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Neonatology Today (NT) is a monthly newsletter for BC/BE neonatologists and perinatologists that provides timely news and information regarding the care of newborns and the diagnosis and treatment of premature and/or sick infants.



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# THE GROWING PROBLEM OF THE NEWBORN! AN APPROACH TO THE DIAGNOSIS OF CYANOTIC NEONATE FOR THE PRIMARY CARE PROVIDER

*By P. Syamasundar Rao, MD*

## INTRODUCTION

Neonates with distress caused by cardiac defects may not survive unless treated appropriately and rapidly [1-4]. Cyanosis is an important manifestation of congenital heart defects (CHD) in the neonate. An echocardiogram and/or a pediatric cardiology consultation may help make the diagnosis; however, this may not be readily available for all neonates. An understanding of issues surrounding the cyanotic neonate by the primary care physician (usually a neonatologist or a pediatrician) may be helpful. It should be understood that:

1. Complex anatomic and physiologic changes occur during the first few hours and days of life [5],
2. Severe cyanotic CHD can be present without murmurs,
3. A loud murmur does not necessarily mean that the cause of the distress in the neonate is of cardiac origin,
4. When a murmur is present, it is not typical for a given heart defect and
5. A small or normal-sized heart on a chest X-ray does not exclude serious CHD.

The objectives of this presentation are to enumerate the causes of cyanosis in the neonate, outline methods to differentiate cardiac from non-cardiac cyanosis and to present an approach to formulate a cardiac diagnosis prior to echo-Doppler and/or angiographic studies.

## CAUSES OF CYANOSIS

The causes of cyanosis may be broadly divided into five groups and these are listed in Table I.

**Table I. Causes of Cyanosis**

- Respiratory disorders
- Cyanotic heart defects
- Persistent fetal circulation
- Central nervous system disorders
- Miscellaneous

The Respiratory Disorders may be subgrouped as pulmonary parenchymal diseases and disorders causing mechanical interference with lung function and are

 

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**Table II. Respiratory Disorders**

***A. Pulmonary parenchymal diseases***

- Hyaline membrane disease
- Aspiration syndrome
- Pneumonia
- Rare disorders like pulmonary hemorrhage or Wilson-Mikety syndrome

***B. Diseases causing mechanical interference with lung function***

- Diaphragmatic hernia
- Pneumothorax and pneumomediastinum
- Tracheo-esophageal fistula
- Lobar emphysema

**Table III. Cyanotic Heart Defects**

***A. Decreased pulmonary vascular markings***

- Tetralogy of Fallot
- Pulmonary atresia (stenosis) with intact ventricular septum
- Tricuspid atresia
- Complex pulmonary stenosis

***B. Increased pulmonary vascular markings***

- Transposition of the great arteries
- Hypoplastic left heart syndrome
- Coarctation of the aorta syndrome
- Multiple left-to-right shunts

***C. Severe pulmonary venous congestion***

- Total anomalous pulmonary venous connection (infradiaphragmatic type)
- Hypoplastic left heart syndrome (with intact atrial septum)
- Severe coarctation of the aorta

**Table IV. Central Nervous System Disorders**

- Intracranial hemorrhage
- Intracerebral malformations
- Severe intracranial infections
- Primary seizure disorders

**Table V. Miscellaneous**

- Polycythemia
- Hypoglycemia
- Methemoglobinemia
- Shock and sepsis
- Maternal drugs

listed in Table II. The CHD may be divided into three groups, based on pulmonary vascular markings on a chest roentgenogram (Table III). Persistent fetal circulation is an important cause of cyanosis, related to right to left shunt at atrial and/or ductal level. In Table IV are listed central nervous system disorders that are likely to cause cyanosis, mostly based on hypoventilation. An interesting group of miscellaneous disorders causing cyanosis are listed in Table V.

