Chronic oedema, compression therapy and static stiffness index

KEY WORDS
- Chronic oedema
- Venous insufficiency
- Compression therapy
- Static stiffness index

When oedema has been present for at least 3 months, it is considered to be chronic. Oedema formation is the result of a complex interaction between filtration and reabsorption into the capillaries and lymphatic vessels. An holistic assessment is the basis for identification of the underlying pathology so that effective treatment can be implemented in concordance with the patient. Bandages that have a static stiffness index ≥10 provide effective therapeutic compression. Once oedema has been reduced, a follow-up programme must be initiated to monitor and control any oedema recurrence.

HILDEGARD CHARLES
Independent Tissue Viability Consultant, London

Chronic oedema is a term used for swelling that has been present for at least 3 months in a limb or limbs and/or mid-line structures, such as the trunk, head, neck, or genitalia, and is often associated with leg ulceration (Moffatt et al, 2003).

Moffatt et al (2003) concluded that chronic oedema is as common as leg ulceration, with a prevalence of 1.33 per 100 000 people, rising to one per 200 people over the age of 65 years. Chronic oedema of the lower limb is not always dealt with promptly or effectively, which can be due to cost, lack of knowledge and inexperience of clinicians, or delay of the patient's contact (Moffatt et al, 2005; Morgan et al, 2005).

Chronic oedema affects leg ulcer healing and adds to the distress of patients in terms of increased exudate, pain, immobility, difficulty with putting clothing on (especially footwear), and the susceptibility to infection (Williams, 2003).

AETIOLOGY OF CHRONIC OEDEMA

There are different aetiologies of chronic oedema. Therefore, it is essential that a correct diagnosis is established before treatment is initiated. This article will examine lymphovenous oedema, dependency oedema and lymphoedema, and the importance of static stiffness index.

The arteries, veins, and lymphatic vessels make up the circulatory system of the body. Fluid and proteins leak out from the blood capillaries known as the filtration rate into the interstitial spaces (spaces between tissue cells) where fluid is always present. The amount of fluid in the interstitial spaces depends on the amount of fluid and other products introduced into the interstitial space, and the rate at which these fluids are reabsorbed back into the capillaries and lymphatic vessels (Mortimer, 1998; Simonian et al, 2008; Rucigay and Zunter, 2011).

Additionally, people who are malnourished, have renal disease, or malignant ascites can have an abnormal loss of plasma proteins (hypoproteinaemia). This can lead to an increased interstitial fluid volume due to the decreased plasma colloid osmotic pressure (Williams, 2003).

In the normal physiological state, entrance and exit of the fluids from the interstitial spaces are approximately equal, so tissues retain their usual morphological appearance and function (Stanton, 2000). The lymph system plays an essential role in maintaining fluid balance. It carries fluids, fats and proteins back into the general circulation from the tissues and so they do not accumulate in the interstitial spaces (Green, 2007). Any increase of filtration into the interstitial spaces will also result in excess fluid accumulation if not reabsorbed. This increase can be due to cardiac failure, increased venous pressure, ulceration, or inflammation (Williams, 2003).

The lymphatic system also provides an important immunological function by carrying immune cells, such as lymphocytes and macrophages, to the lymph nodes (Mortimer, 2000; Mortimer and Levick, 2004). Lymph transport, not venous capillary reabsorption, is the main process responsible for interstitial fluid drainage (Mortimer and Levick, 2004; *Figure 1*).

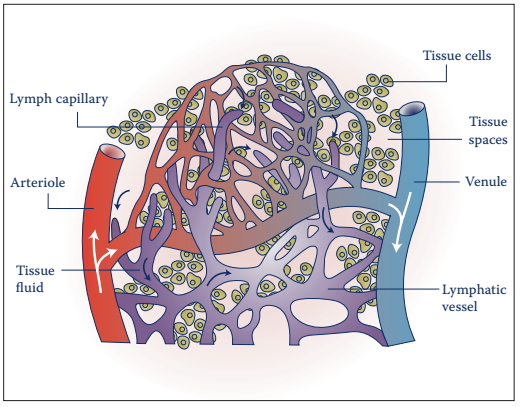

Figure 1. Arterial, venous, and lymph vessels.

