AN APPROACH TO A PAEDIATRIC MURMUR

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AN APPROACH TO A PAEDIATRIC MURMUR

INTRODUCTION

The paediatric patient presents inherent difficulties to the anaesthetist. This is due to the unique neonatal and paediatric physiology as well as pathology. Part of the difficulty in dealing with these patients is that they may have undiagnosed pathology. This may be due to consequences of the disease taking time to be clinically apparent.

Congenital cardiac disease is the most common type of congenital disease making up 30% of the total(11). The incidence of congenital cardiac disease in live-births varies from around 0.5-1 %.(2) This figure is noted to be a worldwide rate and although it may seem small due to the elevated birth rate in countries like South Africa the actual burden of disease may fall largely in developing countries.(18)

Cardiac disease may present with variety of clinical signs and symptoms. One of the most striking to an anaesthetist may be the occurrence of a cardiac murmur. Murmurs especially in the paediatric population are not always linked to pathological lesions.

A pathological murmur is defined as one associated with an anatomical cardiac abnormality and where physiological abnormalities may occur as a result of this. Innocent murmurs in contrast occur where there are no functional or anatomical abnormalities. The incidence of innocent murmurs in children range 50-72 %.(1)

Considering the incidence of congenital heart disease being quite low and the occurrence of innocent murmurs to be quite high. The majority of murmurs are thankfully nonsignificant. Even is a cardiac lesion is present most lesions tolerate minor anaesthetics. However serious complications may occur if a murmur is diagnosed incorrectly or a pathological murmur is misdiagnosed as an innocent one. Congenital heart disease has be shown to double the risk of mortality.(5)

Complications can range from:

- cardiac arrest(18)pediatric perioperative cardiac arrest registry(POCA) documented 34% of anaesthesia-related cardiac arrest occurred in children with congenital or acquired cardiac abnormalities
- haemodynamic instability
- paradoxical air embolism
- reversion to foetal circulation
- infective endocarditis(1)

For these reasons it is important to distinguish the innocent murmurs from pathological ones.
Why do innocent murmurs occur?

Murmurs occur due to turbulent flow, in pathological murmurs, abnormal anatomy is responsible for this. Turbulence is more likely to occur if Reynolds number > 3000 (1)

$$\text{Re} = \frac{pDV}{n}$$

($p$=density, $D$=diameter, $V$=flow velocity, $n$=viscosity)

Therefore any increase in the numerator or decrease in the denominator will result in a higher Reynolds number.

Children have higher cardiac output (200ml/kg/min) ($V$), smaller vessels ($D$) which tend to diverge at acute angles. There is a shorter distance from blood vessels to chest wall and thus the sound intensity of murmurs will be higher and more likely to be heard on auscultation. (1)

When do I delay surgery and when do I proceed?

Meticulous history, examination and appropriate investigations adhered to. Signs and symptoms of cardiac lesions require elicitation.

One of the first factors to take into account is the patient. Infants have a high incidence of structural heart disease in with asymptomatic lesions (50-75%) (7). Thus even the asymptomatic murmur should be referred and a delay in surgery may be warranted. (5)

History

Can often be the first stumbling block to the correct diagnosis of congenital disease as primary patient is the child, caregivers may not present at the time of anaesthetic preoperative evaluation and thus history can be severely limited.

History should start with the prenatal period. In-utero exposure to infections, alcohol, medication should be disclosed. Fetal alcohol syndrome is associated increased occurrences of ASD, VSD and Tetralogy of Fallot. Lithium can cause an increased risk of Ebsteins Anomaly, while valproate has been associated with Coarctation of the Aorta and Hypoplastic Left Heart Syndrome. Maternal rubella has been associated a higher risk of PDA. (3)

Maternal disease such as diabetes should be related as certain congenital heart diseases such as Tetralogy of Fallot, Truncus Ateriosus and Double-Outlet Right Ventricle occur more frequently. (3)

Prematurity is also noted to be associated with higher risks of congenital heart disease. (2)

Syndromes or associations with links to cardiac disease should be sought for-Downs, CHARGE, VACTERL, Turners, and Digeorge are just some examples. (5)

Family history of sudden death, SIDS (Sudden Infant Death Syndrome) or Congenital Heart Disease (CHD) all increases possibility of cardiac disease. Hypertrophic Obstructive Cardiomyopathy being linked sudden deaths, and family history of CHD increases probability of VSD and mitral valve prolapse. (4)
Cardiac signs

Specific cardiac signs or symptoms may also be elicited on history.

