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Early Postoperative Care After Cardiac Surgery

Paul M. H. J. Roekaerts¹ and John H. Heijmans²

¹*Department of Intensive Care*

²*Department of Anaesthesiology
University Hospital Maastricht, Maastricht
The Netherlands*

1. Introduction

The early postoperative course for most patients after cardiac surgery is characterized by a typical pattern of pathophysiologic derangements that benefits from standardized care. Multimodal, multidisciplinary standardization of the care process has been shown to improve use of resources, efficiency, quality, safety and patient satisfaction.

The initial management in the postoperative care after routine cardiac surgery has fundamentally shifted during the past two decades towards a more efficient use of limited postoperative care facilities, early extubation and rapid discharge. The fast-track protocol became feasible after cardiac surgery due to improvements in perioperative anaesthesia management, new surgical techniques, better myocardial protection and cardiopulmonary bypass techniques and to better management of bleeding using point-of-care testing and new hemostatic drugs.

This chapter will briefly discuss the major pathophysiologic derangements and their management during the first 24 hours after surgery. It will then summarize the postoperative care to more specific procedures. Finally, the management of common postoperative complications will be discussed.

2. Pathophysiology during the early postoperative period

Arrival in the intensive care unit

Upon arrival in the ICU, an efficient transfer of care from operation room staff to ICU staff is mandated, while at the same time vital signs are to be maintained stable.

The initial goals in postoperative cardiac recovery are sufficient analgesia, normothermia, adequate oxygenation and ventilation, control of bleeding, restoration of intravascular volume, optimization of blood pressure and cardiac output to maintain organ perfusion and metabolic stabilization.

Hypothermia

Hypothermic cardiopulmonary bypass is usually terminated after the patient has rewarmed to a core body temperature of at least 36 °C. (1) However, patients usually arrive in the ICU with lower core temperatures. This drop in temperature from end of CPB until arrival in the ICU is due to the cool ambient temperatures in the operation room, poor peripheral perfusion and anesthesia-induced inhibition of normal thermoregulation. Even patients

operated under normothermic CPB, have a tendency to significantly cool down before conclusion of surgery.

Hypothermia has many potential adverse effects. (2) It increases the systemic vascular resistance (SVR) which increases myocardial afterload and myocardial oxygen demand. This compensatory mechanism to provide core warming may contribute to slow warming of peripheral tissues. Drugs that provide vasodilation may improve peripheral perfusion. To prevent hypotension, warmed infusions should be administered concomitantly. Peripheral vasodilation augments heat loss, and core hypothermia may therefore persist.

Hypothermia also precipitates shivering, thereby increasing CO₂ production and oxygen consumption, and predisposes to ventricular arrhythmias and coagulation cascade impairments. (3,4)

Therefore, warming should be hastened by forced-air warming blankets, heated humidifiers in the ventilator circuit and warmed infusion fluids. The use of other types of warming blankets or radiant heating hoods can also be considered. (5)

After cardiac surgery, patients may rapidly rewarm and occasionally overwarm to higher temperatures. This phenomenon is attributed to the resetting of the central thermoregulation system.

Blood loss after cardiac surgery

Careful hemostasis in the operation room is the cornerstone in reducing postoperative blood loss. However, bleeding can also be medical and determining the cause of bleeding is often difficult. Although the clinical situation must be individualized for each patient, bleeding in general should not exceed 400 mL/hr during the first hour, 200 mL/hr for each of the first 2 hours, or 100 mL/hr over the first four hours. (6)

There are numerous medical causes for bleeding following cardiac surgery. Residual heparinization is common post cardiac surgery and usually occurs when insufficient protamine is used or heparinized pump blood is transfused following CPB.

Platelet dysfunction is also common following cardiac surgery. The CPB circuit itself leads to contact activation and degranulation of platelets, resulting in their dysfunction. Fibrinolysis frequently occurs after CPB, caused by activation of inflammatory or coagulation pathways.

Coagulation factors may decrease from activation and dilution in the CPB circuit.

There has been a dramatic increase in the iatrogenic use of heparin and newer antiplatelet, antithrombotic and thrombolytic drugs during (interventional) treatment of acute coronary syndromes. If revascularization surgery is warranted immediately after these treatments, the anticoagulant effect of these drugs is notable in the postoperative period.

Conventional coagulation tests are helpful to identify the coagulation abnormality contributing to the bleeding. Common laboratory testing includes Hb, platelet count, aPTT, INR, and fibrinogen level. Thromboelastography is also commonly used and has been demonstrated to reduce transfusion requirements.

The most basic principles of the management of postoperative bleeding are:

1. Diagnose underlying medical cause by coagulation tests;
2. Rule out surgical bleeding;
3. Restore clotting parameters to normal by means of medications, transfusion of blood products or clotting factors, and restore normothermia;
4. Monitor for stability of clotting parameters.

Most cardiac surgical centers use the antifibrinolytic lysine analogues, tranexaminic acid and aminocaproic acid, to reduce intraoperative bleeding. These drugs significantly reduce allogeneic blood transfusion after cardiac surgery.

Although rescue therapy with recombinant factor VII can be life-saving in massive bleeding after cardiac surgery, its safety has been questioned. A recent meta-analysis (N = 4468 from 35 studies) demonstrated that this therapy significantly increased the rate of arterial but not venous thromboembolic events. Given its cost and arterial thrombotic risk, it is likely that this hemostatic intervention will continue to be reserved for life-saving therapy of massive coagulopathy after cardiac surgery.(7)

Blood transfusion management

Although there are guidelines for blood transfusion in cardiac surgery, considerable variability has persisted in clinical practice. This variability also exists in anticoagulation and coagulation management.

