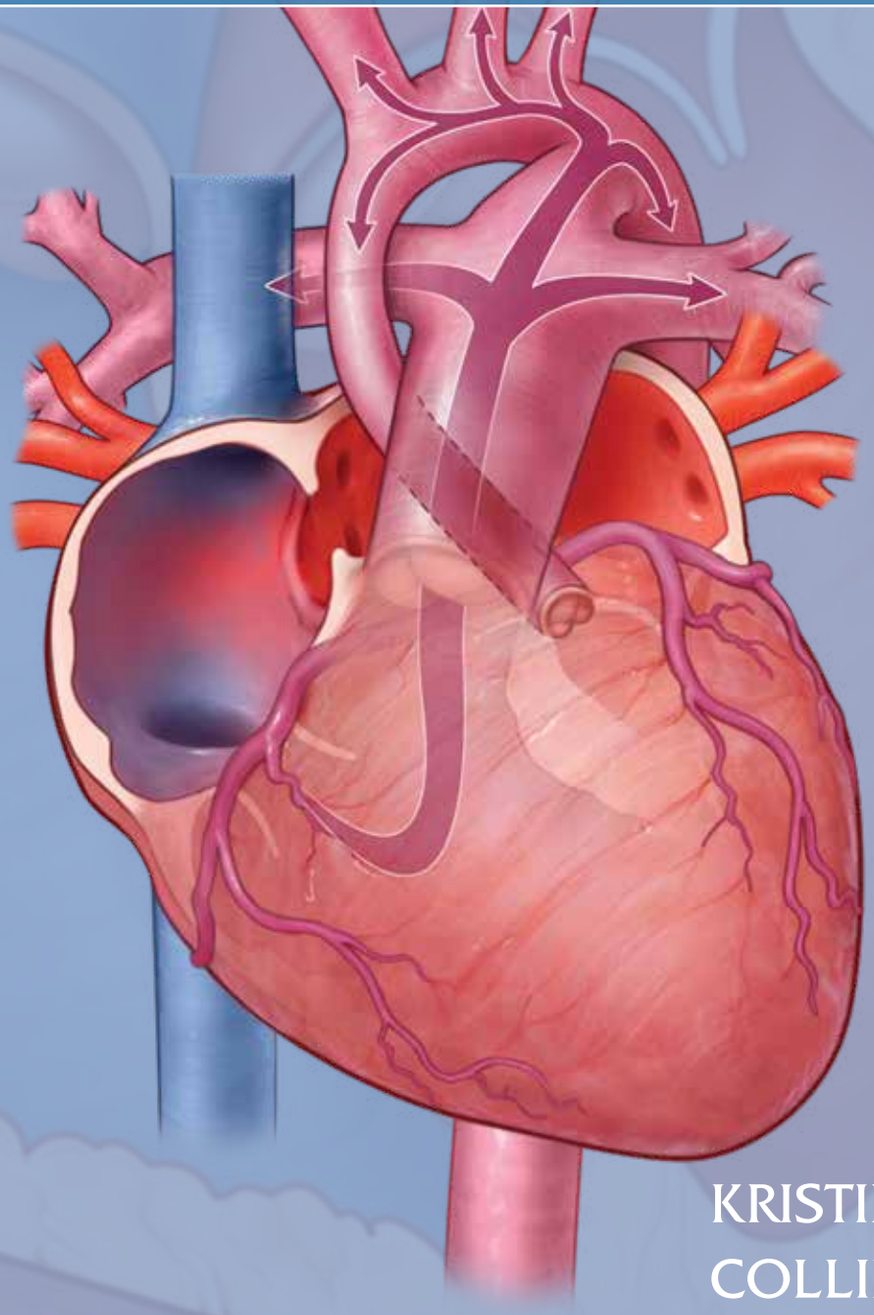
SECOND EDITION

S.T.A.B.L.E.® – Cardiac Module

Recognition and Stabilization of Neonates with Severe CHD



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S.T.A.B.L.E.® – Cardiac Module

Recognition and Stabilization of Neonates with Severe CHD

Second Edition

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Cardiac Module *2nd edition*

THE S.T.A.B.L.E.® Program

Sugar, Temperature, Airway, Blood Pressure, Lab Work, and Emotional Support

Introduction

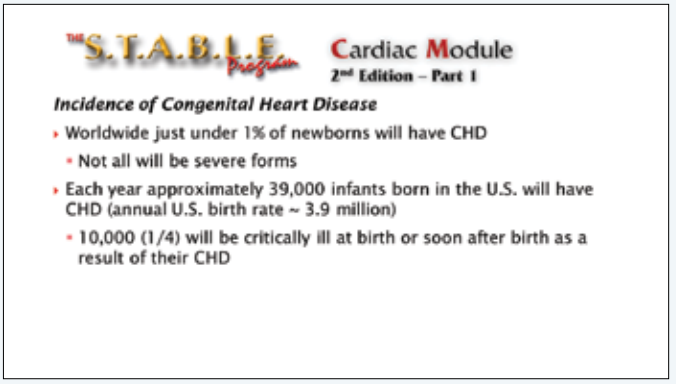
The S.T.A.B.L.E. Program is an educational program designed for all maternal/child healthcare professionals (nurses, physicians, respiratory therapists, and prehospital providers) to address the pretransport stabilization and postresuscitation care of sick neonates. All information in The S.T.A.B.L.E. Program applies to neonates with cardiac conditions. This resource includes additional guidelines for neonates with severe congenital heart disease (CHD).

Part 1 will focus on the history and physical examination of neonates with suspected CHD; Part 2 will focus on the presentation and stabilization of infants with severe structural heart disease; and Part 3 will discuss The S.T.A.B.L.E. Program postresuscitation stabilization modules — Sugar, Temperature, Airway, Blood pressure, Lab work, and Emotional support — and adaptations that may be necessary when caring for neonates with CHD. An Appendix is included to explain the palliative and surgical options to address the cardiac defects described in this book.

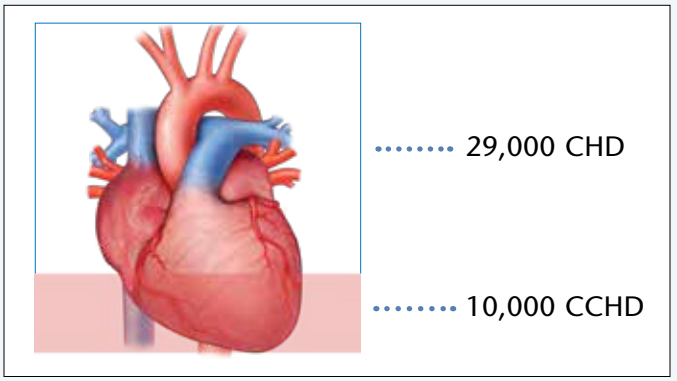
A subscription to a companion PowerPoint® presentation is available for purchase from www.stableprogram.org. The slides on each page of this manual are from that presentation and are included to help associate the book content with the PowerPoint slides.

Incidence of Congenital Heart Disease (CHD)

Worldwide, approximately 8 babies per 1,000 live births, or just under 1% of all babies born, will have some form of CHD.[1-3] Globally, this rate equates to 1.35 million babies born each year with CHD.[3] In the United States, with an annual birth rate just under 4 million,[4] 39,000 infants will have CHD and approximately 25% will have the most severe forms, that are known as critical congenital heart defects (CCHDs).[5,6] These infants will require care by neonatal and cardiac experts in the first days to weeks of life. Prompt, effective care of neonates with CHD can reduce secondary organ damage, improve short- and long-term outcomes, and reduce mortality.[7]

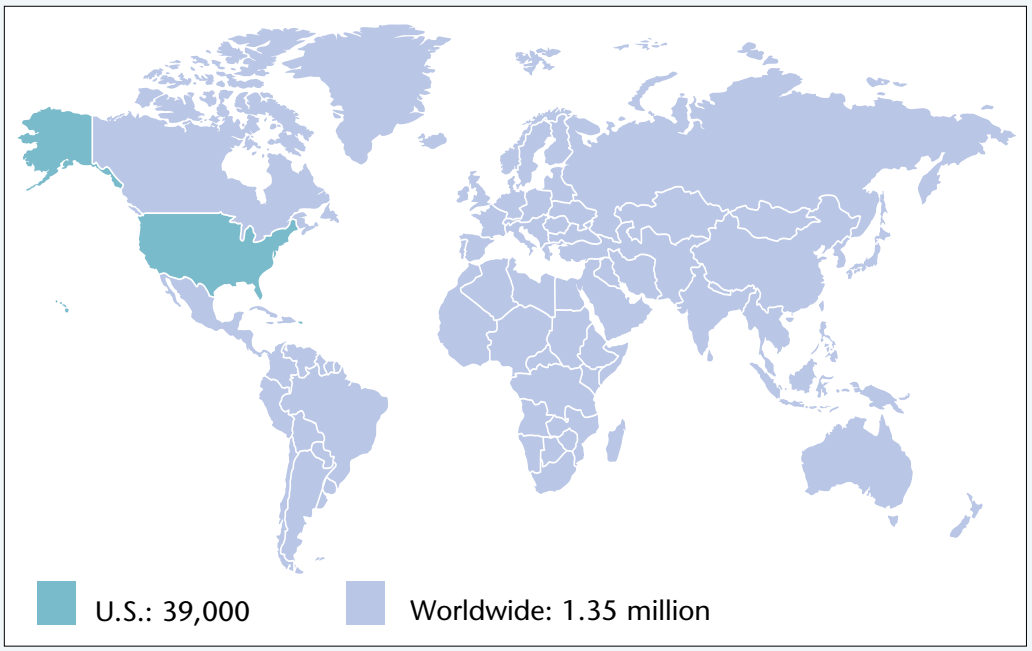


U.S., Annual Rate of CHD



39,000 infants will have CHD and approximately 25% will have the most severe critical congenital heart defects (CCHDs).

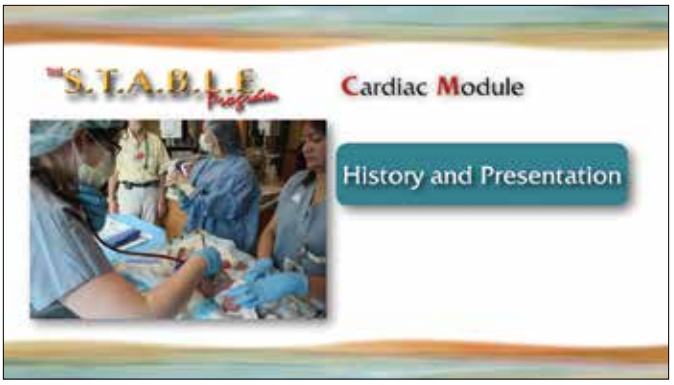
Infants Born with CHD Per Year



Worldwide, approximately 8 babies per 1,000 live births, or just under 1% of all babies born, will have some form of CHD.

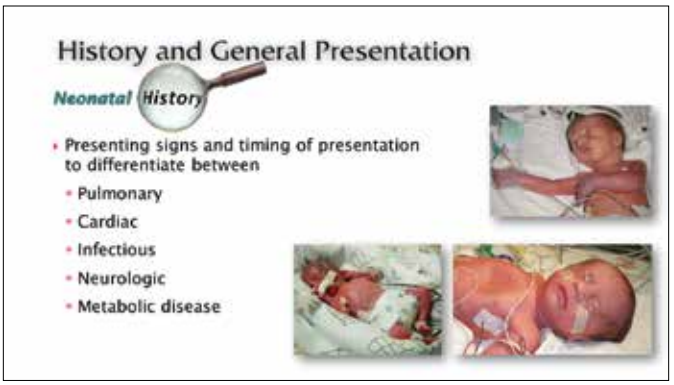
History and Patient Presentation

When a neonate presents with cyanosis, respiratory distress, and/or shock, the process of determining whether signs and symptoms are due to pulmonary, cardiac, infectious, neurologic, or other causes begins.[2] Information valuable to differential diagnosis includes evaluation of the presenting signs and timing of presentation, as well as family, pregnancy, and maternal medical history.



