

INTRODUCTION

Peripheral arterial disease (PAD) is generally defined as a chronic athero-thrombotic occlusive disorder affecting small, medium and large arteries of the lower extremities. Atherosclerosis can affect upper extremity arteries but is best addressed separately as upper limb ischaemia. The importance of PAD extends beyond the traditional concerns of gangrene and limb loss. PAD is becoming increasingly recognized as an important and potent surrogate marker of future cardiovascular events in patients with established disease. These events include myocardial infarction, stroke and vascular death.

EPIDEMIOLOGY

The community prevalence of PAD ranges between 3 – 12%. In general the incidence is 5% for those < 60 years, 10% for those between 60 and 70 years, and 20% for those > 70 years. The risk factors associated with PAD are:

- Smoking
- Diabetes mellitus
- Hypercholesterolaemia
- Hypertension
- Increasing age
- Obesity
- Sedentary lifestyle
- Hyperfibrinogenaemia
- Hyperhomocysteinaemia

Black patients tend to have more aggressive disease than Caucasian patients. Diabetic, female, and renal failure patients with PAD tend to have poorer outcomes.

BASIC ANATOMY

The lower extremity circulation is served by the abdominal aorta. Below the renal arteries the abdominal aorta is a smaller calibre than the supra-renal aorta and generally tapers as it bifurcates at the level of L4 (umbilicus

level) into the R. and L. common iliac arteries. It has paired lumbar arteries, and an unpaired inferior mesenteric artery and median sacral artery.

The common iliac arteries bifurcate at the level of the sacro-iliac joint into the internal iliac artery, which supply the pelvic structures, and the external iliac artery which supply the lower extremity proper. The external iliac artery continues as the common femoral artery at the level of the inguinal ligament. The deep circumflex iliac and the inferior epigastric branches mark the point of transition from the external iliac artery to the common femoral artery. The common femoral artery generally lies anterior to the head of the femur (as seen on angiograms)

The common femoral artery bifurcates generally distal to the head of the femur into the profunda femoris artery, which supplies the thigh, and the superficial femoral artery. The superficial femoral artery courses the adductor canal in the thigh and continues as the popliteal artery at the level of the adductor hiatus formed by the adductor magnus muscle. This transition generally occurs between the mid and distal thirds of the thigh. The popliteal artery divides into 3 branches (calf / crural vessels) below the knee joint. This is often referred to as the popliteal trifurcation, although in practice a popliteal bifurcation is more commonly seen. The popliteal artery bifurcates into the anterior tibial artery lateral and the tibio-peroneal trunk medially. The anterior tibial artery courses in the anterior calf compartment and continues as the dorsalis pedis artery at the level of the ankle ("mid-malleolar point"). The dorsalis pedis artery courses on the dorsum of the foot and dives between the first and second proximal metatarsal heads to join the plantar arch. The tibio-peroneal trunk divides

into the peroneal artery and the posterior tibial artery. The peroneal artery divides into a superficial peroneal artery, which courses through the interosseous membrane in the distal third of the leg into the lateral calf compartment, and the deep peroneal artery. The peroneal artery does not cross the ankle into the foot. The posterior tibial artery courses between the superficial and deep posterior calf compartments, and continues distally behind the medial malleolus. Distal to the medial malleolus the posterior tibial artery divides into the lateral and medial tarsal artery which, together with the dorsalis pedis artery, forms the plantar arch.

Thus the circulation to the lower extremity can be conveniently divided into three segments. Occlusive disease can affect one or more of these segments.

- Aorto-iliac segment
- Femoro-popliteal segment
- Tibio-peroneal segment (“infra-popliteal” / “BTK - below the knee” segment)

PATHOPHYSIOLOGY

The dominant pathology in PAD is atherosclerosis. Stable atherosclerotic plaques may be asymptomatic or may be associated with stable claudication. Unstable or vulnerable plaques are generally associated with the onset of rapidly progressive claudication, critical limb ischaemia or acute limb ischemia.

