Chapter 7:

CARDIOVASCULAR DISEASES

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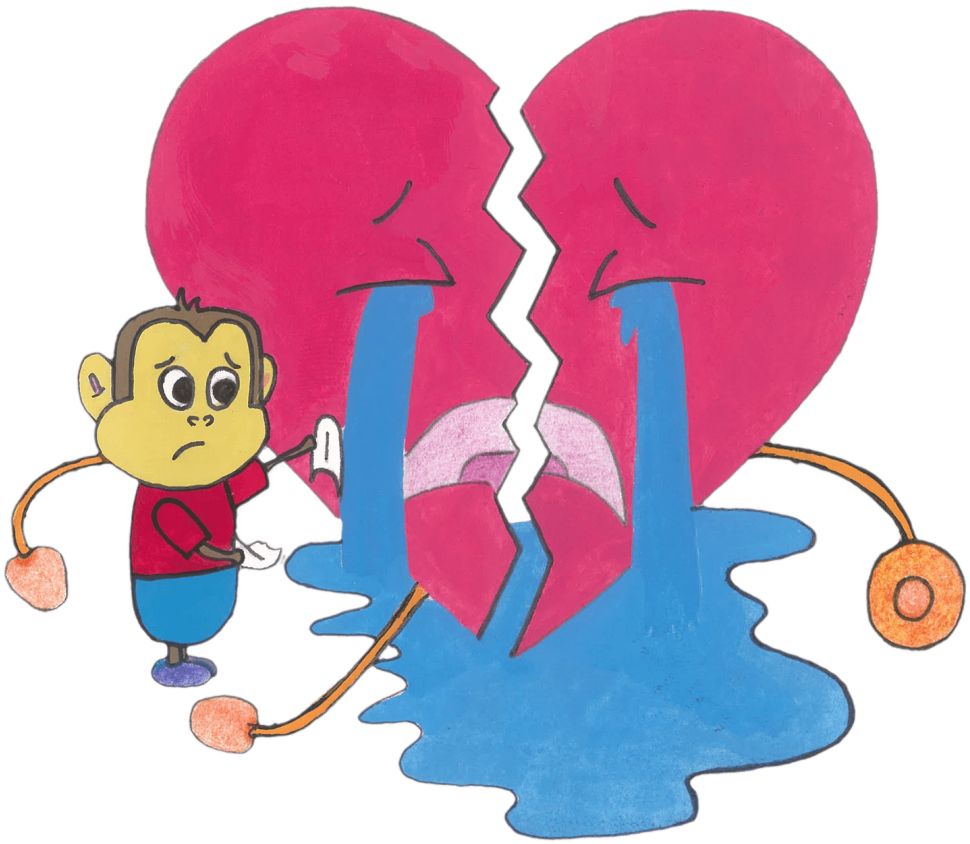

This chapter covers the following topics:

- Murmurs
- Acute rheumatic fever (ARF)
- Rheumatic heart disease (RHD)
- Cardiac failure
- Congenital heart disease
- Prevention of conditions which affect cardiovascular health

MURMURS

Murmurs are distinct sounds which arise when blood flows through narrowed or leaky cardiac valves, or through anatomical defects.

Pathophysiology

Systolic murmurs are heard in systole, between S1 and S2. These are further classified by their timing into early, mid or late systolic murmurs. Murmurs that last throughout systole are known as holo- or pansystolic murmurs. Systolic murmurs can be functional (benign). These are known as innocent murmurs and are common in infancy and childhood, but disappear in adulthood. Febrile or exertional states are commonly associated with these murmurs. The volume of a systolic murmur is graded on a six-point scale.

Diastolic murmurs can be classified into early diastolic (regurgitation through the aortic or pulmonary valves) or mid-to-late diastolic (in mitral or tricuspid stenosis). Diastolic murmurs are almost always pathological but are not as commonly heard as systolic murmurs in children with congenital cardiac defects. The volume of a systolic murmur is graded on a four-point scale. All murmurs (systolic and diastolic) louder than grade 3 are pathological.

