Postoperative Considerations of Cardiopulmonary Bypass in Adult Cardiac Surgery

18

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Abstract

Development of the cardiopulmonary bypass (CPB) technology in the second half of the twentieth century was one of the most important medical advances and has been the main part of cardiac surgery as a routine procedure. Providing a completely motionless, bloodless heart is the main goal of CPB. CPB fulfills the role of the heart (and lungs) by preserving the systemic circulation and gas exchange.

Since the first initial machine, evolution from cross-sectional technique till to minimal extracorporeal circulation continues. The practice of cardiac surgery with CPB is safe and effective (but not perfect). CPB is a non-physiologic state and could cause multi-organ dysfunction. Although it simplifies cardiac surgery, CPB by itself induces a systemic inflammatory response syndrome, mostly due to blood contact with artificial surfaces. It activates complement, leukocyte, coagulation and fibrinolytic cascade, upregulation of proinflammatory cytokines, and production of oxygen free radicals and alters nitric oxide metabolism. Nearly all organs can be affected by these inflammatory mediators. In majority of patients these changes are asymptomatic due to adequate physiologic reserve. With improved knowledge in pathophysiology of CPB effects, efforts led to make new extracorporeal technology with less side effects.

The first section describes the structural parts of CPB. The second section discusses about the CPB effects on vital organs.

Keywords

Cardiopulmonary bypass circuit structures · History · Blood circuit · Cardiotomy Membrane oxygenator · Anticoagulation · Cannulation · Cardioplegia · Heat exchanger · Arterial filter · Minimal invasive extracorporeal circulation

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Ultrafiltration · Cardiopulmonary bypass-related complications · Inflammation Hematologic effect · CPB and Kidney · CPB and Lung · CPB and CNS · Off-pump coronary artery bypass

18.1 Cardiopulmonary Bypass Circuit Structure

18.1.1 History

In spite of other fields of surgery, cardiac surgery was suspended for centuries due to lack of knowledge and technology. The lack of its requirements was the cause of this slow and delayed evolution:

1. Inability to preserve systemic circulation and gas exchange independent to heart contraction and lung function, respectively
2. Need to preserve systemic anticoagulation that could be reversed at the end of the operation (insufficient knowledge about blood group types, transfusion, heparin, and protamine)

The introduction of the heart-lung machine in 1953 and development of CPB are among the most important advances in medicine to permit cardiac surgery. It has become a standard part of cardiac operations to make possible the surgical correction of intracardiac diseases, with a good example of competent team working requirement (surgeon, perfusionist and anesthesiologist).

Its development had a prolonged evolution way from concept of extracorporeal circulation (Le Gallois in 1813) to the hopeless Theodor Billroth attitude in 1881 “No surgeon who wished to preserve the respect of his colleagues would ever attempt to suture a wound of the heart” to the first heart surgery using CPB by John Gibbon in 1953, to the present trend of minimal extracorporeal circulation, assist devices, and total artificial hearts.

The CPB machine is an equipment that provides mechanical circulatory function of the heart and lungs. The machine consists of pumps, membrane oxygenator, venous and arterial cannula, tubing, reservoir cardiotomy, and heat exchanger. This section discusses in brief the parts of CPB system for better understanding of issues related to post-cardiac surgery problems (Hessel 2015).

18.1.2 Blood Circuit

In normal condition, blood enters the heart in the right atrium and passes through the right ventricle. The blood leaves the heart into the lungs where carbon dioxide is extracted from the blood and substituted by oxygen. Then the blood is sent back into the left atrium, enters into the left ventricle, and is injected into the aorta where it transfers to the systemic circulation.

The CPB acts nearly the same performance outside of the body. At the beginning of the operation, polyvinyl chloride or silicone tubing is primed by crystalloid solution. Colloid solutions, mannitol, sodium bicarbonate, heparin, and sometimes blood are prime additives. Venous line removes blood by means of gravity or vacuum from right side of the heart and returns it in oxygenated form to the systemic circulation via arterial line by pump. Pump acts like a ventricle. The two most common types are roller and centrifugal pumps.

18.1.2.1 Roller Pump

This pump consists of two rollers placed on the ends of a rotating arm, opposite to each other. The roller rotates and engages the tubing which is then compressed against the pump's housing. It pushes the blood ahead and forward continuous flow is induced (Fig. 18.1). It has occlusive nature and generates shear stress.

