

# Chapter 35

## Principles of Postoperative Care



Lynze Franko and Kenneth Shelton

### Assessment and Goals for the Immediate Postoperative Period [1–4]

- The first step when the ICU receives a patient from the operating room (OR) is connecting the patient to monitoring and the ICU ventilator to ensure safety. Next, the handoff occurs. This is a critical moment in time when all members of the team should be present, focused, and alert. This includes anesthesia, ICU providers, nurses, and a cardiac surgical team member. Checklists are often utilized, which have been shown to reduce errors and gaps in communication.
- Important points for the cardiac surgery team to convey are pertinent patient history, preoperative cardiac disease, operative findings, surgical interventions, operative complications, bypass configuration, location and number of tubes/drains, placement of pacing wires, underlying cardiac rhythm, need for defibrillation/cardioversion, and desired postoperative care. The surgical team should also discuss expected postoperative course, possible complications to watch for, and desired interventions, including antibiotic needs, blood pressure goals, and anticoagulation plan.
- The anesthesia team conveys information about pre/postop echocardiogram, airway concerns, lines, clamp time, CPB time, circulatory arrest, any difficulty coming off CPB, transfusions, fluids, urine output, medications (inotropes, vaso-pressors, antibiotics, analgesics, paralytics), and other pertinent details.

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L. Franko (✉)

Department of Surgery, Massachusetts General Hospital, Boston, MA, USA

e-mail: [lfranko@mgb.org](mailto:lfranko@mgb.org)

K. Shelton

Heart Center Intensive Care Unit, Department of Anesthesia, Critical Care, and Pain Medicine, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA

e-mail: [kshelton@mgh.harvard.edu](mailto:kshelton@mgh.harvard.edu)

- Critical first assessments after handoff include:
  - Vital signs: It is important to assess for hypothermia upon arrival to the ICU. Blood pressure is closely monitored. The ventilator settings can be changed to obtain appropriate oxygen saturations immediately upon arrival. Additionally, telemetry should be applied, and a 12-lead EKG obtained.
  - Chest X-ray: Evaluate for chest tube position, hemothorax, pneumothorax, and endotracheal tube position.
  - Laboratory values: A full set of labs should be sent upon arrival to the ICU. This should include a CBC, BMP, LFTs, ionized calcium, magnesium, phosphorus, INR, aPTT, fibrinogen, lactate, arterial blood gas, and mixed venous oxygen saturation.
  - Tubes and lines assessment: All tubes and lines should be assessed upon arrival. Central and peripheral intravenous line access should be assessed for patency. Additionally, chest tubes should be assessed for output and drainage. Immediate interventions should be employed if a chest tube appears to be obstructed by clot. Without drainage, blood can collect around the heart or lungs leading to tamponade or hemothorax.
- In cardiac surgical ICU patients, the focus is often on optimizing the patient's cardiac output in the postoperative period in order to supply sufficient end organ perfusion.
  - Cardiac Output = Stroke Volume  $\times$  Heart Rate.

Stroke volume is a function of preload, afterload, and contractility. Interventions focused on modifying these variables can improve cardiac output and end organ perfusion.
  - Preload of the right side is often measured by central venous pressure. Left-sided preload can be assessed with pulmonary capillary wedge pressures. More accurate assessments of preload utilize respiratory arterial pulse pressure variation and stroke volume variation as well. Increasing preload often focuses on giving the patient volume in the form of fluid or transfusions.
  - Afterload is “the workload imposed by any factor that resists ejection of blood from the ventricle” [4]. Afterload is influenced by vascular resistance and wall stress. For example, vasoconstriction often increases afterload, and vasodilation decreases afterload. Afterload is important to maintain blood pressure, though can also be detrimental in decreasing cardiac output and increasing myocardial demand.
  - Contractility is based on the hearts ability to eject blood. This can be modified with inotropic medications.

