How to Treat

PERIPHERAL arterial disease (PAD) is a common problem facing GPs and cardiovascular physicians. Atherosclerosis, the most common cause of symptomatic obstruction in the peripheral arterial tree, remains a leading cause of morbidity and mortality in the world.

The prevalence of PAD increases with age — 2-3% at age 60, increasing to 20-25% in people over 70. PAD occurs most often in elderly men (mean age 66; ratio of men to women 2:1). Although less common in younger people, it is prevalent in patients over 50 with common atherosclerotic risk factors.

Other disease states affecting the peripheral circulation include atheroembolic syndromes, thromboangiitis obliterans (Buerger’s disease), the vasculitides, vasospastic disorders, popliteal entrapment syndrome, fibromuscular dysplasia and hypercoagulable states.

Atherosclerotic PAD typically involves the superficial femoral and popliteal arteries, occurs in older patients and has a close association with coronary artery disease. Patients with diabetes develop extensive and rapidly progressive disease at a younger age, typically multi-segmental in distribution, with frequent involvement of the popliteal trifurcation and tibio-peroneal arteries, as well as sequential lesions within one arterial segment. They also demonstrate an increased prevalence of occlusive disease in distal small vessels, and potential collateral pathways such as the profunda femoris are more frequently involved than in people without diabetes. Aorto-iliac disease tends to occur predominantly in younger patients.

The clinical presentation of patients with PAD is highly variable and depends on the involved vascular territory. Symptoms range from mild lower extremity discomfort during intense exercise, to the presence of constant rest discomfort, painful ulceration, or frank gangrene (chronic critical limb ischaemia).

For the purposes of management, PAD can be classified into the following three major groups:

■ Intermittent claudication
■ Chronic critical limb ischaemia
■ Acute limb ischaemia

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How to treat peripheral arterial disease

Intermittent claudication

Claudication is variably described as pain, tightness, ache, or aching sensation on walking, cramping, or discomfort of the muscles of the lower limbs, typically in the calf, thigh, buttock, or the foot. It occurs with activity and resolves promptly with rest. It is consistently reproducible and the severity is measured in terms of the claudication distance (the distance travelled before the onset of symptoms), which is usually constant.

The sudden onset of severe limb symptoms or conversion of rest pain implies superimposed thrombosis, which invariably accompanies severe atherosclerotic PAD.

Calf claudication alone suggests superficial femoral artery and/or popliteal disease. Calf, thigh, and buttock claudication suggests axillofemoral disease. Isolated foot pain with ambulation may suggest infra-popliteal disease or Buerger’s disease.

Neuropathic conditions may be the cause of symptoms (pseudoclaudication) [table 1] if the severity of the pain varies widely, the pain does not subside quickly with rest or if it is accompanied by numbness and paraesthesia.

Claudication is experienced by only 10-20% of patients with PAD, whereas 50% have atypical symptoms, such as aching or tiredness after walking, or foot or ankle pain while walking.

Symptoms related to PAD rarely occur until the atherosclerotic process has narrowed the vessel diameter by at least 50%. However, the presence of one or more medical conditions (50% stenosis does not imply that the patient will be symptomatic) with complete occlusion of the major blood supply to a limb or organ may result in the development of symptoms if an ample collateral supply is present.

Also, diagnostic claudication may not be recognized because of lack of exercise — satiety individual patients with moderate to severe PAD may not experience claudication.

Physical findings

Thermography is insignificant in the diminution or absence of lower extremity pulses. For example, a normal common femoral pulse but absent popliteal and ankle pulses indicate occlusion of the superficial femoral or popliteal artery. In the absence of a popliteal pulse there may be an increase in skin temperature in the region of the knee, indicating an elevated medial collateral circulation.

Examination should include auscultation for bruits over the abdominal aorta, the common femoral arteries at the groin, the adductor canal and at the popliteal fossa, especially in patients with diabetes. The history should always be inspected for skin integrity and wounds. Palpation of the arterial pulses and auscultation for bruits can provide an indication of the degree of stenosis.

A reduced pulse with a loud bruit over that vessel indicates at least a 70% stenosis. A barely palpable pulse associated with a soft bruit indicates >90% stenosis. With complete occlusion, no pulsations or bruits are detectable, except for bruits in collateral vessels.