- Cyanosis especially central cyanosis-can be missed in patients with darker skin-should be looked for as discolouration of tongue. Pulse oximetry should be utilised to detect the presence of cyanosis.(10)
- Hypercyanotic spells associated with Tetralogy of Fallot- often occur during crying or feeding relieved by squatting(6)
- Symptoms of cardiac failure: in the neonate can be prolonged feeding, sweating, poor weight gain and failure to thrive can also manifest as swollen eyes especially after sleeping. In the older child symptoms usually include breathlessness and limited exercise tolerance and inability to keep up with activity of peers.(2)
- Syncope
- Chest pain(3)

Respiratory signs:
- tachypnoea,
- recurrent cough
- diagnosis of asthma
- recurrent chest infections
(6)

Examination

Quiet room and non-crying child are the prerequisites often necessary for proper clinical examination. (1)
Vitals should be compared to age related norms

![Paediatric Normal Vital Signs](source: APLS guidelines)
General examination

- dysmorphic features

The list of congenital syndromes/associations in which the cardiovascular system may be involved is extensive. Dysmorphic features associated with any of the below syndromes and associations should be considered.

- Turners syndrome
- 1p36 deletion syndrome
- Alcoholic embryopathy
- CHARGE syndrome
- Cri du chat syndrome
- Ellis-van Creveld syndrome
- Holt-Oram syndrome
- Jacobsen syndrome
- Marfan syndrome
- Microdeletion syndrome 22q11.2
- Noonan syndrome
- Rubeolar embryopathy
- Trisomy 13
- Trisomy 18
- Trisomy 21
- VACTERL association
- Williams (Beuren) syndrome
- Wolf-Hirschhorn syndrome(27)

- Signs of breathlessness, tachypnoea or respiratory distress. The increased respiratory rate of the child should be taken into account.
- Clubbing and cyanosis should not be overlooked; room air pulse oximetry is a necessity in a patient in which heart lesion is suspected.
- The jugular venous pulsation is difficult to see in a child<5yrs often due to their short necks and difficulty in immobilisation.(3)
- The liver should also be palpated- important to note- the neonatal liver usually palpable 1cm below costal margin.(6)
- Radiofemoral delay-important to look for in coarctation of aorta-four limb blood pressure measurement should ensue.(24)

Respiratory Examination

- Added sounds i.e.wheezes and crepitations, absent or decreased breath sounds should be auscultated.
- Defective segmentation of sternum (3)
Cardiac Examination

- An active praecordium may be a normal finding in a thin child- although it may also indicate Aortic Stenosis (AS), Ventricular Septal Defect (VSD), and Patent Ductus Ateriosus (PDA)(2)
- Apex beat is generally noted to be at the 4th-5th intercostal space and displacement of this can be due to pathology.(22)
- Auscultation

Characteristics of Innocent and Pathological Murmurs: (28)

- Sensitive: As innocent murmurs are produced by flow, changing the flow should change the murmur.
  - Reducing venous return to the heart will decrease the murmur.
  - Changing position-standing, sitting and squatting will change the intensity and the valsalva manoeuvre will reduce the noise of a murmur by reducing venous return.
  - Valsalva may be utilised in younger children who may be told to push out their stomach against examiners hands.
  - Pathological murmurs do not change on position, a notable exception being Hypertrophic Obstructive Cardiomyopathy(HOCM) which increases on standing.
- Short duration-not holosystolic, pathological murmurs may take up the entirety of systole
- Single-no associated clicks or gallops or thrills
- Small-limited to a small area and non-radiating
- Soft-low intensity (3/6 or less)
- Sweet-non-harsh sounding as harsher sounds denotes high velocity flow of blood from a higher pressure chamber into a lower pressure chamber as seen in VSD and semilunar valve stenosis
- Systolic-never solely diastolic unlike pathological murmurs.

