus arteriosus at three to 14 days of age\textsuperscript{(34,36)}.

Suspicion of cyanosis should be confirmed by the nitrogen washout test or by pulse oximeter\textsuperscript{(37)}. Accurate assessment of height, weight and head circumference with plotting them on standard growth charts is essential as both chronic HF and
chronic cyanosis result in failure to thrive. Growth failure is usually manifested by poor weight gain.

1.8. INVESTIGATIONS.

Important haematological investigations for patients with CHD are determination of haemoglobin and haematocrit. In acyanotic infants with large L-R shunts, the onset of HF often coincides with the normal physiologic anaemia of infancy. Polycythaemia is frequently noted in cyanotic patients with R-L shunts\(^8\).

1.8.1. Specific non invasive investigations.

Diagnosis of congenital heart disease is usually made by clinical examination aided by:

1.8.1.1. Chest roentgenography (CXR).

CXR provides an idea about the: heart size and silhouette, enlargement of specific chambers, pulmonary blood flow or pulmonary vascular markings (PVM), and other information (e.g., lung parenchyma, spine, bony thorax, and abdominal situs).

1.8.1.2. Electrocardiography (ECG).

The standard 12-lead electrocardiogram is a recording of the electrical activity from cardiac muscle cells onto each lead on the surface of the torso. Analysis of the ECG includes the rate, rhythm, P wave morphology, PR interval, QRS complex, QT interval, and ST segment. It also gives an idea about right and left
ventricular hypertrophy, and bundle branch block. The ECG also provides insight into the metabolic state of the cardiac cell such as hyperkalemia\textsuperscript{(38)}.

1.8.1.3. Echocardiography (ECHO).

Echocardiography remains the primary diagnostic modality; it is a non-invasive investigation which dramatically reduced the requirement for invasive studies such as cardiac catheterization. The echocardiographic examination can be used to evaluate cardiac structure in (CHD), estimate intracardiac pressures and gradients across stenotic valves and vessels, quantitate cardiac contractile function (both systolic and diastolic), determine the direction of flow across a defect, examine the integrity of the coronary arteries, and detect the presence of vegetations from endocarditis, as well as the presence of pericardial effusion, cardiac tumors, and chamber thrombi. ECHO may also be used to assist in the performance of pericardiocentesis, balloon atrial septostomy, and endocardial biopsy\textsuperscript{(39)}. A complete ECHO examination usually entails a combination of M-Mode and two-dimensional imaging, as well as pulsed, continuous and colour Doppler flow studies\textsuperscript{(40)}.

Fetal echocardiography can be used to evaluate cardiac structure or disturbance in cardiac rhythm, and may be capable of diagnosing congenital heart disease as early as 17-19 wk of gestation; however accuracy at this early stage is limited. Between 10 and 14 weeks of pregnancy, physicians also may use ultrasound to look
for a thickness at the nuchal translucency, a pocket of fluid in back of the embryo's neck, which may indicate a cardiac defect in 55% of cases\(^{(41)}\).

1.8.1.4. Magnetic resonance imaging (MRI), Electron beam computed tomography, and Radionuclide studies.

MRI provides excellent anatomic evaluation and often yields more information than angiography. It is useful in evaluating areas that are less well visualized by ECHO, such as distal branch PA anatomy and anomalies in systemic and pulmonary venous return. Magnetic resonance angiography shows changes in wall thickening, chamber volume, and valve function\(^{(42)}\).

Electron beam computed tomography is useful in evaluating branch pulmonary arteries, anomalies in systemic and pulmonary venous return, and great vessel anomalies such as COA. Radionuclide angiography is used to detect and quantify shunts and to analyze the distribution of blood flow to each lung, and to quantify the success of balloon angioplasty and intravascular stenting procedures. *Gated blood pool scanning* is used to calculate haemodynamic measurements, quantify valvular regurgitation, and detect regional wall motion abnormalitie. *Thallium imaging* is performed to evaluate cardiac muscle perfusion\(^{(8)}\).
1.8.2. Specific invasive procedures.

1.8.2.1. Cardiac catheterization.

It is an invasive investigation which is only performed in specialized units. It enables the pressure and saturation within the cardiac chambers to be measured and with angiocardiography permits precise anatomical and physiologic diagnosis\(^1\).

Major indications for cardiac catheterization include:

1- Presurgical evaluation of cardiac anatomy or shunt size, or both, in children with CHD when ECHO evaluation is incomplete.

2- Evaluation of pulmonary vascular resistance and its response to vasodilators or oxygen.

3- Follow-up after surgical repair or palliation of complex CHD.

4- Myocardial biopsy for the diagnosis of cardiomyopathy or screening for cardiac rejection after transplantation.

5- Interventional cardiac catheterization.

6- Electrophysiologic study and transcatheter ablation.

1.9. MANAGEMENT.

Most patients with mild (CHD) require no treatment, parents and child should be reassured and made aware that normal life is expected and no restriction of child activity is required.
1.9.1. Medical treatment.

1.9.1.1. Management of heart failure.

- **Supportive therapy**: are important to increase tissue oxygen supply, decrease tissue oxygen consumption, and correct metabolic abnormalities. Infants in heart failure feel better when semi-recumbent, children prefer to sit up. Cool, humidified oxygen by tent, mask or nasal prongs may be useful in hypoxic patients. The need for calories is often urgent. Feeding by nasogastric tube is necessary and if given continuously it may be better tolerated in a sick infant. Continuous overnight tube feeding may help an older child to thrive\(^{(43)}\).

- **Medical Therapy**: These include correction of hypoglycaemia, anaemia, acidaemia, and administration of antibiotics if infection is a contributing factor.

  *Inotropic agents*: in severe acute HF, intravenous infusion of dopamine (5\(\mu\)g / kg /min) will improve cardiac output. In less severe cases digoxin; a cardiac glycoside can be used. Digoxin is particularly effective in infants and children with poor myocardial function (endocardial fibroelastosis, myocaditis, cardiomyopathy or obstructive lesions like coarctation). It is much less effective when there is volume overload (VSD, PDA, AVSD, truncus arteriosus) and often not used in such lesions. It should not be used in preterm infant with a PDA\(^{(43)}\).
Diuretics; frusemide (1-3 mg / kg /day in 2-3 divided doses) is the most effective loop diuretic. If used in long term a potassium supplement (KCl 2 mmol /kg / day in 2 divided doses) or a potassium sparing diuretic such as spironolactone (2 mg / kg /day in 2 divided doses) is necessary.

Vasodilators; these are angiotensin converting enzyme (ACE) inhibitors used in children with valvular lesions, myocardial disease or large L-R shunts to supplement diuretic therapy. They should be used with caution to avoid hypotension. Captopril (0.5 to 3 mg / kg / day in 3 divided doses) and Enalapril (2 to 10 mg bid). It is reported that ACE inhibitors significantly reduce the mortality and hospital admissions for HF. Moreover, they delay the onset of symptoms in patients with asymptomatic left ventricular dysfunction\textsuperscript{(44,45)}.

1.9.1.2. Management of cyanosis.

In a cyanosed newborn, prostaglandin (PGE) often re-opens and maintains the patency of the ductus arteriosus and therefore relieve cyanosis in infants with duct dependent pulmonary blood flow (TA, PA) and improve perfusion in infants with duct dependent systemic blood flow (COA). Caynosis in TGA may be improved but not in persistent foetal circulation or TAPVR. The usual starting dose is intravenous (IV) infusion at a rate $0.005 \mu g / kg / min$, increase in multiples of doses to a maximum of $0.05 \mu g / kg / min$ until the cyanosis improves. Side
effects include central apnea (occurs in 12% of babies, it is dose related and unusual at the quoted doses), fever, hypotension, jitteriness, and cutaneous vasodilatation.

Cyanotic spells is treated by placing the child in a knee-chest position, giving oxygen by mask at 5 to 8 liters per minute and inject morphine intravenously (0.1 mg / kg). If spell is severe sodium bicarbonate, 1 mg / kg IV is given, and volume expansion with crystalloid or blood should be given if cyanosis persists. Propranolol, 0.1 mg / kg IV, may be effective in a protracted spell that does not respond to the preceding measures\(^{46}\).

1.9.2. Surgical treatment.

Surgery is performed in a high proportion of infants with severe cardiac anomalies. Cardiac surgery in the newborn, infant and child is divided into two types of procedures; open-heart procedures and closed procedures. Open-heart procedures utilize cardiopulmonary bypass (CPB) with cardiac arrest and some degree of hypothermia, with or without circulatory arrest, and are required for repair of intracardiac anomalies (e.g., VSD, TOF and TGA). Closed procedures do not require CPB and are performed for repair of extracardiac anomalies (e.g., COA, PDA) or palliative procedures (systemic-to-pulmonary [S-P] shunt procedures or PA banding).

The current trend is to carry out total repair of (CHD) at an early age, whenever such repair is technically possible. This approach is made possible by improved
surgical technique and better understanding of postoperative intensive care of newborns and small infants. Early total repair negates the need for palliative procedures and possibly prevents permanent damage to the cardiovascular system, which is known to develop in certain CHDs. Recommendations for early repair of (CHD) in the neonate or small infant are made on an individual basis in a joint cardiology-cardiac surgery conference; the indications vary among institutions\(^{31}\).

"Palliative" surgery offers relief of major circulatory disturbances, and includes: PA banding, Blalock-Taussig shunt, Blalock-Hanlon operation, Waterston’s shunt, Pott’s operation, Gore-Tex interposition shunt and Noorwood procedure.

"Definitive" surgery attempts to restore the cardiac anatomy to normal, and includes: Rastelli repair, Rashkind procedure, Senning procedure, Fontan procedure, Glenn shunt, Jateene procedure and Mustard technique\(^{47}\).

1.9.3. Interventional Catheterization.

Nonsurgical treatment of certain cardiac defects is routine with interventional cardiac catheterization. Interventional techniques include balloon dilatation of stenotic valves and arteries, embolization of abnormal vascular connections, and catheter closure of both intracardiac and extracardiac defects. The procedure most often used is balloon valvuloplasty, which has yielded excellent results for AS,
although as with surgery, AS often recurs as the child grows and multiple procedures may thus be required\textsuperscript{(35,48,49)}.

Balloon angioplasty is the procedure of choice for patient with re-stenosis of COA after earlier surgery. Valvular PS can be also treated successfully by balloon angioplasty. The clinical results of this procedure are similar to those obtained by open heart surgery, but without the need for sternotomy or prolonged hospitalization. The risk of angioplasty and valvuloplasty procedures on the left side of the heart is higher in younger patients, especially infants less than 1 yr of age, because of complications at the site of femoral artery catheterization. Low-profile catheters have significantly reduced, though not totally eliminated these complications.

The use of catheter-introduced devices to close (CHD) has produced good results. Currently, several devices (Clamshell, Helex, Button) are undergoing clinical trials for closure of small to moderate sized ASDs. Umbrella or bag devices may also be introduced to close a large PDA not amenable to coil closure. Patients with apical muscular VSDs, especially when associated with other cardiac defects, may be candidates for catheter closure with a clamshell-type device because of the higher risk of standard surgery\textsuperscript{(48)}. When all other options fail, some patients may need a heart or heart-lung transplant, an increasing number of patients have undergone these procedures with favorable outcome.
Children with congenital heart disease require lifelong monitoring, even after successful surgery. The American Heart Association recommends regular dental check-ups and the preventive use of antibiotics to protect patients from heart infections, or endocarditis. However, a 2003 study reported that preventive antibiotics are underused in people with congenital heart disease. Many patients did not understand the risk of endocarditis\(^{(50)}\).

1.9.4. Prevention and Prognosis.

Congenital heart disease cannot be predicted and most types cannot be prevented. General measures to ensure the birth of a healthy baby, such as avoiding excess alcohol, not taking drugs, and avoiding exposure to rubella and environmental toxins, will help prevent some cases. The outlook for children with congenital heart disease has improved markedly in the past two decades. Many types of congenital heart disease that would have been fatal now can be treated successfully. Research on diagnosing heart defects when the fetus is in the womb may lead to future treatment to correct defects before birth. Promising new prevention methods and treatments include genetic screening and the cultivation of cardiac tissue in the laboratory that could be used to repair congenital heart defects. As scientists continue to advance the study of genetics, they also will better understand genetic causes of many congenital heart diseases. For example, scientists
just discovered a potential cause of atrioventricular canal defect in the fall of 2003\(^{(2,41)}\).

1.10. COMPLICATIONS.

The complications that can jeopardize the health and life of a child with (CHD) include heart failure, repeated chest infection, growth impairment, bacterial endocarditis (BE) and pulmonary vascular disease (Eisenmenger’s syndrome). Acute and chronic hypoxemias are the major cause of ill health in cyanotic children. Arrhythmia which may be transient or permanent can occur in some (CHD), as a result of drug toxicity (digoxin) or following surgery for (CHD). Sudden death may occur in patients with corrected, palliated or unoperated (CHD) also occurs in patients with long QT syndrome\(^{(8)}\).

