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Anesthetic Consideration for Descending Thoracic Aortic Aneurysm Repair

Ronald A. Kahn, MD, Marc E. Stone, MD,
and David M. Moskowitz, MD

Anesthesia for surgery of the aorta poses some of the most difficult challenges for anesthesiologists. Major hemodynamic and physiologic stresses and sophisticated techniques of extracorporeal support are superimposed on patients with complex medical disease states. In this review, etiologies, natural history, and surgical techniques of thoracic aortic aneurysm are presented. Anesthetic considerations are discussed in detail, including the management of distal perfusion using partial cardiopulmonary bypass. Considerations of spinal cord protection, including management of proximal hypertension, cerebral spinal fluid drainage, and pharmacological therapies, are presented.

Keywords: aorta; thoracic thoracoabdominal aneurysms; surgery; anesthetic management; cerebral spinal fluid drainage; partial cardiopulmonary bypass

Anesthesia for surgery of the aorta poses some of the most difficult challenges for anesthesiologists. Major hemodynamic and physiologic stresses and sophisticated techniques of extracorporeal support are superimposed on patients with complex medical disease states. With the average age of the population increasing, the number of patients presenting for aortic reconstruction is likely to increase. In 1951, Dubost was the first to successfully remove an abdominal aortic aneurysm (AAA) and replace it with an arterial homograft.[1] Later, DeBakey and Cooley[2] and Bahnson[3] further expanded this procedure to involve the thoracic aorta. Gibbon[4] introduced extracorporeal circulation as an adjuvant to thoracic aortic reconstruction. Morbidity has decreased dramatically over the past 3 decades, undoubtedly from better perioperative care and surgical technique. In this review, we discuss the anesthetic considerations of patients presenting for descending thoracic and thoracoabdominal aortic aneurysm repair (TAAA).

From the Department of Anesthesiology, the Mount Sinai School of Medicine, New York, New York (RAK, MS), and the Department of Anesthesiology, Englewood Medical Center, Engelwood, NJ (DMM).

Address correspondence to Ronald A. Kahn, MD, Department of Anesthesiology, Box 1010, 1 Gustave L. Levy Place, New York City, NY 10029-6574; e-mail: ron.kahn@mssm.edu.

Definitions, Incidence, and Etiologies

An aortic aneurysm may be described as a permanent dilation that is at least 1.5 times the diameter of the expected normal value.[5] Aneurysms can be further described according to their location, morphology, and etiology. Location is commonly used to classify aortic aneurysms for both clinical significance and surgical approach. Typically, the aneurysm can be localized to the sinuses of Valsalva (aortoannuloectasia), ascending, arch, descending, and abdominal portions of the aorta. This type of classification may be misleading, because other portions of the aorta may also be involved. The Crawford classification improves on this shortcoming and is commonly used to describe the location of aortic aneurysms. Approximately 6% of thoracic aortic aneurysms affect the distal aorta and arch, whereas 11% involve the thoracoabdominal aorta.[6] Sixty-five percent of all aortic aneurysms occur in the abdominal aorta.

The morphology of an aneurysm is either fusiform or saccular. Fusiform aneurysms are sausage-like in shape; they are dilated symmetrically 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 classified morphologically as either true or false (pseudoaneurysm). True aneurysms involve all layers of the aortic wall. A pseudoaneurysm is a contained rupture that

Table 1. Etiologies of Thoracic Aortic Aneurysms

Congenital or developmental	Marfan's syndrome, Ehlers-Danlos syndrome
Degenerative	Cystic medial degeneration, nonspecific (atherosclerosis)
Traumatic	Blunt or penetrating
Inflammatory	Takayasu's arteritis, Beçhet's syndrome, Kawasaki's disease; microvascular disorders (ie, polyarteritis)
Infectious (mycotic)	Bacterial, fungal, spirochetal, viral
Mechanical	Poststenotic, associated with arteriovenous fistula
Anastomotic (postarteriotomy)	

extends through all layers of the aortic wall. The integrity of the vasculature is maintained by a surrounding tissues and the chronic inflammatory reaction to extravasated blood.[7]

Aneurysms may be congenital or acquired; the latter occurs more frequently than the former (Table 1). Acquired aneurysms may be attributable to atherosclerosis, cystic medial degeneration, infection, trauma, and iatrogenic or inflammatory processes. Congenital etiologies include Marfan's syndrome and Ehlers-Danlos syndrome.

Atherosclerosis is the most common cause of aortic aneurysms. Dissection and degenerative changes of the media are frequent causative factors of aneurysms of the thoracic aorta.[8] Atherosclerotic aneurysms are most commonly located in the infrarenal aorta and are implicated in more than 90% of AAAs. Atherosclerotic ascending aortic dilation is commonly associated with diffuse aortic atherosclerosis. Aneurysms of the ascending aorta are usually associated with cystic medial necrosis of the media (smooth muscle necrosis and elastic fiber degeneration) and atherosclerosis. This pathologic process leads to weakening of the support apparatus with resultant dilation of the aorta. Syphilis is no longer a common cause of ascending aortic aneurysms. The spirochetes lodge in the vasa vasorum during the secondary phase of the disease, resulting in an obliterative endarteritis that eventually leads to weakening of the aortic wall. Although primary aortic infection may result in aortic aneurysmal disease, secondary infections (eg, bacterial endocarditis or mycotic aneurysm) is more common. Iatrogenic causes include previous heart surgery requiring aortic cannulation and/or aortic cross-clamping. Generalized inflammatory processes like Takayasu's arteritis, giant cell arteritis, ankylosing spondylitis, psoriatic arthritis, and other autoimmune processes may be associated with aortic root and aortic dilation.[9-11] In addition, Behçets disease can cause aneurysms of the aorta, pulmonary artery, and other major vessels.[12]

Most aneurysms are associated with advanced age, hypertension, and smoking.[13] The pathogenesis is multifactorial and probably involves a combination of decreased synthesis, increased breakdown, and decreased inhibition of the breakdown of the supporting matrix of the aorta.[14]

The incidence and prevalence of thoracic aortic aneurysms are difficult parameters to quantify. There are no current screening studies nor is screening feasible. Ultrasound and chest radiography are insensitive and nonspecific, whereas computerized tomography and magnetic resonance imaging techniques are not cost-effective, as reported in a population-based study performed in the Midwest region of the United States.[15] Bickerstaff et al[16] reported the incidence of newly diagnosed thoracic aortic aneurysms as 5.9 per 100 000 person-years. The incidence was equal in both sexes and decreased slightly over the 30-year period of this observational study. The age range was 47 to 93 years (median, 65 years for men and 77 years for women). The ascending aorta was involved in 51% of patients, the aortic arch in 11%, and the descending aorta in 38%. Svensjo et al[17] reported on autopsy records from 1958 to 1985 in a town in Sweden that has an 83% autopsy rate. The overall incidence of thoracic aortic aneurysms was 489 per 100 000 autopsies for men and 437 per 100 000 autopsies for women with a prevalence of about 400 per 100 000 autopsies by age 65 that increased to 670 per 100 000 autopsies by age 80.

Bernstein et al[18] demonstrated an average growth rate of aortic aneurysms of 0.4 cm per year. In a follow-up study of high-risk patients, this group reported an overall rupture rate of 3%, a surgical mortality (elective surgery for aneurysm size greater than 6 cm or symptoms) of 4.9%; 34% of the mortality was from causes unrelated to the aneurysm.[19] Szilagyi et al[20] reported that the 5-year survival for untreated AAA greater than 6 cm was less than 10%, whereas the 5 year survival for untreated AAA less than 6 cm was 50%. Elevated diastolic blood pressure, aneurysm anteroposterior diameter

greater than 5 cm, and obstructive pulmonary disease were independent predictors of rupture.[21] Predicted 5-year rupture rates varied from 2% when these risk factors were absent to 100% when all 3 risk factors were present.

