Noninvasive Physiologic Vascular Studies: A Guide to Diagnosing Peripheral Arterial Disease[1]

Robert C. Sibley III, MD
Stephen P. Reis, MD
Jarrod J. MacFarlane, DO
Mark A. Reddick, MD, MS
Sanjeeva P. Kalva, MD
Patrick D. Sutphin, MD, PhD

Abbreviations: ABI = ankle-brachial index, DCA = diagnostic catheter angiography, PAD = peripheral arterial disease, PVR = pulse volume recording, TASC = Trans-Atlantic Inter-Society Consensus Document on Management of Peripheral Arterial Disease, TBI = toe-brachial index, 3D = three-dimensional

RadioGraphics 2017; 37:0000–0000

Published online 10.1148/rg.2017160044

Content Codes: IR US VA

[1]From the Division of Interventional Radiology, Department of Radiology, University of Texas Southwestern Medical Center, 5323 Harry Hines Blvd, Dallas, TX 75390-8834. Presented as an education exhibit at the 2015 RSNA Annual Meeting. Received March 6, 2016; revision requested May 16 and received June 27; accepted July 14. For this journal-based SA-CME activity, S.P.K. has provided disclosures (see end of article); all other authors, the editor, and the reviewers have disclosed no relevant relationships. **Address correspondence to** P.D.S. (e-mail: *Patrick.Sutphin@UTSouthwestern.edu*).

SA-CME LEARNING OBJECTIVES

After completing this journal-based SA-CME activity, participants will be able to:

- Describe diagnostic criteria for PAD using arterial pressures, Doppler waveforms, and PVRs.
- Identify the limitations of arterial pressures, Doppler waveforms, and PVRs.
- Localize the anatomic level of the lesion when given arterial pressures, Doppler waveforms, and PVRs.

See www.rsna.org/education/search/RG.

Noninvasive physiologic vascular studies play an important role in the diagnosis and characterization in peripheral arterial disease (PAD) of the lower extremity. These studies evaluate the physiologic parameters of blood flow through segmental arterial pressures, Doppler waveforms, and pulse volume recordings. Collectively, they comprise a powerful toolset for defining the functionality of the arterial system, localizing the site of disease, and providing prognostic data. This technology has been widely adopted by diverse medical specialty practitioners, including radiologists, surgeons, cardiologists, and primary care providers. The use of these studies increased substantially between 2000 and 2010. Although they do not employ imaging, they remain a critical component for a comprehensive radiologic vascular laboratory. A strong presence of radiology in the diagnosis of PAD adds value in that radiologists have shifted to noninvasive alternatives to diagnostic catheter angiography (DCA), such as computed tomography (CT) and magnetic resonance (MR) angiography, which provide a more efficient, less-expensive, and lower-risk alternative. Other specialties have increased the use of DCA during the same period. The authors provide a review of the relevant anatomy and physiology of PAD as well as the associated clinical implications. In addition, guidelines for interpreting the ankle-brachial index, segmental pressures, Doppler waveforms, and pulse volume recordings are reviewed as well as potential limitations of these studies. Noninvasive physiologic vascular studies are provided here for review with associated correlating angiographic, CT, and/or MR findings covering the segmental distribution of PAD as well as select nonatherosclerotic diagnoses.

Introduction

Atherosclerotic peripheral arterial disease (PAD) is the progressive stenosis, occlusion, or aneurysmal dilatation of the aorta and its noncoronary, nonintracranial branch vessels. In the setting of lower extremity PAD, the affected arteries include the distal aorta and the common iliac, internal iliac, external iliac, femoral, popliteal, and crural arteries. The compromised and progressive reduction in arterial blood flow may lead to claudication, rest pain in the leg or foot, tissue loss, nonhealing wounds or ulcers, infection, gangrene, and loss of limb. However, the implications of PAD extend beyond that of the lower extremity, with many PAD patients experiencing depression, reduced quality of life, and a significantly higher risk of cardiovascular events (1,2). The 5-year rate of nonfatal cardiovascular events, including myocardial infarction and stroke, for patients with symptomatic PAD is approximately 20%, and the 5-year mortality is 15%–30% (3).

TEACHING POINTS

- In PAD, the level of the lesion is grouped into three categories: aortoiliac, femoropopliteal, and crural (tibiopedal).
- An ABI less than 0.90 is diagnostic for PAD in patients with claudication or other signs of ischemia, with 95% sensitivity and 100% specificity.
- A proximal-to-distal decrease in sequential pressures greater than 20 mm Hg or a decrease in segmental-brachial index greater than 0.15 indicates occlusive disease and correlates with the level of the lesion.
- A normal lower extremity arterial Doppler velocity tracing is triphasic, with a sharp upstroke and peaked systolic component, an early diastolic component with reversal of flow, and a late diastolic component with forward flow. A biphasic signal is considered abnormal if there is a clear transition from triphasic signal along the vascular tree. Monophasic waveforms are always considered abnormal.
- Abnormal PVR findings include decreased amplitude, a flattened peak, and an absent dicrotic notch.

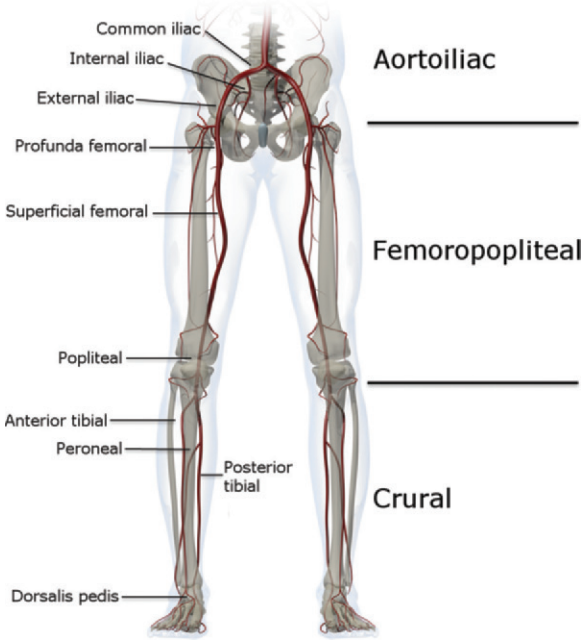


Figure 1. Diagram shows the vascular anatomy of the lower extremity, demonstrating the aortoiliac, femoropopliteal, and crural segments.

Approximately 8 million people in the United States have PAD (4). However, diagnosis and characterization of PAD by clinical factors alone remains a challenge. Patients may have a variable presentation: The authors of the Walking and Leg Circulation Study found that 48.3% of patients with an ankle-brachial index (ABI) less than 0.9 were asymptomatic or had atypical pain (5). With the silent progression of PAD, many have campaigned for screening for PAD. The Trans-Atlantic Inter-Society Consensus Document on Management of Peripheral Arterial Disease (TASC) II advocates for the screening of PAD with the ABI in all patients who have exertional leg symptoms, patients 50–69 years of age with cardiovascular risk factors, all patients greater than or equal to 70 years of age, or patients who have a Framingham Risk Score of 10%–20% (6). Additional evaluation may then be performed with such noninvasive physiologic vascular studies as segmental arterial pressures, pulse volume recordings (PVRs), and Doppler waveforms. The American College of Radiology Appropriateness Criteria state that these studies should be used in patients with symptoms and findings suggestive of PAD (7). Noninvasive physiologic vascular studies provide a more comprehensive evaluation compared with the ABI measurement and can determine the site and severity of disease (8).

There has been an increasing reliance on noninvasive physiologic vascular studies for the diagnosis of PAD. Analysis of Medicare Part B data demonstrated a sharp increase of 84% between 2000 and 2010 in their utilization rates. However, the rate of growth was not uniform among specialties. Utilization increased 180% among primary care physicians, 179% among cardiologists, 61% for radiologists, and 23% for surgeons (9). During the same period, with improved CT and MR angiography techniques, a divergence in the paradigm for the evaluation of PAD emerged between specialties. Review of Medicare Part B databases between 2002 and 2013 by Patel et al (10) revealed that MR and CT angiography nearly replaced diagnostic catheter angiography (DCA) in the diagnosis of PAD among radiologists, whereas the use of DCA rose sharply among cardiologists and surgeons despite available noninvasive alternatives. The use of radiology alongside noninvasive physiologic vascular studies in the diagnosis of PAD provides an opportunity to promote a shift to advanced noninvasive techniques such as CT and MR angiography, which are more efficient, less expensive, and carry a lower risk of complication than DCA.

