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Anesthesia for Vascular Surgery

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PERIPHERAL VASCULAR DISEASE

Atherosclerosis is the most common cause of peripheral vascular occlusive disease. This degenerative process involves the formation of atheromatous plaques that may obstruct the vessel lumen resulting in a reduction in distal blood flow. The pathophysiologic process is systemic, progressive, and primarily affects the arteries due to plaque formation, which can lead to stenosis and potentially occlusion of the vascular lumen; thrombosis from hypercoagulability, resulting in acute organ ischemia; embolism from microthrombi or atheromatous debris; and weakening of the arterial wall, resulting in aneurysm formation. Atherosclerosis is an inflammatory condition that is partially caused by cholesterol plaques, which occur within arteries. In response to cholesterol plaques, immune cells such as macrophages and monocytes liberate proinflammatory cytokines, which leads to a progressive increase in the size of plaques. The lipid cap that envelopes the plaque can rupture, resulting in intraluminal thrombosis or plaque emboli. The most common risk factors associated with atherosclerosis are shown in Box 28.1. Endothelial dysfunction is a potential cause of increased hemodynamic variability during anesthesia. Smoking, elevated proinflammatory mediators, and diabetes mellitus are major risk factors in the pathogenesis of atherosclerosis in the arterial tree. Typical symptoms associated with peripheral occlusive disease include claudication, skin ulcerations, gangrene, and impotence.[1] The extent of disability is primarily influenced by the development of collateral blood flow. The mortality rate in patients with vascular disease is two- to sixfold higher than in the general population.[2] Hypercoagulability resulting from platelet interaction with leukocytes and other cells that modulate the immune response plays a major role in the development of atherosclerosis.[3,4] Researchers have discovered heritable genetic factors that predispose patients to developing vascular disease.[5]

Treatment for peripheral occlusive disease may range from pharmacologic therapy to surgery. Surgical therapy includes transluminal angioplasty, endarterectomy, thrombectomy, endovascular stenting, and arterial bypass. Some common surgical maneuvers used to bypass occlusive lesions are aortofemoral, axillofemoral, femorofemoral, and femoropopliteal procedures. Bypass techniques may be classified as inflow or outflow procedures depending on the level of the obstruction, with the dividing axis occurring at the level of the groin. Temporary occlusion of the operative artery is mandatory during surgical bypass, as this temporarily further reduces blood flow and oxygen delivery. The development of collateral circulation provides alternative vascular blood flow in patients with occlusive disease.[6,7] Initially, angiogenesis or the development of new vessels supplies collateral blood flow that is sufficient to meet tissue oxygen demands. As the disease progresses, the blood flow is decreased, and the oxygen supply is unable to meet the tissues' demand, which could result in myocardial dysfunction, neurologic dysfunction, renal dysfunction, and/or limb ischemia.

Preoperative Evaluation

The atherosclerotic process in occlusive disease is not limited to peripheral arteries and should be expected to be present in the coronary, cerebral, and renal arteries. More than half of the mortality associated with peripheral vascular disease results from adverse cardiac events.[8] There is a clear association between the development of aortic aneurysms and coronary artery disease (CAD).[9] It has been estimated that 42% of patients presenting for abdominal aortic aneurysm (AAA) repair have significant CAD.[10] Preoperative cardiovascular evaluation and treatment are beneficial for reducing not only perioperative risk but also late cardiovascular events. After elective AAA repair, the 5-year survival rate and incidence of major adverse cardiovascular events is 86%.[11] Cardiac pathology, which often occurs in this patient population, must be managed aggressively to optimize cardiac functioning and decrease morbidity and mortality from cardiac causes.

Preoperative Pharmacologic Management

β-blockade. The advantages of β-blockade, as it relates to factors that affect myocardial oxygen supply and demand, have been extensively studied in patients with peripheral vascular disease. The use of β-blockers is recommended in patients at high risk for myocardial ischemia and infarction.[12] For patients having an AAA repair, there is a 10-fold decrease in cardiac morbidity associated with adequate preoperative β-blockade.[13] β-blockade therapy should be instituted days to weeks before surgery and titrated to a target heart rate between 50 and 60 beats per minute (bpm).[14] Perioperative β-blockade started within 1 day or less before noncardiac surgery prevents nonfatal myocardial infarctions (MIs) but increases the risk of hypotension, bradycardia, stroke, and death. Initiating β-blockade between 2 and 7 days before surgery may be preferable, but there is a lack of scientific data to support the benefit of beginning therapy more than a month in advance.[15] Vascular surgery patients with limited heart rate variability after receiving β-blockers exhibit less cardiac ischemia and decreased postoperative troponin values and have a decreased mortality from all causes 2 years postoperatively.[16] For patients taking β-blockers, these medications should be continued up to the day of surgery and during the postoperative period.[17]

Statins. It is suggested that statins decrease perioperative mortality in patients with vascular disease by decreasing adverse cardiovascular and cerebrovascular events and death.[18] These drugs have cardioprotective effects, as they reduce vascular inflammation, decrease the incidence of thrombogenesis, enhance nitric oxide bioavailability, stabilize atherosclerotic plaques, and lower lipid concentrations. It is reasonable to start a statin drug in this patient population. If prescribed, a statin should be instituted 30 days prior to the surgical procedure and continued throughout the postoperative period.[15] Statin therapy that is started preoperatively and continued through discharge has been associated with reduced 30-day mortality and an absolute 18% improvement in

BOX 28.1 Risk Factors Related to the Development of Atherosclerotic Lesions

- Advanced age
- Smoking
- Hypertension
- Diabetes mellitus
- Insulin resistance
- Obesity
- Family history and genetic predisposition
- Physical inactivity
- Gender (males at greater risk than females)
- Hyper/hypohomocysteinemia
- Elevated C-reactive protein
- Elevated lipoprotein
- Hypertriglyceridemia
- Hyperlipidemia

5-year survival after vascular surgery.[18,19] Specifically, patients who were prescribed statins and then underwent endovascular aortic aneurysm repair (EVAR) had greater residual aneurysm sac regression within the first years postoperatively.[20]

Antiplatelet medications. It has been unclear whether patients having noncardiac surgery who are at increased risk for MI should receive aspirin throughout the perioperative period. The results of current evidence and the Perioperative Ischemic Evaluation 2 (POISE-2) research trial indicate that perioperative aspirin does not prevent MI and does not alter the risk of a perioperative cardiovascular event.[21] The outcomes were unchanged for those subjects who took aspirin for a prolonged period compared to those who started aspirin prior to surgery. Aspirin did, however, increase the risk of major bleeding. In patients who have been on a long-term aspirin regimen and have aspirin withheld during the perioperative period, it is important to ensure that aspirin is restarted after the increased risk period for bleeding has passed (i.e., 8–10 days after surgery). On the basis of currently available literature, aspirin should not be administered to patients undergoing surgery unless there is a definitive guideline-based primary or secondary prevention indicated.[22,23]

The presence of concurrent pulmonary, renal, neurologic, and endocrine dysfunction should be identified, and measures should be taken to improve organ function before surgery. Acute kidney injury (AKI) is common during the perioperative period in patients undergoing vascular surgery, and this condition is associated with a high risk for cardiovascular-specific mortality comparable to that seen with chronic kidney disease.[24] Strategies used to help prevent perioperative AKI are discussed later in this chapter. Preoperatively, the greater the number of comorbidities that exist, the greater the risk of morbidity and mortality during the perioperative period.

Monitoring

The extent of perioperative monitoring should be based on the presence of coexisting disease and the type of surgery. The detection of myocardial ischemia should be a primary objective in patients with vascular disease. Methods for assessing cardiac function include electrocardiography (ECG), pulmonary artery pressure, and transesophageal echocardiography (TEE) monitoring. The effectiveness of pulmonary artery catheters (PACs) in improving patient outcomes remains controversial. Many randomized controlled trials have been performed to assess whether they offer any benefit. It was determined that PAC monitoring had no effect on mortality or length of hospital stay. Additionally, there were higher rates of pulmonary embolism, pulmonary infarction, and hemorrhage in the PAC group.[25-27] Furthermore, PACs have not been associated with decreased intraoperative mortality or morbidity, and they are associated with increases in the duration of ventilation and length of stay (LOS) in the intensive care unit (ICU) following cardiac surgical procedures.[26] Specific indications for the use of PAC may be indicated (e.g., complex cardiac surgery).[28]

Due to the global nature of atherosclerotic disease, some degree of systemic cardiovascular disease in patients with peripheral vascular disease should be assumed.[10] Patients with hypertension and/or angiopathology rely on increased mean arterial pressures (MAPs) to perfuse their vital organs. Thus cerebral and coronary autoregulation occur within a higher range compared to patients without peripheral occlusive vascular disease (60–140 mm Hg). Short, sustained periods of hypotension can result in cardiac or neurologic ischemia. Direct intraarterial blood pressure monitoring allows for near–real-time determination of blood pressure values. Information ascertained from an arterial line, such as fluid volume status, acute fluctuations in blood pressure caused by surgical intervention, and titration of vasopressor/vasodilator medications, helps to guide treatment decisions.

In the future, the use of noninvasive hemodynamic monitoring modalities may prove to decrease morbidity and mortality during anesthesia care by allowing the anesthetist to make pharmacologic choices based on cardiac output and myocardial oxygen supply and demand. Guided titration of vasoactive medications along with patient-specific, goal-directed intravenous fluid therapy could improve survivability in this patient population. More scientific evidence will be needed on these interventions prior to making practice guideline recommendations.

Anesthetic Selection

The anesthetic technique chosen for patients having vascular surgery depends on the type of surgical procedure to be performed and the presence of coexisting disease. Maintaining consistent hemodynamic control and avoiding significant episodes of intraoperative hypertension and hypotension are vital to (1) maintaining oxygen delivery to vital organs, (2) decreasing the possibility of increased myocardial oxygen consumption, and (3) decreasing the potential for hemorrhagic stroke. In certain instances, infiltration of local anesthetic and intravenous sedation may be sufficient, whereas more invasive surgical procedures require the use of general anesthesia. Regional anesthesia for surgery on the lower extremities may decrease the overall morbidity and mortality associated with this patient population. Numerous studies have failed to yield demonstrative evidence that any single anesthetic technique decreases morbidity and mortality following vascular surgery. A comprehensive meta-analysis combining data from 141 studies involving 9559 patients suggested a 30% reduction in mortality for those patients who received a combined general anesthetic (GA) and epidural combination. A reduction in the rate of MI, stroke, and respiratory failure was found when epidural anesthesia was used in patients undergoing aortic surgery.[29] A major study has been conducted to evaluate various end points associated with major vascular surgery.[30] None of these studies have definitively concluded that superior outcomes depend on the anesthetic technique used.[31] A positive consideration for administering inhalation and intravenous anesthetic agents is that anesthetic medications decrease the rate of oxygen demand and help to protect neurologic and cardiac tissue in patients having noncardiac surgery.[32] In addition, a meta-analysis reviewing epidural analgesia versus opioids for postoperative pain relief in patients undergoing abdominal aortic surgery showed an overall decreased rate of MI in those patients who received epidural analgesia.[33] Epidural analgesia provided during the postoperative period has significant physiologic advantages. Specific benefits of using an epidural for major abdominal vascular surgery are summarized in Box 28.2. In addition, many patients having vascular surgery are

BOX 28.2 Benefits of the Epidural Technique in Vascular Surgery

Endocrine
- Inhibits surgical stress response
- Inhibits epinephrine and cortisol release
- Inhibits hyperglycemia
- Inhibits lymphopenia and granulocytosis
- Causes nitrogen sparing
- Blocks sympathetic tone
- Inhibits inflammatory mediator release

Cardiovascular
- Decreases myocardial oxygen demand and afterload
- Increases endocardial perfusion at ischemic zone
- Increases hemodynamic stability
- Decreases blood loss
- Decreases general anesthetic medication requirements
- Redistributes blood to lower extremities

Pulmonary
- Decreased effect on FVC, FEV_1, and PEFR
- Decreases ventilation perfusion mismatch
- Improves atrioventricular oxygen differentiation
- Decreases pulmonary postoperative complications
- Decreases incidence of thromboembolism

Renal
- Increases blood flow in the renal cortex
- Decreases renovascular constriction

Geriatric
- Inhibits physiologic stress
- Improves postoperative mental status

Miscellaneous
- Allows earlier extubation, ambulation, and discharge
- Improves postoperative pain control

FEV_1, Forced expiratory volume in 1 sec; *FVC,* forced vital capacity; *PEFR,* peak expiratory flow rate.

receiving anticoagulant therapy and will receive heparin intraoperatively; therefore there is a remote risk that neuraxial anesthesia could lead to epidural hematoma formation.[34]

Postoperative Considerations

Postoperative pain management is important to consider after peripheral vascular surgery. Most clinicians agree that postoperative administration of narcotics not only provides patient comfort but also contributes to cardiac stability. The perioperative use of dexmedetomidine is also advantageous due to its sympatholytic effects, analgesic properties, and minimal effects on respiration. The use of epidural opioids and local anesthetics in patients recovering from vascular surgery is an important component of postoperative care because pain can greatly enhance sympathetic nervous system stimulation. Despite a decrease in discomfort during the postoperative course, these patients must be monitored for possible adverse events, such as MI, hypotension, or respiratory depression, which could be attributed to the administration of epidural opioids and local anesthetics. Acute pain increases inflammatory mediators such as creatinine kinase, C-reactive protein, interleukin-6 (IL-6), and tumor necrosis factor, which can lead to regional blood flow alterations, organ dysfunction, and cell death.[35]

BOX 28.3 Conditions and Traits Associated With Development of Abdominal Aortic Aneurysm

- Smoking
- Older age
- Gender (more common in males than in females)
- Family history
- Coronary artery disease
- High cholesterol
- Chronic obstructive pulmonary disease
- Height (per 7-cm interval)
- Hypertension
- Peripheral vascular occlusive disease
- Whites

Patients having vascular surgery are at increased risk for developing a venous thromboembolism (VTE) during the postoperative period. In one study, VTE was detected in 18.1% of patients with aortoiliac obstruction and 21% of patients after AAA repair.[36] The incidence of VTE continued to be elevated after discharge. All methods intended to prevent the formation of deep vein thrombosis (DVT), including pharmacologic management, should be employed throughout the postoperative period. Low-molecular-weight heparin is frequently used to bridge the time between withholding oral anticoagulants and surgery. It is important to restart oral anticoagulant medications postoperatively after the risk of bleeding is decreased to minimize DVT and VTE. Increased postoperative hematocrit concentration is associated with an increased risk of 30-day mortality from DVT and pulmonary embolism.[37]

ABDOMINAL AORTIC ANEURYSMS

Incidence

The incidence of AAA is estimated to be between 3% and 10% for patients over 50 years of age in the Western world.[38] Improved detection of AAAs is the result of increased screening of asymptomatic aneurysms by noninvasive diagnostic modalities such as computed tomography (CT), magnetic resonance imaging (MRI), and ultrasonography. The occurrence of AAAs has increased because of the increased age of the general population and the vascular changes that occur as a result of aging.[39] AAAs are two to six times more common in men than in women, and are two to three times more common in white men than in black men.[38] Women with AAAs are being treated at older ages and typically have AAAs that are smaller in diameter, as compared to men.[40] In men, AAAs most frequently begin to occur at 50 years of age and peak at 80 years of age.[41]

Risk Factors

The incidence of AAAs in a given population depends on the presence of risk factors (Box 28.3). Independent risk factors thought to be causes rather than markers for the development of an AAA include age, gender, and smoking. Smoking is the risk factor that is most highly correlated with AAA. In cigarette smokers, the incidence of AAAs increases fivefold.[38] There is an association between chronic inflammation and angiopathology. The specific mechanism that links inflammation and vascular disease has not been definitively established, but elevated cytokine levels appear to play a central role.[42]

Mortality

Elective AAA repair is one of the most frequent vascular surgical procedures, with approximately 40,000 operations performed in the

United States annually.[43] Mortality rates for elective abdominal aortic aneurysmectomies have decreased since the 1970s. The present mortality rate ranges from 1% to 11%, although it is most commonly estimated at 5%. This is compared with mortality rates of 18% to 30% in the 1950s.[38,44-47] Even with the advent and increased frequency of EVAR, long-term mortality rates are similar, at approximately 15% to 17%.[48] Advanced detection, earlier surgical intervention, extensive preoperative preparation, refined surgical techniques, higher quality hemodynamic monitoring, and improved anesthetic techniques have all contributed to this improvement in surgical outcomes. Data suggest a low risk of rupture for AAAs less than 4 cm in diameter; however, the risk dramatically increases for AAAs with a 5-cm diameter or greater. Surgical intervention is recommended for AAAs 5.5 cm or greater in diameter.[49] Unfortunately, mortality rates for patients with undetected or untreated ruptured aortic aneurysms have not followed the trend of those who have surgical intervention. Estimates of mortality resulting from ruptured AAAs vary from 35% to 94%.[43,50-52] Combining prehospital mortality with operative mortality, the overall mortality for AAA rupture is 80% to 90%. The 5-year mortality rate for individuals with untreated AAAs is 81%, and the 10-year mortality rate is 100%.[53] Other criteria for surgical intervention for AAA include ruptured AAA, a 4- to 5-cm AAA with greater than 0.5-cm enlargement in less than 6 months, patients who are symptomatic for AAA, and a 5-cm AAA or greater for elective repair for patients with a reasonable life expectancy. Early detection and elective surgical intervention can be lifesaving because elective surgical mortality is less than 5% in most studies.[54]

Diagnosis

Physical Examination

Asymptomatic aneurysms may be detected during routine examination as a pulsatile abdominal mass. Smaller aneurysms are often undetected on routine physical examination. AAA screening rates remain below 50%. AAAs are frequently discovered incidentally by primary practitioners, and some patients undergo unnecessary ultrasound screening.[55] It has been estimated that less than 30% of AAAs are identified during routine physical examination. A more extensive scoring system that includes additional risk factors such as the presence of carotid artery or peripheral arterial disease, obesity, hypertension, smoking, diabetes, and advanced age may increase the rate of detection to almost 90% of AAAs.[56]

Imaging

A minimally invasive method used to initially diagnose the presence of AAA is by ultrasound. Ultrasound is helpful to determine if an AAA is present, but it is not highly accurate in determining the extent of the AAA or if rupture has occurred.[57] CT angiography (CTA) allows for a more precise view of the aneurysm morphology, including aneurysm size, vessel wall integrity, and adjacent anatomic definition such as the iliac arteries. CTA has become the imaging test of choice for AAA because of its high-quality resolution, rapid image acquisition, and wide availability.[58] The information gained from CTA is valuable to the surgeon and interventional radiologist for initial determination of the surgical intervention (e.g., open or EVAR) and the extent of the distal and proximal aneurysm if an endovascular stent graft is to be implanted.

ABDOMINAL AORTIC RECONSTRUCTION

Patient Selection

As a result of recent advances in surgical and anesthetic techniques, the 30-day perioperative mortality rate associated with elective open repair of AAAs is estimated to be 3% to 4.5%.[59] Most patients with abdominal aneurysms, including the elderly, are considered surgical candidates. Although advanced age contributes to an increased incidence of morbidity and mortality, age alone is not a contraindication to elective aneurysmectomy.[60] However, physiologic age is more indicative of increased surgical risk than chronologic age. Contraindications to elective repair include intractable angina pectoris, recent MI, severe pulmonary dysfunction, and chronic renal insufficiency.[61] Patients with stable CAD and coronary artery stenosis of greater than 70% who require nonemergent AAA repair do not benefit from revascularization if β-blockade has been established.[44] Table 28.1 lists characteristics that define high-risk patients; however, in most cases, the presence of an AAA warrants surgical intervention.[53]

TABLE 28.1 Criteria for High Risk in Abdominal Aortic Aneurysm Repair

Parameter	Criterion
Age	>70 yr
Gender	Female
Cardiac	History of myocardial infarction Angina pectoris Myocardial disease Q waves on ECG ST/T wave changes on ECG Ventricular ectopy Hypertension with left ventricular hypertrophy Congestive heart failure
Endocrine	Diabetes
Neurologic	Stroke
Renal	Chronic or acute renal failure
Pulmonary	Chronic obstructive pulmonary disease Emphysema Dyspnea Previous pulmonary surgery

ECG, Electrocardiogram.

