

## The Aorta

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### OUTLINE

- Aneurysmal Disease
- Aortoiliac Occlusive Disease
- Aortic Dissection

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The aorta is a broad topic encompassing the diagnosis and management of aneurysms, occlusive disease, and dissections of the abdominal and thoracic aorta. In the past 2 decades, endovascular therapy has offered a frequently less morbid approach to each of these disease entities. The rapid adoption of endovascular techniques and technologies has clearly revolutionized the management of aortic disease. Endovascular aneurysm repair (EVAR) is now performed much more frequently than open repair and appears to have had an impact on the mortality rate attributable to aortic aneurysm.<sup>1</sup> Thoracic EVAR (TEVAR) is now the recommended first treatment option. The new TransAtlantic Inter-Society Consensus guidelines (TASC III) will recommend endovascular therapy as the first option for almost all degrees of aortoiliac occlusive disease (AIOD). Nevertheless, 2013 data suggest that aortic disease is identified as the cause of nearly 10,000 deaths per year,<sup>2</sup> and others have noted that this number is likely to be significantly higher because of the failure to identify aortic disease in deaths that occur out of the hospital and without autopsy.

We have tried to make this chapter relevant to general surgery residents training in the second decade of the 21st century, with particular attention to the fact that with a shift toward endovascular therapy, the exposure of surgical residents to open reconstructive techniques for management of both the thoracic and the abdominal aorta has declined noticeably. Relatively few centers still offer rich experience in open aortic surgery and, in particular, the most complex cases, and yet mastery of aortic surgery remains a necessity. Dense calcium, involvement of visceral vessels, infections, trauma, small arteries, and failed endografts may and do necessitate formal open reconstruction. We hope that concurrent changes in the training regimen not only will allow the maintenance of standards with regard to the surgeon's skill set and outcomes but also will enable future generations to continue to drive advances in the state of the art of vascular surgery.

### ANEURYSMAL DISEASE

Aneurysms, typically defined as an increase in size of more than 50% above the normal arterial diameter, may occur anywhere along the aorta, from the aortic root to the bifurcation. Aneurysms may be further characterized on the basis of anatomy or etiology. Anatomically, fusiform aneurysms exhibit smooth, circumferential dilation as opposed to saccular aneurysms, which, as their name suggests, appear as a focal outpouching of the arterial wall. Whereas true aneurysms involve all three layers of the vessel wall, false aneurysm or pseudoaneurysm describes a focal defect in the artery with an associated collection of blood contained by adventitia and periarterial tissue; it may be degenerative, infectious, or traumatic in etiology. The majority of aneurysms addressed in this chapter are degenerative in nature. Less frequently, aneurysms may be associated with infection (mycotic aneurysms), inflammation, or autoimmune or connective tissue disease. These cases merit special consideration in their evaluation and management. Aneurysmal enlargement of the aorta is associated with factors that result in weakening of the arterial wall and increased local hemodynamic forces. These may include heritable conditions, such as Marfan syndrome, familial thoracic aortic aneurysm and dissection, and vascular-type Ehlers-Danlos, as well as less well defined entities that contribute to the significantly elevated incidence of aneurysm in patients with a family history of aneurysm. Factors that contribute to the degradation of collagen and elastin are also associated with aneurysmal disease, and research in this area has focused on the role of matrix metalloproteinases and other mediators of tissue enzyme function. Ongoing avenues of investigation in this area also include the role of the immune response and hormone milieu.<sup>3</sup> Aneurysms do also occur as a degenerative complication after aortic dissection.

The incidence of abdominal aortic aneurysm (AAA), based on large screening studies, is estimated to range from 3% to 10%. A number of risk factors, in addition to genetic or familial disorders, for the development, expansion, and rupture of AAAs have been identified (Table 61-1). Risk factors for development of an AAA include age, male gender, concurrent aneurysms, family history,

**VIDEO**

Video 61-1: Total Aortic Replacement

**TABLE 61-1 Risk Factors for Aneurysm Development, Expansion, and Rupture**

SYMPTOM	RISK FACTORS
AAA development	Tobacco use
	Hypercholesterolemia
	Hypertension
	Male gender
	Family history (male predominance)
AAA expansion	Advanced age
	Severe cardiac disease
	Previous stroke
	Tobacco use
	Cardiac or renal transplantation
AAA rupture	Female gender
	↓ FEV <sub>1</sub>
	Larger initial AA diameter
	Higher mean blood pressure
	Current tobacco use (length of time smoking >> amount)
Cardiac or renal transplantation	Critical wall stress–wall strength relationship

Adapted from Chaikof EL, Brewster DC, Dalman RL, et al: The care of patients with an abdominal aortic aneurysm: The Society for Vascular Surgery practice guidelines. *J Vasc Surg* 50:S2–S49, 2009.

tobacco use, hypertension, hyperlipidemia, and height. Female gender, black race, and diabetes appear to be protective.<sup>4–14</sup>

Gender differences extend to the presentation, associations, and natural history of aneurysms. Men with AAA, for instance, are more likely to present with concurrent iliac or femoropopliteal aneurysms.<sup>15</sup> Women are more likely to experience rupture and consistently demonstrate poorer outcomes after repair, perhaps because of a significantly higher incidence of challenging anatomy.<sup>16,17</sup>

### Risk of Rupture

Predicting the behavior of an aneurysm over time is difficult. Published risk factors for rupture include chronic obstructive pulmonary disease (COPD), current tobacco use, larger initial AAA diameter, female gender, cardiac or renal transplantation, and certain patterns of wall stress.<sup>4,18–26</sup>

The most widely adopted surrogate for rupture risk is maximal cross-sectional aneurysm diameter (Table 61-2), although the implications for rupture risk of a particular aortic diameter remain debated. Some data suggest that surgeons tend to overestimate rupture risk.<sup>27</sup> In addition, an observational study suggested that even broadly accepted estimates of risk may overstate the rupture rates of untreated AAA and noted, in patients deemed medically unfit for elective repair, that the risk of death from non–aneurysm-related causes exceeded the risk of death from rupture.<sup>28</sup>

In addition, despite a relative paucity of natural history data regarding growth rate and rupture, most clinicians do consider the rate of enlargement a risk factor for rupture. A rate of growth of more than 5 mm in 6 months or more than 1 cm per year has been widely adopted as an indication for repair, independent of aneurysm size. Size is an imperfect predictor of rupture risk; autopsy studies have discovered evidence of rupture in up to 12% of aneurysms less than 5 cm in diameter.<sup>29</sup> A number of investigational models attempt to quantify rupture risk by calculations

**TABLE 61-2 Estimated Annual Rupture Risk**

AAA DIAMETER (cm)	RUPTURE RISK (%/yr)
<4	0
4-5	0.5-5
5-6	3-15
6-7	10-20
7-8	20-40
>8	30-50

Adapted from Brewster DC, Cronenwett JL, Hallett JW Jr, et al: Guidelines for the treatment of abdominal aortic aneurysms. Report of a subcommittee of the Joint Council of the American Association for Vascular Surgery and Society for Vascular Surgery. *J Vasc Surg* 37:1106–1117, 2003.



**FIGURE 61-1** Gray-scale cross-sectional ultrasound image of an infrarenal aortic aneurysm measuring 6.19 cm in maximal anteroposterior diameter.

of wall stress, observation of particular wall or thrombus characteristics, or the combination of multiple factors thought to contribute to increased wall stress or decreased strength.

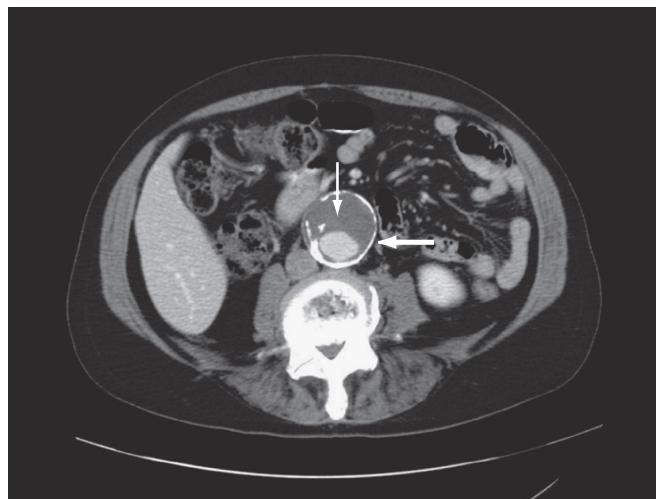
### Diagnosis

Abdominal aneurysm may be detected on physical examination as a palpable pulsatile mass, most commonly supraumbilical and in the midline. The location may be variable, however, as aortic tortuosity can result in a lateral or infraumbilical location. The sensitivity of physical examination is, as one might expect, dependent on the aneurysm's size and the patient's habitus.

The detection and characterization of aneurysms are greatly aided by modern imaging techniques. Ultrasound examination has been demonstrated to afford excellent sensitivity and specificity (Fig. 61-1). Ultrasound may be limited by the patient's habitus or bowel gas, but as it avoids the complications associated with invasive testing, radiation, and contrast media, it is an excellent choice for screening. Ultrasound is not an ideal method for detecting rupture; it is unable to image all portions of the aortic wall, and the nonfasting status of emergently examined patients may further preclude ideal image acquisition. It has been estimated that ultrasound may fail to detect up to 50% of aneurysm ruptures.

Computed tomography (CT) provides excellent imaging of AAA, with greater reproducibility of diameter measurements than by ultrasound. CT, particularly with the adjunctive use of iodinated contrast agents to perform CT angiography (CTA), provides a wealth of anatomic information; it detects vessel calcification, thrombus, and concurrent arterial occlusive disease and permits multiplanar and three-dimensional reconstruction and analysis for operative planning (Fig. 61-2). Drawbacks include substantial radiation exposure, particularly in the setting of serial examinations, and the use of iodinated contrast media in a population with a high incidence of comorbid kidney disease.<sup>4</sup>

Magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) are, like CT, sensitive in the detection of AAA (Fig. 61-3). Unlike CT, MRI does not demonstrate aortic



**FIGURE 61-2** CTA axial plane image of an infrarenal abdominal aortic aneurysm demonstrating aortic wall calcification (thick arrow) and intraluminal thrombus (thin arrow).



**FIGURE 61-3** MRA coronal view of an infrarenal aneurysm (arrow).

wall calcification, which may be important in operative planning. Although the study does not require the use of iodinated contrast material, MRA uses gadolinium, which has been associated with the development of nephrogenic systemic fibrosis in patients with low glomerular filtration rate. The availability of MRI may also be limited by the presence of incompatible metallic implants or foreign bodies. The ability to acquire dynamic images throughout the cardiac cycle may ultimately prove clinically useful.<sup>30</sup>

### Screening and Surveillance Recommendations

Screening recommendations for AAA are informed by the sensitivity and specificity of ultrasound screening, the detection yield of screening based on various risk factor selection criteria, and cost. A major recent compilation of evidence-based recommendations for screening and surveillance of AAA is provided by the 2009 Practice Guidelines developed by the Clinical Practice Council of the Society for Vascular Surgery. The Society for Vascular Surgery committee charged with reviewing available data regarding screening made a strong recommendation for one-time screening of all men aged 65 years and older or men 55 years and older with a family history of AAA. Screening of women is also strongly recommended for those aged 65 years and older with a family history of AAA or a personal smoking history. The evidence basis of these recommendations was deemed to be strong in the former case and moderate in the latter.<sup>4</sup>

The U.S. Preventive Services Task Force issued a more limited recommendation for one-time screening of men between 65 and 75 years of age who have a personal smoking history.<sup>31</sup>

Screening of women remains controversial. Although there is evidence that women may exhibit a stronger association between smoking and aneurysm, it is known that the incidence of aneurysm in women who have smoked exceeds that of men who have never smoked, and mortality data reflect that gender differences in aneurysm-related mortality narrow with advanced age.<sup>4</sup> Payer policies regarding reimbursement may not track either of these recommendations. Medicare, for instance, as a result of the Screening Abdominal Aortic Aneurysms Very Efficiently (SAAVE) Act, reflects an intermediate approach in offering a screening benefit for men with a personal smoking history and men or women with a family history of AAA, although only as a part of the initial Welcome to Medicare physical examination.

Once an aneurysm has been detected, the Society for Vascular Surgery Clinical Practice Council recommends further screening intervals as follows, based on aneurysm size (maximum external aortic diameter) and associated risk of rupture<sup>4</sup>:

- <2.6 cm: no further screening recommended
- 2.6-2.9 cm: reexamination at 5 years
- 3-3.4 cm: reexamination at 3 years
- 3.5-4.4 cm: reexamination at 12 months
- 4.5-5.4 cm: reexamination at 6 months

The recommendation for follow-up of aortic diameters less than 3 cm is controversial and has been criticized on the basis of cost-effectiveness analyses. It is based on findings that a significant proportion of 65-year-old men (13.8%) with an initial aortic diameter of 2.6 to 2.9 cm developed aneurysms exceeding 5.5 cm at 10 years. Given current life expectancy projections, it is evident that a subset of patients deemed "normal" at screening will go on to develop large aneurysms.<sup>32</sup>

### Medical Therapy

Once an aneurysm has been diagnosed, the optimization of medical therapy serves a dual purpose: to potentially minimize

the rate of aneurysm expansion or rupture and to medically prepare for potential repair. Many avenues have been investigated in the search for effective medical treatment to prevent the progression of aortic aneurysm, leading to a recent editorial statement that “the bottom line is that no drug can currently be recommended for the indication of reducing AAA enlargement.”<sup>33</sup> A highly anticipated, randomized trial of doxycycline, an antibiotic and inhibitor of matrix metalloproteinase activity, not only failed to demonstrate benefit but found increased AAA enlargement primarily in the first 6 months of follow-up.<sup>34</sup> Investigation is ongoing, particularly with regard to the role of anti-inflammatory agents.

Currently, many continue to incorporate beta blockade in an effort to control blood pressure and dP/dT that may contribute to harmful wall stress. Studies using propranolol demonstrated mixed results and low patient compliance. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers have yielded mixed results in clinical studies, but their use is based on the goal of blood pressure management as well as on evidence for their utility in the management of patients with aortic disease associated with Marfan syndrome.<sup>35</sup> HMG-coenzyme A reductase inhibitor (statin) therapy has been associated with reduced rates of AAA enlargement and is otherwise appropriate in a population with a high prevalence of concurrent atherosclerotic disease. Anti-platelet therapy using aspirin does, like beta blockers and statins, offer secondary preventive benefit in this population and should be considered. Perhaps the most important intervention, in both regards, is smoking cessation. Current tobacco use has been associated with an increased rate of aneurysm expansion. Smoking cessation may also yield benefits with regard to perioperative morbidity and mortality in the event that the aneurysm ultimately requires repair.

### Surgical Treatment

Surgical treatment is generally recommended for aneurysms more than 5.5 cm in maximal diameter, those demonstrating more than 5 mm of growth in 6 months or more than 1 cm in a year, and aneurysms with a saccular rather than the typical fusiform anatomy. Gender differences in a variety of factors have led some to advocate for consideration of aneurysm repair at a smaller size in women. It has been observed that the average size of “normal” aorta tends to be slightly smaller in women. Evaluation of several indices relating aortic diameter to body build, such as body surface area or wrist circumference, also suggests that measures other than aortic diameter alone may be more accurate in predicting AAA.<sup>36</sup> These observations are consistent with evidence suggesting more rapid aneurysm growth and rupture at smaller sizes in women (average diameter of 5 cm rather than one of 6 cm in male patients).<sup>37,38</sup> The presence of significant aneurysm-related anxiety associated with awareness of the presence of an unrepaired aneurysm has also been cited as affecting quality of life and presenting a potential consideration in managing aneurysms below 5.5 cm in diameter.<sup>38</sup>

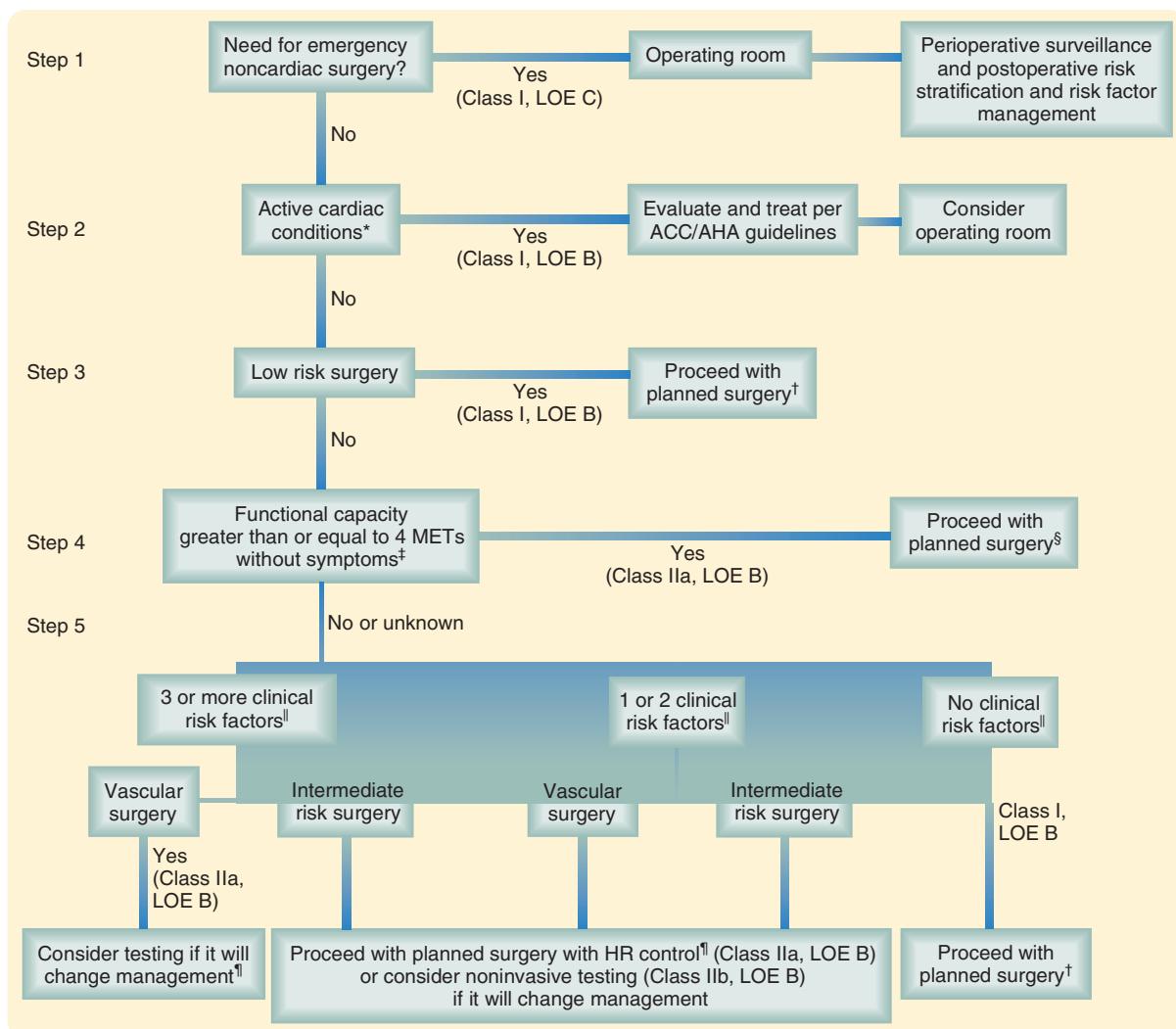
### Preoperative Evaluation

The preoperative evaluation of patients with AAA comprises operative planning as well as identification and management of important medical comorbidities, such as coronary artery disease, renal insufficiency, peripheral arterial occlusive disease, diabetes, and obstructive lung disease. As coronary artery disease is the primary cause of mortality after either open or endovascular repair of AAA, a great deal of attention has been focused on the preoperative

evaluation and management of comorbid coronary artery disease. The guiding principles in this evaluation have traditionally been the identification of information that will alter management and the institution of therapy that will improve cardiac-related mortality. In 2007, the American College of Cardiology/American Heart Association (ACC/AHA) published guidelines regarding the pre-operative cardiac evaluation of patients undergoing noncardiac vascular surgery.<sup>39</sup> These guidelines stratify patients according to the presence or absence of symptomatic cardiac disease, the presence of significant clinical risk factors (mild angina, prior myocardial infarction, compensated congestive heart failure, diabetes mellitus, or renal insufficiency), and the level (quantified in metabolic equivalents) of the patient’s functional capacity. Resting electrocardiography is typically performed before high-risk surgery, such as open aneurysm repair, but it is no longer recommended by the ACC/AHA or European Society of Cardiology for patients without clinical risk factors who are undergoing low-risk surgery. Echocardiography may be used to evaluate the cardiac function of those with a history of heart failure or current dyspnea. Heart failure is a significant consideration; left ventricular ejection fraction less than 35% has been found to have 50% sensitivity and 91% specificity for predicting perioperative cardiac events.<sup>40</sup>

The decision to proceed with noninvasive testing in patients without symptoms of active cardiac disease should be based on the patient’s functional capacity and the presence of three or more significant additional risk factors. Coronary angiography should be considered in patients with evidence of active cardiac disease based on screening questions or evidence of ischemia on noninvasive stress testing. Adjunctive medical therapy may also serve to reduce the risk of perioperative cardiac events. Perioperative beta blockade, statin use, and aspirin use are widely accepted, and there is also evidence to support the use of other antihypertensives during this period (Fig. 61-4). An important caveat has been added to the perioperative use of beta blockade, with the 2009 American College of Cardiology Foundation/American Heart Association Focused Update on Perioperative Beta Blockade advising continuation of previously prescribed beta blockers in the perioperative period and titration of beta blockers to desired heart rate and blood pressure, noting that routine perioperative high-dose beta blockers without titration may be harmful.<sup>41</sup> The withdrawal of statins in the perioperative period may be associated with an increased risk of coronary events.<sup>42</sup>

Renal insufficiency related to renovascular or medical renal disease is a well-established risk factor for morbidity and mortality after AAA repair. Coexisting renal artery occlusive disease may be present in 20% to 38% of patients with AAA.<sup>43</sup> In addition, both open and endovascular repair of AAA may result in further deterioration in the renal function of patients with preexisting renal disease. Concurrent repair of clinically significant renal occlusive disease is appropriate at the time of either open repair or EVAR. A number of strategies for intraoperative renal protection have been proposed. Current recommendations include adequate hydration, perioperative discontinuation of angiotensin-converting enzyme inhibitors and angiotensin receptor blockers, and avoidance of hypotension. There is also evidence of increased perioperative mortality associated with postoperative nonresumption of angiotensin-converting enzyme inhibitors.<sup>44</sup> There is mixed evidence regarding the benefits of antioxidants (mannitol, ascorbic acid, vitamin E, N-acetylcysteine, and allopurinol) and some data supporting the beneficial effects of infused fenoldopam.<sup>45,46</sup> When suprarenal clamp placement is necessary, the



**FIGURE 61-4** Cardiac evaluation and care algorithm for noncardiac surgery based on active clinical conditions, known cardiovascular disease, or cardiac risk factors for patients 50 years of age or older. (From Fleisher LA, Beckman JA, Brown KA, et al: ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines [Writing Committee to Revise the 2002 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery]: Developed in collaboration with the American Society of Echocardiography, American Society of Nuclear Cardiology, Heart Rhythm Society, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, and Society for Vascular Surgery. *Circulation* 116:e418–e499, 2007.)

authors endorse the use of cold saline perfusion of the kidneys, preclamp administration of furosemide and mannitol, and selective use of fenoldopam. An additional consideration, particularly in patients with preexisting renal dysfunction, is contrast-induced nephropathy associated with administration of iodinated contrast

agents for CT imaging or angiography. Current data support intravenous hydration with sodium bicarbonate or normal saline and possibly the use of antioxidants, such as ascorbic acid or *N*-acetylcysteine. When EVAR is contemplated, carbon dioxide may be used as an imaging agent to alleviate or to minimize the

need for iodinated agents as the rate of contrast-induced nephropathy is related to the amount of agent administered as well as age and prior renal function.