In patients with occlusive arterial disease, elevation of the legs at an angle >60° and repeated flexing of the calf muscles (Buerger’s test) produces pallor of the soles of the feet, followed by a rubor of reactive hyperemia when the legs are placed in a dependent position.

Risk factors

All patients with vascular disease should be assessed for risk factors, including smoking, diabetes, dyslipidaemia, hypertension, and hypercoagulable states (Buerger’s test) produces pallor of the soles of the feet, followed by a rubor of reactive hyperemia when the legs are placed in a dependent position.

Risk factors for PAD include hyperlipidaemia, diabetes, arterial hypertension, smoking, and obesity. About 70% of patients with intermittent claudication will have stable symptoms or become less symptomatic after 5-10 years because of collateral vessel development.

Progressive deterioration of claudication to rest pain or hyperpigmentation in 3-5% of patients annually and in 15-20% patients over a period of 10 years. Amputation is required in 1% annually and in 5-10% over 5-10 years.

Patients with diabetes are a unique subgroup — they have a high likelihood of developing critical limb ischaemia, with an amputation rate seven times greater than for patients with PAD but no diabetes.

The natural history of patients with occlusive PAD is influenced by the extent of coexistent coronary artery and cerebrovascular disease. Fifty per cent of patients presenting with symptomatic PAD also have severe coronary artery disease, with an overall reduction in life expectancy of 10 years; 10-20% will have non-fatal MI or stroke and the mortality rate approaches 30%.

The choice of appropriate footwear is important because most limb-threatening complications in patients with occlusive arterial disease arise from trauma.

Diagnosis

PAD is assessed by non-invasive techniques to:

- Establish the diagnosis of PAD objectively.

- Assess the severity of the disease.

- Select appropriate management strategies.

- Provide a prognostic guide and monitor natural progression.

- Assess the ankle brachial index (ABI), calculated from the ratio of ankle systolic pressure to brachial systolic pressure, giving a sensitive estimate of the degree of arterial insufficiency and usually correlates with functional symptoms.

- For example, an ABI of 0.7-0.8 suggests mild intermittent claudication, in the presence of rest pain, an ABI usually <0.5 suggests severe claudication using the ankle peroneal index (ABI).

- Patients should examine their feet on a daily basis, looking for blisters, dryness, rawness, changes in the skin around the heel pressure point, and areas of unusual skin discolouration. Dryness and fissuring of the skin are markers for the need to protect the skin from further trauma.

- Any infection needs to be treated urgently with appropriate antimicrobial therapy.

Conservative measures

Ischaemic tissues tolerate infection poorly. Maintaining skin integrity is therefore important in patients with arterial insufficiency, so meticulous foot, skin, and nail care and good footwear are essential.

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Conservative measures

- Treating coexisting disease (eg, hypertension).
- Avoiding foot problems (eg, hyperlipidaemia).
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- Any infection needs to be treated urgently with appropriate antimicrobial therapy.

Risk factors for PAD

The importance of smoking cessation and aggressive control of hyperlipidaemia, hypertension and diabetes remain fundamental to the management of PAD. The GP must assume a role in lifestyle modification because modifying risk factors decrease the rate of cardiac and vascular ischaemic events.

Patients who stop smoking have significantly better outcomes (table 2), and cessation of smoking contributes to better assessment of PAD, reducing re-occlusion rates in patients with PAD over time.

Meticulous control of diabetes is essential. Hyperglycaemia is a major problem because it increases vascular ischaemic events, reducing vascular thrombogenicity and limiting infected ischaemic lesions, which are typically resistant to treatment and troubling for patients. Aggressive lipid-lowering therapy is indicated in patients with PAD.

Conservative measures

- Treating coexisting disease (eg, hypertension).
- Avoiding foot problems (eg, hyperlipidaemia).
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Prophylaxis against thrombotic events associated with atherosclerosis (eg, with antipileptic drugs).

In contrast to the medical treatment for coronary artery disease, no pharmacological agent has proved efficacious enough to significantly improving symptoms of PAD to gain widespread acceptance or use.

While antplatelet agents have been reported to decrease progression of atherosclerosis in occlusive PAD, no improvement in exercise capacity has been demonstrated. Clopidogrel has been shown to reduce the overall rate of ischemic events in PAD patients.