## CARDIAC VS. NON-CARDIAC

A diagnosis of the cause of the cyanosis (Tables I – V) can often be made with the use of routine clinical and laboratory data. However, there are some infants in whom it is difficult to differentiate severe pulmonary disease from cyanotic CHD. Respiratory causes may be suspected if there were: complications of pregnancy and/or delivery, maternal fever prior to birth, onset of cyanosis within a few hours after birth, prematurity, inappropriate size for gestational age, asphyxia neonatorum, meconium staining, and/or low Apgar score. A chest X-ray will usually help in identifying surgical and parenchymal causes (Table II). Arterial blood gas analysis is of use in separating cardiac from pulmonary disorders; the $PaCO_2$ is elevated in pulmonary disorders, but not uniformly so in cyanotic heart defects.

Response of arterial $PO_2$ to 100% oxygen in an oxyhood for 15 minutes [1,6] is useful in the diagnosis; no significant increase in $PO_2$ (<10 torr) is seen in cyanotic CHD while the $PO_2$ increases significantly with pulmonary disease. A $PaO_2$ >150 torr is highly suggestive of respiratory disease. A higher $PO_2$ in the right radial artery than in the umbilical artery is suggestive of right to left shunting via the ductus arteriosus, seen with persistent fetal circulation and aortic arch obstruction (Interrupted aortic arch and severe aortic coarctation).

Although a lack of increase in $PO_2$ with 100% $O_2$ is highly suggestive of cyanotic CHD, there will be some neonates with severe pulmonary disease who will not show an increase in $PO_2$. In these infants applying continuous positive airway pressure (CPAP) may be helpful [7]. Application of 8 to 10 cm $H_2O$ CPAP with 100% $O_2$ for 10 to 15 minutes via a facemask will result in an increase in

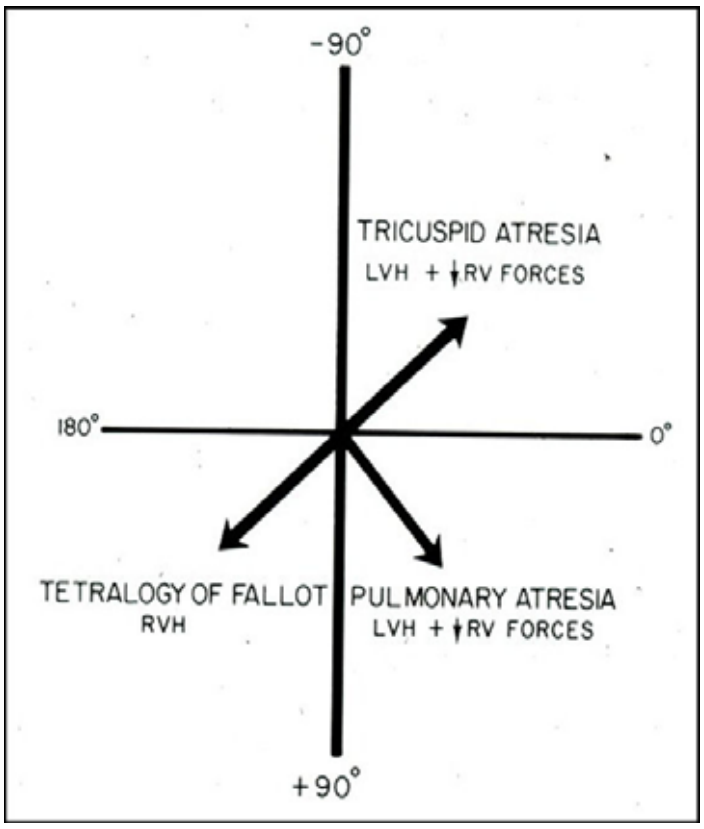


*Figure 1. Figure illustrates the usefulness of frontal plane mean QRS vector (axis) in assisting differential diagnosis of cyanotic heart defects with decreased pulmonary flow. An axis between 0 and $-90^0$ is suggestive of tricuspid atresia, an axis between 0 and $+90^0$ is indicative of pulmonary atresia with intact ventricular septum and an axis between $+90^0$ and $\pm180^0$ is associated with tetralogy of Fallot. The accompanying ECG abnormalities are shown. Cyanotic infants with complex heart defects with severe pulmonary stenosis/atresia may fall in any quadrant.*

$PO_2$ (>10 torr) in pulmonary infants, while a lack of change or a decrease may seen in cyanotic CHD babies [7].