Because of the poor intrathoracic imaging techniques available previously, only a few prospective studies dealing with the natural history of thoracic aortic aneurysms were performed. Juvonen et al[22] described the natural course of 114 patients with moderate sized descending thoracic and thoracoabdominal aortic aneurysms. Of these patients, 8 died of causes unrelated to the aneurysm, 26 died of rupture, 20 eventually met previously determined criteria for operation, and 60 survived without operation or rupture over an approximately 8-year period. Maximal diameter of the aneurysm in the descending aorta and in the abdominal aorta was an independent risk factor for rupture, as well as older age, the presence of uncharacteristic pain, and a history of chronic obstructive pulmonary disease. The average size of the thoracoabdominal aneurysm before rupture was 5.8 cm. Aneurysms 5 to 6 cm in diameter have a faster rate of enlargement than smaller aneurysms.[8,16] Perko et al demonstrated that aortic aneurysms greater than 6 cm were associated with a 5-fold increase in the cumulative hazard of rupture, and they advocate an aggressive surgical approach.

Surgical Approach

The patient is positioned with the thorax in the right lateral decubitus position, with the hips rotated toward a more supine position. This allows access to the descending aorta as well as the femoral vessels, which must be accessible for the institution of partial left heart bypass or partial cardiopulmonary bypass (see below). A posterolateral thoracotomy with extension to an oblique midline vertical abdominal laparotomy is performed. After the aorta is adequately dissected, appropriate cannulae are inserted for perfusion of the distal aorta, and extracorporeal circulation (partial bypass) is begun. Two vascular clamps are placed on the aorta, and the aorta is divided. This perfusion technique allows for anterograde flow to the coronary and brachiocephalic vessels proximal to the aortic clamp and retrograde flow via the femoral artery to tissues distal to the cross-clamp. The anastomosis between the aorta and the proximal aortic graft is performed, and both cross-clamps are moved distally to allow for visceral artery reimplantation. A distal opening is created in the aortic graft, and the visceral button is implanted. After further movement of the aortic cross-clamps, the distal anastomosis is performed, the cross-clamps are released, and partial cardiac bypass is discontinued.

Preoperative Preparation

Patients presenting for aortic surgery nearly all have coexisting medical conditions that can significantly affect anesthetic management. Problems 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, optimize the patient's medical status, and devise an anesthetic technique that minimizes complications. It is not always possible to obtain a complete preoperative evaluation when surgery is required on an urgent basis, and thus preoperative optimization of the patient is not always feasible. Moreover, it is also well established that intrathoracic surgery, aortic surgery, and emergency operations are independent risk factors for major complications, and therefore, these remain high-risk procedures even with an optimal preoperative evaluation.[23]

All antihypertensive and antianginal medications should be continued until the time of surgery. 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 ischemia in patients with concurrent coronary artery disease. Patients presenting for aortic surgery may be very unstable hemodynamically because of ongoing hemorrhage, cardiac tamponade, myocardial ischemia, or congestive heart failure. Organ malperfusion is also a major problem. It is therefore recommended that patients who present for emergency aortic surgery be intensively monitored to control blood pressure and resuscitate appropriately.

Anesthetic Preparation

Standard monitors are placed for all patients presenting for surgery of the aorta. Electrocardiography with the ability to monitor limb and precordial leads (II and V5 at the minimum) is desirable to detect

myocardial ischemia and arrhythmias.[24] Foley catheterization is also prudent to assess volume status and to provide an early indication of renal malperfusion. Temperature monitoring should consist of at least 2 locations that estimate both the core (blood or esophageal) temperature and the visceral (bladder or rectal) temperature. Most clinicians use the tympanic membrane, nasopharyngeal, or esophageal sites as the best indicator of brain temperature, although gradients still exist among different tissue sites.[25]

Extensive hemodynamic changes should be anticipated during aortic surgery. Changes in preload may be attributable to sudden and profound hemorrhage from intercostal and visceral back-bleeding, aortic disruptions, extensive anastomotic suture leaks, and evaporative and third-space losses. Myocardial depression as well as rapid afterload changes may also occur. Large-bore intravenous access is imperative, such as 1 or more large-bore (7F to 9F) venous (introducer) cannulae. Possible sites for these cannulae include the femoral veins, antecubital fossa, subclavian vein, or internal jugular veins. Patient positioning and surgical considerations may preclude the use of certain sites. Rapid infusion systems with blood-warming capacity are necessary. Blood salvaging techniques for autotransfusion should be used. The easiest technique for blood salvaging is the use of a centrifugal device that scavenges and washes erythrocytes. The disadvantages of this technique are the delays involved in filling the centrifugal bowl and processing the blood and the loss of the plasma volume, proteins, coagulation factors, and platelets. To complement the use of an erythrocyte salvager in adequately heparinized patients, a separate cardiotomy suction may be set up to return shed blood to a cardiotomy reservoir for autotransfusion. Autotransfusion 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.[26]

Large-bore intravenous access is extremely important in aortic surgery. Aortic rupture may always occur, and the ability to rapidly infuse intravenous fluids, blood or blood products is necessary. One or 2 large-bore peripheral intravenous lines are recommended along with some form of central venous access. Because the left innominate vein may be injured or intentionally divided during aortic arch surgery, left-side intravenous access should be avoided if aortic arch repair is anticipated. A rapid infusion system with a heat exchanger should be immediately available. Blood should also be immediately available and possibly even primed through the rapid infusion system.

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 the 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. It is necessary to consult with the surgeon before cannulating the femoral vessels because these vessels may be used for extracorporeal perfusion or placement of an intra-aortic balloon pump during the surgical procedure. Alternatively, the pressure in the descending aorta distal to the repair may be measured directly by having the surgeon place a small needle into the aorta and attaching this to a transducer for measurement of the aortic pressure.

Cannulation of a central vein is routinely performed for ascending aortic and aortic arch surgery to measure filling pressures in the heart (using a central venous catheter or a pulmonary artery catheter) and provide a central route for drug administration and for the rapid infusion of fluids. Central venous access can be accomplished with a large-bore cannula (introducer) in the right or left internal jugular veins. If the femoral vein is chosen for central venous access, then consultation with the surgeons is recommended, because the femoral veins may be used for extracorporeal perfusion.

Pulmonary artery catheterization should be strongly considered in all patients undergoing thoracoabdominal aortic repairs. The justifications for this monitor include concomitant cardiac disease with preexisting or intraoperative left ventricular dysfunction and the need for left ventricular preload and afterload assessment. The monitoring of right-side pressures as the sole indication of volume status is probably not sufficient.

Transesophageal echocardiography is a minimally invasive procedure that has a proven safety record.[27] The close anatomic relationship of the esophagus to the aorta and the heart allows TEE to provide excellent high-quality images without significant interference from the overlying structures (lungs and chest wall). With the introduction of biplane and multiplane TEE probes, more complete definitions of the distal ascending aorta and aortic arch are possible; however, imaging of the distal ascending aorta and proximal aortic arch is

still unreliable because of the interposition of the trachea or bronchus between the TEE probe and aorta. TEE is performed in real time, allowing for its unique ability to give functional and hemodynamic information. The extent and severity of the aortic pathology may be identified and confirmed. Ventricular dysfunction and regional wall motion abnormalities may be diagnosed, and the proper placement of venous cannulae can be confirmed. In the presence of an aortic dissection, it is possible to inadvertently cannulate the false lumen of the femoral artery. Using TEE, it is possible to ascertain that there is appropriate flow in the true lumen on commencement of bypass. This enables the evaluation of the aortic valve for regurgitation, the pericardial space for tamponade, and the left ventricle for evidence of dysfunction. Flow in both the true and false lumina can be analyzed with Doppler color flow imaging and pulsed or continuous wave Doppler, allowing for an alternative technique to identify intimal tears when not directly visualized by 2-dimensional imaging. The proximal coronary arteries can be observed for possible involvement in the dissection process.