All patients with PAD should receive antplatelet therapy with at least aspirin. Neither anticoagulant agents nor thrombolytic agents have been shown to be effective in chronic occlusive arterial disease.

Several vasodilators (eg, calcium-channel blockers, alpha-adrenergic antagonists, and metabolic agents (l-carnitine, l-arginine) have been studied but none has conclusively demonstrated to improve symptoms related to PAD. Oxygen therapy, (Tristar), a rheolytic agent, has been shown to increase walking distance by up to 21% over placebo. Patients most likely to benefit were those with moderately severe occlusive arterial disease (ABI ≤0.80) who have been symptomatic for more than one year. However, most investigators agree that oxygen therapy should not be considered as a substitute for exercise and risk factor modification in symptomatic PAD.

Management of associated disorders

All patients being considered for revascularisation and with a history of past vascular events, symptoms of diabetes or multiple risk factors for atherosclerosis should be assessed for myocardial disease using stress test and carotid or extracranial carotid artery duplex ultrasound.

Interventional therapy

Invasive therapy to restore pulsatile flow is the most effective treatment for the immediate relief of symptoms of PAD. Revascularisation is usually reserved for:

- Patients with progressive disease (increasing symptoms for longer than six months).
- Severe or disabling symptoms that interfere with employment or lifestyle (claudication distance usually ≤100m).
- Acute limb ischemia.
- Chronic critical limb ischemia.

When the indications for invasive therapy are clear and the anatomical substrate has been defined, the choice will be between conventional surgery and catheter-based techniques.

Surgery

Surgery typically involves placement of saphenous-venous or prosthetic materials to bypass or subside for a severely diseased native artery. Patency rates for surgical procedures at five years are 80-90% for aorto-bifemoral bypass, 60-80% for above-knee vein grafts, and 50-70% for below-knee vein grafts.

The choice of surgical procedure is influenced by the distribution of disease, the adequacy of distal run-off vessels, and by comorbidities. In some situations (aortic occlusion, common femoral artery bifurcation disease, painful aneurysm), surgery remains the gold standard for revascularisation.

Quality, these operations require general anaesthesia. They involve significant blood loss and fluid shifts in patients who may have profound involvement of the blood supply to other critical organs, thereby increasing operative morbidity and mortality. When possible, use of catheter-based (endovascular) treatments provides a similar level of correction and durability and substantially minimises risk and disability.

Lumbar sympathectomy

Lumbar sympathectomy is used alone in patients not fit for surgery or as an adjunct to arterial revascularisation. There is no good clinical evidence that lumbar sympathectomy improves graft patency or improves limb survival.

Endovascular therapy and anatomical regions of interest

Percutaneous revascularisation has become increasingly popular as first-line therapy for PAD. Intervention is indicated in selected patients with disabling intermittent claudication in whom medical therapy has failed, except in two unique subgroups:

- Patients with diabetes or increased rates of chronic critical limb ischemia and severe occult amputation rates, who need a more aggressive approach on signs of deterioration.
- Patients who develop acute limb ischemia (see page 35).

Aorto-Iliac disease. Considerable controversy exists concerning the optimal treatment in this situation. Percutaneous transluminal angioplasty is generally used for more focal disease of the distal aorta, common iliac arteries, and external iliac arteries (figure 2). However, the availability of hydrophilic guiding-wires has led to an increased ability to revascularise even lengthy, chronic iliac occlusions (figure 3).

Endovascular stents (and drug-coated balloons) have dramatically improved the short-term (90-100% technical success and long-term results (75-90% five-year patency) of percutaneous transluminal angioplasty. This compare favourably with results of surgical revascularisation for aortoiliac disease (primary patency at 7.5 years 75-80%) but at the cost of 2-3% mortality.

Superficial femoral and popliteal artery disease. The role of percutaneous therapy versus surgery is controversial in this group. In superficial femoral artery or popliteal stenotic disease, angioplasty has a high technical success rate and reasonable long-term patency rates (up to 70% (figure 4) and now must be considered as first-line therapy.

Percutaneous revascularisation is effective in lesions up to 70% (figure 4) and now must be considered as first-line therapy.