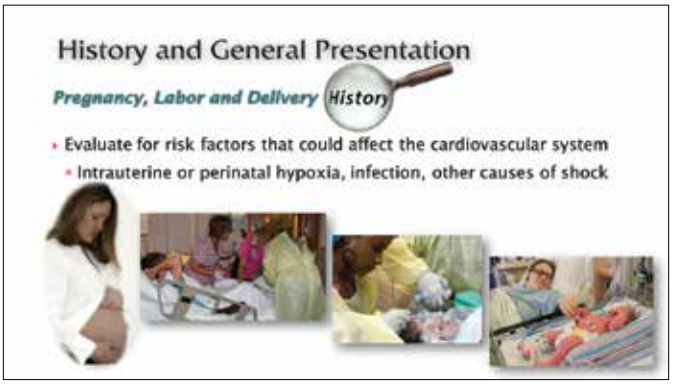
Neonatal History[2,8]

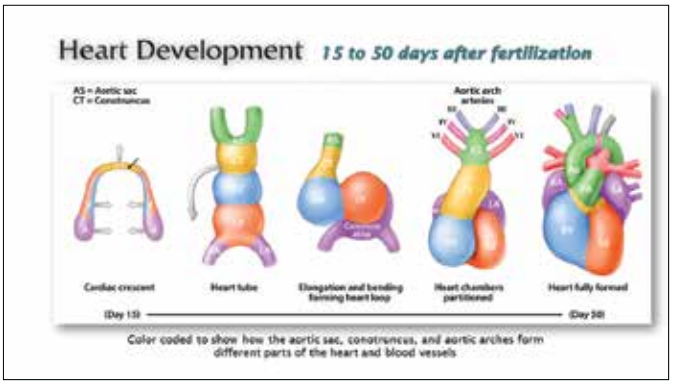
A baby who presents with severe cyanosis, congestive heart failure (CHF), and/or cardiovascular collapse within the first hours, days, or weeks of life must be evaluated for CHD. The timing and history of symptom onset are very important. A term infant who has been well the first few days of life, but who becomes tachypneic, feeds poorly, sleeps more than normal, and/or displays signs of shock should prompt consideration of left-sided obstructive CHD with a closing ductus arteriosus. The infant with early onset respiratory distress following a difficult birth or born to a group B streptococcus positive mother is more likely suffering from pulmonary disease or the effects of sepsis. A preterm infant is more likely to have pulmonary causes of respiratory distress, although cardiac disease does occur in both preterm and term infants.



Pregnancy, Labor, and Delivery History

Pregnancy, labor, and delivery complications should be carefully evaluated for risk factors that could affect the cardiovascular system or conditions that mimic CHD.[2] For example, intrauterine and perinatal hypoxia are risk factors for development of myocardial dysfunction, as well as persistent pulmonary hypertension of the newborn (PPHN). Risk factors for sepsis should be identified, as septic





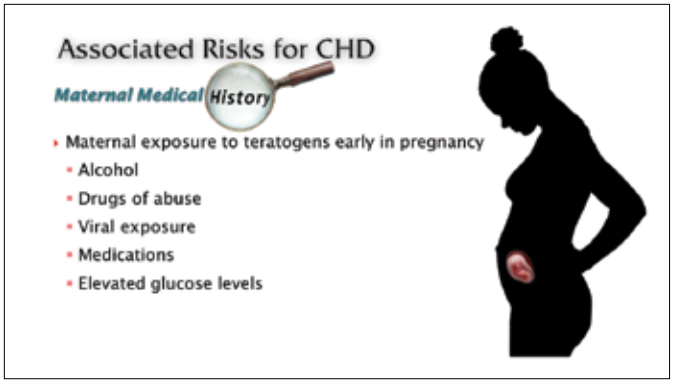
neonates may present in shock similar to those with left heart obstructive lesions. Other causes of shock, including maternal/fetal hemorrhage, should be identified and treated.

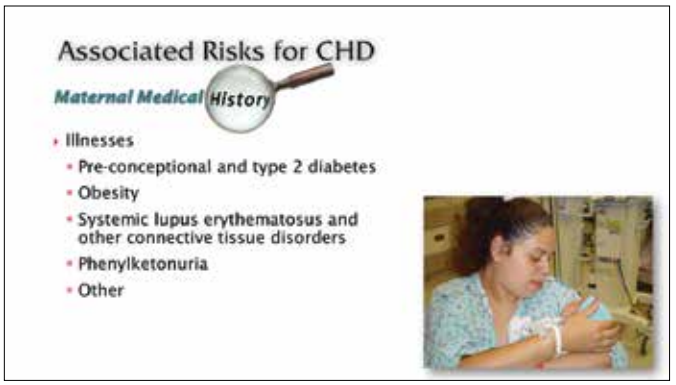
Maternal Medical History[1,9-24]

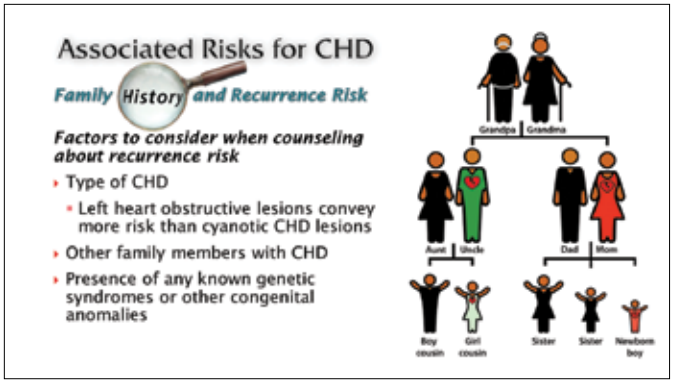
The heart develops very early in pregnancy. Between the 15th and 50th day after fertilization, the four-chamber heart has formed. After that, the heart continues to grow and mature.[25] Exposure to teratogens early in pregnancy, including alcohol, drugs of abuse, viruses, certain medications, and even elevated glucose levels, increases the risk for CHD. Maternal illnesses that may result in fetal anomalies including congenital heart defects, include diabetes, obesity, connective tissue disorders, and phenylketonuria (PKU). However, in the case of diabetes and PKU, it should be noted that strict preconception and intrapartum metabolic control can decrease or even eliminate risk to the developing fetus. Table 1.1 on page 6 summarizes maternal exposures, conditions, and illnesses that increase risk for CHD.

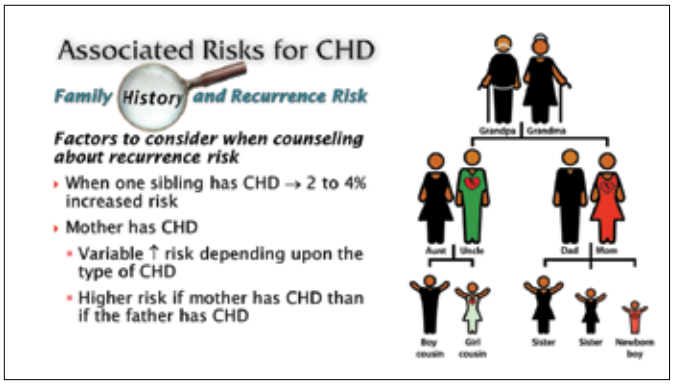
Recurrence Risk[18,26-32]

When a sibling or parent has a congenital heart defect, the question of recurrence risk arises. Family counseling involves consideration of many factors, including the type of CHD, other family members with CHD, and presence of genetic syndromes or other congenital anomalies.[33] Generally speaking, when one sibling has a congenital heart defect, the recurrence risk for other siblings ranges between 2% and approximately 4%. However, there is some evidence that left heart obstructive lesions, such as coarctation of the aorta, hypoplastic left heart syndrome, and aortic stenosis or atresia, are associated with an even higher risk of recurrence than other forms of CHD. Although not clearly understood why, when the mother has a congenital heart defect, the recurrence risk for CHD is higher in her offspring than when the father has CHD.









Patient Assessment

Oxygen Saturation*

Oxygen (O_2) is transported to the tissues bound to Hb. O_2 saturation is the percentage of Hb bound to O_2. When infants are cyanotic, oxygen is given to improve arterial O_2 saturation.

Cyanotic CHD should be suspected when there is a minimal increase in O_2 saturation or arterial PO_2 when the infant breathes 100% O_2.

Infants presenting with shock should be assessed for presence of a **left heart obstructive lesion** such as critical COA. In these infants, the cardiac defect is severe despite the responsiveness of the O_2 saturation and PaO_2 to O_2 administration.

Figure 1.6. Illustration of a right-to-left shunt through the PDA secondary to increased PVR and vasoconstricted pulmonary arterioles. Because of increased PVR, blood shunts via the pathway of least resistance, R to L through the PDA, into the aorta. Typically, significant R to L ductal shunting leads to a saturation in the right hand at least 5 to 10% higher than saturation in either foot. If shunting is primarily R to L at the patent foramen ovale (PFO), then there may be no appreciable difference in preductal and postductal O_2 saturations.

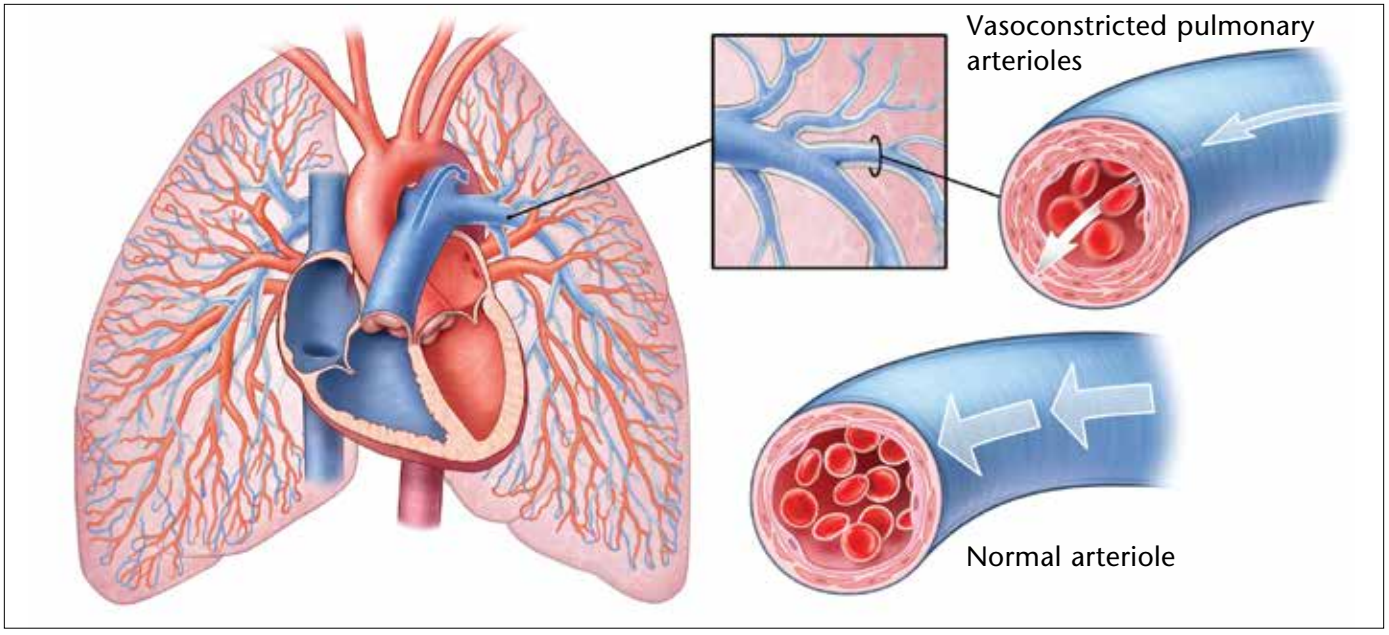
Preductal and Postductal O_2 Saturation

At times, it is of diagnostic value to evaluate both the preductal and postductal O_2 saturation to determine whether there may be a right-to-left shunt at the DA, as may occur when there is a noncardiac clinical problem called persistent pulmonary hypertension of the newborn (PPHN; see Figure 1.6). Characterized by persistently elevated pulmonary vascular resistance (PVR), the pathophysiology of PPHN usually falls into one of three categories, although overlap between categories is possible:

1) Pulmonary arteriolar vasoconstriction in association with lung parenchymal disease such as meconium aspiration syndrome, pneumonia, respiratory distress syndrome, or bronchopulmonary dysplasia;

2) Reduced pulmonary vascular bed with lung hypoplasia, such as seen with congenital diaphragmatic hernia; or,

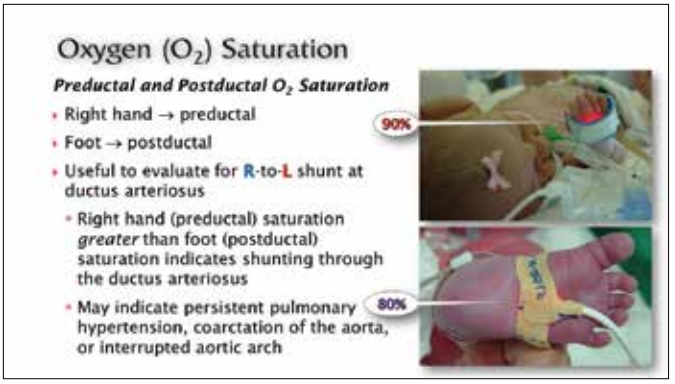
3) When the lung parenchyma is normal, but there is remodeled, muscularized pulmonary vasculature (also called idiopathic).

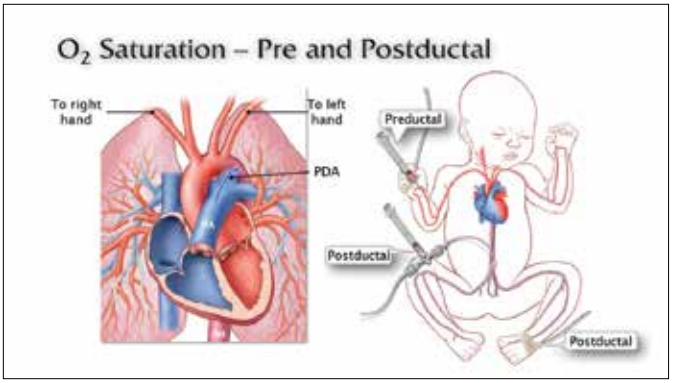


* See the S.T.A.B.L.E. – Cardiac Module PowerPoint presentation for a video on O_2 saturation and normal heart anatomy.

To monitor preductal and postductal saturation, attach a pulse oximeter probe to the right hand and to either foot. Allow the reading to stabilize and then record your findings.

In addition to monitoring preductal and postductal O_2 saturations in ill-appearing infants, pulse oximetry screening to detect critical congenital heart defects (CCHDs) is being performed in many hospitals worldwide.[111-116] Otherwise healthy appearing infants are screened before discharge to home with the purpose of detecting l*ower than normal O_2 saturation values* secondary to previously undiagnosed CCHDs. For information specific to CCHD screening, see page 162.





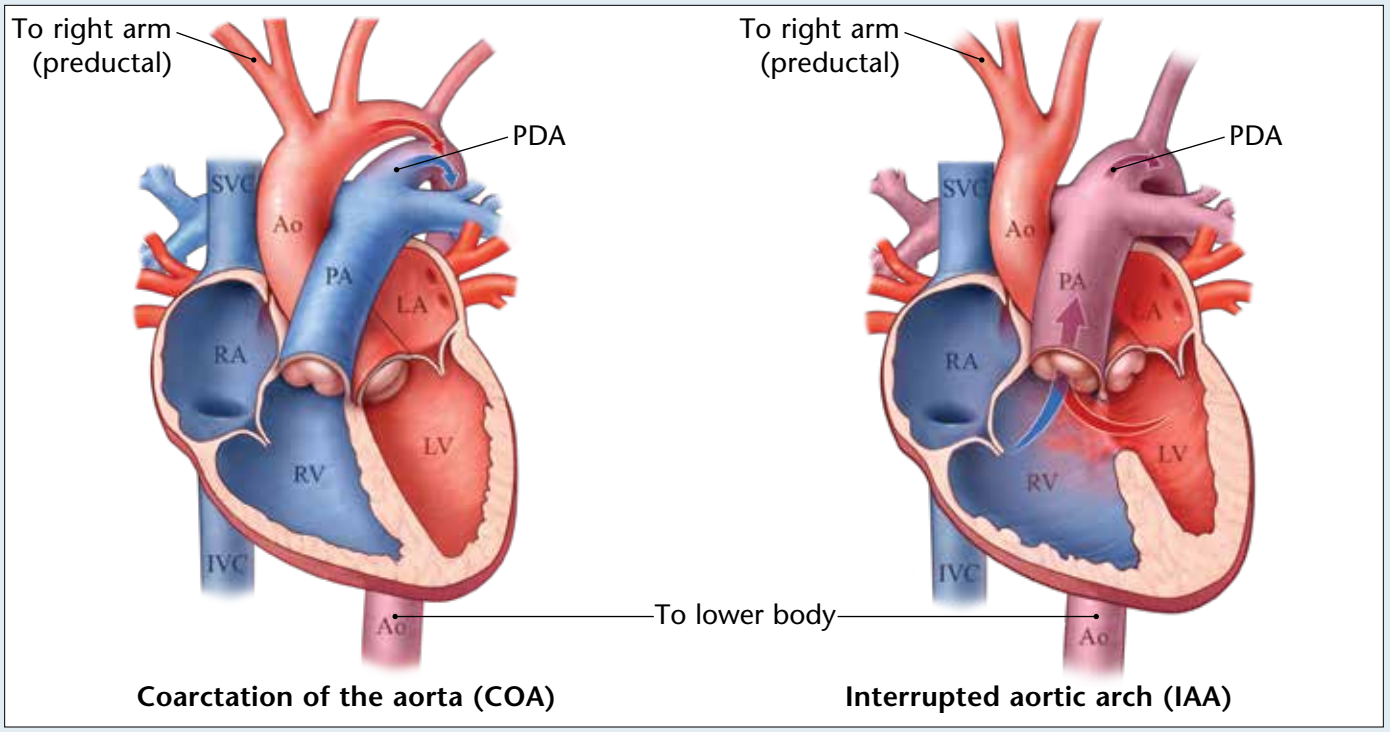
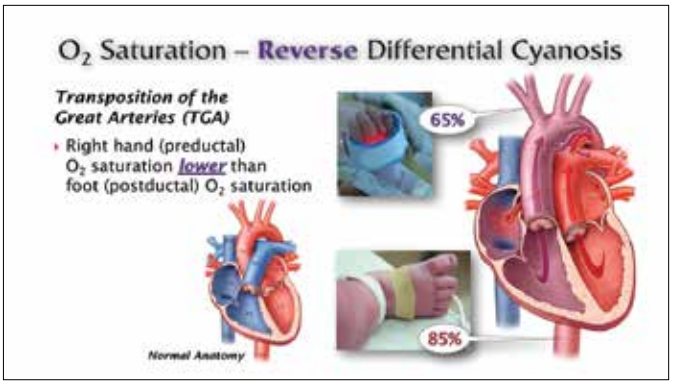


Figure 1.7. Differential cyanosis (higher O_2 saturation in the right hand, lower O_2 saturation in the foot), that may be observed with COA and IAA. While the PDA is open, desaturated blue blood can shunt from the pulmonary artery, through the PDA, and into the aorta. Pulse oximetry screening may detect a difference between the right hand (preductal) and the foot saturation (postductal).

Reverse Differential Cyanosis

A higher O_2 saturation in the right hand compared to either foot indicates a right-to-left ductal shunt as just described. *Reverse differential cyanosis*, where there is a lower O_2 saturation in the right hand compared to the foot, suggests transposition of the great arteries (TGA) with pulmonary hypertension (see Figure 1.8), or TGA with critical COA/IAA. In TGA, the aorta originates from the right ventricle and the pulmonary artery originates from the left ventricle. When infants have TGA, continuous assessment of preductal O_2 saturation is critically important because the O_2 saturation in the right hand is the same O_2 saturation of the blood perfusing the brain.



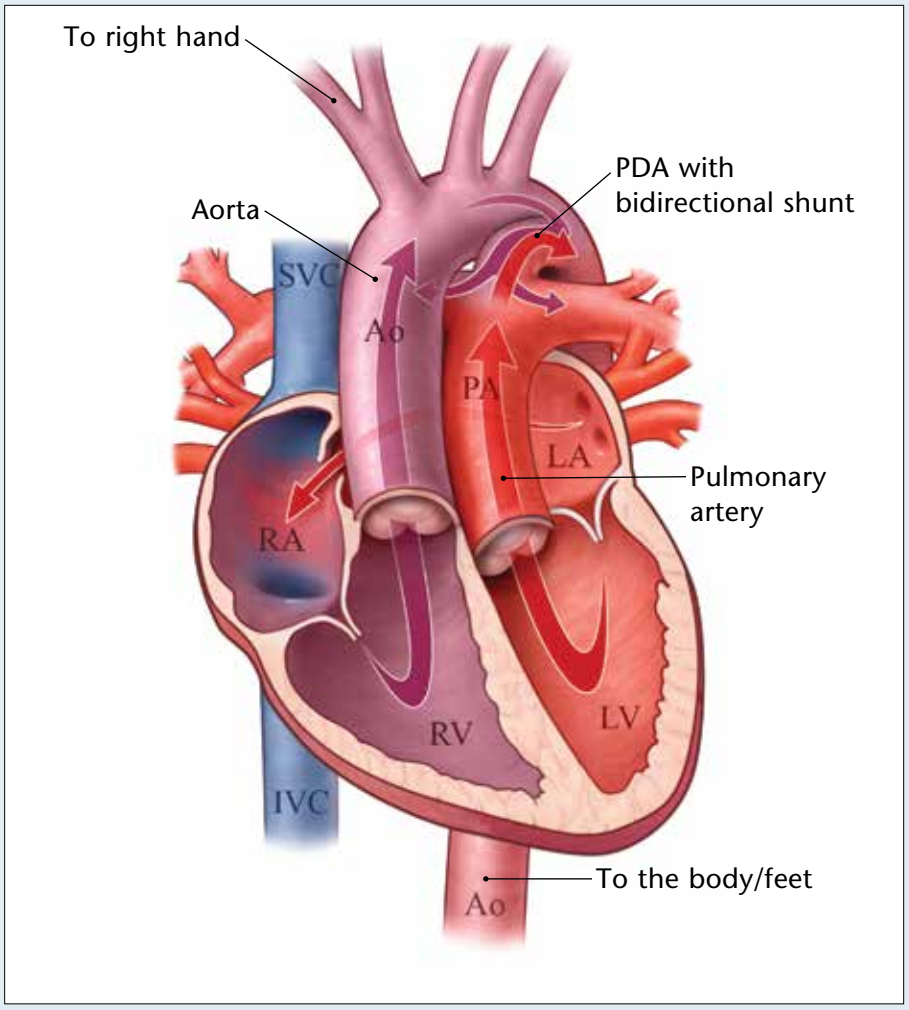


Figure 1.8. Reverse differential cyanosis secondary to Transposition of the Great Arteries (TGA).

