Pediatric Postoperative Cardiac Care

George Ofori-Amanfo, MBChB*, Ira M. Cheifetz, MD, FCCM

INTRODUCTION

Postoperative care of pediatric cardiac patients has evolved dramatically over the past 2 decades, with significant improvement in survival. These improvements are attributable, at least in part, to improvements in diagnostic modalities, surgical techniques, cardiopulmonary bypass (CPB) support, anesthetic management, postoperative care, and the use of extracorporeal life support to manage postoperative refractory shock.1,2 Despite an overall increase in complexity, mortality has decreased in both short-term and long-term follow-up.3 Several factors have contributed to this improvement in outcome, including advances in prenatal and preoperative evaluation and diagnosis,1,5 anesthetic and intraoperative management,6 and standardized approaches to postoperative care.7–9 For example, recent advances in the surgical and perioperative

KEYWORDS

- Congenital heart disease
- Cardiac surgery
- Cardiac output
- Right ventricle
- Left ventricle
- Pediatric
- Neonatal
- Cardiopulmonary bypass

KEY POINTS

- Postoperative care of cardiac patients requires a comprehensive and multidisciplinary approach to critically ill patients with cardiac disease whose care requires a clear understanding of cardiovascular physiology.
- Finely balanced multiorgan interaction maintained during health can be profoundly deranged in disease states.
- When a patient fails to progress along the projected course or decompensates acutely, prompt evaluation with bedside assessment, laboratory evaluation, and echocardiography is essential.
- When things do not add up, cardiac catheterization must be seriously considered. With continued advancements in the field of neonatal and pediatric postoperative cardiac care, continued improvements in overall outcomes for this specialized population are anticipated.

* Corresponding author.
E-mail address: george.ofori@duke.edu

Division of Pediatric Critical Care Medicine, Duke Children’s Hospital, Duke University Medical Center, DUMC 3046, 2300 Erwin Road, Durham, NC 27710, USA

Crit Care Clin 29 (2013) 185–202
http://dx.doi.org/10.1016/j.ccc.2013.01.003
criticalcare.theclinics.com
0749-0704/13/$ – see front matter Published by Elsevier Inc.
management of congenital heart disease in neonates have allowed early primary repair of cardiac lesions, such as tetralogy of Fallot, with results comparable to delayed repair.\textsuperscript{10}

The growing implementation of multidisciplinary clinical care teams with expertise in cardiac surgery, critical care, cardiology, cardiac anesthesia, neonatology, electrophysiology, nursing, respiratory care, pharmacology, and nutritional support have had a positive impact on the care delivered to these complex patients. The development of these dedicated teams along with improved outcomes in premature and low-birth-weight infants have changed the demographics of patients managed in pediatric cardiac ICUs.\textsuperscript{11}

Optimal postoperative care in the ICU requires an accurate preoperative assessment, use of intraoperative findings and data, careful patient risk stratification, and meticulous anticipatory management. When patients fail to progress along their predicted clinical course, prompt hands-on reassessment at the bedside is essential. In some cases, deviations from the predicted clinical course necessitate urgent cardiac catheterization and angiography for diagnostic and therapeutic purposes.

**PREOPERATIVE ASSESSMENT**

Preoperative assessment of pediatric patients with congenital heart disease is essential to optimal intraoperative and postoperative management. The goal of preoperative evaluation is to compile data in a systematic fashion to aid in the establishment of an accurate diagnosis, a determination of patient risk level, and a preliminary formulation of postoperative expectations.

Congenital heart defects can be complex. The preoperative data should be carefully collected, appropriately organized, and synthesized based on the underlying physiologic problems. Clinical assessment should focus on identifying factors that can be modified to optimize a patient’s preoperative clinical status as well as those that could potentially affect the intraoperative and postoperative courses. The optimal approach to assessing patients with congenital heart disease is influenced by age category (ie, neonate or non-neonate) and underlying physiology (ie, pulmonary overcirculation, impaired systemic output, impaired pulmonary blood flow, or parallel circulations).

For neonatal severe congenital heart disease, infants may present from the delivery room with a prenatal diagnosis, may be transferred from another institution, or may present via an emergency department with cyanosis, respiratory distress, and/or circulatory collapse. In most cases, an intensivist has the opportunity to assess patients and develop an overall plan for postoperative management before surgery. The preoperative evaluation of these infants must focus on the clinical effects of the current physiology and the anticipated impact on intraoperative and postoperative courses.

The situation with non-neonates varies in that these children are often admitted from the operating room (OR) after an elective primary repair or staged palliation, and, thus, an intensivist may not have had the opportunity to meet and assess the patient preoperatively. The intensivist must gather and synthesize preexisting data from the cardiologist and cardiothoracic surgeon for risk stratification and guidance of the postoperative course. A comprehensive and systematic handoff from the OR team is essential to optimal postoperative care.

