CHILDHOOD DEVELOPMENTAL SCREENING 2020
https://doi.org/10.33591/sfp.46.5.u6

UNIT NO. 6

# CHILDHOOD CARDIAC CONDITIONS

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## ABSTRACT

**Detection of congenital heart disease (CHD) remains the main focus in the screening of childhood cardiac conditions. History taking and physical examination form the backbone of this screening process. Referral to a paediatric cardiologist is required for diagnosis and management. There is a need to understand which referrals are urgent. Increasingly, the primary care clinician will encounter the growing pool of post-operative CHD and will need to be familiar with the potential morbidities of this group.**

**Kawasaki Disease is the most common acquired childhood cardiac condition. There should be a high index of suspicion in a child with prolonged fever and a constellation of rash, conjunctivitis, mucositis, dactylitis and cervical lymphadenopathy. Timely diagnosis and treatment prevent coronary artery complications.**

**The primary care physician should also be on the alert to recognise children with cardiac arrhythmias, acute myocarditis, cardiomyopathies and valvular heart disease. Other rare conditions include idiopathic pulmonary arterial hypertension. Screening for risk factors for sudden cardiac death may also be important, particularly for children involved in competitive sports. Primary prevention of coronary artery disease and metabolic syndrome starts in childhood. The "at-risk" child should be identified early and healthy lifestyle and diet promoted.**

**Keywords: Screening, congenital heart disease, Kawasaki Disease, Obesity, chest pain**

**SFP2020; 46(5) : 38-43**

## INTRODUCTION

The screening of childhood cardiac conditions tends to focus on congenital heart disease (CHD). However, the landscape of cardiac diseases in children is changing. Apart from acquired heart diseases such as Kawasaki Disease (KD), children should also be screened for pre-cursors to adult-type cardiovascular diseases such as coronary artery disease, hypertension and stroke. As the incidence of obesity is rising, risk factors for the development of metabolic syndrome should also be looked into and addressed in childhood. More young children are being seen for chest pain and palpitations. Identifying red flags in the symptoms and signs is important in differentiating psychosomatic complaints from harbingers of sinister cardiac diseases. More children are participating competitively in sports at a younger age. The primary care physician may need to be familiar with cardiovascular screening in young athletes and risk factors for sudden cardiac death. Connective tissue disorders, e.g. Marfan syndrome, are also associated with potentially life-threatening cardiac conditions. This group should also be identified for screening. A detailed history and thorough physical examination form the backbone in the screening for childhood cardiac conditions.

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## Congenital Heart Disease

The incidence of congenital heart disease (CHD) is approximately one percent of all live births, and approximately 25 percent have critical CHD, likely requiring surgery in the first year of life.[1] Approximately two-thirds of babies with critical CHD survive to adulthood. In comparison, almost all babies with non-critical CHD survive to adulthood. A significant proportion of those with critical CHD has some form of physical or learning disability or impairment. The outcome for critical CHD is much improved where there is early and timely detection, and presentation near extremis is avoided. For this purpose, the screening processes for (critical) CHD are now set in place.

Screening begins in the antenatal period, as early as the first trimester. Nuchal fold thickness and abnormal genetic tests alert the obstetrician to the possibility of CHD. Foetal anomaly scans are done and, where indicated, a referral is made to a paediatric cardiologist for diagnosis on foetal echocardiography and counselling. A more definitive chromosomal and/or genetic test may be offered. Referral to a paediatric cardiologist should also be considered if there is a history of complex CHD in a family member or a previous pregnancy. Rhythm abnormalities are also commonly detected.

