Cardiac surgery and post- operative management

Lee JH, Choo J, Nakao M

The overall goal of post-operative cardiac care is to maintain adequate tissue oxygenation and perfusion. Understanding the pre-operative anatomy and physiology, type of surgical procedures performed and general post-operative management principles are essential components in the clinical care of children after cardiac surgery.

Pre-operative assessment

The assessment of the post-operative paediatric cardiac patient begins with a focussed review of the pre-operative state as well as the underlying anatomy and physiology of the cardiac lesion.

Important points to note in the history include:

- Failure to thrive
- Recurrent chest infections
- Airway obstruction
- Significant co-morbidities (e.g., Down syndrome, DiGeorge syndrome)
- Previous cardiac catheter studies, cardiac surgeries and their anaesthetic and post-operative course

The patient's 2D-echocardiograms (transthoracic, transoesophageal) and cardiac catheter studies should be reviewed with particular attention paid to:

- Suitability of a biventricular repair versus single-ventricle palliation
- Presence of obstruction to ventricular outflow or venous return
- Presence of valvular regurgitation or stenosis
- Haemodynamic data from cardiac catheter studies
- Reversibility of high pulmonary vascular resistance

Intra-operative course

Important details to be obtained from anaesthetists and cardiac surgeons upon arrival in the ICU include:

- Details regarding the surgical procedure
- Surgical complications that may have occurred intra-operatively
- Number and type of drains as well as type of pacing wires inserted
- Findings of the immediate intra-operative transoesophageal 2D-echocardiogram (if performed). In particular, it is important to know the cardiac function and the presence of any residual lesions
- Anaesthetic details and any difficulties encountered during induction, intubation and line placement
- Intra-operative ventilator settings and issues
- Haemodynamic details after coming off bypass (rhythm, blood pressure, central venous pressure and pulmonary pressures if pulmonary arterial line is present)
- Presence of arrhythmias, heart blocks and electrocardiograph (ECG) changes during the surgery
- Duration of cardiopulmonary bypass (CPB), aortic cross-clamp time and circulatory arrest time
- Medications given intra-operatively or prior to patient transfer including inotropes, vasoactive drugs, analgesics and diuretics

- Fluids status: Note any drain losses, blood loss during operation and amount of fluids/blood products given.

Effects of CPB and aortic cross-clamp include:

- Systemic inflammatory response (SIR) leading to capillary leak with third space loss of fluids, changes in systemic vascular resistance and reversible acute kidney injury
- Pulmonary endothelial dysfunction
- Myocardial ischemia–reperfusion injury

Generally, SIR is maximal at 18-24 hours after CPB. The longer the CPB and aortic cross-clamp time the more intense the inflammatory response will be and the more likely that the myocardium will require post-operative support. Genetic predisposition as well as pre-operative status such as the presence of cardiac failure also determines the extent of this inflammatory response. Glucocorticoids may be used prior to CPB to help ameliorate the inflammatory effects of CPB.

Investigations

Post operative investigations include:

- arterial blood gas (ABG), lactate
- hypocount
- full blood count
- renal panel
- liver panel
- coagulation profile
- radiographs (e.g., CXR/AXR) looking at tubes and invasive line placements

The frequency of subsequent laboratory investigations will depend on whether the surgery was performed with or without CPB as well as the clinical post-operative course of the child. Commonly, ABG and lactate levels are performed 2-4 hourly during the immediate post-operatively period and the interval can be spaced out according to patient status and recovery.

Post-operative management

General issues

After obtaining pertinent details during the intra-operative course, the child should be examined for:

- General condition looking for pallor, hemodynamic stability and adequacy of tissue perfusion
- Cardiovascular examination for quality of the first and second heart sounds, murmurs, rubs
- Respiratory examination to assess the position of the endotracheal tube and to clinically detect effusions and atelectasis
- Abdominal examination for hepatomegaly, presence of abdominal distention and ascites
- Neurological examination for adequacy of sedation, gross motor examination
- Sites of all indwelling catheters and tubes

Peri-operative antibiotics may include intravenous cefazolin or a combination of intravenous cloxacillin and gentamicin. Pre-operative methicillin-resistant *Staphylococcus aureus* status should be noted.

