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Perinatal Circulatory Physiology: Its Influence on Clinical Manifestations of Neonatal Heart Disease: Part II

By P. Syamasundar Rao, MD

INTRODUCTION

In the last issue of Neonatology Today [1] the course of the fetal circulation, mechanisms that maintain fetal circulatory pathways, distribution of the cardiac output, fetal myocardial function and postnatal circulatory changes were addressed. In this second part, the influence of postnatal circulatory changes on the presentation of important congenital cardiac defects, as well as their therapeutic implications will be discussed.

Congenital heart disease is usually well-tolerated during fetal life. However, circulatory changes following birth have marked effect on the clinical presentation and on the course of the heart disease in the early newborn period.

DUCTUS ARTERIOSUS

The ductus arteriosus is a tubular muscular structure connecting the pulmonary artery with the descending aorta. The ductus diverts the desaturated blood from the pulmonary artery into the descending aorta and from there into the placenta for oxygenation. It is kept open during fetal life by locally produced and circulating prostaglandins. After the infant is born, the ductus arteriosus constricts and closes spontaneously, presumably secondary to increased PO₂ as well as to decreasing responsiveness of the ductal musculature to prostaglandins with increas-

ing age. The importance of the ductus in various congenital heart defects will be reviewed.

Hypoplastic Left Heart Syndrome (HLHS)

In HLHS, the mitral valve, left ventricle and/or the ascending aorta are markedly stenotic or atretic, and there is no forward flow from the left heart into the body [2,3]. The entire systemic circulation depends upon the flow through the patent ductus arteriosus (Figure 1). Following birth, as the PO₂ increases, the ductus arteriosus constricts and compromises the systemic blood flow. There is concurrent decrease in the pulmonary vascular resistance, again due in part to an increase in PO₂. This will result in a marked increase in pulmonary blood flow. A combination of these events causes severe acidemia (secondary to decreased systemic perfusion) and increase in arterial PO₂ (related to increased pulmonary-to-systemic flow ratio). If untreated, severe congestive heart failure and death will result.

It seems logical not to increase PO₂ by not increasing ambient oxygen concentration so as not to hasten the ductal closure. Administration of prostaglandin E₁ will help keep the ductus patent, thus maintaining systemic perfusion. To further facilitate systemic perfusion through the ductus arteriosus, resistance to flow into the lungs should be increased. Lower FIO₂ than room air is advocated so as to increase pulmonary vasoconstriction.

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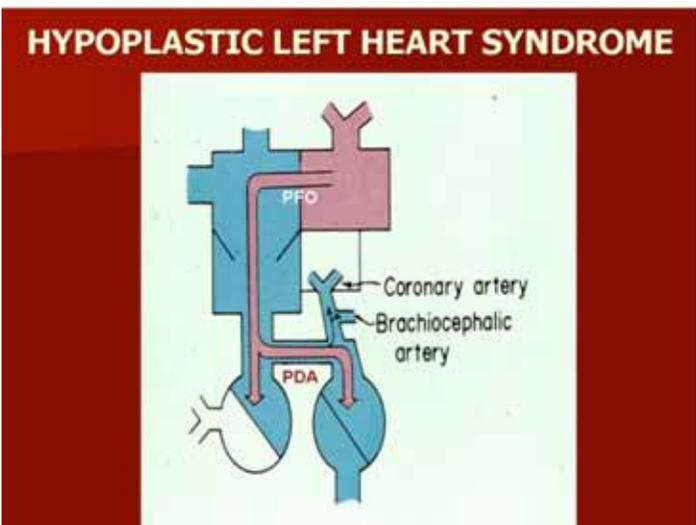


Figure 1. Box diagram of Hypoplastic Left Heart Syndrome. Because there is no forward flow from the left heart into the hypoplastic aorta, the systemic perfusion is dependent upon the patent ductus arteriosus (PDA). Retrograde flow into the brachiocephalic vessels and coronary arteries is also shown. If the ductus constricts the systemic, perfusion is compromised. The pulmonary venous return cannot exit into the left ventricle, and its egress has to be into the right atrium via the patent foramen ovale (PFO). If the foramen ovale is obstructive, the infant will develop signs of pulmonary venous obstruction.

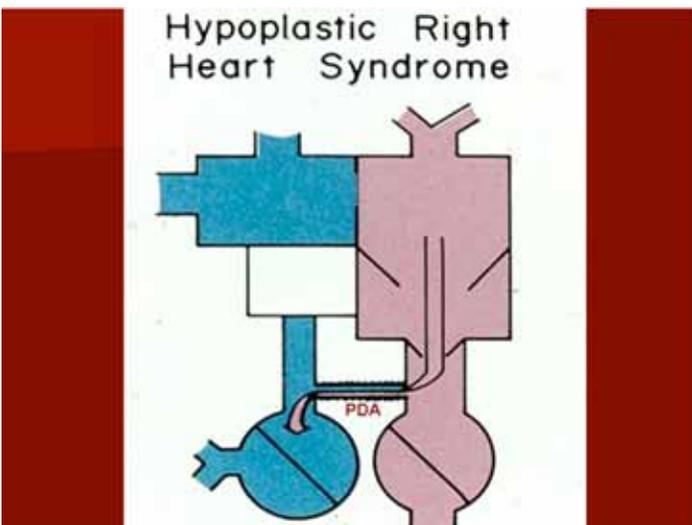


Figure 2. Box diagram of hypoplastic right heart syndrome. Since there is no forward flow into the pulmonary artery from the right ventricle, the pulmonary flow is dependent upon the patent ductus arteriosus (PDA). As the ductus begins to constrict, the pulmonary blood flow will decrease causing severe hypoxemia.

Pulmonary Atresia with Intact Ventricular Septum

Pulmonary atresia with intact ventricular septum is a complex cyanotic congenital heart defect characterized by complete obstruction of the pulmonary valve, two distinct ventricles, a patent tricuspid valve and no ventricular septal defect [4]. The right ventricle is usually, but not invariably, small and hypoplastic. Since there is complete blockage of the pulmonary valve,

Table I. 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

the pulmonary blood flow is entirely dependent upon the patency of the ductus (Figure 2). Initially (at birth) the ductus is patent and the pulmonary blood flow may be adequate with reasonable arterial PO_2 . However, as the ductus begins to close during the natural process of closure, marked hypoxemia and metabolic acidosis will ensue. Prostaglandin E_1 infusion usually helps to keep the ductus open. Several other cardiac defects (Table IA) with severe stenosis or atresia of the pulmonary outflow tract are similarly ductal dependent and are benefited by prostaglandin E_1 infusion.