Nonetheless, TEE has some risks and limitations. There is still the risk (albeit small) for the probe to cause damage in the region of the oropharynx, esophagus, or stomach. Hypotension or hypertension can occur rapidly secondary to complications from the aortic dissection (eg, tamponade) or from stimulation of the oropharynx by the TEE probe, respectively. Thus, continuous intra-arterial monitoring is usually indicated. Additionally, endotracheal intubation should be considered in patients at risk of aspiration of gastric contents. The TEE is limited in its view of the distal ascending aorta and aortic arch despite the advantages that the biplane and multiplane probes have afforded over the monoplane probe. Finally, the abdominal aorta and flow to the major vessels that branch from aorta are not clearly imaged.

A double-lumen endotracheal tube (DLT) should be inserted for lung separation. The use of single-lung ventilation 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. A large descending thoracic aortic aneurysm may compress or distort the left main bronchus. Because of the distortion of airway anatomy, left-side endobronchial tubes should be used with caution. If a right-side DLT is placed, fiberoptic confirmation of tube position relative to the carina is mandatory. After the procedure, the decision to change the DLT to a single-lumen tube should be made after carefully evaluating the extent of airway edema, because these procedures are associated with significant facial and laryngeal edema. Direct laryngoscopy should be performed to confirm the ability to visualize the vocal cords before extubation. Endotracheal tube exchangers may also be used to facilitate changing of the endotracheal tube. This device may be used to identify the laryngeal opening or may be used for jet ventilation if reintubation becomes difficult.

Coagulopathy commonly occurs during and after descending thoracic aortic aneurysm repair. The causes include dilutional coagulopathy, qualitative platelet dysfunction, heparin effects, and fibrinolysis. The extensive blood product and fluid requirements of descending thoracic aortic aneurysm repairs may approach several blood volumes. Qualitative and quantitative platelet deficiencies are the most common cause of perioperative bleeding, most likely attributable to extracorporeal circulation and hypothermia.[28]

Illig and colleagues[29] reported significantly decreased euglobulin clot lysis times, elevated tissue plasminogen activator levels, elevated tissue plasminogen-to-plasminogen activator inhibitor-1 ratios, and reduced α 2-antiplasmin levels within 20 minutes after supraceliac cross-clamping, when compared with infrarenal occlusion. These findings are consistent with a primary fibrinolytic state. Visceral ischemia (which does not occur with infrarenal aortic clamping) may be the condition that initiates the fibrinolysis. During supraceliac occlusion, fibrinolysis was attenuated when superior mesenteric artery perfusion was maintained via a shunt.[30] Peripheral ischemia may also result in fibrinolysis. Within 30 minutes of the onset of acute peripheral ischemia, (infrarenal aortic occlusion) fibrinolytic activity increased, as demonstrated by an increase in tissue-type plasminogen activity (TPA) and a decrease in plasminogen activator inhibitor activity. This increase in TPA resulted from release of stores from ischemic vascular tissues.[31] Endotoxemia during aortic occlusion may also be associated with fibrinolysis.[32]

Antifibrinolytic agents should be considered during the perioperative period. Compared with placebo, tranexamic acid use during thoracic aortic surgery decreases perioperative bleeding with subsequent reduction erythrocyte and total amount of allogeneic transfusion.[33] No increased risk of thrombotic complications was observed. Aprotinin is a nonspecific serine protease activity and inhibits plasmin, thrombin, trypsin, and tissue and plasma kallikrein. In addition, aprotinin prevents activation of platelet thrombin receptors (PAR-1,3,4) and the platelet glycoprotein receptors (GPIbIIa

and GPIIbIIIa).[34] Aprotinin has been effective in decreasing bleeding during and after cardiac surgery.[35,36] It is unclear, however, whether other less expensive fibrinolytic agents are similarly effective. In a meta-analysis, a greater reduction in blood loss was seen with aprotinin use, but this magnitude of this reduction was not clinically significant.[37] Mangano et al[38] used propensity adjusted multivariable logistical regression from observational data of 4374 patients. They reported an association between aprotinin use and adverse renal, cardiac, and neurological outcome. Similarly, in another observational study using propensity scoring, Karkouti et al reported an association of aprotinin use with renal dysfunction.[39] Other studies found no deleterious effects of aprotinin and demonstrated a reduction in cerebrovascular outcomes.[36,40]

Hemodynamic Changes Associated With Aortic Clamping and Unclamping

The hemodynamic consequences of aortic occlusion and release have been previously described in detail (see Table 2).[41] The most consistent hemodynamic response to acute aortic occlusion is an abrupt increase in afterload with a resultant increase in proximal aortic pressure. During supraceliac aortic occlusion, there is an increase in preload attributable to volume redistribution from 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 ischemia. Because of the expected increases in preload from aortic occlusion, the preocclusion preload should be maintained low. Venodilators such as nitroglycerin (NTG) may be titrated to further decrease preload.

The control of proximal hypertension is a difficult problem during aortic reconstruction. Although it is important to attenuate the increase in mean arterial pressure (MAP) that is caused by aortic cross-clamp application, spinal cord perfusion pressure (SCPP) should not be compromised. Proximal arterial pressure control using sodium nitroprusside (SNP) has been well studied. When compared with no treatment of proximal hypertension after experimental thoracic aortic occlusion, SNP increased cerebral spinal fluid (CSF) pressure.[42] In addition to having this effect on CSF pressure, SNP also reduced distal aortic pressure compared with animals in the control group. The combination of increased CSF pressure and decreased distal aortic pressure resulted in a significant decrease in SCPP and spinal cord blood flow in the lower spinal cord segments. There was an earlier loss of somatosensory evoked potentials (SSEPs) after aortic occlusion as well as a poorer neurological outcome with the use of SNP, which would not be likely to improve with a cerebral spinal fluid drain (CSFD).[43] Other investigators have also observed decreases in distal aortic pressure with the use of SNP as well as a delay in the return of SSEP signals after resumption of spinal blood flow and a worsening in neurological outcome.[44]

Table 2. Hemodynamic Changes Associated With Aortic Occlusion

Increased afterload
Increased preload secondary to venous redistribution
Increased contractility secondary to increased catecholamine release
Decreased heart rate

The use of NTG to control proximal arterial hypertension is also problematic.[45] When compared with induced hypovolemia (exsanguination), NTG use resulted in significant decreases in SCPP, which was primarily attributable to decreases in distal aortic pressure (ie, there were no differences in CSF pressures between the animals that were made hypovolemic vs those that received NTG). NTG also resulted in worse neurological outcomes. In another study, SNP with CSFD and NTG with CSFD were compared with NTG alone in the management of proximal hypertension.[46] NTG use was associated with greater distal aortic pressures compared with SNP; however, NTG resulted in low spinal cord perfusion pressures when CSFD was omitted. The authors concluded that the use of NTG alone or SNP (even with CSFD) may result in adverse neurological outcome by impairing SCPP.

Esmolol infusion may be safely used to control proximal hypertension. Esmolol during aortic occlusion may reduce cardiac output and increase ventricular filling pressures. Compared with SNP or no treatment, esmolol was associated with greater SCPP.[47] Increasing the depth of anesthesia may also be effective in controlling proximal hypertension. The use of isoflurane resulted in greater SCPP and was associated with a lower incidence of neurologic injury when compared with nitroprusside in a canine model of thoracic aortic occlusion.[48]

Phlebotomy after aortic occlusion significantly decreased MAP, CVP, and CSF pressure, suggesting that cardiac preload may be an important determinant of CSF pressure.[49] Because there was concomitant hypotension with exsanguination, no change in SCPP was observed. In a study comparing exsanguination, a

Table 3. Differential Diagnosis of Hypotension During Partial Cardiopulmonary Bypass

Hypovolemia
Myocardial depression
Decreases in afterload
Excessive partial bypass pump flow

Table 4. Differential Diagnosis of Hypotension During Reperfusion

Central hypovolemia
Hypoxia-mediated vasodilation
Accumulation of vasoactive or myocardial-depressant metabolites
Acute acidosis
Acute hypocalcemia

combination of nitroprusside and isoflurane, or no treatment, significant increases in CSF pressure and decreases in SCPP were observed with the use of nitroprusside compared with the other 2 groups.[50] Although the incidence of adverse neurologic outcome was least with phlebotomy, these differences did not reach statistical significance. On a practical basis, phlebotomy into a cardiotomy reservoir and intermittent volume loading via the extracorporeal circuit are probably the ideal method of controlling proximal hypertension during thoracoabdominal aortic surgery.

