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Compression for chronic venous disorders of the lower limbs

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Contents

1 Definition and mechanics of chronic venous disorder 2
1.1 Varicose veins 3
1.2 Progression of oedema 4
1.2.1 Classifications and grading 5
1.2.2 Site of accumulation of fluid in lower leg 6
2 Prevalence data 7
2.1 Prevalence of venous disorder 7
2.2 Prevalence of oedema 7
3 Management of chronic venous disease 8
3.1 Oedema management 9
3.2 Ulcer healing 10
3.3 Concomitant ulcers and oedema healing 11
Conclusion 12
References 12

Abstract

Chronic venous disorder refers to circumstances in which venous valves fail to function properly and blood flows backwards in the veins to accumulate in the limb. In turn the pressure in the veins is increased. A range of indicators arise (for example, oedema, ulcer) as a result of venous return failure. This chapter provides an overview of chronic venous disease and its classifications. Prevalence of venous disorder and oedema is reported. Additionally, the use of textile-based compression therapy for the treatment of chronic venous disease, including ulcer healing and oedema management, is highlighted.

Keywords: Chronic venous disease, Oedema, Compression therapy

1 Definition and mechanics of chronic venous disorder

Chronic venous disorder refers to the circumstances in which venous valves fail to function properly, which affects both the deep and superficial venous system. Under normal conditions, the veins and a series of valves propel and direct the blood flow in the limbs from the surface to deep veins and then back to the heart against the forces of gravity. If valves in these veins fail to work efficiently, the blood can flow back through the veins and accumulate in the limb. In turn the pressure in the veins is increased. A range of indicators i.e. varicose veins, skin ulceration, oedema, venous eczema, hyper-pigmentation of skin of the ankle, scar tissue formation due to either swelling or cell damage arise as a result of venous return failure (Kistner, Eklof, and Masuda 1996, Ballard and Bergan 2000, Bergan, Schmid-Schonbein, Smith, Nicolaides et al. 2006, Meissner, Moneta, Burnand, Gloviczki et al. 2007, Kankariya 2021).

Chronic venous disease represents a wide spectrum of disease conditions ranging from reticular veins to venous ulcerations. Treatment of venous disease has suffered from a deficiency of precision in diagnosis, which in turn has led to conflicting recommendations about how to control specific venous disease. Better classification of disease entities has helped to resolve these conflicts (Callam 1994, Kistner, Eklof, and Masuda 1996). Widmer (1978) proposed a classification for clinical appearance of the limb; Sytchev (1985) presented a more comprehensive approach based on hemodynamic and phlebographic data in combination with clinical stages; Enrici and Caldevilla (1992) in Argentina proposed a classification of initial clinical manifestations in patients. None of these classifications has received universal acceptance or widespread usage (Kistner, Eklof, and Masuda 1996, Kankariya 2021).

In 1993, Porter suggested using a similar approach to that of the TNM classification (Tumor/ Node/ Metastasis) for cancer to develop a classification system for venous diseases. After a year of intense discussion in February 1994, an international ad hoc committee of the American Venous Forum developed the first CEAP with the aim being development of a universal accepted venous disease classification. The four components of CEAP classification included the clinical sign of CVI, the Etiology factors, the anatomic distribution of reflux and obstruction, and the underlying pathophysiology findings (Kistner, Eklof, and Masuda 1996, Eklöf, Rutherford, Bergan, Carpentier et al. 2004, Meissner 2005, Robertson, Evans, and Fowkes 2008, Eberhardt and Raffetto 2014, Kankariya 2021).

The clinical sign of venous disease is categorised into seven parts, i.e. class 0 - asymptomatic limbs, class 1 - telangiectasia / reticular, class 2 - varicose veins, class 3 - oedema, class 4 - skin changes without ulceration, class 5 - healed ulcers, class 6 - active ulcers. The etiology being classified as congenital for recognised birth defects, primary for undetermined cause, and secondary for known cause (usually an incident of acute DVT). The underlying anatomic cause of venous dysfunction was divided into three simplified forms i.e. superficial, deep and perforating. The pathophysiologic was categorised as reflux or obstruction or both. The beneficial value of exerting pressure to manage the venous disease varies based on the classification (Kistner, Eklof, and Masuda 1996, Eberhardt and Raffetto 2014).

