Atherosclerosis is a systemic disease that afflicts millions of patients annually in the United States. Historically, most of the clinical focus has been on its coronary artery manifestations, given their frequency and the potentially grave consequences. While vascular medicine and vascular surgical specialists, however, have long recognized that peripheral (extracardiac) arterial occlusive disease may contribute significantly to morbidity and mortality, it is only recently that invasive and interventional cardiologists have become involved in its diagnosis and management. To support that involvement, the scope of this chapter has been extended beyond the usual aortic diseases (such as dissection) to include information on atherosclerosis as it affects other major arterial territories. It reviews the natural history, clinical presentation, noninvasive diagnostic modalities, and angiographic techniques that are of value in patients with peripheral vascular disease, including aneurysmal disease of the thoracic and abdominal aorta, and atherosclerotic disease of the extracranial carotid arteries, renal arteries, and lower-extremity arteries. Additional information regarding interventional techniques is reviewed in Chapter 27, and representative case profiles are reviewed in Chapter 35.

PERIPHERAL IMAGING TECHNIQUES

Aortography and peripheral angiography have a history as long as that of cardiac catheterization. As W. Forssmann was reporting the passage of a catheter from his own arm vein into his right atrium in 1929 (1), dos Santos and colleagues described their experience in performing abdominal aortography by direct needle puncture (2). Seven years later, Nuvoli performed aortography via direct needle puncture of the ascending aorta (3). Fortunately, these direct-access techniques have now been virtually replaced by the same percutaneous (see Chapter 4) and cutdown (see Chapter 5) techniques for catheter introduction that are used for left heart catheterization, albeit with different catheters and filming techniques. Beyond these catheter-based imaging techniques, many disorders of the aorta are now diagnosed using highly refined noninvasive techniques (echocardiography, computed tomography [CT], and magnetic resonance angiography [MRA] techniques) that have the potential to provide detailed two- and even three-dimensional images.

RADIOGRAPHIC IMAGING

Radiographic invasive vascular imaging examinations have achieved a new level of complexity and sophistication over the past decade. Although many of the technical aspects are the same as those previously described for cardiac catheterization (see Chapter 2), some of the requirements for examination of the aorta and peripheral vessels are different. They are summarized in American Heart Association (AHA) task force guidelines relating to the optimal resources for the examination and endovascular treatment of peripheral and visceral vascular systems (4). As in cardiac work, satisfactory imaging requires a radiographic gantry that is capable of angulation in both the axial and sagittal planes. It must also allow the operator to access a variety of potential catheter introduction sites, including the neck via jugular vein, arm (axillary, brachial, and radial arteries), and leg (for antegrade and retrograde femoral as well as more peripheral artery) entry sites. To capture the larger regions of interest (e.g., the entire aortic arch, the pelvic vasculature, or both legs, a larger field (14-inch or 36-cm) image intensifier is optimal.

Conventionally, image recording of peripheral studies was done using film-screen radiographic techniques and mechanical rapid cut-film changers. This has now been replaced by digital angiography, which allows immediate monitor display of the acquired image, as well as electronic processing to enhance contrast, reduce noise, and subtract
Normal abdominal aortogram utilizing iodinated contrast material obtained by digital imaging technique. A preliminary image is recorded immediately prior to contrast injection, so that any background density (bone, calcifications, soft tissue, and air densities) can be subtracted from subsequent images, which then show only the contrast-filled vessels of interest (Fig. 14.1A, B). Further postprocessing features may include reversal, magnification, pixel-shifting, picture integration, and contour enhancement of the subtracted image. Quantitative analysis (QA) may be used to assess vessel diameters and lengths, degree of luminal narrowing, and blood flow velocity. The resulting electronic images may be stored digitally and may also be transmitted to other sites for simultaneous review. Although it is not used in cardiac work (where cardiac motion precludes acquiring a suitable “mask” image), DSA is of great value in peripheral work where it can reduce the volume of contrast required for an examination (improving patient comfort, reducing the risk of local vascular and systemic contrast toxicity), shorten the time needed to perform the procedure, reduce radiation exposure, and lower film cost (because only the selected best images are captured to film).

**FIG. 14.1.**

A: Normal abdominal aortogram utilizing iodinated contrast material obtained by digital imaging technique. B: Same imaging data as for (A); however, enhancement of contrast-filled vessels obtained by the subtraction of all background densities (bones, soft tissue, gas) as recorded on a “mask,” immediately prior to contrast injection.

**CATHETERS AND GUIDEWIRES**

Just as there is a wide range of cardiac catheters and guidewires, vascular angiography and intervention have a wide range of tools to meet different anatomic challenges. In addition to standard, thin-walled 18-gauge needles that will accommodate an 0.038-inch wire, micropuncture (i.e., 21-gauge) needle sets are available that allow conversion to a standard (i.e., 0.035-inch) guidewire in situations with a high risk of bleeding, if anticipating unsuccessful needle punctures or thrombolytic therapy.

**FIG. 14.2.**

Peripheral angiographic catheters. **Left to right:** Pigtail, cobra, multipurpose, headhunter, Simmons, SOS-OMNI, tennis-racquet.

Most peripheral guidewires are made of a stainless steel coil surrounding a tapered inner core that runs the length of the wire for additional strength. A central safety wire filament is incorporated to prevent separation should the wire coil ever fracture. Standard wires vary in diameter from 0.012 to 0.052 inch, with 0.035 and 0.038 being the most commonly used sizes. The length of most standard wires is between 100 and 180 cm; longer exchange-length guidewires (measuring 260 to 300 cm) permit keeping the tip of the wire in a selected position during catheter exchange. Tip configurations include straight or angled tip and J shape. Special features may include the ability to move the wire’s inner core to vary the length of the floppy tip, deflect the wire tip, or transmit torque from the shaft to the tip so that it can be steered within the vascular tree. Varying degrees of shaft stiffness (e.g., extrastiff support wires) allow advancement of stiff devices through tortuous vessels, and low-friction wires with a hydrophilic coating (glidewires) have revolutionized peripheral work and made it possible to perform superselective catheterization, and traverse complex stenoses and long occlusions.

Peripheral angiographic catheters are constructed of polyurethane, polyethylene, Teflon, or nylon, with wire braid to impart torqueability. An ideal catheter has good memory, nonthrombogenicity, sufficient torque control to facilitate rotational positions, ability to accommodate high-pressure injection, and ready trackability, frequently aided by hydrophilic polymer coating. Catheters vary in French size, length, and hole pattern. They may have either a single end-hole for selective injections, both end- and side-holes, or a blocked end with side-holes only. For catheters designed to be positioned in the abdominal aorta, 60- to 80-cm lengths are sufficient; in the thoracic or carotid areas, 100- to 120-cm lengths (similar to those of left heart catheters) may be required. The most common diagnostic catheter sizes are 5F to 7F, although 3F and 4F systems have gained popularity when brachial and radial arteries are used for access.
Several catheter shapes have been designed, which ultimately determines a specific function (Fig. 14.2). They fall into these general families:

- Straight catheters with multiple side ports that are used for rapid injection into large vessels, and for exchange.
- Pigtail or tennis-racquet catheters that are used for nonselective angiography in large vessels (i.e., aorta, pulmonary artery, or cardiac chambers). Multiple side-holes along the distal shaft allow rapid delivery of contrast without a single forceful jet that could cause catheter whipping or subintimal dissection, as might be seen with contrast exiting the end-hole alone.
- Simple curved catheters (e.g., Berenstein, Cobra [Merit Medical, South Jordan, UT], Headhunter), that are used for vessel selection.
- Complex reverse-curve catheters (e.g., Simmons [Cook Medical, Bloomington IN], Sidewinder [Cordis, a Johnson & Johnson Co., Warren, NJ], SOS-OMNI [Angio Dynamics, Queensbury, NY]), that are used for selective catheterization of certain aortic branches.

**CONTRAST AGENTS**

Because high-osmolar contrast materials (e.g., iothalamate, diatrizote) produce general side-effects (such as nausea, vomiting, light-headedness) as well as intense local pain, during peripheral injection, patient tolerance is improved by the use of low-osmolar agents (5). Low-osmolar agents also deliver a lesser osmotic load, thereby reducing any intravascular volume augmentation, which could be hazardous in patients with congestive heart failure or renal dysfunction. They are a necessity when there is a possibility of filling carotid, vertebral, or spinal artery branches and should be used for pulmonary arteriography to avoid pulmonary vasoconstriction.

**FIG. 14.3.**

A: Pelvic arteriogram of 75-year-old female with bilateral hip claudication demonstrating diffuse infrarenal aortic atherosclerosis, right common iliac artery stenosis (black arrow) and left common iliac artery occlusion (white arrow) with external iliac artery reconstitution via collaterals. B: Corresponding MRA with two-dimensional gadolinium-enhanced technique that mirrors the DSA image.