Intraoperative hypotension may result from multiple causes (Tables 3 and 4). Hypovolemia, myocardial depression, and decreases in afterload should be considered. Reperfusion is associated with hypotension. This hypotension may be caused by central hypovolemia attributable to blood pooling in reperfused tissues, hypoxia-mediated vasodilation, and accumulation of vasoactive or myocardial-depressant metabolites, such as lactate. Treatment should be directed toward rapid correction of hypovolemia, acidosis, hypocalcemia and the judicious administration of vasoactive drugs. If there is difficulty obtaining hemodynamic stability, the aorta can be temporarily reoccluded while resuscitation continues.

Renal Protection

The incidence of renal insufficiency after descending thoracic aortic repair is approximately 25%; of these patients, 8% will require hemodialysis. Risk factors that are associated with postoperative renal failure after descending thoracic aortic repair include age greater than 50 years, preexisting renal dysfunction, duration of renal ischemia, administration of greater than 5 units of either packed red cells or salvaged washed autologous ("Cell-Saver") blood, hemodynamic instability, and diffuse atherosclerosis.[51,52]

The attenuation of renal ischemia may decrease the incidence of postoperative renal dysfunction. There is controversy regarding the protective nature of distal perfusion during aortic occlusion. Godet et al[53] observed a decrease in the incidence of renal failure with the use of distal aortic perfusion. Others have observed increases in renal failure with distal bypass.[54] Selective renal artery perfusion during descending thoracic aortic repair may result in uninterrupted urine production throughout the procedure and may decrease the incidence of renal failure postoperatively.[55]

Pharmacological agents may also provide renal protection during descending thoracic aortic repair. Nicolson et al[56] administered mannitol 0.3 g/kg immediately before aortic cross-clamping. No differences in postoperative blood urea level, serum creatinine concentration, or creatinine clearance were observed between the mannitol and control groups. Patients receiving mannitol had greater diuresis on postoperative day 1 and less subclinical glomerular and renal tubular damage. There is little evidence of the effectiveness of furosemide as a renal protective agent. Hager et al[57] randomized patients undergoing major vascular surgery to low-dose continuous intravenous furosemide infusion or placebo. They observed no differences between groups with respect to postoperative creatinine clearance, but there was a significant increase in the incidence of hypokalemia with the use of furosemide.

Low-dose dopamine (1-3 μg/kg/min) dilates renal afferent arterioles and increases renal blood flow, independent of its cardiac effects. Dopamine infusion during aortic clamping results in a significant increase in urine sodium output, potassium output, creatinine clearance, and urine volume.[58] The use of perioperative dopamine during aortic surgery is associated with increases in effective renal plasma flow and glomerular filtration rate as well as fractional excretion of sodium during the postoperative period.[59] Pavoni et al[60] observed a dose-dependent increase in urinary output in most patients administered renal dopamine who exhibited oliguria after major vascular surgery. In a randomized trial during major aortic surgery, the use of perioperative renal-dose dopamine (3 μg/kg/min) was compared with volume loading (euvolemia). There was no statistical or clinically significant effect on blood urea nitrogen or creatinine concentrations or creatinine clearance perioperatively.[61] The authors concluded that renal-dose dopamine administration during

the perioperative period confers no advantage over volume loading (maintenance of euvolemia) in most vascular patients during infrarenal abdominal aortic aneurysm repair.[61,62]

Spinal Cord Protection

Paraparesis and paraplegia are perhaps the most feared complications resulting from repair of the descending thoracic aorta. Although rates as high as 23% have previously been reported, the current incidence is probably somewhere around 2% to 6%.[63-67] In any specific patient, however, the likelihood of neurological complications depends highly on individual anatomy, on whether the aorta is dissected or just aneurysmal, and on whether the pathology is acute, chronic, or both.

Risk factors for perioperative spinal cord injury include emergency surgery, dissection, extensive disease, prolonged aortic cross-clamp time, aortic rupture, level of aortic cross-clamp, patient age, and a history of renal dysfunction. The risk of paraplegia or paraparesis is minimal if the aortic cross-clamp time is less than 15 minutes.[68] Svensson et al[69] reported a 20% risk of neurological injury if aortic cross-clamp time was greater than 60 minutes and less than 10% if the repair was performed in less than 30 minutes. There are, however, reports of paraplegia or paraparesis with aortic cross-clamp times of less than 20 minutes,[70] whereas others have concluded that aortic cross-clamp times greater than 40 minutes did not result in increases in adverse spinal cord outcome if distal perfusion was used.[71]

Spinal Cord Blood Flow

The vertebral arteries supply the spinal cord superiorly with poorly defined circumferential anastomotic channels, whereas the superior posterolateral spinal cord blood flow is occasionally supplied by the posterior inferior cerebellar arteries. These posterior arteries are plexiform, whereas the anterior arteries are more continuous in nature. The anterior spinal artery is unique in that it is a single long vessel that nourishes the motor aspect of the spinal cord. In most cases, very few radicular arteries supply the anterior spinal artery; consequently, the anterior spinal cord has little if any reserve of blood supply. Although the posterior rami of the intercostal arteries may potentially feed the anterior spinal cord, only a fraction of these arteries actually reach the cord. The superior cord is usually supplied by 3 to 5 anterior radicular arteries, the upper thoracic cord has 1 or no anterior arterial supply, and the lower thoracic and lumbar cord is supplied by 3 to 5 anterior thoracic arteries, the most important of which is the artery of Adamkiewicz. This artery arises from T5 to T8 in 15% of individuals, T9 to T12 in about 60% of individuals, and L1 in approximately 25% of individuals. If the cross-clamp is applied distal to this artery (such as during infrarenal aortic reconstruction), the probability of spinal cord ischemia is low. Conversely, if this critical intercostal artery branch is patent and emerges from the aneurysm wall or below the level of the aortic cross-clamp, a significant spinal cord ischemia is possible during surgery.

Many of the intercostal arteries are chronically occluded in patients with thoracoabdominal aneurysms, and spinal cord perfusion in atherosclerotic patients often depends on a collateral network that develops and feeds the anterior spinal artery. Important contributions have been demonstrated to come from lumbar arteries, perivertebral vessels, the paraspinous muscles, and pelvic circulation. It has also been demonstrated that this collateral network is very dynamic and can supply blood to the spinal cord from 1 source when another is reduced. Furthermore, that steal can occur if alternate low-resistance pathways are opened.

Spinal Cord Perfusion Pressure

Spinal cord perfusion pressure and hence spinal blood flow are the difference between distal aortic pressure and CSF pressure. Spinal cord ischemia may therefore be induced by preoperative hypotension, distal aortic hypotension during aortic cross-clamping and surgical repair, transection of major anterior spinal arteries, profound increases in CSF pressure, or postoperative hypotension or hypoxemia. Therefore, the strategies for spinal cord protection are 3-fold: maintenance of spinal cord arterial pressure; minimization of CSF pressure; and attenuation of spinal cord injury after ischemic insult (Table 4).

Maintenance of Spinal Cord Arterial Pressure

As discussed above, the vertebral arteries contribute to the blood flow of the anterior spinal cord. Proximal hypertension may increase the contribution of the vertebral artery–derived blood flow to the spinal cord as well as collateral flow. Other methods

of maintaining arterial flow to the spinal cord include the aggressive reimplantation of major intercostal arteries into the aortic graft. This may be accomplished either by incorporating the posterior aspect of the aorta that contains the intercostal artery origins or incorporating a patch consisting of intercostal origins into the proximal aortic segment. Intrathecal papaverine has also been described as a method of inducing spinal cord arterial dilation and thus increasing spinal cord blood flow.[72]

If aortic cross-clamps are placed relatively close to each other, excluding only the segment of aorta being repaired, mechanical adjuvants (such as partial cardiopulmonary bypass using a left-atrial femoral artery circuit) may be used to provide continued blood flow to the aortic segments distal to the distal aortic cross-clamp. This should provide continued perfusion to the spinal cord and visceral organs during periods of aortic occlusion. In addition to the vertebral artery–derived flow to the anterior spinal artery, the use of these adjuvants allows continued blood flow to the spinal cord through the artery of Adamkiewicz (if it is not divided or originating in the region between the aortic cross-clamps), distal intercostal arteries, and the hypogastric artery–derived component of spinal cord flow.