1.11. HEALTH MAINTENANCE OF THE CHILD WITH CONGENITAL HEART DISEASE AND HIS FAMILY.

Health is a state of complete physical, mental, and social wellbeing and not merely the absence of disease or infirmity\(^{(51)}\). To maintain the health of a child with (CHD), regular visits should be scheduled to a multidisciplinary team. The frequency of visits should be individualized depending on the condition of the child. In these visits several issues should be discussed with the parents. This includes the nature of their child’s (CHD), treatment options, nutrition, vaccination, endocarditis prophylaxis and recommendation for physical activity\(^{(52)}\).
1.11.1. Nutrition and growth.

Infants and children with congenital heart disease present special nutritional challenges. It is not uncommon to have some degree of growth failure. In almost all cases, nutritional intervention is required to ensure adequate nutritional intake to decrease the risk of malnutrition and promote weight gain, linear growth and age-appropriate development.

Many children with (CHD) are able to breast-feed and gain adequate weight as well as enjoy the other benefits of breast feeding\(^{(53)}\). In children who are unable to gain sufficient weight with breast feeding, supplementation options include a formula with a high caloric density, nocturnal enteral feeding or continuous 24-hour feeding with a nasogastric or duodenal tube. The latter is the most effective form of supplementation as proved by Schwartz SM\(^{(54)}\). A caloric intake of 140 to 200 calories per kg per day is needed to induce catch-up growth\(^{(55)}\). Growth often varies according to the type and severity of heart disease. An eight-ounce to one-pound (226.8 to 453.9 grams) gain in a month may be an acceptable weight gain for a baby with a heart defect. Infants and children with CHF or cyanosis tend to gain weight slowly\(^{(56,57,58)}\).

Since children with (CHD) often have poor appetites, high-calorie foods and snacks can play a very important role in providing good nutrition. Restriction of fat in the diet is unadvisable, particularly in the first two years of life. It is needed to
help children grow and develop properly. That is why lower fat milks (2%, 1% or fat-free [skim]) are not recommended until children are two years of age or older\(^{(57)}\).

Generally, growth improves with increasing age as a result of the decreased size of a L-R shunt, closure of a septal defect or development of pulmonary vascular obstructive disease. In most patients, catch-up growth is largely complete within six to 12 months of surgery\(^{(55)}\). Regular follow-up visits are essential to properly assess growth and to re-evaluate nutritional needs for the child.

1.11.2. Developmental considerations.

Children with (CHD) may fall behind in their development for several reasons, including:

- Inadequate nutrition does not meet the body's energy requirements, or allow for proper growth and development of muscles, bones, and brain nerve cells.

- Inadequate nutrition does not meet the body's energy requirements, causing children to tire quickly or not be able to physically keep up with others their same age.

- Illness and frequent or prolonged hospitalizations may prevent the child from receiving stimuli that help with development, such as being played with, talked to, held or touched.

Parents of children with (CHD) can play an active role in promoting the development of their children, at home or in the hospital. Physicians, nurses,
physical therapists and other healthcare team members will provide appropriate guidelines that are tailored for each individual child. Some ways that parents can encourage the development of their children may include the following:

- Touching and talking to the child can soothe him/her and provide reassurance, especially in the intensive care unit or right after surgery, even if he/she have been sedated.
- Encourage light physical activity after surgery, as directed by the child's physician.
- Provide the child with a variety of toys and other objects that stimulate his/her senses of hearing, vision, touch and smell, even while in the hospital.
- A physical therapist can be of assistance in providing exercises that are safe for children of all ages to encourage their development.
- Allow the child to participate in everyday family activities, within his/her physical limitations. Children also learn new skills from interaction with brothers, sisters and friends.

1.11.3. Immunization.

Routine immunization schedule should be followed with a few exceptions: varicella vaccine and measles, mumps and rubella vaccine are indicated at 12 months of age rather than 15 months; pneumococcal vaccine is recommended at two years of ages and influenza vaccine should be given yearly beginning at six months
in this higher-risk population. Children who are having heart surgery should not have the measles-mumps-rubella immunization within 10 days of their surgery and it is best to avoid all immunizations the week before and for 6 weeks after heart surgery. If possible, children should not have oral polio vaccine within 6 weeks of hospital admission\(^{(43)}\).

1.11.4. Prophylaxis against Bacterial Endocarditis.

Prophylaxis against bacterial endocarditis should be instituted in patients undergoing certain procedures, in accordance with the American Heart Association recommendations, as there is a small risk of subacute bacterial endocarditis when children with (CHD) have invasive dental procedures (extractions, filling, scaling and polishing). The importance of antibiotic prophylaxis should be stressed to the parents and it is useful if they have written information to give to their dentist. Oral prophylaxis is satisfactory and amoxicillin is the antibiotic of choice (50 mg / kg orally one hour before dental treatment). Children who are already receiving an antibiotic that is used in prophylaxis should be given an alternate antibiotic prophylaxis regimen. In case of accidental injury, antibiotics may be prescribed for use during the initial healing period\(^{(43,52)}\).

1.11.5. Guidelines Regarding Activity Levels.

Most children with a congenital heart defect can be fully active. They don't need restrictions. In fact, pediatric cardiologists encourage children to do physical
activity that helps keep their hearts fit and can be enjoyed for a lifetime. Such healthful activities include swimming, bicycling, running, rope jumping and tennis. In a very few specific heart conditions, a pediatric cardiologist may advise that a child avoid some strenuous physical activities, such as competitive sports. However guidelines regarding activity and sports participation for children with (CHD) have been developed. Children should be assessed individually, based on their clinical condition and the type of activity in which they plan to engage. Basic assessment by a paediatric cardiologist includes graded exercise testing on treadmill or bicycle to measure heart rate, rhythm and blood pressure. Patients with severe forms of cyanotic (CHD), AS, COA with residual hypertension are at risk of sudden death during exercise. In the absence of pulmonary hypertension, patients with an unrepaired ASD, VSD and PDA are usually asymptomatic with exercise\textsuperscript{59}.

1.11.6. Psychosocial Issues.

When an infant is born with a heart defect, the parents may grieve over the loss of the healthy newborn they had anticipated and experience shock, denial, guilt, anger, despair or confusion on learning that their infant has a cardiac defect. Some parents may be unable at first to respond to their newborn. Even greater stress may occur if the condition is one that requires surgical intervention. The psychosocial stresses faced by the parents of a child with (CHD), should be considered thoroughly by the health care team. The family or parents, as the primary caregiver, are
ultimately responsible for the child care and treatment\(^{(60)}\). Illness impacted the whole family and parents reported marital and sibling problems\(^{(61)}\).

Many factors influence how a child feels about having (CHD), and how it affects him/her mentally and emotionally, including:

- **The type of defect.**
  Different emotions may be experienced with (CHD) that require one operation to repair versus that requires many operations and periodic or constant medical care.

- **The age of the child when the heart defect was diagnosed.**
  A child who was diagnosed at birth and who has grown up with heart defect may adjust differently than a child who learns of his/her heart disease at an older age, after living a seemingly healthy life.

- **The number of hospitalizations.**
  The children who require many diagnostic tests and procedures, surgeries and/or other hospitalizations due to the nature of their illness may feel angry, fearful, resentful or withdrawn.

- **The age of the child.**
  Younger children may have difficulty understanding their illness and may misinterpret the reasons for tests and surgical procedures. Whereas, older
children can better understand information about their illness and what it will take to make them well.

- **The coping skills and temperament of the child.**
  Some children can deal with adversity better than others, and some children are more nervous and anxious than others.

- **Body image.**
  Surgical scar, cyanosis or the need for medical therapies such as oxygen or feeding tubes often make a child feels different from others, and can affect self-esteem and body-image.

- **Family dynamics.**
  A child’s emotions can be affected by the way his/her family members cope with the illness, as well as other issues including the stress felt by the family. Finances, work and insurance problems the family may face or siblings who are jealous from the extra attention the child with (CHD) may receive due to his/her illness, will all affect the child’s emotions.

  Reinforcement of the positive, normal attributes of the child helps the family see the child as an individual with many of the same needs as healthy children. Consultation with a mental health professional may enable the family to recognize and build on strengths that will help them cope with this challenge and circumvent those things that might interfere with their coping ability. Parental support groups
play an important role and are helpful to parents as (CHD) affects not only patients, but entire families\textsuperscript{(62)}. Recognizing and building on the family's strengths and on the child's positive attributes lay the groundwork for normalization. When this process occurs, the family focuses on the child rather than on the condition. Reinforcing the family's successes help them to build up the confidence and the desire to address and manage future issues\textsuperscript{(60)}.

1.12. PARENTAL KNOWLEDGE, ATTITUDES, AND PRACTICE TOWARDS THEIR CHILDREN'S CONGENITAL HEART DISEASE.

Accurate understanding of chronic illness in patients of any age is associated with less distress, less confusion, improved satisfaction with medical care, better compliance with treatment and an improved emotional state, all key factors for good health related quality of life\textsuperscript{(63)}. The knowledge of one’s own disease is an important determinant of health related behaviour\textsuperscript{(64)}. Much effort has therefore been devoted to educating patients with chronic diseases in an attempt to change their health behaviour and maximise their health benefits\textsuperscript{(65)}. For children with congenital heart disease, parents’ knowledge about their children’s heart disease, treatment and prevention of complications may likewise promote a better health related behaviour.
in their children by increasing the understanding of the cardiac problem, improving compliance with treatment, and avoiding risk taking behaviour.

In the past decade, significant advances in transcatheter and surgical management of paediatric cardiac patients have been made. Nonetheless, several recent studies have shown that understanding of illness by children, adolescents and adults with (CHD) remains unsatisfactory\(^{66,67}\). The paediatric cardiac patients may have an entirely wrong concept of their disease\(^{68,69}\), while the majority of adult patients have a poor understanding of symptoms suggestive of cardiac deterioration, risk factors of endocarditis, the impact of smoking and alcohol on heart disease, and the hereditary nature of their cardiac conditions\(^{67,70}\). Given the important role of parents in imparting knowledge of chronic illness to their child, the disappointing findings from previous studies may perhaps reflect inadequacies of parental knowledge.

In China, a study was conducted by Cheuk DK, Wong SM, Choi YP and Cheung YF in Grantham hospital to assess parents’ of 156 children with (CHD), understanding of their child's (CHD) and to identify significant determinants of parental knowledge. The conclusion was that parents of children with (CHD) have important knowledge gaps, and suggested that the current educational program was inadequate and needs to be refined. This is to promote better parental understanding
of their child’s heart disease, with the ultimate aim of enabling parents to impart such knowledge accurately to their children\(^{(71)}\).

In Portugal a study was conducted by Barreira JL, Baptista MJ, Moreira A, Azevedo A and Areias JC to assess the level of understanding among caregivers of children with (CHD) of their disease, risks of BE and attitude concerning oral health and antibiotics prophylaxis of BE. The result of this survey supported the need to reinforce information about risks of IE and prophylaxis recommendation among caregivers of children with CHD\(^{(72)}\).

Bulat DC and Kantoch MJ, University of Alberta, Edmonton, assessed parental knowledge regarding their children (CHD), risk of BE and requirement for BEP and came to a conclusion that many parents are not familiar with their children’s heart disease and do not understand the risks of BE or the need for BEP\(^{(73)}\).

In Granada a study was done by Perez piaya Moreno MR, de Diego Fernandez P, Chinchilla Molina JM, de Haro Lopez MA, Sanchez Calderon M, Rodriguez Vazquez de Rey MM, et al to assess the knowledge that parents of children with congenital cardiac disease have about BE and its prophylaxis. They came to a conclusion that they emphasize the need to reinforce the information about BEP that the cardiologists, pediatricians and general physicians give to the families in order to prevent the morbidity and mortality caused by BE\(^{(74)}\).
Cetta F, Bell TJ, Podlecki DD, Ros Sp, Loyola University Medical Center, Maywood, Illinois: conducted a study to determine parental knowledge of bacterial endocarditis prophylaxis. They came to a conclusion that while most parents know the nature of their children's heart lesion and current medications, parental knowledge of endocarditis and BEP was limited, and intensified education and awareness programs are needed in order to prevent potential major morbidity and mortality for pediatric patients with heart disease\(^{(75)}\).

In UK, a study was conducted by Saunders CP, Robert GJ in Great Ormond Street Hospital for children to investigate the dental knowledge, attitudes and dental health practices of families of children with congenital heart disease. It concluded that parents need more active encouragement to ensure that their children receive adequate preventive advice about dental health and the availability of dental treatment\(^{(76)}\).

Andresova S, Hamalova J, Vyhnalek J, in Czechoslovakia investigated training attitudes of 18 mothers of children aged 6-14 years operated during preschool age for congenital cardiac disease. All mothers preferred democratic training with stimulation of the children's activities. They refused authoritative and restricting attitudes. Mothers did not display excessively centered relations to the child, nor exaggerated protectiveness\(^{(77)}\).
Beeri M, Haramati Z, Rein JJ, Nir A, in Hadassah University Hospital, Mount Scopus, conducted a study to assess parental knowledge and attitudes among outpatients at a hospital pediatric cardiology clinic. They reached to a conclusion that ignorance of their child's problem did not correlate with its severity or complexity but rather with parental background: the less educated the parent, the more likely was the problem perceived incorrectly. An additional finding was that (68%) of the families turn to non-medical personnel for medical advice—an interesting finding not hitherto addressed(78).

