مجلة العلوم الطبية
Journal of Medical Sciences
www.Suj.sebhau.edu.ly
Received 12/02/2018 Revised 08/08/2018 Published online 15/11/2018

# Prevalence and Pattern of Congenital Heart Disease in South Libya

Abraheem M. Mansour
Head of Paediatric Department, Faculty of Medicine, Sebha university, and Chief Neonatology Division, Sebha Medical Center ,Libya.

**Abstract:**
**Aim**: This research was performed to study the prevalence of Congenital Heart Disease (CHD) in south Libya.. Data on the prevalence of CHD were collected and analyzed from the major hospital in South Libya from 2015 to 2016. Congenital heart disease is the most common congenital problem in children. Presentation can vary from asymptomatic, accidental finding to severe cardiac decomposition and death. Early recognition has great implications on prognosis. This study was carried out to identify the specific types of CHD, sex and age distribution and the most common pattern of presentation among hospitalized patient. The study also aims at studying age, gender distribution and frequency of congenital heart disease in children referred to echocardiography in Sebha Medical Center.
**Methods:** This is a prospective descriptive study of most patients (inpatient and outpatient) with clinical finding i.e. cyanosis, systolic murmur, family history of CHD, congenital malformation and recurrent chest infection. From September 2015 to September 2016. Infants of diabetic mothers are referred for echocardiography. Patients from day one of life until 14 years are included in the study.
**Results:** Out of the 1600 total cases, 425 (26.5%) were diagnosed with congenital heart disease. There was a male preponderance. There were 219 males and 206 females with a M:Fratio of 1:0.9 The mean age of all the patients with CHD was days. Of the total cases, 116 (26.8%) had patent ductus arteriosus (PDA), 63 (14.8%) had atrial septal defect (ASD), 63 (14.8%) had PR, 56 (13.2%) had ventricular septal defect (VSD), 43 (10.1%) had Mixed CHD, 18 (4.2%) had TR, 15 (3.5%) had IVSH, 13 (3.1%) had Pulmonary stenosis, 11 (2.6%) had tetralogy of fallot (TOF), and 10 (2.4%) had transposition of great arteries (TGA), 8 (1.9%) had aortic stenosis, 4 (0.9%) had AVCD, 1 (0,2%) each was diagnosed to have Truncus arterioses, Coarctation of aorta, Dextrocardia and Aortic regurgitation.
**Conclusion:** The majority of patients with CHD defects had non-cyanotic CHD. TGA is the most common cyanotic lesion and PDA non-cyanotic lesion. In order to avoid complications, early detection of congenital heart disease is of utmost importance for proper treatment. 2D-echo with Doppler examination forms the gold standard for diagnosis.
**Key words**: Congenital heart disease, 2-Dimention echocardiography, ventricular septal defect, patent ductus arteriosus.

**Introduction:** Congenital heart diseases occur in 0.8% of live births. The incidence is higher in stillborns 3-4%, abortuses 1.0 to 2.5%, and premature infants' about 2%, excluding patent ductus arteriosus PDA.
Congenital cardiac defects have a wide spectrum of severity in infants. About 30-40% of patients with congenital cardiac defects are symptomatic in the 1st year of life, where as the diagnosis can be established in 60% of patients by the 1st month of age. There are 10 common congenital heart lesions PDA, ASD, PR,VSD, IVSH, PS, TOF, TGA, AS and Coarctation of aorta. All together make up 58% of all cases. Although the advent in diagnostic techniques (especially the color Doppler echocardiography), PDA is considered the commonest lesion and constitutes about 27% of all CHD while ASD, PR and VSD are considered the next most common congenital heart diseases. Each lesion forms about 13-115% of all congenital heart diseases. Tetralogy of Fallot accounts for 2.6% of the whole CHD and TGA for 2.4 % of these diseases.

**Types of CHD:**

**1- A cyanotic Heart Disease:**

The most common lesions in acyanotic congenital heart disease that cause left to right shunting and volume overload are: PDA, ASD and VSD.
The direction and magnitude of the shunt across such a communication depends on the size of the defect and relative symptoms. Large lesions such as large VSD may be associated with little shunting and few symptoms during the initial weeks of life. When pulmonary vascular resistance declines over the next several weeks, the volume of the left-to-right shunt increases. If the hole is small, the shunt may be trivial, but if it is large it may represent the majority of cardiac output. So the blood flow through the pulmonary artery may be several times greater than that through the aorta, and this results in signs and symptoms of heart failure and failure to thrive begins to appear. So most patients with CHD and significant left to right shunt have poor weight gain.