In this article, the authors provide and discuss cases illustrating the anatomy and pathophysiology of PAD, the tools used in noninvasive physiologic vascular studies, the distribution of PAD, and findings of select nonatherosclerotic diagnoses encountered with these studies.

Anatomy and Pathophysiology of PAD

In PAD, the level of the lesion is grouped into three categories: aortoiliac, femoropopliteal, and crural (tibiopedal) (Fig 1). Aortoiliac disease includes the infrarenal segment of the abdominal aorta, common iliac arteries, internal iliac arter-

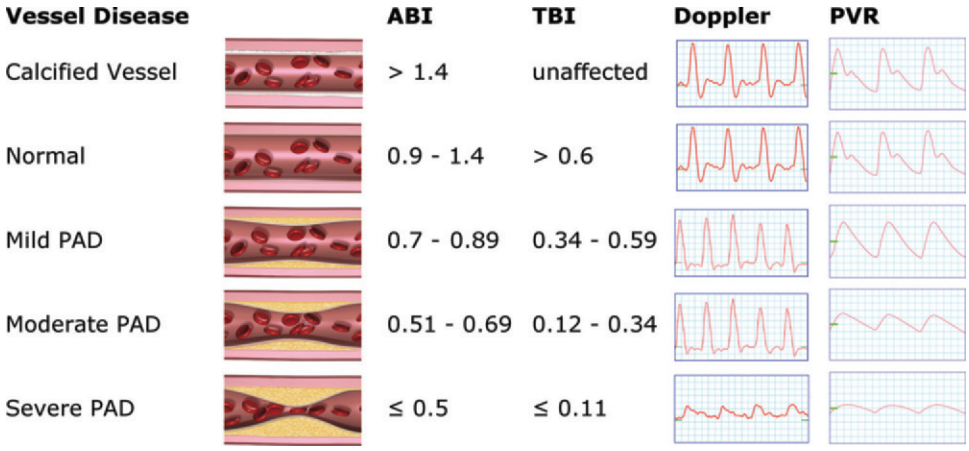


Figure 2. Graphic table shows guidelines for interpreting ABI, TBI, Doppler waveforms, and PVR waveforms in PAD.

ies, and external iliac arteries, proximal to the inguinal ligament or deep circumflex iliac artery. Femoropopliteal disease involves the common femoral arteries, profunda femoral arteries, and superficial femoral arteries, which continue to become the popliteal arteries as they enter the adductor canal and end at the origin of the anterior tibial arteries. Crural disease includes the anterior tibial, posterior tibial, peroneal, dorsalis pedis, and plantar arteries.

Blood flow limitation from areas of stenosis cause the signs and symptoms associated with PAD. Flow velocity and the degree of the stenosis determine whether a lesion is flow limiting (11). All other factors being equal, a stenosis decreasing vessel radius by 50% leads to a 16-fold reduction in flow. At rest, the flow velocity of the femoral artery is estimated to be as low as 20 cm/sec. For a stenosis to be hemodynamically important at this rate, a 90% decrease in luminal radius would be required. During exercise, the flow velocity of the femoral artery may increase up to 150 cm/sec. At this rate, a stenosis of only 50% is estimated to significantly impair arterial flow (8,11). Mild claudication is typically caused by single-segment disease with development of collateral circulation. Severe claudication and critical limb ischemia are associated with multilevel disease. The effects of a stenosis on blood flow allow various approaches to screen for PAD.

Tools for Diagnosing PAD

Arterial Pressure

Measuring systolic blood pressures at various points throughout the vascular tree provides useful information for diagnosing PAD. During a routine arterial pressure examination, pressures are measured at the arm, at the high thigh, above the knee, below the knee, at the ankle, and at the toe, bilaterally using Doppler signals to detect blood flow. Information derived from these pressures includes ABIs, toe-brachial indices (TBIs), segmental pressure differences, and postexercise comparisons (Fig 2).

In the primary care setting, the ABI is a quick and cost-effective examination (3) and should be used to screen patients meeting the TASC II criteria. To calculate the ABI, the pressure measured in the lower extremity is divided by the brachial pressure of the arm with the higher pressure. According to the 2011 American College of Cardiology Foundation (ACCF)/American Heart Association (AHA) guidelines, ABI results should be reported with noncompressible values defined as greater than 1.40, normal as 1.00 to 1.40, borderline as 0.91 to 0.99, and abnormal as 0.90 or less (12). An ABI less than 0.90 is diagnostic for PAD in patients with claudication or other signs of ischemia, with 95% sensitivity and 100% specificity (13). At our institution, in accordance with the Intersocietal Accreditation Commission Vascular Testing standards, an ABI of 0.70–0.89 is considered mild PAD, 0.51–0.69 moderate PAD, and less than or equal to 0.50 severe PAD. Mild-to-moderate PAD is typically associated with claudication (14). An ABI less than 0.50 has been associated with more severe coronary artery disease and increased mortality (15). Severe PAD is associated with multilevel disease, nonhealing ulcers, gangrene, and ischemic rest pain.

The vascular laboratory allows segmental pressures, segmental-brachial indexes, and TBIs to be measured. A proximal-to-distal decrease in sequential pressures greater than 20 mm Hg or a decrease in segmental-brachial index greater than 0.15 indicates occlusive disease and correlates with the level of the lesion (16). A difference of 30 mm Hg at the same level between left and right is also considered abnormal. A TBI less than 0.6 is considered abnormal, and a TBI less than 0.11 is associated with ischemic rest pain (17). Although an absolute toe pressure exceeding 30 mm Hg is required for normal wound healing, in

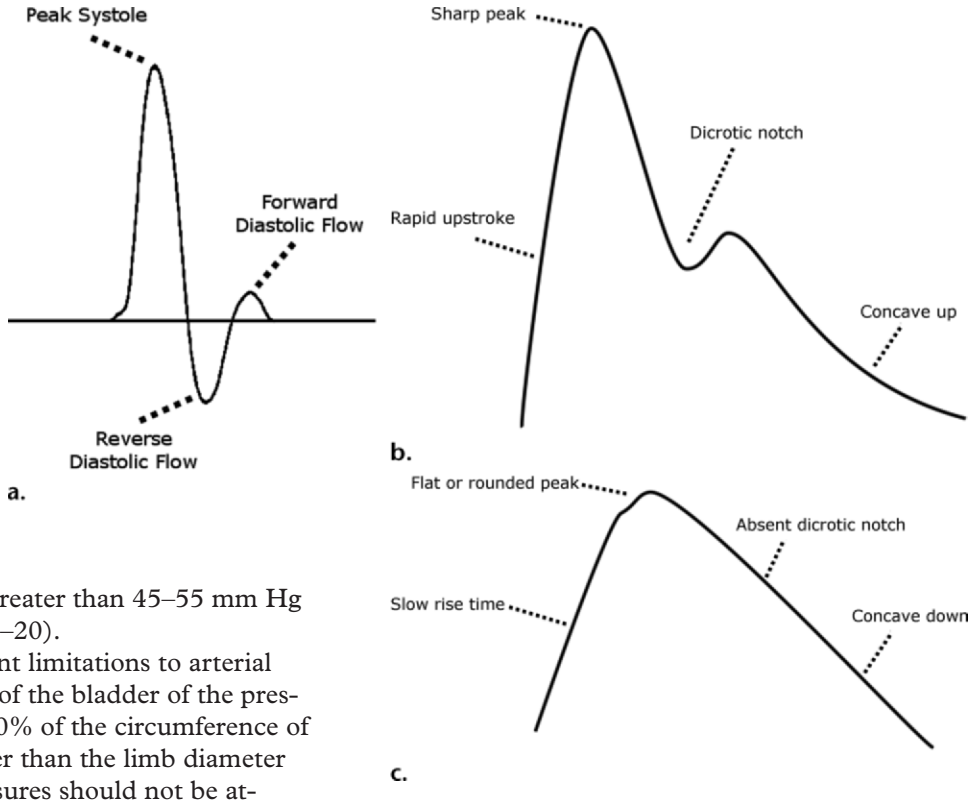


Figure 3. **(a)** Annotated triphasic Doppler waveform demonstrating peak systole, reverse diastolic flow, and forward diastolic flow. **(b)** Annotated normal PVR demonstrating a rapid upstroke, sharp peak, dicrotic notch, and concave-up distal waveform. **(c)** Abnormal PVR with a slow rise time, flattened (or rounded) peak, absent dicrotic notch, and concave-down distal waveform.

diabetics a pressure greater than 45–55 mm Hg may be necessary (18–20).