Total Anomalous Pulmonary Venous Connection (TAPVC)

In TAPVC, the pulmonary veins drain into the right atrium or systemic veins. In the infra-diaphragmatic type of TAPVC, pulmonary venous obstruction is present with resultant pulmonary edema and marked increase in the pulmonary arterial pressures and resistance. If the ductus is open, decompression of pulmonary arterial tree may occur with some relief of supra-systemic pulmonary pressures although this is at the expense of deoxygenated blood bypassing the lungs.

Transposition of the Great Arteries (TGA)

In TGA the aorta arises from the right ventricle and the pulmonary artery from the left ventricle. Consequently the pulmonary and systemic circulations are parallel (Figure 3), in contradistinction to normal, in series arrangement. If there is no inter-circulatory mixing across a patent foramen ovale or patent ductus arteriosus, the infant would not survive because there is no delivery of oxygenated blood to the body. An open ductus may enhance inter-circulatory mixing, thus improving arterial PO_2 . PGE_1 infusion is helpful in abating hypoxemia in some patients with TGA.

Coarctation of the Aorta

Development of coarctation of the aorta is at least in part related to posterior shelf-like structure within folding of medial and intimal tissue of the aortic wall and in part the result of constriction of the ductus arteriosus [5,6]. Blood flow from the proximal part of the aorta into the descending aorta around the posterior shelf of coarctation is facilitated by a patent ductus (Figure 4A). This bypass mechanism is no longer available when the ductus closes (Figure 4B). Acute aortic obstruction develops rapidly

Transposition of the Great Arteries

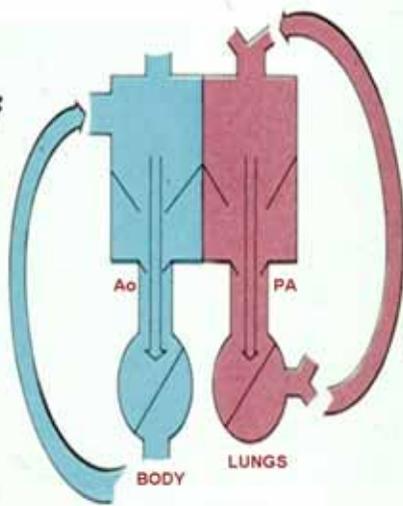


Figure 3. Box diagram of the heart showing parallel circulations in transposition of the great arteries. Note that right ventricle pumps into the aorta (Ao) (because of transposition) which goes to the body and returns into right atrium and back into the body. Similarly left ventricular output goes to the pulmonary artery (PA) and lungs and returns back to the left atrium and left ventricle to be pumped back into the lungs. Unless there are inter-circulatory communications via either a patent foramen ovale or patent ductus arteriosus, the infant cannot survive. Mixing across a ventricular septal defect (VSD) if such is present (not shown in the diagram), would also prevent progressive hypoxemia and death.

with consequent development of symptoms. Temporary relief can be obtained by prostaglandin E₁ infusion.

Interrupted Aortic Arch

This is an uncommon defect with complete lack of communication between the ascending and descending aorta; this interruption may occur at various levels. Irrespective of the level of obstruction, the blood flow goes from the pulmonary artery to the descending aorta via a ductus arteriosus, perfusing the lower part of the body. Again, during the normal course of maturation, the ductus tends to close, causing no perfusion to the lower part of the body. Prostaglandin E₁ infusion opens the ductus, restoring circulation to the lower part of the body.

DUCTUS VENOSUS

The ductus venosus connects the umbilical vein to the inferior vena cava. In the fetus a substantial portion of umbilical venous blood goes through this channel and is presumed to be kept open by the mechanical effect of the blood flowing through it. Following birth, it closes spontaneously because of lack of flow through it, although the mechanisms of closure may be similar to ductus arteriosus, alluded to above.

TAPVC

In infra-diaphragmatic type of TAPVC, some of the pulmonary venous flow will be through the ductus venosus. When the ductus venosus closes, pulmonary venous flow gets obstructed and the infant becomes symptomatic with tachypnea, cyanosis and pulmonary edema because of the necessity of the blood to pass through the liver. This is secondary to high impedance to the passage of blood through the hepatic circulation.

FORAMEN OVALE

The foramen ovale is an opening in the atrial septum formed by the septum secundum above and septum primum below and is kept patent in the fetus because of the mechanical effect of streaming of the inferior vena caval blood into the left atrium. At birth a combination of increase in the left atrial pressure secondary to increased pulmonary venous return and decrease in the right atrial pressure secondary to decreased placental return will result in apposition of the septum primum and septum secundum causing functional closure of foramen ovale. The role of patency of the foramen ovale in congenital heart defects in the neonate will be discussed.

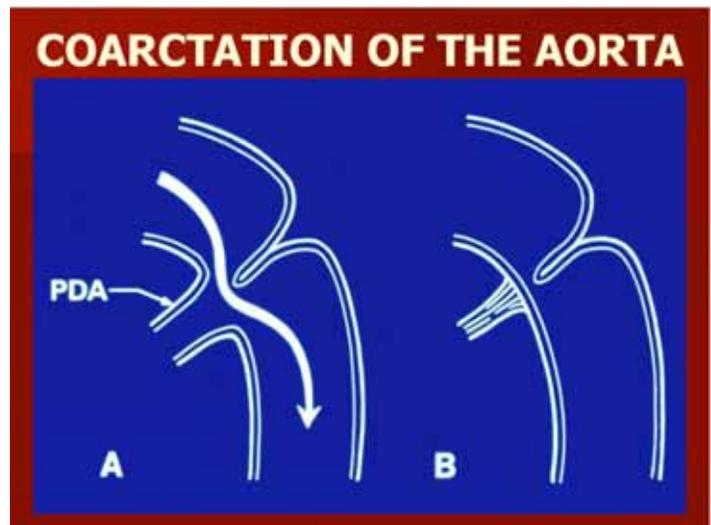


Figure 4. Diagrammatic portrayal of the flow from the aortic isthmus to the descending aorta in coarctation of the aorta: A, with open ductus (PDA), and B, with closed ductus. See the text for discussion.