In recent years, two new contrast agents have emerged as alternatives in patients with severe renal dysfunction or a history of life-threatening contrast allergy. Carbon dioxide (CO2) as a contrast agent has been utilized extensively in many vascular beds (6–8). Its primary advantage is that it obviates any risk of allergic reaction or nephrotoxicity (8–10). Its application is limited to arteries below the diaphragm, to minimize the risk of intracerebral embolization. To be used effectively, digital subtraction equipment is required. Another agent, gadolinium (gadopentetate dimeglumine), has been used traditionally with magnetic resonance imaging. Recently, however, it has also been used as a contrast agent during catheter-directed radiographic peripheral vascular imaging (11),(12). Like CO2, it is relatively nontoxic, although the maximal dose is limited to 0.4 mmol/kg (approximately 60 mL). It lower K-edge absorbance energy may also require some adjustment to radiographic technique (lower kilovolts) compared with iodine-based imaging (Fig. 14.3A,B).

**VASCULAR ACCESS**

Deciding on the optimal puncture site in the patient with peripheral vascular disease is analogous to the surgeon's planning of an incision. The goals are to facilitate the procedure, reduce the likelihood of complications, and shorten the duration of the procedure. The optimal site of access may be determined by the physical examination, complemented with data obtained by noninvasive studies (e.g., duplex ultrasonography), to avoid entry into heavily diseased or occluded vessels. The most common sites remain the common femoral and brachial arteries. If the femoral pulse is diminished or absent (e.g., due to occlusion more proximally), one of several methods may be employed to facilitate successful entry of the artery (13),(14). Fluoroscopic landmarks include the facts that the...
Antegrade femoral artery puncture. The skin nick at the top of the femoral head (needle), with ideal entry at the middle of the common femoral artery with angle less than 45°. Ultrasound guidance, and road mapping of a contrast injection performed via a catheter positioned in the distal aorta from the contralateral groin may be helpful.

One technique unique to peripheral angiography—antegrade puncture of the common femoral artery (CFA)—is required for many infragenual procedures. As in retrograde access, the desired site of entry is in the middle of the CFA below the inguinal ligament; therefore the skin puncture is made at or above the top of the femoral head (not the bottom of the femoral head as during retrograde puncture) (15) (Fig. 14.4). A less acute needle angle, generally less than 45°, should be maintained to facilitate catheter and sheath insertion by avoiding the kinking that may occur with a steeper-angled entry. Single-wall puncture of the artery (rather than the classic through-and-through Seldinger approach) may reduce bleeding complications. Great care should be exercised in advancing and manipulating catheters and guidewires in the severely diseased peripheral circulation, to reduce the chance of embolization related to the traumatic disruption of cholesterol-rich atherosclerotic plaque. This rare but devastating complication of arteriography may lead to livedo reticularis, hypertension, renal failure, gangrene, stroke, or death (see Chapter 3). Although there are no proven therapies effective in the management of this dreadful complication, prostaglandins (e.g., PGE1, PGI2) may serve a palliative role in those cases in which it occurs (16) (17).

FIG. 14.4.

Antegrade femoral artery puncture. The skin nick at the top of the femoral head (needle), with ideal entry at the middle of the common femoral artery with angle less than 45°.

Beyond these general peripheral imaging techniques, there are a number of important considerations relating to each portion of the arterial tree. In this and subsequent chapters relating to the peripheral circulation (Chapters 27 and 35), we will review the territories in a head-to-foot sequence.

THORACIC AORTA

Anatomy

The aortic valve is composed of three leaflets that form the three sinuses of Valsalva: right, left, and posterior (18). The ascending aorta itself begins just beyond the sinus segment and courses in a mostly anterior to posterior direction. The diameter of the ascending aorta varies between 2.2 cm and 3.8 cm in middle-aged adults, and increases slightly with advancing age (19). After it passes over the main pulmonary artery and left mainstem bronchus, the aorta gives rise to the brachiocephalic trunk and then courses posteriorly and leftward in front of the trachea. It then gives rise to the remaining arch vessels—the left common carotid, and left subclavian arteries—from its upper surface (Fig. 14.5A).

FIG. 14.5.

A: Normal ascending and arch aortogram with great vessels. B: Ascending aortic aneurysm due to cystic medial degeneration. C: Stanford type A aortic dissection following aortic valve replacement. The intimal dissection flap (arrows) separates the contrast-filled true lumen (TL) from the false lumen (FL) that compromises the TL as it proceeds distally. D: The dissection extending into the abdominal aorta with origination of the left renal artery from the FL and TL supplying the right renal artery.

Distal to the origin of the left subclavian artery, the aorta narrows slightly at the site of the isthmus where the ligamentum arteriosum (the remnant of the fetal ductus arteriosus) tethers the aorta to the left pulmonary artery. Just
distally to this point, a fusiform dilatation, called the aortic spindle, may occur. The descending aorta then continues anterior to the spine, with a diameter of approximately 2.5 cm. Vessels deriving from the descending portion of the aorta are nine pairs of posterior intercostal arteries (levels T-3 to T-11). The first and second posterior intercostal arteries are supplied by the superior intercostal artery, which is a branch of the subclavian artery. At the level of the fourth to sixth thoracic vertebrae, anteriorly directed bronchial arteries arise to supply each lung.

Disorders of the Thoracic Aorta

Aortic Coarctation

Coarctation of the aorta occurs in 0.02% to 0.06% of the population and may be associated with bicuspid aortic valve (33% of cases), patent ductus arteriosus, ventricular septal defect (VSD), or Turner's syndrome. To bypass the resulting bandlike narrowing of the aorta, collateral flow occurs retrograde into the posterior intercostal branches of the descending aorta. The resultant enlargement and tortuosity of these intercostal arteries are responsible for the “rib notching” seen on chest roentgenograms.

Findings by aortography or MRI are a severe, discrete narrowing of the aorta at the isthmus, dilatation of the ascending aorta, and enlarged internal thoracic and intercostal arteries. Aortography assumes a significant role in differentiating the great variety of abnormal patterns, including complete aortic interruption, hypoplastic aorta, and the most common type—a stenosis at the site of the isthmus, distal to the origin of the left subclavian artery. Both anteroposterior (AP) and lateral (right anterior oblique [RAO] to left anterior oblique [LAO]) aortography should be initially undertaken, with contrast injection performed proximal to the presumed site of coarctation using either large-film or cineangiographic technique. When attempting to traverse the site of narrowing in retrograde fashion, care must be taken to avoid inadvertent perforation of the thin-walled poststenotic segment. Entrance to the prestenotic aorta from the brachial or axillary arteries may thus be preferred.

Patent Ductus Arteriosus

The prevalence of patent ductus arteriosus is one in 5,500 children less than 14 years of age. Selective aortic angiography is sensitive in demonstrating small shunts and surpasses the sensitivity of right heart catheterization with stepwise oximetry (see also Chapters 6, 28, and 34).

Aortic Aneurysms

Thoracic aortic aneurysms (TAAs) and pseudoaneurysms may have various etiologies. These include those that are related to degeneration or atherosclerosis, trauma, infection (syphilitic, bacterial), cystic medial degeneration, connective-tissue disorders, vasculitis, chronic dissection, and congenital (aneurysms of the Valsalva sinus) causes. Degenerative aneurysms involving the descending aorta account for about 75% of TAAs. Cystic medial degeneration (as seen in Marfan's syndrome, see below) may also produce aneurysms of the ascending aorta (Fig. 14.5B). Aneurysms caused by blunt or penetrating trauma often involve the proximal descending thoracic aorta, where the mobile arch segment joins the descending segment that is fixed to the spine (26–28). They often represent pseudoaneurysms-contained ruptures that are lacking intimal and medial components and are contained only by adventitia and periaortic tissue.

The natural history of TAAs is poorly understood as compared with the extensive data available on untreated infrarenal abdominal aortic aneurysms. Many patients with thoracic aortic aneurysms are asymptomatic at the time of diagnosis, with the aneurysm incidentally detected during testing for an unrelated disorder. Thoracic aneurysms appear to enlarge at a more rapid rate than abdominal aneurysms (0.42 versus 0.28 cm/year), and aneurysms larger than 5 to 6 cm in diameter enlarge even faster and have a greater likelihood of rupture (29–31). The cumulative 5-year risk of the enlargement of the aorta, and are usually related to impingement on adjacent structures. In addition to presenting with catastrophic rupture, patients with TAA may report dyspnea, hoarseness, dysphagia, stridor, and plethora with edema from superior vena cava (SVC) compression. Neck or jaw pain may also be present in patients with aneurysms of the aortic arch. Dilatation of the aortic valve annulus and aortic valve may produce aortic regurgitation and congestive heart failure. Aneurysms of the descending thoracic aorta may produce...
pleuritic left-sided or interscapular pain, and thoracoabdominal aortic aneurysms may induce complaints of abdominal pain and left shoulder discomfort from irritation of the left hemidiaphragm.

