14 Physiopathology of Intraoperative Visceral Ischemia and Anesthesiological Management of Supravisceral Aortic Clamping

Fabrizio Monaco, Barucco Gaia, Mattioli Cristina, and De Luca Monica

14.1 Introduction

In patients undergoing aortic surgery, visceral ischemia is the leading cause of visceral dysfunction and injury [1, 2]. In fact, acute kidney injury (AKI), spinal cord ischemia (SPCI), and bowel ischemia are catastrophic complications which may occur during surgical procedures involving the supra- and juxta-renal aorta cross-clamping [2, 3]. Because of the fact that the higher the extent of the surgical aortic repair, the greater the risk of organ dysfunction, thoracoabdominal aortic aneurysm open repair (TAAAr) is more prone to developing visceral ischemia than infrarenal abdominal aortic aneurysm [1, 3]. The application of the aortic clamp, decreasing the blood supply to the organs, is associated with extensive physiological changes which may affect patient outcomes. Therefore, it is important for surgeons and anesthesiologists to understand the pathophysiologic changes occurring during aortic cross-clamping in order to mitigate the deleterious effect of ischemia-reperfusion injuries.

14.2 Physiopathology of Intraoperative Visceral Ischemia

Vascular surgery, in which the aorta is clamped proximally to the celiac artery, is one of the few surgical procedures producing ischemia of the liver, bowel, kidneys, spinal cord, and inferior limbs contemporarily. Aortic cross-clamping produces rapid hemodynamic changes and induces ischemic insults. Following the aortic clamp removal, the reperfusion itself may lead to a sudden drop in blood pressure and cellular damage. Therefore, aortic surgery shows double physiological phenomena named ischemia/reperfusion (I/R) injury which is the major determinant of an extensive systemic inflammatory response and the trigger for postoperative multi-organ dysfunction (MODS) [4, 5]. In particular, after aortic clamping in the district distal to the aortic clamp, visceral tissues suffer a sudden decrease of the blood flow with an acute hypoxic insult, shift from an aerobic to an anaerobic metabolism, production of lactate, and development of acidosis [6]. At the same

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time, cellular membranes increase their permeability, leading to cellular swelling [4, 7, 8]. Following aortic clamp removal, the reperfusion of tissue is responsible for additional injuries on the top of ischemia [4–8]. In some instances, the reperfusion damage may exceed the original ischemic injury; in fact, the restoration of the blood flow account for the activation of several inflammatory pathways and biochemical changes [4]. In the I/R syndrome, polymorphonuclear neutrophils, oxygen radicals (ROS), nitric oxide (NO), complement system, and various cytokines play a pivotal role, showing their effects in both re-perfused tissues and distal organs [9, 10]. The re-oxygenation enhances ROS production, which are associated with lipid peroxidation, complement activation, platelet aggregation, white cell activation, suppression of adenosine triphosphate synthesis, and inactivation of metabolic enzymes [10, 11]. Moreover, the ROS leading to the depletion of antioxidant reserves, disruption of cellular and mitochondrial membrane, derangement of intracellular electrolytes boost the phenomenon of apoptosis [12, 13].

A growing body of literature suggests that polymorphonuclear neutrophils play a central role in the pathophysiology of I/R [10]. In fact, the upregulation of adhesion molecules, chemoattractants, chemokines, and integrins due to I/R stimulates the migration of polymorphonuclear neutrophils from the postcapillary venules to the area of inflammation. Polymorphonuclear neutrophils, then, may disrupt the contiguous tissues by the secretion of proteolytic enzymes, production of free radicals, and microcirculation disarrangement [11]. Of note, the oxidative stress and ROS formation reach their peak during the ischemic attack (15–60 min of clamping), while PMN infiltration is at a maximum during reperfusion [10–12].

Notably, the NO has both cytotoxic and cytoprotective effects. In fact, it is an oxygen-free radical scavenger, maintains normal vascular permeability, inhibits the proliferation of smooth muscle, reduces PMN adherence, and decreases platelet aggregation [13]. However the release of large amounts of NO may account for tissue injury, bacterial translocation, mucosal apoptosis, and pulmonary injury [14–16].

The complement, acting together with ROS, NOS, and PNM, increases vascular permeability and tissue edema [17–20]. It elicits a cascade of pro-inflammatory events with release of high concentration of TNF-α and interleukin (IL)-1 [18, 21]. Finally, the occurrence of edema in the interstitium of the injured organ further decreases the oxygen diffusion gradient from the microcirculation to the cells [9].

14.3 Postoperative Effects of Ischemia/Reperfusion Injury

Interestingly, the I/R injury has an effect on both the organs directly affected by the ischemia and the organs not involved in the ischemic insult, by a systemic release of inflammatory mediators [22]. Therefore, it is not surprising that during aortic surgery, along with the bowel and kidney, even the heart, lungs, and spinal cord may suffer I/R damage [4]. When the damage is extensive, multi-organ failure may occur [22, 23]. Each organ and apparatus has a specific sensitivity to I/R insult and deserves specific considerations.