The simplest technique for descending aortic reconstruction is single-clamp aortic occlusion or "clamp and run."[73,74] It also avoids the problems associated with larger doses of heparin. To minimize visceral ischemic time and possibly spinal cord ischemia, arterial shunts or bypasses may be used for distal perfusion during aortic occlusion. A heparin-bonded Gott shunt may be placed from the left ventricular apex, ascending aorta, arch, or proximal descending aorta to the distal aorta. Flow through this shunt is passive, and a flow probe may be used to monitor the adequacy of distal perfusion. The major disadvantage of this shunt is the inability to control flows and hence proximal or distal aortic pressures. Other modalities of extracorporeal support during descending aortic reconstruction include partial femoral vein–femoral arterial bypass with an interposed oxygenator or left atrial–femoral arterial bypass. The final option is full cardiopulmonary bypass with or without DHCA.[75] The use of cardiopulmonary bypass allows use of more profound degrees of hypothermia and increases the tolerance of the spinal cord to periods of ischemia. These mechanical modalities allow precise control of proximal and distal aortic pressures.

The decision regarding the modality of partial bypass for an individual operation is primarily surgical. The establishment of femoral vein–femoral artery bypass is technically easiest but requires a more complex extracorporeal circuit, oxygenator, and full heparinization. Next in technical ease is an aortoaortic (or aortofemoral arterial) shunt (with or without an interposed pump). This shunt may be used without heparinization but requires aortic manipulation with the risk of atheromatous embolization.

For left atrial–femoral artery bypass, the pericardium is opened through a thoracotomy incision, and either the left atrial appendage or the left inferior pulmonary vein is cannulated. Alternatively, if femoral venous–femoral arterial partial bypass or full cardiopulmonary bypass is used, the femoral vein is cannulated with a long venous cannula, which is threaded into the right atrium. Transesophageal echocardiography may be used to confirm the position of the venous cannula at the junction of the inferior vena cava and the right atrium. Proper positioning of the venous cannula is important to ensure adequate venous outflow. Arterial cannulation is usually at the femoral artery; however, the iliac artery or the distal aorta may occasionally be used. Partial bypass is usually performed using a vortex (centrifugal) pump, which has the advantages of trapping air bubbles (decreasing the chance of embolization) as well as minimizing trauma to blood cells. A cardiotomy reservoir may be placed in the extracorporeal circuit to salvage shed blood in the surgical field. In the case of sudden hypovolemia, the reservoir can be drained into the patient to restore preload. Alternatively, blood may be diverted from the patient into this reservoir to reduce proximal aortic hypertension or to induce short, controlled periods of hypotension. An oxygenator or heat exchanger will be needed if femoral venous–femoral arterial or full cardiopulmonary bypass is used. If an oxygenator is used, full heparinization (300 units/kg) should be administered.

After confirmation of good left atrial cannula position by TEE, bypass is initiated at a flow of 1.5 L/min to ensure adequate ability to pump blood. During aortic occlusion, both proximal and distal arterial pressures are monitored. Generally, the goal is to equilibrate mean proximal and distal pressures. The differential diagnosis of proximal arterial hypotension includes excessive distal aortic flow with distal aortic hypertension, hypovolemia (as diagnosed by TEE or low filling pressures), and myocardial depression. Hypertension should be treated by ensuring anesthetic depth, decreasing preload with controlled hypovolemia or short-acting β-blockers. Because of the risk of spinal cord steal, vasodilators should be avoided if possible. The minimum desirable

distal arterial pressure is 60 mm Hg to ensure adequate spinal cord blood flow, whereas maximal proximal MAP should be about 90 to 100 mm Hg.[76] Excessive proximal aortic pressures may result in proximal anastomotic leak, severe increases in left ventricular end-diastolic pressures, or aortic regurgitation. The maintenance of adequate spinal cord perfusion should be confirmed by the presence of either somatosensory or motor evoked potentials. The pump flow may be restricted by the venous return as well as the resistance to arterial outflow.

Minimizing Cerebral Spinal Fluid Pressure

Application of a cross-clamp to the proximal descending aorta not only creates a major hemodynamic load on the heart but also causes an acute elevation in CSF pressure. When these physiologic alterations are accompanied by a critical reduction in blood supply to the spinal cord, measures should be taken to increase the SCPP. The increase in CSF pressure within the spinal cord is most likely attributable to volume changes in the capacitance veins of the epidural space, but it is theorized that an increase in this pressure does not result in spinal cord ischemia via direct compression (spinal cord compartment syndrome).[77] When CSF pressure exceeds spinal venous pressure, a critical closing pressure is achieved, and the veins collapse independent of inflow pressure. The SCPP is therefore the difference between spinal arterial pressure and CSF pressure. This physical principle is the best rationale for the use of CSF drainage during thoracic aortic surgery. The patency of the compromised arteriolar circulation and the thinned-walled intradural radiculospinal veins must be ensured. Finally, surgical retraction of the aortic arch may also produce significant increases in CSF pressures.[78]

Laboratory evidence supporting the use of CSF drainage is contradictory. Although high CSF pressures are associated with decreased spinal cord perfusion, further decreases in CSF pressure below normal values (ie, <10 mm Hg) do not result in further increases in spinal cord perfusion.[79] In a canine model of thoracic aortic occlusion, the addition of CSF drainage to aortofemoral bypass did not result in improved spinal cord blood flow or neurologic outcome compared with aortofemoral bypass alone.[80] In contrast, other laboratory studies have demonstrated an improvement in neurological outcome after aortic occlusion with CSF drainage.[81,82]

Cosselli et al[83] randomized 145 patients undergoing thoracoabdominal aortic repair with or without CSF drainage. All patients had permissive mild hypothermia, left heart bypass, and reattachment of patent critical intercostal arteries. CSFD (n = 76) was initiated during the operation and continued for 48 hours after surgery with a CSF pressure of 10 mm Hg or less. Both groups had similar risk factors for paraplegia, aortic clamp times, left heart bypass times, and number of reattached intercostal arteries. In 9 patients (13.0%) in the control group, paraplegia or paraparesis developed. In contrast, deficits developed in only 2 patients in the CSFD group (2.6%) (P = .03). No patients with CSFD had immediate paraplegia. The authors concluded that CSFD resulted in an 80% reduction in the relative risk of postoperative deficits.

The clinical use of lumbar CSF drainage is not without risks. Possible complications include headache, spinal or epidural hematoma formation or inflammatory reaction, meningitis, and persistent CSF leaks. Additionally, the introduction of blood into the subarachnoid space may result in vasospasm and decreases in spinal blood flow.[84] Decreases in CSF pressure may be realized with phlebotomy and aggressive use of hyperosmotic agents, and hyperventilation may be as effective as spinal drainage in maintaining spinal cord perfusion pressure.[85,86]

Attenuation of Ischemic Injury

Hypothermia. It was hypothesized originally that hypothermia provides neuronal protection by decreasing cerebral metabolic rate. Recent experimental evidence of cerebral protection by mild degrees hypothermia cannot be explained by metabolic suppression alone. Furthermore, anesthetic agents that are equally suppressant of cerebral metabolism do not confer equal degrees of protection to ischemic insult. It is likely, therefore, that mild hypothermia may provide significant neuronal protection by mechanisms, such as reducing excitatory neurotransmitter release, decreasing free radical production, decreasing postischemic edema, and stabilizing central nervous system blood flow.

