

UNDERSTANDING CHRONIC VENOUS HYPERTENSION

Chronic venous hypertension accounts for about 70% of ulcers on the lower limb. It is important to understand the underlying pathophysiology of the condition in order to rationalise assessment and treatment approaches.

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Figure 1. A venous leg ulcer.

Venous disease is the underlying cause of venous ulceration (Figure 1). Although patients may cite trauma as the cause of their ulcer it is in fact a wound complicated by a disease process in the lower leg (Rainey, 2002). It is important for the practitioner to understand the pathophysiology of venous disease as this will enable them to make rational treatment decisions and explain the importance of self care and compression therapy to the patient. This article aims to explain the pathophysiology of chronic venous hypertension and link this to clinical signs and the symptoms experienced by the patient. The mechanical and cellular processes are very complex and the objective of

this article is to highlight key areas in order to help healthcare professionals understand and explain to patients the complex processes involved in venous hypertension and leg ulcer development.

Veins in the lower leg

Veins carry blood back to the heart and the blood flows at a relatively low pressure compared to arterial blood flow (which carries the blood away from the heart under high pressure) (Tortora and Grabowski, 2000). The arteries branch into smaller and smaller vessels, until the blood flows into capillaries. The walls of capillaries are only one cell thick (Tamir, 2002) so that oxygen, glucose and other

substances can pass through them to nourish the tissues.

Veins are thinner than arteries and have three layers: the tunica

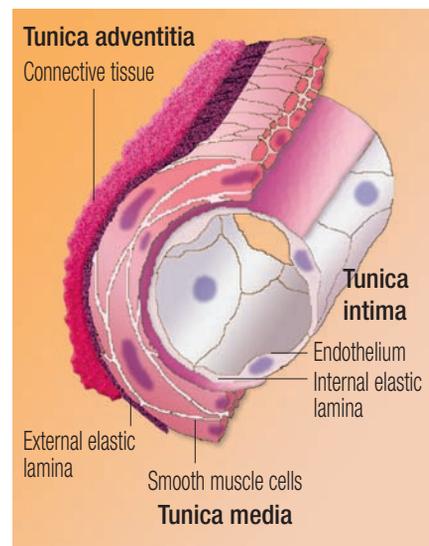


Figure 2. Structure of the vein.



Figure 3. Venous blood flows up the leg and backflow is prevented by one-way valves.

adventitia on the outside, tunica media (middle layer) and the tunica intima, an inner layer of endothelial cells lining the vessel (Figure 2) (Waugh and Grant, 2001). The waste materials of metabolism, such as carbon dioxide and lactic acid, filter in the opposite direction into the capillaries which then join to form slightly larger vessels (venules, or tiny veins) which in turn join up to veins.

The venous system is made up of a deep system and a superficial system, which are joined by perforator (joining) veins (Figure 3). The deep veins of the leg are the femoral, popliteal, anterior and posterior tibial veins, while the superficial veins are the great and small saphenous

(previously called long and short veins [Waugh and Grant, 2001; Meissner et al, 2007]). Deep veins are held within the muscle fascia of the leg while superficial veins are less supported and are nearer the surface of the leg (Waugh and Grant, 2001). These are the veins which are visible, e.g. on the foot.

The perforator veins are named as such because they 'perforate' the fascia layer surrounding the muscles of the legs, linking the superficial and deep veins. Perforator veins allow venous blood to flow from the superficial veins into the deep veins with the highest number of these joining veins being near the calf muscle (Meissner et al, 2007).

All veins in the leg have valves to ensure that venous blood travels back to the heart or from the superficial veins back to the deep veins via the perforators. These valves are particularly important in the lower leg as the blood needs to flow upwards a considerable distance to the heart especially if standing upright. If the valves do not work in the perforator veins, blood is pushed out into superficial veins, increasing the pressure in these vessels.

