Noninvasive Arterial Testing: What and When to Use

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Abstract

Peripheral arterial disease (PAD) represents a growing public health issue that continues to be underdiagnosed. In its most severe form, critical limb ischemia, it contributes to expanding morbidity with minor and major limb amputations. PAD is strongly associated with increased mortality, as it is known to be concomitant with coronary and cerebrovascular disease. Diagnosis of PAD relies on noninvasive arterial testing, a class of tests that can provide physiologic or morphologic information. Physiologic tests such as ankle-brachial index, toe-brachial index, pulse volume recordings, and arterial duplex evaluation are the mainstay of gateway evaluation and surveillance. Morphologic exams such as computer tomographic angiography and magnetic resonance angiography are appropriate for preprocedural anatomic evaluation in patients with established vascular disease. This review focuses on physiologic exams.

Keywords

- noninvasive arterial testing
- ankle-brachial index
- pulse volume recordings
- physiologic arterial test
- interventional radiology

Objectives: Upon completion of this article, the reader will be able to identify the fundamentals of physiologic noninvasive arterial testing including strengths, weaknesses, and commonly applied threshold values.

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Peripheral arterial disease (PAD) is a vascular disease affecting a relatively large and growing percentage of the population worldwide. Its most severe form, critical limb ischemia (CLI), manifests as tissue loss or ischemic rest pain for more than 2 weeks in duration. The estimated prevalence in the United States in 2008 in adults 40 years or older of PAD was 10.7% and CLI was 1.3%.[1] The global estimate of PAD is 202 million people in 2010 with increases in the preceding decade of 28% in low-income or middle-income countries and 13% in high-income countries.[2] Despite the large and growing prevalence of PAD, the disease remains markedly underdiagnosed even with consequences as severe as limb loss in patients with CLI.[3] Noninvasive arterial testing comprises a series of exams used individually or in combination in the evaluation and surveillance of patients with known or suspected PAD or CLI. Testing is divided into exams that provide physiologic information and those that provide morphologic information. The most commonly utilized tests include segmental pressures, pulse volume recordings (PVRs), arterial duplex ultrasound, computed tomographic angiography (CTA), and magnetic resonance angiography (MRA). Components comprising a complete exam depend on the severity of disease at presentation and whether the patient is diabetic.

Segmental Pressures

Segmental pressures measure the systolic arterial pressure in multiple segments of the lower extremities. A blood pressure cuff typically is applied in four segments: high thigh, low thigh, calf, and ankle. It may not be possible to apply two thigh cuffs to shorter or obese patients. Each cuff above the ankle is inflated to a pressure at which a Doppler signal is lost in the dorsalis

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pedis. The ankle cuff is inflated to pressures that result in the loss of signal in the dorsalis pedis and posterior tibial artery, and these values are recorded separately. The systolic pressure in mm Hg is compared to the higher value of the upper extremity systolic brachial pressures, and a ratio is generated. The ratio of ankle pressure, using the greater of the dorsalis pedis or posterior tibial, is the ankle-brachial index (ABI). A drop in pressure of 20 mm Hg between any two levels is considered suspicious for a hemodynamically significant lesion in that segment.[4] In the author's experience, patients find a full segmental pressure exam to be highly uncomfortable.

Ankle-Brachial Index

The ABI is the most commonly used physiologic testing metric in the initial evaluation of patients with symptoms suspected to be secondary arterial insufficiency. The exam is inexpensive, not difficult to perform, straightforward to interpret, and requires minimal technology.[5] 2016 AHA/ACC guidelines recommend a resting ABI with or without segmental pressures and PVRs to establish a diagnosis in patients with a history or physical exam findings suggesting PAD.[6] Measurement of an ABI in asymptomatic patients with increased risk of PAD is considered reasonable by the same guidelines. Recommended classification of results is outlined in ►**Table 1**. Historic data suggest high sensitivity and specificity for the detection of hemodynamically significant arterial disease with an ABI.[5–7] Reproducibility of results between providers of different skill levels and between skilled providers has been questioned.[7,8] A major limitation of the exam is poor applicability to patients with poorly compressible or noncompressible arteries.[7,9,10] Diabetes and advanced chronic kidney disease are associated with medial wall calcification and subsequent poor arterial compressibility. The percentage of patients with noncompressible vessels reported in the literature has increased over time and may be attributable to the increase prevalence of diabetes.[9,11] Noncompressible vessels are reported in up to one-third of patients with diabetes.[9] Even in compressible vessels, medial calcification artificially inflates the ABI, and the metric is considered unreliable in the diabetic population particularly those with isolated infrapopliteal disease.[10]

Patients with history or physical exam findings suggesting PAD who have a normal ABI at rest should undergo further testing with measurement of a postexercise ABI.[12] Typically, exercise consists of walking on a treadmill for a short period of time such as 5 minutes. Ideally, the time spent on the treadmill is enough to elicit claudication-type symptoms. A normal ABI before and after exercise in a nondiabetic patient has a very high negative predictive value for arterial insufficiency as the cause of symptoms.[5]