The cooler the cord, the greater the period of tolerable ischemia. Rabbits were randomized to receive burst-suppressing doses of thiopental at normothermia or halothane 1% at normothermia, mild hypothermia (35°C), or moderate hypothermia (32°C) before 20 minutes of spinal cord ischemia.[87] All hypothermic animals were neurologically intact at 48 hours, which was significantly better than the

2 of 7 animals in normothermic halothane group and 1 of 7 animals in the normothermic thiopental group that had normal neurological outcomes. Marsala et al[88] demonstrated significant improvement of behavioral function, pathologic outcome, and spinal cord blood flow reactivity to CO_2 with induction of mild global hypothermia (34°C).

Arrhythmias, such as atrial fibrillation and even ventricular fibrillation, can occur if hypothermia is too severe. Hypothermia is achieved easily by passive cooling in a cold operating room with a major incision. Cooling blankets and unwarmed intravenous fluids contribute to the cooling. If an extracorporeal circuit is used, a heat exchanger permits regulation of the body temperature in either direction. A temperature of 32°C is usually well tolerated by patients not undergoing full CPB.

Alternatively, epidural cooling may be used to induce regional hypothermia. In a rabbit model, epidural cooling was performed with continuous perfusion of 2°C to 4°C saline before and during 40 minutes of spinal cord ischemia.[89] Rabbits without epidural cooling all developed spastic paraplegia and extensive necrotic changes of the lumbosacral gray matter. In contrast, all animals with epidural cooling had full recovery of spinal cord function. Epidural cooling may be used in the operating room. Cambria et al[90] inserted low thoracic epidural catheters as well as lumbar spinal catheters during thoracic and thoracoabdominal aortic repairs. Selective cooling was accomplished by infusing 4°C saline through the epidural catheter. In their 70 patient cohort, the authors infused 1442 ± 718 mL of infusate and were able to reduce the CSF temperature to 24°C ± 3°C while maintaining a core temperature of 34°C ± 0.8°C. Although this technique was associated with substantial increases in CSF pressure, the authors reported a significant reduction in postoperative neurological deficits.

Postischemic hyperemia occurs in the spinal cord.[91] The magnitude of this hyperemia has been demonstrated to be proportional to the incidence of paraplegia. Possible mechanisms for the increased neurologic injury associated with vascular hyperemia include edema formation with the development of a compartment syndrome and subsequently decreased spinal cord perfusion, and increased oxygen delivery that may result in greater free oxygen radical species generation.

Pharmacology

The efficacy of barbiturates in ameliorating spinal cord injury is controversial. Pretreatment of rabbits with thiopental decreased the incidence of paraplegia after 25 minutes of spinal cord ischemia and permitted longer periods of electrophysiological silence without permanent neurological deficit.[92] Similarly, the duration of spinal cord ischemia necessary to produce neurological deficits in 50% of animals increased from 26 to 41 minutes with introduction of hypothermia or thiopental, with an increase to 57 minutes when they were combined.[93] In contrast, Mutch et al[94] did not observe any differences in neurological outcome after addition of methohexital in doses that produced an isoelectric electroencephalogram compared with isoflurane, 1.4%. Differences in the results of these studies may be attributable to differences in study methodology, differences in the degree of spinal cord ischemia, and, most important, inconsistent management of perischemic temperature.

Steroids and Mannitol

The use of methylprednisolone (30 mg/kg) before and after aortic occlusion may result in better spinal cord protection.[95] The mechanism of this protection is unclear but may be related to decreased spinal cord edema and free radical scavenging. Similarly, mannitol (0.25-1.0 g/kg) has been used to modulate the extent of ischemic spinal cord injury. Mannitol is hypothesized to act in similar fashion to methylprednisolone.

Monitoring of Spinal Cord Function

In general, the comparatively low current incidence of neurological complications is attributable to the routine use of multimodal neurophysiologic monitoring (eg, SSEPs and MEPs) in conjunction with neuroprotective strategies (eg, cerebrospinal fluid drainage, mild hypothermia, distal perfusion techniques, high-dose steroid administration). Although these neuroprotective strategies continue to evolve and may differ slightly in their implementation from center to center, monitoring of evoked potentials during these cases has become common because it provides the surgeon with the opportunity to promptly intervene if alterations in monitored potentials indicate neurologic compromise is occurring.

Evoked potential monitoring is based on the detection of periodic changes in latency and amplitude as a result of a given stimulus. These signals are generated as a result of synaptic transmission during the course of this stimulation. Generally, a decrease in amplitude

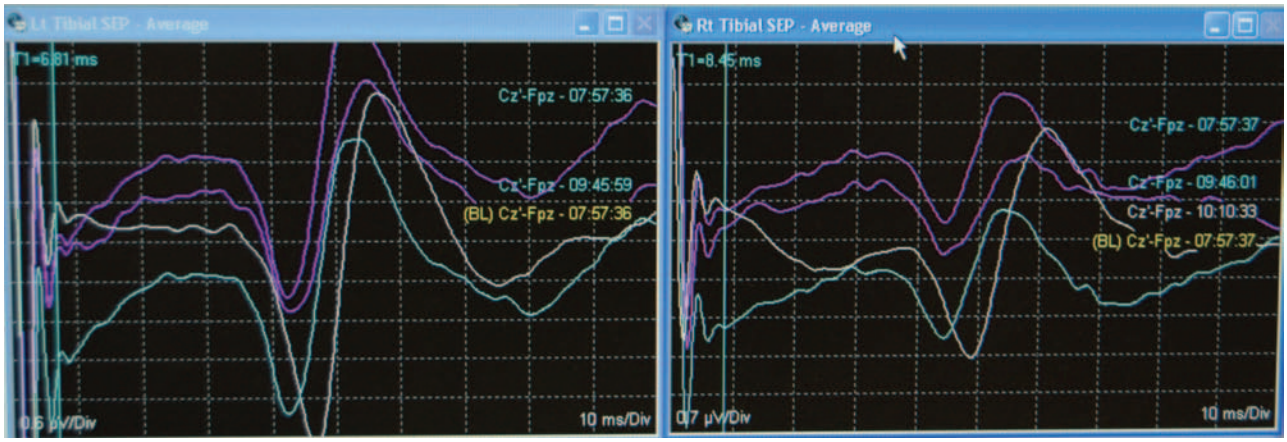

Figure 1. The typical appearance of intraoperatively monitored SSEPs.

or an increase in signal latency is indicative of pathway interruption by either physiological (eg, hypotension or extreme hypothermia) or pathophysiological (eg, spinal cord ischemia) factors. Evoked potential monitoring requires the extraction of low-amplitude signals from a background of higher amplitude noise. More effective extraction of these low-amplitude signals requires optimization of the signal-to-noise ratio. In other words, the signal (ie, wave amplitude) must be maximized, or the noise must be minimized. One method of eliminating noise is the elimination of electromyogenic potentials by using paralytic medications. Obviously, this is not a viable option if myogenic evoked potentials will be used.

SSEPs are recorded at the scalp as cortical signals that are stimulated at the posterior tibial or peroneal nerves, which are conducted to the lateral and posterior columns of the spinal cord.[96] Figure 1 shows the typical appearance of intraoperative tibial SSEP monitoring. Because SEP monitoring is less sensitive to anesthetic drugs and can involve the use of paralytic agents, the use of SEP is less complex than MEP monitoring. It is, however, limited because it is only dependent on the integrity of the sensory pathway: posterior and lateral column. The anterior motor column is more likely to be effected by ischemic injury during aortic reconstruction. It is thus possible to sustain an isolated perioperative anterior column injury without changes in SEP.

In contrast, MEPs are performed by stimulating the motor cortex and recording at the level of the spinal cord, peripheral nerves, or muscles.[96] This stimulus may be a high-voltage, short-duration electrical stimulus or magnetic induction.[97] Neurogenic MEPs are responses recorded at the peripheral nerves, whereas myogenic MEPs are large biphasic responses recorded over the muscle belly. Because the amplitude of the response is proportional to the number of motor neurons being stimulated, these evoked potentials are very sensitive to neuromuscular blocking and anesthetic agents. The typical intraoperative appearance of myogenic MEPs appears in Figure 2.

