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CHAPTER 4

The Cyanotic Neonate

Sanjeev Chetry, Susanta Kumar Badatya, Anup Thakur

ABSTRACT

Cyanosis remains a cardiorespiratory emergency in neonatal period. Prompt attention and a diligent approach to arrive at a diagnosis are important to guide specific management. An appropriate physical examination and interpretation of examination will guide in diagnosis. However basic management should commence while arriving at possible cause for cyanosis. A neonate with minimal respiratory distress with no appreciable response to supplemental oxygen should be evaluated for cyanotic heart disease. Prostaglandin infusion can be lifesaving, when used in cases of suspected critical cyanotic heart disease.

Keywords: Acrocyanosis, Cyanosis, Cyanotic heart disease, Methemoglobin, Prostaglandin E1, Pulmonary circulation.

INTRODUCTION

Well-perfused skin and mucous membrane with oxygenated blood appears pink due to color of oxygenated hemoglobin (Hb). Cyanosis is a clinical condition in which bluish discoloration of skin or mucous membrane occurs due to presence of excess amount of reduced Hb in the circulation (>3 g/dL in arterial blood and 5 g/dL in capillary circulation).

The term cyanosis is derived from the Greek word "*kuaneos*" (means dark blue) which is used to refer the color imparted to skin secondary to reduced Hb. Depending upon underlying pathology, significance of cyanosis may range from a potential cardiorespiratory emergency to a simple disturbance of thermoregulation. Systematic approach for identification and differentiation between these pathologies holds a key to successful management.

CLASSIFICATION

Cyanosis can be classified into peripheral cyanosis and central cyanosis depending upon distribution of bluish discoloration.

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- *Peripheral cyanosis:* When discoloration is limited to the extremities only, it is termed as acrocyanosis or peripheral cyanosis. It occurs due to sluggish circulation through capillary bed leading to accumulation of enough reduced Hb and resultant bluish hue. It is physiologically present in newly borns, cold-induced vasoconstriction, and polycythemia.
- *Central cyanosis:* Central cyanosis denotes involvement of all the mucous membrane and skin due to deoxygenated Hb in major circulation throughout the body. Central cyanosis may be due to underlying cardiac and/or pulmonary disorders. These are more life-threatening situation requiring urgent medical attention.

There are few other clinical scenarios, where upper and lower part of body may be differentially cyanosed. In persistent pulmonary hypertension of the newborn (PPHN) with patent ductus arteriosus, shunting of deoxygenated blood flow into descending aorta gives *differential cyanosis* (upper part—pink, lower part—cyanosed). *Reverse differential cyanosis* (upper part—cyanosed, lower part—pink) is seen in transposition of the great arteries (TGA) with coarctation or interruption of the aortic arch, or PPHN where there is active shunt from pulmonary artery to aorta.

PATHOPHYSIOLOGY

Development of cyanosis is dependent on the absolute concentration of deoxy-Hb, type of the Hb and the degree of sluggishness of peripheral circulation, not on the ratio of oxy-Hb/deoxy-Hb, i.e., oxygen saturation. **Table 1** compares different situation of cyanosis at different level of oxygen saturation.

It is clear from **Table 1** that cyanosis is detectable at a higher value of SaO_2 in polycythemia, whereas in anemia, it is detectable at much lower value of SaO_2. Two neonates with similar extent of desaturations may not result in cyanosis, if one of them is anemic enough as compared to the other one. So cyanosis does not develop in a neonate with severe anemia until the

TABLE 1: Relationship between absolute amount of reduced hemoglobin and cyanosis.

Scenario	*Saturation*	*Hemoglobin level*	*Reduced hemoglobin*	*Cyanosis*
Mild hypoxemia	85%	15 g/dL	2.25 g/dL	No
Severe hypoxemia	77%	15 g/dL	3.45 g/dL	Yes
Polycythemia	85%	23 g/dL	3.45 g/dL	Yes
Anemia	85%	8 g/dL	1.2 g/dL	No
Anemia	60%	8 g/dL	3.2 g/dL	Yes

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saturation is too low, whereas polycythemic neonates develop at relatively higher saturation.

DIFFERENTIAL DIAGNOSIS

Cyanosis in a neonate may be of respiratory origin, cardiac origin, or central nervous system origin. **Table 2** summarizes the possible causes of cyanosis in neonates.

TABLE 2: Differential diagnosis of neonatal cyanosis.