There are important limitations to arterial pressures. The width of the bladder of the pressure cuff should be 40% of the circumference of the limb or 20% wider than the limb diameter (21). Segmental pressures should not be attempted at the level of a previously placed stent or arterial bypass graft. Patients with limb ischemia can rarely tolerate blood pressure measurement in the affected limb. Finally, ABIs greater than 1.40 or pressures reported as noncompressible indicate arterial calcifications. The presence or absence of flow-limiting PAD cannot be determined in these cases.

Doppler Waveform

A continuous-wave Doppler velocity detector senses the Doppler shift of reflected sound waves bouncing off moving red blood cells. The B-mode component of duplex ultrasonography allows the correct angle placement of between 30° and 70°. A normal lower extremity arterial Doppler velocity tracing is triphasic, with a sharp upstroke and peaked systolic component, an early diastolic component with reversal of flow, and a late diastolic component with forward flow (Fig 3a). A biphasic signal is considered abnormal if there is a clear transition from triphasic signal along the vascular tree. Monophasic waveforms are always considered abnormal. Initially, as atherosclerosis develops, the elastic and muscular recoil of the vessel wall is lost, resulting in loss of forward flow during late diastole, creating a biphasic waveform. The loss of vascular resistance in severe PAD results in the loss of reversal of flow and in the monophasic waveform. In the absence of additional obstructions, it is possible for signals distal to an abnormal waveform to normalize. The deterioration of the waveform indicates the level of the lesion (Fig 2). The limitations of Doppler waveforms include technologist dependence, less accuracy in the aortoiliac segments secondary to obesity or bowel gas, and the time required to perform the study. Heat-induced vasodilatation leads to a decrease in the reversal of flow seen in early diastole of Doppler waveforms, and patients with uncompensated congestive heart failure demonstrate dampened waveforms following exercise (8).

Pulse Volume Recording

A PVR is a graph of the pulsatile change in limb volume from blood flow using constant standard pressure. Modern vascular laboratories acquire these tracings using the same pressure cuffs used for segmental limb pressure measurement. Normal PVRs consist of a rapid upstroke with a sharp peak, a dicrotic notch, and a concave-up late diastolic component. Abnormal PVR findings include decreased amplitude, a flattened peak, and an absent dicrotic notch (22,23) (Figs 2, 3b, 3c). An amplitude of less than 5 mm from trough to peak has been used as a criterion for diagnosing vascular claudication (8). Abrupt changes in amplitude and contour indicate occlusion between the two levels. Cardiac output, vasomotor tone, patient movement, and aortic stenosis influence PVRs, making lateral and sequential comparison imperative for interpretation. Heat-induced vasodilatation leads to loss of the dicrotic

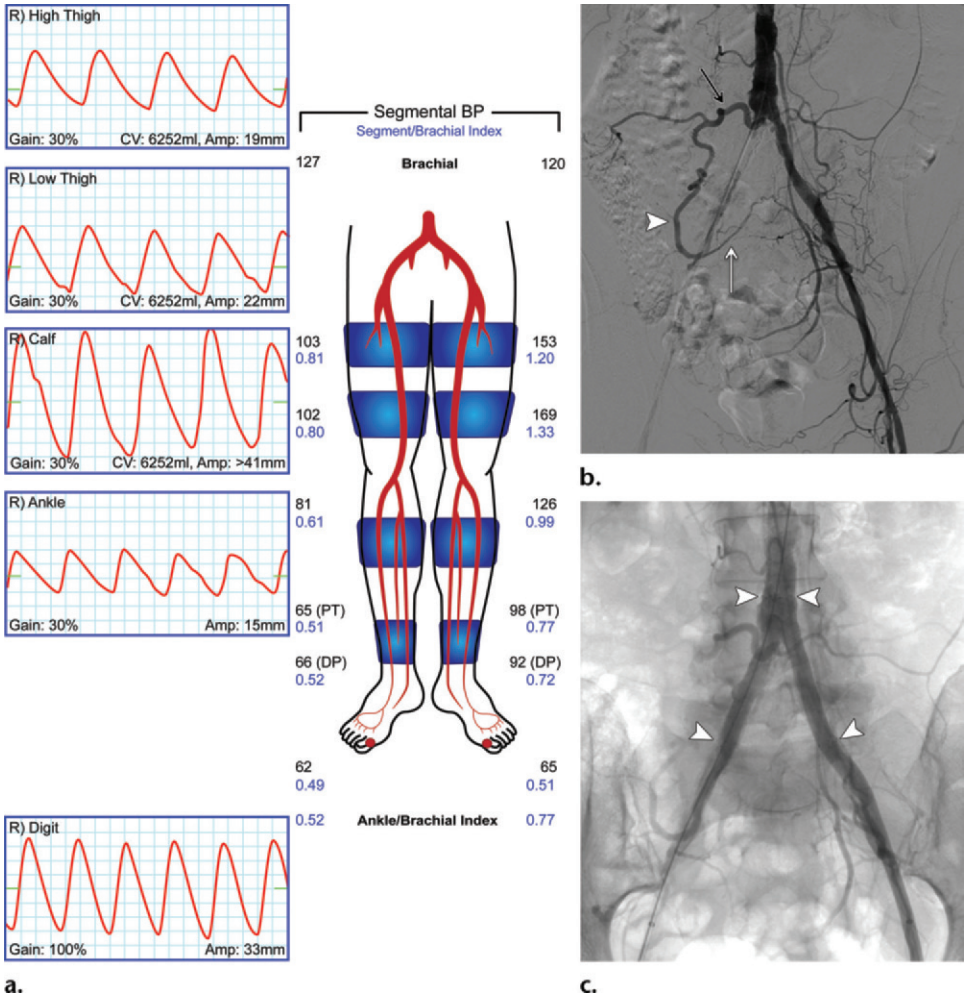


Figure 4. Aortoiliac disease in a 59-year-old man with bilateral lower extremity claudication after walking one block. **(a)** PVRs demonstrate widened waveforms, with loss of the dicrotic notch and concave-down late diastolic components. Right and left ABIs are 0.52 and 0.77, respectively. **(b)** Pelvic angiogram shows aortoiliac disease (greater on the right than on the left) with collateral flow via an enlarged right lumbar artery (black arrow) to the right iliolumbar artery (arrowhead). The right lateral sacral artery (white arrow) is also noted, with collateral flow to the contralateral lateral sacral artery. **(c)** Pelvic angiogram after intervention with kissing iliac stents demonstrates patent iliac arteries. Arrowheads = ends of the kissing iliac stents.

notch (24). Interpretation of PVRs in combination with Doppler waveforms can also help diagnose chronicity of arterial occlusive disease. In acute thrombosis, both the Doppler waveform and the PVR waveform are absent or decreased. With the development of arterial collaterals, as is seen with chronic occlusive disease, the PVR waveform may be relatively preserved compared with the Doppler waveform.

Distribution of Disease

PAD may affect an isolated segment of the aortoiliac, femoropopliteal, or crural vasculature or be distributed in a multisegmental fashion. In a study of 626 patients who underwent angiography, Ozkan et al (25) found that 64% of the patients had multisegmental disease and 22% of patients had disease across all three segments. Select risk factors have been associated with the distribution of PAD. Diabetic patients demonstrate a higher incidence of disease in the crural segments (25,26). Additionally, there is evidence to suggest that aortoiliac disease is more commonly seen in patients with a history of smoking (25,27).