Ventilation strategies

Cardiopulmonary interactions and the response of certain vascular beds to carbon dioxide tension allow the intensivist to manipulate ventilation to optimize the post-operative care of the patient.

Examples of ventilation strategies for various post-operative scenarios include:

- Pulmonary hypertension: avoid respiratory acidosis and consider use of inhaled nitric oxide
- Bidirectional Glenn/ cavopulmonary shunt: accept a higher P_aCO_2 to allow for higher cerebral and thus pulmonary blood flow

- Low cardiac output state, left ventricular dysfunction: administration of positive pressure ventilation reduces systemic ventricular afterload and decreases the transmural pressure of the left ventricle.
- Right ventricular dysfunction: Prevent/manage lung collapse and avoid over-distension of the lungs.

Haemodynamic assessment

Almost all post-operative cardiac patients will have continuous blood pressure monitoring, central venous lines and sometimes atrial/pulmonary pressure lines. These give useful information with regards to the hemodynamic status and intra-cardiac pressures.

Some common scenarios include:

- High right-sided atrial pressures: right ventricular dysfunction, severe (residual) pulmonary stenosis or regurgitation, tricuspid stenosis, pulmonary hypertension, pericardial tamponade
- High left-sided atrial pressures: systemic ventricular dysfunction, residual left to right shunt, mitral regurgitation, pericardial tamponade

Most patients have some degree of myocardial dysfunction during the immediate post-operative period and will often require inotropic support. In our centre, patients are commonly supported with low dose adrenaline and milrinone. Afterload reduction agents (e.g., nitroglycerin or nitroprusside) may be necessary in certain cases where severe hypertension is detrimental (e.g. repair of coarctation of aorta) and to optimize systemic blood flow in single ventricle repairs.

Fluid management

Fluid regimes during the postoperative period are often center-specific. In our center, the fluid management for post-operative cardiac patients is as follows:

Open heart surgery requiring CPB:

- Operative day: 25ml/kg/day
- Post-operative day (POD) 1: 50ml/kg/day
- POD 2: 75ml/kg/day
- POD 3: 100ml/kg/day

Surgeries not requiring CPB:

- Operative day: 50 ml/kg/day
- POD 1: 75 ml/kg/day
- POD 2: 100ml/kg/day

Additional fluid boluses can be given as required based on changes in fluid status and haemodynamics. Diuretics (e.g. frusemide and spironolactone) may be given when there is a concern with residual pulmonary congestion.

Post-operative bleeding

Drainage from pleural and mediastinal drains should decrease rapidly within the first four hours and should not be persistently draining frank blood.

A simple rule to follow is that volume of drainage should be at maximum:

- 4ml/kg/hr for the 1st hour
- decrease by 1ml/kg/hr every subsequent hour

Any persistent drainage of 4ml/kg/hr beyond the first two hours should be of concern and the cardiothoracic surgeon should be notified.

Supportive measures for post-operative bleeding include blood product support with packed red blood cells, platelets, fresh frozen plasma and cryoprecipitate. Surgical re-exploration may be necessary if bleeding is persistent.

Acid-base status

Serial monitoring of the ABG for adequacy of oxygenation, ventilation and tissue perfusion should be performed. Metabolic acidosis should be looked for as it may be first sign of inadequacy of tissue perfusion. A rising lactate level, if available, may also be indicative of a low cardiac output state. In our centre, metabolic acidosis is often corrected with sodium bicarbonate when the base deficit is greater than 5.

Possible causes of metabolic acidosis should also be sought for concurrently. These include intravascular volume depletion, poor cardiac function, presence of residual lesions and sepsis.

Arrhythmias and pacing

Common arrhythmias following cardiac surgery include:

1. Junctional Ectopic Tachycardia (JET)

This is a common arrhythmia in post-operative cardiac patients, especially if the surgery involves the ventricular septum. Maintaining atrio-ventricular (AV) synchrony is vital to optimize cardiac output and oxygen delivery. The most useful investigation for JET is the atrial ECG. The can be obtained by connecting the external atrial pacing wires to the ECG electrodes.