Table 1 Ankle brachial index (ABI) and associated classification

ABI	Classification
≥1.4	Noncompressible (abnormal)
1.0–1.39	Normal
0.9–0.99	Borderline
0.7–0.89	Mild arterial insufficiency
0.4–0.69	Moderate arterial insufficiency
<0.4	Severe arterial insufficiency

Toe-Brachial Index

The toe-brachial index (TBI) is a ratio of the systolic pressure of the great toe to brachial systolic pressure. Digital arteries are less pervasively affected by medial wall calcification in diabetic patients than tibial vessels, and TBI is considered more reliable than ABI in these patients.[10] A TBI less than 0.7 is considered diagnostic of PAD.[6] Patients with foot ulceration, particularly those with diabetes, should be evaluated by measures additional to an ABI.[7] TBIs are both sensitive and cost-effective in identifying patients with arterial insufficiency contributing to a diabetic foot ulcer.[13] A patient with a wound and an absolute toe pressure of less than 30 mm Hg is unlikely to heal without revascularization, and a diabetic patient with an absolute toe pressure less than 45 mm Hg is unlikely to heal a wound.[14] A patient with a TBI less than 0.45 is unlikely to heal a toe or minor foot amputation.[15]

Pulse Volume Recordings

Pulse volume recordings or pulse volume plethysmography are physiologic tests that represent measurements of the change in volume at an interrogated level in the extremity with the cardiac cycle. Volume in an extremity increases transiently in systole and returns to baseline in diastole. PVRs are measured with cuffs, and modern devices can measure segmental pressures and PVRs with the same set of cuffs. To measure PVRs, cuffs are inflated to 60 to 70 mm Hg. The exam typically is better tolerated by patients than segmental pressures due to lower pressure cuff inflations. A normal waveform (►**Fig. 1**) has a brisk upstroke, narrow peak, brisk downstroke, and either a dicrotic notch or an

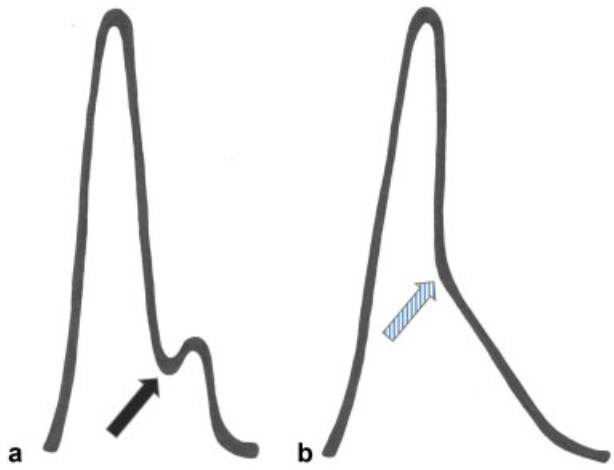

Fig. 1 Normal waveform morphology in a pulse volume recording. Classically, a normal waveform is described as having a dicrotic notch (**a**, *black arrow*). A common manifestation of a normal waveform has a clearly defined inflection point rather than a dicrotic notch (**b**, *dashed arrow*).

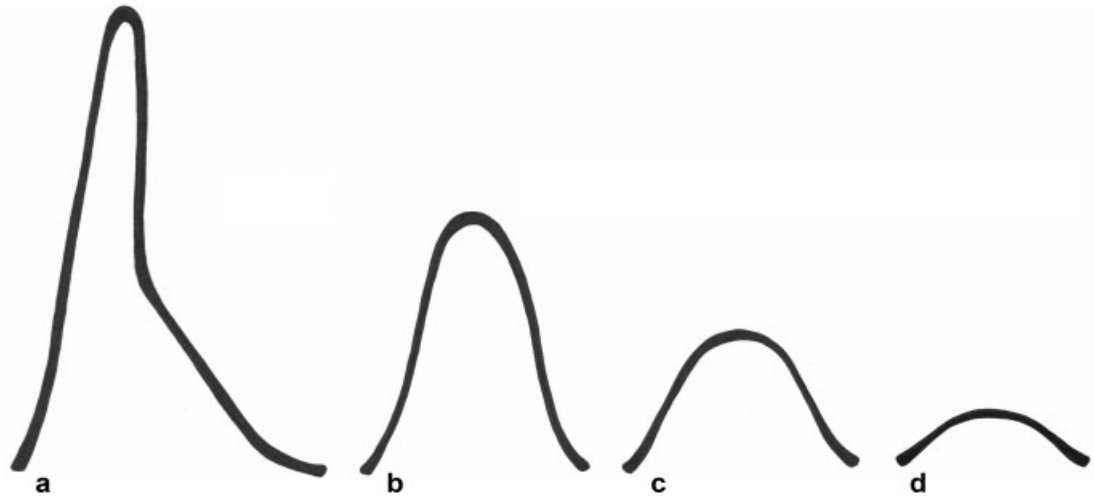

Fig. 2 Normal (**a**), mildly abnormal (**b**), moderately abnormal (**c**), and severely abnormal (**d**) pulse volume recordings. As the waveform degrades, the inflection point disappears first, followed by diminished waveform amplitude and increased upstroke time.

inflection point with a clear change in slope. A dicrotic notch is more common in normal exams of younger individuals, and an inflection point is more common in normal exams of older individuals. If a patient has a hemodynamically significant lesion or occlusion, all PVR waveforms distal to the culprit lesion will degrade (►**Fig. 2**). Waveform degradation follows a typical pattern that begins with early loss of the dicrotic notch or inflection point. Further degradation is marked by increased upstroke time, widening of the peak, and loss of amplitude. Waveforms obtained proximal to a culprit lesion do not degrade.