Right-sided Obstructive Lesions

In defects such as tricuspid or pulmonary atresia, because of atretic pulmonary and/or tricuspid valve, there is no forward flow into the pulmonary circuit. The blood regurgitates back into the right atrium. The right atrial pressure is higher than that in the left atrium; this will keep the foramen ovale open. Therefore, an obligatory right-to-left shunt occurs across the atrial septum (Figures 4). In-



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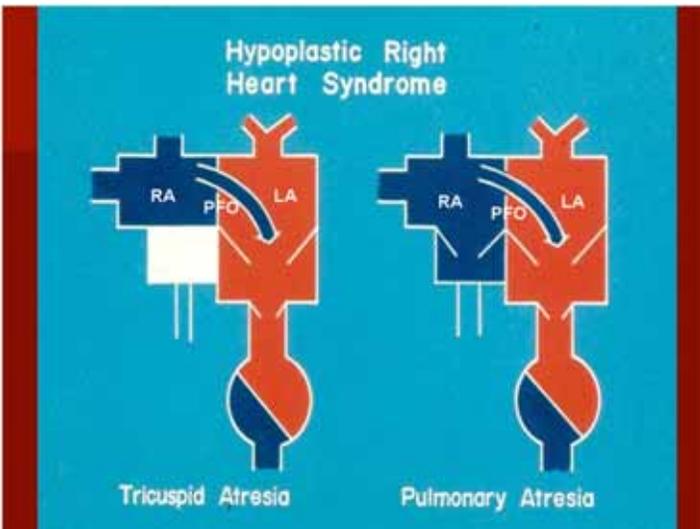


Figure 5. Box diagrams of hypoplastic right heart syndromes. In pulmonary atresia (or tricuspid atresia) the right atrial (RA) blood has to egress into the left atrium (LA) via the patent foramen ovale (PFO). If the PFO is obstructive, signs of systemic venous obstruction will develop.

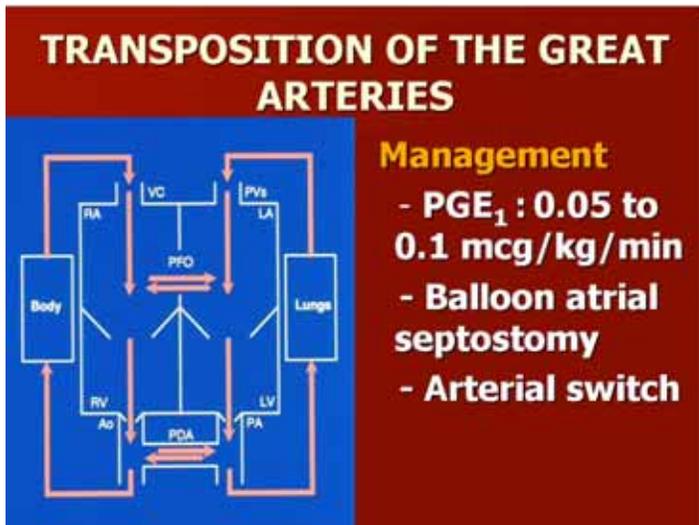


Figure 6. Box diagram of the heart showing parallel circulations in transposition of the great arteries. Note that right ventricle (RV) pumps into the aorta (because of transposition) which goes to the body and returns into right atrium and back into the body. Similarly left ventricular output goes to the pulmonary artery and lungs and returns back to the left atrium and left ventricle to be pumped back into the lungs. Unless there are inter-circulatory communications via either a patent foramen ovale (PFO) or patent ductus arteriosus (PDA), the infant cannot survive. Mixing across a ventricular septal defect (VSD) if such is present (not shown in the diagram) would also prevent progressive hypoxemia and death. PGE₁ infusion to open the ductus and balloon septostomy to enlarge the PFO are usually helpful.

deed, right-to-left shunting at the atrial level is essential for survival of the patient. While rare in neonatal period, the foramen ovale can become restrictive and may need enlargement either by transcatheter or surgical methodology [7].

TAPVC

In TAPVC the entire pulmonary venous return comes into the right atrium or into systemic veins. From there its gets distributed into the right and left heart structures. Consequently the systemic blood flow depends upon the patency of the foramen ovale. All the systemic output must pass through the patent foramen ovale. While obstruction at the foramen level can occur, it is more often than not located at other sites. Should it be the sole obstruction, temporary relief can be provided by septostomy [7].

Left-sided Obstruction Lesions

In HLHS and mitral or aortic atresia, the pulmonary venous return coming into the left atrium can't empty into the left ventricle and therefore has to be shunted into the right atrium. Consequently, the foramen ovale has to remain open in order to direct the pulmonary venous return into the right heart (Figure 1). Should the foramen ovale close, surgical or balloon atrial septostomy should be performed to relieve the interatrial obstruction [7].

Transposition of the Great Arteries

As alluded to above, in TGA the circulation is parallel and some inter-circulatory mixing is essential for survival. If the fetal circulatory pathways close, as they usually do, creation of or enlargement of atrial septal defect (Figure 6) by balloon atrial septostomy is mandatory. Such an atrial communication may sometimes be necessary even in patients with a naturally open or prostaglandin-induced patency of the ductus arteriosus.

Left-to-right shunt lesions

In lesions such as large patent ductus arteriosus (PDA) and ventricular septal defect (VSD), the pulmonary blood flow is markedly increased (see Pulmonary Vascular Bed section below) and consequently the left atrial size increases. This left atrial enlargement may cause stretching of the patent foramen ovale resulting in an additional left-to-right atrial shunting. However, such shunting maintains low pulmonary venous pressure and may even prevent pulmonary edema.