The occlusive atherosclerotic disease in PAD is segmental and classified as follows:

- **Aorto-iliac disease** (absent femoral, popliteal and foot pulses)
- **Femoro-popliteal disease** (bounding femoral pulses but absent popliteal and foot pulses)

- **Tibio-peroneal disease** (bounding femoral and popliteal pulses but absent foot pulses)

Young patients with PAD (< 55 years) may have the following pathologies:

1. Accelerated atherosclerosis (Age: 40 – 55 years)
2. Precocious atherosclerosis (Age: < 40 years)
3. Hypercoagulable states
 - antiphospholipid syndrome
 - hyperhomocysteinaemia
 - cancer patients
4. Vasculitides
 - Scleroderma
 - Rheumatoid arthritis
 - SLE
 - Takayasu`s disease
5. Popliteal entrapment syndrome
6. Anterior compartment syndrome
7. Iliac compression of the cyclist
8. Fibromuscular dysplasia
9. HIV- related PAD

CLINICAL FEATURES

PAD can be classified clinically as:

- **Asymptomatic**
- **Symptomatic**
 - claudication
 - erectile dysfunction
- **Complicated**
 - acute lower extremity ischaemia
 - critical limb ischaemia

Symptoms

Claudication is an exertional symptom that affects predominantly the calf muscles, although the thighs and buttocks may be affected in more proximal occlusive disease. It is often described as “pulling stiff”, cramping, lameness and “giving way” following a period of walking. The symptoms are often relieved following a short period of rest (3-5 minutes).

Claudication may be classified clinically into 4 types:

- **Intermittent claudication:** associated with PAD
- **Venous claudication:** generally associated with iliac vein occlusive disease, rarely with varicose veins alone. Leg swelling is usually the norm and symptoms are relieved following a prolonged period of rest usually in the recumbent position
- **Spinal claudication:** associated with spinal stenosis. These patients are generally elderly patients with chronic spine problems with pain that radiates down the legs. They get out of bed with difficulty and ambulate with difficulty. Symptoms are worse on stepping down stairs with “jarring” pain being experienced with each step. Symptoms may be relieved, paradoxically, by walking uphill. Symptoms are relieved by sitting or leaning forward and take a long time to settle (~ 20 – 30 minutes)
- **Atypical claudication:** A variety of medical conditions may produce atypical leg pain. If claudication is the only presenting symptom and is not compatible with classic intermittent claudication in a patient with established PAD, a systematic clinical approach should be utilized to exclude these conditions, before attributing these symptoms to PAD. **(Table 1 – List of medical conditions presenting with leg pain)**

Erectile dysfunction may be an early indication of PAD, especially in diabetic or pre-diabetic patients. Vasculogenic erectile dysfunction may be seen in patients with a totally occluded aorto-iliac segment (absent femoral pulses) and buttock claudication. This is referred to as the **Leriche syndrome**.

Table 1: Causes of leg pain	
•	Peripheral neuropathy
•	Spinal stenosis
•	Lumbar spondylosis
•	Sciatica
•	Complex regional pain syndromes
•	Osteo-Arthritis
•	Varicose veins
•	Post-thrombotic syndrome
•	Anaemia
•	Restless leg syndrome
•	Tinea pedis
•	Cellulitis

Rest pain is unfortunately a badly named term used to describe foot pain related to severe PAD, but is still used universally currently. Rest pain, or more appropriately “nocturnal ischaemic foot pain”, is an incapacitating ischaemic neuropathic type of pain that involves the toes and forefoot when the patient is in a recumbent position. It is relieved by dependency. Certain conditions may mimic rest pain viz, gout.

The patient with PAD may present with two types of tissue loss:

- **Ischaemic ulcer:** a foot ulcer is ischaemic (associated with PAD) until proven otherwise. These ulcers are pale, white, necrotic (black), or have yellowish slough. These ulcers generally occur at pressure points in the foot: dorsum of the toes; head of the first and fifth metatarsals, lateral malleolus and the heel.
- **Gangrene:** This refers to necrosis of tissue (black). Gangrene may involve the toes or focal areas on the foot and is usually mummified (dry gangrene). Gangrene with sepsis is referred to as wet gangrene.