A recent randomized controlled trial has already demonstrated that restrictive perioperative transfusion does not result in inferior clinical outcome after cardiac surgery. (8) Transfusion burden may in the future be interpreted as a quality indicator in cardiac surgery that must balance risks and benefits to achieve cost-effective optimal clinical outcomes. (9,10)

Perioperative transfusion algorithms for the administration of blood products, coagulation factors and pro-coagulant drugs should assist in preserving resources with improvement in patient safety. (11)

Fluid resuscitation

Cardiac surgery and CPB elicit a systemic inflammatory response which produces a capillary leak. Therefore, fluid resuscitation with cristalloids and/or colloids is necessary to offset the hemodynamic consequences of the capillary leak and the vasodilation that occurs from rewarming and vasodilating drugs. However, the maintenance of intravascular volume in the leakage phase occurs at the expense of expansion of the interstitial space. (12,13)

After the capillary leak has ceased and hemodynamics have stabilized, diuretics are often used to eliminate the excessive salt and water administered during surgery and the early postoperative phase. This forced diuresis may beneficially affect pulmonary function and early successful extubation.

Several intraoperative measures that have been implemented throughout the years caused a reduction in the inflammatory response and may have contributed to the faster recovery times currently observed after cardiac surgery. The measures include the use of membrane oxygenation, centrifugal pumps, anti-fibrinolytic drugs and steroids, leukocyte filters and coated CPB tubings. (14,15)

Perioperative cardiovascular dysfunction

Adequacy of organ perfusion and tissue oxygenation is the primary goal of hemodynamic management in the postoperative cardiac surgical patient. Preload, afterload and contractility should therefore be maintained at their optimal level. This commonly requires atrial or atrioventricular pacing.

Approximately 20 % of cardiac surgical patients develop cardiovascular dysfunction in the perioperative period, resulting in an inability to pump sufficient blood at normal end-diastolic pressures. There are three distinct clinical scenarios of cardiac impairment in the perioperative period of cardiac surgery - precardiotomy, failure to wean and postcardiotomy - differing from each other substantially concerning diagnosis, monitoring and management.

Precardiotomy heart failure

Myocardial ischaemia is one of the most frequent causes of precardiotomy low output syndrome. The dysfunctional myocardium may not be irreversibly damaged and possibly only 'stunned' or 'hibernating'. Revascularization of the reversibly injured heart areas may result in improved cardiac performance. The first priority should therefore be prompt surgery avoiding further alterations in myocardial contractility, possibly by introducing an IABP preoperatively. However, inadequate myocardial protection during cardiac surgery may exacerbate ischaemic injury in some patients. Patients with longer standing previous poor preoperative cardiac function or with recently irreversibly injured ischaemic heart areas, will of course continue to have poor ventricular performance postoperatively.

Failure to wean

For the successful therapeutic approach of failure to wean, a correct diagnosis of the underlying cause is necessary. The heart failure may be procedure related or patient specific and includes inadequate myocardial protection, reperfusion injury, ischaemia, infarction, incomplete revascularization, metabolic, uncorrected pathology, mechanical issues, conduction issues, pulmonary hypertension and right ventricular failure.

Postcardiotomy heart failure

The priority is to preserve end organ function. Preload and heart rhythm should be optimized, and positive inotropic and/or vasopressor drugs are often used to maintain adequate cardiac output and blood pressure. Although this strategy will restore haemodynamics in most patients, mechanical circulatory support may be indicated.

Monitoring and assessing volume status

Heart failure cannot be ascertained unless the volume status is optimal. However, it is difficult to ascertain volume loading using single haemodynamic measures. Pressure estimates such as pulmonary capillary wedge pressure and central venous pressure are generally unreliable indicators of LV and RV preload. Uncoupling between PCWP and LVEDP frequently occurs as a consequence of elevated pulmonary vascular resistance, pulmonary venoconstriction, mitral stenosis and reduction in transmural cardiac compliance.

Volumetric estimates by echocardiography or transpulmonary thermal dilution techniques are more predictive of preload. In predicting fluid responsiveness in ICU patients, it is preferable to use more reliable dynamic indicators reflecting hypovolaemia, such as stroke volume variation, than static parameters. (16) Several devices are now being used to assess cardiac function based on pulse contour analysis of an arterial waveform. (17)

Echocardiography is of great value in the perioperative cardiac surgical setting. It not only is helpful in assessing the optimal volume status, but may also immediately identify causes of cardiovascular failure, including valvular problems, cardiac tamponade, systolic anterior motion of the anterior mitral valve leaflet and pulmonary embolism. Echocardiography may differentiate between acute right, left and global heart failure as well as between systolic and diastolic dysfunction.

If there are echocardiographic signs of RV failure, a pulmonary artery catheter (PAC) preferably with continuous SvO₂ measurement should be introduced. PACs can differentiate between pulmonary hypertension and RV ischaemia, which necessitates a reduction of RV afterload. PAC and TEE are complementary to each other for diagnosis and treatment of the cardiac surgical patient. Indications for the use of a PAC are, high risk and/or complex cardiac surgery, hemodynamic instability, low cardiac output syndrome, pulmonary hypertension,

differentiating between severe right and left ventricular dysfunction, vasodilation/vasoconstriction, hypovolemia. SvO₂ in combination with lactate concentration was used postoperative as a goal-oriented hemodynamic therapy to improve outcome. (18,19)

Risk stratification

Risk stratification is increasingly used in open-heart surgery to help adjust resources to predicted outcome. According to all scoring systems major clinical risks include heart failure, unstable coronary syndromes, significant arrhythmias and severe valvular disease. The euroSCORE is mostly used to calculate operative risk, although updating its sensitivity is warranted. (20,21)

In addition to scoring systems, levels at hospital admission of B-type natriuretic peptide (BNP) and the amino-terminal fragment of pro-BNP (NT pro-BNP) are powerful predictors of outcome with regard to in-hospital mortality and rehospitalisation in heart failure patients. (22)

Perioperative myocardial protection

The ultimate goal of perioperative myocardial protection is to limit the extent and consequences of myocardial ischaemia-reperfusion injury. This injury is caused by free radical formation, calcium overload and impairment of the coronary vasculature. Protective measures include the use of free radical oxygen scavengers, inhibitors of the complement system and neutrophil activation, modulation of intracellular gradients and maintenance of sufficient myocardial high energy phosphate stores. Drugs affecting the complement-inflammation pathways, adenosine modulators, cardioplegia solution adjuvants, Na⁺/H⁺ exchange inhibitors, K_{ATP} channel openers such as volatile anaesthetics and levosimendan, and anti-apoptotic agents are all used for this purpose. (23,24)

Pharmacologic support of myocardial dysfunction

Pharmacological treatment of low cardiac output and reduced oxygen delivery to vital organs is often required in the perioperative cardiac surgical setting. Inadequate treatment may lead to multiple organ failure, one of the main causes of prolonged hospital stay, postoperative morbidity and mortality. Optimal use of inotropes and vasopressors is still controversial and needs further large multinational randomized controlled trials.