Central cyanosis	
Respiratory cause (ventilation/perfusion mismatch, intrapulmonary right-to-left shunt)	
Parenchymal pathology: • Transient tachypnea of the newborn (TTNB) • Respiratory distress syndrome • Pneumonia • Atelectasis • Diaphragmatic hernia • Pulmonary hypoplasia • Pulmonary hemorrhage • Congenital cystic adenomatoid malformation (CCAM)	*Extraparenchymal pathology:* • Pneumothorax • Pleural effusion • Hemothorax • Congenital diaphragmatic hernia *Airway pathology:* • Choanal stenosis or atresia • Pierre Robin syndrome • Tracheal stenosis
Cardiac (intracardiac mixing or intracardiac right to left shunt)	
Decreased pulmonary blood flow: • Critical pulmonary stenosis • Tricuspid atresia • Pulmonary atresia/intact ventricular septum • Tetralogy of Fallot • Ebstein anomaly • Total anomalous pulmonary venous connection with obstruction	*Normal or increased pulmonary blood flow:* • Hypoplastic left heart syndrome • Transposition of the great arteries • Truncus arteriosus • Tetralogy of Fallot/pulmonary atresia • Complete common atrioventricular canal • Total anomalous pulmonary venous connection without obstruction
Central nervous system (hypoventilation/poor respiratory drive)	
• *Central nervous system lesions:* Meningitis, intraventricular hemorrhage, cerebral edema, and malformations • *Neuromuscular diseases:* Myopathy and peripheral neuropathy • *Drugs:* Sedatives	
Peripheral cyanosis	
• *Polycythemia* • *Normal physiological changes* • *Hypothermia* • *Shock of any etiology*	

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APPROACH TO A NEONATE WITH CYANOSIS

Clinical evaluation in conjunction with appropriate investigation helps in clinching the diagnosis. *Clinical history* including relevant antenatal, peripartum, and neonatal history will give pointers toward etiological diagnosis as depicted in **Tables 3 and 4**.

Physical examination along with vitals recording must be done meticulously as this guides diagnosis and course of management of cyanosis. Severity of respiratory distress is usually the differentiating feature between pulmonary and nonpulmonary causes of cyanosis such as cyanotic cardiac lesion or methemoglobinemia. Pulmonary disease typically manifests with laborious breathing, fast breathing, and accessory muscle over activity in form of retractions and nasal flaring. Four limb blood pressure recording, preductal and postductal oxygen saturations, heart sound characteristics, murmur characteristics guide in narrowing down possible cardiac causes of cyanosis.

TABLE 3: Clinical presentation and etiological correlation.

History	***Possible etiology***
Maternal history:	
• Diabetes	• Transient tachypnea of the newborn (TTNB), congenital heart disease
• Drugs	• Narcotic withdrawal
• Pregnancy-induced hypertension (PIH)	• Polycythemia, hypoglycemia
• Polyhydramnios	• Tracheoesophageal fistula (TEF)
• Oligohydramnios	• Pulmonary hypoplasia
Labor and delivery:	
• Prolonged rupture of membranes (PROM)	• Sepsis, pneumonia
• Asphyxia	• Encephalopathy, cerebral edema
• Cesarean section without labor	• TTNB
• Meconium-stained liquor	• Meconium aspiration syndrome (MAS), pneumothorax, or air leak
• Difficulty extraction	• Intracranial hemorrhage, Erb's palsy with phrenic nerve palsy
Examination finding:	
• Respiratory distress—tachypnea, grunting, retraction	• Respiratory
• Inspiratory stridor	• Airway obstruction
• Asymmetric chest	• Pneumothorax
• Scaphoid abdomen	• Congenital diaphragmatic hernia
• Encephalopathy	• Hypoventilation
• Cyanosis improving with cry	• Choanal atresia
• Cyanosis worsening with cry	• Laryngomalacia

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TABLE 4: Age at presentation of cyanosis in cyanotic heart diseases.

0–7 days	*7–28 days*	*>28 days*
• TGA • PS + IVS • HLHS • Ebstein anomaly • Obstructed TAPVC • PPHN	• Truncus arteriosus • TAPVC • TGA with VSD • TOF	• TOF like physiology • TGA with ASD • Truncus

(ASD: atrial septal defect; HLHS: hypoplastic left heart syndrome; IVS: intact ventricular septum; PPHN: persistent pulmonary hypertension of the newborn; PS: pulmonary stenosis; TAPVC: total anomalous pulmonary venous connection; TGA: transposition of the great arteries; TOF: tetralogy of fallot; VSD: ventricular septal defect)

INVESTIGATIONS

For evaluation of cyanosis, investigation tools help in assessment of degree of hypoxia (pulse oximetry, arterial blood gas) and detection of underlying pathophysiology (chest radiograph, echocardiography, and sepsis screen).

