Hemodynamic management of patients in the first 24 hours after cardiac surgery

Arthur C. St. André, MD, FCCM; Anthony DelRossi, MD

**Objective:** To describe the physiologic alterations, evaluation, and hemodynamic management of patients in the first 24 hrs after cardiac surgery.

**Design:** A brief review of preoperative and intraoperative events, postoperative physiology, and a discussion of the evaluation and hemodynamic management of cardiac surgery patients postoperatively based on a review of the literature, known physiology, and clinical experience.

**Results:** After cardiac surgery, patients undergo alterations in cardiac performance related to co-morbid conditions, preoperative myocardial insults and interventions, the surgical procedure, and intraoperative management. Predictable responses evolve rapidly in the first 24 hrs after surgery. Monitoring, diagnostic regimens, and therapeutic regimens exist to address the patterns of response and occasional complications.

**Conclusion:** By understanding preoperative and intraoperative events and their evolution in the intensive care unit, clinicians can effectively manage patients who experience cardiac surgery. (Crit Care Med 2005; 33:2082–2093)

**Key Words:** cardiac surgery; hemodynamic management; postoperative management.

Each year, >500,000 cardiac procedures are performed in the United States (1), and until recently, this number had grown annually. The indications for cardiac procedures include myocardial ischemia, valvular dysfunction, heart failure, dysrhythmias, and disturbances of pulse generation and propagation (2–5). Patient populations, operative and anesthetic management, and diagnostic and therapeutic armamentaria undergo continual rapid evolution and are incorporated into existing surgical and programmatic traditions. Evaluating and managing postcardiac surgery patients present clinicians with an array of challenges. This review will discuss the hemodynamic management of patients during the dynamic first 24 hrs in the intensive care unit (ICU) after coronary and valvular cardiac surgery.

**FACTORS INFLUENCING POSTOPERATIVE MANAGEMENT**

Effective postoperative management depends highly on understanding each patient’s preoperative status. Patients are predominantly men, in a 70:30 ratio, although this ratio is diminishing. Mean age approximates 64 yrs old for men and 67 yrs old for women (6). Co-morbid conditions play a significant role, with cerebrovascular and renal disease, obesity, hypertension, and diabetes manifesting with variable intensity during postoperative care (7). The panoply of medications in use preoperatively has grown dramatically during the past two decades. Current preoperative regimens include angiotensin-converting enzyme inhibitors, beta-blockers, calcium channel blockers, nitrates, aspirin, and clopidogrel. These medications may exert significant effect on intraoperative and postoperative physiology.

Survival is related to preoperative risk factors and to intraoperative and postoperative management. Risks include decreased ejection fraction (EF), especially if <30%, left main coronary artery disease, diabetes mellitus with its associated diffuse coronary obstructions, renal insufficiency (creatinine of >2.0), symptomatic parenchymal lung disease, and advanced age. Risk stratification methodologies exist and allow reasonable prediction of outcomes (6). Patients, agencies, payors, and hospitals use such determinations to compare results among programs and individual surgeons (8–12).

**EFFECT OF INTRAOPERATIVE EVENTS AND MANAGEMENT**

Anesthetic management, with particular attention to myocardial oxygen consumption, is critical in all cardiac surgeries but especially during coronary artery bypass procedures. With coronary obstruction, myocardial oxygen demand must be minimized. Attention to blood pressure and heart rate are essential because increases or decreases in either can lead to myocardial ischemia. The general theme in the “fast-track” era (rapid progress through the ICU and hospital) is to minimize use of long-acting agents during the maintenance phase of anesthesia (13, 14). Inhalational or rapidly metabolized intravenous agents, such as propofol or dexmedetomidine, are utilized in the maintenance phase of the procedure, along with narcotic agents and muscle paralysis.

During the period of no coronary artery blood flow during cardiopulmonary bypass, myocardial oxygen demand must be eliminated to prevent myocardial ischemia and infarction. Traditionally, surgery is performed on the nonbeating heart by rapidly inducing diastolic arrest by injection of a cold KCl cardioplegic solution to eliminate myocardial oxygen demand during cardiopulmonary bypass. The solution is infused directly into the aortic root after applying a clamp to the distal ascending aorta.
Additional cardioplegic solution may be given into the coronary sinus (15–18).

Arterial grafts to coronary arteries are commonly utilized. Arterial grafts, particularly the left internal mammary, reduce short- and long-term mortality and the need for subsequent revascularization. Use of the right internal mammary artery is limited because of technical difficulties (i.e., an inability of the right to reach the posterior descending artery when grafting in situ) and fear that there will be an increase in sternal infection, especially in insulin-dependent diabetic or obese patients. Saphenous veins are easily accessible and are excellent conduits when inserted in a reversed direction relative to the venous valves. The long-term patency of arterial grafts is superior to saphenous vein grafts. Approximately 80% of arterial grafts are patent at 8 yrs. This has precipitated enthusiasm for use of other arteries, including the radial and gastroepiploic. The use of in situ arterial grafts (e.g., where the native take off of the internal mammary artery from the subclavian artery remains intact) eliminates proximal anastomoses to the ascending aorta. Consequently, cerebrovascular accidents that primarily result from ascending aorta emboli may be reduced. Most commonly, surgeons use the left internal mammary as a single graft to the left anterior descending and saphenous veins for the remaining bypasses.

