



Thoracoabdominal Aortic Aneurysm Repair: What Should the Anaesthetist Know?

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Cite this article as: Goel N, Jain D, Savlania A, Bansal A. Thoracoabdominal Aortic Aneurysm Repair: What Should the Anaesthetist Know? *Turk J Anaesthesiol Reanim* 2019; 47(1): 1-11

Abstract

The surgical repair of descending thoracic aortic (DTA) and thoracoabdominal aortic aneurysms (TAAAs) presents one of the greatest challenges for anaesthesiologists. The challenge comes from the fine balance of complex medical issues in the setting of altered physiology that occurs during the perioperative period. Patients presenting for TAAA repair usually have multiple pre-existing comorbid conditions involving their cardiac, pulmonary and renal systems; and aneurysm repair poses a direct and immediate threat to these systems in addition to that to the gastrointestinal and neurologic systems. Operative mortality in thoracoabdominal aortic surgery is quite high to the extent of 5%-12% with a 5-year survival rate of 70%-79% for DTA aneurysm and 59% for thoracoabdominal aortic aneurysm surgeries. Complex haemodynamic changes associated with the clamping and declamping of aorta requires thorough knowledge and expertise for the management of TAAA. We present a brief review on the anaesthetic management and possible complications that anaesthetists should be aware of during TAAA repair.

Keywords: One-lung ventilation, renal protection, spinal cord protection, thoracoabdominal aortic aneurysm

Introduction

Anaesthetic management of thoracoabdominal aortic aneurysm (TAAA) repair poses one of the most difficult challenges for anaesthesiologists. The surgical procedure impacts every major organ system, and patients typically possess multiple comorbidities. The management of these patients requires an understanding of the disease process, a surgical technique and multiple adjunctive therapies for organ protection. The morbidity and mortality associated with TAAA surgery are multifactorial. Neurologic and renal complications are significant for the most extensive forms of aneurysms. Mortality has improved over time as a consequence of increased surgical experience, adoption of a protocolised strategy for repair and secular improvements in anaesthetic and intensive care treatment. Here a brief update on perioperative considerations in TAAA surgery is presented.

Definition

Thoracoabdominal aortic aneurysm is defined as a dilation of the aorta to a diameter at least 50% greater than the expected normal aortic diameter at the diaphragmatic hiatus, with varying degrees of thoracic and abdominal extensions (1). Aneurysms can be further described according to their location, morphology and aetiology. Its location is commonly used to classify aortic aneurysms for both clinical significance and surgical approach. Descending thoracic aneurysms may involve one or more aortic segments (aortic root, ascending aorta, arch or descending aorta), whereas TAAAs will extend through the diaphragm (2). The morphology of an aneurysm is either fusiform or saccular. Fusiform aneurysms are sausage-like in shape and are symmetrically dilated throughout the full circumference of the aorta. Saccular aneurysms involve an outpouching of a portion of the circumference of the aortic wall. Aneurysms may also be morphologically classified as either true or false (pseudoaneurysm). True aneurysms involve all layers of the aortic wall. A pseudoaneurysm is a contained rupture that extends between the two outer layers of

the aortic wall. The integrity of the vasculature is maintained by surrounding tissues and the chronic inflammatory reaction to extravasated blood (3).

Aetiology and Pathophysiology

Atherosclerosis is the most common cause of aortic aneurysms. Dissection and degenerative changes of the muscle or tunica media of aorta layer of the aortic wall are frequent causative factors of aneurysms of thoracic aorta. The strength and elasticity come from the media layer of the aortic wall comprising vascular smooth muscle cells around a matrix of elastin and collagen proteins (4). It is the destruction of these structural components that causes aneurysm development (5). Ascending aortic aneurysms were more common due to medial degeneration, where the wall composition showed an increased proportion of elastin fibres, whereas descending aortic aneurysms were primarily associated with arteriosclerotic lesions, in which the aortic wall had a higher proportion of collagen (6). Medial degeneration can be due to various connective tissue and genetic disorders (2). Aneurysmal dilation in the young population, specifically in the ascending aorta and aortic root, is classically associated with Marfan syndrome (2, 7). Other disorders associated with aortic aneurysms include syphilis, vascular arteritis and Turner’s syndrome (7). Although the majority of TAAAs are due to degenerative diseases, approximately 20% of TAAAs presenting for repair are due to chronic aortic dissection (8). Aortic dissection classically occurs when a false lumen is created by a tear through the intima, separating it from the media. If left untreated, then this chronic dissection will weaken the wall strength and cause aneurysmal growth. The most consistent risk factor associated with the progression of aortic dissection to aneurysm formation is chronic hypertension, specifically elevated diastolic and mean arterial pressures (MAPs) (8-10). Iatrogenic causes of TAAA include a previous heart surgery requiring aortic cannulation and/or aortic cross-clamping.

