Concise Definitive Review

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Postoperative Critical Care of the Adult Cardiac Surgical Patient. Part I: Routine Postoperative Care

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Objectives: Cardiac surgery, including coronary artery bypass, cardiac valve, and aortic procedures, is among the most common surgical procedures performed in the United States. Successful outcomes after cardiac surgery depend on optimum postoperative critical care. The cardiac intensivist must have a comprehensive understanding of cardiopulmonary physiology and the sequelae of cardiopulmonary bypass. In this concise review, targeted at intensivists and surgeons, we discuss the routine management of the postoperative cardiac surgical patient.

Data Source and Synthesis: Narrative review of relevant English-language peer-reviewed medical literature.

Conclusions: Critical care of the cardiac surgical patient is a complex and dynamic endeavor. Adequate fluid resuscitation, appropriate inotropic support, attention to rewarming, and ventilator management are key components. Patient safety is enhanced by experienced personnel, a structured handover between the operating room and ICU teams, and appropriate transfusion strategies. (*Crit Care Med* 2015; 43:1477–1497)

Key Words: cardiac surgical procedures; cardiopulmonary bypass; coronary artery bypass; hemodynamics; intensive care; postoperative care; quality improvement

Cardiac surgical critical care is emerging as an important subspecialty of critical care medicine. Cardiac operations, including coronary artery bypass graft (CABG), cardiac valve, and aortic procedures, represent one of the most common categories of surgeries performed in the United States. With an average inpatient cost of $40,000, the yearly direct cost of these procedures alone is more than $20 billion, representing 1–2% of U.S. healthcare costs (1). As the population ages and care becomes more sophisticated, cardiac surgery is being performed on older, sicker, and more complicated patients (1, 2). Simultaneously, the spectrum of cardiac surgery is expanding, with increasing use of both minimally invasive techniques and mechanical circulatory support devices. Modern cardiac surgery was made possible by the development of cardiopulmonary bypass (CPB) in the 1950s, but "off-pump" techniques are increasingly used, especially for CABG procedures. Regardless of the procedure performed, successful outcomes depend on optimal postoperative care in the ICU. Most preventable deaths after CABG operations have been linked to postoperative problems in the ICU (3). Thus, "failure to rescue" a patient from potentially reversible complications is an important cause of perioperative morbidity and mortality (4). Historically, cardiac surgeons have provided the bulk of perioperative care to their patients, but this has changed as the number of cardiac surgeons has decreased and work-hour restrictions have limited the ICU experience of surgical trainees (5–7). Consequently, cardiac surgical critical care is increasingly being provided by critical care physicians. Close collaboration between the intensivist and operating surgeon remains essential for comprehensive postoperative care. This two-part review is targeted at intensivists, surgeons, and others who participate in the ICU care of adult cardiac surgical patients. In the first installment, we discuss routine postoperative management after cardiac surgery, with an emphasis on the sequelae of CPB. In the forthcoming second half, we will review procedure-specific management, including off-pump surgeries, common complications, and systems and practice improvement.

SEQUELAE OF CPB AND INTRAOPERATIVE EVENTS

The unique physiologic consequences of cardiac surgery dictate much of early postoperative management. Because of this continuum, the cardiac intensivist must have not only a comprehensive understanding of cardiopulmonary physiology but also the knowledge of surgical anatomy, the conduct of the surgical procedures, and the principles and effects of CPB (7, 8). Although operations have been developed which do not require

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CPB, this technology remains a defining aspect of cardiac surgery. "Off-pump" operations depend on CPB as a backup in the event of hemodynamic deterioration, and CPB technology has been developed into long-term extracorporeal support systems such as extracorporeal membrane oxygenation. Conceptually, CPB supports the circulation in an arrested heart. By isolating the heart and lungs from the circulation, the heart can be stopped, allowing both epicardial operations (e.g., CABG) and intracardiac operations (e.g., valve procedures and closures of septal defects) to be accomplished safely. CPB drains venous blood from the patient, usually from the right atrium or femoral vein, provides that blood with oxygen by way of an oxygenator (which also eliminates carbon dioxide), and then sends that oxygenated blood back into the arterial tree, usually via the aorta or the femoral artery. Nonpulsatile flow is generally used, and flow rates can be adjusted depending on perfusion needs. A heat exchanger in the circuit allows patient temperature to be tightly regulated.

Although a proven technology, there are predictable sequelae of CPB (**Table 1**), many of which are due to the interaction between blood and the artificial surface of the CPB circuit. The first of these is a systemic inflammatory response with biochemical similarity to sepsis (9). Inflammatory cytokine levels are increased, resulting in systemic vasodilation and an endothelial leak syndrome that can persist for hours after the conclusion of bypass. Interestingly, a similar systemic inflammatory response is also present after off-pump surgery, though attenuated compared to post-CPB (10). A second predictable sequel is a multifactorial coagulopathy (11, 12). The bypass circuit is thrombogenic, thus CPB requires high doses of systemic heparin to reduce the risk of embolic phenomena. Although reversed with protamine at the conclusion of CPB, remnant heparin activity can contribute to postoperative coagulopathy. The systemic inflammation of CPB results in a consumptive coagulopathy akin to disseminated intravascular coagulation. Platelet activation leads to platelet consumption and postoperative dysfunction. Hypothermia, used for organ protection during periods of relative ischemia, interferes with normal coagulation. Finally, the crystalloid used to prime the CBP circuit leads to a dilutional coagulopathy and a dilutional anemia, altering blood rheostasis (11, 13, 14). Crystalloid priming contributes to a third predictable finding after CPB: total body volume overload, which is also due to continuing volume requirements necessitated by vasodilation and endothelial leak while on CPB (15).

Vascular injury at cannulation sites can cause dissections, hematomas, and impair distal perfusion; right atrial cannulation sites can be foci of atrial arrhythmias (16–18). Clamping of the aorta, though necessary to isolate the heart from the systemic circulation, can cause atheroembolism and aortic dissection (19–21). Once the aorta is clamped, cardioplegia solution is administered (into the aortic root or through a retrograde coronary sinus cannula) to arrest the heart. Inadequate cardioplegia can lead to myocardial dysfunction and conduction abnormalities (22). The nonpulsatile nature of CPB flow may impair microcirculatory perfusion and contribute to leukocyte activation and systemic inflammation (23–25). Similarly, systemic hypotension on CBP, whether intentional or not, can lead to organ malperfusion and hyperstimulate the sympathetic nervous system, leading to postoperative hemodynamic lability (26). Awareness of these by-products of CPB provides a foundation for the postoperative care of the cardiac surgery patient.

Postoperative management begins in the operating room (OR) as the surgical and anesthesia teams work in conjunction to separate the patient from CPB (if used), obtain hemostasis, and ensure hemodynamic stability. Intraoperative hemodynamic optimization routinely involves transesophageal echocardiography (TEE), allowing correlation of functional imaging with hemodynamic measurements, determination of optimal right and left ventricular (LV) preload, and guidance of inotropic therapy. Chest tubes are placed to drain mediastinal and pleural fluid, and, depending on the procedure performed and surgeon preference, temporary epicardial pacing wires may be placed.

ROUTINE POSTOPERATIVE MANAGEMENT

Although some institutions manage uncomplicated patients in a postanesthesia care unit (27), most patients are admitted to a cardiac surgical ICU (CSICU). A dedicated unit staffed by experienced intensivists, nurses, respiratory therapists, pharmacists, and other allied health professionals may improve outcomes (28–36). Specifically after cardiac surgery, involvement of an intensivist decreased postoperative mechanical ventilation time, reduced blood product transfusion, shortened hospital length of stay, and decreased total costs (37, 38). In the 1990s, "fast-track" protocols for perioperative management were developed as an approach to decrease length of stay and resource consumption (39, 40). Fast-track protocols use short-acting anesthetics, judicious narcotics, and relative normothermia to facilitate rapid extubation and transfer out of the ICU (41, 42). Many of the management strategies discussed below are intended to facilitate this rapid progression from initial postoperative care through ICU discharge. Most patients progress rapidly and require critical care for a relatively short time (6–24 hr) before transitioning to a step-down unit.

Admission to the ICU and Transfer of Care

Transfer from the OR to the ICU is inherently risky, and physician presence, resuscitative drugs, functional pacing wires, and continuous hemodynamic and electrocardiographic (ECG) monitoring are essential to safety. Battery-powered infusion pumps allow uninterrupted administration of sedatives and vasoactives. Either a battery-powered ventilator or manual ventilation may be used; the latter approach is simple, but attention must be paid to avoid hypoventilation (43). Close proximity of the ICU or postoperative care area to the OR can shorten transfer time and facilitate rapid return to the OR in the event of an emergency.

Upon arrival to the ICU, a formal handover should occur, with the surgical and anesthesia teams briefing the ICU team. A standardized handover protocol was shown to decrease critical

TABLE 1. Intraoperative Events and Clinical Sequelae of Cardiopulmonary Bypass

Intraoperative Event	Sequelae	Postoperative Manifestation	References
Aortic cannulation and clamping	Atheroembolism	Stroke	19–21
		Splanchnic embolization	
	Aortic dissection	Organ ischemia	
Right atrial cannulation	Atrial wall injury	Bleeding	177
		Arrhythmias	
Femoral artery cannulation	Distal leg ischemia	Muscle injury and necrosis	16–18
		Compartment syndrome	
	Vascular trauma	Hematoma	
		Need for vascular repair	
		Lymphocele	
	Retrograde aortic perfusion	Retrograde embolism	
		Cerebral hypoxemia	
		Left ventricular distension	
Femoral vein cannulation	Vascular trauma	Hematoma	16
		Deep venous thrombosis	
		Lymphocele	
High-dose heparin	Systemic anticoagulation	Coagulopathy	11, 12
		Heparin-induced thrombocytopenia	
Crystalloid priming of CPB circuit	Hemodilution	Volume overload	11, 13–15
		Dilutional anemia	
		Dilutional coagulopathy	
Extracorporeal circulation	Complement activation	Coagulopathy	9–12, 23–25
	Fibrinolysis		
	Systemic inflammatory response	Coagulopathy	
		Vasoplegia, hypotension, inflammatory end-organ damage (e.g., lung injury)	
	Microvascular hypoperfusion		
	Microemboli	Impairment of renal and mesenteric blood flow	
		Stroke (small vessel)	
Cardioplegic arrest	Inadequate cardioprotection	Myocardial injury and dysfunction	22
		Heart failure	
		Conduction disturbance	
		Arrhythmias	
Hypotension on CPB	Sympathetic hyperactivity	Arrhythmias	26
		Blood pressure lability	
	Renin-angiotensin activation		
	Cerebral and visceral hypoperfusion	End-organ damage	

(*Continued*)

TABLE 1. (*Continued*). Intraoperative Events and Clinical Sequelae of Cardiopulmonary Bypass

Intraoperative Event	Sequelae	Postoperative Manifestation	References
Hypothermia	Splanchnic vasoconstriction	Mesenteric and renal ischemia	225–231
	Impairment of coagulation cascade	Coagulopathy, hemorrhage	
	Shivering	Increased O_2 consumption, increased Co_2 production	
	Sympathetic hyperactivity	Arrhythmias	
		Blood pressure lability	
Hypothermic circulatory arrest	Cerebral ischemia	Stroke, encephalopathy	225–227
	Somatic and spinal ischemia	Paralysis, kidney injury, mesenteric ischemia, myonecrosis	

CPB = cardiopulmonary bypass.

omissions, decrease ventilator time, and improve caregiver teamwork (44, 45). The studied protocols included procedural and anesthetic details, as well as doses of vasoactive and sedative infusions, blood products administered, transesophageal echo findings, perioperative antibiotics, and management concerns (**Table 2**); these details can be adapted based on specific institutional needs. As part of the handover, ventilator settings, rates of IV infusions, and, if present, temporary pacemaker variables (settings, sensing thresholds, and pacing thresholds) are communicated and confirmed by the ICU team (45). The receiving ICU team should have the opportunity to ask questions of both the surgical and anesthesia teams.

Monitoring and Initial Studies

Standard monitoring for the postoperative cardiac patient includes continuous ECG monitoring, pulse oximetry, and invasive arterial blood pressure monitoring. Automated ST-segment analysis, though prone to false-positive and false-negative findings, can detect ischemia (46, 47). The 12-lead ECG is more sensitive for the detection of ischemia and should be obtained on arrival to the ICU (48); new q-waves are particularly predictive of mortality (49). The ECG is invaluable for the detection of postoperative conduction abnormalities, which are rarely seen after CABG but more common after valve procedures. Because of multiple confounders (e.g., pericarditis and myocardial inflammation), the postoperative ECG should be interpreted in the overall clinical context, including coronary anatomy and adequacy of revascularization (50).

A chest radiograph is commonly obtained at ICU admission to exclude pneumothorax or hemothorax and to verify endotracheal tube, vascular catheter, and device (e.g., intra-aortic balloon pump) placement. The admission radiograph detects abnormalities in up to 35% of patients, although few of these result in a change in therapy (51). In the absence of clinical indication, further "routine" radiographs are not required (51–53).

A central venous catheter is mandatory for administration of vasoactive medications and allows measurement of central venous pressure (CVP) and analysis of central venous blood. These are typically placed in the OR but remain in situ during ICU care. Routine placement of a pulmonary artery catheter (PAC) is neither required nor helpful in the majority of patients (54–58). In high-risk scenarios such as severely decreased LV function (ejection fraction < 30%), right ventricular (RV) failure, pulmonary hypertension, severe renal insufficiency, or thoracic transplantation, PACs may be useful, although this is controversial (59, 60). Urinary catheters are essential to monitor urine output and are an additional physiologic monitor to aid in assessing perfusion. Chest tubes are placed to wall suction, function checked, and output closely followed.