However, some recommendations can be made:

- Norepinephrine should be used in case of low blood pressure due to vasoplegia to maintain an adequate perfusion pressure. Preload should be assessed regularly to avoid hypovolemia under vasopressors.
- All catecholamines have positive inotropic and chronotropic effects. There is evidence that dobutamine better preserves the myocardial oxygen balance as compared to the other commonly used drugs. Dobutamine increases stroke volume and heart rate while PCWP is moderately decreased. If blood pressures are low, the combination dobutamine-norepinephrine is frequently used.
- Phosphodiesterase III inhibitors are potent vasodilators and cause less tachyarrhythmias as compared to dobutamine. They also have a more favourable effect on the myocardial oxygen balance as compared to the catecholamines.
- Levosimendan, a calcium sensitizer, has recently been introduced for the treatment of low cardiac output in the perioperative period with success. (25,26,27,28)

Mechanical circulatory support

The *intra-aortic balloon pump (IABP)* is recommended in the case of heart dysfunction with suspected coronary hypoperfusion. It's main mechanism of action is a reduction of afterload

and diastolic coronary perfusion pressure. The IABP reduces heart work and myocardial oxygen consumption, favourably modifying the balance of oxygen supply/demand.

Extra-corporeal membrane oxygenation (ECMO) is increasingly used for temporary mechanical circulatory support. Advantages of the system include low cost, availability in all cardiac surgical centers and versatile use for cardiac, pulmonary and renal support. ECMO is used as a bridge to recovery, to transplantation, to long-term assist-device and to decision making.

Ventricular assist devices are used today as an established option for patients with end-stage heart failure to obtain a level of functionality that results in an acceptable quality of life for the patient.

Cardiac arrhythmias

Temporary pacing

Two temporary right atrial and two right ventricular epicardial pacing wire electrodes are usually placed at the conclusion of cardiac surgery. Atrial pacing wires can be used diagnostically to record atrial activity. These recordings, obtained simultaneously with standard limb leads, can distinguish among atrial and junctional arrhythmias and differentiate them from more life-threatening ventricular arrhythmias. (6)

The use of pacing is often required in the per- and postoperative period to increase heart rate. Atrial or AV pacing will nearly always demonstrate superior haemodynamics to ventricular pacing. Reentrant rhythms can be terminated by rapid pacing.

Arrhythmias after cardiac surgery

The development of cardiac arrhythmias following open-heart surgery is fairly common and related to altered impulse formation and conduction. An understanding of these mechanisms and the electrophysiologic effects of antiarrhythmic drugs provides a rational basis for the treatment of the different rhythm disturbances.

Atrial fibrillation

Despite various prophylactic measures, atrial fibrillation and flutter occur in about 35 % of all cardiac surgical patients, most commonly on the second and third postoperative day. Etiologic factors include atrial distension, pericardial inflammation, enhanced sympathetic activity, surgical trauma and poor atrial preservation.

To prevent atrial fibrillation, β -blockers with or without class III (Sotalol) antiarrhythmic properties, are commonly administered orally in the perioperative phase. Dual site atrial pacing and numerous other medications (amiodarone, magnesium sulphate, triiodothyronine, digoxin, steroids, procainamide, verapamil, diltiazem) have all been reported to have some favourable effect on the incidence of atrial fibrillation after cardiac surgery.

Treatment consists of cardioversion in the haemodynamically unstable patient. For the stable patient, rate control and attempts to achieve conversion are usually initiated. Drugs used for rate control include calcium-channel blockers (diltiazem, verapamil), β -blockers (esmolol, metoprolol), magnesium sulfate and digoxin. For conversion to sinus rhythm, magnesium sulphate, IA medications (procainamide, quinidine) , IC (propafenone) or III antiarrhythmics (Ibutilide, Amiodarone) are commonly used. (29,30,31)

Ventilation management

Pulmonary complications following cardiac surgery are common, even in patients with healthy lungs, and include diminished functional residual capacity (FRC) following general anaesthesia and muscle relaxants, reductions in vital capacity (VC) following median

sternotomy and intrathoracic manipulation, atelectasis, increased intravascular lung water, and increased capillary leakage and extravascular lung water due to the inflammatory response to CPB and surgery. Multiple blood product transfusions and excessive fluid loading may further compromise lung functioning. Acute FRC reduction results in arterial hypoxemia due to ventilation-perfusion mismatch and shunting. In the early postoperative phase, restoration of FRC and maintenance of adequate gas exchange in the face of rising VO_2 and VCO_2 are the primary goals. This can be achieved by a lung-protective ventilation strategy with adequate levels of PEEP. (32,33,34)

For several decades, the medical care of the cardiac surgical patients in the perioperative setting consisted of high-dose opioid stress-free anaesthesia and prolonged mechanical ventilation in the ICU. In recent years, the concepts of Fast-Track Cardiac Anaesthesia, Early Extubation and Short-Stay Intensive Care became the backbone of modern perioperative care. Indeed, several randomized trials have shown the safety of fast-tracking. (35,36,37,38,39)

