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MANAGEMENT OF LOWER EXTREMITY PERIPHERAL ARTERIAL DISEASE

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Peripheral arterial disease (PAD), a manifestation of systemic atherosclerosis, is a significant health problem affecting 8 million people in the United States.[1] Although the term PAD is sometimes inclusive of all peripheral arteries, this article will specifically focus on the arteries of the lower extremities. PAD is characterized by a partial or complete failure of the arterial system to deliver oxygenated blood to peripheral tissue. Atherosclerosis is by far the most common etiology of PAD. However, several other processes can lead to the clinical syndrome like arterial entrapment, thrombus, adventitial cyst, embolism, fibromuscular dysplasia, dissection, trauma, vasculitis and vasospasm.

The ankle brachial blood pressure index (ABI), defined as the systolic blood pressure measured at the ankle divided by the systolic blood pressure measured in the arm during supine rest, is the most widely used quantitative measure to determine the presence and severity of PAD. An abnormal ABI value of ≤ 0.90 is generally considered to be the best reference standard of identifying PAD, whereas normal values range between 0.9 to 1.3. The prevalence of PAD is 16 percent in the general population older than 55 years of age when an ABI value of ≤0.90 is used as the criterion of PAD. In the Edinburgh Artery Study,[2] approximately 20% of the men and women ages 55 to 74 years of age had an ABI ≤0.90, and thus were diagnosed as having PAD. The prevalence of PAD increases with age, and at all ages is higher in men than in women. At ages 65 to 69 years, the prevalence of PAD in men from the Cardiovascular Health Study [3] was approximately 7 percent, and approximately 5 percent in women. In subjects age 85 years and older, the prevalence was 23 percent in men and 21 percent in women.

CARDIOVASCULAR DISEASE RISK FACTORS OF PAD

PAD and coronary artery disease (CAD) share risk factors. In addition to age and male sex, risk factors for PAD include smoking,[4, 5] hypercholesterolemia,[6] diabetes,[7] hypertension,[8] chronic kidney disease,[9] hyperhomocystinemia,[10] elevated fibrinogen concentration,[11] having a family history of premature atherosclerosis (suggesting that genetic factors may influence the development of PAD), and being non-white.[1, 12] Although PAD can be seen in the absence of clinical CAD, asymptomatic CAD is frequently present in patients with PAD.[13] Every patient presenting with PAD should be considered to have CAD until proven otherwise. Evaluation and treatment for PAD should include evaluation and control of CAD risk factors.

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Patients with PAD are at increased risk for coronary heart disease, coronary vascular disease, and all-cause mortality. This risk is independent of traditional risk factors, including age, sex, smoking, systolic blood pressure, plasma lipids, fasting glucose, body mass index, and preexisting clinical cardiovascular disease. Risk of mortality as a consequence of coronary heart disease and cardiovascular disease is 3 to 6 times higher in subjects with PAD than in subjects without PAD, even after accounting for traditional risk factors.

PRESENTATION

Clinical PAD has been recognized since as early as 1831 and the disease spectrum varies from asymptomatic PAD to gangrene and limb ischemia requiring amputation. Two schemes, both based on symptoms and clinical measures, are commonly used to classify the severity of PAD (Tables 1 and 2). In the early stages of PAD, the reduction in blood flow and ABI does not result in any noticeable symptoms (asymptomatic PAD) and is defined as stage I according to the Fontaine classification system,[14] and grade I, category 0 according to the Rutherford classification[14] system. As PAD progresses, ischemic pain in the leg musculature occurs when patients walk (intermittent claudication), and is classified as either Fontaine stage II-a or II-b, or Rutherford grade I, category 1, 2, or 3, depending on the walking distance and the change in ABI following walking. In more advanced stages of disease, ABI is reduced to such an extent that pain is experienced even while at rest, classified as either Fontaine stage III or Rutherford grade II, category 4. Further progression of the disease leads to ischemic ulcerations on the lower extremities, gangrene, and tissue loss, classified as either Fontaine stage IV or Rutherford grade II, category 5, or grade III, category 6. Patients in these categories have critical limb-threatening ischemia in which the ischemia endangers part or all of the lower extremity.[14] These patients are candidates for aggressive limb salvage interventions such as percutaneous transluminal angioplasty or bypass surgery.