The flow rate is determined by the diameter of the tubing and the rotation rate of the rollers (per minute). It is not dependent on preload and afterload changes. The flow rate of 2–2.4 $L/m^2/min$ is sufficient for adequate systemic perfusion, with consideration of patient's temperature. Optimal perfusion targets remain to be in controversy, but it is recommended to preserve mean arterial pressure of 50–70 mmHg, CVP < 5 mmHg, hematocrit more than 20%, and mixed venous saturation > 65% during CPB (Gravlee 2008; Saczkowski et al. 2012).

18.1.2.2 Centrifugal Pump

In plastic housing, a complex of nested cones are coupled magnetically to an electric motor. They rotate rapidly; by creating a pressure gradient between the inlet and outlet of the pump, kinetic energy is transferred to blood resulting in forward blood flow (Fig. 18.2). This pump is used less often than roller pump. The flow rate is dependent on preload and afterload. In spite of roller pump, centrifugal pump is nonocclusive and has less shear stress disturbance.

Some studies have shown that centrifugal pump is associated with less trauma to blood elements and postoperative neurologic complications (Asante-Siaw et al. 2006; Gravlee 2008; Saczkowski et al. 2012).

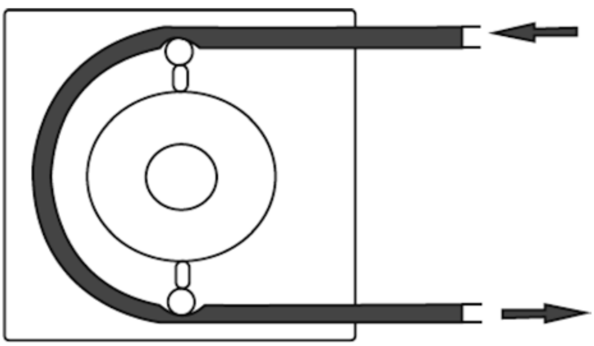

Fig. 18.1 Roller pump (Foroughi 2014)

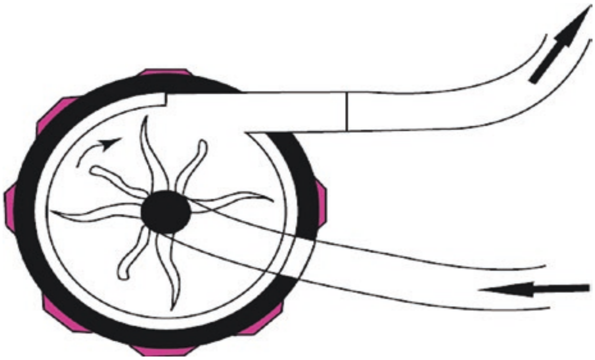

Fig. 18.2 Centrifugal pump

18.1.3 Cardiotomy

A filtered reservoir collects blood drained from the venous circulation. It is for storage, defoaming, and filtration before pumped to oxygenator and arterial circuit. Fluid, blood products, and medication may also be added.

Reservoir design may be open or closed systems. The open system (solid container) has graduated lines that show blood volume in the container. The design is open to atmosphere, allowing blood interface with atmosphere gases. In the closed system, the soft collapsible bag eliminates the air-blood interface. Volume is measured by weight or by changes in radius of the container (Stammers and Trowbridge 2008).

18.1.4 Membrane Oxygenator

Membrane oxygenator is equivalent to lung. A flat sheet of hollow fibers imitates the pulmonary capillary function, by interposing a thin membrane interface between blood and gases, without mixture. Gas flows through the hollow fiber and blood flow is around the fiber. In spite of carbon dioxide, oxygen is not diffusible in plasma well, so the blood is spread very thin to facilitate the transfer of oxygen by increased gradient pressure (Stammers and Trowbridge 2008; Hessel 2015).

18.1.5 Anticoagulation

Adequate anticoagulation is the integral part of CPB to maintain blood fluidity, avoidance of coagulation factor consumption, and thrombosis. Unfractionated heparin is used to achieve adequate systemic anticoagulation, by measuring activated clotting time (ACT) ≥400 s. It remains the absolute choice of anticoagulant drug in cardiac surgery. Heparin is obtained from animal tissues (bovine lung and porcine intestine). It doesn't have anticoagulant properties itself but could potentiate the effects of antithrombin III (AT), a potent endogenous anticoagulant in the body.