Incidence

The incidence of thoracic aortic aneurysms is estimated to vary between 5.9 and 16.3 compared with 350 cases for abdominal aortic aneurysms per 100,000 person-years (11-13).

Type I	From distal to the left subclavian artery to above the renal arteries
Type II	From distal to the left subclavian artery to below the renal arteries
Type III	From the midthoracic descending aorta to below the renal arteries
Type IV	From the diaphragm to below the renal arteries
Type V	From the sixth intercostal space to above the renal arteries

Of thoracic aortic aneurysms, the ascending aorta is affected in 50% of cases, the aortic arch in 10% and the descending thoracic aorta (DTA) in 40% (14). Rigberg et al. (15) have reported a mean age of 70 years for elective TAAA repairs and 72.1 years for ruptured TAAA repairs, with men comprising 62% and 68% of patients, respectively. The 5-yr survival rates of patients with thoracic and abdominal aortic aneurysms not surgically treated are approximately 20% and 16%-19%, respectively (14). Various authors have identified the female sex as an independent risk factor for aortic rupture (10, 12, 16, 17, 19). Additional risk factors that have been associated with aneurysmal dilatation and increased risk for rupture include a history of hypertension (75%), coronary artery disease (30%), diabetes mellitus, age >70 years, tobacco use, pulmonary disease (30%) and renal dysfunction (15%) (18-20). A history of pulmonary disease, specifically COPD, is associated with a 3.6-fold greater rate of aneurysm rupture (20). Predisposing factors for rupture include aneurysm diameter of >5 cm, hypertension, smoking, chronic obstructive pulmonary disease, pain, chronic aortic dissection and age. Although complications after thoracic aortic surgery remain a threat, the overall operative mortality is 5%-12% with a 5-yr survival rate of 70%-79% for DTA aneurysms and 59% for TAAA surgery (21). Major postoperative complications include renal failure (5%-13%) and spinal cord ischaemic injury (4%-30%, depending on the extent of repair and use of adjuncts) (22, 23).

Classification

Thoracoabdominal aortic aneurysms can occur anywhere from the left subclavian artery to the bifurcation of iliac arteries. In 1986, Crawford described the first TAAA classification scheme based on the anatomic extent of the aneurysm (21). Type I involves most DTA from the origin of the left subclavian to the suprarenal abdominal aorta. Type II is the most extensive, extending from the subclavian to the aorto-iliac bifurcation. Type III involves the distal thoracic aorta to the aortoiliac bifurcation. Type I and II TAAA repairs have the highest risk for morbidity and mortality because of their complex multiorgan system involvement. Type IV TAAAs are limited to the abdominal aorta below the diaphragm. Safi’s group modified this scheme by adding Type V, which extends from the distal thoracic aorta, including the celiac and superior mesenteric origins but not the renal arteries (Table 1, Figure 1) (24).

Indications of Repair

Thoracoabdominal aortic aneurysm repair is associated with a significant risk of postoperative mortality and morbidity and with a high risk of postoperative paraplegia, renal failure, stroke, pulmonary complications and myocardial infarction (10, 20, 25). These risks should be weighed against the likelihood of spontaneous rupture to determine the optimal time for repair. All symptomatic aortic aneurysms, regardless of

their size or anatomic extent, should be surgically addressed. These symptoms most commonly present as pain or pressure. In case of DTA, aneurysmal pain is classic, often described as intrascapular or chest pain radiating to the back, with a ‘ripping’ or ‘tearing’ quality. Aneurysmal growth is exponential, with a rate of 0.12 cm per year for aneurysms with diameter >5.2 cm (26). The critical size for rupture or dissection is 6.0 cm for ascending aortic aneurysms and 7.0 cm for descending aortic aneurysms (26). Elefteriades et al. (26) have extensively reported on the natural history and rupture risk of the thoracic aorta stratified by diameter and provided the following guidelines for the repair of DTA:

- (I) Rupture
- (II) Acute dissection resulting in malperfusion or other life threatening complications
- (III) Symptomatic states
 - (i) Pain consistent with rupture and unexplained by other causes
 - (ii) Compression of adjacent organs
- (IV) Documented enlargement of ≥ 1 cm/year or substantial growth approaching absolute size criteria
- (V) Absolute size of >6.5 or >6.0 cm in patients with connective tissue disorders.