In Kingdom of Saudi Arabia, King Khaled University Hospital, a study was conducted by Al-Jarallah AS, Lardhi AA and Hassan AK to determine the parental knowledge of bacterial endocarditis prophylaxis (BEP). It concluded that while most parents know the nature of their child's heart lesion and current medication, parental knowledge of endocarditis and it's prophylaxis was limited. The study suggested intensified education programs in order to prevent potential major morbidity and mortality for paediatric patient with CHD(79).

Noll S, Spitz L, Pierro A, University College London, UK: conducted a study to (1) characterize the source of additional medical information acquired by parents and (2) to verify how much information is correctly interpreted and remembered correctly. The conclusion was that parents commonly obtain additional medical information, however this information does not necessarily improve parents'
understanding of the child's operative risk and long-term problems. In addition, parents' perception of having adequate medical information often is incorrect\(^{(80)}\).

Efforts to provide accurate individualised information for parents and children need to be intensified and the efficacy of different mechanisms for delivering such information audited. Enhanced understanding of the illness and its treatment has implications not only for the physical wellbeing of these patients but also for their psychological welfare, quality of life and their adherence to the prescribed treatment protocols.

1.13. EFFECTS OF CONGENITAL HEART DISEASE ON GROWTH.

Growth in children with (CHD) is often compromised. Cardiac malformations are undoubtedly responsible for malnutrition, which may range from mild undernutrition to severe failure to thrive (FTT). Growth impairment of children with acyanotic (CHD) is directly proportional to the severity of hemodynamic disturbance\(^{(55)}\). The most severely affected infants are those with congestive heart failure\(^{(55)}\). Acyanotic lesions tend to jeopardize weight gain rather than height, Salzer and colleagues showed that infants with left to right shunt tended to gain less weight and to be leaner than those with cyanotic heart disease\(^{(81)}\). Whereas, cyanotic lesions tend to affect both height and weight\(^{(82)}\). Linde and colleagues, found a more pronounced retardation in both height and weight in children with cyanosis than in those with acyanotic heart disease\(^{(83)}\). Infants with cyanotic lesions have an earlier
fall-off in linear growth and often are smaller than infants without this problem\textsuperscript{(55)}. Children with (CHD) and growth impairment typically show caloric deprivation and a reduction in adipose stores. Boys are usually more malnourished than girls\textsuperscript{(55,82)}.

Anorexia and early satiety may be exacerbated by the drugs, such as diuretics\textsuperscript{(82)}. However, in the absence of CHF, a large left-to-right shunt or cyanosis, other causes of failure to thrive should be explored\textsuperscript{(82)}.

Many factors contribute to growth impairment in children with (CHD), including a lower birth weight, increased caloric requirements as a result of increased respiratory rate accompanying CHF, and the presence of concomitant musculoskeletal, central nervous system, renal or gastrointestinal malformations\textsuperscript{(55)}. Mild gastrointestinal abnormalities, mild steatorrhea and excess protein loss are common in infants with CHD\textsuperscript{(55)}. However, inadequate caloric intake appears to be the most important cause of growth failure in CHD\textsuperscript{(84,85)}. A characteristic feeding pattern of children with (CHD) is defined, with a large variation in caloric intake\textsuperscript{(84)}. When heart failure is mild the infant commonly overfeeds, and fluid and sodium overload disturb cardiac haemodynamics, leading to decompensation of heart failure and decreased intake\textsuperscript{(84)}.

Poor nutrition related to anorexia, fatigability, vomiting, fluid restriction and frequent respiratory infection also contributes to growth impairment\textsuperscript{(55)}. Chronic hypoxia may contribute to the feeding problem in cardiac patients and affect growth,
as it is an important factor in anorexia and inefficient processing of nutrients at the cellular level\(^{(83,85)}\). Malabsorption is also thought to play a role in cardiac cachexia, it can result from both congestive heart failure and oxygen lack\(^{(86,87)}\). Hypertrophic cardiac muscle can use up to (30%) of the total oxygen consumption of the body rather than the usual ten percent\(^{(88)}\). After accounting for other energy needs, infants with (CHD) may have only one half as much energy available for growth as healthy infants\(^{(55)}\). Children with heart disease may need as much as (50%) more calories than normal children in order to achieve normal growth\(^{(84)}\).

Delay in surgical repair of congenital heart lesions can lead to worsening of nutrition and growth status of patients. Several reports have documented encouraging results of early repair of critical congenital heart defects in symptomatic neonates and infants rather than palliative operations, and of primary surgical closure of large ventricular septal defects\(^{(89,90,91)}\). Thus, a more intensive nutritional treatment and early corrective surgery should be considered to optimise the outcome.

The effects of (CHD) on growth have been assessed in several studies. Some studies also have been done to show the effect of enteral feeding in infants with (CHD). One of the oldest of these studies was done by Salzer HR and colleagues, University of Vienna, Austria in 1989: to assess the relation between growth and nutritional intake of infants with (CHD), and came to a conclusion that a low level of food intake was not the main cause of inadequate growth of infants with CHD\(^{(81)}\).
In USA a study was conducted by Cameron JW and colleagues to determine the prevalence of malnutrition among hospitalized children with congenital heart disease by age, disease process, and clinical status. It concluded that malnutrition in hospitalized children with congenital heart disease remains common, highlighting the importance of nutritional screening and intervention\(^{(92)}\).

Unger R, Dekleermaeker M, Gidding SS and Christoffel KK, Northwestern University Medical School, Chicago: assessed the nutritional status of patients with (CHD) to evaluate the role of dietary intake in impaired weight for patient length. Underweight patients with (CHD) underwent nutritional counseling to evaluate the role of this intervention in improvement of weight for length. The study concluded that nutritional evaluation of patients with (CHD) demonstrated that underweight children had inadequate diets. Underweight patients with (CHD) who received nutritional counseling showed increased dietary intake and improved anthropometric measurement on follow up\(^{(93)}\).

In New York Medical College, Schwarz SM, Gewitz MH, See CC, Berazin S, Glassman MS, Medow CM, et al conducted a study to determine an effective nutritional regimen for management of growth failure in infants with congenital heart disease and CHF. Patients were randomly assigned to one of three feeding groups: group 1 received continuous, 24-hour nasogastric alimentation; group 2 received overnight, 12-hour nasogastric infusions plus daytime oral feedings as
tolerated; and group 3 received oral feedings alone. For all patients, commercial infant formula (cow's milk or soy protein) was supplemented to a calorie density of approximately 1 kcal/mL. Serial anthropometric measurements demonstrated that only 24-hour infusions (group 1) were associated with significantly improved nutritional status. The study suggested that infants with congenital cardiac defects complicated by malnutrition manifest increased nutrient requirements for growth and weight gain. Continuous, 24-hour, nasogastric alimentation is a safe and effective method for achieving both increased nutrient intake and improved overall nutritional status in these infants\(^{(54)}\).

In Brazil a study was conducted by Leite HP, de Camargo Carvalho AC, Fisberg M to assess the nutritional status of children with congenital heart disease with left-to-right shunt and the nutritional disturbances related to the presence of pulmonary hypertension (PH). The study concluded that the prevalence of malnutrition was high. The presence of PH was associated with higher nutritional disturbance, and that the nutritional assessment may be a good way to identify diagnostic groups at particular risk of failure to thrive. This can be useful in planning a management which ensures these patients to achieve adequate nutritional supplementation in early life\(^{(94)}\).

In Turkey a study conducted by Varan B, Tokel K and Yilmaz G to investigate the effect of several types of (CHD) on nutrition and growth and came to a
conclusion that Patients with (CHD) are prone to malnutrition and growth failure. Pulmonary hypertension appears to be the most important factor, and cyanotic patients with pulmonary hypertension are the ones most severely affected. This study shows the additive effects of hypoxia and pulmonary hypertension on nutrition and growth of children with CHD\(^{95}\).

Venugopalan p, Akinbami FO, Al-Hinai KM, Agarwal AK, Sultan Qaboos University Hospital, Muscat, Oman: assessed the frequency of malnutrition in children with congenital heart defects in a hospital outpatient setting. The study concluded that malnutrition remains a problem among children with congenital heart defects, especially in those with either heart failure or cyanosis, and symptomatic infants are the worst affected. Greater attention is required in the dietary management, early diagnosis and intervention to restore normal growth\(^{96}\).

In Japan a study was done by Sasaki H, Baba K, Nishida Y, Waki K, Konishi N, Mawatari H et al to assess seven prepubertal short children with congenital heart disease were treated with recombinant human growth hormone (GH). Although complete surgical correction was performed for their heart disease at least 2 years before the start of GH therapy, improvement in growth was less than expected in these children. They received 0.5 IU/ kg / week of GH for 2 years or more. The growth rate increased from a mean of 4.3 cm / year before treatment to a mean of 7.8 cm / year in the first year and to a mean of 6.3 cm / year in the second year of
treatment. They came to a conclusion that recombinant GH increases the growth rate in children with congenital heart disease and prepubertal growth retardation\(^\text{(97)}\).
JUSTIFICATION AND OBJECTIVES

Justification.

- Congenital heart disease is a chronic problem with serious complications that can impose psychological, social and financial stresses on the family and affect the growth of their children.
- Knowledge about congenital heart disease, its treatment and risk factors for complications may optimize treatment and reduce incidence of complications.
- Identifying the determinants of parental knowledge can help us to design a program for teaching parents of children with congenital heart disease.
- No similar study has been performed in Sudan before.
Objectives.

- To assess the parents’ knowledge, attitudes and practice towards their children’s congenital heart disease, its management and prevention of its complications.
- To identify determinants of parental knowledge about their children’s congenital heart disease.
- To assess the impact of parental knowledge on the growth of their children.
CHAPTER TWO

PATIENTS
AND
METHODS
CHAPTER TWO

2. PATIENTS AND METHODS

2.1. Nature of the study:

This is a hospital based, cross sectional questionnaire survey.

2.2. Study area:

The study was carried out at Sudan Heart Centre (SHC), a tertiary referral centre for adults and children with heart disease. There are three paediatric cardiology clinics per week. Two clinics are run by one paediatric cardiologist who evaluated the patients in this study and the third clinic run by another paediatric cardiologist. These clinics evaluated 60 patients per week. There is one assistant paediatric cardiologist and two junior staffs. The paediatric cardiologists evaluate the patients clinically and by ECHO and explain to the parents their children’s heart disease and its implications in a lay term aided by a simple diagram.

2.3. The duration of the study:

The data was collected in the period between April and July 2005.

2.4. Study population:

The study population includes children who were accompanying their parents to the outpatient clinic of the tertiary cardiac centre in Khartoum and their parents.
2.5. **Inclusion criteria:**

All children with congenital heart disease, reporting to the centre whose age is less than 15 years.

2.6. **Exclusion criteria:**

- Children with associated syndromes or major other systemic disease such as Down's syndrome, gastrointestinal deficit and any severe abnormalities of the central nervous system.
- Children with a new diagnosis of heart disease.
- Parents and children who refuse to get involved in the study or to comply with its requirement.

2.7. **Sample size:**

The sample size was calculated according to the equation:

\[ N = \frac{Z^2PQ}{d^2} \]

where:
- \( N \) = sample size
- \( Z \) = statistical certainty 1.96 (at 95% of confidence)
- \( P \) = probability problem under study (as fraction of 1) 0.8
- \( Q \) = 1.0 – \( P \)
- \( d \) = desired margin of error

The sample size calculated was 100 children.
2.8. Ethical approval:

- Approval consent of the study was taken from our local committee of Paediatric and Child Health, University of Khartoum.
- Informed consent was taken from cardiac centre and treating doctors.
- Verbal informed consent was obtained from the parents of children with (CHD) after explaining the objectives of the study.

2.9. Methods:

The parents were informed about the purpose of the study; consent was taken and personally interviewed by the author with the help of a pre-structured questionnaire.

2.9.1. Questionnaire:

The questionnaire sheet was designed to include:

- Personal data of the patients such as name, age, tribe and residence. It also provided the demographic data of the parents including age, educational level, occupation, consanguinity, income and socioeconomic status.
- From the medical records, the following data was collected: cardiac diagnosis, family history of congenital heart disease, previous cardiac
operations or cardiac catheterizations, current medication, and history of infective endocarditis.

- The parental knowledge was assessed under three domains: the nature of the heart disease, its treatment and prognosis; the impact of heart lesions on exercise capacity and infective endocarditis and its prevention.

- The parental attitudes were assessed by asking the parents to express their feeling towards their children illness and caring for their children.

- The parental practice of dental health and nutritional considerations was assessed.

- A diagram of the normal heart showing the four cardiac chambers and the normal connection of the great arteries was shown to parents, who were then asked to draw or indicate the heart defect or defects on the diagram.

- The questionnaire sheet also included findings of thorough physical examination, including anthropometric measurement (weight, height, head circumference). The growth parameters were plotted on standardized growth charts. Children with growth parameters below the third percentiles for their age were categorized as failing to thrive\(^9\). Ideal body weight for length was also estimated. Malnutrition was
described as mild, moderate or severe when the patient weight was between 80-90%, 70-80% or less than 70% of ideal weight for length respectively. The impact of parental knowledge on the growth was assessed.