Normal PVRs are distinguished by waveform morphology and ratios between different levels (►**Fig. 3**). Waveform amplitude should increase 20 to 30% at the calf compared to the high thigh. Amplitude should be maintained at the ankle compared to the low thigh. Loss of augmentation at the thigh and failure to maintain amplitude at the ankle are abnormal findings even with normal waveform morphology (see ►**Figs. 4–6**). Ratios will be maintained for a given segment that lacks a hemodynamically significant lesion even if the waveform morphology is not normal. For example (see ►**Fig. 4**), an isolated iliac lesion will degrade waveform

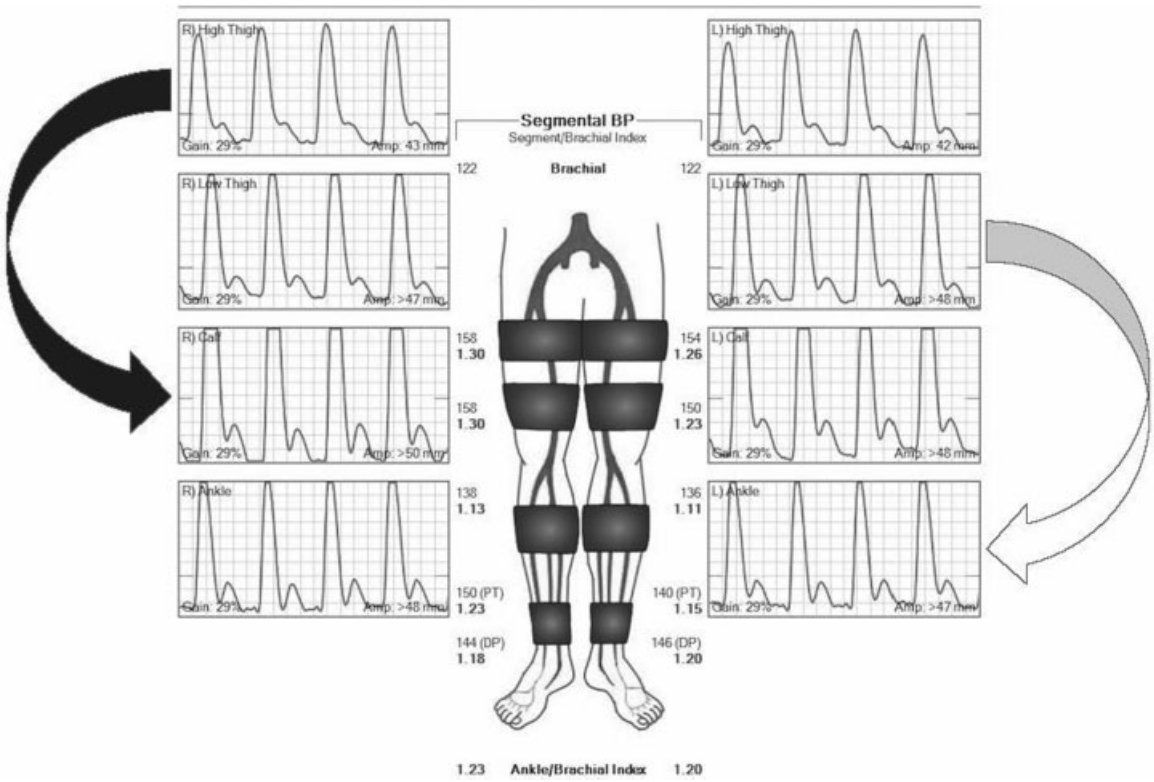

Fig. 3 Segmental pressures and pulse volume recordings in a normal exam. Exam demonstrates normal waveform morphology and ratios. There is appropriate augmentation at the calf (*black arrow*) and maintained amplitude at the ankle (*white arrow*).

