REDELVE INTERNATIONAL PUBLICATIONS

Journal of Applied Sciences | ReDelve
Volume 2019, Issue 02
Review Article: RD-APS-10008

Approach to Congenital Heart Disease - Review

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Citation: *Kaushal M (2019) Approach to Congenital Heart Disease - Review. Redelve Journal of Applied Sciences. ReDelve | RD-APS-10008.*

Received Date: *24 January 2019;* ***Accepted Date:*** *26 February 2019;* ***Published Date:*** *05 March 2019*

Abstract

Congenital Heart Disease (CHD) is the most common congenital disorder in newborn. Critical CHD occurs in one fourth of these babies that may be life threatening if not detected in time. They may mimic with other conditions like respiratory distress, sepsis and inborn error of metabolism. Baby may appear normal at birth and later may become critical. The risk of morbidity and mortality increases when there is delay in diagnosis and treatment. Right approach is early identification before it becomes critical, differentiate it from other conditions and then do appropriate investigations. Timely intervention is the key to success of its survival. This review tries to find the latest and best ways to approach a baby with CHD, which includes pulse oximetry screening, diagnosis and management of CHD.

Keywords: Congenital Heart Disease; Cyanosis; Pulse Oximeter

Introduction

Congenital Heart Diseases (CHD) refer to structural or functional heart diseases, which are usually present at birth although some may be discovered later. The incidence is 8-10/1000 live births. Nearly 33% to 50% of these defects are critical, requiring early intervention but only less than half can be picked up by antenatal ultrasound where only four-chamber fetal heart view is done.

Most newborn with critical CHD is symptomatic and can be identified after birth but some are not diagnosed until after discharge. Risk of mortality and morbidity increases if there is delay in diagnosis. Timing of presentation varies with the underlying lesion and its dependence on Patent Ductus Arteriosus (PDA).

Closure of PDA can precipitate rapid clinical deterioration like severe metabolic acidosis and cardiogenic shock. A recent approach for screening, early diagnosis and treatment is described below.

Asymptomatic at birth and till discharge from hospital

Few or no symptoms at birth: Early detection remains challenging because clinical findings may be subtle or absent immediately after birth, and prenatal screening does not detect all cases of CHD. Pulse oximetry is an effective screening measure for CHD and universal screening is

recommended by the American Academy of Pediatrics.

Pulse Oximetry Screening

Newborn screening is specifically directed towards identifying seven specific lesions:

1. HLHS
2. Pulmonary Atresia (PA)
3. TOF
4. TAPVC
5. TGA
6. Tricuspid Atresia (TA)
7. Truncus Arteriosus (TAC)

- **Timing** - performed after 24 hours of life or as late as possible if early discharge is planned. If done early should be repeated.
- **Instrument** - a motion-tolerant pulse oximeter.
- **Probe placement** - Right hand (preductal) and either foot (postductal). Screening at both locations can occur simultaneously or in direct sequence.
- **Personnel** - Qualified and trained personnel.
- **Criteria for positive screen** - A positive test includes fulfilling **one** of the following three criterion:
- SpO2 <90 %
- SpO2 <95 % in both upper and lower extremities on three measurements, each separated by one hour
- SpO2 difference>3 % between the upper and lower extremities
- Limitation: failure to identify left obstructive lesions, acyanotic lesions, such as VSD. A negative result does not exclude potentially significant CHD.

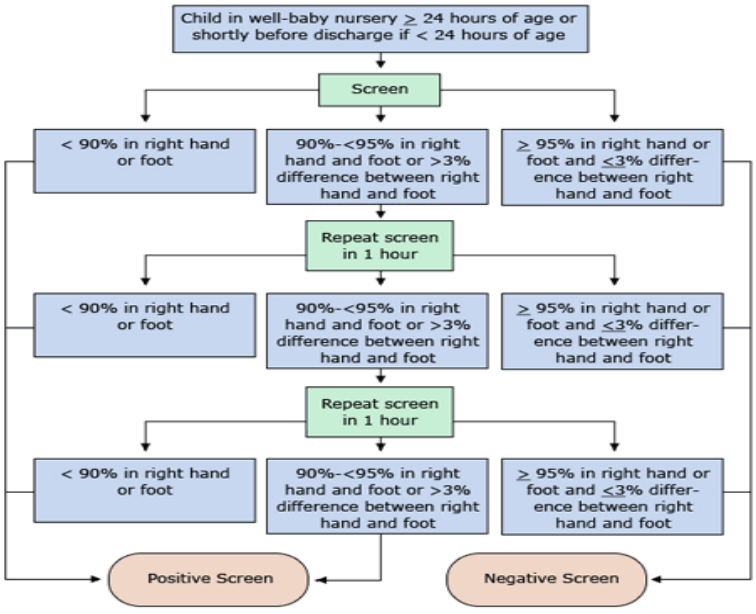

Figure1: Interpretation of screening test.