In recent years, beating-heart surgery that eliminates cardioplegia and cardiopulmonary bypass has become prevalent. Beating-heart surgery is accomplished with a device that stabilizes a small area around the coronary artery target, allowing the surgeon to perform a distal anastomosis with minimal myocardial movement while the remaining heart continues to contract. Some advocate off-pump and normothermic coronary bypass, pointing out that there is a reduction in the use of blood products postoperatively and shorter ICU lengths of stay with normothermia. Proponents of off-bypass coronary surgery claim that results are better and that the prevalence of cerebrovascular accidents and other complications are reduced (19). However, mild hypothermia is helpful in reducing myocardial oxygen demand during the operation, so the optimal approach as it relates to temperature is unclear. Thus far, the more technically difficult off-bypass procedure has results similar to conventional surgery utilizing cardiopulmonary bypass.

Heparin is administered during off-bypass surgery and is also essential for procedures using cardiopulmonary bypass to offset the exposure of blood to the surfaces of the bypass machine, which triggers the coagulation cascade. Protamine is used to neutralize heparin after the procedure, but it is not benign and can cause hypotension during administration, especially in diabetic patients. Hemodynamic instability from protamine results from a nonimmunologic reaction with thrombosome release that induces pulmonary vasoconstriction, bradycardia, and hypotension, with right heart failure or a more severe immunologically mediated anaphylaxis (20, 21). In addition, intraoperative antifibrinolytic agents are used to reduce bleeding and curtail the need for blood products postoperatively.

Valvular replacement and repair are prevalent procedures and are often accomplished along with coronary artery surgery. Mitral valvular repair rather than replacement is preferable when practical (22). The regurgitant mitral valve is well adapted for repair. Repair of mitral valve insufficiency exceeding 2+ is indicated at the time of coronary artery bypass grafting. Surgery for atrial dysrhythmias, such as the Maze procedure (23), can be done to interrupt aberrant conduction pathways during cardiac surgery performed for other indications. Surgeons ablate the right and left atrial pathways with either cryotherapy or bipolar radio frequency energy.

Procedures to ameliorate heart failure are undergoing active investigation. Myocardial tissue is excised in volume reduction surgery, and alternatively, devices are applied to prevent the myocardium from further dilating. In recent years, major advances have occurred in the development of temporary and permanent ventricular assist devices. At present, devices to support the left and right ventricles can be temporarily used to successfully remove patients from cardiopulmonary bypass (24, 25). The management of these devices will not be reviewed.

**POSTOPERATIVE PHYSIOLOGY**

Successful recovery after cardiac surgery depends heavily on preoperative and postoperative myocardial performance rather than coronary anatomy. Postoperatively, contractility is uniformly diminished compared with preoperatively. The magnitude and duration relate to: the severity of chronic dysfunction, recent ischemic events, efficacy and complications of preoperative invasive cardiologic procedures, and intraoperative course. A previous echocardiogram or ventriculogram may provide useful information, but echocardiographic performance just before surgery may be of little assistance when a significant ischemic insult intervened. Thrombolytic, anticoagulant, or antithrombotic therapies and recent neurologic events and dye-related nephropathy are common considerations in postoperative management. In addition to knowing the implications of these details, clinicians must be aware of the natural history of the myocardium’s response to pre-ICU insults.

All intraoperative events play significant roles in the recovery process. Those of most import are: anesthetic management, hypothermia (26, 27), cardiopulmonary bypass, and the duration of cardiopulmonary bypass (28). If in place, an intraaortic balloon pump is more often placed preoperatively for ischemia rather than for dysfunction, whereas dysfunction serves as the reason for intraoperative placement.

Preoperative and intraoperative events and interventions vary in magnitude and duration but typically result in a myocardium of reduced contractility and compliance that affect postoperative management and eventual outcome (29–32). A preoperative EF of <35% and immediate preoperative ischemia or myocardial infarction have substantial effect on postoperative management. Patients with outflow obstruction from hypertrophic disease related to chronic hypertension or aortic stenosis present challenging management issues. Early surgery for valvular insufficiency generally reduces gross postoperative physiologic disturbances. Late intervention for chronic valvular insufficiency is likely to be associated with very poor postoperative ventricular function. Significant right ventricular dysfunction, often of cryptic etiology, remains a particular challenge. The large variety of pre-ICU patient populations results in repetitive postoperative physiologic “themes.”

At ICU admission, one determines the expected physiology. The typical physiologic patterns result from either a 1) pressure-overloaded or 2) a volume-overloaded myocardium. Each has implications for assessment and management of preload, contractility, heart rate, and rhythm. Important in each physiologic pattern is optimizing left ventricular pre-
load, which in the ICU is typically measured by filling pressures. However, one must be aware of when filling pressures do not adequately reflect preload, defined as the ventricular volume at end diastole. In the pressure-overloaded ventricle, especially if there is significant hypertrophy, volume requirements may be significant. With each aliquot of volume resuscitation, the blood pressure, cardiac index (CI), and ventricular filling pressures must be closely scrutinized to avoid both under and over resuscitation. After cardiac surgery, the myocardium generally functions as a pressure-overloaded ventricle regarding volume resuscitation because of reduced postoperative myocardial compliance. As a result of decreased compliance, preload requirements may be underestimated by solely relying on measured filling pressures. Because a ventricle with reduced compliance is more “stiff,” the pulmonary artery occlusion pressure may be relatively higher than the actual preload. Thus, further volume resuscitation may be salutary, even when the pulmonary artery occlusion pressure is normal to high.

Similarly, chronically volume-overloaded ventricles, typically from mitral insufficiency, depend on sufficient volume resuscitation. In the chronically volume-overloaded ventricle, the blood pressure and CI response to volume challenge serve as better guides of adequate preload because the pulmonary artery occlusion pressure and pulmonary artery pressure are insensitive except at the extremes of hypovolemia and hypervolemia. In this situation, the blood pressure and CI may improve remarkably before the filling pressures change. As the time postoperatively increases and the patient warms, myocardial compliance improves, altering the relationship of preload to filling pressures in pressure and volume ventricles.