Data are mixed with regard to the impact of pulmonary disease, particularly COPD, on mortality after AAA repair. However, there is evidence that optimal management of comorbid COPD may improve morbidity and mortality.<sup>47</sup> The authors support obtaining a preoperative pulmonary function assessment, including arterial blood gases, to assess risk and to guide management in the perioperative period. Patients with poor pulmonary function must be made aware of the increased risk that they will require prolonged ventilatory support postoperatively and the attendant possibility that tracheostomy will be required during this period. Smoking cessation before surgery may be beneficial and can be aided by counseling and a variety of pharmacologic therapies. Although several studies have suggested that initiating smoking cessation less than 2 weeks before surgery may actually be associated with worse outcomes, a meta-analysis suggests that smoking cessation at any time within 8 weeks of surgery is not associated with a higher rate of either overall complications or pulmonary complications postoperatively.<sup>48</sup>

The preoperative evaluation should also include a chest radiograph, complete blood count, blood chemistries, and coagulation studies as well as urinalysis. The chest radiograph may demonstrate evidence of infection, thoracic aortic disease, or malignant disease, all of which should be thoroughly investigated before AAA repair. The use of various anticoagulant agents is common in patients with AAA, and management is tailored to the indication for use. Vitamin K antagonists should be stopped 5 to 7 days before surgery and bridging anticoagulation provided, if indicated, with low-molecular-weight or unfractionated heparin. Thienopyridines are typically stopped 7 to 10 days before surgery, although patients receiving thienopyridine therapy for drug-eluting coronary stents necessitate careful consideration of the merits of delaying surgery until therapy is discontinued in light of the additional bleeding risk associated with these drugs. Aspirin is typically continued perioperatively as it may confer some degree of benefit with regard to cardiac complications in the perioperative period.

Careful evaluation of preoperative imaging is crucial in planning repair. Anatomic variations, such as a retroaortic renal vein, variant inferior vena cava, or horseshoe kidney, may significantly affect the selection of surgical approach and, if not appreciated preoperatively, can lead to disastrous complications. CT imaging affords the additional advantage of demonstrating vascular calcification, thus permitting the surgeon to assess the feasibility of clamping the aorta and iliac arteries at various levels (Fig. 61-5). Occlusion balloons may be substituted for arterial clamp placement, most frequently at the iliac arteries, should severe calcification render clamp placement untenable. Finally, the size and patency of branch vessels, such as the inferior mesenteric, accessory renal, iliac, and lumbar arteries, can be assessed and may further contribute to preoperative planning.

### Technique of Open Surgical Repair of Abdominal Aortic Aneurysms

Open surgical repair of AAA may be accomplished by either a transperitoneal or a retroperitoneal approach. The choice of technique is guided by technical advantages and disadvantages afforded by each as well as by the surgeon's experience and preference. Transperitoneal repair through a midline laparotomy incision is the most widely used approach to the usual infrarenal aneurysm



**FIGURE 61-5** Reconstruction of coronal CTA image demonstrating heavy calcification (arrows) extending from above the renal arteries distally through both common iliac arteries.

and offers a rapid exposure, excellent access to renal and iliac vessels, and the ability to fully examine the abdominal contents. Adjunctive measures to improve exposure at or above the level of the renal arteries may include ligation and division of the tributaries (gonadal, lumbar, and adrenal) of the left renal vein, if the vein is to be preserved, or division of the proximal left renal vein itself. Although data are mixed regarding the effect of left renal vein ligation on postoperative renal function, it is essential that these tributaries be preserved to provide collateral outflow should renal vein ligation be planned. Alternatively, repair of the left renal vein after ligation has been reported.

The infrarenal transperitoneal repair begins with the perioperative administration of an antibiotic, typically a first-generation cephalosporin, and scrupulous skin preparation from the nipples to the thighs. If a ruptured aneurysm is being treated, skin preparation and draping are accomplished before the induction of general anesthesia to permit rapid exposure and control should induction incite hemodynamic collapse. The patient is draped, and a generous midline laparotomy incision is made from the xiphoid to just above the pubis. Extension of this incision along the xiphoid may facilitate supraceliac exposure, if necessary. If repair is elective and preoperative imaging has demonstrated iliac disease necessitating extension of a bifurcated graft to the femoral artery on one or both sides, the femoral artery dissection should be accomplished before laparotomy (Fig. 61-6).

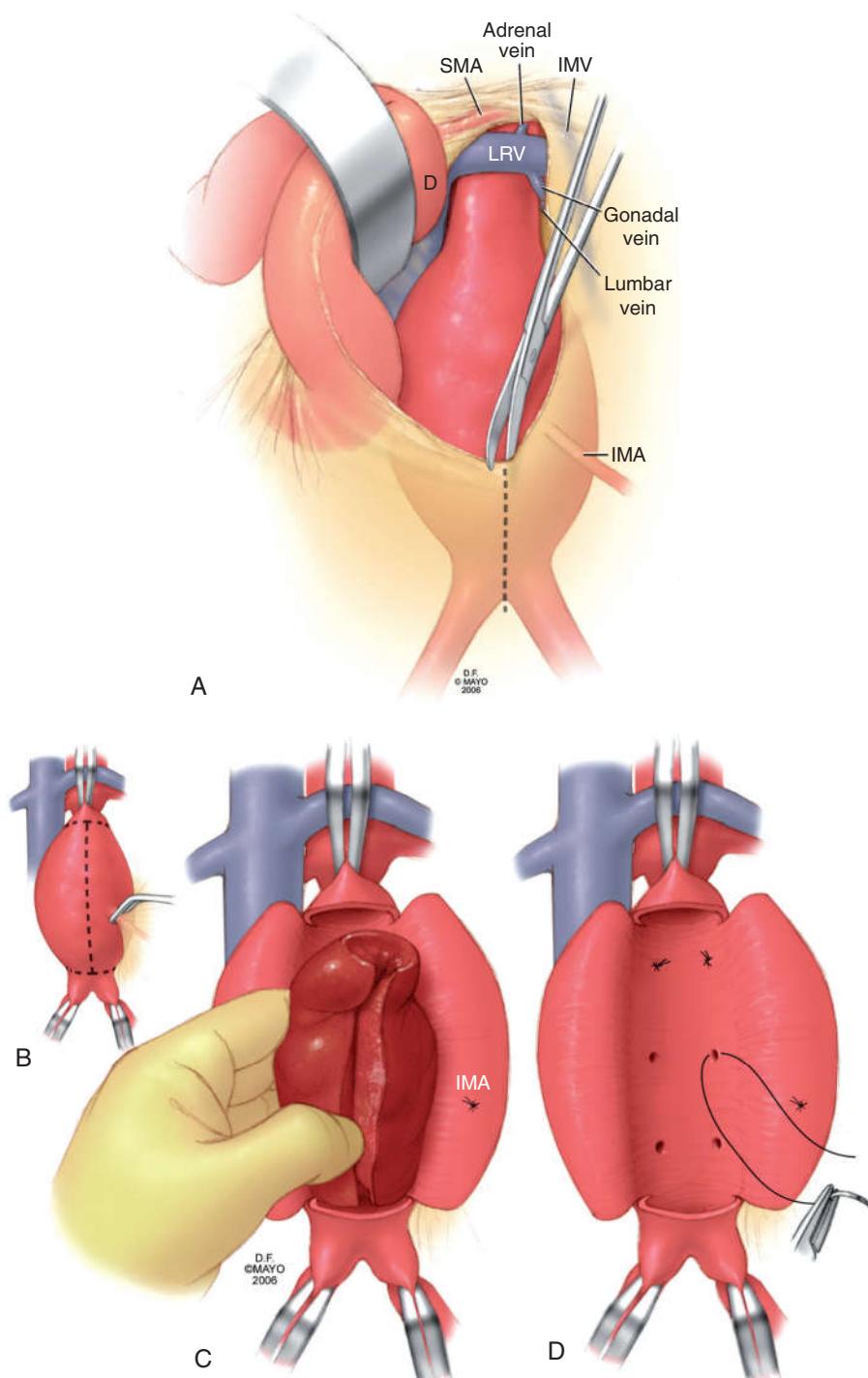
If supraceliac clamp placement is anticipated, the left lobe of the liver is mobilized by division of the triangular ligament, and the esophagus is identified and reflected to the patient's left. Placement of a nasogastric tube facilitates identification and protection of the esophagus. The crural fibers of the diaphragm are divided proximal to the celiac artery to provide adequate exposure and mobilization of the aorta for supraceliac clamp placement. Once these steps have been accomplished, the neck of the aneurysm may be approached. In some cases, the proximal clamp may be moved down to a suprarenal or infrarenal position at this stage, permitting perfusion of visceral and, ideally, renal vessels.

The surgeon may then proceed with iliac dissection and clamp placement.

The surgeon's preference guides the decision with regard to intravenous administration of heparin in rupture. Typical systemic administration of heparin in the elective setting consists of

100 units/kg, administered intravenously and permitted to circulate before clamp placement.

Elective repair permits controlled exposure of the iliac arteries and aneurysm neck before heparinization and clamp placement. Exposure of the infrarenal neck of the aneurysm requires careful



**FIGURE 61-6** Technique of open operative repair of an infrarenal abdominal aortic aneurysm using a straight tube graft (**H**) or a bifurcated aortoiliac or aortofemoral (**I**) configuration. Note the attention to closure of the aneurysm sac over the completed repair, with additional closure of retroperitoneal tissues to exclude the duodenum fully (**J**). *D*, duodenum; *IMA*, inferior mesenteric artery; *IMV*, inferior mesenteric vein; *LRV*, left renal vein; *SMA*, superior mesenteric artery. (Courtesy Mayo Foundation for Medical Education and Research.)

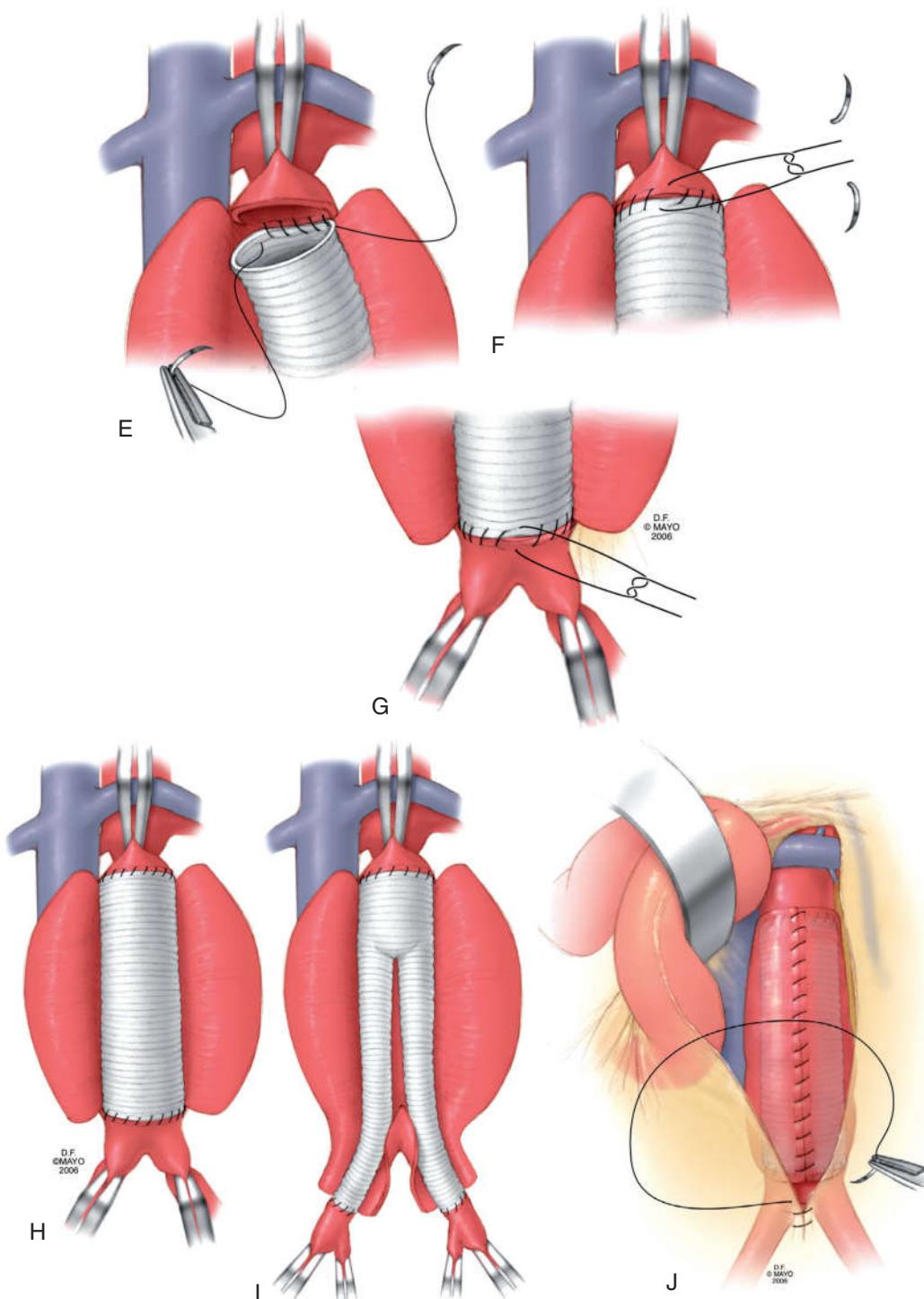


FIGURE 61-6, cont'd

mobilization of the duodenum, distal to the ligament of Treitz, to the patient's right side. The retroperitoneum may then be opened to the level of the iliac bifurcation. Mobilization of the left renal vein facilitates exposure and control of the neck of the aneurysm. At this time, a decision should be made about the necessity of division of either the left renal vein or its tributaries. The iliac arteries may be exposed by careful dissection in the avascular anterior plane, with attention to preservation of the

ureter, which will typically cross at the level of the iliac bifurcation, and the pelvic sympathetics, which cross the bifurcation and proximal left common iliac artery. Extensive dissection of the bifurcation and proximal common iliac arteries is not typically necessary as clamp placement in the mid or distal common iliacs is more typical when the aneurysm terminates at or proximal to the aortic bifurcation, permitting repair with a simple tube graft. When aneurysmal or occlusive disease of the iliacs requires

replacement of the common iliac, clamps may be placed at the proximal internal (hypogastric) and external iliac arteries. Soft iliac arteries may be controlled with vessel loops placed in a Potts fashion or using a Rumel tourniquet. However, the authors prefer to use vascular clamps to avoid circumferential dissection of the iliac arteries, where possible, and the attendant risk of venous injury, which can lead to catastrophic bleeding. Severely calcified iliac arteries may be controlled with occlusive balloons, although the proximal ends may require endarterectomy to permit either anastomosis or oversewing.

Once adequate dissection has been accomplished to permit proximal and distal control and systemic heparin administered, clamps may be placed and the aneurysm sac opened. There are a variety of opinions for the order of clamp placement. Some think that initial proximal clamp placement minimizes the risk of distal embolization; others maintain that initial distal clamp placement permits staging of the hemodynamic effect of clamp placement. The sac should be opened just below the aneurysm neck and the opening extended along the right side of the anterior surface of the aneurysm, leaving the orifice of the inferior mesenteric artery in situ. Lumbar arteries and the middle sacral artery may be ligated from within the sac to prevent backbleeding. An inferior mesenteric artery with brisk, pulsatile backbleeding or one that is chronically occluded, as often occurs in aneurysms, may be safely oversewn at its origin. Poor backbleeding suggests inadequate collateralization and is an indication for reimplantation of the inferior mesenteric artery into either the main graft or the left iliac limb.

Once backbleeding has been controlled, the proximal anastomosis may be addressed, typically in an end-to-end running fashion using nonabsorbable monofilament suture such as Prolene and an appropriately sized woven or knitted polyester graft. An aneurysm terminating at or before the aortic bifurcation may be repaired with a simple tube graft, whereas involvement of the iliac vessels may necessitate a bifurcated graft and distal anastomoses to either the iliac or femoral arteries. Once the proximal anastomosis is complete, it should be examined by placing a second clamp below the anastomosis and carefully removing the proximal clamp. Any areas of bleeding may be readily addressed with repair sutures at this time, before immobilization of the graft by the distal anastomosis. If a tube graft is sufficient, the distal anastomosis may similarly be completed in a running fashion. Iliac anastomoses may frequently be performed at the level of the iliac bifurcation, incorporating both internal and external iliac arteries as a common orifice. If femoral anastomosis is performed, a retroperitoneal tunnel should be created bluntly in the avascular, anatomic plane anterior to the native external iliac artery, passing beneath the ureter. The limb may then be passed to the groin incision using either a blunt clamp passed gently through the tunnel from groin to retroperitoneum or a sterile tape or drain passed along the same course.

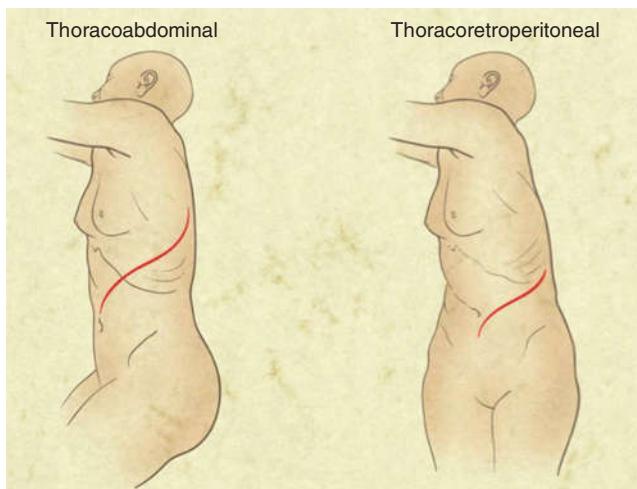
Before completion of the distal anastomosis, the distal iliac or femoral vessels should sequentially be permitted to backbleed to flush out any thrombus or atherosclerotic debris, the proximal clamp briefly removed to flush the graft, and the graft flushed with heparinized saline. The proximal and distal clamps may then be removed. It is imperative that the surgical and anesthesia teams communicate well during this process as clamping and unclamping of the aorta produce profound hemodynamic effects. The patient should be well resuscitated before unclamping as this act is frequently attended by a significant hypotension. Slightly staged release of the iliac arteries or limbs, in the case of a bifur-

cated graft, may alleviate this somewhat. Sodium bicarbonate to counteract acidosis and the use of vasopressor agents may also be required at this time. The inferior mesenteric artery may be reimplanted at this time, if necessary, most commonly as a Carrel patch. If hemostasis appears to be adequate at all anastomoses and the patient is normotensive, protamine may be administered at 0.5 to 1 mg per 100 units of heparin given.

Once aortic replacement has been accomplished, attention should be turned to graft coverage. The aneurysm sac and retroperitoneum may be approximated over the graft to effectively exclude the abdominal contents and, in particular, the third portion of the duodenum, which typically rests just anterior to the proximal, infrarenal suture line. The abdominal and, if present, groin incisions should be closed meticulously. Hernias, as previously noted, occur relatively frequently after open aneurysmorrhaphy. Wound breakdown, particularly at the groin, can be costly and difficult for the patient and significantly increases the risk of catastrophic graft infection. The authors do not routinely drain groin incisions.

The retroperitoneal approach is thought, by some, to reduce physiologic stress on the patient and to result in fewer postoperative pulmonary complications as well as a reduction of postoperative ileus.<sup>49</sup> Both approaches are associated with a significant rate of wound healing complications. Midline incisions for AAA repair are complicated by radiographically apparent abdominal wall defects in approximately 20% of cases in a recent series, although clinically significant hernias are less frequent. Persistent postoperative pain, flank wall laxity, and hernia have been described complicating retroperitoneal repair, and some investigators have reported more frequent occurrence of these complications with use of the retroperitoneal technique. With regard to operative exposure, the retroperitoneal approach does afford greater access to the visceral segment of the abdominal aorta and may be aided, where required, by thoracic extension of the incision and exposure with or without division of the diaphragm.