The presentation of PAD varies widely among patients. PAD can be present without any clinical signs, in which case, diagnosis of PAD can only be made with laboratory tests. With mild disease, the peripheral pulses can be decreased and intermittent claudication may occur while ambulating. With more advanced disease there may be an audible bruit or the distal pulses may be absent. The extremity may be pale or cyanotic at rest, upon raising the leg, or with exercise. The skin of the extremity can be cool, smooth, and shiny with hair loss, and nails can be thickened. Dangling the legs following elevating the extremity can lead to delayed return of color to the skin (usual time about 10 seconds), delayed filling of the veins of the feet and ankles (normal about 15 seconds), and the development of a dusky rubor in the legs. The patient may complain of a sensation of cold or numbness in the foot or toes. Patients with more severe disease will often complain of pain when the extremity is at rest. This pain will often occur at night and will be relieved by dangling the leg over the edge of the bed. Patients with the most severe disease will present with ulcers on their extremity or frank gangrene.

PAD has important implications for physical function. PAD severity (accessed by ABI) is directly related to 6-minute walk distance,[15] free-living energy expenditure, and steps taken per day. Furthermore, PAD is not a static disease. Progression from intermittent claudication to rest pain or gangrene can occur in anywhere from 2 to 7 percent of patients per year. Furthermore, PAD is associated with increased risk for mortality, and this risk becomes greater as the severity of PAD increases.[16]

EVALUATION

A number of non-invasive tests have been used to screen for and to evaluate the extent of PAD. The most basic clinical test is palpation and auscultation of the peripheral pulses. Little is known about the sensitivity of pulse palpation for the diagnosis of PAD.[17] A recent systematic literature review concluded that physical examination findings are not sufficient alone and

must be considered in the context of risk factors for atherosclerosis to improve diagnostic accuracy.[18] In situations in which direct examination of subjects is not feasible, such as epidemiologic studies, the San Diego Claudication Questionnaire,[19] which is a version of the Rose Questionnaire,[20] is often used to screen subjects for PAD. Using these questionnaires, PAD is defined as leg pain associated with walking that goes away with rest. The sensitivity of the Rose Questionnaire has been reported as being anywhere from 10 to 50 percent.

Noninvasive Vascular Tests

The most common measure to assess the presence and severity of PAD is the ABI. PAD is typically defined by an ABI value. The sensitivity of using the ABI cut point of ≤ 0.90 is greater than 90%.[21] Generally, a patient whose ABI is < 0.8 will be symptomatic with intermittent claudication during exercise, and a patient whose ABI is < 0.30 will generally complain of pain at rest. Very high ABI's > 1.3 are considered invalid because they do not reflect the true ankle blood pressure. Rather, it is caused by arteries that have become calcified or non-compressible, termed calcific medial sclerosis, which is often observed in patients with diabetes.

Segmental systolic blood pressure measures in the brachial, upper thigh, lower thigh, and ankle locations have been used to access the extent of PAD.[22] Additional non-invasive tests for PAD include Doppler ultrasonography[23] (ie, measurement of blood flow velocity), plethysmography (pressure-wave tracing),[22] and measurement of post-occlusive reactive hyperemia (PORH).[24] PORH is performed by occluding arterial flow by inflating a blood pressure cuff above systolic pressure at the level of the upper thigh or knee for 3 minutes, followed by measurement of the systolic blood pressure at the ankle or calf blood flow within seconds after releasing the occlusion. When compared to patients without vascular disease, patients with PAD will demonstrate a lower post-occlusive ABI and a delayed return to pre-occlusion pressures. The sensitivity of the post-occlusive ABI is > 95%. For individuals who present with classic claudication and who have ABI values in the borderline-to-normal range (0.91 to 1.30), or who have ABI values above normal (greater than 1.30), alternative diagnostic strategies should be used to confirm the diagnosis of lower extremity PAD. These alternative methods include the toe-brachial index,[25, 26] ABI after treadmill exercise,[27] segmental systolic blood pressures, duplex ultrasound, Computer tomographic angiogram, and magnetic resonance angiogram.