Invasive therapy to restore pulsatile flow is the most effective treatment for the immediate relief of symptoms of PAD.
**Chronic critical limb ischaemia**

This clinical entity describes patients with advanced chronic occlusive PAD who are at particular risk of major lower limb complications, including extensive ischemia, recurrent claudication, and chronic critical limb ischemia.

Clinical features

Chronic critical limb ischaemia is characterised by rest pain, ulceration and skin necrosis or superficial gangrene. Rest pain is usually more severe at night and can only be relieved by placing the legs in a dependent position. If however, prolonged dependency may cause cedema, further thrombosis, microcirculation, with delayed healing of ulceration.

Acute critical ischaemia, characterised by a severe lancinating or burning sensation in the leg and foot, may be precipitated and persistent for many months even after correction of the ischaemia.

**A acute limb ischaemia**

**ACUTE limb ischaemia** is a medical emergency. Successful management depends on early diagnosis and urgent intervention; the patient should be immediately transferred to an institution with facilities for angiography and revascularisation.

### Aetiology

This condition occurs most often after thrombotic occlusion of atherosclerotic native arteries or bypass grafts, or patent popliteal thrombosis with, or without underlying impediment to flow. Thrombotic occlusion of a popliteal artery aneurysm is almost always a serious limb-threatening complication.

Embolic occlusion accounts for about 10% of cases. The source is cardiac (AF, post MI), valvular heart disease in >75% of cases. Emboli may also originate from an aneurysmal disease and atherosclerotic lesions in a thoracic or abdominal aorta.

Rarer causes include Buerger’s disease, arterial trauma (e.g. drug, erg, acute arterial trauma), arterial dissection and popliteal arterial aneurysm. In a thrombotic occlusion of a popliteal artery aneurysm is almost always a serious limb-threatening complication.

### Embolic occlusion

Acute onset of limb ischaemia is associated with pain, paraesthesia, numbness and coldness, muscle tenderness and paresthesia. The extremity is cool, pale and pulseless.

A presentation depends on the duration and level of occlusion, the status of the underlying vessels, and general factors: blood pressure, cardiac output, absence or presence of diabetes, O2 saturation.

A preceding history of stable claudication, abrupt shortening of the claudication distance and the finding of arterial bruits elsewhere are suggestive of the diagnosis.

A popliteal or sciatic artery thrombosis is often due to an in situ atheromatous lesion, and will progress slowly over several days. A popliteal or sciatic artery atheromatous lesion may also be due to an embolic source from the heart or aorta. The ischaemia is usually painless, but may be mistaken for a deep vein thrombosis or a similar condition.

A pre-existing progressive occlusion of a pre-existing arterial narrowing. Embolic events may have a sudden presentation with a cold or blue digit.

A series of clinical categories with well-defined diagnostic criteria help determine whether the affected limb is viable, threatened or already irreversibly damaged (table 3).

### Table: Classification of acute limb ischaemia by clinical features

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
<th>Capillary return</th>
<th>Muscle weakness</th>
<th>Sensory loss</th>
<th>Arterial doppler</th>
<th>Venous doppler</th>
</tr>
</thead>
<tbody>
<tr>
<td>Viable</td>
<td>Not threatened</td>
<td>Intact</td>
<td>None</td>
<td>None</td>
<td>Audible</td>
<td>Audible</td>
</tr>
<tr>
<td>Threatened</td>
<td>Salvageable if properly treated</td>
<td>Intact or slow</td>
<td>Mild, partial</td>
<td>Mild or complete</td>
<td>Not audible</td>
<td>Audible</td>
</tr>
<tr>
<td>Irreversible</td>
<td>Major tissue loss, amputation</td>
<td>Absent</td>
<td>Paralysis</td>
<td>Anaesthesia</td>
<td>Not audible</td>
<td>Not audible</td>
</tr>
</tbody>
</table>

**AP = ankle pressure**

Clinical signs include skin atrophy accompanied by rubor and reduced skin temperature. Hair is thinned in areas of chronic fungal infection and poor nail growth. The ulcers occur most often in areas subject to friction (the sole of the foot) and are typically necrotic, dry and extremely painful, with ill-defined and cyanotic borders. They are often covered with a black eschar (figure 7).

