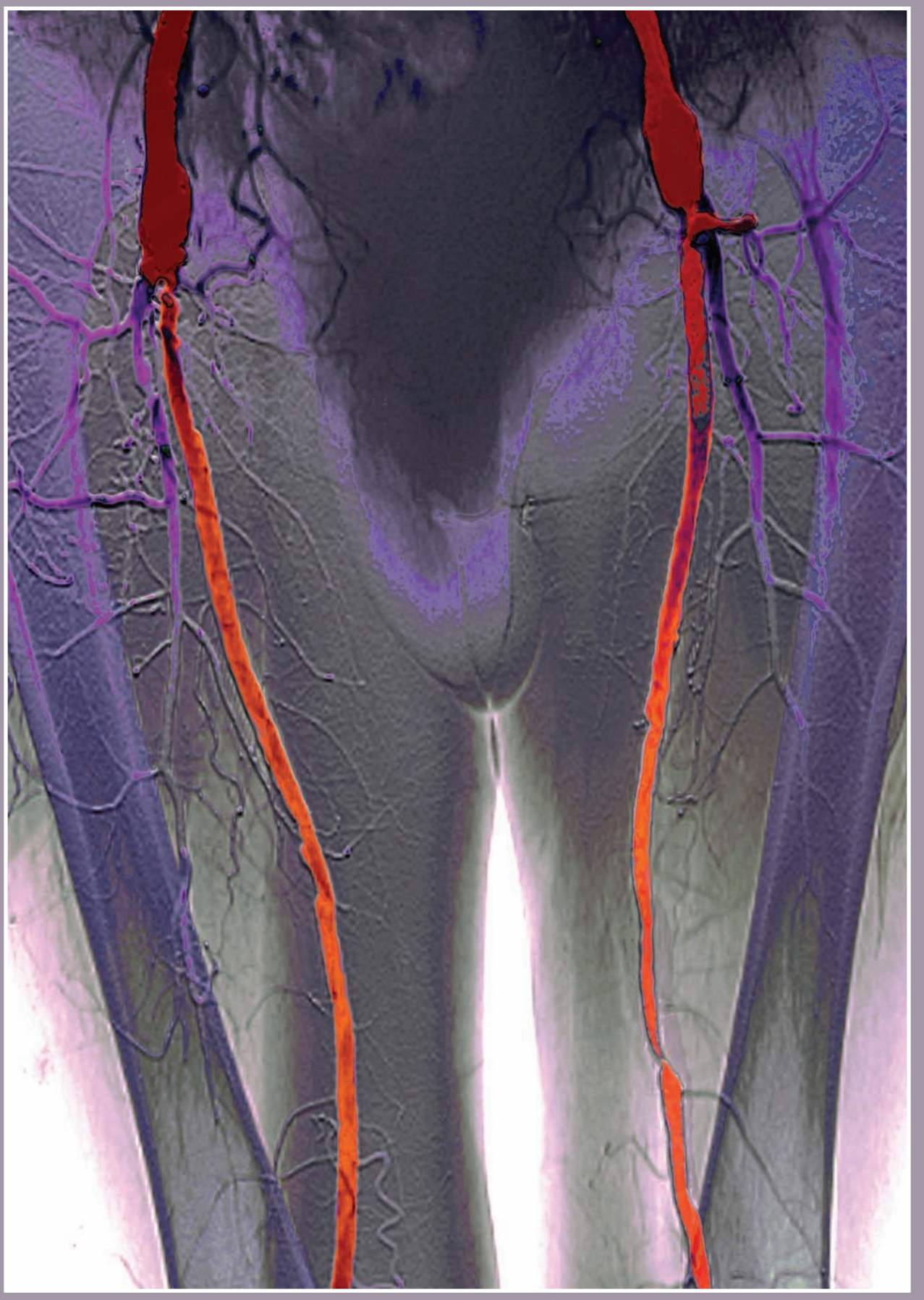

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A closer look at lower extremity peripheral arterial disease