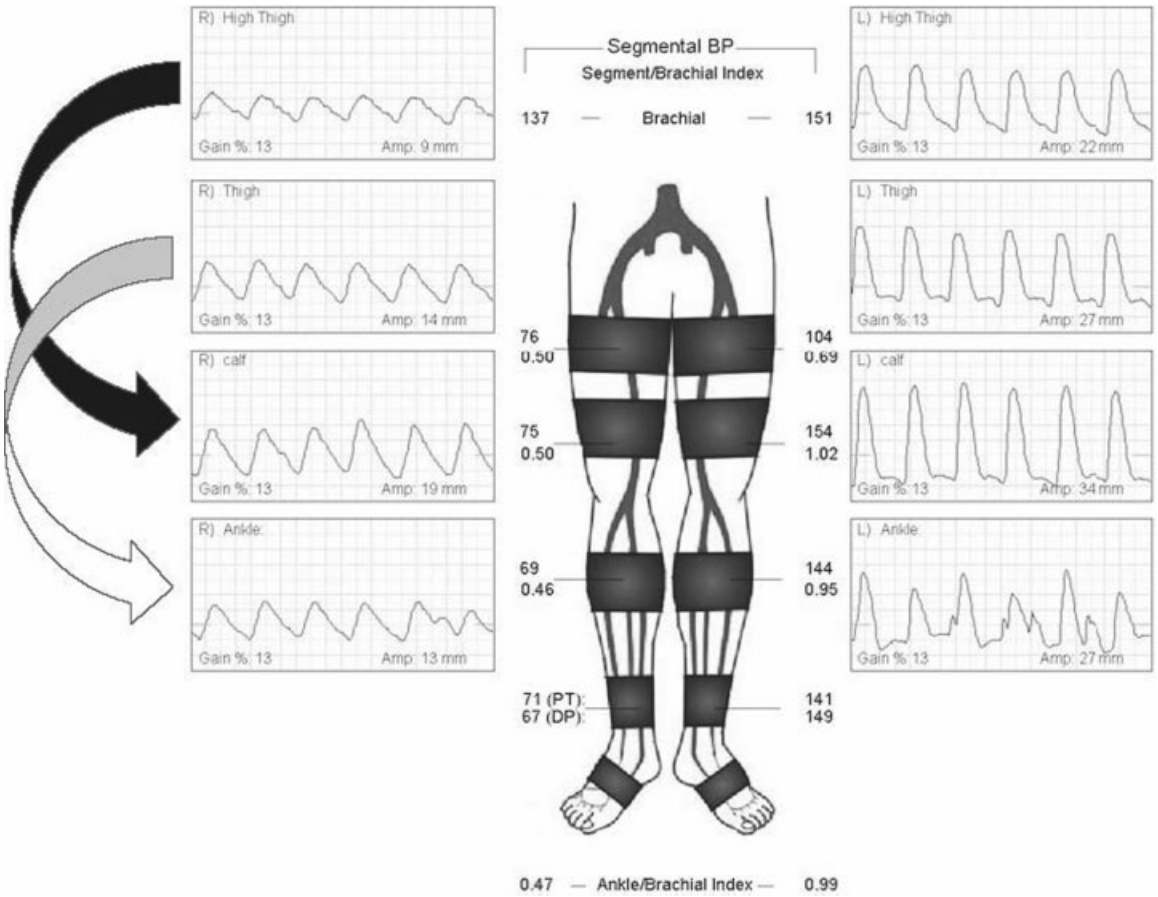

Fig. 4 Isolated unilateral iliac occlusive disease. PVRs on the right demonstrate waveform degradation and diminished amplitudes at the high thigh consistent with significant iliac disease. Augmentation is maintained at the calf (*black arrow*), and amplitude is maintained at the ankle (*white arrow*). PVRs on the left are normal.

morphology at all levels in an extremity. Amplitude ratios will remain normal if there is not a hemodynamically significant infrainguinal lesion in the same extremity. Analysis of both waveform morphology and ratios allows for identification or exclusion of hemodynamically significant lesions in multiple segments.

Unlike ABIs, vascular calcification and poor compressibility do not degrade PVRs.[12,16] As such, PVRs are considered a useful adjunct to segmental pressures in the vascular laboratory.[17,18] When combined with segmental pressures, PVRs have a reported diagnostic accuracy as high as 97%.[19] Not all data support the diagnostic value of PVRs,[10] and the qualitative interpretive component has been cited as a weakness.[7]

Arterial Duplex Ultrasound

Arterial duplex ultrasound of the lower extremity offers both physiologic and morphologic information. The exam combines grayscale imaging for anatomic detail with color Doppler evaluation for physiologic data. Speed and direction of flow can be determined with Doppler imaging. A normal Doppler waveform (►**Fig. 7a**) has rapid systolic acceleration, a narrow peak, rapid deceleration, brief reversal of flow during early diastole, transient return of antegrade flow during mid diastole, and loss of antegrade flow during late diastole. Persistent antegrade diastolic flow is not a typical feature of higher resistance arterial beds such as the lower extremities at rest, but its presence is not necessarily pathologic. It can be seen in conditions that result in diminished peripheral resistance such as paraplegia, muscular dystrophy, lower extremity disuse (wheelchair-bound patients), vascular malformations, and pedal infections.

The Doppler waveform will degrade in the setting of PAD. Proximal to a hemodynamically significant lesion, the waveform first will lose the transient return of antegrade flow during mid diastole with the result of a biphasic waveform (►**Fig. 7b**). As the lesion worsens or the Doppler sample is closer to a more severe stenosis or occlusion, brief reversal of flow during early diastole will be lost and will result in a monophasic, high-resistance waveform (►**Fig. 7c**). Downstream from a severe stenosis or occlusion, the waveform will be monophasic, low resistance with persistent antegrade diastolic flow (►**Fig. 7d**). Downstream from any hemodynamically significant lesion, there will be delayed systolic acceleration.

