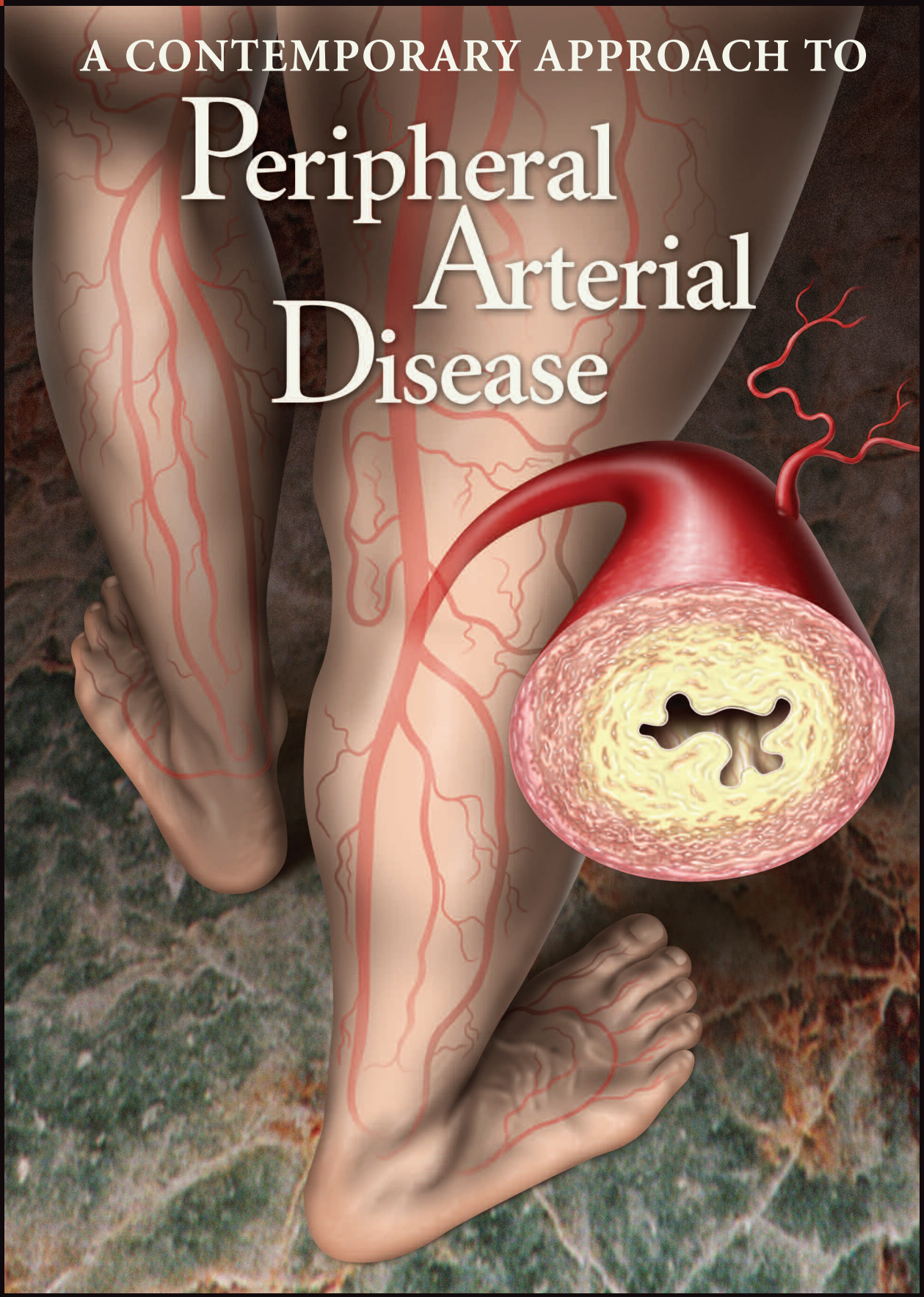
A CONTEMPORARY APPROACH TO Peripheral Arterial Disease



Peripheral arterial disease is often overlooked by primary care providers because early physical findings are subtle and patients may not complain of symptoms until it is in its advanced stages.