**Pulmonary Overcirculation**

Cardiac lesions associated with pulmonary overcirculation are marked by intracardiac (eg, atrial septal defect, ventricular septal defect [VSD], and atrioventricular [AV] septal defect) or extracardiac (eg, patent ductus arteriosus) left-to-right shunting. Infants with such lesions are generally acyanotic and their clinical presentation is one of excessive
pulmonary blood flow and congestive heart failure. In this category of lesions, the pulmonary and systemic circulations are not ductus dependent, and these infants generally do not present an acute surgical emergency. Over time, however, they may develop frequent respiratory infections (especially viral) and failure to thrive.

**Impaired Systemic Output**

This group includes patients with obstructive left-sided heart lesions, such as critical aortic valve stenosis, critical coactation of the aorta, and hypoplastic left heart syndrome (HLHS). Systemic output depends on right-to-left shunting through a patent ductus arteriosus; hence, these neonates are prostaglandin (PGE) dependent. In the event of closure of the ductus arteriosus, these infants appear dusky and can rapidly develop acute circulatory shock due to lack of systemic output.

Aortic stenosis is one of the few congenital heart lesions that present as an acute surgical/interventional catheterization emergency. Critical aortic valve stenosis is only treatable by urgent relief of the obstruction, which, in most cases, can be accomplished by cardiac catheterization. In the situation of critical aortic valve stenosis, PGE infusion provides systemic blood flow; however, profound left ventricular (LV) hypertrophy and elevated LV end-diastolic pressure impede coronary perfusion, potentially resulting in poor LV function.

**Impaired Pulmonary Blood Flow**

Lesions in this category include pulmonary atresia or severe pulmonary stenosis. A PGE infusion is required to provide pulmonary blood flow. For infants with pulmonary atresia and an intact ventricular septum, cardiac catherization is usually required to define the coronary anatomy before determining a surgical plan.

**Parallel Circulation**

The prototype congenital heart lesion in this category is transposition of the great arteries. In this situation, the pulmonary circulation runs parallel to the systemic circulation. As such, the only means of delivering oxygenated blood to the systemic circulation is by intracardiac mixing. Inadequate mixing presents as profound cyanosis that may require an urgent balloon atrial septostomy.

**HISTORY, PHYSICAL EXAMINATION, AND LABORATORY TESTING**

Regardless of physiology and patient age, preoperative assessment requires a complete review of the cardiac and noncardiac history, a thorough physical examination, laboratory tests, ECGs, echocardiograms, and/or other cardiac imaging as available and clinically indicated, including cardiac catheterization, angiograms, and MRI. The critical care team must have an accurate understanding of the anatomic diagnoses and cardiac physiology as well as a clear outline of issues that are likely to have an impact on the postoperative course.

The history and physical examination should ascertain and assess each patient’s cardiopulmonary status, including any comorbid conditions. A detailed review of systems, past medical history, and surgical history is necessary for appropriate risk stratification and planning for the postoperative period. A recent intercurrent illness, especially respiratory infection, can be a manifestation of chronic pulmonary overcirculation and can prolong postoperative mechanical ventilation and ICU length of stay. A history of clinically significant arrhythmias can be an additional risk factor for patients in the postoperative period. Although there are no data indicating that postoperative events repeat themselves, knowledge of events of prior surgeries, such as
postoperative arrhythmia, chylous effusions, and unexplained pulmonary hypertensive crises, is important.

Genetic defects and syndromes/dysmorphisms may have important clinical implications for the perioperative course. For example, Down syndrome is associated with a significant risk of upper and lower airway anomalies, endocrinopathies, hematologic derangements, and immune deficiencies.\textsuperscript{12–14} Patients with conotruncal anomalies may also have chromosome 22q11 deletion and its associated T-cell dysfunction and derangements in calcium metabolism.\textsuperscript{15}

As a minimum, laboratory evaluation should include routine complete blood cell count and serum electrolytes. A high white blood cell count may indicate infection. The hemoglobin level may provide valuable information as to how well a patient is responding to chronic cyanosis, and an adequate platelet count is important for patients undergoing CPB. Electrolyte derangements and acid-base disturbances may be a reflection of the effects of preoperative medications, such as diuretics. Although these pieces of information are necessary for preparing patients for surgery, they may also have implications for the cardiac anesthesiologist. Additional laboratory tests, such as coagulation profile and liver function panels, may be dictated by a patient’s clinical status and institutional practice.

Chest radiography is necessary to assess the cardiac silhouette and lung fields at baseline. An ECG is an important part of the preoperative evaluation to demonstrate a patient’s cardiac rhythm as well as to assess conduction intervals, QRS duration, and T-wave morphology. ECGs may be helpful to identify the presence of subtle arrhythmias, such as first-degree or second-degree heart block.