By Linda Berti-Hearn, MSN, RN, CWOCN, and Brenda Elliott, PhD, RN, CNE

ACCORDING TO the CDC, approximately 8.5 million people in the United States have peripheral arterial disease (PAD).[1] PAD, which includes both upper and lower extremity disease, affects approximately 202 million people worldwide.[2] (See *Incidence and prevalence.*) A study by Slovut et al. found a significant gap between the American Heart Association (AHA)/American College of Cardiology (ACC) management guideline for patients with lower extremity PAD and clinical practice for this patient population compared with patients with coronary artery disease.[3] Assessing patients for PAD is very important because the prevalence of atherosclerosis in the coronary, carotid, and renal arteries is higher in patients with PAD than in those without PAD.[2]

The purpose of this article is to provide a review and update of lower extremity PAD, including a review of current management guidelines and nursing care. Acute limb ischemia and critical limb ischemia are beyond the scope of this article.

Normal anatomy and physiology

Arteries contain three concentric layers of tissue: the intima, the media,

Incidence and prevalence

PAD, also called peripheral vascular disease or arterial insufficiency, is a common cardiovascular disease.[2] It affects 32% of adults ages 40 to 70, and its prevalence increases to 40% in those age 80 or older.[1] Lower extremity PAD is the most common type of PAD.

Those of Hispanic or Black ethnicity tend to have a higher rate of PAD compared with non-Hispanic White people.[1,16] PAD affects both genders, but males are more likely to have more severe or symptomatic disease.[17] The risk of death from a cardiac event in patients with known PAD is much higher than in those without PAD, resulting in mortality that's two to three times higher for those with PAD.[17]

PDSN/Medical Images

and the adventitia. (See *Close up on blood vessel walls*.) Endothelial cells form a continuous lining called the endothelium for the entire vascular system. The endothelium plays an active role in controlling vascular function. (See *Endothelial cell properties and functions*.)

Vascular smooth muscle cells form the predominant cellular layer in the tunica media, thereby controlling vasoconstriction and vasodilation. These cells also synthesize collagen, elastin, and other extracellular matrix components important in both normal vascular repair and pathologic processes such as PAD.[4]

Pathophysiology

PAD refers to arterial disease affecting noncoronary arteries due to atherosclerosis causing vessel stenosis or occlusion.[5] Atherosclerosis results in calcification and plaque buildup in the artery walls.[6] As atherosclerosis progresses, arteries thicken and lumen diameter decreases, diminishing blood flow to tissues.

Blood flow depends on the patency of the artery, as well as sufficient perfusion pressure. When arterial blood flow is diminished from PAD, less oxygen and fewer nutrients reach local tissues leading to ischemia and necrosis. (See *Arteries of the leg*.)

The risk factors for PAD are similar to those that promote the development of coronary atherosclerosis.[2] (See *Risk factors for PAD*.)

Clinical assessment

Assessing the patient at risk for lower extremity PAD begins with obtaining a comprehensive health history and performing a physical assessment.[2] Signs and symptoms of PAD vary, depending on the number and location of arteries affected, degree of arterial stenosis, presence and extent of collateral circulation, and the patient's activity levels. Many patients don't have any complaints until arterial narrowing prevents the blood supply from satisfying metabolic requirements.[5] Generally, an artery has narrowed by 50% before the patient begins to develop signs and symptoms such as intermittent claudication, the classic symptom of lower extremity PAD.[7,8]

The AHA/ACC guideline on management defines claudication as "fatigue, discomfort, cramping, or pain (calf, thigh, or buttock) of vascular origin in the muscles of the lower extremities that's consistently induced by exercise and consistently relieved by rest (within 10 minutes)."[2]

