Review Article

Congenital heart disease: Saving lives and securing liveliness with early primary care and expert family care

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Abstract

Pertinent perspectives for practical applications is advantageous professionalism. Congenital heart diseases (CHDs) are of varied types, severity, and complexity. Simple scientific approach along with sophistication is required for successful management at all levels. We gleaned important information from published evidence and authoritative resources and have put things in practical perspective. These include current prevention, care, correction, cure, and rehabilitation strategies for robust practices. Prospective prevention is with preconception counseling and addressing environmental factors. Fetal echocardiography and nondirective genetic counseling are important. Early detection and expert diligence is desirable. The suspicion is clinical. A number of clinical and investigative diagnostic modalities are available and should be utilized as guided by the clinical cues. Medical management includes general health maintenance, immunizations, monitoring, and complications treatment. Pediatric interventional cardiology is making rapid strides and treating many types of lesions. Understanding of procedure done is a prerequisite of follow-up care. Surgery is of curative, reparative, or palliative types. After surgical correction in early childhood long-term sequelae are rare. After reparative surgery some have life-long sequelae and some have significant late impairment. The number of postintervention/surgery survivors into adulthood is increasing. Microsurgical techniques are leading the way for precision and fewer complications. Follow-up care with "Ten Points Information and Action Plan (TP-IAP)" is suggested. The Armed Forces scenario involves frequent movements to difficult and harsh conditions. Special precautions for affected children needed. Cardiovascular health is enhanced by avoiding a sedentary lifestyle and obesity. Various intrinsic and extrinsic factors limiting functioning need to be suitably addressed.

Keywords: Environmental factors, genetic counseling, interventional cardiology, obesity, physical activity, prospective prevention

Introduction

Congenital heart disease (CHD) is a common cause of major congenital anomalies, the leading cause of death in children with these, and encompasses a diverse range of conditions. Clinically relevant CHD affects 1% of all live births.[1]

Early detection of CHD is of primary importance for improvements in the quality of life and reducing morbidity and mortality of children.[2] Primary care physicians need to be rejuvenated of the manifestations and management for early diagnosis and energetic diligence. Family physicians need to periodically monitor and manage progress. With advances in both corrective and palliative surgery, the number of children with CHD surviving to adulthood has increased dramatically.

In spite of advances in medical and surgical management, CHD continues to be the leading cause of death in children with congenital malformations.[3] Thus, the need for reviewing our present status and scope for progress, with the aim-

"*Simple robust expertise* and *evolving rewarding strategies*"

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Methods

Glean important information, from published evidence and authoritative resources, for comprehensive management of children with CHDs. Analyze trends for tactful applications by all. Put things in proper perspective.

Prospective prevention

Prevention of congenital anomalies is important, as the impact of these can be for life. Advances in our understanding of causation need to be converted into preventive strategies, popularized, and practiced.

Major advances are being made in the field of genetics with regards to diagnosis. The etiology in about 80% of CHD is multifactorial and arises through various combinations of genetic and environmental contributors. In about 20% of cases it is attributed to chromosomal anomalies, Mendelian syndromes, nonsyndromal single gene disorders or teratogens.[4] All this suggests that there is a lot of room for the interplay of multifactorial etiologies, such as the interactions between multiple genes, environmental factors, and spontaneous mutations.[5]

Targeting genetic and environmental factors stands to reasoning. Our present state of knowledge should guide actions. Based on current evidence we suggest two strategies.