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Peripheral arterial disease (PAD) includes vasospastic disorders, aneurysms, and arterial inflammatory disorders, but the most common peripheral arterial disorder is caused by atherosclerosis. Peripheral arterial disease is common in the United States, affecting approximately 12% of all adults or about 8.4 million people.[1] However, it is often overlooked by primary care providers (PCPs) because early physical findings are subtle and patients may not complain of symptoms associated with the disease until it is in its advanced stages.

The prevalence of PAD increases with age. It is more common in men under the age of 65 years, but after age 65, both genders are equally affected. It can be a marker for atherosclerotic cardiovascular and cerebrovascular diseases (CVDs), and even patients without symptoms are at an increased risk for cardiovascular and cerebrovascular mortality.[2-4]

The Problem

Risk factors for developing PAD are similar to those for coronary artery disease (CAD): age, smoking history, and diabetes. Risk factor management is an important intervention, but unfortunately it is often initiated late in the disease process. Early identification and risk factor modification can reduce the morbidity associated with CAD, CVD, and PAD.

Patients with CAD or cerebrovascular disorders may have peripheral vascular symptoms as the first indication of any vascular disorder. Older adults with peripheral arterial occlusive disease may be asymptomatic or present with mild claudication. Intermittent claudication is a symptom that affects up to 20% of the population over 75 years of age. Sometimes this symptom is ignored by patients because it is perceived to be a consequence of aging. Inactive individuals may be asymptomatic, yet harbor risk factors for a stroke or myocardial infarction (MI). Peripheral arterial disease can be detected by a simple, noninvasive test, the ankle brachial index (ABI).

Risk Factors for Arterial Vascular Disease

The primary precursor to PAD, atherosclerosis, is the same for CAD and cerebral vascular disease and should be considered when assessing older patients. Identifying early onset markers for these diseases assists the healthcare provider in planning interventions to reduce modifiable risk factors. Smoking, obesity, hypertension, hyperlipidemia, diabetes, and sedentary lifestyle are all modifiable risk factors.

The single most significant risk factor for arterial disease is tobacco use. Tobacco accelerates atherosclerosis, damages the endothelium, and increases intravascular coagulation. Patients with PAD who continue to smoke have a 40% to 50% 5-year mortality rate.[5] Nicotine causes vasoconstriction, decreasing oxygen available for the tissues. Carbon monoxide further reduces the oxygen available for transport to the distal tissues. Claudication (from the Latin, claudicare, meaning "to limp") is the most frequent symptom associated with lower extremity PAD.

Intermittent claudication is three times more common among smokers than nonsmokers, and the diagnosis of PAD in smokers is made 10 years earlier than in nonsmokers.[6] Claudication is pain that commonly occurs in the calf muscle after walking, but it can occur in any leg muscle after use including the muscles of the thighs and buttocks. The pain, usually described as "cramping," is predictable and occurs at the same distance provided the ambulation pace and incline remain constant. Claudication occurs when there is decreased blood flow to the tissues during muscle use as in ambulation. Relief is obtained with cessation of muscle use. The patient can merely stand and as the arteries fill with blood, the pain disappears. Patients can be assured that walking can be resumed without fear of causing harm; however, it may pro-

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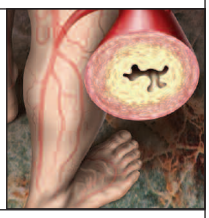
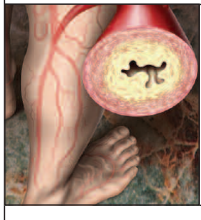


Illustration by Mark Lefkowitz

voke the pain. If the individual has to get into a position such as sitting or stretching to resolve the discomfort, another source is likely causing the pain. Claudication may be present, but it is not the only cause of pain.

One study found that patients with intermittent claudication who smoked 40 pack-years required surgical intervention three times more frequently than those who smoked less.[7] Patients who continue to smoke after bypass procedures are two times more likely to experience graft failure than nonsmokers. The number of cigarettes smoked increases the severity of PAD.[8] Conversely, smoking cessation reduces the incidence of intermittent claudication, and the risk of claudication for those who have quit smoking is the same as for nonsmokers after 1 year.[9]

Diabetes is a significant risk factor for PAD progression. Intermittent claudication is found twice as frequently in diabetic patients than nondiabetic patients. Atherosclerosis occurs in diabetics at a younger age, and arterial disease is associated with more than three-quarters of diabetic deaths.[10] Diabetic neuropathy increases the risk of foot ulcers in diabetics, and diabetes is associated with slow healing. More than one-half of diabetics with gangrenous foot lesions have PAD. Patients with diabetes and PAD have a 21% risk of major amputation compared with 3% in nondiabetic patients.[10]



Hypertension accelerates atherosclerosis and increases the mortality and morbidity of CAD and CVD.