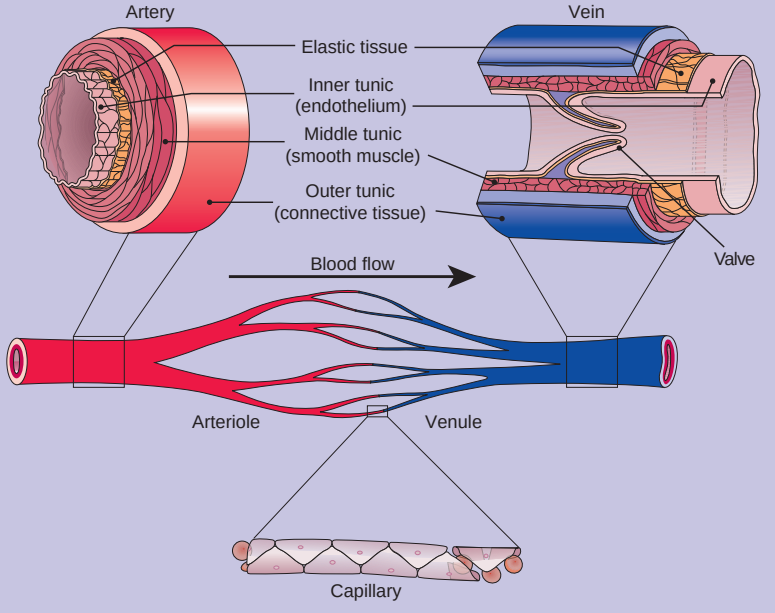
As PAD progresses, patients may experience positional or rest pain in the absence of walking or activity.[9] Positional pain occurs when the legs are elevated, and pain subsides when the legs are placed in a dependent position. Rest pain occurs in the absence of activity, most often at night while the patient is supine and sleeping.[9] Many patients find relief only when sitting up and dangling their legs over the side of the bed.

The vascular assessment of the lower extremities should be done with the patient supine and the legs fully exposed. Inspect both legs from the groin and buttocks to feet (including toes), noting any edema, pigmentation, rashes, or ulcerations. Note the skin color and texture, color of nail beds, and distribution of hair.[10]

Palpate and grade femoral, popliteal, dorsalis pedis, and posterior tibial pulses and note the temperature of the feet and legs. Auscultate for femoral bruits.

Patients with PAD may present with thin, shiny, atrophic skin, thickened and ridged toenails, and loss of hair on foot and toes.[7] Pulses are diminished or absent and the extremity may feel cool to the touch. Ulceration, if present, usually involves toes or points of trauma on the feet. (See *Recognizing advanced PAD*.)

Close up on blood vessel walls



Arterial walls are constructed to accommodate the high pulsing pressure of blood transported by the pumping heart, whereas venous walls are designed with valves that promote the return of blood and prevent backflow.

Source: Weber JR, Kelley JH. *Health Assessment in Nursing*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2013.

Endothelial cell properties and functions

Major properties	Associated functions/factors
Maintenance of a selectively permeable barrier	Controls the transfer of small and large molecules across the vessel wall
Regulation of thrombosis	Elaboration of pro- and antithrombotic molecules (von Willebrand factor, plasminogen activator) and antithrombotic molecules (prostacyclin, heparin-like molecules, plasminogen activator)
Modulation of blood flow and vascular reactivity	Elaboration of vasodilators (nitric oxide, prostacyclin) and vasoconstrictors (endothelins, angiotensin-converting enzyme)
Regulation of cell growth, particularly smooth muscle cells	Production of growth-stimulating factors (platelet-derived growth factor, hematopoietic colony-stimulating factor) and growth-inhibiting factors (heparin, transforming growth factor-beta)
Regulation of inflammatory/immune responses	Expression of adhesion molecules that regulate leukocyte migration and release of inflammatory and immune system mediators (for instance, interleukins, interferons)
Maintenance of the extracellular matrix	Synthesis of collagen, laminin, proteoglycans
Involvement in lipoprotein metabolism	Oxidation of very-low-density lipoprotein, low-density lipoprotein, cholesterol.

Source: Porth CM. *Essentials of Pathophysiology*. 3rd ed. Philadelphia, PA: Wolters Kluwer/Lippincott Williams and Wilkins; 2011.