Modified from Pairolero PC. Repair of abdominal aortic aneurysms in high-risk patients. *Surg Clin North Am.* 1989;69:765–774; Holt PJE, Thompson MM. Abdominal aortic aneurysms: evaluation and decision making. In Cronenwett JL, Johnston W, eds. *Rutherford's Vascular Surgery.* 8th ed. Vol. 2. Philadelphia: Elsevier; 2014; Woo EY, Damraur SM. Abdominal aortic aneurysms: open surgical treatment. In Cronenwett JL, Johnston W, eds. *Rutherford's Vascular Surgery.* 8th ed. Vol. 2. Philadelphia: Elsevier; 2014.

The dimensions of an aneurysm can change over time. AAAs expand by approximately 4 mm/yr.[62] Aneurysmal vessel dimensions correspond to the law of Laplace:

$$T = P \times r$$

where T = wall tension, P = transmural pressure, and r = vessel radius.

As the radius of a vessel increases, the wall tension increases. Wall tension is directly proportional to the vessel radius and intraluminal pressure and inversely proportional to wall thickness. Therefore the larger the aneurysm, the higher the likelihood of spontaneous rupture.

As previously stated, aneurysms measuring more than 5 cm in diameter generally require surgical intervention, but aneurysms measuring less than 5 cm should not be considered benign, and monitoring of the condition is indicated.[44] An aneurysm has the potential to rupture regardless of its size. As the diameter of the aneurysm increases in size, the risk of rupture increases (Table 28.2).[1] In contradiction to the current thought that increased wall shear stress increases the risk of

TABLE 28.2 Range of Potential Rupture Rates for a Given Size of Abdominal Aortic Aneurysm

AAA Diameter (cm)	Rupture Risk (%/yr)
<4	0
4–5	0.5–5
5–6	3–15
6–7	10–20
7–8	20–40
>8	30–50

AAA, Abdominal aortic aneurysm.
From Brewster DC, et al. Guidelines for the treatment of abdominal aortic aneurysms: report of a subcommittee of the Joint Council of the American Association for Vascular Surgery and Society for Vascular Surgery. *J Vasc Surg.* 2003;37:1106–1117.

aortic rupture, it has been shown that aortic rupture may occur more often at sites with low wall shear stress due to blood flow recirculation resulting in thrombus deposition, aortic wall degeneration, and eventual rupture.[63] Due to increased wall stress at the bifurcation of the aorta and the iliac arteries, AAAs most frequently develop in the infrarenal aorta, although approximately 5% to 15% involve the suprarenal aorta. It is estimated that approximately 40% of AAAs also involve the iliac arteries.[1]

Patient Preparation

Perioperative MI is the most common reason for poor outcomes in noncardiac surgery for patients with vascular disease. Optimization of myocardial oxygen supply and demand and modification of cardiac risk factors are the major goals of preoperative risk reduction. β-blockers and statins are the important preoperative pharmacologic treatments for medical management.[17,19] Prophylactic coronary revascularization does not reduce the incidence of perioperative cardiac events.[64] Preoperative cardiac testing is recommended only if interpretation of the results will change anesthetic management.[15,65,66]

Preoperative fluid loading and restoration of intravascular volume are perhaps the most important techniques used to enhance cardiac function during abdominal aortic aneurysmectomies. Reliable venous access must be secured if volume replacement is to be accomplished. Large-bore intravenous lines and central lines can be used to infuse fluids or blood. Massive hemorrhage is an ever-present threat, therefore the availability of blood and blood products should be ensured. Provisions for rapid transfusion and intraoperative blood salvage should be confirmed.

Routine Monitoring

Standard monitoring methods include ECG (with display of lead II for detection of dysrhythmias and the precordial V_5 lead for analysis of ischemic ST segment changes), pulse oximetry, and capnography. An esophageal stethoscope allows for continuous auscultation of heart and breath sounds, as well as temperature monitoring. Placement of an indwelling urinary catheter is necessary for continuous measurement of urinary output and renal function. Neuromuscular function should be routinely monitored.

Invasive Monitoring

Maintaining cardiac function is crucial for a successful surgical outcome. Cardiac function should be closely monitored during abdominal aortic reconstruction. Invasive blood pressure monitoring permits beat-to-beat analysis of the blood pressure, immediate identification of hemodynamic alterations related to aortic clamping, and access for blood sampling. However, information obtained from PACs has been shown to have low sensitivity and low specificity in detecting myocardial ischemia when compared with ECG and TEE. As previously discussed, PACs are not routinely indicated unless a specific purpose warrants their use.[26]

By detecting changes in ventricular wall motion, TEE provides a sensitive method for assessing ventricular wall motion abnormalities. TEE is a primary method of intraoperative cardiac assessment in patients undergoing surgery on the heart and the aorta.[65,67] Wall motion abnormalities also occur much sooner than electrocardiographic changes during periods of reduced coronary blood flow.[52] When TEE is used to guide intraoperative hemodynamic management, patients with left ventricular diastolic dysfunction have a decreased incidence of developing congestive heart failure and atrial fibrillation.[68,69] The most common abnormalities detected by intraoperative TEE include hypovolemia, low ejection fraction, right ventricular failure, segmental wall motion abnormalities, and pulmonary embolus.[70] Myocardial ischemia poses the greatest risk of mortality after abdominal aortic reconstruction. Intraoperative monitoring may enable earlier detection and intervention during ischemic cardiac events.

Aortic Cross-Clamping

Abdominal aortic reconstruction is one of the most challenging situations for the anesthetist due to the frequency and degree of hemodynamic variability during cross-clamping and unclamping of the aorta. This is further complicated by the fact that most patients having an aortic aneurysm repair are elderly and have varying degrees of coexisting disease. Perhaps the most dramatic physiologic change occurs with the application of an aortic cross-clamp. Temporary aortic occlusion produces various hemodynamic and metabolic alterations.

Hemodynamic Alterations

The hemodynamic effects of aortic cross-clamping are affected by the clamp application site along the aorta, the patient's preoperative cardiac reserve, and the patient's intravascular volume. The most common site for cross-clamping is infrarenal because most aneurysms appear below the level of the renal arteries. Less common sites of aneurysm development are the juxtarenal and suprarenal areas.

During aortic cross-clamping, hypertension occurs above the cross-clamp, and hypotension occurs below the cross-clamp. Aortic cross-clamping increases plasma levels of catecholamines, aldosterone, cortisol, prostaglandins, and other stress hormones that are associated with a sympathetic nervous system response. There is an absence of blood flow distal to the cross-clamp in the pelvis and lower extremities.[6] An increase in afterload causes the left ventricular myocardial wall tension to increase, which in turn increases myocardial oxygen demand. Patients with poor left ventricular function are at risk for developing congestive heart failure during this period. MAP and systemic vascular resistance (SVR) also increase. Cardiac output may decrease or remain unchanged. Pulmonary artery occlusion pressure (PAOP) may increase or remain unchanged. Table 28.3 summarizes the physiologic changes associated with aortic cross-clamping.

Patients with adequate cardiac reserve commonly adjust to sudden increases in afterload without the occurrence of adverse cardiac events. However, patients with ischemic heart disease or ventricular dysfunction are unable to fully compensate as a result of the hemodynamic alterations. The increased left ventricular wall stress attributed to aortic cross-clamp application may contribute to decreased global ventricular function and myocardial ischemia. Clinically, these patients experience increases in PAOP in response to aortic cross-clamping. Aggressive

TABLE 28.3 The Physiologic Changes Associated With Aortic Cross-Clamping

Hemodynamic Changes	Metabolic Changes	Intraoperative Interventions
Increased arterial blood pressure above the clamp	Decreased total body oxygen consumption	*Reduce Afterload* Sodium nitroprusside Inhalation anesthetics Milrinone Shunts and aorta to femoral bypass
Decreased arterial blood pressure below the clamp	Decreased total body carbon dioxide production	*Reduce Preload* Nitroglycerin Atrial to femoral bypass
Increased wall motion abnormalities and left ventricular wall tension	Increased mixed venous oxygen saturation	*Renal Protection* Fluid administration Mannitol Furosemide Dopamine N-acetylcysteine Renal cold perfusion
Decreased ejection fraction and cardiac output	Decreased total body oxygen extraction	*Miscellaneous* Hypothermia Decrease minute ventilation Sodium bicarbonate
Decreased renal blood flow	Increased catecholamine release	
Increased pulmonary occlusion pressure	Respiratory alkalosis	
Increased central venous pressure	Metabolic acidosis	
Increased coronary blood flow		

Adapted from Norris EJ. Anesthesia for vascular surgery. In Miller RD, et al., eds. *Miller's Anesthesia*. 9th ed. Philadelphia: Elsevier; 2020; Holt PJE, Thompson MM. Abdominal aortic aneurysms: evaluation and decision making. In Cronenwett JL, Johnston W, eds. *Rutherford's Vascular Surgery*. 9th ed. Vol. 2. Philadelphia: Elsevier; 2019.

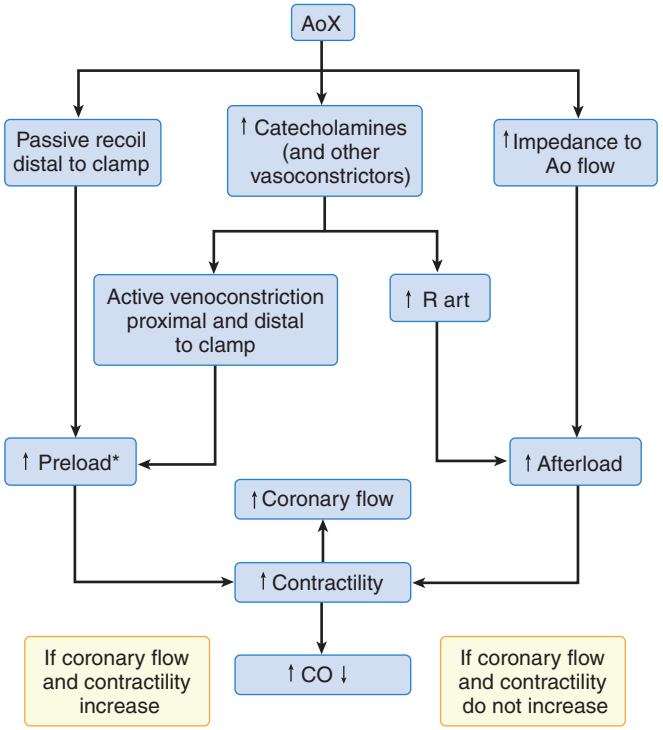

Fig. 28.1 Systemic hemodynamic response to aortic cross-clamping. Preload *(asterisk)* does not necessarily increase with infrarenal clamping. Depending on splanchnic vascular tone, blood volume can be shifted into the splanchnic circulation, and preload will not increase. *Ao,* Aortic; *AoX,* aortic cross-clamping; *R art,* arterial resistance. (Adapted from Gelman S. The pathophysiology of aortic cross-clamping. *Anesthesiology.* 1995;82:1026-1060.)

pharmacologic intervention is required for restoration of cardiac function during this time. An algorithm that depicts the systemic hemodynamic responses to aortic cross-clamping is shown in Fig. 28.1.

Metabolic Alterations

After application of an aortic cross-clamp, the lack of blood flow to distal structures results in a hypoxic and ischemic environment. In response to tissue ischemia, metabolites such as cytokines, prostaglandins, nitric oxide, and arachidonic acid are formed and released into circulation. Furthermore, anaerobic metabolism leads to the accumulation of serum lactate. The release of arachidonic acid derivatives may be a contributing factor leading to cardiac instability and myocardial depression during aortic cross-clamping.[38] Thromboxane A_2 synthesis, which is accelerated by the application of an aortic cross-clamp, may be responsible for the decrease in myocardial contractility and cardiac output that occurs.

Traction on the mesentery is a surgical maneuver used for exposing the aorta. Mesenteric traction syndrome is associated with this procedure. Decreases in blood pressure and SVR, tachycardia, increased cardiac output, and facial flushing are common responses to mesenteric traction. Although the cause of this syndrome is unknown, it has been associated with high concentrations of 6-keto-prostaglandin F1α, a stable metabolite of prostacyclin, at the time of mesenteric traction.[71] The 6-keto-prostaglandin F1α levels and hemodynamic stability return to preclamp values as reperfusion occurs.

The neuroendocrine response to major surgical stress is believed to be mediated by cytokines such as IL-1B, IL-6, and tumor necrosis factor, as well as plasma catecholamines and cortisol.[72] These mediators are thought to be responsible for triggering the inflammatory response that results in increased body temperature, leukocytosis, tachycardia, tachypnea, and fluid sequestration. Patients who have an exaggerated plasma stress mediator release have longer operative and cross-clamp times and require a greater number of blood transfusions.

Effects on Regional Circulation

Acute kidney injury. Hypoperfusion to tissues that are distal to the aortic clamp occurs. Renal insufficiency and acute renal failure are severe complications associated with abdominal aortic reconstruction. Suprarenal and juxtarenal cross-clamping are associated with a higher incidence of altered renal dynamics and can decrease renal blood flow by as much as 80%. However, significant reductions in renal blood flow occur even when aortic cross-clamping is performed below the renal arteries. Infrarenal aortic cross-clamping is associated with a 40% decrease in renal blood flow.[38] Thus renal insufficiency more commonly occurs with suprarenal as compared to infrarenal cross-clamping. AKI may occur in as many as 18% of patients undergoing aortic aneurysm repair. Preoperative evaluation of renal function is the best method of assessing and anticipating which patients may develop postoperative renal dysfunction. Preexisting renal impairment appears

to be the main factor associated with AKI. A complete evaluation of renal function is required during the preoperative period, and patients with a low glomerular filtration rate should be managed with more aggressive renal protection interventions.[73]

Suprarenal cross-clamp times longer than 30 minutes increase the risk of postoperative renal failure. Even though renal blood flow is restored after unclamping, prolonged effects associated with ischemic reperfusion injury (IRI) occur. The injury caused to the renal tubular epithelium decreases the glomerular filtration rate. This effect may lead to acute renal failure, which is fatal in 50% to 90% of patients who have undergone aneurysmectomy.[74] Clamp positioning above the renal arteries is predictive of severe AKI in patients treated with open surgical repair (OSR).[75] AKI is a common problem after elective infrarenal EVAR, and preoperative renal dysfunction appears to be the main factor associated with AKI. AKI is associated with higher mortality rates and long-term cardiovascular events after surgery.[75,76] The administration of renal-dose dopamine, mannitol, sodium bicarbonate, and/or loop diuretics has not been scientifically proven to preserve or improve renal function postoperatively. The use of balanced crystalloid solutions decreases the incidence of AKI.[77] Minimizing the use of nephrotoxic medications such as nonsteroidal antiinflammatory drugs and aminoglycoside antibiotics preoperatively is prudent. Intraoperative renal perfusion with cold solution appears to have a renal protective effect and decrease the incidence of AKI.[78] Atrial natriuretic peptide (ANP) causes vasodilatation of the preglomerular artery, inhibition of the angiotensin axis, and prostaglandin release, which promotes renal vascular dilation. During the AKI reflow period, the natriuretic effect of ANP could be useful in preventing tubular obstruction in patients undergoing major surgery such as cardiovascular surgery.[77] The most important interventions to protect from AKI are aggressive hemodynamic stabilization and minimization of aortic clamp times, which have proven efficacy.[79]

Spinal cord ischemia. Spinal cord damage causing paraplegia can occur during aortic occlusion. The incidence of paraplegia during thoracic and thoracoabdominal aneurysm repair is estimated to be between 1% and 13%.[80] Longitudinal blood flow to the spinal cord includes (1) two posterior and two posterolateral arteries supplying blood to the dorsal or sensory portion of the spinal cord (20% of spinal cord blood flow) and (2) one anterior spinal artery supplying blood to the anterior or motor portion of the spinal cord (80% of spinal cord blood flow). Transverse blood flow originating from the aorta is via the greater radicular artery (artery of Adamkiewicz). The exact location of this artery is variable and depends on an individual's specific anatomic characteristics. The artery most often originates between spinal segments T8 and T12, but it can originate as low as L2 in a small segment of the population. This explains why the presence of paraplegia with aortic cross-clamping is unpredictable. Interruption of blood flow to the greater radicular artery in the absence of collateral blood flow has been identified as a factor that can cause paraplegia in patients having AAA repair. The incidence of neurologic complications increases as the aortic cross-clamp is positioned higher or more proximal to the heart. Somatosensory-evoked potential (SSEP) monitoring has been advocated as a method of identifying spinal cord ischemia. However, SSEP monitoring reflects dorsal (sensory) spinal cord function and does not provide information regarding the integrity of the anterior (motor) spinal cord.[6] Motor-evoked potential (MEP) monitoring is capable of determining anterior cord function. This monitoring modality relies on intact neuromuscular functioning for analysis, which limits its use in abdominal aortic aneurysmectomies because neuromuscular blocking drugs are routinely used. Alternative methods for reliable evaluation of spinal cord ischemia are still under investigation.[81]

Spinal cord protection strategies include distal aortic perfusion, cerebrospinal fluid drainage, and mild hypothermia. Maintenance of normotension (systolic blood pressure ≥120 mm Hg) through the second postoperative day decreased the incidence of paraplegia during thoracic aortic reconstruction.[82,83]

Ischemic colon injury is a well-documented complication associated with abdominal aortic resections. Ischemia of the colon is most often attributed to manipulation of the inferior mesenteric artery, which supplies the primary blood supply to the left colon. This vessel is often sacrificed during surgery, and blood flow to the descending and sigmoid colon depends on the presence and adequacy of the collateral vessels. Increased intraabdominal pressure has also been implicated in ischemic colon injury. Mucosal ischemia occurs in 10% of patients who undergo AAA repair. In less than 1% of these patients, infarction of the left colon necessitates surgical intervention.[74]

TABLE 28.4 Hemodynamic Responses to Aortic Unclamping and Therapeutic Interventions

Hemodynamic Changes	Metabolic Changes	Intraoperative Interventions
Decreased arterial blood pressure	Increased lactate	Decrease anesthetic depth
Decreased myocardial contractility	Increased total body oxygen consumption	Decrease vasodilators
Decreased systemic vascular resistance	Decreased mixed venous oxygen saturation	Increase fluids
Decreased central venous pressure	Increased prostaglandins	Increase vasoconstrictor drugs
Decreased preload	Increased activated complement	Reapply cross-clamp for severe hypotension
Decreased cardiac output	Increased myocardial depressant factors	Consider administration of mannitol and sodium bicarbonate
Increased pulmonary artery pressure	Decreased temperature	
	Metabolic acidosis	

Adapted from Norris EJ. Anesthesia for vascular surgery. In Miller RD, et al., eds. *Miller's Anesthesia*. 9th ed. Philadelphia: Elsevier; 2020; Holt PJE, Thompson MM. Abdominal aortic aneurysms: evaluation and decision making. In Cronenwett JL, Johnston W, eds. *Rutherford's Vascular Surgery*. 9th ed. Vol. 2. Philadelphia: Elsevier; 2019.

Aortic Cross-Clamp Release

While the aorta is occluded, metabolites that are liberated as a result of anaerobic metabolism (such as serum lactate) accumulate below the aortic cross-clamp and induce vasodilation. As the cross-clamp is released, SVR decreases, and blood is sequestered into previously dilated veins, decreasing venous return. Reactive hyperemia causes transient vasodilation secondary to the presence of tissue hypoxia, release of adenine, and liberation of an unknown vasodepressor substance that may act as a myocardial depressant and peripheral vasodilator.[74] This combination of events results in decreased preload and afterload. The hemodynamic instability that may ensue after the release of an aortic cross-clamp is called declamping shock syndrome.[84] Evidence demonstrates that venous endothelin (ET), and specifically ET-1, may be partially responsible for the hemodynamic alterations that accompany declamping shock syndrome. Venous ET-1 has a positive inotropic effect on the heart and a vasoconstricting effect on blood vessels. Table 28.4 summarizes the most commonly observed hemodynamic responses to aortic unclamping and therapeutic interventions.

