



CHILDREN WITH SUSPECTED CONGENITAL HEART DISEASE - A REVIEW

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Abstract

Congenital heart disease (CHD) is the most common congenital cardiac defect in children. It is a significant cause to infant death and morbidity. This article provides an overview of the etiology and epidemiology of CHD. According to the WHO, Bangladesh has a 6% incidence of congenital heart disease. Nine frequent lesions make up 80% of congenital heart disease. The majority of the rare or complex lesions that make up the remaining 20% of congenital heart disease. The study aims to contribute to overview of CHD enhancing patient outcomes and informing public health policies. This review study employs a systematic review approach to compile and analyze existing research on children with suspected congenital heart disease. We looked at recent studies, and searched pub med, Cochrane database, cardiovascular disease journal. The data mainly involve a review of CHD from various studies of sample groups of different ethnicities; age groups and gender were evaluated. After the application of predefined inclusion/exclusion criteria, 50 studies were included in the systematic review. CHD impacts millions of women worldwide children suffer silently without knowing that there is treatment. CHD are revealed by epidemiology research which must recognize the value of routine medical exams, with the help of successful transition program. A thorough review of the most recent research on the pathophysiology, Incidences clinical symptoms, and therapy of CHD was conducted and optimum care of CHD is essential to enhance the quality of life and prevent the progression of the diseases.

Key words: Cardiac abnormality, Congenital heart disease, Epidemiology, Heart defects.

Introduction

The most prevalent congenital lesion and the most prevalent form of heart disease in children is congenital heart disease. It is a significant contributor to infant death and morbidity (Muntean et al. 2017, Wu et al. 2020). A gross anatomical anomaly of the heart or intrathoracic great vessels that is actually or potentially functionally significant is referred to as congenital heart disease (Mitchell et al. 1971, Chowdhury et al. 2022a,b). Congenital heart disease affects about 8 out of every 1000 live births, with stillbirth, spontaneous abortion, and prematurity having greater incidences (Fyler et al. 1980, Jordan and Scott 1989). The incidence of the illness has remained constant over the past three decades, indicating little progress in our understanding of the etiology of CHD, even while surgical and medicinal advancements have increased childhood survival in CHD from 20% in 1950 to >90% today (Muntean et al. 2017, Zimmerman et al. 2020, Wu et al. 2020). Indicating a continuing health inequality, mortality has also remained disproportionately higher in babies and at poorer socio-demographic indices. Additionally, some survivors report having a poor quality of life and having lifelong morbidity, such as developmental disabilities (Wernovsky and Licht 2016).

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According to the World Health Organization (WHO), Bangladesh has a 6% incidence of congenital heart disease, India has a 15% incidence, Burma has a 6% incidence, and Sri Lanka has a 10% incidence of all cardiovascular diseases (Malik 1976). This review offered an in-depth examination of congenital heart disease (CHD) in children, covering its epidemiology, causes, clinical manifestations, diagnostic techniques, treatment approaches, and prognosis. By compiling and analyzing recent knowledge and advancements, this review seeks to elevate awareness and understanding among healthcare providers, facilitate early diagnosis and treatment, and ultimately improve the health outcomes for children with CHD.

Nine frequent lesions make up 80% of congenital heart disease, while the relative frequency of the most common lesions changes depending on the source (Jackson et al. 1996). These include tetralogy of fallout (4%), pulmonary stenosis (9%), atrial septal defect (5%), patent ductus arteriosus (9%), coarctation of the aorta (5%), transposition of the great arteries (5%), and ventricular septal defect (36%). The majority of the rare or complex lesions that make up the remaining 20% of congenital heart disease were reported. (Bernstein et al. 2008). Early detection and timely intervention in children with suspected congenital heart disease improve clinical outcomes and reduce long-term complications. CHD is a frequent congenital defect that places a heavy financial and emotional strain on the entire family. However, the likelihood of long-term complications is lower and the outcome is better if the issues are identified earlier in life. A greater number of children with CHD are surviving into adolescence and adulthood as a result of improved medical and surgical care (Camm and Bunce 2005). With the exception of a few sporadic reports, the clinical pattern of CHD in Bangladeshi children is not well known (Sharmin et al. 2008). The study aims to contribute to the improved diagnosis, treatment, and management of congenital heart disease in children, ultimately enhancing patient outcomes and informing public health policies.

Methodology

This is based on a comprehensive literature search, looking into a recent meta-analysis, and searching PubMed, Cochrane database, Elsevier, and cardiovascular surgery journal. The data mainly involve a review of various studies of cardiovascular disease from sample groups of different ethnicities. We tried to get the studies, by searching for symptomatology and the epidemiology of cardiovascular disease. This review study employs a systematic review and meta-analysis approach to compile and analyze existing research on children with suspected congenital heart disease (CHD). The study follows the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines to ensure comprehensive and transparent reporting of the review process. Inclusion criteria: studies involving children (aged 0-18 years) suspected of having congenital heart disease. Studies that utilize non-invasive diagnostic tools such as echocardiography, electrocardiograms (ECGs), magnetic resonance imaging (MRI), and chest X-rays. Articles published in peer-reviewed journals in English. Studies reporting on the accuracy, sensitivity, and specificity of diagnostic tools, clinical outcomes, risk factors, and intervention strategies. Exclusion criteria: Studies involving adults with congenital heart disease. Case reports, editorials, and review articles. Studies not available in full text or in languages other than English

Data Sources and Search Strategy: A comprehensive literature search will be conducted using the following electronic databases: PubMed, MEDLINE and Cochrane Library. As this study is a review of previously published research, it does not involve primary data collection and does not require ethical approval. However, the review will adhere to ethical standards in research by ensuring accurate and unbiased

reporting of finding. Potential limitations of this study include the variability in study designs, diagnostic criteria, and outcome measures across the included studies, which may introduce heterogeneity and affect the generalizability of the findings. Congenital cardiac abnormalities can be categorized in a variety of ways; the following categories could be used to emphasize the underlying anatomy and pathophysiology like - (a) CHD with shunt between systemic and pulmonary circulation, (b) left heart CHD, (c) right heart CHD, (d) CHD with anomalous origin of great arteries, and (e) miscellanea (Micheletti 2019). Brief information on the basis of mentioned points are given below-

(a) CHD with shunt between systemic and pulmonary circulation

●Atrial Septal Defect:

A communication between the atrial chambers known as an atrial septal defect (ASD) allows for left-to-right shunting (Fig. 1). It is the second most widespread kind of CHD, affecting 7-10% of people overall. It is more common in women (2:1) and is most frequently detected in late childhood or adults. There are various forms of ASD was reported by Anderson et al. (2002).

