The Neonate After Cardiac Surgery: What do You Need to Worry About in the Emergency Department?

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Congenital heart disease (CHD) is the most common congenital malformation, with an incidence of 6 in 1000 live births. Although many patients with CHD are diagnosed prenatally, more than one third of neonates with critical lesions are diagnosed in the newborn nursery, and up to 20% can present after discharge. These neonates can be at high risk of cardiovascular collapse at initial diagnosis as well as life-threatening complications after surgical correction or palliation of their cardiac lesion. This article focuses on common corrective and palliative procedures in neonates with CHD as well as postoperative complications that may be encountered in the emergency department. Specifically, hemodynamic, infectious, and intrathoracic derangements as well as common postoperative dysrhythmias will be discussed. Approaches to airway management, vascular access, and procedural sedation in this patient population will also be addressed.

Keywords: congenital heart disease; neonate; cardiac surgery; shunt

COMMON CARDIAC SURGERIES IN THE NEONATE

Congenital heart disease requiring surgery in the neonatal period falls into 3 broad categories: lesions with either ductal...
dependent systemic blood flow or ductal dependent pulmonary blood flow and ductal independent lesions (Table 1). Neonates with single ventricles comprise a large proportion of neonates admitted to tertiary care cardiac intensive care units.\(^3\) The single ventricle is the right ventricle in hypoplastic left heart syndrome and the left ventricle in patients with tricuspid atresia. Patients with hypoplastic left heart syndrome undergo palliation with a Norwood operation shortly after diagnosis. The Norwood operation consists of atrial septostomy to create a single atrium, ligation of the main pulmonary artery from the right and left pulmonary arteries, creation of a neoaorta arising from the right ventricle, and placement of a synthetic shunt from the systemic circulation to the pulmonary circulation (Figure 1). The shunt may arise from the right ventricle (Sano conduit) or from a major systemic artery such as the innominate artery (modified Blalock-Taussig [BT] shunt). These shunts are approximately 3.5 to 5 mm in diameter\(^4\) and are the sole source of pulmonary blood flow in these patients. A modified BT shunt is also necessary in newborns with tricuspid atresia.

Neonates with critical coarctation of the aorta and interrupted aortic arch will usually undergo reconstruction with left subclavian artery flap aortoplasty.\(^5\) This procedure has important implications when evaluating these neonates postoperatively for restenosis or arch obstruction. Surgical repair of tetralogy of Fallot usually occurs later in infancy after a period of weight gain, but neonates with severe obstruction require relief of the right ventricular outflow obstruction early in life because of severe cyanosis. Placement of a patch across the obstruction or creating a right ventricle to pulmonary artery conduit with a homograft usually involves a ventriculotomy.\(^6\) The neonatal right ventricle is not yet compliant, and these patients are at risk for dysrhythmias and myocardial dysfunction postoperatively.\(^7\)

For patients with transposition of the great arteries, a common corrective procedure performed in the neonatal period is the arterial switch operation. This surgery consists of ligation of both the aorta and pulmonary artery and reanastomosis on the left and right ventricle, respectively. Any accompanying atrial or ventricular septal defect is closed with a patch. The coronary arteries are excised, mobilized, and reanastomosed onto the aorta. Although the arterial switch procedure is currently the most common surgery performed for transposition of the great arteries, some centers may still perform atrial baffling procedures such as the Mustard or Senning operation to direct blood flow to the appropriate ventricle.

### HEMODYNAMIC COMPLICATIONS

#### Obstructed Shunt

**Recognition**

The neonate with a significantly obstructed Sano or BT shunt will present with profound cyanosis and respiratory distress that can progress rapidly to cardiovascular collapse and cardiac arrest. An obstructed shunt should be suspected when a neonate with a history of a shunt procedure has declining oxygen saturations despite administration of supplemental oxygen. Rapid identification of an obstructed shunt can be performed by listening for a continuous murmur usually present at the right upper sternal border. If the murmur is soft or absent, a shunt obstruction is highly likely. In cases where the surgical history is vague and the patient presenting with severe cyanosis has a median sternotomy incision, emergent echocardiography is required to assess for an obstructed shunt.