Critical Limb Ischaemia (CLI) is defined as a patient suffering from PAD with rest pain of more than 2 weeks duration and / tissue necrosis, either ischaemic ulcers or gangrene.

The diagnosis of CLI can be confirmed if the ankle pressures are ≤ 50 mmHg or toe pressures are ≤ 30 mmHg. Diabetic foot ulcers with an ankle pressure ≤ 70 mmHg or toe pressures ≤ 50 mmHg or foot TcPO₂ ≤ 45 mmHg are unlikely to heal.

Signs

Patients with PAD may provide clues to their risk factors for atherosclerosis. All patients must have their height and weight measured. They may have an abnormal body mass index (BMI – normal is 19 to 25). In addition the abdominal girth should also be measured. These patients may have truncal obesity and the metabolic syndrome.

Patients may present with skin tags (acanthosis). **Nicotine stains** may be noticed to involve the fingers, hair, moustache and teeth. Patients may have discoloration of the sclera related to chronic smoking (“muddy sclera”)

Patients with hypercholesterolaemia may have **xanthelasma** involving the eyelids, tendon thickening (especially the Achilles tendon), subcutaneous nodules related to tendons (**xanthomas**), and a white ring in the corneo-scleral junction (**arcus lipidica / arcus cornealis**)

Diabetic patients may have **cataracts, foot deformities or plantar callosities**.

Patients with severe PAD may present with reactive hyperaemia of the ipsilateral foot. The vessels here are maximally dilated resulting in a red, congested looking foot. This is referred to as the **sunset foot**.

Approximately 95% of patients with PAD are smokers. Examination of the chest is important. Patients may have features of hyper-inflation, wheezes and crepitations, features of lung consolidation, pleural effusion, etc.

Patients with PAD are prone to develop coronary artery disease and cerebrovascular disease. A carotid bruit may suggest a carotid stenosis thus identifying a patient at risk for stroke. This is best heard over the carotid pulse at the level of the angle of mandible. A proper cardiac assessment is essential: heart rate and rhythm, abnormal heart sounds, murmurs, displaced apex, features of cardiac failure, etc. The blood pressure must be measured in both arms. A systolic pressure difference of > 20 mmHg in both arms is associated with future cardiovascular events.

Palpation and auscultation of the abdomen is important. Patients with PAD may also have an associated abdominal aortic aneurysm. A bruit in the epigastrium may suggest renal artery stenosis, mesenteric artery stenosis or aortic stenosis.

Examination of the lower extremity in patients with PAD needs to be structured:

- I. **What is the pulse status?**
This will identify the most proximal level of occlusive disease clinically. Pulses may be grade (**Table 2 – grading of peripheral pulses**)
- II. **Are there any trophic (dystrophic features) changes related to PAD?** These may involve the skin and skin appendages, nails and muscle. The skin may be dry and thin. Loss of hair may be appreciated on the dorsum of the toes. There may be atrophy of the nails, nail thickening (onychogryphosis), brittle nails, etc. There may be atrophy of the intrinsic foot muscles with prominence of the dorsal foot tendons. Calf muscle atrophy is rarely appreciated, usually in a patient with unilateral or asymmetrical PAD.

III. **Does the patient have features of CLI?**

Not all patients will have classic rest pain and/or tissue loss. There are a few tests that may identify a patient with a severe circulation problem.

- **The Buerger's test:** The patients lies supine in bed. When the leg is elevated, the foot becomes pale ("cadaveric pallor"). When the patients sits and dangles the foot, it becomes red or dusky ("dependent rubor")
- **Goldflam test:** The patient lies supine in bed. When the leg is elevated the foot pallor is not readily apparent. When the patient wiggles the toes this facilitates drainage of blood and the foot becomes pale.