The pathway of blood flow in this illustration is as follows: Blue, deoxygenated blood returning to the right side of the heart is pumped from the right ventricle into the aorta, to the brachiocephalic artery that branches into the right subclavian artery (to the right hand) and the right carotid artery (to the brain). Red, oxygenated blood returning from the lungs to the left side of the heart is pumped from the left ventricle into the pulmonary artery, through the PDA and into the aorta (to the body). In the presence of pulmonary hypertension, which is not uncommon in TGA, blood is preferentially shunted through the PDA into the aorta. Thus, the color of the blood in the right hand is more desaturated than the color of the blood in the distal aorta (i.e., reverse differential cyanosis). Infants with TGA may also have critical COA or IAA (not shown), which has the same effect on blood flow as pulmonary hypertension.

Patient Assessment

Vital Signs – Respiratory Rate and Effort*

Normal

The respiratory rate is 30 to 60 breaths per minute.

Breathing is not labored.

Symmetric breath sounds are heard.

Abnormal

Tachypnea

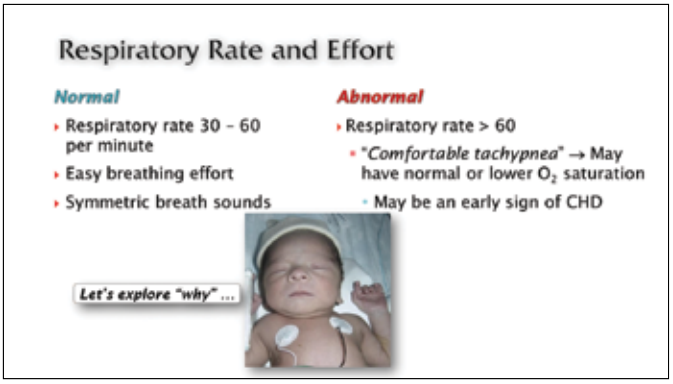
A respiratory rate > 60 breaths per minute, without signs of respiratory distress (comfortably tachypneic), may be one of the first signs of cardiac disease.

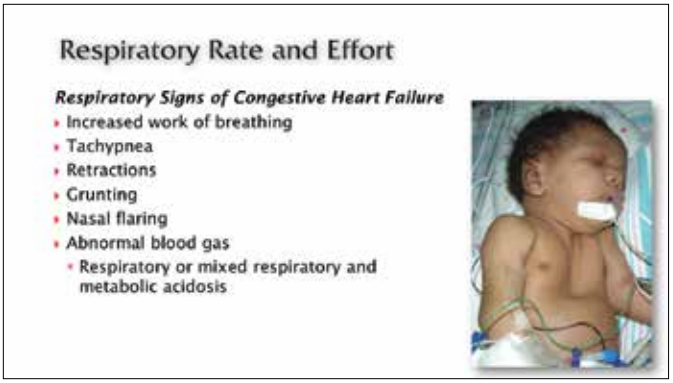
Respiratory Signs of CHF[118] may include:

- **Increased work of breathing**
- **Tachypnea**
- **Retractions**
- **Grunting**
- **Nasal flaring**
- **Abnormal blood gas (respiratory acidosis or mixed respiratory and metabolic acidosis)**

Respiratory Rate < 30 (Bradypnea)

A slow respiratory rate, in association with labored breathing and a declining neurologic status may signal that the infant is becoming exhausted. Bradypnea (respiratory rate < 30 per minute) may also represent a decrease in central respiratory drive because of hypoxic ischemic encephalopathy, prematurity, medications that depress respiratory drive (e.g. opioids), or other illnesses.





Gasping is an ominous sign of impending cardiorespiratory arrest. This extremely critical state should be treated the same as though the infant is apneic. Immediately provide positive pressure ventilation. If the infant's heart rate is low and not rising, establish an advanced airway via tracheal intubation or insertion of a laryngeal mask airway.

*See the S.T.A.B.L.E. – Cardiac Module PowerPoint presentation for two videos that explain Homeostatic Control of Respiration and Congestive Heart Failure.

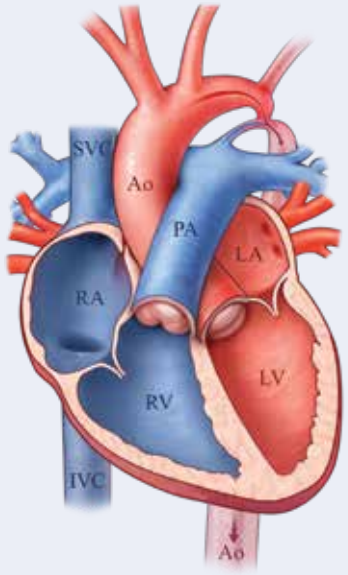
Part 2 of the S.T.A.B.L.E. – Cardiac Module is presented as follows:

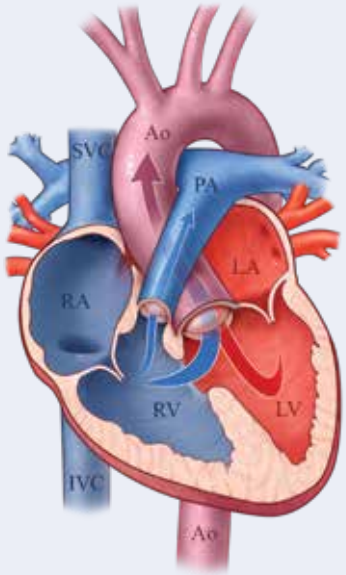
1. **Left-sided obstructive forms of CHD that are ductal dependent for systemic blood flow:**
   - Coarctation of the aorta
   - Interrupted aortic arch
   - Aortic valve stenosis
   - Hypoplastic left heart syndrome

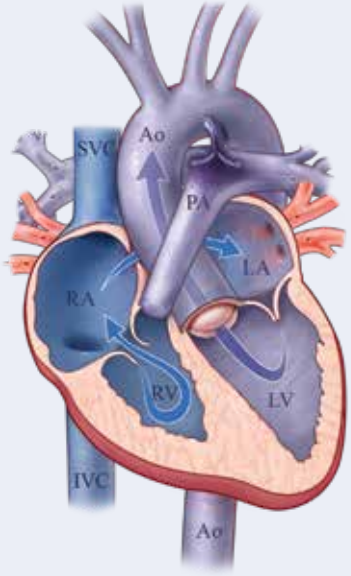
2. **Cyanotic forms of CHD that are not ductal dependent for pulmonary blood flow:**
   - Tetralogy of Fallot*
   - Tricuspid atresia*
   - Truncus arteriosus
   - Total anomalous pulmonary venous connection
   - Ebstein anomaly*

3. **Cyanotic forms of CHD that are ductal dependent for pulmonary blood flow:**
   - Pulmonary atresia with intact ventricular septum
   - Pulmonary atresia with ventricular septal defect
   - Transposition of the great arteries

*Some forms may be ductal dependent







The Appendix on page 179 contains palliative and surgical options for the heart lesions presented in Part 2.

What does it mean?
Let's talk about CHD that is ductal dependent[82,83,169,180]

The ductus arteriosus (DA)

In utero, the placenta (not the lungs) is the site of gas exchange. In the fetus, most of the blood entering the right ventricle bypasses the lungs by flowing through a vessel that connects the pulmonary artery and aorta – the ductus arteriosus (DA). The DA is an artery that is lined with smooth muscle that normally closes shortly after birth. In most term infants, anatomic closure is by 4 days of age, but in some term infants, closure of the DA may be delayed or not happen spontaneously at all.

Factors contributing to vasoconstriction and closure of the DA include increased arterial oxygen content that occurs when the lungs take over gas exchange after birth, and removal of vasodilatory substances from the placenta upon cord separation.

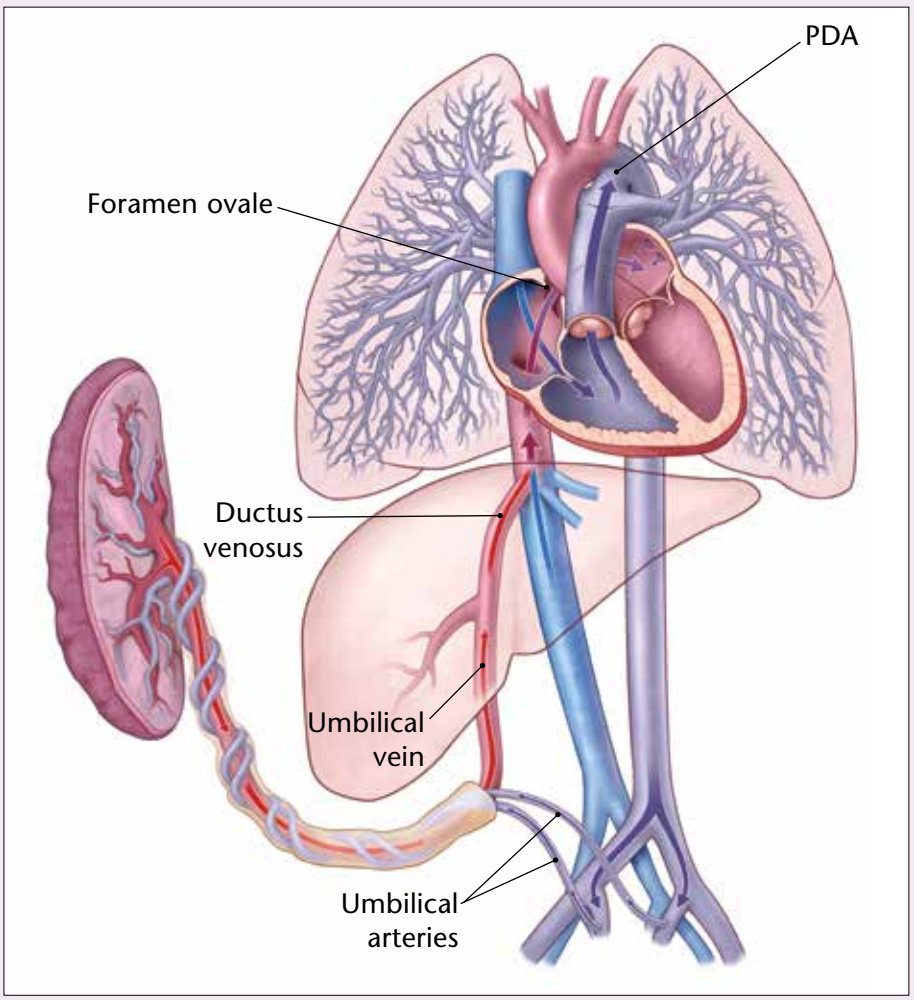
Ductal dependent left-sided obstructive lesions

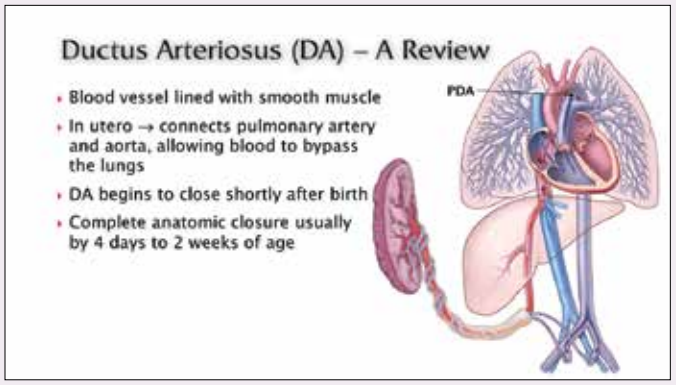
Infants with poor systemic perfusion secondary to critical left-sided obstructive lesions (coarctation of the aorta, critical aortic valve stenosis, interrupted aortic arch, hypoplastic left ventricle) will depend on a **right**-to-**left** shunt through the DA for systemic circulation. As the DA closes, poor cardiac output leads to shock.