**Pathophysiology**

Sano and BT shunts are extremely small in diameter and have an obstruction rate of approximately 12%.\(^8,9\) Obstruction most often occurs because of thrombosis in the setting of dehydration or in instances when anticoagulation is discontinued, such as in anticipation of a surgical procedure. Shunt size less than 4 mm, young age at shunt placement, and low weight are some risk factors associated with shunt obstruction.\(^4,9\) As the shunt is

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**TABLE 1. Common congenital heart lesions requiring surgery in the neonates.**

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<thead>
<tr>
<th>Ductal dependent systemic blood flow/left-sided obstructive lesions</th>
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<tbody>
<tr>
<td>Hypoplastic left heart syndrome</td>
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<tr>
<td>Aortic stenosis</td>
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<tr>
<td>Critical coarctation of the aorta</td>
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<tr>
<td>Interrupted aortic arch</td>
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<tr>
<th>Ductal dependent pulmonary blood flow/right-sided obstructive lesions</th>
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<tr>
<td>Severe tetralogy of Fallot</td>
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<tr>
<td>Pulmonary atresia</td>
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<tr>
<td>Tricuspid atresia</td>
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<td>Ebstein anomaly</td>
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<th>Ductal independent lesions</th>
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<tr>
<td>Transposition of the great arteries</td>
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<tr>
<td>Truncus arteriosus</td>
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<td>Total anomalous pulmonary venous return</td>
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the sole source of pulmonary blood flow in these neonates, blockage causes a dramatic decrease in oxygenation of arterial blood, resulting in rapid decline in arterial oxygen content and delivery to peripheral tissues. The neonate is particularly sensitive to hypoxia. After a period of tachypnea and tachycardia in response to hypoxemia, the neonate will develop apnea and bradycardia because of an immature autonomic system and the carotid body reflex. Lack of oxygen delivery will also result in acidosis and progressive multiorgan dysfunction. If the blockage is not relieved promptly, cardiopulmonary arrest will occur.

Management
As patients with shunt obstruction can rapidly progress to asystole, the most important component of management is adequate cardiopulmonary resuscitation (CPR) until stenting or replacement of the shunt can be performed. Endotracheal intubation is often required, and airway management will be discussed later in this article. Vasopressors such as epinephrine or phenylephrine are often helpful to increase systemic vascular resistance, thereby shunting blood into the pulmonary vasculature. Identification and correction of acidosis are also critical. Anticoagulation with an intravenous bolus of heparin at 50 to 100 U/kg should be administered to prevent progression of the obstructing thrombus. Extracorporeal membrane oxygenation may be necessary to augment CPR, and a cardiothoracic surgeon should be contacted immediately if there is concern of a shunt obstruction. Unfortunately, despite these interventions, mortality is high in patients with shunt obstruction.

Arch Obstruction/Restenosis

Recognition
Aortic arch obstruction after surgery for critical coarctation of the aorta or interrupted aortic arch will present with decreased or absent femoral pulses, poor perfusion in the lower extremities, and, if severe, circulatory shock. Comparison of blood pressures in the right upper extremity to blood pressures in the lower extremities is important for diagnosis. Many neonates undergo left subclavian artery flap repair of their initial coarctation, so blood pressure will often be unobtainable in the left upper extremity at baseline.

Pathophysiology
The rate of restenosis after initial surgical repair is approximately 11% to 14% and is higher if balloon angioplasty of the coarctation was initially performed. Although low weight at initial operation was once thought to be a risk factor for restenosis, a recent study demonstrated no difference in reintervention rates based on weight. The obstruction prevents blood flow through the aortic arch into the descending aorta, which can lead rapidly to multiorgan dysfunction.

Figure 1. A, Norwood operation with neoaorta and BT shunt. B, Norwood operation with Sano conduit. PTFE indicates polytetrafluoroethylene; RV-PA, right ventricle to pulmonary artery. Figure used with permission from http://hlhs-awareness-uk.com.
Management

In addition to management of airway and breathing, emergent cardiology consultation is necessary so that balloon angioplasty of the stenotic area can be performed. While awaiting definitive repair of the obstruction, normal saline boluses may be required to increase lower extremity perfusion. Identification and correction of acidosis should also be performed.

INFECTIOUS COMPLICATIONS

Septicemia

Recognition

As in any neonate, fever may be the only sign of invasive bacterial disease and should prompt evaluation for serious bacterial illness. Signs of septic shock include poor perfusion, lethargy, and respiratory distress.