PULMONARY VASCULAR BED

In utero, the pulmonary vascular resistance is high, most likely due to the low PO₂ to which the pulmonary arterioles are exposed [1]. Following birth, normal breathing and oxygenation of the lungs take place resulting in the fall of pulmonary vascular resistance and pressure. The influence of pulmonary vascular changes in the neonate will be reviewed.

Large Inter-Circulatory Connections

In the presence of a large systemic-to-pulmonary communication such as VSD, the pressures in both ventricles are similar. Therefore, the quantity of left-to-right shunt is to a great extent dependent upon the ratio of pulmonary to systemic vascular resistance. If the pulmonary vascular resistance decreases in a normal fashion within the first few hours to days, a large left-to-right shunt and congestive heart failure will develop during the first few days to weeks of life. However, this does not usually happen in otherwise normal babies until the age of 4 to 12 weeks. This is because of delayed regression of the pulmonary vascular resistance and pulmonary arteriolar muscular thickness. Increased pressure to which the pulmonary arterioles are subjected may be responsible

for this delayed regression. However, the exact mechanism through which the high pressure acts to delay the normal involution of the pulmonary vasculature is not known.

Any cardiac defect with large inter-circulatory connection such as large patent ductus arteriosus, double outlet right ventricle, truncus arteriosus, single ventricle and others (all without associated pulmonary stenosis) affects the pulmonary vascular bed in a manner similar to that described for the VSD.

Abnormal Development of Pulmonary Vasculature

The development of fetal pulmonary vasculature is dependent upon the PO₂ to which it is exposed. Therefore, if the PO₂ of pulmonary blood is increased because of a cardiac defect, pulmonary vasculature (arterioles) remains underdeveloped and less muscular. Consequently, the pulmonary vasculature may regress rapidly producing a decrease in the pulmonary vascular resistance and development of a large left-to-right shunt much earlier than anticipated.

Prematurity

In premature infants, because of lack of complete development of the media of the pulmonary arterioles, there is less pulmonary arteriolar smooth muscle to regress; therefore, the infants with large systemic-pulmonary communications develop heart failure much earlier than expected for normal full-term newborn. But, if they also have pulmonary disease producing hypoxia, a fall in pulmonary vascular resistance may be delayed and as the infant improves from lung disease, a large left-to-right shunt and congestive heart failure will develop.

Other Factors

Chronic hypoxia also delays regression of pulmonary vasculature. Decreased ambient oxygen at high altitude and pulmonary disease may delay the development of large left-to-right shunt and congestive heart failure in patients with large inter-circulatory connections.

High left atrial pressure caused by left ventricular inflow obstructions (mitral stenosis/atresia and HLHS complexes) also cause delayed involution of the pulmonary arterioles, although the mechanism by which this is affected is not understood.

THERAPEUTIC IMPLICATIONS

Therapeutic implications of post-natal circulatory changes in some cardiac defects will be reviewed.

Transposition of the Great Arteries

In TGA severe hypoxemia develops as the PDA and foramen ovale close. Initially prostaglandin E₁ infusion should be started



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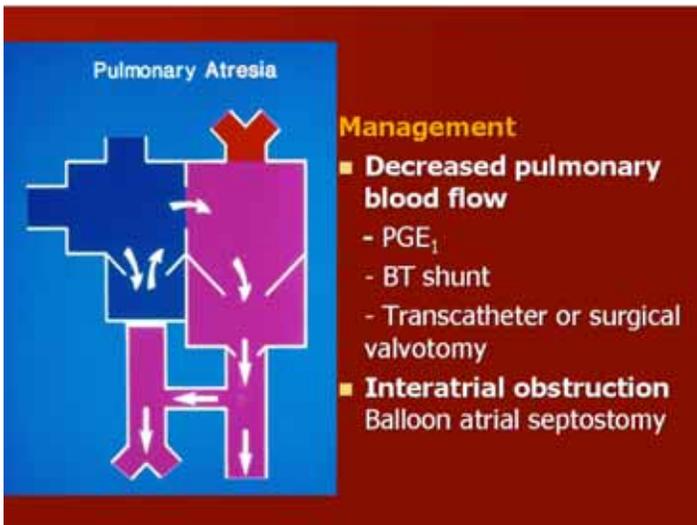


Figure 7. Box diagram of the heart showing pulmonary atresia with intact ventricular septum. Since there is no forward flow into the pulmonary artery from the right ventricle, the pulmonary flow is dependent upon the patent ductus arteriosus (PDA). Administration of PGE₁ followed by transcatheter or surgical opening of the pulmonary valve or creation of an aorto-pulmonary shunt such as Blalock-Taussig (BT) shunt is usually performed. If interatrial obstruction develops, transcatheter or rarely surgical atrial septostomy may become necessary.

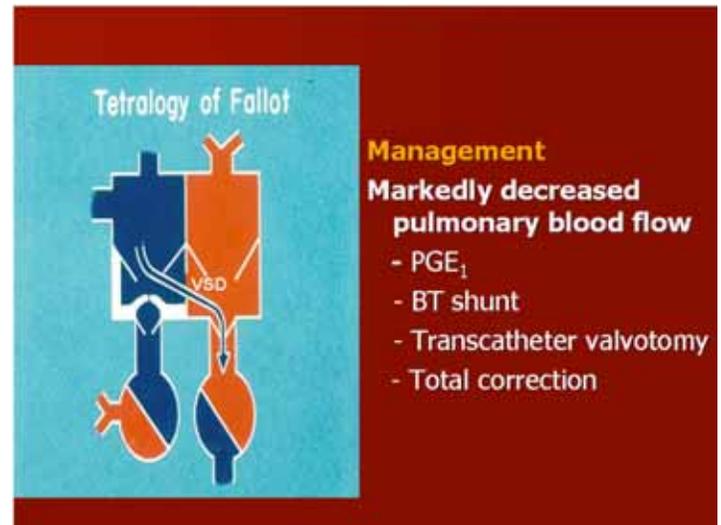


Figure 9. Box diagram of the heart showing tetralogy of Fallot demonstrating right to left shunt across the ventricular septal defect (VSD). When there is associated pulmonary atresia or a very severe pulmonary stenosis the pulmonary flow is dependent upon the patent ductus arteriosus. In some tetralogy patients neonatal intervention is needed. PGE₁ is initially administered to increase pulmonary blood flow and to improve hypoxemia. This is followed by BT shunt, balloon pulmonary valvuloplasty or total surgical correcting depending upon the anatomy.