**DIAGNOSIS OF SPECIFIC TYPE OF CHD**

Once identified as a cardiac baby, further definition of CHD is based on analysis of pulmonary blood flow on a posterior-anterior view of a chest X-ray:

(A.) Decreased pulmonary vascular markings,

(B.) Increased pulmonary vascular markings and

(C.) Severe pulmonary venous congestion.

Good quality chest film with good inspiration and without rotation is mandatory to achieve accurate assessment of pulmonary vascular markings.

**A. Decreased pulmonary vascular markings.** The most commonly encountered lesions in this group are listed in Table III. Diagnosis of infants with decreased pulmonary blood flow can be made by analysis of the electrocardiogram (ECG) (Figure 1). Sophisticated knowledge of ECG interpretation is not necessary; only calculation of axis (mean frontal plane vector) is adequate, which is taught in the medical school.

1. The infants with tetralogy of Fallot exhibit right axis deviation (+90 to $\pm180^0$). Right ventricular hypertrophy may be present.
2. Infants with pulmonary atresia with intact ventricular septum are likely to have an axis of 0 to $+90^0$. There is no right ventricular hypertrophy, instead the right ventricular voltages (R waves in leads V1 and V2 and S waves in leads V5 and V6) may be diminished and left ventricular hypertrophy may be present.
3. Neonates with tricuspid atresia will not only have left axis (superior vector) deviation (0 to $-90^0$) but also left ventricular hypertrophy and diminished right ventricular forces.
4. Complex pulmonary stenosis group includes a number of complex cyanotic CHD, namely single ventricle complexes, double outlet right ventricle, ventricular inversion and others, all associated with severe pulmonary stenosis or atresia. The axis and ventricular hypertrophy patterns vary markedly.

**B. Increased pulmonary vascular markings.** The most common defects in this group include transposition of the great arteries, hypoplastic left heart syndrome and coarctation of the aorta syndrome. Multiple left to right shunt lesions, listed in Table III, present more commonly beyond the neonatal period.

1. The infants with transposition of the great arteries usually present with severe cyanosis, minimal distress, if any, and have no signs of heart failure. Chest X-ray shows minimal cardiomegaly and the ECG is either normal or may show right ventricular hypertrophy. Blood gas analysis shows markedly reduced $PO_2$ and oxygen saturation. The $PO_2$ does not increase with 100% $O_2$ or CPAP.
2. Infants with hypoplastic left heart syndrome present with signs of severe congestive heart failure; they have minimal cyanosis, but have marked respiratory distress, hepatomegaly, poor pulses in all four extremities, massive cardiomegaly on chest X-ray, and electrocardiographic evidence for decreased left ventricular forces and right ventricular hypertrophy. A reasonably good $PO_2$ and oxygen

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**Table VI. Ductal-dependent Cardiac Defects**

***A. Ductal-dependent pulmonary flow***

- Pulmonary atresia or critical stenosis with intact ventricular septum
- Pulmonary atresia with ventricular septal defect
- Severe tetralogy of Fallot
- Tricuspid atresia
- Complex cyanotic heart disease with pulmonary atresia or severe stenosis
- Ebstein's anomaly of the tricuspid valve
- Hypoplastic right ventricle

***B. Ductal-dependent systemic flow***

- Hypoplastic left heart syndrome
- Severe coarctation of the aorta syndrome
- Interrupted aortic arch

saturation are present, but with metabolic acidosis. The $PO_2$ may increase slightly with 100% $O_2$ or CPAP, but rarely, if ever above 150 torr. The distinction between transposition of the great arteries and hypoplastic left heart syndrome is simple: *the problem of infants with transposition of the great arteries is cyanosis, cyanosis and cyanosis, and the problem of infants with hypoplastic left heart syndrome is heart failure, heart failure and heart failure.*

3. Neonates with coarctation of the aorta syndrome are usually not (but not invariably) as sick as hypoplastic left heart syndrome patients. They present with signs of heart failure, have decreased femoral pulses, cardiomegaly on X-ray and biventricular or right ventricular hypertrophy on ECG. The $PO_2$ response to ambient oxygen is excellent since the reason for their cyanosis is pulmonary edema.