Treadmill Testing

The primary effect of PAD has on acute exercise is the development of claudication pain in the leg musculature as a result of insufficient blood flow. As a result, claudication and peripheral hemodynamic measurements obtained from a treadmill test are the primary criteria to assess the effectiveness of an exercise program. The specific claudication variables that are measured to assess the functional severity of PAD include the distances (or times) to onset and to maximal claudication pain. ABI measurements obtained before and after the treadmill test, in addition to claudication measurements, provide a more objective assessment of disease severity.

The primary objective of a treadmill test for patients with PAD is to obtain reliable measures of 1) the rate of claudication pain development, 2) the ABI response to exercise, and 3) the presence of coexisting coronary heart disease. The test should be a progressive test with gradual increments in grade. By having a test with small increases in exercise intensity, claudication distances of patients can be stratified according to disease severity. A highly reliable treadmill tests for patients with PAD uses a constant walking speed of 2 mph and gradual increases in grade of 2 percent every 2 minutes beginning at 0 percent grade. By using this treadmill protocol, typical distances to onset of pain and to maximal pain are approximately 170 meters (3 minutes) and 360 meters (6.5 minutes), respectively. Measurement of the ABI immediately

after a treadmill exercise stress test can help diagnose PAD in difficult cases, as well as determine the extent of impairment of the peripheral circulation. Exercise increases systemic blood pressure (ie, the brachial pressure), while pressure distal to an arterial lesion in the lower extremity falls with exercise as a consequence of dilation of secondary arterioles. As a result, ABI typically drops from a resting value of 0.7 to approximately 0.3 immediately following the treadmill test. The sensitivity of ABI measured after treadmill walking is > 95%.[27]

Gas-exchange measures during the treadmill test show that PAD patients with intermittent claudication have peak oxygen consumption values in the range of 12–15 mL $\cdot$ kg^{-1} $\cdot$ min^{-1} which is approximately 50% of age-matched controls. Favorable changes following a program of exercise rehabilitation should include greater walking distances covered before the occurrence of the onset and maximal claudication pain, an increase in peak oxygen consumption, and possibly a blunted drop in ABI and a faster rate of recovery in ABI to the resting baseline value.

MEDICAL MANAGEMENT

All patients with PAD need aggressive risk factor modification including cessation of cigarette smoking, control of diabetes, hypertension, and hypercholesterolemia, along with dietary restrictions aimed at reducing low density lipoprotein cholesterol and obesity. In addition to risk factor modification, treatment options are geared towards medical therapy which includes antiplatelet agents like aspirin and clopidogrel,[28] vasodilators like cilastazol and pentoxyfylline, exercise rehabilitation, surgical therapy like endovascular interventions, or surgical bypass. Management of patients through medication and exercise should be attempted prior to the more aggressive approaches of endovascular intervention or surgical bypass. Our primary focus will be on exercise therapy aimed at reducing functional disability and increasing walking distance.

Hypercholesterolemia Management

Statins should be prescribed to all patients with PAD irrespective of the presence of CAD. Recent PAD guidelines recommend target LDL concentration to be <100 mg/dl in all PAD patients, with an additional target of <70mg/dL in patients with very high risk of ischemic events. Several studies have reported improvement in walking performance, ABI, and symptoms of claudication after treatment with simvistatin in patients with hypercholesterolemia,[29, 30] and improvement in pain free walking distance.[31]

Hypertension Management

Control of hypertension is critical for prevention of stroke, myocardial infarction (MI) and congestive heart failure (CHF). Antihypertensive therapy may decrease limb perfusion pressure and potentially exacerbate symptoms of claudication or chronic limb ischemia (CLI). However, most patients are able to tolerate therapy without worsening of symptoms, and contrary to prior belief, β-blockers do not worsen intermittent claudication in PAD patients.[32] Recent PAD guidelines recommend using ACE inhibitors in symptomatic PAD patients based on the Heart Outcomes Prevention Evaluation (HOPE) study where ramipril reduced the risk of MI, stroke, or vascular death in patients with PAD by approximately 25%.[33]