Alterations in anaesthetic protocols using short-acting sedatives-hypnotics and analgesics, less invasive surgical and perfusion techniques, improved perioperative haemostasis management, fluid restriction, preservation of normothermia and reduction of the inflammatory response were all crucial steps in the development of fast-track cardiac surgery. As the number of elderly people is growing fast and cardiac surgery is now an accepted practice in these older patients, fast-tracking makes it possible to more efficiently use the limited facilities and resources. (40)

The debate regarding the optimal extubation time, the window of opportunity, is still ongoing. (41) There are several studies on outcome after extubation in the operation room, which show that it is feasible with good results. (42, 43) However, the nadir of ventricular function occurs about 4 hours following cardiopulmonary bypass. Also, the first few hours after cardiac surgery are characterized by periods of haemodynamic instability, temperature dysregulation, increased mediastinal blood loss and other homeostatic disturbances. Patients can rapidly deteriorate in this early postoperative phase and we believe that instabilities can be best anticipated and treated in an ICU setting in sedated and ventilated patients. The window of opportunity for extubation is therefore between 2 and 6 hours postoperatively.

Weaning strategies should be protocolized. (44) In table 1 is shown a nurse-driven weaning protocol which includes the criteria for the start of the weaning procedure, adequate breathing criteria and the extubation criteria. Only three steps in this protocol may mandate the consultation of the ward doctor.

After ventilatory weaning, the next step in the postoperative ICU management is the discharge of the patient to a step-down unit. This is usually accomplished within 8 hours after arrival in the ICU. Intensive Care discharge criteria are shown in Table 2.

The postoperative (intensive) care unit

The advent of Fast-Track Cardiac Anaesthesia and Short-Stay Intensive Care after Cardiac Surgery also started the discussion whether or not these patients should be treated in a conventional ICU setting. Can adequate and safe postoperative care be given to these patients in parallel "special-care units" such as a dedicated Cardiac Recovery Area (CRA). If a hospital has such a highly-equipped special care unit with a competent and qualified ICU doctor on the ward, adequate nurse-patient ratio and immediate access to ICU-OR facilities, then special care may be feasible. Several institutions reported safe and adequate care in these special units. (45,46) However, in the early postoperative phase, the clinical condition of the patient may

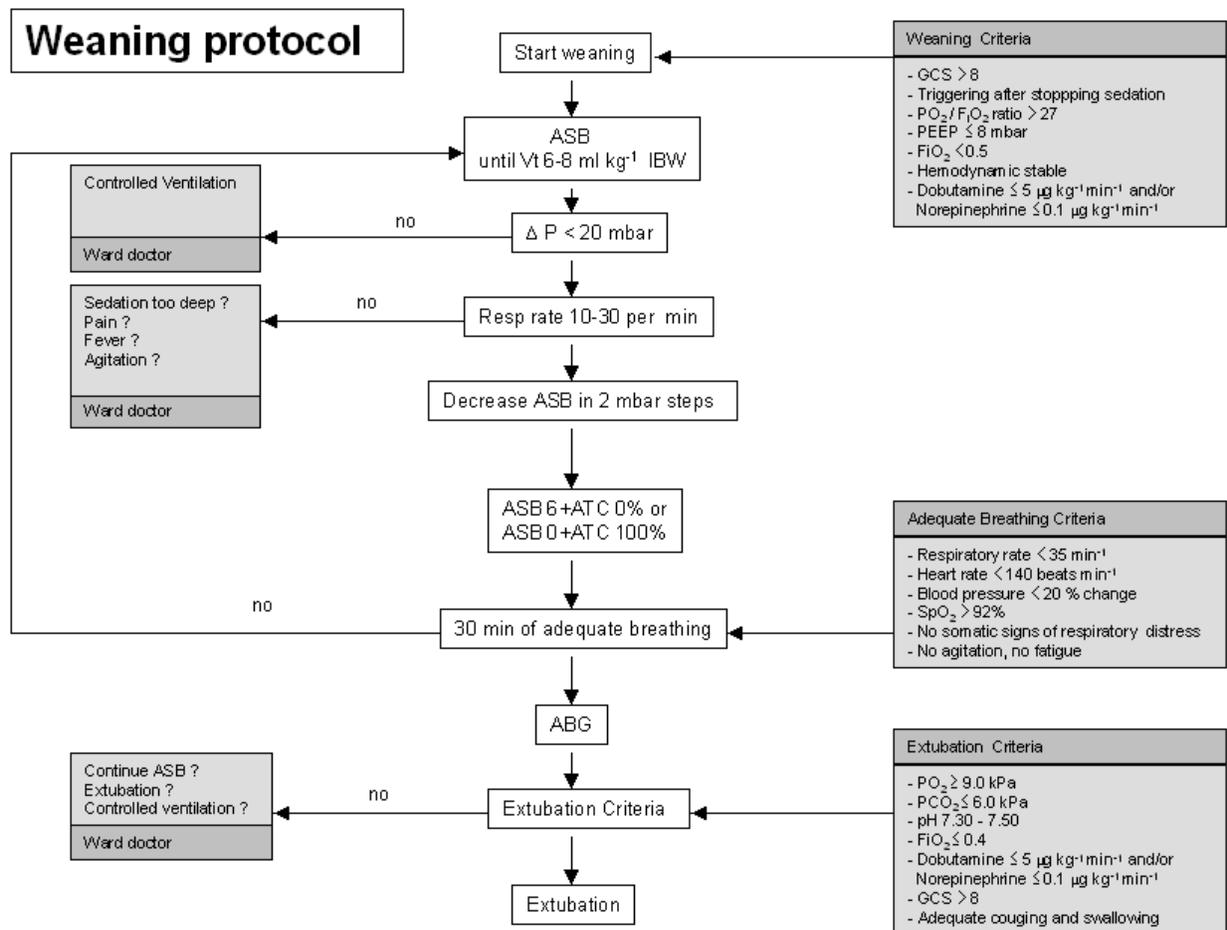


Table 1. Weaning protocol

ICU Discharge Criteria

Pulmonary	Extubation > 30 min Oxygen < 5 L min ⁻¹ nasally Respiratory rate > 10 min and < 25 min PaO ₂ > 9 kPa and PCO ₂ < 6.5 Kpa
Cardiac	No myocardial ischaemia or ongoing infarction No haemodynamically significant dysrhythmias
Fluid balance	Chest tube drainage < 100 mL hr ⁻¹ Diuresis > 0.5 mL Kg ⁻¹ hr ⁻¹
Neurologic	No signs/symptoms of major neurologic complications
Haemodynamic	No iv vasoactive drugs Except dobutamine 2 g Kg ⁻¹ min ⁻¹ and/or nitroglycerine 0.5 g Kg ⁻¹ min ⁻¹ No IABP Cardiac Index > 2 L min ⁻¹ m ⁻²

