Newborn in distress: Subject review

Approach to infants and children with Cyanotic congenital heart disease

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Cyanosis is the bluish discoloration of skin and mucous membranes of tongue, lips, buccal mucosa and conjunctiva resulting from deoxygenation of capillary blood. The term cyanosis has origin from a Greek word ‘Kaunosis’ meaning blueness. Perhaps the first description of a cyanotic heart disease comes from an ancient Egyptian papyrus, 3000 years old which describes a sick child who died – “His lips are ruddy”. Cyanosis can be central or peripheral. In central cyanosis there is definite blue color of tongue and oral mucosa along with skin coloration. Tongue is perhaps the best indicator of cyanosis as it is rich in vascular supply and is less likely to be pigmented. Acrocyanosis, seen most often in neonates, is an evanescent blueness which lasts for a few hours. Any cyanosis lasting for more than 6-8 hours in a neonate is almost always abnormal. Shock sometimes can cause cyanosis due to venous stasis.

Recognizable cyanosis is due to a reduced Hb content of more than 4-6 gm/dl in capillary blood. It can occur also due to high (> 1.5 gm/dl) content of methemoglobin or very rarely high (>0.5 gm/dl) content of sulfhemoglobin. Recognizable cyanosis depends on hemoglobin value also. With a normal or high Hb content, desaturation will produce a significant amount of reduced Hb to cause cyanosis, while in severely anemic infant, even in the presence of desaturation, reduced Hb will not reach the value to cause clinical cyanosis.

Recognizing central cyanosis is an infant, especially a neonate is quite tricky. Ambient light, wall paint, skin color and examiner’s experience are some of the factors determining accurate detection of cyanosis. In a fair skinned child a saturation below 85% is required to produce clinical cyanosis. If any baby is really blue its saturation will be usually around 70 - 75 %.

<table>
<thead>
<tr>
<th>SITUATION</th>
<th>SITUATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial desaturation</td>
<td>&lt; 95%</td>
</tr>
<tr>
<td>Hypoxemia</td>
<td>&lt; 90%</td>
</tr>
<tr>
<td>Clinical cyanosis</td>
<td>&lt; 85%</td>
</tr>
</tbody>
</table>

Central cyanosis in a heart disease is due to a right to left shunting either intracardiac or extra cardiac. To put it in a different way, congenital cyanotic heart diseases are anomalies in which some systemic venous return will inevitably reach the systemic circulation without passing through lung. There are however many potential pitfalls in clinical recognition of congenital cyanotic heart disease.

- As discussed earlier, clinical assessment of cyanosis can be inaccurate depending on hemoglobin status.
- Congenital cyanotic heart disease with increased pulmonary blood flow due to admixture physiology can have saturations varying from 85% to 90% so that there is no clinically evident cyanosis.
- Some acyanotic congenital heart disease (CHD), especially large left to right shunts can develop severe chest infection and CHF and cause central cyanosis.
- Primary lung conditions sometimes can mimic cyanotic heart disease.

These caveats may be remembered when one evaluates an infant or a child with suspected cyanotic CHD.

Cyanosis in a cyanotic CHD is often uniform and constant. Occasionally differential cyanosis can occur – It occurs when upper limbs are pink and lower limbs are blue or alternatively upper limbs blue and lower limbs pink. Clinical differential cyanosis requires at least a difference of 10% saturation between upper limbs and lower
limbs. Even though an interesting term, it is quite rare clinically.

**Differential cyanosis**
Upper limbs pink; Lower limbs blue

Occurs in PDA with Eisenmenger syndrome
PPHN (Persistent Pulmonary Hypertension in Neonate)
Critical Coarctation
Hypoplastic Left Heart Syndrome (HLHS)
Interrupted Aortic Arch Syndrome (IAAS)

**Reversed Differential Cyanosis**
Upper limbs blue; Lower limbs pink

Occurs in Transposition of Great Vessels (d TGA) with large PDA and PAH with / without Coarctation.

Note: PDA Eisenmenger need not have differential cyanosis if it has
Associated ASD or VSD.

**A. Congenital Cyanotic Heart Disease In Newborn**

In a newborn with central cyanosis 70% will have congenital heart disease which are usually complex and 30% will have respiratory cause. Methemoglobinemia, though rare, can cause central cyanosis, but the baby has no distress. It can be due to drug intake or Hb M disease.