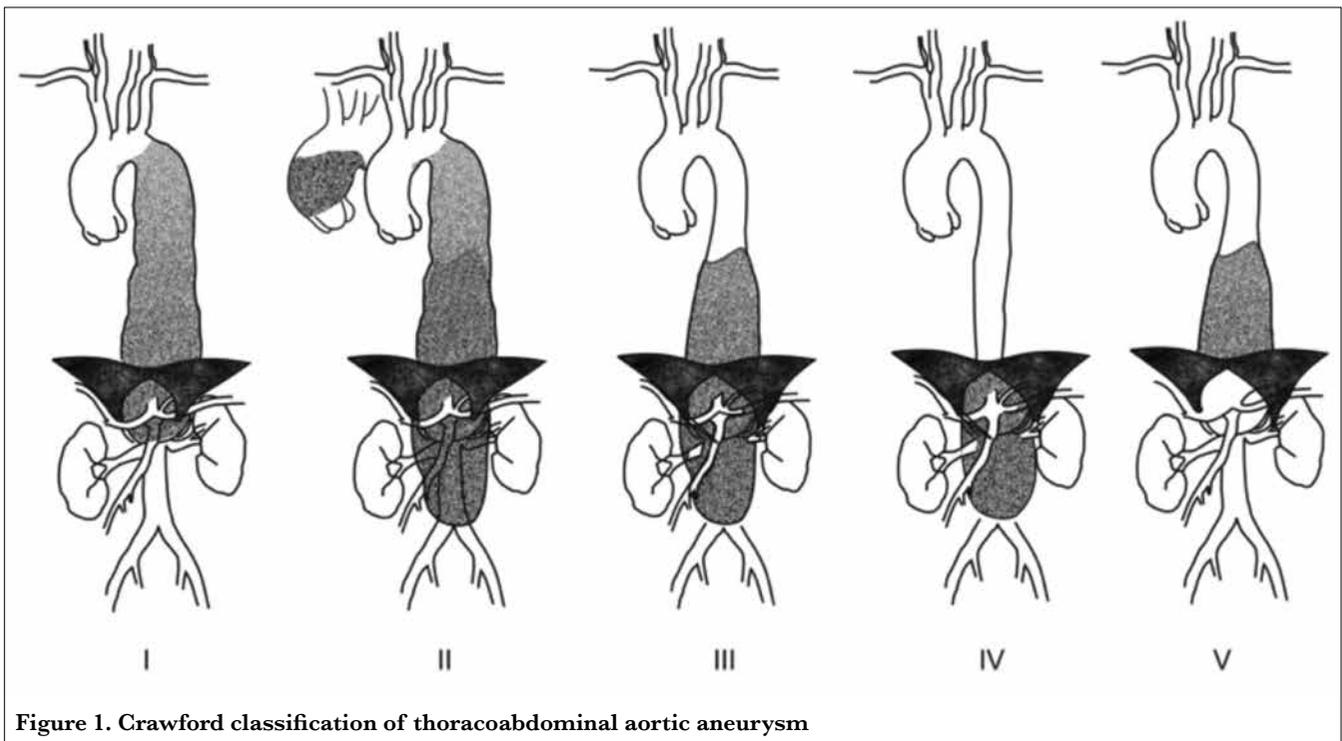


Figure 1. Crawford classification of thoracoabdominal aortic aneurysm

Optimisation of pulmonary functions	<ul style="list-style-type: none"> a) Smoking cessation for 8 weeks prior to surgery b) Get room air arterial blood gases, pulmonary function tests c) Continue with bronchodilators, inhaled steroids, antibiotics d) Chest physiotherapy, spirometry to be continued
Optimisation of cardiac status	<ul style="list-style-type: none"> a) Continue with beta blockers, calcium channel blockers and ACE inhibitors b) Evaluate with coronary angiography, TTE c) Interventional therapy (PTCA, CABG)
Optimisation of renal function	Maintain adequate hydration
Optimisation of haemostasis	<ul style="list-style-type: none"> a) Monitor INR, PTT, platelet function b) Prepare adequate amount of blood product anticipating 5-6 L of blood loss
<p>TAAA: thoracoabdominal aortic aneurysm; ACE: angiotensin-converting enzyme; PTCA: percutaneous transluminal coronary angioplasty; CABG: coronary artery bypass grafting; INR: international normalized ratio; PTT: prothrombin time index</p>	

Table 3. Advantages and disadvantages of distal perfusion techniques	
Advantages	Disadvantages
Control of proximal hypertension Reduction of visceral and renal ischaemia Prevention of acidosis and declamping shock Ability to rapidly warm patients Access for rapid volume infusion Potential reduction in DIC incidence Supplementary oxygenation with extracorporeal oxygenators Reduced incidence of paraplegia	Possible atrial, ventricular, aortic or femoral arterial injury Air emboli/embolic stroke Interference with operative field Increased operative time Potential for excess haemorrhage with anticoagulation Bleeding from the cannulation sites Difficult proximal exposure for atrial/proximal aortic cannulation Shunt dislodgement
DIC: disseminated intravascular coagulation	