Table 2. ICU Discharge Criteria

deteriorate extremely rapidly. Therefore, continuous adequate monitoring and maximal acute treatment or intervention should always be readily possible for these patients in the early postoperative period. We believe that currently in most hospitals, the ICU setting is the safest and best place to recover from cardiac surgery. In the integrated model, in which all patients are admitted to the ICU, the postoperative management such as nursing-to-patient ratio is variable based on patient requirements. The goal is thus a postoperative unit that allows variable levels of monitoring and care based on patient need. In this model, discharge to a step-down unit as soon as possible after extubation and stabilization of vital parameters should be strived after for every single patient.

Postoperative anticoagulation

After *coronary artery surgery*, antiplatelet therapy has been shown to inhibit platelet deposition on vein grafts and may delay or attenuate the development of fibrointimal hyperplasia and atherosclerosis. Aspirin should therefore be started after CABG surgery and continued indefinitely because of its beneficial effects in patients with native coronary disease. (47)

After tissue *aortic valve surgery*, there is some evidence that short-term anticoagulation may reduce the incidence of thromboembolism. Therefore, anticoagulation is generally recommended for 3 months in younger patients or those with no contraindication for anticoagulation, and is then converted to aspirin. If anticoagulation is not used, aspirin is given. (48,49)

After mechanical aortic valve surgery, all patients should receive anticoagulation indefinitely to achieve an INR of 2.5 – 3.5 for tilting and bileaflet valves.

After mitral tissue valve or mitral ring implantation, anticoagulation should be given for 3 months to achieve an INR of 2.0 – 3.0 and should then be converted to aspirin if the patient is in sinus rhythm. Anticoagulation should be continued indefinitely in patients with atrial fibrillation, an enlarged left atrium (> 50 mm in diameter), or a history of thromboembolism. After mechanical mitral valve insertion, anticoagulation is given to achieve an INR of 2.5 – 3.5. The addition of aspirin is safe and may further reduce the thromboembolic risk. (50,51)

Echocardiography

During the past few decades, the effect of perioperative transoesophageal echocardiography's (TEE) influence on perioperative cardiac surgical decision making has become increasingly more appreciated. Data from several clinical investigations have consistently implicated an important, clinically significant, and cost-effective role for TEE as a safe and valuable haemodynamic monitor in identifying high-risk patients, in assessing in the determination of the definitive surgical approach, and in providing a timely post-cardiopulmonary bypass evaluation of the procedure, thereby allowing for the opportunity to immediately re-intervene or to at least triage patients appropriately. In addition, perioperative TEE has been instrumental in diagnosing cardiac and associated great vessel pathology and in identifying structural abnormalities, aortic disease, intracardiac masses, and pericardial disease. TEE is perhaps most useful for the perioperative evaluation of cardiac valvular disease, especially during surgical procedures involving the mitral valve.

In the intra- and early postoperative period of cardiac surgery, an experienced echocardiographer remains an indispensable clinical team member. Increasing numbers of cardiac anaesthesiologists and intensivists are now being trained and certified as perioperative echocardiographers. (52,53,54)

Analgesia and sedation

An essential element of postoperative care is the provision of adequate analgesia and sedation. In the patient in whom delayed extubation is anticipated, the residual effects of anesthetics and midazolam in combination with a narcotic are generally accepted.

With the trend toward earlier extubation, short-acting narcotics and analgesics are administered during surgery. This requires early postoperative administration of short-acting medications for pain relief and sedation. We prefer to give low dose continuous infusions of morphine in combination with propofol in the ICU. This usually produces adequate sedation and pain relief without respiratory depression and allows for fairly early extubation.

3. Management after specific cardiac surgical procedures

Coronary Artery Bypass Grafting

Treatment of coronary artery disease can be medical or interventional. Catheterization procedures include balloon angioplasty, cardiac stenting and drug-eluting stents, which release drugs capable of preventing stenosis. Surgery includes CABG with the use of the cardiopulmonary bypass machine and OPCAB without the use of the CPB machine. OPCAB surgery may include sternotomy, thoracotomy (MIDCAB) or robotically assisted thoracotomy.

With increasing number of treatment options, it is crucial to establish for each patient which option is superior with regard to angina recurrence, graft patency, and long term survival with the least morbidity at the lowest costs. Several studies address this important issue. Analysis of individual patient data from ten randomized trials including 7812 patients concluded that long-term mortality after CABG and PCI in most patient subgroups with multivessel CAD is similar. (55) CABG versus PCI had lower mortality in diabetes and patients older than 65 years. The SYNTAX investigators concluded from their study that in low and intermediate risk patients with multivessel CAD, PCI and CABG have similar outcomes. In high risk patients with multivessel CAD, CABG is preferred. (56)

Although the COURAGE study, which provided optimal medical therapy (OMT) to all patients and demonstrated no incremental advantage of PCI on outcomes other than angina-related quality of life in stable CAD, a recent analysis by Borden et al reported that among patients with stable CAD undergoing PCI, less than half were receiving OMT before PCI and approximately two-thirds were receiving OMT at discharge following PCI. (57,58)

A number of randomized controlled studies comparing OPCAB to on pump CABG have been completed. Although outcomes have been largely comparable, the evidence of benefits of OPCAB from these trials has not been as convincing as was first anticipated. A large adequately powered RCT of OPCAB versus on pump CABG in high risk patients is needed to determine whether this undeniably harder technique is here to stay. (59,60,61)

Aortic Valve Surgery

Aortic valve replacement surgery may be complicated by heart block because the conduction system lies adjacent to the base of the right coronary cusp. If AV pacing is necessary for more than 4 to 5 days, during which time edema or hemorrhage should subside, placement of a permanent DDD pacemaker is necessary because the conduction system then probably has been damaged by sutures or débridement.