Vasodilators

Pharmacologic therapy for intermittent claudication in the United States is limited to pentoxifylline and cilostazol. Pentoxifylline,[34, 35] which has a hemorheologic effect by improving the flexibility of red blood cell membranes and byreducing platelet aggregration, was first studied in the United States in 1982. It was found to increase the distance to onset of claudication pain by 45% and the distance to maximal pain by 32% following 24 weeks of

treatment. These were significantly greater changes than the 23% and 20% increases seen with placebo treatment. Although this initial study demonstrated the efficacy of pentoxyifylline, its usefulness in treating intermittent claudication has been questioned. Cilostazol is a newer medication with more potent vasodilatory and antiplatelet activity than aspirin. Cilostazol was found to increase the distances to onset and to maximal claudication pain by 40% and 42%, respectively, which were significantly greater than the 1% and −14% changes seen with placebo treatment.[36] These studies suggest that pharmacologic intervention may be used to treat intermittent claudication in a large percentage of patients. However, exercise rehabilitation results in greater increases in walking distances in patients who are capable and motivated to walk on a regular basis. Therefore, the use of pharmacologic intervention should be done in combination with an exercise program as an initial approach to improve intermittent claudication. The combined therapy of supervised exercise and cilostazol results in the greatest change in walking distance to maximal pain and in ABI compared to standard medical management and each therapy alone.[37] Cilostazol should not be given to patients with any evidence of CHF because of concern for increased risk of mortality since the drug is in the phosphodiesterase III inhibitor class of drugs.

Exercise Rehabilitation

Intermittent claudication results in impaired walking ability and occurs when the peripheral circulation is inadequate to meet the metabolic requirement of the active leg musculature. Consequently, patients with intermittent claudication experience difficulty in carrying out activities of daily living, and are at increased risk of becoming housebound or dependent on others.[38–41] This translates to decreased quality of life, deconditioning, and worsening of co-morbid conditions like hypertension, obesity, hyperlipidemia, and hyperglycemia. These factors may be treated with a program of exercise.

Exercise therapy was first suggested by Erb in 1898,[42, 43] with the first randomized controlled trial published in 1966 demonstrating an improvement in treadmill walking ability.[44] In contrast to either drug treatment or surgical procedures, the clinical management of intermittent claudication in patients with PAD can be significantly improved with little cost, morbidity, and mortality through physical conditioning. Significant improvements in claudication pain have occurred following exercise rehabilitation. For example, meta-analysis demonstrated, that in 21 exercise rehabilitation studies conducted between 1966 and 1993, the average distance walked on a treadmill to onset of claudication pain increased 179%, from a mean of 126 meters to 351 meters following rehabilitation, and the average distance walked to maximal claudication pain increased 122%, from 326 meters to 723 meters.[45] Similarly a more recent meta-analysis of 22 studies showed a median improvement of 119% in pain free walking ability and 83% improvement in absolute walking ability.[46] Consistent with these meta-analyses, a recent prospective study among patients with PAD showed that self directed walking exercise was associated with significantly less functional decline when performed at least 3 times weekly compared with only 1 to 2 times per week.[47] Exercise therapy has been also shown to increase calf blood flow in patients undergoing supervised exercise therapy.[48, 49]

Potential Mechanisms for the Improvement in Claudication Measurements—

Numerous mechanisms have been proposed to explain the improvement in walking distances to the onset and to maximal claudication pain following exercise rehabilitation.[50] The mechanisms primarily center on hemodynamic and enzymatic adaptations within the exercising musculature of the symptomatic leg(s).[51–55] These mechanisms include an increase in blood flow to the exercising leg musculature, a more favorable redistribution of blood flow, greater utilization of oxygen because of a higher concentration of oxidative enzymes in the mitochondria of exercising muscles, improvement in hemorheologic properties of the blood, a decrease in the reliance upon anaerobic metabolism, and an improvement in the efficiency

of walking. It is likely that a combination of changes in these factors contribute to the improved walking distances. Improvements in psychosocial attitude due to accomplishments that are achieved during exercise rehabilitation may further enhance this effect.