IV. **Does the patient have features of peripheral neuropathy?**

Sensory neuropathic testing is essential, especially with diabetic patients. All sensations need to be tested. Ankle reflexes need to be elicited. Foot deformities need to be clearly documented (claw toes, hammer toes, scissor/overlapping toes, accentuated instep, etc)

V. **Does the patient have features of chronic venous disorders?**

The patient may present with claudication and have a diffusely swollen, hyperpigmented leg from a previous iliofemoral deep vein thrombosis. Alternatively patients may present with varicose veins, absent leg pulses and a non-healing leg ulcer.

VI. **Are there any features to suggest musculo-skeletal disorders?**

Patients may have spine deformities, spine tenderness, positive straight leg raising sign, joint

deformities, swollen joints, etc. These patients may present with atypical claudication.

VII. **Does the patient have unusual skin lesions?**

Patients may present with necrotic skin lesions confounding PAD, e.g. rheumatoid ulcers, the black necrotic lesions seen with sickle-cell anaemia, etc.

VIII. **Does the patient have peripheral aneurysms?**

Peripheral aneurysms may simulate PAD when occluded. Femoral masses are easily palpable. Popliteal aneurysms may be suggested by a vague mass palpable in the popliteal fossa or an exceptionally bounding contralateral popliteal pulse.

Table 2: Grading pulses clinically
++ Normal pulse
0 Absent pulse
+ Diminished pulse compared to a normal reference pulse
+++ Abnormally prominent pulse (e.g. a bounding popliteal pulse may suggest a popliteal aneurysm)
++++ A visible pulsation (e.g. with an abdominal aortic aneurysm)

Natural history

Approximately 7 – 15% of patients with PAD in the community will develop claudication. For ratio of asymptomatic PAD to symptomatic PAD is 3-4:1

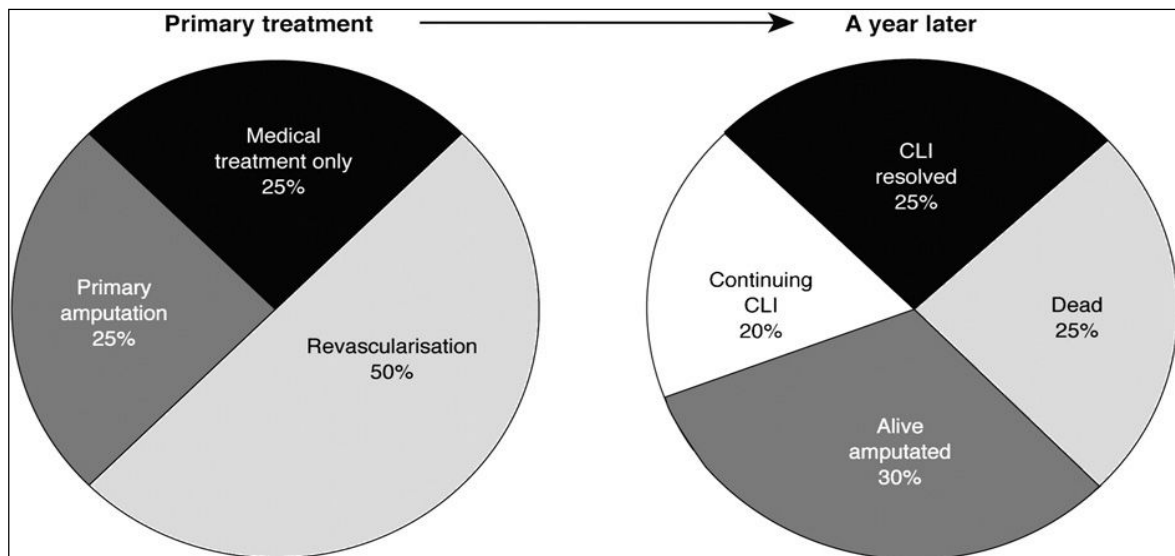
The clinical outcome of claudicants is generally benign: 50% of patients will improve; 25% will remain stable; 25% will have a deterioration of their symptoms; 5 % will require revascularisation and only 1-2% will require a major amputation. For every 100 claudicants 1 patient will develop CLI.

