Cardiac Disease and Murmurs in the Newborn

St. Peter’s Hospital, Neonatal Unit

Diagnosis & Investigation of Congenital Heart Disease in the Neonate

Congenital heart disease (CHD) is one of the commonest congenital malformations, affecting 7 to 8 per 1000 live births\(^1,2\). Although many of the structurally significant heart lesions will present with symptoms early on, a significant proportion may demonstrate no symptoms until they later collapse\(^3\). At the same time, however, symptoms such as murmurs are very common in the newborn period and often do not require any therapeutic intervention.

Common Presentations & Their Management

The common ways in which CHD presents are with cyanosis; respiratory distress; a murmur; cardiovascular collapse; feeding difficulties; or a combination of the above. Assessment of potential CHD must include a thorough pre- and postnatal history, as well as review of any antenatal scans performed.

The routine newborn check must include:
- assessment of feeding and any respiratory distress
- auscultation of the praecordium
- palpation of femoral pulses

Cyanosis (\(+/-\) respiratory distress)

Causes include:
- CHD
- Respiratory disease
- Sepsis
- Persistent pulmonary hypertension of the newborn
- Methaemoglobinaemia (rare)

Clinically, cyanosis is best seen in the tongue and the perioral mucosa, but the clinical pick-up rate of cyanosis is low. Therefore, if there is any doubt at all, lower-limb oxygen saturations (normal >95\%) must be obtained. All babies with cyanosis in the postnatal ward MUST be admitted to the neonatal unit for further investigation, directed at the above causes. A hyperoxia (nitrogen washout) test (see Appendix 1), together with chest X ray & ECG, is still a robust means of distinguishing between cardiac and non-cardiac causes of cyanosis, where echocardiography is not immediately available.

The classical presentation of a cyanotic lesion in the neonatal period is due to the closure of the ductus in one of the duct-dependent circulations, the treatment for which would be prostaglandin E2—see Neonatal Formulary. This must be discussed with the consultant.

Once the cause is confirmed as a duct-dependent cardiac lesion, oxygen therapy must be minimised (to keep saturations at 70% -75%) as hyperoxia can stimulate closure of the PDA and subsequent clinical deterioration. Paediatric cardiology opinion must be sought urgently to advise on further management.

Murmurs:

Murmurs are relatively common in the newborn period, affecting 0.5 – 1% of all newborns\(^5\). Of these, a significant proportion (46% - 62%) are likely to have a clinically insignificant murmur, commonly a PDA or peripheral pulmonary artery stenosis\(^5,6\). In babies with a murmur a full cardiovascular examination, and lower-limb oxygen saturations is a minimum requirement\(^7\).
Features of a clinically insignificant murmur are:

1) Soft (no heaves/thrills) & Systolic (diastolic murmurs are NEVER innocent)
2) Localised
3) Normal pulses & limb oxygen saturations (>95%)^4

A) If the murmur is clinically insignificant, saturations >95% & baby is asymptomatic:
   - re-examine in 24 hours’ time, ideally by the same person.
   - If murmur still present, examination by a senior colleague to confirm that the murmur is clinically insignificant.
   - If so, arrange an appointment in 4 to 8 weeks’ time, provide a parent information sheet on heart murmurs & advise to return to hospital if there is:
     - Central cyanosis
     - Respiratory distress and/or excessive sweating
     - Poor feeding (>2 consecutive feeds of <50% usual volume) or poor weight gain

B) If there is any doubt about the clinical significance of the murmur:
   Any potentially-significant murmurs must be reviewed by a senior colleague. In addition, a chest X ray and ECG must be performed as well as 4-limb blood pressures. Discuss early echocardiography with the consultant.

C) If limb saturations <95% and/or baby is symptomatic:
   See “Cyanosis (+/- Respiratory distress)”

Cardiovascular collapse

Circulatory collapse, with diminished peripheral pulses and poor systemic perfusion, is a classic presentation of a systemic outflow tract obstruction, such as coarctation of the aorta, critical aortic stenosis, interrupted aortic arch and hypoplastic left heart syndrome^2,3. The decompensation occurs as a result of closure of the ductus arteriosus. Cyanosis is not usually present. Care must be taken to distinguish this picture from that of sepsis, which may present in a very similar fashion, and if in doubt treatment for sepsis should be commenced alongside investigation and management of suspected CHD.

The emergency management of any baby presenting in the above manner is:

- Assess & resuscitate according to A(Airway), B(Breathing) & C(Circulation)
- Full examination, noting presence of murmurs & quality of femoral pulses, 4-limb blood pressures, respiratory distress, hepatomegaly and central & peripheral perfusion
- Support ventilation if needed
- Secure vascular access x2
- Admit to the neonatal unit (or other appropriate unit) and inform consultant
- Bloods for FBC/U&E/CRP/blood culture/blood gas including blood sugar
- Commence first-line antibiotics
- Correct metabolic acidosis as indicated
- In addition, inotropic support may be required (particularly in hypoplastic left heart syndrome where, once the ductus has re-opened, the ratio of blood flow in the pulmonary and systemic circulations must be balanced)
- CXR; urgent echocardiography if available
- Consider invasive blood pressure monitoring
- If duct-dependent CHD is suspected, the mainstay of treatment is to re-open the ductus arteriosus with prostaglandin E2—see neonatal formulary for details of administration.
- Early discussion with a cardiology unit is mandatory, both for management advice and potential transfer
Appendix 1:

**Hyperoxia (Nitrogen Washout) Test**

This test differentiates between cardiac and respiratory causes of cyanosis.

1. Place the baby in 100% oxygen for 10 minutes, in order to completely fill the alveoli with oxygen.

2. If the paO2 rises above 100mmHg (14kPa) (or oxygen saturations to 100%) then the cause of the cyanosis is more likely to be respiratory or central (e.g. sepsis).

3. If there is no/little change in the paO2 (<4kPa), or if it rises but stays below 100mmHg/14kPa, the cause is more likely to be due to an intracardiac right-to-left shunt.

Exceptions are TAPVD (which may respond to high-flow oxygen due to the large pulmonary blood flow), or conversely in very severe respiratory disease, or with large intrapulmonary shunting (which will NOT respond to high-flow oxygenation).

**References**


Guideline written by Dr R Cheung, Neonatal SpR 2009
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