**Echocardiography and MRI**

Echocardiography is an essential imaging tool in diagnosing congenital heart disease. Recent advances in technology, including Doppler measurements, 3-D echocardiography, and strain rate imaging, have enhanced the diagnostic accuracy and allowed prenatal diagnosis of cardiac disease as early as the first trimester of gestation.\textsuperscript{16,17} In addition to the anatomic diagnoses, physiologic information can be inferred from an echocardiogram, but intensivists must be aware of the limitations of these data. For instance, right ventricular (RV) volume overload should be carefully assessed if the RV is compared with an underfilled LV. The RV pressure estimate from the jet of tricuspid regurgitation requires an optimal Doppler envelope and a fair estimate of right atrial (RA) pressure, because the number obtained is a pressure gradient between the RV and the RA. When assessing a pressure gradient across an obstruction via echocardiography, it must be remembered that the gradient is flow dependent. For example, the pressure gradient across the aortic valve is dependent on the stroke volume and myocardial contractility. Pressure gradients measured on the venous side of the cardiovascular system and at the interatrial level may be low yet have significant clinical implications.

Furthermore, echocardiography also has some limitations in assessing myocardial function. Although the assessment of ventricular systolic function using volumetric measurements of the LV is accurate, the complex geometry of the RV makes assessment of function challenging.\textsuperscript{18}

In spite of the limitations, echocardiography indicates cardiac situs, intracardiac anatomy, myocardial function, AV and semilunar valve function, and anatomy of the great vessels. It also assesses the pulmonary and systemic venous systems and in the prenatal stage can be used in the diagnosis of arrhythmias. Echocardiography, transthoracic and/or transesophageal, is an essential tool throughout the perioperative period.
In recent years, MRI and magnetic resonance angiography have played an increasingly important role in the noninvasive evaluation of cardiac patients. Because most patients undergoing cardiac MRI have had prior echocardiograms, the cardiac MRI can be targeted and able to characterize the precise location and anatomic severity of primary cardiac lesions. Also identifiable are associated defects and the functional consequences of the primary lesions. For example, cardiac MRI can assess ejection fractions as an indicator of biventricular function. Thus, regurgitant fraction across valves and Qp:Qs in shunt lesions can be calculated.\textsuperscript{19} CT scan with angiography can also be used in the preoperative evaluation, but its use has been limited by the high doses of radiation required and the increasing availability of MRI.

**Cardiac Catheterization and Angiography**

Due to advances in noninvasive imaging modalities, diagnostic cardiac catheterization is no longer indicated in the routine preoperative evaluation of most congenital heart defects. It is used in circumstances, however, in which the anatomy of the congenital heart disease is inadequately defined by noninvasive means or in cases where specific anatomic details or hemodynamic data, such as pulmonary vascular resistance (PVR), are necessary to optimize surgical management.\textsuperscript{20} When cardiac catheterization has been performed, intensivists must know the hemodynamic data, in particular any information that may have an impact on a patient’s physiology and postoperative course. For example, pulmonary venous desaturation may suggest the presence of lung disease. Qp:Qs may provide an objective measure of pulmonary overcirculation. Pulmonary hypertension can be more accurately measured and diastolic function can be better assessed using ventricular end-diastolic pressures.

**POSTOPERATIVE PERIOD**

The intensivist and other members of the critical care team assume the postoperative management after a patient has been successfully transitioned to the ICU environment. This transition includes full monitoring on the ICU (not transport) devices, appropriate support of the respiratory system without manual ventilation, and medication infusions administered via the appropriate ICU equipment. A systematic and comprehensive surgical and anesthesia handoff is essential to optimal patient management.

A comprehensive OR to ICU handoff should include patient history; airway and anesthetic management (including the most recent doses of anesthetic, paralytic, and antimicrobial agents); and detailed description of operative findings, the repair procedure, intraoperative complications, CPB time, cross-clamp time, deep hypothermic circulatory arrest time, presence of intraoperative arrhythmias, and current infusions (eg, vasoactive agents, inotropes, and sedatives/analgesics). Difficulties with airway management, such as a difficult intubation, and problems with myocardial protection must be clearly communicated to the ICU team. Specific cardiac data to be communicated from the conclusion of surgery include systemic arterial pressure, central venous pressure required to maintain a targeted systemic blood pressure, pulmonary arterial pressure, if measured, and systemic oxygen saturation. Vasoactive medication usage, arrhythmias, and atrial or ventricular pacing are also critical components of the handoff. The results of an intraoperative echocardiogram (generally transesophageal) should be reported, including the results of the repair, any known residual defects, and ventricular function. An example of a scripted postoperative handoff sheet is provided in \textit{Fig. 1}.