Pathophysiology

The risk of invasive bacterial disease in febrile young infants is 8% to 12.5%, and neonates postoperative from cardiac surgery have additional risk due to endocarditis and mediastinitis. The incidence of septicemia was 2.6% in a retrospective study of 30,078 children after cardiac surgery, with the highest rate of major infection occurring after the Norwood operation. In addition to the usual neonatal pathogens such as group B Streptococcus, Escherichia coli, and Listeria monocytogenes, neonates after cardiac surgery are also at risk for invasive infection with skin flora such as Staphylococcus aureus as well as other gram-negative organisms including Pseudomonas aeruginosa.

Management

As with any neonate with fever, blood and urine cultures should be obtained, and the infant should be admitted for empiric intravenous antibiotics pending culture results. Although lumbar puncture is often performed in febrile young infants, neonates with recent cardiac surgery for CHD are potentially at higher risk for respiratory and hemodynamic complications with positioning, and therefore, careful attention must be given to heart rate and oxygenation during the procedure. Lumbar puncture should be deferred if the patient is hemodynamically unstable. The neonate presenting in septic shock should receive early broad-spectrum antibiotics and correction of hypocalcemia and hypoglycemia, if present. Fluid resuscitation should be performed with the judicious administration of crystalloid. Boluses of 10 mL/kg may be used, followed by reassessment for signs of congestive heart failure (CHF) such as hepatomegaly. If signs of CHF are not present and the neonate continues to have poor perfusion, repeat boluses of 10 mL/kg of crystalloid can be administered with careful reassessment for signs of CHF.

Endocarditis

Recognition

Fever in the neonate after cardiac surgery should prompt consideration of infective endocarditis (IE). A new murmur, confirmed by patient history or review of postoperative records when available, may occur secondary to dehiscence of a patch of conduit or turbulent flow from the presence of a vegetation. Other clinical manifestations of IE in a neonate are variable and nonspecific and include poor feeding, tachycardia, and signs of sepsis. Embolic phenomena are common, resulting in extracardiac manifestations such as osteomyelitis and neurologic signs such as seizures and hemiparesis. Osler nodes, Roth spots, Janeway lesions, and splinter hemorrhages have not been described in neonates.

Pathophysiology

Congenital heart disease is a known risk factor for IE, and patients who have undergone palliative shunt and conduit operations are at particularly high risk. S aureus was the most common pathogen in IE in children in a recent study by Day et al, and infants had the highest mortality rate.

Management

Blood cultures should be obtained, and echocardiography should be performed to assess for vegetations. Empiric antimicrobial therapy should include broad-spectrum antibiotics with activity against both gram-negative and gram-positive organisms, including S aureus. Indications for surgical intervention include CHF, resistant bacteria, left-sided infection with gram-negative organisms, fungi, multiple embolic events, and vegetations greater than 1 cm on the anterior mitral leaflet.

Mediastinitis

Recognition

Mediastinitis should be suspected with erythema, induration, fluctuance, or purulent drainage from the sternotomy incision, wound dehiscence, or sternal instability as evidenced by separation of the
2 pieces of the sternum. Sternal instability can be diagnosed by pressing firmly with the fingertips along the entire sternum and feeling a “give” or hearing an audible “click.” Respiratory distress and signs of septic shock may also be present.

**Pathophysiology**

The overall incidence of mediastinitis postmedian sternotomy in children is approximately 1% to 2%, although the incidence is highest in neonates, occurring at a rate of 5.5% in a retrospective study by Erez et al. Gram-positive organisms, especially *S. aureus*, are the predominant pathogens, although up to one third of infections are due to gram-negative bacteria including *P. aeruginosa*. In the study by Erez et al, 3 of the 6 neonates had gram-negative infections. Fungal pathogens may also cause mediastinitis in children, although the incidence appears lower than bacterial infection. Up to 47% to 53% of children with mediastinitis have concurrent bacteremia.

**Management**

In addition to broad-spectrum intravenous antibiotics with activity against both *S. aureus* and *P. aeruginosa*, cardiothoracic surgery consultation should be obtained emergently for an unstable sternum. In addition to aggressive wound care and surgical debridement, patients may require either pectoralis muscle flap or other surgical reconstruction.

