

the LV and a transapical approach. General anesthesia is needed for transapical procedures.⁴⁰⁵ Large-bore intravenous access, an arterial catheter, and central venous access are essential, and a PA catheter may be used in patients with preexisting pulmonary hypertension. External defibrillation pads are placed and connected to the defibrillator before the procedure begins (ventricular fibrillation can result from manipulation of catheters within the heart or from rapid ventricular pacing).⁴⁰⁵ Because a period of rapid ventricular pacing is required during deployment of certain of the valves, a transvenous lead for temporary pacing is inserted, typically through the femoral or subclavian vein (or, in the case of transapical procedures, sewn directly on the epicardial surface).⁴⁰⁵

When general anesthesia is used, a TEE probe is introduced after induction of anesthesia, so that structural anatomy can be confirmed after the TAVI procedure. A number of centers have adopted a minimalist approach to transfemoral TAVI in which sedation has replaced general anesthesia, and a fast-track protocol speeds the throughput of these patients. This has led it to be a “same-day” procedure in some sites with relatively good results, although these techniques are highly institutionally dependent and success is predicated upon the dynamics and experience of a well-trained team.^{408,409}

The advantages of this approach include neurologic status monitoring, greater hemodynamic stability, and possibly reduced postprocedure length of stay in the ICU compared with an approach using general anesthesia.⁴¹⁰ However, if TEE is a planned part of the procedure to evaluate valve placement, aortic integrity, and to rule out cardiac complications, general anesthesia is suggested.

TEE has a central role in evaluating annulus size, aortic disease, ventricular function, and MR, as well as determining reasons for hemodynamic instability.^{407,411} A balloon valvuloplasty of the aortic valve often precedes the insertion

of a bioprosthetic valve, which is crimped on a valvuloplasty balloon catheter and implanted by inflation. The device is guided into position through the combined use of real-time TEE and fluoroscopy. Immediately after inflation, TEE is employed to measure the degree and source of any AR⁴¹² and to examine the aorta for any aortic dissection.^{407,411}

A major challenge during the TAVI procedure is maintaining hemodynamic stability. In patients with severe concentric LV hypertrophy and intravascular volume depletion, hemodynamic status may deteriorate rapidly because of ventricular pacing, intracardiac guidewire or catheter manipulation, or balloon aortic valvuloplasty itself.⁴⁰⁵ Avoiding prolonged hypotension and the cycle of hypotension, subendocardial ischemia, and low output is critical to prevent hemodynamic collapse. The frequency and duration of rapid ventricular pacing episodes may need to be limited to allow enough time between episodes for spontaneous circulation to recover. Vasopressors (nor-epinephrine, epinephrine, or phenylephrine), administered as incremental boluses or as a continuous infusion, may be necessary. Constant communication among team members is essential throughout this multidisciplinary procedure.

The TAVI procedure requires state-of-the-art imaging capability, as well as the immediate availability of personnel to secure surgical access and institute CPB.⁴⁰³ The short-term efficacy of TAVI, as shown by echocardiographic measurements, is good.⁴⁰⁶ However, evidence on long-term outcomes awaits further data collection in national TAVI registries.^{405,413}

Mitral Valve Clipping. Percutaneous mitral valve repair by clipping is a catheter-based procedure, in which edge-to-edge repair of the mitral valve is performed by the MitraClip (Abbott Inc., North Chicago, IL) (Fig. 54.50).⁴¹⁴ The MitraClip is used to perform mitral valve repair in patients who are considered too high-risk to undergo conventional mitral valve repair surgery. The percutaneous procedure is performed on a beating heart. TEE plays a crucial role in

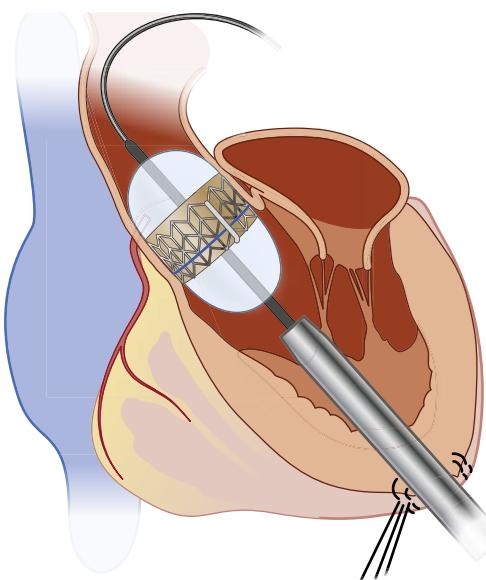


Fig. 54.49 Schematic of transapical aortic valve implantation. The prosthesis is implanted within the native annulus by balloon inflation. (From Walther T, Ralk V, Borger MA, et al. Minimally invasive transapical beating heart aortic valve implantation: proof of concept. *Eur J Cardiothorac Surg*. 2007;31:9–15.)



Fig. 54.50 Close-up view of the MitraClip (Abbott Inc., North Chicago, IL). (From Kothandan H, Vui KH, Khung KY, et al. Anesthesia management for MitraClip device implantation. *Ann Card Anaesth*. 2014;17[1]:17–22.)

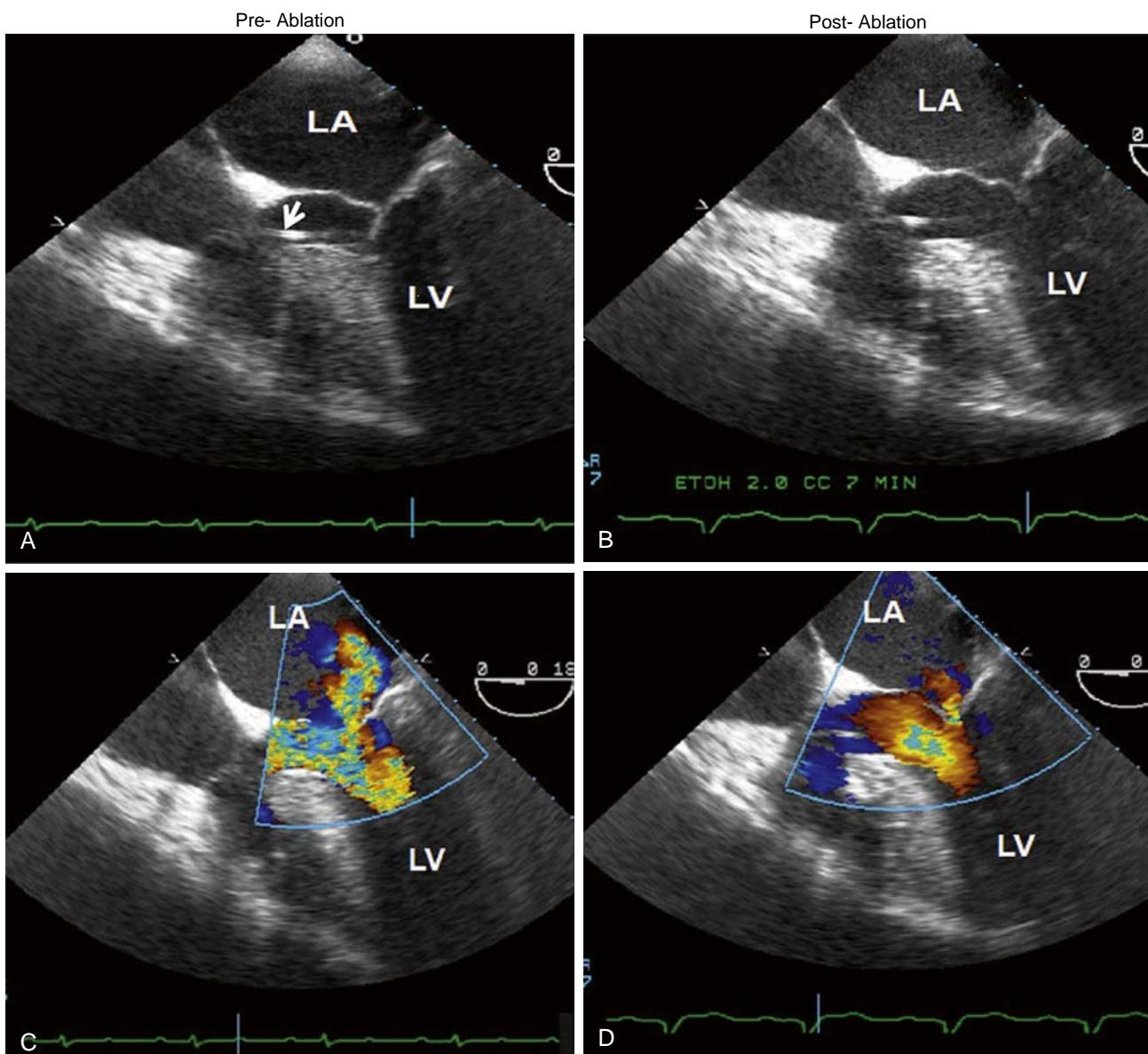


Fig. 54.51 Transthoracic echocardiography (TTE) shows images before and after MitraClip implantation in a patient with functional mitral regurgitation. 4-chamber views showing MR (A) before and (B) after MitraClip implantation. 2-chamber view showing MR before (C) and after (D) MitraClip implantation. LA, Left atrium; LV, left ventricle. (From Kothandan H, Vui KH, Khung KY, et al. Anesthesia management for MitraClip device implantation. *Ann Card Anaesth*. 2014;17[1]:17–22.)

directing the deployment of the device and placement of the clip(s). At the start of the procedure, 3D TEE is performed to provide pertinent annular and leaflet measurements. The device is advanced through the femoral vein and, after interatrial septal puncture, it is advanced into position under the guidance of TEE, taking care to avoid the atrial walls. A successfully performed MitraClip implantation produces a double orifice mitral valve (Fig. 54.51),⁴¹⁴ similar to an Alfieri repair, a mitral valve edge-to-edge stitch that is performed in the operating room with the use of CPB.⁴¹⁴

MitraClip implantation has been shown to significantly reduce mitral regurgitation, improve the NYHA functional class of the patient,⁴¹⁵ and to shorten hospital stay relative to open cardiac surgery (Fig. 54.52). Potential problems associated with the procedure include worsening of the

regurgitation, valvular stenosis, residual ASD, and rupture of the atrial wall with resultant tamponade.⁴¹⁴

A multidisciplinary team comprised of a cardiologist, cardiac surgeon, cardiac anesthesiologist, cardiac specialty nursing staff, and a radiology technician is necessary to ensure success of these procedures.

ANESTHETIC CONSIDERATIONS. Mitral valve clipping is performed under general anesthesia with endotracheal intubation. Standard ASA monitors and arterial line for blood pressure monitoring are used. Two optimally functioning peripheral intravenous lines are generally considered adequate.

After the interatrial septal puncture is made, anticoagulation is achieved by administering heparin to achieve an ACT of approximately 250 seconds. Maintenance of ACT at

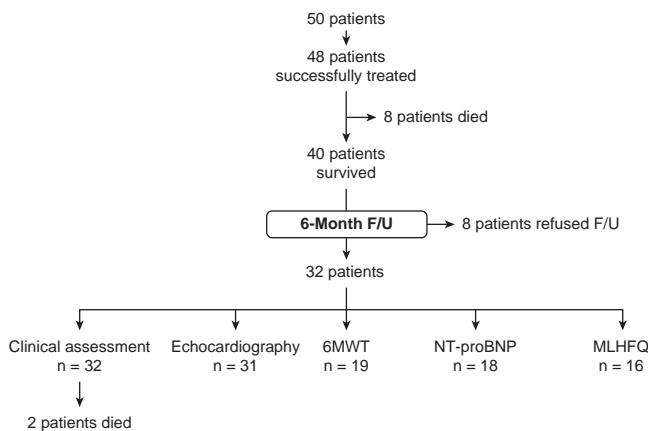


Fig. 54.52 Outcomes of MitraClip implantation at 6 months. Clinical follow-up was obtained from 32 and echocardiographic follow-up from 31 successfully treated patients at a median of 6.1 months (range, 4.2-10.4 months). Eight patients had died by the time of their scheduled follow-up visit, and eight patients did not return for a follow-up visit—one patient because he had received an LV assist device 3 months after MitraClip therapy and the others because they lived too far away from their respective treatment centers. Flow of patients over the 6-month period. F/U, follow-up; MLHFQ, Minnesota Living with Heart Failure Questionnaire; 6MWT, 6-minute walk test. (Redrawn from Franzen O, van der Heyden J, Baldus S, et al. MitraClip therapy in patients with end-stage systolic heart failure. *Eur J Heart Fail*. 2011;13:569-576.)

an adequate level is ensured by checking the values every 30 minutes.⁴¹⁴

During the procedure, ventilation holding may be required at certain critical points. Placement of a second clip may require special attention on the part of the cardiac anesthesiologist. During the advancement of the second clip from atrium to the ventricle there is a potential for damage to the first clip. Some patients may experience transient hypotension during this process, and the use of vasopressors might be needed to support the blood pressure. Blood pressure should be brought to patient's non-anesthetized state at the end of each grasp and before the release of the device to evaluate any residual regurgitation. This will eventually help determine if the clip can be deployed or an additional clip will be needed.⁴¹⁴

At the conclusion, protamine is used to reverse anticoagulation and patients are typically extubated at the end of the procedure, unless there is a medical need to keep the endotracheal tube in place. All patients typically spend the first night after the procedure in an ICU, although trends toward fast-tracking and bypassing the ICU have permeated the mitral clipping procedures as well.

Left Atrial Appendage Occlusion Device Implantation.

AF remains the most common arrhythmia. AF increases the risk of stroke by fivefold due to appendage thrombus embolization, and consequently carries significant morbidity and mortality.⁴¹⁶ Drugs commonly used for the prevention of thrombus formation include warfarin, factor Xa inhibitors, and direct thrombin inhibitors, which reduce the risk of stroke by 60%. Patients for whom anticoagulant medication is contraindicated would be candidates for LAA occlusion techniques, which are increasingly available.

The PROTECT AF clinical trial studied the Watchman LAA system (Boston Scientific Corporation, Natick, MA)

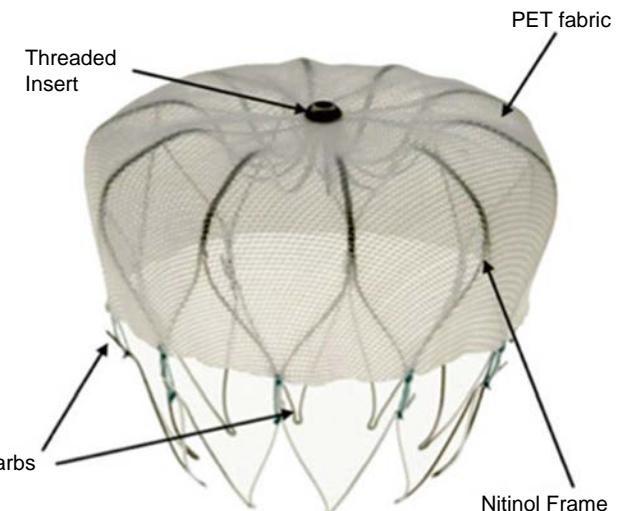


Fig. 54.53 Watchman device (Boston Scientific Corporation, Natick, MA), a self-expanding nitinol frame structure with fixation barbs and a permeable polyester fabric that covers the atrial facing surface of the device. (From Ding J, Zhu J, Lu J, et al. Transcatheter closure of the LAA: initial experience with the Watchman device. *Int J Clin Exp Med*. 2015;8(9):15230-15237.)

under the hypothesis that AF patients can be protected from stroke with the Watchman as effectively as with systemic anticoagulation.⁴¹⁶

Fig. 54.53⁴¹⁶ shows the Watchman device (Boston Scientific Corporation). Designed like a parachute, the Watchman device is deployed percutaneously from the femoral vein approach, and using TEE and fluoroscopic guidance, is directed across the interatrial septum and into the LAA. TEE is used extensively to evaluate the final deployment of the device and to confirm that there is minimal peri-device blood leakage.

ROLE OF TRANSESOPHAGEAL ECHOCARDIOGRAPHY IN THE WATCHMAN PROCEDURE. Preprocedure TEE should be performed to rule out presence of a thrombus in the LAA, as it is a contraindication to the procedure. The presence of spontaneous contrast, on the other hand, is not considered a contraindication. TEE is further used to ascertain the shape of the LAA, the number and location of lobes of the LAA, and compared with the cardiac CT exam. In addition, the width of the orifice is measured using multiple viewing angles to determine the ideal landing zone, 10 mm distal to the ostium of the appendage. It is recommended that the device be sized 3-5 mm larger than the largest diameter.⁴¹⁷

The device is advanced through a transseptal puncture. In order to align the device with the axis of the LAA the septal puncture is made in the inferior and posterior part of the fossa ovalis. Care must be taken to guide the device away from the aortic valve. The midesophageal aortic valve short-axis view and the midesophageal bicaval view can be used to ensure that the device is positioned near the IVC and is being advanced away from the aortic valve. After testing the device with a tug test and using color Doppler to rule out any significant perioccluder blood leakage, the device is finally released from the delivery cable.

Postimplantation it is important to perform a thorough TEE evaluation to rule out complications of implantation.

These complications may include left upper pulmonary vein compression, mitral valve impingement, and circumflex coronary artery compression.⁴¹⁷

ANESTHETIC MANAGEMENT. LAA occluders are deployed under general anesthesia with endotracheal intubation. In addition to the standard ASA monitors, two large-bore peripheral intravenous lines are required for these procedures. At the time of septal puncture, heparin is administered to achieve a target ACT of greater than 250 seconds. Depending on the ACT toward the end of the procedure, heparin may be reversed with protamine. Generally, Watchman procedures are short in duration, in the range of 30 to 45 minutes. Patients are extubated on the table and are kept in the hospital overnight for observation after the procedure.

HEART FAILURE

HF can be defined as a complex clinical syndrome caused by any structural or functional cardiac disorder that impairs the ability of the heart (as a pump) to meet the metabolic demands of the body. Thus HF can result from an impairment of diastolic filling, systolic ejection, or both. Once HF is present, progressive cycles of deterioration and transient compensation ensue that may continue for years. Essentially, increases in end-diastolic volumes are compensated for by endogenously promoted diuresis, which is compensated for by sympathetic activation. This activation promotes further diuresis, which then requires compensation by further sympathetic activation, and so on. As the syndrome progresses, the hemodynamic changes, the cycles of fluid retention and relative hypovolemia, and bodily hypoperfusion perturb many neuroendocrine, humoral, and inflammatory feedback loops (Box 54.13), with resulting progressive and inexorable cycles of physical and functional deterioration of the heart and major bodily organs. In the United States, more than 6 million people currently have HF, and its prevalence is estimated at 10% in people older than 65 years. Although survival with HF has improved, the mortality associated with HF remains high, and at least 50% of patients with HF are expected to die within 5 years of diagnosis.

The ACC/AHA guidelines for the evaluation and management of chronic HF place patients in four classes based on the stages of the syndrome (Box 54.14).⁴¹⁸ Early in the course of the disease, ventricular contractility is maintained by adrenergic stimulation and activation of the renin-angiotensin-aldosterone and other neurohormonal and cytokine systems.^{419,420} Patients in this stage would be considered to be in ACC/AHA class B. However, these compensatory mechanisms become less effective over time, and ventricular dilatation and fibrosis occur, progressively worsening cardiac function. This produces a chronic state of low perfusion and, ultimately, refractory end-stage HF, labeled class D in the ACC/AHA classification scheme. The New York Heart Association (NYHA) functional classification system is also used to assess the severity of functional limitations and correlates fairly well with prognosis (Box 54.15). Some patients may remain asymptomatic for years despite ventricular remodeling, dilatation, and decreased EF.

BOX 54.13 Pathophysiology of Heart Failure: From Injury to Clinical Syndrome

1. Causes
 - a. Myocardial injury
 - i. Ischemia
 - ii. Toxins
 - iii. Volume overload
 - iv. Pressure overload
 - b. Genetic perturbation
2. Cardiac remodeling
 - a. Myocyte growth
 - i. Concentric hypertrophy
 - ii. Eccentric hypertrophy
 - b. Interstitial fibrosis
 - c. Apoptosis
 - d. Sarcomere slippage
 - e. Chamber enlargement
3. Clinical heart failure milieu
 - a. Pump performance
 - b. Circulatory dynamics
 - c. Metabolic abnormalities

BOX 54.14 American College of Cardiology/American Heart Association Classification of Chronic Heart Failure Stages

- A: High risk for heart failure
Hypertension, diabetes mellitus, coronary artery disease, family history of cardiomyopathy
- B: Asymptomatic heart failure
Previous myocardial infarction, left ventricular dysfunction, valvular heart disease
- C: Symptomatic heart failure
Structural heart disease, dyspnea, fatigue, impaired exercise tolerance
- D: Refractory end-stage heart failure
Marked symptoms at rest despite maximal medical therapy

From Hunt SA, Abraham WT, Chin MH, et al. 2009 Focused update incorporated into the ACC/AHA 2005 guidelines for the diagnosis and management of heart failure in adults: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines developed in collaboration with the International Society for Heart and Lung Transplantation. *J Am Coll Cardiol*. 2009;53:e1–e90.

BOX 54.15 New York Heart Association Heart Failure Symptom Classification System

NYHA Class Level of Impairment

- I: Ordinary physical activity not limited by symptoms
- II: Ordinary physical activity somewhat limited by dyspnea
- III: Exercise limited by dyspnea during a mild workload
- IV: Dyspnea at rest or with very little exertion

NYHA, New York Heart Association.

Medical Management of Heart Failure

Medical strategies for treating developing HF aim to limit disease progression, prolong life, and improve quality of life as the syndrome progresses. Medical therapies, such as ACEIs, β -blockers, diuretics, inotropic agents, and antiarrhythmics, represent the usual standard of care for HF patients. In general, β -blockers, ACEIs, and ARBs (for those who cannot tolerate ACEIs) are class I indications in the current ACC/AHA guidelines for class A and B patients with HF who have cardiac structural abnormalities and who have not yet developed symptoms of HF.⁴¹⁸ Signs and symptoms of HF (class C) are class I indications for the use of certain β -blockers that have been shown to prolong life (e.g., bisoprolol, carvedilol, and sustained release metoprolol), ACEIs, and ARBs. Diuretics and salt restriction should be added for patients with fluid retention. The implantation of devices for cardiac resynchronization therapy and defibrillatory capability is recommended, and revascularization and valve repair or replacement should be performed as appropriate. In both symptomatic and asymptomatic patients, current ACC/AHA guidelines list several additional potential therapies with class II and III indications.

However, even multidrug regimens may not prevent progression toward class D HF. A patient who has reached this category has a 2-year mortality risk greater than 75%. Thus, surgical intervention at less advanced stages of HF has become common in an attempt to prevent the inexorable progression of the syndrome.

Surgical Management of Heart Failure

In prior decades, the only established surgical treatment option for advanced HF was cardiac transplantation, which is still associated with excellent survival rates and functional capacity. It is now currently accepted that certain surgical interventions (in conjunction with pharmacological) have the capacity to retard or even potentially reverse the pathophysiology of cardiac failure. Though there are no large multicenter trials showing an independent effect on enhanced survival, specific surgical interventions (e.g., coronary revascularization, mitral valve repair/replacement) are now routinely performed for patients with advanced stages of HF to improve quality of life.

CAD is still the most common cause of HF, as it has been reported to be for many years. Where viable myocardium and feasible targets exist, revascularization of the failing heart can improve cardiac function and NYHA functional class.^{421,422} It can also slow remodeling and decrease the incidence of arrhythmias,⁴²³ and it has been demonstrated to improve survival. For example, Liao and associates found that among patients with CHF, survival was improved in patients who underwent revascularization compared with those who did not.⁴²¹ The optimal method of revascularization has not been conclusively determined, however, and studies continue to examine the issue of whether percutaneous coronary intervention is as effective as the gold standard intervention, CABG, in promoting long-term survival. The issue may be further complex in that certain subpopulations of patients benefit differently (e.g., those with diabetes), that surgical technique is not uniform, and that not all stents are equivalent. A meta-analysis demonstrated that in transplanted patients with HF from coronary allograft

vasculopathy, patients who underwent percutaneous intervention instead of CABG had both a lower early mortality (PCI 4.3% vs. CABG 36.4%; $P < .001$) and a convincing trend regarding overall mortality (PCI 21.4% vs. CABG 42.3%; $P = .049$).⁴²⁴

Carefully selected patients with advanced CHF and MR receive several benefits from mitral valve repair or replacement. Among them are a progressive decrease in LVEDV, reverse remodeling, a progressive improvement in LVEF, improved functional status or NYHA class, improved 6-minute walk-test performance, improved peak oxygen consumption, and lower long-term mortality risk.^{425,426} In fact, the 2017 focused update to the 2014 ACC/AHA Guidelines for the Management of Valvular Heart Disease provide several recommendations regarding surgical interventions for valvular dysfunction in the setting of HF.⁴²⁷ Notably, a reminder is put forth in the guidelines that primary and secondary MR are different diseases. Primary (structural) mitral regurgitation is the subject of both curative and preventative recommendations. From a curative perspective, it is a class I recommendation that primary severe MR should be addressed surgically in the setting of LV dysfunction (LVEF 30%-60%) and/or LV end-systolic dimension (LVESD) ≥ 40 mm, to allow for reverse remodeling and to prevent further progression of disease. From a purely preventative perspective, it is a class IIa recommendation that severe asymptomatic primary MR be addressed surgically when the preserved LVEF progressively falls below 60% and/or the LVESD increases above 40 mm on serial exams. However, once LV dysfunction has progressed, it is only a class IIb recommendation that repair of symptomatic severe MR “may be considered” when the EF is less than 30%. The importance of appropriate patient selection is paramount, however, if optimal benefits are to be realized; the best results occur when mitral valve repair or replacement is undertaken before the geometric derangements to the LV and functional derangements of the valve reach certain levels. Several key predictors of lack of reverse remodeling have been identified: LV end-diastolic dimension greater than 6.5 cm, LVESD greater than 5.1 cm, large LA volume, high LV sphericity index, and severely depressed LVEF.^{321,426-430} The optimal type of repair remains a subject of ongoing study, as does the issue of whether mitral repair or replacement affords the greater survival benefit to these often older and highly comorbid patients. Clearly, the quality of any repair greatly influences its outcome.

With respect to secondary, or functional, MR (most often secondary to ischemic disease), the 2017 ACC/AHA recommendations provide a class IIa recommendation for choosing chordal-sparing mitral valve replacement over “repair” for chronic severe MR in NYHA class III or IV patients. A 2016 trial of 251 patients with severe ischemic MR randomized to repair or replacement demonstrated no difference in reverse remodeling or mortality at 2 years, but an increased recurrence of moderate-severe MR with repair (vs. replacement) at 2 years, as well as an increased incidence of recurrent HF and rehospitalization for HF in the repair group.⁴³¹

The effectiveness and potential survival benefits of combining revascularization with other surgical procedures (e.g., valve repair or replacement, ventricular reshaping) have been and continue to be the subject of large,

multicenter investigations. Surgical ventricular restoration (reshaping), when performed in conjunction with revascularization, has thus far not been shown to improve survival, decrease symptoms, or increase exercise tolerance in patients with HF.⁴³²

In addition to common surgical interventions, electrophysiologic interventions (see chapter XX) play a key role in the initial modern management of HF. Large, international, multicenter trials in symptomatic and asymptomatic HF patients treated with cardiac resynchronization therapy, and/or ICD, have demonstrated improved survival, decreased risk of hospitalization, decreased incidence of sudden cardiac death, improvements in LVEF, decreased LV volumes, and decreased symptomatology.⁴³³⁻⁴³⁷

Today, when pharmacologic, electrophysiologic, and surgical interventions fail to stave off the inevitable progression of HF, mechanical circulatory support (MCS) with VADs is commonly implemented. Short-term MCS is generally used as a “bridge to immediate survival,” a “bridge to recovery,” a “bridge to next decision,” and/or a “bridge to a bridge” after an acute cardiac event. Patients with advanced HF who have little expectation of ventricular recovery (or who have not recovered despite short-term support) now regularly receive intermediate- and long-term VAD support as a potential “bridge to transplantation” and a “bridge to candidacy.” It is known that time spent on MCS can improve multisystem organ function to the point where previously transplant-ineligible patients can become transplant eligible. Since 2002, transplant-ineligible patients may receive approved LVADs as “destination therapy,” a final treatment strategy shown to confer a survival advantage compared with medical management alone. Heart transplantation remains, of course, the ultimate surgical intervention for end-stage HF, but the number of patients with HF far exceeds the number of donor organs available each year worldwide, thus leaving MCS with an LVAD as the best option for most patients with advanced and end-stage HF.

The use of MCS with LVADs as a bridge to cardiac transplantation was previously shown to improve the survival rates and outcomes of patients with decompensated HF and was also found to improve multiorgan function during the time spent on VAD support.⁴³⁸⁻⁴⁴³ This has been the rationale for the tremendous increase in the use of bridging technology.

However, analyses from the International Society for Heart and Lung Transplantation indicate that among patients who received cardiac transplants between July 2004 and June 2009, bridging to transplantation with pulsatile or nonpulsatile VADs did not actually confer a statistically significant survival advantage.⁴⁴⁴ Those bridged patients who received a transplant between January 2002 and June 2009 actually had a worse 6-month posttransplant survival but an equivalent 7-year survival, to patients who were not bridged. However, the preponderance of deaths in the early years of this technology and the statistical methods used to perform the analyses may have great bearing on meta-analyses conducted in the modern era. Nevertheless a subsequent publication found that bridging to transplantation with an LVAD was associated with increased transfusion requirements for blood and blood products perioperatively, a higher incidence of clinically

significant cell-mediated rejection within the first posttransplant year, and a trend toward increased postoperative mortality.⁴⁴⁵ Further analyses are currently under way because overall, there does seem to be a survival advantage in more recent years with modern devices and our more extensive collective experience with patient management. Thus, the indication for bridging (e.g., elective vs. “required” because of severe decompensation) may be a vitally important part of the selection criteria going forward. In this regard, the Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS) profile plays an important role in appropriate patient selection and the timing of intervention.⁴⁴⁶ The INTERMACS profile characterizes patients with HF on the basis of clinical status and symptoms.⁴⁴⁷

- INTERMACS 1: critical cardiogenic shock
- INTERMACS 2: deteriorating on inotropes
- INTERMACS 3: stable, but inotrope dependent
- INTERMACS 4: symptomatic at rest
- INTERMACS 5: exertion intolerant
- INTERMACS 6: limited with exertion
- INTERMACS 7: advanced NYHA class III

With regard to destination therapy in the United States, the most recent statistics from the INTERMACS registry indicate that overall survival has dramatically increased since the introduction of continuous flow devices: 1-year survival is now approximately 80%, 2-year survival is approximately 70%, 3-year survival is approximately 60%, and 4-year survival is approximately 50%.⁴⁴⁸ These numbers are extremely noteworthy considering the approximate 25% rate 1-year survival originally shown in the Randomized Evaluation of Mechanical Assistance for the Treatment of Congestive Heart Failure (REMATCH) trial, which led to FDA approval of the HeartMate VE for destination therapy in September 2002.

The first generation of pulsatile VADs used as bridges to recovery and bridges to transplantation in the 1990s and early 2000s have now been supplanted worldwide by second- and third-generation, nonpulsatile devices, which generate continuous flow and have the advantages of being small, silent, valveless, and significantly more durable than the first generation of devices.

Though the current generation of devices is associated with a significant reduction in specific perioperative and long-term complications that were common with the first-generation pulsatile support devices, it seems that the total burden of “adverse events” has not apparently decreased because new complications have emerged that did not exist with the first generation of devices. Quality of life is arguably more important than quantity of life for many, and it is important to weigh the demonstrated clinical benefits of VADs against the risks of infection, pump malfunction (nowadays, most often due to pump thrombosis or electrical driveline issues), neurologic events, and bleeding complications. That said, the survival of end-stage HF is clearly better with MCS than without.

At the time of this writing, the most commonly used long-term LVAD in the United States is the HeartMate II (HM II; Abbott). The HM II is a small axial flow pump that continuously draws blood from the LV apex by the action of a rapidly rotating impeller and returns the blood to the ascending aorta in a nonpulsatile fashion. It has been approved in the

United States as a bridge to transplantation since 2008 and as destination therapy since 2010. Rates of specific common complications are dramatically lower with the HM II than with the previous generation of pulsatile devices, and the durability of the device far exceeds that of the Heart-Mate I (>10 years, compared with only approximately 18 months). The current rate of successful bridging to transplantation with the HM II is approximately 87%. Chronic anticoagulation to an INR of 2.5 to 3.5 is recommended for all currently available implantable LVADs, but one advantage of the HM II over other current devices appears to be the duration one can be without anticoagulation, which may be helpful if patients tend toward non-compliance.

The Heartware HVAD (Medtronic) is a miniaturized centrifugal flow pump with a magnetically levitated impeller. It was approved as a “bridge to transplantation” in 2012 and for “destination therapy” in 2017. Its small size and intrapericardial site of implantation are advantageous to allow for its use in patients with smaller body surface areas. Successful rates of bridging to transplantation and overall survival with the HVAD are similar to the HM II, but the HVAD appears to be associated with a slightly higher risk of stroke than the HM II.^{449,450} It may be that HVAD would be a less desirable choice in uncontrolled hypertensive patients, as MAPs greater than 90 have been associated with an increased risk for stroke during LVAD support.⁴⁴⁹

The HeartMate 3 (Abbott) is a small intrapericardially positioned continuous flow centrifugal pump with a bearingless, magnetically levitated, magnetically driven impeller. With this centrifugal pump, rotational speeds of 3000 to 5000 rpm can produce upwards of 10 LPM of flow. According to the manufacturer, and as described in the first reported implant in a human,⁴⁵¹ the hemocompatibility of the device was improved through coating of all blood surfaces with the classic HeartMate sintered-titanium microsphere lining. In addition, the risk of thrombosis was reduced by creating three separate flow paths through the device to continuously wash all internal device elements. As well, magnetic levitation of the rotor obviates the need for a bearing, which not only reduces wear-and-tear, but decreases the amount of heat produced during impeller rotation. As has been shown to be advantageous with other “maglev” devices, less heat means less hemolysis, with less resultant plugging of the microvasculature from free hemoglobin in the plasma. In addition, small size and intrapericardial site of implantation allow its use in patients with smaller body surface areas, as with the HVAD.

As overall survival has improved over the past decade, analyses now focus on other details of importance, and the issue of “centrifugal versus axial” design has become an important one. The HM3 was compared to the HM II in the MOMENTUM 3 trial (Multicenter Study of MagLev Technology in Patients Undergoing Mechanical Circulatory Support Therapy with HeartMate 3).⁴⁵² The first analysis of this key multicenter trial of nearly 300 patients demonstrated a significantly higher rate of a composite endpoint of survival free from either disabling stroke or reoperation for device malfunction at 6 months with the HM3 compared to the HMII (86.2% vs. 76.8%; $P < .001$ for non-inferiority; $P = .04$ for superiority). There were **NO** suspected or confirmed pump thromboses in HM3 patients at 6 months compared

to 10.1% of patients implanted with the HMII. Importantly, there were also no significant differences detected between the two groups in the rates of stroke; bleeding; sepsis; RV failure; hepatic, renal, or pulmonary failure; hospital length of stay; rate of discharge from the hospital; arrhythmias; hemolysis; or drive-line infections. Thus, the overall conclusion was that implantation with the HM3 resulted in better outcomes, mainly as a result of fewer reoperations for pump “malfunction.” Two-year follow-up data from MOMENTUM 3 was published in 2018,⁴⁵³ and again, the HM3 was found to be not only “noninferior” to the HM II, but frankly “superior.” Regardless of the indication for implantation (bridge to transplantation vs. destination therapy), at 2 years following implantation, 79.5% of those implanted with the HM3 demonstrated survival free from either disabling stroke or reoperation for device malfunction compared to 60.2% of those implanted with the HM II ($P < .001$ for noninferiority and $P < .001$ for superiority). Driving the finding was again freedom from reoperation for pump malfunction (1.6% in the centrifugal-flow pump group vs. 17.0% in the axial-flow pump group $P < .001$). The rates of death and disabling stroke were similar in the two groups, but the overall rate of stroke was lower in the centrifugal-flow pump group than in the axial-flow pump group (10.1% vs. 19.2%; $P = .02$). The HM 3 received FDA approval for “short-term indications” (e.g., “bridge to transplantation”) in 2017, and approval for “long-term” indications (e.g., “destination therapy”) is anticipated but still pending at the time of this writing.

The CardioWest total artificial heart (TAH-t, SynCardia Systems, Inc, Tucson, AZ) is FDA approved (and approved and available in Europe and Canada) as a bridge to transplantation for patients requiring biventricular long-term support. Two sizes are available: 70 mL ventricles (for patients with a body surface area $>1.7 \text{ m}^2$ and an intrathoracic anteroposterior diameter $>10 \text{ cm}$ at the level of the 10th vertebral level), and 50 mL ventricles (for patients with body surface area $<1.7 \text{ m}^2$). Indications for the TAH include: irreversible biventricular failure, allograft failure (rejection or heart transplant vasculopathy), failure to wean from ECMO, massive MI that affects technical insertion of a VAD, recurrent ventricular tachycardia/fibrillation, intracardiac thrombus, tumor, restrictive cardiomyopathy, post infarction VSD, type A aortic dissection with coronary artery dissection, or end-stage congenital heart disease (CHD).⁴⁵⁴⁻⁴⁵⁶ Survival rates at 3 and 6 months after TAH implantation were reported in 2016 as 76% and 65%, respectively, and 45% of TAH patients from 2005 to 2015 were transplanted by 6 months.⁴⁵⁷ Recently, the TAH-t has seen renewed popularity, mainly because it is the only implantable intermediate- to long-term support solution for patients with biventricular failure that is currently available. In previous years, the TAH-t was reported to enjoy a 79% successful rate of bridging patients to transplantation, but no recent data is published in this regard. According to the manufacturer, more than 1100 TAH-t devices have been implanted.

CARDIOMYOPATHIES

Current understanding(s) and classification(s) of “cardiomyopathies” have changed dramatically since the 1980

WHO/ISFC task force definition as “heart muscle diseases of *unknown cause*” and a classification that simply included:

- Dilated cardiomyopathy
- Hypertrophic cardiomyopathy
- Restrictive cardiomyopathy

That same task force document⁴⁵⁸ defined heart muscle diseases of *known cause* or associated with disorders of other systems as “specific heart muscle diseases” that included a wide variety of infective diseases, metabolic disorders, generalized systemic diseases (e.g., infiltrative and connective tissue disorders), heredofamilial diseases (e.g., muscular dystrophies), sensitivities, and toxic reactions.

By 2006, significant advances in molecular genetics afforded entirely new understandings of the etiologies and pathogenesis of “heart muscle diseases” such that an entirely new definition and classification of cardiomyopathies was proposed. According to the American Heart Association’s 2006 Scientific Statement⁴⁵⁹:

“Cardiomyopathies are a heterogeneous group of diseases of the myocardium associated with mechanical and/or electrical dysfunction that usually (but not invariably) exhibit inappropriate ventricular hypertrophy or dilatation and are due to a variety of causes that frequently are genetic. Cardiomyopathies either are confined to the heart or are part of generalized systemic disorders, often leading to cardiovascular death or progressive HF-related disability.”

“Cardiomyopathies...are not... a direct consequence of other cardiovascular abnormalities such as which occurs with valvular disease, systemic hypertension, CHD, and atherosclerotic coronary artery disease producing ischemic myocardial damage secondary to impairment in coronary flow.”

Thus the current conception of cardiomyopathies classifies them as either primary (a disease confined to the heart) or secondary (cardiac pathology/pathophysiology results as a manifestation of a systemic condition). Primary cardiomyopathies are further subcategorized into genetic, acquired, or mixed in their etiology. Secondary causes of cardiomyopathy are classified by the pathogenesis, and include a wide variety of infectious, endocrine, infiltrative, autoimmune/inflammatory, nutritional, toxic, and neuromuscular diseases. Box 54.16⁴⁶⁰ lists examples of the primary cardiomyopathies. In the current conception, acquired (non-genetic) cardiomyopathies are distinguished from secondary cardiomyopathies (e.g., the myocardial dysfunction that results from ischemic coronary disease is no longer considered a “cardiomyopathy”).

Distinct from the classification of cardiomyopathies for medical purposes, and regardless of whether the cardiac dysfunction is now called primary or secondary, acquired or inherited, the surgical management of “cardiomyopathy” derives directly from the resultant cardiac pathophysiology. Some cardiomyopathies are amenable to surgical interventions, and some are “managed” with electrophysiological procedural interventions, with or without pharmacological therapies; some require all of these.

Given the acute and potentially severe cardiac mechanical dysfunction associated with several of the primary cardiomyopathies (e.g., peripartum, Takotsubo, myocarditis), urgent

BOX 54.16 Primary Cardiomyopathies

Genetic

- Hypertrophic
- Ion channelopathies
- Arrhythmogenic RV dysplasia
- LV noncompaction
- Mitochondrial myopathies

Acquired

- Peripartum
- Stress (Takotsubo)
- Myocarditis
- Tachymyopathy

Mixed

- Restrictive
- Dilated

LV, Left ventricular; RV, right ventricle.

Adapted from Brierle J, Breeden MA, Tucker J. Cardiomyopathy: an overview. *Am Fam Phys*. 2017;96(10):640–647.

MCS or ECMO may be required as a “bridge to immediate survival,” a “bridge to next decision,” a “bridge to recovery,” or a “bridge to a bridge.” The majority of the secondary cardiomyopathies and some of the primary ones (e.g., LV noncompaction and dilated) are progressive in nature from the time of their onset. Aside from acute decompensation, they are managed on a long-term basis with either eventual planned cardiac transplantation, or intermediate- and long-term MCS as a “bridge to transplantation,” a “bridge to candidacy” (for transplantation), and in some cases, “destination therapy.”

Many of the cardiomyopathies associated primarily with electrical dysfunction (but otherwise histologically normal myocardium) are managed with electrophysiologic procedural interventions (e.g., implantable defibrillators [ICDs], catheter-based percutaneous ablations, cardiac resynchronization therapy with biventricular pacing, etc.).

Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is of autosomal dominant genetic inheritance, and is reported to be the most common primary cardiomyopathy, with a prevalence of 1:500 people.⁴⁶¹ HCM is eminently amenable to both surgical and procedural interventions. Though there are a few described variants, in the most common form, asymmetric interventricular septal hypertrophy can result in ventricular arrhythmias, and (often) SAM of the mitral valve, in which systolic LVOT obstruction can occur; the entity is then called hypertrophic obstructive cardiomyopathy, or HOCM. Both arrhythmias and SAM present the risk of sudden death. Thus ICDs and surgical interventions (e.g., septal myectomy, mitral valve repair, release of papillary muscles, etc.) can mitigate both the risk of sudden death and symptomatology. In patients deemed too high-risk for surgery, percutaneous septal alcohol injection can be used to ablate the hypertrophied septal tissue.

Patients with HCM/HOCM should be evaluated regarding their risk of sudden death and the implantation of an ICD.⁴⁶² In addition to the potentially protective effects of an ICD, surgical septal myectomy is the treatment of choice for HOCM in patients with interventricular septal thickness

greater than 15 mm and/or SAM-prone MV anatomy, but the resection of septal tissue alone is not always sufficient to ameliorate the LVOT obstruction. Adjunctive procedures often need to be performed on the mitral valve and subvalvular apparatus to effect a complete repair. The majority of the patients with LVOT obstruction and associated anomalies of the MV apparatus can be corrected with MV repair without the need for MV replacement.

Potential surgical procedures include:

1. The Morrow myectomy: a classic subaortic septal resection, which is not as commonly performed. The subaortic area is easily accessible for resection but is not always the area causing the SAM.⁴⁶³
2. Extended myectomy: more commonly performed in the modern era; the hypertrophied septal myocardium is resected to a point beyond that of mitral-septal contact, usually to the base of the anterolateral papillary muscle, as such resection redirects flow medially and anteriorly away from the mitral leaflets to ameliorate SAM.⁴⁶⁴
3. Papillary muscle release: dividing connections between the papillary muscles and the LV free wall. With release, the mitral apparatus drops posteriorly into the LV, better separating the inflow and outflow portions of the LV, to ameliorate SAM.^{465,466}
4. AML plication: to shorten the redundant anterior leaflet and reduce leaflet slack that predisposes to SAM.⁴⁶⁷

Transthoracic echocardiography (TTE) is generally used at the time of initial presentation to verify the location and extent of the disease process and suitability for surgical repair, but TEE is generally used in the operating room. Prior to surgery, TEE is used to assess the location and degree of septal thickness, the presence of SAM-prone anatomy, coexisting mitral regurgitation of primary etiology, associated abnormalities of the mitral apparatus, and quantitation of the gradient across the LVOT. For the best possible outcome, it is important for the echocardiographer to review and discuss ALL TEE findings with the surgeon before instituting CPB, before separating from CPB, and again after final separation from CPB.

Following surgical intervention, it is especially critical for the echocardiographer to understand the specific surgical procedure(s) performed, as this will allow for a more complete assessment of the effectiveness of the intervention. The post-intervention TEE assessment should include:

- the effectiveness of the intervention, sometimes by employing provocative maneuvers to determine if continued obstruction can be elicited,
- the mitral valve, the chordae tendineae, and the papillary muscles (which may also have been thinned, plicated, resected, and/or released as part of the repair) and,
- evaluation for potential surgical complications of the myectomy (e.g., VSD, lacerated septal perforator(s)).

Postoperatively the magnitude of the gradient across the LVOT is critically dependent on optimization of preload, afterload, heart rate, and contractility, and whether or not there is still SAM. Initial postbypass gradients often disappear once the patient is optimized and there is no SAM. Factors known to promote SAM include: hypovolemia, vasodilatation, a high contractile state, and tachycardia.

One should not attempt to interpret residual gradients until the determinants of hemodynamics more closely mimic the awake state and the patient is rendered normovolemic postoperatively. Residual LVOT gradients between 10 and 30 mm Hg without SAM may be considered acceptable but should prompt consideration of a provocative maneuver to see if it increases. Residual LVOT gradients between 30 and 50 mm Hg and no SAM suggest that the myectomy may be inadequate. Gradients of this magnitude with SAM suggest that hemodynamic optimization may not yet have been realized, and/or that additional repairs of the mitral valve and/or subvalvular apparatus might be needed. A residual LVOT gradient greater than 50 mm Hg due to SAM suggests that further repair of the mitral valve (leaflets, annulus, and/or subvalvular apparatus) may be necessary. If the surgeon has already performed the valve “repair” in addition to the myectomy, this should likely prompt a discussion of possible mitral valve replacement or Alfieri stitch to control the SAM.

Provocative maneuvers that can be used to try to elicit LVOT residual obstruction are not always necessary, but include Valsalva maneuver, reduction in preload (e.g., reverse Trendelenburg position, bolus of NTG, etc.), pharmacological vasodilatation, and/or increase in inotropic state.

An iatrogenic VSD (with flow from left to right ventricles) should be excluded following myectomy, as such will require immediate repair. It is also important, however, to distinguish a VSD from a severed septal branch of the coronary artery anatomy (a coronary-cameral fistula). Such fistulae will demonstrate diastolic flow at the site of the myectomy into the LV. Large severed perforator branches may require ligation or occlusion lest the intracardiac shunt contribute to volume overload and potential cardiac failure.

Historically, such fistulae have generally been reported to be “rare,” but the identification of such fistulae is a common echocardiographic finding of which studies report a prevalence in the range of 19% to 23% following myectomy.^{468,469} The clinical significance of these fistulae depends on their size (most are trivial), and the natural history is that most spontaneously close within a few weeks. Though one study of 40 myectomy patients in the modern era found that nearly onequarter of such fistulae were still detectable by echocardiography at 6 months postoperatively.⁴⁶⁹

Cardiac Transplantation

Discovery of the immunosuppressive agent cyclosporine in the early 1980s made cardiac transplantation an accepted surgical option for end-stage HF.⁴⁷⁰ Currently, 1-year survival is in the range of 80% to 90%, and 5-year survival is approximately 70%.⁴⁴⁴ Patients 30 to 59 years old tend to have the best survival, as do recipients who required transplantation because of nonischemic cardiomyopathy. Patients who required transplantation due to CHD and those receiving a retransplant have a reported 1-year survival of approximately 68%.⁴⁷¹

Most candidates for cardiac transplantation have class D HF that has been maximally treated medically but is nevertheless likely to result in death in less than 1 year. Such patients typically are in cardiogenic shock or have a chronic low-output state that necessitates mechanical or inotropic

support, but candidates also include patients with advanced symptomatic HF and peak oxygen uptake less than 10 mL/kg/min (with the achievement of an anaerobic threshold), patients with NYHA class IV HF caused by advanced hypertrophic or restrictive cardiomyopathy, patients with refractory angina pectoris and inoperable CAD, and patients with life-threatening ventricular arrhythmias that are refractory to all appropriate medical and surgical treatment. Usually, the patient's EF is less than 20%. However, patients with NYHA class III HF who are at risk for sudden death related to malignant arrhythmias are sometimes placed on the waiting list.

