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Evaluation of suspected congenital heart disease in the neonatal period

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Summary Congenital heart disease (CHD) is the most common group of significant congenital abnormalities. It may present in the neonatal period with an asymptomatic murmur detected on the routine neonatal examination or when an infant becomes symptomatic. In assessing an infant with possible CHD, key features in history and examination need to be considered. The investigations appropriate to consider outside the tertiary paediatric cardiology setting will be discussed. We highlight the importance of a thorough assessment of the neonate presenting with an asymptomatic heart murmur to ensure that, where possible, infants with duct-dependent CHD are not discharged home inappropriately. The symptomatic presentation of CHD in the neonatal period is described, considering groups related by physiology rather than concentrating on the details of individual lesions. Important points in the initial stabilisation of a symptomatic infant are outlined.

Practice points

- Cyanosis is easily missed, always check oxygen saturation.
- Loud murmurs (especially associated with thrills) or abnormal pulses must always be taken seriously.
- Dysmorphic features or the presence of associated anomalies increase the risk of a murmur being pathological.
- Always consider congenital heart disease in the sick infant.

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Introduction

Congenital heart disease (CHD) can be defined as a structural abnormality of the heart or intrathoracic great vessels which is actually or potentially of functional significance.[1] It represents a spectrum of conditions, from those that may be fatal in the neonatal period, to those with which a normal lifespan would be expected.

Current guidelines recommend initial screening for CHD in the neonatal period with a further examination at 6–8 weeks.[2] Around 45% of CHD will be detected on routine neonatal examination when a large number of innocent murmurs will also be heard. Even severe lesions may not be detected at this point due to the presence of a widely patent ductus arteriosus.[3] Many lesions, particularly those

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with significant left ventricular outflow tract obstruction will become symptomatic before the second examination and carry a high mortality if they continue to go unrecognised.[4,5] The appropriate assessment of infants who have been found to have an abnormal neonatal cardiac examination and an awareness regarding the range of presentations of CHD in early infancy are therefore vital.

Risk factors for CHD

CHD is the most common group of significant congenital malformations accounting for 40% of the total group.[6] Most studies estimate the incidence of CHD to be between 5 and 8 per 1000 live births, although this figure varies according to case definition and method of case ascertainment.[7]

Family history

Having a sibling with CHD confers a 2% risk on a subsequent child. A mother with CHD has a 6% risk of having an affected offspring and a father a 2% risk overall.[8] There is some variation in these figures depending on the specific lesion present.

Syndromes and associations

The aetiology of CHD is complex, and in most cases multifactorial. There are a number of recognised associations that include:

- Chromosomal abnormalities (Down, Edward, Patau, Turner, cri-du-chat)
- Contiguous gene syndromes (William's, DiGeorge's)
- Single gene defects (Noonan's, Marfan's, isomerisms)
- Teratogens (anticonvulsants, alcohol, lithium)
- Congenital infection (rubella)

Even in conditions, such as Down syndrome, in which the association with CHD is clear, the mechanisms underlying the abnormality are only now beginning to be elucidated.

Antenatal detection

Routine antenatal screening for CHD is limited to a four-chamber view of the heart and is performed at around 20 weeks gestational age as part of anomaly scanning. This will pick up around 25% of significant CHD,[9] although with specific training and assessment of the ventricular outflow tracts, this may be significantly improved.[10] Fetal echocardiography, in the experienced hands, is both sensitive and specific however, its role is limited to those 5% of pregnancies in which an abnormality has been detected on routine antenatal scanning or in which an identified risk factor is present.[10,11] In the majority of cases with no previously identifiable risk factor CHD will continue to go undetected prior to delivery.

Presentation of CHD in the neonatal period

In the neonatal period a diagnosis of CHD may be considered for two reasons: (1) a heart murmur or other cardiovascular abnormality identified in an asymptomatic infant or (2) the development of symptoms and signs that could be attributable to CHD. The initial assessment of these infants will be discussed.

Assessment of a child with suspected CHD

History

The following information needs to be obtained at the initial review:

- *Risk factors*: Family history, maternal medication or congenital infection.
- *Antenatal scans*: Routine anomaly and fetal echocardiography.
- *Perinatal*: Considering risk factors for infection and persistent pulmonary hypertension that may present in a way indistinguishable from CHD.
- *Postnatal*: Breathing and feeding difficulties. Excess weight gain or failure to lose weight after birth.

Examination

A full examination should include:

- *Evaluation of airway, breathing and circulation*: Assessment and management of life-threatening problems.
- Presence of dysmorphic features and other congenital abnormalities.
- *Colour*: Cyanosed or pale. Check oxygen saturation with a pulse oximeter.