LYMPHOVENOUS OEDEMA

Lymphovenous oedema is a combination of venous and lymphatic problems. It occurs when chronic venous disease influences the capillary permeability and increases capillary filtration rates (Nicolaides and Zukowski, 1986; Green and Mason, 2006; Green, 2007). The return of blood from the legs to the heart when the body is in an upright position takes place against the force of gravity. The movement of the ankle and knee activate the calf muscle pump that propels the blood in the deep veins up towards the heart. The venous valves open up as soon as blood is pushed upwards and close when the muscles relax (Schneider and Fischer, 1969; Partsch, 2006; *Figure 2*).

Chronic venous insufficiency occurs when these valves become damaged, allowing the blood to flow backwards. Valve damage may occur as the result of aging, reduced mobility, extended periods of sitting or standing, or a combination of these. The most common cause is valve damage following thrombosis (post-thrombotic syndrome). The backflow of blood increases venous pressure, resulting in venous hypertension. Exposing the connecting and superficial veins to this constant high pressure will increase the filtration rate into the interstitial spaces and lead to an overload of the lymphatic system. As lymphatic drainage becomes further compromised, lymphostatic fibrosis begins to develop, leading to chronic oedema (Green and Mason, 2006; *Figure 3*).

Presentation

The following limb changes may present themselves in lymphovenous oedema: oedema, venous dilatation at the ankle (ankle flare), varicose veins, skin changes – such as hyperpigmentation due to haemosiderin staining – venous eczema, and lipodermatosclerotic changes (Keeley, 2009).

The Clinical signs, Etiology Anatomic distribution and Pathophysiological dysfunction (CEAP) classification for venous insufficiency was developed by Beebe et al (1996) and updated by Eklöf et al (2004). Berridge et al (2010) published new referral recommendations that can be used as a guide for assessment and treatment (*Table 1*).

Management

Guidelines for the treatment of leg ulcers are available locally and nationally (Clinical Resource Efficiency Support Team, 1998; Royal College of Nursing, 2006; Bianchi, 2009; SIGN, 2010). An holistic assessment of people with leg ulceration should include Doppler assessment. Once diagnosis has been established and there are no arterial problems, then compression therapy can be started for venous/lymphovenous leg ulcers (Wingfield, 2009).

Effective compression should provide a balance between exerting too little and too much pressure. Too little pressure is ineffective in reducing oedema and too much pressure is not tolerated by the patient and can cause damage to the arterial system and/or bony prominent parts of the limb. Inelastic (short-stretch) bandages should be applied according to manufacturers' instructions. At full stretch, they should exert a therapeutic resting pressure when the muscle is inactive/relaxed. A working pressure is achieved when the patient is standing, exercising, or walking because the short-stretch bandage does not expand.

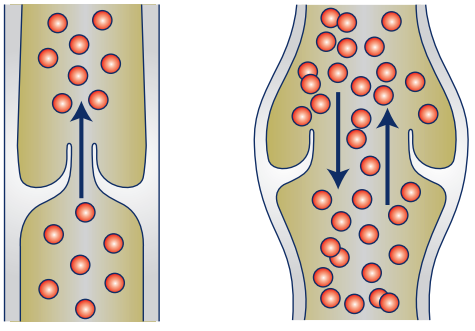

Figure 2. Venous valves.

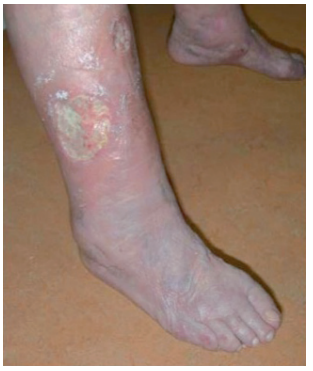

Figure 3. Lymphovenous oedema. Courtesy: Prof Partsch.