First focused approach should be for families with children or relatives with CHD. The risk of CHD increases if a first-degree relative (parent or sibling) is affected. The incidence of CHD in the normal population is 0.8%. After the birth of a child with CHD or if a parent is affected the risk of CHD increases to 2–6% for the 2nd pregnancy. This recurrence risk varies with the type of lesion in the 1st child. If two first-degree relatives have CHD, the risk for a subsequent child may reach to 20–30%. When a 2nd child is found to have CHD, it is of a similar class as the lesion in their first-degree relative (e.g., conotruncal lesions, left-, or right-sided obstructive lesions, atrioventricular septation defects). The severity varies and also the presence of associated defects.[3] Ongoing genetic and molecular studies suggest intriguing links between certain specific cardiac lesions in some familial pedigrees.[6] Clinical significance of all this data is preconception/prenatal counseling. Fetal echocardiography and nondirective genetic counseling also needs implementation. Preventing excessive numbers of births of children with CHD by lowering the fertility rate is a suggested strategy.[7] Also, earlier theories had suggested that etiology of CHD is multifactorial, i.e., combination of a hereditary predisposition and environmental trigger; however, more recent research in molecular biology suggests that a much higher percentage are caused by point mutations.[8,9]

Second environmental factors known to be risk factors in etiology need to be suitably addressed. Known environmental or adverse maternal conditions and teratogenic influences are the cause of 2–4% of all cases of CHD. The excessive alcohol consumption during pregnancy is a risk factors for CHD.[10] A woman with insulin-dependent diabetes mellitus may significantly decrease her risk for having a child with birth defects by achieving good control of her disease before conception. Agents acting on pregnant women that may adversely affect the structure or function of the fetus and newborn's heart include alcohol, amphetamines, mycophenolate mofetil, lithium, warfarin, vitamin A derivatives, statins, antimetabolites, and anticonvulsant agents.[11] Appropriate radiologic equipment and techniques for reducing gonadal and fetal radiation exposure should always be used to reduce the hazards of this potential cause of birth defects.[12]

A recent meta-analysis of observational studies has revealed that maternal viral infection, in particular infection with rubella or cytomegalovirus, in early pregnancy is significantly associated with risk of CHD in offspring. Clinical implication is that early detection and intervention may help to reduce the incidence of CHD.[13]

Worldwide efforts to eliminate endemic rubella virus transmission and occurrence of congenital rubella affections include maintaining high population immunity through vaccination and surveillance.

The thoughtfulness of all the above approaches is best summarized as-

"*Prevention is of utmost importance,*

Particularly when treatment aims cure or near cure or just palliation"

Detection and diagnosis

Early detection and expert diligence is desirable. The most important reason pointed for the dismal state of affairs of children with CHD in our country is that only a very small fraction of CHD cases are detected at birth and during infancy.[14] With early diagnosis nil mortality has been demonstrated.[15]

The suspicion of CHD is clinical. Features in the history are maternal diabetes mellitus; premature birth; birth at a high altitude; fever and or rash in first trimester, and teratogenic medications. Clinical symptoms suggestive are features of heart failure, a history of syncope, chest pain, or squatting; cyanosis; chronic unexplained hoarseness; asymmetric facies with crying; and a physical appearance suggestive of a clinical syndrome. Symptoms of heart failure are age specific. In infants, feeding difficulties are common. Others are poor weight gain; difficulty in feeding; breathes too fast; breathes better when held against the shoulder; inappropriate sweating; irritability and restlessness; persistent cough and wheezing; recurrent pneumonia; edema. In older children, heart failure clinical clues are exercise intolerance, difficulty keeping up with peers during sports, fatiguability and poor growth.[16,17]

Clinicians of all caliber and qualifications should continue to evaluate on the basis of Nadas' Criteria for clinical diagnosis of

CHD. The major criteria include (i) systolic murmur grade III or more, (ii) diastolic murmur, (iii) cyanosis, and (iv) congestive cardiac failure. The minor criteria include (i) systolic murmur grades I or II, (ii) abnormal second sound, (iii) abnormal electrocardiogram, (iv) abnormal X-ray, and (v) abnormal blood pressure. Presence of one major or two minor criteria is essential for indicating the presence of heart disease. Adherence to these criteria will also obviate unnecessary referrals and parental anxiety.

