A Practical Approach to Interpreting Lower Extremity Noninvasive Physiologic Studies

Thomas E. McCann, MD, Leslie M. Scoutt, MD,
Gowthaman Gunabushanam, MD*

KEYWORDS

• Peripheral arterial disease • Vascular laboratory • Ankle brachial index
• Segmental pressure measurements • Pulse volume recording

KEY POINTS

- Resting ankle brachial index (ABI) less than 0.90 is abnormal. Exercise ABI should be done in symptomatic patients with normal ABIs at rest.
- When rest and postexercise ABIs are normal, there is a low likelihood of an abnormal segmental pressure measurement or pulse volume recording (PVR).
- Toe brachial index (TBI) is especially helpful in patients in whom an ABI cannot be reliably obtained because of incompressibility of the calf arteries, often due to arteriolosclerotic mural calcifications. A TBI value less than 0.70 is abnormal.
- Segmental pressure measurements and pulse volume recording (PVR) studies help determine the level of obstruction. A pressure gradient more than 20 mm Hg between different levels, or between the 2 sides at the same level, is considered significant.
- PVR waveforms distal to a site of significant stenosis are characterized by loss of the dicrotic notch, smaller amplitude of the pulse wave, increased time-to-peak, a more rounded peak, and a downslope that is convex away from the baseline.

INTRODUCTION

Peripheral arterial disease (PAD) is an important manifestation of atherosclerosis, with an estimated age-adjusted prevalence of approximately 13% in people older than 50.[1] PAD affects men and women equally and is associated with an increased relative risk of death from cardiovascular causes that is approximately the same as in patients with a history of cardiovascular disease.[2] The major risk factors for PAD include age older than 40 years, smoking, diabetes, hyperlipidemia, hypertension, and hyperhomocysteinemia.[3–6] Because even asymptomatic individuals with PAD have an increased relative risk of death, screening of the at-risk population should be considered to identify the disease and begin treatment.[2] Early intervention with lipid-lowering therapy and antiplatelet drugs may delay disease progression and prevent premature death from cardiovascular causes.[7]

Ten percent to 30% of patients with PAD have symptoms of claudication.[8] Typical claudication is defined as pain in one or both legs on walking, primarily affecting the calves, that does not go

Department of Diagnostic Radiology, Yale University School of Medicine, 333 Cedar Street, PO Box 208042, New Haven, CT 06520, USA
* Corresponding author.
E-mail address: gowthaman.gunabushanam@yale.edu

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away with continued walking and is relieved by rest. However, more than 50% of patients found to have PAD by ankle brachial index screening (ABI) do not have typical claudication or evidence of limb ischemia at rest.[9] Initial evaluation of a patient suspected of having PAD should include a careful history to determine whether the patient has a history of walking impairment or if the patient experiences discomfort at rest. In patients with a history of walking impairment, it is important to quantify the degree of impairment by documenting the degree of exertion (eg, the distance walked) before the development of symptoms. A thorough physical examination is also performed to assess for abnormal pulses, skin discoloration, skin integrity, and ulcerations.[2] PAD can be further classified using the Rutherford Classification Index or the Fontaine classification system.[10,11] The Rutherford Classification Index classifies patients according to the degree of sensory loss, muscle weakness, and arterial and venous measurements in acute and chronic PAD. The Fontaine classification index classifies patients into 1 of 4 disease states, ranging from asymptomatic (stage 1) to tissue necrosis, death, and gangrene (stage 4).

In this review, we focus on the physiologic noninvasive vascular laboratory methods for screening and follow-up of patients with PAD, such as ABI (without or with exercise), toe brachial index (TBI), segmental pressure measurements, and pulse volume recordings (PVRs), which are considered the mainstays for identifying and quantifying the degree of PAD. Noninvasive imaging of PAD with ultrasound, computed tomography angiography (CTA), or magnetic resonance angiography (MRA) is not included in this review.[12–14]

ABI

The ABI is an objective test that can be used as a screening tool in the initial evaluation of PAD and in differentiating vascular etiology from neurologic and musculoskeletal causes of lower extremity pain, such as nerve root compression (for example by a herniated disc), spinal stenosis, or hip arthritis.[8] ABIs are also helpful in the evaluation of patients with PAD after medical or interventional treatment. Furthermore, ABIs have been validated with angiography and have been shown to provide prognostic information regarding limb survival, wound healing, and even all-cause patient survival.[15–17]

Technique

The patient should rest in the supine position for at least 10 minutes before obtaining the ABI. Blood pressure cuffs that are appropriately sized to the limb circumference are placed on both arms and lower calves. Systolic blood pressure (BP_S) is obtained with the aid of a handheld 5-MHz to 10 MHz continuous wave Doppler scanning probe. BP_S from the brachial arteries in the right and left upper extremities and the dorsalis pedis and posterior tibial arteries in the bilateral lower extremities are obtained. The ABI on each side is then calculated to 2 decimal places by dividing the higher of the dorsalis pedis and posterior tibial artery BP_S on that side by the higher of the BP_S in the arms (left or right arm).

Interpretation

A difference in BP_S in the brachial arteries by more than 20 mm Hg may be observed in patients with aortic dissection or stenosis in the subclavian or axillary arteries, and further workup for this should be considered (**Fig. 1**). Pulse wave reflection in healthy individuals causes the ankle systolic pressure to be approximately 10 to 15 mm Hg (10%) higher than the brachial arterial systolic pressure, causing the ABI to be normally greater than 1.00. An ABI less than 0.90 is considered abnormal. Patients with claudication typically have ABIs ranging from 0.41 to 0.89, and patients with critical leg ischemia have ABI values of 0.40 or less (**Table 1**). Patients with heavily calcified arteries, including arteriolosclerotic calcifications in the tunica media (often found in patients with diabetes and chronic renal failure and elderly individuals), may demonstrate falsely elevated systolic pressure measurements or inability to completely occlude the arterial flow (ie, noncompressible vessels). An ABI value of greater than 1.30 or a systolic pressure measurement higher than 250 mm Hg (some use 200 mm Hg) is an indeterminate result, usually secondary to a noncompressible, calcified vessel that prevents measurement of true arterial pressure. Although vessel wall calcification limits ABI and segmental pressure measurements and interpretation, it usually does not affect pulse volume recording waveforms.[18]

With serial ABI measurements following medical, surgical, or percutaneous interventional treatment, a decrease in ABI of 0.10 or greater when associated with a change in clinical status or an isolated decrease in ABI of 0.15 or greater is considered significant.[19]

Pitfalls and Limitations

An abnormal ABI in itself is not a reliable predictor of symptom magnitude, extent, or location of disease, and should be used primarily to identify patients with PAD, and interpreted in conjunction with segmental pressure measurements and PVR