Aortoiliac Disease

Aortoiliac disease, sometimes referred to as inflow disease, describes atherosclerotic disease involving the infrarenal abdominal aorta, common, internal, and external iliac arteries. Although presentations vary, aortoiliac disease may present as buttock, hip, or thigh claudication. Patients often have difficulty ambulating due to pain and weakness. At physical examination, one or both femoral pulses are diminished. Femoral Doppler waveforms for aortoiliac disease are typically biphasic or monophasic and high thigh PVRs are abnormal, indicating proximal disease (Fig 4).

Aortoiliac disease may also manifest as the classic triad of buttock or thigh claudication, erectile dysfunction, and decreased or absent femoral pulses described by French surgeon René Leriche in 1923 and now known as Leriche syndrome. Various collateral pathways develop in occlusive aortoiliac disease. Systemic-systemic pathways connect intercostal arteries, lumbar arteries, and iliolumbar arteries with inferior epigastric and deep circumflex arteries. Visceral-visceral collateral pathways exist between the celiac trunk, superior mesenteric, internal mesenteric, and superior rectal arteries (Fig

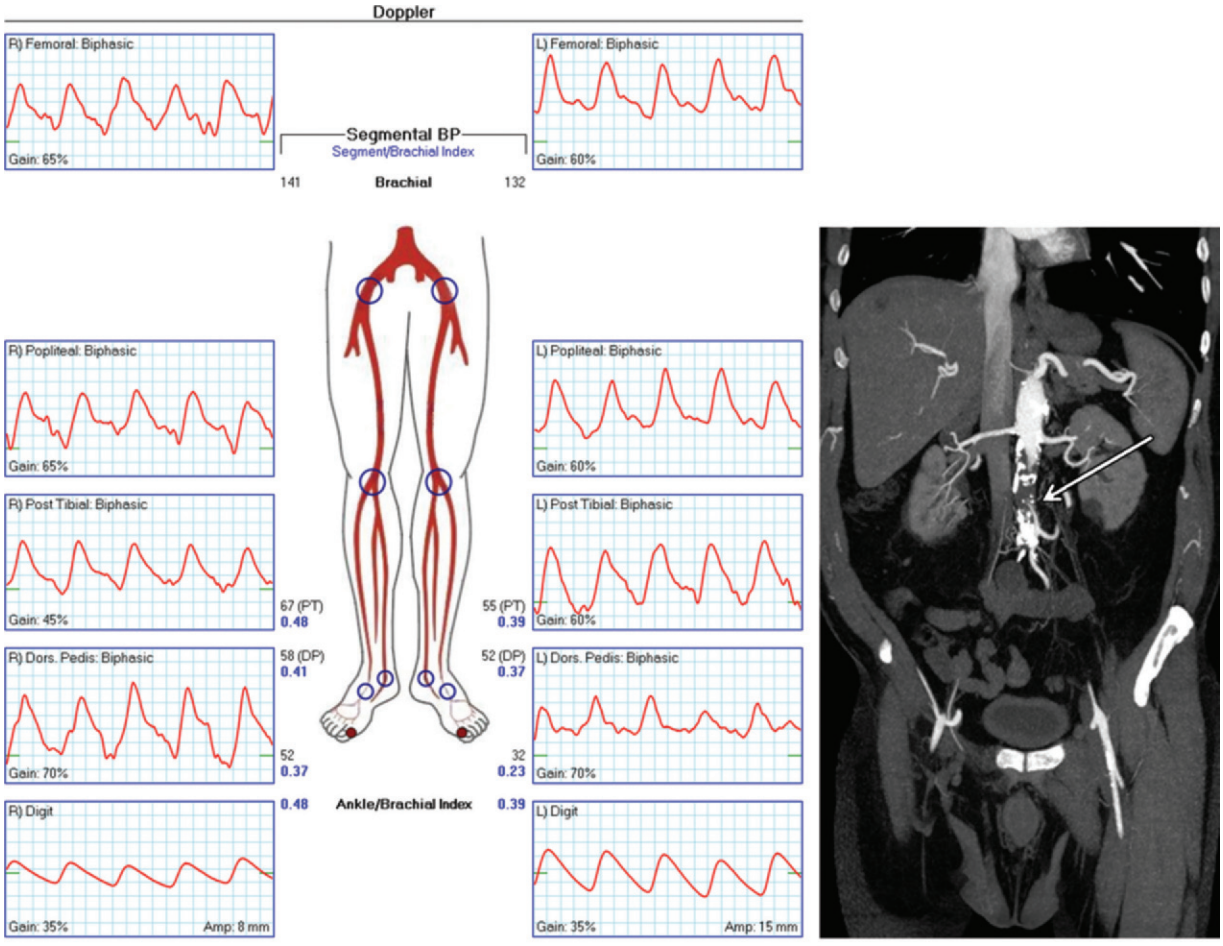


Figure 5. Occluded infrarenal abdominal aorta in a 64-year-old male smoker with claudication of the buttocks, thighs, calves, and feet after walking less than 100 ft (30 m) as well as erectile dysfunction. **(a)** Right and left ABIs are 0.48 and 0.39, respectively. The Doppler waveforms are biphasic throughout bilaterally. **(b)** CT angiogram demonstrates the occluded infrarenal abdominal aorta (arrow) (Leriche syndrome).

5). Rarely, gonadal pathways can arise, with the gonadal artery supplying blood flow to the inferior epigastric artery (28).

Femoropopliteal Disease

Femoropopliteal disease involves the common femoral, profunda femoral, and superficial femoral arteries, which continue down the leg to become the popliteal arteries as they exit the adductor hiatus. The popliteal artery ends at the origin of the anterior tibial artery. Femoropopliteal disease typically produces claudication in the thigh and calf. At physical examination, these patients have normal femoral pulses, but distal pulses are diminished. Calf claudication due to superficial femoral artery stenosis typically causes pain in the upper two-thirds of the calf. Pain in the lower one-third of the calf is associated with popliteal disease. Femoral Doppler waveforms can be triphasic, biphasic, or monophasic, depending on the level of the lesion. Popliteal, posterior tibial, and dorsalis pedis Doppler waveforms are abnormal (Fig 6). High thigh PVRs are typically normal, and above the knee, below the knee, and at the ankle PVRs are typically abnormal, depending on the level of the lesion.

Crural Disease

Crural disease involves the anterior tibial, posterior tibial, peroneal, dorsalis pedis, and plantar arteries. Although foot claudication is uncommon in PAD, it is typically associated with disease of the tibial and peroneal arteries. In crural disease, Doppler waveforms deteriorate from the popliteal level to the posterior tibial, dorsalis pedis, or digital level. PVRs below the knee and above the ankle are abnormal, depending on the level of the lesion. As ABIs are calculated using the high ankle pressure (ie, dorsalis pedis or posterior tibial artery), this screening tool may miss distal crural disease (Fig 7).