An arterial stenosis will result in increased flow velocity through the lesion due to Bernoulli's principle.[20] An exception occurs when the degree of stenosis reaches a critical threshold (>95%) and velocity decreases. There is no consensus regarding velocity thresholds and corresponding degrees of stenosis.[21] The ratio of the peak systolic velocity within the stenosis to the

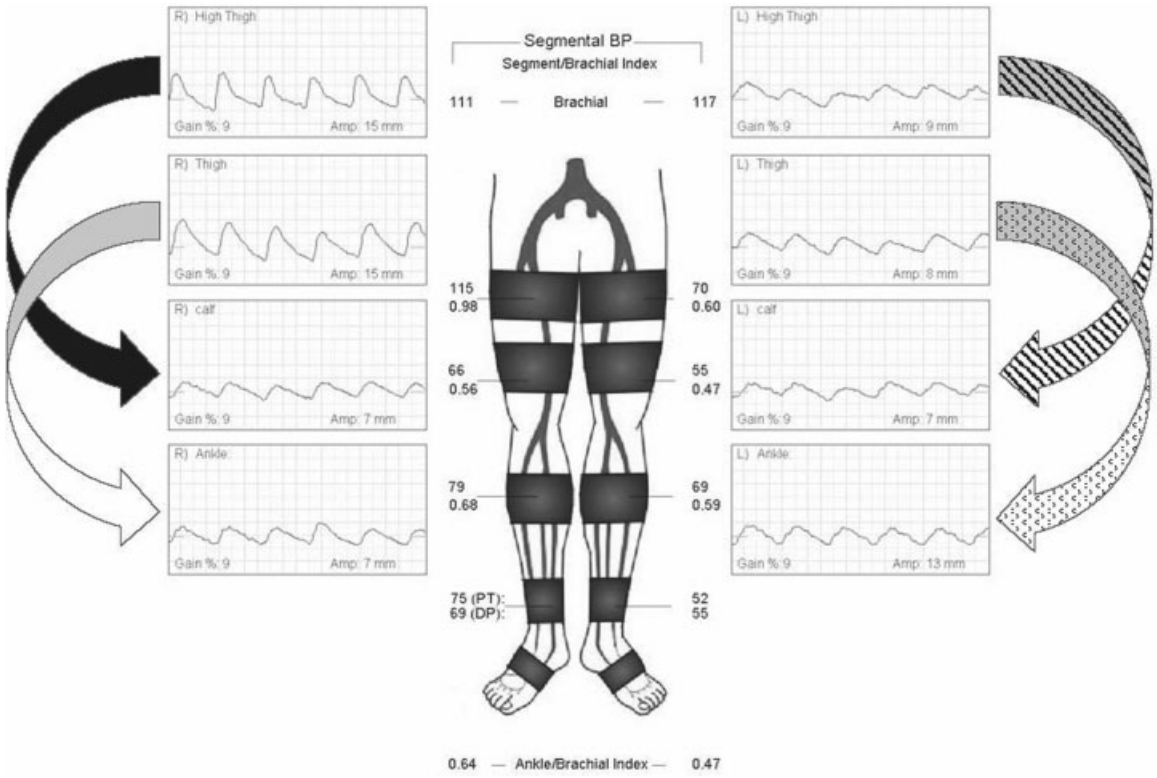

Fig. 5 Multilevel disease bilateral lower extremities. On the right, the high thigh waveform is normal. The calf waveform is degraded with loss of amplitude indicating occlusive fem-pop disease (*black arrow*). There is diminished amplitude at the ankle relative to the low thigh indicating tibial disease (*white arrow*). On the left, the high thigh waveforms and amplitudes are abnormal consistent with occlusive iliac disease. There is no augmentation at the calf consistent with concomitant fem-pop disease (*dashed arrow*). There is maintained amplitude at the ankle consistent with no significant tibial disease (*dotted arrow*).