Postoperative assessment requires a complete initial physical examination followed by regular, focused examinations dictated by a patient’s clinical condition. Laboratory
data include blood gas analyses, serum electrolytes and glucose, complete blood count, coagulation profile, serum lactate, chest radiograph, and a 12-lead or 15-lead ECG. Preoperative, intraoperative, and postoperative data should be used to determine a patient’s risk level and to help direct the postoperative management plan. The Risk Adjustment in Congenital Heart Surgery and the Aristotle basic complexity scoring systems have been used to evaluate risk of mortality, morbidity, and quality of care in patients undergoing congenital heart surgery. Although these tools are relevant in the care of cardiac ICU patients, they are designed to describe populations of patients and not individual patients. The intensivist’s stratification should aim to group each patient based on the following categories: potential for early extubation, risk of major postoperative complications, and risk of prolonged ICU and/or hospital admission. These risk strata not only create a sense of awareness of patient acuity but also help direct clinical efforts toward mitigating risk factors and improving outcomes.

Early extubation after CPB has been shown to be safe, but the practice requires strong collaboration among the ICU, cardiac surgery, and cardiac anesthesia teams. Patients must be carefully selected to minimize the risk of reintubation, which may lead to increased morbidity and an increased total length of mechanical ventilation. Younger age, longer CPB time, and increased inotrope requirement are risk factors for failed early extubation.

The risk for postoperative complications may be anticipated based on a patient’s complexity of diagnoses and surgical repair, presence of intraoperative complications or pulmonary hypertension, excessive bleeding, refractory arrhythmias, and the presence of low cardiac output syndrome (LCOS). Those with complications have a higher risk of cardiac arrest and may have a tendency to drift into the prolonged hospitalization risk category. ICU management should focus on preventing this adverse drift.
Patients at risk for chronic hospitalization tend to be those who develop complications, such as persistent chylous effusions, thrombosis of major vessels, airway problems (eg, vocal cord and diaphragmatic compromise), and difficulty feeding.

**Postoperative Monitoring**

The goals of postoperative monitoring are to establish an objective assessment of each patient’s overall clinical status, predict potential adverse events, and guide proactive management. The level of monitoring is dictated by complexity of diagnoses, surgical repair, and hemodynamic and respiratory data. All patients should have continuous ECG monitoring, invasive or noninvasive blood pressure monitoring, and respiratory monitoring, including pulse oximetry. Assessment of urine output (with or without a Foley catheter) is essential in the immediate postoperative period. In mechanically ventilated patients, other noninvasive respiratory data can be obtained, including end-tidal carbon dioxide, carbon dioxide elimination, dead space ventilation ($V_t/V_r$ ratio of physiologic dead space over tidal volume), respiratory compliance, and airway resistance. The use of capnography is steadily increasing.

Cerebral near-infrared spectroscopy is gaining popularity in postoperative management. Trends in cerebral oximetry for individual patients may be a helpful marker of alterations in cardiac output, and the use of this approach has been suggested as a predictor of outcome after cardiac surgery.27–29

Central venous pressure monitoring is standard for most patients who have undergone CPB. This can be monitored continuously through a percutaneously placed central venous line or a surgically placed transthoracic line. RA pressure monitoring provides a continuous assessment of filling pressures so that low RA pressures in hypotensive patients may suggest the need for fluid resuscitation. Elevated RA pressures may be an indicator of cardiac tamponade resulting from a pericardial effusion, poor RV function/compliance, or acute pulmonary processes, such as pneumothorax.

Indirect assessment of cardiac output can be determined by measuring mixed venous saturation ($SvO_2$) on a specimen drawn from the RA. The arteriovenous oxygen difference ($\text{AVDO}_2$) may reflect cardiac output. A difference of approximately 25% suggests a normal output, and this is applicable for patients with mixing lesions with systemic oxygen desaturation as well. Patients with left to RA level shunts have elevated $SvO_2$, and the $\text{AVDO}_2$ may not necessarily reflect cardiac output. LA and pulmonary artery lines are seldom used in the current era of cardiac critical care, but LA lines can provide useful objective data in the management of patients with LV dysfunction, mitral valve disease, and/or abnormalities in coronary artery perfusion.

**Low Cardiac Output Syndrome**

LCOS is a common postoperative complication after CPB and occurs within the first 6 to 12 hours after surgery. It is reported in approximately 25% of patients undergoing CPB for congenital heart surgery and has been defined by a constellation of signs and symptoms of low cardiac output state: tachycardia, poor peripheral perfusion, and oliguria. LCOS requires increased inotropic support and may result in cardiac arrest.30

The factors believed to account for LCOS are hemodynamically significant residual lesions, myocardial dysfunction probably resulting from prolonged periods of cardioplegia, myocardial ischemia, and reperfusion injury. Other factors include inflammatory response to CPB, with a resulting increase in systemic vascular resistance, PVR, capillary leak, and pulmonary dysfunction.31,32 The risk of LCOS is greatest among neonates undergoing complex surgeries. Additional risk factors are prolonged CPB time, prolonged cross-clamp time, preoperative circulatory collapse, and
preoperative ventricular dysfunction. The hallmarks of management beyond prevention are careful anticipation and aggressive cardiorespiratory treatment.