Preoperative disease and medications, the operative procedure, and medications determine heart rate at arrival to the ICU. Hypertrophic or pressure-overloaded ventricles are intolerant of wide fluctuations in heart rate. When heart rate is excessive, the time for preloading the ventricle may shorten sufficiently to compromise end-diastolic volume and thus cardiac output. In contrast, as heart rate slows below the time needed to develop maximal end-diastolic volume, cardiac output may also decrease in relation to the slower rate. Assuming optimization of preload, a heart rate of 90–100 beats/min is typically optimal for the hypertrophied myocardium. In addition, when compliance is significantly diminished, synchronized atrioventricular contraction plays a more significant role in ensuring maximal preload. Thus, effective preloading of the significantly hypertrophied left ventricle commonly requires the atrioventricular synchrony of sinus rhythm or dual-chamber pacing.

The volume-overloaded ventricle is less susceptible to underfilling and more tolerant of tachycardia and loss of atrioventricular synchrony. At faster heart rates, end-diastolic volume is less as compared with that at slower rates. With reduced end-diastolic ventricular distention at faster heart rates, systolic emptying may improve. Rate-related contractility might also be related to the Bowditch reflex, by which contractility improves at higher rates. In fact, tachycardia (~100 beats/min) may be enlisted as a therapeutic strategy. In these patients, a sinus rhythm <75 tends to be more deleterious than an abnormal rhythm at rates around 90. As the heart rate slows, prolonging the time for diastolic filling, compromise of ventricular EF may occur because the ventricle is more dilated. Because diastolic compliance of the volume-overloaded ventricle is not as severely compromised as that of the pressure-overloaded ventricle, there is less dependence on the atrial “kick” of atrioventricular synchrony to ensure maximal preload. So, in the volume-overloaded ventricle, tachycardia and loss of atrioventricular synchrony may be better tolerated than sinus bradycardia.

Determining the degree of reduced contractility at admission to the ICU is problematic as chronic, immediately preoperative, and operative variables must be taken into account. Key contributors to depressed postoperative contractility include a preoperative EF of <35%, the duration and severity of hypothermia (especially if a patient arrives at <35°C), and cardiopulmonary bypass time (especially if the duration exceeded 120 mins). Of considerable value is a description of intraoperative myocardial performance and the inotropic agents required. With valvular procedures, many patients undergo a post-bypass intraoperative transesophageal echocardiogram performed by a cardiologist or anesthesiologist. These prove quite helpful in assessing valvular placement and function, ventricular performance, and preload. The use of intraoperative transesophageal echocardiograms is expanding as surgeons further recognize their utility in this setting and as anesthesiologists develop the procedural skill.

Understanding the “getting better” process as one of resolution of diminished contractility and decreased compliance is key to effective management. If the preoperative EF was >35% and the operative course was smooth, myocardial performance diminishes the first 4–6 hrs in the ICU and then rapidly returns to baseline or suprabaseline within 24 hrs (Fig. 1). The maximum diminution in function approximates 10–15% of baseline EF.

Patients with a preoperative EF of <35%, perioperative ischemia, or a diffi-
POSTOPERATIVE MANAGEMENT

General

Similarities in monitoring, evaluation, and management of patients exist, despite the wide spectrum of patients, operative procedures, and pre-ICU management. Early continuous monitoring includes the ICU array of electrocardiogram (ECG), invasive arterial pressure, pulse oximetry, and, occasionally, endtidal CO₂ concentration. Either a central venous pressure monitor or pulmonary artery catheter (33) is typically used for preload assessment. Pulmonary artery catheters may be inserted preoperatively, depending on myocardial performance, the intended operative procedure, and the expected physiologic insult or associated mechanical manipulation. Assessment of patient risk may also lead to the use of a pulmonary artery catheter with capability for monitoring cardiac output, pulmonary artery pressures, pulmonary artery occlusion pressure, a measure of left ventricular preload, and mixed venous oxygen saturation. Typical indications for a pulmonary artery catheter include: an EF of <40%, combined aortic valve and coronary surgery, mitral or tricuspid valve surgery, chronic heart failure–myocardopathy surgery, and Jehovah’s Witnesses.

Also, during beating-heart coronary surgery, a pulmonary artery catheter is typically inserted to better assess performance during heart manipulation. As operative teams become more familiar with this procedure and its effects, pulmonary artery catheter use will likely diminish. Pulmonary artery catheter utilization is also determined by other factors related to programmatic and surgeon preferences.

Over time, the use of laboratory measurements has decreased as providers realize that traditional test panels offer little utility. At a minimum, the postoperative panel includes a hemoglobin, potassium, activated partial thromboplastin time, and platelet count that may be used subsequently to assess for the development of heparin-induced thrombocytopenia. An initial arterial blood gas, or one done late in the operating room, is useful. A baseline postoperative 12-lead ECG is performed. Subsequently, the hemoglobin, potassium, and possibly the arterial blood gases are reassessed during the first 6 hrs. Most patients are assessed with a postoperative portable chest radiograph, although important abnormalities are quite uncommon.