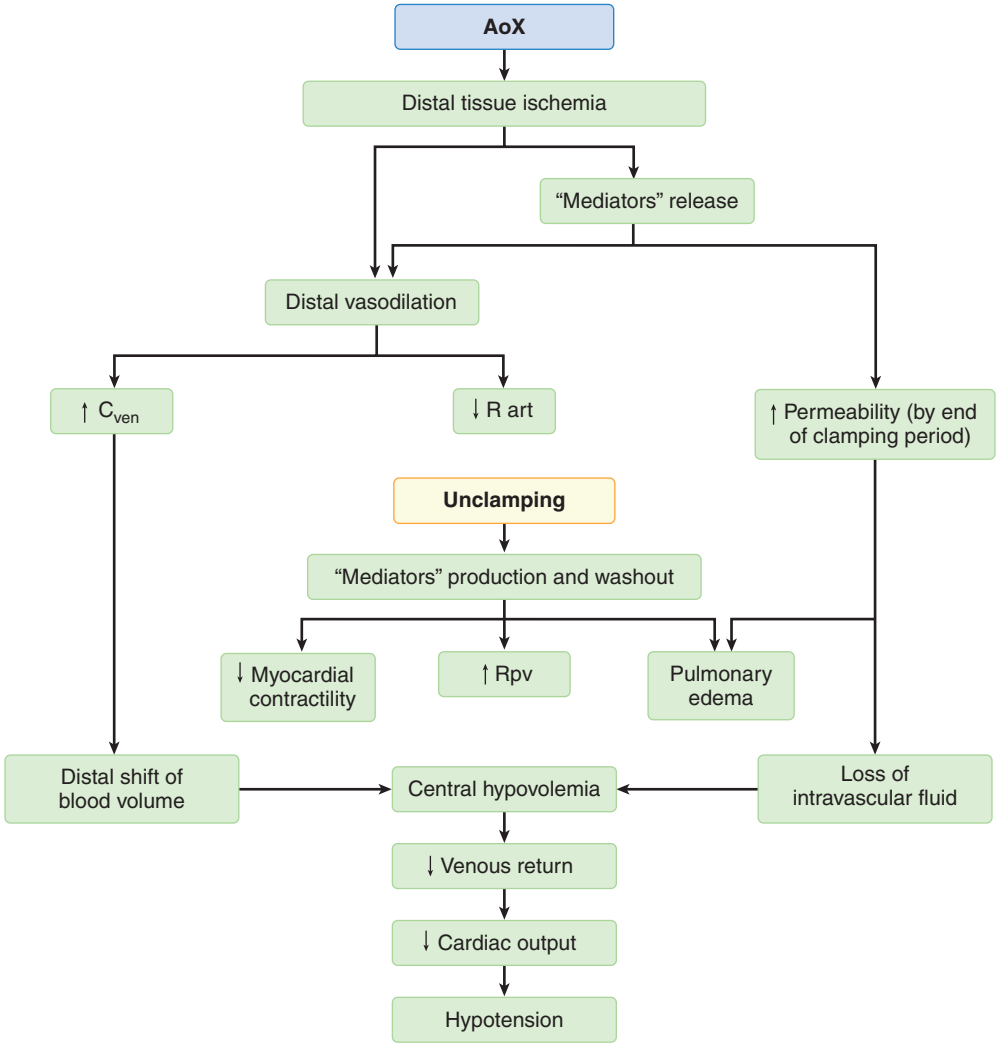

Fig. 28.2 Systemic hemodynamic response to aortic unclamping. *AoX,* Aortic cross-clamping; C_{ven}, venous capacitance; *R art,* arterial resistance; *Rpv,* pulmonary vascular resistance. (From Gelman S. The pathophysiology of aortic cross-clamping and unclamping. *Anesthesiology.* 1995;82:1026-1060.)

The magnitude of the response to unclamping the aorta may be manipulated. Although SVR and MAP decrease, intravascular volume may influence the direction and magnitude of the change in cardiac output. Restoration of circulating blood volume is paramount in providing circulatory stability before release of the aortic clamp.[64,74,85] The site and duration of cross-clamp application, as well as the gradual release of the clamp, influence the magnitude of circulatory instability. Thus effective communication between the anesthetist and the surgical team is vital during the operative procedure. Partial release of the aortic cross-clamp over time often results in less severe hypotension. Vasopressors and/or inotropic agents are administered to help minimize hypotension. An algorithm depicting the systemic hemodynamic response to aortic unclamping is shown in Fig. 28.2.

IRI is a complex metabolic process that occurs during application of the cross-clamp (ischemia) and subsequent unclamping of the aorta (reperfusion). IRI is characterized by metabolic, thrombotic, and inflammatory components. Cells that comprise tissues remain metabolic despite low blood flow, and they liberate cytotoxic mediators during anaerobic metabolism. During cellular ischemia, reactive oxygen species and increased intracellular calcium further inhibit mitochondrial activity and adenosine triphosphate generation. Specific body tissues vary in terms of the time it takes for their cells to become necrotic. The degree of cellular necrosis is primarily determined by the duration of ischemia. The no-reflow phenomenon occurs when the microvasculature is occluded by platelets, neutrophils, and thrombi, causing inadequate perfusion and further increasing cellular necrosis.[1] Reinstituting blood flow increases inflammatory cell influx and cytotoxic substance washout into the central circulation.[82] Myocardial stunning and dysrhythmias can occur due to decreased cellular energy, increased reactive oxygen metabolites, and increased intracellular calcium, necessitating inotropic support.[86] Other manifestations associated with IRI include tissue edema, acute respiratory distress syndrome, compartment syndrome, bacterial translocation, renal failure, and multisystem organ failure.[1]

Surgical Approach

The standard surgical approach for elective abdominal aortic reconstruction is a transperitoneal incision. The advantages of this route include exposure of infrarenal and iliac vessels, ability to inspect intraabdominal organs, and rapid closure.[87] Unfavorable consequences associated with the transperitoneal approach include increased fluid losses, prolonged ileus, postoperative incisional pain, and pulmonary complications.

The retroperitoneal approach is an alternative to the standard route. Its advantages include excellent exposure (especially for both

TABLE 28.5 Comparison of Transperitoneal and Retroperitoneal Approaches

Transperitoneal	Retroperitoneal
Advantages	
Familiarity	Exposure for juxtarenal and suprarenal aneurysms
Access to infrarenal aorta and iliac vessels	Decreased fluid loss
Visualization of intraabdominal viscera	Improved postoperative respiratory function
Rapid opening and closure	Better-tolerated incisional pain Avoids formation of intraabdominal adhesions
Versatility	Does not elicit mesenteric traction syndrome
Disadvantages	
Increased fluid losses	Inaccessibility to distal right renal artery
Less postoperative ileus	
More frequent postoperative respiratory complications	
Increased postoperative incisional pain	

Modified from Sicard GA, et al. Retroperitoneal versus transperitoneal approach of repair of abdominal aortic aneurysms. *Surg Clin North Am.* 1989;69:795–806; Woo EY, Damraur SM. Abdominal aortic aneurysms: open surgical treatment. In Cronenwett JL, Johnston W, eds. *Rutherford's Vascular Surgery*. 9th ed. Vol. 2. Philadelphia: Elsevier; 2019.

juxtarenal and suprarenal aneurysms and in obese patients), decreased fluid losses, less incisional pain, and fewer postoperative pulmonary and intestinal complications. In addition, the retroperitoneal approach does not elicit mesenteric traction syndrome.[87] The reported limitations of this approach are the unfamiliarity of surgeons with this technique, poor right distal renal artery exposure, and the inability to inspect the integrity of the abdominal contents. Table 28.5 compares the standard and retroperitoneal surgical approaches. After cross-clamping, the aneurysm is incised, and a synthetic graft is sewn distally and proximally to the aneurysm. The aortic adventitia is then resewn over the synthetic graft (Fig. 28.3).

Management of Fluid and Blood Loss

Extreme loss of extracellular fluid and blood should be expected during repair of AAAs. The degree of surgical and evaporative losses and third spacing will determine the magnitude of the patient's fluid volume deficit. Furthermore, the surgical approach, the duration of the surgery, and the experience of the surgeon affect the total blood loss. Most blood loss occurs because of backbleeding from the lumbar and inferior mesenteric arteries after the vessels have been clamped and the aneurysm is opened.[88] Anticoagulation with the use of heparin also contributes to blood loss. Excessive bleeding, however, can occur at any point during surgery, and blood replacement is often necessary during open abdominal aortic resections.

Owing to the heightened awareness of transfusion-related morbidity, the use of autologous blood via a cell-saver system is a standard procedure. Presently, three options are available for administering autologous transfusions: preoperative deposit, intraoperative phlebotomy and hemodilution, and intraoperative blood salvage. Ideally, patients donate their own blood to minimize the intraoperative use of homologous blood products and the subsequent risk of transfusion-related viruses. With anemia and decreased hemoglobin, oxygen transport is decreased, thus placing the patient with systemic vascular disease at increased risk for MI and stroke. Autotransfusion blood-salvaging systems may be used for replacing intraoperative blood loss. A study at the Mayo Clinic demonstrated a 75% reduction in transfused banked blood when intraoperative autologous red cell salvage was used. It was a prospective study of 100 patients who underwent elective abdominal aortic resections, and 80% of patients received only their own blood.[88] An increased number of banked red blood cell units infused is an independent risk factor for poor outcome after cardiac surgery.[89] Evidence suggests that there is no difference with respect to 30-day mortality between a hematocrit of 24% and a hematocrit of 30% in patients having cardiac surgery.[90]

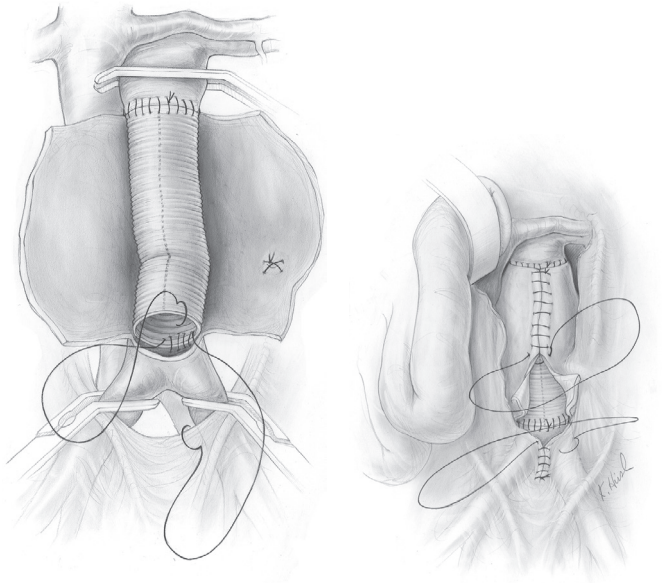

Fig. 28.3 Dacron graft used to repair an aneurysm. (From Zarins CK, Gewertz BL. *Atlas of Vascular Surgery*. 2nd ed. Philadelphia: Churchill Livingstone; 2009.)

Presence of Concurrent Disease

Preoperative Management

The presence of underlying CAD in patients with vascular disease has been well documented. Reports suggest that CAD exists in more than 50% of patients who require abdominal aortic reconstruction and is the single most significant risk factor influencing long-term survivability.[8,91,92] MIs are responsible for 40% to 70% of all fatalities that occur after aneurysm reconstruction.[6,46] Preoperative cardiac evaluation begins with the identification of risk factors that may contribute to adverse cardiac events and subsequent death. When preoperative CAD exists, an increased incidence of postoperative adverse cardiac complications has been demonstrated.[93]

The end point of any method of preoperative cardiac evaluation for aneurysmectomies is identification of functional cardiac limitations. Depending on the degree of cardiac dysfunction, preoperative optimization of cardiac function may range from simple pharmacologic manipulation to surgical intervention. The American College of Cardiology and the American Hospital Association guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery are

BOX 28.4 Optimization of Body Systems Prior to Abdominal Aortic Aneurysm Repair

Cardiac Evaluation
- Quantify risk factors and optimize cardiac function
- Institute appropriate β-blockade
- Institute statin therapy
- Control hypertension
- Institute appropriate anticoagulation therapy

Pulmonary Evaluation
- Advise smoking cessation
- Perform radiologic tests and pulmonary function testing as indicated
- Institute pharmacologic therapy, which may include corticosteroids and bronchodilators

Renal Evaluation
- Assess electrolytes, creatinine, and glomerular filtration rate

Adrenal Evaluation
- Provide steroid supplementation for patients at risk for acute adrenal crises

Deep Vein Thrombosis Prophylaxis
- Administer pharmacologic prophylaxis
- Provide graduated compression stockings
- Provide intermittent pneumatic compression
- Provide venous foot pumps

Musculoskeletal Evaluation
- Assess range of neck motion prior to airway management
- Assess functional limitations for positioning to avoid postoperative paresthesia

Endocrine Evaluation
- Provide short- and long-term glycemic control (mandatory) due to the increased incidence of diabetes

Miscellaneous Considerations
- Order laboratory assessments—complete blood count, coagulation panel, electrolyte panel, blood urea nitrogen, creatinine, albumin, blood sugar, liver function tests, type and crossmatch six units packed red blood cells

From Fleisher LA, et al. 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: executive summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2014;130:2215–2245.

generally followed when preparing patients for these procedures. Optimizing patient preoperative pathophysiologic states (Box 28.4) minimizes the overall rate of morbidity and mortality.

Intraoperative Management

Anesthetic Selection

Several anesthetic techniques are available for abdominal aortic resections. Although each technique has its advantages and disadvantages, no single technique has been proven to be definitively superior. Anesthetic selection should be based on the following objectives: providing optimum analgesia and amnesia, facilitating relaxation, maintaining hemodynamic stability, preserving renal blood flow, and minimizing morbidity and mortality.

General anesthesia. Circulatory stability is especially desirable for patients undergoing AAA reconstruction, especially for those with CAD. All inhalation anesthetics may depress the myocardium and cause hemodynamic instability. Therefore high concentrations of inhalation agents should not be used in patients with a moderate to severe decreased ejection fraction. The degree of myocardial depression is dose dependent, and it is acceptable to administer inhalation agents at lower inhaled concentrations. Beneficial effects attributed to inhalation agents include the ability to alter autonomic responses, reversibility, rapid emergence, potentially earlier extubation, neurologic protection, and cardioprotection.[33] Cardiovascular stability provided by opioids has been well documented, and this feature is especially attractive for patients with ischemic heart disease and ventricular dysfunction. Provision of intense analgesia for the initial postoperative period after major abdominal vascular surgery (i.e., administration of neuraxial opioid) does not alter the combined incidence of major cardiovascular, respiratory, and renal complications.[94] Despite the absence of direct myocardial depression, the sympathetic nervous system inhibition that ensues may decrease SVR and heart rate. Therefore, especially in an individual with a moderate to severely decreased ejection fraction, narcotics should be carefully titrated to the patient's hemodynamic response. Dexmedetomidine is an option since it functions to inhibit the sympathetic nervous system by decreasing central catecholamine release, does not inhibit respiration, and provides postoperative analgesia.

Regional anesthesia. The use of epidural anesthesia for abdominal aneurysmectomies is commonly considered. The benefits of epidural use include (1) decreased preload and afterload, (2) preserved myocardial oxygenation, (3) reduced stress response, (4) excellent muscle relaxation, (5) decreased incidence of postoperative thromboembolism, (6) increased graft flow to the lower extremities, (7) decreased pulmonary complications, and (8) improved postoperative analgesia. Potential disadvantages include the possibility of an epidural hematoma (risk increases with anticoagulation) and severe hypotension during blood loss or unclamping.[34,95]

The use of thoracic epidural analgesia (TEA) in patients having coronary artery bypass surgery decreases the incidence of postoperative supraventricular arrhythmias and respiratory complications. General anesthesia with TEA does not increase the risk of mortality, MI, or neurologic complications compared to GA alone.[96]

Combination techniques. The use of a combined general anesthesia and epidural anesthesia provides the benefits of epidural anesthesia with the ability to provide amnesia and controlled ventilation. Due to neuraxial blockade, a "light" GA can be administered. Postoperative epidural analgesia improves postoperative respiratory function and blood flow to distal tissues. Furthermore, postoperative epidural analgesia reduces postoperative pain and pulmonary complications in patients with chronic obstructive pulmonary disease (COPD), as compared to general anesthesia alone after open aneurysm repair.[97] However, the major risks associated with neuraxial anesthesia are subarachnoid or epidural hemorrhage (resulting in hematoma after heparinization) and hypotension, which may be difficult to treat especially during an episode of acute blood loss.

In summary, all the aforementioned anesthetic techniques can be used safely and can demonstrate positive outcomes. Even more important than anesthetic selection is the clinical management of each patient. Observation, accurate interpretation, and immediate intervention to minimize dramatic hemodynamic variability during the anesthetic process reduce morbidity and mortality to a much greater extent than selection of a superior anesthetic technique.

Fluid Management

Maintaining intravascular volume may be an extreme challenge during abdominal aortic resections. Controversy exists regarding whether the

BOX 28.5 Postoperative Considerations for Patients Having Abdominal Aortic Aneurysm Repair

- Continue invasive hemodynamic monitoring
- Treat acute blood pressure extremes, arrhythmias (atrial fibrillation)
- Assess for postoperative myocardial infarction
- Provide ventilatory management with weaning and extubation
- Assess for abdominal compartment syndrome
- Evaluate hemoglobin, hematocrit, coagulation status, and adequacy of volume replacement
- Assess blood urea nitrogen/creatinine and urine output
- Institute deep vein thrombosis prophylaxis per protocol

BOX 28.6 Potential Complications of Juxtarenal or Suprarenal Aortic Occlusion

- Renal failure
- Hemorrhage
- Distal arterial occlusion
- Infarction
- Pulmonary or cardiac dysfunction
- Impotence
- Paraplegia
- Thrombosis
- Pseudoaneurysm formation
- Aortoenteric fistula

administration of crystalloids or colloids affects the overall incidence of morbidity and mortality. Blood losses initially can be replaced with crystalloids at a ratio of 3:1. The combination of crystalloid and colloid administration is also acceptable. Regardless of the choice of fluid, volume replacement must be dictated by physiologic parameters. Fluid replacement should be sufficient to maintain normal cardiac filling pressures and cardiac output, and a urine output of at least 1 mL/kg/hr. Patients with limited cardiac reserve can develop congestive heart failure if hypervolemia occurs. As mentioned previously, cell-saver blood retrieval can be used, and vascular access should include two large-bore intravenous lines and a central venous catheter. Goal-directed fluid therapy may help to optimize a patient's intravascular volume and hemodynamic status.

Hemodynamic Alterations

Hemodynamic changes are likely to occur throughout the procedure. Adequate preoperative sedation should be given before the placement of invasive monitoring equipment. Fluctuations in heart rate and blood pressure should be anticipated during induction and intubation. Preoperative replacement of fluid deficits prevents exaggerated responses to vasodilating induction agents. For patients with adequate left ventricular function, hemodynamic stability can be preserved with a "slow" and controlled induction using higher doses of opioids and sympathomimetic agents if hypotension develops. The response to mesenteric traction (discussed previously) is also associated with stimulation of the celiac reflex, which results in bradycardia and hypotension.

Postoperative Considerations

Cardiac, respiratory, and renal failure are the most common complications observed postoperatively in patients recovering from abdominal aortic reconstruction. Cardiovascular function must be closely monitored in the ICU for at least 24 hours after surgery. Maintaining adequate blood pressure, intravascular fluid volume, and myocardial oxygenation is paramount during this period. MI frequently contributes to postoperative morbidity and mortality; serial cardiac enzyme analysis may be justified. Pharmacologic agents used in the treatment of hypertension or hypotension must also be available.