**Clinical Condition**  
<table>
<thead>
<tr>
<th>Met Hemoglobinemia</th>
<th>Normal</th>
<th>Low</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cyanotic CHD</td>
<td>Low</td>
<td>Low</td>
</tr>
</tbody>
</table>

**Central Cyanosis: Respiratory or Cardiac?**

<table>
<thead>
<tr>
<th>Respiratory Rate</th>
<th>Cardiac</th>
<th>Respiratory</th>
</tr>
</thead>
<tbody>
<tr>
<td>40-60 / mt</td>
<td>40-60 / mt</td>
<td>Usually &gt; 80/mt</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Respiratory distress</th>
<th>Cardiac</th>
<th>Respiratory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absent</td>
<td>40-60 / mt</td>
<td>Present always</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Grunting / Flaring</th>
<th>Cardiac</th>
<th>Respiratory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absent</td>
<td>40-60 / mt</td>
<td>Present</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Intensity of Cyanosis</th>
<th>Cardiac</th>
<th>Respiratory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deep</td>
<td>40-60 / mt</td>
<td>Less Intense</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Response to 100% oxygen</th>
<th>Cardiac</th>
<th>Respiratory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Limited / No</td>
<td>40-60 / mt</td>
<td>Good</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Depth of breathing</th>
<th>Cardiac</th>
<th>Respiratory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deep</td>
<td>40-60 / mt</td>
<td>Less deep</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pulses</th>
<th>Cardiac</th>
<th>Respiratory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Could be abnormal</td>
<td>40-60 / mt</td>
<td>Normal</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Circulation</th>
<th>Cardiac</th>
<th>Respiratory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Could be poor</td>
<td>40-60 / mt</td>
<td>Mostly normal</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Murmur</th>
<th>Cardiac</th>
<th>Respiratory</th>
</tr>
</thead>
<tbody>
<tr>
<td>More likely</td>
<td>40-60 / mt</td>
<td>Less likely</td>
</tr>
</tbody>
</table>

Hyperoxia test, introduced by Jones in 1976 continues to be a very useful clinical screening test to differentiate between a cardiac cause and respiratory cause for cyanosis, particularly in a neonate where the clinical differentiation can be very difficult. The baby is given 100% oxygen by mask for 10 minutes following a right radial artery sample for ABG - both PO₂ and SaO₂. A repeat ABG is done after oxygen. If PO₂ rises above 200 mm Hg it is very unlikely to be cardiac and if PO₂ fails to rise beyond 150 mmHg it is likely to be cardiac in origin. Between 150-200 mmHg, it is still less likely to be cardiac in cause. Right radial artery is preferred as it is the most proximal peripheral artery and unlikely to be influenced by presence of Coarctation, PDA etc. Other arterial sampling may be subject to misinterpretation in the presence of

1. Coarctation which is proximal to Left subclavian artery
2. Aberrant Right subclavian artery
3. TGA with Coarctation and PDA.

It is also likely to be affected in severe lung pathology where pulmonary vascular resistance is very high which, in the presence of PDA can cause right to left shunting and altered ABG in femoral artery sample.

**HYPOXEMIC BABY**

[Sao2 < 90%]

-↓

Oxygen by mask 100%

10 mts

↓

ABG

PO₂ > 200 PO₂ > 150 PO₂ > 150

Non Cardiac Less likely Cardiac? Cardiac

Pitfalls can occur in hyperoxia test. It is more likely if one uses pulse Oximetry instead of ABG, as good saturation in a neonate can occur with much lower PO₂ and hence assessment of saturation alone is not advocated.

In severe HMD (Hyaline Membrane Disease) or PPHN, PO₂ may not reach 200 mmHg and may be thought of as a congenital cyanotic heart disease.

In TAPVC (Total Anomalous pulmonary venous drainage) 100% oxygen will reduce PVR and hence increase tremendously, pulmonary blood flow thereby increasing both PO₂ and saturation significantly and one might even exclude a potentially lethal but correctable CHD.

Once a potential congenital cyanotic heart disease is diagnosed baby should be referred for cardiac evaluation including echocardiography for final diagnosis.

However certain clinical situations warrant emergency echocardiography.

- PO₂ in ABG < 50%
- Differential cyanosis
- Murmur with cyanosis
- Cyanosis with no respiratory distress

**CYANOTIC NEONATE AND ABG**

<table>
<thead>
<tr>
<th>PH</th>
<th>PCO₂</th>
<th>PO₂</th>
<th>Response to Oxygen</th>
<th>Respiratory</th>
</tr>
</thead>
<tbody>
<tr>
<td>-↓</td>
<td>-</td>
<td>↓</td>
<td>-No-</td>
<td>CCHD. ↓ PBF</td>
</tr>
<tr>
<td>-↓</td>
<td>-</td>
<td>↓</td>
<td>+</td>
<td>CCHD. ↑ PBF</td>
</tr>
<tr>
<td>↓</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
<td>Systemic Hypop erfusion</td>
</tr>
<tr>
<td>↓</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
<td>Alveolar hypoventilation</td>
</tr>
<tr>
<td>-↓</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
<td>Parenchymal lung condition</td>
</tr>
</tbody>
</table>
B. Congenital Cyanotic Heart Disease In Infancy And Childhood

Majority of infants and children present with history of cyanosis - on crying or at rest. A few infants present for the first time with cyanotic spells. Occasional child will present with a cerebrovascular accident or brain abscess.