In the United States, cardiac transplantation is performed in member centers of the United Network for Organ Sharing (UNOS), an umbrella organization responsible for coordinating organ procurement, organ allocation, and statistical information. UNOS allocates donor hearts according to each patient's priority status, ABO group compatibility, body size match, and distance from the donor center. The highest priority is given to inpatients supported by mechanical circulatory assist devices for acute hemodynamic decompensation, patients maintained on assist devices who have significant device-related complications, patients requiring continuous infusion of single or multiple high-dose intravenous inotropic medications, and patients with a life expectancy of less than 7 days without transplantation.

The most common diagnoses in adult patients with HF who require cardiac transplantation include idiopathic or ischemic cardiomyopathy and complex CHD. Less common diagnoses include viral cardiomyopathy, postpartum cardiomyopathy, refractory valvular disease, primary myocardial diseases (e.g., sarcoidosis, amyloidosis), and drug-induced myocardial disease.

The first most common indication is idiopathic dilated cardiomyopathy, which accounted for approximately 46% of all cases in 2018.⁴⁷¹ Worldwide, ischemic cardiomyopathy is now the second most common indication for heart transplantation, accounting for 38% of all registered cases.⁴⁴⁴ Similar to the U.S. experience, nonischemic cardiomyopathy has been the most common indication worldwide, accounting for 53% of all registered cases.

The selection criteria have been broadened for both heart recipients and heart donors.⁴⁷² For example, patients with diabetes and those older than 65 years old are now considered transplantation candidates in many centers, though they were previously considered exclusion criteria.⁴⁷³ In addition, although centers generally prefer donors to be younger than 35 years old, the age criterion has been expanded to include suitable donors (e.g., those without cardiac risk factors or evidence of CAD) who are 60 years old or older. Suitability involves the absence of CAD or of major cardiac risk factors for CAD. Major contraindications to heart transplantation that continue to steer patients toward destination therapy with a VAD instead of transplantation include age greater than 70 years, chronic renal dysfunction, and high body mass index.⁴⁴⁷ Modifiable risk factors (e.g., pulmonary hypertension) remain the subject of ongoing clinical investigation. Classically, a PVR greater than 5 dyne•s•cm⁻⁵ unresponsive to pulmonary vasodilators is a relative contraindication, as it indicates a "fixed"

PVR, and has been associated with an increased risk for early mortality after orthotopic heart transplantation.⁴⁷³ Significant issues of non-compliance, lack of a social support structure, comorbidities with a poor prognosis, and significant drug and/or alcohol abuse may also render a patient ineligible for transplantation.

The manner in which the donor heart is implanted has also changed, with a shift away from the classic biatrial technique (originally described by Lower and Shumway in 1960) to the bicaval Wythenshawe technique described in the early 1990s. The advantages of a bicaval implantation technique include lower incidences of LA dilatation, diuretic use, atrial dysrhythmias, conduction disturbances, mitral and tricuspid valve incompetence, and RV failure. In addition, this technique has been demonstrated to be associated with shorter hospital stays. Furthermore, several series that compared the classic Shumway technique with the bicaval technique showed greater 12-month survival in the bicaval group.^{474,475}

An even more recent implantation technique further reduces the size of the native LA remnant to two islands containing the pulmonary veins on either side. This so-called "total" technique was described by Dreyfus and Carpentier in 1991.⁴⁷⁶

Because the recipient sinoatrial node is no longer routinely retained with either of the modern bicaval techniques, the concept of two P waves on the posttransplant ECG is now only of historical interest. Posttransplantation physiology, however, remains unchanged: the requisite denervation of the donor organ during harvesting results in the absence of both direct afferent and efferent neural innervation via autonomic or somatic neural pathways after implantation. In plain English, the transplanted heart functions in isolation from the recipient's nervous system, although the potential for direct activation of myocardial receptors (e.g., myocardial adrenergic receptors) by circulating factors is retained, as are all intrinsic myocardial reflexes (e.g., the Starling mechanism, the Anrep effect, the Bowditch effect). The loss of parasympathetic tone in the transplanted heart means that the resting heart rate is faster than normal, and heart rates of approximately 90 to 110 beats/min are usual and often necessary to maintain CO.

A clear understanding of what remains intact and what is lost in the transplanted heart dictates and guides posttransplant anesthetic management. Adequate preload is critical. Direct-acting drugs (e.g., epinephrine, isoproterenol) and pacing are maximally and immediately effective when needed to increase inotropy or chronotropy, respectively, in the transplanted heart. Indirect-acting drugs (e.g., epinephrine, dopamine) rely primarily on the release of epinephrine and norepinephrine from the adrenal glands. Digoxin retains its direct inotropic effect on the heart through inhibition of sodium-potassium adenosine triphosphatase, with resultant buildup of intramyocardial calcium, but loses its potential to decrease chronotropy (a parasympathetically mediated effect on the AV node). One should not expect a decrease or increase in heart rate after the administration of medications that cause bradycardia (e.g., fentanyl) or tachycardia (e.g., meperidine, pancuronium) as a side effect since these side effects work through neural mechanisms. Anticholinergic medications (e.g., atropine, glycopyrrolate)

are not expected to increase heart rate in the denervated heart but are always administered at the time of reversal of muscle relaxation by acetylcholinesterase inhibitors to reduce the adverse *extracardiac* cholinergic effects of the anticholinesterase drugs.

The denervated heart is subject to accelerated atherosclerotic disease, causing a significant 5-year incidence of CAD in transplant recipients, but these recipients do not experience angina. Significant dysrhythmias or arrhythmias in a transplanted heart should be considered harbingers of ischemia until proven otherwise.

CONGENITAL HEART DISEASE IN ADULTS

Background and Current Perspective

Medical and surgical advances in CHD surgery since the 1960s have significantly reduced the death rates of patients with CHD. Today, 85% to 95% of babies born with CHD can reach adulthood, and the number of adults with CHD now exceeds the number of children with CHD.⁴⁷⁷ Currently there is no formal database of adults with CHD but this population is increasing at a rate of more than 5% annually. Adult patients who present with CHD can have extremely complex conditions that require careful assessment and operative planning. They may present for cardiac surgery for the first time or for further palliative correction, either definitively or for residua. Most patients with CHD face a lifetime of significant challenges,⁴⁷⁸ ranging from the complications of long-term antibiotic prophylaxis to thromboembolic ventricular dysfunction resulting from cardiac arrhythmias and pulmonary hypertension.

Preoperative Anesthetic Considerations

The unique problems of this population include the multisystem effects of single-ventricle physiology, cyanosis, systemic right ventricle, complex intracardiac baffles, and failing subpulmonary right ventricle.⁴⁷⁷ The initial assessment, history taking, and physical examination of an adult patient with CHD must be broadly systemic to capture any noncardiac organ involvement. A full review of each organ system is beyond the scope of this chapter, but readers are referred to the excellent review articles by Chassot and Betetx⁴⁷⁸ and by Lovell.⁴⁷⁹ Appropriate consultation with the patient's cardiologist (pediatric or adult), as well as access to old medical records, is valuable, especially for patients who have complex CHD or have undergone complex surgical procedures. Preoperative echocardiography is extremely helpful in understanding the anatomy and the primary and compensatory cardiac function of patients with CHD.

One of the early clinical decisions to be made for an individual patient involves determining the appropriateness of surgical care in a given institution. Obviously, emergency procedures give very little latitude, but elective procedures should warrant careful assessment, especially of the abilities and experience of the anesthesiologist who will be caring for a patient with complex corrected or palliated CHD. In 2001, the Thirty-second Bethesda Conference on CHD advocated the creation of regional centers for the care of adults with CHD (ACHD centers), whose personnel would include a cardiac anesthesiologist and other subspecialists with expertise in the management of patients with CHD.⁴⁸⁰

BOX 54.17 Types of Adult Congenital Heart Disease of Great Complexity

- Conduits, valved or nonvalved
- Cyanotic congenital heart disease (all forms)
- Double-outlet ventricle
- Eisenmenger syndrome
- Fontan procedure
- Mitral atresia
- Single ventricle (also called double inlet or outlet, common, or primitive)
- Pulmonary atresia (all forms)
- Pulmonary vascular obstructive disease
- Transposition of the great arteries
- Tricuspid atresia
- Truncus arteriosus or hemitruncus
- Other abnormalities of atrioventricular or ventriculoarterial connection not included here (i.e., crisscross heart, isomerism, heterotaxy syndromes, ventricular inversion)

From Warnes CA, Williams RG, Bashore TM, et al. ACC/AHA 2008 guidelines for the management of adults with congenital heart disease: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Adults with Congenital Heart Disease). Developed in collaboration with the American Society of Echocardiography, Heart Rhythm Society, Angiography and Interventions and Society of Thoracic Surgeons. *Circulation.* 2008;118:2395–2451.

The conference recommended that all adult patients with complex (Box 54.17) and moderate (Box 54.18) CHD be referred to such ACHD centers. Current U.S.^{481,482} and Canadian⁴⁷⁷ updates to these guidelines continue to support these recommendations.

Intraoperative Anesthetic Considerations

Discussion of specific anesthetic management techniques for particular congenital heart defects in adult patients is not within the scope of this chapter. However, a few basic principles are essential for all anesthesiologists to know, especially those who do not have specialized training in cardiac anesthesia or CHD but who may be faced with an older patient with CHD who needs urgent noncardiac surgery.

Preparation of the anesthetic plan requires detailed knowledge of the anatomy and physiology of the lesion.⁴⁷⁹ A skilled multidisciplinary team is very helpful for providing optimal management.^{483,484} During the preoperative assessment of an adult with CHD, an extensive review should be conducted to elicit signs of CHF, cyanosis, or compromised peripheral vascular disease. Anesthetic considerations are lesion specific and are discussed in several reviews.^{479,485,486}

Principle 1: The Presence or Absence of Cyanosis. Cyanosis is usually a marker for complex CHD. Chronic cyanosis can lead to the development of abnormalities of RBC production and hemostasis. Elevated levels of erythropoietin in cyanotic patients can lead to a hyperviscosity syndrome and increased risk of neurologic injury.

In any patient with cyanotic CHD, the anesthesiologist should establish intravenous fluid therapy and monitor

BOX 54.18 Diagnoses in Adult Patients With Congenital Heart Disease of Moderate Complexity

Anomalous pulmonary venous drainage, partial or total
 Aorto-left ventricular fistula
 Atrioventricular septal defect (partial or complete)
 Coarctation of the aorta
 Ebstein anomaly
 Infundibular right ventricular outflow obstruction of significance
 Ostium primum atrial septal defect
 Patent ductus arteriosus (not closed)
 Pulmonary valve regurgitation (moderate to severe)
 Pulmonary valve stenosis (moderate to severe)
 Sinus of Valsalva fistula or aneurysm
 Sinus venosus atrial septal defect
 Subvalvular or supravalvular aortic stenosis (except HOCM)
 Tetralogy of Fallot
 Ventricular septal defect with:
 • Absent valve or valves
 • Aortic regurgitation
 • Coarctation of the aorta
 • Mitral valve disease
 • Right ventricular outflow tract obstruction
 • Straddling tricuspid or mitral valve
 • Subaortic stenosis

HOCM, Hypertrophic obstructive cardiomyopathy.

From Warnes CA, Williams RG, Bashore TM, et al. ACC/AHA 2008 guidelines for the management of adults with congenital heart disease: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Adults with Congenital Heart Disease). Developed in collaboration with the American Society of Echocardiography, Heart Rhythm Society, Angiography and Interventions, and Society of Thoracic Surgeons. *Circulation*. 2008;118:2395–2451.

urine output to overcome the adverse circulatory effects of perioperative fasting, dehydration, and intraoperative hypovolemia. These conditions cause hyperviscosity, which can be associated with hemostatic abnormalities. Many patients with congenital repairs that include a conduit or graft are maintained on antiplatelet medications, and this should be noted. Arterial pressure monitoring may be prudent, and the decision to place a central venous catheter should be made in careful consideration of the patient's anatomy and proposed surgical repair.

Principle 2: The Presence or Absence of Intracardiac or Extracardiac Shunts. Intracardiac shunts are classically located at the level of the atria or ventricles. The magnitude of the shunt is often expressed in terms of the ratio of pulmonary blood flow (Q_p) to systemic blood flow (Q_s): Q_p/Q_s . Balanced flow, or a ratio equal to 1, is the hallmark of normal physiology. A Q_p/Q_s ratio greater than 1 is generally consistent with acyanotic heart disease and luxuriant pulmonary blood flow. A Q_p/Q_s ratio less than 1 would be consistent with cyanosis. Intracardiac shunts and the Q_p/Q_s are often bidirectional, depending on situational physiology, and therefore must always be considered to be a risk for systemic embolization. The location and size of intracardiac shunts are critically important to the management of anesthesia care for patients with CHD.⁴⁷⁸ The direction and magnitude of flow across an intracardiac or extracardiac

shunt is directly related to the balance between PVR and SVR, a relationship that can be managed by vasoactive agent administration.

1. The direction of shunt flow is important. Shunts can be right, left, or bidirectional. Shunt flow can be influenced by changes in driving pressure across a shunt or pressure in the receiving chamber. The high intrathoracic pressure that can occur with mechanical ventilation, coughing, the Valsalva maneuver, bronchospasm, or positive end-expiratory pressure can actually reverse the direction of a left-to-right shunt or at least make it bidirectional. Therefore, paradoxical embolization by clot or air is a real risk in patients with intracardiac shunts and warrants fastidious attention to de-airing intravenous lines and careful administration of drugs by syringes through ports in intravenous lines.
2. The size of the intracardiac defect is important. Shunts are commonly labeled as restrictive or nonrestrictive according to their blood velocity or pressure characteristics measured at catheterization or by echocardiography. Large defects with associated low-pressure gradients across them have nonrestrictive flow. In addition, larger defects can be expected to have a greater effect on downstream structures and pressures. Small defects with high pressure or gradients restrict the amount of blood flow. The mainstays of the anesthetic management of shunt lesions are understanding and controlling factors that may influence the direction and magnitude of the shunt. Shunt flow and direction are directly influenced by both PVR and SVR. Elevated SVR and decreased PVR generally increase the magnitude of left-to-right shunting. Conversely, decreased SVR and/or increased PVR decrease the magnitude of left-to-right shunting, or will create a right-to-left shunt. Mechanical ventilation, the inspired oxygen concentration, carbon dioxide levels, and the hemodynamic effects of anesthetic drugs are all used to achieve certain therapeutic goals in relation to PVR and SVR, and hence, shunt management. Oxygen is a potent pulmonary vasodilator, as is hypocapnia. If the clinical problem is luxuriant pulmonary blood flow, a high FiO_2 should be avoided. Similarly, the direction and magnitude of the shunt flow determine whether one should hyperventilate a patient to achieve a low $Paco_2$ or tolerate a high-normal $Paco_2$.
3. In the presence of a shunt, the potential dilation of the cardiac chambers and resultant increase in chamber volume and pressure are important. A shunt above the level of the AV valves, such as a left-to-right shunt at the atrial level, usually causes right-sided chamber enlargement. Left-to-right shunts at any level that increase pulmonary blood flow can lead to elevations in PVR and pulmonary arterial pressure. Furthermore, exposure to elevated pulmonary pressure and afterload leads to continued right heart enlargement, RV failure, bidirectional shunting, and in extreme cases, a reversal of shunt direction, or right-to-left shunting. Clinically this manifests as cyanosis. When PVR becomes elevated, fixed, and irreversible, this is referred to as Eisenmenger syndrome.

Extracardiac shunts can be native to CHD (e.g., aberrant pulmonary venous return), surgically created (e.g., Blalock-Taussig shunt), or compensatory (e.g., aortopulmonary

collateral formation with long-standing cyanotic heart disease). Special mention of certain aortopulmonary shunts, used commonly in the past to palliate some forms of cyanotic heart disease, is warranted. The proximal connection may be to the ascending aorta, brachiocephalic trunk, or subclavian artery. Because blood flow through this type of shunt is dependent on systemic blood pressure, systemic hypotension may lead to worsening hypoxemia. Furthermore, long-term exposure to these extracardiac shunts can result in left-sided chamber enlargement and dysfunction secondary to chronic volume overload.

Principle 3: The Presence of Pulmonary Hypertension. Pulmonary arterial hypertension is defined as a mean PAP greater than 25 mm Hg or greater than 30 mm Hg with exercise. Some degree of pulmonary hypertension develops in 5% to 10% of adult patients with CHD. This is associated with exercise intolerance and decreased functional capacity, which appear to have important prognostic implications.⁴⁸⁴

The anesthetic management of patients with pulmonary hypertension can be challenging. Invasive monitoring and careful anesthetic titration may be required. Regional anesthesia may be used for appropriate procedures, but neuraxial blocks should be administered with caution because of the sympathetic blockade produced. Patients with significant pulmonary hypertension are very sensitive to preload; therefore, hypovolemia, should be treated immediately and aggressively. Pulmonary hypertension can be managed pharmacologically and mechanically. Factors associated with decreasing PVR and, subsequently, reductions in PAP are listed in Box 54.19.⁴⁷⁹

Principle 4: The Presence of Ventricular Dysfunction. As in adult patients with acquired heart disease, the presence of ventricular dysfunction is an important risk factor for morbidity and mortality, both in the perioperative period and in the long term. Regarding RV dysfunction, predictors of a poor outcome may include pulmonary hypertension, pulmonic valve incompetence, and consequent dysfunction of the subpulmonic ventricle.⁴⁸⁶ LV dysfunction has been observed after repair of tetralogy of Fallot in the adult. Risk factors associated with this dysfunction include male gender, LV enlargement, duration of shunt before repair, history of arrhythmia, long QRS duration, presence of an ICD, and moderate-to-severe RV dysfunction preoperatively.⁴⁸⁷

OTHER CARDIAC PROCEDURES

Surgical Ablation of Atrial Fibrillation

In the United States, 2.7 to 6.1 million persons are estimated to have atrial fibrillation.⁴⁸⁸ Among Medicare patients 65 years old or older, the prevalence of atrial fibrillation increased from 3.2% in 1992 to 6.0% in 2002 and is higher in older patients.⁴⁸⁹ Stroke remains the most feared complication of atrial fibrillation; stroke risk is fourfold to fivefold higher in patients with atrial fibrillation than in patients without this arrhythmia. Additionally, atrial fibrillation causes 24% of strokes in patients older than 80 years old.⁴⁸⁸

With regard to persistent atrial fibrillation, both the left atrium and the pulmonary veins have been implicated in the pathogenesis and maintenance of persistent atrial fibrillation. Paroxysmal atrial fibrillation, conversely, may result

BOX 54.19 Factors Associated With Decreasing Pulmonary Vascular Resistance and Reductions in Pulmonary Artery Pressure

Decrease in Pulmonary Vascular Resistance

- Increasing PaO_2
- Hypocapnia
- Alkalemia
- Minimizing intrathoracic pressure
 - Spontaneous ventilation
 - Normal lung volumes
 - High-frequency and jet ventilation
 - Avoidance of sympathetic stimulation
 - Deep anesthesia
- Pharmacologic methods
 - Isoproterenol
 - Phosphodiesterase III inhibitors
 - Prostaglandin infusion (PGE_1 and PGI_2)
 - Inhaled nitric oxide

Increase in Pulmonary Vascular Resistance

- Sympathetic stimulation
 - Light anesthesia
 - Pain
- Acidemia
- Hypoxia
- Hypercapnia
- Hypothermia
- Increased intrathoracic pressure
 - Controlled ventilation
 - Positive end-expiratory pressure
 - Atelectasis

PaO_2 , Partial pressure of arterial oxygen; PGI_2 , prostacyclin.

From Lovell AT. Anaesthetic implications of grown-up congenital heart disease. *Br J Anaesth.* 2004;93:129–139.

from the juxtaposition of the pulmonary vein endothelium and the LA endocardium. The transition of the electrical signal from one type of tissue to the other is probably responsible for this arrhythmia.

The classic Maze procedure is currently the most effective curative therapy for atrial fibrillation.^{490,491} To simplify the procedure and improve its results, Cox and colleagues modified the operation twice, hence its current name, the Cox-Maze III procedure.⁴⁹⁰ This procedure appears to cure atrial fibrillation in 99% of cases.⁴⁹¹ Current indications for the Cox-Maze III operation include drug intolerance, arrhythmia intolerance, and recurrent embolic events.⁴⁹²

Ablative treatments for atrial fibrillation involve making RA and LA incisions and cryolesions to interrupt the multiple reentrant circuits that cause atrial fibrillation. Isolating the pulmonary veins and excising the LAA are integral parts of the Maze procedure, which requires CPB and cardiac standstill using cardioplegia.^{493–495} This procedure can be performed through a minimally invasive chest incision, as an alternative to sternotomy, while the patient is undergoing CPB.

A study done by Prasad and co-workers found that the Cox-Maze III procedure effectively cured atrial fibrillation when the operation was performed alone or concomitantly with coronary or valve procedures.⁴⁹² In patients who underwent such concomitant operations, the Cox-Maze III

TABLE 54.21 Energy Sources for Surgical Ablation of Atrial Fibrillation

Energy Type	Endocardial Application	Epicardial Application	Flexible Probe	Assess Transmural	No Char	Rapid
Radiofrequency*	+	+	+	+	-	+
Microwave	+	+	+	-	+	+
Cryotherapy	+	+	-	-	+	-

*Radiofrequency energy may be delivered in unipolar or bipolar fashion. A commercially available bipolar instrument allows the operator to assess transmurality. From Gillinov AM, Blackstone EH, McCarthy PM. Atrial fibrillation: current surgical options and their assessment. *Ann Thorac Surg*. 2002;74:2210–2217.

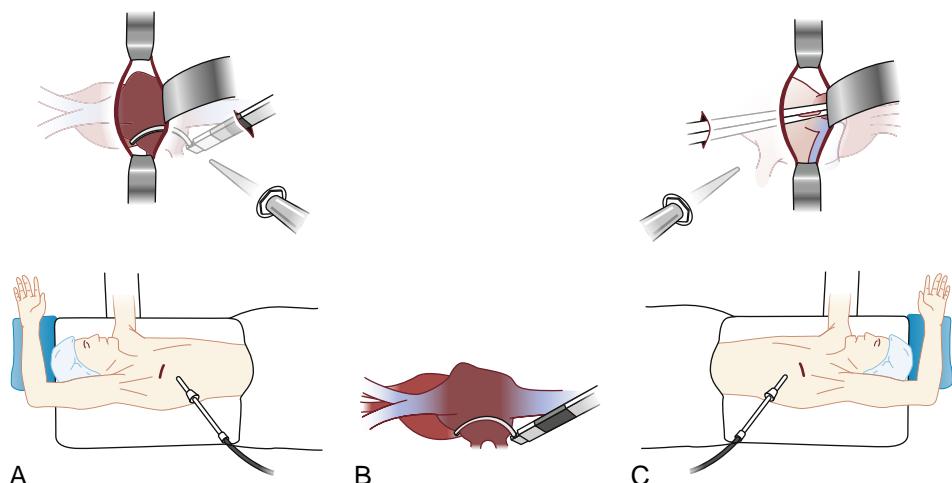


Fig. 54.54 “Keyhole” approach for minimally invasive epicardial pulmonary vein isolation with excision of the left atrial appendage. (A) Access to the right pulmonary veins is gained through a keyhole incision under endoscopic guidance. (B) A bipolar radiofrequency clamp is used to isolate the left atrial cuff adjacent to the right pulmonary veins. (C) Stapled excision of the left atrial appendage after the left pulmonary veins are isolated with a technique similar to that shown for the right pulmonary veins. (From Gillinov AM, Wolf RK. Surgical ablation of atrial fibrillation. *Prog Cardiovasc Dis*. 2005;48:169–177.)

procedure did not significantly add to the mortality or morbidity associated with the revascularization or valve repair procedure.

The underuse of the Maze procedure in the past probably reflected the procedure’s perceived complexity. Currently, newer technologies enable the rapid creation of lines of conduction blockade, which surgeons use to ablate atrial fibrillation in patients undergoing concomitant cardiac surgical procedures. These technologies include alternate energy sources such as radiofrequency and microwave energy, ultrasound, cryotherapy, and laser (Table 54.21). These technologies are also used in minimally invasive surgical ablation procedures to cure isolated atrial fibrillation.⁴⁹¹ Furthermore, the development of epicardial probes has facilitated off-pump ablation of atrial fibrillation in the beating heart.

Because cardiac structures can be visualized directly during open surgical procedures, ablation lines can be created safely, thus avoiding the complication of pulmonary vein stenosis. An epicardial approach to ablation eliminates the risk of esophageal injury.^{490,491} The advantage of a surgical approach is the ease of excising the LAA, which virtually eliminates the risk of stroke. However, isolation of the pulmonary vein and excision of the LAA can now be done through the minimally invasive “keyhole approach” (Fig. 54.54) or a thoracoscopic approach.⁴⁹⁶ Both epicardial approaches can be used without the need for CPB.⁴⁹¹ As experience with the Cox-Maze procedures increases and

the technology continues to advance, the goal is to improve patients’ quality of life and free them from the need for antiarrhythmic and anticoagulant medications.

Pericardial Tamponade and Constrictive Pericarditis

Pericardial Tamponade. The pericardial sac has two layers: the outer, parietal pericardium; and the inner, visceral pericardium (epicardium), which is directly adherent to the surface of the heart. The normal amount of fluid between the two layers is 15 to 30 mL, which generates a pressure that is 5 mm Hg less than the CVP and approximates the pleural pressure.

Cardiac tamponade occurs when the volume of fluid trapped in the pericardial sac increases and compresses the heart, thereby compromising CO.⁴⁹⁷ In classic tamponade, the circulation adapts by increasing the systemic venous and pulmonary venous pressures to equalize with the pericardial pressure so that total collapse of the cardiac chambers is prevented.⁴⁹⁸ The PCWP and the LV, RV, and RA diastolic pressures increase to equalize with the pressure in the pericardium. As a result, the atrial and ventricular diastolic transmural pressures are essentially zero, a hallmark of pericardial tamponade. Although this virtual loss of pressure reduces the stroke volume, high adrenergic tone helps to partially preserve CO by increasing the heart rate.

If enough fluid accumulates, the pericardium reaches a stage at which it can no longer distend; therefore, the

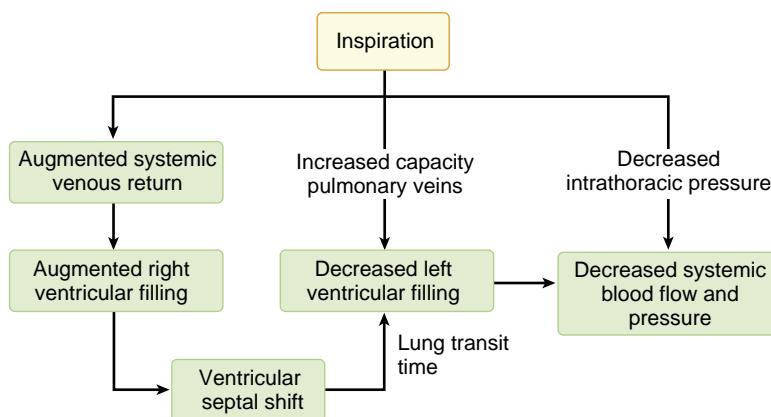


Fig. 54.55 Physiology of paradoxical pulses in cardiac tamponade. (From Fowler NO. Diseases of the pericardium. *Curr Probl Cardiol*. 1978;2:6-38.)

total pericardial volume no longer changes throughout the respiratory cycle.⁴⁹⁸ This results in a phenomenon referred to as ventricular interdependence, wherein any change in the volume of one side of the heart causes the opposite change in the volume of the other side. During inspiration, increased venous return and filling of the right heart cause the interatrial and interventricular septa to bulge to the left, compromising the ability of the LV to eject. Opposite changes occur during expiration. Such ventricular interdependence is evident on echocardiography.

Ventricular interdependence manifests clinically as pulsus paradoxus,⁴⁹⁹ an exaggeration of the normal diminution of the radial pulse on inspiration (Fig. 54.55). *Pulsus paradoxus* is defined as a drop in systolic blood pressure exceeding 10 mm Hg during inspiration.⁵⁰⁰ In particularly severe cases, no brachial or radial pulse may be palpable on inspiration.⁵⁰¹

Cardiac tamponade is not necessary to cause pulsus paradoxus. Wide swings in intrathoracic pressure and conditions such as pulmonary embolism or hypovolemic shock can also give rise to the phenomenon. Additionally, patients with cardiac tamponade may not have pulsus paradoxus if they have coexisting aortic insufficiency, ASD, or preexisting elevated LVEDP resulting from LV hypertrophy or dilatation.

Fig. 54.56 illustrates the pressure-volume relationship in the pericardium, depending on whether the effusion is accumulating slowly or rapidly.⁵⁰¹ This J-shaped curve suggests that a sudden increase in pericardial fluid volume of 100 to 200 mL can increase pericardial pressure to 30 mm Hg or more and cause severe cardiac tamponade. The faster the fluid accumulation, the more severe is the hemodynamic compromise. Causes of this type of acute tamponade include rupture of a thoracic aortic aneurysm, traumatic mediastinal injury, and accidental puncture of a cardiac chamber or blood vessel in the CCL.⁴⁹⁸

When accumulation of pericardial fluid is slower, compliance of the parietal pericardium is greater, and changes in volume produce smaller rises in pressure than they would if fluid accumulated more rapidly. As pericardial volume increases, so does intrapericardial pressure, with a compensatory increase in CVP to maintain a gradient to allow cardiac filling.⁴⁹⁷ When pericardial compliance cannot increase further, the intrapericardial pressure gradually equalizes with the pressures in the cardiac chambers.

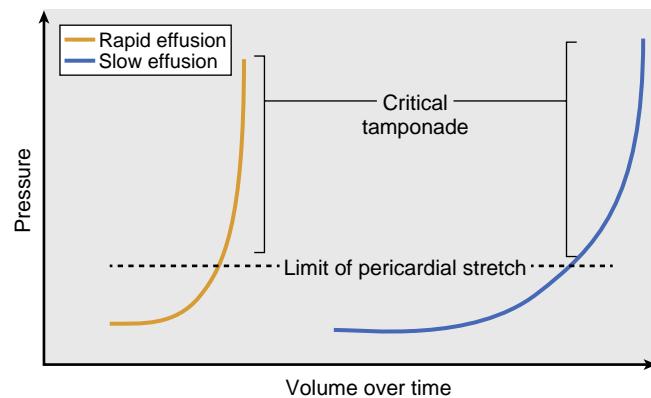


Fig. 54.56 Cardiac tamponade. Pericardial pressure-volume (or strain-stress) curves are shown in which the volume increases slowly or rapidly over time. On the left, rapidly increasing pericardial fluid first reaches the limit of the pericardial reserve volume (the initial flat segment) and then quickly exceeds the limit of parietal pericardial stretch, thus causing a steep rise in pressure, which becomes even steeper as smaller increments in fluid cause a disproportionate increase in the pericardial pressure. On the right, a slower rate of pericardial filling takes longer to exceed the limit of pericardial stretch because more time is available for the pericardium to stretch and for compensatory mechanisms to become activated. (From Spodick DH. Acute cardiac tamponade. *N Engl J Med*. 2003;349:684-690.)

CO gradually declines, accompanied by a compensatory tachycardia, peripheral vasoconstriction, and increased contractility.

SYMPOTMS AND SIGNS. Patients with pericardial tamponade may present with pain or feelings of fullness in the chest, dyspnea, lethargy, fever, cough, weakness, fatigue, anorexia, or palpitations.⁴⁹⁷ The Beck triad may appear as a combination of three signs of severe tamponade: low blood pressure, increased jugular venous pressure, and distant heart sounds. However, patients whose tamponade develops secondary to a chronic medical condition (e.g., malignant disease, end-stage renal disease, collagen vascular disease) may not present with the classic findings of the Beck triad.

ECHOCARDIOGRAPHIC FEATURES OF PERICARDIAL TAMPOADE. Of the various echocardiographic signs of cardiac tamponade (Box 54.20), one of the best established is diastolic collapse of the right atrium or right ventricle. RV collapse, seen in early diastole, appears as an invagination of the RV free wall (Fig. 54.57). In contrast, RA collapse is seen in late

diastole and early systole and appears as an invagination of the RA wall (see Fig. 54.57). The timing of the collapse of the two chambers is related to the lowest intracavitory pressures in those chambers (i.e., early diastole for the right ventricle and late diastole to systole for the right atrium). The presence of both RV and RA collapse indicates hemodynamically significant pericardial effusion. Left-sided heart collapse is rarely seen, both because the thickness and stiffness of the LV enable it to resist collapse and because the left atrium is located posteriorly. However, in patients with very large effusions, fluid does accumulate behind the left atrium and eventually collapses this chamber.⁵⁰² The presence of LA collapse is highly specific for tamponade.⁵⁰¹

ANESTHETIC MANAGEMENT OF PERICARDIAL TAMPONADE. Indications for pericardiocentesis (surgical drainage of the pericardial effusion) were summarized by Soler-Soler and co-workers (Box 54.21).⁵⁰³ Surgical intervention for pericardial disease warrants invasive monitoring, which should involve an intraarterial catheter and possibly a central venous catheter. Large-bore intravenous access should be secured prior to anesthesia induction.

BOX 54.20 Doppler Echocardiographic Signs of Cardiac Tamponade

Exaggerated inspiratory variation of the two ventricles (inspiratory expansion of the right ventricle and simultaneous compression of the left ventricle; reciprocal changes in the expiratory phase)

Right atrial collapse

Right ventricular collapse

Left atrial collapse

Left ventricular collapse

Inferior vena cava plethora

Abnormal increased respiratory variation in transvalvular blood

flow velocities (mitral and aortic flow reduction in the inspiratory phase)

From Pepi M, Muratori M. Echocardiography in the diagnosis and management of pericardial disease. *J Cardiovasc Med* (Hagerstown). 2006;7:533–544.

In patients who are severely hemodynamically compromised, one surgical option is to perform pericardiocentesis or subxiphoid exploration using local anesthesia⁵⁰⁴ and inducing general anesthesia after the tamponade has been partially relieved. If general anesthesia is planned, the watchwords in the management of cardiac tamponade are *fast, full, and strong*. Heart rate should be maintained at a high normal level so as to maintain CO in a stroke-volume-limited ventricle. Intravenous fluids should be administered before induction of anesthesia to optimize preload. Increasing intravascular volume helps to increase the effective filling pressures of the heart, restore the gradient between the chambers, and increase arterial pressure.

Any manipulation that can decrease venous return to the heart should be avoided. This includes controlled positive-pressure ventilation with large tidal volumes, which may significantly decrease preload and CO.⁵⁰⁴ If general anesthesia is required, one option is to perform prep and drape to the point of surgical incision and then, after induction of anesthesia, rapidly perform evacuation of the pericardial effusion. Another option is to induce general anesthesia but to allow the patient to breathe spontaneously, avoiding positive intrathoracic pressure, until the pericardial sac is opened.⁵⁰⁴ Alternatively, a ventilation pattern of high rate and low tidal volumes can be used to minimize mean airway pressure.

Drugs that may cause myocardial depression should be avoided and thus etomidate is often chosen as the induction agent of choice.⁵⁰⁵ Additionally, bradycardia should be avoided because tachycardia is the most important compensatory mechanism for preserving CO. The use of ketamine to induce general anesthesia for creation of a pericardial window has been described.⁵⁰⁶

Once cardiac tamponade is relieved, endogenously generated and exogenously administered catecholamines may cause sudden, severe increases in blood pressure and heart rate. This phenomenon should be anticipated and treated.

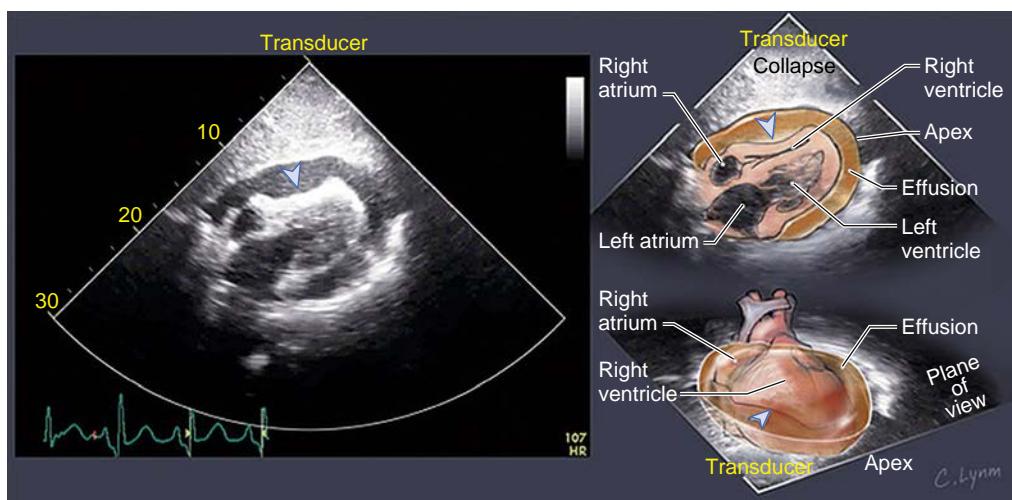


Fig. 54.57 Echocardiogram (and drawings) showing pericardial effusion causing cardiac tamponade. A subcostal view in early diastole shows a large circumferential pericardial effusion compressing the heart, with the right ventricle completely collapsed (arrowheads). (From Roy CL, Minor MA, Brookhart MA, et al. Does this patient with a pericardial effusion have cardiac tamponade? *JAMA*. 2007;297:1810–1818.)

Constrictive Pericarditis

PATOPHYSIOLOGY. Constrictive pericarditis is said to be present when the diastolic filling of the heart is restricted by a thickened and adherent pericardium. Several causes of constrictive pericarditis have been identified, including infectious processes. In addition, radiation to the chest area and cardiac surgery itself have emerged as additional causes of constrictive pericarditis over the past few decades.⁵⁰⁷ If the two pericardial layers—visceral and parietal—become adherent as a result of fibrosis and calcification of the pericardium, the space between the two layers is obliterated.⁵⁰⁷

SIGNS AND SYMPTOMS. Typically, patients with constrictive pericarditis present with signs and symptoms of right-sided HF. A high driving pressure across the valves causes a rapid filling of the ventricles in diastole and results in sudden increase in the ventricular pressures. As the ventricular pressures exceed the atrial pressures, flow abruptly stops, and pressures are elevated and equalized in all four chambers.⁵⁰⁷

Another sign seen in constrictive pericarditis is the Kussmaul sign (an exaggeration of the *x* and *y* descent on the CVP tracing), which is elicited in response to increased blood return to the right side of the heart on inspiration. Because the thickened pericardium no longer allows the heart to expand to accommodate all the blood flowing into it, the pressure generated by this returning volume is transmitted to the central venous system.⁵⁰⁸

As in pericardial tamponade, pulsus paradoxus is also usually seen in constrictive pericarditis. However, in some cases, thickening of the pericardium can shield the myocardium from the effects of the respiratory cycle; therefore, pulsus paradoxus may not be that obvious. Dyspnea and orthopnea are common in patients with constrictive pericarditis because the low CO caused by constrictive pericarditis severely reduces the patient's exercise tolerance and, in extremely advanced cases, causes cardiac cachexia and muscle wasting.⁵⁰⁹ Ascites and peripheral edema may also be seen. Echocardiography is particularly helpful in differentiating constrictive pericarditis from other

disease processes with similar presenting symptoms, such as restrictive cardiomyopathy.^{509,510}

ANESTHETIC CONSIDERATIONS FOR PERICARDIECTOMY. The definitive treatment for constrictive pericarditis is pericardectomy.⁵¹¹ This procedure is usually performed through a sternotomy, but it can also be performed through a left thoracotomy incision. In patients with severe cases of the disease, pericardectomy may involve stripping the pericardium off the heart, which may necessitate the use of CPB.

The hemodynamic management of a patient undergoing pericardectomy for constrictive pericarditis is similar to that of a patient having a surgical intervention for pericardial effusion and tamponade. However, because the pericardium may need to be “peeled” from the surface of the heart, pericardectomy carries an additional risk of arrhythmia and injury to the myocardium itself, including rupture of one of the chambers. The raw surface of myocardium and the peeling of pericardium cause the release of fibrinolytic activators, and the complement and coagulation cascades are activated. Thus, pericardectomy can be associated with generalized bleeding due to consumptive coagulopathy, in addition to actual surgical bleeding. Measures should be taken to avoid and treat arrhythmias and massive intraoperative bleeding.

Cardiac and Aortic Trauma

Penetrating Cardiac Injury. With the increase in violent crime over the past few decades, the number of cases of gunshot and stab wounds to the heart has risen. Patients with penetrating cardiac injury may be critically unstable because of hemorrhage or cardiac tamponade, or they may have a benign injury to the heart.⁵¹² Hemodynamic and ECG changes may be deceptively absent in many cases. The absence of these changes does not rule out the presence of life-threatening injury to the heart or its surrounding structures.⁵¹³ Echocardiography can identify fluid or blood around the heart and can inform the surgical decision regarding whether to perform a sternotomy or to use an alternative approach.^{512,514}

The anesthetic management of patients with penetrating cardiac injury is similar to that of other trauma patients. Unstable patients require emergency surgical intervention. The anesthesiologist must ascertain that blood is available on first knowledge of impending surgery for penetrating cardiac injury. With critically unstable patients, the anesthesiologist must secure the airway, establish intravenous access, and place the monitoring lines while the surgical incision is being made. An assistant can be very helpful in efficiently caring for such a patient. In planning the induction of anesthesia and the securing of the airway, the anesthesiologist should assume that the patient probably has a full stomach and volume depletion. Monitoring lines should include an arterial line and a central venous catheter. Barring any contraindications, on the basis of the scant history that may be available at that time, TEE should be considered as an additional monitor. Anesthesia management involves attempting to maintain stable hemodynamics and prevent recall. As in any trauma case, if the patient is unstable, controlled postoperative ventilation is usually preferred, including transportation to the ICU with full monitoring and ventilatory support.

BOX 54.21 Indications for Pericardiocentesis and Surgical Drainage

Pericardiocentesis is indicated in patients with overt clinical tamponade, in patients with suspected purulent pericarditis, and in patients with idiopathic chronic large pericardial effusion.

Indications for surgical drainage are tamponade, either unresolved or relapsing after pericardiocentesis, and persistent active illness 3 weeks after hospital admission.

Pericardial drainage does not seem to be warranted in the initial management of patients with large pericardial effusions without clinical tamponade because of its low diagnostic yield and poor influence on the evolution of pericardial effusion.

Even the presence of echocardiographic right chamber collapse (suggesting raised intrapericardial pressure) does not by itself warrant pericardial drainage because most of these patients do not develop overt tamponade.

Traumatic Aortic Injury. Blunt aortic injury, which can result in either traumatic aortic transection (TAT) or acute rupture, is one of the most common causes of death from blunt trauma, second only to head injury. On arrival at the hospital, only 25% of patients with blunt thoracic trauma and resultant aortic injury (TAT or acute rupture) are alive.⁵¹⁵ Furthermore, the prognosis of patients who survive the initial injury and remain untreated is poor: 30% die within the first 6 hours, 50% die in the first 24 hours, and 90% die within the first 4 months.⁵¹⁶

In blunt trauma to the chest, the mechanism of injury to the thoracic aorta is sudden traction on the relatively immobile portion of the aorta, the isthmus, as a result of sudden deceleration.⁵¹⁵ The isthmus is distal to the left subclavian artery, proximal to the third intercostal artery, and fixed to the left PA by the ligamentum arteriosum. This area of attachment of the ligamentum arteriosum acts as a hinge for movement of the aortic arch; thus, it receives the brunt of the shearing force during violent injury to the chest area. Hence, it is the most common location for rupture of the aorta (50%-70%), followed by the ascending aorta or the aortic arch in 18% and the distal thoracic aorta in 14% of cases.⁵¹⁵ The spectrum of injuries to the thoracic aorta in blunt thoracic trauma ranges from simple subintimal hemorrhage to complete aortic transection.

The diagnosis of the aortic injury may be made on the basis of a plain chest radiograph, computed tomography, angiography, or TEE. TEE can also detect cardiac tamponade, left pleural effusion, hypovolemia, ventricular dysfunction from myocardial contusion, or vascular injuries from penetrating chest wounds.⁵¹⁷ Diagnosing TAT with TEE requires recognition of a mural flap at the site of intimal disruption and corresponding deformities of the aortic wall caused by a contained rupture. A gap of more than 7 mm between the probe and the aorta at the level of the proximal descending thoracic aorta, in addition to blood between the aortic wall and the pleura, strongly suggests disruption of the aorta. TEE can also be useful in monitoring the progression of a small intimal tear or as a screening tool for patients who have a normal mediastinum after blunt thoracic trauma.

Management of TAT focuses on the high risk of rupture that this injury entails. The unpredictable timing of this event adds to the concerns regarding these patients. Aggressive management of blood pressure in the preoperative period is necessary to minimize the risk of rupture.⁵¹⁸ Systolic blood pressure should not exceed 100 mm Hg, and the heart rate should not exceed 100 beats/min. The use of β -blockers and nitroglycerin or sodium nitroprusside infusions is recommended.

Intraoperative monitoring should include an arterial catheter and central venous access and a large-bore IV should be secured. The keys to anesthetic management of patients with TAT are avoiding wide swings in systolic blood pressure and continuously monitoring the patient for end-organ ischemia. Because many operations on patients with TAT are performed on an emergency basis, it may be necessary to use a modified rapid-sequence induction. Despite all precautions, marked swings in blood pressure are often seen during induction and laryngoscopy. Agents such as nitroglycerin and esmolol should be immediately available to treat any undesirable hypertension during

endotracheal intubation, TEE probe insertion, and surgical incision.

Surgical repair of injuries to the ascending aorta or aortic arch typically requires CPB with DHCA. Injuries to the descending thoracic aorta may require open repair of a short segment. Challenges include preservation of vital organs, particularly the spinal cord. In open procedures, use of partial LHB can facilitate the operation and at the same time preserve visceral organ and spinal cord perfusion. This technique also prevents aggravation of any other injuries that the patient may have sustained because this type of bypass does not require large-dose heparin.⁵¹⁶ Although open repair is possible, transcutaneous endovascular aortic repair has emerged as the preferred intervention whenever possible (STS class I recommendation; level of evidence B).^{519,520}

Ischemic and Other Emergencies in the Cardiac Catheterization Laboratory

With rapid advances in interventional cardiology, the number of patients in the United States and Europe who undergo interventional therapy in a CCL is increasing. Such emergencies include coronary artery dissection, free rupture, tamponade, foreign body embolism, and wire entrapment. Patients may be at high risk for complications in the CCL if they have unfavorable coronary anatomy or severe ventricular dysfunction. In some cases, cardiac surgery and cardiac anesthesia services should be consulted and placed on standby for a possible emergency intervention. Rapid surgical intervention, such as revascularization or temporary mechanical support, is crucial to reducing the mortality and morbidity of patients who suffer angiography-related accidents.

When ischemic complications occur during cardiac catheterization with percutaneous transluminal coronary angioplasty and stenting, pharmacologic intervention and, possibly, a device such as an IABP or VAD may be used to stabilize the patient's hemodynamic condition before the patient is transferred to the operating room on an urgent or emergency basis for open coronary revascularization. Patients may be intubated on an emergency basis in the CCL. Patients with coronary dissection or severe ischemia may require CPR during transport to the operating room.⁵²¹ Although the anesthesiologist, cardiologist, or both may lead these resuscitative efforts, all members of the catheterization team—physicians, nurses, and technologists—should complete a course in basic CPR; additionally, certification in advanced cardiac life support, with annual recertification, is strongly urged.⁵²²

If the patient is not already intubated, the choice of induction agents should be tailored to the patient's hemodynamic condition; one should avoid hypotension and tachycardia while keeping in mind the patient's NPO (nothing by mouth) status. Perioperative monitoring of these patients should include an arterial line and central intravenous access, which should be established as soon as possible. TEE can be of immense importance in both diagnosing the cardiac problem and monitoring the patient intraoperatively.

Other procedures performed in the CCL, invasive vascular laboratory, and electrophysiology laboratory—notably aortic aneurysm stenting and ablation procedures—may also require operating room backup. A dedicated surgical

team should be available in the event of a life-threatening complication. Planning for the possibility of conversion to an open procedure, such as in cases of an aneurysm leak or catheter perforation of a major blood vessel or cardiac chamber, is imperative. A hybrid interventional cardiovascular suite is ideal for such events.⁵²³ In institutions without such a hybrid suite, urgent transportation to a general operating room may be necessary.