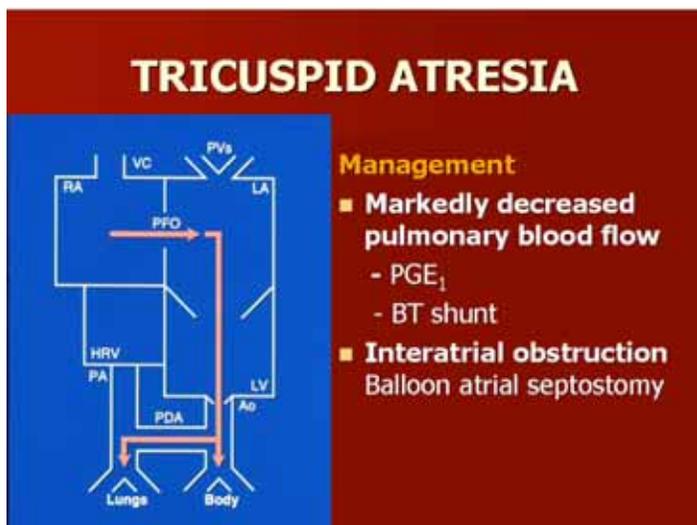


Figure 8. Box diagram of the heart showing tricuspid atresia. When there is associated pulmonary atresia or a very small ventricular septal defect, the pulmonary flow is dependent upon the patent ductus arteriosus (PDA). Administration of PGE₁ followed by an aorto-pulmonary shunt such as Blalock-Taussig (BT) shunt is usually performed. If interatrial obstruction develops, transcatheter or rarely surgical atrial septostomy may become necessary.

to open the ductus (Figure 6) which may allow inter-circulatory mixing and improve hypoxemia. However, the degree of mixing may not be adequate to wait until the arterial switch procedure (Jatene) is performed. In such situations balloon atrial septostomy (Figure 6) should be performed [7].

Right-sided Obstructive Lesions

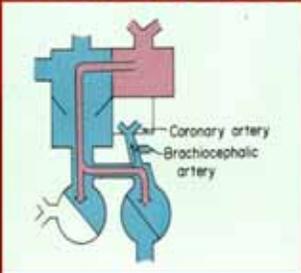
In right heart obstructive lesions such as pulmonary atresia (Figure 7), tricuspid atresia (Figure 8) and severe tetralogy of Fallot (Figure 9), as the ductus begins to close the pulmonary blood flow decreases and hypoxemia develops. Administration of PGE₁ usually restores the pulmonary flow and improves hypoxemia. A more permanent solution to augment and maintain pulmonary blood flow should follow. This includes either transcatheter or surgical opening of the pulmonary valve or creation of an aorto-pulmonary shunt such as Blalock-Taussig (BT) shunt for pulmonary atresia (Figure 8), BT shunt for tricuspid atresia (Figure 9) and BT shunt, balloon pulmonary valvuloplasty [8] or total surgical correction (depending upon the anatomy) for tetralogy of Fallot (Figure 10). A similar approach is used in other lesions associated with severe pulmonary stenosis or atresia (Table 1A). An alternative to BT shunt is implantation of ductal stents [9].

Sometimes interatrial obstruction develops in patients with pulmonary and tricuspid atresia (Figure 8 and 9), and transcatheter or surgical atrial septostomy [7] may become necessary.

Left-sided Obstruction Lesions

In HLHS and mitral or aortic atresia (Figure 10), again the ductus tends to close spontaneously soon after birth. PGE₁ infusion to open the ductus is usually effective. Decrease in pulmonary vascular resistance that follows lung expansion after birth increases flow into the lungs, thus compromising systemic flow. This can in part be reversed by lowering FIO₂ to less than room air (to increase pulmonary vasoconstriction), thus facilitating systemic perfusion through the ductus arteriosus. Maintaining some restriction at the level of patent foramen ovale will also

HYPOPLASTIC LEFT HEART SYNDROME



Coronary artery
Brachiocephalic artery

Balance Pulmonary & Systemic Circulations

Increase pulmonary vascular resistance to promote systemic flow by decreasing FIO₂ (<21%)

Inter-atrial Septal Obstruction

Balloon septostomy/PFO dilatation/stent

Slightly restrictive opening to prevent CHF

Closure of PDA

Initially PGE₁ infusion

Sometimes ductal stents may be needed

Figure 10. Box diagram of the heart showing hypoplastic left heart syndrome and possible therapeutic interventions.

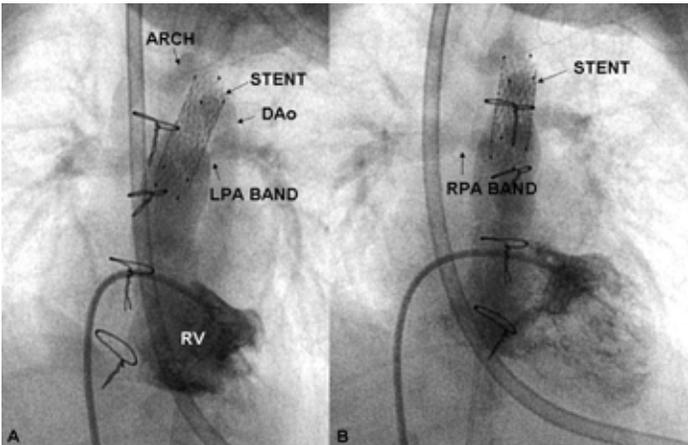


Figure 11. Selected right ventricular (RV) cine-angiographic frames demonstrating a ductal stent implanted during hybrid procedure. Note the opacification of the descending aorta (DAo) with concurrent opacification of the aortic arch (ARCH) by retrograde flow. The left pulmonary artery (LPA) band (arrow) is clearly seen. (A). Slightly different angle of cine projection is used (B) to demonstrate right pulmonary artery (RPA) band.

help prevent rapid fall in pulmonary resistance. These treatment modalities may tide over the patient until the conventional Norwood procedure is performed.