Early serious life-threatening presentation

Must suspect CHD in babies who present with shock, cyanosis, tachypnea and pulmonary

edema.

a. **Shock:** Cardiomegaly or murmur points towards cardiogenic shock and must be differentiated from septic shock. It occurs in left heart obstructive lesions (eg, HLHS, critical AS and IAA) where systemic perfusion is lost.

b. **b. Cyanosis:** Cyanosis is observed in nonductile-dependent lesions like TAPVC, TOF, TA and TAC. Differential cyanosis occurs in critical COA or IAA, where the deoxygenated flow through the ductus supplies the lower half of the body's circulation, but oxygenated blood flow from the left heart supplies the upper body via the vessels proximal to the site of arch obstruction. Cyanosis occurs when reduced hemoglobin is >3 g/dL and may be absent in mild desaturation (>80 %) or anemia. PDA closure can precipitate profound cyanosis in duct dependent pulmonary circulation (critical PS or PA), Duct dependent systemic circulations (including HLHS and critical AS) and mixing between parallel pulmonary and systemic circulations (TGA).

c. **Pulmonary edema:** It occurs in TAC, obstructed TAPVC, and Pulmonary hemorrhage in PDA due to rapid fall in pulmonary vascular resistance.

C. Later presentation - Around two weeks of age so postnatal visits must include detailed cardiac examination.

I. Clinical manifestations

1. Feeding difficulty- interrupted feeding, decreased intake, taking too long time
2. Respiratory distress- fast or hard breathing, persistent cough or wheeze.
3. Central cyanosis or persistent pallor
4. Excessive irritability
5. Excessive sweating during feeding or sleep
6. Poor weight gain
7. Decreased activity or excessive sleeping

II. Examination

General physical examination

Cyanosis (oral mucosa or nail beds), skin mottling, and ashen grey color are clues to severe cardiovascular compromise. Look for Pattern of breathing, work of breathing and use of accessory muscles. Palpation of distal extremities, temperature and Capillary Refill Time (CFT) is imperative. The cool neonate with delayed CFT should be evaluated for CHD. Absent or diminished distal pulse is suggestive of obstruction of the aortic arch.

Cardiovascular examination

1. **Abnormal heart rate** - HR < 90/min or > 160/min for neonates up to six days of age, ECG is performed to look for arrhythmia
2. **Precordial activity** - Precordial palpation ascertains whether the heart is located on the left side of the chest. Dextrocardia is often associated with complex CHD. In addition, palpation may detect the following:

1. Ventricular impulse in the lower left parasternal area suggestive of RV volume or pressure overload.
2. Increased apical activity suggestive of LV volume or pressure overload.
3. Thrill due to outflow tract obstruction or a restrictive VSD.
4. S2 splitting - it is physiological with inspiration and becomes single during expiration.

Splitting is audible in 80 % of normal newborns by 48 hours of age (If HR <150/min). Single S2 occurs in Aortic and pulmonary atresia, Truncus arteriosus and PAH. A widely or fixed split S2 occurs with ASD and RBBB's.

5. Murmurs - the presence of a murmur is often associated with CHD except for HLHS, TGA, PA and cardiomyopathy, AS where murmur is absent.

a. Innocent murmurs

Commonly occurs due to pulmonary branch stenosis (peripheral pulmonary stenosis). A grade 1-2/6, mid-systolic, high-pitched or blowing ejection murmur heard best in pulmonary area with radiation to axilla and back. Still's murmur is due to vibrations of the attachments of the pulmonary valve leaflets. These low pitched, vibratory, musical, grade 1-2/6 systolic ejection murmurs are usually best heard between the lower left sternal border and apex.

Protocol for a baby with innocent murmur

1. Review prior to discharge from hospital.
2. Check post-ductal (leg) 'spot' SpO2 before discharge. If post-ductal SpO2 < 95%, doECG/cardiology review.
3. If the diagnosis of innocent murmur is confirmed, review in OPD after 4 weeks. Inform parents to review earlier if baby develops a change in its feeding/breathing or colour.
4. An in-house echocardiogram is desirable (but is not essential) prior to discharge.

b. Pathologic murmurs

The following features of murmurs are associated with structural heart disease:

1. Murmur intensity grade 3 or higher
2. Harsh quality
3. Pansystolic duration
4. Loudest at upper left, upper right sternal border, or apex
5. Abnormal S2
6. Absent or diminished femoral pulses or noncardiac abnormalities are associated with CHD.