In patients with ulceration exercise is restricted until ulceration has healed. Strict control of diabetes is crucial because uncontrolled diabetes is a hypercoagulable state that also encourages infection. Patients with diabetes also suffer from various neuropathies (sensory, motor, autonomic) that contribute to callous formation, lengthening ulcers, sub-callosus haemorrhage and infection, and ultimately, ulceration. Therefore even minor foot lesions in these patients should be treated vigorously.

Antibiotics (including anabolic cover) should be given in the presence of clinical evidence of cellulitis. In severe infections IV antibiotics are indicated. Topical antibiotics are of no value and should be avoided.

Heparin should be started to prevent arterial thrombosis and to decrease the risk of venous thromboembolism that accompanies ischaemia and reduced limb perfusion. Thrombolytics may be indicated in selected patients. Compression stockings and calf- compression devices cannot be used for DVT prophylaxis in the presence of severe occlusive PAD.

Long-term treatment with anticoagulants reduces the risk of cardiovascular death in those with current MI or coronary disease in patients with PAD and maintains patency after revascularisation.

Prostaglandins by infusion have benefit in patients with chronic critical limb ischaemia and intractable ulceration, frank gangrene before surgery (to accelerate demarcation and healing). Some ulcers heal quickly or demonstrate a marked reduction in size with this technique. However, extremity ulcers, especially of the foot, may take several months even after correction of the ischaemia.
How to treat - peripheral arterial disease

Other selected disease states of the peripheral arteries

Atheroembolism

Atheroembolism is caused by macro-embolisation of friable or ulcerated atherosclerotic plaque debris, or micro-embolisation of cholesterol crystals liberated from the intimal arterial tree, either spontaneously or after provocation (direct surgical or catheter manipulation, antiplatelet therapy, anticoagulation). It is more common in elderly men with advanced atherosclerosis.

Clinical presentation is related on the region affected, the embolic load and the status of the underlying vascular bed. Atheroembolism to the lower extremities may be focal, affecting one or more toes (for toe syndrome) or diffuse, causing bilateral livedo reticularis, cyanosis and ischaemic pain (for hose syndrome) or skin ulceration and gangrene.

Involvement of the renovascular bed may result in deteriorating renal function and/or renal failure. There may be non-specific symptoms suggesting a systemic illness, with fever, malaise, anorexia, weight loss, headache and myalgia, or it may provoke acute catastrophic multi-organ failure.

Atheroembolism should be considered when a patient presents with new limb ischaemia, renal failure, transient cerebral ischaemia or angina not explained by local large artery occlusive disease. Suggestive laboratory findings include an elevated ESR, leucocytosis with eosinophilia, hypercoagulable state, anemia, elevated transaminases and acylasemia.

A diagnosis may be confirmed by fundoscopy or tissue biopsy, showing pathologic cholesterin crystals in new or remote arterioles or small vessels of affected tissues.

Treatment strategies include conservative local measures for the affected tissue and, importantly, appropriate steps to prevent further embolic events, including antiplatelet therapy and avoidance of surgical and catheter manipulation near the presumed source of embolisation.

Occasionally, especially in the case of recurrent atheroembolism, exclusion of the embolic source (either surgical or percutaneous) may be necessary.

Refractory failure often requires dialys support. These patients have a high risk of cardiovascular death in subsequent years.

Buerger’s disease

Buerger’s disease (thromboangiitis obliterans) is an aggressive form of obliterative arteritis affecting predominantly the small and medium-sized arteries and veins of the upper and lower extremities. It is associated with a segmental, inflammatory thrombotic vasculitis and most patients have evidence of other coexisting conditions.

Patients typically present with ischaemic ulceration of one or more digits, which may progress to gangrene. They may have associated arch, foot or calf claudication, cold sensitivity, and episodic superficial thromboembolitis. The disease almost always involves two or more limbs; an abnormal Allen test is present in two-thirds of patients.

Buerger’s disease must be distinguished from premature atherosclerotic arterial occlusion and polyarteritis nodosa, Wegener’s granulomatosis and the hyper vasospastic vasculitides. The disease may present with angina, claudication, and gangrene due to prolonged ischaemia.

Although most attacks are mild, patients with severe Raynaud’s occasionally present with ulceration or gangrene due to prolonged ischaemia.