Exercise Study

In symptomatic patients with normal or borderline ABI at rest, an exercise ABI should be

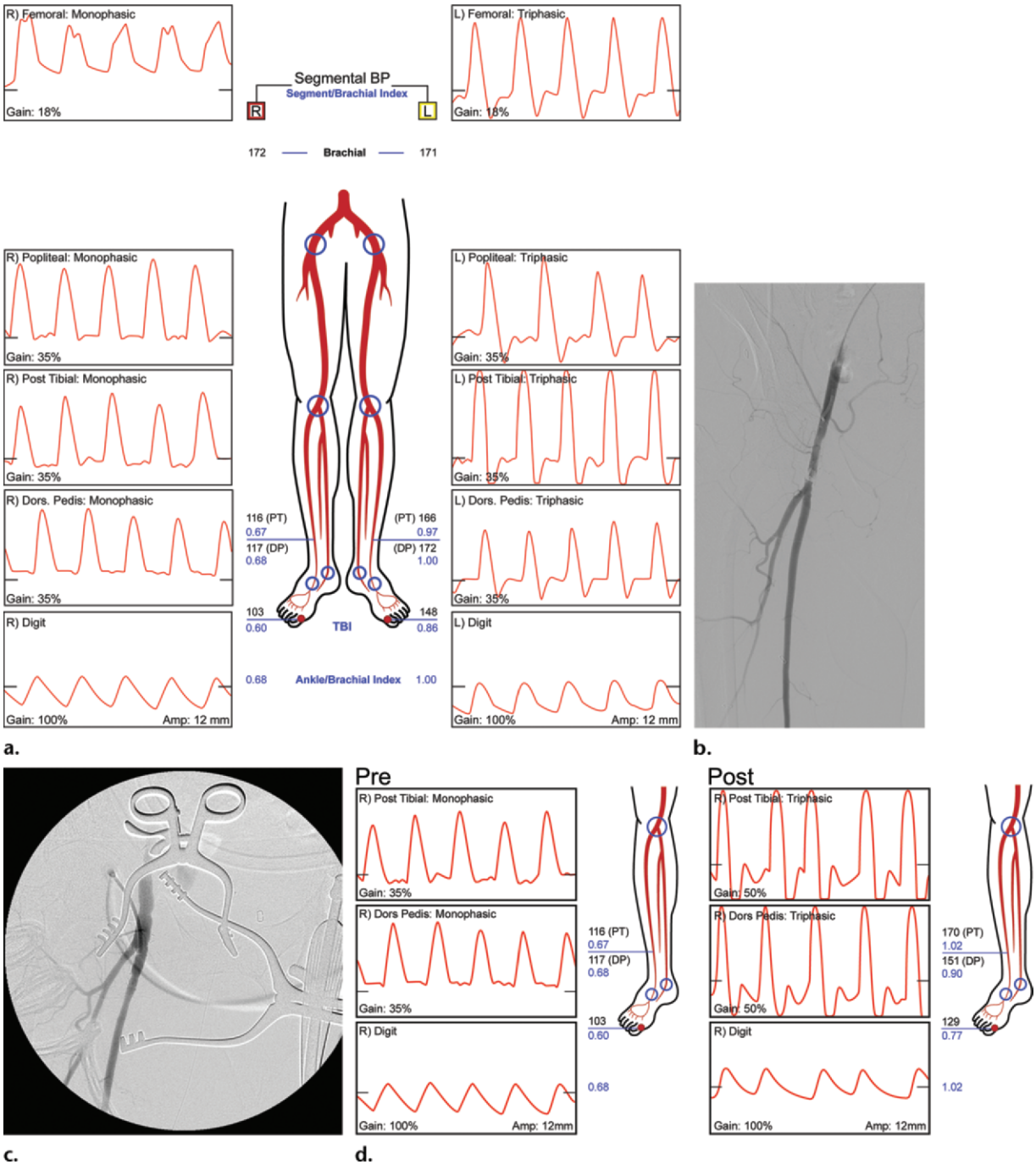


Figure 6. Near-total occlusion of the common femoral artery in a 71-year-old woman with claudication of the right lower extremity associated with walking. **(a)** ABI of 0.68 with monophasic Doppler waveforms on the right and 1.00 with triphasic waveforms on the left. **(b)** Right lower angiogram shows near-occlusion of the common femoral artery. The superficial femoral artery, popliteal artery, and tibiopedal arteries were unremarkable. **(c)** Angiogram of right common femoral artery endarterectomy. **(d)** Comparison of pre- and posttreatment ABIs and Doppler waveforms shows marked improvement in the ABI from 0.68 to 1.02 and associated change in the waveform from monophasic to triphasic.

performed. The sensitivity for the detection of PAD may be increased with postexercise measurements. The patient should walk on a treadmill at 2 mph (3.22 km/h) at a 10%–12% grade for 5 minutes or until claudication symptoms develop. ABIs should be measured immediately after exercise and every minute until ABIs normalize to pre-exercise values. The examination allows assessment of functional limitation and should be reproducible to allow monitoring of response to therapy. A decrease in the ABI after exercise of greater than 0.2 indicates PAD. The time required for the ABI to return to baseline is also useful in detecting PAD. Ankle pressures normally return to baseline within 2 minutes after cessation of exercise. Return

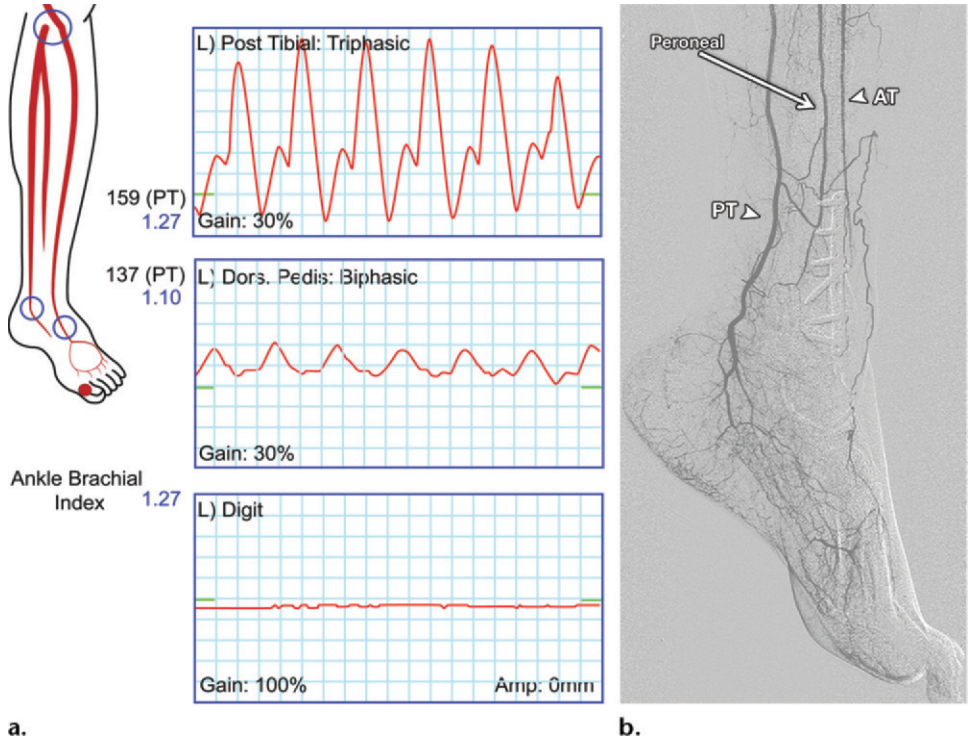


Figure 7. Occlusion of the anterior tibial artery in a 43-year-old woman with a 1-week history of left first through third digit discoloration. **(a)** There is a normal ABI study in the posterior tibial artery, with an ABI of 1.27 and a triphasic waveform. A monophasic and biphasic waveform is noted in the dorsalis pedis artery, with no discernible waveform in the left digit. **(b)** Angiogram of the left lower extremity reveals a normal posterior tibial artery *(PT)* with occlusion of the anterior tibial artery *(AT)* at the level of a previously placed fibular fixation plate.