Most patients require ventilatory assistance during the postoperative period. Vigilant monitoring of respiratory function is mandatory, especially when epidural catheters are used for postoperative analgesia. To address the significant number of serious postoperative complications (Box 28.5), intensive and continuous assessment of the patient condition is vital. Patients are admitted to the ICU for high-acuity monitoring and care.

Juxtarenal and Suprarenal Aortic Aneurysms

Although most AAAs occur below the level of the renal arteries, 2% extend proximally and involve the renal or visceral arteries.[98] Juxtarenal aneurysms are located at the level of the renal arteries, but they spare the renal artery orifice. More proximal suprarenal aneurysms include at least one of the renal arteries and may involve visceral vessels. The effects of aortic cross-clamping for juxtarenal or suprarenal aneurysms are similar to those for infrarenal aortic occlusions; however, the magnitude of hemodynamic alterations increases as the aorta is clamped more proximally.

Renal failure, although possible during infrarenal aortic cross-clamping, occurs more often because of suprarenal aortic occlusion. Maintaining adequate intravascular volume and administering osmotic and loop diuretics may minimize renal ischemia and dysfunction.

Paraplegia is possible when the blood supply to the spinal cord is interrupted by aortic cross-clamping at or above the level of the diaphragm. Increasing the MAP or decreasing cerebrospinal fluid (CSF) pressure by placing a catheter in the subarachnoid space to drain CSF may be used as a means to increase spinal cord perfusion pressure.[98] Total body hypothermia and neurologic monitoring (i.e., SSEPs and MEPs) can be used to decrease the incidence of paraplegia. Early detection and intervention for spinal cord ischemia can decrease the incidence of permanent paraplegia after endovascular stent grafting (EVSG) of the descending thoracic aorta. Neurologic deficits can become evident weeks after surgery. Routine SSEP monitoring, serial neurologic assessment, arterial pressure augmentation, and CSF drainage may benefit patients at risk for paraplegia.[99] Box 28.6 summarizes the complications that may result from juxtarenal or suprarenal aortic occlusion.

Ruptured Abdominal Aortic Aneurysm

A high mortality rate of 80% to 90% is associated with a ruptured AAA, whereas postoperative mortality is estimated to range from 40% to 50%.[100] The mortality after surgery to repair a ruptured AAA does not vary based on the type of surgical repair (open vs endovascular).[101] The most common symptoms of ruptured AAAs include a triad of severe abdominal discomfort or back pain, altered level of consciousness caused by hypotension, and a pulsatile abdominal mass.[102] Other common symptoms include syncope, groin or flank pain, hematuria, and groin hernia. Risk factors associated with an increase in mortality in patients with a ruptured AAA are noted in Box 28.7.

Hypotension and a history of cardiac disease are two factors associated with the poorest prognosis.[51] Patients with these symptoms should be immediately transferred to the operating room for surgical exploration. When hypotension is absent, more time is available for comprehensive preoperative assessment and testing; however, decompensation and cardiovascular collapse can occur rapidly.

Once the patient arrives in the operating suite, performing a brief preoperative evaluation to establish peripheral and central venous access is a priority. Provisions for fluid, blood, and blood product

BOX 28.7 Risk Factors Associated With an Increased Risk of Mortality in Abdominal Aortic Aneurysm Rupture

- Increased age
- Women
- Nonwhite race
- Insurance status (higher for those who self-pay or are on Medicaid in the United States)
- Comorbid conditions
- Congestive heart failure
- Renal failure
- Valvular heart disease

From Cronenwett JL, Johnston W. *Rutherford's Vascular Surgery*. 9th ed. Vol. 2. Philadelphia: Elsevier; 2019.

administration is necessary, as rapid massive hemorrhage is a distinct possibility. The use of blood salvaging techniques and the ability to rapidly infuse blood and fluids are indicated. Insertion of an arterial line is essential, as significant fluctuations in blood pressure should be expected. Vasopressors such as phenylephrine and epinephrine given as a bolus and by infusion should be available. Hemodynamic stability must be the primary objective, and anesthetic induction and maintenance agents must be selected on a case-by-case basis.

Cardiovascular stability is the primary focus until blood loss from the proximal aorta is controlled by surgical intervention. Fluid resuscitation can begin with crystalloids; colloids and blood products can be administered as they become available. Intraoperative blood salvage provisions should be available. Coagulation studies and other laboratory tests, including hemoglobin, hematocrit, and ionized calcium values, should be obtained. Calcium is a positive inotrope, which is necessary for myocardial contractility. Large amounts of citrate used as a preservative in banked blood bind calcium ions and result in relative hypocalcemia. Decreased myocardial contractility—as evidenced by hypotension, increased left ventricular end-diastolic pressure, and increased central venous pressure—can be caused by hypocalcemia. Increased bleeding can also be caused by intraoperative hypocalcemia. If hypocalcemia occurs, calcium chloride can be administered, as guided by ionized calcium levels. Dilutional thrombocytopenia is the most common reason for coagulopathy to develop after massive intravenous fluid and blood administration. The use of fresh frozen plasma has been shown to decrease the total transfusion requirement and the incidence of coagulopathies.[37]

The hemodynamic effects of aortic cross-clamping and release are similar to those for elective surgery; however, responses may be extreme especially if hypotension exists when the clamp is released. Most patients require large amounts of fluid and blood replacement, and therefore postoperative mechanical ventilation is recommended. Patients experiencing hypovolemic shock are exquisitely sensitive to the myocardial depressant and vasodilatory effects of anesthetic agents. Titration of anesthetic agents and the use of vasopressors and/or positive inotropes are warranted. Additionally, ventilation may be difficult due to surgical displacement of the diaphragm cephalad. This will decrease lung expansion and functional residual capacity and increase peak pressures. Because positive pressure ventilation decreases venous return, hypotension can occur. Minimizing peak inspiratory pressures and administering higher concentrations of oxygen will help maximize venous return and maintain oxygen saturation. Manual initiation of a positive pressure breath will improve alveolar recruitment and distention.

TABLE 28.6 Classification Schemes of Acute Aortic Dissection

Classification	Site of Origin and Extent of Aortic Involvement
DeBakey	
Type I	Originates in the ascending aorta and extends at least to the aortic arch and often to the descending aorta (and beyond)
Type II	Originates in the ascending aorta; confined to this segment
Type III	Originates in the descending aorta, usually just distal to the left subclavian artery, and extends distally
Stanford	
Type A	Dissections that involve the ascending aorta (with or without extension into the descending aorta)
Type B	Dissections that do not involve the ascending aorta

From Braverman AC, et al. Diseases of the aorta. In Bonow RO, et al., eds. *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine*. 9th ed. Vol. 2. Philadelphia: Elsevier; 2012:1320.

THORACIC AORTIC ANEURYSMS

The mortality associated with elective thoracic aneurysm repair is 22%, and if rupture occurs, it increases to 54%.[103] Patients with aortic dissections have a predicted survival of only 3 months if they do not undergo surgical repair because the incidence of rupture is high.[104] Aneurysms have been described for hundreds of years; however, the development of the arterial prosthesis that led to successful bypass options did not occur until 1951.[105] The refinement of endovascular stent grafts, surgical and perfusion techniques, and intraoperative management have contributed to improved surgical outcomes.

Classification

Aneurysms of the thoracic aorta may be classified with respect to type, shape, and location. Typically, aneurysms involving all three layers of the arterial wall—tunica adventitia, tunica media, and tunica intima—are considered to be true aneurysms. In comparison, aneurysms that solely involve the adventitia are termed false aneurysms. The shape of the lesion also can serve as a means of characterizing aneurysms. Fusiform aneurysms have a spindle shape and result in dilation of the aorta. Saccular aneurysms are spherical dilations and are generally limited to only one segment of the vessel wall. Aortic dissection is the result of a spontaneous tear within the intima that permits the flow of blood through a false passage along the longitudinal axis of the aorta. If an aortic dissection is extensive, it is difficult for the surgeon to isolate the aneurysm and secure a graft, due to the weakened aortic wall. There are two major classification schemes for aortic dissections, based on the location. These are the DeBakey and Stanford classifications (Table 28.6). Thoracoabdominal aortic aneurysms (TAAAs) are classified using the Crawford classification (Fig. 28.4).

Etiology

Atherosclerosis is the most common cause of aneurysmal pathology. Atherosclerotic lesions occur most often in the descending and distal thoracic aorta and are most often classified as fusiform. Less common causes include aortic dissection and various mechanical, inflammatory, and infectious processes. The various causes of aortic aneurysms are classified in Box 28.8.

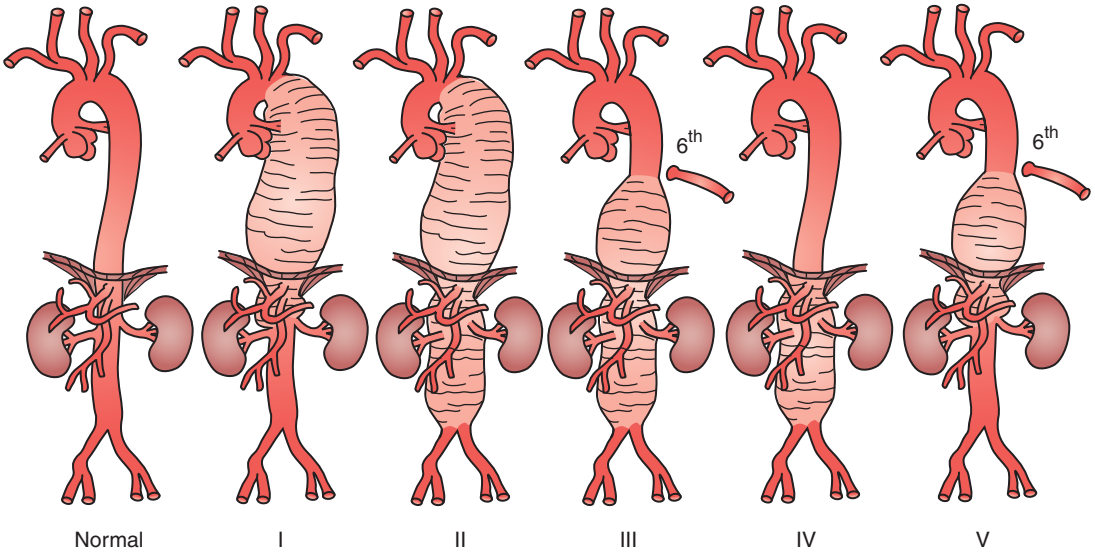

Fig. 28.4 Crawford classification of thoracoabdominal aortic aneurysms. Type I, distal to the left subclavian artery to above the renal arteries. Type II, distal to the left subclavian artery to below the renal arteries. Type III, from the sixth intercostal space to the renal arteries. Type IV, from the 13th intercostal space to the iliac bifurcation (entire abdominal aorta). Type V, below the sixth intercostal space to just above the renal arteries. (From Cronenwett JL, Johnston KW. *Rutherford's Vascular Surgery*. 9th ed. Vol. 2. Philadelphia: Elsevier; 2019:2096.)

BOX 28.8 Etiology of Thoracoabdominal Aortic Aneurysms

Degenerative
- Nonspecific (commonly considered arteriosclerotic), dysplastic (80%)

Mechanical (Hemodynamic)
- Dissections (15%–20%)
- Poststenotic
- Arteriovenous fistula
- Blunt or penetrating trauma

Connective Tissue
- Ehlers-Danlos syndrome
- Marfan syndrome

Inflammatory (Noninfectious)
- Takayasu disease
- Behçet syndrome
- Reiter syndrome
- Kawasaki disease
- Microvascular disorder (e.g., polyarteritis)
- Ankylosing spondylitis
- Rheumatoid aortitis
- Periarterial inflammatory disease (e.g., pancreatitis)

Infectious
- Tuberculosis
- Bacterial
- Fungal
- Spirochetal (syphilis)

Anastomosis
- Postarteriotomy
- Postoperative pseudoaneurysm

Diagnosis

The symptomatology of thoracic aneurysms is often related to the site of the lesion and its compression of adjacent structures. Pain, stridor, and cough may result from compression of thoracic structures. A change in the quality of one's voice, resulting in hoarseness, can occur from impingement by the aneurysm on the left recurrent laryngeal nerve. This can occur because the left recurrent larynx nerve bifurcates from the vagus nerve at the level of the aortic arch. Symptoms related to aortic insufficiency may be observed in aneurysms of the ascending aorta. An upper mediastinal mass may be an incidental finding on conventional chest radiography in an asymptomatic patient. Further investigation with noninvasive methods such as CT scan and MRI can describe the specific anatomic characteristics and location of the aneurysm.

Treatment

As previously described, a high mortality rate is associated with the rupture of thoracic aneurysms, therefore early detection and surgical intervention make a significant contribution to long-term survival. Hemodynamic compromise and increased complexity of the aneurysm are associated with increased postoperative mortality. The surgical approach and the method of aneurysm resection vary according to the location of the lesion within the thoracic aorta. Resection of the ascending aorta and graft replacement necessitate the use of complete cardiopulmonary bypass or partial cardiopulmonary bypass (atrial-femoral: left atrial cannulation to a centrifugal pump, and reinfusion to a femoral artery cannula). If extracorporeal circulation is not indicated, heparin (50–100 units/kg) is required prior to aortic cross-clamping. For complete cardiac bypass, total systemic heparinization with 400 units/kg is necessary, and monitoring of activated clotting time is needed.[106] Depending on the proximity of the aneurysm to the aortic arch, the aortic valve may require replacement. Surgical resection of lesions in the transverse arch compromises cerebral perfusion. For these higher aneurysms various bypass techniques, combined with profound hypothermia and circulatory arrest, have been used.[107]

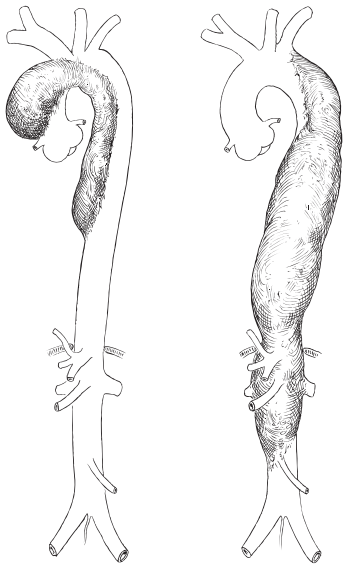

Fig. 28.5 Types of aortic dissection. Type A involves the ascending aorta and may extend into the aortic arch. Type B starts at the proximal descending aorta and extends distally.

Aneurysms of the descending aorta may be resected after application of an aortic cross-clamp; however, perfusion to distal organs can be compromised during this procedure. Arterial line and pulse oximetry monitoring should occur on the right side because impingement of the left subclavian artery, which provides blood flow to the left hand, is possible. A double lumen tube is placed, and deflation of the left lung is necessary to avoid left lung contusion and improve the surgical operating conditions. The patient is positioned in the left lateral decubitus position, and a left-sided thoracotomy is accomplished. The extent of the thoracotomy is determined by the extent of the aneurysm. A lower thoracic incision is associated with a decreased incidence of postoperative pulmonary dysfunction.[108]

AORTIC DISSECTION

Aortic dissection is characterized by a spontaneous tear of the vessel wall intima, permitting the passage of blood along a false lumen. Although the cause of dissections is unclear, lesions that were thought to be related to cystic necrotic processes may actually be caused by variations in wall integrity. Hypertension is the most common factor that contributes to the progression of the lesion. Manipulation of the ascending aorta during cardiac surgery may be associated with aortic dissection.[109] The symptoms of aortic dissection are the result of interruption of blood supply to vital organs. The most serious complication is aneurysm rupture. Diagnosis can be accomplished by the previously mentioned noninvasive techniques.

Treatment of dissecting aortic lesions depends on their location within the thoracic aorta (Fig. 28.5). Type A lesions have the highest incidence of rupture and require immediate surgical intervention. Type B lesions may initially be managed medically, with the administration of arterial dilating and β-adrenergic blocking agents.

In summary, surgical resection of thoracic aortic lesions enhances long-term survival. Refinement of surgical techniques and improvements in perfusion technology have reduced the overall mortality rate. The surgical method used is dependent on the location of the aortic lesion. Anesthesia for aneurysms of the ascending and transverse aorta requires cardiopulmonary bypass.

DESCENDING THORACIC AND THORACOABDOMINAL ANEURYSMS

Preoperative Assessment

Patients who undergo major vascular surgery are often elderly and have varying degrees of concurrent disease. Most patients who develop a descending thoracic aortic aneurysm (DTAA) are asymptomatic. Operative surgical decisions are based on the size, extent, and rate of expansion of the aneurysm. For patients with degenerative aortic disease, surgical repair is advised for aneurysms 6 cm or larger. Independent risk factors for DTAA include pain, increased age, COPD, renal insufficiency, aneurysm size, and aneurysm expansion rate.[110]

The importance of a thorough preoperative evaluation cannot be overemphasized. Special attention should be directed toward cardiac, renal, and neurologic function. Although most fatalities related to thoracic aortic surgery are cardiac in origin, renal and neurologic dysfunction contribute to poor surgical outcomes.[111] Preoperative renal dysfunction is directly related to postoperative renal failure and is thought to be one of the strongest contributors to renal deterioration after surgery.[83,111] Neurologic function should be carefully assessed in the preoperative phase. Paraplegia is one of the most devastating consequences of thoracic aortic surgery, and any alteration in lower extremity function should be noted. Hoarseness related to compression of the recurrent laryngeal nerve should be assessed and documented. The left recurrent laryngeal nerve is most susceptible due to its close proximity to the aortic arch. Bilateral recurrent laryngeal nerve compression or damage can result in respiratory compromise.

Intraoperative Management

Monitoring

Intraoperative monitoring devices used for thoracoabdominal aneurysm resection are the same as those used for abdominal aneurysmectomies. Direct intraarterial blood pressure and pulmonary artery pressure monitoring is standard during extracorporeal circulation. If the aneurysm involves the thoracic region or the distal aortic arch, right radial arterial line monitoring is preferred because left subclavian arterial blood flow may be compromised during surgery. The use of TEE is suggested for cardiac monitoring in patients with myocardial dysfunction. An indwelling urinary catheter is used for assessing renal function. To facilitate exposing the descending thoracic aorta, a double-lumen endotracheal tube is inserted to allow for one-lung ventilation. As a result, careful monitoring of oxygenation is mandatory. Routine use of pulse oximetry may be limited if the left subclavian artery is manipulated; therefore the right hand, the ear, or the nose should be used for monitoring oxygen saturation. Finally, a lumbar intrathecal catheter is inserted to access CSF pressure. SSEPs and/or MEPs are often used to monitor and detect neurologic dysfunction.

Spinal Cord Ischemia

Neurologic dysfunction is a serious complication associated with thoracic aortic aneurysm (TAA) reconstruction. Spinal cord injury is categorized into immediate and delayed paraplegia. The incidence of immediate paraplegia with DTAA ranges from 0% to 3% if surgery is performed with adjunctive procedures or clamp times are less than 10 minutes. However, the incidence of paraplegia and/or paresis for patients having TAA repair is 7.1% ± 6.1% (range 0%–32%).[112] Impending spinal cord injury depends on the type of aneurysm, surgical technique, cross-clamp time, and use of spinal cord protection interventions.[113] The exact incidence of delayed paraplegia is unknown, but it is believed that as many as 25% of all spinal cord injuries are delayed. The primary preoperative risk factors for delayed paraplegia

include type 2 aneurysms, emergency procedures, number of sacrificed segmental segments, and renal failure. The main postoperative factors include hemodynamic instability caused by atrial fibrillation, bleeding, multiorgan failure, and sepsis.[110]

Several techniques have been successfully applied in an effort to decrease the incidence of neurologic dysfunction after thoracic aortic surgery. These include SSEP and MEP monitoring, CSF drainage, hypothermia, reattachment of intercostal arteries, and distal aortic perfusion. Systemic hypothermia and selective cooling of the spinal cord may lengthen ischemic time intervals; however, the clinical benefits are unclear.[114] The use of various bypass mechanisms and distal shunts may minimize the length of aortic occlusion time.