14.3.1 Bowel

Since labile cells are settled at the tips of the villi and supplied by the end of the distribution of a central arteriole, they are much more vulnerable to the ischemia when compared to cells located within the crypts [24]. The various acute-phase proteins, hydrogen peroxide, hormones, and cytokines produced by intestinal mucosa during I/R injury have deleterious effects onto the intestinal microvasculature and may lead to bowel infarction, short-bowel syndrome, systemic inflammatory response syndrome, acute respiratory distress syndrome, and MOF [25]. Moreover, the impairment of the mucosal barrier allows the release in the systemic circulation of the endotoxin which induces the systemic

activation of PMN, complement, and clotting pathways and further increase of the mucosal permeability [26].

14.3.2 Kidney

Acute renal failure (AKI) during aortic surgery is multifactorial and it may occur as a result of I/R damage, hemodynamic changes, bleeding, acute heart failure, and cytotoxic agents [27–36]. The level at which the aortic clamp is applied affects the renal perfusion [37]. In fact, it decreases by 80% in the event of the suprarenal aortic cross-clamping, while it decreases by 45% when the aortic clamp is infrarenal [38]. To counteract the decrease of the renal blood flow, an increase of the renal vascular resistance is mediated by the hormone angiotensin II which redistributes the blood flow away from renal medulla and cortex, significantly decreasing renal perfusion [28, 39]. This effect persists after aortic clamp removal, despite a normal mean perfusion pressure [40]. Similarly, the glomerular filtration rate and renal blood flow may remain impaired for a long period of time after the surgery [31]. Besides this, ROS, complement, IL-1, IL 6, and IL8 released by the damaged mesangial cells increase the local inflammation worsening the renal function [41].

14.3.3 Heart

Aortic surgery is associated with the highest risk of myocardial infarction and cardiovascular complications compared to other noncardiac-related surgeries [42, 43]. The reasons for that include the increase of afterload and preload associated with the aortic cross-clamp, massive bleeding with consequent volume shift, and inflammatory response following I/R injury of abdominal organs [22]. Several authors have proposed that IL-2, IL-1β, IL-6, IFN-γ, and TNF-α may affect the cardiac function [44]. In I/R injury of the heart, the NO is probably involved in the decrease of ventricular compliance [4]. The activation of the NO synthesis leads to higher NO concentration which significantly affects the cardiac adrenergic and cholinergic stimulation [4]. A ventricle with low compliance is "difficult to fill," and it is associated with lower cardiac output and impairment of the coronary blood flow due to a decrease in the aortocoronary pressure gradient. Moreover, the ROS released from the injured myocytes and endothelial cells promotes membrane damage, endothelial injury, and vessel permeability [45, 46]. The treatment of the diastolic dysfunction is challenging: in fact, the administration of exogenous inotropes/vasopressors may further decrease a microcirculation already impaired by the endothelial swelling, exacerbating the ischemic damage [46]. In addition, activation of the coagulation cascade, formation of microthrombi, platelet aggregation stimulated by the use of vasopressors, and accumulation of reactive neutrophils act together impairing the microcirculation and decreasing the myocardial perfusion [47].

14.3.4 Lungs

The respiratory function after aortic surgery is commonly impaired. While in the vast majority of cases, damage is moderate with nonsignificant clinical manifestations; in several circumstances, it may be part of MOF [22]. Since in a physiological state the pulmonary vasculature is a neutrophil reserve, the respiratory system is at the highest risk of developing an inflammatory response during aortic surgery [11]. In fact, it is particularly sensitive to the circulating cytokines released from several organs during the postoperative period [48]. The I/R damage releases "per se" cytokines, which activate the pulmonary endothelium, stimulate the leucocytes migration into the interstitial and alveolar space, and promote inflammation [22]. Similarly, the anaphylatoxins C3a and C5a play a role in increasing the pulmonary vascular tone, favoring capillary leakage, and activating the mast cells which release histamine [49, 50]. The most severe clinical respiratory manifestation of this "vicious circle" is the acute respiratory distress syndrome which is associated with refractory hypoxia and death in a high percentage of patients [51].

14.3.5 Spinal Cord

Spinal cord ischemia is one of the most dreadful complications in aortic surgery [1–3]. The abrupt interruption of the blood flow to the spinal cord leads to ischemic injury [52–56]. Even when the thoracic aorta is not involved (abdominal aorta aneurysm open repair), a profound shock may impair the medulla perfusion pressure causing SCI [53]. The pathogenesis includes oxygen-free radical-induced lipid peroxidation, intracellular calcium overload, leukocyte activation, inflammatory response, and neuronal apoptosis. All these factors acting together cause the disruption of the blood-spinal cord barrier, which in turn exacerbates the spinal cord edema, increases the leukocyte infiltration, and amplifies inflammation and oxidative stress [57].

14.4 Anesthesiological Management of Supravisceral Aortic Clamping

Given the complexity of the physiopathology of the visceral ischemia in procedures involving supravisceral aortic clamping, the aim of the anesthesiological management is to avoid the hemodynamic fluctuations which may induce irreversible damage to organs and apparatus.