Sometimes severe inter-atrial obstruction may develop and may have to be relieved by atrial septostomy [7].

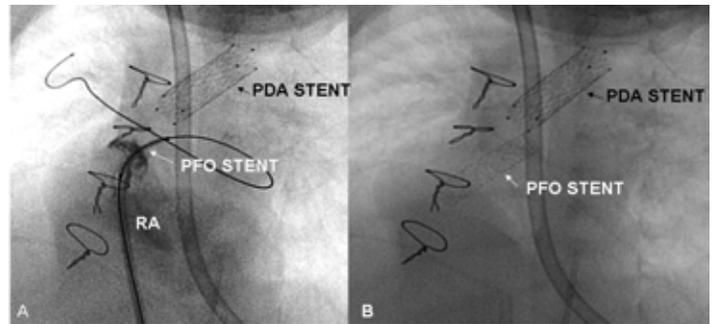


Figure 12. Selected cine frames demonstrating stent implantation (A) in the restrictive patent foramen ovale (PFO) and its position across the atrial septum (B) after deployment and removal of the balloon and guide wire.

Utilizing these principles, hybrid procedures have been developed [10] in which the pulmonary artery bands are placed surgically, constricting both branch pulmonary arteries and a stent implanted within the ductus arteriosus (Figure 11) via a sheath placed in the main pulmonary artery at the time of banding. Some of these infants may develop severe restriction of the atrial septum, requiring placement of a stent in the patent foramen ovale (Figure 12).

Coarctation of the Aorta and Interrupted Aortic Arch

In both these conditions, closure of ductus causes severe decrease in perfusion to the lower part of the body (Table IB) and can be relieved by prostaglandin infusion. This should be followed by surgical correction.

TAPVC

In some patients with TAPVC, spontaneous closure of ductus venosus and ductus arteriosus and restriction of foramen ovale may have therapeutic implication. However, in the majority of patients with infra-diaphragmatic type of TAPVC, the major issue is pulmonary venous obstruction, requiring emergent surgical correction.

VSD

In large ventricular defects, because of high pulmonary resistance at birth the murmur of VSD may not be heard at birth; the murmur becomes manifest in a few weeks after birth. The babies also do not usually go into heart failure at birth. Congestive heart failure does not become apparent until six to eight weeks of age because of delayed involution of pulmonary arterioles.

Exactly the same physiological principles may be applied to patients with other large inter-circulatory communications such as a large PDA and other complex heart defects such as: double-outlet right ventricle, double inlet left ventricle, trans-

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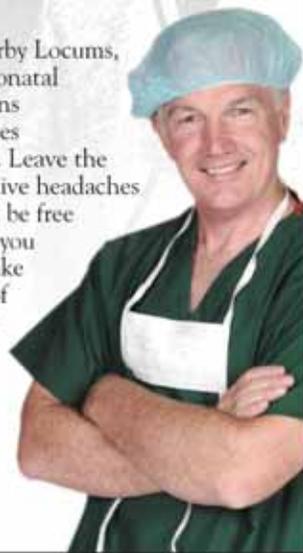
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position of the great arteries with a large VSD, tricuspid atresia with a large VSD and truncus arteriosus, all without associated pulmonary stenosis.

High pulmonary resistance secondary to hypoxemia or recurrent aspiration (tracheo-esophageal fistula) prevents development of a large left-to-right shunt and development of congestive heart failure. When a baby's lung disease gets better, sudden onset of congestive heart failure occurs.

In small VSDs, the murmur of the defect may be heard at birth since the normal fall in pulmonary vascular resistance is not interfered with.

Ebstein's Anomaly of the Tricuspid Valve

In severe forms of Ebstein's, right-to-left shunt at atrial level is increased by high neonatal pulmonary vascular resistance. As the pulmonary vascular resistance falls, the degree of right-to-left atrial may diminish, improving the hypoxemia.

In some patients, administration of PGE₁ is helpful in augmenting pulmonary flow, especially in association with pulmonary valve stenosis or atresia.

SUMMARY AND CONCLUSIONS

Postnatal circulatory changes markedly influence the clinical presentation and clinical course of the neonate with congenital heart defects. Closure of the ductus arteriosus adversely affects:

1. The systemic perfusion in HLHS and aortic arch obstructions,
2. Pulmonary blood flow in cardiac defects with severe pulmonary stenosis or atresia and
3. Inter-circulatory mixing in TGA.

Prostaglandin E₁ infusion is effective in reopening the ductus or maintaining its patency. Longer lasting solutions include BT shunts and ductal stents.

Spontaneous closure or restriction of foramen ovale adversely affects:

1. Right-to-left shunting in right heart obstructive lesions and TAPVC,
2. Left-to-right shunting in left heart obstructive lesions and
3. Inter-circulatory mixing in TGA.

When the foramen ovale is restrictive, transcatheter or surgical septostomy is beneficial.

Pulmonary vascular resistance plays a critical role in patients with large inter-circulatory communications.

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ANNUAL NEONATAL PRACTICES CONFERENCE

SCHEDULED FOR CHICAGO ON JUNE 20-21, 2008

The Fifth Annual Evidence vs Experience in Neonatal Practices® CME conference returns to Chicago June 20-21, 2008

This popular meeting, to be held at the Renaissance Chicago Hotel, will address the timeliest topics in the management of neonates. A faculty of 13 thought leaders in neonatology will describe recent developments in this fast-evolving field, including state-of-the-art treatment options that are improving outcomes for preterm infants.

The two-day conference will focus on a wide range of subjects in areas such as nutrition, surfactants, hemodynamics and ventilation.

Presentations will include:

- Surfactant Treatment for ARDS
- GERD and Gut Dysmotility in Preterm Infants
- Late Preterm Infants: Unique Problems
- Low Systemic Blood Flow and Neurodevelopmental Outcomes
- To Tube or Not to Tube Babies with RDS
- Neonatal Ventilators: How Do They Differ?