The primary treatment for TAAs is surgical repair when the diameter exceeds 5 to 6 cm or symptoms develop (32). The standard procedure is to use a Dacron graft to replace the diseased segment. In most patients undergoing elective thoracic aorta surgical repair, aortography is required to provide information about the location of the aneurysm and its relationship to major aortic branches in the chest and abdomen. Optimal surgical approaches, as well as operative risks, are best defined by imaging the coronary, brachiocephalic, visceral, and renal arteries during injections. Stent-graft devices have been successfully employed as an alternative to surgical grafting for both thoracic and aortic degenerative and posttraumatic descending TAAs (33–35). Early experience has been limited by incomplete aneurysm thrombosis, graft leak and failure. However, further refinements in the technology may make this modality a viable option in poor surgical candidates.

**Aortic Dissection**

Aortic dissection is a longitudinal cleavage of the aortic media by a dissecting column of blood (36). An intimal tear allows the passage of blood into the aortic wall, separating the inner and outer layers of the aortic wall and creating a “double-barrel lumen” (37). Men are affected about twice as frequently as women (38). Most patients are between 50 and 70 years of age, and have arterial hypertension (39). Other risk factors include cystic medial degeneration (40), Marfan's syndrome (41), bicuspid aortic valve (39), aortic coarctation, blunt trauma (39), pregnancy (42), (43), connective-tissue disorders (41), and thoracic aorta operative procedures (44). The dissection may extend proximally from its origin to the aortic annulus, or distally to involve the entire length of the aorta and any or all of its major branches, until terminated by an aortic branch or atherosclerotic plaque. Two classification systems of aortic dissection are widely used. The DeBakey classification is based on the anatomic extent of the dissection (45), (46). In type I, the tear originates in the ascending aorta and extends distally. Type II dissections are confined to the ascending aorta. In type III, the dissection may be confined to the descending aorta (type IIIa) or extend into the abdominal aorta and iliac arteries (type IIIb). The Stanford classification is based solely on the location of the origin of the dissection (47). Type A includes all cases where the ascending aorta is involved, and type B includes those where the ascending aorta is not involved (Fig. 14.5C,D).

Dissection is usually heralded by the sudden onset of excruciating pain described as “tearing, throbbing, lacerating, ripping, or burning” in the anterior chest, neck, or interscapular region (48). Similar pain may occur with rupture or sudden expansion of a chronic dissection. If the acute dissection results in compression of aortic branches, symptoms and signs of acute myocardial infarction (49), stroke or transient ischemic attack, paraparesis (50), mesenteric ischemia, renal failure (51), paraplegia, and extremity ischemia (52) may result. The majority of patients with ascending aortic extension who are treated medically die within 3 months, usually from dissection into the pericardium, mediastinum, or pleural cavity.

Once considered the “gold standard” for diagnosis of aortic dissection, aortography (which has a sensitivity of 80% and specificity of about 95%) has largely been replaced by CT, MRA, and tranesphageal echocardiography (TEE) (53). Intimal flap visualization is the only direct aortographic sign that is pathognomonic of dissection. This is frequently in association with delayed or sluggish filling of a second lumen, although about 20% of patients with aortic dissection have only one visible aortic channel. The presence of a false lumen may still be suspected, however, if that single channel shows evidence of extrinsic compression by a hematoma in the false lumen. Beyond documenting the dissection, aortography provides information about aortic insufficiency and branch vessel or coronary artery involvement, particularly in cases where CT or MRI findings are equivocal and there is a strong clinical suspicion of aortic dissection (54).

When approaching a patient with suspected aortic dissection, the preferred entry point is the femoral artery with the best pulse. An atraumatic diagnostic catheter (e.g., pigtail or tennis racquet) with a soft J-tipped guidewire should be advanced under fluoroscopic guidance with frequent test injections. Since the entry to the false lumen is commonly on the greater (outer) curve of the aorta, the catheter may be used to direct the wire toward the inner curve to maximize the chance of remaining in the true lumen. If this is done successfully, structures like the aortic leaflets and coronary arteries will be observed, and it will be possible to enter the left ventricle. It is not uncommon, however, to enter the false lumen during initial catheter advancement. When this becomes apparent on test injections, care should
be taken to avoid extending the false lumen, pulling the catheter back and using the techniques discussed above to reenter the true lumen.

Surgical repair of Stanford type A aortic dissections entails Dacron graft replacement of the ascending aorta (55). If the aortic valve is abnormal, it is replaced (56). In contrast, most patients with type B acute aortic dissections can be initially treated with medical therapy, reserving surgical intervention for those with signs of impending rupture (persistent pain and hypotension), ischemia of legs or mesentery, renal failure, paraparesis, or paraplegia (57). In cases of chronic dissection, operative treatment should be considered if the diameter of the descending aorta exceeds 5 to 6 cm or symptoms develop. Endovascular stents and balloon fenestration have been successfully used in treating the ischemic complications associated with aortic dissection (58–60).

**Vasculitides**

Vasculitis, highlighted by inflammation of the vessel wall, has two forms which commonly affect the aorta and its branches. These produce dilation of the proximal aorta, narrowing or occlusion of large aortic branches, or both. Takayasu's arteritis is characterized by irregularity of the ascending aorta, narrowing of the descending aorta, obstructions of arch vessels and aortic insufficiency or dissection (61–63). Therapeutic options include surgical bypass or balloon angioplasty demonstrating adjunctive stenting in patients with end-organ ischemia (64),(65). Intervention should generally be reserved until acute inflammation has subsided.

Giant cell or temporal arteritis is a vasculitis of large and medium-sized arteries, and is likely a variant of Takayasu's arteritis. Angiographic evidence of aortic branch involvement shows long, smooth stenoses alternating with relatively normal segments. The intracranial carotid artery and its branches, or the distal subclavian arteries, are usually involved, with aortic disease relatively uncommon (66).

**Connective-Tissue Disorders**

Several inherited diseases—including Marfan's syndrome, Ehlers-Danlos syndrome, and hereditary annuloaortic ectasia—may be responsible for noninflammatory degeneration of the aortic wall. These may lead to aneurysm formation, rupture, or dissection.

Marfan's syndrome is a rare autosomal dominant disorder that may affect the aorta, heart, eye, and skeleton. Cardiovascular complications occur in greater than 50% of patients (67),(68). Cystic medial degeneration accounts for the resultant changes in aortic root dilation with aortic ectasia, aortic insufficiency, aneurysm formation, or dissection (69). In Marfan's syndrome the aortic dilatation is primarily confined to the aortic root. Asymptomatic aortic dissection may be seen in approximately 10% of patients. Treatment for patients with Marfan's syndrome and cystic medial degenerative disease should include elective replacement of the ascending aorta and the aortic sinuses when the greatest diameter of the aorta is 5.0 to 5.5 cm (70). The most commonly performed procedure is replacement of the ascending aorta and the aortic valve with a composite graft containing Dacron and a mechanical valve prosthesis. The coronary arteries are reimplanted in the Dacron graft (71).

Ehlers-Danlos syndrome is a rare set of genetic disorders of collagen production. The literature describes more than nine types of this syndrome with features of hyperextensibility of joints and thick skin. Vascular complications include vessel thrombosis, rupture, or embolization from aneurysms (72).

**Thoracic Aortography**

Arch aortography has historically been used to examine the aorta for aortic valve or root disease; suspected aneurysms, dissections; congenital anomalies, such as vascular rings, coarctation, or patent ductus arteriosus; evaluation of vascular injuries associated with blunt or penetrating chest trauma; and examination of stenoses at the origins of the great vessels. TEE, CT, and MRA have made substantial inroads in the role traditionally reserved for arch aortography.

Thoracic aortography is usually performed from the femoral approach. In cases of suspected aortic dissection with
ABDOMINAL AORTA

Anatomy

The abdominal aorta starts at the level of the diaphragm (T-12) and proceeds anterior to the spine and to the left of the inferior vena cava until it bifurcates into the common iliac arteries at the level of the fourth lumbar vertebra (18) (Fig. 14.6A). The normal diameter of the midabdominal aorta varies between 1.50 cm and 2.15 cm, with a slight increase in size with age and male gender (73). Three main branches of the aorta originate from its ventral surface. The first is the celiac artery at the level of T-12 to L-1. The second branch is the superior mesenteric artery (SMA) which takes off about 1 cm caudal to the celiac axis, at the L-1 to L-2 level. The third is the inferior mesenteric artery (IMA), which originates at the L-3 to L-4 level and takes off in an anterolateral direction, slightly to the left. The renal arteries originate posterolaterally from the aorta at the level of L-1 to L-2 (just below the SMA). Four pairs of lumbar arteries arise in a posterolateral direction, below the main renal arteries.