Hypertension and PAD have a strong association. Hypertension accelerates atherosclerosis and increases the mortality and morbidity of CAD and CVD. Hypertension has been shown to increase risk of PAD 2.5 times for men and 3.9 times for women.[11] When three risk factors are present, the risk for PAD increases sixfold.[9]

PAD Symptoms

Olson and Treat-Jacobson concluded after a literature review that classic claudication symptoms may not be present in all patients with PAD.[10] Patients also may not recognize symptoms of lower-extremity PAD. One study found that up to 54% of those who reported being asymptomatic actually reported symptoms during supervised walking tests.[12] Often, older patients do not exercise to the extent they will elicit symptoms. Others describe atypical symptoms that the PCP may not recognize as PAD.

Rest pain is a symptom of insufficient arterial inflow to maintain tissue survival. The term "rest pain" is descriptive of the circumstances in which it occurs. Patients describe a pain across the dorsum of the foot that wakes them from sleep. In a horizontal sleeping position, the metabolic rate and blood pressure decrease. There is not enough pressure to push against a narrowed atherosclerotic arterial lumen or along collateral vessels to oxygenate the distal tissue. Patients describe getting out of bed, placing feet on the floor, or walking to gain some comfort. Gravity assists with arterial blood flow to the feet when standing. Some of these patients may even report sleeping in a chair sitting upright with legs and feet in a dependent position.

Vascular Clinical Assessment

Assessment of the peripheral vascular patient includes obtaining a comprehensive history. Pertinent questions include family history of diseases, disabilities, and sudden or unexplained death. Family deaths occurring at young ages may indicate a genetic trait for a coagulopathy leading to vascular occlusion.

The body can be divided into four sections with regard to assessment of the arterial system: the head, upper extremities, abdomen and chest, and lower extremities. A focused vascular history includes description of symptoms. Complaints related to the arteries that supply the head include an asymptomatic neck bruit found incidentally on physical examination or symptoms referenced to the anterior or posterior areas of the brain. Anterior (cerebral hemispheric) symptoms related to carotid arteries include amaurosis fugax (painless, monocular loss of vision), a fleeting blindness, which may be represented by a curtain descending over the visual field from top to bottom or from side to side, contralateral motor and sensory deficits, or dysphasias.

Posterior (cerebellar) symptoms related to the vertebral arteries include bilateral visual defects, diplopia, vertigo, ataxia, and dysphagia. Patients may report headache, confusion, or memory loss, although these are more general symptoms with many potential causes. A drop attack is a sudden fall to the ground without loss of consciousness and indicates vertebral insufficiency. Drop attacks may occur with neck rotation or hyperextension. Symptoms may be reproduced with range of motion maneuvers of the head.

Patients with arterial problems in the upper extremities often present with acute arterial symptoms including acute pain, exertional muscle fatigue, or distal digit pain of vasospastic disorders. The sequence of acute arterial ischemia can be described by the six Ps: pain, pallor, pulselessness, poikilothermia, paresthesias, paralysis. This list is progressive, and by the time paralysis is present, survival of the tissue is un-

Basic ABI Equipment

Appropriate Cuff Selection
- Cuff diameter 1.2 X limb diameter
  (too wide—falsely low pressure)
  (too narrow—falsely elevated pressure)

Cuff Placement
- Correct size brachial cuff, both arms
- Correct size ankle cuffs
- Place above medial malleolus
- Wrap cuffs snuggly

Doppler
- High frequency – 8 Mhz
  Majority
  Shallow penetration
- Lower frequency — 4 Mhz
  Obese/edema
  Deep penetration

Probe angle
- 45 to 60 degrees incident to vessel
  Not perpendicular to vessel

Risk Factors for Arterial Vascular Disease

1. Family history
2. Age
3. Race
4. Obesity
5. Medical comorbidities, disorders
   a. diabetes
   b. hypertension
   c. dyslipidemia
   d. coronary artery disease
   e. cerebral vascular disease
   f. clotting disorders
6. Smoking

likely. Acute arterial ischemia may be caused by an embolism from an atherosclerotic plaque, from an aneurysm, or from manipulation of an atherosclerotic artery during a procedure performed proximal to the embolic event. Upper extremity ischemia may be the result of a subclavian or axillary artery stenosis or occlusion, from extreme hypotension, or from vasospastic disorders or vasoconstrictor medications.