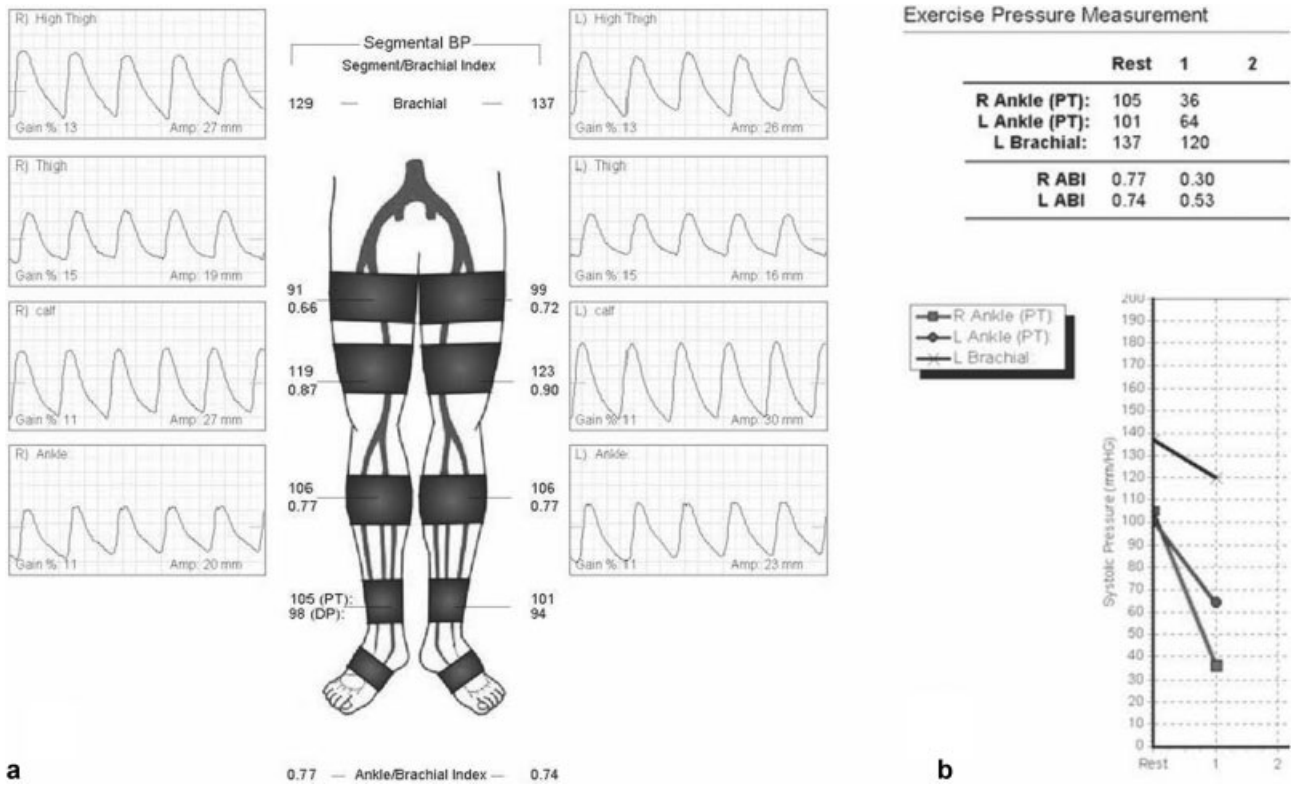

Fig. 6 PVRs and ABIS pre- and postexercise in a patient with aortoiliac occlusive disease. There is subtle abnormality of the PVR waveforms at all levels with no discrete inflection point. Lack of augmentation is due to gain adjustment by the sonographer (**a**). Following exercise, ABIs drop markedly and symmetrically (**b**).

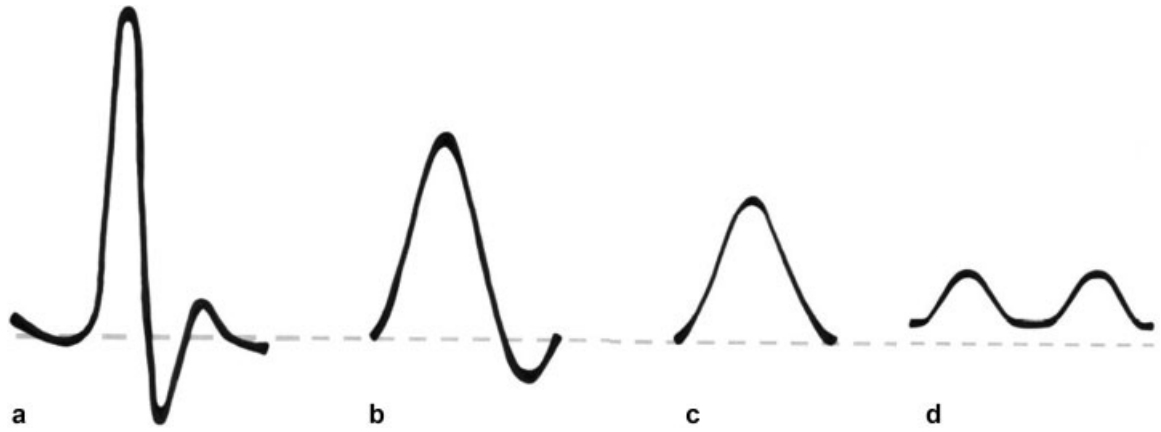

Fig. 7 (**a**) Normal Doppler waveform. (**b**) Biphasic waveform. (**c**) Monophasic high-resistance waveform. (**d**) Monophasic low-resistance waveform with persistent antegrade diastolic flow.

systolic velocity within an immediately proximal nonstenotic segment is considered more reliable for the classification of degree of stenosis with a high degree of sensitivity and specificity (►**Table 2**).[22] A peak systolic velocity ratio (PSVR) greater than 2.4 is generally accepted as indicating a greater than 50% stenosis and is used to designate loss of primary patency following angioplasty or stenting (►**Fig. 8**).[23]