1.1 Varicose veins

The most common clinical manifestations of CVI are subcutaneous dilated, tortuosity veins i.e. reticular veins, varicose veins and telangiectasias (Naoum, Hunter, Woodside, and Chen 2007). Varicose veins are palpable tortuous veins that don’t discolour the overlying skin and have a diameter of greater than 4 mm, which distinguishes them from reticular veins which are nonpalpable, blue dermal veins with diameter of less than 4 mm (Meissner 2005, Eklöf et al. 2004). Common chronic symptoms of varicose veins include skin rashes, leg aching, pruritus and cosmetic embarrassment, which most commonly occur on the leg. The development of varicose veins is the result of incompetence of proximal valve, a flap valve situated in between superficial and deep vein, which leads to reflux of blood from the deep to the superficial system which in turn increase the dilation and tortuosity (Naoum et al. 2007, Golledge and Quigley 2003).

1.2 Progression of oedema

Total body fluid in humans is divided between the extracellular (25%) and intracellular (75%) spaces. The extracellular spaces may further be separated into (a) interstitial compartments, which comprise two third of extracellular fluid volume, and (b) vascular spaces, which contain one third of extracellular fluid volume (Cho and Atwood 2002). The ratio of fluid volume in vascular and interstitial spaces is regulated by two dynamic and opposing forces i.e. hydrostatic and osmotic forces operating across the capillary wall. Capillaries, the very thin blood vessels, perform the role of oxygen, carbon dioxide, nutrients and metabolic waste exchange between the blood and the interstitial fluid, denoted as capillary filtration. The direction and amount of fluid flow across the capillary walls reflects the balance of hydrostatic and osmotic pressures, i.e. net filtration pressure. Hydrostatic pressure pushes the fluid across the capillary wall. At any point on the capillary, the capillary hydrostatic pressure HP_c works against the interstitial hydrostatic pressure HP_{if} acting outside the capillaries and aiding return of fluid to the capillaries. For this reason, the net hydrostatic pressure acting on the capillaries at any point is the difference between HP_c and HP_{if}. Normally the value of HP_{if} is negative to slightly positive so it is assumed to be zero. In contrast to hydrostatic pressure, osmotic pressure pulls the fluid across the capillary membrane which is created by the presence of abundant nondiffusible solutes, i.e. plasma proteins, in a fluid. Because of abundant plasma protein in capillary blood compare to lower protein content in interstitial spaces, the magnitude of OP_c remain higher than OP_{if} (Marieb and Hoehn 2010). At the arterial end of the capillary the dominating hydrostatic pressure over osmotic pressure forces fluid into the interstitial spaces. In contrast, the dominating osmotic pressure over hydrostatic pressure at the venous end of the capillary facilitates reabsorbing the fluid back into the capillary. An estimated nine-tenths of what is pushed out into the interstitial spaces at the arterial end returns at the venous end, with the remaining small amount of excess fluid and proteins returning through the lymphatic system as lymph flow activity (Marieb and Hoehn 2007, Kankariya 2021, Villeco 2012).

Imbalance of the hydrostatic and osmotic pressures which may be the result of venous disorder acting across the capillary wall, increases filtration and allows excess fluid to accumulate in the interstitial spaces. This in turn leads to formation of swelling, i.e. oedema. In the initial stage of the swelling, the lymphatic system compensates for the increased volume of fluid in the tissue spaces by increasing lymph flow activity. Adequate treatment may reverse the failure of the filtration function and re-set the pressure values. However, if the lymphatic system is pushed beyond its maximum working capacity it may be overwhelmed by the additional fluid

and be unable to drain the excess fluid accumulated in the interstitial spaces. The accumulated fluid leads to reduced joint mobility and an observable disparity in appearance. Measurable swelling at this stage is manifested by pitting oedema. Over time and/or in the case of inadequate treatment, the over extension of the system may lead to permanent damage of lymphatic valves. Consequently, fluid accumulation increases along with initiation and progression of protein deposition in the tissue, which is irreversible. Skin in this stage of oedema is smooth. Without treatment, the swelling progresses further where there is large volume of protein content present in the tissue, and oxygenation and nutrition of the tissue may reduce resulting in the skin hardening (Priollet 2006, Simon 2014, Hespe, Matthew, and Babak 2015, Kankariya 2022, Kankariya 2021). Further complexity is added due to oedema increasing the risk of greater severity of this chronic condition. Oedema, by preventing fresh oxygenated blood flow into the limb can also contribute to worsening of ulcerations (Cho and Atwood 2002, Spentzouris and Labropoulos 2009, Dogra and Sarangal 2014, Kankariya 2022). Hence management of oedema in its early stages is necessary if mobility and comfort of patients is to be improved. It is worth noting that a person without any disorders and with an equitable circulatory system may have some lower extremity gravity-dependent oedema (Hettrick 2009).