Optimal care also depends on the ready availability of a stat laboratory. Blood gas analysis, as well as the assessment of electrolytes and hemoglobin or hematocrit, is important in urgent situations. Patients undergoing procedures for coronary revascularization may be receiving platelet inhibitor drugs. Conventional coagulation tests may not be sufficient for diagnosing clinical bleeding in these patients, therefore POC tests that evaluate platelet function and viscoelastic testing to manage perioperative bleeding are useful.⁵²³

Compared with elective cardiac surgical procedures, emergency cardiac surgery carries higher mortality and morbidity risks, especially if the patient is in cardiogenic shock.⁵²⁴ Identifying high-risk patients going to the CCL for interventional procedures, as well as communication about these high-risk patients among the interventional cardiologists, cardiac surgeons, and cardiac anesthesiologists, is of prime importance in improving the outcome of these emergency procedures.

Procedures in the Hybrid Operating Room

GENERAL CONSIDERATIONS

Since the 1990s, the scope of activity in the CCL and electrophysiology laboratory has greatly increased and has diverged considerably from the simple diagnosis and evaluation of valvular heart disease, CAD, and CHD.^{396,397,522,525} Procedures and interventions in these settings are more complex and often involve acutely ill patients.³⁹⁷ Cardiac anesthesiologists may find themselves in a relatively isolated environment with bulky equipment, subdued lighting, and limited access to the patient.^{521,524} Assistance from surgical colleagues, if required, may not be readily available, nor may the services of anesthesia support personnel, the pharmacy, and the stat laboratory. Finally, for patients with moderate or severe cardiac disease who have undergone heavy sedation or general anesthesia, a suitable recovery area may be in a distant location.

Because fluoroscopy is frequently used during interventional procedures in the CCL and hybrid operating rooms, it can present a significant risk of radiation exposure to both patients and healthcare workers. The potential hazards of ionizing radiation include skin injuries and cellular mutation, which can lead to leukemia, bone cancer, and birth defects. The radiation safety program at the Centers for Disease Control and Prevention (CDC) has developed the concept that radiation doses should be “as low as reasonably achievable” (ALARA). Exposure to radiation can be minimized by three means: distance, time, and shielding.⁵²² The distance between the

BOX 54.22 Maximum Allowable Radiation Limits for Medical Workers

- Whole body: 5 rem/year (50 mSv/year)
- Skin: 50 rad/year (500 mGy/year)
- Lens of eye: 2 rad/year (20 mGy/year)
- Fetus (for pregnant worker): 0.5 rad (5 mGy) for total pregnancy or 0.05 rad/month (0.5 mGy/month) (estimated by abdominal badge under lead apron)
- Cumulative exposure (lifetime): 1 rem × age (10 mSv × age)

Modified from Bashore TM, Balter S, Barac A, et al. 2012 American College of Cardiology Foundation/Society for Cardiovascular Angiography and Interventions expert consensus document on cardiac catheterization laboratory standards update: a report of the American College of Cardiology Foundation Task Force on Expert Consensus documents. *J Am Coll Cardiol.* 2012;59:2221–2305.

person and the source should be maximized because the dose rate varies with the inverse square of that distance. In addition, the person’s exposure time should be minimized because the dose rate and time are directly related. Finally, personal shielding and shielding of the radiation source should be maximized.

The rad is a unit of absorbed dose, which is the energy imparted to matter by ionizing radiation per unit mass of irradiated material at the point of interest.⁵²² The rem is a unit of effective dose, which is the estimated total body dose. Healthcare personnel in a radiation environment must wear a dosimeter badge to track cumulative radiation exposure. The dosimeter should be worn on areas at highest risk for frequent exposure, such as the thyroid collar, and outside any shielding garments. Corrective action is recommended if an individual (patient or provider) receives more than 5 rem/year to the whole body (Box 54.22).⁵²² In the case of pregnant women, the total radiation exposure to the fetus throughout gestation should be no more than 0.05 rad/month or 0.5 rad total.

Identifying patients at risk for a reaction to iodinated contrast material is another consideration unique to procedures performed in the CCL or hybrid suite. A previous anaphylactoid reaction and a history of atopic conditions such as asthma are the most significant risk factors for acute hypersensitivity reactions.⁵²² Premedication regimens with histamine (H₂) blockers and steroids are recommended for the highest-risk atopic patients, particularly those with a known prior reaction. Current options include giving 50 mg oral prednisone 13 hours, 7 hours, and 1 hour before the procedure or 200 mg intravenous hydrocortisone, with or without H₂ blockers, 2 hours before the cardiac catheterization.⁵²²

In patients with renal insufficiency, radiocontrast media-induced nephropathy is a concern, and diabetic patients are at particularly high risk for acute renal failure after exposure to contrast agents.^{521,522} These effects can be minimized through careful contrast administration and limitation of the total dose. Preprocedural and postprocedural hydration with normal saline solution, sodium bicarbonate, or both is recommended.⁵²² However, in patients with severe HF or near-end-stage renal disease, one must be careful to avoid volume overload.^{521,522} Elevated serum creatinine levels are common after the administration of

radiocontrast media, and these levels should be monitored for 72 or more hours in patients at risk. Two definitions (an incremental increase >0.5 mg/dL or a rise $>25\%$ in serum creatinine level) are now accepted as indicators of contrast-induced nephropathy. Fortunately, renal dysfunction is usually transient and rarely progresses to acute renal failure.

HYBRID OPERATING ROOM

To address some of the issues regarding the technologic and procedural demands regarding surgical and imaging equipment for selected endovascular and transcatheter procedures, hybrid operating rooms have been built in many institutions. These rooms have complete dual capabilities for procedures that require fluoroscopy, open surgery, or both. Ideally, such rooms are within or adjacent to the regular surgical suite. The physical location of such hybrid rooms may represent an advance in care in that key personnel are more readily available to handle unanticipated complications and emergencies.

The types of procedures that are performed in the CCL or the hybrid operating room vary according to institutional preferences but may include (1) electrophysiology procedures, (2) percutaneous management of valvular lesions, (3) the use of occlusion or umbrella devices to close an ASD or VSD or a PDA, (4) percutaneous VADs, and (5) stenting of abdominal or thoracic aortic aneurysms.⁴⁰⁰⁻⁴⁰²

Although requirements vary depending on the nature of the procedure, sedation or anesthesia improve the efficacy and safety of many procedures.⁵²⁵ Providing stable hemodynamics for organ perfusion and preservation during anesthesia is an important goal. Some procedures can be performed with the aid of monitored anesthesia care or regional blocks, provided a certain patient comfort level can be achieved. However, during difficult and lengthy procedures, patients may have trouble lying still. In many cases, a general anesthetic regimen may be the best option. If indicated, general anesthesia with endotracheal intubation provides a controlled situation: the patient's comfort is maximized, and the airway is secured.⁵²¹ Use of a laryngeal mask or a standard mask airway is also possible, but the constant diaphragmatic movements that occur during spontaneous ventilation may interfere with fluoroscopic visualization of cardiac and vascular structures. In the absence of significant complications or comorbid conditions, the patient may be allowed to emerge from anesthesia before being transferred to the CCL recovery area and, eventually, to a hospital floor. In more complex cases, the patient may be transferred to an ICU.

Percutaneous mitral valve repairs (e.g., correction of MR and commissurotomy for MS) share the same advantages as TAVI with respect to avoiding sternotomy, implementing CPB, and cross-clamping the aorta.³⁹⁹ These procedures are similarly performed with the patient under general anesthesia, with fluoroscopic and TEE guidance.⁴⁰²

Percutaneous closure procedures are used to close ASDs and, less commonly, VSDs, as well as PDAs and fenestrations.^{402,525} Echocardiography is used to help guide device placement and confirm a successful result. If TEE is used, general anesthesia is necessary. If ICE is used, the procedure can potentially be performed with sedation only.⁴⁰²

Large-bore peripheral or femoral venous access is needed, and a radial arterial line is often placed.

Percutaneous VADs (TandemHeart, and Impella Recover LP 2.5 and 5.0) are placed in patients undergoing high-risk coronary interventions or ablation procedures or who are in cardiogenic shock.⁴⁰² These devices can produce CO that completely replaces LV function with nonpulsatile blood flow. Hence, pulse oximetry and noninvasive blood pressure measurement may not work properly since they rely on the presence of a pulse for their mechanism of measurement. Either sedation or general anesthesia can be used, depending on the patient's hemodynamic status and ability to cooperate. Invasive monitoring of the arterial blood pressure is easily available because arterial cannulation is used during the procedure.⁴⁰² Large-bore intravenous access is desirable because a large amount of blood loss is possible. Surgical backup is necessary during these procedures. The institution of ECMO for full cardiorespiratory support is often performed in a catheterization laboratory or a hybrid operating room.

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 Complete references available online at expertconsult.com.

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SAMUEL A. IREFIN

KEY POINTS

- Cardiac arrhythmias are caused by disorders of impulse formation, disorders of impulse conduction, or both. Cardiac arrhythmias may be life-threatening because of a reduction in cardiac output and/or myocardial blood flow or precipitation of a more serious arrhythmia.
- Radiofrequency ablation is the therapy of choice for many types of cardiac arrhythmias.
- Electrophysiologic studies are used to map out normal and abnormal intracardiac structures. In this process, the mechanism of arrhythmia is delineated and ablation can be performed at the same time.
- Pacing technologies have been developed to treat heart failure resulting in increases in pulse pressure, left ventricular stroke volume, cardiac index, and wedge pressure.
- Implantable pacemakers are placed for treatment of symptomatic bradycardia with the ability to respond to changing hemodynamic demands.
- The development of implantable cardioverter-defibrillators (ICDs) to terminate ventricular tachyarrhythmias by delivering high-voltage shocks to the ventricle has revolutionized therapy for cardiac arrhythmias.
- The main purpose of ICD placement is to prevent sudden cardiac death resulting from hemodynamically unstable ventricular arrhythmias.
- An ICD can be placed for cardiac resynchronization. Cardiac resynchronization therapy improves heart failure symptoms, quality of life, exercise capacity, and electrocardiographic variables.
- Anesthetic management of patients for correction of cardiac arrhythmias depends on associated comorbid illness and the procedure that is planned.

In the United States, cardiac arrhythmias are responsible for about 1 million hospitalizations and nearly 50,000 deaths.¹ Pharmacotherapy with potentially toxic medications was used to treat cardiac arrhythmias in the past but electrophysiology has transformed the care of patients with arrhythmias from diagnostic studies to direct therapeutic interventions, as demonstrated in clinical trials. Cardiac arrhythmias are caused by disorders of impulse formation, disorders of impulse conduction, or both. Disorders of impulse formation include enhancement or depression of automaticity, parasystolic activity, and triggered activity. Disorders of conduction include decremental conduction, reentry, entry block, exit block, concealed conduction, and supernormal conduction.²

At the present time, radiofrequency catheter ablation has replaced antiarrhythmic drug therapy as the treatment of choice for many types of cardiac arrhythmias. Before the 1980s, cardiac electrophysiology was primarily used to confirm mechanisms of arrhythmias, with management mainly by pharmacologic means. As a result of shortcomings in antiarrhythmic drug therapy (including the results of randomized trials), radiofrequency ablation and implantable cardioverter-defibrillators (ICDs) were developed.^{3,4}

Historical Perspectives

The treatment of cardiac arrhythmias with device-based therapy may have begun in 1899, when Prevost and Batelli⁵ noted almost as an afterthought that direct electric shock could terminate ventricular fibrillation in dogs. Hooker and colleagues⁶ showed three decades later that the passage of electric current across the heart can initiate and terminate ventricular fibrillation. In 1947, Beck⁷ saved the first human life by the successful use of cardiac defibrillation in a 14-year-old boy who developed ventricular fibrillation during a thoracic procedure and went on to achieve full recovery. These early achievements provided the foundation for the landmark work of Mirowski and Mower⁸ which ultimately led to the development of ICDs in humans in 1980. During the past three decades, an increase has occurred in the numbers of patients with pacemakers and ICDs for the correction of cardiac arrhythmias.

Scope of Cardiac Arrhythmias

Cardiac arrhythmias are common. Some cardiac arrhythmias are life-threatening, and others are merely a nuisance.

Cardiac arrhythmias are caused by abnormalities in impulse formation or conduction that lead to slow or fast, regular or irregular heart rhythms. At the present time, it is not difficult to treat slow rhythms because available pacemakers are able to adapt slow function to the needs of the body.⁹ The situation is different, however, for patients with rapid rhythms. Rapid rhythms may originate anywhere in the heart and result from various mechanisms. These mechanisms may be focal, meaning that the abnormal impulse formation is confined to a small area, or they may be the result of an impulse running in a circuit composed of several interconnected cardiac cells. Such a circuit may be small or large, as in atrial flutter and in arrhythmias in which the normal atrioventricular conduction system and an extra connection between the atrium and the ventricle are incorporated into the circuit of the arrhythmia.¹⁰

Pharmacologic interventions originally were used to terminate and prevent rapid rhythms. However, antiarrhythmic drugs may have serious side effects and sometimes may even be responsible for the occurrence of life-threatening arrhythmias and sudden death.¹¹ As a result of these effects, techniques were developed for localizing the site of origin or pathway of an arrhythmia and then isolating or destroying the tissue that is responsible. By employing an intracardiac catheter, the site of origin or pathway of an arrhythmia can be identified and the rhythm disturbance corrected by applying radiofrequency, laser, ultrasound, microwave energy, or freezing temperatures to the tissue causing the arrhythmia.

Heart failure is a major problem in older patients. Although pharmacologic treatment of heart failure has improved, outcome generally remains poor. New pacing technologies may be used to treat selected patients with heart failure. For many years, permanent pacing has been used to treat symptomatic bradycardia, and pacing may alleviate heart failure when associated with heart block. Several studies have examined the use of conventional dual-chamber atrioventricular-right ventricular pacing for treatment of heart failure in the absence of symptomatic bradycardia or heart block.^{4,12} Biventricular pacing aims to restore synchronous cardiac contraction. When ventricular dyssynchrony is reduced, the heart is able to contract more efficiently and increase left ventricular ejection fraction and cardiac output, while working less and consuming less oxygen.¹³ In addition, reestablishment of left ventricular synchrony can

increase left ventricular filling times, decrease pulmonary capillary wedge pressure, and reduce mitral regurgitation.

Normal Cardiac Rhythm

In the normal heart, the dominant impulse arises in the sinus node with a rate of 60 to 100 beats/min (Fig. 55.1). During sleep, the rate may decrease to 30 to 50 beats/min.¹⁴ Episodes of sinus pauses up to 3 seconds, sinoatrial block, junctional rhythms, and first-degree and second-degree atrioventricular nodal block that occur quite often (especially in trained athletes) are considered to be normal variants.⁹

The impulses generated from the sinoatrial node propagate along three intraatrial conduction pathways: the anterior, middle, and posterior internodal tracts. These tracts are not discrete pathways, but groups of cells that conduct slightly faster than the atrial myocardium.¹⁵ The internodal tracts give rise to interatrial fibers. The electric impulse, whether propagated in the atrial myocardium or along the internodal tracts, converges on the atrioventricular junction. The atrioventricular node located in the atrioventricular junction ultimately receives the impulses generated from the sinoatrial node. The impulses are delayed in the atrioventricular node before they are finally distributed to the ventricular myocardium via the His-Purkinje system.

Normally, the heart rate increases with exercise to at least 85% of the age-predicted maximum of 220 minus age in years; failure to do so is termed chronotropic incompetence. Sinus arrhythmia is defined as sinus rhythm with P-to-P variations of more than 10% (Fig. 55.2). Sinus arrhythmia is due to cyclic variations in vagal tone commonly related to respiration (the rate is faster with inspiration and slower with expiration).¹⁶ Sinus arrhythmia disappears with exercise, breath-holding, and atropine, and is more likely to be seen in individuals who do not have heart disease.¹⁷

Cardiac Arrhythmias

Cardiac arrhythmia is caused by a disorder of impulse generation, impulse conduction, or a combination of both. Cardiac arrhythmia may be life-threatening because of



Fig. 55.1 Normal sinus rhythm. (Courtesy M. Kanj, MD, Cleveland Clinic, Cleveland, OH.)



Fig. 55.2 Sinus arrhythmia. (Courtesy M. Kanj, MD, Cleveland Clinic, Cleveland, OH.)

a reduction in cardiac output, reduction in myocardial blood flow, or precipitation of a more serious arrhythmia.¹⁸ Arrhythmias may be described based on (1) rate (bradycardia or tachycardia), (2) rhythm (regular or irregular), (3) origin of impulse (supraventricular, ventricular, or artificial pacemaker), (4) impulse conduction (atrioventricular, ventriculoatrial, or block), (5) ventricular rate, or (6) special phenomena (e.g., preexcitation).

Reentry is a common electrophysiologic mechanism that predisposes to most ventricular arrhythmias and to most supraventricular tachyarrhythmias. The most common mechanism of reentry is based on the model originally proposed by Erlanger and Schmitt and later modified by Wit.² This model postulates the presence of a ring or loop of cardiac tissue that is functionally separate from neighboring tissue and the presence of transient or permanent unidirectional block in a portion of the loop. Unidirectional block may be anatomic in origin (e.g., bundle branches, fibrosis, dual pathways, atrioventricular node plus accessory pathway) or functional (e.g., ischemia, drug effect).

Atrial flutter is a macro-reentrant arrhythmia identified by flutter waves, often best seen in the inferior leads at 250 to 350 beats/min (Fig. 55.3). Patients often present with a 2:1 atrioventricular conduction with a ventricular rate of 150 beats/min, although the atrioventricular conduction ratio can change abruptly. Atrial fibrillation is a narrow-complex tachyarrhythmia and is the most common in the general population (Fig. 55.4). It is associated with significant morbidity. The prevalence of atrial fibrillation in the general population increases exponentially with age, from 0.9% in individuals 40 years of age to 5.9% in individuals older than age 65 years. The most important risk factors for development of atrial fibrillation in the general population are structural heart disease, valvular heart disease,

and left ventricular hypertrophy.¹⁹ Atrial fibrillation is a significant contributor to the development of angina and stroke, with an estimated stroke risk in untreated individuals of 3% to 5%.²⁰

Ventricular tachyarrhythmia is defined as three or more consecutive ectopic beats at a rate more rapid than 100 beats/min (Fig. 55.5).²¹ Ventricular tachyarrhythmia is traditionally classified as nonsustained or sustained. Sustained ventricular tachyarrhythmia is defined as ventricular tachyarrhythmia lasting more than 30 seconds. Nonsustained ventricular tachyarrhythmia is defined as ventricular tachyarrhythmia that terminates spontaneously within 30 seconds. Sustained ventricular tachyarrhythmia also is traditionally classified as monomorphic (one site of origin) or polymorphic (two or more sites of origin).²² Monomorphic ventricular tachyarrhythmia usually results from reentry, and the site of reentry depends in part on the type of heart disease. In patients with coronary artery disease, the reentry circuit is usually located in ventricular myocardium, whereas in dilated cardiomyopathy with left bundle branch block, bundle branch reentry is common.²³ Monomorphic ventricular tachyarrhythmia may occur in individuals with an otherwise normal heart, whereas polymorphic ventricular tachyarrhythmia may occur in acquired states that produce a marked prolongation of the Q-T interval. Nonsustained ventricular tachyarrhythmia is frequently asymptomatic, but may produce palpitations, weakness, and presyncope.²²

Torsade de pointes is a French term translated as “twisting of the points.” It is a syndrome composed of polymorphic ventricular tachyarrhythmia (Fig. 55.6). It may be due to various medications or electrolyte imbalances. *Torsade de pointes* is usually paroxysmal, but is frequently symptomatic and often produces loss of consciousness. It

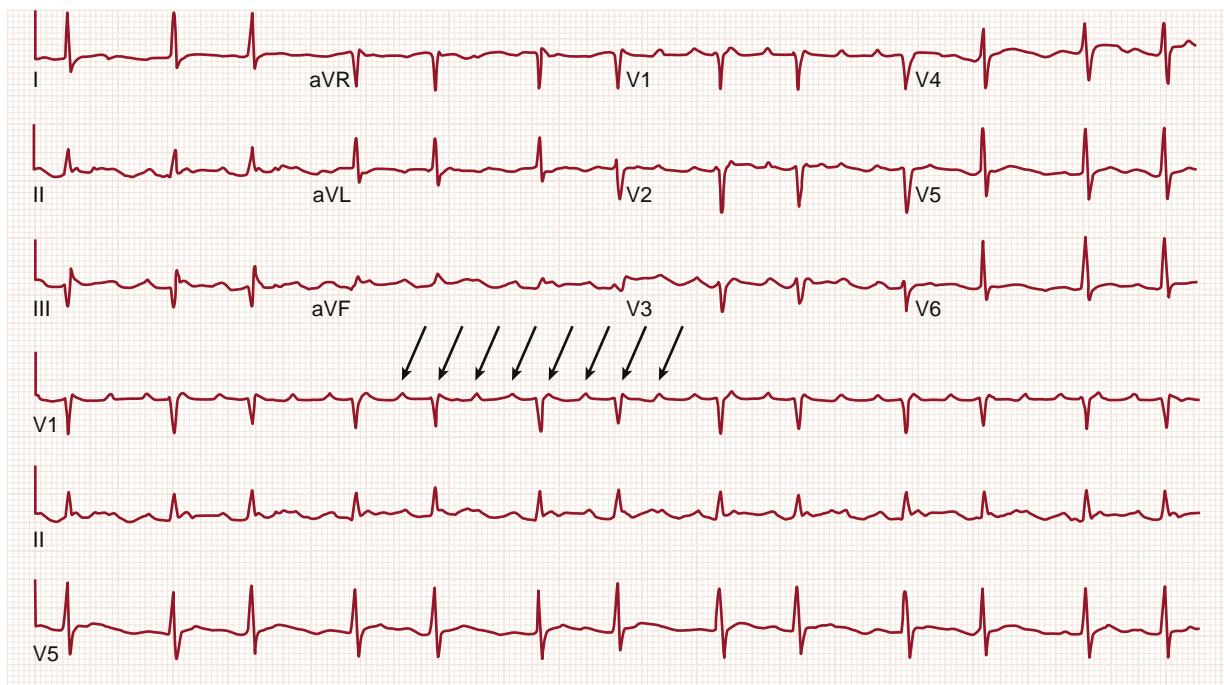


Fig. 55.3 Atrial flutter. Note the flutter wave pattern in lead V1 (arrows). (Courtesy M. Kanj, MD, Cleveland Clinic, Cleveland, OH.)

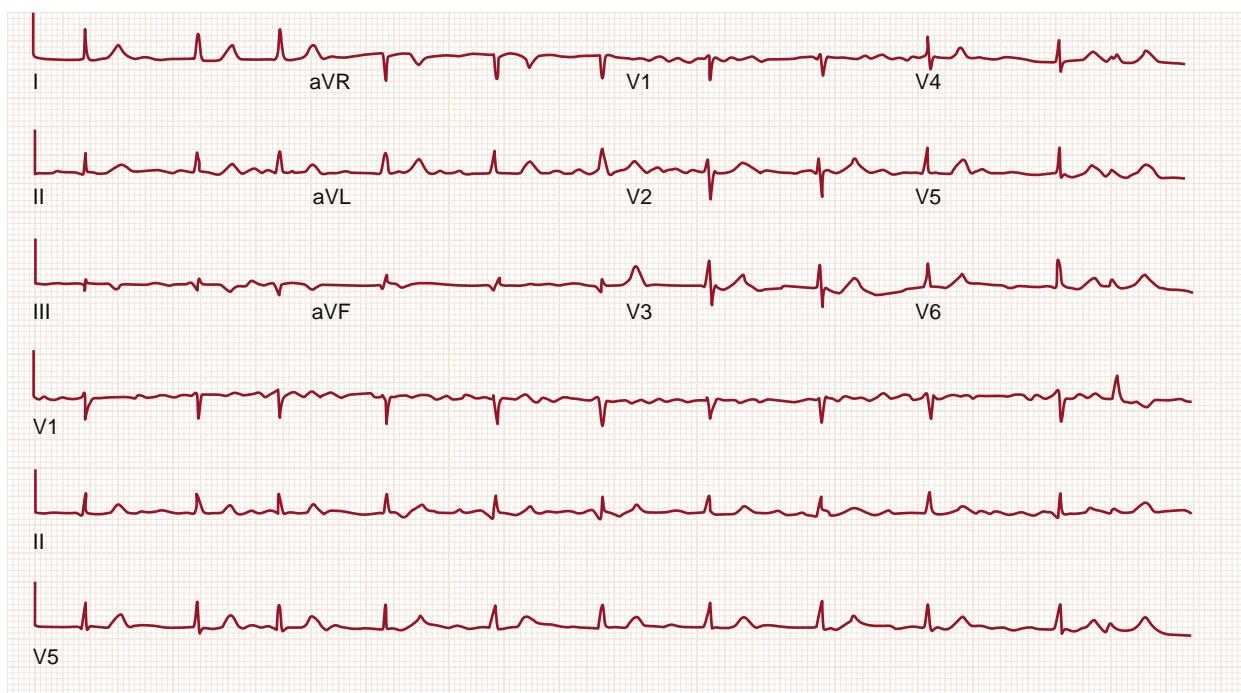


Fig. 55.4 Atrial fibrillation. (Courtesy M. Kanj, MD, Cleveland Clinic, Cleveland, OH.)



Fig. 55.5 Ventricular tachycardia. (Courtesy M. Kanj, MD, Cleveland Clinic, Cleveland, OH.)



Fig. 55.6 Torsade de pointes. (Courtesy M. Kanj, MD, Cleveland Clinic, Cleveland, OH.)

occasionally degenerates to ventricular fibrillation. Ventricular fibrillation accounts for 80% to 85% of sudden cardiac deaths.²²

Ventricular fibrillation is usually preceded by ventricular tachyarrhythmia, but also may occur as a primary arrhythmia (Fig. 55.7). More recent studies suggest that ventricular fibrillation results from multiple wavelengths that disperse randomly, using the leading circle form of reentry.²² The most common cause of ventricular fibrillation is acute myocardial infarction. It also is observed in patients with chronic ischemic heart disease, hypoxia resulting from any cause, acidosis, hypokalemia, and massive hemorrhage.

INDICATIONS FOR CORRECTION OF CARDIAC ARRHYTHMIAS

Intracardiac electrophysiologic studies can give valuable information about normal and abnormal electrophysiology of intracardiac structures (see also Chapters 36, 38, and 86). These studies are used to confirm the mechanism of an arrhythmia, to delineate its anatomic substrate, and to ablate it. The electric stability of the ventricles also can be assessed, as can the effects of an antiarrhythmic regimen.

In addition, pacing technologies have been developed to treat heart failure with promising results, leading to

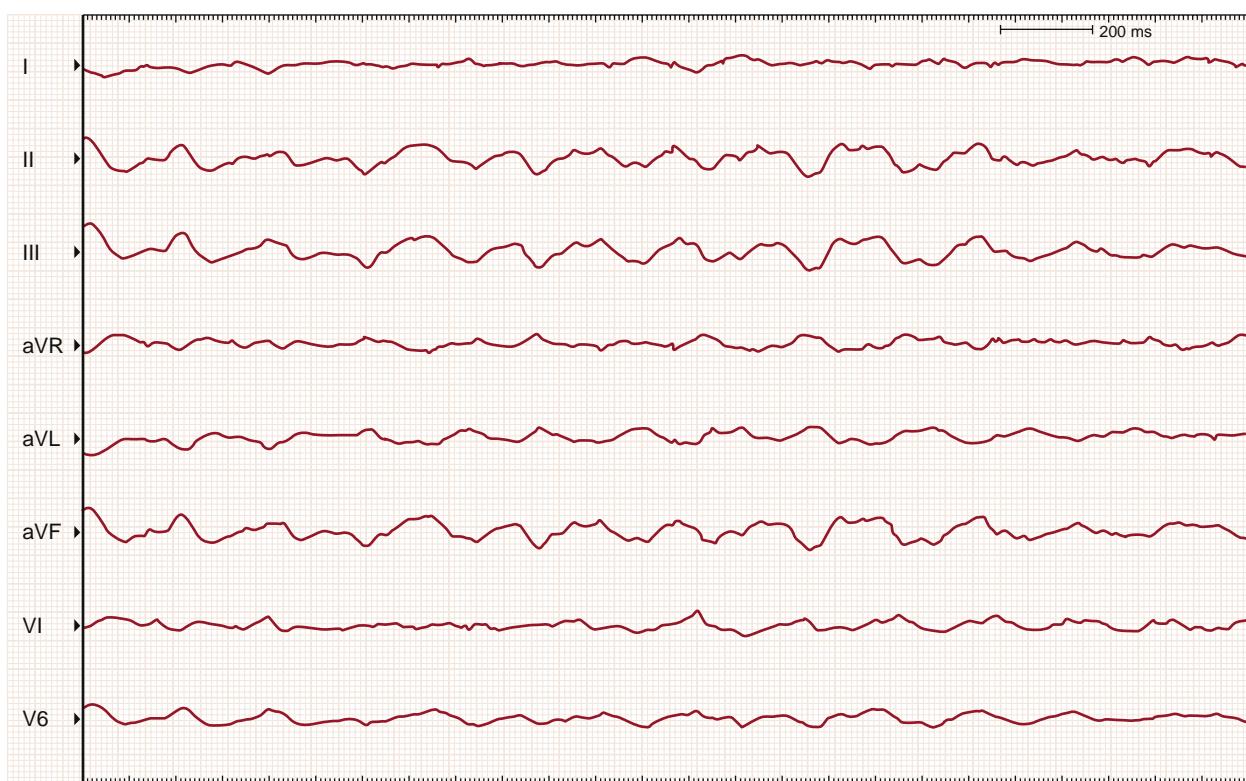


Fig. 55.7 Ventricular fibrillation. (Courtesy M. Kanj, MD, Cleveland Clinic, Cleveland, OH.)

improvement in morbidity and mortality in patients with heart failure.

Hemodynamic responses to biventricular pacing include an increase in the rate of elevation of left ventricular pressure and increases in pulse pressure, left ventricular stroke work, cardiac index, and wedge pressure.²³ Cardiac resynchronization therapy improves ventricular function without increasing myocardial energy consumption, in contrast to the effect of inotropic agents, such as dobutamine.¹³ In addition, cardiac resynchronization therapy may reverse left ventricular remodeling over time.²⁴

Permanent Pacing

Indications for pacemaker therapy have increased in recent years and now include the treatment of bradyarrhythmias and heart failure according to the American College of Cardiology and American Heart Association guidelines.²⁵ These guidelines discuss indications for pacing in patients with sinus node dysfunction, acquired atrioventricular block, chronic bifascicular and trifascicular block, hypersensitive carotid sinus, and neurally mediated syndromes. The guidelines direct the treating physician in selecting patients who would benefit from device therapy.

A Swedish team led by Sennings and Elmquist implanted the first pacemaker in 1958.²⁶ A thoracotomy was required, and pacing was done through electrodes sutured to the epicardium. In these early systems, significant problems with changes in pacing threshold, lead infection, and lead breakage were common. Transvenous lead implantation subsequently developed by Furman and colleagues²⁷ would

resolve many of these issues. In 1958, Furman successfully paced an elderly patient with a catheter electrode inserted transvenously. Other investigators took on the challenge of solving various technical problems, such as device miniaturization; longer-life batteries; and stable, reliable lead material.²⁸ As the indication for implantation expanded from atrioventricular conduction disturbances to management of sinus node dysfunction, the need for implantable pacemakers grew in proportion.²⁸ Technology evolved rapidly with the development of lithium-iodide batteries that had greater longevity. Electronic advances then led to major miniaturization using integrated circuits as opposed to discrete components. Lead materials used in today's pacemaker rely on silicone and polyurethane, which are more biocompatible and reliable than earlier materials. With these technical refinements, present-day pacemakers are small and can pace reliably for 8 to 10 years before generator replacement is needed. The primary functional challenge for contemporary pacemakers is to maintain the heart rate based on circulatory needs, pacing in a manner that mimics the natural physiology of excitation and conduction. In a healthy heart, the sinus node is modulated by the autonomic nervous system, and its rate is determined by a multiplicity of factors, such as physical activity, emotion, and blood pressure. Not only the rate, but also the activation sequence and atrioventricular conduction time vary with demand; these requirements also must be considered. Rate is controlled by pacemaker discharge, and the excitation and conduction sequence depends on the placement of pacing electrodes. Approximately 120,000 pacemakers are implanted each year in the United States. Indication for implantation for most of these cases is sick sinus syndrome.

Other indications include atrioventricular block, carotid sinus hypersensitivity, malignant vasodepressor syndrome, and hypertrophic cardiomyopathy.²⁹ The primary purpose of implantable pacemakers is to treat symptomatic bradycardia. With the extraordinary developments that have occurred in pacemaker therapy for the traditional indication—bradycardia—new uses are now beginning to be explored. Pacemakers have progressed from large, fixed-rate, single-chamber devices to multiprogrammable, multichamber devices with the ability to respond to changing hemodynamic demands. As technology advances, other possible uses are likely to be conceived.

Resynchronization Therapy

Cardiac resynchronization is a pacing therapy aimed at improving coordination of atria and both ventricles. This therapy has been demonstrated to be effective for patients with heart failure who have conduction delay and left ventricular systolic dysfunction.³⁰ In addition, this therapy can reduce hospital readmission rates and improve quality of life in patients with heart failure.³¹

Cardiac resynchronization therapy improves heart failure symptoms, quality of life, exercise capacity, hospitalization, and echocardiographic variables.³² It is indicated in patients with drug-refractory, symptomatic New York Heart Association functional class III and IV heart failure of either ischemic or nonischemic origin.³¹ In addition, these patients are protected from associated risk for sudden cardiac death when combined with an ICD system.³⁰

The development of an automatic internal defibrillator, or ICD, began in the 1960s. External cardiac defibrillation was increasingly being used in coronary care units for the treatment of ventricular fibrillation and sudden cardiac death. Although the idea of automatic external defibrillation had been discussed initially by Zycoto, Mirowski, and colleagues³³ were the first to champion and begin practical development of an automatic internal device. In 1969, Mirowski and Mower developed the prototype of today's automatic internal defibrillator.³⁴

The primary goal of all defibrillators is to terminate ventricular tachyarrhythmias by delivering high-voltage shocks to the ventricle. As with implantable pacemakers, defibrillating devices need to be small and reliable and have adequate longevity. ICDs have evolved not only to perform this function but also to take on additional tasks, such as antitachycardia pacing of the ventricle, dual-chamber pacing, and termination of atrial tachyarrhythmias.

A key difference between pacing and defibrillation of the heart is that for pacing only a very small mass of myocardium needs to be stimulated, whereas for defibrillation, most, if not all, of the myocardium must be stimulated. Because the myocardium is easily excitable throughout diastole, a small wave of depolarization during pacing can readily propagate throughout the whole heart. In contrast, during ventricular fibrillation, multiple reentrant wavefronts usually occur that are continuously changing in location and size and must be quelled. To defibrillate successfully, most of these wavefronts must be interrupted simultaneously; to achieve this, it is necessary to capture most of the tissue that is in a state of relative refractoriness.³⁵ One unique property

of defibrillation success is that it is probabilistic.³⁶ The same energy that can defibrillate the heart on one occasion may be unsuccessful at another time.

The main purpose of ICD placement is to prevent death from hemodynamically unstable ventricular tachyarrhythmias. Although advances in technology have made these devices much more flexible in terms of arrhythmia detection and electric therapy options, their main purpose is to reduce sudden cardiac death, which claims approximately 300,000 lives in the United States annually. Secondary prevention of sudden cardiac death in patients who have survived cardiac arrest is another major indication for ICD placement. In such patients and especially in patients of this group for whom no reversible or curable cause can be found, ICD implantation has been repeatedly documented to provide a major mortality benefit.³⁷ Interest in managing atrial tachyarrhythmias also has grown significantly in recent years. It is now recognized that approximately 30% of patients with ventricular tachyarrhythmia also have atrial tachyarrhythmias.³⁸ Such atrially initiated tachyarrhythmias can worsen patient symptoms, can result in inappropriate ventricular shocks, and may be responsible for initiating ventricular tachyarrhythmias that can exacerbate other pathologic processes, such as heart failure. New strategies for treatment and prevention of atrial tachyarrhythmias are incorporated into devices that are capable of defibrillation and anti-tachycardia pacing in the atrium and ventricle, in addition to combined dual-chamber pacing.³⁹

The relative ease of ICD implantation and longevity of current defibrillators have made them a valuable tool in primary prevention. Patients no longer must survive cardiac arrest to justify the risk in ICD implantation.

PREOPERATIVE EVALUATION

Most patients who require pacemaker or ICD placement have significant cardiovascular disease. In addition, correction of cardiac arrhythmia may require radiofrequency catheter ablation. Radiofrequency catheter ablation has proved highly effective in the treatment of atrioventricular nodal reentrant and accessory pathway tachycardias. Indications for pacemaker and ICD placement continue to evolve as the utility of these devices continues to increase. Although most pacemaker placement is done with local anesthetic infiltration, ICD placement may require monitored anesthesia care or in some cases general anesthesia. The modern ICD unit is capable of delivering the full spectrum of therapy for ventricular tachyarrhythmias and for bradycardia therapy with dual-chamber pacing or sensing, rate modulation, and mode-switching features.

As mentioned earlier, ICD placement has two common indications. One is continued ventricular tachyarrhythmias despite adequate drug therapy, and the other is history of sudden cardiac arrest that is not associated with myocardial infarction. Preoperative evaluation processes necessary for placement of an ICD should be complete by the time the decision is made to place the device. These patients need a thorough preoperative evaluation. This evaluation includes electrophysiologic testing to determine the inducibility of ventricular tachycardia and electrophysiologically guided drug therapy. Preoperative pulmonary

function tests may be necessary in patients on amiodarone to evaluate possible toxicity of this drug, which can result in chronic obstructive pulmonary disease or interstitial lung disease. In some instances, the underlying pathophysiology of malignant ventricular arrhythmias is related to ischemic or idiopathic cardiomyopathy.⁴⁰ These patients often present with poor left ventricular function and more frequent incidence of congestive heart failure. Patients with a history of congestive heart failure should be in optimal condition before surgery.

Generally, all patients who present for correction of cardiac arrhythmia require preoperative evaluations including electrocardiogram (ECG), chest radiograph, hemoglobin value, and electrolyte levels. Patients should receive nothing by mouth (NPO) for at least 8 hours before the procedure. In addition, patients who require device and lead extractions because of malfunction or infection may require blood product transfusions during the procedure. Consequently, type and crossmatch of blood products is frequently necessary for these procedures.

ANESTHETIC CONSIDERATIONS

Pacemakers

Permanent implantable pacemakers have been the standard modality of treatment for patients with all types of bradyarrhythmias. A significant number of these patients present with sick sinus syndrome and are older. Consequently, devices are placed under general anesthesia in these patients. As a result of more recent advances in pacemaker technology, these devices now can be placed as a therapeutic modality to alter hemodynamic states. Surgeons used to be primarily responsible for device insertion. Now the task falls under the services of cardiologists. Device placement is commonly performed in the cardiac catheterization suite under local anesthesia on an outpatient basis. Patients at high risk who have complicated disease now present for pacemaker insertion, however, in addition to those with indications more recently identified by the American College of Cardiology and American Heart Association for these devices. In light of these increased indications, the expertise of anesthesiologists is needed for monitoring and perioperative care of these patients.

Monitored Anesthesia Care

Currently, most pacemaker insertions are performed by cardiologists. Most of these cases are performed under local anesthesia with sedation. Depending on the level of training, administration of sedatives and analgesics can be provided by nurses.

In instances that require deeper sedation for a patient's comfort or for critically ill patients with hemodynamic instability, monitored anesthesia care by an anesthesiologist may be required. Adequate monitoring and resuscitation equipment are required in such situations. The goal of monitored anesthesia care is to provide analgesia, sedation, and anxiolysis, while ensuring rapid recovery with minimal or no side effects. Any sedative-hypnotic medication may be used during monitored anesthesia care with a wide variety of delivery systems.⁴¹ Subanesthetic concentrations of inhaled agents also have been used to supplement local anesthetics. Newer drugs, such as centrally mediated

α_2 -agonists, have been shown to produce anxiolysis, sedation, and reduced requirements for supplemental analgesic medications during monitored anesthesia care.

General Anesthesia

Patients requiring pacemaker placement rarely require general anesthesia for placement. If general anesthesia is required, it should be directed toward underlying cardiac pathophysiology, indications, complications, and hemodynamic goals. Immediate access to life-support equipment, such as a cardiac defibrillator and a transcutaneous pacemaker, is necessary if the device is being placed under general anesthesia.

Implantable Cardioverter-Defibrillator

Since the 1980s, indications for use and implantation of ICDs have steadily increased. Over the past two decades, ICDs have undergone a significant evolution. In the 1970s and 1980s, ICD placement usually required thoracotomy for placement of epicardial patches.

PREOPERATIVE EVALUATION

As mentioned earlier, common indications for ICD implantation include continued ventricular tachyarrhythmias unresponsive to adequate pharmacotherapy and history of sudden cardiac arrest unassociated with myocardial infarction. Newer indications include patients with various forms of the congenital long QT syndrome.⁴² Patients with long QT syndrome who have survived an episode of cardiac arrest or documented polymorphic ventricular tachyarrhythmia, especially if on pharmacotherapy at the time, are increasingly being evaluated as ICD candidates. In addition, patients with hypertrophic cardiomyopathies and without a history of sudden death are usually evaluated for ICD placement.⁴³ In these patients, sustained ventricular arrhythmias, nonexertional syncope, or a strong family history of sudden death with early age of presentation strongly indicates ICD implantation.

In all instances, the evaluation that is necessary for ICD implantation is completed by the time the decision is made to place the device. Electrophysiologic studies may have been done to determine the forms of arrhythmias present. When the pathophysiology of ventricular arrhythmias is related to idiopathic or ischemic cardiomyopathy,⁴⁴ these patients may present with poor left ventricular function and a high incidence of congestive heart failure. Consequently, they should be optimized as much as possible preoperatively.

ANESTHETIC CONSIDERATIONS

In the 1980s, ICD implantation was done with epicardial leads via thoracotomy under general anesthesia with one-lung ventilation. The technologic development of implantable ICDs with transvenous lead systems has simplified their implantation. Consequently, it was reasoned that ICDs can be placed under deep sedation with little or no intervention by the anesthesiologist analogous to what is needed for pacemaker placement.⁴⁵ Placement of an ICD under

general anesthesia may be safer and more comfortable for the patient, however. Patients who present for ICD placement are often critically ill with cardiopulmonary comorbidity. These patients often present with ejection fractions of less than 30% and require vasopressors to support hemodynamics during the procedure. In addition, some form of general anesthesia is necessary for intraoperative testing of defibrillating thresholds.

Monitored Anesthesia Care

Small, new-generation devices and transvenous lead systems lend themselves to the use of local anesthesia and intravenous sedation for ICD implantation. Midazolam and fentanyl are usually the drugs of choice when an ICD is placed under monitored anesthesia care. Monitoring includes pulse oximetry, five-lead ECG, and noninvasive blood pressure. Depth of anesthesia is monitored clinically. One of the major aspects of ICD placement is testing the device. Testing the device may require deep sedation or general anesthesia because the shocks associated with this procedure can be very painful. The presence of an anesthesiology team may be necessary for ICD placement under monitored anesthesia care.

General Anesthesia

Most patients who present for ICD placement typically have comorbidities such as ventricular tachycardia, congestive heart failure with ejection fraction less than 30%, coronary artery disease, pulmonary hypertension, chronic renal insufficiency, or valvular heart disease. These patients may be unable to lie flat for the prolonged period necessary for placement of the ICD. In addition, they may require close hemodynamic monitoring during testing of the device. General anesthesia should be considered in these patients. When general anesthesia is chosen, in addition to standard monitoring, an arterial line may be added. External cardioverter-defibrillator pads are required for all ICD placements. These are employed in cases in which an implanted defibrillator fails. General anesthesia also may be requested for anxious and extremely nervous patients. Because pacemakers and ICDs are placed percutaneously, anesthesiologists must be vigilant for possible complications, such as myocardial infarction, stroke, possible cardiac injury (perforation or tamponade), and pneumothorax from subclavian vascular access.

EXTRACTION OF DEVICES

As a result of continued growth and expanding indications for pacemakers and ICD placement, leads may require extraction because of mechanical dysfunction, the need to upgrade to more complex devices, or local or systemic infection. Lead extractions are probably one of the most challenging procedures that a cardiac electrophysiologist faces today.

Indications for lead extractions can be divided into two categories—patient-related and lead-related. Patient-related indications include infection, ineffective therapy (high defibrillation threshold), perforation, migration, embolization, induction of arrhythmias, venous thrombosis, unrelenting pain, device interactions, and device upgrades.⁴⁶ Lead-related indications include lead recalls,

lead failure, and lead interactions.⁴⁷ Lead extraction is performed via powered sheaths through which energy is delivered to the tip in the form of excimer laser light or electrocautery. These systems burn through scar tissue adherent to the wall of the lead throughout its course. The potential for life-threatening complications, such as lead fracture, venous or myocardial rupture, and tamponade, makes general anesthesia with invasive monitors a prudent choice for lead extractions. There is also a small chance of needing emergent cardiac surgery with lead extraction and therefore the team must be vigilant for signs of cardiovascular decompensation.

POSTOPERATIVE CARE

Postoperative care of patients with pacemaker or ICD implantation depends on various factors surrounding the implantation of the device. As mentioned earlier, most of these patients are quite ill with significant comorbidities. It is not unusual for patients to have congestive heart failure with an ejection fraction less than 30% as a result of poor left ventricular function. Consequently, it is imperative to have these patients monitored in the postanesthesia care unit, especially if the device is placed or extracted under general anesthesia. The spectrum of recovery sites after these procedures may vary from postprocedure units to a coronary intensive care unit. Most of these procedures are done on an outpatient basis, and anesthesia is tailored to ensure rapid recovery after implantation.

Correction of Cardiac Arrhythmias With Ablation Therapy

Catheter ablation is a safe and curative option for most cardiac arrhythmias, with 85% to 98% cure rates among the arrhythmias treated most frequently.⁴⁸ The rate of major complications is less than 3%.⁴⁸ Cardiac ablation therapy involves the delivery of energy through a catheter that is usually placed in the endocardial position in the heart, destroying myocardial tissue that is responsible for the tachyarrhythmia (Fig. 55.8). Multiple electrodes are inserted to locate the arrhythmia and ablate it. Usually the diagnostic portions of the ablation study are done during the same procedure.⁴⁹ The efficacy of catheter ablation depends on accurate identification of the site of origin of the arrhythmia. When the site is identified, the electrode catheter is positioned in direct contact with the site of the arrhythmia, and radiofrequency energy is delivered through the catheter to destroy it.

The current generated by radiofrequency is alternating current and is delivered at cycle lengths of 300 to 750 kHz when used for catheter ablation.⁵⁰ It causes resistive heating of the tissue in contact with the electrode. The degree of tissue heating is inversely proportional to the radius to the fourth power.⁵¹ Consequently, the lesions created by radiofrequency energy are small. Although electric injury may be a contributing factor, the primary mechanism of tissue destruction by radiofrequency current is thermal injury. Acute lesions created by a radiofrequency current consist of a central zone of coagulation necrosis surrounded by a zone of hemorrhage and inflammation.⁵² Cardiac arrhythmias

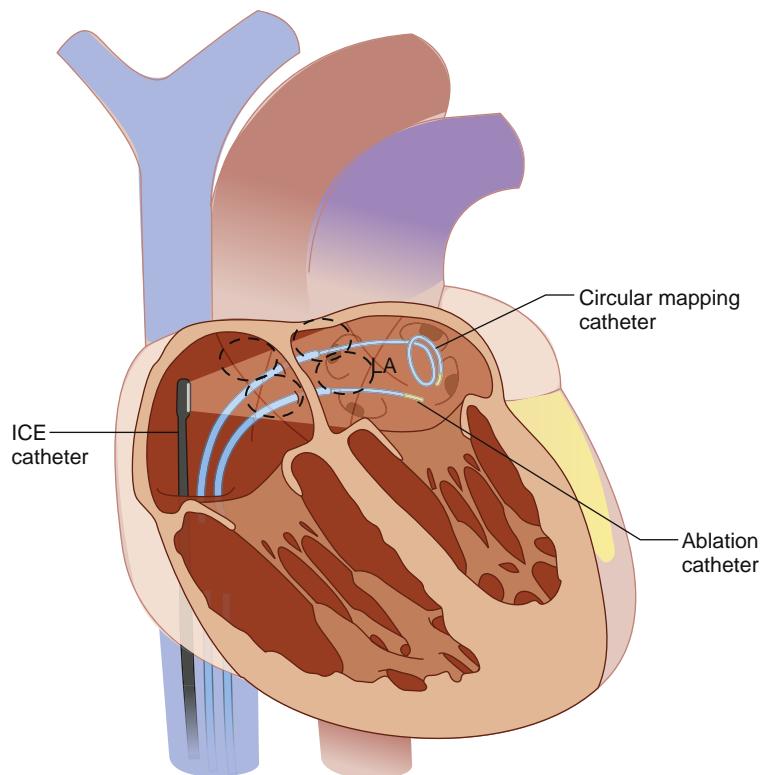


Fig. 55.8 Catheter placement during ablation procedures. Mapping and ablation catheters were placed under the guidance of intracardiac echocardiography. ICE, Intracardiac echocardiography LA, Left atrium. (Courtesy O. Wazni, MD, Cleveland Clinic, Cleveland, OH.)

that can be treated with radiofrequency ablation include paroxysmal supraventricular tachycardia, Wolff-Parkinson-White syndrome, atrial flutter, atrial fibrillation, and idiopathic ventricular tachycardia. Most cardiac arrhythmias treated with radiofrequency ablation are not life-threatening but have a significant impact on a patient's quality of life.⁵³ Advantages of radiofrequency ablation of cardiac arrhythmias include relief of symptoms, improvement in functional capacity and quality of life, and elimination of the need for lifelong antiarrhythmic drug therapy. The principal disadvantage is the risk for complications, which varies depending on the type of ablation procedure and skill of the operator.