The life expectancy of patients with PAD is variable;

1. ~ 20% - 25% of patients with CLI are dead in 1 year
2. ~ 50% of patients with CLI are dead in 5 years
3. ~ 50% of claudicants are dead in 10 years
4. **Being asymptomatic is not protective.** The life-expectancy in this group is similar to that of claudicants

Note: The risk of amputation in 10 years is only 4%

Causes of death in patients with PAD relate to:



- 55% coronary artery disease
- 10% cerebrovascular disease
- 25% non-vascular
- < 10% other vascular

Fig.1: Fate of the critically ischaemic leg with treatment

CLASSIFICATION OF PAD BASED ON SEVERITY GRADING

The following classifications exist defining the severity of PAD.

- **Fontaine classification**
 - Stage 1: Established PAD but asymptomatic
 - Stage 2: Patients with intermittent claudication

- Stage 3: Patients with CLI and rest pain
- Stage 4: Patients with CLI and tissue loss

- **Rutherford classification**
 - Category 0: Established PAD but asymptomatic
 - Category 1: Mild claudication
 - Category II: Moderate claudication
 - Category III: Severe claudication
 - Category IV: CLI with rest pain
 - Category V: CLI with ischaemic ulcer (minor tissue loss)

- Category: CLI with gangrene (major tissue loss)

In general patients with claudication tend to have a single segment PAD, and those with CLI tend to have multi-segment PAD.

Diagnostic appraisal

The diagnostic evaluation may be basic in patients who are asymptomatic or have mild to moderate claudication.

1. All patients should have the following blood tests done:

- full blood count
- urea and electrolytes
- creatinine
- random or fasting blood sugar
- HbA1C if diabetic on treatment
- Fasting lipogram

2. All patients must have Doppler pressure measurements and determination of the ankle-brachial-index (ABI).

- All pressures are determined by using a hand-held Doppler machine. A cuff is placed proximal to the artery of interest. A Doppler probe is used to insonate the artery of interest. The character of the signal is first appreciated. Is the signal mono-phasic (weak signal), biphasic or triphasic (good signal)?. The cuff is inflated until the signal disappears. The cuff is then very slowly deflated until the signal re-appears. This identifies and measures the systolic pressure. Only systolic pressures can be measured in this manner.
- The ankle-brachial index is measured by dividing the ankle pressure by the best brachial pressure. This is the reason both brachial pressures are measured.
- Normal ABI is 0.9 to 1.3
- An ABI < 0.9 suggests PAD
- Claudicants have an ABI between 0.5 and 0.9
- Patients with CLI and rest pain generally have an ABI of 0.4 or less
- Patients with CLI and tissue loss generally have an ABI of 0.2 or less
- Patients with PAD and very calcified calf vessels have an ABI > 1,3

- In diabetic patients with PAD and foot ulcers, an ABI of > 0.6 is inaccurate and should not be used to rationalize the need to revascularize.

Peripheral vascular imaging studies are generally reserved for patients requiring revascularization.

Duplex ultrasound

Duplex ultrasound (DUS) employs B-mode ultrasound for anatomical information and colour Doppler, and spectral Doppler, for physiological information. Duplex ultrasound of peripheral arteries is non-invasive, readily available and provides information regarding areas of stenoses or occlusions. The suitability of veins as vascular conduits can also be evaluated simultaneously. Its results are, however, operator dependent.

Computed Tomography Angiography

Multi-detector computed tomography angiography (MDCTA) is a useful non-invasive angio-imaging tool. The abdominal aorta and its branches, and the peripheral arteries can be imaged rapidly. The use of MDCTA is accompanied by the risk of ionising radiation exposure and complications related to the use of iodinated contrast agents.

Magnetic Resonance Angiography

Contrast-enhanced magnetic resonance angiography (CEMRA), using agents such as gadolinium, is a useful alternative. This modality is not readily available and is costly. It is not recommended in patients who are claustrophobic, and in patients with metal implants e.g. pacemakers, cardiac valves, etc. In patients with renal dysfunction, gadolinium can cause nephrogenic systemic fibrosis.