Initial laboratory studies should include arterial blood gas analysis, hemoglobin, potassium, calcium, and glucose. Depending on the clinical situation, a central or mixed venous blood gas, lactic acid, coagulation profile (prothrombin time, partial thromboplastin time, fibrinogen), and platelet count may be indicated. Immediate postoperative troponin levels are rarely informative, as high levels of troponin may be released even during a successful operation. However, persistent troponin elevation 24 hours postoperatively is associated with a higher cardiac mortality (61).

Hemodynamic Management

Hemodynamic lability is the rule in the early postoperative period. Virtually, all patients have postoperative myocardial dysfunction and decreased ventricular compliance (62), superimposed upon intravascular hypovolemia and vasodilation. It is critical to appropriately manipulate preload, afterload, and inotropic support (63, 64). Equanimity and vigilance are required to avoid overreacting to disquieting but self-limited hemodynamic swings while appropriately intervening on concerning trends or sudden deterioration.

Goals and Indicators of Perfusion. Desired hemodynamic goals are a key element of the OR to ICU handover and may be adapted to individual patient characteristics or clinical scenarios. Commonly targeted hemodynamic variables include blood pressure, indices of preload (cardiac filling pressures), and assessments of cardiac function and output. The overall goal of hemodynamic management is to maintain adequate organ perfusion and oxygen delivery. There is increasing interest in "goal-directed therapy" (GDT) protocols, which place a premium on optimizing cardiac output and systemic oxygen delivery to meet patient-specific perfusion goals (65–68).

TABLE 2. Key Components of Operating Room to ICU Handoff

Surgical Team	Anesthesia Team
Patient name and demographics	Patient name and demographics
Past medical history and allergies	Past medical history and allergies
Indications for surgery	Airway management details
Intraoperative findings	Vascular access details
Details of procedure	Details of anesthetic course
Cardiopulmonary bypass time	Transesophageal echocardiogram findings
Aortic cross-clamp time	Timing of perioperative antibiotics
Optimum filling pressures on separation from cardiopulmonary bypass	Anesthetic problems and complications
Complications	Inotropic/vasoactive infusions
Inotropic/vasoactive infusions	Blood product administration
Blood product administration	IV fluid administration
Hemodynamic goals	Sedative infusions
Management concerns	Ventilator settings
	Temporary pacemaker settings
	Management concerns

References (44, 45).

Although GDT after cardiac surgery has had promising results in small studies and in a meta-analysis of 699 patients, and seem to have a sound physiologic basis, it has not yet been subjected to large-scale study (69). Regardless of whether a protocol is used, hemodynamic management of postoperative cardiac patients requires integration of hemodynamic, clinical, and laboratory data and interpretation of those data within the overall clinical context.

Blood pressure. There are few data to definitively guide blood pressure management after cardiac surgery. It is not known if data on intraoperative blood pressure management apply postoperatively (70, 71). A mean arterial pressure (MAP) of 60–90 mm Hg and a systolic blood pressure of 90–140 mm Hg are reasonable targets (62). A higher MAP may be indicated in hypertensive patients or patients with renal insufficiency, whereas a lower MAP may be desirable in the face of poor ventricular function, mitral repair surgery, vulnerable aortic suture lines, or active bleeding. Although near-infrared spectroscopy monitoring may be helpful in targeting intraoperative blood pressure (72), its postoperative utility is unknown (73). Ultimately, blood pressure is a poor indicator of systemic perfusion and cannot be used as a hemodynamic goal in isolation.

Preload. Cardiac output and venous return are integrally related: optimum cardiac function requires optimum cardiac preload (74–76). Intravascular pressures (e.g., CVP, pulmonary artery occlusion pressure, and pulmonary artery diastolic pressure) are commonly used as surrogates for preload and guides for fluid resuscitation (55). Unfortunately, static intravascular pressures poorly predict fluid responsiveness (77–79); some data suggest more utility after cardiac surgery than in other shock states (80–82). Dynamic approaches to assessing preload, such as respiratory arterial pulse pressure variation (PPV) and stroke volume variation (SVV, based on pulse contour analysis), using an approximate threshold value of more than 11% to indicate a volume deficit, may be more accurate in predicting fluid responsiveness (79, 83, 84). However, these techniques require controlled mechanical ventilation, the absence of spontaneous breathing, and normal cardiac rhythm; they are inaccurate in patients with open chests (85). The use of lower tidal volumes (< 8 mL/kg) may limit the accuracy of PPV and SVV (84, 86). Ultrasound measurement of inferior vena cava diameter does not appear to be useful after cardiac surgery (87).

Clinical and laboratory assessment of perfusion. A physical examination demonstrating warm extremities with strong pulses and good urine output is reassuring for adequate perfusion but should be supplemented by objective data. Lactate is an extremely sensitive marker of impaired perfusion, and even minimally elevated levels (> 2 mmol/L) can identify patients with occult hypoperfusion (88). Higher lactate levels (> 3–4 mmol/L) and slow lactate clearance accurately predict major complications after cardiac surgery (89–91).

Cardiac output and venous oxygen saturation. Thermodilution remains the gold standard for cardiac output measurement (92). However, as noted above, PACs are neither required nor helpful in the majority of cases. Other approaches, such as esophageal Doppler monitoring of aortic blood flow, pulse contour analysis, and transpulmonary thermodilution (TPTD), have been recently reviewed (92). There are limited data examining these techniques in cardiac surgical critical care, consisting of relatively small studies. Several groups have reported good correlation between transesophageal Doppler

and thermodilution; this is not universal (93–96). Pulse contour devices and TPTD have also been reported to correlate well with thermodilution, but lose accuracy during hemodynamic instability and with aortic insufficiency; "uncalibrated" pulse contour devices (not calibrated to TPTD data) seem less accurate (92, 97–101). Pulse contour devices are relatively inaccurate at detecting changes in cardiac output after cardiac surgery, limiting their utility (102, 103). If a device for monitoring cardiac output is in place, a normal cardiac output should be targeted. In most cases, this corresponds to a cardiac index of more than 2.2–2.5 L/min/m^2 (62). There is no benefit in targeting a supranormal cardiac output (104–106).

Based on the Fick principle, central venous ($S_{cv}O_2$) or mixed venous oxygen (S_vO_2) saturations allow global assessment of adequacy of oxygen delivery and cardiac output and can be used in isolation or to corroborate measured cardiac output. $S_{cv}O_2$ and S_vO_2 greater than 70% and greater than 60%, respectively, are generally reassuring (107, 108), although data regarding venous oxygen saturations are mixed (109, 110). Significant discrepancies between $S_{cv}O_2$ and S_vO_2 may predict postoperative complications (111).

Fluid Resuscitation. Appropriate fluid resuscitation is perhaps the most important hemodynamic intervention in the immediate postoperative period and should be first-line therapy for early hemodynamic instability. There are four major contributors to the need for intravascular volume replacement: blood loss, increasing vascular capacitance with rewarming, third space fluid losses due to CBP-induced inflammation, and elevated cardiac preload requirements in the setting of transient cardiac ischemia-reperfusion injury, myocardial stunning, and decreased ventricular compliance. Crystalloids are preferred for fluid resuscitation. The choice of crystalloid is a matter of institutional preference, although emerging data may question equipoise in this matter. IV fluids containing large amounts of chloride, such as normal saline (0.9% sodium chloride), cause hyperchloremic acidosis and may be associated with acute kidney injury (AKI) (112). A change to low-chloride resuscitation fluids was associated with a decrease in AKI (113), and there is physiologic rationale, if few hard data, for using buffered balanced salt solutions such as lactated Ringer's solution or Plasmalyte (Baxter, Deerfield, IL) (112, 114). Synthetic colloids are not superior to crystalloids, can worsen coagulopathy, and are associated with renal failure (112, 115–117). Albumin is effective for volume resuscitation after cardiac surgery (117, 118), but no more so than crystalloid, and cost should preclude its use as a first-line volume expander.

Overexuberant fluid administration can contribute to heart failure, pulmonary edema, hemodilution, intestinal dysfunction, increased transfusion requirements, and prolonged hospital stay (119–124). It is unusual to require more than 2–3 L of crystalloid, particularly after the patient has warmed completely. Patients with significant cardiac hypertrophy often require higher filling pressures (62). Similarly, long aortic cross-clamp periods decrease ventricular compliance, resulting in a need for higher filling pressures. Ongoing fluid requirements should prompt rapid assessment for alternative causes of hemodynamic instability: bleeding, tamponade, tension pneumothorax, valvular dysfunction, cardiac ischemia, and heart failure. Echocardiography may be useful when faced with an unclear hemodynamic picture. Miniaturized TEE probes that can remain in situ for up to 72 hours have been reported to be useful in guiding resuscitation (125, 126).

Inotrope and Vasopressor Support. Ventricular and vascular dysfunction are ubiquitous after cardiac surgery, and many patients require inotropic or vasopressor support upon separation from CPB (127–129). There are few data guiding choice of vasoactive agents, and tremendous variability exists in their use (130–133). Inotropes and vasopressors span multiple drug classes, including catecholamines, phosphodiesterase inhibitors (PDEIs), and hormonal analogs, each with specific characteristics (**Table 3**). Commonly used inotropic catecholamines include epinephrine (134–136), norepinephrine (137), dopamine (138–140), and dobutamine (138, 140–142). Whereas most catecholamines have some vasopressor activity, dobutamine is an inodilator, and often needs to be used with a vasopressor to maintain an adequate MAP. Data in noncardiac surgical patients suggest that 1) the combination of norepinephrine and dobutamine is just as efficacious as and perhaps safer than epinephrine and 2) norepinephrine is superior to dopamine for cardiogenic shock (143, 144). PDEIs, such as milrinone, amrinone, and enoximone, are another important inotropic class (134, 140, 141, 145, 146). PDEIs have attractive systemic and pulmonary vasodilatory properties and may be particularly useful in the settings of right heart failure and pulmonary hypertension (132, 147). Like dobutamine, PDEIs are inodilators and frequently require a concomitant vasopressor to maintain MAP (148). PDEIs have longer half-lives than catecholamines, ranging from 30 to 60 minutes (milrinone) to 3.5 hours (amrinone) (132). This long half-life, along with well-described effects on platelet function and number, is an important consideration with PDEIs (149). There is emerging interest in the calcium sensitizer levosimendan (150–153). However, available data do not yet support a beneficial effect of levosimendan on mortality (154). Levosimendan has been reported to increase the risk of bleeding (155) and is not approved in the United States.

Vasopressors are useful either in the face of excessive vasodilation or inodilator-induced hypotension. Typical agents are norepinephrine and the hormone vasopressin. At low doses (0.02–0.04 U/min), vasopressin is effective at treating postoperative vasodilation and vasoplegia (156, 157). Phenylephrine should rarely, if ever, be used; it both increases afterload and decreases bypass graft flow (158).

Despite their invaluable role in the management of postoperative cardiac patients, caution is mandated with inotropes and vasopressors. Inotropes increase myocardial oxygen demand and are arrhythmogenic; dopamine seems to be the worst offender in this regard (132). The use of inotropes after cardiac surgery may be independently associated with postoperative myocardial infarction, stroke, renal dysfunction, and increased mortality (128, 159). Meta-analyses have also suggested an increase in mortality when milrinone or dobutamine

is used (160–162). Titrating vasopressors to achieve a higher MAP does not necessarily indicate an increase in cardiac output. Indeed, the increase in afterload may be at the expense of stroke volume and systemic perfusion (137). Furthermore, high doses can cause ischemia in peripheral and splanchnic vascular beds. Thus, the use of inotropes and vasopressors should be judicious. Large-scale trials are needed to determine optimum indications and regimens for inotropic therapy after cardiac surgery.

Vasodilators and Afterload Reduction. Although hypotension is common, postoperative hypertension is also a frequent problem (163–165). Hypertension can increase cardiac afterload (and worsen cardiac function), potentiate bleeding, and threaten fragile anastomoses. One large study reported that nearly 90% of patients were treated to lower blood pressure at least once in the perioperative period (166). Vasodilators are commonly used to control blood pressure, reduce cardiac preload (venodilators) or afterload (arterial vasodilators), maximize stroke volume, and prevent native and graft coronary vasospasm. Vasodilators are frequently used in combination with inotropes to minimize afterload and optimize cardiac output (130). In a hypertensive or normotensive patient, reduction of afterload can dramatically increase cardiac output and spare inotropic agents (167–169). Because of the risk of sudden hemodynamic deterioration, short-acting agents such as nitroglycerin and nitroprusside may be preferable, although these can both worsen hypoxemia by antagonizing hypoxemic pulmonary vasoconstriction (170). Nicardipine is an alternative, but it has a longer half-life (171, 172). LV afterload reduction is essential after mitral regurgitation surgery because the newly competent mitral valve no longer serves as a low pressure "pop-off" valve for the LV. This can abruptly increase LV afterload and precipitate LV failure (173). Reduction of systemic blood pressure can mitigate this consequence of a newly competent mitral valve. After aortic surgery, it makes sense to keep blood pressure low to protect the aortic suture line, similarly, in the setting of bleeding, to decrease the pressure driving hemorrhage. Bypass grafts and, less frequently, native coronary arteries can vasospasm (174), causing ischemia and hemodynamic compromise (175). Nitroglycerin is the drug of choice for coronary vasospasm (176).