Spinal cord perfusion pressure can be estimated by calculating the arterial blood pressure minus the CSF pressure. During aortic clamping, CSF pressure increases whereas arterial pressure decreases distal to the clamp. The spinal cord perfusion pressure can therefore be manipulated by altering arterial blood pressure and draining CSF through an intrathecal catheter.[114,115] The most influential interventions used to protect the spinal cord during thoracic aortic cross-clamping include (1) routine CSF drainage (CSF pressure <10 mm Hg), (2) endorphin receptor blockade (naloxone infusion), (3) moderate intraoperative hypothermia (<35°C), (4) avoiding hypotension (MAP >90 mm Hg), and (5) optimizing cardiac function.[116] It is postulated that increased levels of excitatory amino acid neurotransmitters bind to opioid receptors in the spinal cord and induce spinal cord edema. Therefore administration of naloxone may inhibit edema formation. Avoiding the use of sodium nitroprusside is indicated, as arterial dilation may cause a "steal phenomenon," further decreasing spinal cord blood flow.

Methods for detecting spinal cord ischemia were discussed previously. The intraoperative use of SSEPs and MEPs can provide early identification of neurologic dysfunction, but these monitoring modalities do not ensure spinal cord integrity. Factors that contribute to the development of neurologic deficits include the level of aortic clamp application, ischemic time, embolization or thrombosis of a critical intercostal artery, failure to revascularize intercostal arteries, and the urgency of surgical intervention.[114,115] Delayed paraplegia may also be the result of IRI, although the exact mechanism of injury has not been proven.[110,117,118] Additional complications of thoracoabdominal aortic reconstruction are listed in Box 28.9.

Anesthetic Management

The principles of perioperative management of TAAs and DTAAs are similar to those previously discussed for abdominal aortic aneurysmectomies. Anesthetic selection should be based on the presence of concomitant disease processes, with the objective of maintaining cardiovascular stability and minimizing morbidity and mortality. Intraoperative monitoring should focus on detection of myocardial, neurologic, and renal ischemia. The hemodynamic consequences of aortic cross-clamping should be attenuated by the use of pharmacologic adjuncts. Restoration of circulating blood volume minimizes the hemodynamic alterations caused by the release of the aortic clamp.

Postoperative Considerations

After surgery is completed, if a double-lumen endotracheal tube was used, it should be replaced with a standard endotracheal tube since postoperative ventilatory assistance is usually required. Airway anatomy may become edematous during surgery, causing difficulty with ventilation and reintubation. Under these circumstances, the double-lumen endotracheal tube may be left in place. Replacement of the endotracheal tube can proceed in the postoperative period after the airway edema has dissipated. Recurrent laryngeal nerve dysfunction can contribute to breathing difficulties after extubation.

BOX 28.9 Complications Following Thoracoabdominal Aortic Aneurysm Repair

Early Complications
- Respiratory failure (most common complication)
- Hemorrhage
- Myocardial infarction
- Congestive heart failure
- Early paraplegia
- Embolization/thrombosis
- Distal artery occlusion
- Bowel ischemia
- Sexual dysfunction
- Infection
- Renal failure
- Cerebrovascular accident

Late Complications
- Delayed paraplegia
- Graft thrombosis
- Fistula formation
- False aneurysm
- Graft infection

Close observation of neurologic, circulatory, pulmonary, and renal status is warranted in the postoperative phase. Hemodynamic control is vital to maintaining perfusion to vital organs without creating excessive demands on the heart or the aortic graft. Careful monitoring of respiratory status aided by arterial blood gas analysis is indicated. Epidural analgesia with the use of local anesthetics, narcotics, or both can be administered for pain relief.

ENDOVASCULAR AORTIC ANEURYSM REPAIR

In 1991 the first EVSG procedure was performed to repair an infrarenal aortic aneurysm. The development of this technique allows surgeons to repair an AAA in a less invasive manner. Due to severe cardiac and respiratory pathology, as many as 30% of patients with aortic aneurysms are poor surgical candidates.[119] Thus EVAR was initially developed to help patients with severe coexisting disease who were not considered viable surgical candidates. Presently, EVAR is the treatment of choice for the majority of patients with an AAA.[120] In high-risk patients having elective AAA repair, the 30-day and 1-year mortality rates are significantly decreased with EVAR as compared to OSR.[121] Researchers suggest that the patient population that may benefit most from EVAR are high-risk patients.[122] Furthermore, patients who are prone to aortic aneurysm development commonly have significant coexisting diseases (Box 28.10).

Endovascular aortic repair is associated with improved 30-day outcomes (all-cause mortality, readmission, surgical site infection, pneumonia, and sepsis) as compared to OSR.[122-124] In one study, the 30-day mortality rate was 1.7% in the EVAR group vs 4.7% in the OSR group. Secondary interventions most often caused by endoleak were more common in the EVAR group (9.8% vs 5.8%).[125] However, there was no significant difference between the groups with respect to 2-year survival. A comparison of EVAR vs OSR showed that mortality was 0.6% and mean LOS was 5.8 days for EVAR, whereas in-hospital mortality for OSR was 4.6%, and the average LOS was 11.9 days.[126] EVAR is associated with decreased procedure duration, a decreased need for transfusion of blood and blood products, a shorter duration of hospitalization, and decreased morbidity compared to OSR.[127] Perioperative

BOX 28.10 Predisposing Factors for Patients Who Are Prone to Aortic Aneurysm Development

- Hypertension
- Male gender
- Heart disease
- Smoking
- Chronic obstructive pulmonary disease
- Diabetes mellitus
- Renal impairment
- Carotid artery disease
- Peripheral arterial disease
- Family history

Data from Townsend CM, et al. *Sabiston Textbook of Surgery*. 20th ed. Philadelphia: Elsevier; 2017.

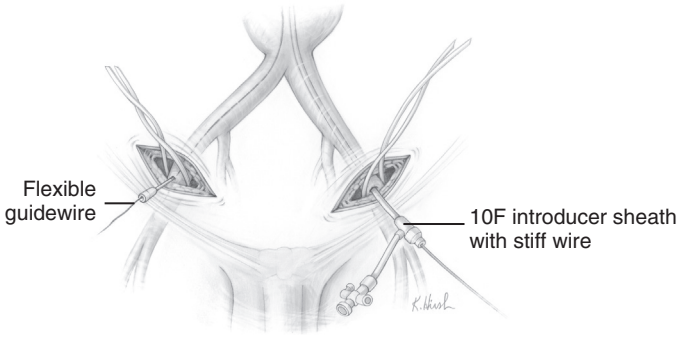

Fig. 28.6 Femoral cutdown and insertion of introducer sheath. (From Zarins CK, Gewertz BL. *Atlas of Vascular Surgery*. 2nd ed. Philadelphia: Churchill Livingstone; 2009.)

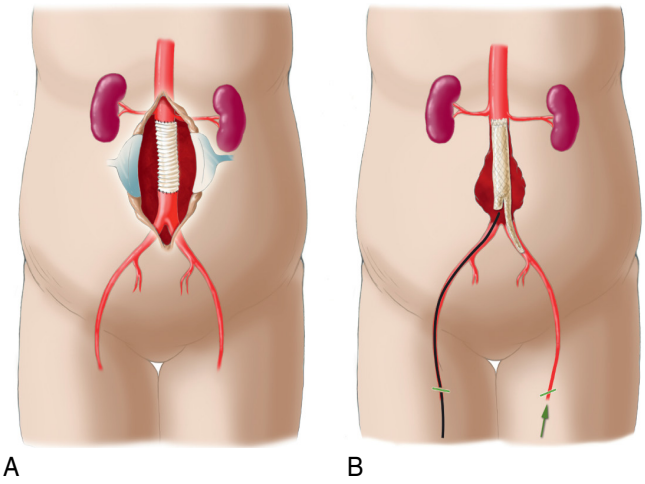

Fig. 28.7 Comparison of (A) open surgical repair and (B) endovascular aortic aneurysm repair.

mortality after EVAR has remained similar in recent years despite improvements in techniques, devices, and proficiency. Randomized trials such as the Endovascular Aneurysm Repair 1 (EVAR 1), Dutch Randomized Endovascular Aneurysm Management (DREAM), and Open versus Endovascular Repair (OVER) trials showed lower 30-day mortality rates for EVAR as compared to OSR. However, late mortality rates (24–36 months postoperatively) are similar for EVAR and OSR.[128]

Reinterventions occur more frequently after EVAR than after OSR.[129] The primary reason for a secondary corrective procedure is due to endoleak. Endoleak is a term that is used to describe the inability of the EVSG to isolate blood flow into the aneurysm sac. Endoleak has been determined to be a significant risk factor for late open conversion. The overall risk of late failure is approximately 3% per year.[124]

EVAR is also being used to treat patients with TAAs. The mortality rate for EVAR for elective DTAA repairs range from 3.5% to 12.5%, as compared with an open approach, where mortality is approximately 10%.[130] Reports also show that EVAR has a low incidence (0%–6%) of spinal cord ischemia and paraplegia.[131] Potential explanations for the decreased incidence of spinal cord trauma as compared to OSR are (1) no thoracic aortic cross-clamping and (2) no prolonged periods of extreme hypotension. Perioperative hypotension (MAP <70 mm Hg) was a significant predictor of spinal cord ischemia in patients undergoing EVAR for TAA.[132]

The overall mortality rate for patients with a ruptured AAA who are alive when diagnosed in emergency departments is 40% to 70%.[133] Since the 1950s, mortality from ruptured AAAs has decreased only 3.5% per decade.[134] Patient survival after emergency repair with EVAR has increased from 2005 to 2011. A significant improvement is noted particularly in those patients who survive the first 24 hours postoperatively.[135] The 30-day mortality rate after AAA rupture is estimated to be 10% to 45%.[136] Even though secondary interventions and EVSG surveillance are required, the use of EVAR for both ruptured AAAs and TAAs in patients with suitable anatomy is a lifesaving option.[137] Medical centers that consider EVAR for ruptured AAA repair must have immediate CT imaging capabilities, trained endovascular teams, adequate endovascular supplies, and a specially arranged surgical suite.

Procedure

The most significant intraoperative advantages with EVAR as compared to OSR are the absence of aortic cross-clamping and the absence of an incision that extends from the xiphoid process to the pubis. EVAR involves deployment of an EVSG within the aortic lumen. The graft restricts blood flow to the portion of the aorta where the aneurysm exists. This procedure is also performed for patients who have TAAs or TAAAs. Cannulation of both femoral arteries is performed. As seen in Fig. 28.6, a guide wire is threaded through the iliac artery to the level of the aneurysm. Next, a sheath is inserted over the guide wire and positioned at the aneurysm location through the use of fluoroscopy. The proximal end of the sheath must extend beyond the aneurysm, and care must be taken to avoid occlusion of the renal arteries. Once the sheath is deployed, radial force or fixation mechanisms such as hooks or barbs on the stent become embedded into the aortic wall to prevent stent migration (Fig. 28.7).

The surgical procedure may take place in a traditional operating room or an interventional radiology suite. Compared with the conventional surgical method, advantages of the endovascular approach include the absence of aortic cross-clamping, improved hemodynamic stability, decreased incidence of embolic events, decreased blood loss, a reduced stress response, decreased incidence of renal dysfunction, and decreased postoperative discomfort.[138,139] Systemic anticoagulation with heparin (50–100 units/kg) is administered prior to catheter manipulation.[140] Administration of a broad-spectrum antibiotic is recommended prior to surgery. The anesthetic techniques that can be used for EVAR include general anesthesia, neuraxial blockade, or local anesthesia with sedation.[141]

Local anesthesia with sedation, as compared to general anesthesia, is associated with decreases in nonfatal cardiac morbidity, respiratory complications, renal failure, and overall mortality.[142,143] There is also decreased pulmonary morbidity as compared with general anesthesia,

and local anesthesia with sedation is associated with a shorter LOS as compared with general and neuraxial anesthesia.[144] The goals for intraoperative management for EVAR include maintaining hemodynamic stability, providing analgesia and anxiolysis, and being prepared to rapidly convert to an open procedure. Local or neuraxial anesthesia is associated with fewer ICU admissions, decreased length of hospitalization, and fewer systemic complications, as compared to general anesthesia.[145] In an alternative analysis there was no difference in 30-day mortality associated with either local anesthesia or general anesthesia provided for EVAR. However, shorter operative times, shorter length of hospitalization, and fewer postoperative complications were associated with a local anesthetic technique.[146]

With infrarenal or suprarenal EVAR, creatinine clearance values can decrease by 10% in the first year.[147] However, proximal endovascular graft migration can occur, causing renal artery occlusion and postoperative renal failure.[148] Fenestrated EVSGs that are constructed to allow blood to flow to the renal arteries can be used safely for those patients who have juxtarenal or suprarenal aortic aneurysms.[149] Plasma catecholamine concentrations and mediators of the systemic immune response are decreased in patients who undergo the endovascular approach as compared with patients who undergo conventional repair.[150,151] Furthermore, there is evidence suggesting that patients who undergo EVAR release less plasma cortisol, develop significantly less sepsis, and may encounter a reduced incidence of systemic immune response syndrome as compared to those having traditional open AAA repair.[152] Some of the complications that can arise from the EVAR approach include (1) endograft thrombosis, migration, or rupture; (2) graft infection; (3) iliac artery rupture; and (4) lower extremity ischemia.[153] Fatal cerebral embolism resulting in sudden respiratory arrest has occurred during EVAR.[154] Box 28.11 lists potential complications associated with EVAR.

Endovascular graft design and durability continue to improve. Graft devices are either unibody (come in one piece) or modular (come in multiple pieces). The endograft fabric is either woven polyester (Dacron) or polytetrafluoroethylene. There is no significant difference in biologic response when comparing these two materials.[155] The graft skeleton is constructed of stainless steel, Nitinol, or Elgiloy (Fig. 28.8). Nitinol stents are popular because they exhibit minimal shortening after deployment when exposed to body temperature. There is considerable interest and research involving drug-eluting stents. In initial clinical trials, researchers have shown that restenosis rates are improved with the newer generation endovascular stents.[156,157] EVSGs have undergone modifications to meet anatomic challenges and improve patient outcomes. In the past, endovascular repair has been limited to infrarenal AAAs and isolated TAAs. The advent of fenestrated and branched endografts have made endovascular repair of thoracoabdominal and juxtarenal aneurysms possible. Fenestrated EVSGs are safe and effective in short- and midterm postoperative follow-up.[158] Continued evolution of endograft technology

BOX 28.11 Potential Complications Associated With EVAR

Graft and Deployment Complications

- Failed deployment
- Microembolization
- Migration/occlusion of major branch arteries (i.e., renal, mesenteric)
- Aortic perforation/aneurysm rupture
- Aortic dissection
- Hematoma formation
- Endoleak
- Stenosis/kink/thrombosis
- Graft tear
- Damage to access arteries (femoral → iliac)
- Infection

Radiologic Implications

- Radiation exposure
- Allergy to contrast dye
- Renal insufficiency from contrast dye

Systemic Complications

- Neurologic (CVA, paraplegia)
- Cardiac morbidity/mortality
- Pulmonary insufficiency
- Renal insufficiency
- Postimplantation syndrome

CVA, Cardiovascular accident; *EVAR,* endovascular aneurysm repair.

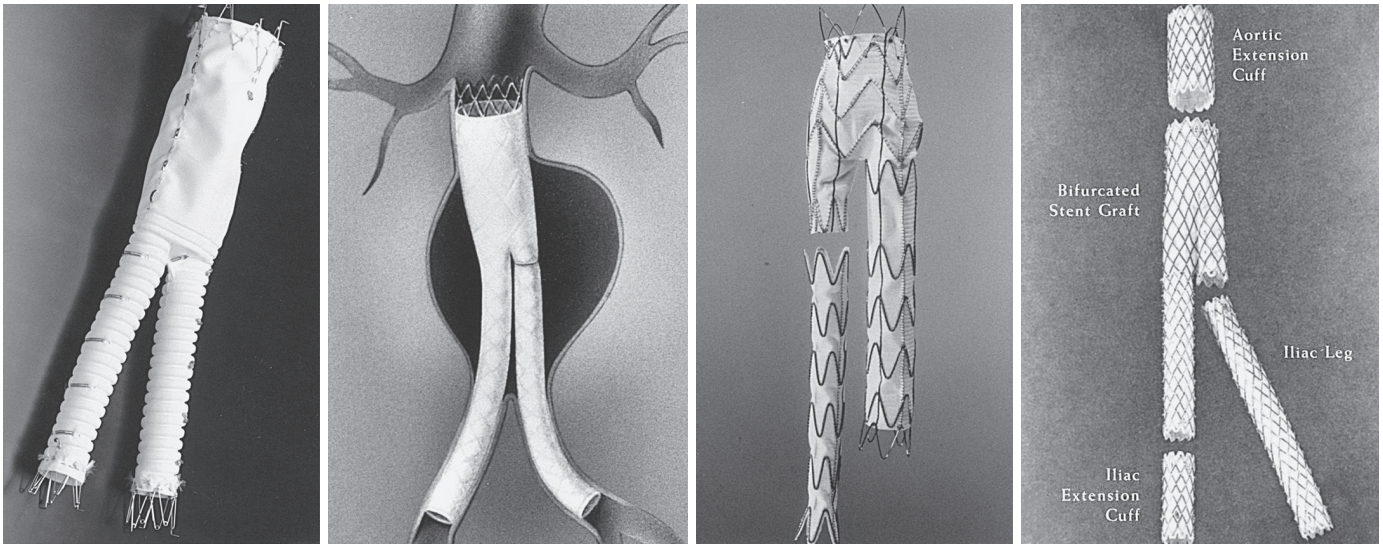

Fig. 28.8 Various types of endovascular grafts. (Adapted from Cronenwett JL, Johnston KW. *Rutherford's Vascular Surgery.* 7th ed. Vol. 2. Philadelphia: Saunders; 2010:1363-1383.)

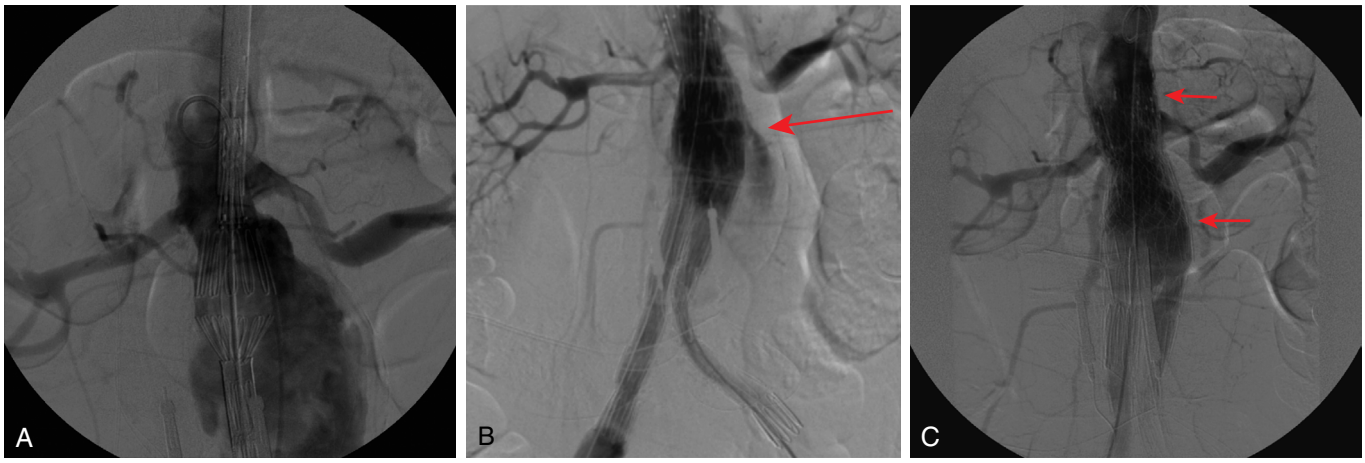

Fig. 28.9 (A) Short aortic neck immediately before endovascular aneurysm repair. (B) Type IA endoleak *(arrow)* after initial endograft placement and molding balloon angioplasty. (C) Resolution of the type IA endoleak after placement of a 5010 giant Palmaz stent (*arrows* show proximal and distal extent). (From Cronenwett JL, Johnston W. *Rutherford's Vascular Surgery*. 8th ed. Vol. 2. Philadelphia: Elsevier; 2014:1348.)