Features suggestive of JET:

- narrow-complex tachycardia
- Rate is usually greater than 180/min
- A-V dissociation
- The atrial activity occurs within or immediately after the QRS complex

Management of JET includes:

- Adequate sedation and analgesia
- Minimizing the use of beta-adrenergic agents such as intravenous adrenaline
- Correcting any electrolyte abnormalities such hypokalaemia, hypocalcaemia and hypomagnesaemia
- Correcting hypovolaemia
- Correcting metabolic acidosis if present
- Avoiding hyperthermia, consider active cooling if JET is refractory
- Intravenous amiodarone (contact cardiologist)
- Overdrive pacing (contact cardiologist)

2. Heart block

Heart block can occur in patients who have had surgery around the AV node, including that of ventricular septal defect repair and mitral valve surgery. The type of heart blocks that usually causes haemodynamic problems are second-degree type II blocks and third-degree blocks.

Temporary external pacing may be required and paediatric cardiologists should be consulted on the pacing mode. VVI and AV sequential pacing are most commonly used. It is important to exclude and treat any electrolyte abnormalities in the meantime such as hypokalaemia, hypocalcaemia and hypomagnesaemia.

Sedation, analgesia and neuromuscular blockade

All post-operative patients should receive adequate pain relief. Intravenous morphine is most commonly used in our unit. Alternatives include intravenous fentanyl. In the immediate post-operative period, intravenous paracetamol is often useful; non-steroidal anti-inflammatory drugs (NSAIDs) can be given when bleeding is not an active concern.

Sedation may be necessary if the intubated patient remains agitated despite adequate pain relief. In our unit, intravenous midazolam is the preferred sedative agent in intubated patients.

Paralysis may be required in certain groups of patients such as those returning from the operating theatre with an open chest or patients with pulmonary hypertensive crises.

Low cardiac output state (LCOS)

It is important to identify LCOS early, search for treatable causes and start the necessary therapy to reverse a potentially deadly vicious cycle.

Signs include:

- Tachycardia
- Poor perfusion
- Widening toe-core temperature difference
- Decreasing urine output
- Metabolic acidosis or rising lactate level on serial ABG readings
- Widened arterial-mixed venous oxygenation difference
- Hypotension (late sign)
- Rising creatinine or liver enzymes level, seizures or other indicators of end-organ damage (late signs)

Important causes to consider include:

- Cardiac tamponade: look for tachycardia, narrowed pulse pressure, elevated central venous pressure (CVP), sudden drop in pericardial drain output suggesting that the pericardial drain could be blocked
- Adequacy of intravascular volume: look for tachycardia, drop in CVP, poor urine output and large variability in arterial pressure waveform with respiratory cycle.
- Arrhythmias/ heart block
- Myocardial dysfunction: consider in a patient with tachycardia, elevated CVP/ left atrial pressure (LAP), worsening acidosis with cool peripheries with no response to fluid challenge.

Issues pertaining to LCOS in specific cardiac lesions are discussed later. General principles for management of LCOS can be classified according to factors that influence cardiac output: preload, myocardial contractility, afterload, heart rate and rhythm. These are outlined below.

1. Preload:

Optimization of intravascular volume is essential. In our unit, 5% albumin is most often used. Other causes of decreased preload should also be considered such as blood loss and these should be addressed accordingly. Preload adjustments should be monitored with changes in atrial pressures as well as ventricular response (clinical assessment of heart rate, changes in blood pressure, perfusion, toe-core temperature difference, central venous pressures and urine output).

2. Contractility:

Often after CPB, manipulation of preload is not enough to address LCOS. The use of inotropic support in post-operative patients is common. Judicious use of inotropes such as adrenaline is often useful to manage the depressed myocardial function after CPB.

