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Research Article

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# Prevalence of Congenital Heart Defects among Neonates in Port Harcourt, Rivers State, Nigeria

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## Abstract

### Introduction

Congenital Heart Defects (CHDs) are structural abnormalities of the heart and intra-thoracic great vessels that are present at birth and may be of functional significance. They are the most frequently occurring congenital anomalies and babies born with severe forms of these defects are likely to die in the neonatal period.

### Objectives

The aim of this study was to determine the prevalence of CHDs among neonates delivered in Port Harcourt, Rivers State, Nigeria

### Methods

Using a stratified sampling technique, 530 neonates were selected from three hospitals in Port Harcourt. The biodata of the parents and socio-demographic information were obtained through an interviewer-administered questionnaire to the mothers. Physical examination and echocardiograghy were performed on all the neonates.

### Results

Five hundred and thirty (530) neonates aged 0-7days (5.2±1.8) participated in this study and the male to female ratio was 1.1:1. Forty-three neonates were found to have CHD giving a prevalence of 8.1% [95%CI: 6.0-10.86]. Thirty-nine were acyanotic and four cyanotic. Congenital Heart Defects were found in 21 (48.8%) males and 22 (51.2%) females. The more common heart defects were isolated Atrial Septal Defect in 16(37.2%), isolated Patent Ductus Arteriosus in 11(25.6%) and isolated Ventricular Septal Defect in 6 (13.9%). The most common cyanotic CHD was Transposition of the Great Arteries in 2 neonates (4.7%). The clinical features identified in

neonates with CHD were tachypnoea, dysmorphia, cyanosis, hypoxia and murmur

## Conclusion

The prevalence of CHD is considerably high in Port Harcourt and further studies need to be carried out to ascertain the risk factors.

## Introduction

Congenital heart defects (CHD) are gross structural abnormalities of the heart or intra-thoracic great vessels that are present at birth and may be of functional significance.[1] This definition does not include cardiomyopathies, cardiac arrhythmias or functionless vascular disorders like persistent left superior vena cava.[2] Congenital heart defects are the most common forms of major birth defects and account for a third of all major congenital abnormalities in the world.[3] Babies born with severe forms of these defects are twelve times more likely to die in the first year of life especially if the defect is missed in the neonatal period.[4]

Worldwide, the estimated incidence of CHD is 8 per 1000 live births.[5] The incidence of CHD has increased steadily from 4-5/1000 in the 1950s to as much as 50-75/1000 in more recent times.[2] This increment could be attributed to newer diagnostic techniques, more expertise in the field of Pediatric Cardiology, varying methodologies applied in these studies or an actual increase in the prevalence of CHD.

In Nigeria, much work has been done on CHD[9-13] but a few were among neonates. Studies done in the neonatal period were focused on creating a normogram for left and right ventricular dimensions.[6,7] Some studies in the post-neonatal period were retrospective,[8,9] while others were prospective and based on clinical assessment without the use of echocardiography.[10,11] Other studies using echocardiography were hospital based and included few neonates.[12,13] Some of the studies were also school based and found mainly ASDSs to be the most common.[14] The prevalence obtained may be an underestimation of the burden of disease as majority of the children with severe CHD do not usually survive beyond 5 years, and would have died before school age.

Echocardiography remains the gold standard for the detection of CHD as it is highly sensitive and specific.[15] Congenital heart defects can be diagnosed in utero as early as the second trimester in at-risk fetuses using fetal echocardiography[16] but this is not not readily available in many Nigerian centres. Transthoracic echocardiography (TTE) is non-invasive and the most widely used in the neonatal period and offers the closest opportunity to estimating the true prevalence of CHD among neonates in Nigeria.

The city of Port Harcourt, situated in the Niger Delta region is known for its oil exploration activities and due to weak environmental laws is highly polluted.[17] Gas flaring poses a potential threat to inhabitants of Port Harcourt as maternal exposure to these chemicals (hydrocarbons, organic solvents, air pollutants) especially in the first trimester is a potential risk factor for CHD.[18,19] This study was structured to identify babies with CHD in the first week of life using a transthoracic echocardiograph.

