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Review Article

Role of Stents and its Complications in Congenital Heart Disease

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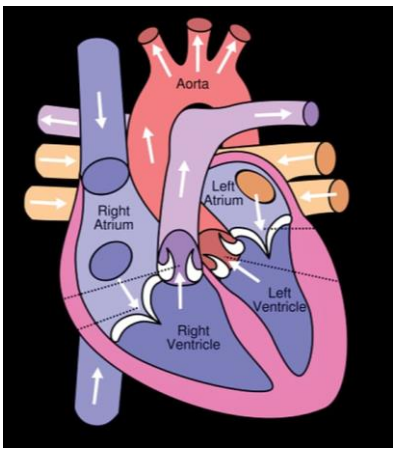
ABSTRACT

In many types of congenital heart disease (CHD), intravascular or intracardiac stenoses are present. As a result, the placement of stents for stenotic lesions in pediatric cardiology is becoming a common interventional technique. Additionally, stents are known to be utilized to prevent intracardiac communications from becoming blocked or to ensure that aneurysms in vessels are excluded. As the initial generation of devices was improved upon, other "modern" stents with various design features emerged. Although technology has advanced dramatically over the past 20 years, the "ideal stent" has yet to be created. As a result, the pediatric interventionalist must choose the best stent for each lesion. On this foundation, the pros and cons of the currently available stents for widespread use in CHD are reviewed. The lack of adaptation to somatic growth is one of the current issues that has been addressed with new ideas and approaches. The currently utilized generation of stents may therefore be replaced in the future by biodegradable or growing stents. The indications for the use of stents in the treatment of CHD may become more widespread.

Keywords: Congenital heart disease, stenting, arterial septal defect, ventricular septal defect.

INTRODUCTION

A deficiency in the anatomy of the heart or major blood vessels that exists at birth is referred to as a congenital heart defect (CHD), also known as a congenital heart anomaly or congenital heart disease. Congenital cardiac defects fall within the category of cardiovascular diseases.[1,2]



(Figure 1, source: https://www.gov.uk/government/publications/congenital-heart-disease-description-in-brief/congenital-heart-disease-information-for-parents-html)

When infectious etiologies are taken into consideration, congenital heart disease (CHD) is the largest cause of birth abnormalities and the leading cause of mortality in the first year of life. CHD is a major cause of childhood morbidity and mortality worldwide, with a prevalence of 19 to 75 per 1,000 live births and a much higher percentage of miscarriages. The etiology of CHD is still not fully understood, despite improvements in medical and surgical therapy, and as more children with CHD grow up and have children, it becomes even more important to comprehend its causes. Traditional research, such as the Baltimore-Washington Infant Study, has discovered that CHD is complex, resulting from both genetic predisposition and environmental factors.

The molecular mechanisms controlling heart development have become better understood over the last few decades. Numerous mouse models with developmental abnormalities in the heart have been produced as a result of the advancement of gene-targeting technology.[3] Numerous transcriptional regulators, signaling molecules, and structural genes that are essential for correct heart morphogenesis have been discovered because of this research. Furthermore, a number of genes have been found to be under the control of these highly conserved molecular pathways. These studies of the molecular mechanisms underlying heart development have aided in the discovery of genetic causes of CHD and offer proof that many genes may play etiologic roles in human CHD.

EPIDEMIOLOGY OF CHD:

CHDs, which affect 1% of new-born and significantly increase infant morbidity and mortality, are the most common deadly abnormality. These represent 30% of all congenital anomalies and have a birth prevalence of 4% to 5% per 1000. 1 The percentage of people surviving to adulthood has increased over the past few decades due to improvements in diagnostic methods and curative therapies for CHDs. The incidence rate of CHD has increased because CHD patients are now more likely to procreate. Between 1930 and 2009, the global birth prevalence grew, and over the previous ten to fifteen years, it has stabilized around 9 per 1,000 live births. A high birth-rate in underdeveloped nations contributes to a high prevalence of in different populations, certain CHDs are