1.2.1 Classifications and grading

Peripheral oedema limited to the lower leg can be one of two types i.e. venous oedema and lymphedema. Venous oedema occurs as fluid from the capillaries accumulates in the interstitial spaces due to an imbalance of net filtration pressure, whereas lymphedema arises as a result of protein rich fluid deposition in the interstitial space due to impairment of the lymphatic system (Simon 2014, Kankariya 2021).

Oedema can also be clinically classified as pitting or non-pitting oedema. Pitting oedema refers to oedema which is severe enough to leave a pit or an indentation when an external force is applied to the swollen area i.e. when depressing the effected skin with a finger, a pit forms which persists for some time after release of the force. The application of external pressure forms a visible indentation on skin, pit, due to displacing of interstitial fluid with the increased drainage through the lymph vessel (Whiting and McCready 2016). The indentation disappears with the removal of pressure. If no pit or indentation forms when an external force is applied, the oedema is classified as non-pitting oedema. Pitting oedema formation generally precedes non-pitting oedema and is more common. Non-pitting oedema arises with the initiating of accumulation of protein concentration in the fluid of the interstitial space, caused by damage

of the lymphatic system. That an external force does not form an indentation is due to the impaired lymphatic system inability to drain the fluid. If unhealed, the pitting oedema may evolve into fibrotic and brawny oedema. Brawny oedema is stiff to touch, with no “pits”, and needs to be treated before it can be mobilised (Villeco 2012, Trayes, Studdiford, Pickle, and Tully 2013, Kankariya 2021, Whiting and McCready 2016).

Pitting oedema is often graded for severity with grades ranging from slight to very severe on a four point scale (Sussman and Bates-Jensen 2007).

4 grade when, pitting size >25.4 mm (1 inch), very severe
3 grade when, 12.7 mm (½ inch) < pitting size >25.4 mm (1 inch), severe
2 grade when, 6.35 mm, (¼ inch) < pitting size > 12.7 mm (½ inch), moderate
1 grade when pitting size < 6.35 mm, (¼ inch), mild

1.2.2 Site of accumulation of fluid in lower leg

In the lower extremities groups of muscles, blood vessels and nerves can be denoted as compartments. Compartments are covered by a thick and tough fascia membrane and are the potential site for oedema development. Inside a closed compartment, tissue pressure exceeding venous pressure impairs the arterial inflow and venous outflow. This leads to accumulation of fluid into the tissue spaces of the compartment and swelling occurs. To determine the site of accumulation of the fluid an understanding of the location, and anatomy of each compartment is needed (Mauser, Gissel, Henderson, Hao et al. 2013).

The foot

The foot is believed to have nine compartments. These nine compartments are divided into four groups i.e. the interosseous compartment which encloses four muscles, central, medial and lateral compartment (Mazzillo, Ansari, and Reichman 2013).

The ankle to calf

This region of lower leg is comprised of four compartments i.e. superficial posterior, deep posterior, anterior and lateral. The posterior and anterior inter-muscular septum separates the lateral muscles from the posterior and anterior muscles respectively (Von Keudell, Weaver, Appleton, Bae et al. 2015).

The thigh

The thigh region consists of three compartments i.e. posterior, anterior and medial. The medial and lateral inter-muscular septum separates the medial and posterior compartments from the

anterior compartment (Von Keudell et al. 2015). The posterior compartment is separated from the medial compartment by a thin fascial plane.

As the foot, ankle, calf and thigh have a number of 'compartments', swelling may occur in any and may be asymmetric or symmetric (Mauser et al. 2013). Correct treatment requires consideration of various factors i.e. location of compartment, symmetric or asymmetric swelling, chronic or acute conditions, and direction of movement of fluid (Kankariya 2021, Gorman, Davis, and Donnelly 2001).