Babies delivered in hospitals are screened prior to discharge, with close attention paid to cardiac murmurs, abnormal heart sounds and femoral pulses. Pulse oximetry is now routinely used as a screening tool for critical CHDs.[2] Oxygen saturation by pulse oximetry (SpO2) is measured after 24 hours of life in the right upper limb and one lower limb. The SpO2 should be ≥95 percent for both limbs with ≤three percent discrepancy, failing which, the neonate should be investigated for causes of hypoxaemia, and urgent referral to a paediatric cardiologist may be warranted. As the overall sensitivity of pulse oximetry for detection of critical CHD is approximately 76 percent, neonates may still present to the emergency department in extremis if they have a duct-dependent systemic or pulmonary circulation.[3] Some examples include coarctation of the aorta,

interrupted aortic arch, hypoplastic left heart syndrome, critical aortic stenosis, Fallot's tetralogy, pulmonary atresia, critical pulmonary stenosis, tricuspid atresia and severe Ebstein anomaly. Obstructive forms of total anomalous pulmonary venous drainage, transposition of the great arteries and truncus arteriosus may also present within the first week of life.

At each encounter with a newborn, the primary care physician must continue to be alert and identify the patient with critical CHD but passed the pulse oximetry screening. A thorough physical examination is the key to this. Urgent referral to a paediatric cardiologist should be made upon detection of central cyanosis (which may be mild), baseline tachypnea, retractions, additional heart sounds (e.g. gallop rhythm), prominent cardiac murmurs and weak or absent femoral pulses as cardiovascular collapse may be imminent. In general, in the first four to six weeks of life, early referral to a paediatric cardiologist for evaluation is recommended when an asymptomatic murmur is detected.

Screening for CHD continues throughout childhood and adolescence as the milder forms of CHD can present at any age. Classically, the pansystolic murmur (PSM) of the ventricular septal defect (VSD) appears nearer at one month of life when the pulmonary pressures physiologically drop to normal. This allows blood to flow from the left ventricle to the right ventricle at a higher velocity, generating the murmur. Likewise, the ejection systolic murmur (ESM) of pulmonary stenosis becomes more apparent at one month of life. In some CHDs (e.g. aortic stenosis), the murmur may initially be soft when the lesion is mild and then gets louder as it progresses. Some CHDs (e.g. partial anomalous pulmonary venous drainage) have very soft, musical murmurs that are difficult to differentiate from innocent murmurs. In such cases, the physician will need to rely on the presence of concomitant physical signs such as cyanosis, failure to thrive, tachypnoea, retractions, precordial bulge, parasternal heave, deviation of the apex beat, weak femoral pulses and radio-femoral delay, fixed splitting of the second heart sound, ejection and mid systolic clicks, mid-diastolic murmurs, gallop rhythm and hepatomegaly, The key to screening and diagnosis in CHD lies in a detailed physical examination.

The primary care physician will increasingly see a growing pool of children who had cardiac surgery or transcatheter intervention for CHD. Simple lesions such as ASD and VSD are associated with very good prognosis post-operatively and usually do not require any exercise restriction. Most of the other complex CHDs require long-term follow-up and further intervention. They are also prone to developing malignant arrhythmias. In general, they should be exempted from strenuous physical activities and competitive sports and; most continue to require antibiotic prophylaxis against infective endocarditis for dental and surgical procedures. Table 1 provides some differential diagnosis for auscultatory findings in children with CHD.