Arterial symptoms in the abdomen are associated with aneurysms and stenoses. Abdominal aortic aneurysms are more often asymptomatic than symptomatic. Most are found during general physical examinations or on radiographic or ultrasound images performed for other reasons. A leaking aneurysm may present with pain in the neck, abdomen, groin, or scrotum. Other arterial problems that occur in the abdomen are renal artery stenosis, which may cause hypertension, and mesenteric ischemia, which presents with intestinal angina 20 to 30 minutes after meals and is associated with food fear and subsequent weight loss.

Lower extremity arterial disease presentation ranges from no symptoms to disabling symptoms. Descriptions of arterial pain include aching, sharp throbbing, pins and needles, stabbing. The description of pain may represent disease severity. It ranges from mild claudication to significant rest pain. Patients who present with claudication experience the pain one joint level below the arterial stenosis. Claudication is relieved with cessation of activity, but rest pain requires placing the limb in a dependent position or taking pain medication. Other causes of pain that may be present and must be differentiated include neurospinal compression, musculoskeletal disease, neuritis, arthritis, sciatica, and causalgia or reflex sympathetic dystrophy. Any of these can be present in addition to PAD pain and must be considered in the differential diagnosis. Treatment is different for each entity. Patients should be asked to describe the symptoms, indicate what part of the leg is involved, relate how long the symptoms last, what distance initiates symptoms, and what provides relief.

Acute or chronic ischemia may occur when an embolism breaks off of a proximal diseased artery. The patient complains of sudden pain in distal tissues. The pain does not subside and the tissues become a blue-grey color with a temperature noticeably cooler than adjacent tissue or the contralateral limb. Acute arterial occlusion requires emergency intervention to preserve the limb.

Focused Physical Examination

Arterial perfusion is assessed by comparing blood pressure, pulses, capillary refill, temperature on palpation, color, and sensation bilaterally. Sensory differences, edema, scars, hair/nail description, and presence of ulcers or pregangrenous changes such as in a localized area compared to the surrounding tissue are included. Capillary refill is assessed by pressing the nail beds of the fingers and toes to create blanching. Normal color returns in less than 3 seconds. Sluggish return indicates reduced arterial pressure. Raising the ischemic limb causes it to become pale, and when it is lowered below heart level, it becomes dark with dependent rubor.

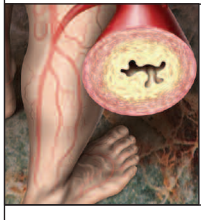
During a comprehensive examination, the eyes are examined with an ophthalmoscope. In the retina, evidence of atherosclerosis can be directly visualized in the arteries by the presence of glistening cholesterol emboli called Hollenhorst plaques.

Arterial examination and neurologic examination overlap. Arterial insufficiency may be the source of central or peripheral neurologic disturbances. A baseline neurologic assessment includes orientation, pupil size and reaction to

light, grasp strength and equality, movement of extremities, facial symmetry, tongue deviation, speech and swallowing, and gait. Arterial disease of the vessels supplying the brain and other parts of the nervous system may alter neurologic function. A neuromotor examination should be performed before and after any invasive procedure that involves insertion of needles into arteries near the neurovascular bundles, particularly in the groin or axillary areas. A hematoma in a confined space can quickly cause paresthesias due to nerve compression.

Inspect the legs for size, symmetry, and deformities. Assess color changes by elevating the feet 30 to 40 degrees above the hips. Observe for pallor on elevation and dependent rubor when the feet are put down. Trophic changes such as hair loss, thin skin, thick nails, tapering toes, and skin breakdown, occur only when there has been long standing ischemia.

Skin palpation for temperature should be performed on the back of both hands simultaneously. A difference of 10 degrees must be present for it to be noticed on palpation. A temperature demarcation will be felt below the site of a major occluded artery. If symmetric coolness is felt, it is likely due to vasoconstriction; whereas an asymmetric coolness indicates an arterial stenosis or occlusion on the cooler limb.