3. Afterload reduction:

Milrinone, a phosphodiesterase III inhibitor, is a unique inotropic agent because of its vasodilatory and lusitropic effect. Other afterload reducing agents such as nitroglycerin and nitroprusside may be indicated when systemic blood pressure is high and cardiac output is deemed to be low or normal.

4. Heart rate and rhythm:

Certain post-operative cardiac patients, especially neonates, often depend on a sufficient heart rate for cardiac output. As such, in cases of heart block or relatively slow heart rate, temporary pacing is required. Control of arrhythmia is also important to ensure that cardiac output is not compromised in these cases.

5. **"Pop off valve":**

In certain cases where significant RV dysfunction (e.g., tetralogy of Fallot with a severe hypertrophy of the RV) is anticipated, preserving or creating a right to left shunt at the atrial level is beneficial to ensure adequate cardiac output (by optimizing preload of the left ventricles) while accepting transient desaturations.

Lines and drains

All central lines should be removed as soon as they are not required in order to minimise line-related infections. Drains should be removed once drainage is minimal and after consultation with the cardiac surgeons.

A chest radiograph should be obtained after removal of a chest drain to look for any intra-thoracic air collections and/or pleural fluid re-accumulation.

Anti-coagulation

For Blalock-Taussig shunts (BTS), bidirectional cavopulmonary connections (BCPC) and Fontan procedures, heparin infusion may be started 4-6 hours post-operatively if the coagulation profile is not significantly deranged and there is no significant surgical bleeding/ drain losses. The surgeons should be consulted prior to starting of heparin in these patients. The usual starting dose is 10 units/kg/hour of intravenous heparin, and titrated to therapeutic goals of anti-coagulation based on the hospital's anti-coagulation guidelines. Loading doses of heparin (eg. 10-25 units/kg) may be required to reach therapeutic targets, this should be discussed with the surgeons and intensivists.

Specific post-operative cardiac lesions

Residual left-to-right shunts

Residual left-to-right shunts can occur in surgery that involves the repair of septal defects, where pre-operative shunts are left unrepaired or unrecognized. These can lead to increased pulmonary blood flow (leading to pulmonary oedema, pulmonary hypertension and volume overload of the systemic ventricle).

Small residual shunts often do not need further management. However, a significant residual lesion predisposes to congestive cardiac failure and may require prolonged course of diuretics with consideration given for the need for further surgery to address the residual lesions.

Residual systemic ventricular outflow tract obstruction

These should be looked for in surgeries involving left outflow tract obstruction such as repair of subaortic stenosis, aortic stenosis and coarctation of aorta. These can limit cardiac output.

Persistent outflow tract obstruction may require cardiac catheter intervention such as balloon angioplasty or a second surgery. It is important to be careful with afterload reducing agents in cases where fixed outflow tract obstruction is present.

Atrio-ventricular valve dysfunction

These may occur after valve repair or when closure of a septal defect results in valvular stenosis. These can limit cardiac output and may require further surgical intervention if severe.

Medical management of AV valve insufficiency is focused on afterload reduction. AV valve stenosis is usually not amenable to medical management.

Ventricular diastolic dysfunction

Diastolic dysfunction is a common complication after any surgery where there is significant ventricular hypertrophy. This is especially true for surgery such as a correction of Tetralogy of Fallot.

The atrial pressures are elevated in diastolic dysfunction. In worsening diastolic dysfunction, there will be a need for higher ventilator support with higher venous pressures leading to pleural effusions and ascites.

Management of diastolic dysfunction includes:
- Maintaining adequate preload
- Infusion of inotropic agents with afterload reducing properties

Pulmonary hypertension
This is a potential problem in patients with long-standing pre-operative left to right shunts, pulmonary venous congestion, mitral valve disease and those who already demonstrate pre-operative pulmonary vascular changes.

Signs of pulmonary hypertension post-operatively include:
- cyanosis (if there's a residual systemic-to-pulmonary shunt)
- acute right ventricular failure (with acute increase in right atrial pressure)
- low cardiac output
- sudden decrease in lung compliance and onset of bronchospasm

If untreated, this can lead to pulmonary hypertensive crises with haemodynamic instability.