more or less common. The prevalence of CHD was reported to be 8.1 per 1,000 live births from 1998 to 2005 in a population-based study from Atlanta, Georgia, in the United States. Population-based studies from Taiwan (2000–2006) and Denmark (1977–2005) both found higher prevalence of CHD, with rates of 10.3 and 13.1 per 1,000 live births, respectively. 6,7 Although other nations have birth defect registries, India does not have such organized studies. According to figures from the 2011 census, India's crude birth rate (CBR) is 21.8 per 1000 people. The total number of live births annually at this CBR is roughly 26 382 216. If the birth prevalence is assumed to be 8 per 1000 live births, then it is likely that close to 211 058 children are born per year with CHD. If spontaneous abortions and neonatal fatalities are included, this number increases significantly.

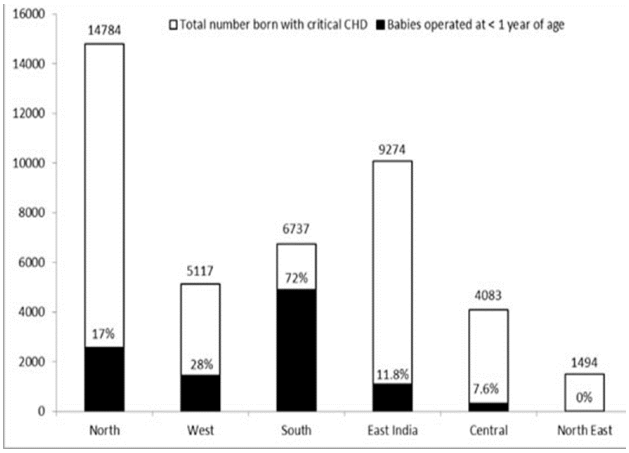
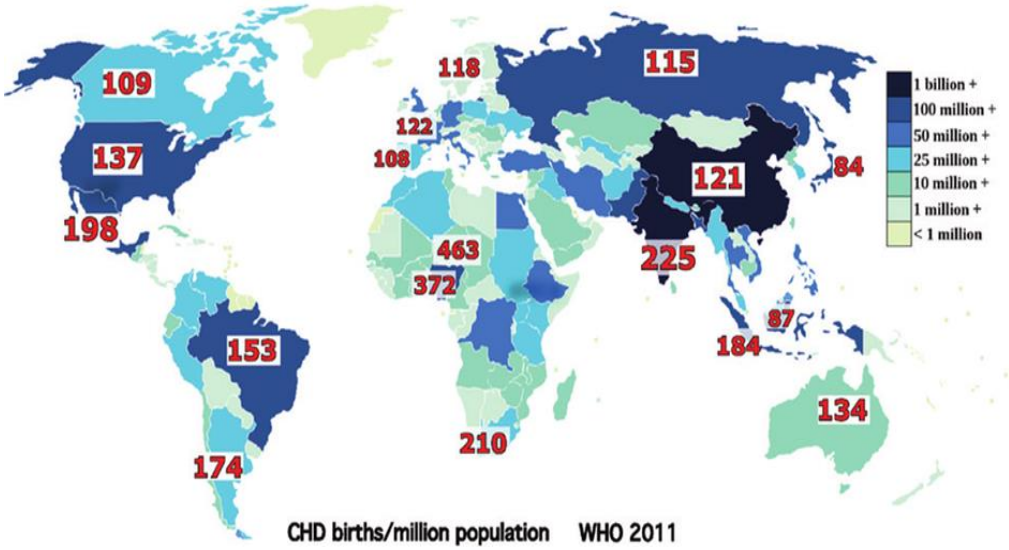


Figure 2: New-borns with critical heart disease who have surgery as a percentage of all infants born with critical heart illness.

(Fig. source: https://indianpediatrics.net/dec2018/dec-1075-1082.htm)



The proportion of a million people who are born with congenital cardiac disease.

Figure 3: Congenital heart disease births worldwide, by continent.