Arrhythmias: Prophylaxis and Management. Supraventricular arrhythmias occur frequently after cardiac surgery and contribute to prolonged hospital stays, higher costs, and increased risk of stroke. The loss of atrial contraction can significantly impair cardiac output. Advanced age, sleep apnea, prior arrhythmia or congestive heart failure, bicaval cannulation, and long CPB runs are all predictors of atrial dysrhythmias (177, 178). Hypothermia, electrolyte abnormalities, myocardial irritation, atrial distension, and proarrhythmic drugs are also other factors. In the absence of prophylaxis, supraventricular arrhythmias occur in 30–40% of patients; most of these are atrial fibrillation or flutter, which occur more commonly after valve procedures than CABG (177) and peak in prevalence on postoperative days 2 and 3 (179). Prophylaxis in appropriate patients can decrease the prevalence of atrial fibrillation by nearly 50% (180–182); options for prophylaxis appear in **Table 4**. In patients without the need for inotropic support, β-blockers provide both anti-ischemic and antiarrhythmic therapies (182–184). Amiodarone has less negative inotropy than β-blockers (179, 182, 185–187) and may be a superior agent for prophylaxis and treatment in patients with compromised cardiac function. Amiodarone has well-described pulmonary toxicity, however, and may confer a higher risk of bradycardia and hypotension (188–190). Sotalol is effective at preventing atrial fibrillation, but potential adverse effects may mitigate against its first-line use (191). Magnesium is safe but is a less effective prophylactic strategy than other pharmacologic approaches; importantly, magnesium is only effective at prophylaxis and should not be used to treat atrial fibrillation (182, 192). Biatrial pacing with temporary epicardial leads is a relatively low-risk prophylactic strategy to prevent atrial arrhythmias (182, 192, 193).

Ventricular arrhythmias are uncommon and must raise suspicion for ongoing ischemia. Amiodarone is useful for pharmacologic cardioversion, but hemodynamic instability mandates immediate cardioversion. Patients with low ejection fractions and continued ventricular arrhythmias may benefit from electrophysiologic consultation and internal cardioverter/defibrillator placement (194).

Bradycardias and Temporary Pacemaker Management. Bradycardias can also occur after cardiac surgery and are often potentiated by anti-tachyarrhythmia prophylaxis. Conductive tissue may be directly traumatized, particularly during valvular surgery, as the atrioventricular node sits in juxtaposition to the annuli of the mitral, aortic, and tricuspid valves. Sinus asystole, sinus bradycardia, junctional bradycardia, atrioventricular conduction delays, and complete heart block are seen. It should be noted that atrioventricular conduction problems may not be immediately apparent but can develop several days after surgery. Accordingly, surgeons may place temporary atrial and ventricular epicardial pacemaker leads to allow pacing if necessary. In low-risk patients, ventricular leads may suffice.

If temporary pacing is desired or required, atrial pacing is preferred, as stroke volume is greatest when the electrical impulse is generated above the atrioventricular node (195). In the event of an atrioventricular conduction block, atrioventricular sequential pacing is the next choice. Ventricular pacing should primarily be used as a rescue mode in the event of cardiac standstill or failure of atrial leads to capture. To that end, pacing wires should always be tested and set in an inhibited (e.g., "VVI") mode to rescue significant bradycardia. If postoperative atrioventricular conduction block persists beyond 5–7 days, permanent pacemaker placement is usually required.

Sedation, Pain Control, and Delirium

Appropriate sedation and analgesia are essential components of postoperative cardiac surgical care (196). As in other critical care arenas, minimizing sedation minimizes delirium, speeds extubation, and facilitates early ambulation and physical rehabilitation (197). However, on arrival to the CSICU, most patients are still under neuromuscular blockade (NMB), and sedation must

TABLE 3. Inotropic, Vasopressor, and Vasodilatory Agents Commonly Used After Cardiac Surgery

Agent	Class	Effect(s)	Indications
Epinephrine	Catecholamine	Inotrope	Low CO
		Vasopressor (higher doses)	Hypotension
Norepinephrine	Catecholamine	Vasopressor	Hypotension
			Excessive vasodilation
		Some inotrope	Vasoplegia
			Low CO
Dopamine	Catecholamine	Inotrope	Low CO
		Some vasopressor	Hypotension
Dobutamine	Catecholamine	Inotrope	Low CO
		Systemic vasodilator	Decrease LV afterload
Milrinone (Amrinone; enoximone)	Phosphodiesterase inhibitor	Inotrope	Low CO
		Systemic vasodilator	Decrease right ventricular afterload
		Lusitrope	Decrease LV afterload
		Pulmonary vasodilator	
Vasopressin	Hormone	Vasopressor	Hypotension
			Excessive vasodilation
			Vasoplegia
Levosimendan	Calcium sensitizer	Inotrope	Low CO
		Lusitrope	
Sodium nitroprusside	NO donor; cGMP stimulator	Arterial vasodilator	Low CO with high BP
			Decrease LV afterload
			Decrease BP
Nicardipine	Calcium channel blocker	Arterial vasodilator	Low CO with high BP
			Decrease LV afterload
			Decrease BP
Nitroglycerin	NO donor; cGMP stimulator	Venous vasodilator	Decrease LV preload
			Decrease BP
			Treat or prevent coronary vasospasm

CO = cardiac output, Vo_2 = oxygen consumption, LV = left ventricle, NO = nitric oxide, cGMP = cyclic guanosine monophosphate, BP = blood pressure. See text for discussion.

continue until NMB has worn off or been reversed. In the setting of hypothermia and altered drug elimination (e.g., hepatic or renal dysfunction), NMB can be prolonged, mandating a longer duration of deep sedation. The ideal sedative reliably maintains adequate sedation but rapidly wears off once weaning is appropriate. Propofol is commonly used and, when combined with intermittent narcotic doses, results in faster postoperative extubation than a combination of fentanyl and midazolam infusions (198). Propofol can cause or contribute to hypotension, and the infusion rate should be carefully monitored. Propofol has no analgesic effects and must be used in concert with an analgesic agent. Retrospective data suggest that dexmedetomidine (which does have analgesic effects) may be a good substitute for propofol in cardiac patients and may result in faster extubation (199) and decreased mortality (200, 201). Dexmedetomidine can cause significant bradycardia and hypotension and must be used with caution. Benzodiazepines should be avoided in the absence of a specific indication.

Advantages	Caveats	References
Effective at increasing CO; may be agent of choice in hypotensive patients with low CO	Increased myocardial V_{O_2}; splanchnic vasoconstriction; increases lactate; arrhythmogenic; increases LV afterload at high doses	132–136
More inotropy than vasopressin; superior to dopamine in cardiogenic shock	Splanchnic vasoconstriction; increases LV afterload; variable effect on CO	137, 143, 144
	Arrhythmogenic; Increased myocardial V_{O_2}; splanchnic vasoconstriction; no evidence to support selective renal vasodilation	132, 138–140
Effective LV afterload reducer; more effective than epinephrine or dopamine	Systemic hypotension; vasopressor support frequently needed; increased myocardial V_{O_2}; increases heart rate; arrhythmogenic; possible increase in mortality	132, 138, 140–144, 162
Increases CO without tachycardia; effective pulmonary vasodilator	Systemic hypotension; vasopressor support frequently needed; long half-life; increased myocardial V_{O_2}; thrombocytopenia; possible increase in mortality	132, 134, 140, 141, 145–149, 160,161
Highly effective; spares catecholamines	Splanchnic vasoconstriction; increased LV afterload	156, 157
Increases CO without increasing myocardial V_{O_2}	Limited data; increased bleeding; not available in United States	132, 150–155
May increase CO in hypertensive or normotensive patients; short half-life	Antagonizes hypoxic pulmonary vasoconstriction Intrapulmonary shunting Cyanide toxicity Systemic hypotension	167–170
May increase CO in hypertensive or normotensive patients	Long half-life Systemic hypotension	171, 172
Short half-life Effective at preventing/treating coronary vasospasm	Limited effect on CO Intrapulmonary shunting Limited effect on BP	176

Pain after cardiac surgery is frequently undertreated (197). Adequate pain control is mandatory to improve pulmonary function, decrease delirium, and increase patient satisfaction (197, 202). Nurse-driven protocols facilitate pain assessment and rapid treatment of postoperative pain (197, 203). Narcotics are the mainstay of analgesia in the early postoperative phase. Fentanyl is commonly used, although there are some data to recommend remifentanil, which has a shorter half-life than fentanyl, and may shorten the time until extubation and provide some degree of cardioprotection (204–206). IV paracetamol is an effective analgesic agent and may spare narcotics (207, 208). There is no evidence of an increased risk of hepatoxicity (209). Once extubated, patient-controlled analgesia (PCA) devices are effective and well received by patients and nurses (202). Nonpharmacologic adjuncts, such as music, may improve pain control (210, 211). Once patients are able to take oral medications, oral narcotic regimens typically suffice. There are some data supporting the use of ketorolac in patients with normal

TABLE 4. Atrial Fibrillation Prophylaxis After Cardiac Surgery

Strategy	Advantages	Caveats	References
β-blockers	Highly effective: OR 0.33 (95% CI, 0.26–0.43) for atrial fibrillation compared with control (pooled analysis); anti-ischemic	Negative inotropy Contraindicated with bradycardia, conduction disturbances	182–184, 192
Amiodarone	Effective: OR 0.43 (95% CI, 0.34–0.54) for atrial fibrillation compared with control (pooled analysis); less negative inotropy than β-blockers	Contraindicated with bradycardia, conduction disturbances, pregnancy, chronic interstitial lung disease. Can cause pulmonary, hepatic, and thyroid toxicity	179, 182, 183, 185–190, 192
Sotalol	Highly effective: OR 0.34 (95% CI, 0.26–0.43) for atrial fibrillation compared with control (pooled analysis)	Potential for adverse effects limits use to high-risk patients	182, 191, 192
Magnesium	Effective: OR 0.55 (95% CI, 0.41–0.73) for atrial fibrillation compared with control (pooled analysis); safe Few adverse effects	Least effective pharmacologic strategy Does not decrease hospital length of stay	182, 192
Biatrial pacing	Effective: OR 0.47 (95% CI, 0.36–0.61) for atrial fibrillation compared with control (pooled analysis); safe	Less effective than β-blockers Equipment costs Requires epicardial pacing leads	182, 192, 193

OR = odds ratio.
ORs are derived from reference (182).

renal function (212–214), but a high degree of caution is suggested with all nonsteroidal anti-inflammatory drugs due to adverse effects on platelet and kidney function.

Delirium is a significant problem after cardiac surgery (215–219) and adversely affects outcomes (197). Risk factors include benzodiazepine use, restraints, and immobilizing therapeutic devices (e.g., ventricular assist devices and intra-aortic balloon pumps) (220). Most delirium in the CSICU is hypoactive, which is likely to go unrecognized and is a risk factor for prolonged mechanical ventilation (221). A dexmedetomidine-based sedation strategy may decrease delirium in cardiac surgical patients (201, 222); melatonin antagonists may also be effective (223). Early mobilization reduces delirium in medical ICU patients, but there are no data confirming this in cardiac ICUs (215, 224). Pharmacologic treatment of delirium with antipsychotics is of questionable efficacy (197, 215); caution is warranted due to the proarrhythmic effects of these drugs.

Hypothermia and Rewarming

Historically, intraoperative hypothermia was deliberately induced to diminish the rate of rewarming of the myocardium during aortic cross-clamp and to provide cardiac protection from ischemic injury during periods of low or absent flow on CPB. In the modern era, deep hypothermia is still used for specialized applications (e.g., aortic procedures using deep hypothermic circulatory arrest [225, 226]), but normothermic CPB, targeting temperatures greater than 34°C, is used for many procedures (227–229). Hypothermia can still result from cold pericardial irrigation, heat loss from open body cavities, and administration of cold or room temperature fluids and blood products. Many patients arrive to the ICU at 34–36°C. Hypothermia interferes with coagulation, predisposes to arrhythmias, decreases cardiac output, and delays weaning from mechanical ventilation (230–232). Forced air warming devices are most effective; warm IV fluids are also useful (233–236). Vasodilation during rewarming can affect hemodynamics, and the need for additional fluid should be anticipated.

Ventilatory Support and Respiratory Management

Cardiac surgery has a marked, if temporary, effect on the respiratory system. Although select patients can be extubated in the OR, most patients arrive to the ICU intubated and mechanically ventilated. Almost all patients have restrictive physiology, pulmonary edema, decreased lung compliance, and atelectasis (119, 237–240); some have phrenic nerve injury (241, 242). Sedation and residual NMB initially mandate controlled ventilation, but rapid extubation (within 6 hr of admission) is associated with early ICU discharge and improved outcomes (234, 243, 244). This requires immediate attention to ventilator management. Standardized protocols with visual cues and staff reminders can increase rates of early extubation (245, 246).

Cardiac surgery is a risk factor for acute respiratory distress syndrome (ARDS), which confers significant morbidity (119, 247, 248). The risk of ARDS can be minimized by preemptively using a lung-protective (low tidal volume) ventilatory strategy (119, 249). Use of low tidal volumes (V_T) (6 mL/kg predicted body weight [PBW]) compared with 10 mL/kg PBW increased the number of patients free of mechanical ventilation at 6 hours postoperation and decreased reintubation rates (250). In addition, V_Ts greater than 10 mL/kg PBW have been linked to multiple organ dysfunction after cardiac surgery (251, 252).

F_{IO_2} should be titrated to target a Pa_{O_2} of greater than 70 mm Hg. In patients at risk for RV failure, a higher Pa_{O_2} target range (85–100 mm Hg) may help reduce RV afterload (253). Appropriate application of positive end-expiratory pressure (PEEP) is invaluable to support oxygenation, and even high levels are safe after cardiac surgery (254). Indeed, some data suggest that higher levels of PEEP in the immediate postoperative period (10 cm H_2O vs 8 or 5 cm H_2O) improve pulmonary compliance (255), although it remains unclear if the routine application of higher levels of PEEP confers any meaningful clinical benefit (254, 256). In the setting of persistent atelectasis accompanied by hypoxemia, recruitment maneuvers (RMs) may be of some benefit, but adverse consequences (e.g., desaturation, hypotension, arrhythmias, and barotrauma) are not infrequent (257–260). RMs performed by increasing PEEP to 20 cm H_2O for 2 minutes may be better tolerated than using continuous positive airway pressure of 40 cm H_2O for 30 seconds (261).