The hypertrophied, noncompliant left ventricle in aortic stenosis depends on adequate preload and on atrial contractions. Loss of sinus rhythm is associated with a 30 % reduction in stroke volume and requires AV pacing.

In aortic regurgitation, the left ventricle is volume and pressure overloaded resulting in a dilated and often hypertrophied chamber. Aortic valve repair for aortic regurgitation is evolving into a standard of care. The systematic classification of aortic regurgitation based on leaflet mobility within the functional aortic annulus makes it possible to study outcomes of the specific interventions. (62,63)

A recent RCT showed that transcatheter aortic valve implantation (TAVI) is significantly superior to medical management of severe aortic stenosis in patients judged to be at excessive risk for conventional aortic valve replacement. (64) TAVI significantly reduced all-cause 1-year mortality. Recent studies have documented rates of cerebral embolism of 70-80 %. (65) Future trials should focus on interventions for stroke reduction after TAVI, including cerebral embolic protection. Techniques for reduction of embolic load may also improve renal dysfunction after TAVI. Although the short and medium term durability of the TAVI valve with preserved hemodynamic performance has been established, further studies are required to elucidate the long term effects. (66,67,68) To this term, guidelines for standardized endpoints in TAVI trials have been published.(69)

Mitral valve surgery

Patients with chronic mitral stenosis often have pulmonary hypertension and usually are diuretic-dependent. They have a small left ventricular cavity with preserved LV function. Common postoperative problems are a low cardiac output syndrome associated with the small LV end-diastolic and end-systolic volumes, RV dysfunction and ventilatory failure due to the pulmonary hypertension, cachexia and fluid overload.

Due to the systolic unloading in patients with mitral valve regurgitation reducing LV wall stress, greater systolic wall stress is required after surgery to achieve adequate cardiac output. Therefore, the use of inotropic support and afterload reduction is often indicated.

In the postoperative period, cardiovascular management is often directed toward increasing filling pressures to above 15-20 mmHg, reduction of pulmonary hypertension and improvement of RV and LV failure. Guiding hemodynamic support with the use of a pulmonary artery catheter may be very helpful. When atrial fibrillation has been present for more than 1 year or when LA dimension exceeds 50 mm, it is very unlikely to maintain sinus rhythm in the postoperative period. AV pacing is often possible after surgery and may improve cardiac performance. (6)

Diseases of the thoracic aorta

Multidisciplinary guidelines for thoracic aortic diseases were published in 2010. (70) We will highlight some concerns that concern the perioperative setting.

Ascending aortic *dilatation* should be carefully measured in patients with a bicuspid aortic valve presenting for surgery. Earlier surgical intervention is warranted to avoid rupture or dissection.

In aortic arch *aneurysm* surgery, hybrid repair has emerged as low risk aortic repair in high-risk patients. Type I repairs have adequate proximal and distal landing zones: after off-pump anastomosis of the brachiocephalic vessels to the ascending aorta, an endovascular stent is deployed for complete arch repair. Type II repairs have adequate distal landing zone but insufficient ascending aorta to serve as a proximal stent landing zone: after ascending aortic replacement with aortic arch debranching, an endovascular stent is deployed for

complete arch repair with the ascending aortic graft serving as proximal landing zone. Type III repairs have inadequate proximal and distal landing zones: after total arch replacement with a distal elephant trunk, the descending thoracic aortic repair is completed by endovascular stenting with the elephant trunk serving as the proximal landing zone.

Concerning aortic *dissection*, the Penn classification of a type A dissection integrates type of clinical presentation with dissection extent to stratify perioperative outcome and facilitate decision-making about the type of surgical repair. (71)

The American Heart Association recently published a position paper on the integrated management of descending thoracic aortic disease that complements the recent guidelines from the Society of Thoracic surgeons. (72) These guidelines together summarize the paradigm shift in the management of descending thoracic aortic pathologies due to endovascular therapies. In Stanford type B aortic dissection, the conservative management of refractory pain and hypertension is associated with significant short-term mortality. Therefore, although a survival advantage has not been demonstrated yet, endovascular intervention of these type B dissections is now more often applied.

Depending on the type of organ protection applied during aortic surgery (deep hypothermic circulatory arrest, selective perfusion of brain and kidneys) coagulopathies and neurologic deficit may occur. Brain damage may be due to ischemia or embolisation and paraplegia may result from crossclamping of the descending aorta. Careful neurologic evaluation before and after surgery are important.

Also, the hypotensive regimen used in the early postoperative period must reduce systolic blood pressure and the force of cardiac contraction. The most common regimens include the use of beta-blockers.

4. Management of complications

Atrial fibrillation

Atrial fibrillation following cardiac surgery is common and occurs in up to 35 % of patients. While the cause of AF is not completely understood, it is associated with an increase in mortality, stroke, and prolonged hospital stay. AF has been discussed in Section I of this chapter.

Low cardiac output syndrome

A low cardiac output state may result from decreased left ventricular preload (hypovolemia, cardiac tamponade, vasoplegia), decreased contractility (myocardial stunning, ischemia or infarction related to poor intraoperative myocardial protection, incomplete myocardial revascularization, anastomotic stenosis, or coronary artery spasm), arrhythmias, increased afterload or diastolic dysfunction.