(Fig. source: https://www.researchgate.net/figure/Total-annual-birth-of-children-with-CHD-by-continent-wise-Total-number-is-roughly_fig2_333758386)

STENT:

A stent is a tiny mesh tube that is often used to keep open body channels, such as weak or restricted arteries, open. [4] The coronary arteries that supply the heart with blood that is high in oxygen are frequently treated with stents. Stents may also be used to treat a lung airway obstruction as well as an aneurysm, a protrusion in the artery's wall.

INDICATIONS OF STENT

Stents are commonly used to treat obstructive lesions in the

(1) Branch and peripheral pulmonary arteries.

(2) Systemic and pulmonary veins.

(3) Aorta and branches stents for congenital heart disease.

(4) Right ventricular outflow tract (RVOT) conduits.

(5) Maintenance of the arterial duct in duct-dependent circulation.

(6) Maintenance of stenosed aortopulmonary collateral arteries or surgically constructed but obstructed shunts

(7) Maintenance of intracardiac connections.

IDEAL PROPERTIES OF STENTS:

The following qualities are ideal for a paediatric cardiology stent design:

(1) Low stent profile;

(2) High trackability;

(3) Flexibility to negotiate steep curves;

(4) Good radio-opacity and visibility;

(5) Compatibility with magnetic resonance imaging (MRI), without artefacts;

(6) Predictable expansion with minimal foreshortening;

(7) Sufficient radial strength; and

(8) Low rigidity with no material fatigue.

STENT IMPLANTATION FOR SPECIFIC CHD CASES

Types of defects:	Indications for intervention	Timing for intervention	Type of intervention
1.ATRIAL SEPTAL DEFECT a) Ostium Sicundum ASDS	Closures are generally not required probably 5mm ASDs (atrial septal defect) are going to close on their own	Elective closure around 4-5yrs of age. Closure during infancy is not undertaken unless the infant is symptomatic	Surgery and trans-catheter closures.
b) Premium Ostium ASDS	Co-exist with severe mitral insufficiency. Digoxin and diuretics should all be started right away.	Surgical repair in asymptomatic patients is typically advised between the ages of 3-5 years old.	Surgical repair is preferred. Atrial defect is sealed with an autologous pericardial patch.
c)Patent foramen ovale	Anti Congestive treatment should be used initially. Pulmonary vasodilators like sildenafil and bosantin. lung transplant could be an option.	VSD closure should be done before 6-12 months of age	Medical, trans-catheter, surgical and hybrid methods of treatment are offered for VSDs (ventricular septal defect)
2) VENTRICULAR SEPTAL DEFECT	Atrioventricular (AV) septal (located in the inlet septum), supracrustal (located in the conal septum in the sub pulmonary region), and muscular (located in the peri membranous ventricular septum in the membranous ventricular septum in the subaortic region)	Regardless of how well heart failure is under control and how much weight has been gained, VSD closure should be done before 6 to 12 months of age (definitely no later than 18 months).	Medical, trans-catheter, surgical, and hybrid methods of treatment are offered for VSDs. The procedure used for a specific patient is determined by the patient's clinical condition, the size, and the location of the VSD
3) STENTS IN PULMONARY ARTERIES	Stent indications and usage for treating PA stenosis have grown recently. Stents are mostly used to treat origin stenosis that develops naturally or after prior surgery, as well as elastic rebound, intimal tears following balloon angioplasty, kinking or tenting of the branch PA (pulmonary artery), external compression of the branch PA, and recanalization of completely occluded veins.		

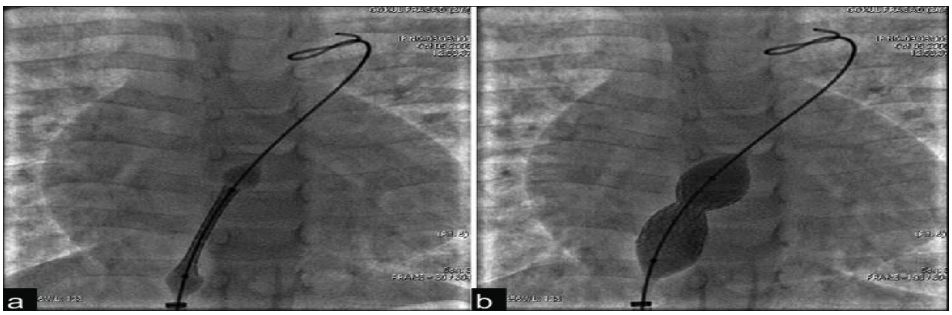


Figure 4: Depiction of stenting in atrial septal defect.