A retroperitoneal aortic exposure may be accomplished with the patient in a modified right lateral decubitus position with the thorax rotated but hips relatively flat to permit access to both groins (Fig. 61-7). A curvilinear incision is made from the costal



**FIGURE 61-7** Patient positioning and incision for thoracoabdominal and thoracoretroperitoneal exposures. Note the open configuration of the hips in the latter, facilitating bilateral access to the iliac and femoral arteries.

margin to below the umbilicus, depending on the extent of exposure required and the patient's habitus. The retroperitoneal plane may be entered at the lateral border of the rectus sheath. The rectus abdominis may be reflected either medially or laterally. Some surgeons prefer lateral reflection as this may result in less difficulty with postoperative body wall laxity. Care is taken to avoid entering the peritoneum. Much of the initial portion of this dissection may be carried out bluntly, with the aid of a tonsil sponge on a ring or Kelly forceps. The abdominal contents, enveloped in peritoneum, may be swept medially. The ureter will be visualized and swept medially. The left kidney may be either elevated or left in situ, although the authors generally prefer to medialize the kidney, which also serves to mobilize the left renal vein. The gonadal tributary, however, must generally be identified, ligated, and divided. Proximally, the spleen is carefully mobilized within its peritoneal covering to expose the underside of the diaphragm. The fibers of the left crus of the diaphragm, when divided, expose the supraceliac and visceral portions of the aorta. The left renal artery should be readily accessible, and the celiac and superior mesenteric arteries may be mobilized by careful dissection. The right renal artery is frequently difficult to isolate before aortotomy. Distally, the iliacs are carefully exposed in the avascular plane by gently mobilizing overlying structures, including the ureters. Again, the full exposure of the right iliac is typically more difficult by this approach, depending on the patient's habitus. The extensive exposure of the supraceliac and visceral portions of the aorta permits full access and nuanced decision making about clamp placement, which may be suprarenal, supramesenteric, or supraceliac. Visceral and renal vessels may be controlled by clamp placement, vessel loops, or, after aortotomy, use of occlusion balloons, with great care taken in handling to avoid dissection or embolization. Occlusive disease or aneurysmal involvement of renal or visceral vessels may be readily addressed by this approach. According to the patient's indications and the surgeon's preference, cardiopulmonary bypass may be used as an adjunct and provides the ability to actively perfuse the renal and visceral vessels should a complex or prolonged reconstruction be anticipated.

Once adequate exposure has been achieved, proximal and distal clamps may be placed. As in the transperitoneal approach, repair is typically accomplished by endoaneurysorrhaphy, using end-to-end proximal and distal anastomoses to replace the diseased portion of aorta as an interposition. Once again, the aneurysm thrombus is removed at the time of aortotomy, and lumbar arteries are ligated within the sac. The same principles of back-bleeding and flushing of the graft before completion of the distal anastomosis apply. This approach does also permit a variety of approaches to reconstruction of the juxtarenal, pararenal, and paravisceral aorta. Branch vessels may be incorporated together by careful beveling of the graft, reimplanted individually as Carrel patches, or reconstructed using short bypass grafts. In treating thoracoabdominal aneurysms, the incision may be extended into the chest at the appropriate rib space and the diaphragm circumferentially divided to afford enough exposure to extend the repair to virtually any level of the descending aorta. The rib may be circumferentially dissected and divided posteriorly to further improve thoracic exposure as needed. When hemostasis is achieved, the sac may again be closed over the graft, although the retroperitoneally placed graft is not as vulnerable to erosion and aortoduodenal fistula as that placed transperitoneally (Fig. 61-8).

Medial visceral rotation, introduced by Mattox for trauma and adapted to aortic reconstruction by Stoney, is a third technique

that may, through an abdominal incision, afford exposure of the entire abdominal aorta. This technique may be used for type IV or high paravisceral aneurysms and is best suited for patients who are not obese or asthenic, with narrow costal margins extending to the iliac crest.

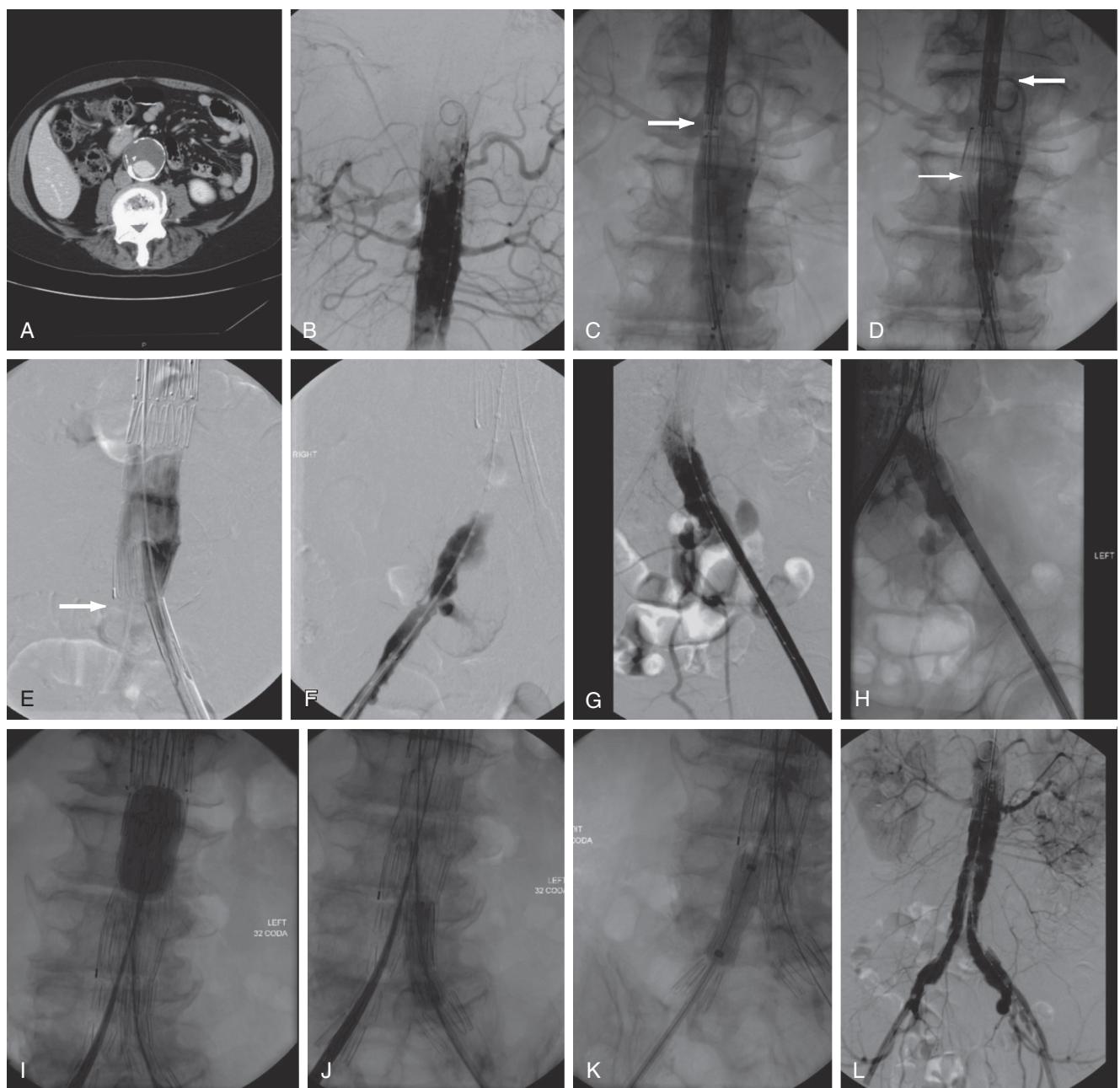
### Management of the Ruptured Aneurysm

Patients who survive to present to the hospital with a ruptured AAA may range from relatively stable to circulatory collapse. Optimal outcomes rely on the establishment of an institutional system for management of this critical aortic emergency that facilitates the early notification of the operative team, the availability of appropriate equipment (including an inventory of implants) and staff to support endovascular or open repair, and a system for rapid anatomic assessment. Several key principles of management must be considered. First, the appropriate hemodynamic parameters are those of permissive hypotension, with systolic blood pressures as low as 50 to 70 mm Hg considered adequate in a conscious patient, and avoidance of aggressive volume resuscitation. Management of a hemodynamically unstable patient or one in whom aortic control is expected to be delayed, prolonged, or complex, whether open or endovascular, may use percutaneous balloon control of the proximal aorta.<sup>50</sup> When possible, contrast-enhanced CTA should be performed preoperatively, although techniques that rely exclusively on angiography have been described. This may establish whether endovascular repair is possible or preferred, in addition to delineating anatomy and identifying associated aneurysmal or occlusive disease.

In the operating room, consideration should be given to the method and timing of anesthesia. Endovascular repair may be performed initially or in its entirety under local anesthesia. When open repair and general anesthesia are required, it is prudent to complete positioning, sterile preparation, and draping of the field before induction of anesthesia. In treating a ruptured aneurysm, a supraceliac control clamp should greatly facilitate resuscitation and provide a measure of hemodynamic stability. A formal protocol for resuscitative endovascular balloon occlusion of the aorta has been developed and can be of great utility in the unstable patient with ruptured aneurysm as well as other forms of hemorrhagic shock. This balloon may be placed percutaneously to establish proximal control in anticipation of open or endovascular repair.<sup>51</sup> Initial wire access to the suprarenal aorta should be followed by passage of a stiff guidewire and placement of a sheath of sufficient size (14 Fr) to accommodate a large, compliant occlusion balloon and length (40 cm) to support the inflated balloon in a suprarenal position against aortic pulsation.

### Postoperative Management

In the immediate postoperative period, patients are typically admitted to an intensive care unit, with continuous cardiopulmonary monitoring. Adequate pain control, appropriate resuscitation, adequate oxygenation, and heart rate control all serve to minimize the risk of postoperative myocardial infarction. Epidural anesthesia and patient-controlled analgesia are both excellent options for postoperative pain management, and epidural anesthesia may actually decrease postoperative complications.<sup>52</sup> The use of appropriate prophylaxis for deep venous thrombosis is important and is not precluded by the use of an epidural catheter. Attention to early mobilization and nutrition of the patient are also essential to recovery.



**FIGURE 61-8** Technique of EVAR. **A**, Initial aortogram profiling the renal arteries. **B**, Device has been advanced over a stiff wire to the level of the renal arteries. **C**, Note radiopaque markers indicating the beginning of fabric coverage (arrow). **D**, Device sheath withdrawn, permitting partial opening of the proximal graft (thin arrow). Note that the top cap continues to constrain the suprarenal fixation wires (thick arrow). **E**, The contralateral iliac limb gate (arrow) has been cannulated; contrast material is introduced with use of a rim catheter to confirm successful cannulation before placement of iliac extension. **F-H**, Angiography of both iliac arteries with marker catheters in place to permit deployment of iliac extensions, with preservation of both internal iliac arteries. **I-K**, Balloon molding of the proximal graft, overlap segments of the main graft and iliac limbs, and distal seal zones of the iliac limbs to facilitate proximal, distal, and intercomponent seals. **L**, Completion aortogram demonstrating successful exclusion of the aneurysm and no evidence of endoleak, which would be manifested as continued filling of the aneurysm sac by contrast material.

Although late events after open surgical repair are relatively rare, a program of surveillance is typical to detect complications such as the formation of anastomotic or para-anastomotic aneurysms, which may occur up to 20% of the time at 15 years after repair.<sup>53,54</sup> The authors typically image patients with CT initially,

then at 5-year intervals after repair. Ultrasound may also be used for surveillance but is operator dependent and lacks the sensitivity of CT for detecting anastomotic or para-anastomotic changes.

Ruptured aneurysm may pose a significant challenge in the postoperative period, whether it is approached in an open or

endovascular fashion. The incidence of serious complications, such as colon ischemia, renal failure, and spinal cord infarction, is significantly higher than after elective repair and is associated with increased mortality. Surgeons must remain vigilant as colon ischemia may be manifested subtly and must have a low threshold for sigmoidoscopy if it is suspected. Ischemia and reperfusion may also contribute to injury to the lungs, development of lower extremity compartment syndrome with rhabdomyolysis, and abdominal compartment syndrome. The abdominal compartment syndrome may necessitate decompressive laparotomy after successful endovascular repair.

### Endovascular Repair

EVAR was first reported by Parodi and colleagues in 1991<sup>55</sup> and has been widely adopted since the first Food and Drug Administration (FDA)-approved devices for EVAR, the AneuRx (Medtronic, Minneapolis, Minn) and the Ancure (Guidant Corporation, Menlo Park, Calif), became available in 1999. In 2006, the Agency for Healthcare Research and Quality published a comparison of EVAR and open surgical repair for AAA that concluded that "EVAR has shorter length of stay, lower 30-day morbidity and mortality but does not improve quality of life beyond 3 months or survival beyond 2 years."<sup>56</sup> These advantages, although limited, have been sufficient to make EVAR more frequently performed in recent years than open surgical repair for aneurysms with suitable anatomy.<sup>57</sup> The advantages of EVAR extend to treatment of ruptured aneurysms, for which multiple studies have confirmed that endovascular repair is associated with lower in-hospital morbidity and mortality.<sup>58</sup> Several anatomic considerations guide patient suitability for EVAR, including the anatomy of the aneurysm neck (size, length, shape, and angulation) and the iliac arteries (caliber, tortuosity, and aneurysmal involvement). The capabilities of currently available devices, as summarized in their approved indications for use, are listed in Table 61-3. The majority of available devices are modular bifurcated grafts consisting of an aortic main body to be used with a variable number of iliac or proximal aortic extension components. Aortouni-iliac devices are also available and may be used, generally in conjunction with femoral-femoral bypass grafting, either primarily or to salvage a failed bifurcated device. Branched devices designed to preserve the hypogastric artery in the setting of an aneurysmal common iliac artery are available in Europe and in clinical trials in the United States.\*

Whereas early morbidity and mortality (0.5% to 1.54% versus 3% to 4.8%) are lower with EVAR, there is overall a higher rate of re-intervention (albeit primarily endovascular) after endovascular than after open aneurysm repair and, after 2 to 3 years, no significant difference in the overall mortality rate.<sup>1,59</sup> This relatively new technology has borne an entirely new set of complications. Early, periprocedural complications include endoleak; access-related complications, which occur in up to 3% of cases and include hematoma, pseudoaneurysm, arterial occlusion or dissection, and iliac artery rupture or transection; peripheral embolization; renal insufficiency; local wound complications; inadvertent renal or hypogastric artery occlusion; and rare occurrences, such as colon or spine ischemia. Late complications

include rupture, which occurs rarely but at a higher rate than after open repair; graft limb occlusion; endoleak or sac enlargement; and graft infection.<sup>60</sup> Some of these, such as access site complications, have diminished in frequency over time as operator experience has improved and newer generations of devices have incorporated smaller diameters and hydrophilic coatings.

Endoleak is the most common indication for re-intervention after EVAR. Type I endoleak is defined as failure to achieve a satisfactory seal at either the proximal (type Ia) or distal (type Ib) seal zone, representing a failure to exclude the aneurysm sac. In general, a type I endoleak should be addressed at the time of detection. More aggressive balloon inflation within the seal zone, placement of additional graft components to extend the seal zone, and placement of balloon-expandable stents within the seal zone to improve wall apposition through increased radial force are among the most common endovascular therapies for type I endoleak. One FDA-approved device uses helical EndoAnchors (Aptus Endosystems, Sunnyvale, Calif) delivered with a deflectable sheath to address both type I endoleak and migration.<sup>61</sup> Embolization using endovascular coils or liquid embolic agents such as Onyx has also been described.

Type II endoleaks are the most common form and represent continued filling of the aneurysm sac by lumbar branches or the inferior mesenteric artery. Further treatment is indicated if a persistent type II endoleak is accompanied by an increase in sac size. Treatment may include embolization of feeding branches by selective catheterization (transarterial technique) or direct sac puncture (translumbar technique) or laparoscopic or open surgical ligation of these vessels. Efforts have recently focused on identifying pre-operative imaging characteristics predictive of persistent type II endoleaks or those that will result in sac enlargement. Aneurysm sac diameter at the level of the inferior mesenteric artery and the number of patent lumbar arteries have been associated with persistent type II endoleak and delayed or recurrent presentation, the presence of inferior mesenteric artery-lumbar artery type of endoleak, and the diameter of the largest feeding or draining artery with sac enlargement.<sup>62-64</sup> Whereas embolization of patent aortic branches at the time of the index procedure seems to decrease the rate of type II endoleak, rupture due to type II endoleak remains rare and difficult to predict; therefore, the role of preemptive branch vessel embolization is controversial.<sup>65,66</sup> Additional techniques, such as perigraft sac embolization, are being investigated.<sup>67</sup>

Type III endoleaks represent failure of an individual component or of the seal between components of a modular graft system. As with type I leaks, all type III endoleaks should be treated, typically by relining the offending area with new graft components. Type IV endoleaks represent seepage through porous graft material and are typically self-limited, resolving when procedural anti-coagulation is reversed. Finally, an entity known as endotension is sometimes considered a fifth type of endoleak. This represents persistent growth of the aneurysm sac in the absence of a detectable leak. It is proposed that this phenomenon is due to either the passage of serous ultrafiltrate across an excessively porous fabric or, as some believe, the existence of an undetected endoleak of one of the prior types.

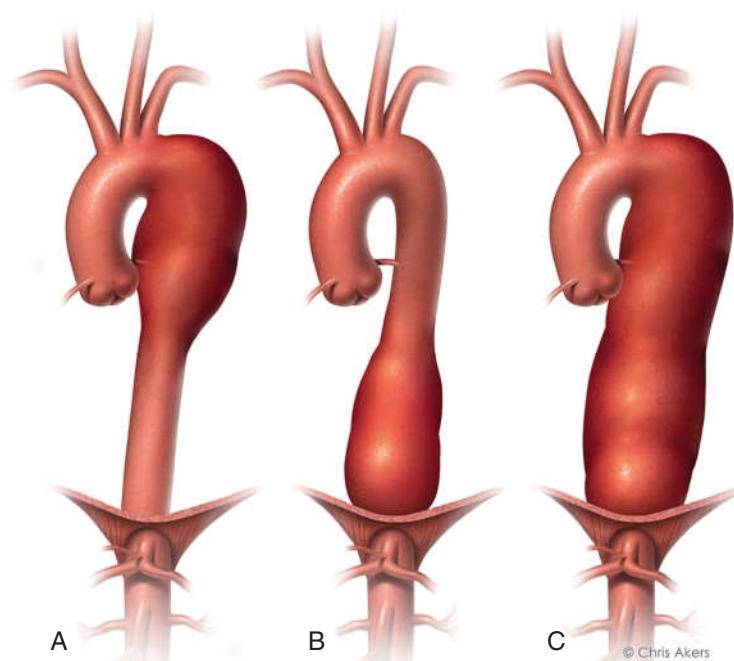
It has also been noted that as operators have gained experience with EVAR, a growing proportion of procedures are performed outside the approved instructions for use. There has, during the same period, been a trend toward treating a greater proportion of patients 80 years of age or older. When demographic and anatomic factors were reviewed, it was noted that only 42% of

\*Gore Excluder Iliac Branch Endoprosthesis, Cook Zenith Branch Iliac Endovascular Graft. <http://www.goremedical.com/eu/excluder/>, <http://zenithglobal.cookmedical.com/zenith-abdominal.html>. Accessed September 29, 2015.

TABLE 61-3 Endovascular Repair Devices: Indications for Use

DEVICE (MANUFACTURER)	STENT OR GRAFT MATERIAL	SHEATH OR DEVICE DIAMETER (MAIN BODY) (mm)	AORTIC DIAMETER* (mm)	ILIAC DIAMETER (mm)	MAXIMUM ANGULATION	MINIMUM NECK LENGTH	OTHER
AneuRx (Medtronic)	Nitinol, polyester	21 Fr	20-28 (graft)	12-24 (graft)	45-degree neck	15 mm	Initial FDA approval 1999 No suprarenal fixation or barbs Sheath not required Suprarenal fixation stent Tapered and flared limbs available Uni-iliac Talent Converter device available
Talent (Medtronic)	Nitinol, polyester	22 Fr	18-32 (aorta) 22-36 (graft)	8-22 (iliac) 8-24 (graft)	60-degree neck	10 mm	Barbed suprarenal fixation stent Like Talent, approved for 10-mm neck Thin fabric, low delivery profile
Endurant (Medtronic)	Nitinol, polyester	18 Fr, 20 Fr	19-32 (aorta) 23-36 (graft)	8-25 (iliac) 10-28 (graft)	60-degree neck	10 mm	Barbed suprarenal fixation stent Tapered limb configurations available
Zenith (Cook Medical)	Stainless steel, polyester	18 Fr, 20 Fr, 22 Fr	18-32 (aorta) 22-36 (graft)	7.5-20 (iliac) 9-24 (graft)	60-degree neck	15 mm	Zenith Reu aortouni-iliac graft available Graft sizing based on outer diameter Available ipsilateral limb sizes vary with size of primary graft Contralateral limb components may be used as iliac extenders
Excluder (W.L. Gore)	Nitinol, ePTFE	18 Fr, 20 Fr	19-29 (aorta) 23-31 (graft)	8-13.5 (ipsilateral graft) 12-14.5 (ipsilateral graft) 8-18.5 (contralateral graft) 12-20 (contralateral graft)	60-degree neck	15 mm	Proximal nitinol anchors Anatomic fixation on aortic bifurcation Seal achieved with suprarenal or infrarenal proximal aortic cuff IntuiTrak system avoids need to cannulate contralateral gate Similar to Powerlink in design Multiple limb configurations Low-profile delivery system for bifurcated graft and proximal cuff
Powerlink (Endologix)	Cobalt-chromium, ePTFE	19 Fr, 21 Fr	18-32 (aorta) 22, 25, 28 (graft) 25, 28, 34 (aortic cuff)	10-23 (iliac) 13-16 (graft) 16-25 (extensions)	60-degree neck 90-degree iliac	15 mm	Uses polymer-filled sealing ring technology for proximal seal Offers integrated crossover lumen as alternative to retrograde cannulation of contralateral gate Combination circular and helical stents to accommodate extreme angulation Proximal fishmouth configuration
AFX (Endologix)	Cobalt-chromium, ePTFE	17 Fr	18-32 (aorta) 22, 25, 28 (graft) 25, 28, 34 (aortic cuff)	10-23 (iliac) 13-16 (graft) 16-25 (extensions)	60-degree neck 90-degree iliac	15 mm	
OvationPrime (Trivascular/Endologix)	Cobalt- Chromium/ ePTFE	14 Fr	16-30 (aorta)	8-25 (iliac)	60-degree neck (45-degree if neck <10 mm)	<10 mm†	
Aorfix (Lombard)	Nitinol/woven polyester	22 Fr	19-29 (aorta) 24-31 (graft)	9-19 (iliac) 10-20 (graft)	90-degree neck	20 mm	

\*Recommended methods of vessel sizing and guidelines for graft oversizing vary by device.  
†Proximal seal based on diameter at 13 mm from lowest renal, as sealing O-ring centered 13 mm from leading edge.