Patients should be critically evaluated, and the evaluation should start with a focused physical examination to identify a cause. For instance, physical examination may reveal murmurs consistent with a residual VSD or AV valve regurgitation. This is followed by laboratory evaluation to help make the diagnosis and to discern the clinical repercussions of the LCOS, such as worsening acidosis and resultant end-organ dysfunction. Additional tests, such as an echocardiogram, may be necessary. Diagnosis of residual lesions is often accurately made in the ICU, but their absolute contribution to a patient’s clinical deterioration may be difficult to ascertain. Therefore, the intensivist may need to err on the side of being more aggressive, which may include cardiac catheterization with angiography.

After the evaluation, the cause of LCOS must be stratified into 2 main categories: those requiring surgical intervention and those amenable to medical therapy. For instance, neonates who undergo tetralogy of Fallot repair generally do not tolerate a significant residual VSD, and infants who have undergone AV canal defect repair generally do not tolerate a persistent patent ductus arteriosus. These clinical scenarios present significant hemodynamic problems with refractory LCOS and often require urgent reoperation.

Therapy for LCOS should be individualized and a patient’s response to interventions closely monitored. Medical therapy is geared toward the perceived cause but all patients must receive adequate fluid resuscitation to maintain preload and systemic blood pressure followed by appropriate use of inotropic agents to support myocardial contractility and afterload, reducing agents to decrease ventricular work load, enhance cardiac output, and improve perfusion. Although the PRIMACORP study has proposed the use of milrinone prophylactically against postoperative LCOS, there are institutional variations in agents used to prevent or treat LCOS, including low-dose epinephrine alone or in combination with milrinone. Several studies have shown drugs, such as milrinone, dopamine, and epinephrine, to have significant arrhythmogenicity in the postoperative period.

In the absence of a surgical cause, when LCOS remains refractory to medical therapy, mechanical support should be considered. Extracorporeal membrane oxygenation (ECMO) has been used with good results in postoperative cardiac patients, including those placed on ECMO for an inability to wean from CPB and those with severe hemodynamic instability with refractory LCOS, and as rescue from cardiac arrest. Because of the associated morbidity, timing of ECMO poses a major decision challenge. Therefore, early engagement of the multidisciplinary team, involving critical care, surgery, anesthesiology, and cardiology, is crucial. Any delay in initiation of extracorporeal life support may have grave repercussions for patients.

**Pulmonary Hypertension**

Elevated PVR and resultant pulmonary arterial hypertension (PAH) is a common postoperative complication after congenital heart surgery. PAH can acutely elevate RV afterload with resultant RV dysfunction and is a common cause of cardiac arrest in the postoperative period. Several factors contribute to its development, including CPB, which is associated with a systemic inflammatory response syndrome, involving mediators, such as interleukin 6, interleukin 10, tumor necrosis factor α, P-selectin and E-selectin, leptin, soluble intercellular adhesion molecule and vascular cell adhesion molecule, and fractalkine. Patient factors that contribute to pulmonary hypertension include cardiac physiology, comorbid conditions, and some genetic syndromes.
The cardiac physiologies most at risk for development of PAH are

1. Those associated with an increased pressure load to the pulmonary arterial system, such as truncus arteriosus communis, VSD, AV canal defect, aortopulmonary window, and patent ductus arteriosus
2. Those associated with impaired egress of blood from the pulmonary arterial tree (eg, obstructed total anomalous pulmonary venous connections), mitral valve stenosis, or restrictive atrial communication in cases of HLHS
3. Heart transplant patients with preexisting pulmonary hypertension (eg, restrictive cardiomyopathy)

Comorbid conditions, such as congenital diaphragmatic hernia, may also pose an independent risk to the development of PAH, and some genetic syndromes, in particular Down syndrome, can also be a risk factor.

The postoperative implication of PAH is pulmonary vascular reactivity. In this setting, a vasospastic stimulus can trigger a potentially lethal episodic pulmonary hypertensive crisis that can result in acute RV failure, tricuspid regurgitation, decreased cardiac output, and myocardial ischemia. The initial approach to postoperative pulmonary hypertension is prevention. Noxious stimuli must be minimized. For example, in a mechanically ventilated patient, endotracheal suctioning should be performed carefully. This might mean limiting suctioning to the tip of the endotracheal tube and administering additional sedation/analgesia/neuromuscular blockade in labile patients. Hypercarbia should be avoided, and supplemental oxygen should be used judiciously for its pulmonary vasodilatory benefit.