(Source: https://www.researchgate.net/figure/Atrial-septal-stenting-in-bidirectional-post-tricuspid-shunt-with-severe-mitral-stenosis_fig3_272421980)

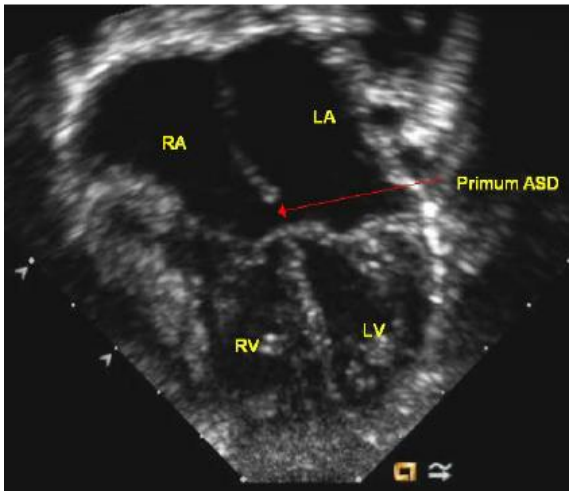


Figure 5: Depiction of stenting in ostium asds.

(Source: https://www.researchgate.net/figure/Four-chambered-view-of-the-heart-demonstrates-ostium-primum-atrial-septal-defect-ASD_fig3_224830715)

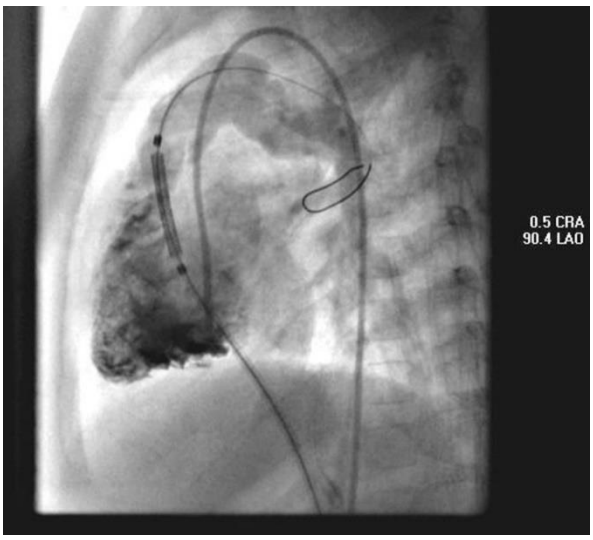


Figure 6: Depiction of stenting in ventricular septal defect

(Source: https://www.hmpgloballearningnetwork.com/site/jic/articles/right-ventricular-outflow-tract-stenting-tetrology-fallot-restrictive-ventricular-septal)

STENTS TO ENSURE PATENCY OF INTRACARDIAC COMMUNICATIONS

An unrestrictive ASD is necessary to achieve an acceptable cardiac output and/or systemic saturation for some severe congenital heart abnormalities, as well as to relieve right or left atrial hypertension.[5] However, the employment of those procedures may be ineffective to achieve and sustain a sufficiently sized communication in infants past the neonatal period or in neonates with hypoplastic left heart syndrome due to the increasing thickness of the IAS(Internal anal sphincter) in these populations. Consequently, atrial septal stent placement has been successfully used to encourage a long-lasting, unrestrictive ASD. Techniques include transeptal puncture or radiofrequency perforation (in the case of an intact septum), balloon or blade septostomy, and static balloon dilatation of the interatrial septum, which are typically used to create or widen an ASD transcatheter (IAS).[6] The use of cutting balloons in the past (Boston Scientific) may have been beneficial, because the microsurgical blades of the cutting balloon allow controlled tearing of the septal wall as opposed to stretching of the thickened ASD as seen with static balloon dilatation. Most of the time, we and other writers use pre-mounted Palmaz-Genesis stents (Cordis; Johnson & Johnson, Miami, FL, USA) with diameters between 7 and 8 mm and lengths between 12 and 26 mm. Sometimes, securing the stent and preventing stent migration involve redilating the stent with a larger balloon to form a "diabolo" configuration. As thrombus formation and increasing blockage may occur, close monitoring is required. Another modality where fenestration may be beneficial is failing Fontan circulation. As a result,the usage of covered and uncovered stents has been discussed in order to maintain the integrity of the recently established communication. Covered stents may be able to lower the immediate risk of bleeding.