Ostium secundum ASD: The most common type of ASD, accounting for 70-80% of all cases, is caused by a defect in the flap valve of the oval fossa, which can manifest as multiple fenestrations, incomplete development, unsuccessful overlapping to the septum secundum, or even total absence. In 10% of instances, abnormal pulmonary venous return is evident.

Ostium primum ASD: A component of the atrioventricular septal defect that is present in 15% of all ASDs and is situated close to the crux.

Sinus venosus defect: While functional closure occurs in 5-10% of infants near the superior and inferior vena cava (SVC) or inferior and posterior to the inferior vena cava (IVC) entries, complete anatomic closure occurs in 70-75% of adults (Schneider et al. 1996). Due to paradoxical emboli, the patent foramen ovale (PFO), which refers to the postnatal maintenance of this normal communication, takes clinical importance in some individuals with cerebral vascular accident and CHD (Furlan et al. 2012).

Diagnosis: From an early age, the second heart sound is widely split and fixed and is accompanied by a low-pitched systolic ejection murmur. The preferred diagnostic test is TTE. To determine pulmonary vascular resistance and evaluate pulmonary circulation vasoreactivity in the case of PH, diagnostic cardiac catheterization is required (Micheletti 2019).

Treatment: The rationale for ASD closure is the existence of a hemodynamically substantial L-R shunt (a pulmonary blood flow to systemic blood flow ratio of >1.5:1.0) and/or right chamber volume overload without considerable PH (pulmonary hypertension). Surgery or, in the event of secundum ASD and acceptable anatomic rims, device placement during interventional catheterization can be used to achieve closure (Butera et al. 2008).

Outcome: The most frequent late problem is atrial arrhythmia, and the risk gets up as child get older after the treatment. Although uncommon in patients who underwent surgery before the age of 25, PH is another late complication that can affect survival, and the risk rises with advancing age at repair as well (Hoffman et al. 2004). Performing closure during adolescence or childhood results in a return to normal life expectancy (Roos-Hesselink et al. 2003).

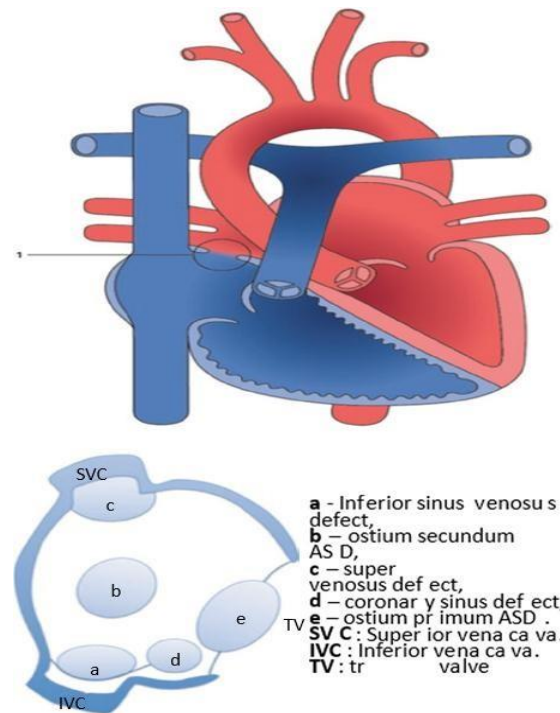


Fig. 1. Atrial septal defect and types (<https://perfusfind.com/en/eisenmenger-syndrome-with-patent-ductus-arteriosus-pda/>).

A communication between the two ventricles is known as a VSD-ventricular septal defect (Fig. 2). It accounts for about 20% of all defects and is the most prevalent CHD. It can happen alone or as a component of a complicated cardiac abnormality (Hoffman et al. 2004). Different types of VSD can be categorized based on the anatomical position of the conduction system. Membranous septal aneurysms are frequently related and may eventually lead to full or partial closure.

Muscular VSD: About 15-18% of all VSDs is completely covered by muscle and can be located within any IVS muscular component. Small defects frequently close on their own; rarely, multiple defects the so-called "Swiss-cheese" septum can be present.

Doubly committed, Juxta-arterial VSD: It occurs more frequently in Asian nations and is confined to the area directly below the artery valves, which are consequently in continuous fibrous tissue.

Inlet defect: frequently seen in people with down syndrome; situated in the inflow part of the septum, immediately inferior to the atrioventricular valve system.

The extent of the defect, ventricular systolic and diastolic performance, the existence of right ventricular outflow tract obstruction (RVOT), and PVR all affect the direction and size of the shunt.

Diagnosis: It may take a few weeks after birth when the shunt reaches its peak before the typical pansystolic murmur can be heard, which is best audible at the left mid to lower sternal border. In general, the

murmur strength increases as the defect size decreases. The presence of a second sound that is physiologically separated from respiration indicates appropriate pulmonary pressure. Variable levels of cyanosis may be seen in POVD connected to VSD. The amount of the L-R shunt is closely correlated with the degree of cardiomegaly and the rise in pulmonary vascular markings on a chest x-ray. TTE is required to evaluate every characteristic, including position, size, number, relationship to other congenital defects or aortic valve dysfunction, direction of the shunt, connection to the tricuspid valve and its cordal apparatus, presence of aneurysmal tissue, and distance between the VSD and aortic valve (Simpson and Miller 2011). To determine PVR and determine the vasoreactivity of the pulmonary circulation, a diagnostic cardiac catheterization may be necessary in some carefully chosen cases.