Heparin is given in the bolus dose of 2.5–4 mg/kg to aim an ACT of 400–600 s, before aortic and venous cannulation. For adequate anticoagulation state additional heparin is repeated to maintain ACT above 400 s during CPB.

An inadequate response of ACT to high doses of heparin (heparin resistance) may be seen in patients with AT deficiency (such as long preoperative heparin use, liver disease, congenital AT deficiency). This condition is corrected by fresh frozen plasma or recombinant AT administration.

At the end of operation, after CPB weaning heparin is reversed by protamine (isolated from fish sperm) to establish normal hemostasis condition after surgery. There are some ways to determine the protamine dose: protamine titration, fixed dose of protamine (in the ratio of 1–1.5 mg for 100 units of heparin previously administrated), ACT/heparin dose–response curve, and heparin concentration.

Protamine has a mild anticoagulant effect too and inhibits platelet-induced aggregation. Although protamine has a short half-life, care should be taken not to infuse too fast as it can induce systemic hypotension due to systemic vasodilation and pulmonary vasoconstriction.

Heparin rebound, a state of recurrent anticoagulant activity of heparin after adequate protamine administration, contributes to postoperative bleeding. Because not all of heparin is bounded to protamine, some of it binds to other plasma proteins and vascular cells, and reappears in circulation gradually.

Direct thrombin inhibitor is recommended in patients who should not receive heparin such as heparin-induced thrombocytopenic setting (Hessel 2015; Sniecinski and Levy 2015; Rehfeldt and Barbara 2016).

18.1.6 Cannulation

Cannula is made from clear polyvinyl chloride; the oxygenator casing and connections are from polycarbonate. Arterial and venous cannulation sites are influenced by the planned operation. In routine valve and coronary surgeries, the ascending aorta and right atrium are selected. Alternative arterial cannulation sites are the femoral artery, axillary artery, and left ventricular apex. Cannulation site for venous access can be the inferior and superior vena cava, femoral vein, and internal jugular vein (Hessel 2015; Ren et al. 2015).

18.1.7 Cardioplegia

A blood-free and motionless operative field is obtained by potassium-based cardioplegic solution. It causes diastolic electromechanical arrest. In addition to stop electrical and consequently mechanical activation, other main goals are preserving myocardial function and attenuating ischemic-reperfusion injury. Combination of electromechanical arrest and hypothermia reduces oxygen consumption up to 97%. Cardioplegic solution can be categorized according to the type of solution (crystalloid vs. bloody), temperature (cold vs. tepid), infusion type (antegrade into aortic root vs. retrograde through coronary sinus), and infusion interval (continuous vs. intermittent vs. single dose). There is no consensus about optimal choice of cardioplegia (Baikoussis et al. 2015; Gong et al. 2015).

18.1.8 Heat Exchanger

The heat exchanger is used in combination with the oxygenator. This device is typically placed just before the oxygenator, to prevent bubble forming in the blood too. Heat exchanger controls the body temperature through cooling or warming the blood passing the circuit, at the beginning and end of CPB, respectively. In heat exchanger, the blood and water lines are separated by a metallic barrier. As the water temperature is changed, the blood temperature which enters the body circulation and the tissue temperature change. There is a consensus on hypothermic protective effect for organ protection during ischemic period. Hypothermia reduces oxygen consumption and metabolic rate. Even mild hypothermic condition can increase brain tolerance against ischemic injury. However hypothermia is associated with side effects such as induced coagulopathy, free radical generation, metabolic acidosis, and leftward shift of oxygen-hemoglobin dissociation curve. Depending on the type of operation, the patient's temperature may be kept normothermic to less than 20 °C. Monitoring the blood temperature during the operation, and the speed and temperature of rewarming, is mandatory. Rewarming too great or too quickly may make important problems. Hyperthermia during rewarming period is known to increase ischemic damages especially in the brain and kidney (Baikoussis et al. 2015; Hessel 2015).

18.1.9 Arterial Filter

Inclusion of arterial filter in CPB circuit is used to reduce embolic events and improve neurologic outcome after cardiac surgery. Arterial filter with 20–40 μm porous screen holds air bubble, particles of platelet aggregation, and thrombus during CPB (De Somer 2012; DeFoe et al. 2014).