Table 4. Intraoperative management of TAAA repair	
Maintain haemodynamics	a) Ensure wide bore intravenous lines b) Rapid infusor set, blood warming available c) Both radial and femoral artery pressure monitoring done d) Prepare both vasodilator and vasopressors e) Distal perfusion techniques preferred
One-lung ventilation	a) Double-lumen tube or endobronchial blocker b) Confirmation of position of DLT in supine and in lateral decubitus position of the patient
Spinal cord protection	a) Permissive hypothermia b) CSF drainage (CSF pressure <10 mmHg) c) Reimplantation of intercostal arteries
Renal protection	a) Hypothermia b) Distal aortic perfusion c) Cold crystalloid perfusate
Coagulopathy	Anticipatory treatment of coagulation disorders (FFP, cryoprecipitate, inhibitors of fibrinolysis)
TAAA: thoracoabdominal aortic aneurysm; CSF: cerebrospinal fluid; FFP: fresh frozen plasma	

Table 5. Postoperative management of TAAA repair	
Haemodynamic	a) BP control to ensure SCPP b) Monitor for signs of myocardial dysfunction
Pulmonary	a) Airway oedema (consider leaving DLT in place) b) Pulmonary oedema, TRALI, ARDS possible
Renal	a) Monitor for signs of renal failure b) Haemodialysis possible
Neurological	a) Assess and monitor the neurologic status b) Maintain SCPP (via BP and CSF drainage)
Coagulation	Ensure normal coagulation and platelet count/function before CSF catheter removal
TAAA: thoracoabdominal aortic aneurysm; BP: blood pressure; SCPP: spinal cord perfusion pressure; DLT: double-lumen tube; TRALI: transfusion-related acute lung injury; ARDS: acute respiratory distress syndrome; CSF: cerebrospinal fluid	

Diagnostic Imaging

Imaging is fundamental for the diagnosis, management and continued monitoring of TAAA. The gold standard radio-

logic modality is computed tomographic angiography (CTA) with 3-dimensional reconstructions (27). High-resolution CTA images are easily and rapidly attainable, making this

modality superior to transoesophageal echocardiography (TEE) and magnetic resonance angiography (27, 28). Key TAAA characteristics to evaluate on CTA imaging include morphology, maximum aortic diameter, extension and relationship to adjacent structures, presence of mural thrombus, calcifications and the presence of leaks or dissections (28).

Preoperative Preparation

Nearly all patients presenting for aortic surgery have coexisting medical conditions that can significantly affect anaesthetic management. These include diseases of the cardiovascular, pulmonary, renal and central nervous systems, among others. The goal of preoperative evaluation is to detect coexisting diseases, assess the risk of adverse outcomes, optimise the patient's medical status and devise an anaesthetic technique that minimises complications. Preoperative sedation should be based on the patient's clinical condition and concurrent medical diseases. Some form of anxiolysis should be administered because hypertension and tachycardia may increase the risk of aneurysm leakage or rupture or induce myocardial ischaemia in patients with concurrent coronary artery disease. A preoperative summary is presented in Table 2.

Pulmonary dysfunction following TAAA repair remain the leading cause of postoperative morbidity and mortality occurring in 20%-40% of patients (29). The presence of preoperative history of chronic obstructive pulmonary disease, cerebrovascular accident, congestive heart failure, renal failure (creatinine >1.5 mg dL⁻¹ or blood urea nitrogen >24 mg dL⁻¹), elderly age group (age >64 years), immunocompromised state and obesity (body mass index >30 kgm⁻²) are risk factors for postoperative pulmonary complications. Preoperative pulmonary assessments provide a baseline measurement to aid in risk stratification and to identify patients whose preoperative respiratory status may be improved. All patients should have preoperative chest radiography, arterial blood gas and pulmonary function tests, if possible, to evaluate respiratory reserve because one-lung ventilation (OLV) is necessary for surgical exposure. In patients with significant pulmonary disease, bronchodilator therapy should be optimised before surgery. Smoking cessation may decrease postoperative complications. Cessation for a minimum period of 8 weeks before surgery is recommended (30). Poor pulmonary function is associated with prolonged mechanical ventilation and increased mortality (31). The lower limit for FEV₁ below which significant activity restriction and carbon dioxide retention occurs is around 0.6-0.7 L. In patients with carbon dioxide retention, the 5-year survival rate is $<10\%$. In patients undergoing abdominal aneurysm surgery, the transient postoperative decrease in FEV₁ is at least 0.6 L. Because of the extensive involvement of the abdomen and thorax, the division of the diaphragm and other physiological derangements in TAAA surgery, the decrease in FEV₁ will be greater. Svenson

suggests an FEV₁ of <1.2 L, a carbon dioxide level of >45 mmHg, an FEV_{25%} of <0.5 Ls⁻¹ and a PaO₂ of <55 mmHg as preoperative indicators of high risk of postoperative respiratory complications (32).