Treatment: Small, restrictive VSDs may close on their own. To avoid difficulties in the future, moderate to large VSDs are often closed through open heart surgery during infancy (Nygren et al. 2005). Although surgery is still the preferred course of treatment due to its low operative mortality, transcatheter closure is now a viable alternative that should only be used in certain situations involving either children or adults (Carminati et al. 2005).

Outcome: A patient with a successful surgically corrected VSD has a great long-term outlook and a life expectancy comparable to that of the general population (Roos-Hesselink et al. 2004). After surgical or transcatheter closure of peri-membranous VSD, acute and/or late-onset complete atrioventricular block (cAVB) caused by conduction system injury is still a serious concern; the incidence of cAVB after device closure that necessitates permanent pacemaker implantation is 2.6%, and the risk is higher in children under the age of six (Holzer et al. 2006, Butera et al. 2007, Carminati et al. 2007).

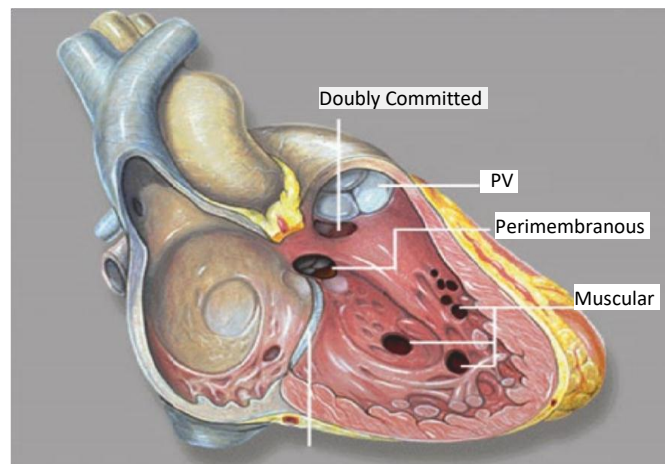


Fig. 2: Different types of VSD according to anatomical position within the septum viewed from RV side, TV tricuspid valve, PV pulmonary valve (<https://www.londoncardiovascularclinic.co.uk/cardiology-info/symptoms-diagnosis/atrial-septal-defect-asd>).

Atrioventricular Septal Defect (AVSDs)

A spectrum of cardiac malformations known as atrioventricular septal defects are characterized by aberrant development of the atrioventricular junction, which typically results from endocardial cushion tissue (Fig. 3).

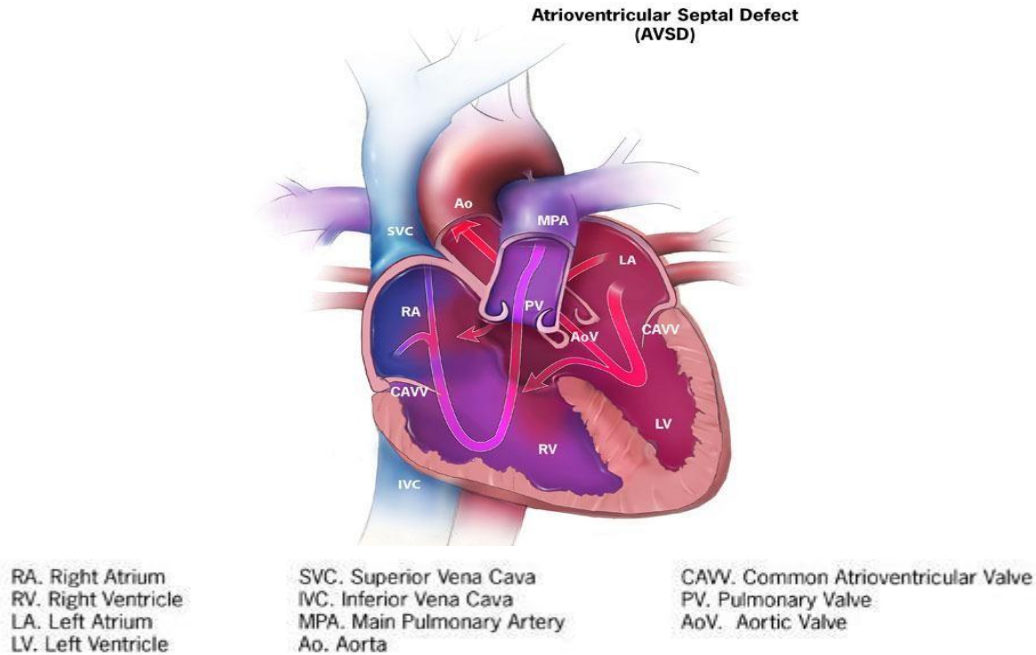


Fig. 3. Atrio-ventricular septal defect (https://en.wikipedia.org/wiki/Dextro-Transposition_of_the_great_arteries)

Diagnosis: It causes a superior and rightward QRS axis deviation on the electrocardiogram that is commonly accompanied by an extended PR interval and QRS duration. Cardiomegaly and increased pulmonary vascular markings on a chest x-ray indicate significant left-to-right intracardiac shunting and/or left AV valve regurgitation; Eisenmenger syndrome is indicated by the presence of prominent central pulmonary arteries along with peripheral "pruning" and an enlarged right heart. To rule out imbalanced ventricles, outflow tract blockages, and related lesions, a thorough and sequential 2D and 3D TTE should be performed (Hlavacek et al. 2006).

Treatment: Although the use of afterload reduction with ACE inhibitors is still debatable, complete AVSD typically requires medical management with diuretics and ACE inhibitors while waiting for surgical repair. Failure to thrive in the majority of infants is a sign that a repair is necessary, which is often done within the first three to six months. After diagnostic catheterization, patients with unrepaired AVSD and Eisenmenger syndrome respond to pulmonary vasodilator treatment (Micheletti 2019). Excellent short-term survival following AVSD repair is currently the norm. The most frequent causes of late morbidity and mortality in postoperative AVSD patients who require reoperation are heart block, subaortic stenosis, persistent septal abnormalities, and failure of the left AV valve (mostly regurgitation) (Najm et al. 1997).