INNOCENT MURMURS (1,3-4)

Classical-Stills Murmur
Most common type of innocent murmur, occurring most frequently in ages 2-6.It is thought to be due to vibrations of the left ventricular outflow tract, fibromuscular band in left ventricle or from the right outflow tract. The intensity is generally grade 1-3, described as musical murmur, squeaky, creaky. Early systolic, loudest at left sternal border, softer on standing and louder on squatting without an associated thrill.

Venous hum
Generally occurs in 3-8 year age group. These are low pitched continuous murmurs but accentuated in diastole, created by blood returning to the heart via vena cava. Due to the low pitch the stethoscope bell is ideal to hear the murmur over the lower neck lateral to sternocleidomastoid. The murmur normally louder in inspiration. Changing position of the head or compressing major veins will cause change in flow and the murmur may be changed or disappear.

Pulmonary flow murmur
Occurs in older children and adolescents. Tends to arise in patients with pectus excavatum or kyphoscoliosis.Thought to be most likely from a relatively smaller right ventricle outflow tract and
a larger flow volume. It has a tendency to occur in high flow conditions such as anaemia or fever. The characteristics include grade 2-3 intensity, crescendo-decrescendo quality and early mid systolic in timing. Best auscultated at the upper left sternal border between 2-3rd intercostal spaces or over apex and lower neck. Important to note this murmur has a harsh quality. It is loudest when supine and decreases when the patient is upright.

**Peripheral Pulmonary arterial stenosis of the newborn**

Occurs in infants due to the comparative discrepancy in the size of the main and branch pulmonary arteries are due the flow differences during foetal life. The main pulmonary artery transports 90% of blood to the ductus arteriosus while only 10% reach the distal arteries, thus the smaller pulmonary arteries remain not fully developed in the infant. The angle at which the pulmonary artery divides is also noted to be more acute in the infant then in later life resulting in greater chance of turbulent flow. The grade of murmur is usually 1-2, it is low-pitched and early-mid systolic. It is heard best over the back and axilla.

**Supraventricular/Brachiocephalic murmur**

Seen in late childhood to early adulthood. Caused by flow through the brachiocephalic vessels as they depart the aorta. It is a brief, low-pitched, crescendo-decrescendo murmur, systolic in timing, heard in the upper chest, supraclavicular region and can extend into the neck. There is a reduction in intensity of the murmur on hyperextension of the shoulders.

**Aortic systolic murmur**

Found in older children and adults. Arises from the left ventricular outflow tract. Short, low-medium pitch, systolic in nature, maximally heard at the aortic area.

**Mammary soufflé**

While not a murmur usually heard in childhood it may occur in adolescence and is included for completeness sake. Commonly heard in third trimester pregnancy or during lactation, caused by an increase in mammary blood flow heard along the anterior chest wall. It is noted to be a high pitched systolic murmur that may extend in diastole.

NOW..symptomatic lesions should hopefully be identified by combination of history and examination.

Asymptomatic lesions (without any abnormalities, except the murmur) may still be a sign of cardiac disease. Further investigations can further delineate the innocent from the not so innocent murmurs.

Echocardiogram continues to be the gold standard in diagnosis of cardiac lesions however at most centres echocardiogram especially in the neonate may be difficult and costly to obtain. Other investigations which are simple, cheap and easy to achieve should be considered namely Electrocardiogram(ECG) and Chest X-Ray(CXR).

While ECG generally has no adverse effects, X-Ray does expose the growing child to ionising radiation. Unfortunately the evidence pertaining to the correct diagnosis to congenital disease via these modalities is low. Several studies have shown low-pick rate on asymptomatic lesions via CXR and ECG.

However certain pathological lesions may present with an obvious abnormality in CXR or ECG, in fact all preoperative ECG is recommended in children suspected of congenital disease, where arrhythmias are generally the area of interest.
Age-related changes may make interpretation of the paediatric ECG a difficult task. The paediatric ECG reflects changes in the cardiovascular system of the growing child. As foetal circulation depends on the right sided circulation to manage flow at birth the right ventricle mass is greater and bigger than the left ventricle. The left ventricle rapidly compensates for the increased flow at birth and it increases in thickness and size. By 6 months left ventricle has overtaken the right.

Differences between children and adults.