14.5 Hemodynamic Response to Cross-Clamping

Generally speaking, the higher the location of the aortic clamp, the greater the increase of the afterload against which the heart has to work [58]. The immediate effect of aortic clamping is a rapid increase of blood pressure due to an increase in systemic vascular resistance (SVR) [58, 59] (Fig. 14.1). Reasons for that are higher impedance to aortic flow, increased venous return (preload) from the viscera, and release of catecholamines and angiotensin [6, 58–62]. In particular, in the event of supraceliac aortic clamping, a rapid decrease of venous capacity in the splanchnic district is associated with a blood volume shift proximal to the clamp site [63]. When the aortic clamp is infra-celiac, the increase of preload is directly related to the splanchnic venous tone: with a lower preload when the venous tone is low and higher preload if the venous tone is higher [38, 63]. The consequence of the increase of afterload and preload is the improvement in contractility [59]. The increase of the left ventricular end-diastolic pressure, following the increase in arterial blood pressure, leads to a transitory subendocardial ischemia which triggers the augmentation of the coronary blood flow toward the endocardia (Anrep effects) [64]. The effect is an increase of contractility and then of cardiac output [65]. On the contrary, patients with low coronary reserve, due to coronary artery disease, fail to respond to the subendocardial ischemia with an increase of the coronary flow leading to subendocardial ischemia and low cardiac output. In this case, vasodilators may improve the Anrep effect, increasing coronary blood flow and reducing, at the same time, pre- and afterload [66]. When a distal perfusion technique is not provided and an aortic clamp is present, the perfusion of the vital tissues distal to the aortic clamp is provided by collateral vessels and depends upon the proximal perfusion pressure [62]. Thus, hypotension should be avoided as much as possible [62, 66, 67].

When the aortic clamp is released, the rapid decrease in vascular resistance produces hypotension. Reperfusion of previous ischemic tissues which are vasodilated for the effect of hypercapnia, acidosis, and high concentration of adenosine and lactate [58] leads to central hypovolemia. Furthermore, the washout into the systemic circulation of myocardial depressant metabolites from ischemic area is associated with further vasodilation and decrease in cardiac output [59] (Fig. 14.2).

Following aortic clamp removal, a transient increase in CO_2 is commonly observed due to both CO_2 washout from the ischemic tissues into the systemic circulation and increased CO_2