Table 1. CEAP classification and recommendations for referral and treatment of venous insufficiency (Berridge et al, 2010)

Classification (Category)	Description	Recommendations
C 0	No visible or palpable signs of venous disease	No intervention
C 1	Telangiectasias or reticular veins (spider veins)	Lifestyle advice exercise, elevation, and compression hosiery
C 2	Varicose veins (>3mm)	As above
C 3	Oedema	As above, if not improved referred appropriately
C 4	Changes in skin and subcutaneous tissue: pigmentation, eczema, lipodermato-sclerosis, or atrophie blanch	Referred to vascular surgeon for full clinical and duplex ultrasound assessment
C 5	Healed venous ulcer	As above
C 6	Active venous ulcer that has failed to heal within 2 weeks	Urgent referral within 2 weeks
Note: Urgent referrals also apply when there is bleeding, varicosities, or superficial thrombophlebitis.		

The static stiffness index (SSI) is the difference between the resting and working sub-bandage pressures. The dynamic stiffness index (DSI) is the change in the sub-bandage pressure when a person exercises the leg (i.e. dorsi-flexion or walking). The amplitude is the difference between the maximal-minimal pressure during dorsi-flexion (*Figure 4*). The SSI is used to differentiate between a long-stretch and short-stretch (inelastic) bandage. An SSI of ≥10 is defined as inelastic compression bandage. This type of bandage produces higher pressure peaks than elastic bandages and results in more effective oedema reduction (Mosti et al, 2008).

The effects of these intermittent pressure peaks will generate a massage effect that mimics the functions of the venous valves, and thus reduces backflow of blood and venous hypertension. These intermittent pressure peaks stimulate lymph flow (Mortimer and Levick, 2004; Földi et al, 2005; Partsch et al, 2006; Mosti et al, 2008; Charles, 2012).

DEPENDENCY OEDEMA

Dependency oedema is also known as "armchair syndrome" or "gravitational oedema". It is usually associated with long-standing immobility due to neurological problems (e.g. multiple sclerosis, cardiac conditions, chronic arthritis, chronic respiratory conditions, general debility, and obesity; Keeley, 2009). The inactivity of the limb develops pressure in the venous circulation, leading to increased capillary permeability. This causes an increase in the lymphatic load and a reduced lymph transport due to the reduced or inactive muscle pump.

Presentation

Oedema starts at the foot and continues up the legs. It is usually soft and pits easily at first (*Figure 5*), but becomes firmer over time. Distortion of limb shape can develop over time and skin breakdown can occur, as well as ulceration (Keeley, 2009).

Management

An holistic assessment must include arterial investigation, initially conducted by Doppler measurements. Early management is advisable before significant limb distortion occurs. Oedema can be reduced by the patient using the calf and foot pump. Dorsi-plantarflexion and circular movements of the ankle promote venous return. Where possible, short phases of standing in conjunction with small steps are beneficial for oedema reduction. Limb elevation while sitting should also be encouraged. Wheelchair-bound individuals need external compression in order to prevent or reduce swelling and induration as long as immobility exists. Meticulous skin care is essential to prevent skin breakdown and infection.

Inelastic bandage compression should be considered for dependent oedema if the arterial blood flow permits compression (i.e. RABPI > 0.8).

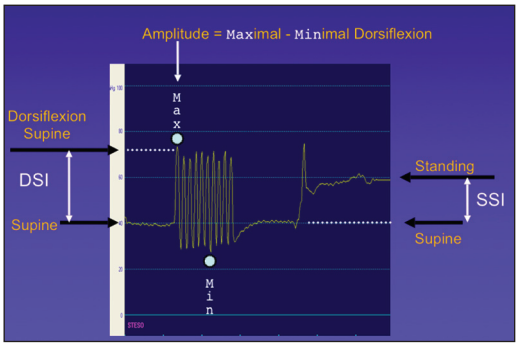

Figure 4. Inelastic sub-bandage pressures (resting and working pressures). Courtesy: H. Charles.