Transoesophageal echocardiography can help define whether a low cardiac output state is related to left ventricular systolic or diastolic dysfunction, right ventricular dysfunction or cardiac tamponade. The management of low cardiac output has been discussed in Section I of this chapter.

Right ventricular dysfunction produces inadequate filling of the left heart resulting in a low cardiac output state. It may be attributable to poor myocardial protection, prolonged ischemic times, coronary embolism, hypotension, RV pressure overload (pulmonary disease, ARDS, pulmonary embolism) or acute pulmonary hypertension due to vasoactive substances, LV dysfunction, protamine or hypoxia and acidosis.

Right coronary artery disease, right ventricular infarction and pulmonary hypertension associated with mitral/aortic disease predispose to RV failure after cardiac surgery.

PAC's and TEE are very helpful in assessing the status of the RV function. In the absence of LV dysfunction, a high RA/PCWP pressure ratio is suggestive for RV dysfunction. The goals of treatment are to optimize RV preload, maintain systemic perfusion pressure, improve RV contractility, and reduce RV afterload by reducing pulmonary vascular resistance. (27)

Diastolic dysfunction, defined as increased resistance to filling of one or both cardiac chambers, is a common finding after cardiac surgery, especially after cardioplegic arrest. Echocardiography has greatly improved the knowledge of diastole by showing the real-time activities in the heart, as related to filling pressures, shape and relaxation. Failure of the RV can contribute to left-sided diastolic dysfunction by increasing cardiac pressures, which causes decreased relaxation of the myocardium yielding decreased myocardial distensibility. Factors responsible for increased chamber stiffness include fibrosis, cellular disarray, and hypertrophy. Factors responsible for decreased relaxation include asynchrony, abnormal loading, ischemia, abnormal calcium ion flux and hypertrophy. Note that ventricular hypertrophy affects both stiffness and relaxation, increasing the risk of diastolic dysfunction. (73)

Cardiac tamponade

Cardiac tamponade is primarily the result of impaired filling of one or more of the cardiac chambers and leads to low cardiac output. Adrenergic and endocrine mechanisms are activated resulting in tachycardia and vasoconstriction.

The diagnosis of cardiac tamponade depends on a high degree of suspicion. Tamponade after cardiac surgery is different from a medical tamponade due to compressing fluid within an intact pericardium. In the setting of cardiac surgery, the pericardial space is often left open and in open communication with one or both the pleural spaces, and the compressing blood is at least in part clotted and able to cause localized compression of the heart. Serious suspicion for tamponade should rise in patients with deteriorating haemodynamics or gradually increasing requirements for inotropic drugs. The classic signs of elevated CVP or equalization of CVP and PAOP are often absent. Cardiac tamponade is difficult to distinguish from biventricular failure. A useful clue may be the pronounced respiratory variation of blood pressure in association with high filling pressures and low cardiac output. TEE may be helpful in diagnosing cardiac tamponade. Echolucent crescents between the RV wall and the pericardium or the posterior LV wall are discernible. A classic sign is diastolic collapse of the right atrium or RV.

A rule of thumb in the acute management of cardiac tamponade is to keep the patient *Full, Fast and Tight*. Full, the delivery of volume expansion in order to achieve an adequate preload. Fast, using pacing or medication to increase the heart rate to maintain cardiac output since the stroke volume is compromised. Tight, applying vasopressor therapy to increase preload, maintain blood pressure and coronary perfusion pressure.

The definitive treatment of tamponade is surgical exploration with evacuation of hematoma.

Renal insufficiency

No clear definition exists as to what constitutes renal impairment or failure following CPB. Renal failure requiring dialysis is infrequent following CPB, although reductions in creatinine clearance are more frequent. There are several risk factors for postoperative renal

failure, including postoperative low cardiac output, repeat cardiac surgery, valve surgery, age greater than 65, and diabetes.

The primary cause may be prerenal (low pressure, low output, ACE, NSAID's), renal (Acute Kidney Injury) from ischaemic insult or interstitial drug-related nephritis or postrenal.

Management of these patients consists of supportive treatment ensuring adequate cardiac output, perfusion pressure and volume status and of determining the primary cause, and then directing specific treatment as necessary such as discontinuing the offending drug.

If patients do require dialysis, continuous dialysis may be better than intermittent dialysis. (6,74,75,76)

Impediments to weaning and extubation

The most important factors limiting weaning and extubation in the early postoperative period after cardiac surgery include:

1. neurologic dysfunction

- agitation, restlessness and disorientation may occur after discontinuation of the sedative medication. The ethiology of this syndrome is multifactorial and includes patient characteristics, perioperative psychotropic drugs used for anaesthesia, pain relief and sedation, and brain ischaemia and inflammation. Initial management consists of reassurance and orientation of the patient and control of pain with opioids. Resedation for a period or the use of haloperidol may be useful until the patient is oriented and tranquil.
- diaphragmatic paralysis may complicate cardiac surgery, especially after reoperations, due to surgical lesion of the phrenic nerve in fibrotic pericardial tissue. The phrenic nerve can also be injured or transected during dissection of the internal mammary arteries or during mobilization of the heart in redo surgery. Transient diaphragmatic paralysis can also occur secondary to cold injury by the cold cardioplegic solutions to the phrenic nerve. (6)

The diagnosis of diaphragmatic paralysis should be considered whenever a patient fails to wean from mechanical ventilation and can be documented by observing paradoxical movement of the diaphragm during inspiration.

2. unstable haemodynamics

Postoperative cardiac surgical patients with unstable haemodynamics and/or low cardiac output syndromes may not well tolerate the extra work of breathing associated with weaning. Weaning is difficult and may further deteriorate the already compromised myocardium.