**Table 1. Auscultatory Findings and their Differential Diagnoses for CHD**

| Location | Physical Sign | Native Lesion | Post-op Finding |
|---|---|---|---|
| Apex | S4 gallop | VSD<br>PDA | |
| | Mid systolic Click | MVP | |
| | Mechanical click | | Mitral valve replacement |
| | PSM | MR<br>AVSD | MR<br>AVSD |
| | MDM | MS | AVSD (MS) |
| | EDM | AR | |
| LLSE | S3 gallop | Normal | |
| | S4 gallop | ASD<br>Primum ASD (AVSD) | |
| | Mid-systolic click | TVP<br>Ebstein anomaly | |
| | PSM | VSD<br>TR<br>AVSD | |
| | MDM | Ebstein anomaly<br>Rhabdomyoma | |
| ULSE | S2 – fixed split | ASD<br>Primum ASD (AVSD) | |
| | P2 - loud | PHT<br>Eisenmenger syndrome | |
| | Single S2 | Absent pulmonary valve syndrome<br>Pulmonary atresia<br>Truncus arteriosus | Post-op FT<br>Post Pulmonary valvotomy<br>Post-op Pulmonary atresia |
| | Ejection systolic click | Pulmonary valve stenosis<br>Truncus arteriosus | RV-PA conduit |
| | ESM | Innocent murmur<br>PS<br>LPAS<br>ASD<br>Primum ASD (AVSD)<br>TAPVD / PAPVD<br>CoA<br>FT<br>Complex CHD + PS e.g. DORV + PS | PS (residual PS in Post-op FT or other complex CHD) |
| | EDM | PR<br>AR | FT (PR) |
| | Continuous murmur | Venous hum<br>PDA<br>APW<br>CAVF | Left BT shunt<br>Central shunt |
| URSE | Ejection systolic click | Bicuspid aortic valve<br>Aortic valve stenosis | |
| | Mechanical click | | Aortic valve replacement |
| | ESM | AS<br>RPAS | AS |
| | EDM | AR | AR |
| | Continuous murmur | CAVF | Right BT shunt |
| Left infraclavicular region | Continuous murmur | Venous hum<br>PDA | Left BT shunt |
| | ESM | CoA | |
| Right infraclavicular region | Continuous murmur | Venous hum | Right BT shunt |
| Interscapular region | ESM | CoA | |
| | Continuous murmur | Collateral arteries | |

APW- aortopulmonary window; AR- aortic regurgitation; AS-aortic stenosis; ASD-atrial septal defect; AVSD-atrioventricular septal defect; BT shunt-Blalock-Taussig shunt; CAVF- coronary arteriovenous fistula; CHD-congenital heart disease; CoA-Coarctation of the aorta; DORV-double outlet right ventricle; EDM-Early diastolic murmur; ESM-Ejection systolic murmur; FT-Fallot's tetralogy; LLSE-lower left sternal edge; LPAS-left pulmonary artery stenosis; MDM-mid-diastolic murmur; MR-mitral regurgitation; MS-mitral stenosis; MVP-mitral valve prolapse; PAPVD- partial anomalous pulmonary venous drainage; PDA-patent ductus arteriosus; PHT-pulmonary hypertension; PR-pulmonary regurgitation; PS-pulmonary stenosis; RPAS-right pulmonary artery stenosis; RV-PA conduit-right ventricle to pulmonary artery conduit; TAPVD-total anomalous pulmonary venous drainage; TR-tricuspid regurgitation; TVP-tricuspid valve prolapse; ULSE-upper left sternal edge; URSE -upper right sternal edge; VSD- ventricular septal defect

## Kawasaki Disease

Kawasaki Disease (KD) is the most common acquired heart disease in children. Termed the "the great mimicker," the diagnosis of KD can be easily missed. KD is a clinical diagnosis that typically presents as a constellation of fever of at least five days with four out of five other criteria – generalised rash, non-suppurative conjunctivitis, mucositis, cervical lymphadenopathy and, erythema and oedema of the hands and feet. It typically occurs between six months to five years of age.

Some patients may have incomplete (or atypical) KD, especially if they fall outside this age range. There are guidelines for the diagnosis of incomplete (or typical) KD from the American Heart Association (AHA) and the Japanese Cardiac Society (JCS).[4,5] Investigations that support the diagnosis of incomplete KD include elevated inflammatory markers, elevated platelet counts, anaemia for age, elevated white cell count, hypoalbuminaemia, elevated alanine transferase (ALT) levels, and pyuria. (See Table 2) Other supportive features for KD include erythema and induration of the BCG scar, extreme irritability and hydrops of the gallbladder. Although KD is a clinical diagnosis, an echocardiogram is required to assess for coronary artery dilatation as well as mitral and aortic regurgitation, pericardial effusion and myocarditis. ECG changes may also be present. As KD is often triggered by a viral or bacterial infection, it is important to note that the diagnosis of a concomitant infection does not exclude the diagnosis of KD.