"The early stages of lymphoedema are often ignored by both patient and clinicians since there are no distinctive clinical signs."

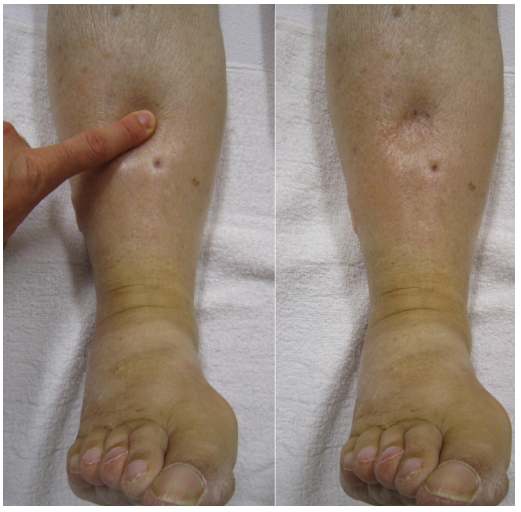

Figure 5. Dependency oedema. Courtesy: Dr J. Heilman.

It has been shown that inelastic bandages (short-stretch) can provide pressure peaks even during small ankle flexions (Franks et al, 2004; Partsch, 2006), thus aiding oedema reduction. When oedema is reduced, properly fitted compression hosiery will help to prevent oedema recurring.

LYMPHOEDEMA

Lymphoedema is the result of an accumulation of fluid and other elements (e.g. proteins) in the interstitial spaces due to an imbalance between the amount of interstitial fluid and its transport. It arises from congenital malformation of the lymphatic system, or damage/loss to lymphatic vessels and/or lymph nodes (International Society of Lymphology, 2003).

Lymphoedema can be divided into primary and secondary. Primary lymphoedema occurs due to an abnormality in the development of the lymphatic system, leading to poor lymph drainage. Secondary lymphoedema arises because of lymphatic damage by some extrinsic process (e.g. radiotherapy, infection, such as cellulitis, surgery, trauma and lymphadenopathy due to cancer; Keeley, 2009).

Note that lymphoedema swelling consists of an increase in fibro-fatty tissue and not just fluid.

Presentation

The early stages of lymphoedema are often ignored by both patient and clinicians since there are no distinctive clinical signs. If untreated, this can progress to lymphovenous oedema (as described above). This progression may first appear as pitting and will be relieved with leg elevation during the day or overnight (Williams, 2003).

As the fluid and waste products accumulate in the tissues, they cause thickening or tissue fibrosis. Tissue becomes hard, non-pitting, and the swelling does not reduce on elevation (*Figure 6*). A positive Stemmer's sign is used to confirm lymphoedema – this is the inability to pick up a fold of skin at the base of the second toe/digit (Stemmer, 1969; 1976).

The International Society for Lymphology has developed a scale for the assessment and categorisation of lymphoedema (*Table 2;* International Society for Lymphology, 2003).

Management

Patients with lymphoedema require a modified approach to the usual venous leg ulcer bandaging regimens (Moffatt et al, 2005).

The British Lymphology Society recommends a minimum standard of care for patients with lymphoedema that comprises four components and should be used in combination to provide the most effective care (British Lymphology Society, 2002). The Society's recommendations are:

- Skin care includes the maintaining of skin integrity by moisturising the skin to prevent infection.
- Exercise to maintain joint mobility and enhance lymphatic and venous flow.
- Compression therapy with multilayer in-elastic bandages and follow-up with compression hosiery.
- Manual lymphatic drainage by a specially trained professional.