Multiple studies demonstrate the benefit of routine pulse oximetry screening for all newborns to detect unsuspected critical cyanotic CHD. Such screening has been endorsed by the American Academy of Pediatrics, American Heart Association, American College of Cardiology, and the March of Dimes.[18]

Diagnostic aids and modalities available are echocardiogram or transoesophageal echocardiogram, electrocardiogram, chest X-ray, cardiac catheterization, and magnetic resonance imaging methods are used to diagnose CHD.[10] These should be suitably utilized guided by the clinical cues.

The diligence and distinction for thoughtful detection and tactful diagnoses required is-

"*Clinical skills for suspicion,*

Sophisticated comprehensive skills for diagnosis"

Medical management

General health maintenance, including a well-balanced "heart-healthy" diet, aerobic exercise, and avoidance of smoking (active and passive) needs to be encouraged.

Most patients who have mild CHD should be made aware and advised that a normal life is expected. Even patients with moderate to severe heart disease need not be restricted from all physical activity, and these children tend to limit their own activities. A formal assessment of the child's physical activity capacity should be done in dedicated pediatric exercise laboratory. This should be ensured for conditions in which exercise testing has proved useful. These are unoperated or palliated cyanotic defects, suspected tachy-arrhythmias; after repair of aortic coarctation, tetralogy of Fallot, and Ebstein's anomaly; and post Fontan operation.[19] Physical activity should be guided according to the child's capacity, including recreational and competitive athletics. Caution is required for some patients with CHD that are at risk for cardiac decompensation and sudden death, depending on specific lesions and their severity.[20] The conditions at highest risk during strenuous activity are conditions with severe ventricular outflow obstruction, congestive heart failure, coronary insufficiency, pulmonary hypertension, hypertrophic cardiomyopathy, Marfan syndrome, and aortic dilation.[21]

Routine immunizations should be ensured. Inactivated influenza vaccination (either trivalent or quadrivalent), starting from 6 months of age, 2-4 weeks before influenza season: two doses at the interval of 1 month in the first year, and one dose annually thereafter is useful.[22] During respiratory syncytial virus season prophylaxis against it is recommended in young infants with unrepaired CHD and significant hemodynamic abnormalities.[23]

Bacterial endocarditis prophylaxis should be carried out during dental or oral surgical procedures for appropriate patients as per American Heart Association (AHA) guideline 2007.[24] It is no longer recommended for gastrointestinal or genitourinary procedures.

Cyanotic patients should be monitored for noncardiac manifestations of oxygen deficiency, particularly excessive polycythemia. Dehydration leads to increased viscosity and increases the risk of stroke. It needs to be prevented and promptly treated. Decompensation may occur rapidly during childhood respiratory or gastrointestinal infections, when respiratory function and enteral intake are impaired or fluid losses are increased. Diuretics should be used carefully or discontinued depending on hemodynamic status.

Maintaining adequate hemoglobin levels in all CHD patients is useful. Cyanotic patients may have a low mean corpuscular hemoglobin concentration despite polycythemia. Iron deficiency should be looked for and corrected.

Overall family physicians' and pediatricians' relevant role rhetoric is-

"*Medical management is best for some severity,*

Medical management at its best for all stages in the life-cycle of all severities"

Interventional techniques and advances

Pediatric interventional cardiology is making rapid strides. Many more types of CHDs can be now treated in the cardiac catheter laboratory than ever before.[25] Advances in cardiac imaging make possible complex interventional procedures with precision. Currently defect closure, dilatation, valve implantation, and hybrid techniques are used. Understanding of procedure done is a prerequisite of follow-up care.

Defect closure is commonly done for atrial septal defect, patent duct arteriosus (PDA), and abnormal vascular connections. Substantial experience has accumulated with device closure of membranous and muscular ventricular septal defects, as well as more complex shunts such as baffle leaks after atrial switch repair and ventricular pseudo-aneurysms.[26]

Dilatation is performed for pulmonary valvular stenosis, aortic valvular stenosis, and restenosis of coarctation of aorta after earlier surgery. Other indications of dilatation include amelioration of mitral stenosis, branch pulmonary artery narrowing, and dilation of systemic or pulmonary venous obstructions. Intravascular

stents have better results in branch pulmonary stenosis and these are also used in coarctation of the aorta.