Clinical Manifestations of Abdominal Aortic Disease

In patients with abdominal aortic aneurysms (AAA), the goals of preoperative imaging are detection, staging, surveillance, and diagnosis of rupture (74),(75). Important information in planning a management strategy includes the size and length of the AAA, proximal and distal margins, number, location and patency of renal and mesenteric arteries, presence of lower-extremity occlusive disease, and any associated aneurysmal disease (e.g., iliac, hypogastric, femoral, or other intraabdominal vessels) (Fig. 14.6B). The role of abdominal aortography in the preoperative assessment of patients with AAA has diminished with the advent of CT, MRI, and sonography. Preoperative angiography may be useful in the cases of suspected suprarenal or juxtarenal aortic aneurysm involvement, renal or mesenteric artery stenosis, horseshoe kidney, and iliofemoral occlusive disease.

Atherosclerotic occlusive disease (ASO), or asteriosclerois obliterans, may warrant arteriographic examination of the aorta. ASO may result in complete occlusion of the aorta (76) (Fig. 14.6C). The etiology usually is a chronic thrombotic occlusion superimposed on severe atherosclerosis of the distal aorta and iliac arteries. Leriche syndrome is a chronic aortic occlusion that consists of buttock and thigh claudication, impotence, and the absence of femoral pulses (77). Congenital coarctation syndromes, which include Williams’ syndrome (78), neurofibromatosis (79), congenital rubella (80), and tuberous sclerosis (81), may also involve the abdominal aorta and its branches. Aortography reveals a smooth, tapered proximal and midabdominal aorta with proximal renal artery involvement, and narrowing of the superior mesenteric or celiac arteries. Middle aortic syndrome (abdominal aortic coarctation) produces stenoses of the midaorta and its associated major branches (82). Treatment options include surgical bypass or percutaneous transluminal angioplasty with endovascular stenting in certain cases, although experience is limited, and the exact role of the latter is controversial (83).
Abdominal Aortography Technique

Abdominal aortography is performed from a femoral approach, utilizing a 4F or 5F multiple-side-hole pigtail or tennis-racquet diagnostic catheter. If the femoral pulse is not palpable on either side, other options include translumbar, axillary, brachial, or radial access. The tip of the catheter should be positioned at the T-12 or L-1 level, thus placing the side-holes adjacent to the first and second lumbar vertebrae. Contrast medium should be injected (30 to 60 mL at a rate of 15 to 30 mL/sec). At least three frames per second should be obtained when evaluating the mesenteric or renal arteries. Two views of the aorta-anteroposterior and lateral—generally provide sufficient information regarding the aorta and mesenteric vessels. When performing arteriography in an aorta with suspected or known aneurysmal disease or severe atherosclerotic involvement, meticulous care should be taken to avoid dislodging mural thrombus or plaque, potentially liberating distal atheroemboli.

SUBCLAVIAN AND VERTEBRAL ARTERIES

Anatomy

The brachiocephalic, left common carotid, and left subclavian arteries arise from the aortic arch after it passes over the main pulmonary artery and left main stem bronchus (18). While the right subclavian artery and right common carotid originate as branches of the brachiocephalic trunk (also known as the “innominate” artery), the left common carotid and left subclavian usually originate separately from the aortic arch. An aortic arch variant in which the brachiocephalic and left common carotid artery may have a common origin (i.e., “bovine arch”) is present in about 10% of the population (84). The major branches of the subclavian artery that deserve special attention are the internal mammary and vertebral arteries; the latter originate from the superior aspect of the vessel (opposite the internal mammary) and proceed into the skull through the cervical transverse processes.

Manifestations of Subclavian Disease

Atherosclerosis of the proximal subclavian artery may manifest clinically as arm claudication, subclavian-steal syndrome (85), or (in patients with previous internal mammary grafting) coronary ischemia (86). In classic subclavian steal, stenosis or occlusion of the proximal subclavian artery causes blood from the contralateral vertebral artery to flow antegrade across the basilar system and then retrograde down the ipsilateral vertebral to fill the subclavian artery distal to the lesion (Fig. 14.7A). In rare cases, this may cause cerebral ischemia during upper-extremity exercise. In patients who have undergone internal mammary artery bypass grafting to a coronary artery, a proximal subclavian obstruction may cause retrograde flow in the graft during arm exercise and lead to coronary ischemia (coronary-subclavian-steal) (Fig. 14.7B,C). Stenosis of the vertebral origin is relatively common, particularly at its origin from the subclavian artery; however, cerebral symptoms are unusual, given the dual blood supply (from both vertebrales and the carotid arteries by way of the posterior communicating artery) unless both vertebrales are diseased.

FIG. 14.7.

A: Selective left subclavian arteriogram depicting severe ostial stenosis (arrow) and retrograde flow through left vertebral artery (white). B: Arteriogram of subclavian artery in a patient after a coronary artery bypass graft with a left internal mammary artery (LIMA) graft and high-grade ostial stenosis (double arrow) resulting in poor visualization of the graft (arrow). C: Following successful stenting of the subclavian artery stenosis and restoration of antegrade flow into the LIMA graft

Subclavian and Vertebral Arteriography
An aortic arch arteriogram with a 5F pigtail catheter can visualize the origin of the great vessels to evaluate for atherosclerotic occlusive disease. (See the section on the thoracic arteriogram.) To catheterize the left subclavian artery selectively, a guidewire usually can be advanced directly from the descending thoracic aorta (see Chapter 11). If a proximal vertebral artery stenosis is expected, selective injection of the ipsilateral subclavian artery in the anteroposterior projection is usually diagnostic. Modest angulation may be necessary.

CAROTID ARTERIES

Anatomy

The brachiocephalic artery bifurcates into the right subclavian and right common carotid arteries as the first main branch off the aorta. The left common carotid is typically the second main branch of the aorta. Each common carotid runs within a fascial (carotid) sheath, lateral to the vertebrae, and bifurcates into an external and internal carotid artery branch at the fourth cervical vertebrae (18). While the internal carotid artery normally has no main branches prior to entering the skull, it forms a tortuous portion known as the carotid siphon within the cavernous and supraclinoid segment and thereafter divides into the anterior and posterior cerebral arteries. The external carotid artery has several major branches named for their territory of supply.

Extracranial Carotid Atherosclerosis

Approximately 700,000 strokes occur annually in the United States. It is estimated that 25% to 30% of these events are due to extracranial carotid artery disease. In the Minneapolis–St. Paul, Minnesota, metropolitan area, in 1985 there were 1,792 hospital discharges with the diagnosis of acute stroke, representing an event rate of 828/100,000 population in men and 551/100,000 in women (87). Patients with carotid disease frequently have severe coronary artery disease. In a population of 506 patients undergoing evaluation for potential carotid revascularization, 16% of patients without clinical clues suggestive of coronary heart disease were found to have severe, surgically correctable coronary artery disease (88). Even patients with asymptomatic carotid artery stenosis have an increased risk of coronary events. In one study of 444 male patients, the 4-year mortality rate was 37%, with 61% of the deaths due to coronary artery disease. Multivariate analysis shows diabetes mellitus, an abnormal electrocardiogram, and the presence of intermittent claudication to be associated with an increased mortality risk. (Two or three risk factors revealed annual mortality rates of 11.3% and 13%, respectively.) Just the finding of increased carotid intima-media thickness on duplex ultrasonographic examination predicts a higher risk of myocardial infarction or stroke, as much as 3.87 times that compared with patients with minimal thickness (89).

The majority of patients with extracranial carotid artery disease, often identified by the discovery of a carotid bruit on physical examination, have no referable symptoms. Estimates of the prevalence of asymptomatic carotid bruits in adults range from 1% (90) to 2.3% in patients age 45 to 54 years and 8.2% in patients more than 75 years of age (91). Among patients scheduled to undergo other vascular surgical procedures, however, the incidence of cervical bruits ranged from 6% (92) to 16% (93), with a mean prevalence of 10% (94). An asymptomatic carotid bruit carries a 1.5% annual incidence of stroke and a 3-year stroke risk of 2.1% (as demonstrated by the European Carotid Surgery Trialists). Among patients with an asymptomatic bruit and severe (70% to 99%) carotid stenosis, the 3-year risk of stroke was 5.7% (95). Absence of a bruit, however, does not imply absence of significant carotid disease. In a substudy of the North American Symptomatic Carotid Endarterectomy Trial (NASCET), 1,268 patients with recent transient cerebral ischemia or nondisabling stroke were examined for the presence of a carotid bruit. Fifty-eight percent of patients had a bruit localized to the ipsilateral carotid artery; 31% had a carotid bruit involving the contralateral vessel; and 24% had bilateral carotid bruits. The sensitivity and specificity of a focal bruit to predict high-grade ipsilateral carotid stenosis was 63% and 61%, respectively. In this patient subgroup, absence of a bruit lowered the pretest probability of a 70% to 99% carotid stenosis only from 52% to 40% (96).