TABLE 28.7 Classification of Types of Endoleak

Classification	Description	Treatment
Type I endoleak	Attachment site leaks	Proximal or distal graft extension Secondary endograft Open repair
Type II endoleak	Branch leaks (i.e., lumbar artery, renal artery, internal iliac artery, inferior mesenteric artery)	Monitoring for enlargement Laparoscopic clip application Embolization
Type III endoleak	Graft defect (fabric tear, modular disconnection)	Secondary endograft Open repair
Type IV endoleak	Graft wall fabric porosity/suture holes	Observation Open repair
Endotension	Systemic pressure in aneurysm sac despite no evidence of endoleaks	Secondary endograft Open repair

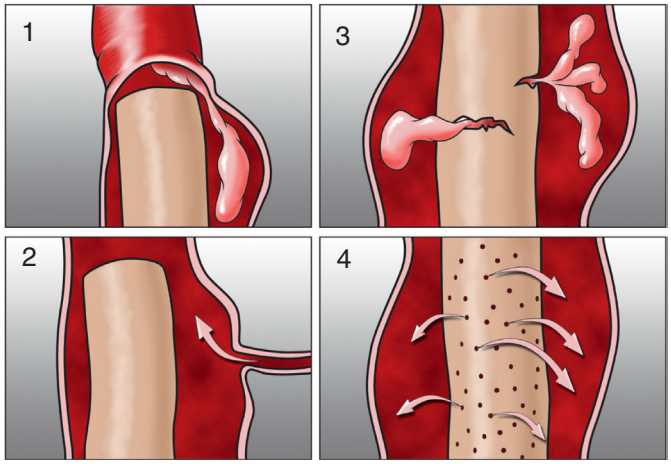

Fig. 28.10 *1,* Type I endoleak; *2,* type II endoleak; *3,* type III endoleak; *4,* type IV endoleak.

will maximize the benefit and minimize complications in patients with a range of aneurysmal disease.

Endoleak (Fig. 28.9), which was noted earlier as persistent blood flow and pressure (endotension) between the endovascular graft and the aortic aneurysm, is a serious complication of this procedure. Types of endoleaks are listed in Table 28.7 and shown in Fig. 28.10. Endoleak diagnosed by postoperative CT scan has been reported to occur in 15% to 52% of patients.[159] The majority of endoleaks are type II, and 70% close spontaneously within the first month after implantation.[160] Type II endoleaks are caused by collateral retrograde perfusion and are associated with long-term complications. Types I and III endoleaks are caused by device-related problems and most often occur soon after EVSG implantation.[161] The most frequent interventions used to correct these complications include implantation of a second endograft or open repair.[162] One long-term study has demonstrated that EVAR yields good results as compared to an open repair, but the overall durability of the open surgical procedure is superior.[163]

As described in the EVAR 1 study, reinterventions due to endoleak were required in three times as many patients who had EVAR as compared to an open procedure. Of these endoleaks requiring reintervention, 7% were discovered within 1 month of implantation, and another 13% occurred within 4 years postoperatively.[148] A comparison of the outcomes comparatively evaluating EVAR and open AAA repair (in nearly 40,000 patients) has been reported. Perioperative mortality (≤30 days postoperative) and the risk of mortality 3 years postoperatively were lower after EVAR compared with OSR. At 3 years postoperatively, the risk of mortality was similar to patients having an OSR. Follow-up interventions were more common after EVAR, most often due to the EVSG. Lastly, the risk of rupture was greater with EVAR within an 8-year postoperative period.[122]

As EVSG-related endoleaks are the most likely causes of late aneurysm rupture, post-EVAR surveillance is an important factor in avoiding the risk of late aneurysm rupture.[164] A large proportion of late ruptures are amenable to endovascular treatment. Postoperative follow-up care for patients who have undergone EVAR is vital because long-term outcomes have not been quantitatively established. Physical examination and contrast-enhanced CT scans are recommended at 1, 6, 12, and 18 months postoperatively, and then annually.[165] Additionally, abdominal x-rays should be obtained on a regular basis. Lifelong

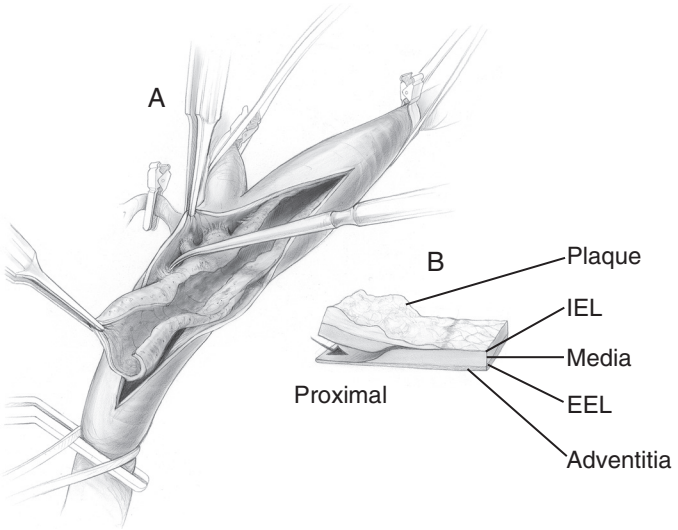

Fig. 28.11 (A) Removal of plaque from carotid artery. (B) Formulation of plaque on the intima of the carotid artery. *EEL,* External elastic lamina; *IEL,* internal elastic lamina. (From Zarins CK, Gewertz BL. *Atlas of Vascular Surgery*. 2nd ed. Philadelphia: Churchill Livingstone; 2009.)

radiographic evaluation and surveillance is necessary to monitor aneurysm size, graft migration, and endoleak. Intensive follow-up care, the need for reinterventions, and the cost of the endograft make EVAR more expensive than open repair.[166]

CEREBROVASCULAR INSUFFICIENCY AND CAROTID ENDARTERECTOMY

Carotid endarterectomy (CEA) is the second most common vascular operation performed in the United States every year (the first being coronary revascularization). Cerebrovascular accidents (CVAs; strokes) are the third leading cause of death in the United States.[167] More strokes are caused by cerebral ischemia than by intracranial hemorrhage. In carotid atherosclerotic disease, subintimal fatty plaques can increase in size over time and incrementally occlude the vascular lumen, which results in decreased cerebral blood flow (CBF). The plaque may rupture and release fibrin, calcium, cholesterol, and inflammatory cells. This phenomenon can lead to abrupt occlusion of the lumen from thrombosis due to platelet activation, or an embolus may form and decrease CBF distal to the carotid artery. In each scenario, an abrupt decrease in CBF leads to transient ischemic attacks (TIAs) or strokes. Note the anatomic details associated with the removal of plaque and the involvement within the layers of the carotid artery in Fig. 28.11.

More than half of all strokes are preceded by a TIA. The Framingham study reported that the risk of a stroke was 30% 2 years after a TIA had occurred and approximately 55% 12 years after a TIA had occurred.[168] It is this increased risk of stroke associated with TIA that provides the rationale for the use of CEA, the surgical procedure in which the internal carotid artery is incised and the plaque within the carotid arterial lumen is removed to improve CBF.

Indications

Since 1954, specific indications for and expected outcomes of CEA have been the subject of heated debate. Ischemic stroke accounts for approximately 80% of first-time strokes and is primarily caused by atheromatous plaques. The initial indication for CEA was symptomatic stenosis but not complete occlusive carotid disease. This presentation occurs in most patients who undergo carotid surgery. Some centers have extended the indications to include evolved (nondense), nonhemorrhagic strokes and asymptomatic severe stenosis or lesser stenosis associated with contralateral occlusive disease. The North American Symptomatic CEA Trial concluded that CEA for patients with recent hemispheric TIAs and high-grade stenosis (70%–99%) had a risk reduction of 65% for the development of an ipsilateral stroke 2 years after surgery, compared with patients whose conditions were medically managed.[169] The Executive Committee for the Asymptomatic Carotid Atherosclerosis Study demonstrated that asymptomatic patients with at least 60% carotid artery stenosis who underwent CEA had a 53% lower 5-year risk of ipsilateral stroke than patients who were treated medically.[170] Other widely reported large-scale studies, including the Asymptomatic Carotid Atherosclerosis Study (ACAS) and the European Asymptomatic Carotid Surgery Trial (EACST), demonstrated a benefit of CEA with medical therapy (aspirin and atherosclerotic risk factor reduction) over medical therapy alone for patients with carotid stenosis in the 60% to 99% range.[171,172] These trials showed a similar absolute and relative reduction in risk for stroke of approximately 5% and 50%, respectively, at 5 years for CEA over medical therapy. Symptomatic patients are at a higher risk than asymptomatic patients for perioperative adverse events. However, the benefit of CEA in patients with recent ipsilateral carotid territory symptoms and moderate to severe carotid stenosis is much greater than the benefit of CEA in asymptomatic patients.[173]

Morbidity and Mortality

The surgical outcomes reported for CEA vary due to differences in patient populations and varying degrees of surgical expertise. Other variables that cannot be stratified in studies but may affect patient outcomes include the state of collateral flow through the circle of Willis, the presence of concurrent atherosclerotic disease in the cerebral vasculature, the size and morphology of the offending plaque, the specific presenting symptoms, and the presence of concurrent cardiovascular disease.[174] Carotid artery stenosis is the primary cause of approximately 20% of all strokes.[175] The recommended acceptable perioperative stroke rates are less than 3% in asymptomatic patients, less than 5% in symptomatic patients, and 10% or less in patients with recurrent disease or existing strokes.[176] Morbidity rates related to CEA have been reported to be at or below these recommended limits.[176,177] The perioperative MI rate of 2% to 5% illustrates the global nature of atherosclerotic disease and represents the greatest contribution to overall morbidity. The perioperative mortality rate for CEA is approximately 0.5% to 2.5%,[178,179] and the long-term postoperative stroke incidence ranges from 1% to 3% per year.[180] In a multicenter cohort of black and white adults in the United States, the incidence and mortality rates associated with a stroke decreased from 1987 to 2011. The decreases varied across age groups but were similar across sex and race.[181]

Patient Selection

The risks associated with CEA and stroke must be measured against the risks associated with undergoing medical management. The patients who benefit most from CEA are those with stenosis of greater than 70%; it is less beneficial in symptomatic patients with 50% to 69% stenosis.[175] Surgical intervention is most beneficial in men who are older than 75 years and are within 2 weeks of their last ischemic event.[182] As mentioned previously, the Framingham study identified the incidence of stroke after TIAs and demonstrated an increased risk of stroke in patients with untreated disease. Preoperative neurologic dysfunction was found to be the most significant factor for predicting postoperative stroke incidence (4%). Several conditions that can increase the risk of perioperative complications include severe preoperative hypertension, CEA performed in preparation for coronary artery bypass, angina, internal carotid artery stenosis near the carotid siphon, age older than

BOX 28.12 Factors Contributing to Morbidity During Carotid Endarterectomy

- History of stroke
- Operative timing
- Hyperglycemia
- Multiple comorbidities
- Age
- Contralateral carotid artery disease
- Progressing stroke
- Ulcerative lesion
- Intraoperative hemodynamic instability
- Surgery with shunt
- Surgery without shunt

75 years, and diabetes mellitis.[183] Box 28.12 identifies various factors that contribute to morbidity during CEA.

In the past, emergent CEA or carotid artery stenting (CAS) after acute CVA was not considered safe. However, more evidence suggests that these surgical interventions, after an acute neurologic ischemic episode, are performed to reduce the risk of neurologic damage and recurrent stroke. Even within 72 hours after tissue plasminogen activator administration for thrombolysis, patients were not at risk for increased complications associated with the surgical procedure.[184]

Diagnosis

The neurologic symptoms associated with cerebral vascular dysfunctions such as TIAs and strokes are often related to decreased CBF. Whereas there are multiple causes for symptoms such as lightheadedness, altered levels of consciousness, aphasia, and acute motor deficits, these deficits warrant testing to determine if carotid stenosis is present. Asymptomatic carotid bruits may be a sign of carotid artery disease. However, not all carotid bruits indicate the presence of significant carotid artery disease. Amaurosis fugax manifests as monocular blindness and occurs in 25% of patients with high-grade carotid artery stenosis. This syndrome is believed to be caused by microthrombi that migrate into the internal carotid artery and decrease the blood supply of the optic nerve via the ophthalmic artery. Standard diagnostic imaging techniques used to assess the extent of carotid disease include duplex ultrasonography, digital subtraction angiography, CTA, and magnetic resonance angiography.[185]

Preoperative Assessment

The presence of concurrent CAD and carotid stenosis is well documented. Although stroke is a devastating consequence of CEA, MI contributes more often to poor surgical outcomes than stroke. Although coronary angiography may not be justified in all patients undergoing CEA, a systematic approach for identifying CAD and its subsequent risks should be performed before elective surgery.

Patients with no significant medical history, normal physical examination, and normal ECG should proceed directly to surgery; these patients have low surgical risks. If abnormal cardiac function is discovered during the preoperative assessment, further evaluation and testing should be performed. The presence of significant comorbidities will determine the extent to which further preoperative testing is appropriate. Box 28.13 lists preoperative risk factors in patients having CEA. Preoperative pharmacologic optimization for patients with vascular and cardiac disease was discussed earlier in this chapter. Vascular surgery is associated with an increased risk of major adverse cardiac events.[186] For a complete discussion of cardiac optimization, see Chapter 20. The American College of Cardiology Foundation (ACCF) and American Heart Association (AHA) recommendations for perioperative cardiac assessment are in Table 28.8.

BOX 28.13 Preoperative Risk Factors for Patients Scheduled for CEA

- Neurologic (cerebrovascular accident)
- Coronary artery disease
- Hypertension
- Diabetes
- Renal disease
- Thromboembolism

CEA, Carotid endarterectomy.

Intraoperative Considerations

Cerebral Physiology

CBF can remain relatively constant at different cerebral perfusion pressures as a result of cerebrovascular autoregulation. Cerebral perfusion pressure can be expressed as the difference between MAP and intracranial pressure (ICP). During CEA, ICP is usually not elevated, therefore MAP plays the predominant role in determining cerebral perfusion pressure. When MAP is maintained between 60 and 160 mm Hg, CBF remains constant. However, the adverse effects of chronic systemic hypertension shift the patient's cerebral autoregulatory curve to the right, and therefore a higher than normal MAP may be required to ensure adequate cerebral perfusion. CBF is also influenced by arterial carbon dioxide and oxygen concentrations, as well as anesthetic agents. Profound hypocarbia causes cerebral vascular constriction by decreasing CBF. Cerebral steal can occur with hypercarbia as it leads to cerebral vascular dilation in cerebral vessels. However, in those areas of the brain that are at risk of developing ischemia caused by atherosclerotic plaques, the cerebral vessels are maximally dilated. Therefore causing profound cerebral vascular dilation decreases CBF and increases the potential for regional ischemia. Inhalation agents increase CBF due to cerebral vascular dilation in a dose-dependent fashion. Anesthetic agents, with the exception of ketamine, decrease the cerebral metabolic rate of oxygen consumption ($CMRO_2$).

Normal CBF is approximately 50 mL/100 g per min. Neuronal function is generally maintained at levels greater than 25 mL/100 g per min. Blood flow that is less than this critical value jeopardizes cellular function. Decreased perfusion and ischemia can be reflected in changes in consciousness. Cellular death occurs at levels less than 6 mL/100 g per min, as evidenced by the flattening seen on an electroencephalogram (EEG).

Carotid occlusive disease jeopardizes the cerebral perfusion pressure in the ipsilateral artery. Ischemia leads to the disruption of autoregulation and compensatory vasodilation, causing blood flow to become pressure dependent. During CEA, a primary goal is to ensure adequate CBF by maintaining and, if necessary, augmenting MAP.

Cerebral Monitoring

In addition to standard monitoring, direct intraarterial pressure must be continuously assessed via arterial line placement. During CEA, hemodynamic variability frequently occurs. Owing to the high incidence of CAD and neurovascular disease in patients having a CEA, prompt and tight control of blood pressure is imperative.

During repair, the carotid artery cross-clamp is applied distally and proximally to the carotid incision. Various monitoring techniques have been proposed for assessing the adequacy of CBF during this maneuver. A summary of select cerebral monitoring techniques is presented

TABLE 28.8 ACCF/AHA Recommendations for Perioperative Cardiac Assessment

	Scenario	Recommendation
Class I	Patients in need of emergency noncardiac surgery	Proceed directly to the operating room.
	Patients with active cardiac conditions, unstable or severe angina (not stable angina), decompensated heart failure, significant dysrhythmia (high-grade AV block, Mobitz type II block, third-degree AV block, symptomatic ventricular arrhythmias, symptomatic bradycardia, and supraventricular arrhythmias including atrial fibrillation with HR >100 beats/min), and severe valvular disease	Provide perioperative surveillance, risk stratification, and risk factor management.
	Patients undergoing low-risk procedures	Proceed with planned surgery.
	Patients with poor (<4 METs) or unknown functional capacity and no clinical risk factors	
Class IIa	Patients with functional capacity ≥4 METs without symptoms	Proceed with planned surgery.
	Patients with poor (<4 METs) or unknown functional capacity and three or more clinical risk factors* who are undergoing intermediate-risk surgery	Proceed with planned surgery with heart rate control.
	Patients with poor (<4 METs) or unknown functional capacity and one or two clinical risk factors* who are undergoing vascular surgery or intermediate-risk surgery	
	Patients with poor (<4 METs) or unknown functional capacity and three or more clinical risk factors* who are undergoing vascular surgery	Consider further testing if it will change management.
Class IIb	Patients with poor (<4 METs) or unknown functional capacity and three or more clinical risk factors* who are scheduled for intermediate-risk surgery	Consider noninvasive testing if it will change patient management.
	Patients with poor (<4 METs) or unknown functional capacity and one or two clinical risk factors* who are scheduled for vascular surgery or intermediate-risk surgery	

*Clinical risk factors include ischemic heart disease, compensated or prior heart failure, diabetes mellitus, renal insufficiency, and cerebrovascular disease.

Class I recommendations suggest that procedures/treatments should be performed; class IIa recommendations suggest that it is reasonable to perform the procedure/treatment; class IIb recommendations imply that the procedure/treatment should be considered; and in class III the intervention should not be performed because it may not be helpful and may potentially be harmful to the patient.

ACCF/AHA, American College of Cardiology Foundation/American Heart Association; *AV,* atrioventricular; *HR,* heart rate; *METs,* metabolic equivalents.

From Cronenwett JL, Johnston KW. *Rutherford's Vascular Surgery*. 9th ed. Philadelphia: Elsevier; 2019: 507.

BOX 28.14 Cerebral Monitoring Modalities During General Anesthesia for CEA

- Electroencephalogram: assesses cortical electrical function
- Somatosensory-evoked potential: assesses sensory-evoked potentials
- Carotid stump pressure: assesses perfusion pressure in the operative carotid artery
- Transcranial Doppler: assesses blood flow velocity in the middle cerebral artery
- Cerebral oximetry: assesses cerebral regional oxygen saturation (near infrared spectroscopy)

CEA, Carotid endarterectomy.

in Box 28.14. Each of these monitoring modalities has limitations. The most sensitive and specific measure of adequate CBF is responsiveness in an awake patient.