- *Signs of respiratory distress*: respiratory rate, recession and grunting.
- *Heart rate*.
- *Palpation of pulses*: Discrepancy between right and left brachial pulses with weak femorals suggest the possibility of an aortic arch abnormality.
- *Presence of thrills*: In the upper sternal edge and suprasternal notch will suggest severe or critical pulmonary or aortic stenosis that may be duct-dependent.
- *Auscultation for murmur*: Timing, site and radiation.
- *Palpation for hepatomegaly*: A large liver indicates heart failure or elevated right heart pressures.
- *Auscultation for bruit*: For cerebral or hepatic arterio-venous malformations.

Investigations

Oxygen saturations

Central cyanosis is the bluish discolouration of the lips and mucous membranes due to the presence of greater than 5 g/dl of deoxygenated haemoglobin. It may not be visible until the oxygen saturation of arterial blood drops below 85%. It can be particularly difficult to determine in dark-skinned infants. Clinical assessment of cyanosis is inadequate to exclude low arterial oxygen level and pulse oximetry should always be performed. Lower oxygen saturations in the lower limbs compared with the right arm, indicates right-to-left shunting across the ductus arteriosus which may be associated with persistent pulmonary hypertension or coarctation of the aorta. Arterial oxygen measurement needs to be carried out to confirm this.

Blood pressure (BP) measurement

BP measurement is routinely carried out as part of the initial assessment of all neonates suspected of having CHD. Non-invasive BP measurements rely on the choice of an appropriate cuff size for the infant. The width of the air bladder should be 40–50% of the circumference of the arm. Oscillometric devices, are normally used for measuring BP and these have been validated in neonates.[12,13] In the normal infant the BP in the thigh should be 5–10 mmHg higher than that in the arm, a lower value in the legs raises the possibility of a coarctation or interruption of the aorta. A normal value does not exclude the conditions particularly if the femoral pulses are of low volume or impalpable.

Electrocardiogram (ECG)

ECG findings change in the first few days of life, and unless very familiar with analysing ECGs in this age group, we recommend referral to available standard texts for information (see further reading). In an asymptomatic infant an abnormal ECG may support a diagnosis of CHD, however, a normal ECG does not exclude a lesion.

Chest X-ray (CXR)

Table 1 indicates the range of information that may be obtained from a CXR. Although the CXR continues to be part of the initial investigations for neonates with an asymptomatic murmur, they need to be interpreted with caution. Even when reported by a group of paediatric radiologists, the test has a positive predictive value of only 0.4 and a negative predictive value of just 0.8 for CHD. This will result in unnecessary investigations in some and a missed opportunity for early diagnosis in others if inappropriate weight is placed on the findings of the CXR.[14]

Echocardiography

It is not possible to arrange an urgent local echocardiography examination by a paediatric cardiologist in many centres, and is logistically impossible for all infants noted to have an asymptomatic murmur.[15] The diagnostic accuracy of echocardiography in the hands of non-paediatric cardiologists is user-dependent and highly variable. With appropriately trained personnel good diagnostic accuracy, resulting in treatment alteration, has been reported.[16] However, another study reports far less favourable accuracy with significant errors occurring, particularly in those below 1 month of age.[17] Although an increasing number of neonatologists are gaining these skills, placing excess weight on unreliable findings, both positive and negative, may be detrimental. Telemedicine, in which locally obtained echocardiographic images are transmitted for review by a tertiary paediatric cardiologist, has been shown to be effective and may avoid some of these problems.[18] Echocardiography should not delay the initial stabilisation of an infant, as in most cases this will not be dependent on the echocardiographic description of the individual lesion.

The heart murmur in the asymptomatic infant

The identification of a heart murmur on routine neonatal examination is common. A murmur may

Table 1 Information that may be obtained from a neonatal chest X-ray.

Heart position	Right, left or central	Dextrocardia may be a isolated or may be found in conjunction with a range of cardiac defects including transposition of the great arteries (TGA)
Bronchial or abdominal situs	Usual, inverted, ambiguous	Suggestive of isomerisms
Aortic arch	Left or right	
Thymic shadow	Presence or absence	Absence may indicate DiGeorge's syndrome. A large thymic shadow is easily mistaken for cardiac enlargement
Heart size	A cardiothoracic ratio of up to 60% may be normal in the neonatal period	An enlarged heart may indicate heart failure or cardiomyopathy
Heart contour	Boot shaped Egg-on-the-side shaped Snowman shaped	Tetralogy of Fallot TGA Supracardiac TAPVD
Pulmonary vascular markings	Increased or decreased	Lesions associated with high and low pulmonary blood flow
Other pulmonary pathology	Collapse, consolidation, diaphragmatic hernia	
Skeletal abnormalities	Spinal abnormalities	Associated with syndromes, e.g., Alagilles or VACTERL association