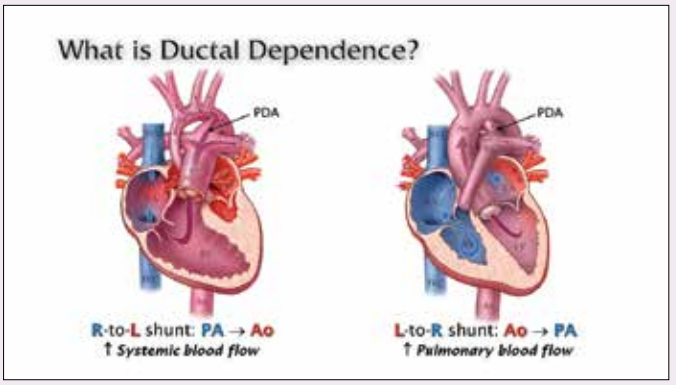
Ductal dependent right-sided obstructive lesions

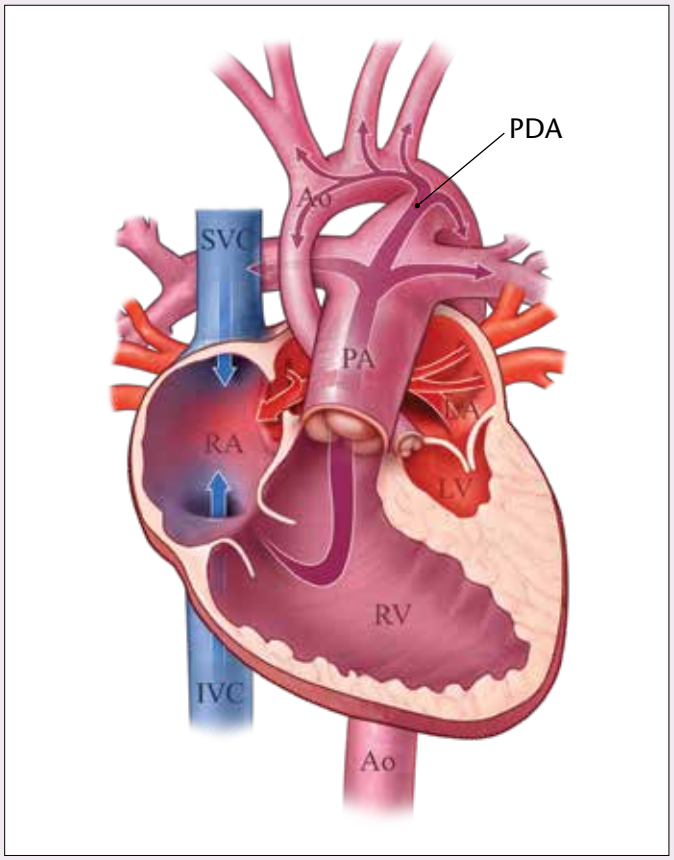
Infants with significantly decreased pulmonary blood flow secondary to right-sided obstructive lesions (pulmonary atresia with intact ventricular septum, severe pulmonary stenosis, tetralogy of Fallot with severe pulmonary stenosis or atresia, and tricuspid atresia with normally related great vessels, restrictive VSD and inadequate pulmonary blood flow), will depend on a **left**-to-**right** shunt from the aorta through the DA to the pulmonary arteries and lungs.

With transposition of the great arteries with an intact ventricular septum, maintaining ductal patency helps increase intercirculatory mixing by increasing the pressure in the left atrium to help drive oxygenated blood across the atrial septum to the right atrium, right ventricle, and out the aorta.

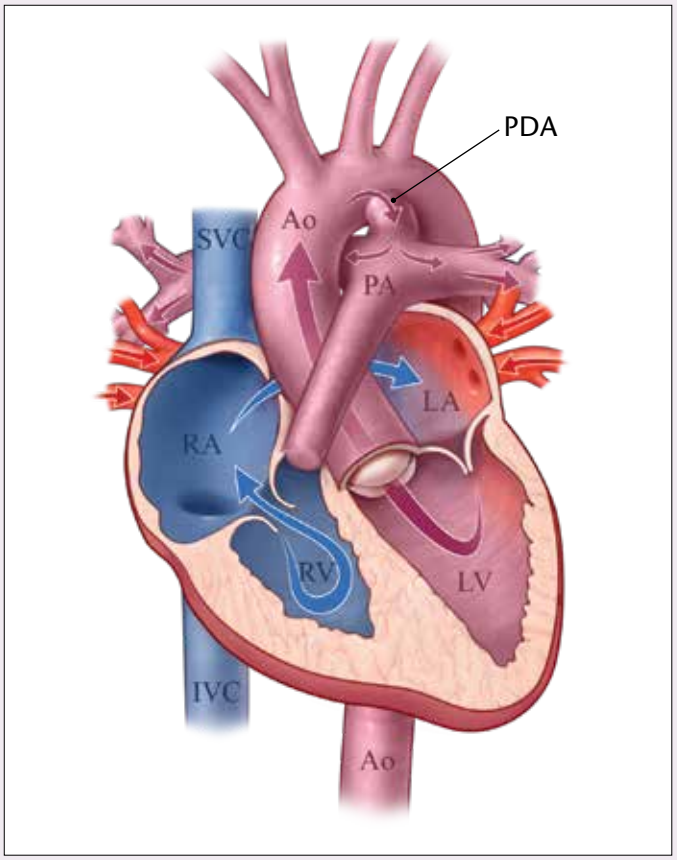








Hypoplastic left heart syndrome dependent upon a right-to-left shunt at the DA for systemic perfusion. Oxygenated blood drains from the pulmonary veins to the left atrium. Because of left ventricular obstruction, blood in the left atrium will shunt across the foramen ovale or ASD into the right atrium. Blood will then shunt right-to-left from the pulmonary artery through the DA to the aorta to perfuse the body, brain (head and neck vessels), and coronary vessels.



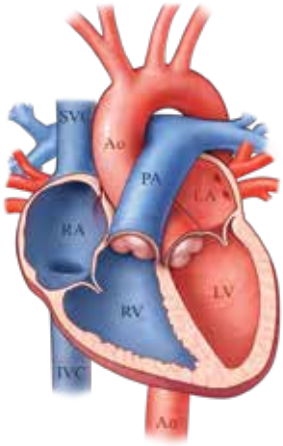
Pulmonary atresia dependent upon a left-to-right shunt at the DA for pulmonary perfusion. Because of outflow obstruction secondary to pulmonary valve atresia, blood that enters the right ventricle regurgitates back into the right atrium where it shunts across the foramen ovale or ASD to the left atrium, left ventricle, then into the aorta. To perfuse the lungs, blood shunts from the aorta, left-to-right through the DA, to the pulmonary arteries and lungs. In this setting, the orientation of the DA is reversed in comparison to the orientation of the normal DA with right-to-left flow in utero.

Tetralogy of Fallot (TOF)[71,73,171,186,188,194-196]

TOF affects approximately 10% of all infants with CHD and occurs in males slightly more than females. Four primary abnormalities characterize TOF:

- A large ventricular septal defect (VSD)
- RVOT obstruction secondary to infundibular (subvalvar) stenosis (most common) and/or pulmonary valve stenosis
- Aortic override of the ventricular septum
- Right ventricular hypertrophy secondary to systemic right ventricular pressure in the setting of a large VSD (i.e. the blood pressures in the ventricles are equal, whereas without a VSD, the RV blood pressure should be much lower)

A right aortic arch is present in 25% of cases of TOF. The pulmonary valve annulus may be near normal in size to severely hypoplastic. The severity of cyanosis relates to the degree of RVOT obstruction. The most severe form of TOF, pulmonary atresia with a VSD, is described in more detail on page 132.



Normal heart

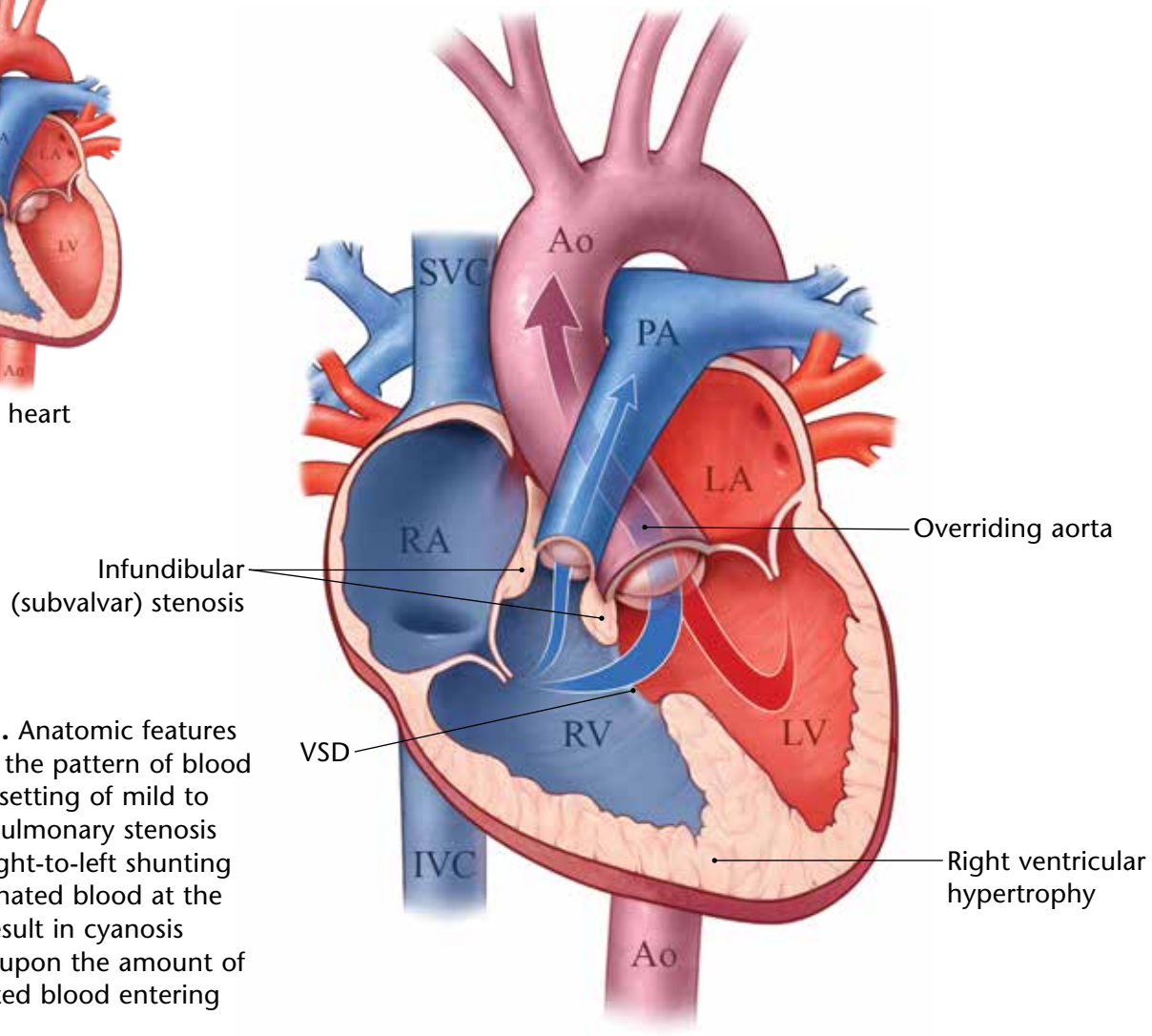
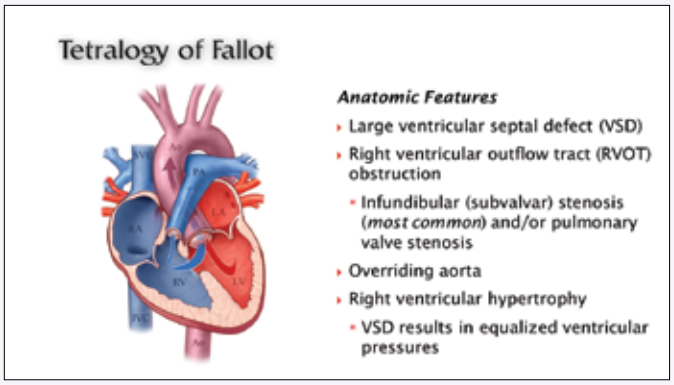
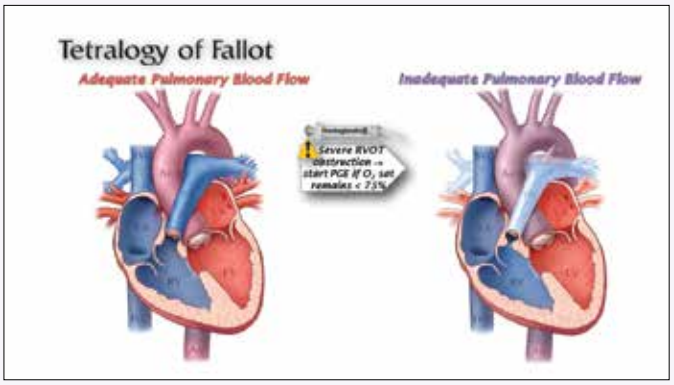


Figure 2.9. Anatomic features of TOF and the pattern of blood flow in the setting of mild to moderate pulmonary stenosis (PS). The right-to-left shunting of deoxygenated blood at the VSD may result in cyanosis depending upon the amount of deoxygenated blood entering the aorta.