EEG monitoring constitutes the gold standard in identifying neurologic deficits related to carotid artery cross-clamping.[175,187] EEG has demonstrated reliability in monitoring cortical electrical function.[188] Loss of β-wave activity, loss of amplitude, and emergence of slow-wave activity are all indicative of neurologic dysfunction. Limitations surrounding EEG monitoring include (1) the effect of blood pressure, temperature, and anesthetic agents on monitoring; and (2) the fact that this modality detects EEG changes only on the superficial layers of the brain and not in deep cortical structures such as the brainstem.

Carotid stump pressure has been used as a means of assessing collateral flow.[189] After the carotid cross-clamp is placed, blood flow from the nonoperative carotid artery and the basilar artery provides blood flow to the circle of Willis. A catheter is placed into the distal portion (above the cross-clamp) of the operative internal carotid artery, and the pressure can be monitored. Carotid stump pressure is a gross measurement of the pressure within the circle of Willis. A carotid stump pressure of less than 40 to 50 mm Hg reflects neurologic hypoperfusion and is a criterion for shunt placement. However, there is no correlation between stump pressures and EEG changes.[177] In one study, a carotid stump pressure of less than 50 mm Hg had a positive predictive value for only 36% of patients who exhibited ischemic changes on EEG during carotid artery cross-clamping.[190] A combination of stump pressure and either transcranial Doppler (TCD) or EEG appears to improve the detection of cerebral ischemia during carotid artery cross-clamping.[191]

SSEP monitoring can be used to identify inadequate CBF during cross-clamping; however, false-positive results can occur. In addition, SSEPs reflect the sensory integrity of the spinal cord and the brain, therefore a motor deficit can occur despite a normal SSEP waveform. Additionally, there are no values for decreased amplitude and increased latency that definitively correlate with cerebral ischemia. Monitoring both SSEP and EEG is more sensitive for predicting perioperative deficits as compared to using either monitoring modality in isolation. Patients who experience perioperative strokes are 17 times more likely to have a change in either EEG or SSEP than other patients.[192]

TCD velocity monitoring has been used to detect adverse cerebral events during CEA. TCD is noninvasive and measures cerebrovascular dynamics through the CBF velocity. The use of TCD during CEA to determine if carotid shunt placement is necessary is a reliable method to decrease adverse neurologic outcomes.[193] TCD can also be used during the postoperative period to detect ischemia and the presence of cerebral hyperperfusion syndrome (CHS).

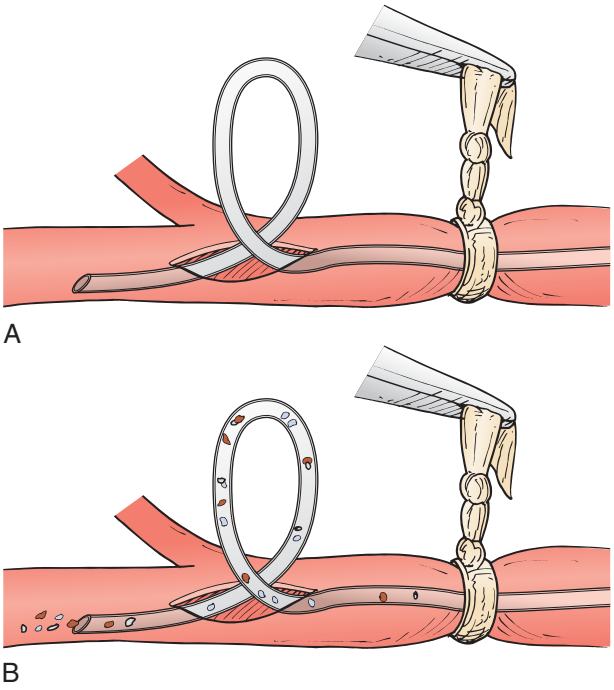

Fig. 28.12 Pitfalls of carotid shunt placement. (A) Potential traumatic injury to the distal internal carotid artery intima. (B) Potential for embolization of atherosclerotic debris or air. (From Cronenwett JL, Johnston W. *Rutherford's Vascular Surgery*. 7th ed. Vol. 2. Philadelphia: Saunders; 2010:1452.)

Near infrared spectroscopy (NIRS) measures cerebral oxygenation. A greater than 20% reduction in regional cerebral oxygenation coincides with regional and global cerebral ischemia during CEA.[194] Despite the established advantages of using NIRS monitoring in cardiac surgery, its routine use is less established during noncardiac procedures. Both NIRS and TCD monitoring are independently accurate in predicting the need for selective shunting by detecting cerebral ischemia during CEA and general anesthesia.[195] As compared with stump pressure monitoring, cerebral oximetry more accurately predicts cerebral oxygenation.[196] Box 28.14 outlines the cerebral monitoring modalities that can be used during general anesthesia for CEA.

Cerebral Protection

The major objective during carotid artery revascularization is to maintain cerebral CBF and oxygenation. Prevention of cerebral ischemia can be accomplished in one of two ways: by increasing collateral flow (placement of intraluminal shunt) or by decreasing cerebral metabolic requirements (anesthetic medications). Multiple interventions are available for cerebral protection, including avoiding hyperglycemia, hemodilution, maintenance of normocarbia, and tight control of arterial blood pressure. Anesthetics, except for etomidate, have cerebral protective properties and may be used to minimize the degree of cerebral ischemia. Shunt placement is commonly used to allow blood to flow proximally and distally to the carotid cross-clamp during intimal plaque dissection. Potential complications associated with carotid shunt placement are depicted in Fig. 28.12.

Cerebral ischemic events are most often the result of embolic complications and frequently occur during the postoperative period. The need for shunt placement is based on surgeon preference and information obtained using intraoperative monitoring techniques to determine CBF. Furthermore, propofol decreases $CMRO_2$ to 40% below normal values.[114] Dexmedetomidine also decreases cerebral oxygen consumption and CBF in animal models.[197] During transient focal ischemia, propofol decreases the $CMRO_2$, which results in cerebral protection. The disadvantages of administering propofol during CEA surgery include myocardial depression and hypotension. The inhalation agents also decrease $CMRO_2$ in a dose-dependent fashion. Nitrous oxide should be avoided due to the potential for pneumocephalus from microbubble expansion after carotid artery unclamping.[175,182]

Blood Pressure Control

The presence of hypertension in patients with cerebrovascular disease is well known, and therefore one of the most challenging aspects of care associated with anesthesia for CEA is blood pressure control. Patients with cerebral insufficiency are vulnerable to perioperative blood pressure instability. Hypotension occurs in 10% to 50% of patients who undergo CEA and is believed to be the result of carotid sinus baroreceptor stimulation. Conversely, 10% to 66% of patients experience hypertension, which is attributed to surgical manipulation of the carotid sinus.[198] Preoperative blood pressure control, volume status, and depth of anesthesia can also contribute to intraoperative hemodynamic instability. During carotid artery cross-clamping, maintaining the MAP at 20% or greater of the patient's preoperative mean pressure decreases postoperative cognitive dysfunction.[199]

Blood pressure control must begin in the preoperative phase. All patients should continue taking their antihypertensive medications until the time of surgery. Patients with systolic blood pressure greater than 180 mm Hg may be at increased risk of stroke and death.[182] Additional pharmacologic agents may be required in the preoperative period, especially during the insertion of intravenous and intraarterial catheters, to reduce increases in heart rate and blood pressure. The induction of anesthesia, the initial incision, dissection, manipulation of the carotid sinus, and emergence from anesthesia are all events that precipitate blood pressure fluctuations. The use of pharmacologic adjuncts, such as short-acting β-adrenergic blockers, may stabilize blood pressure during induction and emergence. Continuous intravenous use of nitroglycerin or sodium nitroprusside should be available to treat hypertension. Patients with chronic hypertension are predisposed to dramatic decreases in blood pressure after the induction of general anesthesia. This condition must be treated promptly and can be successfully managed by providing intravenous fluids or administering appropriate vasopressors. Hypotension and bradycardia, which result from carotid sinus baroreceptor manipulation, may be inhibited by stopping surgical stimulation, infiltrating the region with local anesthesia, and administering an anticholinergic (as necessary).

Anesthetic Management

The anesthetic objectives for vascular surgery are similar to those for any type of elective procedure: to provide analgesia and amnesia, to facilitate surgical intervention, and to minimize operative morbidity and mortality. Goals that are specific to CEA include maintaining cerebral and myocardial perfusion and oxygenation, minimizing the stress response, and facilitating a smooth and rapid emergence. However, it may be difficult to maintain the integrity of one system without adversely affecting the other. For example, raising the arterial blood pressure to augment cerebral perfusion can increase myocardial oxygen demand, which may lead to ischemia. In addition, significantly decreasing blood pressure can lead to cerebral hypoperfusion. Therefore the anesthetic goal is to optimize perfusion to the brain, minimize myocardial workload, ensure cardiovascular stability, and

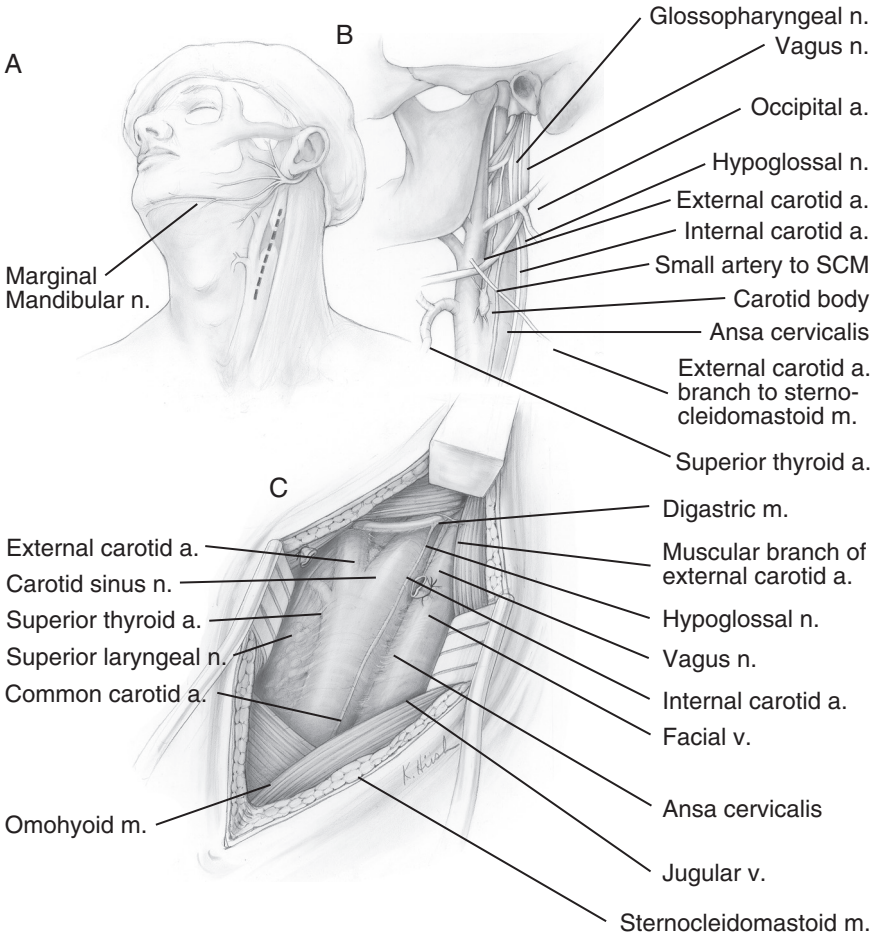

Fig. 28.13 Carotid endarterectomy. (A) Incision site. (B, C) Anatomic structures presented at the carotid surgical site. *a,* Artery; *m,* muscle; *n,* nerve; *SCM,* sternocleidomastoid; *v,* vein. (From Zarins CK, Gewertz BL. *Atlas of Vascular Surgery.* 2nd ed. Philadelphia: Churchill Livingstone; 2009.)

allow for rapid emergence. Anticoagulation is achieved via administration of heparin (50–100 units/kg) prior to carotid artery cross-clamping. The decision to administer protamine upon completion of the surgical procedure is based on the surgeon's impression. Protamine administration is associated with a reduction in bleeding complications without increasing major thrombotic outcomes, including stroke, MI, or death after CEA.[200] Protamine administration is associated with hypotension. Anaphylaxis is a rare but life-threatening side effect. An understanding of the physiology of the cerebrovascular system is important for optimal anesthetic management. Fig. 28.13 illustrates the anatomy of structures in this region. This knowledge enables the selection of appropriate monitoring and anesthetic techniques that will protect and improve cerebral and myocardial perfusion.

Anesthetic Selection

The long-standing question has been whether there is an advantage of regional vs general anesthesia for CEA. The General Anesthetic versus Local Anesthetic for Carotid Surgery Trial (GALA), as well as a Cochrane meta-analysis, indicate no significant difference between the two anesthetic techniques.[201,202] The anesthetic selection is based on the surgeon's preference, the patient's condition, and the preoperative evaluation. Advantages of a regional technique are that an awake patient can respond to commands and allow for continuous assessment of neurologic function. If the patient's level of consciousness decreases as a result of cerebral hypoperfusion, surgeons can then place a shunt. Other potential benefits include improved patient satisfaction, decreased cost, and minimization of potential postoperative cognitive effects associated with general anesthesia.[203-205] Disadvantages include patient agitation and inability to remain still, minimal airway control, seizure or stroke during carotid artery clamping, and limited ability to give anesthetic medications. Advantages of general anesthesia include the ability to perform more extensive and difficult surgical procedures, better airway control, the ability to administer cerebral protectants, and improved blood pressure control.[175,182]

Regional anesthesia. A regional anesthetic (RA) technique during CEA requires a deep and superficial cervical plexus block, which is accomplished by anesthetizing cervical nerves II to IV.[175] Superficial cervical blocks do not anesthetize the region at the angle of the mandible, which is innervated by the trigeminal nerve. Local infiltration may be required. As noted previously, the greatest advantage of RA is the ability to directly assess neurologic function in an awake individual. Assessing level of consciousness is the most effective method of assessing the adequacy of CBF and detecting cerebral ischemia. In fact, assessment of consciousness in the awake patient may be more sensitive than conventional EEG in detecting cerebral ischemia. Researchers reviewed data from 399 patients who underwent CEA while receiving either GA or RA. The authors concluded that perioperative strokes occurred less often when RA was administered, especially in high-risk patients.[206] Another study compared middle cerebral artery blood flow velocity using TCD

monitoring in patients undergoing CEA with either local or GA.[207] There is significant evidence to support that the preservation of cerebral circulation is better maintained in patients who receive local anesthesia.[208-211] In addition, further evidence suggests that patients who receive GA are more likely to receive a shunt compared with those who receive RA.[212] RA resulted in fewer hemodynamic fluctuations and fewer intraoperative vasoactive medication requirements as compared with GA during perioperative management of CEA.[213] However, despite these seemingly physiologic advantages, no differences were observed in outcomes between the RA and GA groups. The use of RA has been associated with shorter operative times, fewer cardiopulmonary complications, and a shorter duration of postoperative hospitalization.

One limiting factor for the use of RA is patient tolerance. The individual is mildly sedated; therefore preoperative education is essential, and patient cooperation during surgery is vital. Anxiety, fear, and apprehension can initiate sympathetic stimulation, and as a result extreme hemodynamic responses can occur. Deep sedation, which is sometimes necessary in an apprehensive patient, may confound the neurologic assessment, subsequently negating the advantages of a regional technique. Additionally, hypercarbia can result from hypoventilation, and dysphoria is more likely to occur. Furthermore, converting to a GA technique once surgery has begun can be problematic. Symptoms indicating that adequate cerebral perfusion has been compromised include dizziness, contralateral weakness, decreased mentation, and loss of consciousness. In the event that this scenario occurs, immediate shunt placement is warranted. If symptoms associated with cerebral hypoperfusion do not resolve rapidly with shunt placement, emergent airway management is necessary.

General anesthesia. Although the use of RA has numerous advantages, GA is also used during CEA. Perhaps the greatest benefit of this technique is that it counters the most cited disadvantage of regional anesthesia: lack of patient tolerance during the procedure. GA promotes a motionless field during surgery. In addition, inhalation agents may provide hemodynamic stability and may have beneficial effects on cerebral circulation.[214] By decreasing cerebral and cardiac metabolism, the inhalation agents provide a degree of protection against ischemia, an effect called anesthetic preconditioning.[215-217]

There is no scientific evidence to suggest that patient outcome is improved when inhaled agents are used compared to narcotic-based techniques. In studies of inhalation agents, the critical regional CBF (the blood flow measurement for which EEG signs of ischemia occur) during isoflurane anesthesia was less than when other volatile anesthetics were used.[214,218] The effects of sufentanil on cerebral hemodynamics were similar to those of isoflurane.[219] Remifentanil can be used, and its rapid metabolism improves neurologic recovery. The inhalation agents may alter the monitoring methods used for detecting cerebral ischemia, such as EEG and SSEP monitoring. In these cases, GA may require modification, and direct communication between the anesthesia and surgical teams is vital. When carotid artery cross-clamping without shunting occurs, MAP values must be 20% or greater of the patient's preoperative MAP to help ensure adequate cerebral perfusion through the contralateral carotid artery and decrease the possibility of postoperative cognitive dysfunction.[199] The use of nitrous oxide during CEA can potentially increase the incidence of a clinically significant pneumocephalus. During shunt placement and carotid artery cross-clamp release, microbubbles can be entrained into the carotid artery blood flow. Nitrous oxide is also known to cause hyperhomocysteinemia. Increased homocysteine can increase the postoperative cardiac risk and long-term mortality and increase the potential for carotid artery restenosis.[220,221] If nitrous oxide is used, it should be discontinued before removal of the carotid artery cross-clamp.[175,182]

In summary, there is no scientific consensus supporting the notion that a specific anesthetic technique is superior in decreasing perioperative morbidity and mortality post-CEA. Ensuring adequate cerebral and cardiac perfusion by treating hypertensive and hypotensive episodes aggressively is important. An anesthetic plan that allows for a rapid assessment of neurologic function at the completion of surgery should be selected.

Postoperative Considerations

Perhaps the most common problem experienced in the postoperative period is hypertension. Although the specific cause remains unclear, postoperative hypertension is likely related to changes in sensitivity of the carotid baroreceptor reflex. A systolic blood pressure greater than 180 mm Hg is associated with an increased incidence of TIA, stroke, or MI.[183] Patients with a systolic blood pressure of 145 mm Hg or less tend to have fewer postoperative complications.[169] A postoperative blood pressure of 140/80 mm Hg or less is recommended.[222] Chronic hypertension during the postoperative period can lead to the development of CHS as described later. Postoperative hypertension often resolves within 24 hours after surgery. Postoperative hypotension is less common but can be more difficult to treat because raising the blood pressure increases myocardial oxygen demand. Reestablishing normal pressures can be accomplished by careful titration of fluids and vasopressors.