ANESTHETIC CONSIDERATIONS

Catheter ablation was introduced into clinical practice in 1982. Initially, ablation was performed with direct electric shocks.⁵⁴ As a result of several advantages over direct current, radiofrequency ablation has replaced direct current ablation. These advantages include the absence of skeletal and cardiac muscle stimulation, minimal discomfort during delivery of energy, the possibility of performing the procedure in conscious patients, and the discrete nature of resulting lesions.⁵⁵

Until recently, most cardiac ablation therapy for correction of arrhythmias could be performed under moderate sedation or monitored anesthesia care. In some of these cases, deep sedation may have been required as the case progressed. General anesthesia is now being employed routinely in most cases of cardiac ablation therapy due to patient anxiety and the extended period required for these

procedures. General anesthesia may be implemented with standard American Society of Anesthesiologists monitors with adequate vascular access. Catheter ablation is the first-choice treatment for most cardiac arrhythmias. It is a safe treatment and is usually effective as a single procedure. Because it is curative in many patients, it is offered to all patients who would otherwise be committed to long-term drug therapy.

Future Trends

Correction of cardiac tachyarrhythmias has improved dramatically in the past two decades. Emphasis has shifted from pharmacologic therapy to nonpharmacologic therapy of tachyarrhythmias; this has led to a significant increase in the number of radiofrequency catheter ablations and defibrillator implantations. These developments were triggered by technologic advances that showed superiority of these procedures over the use of antiarrhythmic drugs.⁵⁵ As a result, treatment of supraventricular tachycardias and tachycardias involving accessory atrioventricular pathways will probably remain the domain of catheter ablation. The cure rate of patients treated with catheter ablation is very high. In addition, treatment of life-threatening ventricular tachyarrhythmia will remain in the domain of ICDs for the foreseeable future. The role of ICD therapy has been clearly defined with respect to prolongation of life and has been expanded to include primary prophylaxis of sudden death in high-risk populations.⁵⁶

The demand for perioperative care in the electrophysiology suite, especially for those undergoing ablation

procedures, has resurrected the use of high-frequency jet ventilation (HFJV) in recent years.⁵⁷ Although the use of HFJV in the electrophysiology suite is relatively new, studies have demonstrated improved outcomes and decreases in procedure time.⁵⁸ HFJV has been demonstrated to provide a more stable environment, especially in the posterior wall of the atrium. As a consequence, in the future HFJV use will increase and provide an attractive alternative to the conventional mode of ventilation during catheter ablation.^{20,59} The anesthetic implications of this modality in the electrophysiology suite has recently been reviewed.⁶⁰

As a result of these developments, the presence of an anesthesiology team will continue to increase in cardiology suites. Patients who are being cared for in these areas are sicker with significant comorbidities. The role of conscious sedation will continue to diminish in the performance of these procedures. These patients will require full monitoring and care under the direction of an anesthesiologist.

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AHMED SHALABI and JOYCE CHANG

KEY POINTS

- Perioperative management of patients undergoing vascular surgery requires an understanding of the underlying pathophysiology of the specific vascular lesion.
- Major vascular surgery is particularly challenging to the anesthesiologist because these are high-risk operations in a patient population with a high prevalence of either overt or occult coronary artery disease, which is the leading cause of perioperative and long-term mortality after vascular surgery.
- Accurate clinical assessment of the pretest probability of significant coronary artery disease is necessary for prudent use and rational interpretation of preoperative cardiac testing.
- Guidelines on perioperative cardiovascular evaluation and care suggest that coronary intervention is rarely necessary to simply decrease the risk for surgery unless such intervention is indicated, irrespective of the preoperative context. Prophylactic coronary revascularization has not been shown to reduce perioperative or long-term morbidity after major vascular surgery. Medical therapy is the cornerstone for the management of coronary artery disease.
- Patients should take their usual cardiovascular medications throughout the perioperative period. Antiplatelet therapy requires special consideration and must be individualized to each patient.
- Prevention and treatment of perioperative myocardial ischemia require careful control of the determinants of myocardial oxygen supply and demand. ST-segment monitoring, particularly with computerized ST-segment analysis, should be used to detect myocardial ischemia during the perioperative period.
- Initiation of perioperative β -adrenergic blocker therapy has potential benefits and risks.
- All patients who require statin therapy on an ongoing basis should also receive statins in the perioperative period.
- The clinical usefulness of any intraoperative monitoring technique ultimately depends on patient selection, accurate interpretation of data, and appropriate therapeutic intervention.
- Maintenance of vital organ perfusion and function by the provision of stable perioperative hemodynamics is more important to overall outcome after aortic surgery than is the choice of anesthetic drug or technique.
- The pathophysiology of aortic cross-clamping and unclamping is complex and depends on many factors, including the level of the cross-clamp, the extent of coronary artery disease and myocardial dysfunction, intravascular blood volume and distribution, activation of the sympathetic nervous system, and the anesthetic drugs and techniques.
- The degree of preoperative renal insufficiency is the strongest predictor of postoperative renal dysfunction.
- Endovascular aortic surgery has become an established, less invasive alternative to conventional open aortic repair. Endoleak, or the inability to obtain or maintain complete exclusion of the aneurysm sac from arterial blood flow, is a complication specific to endovascular aortic repair.
- The primary clinical utility of cerebral monitoring during carotid endarterectomy is to identify patients in need of carotid artery shunting; second, such monitoring is used to identify patients who may benefit from an increase in arterial blood pressure or change in surgical technique.
- Postoperative hypothermia is associated with many undesirable physiologic effects and may contribute to adverse cardiac outcome.

Preoperative Evaluation

COEXISTING DISEASE

Patients undergoing vascular surgery have a high incidence of coexisting disease, including diabetes mellitus, hypertension, renal impairment, and pulmonary disease, all of which

should be assessed and, if possible, optimized before surgery. Because of the systemic nature of atherosclerotic disease, patients with vascular disease frequently have arterial disease affecting multiple vascular territories. Coronary artery disease (CAD) is the leading cause of perioperative mortality at the time of vascular surgery, and long-term survival after vascular procedures is significantly limited by the frequent

occurrence of morbid cardiac events.¹ Less than 10% of patients who undergo vascular surgery have normal coronary arteries, and more than 50% have advanced or severe CAD. Unrecognized myocardial infarction (MI) (determined by wall motion abnormalities at rest in the absence of a history of MI) and silent myocardial ischemia (determined by stress-induced wall motion abnormalities in the absence of angina) often occur in vascular surgery patients (23% and 28%, respectively) and are associated with increased long-term mortality and adverse cardiac events.² Left ventricular systolic dysfunction (LVSD) is five times more common in patients with vascular disease than in matched controls.³ It is not clear whether any specific category of vascular disease is associated with a greater likelihood of coexisting CAD. Some investigators have shown a similar incidence and severity of CAD in patients with aortic, lower extremity, and carotid disease. Others have shown that patients with lower extremity vascular disease are more likely to have significant CAD and to experience perioperative morbidity. Medical therapy is the cornerstone of the management of CAD.

PERIOPERATIVE AND LONG-TERM CARDIAC OUTCOMES

Preoperatively, the potential for MI and death in patients undergoing vascular surgery must be considered (Table 56.1). Nonfatal and fatal MIs are the most important and specific outcomes that determine perioperative cardiac morbidity. When multiple recent studies are pooled, the overall prevalence of perioperative MI and death is 4.9% and 2.4%, respectively. When outcomes are assessed over the long term (2 to 5 years), the prevalence of MI and death is 8.9% and 11.2%, respectively. This perioperative and long-term morbidity and mortality persist despite aggressive medical and surgical therapy.⁴

GUIDELINE-BASED APPROACH

A guideline-based approach to health care is relatively new and originated primarily in the United States. The American College of Cardiology (ACC) Foundation and the American Heart Association (AHA) jointly produced guidelines in the area of cardiovascular disease for more than 2 decades. The ACC/AHA Task Force on Practice Guidelines published "Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery" in 1996. This evidence-based approach to perioperative evaluation and management was updated in 2002, 2007,¹⁸ 2009,¹⁹ and 2014.²⁰ A stepwise approach (simplified from the 2007 guidelines) to perioperative cardiac evaluation and care for noncardiac surgery is provided in Chapter 31. The authors emphasize that the purpose of the preoperative evaluation is not to give medical clearance but rather to perform an evaluation of the patient's current medical status; make recommendations concerning the evaluation, management, and risk for cardiac problems; and provide a clinical risk profile that the patient and caregivers can use in making treatment decisions that may influence perioperative and longer-term cardiac outcomes. The overriding theme of the perioperative guidelines is that intervention is rarely necessary to simply lower the risk associated with surgery unless such intervention is indicated, irrespective of the preoperative context.

TABLE 56.1 Rates of Myocardial Infarction and Death in Patients Undergoing Vascular Surgery

Study	MI (%)	Death (%)	Comments
SHORT-TERM FOLLOW-UP (IN HOSPITAL OR 30-DAY)			
Ouyang et al. ⁵	8	0	Small study
Raby et al. ⁶	2.3	0.06	Aortic, lower extremity, carotid
Mangano et al. ⁷	4.1	2.3	Vascular patient only reported
Bode et al. ⁸	4.5	3.1	All lower extremity
Christopherson et al. ⁹	4.0	2.0	All lower extremity
Mangano et al. ⁷	5.0	0	Vascular patient only reported
Fleisher et al. ¹⁰	6.0	3.0	Vascular patient only reported
Pasternack et al. ¹¹	4.5	1.0	Aortic, lower extremity, carotid
Krupski et al. ¹²	2.1	2.9	Aortic, lower extremity
Baron et al. ¹³	5.9	4.0	All aortic
Norris et al. ¹⁴	3.3	5.4	All aortic
Fleron et al. ¹⁵	5.5	4.1	All aortic
McFalls et al. ⁴	8.4	3.2	Aortic, lower extremity
Average	4.9	2.4	
LONG-TERM FOLLOW-UP (IN HOSPITAL AND AFTER DISCHARGE)			
Raby et al. ⁶	7.4	5.1	20-mo follow-up
Mangano et al. ⁷	4.7	3.5	15-mo follow-up
Mangano et al. ¹⁶	19.4	13.5	24-mo follow-up
Hertzer et al. ¹⁷	12		60-mo follow-up
Krupski et al. ¹²	3.9	11.2	24-mo follow-up
McFalls et al. ⁴	22		30-mo follow-up
Average	8.9	11.2	

MI, Myocardial infarction.

Thus, preoperative testing should not be performed unless it is likely to influence patient care. The particular challenge that the vascular surgery patient presents is emphasized throughout the document. Aspects of the updated guidelines and their evidence-based approach will be discussed throughout this chapter.

CARDIAC RISK ASSESSMENT

The preoperative cardiac assessment presents an opportunity to initiate and optimize pharmacologic management, perform appropriate diagnostic and therapeutic interventions, and adjust overall care to decrease not only perioperative risk but also long-term risks from cardiovascular events. The challenge for clinicians is to accurately assess risk for cardiac morbidity while maintaining a cost-effective, clinically relevant, and evidence-based strategy. The ACC/AHA stepwise approach considers vascular surgery distinct from other noncardiac surgical procedures and is reviewed in detail in Chapter 31. Only issues specific to vascular surgery are reviewed in this chapter.

After assessment of cardiac risk, the additional challenge exists of modifying perioperative management to reduce

risk by adjusting or adding cardiac medications (e.g., β -adrenergic blocker), direct coronary intervention (e.g., percutaneous coronary intervention [PCI] or coronary artery bypass grafting [CABG]), modifying or intensifying perioperative management (e.g., invasive hemodynamic monitoring), or changing preoperative plans (e.g., performing endovascular aneurysm repair [EVAR] rather than open aortic repair). Coordination is essential among surgeons, anesthesiologists, and cardiologists, each of whom may have different criteria for risk assessment and different objectives for risk modification.

Clinical Risk Indices

Assessing cardiac risk in patients before vascular surgery is a controversial and difficult task. Although risk indices are a cost-effective screening method for determining which patients may require further cardiac evaluation (i.e., additional risk stratification with noninvasive technologies), the high pretest probability of CAD in vascular surgery patients makes the risk index somewhat less useful. Vascular surgery-specific indices have been recently developed to optimize the prediction of perioperative mortality²¹ and cardiac morbidity²² in patients undergoing elective and urgent vascular surgery. Risk indices do not provide specific risk prediction for individuals, but rather place patients in general risk categories, most commonly designated as low (cardiac risk generally <1%), intermediate (cardiac risk of 1% to 5%), or high (cardiac risk often >5%). Clinical risk variables identified by logistic regression in vascular surgery cohorts can be used along with noninvasive cardiac testing to optimize preoperative assessment of cardiac risk before vascular surgery. From the registry of the Coronary Artery Revascularization Prophylaxis (CARP) trial, the absence of multiple preoperative cardiac risk variables identifies patients with the best long-term survival after elective vascular surgery.²³

Noninvasive Diagnostic Cardiac Testing

Accurate clinical assessment of the pretest probability of significant CAD is extremely important. In general, noninvasive cardiac testing before vascular surgery is best directed at patients considered to be at intermediate clinical risk. Such testing should not be undertaken if it is unlikely to alter patient management and should not be considered as a preliminary step leading to coronary revascularization. A revascularization procedure is rarely needed solely for the purpose of getting a patient through the perioperative period. Extensive cardiac evaluation before vascular operations can result in morbidity, delays, and patient refusal to undergo vascular surgery. A complete review of this subject is found in [Chapter 31](#).

Cardiac Catheterization and Prophylactic Revascularization

The largest series on outcome in vascular surgery patients is that of Hertzer and colleagues²⁴ from the Cleveland Clinic. These investigators performed cardiac catheterization in 1000 consecutive patients scheduled to undergo peripheral vascular surgery (aortic aneurysm resection, carotid endarterectomy, and lower extremity revascularization). The incidence and severity of CAD were assessed according to the following classification: normal coronary arteries; mild-to-moderate CAD with no lesion exceeding 70%

TABLE 56.2 Results of Coronary Angiography in 1000 Patients with Peripheral Vascular Disease

Angiographic Classification	CLINICAL CAD					
	NONE		SUSPECTED		TOTAL	
	No.	%	No.	%	No.	%
Normal coronary arteries	64	14	21	4	85	8.5
Mild-to-moderate CAD	218	49	99	18	317	32
Advanced, compensated CAD	97	22	192	34	289	29
Severe, correctable CAD	63	14	188	34	251	25
Severe, inoperable CAD	4	1	54	10	58	5.8

CAD, Coronary artery disease.

Data from Hertzer NR, Beven EG, Young JR, et al. Coronary artery disease in peripheral vascular patients: a classification of 1000 coronary angiograms and results of surgical management. *Ann Surg*. 1984;199:223–233.

stenosis; advanced, compensated CAD with one or more lesions exceeding 70% stenosis but with adequate collateral circulation; severe, correctable CAD with more than 70% stenosis in one or more coronary arteries; and severe inoperable CAD with greater than 70% stenosis in one or more coronary arteries and severe distal disease or poor ventricular function. The most remarkable findings were that only 8.5% of patients had normal coronary arteries and 60% had advanced or severe coronary lesions (>70% stenosis). Even when CAD was not suspected by the clinical history, more than a third of patients had advanced or severe coronary lesions ([Table 56.2](#)).

In the Hertzer series, patients with severe correctable CAD were offered CABG before their vascular surgery, patients with normal or mild-to-moderate CAD went directly to vascular surgery, and those with severe inoperable CAD were treated on an individual basis. Combined mortality rates over the immediate- and long-term (4.6-year follow-up) postoperative period are shown in [Table 56.3](#).¹⁷ Of the 216 patients who underwent coronary revascularization (CABG), 12 (5.5%) died after this surgery. This mortality rate is higher than that reported for patients undergoing CABG surgery without peripheral vascular disease (1% to 2%). Perhaps the risks associated with CABG should be seriously considered as part of the preoperative evaluation of these patients. When overall early and late mortality (>5 years) is considered, death occurred in 12% versus 26% of patients who did or did not undergo CABG. Although these data appear to support the beneficial effect of CABG on outcome, the mortality from CABG itself (5.5%) reduces its apparent benefits.

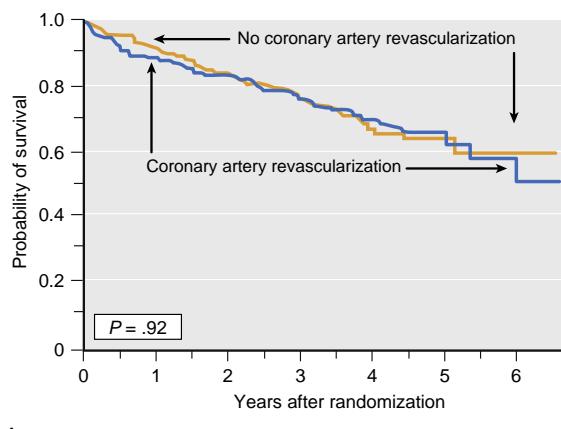
Two randomized clinical trials have been performed to determine the impact of prophylactic coronary artery revascularization on outcome after open aortic and lower extremity arterial vascular surgery.^{4,25} Of the 5859 patients screened in the CARP trial, 41190 underwent coronary angiography based on a combination of clinical risk factors and noninvasive stress imaging data.²⁶ The incidence and severity of CAD on these angiograms were 43% of patients had one or more major coronary arteries with at least a 70% stenosis suitable for revascularization (and were randomized to either revascularization or no revascularization before vascular surgery); 31% had

TABLE 56.3 Perioperative and Late Cardiac Deaths after Peripheral Vascular Reconstruction in Patients Monitored over a 5-Year Period According to Coronary Angiographic Classification

Clinical Features	Total No. of Patients	NORMAL OR MILD-TO-MODERATE CAD		ADVANCED, COMPENSATED CAD		SEVERE, CORRECTABLE CAD		SEVERE, INOPERABLE CAD		TOTAL CARDIAC DEATHS	
		No.	%	No.	%	No.	%	No.	%	No.	%
Men	685	10/242	4.1	33/204	16	13/174	7.5	6/24	25	14/41	34
Women	315	5/160	3.1	12/85	14	12/42	29	3/11	27	8/17	47
Age <70 yr	722	10/328	3.0	29/198	15	19/148	13	3/20	15	13/28	46
Age >70 yr	278	5/74	6.8	16/91	18	6/68	8.8	6/15	40	9/30	30
Normotensive	403	7/185	3.8	15/102	15	8/82	9.8	2/15	13	8/19	42
Hypertensive	597	8/217	3.4	30/187	16	17/134	13	7/20	35	14/39	36
Nondiabetic	830	12/348	3.4	28/232	12	17/183	9.3	8/30	27	13/37	35
Diabetic	170	3/54	5.5	17/57	30	8/33	24	1/5	20	9/21	43
Total	1000	15/402	3.7	45/289	16	25/216	12	9/35	26	22/58	38
										116	12

CABG, Coronary artery bypass grafting; CAD, coronary artery disease.

Data from Hertzler NR, Young JR, Beven EG, et al. Late results of coronary bypass in patients with peripheral vascular disease. II. Five-year survival according to sex, hypertension, and diabetes. *Cleve Clin J Med*. 1987;54:15–23.



No. at Risk

Revascularization 226 175 113 65 18 7

No revascularization 229 172 108 55 17 12

Fig. 56.1 Long-term survival in patients randomized to undergo coronary artery revascularization or no coronary artery revascularization before elective major vascular surgery (Coronary Artery Revascularization Prophylaxis trial). (From McFalls EO, Ward HB, Moritz TE, et al. Coronary-artery revascularization before elective major vascular surgery. *N Engl J Med*. 2004;351:2796–2804.)

nonobstructed coronary arteries; 18% had coronary stenosis considered unsuitable for revascularization; and 5% had left main coronary artery stenosis of 50% or more. The CARP trial showed that prophylactic revascularization (by CABG or PCI) was generally safe but did not improve long-term outcome after vascular surgery. Long-term mortality (2.7 years) was 22% in the revascularization group and 23% in the group considered inappropriate for revascularization (Fig. 56.1). Although the trial was not designed to test the short-term benefit of prophylactic revascularization, perioperative outcomes were not decreased, including death (3.1% vs. 3.4%) and MI (12% vs. 14%). The CARP trial results can be applied to most of the vascular surgery patients; however, they cannot be extrapolated to patients with unstable cardiac symptoms, left main

coronary artery disease, aortic stenosis, or severe left ventricular dysfunction because these conditions excluded patients from study participation. The DECREASE-V trial²⁶ screened 1880 vascular surgery patients, 430 of whom with three or more clinical risk factors underwent noninvasive stress testing using stress-echo or perfusion imaging. Patients with extensive stress-induced ischemia (26%) were randomly assigned to revascularization or no revascularization. Coronary angiography showed two-vessel disease in 24%, three-vessel disease in 67%, and left main coronary artery disease in 8%. Prophylactic coronary revascularization (CABG or PCI) did not improve perioperative or long-term outcome (Table 56.4). The incidence of all-cause death or nonfatal MI at 30 days in patients who underwent revascularization or who did not was 43% versus 33%, respectively. The incidence of the composite end point at 1 year was similar, 49% versus 44%, respectively. As noted previously, this trial has come under scrutiny based on concerns of scientific misconduct by the principal investigator.

The lack of benefit of prophylactic coronary revascularization in the CARP and DECREASE-V trials is difficult to reconcile with the more favorable data from Hertzler and co-workers²⁵ and other studies (Coronary Artery Surgery Study [CASS]²⁷ and Bypass Angioplasty Revascularization Investigation [BARI]²⁸). Clearly, issues are involved that go beyond critical coronary lesions; perhaps the current understanding of the pathophysiology of perioperative MI is incomplete. For example, perioperative MI may be caused by culprit lesions (i.e., vulnerable plaques with high likelihood of thrombotic complications) often located in coronary vessels *without* critical stenosis.²⁹ For this type of MI (atherothrombotic), perioperative strategies aimed at reducing potential triggers of coronary plaque destabilization and rupture may be more appropriate than those leading to coronary revascularization. Demand ischemia is likely the predominant cause of perioperative MI, which has been confirmed by a recent angiographic study.³⁰

TABLE 56.4 Perioperative and Long-Term Patient Outcomes from the Dutch Echocardiographic Cardiac Risk Evaluation Applying Stress Echocardiography (DECREASE)-V—V TRIAL

	Revascularization No. (%)	No Revascularization No. (%)	HR (95% CI)	P-value
NO. OF PATIENTS	49	52		
Events before surgery	2 (4.1)	0		0.23
All-cause mortality	1 (2.1)	0		0.11
Myocardial infarction	3 (6.1)	0		
Composite				
Events up to 30 days after surgery	11 (22.5)	6 (11.5)	2.2 (0.74-6.6)	0.14
All-cause mortality	17 (34.7)	16 (30.8)	1.4 (0.73-2.8)	0.30
Myocardial infarction	21 (42.9)	17 (32.7)		
Composite				
Events up to 365 days after surgery	13 (26.5)	12 (23.1)	1.3 (0.55-2.9)	0.58
All-cause mortality	18 (36.7)	19 (36.5)	1.2 (0.68-2.3)	0.48
Myocardial infarction	24 (49.0)	23 (44.2)		
Composite				

CI, Confidence interval; HR, hazard ratio.

From Poldermans D, Schouten O, Vidakovic R, et al. A clinical randomized trial to evaluate the safety of a noninvasive approach in high-risk patients undergoing major vascular surgery. The DECREASE-V Pilot Study. *J Am Coll Cardiol*. 2007;49:1763–1769.

ASSESSMENT OF PULMONARY FUNCTION

Postoperative pulmonary complications are potentially serious in patients undergoing vascular surgery, with the most significant morbidity seen in patients undergoing open aortic procedures (see also [Chapter 54](#)). The most important pulmonary complications are atelectasis, pneumonia, respiratory failure, and exacerbation of underlying chronic disease. Given the prevalence of cigarette smoking in this population, chronic obstructive pulmonary disease (COPD) and chronic bronchitis are common and, when present, place the patient at increased risk for postoperative pulmonary complications. When clinical assessment suggests severe pulmonary compromise, pulmonary function tests may be useful in evaluating and optimizing respiratory function (see also [Chapters 32 and 41](#)). Preoperative analysis of arterial blood gases should be used to establish a baseline for postoperative comparison. Baseline hypercapnia (partial pressure of arterial carbon dioxide >45 mm Hg) indicates a more frequent risk for postoperative morbidity. Bronchodilator therapy may be indicated on the basis of results of pulmonary function tests, although the risk for β -adrenergic agonist-induced arrhythmia or myocardial ischemia also must be considered. Preoperative treatment with a short course of glucocorticoids (prednisone 40 mg/day for 2 days) may be helpful for patients with significant COPD or asthma. Evidence of pulmonary infection should be treated with appropriate antibiotics. Although improved pulmonary outcome with regional anesthesia is not clear, patients with significant pulmonary disease may benefit from epidural techniques. Use of these techniques in the postoperative period helps to avoid respiratory depression from systemic opiates (see also [Chapter 81](#)). Pulmonary complications in the postoperative period are difficult to avoid. Incentive spirometry and continuous positive airway pressure (CPAP) do provide benefit.³¹ Given proper pulmonary care, even patients with severe pulmonary insufficiency, however, may undergo aortic surgery with acceptable morbidity and mortality outcomes.³²

ASSESSMENT OF RENAL FUNCTION

Chronic renal disease is common in vascular surgery patients and is associated with an increased risk for death and cardiovascular disease (see also [Chapters 30 and 42](#)).³³ Chronic renal disease strongly predicts long-term mortality in patients with symptomatic lower extremity arterial occlusive disease irrespective of disease severity, cardiovascular risk, and concomitant treatment.³⁴ Cardiovascular disease is independently associated with a decline in renal function and the development of kidney disease.³⁵ Serum creatinine and creatinine clearance are used to assess renal function perioperatively. A preoperative serum creatinine level more than 2 mg/dL is an independent risk factor for cardiac complications after major noncardiac surgery.³⁶ Preoperative creatinine clearance less than 60 mL/min is an independent predictor of both short-term and long-term mortality after elective vascular surgery.³⁷ Perioperative β -adrenergic blocker³⁸ and statin³⁷ administration decrease risk for death in vascular surgery patients with renal impairment. Atherosclerotic disease in the abdominal aorta or renal arteries may compromise renal blood flow and renal function. Conversely, renal artery stenosis causes hypertension through renin-induced and angiotensin-induced vasoconstriction. Hypertension itself may cause renal insufficiency or failure. Diabetic nephropathy is also common (see also [Chapter 32](#)). Superimposed on baseline abnormalities in renal function are the preoperatively and intraoperatively administered angiographic dyes, which are directly nephrotoxic. Renal ischemia occurs with interruption of renal blood flow from aortic cross-clamping. Even with infrarenal aortic cross-clamps, renal blood flow may decrease despite normal systemic arterial blood pressure and cardiac output. Embolic plaque can be showered into the renal arteries, especially when suprarenal aortic cross-clamps are applied and released. Fluctuations in intravascular volume and cardiac output can compromise renal perfusion during the intraoperative and postoperative periods. In one series of more than 500 patients, the prevalence of acute renal failure was 7% after abdominal aortic reconstruction.

PERIOPERATIVE β -ADRENERGIC BLOCKER THERAPY

Perioperative β -adrenergic blocker therapy is an important and controversial topic, particularly in patients undergoing vascular surgery, and is reviewed more fully in [Chapters 31 and 32](#). Patients receiving chronic β -adrenergic blocker therapy should continue taking β -adrenergic blockers throughout the perioperative period. However, β -adrenergic blockers should not be used as the initial or primary treatment of tachycardia caused by perioperative events, such as hypovolemia, anemia, pain, or infection, because these conditions require prompt treatment of the underlying cause. Treatment of tachycardia caused by the sympathetic stimulation associated with surgical stress should be considered in high-risk patients, particularly those with known ischemic potential (i.e., ischemia on preoperative testing). Hypotension and bradycardia should be avoided. Acute initiation of large-dose β -adrenergic blockade in the perioperative period should be avoided. If a decision is made to initiate β -blocker treatment in the perioperative period to reduce cardiac risk, the safest approach may be to initiate therapy with a small dose and titrate to effect over a 7- to 10-day period before the planned surgery. Perioperative β -adrenergic blocker therapy can decrease the number of patients referred for preoperative cardiac testing. However, such testing should not be eliminated, and its risk-to-benefit ratio should be carefully assessed.

PERIOPERATIVE STATIN THERAPY

In addition to their lipid-lowering properties, statins have beneficial anti-inflammatory, plaque-stabilizing, and antioxidant effects (see also [Chapter 32](#)). Over the last decade, statin use has emerged as a promising strategy for the prevention of perioperative cardiovascular complications in patients undergoing vascular surgery.³⁹ This approach is supported by the double-blind, placebo-controlled DECREASE-III trial. Unfortunately, controversy exists regarding this trial because of scientific misconduct identified by a recent investigation by Erasmus University.⁴⁰ Statin use can help preserve renal function after aortic surgery and improve graft patency after lower extremity bypass surgery. Although current guidelines recommend the use of statins in all patients with peripheral arterial disease, the optimal timing and dosing of statins for perioperative use have not been established.

PERIOPERATIVE DUAL ANTIPLATELET THERAPY

The timing of noncardiac surgery in patients treated with coronary stents with the risk of stent thrombosis if the dual antiplatelet therapy (DAPT) is discontinued versus the risk of increased intraoperative bleeding if DAPT is continued.^{20,41} Previous recommendations regarding the duration of DAPT and timing of noncardiac surgeries were based on observations of those treated with first-generation stents. Currently, used newer generations of stents, particularly the newer drug eluting stents (DES), have lower risk of in-stent thrombosis and require a shorter minimum duration of DAPT.⁴² The safety of treating patients with newer generation DES treated for shorter durations of DAPT (3

to 6 months) has been demonstrated in a meta-analysis of four trials.⁴³ Also, in the PARIS (Patterns of Nonadherence to Antiplatelet Regimens in Stented Patients) registry, interruption of DAPT based on the physician judgment in patients undergoing surgery at any point did not affect the risk of major cardiac events.⁴⁴ Hence, the ACC/AHA guidelines were changed in 2016 to reflect those changes (see [Chapters 31 and 32](#)).⁴⁵

Abdominal Aortic Reconstruction

Anesthesia for conventional abdominal aortic reconstruction requires an understanding of the pathophysiology, extensive knowledge of the surgical procedure, the ability to interpret sophisticated hemodynamic data, and skillful pharmacologic control and manipulation of hemodynamics. Preoperative and intraoperative communication with the surgical team is essential. All open operative procedures on the abdominal aorta and its major branches require large incisions and extensive dissection, clamping and unclamping of the aorta or its major branches, varying duration of organ ischemia-reperfusion, significant fluid shifts and temperature fluctuations, and activation of neurohumoral and inflammatory responses. The major objectives of surgical treatment of the aorta are to relieve symptoms, reduce the frequency of associated complications, and in the case of aortic aneurysm, prevent rupture. Over the last two decades, the growth and development of catheter-based technology for the treatment of peripheral arterial disease have generated tremendous interest for less invasive methods to treat aortic disease.

Endovascular aortic aneurysm repair (discussed later) has become an established, less invasive alternative to conventional open repair, and its use has expanded to more than 75% of elective repairs and 30% of rupture repairs.⁴⁶ The endovascular field continues to evolve rapidly with new devices, innovations, and indications for aortic disease.

NATURAL HISTORY AND SURGICAL MORTALITY

Abdominal Aortic Aneurysm

Abdominal aortic aneurysms (AAAs) occur frequently in elderly men, with an incidence approaching 8% (see also [Chapter 65](#)). Increasing age, smoking, family history of AAA, and atherosclerotic disease are established risk factors. Although the prevalence is less frequent in women, the risk factors for AAA resemble those in men. More than 30,000 deaths result from rupture of AAAs each year in the United States.⁴⁷ The number of hospital discharges each year with the first diagnosis of aortic aneurysm is nearly 70,000. Approximately 40,000 patients undergo repair of AAA each year in the United States, at a cost likely to exceed a billion dollars. The incidence of AAA appears to be increasing and is age- and gender-dependent.

AAA is a multifactorial disease associated with aortic aging and atherosclerosis. Although no unified concept of pathogenesis exists, genetic, biochemical, metabolic, infectious, mechanical, and hemodynamic factors may contribute to the development of AAA disease. Adventitial elastin degradation (elastolysis), a hallmark of AAA formation, may be the primary event. Chronic inflammation plays a

fundamental role in the destruction of connective tissue in the aortic wall. Concomitant aortoiliac occlusive disease is present in approximately 20% to 25% of patients with AAA. Approximately 5% of patients undergoing abdominal aortic resection have inflammatory aneurysms. Rare causes of AAA disease include trauma, mycotic infection, syphilis, and Marfan syndrome.

Most AAAs are detected incidentally when imaging is performed for other reasons or through screening programs. The natural history of AAA disease is progressive enlargement and ultimate rupture and death. The diameter and rate of expansion of asymptomatic AAAs are the best predictors of the risk for rupture. Current guidelines emphasize that it is not possible to recommend a single threshold diameter for operative intervention that can be generalized to all patients. Yet, elective repair should be undertaken in all patients with AAA 6 cm or larger in diameter. Although some controversy exists regarding elective AAA repair when its diameter is in the 5.5- to 5.9-cm range, the risk for rupture of a 5.5-cm aneurysm (per year) is equal to or greater than the risk for perioperative mortality, and thus surgical repair is indicated. The 1-year incidence of probable rupture in patients refusing or unfit for elective repair is 9.4%, 10.2%, and 32.5% for aneurysms 5.5 to 5.9 cm, 6.0 to 6.9 cm, and 7.0 cm or greater, respectively.⁴⁸ Over 90% of AAAs are less than the current threshold (5.5 cm) for surgical repair at the time of detection. Randomized controlled trials in patients with AAAs 4.0 to 5.5 cm in diameter have provided important insight into the natural history of small asymptomatic aortic aneurysms.⁴⁹ Four trials have demonstrated that surveillance of small aneurysms (4.0 to 5.5 cm) is a safe management option and that early repair (open or endovascular surgery) did not result in any long-term survival benefit. Surgical repair is often considered if small aneurysms become symptomatic or expand more than 0.5 cm in a 6-month period. Although significant interest exists in medical treatment (e.g., antibiotics, β -adrenergic blockers, statins) to delay or reverse expansion of small aneurysms, evidence for a protective effect is limited.⁵⁰ Aneurysms less than 4.0 cm in diameter are thought to be relatively benign in terms of rupture and expansion.

Perioperative mortality from elective resection of infrarenal AAAs has progressively declined from 18% to 20% during the 1950s, 6% to 8% in the mid-1960s, 5% to 6% in the early 1970s, and 2% to 4% in the 1980s, at which time it plateaued. A publication of data from 1000 consecutive elective open infrarenal abdominal aneurysm repairs over a 15-year period reported a perioperative mortality rate of 2.4%.⁵¹ Hertzler and co-workers⁵² reported a mortality rate of 1.2% for 1135 consecutive elective open infrarenal abdominal aortic repairs at the Cleveland Clinic. This single-center mortality rate is considerably less than the mortality rates of 5.6% to 8.4% reported from large national data sets. These more frequent mortality rates on the national level suggest that all the technologic and treatment advances over the last 2 decades have not had an impact on outcomes of patients requiring open AAA repair. Regionalization of patient care and endovascular treatments currently hold the most promise for improvement in operative mortality.

For ruptured AAAs, perioperative mortality has not changed significantly over the last 4 decades and remains nearly 50%, with few exceptions. Including patients with

rupture who die before reaching a hospital, the overall mortality rate after rupture may very well exceed 90%.

The long-term durability of open infrarenal AAA repair is excellent and well established. The incidence of late graft complications is infrequent (0.4% to 2.3%). Postoperative survival rates after repair of non-ruptured AAA are 92% at 1 year and 67% at 5 years.

Aortoiliac Occlusive Disease

The infrarenal aorta and the iliac arteries are two of the most common sites of chronic atherosclerosis. Because of the diffuse and progressive nature of aortoiliac atherosclerosis, plaque enlargement may reduce blood flow to the lower extremities below a critical level and result in symptoms of ischemia. Unlike patients with aortic aneurysmal disease, patients undergo surgery for aortoiliac occlusive disease only if they are symptomatic. Surgical intervention is indicated for disabling intermittent claudication and limb-threatening ischemia. Intervention is directed toward restoring peripheral pulsatile circulation to relieve claudication and toward preventing amputation. Patients with localized aortoiliac occlusive disease typically have claudication because collateral circulation adequate to prevent critical lower extremity ischemia usually exists. Perioperative mortality is lower in patients undergoing aortoiliac reconstruction than in those undergoing abdominal aortic surgery.

Therapeutic options for managing aortoiliac occlusive disease include anatomic or direct reconstruction (i.e., aortobifemoral bypass), extra-anatomic or indirect bypass grafts (i.e., axillofemoral bypass), and catheter-based endoluminal techniques (i.e., percutaneous transluminal angioplasty [PTA] with or without stent insertion). Aortobifemoral bypass is viewed as the gold standard in treating aortoiliac occlusive disease. Extra-anatomic bypass grafts are generally reserved for specific indications, usually patients with infection, failure of previous reconstruction, or prohibitive risk. Reduced long-term patency and inferior functional results are frequently the trade-off for lower perioperative morbidity and mortality. Catheter-based endoluminal techniques, such as PTA, are used for relatively localized disease and may be reasonable alternatives to aortobifemoral bypass in 10% to 15% of patients with aortoiliac occlusive disease.

Renal and Visceral Arterial Insufficiency

Atherosclerosis is the most common cause of renal artery stenosis. Occlusive lesions are located almost exclusively in the proximal segment and orifice of the renal artery and are usually an extension of aortic atherosclerosis. Fibromuscular dysplasia is an important, but less common, cause of renal artery stenosis and most frequently involves the distal two thirds of the renal arteries. Hemodynamically significant renal artery stenosis may cause hypertension by activation of the renin-angiotensin-aldosterone system, and bilateral involvement may result in renal failure. Patients with renovascular hypertension frequently have poorly controlled hypertension despite maximal medical therapy. These patients often have severe bilateral renal artery stenosis and may have recurrent congestive heart failure or flash pulmonary edema. Indications for intervention include control of hypertension and salvage of renal function.

Operative interventions include aortorenal bypass, extra-anatomic bypass (hepatorenal or splenorenal bypass), or transaortic endarterectomy. Suprarenal or supraceliac aortic cross-clamping is frequently required for open operative interventions. PTA with stenting of the renal artery is used as the first-line treatment in selected patients.

Stenosis at the origin of the celiac and mesenteric arteries occurs as a result of extension of aortic atherosclerosis. The inferior mesenteric artery is by far the most commonly involved, followed by the superior mesenteric artery and the celiac artery.

Occlusion of a single vessel rarely causes ischemic symptoms because of the extensive nature of visceral collateralization. However, occlusion or significant stenosis of any two vessels may compromise collateral flow sufficiently to give rise to chronic visceral ischemia. Operative repair of visceral artery stenosis is reserved for symptomatic patients. Operative interventions include transaortic endarterectomy and bypass grafts, which frequently require supraceliac aortic cross-clamping. Mortality rates for such procedures range from 7% to 18%. To avoid the high mortality associated with open repair, PTA with stenting has increasingly been applied in patients with chronic visceral ischemia. Acute visceral artery occlusion can be caused by an embolus or, less commonly, by thrombosis. To avoid the extremely high mortality associated with acute visceral ischemia, diagnosis and surgical intervention must occur before gangrene of the bowel develops.

AORTIC CROSS CLAMPING

The pathophysiology of aortic cross-clamping is complex and depends on many factors, including level of the cross-clamp, status of the left ventricle, degree of periaortic collateralization, intravascular blood volume and distribution, activation of the sympathetic nervous system, and anesthetic drugs and techniques. Most abdominal aortic reconstructions require clamping at the infrarenal level. However, clamping at the suprarenal and supraceliac levels is required for suprarenal aneurysms and renal or visceral reconstructions and is frequently necessary for juxtarenal aneurysms, inflammatory aneurysms, and aortoiliac occlusive disease with proximal extension. These higher levels of aortic occlusion have a significant impact on the cardiovascular system, as well as on other vital organs rendered ischemic or hypoperfused. Ischemic complications may result in renal failure, hepatic ischemia and coagulopathy, bowel infarction, and paraplegia. With EVAR now common, an increasing proportion of patients undergoing open repair have anatomically complex aneurysms, many of which require suprarenal cross-clamping.⁵³

Hemodynamic and Metabolic Changes

The hemodynamic and metabolic changes associated with aortic cross-clamping are summarized in **Box 56.1**. The magnitude and direction of these changes are complex, dynamic, and vary among experimental and clinical studies. However, several important factors must be considered (**Box 56.2**). The systemic cardiovascular consequences of aortic cross-clamping can be dramatic, depending primarily on the level at which the cross-clamp is applied. Arterial hypertension above the clamp and arterial hypotension

BOX 56.1 Physiologic Changes With Aortic Cross-Clamping* and Therapeutic Interventions

Hemodynamic Changes

- ↑ Arterial blood pressure above the clamp
- ↓ Arterial blood pressure below the clamp
- ↑ Segmental wall motion abnormalities
- ↑ Left ventricular wall tension
- ↓ Ejection fraction
- ↓ Cardiac output^{†,‡}
- ↓ Renal blood flow
- ↑ Pulmonary occlusion pressure
- ↑ Central venous pressure
- ↑ Coronary blood flow

Metabolic Changes

- ↓ Total body oxygen consumption
- ↓ Total body carbon dioxide production
- ↑ Mixed venous oxygen saturation
- ↓ Total body oxygen extraction
- ↑ Epinephrine and norepinephrine
- Respiratory alkalosis
- Metabolic acidosis

Therapeutic Interventions

- Afterload reduction
 - Sodium nitroprusside
 - Inhalational anesthetics
 - Amrinone
 - Shunts and aorta-to-femoral bypass
- Preload reduction
 - Nitroglycerin
 - Controlled phlebotomy
 - Atrial-to-femoral bypass
- Renal protection
 - Fluid administration
 - Distal aortic perfusion techniques
 - Selective renal artery perfusion
 - Mannitol
 - Drugs to augment renal perfusion
- Other
 - Hypothermia
 - ↓ Minute ventilation
 - Sodium bicarbonate

*These changes are of greater significance with longer duration of cross-clamping and with more proximal cross-clamping.

†Cardiac output may increase with thoracic cross-clamping.

‡When ventilatory settings are unchanged from pre-clamp levels.

below the clamp are the most consistent components of the hemodynamic response to aortic cross-clamping at any level. The increase in arterial blood pressure above the clamp is primarily due to the sudden increase in impedance to aortic blood flow and the resultant increase in systolic ventricular wall tension or afterload. However, factors such as myocardial contractility, preload, blood volume, and activation of the sympathetic nervous system also may be important.⁵⁴ Cross-clamping of the aorta at or above the diaphragm results in the most profound increases in arterial blood pressure unless diverting circulatory support or IV vasodilators are used. Changes in cardiac output and filling pressure with aortic cross-clamping are not consistent and require an integrated approach in understanding

BOX 56.2 Factors That May Influence the Magnitude and Direction of Physiologic Changes Occurring With Aortic Cross-Clamping

- Level of aortic cross-clamp
- Species differences
- Anesthetic agents and techniques
- Use of vasodilator therapy
- Use of diverting circulatory support
- Degree of periaortic collateralization
- Left ventricular function
- Status of the coronary circulation
- Volume status
- Neuroendocrine activation
- Duration of aortic cross-clamp
- Body temperature

TABLE 56.5 Percent Change in Cardiovascular Variables on Initiation of Aortic Occlusion

Cardiovascular Variable	PERCENT CHANGE AFTER OCCLUSION		
	Supraceliac	Suprarenal-Infraceliac	Infrarenal
Mean arterial blood pressure	54	5*	2*
Pulmonary capillary wedge pressure	38	10*	0*
End-diastolic area	28	2*	9*
End-systolic area	69	10*	11*
Ejection fraction	-38	-10*	-3*
Patients with wall motion abnormalities	92	33	0

*Statistically different ($P < .05$) from group undergoing supraceliac aortic occlusion.

From Roizen MF, Beaupre PN, Alpert RA, et al. Monitoring with two-dimensional transesophageal echocardiography: comparison of myocardial function in patients undergoing supraceliac, suprarenal-infraceliac, or infrarenal aortic occlusion. *J Vasc Surg*. 1984;1:300–305.

aortic cross-clamping causes significant increases in left ventricular end-systolic and end-diastolic area (69% and 28%, respectively), as well as wall motion abnormalities indicative of ischemia in 11 of 12 patients (Table 56.5). Aortic cross-clamping at the suprarenal level causes similar but smaller cardiovascular changes and clamping at the infrarenal level is associated with only minimal changes and no wall motion abnormalities.