Exercise Program Components Predicting Improved Claudication Measurements—Although substantial increases in the average distances to onset of pain and to maximal claudication pain during treadmill walking have been noted following exercise, considerable variability among the studies exists; for example, the increased distance to onset of pain ranges between 73% and 746%, and the increased distance to maximal pain ranges between 61% and 765%. Differences in the components of exercise programs (eg, intensity, duration, and frequency of exercise sessions) may largely account for these widely divergent responses.[45, 56–58]

To examine the contributions of the components of an exercise rehabilitation program, a meta-analysis was carried out.[45] As displayed in Table 3, 6 components were examined: 1) duration of exercise (minutes per session); 2) frequency of exercise (sessions per week); 3) length of the program (weeks); 4) claudication pain endpoint used in the program (onset versus near maximal pain); 5) mode of exercise (walking versus a combination of exercises); and 6) level of supervision. All of the exercise rehabilitation components had a significant effect on the magnitude of change in the claudication distances except for the level of supervision. For example, programs that exercised patients to near-maximal claudication pain were more effective than programs that exercised patients to only the onset of pain. Additionally, programs consisting of higher exercise duration, higher frequency, greater program length, and walking as the only mode of exercise were more effective than programs consisting of lower exercise duration, lower frequency, shorter program length, and having patients train by a variety of exercise modes. The addition of home exercise to supplement the amount of exercise performed in a supervised setting did not result in further ambulatory benefit. Of the 5 components that had an effect on the change in the claudication distances, only 3 were found to have an independent effect through multivariate analyses. These components were the claudication pain endpoint used in the program, the length of the program, and the mode of exercise. The combination of these components explained nearly 90% of the variance in the increase in the walking distances following exercise rehabilitation.

Recommended Exercise Program for Treating Intermittent Claudication—Optimal improvements in claudication symptoms are elicited by having patients walk intermittently beyond the onset of pain for as long as they can safely tolerate, and perform this exercise program for a minimum of 6 months. Although the duration and frequency of the exercise sessions are not independent predictors of the change in claudication pain times, patients should walk for at least 30 minutes per session and for at least 3 sessions per week, as these amounts were more beneficial than programs using a lower exercise duration and frequency.

A review of only 5 controlled trials recommends that the optimal exercise program for treating intermittent claudication consists of exercising under supervised conditions for at least 2 months and at high intensity.[59] However, the appropriate exercise intensity to use during training cannot be determined at this time because no study has addressed this issue. There is a common misconception that walking beyond the onset of pain to near maximal pain is an increase in intensity when, in fact, it is merely an increase in duration. The rate of work performed while walking, regardless of the duration, is the important consideration when setting the appropriate exercise intensity. Because heart rate is commonly used as a means to adjust the intensity of exercise, a conservative recommendation for patients with claudication who are beginning rehabilitation is to walk at an appropriate speed and grade on a treadmill to elicit an intensity of approximately 50% of the heart rate reserve, and to gradually increase the

intensity to 70 to 80% of their heart reserve by completion of the program. Table 4 summarizes recommendations for an exercise program for patients with PAD. Supervised programs now have a specific Current Procedural Terminology code (CPT 93668) for reimbursement.

Revascularization Therapy

Patients with life style limiting claudication that interferes with life or having resting leg pain can be considered for revascularization therapy which includes endovascular or surgical therapy. Endovascular interventions have seen a exponential growth since their first introduction in 1964.[59] They are recommended in case of failure of pharmacological or exercise therapy and in cases where a favorable risk benefit ratio exists, such as in patients with focal aorto iliac disease and focal femoro popliteal disease as discussed in detail in the recent guidelines of the American College of Cardiology/American Heart Association and Trans Atlantic Inter-Society Consensus for the Management of PAD (TASC II).[60, 61] Endovascular interventions include percutaneous transluminal angioplasty (PTA), stenting, stent grafting, atherectomy, and cryotherapy.[62, 63] Initially PTA and stenting was thought to be a procedure of choice for shorter, more focal stenoses, whereas long occlusions required a surgical therapy. However, with improvement in endovascular techniques and experience, this is changing as more complex procedures involving longer infra inguinal lesions are being treated by endovascular interventions.[64]

The indications for surgical treatment are the same as those for endovascular treatment. Comparable efficacy can often be achieved, with less risk imposed by endovascular intervention when both procedures are feasible. Choosing between endovascular vs. surgical therapy depends on several factors including age, co morbidities, and type of the lesion (TransAtlantic Inter-Society Consensus [TASC] lesion classification). Surgery is usually reserved for individuals who have limb arterial anatomy that is favorable to obtaining a durable clinical result, and in whom the cardiovascular risk of surgical revascularization is low. In general it is agreed that endovascular treatment would be treatment of choice for TASC A lesions and surgery would be preferable for TASC D lesions.