Venous flow in the lower leg

Venous blood returns to the heart mainly via the deep veins, which are bigger and stronger than the superficial veins, and there are three key mechanisms by which this blood flow is encouraged:

- ▶▶ Gravity: blood from the lower leg has to travel a long way against gravity to return to the heart; therefore when the body is laying flat or when the legs are elevated the flow

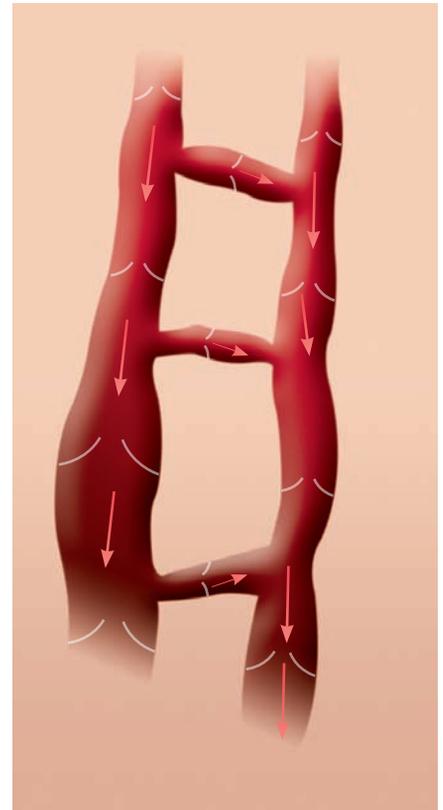


Figure 4. Chronic venous hypertension: backflow of venous blood increases the blood volume in the leg veins forcing the one-way valves apart.

meets less resistance than when standing

- ▶▶ Breathing: On breathing in there is a negative pressure created in the chest cavity. The abdominal pressure increases as the diaphragm lowers and both mechanisms encourage venous return
- ▶▶ The calf-muscle pump: this will now be discussed in more detail.

The calf-muscle pump

When blood is pumped into the arteries by the heart, it is pushed forward under high pressure. Only a little of this pressure is left once it has passed through the capillaries into the veins so the action of the muscles provides a pumping action that helps to push the blood up through

the veins. This muscle pump is particularly important in the legs, because on standing, blood has to travel a long way, against gravity, to return to the heart.

As the calf muscle contracts, the valves in the deep veins are squeezed and they open to allow the blood to travel upwards. As the calf muscle relaxes the valve closes and creates a negative pressure as the section between valves empties. This negative pressure draws blood from the superficial veins, through the perforators to refill the chamber ready for the next contraction of the calf muscle (Meissner et al, 2007).

In the lower limb the normal venous pressure may range from about 100mmHg when standing still, falling to about 22mmHg when walking (Meissner et al, 2007). These pressure changes also occur on ankle flexion and during exercises where the heel is raised and the person stands on tiptoe. These changes in pressure on activity illustrate the key role of exercise in the management of venous hypertension. Even moderate exercise in patients with reduced mobility can still be undertaken.

Chronic venous hypertension

The valves of the leg can become damaged by trauma, surgery to the lower leg that has involved cutting into veins, pregnancy, obesity, increased vein capacity and thrombosis. If the valves are incompetent they will allow backflow (reflux) of venous blood in the deep vein and the volume of blood in the lower leg will increase. This increased load will cause a rise in the pressure

