Peripheral Vascular Disease

History
- Brought on by exercise and relieved by rest (not true of neurogenic pain)
- Affects a muscle group(s)
- Reproducible on exercise (claudication distance)
- Musculoskeletal pain – worse on m’ment / weight bearing, radiates, not repro by exercise
- May be co-existing cardiac dx – ask about breathlessness, angina etc
- Risk factors – smoking, diabetes, hypertension, hypercholesterolaemia, AF….
- Beta blockers may make it worse

Examination
- Inspection: colour, hair loss, ulcers, surgical scars, venous guttering (reduced filling)
- Palpation:
  - temperature (compare to other side)
  - capillary refill
  - Buerger’s Test (leg to 45deg for 2mins foes pale, drop over side goes hypereamic, cyanotic)
  - Pulses: femoral, popliteal, posterior tibial, dorsalis pedis (consider radials, brachials)
- Auscultate for bruits over femorals and abdominal aorta
- Consider power and sensation to rule out neuro problem

Investigation
- Ankle-brachial pressure index (ABPI with u/s probe for both pulses. Should be 1:1, below 0.4 is critical)
- Post exercise ABPI when the claudication point. A fall in ABPI shows significant disease
- Doppler duplex for large vessels
- Angiogram (able to visualise smaller vessels – also CT + MR angio)
  - Complications: 1% amputation risk – 2% with angioplasty. Contrast toxicity
  - Look for Inflow and Outflow: should be 4 vessel runoff from superficial femoral: popliteal, anterior tibial, posterior tibial, common peroneal – at least one patent needed for a bypass
- FBC, U+E, glucose, lipids, INR/co-ag

Treatment
- Conservative: lifestyle: stop smoking etc, supervised exercise (stimulates collateral formation)
- Medical: antihypertensives, statins, antiplatelets, nifedipine
- Surgical / Radiological – bypass or angioplasty
- Lumbar sympathectomy (blocks sympathetic chain chain causing dilatation) – last resort resort, often prior to amputation, may relieve rest pain for a while

Acute Limb Ischaemia
- History of AF / aneurysm / recent MI / infective endocarditis
- Not usually a background history of claudication
- 6 P’s – pain, pale, pulseless, paraesthesia, paralysis, perishing cold
- Treatment:
  - O2, morphine, aspirin
  - Bolus 5000 units of heparin
  - Surgery – embolectomy
  - Locate source of embolus
  - Later: fragmin as inpatient then warfarin (INR 2-3 for life
Lower Limb Arterial Disease

Fontaine I: asymptomatic lower limb arterial disease. Fontaine IIa: claudication > 200m. Fontaine IIb: claudication < 200m. Fontaine III: rest pain. Fontaine IV: ulceration and/or gangrene.

Treatment of intermittent claudication is usually conservative until critical ischaemia ensues or with severe lifestyle limitations. Stop smoking, treat hypertension, diet/statins, exercise. Intervention is by angioplasty or surgery.

Patients with critical limb ischaemia (rest pain of 2/52 or ischaemic tissue loss) present with: rest pain, gangrene, ulceration or a combination of these.

Arterial Ulceration

The following factors predispose to ulceration:
- Neuropathy (e.g. diabetes)  Poor nutrition
- Severe ischaemia  Prolonged-bed rest
- Infection  Trauma

An ulcerated lesion requires a greatly enhanced blood flow to heal; therefore many ulcers fail to heal where CLI is present.

Acute limb ischaemia is caused by embolism, thrombosis of a peripheral aneurysm, trauma, IVDU etc. Presents with the 6 P's: Pain, Pulseless, Pallor, Parasthesia, Paralysis, Perishing cold. Treatment: pain control, anticoagulation, revascularisation (thrombolysis, embolectomy, bypass, PTA) or amputation.

Venous Ulceration

- Most common in pts with constant ambulatory venous hypertension (from damaged valves – see varicose veins bit). Accounts for 80-90% of all leg ulcers. Pathophysiology uncertain but probably due to a combination of factors leading to skin hypoxia.
- Most frequently medial aspect of calf.
- When it occurs, 1st use Doppler to check for any coexisting arterial component.
- Healing is often very slow. Treat by application of compression stockings over appropriate dressings to improve venous flow and reduce stasis. Elevate the foot. Treat any local infection. Occasionally a skin graft may be required.
- Once healed, a patient needs to wear compression stockings for life to prevent recurrence.
- Other causes of lower-limb ulceration: arterial, neuropathic, malignant.

Arterial ulcers are usually on the toes or lateral aspect of the calf above the malleolus. Its important to differentiate from venous, as compression makes them worse.

Neuropathic ulcers occur on weight/pressure bearing areas due to reduced sensation e.g. diabetes.

If an ulcer does not heal, consider malignancy and take biopsy.
Varicose Veins

Common, affecting 50% population at some time in their lives. 80% are women. They are rare in many non-western countries. They can be primary (idiopathic) or secondary – caused by deep venous insufficiency (damage to valves, after a DVT, fracture, surgery, pregnancy, prolonged bed rest).

Pathophysiology: superficial veins (long + short saphenous veins) drain skin + subcut tissues. Deep veins drain muscles of the leg. The systems are connected at the sapheno-femoral and sapheno-popliteal junctions and by perforating veins with one-way valves allowing blood flow from superficial to deep veins.

High pressure is prevented in superficial veins by the ‘muscle pump effect’ and the valves preventing backflow. Failure of perforator or deep venous valves allows backflow and increases superficial venous pressure causing dilatation (varicosities).

Clinical features + examination: cosmetics, aching, discomfort or sensation of heaviness in legs on standing, haemosiderin skin staining. No severe pain unless evidence of superficial phlebitis. Ask about previous history of DVT.

Examination:

- Inspect for ulcers, brown staining (heamosiderin), eczema, thin skin
- Examine anterior from thigh to medial calf (long saphenous) and posterior calf (short saphenous) for vein tenderness (phlebitis) and hardness (thrombosis)
- Cough impulse at sapheno-femoral junction = incompetence
- Percuss vein and feel distally for thrill (interrupted by competent valves)
- Trendelenberg Test for sapheno-femoral incompetence: lie pt down, raise leg to empty vein, place 2 fingers on s-f jcn (5cm below + medial to femoral pulse). Keep fingers in place and ask pt to stand – if varicosities controlled, they will not rapidly refill – release fingers to confirm they then refill. This shows s-p incompetence and that the Trendelenberg op (s-f disconnection) should help. If they do refill rapidly, the incompetence is at a lower level.
- Tourniquet test: as above, but use tourniquet instead of finger and move it down the leg until area of incompetence is identified.
- Perthes test: tourniquet around mid-thigh with pt standing, then walks for 5 mins. If s veins collapse below tourniquet, the deep veins are patent and communicating veins are competent. If unchanged, both sphenous and communicating veins are incompetent. If veins more prominent + pain, the deep veins are occluded.
- Can use Doppler ultrasound to listen to flow in incompetent valves

Treatment: reassurance (avoid prolonged standing, loose weight, walk – muscle pump), compression stockings, sclerotherapy (injection to ablate the superficial varicosities – may need multiple, not possible for major varicosities, risk of damage to deep veins + ulceration), surgery (often good results from sapheno-femoral ligation, above knee saphenous stripping or multiple avulsion).

Complications: uncommon – rarely ulceration, superficial thrombophlebitis is more common in severe disease – may be mistaken for DVT. Use anti-inflammatory’s and support bandages. If thrombophlebitis extends to s-f junction, there is increased risk of PE.