Assess for postural color changes by raising both legs to about 60 degrees until maximal pallor of the feet develops, usually within 1 minute. Then ask the patient to sit and dangle his or her legs. Compare both feet, noting the time required for color to return to normal. Color changes may be difficult to see in darker-skinned patients, so inspect the soles of the feet for color changes. Elevation pallor and dependent rubor suggest PAD.[10]

Diagnostic studies

History and physical assessment findings suggesting PAD require diagnostic testing.[2] Noninvasive assessment includes calculating the ankle-brachial index (ABI), which is the initial diagnostic test for PAD and may be the only test required to establish the diagnosis and institute treatment.[2] The ABI can be calculated at the bedside or in the patient's home by a nurse using a handheld Doppler and a BP cuff. For accurate results, the patient should rest for 15 to 30 minutes before measuring pressures. Measure arm systolic BPs (brachial arteries) and ankle systolic BPs (dorsalis pedis and posterior tibial arteries) in the supine position by using a Doppler device. Calculate the ABI of each leg by dividing the higher of the dorsalis pedis pressure or posterior tibial pressure by the higher of the right or left arm pressure.[11]

Resting ABI results should be reported as abnormal (ABI ≤ 0.90), borderline (ABI, 0.91-0.99), normal (ABI, 1.00-1.40), or noncompressible (ABI > 1.40).[2]

Results can be falsely elevated in patients with diabetes and in older adults because of calcification in the arteries. Depending on the patient's clinical presentation and the ABI results, additional vascular studies may be indicated. These could include toe-brachial index (TBI) when the ABI is greater than 1.40, exercise treadmill ABI testing, or additional perfusion assessment measures such as transcutaneous oxygen pressure or skin perfusion pressure.[2]

Risk factors for PAD

The ACC/AHA guidelines on PAD identified the following groups with a higher prevalence of PAD:

- age 70 or older
- ages 50 to 69 with a history of smoking or diabetes
- ages 40 to 49 with diabetes and at least one other risk factor for atherosclerosis
- leg symptoms suggesting claudication with exertion, or ischemic pain at rest
- abnormal lower extremity pulse examination
- known atherosclerosis at other sites (for instance, coronary, carotid, or renal artery disease)
- other risk factors, including a family history of atherosclerosis, smoking, hypertension, hyperlipidemia, and homocysteinemia.

Source: Neschis DG, Golden MA. Clinical features and diagnosis of lower extremity peripheral arterial disease. 2017. www.uptodate.com.

Anatomic imaging studies, such as duplex ultrasonography, magnetic resonance angiography, computed tomography angiography, and invasive angiography, are reserved for patients who are highly symptomatic and those in whom revascularization is being considered.[2]