## Subjects and Methods

This cross-sectional study was carried out among neonates delivered in Port Harcourt, Rivers State. It was done over a five-month period in three health facilities in Port Harcourt from November 1st 2019 to March 30th 2020. Port Harcourt is the capital and largest city of Rivers State Nigeria. It is located in the Niger Delta region and is a major oil-producing city and is home to the first oil refinery in Nigeria.[20] It has two tertiary hospitals, the University of Port Harcourt Teaching Hospital (UPTH) and River State University Teaching Hospital, three secondary health centres and 27 Primary Health Centres. The Government health facilities in Port Harcourt City were stratified according to the type of facility (Primary, Secondary and Tertiary Hospitals) and a facility was selected from each of these strata by simple random sampling. Based on the estimated number of monthly deliveries per hospital, eligible neonates were recruited using proportionate to size allocation. The inclusion

criteria for the study population consisted of neonates ≥28 weeks of gestation aged 0-7 days and delivered in the selected hospitals in Port Harcourt. Preterm neonates with solitary Patent Ductus Arteriosus and all neonates with an isolated Patent Foramen Ovale were excluded. The study was carried out in the neonatal and immunization units of University of Port Harcourt Teaching Hospital, Obio Cottage Hospital and Primary Health Centre Rumuigbo.

## Ethical Considerations

Ethical clearance for the study was obtained from the Research and Ethics Committee of the University of Port Harcourt Teaching Hospital (UPTH) before the commencement of the study. Permission was obtained from the Management of Obio Cottage Hospital and the Permanent Secretary of the Rivers State Primary Health Care Management Board. A written informed consent was obtained from the parents of the neonates selected for this study and the information retrieved in this study was kept confidential. All neonates with congenital heart defects were referred for management and long term follow up in the Paediatric Cardiology Unit of the University of Port Harcourt Teaching Hospital. The cost of the echocardiography was borne by the researcher.

## Data Collection

The three selected hospitals were visited before the start of the study and the purpose and scope of the study were explained to the Chief Medical Directors, Heads of the neonatal units and Matrons at the immunization centres. On each day of the study, the researcher first gave a talk about the study to mothers and caregivers at the immunization centres and neonatal units. Data was collected using pretested interviewer-administered questionnaires. All the babies recruited into the study had a physical examination and echocardiodiograph done. A portable SONOSITE MICRO MAXX transthoracic echocardiograph machine with an 8-4MHz transducer was used and echocardiography was done by the researcher according to the American Society of Echocardiography Guidelines for performance of pediatric echocardiography.[21]

The echocardiograph clips were stored and reviewed by a consultant cardiologist. A repeat echocardiograph was done at 6 weeks for all term neonates with isolated PDA and those with persistent patent ductus arteriosus were classified to have CHD. Neonates with ASD <4mm with no hemodynamic compromise were classified as having structurally normal hearts. Neonates with CHD were referred to the Pediatric Cardiologist at UPTH for follow up. The data collected were entered and analyzed using the Statistical Package for Social Sciences (SPSS) software version 25 and p-value of <0.05 was considered statistically significant.

## Results

*General Characteristics of the Subjects*

There were 530 neonates included in this study. The neonates were between 0-7days old with a mean age of 5.2 ±1.8 days. Of the 530 neonates studied, 282 (53.2%) were males and 248 (46.8%) were females giving a male to female ratio of 1.1:1. The mean birth weight of the babies was 3.37±0.58kg. Twenty-one (4.0%) of the babies were products of multiple gestation. Table I shows the biodata of the study population.

*Socio-Demographic Characteristics of the Parents*

Table II shows the socio-demographic characteristics of the parents. Three hundred and eighty-one (71.9%) of the parents were married for 1-5 years. Twenty five (4.7%) of neonates had a family history suggestive of CHD (early neonatal deaths -18, sibling living with CHD- 4, stillbirths-3).

*Mode of Conception, Antenatal Supervision, Place and Mode of Delivery*

Table III shows the mode of conception, antenatal care, place and mode of delivery. Ten of the neonates were conceived via assisted reproductive technology, while the other 520 were conceived naturally. In 258 mothers (48.7%), the pregnancy was supervised in a secondary health facility and spontaneous vertex was the predominant mode of delivery (62.3%).