**INTRATHORACIC COMPLICATIONS**

**Pericardial Effusion and Cardiac Tamponade**

**Recognition**

The neonate with cardiac tamponade may present with tachypnea, tachycardia, hepatomegaly, and poor perfusion. Pulsus paradoxus is a sensitive sign for cardiac tamponade. Using a manual sphygmomanometer, a 10 mm Hg or greater difference between the systolic blood pressure at which the first Korotkoff sounds are present with expiration and when the Korotkoff sounds are present during both exhalation and inspiration indicates pulsus paradoxus. However, this maneuver is difficult to obtain in the neonate. Therefore, using pulse oximetry to detect pulsus paradoxus is recommended in this population. A pronounced drop in the highest value of the upper plethysmographic peak of the pulse oximetry waveform with inspiration is indicative of pulsus paradoxus (Figure 2). Cardiomegaly on chest x-ray is also a sensitive indicator of a large pericardial effusion.

**Pathophysiology**

Postoperative pericardial effusion in the neonate is uncommon but can occur due to traumatic pericarditis or due to postpericardiotomy syndrome. Although the pericardium is very compliant, there is a point at which it is inextensible, and a small amount of fluid that accumulates quickly can result in hemodynamic compromise. When the pericardium cannot stretch further, the intrapericardial pressure will equal the right and left ventricular end diastolic pressures, resulting in decreased cardiac output.

**Management**

In the neonate with cardiac tamponade, pericardiocentesis must be performed emergently. Preload is reduced because of the high intrapericardial pressure and restricted right atrial filling. Therefore, crystalloid boluses of 10 mL/kg should be administered to enhance cardiac output while awaiting pericardiocentesis. Various vasopressor and

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**Figure 2.** Pulse oximetry tracing demonstrating pulsus paradoxus. A pronounced drop in the amplitude of the plethysmographic peak of the pulse oximetry waveform (lower circle) is seen on inspiration (top circle). Figure provided by Geoffrey Bird, MD.
inotropic agents have been studied in cardiac tamponade. Although hemodynamics were improved with the use of dopamine, isoproterenol, and norepinephrine in dog models, these agents did not improve the cardiac index to the same degree in human patients. Dobutamine improved cardiac index and tissue oxygen delivery with increasing levels of intrapericardial pressures compared with norepinephrine in a randomized controlled trial involving dogs. In summary, although data are limited on the use of inotropic agents in patients with cardiac tamponade, inotropic agents, particularly dobutamine, may be used in addition to fluid therapy while awaiting pericardiocentesis.

**Postpericardiotomy Syndrome**

**Recognition**
Postpericardiotomy syndrome should be suspected in the neonate presenting 1 to 2 weeks postoperatively with fever and signs of a pericardial effusion such as a friction rub, cardiomegaly on chest x-ray, or cardiac tamponade as described above. Other symptoms may include poor feeding, decreased activity, tachypnea, or gastrointestinal distress. Pleural effusions can also occur as a component of the syndrome.

**Pathophysiology**
Postpericardiotomy syndrome occurs after wide incision of the pericardium. The incidence is approximately 27% to 30% after surgery with entry into the pericardium, although the incidence is only 1.6% to 3.5% in children younger than 2 years old. Acute or reactivated viral infections and an immunologic response resulting in antiheart antibodies are both associated with the development of postpericardiotomy syndrome. The resultant inflammatory response causes accumulation of pericardial and often pleural fluid. Although cardiac tamponade can occur, the syndrome usually resolves spontaneously over a period of days to months.

**Management**
Because of its self-limiting pattern, there are no clinical trials evaluating therapy in postpericardiotomy syndrome. Salicylates and corticosteroids are most commonly used. Prompt resolution of symptoms has been reported with corticosteroids. Pericardiocentesis should be performed in neonates who develop cardiac tamponade.

**DYSRHYTHMIAS**

**Recognition**
The neonate with a postoperative dysrhythmia may present with symptoms such as irritability, poor feeding, and tachypnea that may be mistaken for other etiologies such as bacterial or viral infection. Clinical findings may include poor perfusion evidenced by delayed capillary refill, cool extremities, and mottled skin. In addition, if prolonged, the dysrhythmia may result in signs of CHF such as hepatomegaly and tachypnea. Although neonates may have sinus tachycardia at rates up to 220 beats per minute (bpm), supraventricular tachycardia or junctional ectopic tachycardia (JET) should be suspected when the heart rate is above 220 bpm or is at a fixed rate without variation. Ectopic atrial tachycardia (EAT) can occur with heart rates less than 220 bpm.