**FIGURE 61-9** Classification, descending thoracic aortic aneurysm. **A**, Type A, distal to the left subclavian artery to the sixth intercostal space. **B**, Type B, sixth intercostal space to above the diaphragm (twelfth intercostal space). **C**, Type C, entire descending thoracic aorta, distal to the left subclavian artery to above the diaphragm (twelfth intercostal space). (Courtesy Chris Akers, 2006.)

patients met the conservative definition of instructions for use, whereas 69% met the most liberal definition. Independent predictors of post-EVAR sac enlargement at 5 years were the presence of endoleak, age of 80 years or older, aortic neck diameter of 28 mm or more, aortic neck angulation of 60 degrees or more, and common iliac artery diameter of more than 20 mm, suggesting that factors associated with the gradual liberalization of anatomic criteria over time are associated with sac enlargement and, by implication, worse outcomes.<sup>68</sup> As techniques and devices extend the proximal seal zone into the visceral segment and beyond, one may expect that the trend toward treatment outside instructions for use with infrarenal devices may be reversed.

Device migration, either intraprocedurally or over time, may occur. In the EVAR setting, migration may be facilitated by unfavorable aneurysm neck anatomy. Manufacturers have attempted to address this issue by mechanisms including increased radial force, use of barbs or suprarenal fixation, or use of “anatomic fixation” at the aortic bifurcation. Device failure resulting from fracture of metallic components or fabric failure may also occur. The iliac limbs of these devices are also subject to thrombosis and occlusion, possibly at a higher rate than bifurcated grafts placed during open surgical repair.<sup>69</sup>

It is recommended that contrast CT surveillance be conducted at 1 month, 6 months, and 12 months after graft implantation and annually thereafter. Concern about accumulated lifetime radiation exposure and the use of nephrotoxic contrast agents has driven the expansion of the role of color Doppler and contrast-enhanced duplex ultrasonography in graft surveillance.<sup>70,71</sup> Implantable sensor technology has been approved by the FDA to monitor pressure within the aneurysm sac and may evolve to augment or even to supplant current imaging techniques for postoperative aneurysm surveillance.

### Thoracic Aortic Aneurysm

Aneurysms of the descending thoracic aorta may be classified as type A, B, or C, depending on whether the aneurysm involves the proximal, mid, or distal third of the descending aorta, respectively (Fig. 61-9). Thoracoabdominal aneurysms are typically distinguished according to the Crawford classification system (Fig. 61-10). As with aneurysms of the abdominal aorta, rupture risk is closely associated with aneurysm size and, to a lesser extent, female gender. Current guidelines recommend repair of the descending thoracic aorta at 5.5 cm.

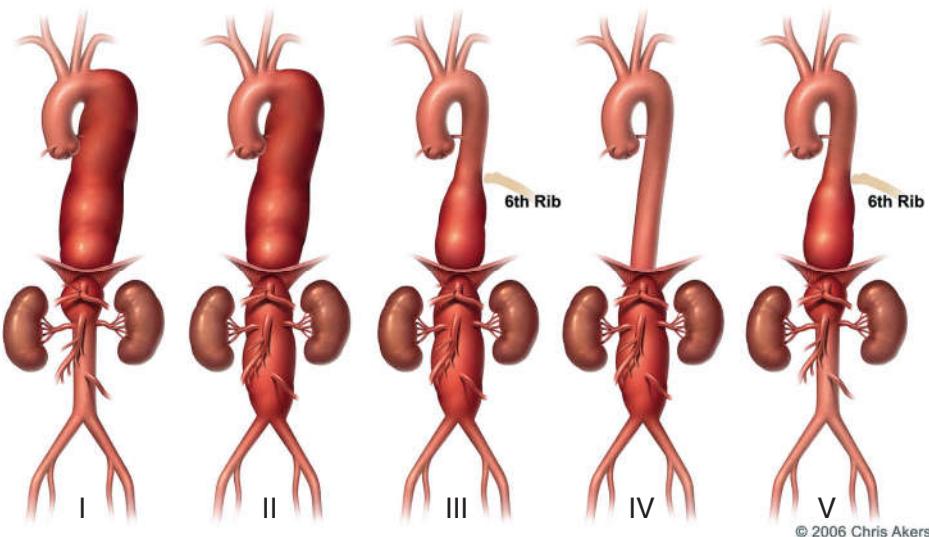
### Open Repair of Thoracic and Thoracoabdominal Aneurysms

The level of entry into the thoracic cavity for repair of thoracic or thoracoabdominal aneurysm is guided by the proximal extent of the aneurysm. Incision at the fifth or sixth interspace provides excellent exposure of the proximal descending aorta; at the eighth or ninth interspace, the mid-descending aorta; and at the tenth or eleventh interspace, the infradiaphragmatic portion of the aorta. Cardiopulmonary bypass combined with the selective use of distal aortic and visceral perfusion and hypothermic circulatory arrest have yielded exemplary results in experienced hands.<sup>72</sup>

### Endovascular Management of Thoracic Aneurysms

In 2005, the FDA approved the GORE TAG Thoracic Endoprosthesis (W.L. Gore & Associates, Flagstaff, Ariz) for treatment of the descending thoracic aorta. Since that time, open surgical therapy for aneurysms of the descending thoracic aorta has largely been supplanted by TEVAR for anatomically suitable lesions.<sup>73</sup>

As with abdominal aortic endografts, initial and subsequent studies have demonstrated a significantly lower rate of short-term morbidity and mortality than with open repair. Unlike EVAR, TEVAR appears to offer a significant, long-term aneurysm-related



**FIGURE 61-10** Normal thoracoabdominal aorta aneurysm classification: extent I, distal to the left subclavian artery to above the renal arteries; extent II, distal to the left subclavian artery to below the renal arteries; extent III, from the sixth intercostal space to below the renal arteries; extent IV, from the twelfth intercostal space to the iliac bifurcation (total abdominal aortic aneurysm); extent V, below the sixth intercostal space to just above the renal arteries (modified Crawford classification). (Courtesy Chris Akers, 2006.)

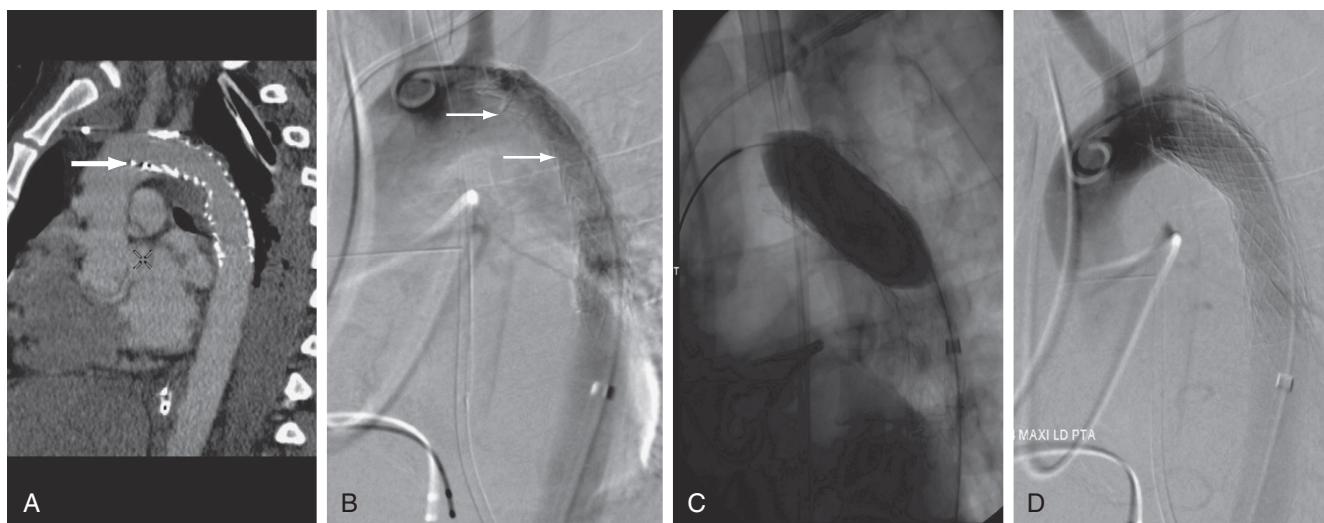
mortality advantage. The most frequent complications of TEVAR are related to injuries to femoral or iliac access vessels. As the diameters of delivery devices for thoracic endografts may be considerably larger than those required for EVAR, many surgeons have developed a distinctly conservative approach to device access, performing open femoral artery exposure or addressing diminutive iliac vessels through the use of iliac or aortic conduits, open aortic or iliac exposure and direct puncture, or the use of endovascular iliac conduits to prevent injury. Recently, several investigators have pioneered the technique of caval-aortic access for delivery of larger diameter devices to the thoracic aorta or aortic valve.<sup>74</sup> The combination of operator experience and the development of lower profile and hydrophilic delivery systems has, over time, reduced the rate of access-related complications of TEVAR.<sup>75</sup>

Because of the passage of endovascular wires, catheters, and other devices through the aortic arch, TEVAR carries the additional risk of embolic stroke.<sup>76</sup> Nevertheless, TEVAR has consistently yielded lower rates of early mortality and common postoperative complications than in open repair, with Bavaria and colleagues reporting perioperative mortality rates of 2.1% versus 11.7%, spinal cord ischemia rates of 3% versus 14%, respiratory failure rates of 4% versus 20%, and renal insufficiency rates of 1% versus 13% in low-risk patients after endovascular and open repair, respectively.<sup>77</sup>

Several anatomic considerations guide the selection of patients for TEVAR. As with EVAR, the size and configuration of the proximal aneurysm neck must suit the configuration and capabilities of available grafts. Commercially available thoracic endograft diameters currently range from 21 to 46 mm, creating limitations in patients with large proximal necks or small-caliber aortas. Tapered configurations are also available. The radius of the aortic arch and proximal descending aorta can also challenge device conformability and may result in a “bird’s beak” deformity on deployment and, potentially, device collapse with consequent compromise of the aortic lumen (Fig. 61-11). In addition, coverage of one or more supra-aortic vessels may be required to achieve

an adequate proximal landing and seal zone for the graft, necessitating decisions about extra-anatomic reconstruction. Most commonly, the left subclavian artery origin is covered. Justifications for surgical reconstruction of the subclavian artery, generally by carotid–subclavian artery bypass or subclavian artery transposition, include prevention of arm claudication, preservation of flow to a dominant left vertebral artery, and perhaps most important, maximization of collateral spinal cord perfusion. More recently, endovascular fenestration of the left subclavian artery by a laser technique has been reported. Ultimately, branched devices such as the GORE TAG Thoracic Branch Endoprosthesis are being trialed to extend the proximal seal zone into the aortic arch. Endoleaks, primarily type I, complicate a significant proportion of TEVAR procedures. Some close spontaneously, perhaps because of reversal of intraprocedural heparin anticoagulation. Others require repair by the same techniques used for endoleaks encountered after EVAR.<sup>78</sup>

As with open repair of thoracic and thoracoabdominal aneurysms, prevention of spinal cord ischemia, which may be manifested as temporary or permanent, unilateral or bilateral paraparesis or paraplegia, is of great importance. Delayed rather than immediate postoperative spinal cord ischemia seems to be more common after TEVAR than after open surgical repair, and these patients may be more likely to experience functional improvement after injury.<sup>79</sup> Risk factors for spinal cord ischemia include the length of aortic coverage, coverage of the distal thoracic aorta, compromise of multiple collateral territories, preoperative renal insufficiency, and hypotension.<sup>80</sup> Whereas institutional protocols vary, particularly with regard to selection of patients for cerebrospinal fluid drain placement, the mainstays of prevention of spinal cord ischemia and the treatment of delayed-onset symptoms remain avoidance of hypotension, blood pressure augmentation (target mean arterial pressure > 90 mm Hg), and cerebrospinal fluid drainage to minimize ischemia related to cord compression in the setting of elevated cerebrospinal fluid pressure after ischemia-reperfusion.



**FIGURE 61-11** **A**, CT scan demonstrating thoracic endograft with bird's beak deployment along the lesser curve of the aorta, leaving the leading edge of the endograft projecting into the lumen (arrow). **B**, Thoracic aortogram demonstrating subsequent collapse of the endograft caused by pressure on the protruding proximal portion of the endograft, resulting in distal hypoperfusion (arrows). **C** and **D**, Deployment of a balloon-expandable Palmaz stent to reopen the proximal graft.

### Endovascular Repair of Aneurysms Involving the Visceral Segment: Snorkels, Fenestrations, and Branched Grafts

Endovascular therapy for juxtarenal, suprarenal, and thoracoabdominal aneurysms was, until recently, limited to a few centers with access to investigational fenestrated devices or with substantial experience in either creating customized fenestrated devices (Fig. 61-12) or using debranching (antegrade grafts from the thoracic aorta or retrograde iliac grafts to the renovisceral vessels, permitting stent graft coverage of the perivisceral segment) or “snorkel” or “chimney” techniques (use of covered stents extending from the branch vessels beyond the proximal or distal extent of an aortic stent graft) to maintain perfusion to branch vessels.<sup>81,82</sup>

At present, a broader range of centers have gained critical experience in the use of surgeon-modified grafts or snorkel techniques for the treatment of aneurysms involving the visceral segment of the aorta.<sup>83</sup> In 2012, the FDA issued the first U.S. approval for a fenestrated device for the treatment of juxtarenal and pararenal aneurysms, the custom-made Zenith Fenestrated AAA Endovascular Graft (Cook Medical, Bloomington, Ind.). This approval mandated a rigorous physician training program including an intensive 2-day training session for the initial group of implanting physicians, selected for their extensive EVAR experience, followed by physician proctoring. Additional branched or fenestrated devices have gained European CE Mark approval or are in investigational stages either in the United States or abroad. There is particular interest in the development of “off-the-shelf” devices that would permit accommodation of the majority of anatomic configurations without the 3- to 4-week manufacturing period required to obtain the custom-manufactured grafts. Devices currently in U.S. clinical trials include the Cook Zenith p-Branch and Endologix (Irvine, Calif) Ventana and the Cook Zenith t-Branch Thoracoabdominal Endovascular Graft, which incorporates four caudally oriented branches into an off-the-shelf design for the treatment of thoracoabdominal aneurysms. The



**FIGURE 61-12** CT reconstruction demonstrating successful placement of an endovascular graft with fenestrations at the level of the renal arteries, permitting renal stent placement and use of a proximal seal zone above the renal arteries, with preservation of renal perfusion. (Courtesy Dr. Gilbert R. Upchurch, Jr.)

Gore Excluder Thoracoabdominal Branch Endoprostheses, which began enrolling in clinical trials outside the United States in November 2014 and anticipates initiating enrollment at six U.S. sites in September 2015, includes four visceral branches that are precannulated to facilitate branch vessel selection. The features of the next generation of thoracoabdominal grafts address many of the drawbacks of current custom-made fenestrated grafts and, certainly, of the surgeon-modified branched or fenestrated and various snorkel configurations.

### **Blunt Thoracic Aortic Injury**

In the modern era, the majority of blunt aortic injury is related to motor vehicles and involves the thoracic aorta. True transections are rapidly fatal, generally before arrival at the hospital; some estimates of prehospital mortality are as high as 80% to 90%. Patients may, however, arrive relatively stable with injuries ranging from contained pseudoaneurysms to intramural hematoma to relatively subtle intimal disruptions. The current “gold standard” for imaging of aortic trauma is contrast-enhanced CTA. The most frequent site of injury is the aortic isthmus, and it is typically a transverse tear that can encompass partial or full aortic circumference and may be either partial or transmural. It is thought that injury at this location is a result of rapid deceleration, which creates severe torsion and shearing forces where relatively mobile segments of the aorta become fixed, such as the ligamentum arteriosum, aortic root, and diaphragm.

Particularly in the setting of other distracting or disabling injuries, the history and physical examination may be unrevealing. Chest radiography may demonstrate mediastinal widening, apical pleural cap, loss of the aortopulmonary window, rightward deviation of the mediastinal structures or instrumentation, or depression of the left mainstem bronchus. CT is sensitive and may distinguish between mediastinal hematoma with a preserved peri-aortic plane and true periaortic hematoma. One must also distinguish between ductus remnants or diverticula and acute injury.

Since the first report of endovascular repair of a traumatic injury to the thoracic aorta in 1997, data have rapidly accumulated to support preferential use of endovascular stent graft over traditional open repair because of consistently reduced rates of mortality (8% to 9% versus 19%), paraplegia (0.5% to 3% versus 3% to 9%), and end-stage renal disease (5% versus 8%) as well as a comparable stroke rate (2.5% versus 1%).<sup>84,85</sup> In practice, endovascular repair was adopted rapidly despite initial concerns about its durability, accounting for 65% of blunt thoracic aortic injury repairs within a decade of the initial publication. During this time, despite technical challenges related to the smaller aortic sizes, endovascular repair offers the additional benefits of reduced hemodynamic impact, lower complication rate, and less overall morbidity. The lower risk nature of the procedure itself may permit earlier aortic repair in patients with significant concurrent injuries.

### **AORTOILIAC OCCLUSIVE DISEASE**

In 1950, the first aortic reconstruction for AIOD (Leriche syndrome) was performed by Jacques Oudot in France. This was performed through a retroperitoneal approach using homograft. Following the investigational and clinical work of Arthur Voorhees at Columbia University, prosthetic grafts of Vinyon-B and nylon were used for reconstruction of aortic occlusive as well as aneurysmal disease. Both of these materials had significant prob-

lems associated with them. Wylie introduced aortoiliac endarterectomy to the United States in 1952, and that technique was the most commonly used during the 1950s. In 1958, DeBakey introduced Dacron grafts, and aortofemoral grafting with Dacron became the most widely used technique for open reconstruction, although aortoiliac endarterectomy was still performed at certain centers, notably San Francisco, Boston, and Portland, Oregon. It is still useful for patients with aortoiliac disease confined to the aorta and common iliac arteries, especially those patients with small aortas and iliac arteries, for whom endovascular repair may not be optimal. Axillofemoral artery grafting and femoral-femoral artery grafting were introduced to provide inflow procedures for poor-risk patients and patients with unilateral iliac disease, respectively.

Endovascular repair for occlusive disease of the aorta and iliac arteries was introduced in the 1990s. The use of kissing stents and the size of the common iliac arteries especially have allowed this modality to work extremely well for the majority of patients with AIOD. The TASC document on management of peripheral arterial disease (TASC I) was published in January 2000.<sup>86,87</sup> These guidelines were developed to help in the rational choice of an open or endovascular approach to aortoiliac disease in particular patients. At present, endovascular treatment is the treatment of choice for type A lesions. It is also the most commonly used modality for type B. For type C lesions with more extensive disease of the external iliacs or bilateral occlusions of the common iliacs, surgical treatment has been more often recommended. For type D lesions, that is, extensive disease of the common and external iliac arteries, surgery has been the treatment of choice. Nonetheless, multiple authors have documented good success with endovascular treatment even for TASC C and D lesions.<sup>88-90</sup>

The next iteration of the TASC recommendation is shortly expected, and it is likely that endovascular treatment will be the recommended first-line treatment for all patients. It is difficult, however, to imagine endovascular therapy to be as good or as long lasting for AIOD for these extensive lesions, especially in young patients. It is worth noting the proportion of aortofemoral bypass patients whose concomitant femoral disease requires endarterectomy at the time of the index operation.<sup>91</sup> These patients as well as patients with a significant aortic component of their AIOD may not be well served by endovascular therapy alone. It is also difficult to imagine its utility for juxtarenal aortic occlusion, with its “dunce’s cap” of chronic thrombus extending upward between the renal arteries, without a disproportionate increase in renal failure. Hybrid techniques, such as the combination of femoral endarterectomy with the placement of iliac stents, may address some of the limitations of the percutaneous-only approach.

Concomitant with the change in TASC definitions has been a marked increase in the use of endovascular techniques in comparison to open techniques. This increase corresponds to improvements in the delivery systems and stents that are employed as well as to improved skill sets of vascular radiologists, cardiologists, and vascular surgeons. Upchurch and colleagues documented an increase of 850% in endovascular use by 2000 with a concomitant 16% decrease in open cases. There was a 34% increase in treated disease without an increase in the prevalence of the disease.<sup>92</sup> These trends have continued and amplified; endovascular repair for AIOD is performed much more commonly than open repair.