Cyanotic children may be classified into those who have decreased pulmonary blood flow (↓ PBF) or those with increased pulmonary blood flow (PBF). This is primarily answered by history, physical examination and ultimately by chest x-ray.

CCHD with reduced PBF can be divided into two major groups.

1. With pulmonic stenosis
2. With pulmonary hypertension

Both effectively reducing blood flow across lungs and causing central cyanosis. There is a right to left shunt at some level, Intracardiac.

Pulmonic stenosis most often occurs in association with VSD (ventricular septal defect). Majority of CCHD with reduced PBF have an interventricular communication and some degree of PS. TOF (Tetralogy of Fallot) is the prototype of this group. In some, PS is associated with intact IVS (interventricular septum) and right to left shunt occurs through a small ASD or PFO (Patent Foramen Ovale).

**Group I (CCHD; ↓ PS; VSD)**

This group will have history of progressive central cyanosis from infancy, reasonably deep cyanosis, history of spells in infancy and squatting in childhood. They do not have recurring chest infections and do not exhibit features of CHF (Congestive heart failure). The physical findings include:

- Significant cyanosis
- No CHF
- No Cardiomegaly
- Quiet precordium
- Single S2 (Second sound)
- An outflow murmur of varying intensity

Precordium will be devoid of left parasternal heave or hyperkinetic impulses, JVP is usually not elevated and usually a single murmur is heard. Radiology of such children will reveal normal cardiothoracic ratio, pulmonary oligemia, small sized pulmonary arteries and sometimes right sided aortic arch. Electrocardiography will be variable and is of greater use in differentiating between individual CHD among this group.

The common lesions in this group are

1. Tetralogy of Fallot (TOF)
2. Pulmonary Atresia with VSD
3. Tricuspid Atresia*
4. DORV. VSD. PS
5. L-TGA. VSD. PS. (Corrected TGA)
6. D-TGA. VSD. PS
7. AV Septal Defect with PS
8. Single Ventricle with PS
9. Truncus Arteriosus with PS

Almost all of them share features like central cyanosis, absent CHF, normal or near normal cardiac size, single S2 and an outflow murmur. Some may have some deviation from the set pattern, which may help in reaching a more specific diagnosis.

**Pulmonary Atresia with VSD**

There is no outflow murmur. Instead children will have a high volume pulse, a murmur of either PDA or Major Aorto pulmonary collateral arteries (MAPCA) or occasionally an ejection click due to dilated aorta.

**Tricuspid Atresia**

It is not a true TOF physiology as it often has a restrictive VSD. It will have a very early presentation with deep cyanosis, absent right ventricular impulses, prominent ‘a’ wave in JVP and sometimes a left ventricular apical impulse.

**DORV. VSD. PS**

Clinically indistinguishable from TOF.

**L-TGA.VSD.PS.**

More often associated with dextrocardia; shares all the clinical features of TOF but for a very loud single S2 at pulmonary area due to an L-posed anterior aorta. There could be an apical murmur- MR like, due to Left AV valve (Tricuspid) regurgitation.

**D-TGA. VSD. PS**

Will have intense, early onset cyanosis – from newborn days onwards and outflow murmur. There could be mild cardiomegaly.

**AV Septal defect with PS**

Can have an LV impulse, an apical murmur due to
Left AV valve regurgitation (Mitral) in addition to RVOT murmur.

Single Ventricle with PS

Will be almost be like TOF; there could be an LV apical impulse, mild cardiomegaly and a long systolic murmur in spite of significant cyanosis.

Chest X-ray in group I

Both TOF and PA with VSD will have right aortic arch in 25-30% of cases, Tricuspid atresia will have no upturned apex but will have prominent right atrium. L-TGA will have an L-posed Aorta forming upper part of cardiac border. D-TGA will have prominent RA enlargement and narrow pedicle.

ECG in Group I

Include RAD. RVH with out strain. TOF will have early transition in precordial leads. RAE is not a feature in this group. The individual lesions will have certain characteristic ECG abnormalities.