**Pathophysiology**
Postoperative arrhythmias are a significant cause of morbidity and mortality after palliative and corrective procedures for CHD, occurring at a rate of 14% to 34%. Junctional ectopic tachycardia was the most common postoperative dysrhythmia in a study of 402 children undergoing surgery for CHD and occurs most frequently after the arterial switch and Norwood operations. Ectopic atrial tachycardia can also occur after the arterial switch procedure. Ventricular tachycardia and fibrillation are uncommon in this population. Postoperative arrhythmias can result from direct surgical injury caused by myocardial incision, sutures that affect function of the conduction system, and rapid changes of intracardiac pressures caused by volume and pressure fluctuations after cardiopulmonary bypass. Because of ineffective cardiac contraction, patients with postoperative dysrhythmias will have poor cardiac output that can result in ischemia and CIHF.

**Management**
Management consists of careful attention to airway, breathing, and circulation. As supraventricular tachycardia and JET may be difficult to distinguish without cardiology consultation, vagal maneuvers should be attempted while establishing intravenous access. Vagal maneuvers in the neonate consist of placing ice over the mouth to stimulate the diving reflex or placing the infant’s knees to the chest. If these maneuvers fail to resolve the
arrhythmia, adenosine may be administered through an intravenous line that is placed as close to the heart as possible. Junctional ectopic tachycardia may respond to amiodarone, which should be administered in the presence of a cardiologist. β-Blockers and digoxin may be used in patients with EAT to slow the ventricular rate. Reducing sympathetic output through calming maneuvers and treating fever is also important. Gentle fluid resuscitation in boluses of 5 to 10 mL/kg may be used in the dehydrated neonate. Identification and correction of electrolyte disturbances, including hypomagnesemia, should be performed as these abnormalities may propagate a dysrhythmia.

**PROCEDURES**

**Airway Management**

Airway management in the neonate postoperative from cardiac surgery has several unique considerations in addition to the normal challenges of neonatal airway management. Interventions such as positive pressure ventilation and hyperoxia in addition to medications used for rapid sequence intubation can all have significant cardiovascular effects in the postoperative period. Conversion to positive pressure ventilation increases intrathoracic pressure and is associated with reduced venous return resulting in decreased preload and cardiac output. These hemodynamic effects are particularly significant in the neonate postoperative from cardiac surgery because of the neonate’s stiff immature myocardium that cannot easily tolerate changes in volume and pressure. In addition, the increased intrathoracic pressure will also reduce pulmonary blood flow in patients with systemic to pulmonary shunts. Therefore, several issues must be addressed. First, maximizing preload before and after conversion to positive pressure ventilation is critical. Normal saline boluses of 10 mL/kg should be readily available to be given rapidly if signs of decreased cardiac output develop. In addition, vasopressor therapy such as dopamine should be available at the bedside. Pulmonary blood flow and cardiac output can decrease dramatically in these patients. Hypoxia and acidosis can also occur rapidly, resulting in bradycardia and possibly cardiac arrest. Therefore, it is critical to anticipate cardiovascular collapse. In addition to preparation to deliver adequate CPR, intravenous epinephrine should be readily available at the bedside. Finally, careful attention must be given to the end-expiratory and inspiratory pressures used during bag mask ventilation. A pressure manometer should be connected to the self-inflating bag valve mask, and positive end-expiratory pressure should not exceed 5 mm Hg.

The second special consideration in neonates is to be aware of the patient’s baseline oxygen saturation. Patients with Sano or BT shunts will have complete mixing of deoxygenated and oxygenated blood and will have arterial oxygen saturations of 75% to 85%. Achieving oxygen saturations above this level will result in pulmonary vasodilation and overcirculation of the pulmonary vasculature at the expense of the systemic circulation. Pulmonary overcirculation will result in CHF and worsened pulmonary dynamics in a patient who may already have hemodynamic compromise. Inspired oxygen concentration and positive end-expiratory pressure should be carefully adjusted to maintain the patient’s baseline oxygen saturation.