- additional leads are often incorporated including V3R, V4R, V7. The normal ranges for heart rate, axis, intervals and R wave height and S wave depth change over the years of the child's life.

<p>| Table 1 |
|---------------------|---------------------|---------------------|---------------------|---------------------|---------------------|---------------------|</p>
<table>
<thead>
<tr>
<th>Age</th>
<th>HR (bpm)</th>
<th>QRS axis (degrees)</th>
<th>PR interval (sec)</th>
<th>QRS interval (sec)</th>
<th>R in V1 (mm)</th>
<th>S in V1 (mm)</th>
<th>R in V6 (mm)</th>
<th>S in V6 (mm)</th>
</tr>
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<tbody>
<tr>
<td>1st wk</td>
<td>90-160</td>
<td>60-180</td>
<td>0.08-0.15</td>
<td>0.03-0.08</td>
<td>5-26</td>
<td>0-23</td>
<td>0-12</td>
<td>0-10</td>
</tr>
<tr>
<td>1-3 wk</td>
<td>100-180</td>
<td>45-160</td>
<td>0.08-0.15</td>
<td>0.03-0.08</td>
<td>3-21</td>
<td>0-16</td>
<td>2-16</td>
<td>0-10</td>
</tr>
<tr>
<td>1-2 mo</td>
<td>120-180</td>
<td>30-135</td>
<td>0.08-0.15</td>
<td>0.03-0.08</td>
<td>3-18</td>
<td>0-15</td>
<td>5-21</td>
<td>0-10</td>
</tr>
<tr>
<td>3-5 mo</td>
<td>105-185</td>
<td>0-135</td>
<td>0.08-0.15</td>
<td>0.03-0.08</td>
<td>3-20</td>
<td>0-15</td>
<td>6-22</td>
<td>0-10</td>
</tr>
<tr>
<td>6-11 mo</td>
<td>110-170</td>
<td>0-135</td>
<td>0.07-0.16</td>
<td>0.03-0.08</td>
<td>2-20</td>
<td>0.5-20</td>
<td>6-23</td>
<td>0-7</td>
</tr>
<tr>
<td>1-2 yr</td>
<td>90-165</td>
<td>0-110</td>
<td>0.08-0.16</td>
<td>0.03-0.08</td>
<td>2-18</td>
<td>0.5-21</td>
<td>6-23</td>
<td>0-7</td>
</tr>
<tr>
<td>3-4 yr</td>
<td>70-140</td>
<td>0-110</td>
<td>0.09-0.17</td>
<td>0.04-0.08</td>
<td>1-18</td>
<td>0.5-21</td>
<td>4-24</td>
<td>0-5</td>
</tr>
<tr>
<td>5-7 yr</td>
<td>65-140</td>
<td>0-110</td>
<td>0.09-0.17</td>
<td>0.04-0.08</td>
<td>0.5-14</td>
<td>0.5-24</td>
<td>4-26</td>
<td>0-4</td>
</tr>
<tr>
<td>8-11 yr</td>
<td>60-130</td>
<td>-15-110</td>
<td>0.09-0.17</td>
<td>0.04-0.09</td>
<td>0-14</td>
<td>0.5-25</td>
<td>4-25</td>
<td>0-4</td>
</tr>
<tr>
<td>12-15 yr</td>
<td>65-130</td>
<td>-15-110</td>
<td>0.09-0.18</td>
<td>0.04-0.09</td>
<td>0-14</td>
<td>0.5-25</td>
<td>4-25</td>
<td>0-4</td>
</tr>
<tr>
<td>&gt;16 yr</td>
<td>50-120</td>
<td>-15-110</td>
<td>0.12-0.20</td>
<td>0.05-0.10</td>
<td>0-14</td>
<td>0.5-23</td>
<td>4-21</td>
<td>0-4</td>
</tr>
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(23)

Axis
Right ventricular dominance and right axis deviation is seen in the neonate. There is also an increased R/S wave ratio in leads V1, V2 and a decreased R/S Ratio in V5, V6

PR interval
PR intervals increase with increasing cardiac muscle mass. It is essentially very short in the newborn 0.08-0.15 and increases to normal lengths by 16 years. Thus the diagnosis of atrioventricular block may be missed.