### ***Hemodynamic Evaluation [2, 3, 5–16]***

- Standard hemodynamic monitoring in the ICU is based on blood pressure. For cardiac surgery patients, this is often through arterial line monitoring and intermittent cuff pressures. However, blood pressure alone is insufficient as a marker of perfusion.
  - The first step when a significant blood pressure change occurs is to check the arterial wave form to ensure it is not dampened (false hypotension) and does not have an exaggerated wave form/whip (false hypertension). A cuff blood pressure should be done to make sure there is not an error in arterial line monitoring.
  - Avoiding significant hypertension is important, particularly in cases with an aortic anastomosis, to prevent bleeding and excessive stress on a new suture line. Hypotension is important to avoid as it can lead to reduced cardiac and end organ perfusion. Blood pressure goals after specific surgeries are often quite surgeon dependent. Commonly, blood pressure goals include mean arterial pressures (MAP) greater than 60–65 or a systolic blood pressure between 100 and 140 mmHg.
  - Baseline blood pressure of the patient and presence of kidney disease is important to understand when setting blood pressure and MAP goals for patients. Chronically hypertensive patients or those with kidney disease may require higher blood pressures for sufficient end organ perfusion.
- Pulmonary Artery Catheters (PACs): PACs provide information regarding right and left heart function. There is controversy regarding utilization of PAC given few studies showing improvement in patient outcomes utilizing this device. Because of this, PACs are not often utilized for non-cardiac surgery patients who are critically ill. Further, PACs are no longer utilized for all cardiac surgeries. However, PACs are utilized in cardiac surgery patients with high risk of cardiac dysfunction, including those with preoperative reduced EF, pulmonary HTN, or right ventricular failure. Additionally, a Class 1A recommendation for heart transplant patients is intermittent right heart catheterization to evaluate pulmonary artery pressures, which can be assessed with a PAC.
  - The PAC is placed through a sheath in the central line. Right-sided internal jugular lines can lead to easier placement of PAC than left. In extreme situations, the PAC can be placed through a femoral sheath though this is significantly more difficult. To place a PAC, the balloon at the catheter tip will be inflated after the PAC is inserted approximately 15–20 cm. The inflated balloon at the tip of the catheter allows it to flow with blood through the right atrium, tricuspid valve, right ventricle, and pulmonic valve into the pulmonary artery without injuring these structures. Location of the balloon is identified based on

transduced wave form and pressures. For example, lower diastolic pressures are seen in the right ventricle compared to the pulmonary artery. Placement is confirmed by monitoring transduced wave forms. It is important never to withdraw a PAC with the balloon inflated as this can lead to injury, so the balloon is always deflated before withdrawing during placement or manipulation.

- Once the PAC is in the main pulmonary artery, the catheter is advanced until it “wedges” in a branch of either the right (more common) or left pulmonary artery. Wedging is identified by changing from an arterial wave form to a venous wave form (reduction in systolic pressure and loss of a dicrotic notch). It is often around a distance of 50 cm when placed. The catheter should have a wedged wave form when the balloon is inflated and pulmonary artery waveform when deflated. If the PAC has a wedged wave form when the balloon is deflated, it needs to be slightly withdrawn (with the balloon deflated). Having the balloon inflated for a significant amount of time or too deep can lead to ischemia and complications. PAC placement can be monitored by transducing the PAC and X-ray. On X-ray, the catheter should not be 1–2 cm past the mediastinum.
- Complications related to PAC include valve injury, subclavian vein thrombosis, arrhythmias, pulmonary infarct, and pulmonary artery rupture. Pulmonary artery rupture is a life-threatening complication. This should be suspected in patients with a PAC who have hemoptysis. This can be caused by distal migration of the catheter with subsequent balloon over inflation or perforation of the artery by the tip of the catheter. If significant, this can require interventional radiology embolization or surgical resection. The initial step in treatment is often inflating the balloon to tamponade the bleeding.
- Pulmonary capillary wedge pressure (PCWP, normal = 4–12 mmHg) measures the filling pressure of the left heart, which is equivalent to left atrial and therefore left ventricle end diastolic pressure in patients without mitral stenosis. This is particularly helpful in a patient with rising CVP and filling pressures to help identify if the source of increased pressure is the right heart, lungs, or left heart.
- Cardiac Output (CO, normal = 4–8 L/min)/Cardiac Index (CI, normal = 2.2–4.0 L/min/m<sup>2</sup>).
  - Cardiac Index = Cardiac Output/Body Surface Area.
  - CO/CI are most often calculated utilizing data from PAC. Information regarding CI can be helpful in monitoring postoperative patients and managing inotrope support. While there are newer techniques to monitor CI continuously, most often CI is measured at specific intervals in the initial postoperative period and with changes in the patient’s condition.
  - Thermodilution is one way to measure cardiac output with a PAC. Most often, a small volume of room temperature or cool fluid is injected into the catheter at a proximal port (often the RA port). It then measures resulting temperature change in the pulmonary artery with a sensor near the end of the catheter. It utilizes a temperature curve to calculate the cardiac output. This method can be inaccurate in patients with tricuspid regurgitation with the CO being falsely underestimated.
  - Fick’s principle is another way to calculate cardiac output. This also requires a PAC. However, in this instance, the PAC is used to obtain a mixed venous