TAPVD, total anomalous pulmonary venous drainage.

be innocent, whereby it is not associated with either anatomical or physiological abnormalities, or pathological whereby it is associated with underlying CHD.[19] It is not always possible to reliably distinguish the groups on auscultation alone. Newborns noted to have a murmur will fall into these two groups in approximately equal proportions.[20] Table 2 outlines the causes of a neonatal murmur, both innocent and pathological.

The primary consideration when assessing an asymptomatic neonate with a murmur is whether it could be associated with duct-dependent CHD, which would require assessment and management before an infant could be discharged from hospital. Table 3 provides a checklist for use in the postnatal setting that is designed to minimise the likelihood of significant lesions being missed. If there is any doubt a senior review should be sought and the infant should continue a period of observation in hospital or be referred to a paediatric cardiologist for a definitive assessment.

All infants with a heart murmur, in whom a definitive diagnosis has not been made before hospital discharge require follow-up with a paediatrician or a paediatric cardiologist, depending on local practice, within 4–6 weeks. It is important to inform parents of the symptoms of CHD before hospital discharge.

The symptomatic infant

CHD may become symptomatic in the neonatal period in three ways:

- Cyanosis
- Heart failure
- Collapse

Some anomalies may present with a combination of these features according to the timing of presentation and severity. There will usually be a number of, generally more common, differential diagnoses that could explain each of these presentations, including respiratory and infective conditions. For this reason consideration of CHD and the ways it may present in early life is vital to

Table 2 Causes of heart murmurs in the neonatal period.

Pathological		Innocent or physiological	
Obstruction			
Pulmonary Aortic Valvar Subvalvar Supravalvar		Physiological pulmonary branch narrowing	Maximal in pulmonary area, transmitted throughout the chest Resolves by 6 months
Coarctation	Particularly as ductus arteriosus closes		
Regurgitation			
Tricuspid Mitral	Associated with trioventricular septal defect and other complex lesions	Tricuspid regurgitation	May be confused with a ventricular septal defect (VSD) clinically Resolves as pulmonary vascular resistance falls-
Shunting			
Persistent ductus arteriosus (PDA) and VSD	May be minimal murmur if large defect or high pulmonary vascular resistance	PDA	90% resolve spontaneously
Atrial septal defect (ASD)	Rarely in neonatal period		
Atrioventricular septal defect	Murmur primarily due to atrioventricular valve regurgitation		

Table 3 Checklist for use in assessing the asymptomatic murmur in a neonate.

History	Are there any risk factors Is the infant symptomatic
Examination	No dysmorphic features No respiratory distress No soft systolic murmur Tachypnoea or tachycardia Good volume femoral pulses
Oxygen saturations	Normal ($>95\%$) in arms and legs
Blood pressure	Normal and higher in legs than arms
Electrocardiogram	Normal
Chest X-ray	Normal
Parents informed of features of symptomatic CHD	

ensure timely diagnosis and appropriate initial management.

Cyanosis

Table 4 outlines the differential diagnoses of central cyanosis in the newborn period and the features that may be useful in distinguishing the groups.

The hyperoxia test

The hyperoxia test may help to differentiate respiratory causes from cyanotic CHD. The infant is placed in as near to 100% oxygen as possible for 10 min and a right radial (pre-ductal) arterial gas is

Table 4 Causes of central cyanosis in the neonatal period.

Causes of neonatal central cyanosis	History and examination	Investigations			
		Chest X-ray	Electrocardiogram (ECG)	Oximetry and arterial blood gas	Hyperoxia test
Respiratory disease	Respiratory distress present	Usually abnormal or diagnostic	Usually normal	Low PO_2, high PCO_2	Likely to pass
CHD	Respiratory distress less common. May have other abnormal cardiac findings	Heart size and vascular marking may be suggestive	May be helpful if abnormal. Normal ECG does not exclude CHD	Low PO_2, normal or low PCO_2	Likely to fail
Persistant pulmonary hypertension	History suggestive of cause, e.g., meconium aspiration, asphyxia or sepsis	May suggest underlying cause, e.g., meconium aspiration or congenital pneumonia	Usually normal	Right arm saturations may be higher than legs Low PO_2, normal or high PCO_2	May pass or fail
Methaemo-globinaemia	Otherwise well, may be a family history	Normal	Normal	Normal	Pass

sampled at the end of the period. A PO_2 of less than 20 kPa, supports a diagnosis of cyanotic CHD over a respiratory disorder. However, it is important to be aware that common mixing conditions with a high pulmonary blood may raise the PO_2 above this level, and that respiratory conditions with a severe ventilation perfusion mismatch may not. As such the test needs to be taken in the context of the rest of the assessment. If a competent paediatric echocardiographer is available the hyperoxia test will be less vital, the distinction being made by echocardiography.