Trans-catheter valve implantation is a great advance. Presently, the most common application in children is replacement of the pulmonary valve (melody valve) in patients who have had prior surgery for tetralogy of Fallot (usually because of residual pulmonary insufficiency). Other indication currently being studied is tricuspid insufficiency.[27]

Hybrid approach involves the interventional technique along with surgery. It improves the outcomes by overcoming the limitations of the two approaches. Initial hybrid interventions included intraoperative stenting of pulmonary arteries and per-ventricular device closure of ventricular septal defects.[28] The perventricular approach for closure of membranous and muscular ventricular septal defects (VSDs) using hybrid methodology obviates the need of cardiopulmonary bypass. Other hybrid procedure indications are hypoplastic left heart syndrome, pulmonic valve replacement,[29] intraoperative stenting,[30] and modified hemi-Fontan operation and subsequent nonsurgical Fontan completion.[31]

The spirit and success is best summarized as-

"*Minimal invasiveness, Maximum benefits*"

Surgical strategies: Fruitfulness and follow-up

Curative surgery is possible for PDA, secundum atrial defect, and uncomplicated ventricular septal defect. After surgical correction in early childhood long-term sequelae are rare.

Reparative surgery is performed for aortic stenosis, atrioventricular canal, coarctation of the aorta, partial anomalous pulmonary venous return, pulmonary stenosis, tetralogy of Fallot, total anomalous pulmonary venous return, d-transposition of the great arteries, and l-transposition. These patients improve after surgery; however, some have life-long sequelae and some have significant late impairment. All this needs medical support.

Palliative surgery is performed for Eisenmenger syndrome, hypoplastic left heart syndrome, malaligned atrioventricular canal with single ventricle repair, single ventricle, tricuspid atresia, and unrepaired cyanotic heart disease. These surgeries do not correct the underlying defects and patients have significant late impairment. Monitoring and suitable medical support is required.

With improvements in interventional pediatric cardiology, surgery, and palliation of more complex lesions, the number of survivors of CHD is increasing.[32] Even with evolving best expertise, residual defects after surgical repair may contribute to functional impairment, which needs to be managed by highest medical competencies.

Follow-up strategies need to be clearly out-lined. These should include monitoring and management plan for primary care providers and family physicians, as well as guidelines for parents. It is important that discharge summary includes ten points information and action plan (10Ds - IAP), details at Table 1.

Surgical progress with microsurgical techniques is leading the way for precision, perfection, and fewer complications. Surgical aims are summarized as-

"*Restoring anatomy* and *physiology,*

Rewarding Cardio-vascular surgery"

Ensuring liveliness

Cardiovascular health is enhanced in almost all children with CHD by promoting tolerated physical activity. Avoiding a sedentary lifestyle and obesity is important.[33] Various factors contributing to these need to be suitably addressed.

Intrinsic and extrinsic factors limit functioning, whether it is play, school activities, or work ability in CHD, and need to be taken care of. Intrinsic factors may include arrhythmia, chronic heart failure, depression, learning disability, and pulmonary hypertension. Extrinsic factors may include intolerance of lifting weight, intolerance of heat or humidity, lack of stamina, and decreased aerobic capacity.[32] Intrinsic factors need to be addressed with medical management. Extrinsic factors effects can be modified with the motto of making best use of individual capabilities.

Adults with CHD need to be monitored and managed by categorizing disease severity comprehensively using the Adult CHD Anatomic and Physiological classification system (ACHD AP Classification).[34] Treatment and activity recommendations should be according to current classification with periodic assessments. The reasoning for this is that patients may move from

Table 1: Ten point information and action plan (10Ds - IAP)