**C. Severe pulmonary venous congestion**. In the neonate this is often related to total anomalous pulmonary venous connection, usually of the infra-diaphragmatic type. There is severe obstruction to the pulmonary venous return and therefore, the pulmonary venous obstructive pattern on the chest X-ray. Occasionally hypoplastic left heart syndrome with intact atrial septum and coarctation of the aorta with intact atrial and ventricular septae may cause a similar X-ray appearance.

**COMMENTS**

During the process of identification and work-up, prevention of hypothermia, maintenance of a neutral thermal environment, monitoring for and prompt treatment of hypoglycemia, treatment of hypocalcemia, monitoring acid-base status and treatment of metabolic acidosis with sodium bicarbonate ($NaHCO_3$) and management of respiratory acidosis with suction, intubation and assisted ventilation as deemed necessary are important, and should be diligently undertaken. In patients with cyanotic heart disease 30-40% $O_2$ is adequate and 100% $O_2$ is not necessary. If ductal dependant CHD is suspected (Table VI), intravenous infusion of prostaglandin E1 (0.05 to 0.1 mcg/kg/minute) should be started [8] while awaiting for confirmatory diagnosis.

*"Neonates with distress caused by cardiac defects may not survive unless treated appropriately and rapidly [1-4]. Cyanosis is an important manifestation of congenital heart defects (CHD) in the neonate."*

**SUMMARY AND CONCLUSIONS**

Cyanosis is a major manifestation of CHD in the neonate. A number of cardiac and non-cardiac entities cause cyanosis in the neonate. The majority of patients can be diagnosed based on their well-described clinical and laboratory findings. In a few patients in whom a clear-cut diagnosis is not possible, analysis of $PO_2$ response to 100% $O_2$ and CPAP may help distinguish cardiac from non-cardiac cyanosis. Once diagnosed to be a cardiac baby, careful analysis of pulmonary vascular marking on a chest X-ray may facilitate categorization into subgroups from which additional analysis of ECG and other features may assist in coming up with a reasonably accurate diagnosis. Echo-Doppler studies can certainly aid in achieving an accurate diagnosis and the author is not opposed to such studies. The exercise presented is an approach that could be taken when echo is not readily available or used prior to the arrival of echocardiographer.

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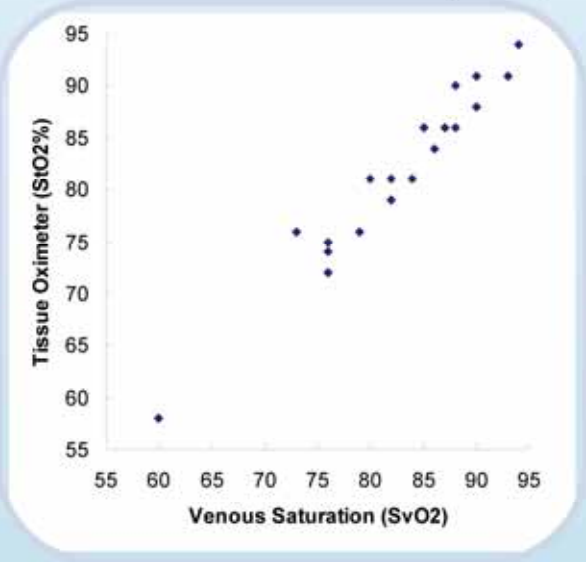


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### Preterm Birth Contributes More Than One-Third of Infant Deaths

According to the National Vital Statistics report released in early May, preterm birth contributes to more than one-third of the nearly 28,000 US infant deaths.