2 Prevalence data

2.1 Prevalence of venous disorder

Venous disorder of the lower extremity is a debilitating chronic condition, having an estimated prevalence of 25% of the adult population in western countries (Lohr and Bush 2013). Studies conducted on diverse populations show that prevalence of chronic venous disorder varies widely, influenced by factors such as population and/or sex of those examined i.e. from 1% to 40% of women and 1% to 17% of men (Ruckley, Evans, Allan, Lee et al. 2002, Criqui, Jamosmos, Fronek, Denenberg et al. 2003, Beebe-Dimmer, Pfeifer, Engle, and Schottenfeld 2005, Lohr and Bush 2013). Venous disorder not only affects the patient's quality of life but also influences the economy. In the United States, over $1 billion was spent and 4.6 million workdays were lost annually in the treatment of chronic venous disease (Spentzouris and Labropoulos 2009). In the United Kingdom, the cost of venous disease of the leg was estimated to be £300 - £600 million per year (O'Meara, Tierney, Cullum, Bland et al. 2009). Note that results of these investigations were published in the early 2000s (2002 – 2013), and no further data has been investigated.

2.2 Prevalence of oedema

In the Bonn Vein Study, conducted during November 2000 to March 2002 and featuring 3072 randomly selected people aged from 18 to 79 years, prevalence of oedema was found to be 135 per 1000 (149 per 1000 in women and 116 per 1000 in men) (Rabe and Pannier 2006). In the Bonn vein study, age distribution prevalence of the Bonn Vein population for oedema is provided, however, age distribution prevalence is not available for men and women. The age distribution prevalence of oedema increased with age and was more than 200 per 1000 for the 60+ year age group. Prevalence of chronic oedema was also higher in women 5.4 per 1000, compared to men 2.5 per 1000, in the Derby city study (United Kingdom), conducted in 2012

and featuring 971 patients aged from 5 years to 85+ years (Moffatt, Keeley, Franks, Rich et al. 2017). Prevalence of chronic oedema in specific patient age groups were again found to be higher in women (15-44 years: 1.03 and 0.46 per 1000; 45-64 years: 7.09 and 4.05 per 1000; 65-74 years: 13.16 and 4.75 per 1000; 75-84 years: 21.67 and 7.53 per 1000; 85+ years: 30.54 and 11.73 per 1000) than to men (15-44 years: 0.47 and 0.09 per 1000; 45-64 years: 3.06 and 0.56 per 1000; 65-74 years: 7.37 and 1.99 per 1000; 75-84 years: 12.71 and 3.68 per 1000; 85+ years: 25.26 and 5.75 per 1000) in both the Derby city study, conducted in 2012, and the West London study, conducted in 2001 (Moffatt, Franks, Doherty, Williams et al. 2003, Moffatt et al. 2017). Data shows the rate of oedema increases with increasing age and accelerates more rapidly in the older age population i.e. 64+ years. The evidence suggests that chronic oedema is more than twice more prevalent in females than males. More recent data on prevalence of chronic oedema in male and female has not been identified (Kankariya 2021, Kankariya, Laing, and Wilson 2021).

3 Management of chronic venous disease

Managing the chronic venous disease is defined as a complex active process that restores the function and continuity of venous return. The objectives of healing venous disease are to treat and remove the precipitating causes, to encourage circulation and to improve the venous return. Dressings in conjunction with compression therapy are the clinical means of cleaning, protecting and compressing in the direction appropriate for reducing oedema induced swelling and/ healing the chronic ulcers (Kan and Delis 2001, Parsa, Zangivand, and Hajimaghsoudi 2012). Healing of venous disease relies on a variety of approaches including compression therapy, drug therapy and surgery (Alguire and Mathes 1997, Felty and Rooke 2005, Perrin and Ramelet 2011). This study focused on management of the chronic venous disease limited to the lower leg, using textile-based compression interventions.

Compression therapy is a recognised method of choice for the management of all kinds of swollen extremities i.e. oedema (Partsch 1991). In resting supine positions, textile compression products apply on-going pressure against the patient's skin surface. In resting upright positions, as the venous pressure is increased, greater external pressure is needed than that in the resting supine position if the same effect is to be achieved. When 'working' (e.g. walking), the underlying muscles contract and expand exerting pressure against the compression fabric and pressure in the veins of the limb is increased. This increased pressure stimulates pumping of the lymphatic system and encourages re-absorption of lymphatic fluid, thus oedema is reduced

(Partsch and Partsch 2005, Lymphoedema Framework 2006, Lim and Davies 2014, Kankariya 2021, Kankariya et al. 2021).