2.10. **Data entry and statistical methods:**

The data was analyzed using statistical package of social sciences (SPSS). Data was expressed as percentage. Descriptive statistics were performed. Univariate analysis to assess for association between demographic and clinical variables with selected items of knowledge and the association between growth and parental knowledge was performed with the Chi-square test, Fisher's exact test. Multivariate analysis by logistic regression was used to identify significant determinants of selected items of knowledge. The dependent knowledge variables were dichotomized as a correct or incorrect answer. Incorrect answers were further deemed as incomplete, incorrect or don't know answers. A probability value of $p < 0.05$ was considered significant.

A scoring system was also used to assess the general knowledge of parents of (CHD) children. Using scores of one or zero for correct and incorrect responses respectively. The total of responses were analyzed and a cut-off point of (60%) was used to divide the responses to good or poor knowledge. Scores more than (60%)
were considered as good knowledge and scores equal to or below (60%) were considered as poor knowledge.

2.11. Difficulties encountered:

- Refusal of parents to participate in the study.
- Insufficient data on outpatient medical records.

2.12. Input of the author:

The input of the author was to:

1. Design the study and questionnaire.
2. Make necessary contact and permission.
3. Interview the parents and fill the questionnaire.
4. Perform a thorough physical examination including anthropometric measurements, and plotting the growth parameters on standardized growth charts.
CHAPTER THREE

RESULTS
CHAPTER THREE

3. RESULTS

A total of 100 children with (CHD) were enrolled in this study. Their parents were interviewed using the questionnaire and data obtained.

3.1. SOCIO-DEMOGRAPHIC CHARACTERISTICS OF THE STUDY GROUP AND THEIR PARENTS.

3.1.1. Age and gender distribution of the study group.

The age of the patients in the study group ranged between 6 months to 15 years. Children below one year of age were 18 children (18%), between one to less than five years were 42 children (42%), between 5 to less than 10 years were 24 children (24%) and 16 children (16%) were in the age group 10-15 years. Females constituted 53 patients (53%) while 47 patients were males (47%). Female to male ratio was 1.13:1, (Figure 1, 2).

3.1.2. Tribe distribution of the study group.

The majority of the children in the study belonged to Arab tribes and they were 72 patients who constituted 72%, Kurdofanian and Darforian descents were 12 patients (12%), Nubian constituted 9 patients (9%), 4 patients (4%) were Bija, 2 patients (2%) were from the ethnic group Nuba and only one patient (1%) belonged to equatorial tribes, (Figure 3).
Fig (1) Age distribution of the 100 patients with Congenital Heart Disease.
Fig (2) Gender distribution in the 100 patients with Congnital Heat Disease.

- Female: 47%
- Male: 53%
Fig (3) Ethnic distribution of the children in the study group.
3.1.3. Residence distribution of the study group.

The majority of the patients in the study group were from urban areas and they were 66 patients (66%), 21 patients (21%) were from rural areas and the remaining 13 patients (13%) were from suburban areas, (Figure 4).

3.1.4. Parental age distribution of the study group.

Paternal age was less than 30 years in five of the fathers (5%), between 30-40 years in 33 of the fathers (33%) and more than 40 years in 62 of the fathers (62%). Regarding maternal age, 10 of the mothers (10%) were less than 25 years, 50 of the mothers (50%) were between 25-35 years, 37 of the mothers (37%) were more than 35 years and three of the mothers were dead (03%) as shown in (Table 1, 2).

3.1.5. Parents’ educational level.

Regarding paternal educational level, seven of the fathers (07%) were illiterate, six received khalwa (06%), 34 completed their primary school (34%), 38 had their secondary school (38%) while 15 were university graduate (15%). On the other hand, 14 of the mothers (14%) were illiterate, eight received khalwa (08%), 32 of the mothers (32%) had their primary school and another 32 of the mothers (32%) had their secondary school, 11 were university graduate (11%) and three of the mothers were dead (03%) as shown in (Figure 5).
Fig (4) Residence distribution of the study group.
Table (1) Paternal age of the 100 patients with Congenital Heart Disease.

<table>
<thead>
<tr>
<th>Paternal age In years</th>
<th>No</th>
<th>Percentage %</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 30</td>
<td>05</td>
<td>05 %</td>
</tr>
<tr>
<td>30-40</td>
<td>33</td>
<td>33 %</td>
</tr>
<tr>
<td>&gt; 40</td>
<td>62</td>
<td>62 %</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>100</strong></td>
<td><strong>100%</strong></td>
</tr>
</tbody>
</table>
Table (2) Maternal age of the 100 patients with Congenital Heart Disease (3 mothers are dead).

<table>
<thead>
<tr>
<th>Maternal age in years</th>
<th>No</th>
<th>Percentage %</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 25</td>
<td>10</td>
<td>10 %</td>
</tr>
<tr>
<td>25-35</td>
<td>50</td>
<td>50 %</td>
</tr>
<tr>
<td>&gt; 35</td>
<td>37</td>
<td>37 %</td>
</tr>
</tbody>
</table>
Fig (5) Parental educational level of the 100 patients with Congenital Heart Disease.

Educational level:
- Illiterate
- Khalwa
- Primary
- Secondary
- University

Parents:
- Fathers
- Mothers

- Illiterate: Fathers 7%, Mothers 6%
- Khalwa: Fathers 8%, Mothers 6%
- Primary: Fathers 34%, Mothers 32%
- Secondary: Fathers 38%, Mothers 32%
- University: Fathers 15%, Mothers 11%
3.1.6. Parents' occupation.

Ten of the fathers (10%) were professionals, 11 were businessmen (11%), 21 were small scaled businessmen (21%), 19 were employees (19%), 13 were skilled laborers (13%), 16 were unskilled laborers (16%) and 10 of the fathers were unemployed (10%). The majority of the mothers were housewives and they were 88 mothers (88%), only one mother (01%) was a professional, while eight were employees (08%) and three of the mothers were dead (03%) as shown in (Figures 6, 7).

3.1.7. Degree of consanguinity of the parents of the study group.

Consanguinity between parents was found in fifty six of the Study group (56%). First degree cousins constituted thirty six (64.3%) couples, six were second degree cousins (10.7%) and 14 of the parents (25%) were far cousins, (Figure 8).

3.1.8. Socio-economic status of the children with CHD.

According to socio-economic status; nearly half of the patients (51%) were of moderate social class, while 35 of the patients (35%) were of low social class and only fourteen (14%) were of high social class, (Figure 9).
Fig (6) Paternal occupation of the 100 patients with Congenital Heart Disease.

- Professional: 10%
- Businessmen: 11%
- Small scaled businessmen: 21%
- Employee: 19%
- Skilled laborer: 16%
- Unskilled laborer: 13%
- Unemployed: 10%
Fig (7) Maternal occupation of the 100 patients with Congenital Heart Disease (3 mothers are dead)

- Housewife: 88%
- Professional: 1%
- Employee: 8%
Fig (8) Degree of consanguinity of the parents of 56 patients with Congenital Heart Disease.

- First degree cousins: 64.30%
- Second degree cousins: 10.70%
- Far cousins: 25%
Fig (9) Socio-economic status of the 100 children in the study group.

35% High
14% Moderate
51% Low
3.2. HISTORY AND CLINICAL DATA OF THE PATIENTS.

3.2.1. Diagnosis.

The study showed that 59 of the patients (59%) had acyanotic (CHD) while 41 of the patients (41%) had cyanotic (CHD). The frequency of the lesions showed (VSD) as the commonest lesion; it was encountered in 22 patients (22%) as isolated lesion. Tetralogy of Fallot was diagnosed in 18 patients (18%) while (PDA) in 15 patients (15%) and isolated (ASD) in eight patients (08%). Double outlet right ventricle was diagnosed in seven patients (07%) and (PS) in five patients (05%) while (D-TGA+VSD) was diagnosed in four patients (04%). Ebstein's anomaly was encountered in three patients (03%). Two patients (02%) were diagnosed with each of the following lesions (AVSD, ASD+VSD, ASD+PS and TA+PS+VSD+TGA). Transposition of the great arteries was encountered in only one patient (01%). Similarly only one patient (01%) was diagnosed with each of the following lesions (VSD+MVP, L-TGA, ASD+PAPVD), unbalanced (AVSD), truncus arteriosus and (TA+VSD, DILV, TGA+VSD+PS) as shown in (Table 3, 4).

3.2.2. Treatment offered:

3.2.2.1. Current medication:

Fifty seven of the patients (57%) were taking medical treatment for their problems.
Table (3) Cardiac diagnosis of 59 children with Acyanotic Congenital Heart Disease (Total 100 children).

<table>
<thead>
<tr>
<th>Cardiac diagnosis</th>
<th>No</th>
<th>Percentage %</th>
</tr>
</thead>
<tbody>
<tr>
<td>VSD</td>
<td>22</td>
<td>22%</td>
</tr>
<tr>
<td>PDA</td>
<td>15</td>
<td>15%</td>
</tr>
<tr>
<td>ASD</td>
<td>08</td>
<td>08%</td>
</tr>
<tr>
<td>PS</td>
<td>05</td>
<td>05%</td>
</tr>
<tr>
<td>AVSD</td>
<td>02</td>
<td>02%</td>
</tr>
<tr>
<td>ASD +VSD</td>
<td>02</td>
<td>02%</td>
</tr>
<tr>
<td>ASD +PS</td>
<td>02</td>
<td>02%</td>
</tr>
<tr>
<td>VSD + MVP</td>
<td>01</td>
<td>01%</td>
</tr>
<tr>
<td>ASD + PAPVD</td>
<td>01</td>
<td>01%</td>
</tr>
<tr>
<td>Unbalanced AVSD</td>
<td>01</td>
<td>01%</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>59</td>
<td>59%</td>
</tr>
</tbody>
</table>
Table (4) Cardiac diagnosis of 41 children with Cyanotic Congenital Heart Disease (Total 100 children).

<table>
<thead>
<tr>
<th>Cardiac diagnosis</th>
<th>No</th>
<th>Percentage %</th>
</tr>
</thead>
<tbody>
<tr>
<td>TOF</td>
<td>18</td>
<td>18 %</td>
</tr>
<tr>
<td>DORV</td>
<td>07</td>
<td>07 %</td>
</tr>
<tr>
<td>D-TGA +VSD</td>
<td>04</td>
<td>04 %</td>
</tr>
<tr>
<td>Ebstein's anomaly</td>
<td>03</td>
<td>03 %</td>
</tr>
<tr>
<td>TA + PS + VSD +TGA</td>
<td>02</td>
<td>02 %</td>
</tr>
<tr>
<td>TGA</td>
<td>01</td>
<td>01 %</td>
</tr>
<tr>
<td>L-TGA</td>
<td>01</td>
<td>01 %</td>
</tr>
<tr>
<td>TA + VSD</td>
<td>01</td>
<td>01 %</td>
</tr>
<tr>
<td>DILV</td>
<td>01</td>
<td>01 %</td>
</tr>
<tr>
<td>TGA + VSD + PS</td>
<td>01</td>
<td>01 %</td>
</tr>
<tr>
<td>TGA + PS</td>
<td>01</td>
<td>01 %</td>
</tr>
<tr>
<td>Truncus arteriosus</td>
<td>01</td>
<td>01 %</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>41</strong></td>
<td><strong>41 %</strong></td>
</tr>
</tbody>
</table>
3.2.2. Previous catheterization or surgery.

Of the 100 patients, twenty (20%) had undergone previous catheterization of whom twelve (12%) it was diagnostic and for eight (08%) it was therapeutic and twenty two of the patients (22%) had undergone previous surgery, (Table 5).

3.2.3. Family history of (CHD).

Positive family history of (CHD) was found in 18 of the children of the study group (18%); five were siblings (27.8 %), 12 of them (66.7%) were cousins and one was a paternal uncle (05.5 %) as shown in (Table 6).

3.2.4. Past history of infective endocarditis.

Only one patient had a documented episode of infective endocarditis

3.2.5. Past history of heart failure.

Past history of heart failure was found in 19 of the patients (19%).

3.3. ANTHROPOMETRIC MEASUREMENT.

3.3.1. Weight.

Fifty five patients of the study group their weight was below the 3rd percentile for age (55%), between 3rd-50th percentile were thirty seven patients (37%) and only eight of them (08%) were between 50th-97th percentile for age. Regarding ideal body weight (IBW %), 12 of the patients (12%) were below 70% of their ideal body weight for height (severe malnutrition). While 28 of the
Table (5) Treatment offered to the 100 patients with Congenital Heart Disease.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>No</th>
<th>Percentage %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medications</td>
<td>57</td>
<td>57 %</td>
</tr>
<tr>
<td>Catheterization:</td>
<td>(20)</td>
<td>(20 %)</td>
</tr>
<tr>
<td>- Diagnostic</td>
<td>12</td>
<td>12 %</td>
</tr>
<tr>
<td>- Therapeutic</td>
<td>08</td>
<td>08 %</td>
</tr>
<tr>
<td>Surgery</td>
<td>22</td>
<td>22 %</td>
</tr>
<tr>
<td>No medication</td>
<td>01</td>
<td>01 %</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>100</strong></td>
<td><strong>100 %</strong></td>
</tr>
</tbody>
</table>

* Total number of catheterization
Table (6) Distribution of family history of (CHD) in 18 patients with Congenital Heart Disease.