Inhaled nitric oxide decreases vascular tone and is an effective agent in the treatment of pulmonary hypertension in the postoperative period. Although its prophylactic use remains controversial, preemptive use should be considered in those critically ill patients who are less likely to tolerate an acute decompensation. Other agents used to treat postoperative pulmonary hypertension include inhaled illoprost and intravenous sildenafil.

Postoperative Arrhythmia

Postoperative arrhythmias, with a reported incidence of 15% to 50%, can cause significant hemodynamic compromise. Although most arrhythmias are clinically unimportant, junctional ectopic tachycardia (JET), reentrant supraventricular tachycardia, ectopic atrial tachycardia (EAT), and ventricular tachycardia, when they occur, can result in prolonged mechanical ventilation, increased inotrope use, prolonged ICU length of stay, increased risk of cardiac arrest, and decreased survival. Risk factors for the development of tachyarrhythmias include younger age at surgery, long CPB and cross-clamp times, and use of deep hypothermic circulatory arrest. Ventricular tachycardia, although not common in the postoperative period, can lead to rapid hemodynamic compromise. It presents as a wide complex tachycardia that must be rapidly differentiated from an aberrantly conducted supraventricular tachycardia.

Accurate diagnosis of the arrhythmia is paramount to optimal management. Narrow complex tachycardias are classified into automatic or reentrant rhythm. Reentrant arrhythmias have sudden onset and respond to pharmacologic agents (eg, adenosine) or electrical cardioversion. They also respond to overdrive pacing and have the characteristic of abrupt termination. The automatic arrhythmias (JET and EAT) demonstrate warm-up and cool-down phenomena (ie, slow increase and slow decline in heart rate); they are catecholamine responsive and do not respond to overdrive pacing or cardioversion. A 12-lead or 15-lead ECG with rhythm strip may be necessary to
make the diagnosis, and in some cases an atrial electrogram is necessary to identify the location of P waves.

Although there is some suggestion of prophylactic amiodarone for prevention of postoperative JET, the hallmark of prevention of JET and other postoperative tachyarrhythmia is aggressive repletion of electrolytes and treatment of significant acid-base disturbances. In cases of the automatic arrhythmias, minimizing a patient’s catecholamine state (eg, avoidance of fever, instituting appropriate sedation, when indicated, and adequate neuromuscular blockade) may decrease risk of arrhythmia. Once arrhythmia occurs, the treatment algorithm largely depends on whether a patient is hemodynamically stable or unstable.

Fig. 2 provides a suggested diagnostic algorithm and a guide to the approach to therapy. Ventricular tachycardia is managed per the Pediatric Advanced Life Support algorithm. supraventricular tachycardia with aberrant conduction may appear, electrographically, similar to ventricular tachycardia. The type of supraventricular tachycardia needs to be established and treated appropriately. The recommended treatment of stable reentrant tachycardia is vagal maneuvers, adenosine, or β-blockers. Unstable patients should be treated with adenosine (if it can be administered promptly) or synchronized cardioversion. For EAT, β-blockers (eg, esmolol) are effective initial therapy. For JET, the traditional therapeutic modalities should be implemented: cooling to approximately 36°C, decreasing catechol infusions as tolerated, adequate sedation, and appropriate neuromuscular blockade. Cooling must be cautiously performed because shivering can cause a catecholamine surge and counteract the therapy. Amiodarone is a suggested first-line pharmacologic therapy for JET. It can be effective, but its dose-related adverse effects (ie, α-blockade) should be considered.

Bradyarrhythmias encountered in the postoperative period include sinus bradycardia, as seen in sick sinus syndrome, and varying degrees of AV node block. These
respond well to pacing using temporary pacing wires placed at the time of surgery. In isolated sick sinus syndrome, atrial pacing alone is adequate. AV node block often requires AV sequential pacing.

**Respiratory Management**

Positive pressure ventilation may have major influences on hemodynamics after congenital heart surgery. Patients most affected are those with significantly impaired myocardial function, pulmonary vascular disease, and passive pulmonary blood flow (e.g., bidirectional Glenn or Fontan circulation). The key to optimal mechanical ventilation is to adopt strategies to achieve adequate gas exchange while minimizing the adverse effects on hemodynamics.

In terms of postoperative respiratory management, the increasingly common approach is early extubation. Advantages of early extubation include minimizing the need for postoperative sedation, reducing the length of ICU admission, reducing the incidence of nosocomial infection, and improving family and patient satisfaction. A discussion regarding the candidacy for early extubation should be made in consultation among the intensivist, cardiothoracic surgeon, and anesthesiologist.