oxygen saturation (SvO<sub>2</sub>). In addition, arterial oxygenation saturation (SaO<sub>2</sub>) is collected from arterial blood gas samples. A calculation utilizing this information is then performed based on estimated oxygen consumption over the arteriovenous oxygen difference.

- Systemic vascular resistance (SVR): SVR is a calculated measurement based on CO and MAP to describe that the amount of systemic (non-pulmonary) resistance blood flow is experiencing. An increase in SVR can be equated to be an increase in afterload and myocardial demand. Given it is a calculated number, SVR can often be misleading; however, it can be beneficial to trend.
- Central venous pressure (CVP, normal 0–8 mmHg): CVP is hemodynamic assessment tool used to monitor preload or filling pressures of the heart. Extremes of CVP (high or low) or overall trends can be helpful in determining fluid status and evaluate overall right heart function. Relative trends in CVP are often more helpful than exact values.
- EKG/Telemetry: Telemetry allows for continuous monitoring. A 12-lead EKG is beneficial as it can be used to identify underlying conduction abnormalities and evidence of acute ischemia. A 12-lead EKG is best obtained with the pacer on pause, if hemodynamically possible, to allow for an accurate assessment of the underlying rhythm.
  - Conduction delays can be seen immediately after cardiac surgery, particularly with valve procedures. These abnormalities can often improve with time as the stunned tissue recovers. Evidence of ischemia is particularly important to monitor for after coronary artery bypass grafting.
- Pacing: A patient's cardiac rhythm after surgery can be abnormal due to injury or stunning of the conduction system. Often times, this abnormality is temporary and improves with time. Given this, temporary epicardial pacing wires are often placed during cardiac surgery. Pacing can be beneficial in cases of heart block, bradycardia, tachyarrhythmias, and bradycardia-induced arrhythmias. Atrial-ventricular pacing has the benefit of maintaining the atrial kick, which can contribute up to 25% of the cardiac output. Ventricular pacing is often utilized as a backup mode.
  - Pacer wires should be evaluated every shift. This includes assessment of underlying rhythm, current pacer settings, and settings required for atrial and ventricular capture. Caution should be applied when checking underlying rhythms as patients with heart block or bradycardia can develop significant hypotension with even a brief pause in pacing.
- Echocardiogram: In the event of decreased cardiac output or changes in patient condition, an echocardiogram can be particularly useful. An echocardiogram can be either transthoracic (TTE) or transesophageal (TEE). In postoperative patients, TEE is often more useful due to difficulty getting good views with TTE. Evaluation for overall ventricular function, localized wall motion abnormalities, valve dysfunction, or pericardial fluid collection can be helpful in identifying the underlying cause of hemodynamic instability.

- Vasopressors/Inotropes: Vasopressors are medications that cause vasoconstriction, which can assist in increasing blood pressure. However, these medications must be used cautiously as increasing afterload without supporting cardiac function can lead to reduced cardiac output. Inotropes are medications that improve cardiac contractility, which work to raise cardiac output. A list of commonly utilized vasopressors, inotropes, and other common medication drips in the cardiac surgery patient can be seen in Table 35.1.

