VARICOSE VEINS:

These are dilated tortuous veins. They are divided into primary and secondary.

Primary varicose veins are the most common and are often familial. They are possibly due to weakness of the superficial vein wall that allows dilatation of the valve ring allowing the valve to become incompetent with retrograde flow. This then leads to increased venous pressure and further valve failure. Other contributory factors include prolonged standing, family history, hormonal factors (more common in pregnancy - progesterone has an effect on the vein wall), ageing.

Secondary varicose veins may be classified as follows:

- Obstruction to venous outflow, e.g. pregnancy, fibroids, ovarian cysts, abdominal lymphadenopathy, pelvic cancer, iliac vein thrombosis or retroperitoneal fibrosis

- Valve destruction, e.g. DVT

- High flow and pressure, e.g. AV fistulae.

Symptoms and signs:

Tortuous dilated veins of the great or small saphenous system. Aching discomfort worse towards the end of the day, relieved by sitting with legs elevated. May present with complications (see below). Examine the patient standing up and assess the site and size of the veins. Palpate for defects in the fascia. Check the state of the skin and subcutaneous tissue. Carry out Trendelenburg's test to assess the site of incompetent

perforating veins. This is carried out with the patient supine, the leg being elevated and the tourniquet applied just below the saphenofemoral junction. The patient then stands erect for 30 s. If the saphenous fills rapidly from below with the tourniquet in place, the perforators lower the legs are incompetent. If the long saphenous vein fills rapidly from above following removal of the tourniquet, the valve at the saphenofemoral junction is incompetent. Repeat at different levels down the leg to determine the level of incompetent perforators.

In practice, Trendelenburg's test is rarely used nowadays and has been replaced by hand-held Doppler machine (see below). If a swelling is apparent over the saphenofemoral junction (saphena varix), the diagnosis should be confirmed by placing the hand over the swelling and tapping the varicose veins lower down the legs. A palpable thrill at the groin will confirm the presence of a saphena varix. Auscultation over the veins should be carried out to exclude arteriovenous fistulae. Perthes test may be performed to exclude deep venous obstruction (tourniquet applied below the saphenofemoral junction and the patient has to exercise on the spot. Severe 'bursting' pain indicates obstruction of the deep venous system.) but this is usually assessed by duplex Doppler nowadays.

Investigations:

Diagnosis usually made on clinical grounds with hand-held Doppler assessment. In difficult or recurrent cases, Duplex ultrasound may be used. If a secondary cause is suspected, then abdominal ultrasound can be performed.

Complications:

Superficial thrombophlebitis. Hemorrhage. Varicose eczema. Varicose pigmentation due to haemosiderin deposition. Lipodermatosclerosis. Chronic venous ulceration. Long-standing venous stasis ulcers may become malignant (Marjolin's ulcer).

Treatment:

Mild varicosities - compression stockings and periodic elevation of the legs. Small varicosities below the knee are suitable for injection with a sclerosing agent and compression bandaging worn for 2 weeks. This treatment can also be used for remaining varicosities after surgery. Surgery is indicated for saphenofemoral incompetence, saphenopopliteal incompetence, or thigh perforators. Mark out the varicose veins prior to surgery. Surgery includes saphenofemoral disconnection and stripping for incompetence of the great saphenous vein. Stripping is generally only performed to knee level. Saphenopopliteal disconnection can be used for varicosities involving the small saphenous system. Stripping is not performed, as this will risk damage to the sural nerve. Stab avulsions through tiny incisions at previously marked sites are used for residual veins. Compression bandaging is required postoperatively.
Encourage early mobilization. Walk for 5-10 min every hour during the day. Sit with legs elevated postoperatively.
Endoluminal techniques are being increasingly used, which result in quicker recovery and reduced postoperative pain. Techniques include laser and radiofrequency ablation.

CHRONIC VENOUS INSUFFICIENCY:

is caused by persistent and sustained ambulatory venous hypertension. Causes include:

• Muscle pump dysfunction. e.g. fused ankle in arthritis or neurological impairment after CVA .

• Abnormal valve function which may be primary (affecting both the superficial and deep veins) or secondary after DVT with destruction of deep valves (known as post-phlebitic limb)

• Congenital valve absence (very rare).

Along with changes in the large veins that result in venous hypertension, there are abnormalities at the microcirculatory level. These include:

• White blood cell trapping. WBCs become trapped in capillaries and cause endothelial activation and release of inflammatory cytokines which increase vascular permeability and contribute to the presence of a fibrin cuff together with release of proteolytic enzymes and free radicals. In addition they cause tissue ischemia by blocking capillaries.

• Fibrin cuff. An increase in venous pressure is transmitted to the capillaries and opens endothelial pores resulting in molecules such as fibrinogen moving into the interstitial space. This polymerizes to fibrin and may act as a barrier to oxygen diffusion causing local tissue ischemia.

Symptoms and signs:

• Peripheral edema. This gets worse towards the end of the day and tends to improve with elevation

• Varicose veins

• Venous eczema and brawny induration of the skin

• Venous pigmentation associated with haemosiderin deposits in the tissues

• Lipodermatosclerosis.

• Venous stasis ulceration, which is common in the area of the medial malleolus:

• There is often severe pain associated with the swelling and ulceration

• Venous claudication. This indicates obstruction of the deep venous system. It described as a 'bursting' pain which comes on with exercise and takes to settle.

Investigations:

• Hand-held Doppler - to assess superficial venous incompetence and can assessment of arterial system (via ABPIs)

• Duplex Doppler - allows assessment of both superficial and venous systems

• Venography (very rarely used).

Treatment:

Difficult to treat and patient rarely gets complete relief.

Medical: Advise patient to avoid long periods of standing and to sit with legs elevated. Graduated compression stockings or 4-layer bandaging (ensure ABPI >0.8 before using compression). Treat venous stasis ulcers by reducing swelling of the leg by bandaging, excision of necrotic tissue and control of any cellulitis with antibiotics. Clean ulcers regularly. Emollients for venous eczema.

Surgery: Options for surgical intervention include:

• Superficial venous surgery. As for management of varicose veins and includes ligation of the SFJ and stripping of the GSV. Useful in isolated superficial combined deep and superficial reflux. Has been shown to aid healing of ulcers

• Perforator vein surgery.

• Deep vein surgery. Rarely indicated. Options include veno-venous bypass, transposition and vein valve transplantation

• Ulcer debridement and skin grafting

• Amputation. Carried out if ulceration becomes complicated or is intractable.

THROMBOPHLEBITIS:

This is characterized by a local inflammation of a segment of superficial vein. The vein is tender, red and feels like a 'cord'. The causes are shown in Table 15.3. Treatment is usually symptomatic and depends on the underlying cause. However, when it involves the great Saphenous vein at the saphenofemoral junction, this has a risk of thromboembolism and is a DVT with anticoagulation or urgent ligation.