As the cardiac system, specifically the myocardium, goes through a period of temporary deterioration and resolution, predictable physiologic patterns evolve as described. Occasionally, physiology is disturbed in degree or kind, raising additional diagnostic concerns. At arrival, patients typically exhibit sinus rhythm in the range of 90–100 and a mean arterial pressure (MAP) of 70–90 mm Hg. The central venous pressure, pulmonary artery diastolic pressure, and pulmonary artery occlusion pressure are predicated on many factors, although they are commonly in the range of 12–18 mm Hg. CI is variable but is usually ≥2.2 and hopefully >2.0, unless severe depression exists. The crux of ICU management relates to manipulation of these variables, understanding that filling pressure, heart rate, and rhythm goals to optimize myocardial performance are individualized. Support at admission may include a wide variety of vasoactive agents to increase (more frequently) or decrease (less frequently) blood pressure or inotropic medications for excessively depressed myocardial performance. An intraaortic balloon pump may have been placed preoperatively to ameliorate myocardial ischemia and, rarely, preoperatively or intraoperatively for myocardial depression. When not used for myocardial depression, it is removed in the first few hours postoperatively.

The first 6–8 hrs of ICU care is a dynamic period. Management is typically responsive rather than proactively preventive. Tachycardia is generally unhelpful as a predictor of low preload because of multiple preoperative and operative factors affecting its correlation to hypovolemia. A moderately low pulmonary artery occlusion pressure or central venous pressure does not prompt therapy in the face of an adequate MAP and CI. Fluctuations in the MAP and CI, if there is a pulmonary artery catheter, are the primary indications for intervention.
The MAP, manifest as hypotension, is the most dynamic physiologic variable during the first hour of postoperative ICU care. This physiologic response typically occurs secondary to a reduction in both preload and ventricular compliance. Vasodilation from a loss of vasomotor tone is also a frequent early contributor to a decreased MAP. Less common are deteriorating contractility, reduced heart rate, and loss of atrioventricular synchrony from dysrhythmia or conduction disturbances.

The goal of therapy is a MAP of 70–80 mm Hg. Occasionally, a more substantial goal has import when the central nervous system and renal systems are accustomed to a higher MAP as in chronic hypertension. Infrequently, the goal must be particularly low due to considerations such as a “friable aorta,” an excessively “wet mediastinum,” or myocardial “thinning” (e.g., as the posterior ventricular wall sometimes is after replacement of the mitral valve). In these circumstances the surgeon has determined the risk of bleeding to be greater than usual. Scientific investigation lacks sufficient evidence of the best resuscitation fluid to use in this patient population. So, clinicians choose based on their understanding of the colloid/crystalloid literature. Some insist on albumin or similar colloid, whereas others utilize normal saline or hypertonic solutions. Hetastarch of up to 2 liters has been utilized successfully without excessive bleeding, although one should be mindful of associated hemodilution and the possibility of coagulopathy. In response to hypotension, volume boluses of 250–500 ml are rapidly administered as the initial therapy. Volume resuscitation continues until the MAP responds, vasodilation is deemed the primary contributor or high filling pressures occur. In our experience, most volume resuscitation occurs during the initial 5 hrs of postoperative ICU care, resulting in the great majority of patients receiving over a liter during this period. In the subsequent 5 hrs, a minority requires volume, and the original group often receives additional aliquots for an overall “bolus” volume of 1–2 liters. Crystalloid resuscitation requires more total volume resuscitation than colloidal. Often, fluid resuscitation alone is fully successful in maintaining an effective MAP and perfusion.

Other diagnostic and therapeutic considerations arise during preload resuscitation if the MAP becomes significantly compromised or usual fluid resuscitation maximums are reached without normalization of the MAP. In contrast to more than a decade ago, when significant hypertension was the extant physiologic pattern, vasodilation with associated, often-severe, hypotension has become the prevalent hemodynamic pattern (34). The pathogenesis (35) and mechanisms of the vasodilatory phenomenon remain elusive, although alcohol dehydrogenase depletion and increased nitric oxide are likely contributory candidates. Consequently, as volume resuscitation proceeds, vasopressor therapy (18) is added, while remaining mindful of situations in which significant diminution of contractility or other causes of hypotension may be contributing. Those agents with vasopressor activity to consider include epinephrine, norepinephrine (36), phenylephrine, vasopressin, and rarely methylene blue (37). In some circumstances, vasopressin (38) is added to a traditional vasopressor. Vasopressin has physiologic rationale, although its effects on outcome are not available, so its use may be simply additive to other agents rather than superior. Assurance of adequate preload is imperative before and during use of inotropic or vasopressor support (39). The duration of excessive vasodilation requiring vasopressor support can be less than an hour, although it often remains for 6–8 hrs. Occasionally, the duration extends to few days in patients otherwise recovered.

In contrast to hypotension, some patients present during their intraoperative or postoperative period with significant hypertension (40, 41). An excessive MAP may augment bleeding and create excessive afterload, while myocardial contractility and compliance are compromised. Medications utilized for this purpose include rapidly acting and metabolized vasodilators such as nitroprusside and intravenous nitroglycerin. Beta blockade should be used with caution, as discussed subsequently.

**Contractility/CI**

Myocardial contractility may further diminish during the immediate postoperative period (29, 30, 32), mandating inotropic support or augmentation of support begun in the operating room. The contractility patterns that evolve are a) a good and improving CI throughout, b) a persistently poor CI from arrival, and c) an initially satisfactory CI with deterioration over 4–8 hrs followed by improvement or occasionally persistent dysfunction. The risk of hypoperfusion increases substantially as the CI decreases to <2.2 and especially to <2.0, unless hypothermia exists. A decreasing CI should be addressed before signs of overt hypoperfusion develop, even if the preprocedure CI was similar.