**2- Cyanotic Heart Disease:**

Some infants and children with congenital heart disease are centrally cyanosed because the unsaturated blood bypasses the lungs. Cyanotic heart disease can be subdivided into two types.
In the first type, the lungs are under-perfused as blood shunts from right to left bypassing the lungs, TOF is the example, the essential features in TOF, being the most cyanotic CHD, are a large VSD and stenosis of the right ventricular outflow tract. There is resistance to the flow of blood through the pulmonary valve with a consequent shunt of blood from the right to left ventricle and thence to the aorta. So the degree of cyanosis depends on the degree of obstruction to pulmonary blood flow. The newborn with this lesion and mild degree of obstruction usually is

pink and cyanosis develops and increases over the next few weeks or months. If the obstruction is severe, the cyanosis may be present during the neonatal period. Hyper cyanotic spells can be a particular problem during the first two years. Heart failure is extremely rare in TOF, so poor weight gain is seldom a problem in such patients.
In the second type, the lungs are normally filled or even over-perfused with blood, but cyanosis results because there is inadequate mixing of both the systemic and the pulmonary circulations. Transposition of the great arteries is the most common example.
In patients with transposition of great arteries, which is the most common cause of cyanosis during the neonatal period, progressive cyanosis within the first few hours or days of life and the affected baby becomes increasingly blue without treatment. This disease has lethal course and few patients survive the first year of life.

**Material and Methods:**
This is a prospective chart review conducted in the Pediatric echo-cardiac clinic from Sep. 2015 to Sep. 2016 in Sabha Medical Center in Libya. Data on the prevalence of CHDs were collected and analyzed. The center receives large numbers of patients, including children, who are referred by other hospitals from in and out of Sebha. 1600 patients participate in the study. Some of them have been examined for various illnesses in SMC by pediatrician. They were divided into five groups, according to age (table. 1, fig. 1).
1) 0 to 28 day,
2) 1 month to 1 years,
3) 1 year to 5 years
4) 5 years to 12 years.
5) more than 12 years
The source of information was the medical and echocardiography records at the hospital. The suspected CHD patients who showed symptoms like poor feeding, failure to thrive, respiratory problem, discrepancy in pulse, cyanosis, heart murmur, abnormal chest x-Ray, and previous family history and infant of diabetic mother have been subjected by the researchers for further clinical diagnosis. Extensive X-ray analysis, ECG and echocardiography examination for the confirmation of the CHD were performed. Prevalence, age and sex specific frequency of all kinds of CHD were computed. For each patient, the following data have been collected: age, sex, residence. The diagnosis of CHD was done by two dimensional echocardiography and Doppler studies.
The echocardiography studies were performed with a mechanical sector scanner at 3 & 5 MHz. Patients were studied in the supine or left lateral decubitus position to obtain different views. Neither catheterization nor surgical interventions were performed to any patient.
CHD is defined as the structural heart disease or intrathoracic great vessels that is actually or potentially of functional significance present at the time of birth even if there was a delay in detection. One thousand and six hundred children were studied. Age ranged from day1 until 14 years of age, Clinical data were reviewed. Consideration was given to the total number of cases with CHD, age at diagnosis, sex distribution and type of CHD.