18.1.10 Minimal Invasive Extracorporeal Circulation

Minimal invasive extracorporeal circulation (MiECC) is an alternative option to conventional CPB. It consists of membrane oxygenator, centrifugal pump, short heparin-coated closed circuit, heat exchanger, venous bubble gas detector, and arterial filter. Priming volume is reduced. There is no cardiotomy suction and venous reservoir, and the blood-air interface is limited, while conventional CPB circuit is an open circuit because of blood-air free contact. The shed blood is washed through cell-saving device before return to arterial line.

It seems that these differences lead to attenuation of the adverse effect of conventional CPB: less inflammatory response, reduced hemodilution (less need to blood transfusion), and less changes in hemostasis system. Some studies showed that cardiac surgery with MiECC is associated with less postoperative neurologic deficit, less postoperative bleeding, and improved end-organ protection (Hessel 2015; Anastasiadis et al. 2016; Ganushchak et al. 2016).

18.1.11 Ultrafiltration

At the beginning of CPB, acute hemodilution is an inevitable event, as there is a mixture of patients' blood with crystalloid priming fluid of CPB circuit. Although hemodilution decreases blood viscosity and facilitates tissue perfusion in hypothermic setting, studies have shown that intraoperative hematocrit less than 20% is associated with disturbance in oxygen-carrying capacity, interstitial edema (decreased oncotic pressure) in vital organs, and increased mortality.

Ultrafiltration removes plasma water and low-molecular-weight materials from blood to a filtrate part under hydrostatic pressure through a hollow fiber semipermeable membrane. It reverses excessive hemodilution during pump and need for blood transfusion. It attenuates systemic inflammatory response by removing inflammatory mediators. Several studies have shown improvement in pulmonary compliance and shorter time on mechanical ventilation with ultrafiltration.

There are three types of ultrafiltration. Conventional ultrafiltration is done during CPB and hemoconcentration causes less blood reservoir volume after CPB. If the patient is in stable hemodynamic condition, modified ultrafiltration is performed after CPB weaning to concentrate circulating blood and remaining reservoir volume. Zero-balance ultrafiltration is done after rewarming during CPB. Filtrated volume is replaced by the balanced electrolyte solution. It corrects electrolytes and acid-base balance, and removes inflammatory mediators in the setting of constant blood volume (Wang et al. 2012; Foroughi et al. 2014; Landis et al. 2014).

18.2 Cardiopulmonary Bypass-Related Complications

The practice of cardiac surgery with CPB is safe and effective (but not perfect). CPB is a non-physiologic state and could cause multi-organ dysfunction. In the majority of patients these changes are asymptomatic due to adequate physiologic reserve.

18.2.1 Inflammation

Blood contact with synthetic surface of CPB circuit can promote whole-body systemic inflammatory response that plays an important role in multi-organ failure. Continuous exposure of heparinized blood to artificial surfaces in the perfusion circuit and nonendothelial cells in the wound could activate complement anaphylatoxins, adhesion molecules, proinflammatory cytokines, vasoactive substances, coagulation, and fibrinolytic cascades. In addition, ischemic reperfusion injury (due to aortic clamp, myocardial ischemia, cardioplegic arrest, and declamping), endotoxemia (due to splanchnic hypoperfusion), hypothermia, surgical trauma, bleeding, and blood transfusion may contribute to activation of inflammatory cascade. Accumulation of activated neutrophil is responsible for initiating the release of inflammatory mediators that have negative impact on all organs. The severity of this exaggerated response can range from subclinical state to severe complications (respiratory failure, coagulopathy, arrhythmias, and organ dysfunction).

There are suggested strategies to limit and attenuate inflammatory response, though there is not a consensus regarding their clinical outcome and benefits; these include heparin-coated circuit, steroids, hemofiltration, leukocyte depletion, minimized extracorporeal circulation, aprotinin, complement inhibitors, free radical scavengers, statins, and antioxidants (Landis et al. 2014; Hessel 2015; Zakkar et al. 2015; Ebrahimi et al. 2016).

18.2.2 CPB and Hematologic Effect

All blood elements have been impressed during CPB by different cascades. Close interaction of coagulation and inflammatory system aggregates this effect. Prolonged pump time and influence of biomaterial substances in surface area of CPB have the most effect on the severity of humoral and cellular activation. In coagulation system, reduction in platelet count and function, platelet activation, dilution, and consumption of coagulation factors and increased activity in fibrinolytic field occur. Blood contact with artificial and non-physiologic surfaces of CPB circuit makes intrinsic coagulation pathway activation. Extrinsic coagulation pathway is activated in pericardial blood, which is usually aspirated and returned to pump circulation. In addition to mechanical trauma to red blood cell and increased fragility of cell elements by shear stress force, mixture of patients' blood with priming solution brings to a significant hemodilution in the beginning of CPB.