The diagnosis of hypoperfusion may be obvious but is often subtle and requires proactive and presumptive therapy based on a combination of expectations, trends, and subtle indicators of reduced flow (42–44). Diminished cardiac performance secondary to altered contractility presents as reduced CI, hypotension, or with an end-organ indicator of low urine output, mottling, or metabolic acidosis. Signs of end-organ dysfunction are not particularly sensitive indicators of early hypoperfusion. Excessive urine output, especially in the initial few hours, commonly occurs and is related to intraoperative management. This initial excess often remains when cardiac performance is marginal, although it is temporary. By contrast, low urine output may indicate diminished contractility, preoperative renal dysfunction, or tamponade. Mottling after cardiac surgery may be associated with hypoperfusion, shivering, hypothermia, or peripheral vascular disease. When due to hypoperfusion, it is typically a late manifestation, is associated with cool limbs, and dissipates slowly, lagging behind improvement in perfusion and temperature. Similarly, metabolic acidosis presents along with a mix of findings suggesting hypoperfusion, unless related to epinephrine (discussed subsequently). Lactate measurement has little value. Occasionally, the pulmonary artery oxygen saturation of hemoglobin (mixed venous oxygen saturation) is useful.

An array of agents, the intraaortic balloon pump, and cardiac assist devices exist to manage compromised contractility. One must be acutely aware of the inotropic, vasodilatory, or constractive and chronotropic effects of each medication. Understanding each agent’s cardiovascular actions and mechanisms serves as the basis for selection because each effect may be a benefit or detriment, depending on the physiologic situation and patient tendencies (Table 1). Medications utilized include epinephrine, milrinone, dobutamine, isoproterenol, and dopamine for inotropy. Dose response ranges are typical, although higher dosages and combinations are utilized in the most dire sit-
Table 1. Cardiovascular medications

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dose</th>
<th>Contractility</th>
<th>Heart Rate</th>
<th>Vasoconstriction</th>
<th>Vasodilation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isoproterenol</td>
<td>1–5 µg/min</td>
<td>4+</td>
<td>4+</td>
<td>0</td>
<td>4+</td>
</tr>
<tr>
<td>Milrinone</td>
<td>0.125–0.5 µg · kg⁻¹ · min⁻¹</td>
<td>4+</td>
<td>0</td>
<td>0</td>
<td>3+</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>2–20 µg · kg⁻¹ · min⁻¹</td>
<td>3–4+</td>
<td>1–2+</td>
<td>0</td>
<td>2+</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>1–20 µg/min</td>
<td>4+</td>
<td>4+</td>
<td>4+</td>
<td>3+</td>
</tr>
<tr>
<td>Dopamine</td>
<td>1–4 µg · kg⁻¹ · min⁻¹</td>
<td>1+</td>
<td>1+</td>
<td>0</td>
<td>1+</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>2–40 µg/min</td>
<td>2+</td>
<td>1+</td>
<td>4+</td>
<td>0</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>20–200 µg/min</td>
<td>0</td>
<td>0</td>
<td>3+</td>
<td>0</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>0.01–0.04 units/min</td>
<td>0</td>
<td>0</td>
<td>4+</td>
<td>0</td>
</tr>
</tbody>
</table>

Modified from Parrillo (115).

Table 2 depicts a scheme of medication choices for various hemodynamic situations. It should be noted that dopamine in the initial hours, more so than subsequently, may result in excessive tachycardia and a marked increase in urine output. To decrease the prevalence of hypotension, milrinone does not have to be loaded. The chronotropic response to dobutamine may be of assistance or detrimental, depending on baseline heart rate. Isoproterenol, a much-vilified drug when myocardial ischemia is a risk, may result in an excellent response, especially in the face of significant right ventricular dysfunction or in the face of relative bradycardia. It has been utilized successfully, even when the basal heart rate is within the normal range, and does not result in significant hypotension.

With just central venous pressure monitoring demonstrating adequate filling pressures after volume resuscitation, one will be uncertain if persistent hypotension is related to vasodilation, poor contractility, or both. The combined alpha and beta effects of epinephrine provide a good balance to address both pathogeneses. Thus, epinephrine may be the first agent utilized to treat hypotension of unclear cause. If a pulmonary artery catheter is not in place and a satisfactory response to lower doses of epinephrine (<5 µg/min) occurs, a pulmonary artery catheter may be avoided. Unfortunately, troublesome side effects may result from epinephrine. These include the non–dose-dependent development of significant metabolic acidosis (45) and severe hyperglycemia that arise when epinephrine is begun within the first 6–8 hrs after the procedure. They typically dissipate by hour 12. Any dose of epinephrine may result in their emergence. The metabolic acidosis may be severe, with an HCO₃ of 15 meq/liter at its worst, although the range of 17–21 meq/liter occurs most commonly. Increased lactate does occur (46). Hypoperfusion does not seem to be the physiology creating this acidosis. Often, the CI is stellar and the mixed venous oxygen saturation is higher than expected, simulating the hypotensive hyperdynamic profile of sepsis. Splanchnic perfusion is preserved. Patients are deemed well perfused and therapy remains unaltered until spontaneous resolution. However, when acidosis occurs during epinephrine therapy with a marginal CI, the diagnostic situation becomes more complex, and the acidosis must be treated as if it was related to tissue hypoperfusion. Here, management depends on the magnitude of dysfunction and may result in the addition of other inotropes while decreasing or reobtaining the epinephrine. The role of bicarbonate therapy for epinephrine-induced metabolic acidosis is unclear.

Epinephrine may produce significant hyperglycemia, even in the nondiabetic. Continuous intravenous insulin brings the blood sugar under control with much difficulty during epinephrine therapy. Aggressive management is mandatory, considering better outcomes with glycemic control (47). High-dose insulin infusion requirements usually disappear quickly because the effects of epinephrine on glucose regulation dissipate rapidly. The effects on long-term outcome remain unreported. Acidosis and hyperglycemia resolve within a few hours and generally last no more than 6 hrs. The phenomenon occurs frequently, but once it disappears, it does not return, even if epinephrine is reintroduced.