The motor and sensory tracts in the spinal cord are perfused from different arterial supplies. The anterior motor tracts are served by the anterior spinal artery and the posterior sensory tracts by the posterior spinal artery. However, many intercostal and lumbar arteries are occluded in patients with TAAAs, and spinal cord perfusion often depends on a collateral network, which includes lumbar arteries and pelvic circulation.[66]

Although it has been shown that injury to the anterior (motor) pathways is unlikely to occur in the absence of a detected injury to the posterior (sensory) pathways during surgery to correct scoliosis,[98] monitoring of SSEPs alone has not been demonstrated to improve outcomes in patients undergoing TAAA repair. Studies have shown that SSEP monitoring is associated with delayed ischemia detection compared with transcranial MEPs[99] as well as high rates of both false-negative and false-positive results.[99,100] Because the anterior motor tracts are of greater clinical interest than the posterior sensory tracts, the monitoring of

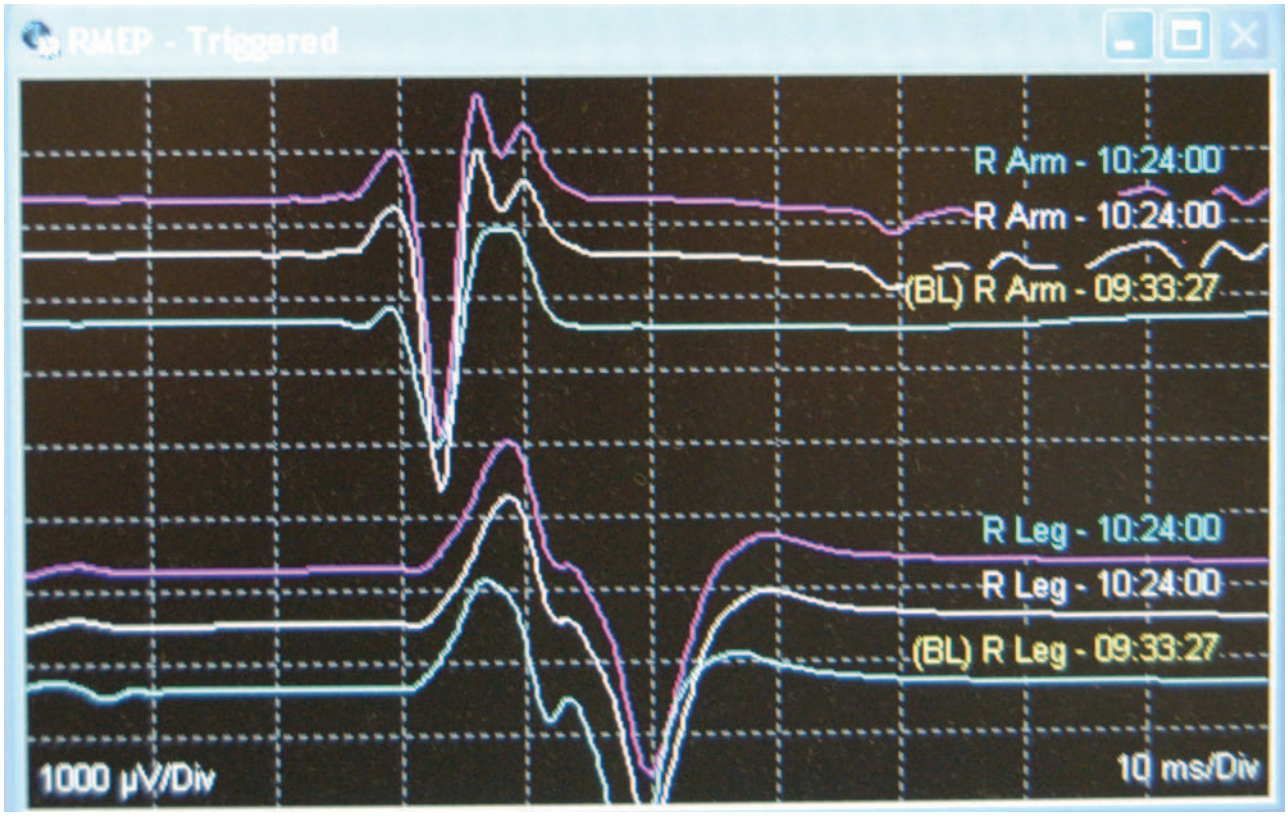

Figure 2. The typical appearance of intraoperatively monitored MEPs.

MEPs is important for identifying segmental arteries that are actually critical to spinal cord perfusion. If acute changes in evoked potentials are noted following segmental arterial sacrifice at a given vertebral level, hemodynamics must be optimized (eg, one must increase arterial blood pressure and/or distal perfusion pressure) in an attempt to return potentials to baseline. If potentials do not improve, the surgeon must attempt to revascularize the territory (eg, by vertebral artery reimplantation, if feasible, or by endarterectomy) to prevent an ischemic insult to the spinal cord. Figure 3 shows the typical appearance of the loss of SSEPs and lower extremity MEPs intraoperatively.

In general, most anesthetic agents act by inhibiting neurological pathways (at synaptic junctions or along axons) or by altering the balance between excitatory and inhibitory neurotransmitters in the central nervous system and the spinal cord. Monitoring of evoked potentials relies on transmission through these same neurological pathways, and thus the mechanism of action of a given agent will determine the extent of potential interference with the monitoring.[101] In general, those agents that directly inhibit synaptic pathways tend to have the most significant effect on neurophysiologic monitoring. However, the different types of evoked potentials differ in their sensitivity to anesthetic agents depending on the neurological pathways involved and the specific anesthetic agents used.[102-105] In general, transcranial MEPs and visual-evoked potentials are extremely sensitive to many of our agents, SSEPs are intermediate in their sensitivity, and brainstem-evoked potentials are not significantly affected.

Evoked potential tracings are described in terms of the amplitude and the frequency of the waveforms. Many commonly used anesthetic agents depress evoked response amplitude and increase latency. As a broad generalization, well-known offenders include potent volatile inhaled agents, nitrous oxide, benzodiazepines, and barbiturates. Opioids have generally been demonstrated to have only mild inhibitory effects on SSEPs and MEPs. Muscle relaxants have a profound inhibitory