The classic indications for AIOD were claudication, rest pain, and threatened limb viability, manifested by tissue loss, nonhealing ulcers, or frank gangrene. Rest pain and threatened limb

viability implied extensive disease of either the deep femoral artery or the femoropopliteal segments in addition to the aortoiliac disease. The advent of endovascular techniques has, as stated before, broadened the indications, with mild claudication being treated much more frequently than in the past. There is certainly more justification for stenting of short-segment iliac stenoses than there is for performing aortofemoral bypass grafting for them.

Nonetheless, open surgery remains the gold standard for long-term patency. Chiu and associates in Birmingham, England, performed a meta-analysis of aortofemoral bypass grafting, iliofemoral bypass grafting, and aortoiliac endarterectomy. Their analysis yielded 29 studies including 5738 patients for aortofemoral bypass grafting, 11 studies incorporating 778 patients for iliofemoral bypass grafting, and 11 studies including 1490 patients for endarterectomy. Operative mortality was 4.1% for aortofemoral bypass grafting, 2.7% for iliofemoral bypass grafting, and 2.7% for aortoiliac endarterectomy. Morbidity was 16%, 18.9%, and 12.5%, respectively. Five-year primary patency rates were 86.3%, 85.3%, and 88.3%, respectively.<sup>93</sup> A meta-analysis published in 2009 suggests that formal aortic reconstruction is still the procedure of choice in terms of long-term patency. That durability may be more appealing to younger, healthier patients than return trips for further endovascular intervention. Similarly, an earlier meta-analysis covering the years 1970 to 1996 showed constant patency rates for aortic bifurcation grafts and declining mortality and morbidity over time. Mortality after 1975 was 3.3%.<sup>94</sup>

Axillofemoral artery bypass grafting is reserved for extremely poor risk patients with rest pain or tissue loss. Its suspect patency makes it a poor choice for claudicants. It is of course used as one of the mainstays of reconstruction for infected aortic grafts or aorta-enteric fistulas. This modality is offered to a different, higher risk group than is aortofemoral bypass grafting. Hertzler reported a 12% mortality compared with 5.6% for femoral-femoral grafting and 2.3% for aortic reconstruction. Within that group, mortality was only 1.2% for aortofemoral bypass grafting but 5.6% for aortoiliac endarterectomy or aortoiliac grafting.<sup>95</sup>

Ilio-femoral grafting yields patency rates superior to femoral-femoral bypass in the treatment of unilateral external iliac occlusive disease not amenable to endovascular therapy or when endovascular therapy has failed. Ricco and Probst compared the two operative methods in 143 patients. Primary 5-year patency for iliofemoral grafting was 92.7% versus 73.2% for femoral-femoral grafting.<sup>96</sup>

As in all vascular beds, endovascular repair of the aortoiliac segments requires much more re-intervention than its open counterpart, but the mortality is lower. At some "aortic centers," there is no difference in mortality, but on average, open mortality is about 4%. Hertzler reported mortality of 2.3% for direct aortic reconstructions in an elegant paper.<sup>95</sup> Both Hertzler and Reed and colleagues found increased limb occlusion in patients with small arteries, especially women.<sup>97</sup> Primary assisted and secondary patency rates for endovascular repair nearly equal the primary patency rates of open reconstruction, and this has indeed been the rationale for the widespread application of endovascular techniques for most patients with AIOD, accepting repeated intervention as a necessary component of treatment.

Multiple authors have written about the endovascular treatment of TASC C and D lesions as well as aortic occlusion. Klonaris and colleagues recommended primary stenting for all aortic occlusive disease, including occlusion. Their study, however, did not include juxtarenal aortic occlusions as opposed to more distal aortic obstruction.<sup>98</sup>

Jongkind and colleagues reviewed all published articles of patients undergoing endovascular treatment of TASC C and D lesions from 2000 to 2009. There were 1711 patients identified. Technical success was achieved in 86% to 100% of those studies. Clinical symptoms were improved in 83% to 100%. Mortality ranged from 1.2% to 6.7% and complication rates varied, being reported between 3% and 45%. Primary patency ranged from 60% to 86% and secondary patency from 80% to 98%.<sup>88</sup>

Ichihashi and colleagues reported technical success in 99% of their 125 patients with TASC C and D lesions. Complications were significantly higher than for their TASC A and B patients (9% versus 3%). Their 5-year patency was 83%, among the very highest reported.<sup>89</sup>

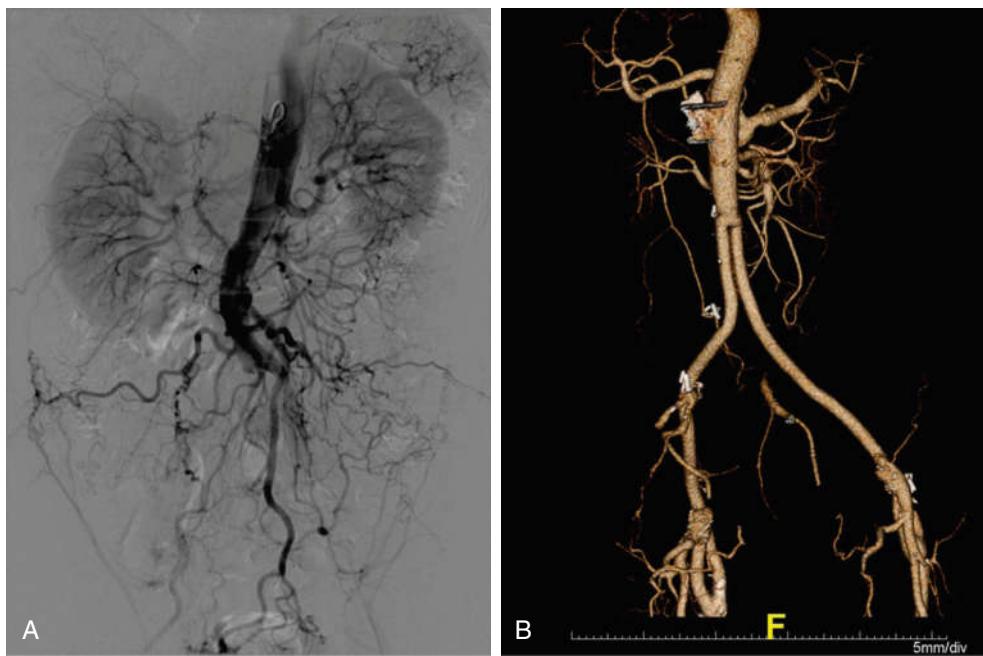
Ye and associates performed a meta-analysis of TASC C and D patients undergoing endovascular reconstruction. TASC C patients had a 93.7% technical success rate and a 1-year primary patency of 89.6%. TASC D patients had 90.1% technical success and 87.3% 1-year patency.<sup>90</sup> Indes and colleagues, reviewing the Nationwide Inpatient Sample for 4119 patients, found that endovascular procedures were associated with lower cost, lower complication rates, and shorter length of stay. Mortality was not different statistically, being 1.8% for endovascular procedures and 2.5% for open procedures.<sup>99</sup> There is a third option for aortic reconstruction, and that is laparoscopic aortic surgery. Its greatest proponents have been in Europe, particularly France, and Québec. This has not gained widespread popularity in the United States.<sup>100</sup>

## Presentation and Evaluation

Patients with AIOD may present with claudication. It is a much more likely presentation for AIOD than is rest pain or tissue loss. Rest pain or tissue loss, as stated previously, indicates disease of the deep femoral artery or the femoral popliteal segments in addition to the aorta and iliac segments. Physical examination historically has been accurate in these patients. A decreased femoral pulse is indicative of at least common femoral disease or more proximal aortoiliac disease. With the advent of the obesity epidemic in this country, physical examination of femoral pulses is not as accurate as it once was. Consequently, vascular laboratory examination is of even more importance than in the past. Wave patterns as well as ankle-brachial indices are necessary to localize the disease to aortoiliac segments, femoral-popliteal segments, or both. It is also vital in identifying the contribution of AIOD to patients with multiple diseases contributing to their lower extremity problems, including neurogenic claudication, spinal stenosis, and hip arthritis, either alone or in combination with arterial disease.

With a tentative diagnosis of AIOD, the most commonly used modality to visualize the arteries is CTA. There are some patients in whom the calcium load is so great that either MRA or conventional arteriography is necessary to determine whether areas of calcific involvement are highly stenotic. Those surgeons for whom endovascular treatment is uniformly their first choice may proceed directly to conventional arteriography with planned endovascular intervention at the same time.

As with all vascular patients, cardiac risk is the greatest one at operation. Consequently, most authors recommend that all of these patients undergo cardiac function evaluation by stress testing. Those patients with myocardium at risk are usually treated by cardiologists or cardiac surgeons before embarking on an aortic reconstruction in an elective situation. Hertzler showed a marked decrease in cardiac mortality when patients underwent preoperative cardiac evaluation and treatment.<sup>95</sup>



**FIGURE 61-13** **A**, Preoperative aortogram demonstrating occlusion of the distal aorta and iliacs with extensive collateralization. **B**, Postoperative three-dimensional CT reconstruction demonstrating revascularization with an aortofemoral bypass graft.

As endovascular techniques gain wider acceptance, the most common indication for formal aortic reconstruction may be claudication or severe ischemia in the setting of failed multiple attempts at endovascular repair. These occluded stents make the operation more complex, with the need for suprarenal aortic clamping and more extended profundaplasties in many cases.

### Technique of Open Reconstruction

#### Aortofemoral Bypass Grafting

For aortofemoral bypass grafting (Fig. 61-13), the patient is prepared from the nipples to the knees. If concomitant distal bypass grafting will be necessary, the patient is prepared to the toes. This would be done for tissue loss with multilevel disease only. Epidural catheters may be used to alleviate postoperative pain. Bilateral groin incisions are made. These are usually done in a vertical or slight curvilinear fashion. The common, superficial, and deep femoral arteries are dissected free. These need to be dissected distally to where they are soft and suitable for anastomosis. Most surgeons use a midline incision for the aortic exposure, although a transverse incision or a retroperitoneal approach can be used. The authors favor standard midline with infracolic exposure. The abdominal contents are mobilized so that the retroperitoneum can be entered. If the mobilized viscera can be kept inside the abdominal cavity rather than being placed on the abdominal wall, the patient's gastrointestinal functional recovery is usually a bit quicker. The retroperitoneum is entered. Care is taken to stay to the patient's right of the inferior mesenteric vein to avoid violating the left mesocolon. The aorta is dissected free below the renal arteries. The surgeon needs to remember that this disease extends from the renal arteries and not from the lower-lying renal vein level. The vein may be mobilized by division and ligation of its tributaries. In general, the exposure required for occlusive disease

is less than is necessary in aneurysmal disease. The aorta is exposed down to the level of the inferior mesenteric artery. Retroperitoneal tunnels connecting this wound with the groin wounds are made. On the left side, a counterincision in the gutter lateral to the white line of Toldt may be necessary. The tunnels should be made posterior to the ureters. It is our habit to create the left tunnel posterior to the inferior mesenteric artery as well to allow the left limb of the graft to be isolated from the gastrointestinal tract by the left mesocolon after completion of reconstruction. The patient is heparinized.

Control is obtained of the aorta below the renal arteries and of the distal aorta. The aorta is divided. Distally, a portion of it is resected on an angular bias and the distal aorta oversewn. That area of excision should be done proximal to the inferior mesenteric artery. This allows comfortable placement of an end-to-end graft. An appropriately chosen graft is fashioned to fit and sewn to the end of the proximal aorta using running 3-0 or 4-0 permanent suture. If one extremity has been less symptomatic than the other, that side is reconstructed first as it is less used to ischemia. The graft is brought through the tunnel into the groin, and control is obtained of the femoral arteries. An arteriotomy is made running from the common femoral artery down to the appropriate level. In many cases, this would be down to a point on the deep femoral artery. If endarterectomy of the common and deep femoral arteries is necessary, it is undertaken at this point. The graft is fashioned to fit and sewn end to side using either running 4-0 or 5-0 permanent suture. Appropriate backbleeding and forward bleeding is allowed before that. The opposite is done in a similar manner. When flow is restored into the limbs, it is restored first to the common, then to the deep, and last to the superficial femoral artery.

In general, end-to-end anastomoses are performed. These may lessen the chance of aortoenteric fistula. They have better flow

characteristics than end-to-side proximal anastomoses. If the patient has bilateral external iliac artery occlusions, either a proximal end-to-side aortic anastomosis or a formal reconstruction of one of the internal iliac arteries is necessary to ensure continued blood flow into the pelvis.

The retroperitoneum should be carefully closed in layers to exclude the graft from the gastrointestinal tract. If there is insufficient tissue, an omental flap should be created and placed over the graft to isolate it from the duodenum.

#### Axillofemoral Bypass Grafting

Axillofemoral bypass grafting (Fig. 61-14) was introduced in the 1960s to provide inflow to patients who were poor physiologic candidates for aortic reconstruction. Its use has been extended to those patients having aortic graft infections or otherwise hostile abdomens for whom an in-line aortic reconstruction is considered too hazardous.

The patient is prepared from the shoulders down to the knees. It is our practice to extend the upper extremity on the side on which the graft is to be based 90 degrees. This will prevent the graft from being too taut when the patient moves the extremity. Reports of pseudoaneurysms and indeed ruptures secondary to short grafts have been published. A transverse incision is made in the deltopectoral groove. The axillary artery is exposed as medial as is possible. The more medial the anastomosis, the less excursion of the graft with use of the upper extremity. The pectoralis minor tendon may be incised to facilitate this exposure. If the tendon is not incised, the tunnel should be posterior to the tendon and then brought to the midaxillary line. In general, the right side is chosen if at all possible as the right subclavian artery is less prone to atherosclerotic disease than the left. Furthermore, if a later formal aortic reconstruction is planned, a right axillofemoral graft is much less a hindrance than one on the left, especially if a retroperitoneal approach is planned. Bilateral groin incisions are made. If the tunnel connecting the ipsilateral groin incision to the axillary incision can be created without a counterincision, this maneuver should be performed in that manner. If not, a counterincision can be made in the patient's flank. The counterincision seems to be prone to infection, and we try to avoid its use. Grafting is then done in the usual manner. The long portion of the graft should be at least 8 mm and preferably 10 or 12 mm in diameter to prevent a functional aortic stenosis. Mortality figures are higher for these patients than for patients having aortic reconstruction, ranging from 10% to 15%. This is because this is a population of much sicker patients. Five-year primary patencies vary greatly, but approximately 50% failure can be expected during the course of 5 years.

#### Femoral-Femoral Artery Bypass Grafting

Femoral arteries are exposed in the standard manner. Most surgeons now use a bucket-handle approach rather than trying to create a completely antegrade sigmoid-shaped reconstruction. This is done superior to the pubis at the subcutaneous level. Either polyester or expanded polytetrafluoroethylene can be used. Grafts of at least 7 or 8 mm are usually preferred. Patency of this has not been nearly as good as was first expected, ranging from 60% to 80% at 5 years. See Figure 61-15.

#### Iliofemoral Artery Bypass Grafting

In-line reconstruction of isolated external iliac artery lesions is preferable to femoral-femoral artery grafting if the patient's physiology and anatomy will allow. A flank incision is made and the

retroperitoneal plane developed. The proximal anastomosis is performed to the common iliac artery or to the distal aorta as is necessary and then brought through that tunnel into the groin. Patency rates for this at 5 years are in the 90% range. See Figure 61-16.

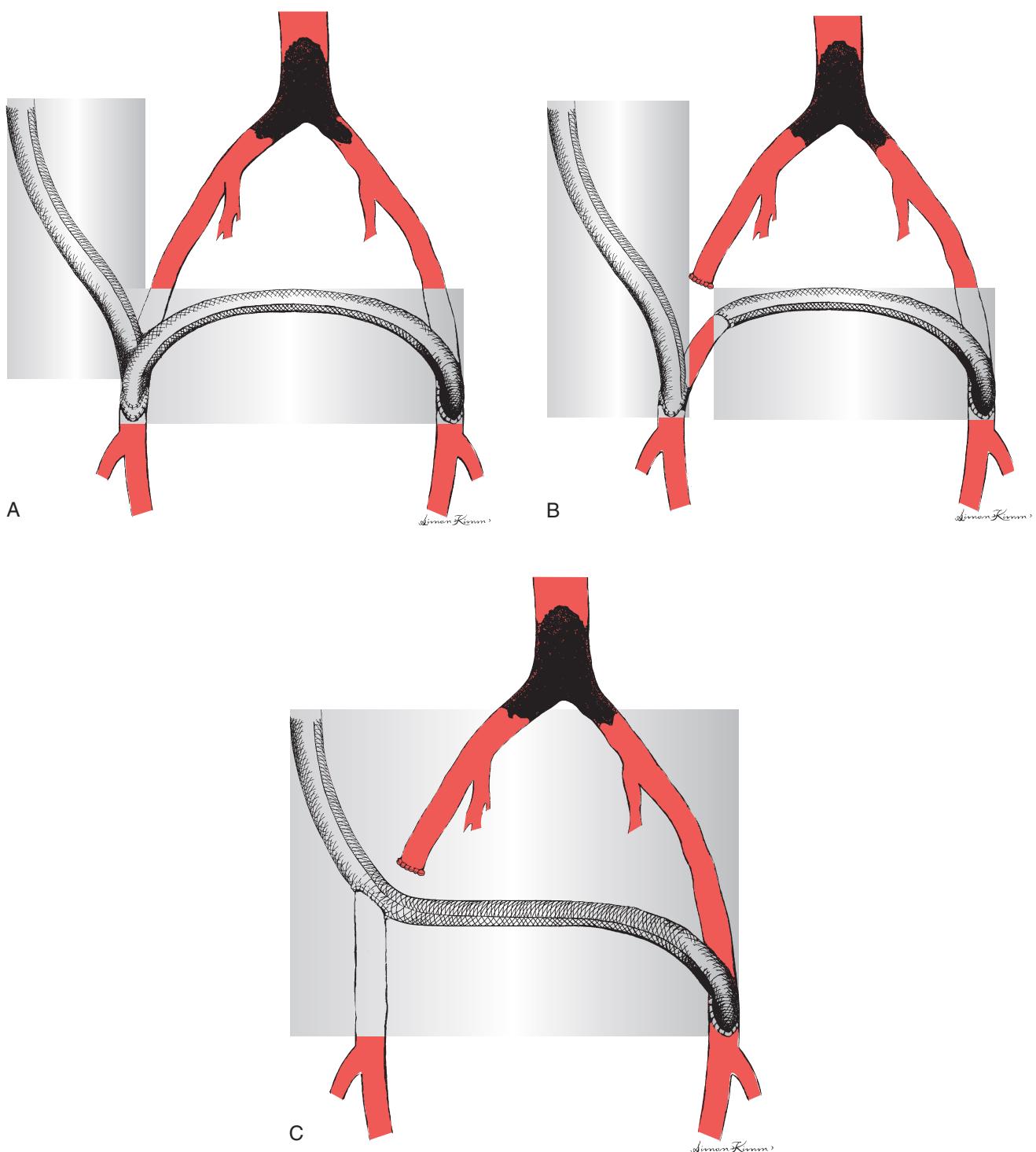
#### Aortoiliac Endarterectomy

This operation is usually done through a midline incision. In contradistinction to exposure for aortic grafting, for endarterectomy, the aorta, the common iliac arteries, and the origins of the internal and external iliac arteries all need to be circumferentially exposed. In addition to the clamping of the aorta and the iliac arteries, it is also best to clamp the lumbar arteries with small clamps to prevent annoying backbleeding, which impedes accurate endarterectomy. The patient is heparinized. Control is obtained of the aorta and the iliac arteries. A vertical aortotomy is made. With use of an elevator, an endarterectomy of the aorta down to the origins of the common iliacs is performed. At this point, either transverse incisions, which is our preference, or vertical incisions may be made at the distal common iliac arteries. The endarterectomy plane is begun here. There may be a tongue of atherosclerosis extending into the origin of the external iliac and the internal iliac. These are elevated. A stripper, such as a Wylie stripper, is used. Classically, this is passed in a retrograde manner. It may be that in some patients with deep pelvises, passing in an antegrade manner is more advantageous. An appropriate-sized stripper is picked, and the endarterectomy of the iliac artery is completed. The atherosclerotic plaque from both the aorta and the iliac arteries may be brought out as a single specimen if retrograde iliac endarterectomy has been performed. The arterial incisions are closed primarily with fine permanent suture after appropriate flushing and ascertainment of good end points. When flow is restored, interestingly enough, the aorta remains essentially the same size and the common iliac arteries balloon up much like pantaloons. The retroperitoneum and abdomen are then closed in standard manner. See Figure 61-17.