<table>
<thead>
<tr>
<th>The relation to Parents</th>
<th>No</th>
<th>Percentage %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sibling</td>
<td>05</td>
<td>27.8 %</td>
</tr>
<tr>
<td>Cousin</td>
<td>12</td>
<td>66.7 %</td>
</tr>
<tr>
<td>Paternal uncle</td>
<td>01</td>
<td>05.5 %</td>
</tr>
<tr>
<td>Total</td>
<td>18</td>
<td>100 %</td>
</tr>
</tbody>
</table>
patients (28%) were between 70-80% of their IBW (moderate malnutrition), 24 of the patients (24%) were between 80-90% of their IBW (mild malnutrition) and 36 of the patients (36%) were between 90-110% of their IBW (normal) as shown in (Tables 7,8).

3.3.2. Height.

Thirty six of the patients (36%) their height was below the 3rd percentile for age, 51 patients (51%) were between the 3rd-50th percentile and only 13 patients (13%) were between 50th-97th percentile for age. The patients with both weight and height below the 3rd percentile were 31 of the patients (31%) and were described as failing to thrive, (Table 7).

3.3.3. Head circumference.

Head circumference below the 3rd percentile was found in ten of the patients (10%), 62 of the patients (62%) were between 3rd-50th percentile, between 50th-97th percentile were twenty seven patients (27%) and only one patient was more than the 97th percentile, (Table 7).

3.4. PARENTAL KNOWLEDGE.

While (88%) of the parents mentioned that their doctor gave them full explanation of their child’s illness, (64%) of them felt they had received enough information and (59%) thought that they well understood the condition.
Table (7) Anthropometric measurement of the 100 patients with Congenital Heart Disease.

<table>
<thead>
<tr>
<th>Growth parameter</th>
<th>No</th>
<th>Percentage%</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Weight percentile</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 3&lt;sup&gt;rd&lt;/sup&gt; percentile</td>
<td>55</td>
<td>55 %</td>
</tr>
<tr>
<td>3&lt;sup&gt;rd&lt;/sup&gt;-50&lt;sup&gt;th&lt;/sup&gt; percentile</td>
<td>37</td>
<td>37 %</td>
</tr>
<tr>
<td>50&lt;sup&gt;th&lt;/sup&gt;-97&lt;sup&gt;th&lt;/sup&gt; percentile</td>
<td>08</td>
<td>08 %</td>
</tr>
<tr>
<td>&gt; 97&lt;sup&gt;th&lt;/sup&gt; percentile</td>
<td>_</td>
<td>_</td>
</tr>
</tbody>
</table>

| **Height percentile**    |    |             |
| < 3<sup>rd</sup> percentile | 36 | 36 %        |
| 3<sup>rd</sup>-50<sup>th</sup> percentile | 51 | 51 %        |
| 50<sup>th</sup>-97<sup>th</sup> percentile | 13 | 13 %        |
| > 97<sup>th</sup> percentile | _  | _           |

| **Head circumference**   |    |             |
| < 3<sup>rd</sup> percentile | 10 | 10 %        |
| 3<sup>rd</sup>-50<sup>th</sup> percentile | 62 | 62 %        |
| 50<sup>th</sup>-97<sup>th</sup> percentile | 27 | 27 %        |
| > 97<sup>th</sup> percentile | 01 | 01 %        |
Table (8) Ideal body weight for height (IBW %) of the 100 patients with Congenital Heart Disease.

<table>
<thead>
<tr>
<th>IBW %</th>
<th>Degree of Nutritional Status</th>
<th>No</th>
<th>Percentage %</th>
</tr>
</thead>
<tbody>
<tr>
<td>90-110</td>
<td>Normal</td>
<td>36</td>
<td>36 %</td>
</tr>
<tr>
<td>80-90</td>
<td>Mild malnutrition</td>
<td>24</td>
<td>24 %</td>
</tr>
<tr>
<td>70-80</td>
<td>Moderate malnutrition</td>
<td>28</td>
<td>28 %</td>
</tr>
<tr>
<td>&lt; 70</td>
<td>Severe malnutrition</td>
<td>12</td>
<td>12 %</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td><strong>100</strong></td>
<td><strong>100 %</strong></td>
</tr>
</tbody>
</table>

Although 30 of the parents (30%) correctly named their child's (CHD), only twenty one of them (21%) correctly indicated the heart lesion or lesions on the diagram. Seventy three of the parents (73%) mentioned that their child disease was a congenital condition without knowing the possible aetiological causes. Five of the parents (05%) related it to urinary tract infection during pregnancy and another two of them (02%) related it to chest infection during early neonatal period. Another two of the parents (02%) mentioned that the possible cause is prematurity and another two of the parents (02%) related it to birth asphyxia. One parent (01%) related the cause to toxoplasmosis infection during pregnancy and another one parent (01%) related it to Rh incompatibility. Also another one parent (01%) mentioned that the only possible cause is witchcraft.

Ninety two (92%) of the parents correctly mentioned the symptoms attributable to the disease and for which they contact their cardiologist. Of the 57 parents whose children were taking cardiac medication at the time of the study, forty two of them (73.7%) were aware of the name, dose and schedule of the medication. Moreover, only twenty five of the parents (43.9%) and nine of them (15.8%) knew correctly the functions and the important side effects of the medications respectively. Nonetheless, parents were very knowledgeable about their children's past interventions, including surgical and catheter interventions. Nineteen of the 20
parents (95%) and 17 of the 22 parents (77.3%) were aware of the indication and aims of catheter and surgical interventions respectively. More than 90% were aware of the purpose and importance of follow up, and 71% were aware of the disease prognosis, (Tables 9, 10).

3.4.2. Impact of heart disease on exercise capacity.

Sixty five parents gave responses to this part of the questionnaire; twenty nine of the parents (44.6%) mentioned that they have received advices regarding safe exercise. Thirty seven of the parents (56.9%) answered correctly on the necessity for exercise restriction appropriate for their children's heart condition.

3.4.3. Infective endocarditis and its prevention.

Only one patient in this study had documented episode of infective endocarditis. Only twenty seven of all parents (27%) had heard of the term (IE). Fifteen of the 27 parents (55.6%) who had heard of infective endocarditis correctly stated the symptoms of (IE). Dental caries, the most well known risk factor of (IE) was correctly identified by 21 of the parents (77.8%). Skin infection, body cautery and poor nail and skin care were identified correctly as risk factors of (IE) by 19 of the parents (70.4%), 13 of the parents(48.1%) and 11 of the parents (40.7%) respectively, (Table11).
Table (9) Parental knowledge about the nature of heart disease.

<table>
<thead>
<tr>
<th>Items</th>
<th>Correct</th>
<th>Incomplete</th>
<th>Incorrect</th>
<th>Don't know</th>
</tr>
</thead>
<tbody>
<tr>
<td>Name</td>
<td>30 (30 %)</td>
<td>40 (40%)</td>
<td>13 (13 %)</td>
<td>17 (17 %)</td>
</tr>
<tr>
<td>Diagrammatic location</td>
<td>21 (21 %)</td>
<td>03 (03 %)</td>
<td>02 (02 %)</td>
<td>74 (74 %)</td>
</tr>
<tr>
<td>Aetiology</td>
<td>73 (73 %)</td>
<td>_____</td>
<td>14 (14 %)</td>
<td>13 (13 %)</td>
</tr>
<tr>
<td>Symptoms</td>
<td>92 (92 %)</td>
<td>01 (01 %)</td>
<td>07 (07 %)</td>
<td>_____</td>
</tr>
</tbody>
</table>
Table (10) Parental Knowledge about the treatment of heart disease.

<table>
<thead>
<tr>
<th>Items</th>
<th>Correct</th>
<th>Incomplete</th>
<th>Incorrect</th>
<th>Don’t know</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment offered</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medications (n=57)</td>
<td>42 (73.7 %)</td>
<td>07 (12.3 %)</td>
<td>02 (03.5 %)</td>
<td>06 (10.5 %)</td>
</tr>
<tr>
<td>Catheterization(n=20)</td>
<td>19 (95 %)</td>
<td>___</td>
<td>___</td>
<td>01 (05 %)</td>
</tr>
<tr>
<td>Surgery (n=22)</td>
<td>17 (77.3 %)</td>
<td>___</td>
<td>03 (13.6 %)</td>
<td>02 (09.1 %)</td>
</tr>
<tr>
<td>Side effects of medication</td>
<td>09 (15.8%)</td>
<td>01 (01.7%)</td>
<td>03 (05.3%)</td>
<td>44 (77.2%)</td>
</tr>
</tbody>
</table>
Table (11) Parental knowledge about the infective endocarditis and its prevention.

<table>
<thead>
<tr>
<th>Items</th>
<th>Correct</th>
<th>Incomplete</th>
<th>Incorrect</th>
<th>Don't know</th>
</tr>
</thead>
<tbody>
<tr>
<td>Awareness of IE</td>
<td>27 (27%)</td>
<td></td>
<td></td>
<td>73 (73%)</td>
</tr>
<tr>
<td>Symptoms of IE</td>
<td>15 (55.6%)</td>
<td>03 (11.1%)</td>
<td></td>
<td>09 (33.3%)</td>
</tr>
<tr>
<td>Risk factors of IE</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dental caries</td>
<td>21 (77.8%)</td>
<td></td>
<td></td>
<td>06 (22.2%)</td>
</tr>
<tr>
<td>Skin infection</td>
<td>19 (70.4%)</td>
<td></td>
<td></td>
<td>08 (29.6%)</td>
</tr>
<tr>
<td>Poor skin &amp; nail care</td>
<td>11 (40.7%)</td>
<td></td>
<td></td>
<td>16 (59.3%)</td>
</tr>
<tr>
<td>Body cautry</td>
<td>13 (48.1%)</td>
<td></td>
<td></td>
<td>14 (51.9%)</td>
</tr>
</tbody>
</table>
3.5. DETERMINANTS OF PARENTAL KNOWLEDGE.

Univariate analysis showed that:

3.5.1. Nature of the heart disease.

The ability to mention correctly the cardiac lesions was not related significantly with the type of cardiac defect; whether acyanotic or cyanotic one, 35.6% versus 21.9%, (P =0.122). Parents with educational attainment of university and secondary school level were more likely to specify the heart lesions correctly than those with primary school level or lower, 45.3% versus 15%, (P=0.002) for fathers, and 46.5 % versus 22.5 %, (P=0.00) for mothers. Also professional, businessmen and skilled workers were more likely to mention the heart lesion correctly than unskilled laborers and unemployed parents, 35.8% versus 21.3%, (P= 0.006) for fathers but insignificant for mothers (P= 0.628).

3.5.2. Diagrammatic location.

The ability to indicate the heart defect on the diagram was related to the cardiac diagnosis, that acyanotic cardiac lesions were indicated correctly more often than cyanotic ones, 32.2% versus 4.9%, (P=0.003). Parents' educational level was related to the ability to indicate the heart lesion or lesions on the diagram. The parents with educational attainment of university and secondary school level were more likely to indicate the lesion or lesions on the diagram correctly than those with primary school level or lower, 30.2% versus 12.5%, (P=0.011) for fathers, and 30.2% versus
17.5%, (P= 0.024) for mothers. The fathers' occupation was related to the ability to correctly indicate the heart lesion or lesions on the diagram, 28.3% versus 12.8%, (P= 0.008) for fathers, on the other hand mothers' occupation was not related (P=0.256).

3.5.3. **Aetiology of heart disease.**

Cardiac diagnosis whether acyanotic or cyanotic was not associated with the awareness of the possible aetiology of (CHD), 72.9% versus 73.2%, (P= 0.410). Fathers with high educational attainment were more likely to be correct regarding the cause of the congenital heart disease (P= 0.004), while mothers' education was not related (P= 0.229). Regarding parental occupation, it was not correlated for both fathers and mothers (P= 0.683) for fathers and (P= 0.256) for mothers with the possible aetiology of (CHD).

3.5.4. **Symptoms of heart disease.**

The ability to describe the cardiac symptoms was not related to the cardiac diagnosis (P= 0.479). Furthermore parents' education was not related to the ability to correctly describe cardiac symptoms (P= 0.070) for fathers and (P= 0.685) for mothers. Likewise parents' occupation was not related to the ability to correctly describe cardiac symptoms (P= 0.448) for fathers and (P= 0.992) for mothers.
3.5.5. Physical exercise capacity.

Parents whose children were older, mean age 9.7 versus 1.6 years, were more likely to give correct answers to the question on exercise guidelines regarding activity levels and sports participation (P=0.001).

3.5.6. Awareness of infective endocarditis.

Awareness of infective endocarditis was related to the fathers' educational attainment, 37.5% versus 17.5%, (P= 0.039), while it was not related to mothers' educational attainment (P= 0.083). Again fathers' occupation was related to awareness of (IE), 34% versus 19.1%, (P= 0.023), while mothers' occupation was not related (P=0.194).

Multivariate analysis by logistic regression showed that cardiac diagnosis, educational attainment and occupation of the parents were the main determinants of parents' knowledge of the nature of their child's heart disease. Acyanotic heart lesions were more likely than cyanotic lesions to be mentioned (P=0.021) and indicated on the diagram correctly (P=0.00). Parents with high educational attainment were likewise more likely to give correct answers when asked to mention the diagnosis (P=0.002), indicate on a diagram (P=0.037) and provide the possible causes of congenital heart disease (P=0.014). Professionals, skilled workers and employees were more likely to indicate on a diagram the heart lesion or lesions (P=0.022) and to give possible causes of congenital heart disease (P=0.009). No
significant determinants of knowledge regarding symptoms, the impact of heart
disease on exercise capacity and infective endocarditis were identified by logistic
regression, (Table 12).