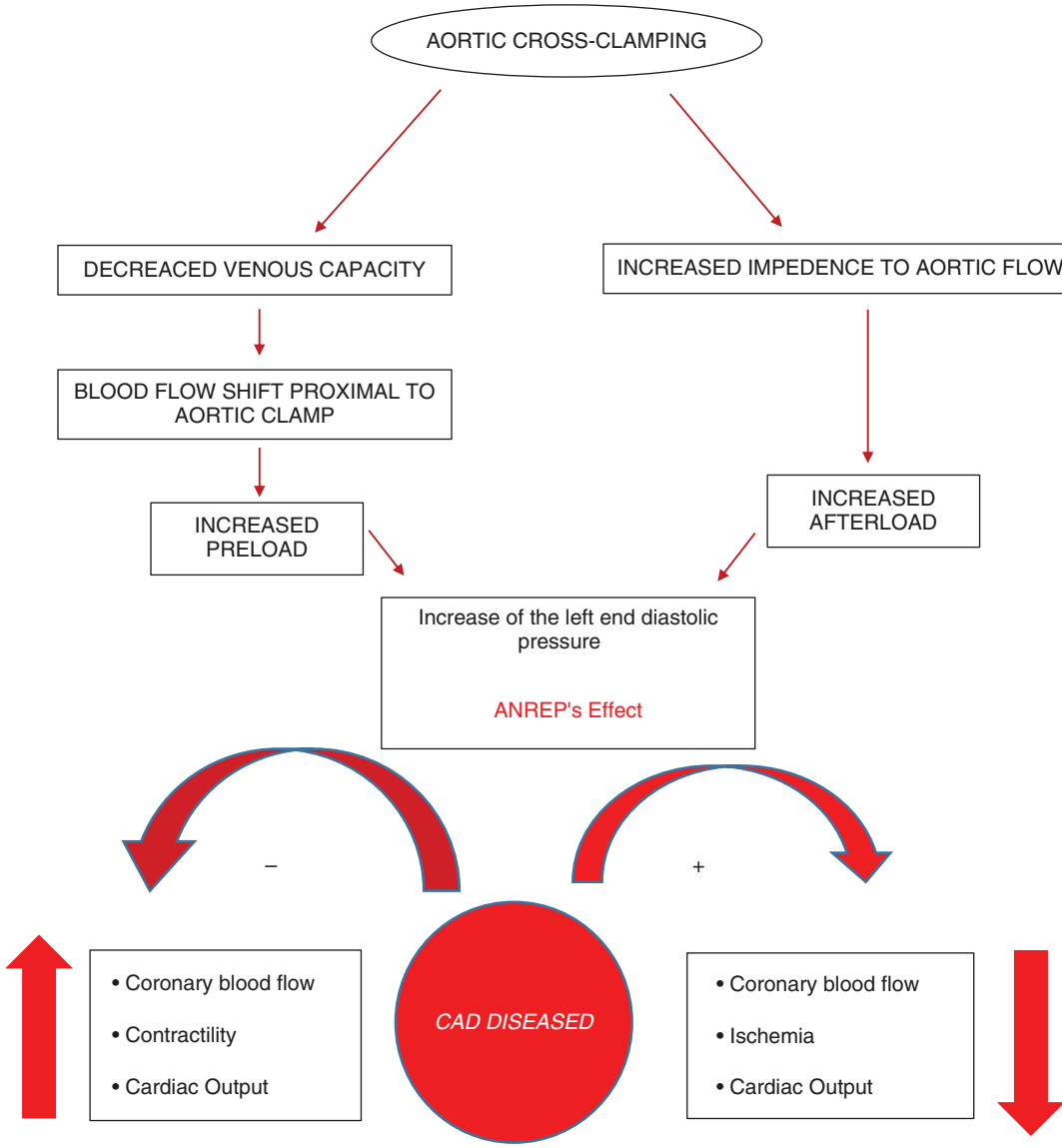

Fig. 14.1 Hemodynamic response to aortic cross-clamping

production secondary to increased oxygen consumption of the re-perfused tissues [58, 59]. Carbon dioxide causes further vasodilation [68].

Hypotension after aortic cross-clamp release can be prevented and treated with volume loading, infusion of vasoactive medications, prompt treatment of metabolic abnormalities, and gradual release of aortic cross-clamp. In this dynamic setting, the anesthesiologist has to continuously assess the patient's global hemodynamic status, integrating cardiac function, intravascular volumes (estimated from transesophageal echocardiography, filling pressures, or both), blood loss, and the total amount of fluid administered [59, 60]. Moreover, adequate tissue perfusion is based on the availability of oxygen delivered. In situations where the blood flow is suboptimal, an arterial oxygen saturation as high as possible and a hemoglobin concentration above 10 g/dL are mandatory [69].