Cardiac complications, including perioperative myocardial infarction and cardiac death, are estimated to occur in 10%-15% of TAAA repairs (33, 34). It is prudent to have an extensive cardiac work-up in these patients because coronary artery disease is quite common in most patients (35). In addition to the ACC/AHA guidelines for cardiac evaluation, electrocardiography, stress ECHO and cardiac catheterisation are routinely utilised (35). In accordance with the ACC/AHA guidelines, patients with severe myocardial dysfunction and with evidence of severe coronary artery disease amenable to percutaneous angioplasty should undergo revascularisation before elective TAAA repair (36). Medical risk reduction strategies, including preoperative beta-blocker and statin therapy, should also be preoperatively continued as instructed by the AHA guidelines (35).

The presence of preoperative renal insufficiency, advanced age and aneurysmal involvement of renal arteries is associated with an increased risk of postoperative renal dysfunction (36). The preoperative assessment of serum creatinine, blood urea nitrogen and creatinine clearance measurement is essential.

Liver dysfunction is associated with bleeding problems and has increased mortality. TAAA repair is associated with major blood losses, often exceeding the patient's intravascular volume. The finding of intraoperative and postoperative deficiencies of clotting factors, along with thrombin generation and activation of the thrombolytic system, is reflective of massive blood losses, visceral ischaemia and massive transfusions. An aggressive strategy of transfusion of blood products is critical for the prevention of clinically significant coagulopathy during surgery. An initial crossmatch of 12-16 units of packed red blood cells, 10 units of fresh frozen plasma and 10 units of platelets should be ready. Low-dose aprotinin, tranexamic acid or epsilon-aminocaproic acid, intraoperative blood salvaging and acute normovolaemic haemodilution can also be tried as an adjunct to reduce blood loss and blood product use.

Intraoperative Period

The cardiac anaesthesiologist should have a thorough understanding of the surgical procedure during the technically challenging, multistep repair. The knowledge of haemodynamic fluctuations during aortic clamping and de-clamping and the control of these fluctuations are absolutely necessary before the induction of anaesthesia. The primary aim of open TAAA repair is to minimise the ischaemic time to vital organ system. Current management techniques include

distal aortic perfusion with left heart bypass (LHB), sequential aortic clamping, permissive systemic hypothermia and neuroprotective strategies including motor evoked potential monitoring, cerebrospinal fluid (CSF) drainage and intercostal or lumbar artery reimplantation (37). We will discuss the anaesthetic management of elective TAAA repair with notation of specific adjunctive surgical protocols that are routinely performed at our institute.

Induction of Anaesthesia

After shifting the patient to the operation theatre, the standard American Society of Anaesthesiologists monitoring comprising 5-lead ECG, pulse oximetry and blood pressure cuff is placed. Foley catheterisation is also prudent to assess volume status and to provide an early indication of renal malperfusion. In repairs of aortic coarctation and descending thoracic aneurysms, simultaneous femoral and right radial arterial monitoring may help in performing partial cardiopulmonary bypass for perfusion distal to a cross-clamp and in determining the adequacy of surgical repair (by documenting the pressure gradient across the repair). The right radial artery is the preferred site because it should reflect the pressure in the carotid arteries, whereas the left radial artery may be rendered useless if the left subclavian artery is affected by the aortic cross-clamp (38). It is necessary to consult with the surgeon before cannulating the femoral vessels because these vessels may be used for extracorporeal perfusion or for the placement of an intra-aortic balloon pump during the surgical procedure. General anaesthesia is induced with etomidate, fentanyl and rocuronium. Isoflurane or sevoflurane is then continued for the maintenance of anaesthesia.

Haemodynamics: Monitoring and Management

Extensive haemodynamic changes should be anticipated during aortic surgery. Securing a large-bore intravenous access is imperative, such as 1 or more large-bore (7F-9F) venous (introducer) cannulae. Besides securing invasive arterial monitoring, two introducers are placed in the same central vein after induction along with pulmonary artery catheter to obtain sufficient central venous access. Rapid infusion systems with blood-warming capacity are necessary. Blood salvaging techniques for auto transfusion should be used. The easiest technique for blood salvaging is the use of a centrifugal device that scavenges and washes erythrocytes (38). Auto transfusion of unwashed, filtered whole shed blood offers significant advantages over allogeneic blood replacement with respect to the preservation of platelets and coagulation factors as well as decreasing allogeneic blood exposure (39).