Patent Ductus Arteriosus

The descending aorta and the roof of the pulmonary arterial trunk are connected by a vascular structure called the patent ductus arteriosus (PDA), which is located close to the origin of the left pulmonary artery (Fig. 4).

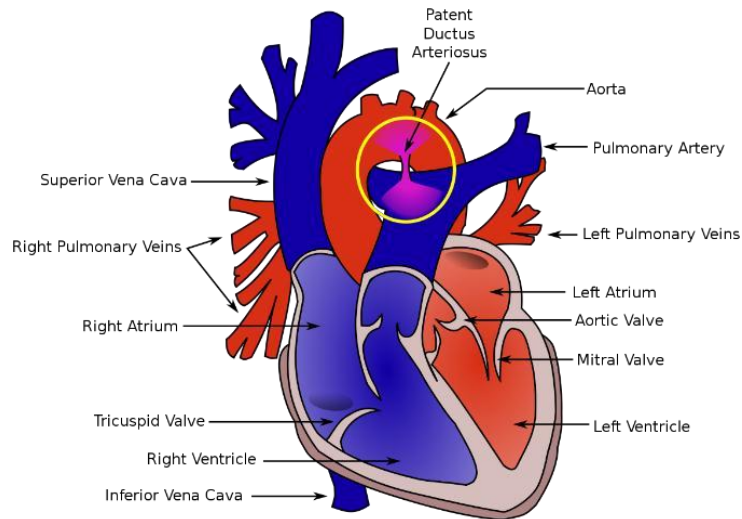


Fig. 4. Patent ductus arteriosus (https://commons.wikimedia.org/wiki/File:Patent_ductus_arteriosus.sv).

Diagnosis: A normal ECG or left ventricular hypertrophy (LVH) is seen with small to moderate PDA. Biventricular hypertrophy is seen with large PDA and pulmonary hypertension. Classically, chest x-ray reveals enlarged cardiac silhouette when the relative pulmonary-to-systemic flow ratio is $>2:1$; cardiomegaly occurs with the enlargement of left atrium (LA) and left ventricle and ascending aorta. Pulmonary vascular markings are increased. TTE evaluation gives accurate information about ductal anatomy and physiology and is practically feasible in almost all infants, in children, and in many adults (Micheletti 2019).

Treatment: Even asymptomatic patients with LV volume excess should have their PDA closed, as should patients with PAH whose PAP or PVR are less than two thirds of their systemic pressure or two thirds of their systemic vascular resistance, respectively. Avoid closure in PDA-Eisenmenger and "silent" ducts, which are very small and without a noise. Percutaneous closure is an option for patients who meet certain requirements for body weight and ductal size; it is practical and safe for kids above 5 kg with a PDA diameter of 2.5- 3.0 mm. This technique is still debatable for newborns of lower birth weight although thoracotomy surgery is now the best option for treating bigger PDAs (Backes et al. 2016, Schwartz et al. 2016).

(b) Left-Heart Congenital Heart Disease

Cor Triatriatum Sinister

A fibrous membrane separates the left atrium into two chambers in cor triatriatum sinister (CTS), a rare congenital anomaly that affects the mitral valve and left atrial appendage. The proximal chamber gets the

pulmonary venous drainage. CTS can happen alone (classic) or together with other congenital heart defects (atypical), like an ASD (Fig. 5).

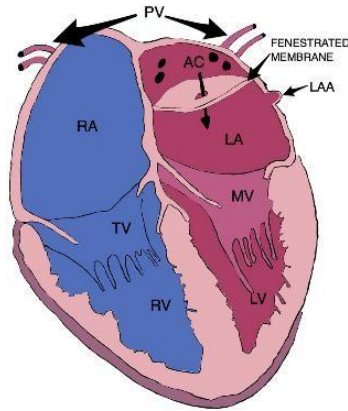


Fig. 5: Cor Tri-atriatum Sinister (Avari et al. 2017).

Diagnosis: Right ventricular hypertrophy (RVH) and right axis deviation are frequently seen on ECGs. On a chest x-ray, the right heart is enlarged to varied degrees, there is significant PA, and there is evidence of pulmonary venous congestion or pulmonary edema. To evaluate the intra-atrial membrane features and the presence of related CHD, TTE is essential.

Treatment: Any age should be taken into consideration if there are any associated symptoms or consequences because surgery is the only effective treatment.

Outcome: Following surgery, there is little chance of recurrence intra-atrial blockage.

Congenital Mitral Valve Stenosis

Congenital mitral stenosis (CMS) is a congenital heart malformation comprising a spectrum of morphologically heterogeneous developmental anomalies that result in functional and anatomic obstruction of inflow into the left ventricle of a patient (Fig. 6).

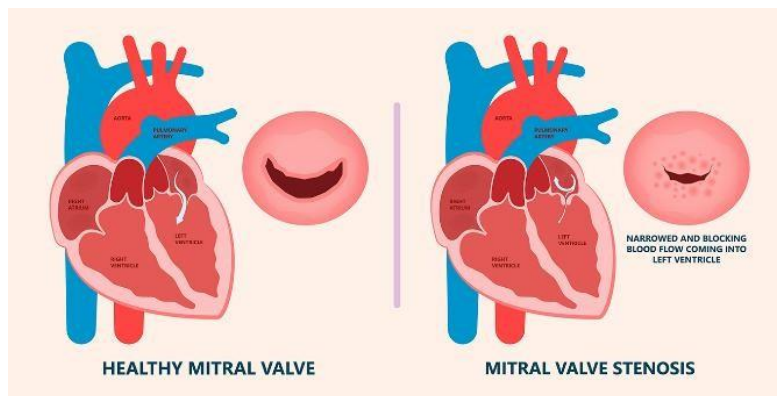


Fig. 6: Congenital mitral valve stenosis (<https://drdraghu.com/services/conditions/mitral-valve-stenosis>).