Approach to a baby with pathologic murmur

1. Review by senior specialist to confirm clinical features and/or assess need for admission to the Neonatal Unit.
2. Check post-ductal (leg) 'spot' SpO2 before discharge. If post-ductal SpO2 < 95%, do ECG/cardiology review.
3. Perform 4 limbs BP measurement.
4. ECG and ECHO prior to discharge.
5. **Peripheral arterial pulses** - Decreased or absent pulses in the lower extremities with strong upper extremity pulses suggests COA or IAA.
6. **Four-extremity blood pressure** - A systolic pressure more than 10 mm Hg higher in upper body compared to lower body is suggestive of COA or IAA.

C. **Respiratory abnormalities**- Tachypnea, grunting, nasal flaring, retractions and head bobbing are the features of pulmonary congestion due to LA volume overload.

D. **Hepatomegaly** - Hepatomegaly occurs in HLHS, coarctation, critical AS and cardiomyopathy or infradiaphragmatic TAPVC. A midline liver suggests heterotaxy syndromes associated with asplenia or polysplenia. Pulmonary disease causes push down

liver, due to hyperinflation and flattened diaphragm but the liver span is not enlarged.

E. Extra cardiac abnormalities - Skeletal abnormalities, involving arms are often associated with cardiac malformations. CHD may be a component of many specific syndromes and chromosomal disorders.

III. Investigations

An Arterial blood gas - ABG is mandatory in neonates with cyanosis. An elevated PCO2 usually indicates pulmonary disease or pulmonary congestion. Metabolic acidosis indicates poor cardiac output and pending shock. Low Sao2 and normal Pao2 indicates to methemoglobinemia.

	PaO2 (Spo2) @ FiO2 = 0.21		PaO2 (Spo2) @ Fi02 = 1	CO2 Mm Hg
Normal	>70 (>95)		>300 (100)	35
Pulmonary disease	50 (85)		>150 (100)	50
Neurologic disease	50 (85)		>150 (100)	50
Methemoglobinemia	>70 (<85)		>200 (<85)	35
Cardiac disease				
Parallel circulation	<40 (<75)		<50 (<85)	35
Mixing with reduced PBF	<40 (<75)		<50 (<85)	35
Mixing without restricted PB	40 to 60 (75 to 93)		<150 (<100)	35
Differential cyanosis	Preductal	Postductal	Variable	35-50
	70 (95)	<40 (<75)		
Reverse Differential cyanosis	<40 (<75)	>50 (>90)		

Table 1: Hypoxia test for diagnosis.

Hyperoxia test

The hyperoxia test is useful in distinguishing cardiac from noncardiac causes of cyanosis, especially pulmonary disease. In this test, PaO2 is measured in the right radial artery (preductal) and in a lower extremity artery (post ductal) during the administration of room air and 100 % oxygen. The changes in PaO2 are used to differentiate the various cardiac and noncardiac causes of neonatal cyanosis (Table 1).

Chest radiograph

The features suggestive of specific cardiac lesions are heart size or shape, pulmonary vascular markings, and situs of the aortic arch.

a. Heart size or shape

i. **Heart size:** Patients with left-sided obstructive lesions may have cardiomegaly due to heart failure. Extreme cardiomegaly due to dilated RA (since this chamber is very compliant) is seen in pulmonary atresia with intact ventricular septum or Ebstein's anomaly. Enlarged RV gives horizontal appearance of heart.

Diagnosis	Physical examination		Chest xray			ECG	
	S2	Murmur	Heart size	PBF	Percent right aortic arch	QRS Axis	Hypertrophy
TGA	Single	None	↑	↑	4	90-150	Nml
TOF	Single	Sys	Boot	↓	20	90-150	Nml
HLHS	Single	Sys	↑	VC		90-150	↓ LV forces
PA-IVS	Single	Sys	↑↑	↓		30-90	LVH, RAE
PS	Single	Sys	↑	↓		30-90	LVH, RAE
TAPVC	Split	Sys	↑/nil	↑VC		90-150	RAE
Tricuspid atresia	Single	Sys	↑	↓		-30 to -90	LVH, RAE
Truncus arteriosus	Single	Sys+dys	↑	↑	30	90-150	Nml
Ebsteins	Split	Sys	↑↑	↓		90-150	RAE

Table 2: Findings on Examination, X-ray and ECG of Cyanotic Heart disease.

ii. **Heart shape:** Characteristic abnormalities of heart shape are associated with specific lesions.