3.6. PARENTAL ATTITUDES.

While 63 of the parents (63%) talked to their doctor regarding advices to cope
with their children, 37 of the parents (37%) did not discuss the problem with their
doctors.

3.6.1. Parental feeling towards their child's illness.

Fifty two of the parents (52%) were worried about their children's illness while
19 were depressed (19%). Those who were feeling sad were 15 of the parents (15%),
ten of the parents (10%) were anxious while only four of the parents (4%) felt guilty,
(Figure 10).

3.6.2. Caring and protection of the child with CHD.

The majority of the parents (93%) tend to give more attention and care to their
children with (CHD) comparing to their siblings, and all of them (100%) tend to
protect them from sources of infection as much as they could, (Figure 11).

3.6.3. Blaming oneself to have a child with CHD.

Nineteen of the parents (19%) blame themselves to have a child with congenital
heart disease. This feeling conveyed indirectly to the author during interview.
Table (12) Determinants of parental knowledge about nature, location on diagram and aetiology of the Congenital Heart Disease.

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Covariate</th>
<th>P value</th>
<th>R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nature:</td>
<td>Acyanotic V cyanotic</td>
<td>0.021</td>
<td>0.304</td>
</tr>
<tr>
<td></td>
<td>University or secondary V primary*</td>
<td>0.002</td>
<td></td>
</tr>
<tr>
<td>Location on diagram:</td>
<td>Acyanotic V cyanotic</td>
<td>0.000</td>
<td>0.334</td>
</tr>
<tr>
<td></td>
<td>University or secondary V primary*</td>
<td>0.037</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Professional or employee V unemployed</td>
<td>0.022</td>
<td></td>
</tr>
<tr>
<td>Aetiology:</td>
<td>University or secondary V primary</td>
<td>0.014</td>
<td>0.160</td>
</tr>
<tr>
<td></td>
<td>Professional or employee V housewives</td>
<td>0.009</td>
<td></td>
</tr>
</tbody>
</table>

* Mothers
Fig (10) Parental feeling towards their children with Congenital Heart Disease.
Fig (11) Parental attention and care for children with Congenital Heart Disease.

- Needs more care: 93%
- As his siblings: 7%

Legend:
- Needs more care
- As his siblings
3.7. PARENTAL PRACTICE.

3.7.1. Dental health.

Fifty three of the parents gave response to this part of the questionnaire. Regarding regular dental check up, only 10 of the parents (18.9%) visited the dentist regularly with their children for check up, while five of the parents (09.4%) knew that antibiotics had to be taken before the dental procedures. Fifty one of the children (96.2%) used to clean their teeth at least once a day, (Table 13).

3.7.2. Nutritional considerations.

Almost half of the parents (51%), asked their doctors for nutritional suggestions and advices. Regarding frequency of the feeding, the majority of the parents used to offer three main feeds and they were 59 of the parents (59%), while 38 of the parents (38%) offer small frequent feeding and only three of the parents (3%) offer feeding on demand, (Figure 12).

To maintain weight and growth 81 of the parents (81%) offered extra calories to their children. Seventy six of the parents (76%) offered home made high calories milk, 23 of the parents (23%) added special nutritional supplements to milk and 59 of the parents (59%) used to offer snacks that are rich in calories. However, on the other hand 59 of the parents (59%) avoided to give their children foods that have low calories, (Figure 13).
Table (13) Dental health practice in 53 patients with Congenital Heart Disease.

<table>
<thead>
<tr>
<th>Dental health practice</th>
<th>No</th>
<th>Percentage %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regular dental check up</td>
<td>10</td>
<td>18.9 %</td>
</tr>
<tr>
<td>Antibiotic before dental</td>
<td>05</td>
<td>09.4 %</td>
</tr>
<tr>
<td>Procedures</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brushing teeth at least</td>
<td>51</td>
<td>96.2 %</td>
</tr>
<tr>
<td>once a day</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Fig (13) Parental practice to increase the calories intake.

- Offer high calories milk: 76%
- Add special nutritional supplements to formula: 23%
- Offer nutritious food and snacks: 59%
- Avoid low calories foods: 59%
3.8. KNOWLEDGE SCORES.

The possible maximum score for the correct answers in knowledge oriented questions was 16, the attained scores by the parents ranged between 1-15. Thirty six of the parents (36%) had good knowledge, more than (60%) of the correct answers, while poor knowledge was found in 64 of the parents (64%) as shown in (Figure 14).

3.9. IMPACT OF PARENTAL KNOWLEDGE ON GROWTH.

Regarding the impact of parental knowledge on the growth of their children, no correlation was found between parental knowledge and improved growth. Regarding parents with good knowledge scores had 20 of their children (36.4%) having weight below 3rd percentile, 14 children (37.8%) were between the 3rd and 50th percentile and only two children (25%) were between 50th and 97th percentile. On the other hand parents with poor knowledge scores had 35 of their children (63.6%) having weight below the 3rd percentile, 23 children (62.2%) were between 3rd and 50th percentile and six children were between the 50th and 97th percentile. Regarding weight there was no statistically significant difference between the two groups (P=0.788). Likewise there was no correlation between the height of the patients and parental knowledge, as parents with good knowledge scores had 10 children (27.8%) with their height below the 3rd percentile, 23 children (45.1%) were between the 3rd and 50th percentile.
Fig (14) Parental Knowledge score.

- 64% Good knowledge
- 36% Poor knowledge
Only three children (23.1%) were between the 50th and 97th percentile. On the other hand parents with poor knowledge had 26 of their children (72.2%) having height below the 3rd percentile, 28 children (54.9%) were between the 3rd and 50th percentile and 10 children (76.9%) were between the 50th and the 97th percentile. Regarding height, again there was no statistically significant difference between the two groups (P=0.147). When considering the head circumference as growth parameter, the parents with good scores had four of their children (40%) below the 3rd percentile, 21 children (33.9%) were between the 3rd and 50th percentile and 11 children (40.7%) between the 50th and 97th percentile. On the other hand the parents with poor knowledge had six of their children (60%) below the 3rd percentile, 41 children (66.1%) were between the 3rd and 50th percentile, 16 children were (59.3%) were between the 50th and 97th percentile and only one child (100%) was above the 97th percentile. Although head circumference was the least to be affected again there was no statistically significant difference between the two groups (P=0.797).

Likewise, when considering ideal body weight for height, the parents with good knowledge scores had 15 of their children (41.7%) between 90-110% of their (IBW), 10 children (41.7%) were between 80-90%, seven children (25%) were between 70-80% and only four children (33.3%) were below 70%. On the other hand the parents with poor knowledge scores had 21 of their children (58.3%) between 90-110%, 14 children (58.3%) were between 80-90%, 21 children (75%)
were between 70-80% and eight children (66.7%) were below 70%. Again there was no correlation between (IBW) for height and parental knowledge (P=0.504) as shown in (Table 14).
Table (14) Relation of parental knowledge and growth parameters.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Good knowledge</th>
<th>Poor knowledge</th>
<th>X²</th>
<th>P. value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No ( % )</td>
<td>No ( % )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight percentile</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 3rd</td>
<td>20 (36.4%)</td>
<td>35 (63.6%)</td>
<td>0.478</td>
<td>0.788</td>
</tr>
<tr>
<td>3rd-50th</td>
<td>14 (37.8%)</td>
<td>23 (62.2%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>50th-97th</td>
<td>02 (25%)</td>
<td>06 (75%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height percentile</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 3rd</td>
<td>10 (27.8%)</td>
<td>26 (72.2%)</td>
<td>3.831</td>
<td>0.147</td>
</tr>
<tr>
<td>3rd-50th</td>
<td>23 (45.1%)</td>
<td>28 (54.9%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>50th-97th</td>
<td>03 (23.1%)</td>
<td>10 (76.9%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Head circumference</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percentile</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 3rd</td>
<td>04 (40%)</td>
<td>06 (60%)</td>
<td>1.017</td>
<td>0.797</td>
</tr>
<tr>
<td>3rd-50th</td>
<td>21 (33.9%)</td>
<td>41 (66.1%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>50th-97th</td>
<td>11 (40.7%)</td>
<td>16 (59.3%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 97th</td>
<td>_</td>
<td>01 (100%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IBW %</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>90-110</td>
<td>15 (41.7%)</td>
<td>21 (58.3%)</td>
<td>2.344</td>
<td>0.504</td>
</tr>
<tr>
<td>80-90</td>
<td>10 (41.7%)</td>
<td>14 (58.3%)</td>
<td></td>
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</tr>
<tr>
<td>70-80</td>
<td>07 (25%)</td>
<td>21 (75%)</td>
<td></td>
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</tr>
<tr>
<td>&lt; 70</td>
<td>04 (33.3%)</td>
<td>08 (66.7%)</td>
<td></td>
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</tbody>
</table>
CHAPTER FOUR

DISCUSSION
CHAPTER FOUR

4. DISCUSSION

This study was designed to assess parental knowledge, attitudes and practice towards their children's (CHD) and to study the impact of that on growth, which differs from most of the previous studies that focused on other aspects\(^{(71,72,75)}\).

4.1. SOCIO-DEMOGRAPHIC CHARACTERISTICS OF THE STUDY GROUP AND THEIR PARENTS.

4.1.1. Age and gender distribution of the study group.

The majority of patients (60\%) were less than 5 years old. The median age at the time of the study was 3 years, ranged between 6 months and 15 years. That was different from the findings of Cheuk study which reported a median age of 6 years, ranged between one month and 21 years\(^{(71)}\). This may reflect the early presentation and improvement in detection of patients with (CHD) in Sudan as it is consistent with the previous studies done in Sudan\(^{(23,26)}\). Regarding gender distribution the study showed slight increase in female preponderance. This is in contrast with findings in the previous study in Sudan and in the literature which showed equal distribution\(^{(26,71)}\).
4.1.2. Tribe distribution of the study group.

The study population truly represents different Sudanese ethnic groups and different geographical areas. There was predominance of Arab tribes (72%), followed by Nubian. As it was consistent with that reported by previous study done in Sudan, this could reflect the representation of these ethnic groups in the community\(^\text{(26)}\).

4.1.3. Residence distribution of the study group.

The majority of the study population came from urban areas. This was consistent with the fact that, urban population benefited most from health services.

4.1.4. Parental age distribution of the study group.

The majority of paternal ages (62%) were more than 40 years, while (50%) of maternal ages were between 25-35 years. This mostly reflects the normal pattern of parental age in our community.

4.1.5. Parental educational level.

Fifty three per cent of the fathers and 43% of the mothers, had at least completed high school education. This was not consistent with the findings in the literature which reported that the majority of the parents, (83%) of fathers and (82%) of mothers had at least completed high school education\(^\text{(71)}\). So, a significant proportion of parents in our study was either illiterate or had only completed primary
school level. This may have its effect on the disease understanding and the socioeconomic status of the family.

4.1.6. Parental occupation.

Most of the fathers were doing jobs (90%) and this was comparable with the findings in literature that (93.5%) were doing jobs. Most of the mothers in the study population were housewives (88%) which are not keeping with the reports in the literature that only (56.4%) were housewives\(^{(71)}\). However, might be a privilege for the mothers of the study group to pay more attention to their children and provide extra care. On the other hand this may reflect the low level of education of the mothers as there was no correlation between mothers being housewives and the children’s growth.

4.1.7. Degree of consanguinity of parents of the study group.

Consanguinity between parents was found in (56%) of the study group. This was keeping with that reported previously in Sudan\(^{(26)}\). Probably this is a pattern in Sudanese community; in addition this may point to a recessive genetic influence in the aetiology of congenital heart disease\(^{(27)}\).

5.1.8. Socio-economic status of the study group.

Almost half of the parents (51%) belonged to families of moderate social class, while (35%) were of low social class and only (14%) belong to families of high social class. This could be a reflection of the social background in our community
and would affect the attitudes of the family towards their children’s illness. In addition, this may affect the management of the patients since most of them need surgical corrections which are usually costly and out of reach of most of these families.

4.2. HISTORY AND CLINICAL DATA OF THE STUDY GROUP.

4.2.1. Diagnosis.

Acyanotic patients comprised (59%) of the patients and cyanotic patients constituted (41%) of the study group. This is not consistent with the results from China where acyanotic comprised (73.5%) and (27.5%) of the children had the cyanotic type of congenital heart disease\(^7\).

4.2.2. Treatment offered.

4.2.2.1. Current medications.

Fifty seven of the study group (57%) was receiving cardiac medications; this was not consistent with the literature where (16%) were taking cardiac medications\(^7\). This reflects the longer duration of medical treatment as surgery is usually delayed more in Sudan than in other countries.

4.2.2.2. Previous catheterization and surgery.

Thirty three of the patients (33%) had undergone previous operation and/or cardiac catheterization which was much less than that reported in the literature where (64.7%) had undergone previous surgery and/or cardiac
catheterization. This can be explained by the limited access to surgery and catheterization in Sudan\(^{(71)}\).