Although the national infant mortality rate is the lowest it's been since the US started collecting data a century ago, there's been little change recently – 6.78 deaths for every 1,000 live births in 2004 compared to 6.89 in 2000, the National Center for Health Statistics report found.

The report, "Infant Mortality Statistics from the 2004 Period Linked Birth/Infant Death Data Set" includes a new analysis tracking preterm birth-related infant deaths. The analysis, first published in the October 2006 edition of *Pediatrics*, found preterm birth contributes to nearly twice as many infant deaths within the first year of life than previously estimated. The new method reviews all causes of infant death and combines conditions, such as respiratory distress syndrome, which frequently occur in premature babies. The analysis looked at only the top 20 leading causes of infant death.



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"We have long known babies born too soon face many developmental challenges – even death," said Joann Petrini, PhD, director of the March of Dimes Perinatal Data Center. "This closer look at preterm birth gives us a better understanding of the impact of prematurity on infant survival and provides insights into the factors that have contributed to the lack of improvement in the US infant mortality rate."

Birth defects remain the leading cause of infant death, followed by prematurity, according to official reporting systems. But, using this new classification, premature birth would be the most frequent cause of infant death. The traditional methods cannot accurately gauge the true impact of preterm birth on the infant mortality rate, the NCHS said.

More than a half million babies are born too soon each year and the preterm birth rate has increased more than 30% since 1981. Babies who do survive face risks of lifelong health and developmental challenges. For more information, visit www.marchofdimes.com.

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**Study Recommends Universal Newborn Screening for Cystic Fibrosis**

Newborn screening for cystic fibrosis saves on treatment costs and would offset the actual costs of the screening programme. This new economic evidence suggests that universal newborn screening programmes for cystic fibrosis should be adopted internationally, according to an article in an April issue of *The Lancet*.

The study also showed that newborn cystic fibrosis screening reduced hospital admissions for invasive therapy.

Cystic fibrosis is a life-shortening hereditary lung disease, but treatments are available. In some regions newborn babies have been screened for cystic fibrosis for more than 25 years, and early diagnosis is associated with improvements in some clinical outcomes. Furthermore, the clinical benefit of those screened as newborn babies is associated with a lower treatment burden compared with clinically diagnosed groups. Whether these potential cost savings attributed to reduced therapeutic requirements would offset the cost of a newborn screening programme had not previously been studied.

Cystic Fibrosis screening hit the headlines in November 2006, when it was announced that UK Chancellor Gordon Brown's baby son Fraser had been diagnosed. Babies are routinely screened for the condition in Scotland (since 2003), Wales and Northern Ireland, but this is not yet the case for all areas of England including London.

Dr. Erika Sims (University of East Anglia, Norwich, UK) and colleagues from the University of Dundee, UK, used data from the UK cystic fibrosis database for 2002 to compare the treatment costs of 184 children aged 1-9 years who had cystic fibrosis that was identified by newborn screening with those of 950 children in the same age-group, who were identified after clinical presentation of the disease. Patients diagnosed by newborn screening cost significantly less to treat than those who were diagnosed clinically. Patients diagnosed on the basis of clinical presentation alone received therapy costing an estimated 60–400% more than patients diagnosed by newborn screening.

The authors conclude: "Newborn screening is associated with lower estimated treatment costs and reduced hospital admissions for invasive therapy, which suggests that indirect costs and disruption to family life will also be less. Furthermore, the potential cost savings to the yearly treatment budget could offset some, if not all, of the costs of a newborn screening service."

In an accompanying Comment, Bridget Wilcken and Kevin Gaskin (both of both the Children's Hospital at Westmead, and University of Sydney, NSW, Australia) state: "If clear clinical benefit does not always persuade governments to implement screening, cost benefits might... Some parts of the world - e.g. the middle east and possibly India—have a high frequency of cystic fibrosis, and screening might have potential in countries with a stable health-care system."

For more information, contact: Dr. Erika Sims at e.sims@uea.ac.uk.

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