1. TOF - Boot-shaped
2. D-TGA - Egg-on-a-string pattern caused by a narrow mediastinal shadow produced by the anterior-posterior rather than right-left relationship of the great arteries.
3. TAPVC - Early feature is white out lung like severe HMD and Figure of eight or snowman heart is a late appearance.

b. Pulmonary vascular markings

Pulmonary vascular markings are increased in patients with truncus arteriosus or common AV canal defects. Pulmonary venous congestion due to heart failure is characterized by indistinct vascular markings spreading in a butterfly distribution from the central region of the chest often seen in obstructed TAPVC, HLHS, severe COA and Cardiomyopathy.

c. Situs of aortic arch

The normal anatomy is a left-sided aortic arch with indentation of the left side of trachea as the arch crosses over the left main stem bronchus. Right aortic arch is found in TOF (20%) and of TAC (30%). Any lesion with right sided aortic arch is considered as TOF as it is much more common.

Diagnosis	Physical examination		Chest x-ray		ECG	
	S2	Murmur	Heart size	PBF	QRS Axis	Hypertrophy
TGA	Single	None	↑	↑	90-150	Nml
TOF	Single	Sys	Boot	↓	90-150	Nml
HLHS	Single	Sys	↑	VC	90-150	↓ LV forces
PA-IVS	Single	Sys	↑↑	↓	30-90	LVH, RAE
PS	Single	Sys	↑	↓	30-90	LVH, RAE
TAPVC	Split	Sys	↑/nil	↑VC	90-150	RAE
Tricuspid atresia	Single	Sys	↑	↓	-30 to -90	LVH, RAE
Truncus arteriosus	Single	Sys+dys	↑	↑	90-150	Nml
Ebsteins	Split	Sys	↑↑	↓	90-150	RAE

Table 2: Findings on Examination, X-ray and ECG of Cyanotic Heart disease.

C. Electrocardiogram (ECG) When interpreting ECG, we should look at:

1. Rate and rhythm
2. P, QRS and T axes
3. Intracardiac conduction interval
4. Chamber enlargement or hypertrophy
5. Pericardial disease, ischemia, infarction, or electrolyte abnormalities.

In the fetus, RV has larger volume load than LV since there is limited pulmonary flow and thus reduced blood volume in the left heart. As a result, the normal neonatal ECG has RAD (QRS axis +90 to +180 degrees) and a precordial pattern of RVH. Lesions with small RV: LAD for age (for pulmonary atresia intact ventricular septum typically +30 to +90 degrees; for tricuspid atresia with normally related great arteries typically -30 to -90 degrees). Right atrial enlargement - Tall peaked P waves most easily identified in lead II.

D. Echocardiography

Indications for Echocardiography

1. Murmur.
2. Failed hyperoxia test or has an equivocal result.
3. Hypoxemic respiratory failure -extra-pulmonary shunting/PPHN.
4. Diagnose and assess hemodynamically significant PDA.
5. Differential saturation or BP between upper and lower limbs.

6. Cardiomegaly in X-ray.
7. Screening ECHO e.g. antenatal concerns or family history of CHD.

Echocardiography includes

1. Detailed evaluation from multiple views (subcostal, apical, parasternal and suprasternal)
2. 2Dand M mode echo helps in assessing systolic ventricular function.
3. Doppler techniques to assess pressure gradient across stenosed valves, reduced flow in descending aorta, and to estimate RV pressure by measuring pressure gradient between RV and RA.
4. Color Doppler defines direction of blood flow in shunts and regurgitant valves, acceleration of flow as across ASD or stenosed valves and finds collateral vessels and AV fistulas.

E. Diagnostic catheterization:

Tosee aortopulmonary collaterals in TOF, RV to coronary fistula in PA and to study hemodynamics after initial palliative surgery.

IV. Initial management

A. General supportive care

1. Temperature maintenance.
2. Vital signs including Four limb saturation and BP.
3. 12 lead ECG.
4. An adequate airway should be established immediately.
5. Oxygen and/or mechanical ventilation as needed.
6. Fluids and inotropes if poor perfusion and hypotension.
7. IV access- UVC/UACs.
8. Correction and monitoring of acid-base balance.
9. Metabolic derangements (hypoglycemia, hypocalcemia) corrected.
10. Treat sepsis with antibiotics and polycythemia (PCV > 70%) with partial exchange.

B. Specific CHD measures

A Prostaglandin E1

Start PGE1 if suspecting duct dependent lesion do not wait for the ECHO.