QRS complex
Again the duration of this complex is reduced in children. QRS complex duration is important for the diagnosis of bundle branch blocks or conduction abnormalities.

T waves
Inverted and flattened T waves are normal in the newborn. In V1-V3 T waves maybe inverted until 8 years of age. Upright T waves after 72hrs maybe a sign of right ventricular hypertrophy.
Atrial and ventricular enlargement

- As structural cardiac disease often presents with signs of atrial or ventricular enlargement, paediatric ECG criteria are important to recognise.
- Right atrial enlargement—P waves are taller than 2mm in infants and 3mm in adolescents (p pulmonale)
- Left atrial enlargement—broad p waves greater than 2mm in infants and 3mm in adolescents.
- Right ventricular hypertrophy—in leads V1, V2 with rSR, QR (no S), only R (no Q, S) wave. Large S waves in V6, upright T waves in V1-V3 or persistence of right ventricular dominance after 6 months.
- Left Ventricular hypertrophy—tall R waves in V5, V6 (>4mV), large S wave in V1, adult pattern of R wave progression in the neonate.

Thus depending on the lesion and the cardiac abnormality it produces signs may be seen on ECG.

THE PATHOLOGICAL PAEDIATRIC X-RAY

Chest radiograph may provide clues as to the diagnosis of pathological lesions as many present with characteristic pictures. Again paediatric x-rays differ from adult x-rays in terms of normal.

Differences between paediatric and adult x-rays

- anterioposterior view is usually taken as the baby/small child lies on the x-ray plate.
- normal cardiothoracic ratio 60%
- normal thymus in patients under 18 months causes a widened superior mediastinum
An approach to chest radiograph interpretation in the paediatric patient - with reference to congenital cardiac disease

Certain anatomical aspects need to be looked at:

1. **Pulmonary vasculature: normal, congested, oligaemic**
   
   **Congested-active vs. passive**
   
   **Active:** increased blood flow, seen in left to right shunts, enlarged vessels are seen more peripherally and maybe tortuous the margins remain distinct
   
   **Passive:** increased pulmonary venous pressure suggests left-sided cardiac dysfunction; margins are not distinct as interstitial oedema occurs.

   **Oligaemic vasculature:** Decreased pulmonary vasculature arises as decreased blood flows through the lungs due to right to left shunt and right ventricle outflow obstruction. If the proximal pulmonary vessels seem enlarged while peripherally they appear small or absent pulmonary hypertension should be suspected.

2. **The Aorta**
   
   An increase in size of the aortic knuckle can be secondary to increased blood flow which occurs in
   
   - PDA, truncus ateriosus, valvular insufficiency.

   A decrease in aortic knuckle size is due to decreased blood flow in ASD, VSD or hypoplasia(hypoplastic left heart syndrome).

   Appearance of a right-sided aortic arch can be associated with heart disease in 10% of patients

   Abnormalities in shape generally associated with coarctation.

3. **Cardiac size and characteristic shapes**
   
   Enlargement of the cardiac shadow represents chamber expansion.

4. **Bony structures**
   
   Scoliosis present in 6% patients with CHD
   
   Ribs may display notching. Patients with downs syndrome may have 11 ribs or hypersegelemented sternums.

**SPECIFIC SIGNS SEEN IN PATHOLOGICAL MURMURS (3,6,25)**

**Ventricular Septal Defect(VSD)**

Most common congenital heart lesion. Maybe missed in the postnatal period as pulmonary vascular resistance is high. Thus the gradient allowing the VSD to occur may not be as apparent. Typically presents as holosystolic murmur, harsh quality characteristics depend on the size of the lesion. Small defects are usually asymptomatic with a loud murmur. Large defects may have a thrill at LLSB, split or loud S2. A mid-diastolic rumble may also be heard. Large defects may also present with cardiac failure and respiratory symptoms.ECG typically left atrial and ventricular enlargement, if pulmonary hypertension occurs-QRS axis shifts to the right and right atrial and ventricular enlargement ensue. On Chest X-ray-marked cardiomegaly and increased pulmonary vascularity occur.
**VSD**
Marked cardiomegaly and increased vascularity noted in the lung fields.