Table 1. Biodata of the study population

| Variables | n= (530) | % |
|---|---|---|
| **Gender** | | |
| Male | 282 | 53.2 |
| Female | 248 | 46.8 |
| **Birth Order** | | |
| First Child | 203 | 38.3 |
| Second Child | 153 | 28.9 |
| Others | 174 | 32.8 |
| **Gestational age status** | | |
| Term | 493 | 93 |
| Preterm | 37 | 7 |
| **Birth weight Classification** | | |
| NBW | 432 | 81.5 |
| Macrosomia | 54 | 10.2 |
| LBW | 44 | 8.3 |
| **Gestational Plurality** | | |
| Singleton | 509 | 96 |
| Multiple | 21 | 4 |

NBW-Normal Birth Weight, LBW-Low Birth Weight

Table 2. Socio-demographic Characteristics of the parents

| Variable | n= (530) | % |
|---|---|---|
| **Parent's Marriage Duration** | | |
| 1-5 | 381 | 71.9 |
| 6-10 | 128 | 24.1 |
| > 10 | 21 | 4 |
| **Socio-economic Class** | | |
| Lower | 344 | 64.9 |
| Middle | 154 | 29.1 |
| Upper | 32 | 6 |
| **Maternal Age (years)** | | |
| ≤34 | 421 | 79.4 |
| ≥35 | 109 | 20.6 |
| **Paternal Age (years)** | | |
| ≤34 | 149 | 28.1 |
| ≥35 | 381 | 71.9 |
| **Family History of CHD** | | |
| No | 505 | 95.3 |
| Yes | 25 | 4.7 |

Figure 1 illustrates that out of the 530 neonates who had echocardiographic evaluation for CHD, 47 had abnormal echo findings; of these, 43 (8.1%) [95%CI: 6.0-10.86] had CHD and 4 had other echocardiographic abnormalities other than CHD (three patients had hypertrophic cardiomyopathy and one had persistent pulmonary hypertension of the newborn). The prevalence of CHD was 8.1% [95%CI: 6.0-10.86] i.e. 81 per 1000 live births. Thirty-nine of the neonates had acyanotic CHD and four had cyanotic CHD. Congenital heart defect was found to be present in 21 (48.8%) males and 22 (51.2%) females giving a male to female ratio of 1:1 and this was not statistically significant.

*The Frequency and Pattern of CHD among the Study Population*

Table IV shows the pattern of CHD in the study population. All the 16 neonates with ASD had the ostium secundum type and the sizes ranged between 4-8mm. Forty-four term neonates had a solitary PDA at birth, of which 33 (75%) closed spontaneously by 6 weeks and 11 persisted. The sizes of the PDAs ranged from 2-4 mm. Four neonates had peri-membranous VSDs while 2 had mus cular VSDs and the sizes of the VSDs ranged from 3-6mm. Cyanotic CHDs were found in 4 neonates. Two had TGA and the other two neonates had a TOF and tricuspid atresia respectively.

*Congenital Heart Defects Associated with Clinical Features*

Out of the 43 neonates with a CHD, 20 (3.7% or 37/1000) of them had one or more clinical features while 23 were asymptomatic. They were eight neonates with dysmorphic features and CHD. Six of the neonates had facial and limb anomalies associated with Down's syndrome (low-set ears, upward slanting palpebral fissures, flat nasal bridge, ocular hypertelorism, sandal gap). The neonates with Down's syndrome had 2 AVSD, one each of TA, VSD, PDA and ASD/PDA respectively. The other dysmorphic neonates with achondroplasia and myelomenigocele each had a PDA.

*The Relationship between Clinical Features and CHD in the Study Population*

Table VI presents the relationship between clinical features and CHD in the study population. A total of 11 newborns had dysmorphic features and they include Down syndrome(7), achondroplasia(1), spinal bifida cystica(1), cryptochidism(2). All the clinical features were significantly associated with CHD except for tachycardia. Out of the seven cyanotic neonates, five had CHD while two had severe respiratory tract infections. Two of the neonates with grade 2 systolic murmurs had structurally normal hearts.

## Discussion

The prevalence of CHD among neonates in Port Harcourt City was 8.1%. The prevalence is much higher than previously reported worldwide among neonates (7.8-75 per 1000 live births).[22,23,24,25,26]The cause of the high prevalence is probably due to the following reasons; Firstly many PDAs, small VSDs and ASDs which may have closed spontaneously early were included due to the timing of the study. It has been shown that performing echocardiography on all neonates has a tendency of picking up many ASDs, VSDs and PDAs that may close spontaneously by one year and this was demonstrated by Ooshima et al.[27] The importance of including all these lesions is that it gives a better picture and understanding of the burden of disease and its aetiology. Also complex lesions such as TGA, TA which otherwise may have resulted in early neonatal deaths were included.