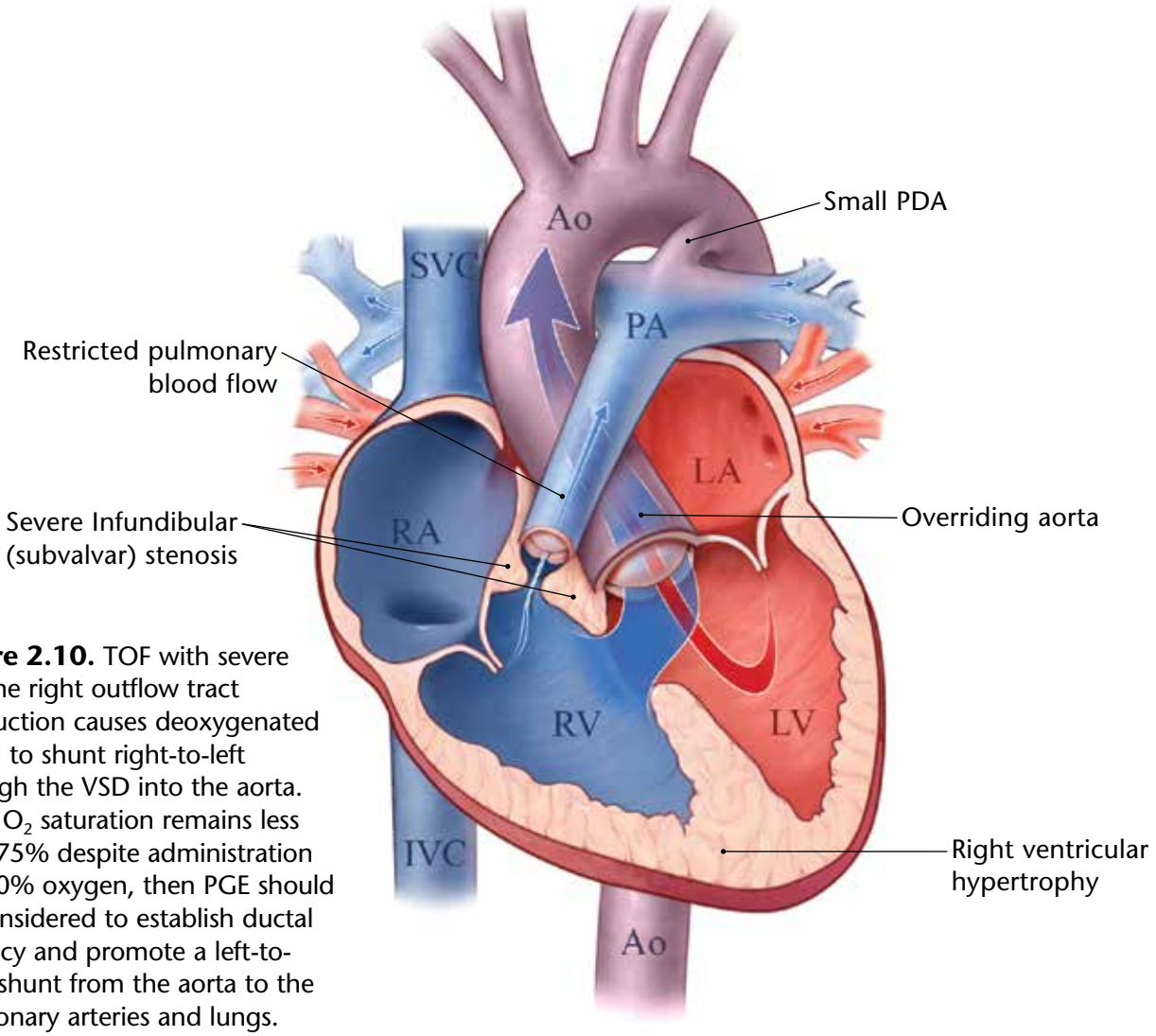


Figure 2.10. TOF with severe PS. The right outflow tract obstruction causes deoxygenated blood to shunt right-to-left through the VSD into the aorta. If the O_2 saturation remains less than 75% despite administration of 100% oxygen, then PGE should be considered to establish ductal patency and promote a left-to-right shunt from the aorta to the pulmonary arteries and lungs.

Sugar, Temperature, Airway, Blood Pressure, Lab Work, and Emotional Support

Introduction

Healthcare professionals involved with neonatal and emergency care will occasionally encounter neonates who are ill with cardiac conditions. Prompt, effective, and appropriate care can reduce secondary organ damage, improve short- and long-term outcomes, and reduce morbidity and mortality. This section will briefly discuss the six S.T.A.B.L.E. assessment components (Sugar, Temperature, Airway, Blood pressure, Lab work, and Emotional support) and any modifications specific to neonates with suspected or confirmed CHD.[230]

Care must be provided based on the neonate's condition at presentation. If the infant is in shock, attention must first be devoted to reducing the work of breathing and identifying and treating primary causes of acid-base disturbances. Supportive and resuscitative measures include assisting ventilation and improving oxygenation, improving blood pressure and perfusion, establishing intravenous (IV) and arterial access, administering medications, and maintaining a normal body temperature.

Sugar and Safe Care Module

Sick infants often do not tolerate enteral (oral or gavage) feedings, and if the infant experienced shock, then intestinal perfusion may have been impaired. In the presence of ductal dependent lesions, as the DA closes, infants will display signs of increased distress that include disinterest in feedings, a weak suck, and development of intestinal ileus and vomiting. When an infant has suspected CHD, the safest approach is to withhold feedings and establish IV access as soon as possible to ensure an IV line is available for medications and fluid resuscitation. In addition, placement of an IV will allow the infusion of appropriate glucose-containing solutions to support the infants' increased energy demands.

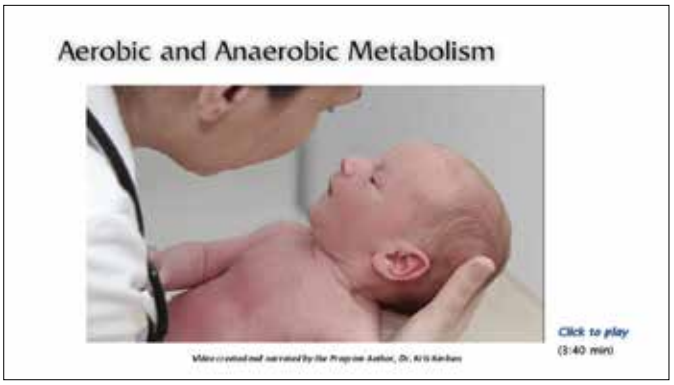
Glucose Production and Utilization Rate[231-235]

Glucose is the primary fuel used by the brain; therefore, adequate blood glucose levels are essential for normal brain function. In healthy term neonates, the liver glycogen breakdown and glucose production rate are approximately 4 to 6 mg/kg/minute. Sick infants are at increased risk for hypoglycemia because of an increased glucose utilization rate that may exceed their glucose production or availability rate. Neonates with CHD may also have concurrent illnesses that place them at higher risk for hypoglycemia, including chromosomal or genetic conditions, hyperinsulinemia, intrauterine growth restriction, and prematurity.

Initial IV Fluid Rate and Target Glucose Levels[230,232,233,235-242]

The S.T.A.B.L.E. Program defines hypoglycemia as "glucose delivery or availability that is inadequate to meet glucose demand."
The exact blood glucose value that defines hypoglycemia remains controversial. In addition, glucose values tolerated by individual infants may vary because of their specific diagnoses and medical problems. When the blood sugar is low, action should be taken to restore the blood sugar to an euglycemic, or normal blood glucose concentration.[243,244]

Once IV access is established, administer $D_{10}W$ at 80 mL/kg/day to provide a glucose infusion rate of 5.5 mg/kg/minute. Infants with hyperinsulinemia and/or accelerated glucose utilization may need a higher dextrose concentration than $D_{10}W$. In the first 2 days of age, the goal blood sugar is 50 to 110 mg/dL (2.8 to 6.1 mmol/L) and after 48 hours, the goal blood sugar is 60 to 110 mg/dL (3.3 to 5.1 mmol/L). The addition of electrolytes, calcium, and magnesium should be considered once the infant is greater than 48 hours old.



A neonate who develops cardiogenic shock or obstructive shock related to ductal closure is at increased risk for acute renal failure and oliguria. Hyponatremia, the appearance of edema, and development of CHF are associated with an imbalance between intake and output. If shock has occurred and the patient is normoglycemic, consider decreasing the baseline fluid rate to 60 mL/kg/day. If the infusion rate is decreased, monitor the blood sugar regularly to ensure it remains consistently >50 mg/dL (<48 hours of age), or >60 mg/dL (>48 hours of age).

If the blood sugar is lower than desired, administer a 2 mL/kg $D_{10}W$ glucose bolus, which equals 200 mg/kg of glucose, at a rate of 1 mL per minute. More than one glucose bolus may be required to stabilize the blood sugar. To prevent hyperglycemia and rebound hypoglycemia, do not give $D_{25}W$ or $D_{50}W$ dextrose concentrations. If the blood

Blood Pressure Module[125,130-133,135,260]

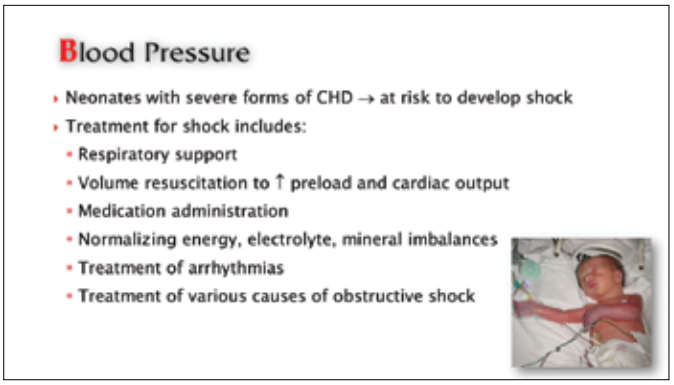
A neonate with severe CHD, poor cardiac output, and/or severe hypoxemia is at risk for developing shock. Delayed or insufficient treatment may lead to permanent tissue injury, organ damage, and death. Rapid, effective treatment of shock may include the following:

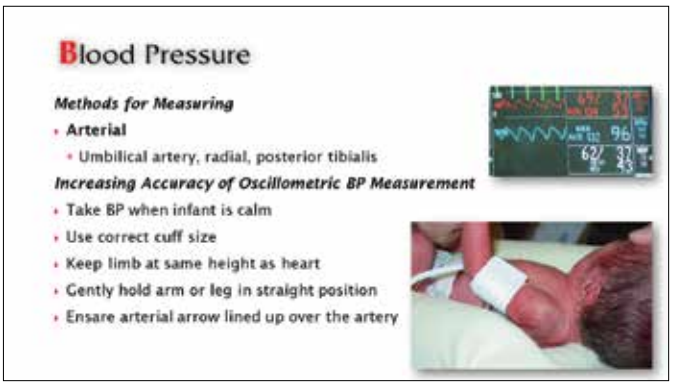
- Respiratory support to increase tissue oxygenation, assist with CO_2 removal, and decrease work of breathing.
- Volume resuscitation to increase preload and cardiac output.
- Administration of medications to improve heart function and systemic and pulmonary blood flow (including PGE, as applicable).
- Normalizing energy (glucose), electrolytes (sodium, potassium, and chloride), or mineral imbalances (magnesium and calcium), that reduce heart contractility when abnormal.
- Treatment of arrhythmias (too fast or too slow) that impair cardiac output.
- Relief of noncardiac causes of obstructive shock that may be secondary to cardiac tamponade, air leak, or hyperinflation.
- Obstructive shock secondary to left outflow tract obstructive lesions is treated with PGE.