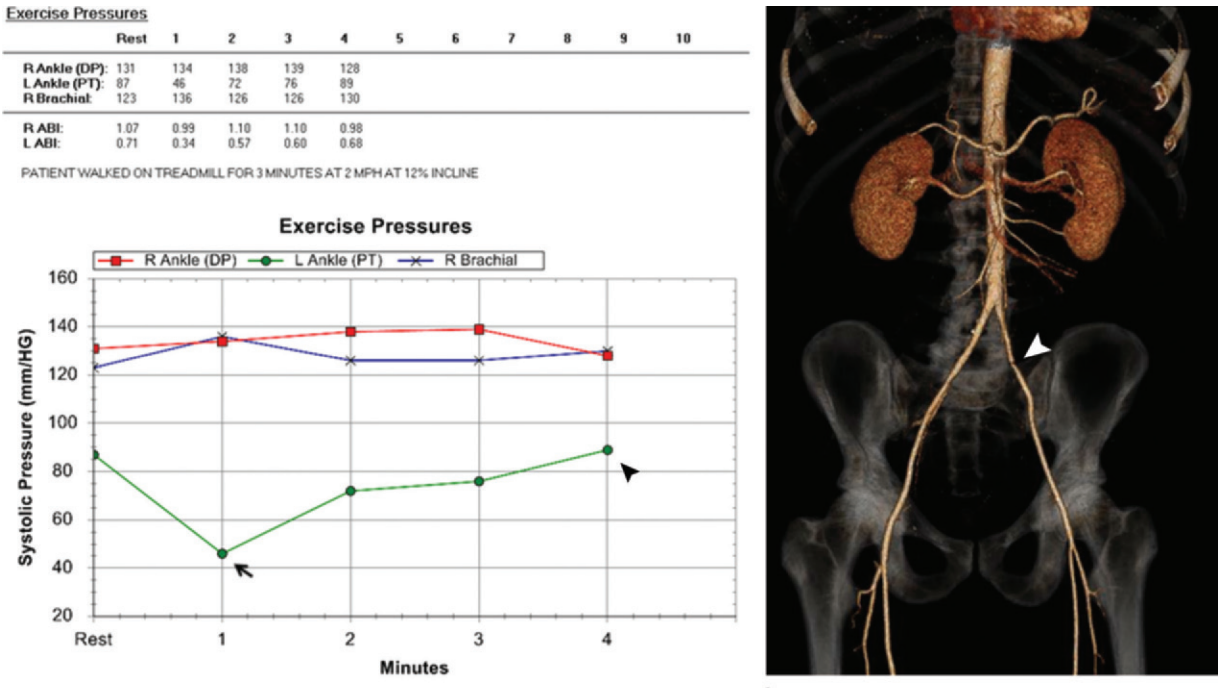
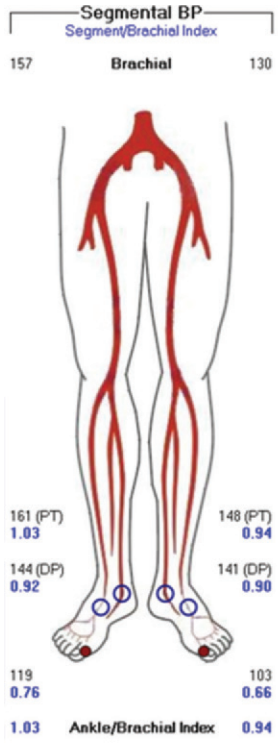


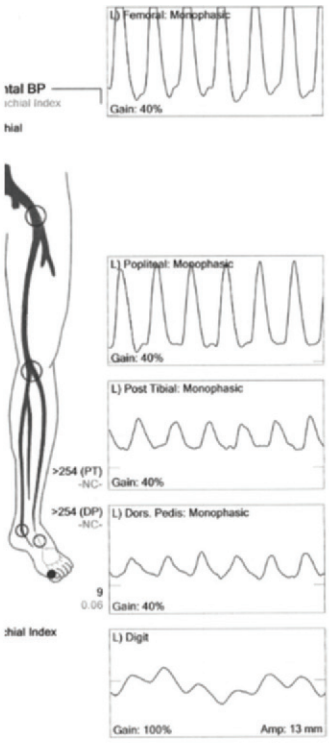
Figure 8. Iliac arterial stenosis in a 53-year-old man with left leg claudication. An ABI of 1.07 on the right and 0.71 on the left were found at rest. Doppler waveforms (not shown) were triphasic on the right and monophasic on the left from the common femoral artery to the ankle. **(a)** Exercise study demonstrates a drop in the left ABI of 0.34 1 minute after exercise (arrow) that returned to baseline after 4 minutes (arrowhead). The right ABI remained stable. **(b)** Reconstructed three-dimensional (3D) image demonstrates a focal segment of severe stenosis (arrowhead) in the left external iliac artery.

to baseline after 2–6 minutes of rest indicates single-segment disease (Fig 8), whereas return to baseline in 6–12 minutes indicates multisegment disease and return to baseline in greater than 15 minutes typically indicates rest pain (13).

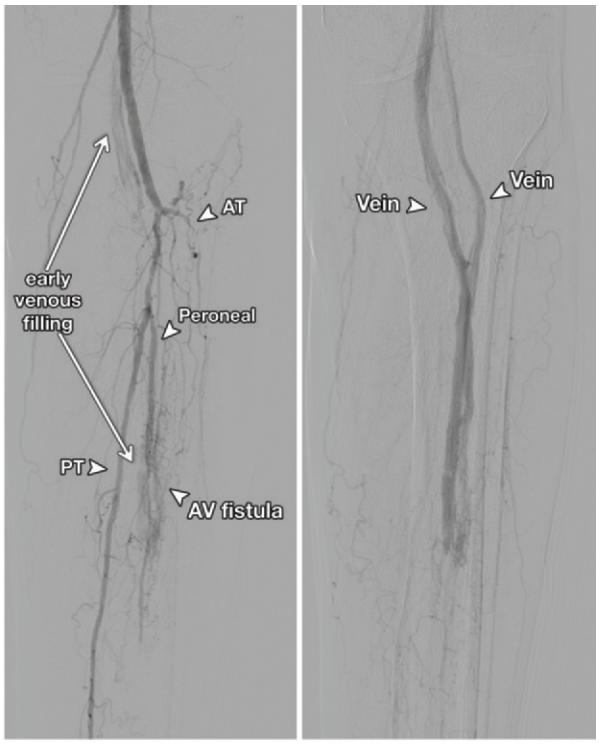
In addition, an exercise study may be useful to determine quantitative limitation in functional capacity secondary to claudication, which can then be used to assess response to therapy or an exercise program (29).



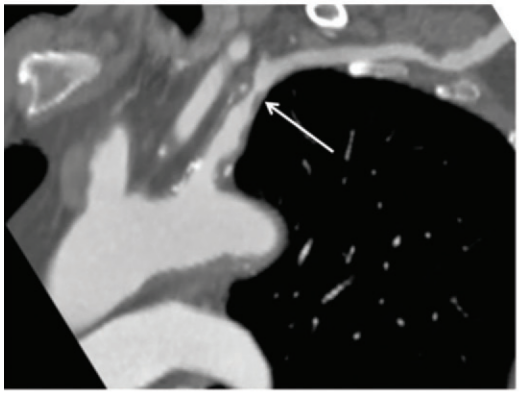
9a.



10a.



10b.



9b.

Figures 9, 10. **(9)** Subclavian arterial stenosis in a 59-year-old man with bilateral hip pain associated with walking. **(a)** There is a 27 mm Hg difference in brachial artery pressures (greater on the right than on the left). **(b)** CT angiogram shows severe left subclavian arterial stenosis (arrow). **(10)** Peroneal AVF in a 65-year-old man with left second and third gangrenous toes. **(a)** Systolic pressures are not measurable due to noncompressibility of calcified vessels. Doppler waveforms demonstrate monophasic waveforms throughout the left. **(b)** Left lower extremity angiogram (left) demonstrates a peroneal arteriovenous *(AV)* fistula with early venous filling. The approximate location of the AVF is noted along with an anterior tibial artery *(AT)* that is occluded proximally, as well as the posterior tibial *(PT)* and peroneal arteries. Draining veins are noted on an image (right) from a later phase of the angiogram.

Alternative Diagnoses

Although the primary objective of noninvasive physiologic vascular studies is to diagnose and characterize atherosclerotic PAD of the lower extremity, the noninvasive studies described may provide evidence for other disease entities. When there is a difference in brachial pressures of greater than 20 mm Hg, the patient should be evaluated for subclavian stenosis, extrinsic compression of the arterial supply to the upper extremity, and aortic dissection (Fig 9). Many disease processes may affect the arterial system of the lower extremity and therefore may also cause abnormal noninvasive vascular studies.