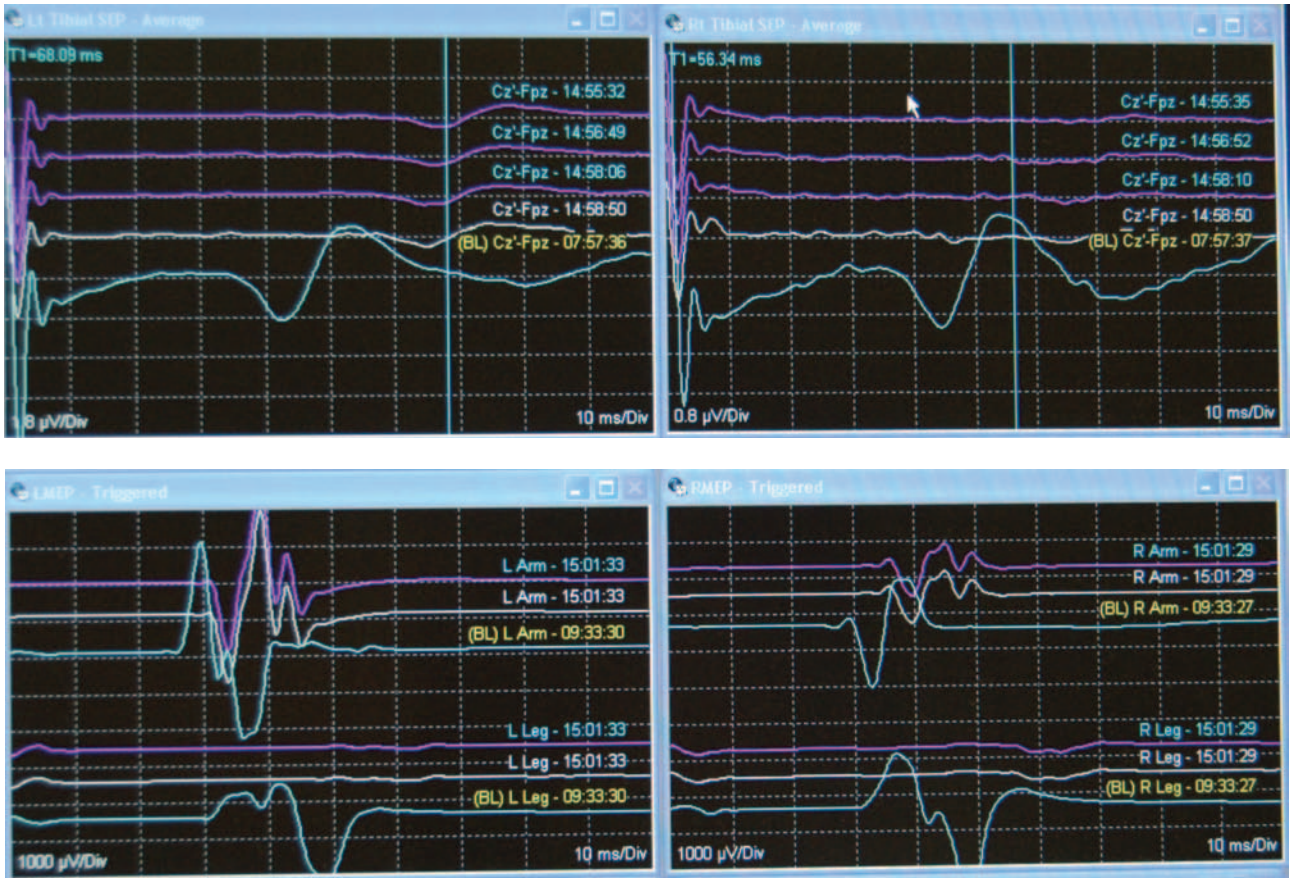

Figure 3. The typical appearance of the loss of intraoperatively monitored SSEPs (top frames) and lower extremity MEPs (bottom frames). By comparison to the baseline tracings from this patient shown in Figures 1 and 2, one can appreciate that there has been nearly a complete loss of the amplitude associated with the potentials, resulting in flat lines. In the bottom frames, one can see that while the potentials have been lost from the lower extremities bilaterally, the tracings from the upper extremities are similar to their respective baselines (see Figure 2). In this particular case, the potentials were lost due to cross clamping of the aorta below the level of the left subclavian artery. Hypothermia may also result in loss of evoked potentials, but the upper extremities would also have been affected if this were the reason in this case. Unilateral loss of potentials would imply an etiology of a surgical nature, such as prolonged limb ischemia.

effect on MEPs, but SSEPs are predictably unaffected. Interestingly, some anesthetic agents (eg, etomidate and ketamine) can enhance SSEP and MEP amplitudes, perhaps by attenuating inhibition.[105] However, dose-dependent effects do exist for our agents and can potentially explain many of the findings published in the literature. For example, despite the potential enhancement of MEP tracings mentioned above, ketamine at high doses has been shown to cause moderate reductions in compound muscle action potential amplitudes.[106]

Equally important with respect to the intrinsic potential interference from our agents are the anatomic locations from which the potentials are stimulated and recorded. For example, propofol appears to cause a significant decrease in the amplitude of cortical SSEPs[107] but it does not cause any significant amplitude depression when the SSEP is recorded in the epidural space.[108] The same has been demonstrated for nitrous oxide,[101] which otherwise causes profound interference with transcranial MEPs. Similarly, opioids, propofol, and thiopental have been demonstrated to suppress myogenic but not neurogenic MEPs in a dose-dependent fashion.[106] In clinical practice, however, it is most common to stimulate the potentials transcranially and record the myogenic responses distally (eg, at the tibialis anterior for MEPs). Although this may be more convenient, it does predispose to potentially increased interference from our agents.

Halothane, enflurane, isoflurane, and nitrous oxide all result in dose-related reductions in the amplitude and increases in the latency of the cortical component

of the SSEP.[109] This effect was most profound with enflurane and least with halothane. Clinically, the authors suggest that 0.5 minimum alveolar concentration (MAC) of isoflurane with nitrous oxide 60% is compatible with intraoperative SSEP monitoring; if nitrous oxide is not used, higher doses of isoflurane may be used. Nitrous oxide appears to be less suppressive of MEP compared with volatile anesthetic agents.[97] Because of the need to use 1-lung ventilation during aortic reconstructions and the need to provide high concentrations of oxygen to prevent hypoxemia, the use of nitrous oxide is limited in these procedures. In comparison to volatile anesthetics and nitrous oxide, clinically relevant anesthetic infusions of propofol (120 $mg \cdot kg^{-1} \cdot min^{-1}$) result in significantly less attenuation of SSEP amplitude with greater levels of depth of anesthesia.[110]

Chen[111] examined isoflurane versus propofol administration at 4 levels of anesthesia (bispectral index range 25-35, 35-45, 45-55, and 55-65). Both propofol and isoflurane depressed SEP amplitudes and increased SEP latencies. Although isoflurane caused a dose-dependent effect, these dose-dependency changes were not observed with propofol administration. With higher doses of isoflurane, a lower percentage of MEP potentials were recordable; at the highest dose, only 6% of the patients had recordable MEP tracings. In contrast, all patients receiving propofol had recordable MEP tracings independent of propofol dose. Increasing propofol doses did not have any effect on MEP latency or amplitude.

"Anesthetic fade" is the reduction of MEP response over the duration of surgery with exposure to general anesthesia. In a retrospective study of 418 patient undergoing spine surgery, Lyon et al[112] identified 62 patients with stable anesthetic depth and hemodynamic conditions. The authors reported a significantly higher voltage threshold for the generation of a 50-μV amplitude signal at the end of the procedure compared with the beginning of the procedure. This increased voltage threshold was directly proportional to the length of anesthetic exposure. The recognition of this phenomenon is important in the interpretation of MEP responses to avoid false positive findings.

The perioperative use of SSEP may identify critical spinal cord ischemia. SSEP monitoring influenced the surgical strategy in 17 of 63 patients (27%) undergoing descending aortic reconstruction.[113] Partial cardiac bypass was initiated in 1 patient, critical intercostals arteries or distal clamp was reimplanted in 13 patients, and the proximal clamp was repositioned in 3 patients with left carotid ischemia. The authors reported no cases of unexplained SSEP abnormalities. New immediate paraplegia was observed in 1 patient with sustained SSEP absence, and 2 patients presented with delayed paraplegia with normal perioperative SSEP.

Lips et al[114] used a porcine model to define the relationship between the duration of MEP signal loss and resultant spinal cord injury during lumbar spinal segmental artery occlusion. If spinal cord blood flow was reestablished after 10 minutes of loss of MEP, there was no resultant spinal cord functional or histopathological injury. MEP loss of greater than 10 minutes was associated with histopathological injury and loss greater than 40 minutes was associated with functional impairment as well.

To demonstrate the greater sensitivity of MEP than SEP monitoring, Dong et al[65] reported their experience with 56 patients undergoing during descending aortic reconstruction. All patients were monitored with both MEP and SEP. Sixteen patients (29%) had MEP evidence of perioperative spinal cord ischemia compared with 4 patients (7%) with SEP changes. These changes were reversed in 13 patients with either segmental artery reimplantation or opimization of hemodynamics. Although none of these 13 patients awoke with immediate paraplegia, the remaining 3 patients awoke paraplegic. All 3 patients had normal perioperative SEP.

Conclusion

Management of patients undergoing repair of descending thoracic aortic aneurysm is very challenging from an anesthetic standpoint. The level of monitoring, hemodynamic 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. It is likely that current perioperative care will continue to evolve in the future given that clinical and laboratory data are constantly influencing clinical practice.

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