Diagnosis: A normal ECG throughout infancy may change as the condition advances to LA enlargement, RVH enlargement, and RA enlargement. Atrial arrhythmias could develop if the atrium continues to dilate. LA, PA, and RV are dilated on a chest x-ray, and Kerley's B lines might be visible in cases of severe disease. In terms of MV anatomical and functional aspects, TTE is diagnostic of the disease.

Treatment: To treat pulmonary venous congestions, patients may need diuretics. Surgery to repair the valve or replace it with a prosthetic valve may be necessary in cases of severe obstruction, although doing so bears risk of morbidity and mortality.

Congenital Mitral Valve Regurgitation

Congenital means it is present at birth and the mitral valve prolapse (MVR) can cause blood to leak backward, a condition called mitral valve regurgitation (Fig. 7). Congenital mitral valve anomalies are problems with the valve between the heart's two left chambers.

Diagnosis: At the apex, there is a regurgitant holosystolic murmur that radiates to the left axilla and left back. Additionally, a strong S3 and an apical rumble could be heard. ECG and CXR results for moderate to severe regurgitation may indicate hypertrophy and enlargement of the left chambers. Echocardiography, which may be used for both qualitative and quantitative evaluation, is the primary method for identifying congenital MV regurgitation.

Treatment: Diuretics and ACE inhibitors are needed for patients who have congestive heart failure in order to lower afterload and increase cardiac output (Hayek et al. 2005).

Aortic stenosis: Aortic stenosis (AS) affects 3-6% of CHD patients overall, and males are more likely to develop it (4:1) than females. Valvular (70%), sub-valvular (23%), or supra-valvular (7%) stenosis are all possible. Typically, a bicuspid aortic valve with a fused commissure and an eccentric orifice causes valvular AS (Fernandes et al. 2004).

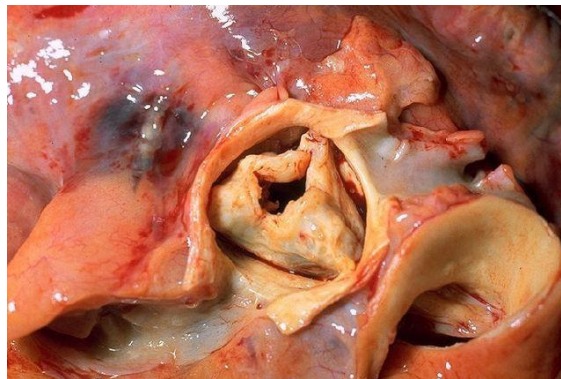


Fig. 7: Stenotic aortic valve (macroscopic appearance) (<https://emedicine.medscape.com/article/150638-overview>).

Treatment: Inotropic medications and diuretics should be initiated to treat CHF in critical, newborn valvular AS, and prostaglandin E1 infusion should be administered to reopen the arterial duct. As quickly as feasible, relief from AS should be accomplished with either surgical valvotomy or percutaneous balloon valvuloplasty (PBV). In many clinics, percutaneous valvuloplasty has taken over as the treatment of choice, including for children and young adults (Maskatia et al. 2011).

(C) Right-Heart Congenital Heart Disease

Pulmonary Valve Stenosis

Pulmonary valve stenosis (PVS) is a type of heart valve disease that involves the narrowing of the pulmonary valve, which controls the flow of blood from the heart's right ventricle into the pulmonary artery to carry blood to the lungs (Fig. 8). 8-12% of all CHDs are caused by pulmonary valve stenosis (PVS) with normal cardiac connections.

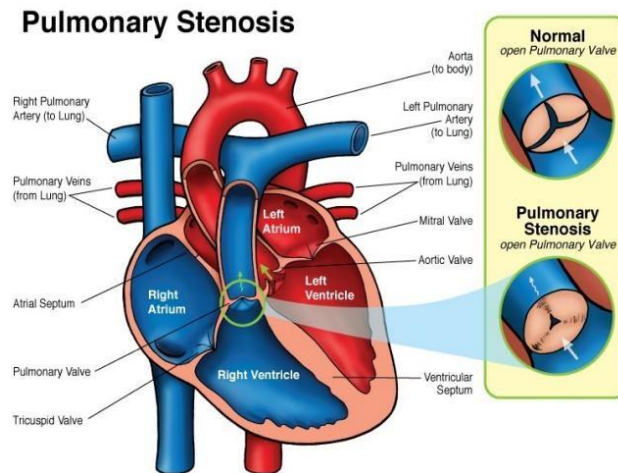


Fig. 8: Pulmonary valve stenosis (<https://www.amrihospitals.in/blog/pulmonary-valve-stenosis-causes>).

Diagnosis: When there is substantial stenosis, the ECG typically reveals right axis deviation, RVH, and a strain pattern; the severity of the disease is inversely correlated with the degree of RVH. Neonatal with significant stenosis have oligemic lungs and various degrees of cardiomegaly visible on chest x-rays. Most children with moderate to severe PVS have normal cardiac silhouettes that will last for a very long time. As these children age, however, major PA enlargement may become more noticeable, which is consistent with post-stenotic dilatation. There is still limited value for CT and MRI for diagnostic purposes in this age group, as well as cardiac catheterization, which is strictly an interventional procedure. TTE is the primary diagnostic modality, able to assess the features of the valve, the annulus size, the RV hypertrophy and dimensions, and the presence of interatrial shunt.

Treatment: PGE1 infusion should be started as soon as possible to increase the size of PDA and thereby improve pulmonary blood flow (PBF). Once stabilized, neonates should undergo cardiac catheterization for percutaneous pulmonary valve balloon dilation, which is the preferred procedure, in order to reduce mortality in newborns with critical PVS. After successful balloon dilation, PGE1 may then be stopped, but it's important to keep in mind that some neonates may still momentarily need another source of PBF. This can happen when RV stiffness prevents proper diastolic ventricular filling or when infundibular muscular hypertrophy becomes more noticeable after the procedure and obstructs antegrade PV flow. A PDA stent or a surgical mBT Scan serve as this additional source. In critically sick patients, immediate surgical pulmonary valvotomy is recommended if percutaneous balloon dilatation fails (Coles et al. 1984).