CCHD; PBF; VSD with PS - ECG Findings

<table>
<thead>
<tr>
<th>P Wave</th>
<th>PR Interval</th>
<th>QRS Axis</th>
<th>RVH</th>
<th>LVH</th>
<th>BVH</th>
</tr>
</thead>
<tbody>
<tr>
<td>TOF</td>
<td>N</td>
<td>RAD</td>
<td>++</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>PA/VSD</td>
<td>N</td>
<td>RAD</td>
<td>++</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>TA</td>
<td>RAE</td>
<td>N</td>
<td>LAD</td>
<td>0</td>
<td>++</td>
</tr>
<tr>
<td>DORV</td>
<td>N</td>
<td>Prolonged</td>
<td>RAD-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>L-TGA</td>
<td>N</td>
<td>Prolonged</td>
<td>RAD-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>D-TGA</td>
<td>N</td>
<td>RAD</td>
<td>++</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>AVSD</td>
<td>RAE</td>
<td>N</td>
<td>LAD</td>
<td>++</td>
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<tr>
<td>SV</td>
<td>N</td>
<td>N</td>
<td>LAD?</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Truncus</td>
<td>N</td>
<td>N</td>
<td>RAD</td>
<td>-</td>
<td>+</td>
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Group II

Severe PS; Intact IVS (Inter ventricular septum)

Severe valvar PS in the long run can progress and cause raised filling pressures in right ventricle, which is transmitted to right atrium. If there is a PFO or ASD, right to left shunt occurs through it and causes central cyanosis later Right ventricle dilates and develops dysfunction causing considerable cardiac enlargement. The clinical presentation is unique among CCHD with decreased PBF.

ECG in Group II

ECG will show severe RVH with strain pattern. This is in contrast to TOF situation where there is no RV strain pattern as RV is used to systemic pressures from outset. RV pressure in this group can be suprasystemic causing a qR in V1 or V3 R.

ECG Findings

- NSR. RAD
- RVH. qR in V1 / V3 R
- RV strain – ST – T changes in V1-V6

Group III

CCHD; increased PBF with PAH

Just as PS causes impedance to right ventricular blood flow to lungs, severe PAH due to Pulmonary Vascular Occlusive Disease (PVOD) will cause considerable impedance to blood flow to lungs causing reduced pulmonary flow. A communication between chambers at any level will set up a right to left shunt causing central cyanosis. The classical condition, Eisenmenger syndrome, which is defined as a condition in which a left to right shunt develops severe PVOD so that bidirectional or right to left shunt occur causing central cyanosis. Eisenmenger reaction can occur in ASD, VSD, PDA, AVSD and AP window. Except ASD all Eisenmenger conditions behave similarly clinically. They will start as significant left to right shunt in infancy and develop cyanosis in the 2nd or 3rd decade, will have mild - moderate cyanosis, minimal or no cardiomegaly, no CHF and clinical findings of severe PAH which include parasternal heave, palpable P2, load and single S2, ejection murmur and early diastolic murmur from pulmonary area and no murmur from shunt lesion.

ASD will have slightly differing clinical presentation – prominent JVP, cardiomegaly, hyperdynamic precordium and in a majority, split S2 with loud P2.

Chest X-ray in group III

There will be no cardiomegaly (except in ASD). Significant dilatation of PA s, and oligemia with peripheral pruning of lung vessels are characteristic.

ECG in Group III

ECG in all conditions will show right axis deviation, right ventricular hypertrophy without strain pattern. qR in V1 or V3 is unlikely as RV pressure seldom crosses systemic levels.

Cyanotic heart disease with increased pulmonary flow will have a characteristic combination of mild cyanosis and severe CHF. This group is again divided into 2 sub groups.
**Group IV: CCHD. Increased PBF: Parallel circulation**

Transposition of great arteries (TGA) is the unique CHD belonging to this group. It will present with severe cyanosis and mild CHF - especially TGA with intact IVS. Clinical findings will be intense early cyanosis, mild cardiomegaly, mild CHF and minimal or no murmur. Murmur in this group will be due to associated PS, small VSD or PDA. **Chest X-ray** will show mild-moderate cardiomegaly, right atrial enlargement, RV contour, increased pulmonary vascular with narrow base.

ECG will show monotonous findings, right ventricular hypertrophy with RAD.

D-TGA with large VSD will behave with more CHF, severe PAH and less intense cyanosis. Similar clinical picture can occur in DORV. Sub pulmonic VSD and PAH (Taussig - Bing anomaly).

**Group V: CCHD. Increased PBF: Admixture**

If there is a complete mixing of systemic and pulmonary venous return due to a common mixing chamber, admixture lesions result. Single atrium, single ventricle and Truncus arteriosus are the classical lesions. Total anomalous pulmonary venous connection (TAPVC) and DORV. VSD. PAH also belong to this group.