A few examples of abnormal vascular studies with causes other than atherosclerotic PAD follow. In arteriovenous fistulas (AVFs) of the lower extremity that are acquired, a shunt connects arterial blood flow directly to a vein. The functional impact of lower extremity AVFs can be assessed with noninvasive physiologic vascular studies (Fig 10). Takayasu arteritis, a chronic vasculitis of unknown cause most commonly found in Asian women, causes inflammation of the arterial wall. The aorta and its primary branches are primarily affected. In patients with presentations suspicious for Takayasu arteritis or a diagnosis of the disease, initial vascular lesions frequently occur in the subclavian artery, leading to decreased brachial pressures (Fig 11). Thromboangiitis obliterans, also known as Buerger disease, is a vasculitis that affects small to medium-sized vessels of the extremities of young patients with a smoking history. Thrombangiitis obliterans can manifest as a low ABI, but a normal ABI does not rule it out. The disease may be limited to distal vasculature, so digital pressures and PVRs may show decreased pressure and one or more flattened waveforms, respectively. Wrist-brachial indexes

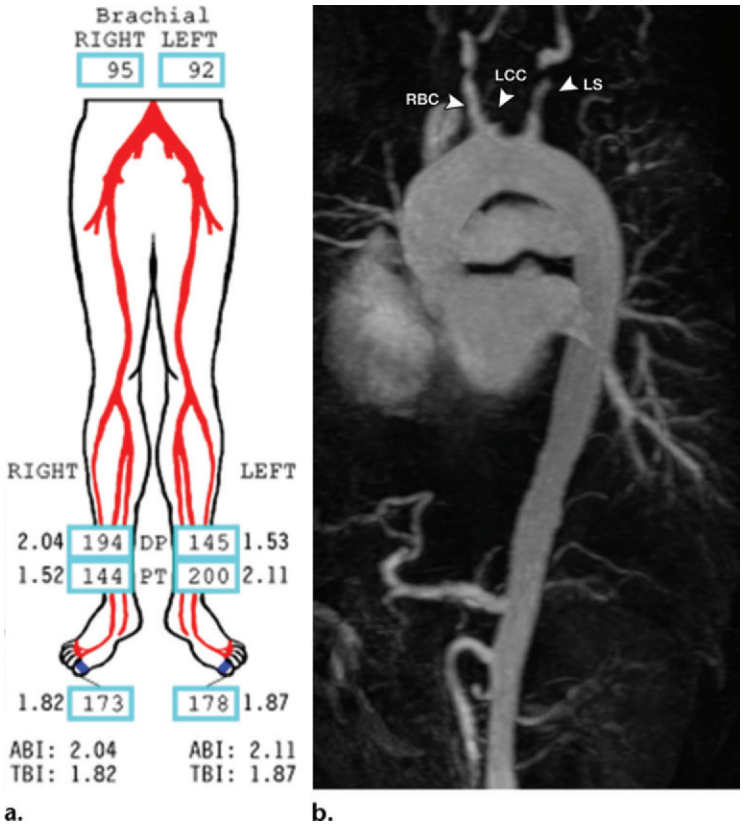


Figure 11. Takayasu arteritis in a 23-year-old woman with light-headedness, arm claudication, and shortness of breath. **(a)** Right and left ABIs are markedly elevated, at 2.04 and 2.11, respectively. **(b)** Maximum intensity projection candy-cane view of the aorta shows irregularity of the right brachiocephalic artery *(RBC)*, occlusion of the left common carotid artery *(LCC)*, and irregularity and occlusion of the left subclavian artery *(LS)* (arrowheads). The image was acquired with the blood-pool agent gadofosveset and rendered with 3D software.

are warranted in patients with upper extremity involvement. Given the role of smoking in PAD and thrombangiitis obliterans, noninvasive physiologic vascular studies should be used to exclude concomitant proximal lesions. Angiographic studies may demonstrate characteristic corkscrew collaterals (30) (Fig 12). Other conditions that can alter findings of noninvasive physiologic vascular studies include coarctation of the aorta, popliteal artery entrapment syndrome, cystic adventitial disease, endofibrosis of the iliac artery, fibromuscular dysplasia, and idiopathic midaortic syndrome (31).

Conclusion

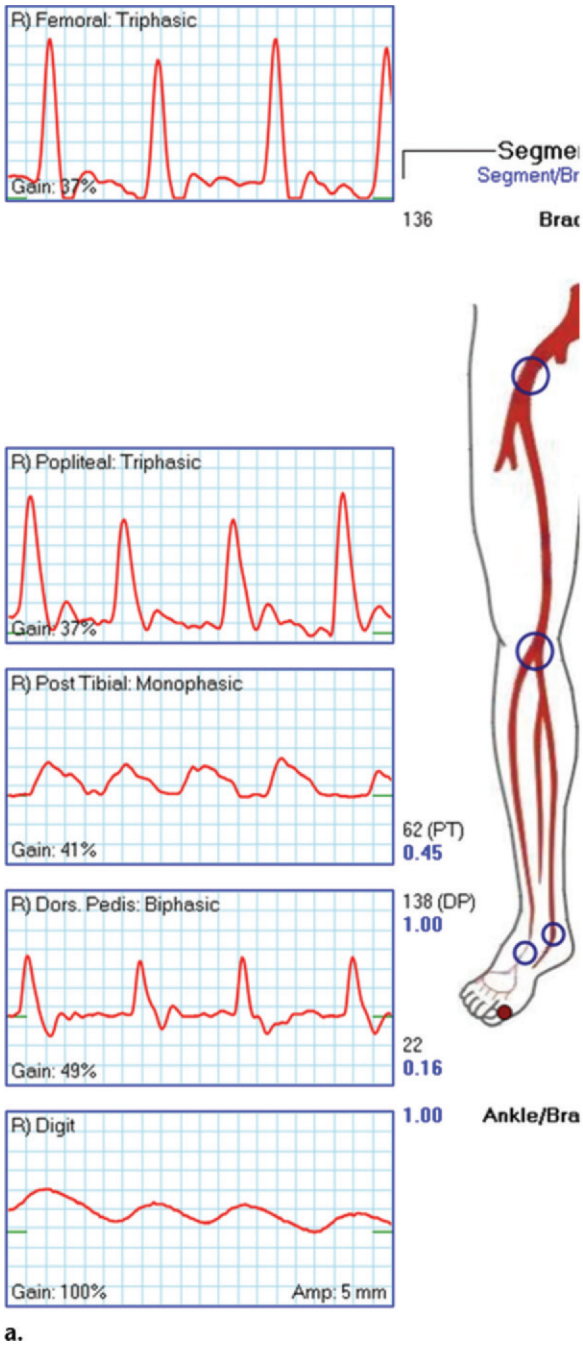
PAD affects a large portion of the population of the United States and is associated with serious morbidity and mortality. Early identification not only allows treatment of PAD but also modification of risk factors to reduce the risks associated with cardiovascular disease. Noninvasive physiologic vascular studies are an important tool in the diagnosis of PAD. Interpretation of these studies requires an understanding of the anatomy and physiology of arterial blood flow as well as the potential limitations of each modality. When interpreted together, these tools allow characterization of the site and severity of PAD. Although noninvasive physiologic vascular studies do not directly employ imaging, the role of the radiologist in interpretation is important, as these studies are a gateway to additional evaluation. When further evaluation of PAD is required, radiologists more frequently rely on the more efficient, less expensive and less risky noninvasive studies such as CT and MR angiography, reducing costs and risks to patients with PAD compared with other specialties.

Acknowledgment.—The authors wish to thank Erin Moore for supplying the medical illustrations.

Disclosures of Conflicts of Interest.—**S.P.K.** *Activities related to the present article:* disclosed no relevant relationships. *Activities not related to the present article:* royalties from Elsevier and Springer, personal fees from CeloNova Biosciences and the Koo Foundation. *Other activities:* disclosed no relevant relationships.