Table 5 outlines the three groups of CHD that will present with neonatal cyanosis and includes the most common lesions presenting in each group.

Right-to-left shunting

Right-to-left shunting, associated with inadequate blood flow through the lungs, results from obstructive lesions on the right side of the heart. Deoxygenated blood shunts from the right side into the left via a patent foramen ovale, atrial septal defect (ASD) or ventricular septal defect (VSD). Deoxygenated blood therefore enters directly into the systemic circulation without passing through the lungs. The level of cyanosis will be determined by the degree of right-sided obstruction and the ease with which blood can pass between the right and the left sides of the heart. With severe right-sided obstruction or atresia the only blood going to the pulmonary circulation will be through the ductus arteriosus unless collateral vessels are present. A duct-dependent pulmonary circulation is therefore present, and infants with these lesions may also present with profound cyanosis and collapse as the duct closes. These lesions will be associated with oligaemic lung fields on CXR.

Common mixing conditions

In common mixing conditions oxygenated blood, having been through the pulmonary circulation, mixes again with deoxygenated blood. This may occur at a number of levels. In total anomalous pulmonary venous drainage (TAPVD) oxygenated blood returns to the vena cava or other large systemic veins. Once mixed the oxygenated and deoxygenated blood passes through an ASD or VSD into the systemic arterial circulation. In truncus

Table 5 CHD presenting with neonatal cyanosis.

Right-to-left shunts	Common mixing	Transposition of the great arteries	
Cyanosis ± collapse • Tricuspid atresia • Pulmonary atresia • Critical pulmonary stenosis • Tetralogy of Fallot	Cyanosis ± heart failure • Total anomalous pulmonary venous drainage • Truncus arteriosus • Double inlet ventricle • Double outlet ventricle	Cyanosis ± heart failure With ventricular septal defect/persistent ductus arteriosus	Cyanosis ± collapse Without ventricular septal defect/persistent ductus arteriosus

arteriosus blood mixes at the level of a common pulmonary arterial and aortic trunk. The mixed blood is thus transmitted to the pulmonary and systemic circulation. Infants with common mixing conditions present with heart failure and cyanosis. Heart failure will predominate if blood flow is unrestricted through the lungs resulting in excessive blood circulating around the pulmonary circulation. This would be more common with truncus arteriosus, when cyanosis may be less obvious initially. In obstructed TAPVD free flow through the lungs is compromised due to obstruction at the level of the pulmonary veins, and infants will present with profound cyanosis and breathlessness. CXR findings will similarly depend on this balance.

Transposition of the great arteries (TGA)
TGA forms the third group of lesions that present with neonatal cyanosis. The systemic and pulmonary circulations are in parallel resulting in oxygenated blood being pumped round the lungs and deoxygenated blood round the body. Without the presence of a VSD infants rely on the ductus arteriosus and foramen ovale to transmit oxygenated blood into the systemic circulation. Such infants will be cyanosed from birth and this will proceed to profound cyanosis and collapse as the ductus arteriosus closes unless free flow across the foramen ovale is maintained. Infants with TGA associated with a VSD or large persistent ductus arteriosus shunting will have less marked cyanosis. These infants may develop heart failure some weeks later due to excess pulmonary blood flow.

Heart failure

Poor feeding, sweating and breathlessness are common presenting features of heart failure. Tachypnoea with grunting, tachycardia and hepatomegaly are found on examination. Oedema is rare unless antenatal heart failure produces hydrops fetalis. The presentation overlaps with the features of common respiratory illnesses such as bronchiolitis. Cardiac muscle disease, arrhythmias, anaemia and fluid overload may also lead to heart failure in the neonatal period. Table 6 indicates the groups of CHD presenting with heart failure in the neonatal period.

Left-sided obstructive lesions
The degree of obstruction to forward flow of blood out of the left ventricle will determine the initial presentation of this group of lesions. If the obstruction is severe or complete the systemic circulation will be supplied primarily or entirely by the ductus arteriosus resulting in a duct-dependent systemic circulation. These infants may present with poor perfusion and collapse as the duct closes. With lesser, although still significant obstruction, infants will present with heart failure over the first 2 weeks of life.