Presentation include

| CHF in infancy |
| Mild cyanosis. |
| Cardiomegaly with hyperdynamic heart |
| Evidence of PAH |
| Abundant murmurs - systolic and diastolic |
| Growth failure. |

Clinically they will behave similar to a large Left to right shunt with mild cyanosis.

**ASD Like Presentation:**

CHF; Cyanosis - TAPVC, Single Atrium

**VSD like presentation:**

CHF; cyanosis - Single ventricle. DORV

**PDA like presentation:**

CHF; Cyanosis - Truncus

**X ray:** All these lesions will cause cardiomegaly and plethora of lung fields. TAPVC and single Atrium (pre tricuspid lesions) will have significant right atrial enlargement. Post tricuspid lesions - Single ventricle, DORV and Truncus will have LA enlargement or bi atrial enlargement. Truncus will have a right sided aortic arch in 40-50% of cases.

ECG in pre tricuspid admixture lesions will have right axis deviation, right ventricular hypertrophy and frequently RSR pattern in V1 or V3R. In post tricuspid lesions RAD, BVH are common.

Some congenital heart diseases defy categorization in to any of the above groups. They include Ebstein anomaly, pulmonary AV fistula (PAVF). Ebstein anomaly will have early cyanosis, cardiomegaly, prominent JVP, split S1 and S2, S3 and S4 and murmurs. In ECG, Ebstein will show RAD, VSR or splintered QRS in V1-V2, occasionally WPW syndrome. RVH is an exception. CXR will reveal cardiomegaly, pulmonary oligemia and right atrial enlargement. PAVF is a curious clinical entity which presents as cyanotic infant or child with minimal or no cardiac findings. CXR and ECG are most often normal. Echocardiographic anatomy of heart is also normal. A contrast echocardiography and later a pulmonary angiography are needed to clinch the diagnosis.

**TOF like clinical picture with**

A. High volume pulse : TOF with PDA / collaterals
   PA with VSD with MAPCA
   Truncus arteriosus with PS
   TOF with BT shunt
   TOF with AR.

B. Prominent JVP : Tricuspid Atresia
   AVSD with PS
   TOF with prolapsed TV into VSD

C. Left ventricular apex : Tricuspid Atresia
   Single ventricle, Truncus Arteriosus (+)

D. Apical murmur : L-TGA, VSD, PS.
   AVSD with PS with MR.
   Single ventricle + HLHS - Obstr. TAPVC

**CONGENITAL CYANOTIC HEART DISEASE**

**CXR / ECG correlation**

1. **CCHD. **↓ PBF: PAH; RAD. RVH
   Eisenmenger syndrome
   TGA with PVOD
   TAPVC with PVOD
   Taussig - Bing Anomaly

2. **CCHD; **↓ PBF; PAH; LAD; RVH
   AVSD with PVOD
   Single Atrium with PVOD
   DORV. VSD. PAH / PVOD
   (Sub aortic)

3. **CCHD; **↓ PBF; PAH: LV Dominance
   Truncus with PVOD
   Single ventricle of LV type

4. **CCHD; **↓ PBF; PS; RAD; RVH
   TOF
   PA with VSD
   PS with IVS
   PA with IVS
   TGA. VSD. PS
   (Sub pulm ) DORV. VSD. PS.

5. **CCD; **↓ PBF; PS; LAD: RVH
   DORV. (Sub aortico) PS
   AVSD with PS

6. **CCHD; **↓ PBF; PS: LV Dominance
   Tricuspid Atresia
   PA with intact IVS. Hypoplastic RV.
   Single ventricle PS (LV)
   TOF with Hypoplastic RV

7. **CCD; **↑ PBF; RAD: RVH
   TGA
   DORV. VSD. PAH
   TAPVC
8. **CCHD; ↑ PBF; LVD Dominance**  
   Single Ventricle. PAH  
   Truncus

9. **CCHD; ↑ PBF; LVD: RVH**  
   Single Atrium  
   AVSD with PAH

10. **CCHD: Normal ECG**  
    PAVF  
    Unroofed Coronary Sinus.  
    Lt. SVC to LA Straddling SVC with sinus venous ASD.

**Suggested Reading**


4. JA Tharakan Clinical Approach to children with Cyanotic Heart Disease in Progress in Pediatric Cardiology Vol. 1 No.2, 200 Congenital Heart Disease Ed M. Zulfikar Ahamed Pediatric Cardiology Division, Trivandrum, Kerala.


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