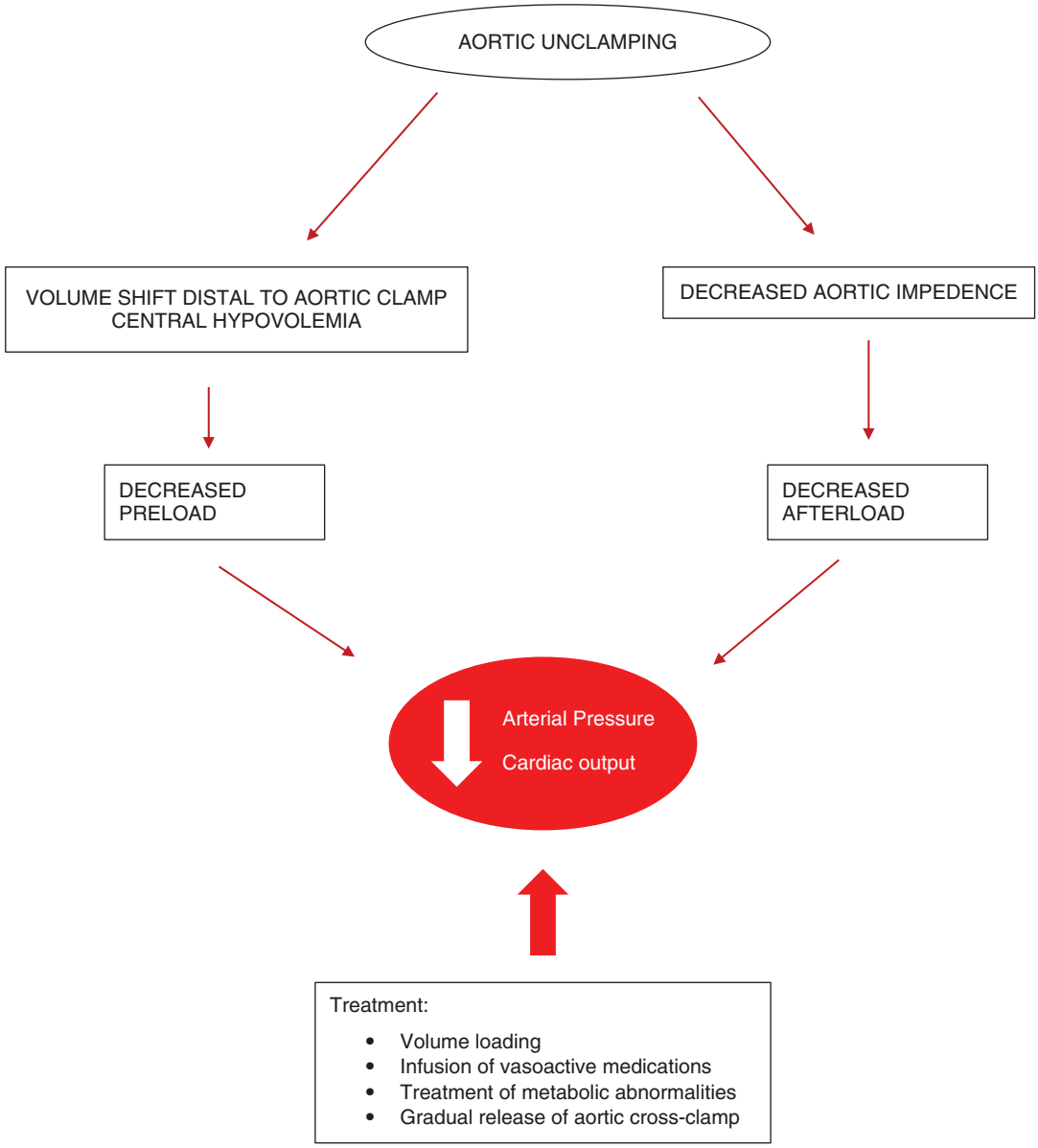

Fig. 14.2 Hemodynamic response to aortic unclamping

14.6 The Distal Perfusion Technique as a Strategy to Prevent Visceral Ischemia

Cross-clamping of the descending thoracic aorta produces visceral, spinal cord, kidney, bowel, and limb ischemia and is also challenging for the heart due to an abrupt increase of the pre- and afterload. In order to prevent and mitigate the consequences of visceral ischemia, several pharmacological and mechanical strategies have been proposed [70]. To date, distal organ perfusion is universally recognized as the best technique to limit ischemic injury in the organs distal to the clamp site, to support the heart, and to control proximal hypertension during thoracoabdominal aneurysm open repair [71, 72]. Distal perfusion may be performed by partial cardiopulmonary bypass (CPBP), left bypass (LBP), or left heart bypass (LHBP). Among these, LHBP is

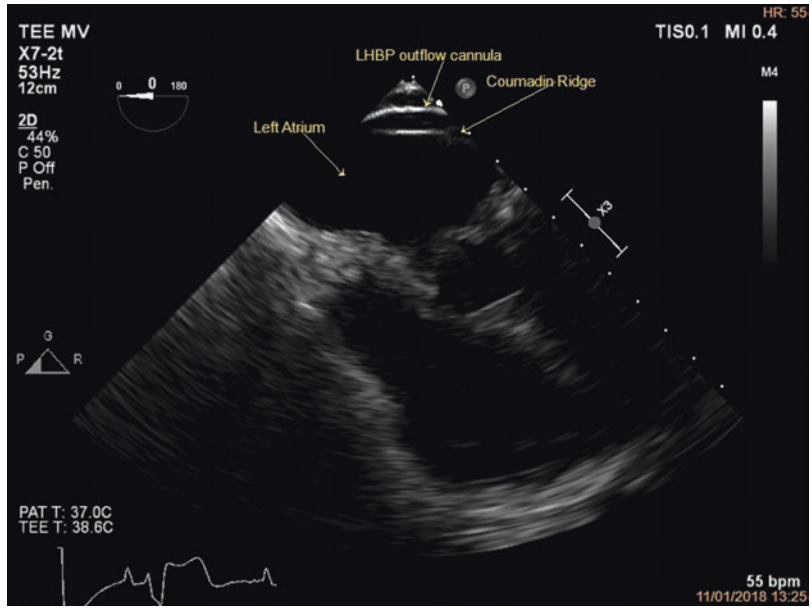

Fig. 14.3 Transesophageal echocardiography. Off-axis view of the outflow cannula joining the left atrium by the inferior left pulmonary vein

associated with a low risk of bleeding due to a mild heparinization [73]. Briefly, the basic circuit for LHBP is composed by an inflow cannula, a centrifugal pump, and an outflow cannula. In the left heart bypass (LHBP), the inflow cannula is placed in the pulmonary vein or left atrial appendage (Fig. 14.3), while the outflow cannula is positioned in the distal aorta or the femoral artery [74]. During the surgical anastomosis of the visceral vessels, perfusion of the abdominal organs is usually guaranteed by a selective catheterization of individual arteries [75, 76]. A flow of 200–300 mL/min of normothermic oxygenated blood for each catheter maintains a visceral perfusion pressure around 70 mmHg. The kidneys are perfused by cold crystalloids or cold Custodiol. Several studies have reported that a cold Ringer lactate solution is superior to normothermic oxygenated blood in terms of prevention of renal dysfunction during selective renal artery perfusion and supraceliac aortic cross-clamp [77–79]. Moreover, Tshomba et al. observed that in patients undergoing TAAA open repair, a selective renal perfusion with histidine-tryptophan-ketoglutarate solution (Custodiol; Dr. Franz-Kohler Chemie GmbH, Bensheim, Germany) significantly decreases the incidence of postoperative renal failure when compared with cold Ringer lactate [80].