(d) CHD with Anomalous Origin of Great Arteries**Truncus arteriosus**

One to four percent of CHDs are truncus arteriosus (TA) cases. TA may be connected to another poly malformative syndrome like CHARGE association, which includes coloboma, heart disease, choanal atresia, retardation of growth, genitourinary abnormalities, and esophageal atresia, in approximately 35% of cases of the other cono-truncal anomalies (TOF, TGA and DORV) (McGoon et al. 1968) (Fig. 9).

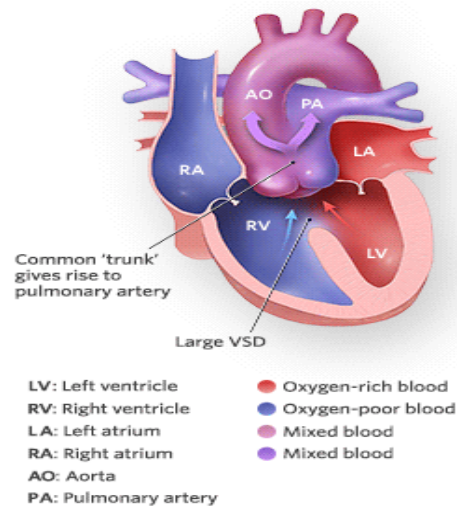


Fig. 9: Truncus arteriosus (<https://www.chop.edu/conditions-diseases/truncus-arteriosus>).

Treatment: The Rastelli procedure involves separating the PA from the arterial trunk and connecting it to the RV with the interposition of an RV-to-PA conduit; in this way, the truncal valve will become the "neo-aortic" valve. Surgery is typically performed within the first six months of life (McGoon et al. 1968).

Heterotaxy

Heterotaxy is a term used to describe a variety of cardiac and systemic defects that result from an embryological failure to differentiate and are defined by an aberrant arrangement of the internal thoracic and abdominal organs along the left-right axis of the body. The primary characteristic of heterotaxy is the mirror-image duplication of typically unilateral structures; some individuals appear to have left isomerism (bilateral left-sidedness), while others appear to have right isomerism (bilateral right-sidedness). At the cardiac level, atrial appendages may show isomerism of the pectinate muscles, and bronchial morphology, the lungs, and atrial appendages are all strongly correlated. The appendages are the most consistent elements within the atrium; their shape and the unique morphology of their junction with the remainder of the atrium allow them to be identified as being morphologically right or left. CHDs are typically present in heterotaxy and range in complexity from mild to severe; some CHDs, like AVSD, can be observed in both forms of isomerism but are more common in the right one (approximately two-thirds of cases) than the left (about half of cases). Other CHDs, like pulmonary stenosis, TGA, and TAPVC, are substantially more prevalent in right isomerism (Micheletti 2019).

Quality of Life of Children with Congenital Heart Disease

A previous study showed overall, 52% of the kids who died from non-cardiac causes between the years 1954 and 1979 and had a congenital heart condition. In 1984, we added a prospective study to this retrospective investigation. We discovered that 39% of the kids most likely passed away for reasons unrelated to the heart, and another 5% most likely did as well. In other words, it's likely that 44% of newborns with heart problems who passed away did not do so as a result of a heart issue (Samánek et al. 1986).

A previous study showed Both parents and children with CHD had their TNO-AZL Child Quality of Life Questionnaire (TACQOL) results compared to those of a reference group of children who were the same age. The whole sample of ConHD children (n = 113, 8-15 years old) had significantly lower mean scores on positive emotional functioning, motor functioning, and cognitive functioning than reference peers, indicating that they were functioning poorer than their reference peers. Aged 8 to 11 years, CHD children had poorer mean scores on 5 of the 7 TACQOL scaled than reference peers. In comparison to 12- to 15-year-old CHD children, they also scored lower on positive emotional functioning. There were no noticeable differences in health-related quality of life between CHD boys and girls or between other diagnostic categories (Spijkerboer et al. 2006).

Congenital Heart Disease - Bangladesh Perspective

A prospective hospital-based study was carried out in the pediatric cardiology department of the Combined Military Hospital (CMH) Dhaka over the course of three years, from January 2006 to December 2008. The study's data set consisted of all 5,658 live births that were delivered at CMH Dhaka between January 2006 and December 2008. The study's claimed incidence, which is 25/1000 live births, is significantly greater than any previous study carried out to date (Fatema et al. 2008).

Another study was carried out in Dhaka Shishu Hospital for a period of 2 years, from January 2008 to December 2009 prospectively, and another 2 years, from January 1998 to December 1999 retrospectively, data were acquired from hospital records. In the study, patients ranging in age from the first day of life to 12 years old were enrolled. In total, 539 people received a CHD diagnosis between January 2008 and December 2009. Only 11.9% of the 312 patients with CHD diagnosed between January 1998 and December 1999 were doing so during the newborn era. However, from January 2008 to December 2009, 27.5% of CHD cases were discovered at the neonatal stage (Hussain et al. 2010). Another prospective study (Sharmin et al. 2008) was conducted among the infants through 12-year-olds admitted to the pediatrics department of Rajshahi Medical College & Hospital over the course of a year. Ventricular septal defect was the most prevalent form of congenital cardiac disease in this investigation. This is consistent with much other research (Mitchell et al. 1971, Bound and Logan 1977, Jullen et al. 2002, Bernstein 2008).

Conclusion

The epidemiology and etiology of CHD also aid in prioritizing the regions that require intervention and any new rules that public health officials may enact. Patients and the parents of infants with CHD must recognize the value of routine medical exams, which can be done with the help of successful transition program and provider teamwork. Early diagnosis and effective management of congenital heart disease (CHD) in children are crucial for enhancing outcomes. Comprehensive care requires the collaboration of pediatric cardiologists,

surgeons, and various healthcare professionals. Ongoing research and advancements in medical technology are essential and offer hope for further improving the prognosis and quality of life for children with CHD.