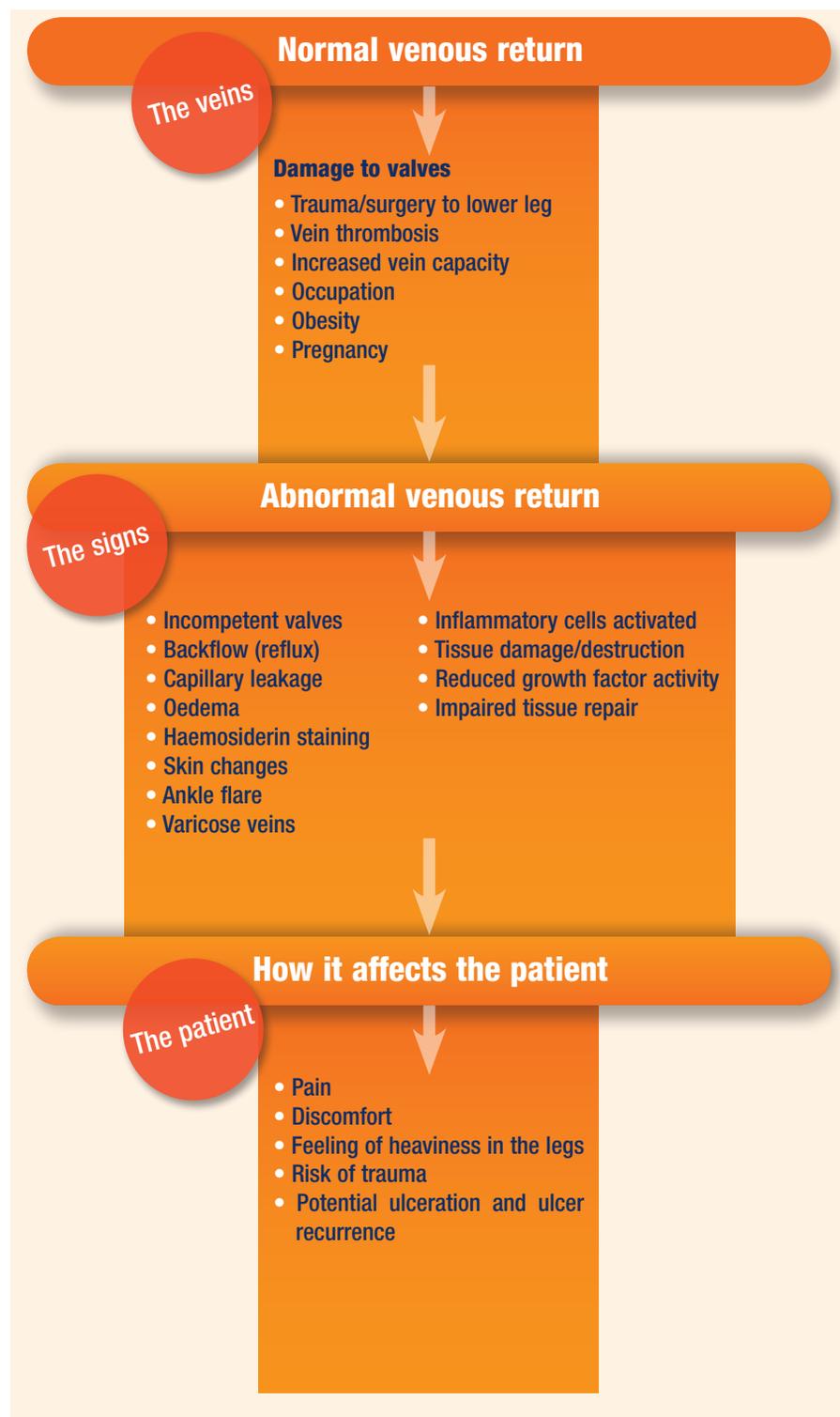


Figure 5. Pathophysiology of chronic venous hypertension.

on the vein wall and will affect other parts of the venous system namely the perforators and the superficial system. As these veins stretch their valves here will not close properly and the volume of blood — and therefore the venous pressure — will

increase. As the valves are unable to prevent backflow of blood, chronic venous hypertension results (Figures 4 and 5). Chronic venous hypertension is the main underlying cause of venous leg ulceration (Morison and Moffatt, 2004).

Table 2

Signs of chronic venous hypertension

Signs	Pathophysiology	Common related signs and symptoms
Ankle flare	Chronic venous hypertension causes the capillaries to swell.	Visible capillaries through the skin, most often on the medial (inner) malleolus (ankle). The area may also become itchy. 
Varicose veins	Veins which are continually stretched lose elasticity and the tunica media layer of the vein wall becomes fibrosed. Increased volumes of blood cause the stretching and the valves can no longer close properly which increases the blood volume further because of backflow. The great and small saphenous and anterior tibial veins are most affected (Waugh and Grant, 2001).	Prominent veins, varicosity may be palpable before becoming visible. Itch, pain especially after standing for long periods. Heavy bleeding if the vein is punctured. 
Oedema	Swollen capillaries allow fluid to leak into the tissue. Normally excess tissue fluid would be reabsorbed back into the capillaries but this may not be possible as the capillary is already congested due to backflow of blood in the veins (Waugh and Grant, 2001)	Pain, discomfort, difficulty walking and finding suitable footwear, skin trauma, fluid leakage through skin, blistering. Risk of infection such as cellulitis.
Varicose eczema	Inflammation, poor tissue nutrition and hydration. Link with venous hypertension is not fully understood.	Dry flaky skin, weeping skin, caused by sensitisers. Itch, skin trauma, skin sensitivities. The skin initially is red and itchy, perhaps with scales and weeping blisters. As the condition becomes more chronic the skin can become much dryer and flaky, even with thick layers of dry scale known as hyperkeratosis (Cameron, 2007).
Lipodermatosclerosis	Prolonged inflammation, tissue fibrosis.	Dry, hard, woody skin. Changes in lower leg shape known as the 'inverted champagne bottle'.
Haemosiderin staining	Leakage of red cells from stretched capillaries stain the tissue a brown or rusty colour (Dealey, 2005).	Dark staining particularly in the area of the medial malleolus.