There is no agreement on which is an adequate distal perfusion pressure when the LHBP is used. Some authors, for instance, suggest that a flow as high as 40 mL/kg/min is adequate to guarantee an optimal distal perfusion. On the contrary, others report that a flow ranging between 1.5 and 3 L/min with a mean femoral artery pressure of 70 mmHg can be considered sufficient for organ perfusion [81]. As practical rule, during aortic cross-clamping, a distal aortic pressure above 70 mmHg and a proximal perfusion pressure above 90 mmHg can be considered optimal for organ and spinal cord perfusion. Further indices of optimal organs perfusion are renal output above 1 mL/kg/h without diuretics and no lactate production [61].

14.7 Strategy to Prevent Postoperative Organ Dysfunction in Visceral Ischemia

Although the distal perfusion technique mitigates the effects of visceral ischemia, postoperative complications due to organ hypoperfusion remain relatively high [1–4]. Several strategies have shown to be effective in mitigating organ ischemia following aortic clamp.

14.7.1 Renal Protection

Postoperative acute kidney injury (AKI) after vascular surgery is a major cause of morbidity and mortality [33, 82]. The etiology of renal failure in the setting of vascular surgery is multifactorial [83]. Ischemic injury (clamp time), nephrotoxic agents (antibiotics, anesthetic agent, contrast media, diuretics, myoglobin), and pre-existing renal failure are major factors related to the development of acute renal failure after aortic surgery [31, 34, 83–85]. In light of this, the avoidance of nephrotoxic insult, prevention of renal hypoperfusion by adequate cardiac output, and MAP have recently been reported as the only measures effective in decreasing the incidence of AKI [86–88]. On the contrary, the use of drugs such as dopamine and fenoldopam is not able to prevent perioperative renal dysfunction [89–91]. Since methylprednisolone, at a dosage of 30 mg/kg, may be a scavenger of free radicals with immunomodulatory proprieties, some authors have postulated that its administration may prevent renal ischemia/reperfusion injury [92–95]. Unfortunately, the results are elusive [96]. Even mannitol (0.5 g/kg), which theoretically has a favorable profile in terms of renal failure prevention due to the induction of osmotic diuresis, the prevention of tubular obstruction, the decrease of epithelial and endothelial cell swelling, the action of free radical scavenger, and the stimulation of the synthesis of intrarenal prostaglandin with a renal vasodilation effect, has shown to be ineffective in preventing AKI [86, 97]. Moreover side effects such as volume depletion and an increased medullary consumption of O_2 are very well known and may have a detrimental impact on renal function [98].

14.7.2 Spinal Cord Protection

Paraplegia caused by ischemic spinal cord injury is a devastating potential complication of aortic surgery [1, 99, 100]. Patients with SCI have poorer long-term survival compared to those who do not [1]. The position of aortic cross-clamping may affect the spinal cord perfusion, with the highest risk during extent II repair (7–10%) and the lowest risk in extent IV (1%) [1, 54, 55]. During surgery, the maintenance of a spinal cord perfusion pressure (SCPP) above 80 mmHG may prevent the development of paraplegia [101, 102]. Notably, the SCPP is the difference between the MAP and the cerebrospinal fluid (CSF) pressure. Interestingly, CSF pressure is influenced by the central venous pressure (CVP). After surgical occlusion of the spinal arteries, the perfusion of the spinal cord depends on the collateral network fed by hypogastric arteries, internal thoracic arteries, and branches from the subclavian arteries. The SCPP is a balance between the driving pressure affected by MAP, cardiac output, and blood volume and outflow pressure which depends on CSF and venous pressure. An increased CVP is associated with higher pressure in the extensive vertebral venous and impairment of spinal cord outflow. For the reasons mentioned above, the use of LHB, inotropes, and vasopressor, acting on the MAP and CVP, prevents paraplegia. The use of CSF drainage has been shown to decrease the risk of paraplegia reducing the CSFP [103]. In a large randomized controlled clinical trial the CSF drainage strategy in patients undergoing TAAA open surgery has shown an 80% decrease of postoperative paraplegia rate [104]. Recently Tshomba et al. observed that the use of the LiquoGuard automated device (Möller Medical GmbH, Fulda, Germany) during TAAA open repair is safe and effective in maintaining the desired CSF pressure values with a significant reduction in complication rates when compared with a standard catheter connected to a dripping chamber [105].

Somatosensory-evoked potentials are used to monitor the integrity of the posterior (sensory) spinal cord, and motor-evoked potentials (MEPs) are used to monitor dysfunction of the anterior (motor) spinal cord, detecting the spinal cord ischemia during the surgery [106–108]. Hypothermia has protective effects on the spinal cord and central nervous system by reducing both metabolic rate and oxygen requirement [109, 110].