Once established, extracranial carotid artery stenosis progresses in approximately 20% to 40% of cases. In one prospective natural history study of 232 patients with mild (<50%) and moderate (50% to 79%) carotid stenosis followed with annual carotid duplex ultrasonography for a mean of 7 years, 23% demonstrated disease progression. One-half of these patients developed severe stenosis (80% to 99%) or occlusion. Progression to either 80% to 99% stenosis or occlusion was more likely in patients whose initial stenosis was 50% to 79% rather than less than 50%.
More recent data in 425 asymptomatic patients with 50% to 79% carotid stenosis followed for a mean of 38 months demonstrated progression of stenosis in 17% of 282 arteries with at least two serial carotid duplex examinations. In general, this carried a moderately low incidence of ipsilateral stroke (0.85% at 1 year; 3.6% at 3 years; 5.4% at 5 years) (98), but patients with 80% to 99% carotid stenosis had an annual neurologic event rate of 20.6% (99).

Many carotid lesions are discovered only after the patient begins to experience symptoms, which may vary from transient monocular blindness (amaurosis fugax) to expressive or receptive aphasia, hemiparesis/hemiplegia, and mental status changes. These episodic symptoms that last minutes to hours and then completely resolve are harbingers of recurrent and potentially nonreversible events, and thus warrant urgent evaluation and therapy in an attempt to prevent a catastrophic stroke. The first study in this evaluation is carotid duplex ultrasonography, which provides two-dimensional images of the extracranial carotid arteries and may provide information about plaque morphology (Fig. 14.8A). Color flow can detect increased velocities of blood flow, which correlate to greater degrees of stenosis, while Doppler waveforms and velocities can also be measured to evaluate stenosis severity when performed by skilled vascular ultrasonographers (100) (Fig. 14.8B). Once a significant stenosis is identified, contrast or MRA can be performed to corroborate the ultrasound findings (101) (Fig. 14.8C). Conversely, if the ultrasound is performed by a reliable vascular laboratory, many surgeons proceed with endarterectomy based on this diagnostic test alone (102). Stent-assisted carotid angioplasty is now being offered at some centers as an alternative to surgery, particularly in patients at high risk for surgical correction (see Chapters 27 and 35) (Fig. 14.8D).

A: Color duplex image of severely narrowed right ICA. B: Corresponding spectral waveform of ICA showing accelerated peak systolic and end-diastolic velocities. C: Carotid arteriogram confirming severe stenosis involving the right ICA. D: Arteriogram of carotid bifurcation following successful stenting of stenotic right ICA.

**Carotid Arteriography**

Carotid arteriography remains the gold standard in assessing the presence and quantitative narrowing of the carotid and intracerebral vasculature. Despite the advances made with noninvasive techniques such as duplex ultrasonography, MRA, and spiral computed tomoangiography (CTA), selective carotid catheterization may be indicated to more accurately delineate the degree of stenosis involving the distal common and internal carotid arteries and the extent of disease at the bifurcation, as well as to provide information about the intracranial circulation, including collateral flow patterns. The carotid artery may be selectively catheterized by a number of 5F (simple) catheters. Tortuous proximal great vessels, however, may require a complex-curve catheter.

Once the catheter is beyond the aortic arch, careful double-flushing is mandatory to minimize risk of embolization. Injections of low-osmolar contrast injections are typically performed at a maximum rate of 8 mL/sec for 10 cc total in the CCA, 8 mL/sec for 8 cc total in the internal carotid artery (ICA), and 7 mL/sec for a total of 7 cc in the vertebral artery. Film rates of two to four frames per second should be used during the arterial phase and slower rates should be used for the venous phase. Multiple oblique projections are necessary, including anteroposterior, lateral, and oblique views to visualize narrowing at the carotid bifurcation and proximal ICA optimally. The lateral projection is best to visualize the proximal ICA and carotid siphon. The Towne and lateral views are best for visualizing the intracerebral anatomy. The Towne view is centered like an AP skull radiograph, with slight angulation so that the petrous ridge lies over the roof of the orbit.

To calculate the percentage diameter stenosis, the projection that demonstrates the highest degree of stenosis should be used. Many different methods of calculating carotid artery stenosis have been employed in previous trials; however, the North American Symptomatic Carotid Endarterectomy Trial (NASCET) methodology is the most widely accepted. It compares the stenotic area with the most normal-appearing artery distal to the stenosis.
Anatomy

The renal arteries arise from the lateral aspect of the aorta at the L-1 to L-2 level (18). Accessory renal arteries may occur in 25% to 35% of cases and usually supply the lower pole of the kidney. These may originate anywhere from the suprarenal aorta down to the iliac arteries.

Atherosclerotic Renal Artery Stenosis

Atherosclerotic renal artery stenosis (ARAS) is clearly more common than previously believed, with increasing prevalence in certain patient populations. In one series of 395 arteriograms performed in patients with abdominal aortic aneurysms, aortoiliac or infrainguinal atherosclerosis, 33% to 50% had renal artery stenosis of more than 50% (103). In 346 patients with aneurysmal or occlusive vascular disease prompting arteriography, 28% had significant ARAS. The presence of coronary artery atherosclerosis is also a marker for ARAS. In a prospective study of 1,302 patients undergoing coronary arteriography, concurrent abdominal aortography demonstrated significant ARAS in 15% of patients. The number of coronary arteries involved with atherosclerosis also appears to predict the likelihood of renal artery stenosis in this series. For example, if one coronary artery demonstrated atherosclerosis, the incidence of significant ARAS was 10.7%. If three coronary arteries and the left main trunk are involved with atherosclerosis, the incidence of ARAS was 39% (104). Conversely, 58% of patients with ARAS had clinically overt coronary artery disease.

A number of clinical clues may suggest the presence of ARAS. Patients who develop diastolic hypertension after 55 years of age who have exacerbation of previously well-controlled hypertension, who demonstrate refractory hypertension (uncontrolled hypertension despite treatment with three antihypertensive medications of synergistic classes at maximal doses), who develop azotemia after treatment with an angiotensin converting enzyme inhibitor, or who present with malignant hypertension (severe hypertension and acute myocardial infarction, acute stroke or transient ischemic attack, aortic dissection, acute renal failure) should be suspected of having renal artery stenosis. A discrepancy in renal size, the physical finding of a systolic and diastolic abdominal bruit with radiation to one or both flank regions, unexplained azotemia, or the presence of diffuse atherosclerosis with hypertension and azotemia without obvious cause must prompt the physician to search for renal artery disease. Up to 24% of patients with end-stage renal disease (ESRD) being considered for dialysis in one series had severe ARAS (105). The 15-year survival of patients committed to ESRD because of ARAS was 0, compared with 32% in patients committed to dialysis for other causes such as polycystic kidney disease.

The natural history of ARAS has been studied extensively in many retrospective series, which suggest that approximately 50% of renal arteries progress over time (106). More recent prospective data utilizing duplex ultrasonography to assess renal artery patency demonstrated that 48% of renal arteries whose baseline stenosis was less than 60% progressed to more than 60% stenosis after 36 months, compared with only 8% in vessels with no stenosis at baseline (107).

A number of noninvasive diagnostic tests have been used to determine if renal artery stenosis is present. Historically, rapid sequence intravenous pyelography was used; this has now been shown to be inaccurate. Equally inaccurate are plasma renin levels, only elevated in 50% to 80% of patients with RAS (108). Captopril stimulated nuclear renography is a prominent diagnostic test for patients with suspected renal artery stenosis, with sensitivity and specificity in the range of 90% (109). However, in a recent comparison for the diagnosis of renal artery stenosis, the isotopic renal scan was no better than the clinical prediction rule to predict renal artery stenosis, particularly in the presence of bilateral renal artery stenosis or impaired renal function.