#### Complications of Open Aortic Surgery

A number of complications may arise after operations to repair the aorta, whether for aneurysmal or occlusive disease.<sup>101</sup> These may be site-specific complications, such as wound infection or hematoma, also commonly seen with endograft approaches. Of more concern and major morbidity are the intra-abdominal and systemic complications. Cardiac ischemia is the most frequent complication of open aortic surgery, and in the very best of hands one can expect that 50% of deaths related to aortic reconstruction will be attributable to the heart. Only a minority of patients with occlusive disease have normal coronary arteries. Stress testing, cardiac angiography, and coronary intervention (catheter based or, more rarely, open) have reduced mortality for direct aortic operations. Some specialty centers with aggressive heart evaluation management schemes have reported mortality in the range of 1% to 2.5%.<sup>95</sup>

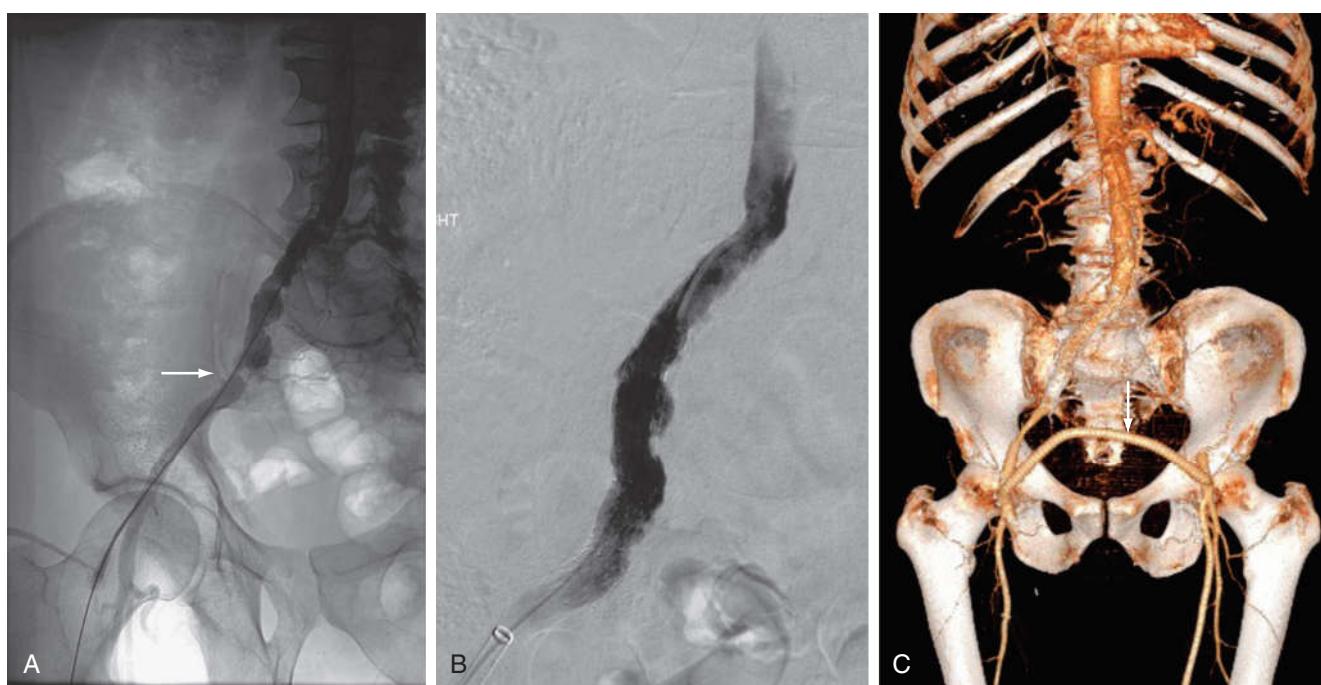
Renal insufficiency is a common complication and may result from embolization from clamping, prolonged ischemia with suprarenal clamping, intrinsic renal artery disease, hypovolemia, or hypoperfusion. It is exacerbated by paravisceral aortic repair and intraoperative complications. It most probably relates directly to the patient's preoperative renal and cardiac status. Accurate assessment of the patient's anatomy and a precise preoperative plan for clamping site and sequence are necessary to minimize the incidence of perioperative renal insufficiency.



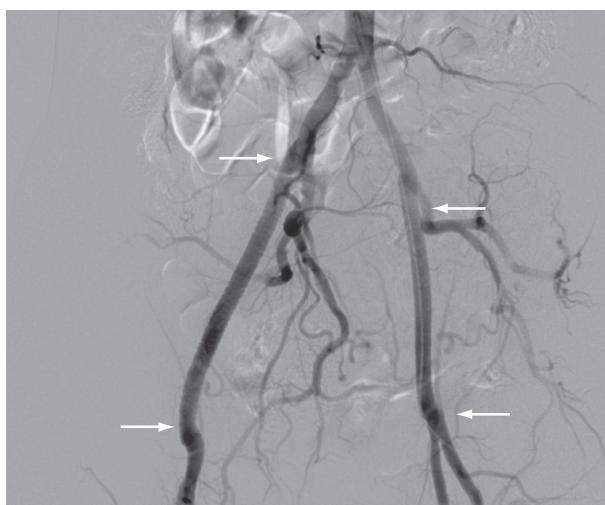
**FIGURE 61-14** Three configurations of axillobifemoral bypass grafts. All three are shown with a right-sided axillofemoral graft component. **A**, The most common configuration. **B** and **C**, Modifications described by Blaisdell and associates (**B**) and Rutherford and Rainer (**C**), designed to prevent competitive inflow from a patent ipsilateral iliac system. (From Cronenwett J, Johnston KW, editors: *Rutherford's vascular surgery*, ed 7, Philadelphia, 2011, Elsevier.)

Pulmonary dysfunction is a frequent and serious complication. This, too, is more prevalent with proximal and paravisceral aortic procedures. Transverse abdominal incisions, epidural analgesia, and retroperitoneal approaches may mitigate pulmonary complications.

Abdominal wall hernia is a common late complication of open aneurysm repair, occurring at a much higher rate (10% to 33%) than after other open operations requiring midline laparotomy, suggesting that there are patient factors associated with the pathology of AAA that predispose this population to hernia. However,



**FIGURE 61-15** **A**, Preoperative angiogram demonstrating occlusion of the left iliac system in severe focal atherosclerotic disease of the right iliac artery (arrow). **B**, Magnified view of the right iliac system after angioplasty and stent placement to establish adequate inflow for femoral-femoral bypass graft. **C**, Three-dimensional CT reconstruction demonstrating completed right to left femoral-femoral bypass graft (arrow).



**FIGURE 61-16** Arteriogram demonstrating iliofemoral bypasses extending from the bilateral common iliac to common femoral arteries (arrows).

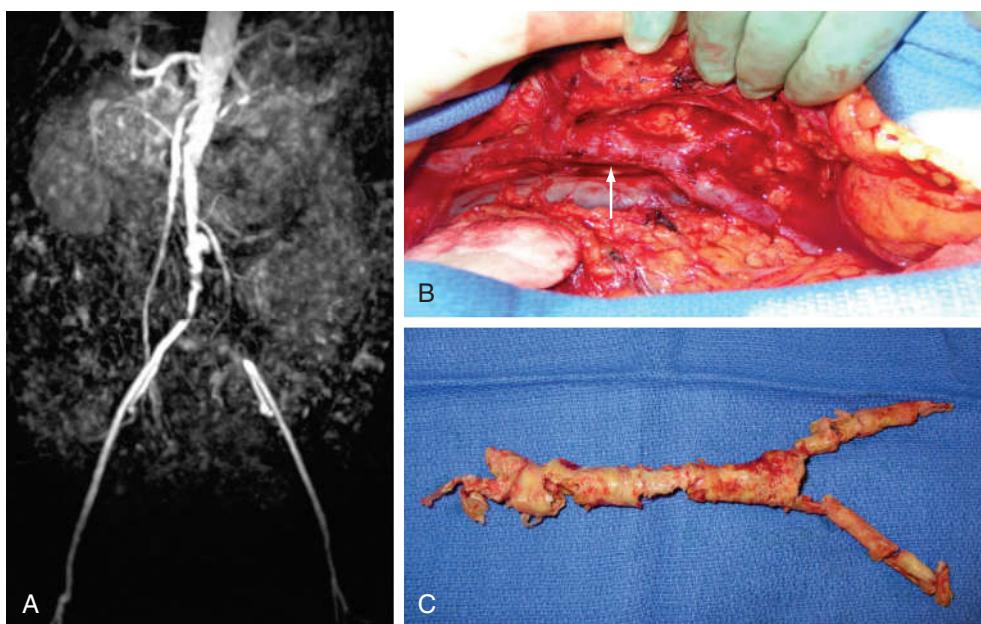
attention to fascial closure remains important to minimize this complication; a prospective study has demonstrated a relatively low 11.6% rate of midline hernia, with a suture length to wound length ratio of less than 4:1 predictive of hernia development. This study, contrary to earlier findings, did not note a statistically significant difference between the aneurysm and occlusive disease groups.<sup>102</sup> Retroperitoneal exposures crossing the flank are prone to subsequent abdominal wall laxity, which is not a true hernia but remains bothersome to patients.

Graft limb thrombosis occurs in 5% to 10% of patients and is associated with female gender, younger patients, and extra-anatomic bypass graft.<sup>96,97</sup>

An anastomotic pseudoaneurysm may be a sterile process or the result of infection. These pseudoaneurysms occur more frequently at the femoral anastomosis than at iliac and aortic anastomoses, which may reflect the higher rate of wound complications and graft infection in this region and the more clinically apparent nature of degeneration at the femoral site. Anastomotic pseudoaneurysms may result from degeneration of the suture line. True aneurysms tend to be para-anastomotic in nature, forming in the aorta proximal to or the iliac or femoral arteries distal to an aortic graft. True aneurysms occur more frequently in patients treated for aneurysmal than occlusive disease. Hypertension, COPD, smoking, hyperlipidemia, suture type, technical failures, and post-operative wound complications may be associated with this phenomenon.

Detection of anastomotic pseudoaneurysms in the iliac or aortic positions is highly reliant on imaging. Prospective studies using routine imaging of arterial grafts in a variety of anatomic positions have demonstrated higher rates of anastomotic pseudoaneurysm than those relying on clinical detection. Routine surveillance with ultrasound, for instance, demonstrated intra-abdominal anastomotic pseudoaneurysms in 10% of patients and 6.3% of aortic anastomoses after abdominal aortic graft placement at a mean interval of 12 years from operation.<sup>54</sup> CT and MRI provide excellent visualization of pseudoaneurysm and aneurysmal degeneration of the para-anastomotic area (Fig. 61-18).

Although some anastomotic pseudoaneurysms are sterile, it is prudent to begin evaluation and treatment with a presumption of infection. Diagnosis should include history (fever, chills, malaise, or weight loss), physical examination (erythema, fluctuant mass,



**FIGURE 61-17** **A**, Preoperative MRA demonstrating severe atherosclerotic disease involving the infrarenal aorta and both common iliac arteries. **B**, Intraoperative photograph of completed aortoiliac endarterectomy showing suture line of primary closure (arrow). **C**, Photograph of intact specimen demonstrating contiguous near-occlusive plaque.



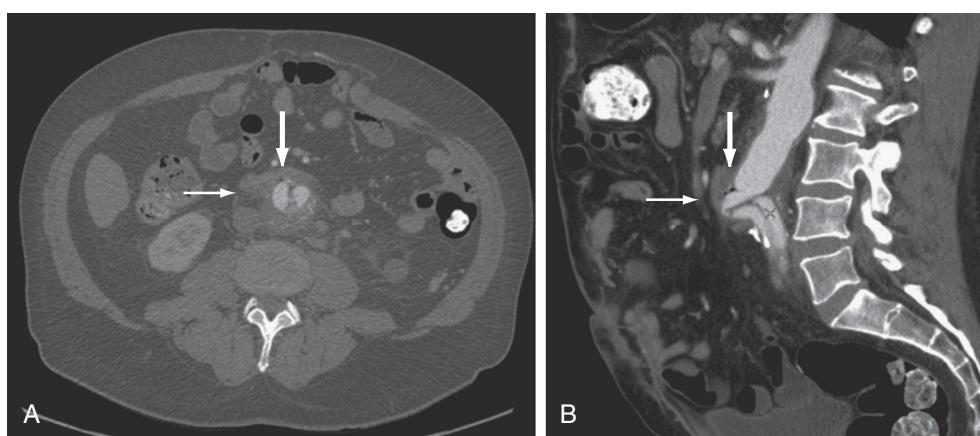
**FIGURE 61-18** CT demonstrating large anastomotic pseudoaneurysm arising at the right femoral anastomosis of an aortofemoral graft.

induration, drainage, or tenderness to palpation), and laboratory evaluation (complete blood count, blood and fluid cultures, C-reactive protein level, or erythrocyte sedimentation rate). With regard to imaging, ultrasound may demonstrate the pseudoaneurysm itself as well as perigraft fluid suggestive of infection. CT

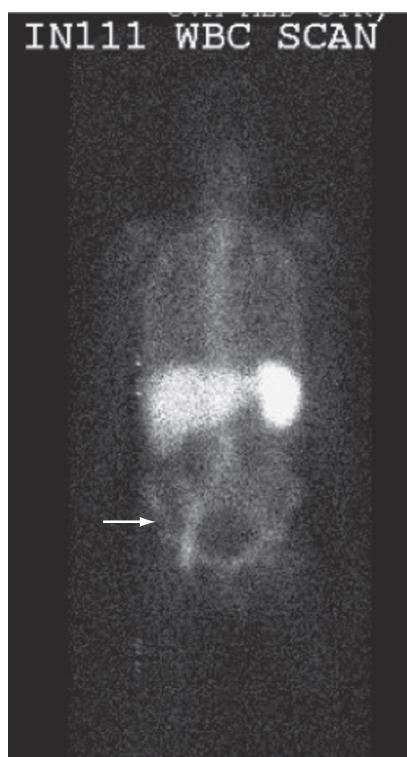
and MRI may more completely characterize these findings (Fig. 61-19). The use of nuclear medicine modalities, such as indium In 111 or technetium Tc 99m tagged white blood cell scans, has greatly improved the surgeon's ability to evaluate for infection in a noninvasive manner (Fig. 61-20). Whereas positive cultures are definitive, many of the organisms common in graft infections are fastidious and may yield multiple negative cultures despite clear clinical evidence of infection. When diagnostic investigation yields evidence of infection, thorough débridement of infected material accompanied by *in situ* or extra-anatomic arterial reconstruction is the mainstay of management. Surgical management of anastomotic pseudoaneurysm is dictated by the presence or absence of infection, the nature of presentation, and the surgeon's experience and preference. In an uninfected field, débridement and interposition grafting may suffice. Whereas aneurysmal degeneration at or near the graft anastomoses historically necessitated open repair, a growing proportion are now successfully managed by endovascular techniques.<sup>103</sup> In any event, expeditious treatment is appropriate for a large, enlarging, or symptomatic lesion.

#### Surgical Treatment of Aortic Graft Infection

Infection should be managed by removal of the entire affected graft with débridement of all infected or devitalized tissue, traditionally accompanied by extra-anatomic reconstruction. Staging of this process by performing the extra-anatomic bypass and then either proceeding to débridement or permitting a recovery interval of up to several days greatly improved the historically substantial morbidity and mortality associated with primary graft resection followed by reconstruction.<sup>104</sup> Further investigation has suggested that in instances of infection limited to a single limb of the graft, satisfactory results may be achieved by limiting resection to the involved limb or limb segment, followed by *in situ* or extra-anatomic reconstruction and, according to the surgeon's



**FIGURE 61-19** Axial (**A**) and sagittal (**B**) CT views of an infected aortofemoral graft after repair of an infrarenal aneurysm. Foci of gas (thick arrows) and extensive inflammation (thin arrows) are visible surrounding the graft. Of note, the tortuosity of the limbs of this graft represents a technical error.



**FIGURE 61-20**  $^{111}\text{In}$ -tagged white blood cell scan image at 20 hours of delay demonstrates abnormal uptake in the region of the right limb of an aortofemoral graft (arrow).

preference, combined with sterile antibiotic irrigation of the field through operatively placed drains.<sup>105</sup> However, recurrent graft infection, graft thrombosis, and the nearly invariably fatal complication of aortic stump infection or disruption continue to contribute to significant morbidity, mortality, and limb loss after this operation. Thorough débridement, layered closure, and vascularized pedicle flap coverage of the aortic stump are considered of paramount importance in avoiding the last complication.

In situ reconstruction may be accomplished by using rifampin-soaked or silver-coated polyester graft, cryopreserved arterial

allograft, or saphenofemoral vein allograft.<sup>106-109</sup> The first of these is the most expeditious but yields a higher rate of reinfection as well as poor results in grossly purulent operative fields. Those espousing its use tend to embrace adjuncts such as wrapping the new graft and anastomoses in vascularized pedicle flaps, antibiotic irrigation therapy, or creation of clean retroperitoneal tunnels. The neoaortoiliac system venous autograft reconstruction described by Clagett is a lengthy procedure that places significant demands on both the patient and the operative team but yields the lowest reported rate of reinfection. Reported results using cryopreserved arterial allograft for in situ reconstruction have been mixed but generally reflect an intermediate rate of reinfection.<sup>106</sup> Endovascular treatment of anastomotic disruptions is generally limited to cases without evidence of infection and may use covered stent exclusion of the pseudoaneurysm or embolization of the pseudoaneurysm, generally with coils or occlusion devices. Aortoenteric fistula represents the most severe manifestation of aortic graft infection. It rarely occurs as a primary process, generally due to erosion of an untreated aneurysm into the duodenum. Most commonly, the lesion is at the point of contact of the third portion of the duodenum with the proximal graft anastomosis. Aortoenteric fistula has been reported in association with aortic stent graft placement. This complication generally is manifested with herald upper or lower gastrointestinal bleeding, which, if left untreated, may be followed by exsanguinating hemorrhage. Aortoenteric fistula should be suspected when gastrointestinal bleeding develops in a patient with a history of abdominal aortic surgery or endograft. Although endoscopy may confirm the diagnosis, demonstrating either an erosion or frank exposure of graft most frequently in the duodenum, CT is a more sensitive diagnostic study. The placement of synthetic or endovascular grafts in the setting of infected pseudoaneurysm, graft infection, primary aortic infection, or aortoenteric fistula is perhaps best viewed as a temporizing measure and is attended by high rates of re-intervention, morbidity, and mortality.<sup>110</sup> Repair of aortoenteric fistula or primary aortic infection follows many of the same principles as for infected graft removal: consideration of staged extra-anatomic bypass, appropriate selection of conduit if in situ reconstruction is elected, establishment of safe proximal control, wide débridement of infected material, coverage of in situ graft with a pedicle flap such as omentum, and appropriate use of antibiotic and antifungal therapy.

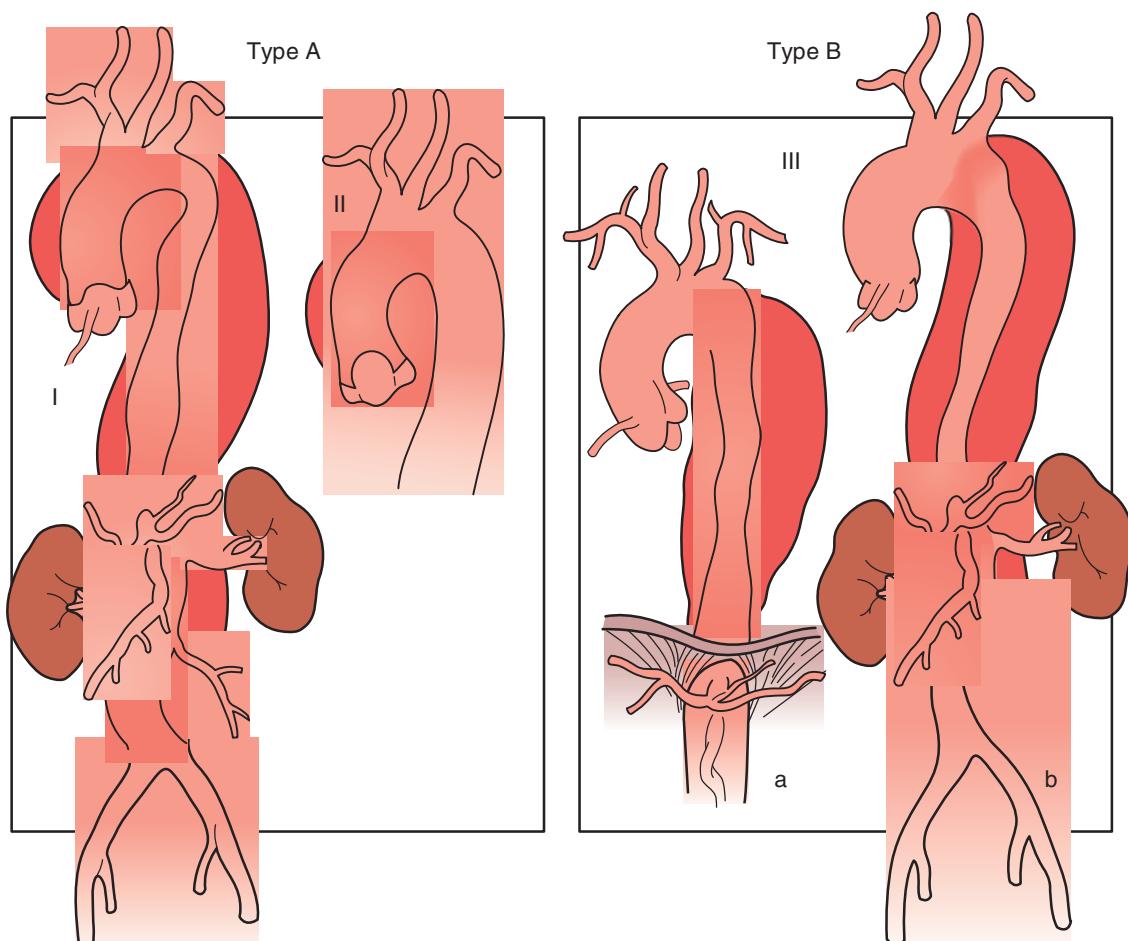
## AORTIC DISSECTION

Aortic dissection occurs when a defect in the intimal layer of the vessel permits blood to create a false channel within the aortic wall, typically between media and adventitia. The aorta is divided into true and false lumens, separated by a septum referred to as the dissection flap. A number of conditions, including connective tissue disorders such as Marfan syndrome, hypertension, and pregnancy, are associated with the development of aortic dissection, as are activities such as cocaine abuse and power weightlifting.<sup>111</sup>

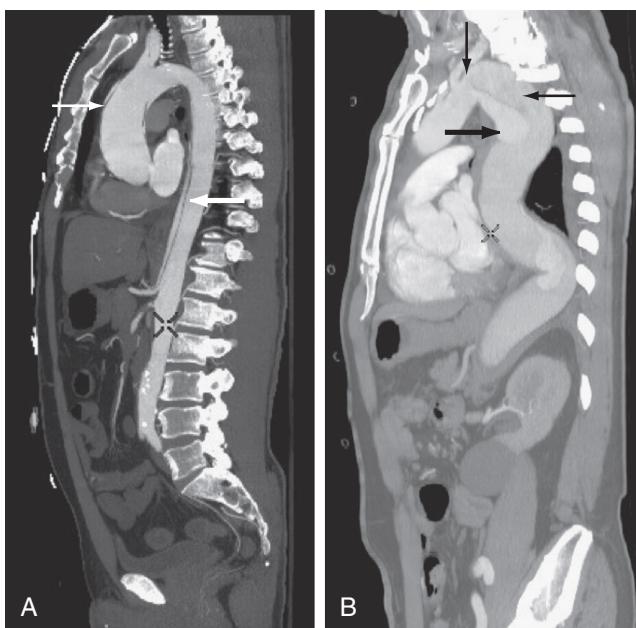
A number of important distinctions must be made once the diagnosis of aortic dissection is made. The DeBakey and Stanford classification systems define dissections on the basis of anatomic extent. DeBakey type I (involving both ascending and thoracoabdominal aorta) and type II (limited to the ascending aorta) dissections correspond to the Stanford type A (any involvement of the ascending aorta); DeBakey type IIIa (confined to the descending thoracic aorta) and type IIIb (involving the descending thoracic and abdominal aorta) correspond to the Stanford type B (not involving the ascending aorta; Figs. 61-21 and 61-22).