Methods for Measuring BP[138,295]

Arterial

Common sites for arterial, invasive BP monitoring in neonates include the umbilical, radial, and posterior tibial arteries. When the transducer is located at the level of the heart, the catheter and transducer are free of air bubbles, and the waveform is not dampened by clots or other obstructions, arterial BP is the most accurate method for assessing BP.





Oscillometric Measurement

Noninvasive blood pressure (NIBP) monitoring allows for the determination of systolic, diastolic, and mean arterial BP. Practitioners should be aware of circumstances in which NIBP monitoring may yield inaccurate results compared to results obtained by invasive BP (IBP) monitoring. Poor correlation between NIBP and IBP measurements is observed in infants with lower gestational ages and weights, lower postmenstrual age, ill preterm and term infants, and infants receiving mechanical ventilation.[147,295-297] The direction of inaccuracy is toward false elevations of mean, systolic, and diastolic BPs when compared to IBP measurements.[147,297] Thus, it is essential that thorough patient assessment accompanies interpretation of NIBP measurements since results obtained may overestimate the actual BP. Figure 3.15 provides information about how oscillometric BP is determined and guidelines about how to improve accuracy when taking a cuff BP.

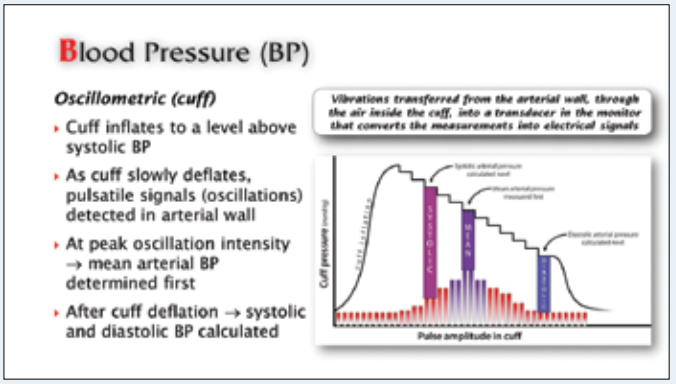
Oscillometric Measurement – How Does it Work?[137,147,148,295,298-304] and personal communication

Bruce Friedman D.Eng., Principal Engineer Analytics, GE Healthcare Technologies, June 9, 2020

The cuff inflates to a level above systolic BP. As the cuff slowly deflates, arterial blood flow increases, and pulsatile signals or oscillations are detected in the arterial wall. The oscillations increase in intensity; when they reach their peak, the mean arterial BP is determined first. The cuff further deflates and when blood flows smoothly through the artery without further detection of oscillations, systolic and diastolic BPs are calculated.

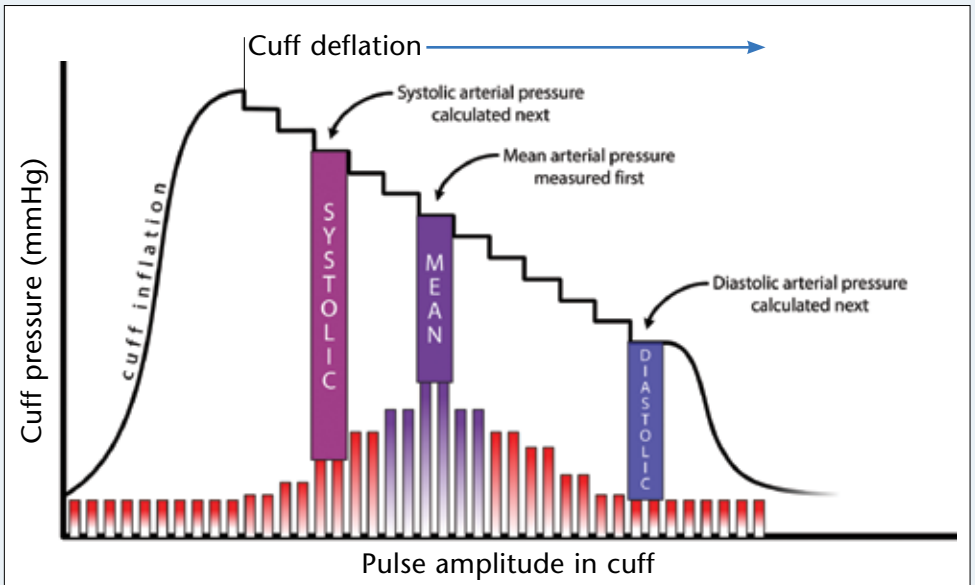
Information obtained during cuff deflation is sensed by a pressure transducer located in the NIBP monitor. The monitor analyzes data via a proprietary algorithm, resulting in display of a systolic, diastolic, and mean arterial BP.

The cuff may be placed on the arm, thigh, or calf. To enable comparison of results, it is helpful to take the BP on the same limb (right upper arm, if possible). Abnormal BP results should be repeated and always correlate findings with patient assessment.



The following items help improve accuracy of oscillometric BP measurement:

- Take the BP when the infant is in a calm state because movement interferes with accuracy of the reading.
- Use the correct cuff size. Calf and arm BPs correlate closely, providing the proper cuff size is selected based on the circumference of the midarm or calf. Too small of a cuff overestimates BP, whereas too large of a cuff underestimates BP.
- Keep the arm or leg at the level of the heart. Gently hold the extremity in a straight position distal to the cuff placement.
- Line up the arterial arrow that is on the cuff with the brachial artery (for an arm BP) or the popliteal artery (for a leg BP). The bladder of the cuff underlies where the tubing is located, and it is the bladder that is detecting the oscillations in the artery.



Graph adapted with permission of Bruce Friedman (GE Healthcare) © 2020.

Figure 3.15. Noninvasive oscillometric blood pressure (BP) measurement in neonates.

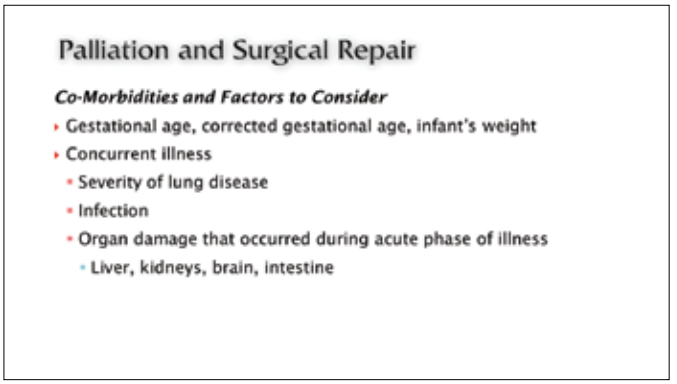
Introduction

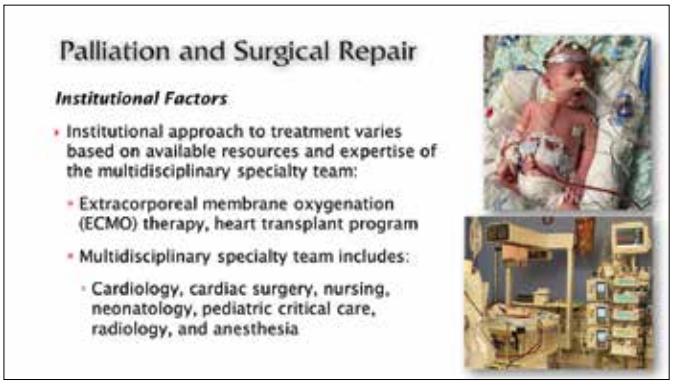
Complete surgical repair of heart defects is performed when possible. However, some infants may be too small or have co-morbidities or other factors that need to be considered when deciding the best timing and approach. These co-morbidities and factors include the following:

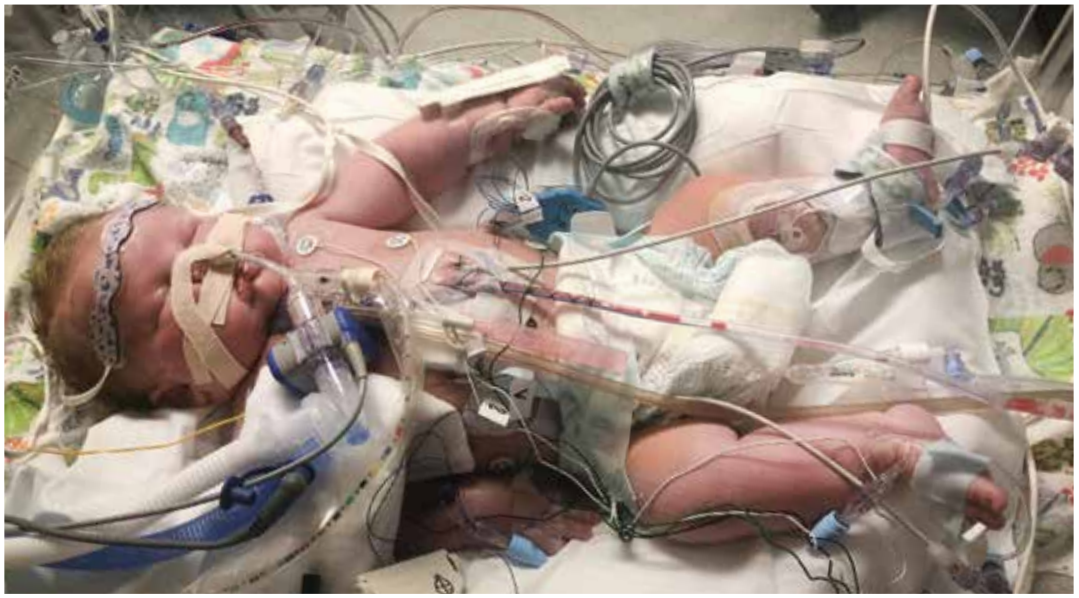
- Prematurity, corrected gestational age, and the infant's weight.
- Concurrent illnesses including lung disease and infection.
- Any organ damage that occurred during the acute phase of illness.

Decisions regarding surgical approach are based upon factors including:

- Institutional approaches based on available resources and expertise of the multidisciplinary specialty teams.
- Availability of extracorporeal membrane oxygenation therapy.
- Whether there is a heart transplant program.