Conflict of interest: The authors declare that there is no competing interest.

References

- Anderson RH, Brown NA and Webb S (2002). Development and structure of the atrial septum. *Heart* 88: 104-10.
- Avari M, Nair S, Kozłowska Z and Nashef S (2017). Cor triatriatum sinistrum: presentation of syncope and atrial tachycardia. *Case Reports*, p.bcr2016218395. <http://dx.doi.org/10.1136/bcr-2016-218395>.
- Backes CH, Cheatham SL, Deyo GM, Leopold S, Ball MK, Smith CV, Garg V, Holzer RJ, Cheatham JP and Berman DP (2016). Percutaneous patent ductus arteriosus (PDA) closure in very preterm infants: feasibility and complications. *Journal of American Heart Association* 5(2): e002923.
- Bernstein D (2008). Epidemiology and Genetic Basis of Congenital heart disease. *In*: Behrman RE, Kligman RM, Jenson HB, editors. *Nelson textbook of Pediatrics*. Philadelphia: Saunders, 19th Edition, pp. 1878-1881.
- Bound JP and Logan WF (1977). Incidence of congenital heart disease in Black pool: 1957-1971. *British Heart Journal* 39: 445-450.
- Butera G, Carminati M, Chessa M, Piazza L, Micheletti A, Negura DG, Abella R, Giamberti A and Frigiola A (2007). Transcatheter closure of perimembranous ventricular septal defects. Early and long term results. *Journal of the American College of Cardiology* 50: 1189-95.
- Butera G, Romagnoli E, Carminati M, Chessa M, Piazza L, Negura D, Giamberti A, Abella R, Pomè G, Condoluci C and Frigiola A (2008). Treatment of isolated secundum atrial septal defects: impact of age and defect morphology in 1,013 consecutive patients. *American Heart Journal* 156(4): 706-712.
- Camm AJ and Bunce NH (2005). *Cardiovascular disease*. Kumar P, Clark M *Clinical medicine*. 6th ed. London: Elsevier Saunders Limited, pp.725-871.
- Carminati M, Butera G, Chessa M, De Giovanni J, Fisher G, Gewillig M, Peuster M, Piechaud JF, Santoro G, Sievert H, Spadoni I and Walsh K (2007). Transcatheter closure of congenital ventricular septal defects: results of the European Registry. *The European Heart Journal* 28: 2361-8.
- Carminati M, Butera G, Chessa M, Drago M, Negura D and Piazza L (2005). Transcatheter closure of congenital ventricular septal defects with Amplatzeroccluders. *The American Journal of Cardiology* 96: 52L-8L.
- Chowdhury MNI, Ullah MA, Hossain MB and Islam SMS (2022a). Prevalence and clinical features of cardiac murmur with its subtypes of congenital heart disease in Rajshahi, Bangladesh. *Journal of Bio-Science* 30(1): 115-121.
- Chowdhury MNI, Ullah MA, Hossain MB and Islam SMS (2022b). Pattern of congenital heart diseases in children in a tertiary care hospital of Bangladesh. *Journal of Bio-Science* 30(2): 39-45.
- Coles JG, Freedom RM, Olley PM, Coceani F, Williams WG and Trusler GA (1984). Surgical management of critical pulmonary stenosis in the neonate. *The Annals of Thoracic Surgery* 38(5): 458-65. doi: 10.1016/s0003-4975(10)64184-7.
- Fatema NN, Chowdhury RB and Chowdhury L (2008). Incidence of congenital heart disease among hospital live birth in a tertiary hospital of Bangladesh. *Cardiovascular Journal* 1(1): 14-20.
- Fernandes SM, Sanders SP, Khairy P, Jenkins KJ, Gauvreau K, Lang P, Simonds H and Colan SD (2004). Morphology of bicuspid aortic valve in children and adolescents. *Journal of the American College of Cardiology* 44: 1648-51.