Hypercarbia can increase RV afterload, thus effective ventilation is essential. Normocarbia or a mild respiratory alkalosis should be targeted, with a goal pH of 7.35–7.45 (262). Blood gases should be regularly monitored. As the patient is warmed, Co_2 production increases and lactate is flushed from previously constricted vascular beds, causing a combined metabolic and respiratory acidosis. Minute ventilation should be increased to compensate for this developing acidosis, preferably via increases in respiratory rate so as to maintain lung-protective tidal volumes. If hemodynamics allow, the head of the bed should be raised to 30° to minimize the risk of aspiration and ventilator-associated pneumonia (263).

Once the patient has reached relative normothermia (35.5°C), NMB is reversed and sedation rapidly weaned. Reversal at cooler temperatures can increase shivering and Co_2 production. As spontaneous breathing returns, and if the patient is hemodynamically stable with no ongoing acidosis, the ventilator is switched to a minimal pressure support (PS) mode, with the goal of rapid extubation. Readiness for extubation can be gauged based on tolerance of a spontaneous breathing trial (SBT). SBTs can be completed using either minimal PS settings (e.g., 5 cm H_2O) or "t-pieces" (264). The utility of measuring respiratory mechanics and extubation predictors is debatable, and these measurements may delay extubation (265, 266). Newer modes of mechanical ventilation, such as adaptive support ventilation, have been postulated to speed weaning, but the data do not yet support their routine use (267–269). Important adjuncts to rapid extubation are minimizing fluid overload and blood transfusion during early resuscitation (116, 270). Elevated levels of B-type natriuretic peptide (BNP) (measured at ICU admission and after a SBT) have been shown to predict failure to wean from mechanical ventilation after cardiac surgery (271); the elevated BNP levels likely reflect volume overload and resultant ventricular dysfunction (272).

Early extubation is the best prevention for complications such as ventilator-associated pneumonia or prolonged ventilator dependence. Indeed, mechanical ventilation for more than 16 hours after cardiac surgery predicts a poor prognosis (243, 273). For those who have persistent respiratory failure, standard mechanical ventilation practices, such as daily SBTs, chlorhexidine mouth hygiene, elevated head of bed, and daily sedation interruptions, are essential to minimize time on mechanical ventilation and improve outcomes (263). Few patients will require reintubation; those that do typically have preextant pulmonary dysfunction had more complicated operative courses (274).

Electrolyte and Acid-Base Management

Electrolyte repletion, particularly of potassium, is usually required after cardiac surgery. Magnesium is also typically lost during CPB; magnesium repletion decreases the risk of arrhythmias postoperatively (275, 276). Rapid correction of electrolyte abnormalities is facilitated by electrolyte repletion protocols. Most patients will have a metabolic acidosis due to relative ischemia, anaerobic metabolism, lactate production, and depletion of bicarbonate stores during CPB. As discussed earlier, the acidosis may worsen with hyperchloremic fluids, increasing Co_2 production, and reopening of vascular beds. Severe acidosis can predispose to arrhythmias, increase RV afterload, and depress myocardial function, although animal data suggest that cardiac output is maintained until pH falls below 7.1–7.2 (277). Often the acidosis can be controlled by increasing minute ventilation, but some intensivists administer bicarbonate (278). There are no data that bicarbonate either improves cardiovascular function or decreases mortality (277, 279–282). Exogenous bicarbonate can cause hypernatremia, volume overload, rebound alkalosis, paradoxical intracellular acidosis, and increased Co_2 production. Ongoing metabolic acidosis must be treated as evidence of inadequate perfusion until proven otherwise (283).

Glycemic Control

Prevention of postoperative hyperglycemia reduces the risk of deep sternal wound infections, all-cause infections, sepsis, and mortality (284, 285), and adequacy of glycemic control has been used as a quality measure (286). A high degree of glycemic variability may predict adverse events (287). Glycemic control is complicated in the immediate postoperative period by the stress response to surgery and exogenous catecholamines used for hemodynamic support and can be challenging in both diabetics and nondiabetics (288). Current data and practice guidelines support keeping blood sugar less than 180 mg/dL for the first two postoperative days (289, 290). This typically requires an insulin infusion for the first 12–24 hours, with subsequent transition to subcutaneous insulin (291, 292). Strategies need to be individualized to account for insulin-resistant and diabetic patients, with caution to avoid both hyper- and hypoglycemia (293).

Management of Bleeding and Transfusion Strategies

Some bleeding is expected after cardiac surgery, but some patients experience significant hemorrhage. Unfortunately, definitions of excessive postoperative bleeding have varied

substantially; no discrete value exists to identify clinically significant bleeding. Chest tube drainage is commonly used to define excessive bleeding, but values ranging from 200 mL/hr to 1,500 mL/8 hr have been used (294–297). A universal definition of perioperative bleeding in adult cardiac surgery was recently proposed by an expert panel (298). This definition identifies five classes of bleeding, ranging from insignificant (class 0) to massive (class 4), based on several variables: delayed sternal closure, chest tube output over 12 hours, blood products transfused, and need for surgical reexploration. This classification scheme appears to predict risk of mortality and other complications (299), but its applicability to clinical care in the ICU remains undetermined. Beyond total amounts of drainage, sudden increases in chest tube output are of obvious concern. Sudden cessation of bleeding suggests tube occlusion and the potential for accumulating hemopericardium or hemothorax.

Bleeding and hemodilution, whether intraoperative or postoperative, lead to extensive, but variable, use of blood products. Approximately 60% of patients receive allogenic blood products (300–304), accounting for 20% of annual transfusions in the United States (305). Most transfusions are of packed RBCs (PRBCs) and are intended to correct anemia. Historic goals, driven by data relating adverse outcomes to the nadir of perioperative anemia (305), targeted a hemoglobin greater than 10 mg/dL in the postoperative period. However, in observational studies, transfusions are a risk factor for short- and long-term mortality after cardiac surgery (306–311). Mortality risk and the risk of adverse cardiac events increase after only one or two units and is additive with each additional transfused unit of PRBCs (312, 313). Transfusion-related acute lung injury (TRALI) and transfusion-related volume overload are significant problems (270, 314, 315). Transfusion is also associated with increased risk of pneumonia, bacteremia, sternal wound infection, and *Clostridium difficile* after cardiac surgery (303, 316–319). Randomized trials have demonstrated the equivalence or superiority of restrictive transfusion strategies (transfusion trigger hemoglobin ≤ 7–8 mg/dL) compared with liberal strategies (trigger hemoglobin ≤ 9–10 mg/dL) in ICU patients, orthopedic patients with cardiovascular disease, and in active gastrointestinal hemorrhage (320–322). Two randomized controlled trials have examined transfusion goals after cardiac surgery. In the Transfusion Requirements After Cardiac Surgery trial (323), 502 patients were randomized to either a restrictive (maintain hematocrit ≥ 24%) or liberal (hematocrit ≥ 30%) transfusion strategy. There was no difference in mortality or major morbidity, and PRBC use was decreased by 60% by the restrictive strategy. The second trial is the recently published Transfusion Indication Threshold Reduction trial, which randomized 2003 elective cardiac surgical patients to a restrictive (maintain hemoglobin ≥ 7.5 g/dL) or liberal (hemoglobin ≥ 9 g/dL) transfusion strategy (324). In this study, blood utilization was significantly decreased by nearly 40% in the restrictive group (53% of patients received a transfusion vs 92% in the liberal group). There was no difference between the groups in the primary composite outcome of serious infection or ischemic event at 3 months. However, while there was no difference in 30-day mortality, there was an unexplainable but significant difference in all-cause 90-day mortality, a secondary outcome, which favored the liberal strategy (2.6% mortality vs 4.2% in the restrictive group). The mechanism of this unanticipated (and delayed) mortality difference is unclear and warrants further study. However, based on the balance of available data, restrictive transfusion protocols in cardiac surgical patients decrease blood utilization. Targeting a hematocrit goal of 24% appears safe and effectively decreases costs, resource use, and complications (304, 325).

Fresh frozen plasma (FFP), platelets, and factor concentrates are also extensively used, especially in the setting of excessive bleeding. FFP and platelets are each used in about 25% of patients; however, these products cannot be used with impunity. Plasma-containing products confer a higher risk of TRALI than PRBCs, and transfusion of FFP in critically ill surgical patients is associated with increased risk of infection (315, 326). We discuss the management of excessive bleeding in the forthcoming second part of this review.

De-Escalation, Diuresis, and Rehabilitation

As the inflammatory effects of surgery and CPB subside and hemodynamic stability ensues, inotropes and vasoactive agents can be weaned off, usually 6–12 hours after admission to the ICU. Care must be taken to ensure that perfusion remains adequate. Between intraoperative and ICU fluid administration, most patients will gain at least 6 L of volume during the first postoperative day (15). Thus, as the inflammatory response subsides and the myocardium recovers, volume and sodium overload become significant problems. Absent significant vasodilation or an ongoing fluid requirement (usually by the morning of postoperative day 1), low-dose IV diuretics (e.g., furosemide 20 mg every 12 hr) should be started with a typical aim of a net negative fluid balance of 1–2 L daily; this goal should be adapted to individual patient characteristics. Diuresis can begin even in the presence of low-dose inotropic or vasopressor support. To minimize the risk of continued fluid overload, maintenance IV fluids should be avoided if possible (327, 328). Rather, shortly after extubation, the patient's swallowing function should be evaluated and, if safe, oral intake of clear liquids commenced (329). The diet can then be advanced as tolerated.

Once an acceptable response to diuresis is confirmed, the Foley catheter should be removed as soon as possible to minimize infectious risk (330, 331). Central venous catheters represent another potential infectious source and should also be removed expeditiously (332). If there is no evidence of conduction system injury or bradyarrhythmias, epicardial pacing leads, if present, can usually be removed on postoperative day 1 or 2, although there is no harm in leaving them in place longer in the event of an unforeseen arrhythmia (333). In the absence of an air leak, chest drains are removed as soon as output drops to an acceptable volume (e.g., < 100 mL/8 hr), also typically on postoperative day 1 or 2 (334, 335). There is no need for a routine chest radiograph after chest tube removal (336, 337).

Physical therapy and rehabilitation are a priority and should be begun as soon as possible. Patients are mobilized rapidly and often ambulate on postoperative day 1 (224, 338–340). Many patients are ready to leave the ICU within 24–48 hours after surgery (42), but cardiac rehabilitation should be continued upon transfer from the ICU and hospital discharge (341).

Predicting Fast-Track Failure and Complicated Courses

The above discussion reflects the current trend toward "fast-track" management of cardiac surgical patients. However, although "fast-track" management is safe and effective at shortening ICU length of stay, not all patients will progress rapidly through initial postoperative care (42, 342). Risk factors for fast-track failure have been an active area of investigation. Described risk factors include advanced age, preoperative heart failure (New York Heart Association class > 3), American Society of Anesthesiologists class greater than 3, complex operations, long operative times, emergent surgeries, recent acute coronary syndrome, and preoperative renal dysfunction (343–346). Patients with these characteristics will still benefit from most of the strategies described in this review but should be considered at higher risk of complications and managed accordingly.

SUMMARY

The immediate postoperative period after cardiac surgery is a dynamic time, characterized by predictable hemodynamic lability and attendant significant fluctuations in vascular tone, large fluid shifts, and coagulopathy. To achieve the goal of hemodynamic stability, all organ systems must be appropriately managed, with attention paid to ventilatory status, acid-base state, electrolytes, sedation, and pain control. The cardiac intensivist must both manage swings in stability and work to rapidly wean sedation and mechanical ventilation. Clear patterns emerge in the management of open-heart surgical patients, and we have attempted to present a framework for routine management of these patients. In the majority of patients, the systemic inflammatory response resolves within 12–24 hours, allowing rapid de-escalation, and a pivot toward rehabilitation. In the forthcoming second half of this review, we will focus on procedure-specific management issues, management of common complications, and quality improvement in the CSICU.

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Postoperative Critical Care of the Adult Cardiac Surgical Patient: Part II: Procedure-Specific Considerations, Management of Complications, and Quality Improvement

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Objectives: The armamentarium of cardiac surgery continues to expand, and the cardiac intensivist must be familiar with a broad spectrum of procedures and their specific management concerns. In the conclusion of this two-part review, we will review procedure-specific concerns after cardiac surgery and the management of common complications. We also discuss performance improvement and outcome assurance.

Data Source and Synthesis: Narrative review of relative English language peer-reviewed medical literature.

Conclusions: Knowledge of procedure-specific sequelae informs anticipation and prevention of many complications after cardiac surgery. Most complications after cardiac surgery fall into a limited number of categories. Familiarity with common complications combined with a structured approach to management facilitates response to even the most complicated postoperative situations. Standardized care and constant self-examination are essential for programmatic improvement and consistent high-quality care. (*Crit Care Med* 2015; XX:00–00)

Key Words: aorta; cardiac surgical procedures; coronary artery bypass; intensive care; off-pump; postoperative care; quality improvement

The general principles of postoperative management discussed in the first installment of this review are applicable to most cardiac surgical patients. However, many procedures have important idiosyncrasies in the postoperative phase. Knowledge of these procedure-specific concerns is essential for competent care of the full spectrum of cardiac surgical patients. Therefore, we discuss specific aspects of postoperative management after coronary artery bypass graft (CABG) procedures, valve surgeries, ascending aortic and aortic arch procedures, and minimally invasive cardiac operations. We do not discuss other operations such as arrhythmia surgery, adult congenital heart surgeries, pulmonary endarterectomy, management of cardiac trauma or acquired defects, and thoracic transplantation; these highly specialized operations are beyond the scope of this review. Regardless of the surgery performed, after the initial resuscitative phase, attention turns to preventing complications, such as nosocomial infections, deep venous thrombosis, and musculoskeletal deconditioning. Even in the face of optimum care, complications occur after cardiac surgery. Most of these fall into several distinct categories, and knowledge of the pathogenesis and management of these complications can allow rapid rescue of a patient from morbidity or mortality. Finally, consistent performance of a cardiac critical care program depends on a rigorous and ongoing quality improvement process, to identify safety concerns and areas for improvement. These topics are discussed in the conclusion of this review on postoperative critical care of the cardiac surgical patient.