The hemodynamic effects of medications used for rapid sequence intubation are an important consideration in all patients and especially for neonates postoperative from cardiac surgery. Premedication with atropine is recommended for its vagolytic properties to prevent reflex bradycardia, which can be hemodynamically significant in these neonates. Glycopyrrolate is also an effective vagolytic but has not been well studied in neonates. For sedation and analgesia, use of medications with minimal effect on cardiovascular stability is beneficial. Using an opioid analgesic alone is an acceptable option. Fentanyl did not adversely affect systemic or cerebral hemodynamics in a case series of 15 premature neonates. Remifentanil has also been associated with stable hemodynamics and successful intubation in preterm infants. Chest wall rigidity is a risk in neonates, but this can be prevented with slow administration or use of naloxone or coadministration of a muscle relaxant. Thiopental has been used successfully for endotracheal intubation in neonates but has potential to cause hypotension and therefore is not a first-line agent in neonates with CHD. Propofol has been used with conflicting results in preterm neonates undergoing endotracheal intubation and in older children with CHD undergoing cardiac catheterization. Because of its effect of lowering systemic vascular resistance and the paucity of evidence in neonates, it is not currently recommended in the neonate postoperative from cardiac surgery. Midazolam is also not recommended as a first-line agent in this population because of a prematurely stopped trial that demonstrated frequent desaturations and a trend toward increased need for CPR in preterm infants when midazolam was used as a sedative for endotracheal intubation. In addition, there is a
risk of hypotension with its use.\(^\text{46}\) Finally, a muscle relaxant should be administered to provide optimal conditions and reduce the physiologic response of the neonate to endotracheal intubation.\(^\text{36}\) Although muscle relaxants have not been studied in neonates postoperative from cardiac surgery, there are several clinical trials showing benefits with both depolarizing and nondepolarizing agents in preterm and term neonates.\(^\text{57-59}\)

### Vascular Access

The principles of vascular access in the neonate postoperative from cardiac surgery are the same as for any neonate with a few important features to highlight. Obtaining rapid vascular access is paramount in these often critically ill patients, and early consultation with a surgeon or anesthesiologist experienced with neonatal vascular access may be lifesaving. Intravenous catheters should ideally be placed in the neck or upper extremities above the nipple line for proximity to the heart. If an intravenous line cannot be placed above the waist in a rapid fashion, placing a lower extremity intravenous or intraosseous catheter is acceptable. Ultrasound-guided vascular access has been used in infants and children with good success.\(^\text{60}\)

### Procedural Sedation

Many of the medications recommended for endotracheal intubation may also be used for procedural sedation. Dexmedetomidine is a central \(\alpha\)-2 adrenergic receptor agonist with sedative and mild analgesic properties. One case series by Barton et al\(^\text{61}\) described the use of dexmedetomidine for invasive procedures in 5 infants, including 2 neonates, with CHD. Three patients required additional sedation with ketamine. All of the infants were placed on prophylactic high-flow nasal cannula throughout the procedure. No respiratory depression was seen, and 1 neonate developed mild hypotension that responded to a fluid bolus without effect on perfusion.\(^\text{61}\) Ketamine is used frequently in older infants and children with CHD for painful procedures because of its maintenance of cardiovascular stability.\(^\text{62}\) Although its use has not been well studied in neonates, low doses of ketamine may be used alone or in combination with dexmedetomidine as described previously.\(^\text{61}\) Propofol has been used in older pediatric patients with CHD undergoing cardiac catheterization with conflicting results. In 1 study of 30 children, propofol reduced systemic vascular resistance causing increased right to left shunting in patients with intracardiac shunts.\(^\text{52}\) A second study showed that although both pulmonary and vascular resistances were reduced, propofol use did not alter the intracardiac shunt.\(^\text{53}\) Further study is needed in neonates.

### SUMMARY

Neonates postoperative from cardiac surgery are susceptible to multiple life-threatening complications and should receive rapid triage and assessment when presenting to the emergency department. Cyanosis in a neonate with a median sternotomy incision should raise a high suspicion for an obstructed systemic to pulmonary shunt and prompt emergent echocardiography. Comprehensive investigation should be undertaken for fever in this population, and the pulse oximeter is a valuable tool for diagnosing pulsus paradoxus in the postoperative neonate with suspected cardiac tamponade. Finally, careful consideration must be given to the initiation of positive pressure ventilation and optimal medications when airway management is required. The care of these neonates necessitates a multidisciplinary approach with early consultation with cardiologists and cardiothoracic surgeons.

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### REFERENCES