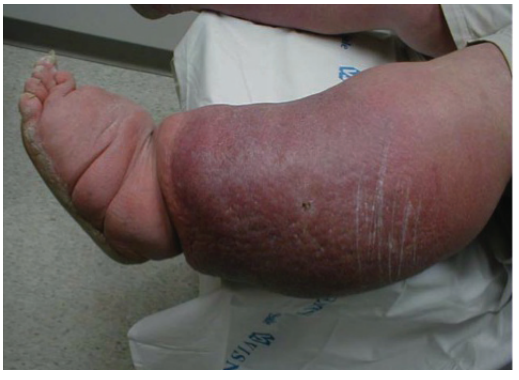

Figure 6. Lymphoedema. Courtesy: H. Charles.

Table 2. Staging of lymphoedema (International Society of Lymphology, 2003)

Stage	Description
Stage 0	A subclinical state where swelling is not evident despite impaired lymph transport. This stage may exist for months or years before oedema becomes evident.
Stage I	This represents early onset of the condition where there is accumulation of tissue fluid that subsides with limb elevation. The oedema may be pitting at this stage.
Stage II	Limb elevation alone rarely reduces swelling and pitting is manifest at late stage II.
Stage II late	There may or may not be pitting as tissue fibrosis is more evident.
Stage III	The tissue is hard (fibrotic) and pitting is absent. Skin changes such as thickening, hyperpigmentation, increased skin folds, fat deposits and warty overgrowths develop.

Patients with mild lower-limb oedema (International Society for Lymphology, Stage I), minor pitting, no significant tissue changes, and no or minimal shape distortion are suitable for initial compression hosiery.

A follow-up programme in partnership with the patient has to be developed during the treatment period (British Lymphology Society, 2002).

CONCLUSION

The management of chronic oedema must be holistic, and in collaboration with the patient and other clinicians as necessary. Management should include:

- Identification and treatment of the underlying medical conditions.
- Reduction of oedema through leg elevation, exercise, and effective compression bandaging.
- Skin care to prevent breaks and possible infection and cellulitis.
- Follow-up to monitor and support the patient. WUK

REFERENCES

Beebe HG, Bergan JJ, Bergqvist D et al (1996) Classification and grading of chronic venous disease in the lower limbs: a consensus statement. *Eur J Vasc Endovasc Surg* 12(4): 487–91

Berridge D, Bradbury AW, Davies AH et al (2010) *Recommendations for the referral and treatment of patients with lower limb chronic venous insufficiency (including varicose veins).* Venous Forum of the Royal Society of Medicine, London. Available at: www.rsm.ac.uk/academ/forvenou.php (accessed 31.05.2013)

Bianchi J (2009) Arterial assessment in patients with chronic oedema of the lower limb. In: *Skills for Practice: Management of Chronic Oedema in the Community.* Wounds UK, Aberdeen

British Lymphology Society (2002) *Strategy for Lymphoedema Management.* BLS, Sevenoaks

Charles H (2012) The function and composition of next generation bandages. *Wounds UK* 8(1 Suppl 1): S16–9

Clinical Resource Efficiency Support Team (1998) *Guidelines for the Assessment and Management of Leg Ulceration.* CREST, Belfast

Eklöf B, Rutherford RB, Bergan JJ et al (2004) Revision of the CEAP classification for chronic venous disorders: Consensus statement. *J Vasc Surg* 40(6): 1248–52

Földi E, Junger M, Partsch H (2005) The science of lymphoedema bandaging. In: *European Wound Management Association Focus Document. Lymphoedema Bandaging in Practice.* MEP, London

Franks PJ, Moody M, Moffatt CJ (2004) Randomized trial of cohesive short-stretch versus four-layer bandaging in the management of venous ulceration. *Wound Repair Regen* 12(2): 157–62

Green T (2007) Leg ulcer management in patients with chronic oedema. *Wounds Essentials* 2: 46–58