Table 7.1: Six-point (Levine) Grading Scale for Systolic Murmurs

Grade	Volume	Thrill?
1/6	Barely audible	No
2/6	Soft	No
3/6	Easily audible	No
4/6	Easily audible	Yes
5/6	Can be heard with the stethoscope partially off the chest	Yes
6/6	Audible with stethoscope completely off the chest	Yes

Table 7.2: Four-point Grading Scale for Diastolic Murmurs

Grade	Volume	Thrill?
1/4	Soft	No
2/4	Easily audible	No
3/4	Can be heard with the stethoscope partially off the chest	Yes
4/4	Audible with stethoscope completely off the chest	Yes

See related figure available here.

Table 7.3: Listening Areas for Common Paediatric Heart Murmurs

Area	Murmur
Upper right sternal border	Aortic stenosis or regurgitation
Upper left sternal border	Pulmonary stenosis, pulmonary flow murmurs, right ventricular outflow tract obstructive murmurs, patent ductus arteriosus
Lower left sternal border	Still's murmur, ventricular septal defect, tricuspid valve regurgitation, hypertrophic cardiomyopathy, subaortic stenosis
Apex	Mitral regurgitation

Clinical Features

The clinical presentation of a child with heart disease depends on the cause and severity of the heart lesion and/or the presence of complications. Below is a list of important clinical features:

- Cyanosis
- Easy fatiguability (in paediatrics, it is often related to feeding)
- Poor exercise tolerance
- Failure-to-thrive (FTT; inability to meet and maintain appropriate growth standards)
- Diaphoresis (sweating)
- Wheezing in babies
- Chronic cough

Older children and teenagers may also present with:

- Dizziness
- Near-syncope or syncope
- Palpitations
- Chest pain

Investigations

The child with heart disease should have the following investigations:

- ECG
- Chest X-ray
- Echocardiogram

Management of Valvar Disease

The severity of the patient's symptoms and the severity of the lesion must be assessed (history, examination and investigations). In most valvar lesions, the patient's symptoms are treated medically until they worsen or a specific echocardiographic variable (e.g. chamber size, gradient across a valve) necessitates further surgical management i.e. reaches a recognisable threshold. Thereafter, surgery to repair or replace the valve is recommended.

ACUTE RHEUMATIC FEVER

Acute rheumatic fever (ARF) is an autoimmune, inflammatory process that develops as a sequela to a group A β-haemolytic streptococcal infection e.g. 'strep' throat or scarlet fever. Rheumatic fever mainly affects children aged 5-15 years with a history of group A β-haemolytic streptococcal infection. It develops several weeks after the pharyngitis has resolved in some children and adolescents (0.3-3% of pharyngitis cases); see also image with signs and symptoms of rheumatic fever here.

Pathophysiology

Molecular mimicry is responsible for the tissue injury observed in ARF. The humeral and cellular immune responses of an individual who is genetically predisposed are both implicated in the development of the disease. In this process, the patient's immune response (both B-cell- and T-cell-mediated immunity) is unable to differentiate between the invading microorganism and some host tissues, leading to inflammation. The resultant inflammation may persist well beyond the acute infection and produce the manifestations of ARF. Cardiac involvement is the most serious complication of ARF and causes significant morbidity and mortality.

Clinical Features and Diagnosis

The revised Jones criteria are used to diagnose ARF. ARF is diagnosed if the patient has 1 required criterion and one of the following:

- 2 major criteria
- 1 major and 2 minor criteria

The required criteria are evidence of recent streptococcal infection:

- Throat cultures growing group A β-haemolytic streptococcal infection
- Elevated antistreptolysin O titres