Prevention of pulmonary hypertension is the most effective treatment. Management strategies include:
- Adequate sedation and analgesia, paralysis if severe and unstable
- Avoiding hypoxia
- Maintaining an arterial pH of between 7.4 to 7.5 with mild hyperventilation
- Avoiding hyperthermia
- Avoiding lung over-distension or under-inflation
- Pulmonary vasodilators such as inhaled nitric oxide and sildenafil

Single ventricle lesions
Post-operative management of single-ventricle lesions is one of the most challenging aspects of post-cardiac surgery care. The principles behind the first stage of palliation and the second/third stages are different.

In the first stage of palliation, the pulmonary and systemic blood flow is dependent on the systemic vascular resistance, the size of the connection to the pulmonary artery and to a lesser degree pulmonary vascular resistance. The main priority in this group of patients is to maintain an adequate systemic perfusion. Poor systemic perfusion may be due to a low total cardiac output state or a high pulmonary flow to systemic flow ratio (Qp:Qs). On the other hand, cyanosis with preserved haemodynamics may be due to low pulmonary flow to systemic flow ratio or a primary pulmonary problem.

General principles to keep in mind in management of these patients are:
- Maintenance of good total cardiac output
- Best way to manipulate Qp:Qs is with systemic afterload reduction. The largest component of pulmonary resistance is at the site of a pulmonary artery band or shunt.

In the second and third stage of palliation, the pulmonary blood flow is now dependent on non-pulsatile venous flow.

Bidirectional cavopulmonary anastomosis
Pulmonary blood flow in this case is dependent on the resistance of the cerebral and pulmonary vascular bed. The opposite physiological response of these two vascular beds to carbon dioxide,

acid-base balance and oxygen makes management of low arterial saturation challenging. Inhaled nitric oxide may be required in such situation with mild hypoventilation.

In cases where the degree of cyanosis is not severe, expectant management with optimisation of haemoglobin and good hemodynamic support usually suffice as oxygen saturation tend to improve slowly over the first few post-op days.

Fontan circulation

Now, the pulmonary blood flow is dependent on the whole systemic venous return and the pulmonary vascular pressure. Elevated pulmonary pressure is associated with worse outcomes as it will result in high CVP, which will then cause large third-space fluid loss. It is important to exclude mechanical pulmonary artery obstruction or myocardial dysfunction causing high pulmonary pressures.

Use of inotropic agents that do not increase ventricular afterload such as milrinone, dobutamine or low dose adrenaline may be helpful. It is important to maintain adequate functional residual capacity (where pulmonary resistance is the lowest) and tidal volume in these cases as well.

The presence of a fenestration may cause mild systemic arterial desaturation but it is helpful in the situation where high pulmonary pressures are expected during the early post-operative period as it provides a systemic blood flow that is not dependent on passing through the pulmonary circulation.

REFERENCES

1. Bronicki RA and Chang AC. Management of the postoperative pediatric cardiac surgical patient. Crit Car Med 2011; 39: 1974-1984
2. Roth SJ. Chapter 13: Postoperative Care. In: Chang AC. Hanley F, Wernosky G and Wessel DL, eds. Pediatric Cardiac Intensive Care. 1st edition.Lippincott Williams and & Wilkins 1998.
3. Schwartz SM. Cardiac Surgery and Postoperative Management. In: Conway EE Jr, Relvas MS, eds. Pediatric Multiprofessional Critical Care Review. Society of Critical Care Medicine 2006.
4. Wessel DL and Laussen P. Chapter 29. In: Cardiac Intensive Care. In: Fuhrman BP and Zimmerman J, eds. Pediatric Critical Care. 3rd edition. Elsevier 2006.
5. Wessel DL and Fraisse A. Chapter 70A: Postoperative Care of the Pediatric Cardiac Surgical Patient: General Considerations. In: Nichols AG, ed. Roger's Textbook of Pediatric Intensive Care. 4th Edition. Lippincott Williams & Wilkins 2008.