There are strategies to decrease these responses: heparin-coated CPB circuit, corticosteroids, leukocyte filter, and ultrafiltration (Ranucci 2015; Ebrahimi et al. 2016).

18.2.3 CPB and Kidney

There is no consensus in the definition of acute kidney injury (AKI) after cardiac surgery; its prevalence has been reported between 1 and 30%, according to definitions used for AKI. Postoperative need to renal replacement therapy is a serious complication; it increases patient mortality as much as 50%.

Attention to preventable causes, early detection, and treatment are among the valuable issues for prevention of AKI after cardiac surgery.

After cardiac surgery, only minimal changes of serum creatinine as small as 0.3 mg/dL or 25% increase over baseline could predict adverse prognosis, even it remains in normal range or recovers to baseline. For active management to prevent further progression, there is a narrow period of reversibility shortly after insult. Early detection of AKI is an opportunity to improve clinical outcome. Delay from injury to diagnosis and after established AKI could be an explanation of limited success for AKI treatment.

Kidney function is usually evaluated by serum creatinine and urinary output. But they are poor indicators during acute stage of AKI. Rise in serum creatinine indicates the significant reduction of glomerular filtration rate, but it occurs slowly in 2–3 days after proved injury. It could be changed by nonrenal factors as age, muscle mass, protein intake, fever, trauma, and medication. It is not a specific and sensitive marker during early stages of kidney injury. Multiple studies have suggested that NGAL (neutrophil gelatinase-associated lipocalin) is a sensitive biomarker that shows proximal tubular damage as early as 2 h after the event. It can be used as a reliable biomarker for diagnosis of acute kidney injury in post-cardiac surgery. Other experimental biomarkers of AKI are cystatin C, interleukin18, and kidney injury molecule-1 (KIM-1).

The most important causes of AKI during CPB, which overlap, and consequences of multiple pathways are:

1. Hemodynamic insults (perioperative low cardiac output, hypovolemic state, and vasoconstriction)
2. Ischemic reperfusion injury and release of inflammatory agents
3. Embolic events (both gaseous and particulate)
4. Patient-related predisposing factors (left ventricular dysfunction, emergent surgery, preexisting kidney dysfunction, advanced age, nephrotoxic agents, preoperative anemia, blood transfusion, and need to IABP)

CPB per se is responsible for decreased renal blood flow and GFR due to nonpulsatile flow. Other factors that are related to CPB include duration of CPB time and cross-clamp time, hemolysis, and hemodilution.

1. *Duration of CPB and cross-clamp:* The consequences of prolonged CPB are extension of hypoperfusion time and release of more inflammatory mediators.
2. *Hemolysis:* Hemolysis is the result of prolonged CPB time, cardiotomy suction, overocclusion roller pump, blood exposure to artificial surface, and shear forces.
3. *Hemodilution:* Because of priming solution in CPB tubes, hemodilution is an inevitable event. It is suggested that hemodilution improves regional blood flow (by reduction of blood viscosity) in the setting of hypothermia and hypoperfusion. Although it was thought that improvement in regional blood flow compensates the risk of acute anemia (loss of O_2-carrying capacity of blood), recent studies have expressed that intraoperative hematocrit less than 20% is an independent risk factor for postoperative AKI. Kidneys receive 20% of cardiac

output. Highly metabolic renal medulla has limited reserve and is more sensitive to hemodynamic insults (anemia and hypoperfusion).

Type of operation has an effect on AKI. Among adult cardiac surgeries, coronary artery bypass graft (CABG) has the lowest incidence of AKI, while the combination of CABG and valve surgery is the highest risk factor.

Perioperative hemodynamic optimization is the key to renoprotection. Modifiable factors that reduce the risk of AKI are pulsatile CPB, preserved mean arterial pressure ≥ 60 mmHg (and higher threshold in diabetic and hypertensive patients) during CPB, prevention of excessive hemodilution, shorter duration of CPB, adequate pump flow (to improve O_2 delivery), retrograde autologous priming, decreased prime volume, optimal glucose control, euvolemia, prevention of low cardiac output to preserve adequate renal perfusion pressure, and avoidance of nephrotic agents.