Once extubated, most patients require supplemental oxygen via a standard nasal cannula. Others, however, require increased support including high-flow nasal cannula (HFNC) and noninvasive ventilation (NIV), whereas a small subset requires reintubation. Although a comprehensive discussion of high-flow oxygen therapy and NIV is beyond the scope of this article, a few key points are provided.

High-flow nasal cannula is an increasingly common approach to the management of patients who require mild-to-moderate respiratory support. Although definitive data are lacking in the pediatric population, there is growing evidence that such an approach is well tolerated with minimal adverse effects. The concept underlying this approach is to provide patients with an elevated flow of oxygen-enriched, warmed, and humidified gas. Flow rates of up to 8 L per minute and 40 L per minute are used for infants and children, respectively.

Multiple mechanisms of action have been proposed for the beneficial effects of high-flow nasal cannula therapy. The increased flow of gas provides pressure to the airways, similar to a continuous positive airway pressure approach. Data show, however, that the pressure generated by HFNC is lower than with continuous positive airway pressure. Thus, this mechanism alone is unlikely to be the sole benefit. Other proposed mechanisms of action include the beneficial secretion clearing and positive mucosal effects of heated and humidified gas, the washout of deadspace by the continuous flow of gas to the upper airways, and the provision of inspiratory gas flow that more closely approaches that of spontaneous respiration, thus minimizing the entrainment of room air. Further investigation on the mechanisms of action for HFNC therapy is under way.

As an intermediate step between HFNC and invasive mechanical ventilation, NIV can be considered. NIV involves the administration of airway pressure at either single-level (continuous positive airway pressure) or bilevel positive airway pressure without the need for intubation. The advantage of NIV focuses on the lack of an endotracheal tube, which is associated with a reduced need for pharmacologic sedation and a reduction in the incidence of ventilator associated pneumonia. The downside of NIV in the pediatric population is a lack of Food and Drug Administration–approved devices and interfaces to efficiently provide this respiratory support for all infants and children.

**Cardiorespiratory Interactions**

When management of postoperative patients requires the use of positive airway pressure (regardless of modality), the cardiorespiratory effects of such an approach must
be considered. Positive airway pressure by definition increases the mean intrathoracic pressure. If the increase in mean intrathoracic pressure is significant, systemic venous return can be reduced due to a decreased pressure gradient between the superior and inferior vena cava and the RA. The result is decreased RV output, and such a situation can be corrected by volume augmentation. The volume required is approximately 5 mL/kg, which can be repeated as clinically indicated.

This physiologic effect is most prominent in those patients with passive pulmonary blood flow (eg, bidirectional Glenn shunt or Fontan shunt), who rely on a low pulmonary artery pressure to establish a flow gradient between the systemic and pulmonary venous systems. In these patients, either high mean airway pressure or atelectasis can adversely affect venous return and cardiac output by compromising the flow gradient. Although there is often hesitation to use positive end-expiratory pressure in such patients, avoidance of positive end-expiratory pressure could cause atelectasis with untoward hemodynamic consequences.

The effects of mechanical ventilation on PVR and RV afterload are variable and dependent on lung volume. PVR and RV afterload are optimized at an optimal lung volume, approximating functional residual capacity. Hyperinflation increases PVR by alveolar overdistension and subsequent compression of perialveolar capillaries. Pulmonary hypoinflation, alternatively, causes lung collapse and elevated PVR from hypoxia-induced pulmonary vasoconstriction and increased impedance of the large pulmonary vessels. Therefore, in patients with RV dysfunction, in whom elevated PVR adds further stress to the RV, it is crucial to ventilate with settings that prevent overdistension and avoid atelectasis.

In terms of the LV, the preload effects are variable and principally dependent on the effects of mechanical ventilation on the RV. This exemplifies the concept of ventricular interdependence. The effects of mechanical ventilation on LV afterload are clearer. The mean intrathoracic pressure generated by positive pressure ventilation reduces LV afterload by reducing the LV transmural pressure. Thus, mechanical ventilation can be an effective approach to patients with LV dysfunction. As an example, patient status postreimplantation of anomalous left coronary artery from the pulmonary artery will likely benefit from mechanical ventilation. Similarly, in patients with poor LV function, clinicians should anticipate a possible deterioration in hemodynamics after extubation. Furthermore, mechanical ventilation may benefit patients with ventricular dysfunction/failure by reducing the respiratory work of breathing (ie, reducing oxygen consumption).

**Cardiac support**

Cardiac support for postoperative patients with congenital heart disease must be directed by patient pathophysiology, including an assessment of changes in loading conditions (described previously). Clinicians must consider the effects of the various inotropes/vasoactive agents available in relation to patient physiology. The physiologic balance most commonly involves reducing the afterload to the ventricles, optimizing ventricular contractility, and providing an appropriate perfusion pressure for both ventricles. Additionally, it should be acknowledged that with the lack of definitive data showing that any one approach is more beneficial than another, institutional preference often plays a large role in clinical decision making. A common approach for postoperative management includes the use of milrinone as an inodilator with supplemental low-dose epinephrine as clinically indicated.