**Results:**
A total of (425) children were included. There were (219) males (51.52%) and 206 were females (48.47%), with a male: female ratio of 1.2:1 (table 2, fig 2). Four hundred and three children (94.82%) of the total cases had acyanotic cardiac defects. Cyanotic heart defects were seen in 403 patients. PDA followed by a trial septal defect, PR, VSD, were the most common. Cyanotic congenital heart lesion, TOF, TGA respectively. Whereas TOF followed by TGA and AVCD were the commonest cyanotic congenital heart lesions as shown in Figure 3 compares acyanotic and cyanotic congenital heart diseases. Female dominance of PDA and A-V canal lesions 63% and 68% of cases respectively were seen. Whereas a male dominance in the aortic valve lesions was seen. The age of the patient ranged from one day to 14 years. Among the 425 studied patients, there were 116 patients (27%) with PDA, 11 patients (26%) diagnosed as TOF and 56 patients (13,2%) showed to have VSD, 10 patients (2.4%) had TGA, 13 patients (3.1%) had pulmonary stenosis. The least common congenital heart disease were Epstein anomalies, Dextrocardia, Truncus arteriosus, and Aortic regurgitation, diagnosed in 4 patients (0.2%) for each defect only (table 3) (fig. 3-4).
The study of sex distribution among different CHD showed that PDA and ASD were more common in female, while other lesions of congenital heart disease had equal male to female, or slightly more common in male incidence (table 4).
There were 317 cases from Sebha (74.6%), 27 from Ashatti (6.4%), 36 cases from Morozok (8.5%), followed by 33 cases from Obary (7.8%) (table 5, fig. 5).
The study of the time of presentation of CDH revealed that 179 patients (42.1%) presented in the 1 day to 28 days of life, 117 patients (27.5%) from one month to one year of life, 49 patients (11.5%) from 1 year to 5 year, 76 patients (17.5%) from 5 year to 12 years and 4 patients (0.9%) were diagnosed after 12 years of age (table 1, fig. 1).

**Discussion:**
Our hospital received referred cases from different other hospitals in south Libya for Echocardiology, because of the increase in the orientation about the congenital heart disease in south Libya in the last few years.
This study proved that PDA is the commonest CHD both in community and hospital based studies, (26.8%) because of recent use of Doppler study by which PDA is easily defected. ASD and PR were the second most CHD in our study (14.8%, 14.8%) respectively. VSD showed to be the third most common congenital lesions (13.2%).
There is higher incidence of TGA than in other studies and this is due to increasing the orientation about the necessity of early referring of such patients to our hospital for paper diagnosis.

This study revealed that there was lower incidence of Truncus arteriosus, Coarctation of aorta, Dextrocardia and Aortic regurgitation.

The distribution of incidence of CHD between Male and Female confirmed that the female patients with PDA and ASD outnumber males, while in all the other lesion than was slight male predominance.

Regarding the age of which the diagnosis had been done, our study indicates that the patients with left to right shunt (PDA), ASD and VSD mostly presented during the first year of life, while the patient with pulmonary regurgitation mostly presented between 5-12 years of life.

All patients who were diagnosed as TOF and TGA presented within 29 day – 1 year of life and this compatible is with the natural history of this disease (table 3).

In our study regarding to geographic distribution large numbers of patients were from Sebha (74%), followed by Shatte (6.4%), Morozok (8.5%), Obary (7.8%), Katroon (0.9%), and Gaat (1.2%) (tab. 6-7, fig. 5)

**Conclusion:**

Congenital heart diseases are an important group of diseases that cause great morbidity and mortality in children. Our aim was to show our experience and compare it to others. The present study shows, for the first time, the prevalence and pattern of CHD in South Libya. These findings will help establish a database for future studies, which are hoped to focus on etiology and ethnic disparity of CHD in the region. The findings can help to establish valuable changes in health policies for the improvement of diagnostic and therapeutic facilities. It is generally accepted that the improvement in diagnosis, attention and awareness among general pediatrician and early referral to pediatric cardiologists have resulted in an increase of reported prevalence of CHD. The present study indicates that CHD is an important pediatric cardiac problem in our study group.

**Table 1: The Age Distribution among various Types of CHD.**

| Age | N | % |
|---|---|---|
| 1 d- 28 d | 179 | 42.1 |
| 29 d- 1 y | 117 | 27.5 |
| 1.2 y- 5 ys | 49 | 11.5 |
| 5.2 ys- 12 ys | 76 | 17.9 |
| More than 12ys | 4 | 0.9 |
| Total | 425 | 100 |