Type A dissection typically is manifested acutely with chest or back pain, commonly described as ripping or tearing in nature. This may be accompanied by profound hypotension, particularly in the setting of pericardial tamponade or disruption of the aortic valve, and distal hypoperfusion as seen in type B dissections. Distal pulse deficits or other evidence of malperfusion in a patient presenting with sudden-onset, severe chest or back pain should immediately prompt evaluation for aortic dissection. CT and echocardiography both provide the ability not only to diagnose dissection but to rapidly assess the status of the proximal aorta, permitting the critical distinction between type A and type B lesions. Acute type A dissection is generally considered a surgical emergency. Repair necessitates the use of adjuncts such as cardiopulmonary bypass and hypothermic circulatory arrest and, on occasion, replacement of the aortic valve in addition to replacement of the ascending aorta.<sup>73</sup>

Type B dissection is further characterized as acute ( $\leq 14$  days from onset of symptoms) or chronic ( $> 14$  days from initial symptoms) and, within these categories, as uncomplicated or complicated. Acute type B dissections will also frequently be manifested with tearing chest or back pain, often in the setting of severe



**FIGURE 61-21** Classification systems for aortic dissection. Stanford type A corresponds to DeBakey type I (involving the ascending and descending aorta) and type II (involving the ascending aorta). Stanford type B corresponds to DeBakey type III (origin of the dissection distal to the left subclavian artery and involving the descending aorta only or the descending and abdominal aorta).

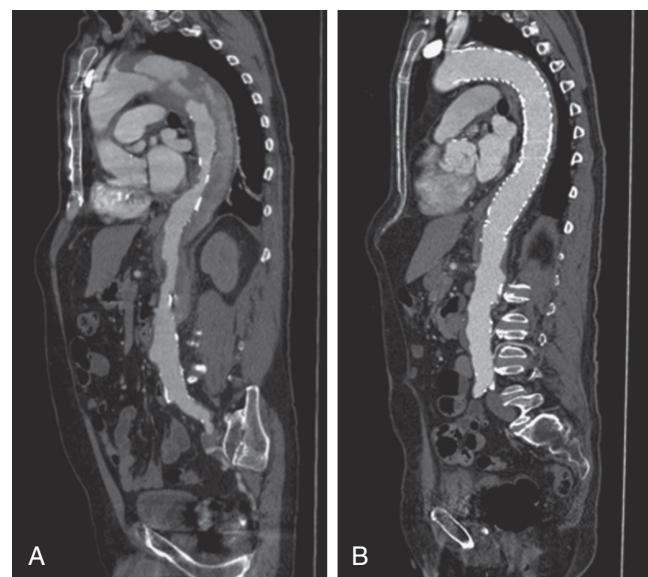


**FIGURE 61-22** **A**, Stanford type A dissection with dissection flap visible in both the ascending (thin arrow) and descending (thick arrow) aorta. **B**, Stanford type B dissection demonstrating proximal entry tear, fenestration distal to the left subclavian artery (thin vertical arrow) and differential filling of true (thick arrow) and false (thin horizontal arrow) lumens.

hypertension. Malperfusion of the spine and renal, visceral, or lower extremity vessels may complicate the presentation. Rarely, patients may present with frank rupture. Anatomically, type B dissections generally originate from a primary tear in the proximal descending thoracic aorta just distal to the origin of the left subclavian artery. Extension is typically antegrade, extending as far as the iliac or femoral arteries, although retrograde extension may occur. Fenestrations or openings in the dissection flap may permit communication between true and false lumens at intervals along the length of the dissection.

Penetrating atherosclerotic ulcer and intramural hematoma are also considered to be variants of aortic dissection. Penetrating atherosclerotic ulcer represents focal intimal ulceration of the aorta within a region of preexisting atherosclerotic disease. These lesions may eventually extend into the media and evolve into a true dissection. Intramural hematoma occurs when intramural thrombus is found without evidence of associated intimal disruption, possibly as a result of disrupted vasa vasorum within the aortic wall. Like penetrating atherosclerotic ulcer, intramural hematoma may evolve into dissection should a frank intimal tear develop.<sup>112</sup>

CT remains the mainstay of diagnostic imaging as it provides excellent anatomic data, ability to localize the entry tear and fenestrations, assessment of branch vessel patency, and detection of extravasation of contrast material consistent with rupture (Fig. 61-23). Electrocardiography-gated techniques have recently enabled the acquisition of motion-free images of the proximal aorta. CT imaging is widely available, and excellent studies may be obtained rapidly with modern multidetector helical scanners, often in a matter of a few minutes. As in abdominal aortic imaging, the effect of iodinated contrast material on renal function remains the principal drawback of CT.



**FIGURE 61-23** Reformatted CT images demonstrating a type B dissection before (**A**) and after (**B**) placement of a thoracic endograft. Note coverage of the proximal fenestration, with resulting false lumen thrombosis and aortic remodeling.

MRI is substantially more time-consuming to obtain and may be limited by patient factors, such as the presence of metallic debris or medical implants, that preclude its use. Electrocardiography gating of contrast-enhanced MRI can provide exceptional motion-free images of the proximal aorta. In addition, unlike contrasted CT, MRI permits appreciation of direction of blood flow as well as the calculation of values such as peak flow and velocity.

Catheter angiography provides excellent information about aortic and branch anatomy and involvement; it has the ability to visualize dissection flap anatomy and fenestrations (particularly with the adjunct of intravascular ultrasound) and to evaluate dissection physiology through measuring true and false lumen pressures and pressure differentials across fenestrations. In addition, angiography permits intervention on the coronary vessels, aorta, and branch vessels at the same setting.

The treatment of type B dissection is in evolution, particularly since the advent of endovascular therapy. Traditionally, intensive medical management has been the mainstay of therapy for uncomplicated dissection; dissections complicated by rupture, aneurysmal expansion, and evidence of malperfusion or, according to some sources, intractable pain have been managed surgically.<sup>113</sup> Aortic branch vessels to the lower extremities, viscera, and spine may originate from either true or false lumen. Malperfusion may result from dynamic compression of the true lumen by the false lumen, thrombosis of one or both lumens, or static obstruction due to extension of the dissection into the branch vessel. It may be manifested as new-onset renal dysfunction, abdominal pain and mesenteric ischemia, lower extremity ischemia, or neurologic function ranging from paresthesia to paraplegia.

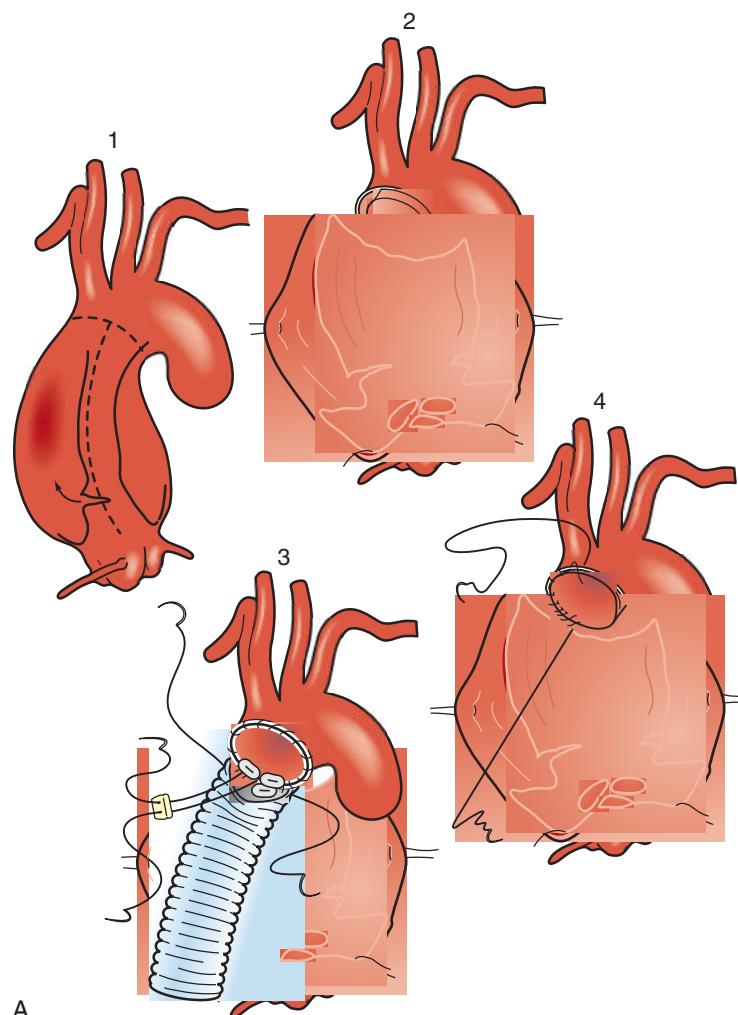
Open surgical therapy for type B dissection may consist of replacement of the descending aorta or fenestration of the abdominal aorta to address visceral or limb malperfusion. Whereas replacement addresses the risk of further aortic enlargement or rupture within the replaced segment of aorta, fenestration addresses malperfusion exclusively. This procedure, which is rarely

performed, involves creating a transverse (if interposition graft is anticipated) or longitudinal aortotomy in the dissected but non-aneurysmal paravisceral aorta to excise a portion of the dissection flap to permit perfusion of the mesenteric and renal arteries or in the infrarenal aorta to reperfuse the lower limbs. Similar effect may be achieved in an endovascular fashion by traversing the dissection flap at the level of the desired fenestration and using an angioplasty balloon to enlarge the opening. Angioplasty may be used alone or with subsequent stent placement within the fenestration.<sup>114</sup>

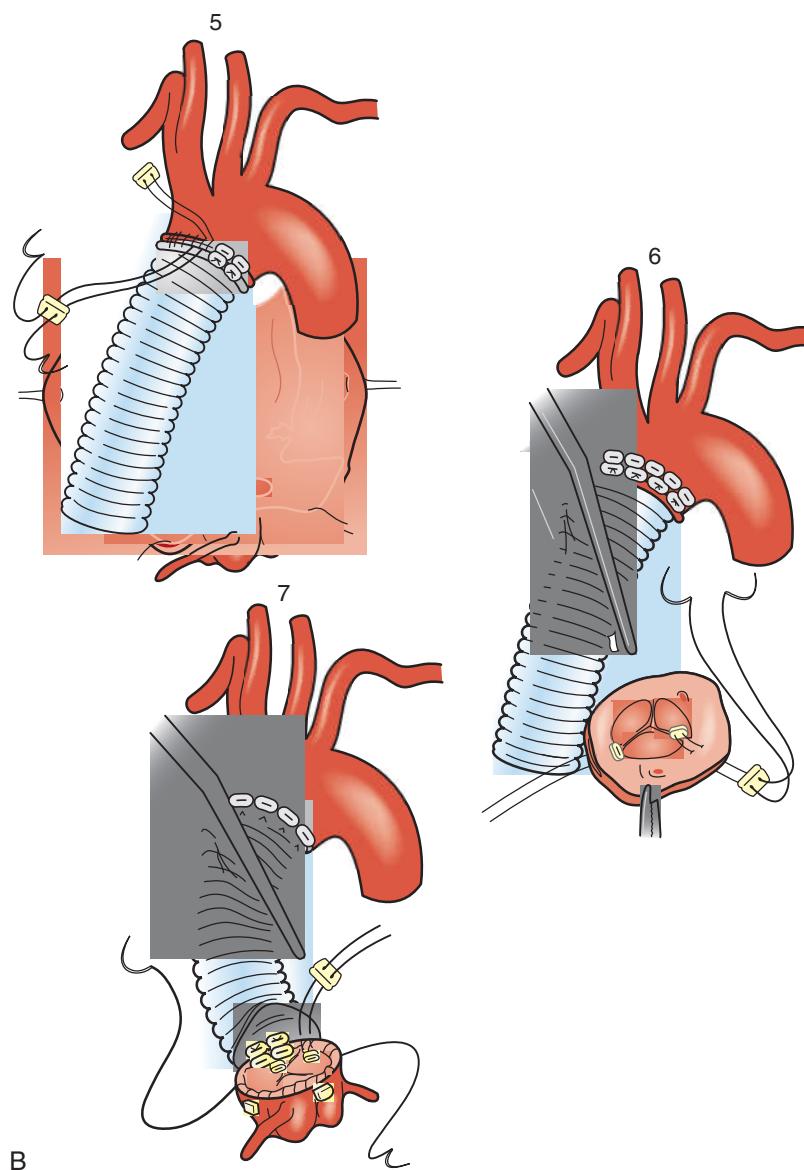
Historically, open surgical therapy for acute, complicated type B dissection has been associated with high rates of morbidity and mortality. A review of data from the International Registry of Acute Aortic Dissection (IRAD) yielded a 29.3% overall rate of in-hospital mortality among patients treated surgically for acute, complicated type B dissection, whereas Panneton and colleagues reported a 43% rate of operative mortality for emergency open surgical fenestration. In the IRAD report, 69% of the patients were treated with replacement of the descending aorta, 28% with partial or complete replacement of the aortic arch, and 9% with surgical fenestration. Surgical or endovascular approaches to

revascularization of malperfused branch territories were used in 20% and 9% of patients, respectively.<sup>114,115</sup> Open repair for chronic dissection may yield more favorable results; a recent series of 69 patients reflected an exemplary 5.8% 30-day mortality rate, 3% stroke rate, and 6% rate of spinal cord ischemia, and a recent systematic review found that other contemporary series also suggested acceptable results.<sup>116,117</sup>

In 1999, Dake and colleagues reported the placement of endovascular stent grafts for acute aortic dissection as an alternative to traditional surgical therapy. The principle of endovascular stent graft therapy is coverage of the entry tear, depressurizing the false lumen; this results in expansion of the true lumen and thrombosis of the false lumen with subsequent remodeling of the aorta, with an increase in the diameter of the true lumen and a decrease in the diameter of the false lumen and, ideally, the overall aortic diameter (Fig. 61-24).<sup>118</sup> In the setting of chronic dissection, progressive thickening of the intimal flap has generally occurred and distal "reentry" fenestrations are well established, rendering false lumen thrombosis more difficult to induce than in acute dissection and probably decreasing the overall occurrence of favorable remodeling.<sup>112</sup>



**FIGURE 61-24 A.** Ascending aorta dissection repair. Under circulatory arrest, the ascending aorta and transverse arch are opened, exposing the dissection (1, 2). The false lumen is obliterated (3). The graft is sutured to the transverse arch in an open distal anastomosis (4).



**FIGURE 61-24, cont'd B,** The distal anastomosis is reinforced (5). The aortic valve is resuspended (6). The proximal anastomosis is constructed (7).

Adjunctive techniques, such as fenestration or direct stent placement to restore flow to obstructed branch vessels, are also important components of modern therapy for dissection. As in the endovascular management of thoracic aortic aneurysm, cervical reconstruction of the supra-aortic branches may facilitate coverage of portions of the aortic arch to achieve an adequate proximal seal zone for the device.

At present, the standard of care for acute, uncomplicated type B dissection is in flux. Whereas medical therapy remains the standard of care, the dismal natural history of medically managed type B dissection is clear, as the majority of patients fail to respond to therapy over time. A review of a cohort of 298 patients presenting with acute type B dissection and managed medically demonstrated early failure (within the first 15 days) in 37 patients (12%), resulting in 25 interventions and 15 deaths. During the study period, there were 174 failures (58.4%), primarily related to aneurysmal degeneration, resulting in a total of 87 interventions and

119 deaths. Ultimately, intervention-free survival was only 55% at 3 years and dropped to 41% by 6 years.<sup>119</sup> In the setting of these prospects, the potential for aortic remodeling and the prevention of aneurysmal dilation over time has prompted growing interest in early stent grafting in this population. A randomized trial of TEVAR and optimal medical treatment versus optimal medical treatment alone (INSTEAD-XL trial) has recently reported landmark analysis suggesting a benefit for TEVAR for all end points, including all-cause mortality, aorta-specific mortality, and disease progression, between 2 and 5 years, with favorable results in each category significantly associated with stent graft-induced false lumen thrombosis.<sup>120</sup>

The quest for aortic remodeling has spurred additional investigation, including new approaches such as the combination of a covered proximal stent graft with a distal bare-metal aortic stent as part of the STABLE trial.<sup>121</sup> A version of this device, the Cook Zenith Dissection Endovascular System, is commercially available

in European markets. Another potentially important development in this area is the application of fenestrated and branched graft techniques to address aneurysmal degeneration of the abdominal aorta and even the aortic arch.<sup>122</sup>

Procedural complications of endovascular stent graft therapy for dissection generally parallel those of TEVAR. There are, however, several complications largely specific to the treatment of dissection, including retrograde dissection (converting a type B into a type A dissection), creation or worsening of malperfusion, and continued false lumen filling with inability to induce thrombosis.

## REFERENCES

- Schermerhorn ML, Giles KA, Sachs T, et al: Defining perioperative mortality after open and endovascular aortic aneurysm repair in the US Medicare population. *J Am Coll Surg* 212:349–355, 2011.
- Deaths: Final data for 2013. NCVR Volume 64, Number 2. CDC. <[http://www.cdc.gov/nchs/data/nvsr/nvsr64/nvsr64\\_02.pdf](http://www.cdc.gov/nchs/data/nvsr/nvsr64/nvsr64_02.pdf)>. Accessed September 23, 2015.
- Wassef M, Upchurch GR, Jr, Kuivaniemi H, et al: Challenges and opportunities in abdominal aortic aneurysm research. *J Vasc Surg* 45:192–198, 2007.
- Chaikof EL, Brewster DC, Dalman RL, et al: The care of patients with an abdominal aortic aneurysm: The Society for Vascular Surgery practice guidelines. *J Vasc Surg* 50:S2–S49, 2009.
- Allardice JT, Allwright GJ, Wafula JM, et al: High prevalence of abdominal aortic aneurysm in men with peripheral vascular disease: Screening by ultrasonography. *Br J Surg* 75:240–242, 1988.
- O'Kelly TJ, Heather BP: General practice-based population screening for abdominal aortic aneurysms: A pilot study. *Br J Surg* 76:479–480, 1989.
- Bengtsson H, Norrgard O, Angquist KA, et al: Ultrasonographic screening of the abdominal aorta among siblings of patients with abdominal aortic aneurysms. *Br J Surg* 76:589–591, 1989.
- Shapira OM, Pasik S, Wassermann JP, et al: Ultrasound screening for abdominal aortic aneurysms in patients with atherosclerotic peripheral vascular disease. *J Cardiovasc Surg (Torino)* 31:170–172, 1990.
- Webster MW, Ferrell RE, St Jean PL, et al: Ultrasound screening of first-degree relatives of patients with an abdominal aortic aneurysm. *J Vasc Surg* 13:9–13, discussion 13–14, 1991.
- Bengtsson H, Sonesson B, Lanne T, et al: Prevalence of abdominal aortic aneurysm in the offspring of patients dying from aneurysm rupture. *Br J Surg* 79:1142–1143, 1992.
- MacSweeney ST, O'Meara M, Alexander C, et al: High prevalence of unsuspected abdominal aortic aneurysm in patients with confirmed symptomatic peripheral or cerebral arterial disease. *Br J Surg* 80:582–584, 1993.
- Lindholt JS, Juul S, Henneberg EW, et al: Is screening for abdominal aortic aneurysm acceptable to the population? Selection and recruitment to hospital-based mass screening for abdominal aortic aneurysm. *J Public Health Med* 20:211–217, 1998.
- Lederle FA, Johnson GR, Wilson SE, et al: Prevalence and associations of abdominal aortic aneurysm detected through screening. Aneurysm Detection and Management (ADAM) Veterans Affairs Cooperative Study Group. *Ann Intern Med* 126:441–449, 1997.
- van der Graaf Y, Akkersdijk GJ, Hak E, et al: Results of aortic screening in the brothers of patients who had elective aortic aneurysm repair. *Br J Surg* 85:778–780, 1998.
- Lawrence PF, Lorenzo-Rivero S, Lyon JL: The incidence of iliac, femoral, and popliteal artery aneurysms in hospitalized patients. *J Vasc Surg* 22:409–415, discussion 415–416, 1995.
- Biebl M, Hakaim AG, Hugl B, et al: Endovascular aortic aneurysm repair with the Zenith AAA Endovascular Graft: Does gender affect procedural success, postoperative morbidity, or early survival? *Am Surg* 71:1001–1008, 2005.
- Velazquez OC, Larson RA, Baum RA, et al: Gender-related differences in infrarenal aortic aneurysm morphologic features: Issues relevant to Ancure and Talent endografts. *J Vasc Surg* 33:S77–S84, 2001.
- Brown LC, Powell JT, UK Small Aneurysm Trial Participants: Risk factors for aneurysm rupture in patients kept under ultrasound surveillance. *Ann Surg* 230:289–296, discussion 296–297, 1999.
- Brown PM, Zelt DT, Sobolev B: The risk of rupture in untreated aneurysms: The impact of size, gender, and expansion rate. *J Vasc Surg* 37:280–284, 2003.
- Cronenwett JL, Murphy TF, Zelenock GB, et al: Actuarial analysis of variables associated with rupture of small abdominal aortic aneurysms. *Surgery* 98:472–483, 1985.
- Norman PE, Powell JT: Abdominal aortic aneurysm: The prognosis in women is worse than in men. *Circulation* 115:2865–2869, 2007.
- Englesbe MJ, Wu AH, Clowes AW, et al: The prevalence and natural history of aortic aneurysms in heart and abdominal organ transplant patients. *J Vasc Surg* 37:27–31, 2003.
- Sonesson B, Sandgren T, Lanne T: Abdominal aortic aneurysm wall mechanics and their relation to risk of rupture. *Eur J Vasc Endovasc Surg* 18:487–493, 1999.
- Hall AJ, Busse EF, McCarville DJ, et al: Aortic wall tension as a predictive factor for abdominal aortic aneurysm rupture: Improving the selection of patients for abdominal aortic aneurysm repair. *Ann Vasc Surg* 14:152–157, 2000.
- Fillinger MF, Marra SP, Raghavan ML, et al: Prediction of rupture risk in abdominal aortic aneurysm during observation: Wall stress versus diameter. *J Vasc Surg* 37:724–732, 2003.
- Fillinger MF, Raghavan ML, Marra SP, et al: In vivo analysis of mechanical wall stress and abdominal aortic aneurysm rupture risk. *J Vasc Surg* 36:589–597, 2002.
- Lederle FA: Risk of rupture of large abdominal aortic aneurysms. Disagreement among vascular surgeons. *Arch Intern Med* 156:1007–1009, 1996.
- Parkinson F, Ferguson S, Lewis P, et al: Rupture rates of untreated large abdominal aortic aneurysms in patients unfit for elective repair. *J Vasc Surg* 61:1606–1612, 2015.
- Darling RC, Messina CR, Brewster DC, et al: Autopsy study of unoperated abdominal aortic aneurysms. The case for early resection. *Circulation* 56:II161–II164, 1977.
- Engellau L, Albrechtsson U, Dahlstrom N, et al: Measurements before endovascular repair of abdominal aortic aneurysms. MR imaging with MRA vs. angiography and CT. *Acta Radiol* 44:177–184, 2003.