Cannulation of a central vein is routinely performed for ascending aortic and aortic arch surgery to measure the filling pressures in the heart (using a central venous or pulmonary artery catheter) and to provide a central route for drug ad-

ministration and for the rapid infusion of fluids. Central venous access can be accomplished using a large-bore cannula (introducer) in the right or left internal jugular veins. TEE can also be a valuable adjunct for monitoring fluid shifts and ventricular function.

One-Lung Ventilation

Surgical access is best achieved via a left thoracotomy. A left-sided double-lumen endotracheal tube (DLT) should be inserted for lung separation. The right lung is isolated and protected in the event of an intrapulmonary bleed or rupture of the aneurysm into the left main bronchus. In some situations, large aneurysms may distort the bronchial anatomy and make the placement of left-sided DLT impossible. In these settings, a right-sided DLT or a bronchial blocker may be placed. The use of OLV provides better surgical exposure, reduces the need for pulmonary retraction, may decrease the severity of iatrogenic pulmonary contusion and protects the right lung from contamination by blood and secretions (38). This decreases the cross-clamp time, thereby reducing the likelihood of spinal cord, renal and visceral ischaemic injury and also reduces left lung retraction and trauma. At the end of surgery, provided there is no facial swelling, airway oedema or evidence of bleeding, DLT should be changed to a single-lumen tube. Leaving the DLT for 12-24 hrs is beneficial only if there is any possibility of re-exploration.

Transoesophageal Echocardiography

Transoesophageal echocardiography may be used for the intraoperative monitoring of myocardial function and fluid status and is often more sensitive than pulmonary artery catheters or electrocardiogram for the detection of myocardial ischaemia via the visualisation of ventricular wall motion abnormalities (40). TEE may also help to guide the placement of LHB cannulae.

Haemodynamic Changes Associated with Aortic Clamping and Unclamping

During aortic cross-clamping, profound haemodynamic changes occur. The most consistent haemodynamic response to acute aortic occlusion is an abrupt increase in afterload with a resultant increase in proximal aortic pressure. This is accompanied with an increase in preload, which is attributed to volume redistribution from the veins distal to the site of aortic occlusion. These increases in afterload, preload and possibly contractility resulting from aortic occlusion result in increases in myocardial oxygen demand and possibly myocardial ischaemia (41). Generally, the more proximal the aortic clamp, the more extreme the augmentation of blood pressure. Nicardipine, nitroglycerine, nitroprusside and/or vasodilating properties of inhaled volatile agents may be used to help offset the increase in afterload seen with aortic cross-clamping. Esmolol infusion may be safely used to control proximal hyper-

tension. Esmolol during aortic occlusion may reduce cardiac output and increase ventricular filling pressures. With prolonged duration of the aortic cross-clamp, systemic vascular resistance increases and cardiac output decreases (41). However, intraoperative hypotension can also occur due to hypovolaemia, myocardial depression and excessive partial bypass pump flow. Reperfusion is also associated with hypotension, which may be caused by central hypovolaemia attributable to blood pooling in reperfused tissues, hypoxia-mediated vasodilation and the accumulation of vasoactive or myocardial-depressant metabolites such as lactate. Treatment should be directed towards the rapid correction of hypovolaemia, acidosis, hypocalcaemia and the judicious administration of vasoactive drugs such as noradrenaline and phenylephrine to maintain MAP at 80-100 mmHg.

Distal Perfusion Techniques

There is a two-fold rationale behind distal perfusion techniques—the attenuation of proximal hypertension and adequate perfusion of tissues below the aortic cross-clamp. By providing blood flow to the lower body, bypass and shunt strategies diminish the incidence of renal failure and possible paraplegia (42, 43) and prevent metabolic acidosis and dramatic hypotension that are characteristic of the declamping syndrome that accompanies simple aortic cross-clamping. The advantages and disadvantages of distal perfusion techniques are presented in Table 3.

The primary haemodynamic derangement typically seen with aortic cross-clamp release is decreased systemic vascular resistance and arterial blood pressure (41). The hypotension seen with unclamping is multifactorial (41). Blood volume redistribution to the lower extremities causes central hypovolaemia. Hypoperfusion of tissues distal to the aortic cross-clamp causes the accumulation of vasoactive and myocardial-depressant metabolites (*e.g.* lactate), which are released upon cross-clamp removal. Hypotension after aortic cross-clamp release can be combated by volume loading, the infusion of vasoactive medications, the prompt treatment of metabolic abnormalities, minimised aortic cross-clamp time and the gradual release of aortic cross-clamp (41). Sodium bicarbonate may be given to counteract this metabolic acidosis, but should be cautiously given in the setting of a mixed respiratory acidosis. Generally, it is advisable to maintain a higher blood pressure than normal after cross-clamp release to ensure adequate renal and spinal cord perfusion (44).