Computed Tomography Angiography

Computed tomography angiography (CTA) is a noninvasive alternative to digital subtraction angiography (DSA) for morphologic evaluation of patients with known or suspected PAD. CTA is readily available, reasonable in cost, and provides excellent anatomic detail.[24] Meta-analysis has shown CTA to be 95% sensitive and 96% specific in detecting lesions with more than 50% stenosis or occlusion.[25] The examination does not provide any physiologic information, and patients may be ill served with CTA as an isolated exam if they have multisegment disease. If a patient has multiple lesions, CTA cannot distinguish between their relative hemodynamic significance. The exam utilizes ionizing radiation and iodinated contrast. Iodinated contrast can be problematic in patients with renal insufficiency, as it can potentiate acute kidney injury. Multiple protocols for mitigating renal injury have been proposed over time, but results from the PRESERVE trial suggest that hydration with intravenous 0.9% sodium chloride is adequate to any alternative treatment.[26]

Calcium in artery walls causes bloom artifact on CTA, a phenomenon known to exaggerate the visual degree of a stenosis.[25] Recent advances with dual-energy CTA, a technology that uses either two separate energy source beams or a single beam that rapidly switches between different voltages, allow for subtraction of high-density material such as bone and calcified plaque to generate images considered analogous to DSA.[27]

Table 2 Peak systolic velocity ratio and corresponding degree of stenosis

Stenosis (%)	PSVR	Sensitivity (%)	Specificity (%)
≥30	≥1.6	90	91
≥40	≥2.1	84	92
≥50	≥2.4	87	94
≥60	≥2.9	84	91
≥70	≥3.4	91	98
≥80	≥4.0	90	98
≥90	≥7.0	88	97

Abbreviation: PSVR, peak systolic velocity ratio.
Source: Reprinted with permission from Jaffer et al.[22]

Magnetic Resonance Angiography

Magnetic resonance angiography utilizes magnetic resonance imaging to create predominantly morphologic vascular information. Limited physiologic information can be generated from time of flight (ToF) imaging and time-resolved imaging of contrast kinetics (TRICKS). ToF provides low-resolution information on direction of flow. TRICKS provides the equivalent of time-resolved angiography with cine-type loops that allow for differentiation between vascular structures that fill at different times following contrast administration. TRICKS is particularly useful for imaging of vascular malformations.[28]

MRA is not susceptible to artifact from heavily calcified vessels, and imaging of tibial vessels may be superior to CTA.[29] Disadvantages of MRA compared to CTA include the time necessary for image acquisition, cost, and paucity of extravascular anatomic information. An implanted pacemaker or defibrillator has been considered a safety issue for MR imaging unless a patient has an MRI-conditional product. Recent data, however, suggest that risk may be minimal to nonexistent in patients with newer devices.[30]

The use of gadolinium-based contrast agents (GBCAs) for contrast-enhanced magnetic resonance imaging has been limited in the past decade due to concerns of the risk of nephrogenic systemic fibrosis (NSF) in patients with poor renal function. As patients with PAD (particularly those with CLI) often have concomitant chronic renal disease, the utilization of MRA in this patient population has been limited.