There is no specific therapy to prevent and treat AKI. Several studies have shown that mannitol, dopamine, $NaHCO_3$, N-acetylcysteine, fenoldopam and ultrafiltration have no renal protective effects (Foroughi et al. 2014; Ho et al. 2015; Kramer et al. 2015; Long et al. 2015; Hu et al. 2016)

18.2.4 CPB and Lung

Pulmonary dysfunction and prolonged ventilation after cardiac surgery are well-known problems and important causes of morbidity. It occurs due to combined effects of anesthesia, surgical trauma, and CPB. Its pathophysiology is complex and multifactorial:

1. Inadequate lung perfusion
2. Ischemic-reperfusion injury
3. Change in respiratory mechanics (pleural opening and lack of chest wall integrity)
4. Hyperoxemia
5. Blood product transfusion
6. Blood contact with artificial surfaces of CPB circuit
7. Local and systemic inflammatory reaction

The pulmonary blood flow is stopped during aortic cross-clamp period and the lung perfusion is limited to bronchial arterial system. This ischemic insult can exaggerate the damaging effect of other factors.

Studies have shown that neutrophil accumulation in lung during CPB, its activation, release of chemical mediators, and proteolytic enzymes are in charge of lung injury. In addition to inflammation response, volume overload during CPB plays an important role in accumulation of interstitial fluid in lung too. It was shown that prolonged CPB time, aortic valve surgery, and combined valve/CABG procedures are independent factors of postoperative respiratory failure. The clinical importance of this injury has a range from subclinical atelectasis to ARDS. During CPB there is some degree of lung edema, atelectasis, increased alveolar-arterial oxygen gradient, abnormal gas exchange, increased intrapulmonary shunting, and decreased lung compliance.

There are some therapeutic interventions to prevent or attenuate pulmonary dysfunction, such as off-pump surgery, corticosteroid administration, leukocyte filter, biocompatible circuit, ultrafiltration, continuous ventilation during CPB, maintaining lung perfusion during CPB, pump time reduction, use of MiECC, and modifying ventilator setting from traditional to lung-protective strategies (by lower tidal volume to 6–8 cm^3/kg, FIO_2 below 0.40, higher PEEP to 8–12 cmH_2O, and increased respiratory rate) (Young 2014; Al Jaaly et al. 2015; Lellouche et al. 2015).

18.2.5 CPB and CNS

Neurologic abnormalities including stroke (prolonged or permanent focal neurologic deficit) and postoperative cognitive dysfunction are relatively common problems despite improvement in anesthetic and surgical techniques. Cognitive disorder, a transient decrease in baseline performance, is the most notable complication after adult cardiac surgery (up to 50% of patients during the first week after surgery) by the same mechanisms.

The patient-related factors that predict neurocognitive impairment are:

1. Peripheral vascular disease
2. Diabetes mellitus
3. History of cerebrovascular disease
4. High transfusion requirement
5. Urgent operation
6. Type of surgery (coronary bypass, valvular operation, or aortic arch surgery)
7. Age
8. Postoperative atrial fibrillation

CPB-related mechanisms include:

1. Macro- or microemboli (gas, atheromatous plaque, inorganic and biologic debris from open cardiac procedures)
2. Systemic inflammatory response
3. Perioperative hypoperfusion and low cardiac state
4. Extensive aortic calcification
5. Prolonged CPB time
6. Hemorrhage
7. Cerebral hyperthermia

The majority of strokes are due to embolic event, and watershed component is seen in few patients. The most atheromatous embolic event happens during manipulation of atherosclerotic aorta (cannulation, cross clamp, and declamping). Its consequences are crack or rupture of atherosclerotic plaque leading to debris release, cerebral embolization, and ischemic brain injury. Aortic clamping in the presence of exophytic plaque is contraindicated, and elimination of CPB is recommended in this condition. It is suggested that epiaortic ultrasonography be used to identify atheromatous plaque and help to select the suitable site for aortic cannulation in the diseased ascending aorta. It is more sensitive than digital palpation and transesophageal echocardiography, and gives information about the entire length of the ascending aortic wall. Epiaortic ultrasonography can affect the rate of stroke whenever operative strategies are changed.

Cerebral autoregulatory mechanism is functional during CPB. Blood pressure outside the regulatory range would be associated with adverse outcome.

In patients who have impaired autoregulatory mechanism in cerebral circulation (such as old, diabetic, and hypertensive patients), prolonged period of hypoperfusion is a well-known predisposing factor of ischemic events.