Action no	Details
1	*D*iagnosis with details of management including intervention/ surgery, and management of residual defects if any
2	*D*ata of anthropometry and vitals, for monitoring progress and early detection of deterioration. This should include oxygen saturation measurements
3	*D*rugs prescribed with dosages
4	*D*iligent activities plan based on functional capacity
5	*D*ietary recommendations
6	*D*esired monitoring and investigations schedule
7	*D*edicated immunization schedule, routine as well as specially indicated
8	*D*istinctive care plan during concurrent illness
9	*D*etailed emergency action plan with cardiopulmonary resuscitation (CPR) steps, automatic external defibrillator use, and emergency use medications
10	*D*efinitive emergency contact details, including ambulance service provider

one ACHD AP classification to another over time similar to the New York Heart Association classification of functional status. Women with CHD should receive counseling regarding risks prior to conceiving with focused evaluation for functional capacity. Functional capacity influences maternal and fetal outcomes.

Advances in the care of patients with CHD have resulted in an older patient population with complex cardiovascular conditions, presenting challenges for health care delivery.[35] Research with data analytics of multicentric life-long data should lead to best practices and better personalized medicine.

The Armed Forces scenario involves frequent movements and to difficult terrains with adverse weather conditions. High altitudes and sudden changes in the thermal environment are deleterious and should be avoided. Prudent policies and particular care for affected children needed. Staged acclimatization and special monitoring should be carried out. Parents need to be made aware of warning signs of deterioration and advised to seek early medical attention.

All our efforts for the best of health and happiness for children with CHD can be summarized as-

"*Making child fit for the environment,* and

Modifying the environment for the child"

COVID-19 and congenital heart disease

Although the respiratory system is the main site of infection during COVID-19, atypical presentations in children have been reported.[36] CHD patients are known to have higher risk for complications with viral illnesses, including respiratory syncytial virus and influenza, hence concern with COVID-19. Primary care and family physicians assume an important role because of their close proximity to the population and importantly when travel restrictions exist.

Increased risk for COVID-19 likely in (i) infants with unrepaired significant CHD (ii) those with chronic cyanosis and depressed ventricular function, (iii) individuals with severe pulmonary hypertension, (iv) immune-compromised patients (including those who have undergone heart transplantation), (v) single ventricle patients after Fontan operation, and (vi) adults with CHD complicated by coronary artery disease or systemic hypertension.[37]

Low risk is in asymptomatic CHD children, those with normal heart rate, rhythm, size and shape, and those with no pulmonary hypertension, and normal heart valves.

Even if low risk, having CHD is a priority indication for vaccination.

It is recommended that all cardiac medications, including aspirin, anticoagulants, angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers, beta blockers, diuretics, and antiarrhythmic medications be continued during COVID-19 illness, unless a clear contraindication develops.

The clinician must be cognizant of QT prolonging effects of some of the medications that are used in COVID-19 therapy, including hydroxychloroquine.

Conclusion

Timely diagnosis, technologically advanced treatment, tactful supportive follow-up strategies can ensure healthy well-being of CHD children. Basic clinical excellence and best comprehensive expertise from primary to tertiary level of healthcare is the need for children with CHD. Raising awareness and realizing goals with strategies suggested will go a long way in ensuring-

"*Health, happiness, liveliness,* and *longevity*"

Key Messages

- Professional approach for early detection and expert diligence
- Prospective prevention including environmental factors attention
- Progress in correction of defects phenomenal, subsequent care with "Ten-Points Information and Action Plan"
- Particular care required for affected Armed Forces personnel's children
- Physical activity guided by functional ability ensures liveliness.

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Conflicts of interest

There are no conflicts of interest.

References

1. Mitchell ME, Sander TL, Klinkner DB, Tomita-Mitchell A. The molecular basis of congenital heart disease. Semin Thorac Cardiovasc Surg 2007;19:228-37.
2. Meshram RM, Gajimwar VS. Prevalence, profile, and pattern of congenital heart disease in Central India: A prospective, observational study. Niger J Cardiol 2018;15:45-9.
3. Bernstein D. Epidemiology and genetic basis of congenital heart disease. In: Kliegman RM, St. Geme JW, Blum NJ, Shah SS, Tasker RC, Wilson KM, editors. Nelson Textbook of Pediatrics. 21st ed. Philadelphia, PA: Elsevier; 2020. p. 9336-49.
4. Blue GM, Kirk EP, Sholler GF, Harvey RP, Winlaw DS. Congenital heart disease: Current knowledge about causes and inheritance. Med J Aust 2012;197:155-9.
5. Khatami AD. Advances and research in congenital heart disease. Transl Pediatr 2016;5:109-11.