**Atrial Septal Defect (ASD)**
Most common congenital heart defect detected in adults (4). Small defects are often asymptomatic. Its characteristic murmur-grade 2-3 systolic ejection murmur, best heard on the upper left sternal border—maybe mistaken for the innocent pulmonary flow murmur. Important differences include widely split second heart sound due to right ventricular volume overload.
**ASD**
Associated cardiomegaly and fullness of the right heart border in keeping with right atrial enlargement. Prominent pulmonary arteries extending to the periphery of the lung.

**Patent Ductus Ateriosus (PDA)**
Preterm infants are at higher risk of failure of closure of PDA. A continuous systolic and diastolic murmur in ULSB or left infraclavicular area is auscultated. A thrill or hyperdynamic left ventricular impulse may be present. ECG and CXR-Left ventricular hypertrophy. If pulmonary hypertension is present right ventricular hypertrophy may be seen.
**Patent Ductus Arteriosus (PDA)**
The PDA may be seen extending from the pulmonary trunk to the aorta. Occasionally this vessel may be calcified. The main pulmonary artery and its branches are dilated.

**Aortic Stenosis**
These occur secondary to bicuspid aortic valves. Often present after 15 years. A systolic murmur present over the aortic area that radiates to the neck is perceived. Congenital supravalvular aortic stenosis (SVAS) - characteristic appearance prominent facial bones, pursed upper lip and rounded forehead, in these patients myocardial ischaemia is implicated in cases of sudden death associated with anaesthesia or sedation. ECG changes commonly are due to left ventricular hypertrophy and ST depression during exercise. Radiographic changes will confirm left ventricular hypertrophy and possible poststenotic dilatation.

**Pulmonic Stenosis**
Maybe supravalvular or subvalvular. Supravalvular exists with other congenital abnormalities and is feature of Williams’s syndrome. Loud systolic ejection murmur, graded 2-5, heard in the pulmonary area (2nd left intercostal space on the left) radiation may occur back, axillae, infraclavicular. Systolic ejection click can be auscultated. The duration and intensity of the murmur is proportional to the underlying disease.

**Coarctation of the aorta**
Preductal coarctation proximal to left subclavian is most likely to present in infants. It is also seen in Turner’s syndrome. It is usually an asymptomatic lesion and diagnosis is made from differing upper limb versus lower limb pressures with upper limbs being hypertensive and lower limbs being hypotensive with weak or absent femoral pulsations. This murmur results in a harsh systolic ejection murmur heard over the back or the left sternal border. Importantly in preductal coarctation there is no difference in systemic blood pressure in upper/lower limbs - due to collateral circulation involving subclavian, scapular, internal thoracic and intercostal arteries. The result is murmur in the interscapular area. ECG will reflect left ventricular hypertrophy in the older child while the infant will still have the right axis and right ventricular hypertrophy seen in infancy. On radiograph due to collateral blood flow notching in the posterior 3rd of ribs 3-8 is seen. X-ray features of the coarctation itself may be seen as a reversed E or 3 sign.
Coarctation of the aorta
The X-ray demonstrates the classic “reverse E or 3” sign caused by narrowing and post-stenotic dilatation. Inferior notching is noted the ribs are noted on the left as the intercostal arteries dilate as collateral pathways.

Hypertrophic Obstructive Cardiomyopathy (HOCM)
The involvement of the right ventricle may be reflected a systolic ejection murmur at the lower left sternal border due to obstruction to right-sided outflow.

If the left side of the heart is implicated a displaced apex beat will be noted , the murmur present is usually grade 1-2 increasing in intensity on standing or evoking the valsalva manoeuvre. If subaortic obstruction is present the murmur at apex is grade 3-4 and it radiates to the left sternal border, a double or triple apex beat may also be palpated. ECG findings comprise left or right sided enlargement depending on the hypertrophied chambers,(generally more likely to be left-sided) with ST segment changes, Q waves, inverted T waves and small R waves in the limb leads. Arrhythmias are also common and may different rhythm abnormalities may be present including atrial or ventricular extrasystolic complexes, supraventricular tachycardia and atrial fibrillation. (29)