Maintaining higher mean arterial pressure (80–90 mmHg) is recommended in these patients during CPB.

Neurologic monitoring is important during cardiac surgery. Near-infra red spectroscopy can be used to assess cerebral perfusion adequacy during cardiac surgery. Whenever cerebral hypoperfusion is detected, hemodynamic management could be achieved by increased pump flow, increased perfusion pressure, and blood transfusion.

Decrease and increase in temperature of brain (during cooling and rewarming period) happen more quickly than other parts of the body because of the well-perfused state. Rapid rewarming at the end of CPB can lead to cerebral hyperthermia and exacerbated neurologic dysfunction.

During complex aortic arch surgery, neurologic complication is less associated in antegrade cerebral perfusion (through right axillary artery and or right common carotid artreycannulation) than retrograde cerebral perfusion and deep hypothermic circulatory arrest.

Those who experienced stroke during 24 h of CPB had adverse outcome in contrast to late presentation, delayed stroke. The majority of strokes happen after initial normal neurologic recovery of CPB.

Prevention of microemboli is accomplished by the use of membrane oxygenator, prevention of air entering the circuit, and use of a microfilter that will effectively trap microemboli and prevent from reentering the bloodstream (Ganavati et al. 2009; Goto and Maekawa 2014; Ono et al. 2014; Scott et al. 2014; Fraser et al. 2015).

18.2.6 Off-Pump Coronary Artery Bypass

In an attempt to decrease the negative effects of CPB, off-pump coronary artery bypass (OPCAB) on the beating heart was suggested as a solution and less invasive option from the early 1990s.

It is important to balance the advantages/disadvantages of OPCAB vs. CPB. During CPB, activation of physiologic mechanisms (due to blood contact with artificial surfaces of bypass circuit, surgical trauma, and ischemic reperfusion injury) is the cause of systemic inflammatory response, affecting multiple-organ systems. This response is significantly less in OPCAB operations.

Coagulopathy and activation of fibrinolytic cascade attributed largely to CPB do not happen in OPCAB patients. There is less blood loss and transfusion requirement in OPCAB operations. Better preservation of renal function is seen in OPCAB patients by prevention of renal hypopersufion and non-pulsatile flow in CPB state. Even in patients with chronic kidney disease, OPCAB operation is associated with less hospital mortality and need for renal replacement therapy.

There is better myocardial protection and rapid recovery in OPCAB. It offers no aortic cross clamp and cardiac arrest. Using of intracoronary shunt maintains blood flow to distal part of coronary artery during surgery. However, there is no convincing difference with regard to short-term mortality and postoperative MI.

OPCAB patients with preexisting pulmonary disease have better postoperative clinical course. It may be interpreted by more inflammatory reactions in the lung and fluid retention observed with CPB operations. The protective effect of OPCAB in end-organ function and safer outcome profile was shown in reoperative surgeries.

There is no trend for reduction in neurologic complication (embolic stroke and cognitive dysfunction) in OPCAB patients. It may be related to side-biting aortic clamp used for proximal anastomoses and embolization risk from atherosclerotic plaque. To reduce postoperative neurologic events in the presence of diseased ascending aorta, OPCAB with total arterial revascularization (no-touch aortic technique) was described to preclude aortic manipulation.

The concerns about OPCAB are incomplete revascularization, less grafts per patients, suboptimal anastomoses and graft patency, hemodynamic instability during operation, and increased coronary re-intervention. It is a challenging technique, and requires more time and experience with a long learning curve that may compromise long-term patient's outcome.

It is not recommended for small, intramyocardial coronary arteries and moderate aortic or mitral regurgitation. When coronary surgery is indicated in acute coronary syndrome, on-pump beating heart surgery is a more effective option. Decompression of ventricles by CPB reduces myocardial oxygen consumption, while OPCAB surgery prevents global myocardial ischemia by avoiding aortic cross clamp.

Although CABG using CPB is the standard surgical treatment for ischemic heart disease, studies have shown that in experienced hands and in high-volume OPCAB centers the outcome parameters are comparable, effective, and safe as on-pump surgery. It may be acceptable and beneficial in selected moderate- to high-risk patients (Euroscore ≥5) (Afilalo et al. 2012; Møller et al. 2012; Sepehripour et al. 2014; Puskas et al. 2015; Kowalewski et al. 2016).

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