CSF Drainage

Cerebrospinal fluid drainage substantially reduces the risk of paraplegia in patients undergoing TAAA repairs, particularly extents I, II and possibly III (45). At our institution, a CSF drain is placed following the induction of general anaesthesia. The patient is placed in the right lateral decubitus position

with both hips and knees flexed, and the drain is inserted under strict and completely sterile conditions. A 14-G needle is inserted in the subarachnoid space below the level of L2-L3 to reduce the risk of spinal cord injury. Once CSF is obtained, the spinal drain is inserted to 8-10 cm within the intrathecal space. The drain is then secured with clear dressings and taped to the right side of the back to avoid surgical incision. Care is taken to ensure that it is kept sterile and protected during repositioning. Free withdrawal of CSF is confirmed after placement, taping and repositioning.

Positioning

For surgical incision and aortic exposure, the patient is placed in the cork-screw right lateral decubitus position with the upper body at 60° and the hips at 30° from the horizontal. This allows for femoral access in the groin area if required (46). An axillary roll is placed for brachial plexus protection, and additional padding is placed at sites of potential pressure points. The left arm is superiorly extended in a freestyle swimming stroke position (46). It is secured in this position using arm boards or slings.

Surgical Incision

The administration of antibiotics before or within 1 h of surgical incision is essential to prevent postoperative infection. Heparin is also administered to maintain moderate anticoagulation at a dose of 1 mg kg⁻¹. Heparin is primarily used to prevent stasis-induced thrombosis of small vessels during the clamp period of TAAA repair. For extent I, II and III repairs, a curvilinear thoracoabdominal incision crossing the inferior costal margin is used. Entry into the chest for extent I and II aneurysms typically occurs at the sixth thoracic intercostal space, allowing for the placement of the proximal aortic clamp at the distal aortic arch (37, 46, 47). Extent III aneurysms do not require distal aortic arch clamping; therefore, entry into the chest is generally at the seventh or eighth thoracic intercostal space. Extent IV TAAAs are performed through an oblique incision through the ninth or tenth intercostal space (46, 47).

Renal Protection

The incidence of acute renal injury after DTA and TAA surgery is 5%-13% (48). Acute renal injury is usually due to decreased renal blood flow and reperfusion injury. Increased cross-clamp time (>30 min) is the main risk factor. Other predisposing factors include advanced age, preoperative renal dysfunction, sustained perioperative hypotension and low cardiac output and failure to use atriobypass during operation. Renal protective measures that are the most effective include minimal cross-clamp time and the maintenance of adequate intravascular volumes, cardiac output and perfusion pressures. Adjuncts such as N-acetylcysteine, distal perfusion and hypothermia (both selective renal and systemic) also improve renal

outcomes (49). Requirements for renal replacement therapies, including intermittent haemodialysis and continuous renal replacement therapy, should be evaluated using KDIGO 2012 definition during the perioperative period (50).

Neuroprotection

A devastating complication of TAAA repair is paraplegia, with incidences ranging from 2.7%-20% (51, 52). Extent II procedures have the highest risk of postoperative paraplegia at 6.2%, followed by extent I at 3.3% and extent III at 2.6% (53). Paraplegia is associated not only with significant morbidity but also with increased mortality (54). CSF drainage, the maintenance of MAP, LHB, hypothermia and the reimplantation of intercostal/radicular arteries are effective in decreasing the incidence of paraplegia (47). However, no single method has been proved entirely effective.

Spinal cord perfusion pressure is equal to MAP-CSF pressure. With the placement of an aortic cross-clamp, there is a compensatory rise in venous pressure, resulting in increased intraspinal venous congestion and thereby, causing a rise in CSF pressure (55). Therefore, measures to increase MAP or decrease CSF pressure will augment spinal cord perfusion pressure. CSF removal decreases intrathecal pressure, thereby improving spinal cord perfusion pressure. CSF drainage is facilitated via spinal drain, placed in the lower lumbar region, with a goal of maintaining CSF pressure at approximately 10-15 mmHg (55). A meta-analysis of 14 randomised controlled studies conducted by Cina et al. (56) demonstrated a reduction in the incidence of postoperative paraplegia and lower-limb neurological deficits in patients undergoing CSF drainage. The number needed to treat with CSF drainage was 10, indicating that only 10 patients need to be treated to prevent paraparesis or paraplegia in one patient (56).