### 4.2.3. Family history of CHD.

Positive family history of (CHD) was present in (18\%) of the study group; in sibling was found in (27.8 \%) of the study group and in cousins in (66.7\%). This was rather more than but still comparable to (15.4\%) which is reported in literature\(^{(71)}\). This strengthened the fact that the risk of recurrence of (CHD) increases when there is positive family history\(^{(27)}\).

### 4.2.4. Past history of infective endocarditis.

Only one patient had documented episode of infective endocarditis, this could be explained by the small number of the study group which did not allow us to encounter more patients with infective endocarditis. Also there is a tendency by the doctors to treat any febrile patient with (CHD) with antibiotics for 2-4 weeks without doing blood culture which makes documentation of the diagnosis of (IE) difficult.

### 4.2.5. Past history of heart failure.

Nineteen percent of the patients in the study group had past history of heart failure. Fifteen of them (78.9\%) were on anti-failure medications. Moreover, 15 of them (78.9\%) having their weight below the 3\textsuperscript{rd} percentils and four encountered
severe malnutrition. This might strengthen the fact that congestive heart failure contributed towards the severity of the nutritional impairment\(^{(55,86,96)}\).

4.3. ANTHROPOMETRIC MEASUREMENT.

4.3.1. Weight.

More than half of the patients (55\%) had a weight below the 3\textsuperscript{rd} percentile. Thirty one per cent were described as failing to thrive\(^{(98)}\). This was less than that reported by Varan and his colleagues in Turkey, where (65.2\%) of their patients were below 3\textsuperscript{rd} percentile for weight and (41.6\%) of the patients were failing to thrive. Because most of the patients in Varan study had severe cardiac lesions, this might be well explained by the fact that the degree of growth impairment being closely associated with severity of the hemodynamic impairment\(^{(82,95)}\). On the other hand, (40\%) of the patients in our study were below (80\%) of their IBW for height, moderate to severe malnutrition, and (24\%) of the patients had mild malnutrition\(^{(99)}\). This is still not keeping with the findings of Varan study where (27\%) of his patients were below (80\%) of their (IBW) and (36\%) had mild malnutrition. This might indicate that patients with (CHD) are more prone to malnutrition and growth failure, highlighting the importance of nutritional assessment and adopting the most effective nutrition strategies\(^{(92,93)}\).
4.3.2. Height.

Thirty six per cent of the patients were below 3rd percentile, and were considered stunted, this was not keeping with the findings reported in Varan study where (48%) of the patients were stunted. Most of the patients in our study with short stature (31%) were also below the 3rd percentile for weight. This has strengthened the fact that malnutrition is prevalent and chronic malnutrition which affects both weight and height is also an important problem in congenital heart disease (93,95).

4.3.3. Head circumference.

Ten per cent of the children had head circumference below the 3rd percentile, (62%) between the 3rd-50th percentile, (27%) between 50th-97th percentile and only (1%) was above the 97th percentile, this reflect that head circumference is not much affected by the state of nutrition or haemodynamic effect.

4.4. PARENTAL KNOWLEDGE.


In the present study (30%) of the parents mentioned the specific diagnosis of their children's heart defect correctly. This was lower than (59%) which was reported by Cheuk and his colleagues, and much lower than (71%) and (98%) that were reported by Bulat and by Al-Jarallah and his colleagues respectively (71,73,79). This difference is perhaps not unexpected when determinants of
parental knowledge in this domain are considered. Cheuk and his colleagues reported that parental understanding of heart defects correlate with parental occupation and educational level\(^{(71)}\). Similarly these were identified as significant determinants in the present study. Septal defects were more likely to be mentioned as the diagnosis but interestingly, Ebstein's anomaly was correctly mentioned by parents of two patients out of the three patients encountered in this study. This is probably because the name Ebstein as the clinical diagnosis itself is specific. Moreover, because the cardiologist tend to spend more time explaining to patients with Ebstein's anomaly as they have noticed a remarkably high incidence in Sudan\(^{(32)}\). Twenty one per cent of the parents were able to indicate the location of the defect correctly on the diagram. This is rather less than but comparable to (28.8\%) reported in literature, considering the fact of the higher percentage of illiteracy in our study\(^{(71)}\).

Seventy three per cent of the parents correctly mentioned that the heart defect was congenital without knowing the possible aetiological causes. This was not consistent with the findings of Cheuk’s study, in which half of the parents were aware of the hereditary nature and possible aetiology\(^{(71)}\). This may reflect the lack of knowledge of the parents in our study; it could be due to high percentage of illiteracy. Ninety two per cent of the parents were aware of the symptoms attributable to the disease. This was higher than what was reported in Cheuk’s study.
which was only (51.3%). This could be due to a relatively simple acyanotic (CHD) presented in the study group. Of the 57 parents (57%) whom their children were taking cardiac medications at the time of the study, (73.7%) knew the name, dose and schedule of the medication. However, this was not consistent with (92%) which was reported by Al-Jarallah in his study[79]. Only (15.8%) of the parents in the study were aware of the side effects of cardiac medications. Although this is considered poor knowledge but still it is higher than the (07.1%) which was reported in the literature[71]. This poor knowledge is perhaps related to the emphasis being placed on the potential beneficial effect of the medications and the importance of compliance on the part of the medical staff. Moreover, it may also be due to the relative lack of emphasis on the potential side effects, especially when the parents did not take the initiative to ask and enquire about the possibility of such side effects.

4.4.2. Impact of heart disease on physical exercise capacity.

Parents of children with (CHD) had misconception about their children participating in various type of physical exercise. Of the sixty five children whose their mean age was 9.7 years. Only (56.9%) of the parents answered correctly on the necessity for exercise restriction appropriate for their children's heart condition. It is perhaps not surprising that parents of older children had a better understanding, as
questions regarding the need for exercise restriction are usually asked when their children join the primary school.

4.4.3. **Infective endocarditis and its prevention.**

Only (27%) of the parents were aware of the existence of (IE) in the study. The poor awareness of (IE) was not unique to this survey as three previous studies had reported that only (25%, 26.9%, 35%) of the parents knew about infective endocarditis\(^{(71,72,73)}\).

4.5. **DETERMINANTS OF PARENTAL KNOWLEDGE.**

4.5.1. **Name of the heart disease.**

The ability to mention the child’s disease correctly was not related to the type of the cardiac defect whether it is acyanotic or cyanotic (P =0.122). This was not consistent with that reported in the literature where the child’s cyanotic heart disease was mentioned correctly more often than acyanotic one (P <0.001). Probably because the name of the diagnosis of cyanotic (CHD) such as tetralogy of Fallot and transposition of great artery itself is composite and specific. Parents with higher educational attainment were more likely to mention the heart lesions correctly than those with lower educational level (P=0.002) for fathers, and (P = 0.00) for mothers, this was comparable with Cheuk study which reported (P=0.041) for fathers and (P=0.046) for mothers\(^{(71,80)}\). The correlation between parents' occupation and
knowledge of the name of the heart lesion was statistically significant in this study for the fathers (P=0.006). This was similar to the findings in a study by Cheuk (71).

4.5.2. Diagrammatic location.

The acyanotic cardiac lesions were indicated correctly more often than cyanotic ones (P = 0.003) this contradicting with previous study where cyanotic lesions were more likely to be indicated on the diagram (67,71). Parents' educational level was related to the ability to indicate the heart lesion or lesions on the diagram (P=0.011) for fathers and (P=0.024) for mothers. This finding was consistent with a previous study (71). Fathers' occupation was related to the ability to correctly indicate the heart lesion or lesions on the diagram (P= 0.008) for fathers. Moreover, this again comparable with the previous study (71). This might point to the positive correlation between parental educational level and occupation with their knowledge about (CHD).

4.5.3. Aetiology of the heart disease.

Cardiac diagnosis was not associated with the awareness of the possible aetiology of congenital heart disease (P=0.410). Fathers with high educational attainment were more likely to be correct regarding the cause of congenital heart disease (P= 0.004). The possible aetiology of (CHD) was not correlated with parents' occupation (P= 0.683) for fathers and (P= 0.256) for mothers. This strengthened the positive correlation between parental knowledge and their educational level.
4.5.4. Symptoms of the heart disease.

The ability to describe cardiac symptoms was statistically insignificant with the cardiac diagnosis (P= 0.479). Regarding parents' education the (P=0.070) was for fathers and (P= 0.685) for mothers and for their occupation the (P= 0.448) for fathers and (P= 0.992) for mothers. This was inconsistent with previous study by Cheuk(71).

4.5.5. Physical exercise capacity.

Parents whose children were older (mean age 9.7 versus 1.6 years) constituted (56.9%) and were more likely to give correct answers to the question on guidelines regarding activity levels and sports participation (P<0.001). This was consistent with (59%) that reported in Cheuk study(71).

4.5.6. Awareness of infective endocarditis.

Awareness of infective endocarditis was related to the fathers' higher educational attainment, 37.5% versus 17.5%, (P= 0.039). This was comparable with the findings in the literature, 30.2% versus 11.1%, (P=0.042). Furthermore fathers' occupation such as professional, businessmen and skilled worker was related to awareness of (IE), 34% versus 19.1%, (P= 0.023). Similarly this was consistent with that reported in the China, 32.1% versus 14.9%(71). Although parental knowledge of bacterial endocarditis was limited and intensified education are needed(74). Yet it is correlated with higher educational attainment(70,72,73).
Logistic regression analysis showed that cardiac diagnosis, educational attainment and occupation of the parents were the main determinants of parents' knowledge of the nature of their child's heart disease. Acyanotic heart lesions were more likely than cyanotic lesions to be mentioned (P=0.021) and indicated on the diagram correctly (P=0.00). This was not consistent with the findings in the study done by Cheuk\(^{(71)}\). Where cyanotic heart lesions were more likely to be mentioned and indicated on the diagram correctly (P<0.001). Regarding parents with high educational attainment were likewise more likely to give correct answers when asked to mention the diagnosis (P=0.002), indicate on a diagram (P=0.037) and provide the possible causes of congenital heart disease (P=0.014). Moreover professionals, skilled workers and employees were more likely to indicate on a diagram the heart lesion or lesions (P=0.022) and to give possible causes of congenital heart disease (P=0.009). This was consistent with the finding of the study by Cheuk\(^{(71)}\).

4.6. PARENTAL ATTITUDES.

4.6.1. Parental feeling towards their child's illness.

Almost half of the parents (52%) were worried about their child's illness, (19%) were depressed, those who were feeling sad constituted (15%) of the parents, (10%) of the parents were anxious and only (4%) of the parents felt guilty. This might reflect the negative attitudes of the parents towards (CHD). However, parents
are usually worried about their child's social life, as the children with (CHD) are usually considered different, or may have limited capacity in participating in sports and physical activities.

4.6.2. Caring and protection of the child with CHD.

The majority of the parents (93%) tended to give more attention and care to their children with (CHD) compared to their siblings. All of them (100%) tended to protect their children from sources of infection as much as they could. This was not consistent with the findings reported by Andresova in Czechoslovakia, where the mothers did not display excessively centered relation to the child with (CHD), nor exaggerated protectiveness\(^7\). The parents of children with (CHD) usually concern about how their sick children will be responsible for their own management in the future. Although the parents would like their children to become independent, yet they also had misgivings and occasionally were overprotective.

4.6.3. Blaming oneself to have a child with CHD.

Nineteen of the parents blame themselves to have a child with (CHD). This feeling could be due to ignorance of the parents about their children’s heart disease which had been conveyed to me indirectly during the interview. Moreover this might reflect the negative attitudes of the parents towards (CHD).
4.7. PARENTAL PRACTICE.

4.7.1. Dental health.

Regarding regular dental check up, only (18.9%) of the parents visited the dentist regularly with their children for check up, this was much less than (52%) which was reported by Barreira in Portugal in his study\(^{72}\). Likewise only five of the parents (9.4%) in this study knew that antibiotics had to be taken before dental procedures, although they may not be aware of the underlying reason for doing so. This was inconsistent with (48%) reported by Barreira in his study\(^{72}\). Fifty one (96.2%) of the parents declared that their children brushed their teeth at least once a day. This was comparable with (92%) that reported in previous study by Barreira, but inconsistent with that reported by Saunders where (21%) of the children with (CHD) never or hardly ever brushed their teeth twice a day\(^{72,76}\).

4.7.2. Nutritional consideration.

Almost half of the parents (51%), asked their doctors for nutritional suggestions and advices. Regarding frequency of the feeding, the majority of the parents (59%) used to offer three main feeds, while (38%) gave small frequent feeding and only (03%) offered feeding on demand. To maintain weight and growth (81%) of the parents mentioned that they try to give extra calories to their children as much as they could. Seventy six percent of the parents offered high calories milk which was usually home made by the possible available items, such as adding
vegetable oil or eggs to enrich cow milk, the commonly used milk in our community. As, high calories ready made formula and additives like that based on glucose such as Polycose powder and Forticol and those based on fat like Medium chain triglyceride oil and Calogen are not readily available. Twenty three per cent of the parents add special nutritional supplements to milk such as the ready made fortified cereals. Likewise, (59%) used to offer snacks that are rich in calories, while (59%) avoided giving their children foods that have low calories. As, children with (CHD) become tired quickly during feeding or even sleep through it. They may not have enough energy to eat properly. So, it was usually recommended to offer a balanced diet, as well as one that higher in calories.