**Table 2. Diagnostic criteria for Classic and Incomplete Kawasaki Disease in children adapted from the American Heart Association (2017)[4]**

| Classic KD | Incomplete KD |
|---|---|
| Fever ≥five days<br>AND the presence of ≥four of the principle clinical features<br>1. Erythema and cracking of the lips, strawberry tongue, and/or erythema of oral and pharyngeal mucosa<br>2. Bilateral bulbar conjunctival injection without exudate<br>3. Rash: maculopapular, diffuse erythroderma, or erythema multiforme-like<br>4. Erythema and oedema of the hands and feet in acute phase and/or periungual desquamation in subacute phase<br>5. Cervical lymphadenopathy (≥1.5cm diameter), usually unilateral | Children with fever ≥five days and two to three compatible clinical criteria<br>OR Infants with fever ≥seven days without other explanation<br><br>AND CRP ≥3.0 mg/dL and/or ESR ≥40 mm/hr<br><br>AND three or more laboratory findings:<br>1. Anaemia for age<br>2. Platelet count ≥450,000/mm$^3$ after day seven of fever<br>3. Albumin ≤3.0 g/dL<br>4. Elevated ALT level<br>5. WBC count ≥15,000/mm$^3$<br>6. Urine ≥10 WBC/hpf<br><br>OR Positive echocardiogram |

Intravenous immunoglobulin (2g/kg) with an anti-inflammatory dose of aspirin (50-100mg/kg/day in three to four divided doses) is the standard treatment for KD. The treatment is best given before day ten of illness. This reduces the overall risk of coronary artery complications from 25 percent to five percent. In the convalescent phase of KD, aspirin is maintained at the anti-platelet dose (3-5 mg/kg/dose once daily) for at least a further four to six weeks. Partial response to treatment and recurrence of KD should be highlighted to parents. The risk of recurrence is highest in the first two years after acute Kawasaki disease.

In some children, the diagnosis of KD may become apparent only when periungual desquamation of the fingers and toes occur in the subacute phase. Retrospectively, the pieces of the jigsaw then fall into place. Laboratory findings at this stage usually show thrombocytosis and elevated ESR. CRP levels are on the decline but may still be elevated. An echocardiogram should be done to screen for cardiac complications. Thromboprophylaxis is usually given for four to six weeks as a precautionary measure even in the absence of coronary artery abnormalities.

Primary care physicians play a role in the long-term care of patients who have recovered from acute KD. Although it is unclear whether long term cardiovascular abnormalities occur in patients who did not have coronary artery involvement in the acute phase of KD, the American Heart Association deems it reasonable to discharge patients from paediatric cardiology care a year after the acute episode. A cardiovascular risk assessment by the primary care physician is recommended annually, and the need to maintain a healthy lifestyle and dietary habits should be emphasised to the parents and patient.

## Obesity, Metabolic Syndrome & Cardiovascular Risk Factors

Obesity in childhood and adolescence is a risk factor for obesity in adulthood and is associated with significant disease burden. As its prevalence is rising, the primary care physician plays a vital role in prevention. Although interventions may not result in a significant change in body mass index (BMI), it is still important to encourage a healthy lifestyle and dietary habits.[6] Parental obesity, maternal gestational diabetes and smoking during pregnancy are important prenatal risk factors, and expectant mothers should be counselled accordingly.

With obesity, there is an increased risk of coronary artery disease (CAD), type 2 Diabetes Mellitus (T2DM), hypertension, dyslipidaemia and the metabolic syndrome (MetS).[7] There are controversies in defining MetS in childhood. However, the importance lies in identifying paediatric cardiometabolic risks.[8] These include a family history of CAD, hypertension and T2DM, obesity; ethnicity; low birth weight, especially with rapid catch-up growth and rebound adiposity; short sleep duration, low physical activity levels, excessive screen time, tobacco smoke exposure and; the presence of acanthosis nigricans.