14.7.3 Heart

Aortic surgery is a deeming procedure for the heart due to aortic clamp and large volume shift. Since the driving pressure for organs and apparatus depends on the native cardiac performance, it is crucial to optimize preload, afterload, and contractility. In light of this, TEE is an invaluable tool allowing for quick diagnosis and guiding the use of inotropes/vasopressors [111, 112]. Markers of right ventricle dysfunction are CVP over 12 mmHg, tricuspid annular plane systolic excursion below 16 mm, tissue Doppler index below 10 cm/s, right mid-cavity diameter above 42 mm, and longitudinal diameter longer than 9.2 mm (Figs. 14.4–14.6). With pressure or volume overload, the septum becomes flat and the LV assumes a D shape at the end of the systole or diastole, respectively (Videos 14.1 and 14.2). The RV is particularly sensitive to the increase of the pulmonary vascular resistance secondary to hypercapnia, hypoxia, acidosis, protamine and blood transfusion, and reduction of the pulmonary vascular bed, commonly observed during single-lung ventilation**.** Therefore, the first-line treatment of the right ventricular dysfunction is gas exchange optimization with high FiO2, moderate hyperventilation, and alkalization (pH > 7.40). Central venous pressure above 15 mmHg affecting SCPP should be treated with aggressive diuretic therapy. For moderate RV dysfunction, dobutamine is the drug of choice, while epinephrine is indicated in the event of poor RV contractility with hypotension associated (or not) to left ventricular failure. When RV failure coexists with low systemic vascular resistance, norepinephrine is effective in maintaining coronary perfusion pressure.

Markers of left ventricle dysfunction are wedge pressure above 15 mmHg, ejection fraction below 50%, and left ventricular outflow tract velocity time integral below 20 cm/s with good RV function. In transgastric midpapillary short-axis view, the TEE allow to identify whether the hypotension depends on low preload (papillary kissing) or poor contractility (increase end-diastolic diameter) (Videos 14.3 and 14.4). Poor contractility is managed with epinephrine or dobutamine. Mean arterial pressure is a critical factor, and it is not unusual to observe a significantly altered ST segment and regional wall motion abnormalities with low MAP that become almost normal with adequate systemic perfusion pressure. When the increased afterload is associated with a systolic ventricular dysfunction, "inodilators" are suggested,

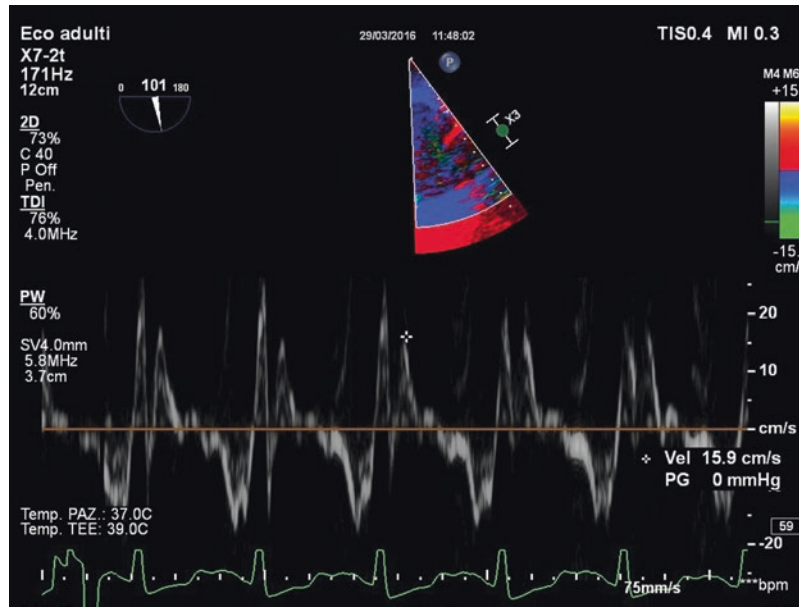

Fig. 14.4 Tissue Doppler index of the right ventricle. A value above 10 cm/s is normal under general anesthesia

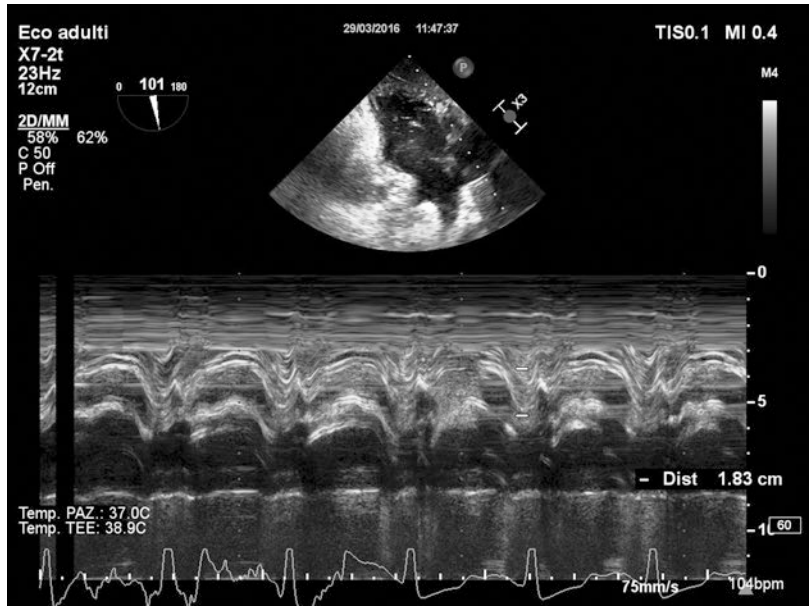

Fig. 14.5 Tricuspid annular plane systolic excursion. A value above 16 mm is normal under general anesthesia

