Concise Definitive Review

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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; 43:1995–2014)

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

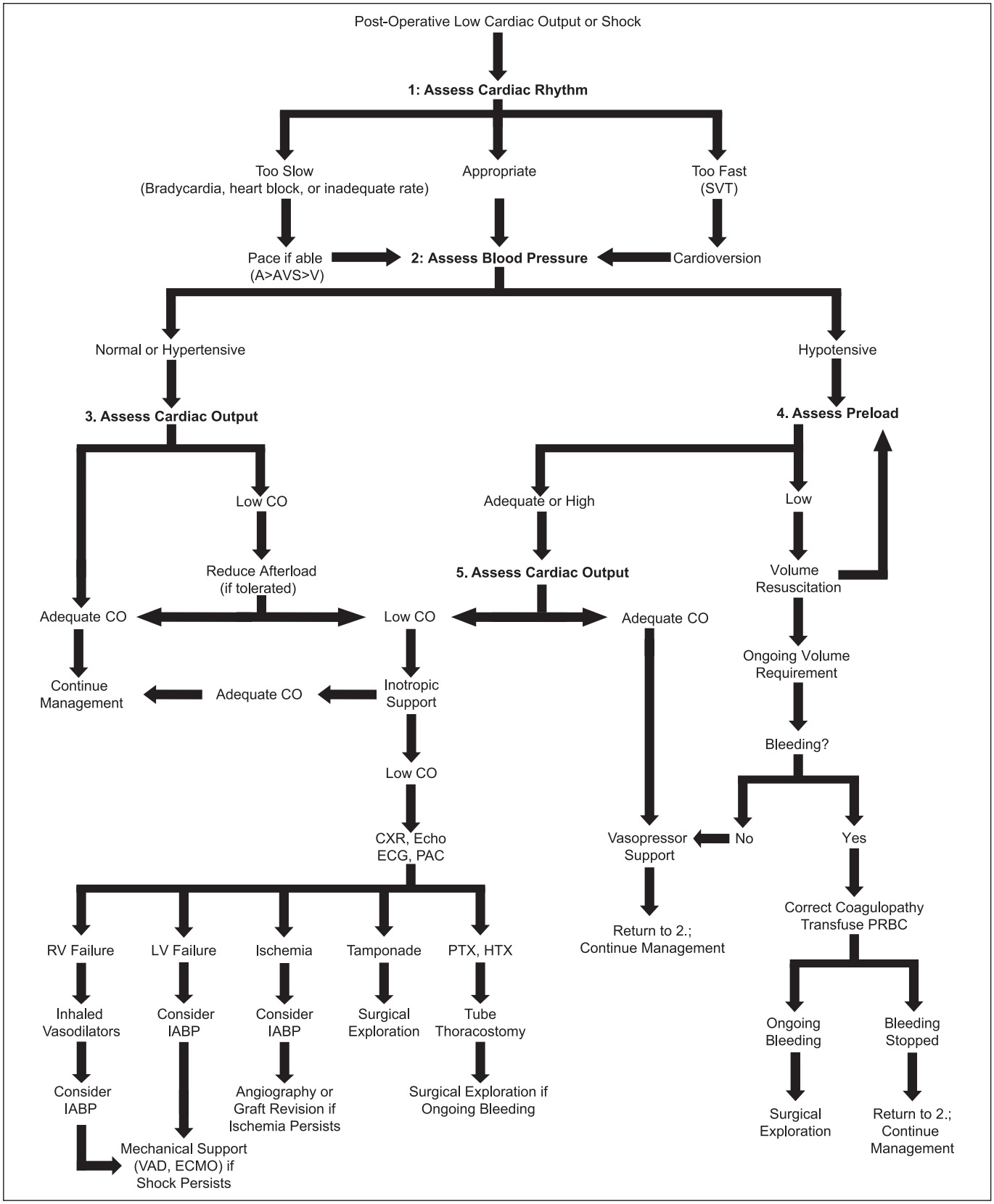

Figure 1. Algorithm for management of low cardiac output and refractory shock after cardiac surgery. This is one approach to the management of low cardiac output and refractory shock after cardiac surgery. It is meant to be illustrative of key thought processes and important factors to consider. See 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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