RVOT STENTING AS PALLITATIVE TREATMENT

Primary repair might not be possible in cases of severe tetralogy of Fallot (TOF) that include PA hypoplasia, serious RVOT (Right ventricular outflow tract) blockage, and the presence of MAPCAs (major aortopulmonary collateral arteries). Early surgical alternatives for this type of two-staged repair include ductal stenting, an RV outflow patch, a central aorto-pulmonary anastomosis, and a modified Blalock-Taussig anastomosis. To ensure sufficient oxygen delivery and to encourage PA development through pulsatile flow, RVOT stent placement may occasionally be an additional option to surgical palliative care.[7]

In cases of pulmonary atresia with high-frequency (HF) perforation, RVOT stenting may also be taken into consideration. Pre-mounted coronary or biliary stents, such as the Palmaz Genesis medium on Slalom balloon, are preferred for these children due to their small size.

COMPLICATIONS OF STENTING

Complications happen in 3.5–19% of instances and , resulting in a 2.3% procedure mortality overall. Location-specific stenting has a significant impact on complication rates. Interventions like arterial duct stenting are still difficult. Numerous issues that arise during the placement of stents are temporary and can be resolved without stopping the process; other issues may require surgery [8] As a result, congenital heart surgery and stent placement should be done in specialised facilities. The level of operator experience also influences the rate of complications. The placement of stents should only be done by experts.

1. Early Emobilization and Migration

The most frequent complications—stenting mal-positioning, migration, and early embolization—occur in 7.7% of all implantations and are reliant on the operator's skill.

2. Stent Mismatch

A relative restenosis of the vessel results from the stented vessel's inability to expand with the developing youngster. The use of stents in this rapidly expanding population may be constrained by the need for repeated stent dilations or surgical removal. Even with a covered CP stent, redilation with a larger balloon is an effective way to increase the diameter of previously implanted balloon-expandable stents to allow for somatic growth .Another method for expanding a stent with a vascular mismatch is to fracture a non-dilatable stent with a high-pressure balloon.[9]

3. Vascular Injury

Aortic wall injury occurs in less than 1% of native aortic coarctation stenting patients, more frequently in Turner syndrome, and even less frequently following PA branch stenting. Such trauma may cause vascular rupture, aneurysm, or dissection. Different interventional treatments are used to treat these problems (coil or device occlusion of a PA branch, implantation of a covered stent). Sometimes, emergency surgery is necessary.

4. COMPRESSION OF ADJACENT STRUCTURES

Patients with CHD who have stents implanted in chest circulatory structures may experience mechanical compression of neighboring structures.[10] Following transcatheter pulmonary valve replacement and angioplasty of the right PA (Video 4), coronary artery compression has been documented. Compression of the bronchi can be quite harmful.

5. Endocarditis

Rare reports of endocarditis following stent placement exist. However, reports claiming endocarditis occurs in 0.88 to 2.4% of patients annually since the introduction of valved stents and transcatheter pulmonary valve replacement have caused serious concerns. It is still unknown what the pathogenic mechanisms and risk factors are.