Weight, height, body mass index (BMI) and waist circumference (WC) should be measured and tracked regularly. WC percentiles for Singaporean children and adolescents are now available.[9] The overweight or obese child and the child who is on the path to becoming overweight (BMI trending toward the 85$^{th}$ percentile) should be referred for weight management. The co-morbidities of obesity, such as obstructive sleep apnoea syndrome (OSAS), polycystic ovarian syndrome (PCOS), and non-alcoholic fatty liver disease (NAFLD), should be identified and treated. Where risk factors for MetS are present, the child should be screened for T2DM, hypertension and dyslipidaemia.[8] Fasting glucose and/or glycated haemoglobin [HbA1c] should be considered once every three years starting age ten or at the onset of puberty, whichever is sooner. Blood pressure (BP) should be obtained annually, starting at three years of age. (Reference ranges for gender, age and height are available.[10]) Fasting lipid profile should be considered in obese children aged two to eight years and repeated at 12-16 years of age.[7]

The patient and parents should be encouraged to adopt a healthy lifestyle. Children should have 20-60 minutes of vigorous short bursts of physical activity per day, at least three

to five days per week. Healthy sleeping habits and limiting screen time (to less than two hours a day) also help prevent obesity. Children should be encouraged to eat more fruits and vegetables, consume less dietary fats, and avoid carbonated drinks and processed foods.[6] The American Academy of Pediatrics recommends that fruit juice intake should be limited to 4-6 ounces a day for children six months to six years and 8-12 ounces per day for older children. In fact, it would be best to replace fruit juice with whole fruits for additional nutritional value. In addition, the primary care physician should also screen obese children for depression and other mood disorders.

### Common complaints in a young child

From time to time, the primary care physician may encounter the child with chest pain, palpitations and breathlessness. Often, the causes of chest pain are benign and are idiopathic or musculoskeletal. Generally, psycho-emotional stress factors are present. Classically, the precordial catch presents as sharp, short, localised central chest pain that is unrelated to physical activity and is not associated with other symptoms. Some encountered concurrent diagnoses are bronchitis, pneumonia, oesophagitis, and referred pain from the abdominal causes. Approximately five percent of cases are attributable to cardiac issues such as structural abnormalities, pericarditis, myocarditis, arrhythmias and cardiomyopathies.[11]

Though rare, cardiac causes are potentially fatal, and the red flags in history and physical examination need to be recognised. Pressing, dull and severe central chest pain lasting more than 20 minutes that is associated with symptoms of haemodynamic compromise (cold and sweaty extremities, pallor, light-headedness, and near syncope) often indicate a cardiac cause. Palpitations and shortness of breath may be present. Chest pain that occurs consistently with exertion also warrants further investigations. When syncope occurs during exertion, all physical activity should be stopped and an urgent referral made to the paediatric cardiologist. Potential underlying causes may include an anomalous coronary artery (e.g. inter-arterial course of the right or left coronary artery and intramural course of the left anterior descending artery) or idiopathic pulmonary arterial hypertension. The child should also be evaluated for risk factors for sudden cardiac death (see below). Other uncommon but important causes of chest pain include uncontrolled asthma and depression with suicide risk.