Vasculitic diseases

Vasculitic diseases can produce transient ischaemic symptoms in the extremities, especially in patients with altered perfusion or smokers.

There may be non-specific symptoms or signs and symptoms of systemic involvement.

Vasospastic diseases

Vasospastic diseases are associated with cutaneous manifestations and may be idiopathic or occur in association with systemic diseases.

The vasculitic syndromes are quite heterogeneous in their pathophysiology, clinical presentation, and prognosis. The vasculitic syndromes are quite heterogeneous in their pathophysiology, clinical presentation, and prognosis. Raynaud’s and other vasospastic disorders is aimed at local (warming the extremity) and pharmacological measures that facilitate vasodilatation.

Poliarticular entrapment syndrome

This syndrome is caused by a congenital abnormality in the relationship between the popliteal artery and the medial head of gastrocnemius muscle. It may be present without symptoms or cause acute or chronic lower limb ischaemia. The diagnosis should be considered in active people who present with exercise-induced calf claudication.

Normal ankle pulses are present with the ankle in extension but become decreased or absent when the ankle is flexed. Angiography demonstrates segmental occlusion of the medial segment of popliteal artery, median dilatation, post-stenotic dilatation or poliptal aneurysm formation.

Treatment consists of operative release of the entrapped artery, thrombolytic of obstructed segments and/or vein bypass grafts for symptomatic individuals as well as asymptomatic patients at risk for severe limb-threatening ischaemia.

Hypercoagulable states

Hypercoagulable states may present with clinical features of arterial insufficiency. Some (eg, hypercoagulable states) are associated with premature onset of peripheral atherosclerosis and, secondarily, exacerbation to thrombosis.

Others, including the primary hypercoagulable states (eg, anti thrombin III deficiency, protein C or S deficiency) and antiphospholipid antibody syndrome, predispose patients to early thrombosis of arterial grafts and veins.

The therapeutic approach to these patients includes early recognition of the abnormality and judicious use of anticoagulants and/or antithrombotic agents.

The future

In the future, invasive therapy for PAD will continue to shift toward a predominantly percutaneous, as opposed to surgical, approach. As interventions become more effective and safe, the threshold for treatment will be lowered and the number of percutaneous interventions will rise.

For patients who have had to opt for amputation in the absence of viable options for conventional revascularization, the possibility of encouraging the formation of new vascular conduits by intro ducting angiogenic factors or stem cells may offer new hope for limb salvage.

Author’s case study

Early revascularisation is essential for limb salvage. M R, aged 20-40, especially women. The disease affects major arteries. Coronary and mesenteric arteries are infrequently involved. It may be more prevalent in young adults aged 20-40, especially women.

Preliminary findings on the region involved, Angiographic appearance is a ‘string of beads’, with focal stenoses and post-stenotic dilatation. Treatment is angioplasty of the affected vessel in symptomatic patients.

Figure 5: A: Mr JW, 82, with necrotic ulceration of three toes, ischaemic rubor, atrophic skin and loss of hair. B: The left lower limb diagnostic angiogram three months after percutaneous revascularisation. Note the healing of necrotic ulceration and improved skin colour.

Figure 10: A: Pre-PTA DSA image of Mr JW’s left lower leg, showing 50% stenosis of the popliteal artery, 50% stenosis of the tibio-peroneal trunk, occluded peroneal artery and subtotal occlusion of the anterior tibial artery. B: Post-PTA DSA after percutaneous revascularisation of the popliteal artery, tibio-peroneal trunk and all three infrapopliteal run-off vessels, with restoration of antegrade flow to the foot.
**How to treat – peripheral arterial disease**

**GP’s contribution**

Revascularisation would be indicated.

GEOFFREY has multiple athrosclerotic risk factors and should therefore also undergo complete cardiovascular assessment including coronary, renal and carotid circulations, as well as assessment of the effects of hypertension and smoking.

He should also have X-rays of the foot and ankle to exclude any mechanical pathology, routine blood testing and testing specifically for autoimmune markers, uric acid and rheumatoid factor.

General questions for the author.

Rest pain in chronic critical ischaemia is usually worse at night. However it can be clinically differentiated from pain of neurospinous origin.