It is not just valve incompetence that leads to venous hypertension but also vein obstruction. After a venous thrombosis the clot is broken down but may not completely resolve and the residue may become fibrous causing an obstruction. If this also happens to be in the area of a valve it may stop the valve working properly

as it becomes tangled in the fibrosis (Meissner et al, 2007).

Effects of chronic venous hypertension

The effects of chronic venous hypertension on the patient are outlined in *Table 1*. An early sign of chronic venous hypertension is oedema. It is caused when increased pressure in congested

veins prevents excess tissue fluid being drawn back into the capillaries (Waugh and Grant, 2001).

Normally there is a dynamic balance between the fluid in the tissue and in the capillary; as tissue pressure rises fluid is absorbed into the capillary and as capillary pressure rises, fluid is

pushed out to the tissue. When there is venous congestion this mechanism is less likely to work, as the pressure in the capillary does not fall sufficiently to allow more fluid to be absorbed from the tissue.

When fluid leaks from the capillary into the tissue, protein is one of the molecules that leaks out. These plasma proteins then attract more fluid from the capillaries due to tissue osmotic pressure (Waugh and Grant, 2001), which means the tissue oedema increases unless there is some kind of intervention to help prevent it such as compression therapy or leg elevation (Nicolaidis, 2005). Compromised lymph drainage exacerbates the problem of oedema, as the lymphatic system is impaired in venous disease, with or without ulceration (Williams and Mortimer, 2007).

Skin changes

Congestion means that nutrients do not get to the tissues including the skin and this can result in dryness, eczema and so on (*Table 1*). Skin changes are very common in venous disease. Herrick et al (2002) state that up to 94% of people with venous disease (including deep vein thrombosis) have skin changes. There does not, however, seem to be a relationship between the degree of disease and these changes, which suggests that there is much that is not yet understood about the progression and causes of venous disease.

Lipodermatosclerosis is hardening of the skin with a 'woody' feel and appearance, sometimes called induration. The most

common site of this is around the medial malleolus. The skin will feel hard with no 'give' in the tissue and may be extremely tender and is very vulnerable to trauma. As this condition develops the skin changes result in thickening that constricts the ankles and together with the build up of oedema and tissue fibrosis results in a change in the shape of the lower leg; referred to as an 'inverted champagne bottle' shape. Sometimes there is a condition called atrophie blanche visible, which are tiny white, scarred areas (Herrick et al, 2002) that are very painful and vulnerable to trauma. Haemosiderin staining is visible on the skin as a brown or rusty discoloration due to the leakage of red blood cells from stretched capillaries.

Varicose veins

Venous congestion will give rise to varicose veins and a condition commonly referred to as ankle flare (*Table 1*) where small capillaries and venules are visible under the skin mostly around the inner malleolus. Varicose veins may affect 10–20% of the adult population and 3% of those with varicose veins are thought to experience leg ulceration (Morison and Moffatt, 1999), however, not everyone with a leg ulcer will also have overt signs of varicose veins. Interestingly, a long-term study of children found that 10% of 10–12 year olds had early varicose vein development and this rose to 75% by the time they were 30 years old (Nicolaidis, 2005). Nicolaidis points out that a range of studies in various countries and age groups suggest symptoms of chronic venous disease may be present in 25–84% of the adult

population. These symptoms include night cramps, 'restless legs', leg heaviness and pain (Nicolaidis, 2005).

Risk factors for venous hypertension

Evidence for a link between social class or income to venous disease is weak but Lee et al (1999) suggested in a review of studies from many countries that venous disease prevalence may be lower in those educated to degree level than in those educated to secondary school level; this could be taken as a crude indication of likely occupation. Occupations involving standing for long periods especially if combined with heavy lifting may be significant, as may being overweight, especially for women.

There appears to be a link between pregnancy and varicose veins but it is unclear what the direct effects are especially as varicose veins often appear in early pregnancy before there is significant extra weight. Burnand (1999) adds that the most significant effect appears with the first pregnancy, although it is not clear why. Any studies on the relationship between the oral contraceptive pill, or menopause and venous hypertension remain inconclusive. There may be a link between venous hypertension and smoking and this seems to affect men more than women (Lee et al, 1999). A significant risk factor for venous disease and varicose vein formation may be a diet deficient in fibre especially in the presence of constipation. This may cause increased abdominal pressure with consequent effects on deep and superficial veins of

the legs through straining (Lee et al, 1999).