**Table 35.1** Commonly utilized vasopressors, inotropes, and other common medication drips in the cardiac surgery

Medication	Mechanism of action	Effect	Side effects/adverse effects
<b>Vasopressor</b>			
Vasopressin	Vasopressin 1 and 2 receptor agonist	<ul style="list-style-type: none"> <li>– Vasoconstriction</li> <li>– Increases afterload</li> </ul>	<ul style="list-style-type: none"> <li>– Decreases splanchnic flow</li> <li>– Decreases cardiac output through increased afterload</li> </ul>
Phenylephrine	Alpha agonist		<ul style="list-style-type: none"> <li>– Decreases cardiac output through increased afterload</li> </ul>
Angiotensin II	Angiotensin II agonist		
<b>Vasopressor and inotrope</b>			
Norepinephrine	Strong alpha agonist, moderate beta 1 agonist	<ul style="list-style-type: none"> <li>– Vasoconstriction</li> <li>– Increases afterload</li> <li>– Increases contractility</li> <li>– Increases heart rate</li> </ul>	<ul style="list-style-type: none"> <li>– Tachycardia</li> <li>– Arrhythmia</li> <li>– Increases myocardial oxygen demand</li> </ul>
Epinephrine	Strong alpha agonist, strong beta 1 agonist, moderate beta 2 agonist	<ul style="list-style-type: none"> <li>– Vasoconstriction</li> <li>– Increases afterload</li> <li>– Increases contractility</li> <li>– Increases heart rate</li> <li>– Bronchodilation</li> </ul>	
Dopamine	Low dose—dopamine agonist Intermediate dose—dopamine agonist, beta 1 agonist High dose—alpha 1, beta 1 agonist, dopamine agonist	<ul style="list-style-type: none"> <li>– Low dose—increases renal blood flow</li> <li>– Low dose—systemic vasodilation</li> <li>– Intermediate dose—increases contractility</li> <li>– Intermediate dose—increases heart rate</li> <li>– High dose—vasoconstriction</li> <li>– High dose—increases afterload</li> <li>– High dose—increases contractility</li> <li>– High dose—increases heart rate</li> </ul>	<ul style="list-style-type: none"> <li>– Tachycardia</li> <li>– Arrhythmia</li> <li>– Increases myocardial oxygen demand</li> </ul>
<b>Inotrope</b>			

**Table 35.1** (continued)

Medication	Mechanism of action	Effect	Side effects/adverse effects
Milrinone	Phosphodiesterase 3 inhibitor	<ul style="list-style-type: none"> <li>– Increases contractility</li> <li>– Pulmonary and systemic vasodilation</li> <li>– Increases relaxation (Lusitropy)</li> </ul>	<ul style="list-style-type: none"> <li>– Hypotension</li> <li>– Tachycardia</li> <li>– Arrhythmia</li> </ul>
Dobutamine	Strong beta 1 agonist, moderate beta 2 agonist, faint alpha 1 agonist	<ul style="list-style-type: none"> <li>– Increases contractility</li> <li>– Increases heart rate</li> <li>– Pulmonary vasodilation</li> </ul>	<ul style="list-style-type: none"> <li>– Arrhythmia and tachycardia</li> </ul>
Isoproterenol	Strong beta 1 and beta 2 agonist	<ul style="list-style-type: none"> <li>– Increases contractility</li> <li>– Increases heart rate</li> <li>– Pulmonary vasodilation</li> <li>– Bronchodilation</li> </ul>	<ul style="list-style-type: none"> <li>– Increases myocardial oxygen demand</li> <li>– Arrhythmia and tachycardia</li> <li>– Hypotension</li> </ul>
Other			
Inhaled nitric oxide	Direct delivery of nitric oxide to pulmonary vascular endothelium	<ul style="list-style-type: none"> <li>– Selective pulmonary vasodilation</li> <li>– Reduces RV afterload</li> <li>– Minimal systemic vasodilation</li> <li>– Decreases ventilation/perfusion mismatch</li> </ul>	<ul style="list-style-type: none"> <li>– High cost</li> <li>– Production of nitrogen dioxide with direct lung tissue damage</li> <li>– Rebound pulmonary hypertension</li> <li>– Methemoglobinemia</li> </ul>
Inhaled epoprostenol	Prostaglandin I <sub>2</sub> (prostacyclin)		<ul style="list-style-type: none"> <li>– Rebound pulmonary hypertension</li> <li>– Inhibition of platelet aggregation</li> </ul>
Nitroglycerin	Converts to nitric oxide in blood stream	<ul style="list-style-type: none"> <li>– Arterial and venous vasodilation</li> <li>– Decreases preload and afterload</li> </ul>	<ul style="list-style-type: none"> <li>– Headache</li> </ul>
Nitroprusside	Releases nitric oxide upon breakdown		<ul style="list-style-type: none"> <li>– Cyanide toxicity (treated with hydroxocobalamin and sodium thiosulfate)</li> <li>– Methemoglobinemia</li> </ul>
Nicardipine	Dihydropyridine calcium channel blocker	<ul style="list-style-type: none"> <li>– Arterial vasodilation</li> <li>– Decreases afterload</li> </ul>	<ul style="list-style-type: none"> <li>– Rebound hypertension</li> </ul>