Weaning affects cardiac output due to changes in pulmonary vascular resistance. Increased pulmonary vascular resistance (PVR) leads to septal shifts and reduced efficiency of biventricular function. It is therefore better to keep the patient sedated on full ventilator support until the cardiac problem is resolved. (77)

3. fluid overload

Cardiac surgery and CPB result in a systemic inflammatory response syndrome which produces a capillary leak. The duration and severity of this syndrome include factors related to the patient characteristics, severity of the surgical trauma, administration of blood products and CPB management. The use of heparin-coated tubings, membrane oxygenator, centrifugal pumps, steroids and leukocyte filters may reduce the SIRS. The capillary leak syndrome is usually most predominant the first 6 to 8 hours after the termination of CPB.

During this period, fluid resuscitation is necessary to offset the capillary leak syndrome and the vasodilation secondary to medications and rewarming. Crystalloid and colloid infusions are used to maintain intravascular volume, although this usually occurs at the expense of expansion of the interstitial space. After the capillary leak has ceased and haemodynamics are stable, diuretics contribute to a faster recovery from surgery.

Successful early extubation is compromised by fluid overload. Optimal monitoring and adequate measures should therefore be taken in the operation room and in the intensive care to minimize the positive fluid balance while maintaining adequate tissue perfusion. (78)

Central nervous system dysfunction

Neurologic complications are dreaded sequelae of cardiac surgery. Notwithstanding a progressive decrease in cardiac surgical mortality over the past decades, the incidence of postoperative neurological complications remains relatively unchanged.

Focal neurologic complications

Focal neurologic events complicate approximately 2 % of cardiac procedures requiring CPB, but may increase as more patients with advanced age and diffuse vascular disease undergo cardiac surgery. Focal deficits may include hemiparesis or hemiplegia, aphasia, dysarthria, hand incoordination and visual field deficits.

Preoperative risk factors include increasing age (risk of up to 10 % in patients older than age 75), pre-existing cerebrovascular disease, hypertension, peripheral vascular disease, and poor LV function. Intraoperative and postoperative risk factors include: ascending aortic atherosclerosis and calcification, LV mural thrombus, complex surgery and prolonged bypass and haemodynamic instabilities.

The mechanisms for neurologic injury include some combination of cerebral embolism, hypoperfusion, and inflammation; associated vascular disease and cerebral autoregulatory dysfunction make the brain more susceptible to injury. Particulate embolism due to atherosclerotic plaque, blood thrombus embolus, and air and platelet-fibrin debris is the most common cause of stroke. Cerebral hypoperfusion may be the result of systemic hypotension or impaired regional cerebral blood flow. Although cerebral autoregulation should protect the brain during CPB, hypothermia, blood gas regulation, diabetes and pre-existing hypertension may affect the adequacy of cerebral autoregulation. (6,79,80,81)

In the prevention of focal neurologic complications, preoperative evaluation for extracranial carotid disease should be considered in any patient with neurologic symptoms. Symptomatic carotid disease warrants carotid endarterectomy (CE) prior or at the time of cardiac surgery. Asymptomatic carotid disease in the presence of a carotid bruit should be evaluated by non-invasive testing. There is a trend toward performance of combined CABG-CE in these patient groups. (82)

Intraoperative echocardiographic scanning of the ascending aorta to identify atherosclerosis might alter cannulation sites and clamping - and manipulation techniques of this diseased aorta. Techniques to avoid embolic load include the use of membrane oxygenators, arterial filters in the CPB circuit, meticulous débridement and irrigation of valves, removal of LV thrombi and of air after intracardiac procedures.

In general, in patients with hypertension or intracranial vascular disease, blood pressure during CPB should be maintained at a higher level.

In the treatment of embolic stroke, heparin is recommended when there is no evidence of intracranial hemorrhage on the CT scan. Heparin prevents propagation of intracardiac

thrombus and improves cerebral microcirculation, but is of unclear benefit in preventing further atheroembolism from dislodged plaque.

Encephalopathy

Encephalopathy is fairly common after cardiac surgery and is usually manifested by disorientation and confusion, lethargy or agitation, and paranoia and hallucinations.

The etiology of this syndrome is multifactorial. It may be related to brain inflammation, cerebral hypoperfusion or microemboli from the CPB circuit. Other factors include patient characteristics, hypoxia, metabolic disturbances, perioperative psychotropic drugs used for anaesthesia, pain relief and sedation, and drug or alcohol withdrawal.

Initial management consists of reassurance and orientation of the patient and control of pain with opioids. Resedation for a period or the use of haloperidol may be useful until the patient is oriented and tranquil. The encephalopathy has a fluctuating course but is usually transient. (79,81)

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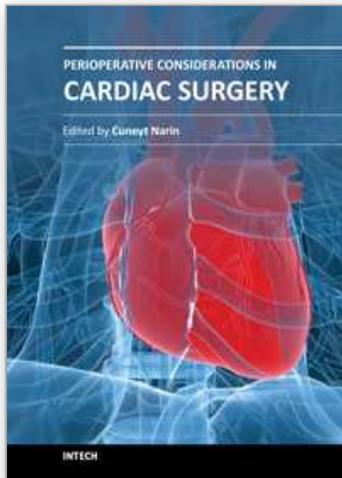
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This book considers mainly the current perioperative care, as well as progresses in new cardiac surgery technologies. Perioperative strategies and new technologies in the field of cardiac surgery will continue to contribute to improvements in postoperative outcomes and enable the cardiac surgical society to optimize surgical procedures. This book should prove to be a useful reference for trainees, senior surgeons and nurses in cardiac surgery, as well as anesthesiologists, perfusionists, and all the related health care workers who are involved in taking care of patients with heart disease which require surgical therapy. I hope these internationally cumulative and diligent efforts will provide patients undergoing cardiac surgery with meticulous perioperative care methods.

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University Campus STeP Ri
Slavka Krautzeka 83/A
51000 Rijeka, Croatia
Phone: +385 (51) 770 447
Fax: +385 (51) 686 166
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InTech China

Unit 405, Office Block, Hotel Equatorial Shanghai
No.65, Yan An Road (West), Shanghai, 200040, China
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元
Phone: +86-21-62489820
Fax: +86-21-62489821

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