Venous disorders

Introduction

Venous disorders are very common and especially affect the lower limb. Twenty per cent of the population suffer with varicose veins and 2 per cent have skin changes which may precede venous ulceration.

Anatomy of the venous system in the limbs

Arterial blood flows through the main axial arteries to the upper and lower limbs. It returns via the deep and superficial veins. In the upper limb the superficial veins are more important in carrying blood back to the heart. In the lower limb, the superficial veins carry only about 10 per cent of the blood, while the remainder passes via the deep veins. The superficial veins lie superficial to the muscle fascia of the limb. The principal superficial veins in the leg are the long and short saphenous veins. In the arm, the cephalic and basilic veins are the principal superficial veins.

Interestingly, venous diseases occur much more frequently in the lower limb than in the upper limb, and most often in the superficial veins. The deep veins of the lower limb may be the site of life threatening venous thrombosis or venous valvular incompetence resulting in leg ulceration. The superficial and deep veins join at a number of points. The short saphenous vein terminates at the saphenopopliteal junction (SPJ) and the long saphenous vein at the saphenofemoral junction (SFJ) in the groin. Here the flow in the superficial veins joins that in the deep veins. There are, in addition, a number of places in the calf and thigh where flow in the superficial veins may also join that in the deep veins. These are the ankle, calf and thigh communicating or perforating veins

Venous pathophysiology

 Blood flows into the leg because it is pumped by the heart along the arteries.

 By the time it emerges from the capillaries it is at a low pressure (about 20 mmHg), but this is enough for the blood to return to the heart.

 Valves prevent the flow of blood from the deep to the superficial system.

 The venous pressure in the foot vein on standing is equivalent to the height of a column of blood, extending from the heart to the foot. However, the same is true of the arterial system so that on standing the arterial blood pressure at the ankle rises by 80—100 mmHg, depending on the height of the person. So the blood continues to circulate, even in the absence of muscle activity.

 We also have a sophisticated series of muscle pumps that act as peripheral hearts in the venous system.

 The direction of venous blood flow is controlled by the venous valves. The pressure within the calf compartment rises to 200—3 00 mmHg during walking and this is more than enough to propel the blood in the direction of the heart. During the muscle relaxation phase, the pressure within the calf falls to a low level and blood from the superficial veins flows through the perforating veins into the deep
veins. The consequence of this is that the pressure in the superficial veins falls during walking.
 Normally the pressure in the superficial veins of the foot and ankle falls from a resting level of 80—100 mmHg to about 20 mmHg.This ability to reduce the pressure in the superficial venous system is crucial to the health of the lower limb. Patients with damage to the veins in whom the superficial venous pressure does not fall during exercise may develop varicose eczema, skin damage and, eventually, leg ulceration.

Venous Incompetence - varicose Veins

One of the most common problems with the veins of the leg is failure of their valves. This occurs frequently in the superficial venous system resulting in varicose veins.

The mechanisms that cause the superficial vein valves to fail have not been fully established. What appears to happen is that first a small gap appears between the valve cusps at the commisure (where the valve leaflets join the vein wall). This gap widens and more reverse flow (venous reflux) is allowed. The valve cusps degenerate and holes develop in them. Eventually they disappear completely. The vein below the valve responds by dilating. Varicose veins may eventually reach five times their usual size if left to develop for long enough.

Varicose veins are thought to develop more often in people who stand during their work. They often develop during pregnancy under the influence of oestrogen and progesterone which cause the smooth muscle in the vein wall to relax.

Clinical features

They may either give no symptoms or cause aching and discomfort in the legs. Varices are recognised as tortuous dilated veins in the leg, but physiologically speaking a varicose vein is one which permits reverse flow through its faulty valves. Patients may develop much smaller varices. These range from 0.5-mm diameter vessels in the skin, which are commonly referred to as thread veins or dermal flares, and are usually purple or red in colour.

Cosmetic appearance is unsatisfactory. Patients may also report aching especially on standing, itching, ‘restless legs’ and ankle swelling. The severity of the symptoms is unrelated to the size of the veins, and is often more severe during the early stages of development of varices.

Complications of varicose veins

1) Thrombosis, which is referred to as superficial thrombophlebitis. Usually this remains in the superficial veins and may cause considerable discomfort.

2) Spectacular haemorrhage can occur when large superficial varices are damaged. This is easily controlled by lying the patient down, elevating the leg and applying a compression bandage.

3) The most serious problem is venous ulceration which complicates varicose veins.

Venous incompetence — deep vein incompetence

Valvular incompetence of the deep veins may develop in the same way as in the superficial venous system, with the degeneration of the valve cusps resulting in reverse flow in these veins. In other patients it may develop following a deep vein thrombosis. In the leg the long and short saphenous veins may act as collateral channels and may double in size to accommodate the additional blood flow. In patients with chronic iliac vein occlusion large suprapubic or abdominal varices may be seen carrying the collateral flow.

Clinical features of deep vein incompetence

1) The calf muscle increases in size, apparently in response to the greater work in returning blood from the leg.
2) There may be some ankle oedema
3) A proportion of patients develops skin complications. These may range from mild eczema to severe ulceration.
4) An early sign of skin injury is brown pigmentation due to haemosiderin deposition in the skin. This occurs because the high venous pressures cause red blood cells to be forced out of capillaries in the skin where their haemoglobin breaks down to form haemosiderin.
5) A later and more serious stage is lipodermatosclerosis in which palpable induration develops in the skin and subcutaneous tissues.
6) Atrophie blanche may also develop. In this condition the superficial blood vessels are lost from the skin and white patches develop. These indicate that the skin has been severely damaged by the venous valvular incompetence. Venous ulceration may develop in these areas.