- *Pulse oximetry:* Pulse oximetry measures the SpO_2 continuously giving noninvasive estimation of oxygen saturation. Preductal measurement of oxygen saturation in right hand or right wrist is preferable especially in immediate neonatal period where ductus arteriosus is patent. SpO_2 value in immediate postnatal period should be interpreted according to the age in minutes before considering supplemental oxygen to relieve cyanosis. SpO_2 is typically targeted between 60 and 65% in first minute which gradually reaches to 80–85% at 5 minutes of life and 85–90% by 10 minutes of life.[1] Simultaneous measurements from preductal and postductal site gives information about mixing of blood flow through ductus arteriosus. Differential cyanosis or reverse differential cyanosis occurs due to mixing of blood through the ductus arteriosus.
- *Arterial blood gas:* This gives measurement of pO_2 in blood and associated metabolic and respiratory status. Cyanosis with structurally normal heart is always a challenge to clinician where methemoglobinemia remains an important differential diagnosis. A low oxygen saturation measurement in pulse oximetry in neonates with normal or higher pO_2 in arterial blood gases should alert clinician to investigate for acquired or inherited methemoglobinemia.
- *Chest radiograph:* Chest X-ray is a cost-effective, easily available bedside investigation in evaluation of cyanosed neonate. X-ray may provide characteristic finding regarding both pulmonary and cardiac conditions. Parenchymal lung lesions, extraparenchymal air or fluid compressions and breech in the integrity of the diaphragm can be easily picked up in a radiograph.[2] Lung malformations require CT scan for exact location and characteristics. Shape, size, and location of cardiac silhouette may give

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hint toward specific cardiac disorder. The "boot shape" heart in tetralogy of Fallot, the "egg on side" appearance of transposition of great arteries, and the massive cardiomegaly of Ebstein's anomaly, and figure of eight in total anomalous pulmonary venous return are few examples to mention.[3]

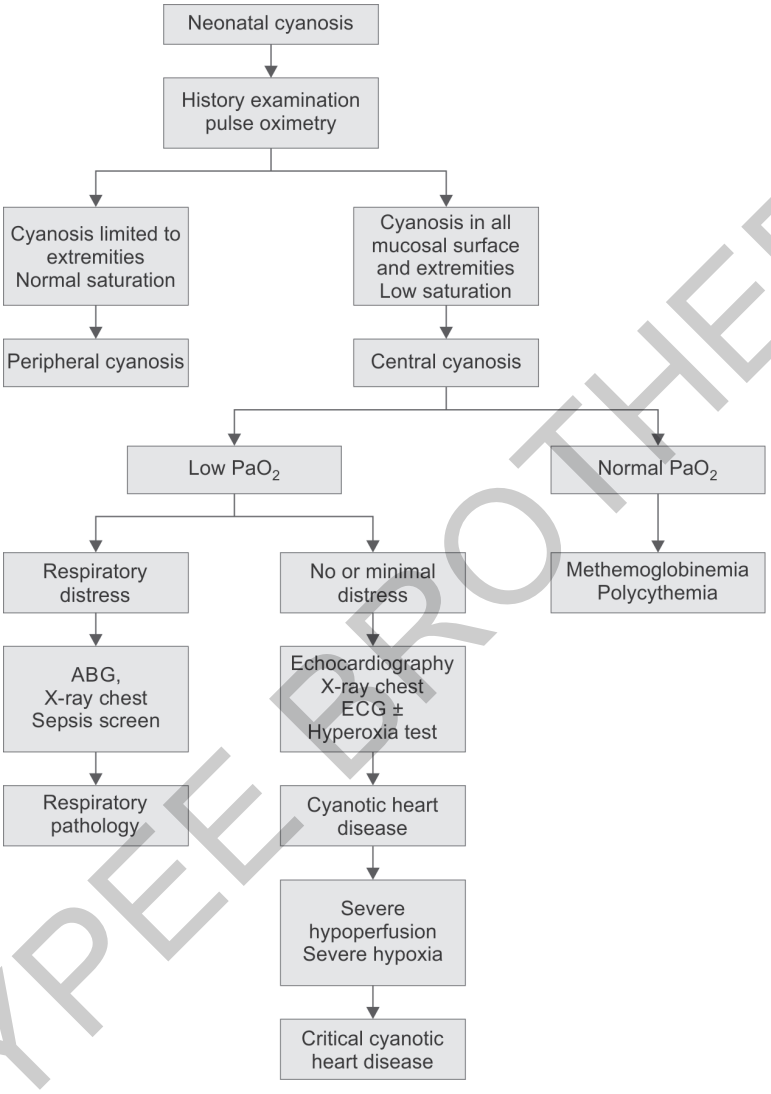
- *Electrocardiogram (ECG):* This is a useful modality for evaluation of the infant with structural congenital heart disease and cardiac arrhythmias. Neonate with left axis deviation due to left ventricular hypertrophy strongly suggests tricuspid atresia.
- *Echocardiography:* Whenever available, echocardiography is the investigation of choice for evaluation of cardiac causes of cyanosis. This gives not only the structural details of cardiac chamber and great vessels but also gives functional measurements and characteristics of intracardiac and extracardiac shunts.[4]
- *Hyperoxia test:* This serves as a clinical tool to differentiate between pulmonary and cardiac causes in cyanotic neonates. Principle underlying this test assumes that 100% oxygen will increase alveolar PO_2, leading to an increase in systemic arterial PO_2 in the absence of fixed cardiac shunts. Neonates with cyanotic congenital heart disease will fail hyperoxia test in which they will show little or no rise in pO_2 even after breathing 100% O_2. Fixed cardiac shunts means presence of shunt through the ductus arteriosus and the foramen ovale in which there will be no rise in pO_2 despite supplemental oxygen. Babies with pulmonary pathology show rise in pO_2 and pass the test. Pulmonary hypertension of neonate is an exception in which hyperoxia test fails despite structurally intact heart due to operating shunts as explained above.[5]
- *Evaluation for sepsis:* Sepsis screen and blood culture should be done to rule out sepsis **(Flowchart 1)**.