6. Fesslova V, Brankovic J, Lalatta F, Villa L, Meli V, Piazza L, *et al.* Recurrence of congenital heart disease in cases with familial risk screened prenatally by echocardiography. J Pregnancy 2011;2011:368067.
7. Hoffman JIE. The global burden of congenital heart disease. Cardiovasc J Afr 2013;24:141-5.
8. Lin JP, Aboulhosn JA, Child JS. Congenital heart disease in adolescents and adults. In: Fuster V, Harrington RA, Narula J, Eapen ZJ, editors. Hurst's The Heart. 14th ed. New York: McGraw-Hill Medical; 2017. p. 1363-94.
9. Belmont JW. Recent progress in the molecular genetics of congenital heart defects. Clin Genet 1998;54:11-9.
10. Sun R, Liu M, Lu L, Zheng Y, Zhang P. Congenital heart disease: Causes, diagnosis, symptoms, and treatments. Cell Biochem Biophys 2015;72:857-60.
11. Suhrie KR, Tabbah SM. Medication and teratogen exposure. In: Kliegman RM, St. Geme JW, Blum NJ, Shah SS, Tasker RC, Wilson KM, editors. Nelson Textbook of Pediatrics. 21st ed. Philadelphia, PA: Elsevier; 2020. p. 3825-36.
12. Webb GD, Small Horn JF, Therrien J, Redington AN. Congenital heart disease. In: Bonow RO, Mann DL, Zipes DP, Libby P, editors. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 10th ed. Philadelphia: Elsevier Saunders; 2015. p. 1391-445.
13. Ye Z, Wang L, Yang T, Chen L, Wang T, Chen L, *et al.* Maternal viral infection and risk of fetal congenital heart diseases: A meta-analysis of observational studies. J Am Heart Assoc 2019;8:e011264.
14. Saxena A. Congenital heart disease in India: A status report. Indian J Pediatr 2005;72:595-8.
15. Jain S, Chandra N, Thapar RK. Paediatric surgery experiences of a tertiary referral hospital: International classification of diseases spectrum for teaching, planning, and scaling up services. Indian J Child Health 2019;6:313-9.
16. Moss AJ. Clues in diagnosing congenital heart disease. West J Med 1992;156:392-8.
17. Kumar RK, Raj M. Disorders of cardiovascular system. In: Paul VK, Bagga A, editors. Ghai Essential Pediatrics. 9th ed. New Delhi: CBS; 2019. p. 394-459.
18. Bernstein D. Evaluation and screening of the infant or child with congenital heart disease. In: Kliegman RM, St. Geme JW, Blum NJ, Shah SS, Tasker RC, Wilson KM, editors. Nelson Textbook of Pediatrics. 21st ed. Philadelphia, PA: Elsevier; 2020. 9350-8.
19. Fleg JL, Pina IL, Balady GJ, Chaitman BR, Fletcher B, Lavie C, *et al.* Assessment of functional capacity in clinical and research applications: An advisory from the Committee on Exercise, Rehabilitation, and Prevention, Council on Clinical Cardiology, American Heart Association. Circulation 2000;102:1591-7.
20. Maron BJ, Zipes DP, Kovacs RJ. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: Preamble, principles, and general considerations: A scientific statement from the American Heart Association and American College of Cardiology. Circulation 2015;132:e256-61.
21. Lantin-Hermoso MR, Berger S, Bhatt AB, Richerson JE, Morrow R, Freed MD, *et al.* The care of children with congenital heart disease in their primary medical home. Pediatrics 2017;140:e20172607.
22. Balasubramanian S, Shah A, Pemde HK, Chatterjee P, Shivananda S, Guduru VK, *et al.* Indian Academy of Pediatrics (IAP) Advisory Committee on Vaccines and Immunization Practices (ACVIP) recommended immunization schedule (2018-19) and update on immunization for children aged 0 through 18 years. Indian Pediatr 2018;55:1066-74.