Despite this effort, the growth of their children was still far from that expected. However, this might strengthen the fact that parents' perception of having adequate information regarding nutritional supplement often was incorrect, along with inadequate nutrient intake caused by propensity for fatigue or tachypnea.

**4.8. KNOWLEDGE SCORES.**

The majority of the parents (64%) were found to have poor knowledge about (CHD). While only (36%) of the parent were found to have good knowledge. This reflects the insufficient knowledge and inadequate information attained by the parents. This could be explained by the inadequate current setup of the cardiac clinic. All the procedures such as clinical examination, (ECHO) and the parents'
education, both medical and nutritional were offered by a single paediatric cardiologist. Supporting staff like dietitian, social worker or specially trained nurse were not readily available in the clinic. In addition primary care health centres and health workers play a weak role in providing health and nutritional education to the community. Along with the unavailability of high calories ready-made supplements.

4.9. **IMPACT OF PARENTAL KNOWLEDGE ON GROWTH.**

No correlation was found between parental knowledge and improved growth. Regarding weight there was no relation between good knowledge and better weight gain (P=0.788). Moreover, there was no correlation between the height of the patients and parental knowledge (P=0.147). Although head circumference was the least to be affected, yet there was no relation between it and parental knowledge (P=0.797). The nature of heart disease such as those conditions with intractable congestive heart failure also contributed towards the severity of the nutritional impairment. Certainly the long delay of corrective surgery will put more patients at risk for malnutrition. Several reports had documented encouraging results of early repair of critical congenital heart defects in symptomatic neonates and infants rather than palliative operations and primary surgical closure of large VSD\(^{(89,90,91)}\). Ultimately, a more intensive nutritional treatment should be considered to optimize the outcome.
CONCLUSION

- Congenital heart disease presented mostly below 5 years of age with slight female preponderance.
- The majority of the patients belonged to Arab tribes and were of moderate social class.
- Fifty five per cent of the patients were below 3rd percentile for weight and (31%) were labeled as failing to thrive. Forty percent of the patients were below (80%) of their ideal body weight for height, with moderate to severe malnutrition. So this adds to the fact that patients with (CHD) are prone to malnutrition and growth failure.
- Despite the usual practice in cardiac clinics to explain a patient's heart disease and its short and long term implications in lay terms, the findings of this study suggested that parents had significant knowledge gaps.
- Most of the parents could mention the cardiac lesions, either in part or completely, and were knowledgeable concerning previous surgical or catheter interventions.
- The knowledge of the parents about the aetiology and their awareness of the impact of their children's heart disease on exercise capacity, were limited. Moreover, their knowledge about infective endocarditis and side effects of cardiac medications was quite deficient.
• Parental educational level, occupation and cardiac diagnosis correlated positively with knowledge of (CHD).

• The parents’ negative attitudes and high concern were prevalent, as most of the parents were worried and depressed. In fact this might have its impact on the patients’ quality of life and wellbeing.

• Nutritional practice was inefficient in promoting growth, mainly because of the lack of nutritional supplement in the market while the dietitians play a very weak role in providing nutritional educations.

• Parental good knowledge did not correlate with growth improvement.
RECOMMENDATIONS

- Cooperation between paediatricians and paediatric cardiologists is needed for intensified continuous educational programs and frequent assessment of parental awareness about congenital heart disease. This will reduce the congenital heart disease morbidity and mortality. An additional benefit is to help parents to impart accurately their knowledge to their children.

- It should be a practice to advise that children with congenital heart disease should seek out and attend dentists for regular dental check up.

- Nutritional and health educational programs along with regular follow up visits are essential, to properly assess growth and to re-evaluate nutritional needs for the children with congenital heart disease. To optimize this, a registered dietitian who works in conjunction with paediatrician should be available.

- High calories ready-made supplements are needed and should be available for all patients.

- It is important to establish programs, like parents support groups, to help the parents overcome the negative psychological impacts of congenital heart disease.

- To improve health services and facilities for management care for these children in all States in the country.
• The school health service program is particularly required to be involved in all aspects of advice, care and education concerning these children.

• More efforts should be made by the Central and Federal Government to reduce the high illiteracy rate in the country.
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Parental knowledge, attitudes, and practice towards their children’s congenital heart disease and its impact on their growth
in Sudan Heart Centre

Questionnaire

0. STUDY DATA :
0.1. Serial No. 0.2. Tel No.
0.3. Hospital : 0.4. Date :
0.5. Unit :

1. THE PERSONAL DATA
1.1. Name : 
1.2. Age : 1.2.1 ( <1 yr) 1.2.2. (1yr – <5yrs)
1.2.3. (5yrs – <10yrs) 1.2.4. ( 10yrs -15yrs )
1.3. Gender : 1.3.1. Male 1.3.2. Female
1.4. Tribe : 
1.5. Ethnic group :
1.6. Residence : 1.6.1. Rural 1.6.2. Urban 1.6.3. Suburban

2. DEVELOPMENTAL HISTORY:
2.1. Early childhood development :
2.1.1. Normal 2.1.2. Delayed
2.2. School level :
2.2.1. Kinder garden 2.2.2. Basic school 2.2.3. Secondary school
2.3. School attendance : 2.3.1. Good 2.3.2. Not good
2.4. School performance :
2.4.1. Excellent 2.4.2. Good 2.4.3. Moderate 2.4.4. Weak

3. FAMILY HISTORY:
3.1. Father : 3.1.1. Alive/well 3.1.2. Alive/ill 3.1.3. Dead
3.2. Mother: 3.2.1. Alive/well 3.2.2. Alive/ill 3.2.3. Dead
3.3. Consanguinity:
3.3.1. 1st degree 3.3.2. 2nd degree 3.3.3. Far relative 3.3.4. No
3.4. Number of siblings: 

3.5. Family history of CHD: 3.5.1. Yes 3.5.2. No

4. PAST HISTORY:
   4.2. History of infective endocarditis: 4.2.1. Yes 4.2.2. No
   4.3. History of arrhythmia: 4.3.1. Yes 4.3.2. No
   4.4. Others specification

5. VACCINATION:
   5.1. Up to date 5.2. Incomplete 5.3. Not vaccinated

6. DIAGNOSIS:
   6.1. VSD 6.2. ASD 6.3. AVSD 6.4. PDA
   6.5. AS 6.6. PS 6.7. TOF 6.8. TGA
   6.9. Others

7. MANAGEMENT (TREATMENTS OFFERED):
   7.1. Current medications:
      7.1.1. Inotropic agent: Digoxin
      7.1.2. Diuretics: Frusemide, Spironolactone
      7.1.3. Vasodilators: Captopril
      7.1.4. Others
   7.2. Previous operations: 7.2.1. Yes 7.2.2. No
   7.3. Previous catheterization: 7.3.1. Yes 7.3.2. No

8. ANTHROPOMETRIC MEASUREMENTS:
   8.1. Weight --kg
      8.1.1. <3rd percentile 8.1.2. 3rd -50th percentile
      8.1.3. 50th -97th percentile 8.1.4. >97th percentile
   8.2. Height (Length) --cm
      8.2.1. <3rd percentile 8.2.2. 3rd -50th percentile
      8.2.3. 50th -97th percentile 8.2.4. >97th percentile
   8.3. Head circumference --cm
      8.3.1. <3rd percentile 8.3.2. 3rd -50th percentile
      8.3.3. 50th -97th percentile 8.3.4. >97th percentile
   8.4. Ideal body weight --%
9. SOCIAL HISTORY (The parents data):

9.1. Responder:  
9.1.1 Father  
9.1.2 Mother

9.2. Age:  
9.2.1 Father…….  
9.2.2 Mother……

9.3. Educational level:

9.3.1. Father  
9.3.1.1 Illiterate.  
9.3.1.2 Khalwa.  
9.3.1.3 Primary school  
9.3.1.4 Secondary school  
9.3.1.5 University.  
9.3.1.6 Postgraduate.

9.3.2. Mother  
9.3.2.1 Illiterate.  
9.3.2.2 Khalwa.  
9.3.2.3 Primary school  
9.3.2.4 Secondary school  
9.3.2.5 University.  
9.3.2.6 Postgraduate.

9.4. Occupational status:

9.4.1. Father  
9.4.1.1 Professionals  
9.4.1.2 Businessman  
9.4.1.3 Small scale businessman  
9.4.1.4 Employee  
9.4.1.5 Skilled laborer  
9.4.1.6 Unskilled laborer  
9.4.1.7 Unemployed

9.4.2. Mother  
9.4.2.1 Housewife  
9.4.2.2 Professionals  
9.4.2.3 Businesswoman  
9.4.2.4 Small scale businesswoman  
9.4.2.5 Employee  
9.4.2.6 Skilled laborer  
9.4.2.7 Unskilled laborer  
9.4.2.8 Unemployed

9.5. Income:  
9.5.1 High  
9.5.2 Moderate  
9.5.3 Low

9.6. Housing condition:

9.6.1 Good  
9.6.2 Average  
9.6.3 Poor

10. PARENTAL KNOWLEDGE:

10.1. Did your doctor offer any explanation?  
10.1.1 Yes  
10.1.2 No

10.2. Do you think you have received enough information about your child heart disease?  
10.2.1 Yes  
10.2.2 No

10.3. Do you think that you have well understood the problem?  
10.3.1 Yes  
10.3.2 No
10.4. Nature of heart disease and its treatment:

10.4.1. What is the name of your child heart defect?

10.4.2. Describe or indicate on the diagram where your child heart defect is located?

10.4.3. What do you think the cause of CHD?
   - Hereditary
   - Congenital
   - Infection
   - Drugs

10.4.4. Mention all symptoms which may occur, and for which you have to contact your child's cardiologist:
   - Shortness of breath
   - Bluish discoloration.
   - Sweating associated with feedings.
   - Difficulty in feeding
   - Swollen feet.
   - Squatting.
   - Others.

10.4.5. Treatments offered:
   - Medications:
     - Name of the drug, dose and schedule
   - Catheterization
   - Surgery

10.4.6. How often does your child have to come for follow up?
   - As advised
   - When needed

10.4.7. What is the purpose of the follow up?
   - To detect any clinical deterioration
     - Yes
     - No
10.4.8 What is the expected disease prognosis?
   10.4.8.1. Can be cured
     10.4.8.1.1. Yes  10.4.8.1.2. No
   10.4.8.2. Life long disease
     10.4.8.2.1. Yes  10.4.8.2.2. No

10.5. Impact of heart disease on exercise capacity:
   10.5.1. Do you think it is necessary to follow any guidelines regarding activity levels and sports participation for your child with CHD?
     10.5.1.1. Yes  10.5.1.2. No
   10.5.2. Is any advices offered regarding safe exercise?
     10.5.2.1. Yes  10.5.2.2. No

10.6. Infective endocarditis (IE) and its prevention:
   10.6.1. Do you know what is endocarditis?
     10.6.1.1. Yes  10.6.1.2. No
   10.6.2. Do you know how it presents?
     Unexplained fever for > 5 days:
     10.6.2.1. Yes  10.6.2.2. No
   10.6.3. Do the following factors contribute to the onset of IE?
     10.6.3.1. Dental caries  10.6.3.2. Skin infection
     10.6.3.3. Poor nail and skin care  10.6.3.4. Body cautery

11. PARENTAL ATTITUDES:
   11.1. What was your feeling when you were first informed about your child’s problem?
     11.1.1 Worried  11.1.2. Depressed  11.1.3. Guilty
     11.1.4. Incriminate  11.1.5. Others
   11.2. Do you give more attention and care to your child with CHD?
     11.2.1. Yes  11.2.2. No
   11.3. Do you try to protect your child from infection?
     11.3.1. Yes  11.3.2. No
   11.4. Do you talk with your doctor to give you advices to cope with your child?
11.4.1. Yes    11.4.2. No

11.5. Do you blame yourself to have a child with CHD?
    11.5.1. Yes    11.5.2. No

12. PARENTAL PRACTICE:

12.1. Do your child with CHD has a dental check up regularly?
    12.1.1. Yes    12.1.2. No

12.2. Does your child take A/B before every visit to the dentist?
    12.2.1. Yes    12.2.2. No

12.3. Does he clean his teeth at least once a day?
    12.3.1. Yes    12.3.2. No

12.4. How often do you offer feeds to your child with CHD?
    12.4.1. On demand.
    12.4.2. 3 main feeds.
    12.4.3. Frequent small feeds.

12.5. Do you give extra calories in order to maintain weight and growth of your child?
    12.5.1. Yes    12.5.2. No

12.6. How do you increase the calories intake?
    12.6.1. Offer high calories milk.
        12.6.1.1. Yes    12.6.1.2. No
    12.6.2. Add special nutritional supplements to formula.
        12.6.2.1. Yes    12.6.2.2. No
    12.6.3. Offer nutritious foods and snacks that are high in calories.
        12.6.3.1. Yes    12.6.3.2. No
    12.6.4. Avoid giving your child foods that have low calories; foods with a lot of sugar and few nutrients.
        12.6.4.1. Yes    12.6.4.2. No

12.7. Do you ask your doctor for nutritional suggestions?
    12.7.1. Yes    12.7.2. No

12.8. As your child has CHD, should you gave him A/B immediately if he has a fever without consulting his doctor?
    12.8.1. Yes    12.8.2. No
CARDIAC DIAGRAM