MANAGEMENT

- Cyanosis is a clinical sign which requires definitive diagnosis for management, but supportive therapy needs to be initiated while diagnosis is being established. Clinicians should differentiate peripheral cyanosis from central cyanosis. Central cyanosis needs to be managed urgently.
  - *Supportive management:* Neonates should be resuscitated by addressing the derangements in the TABC (temperature-airway-breathing-circulation). Neonate should be nursed in a thermoneutral environment using a radiant warmer. Hypothermia and hypoglycemia should be managed if present.
  - *Oxygen/ventilation:* Oxygenation will be increased by increasing alveolar oxygen concentration and/or ventilation with simultaneously resolving the lung pathology [recruitment of alveoli in respiratory distress syndrome (RDS)], expansion of lungs in collapsed lung,

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Flowchart 1: Algorithmic approach for evaluation of neonatal cyanosis.



drainage of accumulated air or fluid in pneumothorax or pleural effusion from pleural space). Central cyanosis other than that due to abnormal Hb and cyanotic cardiac lesions respond to supplemental oxygen. Excessive supplemental O_2 do have its deleterious effect in terms of oxidative stress, lung injury and retinopathy of prematurity; therefore, the use of 100% O_2 is discouraged without addressing the etiology. Minimal respiratory distress with negligible response to oxygen indicates potential cardiac disease. Oxygen supplementation in duct-dependent systemic circulation (such as critical aortic

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stenosis, interrupted aortic arch and hypoplastic left-sided heart syndrome) with moderate cyanosis may prove counterproductive as it promotes ductus closure.[6] Echocardiography is a must for babies with central cyanosis with suboptimal response to oxygen and no/minimal respiratory distress with no apparent lung pathology.

- *Prostaglandin E_1:* Cyanosis due to duct-dependent pulmonary circulations will need alprostadil to maintain ductal patency to maintain pulmonary blood flow or sufficient mixing. Typically administered dose is continuous intravenously infusion at rate of 0.05–0.1 μg/kg/min. Echocardiography should be arranged in priority for confirmation of etiology. For transportation of the baby, baby should be intubated as apnea is the most common side effect of PGE_1 therapy.[7,8]
- *Advanced cardiorespiratory support:* Patients with severe hypoxemic respiratory failure responding poorly to initial ventilator management need referral to a tertiary center with facility for high frequency ventilation, inhaled nitric oxide, or extracorporeal membrane oxygenation (ECMO).

- *Definitive management:* Once the diagnosis gets established, specific management should be initiated according to pathology. Treatment options and its prognosis are largely dependent on the diagnosis itself.

CONCLUSION

Cyanosis in a neonate is merely a sign of underlying pathology of varied etiology, so a prompt, systematic, and rational approach is needed for the diagnosis and initiation of therapy. Careful examination and high index of suspicion is needed for diagnosing cyanosis due to abnormal Hb and cardiac lesions. One should not hesitate to order investigations to differentiate between airway obstruction, parenchymal, or extraparenchymal pathology, and cardiac disease. It should be managed as an emergency by giving attention to oxygenation, maintenance of perfusion, and cause-specific management. Prognosis and treatment outcome is dependable on the underlying diagnosis.

KEY LEARNING POINTS

- Cyanosis may be difficult to interpret in immediate newborn period owing to physiological transitory changes taking place.
- Pulmonary causes for cyanosis are more common and work of breathing may give a clue for this.
- Cyanosis not responding to O_2 is an indication to look for cardiac cause for cyanosis.
- A cyanotic neonate without any respiratory distress with normal oxygen saturation points to a diagnosis of methemoglobinemia.
- Basic management should commence while arriving at possible cause for cyanosis.

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Dr Suraj Gupte figures in most of the iconic national and international *Who's Who* volumes, including the *Morqui's Who's Who*—a pre-eminent database of outstanding global personalities.

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