PROCEDURE-SPECIFIC CONSIDERATIONS

CABG

More than 150,000 CABG procedures are performed each year in the United States (1). Durable success depends on graft patency and modification of cardiovascular risk factors. Long-term graft patency has been dramatically improved by the use of arterial conduits (2–4); the left internal mammary artery (LIMA) is the conduit of choice for bypassing the left anterior descending coronary artery (5, 6). Saphenous venous grafts are commonly used to bypass other vessels. Aspirin, at recommended doses of 100–325 mg daily, increases long-term graft patency and reduces mortality, myocardial infarction, stroke,

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bowel infarction, and renal failure after CABG (2, 7–10). Aspirin should be administered to all patients preoperatively and should be re-administered (or started if not given preoperatively) within 6 hours postoperatively (once immediate bleeding has subsided) and continued indefinitely (2). Clopidogrel or other antiplatelet agents (e.g., prasugrel and ticagrelor) should not be routinely added to aspirin after CABG (2, 11), but these agents are options in aspirin-allergic patients. If at all possible, nonaspirin antiplatelet agents should be held prior to elective cardiac surgery to decrease the risk of major postoperative bleeding. There appears to be no difference in the rates of bleeding between clopidogrel and ticagrelor, which should both be held for at least 5 days preoperatively if at all possible (12–15). The rates of bleeding are substantially higher with prasugrel, which should be held for at least 7 days preoperatively (16). The exception is in patients with recently placed coronary stents, which must remain patent. In these patients, dual antiplatelet therapy (e.g., the combination of clopidogrel and aspirin) should be continued throughout the perioperative period to minimize the chance of in-stent thrombosis. Increased bleeding should be anticipated in this group of patients.

All CABG patients should be treated with a 3-hydroxy-3methylglutaryl-coenzyme A reductase inhibitor (statin). Statins decrease atrial fibrillation, adverse coronary events, graft occlusion, renal dysfunction, and all-cause mortality after cardiac surgery (2, 17–21). In the absence of contraindications (hepatic dysfunction, myositis, and rhabdomyolysis), a statin should be started as soon as the patient can tolerate oral medications and continued indefinitely. The mechanism of the salutary effects of statins is unclear (22, 23), as is the optimum choice and dose of statin; much of the data are based on atorvastatin (40–80 mg daily). Although the benefits of statins have primarily been shown after CABG, there may be benefit to treating other cardiac surgical patients; for example, a single-center study suggested benefit of statins on long-term survival after aortic valve replacement with a biologic prosthesis (although not with mechanical valves or mitral valve replacement [MVR]) (24).

Preoperative administration of β-blockers has been used as a quality metric in cardiac surgery, based on retrospective data suggesting decreased mortality with this intervention (25, 26). More recent data have questioned the role of preoperative β-blockade (27). Postoperatively, inotropic requirements may preclude immediate β-blockade, but current guidelines suggest that β-blockers should be started as soon as possible after CABG (2). β-blockers reduce the risk of postoperative atrial fibrillation and may also reduce myocardial ischemia and mortality (25, 28, 29). It is reasonable to start with a low dose (e.g., metoprolol 12.5–25 mg twice daily) and increase as tolerated by heart rate and hemodynamics.

The role of angiotensin-converting enzyme inhibitors (ACE-Is) or angiotensin receptor blockers (ARBs) after cardiac surgery is controversial because they have been associated with perioperative vasoplegia, hypotension, and postoperative renal dysfunction (30–33). However, it is recommended that patients who were on preoperative ACE-Is or ARBs be restarted on therapy as soon as stable, and that de novo ACE-Is or ARBs be started upon stability in patients who have decreased left ventricular (LV) ejection fraction, diabetes, or chronic kidney disease (2, 31, 34–36).

Off-Pump CABG

Conventional CABG requires cardiopulmonary bypass (CPB), cross-clamping of the aorta, and cardioplegic arrest, all of which carry significant postoperative consequences. In an attempt to avoid these maneuvers, techniques have been developed for off-pump CABG (OP-CABG). However, despite the theoretical benefits, there are as yet no convincing data that OP-CABG is superior to conventional (on-pump) CABG; indeed, long-term graft patency, complete revascularization, and overall survival may be better with conventional CABG (2, 37–41). Still, OP-CABG comprises 15–20% of all CABG procedures in the United States (42). Compared with conventional CABG, OP-CABG patients are less coagulopathic, have less bleeding, and require fewer transfusions; some studies have reported fewer immediate postoperative respiratory and renal complications than after on-pump CABG (40, 43, 44). The rate of immediate perioperative strokes appears to be reduced, and OP-CABG may have a particular niche when aortic atherosclerosis precludes cross-clamping (45, 46). It should be noted, however, that there appears to be no difference between OP-CABG and conventional CABG in risk of renal injury requiring dialysis, risk of stroke or risk neurocognitive dysfunction at either 30 days or 1 year postoperatively (40, 47).

OP-CABG requires optimal positioning and stabilization of a beating heart to complete the bypass anastomoses. These maneuvers can cause significant hemodynamic compromise, due to cardiac compression and a functional decrease in cardiac preload (48). This is treated by intraoperative administration of fluid, which can result in significant volume overload. Tolerance for postoperative bleeding should be less after OP-CABG than conventional CABG, and in the absence of CPB-induced coagulopathy, any bleeding is more likely to be from an anastomosis or an uncontrolled bleeding vessel and require operative repair. The risk of incomplete coronary revascularization is present, and vigilance for ischemia is required (40, 41, 49, 50).

Cardiac Valve Surgery

Valve surgery is riskier than CABG, with unadjusted mortalities increased by nearly two-, three-, and four-fold for aortic, mitral, and tricuspid replacement, respectively (1). Combination of valve procedures with CABG further increases operative complexity. Valve repair, if feasible, obviates the concern of valve thrombosis. After replacement with a bioprosthetic valve, antiplatelet therapy with aspirin is usually sufficient although some recommend short-term anticoagulation. Mechanical prostheses require life-long anticoagulation; this is typically started on postoperative day 1 or 2. Anticoagulation practices vary, with some surgeons preferring to use systemic heparin followed by oral vitamin K antagonists, and others forgoing

heparin and simply starting oral anticoagulation (51). Postoperative management is informed not only by characteristics of the repair itself but also by the adaptive cardiac response to the underlying valve pathology.

Mitral Valve. In the United States, approximately 6,500 isolated MVRs and 9,000 isolated mitral repairs are performed yearly (1). An additional 7,500 mitral procedures are performed concomitantly with CABG. The management of mitral surgery patients is complex because the physiology of mitral disease can predispose patients to both LV and right ventricular (RV) failure in the postoperative period. Correction of severe mitral regurgitation by mitral repair or replacement can cause a dramatic increase in LV afterload, precipitating LV failure and decreased cardiac output (52). The increase in LV afterload has been thought to be due to the elimination of regurgitation into the left atrium as a low resistance LV ejection pathway although more recent studies have questioned this framework (53–55). Regardless, it remains a tenet of care to provide appropriate LV afterload reduction and inotropic support to prevent the development of LV failure and unnecessary strain on the repair (56–58). Long-standing mitral disease can cause pulmonary hypertension and RV compromise; the stress of surgery and CPB can incite acute postoperative RV failure. Inhaled pulmonary vasodilators may be useful if RV failure develops (59). A unique feature of mitral valve repair is the development of dynamic LV outflow tract obstruction due to systolic anterior motion (SAM) of the anterior leaflet of the mitral valve, which is typically due to a mismatch between leaflet tissue and mitral annular size and occurs in approximately 5% of patients after mitral repair (60–63). SAM occurs when the anterior leaflet or chordae of the mitral valve paradoxically moves toward the interventricular septum during systole, causing dynamic LV outflow tract obstruction, reduced cardiac output, and potential hemodynamic collapse (63). SAM is exacerbated by an underfilled, hyperdynamic LV, thus management consists of adequate volume resuscitation, avoidance of inotropes, minimizing tachycardia, and early β-blockade (61, 64, 65). With these measures, surgical revision is rarely required. Atrioventricular groove disruption is a devastating complication of MVR, which occurs in 1.2% of replacements and confers a mortality of roughly 75% (66, 67). Usually, this is apparent in the operating room when significant bleeding occurs from behind the heart upon volume loading and ejection against systemic pressure, but on occasion, it does not manifest until the ICU. Atrioventricular groove disruption should be suspected when massive bleeding occurs after mitral surgery, especially if the surgeon reported extensive debridement of a calcified mitral annulus. Surgical repair is mandatory.

Aortic Valve. Over 30,000 isolated aortic valve replacements (AVR) are performed each year in the United States, with an additional 20,000 combined procedures (AVR-CABG; AVR/MVR) (1). Perioperative mortality continues to decrease, despite an increasingly complex patient population (68). Appropriate fluid management is essential, especially when surgery is performed for aortic stenosis (AS), as the hypertrophied LV is exquisitely sensitive to preload. Blood pressure control after aortotomy is important to limit stress on the aortic suture line. Any sudden increase in bleeding should raise concern regarding the integrity of the aortotomy closure. The postoperative electrocardiogram must be evaluated for conduction disturbances and ischemia, as injury to the conduction system occurs not infrequently, often from placement of sutures through conduction tissue (69). Conduction disturbances typically manifest within the first three postoperative days (70). Many patients require epicardial pacing for transient conduction disturbance; most of these will recover. A minority of patients (≈2–7%) will require a permanent pacemaker (71, 72); pacemaker placement should usually be delayed for 5–7 days post surgery to allow adequate time to prove that the conduction system will not recover (73–75). Malpositioned aortic valve prostheses can occlude either coronary ostia; the right is particularly at risk (76, 77). Coronary occlusion should be suspected in the face of right or LV failure or refractory ventricular arrhythmias. Manipulation of the aorta is a risk factor for cerebral embolism, and a postoperative neurologic examination should be performed once feasible.

Tricuspid and Pulmonic Valves. Tricuspid and pulmonic procedures are less common than other valve operations. Most tricuspid surgeries are performed in concert with another procedure. Mortality after tricuspid surgery is approximately 8% (78). Tricuspid replacement carries a higher risk of mortality than tricuspid repair; major causes of mortality after tricuspid operations are heart failure and injury to the conduction system (79). The risks of RV failure, renal failure, and mortality are higher after valve replacement than repair although this may be due to preoperative patient characteristics (80). Pulmonic valve procedures are rare in adults, but are generally well tolerated. Specific postoperative concerns focus on RV function.

Ascending Aorta and Arch Surgery

Ascending aortic procedures include aneurysm repair with interposition tube grafts, aortic root replacements, aortic arch replacements, and emergent repair of dissections. Complications specific to aortic surgery are predominantly neurologic and hemorrhagic, although if the aortic root is replaced, whether in a valve-sparing fashion or not, the complications of aortic valve surgery can occur as well (81). Neurologic injury can result from embolization of atherosclerotic debris or entrainment of air into the open arch or head vessels (82). Arch procedures often use hypothermic circulatory arrest with temperatures as low as 18°C to allow periods of cerebral and somatic ischemia. Even with hypothermic protection, global neurologic and somatic injury may result from these ischemic periods. Delayed awakening after arch procedures may be predicted by intraoperative regional cerebral oxygen saturation measured by near-infrared spectroscopy (83). When hypothermic circulatory arrest is used, the associated hypothermia and long CPB times can worsen coagulopathy and contribute to postoperative bleeding (84, 85). As with any aortic surgery, blood pressure should be tightly controlled to limit the risk of anastomotic disruption. At a minimum, arterial blood

pressure should be monitored via arterial catheterization of the right upper extremity (typically the right radial artery), as this will reflect perfusion pressure to the coronary vessels and proximal aortic arch, including the right internal carotid, which arises from the same origin (the brachiocephalic trunk) as the right subclavian artery. It is often useful to monitor arterial blood pressure in another site, such as the left radial artery or either femoral artery. Any evidence of asymmetric perfusion (e.g., markedly different blood pressures in different locations, absence of pulses in an extremity, or asymmetric mottling) should raise suspicion for iatrogenic dissection or vascular occlusion. In aortic root replacement procedures (e.g., valve-sparing root replacement or replacement of the aortic root, valve, and ascending aorta with a composite prosthetic valve and graft [the Bentall procedure]), the coronary arteries are reimplanted into the graft (86, 87) and coronary occlusion or kinking with resultant myocardial ischemia is possible. This typically involves the right coronary artery, and new RV failure should raise concern for right coronary artery occlusion (88). Anticoagulation is required if a mechanical valve prosthesis is used in an aortic root replacement; this is typically started once the risk of bleeding has passed, on postoperative day 1 or 2. Aortic surgery patients are at higher risk of developing postoperative acute respiratory distress syndrome (ARDS) than other cardiac patients; empiric lung-protective mechanical ventilation is suggested (89, 90).

Minimally Invasive Cardiac Surgery

There is increasing interest in minimally invasive cardiac surgery, using small incisions, endoscopic techniques, robotic technology, and percutaneous approaches to minimize surgical insult and achieve shorter recovery times. The most common of these is probably the "mini-mitral," which involves replacement or repair of the mitral valve via a small right thoracotomy (91). Minimally invasive direct coronary artery bypass and endoscopic coronary artery bypass both use a small left anterior thoracotomy for off-pump bypass of the LAD with the LIMA. The LIMA is harvested via open technique or thoracoscopic techniques, respectively. Robotic cardiac surgery is also growing in popularity, especially for mitral procedures (92). Minimally invasive procedures carry many of the same complications and considerations as their conventional counterparts, with a few modifications. Pain can be a significant issue due to the rib retraction required for exposure. Less bleeding is expected with minimally invasive procedures, particularly robotic procedures. However, the limited exposure necessitated by smaller incisions can complicate intraoperative hemostasis and accordingly, the threshold of concern for bleeding should be lower: atelectasis is a common problem because most minimally invasive approaches depend on some period of single lung ventilation. With femoral access for perfusion, and long perfusion times, peripheral arterial pulses and lower limb perfusion need to be carefully monitored (92).