- Furlan AJ, Reisman M, Massaro J, Mauri L, Albers GW, Felberg R, Herrmann H, Kar S, Landzberg M, Raizner A, Wechsler L and Closure I (2012). Closure or medical therapy for cryptogenic stroke with patent foramen ovale. *The New England Journal of Medicine* 366: 991-9.
- Fyler DC, Buckley LP, Hellenbrand WE and Cohn HE (1980). Report of the New England Regional Infant Cardiac Program. *Pediatrics* 65(Suppl): 375-461.
- Hayek E, Gring CN and Griffin BP (2005). Mitral valve prolapse. *Lancet*, 365(9458): 507-18.
- Hlavacek AM, Crawford FA Jr, Chessa KS and Shirali GS (2006). Real-time three-dimensional echocardiography is useful in the evaluation of patients with atrioventricular septal defects. *Echocardiography* 23: 225-31.
- Hoffman JI and Kaplan S (2002). The incidence of congenital heart disease. *Journal of the American College of Cardiology* 39(12): 1890-1900.
- Hoffman JI, Kaplan S and Liberthson RR (2004). Prevalence of congenital heart disease. *The American Heart Journal* 147: 425-39.
- Holzer R, De Giovanni J, Walsh K, Tometzki A, Goh T, Hakim F, Zabal C, de Lezo JS, Cao QL and Hijazi ZM (2006). Transcatheter closure of perimembranous ventricular septal defects using the Amplatzer membranous ventricular septal defect device occluder: immediate and midterm results of an international registry. *Catheterization and Cardiovascular Interventions* 68: 620-8.
- Hussain M, Tahura S, Sayeed MA, Rahman MM, Rahman MM and Kar SK (2010). Past and present pattern of congenital heart disease at Dhaka Shishu Hospital: a situation analysis. *Bangladesh Journal of Child Health* 34(2): 51-55.
- Jackson M, Walsh KP, Peart I and Arnold R (1996). Epidemiology of congenital heart disease in Merseyside – 1979 to 1988. *Cardiology Young* 6: 272-80.
- Jordan SC and Scott O (1989). *Heart disease in Paediatrics*. 3rd Ed. Butterwoths, eBook ISBN: 9781483161433. <https://shop.elsevier.com/books/heart-disease-in-paediatrics/jordan/978-0-7506-0537-3>.
- Kliegman RM, Behrman RE and Jenson HB (2007). *Nelson Text Book of Pediatrics*. Philadelphia: WB Saunders Co., 1384, pp. 99. <https://www.scirp.org/reference/referencespapers?referenceid=2800380>.
- Malik A (1976). Congenital and acquired heart diseases: (A survey of 7062 persons). *Bangladesh Medical Research Council Bulletin* 2(2): 115-119.
- Maskatia SA, Ing FF, Justino H, Crystal MA, Mullins CE, Mattamal RJ, Smith EB and Petit CJ (2011). Twenty-five-year experience with balloon aortic valvuloplasty for congenital aortic stenosis. *The American Journal of Cardiology* 108(7): 1024-1028.
- McGoon DC, Rastelli GC and Ongley PA (1968). An operation for the correction of truncus arteriosus. *The Journal of the American Medical Association* 205(2): 69-73. doi:10.1001/jama.1968.03140280023007.
- Micheletti A (2019). Congenital heart disease classification, epidemiology, diagnosis, treatment, and outcome. *Congenital Heart Disease: The Nursing Care Handbook*, pp. 1-67. doi: 10.1007/978-3-319-78423-6_1.
- Mitchell SC, Korones SB and Berendes HW (1971). Congenital heart disease in 56,109 births. Incidence and natural history. *Circulation* 43: 323-332.
- Muntean I, Togănel R and Benedek T (2017). Genetics of congenital heart disease: past and present. *Biochemical Genetics* 55(2): 105-123. <https://doi.org/10.1007/s10528-016-9780-7>.

- Najm HK, Coles JG, Endo M, Stephens D, Rebeyka IM, Williams WG and Freedom RM (1997). Complete atrioventricular septal defects: results of repair, risk factors, and freedom from reoperation. *Circulation* 96(9 Suppl): II-311. <https://pubmed.ncbi.nlm.nih.gov/9386116>.
- Nygren A, Sunnegard J and Berggren H (2005). Preoperative evaluation and surgery in isolated ventricular septal defects: a 21 years' perspective. *Heart (MBJ Journal)* 83: 198-204. doi: 10.1136/heart.83.2.198.
- Radford DJ, Perkins L, Lachman R and Thong YH (1988). Spectrum of Di George syndrome in patients with truncus arteriosus: expanded Di George syndrome. *Pediatric Cardiology* 9: 95-101.
- Roos-Hesselink JW, Meijboom FJ, Spitaels SE, Van Domburg R, Van Rijen EH, Utens EM, Bogers AJ and Simoons ML (2004). Outcome of patients after surgical closure of ventricular septal defect at a young age: longitudinal follow-up of 22-34 years. *European Heart Journal* 25: 1057-62.
- Roos-Hesselink JW, Meijboom FJ, Spitaels SEC, Van Domburg R, Van Rijen, EHM, Utens EMWJ, Bogers AJJC and Simoons ML (2003). Excellent survival and low incidence of arrhythmias, stroke and heart failure long-term after surgical ASD closure at young age: a prospective follow-up study of 21–33 years. *European Heart Journal* 24(2): 190-197.
- Roy K, Shahed M, Roy K, Sarah QS and Chowdhury NS (2020). Clinical presentation and complications of different congenital heart disease in children. *American Journal of Pediatrics* 6(4): 481-487.
- Samanek M, Goetzova J and Benesova D (1986) Causes of death in neonates born with a heart malformation. *International Journal of Cardiology* 11: 63-74. doi: 10.1016/0167-5273(86)90200-7.
- Schneider B, Zienkiewicz T, Jansen V, Hofmann T, Noltenius H and Meinertz T (1996). Diagnosis of patent foramen ovale by transesophageal echocardiography and correlation with autopsy findings. *The American Journal of Cardiology* 77(14): 1202-1209.
- Schwartz MC, Nykanen D, Winner LH, Perez J, McMahan M, Munro HM and Narasimhulu SS (2016). Transcatheter patent ductus arteriosus occlusion in small infants. *Congenit Heart Disease* 11(6): 647-655. doi: 10.1111/chd.12360.
- Sharmin LS, Haque MA, Bari MI and Ali MA (2008). Pattern and clinical profile of congenital heart disease in a teaching hospital. *TAJ: Journal of Teachers Association* 21(1): 58-62. doi:10.3329/TAJ.V21I1.3221.
- Simpson JM and Miller O (2011). Three-dimensional echocardiography in congenital heart disease. *Archives of Cardiovascular Diseases* 104: 45-56.
- Spijkerboer AW, Utens EM, De Koning WB, Bogers AJ, Helbing WA and Verhulst FC (2006). Health-related quality of life in children and adolescents after invasive treatment for congenital heart disease. *Quality of Life Research* 15: 663-73.
- Wernovsky G and Licht DJ (2016). Neurodevelopmental outcomes in children with congenital heart disease- What can we impact? *Pediatric Critical Care Medicine* 17(8_suppl Suppl 1): S232. <https://doi.org/10.1097/PCC.0000000000000800>.
- Wu W, He J and Shao X (2020). Incidence and mortality trend of congenital heart disease at the global, regional, and national level, 1990-2017. *Medicine (Baltimore)* 99(23): e20593. doi: 10.1097/MD.00000000000020593.
- Zimmerman MS, Smith AGC, Sable CA, Echko MM, Wilner LB, Olsen HE, Atalay HT, Awasthi A, Bhutta ZA, Boucher JL and Castro F (2020). Global, regional, and national burden of congenital heart disease, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. *The Lancet Child and Adolescent Health* 4(3): 185-200.