SUMMARY

PAD is a common disorder with increasing prevalence in the elderly population. Conservative management of patients with asymptomatic PAD and patients with intermittent claudication is recommended to modify risk factors and improve ambulatory ability, while patients with more severe PAD typically require revascularization of the lower extremities. Exercise rehabilitation is a highly effective, conservative treatment to improve ambulation in patients with intermittent claudication. To date, the primary focus of attention on the benefits of exercise rehabilitation has centered on the increase in walking distances to onset and to maximal claudication pain during a treadmill test. Future research should focus on the improvement in other functional outcomes which may be more representative of everyday activities such as submaximal exercise performance, walking economy, balance, flexibility, and lower extremity strength. Until these measures are obtained, the full benefit of exercise rehabilitation for PAD patients remains undefined.

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summary 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. J Am Coll Cardiol Mar 21;2006 47(6):1239–1312. [PubMed: 16545667]

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TABLE 1

FONTAINE CLASSIFICATION OF PERIPHERAL ARTERIAL DISEASE[14]

Stage	Symptoms
I	Asymptomatic
II	Intermittent claudication
IIa	Pain-free, claudication walking >200 m
IIb	Pain-free, claudication walking <200 m
III	Rest/nocturnal pain
IV	Necrosis/gangrene

Table 2

RUTHERFORD CLASSIFICATION OF PERIPHERAL ARTERIAL DISEASE[14]

Grade	Category	Clinical Description
I	0	Asymptomatic; not hemodynamically correct
	1	Mild claudication
	2	Moderate claudication
	3	Severe claudication
II	4	Ischemic rest pain
	5	Minor tissue loss; nonhealing ulcer, focal gangrene with diffuse pedal ischemia
III	6	Major tissue loss extending above transmetatarsal level; foot no longer salvageable

Table 3

THE EFFECTS OF EXERCISE PROGRAM COMPONENTS ON CHANGES IN CLAUDICATION PAIN DISTANCES FROM 21 STUDIES*

Exercise Programs Components	Change in the Distance to Onset of Pain (M)	Change in the Distance to Maximal Pain (M)
Exercise duration		
≤30 min/session (n = 8)	143 ± 163	144 ± 419
>30 min/session (n = 6)	314 ± 172**	653 ± 364†
Exercise frequency		
<3 sessions/week (n = 7)	178 ± 130	249 ± 349
≥3 sessions/week (n = 11)	271 ± 221**	541 ± 263**
Length of program		
<26 weeks (n = 10)	132 ± 159	275 ± 228
≥26 weeks (n = 11)	346 ± 162†	518 ± 409†
Claudication pain end point used During training sessions		
Onset of pain (n = 15)	105 ± 91	195 ± 78
Near-maximal pain (n = 6)	350 ± 246†	607 ± 427†
Mode of exercise		
Walking (n = 6)	294 ± 290**	512 ± 483**
Combination of exercises (n = 15)	152 ± 158	287 ± 127
Level of supervision		
Supervised (n = 11)	238 ± 120	449 ± 292
Combination of home and supervised (n = 8)	208 ± 198	339 ± 472

*Values for each component are adjusted means ± standard deviations of the change in the distances to onset and to maximal claudication pain after statistically controlling for the other five exercise programs components.

**Significant difference in the change scores between groups, ($P \leq .05$) [45]

†Significant difference in the change scores between groups, ($P \leq .01$)[45]

Table 4

RECOMMENDED EXERCISE PROGRAM FOR PATIENTS WITH PERIPHERAL ARTERIAL DISEASE[45]

Exercise Component	Comment
Frequency	Three exercise sessions per week.
Intensity	Initially, 50% of peak exercise capacity, with gradual progression to 80% by the end of the program.
Duration	Initially, 15 minutes of exercise per session, with gradual progression to 40–50 minutes by the end of the program.
Mode	Weight bearing (e.g., walking, stair climbing). Nonweight-bearing tasks (e.g., bicycling) may be used for warming up and cooling down.
Type of Exercise	Intermittent walking to a claudication pain score of 3 using a 4-point pain scale.
Program Length	Approximately 6 months.