Techniques for percutaneous approaches to valve replacement are another recent development and are rapidly being integrated into clinical practice. Transcatheter aortic valve replacement (TAVR) is an option for severe AS in high-risk or inoperable patients (93–96). The postoperative management of TAVR patients has recently been reviewed (97), and many of these patients do not require an ICU admission, but a few salient points deserve mention. Like all patients with LV hypertrophy due to AS, TAVR patients may be very volume sensitive. Stroke is a major risk, and postoperative neurologic assessment is important (98–100). Conduction problems are common; up to 20% of TAVR patients will require permanent pacemakers (93). Vascular access points need to be assessed for hematoma, especially in the face of hypotension (93, 97). The requisite contrast to guide valve placement can contribute to acute kidney injury (AKI), as can bleeding and hypotension, and renal function and urine output should be closely monitored (101). Catastrophic complications can occur after TAVR, including aortic rupture and coronary obstruction (102, 103).

MANAGEMENT OF COMMON PROBLEMS AND COMPLICATIONS

Although the majority of cardiac surgery patients have an uncomplicated postoperative course, there are a set of problems and complications which predictably and frequently occur. Anticipation of problems and appropriate management allows "rescue" from otherwise unsurvivable situations (104, 105).

Excessive Bleeding

Given the coagulopathy associated with CPB, some postoperative bleeding is expected (106). In most cases, both the coagulopathy and the expected minor bleeding will resolve shortly after surgery, and no blood products will be required. But approximately 10% of patients have "excessive" postoperative bleeding, which is associated with adverse outcomes and increased costs (107, 108). Unfortunately, as discussed in the first part of this review, the definition of "excessive" varies substantially. Chest tube drainage is easily quantifiable and forms the basis for most bleeding definitions. Amounts ranging from greater from 200 mL/hr to 1,500 mL over 8 hours have been suggested as "excessive" (107, 109–111). An alternative scheme identifies excessive bleeding as more than 400 mL in the first hour, 300 mL/hr for the first 2 hours or 200 mL/hr for three consecutive hours (110). A recent expert panel defined "severe" bleeding as postoperative chest tube blood loss of 1,001–2,000 mL in the 12 hours or transfusion of 5–10 U of packed RBCs (PRBCs) or fresh frozen plasma (FFP). "Massive" bleeding was defined as more than 2,000 mL of chest tube bleeding in the 12 hours or need for more than 10 U of PRBCs or FFP (112). Regardless of definition, postoperative bleeding must be taken seriously: bleeding of more than 200 mL/hr in 1 hour, or 1,000 mL in the first 24 hours, is associated an increased risk of death (113, 114).

Risk factors for excessive bleeding include age, preoperative anemia, emergent or complex operations, use of an IMA, long CPB time, decreased cardiac function, lower body mass, and male sex; surgeon-specific factors (e.g., attention to hemostasis) also contribute to risk of bleeding (115–117). Preoperative dual antiplatelet therapy (aspirin and clopidogrel, prasugrel, or

ticagrelor) confers a major bleeding risk of approximately 15% (15, 118). Although guidelines suggest discontinuing dual antiplatelet therapy 5 days before surgery, this is often impossible in emergencies (119). The Papworth Bleeding Risk Score, derived from a prospective database of more than 11,000 patients, identifies five risk factors and assigns a value of either 0 or 1 point to each: 1) surgery priority (elective [0] or emergent [1]); 2) surgery type (CABG/single valve [0] or all others [1]); 3) aortic valve disease (none [0] or present [1]); 4) body mass index (≥ 25 [0] or < 25 [1]); and 5) age (< 75 years [0] or ≥ 75 years [1]) (120). Patients are rated as low (0 points), medium (1–2 points), and high risk (≥ 3 points), corresponding to rates of excessive bleeding of 3%, 8%, and 21%, respectively. Performance of the Papworth Score has been mixed (121).

Management of a bleeding patient requires attention to multiple details. Crystalloid administration should be limited to prevent hemodilution, and hypothermia and acidosis rapidly corrected (122, 123). As blood pressure, rather than cardiac output, drives bleeding, systolic blood pressure should be no higher than 90–100 mm Hg (124–126) in the early postoperative period. Short-acting agents like nitroglycerin or nitroprusside can be used to lower blood pressure if needed although maintaining adequate cerebral and somatic perfusion is essential. Increasing positive end-expiratory pressure may help control bleeding (127, 128). These are all adjuncts, however, and appropriate support with blood products is essential (115, 116). On the basis of trauma literature, it is reasonable to use a ratio of PRBC-to-FFP and platelets of 2:2:1 in the actively bleeding patient (122, 129). This ratio should be tailored based on assays of coagulation function (130). Thromboelastography may help guide blood product administration as it gives insight into the physiologic activity of clotting factors, platelet function, and fibrinogen and plasminogen activity (131, 132). In the setting of hypofibrinogenemia (< 100 mg/dL), administration of cryoprecipitate can be useful and can spare volume compared with FFP administration (130). Some advocate targeting a higher fibrinogen threshold (e.g., 150 mg/dL) (131). Prothrombin complex concentrates are increasingly used in bleeding cardiac surgical patients; there are no randomized controlled trials supporting this off-label practice (131, 133). There are more data regarding the off-label use of recombinant factor VIIa to treat severe hemorrhage after cardiac surgery (134–136). Factor VIIa does seem to decrease bleeding, but there are no data that mortality is reduced (137, 138), and the potential benefit must be balanced against the real risk of thrombotic complications, the incidence of which ranges between 25% and 50% in postoperative cardiac patients (135, 137, 139). Surgical exploration for uncontrolled hemorrhage is required approximately 3% of cases (117, 140, 141). Surgical re-exploration should be prompt; delays are associated with adverse outcomes (141). In the unstable patient, exploration can occur at the bedside (142).

Refractory Shock

Refractory postcardiotomy shock can manifest in the operating room as failure to separate from CPB, but typically presents in the ICU as either sustained hypotension and hypoperfusion, or sudden hemodynamic collapse. Successful management depends on accurate identification of the cause and appropriate intervention. In the face of an inadequate heart rate or heart block, epicardial pacing at a faster rate can significantly improve cardiac output. Similarly, termination of a supraventricular arrthymia can quickly normalize hemodynamics. Chest x-ray or ultrasound can rule out tension pneumothorax, an undrained hemothorax, or tamponade. Both pulmonary artery catheters (PAC) and transesophageal echocardiography (TEE) can be extremely helpful in patients with undifferentiated shock. The algorithm presented in **Figure 1** summarizes an approach to refractory shock.

Vasoplegic Syndrome. Although many patients need low-dose vasopressor support after cardiac surgery, vasoplegic syndrome, first described in the 1990s, is an extremely low systemic vascular resistance state that occurs in 5–25% of patients after CPB, requires high-dose vasopressors (e.g., > 0.1 μg/kg/min of norepinephrine), and confers a significant increase in morbidity and mortality (32, 143, 144). Vasoplegia is thought to be due to an exaggerated systemic inflammatory response and is associated with preoperative ACE-inhibitors and ARB use, longer CPB times, preoperative LV dysfunction, and blood transfusion. Catecholamine vasopressors are first-line therapy for vasoplegia; norepinephrine is probably the agent of choice. Many cases of vasoplegia, however, are refractory to catecholamines. The addition of vasopressin, at doses up to 0.04 U/min, is effective for both prevention and treatment of vasoplegia (145, 146). Higher doses of vasopressin have been associated with mesenteric ischemia. In refractory vasoplegia, an infusion of methylene blue (2 mg/kg bolus followed by 0.5 mg/kg/hr for 6 hr) has been reported to reverse hypotension although data on this approach are limited (147, 148).

Cardiac Tamponade. Tamponade should always be suspected in the setting of postoperative low cardiac output. Unlike "medical" tamponade, postoperative cardiac tamponade can result from a relatively small posterior pericardial fluid collection with associated compression of an adjacent cardiac chamber; low-pressure chambers (e.g., the atria and the RV) are particularly susceptible (149). In the early postoperative period, pericardial collections are typically undrained blood or clot; inflammatory pericardial effusions and tamponade can develop later (5–7 d) after surgery (150). Diagnosis can be challenging because classic signs, such as pulsus paradoxus, are frequently absent. Similarly, central venous pressure need not be elevated although an increasing central venous pressure in the face of hypotension and low cardiac output should be concerning. Suspicion for pericardial fluid accumulation is also warranted when chest tube drainage abruptly ceases. Emergent echocardiography may be helpful although the sensitivity of transthoracic echocardiography is poor, and even a "normal" transthoracic echocardiogram cannot exclude tamponade (151). Classic echocardiographic findings of tamponade are frequently absent, and small collections of pericardial fluid can cause localized compression of cardiac chambers with impressive hemodynamic effects (149). Transesophageal

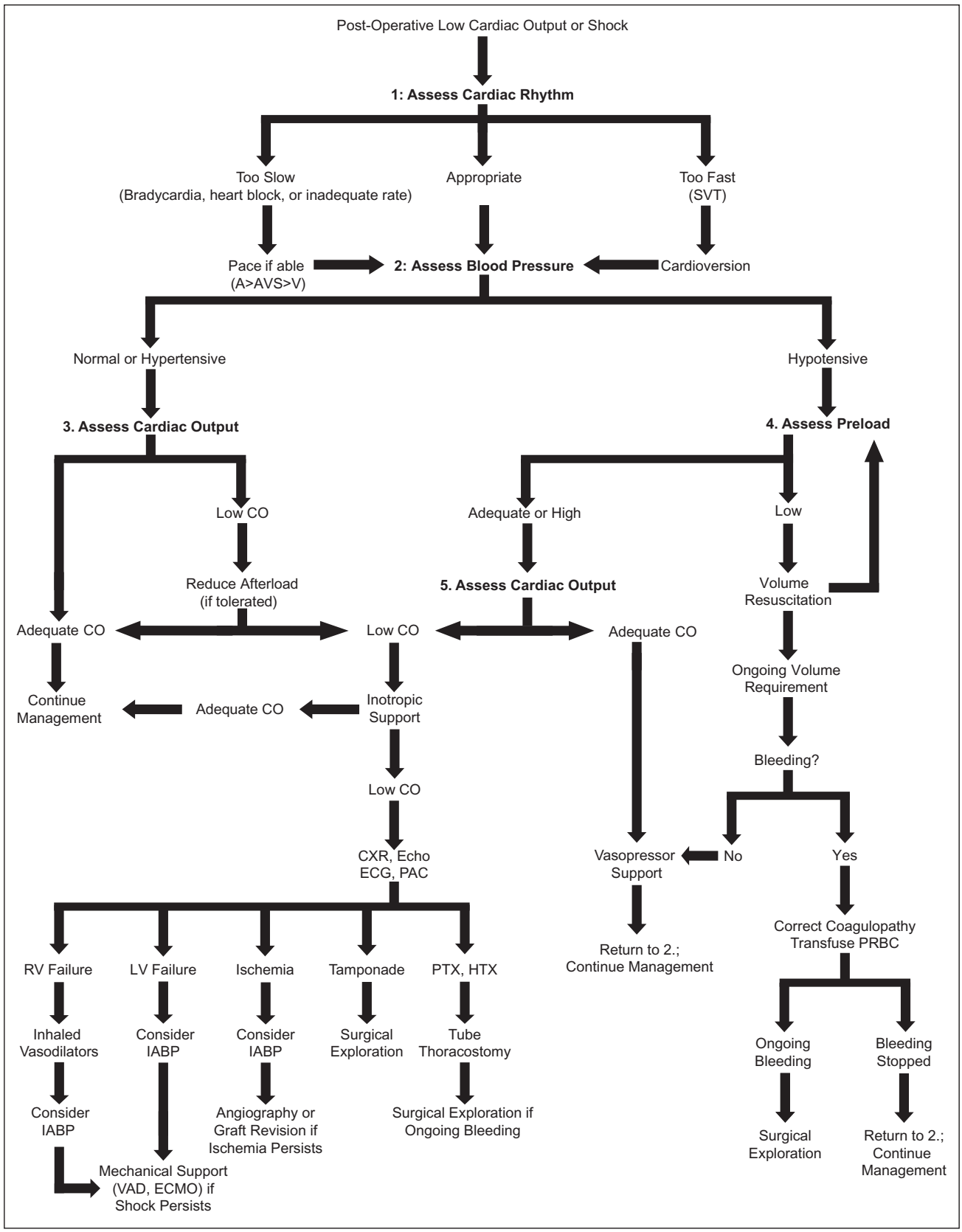

Figure 1. Algorithm for management of low cardiac output and refractory shock after cardiac surgery. This is one approach to the management of low cardiac output and refractory shock after cardiac surgery. It is meant to be illustrative of key thought processes and important factors to consider. See text for detailed discussion. A = atrial; AVS = atrial-ventricular sequential; CO = cardiac output; CXR = chest x-ray; ECG = electrocardiogram; Echo = echocardiogram; ECMO = extracorporeal membrane oxygenation; HTX = hemothorax; IABP = intra-aortic balloon pump; LV = left ventricle; PAC = pulmonary artery catheter; PRBC = packed red blood cells; PTX = pneumothorax; RV = right ventricle; SVT = supraventricular tachycardia; V = ventricular; VAD = ventricular assist device.

echocardiogram is more sensitive (151), and if tamponade is confirmed, or if suspicion is sufficiently high, early surgical exploration and drainage are indicated. Hemodynamic collapse is an indication for emergent ICU thoracotomy (142).