The marked increases in ventricular filling pressure (preload) reported with high aortic cross-clamping have been attributed to increased afterload and redistribution of blood volume, which is of prime importance during thoracic aortic cross-clamping. The splanchnic circulation, an important source of functional blood volume reserve, is central to this hypothesis. The splanchnic organs contain nearly 25% of the total blood volume, nearly two thirds (>800 mL) of which can be autotransfused from the highly compliant venous vasculature into the systemic circulation within seconds.⁵⁷ Primarily because of smaller splanchnic venous capacitance, blood volume is redistributed from vascular beds distal to the clamp to the relatively noncompliant vascular beds proximal to the clamp (Fig. 56.3). Both passive and active mechanisms lower splanchnic venous capacitance with thoracic aortic cross-clamping. Cross-clamping the aorta above the splanchnic system dramatically reduces splanchnic arterial flow, which produces a significant reduction in pressure within the splanchnic capacitance vessels.⁵⁸ This decreased pressure allows the splanchnic veins to passively recoil and increase venous return to the heart and blood volume proximal to the clamp. Thoracic aortic cross-clamping also results in significant increases in plasma epinephrine and norepinephrine, which may enhance venumotor tone both above and below the clamp. The splanchnic veins are highly sensitive to adrenergic stimulation. The major effect of catecholamines on the splanchnic capacitance vessels is vasoconstriction, which actively forces out splanchnic blood, reduces splanchnic venous capacitance, and increases venous return to the heart.⁵⁸

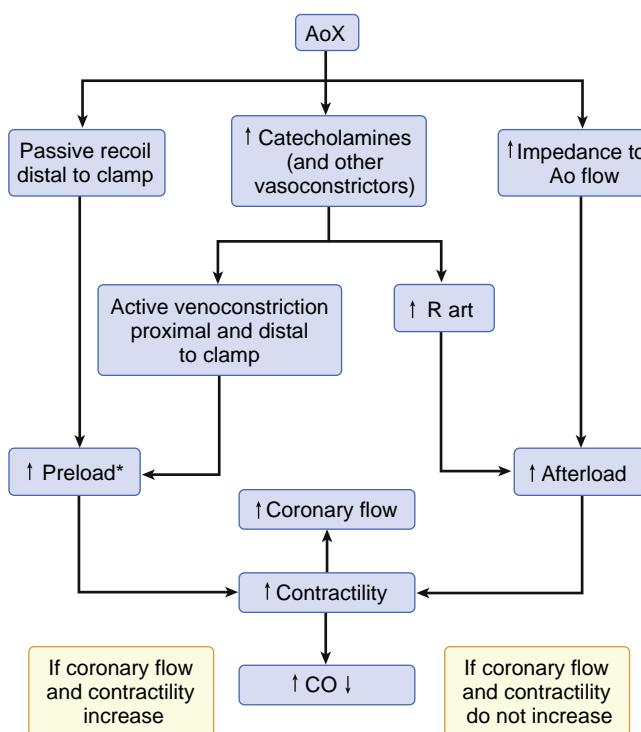


Fig. 56.2 Systemic hemodynamic response to aortic cross-clamping. Preload (asterisk) does not necessarily increase with infrarenal clamping. Depending on splanchnic vascular tone, blood volume can be shifted into the splanchnic circulation and preload will not increase. Ao, Aortic; AoX, aortic cross-clamping; CO, cardiac output; R art, arterial resistance.

the direction and magnitude of such changes (Fig. 56.2). Cross-clamping of the proximal descending thoracic aorta increases mean arterial, central venous, mean pulmonary arterial, and pulmonary capillary wedge pressure by 35%, 56%, 43%, and 90%, respectively, and decreases the cardiac index by 29%.⁵⁵ Heart rate and left ventricular stroke work are not significantly changed. Supraceliac aortic cross-clamping increases mean arterial pressure by 54% and pulmonary capillary wedge pressure by 38%.⁵⁶ Ejection fraction, as determined by two-dimensional echocardiography, decreases by 38%. Despite normalization of systemic and pulmonary capillary wedge pressure with anesthetic agents and vasodilator therapy, supraceliac

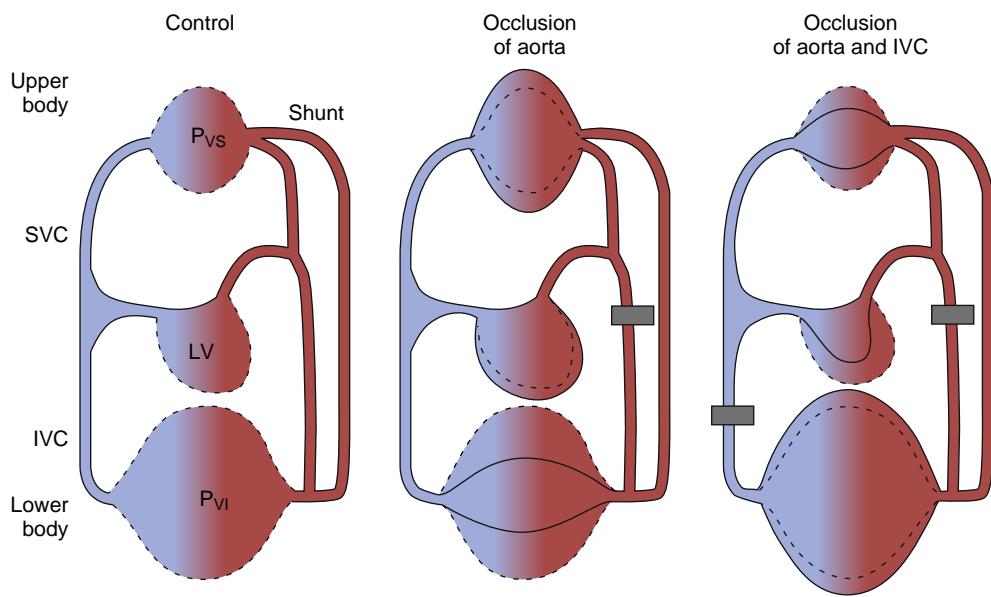


Fig. 56.3 Schematic drawing of the circulation. Compliant regions (dashed lines) of the upper and lower part of the body and end-diastolic volumes of the left ventricle in control state (left panel) are shown after occlusion of the aorta alone (middle panel) and combined occlusion of the aorta and inferior vena cava (right panel). IVC, Inferior vena cava; LV, left ventricle; PVS and PVI, pressure in compliant regions of the upper and lower body, respectively; Shunt, physiologic shunt; SVC, superior vena cava.

Several animal studies support the blood volume redistribution hypothesis. Cross-clamping the thoracic aorta in dogs results in marked increases in mean arterial pressure and end-diastolic left ventricular pressure (84% and 188%, respectively) and no significant change in stroke volume.⁵⁹ In this same experimental model, simultaneous cross-clamping of the thoracic aorta and the inferior vena cava resulted in no significant change in mean arterial pressure or preload (see Fig. 56.3). Stroke volume was reduced by 74%. By transfusing blood (above the clamps) during this period of simultaneous clamping, the authors reproduced the hemodynamic effect of thoracic aortic cross-clamping alone. This study also demonstrated that thoracic aortic cross-clamping is associated with a significant and dramatic increase (155%) in blood flow above the level of the clamp whereas no change in blood flow occurred with simultaneous aortic and inferior vena cava clamping. In other animal models, the proximal aortic hypertension and increased central venous pressure (CVP) occurring after thoracic aortic cross-clamping were completely reversed by phlebotomy.⁶⁰ Aortic cross-clamping at the thoracic and suprarenal levels in dogs both resulted in proximal aortic hypertension, but only occlusion at the thoracic level increased central venous pressure.⁶¹ In this study, thoracic aortic occlusion increased blood volume in organs and tissues proximal to the clamp whereas no such increase occurred with suprarenal aortic cross-clamping. These experimental data strongly support the hypothesis of blood volume redistribution during aortic cross-clamping and help explain the marked differences in hemodynamic responses observed after aortic cross-clamping at different levels.⁵⁶

Afterload-dependent increases in preload also occur with aortic cross-clamping, usually in the setting of impaired myocardial contractility and reduced coronary reserve. The impaired left ventricle may respond to increased afterload with an increase in end-systolic volume and a concomitant reduction in stroke volume (afterload mismatch). The

reduction in stroke volume may be due to limited preload reserve, myocardial ischemia, or inability of the heart to generate a pressure-induced increase in contractility (the Anrep effect). If right ventricular function remains normal, the pre-clamp right ventricular stroke volume added to the increased left ventricular end-systolic volume results in left ventricular dilation and elevated end-diastolic volume. If corrective measures are not undertaken, overt left ventricular overload may result, with severe peripheral organ dysfunction and pulmonary edema.

Most clinical studies indicate that cardiac output decreases with thoracic aortic cross-clamping (without vasodilator therapy or diverting circulatory support), whereas most animal studies show no significant change or an increase in cardiac output.

However, the status of the left ventricle clearly plays a major role. Whereas a normal intact heart can withstand large increases in volume without significant ventricular distension or dysfunction, an impaired heart with reduced myocardial contractility and coronary reserve may respond to such increase in volume conditions with marked ventricular distension as a result of acute left ventricular dysfunction and myocardial ischemia. Although impaired myocardial contractility and reduced coronary reserve are rare in animal experiments, such disorders are frequent in the elderly population undergoing aortic reconstruction. The increase in ventricular loading conditions seen with thoracic and supraceliac cross-clamping^{55,56} in the clinical setting may increase left ventricular wall stress (afterload), with resultant acute deterioration of left ventricular function and myocardial ischemia.

Impaired subendocardial perfusion caused by high intramyocardial pressure may be the cause of wall motion abnormalities and changes in ejection fraction. Reflex mechanisms causing immediate feedback inhibition may also explain the reduction in cardiac output with aortic cross-clamping. For example, baroreceptor activation resulting from increased aortic pressure should depress the heart rate, contractility,

and vascular tone. Thoracic aortic cross-clamping with the use of vasodilator therapy to normalize ventricular loading conditions maintains or increases cardiac output.⁶⁵ The metabolic effects of aortic cross-clamping are summarized in **Box 56.1**. Cross-clamping of the thoracic aorta decreases total-body O₂ consumption by approximately 50%. For reasons that are unclear, O₂ consumption decreases in tissues above the clamp. In clinical studies, increased mixed venous O₂ saturation occurs with aortic cross-clamping above the celiac axis. This increase in mixed venous O₂ saturation may be explained by a reduction in O₂ consumption that exceeds the reduction in cardiac output, thus decreasing total body O₂ extraction. Central hypervolemia and increased arteriovenous shunting in tissues proximal to the aortic clamp may play a role in reducing total body O₂ extraction. Arterial blood pressure, blood flow, and O₂ consumption distal to a thoracic aortic cross-clamp decrease by 78% to 88%, 79% to 88%, and 62%, respectively, from baseline values before clamping. Blood flow through tissues and organs below the level of aortic occlusion is dependent on perfusion pressure and is independent of cardiac output. Administration of sodium nitroprusside to maintain proximal aortic pressure above the cross-clamp at pre-clamp levels has been shown to further reduce arterial pressure distal to the clamp by 53%. As discussed later, these data have significant implications regarding vital organ protection during aortic cross-clamping.

The cardiovascular response to infrarenal aortic cross-clamping is less significant than with high aortic cross-clamping (see **Table 56.5**). Although several clinical reports have noted no significant hemodynamic response to infrarenal cross-clamping, the hemodynamic response generally consists of increases in arterial pressure (7% to 10%) and systemic vascular resistance (20% to 32%), with no significant change in heart rate. Cardiac output is most consistently decreased by 9% to 33%. Reported changes in ventricular filling pressure have been inconsistent. Blood volume redistribution may affect preload with infrarenal aortic cross-clamping (see **Fig. 56.3**). In this situation, blood volume below the clamp shifts to the compliant venous segments of the splanchnic circulation above the clamp, thereby dampening the expected increase in preload. The preload changes with infrarenal aortic cross-clamping also may depend on the status of the coronary circulation. Patients with severe ischemic heart disease responded to infrarenal aortic cross-clamping with significantly increased central venous (35%) and pulmonary capillary (50%) pressure, whereas patients without CAD had decreased filling pressure. Echocardiographically detected segmental wall motion abnormalities occur in up to 30% of patients during infrarenal aortic reconstruction, with over 60% occurring at the time of aortic cross-clamping. Patients with aortoiliac occlusive disease may have less hemodynamic response to infrarenal aortic cross-clamping than do patients with AAA disease, perhaps as a result of more extensive periaortic collateral vascularization.

RENAL FUNCTION AND PROTECTION

Preservation of renal function is highly important during aortic reconstructive surgery. Acute renal failure occurs in approximately 3% of patients undergoing elective infrarenal

aortic reconstruction, and mortality resulting from postoperative acute renal failure is more frequent than 40%. Despite significant improvements in the perioperative care of these patients, the frequent incidence of morbidity and mortality resulting from acute renal failure has remained largely unchanged over the last several decades. Most of the morbidity associated with significant postoperative renal dysfunction is nonrenal.

The adequacy of renal perfusion “cannot” be assumed by urine output. Although urine output is closely monitored and often augmented during aortic surgery, intraoperative urine output does not predict postoperative renal function. Procedures requiring aortic cross-clamping above the renal arteries dramatically reduce renal blood flow. Experimental studies report an 83% to 90% reduction in renal blood flow during thoracic aortic cross-clamping. Infrarenal aortic cross-clamping in humans is associated with a 75% increase in renal vascular resistance, a 38% decrease in renal blood flow, and a redistribution of intrarenal blood flow toward the renal cortex. These rather profound alterations in renal hemodynamics occurred despite no significant change in systemic hemodynamics, and they persisted after unclamping. The sustained deterioration in renal perfusion and function during and after infrarenal aortic cross-clamping has been attributed to renal vasoconstriction, but the specific pathophysiologic process remains unknown. Renal sympathetic blockade with epidural anesthesia to a T6 level does not prevent or modify the severe impairment in renal perfusion and function that occurs during and after infrarenal aortic cross-clamping. Although plasma renin activity is increased during aortic cross-clamping, pretreatment with converting enzyme inhibitors before infrarenal aortic cross-clamping does not attenuate the decreased renal blood flow and glomerular filtration rate. Other mediators, such as plasma endothelin, myoglobin, and prostaglandins, may contribute to the decreased renal perfusion and function after aortic cross-clamping.

Acute tubular necrosis accounts for nearly all the renal dysfunction and failure after aortic reconstruction. The degree of preoperative renal insufficiency remains the strongest predictor of postoperative renal dysfunction. In addition to aortic cross-clamping-induced reductions in renal blood flow, ischemic reperfusion injury, intravascular volume depletion, embolization of atherosclerotic debris to the kidneys, and surgical trauma to the renal arteries all contribute to renal dysfunction.

Mannitol, loop diuretics, and dopamine are used clinically to preserve renal function during aortic surgery. Significant controversy exists regarding the use of these drugs, as well as the mechanisms by which they may offer a protective effect. Although not proved, pharmacologic “protection” before aortic cross-clamping is believed to be beneficial and is therefore given. The use of mannitol 12.5 g/70 kg to induce osmotic diuresis before aortic cross-clamping is ubiquitous in clinical practice. Mannitol improves renal cortical blood flow during infrarenal aortic cross-clamping and reduces ischemia-induced renal vascular endothelial cell edema and vascular congestion. Other mechanisms by which mannitol may be beneficial include acting as a scavenger of free radicals, decreasing renin secretion, and increasing renal prostaglandin synthesis. Loop diuretics and low-dose dopamine (1 to 3 µg/kg/min) are used to

protect the kidneys from aortic cross-clamp-induced injury by increasing renal blood flow and urine output intraoperatively. Routine use of these drugs is common for patients with preoperative renal insufficiency and for procedures requiring suprarenal aortic cross-clamping. Intraoperative use of these drugs requires increased surveillance of intravascular volume and electrolytes during the postoperative period. Therapy with these drugs could actually be harmful because of hypovolemia and resultant renal hypoperfusion. In addition, dopamine's positive inotropic and chronotropic activity may cause tachycardia and increase myocardial O₂ consumption in patients with limited coronary reserve.

Fenoldopam mesylate, a selective dopamine type 1 agonist that preferentially dilates the renal and splanchnic vascular beds, has shown some promise as a renoprotective drug. However, its role in the prevention of renal dysfunction after aortic surgery is not known. Statin use is associated with preserved renal function after aortic surgery requiring suprarenal aortic cross-clamping.⁶³ Remote ischemic preconditioning reduces the incidence of renal impairment after open aortic surgery.⁶⁴ Optimal systemic hemodynamics, including maintenance of intravascular volume and hematocrit, is generally considered the most effective means of renal protection during and after aortic cross-clamping. The goal is to achieve a preload adequate to allow the left ventricle to cope with cross-clamping-induced changes in contractility and afterload while maintaining cardiac output. However, in providing such therapy, excessive intravascular volume should be avoided because it may lead to inappropriate increases in preload or pulmonary edema in patients with decreased myocardial reserve.

THERAPEUTIC STRATEGIES

Patients with preexisting impaired ventricular function and reduced coronary reserve are most vulnerable to the stress imposed on the cardiovascular system by aortic cross-clamping. Rational therapeutic strategies to prevent the deleterious effect of aortic cross-clamping primarily include measures to reduce afterload and maintain a normal preload and cardiac output. Vasodilators, positive and negative inotropic drugs, and controlled intravascular volume depletion (i.e., phlebotomy) may be used selectively.

Patients with impaired ventricular function requiring supraceliac aortic cross-clamping are the most challenging. Myocardial ischemia, reflecting an unfavorable balance between myocardial O₂ supply and demand, may result from the hemodynamic consequences of aortic cross-clamping. Controlled (i.e., slow clamp application) supraceliac aortic cross-clamping is important to avoid abrupt and extreme stress on the heart. Both afterload and preload reduction are often required. Afterload reduction, most commonly accomplished with the use of sodium nitroprusside or clevidipine (predominantly arteriolar dilators), is necessary to unload the heart and reduce ventricular wall tension. In a large series of patients requiring cross-clamping of the descending thoracic aorta, stable left ventricular function was maintained with sodium nitroprusside during cross-clamping. Sodium nitroprusside most likely allowed adequate intravascular volume before unclamping, which resulted in stable unclamping hemodynamics. A normal preload is equally important and involves careful IV fluid

BOX 56.3 Physiologic Changes With Aortic Unclamping* and Therapeutic Intervention

Hemodynamic Changes

- ↓ Myocardial contractility
- ↓ Arterial blood pressure
- ↑ Pulmonary artery pressure
- ↓ Central venous pressure
- ↓ Venous return
- ↓ Cardiac output

Metabolic Changes

- ↑ Total body oxygen consumption
- ↑ Lactate
- ↓ Mixed venous oxygen saturation
- ↑ Prostaglandins
- ↑ Activated complement
- ↑ Myocardial-depressant factor(s)
- ↓ Temperature
- Metabolic acidosis

Therapeutic Interventions

- ↓ Inhaled anesthetics
- ↓ Vasodilators
- ↑ Fluid administration
- ↑ Vasoconstrictor drugs
- Reapply cross-clamp for severe hypotension
- Consider mannitol
- Consider sodium bicarbonate

*These changes are of greater significance with longer duration of cross-clamping and with more proximal cross-clamping.

titration and vasodilator administration. Nitroglycerin can be used because it increases venous capacity more than does sodium nitroprusside.

In patients without evidence of left ventricular decompensation or myocardial ischemia during supraceliac aortic cross-clamping, a proximal aortic mean arterial pressure of up to 120 mm Hg is acceptable. The surgeon may request lower proximal arterial pressure if friable aortic tissue is encountered. Blood flow below the aortic clamp depends on pressure and decreases further during therapy with vasodilators. In this setting, vital organs and tissues distal to the clamp are exposed to reduced perfusion pressure and blood flow. Though infrequent, maintenance of adequate cardiac output may require active intervention with inotropic drugs.

AORTIC UNCLAMPING

The hemodynamic and metabolic effects of aortic unclamping are listed in **Box 56.3**. The hemodynamic response to unclamping depends on many factors, including the level of aortic occlusion, total occlusion time, use of diverting support, and intravascular volume. Hypotension, the most consistent hemodynamic response to aortic unclamping, can be profound, particularly after removal of a supraceliac cross-clamp (**Fig. 56.4**). Reactive hyperemia in tissues and organs distal to the clamp and the resultant relative central hypovolemia are the dominant mechanisms of the hypotension. Washout of vasoactive and cardiodepressant mediators from ischemic tissues, as well as humoral factors,

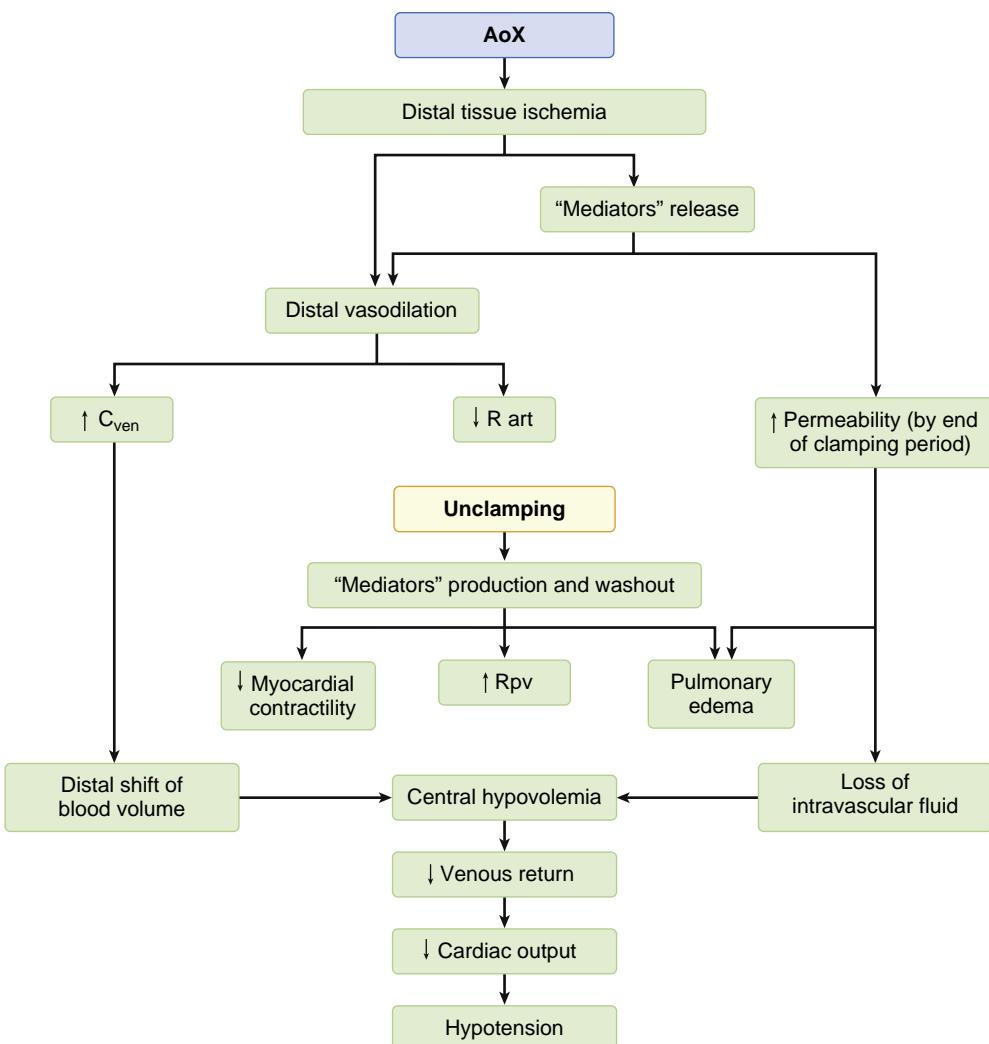


Fig. 56.4 Systemic hemodynamic response to aortic unclamping. AoX, Aortic cross-clamping; C_{ven} , venous capacitance; R_{art} , arterial resistance; R_{pv} , pulmonary vascular resistance.

may also contribute to the hemodynamic responses after unclamping the aorta. These humoral factors and mediators, which may also play a role in organ dysfunction after aortic occlusion, include lactic acid, renin-angiotensin, O₂ free radicals, prostaglandins, neutrophils, activated complement, cytokines, and myocardial-depressant factors.⁵⁴

Avoidance of significant hypotension with unclamping requires close communication with the surgical team, awareness of the technical aspect of the surgical procedure, and appropriate administration of fluids and vasoactive drugs. It is essential that correction of preoperative fluid deficits, maintenance of intraoperative fluid requirements, and replacement of blood loss be accomplished before unclamping. Vasodilators, if used, should be gradually reduced and discontinued before unclamping. The inspired concentrations of volatile anesthetics should be decreased. Moderate augmenting of intravascular volume by administration of fluids (~500 mL) during the immediate prerelease period is indicated for infrarenal unclamping. More aggressive intravascular fluid administration is required in the period immediately preceding supraceliac unclamping. Maintaining increased central venous or pulmonary capillary wedge pressure during the cross-clamp period is not indicated

and may result in significant overtransfusion of fluids and blood products. If significant hypotension results, gradual release of the aortic clamp and reapplication or digital compression are important measures in maintaining hemodynamic stability during unclamping. Although vasopressor requirements are minimal after release of the infrarenal clamp, significant support is often needed after the removal of supraceliac clamps. Caution must be observed when vasopressor support is used in this setting because profound proximal hypertension may occur if reapplication of the cross-clamp is required above the celiac axis. In addition, hypertension should be avoided to prevent damage to or bleeding from the vascular anastomoses.

ANESTHETIC MANAGEMENT

Intraoperative Monitoring

The potential for significant and rapid blood loss cannot be underestimated. A central line and two peripheral lines are usually used as intravenous (IV) access. The choice of the type and the size of the central line can be decided on a case-by-case basis. Placement of an arterial catheter should be routine in all patients undergoing aortic reconstruction.

As with other vascular procedures, the radial artery is most commonly selected for cannulation because of its superficial location, easy accessibility, and low complication rate. A noninvasive blood pressure cuff should be placed on the arm contralateral to the arterial catheter in the event of catheter malfunction.

A central venous catheter should be used for all open aortic procedures. It allows monitoring of CVP and administration of drugs directly into the central circulation. The routine, nonselective use of pulmonary artery catheter monitoring is not recommended. It should be reserved for patients with severely limited cardiopulmonary function or complex aortic reconstruction. In patients with good left ventricular and pulmonary function, CVP correlates well with left ventricular filling pressure. The invasive monitoring catheters can be placed before or after induction of general anesthesia. The advantage of preinduction catheter placement is assessment of the patient's awake (i.e., baseline) cardiovascular status, which allows correction of significant abnormalities in cardiac filling and function before induction.

With selective use, accurate interpretation of data, and rational treatment strategies, pulmonary artery catheter monitoring may be beneficial in high-risk patients undergoing complex aortic reconstruction. Yet the clinical value of pulmonary artery catheter monitoring in high-risk patients has not been established.⁶⁵ Clinical studies over the last 2 decades have yielded quite conflicting and variable results, including both increases and decreases in mortality. The National Heart, Lung and Blood Institute and the Food and Drug Administration (FDA)⁶⁶ sponsored a large prospective, randomized trial that compared goal-directed therapy guided by a pulmonary artery catheter with standard care without the use of a pulmonary artery catheter in high-risk surgical patients. The result found no benefit in treatment guided by a pulmonary artery catheter.⁶⁷ Yet this study did not find an increase in mortality with insertion and use of a pulmonary artery catheter.

Transesophageal echocardiography (TEE) has been used intraoperatively to assess global ventricular function, guide intravascular fluid therapy, and monitor for myocardial ischemia. Patients requiring supraceliac aortic cross-clamping have significant increases in the end-diastolic area and significant decreases in ejection fraction on echocardiography that are not completely normalized with vasodilators and frequently are not detected by pulmonary artery catheter monitoring.⁵⁶

The optimal intraoperative monitoring techniques for patients undergoing abdominal aortic reconstruction have not been established. Existing clinical studies offer insufficient data to conclusively answer the question of whether pulmonary artery catheter or TEE monitoring improves outcome. The clinical usefulness of any monitoring technique ultimately depends on patient selection, accurate interpretation of data, and appropriate therapeutic intervention.

Cell Salvage

Intraoperative cell salvage is a widely used technique combined with allogenic blood transfusion and in some centers is considered routine. The equipment is expensive and requires significant training and expertise. An early, non-randomized study reported a 75% reduction in the number

of allogeneic red blood cell (RBC) units transfused during elective aortic surgery with the use of cell salvage. Later randomized studies have reported conflicting results. The routine use of cell salvage during aortic surgery may not be cost-effective and thus it may best be reserved for a select group of patients with an expected large blood loss. A cost-effective option is to use the cell salvage reservoir for blood collection and activate the full salvage process only if large blood loss occurs.

Anesthetic Drugs and Techniques

Various anesthetic techniques, including general anesthesia, regional (epidural) anesthesia, and combined techniques, have been used successfully for abdominal aortic reconstruction. Combined techniques most commonly use a lumbar or low thoracic epidural catheter in addition to general anesthetic. Local anesthetics, opioids, or, more commonly, a combination of the two may be administered by bolus or continuous epidural infusion. Maintenance of vital organ perfusion and function by the provision of stable perioperative hemodynamics is more important to overall outcome than is the choice of anesthetic drug or technique.¹⁴ Therefore, the specific anesthetic technique for patients undergoing abdominal aortic reconstruction is important insofar as it allows rapid and precise control of hemodynamic parameters. Given the frequent incidence of cardiac morbidity and mortality in patients undergoing aortic reconstruction, factors that influence ventricular work and myocardial perfusion are of prime importance. Induction of general anesthesia should ensure that stable hemodynamics are maintained during loss of consciousness, laryngoscopy and endotracheal intubation, and the immediate postinduction period. A variety of IV anesthetics (propofol, etomidate, thiopental) are suitable. The addition of a short-acting, potent opioid such as fentanyl or sufentanil usually provides stable hemodynamics during and after induction of anesthesia. Volatile anesthetics may be administered in low concentrations before endotracheal intubation during assisted ventilation as an adjunct to blunt the hyperdynamic response to laryngoscopy and endotracheal intubation. Esmolol 10 to 25 mg, sodium nitroprusside 5 to 25 µg, nitroglycerin 50 to 100 µg, or clevipidine 100 mcg and phenylephrine 50 to 100 µg should be available for bolus administration during induction if needed to maintain appropriate hemodynamics.

Maintenance of anesthesia may be accomplished with a combination of a potent opioid (fentanyl or sufentanil) and an inhaled anesthetic (sevoflurane, desflurane, or isoflurane) (i.e., balanced anesthesia). Patients with severe left ventricular dysfunction may benefit from a pure opioid technique, but a balanced anesthetic technique allows the clinician to take advantage of the most desirable characteristics of potent opioids and inhaled volatile anesthetics while minimizing their undesirable side effects. Nitrous oxide can be used to supplement either an opioid or an inhaled anesthetic.

Various regional anesthetic and analgesic techniques have been used effectively during and after aortic reconstruction. For over 2 decades, interest has focused on the use of regional anesthetic and analgesic techniques to reduce the incidence of perioperative morbidity in patients undergoing aortic reconstruction. The benefits of combined

general and epidural anesthesia intraoperatively, with or without epidural analgesia continued into the postoperative period, remain controversial.^{13,14,68-71} Moreover, studies that have reported improved outcome do not determine whether the benefit results from the intraoperative anesthetic technique or the postoperative analgesic regimen (or a combination of the two). In a randomized trial using epidural morphine in patients undergoing aortic surgery, Breslow and associates⁷² found attenuation of the adrenergic response and a less frequent incidence of hypertension in the postoperative period. A large randomized trial reported no reduction in nonsurgical complications with the use of intrathecal opioid.¹⁵ The effects of the anesthetic or analgesic technique on the incidence of perioperative myocardial ischemia have received considerable attention. Four randomized trials, with nearly 450 combined patients undergoing aortic reconstruction, failed to demonstrate a reduction in the incidence of perioperative,^{14,73} intraoperative,⁷⁴ or postoperative⁷⁰ myocardial ischemia when epidural techniques were used. Additionally, randomized trials have not demonstrated a reduction in the incidence of cardiovascular, pulmonary, or renal complications after aortic surgery with the use of epidural techniques.^{13,14,69,70,75}

The duration and intensity of postoperative care after aortic surgery are critically dependent on the physiologic derangements incurred during the perioperative period (i.e., depression of consciousness, hypothermia, excessive intravascular fluids, incisional pain, ileus, and respiratory depression), as well as on the development of certain less common, but more severe postoperative complications (i.e., MI, pneumonia, sepsis, renal failure, and decreased tissue perfusion). Length of hospital stay may therefore be considered the outcome variable most directly proportional to an integrated final negative effect of all significant perioperative morbidity (excluding in-hospital death) and the variable most likely to be altered by the anesthetic or analgesic technique. Randomized trials have not demonstrated any reduction in length of hospital stay after aortic surgery with the use of regional techniques. Norris and colleagues¹⁴ reported the results of a randomized clinical trial comparing alternative combinations of intraoperative anesthesia (i.e., general or combined epidural and general) and postoperative analgesia (i.e., IV patient-controlled analgesia [PCA] or epidural PCA) with respect to length of stay after abdominal aortic surgery. Two unique features of the trial included a factorial design (Fig. 56.5), which allowed the inclusion of all four combinations of intraoperative anesthesia and postoperative analgesia and the ability to separate the influence of time period and technique, and a double-blind design, which helped eliminate investigator and treating physician bias. The study rigorously protocolized perioperative management, standardized postoperative surgical care, and optimized postoperative pain management. Although the overall length of stay was much shorter (median, 7.0 days) than that reported in other studies,^{13,68,70,75} they were not able to demonstrate a reduction in length of stay or direct medical costs based on anesthetic or analgesic technique (Table 56.6). The overall incidence of postoperative complications in the trial was low and not different based on anesthetic or analgesic technique. Postoperative pain was well controlled overall, with similar pain scores in both analgesic treatment groups. Thus, if perioperative care and

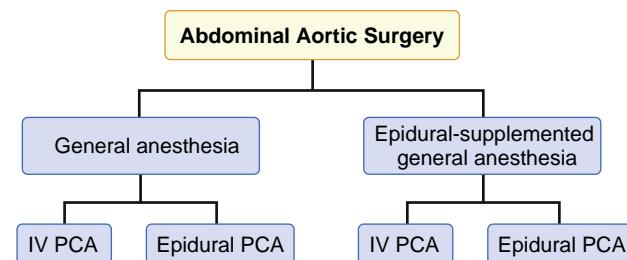


Fig. 56.5 Outline of factorial study design. This design allows the inclusion of all four possible combinations on intraoperative anesthesia and postoperative analgesia and the ability to separate the influences of time period and technique. Data analysis by treatment group, intraoperative treatment, postoperative treatment, and any epidural activation, as well as simultaneous consideration of both intraoperative and postoperative treatments in the same model (factorial analysis), is possible and allows improvement in outcome to be attributed to the intraoperative anesthesia, postoperative analgesia, the combination of the two, or to unrelated factors. IV, Intravenous; PCA, patient-controlled analgesia.

pain relief are optimized, epidural anesthetic and analgesic techniques for aortic surgery offer no major advantage or disadvantage over general anesthesia and IV PCA.

The use of epidural local anesthetics in combination with general anesthesia during aortic reconstruction poses several problems, including hypotension at the time of aortic unclamping and the need for increased intravascular fluid and vasopressor requirements. Supraceliac aortic cross-clamping may significantly exaggerate these disadvantages, and, as a result, some clinicians avoid running local anesthetics in the epidural around the period of aortic clamping and unclamping. Epidural opioids without local anesthetics can be used in the interim and local anesthetic can be given later, after aortic unclamping, when hemodynamics and intravascular volume have stabilized. Although elective aortic reconstruction via the retroperitoneal approach using straight epidural anesthesia (no general anesthetic) has been reported, this technique is not recommended for routine use.

Hypertension and tachycardia are aggressively controlled during emergence by the use of short-acting drugs such as esmolol, nitroglycerin, clivipine, or sodium nitroprusside. Emergence from anesthesia should be conducted after restoration of circulation and establishment of adequate organ perfusion. Hemodynamic, metabolic, and temperature homeostasis must be achieved before extubation; otherwise, patients should be transported intubated to the intensive care unit (ICU).

Temperature Control

Postoperative hypothermia is associated with many undesirable physiologic effects and may contribute to adverse outcomes (see also Chapter 80). Normothermia should be maintained before skin incision by increasing ambient temperature in the operating room, applying warm cotton blankets, and warming IV fluids. If significant hypothermia occurs early in the procedure, normothermia is extremely difficult to achieve, and emergence and tracheal extubation may be delayed. During surgery, all fluids and blood products should be warmed before administration. A forced-air warming blanket should be applied over the upper part of the body. The lower part of the body should not be warmed

TABLE 56.6 Duration of Hospital Stay and Direct Medical Costs by Randomized Treatment Assignment for Patients Surviving to Discharge after Abdominal Aortic Surgery

	GA-IVPCA	RSGA-IVPCA	GA-EPCA	RSGA-EPCA	Total	P-value
No. of Patients	35	36	36	44	151	
Duration of hospital stay (days)*	7.0 (2.2)	8.0 (2.8)	7.0 (2.0)	7.0 (2.8)	7.0 (2.2)	.833†
Range	4–43	5–28	5–20	5–18	4–43	
95% CI	7.0–13.3	7.4–10.2	6.9–8.8	7.6–9.6	7.9–9.7	
Direct medical costs (US\$ 1997)*						
Inpatient	12,413 (2867)	13,786 (4413)	12,492 (3111)	13,767 (3900)	12,793 (3777)	.242
Physician	10,394 (5993)	10,288 (4538)	9609 (3866)	9790 (3567)	9934 (4072)	.459
Total	22,674 (8783)	23,001 (6079)	22,182 (3914)	22,727 (3961)	22,674 (4903)	.851

CI, Confidence interval; GA-EPCA, general anesthesia and epidural patient-controlled analgesia; GA-IVPCA, general anesthesia and intravenous patient-controlled analgesia; RSGA-EPCA, regional supplemented general anesthesia and epidural patient-controlled analgesia; RSGA-IVPCA, regional supplemented general anesthesia and intravenous patient-controlled analgesia.

From Norris EJ, Beattie C, Perler B, et al. Double-masked randomized trial comparing alternate combinations of intraoperative anesthesia and postoperative analgesia in abdominal aortic surgery. *Anesthesiology*. 2001;95:1054–1067.

during the cross-clamp period because doing so can increase injury to ischemic tissue distal to the cross-clamp by increasing metabolic demands.

THORACOABDOMINAL AORTIC SURGERY

Open repair of the thoracoabdominal aorta is widely regarded as the most challenging surgical procedure in terms of overall anesthetic and perioperative management. Surgical repair is required for a spectrum of disease, including degenerative aneurysm, acute and chronic dissection, intramural hematoma, mycotic aneurysm, pseudoaneurysm, penetrating aortic ulcer, coarctation, and traumatic aortic tear. Since the first thoracoabdominal aortic aneurysm (TAA) repair in 1955, tremendous advances have been made in the field. These advances have led to significant reductions in operative mortality and perioperative complications. However, even in centers where numerous procedures are performed, morbidity and mortality are frequent, especially in patients with dissecting or ruptured aneurysms. To successfully care for these patients, the anesthesiologist must be knowledgeable in the areas of one-lung ventilation; extracorporeal circulatory support, including circulatory arrest; renal and spinal cord protection; induced hypothermia; invasive hemodynamic monitoring, including TEE; massive transfusion; and management of coagulopathy. Intraoperative management requires a team effort with intimate cooperation among surgeons, anesthesiologists, perfusionists, nurses, and electrophysiologic monitoring staff. Endovascular stent-graft repair of lesions that affect the descending thoracic and thoracoabdominal aorta is evolving rapidly. As discussed later, accumulating experience with stent-graft repair of thoracic aortic aneurysm, dissection, and traumatic tear has demonstrated this modality to be an effective alternative to open repair for select patients.

ETIOLOGY AND CLASSIFICATION

Aneurysms of the thoracoabdominal aorta occur primarily because of atherosclerotic degenerative disease (80%) and chronic aortic dissection (17%).⁷⁶ The remainder are caused by either trauma or connective tissue diseases involving the aortic wall from conditions such as Marfan syndrome, cystic medial degeneration, Takayasu arteritis, or syphilitic aortitis. The true incidence of TAA is unknown, but population

studies suggest a prevalence much less than that of infrarenal AAA. Degenerative and dissecting TAAs differ in their associated risk factors, extent of aortic involvement, and natural history. Thus complete characterization of each TAA is required to formulate a comprehensive treatment plan. Development of both degenerative and dissecting TAAs is ultimately related to weakening of the aortic wall. Although the natural history of TAA without surgery is uncertain, enlargement tends to be progressive and nonoperative management is generally associated with a poor prognosis. With progressive enlargement, nutritional blood flow to the aorta is compromised. The increasing diameter is associated with increased wall tension, even when arterial pressure is constant (law of Laplace). The frequent incidence of associated systemic hypertension enhances aneurysm enlargement.

Degenerative and dissecting TAAs are symptomatic at initial evaluation in 57% and 85% of patients, respectively. The most common initial complaint is back pain. Additional symptoms can be caused by compression of organs or structures adjacent to the aneurysm. Aortic rupture, as a manifestation of TAA, occurs with equal frequency (9%) in both degenerative and dissecting aneurysms. Rupture of the thoracic and abdominal segments occurs with equal frequency and primarily in patients with aneurysms larger than 5 cm. Surgical repair is usually recommended when aneurysm diameter exceeds 6 cm, but earlier repair may be offered to patients with Marfan syndrome and those with a strong family history of an aortic aneurysm.

In addition to cause, aneurysms of the thoracoabdominal aorta may be classified according to their anatomic location. In 1986, Crawford and colleagues,⁷⁶ recognizing the correlation between aneurysm extent and clinical outcome, proposed a classification based on the extent of aortic involvement (Fig. 56.6). The Crawford classification defines aneurysms as types I, II, III, and IV and is appropriately applied to aneurysms of all causes (degenerative and dissecting). Type I aneurysms involve all or most of the descending thoracic aorta and the upper abdominal aorta. Type II aneurysms involve all or most of the descending thoracic aorta and all or most of the abdominal aorta. Type III aneurysms involve the lower portion of the descending thoracic aorta and most of the abdominal aorta. Type IV aneurysms involve all or most of the abdominal aorta, including the visceral segment. Types II and III are the most difficult to repair because they involve

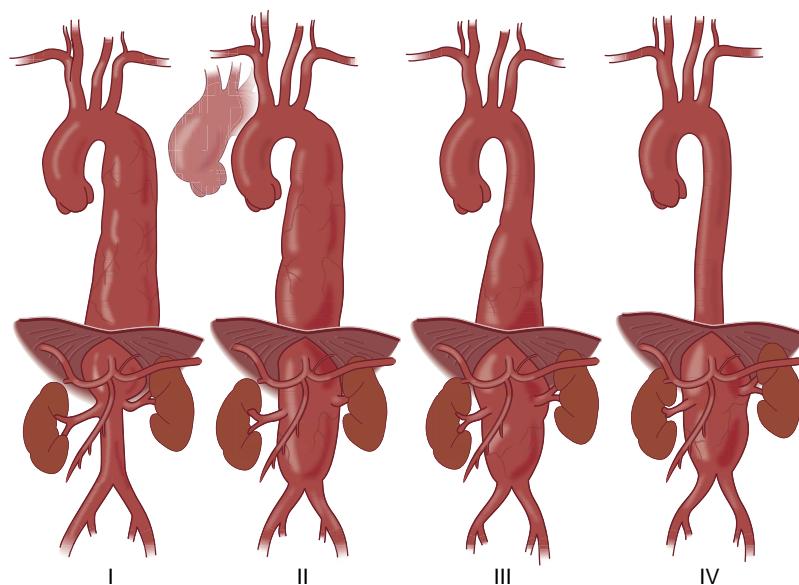


Fig. 56.6 The Crawford classification of thoracoabdominal aortic aneurysms is defined by anatomic location and the extent of involvement. *Type I* aneurysms involve all or most of the descending thoracic aorta and the upper abdominal aorta; *type II* aneurysms involve all or most of the descending thoracic aorta and all or most of the abdominal aorta; *type III* aneurysms involve the lower portion of the descending thoracic aorta and most of the abdominal aorta; and *type IV* aneurysms involve all or most of the abdominal aorta, including the visceral segment.

both the thoracic and the abdominal segments of the aorta. Patients with Crawford type II aneurysms are at greatest risk for paraplegia and renal failure from spinal cord and kidney ischemia during cross-clamping. Even with extracorporeal circulatory support, an obligatory period occurs when blood flow to these organs is interrupted because the origin of the blood flow is between the cross-clamps. For this reason, protective measures to prevent ischemic injury are important in reducing morbidity.

Aortic dissection, with or without aneurysm formation, has likewise been classified according to the extent of aortic involvement. The most widely used classification, proposed by DeBakey and colleagues, defines aortic dissection as types I, II, and III (Fig. 56.7).

Type I aneurysms begin in the ascending aorta and extend throughout the entire aorta. These lesions are usually repaired via a two-stage approach, with the first procedure on the ascending aorta and aortic arch and the second procedure on the descending thoracic aorta. Type II aneurysms are confined to the ascending aorta. Both types I and II often involve the aortic valve and cause aortic regurgitation, and sometimes they involve the ostia of the coronary arteries. Type III aneurysms begin just distal to the left subclavian artery (SCA) and extend either to the diaphragm (type IIIA) or to the aortoiliac bifurcation (type IIIB).

Another commonly used classification of aortic dissection is the Stanford classification. This more simplified classification divides aortic dissection into those that involve the ascending aorta (Stanford type A) and those that do not involve the ascending aorta (Stanford type B). Aortic dissection is also classified by duration, with those less than 2 weeks classified as acute and those greater than 2 weeks classified as chronic. This classification has very significant mortality implications, with much higher mortality in the acute phase.

Acute aortic dissection involving the ascending aorta (DeBakey types I and II, Stanford type A) is a surgical emergency that requires immediate cardiac surgical repair (see also Chapter 54). Acute dissections involving the descending aorta

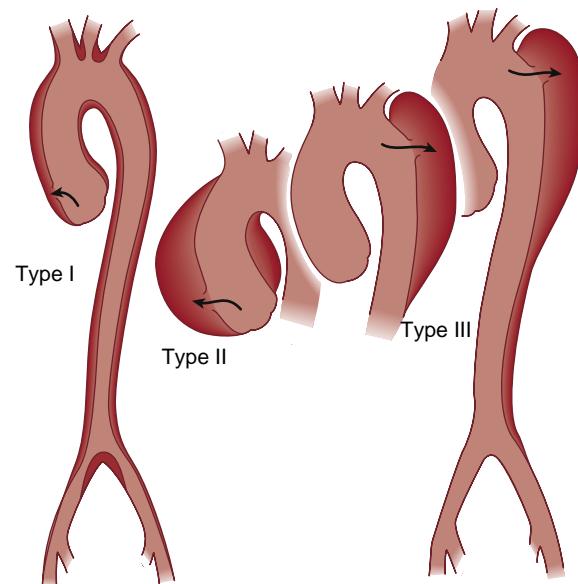


Fig. 56.7 The DeBakey classification of dissecting aneurysms of the aorta. *Type I* has an intimal tear in the ascending aorta with dissection extending down the entire aorta. *Type II* has an intimal tear in the ascending aorta with dissection limited to the ascending aorta. *Type III* has an intimal tear in the proximal descending thoracic aorta with dissection either limited to the thoracic aorta (type IIIA) or extending distally to the abdominal aorta or aortoiliac bifurcation (type IIIB).

(DeBakey type III, Stanford type B) are most often treated conservatively (i.e., arterial blood pressure, heart rate, and pain control) because surgical repair has no proved benefit over medical or interventional treatment in stable patients. Early surgical intervention may be required for a variety of reasons, including aneurysmal formation, impending rupture, organ or leg ischemia, and inadequate response to medical therapy. In approximately 20% to 40% of patients with chronic aortic dissection, significant aneurysmal dilatation of the descending thoracic or thoracoabdominal aorta will develop.

MORBIDITY AND MORTALITY

Despite tremendous development in surgical and anesthetic technique, mortality and complication rates remain frequent for open surgical repair of TAA. Patients who undergo replacement of the entire thoracoabdominal aorta (Crawford extent type II) have the most frequent perioperative risk. Contemporary mortality rates reported from large institutions range from 5% to 14%. Statewide and nationwide mortality rates may be considerably more frequent (~20%). The perioperative mortality rate may significantly underestimate the risk associated with TAA repair. In a large statewide series, the mortality with elective TAA repair was 19% at 30 days and 31% at 365 days.⁷⁷

The incidence of paraplegia or paraparesis in patients undergoing surgical repair of TAA is reported to be 3.8% to 40%, depending on complex factors such as anatomic location, the duration of cross-clamping, the use of protective measures, the degree of dissection, and whether the aneurysm has ruptured. Extensive dissecting TAA repair carries the highest risk for neurologic deficit. A contemporary report of 210 consecutive open TAA repairs reported three patients with paraplegia and two with temporary paraparesis for an overall rate of neurologic deficit of 2.4% (1.4% permanent).⁷⁸ Renal failure occurs in 3% to 30% of patients, depending on similar factors noted earlier. Overall, approximately 6% of patients need postoperative dialysis after TAA repair, which is associated with high mortality (30% to 60%). Gastrointestinal complications occur in approximately 7% of patients and are associated with a mortality approaching 40%. Not surprisingly, pulmonary complications are the most common problem associated with TAA repair. The incidence of postoperative pulmonary insufficiency approaches 50%, with 8% to 14% of patients requiring tracheostomy. As with all other vascular surgical procedures, cardiac complications are common and a leading cause of perioperative mortality. In general, perioperative mortality and major complication rates after open isolated descending thoracic aortic replacement are lower than those described for TAA repair.

PREOPERATIVE PREPARATION AND MONITORING

Open surgical TAA repair requires extensive preoperative evaluation and planning. The evaluation and management of coexisting cardiac and pulmonary disease are discussed earlier. Before the day of surgery, the anesthesiologist and vascular surgeon should discuss, at a minimum, extent of the aneurysm and technique of surgical repair, plans for distal aortic perfusion, monitoring for spinal cord ischemia (SCI), renal and spinal cord protection, hemodynamic monitoring, and ventilation strategy. Depending on the extent and the location of the aortic disease in the descending aorta, the decision to use left heart bypass or not is usually made.

Blood loss during TAA repair can be profound, and the need for massive transfusion must not be underestimated. This author routinely has 10 units of packed RBCs, 10 units of thawed fresh frozen plasma, and several units of platelets immediately available in the operating room, and additional units must be readily obtainable.