3.1 Oedema management

In order to manage oedema using compression therapy, two treatment phases occur i.e. the primary phase of deflation or oedema reduction, followed by the maintenance phase (Sackheim, De Araujo, and Kirsner 2006, Dissemond, Assenheimer, Bültemann, Gerber et al. 2016). Due to the larger dimension of oedematally swollen legs in the initial phase, using stockings may not be appropriate. In the deflation phase, compression bandages need to be re-applied over shorter periods frequently. A disadvantage of bandages is that when the bandage is applied over pronounced oedema, the blood may deplete rapidly, and the bandage slacken. Bandages also do not necessarily follow the contour of the leg, which may lead to slippage, creasing in the bandage, and may cause skin breakdown and discomfort. Therefore, frequent bandage changes may be required until the leg volume reduction has reached steady state (Solne-Rivera and Wu 2012). Applied pressure during the maintenance phase needs to be sufficiently high to prevent any new oedema formation. In this phase stockings are commonly used. An optimum level of pressure needs to be maintained, the level of which is associated with comfort and the quality of the patient's life (Dissemond et al. 2016). Successful compression intervention typically occurs when principles behind the compression therapy are well understood and all factors related to materials used and human body requirements are considered. Management of oedema is necessary in order to improve patients' comfort and mobility, as well as being a key element in healing venous ulcerations (Hettrick 2009, Kankariya et al 2021).

Healed oedema is assessed and quantified by determining either the circumference or the volume of the limb. For example, leg circumference can be measured using a tape measure or a Leg-O-Meter. Leg-O-Meter designed by Dr. F. Zuccarelli, contained the tape measure attached to a stand which in turn fixed to a small board on which the patient is in standing position (Bérard, Kurz, Zuccarelli, Ducros et al. 1998). Unlike the tape measure, Leg-O-Meter also keeps records of height at which the circumference has been measured (Bérard et al. 1998, Bérard, Kurz, Zuccarelli, Abenhaim et al. 2002). A reference line measurement of circumference of both lower legs should be taken at the time of initial assessment and at fixed intervals throughout compression therapy. Leg circumference is measured at the smallest portion of the ankle and at the largest portion of the calf (Guex and Perrin 2000). The

measurement of circumference is not always correlated with leg volume measurement. The leg volume may be measured using a water displacement volumetric technique. Volumetry technique is used to measure the oedema in uneven surfaces i.e. ankle and foot. Volumetry water displacement technique follows a simple physics principle which says if a leg is immersed into a vessel filled completely to a water, the overflowing volume of water represents the volume of the immersed leg. The overflown water can be measured in a calibrated container (Reis, Ribeiro, Carvalho, Belchior et al. 2004, Rabe, Stücker, and Ottillinger 2010).

3.2 Ulcer healing

Leg ulcers are skin disorders which reduce quality of life because of the debilitating nature of the disorder (Spentzouris and Labropoulos 2009, Dogra and Sarangal 2014). Healing of venous ulcers may be referred to as a therapeutic challenge, the immediate objective of which is to reconstruction of tissues by means of improving microcirculation and proper oxygen delivery (Collins and Seraj 2010). Care of the skin surrounding an ulcer is crucial to minimize the risk of further ulceration and also to accelerate the ulcer healing. Compression therapy plays a vital role in healing of the venous ulcers. Compression therapy generates pressure in the tissue under the skin preventing the blood from flowing backward and reducing the swelling by stopping the leaking of the excess fluid into the interstitial space. Thus the skin is able to receive needed oxygen and nutrients essential for ulcer healing (Moneta and Partsch Accessed: 31/3/2017). Non-healing venous ulcers require an accurate diagnosis, assessment and treatment involving correction of the underlying contributing systemic causes (Dabiri, Hammerman, Carson, and Falanga 2015). There are two phases to consider: effective venous ulcer healing with compression therapy i.e. the initial therapy phase; and the subsequent maintenance phase (Partsch 1991, Dissemond, Eder, Läuchli, Partsch et al. 2017). The therapy phase refers to complete healing time for the limb ulceration in which compression bandages are commonly still recommended, whereas the maintenance phase denotes prevention of ulcer recurrence during which stocking systems are frequently used (Dissemond et al. 2017).