The best way to obtain objective information about the presence of PAD is to calculate the ABI.

Palpating pulses is a significant part of the physical examination. The carotid arteries are palpated separately to avoid excessive carotid sinus manipulation, which might cause reflex bradycardia. Palpating the common carotid does not insure patency of the internal carotid as the external carotid may be open, keeping the common carotid open as well. The radial pulses are palpated simultaneously to detect differences between them. Patency of the palmar arch vessels is assessed by performing the Allen test.

The abdomen is palpated for the aortic pulse and for detecting an abdominal aneurysm. The normal aorta is about the size of the individual's thumb. The examiner's hand is placed over the abdomen with thumb and fingers gently pushed to feel the sides of the aorta. An aorta greater than 4 to 5 cm diameter is suspect and should be further evaluated for abdominal aortic aneurysm. This assessment is ineffective in the obese patient.

Femoral and popliteal pulses are palpated and compared bilaterally. Many healthcare providers find palpating the popliteal pulses difficult due to location. A Doppler can be used to assess popliteal pulses. Popliteal pulse examination should be attempted to avoid missing a popliteal aneurysm.

At the foot, the dorsalis pedis and posterior tibial pulses are palpated. About 10% of the population with a normal arterial system will have an absent dorsalis pedis pulse.[9] Patients with arterial stenosis may have a palpable pulse at rest and nonpalpable with exercise. This is the "disappearing pulse" phenomenon[6], which is consistent with intermittent claudication. Pulses are assessed for presence, rate, regularity, and strength. The arterial pressure must be at least 70 mmHg to be palpated. Pulses are documented as "present" or "absent". There is no universal grading system for pulse intensity. Using a numerical system is subjective and not useful for subsequent assessments. It is more useful to document a variation between corresponding pulses in the same individual.

Bilateral brachial blood pressures are obtained in the initial vascular examination. A discrepancy of 20 mmHg or greater between the right and left brachial pressures indicates a proximal stenosis on the side of the lower pressure. The higher of the two systolic pressures is considered accurate and is used for systemic assessment and medication titration. The carotid, renal, and femoral arteries are auscultated for bruits. Blood flow through a normal vessel is laminar, but a stenosis causes turbulence distal to the stenosis. Turbulence creates the sound called a bruit. Bruits can be high or low pitched. There is no way to determine by auscultation the percent stenosis of the artery. Objective tests must be performed to assess the location and percent of stenosis. Palpation is also used to detect a thrill. This sensation is commonly felt over an arterial venous shunt created for hemodialysis. It may be felt over the femoral artery after a diagnostic procedure or intervention, which inadvertently created an arterial venous connection.

A Doppler is used to access signals in blood vessels. This was first demonstrated by the Austrian Christian Doppler who noted that, "The pitch of a sound rises as it nears and lowers as it moves away." This change in frequency is called the Doppler shift. The Doppler sends high frequency waves to the moving red blood cells. They in turn reflect a wave to the Doppler probe. A Doppler can pick up a signal with pressures as low as 20 mmHg, such as in the capillaries. This signal does not equal a palpable pulse. This assessment finding should be documented as a Doppler signal, not a Doppler pulse.

Diagnostic Screening

The best way to obtain objective information about the presence of PAD is to calculate the ABI. This is the ratio of the

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ankle systolic blood pressure to the brachial arterial systolic blood pressure.

The U.S. Preventive Services Task Force recommends against routine screening for PAD in asymptomatic adults in the general population because the prevalence is low. They state that counseling for smoking cessation and encouraging physically inactive patients to increase their exercise are interventions that are already offered to all patients to encourage healthy lifestyles and do not necessarily offer additional benefit for persons with screen-identified PAD.[14] Professional organizations, however, support screening for PAD in certain populations. The American Diabetes Association recommends annual screening for people with diabetes.[15] The American College of Cardiology (ACC)/American Heart Association Guidelines for the Management of Patients with Peripheral Arterial Disease (Lower Extremity, Renal, Mesenteric, and Abdominal Aortic) is the recent result of a collaborative effort among interested professional organizations and recommends using the ABI to evaluate patients who are at risk for PAD.[16]