31. U.S. Preventive Services Task Force: Abdominal aortic aneurysm: Screening. <<http://www.uspreventiveservicestaskforce.org/uspstf/uspsaneu.htm>>. Accessed August 2, 2011.
32. McCarthy RJ, Shaw E, Whyman MR, et al: Recommendations for screening intervals for small aortic aneurysms. *Br J Surg* 90:821–826, 2003.
33. Lederle FA: Abdominal aortic aneurysm: Still no pill. *Ann Intern Med* 159:852–853, 2013.
34. Meijer CA, Stijnen T, Wasser MN, et al: Doxycycline for stabilization of abdominal aortic aneurysms: A randomized trial. *Ann Intern Med* 159:815–823, 2013.
35. Baxter BT, Terrin MC, Dalman RL: Medical management of small abdominal aortic aneurysms. *Circulation* 117:1883–1889, 2008.
36. Sconfienza LM, Santagostino I, Di Leo G, et al: When the diameter of the abdominal aorta should be considered as abnormal? A new ultrasonographic index using the wrist circumference as a body build reference. *Eur J Radiol* 82:e532–e536, 2013.
37. Hannawa KK, Eliason JL, Upchurch GR, Jr: Gender differences in abdominal aortic aneurysms. *Vascular* 17(Suppl 1):S30–S39, 2009.
38. Suckow BD, Schanzer A, Hoel AW, et al: A novel quality of life instrument for patients with an abdominal aortic aneurysm. *J Vasc Surg* 61:43S–44S, 2015.
39. Fleisher LA, Beckman JA, Brown KA, et al: ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery): Developed in collaboration with the American Society of Echocardiography, American Society of Nuclear Cardiology, Heart Rhythm Society, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, and Society for Vascular Surgery. *Circulation* 116:e418–e499, 2007.
40. Shah TR, Veith FJ, Bauer SM: Cardiac evaluation and management before vascular surgery. *Curr Opin Cardiol* 29:499–505, 2014.
41. Fleischmann KE, Beckman JA, Buller CE, et al: 2009 ACCF/AHA focused update on perioperative beta blockade: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *Circulation* 120:2123–2151, 2009.
42. Schouten O, Hoeks SE, Welten GM, et al: Effect of statin withdrawal on frequency of cardiac events after vascular surgery. *Am J Cardiol* 100:316–320, 2007.
43. Corriere M, Edwards M, Hansen KJ: Abdominal aortic aneurysm and renal artery stenosis. *Vasc Dis Manage* 5:16–21, 2008.
44. Lee SM, Takemoto S, Wallace AW: Association between withholding angiotensin receptor blockers in the early post-operative period and 30-day mortality: A cohort study of the Veterans Affairs Healthcare System. *Anesthesiology* 123:288–306, 2015.
45. Hersey P, Poullis M: Does the administration of mannitol prevent renal failure in open abdominal aortic aneurysm surgery? *Interact Cardiovasc Thorac Surg* 7:906–909, 2008.
46. Wijnen MH, Vader HL, Van Den Wall Bake AW, et al: Can renal dysfunction after infra-renal aortic aneurysm repair be modified by multi-antioxidant supplementation? *J Cardiovasc Surg (Torino)* 43:483–488, 2002.
47. Upchurch GR, Jr, Proctor MC, Henke PK, et al: Predictors of severe morbidity and death after elective abdominal aortic aneurysmectomy in patients with chronic obstructive pulmonary disease. *J Vasc Surg* 37:594–599, 2003.
48. Myers K, Hajek P, Hinds C, et al: Stopping smoking shortly before surgery and postoperative complications: A systematic review and meta-analysis. *Arch Intern Med* 171:983–989, 2011.
49. Sicard GA, Toursarkissian B: Midline versus retroperitoneal approach for abdominal aortic aneurysm surgery. In Calligaro KD, Dougherty MJ, Hollier LH, editors: *Diagnosis and treatment of aortic and peripheral arterial aneurysms*, Philadelphia, 1999, WB Saunders, pp 135–148.
50. Berland TL, Veith FJ, Cayne NS, et al: Technique of supraceliac balloon control of the aorta during endovascular repair of ruptured abdominal aortic aneurysms. *J Vasc Surg* 57:272–275, 2013.
51. Stannard A, Eliason JL, Rasmussen TE: Resuscitative endovascular balloon occlusion of the aorta (REBOA) as an adjunct for hemorrhagic shock. *J Trauma* 71:1869–1872, 2011.
52. Nishimori M, Ballantyne JC, Low JH: Epidural pain relief versus systemic opioid-based pain relief for abdominal aortic surgery. *Cochrane Database Syst Rev* (3):CD005059, 2006.
53. Ylonen K, Biancari F, Leo E, et al: Predictors of development of anastomotic femoral pseudoaneurysms after aortobifemoral reconstruction for abdominal aortic aneurysm. *Am J Surg* 187:83–87, 2004.
54. Edwards JM, Teeffey SA, Zierler RE, et al: Intraabdominal paraanastomotic aneurysms after aortic bypass grafting. *J Vasc Surg* 15:344–350, discussion 351–353, 1992.
55. Parodi JC, Palmaz JC, Barone HD: Transfemoral intraluminal graft implantation for abdominal aortic aneurysms. *Ann Vasc Surg* 5:491–499, 1991.
56. Wilt TJ, Lederle FA, Macdonald R, et al: Comparison of endovascular and open surgical repairs for abdominal aortic aneurysm. *Evid Rep Technol Assess (Full Rep)* 144:1–113, 2006.
57. Dimick JB, Upchurch GR, Jr: Endovascular technology, hospital volume, and mortality with abdominal aortic aneurysm surgery. *J Vasc Surg* 47:1150–1154, 2008.
58. Ali MM, Flahive J, Schanzer A, et al: In patients stratified by preoperative risk, endovascular repair of ruptured abdominal aortic aneurysms has a lower in-hospital mortality and morbidity than open repair. *J Vasc Surg* 61:1399–1407, 2015.
59. Chang DC, Parina RP, Wilson SE: Survival after endovascular vs open aortic aneurysm repairs. *JAMA Surg* 2015. [Epub ahead of print].
60. Heyer KS, Modi P, Morasch MD, et al: Secondary infections of thoracic and abdominal aortic endografts. *J Vasc Interv Radiol* 20:173–179, 2009.
61. Mehta M, Henretta J, Glickman M, et al: Outcome of the pivotal study of the Aptus endovascular abdominal aortic aneurysms repair system. *J Vasc Surg* 60:275–285, 2014.
62. Muller-Wille R, Schotz S, Zeman F, et al: CT features of early type II endoleaks after endovascular repair of abdominal aortic aneurysms help predict aneurysm sac enlargement. *Radiology* 274:906–916, 2015.

63. Zhou W, Blay E, Jr, Varu V, et al: Outcome and clinical significance of delayed endoleaks after endovascular aneurysm repair. *J Vasc Surg* 59:915–920, 2014.
64. Guntner O, Zeman F, Wohlgemuth WA, et al: Inferior mesenteric arterial type II endoleaks after endovascular repair of abdominal aortic aneurysm: Are they predictable? *Radiology* 270:910–919, 2014.
65. Alerci M, Giamboni A, Wyttenbach R, et al: Endovascular abdominal aneurysm repair and impact of systematic pre-operative embolization of collateral arteries: Endoleak analysis and long-term follow-up. *J Endovasc Ther* 20:663–671, 2013.
66. Sidloff DA, Gokani V, Stather PW, et al: Type II endoleak: Conservative management is a safe strategy. *Eur J Vasc Endovasc Surg* 48:391–399, 2014.
67. Quinones-Baldrich W, Levin ES, Lew W, et al: Intraprocedural and postprocedural perigraft arterial sac embolization (PASE) for endoleak treatment. *J Vasc Surg* 59:538–541, 2014.
68. Schanzer A, Greenberg RK, Hevelone N, et al: Predictors of abdominal aortic aneurysm sac enlargement after endovascular repair. *Circulation* 123:2848–2855, 2011.
69. Greenhalgh RM, Brown LC, Powell JT, et al: Endovascular versus open repair of abdominal aortic aneurysm. *N Engl J Med* 362:1863–1871, 2010.
70. Gurtler VM, Sommer WH, Meimarakis G, et al: A comparison between contrast-enhanced ultrasound imaging and multislice computed tomography in detecting and classifying endoleaks in the follow-up after endovascular aneurysm repair. *J Vasc Surg* 58:340–345, 2013.
71. Nagre SB, Taylor SM, Passman MA, et al: Evaluating outcomes of endoleak discrepancies between computed tomography scan and ultrasound imaging after endovascular abdominal aneurysm repair. *Ann Vasc Surg* 25:94–100, 2011.
72. Kouchoukos NT, Masetti P, Murphy SF: Hypothermic cardiopulmonary bypass and circulatory arrest in the management of extensive thoracic and thoracoabdominal aortic aneurysms. *Semin Thorac Cardiovasc Surg* 15:333–339, 2003.
73. Hiratzka LF, Bakris GL, Beckman JA, et al: 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with Thoracic Aortic Disease: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine. *Circulation* 121:e266–e369, 2010.
74. Greenbaum AB, O'Neill WW, Paone G, et al: Caval-aortic access to allow transcatheter aortic valve replacement in otherwise ineligible patients: Initial human experience. *J Am Coll Cardiol* 63:2795–2804, 2014.
75. Vandy FC, Girotti M, Williams DM, et al: Iliofemoral complications associated with thoracic endovascular aortic repair: Frequency, risk factors, and early and late outcomes. *J Thorac Cardiovasc Surg* 147:960–965, 2014.
76. Gutsche JT, Cheung AT, McGarvey ML, et al: Risk factors for perioperative stroke after thoracic endovascular aortic repair. *Ann Thorac Surg* 84:1195–1200, discussion 1200, 2007.
77. Bavaria JE, Appoo JJ, Makaroun MS, et al: Endovascular stent grafting versus open surgical repair of descending thoracic aortic aneurysms in low-risk patients: A multicenter comparative trial. *J Thorac Cardiovasc Surg* 133:369–377, 2007.
78. Adams JD, Tracci MC, Sabri S, et al: Real-world experience with type I endoleaks after endovascular repair of the thoracic aorta. *Am Surg* 76:599–605, 2010.
79. DeSart K, Scali ST, Feezor RJ, et al: Fate of patients with spinal cord ischemia complicating thoracic endovascular aortic repair. *J Vasc Surg* 58:635–642.e2, 2013.
80. Czerny M, Eggebrecht H, Sodeck G, et al: Mechanisms of symptomatic spinal cord ischemia after TEVAR: Insights from the European Registry of Endovascular Aortic Repair Complications (EuREC). *J Endovasc Ther* 19:37–43, 2012.
81. Duwayri Y, Jim J, Sanchez L: Alternative techniques to abdominal debranching. *Vasc Dis Manage* 7:E210–E213, 2010.
82. Greenberg R, Eagleton M, Mastracci T: Branched endografts for thoracoabdominal aneurysms. *J Thorac Cardiovasc Surg* 140:S171–S178, 2010.
83. Donas KP, Lee JT, Lachat M, et al: Collected world experience about the performance of the snorkel/chimney endovascular technique in the treatment of complex aortic pathologies: The PERICLES registry. *Ann Surg* 262:546–553, 2015.
84. Fox N, Schwartz D, Salazar JH, et al: Evaluation and management of blunt traumatic aortic injury: A practice management guideline from the Eastern Association for the Surgery of Trauma. *J Trauma Acute Care Surg* 78:136–146, 2015.
85. Murad MH, Rizvi AZ, Malgor R, et al: Comparative effectiveness of the treatments for thoracic aortic transection [corrected]. *J Vasc Surg* 53:193–199.e1–21, 2011.
86. Dormandy JA, Rutherford RB: Management of peripheral arterial disease (PAD). TASC Working Group. TransAtlantic Inter-Society Consensus (TASC). *J Vasc Surg* 31:S1–S296, 2000.
87. Norgren L, Hiatt WR, Dormandy JA, et al: Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC II). *J Vasc Surg* 45(Suppl S):S5–S67, 2007.
88. Jongkind V, Akkersdijk GJ, Yeung KK, et al: A systematic review of endovascular treatment of extensive aortoiliac occlusive disease. *J Vasc Surg* 52:1376–1383, 2010.
89. Ichihashi S, Higashira W, Itoh H, et al: Long-term outcomes for systematic primary stent placement in complex iliac artery occlusive disease classified according to Trans-Atlantic Inter-Society Consensus (TASC)-II. *J Vasc Surg* 53:992–999, 2011.
90. Ye W, Liu CW, Ricco JB, et al: Early and late outcomes of percutaneous treatment of TransAtlantic Inter-Society Consensus class C and D aorto-iliac lesions. *J Vasc Surg* 53:1728–1737, 2011.
91. Kashyap VS, Pavkov ML, Bena JF, et al: The management of severe aortoiliac occlusive disease: Endovascular therapy rivals open reconstruction. *J Vasc Surg* 48:1451–1457, 1457.e1–3, 2008.
92. Upchurch GR, Dimick JB, Wainess RM, et al: Diffusion of new technology in health care: The case of aorto-iliac occlusive disease. *Surgery* 136:812–818, 2004.

93. Chiu KW, Davies RS, Nightingale PG, et al: Review of direct anatomical open surgical management of atherosclerotic aorto-iliac occlusive disease. *Eur J Vasc Endovasc Surg* 39:460–471, 2010.
94. de Vries SO, Hunink MG: Results of aortic bifurcation grafts for aortoiliac occlusive disease: A meta-analysis. *J Vasc Surg* 26:558–569, 1997.
95. Hertzler NR, Bena JF, Karafa MT: A personal experience with direct reconstruction and extra-anatomic bypass for aortoiliofemoral occlusive disease. *J Vasc Surg* 45:527–535, discussion 535, 2007.
96. Ricco JB, Probst H: Long-term results of a multicenter randomized study on direct versus crossover bypass for unilateral iliac artery occlusive disease. *J Vasc Surg* 47:45–53, discussion 53–54, 2008.
97. Reed AB, Conte MS, Donaldson MC, et al: The impact of patient age and aortic size on the results of aortobifemoral bypass grafting. *J Vasc Surg* 37:1219–1225, 2003.
98. Klonaris C, Katsarypis A, Tsekouras N, et al: Primary stenting for aortic lesions: From single stenoses to total aortoiliac occlusions. *J Vasc Surg* 47:310–317, 2008.
99. Indes JE, Mandawat A, Tuggle CT, et al: Endovascular procedures for aorto-iliac occlusive disease are associated with superior short-term clinical and economic outcomes compared with open surgery in the inpatient population. *J Vasc Surg* 52:1173–1179, 1179.e1, 2010.
100. Cau J, Ricco JB, Corpataux JM: Laparoscopic aortic surgery: Techniques and results. *J Vasc Surg* 48:37S–44S, discussion 45S, 2008.
101. Cherry KJ: Complications following reconstructions of the pararenal aorta and its branches. In Towne JB, Hollier LH, editors: *Complications in vascular surgery*, ed 2, New York, 2004, Marcel Dekker, pp 275–287.
102. Gruppo M, Mazzalai F, Lorenzetti R, et al: Midline abdominal wall incisional hernia after aortic reconstructive surgery: A prospective study. *Surgery* 151:882–888, 2012.
103. Sachdev U, Baril DT, Morrissey NJ, et al: Endovascular repair of para-anastomotic aortic aneurysms. *J Vasc Surg* 46:636–641, 2007.
104. Reilly LM, Stoney RJ, Goldstone J, et al: Improved management of aortic graft infection: The influence of operation sequence and staging. *J Vasc Surg* 5:421–431, 1987.
105. Oderich GS, Bower TC, Cherry KJ, Jr, et al: Evolution from axillofemoral to in situ prosthetic reconstruction for the treatment of aortic graft infections at a single center. *J Vasc Surg* 43:1166–1174, 2006.
106. Brown KE, Heyer K, Rodriguez H, et al: Arterial reconstruction with cryopreserved human allografts in the setting of infection: A single-center experience with midterm follow-up. *J Vasc Surg* 49:660–666, 2009.
107. Noel AA, Gloviczki P, Cherry KJ, Jr, et al: Abdominal aortic reconstruction in infected fields: Early results of the United States cryopreserved aortic allograft registry. *J Vasc Surg* 35:847–852, 2002.
108. Batt M, Magne JL, Alric P, et al: In situ revascularization with silver-coated polyester grafts to treat aortic infection: Early and midterm results. *J Vasc Surg* 38:983–989, 2003.
109. Clagett GP, Bowers BL, Lopez-Viego MA, et al: Creation of a neo-aortoiliac system from lower extremity deep and superficial veins. *Ann Surg* 218:239–248, discussion 248–249, 1993.
110. Lonn L, Dias N, Veith Schroeder T, et al: Is EVAR the treatment of choice for aortoenteric fistula? *J Cardiovasc Surg (Torino)* 51:319–327, 2010.
111. Cherry KJ, Dake MD: Aortic dissection. In Hallett JW, Mills JL, Earnshaw JJ, et al, editors: *Comprehensive vascular and endovascular surgery*, ed 2, Philadelphia, 2009, Mosby, pp 517–531.
112. Swee W, Dake MD: Endovascular management of thoracic dissections. *Circulation* 117:1460–1473, 2008.
113. Estrera AL, Miller CC, 3rd, Safi HJ, et al: Outcomes of medical management of acute type B aortic dissection. *Circulation* 114:I384–I389, 2006.
114. Panneton JM, Teh SH, Cherry KJ, Jr, et al: Aortic fenestration for acute or chronic aortic dissection: An uncommon but effective procedure. *J Vasc Surg* 32:711–721, 2000.
115. Trimarchi S, Nienaber CA, Rampoldi V, et al: Role and results of surgery in acute type B aortic dissection: Insights from the International Registry of Acute Aortic Dissection (IRAD). *Circulation* 114:I357–I364, 2006.
116. Tian DH, De Silva RP, Wang T, et al: Open surgical repair for chronic type B aortic dissection: A systematic review. *Ann Cardiorhorac Surg* 3:340–350, 2014.
117. Kouchoukos NT, Kulik A, Castner CF: Open thoracoabdominal aortic repair for chronic type B dissection. *J Thorac Cardiovasc Surg* 149:S125–S129, 2015.
118. Dake MD, Kato N, Mitchell RS, et al: Endovascular stent-graft placement for the treatment of acute aortic dissection. *N Engl J Med* 340:1546–1552, 1999.
119. Durham CA, Cambria RP, Wang LJ, et al: The natural history of medically managed acute type B aortic dissection. *J Vasc Surg* 61:1192–1198, 2015.
120. Nienaber CA, Kische S, Rousseau H, et al: Endovascular repair of type B aortic dissection: Long-term results of the randomized investigation of stent grafts in aortic dissection trial. *Circ Cardiovasc Interv* 6:407–416, 2013.
121. Hofferberth SC, Newcomb AE, Yii MY, et al: Combined proximal stent grafting plus distal bare metal stenting for management of aortic dissection: Superior to standard endovascular repair? *J Thorac Cardiovasc Surg* 144:956–962, discussion 962, 2012.
122. Kitagawa A, Greenberg RK, Eagleton MJ, et al: Fenestrated and branched endovascular aortic repair for chronic type B aortic dissection with thoracoabdominal aneurysms. *J Vasc Surg* 58:625–634, 2013.