COMPLICATIONS BY IMPLANTATION:

The most common procedures involved stenting the aorta and branch pulmonary arteries [11] In one treatment setting, the stenting of pulmonary artery stenosis on both the left and right sides was performed on 33 patients. For all other anatomical sites, no more than 25 stent implants were made. The right ventricular outflow tract/pulmonary trunk group had the greatest complication rates (45%); this was a small cohort that included several post-surgical patients. Additionally, the aorto-pulmonary shunt group (22%), the pulmonary vein group (20%), and the arterial duct group all had relatively high complication rates. An even more specific breakdown of problems per implantation site is shown in table:

Complication rates per site:

Stented site	Total complications (%)	Major complications (%)
Right pulmonary artery	12 (13)	4 (4)
Left pulmonary artery	25 (19)	10 (8)
Arterial duct	8 (35)	3 (13)
AS fenestration	2 (13)	0 (0)
RVOT, pulmonary trunk	5 (46)	2 (18)
Aorto-pulmonary shunt	2 (22)	1 (11)
Arterial duct	8 (35)	3 (13)
Systemic vein	3 (15)	0 (0)
Aorta	9 (13)	2 (3)
Mustard baffle	1 (8)	1 (8)
MAPCA	4 (16)	0 (0)
Coronary artery	0 (0)	0 (0)
Pulmonary vein	1 (20)	1 (20)
Other sites	0 (0)	0 (0)
Total	72 (17)	24 (5.7)

Complication categories (major/minor) by implantation site

Distribution of complication categories:

Complication type	Total	Major	Mortality
Stent malposition/ migration/ embolization	32	8	0
Stent-induced pulmonary oedema	2	2	1
Arrhythmia	5	1	0
Inaccessible site	8	2	0
Vessel dissection	6	4	1
Balloon rupture	11	1	0
Other	8	6	5

MORTALITY:

A 2.3% procedural fatality rate was attributable to the deaths of seven patients during the stent implantation process. The following table lists the specifics of these cases. Three of the babies were under a year old (4.5% of new born fatal complications). The majority of these babies were gravely ill. The fatal complication rate for patients older than 1 year was 1.1%.

Age	Weight (kg)	Sex	Diagnosis	Cause of death	Stent location
7 months	6	Female	Aortic re-coarctation	Retro-peritoneal bleeding	Aorta
51 years	79	Male	Native right pulmonary artery stenosis	Hyper-perfusion right lung, followed by acute pulmonary oedema	Right pulmonary artery
11 years	50	Female	Post-operative Fallot, pulmonary branch stenosis	Coronary ischaemia due to stent induced compression, fatal LV dysfunction	Right pulmonary artery
1 month	3	Male	Severe aortic valve stenosis, LV hypoplasia	Ductal closure due to insufficient ductus coverage by stent	Arterial duct
2 years	13	Female	Idiopathic pulmonary vein stenosis	Thrombotic middle cerebral artery occlusion secondary to pulmonary vein stent thrombosis	Pulmonary vein
4 years	21	Female	Post-operative Fallot, LV hypoplasia	Possible vessel rupture, hypotension and tachycardia at ICU after procedure	Blalock-Taussig shunt
4 months	3	Female	Fallot, severe right ventricular outflow tract obstruction	Severe bradycardia prior to stent dilation	RVOT-pulmonary artery trunk

CONCLUSION

The usefulness of stents in CHD patients has been amply demonstrated during the past 20 years.[12] For a variety of pre- and post-operative vascular stenoses that do not respond to pure balloon dilatation, stenting is now recognized as a safe and effective treatment option. Therefore, stent therapy is currently the norm and first-line treatment for COA (contraction of aorta) in adults or post-operative PA branch stenoses.[13] In order to avoid high-risk recurrent procedures, the majority of stent applications seek to provide a long-lasting cure or tolerable palliation. Stents can now be used to keep the patency of inter-circulatory contacts that are either constructed with a specific purpose (like an arterial duct or an ASD) or naturally existent (like a vein).[14] Second-generation versions have replaced first-generation models, such as the original Palmaz stent, as a result of a considerable breakthrough in stent technology.[15] However, according to the criteria, the ideal stent for those with CHD has not yet been developed. It follows that ideas for the application of stents in extremely small patients that permit modification for somatic growth until adult vascular size is reached are necessary.[16]

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