ABI/TBI

The brachial systolic pressure is taken in both arms using an appropriately sized blood pressure cuff and a Doppler probe. With the patient in the supine position, arterial systolic blood pressures are measured in the posterior tibial artery and the dorsalis pedis artery of each foot. The ankle pressure is divided by the higher of the two brachial pressures to obtain the ABI. An ABI of < 0.90 indicates that PAD may be present. This assessment can be performed in the PCP's office. This simple office diagnostic maneuver will provide enough information to accurately identify a potential life- or limb-threatening diagnosis in its early stages. If PAD is suspected, the patient should be referred to a certified vascular laboratory to obtain a more comprehensive vascular arterial study, which includes sequential arterial pressures and waveforms as well as a repeat ABI and an arterial duplex scan. If acute arterial vascular occlusion is suspected, the patient should be sent to the emergency department for immediate evaluation and treatment.

Patients with diabetes may have elevated ABIs due to calcified noncompressible arterial walls. These patients should have a toe brachial index (TBI) performed to accurately assess the distal vascular pressure. Toe arteries are less likely to have calcified walls and reflect a more accurate distal arterial pressure. The PCP is not likely to have toe cuffs in the office; however, a certified vascular laboratory will have all the necessary equipment for this evaluation, including a qualified professional to interpret the findings.

The purpose of the ABI is to obtain a rapid estimation of the presence or absence of arterial occlusive disease. It is also used for postoperative evaluation of arterial grafts, outcomes of angioplasty and stenting, determination of the stability or progression of PAD, and to differentiate vascular from nonvascular disorders. The presence and severity of arterial diseases are confirmed by tests performed in the vascular laboratory. Noninvasive vascular laboratory tests include Doppler ultrasound, ABI, segmental pressures, and waveforms analysis. Normal arterial waveforms are triphasic, and as disease progresses in the artery, the third portion, then the second portion of the triphasic waveform are lost. As the vessel becomes less compliant, the first wave flattens. Exercise treadmill tests are performed in the vascular laboratory to assess and quantify claudication. An individual with claudication may have a near normal ABI in the supine position but have the ankle pressure drop significantly with exercise. Segmental blood pressures are used to determine the location of arterial lesions. A 20 mmHg pressure gradient from one segment to the next signifies an arterial lesion.

Obtaining an Ankle-Brachial Index

To obtain an ankle-brachial index (ABI) you will need a sphygmomanometer and a Doppler device. The procedure is performed as follows:

- Take the bilateral brachial blood pressure while the patient is supine. The higher of the two systolic pressures is used as the brachial pressure in the ratio.
- Place the blood pressure cuff on the leg just above the malleoli. Place the Doppler probe at a 45-degree angle to the dorsalis pedis or posterior tibial artery.
- Inflate the cuff until the Doppler signal is obliterated. With the Doppler probe over the artery, slowly deflate the cuff until the Doppler signal returns. Record the number as the ankle systolic pressure.
- Divide the ankle pressure by the higher of the two systolic pressures. The ratio obtained is the ABI.

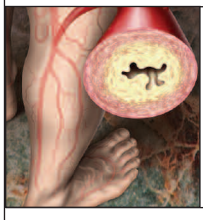
ABI Interpretation

ABI	Interpretation
1.0	Normal
0.75 to 0.90	Moderate disease
0.50 to 0.75	Severe disease
Less than 0.5	Rest pain or gangrene
Unreliable	Diabetes

Hypercoagulation Work-up

Sudden arterial occlusion in a young person without obvious atherosclerosis should raise suspicions of a coagulopathy. Women under 30 years of age with a familial

thrombosis, fetal thrombosis, or recurrent fetal loss should have a hypercoagulation work-up. This includes testing for Factor V Leiden, which is probably the most common cause of arterial thrombosis, prothrombin G20210A mutations, activated protein C resistance, prothrombin factor assay, proteins C, S, and antithrombin and homocysteine concentrations, fibrinogen, plasminogen, and heparin cofactor II. A consult with a hematology specialist is indicated if any of these tests are positive. It may be necessary for these patients to be on long-term anticoagulation medications.[16]



About one-quarter of those with intermittent claudication will significantly deteriorate, and this occurs within the first year after diagnosis.