Digital Subtraction Angiography

Conventional digital subtraction angiography (DSA) has evolved from being used as a purely diagnostic modality, and is currently

recommended as an interventional tool in patients who are suitable for endovascular procedures. It is an invasive procedure requiring arterial puncture and access using a sheath. The following complications are related to DSA:

- Ionising radiation exposure
- Anaphylaxis
- Contrast nephropathy
- Groin haematomas
- Pseudo-aneurysms
- Arterial dissection and thrombosis resulting in acute limb ischaemia
- Thrombo-embolism

In patients with renal dysfunction, CO₂ (carbon dioxide) angiography may be employed to image and treat PAD.

Patients not suitable for revascularization may be candidates for major amputations. In the absence of pulses the healing potential of designated amputation sites may be questionable. This is currently best addressed by measuring transcutaneous oxygen tensions (TcPO₂) using a special apparatus.

MANAGEMENT OF PAD

Medical management

There are 3 integrated treatment strategies:

- **Risk factor reduction / modification (secondary prevention strategies)**
 - Weight reduction
 - Dietary modification
 - Management of hypertension
 - Management of diabetes mellitus
 - Smoking cessation strategies
 - Antiplatelet therapy
 - Lipid lowering strategies
- **Symptomatic treatment**
 - Graded exercise programmes
 - Pharmacotherapy

- **Foot care**

Patients who are overweight should be encouraged to lose weight. Referral to a dietician may be required. There is little evidence for dietary manipulation in PAD but the Mediterranean diet is generally recommended.

Hypertension needs to be managed according to current / updated treatment guidelines. Anti-hypertensive agents that are more cardio-protective and metabolically stable are recommended. Angiotensin converting enzyme inhibitors (ACEI) have been shown in trials (viz. the HOPE trial) to reduce cardiovascular event rates. These vascular events were reduced even when used in normotensive patients. The ACEI appear to have clinical effects that extend beyond blood pressure reduction.

There is very little evidence of benefit for macrovascular disease following tight glycaemic control in diabetic patients. There does appear to be some benefit regarding limiting microvascular disease. An HbA_{1c} level target of < 7 is generally recommended.

Smoking cessation strategies

Counselling is an important, yet often neglected, aspect of smoking cessation. Even a 3 minute counselling session doubles the cessation rate. Current tobacco users willing to quit should be counselled using the five A's:

- **Ask** about their willingness to quit at every visit
- **Advise** on the benefits of complete cessation
- **Assess** their ability and commitment to quit
- **Assistance** should be provided with pharmacotherapy, and

- **Arrangement** of regular follow-up should be provided

Tobacco users not willing to quit may be frustrated by previous quitting failures or lack information about the harmful effects. They require a different approach and will respond poorly to direct confrontation. Counselling techniques involves informing them about the **five R's**:

- **Relevance** (why it is important to quit smoking?)
- **Risk** involved as a consequence of the on-going habit
- **Rewards** and benefit of cessation
- **Roadblocks** to quitting should be addressed (withdrawal, fear, weight gain and peer pressure)
- **Repetition** at every visit.

The health care provider has to emphasize the benefits and address threats to abstinence. The highest cessation rates are in patients whom have experienced a recent acute smoking related event: myocardial infarction, exacerbation of COPD or a threatened limb. This is the so-called '**teachable moment**'.

Every patient should be offered pharmacotherapy:

- Nicotine replacement strategies (nicotine gums, skin patches, inhalers, etc)
- Varenicline (partial $\alpha 4\beta 2$ nicotinic receptor agonist): a 12 week course is available.
- Bupropion (an anti-depressant drug): 150mg twice daily is recommended for 12 weeks.

Patients should be screened for psychiatric issues before prescribing Varenicline or Bupropion. Regular counselling, nicotine replacement therapy and either Varenicline or Bupropion is the current accepted strategy to aid smoking cessation

Anti-platelet therapy

There is currently good evidence for the use of antiplatelet agents in patients with established PAD. They have been consistently shown to reduce cardiovascular events and stroke rates even in patients with PAD.