23. Bernstein D. General principles of treatment of congenital heart disease. In: Kliegman RM, St. Geme JW, Blum NJ, Shah SS, Tasker RC, Wilson KM, editors. Nelson Textbook of Pediatrics. 21st ed. Philadelphia, PA: Elsevier; 2020. p. 9571-603.
24. Wilson W, Taubert KA, Gewitz M, Lockhart PB, Baddour LM, Levison M, *et al.* Prevention of infective endocarditis: Guidelines from the American Heart Association: A guideline from the American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group. Circulation 2007;116:1736-54.
25. Kim SH. Recent advances in pediatric interventional cardiology. Korean J Pediatr 2017;60:237-44.
26. Wilson W, Osten M, Benson L, Horlick E. Evolving trends in interventional cardiology: Endovascular options for congenital disease in adults. Can J Cardiol 2014;30:75-86.
27. Bernstein D. Diagnostic and interventional cardiac catheterization. In: Kliegman RM, St. Geme JW, Blum NJ, Shah SS, Tasker RC, Wilson KM, editors. Nelson Textbook of Pediatrics. 21st ed. Philadelphia, PA: Elsevier; 2020. p. 9325-34.
28. Bearl DW, Fleming GA. Utilizing hybrid techniques to maximize clinical outcomes in congenital heart disease. Curr Cardiol Rep 2017;19:72.
29. Simpson KE, Huddleston CB, Foerster S, Nicholas R, Balzer D. Successful subxyphoid hybrid approach for placement of a Melody percutaneous pulmonary valve. Catheter Cardiovasc Interv 2011;78:108-11.
30. Ungerleider RM, Johnston TA, O'Laughlin MP, Jaggers JJ, Gaskin PR. Intraoperative stents to rehabilitate severely stenotic pulmonary vessels. Ann Thorac Surg 2001;71:476-81.
31. Konertz W, Schneider M, Herwig V, Kampmann C, Waldenberger F, Hausdorf G. Modified hemi-Fontan operation and subsequent nonsurgical Fontan completion. J Thorac Cardiovasc Surg 1995;110:865-7.
32. Institute of Medicine (US) Committee on Social Security Cardiovascular Disability Criteria. Cardiovascular Disability: Updating the Social Security Listings. Washington (DC): National Academies Press (US); 2010.
33. Lantin-Hermoso MR, Berger S, Bhatt AB, Richerson JE, Morrow R, Freed MD, *et al.* The care of children with congenital heart disease in their primary medical home. Pediatrics 2017;140:e20172607.
34. Stout KK, Daniels CJ, Aboulhosn JA, Bozkurt B, Broberg CS, Colman JM, *et al.* 2018 AHA/ACC guideline for the management of adults with congenital heart disease: A report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation 2018;139:e698-800.
35. Diller GP, Arvanitaki A, Opotowsky AR, Jenkins K, Moons P, Kempny A, *et al.* Lifespan perspective on congenital heart disease research: JACC State-of-the-Art review. J Am Coll Cardiol 2021;77:2219-35.

36. Haji Esmaeil Memar E, Pourakbari B, Gorgi M, Sharifzadeh Ekbatani M, Navaeian A, Khodabandeh M, *et al*. COVID-19 and congenital heart disease: A case series of nine children. World J Pediatr 2021;17:71-8.
37. Alsaied T, Saidi A. COVID-19 in congenital heart disease: Ten points to remember. American College of Cardiology Expert analysis. 2020. Available from: https://www.acc.org/latest-in-cardiology/articles/2020/06/04/10/53/covid-19-in-congenital-heart-disease. [Last accessed on 2021 Jun 27].