Large-bore IV access is obviously important, especially if partial bypass (in contrast to full bypass) is planned, because it is difficult or impossible for the perfusionist to administer fluid or blood products into the closed partial bypass circuit. Large-sized central venous catheters with introducer sheaths for pulmonary artery catheters and large bore peripheral IVs are recommended. Rapid infuser systems are used to allow the delivery of up to 1500 mL/min of blood products at a temperature of 37°C to 38°C. A right radial arterial catheter is used for aneurysms involving the proximal descending thoracic aorta because occasionally the cross-clamp is placed proximal to the left subclavian artery, thus occluding flow to the left upper extremity. When distal aortic perfusion techniques are used, arterial blood pressure distal to the cross-clamps can be monitored. This can be accomplished with the placement of a right femoral arterial catheter, or the surgical team can place a catheter directly into the femoral artery or distal aorta. This catheter monitors perfusion pressure to the kidneys, spinal cord, and mesenteric circulation during the time when the cross-clamps are high on the descending aorta and the lower body region is perfused by a shunt or bypass circuit. Radial and femoral arterial pressure should be simultaneously displayed on the anesthesiologist's monitor and a monitor visible to the surgeons and the perfusionists. TEE is used routinely during TAA repair (see also [Chapter 37](#)). When TEE is used by a properly trained individual, assessment of left ventricular end-diastolic volume, myocardial ischemia, and valvular function is possible. It is also possible to determine the size and extent of the aneurysm.

A double-lumen endobronchial tube or a bronchial blocker should be inserted for the purpose of one-lung ventilation (see also [Chapter 53](#)). One-lung ventilation provides optimal visualization of the surgical field and reduces retraction-related trauma to the left lung. The double-lumen tube is usually changed, if possible, to a single-lumen tube at the completion of surgery. This facilitates ICU management of pulmonary hygiene and reduces resistance to breathing during weaning in the postoperative period. Many centers use electrophysiologic monitoring with somatosensory evoked potentials (SSEPs) or motor evoked potentials (MEPs) to monitor for SCI (see also [Chapter 39](#)). These monitoring techniques may be helpful in both identifying the important intercostal arteries that perfuse the spinal cord and confirming successful reimplantation into the aortic graft. If SCI is identified, cross-clamps can often be repositioned, upper or lower body blood pressure can be increased to augment perfusion through collateral channels, or other measures may be taken to protect the spinal cord (i.e., cerebrospinal fluid [CSF] drainage, induced hypothermia, or intrathecal pharmacologic agents). These techniques are discussed later. Three general problems exist with SSEP monitoring when used during TAA repair. First, sensory monitoring is more likely to detect lateral and posterior sensory column ischemia and is a poor monitor for the anterior motor column. As a result, paraplegia can occur despite normal SSEP signals. Second, inhaled anesthetics and hypothermia can significantly interfere with SSEP signals. Third, ischemia affects peripheral nerves, and ischemia in the lower extremities delays conduction from the usual stimulation sites (e.g., posterior tibial nerve). To eliminate the

peripheral nerves as a confounding factor, spinal stimulation via a lumbar epidural electrode can be used, which may be more specific for ischemic injury than peripheral monitoring alone. Lower extremity and peripheral nerve ischemia can be avoided with the use of distal aortic perfusion techniques. To avoid lower extremity ischemia from occlusion of the left femoral artery at the insertion site of the retrograde perfusion cannula, some surgeons suture a small-caliber graft onto the femoral artery (end to side) for insertion of the cannula, which allows both antegrade and retrograde perfusion. These limitations of SSEP monitoring probably account for the lack of identification of critical spinal arteries. The transcranial MEP technique has been used successfully to monitor the anterior columns of the spinal cord. The technique is relatively simple and can be viewed as a “train-of-four” for the brain and spinal cord. Electrical stimulation over the motor cortex activates α -motor neurons, and evoked electromyographic responses are obtained in lower extremity muscle. Only electromyogenic responses are specific for the status of the motor neurons in the anterior horn gray matter. Bilateral recording needles should be placed in the popliteal fossae (i.e., popliteal nerve) and bilateral surface electrodes over the gastrocnemius and tibialis anterior muscles. Bilateral stimulating needles are routinely placed in the popliteal fossae to monitor direct muscle responses and the level of neuromuscular blockade. During aortic cross-clamping, MEPs are monitored every minute. A reduction in MEP amplitude to less than 25% of baseline is considered an indication of SCI and requires corrective measures. Because signal averaging is not required and the anterior horn cells react with an almost immediate functional loss after the onset of ischemia, the technique can be used to rapidly identify intercostal arteries supplying the spinal cord. Additionally, the technique can be used to evaluate the adequacy of distal aortic perfusion and the patency of reimplanted critical intercostal arteries. Careful titration of a short-acting neuromuscular blocker is required to maintain a stable level of neuromuscular blockade. Complete neuromuscular blockade makes MEP monitoring impossible. Isoflurane, desflurane, sevoflurane, and N2O depress synaptic conduction and significantly decrease the amplitude of myogenic MEPs. Although modifications of the stimulating technique have somewhat improved monitoring with inhaled anesthetics, a total IV anesthetic technique may be optimal. Fentanyl and ketamine have little effect on myogenic MEPs and have been used successfully as a combined anesthetic in a large series of patients undergoing TAA repair with MEP monitoring.⁷⁸ This series of 210 consecutive patients had the lowest rate of neurologic deficit (2.4%) and permanent paraplegia (1.4%) reported.⁷⁸ Body temperature should be monitored at two sites (core and peripheral) to assess cooling and warming when bypass techniques are used. However, an important difference exists between full and partial bypass in temperature monitoring. With full bypass, perfusion is usually into the ascending aorta, and typically the upper body core temperature (i.e., nasopharynx or esophagus) cools and warms fastest, whereas the lower body temperature changes more slowly. With partial bypass, the opposite is true. The blood from bypass is returned into the femoral artery, and the lower part of the body (i.e., rectum

or bladder) changes before the upper part changes. This difference is important to recognize to achieve complete cooling and warming because the lagging temperature should be the end point for cooling and warming.

ANESTHETIC MANAGEMENT

Simple Aortic Cross Clamping

Descending thoracic and thoracoabdominal aortic surgery can be performed without extracorporeal support (i.e., left heart bypass or cardiopulmonary bypass). The “clamp-and-sew” technique has had relatively favorable outcomes, but these cases are from institutions with extensive clinical experience and the shortest cross-clamp times. Advocates of this technique favor its surgical simplicity. However, the benefits of avoiding the complexity and complications of bypass must be weighed against the risk for vital organ ischemia and complications such as renal failure and paraplegia. Other than the location and extent of the aneurysm, the duration of cross-clamping on the aorta is the single most important determinant of paraplegia and renal failure with the clamp-and-sew technique. With longer clamp times, specific adjuncts directed against end-organ ischemic complications have been described in literature. Such adjuncts include epidural cooling for spinal cord protection, regional hypothermia for renal protection, and in-line mesenteric shunting to reduce visceral ischemia, although they lack enough clinical evidence to support any of them.

When the simple clamp-and-sew technique is used, the application of the aortic cross-clamp results in significant proximal hypertension, which requires active pharmacologic intervention. Management strategies have been discussed previously in the section on abdominal aortic reconstruction.

Left Heart Bypass

Maintaining lower body perfusion with the use of retrograde distal aortic perfusion reduces ischemic injury and improves outcome, provided the pressure is high enough to perfuse the organs. The simplest method of providing distal aortic perfusion is a passive conduit or shunt. The heparin-bonded Gott shunt was developed to avoid the need for systemic heparinization and is used to divert flow passively from the left ventricle or proximal descending thoracic aorta to the distal aorta. Some centers place a temporary axillary-to-femoral artery graft to function as a shunt during aortic cross-clamping. Partial bypass, also referred to as left heart bypass or left atrial-to-femoral bypass, is the most commonly used distal aortic perfusion technique (Fig. 56.8). This technique allows adjustment of blood flow and usually draws blood from the left atrium and returns blood to the left femoral artery. A centrifugal pump is used (Biomedicus, Eden Prairie, MN), and full-dose systemic heparin is not needed because the circuit is coated with heparin. The typical heparin dose for partial bypass is 100 units/kg. With this technique, an oxygenator is unnecessary because only the left side of the heart is bypassed. Insertion of a heat exchanger into the circuit allows cooling and warming, which is beneficial but not essential. Variations of left heart bypass include cannulating the aortic arch or proximal descending thoracic aorta instead of the left atrium. With this circuit, the left ventricle is relieved of the increased afterload during

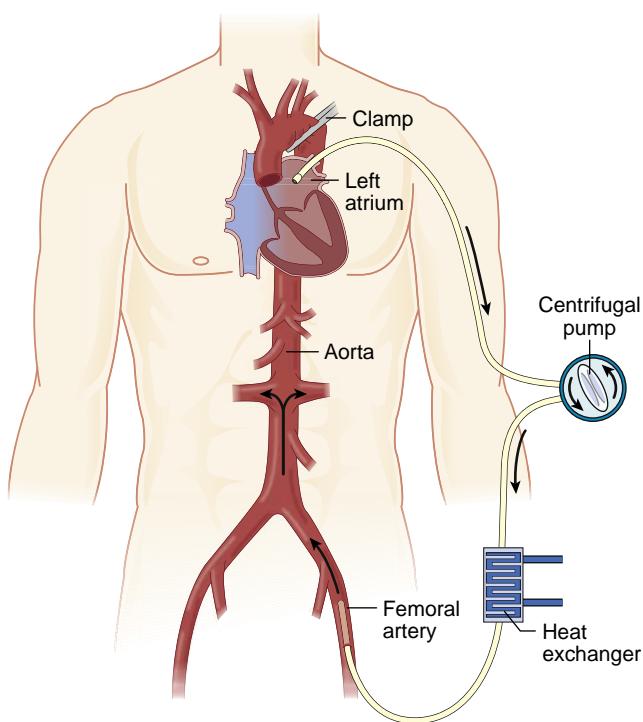


Fig. 56.8 Diagram of left atrial-femoral bypass. The left atrium and the left femoral artery are cannulated, and a centrifugal pump is used with heparin-coated tubing. A heat exchanger may be added into the circuit for cooling and rewarming.

aortic cross-clamping. With left atrial cannulation, the left ventricle is relieved of preload and cardiac output is reduced. Either way, proximal hypertension is controlled, the work of the ventricle is decreased, and perfusion is provided to the distal aorta. When hypothermia (30°C) is combined with atrial cannulation, approximately 15% of patients experience new atrial fibrillation. Although most patients revert to sinus rhythm on rewarming, direct cardioversion may be required.

During left heart bypass, it is essential that arterial blood pressure be monitored above and below the aortic cross-clamps. Careful control of intravascular volume, bypass pump flow, and vasoactive drugs is required to achieve the target blood pressures. Management of left heart bypass requires continuous communication and cooperation between the surgeon, anesthesiologist, and perfusionist. We typically set the initial pump flow to approximately 50% of the patient's cardiac output with application of the proximal aortic clamp. Flow is then adjusted to maintain target proximal and distal pressures. Administration of vasodilators is very infrequently required at this stage. With no vital organ ischemia, the surgeon can complete the proximal anastomoses in an unhurried fashion. With sequential aortic clamping, intercostal arteries can be reimplanted with minimal adjustments of pump flow. Pump flow is eventually reduced significantly during reimplantation of the visceral and renal arteries. At this point, distal perfusion is only to the lower extremities. Moderate hypothermia (32°C) during bypass is used to protect the vital organs during obligate periods of ischemia. After completion of the distal anastomoses, pump flow is increased, and the patient is actively warmed to 37°C.

Deep Hypothermic Circulatory Arrest

Complex aneurysms involving the aortic arch often require elective cardiopulmonary bypass with an interval of deep hypothermic (15°C) circulatory arrest (DHCA) because cerebral blood flow is transiently interrupted during surgery (see also [Chapter 54](#)). Bypass can be accomplished by cannulation of the femoral artery and the femoral vein (i.e., femoral-femoral bypass). During the interval of DHCA, some centers also use anterograde (i.e., innominate artery) or retrograde (i.e., internal jugular vein) selective cerebral perfusion with cold oxygenated blood to extend the safe maximum duration of circulatory arrest. Without this technique, 45 to 60 minutes is thought to be the safe limit of DHCA, but 90 minutes has been reported with selective cerebral perfusion. DHCA also may be necessary whenever the location, extent, or severity of aortic disease precludes placement of a proximal aortic clamp during thoracic or thoracoabdominal aortic repair. This is often the case in patients with previous aortic arch repair, in which adhesions and scarring make application of the proximal aortic cross-clamp difficult or impossible during TAA repair. DHCA eliminates the need for proximal aortic clamping and allows a bloodless field for the proximal aortic anastomosis. Some centers advocate the routine use of DHCA during complex aortic reconstruction because deep hypothermia may provide better end-organ and spinal cord function. This potential benefit must be carefully weighed against the risks associated with prolonged cardiopulmonary bypass and circulatory arrest. After completion of the proximal anastomosis and intercostal artery-to-graft anastomoses under DHCA, the aortic graft is cannulated and bypass flow is reestablished to the upper part of the body. During a period of hypothermic low bypass flow, the distal anastomoses are completed and then rewarming is initiated.

Anesthetic Technique

No single anesthetic technique is best for TAA repair. Usually, balanced anesthesia is provided with a combination of an opioid, a low-dose potent volatile anesthetic, a benzodiazepine, and a muscle relaxant. A total IV technique may be optimal if transcranial MEP monitoring is used. Induction of general anesthesia should be slow and controlled. Hypertension should be avoided because acute stress on the aneurysm can cause rupture. The heart rate should be maintained at or below baseline because myocardial ischemia is often related to the heart rate. Extubation should always take place in the ICU and only after a significant period of hemodynamic and metabolic stability. The postoperative analgesic regimen should focus on pain control and stable hemodynamics.

Spinal Cord Ischemia and Protection

Paraplegia is a devastating complication of aortic surgery. The incidence of paraplegia is reported to be 0.5% to 1.5% for coarctation repair, 0% to 10% for thoracic aneurysm repair, 10% to 20% for thoracoabdominal repair, and as high as 40% for extensive dissecting TAA repair. The spinal cord receives its blood supply from two posterior arteries ($\approx 25\%$) and one anterior spinal artery ($\approx 75\%$) ([Fig. 56.9](#)). The posterior spinal arteries, which supply the sensory tracts in the spinal cord, receive flow from the posterior and inferior cerebellar arteries, the vertebral arteries, and

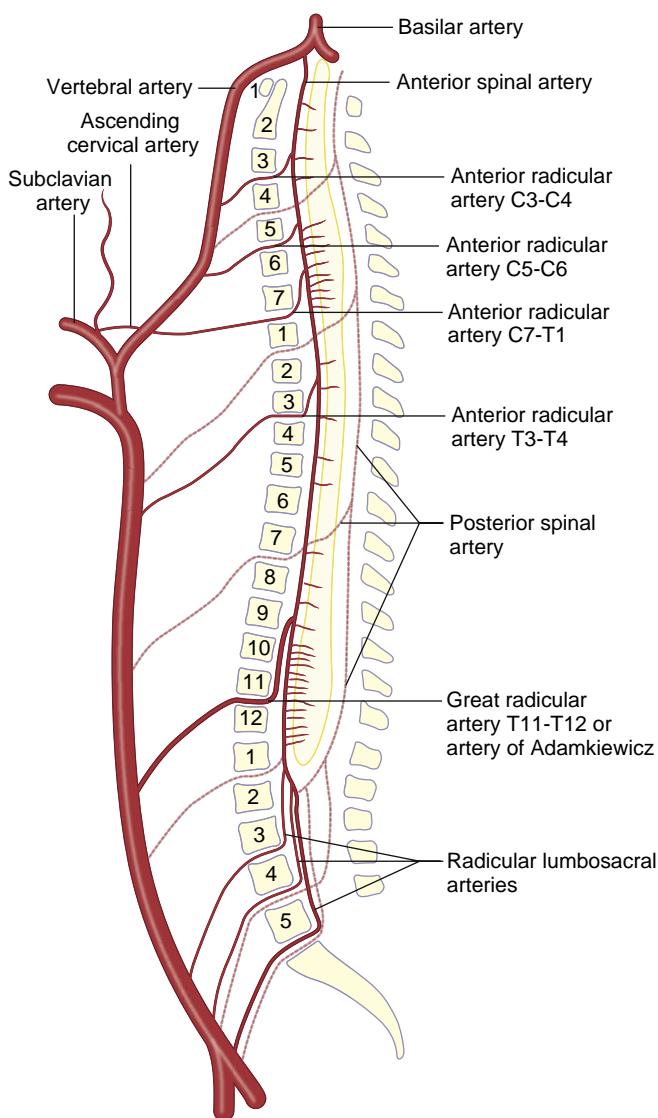


Fig. 56.9 Diagram of the blood supply to the spinal cord showing the anterior and posterior radiculomedullary branches seen in a lateral view. The primary blood supply to the thoracolumbar portion of the spinal cord is derived from the artery of Adamkiewicz; its origin varies but usually branches off the aorta in the T9 to T12 region.

the posterior radicular arteries. The anterior spinal artery, which supplies the motor tracts in the spinal cord, is formed by two branches of the intracranial portion of the vertebral arteries. The upper cervical segment of the spinal cord receives most of its blood flow from the vertebral arteries. The thoracic portion of the anterior spinal artery is supplied by the anterior radicular arteries (one or two cervical, two or three thoracic, and one or two lumbar). The largest of the radicular arteries is called the great radicular artery (GRA) or the artery of Adamkiewicz (AKA). The AKA is the major blood supply to the lower two thirds of the spinal cord. The segmental supplier of the AKA is variable (T5-L5) but is located between T9 and T12 in approximately 75% of cases. The variation in origin of the AKA explains why even infrarenal aortic aneurysm repair is associated with a 0.25% incidence of paraplegia. The specific impact of extensive segmental artery sacrifice on spinal cord perfusion during TAA repair is poorly understood.

Various methods can facilitate preventing ischemic injury to the spinal cord. Distal aortic perfusion with extracorporeal support reduces the incidence of paraplegia. Any of the various methods of distal bypass are likely to be beneficial when the anticipated cross-clamp time is longer than 30 minutes, but they are probably not beneficial when cross-clamp time is less than 20 minutes. CSF drainage is frequently used to improve spinal cord perfusion during TAA repair and is often used in combination with distal aortic perfusion. Spinal cord perfusion pressure is defined as distal mean aortic pressure minus CSF pressure or central venous pressure, whichever is greatest. Autoregulation of spinal cord blood flow is similar to cerebral autoregulation, and blood flow is relatively constant over the range of 50 to 125 mm Hg. During hypoxia or hypercapnia, autoregulation is lost, and flow becomes linearly related to perfusion pressure. Thus, significant flow may remain even at very low perfusion pressure. Drainage of CSF is important because CSF pressure often increases (by 10 to 15 mm Hg) with cross-clamping of the descending thoracic aorta. The increase in CSF pressure reduces spinal cord perfusion pressure and increases the likelihood of ischemic spinal cord injury.

Despite evidence from animal studies that CSF drainage protects the spinal cord, clinical use of this technique is controversial. One randomized trial reported a reduced incidence of paraplegia, but another reported no benefit. Most of the evidence in support of CSF drainage comes from nonrandomized historical cohort studies in which the technique is used in combination with other adjuncts, such as intrathecal papaverine and hypothermic partial bypass. Coselli and colleagues⁷⁹ offered the strongest evidence supporting the efficacy of CSF drainage. They conducted a prospective, randomized clinical trial to evaluate the impact of CSF drainage on the incidence of spinal cord injury after Crawford type I and II TAA repair. CSF drainage resulted in an 80% reduction in the relative risk for a postoperative deficit. Nine patients in the control group (13%) had paraplegia or paraparesis versus only two patients in the CSF drainage group (2.6%). Left heart bypass, moderate heparinization, permissive mild hypothermia, and reimplantation of patient intercostal and lumbar arteries were performed in both treatment groups. The target CSF pressure was 10 mm Hg. CSF drainage also reverses delayed-onset neurologic deficit after open and endovascular TAA repair.⁸⁰

Although CSF drainage is widely used during TAA repair, it has risks. Potential complications include headache, meningitis, chronic CSF leakage, spinal or epidural hematoma, and subdural hematoma. The possibility of intraspinal pathologic processes should be considered in any patient with a postoperative lower extremity neurologic deficit. A retrospective review of 230 patients who underwent TAA repair with CSF drainage reported eight subdural hematomas (3.5%).⁸¹ High-volume CSF drainage was identified as a risk factor for its occurrence. Six patients had subdural hematomas detected during hospitalization, with an associated mortality of 67%. Two patients were seen in a delayed fashion, and both required an epidural blood patch to control chronic CSF leakage.

Hypothermia is probably the most reliable method of neuroprotection from ischemic injury. By reducing O₂ requirements by approximately 5% for each degree centigrade,

a two-fold prolongation of tolerated cross-clamp time is achieved by cooling even to mild hypothermia (34°C). Because the reduction in metabolic rate is linearly related to temperature, moderate or profound hypothermia provides even greater protection. Both systemic and regional spinal cord cooling could be beneficial. Systemic hypothermia can be achieved with either full cardiopulmonary bypass (with or without DHCA) or partial bypass. Cooling to 30°C to 32°C with left atrial-to-femoral bypass in conjunction with CSF drainage was associated with no permanent neurologic sequelae in a series of 20 patients despite a relatively long average cross-clamp time (\approx 70 minutes).⁸² Regional cooling of the spinal cord by cold perfusion of the AKA with blood or crystalloid provides significant protection during spinal ischemia in animal models. Regional cooling is beneficial in humans who received epidural infusions of 4°C saline. Even if active cooling is not used, it is advantageous to allow patients to passively cool to 33°C to 34°C during TAA repair. With passive cooling, the challenge is rewarming after the surgical repair. This is most easily accomplished with the use of a forced-air blanket over the upper part of the body. The lower body region should not be actively warmed because warming ischemic tissue increases metabolic requirements, acidosis, and ischemic injury.

Many drugs have been studied in an attempt to reduce the incidence of ischemic spinal cord injury. Barbiturates provide significant protection. Corticosteroids provide protection in dogs but were beneficial in humans only when they were combined with CSF drainage. Calcium channel blockers were not consistently shown to be protective against SCI. N-methyl-D-aspartate (NMDA) receptor antagonists have been investigated because ischemic injury appears to be related to increased levels of excitatory amino acids (particularly glutamate), which allow increased permeability to Ca²⁺ ions and high intracellular Ca²⁺ concentrations. Dextrorphan (a noncompetitive NMDA receptor antagonist) showed benefit during SCI in animals. Magnesium, another NMDA receptor antagonist, improves recovery from SCI in rat and dog models when administered intrathecally. Naloxone is protective in patients with traumatic spinal cord injuries and in a rabbit model of spinal ischemia. Other than the use of corticosteroids and naloxone at a few centers, most of these agents are considered investigational. Preoperative spinal cord angiography has been used in patients with TAA. The rationale for this highly invasive angiographic procedure is that precise identification of intercostal arteries giving rise to the AKA will allow focused reimplantation of these vessels during surgical repair and help prevent spinal cord injury. Selective intercostal angiography identifies the AKA when an intercostal branch is found making a cephalad hairpin turn to enter the spinal canal and supply a midline longitudinal artery (i.e., the anterior spinal artery) (Fig. 56.10). The AKA can be identified in 43% to 86% of patients studied with traditional angiography. Higher detection rates for AKA localization have been reported with computed tomographic angiography (CTA) and magnetic resonance angiography (MRA), with the latter achieving rates of nearly 100%.⁸³

The importance of reimplanting the intercostal arteries identified as supplying the AKA is not universally accepted. Even in patients with an identified and reimplanted AKA,

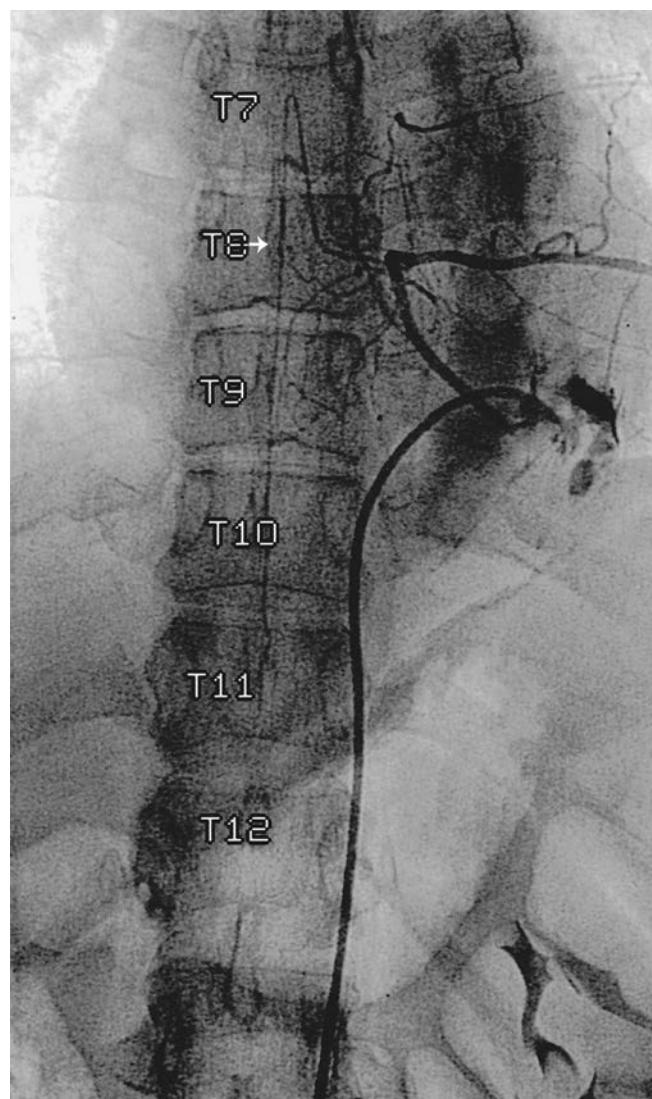


Fig. 56.10 Spinal cord angiogram of an extensive degenerative thoracoabdominal aortic aneurysm. Selective injection of the intercostal artery at T8 (arrow) demonstrates the great radicular artery and the extensive paravertebral collateralization.

spinal cord injury is not always prevented. Some investigators have concluded that preoperative localization of the AKA has little impact on neurologic outcome after TAA repair. A report⁸⁴ found no improvement in overall neurologic outcome with preoperative spinal cord angiography, but it offered important insight regarding the type of aneurysm, identification of the AKA, and neurologic outcome. In patients undergoing TAA repair for extensive degenerative aneurysms, spinal cord injury occurred in 0 of 45 patients versus 10 (12%) of 81 patients with and without an identified AKA, respectively. In contrast, identification of the AKA was not helpful in the case of chronic expanding aortic dissection, with 3 (15%) of 20 patients versus 3 (6%) of 49 patients suffering spinal cord injury with and without an identified AKA, respectively. The investigators hypothesized that mural thrombus in degenerative aneurysms results in the occlusion of many intercostal arteries and favors the development of extensive paravertebral collateral channels (see Fig. 56.10). Identification of an AKA allows focused reimplantation with uniform success. In

patients with chronic dissection, most intercostal arteries are patent, collateralization is minimal, and reimplantation of one or two intercostal arteries may be insufficient to supply blood flow to the spinal cord. This collateral blood supply concept is further supported by clinical studies demonstrating that clamping the segmental supplier to the AKA during TAA repair does not produce critical SCI in the majority of patients.⁸³ Sufficient collateral blood supply, independent of the AKA, must therefore exist to maintain spinal cord integrity.

Delayed-onset neurologic deficits are common after TAA repair.⁸⁵ In a large series of 2368 TAA repairs, 93 (3.9%) patients had postoperative paraplegia or paraparesis, 34 (37%) of whom initially had intact spinal cord function but a deficit developed later.⁸⁶ Preoperative renal dysfunction, acute dissection, and extent type II TAA are significant predictors of delayed neurologic deficit. Postoperative hypotension and CSF drain malfunction may play an important role in the development of these deficits. Neurologic function can frequently be recovered by maintaining an optimal arterial blood pressure and CSF drainage.

Renal Ischemia and Protection

Renal failure after TAA repair results from preexisting renal dysfunction, ischemia during cross-clamping, thrombotic or embolic interruption of renal blood flow, and hypovolemia and hypotension. Approximately 6% of patients require postoperative dialysis, even in centers with the most clinical experience. The associated mortality can be high. The primary predictor of postoperative renal failure is preoperative renal dysfunction. The duration of cross-clamp time is very important with the clamp-and-sew technique. Retrograde distal aortic perfusion techniques are widely used to preserve renal function during the cross-clamp period. Adequate bypass flow and arterial blood pressure are essential for maintaining renal function. Systemic and regional hypothermia, by reducing O₂ requirements, protects the kidneys during ischemia. Some centers advocate the use of DHCA in the treatment of distal TAAs (i.e., extent type III and IV) to preserve renal function.

The role of pharmacologic protection is somewhat controversial. Mannitol 12.5 to 25 g/70 kg is often given before cross-clamping. Mannitol improves renal cortical blood flow and the glomerular filtration rate in animal models of ischemia. Endothelial cell swelling is decreased, and an osmotic diuresis occurs. Evidence demonstrates free radical scavenging with mannitol and subsequent protection from ischemia in animals. Loop diuretics are sometimes given, but these drugs have been less effective than mannitol in experimental models. In clinical studies, the prophylactic use of loop diuretics has not been shown to improve outcome or reduce the need for dialysis for patients with acute renal failure. Dopamine given in low doses (1 to 3 µg/kg/min) dilates renal blood vessels and increases renal blood flow and urine output. Despite these beneficial effects, whether dopamine provides renal protection during ischemia is not clear. Fenoldopam mesylate, a selective dopamine type 1 agonist that preferentially dilates the renal and splanchnic vascular beds, has shown some promise as a neuroprotective drug; however, there is no evidence to support its routine use.

At the present time, optimal renal protection during TAA surgery should rely on hypothermia, mannitol, and prevention of hypotension and hypoperfusion of the kidneys.

Coagulation and Metabolic Management

Coagulopathy is a frequent complication during TAA repair. A dilutional coagulopathy in which platelets become deficient after approximately one blood volume of replacement develops during massive transfusion (see also [Chapters 49 and 50](#)). At between one and two blood volumes of replacement, coagulation factors are diluted to levels low enough to increase bleeding. Other contributing factors are residual heparin; ischemia of the liver, in which most coagulation factors are produced; and persistent hypothermia after weaning from bypass. With the early use of fresh frozen plasma and platelets, severe coagulopathy often can be avoided. The prothrombin time, partial thromboplastin time, fibrinogen level, and platelet count should be measured frequently. Point of care thromboelastography (TEG or ROTEM) can be used as well. Cryoprecipitate may be necessary to correct coagulopathy, especially when the prothrombin time and partial thromboplastin time are prolonged and hypervolemia prevents the administration of significant volumes of fresh frozen plasma. When coagulopathy persists despite these efforts, α -aminocaproic acid or tranexamic acid are beneficial as antifibrinolytic therapy, and desmopressin can be given to increase circulating levels of von Willebrand factor and factor VIII. Normothermia should be achieved by complete rewarming before separation from bypass, by increasing ambient temperature after separation from bypass, and by forced-air warming over the upper body skin surface.

Analysis of arterial blood gases and electrolyte levels should be performed frequently. Hyperkalemia should be treated aggressively, especially in oliguric or anuric patients. Calcium chloride, sodium bicarbonate, insulin, and glucose are the primary acute treatments of hyperkalemia.

Endovascular Aortic Repair Surgery

HISTORY OF ENDOVASCULAR AORTIC REPAIR

The first EVAR of the aorta was performed and reported by the Ukrainian surgeon Dr. Nicholas Volodos and his colleagues. On March 24, 1987, they performed the world's first human endovascular repair of a posttraumatic pseudoaneurysm of the descending thoracic aorta and the patient survived 18 years after the stent graft insertion without any evidence of stent-related complications. However, the case was performed behind the iron curtain, which prevented the news from spreading globally and their work was first published in Russian in 1988.⁸⁷

On September 7, 1990, the Argentinian surgeon Juan Parodi, the Argentinian radiologist Julio Palmaz, and their colleagues performed the first successful EVAR in the western world. After many years of experimenting with stents on dogs with minimal funding, Parodi finally got permission from his institution to perform cases on humans on the condition that these patients had to have been rejected for intervention previously by at least two

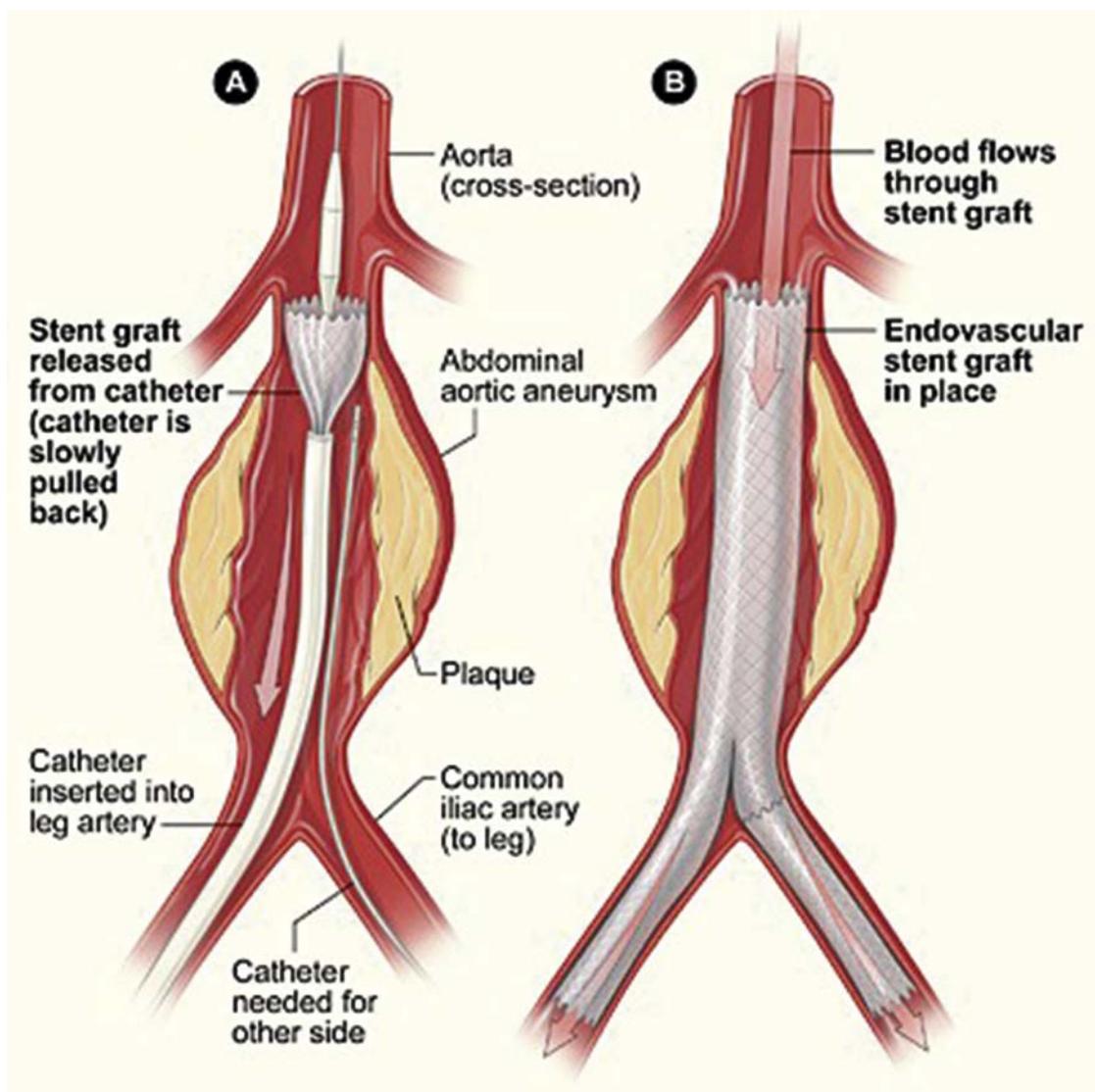


Fig. 56.11 Endovascular aneurysm repair, abdominal aortic aneurysm. (A) A catheter is inserted into an artery in the groin (upper thigh). The catheter is threaded to the abdominal aorta, and the stent graft is released from the catheter. (B) The stent graft allows blood to flow through the aneurysm. (Retrieved from: <https://surgery.ucsf.edu/conditions--procedures/endovascular-aneurysm-repair.aspx>.)

centers. In 1990, he received a phone call from the Argentinian president asking him to use his device on his cousin, who had an aortic aneurysm and severe COPD. Parodi went ahead with the operation and it became his first successful EVAR. Three months later, the patient developed a distal endoleak and he was treated by an aorto-uni-iliac endograft, occlusion of the contralateral common iliac, and a femorofemoral bypass. The patient lived for 9 years and died due to pancreatic cancer.⁸⁸

For all types of aortic diseases, endovascular technique is being utilized as the most feasible treatment option and a perfect alternative to treat AAA with the aid of endoluminal devices (Fig. 56.11).⁸⁹ Endovascular grafting is being used in diverse types of traumatic injuries, ruptures, aortic dissection, and in the diseases of thoracoabdominal and thoracic aorta. The EVAR is considered as one of the main treatment choices for patients who are affected with AAA because it is a less invasive treatment option, associated with reduced perioperative mortality and morbidity, and requires a short stay at the hospital.⁹⁰

ADVANCEMENTS IN STENT-GRAFT DEVICES

The endovascular technique started in the 1960s on an experimental basis and has progressed through a number of developmental eras that have resulted in EVAR as the most common technique for repair of AAAs. More than 2 decades ago, Parodi and fellow colleagues described the first endovascular repair of an AAA.⁹¹ At that time, endografts for AAAs were being used on patients who had severe surgical risks for an open AAA repair. After analysis and detailed comparison of the developments and advancements in EVAR over the previous two decades, the researchers were able to show considerable success in the procedures, outcomes, and efficiency. These grafts are made up of modular bifurcated devices composed of synthetic fabric which is deployed in the human body through a catheter. After the successful treatments in endovascular repair of the descending aorta, the stents received undeniable success and accolades in cases of the infrarenal aorta. Dake and colleagues, in 1994, provided the first report of using these

stent devices for patients with descending thoracic aortic aneurysms.⁹² They conducted tests on 13 patients by using self-expanding stent-grafts and reported 100% success, thereby gaining approval of using these stent-grafts by the FDA after a decade in 2005. Since then, the FDA approved many other stent-graft devices for an array of aortic diseases such as traumatic transections, ruptures, penetrating ulcers, dissections, and aneurysms. In 2012, the FDA approved fenestrated stent-grafts for correcting pararenal and juxtarenal aortic aneurysms through endovascular mechanisms.⁹³ In these devices, the graft fabric openings or scallops are easily aligned with the arteries splitting off the aorta, which significantly paves the way to exclude the aneurysm yet maintain the end-organ perfusion. These fenestrated grafts are referred to as FEVAR, or fenestrated EVAR, whereas the original EVAR name is commonly used to refer to the treatment of infrarenal aneurysms by bifurcated grafts.

It is also worth noting that off-the-shelf stent-grafts are ready-to-go types of stent-grafts used to treat those patients who fall prey to complex AAAs in the acute setting. These standard off-the-shelf fenestrated stent-grafts were studied by the researchers, who found no reported cases of perioperative death, rupture, aneurysm dilation, and stent migration.⁹⁴ However, there is still a dearth of significant clinical evidence for the potential benefits of off-the-shelf fenestrated and branched stent-grafts; hence, more research is suggested to support the view that these stents have the true potential of lessening the requirement for perioperative customization of graft for AAA patients. The main difference between the fenestrated grafts (FEVAR) and multi-branched grafts (mBEVAR) is that the latter has axially oriented cuffs that serve as routes to cannulate and stent the target mesenteric or renal vessels. This gives surgeons less need for absolute precision to align the scallops of the FEVAR right at the origin of the vessels. It also gives them a small margin of moving safely with the device up or down the aorta. This comes at the expense that the BEVARs tend to be longer devices and end up covering a significant portion of the descending thoracic aorta; hence, the risk of spinal cord injury exists, and spinal cord protection strategies should be considered.⁹⁵

The endovascular approach is the best alternative, as it is devoid of open incisions, considerable fluid shifts and blood loss, wide dissections, and long aortic cross-clamp times as in open repairs. Careful attention must be given in choosing the optimal treatment strategy for patients with AAAs. The endovascular technique involves the placement of the endovascular stent graft based at the level of the disease and the size of the vessel. The femoral arteries are accessed either by cutdowns or percutaneous punctures. In general, smaller devices are available commercially that are used percutaneously for EVARs, FEVARs and BEVARs. The decision to do a cutdown versus a percutaneous access depends on the size of the device used, the size of the femoral arteries, preexisting femoral or iliac diseases or surgeries, and the surgeon's experience. If the patients have critically diseased iliac or femoral arteries, then they may need a concomitant, local endarterectomy or balloon angioplasty. Additionally, during the endovascular AAA, around 20% patients are recommended to undergo adjunctive retroperitoneal procedures where the common iliac artery is exposed by

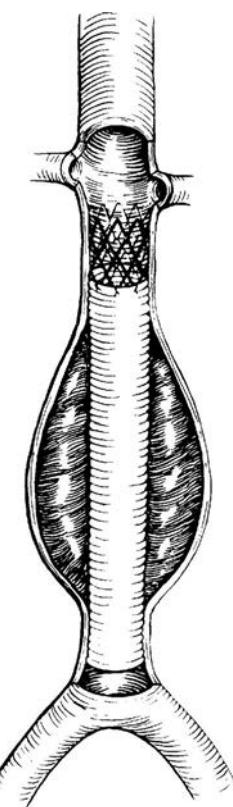


Fig. 56.12 Graft-stent combination with cephalic stent. (From Yao JST, Eskandari MK. Transfemoral intraluminal graft implantation for abdominal aortic aneurysms: two decades later. *Ann Vasc Surg*. 2012;26[7]:895-905.)

a retroperitoneal approach to place a synthetic iliac conduit. The same iliac conduit is used to endoluminally place the delivery system into the aorta. Beyond any reasonable doubt, these adjunctive retroperitoneal procedures allow a significant number of patients to experience EVAR, though these procedures are not free from longer hospital stays, lengthy procedure time, and extensive blood loss because of their hybrid nature.⁹⁶

Understanding the surgical approach and vascular access is important to tailor the anesthesia plan. Most EVARs today are done via percutaneous access of the femoral arteries, which makes monitored anesthesia care (MAC) with local infiltration to the access site a viable safe alternative (Figs. 56.12 to 56.14). On the other hand, multi-branched grafts are much bigger devices and the procedure takes much longer, as it often involves cannulation of the visceral branches. Cannulation of caudally oriented vessels, as the celiac axis and the superior mesenteric arteries, is easier if performed via a left axillary artery access. However, today there are deflectable, steerable guiding sheaths that allow the surgeon to cannulate the caudally oriented vessels from the same femoral access used for insertion of the aortic component, without the need for an additional arterial cutdown in the left arm.

ANESTHETIC MANAGEMENT

To sketch the anesthetic management options associated with EVAR, it is worth mentioning that in the past, the long

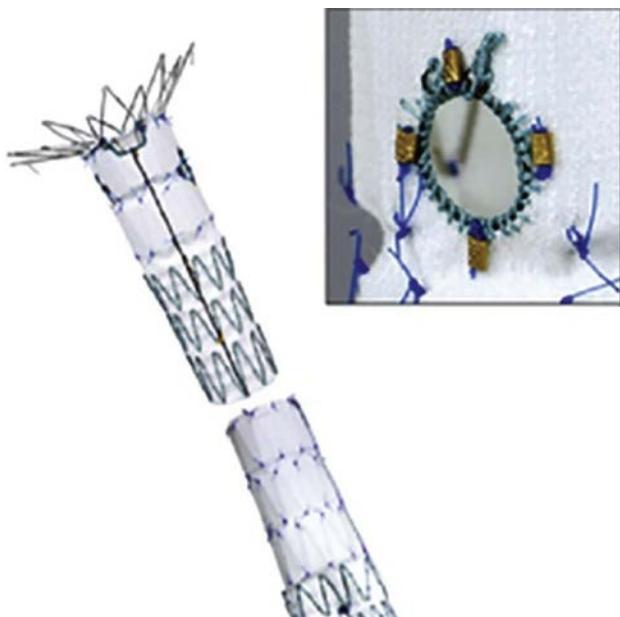


Fig. 56.13 Customized fenestrated stent-graft. (Modified from Kothandan H, Chieh GLH, Khan SA, et al. Anesthetic considerations for endovascular abdominal aortic aneurysm repair. *Ann Cardiac Anaesthesia*. 2016;19[1]:132.)

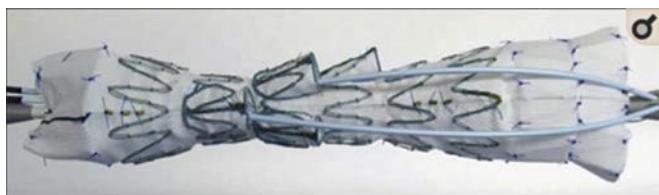


Fig. 56.14 Customized branched stent-graft. (From Kothandan H, Chieh GLH, Khan SA, et al. Anesthetic considerations for endovascular abdominal aortic aneurysm repair. *Ann Cardiac Anaesthesia*. 2016;19[1]:132.)

surgical procedures used general anesthesia. But with the passage of time, surgical procedure times were reduced, and procedures started changing due to the inclusion of newer generation devices. The local and regional anesthetic techniques were employed with IV sedation. Despite the significant use of local, regional, and general anesthesia, conflicting reports raised their voices concerning anesthetic management. According to a report, the regional or local anesthesia might decrease the hospital stay time, ICU admission, and reduce early complications.⁹⁷ Anesthesiologists must consider things like a patient's functional status, co-morbidities, aneurysm complexity, and surgical urgency. According to Kothandan and associates, when it comes to examining a unique risk stratification model in endovascular surgery scenarios, there is no single useful model.^{97a} Few anesthesiologists take leverage of the *risk scores* in this context, such as revised *cardiac risk index* or the modified *customized probability index* when they are engaged in endovascular surgery scenarios.^{98,99}

Spinal Cord Blood Supply

It is critically important to get an overview of the spinal cord blood supply since it has a complex anatomy, but the latest imaging technology has expanded the very small and

complex vessels to a significant degree. Hence, it is now easier to determine the SC vascular pattern as compared to the past, as explained above in the open thoracoabdominal repair section. To better understand the blood supply to the spinal cord, it is important to adapt the collateral network concept. The concept can be summarized as follows:

- There is an axial network of arteries that supply the spinal cord and run in the paravertebral tissue.
- Input to this axial network comes from segmental arteries, subclavian arteries, and hypogastric arteries.
- Blood supply to the cord can increase from one source when another is compromised. An example of this steal phenomenon is the back bleeding from intercostals into an open aortic sac after aortic cross-clamping.¹⁰⁰

This extensive spinal cord arterial system thus consists of an extrinsic and an intrinsic vascular system. The extrinsic network consists of segmental vessels arising from the intercostal and lumbar arteries, subclavian arteries, and the hypogastric arteries. The intrinsic network which receives supply from the extrinsic network is formed by the two posterior spinal arteries supplying the posterior region of the cord and a single anterior spinal artery supplying the anterior region of the cord (Fig. 56.15). The posterior spinal cord is relatively protected from ischemia because the collateral blood flow from the two posterior spinal arteries compared to a single anterior spinal artery make the anterior portion of the cord more susceptible to ischemia. The anterior spinal artery originates from the terminal branches of both vertebral arteries and courses along the anterior longitudinal fissure of the spinal cord receiving reinforcing blood supply from the radicular or segmental medullary branches, the branch of the posterior intercostals, or the lumbar arteries. The largest of the anterior segmental branches is the AKA, which supplies a large portion of the caudal two thirds of the anterior spinal artery. Because it is the only major artery supplying this zone, it is susceptible to watershed ischemia. The AKA originates from a left posterior intercostal artery off the descending aorta usually between T9 and T12 but has originated in some instances from lower level up to L5. A unique identifying feature of the AKA is the "hairpin configuration" on imaging that can be explained by the differential growth of the spinal cord and vertebral column during embryologic development.