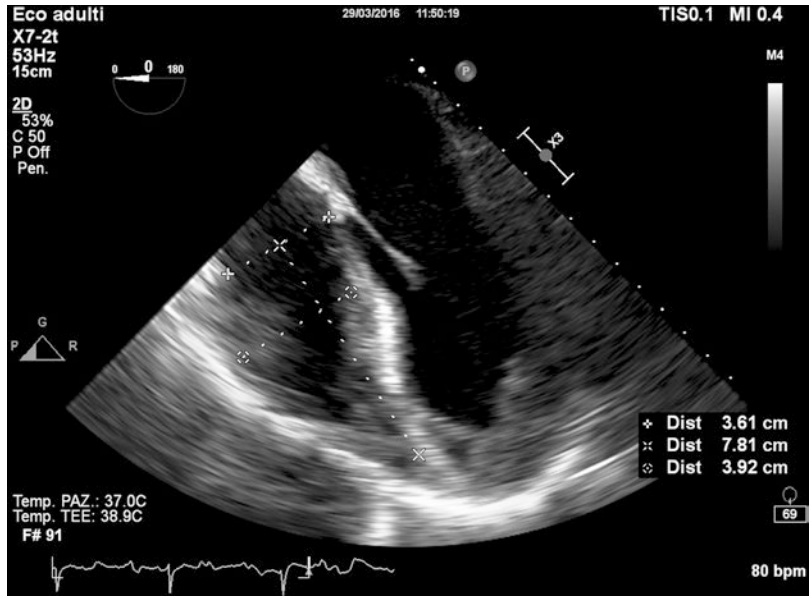

Fig. 14.6 Transesophageal echocardiography. In the midesophageal four-chamber view, the diameters of the right ventricle are best assessed

while with an almost normal systolic contraction brief, acting vasodilator drugs (nitroglycerine) are recommended. As proximal aortic hypotension compromises the perfusion to collateral-dependent tissue beds distal to the aortic clamp, the use of short-term drugs are recommended. Short-acting beta-blockers are preferred agents when a hypertensive episode is associated with tachycardia.

14.7.4 Abdominal Viscera

Surgical occlusion by aortic clamp of the celiac axis and superior and inferior mesenteric leads to hypoxia of the abdominal viscera. The clamping time plays a pivotal role in the development of visceral ischemia, and the adoption of the selective perfusions of the visceral arteries with warm blood mitigates this phenomenon [78]. Even a

transitory period of bowel ischemia, affecting the mucosal integrity, promotes the translocation of intestinal bacteria into the circulation promoting systemic infection and sepsis.

With the aortic clamp release, the washout of both the endotoxins produced by intestinal bacteria and the cardio-depressant metabolites from the ischemic area contributes to the systemic vasodilation and hemodynamic instability observed after visceral reperfusion [16]. Therefore, acid-base alterations occurring throughout the surgery should be promptly treated even by the administration of sodium bicarbonate.

In addition, visceral ischemia may be associated with systemic coagulopathy due to increased intestinal permeability, bacterial translocation, hepatic ischemia, and primary fibrinolysis. Therefore, the use of antifibrinolytic such as tranexamic acid or aminocaproic acid is strongly suggested [18].

14.7.5 Lungs

Postoperative pulmonary complications are common after aortic surgery [1, 3]. In addition to surgical trauma, diaphragm incision, and need of the one-lung ventilation (OLA), lung manipulation, blood transfusion, and fluid overload are very well-recognized risk factors. Furthermore, preoperative risk factors, such as COPD and history of smoking, significantly increase the chance of postoperative lung dysfunction. With this in mind, the adoption of a "protective ventilation" with low tidal volume, higher levels of positive end-expiratory pressure, and low plateau pressure is able to decrease the occurrence of acute lung injury [113]. However during the OLV, the priority is to guarantee oxygen saturation above 90%. A drop in oxygen saturation may lead to a significant decrease in the delivery oxygen with relative tissue hypoxia. A parsimoniously administration of blood products contributes to decreasing the risk of transfusion-related acute lung injury and transfusion-related immune modulation. To avoid large-volume transfusion, a ROTEM-guided protocol may be useful. Further studies are needed to confirm this data.

14.8 Conclusion

Visceral ischemia during supraceliac aortic cross-clamp is a multifactorial complex syndrome associated with an increased risk of developing severe postoperative organ dysfunction, requiring therefore an extensive and detailed anesthesiological and surgical workup. Often, visceral ischemia is devious, and only a prompt treatment may avoid severe postoperative complications. For all these reasons, adequate preoperative assessment and risk stratification, a skillful anesthetic technique, a meticulous intraoperative monitoring, and an appropriate postoperative course are all necessary measures to guarantee an uneventful procedure and avoid potentially fatal complications.

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