Secondly, the difference in the study population and methodology could also affect the prevalence. Studies in which echocardiography was done on symptomatic and asymptomatic neonates gave a high prevalence.[22,27] If only the 20 symptomatic neonates were analyzed, the prevalence of CHD in this study would have been 3.7% (37/1000). This implies that about half of the CHDs would have been missed and the asymptomatic 23 neonates with CHD would otherwise be labeled as having structurally normal hearts. This is similar to a finding noted in Japan where Ishikawa et al[22] obtained a prevalence of 50.3/1000

Table 3. Mode of conception, antenatal care, place and mode of delivery

| Variables | n= (530) | % |
|---|---|---|
| **Mode of conception** | | |
| Natural conception | 520 | 98 |
| Assisted Reproductive Technology | 10 | 2 |
| **Place of antenatal care** | | |
| Primary Health Centre | 127 | 24 |
| Secondary Health Centre | 258 | 48.7 |
| Tertiary Centre | 37 | 7 |
| Traditional Birth Attendant place | 58 | 10.9 |
| Private Hospital | 50 | 9.4 |
| **Place of Delivery** | | |
| Primary Health Centre | 132 | 24.9 |
| Secondary Health Centre | 303 | 57.2 |
| Tertiary Centre | 95 | 17.9 |
| **Mode of Delivery** | | |
| Spontaneous vertex delivery | 330 | 62.3 |
| Caesarean Section | 200 | 37.7 |

Table 4. The Frequency and pattern of CHD among the study population

| CHD | Frequency | % |
|---|---|---|
| Isolated ASD | 16 | 37.2 |
| Solitary PDA | 11 | 25.6 |
| Isolated VSD | 6 | 13.9 |
| ASD/PDA | 4 | 9.3 |
| AVSD | 2 | 4.7 |
| TGA | 2 | 4.7 |
| TA | 1 | 2.3 |
| TOF | 1 | 2.3 |
| **Total** | 43 | 100 |

ASD-Atrial Septal Defect , PDA-Patent Ductus Arteriosus , VSD-Ventricular septal defect, AVSD-Atrio-ventricular Septal Defect , TGA-Transposition of Great Arteries , TA-Tricuspid Atresia, TOF-Tetralogy of Fallot

Table 5. Congenital Heart Defects associated with Clinical Features

| | CHD | CLINICAL FEATURE (S) |
|---|---|---|
| 1 | ASD | Tachypnea |
| 2 | ASD | Murmur |
| 3 | ASD | Hypoxia |
| 4 | ASD | Hypoxia/ Murmur |
| 5 | VSD | Murmur |
| 6 | VSD | Dysmorphia/ Murmur |
| 7 | VSD | Hypoxia/ Murmur |
| 8 | VSD | Tachypnea/ Murmur |
| 9 | PDA | Dysmorphia/Hypoxia/ Murmur |
| 10 | PDA | Dysmorphia/Hypoxia/ Murmur |
| 11 | PDA | Dysmorphia/Hypoxia |
| 12 | PDA | Hypoxia/Cyanosis/Murmur |
| 13 | PDA | Hypoxia/Murmur |
| 14 | ASD/PDA | Dysmorphia/ Murmur |
| 15 | AVSD | Dysmorphia/ Tachypnea/Hypoxia /Murmur |
| 16 | AVSD | Dysmorphia/ Tachypnea/Murmur |
| 17 | TGA | Cyanosis/Hypoxia/Tachypnea/Tachycadia /Murmur |
| 18 | TGA | Cyanosis/Hypoxia/Tachypnea/Tachycadia /Murmur |
| 19 | TOF | Cyanosis/Hypoxia/ Murmur |
| 20 | TA | Dysmorphia/Cyanosis/Hypoxia/Tachypnea/ Murmur |