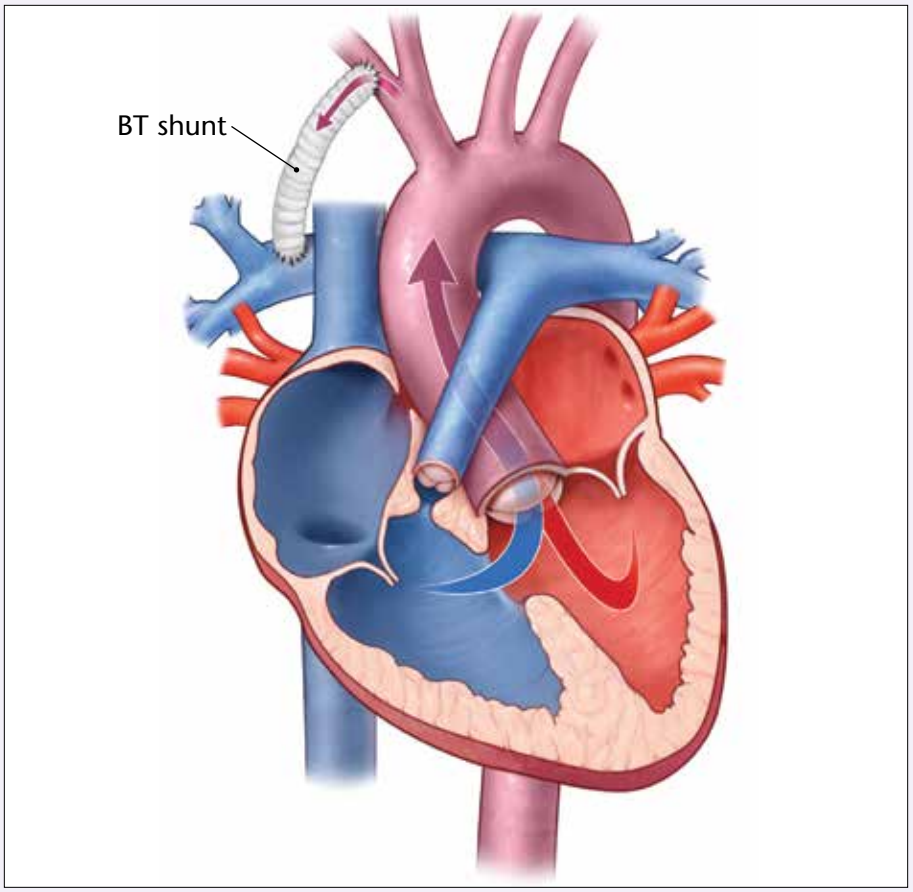
Infant with DORV and dextrocardia following surgical intervention.

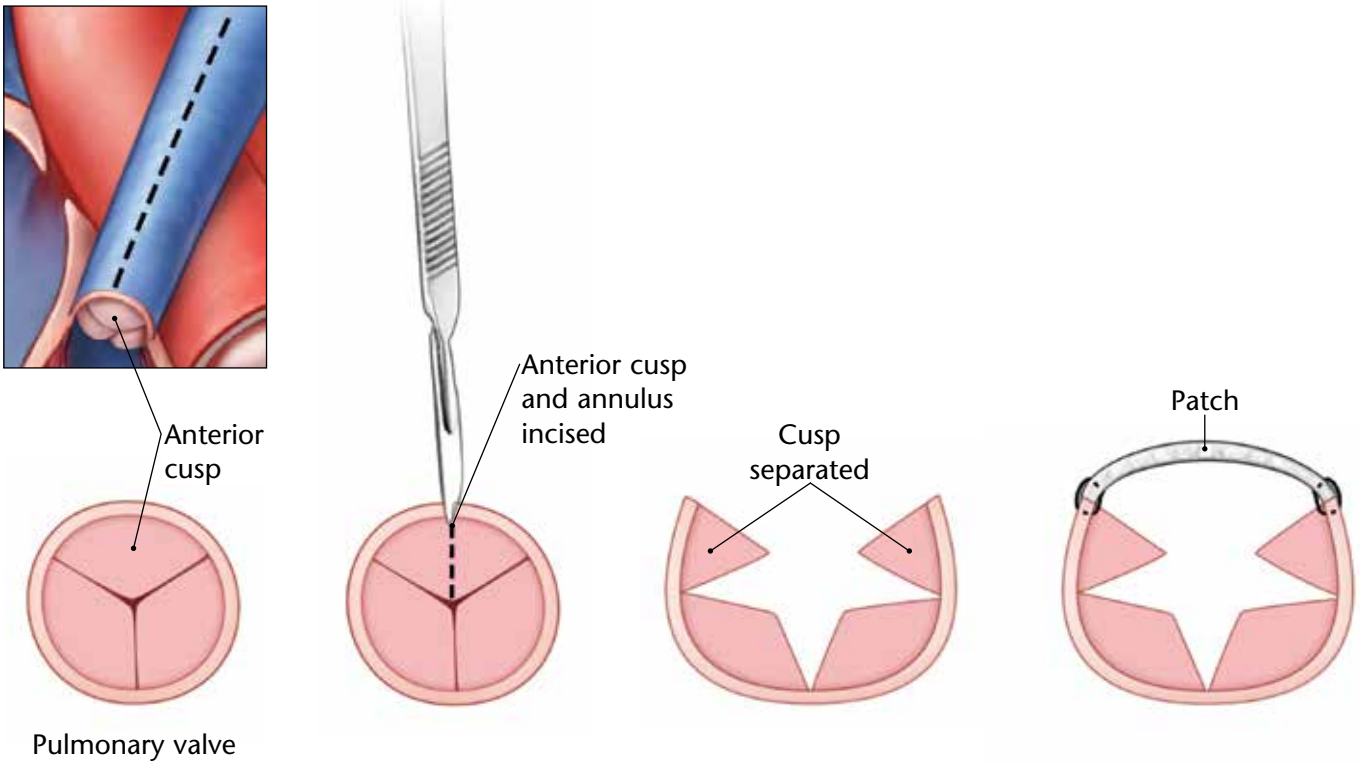
*This Appendix of palliative and surgical repair options will follow the order of the lesions presented in Part 2.

Tetralogy of Fallot – Palliative Options

Systemic-pulmonary artery shunt (Blalock-Taussig/ BT shunt)

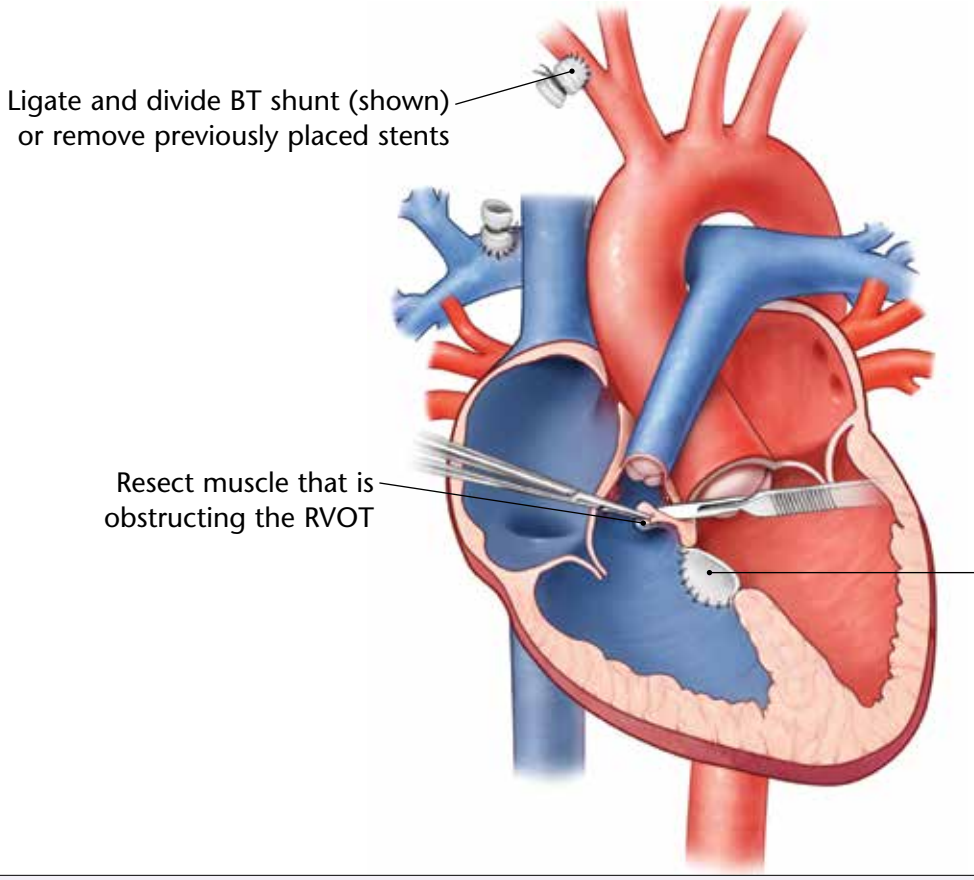
To provide adequate pulmonary blood flow *(surgical procedure)*.

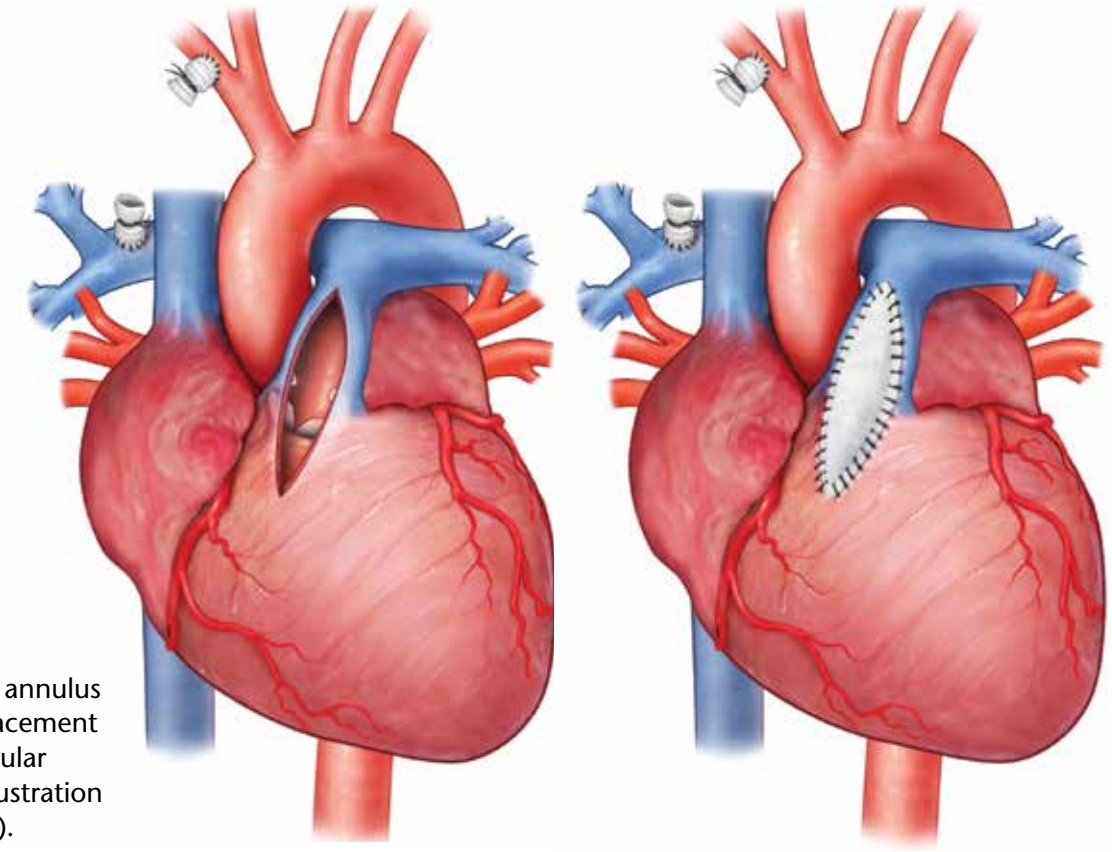




Patient will require future placement of a competent pulmonary valve.

Tetralogy of Fallot – Surgical Repair





If pulmonary annulus too small, placement of a transannular patch (see illustration on page 200).