Renal artery duplex ultrasonography can be an excellent test to diagnose renal artery stenosis if performed by a skilled operator. In one prospective series of 29 patients (58 renal arteries) who underwent contrast arteriography and duplex ultrasonography, sensitivity of the latter was 84%, specificity was 97%, and positive predictive value was 94% for detection of more than 60% stenosis (110). Utilizing criteria of peak systolic velocity within the renal artery of more than 180 cm/sec, duplex scanning was able to discern between normal and diseased renal arteries with a sensitivity of 95% and specificity of 90% (111). The ratio of peak systolic velocity (PSV) in the area of renal artery stenosis compared with the PSV within the aorta (renal to aortic ratio [RAR]) of more than 3.5 predicts the presence
of more than 60% renal artery stenosis with a sensitivity of 92%. In another large prospective series of 102
consecutive patients who underwent both duplex ultrasonography and contrast arteriography within 1 month of each
other, 62 of 63 arteries with less than 60% stenosis, 31 of 32 arteries with 60% to 79% stenosis, and 67 of 69 arteries
with 80% to 99% stenosis were correctly identified by duplex ultrasonography. Occluded renal arteries were
correctly identified in 22 of 23 cases. The overall sensitivity of duplex ultrasonography was 98%; specificity, 99%;
positive predictive value, 99%; and negative predictive value, 97% (112). Limitations of direct ultrasound
visualization of the renal arteries include large body habitus and overlying bowel gas obscuring identification of the
renal arteries. Some authors have suggested that renal hilar scanning is easier and as accurate as complete
interrogation of the renal arteries (113). However, direct comparison of both techniques has revealed limitations of
hilar scanning, including low sensitivity, inability to discriminate between stenosis and occlusion, and inadequate
determination of accessory renal arteries. The sensitivity was 67% for hilar scanning, with a specificity of 89% to
99% (114). Given that many patients have both main renal artery disease and intraparenchymal disease, the addition
of resistive indices within the parenchyma may help predict which patients will benefit from revascularization (115).
Duplex ultrasonography is an excellent method for determining patency following revascularization (116). Given the
proliferation of endovascular therapy (percutaneous angioplasty with stent deployment) (117), duplex
ultrasonography is helpful in detecting restenosis.

Magnetic resonance arteriography has demonstrated great promise as a highly accurate noninvasive test for the
diagnosis of renal artery stenosis (118). Limitations of this technology, predominantly overestimating degrees of
stenosis, are decreasing with the addition of intravenous gadolinium, a nonnephrotoxic contrast agent (119), and
perhaps, captopril (120).

Arteriography

Arteriography of the renal vasculature should begin with an aortogram to assess the degree of aortic disease, ostia of
the renal arteries, and presence of accessory renal arteries. Images are obtained with a 4F or 5F pigtail or tennis-
racquet catheter, with the side-holes positioned at the L-1 to L-2 level (Fig. 14.9A). Anteroposterior and oblique
(RAO and LAO) views, often with cranial or caudal angulation, may be necessary to delineate the ostium and
proximal renal arteries that are most commonly involved in atherosclerosis (Fig. 14.9B). Selective renal
arteriography may be indicated in the case of ostial disease, accessory renal arteries not visualized by the initial
arteriogram, suspected intrarenal vascular disease (e.g., fibromuscular dysplasia, Takayasu's arteritis, radiation,
anerysms, vasculitis), or need to measure pressure gradients across lesions of equivocal hemodynamic significance
(Fig. 14.9C). The renal arteries can be engaged selectively with 4F or 5F (e.g., renal double curve, Cobra, SOS-
OMNI, hockey-stick, or internal mammary) catheters. Injection rates of contrast media should be 5 to 10 mL at a rate
of 5 mL/sec. Filming sequences should include the early arterial phase as well as the nephrographic phase, which
characterizes contrast in both the nephrons and capillaries. The venous phase occurs 5 to 10 seconds after the initial
injection and shows filling of the renal veins. If surgical revascularization (e.g., hepatorenal or splenorenal bypass) is
contemplated, a lateral view of the aorta should be obtained to delineate the origins of the celiac and superior
celiac arterial and evaluate them for the presence of inflow disease.

**FIG. 14.9.**

A: Abdominal aortogram demonstrating normal renal arteries. B: Atherosclerosis of the aorta resulting in bilateral
renal artery stenosis. C: Selective injection of the left renal artery depicting an apparent moderate degree of luminal
narrowing. D: An intraarterial pressure tracing obtained across the lesion demonstrates a peak systolic and mean
gradient of 23 mm Hg and 12 mm Hg, respectively, indicating a hemodynamic significant lesion.

For equivocal renal artery stenoses, measurement of a transstenotic gradient may be helpful—gradients greater than 10
mm Hg mean or 20 mm Hg systolic as measured using a 4F catheter are taken as significant (Fig. 14.9D). This can
be done by advancing these catheters into the renal artery over flexible- or tapered-tip (e.g., Wholey, Bentson) wires
to avoid trauma, spasm, embolization, or perforation of the distal renal branches. Even so, any guidewire
manipulation within the renal artery should be minimized, and the operator must be prepared to move immediately to
renal stenting (see Chapter 27) if measurement of the gradient causes any disruption within the renal artery stenosis).
PELVIC AND LOWER EXTREMITIES

Anatomy

The bifurcation of the abdominal aorta into the common iliac arteries (CIA) occurs at the level of L-4 to L-5 (18) (Fig. 14.10A). The common iliac arteries divide at the lumbosacral junction, with the internal iliac arteries (IIA) taking off medially and posteriorly, and the external iliac arteries (EIA) continuing anteriorly and laterally to the groin, where they exit the pelvis just posterior to the inguinal ligament. The inferior epigastric artery takes off medially at the junction of the EIA and common femoral artery. The deep iliac circumflex artery takes off laterally and superiorly.

FIG. 14.10.

Normal pelvic and lower-extremity arteriogram. A: The distal abdominal aorta bifurcating into the iliac arteries. B: The common femoral artery (CFA) dividing into the deep femoral (DFA) and superficial femoral arteries (SFA). C: The SFA traversing the thigh into the popliteal artery as it dives through the adductor (Hunter's) canal. D: The popliteal artery dividing laterally into the anterior tibial (AT) artery and continues directly into the tibioperoneal trunk (TPT), which bifurcates into the posterior tibial (PT) and peroneal arteries (PER). E: The dorsalis pedis (DP) artery originates from the AT artery beyond the ankle and PT artery, which gives off plantar branches. (CIA, common iliac arteries; IIA, internal iliac arteries; EIA, external iliac arteries.)

The common femoral artery (CFA) is an extension of the EIA, which originates at the inguinal ligament and then bifurcates (usually at the lower portion of the femoral head) into the superficial femoral artery (SFA) anteromedially and the deep femoral artery (DFA), or “profunda,” posterolaterally (Fig. 14.10B). The DFA has two major branches, the lateral circumflex and medial circumflex femoral arteries. The SFA proceeds down the anteromedial thigh and dives deep at adductor (Hunter's) canal, where it becomes the popliteal artery running posterior to the femur. Major popliteal branches include the sural and geniculate (superior, middle, and inferior) arteries around the knee (Fig. 14.10C).

Below the knee, at the border of the popliteus muscle, the popliteal artery divides, with the anterior tibial (AT) artery proceeding laterally and anterior to the tibia toward the foot. As it passes over the ankle onto the dorsum of the foot, it continues as the dorsalis pedis (DP) artery. After the take-off of the AT, the popliteal continues as the tibio-peroneal trunk (TPT), which subsequently bifurcates into the posterior tibial (PT) and peroneal (PER) arteries. The PT courses posteromedially in the calf, while the peroneal runs near the fibula between the AT and PT arteries. The peroneal artery then rejoins the PT above the ankle via its posterior division, and the AT via its anterior division (Fig. 14.10D). On the dorsum of the foot, the DP artery has lateral and medial tarsal branches. After the PT artery passes behind the medial malleus, it divides into medial and lateral plantar arteries. The lateral plantar and distal DP arteries join to form the planter arch (Fig. 14.10E).

Lower-Extremity Arterial Occlusive Disease

The prevalence of peripheral arterial occlusive disease (PAD) remains difficult to appreciate among the general population. Since a significant segment of the population with PAD has no symptoms of the disorder, this makes true prevalence rates even more difficult to ascertain. Patients with asymptomatic PAD are at minimal risk of developing critical limb ischemia that threatens limb survival, with the obvious exception of the patient who suffers acute limb ischemia from an embolic event or trauma. Instead, patients first develop intermittent claudication to some degree, before progressing to rest pain, a nonhealing ischemic ulcer, or gangrene.