### Cardiovascular Screening in Young Athletes and the risk of Sudden Cardiac Arrest

More children are taking up sports at a competitive level at a younger age. Some parents are concerned that the child may be at risk of sudden cardiac arrest (SCA) and request for cardiovascular screening. The more common causes of SCA in children and young adults are hypertrophic cardiomyopathy (HCM), coronary artery abnormalities, rhythm disorders and acute myocarditis. Other less common causes include congenital heart disease, aortic rupture and aortic stenosis. A family history of SCA, premature unexpected death or syncope is a risk factor. The child should be screened for symptoms of dizziness, chest pain, syncope, palpitations and dyspnea, as well as abnormalities on cardiovascular examination. An echocardiogram to assess for aortic root dilatation should be considered in patients with evolving features of Marfan syndrome and other connective tissue disorders. A baseline ECG would be useful to identify patients with Wolff-Parkinson-White syndrome, long QT syndrome, Brugada syndrome and various degrees of heart block. Where necessary, referral to a paediatric cardiologist should be made for further assessment. However, it is important to emphasise to the parents that despite detailed evaluation, not all SCAs can be foreseen and predicted. At present, no standardised screening protocol has been agreed on and proven to be effective.[12]

## CONCLUSION

Childhood cardiac conditions can present at any age. (See table 3) Careful attention should be paid to potentially life-threatening conditions. Recognition of red flags in the history and physical examination is paramount. Timely referral to the paediatric cardiologist can avert a cardiovascular collapse in the newborn and significantly reduce morbidities associated with congenital heart disease. Prevention of obesity and the development of other cardiovascular risk factors start in childhood. Regular tracking of BMI is important, and intervention should start once the BMI trends toward the 85$^{th}$ percentile. Parents must be involved in promoting a healthy lifestyle and dietary habits.

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**LEARNING POINTS**

- **A thorough and detailed physical examination is the key to screening in congenital heart disease.**
- **Primary care physicians play an important role in the management of obesity, metabolic syndrome and other risk factors (including a past history of Kawasaki Disease) that contribute to cardiovascular diseases in adulthood.**
- **The primary care physician should be aware of the red flag symptoms and signs that may be harbingers of sudden cardiac arrest in children presenting with chest pain, palpitations and breathing difficulty.**

**Table 3. Screening for Childhood Cardiac Conditions**

| Condition | Birth | 1-2 m | 6m-2y | 3-6y | 7-12y | 12-16y |
|---|---|---|---|---|---|---|
| **Congenital Heart Disease** | | | | | | |
| • Critical CHD | x | x | x | | | |
| • PS / AS / ASD / others | | x | x | x | x | x |
| • Anomalous Coronary arteries | | x | x | x | x | x |
| **Other Structural Heart Disease** | | | | | | |
| • MVP / MR | | | | | x | x |
| • Aortic Root Dilatation | (Rare) | (Rare) | (Rare) | (Rare) | x | x |
| • Bicuspid aortic valve | | | | | x | x |
| • Connective Tissue Disorders e.g. Marfan syndrome | (Rare) | | | | x | x |
| **Kawasaki Disease** | | | x | x | F/U | F/U |
| **Cardiomyopathies** | | | | | | |
| • HCM | (Rare) | (Rare) | (Rare) | | x | x |
| • DCM | (Rare) | (Rare) | | | x | x |
| **Acute Myocarditis** | | | x | x | x | x |
| **Rhythm Disorders** | x | x | (Rare) | (Rare) | (Rare) | x |
| **Idiopathic PAH** | | | | | x | x |
| **Obesity & MetS** | | | | Edn | x | x |
| • Risk factors/FHx | | | | x | x | x |
| • Lifestyle & Diet | | | | x | x | x |
| • BMI | | | | x | x | x |
| • WC | | | | | x | x |
| • Co-morbidities | | | | | x | x |
| • Laboratory tests (T2DM, dyslipidaemia) | | | | | x | x |
| **Hypertension** | | | | x | x | x |
| **Young athletes** | | | | | x | x |

*AS-aortic stenosis; ASD- atrial septal defect; BMI- Body Mass Index; CHD- congenital heart disease; DCM- dilated cardiomyopathy; Edn- Education; HCM- Hypertrophic cardiomyopathy; FHx- Family History; F/U- follow-up; MetS- Metabolic syndrome; MR- mitral regurgitation; MVP- mitral valve prolapse; PAH- pulmonary arterial hypertension; PS- pulmonary stenosis; T2DM- Type 2 Diabetes Mellitus; WC- Waist Circumference*