References

1. Ruo B, Liu K, Tian L, et al. Persistent depressive symptoms and functional decline among patients with peripheral arterial disease. Psychosom Med 2007;69(5):415–424.
2. McDermott MM, Liu K, Greenland P, et al. Functional decline in peripheral arterial disease: associations with the ankle brachial index and leg symptoms. JAMA 2004;292(4):453–461.
3. Lau JF, Weinberg MD, Olin JW. Peripheral artery disease. I. Clinical evaluation and noninvasive diagnosis. Nat Rev Cardiol 2011;8(7):405–418.
4. Rosamond W, Flegal K, Furie K, et al. Heart disease and stroke statistics: 2008 update—a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation 2008;117(4):e25–e146.
5. McDermott MM, Greenland P, Liu K, et al. The ankle brachial index is associated with leg function and physical activity: the Walking and Leg Circulation Study. Ann Intern Med 2002;136(12):873–883.



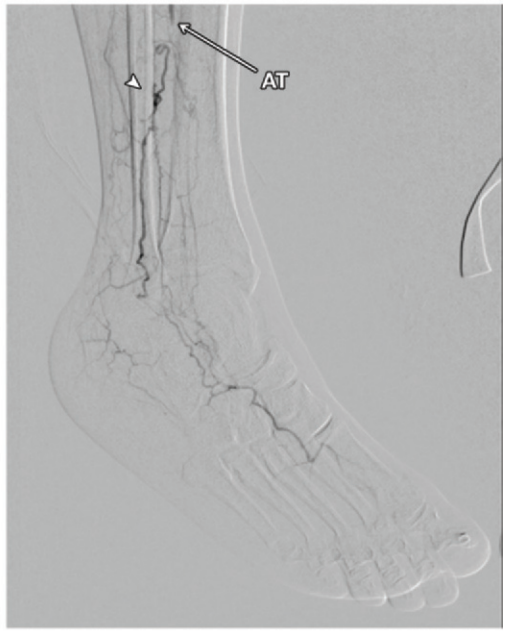


Figure 12. Right toe wound and thrombangiitis obliterans in a 45-year-old woman with a history of smoking, hypertension, and bilateral rest pain in the lower extremities. **(a)** Right lower extremity with ABI of 1.00 and posterior tibial monophasic Doppler waveforms. **(b)** Angiogram shows the anterior tibial artery *(AT)* occluded in the distal calf and classic corkscrew collaterals extending distally (arrowhead). The aortoiliac and femoropopliteal segments were unremarkable (not shown), and the posterior tibial and peroneal arteries were occluded proximally (not shown).

6. Norgren L, Hiatt WR, Dormandy JA, et al. Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC II). J Vasc Surg 2007;45(suppl S):S5–S67.
7. Dill KE, Rybicki FJ, Desjardins B, et al. Claudication: suspected vascular etiology. In: ACR Appropriateness Criteria. Reston, Va: American College of Radiology, 2012.
8. AbuRahma AF, Bergan JJ. Noninvasive vascular diagnosis: a practical guide to therapy. 2nd ed. London, England: Springer, 2006.
9. Levin DC, Gardiner GA Jr, Parker L, Rao VM. Vascular ultrasound and noninvasive physiological testing for peripheral arterial disease: are these tests being overused? J Am Coll Radiol 2016;13(3):249–254.
10. Patel MC, Levin DC, Parker L, Rao VM. Have CT and MR angiography replaced catheter angiography in diagnosing peripheral arterial disease? J Am Coll Radiol 2015;12(9):909–914.
11. Young DF, Cholvin NR, Kirkeeide RL, Roth AC. Hemodynamics of arterial stenoses at elevated flow rates. Circ Res 1977;41(1):99–107.
12. Olin JW, Allie DE, Belkin M, et al. ACCF/AHA/ACR/SCAI/SIR/SVM/SVN/SVS 2010 performance measures for adults with peripheral artery disease: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Performance Measures, the American College of Radiology, the Society for Cardiac Angiography and Interventions, the Society for Interventional Radiology, the Society for Vascular Medicine, the Society for Vascular Nursing, and the Society for Vascular Surgery (Writing Committee to Develop Clinical Performance Measures for Peripheral Artery Disease). Vasc Med 2010;15(6):481–512.
13. Mohler ER 3rd. Peripheral arterial disease: identification and implications. Arch Intern Med 2003;163(19):2306–2314.
14. Wolf EA Jr, Sumner DS, Strandness DE Jr. Correlation between nutritive blood flow and pressure in limbs of patients with intermittent claudication. Surg Forum 1972;23(0):238–239.

15. McDermott MM, Feinglass J, Slavensky R, Pearce WH. The ankle-brachial index as a predictor of survival in patients with peripheral vascular disease. J Gen Intern Med 1994;9(8):445–449.
16. Hirsch AT, Haskal ZJ, Hertzer NR, et al. ACC/AHA 2005 practice guidelines for the management of patients with peripheral arterial disease (lower extremity, renal, mesenteric, and abdominal aortic): a collaborative report from the American Association for Vascular Surgery/Society for Vascular Surgery, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, Society of Interventional Radiology, and the ACC/AHA Task Force on Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Peripheral Arterial Disease)—endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation; National Heart, Lung, and Blood Institute; Society for Vascular Nursing; TransAtlantic Inter-Society Consensus; and Vascular Disease Foundation. Circulation 2006;113(11):e463–e654.
17. Ramsey DE, Manke DA, Sumner DS. Toe blood pressure: a valuable adjunct to ankle pressure measurement for assessing peripheral arterial disease. J Cardiovasc Surg (Torino) 1983;24(1):43–48.
18. Vitti MJ, Robinson DV, Hauer-Jensen M, et al. Wound healing in forefoot amputations: the predictive value of toe pressure. Ann Vasc Surg 1994;8(1):99–106.
19. Apelqvist J, Castenfors J, Larsson J, Stenström A, Agardh CD. Prognostic value of systolic ankle and toe blood pressure levels in outcome of diabetic foot ulcer. Diabetes Care 1989;12(6):373–378.
20. Carter SA, Tate RB. Value of toe pulse waves in addition to systolic pressures in the assessment of the severity of peripheral arterial disease and critical limb ischemia. J Vasc Surg 1996;24(2):258–265.
21. Kirkendall WM, Feinleib M, Freis ED, Mark AL. Recommendations for human blood pressure determination by sphygmomanometers: Subcommittee of the AHA Postgraduate Education Committee. Circulation 1980;62(5):1146A–1155A.
22. Darling RC, Raines JK, Brener BJ, Austen WG. Quantitative segmental pulse volume recorder: a clinical tool. Surgery 1972;72(6):873–877.
23. Kempczinski RF. Segmental volume plethysmography in the diagnosis of lower extremity arterial occlusive disease. J Cardiovasc Surg (Torino) 1982;23(2):125–129.
24. Raines JK, Jaffrin MY, Shapiro AH. A computer simulation of arterial dynamics in the human leg. J Biomech 1974;7(1):77–91.
25. Ozkan U, Oguzkurt L, Tercan F. Atherosclerotic risk factors and segmental distribution in symptomatic peripheral artery disease. J Vasc Interv Radiol 2009;20(4):437–441.
26. Strandness DE Jr, Priest RE, Gibbons GE. Combined clinical and pathologic study of diabetic and nondiabetic peripheral arterial disease. Diabetes 1964;13:366–372.
27. Diehm N, Shang A, Silvestro A, et al. Association of cardiovascular risk factors with pattern of lower limb atherosclerosis in 2659 patients undergoing angioplasty. Eur J Vasc Endovasc Surg 2006;31(1):59–63.
28. Hardman RL, Lopera JE, Cardan RA, Trimmer CK, Josephs SC. Common and rare collateral pathways in aortoiliac occlusive disease: a pictorial essay. AJR Am J Roentgenol 2011;197(3):W519–W524.
29. Anderson JL, Halperin JL, Albert NM, et al. Management of patients with peripheral artery disease (compilation of 2005 and 2011 ACCF/AHA guideline recommendations): a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation 2013;127(13):1425–1443.
30. Fujii Y, Soga J, Hidaka T, et al. Color Doppler flows of corkscrew collaterals in thromboangiitis obliterans (Buerger's disease) using color duplex ultrasonography. J Am Coll Cardiol 2011;57(25):2539.
31. Weinberg I, Jaff MR. Nonatherosclerotic arterial disorders of the lower extremities. Circulation 2012;126(2):213–222.