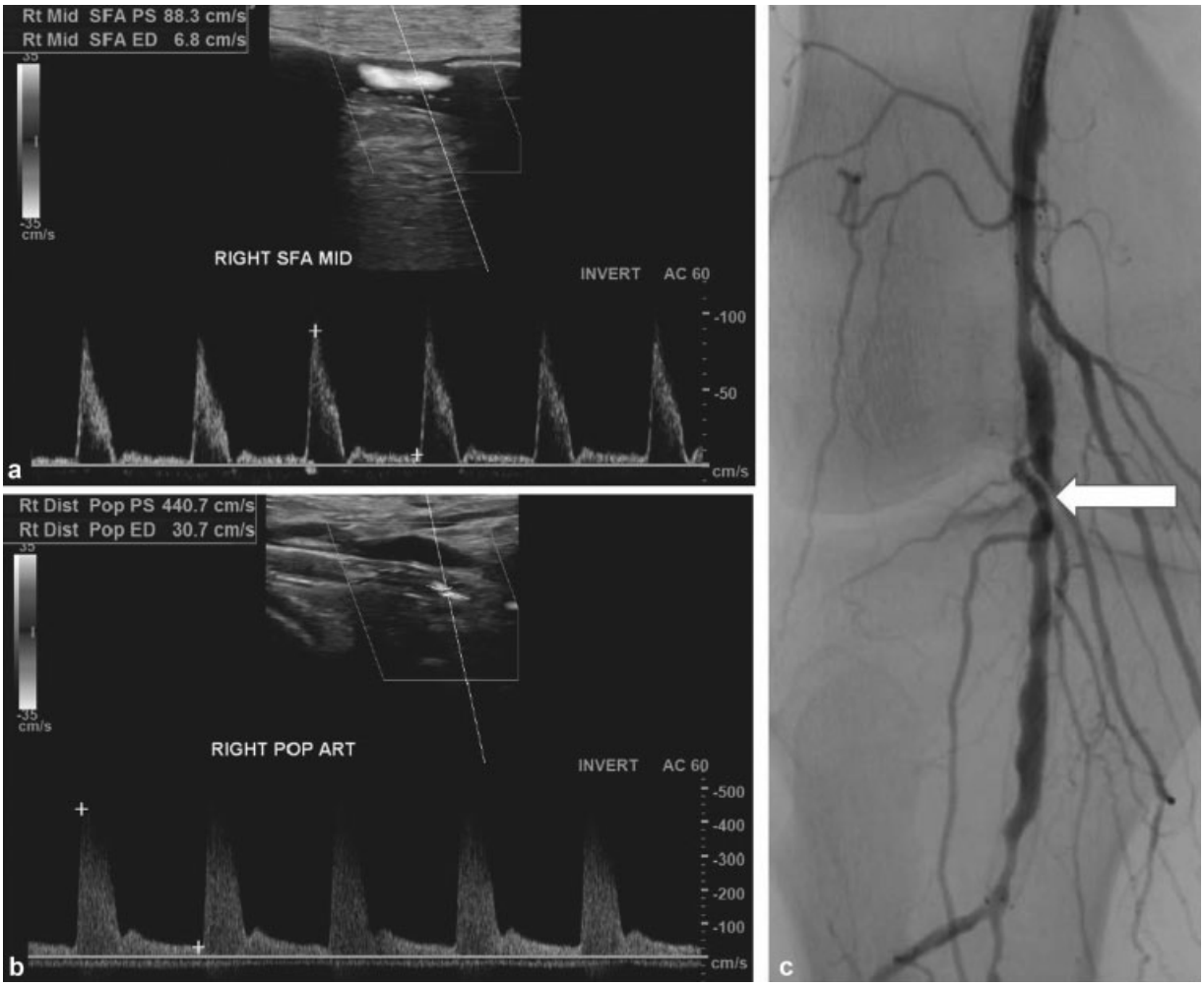

Fig. 8 Duplex image with arterial velocity measurement immediately proximal to the stent within the P2 and P3 segments of the popliteal artery (**a**). Duplex image of peak systolic velocity within the stented segment (**b**). PSVR is 5, consistent with a high-grade stenosis. Angiogram of the stented segment with the point of maximal stenosis obscured by a large crossing collateral (*white arrow*).

Recently, the American College of Radiology has deemed the risk of NSF from Group II GBCAs (►**Table 3**) to be sufficiently low or nonexistent that routine renal function testing is no longer recommended.[31] As such, it is considered safe to administer these agents to patients with poor renal function.

Table 3 Group II gadolinium-based contrast agents associated with few, if any, cases of nephrogenic systemic fibrosis

Agent (commercial name)	Manufacturer
Gadobenate dimeglumine (MultiHance)	Bracco Diagnostics (New Jersey)
Gadobutrol (Gadavist)	Bayer HealthCare Pharmaceuticals (Germany)
Gadoterate acid (Dotarem)	Guerbet (France)
Gadoteridol (ProHance)	Bracco Diagnostics (New Jersey)

Surveillance

Patients who have undergone endovascular or surgical revascularization should undergo routine surveillance that includes ABIs, PVRs, and duplex. There is no consensus that supports surveillance in patients with PAD who have not undergone prior intervention. The goal of surveillance is to detect a trend toward failure of the prior intervention that may not be apparent from clinical history and physical exam. There are little data and lack of consensus on surveillance intervals for endovascular patients. ACC/AHA 2017 guidelines recommend follow-up noninvasive 1 month following intervention and annually thereafter.[6] A more rigorous schedule in patients with infrainguinal stents (or stent grafts) is outlined in ►**Table 4**. A PSVR greater than 2.0 and less than 4.0 within or at the edge of a stent warrants closer surveillance at 3-month intervals until the lesion stabilizes or progresses to a threshold justifying reintervention. A PSVR $\geq$ 4 or peak systolic velocity $>$ 400 cm/second (consistent with $>$75% stenosis) should prompt reintervention due to threat of progression to thrombosis.[32]

Table 4 Surveillance schedule in patients with infrainguinal stents or stent grafts

Interval	Comments
1 mo	Following initial or any repeat intervention
3 mo	Following 1-mo exam or any exam with PSVR $\geq$2 or $\leq$4
6 mo	Following 3-mo exam with PSVR $\leq$2. Exam repeated every 6 mo unless PSVR $\geq$2
Repeat angiography	PSVR $\geq$ 4 or occlusion

Abbreviation: PSVR, peak systolic velocity ratio.

Conclusion

The components of a complete noninvasive arterial exam will vary by institution and vascular laboratory. Protocols may vary depending on whether a patient already carries a diagnosis of PAD, has had prior endovascular procedures or vascular surgery, or has tissue loss. Limitations may be placed by insurers that may, for example, cover only an ABI as an initial exam in a patient with claudication-type symptoms and no preexisting diagnosis of PAD. There is value in a multicomponent physiologic arterial test, as a positive exam yields considerable information regarding clinical severity of disease and the contribution of particular arterial segments to the patient's symptoms. A negative exam allows for the virtual exclusion of arterial insufficiency as an explanation for a patient's symptoms at relatively little cost. Morphologic arterial testing has value predominantly in patients with established disease for whom endovascular or surgical revascularization is planned.

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