Management

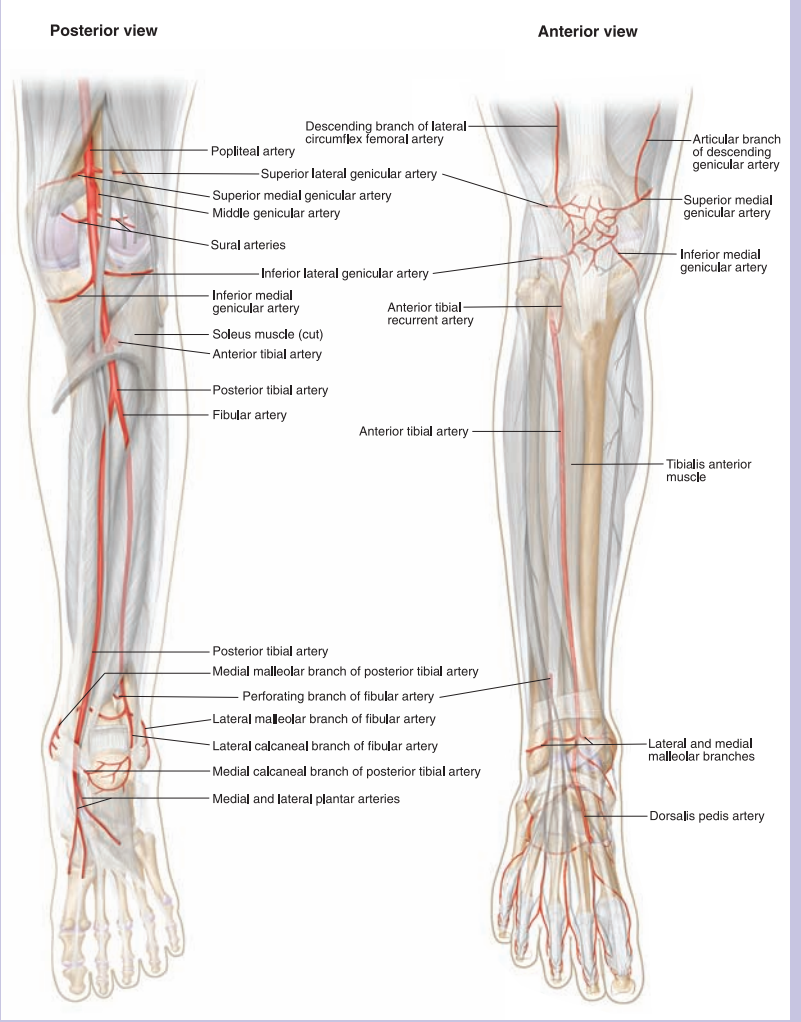
Patients with lower extremity PAD should receive a comprehensive treatment plan that includes a structured exercise program with recommended lifestyle modifications to reduce ischemic events and improve functional status.[2] Relieving signs and symptoms and preserving tissue are the primary goals for treating PAD.[7]

Medical management includes risk factor modification and drug therapy. Smoking cessation is essential for slowing PAD progression. Help patients develop a plan to quit, which should include pharmacotherapy such as bupropion, and/or referral to a smoking cessation program.[2]

A structured exercise program in combination with smoking cessation is the primary nonpharmacologic therapy for intermittent claudication. Walking is the most effective exercise. Intermittent periods of walking to moderate-to-maximum claudication, alternating with periods of rest, are performed for a minimum of 30 minutes/session, at least three times a week, for a minimum of 12 weeks.[2]

Pharmacotherapy, which includes antiplatelet and statin agents, is individualized to additional risk factors, such as diabetes mellitus or hypertension. Cilostazol is effective for treating leg symptoms and walking impairment due to claudication. Adverse reactions include headache, diarrhea, dizziness, and palpitations.[2]

Arteries of the leg



Source: Tank PW, Gest TR. *Lippincott Williams & Wilkins Atlas of Anatomy.* Baltimore, MD: Wolters Kluwer Health; 2009.

Minimizing tissue loss

Patients with PAD are at increased risk for infection and amputation.[6] Preventing lower extremity wounds through patient education, foot inspection, and prompt recognition and treatment of pressure injuries and/or foot infection is important to minimize tissue loss among patients with PAD. Minor tissue loss includes nonhealing ulcer and focal gangrene with diffuse pedal ischemia. Major tissue loss includes tissue loss above the transmetatarsal level and loss of foot function.[2]

Arterial wounds may erupt suddenly or can result from trauma to the feet or toes, such as from stubbing a toe or developing a blister from a constricting shoe. Many patients with PAD as their primary etiology have arterial ulcers on the toes, around the nail bed of a toe, or in the web spaces.[9] If PAD is a contributing factor, then other areas exposed to prolonged pressure or trauma, such as heels and malleoli, are also susceptible.

Arterial wounds are often small and appear punched out with well-defined smooth wound edges.[12] The wound is pale with little or no granulation tissue and may be necrotic or contain eschar. Arterial wounds

have minimal drainage and are very painful; pain worsens with walking or elevation.