LV Failure. LV failure after cardiac surgery can result from transient dysfunction ("stunning") due to prolonged CPB and cross clamp times, coronary malperfusion, valve pathology, and changes in afterload or preload. PACs can help assess volume status and response to volume loading although with the caveats discussed in the first part of this review. TEE can also assist in assessing volume status and can also identify segmental wall abnormalities consistent with ischemia and valve stenosis or insufficiency. Electrocardiography may also be helpful in identifying ischemia. Optimization of preload and afterload is essential. If the patient is hypertensive, afterload reduction with a short-acting agent such as nitroprusside can dramatically improve cardiac output. If cardiac output remains low, increased inotropic support is required (152). As discussed in the first part of this review, there are no clear data guiding inotrope choices (153). In hypertensive patients with low cardiac output, dobutamine or a phosphodiesterase inhibitor (e.g. milrinone) is an option; the shorter half-life of dobutamine can facilitate titration. In hypotensive patients, norepinephrine or epinephrine is an appropriate choice. Alternatively, the combination of norepinephrine and dobutamine is effective and may be safer than epinephrine (154). If ischemia is suspected, caution is warranted with inotropes because they increase myocardial oxygen demand. Coronary angiography or return to the operating room for revascularization may be indicated. If these measures fail to restore adequate perfusion, mechanical circulatory support should be considered.

RV Failure. RV failure can be provoked by postbypass stunning, coronary malperfusion, or LV failure. In addition, the RV is susceptible to acute changes in afterload and preload, and a sudden increase in either can precipitate acute RV failure (155). PACs and TEE are extremely helpful in assessing RV function; the PAC is especially useful (152). If RV failure is identified, inhaled pulmonary vasodilators (nitric oxide or prostacyclin) can reduce RV afterload and significantly improve RV function (59, 156–158). Hypoxemia, hypercarbia, and acidemia all increase pulmonary vascular resistance (PVR) and can potentiate RV failure. Maintaining a PaO_2 greater than 90 mm Hg and a pH greater than 7.45 can markedly improve RV function (156, 159–161). Excessive positive end-expiratory pressure can also increase PVR and RV afterload although this may be counterbalanced by improved oxygenation and decreased hypoxic vasoconstriction. RV preload should be considered, and if the RV is massively dilated and impairing LV filling, efforts should be made to remove intravascular volume via diuresis or ultrafiltration (161). If these measures are unsuccessful, additional inotropy may be required. Dobutamine and milrinone increase cardiac output and have pulmonary vasodilatory effects, making them attractive agents. Dobutamine should be used at low doses (2–5 μg/kg/min) because higher dose ranges do not further reduce PVR (161). Milrinone and other phosphodiesterase inhibitors are effective at decreasing PVR and increasing cardiac output, but their long half-lives complicate titration. Both milrinone and dobutamine can cause or worsen hypotension due to peripheral vasodilation. This is problematic because maintaining adequate blood pressure is important to preserve RV perfusion (156, 161), and dobutamine or milrinone may need to be used in conjunction with peripheral vasoconstrictors such as norepinephrine or vasopressin. Catecholamine vasopressors will increase PVR in addition to systemic vascular resistance, potentially worsening RV function. Some data suggest that vasopressin increases systemic vascular resistance with either no effect on PVR or a pulmonary vasodilatory effect, making vasopressin potentially attractive as a vasopressor in RV failure (162–164). If RV failure is refractory to these measures, mechanical support should be considered (165–167).

Mechanical Circulatory Support. In refractory heart failure, whether LV or RV, if optimization of preload, afterload, inotropic support, and vasopressors support do not restore adequate perfusion, mechanical circulatory support may be indicated. Because high inotrope doses are associated with worse outcomes in postcardiotomy shock, mechanical support should be considered early in the course of refractory shock, before multiorgan dysfunction develops (168, 169). Options for mechanical circulatory support are summarized in **Table 1**.

An intra-aortic balloon pump (IABP) is often first-line mechanical support, especially when coronary ischemia is suspected (170). IABP placement is contraindicated in the setting of aortic dissection or aortic insufficiency (199). Insertion is typically via the femoral artery and support is usually started at a 1:1 augmentation ratio (IABP inflates during every cardiac cycle). This can be changed to 1:2 or 1:3 during weaning or in the face of significant tachycardia. IABPs inflate during diastole, increasing diastolic blood pressure (and theoretically, coronary perfusion), and deflate during systole, decreasing LV afterload. Although IABPs have strong physiologic rationale, they do not improve mortality in cardiogenic shock after acute myocardial infarction (171) and have not been rigorously studied after cardiac surgery. Nevertheless, they are a mainstay of mechanical support, and are commonly used, both when encountering difficulty separating from CPB, and postoperatively in the ICU (172). The need for IABP support is associated with a significant increase in perioperative mortality (200). Because delays in IABP insertion for refractory shock are associated with poor outcomes, some have advocated preoperative insertion in high-risk patients (LV ejection fraction < 35%) although not all studies agree on this point (201–203). Ideally located with the tip just distal to the left subclavian artery, IABPs are frequently malpositioned: at least one visceral artery is occluded in 97% of patients (173). Distal perfusion of the leg also needs to be carefully monitored. Persistent shock, acidosis, lactate production, high inotrope/vasopressor requirements, and oliguria after IABP insertion are all predictors of IABP failure (204).

If hemodynamics or perfusion derangements are not rapidly corrected by IABP insertion, or if IABP use is contraindicated or the patient is too unstable to attempt IABP placement,

TABLE 1. Options for Mechanical Circulatory Support after Cardiac Surgery

Device	Advantages	Disadvantages	Comments	References
IABP	Easily inserted; commonly used; familiar to many providers; strong physiologic rationale	Limited increase in cardiac output (0.5–1 L/min); occlusion of mesenteric/renal arteries; impaired distal leg perfusion; no data showing improved mortality	Second-line support after inotropes; did not improve mortality in acute myocardial infarction with cardiogenic shock	170–173
Abiomed BVS	High levels of cardiac support (> 4–5 L/min); Can be used as RVAD or LVAD	OR insertion required; requires anticoagulation; limited data	Pulsatile pneumatic pump; predominantly replaced by centrifugal pumps.	168, 174–176
Impella	Decompresses LV; surgical insertion: impella 2.5 and 5; peripheral insertion: only impella 2.5 (with fluoroscopy); does not require anticoagulation	OR/catheterization laboratory insertion required; easily malpositioned; LV support only; limited data	Device positioned across aortic valve; no mortality data	168, 174, 177–181
TandemHeart	Can be used as LVAD or RVAD; BiVAD if centrally placed; high levels of cardiac support (> 4–5 L/min)	OR/catheterization laboratory insertion required. Peripheral insertion requires trans-septal puncture; anticoagulation required; no respiratory support; limited data	No mortality data	168, 174, 182, 183
Centrimag	Can be used as LVAD, RVAD, or BiVAD; high levels of cardiac support (> 4–5 L/min); approved for up to 30 days of use	OR insertion required; anticoagulation recommended; limited data.	Centrifugal pump; can be used with oxygenator in ECMO configuration	168, 174, 184–186
Venoarterial-ECMO	Complete cardiopulmonary support; central or peripheral cannulation ± LV decompression; rapid percutaneous cannulation possible in ICU; may decompresses heart	May increase LV afterload; cerebral and coronary hypoxemia if pulmonary dysfunction and LV ejection; risk of systemic thromboembolism; risk of impaired distal leg perfusion if femoral artery used; anticoagulation required	Increasingly used as third-line support after inotropes and IABP	168, 174, 186–198

IABP = intra-aortic balloon pump, RVAD = right ventricular assist device, LVAD = left ventricular assist device, OR = operating room, LV = left ventricular; BiVAD = biventricular assist device, ECMO = extracorporeal membrane oxygenation.

additional mechanical support strategies should be considered. These include either a temporary ventricular assist device (VAD) or venoarterial extracorporeal membrane oxygenation (VA-ECMO). This is a dire circumstance; although more than 50% of postcardiotomy shock patients can be weaned from mechanical support, only 25% are ever discharged to home, and only 15–30% of these patients survive beyond 1 year (174,205–207). Notably, the emergent initiation of mechanical support for refractory shock differs from the use of long-term implantable devices for end-stage heart failure, which is increasingly used to bridge patients to transplantation or as destination therapy (208). The ICU management of implantable continuous flow LV assist devices was recently reviewed in this journal (209).

Several temporary VAD systems are available and have been recently reviewed elsewhere (168, 174). Options include pneumatic pumps (e.g., Abiomed BVS, Abiomed, Danvers, MA) (175, 176), axial flow pumps (e.g., Impella system, Abiomed) (177–181), and centrifugal pumps (e.g., TandemHeart, CardiacAssist, Inc, Pittsburgh, PA [182, 183]; Centrimag, Thoratec, Pleasanton, CA [184, 185]). There are no data to guide choice of specific VAD, and this decision primarily depends on institutional and surgeon preference. Many of these need to be implanted either in the operating room or in the cardiac catheterization laboratory under fluoroscopic guidance, limiting ability for immediate deployment in the ICU. In addition, many of these devices can provide only single ventricular support, mandating a second device in the event of biventricular failure.

Increasingly, some centers are proceeding directly to VA-ECMO as the first-line circulatory support modality in the

face of refractory postcardiotomy shock or postoperative cardiac arrest (186–189). The ECMO circuit typically consists of a centrifugal pump, membrane oxygenator, and heat exchanger. VA-ECMO can be rapidly instituted either via central cannulation (via pre-existing sternotomy) or peripherally using percutaneous techniques, which do not require fluoroscopic guidance. Of note, some devices (e.g. the Centrimag) can function both in VAD or ECMO configurations (184, 186). VA-ECMO allows the provision of both immediate complete biventricular circulatory support and respiratory support, which can be an advantage in severe shock with respiratory failure (190). Percutaneous cannulation is typically performed with the inflow cannula placed in the femoral vein and the outflow cannula in the femoral artery. This configuration, while facilitating rapid ECMO initiation, does increase LV afterload, and can lead to LV distention (168, 187). In the setting of pulmonary dysfunction and significant remnant native cardiac output, the retrograde aortic flow produced by femoral arterial cannulation can lead to mixing in the aortic arch and cerebral hypoxemia (190). Femoral arterial cannulation can significantly impair distal perfusion to the involved leg; perfusion needs to be carefully monitored, and if insufficient, consideration given to inserting a small antegrade perfusion cannula in the femoral artery. Some groups have advocated combining VA-ECMO with IABP support to reduce LV afterload, improve coronary perfusion, and restore a measure of pulsatility to the circulation; the benefit of this approach has not yet been proven (191–193). Once on VA-ECMO, typical target flow rates are 60–80 mL/kg/min (194). Inotropes are typically minimized or discontinued to "rest" the myocardium, and vasopressors used as needed to support blood pressure. Anticoagulation is typically required on VA-ECMO support, due to the risk of arterial and venous thromboembolization (195), but may be precluded if the risk of hemorrhage is high. Appropriate hemoglobin targets on VA-ECMO are not known; in an attempt to maximize oxygen-carrying capacity, many centers target near-normal hemoglobin levels although recent data suggest that more conservative transfusion goals may be safe in veno-venous ECMO (196, 197, 206). Weaning trials should be conducted once evidence of cardiac function has returned (198).

Postoperative Cardiac Arrest

Cardiac arrest can occur as progression from refractory postoperative shock, or as an unheralded event. Resuscitation protocols should be immediately initiated; however, the applicability of Advanced Cardiac Life Support protocols is limited in postoperative cardiac patients. Specific guidelines for the ICU resuscitation of postoperative cardiac arrest, known as *Cardiac Advanced Life Support-Surgical* or *Cardiac Surgery Unit-Advanced Life Support* in the United States and United Kingdom, respectively, have been published (210, 211). These include up to three immediate attempts at defibrillation of either ventricular fibrillation or ventricular tachycardia. Timely defibrillation is critical. Similarly, epicardial pacing can be attempted for asystole or severe bradycardia if epicardial leads are in place. Attempts at defibrillation or pacing should take precedence over chest compressions unless a defibrillator/pacer is not immediately available (i.e., within 1 min) (210, 211). In most cases, after unsuccessful defibrillation/pacing or in the absence of a shockable rhythm, chest compressions should be performed although significant injury can be incurred from chest compressions due to disruption of suture lines, cardiac laceration by sternal edges, and sternal fracture (210). Chest compressions should generally not be performed in patients with VADs or on ECMO because compressions can dislodge cannulae and interfere with device function. Boluses of epinephrine or vasopressin should be used with caution because they can cause severe hypertension in the event that a regular rhythm is rapidly restored, with resultant stress on anastomoses or aortotomies. If there is no response to resuscitative measures within 5 minutes of the arrest (or three shocks), emergency resternotomy and internal cardiac massage should be performed (210, 212). Equipment for emergency resternotomy should be immediately available. Earlier resternotomy should be considered for pulseless electrical activity arrest, which may be due to tamponade, tension pneumothorax, or intrathoracic hemorrhage and for which emergent chest exploration in the ICU can be lifesaving (142, 213). In cardiac arrest that persists despite resternotomy, ECMO may be initiated as a salvage measure (214); a primed ECMO circuit on standby in the ICU can facilitate rapid deployment.

Neurologic Injury

Cardiac surgery is associated with an array of neurologic complications, ranging from mild cognitive impairment to catastrophic cerebrovascular accident (215, 216). After CABG, the incidence of stroke is nearly 4%; this reaches nearly 10% after complex valve or aortic surgery (217). Most of these are embolic and occur in the postoperative period (217, 218). Many other strokes may be asymptomatic, and indeed, routine MRI of patients after cardiac surgery identifies strokes in 18% of patients (219, 220). The occurrence of stroke is associated with markedly worse long-term outcomes (218). Management is supportive, with maintenance of adequate hemodynamics, aspirin treatment, and rehabilitation playing prominent roles.