Left-to-right shunts
The only left-to-right shunt that will routinely present with heart failure in the neonatal period is a PDA in premature neonates. Other shunt lesions may be noted due to the presence of an asymptomatic murmur at this stage. Large VSDs will not present until a few weeks of age when pulmonary vascular resistance will have fallen sufficiently. Large systemic arteriovenous malformations represent a rare but important cause of heart failure in the neonatal period, particularly in infants with no other signs of cardiovascular disease. They occur most commonly at cerebral and hepatic sites. A bruit may be heard on auscultation over the appropriate site aiding the diagnosis.

Common mixing conditions
These conditions have been discussed previously discussed; they present with a combination of heart failure and cyanosis. Heart failure will predominate, and cyanosis will be less marked, if

Table 6 CHD presenting with neonatal heart failure.

Left-sided obstruction	Common mixing	Left-to-right shunts
Heart failure+collapse • Hypoplastic left heart • Coarctation of the aorta • Interrupted aortic arch • Critical aortic stenosis • Aortic atresia	Heart failure+cyanosis • Total anomalous pulmonary venous drainage • Truncus arteriosus • Double inlet ventricle • Double outlet ventricle	Heart failure • Persistent ductus arteriosus • Ventricular septal defect • Arteriovenous malformation

Table 7 CHD presenting with collapse in the neonatal period.

Duct-dependent systemic circulation	Duct-dependent pulmonary circulation	Transposition of the great arteries without VSD/PDA
Collapse+heart failure • Hypoplastic left heart • Coarctation of the aorta • Interrupted aortic arch • Critical aortic stenosis • Aortic atresia	Collapse+cyanosis • Tricuspid atresia • Pulmonary atresia • Critical pulmonary stenosis	Collapse+cyanosis

VSD/PDA, ventricular septal defect/persistent ductus arteriosus.

pulmonary blood flow is unrestricted by pulmonary stenosis or venous obstruction in TAPVD.

Sudden collapse

The presentation of a collapsed infant with poor systemic circulation or extreme cyanosis and acidosis will suggest a number of differential diagnoses, including sepsis and metabolic derangement. It is essential to consider a cardiac cause for such a presentation. Failure to do so will lead to unsuccessful resuscitation and high morbidity.[4,5] These infants will, in many cases, have had a normal neonatal examination and present in extremis over the first week of life as the ductus arterious closes. Table 7 indicates the cardiac causes of neonatal collapse.

Management of a sick neonate with suspected duct-dependent CHD

Optimising general condition

- Airway, breathing and circulation: Consider early endotracheal intubation and ventilation.
- Prostaglandin E infusion.
- Correction of acidosis, hypoglycaemia, hypocalcaemia and other electrolyte abnormalities if present.
- Sepsis and metabolic diseases should be considered in the differential diagnosis and appropriately treated.
- Echocardiography—if a competent echocardiographer is available.

When to use Prostaglandin E

Prostaglandin E given as an intravenous infusion will reopen or keep open the ductus arteriosus. It should be considered in: (1) infants with profound or increasing cyanosis in whom a duct-dependent pulmonary circulation is suspected; or in (2) infants presenting with acidosis and shock in whom a duct-dependent systemic circulation is suspected. The initial starting dose is usually 5 ng/kg/min. This dose may need to be increased by 5 ng/kg/min increments. Doses as high as 50 ng/kg/min may be required, however, alternative diagnoses should be considered if the clinical response is poor. Apnoea is the most significant side effect and this should be closely monitored for. Other side effects include jitteriness, convulsions, low-grade pyrexia, flushing and diarrhoea and should resolve on dose reduction.

Other aspects to management

Early discussion with a tertiary paediatric cardiology facility will allow optimisation of management throughout. All infants becoming symptomatic with CHD will require transfer to tertiary services for diagnosis and definitive management. Transfer of the infant should be carried out by an expert team of intensivists. The parents should be informed of their infant's progress throughout.

Conclusion

Careful evaluation and early diagnosis of CHD is important. In most centres echocardiography is not immediately available and therefore careful clinical assessment is vital in deciding whether a newborn baby is reasonably safe to send home. Despite careful evaluation, a few babies will present in poor condition when the duct closes and CHD should be considered in any baby becoming unwell in the first weeks of life. These babies may have had no or minimal cardiovascular signs at the newborn examination. Echocardiography remains the gold standard for the description of specific lesions however most infants can be stabilised without this facility before transfer to paediatric cardiology facilities.

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