Most patients respond well to vasopressor intervention for vasodilation and inotropic agents for poor contractility. The frequency of vasopressor use varies but may approach 40%, whereas agents for poor contractility reach only 20% (48). Each is dependent on the mix of patient acuity and intraoperative management. Despite success with inotropic agents, patients occasionally require a mechanical device for poor myocardial performance. An intraaortic balloon pump may offer significant hemodynamic utility and is considered throughout the management of significantly compromised contractility, especially when related to the volume-overloaded myocardium. Rarely, patients arrive from the operating room with, and even less often return to the operating room for insertion of, a ventricular assist device.

Other Hemodynamic Diagnostic Issues

Hypotension or hypoperfusion may result from insults not directly related to the anticipated effects of the operative procedure and include cardiac tamponade, new myocardial ischemia, tension pneumothorax, significant hemothorax, and retroperitoneal or thigh hemorrhage related to recent femoral artery cannulation. Rarely, acute thrombosis of a bypass graft, graft spasm (49), or coronary emboli to a graft occur. An ECG may be of diagnostic help because the initial postoperative ECG is expected to be unchanged from preoperatively or reveal subtle ST-T wave abnormalities. If more dramatic changes appear on a repeat ECG, loss of a graft becomes a strong consideration. A return to the operating room or to the cardiac catheterization
operative abnormality. Abnormal patterns within the first 24 hrs include relative bradycardia (70–80 beats/min), “excessive” sinus tachycardia (>110 beats/min), and if valvular surgery, junctional tachycardia with atrioventricular interference or heart block. Valvular surgery augments the frequency of rate and conduction disturbances. Inconsequential right bundle branch block occurs in approximately 10% of patients. Atrial tachydysrhythmias in the first 24 hrs are uncommon, especially in comparison with later in the postoperative course. Infrequently, significant ventricular dysrhythmias arise and, if present, may be resistant to therapy. Altered rate or conduction may require no intervention or require aggressive management, based on the severity and underlying myocardial performance.

Patients return from the operating room with epicardial pacing leads brought to the surface via the subxiphoid region and include a ventricular lead, a subcutaneous ground, and possibly an atrial lead. Thus, ventricular or atrial pacing, sometimes used in combination with chronotropic agents, is available. The heart rate most useful to assist perfusion is higher than usually thought necessary. When blood pressure or CI is marginal, consider augmenting heart rate, even if it is already in the normal range. When managing the chronically pressure-overloaded heart, as from the hypertrophy of aortic stenosis, rate and rhythm play larger roles. Sinus rhythm at 100 may be required, whereas sinus rhythm at a rate of 90 or atrial fibrillation at 100 may result in noticeable deterioration. A junctional rhythm of 110–120 may be more efficacious than a sinus rhythm of 80. When the risk of progressive block seems excessive, ventricular demand pacing at a “safety rate” (usually 50–60 beats/min, or that which is approximately 10 beats/min below the current intrinsic rate and consistent with effective perfusion and pressure) is utilized.

Management of dysrhythmias includes empirical therapy with magnesium and potassium, although they do not often result in conversion. Restitution of sinus

<table>
<thead>
<tr>
<th>Low Cardiac Index</th>
<th>High Cardiac Index</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low blood pressure</td>
<td>Epinephrine</td>
</tr>
<tr>
<td></td>
<td>Milrinone or dobutamine with norepinephrine, vasopressin, or phenylephrine</td>
</tr>
<tr>
<td></td>
<td>No norepinephrine, vasopressin, or phenylephrine</td>
</tr>
<tr>
<td>Low heart rate</td>
<td>Pace, epinephrine, isoproterenol, dopamine</td>
</tr>
<tr>
<td></td>
<td>No therapy</td>
</tr>
<tr>
<td>High blood pressure</td>
<td>Afterload reduction</td>
</tr>
<tr>
<td>High heart rate</td>
<td>Epinephrine</td>
</tr>
<tr>
<td></td>
<td>Milrinone or dobutamine with norepinephrine, vasopressin, or phenylephrine</td>
</tr>
<tr>
<td></td>
<td>No therapy, rarely beta blockade</td>
</tr>
</tbody>
</table>

Heart Rate/Rhythm

Altering in heart rate (55, 56) or conduction (57) may subtly contribute to hypotension or hypoperfusion. Heart rates of 90–100 beats/min are the norm, unless ameliorated by preoperative medication or preexisting disease of pulse generation or propagation. Heart rate and conduction disturbances may occur in the postoperative period, related to the operative procedure, but are often a preoperative abnormality. Abnormal patterns within the first 24 hrs include relative bradycardia (70–80 beats/min), “excessive” sinus tachycardia (>110 beats/min), and if valvular surgery, junctional tachycardia with atrioventricular interference or heart block. Valvular surgery augments the frequency of rate and conduction disturbances. Inconsequential right bundle branch block occurs in approximately 10% of patients. Atrial tachydysrhythmias in the first 24 hrs are uncommon, especially in comparison with later in the postoperative course. Infrequently, significant ventricular dysrhythmias arise and, if present, may be resistant to therapy. Altered rate or conduction may require no intervention or require aggressive management, based on the severity and underlying myocardial performance.