### ***Laboratory Evaluation [2, 17, 18]***

- Lactate levels are utilized in cardiac surgery as a marker of perfusion. A lactate greater than 2, slow clearance, or a rising lactate can be a marker of global hypoperfusion. Of note, some patients on epinephrine have been shown to have elevated lactate levels without hypoperfusion thought to be due to sympathetic beta-2 mediated stimulation. Lactate is less reliable marker in patients with significant liver dysfunction.
- Mixed venous oxygen saturation (SvO<sub>2</sub>) describes the amount of oxygen saturation of hemoglobin remaining after all blood has returned to the heart. This is drawn from the distal port of a PAC. This provides a true mixed venous sample from the entire body, including the superior vena cava, inferior vena cava, and coronary veins. This can be used as a marker of perfusion. A level greater than 60% is often associated with good perfusion. The trend is also helpful as a marker of recovery if improving. SvO<sub>2</sub> is utilized to calculate CO via the Fick's principle. Alternatively, central venous oxygen saturation (ScvO<sub>2</sub>) is drawn from a central line and is only indicative of the venous oxygen present within the superior vena cava. This is a less accurate marker of perfusion.
- Monitoring for anemia, coagulopathy, and thrombocytopenia is important postoperatively. Trending hemoglobin and hematocrit is important to monitor for bleeding and to guide transfusions. Additional frequently monitored labs include PT/INR, PTT, fibrinogen, and platelets. In the event of bleeding, transfusions can be targeted based on deficits identified.
- Electrolytes, including potassium, magnesium, calcium, and phosphorus, should also be monitored and replaced to avoid cardiac arrhythmias. Blood glucose monitoring and treatment of hyperglycemia are important. Monitoring of the kidney function, liver function tests, and lipase can monitor for organ function and/or the development of complications. LDH level trends can be helpful in mechanical circulatory support patients as a marker of hemolysis. Arterial blood gases can be used to titrate ventilator settings in addition to provide more information about the acid/base balance of the patient.
- Of note, postoperative troponin levels are not often followed due to troponin release during standard cardiac surgery; however, if concerned for ischemia, troponin levels can be followed. Persistently high levels of troponin had been associated with increased mortality.

### ***Chest X-ray Evaluation [2, 19]***

- Chest radiographs (chest X-rays) are a critical assessment tool. Immediately upon arrival to the ICU, a chest X-ray should be obtained to assess endotracheal tube and line positions to quickly identify malposition. Chest X-rays should also be performed when there is a change in the patient's condition to aid in identifi-



cation of the problem. While some institutions do perform chest X-rays daily, some experts would suggest only performing chest X-rays when clinically indicated rather than routinely after the first 24 h.

- Several abnormalities can be seen on chest X-rays including widening cardiac silhouette (potential cardiac tamponade), pneumothorax, hemothorax, atelectasis, or consolidation. Further, fluid status can be evaluated by identifying signs of pulmonary edema.

## **Warming, Weaning Sedation, Waking [2, 3]**

- Obtaining normothermia is an important first step in recovery before further steps toward hemodynamic stability and extubation can be taken. Hypothermia is associated with decreased cardiac function, arrhythmias, and coagulopathy. Forced air warming devices are often utilized in the ICU to treat hypothermia. Ideal body temperature is greater than 36.0 °C.
- Weaning of sedation starts after the patient has been stabilized, and normothermia has been obtained. It is important to ensure paralysis has been reversed or worn off prior to waking. Further, the patient must be hemodynamically stable. It is important to treat pain as sedation is weaned. Quick stabilization and weaning of sedation support early extubation as described below.