Ischaemic rest pain is often relieved with the limb placed in the dependent position (ie, downwards). Therefore, the patient may gain relief if the foot of the bed is lowered and if they hang their foot over the side of the bed. Also, patients often cannot tolerate the best diagnostic method.

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**took at:** Sydney, NSW

**INSTRUCTIONS**

Complete this quiz to earn 2 CPD points and/or 2 PDP points by marking the correct answer(s) with an X on this form. Fill in your contact details and return to be used for fax or free post.

**Fax back free post online**

**www.australiandoctor.com.au/cpd** for immediate feedback

**How To Treat Quiz**

Peripheral arterial disease – 13 May 2005

1. Which TWO statements about PAD are correct?
   - a) Diastolic hypertension is a better predictor of PAD than systolic hypertension
   - b) PAD is twice as common in people with diabetes than those who do not
   - c) Dyslipidaemia increases the rate of progression of PAD
   - d) Tobacco smoking is the single most significant risk factor in PAD

2. Trevor, 75, has exercise-induced thigh and calf pain. Which feature would alert you to the possibility that the claudication is a result of PAD and not secondary to neurospinous disease?
   - a) Leg clamminess and weakness
   - b) Pain with standing
   - c) Pain relieved by rest
   - d) Variation in the distance walked before pain begins

3. Trevor has absent ankle pulses and a right femoral bruit. His ABI is 0.8 bilaterally. Which THREE statements about the information provided by an ABI are correct?
   - a) An ABI has a prognostic value in determining both limb survival and survival for all-cause mortality
   - b) It can differentiate stenosis from occlusion
   - c) It is a sensitive indicator of the degree of arterial insufficiency
   - d) It is useful for functional assessment in conjunction with treadmill exercise testing

4. Which TWO treatments may increase Trevor’s exercise capacity?
   - a) Dopetidyl (Trental)
   - b) Regular exercise
   - c) Antispasmodic therapy
   - d) Vasodilators such as calcium-channel blockers

5. George, 82, with a past history of cerebrovascular disease and smoking, has chronic critical limb ischaemia. He has developed rest pain and a punched-out ulcer on the dorsum of his foot. Superficial femoral artery stenosis is identified on angiography. Which ONE procedure may improve limb survival with minimal risk to the patient?
   - a) Limb sympathectomy
   - b) Endovascular therapy
   - c) Surgical revascularisation
   - d) Laser angioplasty

6. George is concerned about the incidence of re-stenosis after endovascular therapy. Which THREE statements about re-stenosis are correct?
   - a) Cigoprilod is used for 6 months after stent insertion to reduce the risk of re-stenosis
   - b) Healing of the ulcer will typically occur if the artery remains patent for 3–6 months
   - c) If re-stenosis occurs, amputation is the only solution
   - d) Risk factors for PAD should be strictly controlled

7. In which TWO ways should George’s leg be managed?
   - a) Meticulous care is necessary to prevent pressure sores
   - b) Topical antibiotics should be applied to any ulcerated areas
   - c) Compression stockings should be used to reduce the risk of DVT
   - d) Topical nitrates may be helpful

8. Vince, 72, has atherosclerosis and presents with an acutely ischaemic lower limb. The ankle pressure is <40mmHg and there is evidence of slow capillary return and mild sensory and motor loss. However, there is an audible venous Doppler and the limb is not considered to be irreversibly threatened. What is the most important part of the initial management (choose ONE)?
   - a) Antibiotic therapy
   - b) Surgical revascularisation
   - c) Invasive treatment (surgical or percutaneous)
   - d) Elevate the limb

9. Vince has percutaneous catheter-based thrombolytic therapy. Which ONE statement about this therapy is correct? 
   - a) Thrombolytic agents can be given IV
   - b) Thrombolytic agents are given IV
   - c) The procedure needs to be done within 24 hours
   - d) This is the treatment of choice for an irreversible threatened limb

10. Which TWO statements about thromboangiitis obliterans (Buerger’s Disease) are correct?
   - a) Antibiotic therapy is useful in management
   - b) It mostly affects men >50 who smoke
   - c) Angiography demonstrates normal anatomy
   - d) Invasive treatment including revascularisation

The mark required to obtain points is 80%. Please note that some questions have more than one correct answer. Your CPD activity will be updated on your RACGP records every January, April, July and October.