Patients return from the operating room with epicardial pacing leads brought to the surface via the subxiphoid region and include a ventricular lead, a subcutaneous ground, and possibly an atrial lead. Thus, ventricular or atrial pacing, sometimes used in combination with chronotropic agents, is available. The heart rate most useful to assist perfusion is higher than usually thought necessary. When blood pressure or CI is marginal, consider augmenting heart rate, even if it is already in the normal range. When managing the chronically pressure-overloaded heart, as from the hypertrophy of aortic stenosis, rate and rhythm play larger roles. Sinus rhythm at 100 may be required, whereas sinus rhythm at a rate of 90 or atrial fibrillation at 100 may result in noticeable deterioration. A junctional rhythm of 110–120 may be more efficacious than a sinus rhythm of 80. When the risk of progressive block seems excessive, ventricular demand pac-
rhythm rather than rate control is the typical ultimate goal. Amiodarone is the mainstay of therapy, and digoxin may be in the medication mix in the management of patients with chronic heart failure and atrial fibrillation. Beta blockade is safe and more easily utilized after 24 hrs, when myocardial performance has improved. Beta blockade used to improve long-term outcome from MI is typically restarted after 24 hrs, even when no atrial dysrhythmias are evident. When chemical restoration of sinus rhythm fails, electrical cardioversion is an important additional therapeutic consideration once rate control is attained. It often fails when used early or before rate control during this period of heightened adrenergic activity. Once in sinus rhythm, the medication regimen for atrial dysrhythmias continues for a month or longer. After 48 hrs of atrial fibrillation, anticoagulation is considered, despite recent surgery, and is continued for a similar period as the antidyssrhythmias.

Post–cardiac surgery atrial tachydysrhythmias have a direct effect on outcome and resource consumption. The hospital rate of cerebrovascular accidents is 3–5%, and it increases to 5–7% in the event of atrial fibrillation (60). Hospital length of stay becomes prolonged (61), patients require additional medications, and when persistent, anticoagulation. Consequently, innumerable medication regimens starting preoperatively or postprocedure to avoid atrial fibrillation have been studied (62–64). Most are partially successful, but none have proved stellar (65).

Cardiac Arrest

A combination of events may result in rapidly evolving pulseless electrical activity (66). In addition to basic life support and advanced cardiac life support protocols, clinicians must consider opening the sternum in the ICU. Occasionally, the cause of pulseless electrical activity is cryptic tamponade, and restitution of blood pressure and perfusion is rapid after gaining access to the mediastinum. Simultaneously, an anatomic cause, such as graft closure, stretching, or kinking of a graft, or intrathoracic hypertension, which is analogous to intraabdominal compartment syndrome, may become evident once the sternum is opened. Tamponade has been reported secondary to pleural effusion (67). On opening the chest, responsibilities beyond advanced cardiac life support arise for the critical care practitioner, who then becomes simultaneously responsible for resuscitation, anesthesia, and supporting the surgical team. Often, the initial surgeon is a house physician or resident awaiting the arrival of a cardiac surgeon. Most important is for the clinician to determine when to open the sternum and to possess skills in managing an extended team, not frequently faced with this scenario.

Bleeding

General Considerations. After cardiac surgery, the management of hypotension, hypoperfusion, and alterations in rate or rhythm demand much attention. Anemia may be present and be due to preoperative anemia and the usual intraoperative and postoperative hemodilution and blood loss (68, 69). Invasive cardiologic procedures that preceded cardiac surgery increase the risk of preoperative anemia. The cardiac surgery patient population consumes enormous blood product resources when considered on a national scale. The societal and individual patient implications result in efforts to minimize use. The minimum necessary hemoglobin has drifted down substantially in recent years. The hemoglobin goal is determined by myocardial performance, age, and other overt clinical risks. These considerations have resulted in programmatic algorithms (70, 71). For example, a hemoglobin of 7.5 may be the acceptable minimum in those <75 yrs old with good performance and no other risks. By contrast, 8.5 may be the chosen threshold for those >75 yrs old. Some argue that hemoglobin thresholds established to conserve blood and reduce associated complications of transfusion require upward revision (72). Despite guidelines, an average of 2 units of packed red blood cells is given to 30–50% of patients transfused (73, 74). Those refusing blood products, such as Jehovah’s Witnesses, present significant challenges. Management of Jehovah’s Witnesses mandates particular attention to minimizing surgical blood loss, decreased lab testing, and intervention with inotropic or vasopressor medications for hemodynamic compromise rather than volume resuscitation.

Mediastinal and Pleural Tubes. Postoperative bleeding via mediastinal and pleural drains (and to a lesser extent, saphenous vein harvest sites) contributes to anemia and, if excessive, may signify need for medical or surgical therapy. The pathogeneses of bleeding are medical and occasionally surgical. Thrombocytopenia arises from a variety of mechanisms, most important of which are hypothermia, recent antiplatelet therapy, and cardiopulmonary bypass (75, 76). Dilution and heparin contribute to the coagulopathic state. Thrombocytopenia commonly occurs, but the platelet level typically remains >100,000. Primary or significant fibrinolysis and disseminated intravascular coagulation rarely occur. Intraoperatively, most patients receive epsilon amino caproic acid (Amicar) or the more expensive aprotinin (Trasylol) to minimize bleeding (77), without apparent risk of secondary graft thrombosis. The routine use of fresh frozen plasma or platelets in the operating room is questionable. A minor degree of “background” bleeding is the norm during the
first 10 hrs (Fig. 3). Programs experience a broad range of bleeding, resulting in no uniform definition of excessive. Commonly, 10-hr ICU blood loss is <500 ml, with >1000 ml occurring infrequently. Most loss occurs in the first 4 hrs. Mild to moderate bleeding usually subsides without intervention. More than 200 ml/hr for 2 hrs may be considered excessive and generally mandates intervention. Medical intervention includes initial empirical platelet transfusion and additional protamine, if indicated. Fresh frozen plasma is ordered subsequently. Red blood cells may be required. In the most severe bleeding, blood products (fresh frozen plasma and platelets) are ordered simultaneously and empirically. Very rarely, cryoprecipitate and activated factor VII play roles after platelet and fresh frozen plasma transfusions have failed. The specific situations for use of activated factor VII remain to be elucidated. Two situations require a return to the operating room. They include persistent excessive bleeding and tamponade (78, 79). The decision for exploration is ordinarily individualized, although algorithms (Table 3) based on quantitative blood loss, hemodynamic compromise, and risk are utilized occasionally. Often on return to the operating room, only diffuse “venous” oozing is noted and is managed by electrocautery, additional blood products, and lavage to remove clot and fibrinolytic byproducts. Sources of significant surgical bleeding include tributaries of the internal mammary arteries and saphenous veins, atrial bypass cannulation sites, aortic or coronary anastomotic targets, and cardiopulmonary cannulation sites.