- If the ductus is large start with 0.01 mcg/kg/min (maximum dose of 0.1 mcg/kg/min) and if restrictive or the status of the ductus is unknown, then start at 0.05 mcg/kg/min
- Complications include hypotension, tachycardia, and apnea. Intubation if apnea occurs at any time during infusion.
- Deterioration after starting PGE1occurs in obstructive TAPVC, HLHS, cortriatriatum, severe mitral stenosis or atresia, or D-TGA associated with restrictive atrial shunting. Urgent echo followed by interventional cardiac catheterization or surgery.
- Prostaglandin E2 = Dinoprostone: may also be used. Dose is Oral 20-25 mg/kg/hourly and IV 0.003mcg/kg/minute initially and upto 0.02mcg/kg/minute.

B Cardiac failure

- Oxygen therapy.
- Restrict fluid intake to 100-120 ml/kg/day.
- Check urea and electrolytes for base line values.

- Furosemide 1-2mg/kg once or twice daily.
- Oral digitalization: 15mcg/kg/dose and total 3 doses at interval of 8 to 12 hours.
- Maintenance oral: 10mcg/kg/dose every 24 hours.
- Check electrolytes regularly and give potassium supplements if needed.
- Modify the dose according to weight.
- When digoxin toxicity is suspected check digoxin levels.

C. Arrhythmias

Paroxysmal supra ventricular tachycardia

It is the commonest serious arrhythmia HR- 180-300 /min with normal QRS complexes on ECG. Look for abnormal P waves, short PR interval and delta waves suggestive of WPW syndrome.

Treatment

- Try vagal stimulation first ice bag on the face or massage the eyes for a few seconds. Try twice under ECG monitoring if no response:
- Adenosine is the drug of choice. Give rapid push 50mcg/kg/dose and increase by 50 mcg/kg/dose every 3 to 4 minutes to a maximum of 300 mcg/kg/dose. Response is immediate with transient flushing and arrhythmia seen on ECG. Recurrence is common but can be repeated at 2 to 5 minutes' intervals.
- DC Cardio version: If hemodynamically unstable cardio version with 0.5 – 2 joules /kg.
- Maintenance drugs: Digoxin- 5 mcg/kg BD or propranolol 0.5-1mg/kg/dose TDS. Propranolol may cause apnea and hypoglycemia. Digoxin should not be given in WPW syndrome because of risk of enhancing ante grade conduction across the accessory pathway. Amiodarone may also be used. Verapamil is contraindicated.

1. **Atrial flutter and AV reciprocating tachycardia:** Rapid trans esophageal atrial pacing.
2. **Ventricular Tachycardia:** It is associated with hypoxemia, shock, electrolyte disturbance, digoxin and catecholamine toxicity, prolonged QTc syndrome and intra myocardial tumors.

Treatment

- Treat underlying cause.
- Hemodynamically stable- lidocaine bolus (1-2mg/kg) and maintenance (20-50ug/kg/min).
- Hemodynamically compromised is given DC cardio version 1-2j/kg.

Ventricular Fibrillation

It is a preterminal arrhythmia. There is coarse irregular pattern on ECG with no identifiable QRS complexes. There are no peripheral pulses or heart sounds on examination. CPR should be initiated and defibrillation starting of 1-2 j/kg performed. A bolus of lidocaine 1mg/kg followed by infusion should be started. Once the neonate is resuscitated the underlying problem evaluated and treated.

Congenital Heart Block

May be isolated or associated with a-v septal defects, maternal SLE and anti ro antibodies. Usually asymptomatic if HR >50/min, narrow QRS, normal heart size; treatment of Heart failure (HR < 40/min) or associated heart disease.

Treatment

- Diuretics if heart failure.
- Isoprenaline infusion in 5% Dextrose, 0.2mcg/kg/min.
- Cardiac Pacing.

d. Cardiac catheterization - palliative by improving cyanosis or be corrective by relieving obstruction to flow.

1. Balloon atrial septostomy - D-TGA with restrictive ASD.
2. Balloon valvuloplasty - critical PS or AS.
3. Trans catheter occlusion of pulmonary AV malformations.

VI. Transport

There should be detailed and complete communication of information between the Referring hospital staff, the transport team and accepting hospital staff under instructions of pediatric cardiologist.

1. Communication to referral hospital
2. Reliable vascular access and if possible UVC to be inserted
3. Prostaglandin infusion with infusion pump titrate to keep spo2 at least 80%
4. Inotropes if required
5. Patent airway if necessary, intubate and ventilate
6. Correct blood pressure, acidosis and maintain euglycemia.
7. Avoid 100% oxygen for the risk of duct closure.
8. Hemodynamic status including Heart rate, BP, and distal perfusion should be reassessed.

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