It's important to assess for complications such as infection, gangrene, or osteomyelitis. Infected wounds and cellulitis in patients with PAD are potentially limb-threatening complications that require immediate referral for evaluation and treatment.[2,12]

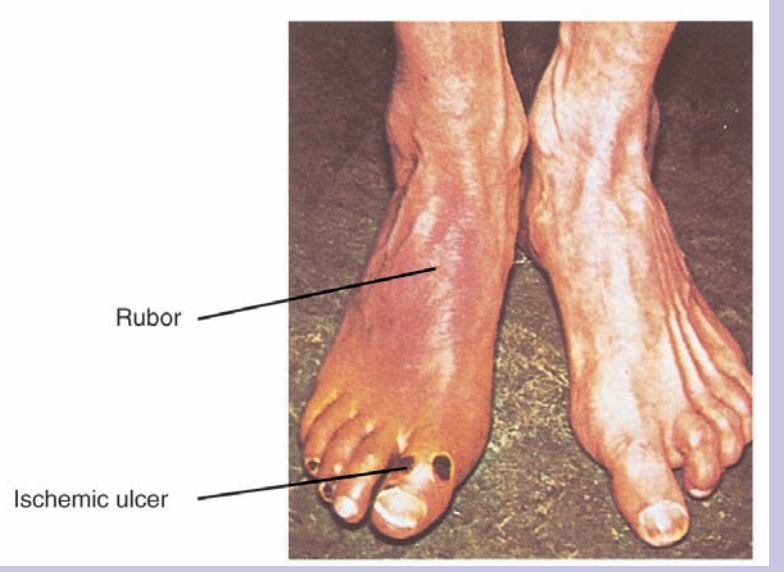
Managing patients with PAD, especially with a nonhealing wound, requires a multidisciplinary team to coordinate revascularization and wound care.[2] Treatment goals include treating the underlying cause, preventing trauma and infection, and educating the patient.

Revascularization's role in managing claudication

For patients with claudication, revascularization can improve symptoms, function, and quality of life. Revascularization is a reasonable option for patients with PAD whose lifestyle is affected by claudication despite receiving management and therapy according to the guidelines, including structured exercise therapy. What is lifestyle-limiting claudication? It depends on the patient, but it can be described as impaired activities of daily living or impaired vocational or recreational activities, or any combination of these, due to claudication. Revascularization approaches should be individualized for each patient.[2]

Endovascular treatment for claudication depends on the characteristics of the lesion, such as its location, length, and degree of calcification, and the experience of the interventionist. Procedures include angioplasty (such as the use of balloon dilation, cutting balloons, or drug-coated balloons), stents (including covered stents and drug-eluting stents), and atherectomy.[2]

Recognizing advanced PAD



Pain	Intermittent claudication, progressing to pain at rest
Mechanism	Tissue ischemia
Pulses	Decreased or absent
Color	Pale, especially on elevation; dusky red on dependency
Temperature	Cool
Edema	Absent or mild; may develop as the patient tries to relieve rest pain by lowering the leg
Skin changes	Trophic changes: thin, shiny, atrophic skin; loss of hair over the foot and toes; nails thickened and ridged
Ulceration	If present, involves toes or points of trauma on feet
Gangrene	May develop

Source: Bickley LS. *Bates' Guide to Physical Examination and History-Taking.* 10th ed. Philadelphia, PA: Wolters Kluwer/Lippincott Williams and Wilkins; 2009.

Surgical procedures can be used instead of endovascular procedures for revascularization for appropriate patients when the advantages of surgery outweigh those of endovascular procedures and when patients don't have risk factors that would rule out surgery.[2]

When claudication occurs, stenosis or occlusion is most commonly found in the superficial femoral and proximal popliteal arteries. For this reason, femoral–popliteal bypass is often the surgical choice for patients with claudication.[2]

Postrevascularization nursing care

To detect complications after a revascularization procedure, monitor patients closely. Assessment of the lower extremities includes color, temperature, capillary refill time, presence and intensity of pulses, pain intensity, and motor and sensory function. A cool extremity may indicate an occluded graft or stent.