Table 7.4: 2015 (Revised) Jones Criteria

Low-Risk Population ARF incidence ≤2 per 100 000 school-aged children or all-age rheumatic heart disease (RHD) prevalence of ≤1 per 1000 population year	**Moderate/High-Risk Population** Children not clearly from a low-risk population
MAJOR CRITERIA	
Clinical and/or subclinical carditis	Clinical and/or subclinical carditis
Polyarthritis	Monoarthritis, polyarthritis and/or polyarthralgia
Sydenham's chorea	Sydenham's chorea
Erythema marginatum	Erythema marginatum
Subcutaneous nodules	Subcutaneous nodules
MINOR CRITERIA	
Prolonged PR interval	Prolonged PR interval
Polyarthralgia	Monoarthralgia
Fever (temperature ≥38.5°C)	Fever (temperature ≥38.5°C)
Inflammatory markers (peak ESR ≥60 mm in 1 hour and/or CRP ≥3.0 mg/dL)	Inflammatory markers (peak ESR ≥30 mm in 1 hour and/or CRP ≥3.0 mg/dL)
Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4832790/	

Note: Subclinical carditis is valvar involvement diagnosed on echocardiography while clinical carditis is the presence of a murmur consistent with aortic or mitral regurgitation.

Investigations

Relevant investigations include:

- Throat culture (usually negative by the time symptoms develop)
- Anti-streptococcal antibodies:
    - Symptoms of ARF develop when the antibodies are at their peak, so this test is very useful for detecting previous streptococcal infection. They should be checked at fortnightly in order to detect a rising titre.
    - Example of an extracellular antistreptococcal antibody – antistreptolysin O titre (ASOT).

    - Examples of intracellular antistreptococcal antibodies – anti-streptococcal polysaccharide, anti-teichoic acid antibody, anti-M protein antibody.
- Acute phase reactants – CRP and ESR (have a high sensitivity but low specificity for ARF)

Treatment

Primary Prevention

Administering a penicillin or another appropriate antibiotic for ARF in the patient with a sore throat (strep infection) decreases the risk of ARF by ~80%. One can prevent ARF with a 10-day course of oral penicillin V to treat pharyngitis. One may also give IM benzathine penicillin G or a benzathine/procaine penicillin combination. A single dose of IM is likely better to avoid non-compliance.
Should there be a penicillin allergy, one of the following antibiotics may be prescribed:

- Erythromycin
- Azithromycin for 5 days
- Clarithromycin for 10 days
- Narrow-spectrum (first-generation) cephalosporin for 10 days

Secondary Prevention

The aim here is to prevent additional streptococcal infections as patients with a history of ARF are at a high risk of recurrence, which may cause further damage to the heart. The patient should be given a course of antibiotics (penicillin G benzathine; if penicillin-allergic, erythromycin or sulfadiazine). The duration of the antibiotic course depends on his/her clinical features:

- ARF with carditis and clinically significant residual heart disease – for ≥10 years following the most recent episode (at least until the age of 40-45 years; usually lifelong prophylaxis).
- ARF with carditis and no residual heart disease besides mild mitral regurgitation – for 10 years or until 25 years old (whichever is longer).
- ARF without carditis – for 5 years or until 18-21 years old (whichever is longer).

The patient should also be given:

- Anti-inflammatory drugs (salicylates and corticosteroids)
    - It is important to avoid anti-inflammatories until the diagnosis of ARF is confirmed, as they may mask signs and symptoms essential to making the diagnosis.
- Analgesia (paracetamol for joint pain)

Patients must be closely monitored until all acute symptoms have resolved and they have returned to baseline function. ARF is a notifiable condition and all cases must be reported to the relevant local authority.

RHEUMATIC HEART DISEASE (RHD)

Pathophysiology

It is a cardiac manifestation of ARF (cardiac inflammation and scarring triggered by autoimmune reaction to a group A β-haemolytic streptococcal infection. In acute disease, the patient will have pancarditis [endo-, myo-, and pericarditis]). In chronic disease (2-10 years following acute rheumatic fever), the patient will have:

- Valvar fibrosis (leading to stenosis and/or valve insufficiency/regurgitation)
- Atrial dilatation
- Arrhythmias (including atrial fibrillation)
- Ventricular dysfunction (most serious complication of RHD)

The mitral valve is the most commonly affected valve and the aortic valve is the second most commonly affected valve. The tricuspid valve is only affected in 10% of cases and this is often in association with mitral and aortic lesions. The pulmonary valve is rarely affected. Severe valve insufficiency in the acute state may result in heart failure and/or even death.