Encephalopathy is another important neurologic complication after cardiac surgery, and, with an incidence up to 32%, occurs much more frequently than stroke (221). Encephalopathy has also been associated with worse in-hospital and long-term outcomes. The etiology is unknown, and proposed contributors have included atherosclerotic embolization during aortic manipulation, microembolization of air, and thrombi during CPB, hypoperfusion during CPB, and pre-extant cerebrovascular disease; the degree to which each of these contributes is unclear. The utility of CT imaging in the setting of abnormal neurologic findings is limited; positive findings (e.g., infarction, hemorrhage) are seen rarely with nonfocal deficits, and only 30% of the time with a focal neurologic deficit (222). Management is supportive.

Respiratory Failure and ARDS

Transient pulmonary complications are common after cardiac surgery, but relatively few patients (~5–8%) require mechanical

ventilation for more than 72 hours (223, 224). Causes of persistent respiratory failure include pneumonia, pulmonary edema, phrenic nerve injury, and ARDS (90). Pneumonia is the most common complication following mitral valve surgery (occurring in 5.5% of patients) and increases average hospital costs and length of stay by nearly $30,000 and 10 days, respectively (225, 226). The risk of ARDS depends on the surgical procedure performed; up to 17% of aortic surgery patients will develop ARDS (89). Mortality in these patients may be as high as 80% (227, 228). Little about the management of respiratory failure is specific to cardiac surgery. Respiratory status can be optimized, and complications are limited, by close attention to fluid status, lung-protective ventilation, minimization of sedation, daily spontaneous breathing trials, and liberation from the ventilator as early as possible (90). When tracheostomy is required, there appears to be no benefit, and possible harm, to delaying tracheostomy past postoperative day 10, despite anecdotes of increased risk of sternal infection with early tracheostomy (224).

AKI

AKI is a significant problem after cardiac surgery. Half of all patients will experience a significant reduction in renal function (25% increase in serum creatinine); up to 5% will require renal replacement therapy (RRT) (229, 230). The cause of AKI after cardiac surgery is not completely understood, but probably includes contributions from hypoperfusion, hemolysis, and inflammatory cytokines. AKI, especially that requiring RRT, significantly increases the risk of mortality (231); even after adjusting for comorbid conditions, AKI requiring RRT increases the risk of perioperative death by 27-fold compared with patients without AKI (232). Even if RRT is not required, AKI of any magnitude is associated with a significant increase in 90-day mortality (233) Beyond mortality, AKI increases length of ICU and hospital stay (233).

The consequences of AKI persist far beyond the postoperative period: the risk of 5-year cardiovascular mortality is significantly increased in patients who sustained any stage of AKI after cardiac surgery compared with those without AKI (234, 235). Preoperative AKI risk factors include pre-existing renal insufficiency, age, diabetes, tobacco use, and antecedent coronary angiography (236, 237). Intraoperative risk factors include CPB itself, long aortic cross-clamp times, and hypotension/poor renal perfusion. Kidney injury can also occur in the postoperative period if persistent hemodynamic instability impairs renal perfusion; inotropic exposure is also linked to AKI (238). Medications such as ACE-inhibitors and nonsteroidal anti-inflammatories should be avoided in high-risk patients. Unfortunately, no preventative strategy has been shown to be effective at decreasing the risk of AKI after cardiac surgery.

Nosocomial Infection

Nosocomial infections occur in 10–20% of cardiac surgical patients (239), including surgical site infections, vascular catheter infections, and urinary catheter infections. Many of these are preventable.

Surgical Site Infections. Deep sternal wound infections and mediastinitis occur in 1–2% of patients, with an associated mortality of up to 50% (240–243). Approximately 3% of patients develop superficial surgical site infections. Risk factors include diabetes, obesity, re-exploration for bleeding, use of the internal mammary arteries, blood transfusion, and prolonged mechanical ventilation and ICU stay (240, 243, 244). Perioperative antibiotic prophylaxis can markedly decrease the risk of surgical site infection. Current guidelines suggest a first- or second-generation cephalosporin in patients without methicillin-resistant *Staphylococcus aureus* (MRSA) colonization, and vancomycin in patients colonized by MRSA or allergic to penicillin (239, 245). Antibiotics should be continued for up to 48 hours postoperatively (246, 247). In MRSA-colonized patients, nasal decontamination with mupirocin ointment and chlorohexidene sponge baths can decrease MRSA wound infections (248, 249). This paradigm has been extended to methicillin-sensitive *S. aureus* (239, 250, 251). Signs of sternal infection and mediastinitis include wound erythema, fluctuence, sternal instability, disproportionate chest pain, fever, and leukocytosis. Effective therapy depends on rapid diagnosis, aggressive surgical debridement, and prolonged antibiotics.

Vascular and Urinary Catheter Infections. Central venous catheters are well recognized as a potential infectious source (252, 253). Catheter-related infection should be suspected in all patients with evidence of infection and no obvious alternative source. Arterial lines, particularly femoral, should not be overlooked (254–256). All catheters should be removed as soon as no longer needed. Similarly, the risk of urinary catheter-associated infection is decreased by 50% if the catheter is removed by postoperative day 2 (257); this should be the goal in all patients.

Venous Thromboembolism

Up to 20% of cardiac surgical patients will develop deep venous thrombosis or pulmonary embolism although few of these are symptomatic (258–260). However, a pulmonary embolism can be a devastating event (261). OP-CABG patients may be at higher risk than on-pump, presumably because of the fibrinolytic effects of CPB (262). Effective prophylaxis is essential, but there are few data for venous thromboembolism prophylaxis specific to cardiac surgery. Guidelines from the American College of Chest Physicians suggest using a combination of mechanical and pharmacologic prophylaxis, depending on a specific patient characteristics (263). For patients with low thrombotic risk and an uncomplicated postoperative course, intermittent pneumatic compression devices are probably sufficient and should be applied immediately to the legs upon arrival to the ICU (including legs used for saphenous vein harvest). Elastic compression stockings may be used in addition to compression devices (258). In patients with a higher risk of VTE or a complicated course (due to nonhemorrhagic events), pharmacologic prophylaxis with subcutaneous unfractionated heparin or low molecular weight heparin should be added to intermittent compression prophylaxis (263) although some authors recommend pharmacologic prophylaxis in all patients (260). If thromboembolism occurs, management hinges on

the temporal distance from surgery and the patient's perceived hemorrhagic risk. In patients at low risk for bleeding, therapeutic anticoagulation is generally acceptable. The management of a patient in circulatory shock due to a pulmonary embolism is more complex because thrombolysis is generally contradicted within 10 days of major surgery (264). Surgical embolectomy remains an option.

Skin Breakdown and Pressure Ulcers

Cardiac surgical patients are at high risk for skin breakdown and pressure ulcer development (265). Frequent skin assessment, preventative care, and early intervention on wounds are essential (266, 267). Early mobilization is an important tool to prevent skin breakdown. Leg wounds after saphenous vein harvest can be problematic, especially after open vein harvest (compared with endoscopic) (268, 269). Close attention should be paid to harvest sites, with attention to any evidence of dehiscence, seroma, hematoma, or infection.

PHYSICAL THERAPY AND REHABILITATION

In addition to having the physical consequences of critical illness, cardiac surgical patients may be significantly deconditioned due to functional limitations of their index disease (e.g., exercise limitation from angina or valve dysfunction). Long-term participation in cardiac rehabilitation decreases 10-year all-cause mortality after CABG (270, 271). The benefits of early physical therapy and rehabilitation in critically ill patients have been documented (272, 273). Although there are few data specific to cardiac surgical patients, it is reasonable to start physical therapy and rehabilitation as possible postoperatively. Uncomplicated patients typically ambulate in the hall on postoperative day 1 (274). More complicated patients, including mechanically ventilated patients, patients on vasoactive infusions, and even patients with mechanical circulatory support devices, may be able to participate in rehabilitation therapy (275–277). Recommended safety criteria for the mobilization of critically ill patients have recently been published; these address considerations relevant to cardiac surgery, such as the presence of IABPs and mechanical support devices (278). Additional precautions are often taken to protect the fresh sternotomy; these include weight limits on lifting with the upper limbs, keeping the upper arms close to the body, and restrictions on using the arms to pull or push while getting out of bed or ambulating with assist devices (279–282). However, these precautions are variably applied, with few data supporting their use, and have been criticized as overly restrictive (280, 282, 283).

QUALITY AND PERFORMANCE IMPROVEMENT IN CARDIAC SURGICAL CRITICAL CARE

In as high-stakes an endeavor as cardiac surgical critical care, efforts to maintain and improve the quality of care are essential. In the opinion of these authors and others, the keys to quality are as follows: agreed upon outcomes to serve as surrogates for quality, standardization of care when possible, and continual review of outcomes.

Metrics

The Society of Thoracic Surgeons (STS) (284, 285), the Joint Commission (286), the University Healthcare Consortium (287), and individual states have created both public and private reports, which grade cardiac surgery programs (288, 289). The STS database includes over 3 million patients collected since 1990; more than 90% of U.S. cardiac surgery programs participate (285). The STS uses risk normalized observed to expected mortality ratios, postoperative complication rates (reoperations for bleeding, renal failure, prolonged ventilation, and mediastinitis), and an evaluation of a program's "systems approach to care" (rates of preoperative β-blocker administration and rates of discharge prescriptions for lipid-lowering agents, antiplatelet drugs, and β-blockers) to assign a "1-Star," "2-Star," or "3-Star" rating to a program. In contrast, the Joint Commission assesses Surgical Care Improvement Project metrics, substituting easily measured surrogates for quality (e.g., use of prophylactic antibiotics) (available at http://www.jointcommission.org). In Europe, the EuroSCORE logistic model, which predicts mortality after cardiac surgery, is used not only as a predictor for individual patient outcomes but also to identify programmatic mortality benchmarks (290–292). New metrics remain in development. Recently, the STS database has been used to develop a 30-day all-cause hospital readmission after CABG metric for future public reporting (293). Other metrics that have been proposed in the literature include blood product use (294) and failure to rescue from complications (105). These metrics are summarized in **Table 2**.

Standardized Care

Standardization of processes has been shown to improve quality and reduce costs in a number of fields. Standardization of practice seems particularly well suited to operations such as CABG, where patients are fairly homogeneous, the operative procedure well scripted, and the postoperative course relatively predictable (295). But all patients undergoing cardiac surgery are likely to benefit from standardized management protocols (296, 297). Standardizing systems or using clinical pathway guidelines improve quality in a variety of arenas (298–300). For example, cardiac surgical ICUs with order sets for sedation, analgesia, and delirium that are more consistent with guidelines have shorter ventilator times than hospitals with lower quality order sets (301). Whether using ventilator-acquired pneumonia "bundles," instituting hemoglobin concentration as a trigger for transfusion, or standardizing extubation protocols to improve early extubation, eliminating the variability innate to individual care givers can markedly improve performance (302–304).

Continued Review of Outcomes

Continuous review of performance metrics is essential to both quality maintenance and improvement. Many institutions use a "dashboard," which present data on selected outcome in

TABLE 2. Performance Metrics and Quality Improvement in Cardiac Surgery Critical Care

Metric	Comparison	Publicly Reported	Comments	References
STS 3-Star Composite Rating	Overall program quality, CABG and aortic valve replacement only	Voluntary	Strong association with quality	288
Procedural volume		Yes	Weak association with outcomes for CABG	289
Perioperative mortality	Expected risk-adjusted mortality Similar programs	Yes	STS composite EuroSCORE	288, 290–292
Perioperative renal failure	Similar programs	Yes	STS composite	288, 289
Perioperative stroke	Similar programs	Yes	STS composite	288, 289
Sternal infection or mediastinitis	Similar programs	Yes	STS composite	288, 289
Reoperation for bleeding	Similar programs	Yes	STS composite	288, 289
Prolonged mechanical ventilation (> 24 hr)	Similar programs	Yes	STS composite	288, 289
Preoperative β-blocker	Similar programs National average	Yes	STS composite Medicare	288, 289; http://www.medicare.gov
Administration of prophylactic antibiotics	National average	Yes	Medicare	http://www.medicare.gov
Discharge for lipid-lowering agent	Similar programs	Yes	STS composite	288, 289
Discharge for antiplatelet agent	Similar programs	Yes	STS composite	288, 289
Discharge for β-blocker	Similar programs	Yes	STS composite	288, 289
Hospital readmission	N/A	Not yet	Proposed	293
Blood product use	N/A	Not yet	Proposed	294
"Failure to rescue" rate	N/A	Not yet	Proposed	105

STS = Society of Thoracic Surgeons, CABG = coronary artery bypass graft, N/A = not applicable.

a standard format. This dashboard is reviewed on a regular basis to monitor performance and allow rapid identification of either positive or worrisome trends. The data reviewed can be adapted according to institutional needs. In many instances, this process has led to improvement in care (297, 299, 300).

SUMMARY

Rapid advances in technology and surgical technique have broadened the armamentarium of cardiac surgeons. Consequently, the cardiac intensivist must be aware of the specific aspects and management concerns of an ever-increasing catalogue of procedures. Although cardiac surgery is nominally performed on the heart and great vessels, its sequelae can affect virtually every organ system. Thus, the cardiac intensivist must also possess broad general medical knowledge and a comprehensive understanding of multisystem pathophysiology. Fortunately, the majority of complications after cardiac surgery fall into a limited number of categories. Familiarity with the presentation and management of these stereotypical problems and pitfalls allows anticipation and rapid reaction when an issue develops. A structured approach to complication management provides a framework for handling even the most complicated postoperative situations. As the use of ECMO increases for severe circulatory failure and severe respiratory failure, cardiac intensivists, by virtue of their extensive experience with mechanical support devices, are well equipped to participate in the expansion of extracorporeal life support technology. High-quality care and good outcomes are enhanced by protocols and standardization, but absolutely depend on constant self-examination and programmatic improvement.

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