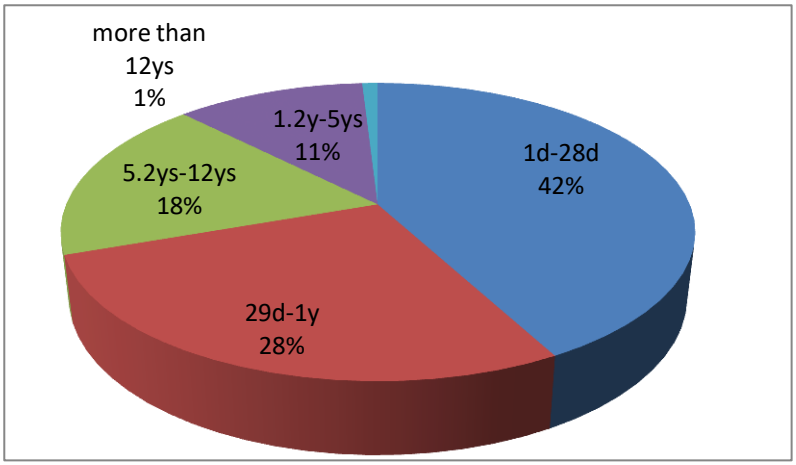


**Figure 1: The Age Distribution among various Types of CHD.**

**Table 2: The sex distribution at diagnosis of different types of CHD.**

| Sex | % | N |
|---|---|---|
| Male (M) | 51.5 | 219 |
| Female (F) | 48.5 | 206 |
| Total | 100 | 425 |