Monitor vital signs frequently. Hypotension and tachycardia may be signs of hypovolemia, which

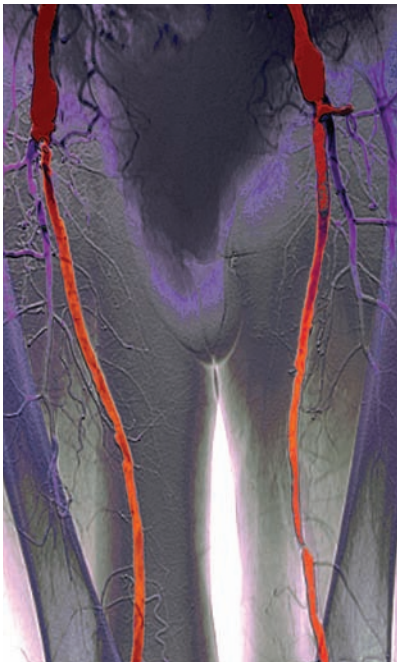
can increase the risk of thrombus formation.[13] Also monitor the catheter site or surgical site for signs of bleeding and infection. Notify the provider immediately about any changes in the patient's status, such as loss of palpable pulses.

Additional patient education

Teach patients that PAD is a chronic disease with frequent and long-term complications. Teach them they can help improve the blood supply to their legs and feet by avoiding elevating or crossing their legs.[12] Patients should also refrain from wearing constrictive clothing to prevent vasoconstriction and edema.

Educate patients to protect their lower extremities from injuries; for example, when getting into and out of cars or when walking past a coffee table, because they're at risk for poor wound healing should an injury occur. Instruct them to inspect their feet and legs daily, keep them clean and dry, and moisturize the skin to prevent cracks and fissures that can lead to infection. To prevent fungal growth, advise them to avoid applying moisturizer between their toes.

Assess patients' feet and evaluate their footwear; wearing properly fitting shoes can help prevent blisters and calluses. Patients with diabetes, in particular, should never walk barefoot. Immobilized patients should be positioned with their heels elevated to prevent pressure injuries.



For patients with claudication, revascularization can improve symptoms, function, and quality of life.

Encourage patients to walk or engage in other physical activity as recommended by the healthcare provider. Evaluate barriers to activity, such as intermittent claudication, and develop a plan of care with patients to help decrease or eliminate these barriers.[8,14]

Nursing care of patients with PAD or at risk for PAD should include education about tobacco and smoking cessation and managing hypertension, diabetes, and dyslipidemia. This includes taking medications as prescribed, ensuring adequate nutrition and protein intake to maintain intact skin or promote wound healing, and tight glycemic control.

Assessing the needs of the spouse or caregiver of patients affected by this disease is also important in providing holistic nursing care.[15] Living with any long-term illness or pain can have many short-term and long-term effects for both patient and family, leading to decreased quality of life, feelings of loss of control, and social isolation.

Ongoing care

PAD is a lifelong, chronic, progressive medical disorder.[2] Advise patients that they can help slow its progression and manage symptoms with regular visits to a podiatrist, primary healthcare provider, and/or vascular surgeon. The healthcare team should assess ABIs at least yearly, monitor cardiovascular and cerebrovascular status closely, adjust medications as indicated, and

Select resources for nurses

Resource	Website
• Wound, Ostomy and Continence Nurses Society – Venous, Arterial, and Neuropathic Lower-Extremity Wounds: Clinical Resource Guide	http://c.ymcdn.com/sites/www.wocn.org/resource/resmgr/publications/Venous_Arterial__Neuropathic.pdf
• Society for Vascular Surgery – Peripheral Arterial Disease Resources	https://vascular.org/news-advocacy/peripheral-arterial-disease-resources
• 2016 AHA/ACC Guideline on the Management of Patients With Lower Extremity Peripheral Artery Disease: Executive Summary	http://circ.ahajournals.org/content/early/2016/11/11/CIR.0000000000000470
• American Heart Association – Symptoms and Diagnosis of PAD	www.heart.org/HEARTORG/Conditions/VascularHealth/PeripheralArteryDisease/Symptoms-and-Diagnosis-of-PAD_UCM_301306_Article.jsp#.WP1Vh9LyuM8

reinforce patient education. For additional resources in the care of patients with PAD, see *Select resources for nurses*. ■

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