**Inhaled nitric oxide**

For those patients with significant RV dysfunction and/or pulmonary hypertension, inhaled nitric oxide may represent a beneficial approach. As a selective pulmonary
vasodilator, inhaled nitric oxide reduces PVR and may benefit those patients with pulmonary hypertension. More controversial is the use of inhaled nitric oxide for patients with normal PVR and RV dysfunction/failure. Some data suggest that the administration of inhaled nitric oxide despite a normal pulmonary vasculature may improve RV performance.

**Special Considerations in the Postoperative Period**

Although it is impossible to comprehensively describe all clinical considerations in a review article, a few special considerations should be discussed. One example is patients with significant chest tube or surgical site bleeding refractory to blood product administration, including platelets, cryoprecipitate, and fresh frozen plasma. Once the platelet count and clotting parameters have been corrected (ie, medical bleeding) and active bleeding continues, a surgical reason must be considered. Close communication with the surgeon throughout such a process is important.

A second group requiring reoperation is shunt-dependent patients with an acute shunt occlusion (eg, thrombosis). With shunt-dependent pulmonary blood flow, an acute desaturation without clear explanation and resolution requires prompt evaluation of the shunt by echocardiography. Once shunt malfunction or occlusion is suspected, evaluation and therapy must be initiated promptly and concurrently. Appropriate therapy includes aggressive fluid administration and heparin bolus followed by continuous infusion. Unless shunt occlusion/malfunction is conclusively ruled out with the initial evaluation, urgent cardiac catheterization with intervention or surgical exploration is the appropriate next step. The effects of profound cyanosis, acidosis, aggressive fluid resuscitation, and/or high doses of pharmacologic cardiovascular agents may have a negative impact on clinical status and elevate the surgical risk for the reoperation.

Special consideration should also be provided for cardiac patients undergoing noncardiac surgery. This population presents unique preoperative and postoperative issues that require careful consideration and assessment. A key example is a single ventricle patient who has undergone stage I palliation with a Blalock-Taussig shunt who may be at risk for acute shunt thrombosis from dehydration. The preoperative and intraoperative management must include careful consideration of this complex physiology. Preoperative intravenous hydration while patients are withheld food and fluids is essential. Also, close consideration should be given to obtaining a preoperative echocardiogram. Careful attention to hydration status and perfusing pressures is essential throughout the perioperative period. Also, during the intraoperative course, abdominal insufflation during laparoscopic gastric tube placement can increase systemic vascular resistance and compromise cardiac output. Support of the function of the single ventricle as clinically indicated is essential.

**EXTRACORPOREAL MEMBRANE OXYGENATION**

ECMO can be life saving for patients with refractory cardiac and/or respiratory failure, including during the postoperative period. The decision to proceed with ECMO support is subjective and must be made in conjunction with a patient’s clinical course and trajectory. It should be stressed that ECMO does not correct cardiac and/or respiratory failure but is rather a bridge to recovery, transplantation, or decision making. In cases of planned cardiac transplantation due to a failure of ventricular recovery, ECMO can be transitioned to a ventricular assist device as a bridge to transplantation.

Although ECMO in the postoperative period can be life saving, overall mortality rates for the neonatal and pediatric populations remain suboptimal at 40% and 49%.
respectively (Extracorporeal Life Support Organization Database, July 2012). The optimal timing for ECMO cannulation and the best candidates for ECMO remain controversial.

One of the growing uses of ECMO in the postoperative congenital heart population is in the situation of cardiac arrest (extracorporeal cardiopulmonary resuscitation). Extracorporeal cardiopulmonary resuscitation is the extension of cardiopulmonary resuscitation to ECMO when standard resuscitative efforts are failing. Reported survival with good neurologic outcomes has been reported as 30% to 40%. As ECMO systems become simpler and can be set up more quickly, it is reasonable to expect the use of extracorporeal cardiopulmonary resuscitation to continue to grow. Additional outcomes-based research is needed.

SUMMARY

Postoperative care of cardiac patients requires a comprehensive and multidisciplinary approach to critically ill patients with cardiac disease whose care requires a clear understanding of cardiovascular physiology, multiorgan system function, and organ interactions in health and in disease. When patients fail to progress along the projected course or decompensate acutely, prompt evaluation with bedside assessment, laboratory evaluation, and echocardiography is essential. When things do not add up, cardiac catheterization must be seriously considered. With continued advancements in the field of neonatal and pediatric postoperative cardiac care, continued improvements in overall outcomes for this specialized population are anticipated.

REFERENCES