Trajectory of PAD

Patients with leg pain can expect to have improvement or no change, an increase in claudication symptoms requiring an intervention to increase blood flow, or progress to tissue death requiring an amputation. Most patients with claudication do not progress to limiting claudication or limb-threatening disease. About one-quarter of those with intermittent claudication will significantly deteriorate, and this occurs within the first year after diagnosis.[9] Major amputation is rare (1% to 3.3% within 5 years) in this group of patients.[9] Patients with intermittent claudication who have heard that amputation is the natural outcome of PAD can be reassured by the statistics that amputation is far from inevitable.

Patients with PAD, whether symptomatic or asymptomatic, have generalized arterial disease and an increased risk of stroke, MI, and cardiovascular death. A low ABI (< 0.5) is associated with an increase in mortality because of its correlation with the severity of CAD.

Critical Limb Ischemia

The term critical limb ischemia (CLI) is used to describe patients who have rest pain, tissue loss, or gangrene. These patients require active intervention. Approximately 40% of patients who cannot undergo arterial reconstruction will lose a limb within 6 months and up to 20% will die.[9] These patients should undergo early evaluation and treatment and cardiac evaluation if surgery is anticipated. Patients at risk for CLI should have regular foot inspections. Patients with acute symptoms should be assessed immediately and treated by a vascular disease specialist.[16]

Treatment

The first line treatment is to modify risk factors[16] with the goal of helping patients gain control over the disease, decrease pain, improve exercise tolerance, and treat any problems associated with the disease. A plan for risk factor management includes a smoking cessation plan, hypertension and hyperlipidemia control, an exercise program, weight loss, and dietary management. Patients have to buy into this treatment plan because the burden to follow through is on them. Exercise is an important part of the treatment, but vascular rehabilitation programs are not readily accessible yet and many that are available do not receive insurance reimbursement. Patients should be encouraged to join a supervised exercise training program that provides 30 to 45 minutes of exercise three times per week. Patients who are unable to participate in supervised programs may create their own exercise programs; however, unsupervised programs have not been as effective in reducing risk factors. Pharmacologic intervention may be helpful in reducing symptoms for some patients with PAD. In general, all patients who have PAD require platelet inhibition to reduce cardiovascular morbidity and mortality. Daily aspirin (75 mg to 325 mg) is recommended to reduce the risk of MI, stroke, or vascular death. Clopidogrel (Plavix), (75 mg daily) is recommended as an alternative antiplatelet therapy to aspirin to reduce the risk of MI, stroke, or vascular death.[16] Antihypertensive and lipid-lowering drugs are recommended for treatment of hypertension and dyslipidemia in patients with PAD.[16] Cilostazol (100 mg twice daily) has been shown to increase walking distance and reduce symptoms of claudication, and is recommended (in the absence of heart failure) by the guidelines.[16]

Gaining control over the disease may include interventions to restore blood flow. Interventions include surgical operations and percutaneous procedures. Surgical interventions include bypass procedures using in situ vein grafts and artificial grafts. Percutaneous procedures, such as balloon angioplasty and stent placement, are increasing. Research is ongoing for new methods and new techniques to restore arterial flow.

Increasing Importance

It is becoming increasingly important for PCPs to be able to recognize PAD and to be familiar with the various treatments. Interpretation of diagnostic tests and treatment options is necessary for the PCP to help patients make appropriate healthcare decisions and to know when to recommend a specialist.

The NP is in a unique position to assess and intervene when a patient presents with peripheral vascular disease. A significant part of the intervention is patient education and risk management—areas in which NPs have expertise. Patients are becoming more aware of their own health needs. Providing them with information for self-care and monitoring will reduce the long-term effects of risk factors.

Patients with risk factors for PAD, CAD, and CVD, smoking history, leg symptoms, nonhealing wounds, age over 50 years with risk factors, or those who CAD, CVD, or chronic renal failure, are all at high risk for PAD. Up to three-quarters of patients with peripheral arterial vascular disease are asymptomatic. As a marker for other diseases, PAD should be assessed in the office with a screening ABI. Early risk factor management may prevent morbidity associated with PAD, CAD, and CVD.

Although there are new interventions in development to repair damaged arteries, nothing substitutes for life-long risk factor modification. NP

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AUTHOR DISCLOSURE

The author has disclosed that she has no significant relationship with or financial interests in any commercial companies that pertain to this education activity.

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