Kris Sekar, MD, FAAP

In their in-depth lectures, speakers will describe the use of surfactant pre-extubation, developments in synthetic vs. natural surfactants, and prebiotics/probiotics. Research into inhaled nitric oxide, relative adrenal insufficiency, and hypotension in preterm infants will also be discussed. Nicholas J. Evans, DM, MRCPCH, of the University of Sydney and Royal Prince Alfred Hospital in Australia will deliver the keynote address on "Low Systemic Blood Flow and Neurodevelopmental Outcome."

Kris Sekar, MD, FAAP, of the University of Oklahoma Health Sciences Center and

The Children's Hospital in Oklahoma City, will serve as Chair of this thought-provoking conference. Other members of the Organizing Committee are Jatinder Bhatia, MBBS (Medical College of Georgia), Rangasamy Ramanathan, MD (Keck School of Medicine of USC), and Istvan Seri, MD, PhD (also of the Keck School of Medicine). Faculty members include neonatologists from the United States, Europe and Australia.

The target audience for this important conference includes physicians, nurse practitioners, nurses and other clinicians caring for preterm infants. The faculty will encourage the audience to participate fully in the program, interacting with not only the speakers but also with one another, and offering their own insights and perspectives into recent research and patient care in neonatal medicine. An Audience Response System will be used to help determine what participants have learned.

This annual conference will be co-sponsored by the Annenberg Center for Health Sciences at Eisenhower and the Keck School of Medicine of USC and supported by an independent educational grant from DEY, L.P.

To register online for **Evidence vs. Experience in Neonatal Practices®**, or for additional conference information, program updates, and accreditation information, visit the conference. Website: www.5StarMedEd.org/neonatal.

Questions about this event can be addressed to the Annenberg Center for Health Sciences at Eisenhower by calling Nina Pratt at 800-321-3690 (toll-free) or 760-773-4500 (8 a.m. to 5 p.m. Pacific time).

Introduction

In 2004, there were more than half a million preterm births in the US (about 12.5% of live births). The problems encountered by a premature infant are related to the immaturity of the organ systems. The infant requires specialized care until his or her organ systems have developed enough to sustain life without specialized support. Depending on the extent of prematurity, this may take weeks to months. This meeting will continue the examination of newly developing treatment options for these problems, while reviewing current evidence for treatment protocols. International thought leaders in the field will help clarify desired and efficacious treatment options.

Learning Objectives

Upon completion of this activity, participants should be better able to:

1. Apply critical nutritional care to their preterm infants
2. Incorporate current knowledge of surfactants into practice
3. Utilize appropriate interventions for hemodynamic problems
4. Provide appropriate ventilator care to preterm infants

Accreditation and Certification

The Annenberg Center for Health Sciences at Eisenhower is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

The Annenberg Center designates this educational activity for a maximum of 11.25 AMA PRA Category 1 Credits™. Physicians should only claim credit commensurate with the extent of their participation in the activity.

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1. How many years has it been since your Fellowship? _____ Years
2. Are you Board Certified in Neonatology (Place an "X" in the appropriate box)?
 a Yes b No c In the process of becoming B/C
3. In terms of your compensation, what is your base salary? \$ _____
- 3A. How much is your bonus? \$ _____
4. What type of position do you hold (check all that apply)? a Academic b Clinical
5. What type of healthcare benefits do you receive (check all that apply)?
 a Healthcare c Dental
 b Vision d None
6. If you receive healthcare, is it (check one)? a Individual Only b Family
- 7A. Is your healthcare (check one) a Fully Paid by Employer b Partially Paid by Employer
- 7B. How many vacation weeks per year do you get? _____ Weeks
8. Do you get a CME allowance (check one)? a Yes b No If yes, how much? \$ _____
9. Is your Malpractice Insurance paid by your employer (check one)? a Yes b No c Partially paid
- 10A. What type of Retirement Plan do you have through your place of employment (check one)?
 a 401K b 401B c Other d None
- 10B. What is the approximate value of your employer's participation in your retirement? \$ _____
11. How many years have you been at your current place of employment? _____ Years
12. For demographic analysis only, what is your 5-digit residential (home) Zip or postal code? _____
13. **Optional:** Do you have any additional comments you would like to share: _____

Thank you,

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Medical News, Products and Information

Code Medication Calculator Compliments of Doug Norwood, Respiratory Care Supervisor, Rockdale Medical Center

Neonatology Today, is making available a drug calculation program to print bedside Code Cards in the NICU.

This basic drug calculation program is provided by Doug Norwood, Respiratory Care Supervisor in the Birth Place at Rockdale Medical Center in Conyers, Georgia. Dosages are calculated based on the infant's weight. Patient name and ID number may be stored and selected from a pull-down menu. The author wishes to share the program with anyone who would like to use it. Please note that the epinephrine dose is calculated at 0.1 ml/kg. Current NRP guidelines recommend 0.1-0.3/kg ml IV or 0.3-1.0/kg ml ETT.

Feel free to modify any part of the program as needed for your own use. Please, always verify any information on it before you use for patient care.

Rockdale Medical Center is a suburban Atlanta hospital that houses a 16 bed level III NICU.

Readers who wish a copy of the program, may send an email to: CodeCard@Neonate.biz. Please include your name and title. Readers may also go the following link, to download the program directly:
<http://digbig.com/4wmpk>

Neonatologists and Pediatricians Highlight Progress and Barriers to Preemie Care

Newswise — A recent survey of 202 neonatologists and pediatricians, which examined current attitudes and practices when caring for the specialized health needs of preterm infants, revealed that

most respondents (70%) feel the United States' healthcare system does not place enough emphasis on or dedicate enough resources to preventive healthcare for preemies. The survey was sponsored by MedImmune, Inc.

The incidence of preterm birth, when infants are born at less than 36 weeks gestation, has increased steadily in the United States since the mid-1990s. Because these babies lack the usual complement of antibodies, which are supplied by the mother to babies in late gestation, preterm babies are at high risk of getting a host of infectious diseases, including respiratory syncytial virus (RSV), the leading cause of infant respiratory hospitalization in the United States. That risk can be even greater among infants that have an array of complex health problems including immune deficiencies, chronic lung disease, congenital heart disease and neurological disorders.