Anterior spinal cord syndrome happens due to infarction of the portion of the spinal cord supplied by the anterior spinal artery. This may happen due to disruption of the AKA or the anterior spinal artery itself at the thoracoabdominal levels. Etiologies may vary between aortic dissection, emboli, covering of large segments of the thoracoabdominal aorta by endovascular stents, and surgical dissection during repair of thoracoabdominal aortic aneurysm (TAAA). The clinical presentation of the syndrome will depend on the spinal cord tracts and the level of cord injury. Classically, the anteromedial portion of the cord which contains the corticospinal and the corticobulbar tracts receives blood supply only from the anterior spinal artery, leading to motor paralysis below the level of injury if this supply is disrupted. The anterolateral aspect of the cord contains the

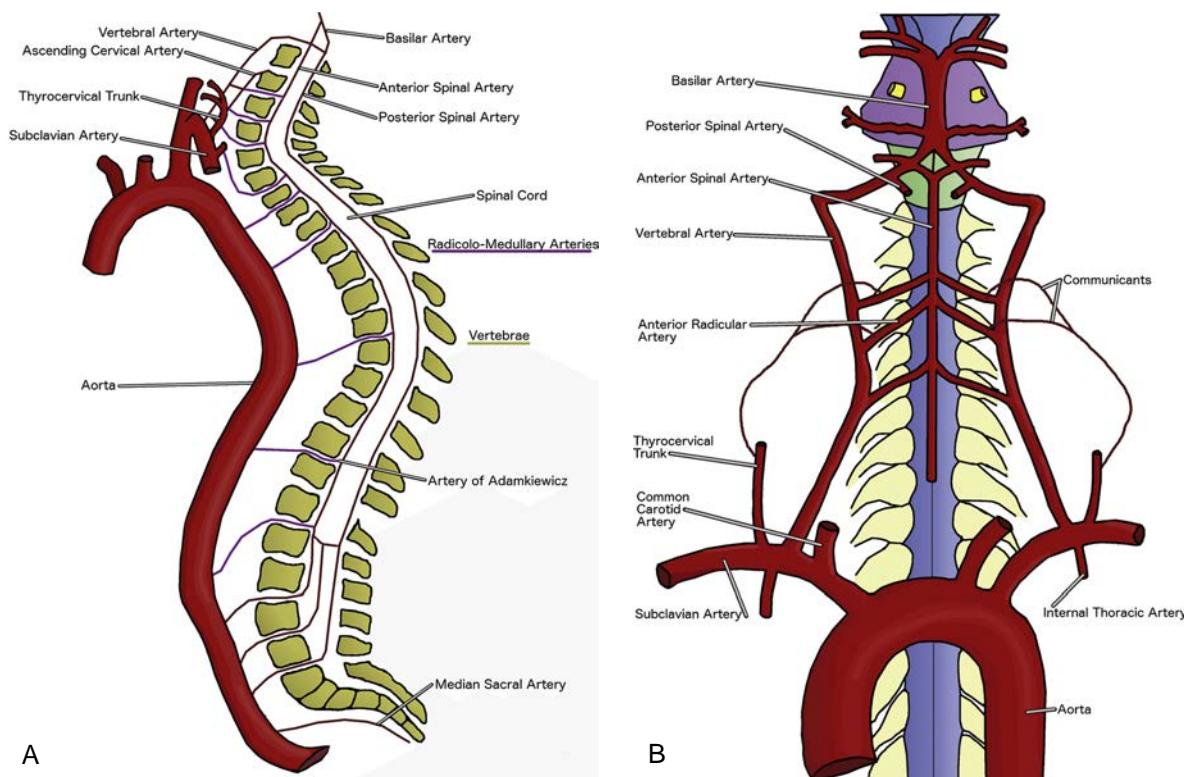


Fig. 56.15 An overview of blood supply to the spinal cord and the cervical spinal cord and the origin of the anterior spinal artery. (A) An overview of blood supply to the spinal cord. The spinal cord predominantly receives blood from three arteries originating around the cervical-cranial junction. These arteries run longitudinally along the spinal cord, terminating at the caudal end. These three arteries, the anterior spinal artery, and paired posterior spinal arteries, receive their blood supply mainly from the vertebral arteries, as well as the ascending cervical arteries, branches of the thyrocervical trunk. The thyrocervical trunk additionally supplies the cervical spinal cord via numerous anterior and posterior radiculomedullary arteries. These arteries do not anastomose with the spinal arteries (anterior and posterior); rather, they directly supply the spinal cord at the level they enter the vertebral canal. As the spinal cord extends caudally, blood supply becomes scarce. Anterior and posterior radiculomedullary branches from the thoracic and abdominal aorta continue to supply the spinal cord directly, but the spinal arteries do not receive any new anastomoses until the artery of Adamkiewicz at the level of the lower thoracic or lumbar vertebrae. The lumbar and sacral spinal cord additionally receives blood from the median sacral artery anteriorly. Magenta lines represent radiculomedullary arteries and yellow boxes represent vertebrae. (B) The cervical spinal cord and origin of the anterior spinal artery. The anterior spinal artery originates from the vertebral arteries around the level of the cervical-cranial junction. Additionally, the anterior spinal artery receives blood from the anterior radicular arteries (branches of the vertebral arteries) and the ascending cervical arteries (branches from the thyrocervical trunk). Following these original tributaries, the anterior spinal artery does not receive blood from any other anastomosis until the lower thoracic and lumbar areas via the Artery of Adamkiewicz (not shown). The anterior spinal artery supplies the bulk of oxygenated blood to the anterior spinal cord. (From Hoehmann CL, Hitscherich K, Cuoco JA. The artery of Adamkiewicz: vascular anatomy, clinical significance and surgical considerations. *J Cardiovasc Res*. 2016;5:6.)

spinothalamic and the spinocerebellar tracts, a watershed area that receives blood supply from both anterior and posterior spinal arteries, but it can still be infarcted in anterior spinal cord syndrome, affecting pain and temperature sensation. The dorsal columns responsible for vibratory sensation and proprioception remain intact, as they are usually supplied by the posterior spinal arteries. Patients with anterior spinal cord syndrome could also present with autonomic dysfunction as bowel and bladder incontinence, orthostatic hypotension, and sexual dysfunction.¹⁰¹

Anesthesia Roadmap for EVARs

Anesthesia administration mainly focuses on

- Hemodynamic stability of the patient
- The preservation of perfusion to the key organs such as splanchnic vessels, kidney, spinal cord, heart, and brain
- Early identification and management of blood loss and maintaining intravascular volume

- Maintaining the normal core body temperature (perioperative normothermia)

Karthikesalingam and associates argue that there is very little evidence indicating the best anesthetic technique in the case of standard EVAR, and even more limited evidence in the case of complex EVAR,¹⁰² because most of the literature relies on the retrospective and descriptive studies. Therefore, the selection of feasible technique should be based on several factors that include aneurysm complexity, patient's premorbid state, vascular team experience, choice of anesthesiologist, and the patient. Fleisher and associates favor the opinion that there is not a particular mode of anesthesia administration on cardiac disease patients in the ACC/AHA guidelines, because there is no sufficient evidence available in this regard.¹⁰³ It is not very common that the surgery turns into open repair scenario (the rates are less than 2%), yet the anesthesiologist should always be ready for such intraoperative rupture with massive blood loss by having adequate vascular access.¹⁰⁴ The following checklist provides an anesthesia roadmap that

the clinician can refer to when considering how to proceed with endovascular aortic procedures.

1. Choice of Anesthesia
2. Radiation Safety
3. Renal Protection Vascular Access and
4. Blood Loss
5. Temperature Control
6. Spinal Cord Protection

CHOICE OF ANESTHESIA

In 2015 Broos and associates studied the anesthesia choices, such as general, regional, or local, by reviewing the outcomes of EVAR repairs. They concluded that the choice of anesthesia was independent of perioperative morbidity and mortality.¹⁰⁵ At the same time, the local or regional anesthesia has an upper edge over general anesthesia in terms of postoperative hospital stay, ICU stay, and shorter procedure times. Edwards and associates examined the results of 6009 elective EVAR procedures against the anesthetic procedures such as local/ MAC, regional, and general anesthesia.¹⁰⁶ They concluded that general anesthesia was strongly linked with pulmonary morbidity and increased hospital stays after the EVARs when compared with local/ MAC and spinal cord anesthesia. Similarly, Karthikesalingam and associates conducted a systematic review and meta-analysis of 10 studies comprising of 13,459 patients who had EVAR surgeries under the administration of local or general anesthesia.¹⁰² They also reported that the group associated with local anesthesia had less postoperative complications and shorter postoperative hospital stays and procedure times when compared to the group associated with general anesthesia. Despite those retrospective studies and systemic reviews, current literature still lacks evidence based on randomized prospective data. Also, the continuous change is the commercially available devices and the increase in surgical experience with endovascular repairs, both of which lead to a composite decrease in operative times, today much less than when the initial endovascular aortic trials were performed.

The point is that there is no substantial evidence which proves that one anesthetic technique has an upper hand over another, and similarly, EVAR success does not point toward using any specific type of anesthesia, either. Generally speaking, simple EVARs performed through percutaneous punctures of the groin can be done under local anesthesia and MAC, neuraxial anesthesia, or general anesthesia provided that there is no other contraindication for the choice of anesthesia. More complex endovascular repairs, like FEVARs or BEVARs, that require multiple arterial cutdowns or combined groin and arm access will require general anesthesia.

Radiation Safety

Endovascular procedures are gaining much more popularity on the score that they exhibit lower access site-related blood loss, but at the same time, the patients become vulnerable to radiation exposure risk as well.¹⁰⁷ In this regard, the key principle of “As low as reasonably achieved” (ALARA) remains the bedrock foundation to limit the staff and patient’s exposure to radiation accordingly.¹⁰⁸⁻¹¹⁰ In this regard, the clinician can take leverage from real-time

radiation monitoring procedures so that he is aware of the situation and can adjust the radiation dose.¹¹¹

Renal Protection

Contrast-induced nephropathy (CIN) is renal function impairment associated with a baseline increase in serum creatinine concentration of 25% or more or an absolute increase of 0.3 to 0.5mg/dL within 2 to 3 days of administration of IV contrast.¹¹²

The recently published PRESERVE Trial, which was a 2-by-2 factorial design, double blinded, placebo and comparator drug controlled randomized study, was conducted to answer the question of whether the outcomes changed after angiography with sodium bicarbonate and acetylcysteine in patients at high risk for CIN. The study concluded that there was no benefit for intravenous sodium bicarbonate over intravenous sodium chloride, nor for acetylcysteine over placebo in prevention of death, need for dialysis, persistent decline in kidney function at 90 days, or for the prevention contrast-induced acute kidney injury.¹¹³

The two most important factors that contribute to CIN are the contrast load and the preexisting kidney disease. Limiting the contrast load, and adequate hydration to decrease the viscosity of the iodine-based dyes, thus decreases the oxidative stress exerted on the proximal convoluted tubules.

Vascular Access and Blood Loss

The surgeons normally use either axillary or left brachial access for the insertion of snorkels or visceral stents of the FEVARs. Cheng underlines that there are specific challenges in anesthetic management scenarios such as prevention of paraplegia and stroke and blood pressure control.¹¹⁴ Apart from this, the surgeons might need to perform bilateral femoral cutdowns as well as left axillary access. This would leave one limited option (i.e., right arm for intraaortic balloon pump [IABP] access). A central venous access is not a must but would be used when complex branched grafts are used with multiple comorbidities and limited peripheral venous access. Lengthy procedures like branched grafts are performed under anticoagulation with heparin. This creates a potential of continuous slow blood loss from the multiple arterial cutdown sites that could be overlooked in the dark hybrid rooms. It is not very common to experience sudden hemodynamic instability in patients unless there is a ruptured aneurysm or an aortic occlusion balloon is being used.

Temperature Control

The patient is vulnerable to perioperative hypothermia with adverse outcomes due to many untoward physiologic effects. Hence, maintaining normothermia is highly recommended and needed before the skin incision process. This could be accomplished by enhancing the operating room’s ambient temperature, by using warming IV fluids, and by using radiolucent underbody warmers. Hypothermia could delay tracheal extubation, which is sometimes preferred to be as early as possible to perform a neurological exam if there are risks of strokes or spinal cord injury.

Spinal Cord Protection

SCI remains one of the devastating possible complications of thoracic and thoracoabdominal aortic repair.^{115,116}

There could be many reasons for the occurrence of SCI such as occlusion risk of stent grafts associated with the AKA, considered the main collateral artery, or due to damage of middle sacral arteries, inferior mesenteric arteries, or internal iliac arteries. Risk factors for spinal cord injury include emergency surgery, aortic dissection, extensive aortic disease, aortic rupture, prior abdominal surgery, hypogastric artery occlusion, and history of renal dysfunction. Therefore, many strategies are utilized such as arterial pressure augmentation, CSF drainage, and lowering the CVP.¹¹⁷ The 2014 European guidelines on the management of aortic disease stated that CSF drainage can be beneficial in high-risk patients (class IIa, level of evidence C).¹¹⁸ Similarly, the ACCF/AHA guidelines for the thoracic aortic disease recommended CSF drainage in TEVAR patients who are at high risk for spinal cord injury (class I, level of evidence B).¹¹⁹ Spinal drains may be placed preinduction of general anesthesia or postinduction if the patient cannot tolerate the procedure. For patients receiving anticoagulant or antiplatelet therapy, the American Society of Regional Anesthesia and Pain Medicine (ASRA) Guidelines should be followed, and the perioperative management of those medications should be arranged with the perioperative team.¹²⁰

To optimize spinal cord perfusion, CSF pressure is usually monitored and CSF periodically drained. Most institutions have their protocols for management of spinal drains and CSF drainage. Most local guidelines and protocols advocate for CSF pressure of 10 to 15 mmHg. Some institutions drain CSF periodically during the intraoperative and postoperative periods while others drain according to CSF pressure or if there are any signs of SCI. Care should be taken to avoid over-draining CSF. Excessive rapid CSF drainage, particularly during the intraoperative period of heparinization or postoperative coagulopathy, could lead to intracranial hypotension and increase the risk of intracranial hemorrhage.

If bloody CSF is encountered during the placement, discussion with the surgeon and the patient if the placement was attempted preinduction, and rescheduling an elective case should be considered. Some institutions encourage routine fluoroscopic-guided spinal drain insertion to decrease the number of passes. Ultrasound-guided placement could be considered.

Other measures to decrease the risk of SCI is the maintenance of flow through the left subclavian artery and

the internal iliac arteries. Subclavian artery flow can be decreased if a TEVAR graft covers the origin of the left subclavian. Flow can be increased by a partial arch debranching through a left carotid to a left subclavian bypass. In the case of internal iliac arteries, minimizing the occlusion by large introducer sheaths will improve the spinal collateral flow.¹²¹

POTENTIAL COMPLICATIONS

Early and Late Complications

The early complications are those that occur from day 2 to day 30 after the surgery. These types of complications include postimplantation syndrome (PIS), paraplegia, stroke, acute renal failure, lower extremity and pelvic ischemia, aneurysm rupture, and others. The late complications that occur 31 days after surgery are associated with endoleak, but they also include the aneurysm rupture, its elongation, degenerated proximal neck, device migration, limb occlusion, and graft infection.

Endoleaks

EVAs are vulnerable to a condition where the aneurysm sac experiences arterial blood flow, despite the exclusion by the stent graft, and is thereby unable to maintain or accomplish full exclusion from the blood flow (Figs. 56.16 and 56.17). Chen and Stavropoulos explain the type 1 endoleak where the stent-graft fails to accomplish a circumferential seal at the distal (IB) or the proximal (IA) positions, thereby demanding immediate treatment due to aneurysm sac systemic pressurization.¹²² If not treated promptly, it can result in either expansion of the aneurysm or even rupture. An increased occurrence of IA needs early endovascular intervention and can occur with hostile proximal aortic neck anatomy. There are certain technologies which can detect these endoleaks, such as cone-beam computed tomography. Endoleaks are normally treated by balloon angioplasty of the proximal attachment site so that the desired seal is obtained through remodeling of the stent-graft. The endoleak can also be bridged by covered extension of the native endograft.¹²³ Embolization is another treatment option specifically when the space between the renal arteries and the endograft is not adequate. Fenestrated or branch graft extensions are also an alternative for more complex proximal endoleaks.

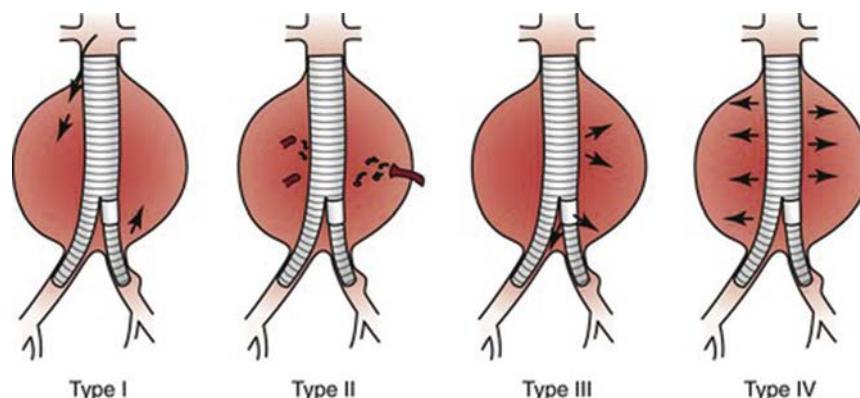


Fig. 56.16 Endoleak types. (From White GH, May J, Waugh RC, et al. Type III and type IV endoleak: toward a complete definition of blood flow in the sac after endoluminal AAA repair. *J Endovasc Ther*. 1998;5:305–309.)



Fig. 56.17 Computed tomography scan performed 2 days after endovascular aneurysm repair revealing a progressively enlarging type 1A endoleak. (From Tureli D, Baltacioglu F. Type I endoleak management after endovascular repair of infrarenal abdominal aortic aneurysm. *Vasc Dis Manag*. 2014;11:E91–E97.)

Type II endoleak occurs when the aneurysm sac undergoes reverse filling from the branch vessels through either lumbar or inferior mesenteric arteries.^{124,125} This can result in various anomalies such as aneurysm rupture, sac enlargement, and enhanced intrasac pressures.¹²⁶ Type II endoleaks can be treated by transarterial embolization through the iliac arteries or retrograde embolization through the superior mesenteric or inferior mesenteric arteries. They can also be treated by translumbar computed tomography angiogram (CTA)-guided embolization.¹²⁷ A transscaval approach can be used if the type II endoleak is close to the IVC. A transjugular sheath is advanced through the IVC and a needle is used to access the aneurysm sac to embolize it. Potential risks of such procedure include retroperitoneal bleeding, pulmonary embolization from non-targeted embolization, and aorto-caval fistulas.¹²⁸

Type III endoleaks trigger when the stent-graft undergoes a structural failure, thereby causing the blood flow to move into the aneurysm sac. These endoleaks occur either due to device failure such as detachment of modular graft components, a junctional leak, or due to erosion of fabric. They also require immediate intervention and treatment to avoid the risk of rapid expansion and rupture of aneurysm. These endoleaks are treated by inserting a new fabricated stent graft over the faulty position with angioplasty so that the desired seal is accomplished. The type IV endoleak is associated with graft porosity while Type V endoleak shows the aneurysm sac enlargement which is devoid of any demonstrable trace in the imaging procedures.

Open surgical treatment remains an option if endovascular treatment of endoleaks fails or is not possible. Surgical treatment could range from ligation of offending arteries feeding an endoleak to graft excision and open aneurysm repair with exogenous graft. Careful preoperative planning between the anesthesia and surgical teams requires all to be ready for the possibilities of rupture of the sac

intraoperatively or conversion into an open surgery if endovascular treatment fails.

Postimplantation Syndrome

PIS is a poorly understood phenomenon that could occur after endovascular aortic procedures and varies widely in incidence and clinical presentation. Possible explanations could be a sort of systemic inflammatory response syndrome (SIRS) in response to the instrumentation of the vascular endothelium or to the stent graft material. Clinical presentation could include a combination of fever, leukocytosis, thrombocytopenia, and coagulopathy. Treatment should be supportive treatment, especially with antipyretics, platelets, or fresh frozen plasma transfusions to treat the coagulopathy.¹²⁹

Hybrid Arch Repairs

Since the introduction of thoracic endovascular aortic repair (TEVAR) in the early 1990s,¹³⁰ the less invasive endovascular approach to descending and thoracoabdominal aneurysms has been advanced with the commercial availability of different types of thoracic stent grafts. Related to the morbid invasiveness and complexity of open arch surgical techniques, hybrid aortic arch repairs have been developed incorporating the use of TEVAR with a conventional elephant trunk repair, or combined with an open debranching of the cerebral vessels, frequently in combination with extraanatomical bypasses.^{131,132} This has simplified aortic arch reconstruction and made aortic arch repair feasible in high-risk individuals with comorbid conditions. Aortic arch pathology is also amenable to TEVAR but often requires a concurrent or staged open debranching procedure such as carotid-carotid or carotid-subclavian bypass. Debranching permits stent deployment across the origins of the arch vessels while maintaining perfusion to the head and upper extremities.¹³³

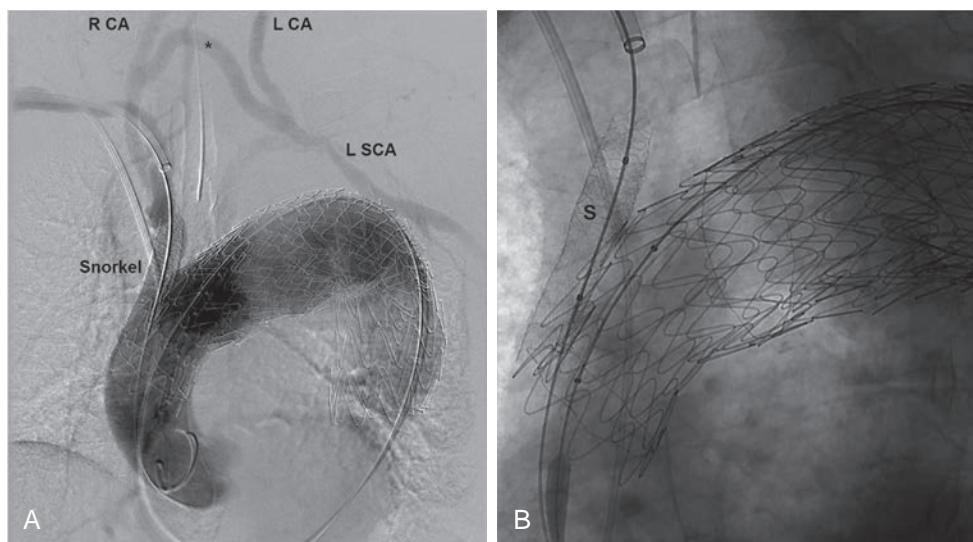


Fig. 56.18 Intraoperative digital subtraction angiography showing completed thoracic endovascular aortic repair with arch debranching after stent deployment with rapid ventricular pacing (RVP). (A) Overview of arch revascularization showing arch stent graft, innominate artery snorkel stent, and carotid-carotid-subclavian bypass (denoted by asterisk). Right common carotid artery (R CA), left common carotid artery (L CA), and left subclavian artery (L SCA) are labeled. The temporary pacer wire used to deliver RVP is visible entering the image from the top left. (B) Close-up of snorkel stent (S) extending from the ascending aortic arch into the innominate artery. The snorkel stent maintains flow to the carotid and subclavian arteries and allows deployment of a stent graft with proximal landing zone zero. (From Bokoch MP, Hiramoto JS, Lobo EP, et al. Rapid ventricular pacing for landing zone precision during thoracic endovascular aortic arch repair: a case series. *J Cardiothorac Vasc Anesth*. 2017;31(6):2141–2146.)

ANESTHETIC MANAGEMENT OF HYBRID ARCH REPAIRS

Anesthetic management of TEVAR must facilitate precise deployment of stents at short proximal landing zones near the arch vessels.¹³⁴ Deployment is complicated by the hydrodynamics of aortic blood flow that force the stent distally (windsock effect). Depending on the proximity of the planned stent graft to the left ventricular outflow tract, transient hypotension (systolic blood pressure of 60 mmHg) may be highly beneficial during deployment to limit stent migration. Various drugs have been used for this purpose,¹³⁵ but rapid ventricular pacing (RVP) is usually the preferred technique (Fig. 56.18).¹³⁶ RVP is reported to produce more profound hypotension more quickly, and with shorter duration as compared to sodium nitroprusside.^{137,138} While adenosine has been used for transient asystole in the setting of TEVAR and intracranial procedures, an unpredictable duration of asystole and wide individual variation in dose requirement (0.3 to 41 mg/kg) have been reported. Unexpected return of left ventricular contractions can complicate stent deployment at a critical moment. Unlike pharmacologic techniques, the onset and duration of RVP can be controlled precisely. The bulk of published data on RVP arises from the transcatheter aortic valve replacement (TAVR) literature, in which RVP is utilized during balloon valvuloplasty and valve deployment. Small studies have shown efficacy and safety of RVP for TEVAR, but deaths have been reported (Fig. 56.19).¹³⁹

Fig. 56.20 illustrates the hybrid aortic arch repair types I, II, and III.

Hybrid approaches for the treatment of aortic arch aneurysmal pathology are being performed with increasing frequency. This reflects the increasing comfort level of the surgeons with endovascular technology, and the improving technology of endovascular platforms to successfully

land stent grafts in the proximal thoracic aorta. As the patient population with thoracic aortic disease seeking intervention get older, embracing and honing these skills will be crucial. In addition to the associated operative mortality of complex operations such as aortic arch hybrid procedures, the Achilles heel of this intervention remains neurologic complications. Several groups have shown that arch hybrid procedures can be performed with acceptable mortality, with very minimal postoperative and long-term endoleak rates. Neurologic complications, including stroke and SCI, remain significant causes of morbidity and associated mortality.¹⁴⁰

Carotid Endarterectomy

The strong association between stroke and carotid artery disease is well known. The principal cause of carotid artery disease is atherosclerosis, which most commonly involves the bifurcation of the common carotid artery with frequent extension into both the internal and external carotid arteries. The clinical manifestations of carotid artery disease represent a spectrum of conditions, with fatal or debilitating stroke secondary to cerebral infarction at one end of the spectrum and ranging successively through non-debilitating stroke, transient ischemic attack, and amaurosis fugax (transient attack of monocular blindness) to an asymptomatic bruit. Cerebrovascular sequelae of carotid atherosclerosis may result either from embolization of thrombus or atheromatous debris or from a reduction in flow (hypoperfusion) secondary to stenosis. The latter probably accounts for less than 10% of the cerebrovascular sequelae of carotid atherosclerosis. Although much is known about the genesis and evolution of atherosclerosis, significantly less is known about the circumstances that lead to plaque instability and rupture. Regardless of the mechanism, the degree of

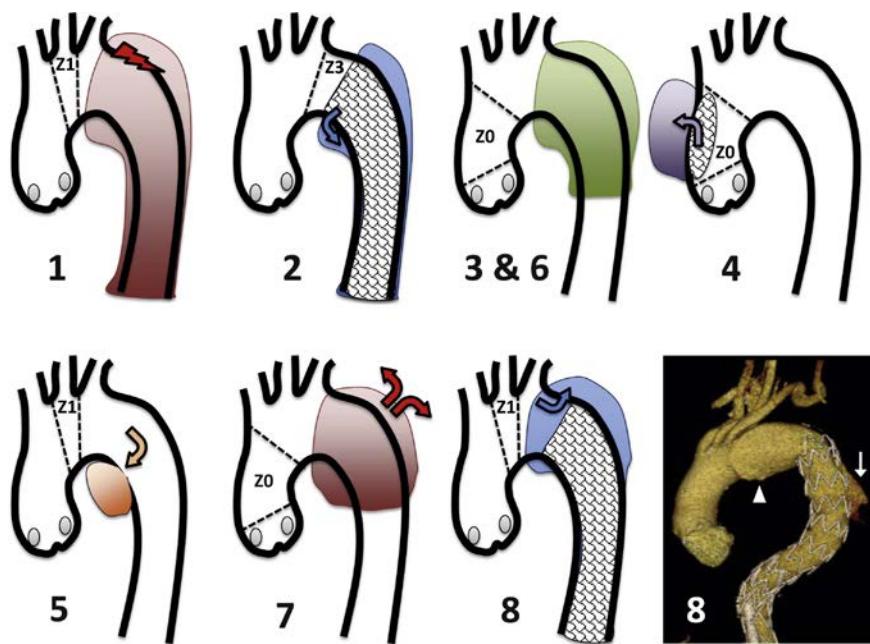


Fig. 56.19 Schematic of aortic arch pathology in patients undergoing thoracic endovascular aortic repair (TEVAR) with rapid ventricular pacing (RVP) in this study. (Patient #1) Aneurysm with chronic type-B dissection, (#2) aneurysm enlarging due to endoleak after prior TEVAR, (#3 and #6) isolated arch aneurysm, (#4) ascending aortic pseudoaneurysm due to leaking patch at prior aortic cannulation site from coronary artery bypass grafting, (#5) penetrating atherosclerotic ulcer of the aortic arch, (#7) ruptured aortic arch aneurysm, and (#8) enlarging aortic arch aneurysm and endoleak after prior TEVAR. Dashed lines indicate landing zone (Z) for proximal margin of stent deployed under RVP. Arrows indicate leak or extravasation of blood. Lightning bolt indicates dissection. (Bottom right) Preoperative three-dimensional reconstructed computed tomography angiogram for patient #8 showing aneurysmal dilation (arrowhead) and endoleak (arrow). (From Bokoch MP, Hiramoto JS, Lobo EP, et al. Rapid ventricular pacing for landing zone precision during thoracic endovascular aortic arch repair: a case series. *J Cardiothoracic Vasc Anesthesia*. 2017;31[6]:2141–2146.)

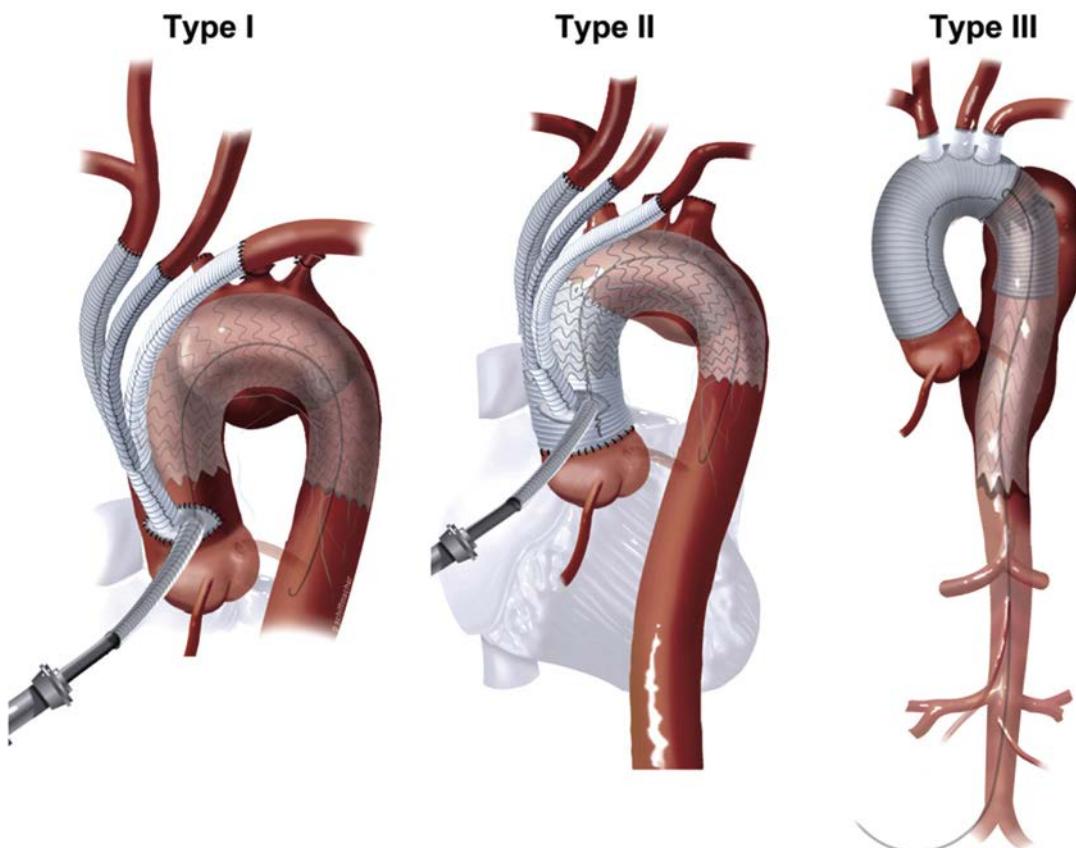


Fig. 56.20 Hybrid aortic arch repair types I, II, and III. (From Vallabhajosyula P, Szeto W, Desai N, et al. Type I and type II hybrid aortic arch replacement: postoperative and mid-term outcome analysis. *Ann Cardiothoracic Surg*. 2013;2[3]:280.)

cerebral injury depends on such factors as plaque morphology, characteristics of the embolus, duration of hypoperfusion, cerebrovascular vasoreactivity, integrity of the circle of Willis, and cerebral collateral circulation. A multi-society guideline is available for the management of carotid artery disease.¹⁴¹

Stroke is a major public health burden worldwide. It is the fifth leading cause of death and the leading cause of serious, long-term disability in the United States. Stroke is also a major contributor to health care costs. The direct and indirect costs of stroke in the United States in 2008 are estimated at \$65.5 billion.¹⁴² Approximately 780,000 people experience a new ($\approx 600,000$) or recurrent ($\approx 180,000$) stroke each year in the United States.¹⁴² Annually, more than 95,000 hospitalizations and 165,000 deaths occur from stroke. Well-defined risk factors exist in patients with stroke, the most important of which is hypertension. Approximately 83% of strokes are ischemic (i.e., cerebral thrombosis or embolism), and 7.6% of ischemic strokes result in death within 30 days of initial evaluation.¹⁴³ Extracranial atherosclerotic disease accounts for up to 20% of all ischemic strokes. Less than 20% of strokes are preceded by a transient ischemic attack. Despite a well-documented decline in stroke mortality, the annual incidence rate of stroke may be increasing. This increase is probably due to growth in high-risk populations. The incidence of perioperative stroke in unselected patients, patients with asymptomatic carotid bruit, and patients with at least 50% carotid stenosis undergoing general anesthesia and surgery is approximately 0.1%, 1.0%, and 3.6%, respectively.

Although treatment options to reverse the effect of acute ischemic stroke are limited, outcomes may be improved with appropriate therapy. One approved therapy is IV recombinant tissue plasminogen activator (rtPA). Given the narrow 3- to 4.5-hour treatment window from the onset of symptoms, prompt evaluation and diagnosis of ischemic stroke are critical. The addition of endovascular treatment options has improved outcomes. Three randomized controlled trials published in 2015 demonstrated a clear benefit of the addition of clot retrieval compared to standard therapy with thrombolytic. The 2015 American Heart Association/American Stroke Association guidelines now recommend endovascular treatment in acute ischemic stroke if the patient meets criteria (large vessel anterior circulation occlusion, independent at baseline, treatment within 24 hours).¹⁴⁴

INDICATIONS

Endarterectomy of the carotid bifurcation has been used to reduce symptoms and prevent stroke for more than 50 years. Although the efficacy of carotid endarterectomy for prevention of ipsilateral stroke in patients with and without symptoms has been demonstrated in large-scale randomized clinical trials,^{145,146} multiple factors including perioperative risk, comorbidities, and life expectancy must be considered in the overall assessment. In centers of excellence, it is a low-risk procedure with excellent long-term durability. Carotid endarterectomy is the most common peripheral vascular surgical procedure performed in the United States, with an estimated 130,000 procedures performed annually. The rate and number of carotid endarterectomies have fluctuated significantly since the early 1970s. With marked growth

in the specialty of vascular surgery and an expanding list of surgical indications, the number of carotid endarterectomies performed in nonfederal hospitals increased from 15,000 in 1971 to 107,000 in 1985 and then declined substantially over the next 5 to 6 years. The decline was probably due to publications questioning the indications for the procedures and isolated reports citing excessively frequent rates of operative morbidity and mortality.

In 1992, a marked increase in the number of carotid endarterectomies occurred after the results of two large-scale, prospective randomized trials were published. The North American Symptomatic Carotid Endarterectomy Trial (NASCET) and the European Carotid Surgery Trial both reported definitive results for symptomatic patients with high-grade carotid stenosis (70% to 99%).^{145,147} In NASCET, follow-up at 2 years showed a stroke rate for surgical patients of 9% versus 26% for medical patients. This benefit of carotid endarterectomy has persisted at 8 years of follow-up.¹⁴⁸ In the European Carotid Surgery Trial, the long-term stroke rate was 2.8% for surgical patients, excluding a perioperative stroke and death rate of 7.5%, and 16.8% for medically managed patients.

The efficacy of carotid endarterectomy in asymptomatic patients with carotid stenosis has been evaluated in five randomized trials.^{146,149-152} The Carotid Artery Surgery Asymptomatic Narrowing Operation Versus Aspirin trial, the first to publish its results, concluded that carotid endarterectomy was not indicated for asymptomatic patients with 50% to 90% carotid stenosis.¹⁴⁹ Unfortunately, this study was seriously flawed and the results questioned. The Mayo Asymptomatic Carotid Endarterectomy Study was terminated early because of a significantly increased number of MIs and transient cerebral ischemic events in the surgical group.¹⁵⁰ Most of these events were not related to the surgery itself but rather to the absence of aspirin in the surgical group. The Department of Veterans Affairs trial was designed to compare the effects of carotid endarterectomy plus aspirin versus medical treatment (i.e., aspirin) in asymptomatic male patients with 50% or greater carotid stenosis.¹⁵¹ This trial demonstrated a significant reduction in ipsilateral neurologic events in the surgical group (8%) versus the medical group (20.6%). However, the combined incidence of stroke and death was not different between study groups. The Asymptomatic Carotid Atherosclerosis Study (ACAS) demonstrated that patients with asymptomatic carotid stenosis ($\ge 60\%$) who were treated with carotid endarterectomy and aspirin have a reduced 5-year risk for ipsilateral stroke compared with patients treated with aspirin alone (5.1% vs. 11.0%).¹⁴⁶ These results reflect only a 5.9% absolute risk reduction in 5 years, which is just above 1% per year. Of note, improvement in outcome for patients randomized to undergo endarterectomy in this trial did not reach significance until 3 years after surgery. The European Asymptomatic Carotid Surgery Trial, the largest trial to date, largely replicated the results of ACAS, but in a somewhat more pragmatic setting.¹⁵² This trial demonstrated that patients with asymptomatic carotid stenosis ($\approx 70\%$) on ultrasound who were treated by immediate carotid endarterectomy plus medical treatment have a reduced 5-year risk for stroke compared with patients treated with medical therapy alone (6.4% vs. 11.8%). Of note, half of this 5-year benefit involved disabling or fatal strokes.

Although landmark randomized clinical trials have defined individuals who are likely to benefit from carotid endarterectomy (and set the standard for developing evidence-based practice guidelines throughout the world), it has been suggested that the significant increase in the number of carotid endarterectomies performed over the last decade may be due in part to the extrapolation of trial results to patients and settings not directly supported by the trials. For example, both NASCET and ACAS restricted enrollment to patients younger than 80 years of age, and both trials carefully selected institutions and surgeons to optimize the results of surgery. Additionally, subgroup analysis of ACAS could not demonstrate a significant benefit for women.¹⁴⁶ With the advent of a second interventional treatment modality, percutaneous carotid angioplasty and stenting (discussed later), and the evolution of intensive medical therapy, this issue has become more complex.

PERIOPERATIVE MORBIDITY AND MORTALITY

Although the randomized trials just noted have demonstrated a protective effect of carotid endarterectomy on ipsilateral stroke, the critical determinants of benefit for any given patient must include the overall perioperative event rate and expected long-term survival. Thus, the perioperative stroke and death rate for carotid endarterectomy needs to be very low to maintain the beneficial effects of surgery over medical therapy alone. Further, to compensate for the perioperative risk associated with surgery, the patient must have a reasonable life expectancy (12 to 18 months). The 30-day stroke and death rate of 2.3% for asymptomatic patients in ACAS (1987 to 1993) and 5.0% for symptomatic patients in NASCET (1988 to 1991) are often cited as benchmarks. More recent reports suggest a considerably less frequent event rate. For example, a prospective database study of 13,316 carotid endarterectomies performed in 2007 and 2008 reported a 30-day stroke and death rate of 1.3% in asymptomatic patients and 2.9% in symptomatic patients.¹⁵³ The 30-day mortality was significantly more frequent in patients who developed a stroke than in those who did not (12.9% vs. 0.6%). Patients with high-risk anatomy, such as restenosis and contralateral carotid arterial occlusion, have the highest risk for perioperative stroke and death. Neurologic deficits occur more commonly in patients with poorly controlled preoperative hypertension and in those with hypertension or hypotension postoperatively. The incidence of perioperative MI in patients undergoing carotid endarterectomy ranges from 0% to 5%. Recent reports suggest the incidence of MI is relatively low. The General Anesthesia versus Local Anesthesia for Carotid Surgery (GALA) trial (discussed later) results reported only 13 patients of 3526 (0.37%) had a perioperative MI.¹⁵⁴ The four fatal perioperative MIs accounted for only 8.9% of the total 30-day mortality. Although the role of carotid endarterectomy in patients older than 80 years of age remains a concern, recent reports suggest that carotid endarterectomy can be performed safely in the very elderly and those deemed high risk, with combined stroke or death rates being comparable to those found in randomized trials (NASCET and ACAS).

PREOPERATIVE ASSESSMENT

The optimal preoperative assessment for patients undergoing carotid endarterectomy continues to be debated (see also [Chapter 31](#)). Patients with recently symptomatic carotid disease present a particular challenge because strong evidence exists to support surgical intervention within 2 weeks after manifestation of symptoms, thus limiting the time available for evaluation and optimization of relevant comorbidities as well as the initiation of new medications.¹⁵⁵ The medical management of patients with asymptomatic carotid disease should be optimized and includes β -blockers, statins, and antiplatelet agents. Poorly controlled hypertension should be addressed with the patient's internist. The gradual decreasing of the arterial blood pressure over several weeks before surgery will restore intravascular volume, reset cerebral autoregulation to a more normal range, and improve perioperative management. Poorly controlled diabetes also warrants preoperative optimization, which may improve perioperative outcome.¹⁵⁶

CAD is common in patients undergoing carotid endarterectomy and is a leading cause of both early and late mortality. Hertzler and co-workers¹⁵⁷ performed coronary angiograms in 506 patients scheduled for carotid endarterectomy and found significant (>70%) stenosis in one or more coronary artery, CAD in 83% of patients suspected of having CAD, and CAD in 40% thought to have no CAD. Despite the known frequent incidence of CAD in patients undergoing carotid endarterectomy, preoperative studies for the evaluation of myocardial function or ischemic potential are rarely undertaken. Exceptions to this practice are patients with unstable angina, recent MI with evidence of ongoing ischemia, decompensated congestive heart failure, and significant valvular disease. In general, specialized cardiac testing would be unlikely to result in cancellation of the procedure or alter perioperative management. Further, the relatively infrequent overall rates of perioperative nonfatal and fatal MI after carotid endarterectomy make aggressive strategies leading to prophylactic coronary revascularization less appealing.¹⁵⁴ A recent clinical trial reported on the safety and efficacy of coronary angiography and revascularization in preventing postoperative cardiac ischemic events after carotid endarterectomy. In a randomized fashion, 426 patients with no history of CAD were randomized to either coronary angiography before carotid endarterectomy (216 patients) or carotid endarterectomy without coronary angiography (210 patients). In the angiography group, 68 patients had a significant stenosis of the coronary arteries and underwent revascularization with PCI (66 patients) or CABG (2 patients). PCI was performed 1 to 8 days before surgery and always consisted of angioplasty and stenting. No patients in the angiography group had a postoperative cardiac ischemic event or complication related to PCI, whereas nine patients in the group without angiography had an ischemic event (one fatal MI and eight ischemic events treated medically). Although all PCI patients received dual-antiplatelet therapy, no major bleeding or neck hematomas were observed. Long-term follow-up was not reported.

Patients with combined carotid stenosis and CAD requiring coronary revascularization represent somewhat of a management dilemma because it is often unclear which

disease should be treated first.¹⁵⁸ The severity of carotid and coronary disease must be evaluated in terms of clinical symptoms and anatomic lesions, and a decision must be made to perform a combined, staged (carotid endarterectomy first), or reverse-staged (CABG first) procedure. Carotid revascularization is recommended before CABG (staged procedure) in patients with symptomatic carotid disease and bilateral severe asymptomatic carotid stenosis. The optimal management of severe unilateral asymptomatic carotid stenosis in patients undergoing CABG is unclear. The only randomized clinical trial to date randomized 185 patients with severe unilateral asymptomatic carotid stenosis undergoing CABG to a staged or combined procedure (94 patients) or a reverse-staged procedure (90 patients).¹⁵⁹ Although the perioperative mortality rates were equivalent (~1.0%), the 90-day stroke and death rates were significantly lower in the staged and combined group (1.0% vs. 8.8%). Given the overall paucity of high-quality evidence, management of an individual patient should be guided by careful assessment of the relative severity of the coronary and carotid disease with particular emphasis on both surgeon-specific and institution-specific results in these patient populations. Carotid artery angioplasty and stenting is widely being applied as an alternative revascularization modality before staged CABG. More recently, a combined procedure (carotid angioplasty/stenting and CABG) has been introduced. In a small feasibility and safety study (90 patients), carotid artery angioplasty and stenting under local anesthesia followed immediately by CABG reported a 30-day stroke and death rate of 2.2%.¹⁶⁰

ANESTHETIC MANAGEMENT

Anesthetic management goals for carotid endarterectomy include protection of the heart and brain from ischemic injury, control of the heart rate and arterial blood pressure, and ablation of the surgical pain and stress responses. These goals must be achieved with another important goal in mind—to have an awake patient at the end of surgery for the purpose of neurologic examination.

The preoperative visit is particularly important in patients undergoing carotid surgery. During this visit, a series of arterial blood pressure and heart rate measurements are obtained from which acceptable ranges for perioperative management can be determined. Patients are instructed to continue all long-term cardiac medications up to and including the morning of surgery. Aspirin therapy should be continued throughout the perioperative period. As noted earlier, discontinuation of aspirin therapy may be related to an increased rate of MI and transient ischemic events in patients undergoing carotid endarterectomy. When patients arrive at the hospital on the day of surgery, they are queried regarding any new cardiovascular or cerebrovascular symptoms. Long-term cardiovascular medications not taken at home should be administered in the preoperative holding area whenever possible. Patient reassurance is particularly important at this time because anxiety is associated with increases in heart rate, systemic vascular resistance, and myocardial O₂ consumption, which in this patient population could precipitate myocardial ischemia.

ECG monitoring should include continuous leads II and V₅ for detection of rhythm disturbances and ST-segment

changes. An intraarterial catheter for beat-to-beat blood pressure monitoring should be considered routine. Non-invasive arterial blood pressure measurement in the contralateral arm is recommended. Central venous and pulmonary artery catheters are rarely indicated for carotid surgery. The rare patient with uncompensated heart failure or recent MI with ongoing ischemia requiring emergent surgery is a possible exception. If such monitors are used, the subclavian or femoral insertion sites are most practical because inadvertent carotid puncture could compromise blood flow as a result of hematoma. Oftentimes, the most common reason for central access is difficult or inadequate peripheral access. IV access for fluid and drug administration can be accomplished with two secure, medium-bore (16-gauge) peripheral IV catheters. Because both arms will be tucked to the patient's sides, the IV catheters must run well after patient positioning.

General Anesthesia

Any of the drugs commonly used to induce anesthesia, maintenance anesthetics, and nondepolarizing muscle relaxants can be used safely during carotid endarterectomy, given that stable hemodynamics are maintained and the patient is awake at the end of the procedure. A conventional technique is as follows. Sedative premedication (e.g., midazolam) has the potential to compromise early neurologic assessment and is universally avoided. After placement of routine monitors and administration of O₂ by facemask, small doses of opioid (e.g., fentanyl 0.5 to 1 µg/kg) are administered during arterial line placement. Induction of anesthesia is accomplished with incremental dosages of Propofol supplemented with additional opioid (total fentanyl dose 2 to 4 µg/kg). Etomidate also may be used and is preferred in patients with limited cardiac reserve. Neuromuscular relaxation with a short-acting to intermediate-acting nondepolarizing muscle relaxant such as rocuronium facilitates tracheal intubation. Esmolol is particularly effective in blunting the increases in heart rate and blood pressure responses during laryngoscopy and endotracheal intubation and is used liberally during the induction period. Arterial blood pressure responses during and after endotracheal intubation are unpredictable in this patient population, and the clinician must be prepared for immediate treatment of extremes in blood pressure. This can be achieved with the use of short-acting drugs, such as phenylephrine 50 to 100 µg for hypotension and sodium nitroprusside 5 to 25 µg or clevipidine 100 to 250 µg for hypertension. Patients with poorly controlled hypertension (diastolic blood pressure >100 mm Hg) require special care. These patients are often intravascularly volume depleted and may have significant hypotension with induction of anesthesia. Administration of fluids intravenously, careful titration of anesthetics, and immediate treatment of hypotension are especially important.