Tetralogy of Fallot
The infant with tetralogy will often appear pink at birth and the onset of cyanosis only occurs between 2-6 months. Auscultatory findings include a systolic ejection murmur on the left sternal border and this is due to turbulence caused by pulmonary stenosis or narrowed right ventricular outflow tract. In this lesion the murmur does not parallel severity as in pulmonary stenosis. The holosystolic murmur of VSD may be noted. CXR features will often show diminished pulmonary vascular markings. The shape of the heart has been described as boot-shaped, the right ventricular apex is turned upwards and the main pulmonary artery is concave. ECG tends to right axis deviation and right ventricular hypertrophy.
**Tetralogy of Fallot**
The mediastinum is narrowed due to hypoplasia of the pulmonary valve. The left ventricular apex is displaced laterally and upward due to right ventricular hypertrophy – “boot shaped heart”. The lung fields demonstrate decreased vascularity due to decreased blood flow across stenosed pulmonary valve and left to right shunting across the VSD.

**Ebsteins anomaly**
Depending on the age group of presentation features may include: in the neonate cyanosis and congestive cardiac failure post ductal closure and in adolescents with supraventricular arrhythmias. A systolic murmur on the lower left sternal border is heard. ECG commonly shows tall p waves-first degree heart blocks, paroxysmal SVT and VT. Woolf-Parkinson White may also be present. On radiograph significant enlargement of the right atrium and massive cardiomegaly is appreciated. The heart is typically globe or box shaped and may fill the entire chest cavity.
**Ebstein’s anomaly**
Globular shaped cardiomegaly with oligaemic lung fields. The heart has a box shape.

**Tricuspid Atresia**
These patients will either have a murmur of VSD or PDA depending on what lesion allows peripheral oxygenation. Radiologically no consistent pattern is seen with features depending on total pulmonary blood flow. ECG demonstrates right atrial enlargement, left axis deviation and left ventricular hypertrophy.

**Chest radiograph in a patient with tricuspid atresia.**
There is pulmonary oligaemia, a small pulmonary artery, and a prominent rounded left ventricular curve to the left heart border

**Transposition of the great arteries**
Of note a murmur may be absent is this abnormality, a soft grade1-2 ejection systolic murmur maybe heard. VSD murmur may also be the only murmur heard. ECG-right axis deviation, right ventricular hypertrophy. The eggshaped heart with a narrow shaft may confirm the diagnosis.
Transposition of the great vessels
Narrowed mediastinum, enlargement of the cardiac silhouette with abnormal convexity of the right atrial border.

Total anomalous pulmonary venous return
Murmurs of ASD, PDA may be heard. Right sided enlargement is seen on ECG, on x-ray cardiomegaly and pulmonary oedema maybe seen-illustrated by snowman in snowstorm picture.
TAPVR – the classic snowman appearance.
The pulmonary veins converge behind the left atrium and form a common pulmonary vein that drains into the left brachiocephalic.

The angiographic view demonstrates the common pulmonary vein communicating with the left brachiocephalic vein via a vertical vein.
CONCLUSION

The child with a murmur who presents for non-cardiac surgery should be evaluated by preoperative assessment.

The child with should first be differentiated into 2 different age-related categories >1 year and <1 year, due to the high incidence of asymptomatic murmurs with pathological lesions infants should be referred for echocardiogram prior to theatre. Again two groups may be separated following history and examination, those with symptomatic disease and those without.

Those with symptomatic disease should be assessed prior to commencement of anaesthesia. Part of this assessment may include ECG, CXR or echocardiogram to further delineate the lesions into those safe to undergo non-cardiac procedures and those that require further workup or cardiac surgery first.

Asymptomatic patients over 1 year should now be evaluated for the possibility of serious cardiac disease; here ECG has been advocated as ECG changes occur in most problematic cardiac conditions such as HOCM, aortic and pulmonary stenosis, coarctation and tetralogy of fallot. (2)
Murmur discovered preoperatively

Soft, early systolic murmur, no thrill?

Venous hum?

NO (i.e. other murmurs)

Postpone surgery and refer for investigation

YES

<1 year old

NO

Asymptomatic

YES

ECG normal

YES

Dirty surgery?

NO

Proceed with surgery
No antibiotics
Refer for postoperative investigation

YES

Proceed with surgery
Appropriate antibiotic cover
Refer for postoperative investigation

Fig. 1. An assessment algorithm.

(2)
REFERENCES