Theories of leg ulcer formation

The link between chronic venous hypertension and ulceration is not fully understood (Coleridge Smith, 2006) and this makes for a real clinical challenge. There are theories about the effect of venous hypertension on capillaries, tissue and skin and how these effects contribute to the development of a leg ulcer. There is some discussion and debate about these theories but the general consensus is that all may contribute to ulcer formation and delayed healing although it is not clear how (Coleridge Smith, 2006).

The fibrin cuff theory suggests that as cells leak out of stretched capillaries fibrinogen (a substance involved in blood clotting) collects on the outside of the capillaries as a cuff and over time forms a fibrotic structure with other cells such as collagen. These cuffs form a barrier, which may prevent nutrients from reaching tissues and skin (Agren and Gottrup, 2007). Another theory involves white cell trapping. When venous pressure is high, white cells such as leucocytes and neutrophils increase in number and accumulate in the capillary becoming 'trapped' and causing a prolonged inflammatory response. This inflammatory response includes the release of oxygen free radicals (destructive cells) and proteinases (cells that break down tissue proteins), which lead to tissue destruction and the formation of skin changes such as lipodermatosclerosis (Coleridge Smith, 2006). Another

hypothesis is that growth factors may become trapped in the dermis, which renders them unable to repair damaged tissue (Agren and Gottrup, 2007). Clearly there is great complexity in the role of, and interaction between, cells.

Recurrence rate of venous ulcers and control of venous hypertension

The recurrence rate of venous ulcers is significant because chronic venous hypertension remains as a disease process. It has to be managed for the patient's lifetime unless something can be done to correct the problems with the valves. Obermayer et al (2006) looked at outcomes in 173 patients with 239 ulcerated legs over seven years. The patients had compression therapy and surgery to superficial veins and perforators. The major outcome was that the recurrence rate was 1.7% at six months and 4.6% at five years. This compares very favourably with reported recurrence rates of 28% with compression therapy alone (Moffatt and Dorman, 1995). Interestingly the Obermayer et al study included people with mixed aetiology (a degree of arterial disease present alongside the venous disease) and this group showed the same rate of healing and recurrence as the others, indicating that correcting the venous hypertension is key to managing ulceration.

Management of chronic venous hypertension

The main treatment for chronic venous hypertension with and without ulceration is compression therapy, which assists in

correcting valve closure where possible, reducing reflux of venous blood. Compression therapy speeds up the blood flow, which reduces the pressure in the capillaries allowing them to drain fluid from the tissues to reduce oedema. The improved circulation has an effect on the skin due to the improved delivery of nutrients and may soften lipodermatosclerotic skin (Oduncu et al, 2004) and reduce dryness. Haemosiderin staining will remain and the skin will still be at risk of sensitivity but the overall condition should improve. Exercise will help to reduce backflow by maximising the effect of the calf muscle. Leg elevation will help venous return by utilising the effects of gravity for drainage. Vein surgery to control reflux in superficial and perforating veins aims to reduce venous hypertension (Obermayer et al, 2006). Jull (2007) reviewed systemic pharmaceutical agents for the management of venous disease concluding that much

Key Points

- ▶ Chronic venous hypertension accounts for about 70% of ulcers in the lower limb.
- ▶ It occurs as a result of damage to the valves of the leg which allows backflow of venous blood and an increase to the volume of blood in the lower leg.
- ▶ The effects of chronic venous hypertension include oedema, skin changes, pain discomfort and potential ulceration.
- ▶ Treatment centres on compression, exercise, skin care and elevation.

more evidence is required for all agents. In addition, Jull stated that aspirin or cinnarizine (a histamine antagonist) may play a role in conjunction with compression therapy in ulcer healing, although this is by no means conclusive. Pentoxifylline may play a role in reducing the effects of leucocyte adhesion and zinc sulphate may positively affect ulcer healing but only where a zinc deficiency exists, which is difficult to establish.

Conclusion

It is clear that there is still much to be understood about venous hypertension and management will centre on compression, exercise, skin care and elevation for some time to come. The role of surgery is important but is perhaps not as available to patients in the UK as it could be — whether this is due to a lack of resources or complexity of patient conditions is unclear. If practitioners understand risk factors, the underlying disease process and the effects of intervention, they are much more likely to communicate effectively with patients and secure their engagement in management of the disease. **WE**

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