Concise Definitive Review

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

R. Scott Stephens, MD[1,2]; Glenn J. R. Whitman, MD[2,3]

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

[1]Division of Pulmonary and Critical Care Medicine, Department of Medicine, Johns Hopkins University, Baltimore, MD.

[2]Cardiovascular Surgical Intensive Care Unit, Johns Hopkins Hospital, Baltimore, MD.

[3]Division of Cardiac Surgery, Department of Surgery, Johns Hopkins University, Baltimore, MD.

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For information regarding this article, E-mail: rsteph13@jhmi.edu

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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 Vo_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 Vo_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 Vo_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 Vo_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 Vo_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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