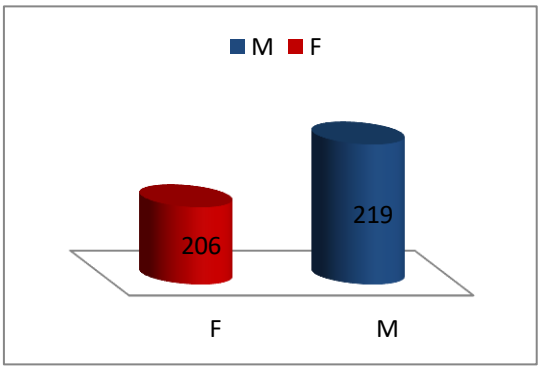


**Figure 2: The sex distribution at diagnosis of different types of CHD.**

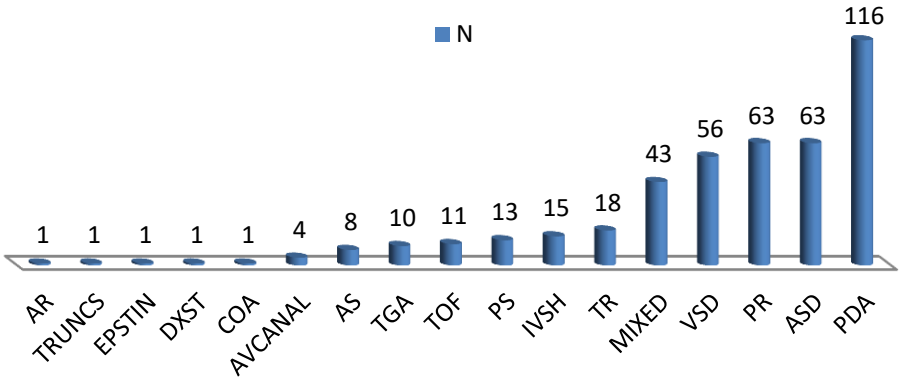


**Fig. 3: Pattern and Frequency of Congenital Heart Disease CHD**

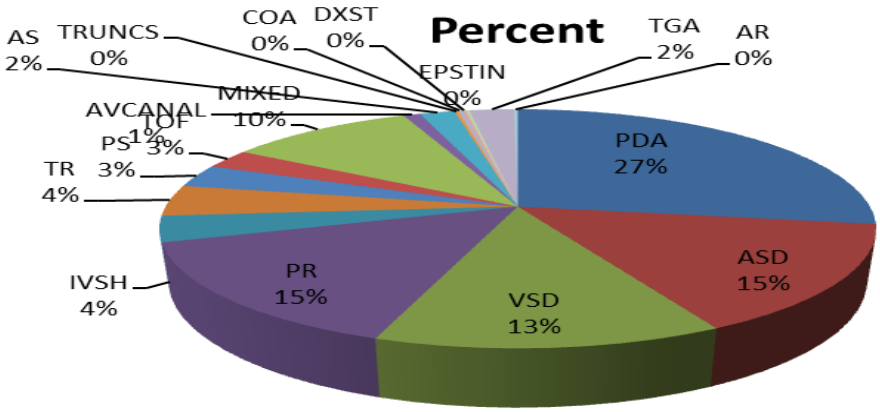


**Figure 4: Pattern and Frequency of Congenital Heart Disease CHD.**

**Table 3: Diagnoses Age Cross Tabulation**

| Diagnoses | Age | | | | | Total |
|---|---|---|---|---|---|---|
| | 1d-29d | 29d-1y | 1.2y-5ys | 5.2ys-12ys | More than 12ys | |
| PDA | 83 | 22 | 7 | 4 | 0 | 116 |
| ASD | 23 | 30 | 5 | 5 | 0 | 63 |
| VSD | 15 | 23 | 6 | 12 | 0 | 56 |
| PR | 3 | 6 | 16 | 34 | 4 | 63 |
| IVSH | 14 | 1 | 0 | 0 | 0 | 15 |
| TR | 9 | 1 | 5 | 3 | 0 | 18 |
| PS | 4 | 6 | 0 | 3 | 0 | 13 |
| TOF | 2 | 4 | 3 | 2 | 0 | 11 |
| MIXED | 20 | 13 | 3 | 7 | 0 | 43 |
| AVCANAL | 0 | 3 | 1 | 0 | 0 | 4 |
| AS | 0 | 1 | 2 | 5 | 0 | 8 |
| TRUNCUS | 0 | 1 | 0 | 0 | 0 | 1 |
| COA | 0 | 1 | 0 | 0 | 0 | 1 |
| DXST | 1 | 0 | 0 | 0 | 0 | 1 |
| EPSTIN | 1 | 0 | 0 | 0 | 0 | 1 |
| TGA | 4 | 5 | 1 | 0 | 0 | 10 |
| AR | 0 | 0 | 0 | 1 | 0 | 1 |
| Total | 179 | 117 | 49 | 76 | 4 | 425 |

**Table 4: Diagnosis Sex Cross Tabulation**

| Diagnoses | Sex | | Total |
|---|---|---|---|
| | Male (M) | Female (F) | |
| PDA | 57 | 59 | 116 |
| ASD | 30 | 33 | 63 |
| VSD | 31 | 25 | 56 |
| PR | 36 | 27 | 63 |
| IVSH | 11 | 4 | 15 |
| TR | 8 | 10 | 18 |
| PS | 3 | 10 | 13 |
| TOF | 4 | 7 | 11 |
| MIXED | 23 | 20 | 43 |
| AVCANAL | 2 | 2 | 4 |
| AS | 5 | 3 | 8 |
| TRUNCUS | 1 | 0 | 1 |
| COA | 1 | 0 | 1 |
| DXST | 0 | 1 | 1 |
| EPSTIN | 1 | 0 | 1 |
| Total | 219 | 206 | 425 |

**Table 5: Diagnoses Residence Cross Tabulation.**

| Diagnoses | Residence | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Sebha | Shatte | Morozok | Obary | Katroon | Gaat | Chwerf | Beng | Jfra | Total |
| PDA | 85 | 8 | 11 | 9 | 1 | 2 | 0 | 0 | 0 | 16 |
| ASD | 49 | 4 | 3 | 4 | 2 | 0 | 0 | 1 | 0 | 63 |
| VSD | 45 | 3 | 4 | 3 | 0 | 1 | 0 | 0 | 0 | 56 |
| PR | 47 | 4 | 6 | 5 | 0 | 1 | 0 | 0 | 0 | 63 |
| IVSH | 8 | 3 | 2 | 2 | 0 | 0 | 0 | 0 | 0 | 15 |
| TR | 16 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 18 |
| PS | 11 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 13 |
| TOF | 8 | 1 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 11 |
| MIXED | 26 | 3 | 6 | 6 | 1 | 0 | 1 | 0 | 0 | 43 |
| AVCANAL | 3 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 4 |
| AS | 6 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 8 |
| TRUNCUS | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| COA | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| DXST | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| EPSTIN | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| EPSTIN | 8 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 10 |
| TGA | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| Total | 317 | 27 | 36 | 33 | 4 | 5 | 1 | 1 | 1 | 425 |