Correct pre-assessment plays a vital role in effective leg ulcer healing. The ulcers assessment includes colour and type of exudate, ulcer location, duration and ulcer size in terms of surface area or two maximum perpendicular diameters (Hendricks and Swallow 1985, Brizzio, Blattler, Rossi, Chirinos et al. 2006, Bongiovanni 2014). Patients with chronic venous disease may also have severe arterial disease and failure to recognise this arterial disease will consequence in the hazardous application of high compression therapy. Arterial disorder should be evaluated

using the Doppler to determine the ankle-brachial pressure index (ABPI). Ankle-brachial pressure index refers to the ratio of systolic blood pressure at the ankle to systolic blood pressure at the upper arm. A leg ulcer can be assigned with following assessment in terms of ankle-brachial pressure index, as (Al-Qaisi, Nott, King, and Kaddoura 2009):

- Ankle-brachial pressure index > 1.0, no arterial disease
- 0.8 < Ankle-brachial pressure index ≥ 1.0, either no or mild arterial disease
- 0.5 ≤ Ankle-brachial pressure index ≥ 0.8, moderate disease
- Ankle-brachial pressure index < 0.5, severe disease

3.3 Concomitant ulcers and oedema healing

In order to healing the ulcers concomitant with oedema using compression therapy, three distinction phases must be followed i.e. the primary phase of deflation or called as oedema reduction phase, the secondary phase of ulcer healing, followed by the maintenance phase (prevention of leg oedema and avoidance of ulcer recurrences) (Dissemond, Assenheimer, Bültemann, Gerber et al. 2016). Due to the bigger dimension of oedematally swollen leg at the initial phase, using ulcer stocking would not be a sensible decision. In the deflation phase, the compression bandages must be reapplied over shorter periods in a frequent fashion. When the bandage is applied over pronounced oedema, the blood is depleted rapidly, and the bandage is slackened. Bandages don't follow the contour of the leg, which leads to slippage resulting in ceases in the bandage causes of skin breakdown and discomfort. Therefore frequently changing of bandage is required until the leg volume reduction has reached a steady condition (Nancy and Stephanie 2012). Resulting successful completion of the initial deflation phase, the secondary phase initiates. In the secondary phase, as the leg volume has already been reduced and reached at the steady state which ensures the proper availability of nutrients and oxygen, resulting in frequent transition of venous leg ulcers into a progressive healing process. Applied pressure during this phase is high enough to ensure the prevention of any newly oedema formation. In the subsequent maintenance phase, ulcer stocking devices are used in order to prevent the ulcer recurrences. An optimum level of pressure is maintained which are associated with comfort and quality of patient's life (Dissemond et al. 2016).

Many patients with chronic venous disorder have ulcers also accompanied by swollen legs. Oedema and ulcers have a complex relationship, however, both may have a similar cause i.e. venous disorder. The oedema induced swollen leg in the uncompressed phase usually has a considerable larger leg circumference (Dissemond et al. 2017). In this case the nutrients are

required to travel more average distance from the capillary wall resulting in oedema creates some barriers for the available tissue's nutrients and oxygen which is also associated with the decrease the difference of hydrostatic (pushes the fluid across the boundary) and osmotic pressure (pulls fluid across the boundary). The larger or wider circumference of the limb also refers to rise in the distance between the epithelial ends, which aid in increasing of ulcer size. An open ulcer allows the infection to be increased progress to further oedema (Macdonald and Ryan 2010, Gogia and Gogia 2012).

Conclusion

Chronic venous disorders refer to a set of conditions in which the valves of the veins do not function effectively, causing blood to reverse its course and accumulate in the limb. From reticular veins to oedema to venous ulcerations, chronic venous disorder encompasses a broad spectrum of disease states. An estimated 25% of adults in western nations suffer with venous disease of the lower extremities.

For the treatment of all types of venous diseases, compression therapy is widely accepted as the best option. Compression interventions are most effective when there is a deep understanding of the foundational principles of therapy, comprehensive consideration of all aspects related to materials used, and careful attention to the specific needs of the human body.

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