Clinical Features

The clinical manifestations of RHD are the same as in ARF, with/without the signs and symptoms of the associated complications.

Investigations

An ECG may be done in addition to those for ARF (listed above). Sinus tachycardia often accompanies RHD, but sinus bradycardia may also occur. Other arrhythmias

may develop in RHD. ECGS may also help determine which chambers are enlarged.

Treatment

It may be

- Medical:
    - The aim here is to prevent recurrent attacks of rheumatic fever.
    - One may prescribe oral penicillin V (250mg twice daily), an oral sulphonamide (0,5-1 g twice daily) or erythromycin (250 mg twice daily).
- Surgical:
    - The affected valves may be surgically corrected or replaced.

CARDIAC FAILURE

Pathophysiology

Congestive cardiac failure (CCF) occurs when the heart fails to meet the body's metabolic demands at normal, physiologic venous pressure. Typically, the heart can compensate for increased demands by:

- Increasing the heart rate through humoral and neural responses
- Increasing ventricular contractility of the ventricles (secondary to the release and circulation of catecholamines and autonomic input)
- Enhancing the preload through renal preservation of intravascular volume (sodium and fluid retention)

When these compensatory mechanisms fail, the signs and symptoms of CCF appear.

Systolic dysfunction is characterised by decreased ventricular contractility. It leads to an impaired ability to increase the stroke volume to meet systemic demands. Diastolic dysfunction is characterised by decreased ventricular compliance. It leads to an increase in venous pressure to maintain adequate ventricular filling.

During acute CCF, the sympathetic nervous system and renin-angiotensin aldosterone system (RAAS) maintain blood flow and blood pressure to the vital organs. Increased neurohormonal activity leads to increased myocardial

contractility, selective peripheral vasoconstriction, salt and fluid retention, and, ultimately, blood pressure maintenance. As a chronic state of failure continues, these same mechanisms cause adverse effects.

In chronic heart failure, myocardial cells die from lack of nutrients, cytotoxic mechanisms (cause necrosis) or the acceleration of apoptosis. Necrosis stimulates fibroblast proliferation, which leads to the replacement of myocardial cells with collagen and scarring. The loss of myocardial cells results in cardiac dilation, an increased afterload and wall tension, and further systolic dysfunction.

Aetiology

The most likely causes of cardiac failure depend on the age of the child.

Table 7.5: Causes of Cardiac Failure Categorised by Age Group

Foetus	**Neonates and Infants <2 Months Old**	**Older Children**
CHF/ hydrops can be detected with a foetal echocardiogram. At this stage, CHF may be due: ● Underlying anaemia (reduced oxygen-carrying capacity) e.g. Rh sensitization, foetomaternal transfusion ● Arrhythmias (usually supraventricular tachycardia) ● Myocardial dysfunction (myocarditis, cardiomyopathy) ● Structural heart disease (rare)	● Respiratory illness (very common cause of heart failure) ● Critical congenital heart disease (CHD) – can present with CCF due to structural disease; systemic and pulmonary circulation may depend on the PDA, especially in those presenting in the first few days of life ● Primary myopathic abnormalities or inborn errors of metabolism (especially if associated with muscle weakness or lactataemia)	● Left-sided obstructive disease (valvar or subvalvar aortic stenosis or coarctation) ● Myocardial dysfunction (myocarditis, cardiomyopathy) ● Renal failure (fluid overload and anaemia) ● Arrhythmias (rare) ● Hypertension (rare) ● Illicit drug use (esp. in unexplained CHF) e.g. cocaine, other stimulants

	● Anaemia ● Infection	

Clinical Features

They depend on the severity of the heart failure and whether it is compensated or not. Clinical features include:

- Venous congestion
    - Right-sided heart failure:
        - In infants – hepatosplenomegaly (oedema and ascites are less common and jugular veins are difficult to assess)
        - In older children – abdominal pain, hepatosplenomegaly, jugular venous distention, oedema, ascites and/or pleural effusions
    - Left-sided heart failure – tachypnoea from pulmonary oedema, respiratory distress (retractions, nasal flaring, grunting), crackles and wheeze (cardiac asthma)
- Low cardiac output manifesting as:
    - Fatigue
    - Pallor
    - Sweating (during feeding in infants)
    - Cool extremities
    - Nausea and vomiting
    - Poor growth
    - Dizziness
    - Altered level of consciousness
    - Syncope
- Respiratory failure