The United States National Institutes of Health suggests that lower-extremity arterial occlusive disease causes more than 60,000 hospitalizations annually, each stay lasting an average of more than 11 days (121).
arteriosclerotic obliterans—namely, diminished pedal pulses and femoral bruises—occur with increasing frequency as the population ages. While intermittent claudication occurs more often in men at any age, physical examination findings of peripheral arterial disease occur with identical frequency in men and women (122). Several investigators have attempted to define the prevalence of PAD using noninvasive testing modalities and symptom questionnaires. One series of 613 men and women with a mean age of 66 years, utilizing segmental limb blood pressures, Doppler flow velocities, reactive hyperemia, and pulse reappearance times found an 11.7% incidence of large-vessel PAD (123). Although 11.7% of the population thus had evidence of PAD, only 2.2% of men and 1.7% of women had intermittent claudication. In this same population, however, 20.3% of men and 22.1% of women had abnormalities in the femoral or posterior tibial artery pulse examination.

The currently accepted methods of determining the presence of PAD include an historical review of patient symptoms and atherosclerotic risk factors, physical examination, and use of noninvasive vascular tests. A common simple test is the ankle-brachial index (ABI). This test compares the blood pressure obtained with a hand-held Doppler in the dorsalis pedis or posterior tibial artery (whichever is higher) to the blood pressure in the higher of the two brachial pressures. Generally, an ABI of more than 0.9 is considered normal, one of more than 0.5 to less than 0.9 reflects mild to moderate PAD, and an ABI of less than 0.5 suggests severe arterial occlusive disease.

It is widely accepted that the presence of PAD increases the likelihood of myocardial infarction, stroke, renovascular disease (124), and cardiovascular mortality. The 5-year survival of a patient with intermittent claudication is only 70%, with 75% of these deaths attributable to cardiovascular events (125). Many studies have confirmed the association between cardiovascular morbidity and mortality and an abnormal ABI (126–132). Some have suggested that there is a significant proportion of the population with asymptomatic PAD, and their risk of cardiovascular morbidity and mortality is similar to their symptomatic counterparts. However, it is assumed that because of their lack of symptoms, this risk may not be recognized until an event has occurred.

The risk factors for the development of PAD include hypertension, hypercholesterolemia, tobacco use, and diabetes mellitus. Tobacco use remains the most important modifiable risk factor for PAD. Hughson et al. found that 56% of patients with intermittent claudication were active users of cigarettes, and 24% were former smokers. In addition, active cigarette smoking causes more severe claudication pain and diminished peripheral circulation than is found among patients who do not smoke, leading to a reduction in the exercise capacity of patients with claudication (133). Finally, the risk of progression of PAD and atherosclerosis in other vascular beds is significantly greater in patients who continue to smoke than it is in those who stop smoking. In 343 patients with intermittent claudication, only 11% stopped smoking 1 year after the diagnosis. Ischemic rest pain developed in 16% of continued smokers after 7 years, whereas none of the former smokers suffered from rest pain. The incidence of myocardial infarction 10 years after the diagnosis of claudication was 11% in former smokers and 53% in active smokers. Ten-year overall survival rates were 82% in former smokers and 46% in active smokers (134).

Diabetes mellitus and PAD is an ominous combination. Although the prevalence of PAD is higher in the diabetic than in the nondiabetic population, it is the relatively rapid progression to ischemic rest pain and ulceration that portends a poor prognosis for the patients with diabetes. There is a two- to threefold increase in risk of intermittent claudication in diabetic patients when compared with the nondiabetic population (135). This holds true for both men and women. The severity of PAD is also greater in the diabetic population. In a study of 47 patients with diabetes mellitus, all of whom had intermittent claudication at baseline, in comparison with 224 patients with intermittent claudication but no diabetes, the incidence of ischemic rest pain and/or gangrene after 6 years of follow-up was 40% and 18%, respectively (136). The duration of diabetes and the type of diabetes therapy (i.e., diet, oral hypoglycemic agent, and insulin) did not play a role in the incidence or severity of PAD.

Independent predictors of progression of PAD in diabetic patients include a decreased postexercise ankle-brachial index, increased arm systolic blood pressure, and current smoking, demonstrating the additive effects of atherosclerotic risk factors on the natural history of PAD (137). Interestingly, among the risk factors for amputation in patients with diabetes mellitus, neuropathic symptoms and lack of outpatient diabetes education are of importance and must be viewed concomitantly with the location and severity of PAD (138). Unfortunately, there remains no definitive evidence that strict glycemic control can prevent macrovascular complications from diabetes mellitus (139). There are several other potential risk factors for peripheral arterial occlusive disease, including Lp(a) (140), hyperhomocysteinemia (141), fibrinogen (142), and C-reactive protein (143). The specific role of each of these factors in the prevention and therapy of peripheral arterial disease remains unclear.
The most common symptom described by patients with peripheral arterial disease is intermittent claudication. Although the description of the symptom may vary among patients from pain, to ache, to numbness and weakness, there are several distinct characteristics of intermittent claudication. The discomfort is usually brought on by walking and alleviated by rest. The discomfort generally involves muscle groups immediately distal to the arterial segments involved (i.e., superficial femoral artery stenosis causes calf discomfort). The onset of intermittent claudication is quite predictable and occurs at similar distances, providing that the speed, incline, and terrain have remained unchanged. Patients generally stop, stand, and wait for 1 to 5 minutes for relief prior to resumption of walking.

Progression to critical limb ischemia is manifest by ischemic pain at rest, generally in the arch of the foot or toes. This occurs with the patient lying supine and is relieved by hanging the foot over the bedside. Paradoxically, patients with ischemic rest pain may note improvement in their pain with walking. Patients may resort to sleeping in a reclining chair, to provide a dependent position to the foot. Ischemic ulcerations occur as a result of trauma to toes or areas where bony prominences are exposed. Even minimal trauma, such as an ill-fitting shoe, may result in ulceration. The presence of ischemic rest pain or ulceration warrants a prompt and aggressive strategy for revascularization.

Physical examination must include palpation of all pulses, including the superficial temporal and carotid arteries, the arteries of the upper extremities, and the arteries of the lower extremities. Auscultation for bruits in the region of the cervical carotid arteries, abdomen, flank, and inguinal regions should be routinely performed, and the phase of the cardiac cycle during which the bruit occurs should be noted. Attempts to palpate the abdominal aorta for aneurysmal dilatation should me made. Close inspection of the feet and toes should include a search for ischemic ulceration or tinea infection. Kissing ulcerations between the toes in the web spaces are often subtle and easily missed on examination.

Once the ankle-brachial index has been performed, providing objective evidence of the overall severity of PAD in a limb, more specific noninvasive information can be obtained in the vascular laboratory. The addition of segmental limb pressures can aid in localizing stenoses or occlusions. Limb pressure cuffs are placed on the thigh (some centers prefer high- and low-thigh cuffs), calf, ankle, transmetatarsal region of the foot, and digit. The ABI is calculated and then the pressure is sequentially inflated in each cuff to approximately 20 to 30 mm Hg above systolic pressure. Utilizing a continuous-wave Doppler probe placed at a pedal vessel, the pressure in the cuff is gradually released, and the pressure at each segment is measured. If a decrease in pressure between two consecutive levels of more than 30 mm Hg is identified, this suggests arterial occlusive disease of the artery proximal to the cuff. In addition, comparing the two limbs, a 20 to 30 mm Hg discrepancy from one limb to the other at the same cuff level also suggests a significant arterial stenosis or occlusion proximal to the cuff (144).

Pulse volume recordings (PVR) are plethysmographic tracings that detect the changes in the volume of blood flowing through a limb. Using similar equipment as described previously, the cuffs are inflated to 65 mm Hg, and a plethysmographic tracing is recorded at various levels (145). The normal PVR is similar to the normal arterial pulse wave tracing and consists of a rapid systolic upstroke and rapid downstroke, with a prominent dicrotic notch. With increasing severity of disease, the waveform becomes more attenuated, with a wide downslope and, ultimately, virtually absent waveforms. Ankle-brachial indices, segmental pressures, and pulse volume recordings are useful objective tests in patients with suspected lower-extremity arterial occlusive disease, in those with limb discomfort without an obvious cause, as a method of evaluating the success of an intervention, and as a method of follow-up. The tests are inexpensive, painless, reproducible, and relatively easy to perform. The equipment required to perform these examinations is significantly less expensive than modern color-flow duplex ultrasound units.