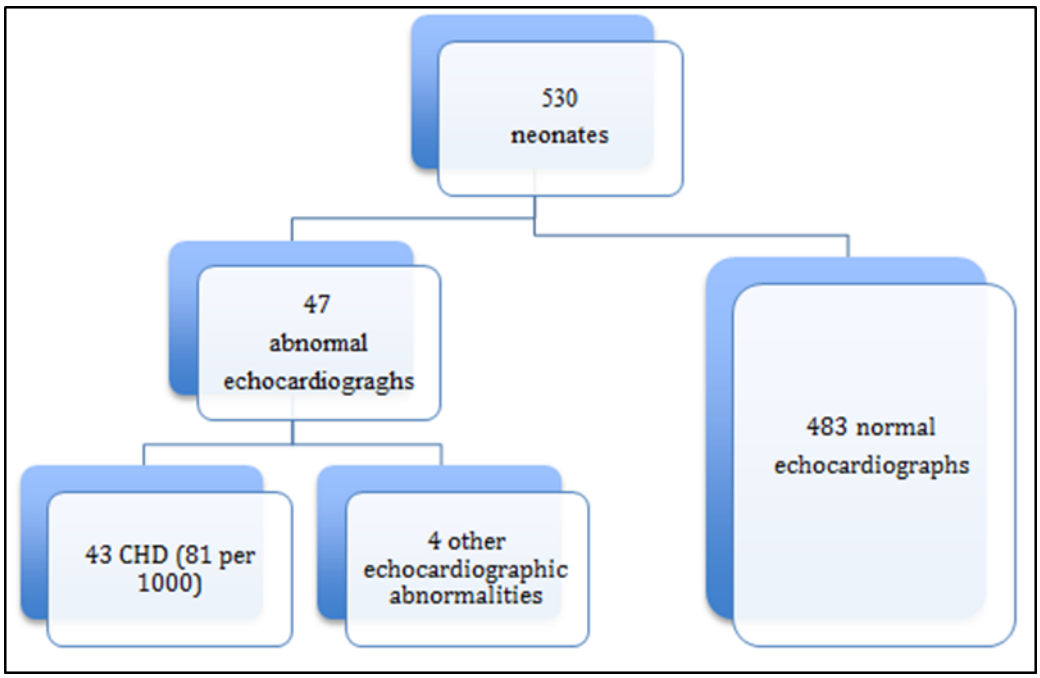


Figure 1. Prevalence of Congenital Heart Defect in the study population

Table 6. The relationship between Clinical features and CHD in the study population.

| | **CHD Status** | | | | |
|---|---|---|---|---|---|
| | **Present** | **Absent** | **Total** | **Chi-square** | |
| **Variable** | **n (%)** | **n (%)** | **n (%)** | | ***p-value*** |
| **Dysmorphic Features** | | | | | |
| Yes | 8 (72.7) | 3 (27.3) | 11 (100.0) | | |
| No | 35(6.7) | 484(93.3) | 519 (100.0) | | |
| **Total** | 43 (8.1) | 487 (91.9) | 530 (100.0) | 62.909 | <.0.001#* |
| **Tachypnoea** | | | | | |
| Yes | 7 (25.0) | 21(75.0) | 28(100.0) | | |
| No | 36 (7.2) | 466 (92.8) | 502 (100.0) | | |
| **Total** | 43 (8.1) | 487 (91.9) | 530 (100.0) | 11.308 | 0.005* |
| | | | | | |
| **Cyanosis** | | | | | |
| Yes | 5 (71.4) | 2 (28.6) | 7 (100.0) | | |
| No | 38 (7.3) | 485(92.7) | 523 (100.0) | | |
| **Total** | 43 (8.1) | 487 (91.9) | 530 (100.0) | 38.146 | <0.001#* |
| | | | | | |
| **Tachycardia** | | | | | |
| Yes | 2(5.6) | 34 (94.4) | 36 (100.0) | | |
| No | 41(8.3) | 453(91.7) | 494(100.0) | | |
| **Total** | 43 (8.1) | 487 (91.9) | 530 (100.0) | 0.339 | .758# |
| Hypoxia | | | | | |
| Yes | 78(85.7) | 13 (14.3) | 91(100.0) | | |
| No | 30(6.8) | 409 (93.2) | 439 (100.0) | | |
| Total | 43 (8.1) | 487 (91.9) | 530 (100) | 5.615 | 0.032* |
| Cardiac Murmur | | | | | |
| Yes | 17(89.5) | 2 (10.5) | 19(100.0) | | |
| No | 26(5.1) | 485 (94.9) | 511 (100.0) | | |
| Total | 43 (8.1) | 487 (91.9) | 530 (100) | 174.981 | <0.001#* |

live-births when all neonates were analyzed but 21.3 per 1000 live births when only symptomatic babies where used to compute the prevalence.