Uncompensated congestive heart failure in infants is mainly characterised by failure to thrive. This may be followed by renal and hepatic failure in severe cases.
Uncompensated CCF in older children is characterised by fatigue. The child may complain of cool extremities, abdominal pain, nausea/vomiting, exercise intolerance, dizziness, or syncope.
Marked failure of one ventricle may lead to failure of the other.

Investigations

One should perform:

- Blood tests – arterial blood gas, FBC, cardiac biomarkers, RFTs, LFTs, RFTs (findings of renal and liver dysfunction may be present)
- Imaging – CXR, echocardiogram (should be performed to assess cardiac function and identify potential cardiovascular causes in the child with unexplained CCF e.g. structural heart lesions)
- ECG

Treatment

The goals of medical therapy for CCF are to:

- Reduce the preload
- Enhance cardiac contractility
- Reduce the afterload
- Improve oxygen delivery
- Enhance nutrition

Thus, the following medications are given

- Oral/IV diuretics (e.g. furosemide, thiazides, metolazone) – to reduce preload
- IV agents (e.g. dopamine) or mixed agents (e.g. dobutamine) – to enhance contractility
- Oral ACE-inhibitors – for afterload reduction

IV hydralazine, nitroprusside and alprostadil are only used for afterload reduction in the ICU and are not easily sourced.

Other, appropriate treatment should be instituted.

CONGENITAL HEART DISEASE

The critical congenital heart diseases include:

- The five T's
    - Truncus arteriosus
    - Transposition of the great arteries
    - Tricuspid atresia/tricuspid regurgitation (Ebstein's anomaly)
    - Tetralogy of Fallot (TOF)

    - Total anomalous pulmonary venous drainage (TAPVD)
- The four left-sided lesions:
    - Interrupted aortic arch
    - Critical aortic stenosis
    - Univentricular heart
    - Hypoplastic left heart syndrome

Acyanotic Heart Diseases

Left-to-right shunting occurs because blood flows from areas of high pressure (left heart) to areas of low pressure (right heart) through a congenital structural defect in the heart.

Acyanotic heart defects include:

- Atrial septal defect (ASD)
- Ventricular septal defect (VSD)
- Patent ductus arteriosus (PDA)
- Atrioventricular septal defect (AVSD) / endocardial cushion defect (ECD)

Atrial Septal Defect (ASD)

In this condition, blood flows from the left atrium (LA) through ASD and into the right atrium (RA) i.e. is added to normal atrial flow. On auscultation, one will hear a fixed, split second sound and grade 2/6 pulmonary ejection systolic murmur (flow murmur across the pulmonary valve). A tricuspid diastolic murmur may be heard. The child may suffer from recurrent chest infections.

Investigations

One may request:

- Chest X-ray – will show a large main pulmonary artery and plethoric lung fields)
- ECG – will show right axis deviation in primum defects (inferior), left axis deviation in secundum (upper portion) and RsR pattern in V1; these changes are the result of RA enlargement and right ventricular (RV) hypertrophy

Management

The child should undergo surgical or device closure, preferably before s/he reaches school-going age (especially if the defect is large).

Ventricular Septal Defect (VSD)

It is the most common congenital heart disease (see also related image here). Children with large defects present when they are 2-6 weeks old (slightly later but later if live at higher altitudes) as the pulmonary vascular resistance decreases from the foetal level around this time.

Clinical Features

The child will present with FTT because s/he becomes breathless during feeds as a result of the large L-to-R shunt which causes overloading and failure of the left ventricle (LV).