Anesthesia is maintained with inhaled volatile anesthetics. Sevoflurane or desflurane may be preferred because of more rapid emergence. Studies using EEG and regional cerebral blood flow (rCBF) measurements suggest that the critical rCBF (the rCBF below which EEG changes of cerebral ischemia occur) is lower for isoflurane. Sevoflurane's critical rCBF in patients undergoing endarterectomy is similar to that determined with isoflurane.^{161,162} This use of 50%

N_2O in O_2 was previously favored; however, its use has been associated with increased post-operative nausea and vomiting. Maintenance anesthesia can also be achieved with a combination of inhaled volatile anesthetic in combination with propofol, remifentanil, or dexmedetomidine. If neuro-monitoring is utilized, it would require inhaled volatile concentrations less than 0.5 minimum alveolar concentration (MAC) with supplementation of intravenous medications. Otherwise, clinical studies show no differences between volatiles and total intravenous anesthesia (TIVA).¹⁶³ With general anesthesia, a superficial plexus block is not required but can be considered as an adjunct.

Despite only modest surgical stimulation, hemodynamic fluctuations are common during carotid endarterectomy. Arterial blood pressure and heart rate are controlled within predetermined and individualized ranges during the surgical procedure with short-acting drugs whenever possible (phenylephrine, esmolol, nitroglycerin, sodium nitroprusside, nicardipine, clevipipine). Arterial blood pressure should be maintained in the high-normal range throughout the procedure and particularly during the period of carotid clamping to increase collateral flow and prevent cerebral ischemia. In patients with contralateral internal carotid artery occlusion or severe stenosis, induced hypertension to approximately 10% to 20% above baseline is advocated during the period of carotid clamping when neurophysiologic monitoring is not used. Arterial blood pressure preservation or augmentation can be accomplished by maintaining light levels of general anesthesia or by administering sympathomimetic drugs such as phenylephrine, ephedrine, or norepinephrine. Some caution must be exercised when using vasopressors to augment blood pressure during carotid endarterectomy because the increases in blood pressure and heart rate may increase myocardial ischemia¹⁶⁴ or infarction. The restrictive use of vasopressors for specific instances of cerebral ischemia has been advocated.¹⁶⁵ In one report, induced hypertension during the period of carotid occlusion was not associated with myocardial ischemia.¹⁶⁶

Surgical manipulation of the carotid sinus with activation of the baroreceptor reflexes can cause abrupt bradycardia and hypotension. Cessation of surgical manipulation promptly restores the hemodynamics, and infiltration of the carotid bifurcation with 1% lidocaine usually prevents further episodes. Infiltration may, however, increase the incidence of both intraoperative and postoperative hypertension.¹⁶⁷

On application of the surgical dressings, drugs used to reverse neuromuscular blockade (i.e., neostigmine or sugammadex) are administered and O_2 is increased to 100%. Ventilation is gently assisted until the patient exhibits spontaneously eye opening or movement. With rare exceptions, all tracheas are extubated after neurologic integrity is established. Neurologic deficits on emergence require immediate discussion with the surgeon about the need for angiography, reoperation, or both. The period of emergence and extubation may be associated with marked hypertension and tachycardia, which may require aggressive pharmacologic intervention. Tight hemodynamic control during this period is likely to be more demanding than during induction. Greater hemodynamic stability and decreased pharmacologic intervention during emergence have been reported in patients undergoing carotid

endarterectomy with propofol versus isoflurane. In addition, a significantly less frequent incidence of myocardial ischemia on emergence was found in the propofol group than in the isoflurane group (1 of 14 vs. 6 of 13). Of particular note, all patients with myocardial ischemia on emergence had systolic blood pressure higher than 200 mm Hg.

Regional and Local Anesthesia

Regional and local anesthetic techniques for carotid endarterectomy have been in use for more than 50 years, and many centers consider them to be the techniques of choice. Regional anesthesia is accomplished by blocking the C2 to C4 dermatomes by use of a superficial, intermediate, deep, or combined cervical plexus block (see also [Chapter 46](#)). Adequate anesthesia can be obtained with an isolated superficial or intermediate cervical plexus block, likely as a result of spread of local anesthetic to the cervical nerve roots.¹⁶⁸ Local infiltration of the incisional site and surgical field can also provide the necessary sensory blockade. A recent systematic review including over 10,000 cervical plexus blocks for carotid endarterectomy found that the deep (or combined) block was associated with a higher serious complication rate related to the injecting needle compared with a superficial (or intermediate) block (0.25% vs. 0%).¹⁶⁹ The conversion rate to general anesthesia was also higher with the deep block (2.1% vs. 0.4%). No difference was found in the incidence of serious systemic complications between the blocks. Although the incidence of serious complications from a cervical plexus block is infrequent, near-toxic levels of local anesthetic occurs in almost half of patients after superficial and deep cervical plexus block.¹⁷⁰ Although no major complications related to local anesthetic toxicity occurred, some caution should be exercised when requesting the surgeon to supplement with additional local anesthetic.

Regional and local anesthesia allows continuous neurologic assessment of the awake patient, which is widely considered to be the most sensitive method for detecting inadequate cerebral perfusion and function. Awake monitoring reduces the need for shunting and avoids the expense associated with indirect monitors of cerebral perfusion. Other advantages that have been reported include greater stability of blood pressure and decreased vasopressor requirements, reduced operative site bleeding, and reduced hospital costs. Potential disadvantages of local or regional anesthesia include an inability to use pharmacologic cerebral protection with anesthetics, patient panic or loss of cooperation, seizure or loss of consciousness with carotid clamping, and inadequate access to the airway should conversion to general anesthesia be necessary. The reported incidence of intraoperative neurologic changes during carotid endarterectomy under local or regional anesthesia varies widely (2.4% to 24%). Rates of conversion from regional anesthesia to general anesthesia of approximately 2% to 6% have been reported. Phrenic nerve paresis is common after cervical plexus block and is of little clinical consequence except in patients with severe COPD or contralateral diaphragmatic dysfunction.

Regional and local anesthesia requires significant patient cooperation throughout the procedure and is best maintained with constant communication and gentle handling

of tissues. Supplemental infiltration of local anesthetic by the surgeon, especially at the lower border and ramus of the mandible, is frequently helpful. Sedation, if used at all, must be kept to a minimum to allow continuous neurologic assessment. The surgical drapes are “tent” over the head and face area to minimize claustrophobic anxiety. Levels of consciousness, speech, and contralateral handgrip are assessed throughout the procedure. If both arms are tucked to the patient’s sides, handgrip can be assessed with the use of a squeaky toy. Blood pressure is augmented with phenylephrine when patients exhibit neurologic changes during carotid artery test clamping or after shunt placement. A 2- to 3-minute test clamp in awake patients allows prompt identification of those who would benefit from shunt placement. Patient acceptance of regional anesthesia is frequent and common, as evidenced by a 92% preference for repeat cervical plexus block for future carotid endarterectomy. Regional anesthesia should be avoided under the following circumstances: strong preference for general anesthesia expressed by the patient (i.e., claustrophobia), language barriers that make communication difficult, and difficult vascular anatomy. Difficult anatomy is usually manifested by a patient with a short neck and a high (more cephalad) bifurcation and may require vigorous submandibular surgical retraction.

Regional Versus General Anesthesia

For decades, the impact of anesthetic technique on outcome for carotid endarterectomy has been debated and studied. Until recently, nonrandomized studies dominated and largely supported that regional anesthesia was associated with significant reductions in the risk for perioperative death, stroke, MI, and pulmonary complications.¹⁷¹ The lack of randomized data was addressed with the landmark GALA trial.¹⁵⁴ This multicenter, randomized controlled trial included 3526 patients with symptomatic or asymptomatic internal carotid stenosis from 95 medical centers in 24 countries. Patients were randomly assigned to carotid endarterectomy under general anesthesia (1753 patients) or local anesthesia (1773 patients) between 1999 and 2007. The primary outcome was a composite of perioperative death, MI, and stroke (including retinal infarction). The main finding was that anesthetic technique was not associated with a significant difference in the composite end point (4.8% for general vs. 4.5% for local). Anesthetic technique was not associated with a significant difference in secondary outcomes, including duration of surgery, duration of ICU stay, length of hospital stay, or quality of life at 1 month after surgery. Other outcomes, including cranial nerve injury (10.5% vs. 12.0%), wound hematoma (8.3% vs. 8.5%), wound hematoma requiring reoperation (2.6% vs. 2.3%), and chest infection (2.0% vs. 1.9%) were similar between patients receiving general anesthesia and local anesthesia, respectively. Of note, 4.4% of patients under local anesthesia (93% received a cervical plexus block) had complications that lead to cancellation of surgery or conversion to general anesthesia. Important limitations of the GALA trial include lack of standardization, absence of blinding, and possible investigator bias. Using patient-level data from the GALA trial, a recent report found that local anesthesia had a cost-effectiveness benefit over general anesthesia.¹⁷²

Although randomized clinical trials such as GALA are considered to be the gold standard in clinical research, the conclusions are not always generalizable and therefore may not reflect actual practice in treating unselected patients. A recent report from a large international vascular registry, including 20,141 carotid endarterectomies performed in 10 countries between 2003 and 2007, found that anesthetic technique had no effect on perioperative mortality (0.5% overall) or stroke rate (1.5% overall).¹⁷³ These real-world results complement the data from the GALA trial. Thus, using major perioperative complications as a guide, there is no reason to routinely prefer one anesthetic technique over the other for carotid endarterectomy. The ultimate decision to use general anesthesia or regional anesthesia should be based on surgeon and the anesthesiologist experience and patient preference.

Carbon Dioxide and Glucose Management

Cerebrovascular CO₂ reactivity is part of the complex autoregulatory system to control cerebral blood flow. Normal cerebral autoregulation responds to acute changes in PaCO₂ by decreasing cerebral blood flow (i.e., vasoconstriction) with hypocapnia and increasing cerebral blood flow (i.e., vasodilatation) with hypercapnia. In patients with carotid artery stenosis or occlusion, ipsilateral cerebral blood flow may be impaired because of poor intracerebral collateral blood flow. In the setting of poor collateralization and resultant cerebral hypoperfusion, cerebral resistance vessels in the hypoperfused territories will dilate to maintain cerebral blood flow. These chronically dilated resistance vessels may demonstrate a diminished or absent (i.e., vasomotor paralysis) cerebral blood flow response to CO₂. Impaired cerebrovascular reactivity to hypercapnia may play a role in the development of stroke ipsilateral to carotid stenosis or occlusion. Although one might expect that impaired CO₂ reactivity would increase the risk for cerebral ischemia after carotid artery clamping, the results of intraoperative cerebral monitoring suggest that no such relationship exists. Impaired cerebrovascular reactivity to CO₂ will significantly improve after carotid endarterectomy.

Control of ventilation and end-tidal CO₂ (ETCO₂) during general anesthesia is a matter of some controversy. Hypercapnia may cause a “steal” phenomenon (i.e., shunting of blood away from hypoperfused territories with dilated vasculature) and is generally avoided. Hypocapnia, with its associated cerebral vasoconstriction, has been advocated to promote a reversal of this steal phenomenon. However, little clinical evidence exists for this “reverse” steal effect. Additionally, experimental data do not support the use of hypocapnia as a therapeutic maneuver to produce a favorable redistribution of blood flow during focal cerebral ischemia. Indeed, in this animal model of focal cerebral ischemia, hypocapnia (PaCO₂ of 23 mm Hg) actually increased the size of the region at risk for ischemia. It is therefore common practice to maintain normocapnia or mild hypocapnia during carotid endarterectomy.

Evidence demonstrates increased ischemic injury to neural tissue when ischemia occurs in the presence of hyperglycemia. Data from The Johns Hopkins Hospital found that operative-day glucose greater than 200 mg/dL at the time of carotid endarterectomy was associated with an increased risk for perioperative stroke or transient ischemic attack,

MI, and death.¹⁵⁶ Thus, until additional data become available, it may be beneficial to maintain a blood glucose level below 200 mg/dL in patients undergoing carotid endarterectomy. If hyperglycemia is treated with insulin preoperatively or intraoperatively, the blood glucose level should be carefully monitored, especially during general anesthesia, to avoid the dangers of hypoglycemia.

NEUROLOGIC MONITORING AND CEREBRAL PERFUSION

Intraoperative monitoring for cerebral ischemia or hypoperfusion and, more recently, for cerebral emboli during carotid endarterectomy is controversial (see also [Chapter 39](#)). Monitoring techniques include internal carotid artery stump pressure determinations, rCBF measurements, EEG monitoring, SSEP monitoring, transcranial Doppler ultrasonography (TCD), and cerebral oximetry monitoring. The rationale for the use of such monitoring is based on the need to prevent intraoperative strokes. The primary clinical utility of cerebral monitoring is to identify patients who may benefit from shunting during the period of arterial clamping. Secondarily, cerebral monitoring is used to identify patients who may benefit from blood pressure augmentation or change in surgical technique. Despite a tremendous amount of investigative effort, only limited data support the assumption that cerebral monitoring actually improves patient outcome after carotid endarterectomy. To further complicate the issue, several large series have reported excellent results from carotid endarterectomy with routine shunting, routine no shunting, and selective shunting using one or more of the methods discussed later. In a review, the mean reported stroke rate with routine shunting was 1.4% and for routine no shunting was 2%.¹⁷⁴ The mean perioperative stroke rates for selective shunting were 1.6% using stump pressure, 1.6% using EEG, 1.8% using SSEP, and 4.8% using TCD.¹⁷⁴

Carotid Artery Stump Pressure

The internal carotid artery stump pressure represents the back-pressure resulting from collateral flow through the circle of Willis via the contralateral carotid artery and the vertebrobasilar system. The advantages of monitoring carotid stump pressure are that it is inexpensive, relatively easy to obtain, and continuously available during carotid clamping (dynamic stump pressure). Despite these advantages, few centers use stump pressure monitoring. A recent single center report of 1135 consecutive carotid endarterectomies under general anesthesia used a stump pressure of below 45 mm Hg as a guide for selective shunting.¹⁷⁵ The 30-day stroke rate was 3% for patients selectively shunted (21%), 0.5% for patients not shunted (79%), and 1% overall. The overall 30-day mortality was 0.5%. Of note, no patient had a stroke caused by global intraoperative cerebral hypoperfusion. A recent prospective randomized trial comparing routine shunting versus selective shunting based on stump pressure below 40 mm Hg in 200 patients undergoing carotid endarterectomy under general anesthesia found both methods were associated with an infrequent perioperative stroke rate (0% vs. 2%).¹⁷⁶ The two strokes in the selective shunting cohort were related to carotid artery thrombosis. No patients died perioperatively. Although an old method, stump pressure monitoring appears to have survived the test of time.

Regional Cerebral Blood Flow

rCBF measurements during carotid endarterectomy are obtained by IV or ipsilateral carotid artery injection of radioactive xenon and analysis of decay curves obtained from detectors placed over the area of the ipsilateral cortex supplied by the middle cerebral artery. Measurements are typically obtained before, during, and immediately after carotid clamping. This technology, combined with the EEG monitoring, has provided important insight into the relationship between rCBF and EEG evidence of cerebral ischemia and the critical rCBF associated with various anesthetics.^{177,178} The critical rCBF varies depending on the volatile anesthetic used. In patients receiving N₂O plus a volatile anesthetic, rCBF is approximately 20, 15, 10, and 10 mL/100 g of brain tissue per minute for halothane, enflurane, isoflurane, and sevoflurane, respectively.^{161,177,178} The expense and the expertise required to make and interpret these blood flow measurements have limited the use of this technology to only a few centers.

Electroencephalography

Many centers advocate intraoperative use of EEG monitoring for the detection of cerebral ischemia and subsequent selective shunting (see also [Chapter 39](#)). The full 16-channel strip-chart EEG and the processed (compressed spectral array) EEG monitor are used for this purpose. Although the processed EEG monitor is more easily interpreted, it is less sensitive than the raw EEG monitor. Significant ischemic EEG changes occur in 7.5% to 20% of monitored patients during carotid clamping under general anesthesia. Significant EEG changes occur more frequently in patients with contralateral carotid disease than in those without (14.3% vs. 5.1%). The presence of a contralateral carotid occlusion may increase the rate of significant ischemic EEG changes to nearly 50% during carotid clamping. Because contralateral occlusion is highly predictive of ischemic EEG changes with carotid clamping, it has been recommended that EEG monitoring be eliminated in this circumstance. Ischemic EEG changes may also be seen with shunt malfunction, hypotension, or cerebral emboli.

When the electroencephalogram is used for cerebral ischemia monitoring during carotid endarterectomy, a stable physiologic and anesthetic milieu is mandatory. Isoflurane, desflurane, and sevoflurane produce similar ECG changes at equipotent levels and, when used at 0.5 MAC, allow for reliable ECG cerebral ischemia monitoring.

The clinical usefulness of intraoperative EEG monitoring for ischemia during carotid endarterectomy is limited by several factors. First, it may not detect subcortical or small cortical infarcts. Second, false-negative results (i.e., neurologic deficit with no ischemic EEG changes intraoperatively) are common. Patients with preexisting stroke or reversible neurologic deficits may have a particularly high incidence of such results. Third, EEG monitoring is not specific for ischemia and may be affected by changes in temperature, blood pressure, and anesthesia depth. Fourth, false-positive results (i.e., no perioperative neurologic deficit with significant ischemic EEG changes intraoperatively) occur because not all cerebral ischemia uniformly proceeds to infarction. Finally, intraoperative EEG monitoring is inherently limited because most intraoperative strokes are thought to be thromboembolic and most perioperative strokes occur

postoperatively. At present, no consistent data demonstrate that EEG monitoring is clearly superior to other methods of intraoperative cerebral monitoring or that the use of EEG monitoring improves outcome.

Somatosensory Evoked Potentials

SSEP monitoring is based on the response of the sensory cortex to electrical impulses from peripheral sensory nerve stimulation. The sensory cortex, being primarily supplied by the middle cerebral artery, is at risk during carotid artery clamping. SSEP monitoring, unlike EEG monitoring, can detect subcortical sensory pathway ischemia. Characteristic SSEP tracings (i.e., decrease in amplitude, increase in latency, or both) occur with decreased rCBF and are abolished in primates when flow decreases to less than 12 mL/100 g of brain tissue per minute. No specific reduction in amplitude or increase in latency has been established as a physiologic marker of impaired rCBF under operative conditions in humans. Anesthetics, hypothermia, and blood pressure may affect SSEPs significantly, and false-negative results have been reported. The validity of SSEPs as an intraoperative monitor of cerebral ischemia during carotid endarterectomy has not been definitively established.

Transcranial Doppler Ultrasonography

TCD allows continuous measurement of mean blood flow velocity and detection of microembolic events in the middle cerebral artery (see also [Chapter 39](#)). These parameters have important clinical implications because most perioperative neurologic deficits are thought to be thromboembolic in origin. With TCD, intraoperative embolization has been detected in more than 90% of patients undergoing carotid endarterectomy. Most intraoperative emboli are characteristic of air and are not associated with adverse neurologic outcomes. TCD may provide useful information regarding shunt function, malfunction, and the incidence of emboli during shunt insertion. Embolization during carotid artery dissection may indicate plaque instability and the need for early carotid artery clamping. Embolization during dissection and wound closure is associated with operative stroke. One center has reported that combined TCD monitoring and completion angiography resulted in a reduction in the intraoperative stroke rate from 4% to 0%. Early postoperative embolization has been detected in more than 70% of patients after carotid endarterectomy and is exclusively particulate in nature. Most TCD-detected emboli occur in the first 2 to 3 hours after surgery. Persistent particulate embolization in the early postoperative period has been shown to predict thrombosis and the development of a major neurologic deficit. Frequent early postoperative TCD embolic signals have been demonstrated to be highly predictive of early postoperative ipsilateral focal cerebral ischemia. Intervention with dextran has been shown to reduce and ultimately stop sustained embolization after carotid endarterectomy. Perioperative microembolization is more common in women and patients with symptomatic carotid disease. TCD monitoring has been reported to detect early asymptomatic carotid artery occlusion and hyperperfusion syndrome after carotid endarterectomy. Although TCD monitoring holds some promise, conclusive evidence demonstrating improved outcome has not been reported. Additionally, the high rate of technical failures significantly limits the clinical utility of this monitoring modality.¹⁷⁹

Cerebral Oxygenation

Direct monitoring of cerebral oxygenation can be obtained with jugular bulb venous monitoring. Such monitoring allows determination of the arterial-jugular venous O₂ content difference and jugular venous O₂ saturation and therefore provides information on global cerebral O₂ metabolism. Jugular venous samples are obtained from a catheter inserted into the jugular bulb ipsilateral to the surgical site. Continuous fiberoptic jugular venous oximetry catheters are available as well. Significant technical and methodologic shortcomings have limited the clinical application of this monitoring during carotid endarterectomy.

Near-infrared spectrophotometry is a noninvasive technique that allows continuous monitoring of regional cerebral O₂ saturation through the scalp and skull. Similar to pulse oximetry, cerebral oximetry is based on the different absorption characteristics of the near-infrared spectrum of oxygenated and deoxygenated hemoglobin. However, unlike pulse oximeters, cerebral oximeters measure the O₂ saturation of hemoglobin in the entire tissue bed (i.e., brain tissue and arterial and venous blood), which is predominately venous blood, and therefore approximates venous blood O₂ saturation. A commercially available cerebral oximetry sensor is applied to the forehead skin ipsilateral to the surgical site, and regional cerebral O₂ saturation from a small sample of the frontal cortex below the sensor is provided. To date, wide patient-to-patient variability in baseline cerebral O₂ saturation and the lack of a clinical threshold of a decrease in cerebral O₂ saturation predictive of the need for shunt placement have impeded the widespread use of this novel monitoring modality.

POSTOPERATIVE CONSIDERATIONS

Most neurologic complications (transient and permanent) after carotid endarterectomy are explained by intraoperative embolization, hypoperfusion during carotid clamping, and postoperative embolization or thrombosis from the endarterectomy site. It is generally accepted that most neurologic complications are related to surgical technique. Thromboembolic (rather than hemodynamic) factors appear to be the major mechanism of perioperative neurologic complications and most occur in the postoperative period. Neurologic complications attributable to carotid artery thrombosis may occur with an incidence as frequent as 3.3% and are associated with a high rate of major stroke or death despite immediate operative intervention. Other important, but less common neurologic complications include intracerebral hemorrhage and cerebral hyperperfusion. The reported incidence of intracerebral hemorrhage after carotid endarterectomy ranges from 0.4% to 2.0%. Most intracerebral hemorrhages occur 1 to 5 days after the operation and are associated with significant morbidity and mortality.

Hypertension is common in the postoperative period after carotid endarterectomy. Not surprisingly, patients with poorly controlled preoperative hypertension often have severe hypertension postoperatively. The causes are not well understood, but surgical denervation of the carotid sinus baroreceptors is probably contributory. Regional anesthesia is associated with less hypertension. Other causes of postoperative hypertension, such as hypoxemia,

hypercapnia, bladder distention, and pain, should be excluded or treated. Because neurologic and cardiac complications may be associated with postoperative hypertension, blood pressure should be aggressively controlled to near preoperative values after surgery. Short-acting drugs are the safest and most effective. Patients with persistent hypertension can be converted to longer-acting IV or oral agents before discharge from the ICU.

Postoperative cerebral hyperperfusion syndrome is an abrupt increase in blood flow with loss of autoregulation in the surgically reperfused brain and is manifested as headache, seizure, focal neurologic signs, brain edema, and possibly intracerebral hemorrhage. Unfortunately, little is actually known about the cause and management of this syndrome. Typically, this syndrome does not occur until several days after carotid endarterectomy. Patients with severe postoperative hypertension and severe preoperative internal carotid artery stenosis are believed to be at increased risk for this syndrome. However, more recent data do not corroborate this common belief and suggest that recent contralateral carotid endarterectomy may be predictive of cerebral hyperperfusion.¹⁸⁰

Postoperative hypotension occurs almost as frequently as hypertension after carotid endarterectomy. Carotid sinus baroreceptor hypersensitivity or reactivation probably plays an important role. Postoperative hypotension may be more common after regional anesthesia. To avoid cerebral and myocardial ischemia, hypotension should be corrected promptly. Cardiac output is frequently normal or elevated and systemic vascular resistance reduced in hypotensive patients after carotid endarterectomy. Intensive surveillance for evidence of myocardial and cerebral ischemia and judicious use of fluids and vasopressors are recommended for postoperative hypotension. In most cases, the hypotension resolves over a period of 12 to 24 hours.

Cranial and cervical nerve dysfunction after carotid endarterectomy is well documented in the literature. Although most injuries are transient, permanent injuries can lead to significant disability. Patients should be examined for injury to the recurrent laryngeal, superior laryngeal, hypoglossal, and marginal mandibular nerves shortly after extubation. Unilateral recurrent laryngeal nerve injury may result in ipsilateral true vocal cord paralysis in the paramedian position. Although most patients have hoarseness and an impaired cough mechanism, the injury is usually well tolerated. However, bilateral recurrent laryngeal nerve injury and resultant bilateral vocal cord paralysis can result in life-threatening upper airway obstruction. This situation must be anticipated in patients who have previously undergone contralateral carotid endarterectomy or neck surgery.

Carotid body denervation may occur after carotid endarterectomy as a result of surgical manipulation. Unilateral loss of carotid body function may result in an impaired ventilatory response to mild hypoxemia and is rarely of clinical significance. Bilateral carotid endarterectomy is associated with loss of the normal ventilatory and arterial pressure responses to acute hypoxia and an increased resting partial pressure of arterial CO₂. In this situation, the central chemoreceptors are the primary sensors for maintaining ventilation, and serious respiratory depression may result from opioid administration. Fortunately, most patients require little more than acetaminophen or ketorolac for postoperative pain.

Wound hematoma probably occurs more frequently than reported in the literature. In the NASCET,¹⁴⁵ 5.5% of patients had wound hematomas. Most cases are the result of venous oozing and require little more than external compression for 5 to 10 minutes. Expanding hematomas require prompt evaluation at the bedside and immediate evacuation if airway compromise is evident. Aggressive postoperative blood pressure control may help reduce the incidence of hematoma.

Although some clinicians believe that intensive care monitoring is not routinely required after carotid surgery, a significant number of patients do require intensive monitoring and active intervention. I think all patients should be monitored in an intensive care setting for at least 8 hours after carotid endarterectomy, because most events requiring intervention occur within this timeframe.^{181,182}

Endovascular Treatment of Carotid Disease: Carotid Artery Stenting

Endovascular treatment of carotid disease is an innovation in evolution for stroke prevention and currently involves percutaneous transluminal angioplasty and stenting. Significant procedural advancements include the use of dual antiplatelet therapy, self-expanding stents, and emboli protection devices. Over the last decade, major randomized clinical trials comparing carotid endarterectomy with carotid artery stenting have been published. A recent systematic review of randomized trials (16 trials involving 7572 patients) found that endovascular treatment (including balloon angioplasty or stenting) was associated with an increased risk for periprocedural stroke or death compared with endarterectomy.¹⁸³ Of note, the increase in risk appeared to be limited to patients 70 years of age and older. Endovascular treatment was associated with lower risks for MI, cranial nerve palsy, and access site hematomas. The rate of ipsilateral stroke after the periprocedural period was not different between treatment groups. Among patients unfit for surgery, the rate of death or stroke did not differ between endovascular treatment and medical care. Updated guidelines provide specific recommendations for revascularization of symptomatic and asymptomatic patients.¹⁴⁹

The carotid artery stenting procedure consists of the following steps: femoral access, aortic arch angiogram, selective cannulation of the common carotid artery origin and angiogram, guidewire advancement into the external carotid artery, carotid sheath placement and advancement into the common carotid artery, placement of embolic protection device, balloon angioplasty of lesion, advancement of stent delivery catheter across dilated lesion, deployment of self-expanding stent, balloon dilation of stent, completion angiogram, and access site management. The femoral artery approach is considered standard, but brachial artery and high radial artery access have been reported with high procedural success. Embolic protection devices are considered mandatory and include distal protection in the form of a filter or occlusion balloon and proximal protection in the form of flow interruption or flow reversal. Cardiologists and radiologists currently perform a large percentage of these procedures in specialized endovascular suites.

Most carotid artery stenting procedures are performed under local anesthesia with light or no sedation to facilitate patient cooperation and continuous neurologic assessment. In addition to routine monitors, an arterial line is placed for continuous blood pressure monitoring. Some degree of hemodynamic instability is common in patients during and after carotid artery stenting. Bradycardia and hypotension occur much more frequently after carotid artery stenting with balloon angioplasty than without angioplasty.¹⁸⁴ A recent, large retrospective study reported asystole in 4.9% of patients after carotid stenting.¹⁸⁵ Asystole was more likely to occur in patients undergoing a right-sided procedure, in those with significant contralateral stenosis, and in those with a reduced left ventricular ejection fraction. The administration of prophylactic atropine before balloon inflation decreases the incidence of intraoperative bradycardia and cardiac morbidity in primary carotid stenting patients.¹⁸⁶

Lower Extremity Revascularization

PERIPHERAL ARTERY DISEASE

The peripheral artery disease (PAD) or atherosclerotic occlusive disease of lower extremities is one of the resources-consuming diseases in the United States health care system.¹⁸⁷ The key risk associated with PAD is the amputation of lower extremity, and this disease also accounts for atherosclerosis in the renovascular, cerebrovascular, cardiovascular beds. These are the reasons the PAD patient is vulnerable to stroke, myocardial infarction (MI), and even death.¹⁸⁸ Studies also show that diabetic patients are at high risk of developing significant long-term disability.¹⁸⁹ Owing to these facts, Hirsch et al. highlighted that the PAD patients undergo expensive treatment due to a broad range of therapeutic procedures and diagnostic tests involved.¹⁹⁰

PERIOPERATIVE MANAGEMENT OF ANTIPLATELETS AND ANTICOAGULANT IN PAD PATIENTS

The long-term management of PAD patients includes an increasing use of effective antiplatelet and anticoagulant medications wherein managing these medications during the perioperative period becomes challenging due to the various risks and benefits.¹⁹¹ The increased risk of bleeding is one of the major concerns associated with perioperative management and if the antiplatelet therapy is paused, the patients are at higher risk of thrombotic complications due to the surgery-related prothrombotic effects.¹⁹²

Single Antiplatelet Therapy in Symptomatic Peripheral Artery Disease

According to Trialists Collaboration, the PAD patients showed 23% reduction in critical vascular events when they performed 42 randomized clinical trials and conducted a meta-analysis of 9214 patients who were prescribed single antiplatelet therapy (SAPT).¹⁹³ Another important study conducted by Mahmoud and associates includes an investigation regarding the efficacy of aspirin in both asymptomatic and symptomatic patients with peripheral vascular disease

and they concluded that neither the worse bleeding outcomes nor the improved cardiovascular results were strictly associated with the use of aspirin.¹⁹⁴ They concluded that there is a great need to conduct large-scale randomized trials to confirm the efficacy of aspirin in these conditions.

Dual Antiplatelet Therapy in Symptomatic Peripheral Artery Disease

Field and Benavente highlighted the Clopidogrel for High Atherothrombotic Risk and Ischemic Stabilization Management and Avoidance (CHARISMA) study wherein they compared the aspirin alone with aspirin plus clopidogrel with (75 to 162 mg/d) and (75 mg/d) in 15,603 patients who were affected with asymptomatic multiple vascular risk factors and with symptomatic vascular disease.¹⁹⁵ The results revealed no noticeable difference in both patients' groups, and as a matter of fact, an increased bleeding risk was shown in the patients.

Another study, the PEGASUS-TIMI 54 trial, "Prevention of Cardiovascular Events in Patients with Prior Heart Attack Using Ticagrelor Compared to Placebo on a Background of Aspirin-Thrombolysis in Myocardial Infarction" was conducted by Bonaca and associates, in which they studied the use of ticagrelor (a reversible P2Y12 inhibitor) in the patients with prior history of heart attack and the findings suggested that ticagrelor significantly reduced the risk of cardiovascular death, myocardial infarction, and strokes at the expense of an increased risk of major bleeding.¹⁹⁶

The Rivaroxaban for the Prevention of Major Cardiovascular Events in Coronary or Peripheral Artery Disease (COM-PASS) trial randomly assigned 27,395 participants with stable atherosclerotic vascular disease to receive rivaroxaban (a direct factor Xa inhibitor) (2.5 mg twice a day) plus aspirin (100 mg daily), rivaroxaban alone (5 mg twice a day), or aspirin alone (100 mg daily).¹⁹⁷ The primary outcome was a composite of cardiovascular death, stroke, or myocardial infarction. The trial was stopped because of an overwhelming efficacy. Total mortality, CAD mortality, and cardiovascular mortality were lowered by 20%. The COM-PASS trial provided a strong evidence for an alternative to clopidogrel when it comes to patients who are pharmacogenetically resistant to it (slow responders) due to genetic variations in the CYP2C19 enzyme responsible for its activation in the liver. It is assumed that 30% of patients may be pharmacogenetically resistant to clopidogrel.

OPEN BYPASS SURGERIES AND ANESTHESIA MANAGEMENT

Generally, the open bypass surgery and the endovascular revascularization are the two options to operate on PAD patients,¹⁹⁸ particularly when the patient doesn't show improvement after medical therapy and also those patients who are affected with critical limb ischemia.^{199,200} In this regard, due to the presence of significant comorbidities in these patients, the patients are at risk for major perioperative complications. While general anesthesia is usually used for open peripheral revascularization surgeries, regional and neuraxial anesthesia could also be employed.²⁰¹ They could add the advantage of postoperative pain control and improved perioperative hemodynamic stability. The ASRA guidelines for antithrombotic should be considered before

performing neuraxial or regional anesthesia, especially because moderate doses of heparin are used in distal bypass surgeries during arterial clamping.¹⁵⁵

ENDOVASCULAR TREATMENT OF PERIPHERAL ARTERIAL DISEASE

General anesthesia, neuraxial anesthesia, or MAC can be used for peripheral arterial stenting procedures since they are usually done through percutaneous punctures. We often use MAC unless there are other indications for different techniques. Like any other percutaneous intervention, the same indications and contraindications apply such as patient consent to awake or conscious sedation, patient's ability to lie flat for femoral access, and lying still for the duration of the procedure. What is different in those cases is that the percutaneous access could be affected by the disease process, for example, common femoral stenosis, which may need an open cutdown or even a combined common femoral endarterectomy before stenting the distal arteries. If this is the cases the choices for anesthesia will be limited to either general anesthesia or neuraxial anesthesia. Again, ASRA guidelines should be respected if neuraxial anesthesia is considered.¹⁵⁵ It should be noted that several research studies underline the fact that there is no noticeable difference among patients who receive either general anesthesia or neuraxial block in terms of cardiac outcomes.^{8,9}

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KEY POINTS

- For the purposes of planning a strategy for controlling intracranial pressure (ICP), the four subcompartments of the intracranial space should be considered: cells, interstitial and intracellular fluid, cerebrospinal fluid (CSF), and blood.
- The clinician should make a preoperative assessment of the probable intracranial compliance reserve as the basis for selection of appropriate anesthetic drugs and techniques.
- The venous side of the cerebral circulation is a largely passive compartment that is often the cause of increased ICP, or “tightness,” of the surgical field.
- Cerebral perfusion pressure (CPP) should be supported at or near normal waking levels in patients with recent cerebral injuries (e.g., traumatic brain injury [TBI], subarachnoid hemorrhage [SAH], and spinal cord injury [SCI]) because of low resting cerebral blood flow and impaired autoregulation.
- When neurosurgical procedures are performed in the sitting position, blood pressure should be corrected to the level of the external auditory meatus and mean arterial pressure (MAP) should be maintained at 60 mm Hg in normotensive adults.
- Monitoring for venous air embolism (VAE) in at-risk situations includes the precordial Doppler and end-tidal carbon dioxide analysis.
- Despite encouraging preclinical data, therapeutic mild hypothermia cannot be advocated in the care of head-injured patients in the intensive care unit (ICU) or during the operative management of patients with intracranial aneurysms because of negative human trial results for those patient groups.
- The most important consideration in the anesthetic management of patients undergoing clipping or coiling after acute SAH is the prevention of paroxysmal hypertension with its attendant risk of aneurysm rerupture. Nonetheless, adequate perfusion pressure is needed if temporary clips are used during management of a cerebral aneurysm.
- Although induced hypotension is rarely used electively in aneurysm surgery, the clinician must be ready to reduce blood pressure immediately and accurately in the event of aneurysm rupture.
- Tracheal intubation of a head-injured patient with an undefined cervical spine injury can be accomplished using rapid sequence induction with manual in-line stabilization (the occiput held rigidly to the backboard), with only a very small risk of injury to the spinal cord.
- CPP (CPP = MAP - ICP) should be supported to a target range of 60 to 70 mm Hg in the first 48 to 72 hours after TBI in adults.
- Hypocapnia has the potential to cause cerebral ischemia, particularly in a recently injured brain and in a brain beneath retractors; it should be used only when absolutely necessary for the control of critically increased or uncertain ICP.

This chapter provides guidelines for the management of common situations in neurosurgical anesthesia. Issues that arise in connection with a wide variety of neurosurgical procedures—those constituting a checklist that the practitioner should review before undertaking anesthesia for any neurosurgical procedure—are reviewed first, followed by

procedure-specific discussions. This chapter assumes familiarity with the cerebral physiology and effects of anesthetics as described in [Chapter 11](#), and with neurologic monitoring as described in [Chapter 39](#). Carotid endarterectomy (CEA) and carotid angioplasty and stenting are discussed in [Chapter 56](#).

Recurrent Issues in Neuroanesthesia

Several basic elements of neurosurgical and neuroanesthetic management are recurrent and, in the absence of an established understanding between surgeon and anesthesiologist, should be discussed and agreed upon at the outset of every neurosurgical procedure (Box 57.1). The list varies by procedure and may include the intended surgical position and requisite positioning aids; intentions with respect to the use of steroids, osmotic agents/diuretics, anticonvulsants, and antibiotics; the surgeon's perception of the "tightness" of the intracranial space and the remaining intracranial compliance reserve; appropriate objectives for the management of blood pressure, carbon dioxide tension, and body temperature; anticipated blood loss; the intended use of neurophysiologic monitoring (which may impose constraints on the use of anesthetics or muscle relaxants, or both); and, sometimes, the perceived risk of air embolism. The considerations driving the decisions made about these issues are presented in this section. One additional recurrent issue, brain protection, is discussed briefly in the section on aneurysms and arteriovenous malformations (AVMs) and in detail in Chapter 11.

CONTROL OF INTRACRANIAL PRESSURE AND BRAIN RELAXATION

The necessity of preventing increases in intracranial pressure (ICP) or reducing ICP that is already increased is recurrent in neuroanesthesia. When the cranium is closed, the objectives are to maintain adequate cerebral perfusion pressure (CPP) (CPP = mean arterial pressure [MAP] – ICP) and prevent the herniation of brain tissue between intracranial compartments or through the foramen magnum (Fig. 57.1).¹ When the cranium is open, the issue may be to provide relaxation of the intracranial contents to facilitate surgical access or, in extreme circumstances, reverse ongoing brain herniation through the craniotomy site. The principles that apply are similar whether the cranium is open or closed.

The various clinical indicators of increased ICP include headache (particularly headache that awakens the patient at night), nausea and vomiting, blurred vision, somnolence, and papilledema. Computed tomography (CT) findings suggestive of either increased ICP or reduced intracranial compliance reserve include midline shift, obliteration of the basal cisterns, loss of sulci, ventricular effacement (or enlarged ventricles in the event of hydrocephalus or ventricular trapping), and edema. Edema appears on a CT scan as a region of hypodensity. The basal cisterns appear on CT as a dark (hypodense fluid) halo around the upper end of the brainstem (Fig. 57.2). They include the interpeduncular cistern, which lies between the two cerebral peduncles, the quadrigeminal cistern, which overlies the four colliculi, and the ambient cisterns, which lie lateral to the cerebral peduncles.

Fig. 57.3 presents the volume-pressure relationship of the intracranial space. The plateau phase occurring at low volumes reveals that the intracranial space is not completely closed, which confers some compensatory latitude. Compensation is accomplished principally by the translocation of cerebrospinal fluid (CSF) and venous blood to the spinal CSF space and the extracranial veins, respectively.

BOX 57.1 Recurrent Issues in Neuroanesthesia

- Control of intracranial pressure/brain relaxation
- Management of PaCO_2
- Management of arterial blood pressure
- Use of steroids
- Use of osmotherapy
- Use of diuretics
- Use of anticonvulsants
- Patient positioning
- Pneumocephalus
- Venous air embolism
- Monitoring
- Intravenous fluid management
- Hypothermia
- Glucose management
- Emergence from anesthesia

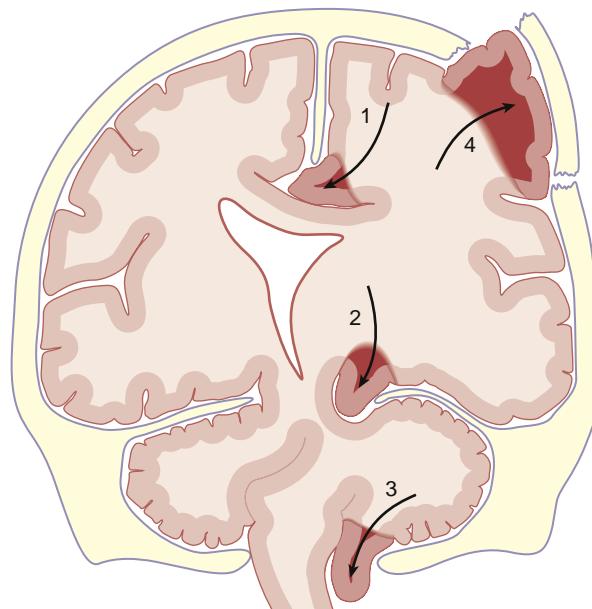


Fig. 57.1 Schematic representation of various herniation pathways. (1) Sub-falcine, (2) uncal (transtentorial), (3) cerebellar, and (4) transcalvarial. (From Fishman RA. Brain edema. *N Engl J Med*. 1975;293:706–711.)

Ultimately, when the compensatory potential is exhausted, even tiny incremental increases in volume can substantially increase ICP. These increases have the potential to result in either herniation of brain tissue from one compartment to another (or into the surgical field) (see Fig. 57.1), with resultant mechanical injury to brain tissue, or in reduction of perfusion pressure, leading to ischemic injury.

Several variables can interact to cause or aggravate intracranial hypertension (Fig. 57.4). For clinicians faced with the problem of managing increased ICP, the objective is, broadly speaking, to reduce the volume of the intracranial contents. For mnemonic purposes, the clinician can divide the intracranial space into four subcompartments (Table 57.1): cells (including neurons, glia, tumors, and extravasated collections of blood), fluid (intracellular and interstitial), CSF, and blood.

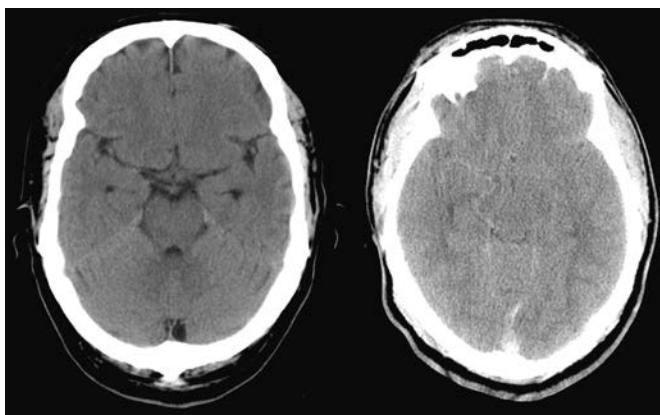


Fig. 57.2 Computed tomography scan depicting normal (left) and compressed (right) basal cisterns. The basal, or perimesencephalic, cerebrospinal fluid space consists of the interpeduncular cistern (anterior), the ambient cisterns (lateral), and the quadrigeminal cisterns (posterior). In the right panel, the cisterns have been obliterated in a patient with diffuse cerebral swelling (caused by sagittal sinus thrombosis). (Courtesy Ivan Petrovitch, MD)

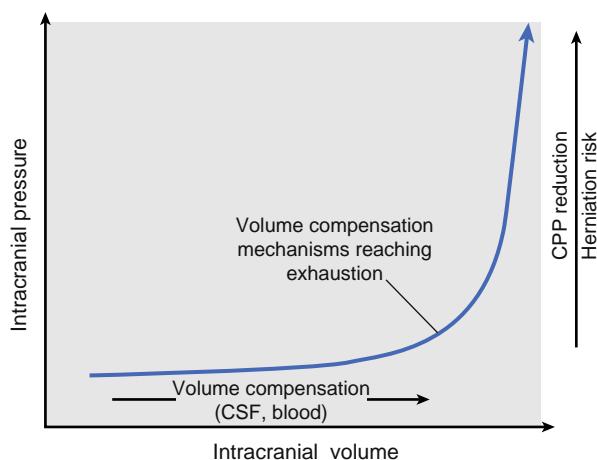


Fig. 57.3 The intracranial volume-pressure relationship. The horizontal portion of the curve indicates that there is initially some latitude for compensation in the face of an expanding intracranial lesion. That compensation is accomplished largely by displacement of cerebrospinal fluid (CSF) and venous blood from intracranial to extracranial spaces. Once the compensatory latitudes are exhausted, small-volume increments result in large increases in intracranial pressure with the associated hazards of herniation or of decreased cerebral perfusion pressure (CPP), resulting in ischemia.

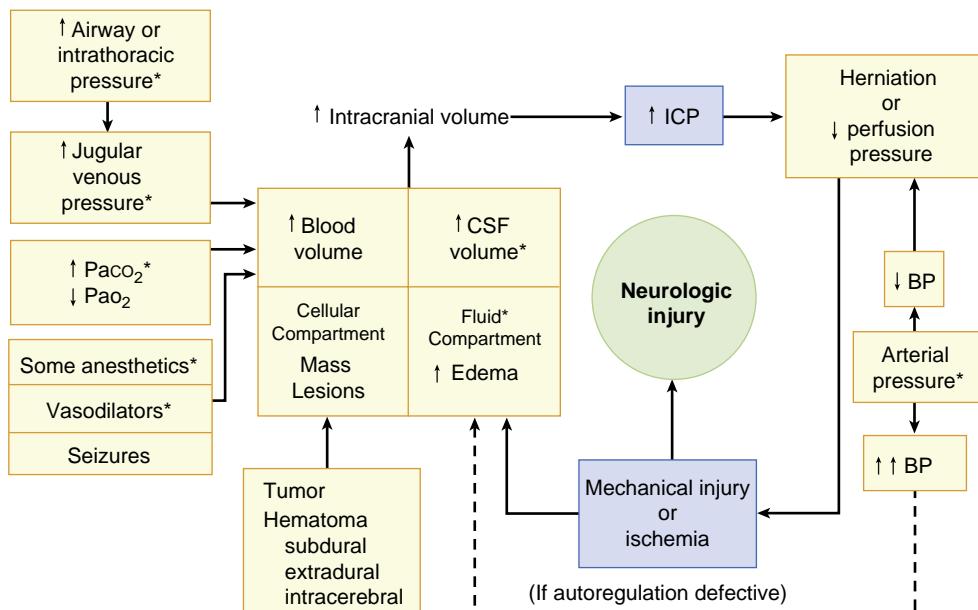


Fig. 57.4 The pathophysiology of intracranial hypertension. The figure depicts the manner in which increases in the volumes of any or all of the four intracranial compartments, blood, cerebrospinal fluid (CSF), fluid (interstitial or intracellular), and cells, result in intracranial pressure (ICP) increases and eventual neurologic damage. The elements that are most readily under the control of the anesthesiologist are indicated with asterisks (*). (Control of CSF volume requires the presence of a ventriculostomy catheter.) BP, Blood pressure; $PaCO_2$, partial pressure of carbon dioxide in the arterial blood; PaO_2 , partial pressure of oxygen in the arterial blood.

1. *The cellular compartment.* This compartment is largely the province of the surgeon. However, it may be the anesthesiologist's responsibility to pose a well-placed diagnostic question. When the brain is bulging into the surgical field at the conclusion of evacuation of an extra-axial hematoma, the clinician should ask whether a subdural or extradural hematoma is present on the contralateral side that warrants either immediate burr holes or immediate postprocedure radiologic evaluation.
2. *The CSF compartment.* There is no pharmacologic manipulation of the CSF space with a time course and magnitude that is relevant to the neurosurgical operating room. The only practical means of manipulating the size of this compartment is by drainage. A tight surgical field can sometimes be improved by passage of a brain needle by the surgeon into a lateral ventricle to drain CSF. Lumbar CSF drainage can be used to improve surgical exposure in situations with no substantial hazard of uncal or transforaminal magnum herniation.