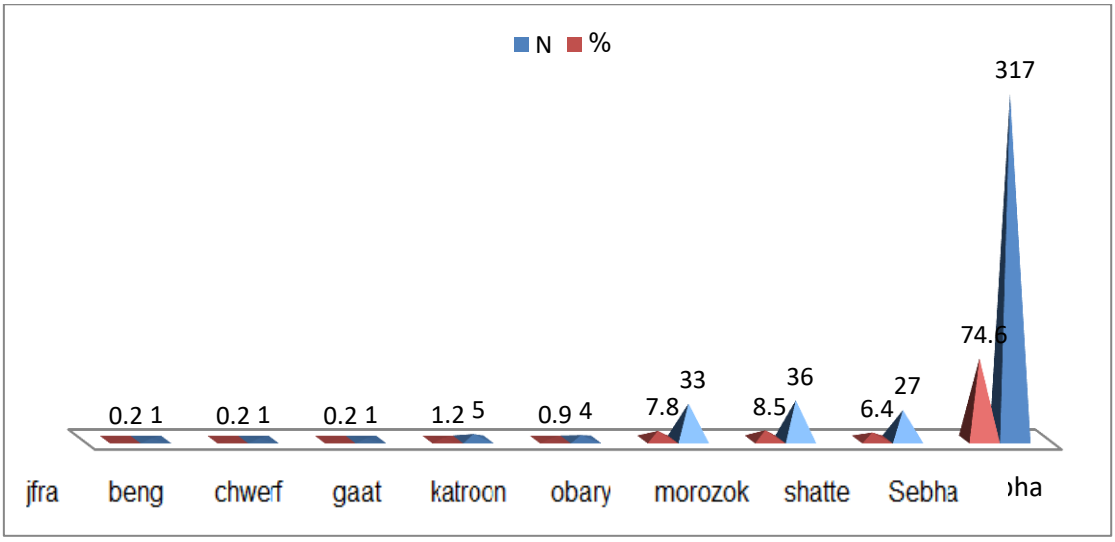


**Figure 5: Geographic distribution of various Types of CHD.**

**Table 6: Residence Sex Cross Tabulation**

| Residence | Sex | | Total |
|---|---|---|---|
| | Female (F) | Male (M) | |
| Sebha | 149 | 168 | 317 |
| Shatte | 16 | 11 | 27 |
| Morozok | 17 | 19 | 36 |
| Obary | 18 | 15 | 33 |
| Katroon | 2 | 2 | 4 |
| Gaat | 3 | 2 | 5 |
| Chwerf | 1 | 0 | 1 |
| Beng | 0 | 1 | 1 |
| Jfra | 0 | 1 | 1 |
| Total | 206 | 219 | 425 |

**Table 7: Geographic distribution of various Types of CHD.**

| Residence | N | % |
|---|---|---|
| Sebha | 317 | 74.6 |
| Shatte | 27 | 6.4 |
| Morozok | 36 | 8.5 |
| Obary | 33 | 7.8 |
| Katroon | 4 | 0.9 |
| Gaat | 5 | 1.2 |
| Chwerf | 1 | 0.2 |
| Beng | 1 | 0.2 |
| Jfra | 1 | 0.2 |
| Total | 425 | 100 |

**References**:


[1]- Richard E Behman Vector C Vanghan: Nelson textbook of pediatrics, disease of the cardiovascular system, 17 th Ed. Saunders comp. 2004; 1499-150.

[2]- Al-jumaily SA: pattern of congenital heart disease in Iraqi children, a thesis submitted to Iraqi commission for medical specialization, 1991, Battie RM: Essential Revision note a pediatrics 1th Ed. 2002 chapter one: 14.

[3]- Saad S AL-Ani: pattern of congenital heart disease in Ramadi hospital.Al-Anbar UnivJ No 11996; 50-5.

[4]- Jose VJ, Gomathi M. Declining prevalence of rheumatic heart disease in rural school children in India: 2001-2002. *Indian Heart J* 2003; 55:158-60.

[5]- Billett J, Majeed A, Gatzoulis M, et al. Trends in hospital admissions, in-hospital case fatality and population mortality from congenital heart disease in England, 1994 to 2004. *Heart* 2008;94(3):342–8.

[6]- Fixler DE, Pastor P, Chamberlin M, et al. Trends in congenital heart disease in Dallas County births. 1971-1984. *Circulation* 1990;81(1): 137-42.