Green T, Mason W (2006) Chronic oedemas: identification and referral pathways. *Br J Community Nurs* 11(4): S8–16

International Society of Lymphology (2003) The diagnosis and treatment of peripheral lymphedema. Consensus document of the International Society of Lymphology. *Lymphology* 36(2): 84–91

Keeley V (2009) Chronic oedema of the lower limb: causes, presentation and management priorities. In: *Skills for Practice: Management of Chronic Oedema in the Community.* Wounds UK, Aberdeen

Moffatt CJ, Franks PJ, Doherty DC et al (2003) Lymphoedema: an underestimated health problem. *QJM* 96(10): 731–8

Moffatt CJ, Morgan P, Doherty D (2005) The Lymphoedema Framework: a consensus on lymphoedema bandaging. In: *European Wound Management Association Focus Document. Lymphoedema Bandaging in Practice.* MEP, London

Morgan PA, Moody M, Franks PJ et al (2005) Assessing community nurses' level of knowledge of lymphoedema. *Br J Nurs* 14(1): 8–13

Mortimer PS (1998) The pathophysiology of lymphoedema. *Cancer* 83(12 Suppl American): 2798-802

Mortimer P (2000) Acute inflammatory episodes. In: Twycross R, Jenns K and Todd J, eds, *Lymphoedema.* Radcliffe Medical Press, Oxford. pp. 130–9

Mortimer PS, Levick JR (2004) Chronic peripheral oedema: the critical role of the lymphatic system. *Clin Med* 4(5): 448–53

Mosti G, Mattaliano V, Partsch H (2008) Inelastic compression increases venous ejection fraction more than elastic bandages in patients with superficial venous reflux. *Phlebology* 23(6): 287–94

Nicolaides AN, Zukowski AJ (1986) The value of dynamic venous pressure measurements. *World J Surg* 10(6): 919–24

Partsch H (2006) Compression therapy of venous ulcers. *EWMA Journal* 6(2): 16–20

Partsch H, Clark M, Bassez S et al (2006) Measurement of lower leg compression in vivo: recommendations for the performance of measurements of interface pressure and stiffness: consensus statement. *Dermatol Surg* 32 (2): 224–33

Royal College of Nursing (2006) *Clinical Practice Guidelines: The Nursing Management of Patients with Venous Leg Ulcers.* RCN, London. Available at: http://bit.ly/Umsm84 (accessed 28.05.2013)

Rucigay TP, Zunter VT (2011) Lymphedema: clinical picture, diagnosis and management. In Singh N, *Radioisotopes. Applications in Bio-Medical Science.* InTech, Rijeka, Croatia

Schneider W, Fischer H (1969) *Die Chronische Venöse Insuffizienz.* Enke, Stuttgart

SIGN (2010) *Management of Chronic Venous Leg Ulcers.* SIGN, Edinburgh

Simonian SJ, Morgan CL, Tretbar LL, Blondeau B (2008) Differential diagnosis of lymphedema. In: Tretbar LL, Morgan CL, Lee BB et al, eds. *Lymphedema.* Springer, London. pp. 12–31

Stanton A (2000) How does tissue swelling occur? The physiology and pathophysiology of interstitial fluid formation. In: Tywcross R, Jenns K, Todd J, eds. *Lypmphoedema.* Radcliffe Medical Press, Oxford

Stemmer R (1969) Ambulatory elastocompressive treatment of the lower extremities particularly with elastic stockings. *Der Kassenarzt* 9: 1–8

Stemmer R (1976) A clinical symptom for the early and differential diagnosis of lymphedema. *VASA* 5(3): 261–2 [article in German]

Williams A (2003) An overview of non-cancer related chronic oedema – a UK perspective. Available at: http://bit.ly/11G8z60 (accessed 31.05.2013)

Wingfield C (2009) Chronic oedema and the importance of skin care. In: *Skills for Practice: Management of Chronic Oedema in the Community.* Wounds UK, Aberdeen