## **Fluid Management [2, 3, 10, 20]**

- Administration of fluid is often done to optimize preload and support CO to ensure end organ perfusion. Fluid administration should be individualized based on markers of perfusion, though most patients after cardiac surgery receive approximately 2–3 L of fluid in the immediate postoperative period.
- Determining if fluid will be beneficial to the patient versus harmful can be difficult. Both hypovolemia and fluid overload should be avoided. Fluid overload is associated with pulmonary edema, heart failure, delayed intestinal motility, and prolonged hospital stays. It is important to determine which patients are fluid responsive. Assessments such as monitoring blood pressure and pulse pressure during passive leg raise or fluid boluses can be helpful. A patient who is not responsive to fluid can be managed with vasopressors and inotropes, which can help to avoid fluid overload.
- Goal directed resuscitation, or fluid administration, is often utilized in the cardiac ICU in order to optimize cardiac output and oxygen delivery. Resuscitation in goal-directed therapy is based on signs of hypovolemia, including lactate trends, SvO<sub>2</sub> less than 60%, cardiac index (CI) less than 2.0, MAP <60, and urine output less than 0.5 mL/kg/h.

- The choice of which type of fluid, crystalloid, or colloid to administer is debated. Crystalloids are generally preferred for early resuscitation. The use of normal saline has decreased given high sodium and chloride content relative to physiologic levels, which can be associated with hyperchloremic acidosis and increased risk of acute kidney injury. For this reason, lactate ringers is often utilized as the crystalloid of choice. Albumin is a frequently utilized colloid in fluid resuscitation. Colloids theoretically have the benefit of increasing oncotic pressure and increasing plasma volume with less third spacing of fluid. However, there are not large-scale clinical trials to specifically support an exact fluid resuscitation protocol for cardiac surgery patients regarding crystalloid versus colloid at this time. Albumin does have an increased cost compared to crystalloids, so some experts suggest against its routine use.

### **Use of Sodium Bicarbonate [21–23]**

- Initial studies identified a reduction of cardiac surgery associated acute kidney injury with the utilization of sodium bicarbonate loading and continuous infusions in patients undergoing cardiopulmonary bypass. However, further research found no benefit to sodium bicarbonate infusions. There was even association of this intervention with increased ventilator time, higher risk of alkalemia, and longer ICU length of stay.
- Sodium bicarbonate is still utilized as needed to buffer the pH to prevent significant acidosis during the immediate postoperative recovery.

### **Goals and Criteria for Extubation [2, 24, 25]**

- The goal for patients after cardiac surgery is for early extubation, which is defined as extubation within 6 hours after surgery. Early extubation is used as a quality marker. This goal was developed as prolonged ventilation is associated with higher costs, increased complications, and longer hospital stays. Given the benefits of early extubation, many institutions have developed multidisciplinary protocols to assist in fast-track extubation pathways.
- Criteria for extubation first rely on decreased ventilator support. Patients should be able to maintain oxygenation with  $FiO_2$  at or below 40% while on relatively low ventilator support. Once this is obtained, the patient's readiness for extubation can be further assessed. The patient should have spontaneous breathing without significant tachypnea, awake mental status, ability to follow commands, hemodynamic stability, and no evidence of significant bleeding. Often prior to extubation, a pressure support trial with decreased ventilator settings is completed to ensure the patient can maintain oxygenation and ventilation without ventilator support.

## Complications After Cardiac Surgery

### *Postoperative Bleeding [6, 26–28]*

- Excessive postoperative bleeding is associated with increased blood transfusions, hospital length of stay, and cost. Postoperative hemorrhage occurs in approximately 2–6% of cardiac surgery patients. Management of postoperative bleeding starts before even leaving the OR. Utilization of a checklist to ensure common areas of bleeding are checked and treated prior to closing has been associated with reduced rates of re-exploration. Often, time will be spent in the OR after the chest is closed monitoring chest tube output to identify patients who require surgical interventions to obtain hemostasis.
- An important focus in the immediate postoperative period is maintaining chest tube patency. If the chest tubes become clogged with clotted blood, this can lead to cardiac tamponade or clinically significant hemothorax, which can require invasive interventions.
- Close monitoring for coagulopathy and thrombocytopenia is critical in cardiac surgery patients as is treatment with transfusions to correct these abnormalities. Treating hypothermia can also help to reverse coagulopathy. Anti-fibrinolytic agents, like epsilon-aminocaproic acid (Amicar) and tranexamic acid (TXA), have been found to be associated with reduced transfusion requirements in cardiac surgery patients without significant increase in morbidity. Red blood cell transfusions can replace blood loss while providing time for correction of coagulopathies.
- If the above interventions are not successful in reducing blood loss, re-exploration and washout in the OR are definitive treatments for postoperative blood loss.