- Aspirin (81 mg – 150mg daily) is generally prescribed. There is no benefit at higher doses but this increases gastrointestinal complications.
- Clopidogrel (75mg daily) has modestly better results than aspirin in patients with PAD, but are expensive and can only be recommended as a second line drug in patients with aspirin intolerance.

Lipid lowering strategies

There is currently overwhelming evidence that lipid lowering drugs, especially the statins (HMGCoA receptor blockers), is associated with a significant reduction in cardiovascular events and stroke rates in patients with established PAD.

- Simvastatin: commenced at 40mg nocte.
- Other statins are available but are more costly (pravastatin, atorvastatin, etc)
- Fibrates may be considered in certain circumstances (e.g. high triglycerides, etc)
- Cholesterol-binders (e.g. cholestyramine, Ezetimibe, etc)
- Dietary manipulation

Statin therapy should target a LDL-cholesterol level < 2.6 mmol/L in low-intermediate risk patients and < 1.8 mmol/L in high risk patients (previous MI, diabetics, PAD, etc)

Statins have both lipid lowering and **pleiotropic** (non-lipid lowering) effects viz. decreases inflammation, plaque stabilisation, decreases thrombosis, decreases platelets adhesiveness and improvement in endothelial dysfunction.

Exercise therapy

Graded exercise therapy has been consistently shown to improve clinical outcomes in patients with PAD. Claudication distance improves with exercise therapy. A supervised exercise programme has been shown to be more effective than unsupervised exercise treatment programmes. Exercise may include walking, swimming, cycling, etc. A programme incorporating 30 minutes of exercise 3 times a week, building up to 1 hour 3 times a week is considered adequate. Diabetic patients with foot problems need proper footwear when exercising. Studies have shown that exercise therapy is more durable than balloon angioplasty without exercise.

Pharmacotherapy for claudication

With the exception of statins which have been shown to improve claudication, very few drugs have shown benefit in patients with claudication.

- **Cilostazol** is a phosphodiesterase type 3 inhibitor that increases cyclic adenosine monophosphate (cAMP) levels. It also has vasodilatory and platelet inhibitory effects.
- **Naftidrofuryl** has been available for treating intermittent claudication for over 20 years in several European countries. It is a 5-hydroxytryptamine type 2 antagonist and may improve muscle metabolism, and reduce erythrocyte and platelet aggregation.
- **L-carnitine and propionyl-L-carnitine** interact with skeletal muscle oxidative metabolism, and these drugs are associated with improved treadmill performance.

There is very little evidence for the use of other drugs, including **Pentoxifylline**, for the treatment of claudication. Vasodilators may be

used in patients with Reynaud's symptoms.

Surgical management

Indications

- Severe life-style limiting claudication
- CLI with rest pain
- CLI with ischaemic foot / leg ulcers
- CLI with gangrene
- To facilitate stump salvage in a patient requiring a major amputation

Operative treatment

These should be considered in patients who are fit for surgery and who have a longer life-expectancy requiring a more durable procedure.

Aorto-iliac procedures include:

- Direct aortic reconstruction (aorto-femoral bypass graft)
- Extra-anatomical reconstruction (femoro-femoral crossover, ilio-femoral bypass, Axillo-bifemoral bypass, etc)

These procedures generally employ prosthetic grafts

Infra-inguinal procedures include:

- Femoro-popliteal bypass
- Femoro-distal bypass (onto crural vessels)
- Pedal artery bypass

These procedures have better outcomes when a vein conduit is used compared to prosthetic conduits for bypass.