Native vessel arterial duplex ultrasonography is widely performed. This examination is generally accepted as a method of defining arterial stenoses or occlusions (Fig. 14.11A). The sensitivity of duplex ultrasonography to detect occlusions and stenoses has been reported to be 95% and 92%, with specificities of 99% and 97%, respectively (146). Limitations have included tandem stenoses (147), tibial vessel imaging (148), and difficulty imaging the inflow arteries (149). Using a 5.0- to 7.5-MHz transducer, imaging of the supra- and infrainguinal arteries is performed. The vessels are studied in the sagittal plane, and Doppler velocities are obtained using a 60° Doppler angle. Vessels are classified into one of five categories: normal, 1% to 19% stenosis, 20% to 49% stenosis, 50% to 99% stenosis, and occlusion. The categories are determined by alterations in the Doppler waveform and by increasing peak systolic velocities. For a stenosis to be classified as 50% to 99%, for example, the peak systolic velocity must increase by 100% in comparison with the normal segment of artery proximal to the stenosis (150) (Fig. 14.11B).
Severe stenosis of the left superficial femoral artery (SFA) as depicted by (A) color duplex scanning. B: Spectral waveform shows an increased peak systolic velocity of 300 cm/sec. C: Arteriogram confirming a severe SFA stenosis at the corresponding site. D: Nine-month follow-up arteriogram demonstrating a widely patent SFA at the site of revascularization with adjunctive pHVEGF165 to accelerate reendothelialization.

Arterial duplex ultrasonography has been used to guide the interventionist toward appropriate access to a lesion potentially amenable to endovascular therapy (151) (Fig. 14.11C). This technology has also been used after endovascular therapy to determine technical success (152) and durability of the procedure (153) (Fig. 14.11D). Unfortunately, it appears that duplex ultrasonography soon after balloon angioplasty may overestimate residual stenosis and may limit the use of this technology after endovascular therapy (154).

In patients who have undergone surgical bypass graft revascularization, particularly with saphenous vein, stenoses will develop in 21% to 33% of cases. Once the graft becomes thrombosed, secondary patency rates are dismal. If the stenosis is detected and repaired before graft thrombosis, however, an estimated 80% of grafts can be salvaged (155). A well-organized graft surveillance program is thus crucial to preserving patency of bypass grafts. In one series of 170 saphenous vein bypass grafts, 110 stenoses were detected over a 39-month period. In those grafts that underwent surgical revision once a stenosis was detected, the 4-year patency was 88%, whereas in those grafts that did not undergo revision despite the detection of a stenosis, the 4-year patency was 57% (156). The use of an intensive surveillance program has been less beneficial in prosthetic grafts (157).

The procedure for graft surveillance is performed in a manner similar to that used in native vessel arterial duplex ultrasonography. The inflow artery to the bypass graft is initially imaged using a 5.0- to 7.5-MHz transducer and a Doppler angle of 60°. Subsequently, the proximal anastomosis; proximal, middle, and distal graft; distal anastomosis; and outflow artery are interrogated. Peak systolic and end-diastolic velocities are obtained at each segment and compared with the segment of graft proximal to the area being studied. If the ratio of the peak systolic velocity within a stenotic segment relative to the normal segment proximal to the stenosis is more than 2, this suggests 50% to 75% diameter reduction. The addition of end-diastolic velocities of more than 100 cm/sec suggests more than 75% stenosis (158).

Vein bypass grafts should be studied within 7 days of formation and then in 1 month, followed by 3-month intervals for the first year. If the graft remains normal after year 1, follow-up surveillance should be done every 6 months thereafter. Ankle pressures and waveforms should be performed at the time of each surveillance study. The development of a stenosis during a surveillance examination should prompt consideration toward arteriography, either with contrast or with magnetic resonance (159).

MRA has been promoted as an excellent method of evaluating the anatomy of the lower-extremity arteries. Initially touted as a unique and effective method of identifying angiographically occult runoff arteries that would be suitable as targets for surgical revascularization (160), investigators have studied the utility of MRA as the sole imaging modality prior to surgical revascularization (161). Recent comparative trials of MRA and standard contrast arteriography have revealed high sensitivity and specificity (97.1% and 99.2%, respectively) for MRA in patients suspected of having PAD (162).

Given the impressive advances in the field of endovascular therapy for PAD with percutaneous transluminal angioplasty, stent deployment, atherectomy, and stent grafting for aneurysmal and occlusive disease, however, diagnostic arteriography continues to play an important role in the management of patients with PAD.

**Pelvic and Lower-Limb Arteriography**

Arteriography is still considered the gold standard for evaluation of patients with peripheral vascular disease (PVD).
With the advent of reliable noninvasive vascular laboratory testing, however, arteriography should be reserved only for patients in whom endovascular or surgical intervention is contemplated. Information obtained from the history, physical examination, and noninvasive testing should be able to provide the clinician with the ability to ascertain the level and distribution of obstructive vascular disease. Indications for arteriographic study of the upper and lower extremities include ischemia (either exertional or resting) due to atherosclerosis, embolus, thrombosis, and vasculitis. Other potential etiologies warranting arteriography are peripheral aneurysms, vascular tumors, trauma, extrinsic compression (e.g., popliteal artery entrapment syndrome, cystic adventitial disease, and vasculitis [Buerger's disease], collagen vascular disease, and radiation).

Pelvic arteriography is usually performed from a femoral approach using a pigtail or tennis-racquet side-hole catheter placed just above the aortic bifurcation (L-4 to L-5). Other options include translumbar, axillary, or brachial artery sites. Axillofemoral bypass grafts may be directly punctured as they pass over the ribs. No consensus has been reached as to which common femoral artery should be punctured, the one on the side of the more symptomatic or that on the less symptomatic leg. The advantages of accessing the less symptomatic leg are that groin complications would not interfere with surgical bypass procedures, there is less risk of iliac artery trauma (e.g., dissection or occlusion), and the possibility remains of performing an antegrade puncture of the affected leg.

When one of the iliac arteries is occluded, the catheter should be positioned just below the renal arteries to visualize the lumbar arteries that serve as important collaterals into the pelvis (Fig. 14.12). To adequately assess for iliac disease, anteroposterior (AP) and both oblique (25° to 30°) pelvic projections should be obtained, to image the iliac and femoral artery bifurcations. The RAO projection opens the left iliac and right femoral bifurcations, and the LAO projection opens the opposite bifurcations. When lower-extremity arteriography is performed, visualization of the arteries to both feet is necessary in planning definitive treatment, by either surgical or endovascular techniques. Images may be recorded on cut-film, long-leg changer or step table, digital bolus-chase method, or serial digital filming techniques. If cut-film step table technique is used, a single bolus of contrast material is injected from the catheter at the aortic bifurcation at 7 to 9 mL/sec for a volume of 70 to 120 mL and images are obtained along the course of the contrast from the aorta to the feet.

**FIG. 14.12.**

Pelvic arteriogram showing right iliac artery occlusion with common femoral artery (arrow) reconstitution via collaterals.

Digital imaging affords better resolution of images with a smaller diluted contrast load than with cut film. Serial digital filming is performed by obtaining stationary images over an arterial segment while contrast material is injected at a rate of 8 to 12 mL/sec for 2 to 3 seconds. The imager is positioned over each segment with similar injections and sequence at all respective levels, until pedal arteries are visualized. The digital bolus-chase method performs angiography by acquiring digital images series without contrast material and then acquires the image as it “chases” the contrast bolus. Advantages have been previously outlined in the text.

Atherosclerotic plaque may be eccentric, particularly in the aorta, iliac, or femoral bifurcation, where it tends to be located in the posterior wall of the deep femoral artery. In these cases, multiple oblique projections are necessary to uncover significant narrowings. Even so, angiography may underestimate the degree of luminal diameter narrowing, especially in tortuous iliac arteries, in particular the ostium of the common iliac and iliac bifurcation (163). Intraarterial pressure monitoring may thus be more accurate than multiple angiographic images in assessing the hemodynamic significance of a vascular lesion (164). There exists no consensus as to the threshold that defines a significant gradient. However, a resting peak systolic gradient of 5 mm Hg or an increase of greater than 10 mm Hg after augmentation with a vasodilator (e.g., nitroglycerin) is considered of hemodynamic significance (165). Intravascular ultrasound permits direct planimetry of luminal cross-sectional narrowing, obviating the multiple, oblique views required to unwind and/or eliminate overlap, which may obscure important luminal obstructions (166).

To optimize the visualization of the tibial or pedal arteries, selective catheter positioning into the superficial femoral
artery with the use of vasodilating agents, such as nitroglycerin (100 to 300 µg), papaverine (30 to 60 mg), or tolazoline (12.5 to 25 mg) may enhance digital images (167). When selection of the contralateral iliac artery is desired, a pigtail or tennis-racquet catheter, if previously placed in the distal aorta, may be used by gently unfolding it with a guidewire and engaging the aorta bifurcation. Once the guidewire is successfully advanced into the contralateral iliac artery, the catheter may be replaced with a straight catheter. Other options include Cobra, SOS-OMNI, hook, or internal mammary artery guide catheters, which are advanced over the aortic bifurcation. Reverse-curve catheters may facilitate engaging the internal iliac arteries from the ipsilateral side, although an easier access may be approached from an antegrade direction, if revascularization is a consideration.