### *Tamponade [2, 3, 6, 29–31]*

- Cardiac tamponade occurs when the chambers of the heart become compressed by external forces, often fluid or blood. This leads to an equalization of pressures between the four chambers and reduced venous filling. This reduces end diastolic volume and subsequently cardiac output. Tamponade can occur in the cardiac surgery patient when there is bleeding without sufficient drainage. This is often signaled by tachycardia, narrowed pulse pressure, increased CVP, and reduced cardiac output. Pulsus paradoxus can also be seen. Pulsus paradoxus is a reduction in systolic blood pressure greater than 10 mmHg during inspiration. Tamponade is also associated with increased vasopressor requirement and worsening markers of perfusion.
- In non-surgical patients, TTE is often utilized to be the gold standard of diagnosis for cardiac tamponade. However, TEE has a greater ability to visualize pericardial fluid/blood in patients after cardiac surgery. This is because TTE can be

limited by chest tubes, patient position, incision, and ventilation. Further, TEE also allows for visualization of the posterior pericardium, where fluid or clot may have accumulated.

- Echocardiographic evidence of cardiac tamponade includes collapse of the atria during systole or ventricles during diastole, respiratory variation of mitral and tricuspid flow, and enlargement of the inferior vena cava with reduced pulsatility. A chest X-ray can show an enlarged cardiac silhouette, and EKG can show low voltage. However, classic signs of tamponade after cardiac surgery may not be initially present so tamponade should be suspected in patients with hemodynamic instability and a pericardial collection.
- Maintaining chest tube patency is critical in the postoperative period to prevent the accumulation of blood around the heart. Additionally, avoiding significant coagulopathy can reduce bleeding postoperatively. In the immediate postoperative period, a definitive measure to treat tamponade is re-exploration in the OR, including opening of the chest and washout with identification and treatment of any areas of bleeding. In decompensating patients, the chest can be opened at the bedside. In patients who are several days from surgery, pericardial drain placement can relieve tamponade.

### ***Atrial Fibrillation [2, 5, 6, 32–34]***

- Atrial fibrillation is common after cardiac surgery. Rates of atrial fibrillation are between 10 and 50% after cardiac surgery, depending on the patient population and surgery. High rates of atrial fibrillation are associated with increased age, mitral valve disease, COPD, and valve surgery. Of note, medications likely epinephrine and norepinephrine can increase the risk of atrial fibrillation because of sympathetic stimulation. Atrial fibrillation in the postoperative period is associated with significant morbidity and increased length of stay.
- Beta blockers have been utilized at some institutions prophylactically to prevent postoperative atrial fibrillation. Amiodarone has also been used prophylactically, but often second line at many institutions due to possible toxicities and adverse effects.
- Pericardial effusions have been associated with increased rates of atrial fibrillation, thought to be due to the effusion causing local inflammation and oxidative damage. Posterior left pericardiotomy has been shown to reduce rates of atrial fibrillation without increasing complications. This procedure involves an incision in the posterior pericardium that allows postoperative pericardial fluid to drain into the left pleural space.
- The decision to anticoagulate postoperative patients with atrial fibrillation is somewhat controversial though most patients with persistent atrial fibrillation and higher CHA<sub>2</sub>DS<sub>2</sub> VASc score are recommended to start on anticoagulation.

## **Stroke [6, 35, 36]**

- The overall risk of stroke with neurological deficits after cardiac surgery is approximately 2–3%. The risk of stroke is associated with increased age, diabetes, known preoperative cerebrovascular disease, and emergent operations. The risk of stroke is highest in aortic and mitral valve surgery. Stroke is associated with increased morbidity and mortality after cardiac surgery.

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