Complications of surgery:

1. Systemic complications generic to all major surgical procedures (e.g. pulmonary atelectasis, myocardial infarction, renal failure, etc)
2. Wound sepsis
3. Wound healing complications
4. Haemorrhagic complications
5. Graft occlusion (early or late)
6. Graft sepsis (early or late)
7. Complications related to aortic surgery
 - Bowel injury
 - Ureteric injury

- Venous injuries (IVC, iliac veins, renal veins, etc)
 - Erectile dysfunction
 - Ischemic sigmoid colitis
 - Paraplegia (rare)
 - Incisional hernia
8. Nerve injury
9. Deep vein thrombosis

Endovascular treatment

Endovascular techniques are considered in patients with endo-suitable aorto-iliac disease irrespective of indication. The results are comparable to open surgery.

Only patients with CLI or are not fit for open surgery or have an inadequate vein conduit should be considered for infra-inguinal endovascular therapy. These procedures are less durable than bypass grafting. Iliac interventions have better results than femoral or tibial interventions.

- Balloon angioplasty
- Peripheral stenting
- Drug eluting balloons
- Drug-eluting stents
- Covered stents
- Percutaneous atherectomy devices

Complications of endovascular therapy

- Access related complications (haematoma, pseudo-aneurysm, etc)
- Arterial dissection
- Arterial perforation / rupture
- Thrombo-embolism
- Restenosis following balloon angioplasty
- In-stent stenosis
- Stent thrombosis / occlusion
- Stent malposition
- Stent fracture

SCREENING FOR PAD

If we are to improve the life-expectancy and reduce the cardiovascular risk in patients with PAD, and reduce the amputation rates, then a programme of early detection is essential to implement cardiovascular risk reduction

strategies and implement evidence based optimum medical treatment in all patients with established PAD

Who should be screened?

- All patients > 55 years
- All patients < 55 years with risk factors for atherosclerosis
- Diabetic patients

Screening should include:

- Palpation of foot pulses
- Arterial Doppler pressure measurements and ABI testing

AMPUTATIONS

Amputations in patients with PAD may be classified as:

- **Primary** (no revascularization was needed or performed)
- **Secondary** (following an attempt at revascularization)

Lower extremity amputations may also be classified anatomically into

- **Foot amputations.** These are also referred to as **minor amputations**, considering that no prosthesis is required, for mobilization, after surgery. Foot amputations may be performed after revascularization because of gangrene or sepsis. This is referred to as "limb salvage".
 - **Toeectomy** (removal of the toe with preservation of the metatarso-phalangeal joint)
 - **Ray amputation** (The toe is removed together with the metatarso-phalangeal joint and the distal part of the metatarsal bone)
 - **Transmetatarsal amputation (TMA)** - All the toes and the distal portions of all the metatarsal bones are surgically removed)

- **Ankle level amputations** (Syme`s, Lisfranc`s, Chopart`s, etc). These have been traditionally performed previously in diabetic patients without PAD. They have fallen out of favour in recent times, because of suboptimum rehabilitation, and are rarely performed.
- **Major amputations.** These patients require a prosthesis to mobilize with. Some patients may require revascularization to facilitate stump healing. This is referred as “stump salvage”.
 - **Below knee amputation**
 - **Through knee amputation**
 - **Above knee amputation**
 - **Hip disarticulation**

Indications

Only **select patients with CLI** and intractable rest pain, and/or tissue loss, may be considered for **major primary amputation.**

- Patients that have non-reconstructable vascular disease on imaging.
- Patients who have significant co-morbidities precluding any form of revascularization (e.g. refractory cardiac failure, end-stage renal failure, major stroke, etc)
- Patients with a fixed-flexion deformity of the knee
- Patients who are not ambulant (confined to a wheel chair or bed-ridden)
- Patients who have major tissue loss of the foot precluding limb salvage (e.g. gangrene extending beyond the TMA level)

Healing following amputation at a particular level may be of concern if

one cannot palpate a pulse proximal to that level. Stump healing at this level may be assessed using:

- Lower extremity Doppler pressures
- The information from angio-imaging (the presence of an internal iliac artery or profunda femoral artery, collaterals, etc)
- Thermography (generally not used in practice)
- Xenon skin clearance (a radio-isotope study; generally not used in practice)
- Estimation of **transcutaneous oxygen tension (TcPO₂)** levels. This is currently the most useful test to guide level of amputation.



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