With an early VSD:

- Blood flows from the LV blood through the VSD and into the RV, in addition to the blood from the RA. Thus, there is LA and LV enlargement in early VSD.
- A small VSD produces a pansystolic murmur (heard at the left lower sternal border).
- A medium-to-large VSD produces ejection systolic and mid-diastolic murmurs (heard at the apex). If there is pulmonary hypertension, there will also be a loud P2.
- If the child fails to respond to anti-failure therapy, a co-existing defect must be suspected (e.g. PDA or COA) and earlier intervention will be required sooner.
- Moderately sized defects may become smaller, and small defects may spontaneously close, whereas children with large defects present with CCF and require surgical closure.

With a late VSD:

- There is RV hypertrophy and increased pulmonary vascular resistance. This results in the reversal of the shunt from right to left – Eisenmenger syndrome.
- Signs then include a very loud P2 (and irreversible pulmonary HPT), RV heave and eventually a single P2.

- Eisenmenger syndrome occurs in any longstanding L-to-R shunt. Patients become cyanosed and are generally >10 years old e.g. the child with Down syndrome and an AVSD (at high risk of early irreversible change).

Although currently less common, the child with a VSD may also have a pulmonary artery band (PAB). In these patients, there is a higher RV outflow tract gradient, ejection systolic murmur and mild cyanosis.

Patent Ductus Arteriosus (PDA)

Preterm neonates often have a PDA and develop a significant L-to-R shunt in the first week of life, especially if they have been hypoxic.

Clinical Features

The infant will present with:

- Tachypnoea
- Systolic murmur on the left sternal border just below the clavicle
- Mid-systolic murmur at the apex if the PDA is large
- Bounding/collapsing peripheral pulses

Management

PDAs usually close by the time the children reach their expected term dates. However, non-steroidal anti-inflammatory drugs (NSAIDs) or paracetamol may be used to close the PDA in preterm neonates (not effective in term infants). Surgical ligation is performed if NSAIDs are not working. Ligation is required in all term infants even if they are asymptomatic because PDAs in these children do not spontaneously close (should be performed before 6-12 months). Percutaneous closure using PDA devices is the most common intervention in children beyond the neonatal period.

Atrioventricular Septal Defect (AVSD) / Endocardial Cushion Defect (ECD)

AVSDs may be complete (there is an ASD in the ostium primum and an inlet VSD with a common atrioventricular valve) or partial (there is only an ASD in the ostium primum).

It is common for a child to present with a primum ASD and a cleft in the mitral valve, which causes mitral regurgitation or allows a L-to-R shunting from the LV to the RA. The defect may be large enough to cause cardiac failure early in life. 50% of these children have Down syndrome. Thus, AVSD must be actively excluded in children with Down syndrome (must have an echo before 6 weeks of age).

Clinical Features

The child with a complete AVSD will have a pansystolic murmur at the apex (due to mitral regurgitation) and an ejection systolic murmur (due to increased pulmonary valve flow) at the left sternal border or apex. However, the absence of a murmur does not exclude a VSD.

An ECG will show left anterior hemiblock and an RSr' pattern in V1 if there is RV hypertrophy.

Cyanotic Heart Diseases

They are the result of R-to-L shunts and may have increased or decreased pulmonary blood flow.

They may be classified as:

- Disorders with right ventricular outflow obstruction
- Mixing disorders
- Mixing disorders with RV outflow obstruction
- Disorders with parallel circulation

Tetralogy of Fallot (TOF)

It is the most common cyanotic heart disorder. In this condition, there is decreased pulmonary flow as it is characterised by:

- Infundibular and valvar pulmonary stenosis
- RV hypertrophy
- Overriding aorta (the aorta is placed over the VSD)
- VSD

One must always suspect TOF in any infant 6 months to 5 years old presenting with central cyanosis, RV hypertrophy, a single second heart sound and an ejection

systolic murmur over the pulmonary area radiating to the left clavicle; see also diagram depicting features of Tetralogy of Fallot available here.

Pathophysiology and Clinical Features

The clinical picture depends on the degree of RV outflow obstruction. If the stenosis is severe, there will be a R-to-L shunt via the VSD. This leads to early persistent central cyanosis, clubbing, polycythaemia and acidosis (sends positive feedback to the stenosis). The lungs will be oligaemic and dark lung fields will be seen on CXR (due to the decreased pulmonary blood flow).