Tamponade becomes a diagnostic consideration during excessive bleeding. It may result when bleeding remains within the norm or abruptly stops. It occurs in the first 12 hrs, although delayed-onset tamponade may present a week or more later, especially when patients are anticoagulated. A high index of suspicion is most important in making this diagnosis. An inappropriately fluctuating MAP, low CI, sudden oliguria, sudden cessation of mediastinal bleeding, or upper torso plethora, either separately or together, should stimulate consideration of tamponade. The classic clinical signs of diminished heart sounds, pulsus paradoxus, and distended neck veins do not help. Often, a strong clinical suspicion or overt tamponade are present before an increase and equalization of pulmonary artery and right ventricular and atrial pressures occur. Echocardiograms (80), both transthoracic and transesophageal, may help if positive but may falsely indicate no tamponade. Be aware that the pericardium is left open and that occasionally tamponade results from localized clot preventing filling. Consequently, the echocardiographer must realize these possibilities to astutely evaluate test results (81, 82). Surface echocardiograms may be insensitive because of these issues and because of technical difficulties in performing the study that occur during mechanical ventilation. Patients may need a return to the operating room based on clinical judgment with a “negative” echocardiogram. Seasoned clinicians act on a presumptive diagnosis without an echocardiogram.

THROUGHPUT AND OUTCOME

After the initial intense 24 hrs, the majority of patients recover smoothly. Rapid throughput programs are designed to minimize the effects of surgery and allow patients to recover quickly (83–101). In them, 85–90% leave the ICU for a telemetry unit within 24 hrs, often in 15–20 hrs. The next 5% move to less intense settings by 48–72 hrs. The final 5% remain in an ICU for many days and are at the same risks as all ICU patients. The post-ICU length of stay is short for most, resulting in discharge to home after a hospitalization of 5–7 days. At hospital discharge, patients require numerous medications that are begun during the hospital postoperative course and include aspirin (102, 103), beta blockade, angiotensin-converting enzyme inhibition, and occasionally clopidogrel. Overall mortality is <5% and is as low as 1–2% for first-time coronary artery surgery patients. Most, even those with severe myocardial dysfunction, survive (104, 105). Occasionally, prolonged low-dose inotropic support carries this group through the stress of increased oxygen consumption related to ventilator weaning. The inotrope is then empirically titrated off a few days after extubation. Complications atypical of other ICU patients may intervene in those with a prolonged ICU length of stay. Mediastinitis (106, 107) or other wound infections, late tamponade (108), and mesenteric ischemia are three complications to consider if the cause of deterioration seems cryptic. Also, because this patient population receives multiple doses of heparin, scrutiny of serial platelet counts is needed because heparin-induced thrombocytopenia may occur (109).

CONCLUSIONS AND FUTURE DIRECTIONS

Despite the risks and intense management required for cardiac surgery patients, full recovery and rapid discharge from the hospital are normative. This dynamic field continues to rapidly evolve. Patient populations shift as demographic, medications, and invasive cardiologic procedures change. Numerous advances in preoperative, intraoperative, and postoperative management have emerged in recent years. Operating room management evolves as new technologies, surgical techniques (110), medications, and management schemes are adopted. Areas ripe for advancement include artificial valve technology, valve repair surgery, cardioprotective strategies (111), beating-heart surgery, and other less invasive techniques. The growing population of patients with heart failure and paucity of human heart transplants has resulted in surgical advances for chronic heart failure (112, 113). Numerous surgical techniques to ameliorate heart failure and an explosion of assist devices are now the subject of intense study and debate. Xenotrophic transplantation may play a role in care of such patients. Layered on top of these evolutionary pathways must be the development of more risk-predictive methodologies coupled to the intended procedure, surgeon, and program. Astute incorporation of significant advances demands sophisticated provider systems.

In summary, after cardiac surgery, patients undergo significant and predictable, usually temporary, physiologic aberrations. These physiologic aberrations vary as the patient population, surgical/anesthetic techniques, and programmatic postoperative care differ. As the contin-

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**Table 3. Indications for exploration for excessive hemorrhage**

<table>
<thead>
<tr>
<th>Blood Loss Per Hour, mL</th>
<th>No. of Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;200</td>
<td>4</td>
</tr>
<tr>
<td>&gt;300</td>
<td>3</td>
</tr>
<tr>
<td>&gt;400</td>
<td>2</td>
</tr>
<tr>
<td>&gt;500</td>
<td>1</td>
</tr>
</tbody>
</table>

Typical indications for mediastinal exploration after cardiac surgery based on the number of hours of excessive mediastinal drainage tube output; tamponade is an indication for exploration regardless of the quantity of drainage.
uum of care grows in complexity, surgeons look to their critical care colleagues to assist in patient care, process management, and programmatic development (115).

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