[7]- Vaidyanathan B, Kumar RK. The global burden of congenital heart disease. *Congen Cardiol Today* 2005;3:1-8.

[8]- Ferencz C, Rubin JD, McCarter RJ, et al. Congenital heart disease: prevalence at live birth. The Baltimore-Washington Infant Study. *Am J Epidemiol* 1985;121(1):31-6.

[9]- Bolisetty S, Daftary A, Ewald D, et al. Congenital heart defects in Central Australia. *Med J* 2004; 180(12):614-7.

[10]- Storch TG, Mannick EE. Epidemiology of congenital heart disease in Louisiana: an association between race and sex and the prevalence of specific cardiac malfor-mations. *Teratology* 1992;46(3):271-6.

[11]- Alabdulgader AAA. Congenital heart dis-ease in 740 subjects: epidemiological aspects. *Ann Trop Paediatr* 2001;21 (2): 111–8.

[12]- Rahim F, Ebadi A, Saki G, et al. Prevalence of congenital heart disease in iran: a clinical study. *J Med Sci* 2008;8(6):547-52.

[13]- Begić H, Latifagić A, Tahirović H, et al. Epidemiological and clinical aspects of congenital heart disease in children in Tuzla Canton, Bosnia-Herzegovina. *Eur J Pediatr* 2003; 162(3):191-3.

[14]- Rodríguez Dehli C, Ariza Hevia F, Riaño Galán I, et al. The epidemiology of congenital heart disease in Asturias (Spain) during the period 1990-2004. *An Pediatr (Barc)* 2009;71(6):502- 9. [In Spanish].

[15]- Tanner K, Sabrine N, Wren C. Cardiovas-cular malformations among preterm infants. Pediatrics 2005;116( 6):e833-e838.

[16]- Bache A, Garne E. Congenital heart defects in the county of Fyn. Epidemiology and mortality 1986-1995. *Ugeskr Laeger* 2002; 164(36): 4169-72. [In Danish]

[17]- Schoetzau A, van Santen F, Sauer U, et al. Cardiovascular abnormalities in Bavaria 1984- 1991. *Z Kardiol* 1997; 86(7):496-504. [Article in German].

[18]- Subramanyan R, Joy J, Venugopalan P, et al. Incidence and spectrum of congenital heart disease in Oman. *Ann Trop Paediatr* 2000; 20(4):337-41.

[19]- Bassili A, Mokhtar SA, Dabous NI, et al. Congenital heart disease among school children in Alexandria, Egypt: an overview on prevalence and relative frequencies. *J Trop Pediatr* 2000;46(6):357-62.

[20]- El Hag AI. Pattern of congenital heart disease in Sudanese children. *East Afr Med J* 1994 ;71(9): 580-6.

[21]- Pongpanich B, Dhanavaravibul S, Limsuwan A. Prevalence of heart disease in school children in Thailand: a preliminary survey at Bang Pa-in. *Southeast Asian J Trop Med Public Health* 1976; 1:91-4.

[22]- Hassan I, Haleem A, Bhutta Z. Profile and risk factors for congenital heart disease. *J Pak Med Assoc* 1997;47(3):78-81.

[23]- Squarcia U, Agnetti A, Carano N, et al. Epidemiological study of congenital heart diseases in the province of Parma during the years 1972-80. *Pediatr Med Chir* 1981;3(6): 459-62.

[24]- Manetti A, Pollini I, Cecchi F, et al. The epidemiology of cardiovascular malformations. III. The prevalence and follow-up of 46,895 live births at the Careggi Maternity Hospital, Florence, in 1975-1984. *Ital Cardiol* 1993; 23(2):145-52.

[25]- Robida A, Folger GM, Hajar HA. Incidence of congenital heart disease in Qatari children. *Int J Cardiol* 1997; 60 (1):19-22.

[26]- Stephensen SS, Sigfusson G, Eiriksson H, et al. Congenital cardiac malformations in Iceland from 1990. *Cardiol Young* 2004; 14(4):396-401.

[27]- Kapoor R, Gupta S. Prevalence of con-genital heart disease, Kanpur, India. *Indian Pediatrics* 2008;45(4):309-11.