The prevalence of CHD in this study is much higher than that noted in Bangladesh[25] (7.8/1000) and Germany[28] (10.8/1000) and this is probably because, in those studies, echocardiography was only done among neonates with clinical features suggestive of CHD. School based studies in which only symptomatic babies have an echocardiography tend to give a much lower prevalence because children with critical CHD may die before school age, some lesions may remain asymptomatic and many of the defects may close spontaneously before the study.[14,29]

Furthermore, the high prevalence in this study could be due to the high amount of air and land pollution in the oil-producing city of Port Harcourt.[17,30,31] Port Harcourt is situated in the Niger Delta region and has some of the world's largest oil and gas producing companies in it and has also experienced some of the world's worst oil spills.[32,33] Studies have shown that people who live in oil exploration communities may suffer adverse effects due to the toxic waste products that are emitted from these facilities. [17,34,35,36]

Oil spillage and gas flaring are off shoots of oil exploration and have been documented to probably have adverse effects on the inhabitants of Port Harcourt. [37] Ordinioha and Brisibe[17] in 2013 estimated that an average of 240,000 barrels of crude oil are spilled on a yearly basis in the Niger Delta and these oils contaminate the groundwater, surface water, air and crops with hydrocarbons.[34] These toxic chemicals may cause a genetic mutation in foetuses during the period of organogenesis and lead to various congenital anomalies. In a 2014 study by Otaigbe and Tabansi[12] in the Niger Delta, the prevalence was as high as 14.4 per 1000 live births even though only 9% of the study population were neonates Port Harcourt has become more polluted in the last few years with the increased amount of air, land and water pollution making this high prevalence tenable.[31,32] The findings of this study is also much higher than 3.5 per 1000 obtained by Gupta and Antia[10] in Ibadan. However the methodology was different, as no echocardiographic diagnosis was made and moreover, Ibadan is not an oil-producing city.

A preponderance of acyanotic defects (90.7%) as observed in this study has been noted in other studies done in the neonatal period.[22,23,27,38] This is probably because most neonates with cyanotic CHDs may have been spontaneous abortuses and this study was able to capture many asymptomatic and acyanotic lesions. Atrial Septal Defect was the most common defect and this is similar to reports in Iran[39] and China.[40] This is in contrast to other studies where VSD was found to be the commonest.[22,23,28] The reason for this difference is not known but it has been found that ASD is commoner in regions with a high level of particulate matter less than 10μm pollution[41,42] and studies have shown a high level of particulate matter pollution in Port Harcourt.[30,32] The most common cyanotic CHD was TGA and this was in keeping with a study done in India.[23] None of the neonates had aortic stenosis or coarctation of the aorta and this was in keeping with findings from other Nigerian authors[12,43] and this in contrast with reports from Asia. [22,23,27]

The findings that symptomatic neonates with CHD presented with murmur, cyanosis, hypoxia, tachypnoea and tachycardia have been demonstrated in other studies.[15,25,48] The reason for the absence of symptoms in the asymptomatic neonates may be the small size of the defect, absence of chamber enlargement or hemodynamic stability.[15] Even a large VSD may be associated with few symptoms in the 1st weeks of life due to high pulmonary pressure and pulmonary vascular resistance.[15] With a drop in pulmonary vascular resistance (PVR) after the first few weeks, the magnitude of the shunt increases and signs of pulmonary volume overload and congestive cardiac failure occur between 6 to 10 weeks.[44] Although dysmorphic facies is not always associated with CHD, more than two-third of the neonates in the study population with dysmorphic facies had CHD. It is similar to a report by Animashaun et al[45] where 73.5% of children with dysmorphic had CHD. This study buttresses the

importance of screening for CHD in patients with dysmorphic features.

## Conclusion

The prevalence of congenital heart defect among neonates in Port Harcourt City is 81 per 1000 live-births. There was an equal male to female preponderance of CHD and acyanotic CHDs were the most common. The clinical features associated with CHD were dysmorphia, cyanosis, tachypnoea, hypoxia and murmur. Atrial septal defect and patent ductus arteriosus were the more common forms of congenital heart defect among newborns in Port Harcourt.

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