"This survey reminds us that, while progress in preemie healthcare has been made, more still needs to be done to ensure that every preemie, regardless of his or her circumstances, receives the care he or she deserves," said Richard J. Martin, MD, division chief of neonatology, Rainbow Babies and Children's Hospital, Cleveland, Ohio.

Additional key survey findings shed light on reasons why premature infants may not receive the specialized care they require:

- Preemie care practices differ among doctors with varying levels of experience. More than half (53%) of pediatricians with 10 years of experience or less relied on parents to find out if a patient was born prematurely, compared with just 14% of pediatricians with 21 plus years of experience. The more experienced pediatricians favored the hospital discharge summary (43%) or communication with the

child's neonatologist for this information (36%).

- Twenty-one percent of neonatologists with more than 10 years of experience said providing parents with a copy of their child's discharge plan is the most important step when discharging a preemie from the hospital. Only three percent of neonatologists with fewer years of experience named this as the most important step.
- Most pediatricians (56%) with 10 years of experience or less said they stop working with a preemie's neonatologist immediately following discharge, whereas most pediatricians (54%) with 21 plus years of experience keep working with the neonatologist until their patient is at least three months old.
- Late-preterm infants (defined as 34-to-35 weeks gestational age for the purpose of the survey) may not be on their doctors' "radars" because of misconceptions about the risks these babies face.
- Fifty-eight percent of 34-to-35 week infants are perceived, by their surveyed doctors, as healthy (not at high-risk), even though they are premature and at high risk for RSV disease.
- Doctors agree that there are a number of reimbursement and managed-care barriers to effective preemie care.
- Most physicians (70%) feel that the U.S. healthcare system does not dedicate enough emphasis and resources to preventive healthcare for premature infants. Eighty-four percent of these physicians say they are willing to personally advocate for more preventive health services for preemies.
- Most pediatricians (69%) say their office staff spends more time on reimbursement for premature infants than for full-term babies. Seventy-seven percent say they spend more time on reimbursement for premature babies at high risk for serious RSV than for preemies who are not at high risk for serious RSV.



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About the Survey

HCD Research, an independent research company, surveyed a random sample of 202 neonatologists and pediatricians from September 5 to 25, 2007. To qualify, respondents had to have spent at least 50 % of their time in a clinical setting, with neonatologists treating at least three preemies per month and pediatricians treating at least three preemies in the past four months. Respondents with an existing financial relationship with an advertising agency, the U.S. Food and Drug Administration or a market research firm were excluded. No incentive was offered in exchange for respondents' participation.

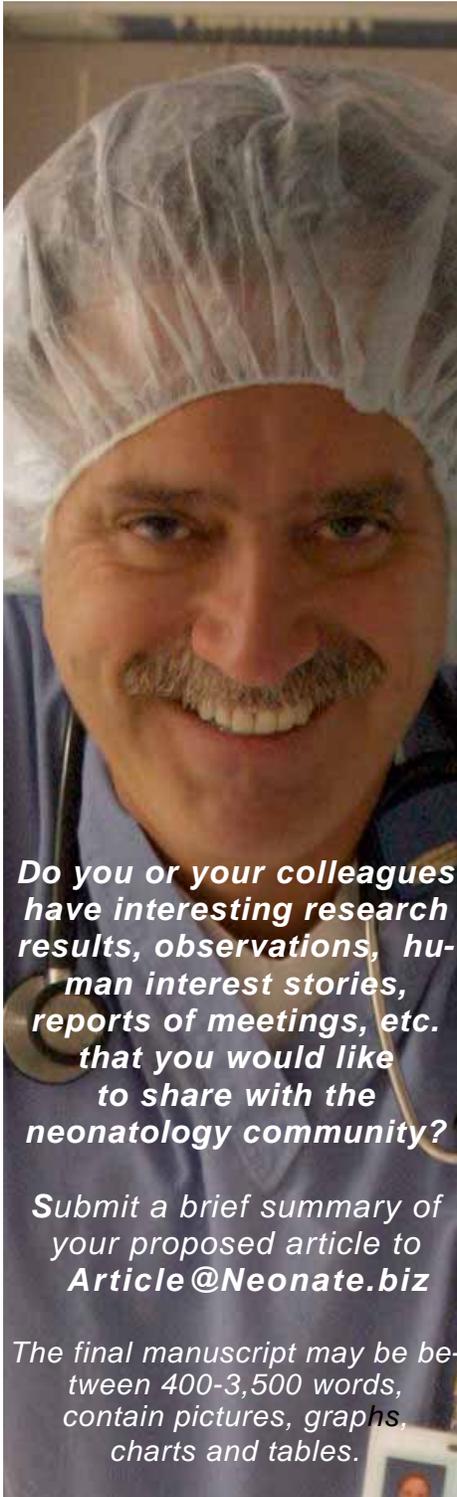
Ninety-seven neonatologists participated in the survey. Thirty-two neonatologists had 10 years of experience or less, 37 neonatologists had between 11 and 20 years of experience, and 28 neonatologists had at least 21 years of experience. A total of 105 pediatricians participated in the survey. Twelve were pediatric pulmonologists and 15 were pediatric cardiologists. Thirty-two pediatricians had 10 years of experience or less, 45 pediatricians had between 11 and 20 years of experience, and 28 pediatricians had at least 21 years of experience.

About RSV

Each year, up to 125,000 infants in the United States are hospitalized with severe RSV infections, the leading cause of infant hospitalization in the U.S. Approximately one-half of all infants are infected with RSV during the first year of life, and nearly all children have been infected at least once by the time they reach their second birthday. In the United States, RSV causes up to 1.7 million physician office visits; 400,000 emergency room visits and more than 230,000 hospital outpatient emergency room visits per year. RSV is the most common respiratory infection in infancy or childhood. Children born prematurely as well as those with chronic lung disease or congenital heart disease are at highest risk for severe disease and hospitalization due to RSV. In addition, some 25-40% of infants in the first year of life infected with RSV develop lower respiratory tract infections (such as bronchiolitis or pneumonia) which can additionally compromise the hearts and lungs of these high-risk infants. The virus may also cause severe illness in populations such as the elderly, those with underlying respiratory or cardiac disease, and those with compromised immune systems (e.g., bone marrow transplant patients).

About MedImmune

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