Although an uncommon complication, carotid artery hemorrhage can occur in the postoperative phase. Hemorrhage is a devastating event that requires immediate surgical intervention. One of the initial manifestations of hemorrhage may be upper airway obstruction, which may make reintubation difficult due to tracheal deviation. Emergency management of a patient with airway compromise as a result of carotid artery hemorrhage and hematoma includes immediate evacuation of the hematoma. In addition, recurrent laryngeal nerve damage can occur, which routinely manifests as inspiratory stridor. Respiratory insufficiency can be problematic for patients who have preexisting respiratory conditions. Tension pneumothorax also can occur because the apices of the lungs extend above the clavicles toward the surgical site. Treatment includes immediate lung reexpansion via chest tube insertion or needle decompression. Damage to the carotid body can lead to blunting of the chemoreceptor reflex, necessitating the administration of supplemental oxygen. Lastly, CHS may result from increased blood flow to the brain as a result of a loss of cerebral vascular autoregulation. The mechanism of action that causes this phenomenon is unclear; however, it is hypothesized that CHS may occur as a result of chronic cerebral ischemia or altered cerebrovascular autoregulation. Signs and symptoms of CHS include severe headache, visual disturbances, altered level of consciousness, and seizures. CHS may occur more often in patients who have had a contralateral CEA within the last 3 months and undergo a second CEA for occlusion on the ipsilateral side.[223]

The incidence of postoperative stroke after CEA was discussed previously. Unfortunately, even after successful revascularization of the carotid artery, occlusion can recur at a rate of 3% per year.[179] Although symptoms are present in only a small percentage of patients (3%–5%), the incidence of recurrent carotid stenosis may be much larger than that reported because asymptomatic cases may be overlooked.[223] As many as 25% of patients experience a neurocognitive decline up to 1 month after surgery. Patients who are predisposed to this decline are those with diabetes and advanced age.[224] The exact mechanism

responsible for postoperative cognitive dysfunction has not been scientifically identified. Postoperative complications associated with CEA are listed in Box 28.15.

Owing to the anatomic location of and potential neurologic complications after CEA, postemergence neurologic integrity should be assessed. In addition to neurocognitive functioning, clinical assessment of cranial nerve function should be performed (Table 28.9). The anatomic locations of the cranial nerves in relation to the internal, external, and common carotid arteries are shown in Fig. 28.14.

CAROTID ARTERY ANGIOPLASTY STENTING

A less invasive surgical approach for the treatment of carotid artery stenosis is carotid artery angioplasty and stenting (i.e., CAS). Controversy exists regarding the degree of success that this procedure affords as an alternative to CEA. The best application of CAS is still evolving, and many studies comparing stenting with endarterectomy are ongoing. Best practices regarding proper patient selection, technique, and timing of the procedure are still being explored.[225,226] The current incidence of stroke after CEA is approximately 2%. A meta-analysis noted that, compared to CAS, CEA decreases the risk of stroke at 30 days, increases the risk of MI, and has no effect on the risk of death.[227]

The first large multicenter randomized controlled trial comparing CEA vs CAS was the Stenting and Angioplasty with Protection Patients at High Risk for Endarterectomy (SAPPHIRE) trial.[228] The rate of event-free survival at 1-year postsurgery was 88% for the CAS group and 79.9% for the CEA group. The stroke rate after 1 year was lower in the CAS group as compared with the CEA group (6.2% vs 7.9%, respectively). As for cardiac morbidity, the rate of MI for CAS vs CEA was 1.9% vs 6.6% at 30 days postoperatively. Overall, cardiac morbidity was 3% for CAS and 6.2% for CEA. The conclusion drawn from the SAPPHIRE trial was that CAS does not yield inferior outcomes as compared with CEA. However, the study methodology was criticized, and some experts questioned whether the results could be replicated.[229] A new 3-year follow-up report of the SAPPHIRE study group indicates that in patients with severe carotid artery stenosis and increased surgical risk, no significant difference could be shown in long-term outcomes between patients who underwent CAS with an embolic protection device and those who underwent endarterectomy.[230]

The Endarterectomy versus Angioplasty with Symptomatic Severe Carotid Stenosis (EVA-3S) trial was designed to compare the outcomes from CAS vs CEA. The study population included patients with symptomatic carotid stenosis of at least 60%. The study was stopped early because of a high incidence of stroke and death (9.6% compared with 3.9% for CEA at 30 days after surgery). The conclusion was that CEA was superior to CAS for this patient population when considering risk of stroke at 30 days and 6 months postoperatively.[231] Another randomized controlled trial, the Stent-Protected Angioplasty versus Carotid Endarterectomy (SPACE) trial, yielded high but similar statistics for 30-day stroke death rates (6.8% for CAS and 6.3% for CEA).[232]

The goal of the Carotid Revascularization Endarterectomy versus Stenting Trial (CREST), a randomized controlled trial, was to determine which procedure (CAS or CEA) was more effective in preventing stroke and death. Inclusion criteria were patients who were symptomatic and had greater than 50% carotid artery stenosis and those who were asymptomatic with greater than 60% carotid artery stenosis. The preliminary results from the first stage of the trial, which included 1000 patients, are encouraging and compare favorably with CEA. The rate of death or stroke from any cause during the 30 days after the procedure was 3% for asymptomatic patients under 80 years of age and 2.7% for symptomatic patients under 80 years of age.[233] Initial indications were that CAS was associated with an increased incidence of stroke in octogenarians. However, it has now been determined that the incidence of stroke resulting from CAS is similar to the CEA results for all age groups.[234] The CREST data show that the health-related quality of life in patients who underwent CAS is superior to those who underwent CEA for up to 1 year postoperatively.[235] The CREST study was conducted over a 10-year period. It was determined that there were no significant differences between patients who had CEA vs CAS with respect to stroke, MI, and death.[236]

BOX 28.15 Postoperative Complications of Carotid Endarterectomy

- Hemodynamic instability
- Myocardial ischemia/infarction
- Cerebral hyperperfusion syndrome
- Stroke
- Respiratory insufficiency
- Recurrent/superior laryngeal nerve damage
- Hematoma
- Carotid body dysfunction
- Tension pneumothorax
- Acute carotid occlusion

TABLE 28.9 Cranial Nerve Assessment for the Patient Scheduled for CEA

Cranial Nerve	Function	Abnormal Response
VII (facial)	Muscles of facial expression, saliva secretion	Inability to smile symmetrically; contralateral asymmetry indicates possible stroke; nerve injury on ipsilateral side
IX (glossopharyngeal)	Swallowing, pharyngeal muscle	Difficulty swallowing with ipsilateral Horner syndrome (i.e., ptosis, miosis, exophthalmos, reduced sweating)
X (vagus) → superior and recurrent laryngeal nerves	Laryngeal muscle movement	Minor swallowing problems, fatigued voice; vocal cord paralysis, hoarseness, inadequate gag reflex; may test speech by having the patient say "EEE"
XI spinal accessory	Shoulder muscles	Ipsilateral weakness in neck and shoulder with shrugging
XII (hypoglossal)*	Muscles of tongue	Tongue sticks out and moves side to side; tongue droops to ipsilateral side; difficulty with speech and chewing, high-pitched sounds, hoarseness

*This nerve traverses the internal carotid artery.
CEA, Carotid endarterectomy.
From Heffine MS. Care of the vascular surgical patient. In Odom-Forren J, ed. *Drain's Perianesthesia Nursing: A Critical Care Approach.* 6th ed. St Louis: Elsevier; 2013.

Case selection guidelines for CAS are listed in Table 28.10. Prior to CAS, a high-resolution MRI is taken of the patient's aortic arch and carotid arteries, as well as a cerebral angiogram. This allows evaluation of the individual anatomy and angiopathology of the aortic arch, brachiocephalic artery (for right carotid artery stent), or left common carotid artery. The type of sheaths, stents, and cerebral embolic protection device needed can then be determined. Femoral artery access is obtained, and a sheath is then threaded through the aortic arch and into the operative carotid artery. The guide wire/embolic protection device is advanced through the sheath and positioned across the stenotic region. An embolic protection device sequesters emboli during angioplasty and stenting to avoid distal occlusion in cerebral arteries (Fig. 28.15). A distal embolic protection device lowers the risk of intraoperative and postoperative adverse events.[237] This filterlike device is inserted distal to the area of stenosis prior to the angioplasty and stent deployment to catch microthrombi and pieces of plaque that could lodge within the brain. Angioplasty with a 5-mm balloon dilates the carotid artery, and then the stent is deployed. The guide wire/device wire is removed after angiographic confirmation that carotid artery dissection or occlusion has not occurred. Fig. 28.16 shows carotid artery patency after angioplasty and stent placement.

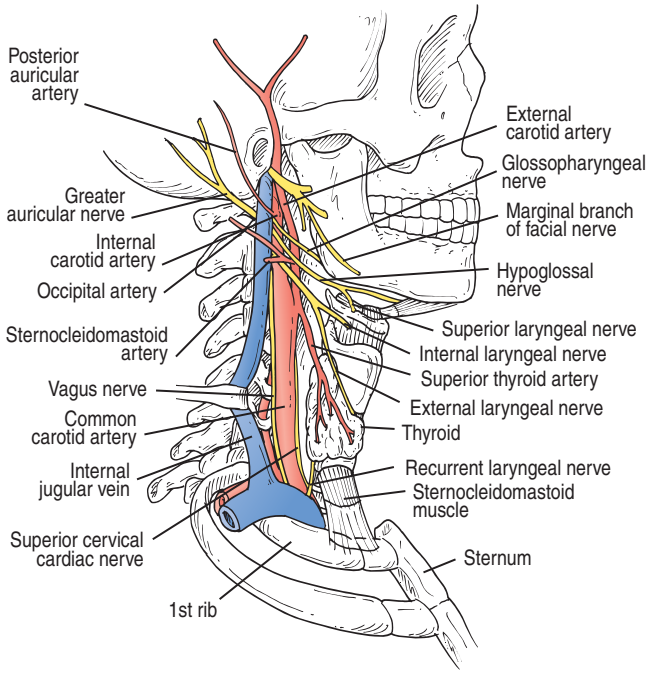

Fig. 28.14 Relationship of the cranial nerves and their major branches to the common, internal, and external carotids in the neck. (From Eisele DW, Smith RV, eds. *Complications in Head and Neck Surgery*. Philadelphia: Elsevier; 2009.)

Anesthetic Considerations

The anesthetic technique used most often for patients having CAS is local anesthesia at the femoral insertion site, minimal sedation, antithrombotic therapy, and observation for hypotension and bradycardia.[238] Anticoagulation is initiated with a heparin bolus (50–100 units/kg) to maintain an activated clotting time greater than 250 seconds.[239] Balloon inflation in the internal carotid artery can stimulate the baroreceptor response, resulting in prolonged bradycardia and hypotension. Glycopyrrolate or atropine can be given prior to inflation to offset this vagal response. Fluoroscopy will be used throughout the surgery

TABLE 28.10 Case Selection for Carotid Artery Stenting

CAS Worse	CAS Better
Clinical Features	
Advanced age (≥80 yr)	COPD
Intolerance of antiplatelet agents	CHD with an abnormal cardiac stress test, unstable angina, or myocardial infarction <1 mo ago
Severe renal dysfunction	Valvular heart disease
	Congestive heart failure (EF <30%)
	Contralateral recurrent laryngeal nerve dysfunction
	Severe obesity
Anatomic Features	
Access related	Previous neck irradiation
Shaggy aorta	Previous radical neck surgery
Eggshell aorta	Tracheostomy
Severely angulated type III aortic arch	Neck immobility
Aortoiliac occlusive disease	Recurrent stenosis
Target vessel related	High lesions (>C2)
Heavy calcification	Contralateral carotid occlusion
Severe tortuosity	
String sign	
Fresh thrombus	
Unstable plaque	

CAS, Carotid artery stenting; *CHD,* coronary heart disease; *COPD,* chronic obstructive pulmonary disease; *EF,* ejection fraction.
Adapted from Cronenwett JL, Johnston W. *Rutherford's Vascular Surgery*. 9th ed. Vol. 2 Philadelphia: Elsevier; 2019.

so it is important that all operating room personnel are protected with lead shielding.

Complications associated with CAS are listed in Box 28.16. The most common complication associated with this procedure is stroke caused by thromboembolism.[240] Interventions for a patient with an acute stroke include airway and hemodynamic management. Immediate CT scan and identification of the presence of an embolus is critical. Neurologic deficits are significantly reversible if CBF is restored within 2 hours. Treatment with catheter-directed recombinant tissue plasminogen activator is approved for acute ischemic stroke that is believed to be caused by an embolus. Catheter-based thrombectomy using snares or balloon angioplasty to restore blood flow and remove the thromboembolic material has also been used successfully. In general, the incidence of CVA is greater with CAS,

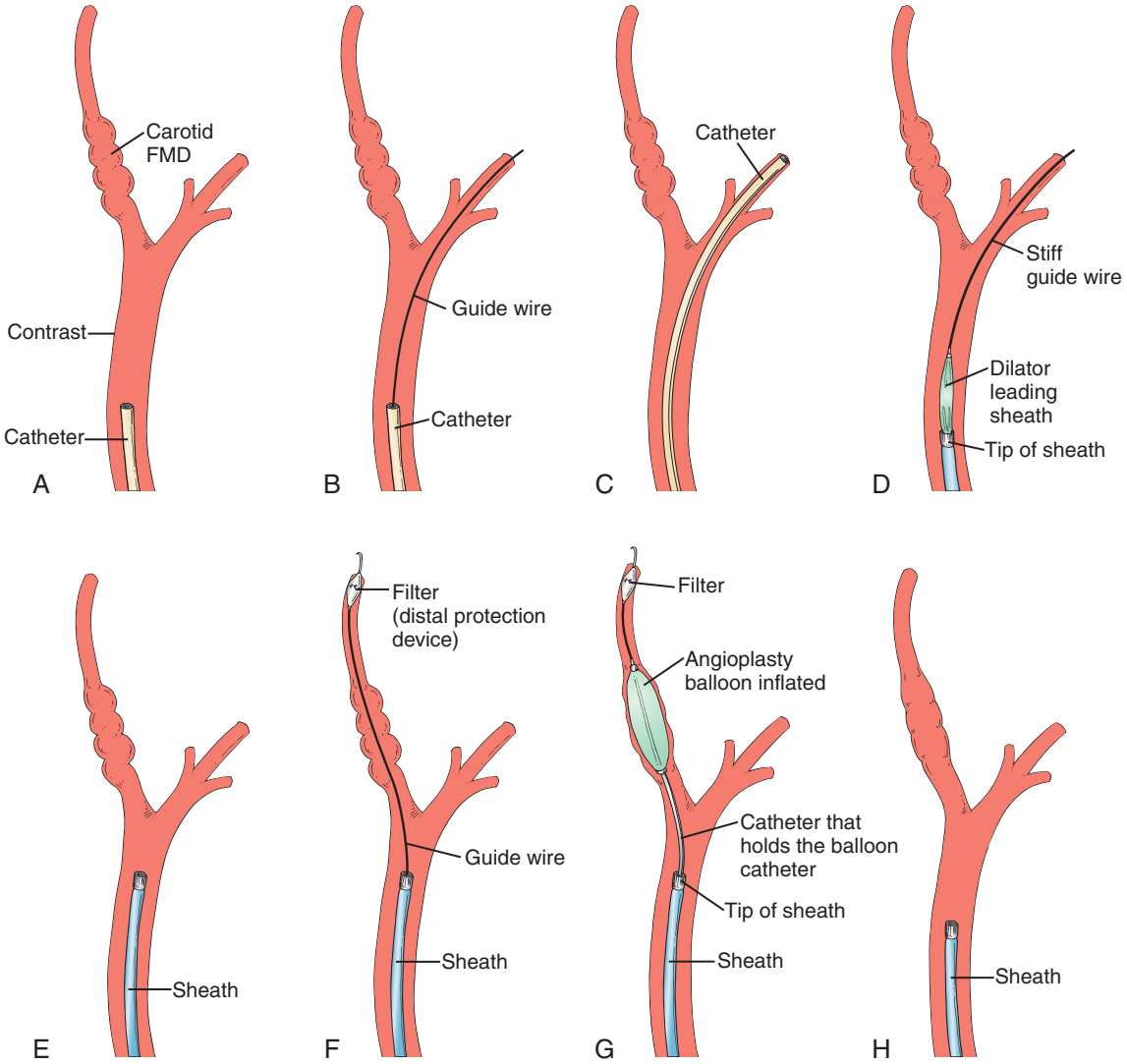

Fig. 28.15 Endovascular technique. (A) Internal carotid artery narrowed by fibromuscular dysplasia. An arteriogram was performed through a carotid catheter. (B) Guide wire placed in the external carotid artery by using a roadmap of the carotid bifurcation. (C) Cerebral catheter advanced into the external carotid artery. (D) Stiff guide wire advanced into the external carotid artery. The carotid access sheath is advanced over the exchange guide wire. (E) Carotid sheath in place with the tip of the sheath in the distal common carotid artery. (F) Cerebral protection device in place in the distal internal carotid artery. (G) Balloon angioplasty of the fibromuscular lesion in the internal carotid artery. (H) After balloon angioplasty, the patency of the lumen has improved significantly. *FMD,* Fibromuscular dysplasia. (From Cronenwett JL, Johnston W. *Rutherford's Vascular Surgery.* 9th ed. Vol. 2. Philadelphia: Elsevier; 2019:1528.)

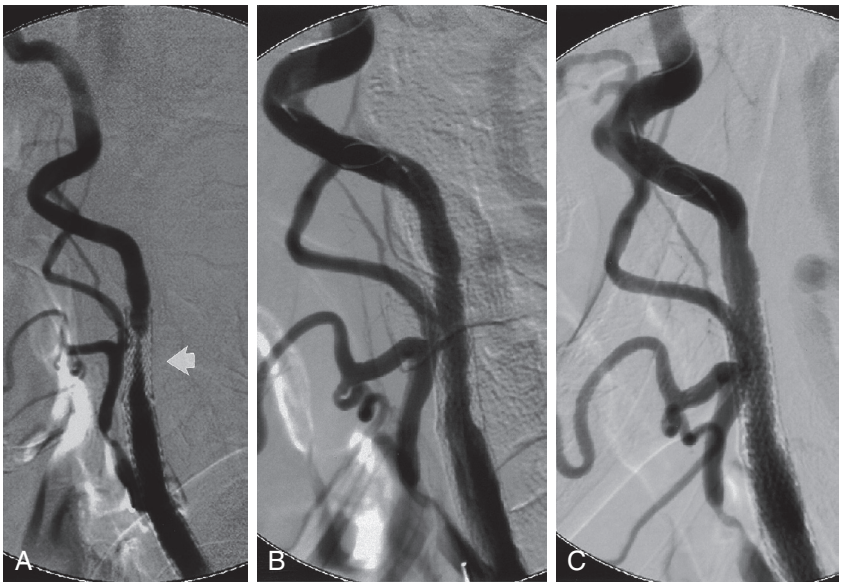

Fig. 28.16 (A) High-grade restenosis of internal carotid artery *(arrowhead)* 11 months after carotid angioplasty/stenting with a wall stent. (B) After angioplasty alone. (C) After placement of a nitinol stent. Note filter protection device in distal internal carotid artery. (From Rutherford RB. *Vascular Surgery*. 8th ed. Vol. 2. Philadelphia: Elsevier; 2014.)

and the incidence of MI is greater with CEA. The long-term morbidity and mortality comparing the two surgical interventions are presently inconclusive. Due to the increased risks of periprocedural stroke, CEA is thought to be the preferred method for the management of asymptomatic carotid stenosis.[241]

Patients typically remain in the postanesthesia care unit for 30 minutes after carotid stent placement and are then transferred to a monitored floor. A carotid duplex scan is performed prior to discharge and then routinely obtained at 6 weeks, 6 months, 1 year, and yearly thereafter. Patients remain on aspirin therapy for anticoagulation for life.[242]

BOX 28.16 Complications Associated With Carotid Artery Stenting

- Stroke
- Myocardial ischemia/infarction
- Bradycardia
- Hypotension
- Deformation of expandable stent
- Stent thrombosis
- Horner syndrome
- Cerebral hyperperfusion syndrome
- Carotid artery dissection
- Carotid artery rupture
- Hemorrhage resulting from anticoagulation

SUMMARY

As the mean age of people in the United States increases, treatment of vascular disease is one of the fastest changing areas of medicine. Minimally invasive vascular surgical techniques are being introduced that are revolutionizing the options available for treatment. Many highly invasive surgical techniques are now being performed as interventional radiologic procedures. Anesthetic management for vascular procedures is far different from just a few years ago and requires that we adapt to ever-new treatment strategies. As technology practice evolves, we will be better able to assess growing evidence that suggests the superiority of these procedures in decreasing patient morbidity, mortality, and convalescence.

REFERENCES

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