Clinical features include:

- Cyanosis
- Ejection systolic murmur
- Single S2

The child may also experience tetralogy/tet spells. During the spells, the child suddenly becomes cyanosed after crying or feeding, or when agitated due to the rapid drop in the levels of oxygen in blood.

Treatment

Emergency treatment for tet spells includes

- Soothing the child
- Administering oxygen and putting him/her in the knee-chest
- Sedating the child with chloral hydrate (if available and necessary)
- Administering morphine 0.1- 0.2mg/kg IV, SC or IM
- Siting a drip and giving fluids (10 mL/kg; feel the liver)
- Administering sodium bicarbonate (2 mL/kg of a 4.5% solution) to counteract the metabolic acidosis
- Administering esmolol (0.5 mg/kg) or propranolol (0.1 mg/kg IV or 1-5mg/kg/day orally in divided doses)

Definitive treatment of TOF is surgical repair, which is usually performed when the child weighs around 8-10 kg. It is important to note that there may be long-term issues post-repair, therefore the patient will need lifelong follow up.

Palliative procedures which may be performed include:

- Right modified Blalock-Taussig-Thomas shunt (between the aorta and R pulmonary artery) – post-surgery, the child will have mild cyanosis, a lateral thoracotomy scar and a shunt murmur
- Central shunt (between the aorta and main pulmonary artery; usually via a median sternotomy)

Transposition of the Great Arteries (TGA)

In TGA, the great arteries arise from the incorrect ventricles – the aorta from the RV and pulmonary trunk from LV. Therefore, deoxygenated, systemic venous blood travels through the RA, RV and aorta and back to the rest of the body, while oxygenated, pulmonary venous blood travels through the LA and LV and back to the pulmonary circulation. It is, therefore, a cyanotic congenital heart disease with increased pulmonary blood flow. The child has two separate parallel circuits, which is incompatible with life.

Clinical Features

The child will present with cyanosis in the 1st week of life (usually soon after birth) and is dependent on a PDA, VSD or ASD for survival. As RV outflow goes to the aorta and the LV empties into the pulmonary artery, there is severe cyanosis, CCF and LA and LV enlargement. Murmurs are not usually heard. The child develops extreme right ventricular outflow obstruction when the duct closes. Thus, mixers with unrestricted flow present when pulmonary vascular resistance drops between 4–6 weeks.

Investigations

All children with suspected TGA must be referred for specialist assessment and management. ECGs may point to the diagnosis but echocardiography is the main diagnostic tool (is essential). Catheterisation and CTA angiogram may be performed to assess pulmonary artery structure and size. Here is a visualisation of Transposition of Great Vessels.

The CXR will be plethoric (due to the increased pulmonary blood flow) and an egg-on-side cardiac silhouette with a narrow pedicle will be seen.

Treatment

While the child is at the peripheral hospital or awaiting surgery, one should give him/her oral or (if needed) IV prostaglandin to keep the duct open. Other interventions which may need to be employed include:

- Compression of the abdominal aorta with a BP cuff
- Sedating the child with IV ketamine (0.5-1 mg/kg; may need to repeat give repeat ketamine doses or sodium bicarbonate/esmolol)
- Intubating the child
- Transfusing the child if s/he is anaemic

Once at the referral hospital, the child will be intubated (if not already) and admitted to the PICU where she will be paralysed (or given more ketamine) and started on phenylephrine (via an arterial line). If there is no improvement, the child will have to be taken for surgery.

These children are palliated with a Rashkind atrial/balloon septostomy – allows the blood to flow from the L heart to the R heart through the septum i.e. raises peripheral SaO2 by increasing pulmonary to systemic shunting. It is then followed by an arterial switch operation.

PREVENTION OF CONDITIONS AFFECTING CARDIOVASCULAR HEALTH

Some of the causes of adult heart disease that start in childhood and which can be prevented include:

- Obesity
- Atherosclerosis (build-up of plaque/fat deposits in the arteries)
- Unhealthy changes in cholesterol levels
- High blood pressure
- Diabetes