Intraoperatively, CSF drain is connected to a pressure transducer and zeroed at the level of the right atrium. CSF pressure transducer should be connected to a non-pressurised saline bag separate from the other invasive pressure transducers. CSF is manually drained to maintain a CSFP <15 mmHg, without exceeding a maximal drainage rate of 10-15 mL h⁻¹. At times of neurological deterioration, brief periods of increased CSF drainage (20 mL h⁻¹) are accepted. The drain is kept in place for up to 72 hours postoperatively. The majority of neurological complications appear immediately after repair; however; delayed neurological deficits (DNDs) have also been reported (57, 58). DNDs, defined as a decline in the neurological status after initial postoperative evaluation, showed the patient to be neurologically intact; this may occur several hours or days following surgery (57). The aetiology of DNDs is thought to be a result of compromised spinal blood flow from an episode of hypotension or increased CSFP leading to further ischaemia (58).

If neurological deficits develop, then the rate of CSF drainage should be increased to lower CSFP; concurrently, MAP should be increased to further increase the SCPP (57).

Although there is a clear benefit of CSF drainage in patients undergoing TAAA repair, it is not without risks. Dardik et al. (59) revealed a 3.5% incidence of subdural haematomas in patients undergoing CSF drainage, which also strongly correlated with the amount of CSF drained. In their study, patients who developed a subdural haematoma had on average 690±79 mL CSF removed, whereas those who did not develop a subdural haematoma had on average of 359±23 mL CSF removed. Other potential complications of CSF drainage include meningitis, spinal or epidural haematomas and puncture headache.

Massive Blood Loss and Coagulopathy

Thoracoabdominal aortic aneurysm repair is associated with significant haemorrhage, fluid shifts and altered coagulation profiles due to extensive surgical repair, prolonged operative times, moderate hypothermia and heparin administration. The extent of TAAA repair has prognostic significance with respect to intraoperative blood loss and coagulopathy. A more proximal aortic cross-clamp and longer duration of aortic clamping correlate with larger reductions in platelets, coagulation factors and fibrinogen levels (60). Coagulopathy commonly occurs during and after DTA aneurysm repair. The causes include dilutional coagulopathy, qualitative platelet dysfunction, heparin effects and fibrinolysis. The extensive blood product and fluid requirements of DTA aneurysm repairs may approach several blood volumes. Qualitative and quantitative platelet deficiencies are the most common causes of perioperative bleeding, most likely attributable to extracorporeal circulation and hypothermia (61). Techniques for monitoring coagulation including thromboelastogram, platelet function analyser and prothrombin time and activated partial thromboplastin time should be used. Blood, platelets, fresh frozen plasma and cryoprecipitate should be readily available in addition to cell saver. Antifibrinolytic therapy in the form of tranexamic acid or α-aminocaproic acid has been considered a viable option for the treatment of coagulopathy in TAAA repair. A summary of intraoperative anaesthetic management is presented in Table 4. After the completion of surgery, protamine administration for the reversal of heparin should be performed as early as feasible for the treatment of ongoing bleeding and reassessment of coagulopathy.

Postoperative Management

Vigilance for the first 48-72 h is of paramount importance in the ICU. Overall goals are haemodynamic stability, normal coagulation and neurological examination. After surgery, patients are cared in the supine position with a slight reversed Trendelenburg tilt if a CSF drainage catheter is in situ. A

single-lumen tracheal tube is exchanged for DLT. MAP is maintained between 80 and 90 mmHg to prevent decrease in spinal cord perfusion pressure. Patients are woken up, and extubation is attempted as soon as possible to evaluate the neurological status. Even if there is no immediate neurological deficit, delayed deficit should be considered. CSF drainage is usually discontinued after 72 h. Pain management can be achieved by continuous opioid infusions titrated to effect or epidural infusions of low-dose local anaesthetic and opioid mixtures. Patients must be closely monitored for any neurological and haemodynamic change. A summary of postoperative management is presented in Table 5.

Conclusion

Although extensive progress has been made over the past decades in reducing TAAA surgical complication rates, continued research efforts are needed to further reduce and eliminate devastating sequelae, in particular, paraplegia and renal failure. Compared with the large number of studies related to spinal cord protection during TAAA repair, those focusing on renal protection in this setting are quite limited. Consequently, major progress in preventing renal failure after TAAA repair remains elusive. The principal strategies for protecting the kidneys during TAAA repair are maintaining renal hypothermia during the ischaemic period, maintaining an adequate perfusion pressure and avoiding the administration of nephrotoxic agents (62). The management of patients undergoing DTA aneurysm repair is very challenging from an anaesthetic standpoint. The level of monitoring, haemodynamic manipulation and neuronal protection strategies are on the cutting edge in every respect. The wide range of therapeutic options is very confusing, occasionally contradictory and continually expanding. Current perioperative care may continue to evolve in the future given that clinical and laboratory data are constantly influencing clinical practice.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – N.G.; Design – D.J.; Supervision – A.S.; Literature Search – A.B.; Writing Manuscript – N.G.; Critical Review – D.J.

Conflict of Interest: The authors have no conflicts of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

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