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Compression Therapy

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Therapeutic limb compression is used to prevent edema formation, to reduce existing edema, and to prevent reaccumulation of edema once it is reduced. Forms of therapeutic compression for limb edema, lymphedema, and venous ulcers include compression bandaging, pneumatic compression, and compression garments (stockings/sleeves). Of these, compression bandaging is usually applied as an initial therapeutic intervention to reduce edema during a "decongestive" phase (obtain a result) and compression garments are used for maintenance (sustain the result) or preventatively as with chronic venous insufficiency. Pneumatic compression may be used in conjunction with bandages or garments. This chapter describes major relevant aspects and offers some practical pearls and guidelines.

1. COMPRESSION BANDAGING

1.1. Bandage Features and Function

Differences in bandage materials and structure give rise to functional differences. A bandage material that contains a high proportion of elastic fibers is referred to as a "long-stretch" bandage. These behave in a manner similar to stretched springs; the more you stretch it, the greater is the recoil force. Long-stretch bandages can be stretched to a length two to three times their zero tension length, as they are applied to a limb. The reactive tension in the bandage causes a sub-bandage pressure (SBP), which is the basis for an inward radial directed "resting pressure." Here the term "resting" is used to distinguish an SBP in a muscularly relaxed limb from one in which muscular contraction is occurring resulting in a dynamic pressure (Fig. 1). A "short-stretch" bandage has few, if any, elastic fibers; so, it exerts much less recoil tension on the limb. During bandaging, a small amount of stretch is made possible by the weave of the bandage fabric and this feature is used to allow the bandage to be molded to the shape of the limb. As the recoil force is low, so is the resting pressure. At the limit of the short-stretch type is the "zero-stretch" or inextensible bandage. Typically, these consist of open-weave cloth or gauze impregnated with a zinc oxide gel that may be applied directly over wounds and skin irritations. These bandages are applied without tension and are molded to the leg while wet to form a cast-like

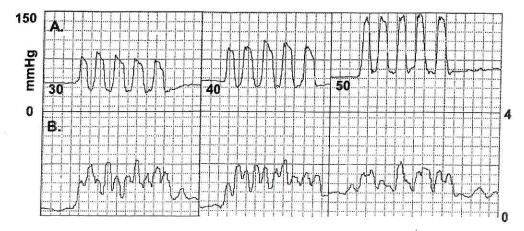


Figure 1 Effect of increased compression on sub-bandage resting and dynamic pressures. A subject was exposed to increasing levels of calf compression (30–40–50 mmHg) as they stood flat-footed. Sub-bandage pressure (A) was monitored with static and dynamic sensors and blood perfusion (B) was monitored with laser-Doppler at the posterior calf during rest and during a sequence of five calf contractions by consecutive heel-up maneuvers. As pressure increases, resting counter-pressure increases and, as radial expansion is restricted, dynamic pressures also increase. Time lines are 1 sec.

bandage. In the case of short-stretch and zero-stretch bandage, additional layers of bandaging can be used to increase pressures (Fig. 2). Padding may be used to protect bony prominences and to make pressures more uniform over irregular-shaped limb contours. Another bandage is a cohesive type that does not stick to the skin, but its individual layers adhere to each other. This material contains some elastic fibers, but the amount of extension has not been determined when the layers cohere. Clinical experience suggests they act like short-stretch bandages as applied to the limb.

Another feature that distinguishes bandages is their "stretchability" when exposed to radial-directed outward forces caused by muscular contraction. A

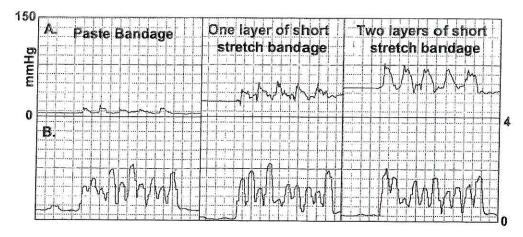


Figure 2 Effect of increasing the number of bandage layers. Same measuring conditions as with Fig. 1. Application of a paste bandage with very low recoil has little effect on resting pressure. Adding one and then two layers of bandaging raises resting pressures somewhat and increases dynamic pressures because of the short-stretch feature of the bandage.

bandage material that has little or no elastic fibers (short stretch) will stretch less than a material with many elastic fibers (long stretch). The most widely known "inelastic" bandage is Unna's boot, which after hardening, may act as a near-rigid shell around the limb. With muscle contraction, the reduced expansion of the bandage results in larger dynamic pressure ("working pressure") than would occur with highly elastic bandage types (Fig. 3).

Thus, under static conditions (muscles relaxed), SBPs generated by bandage compression depend on bandage structure and manner of wrapping. A low-stretch bandage made to form fit a limb without additional tension results in a low SBP (low resting pressure). In contrast, a high-stretch bandage that is applied with some amount of initial stretch results in an SBP that depends on the bandage restoring force and on limb contour and properties.

Under resting conditions, a portion of the bandage-related SBP is transmitted interiorly and serves to raise interstitial/tissue pressures (P_T), which thereby reduces vascular transmural pressures. Direct passive effects of this serve to reduce vessel diameters and reduce transcapillary filtration into tissue. Under dynamic conditions, internal pressures assume much greater values when low- or no-stretch bandages are used. This is explained by the fact that the effective dynamic compliance of the limb to volume expansion is less due to the relatively more rigid surface covering. Thus, rather than significant limb radial expansion, as in the case of a high-stretch bandage, expansion is limited with the short stretch, resulting in much higher dynamic or "working pressures" as shown in Figs. 2 and 3. These dynamic pressures play an important role in controlling edema/lymphedema via their favorable effects on interstitial fluid movement which, together with lymphatic activation, help reduce localized tissue edema.

1.2. Rationale for Compression

A basic goal of compression for treating venous ulcers is to try to normalize altered venous and microcirculatory hemodynamics that contribute to ulcer development and prolongation. Normal lower extremity venous hemodynamics and volumes rely

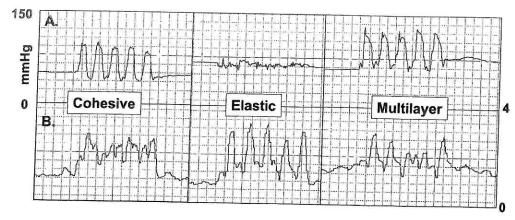


Figure 3 Low dynamic pressures with pure long-stretch bandages. Same measuring conditions as with Fig. 1. The cohesive bandage is associated with a low resting pressure but significant dynamic pressure. The pure elastic and multilayer bandages both are associated with higher resting pressures, but for the elastic bandage, there is virtually no dynamic pressure as the limb radial expansion has little restriction.

on valve competency of superficial, perforating, and deep venous systems that protect against both gravitational and muscle pump pressures. Normal venous return for muscle is via the deep system and via the superficial system for skin and subcutaneous structures. Properly functioning valves in perforating veins prevent superficial veins from being exposed to high pressures developed in deep veins, as they compress against fascia during calf contraction. They also permit unidirectional flow from superficial to deep during relaxation. Normally functioning deep veins, with competent valves, provide a unidirectional, low-resistance pathway for venous return, resulting in adequate blood volume ejection fraction to keep venous and leg volumes at normal levels.

Valve dysfunction alters this situation. If perforator vein valves are dysfunctional, some deep vein volume at high pressure is transmitted to the superficial system with each calf contraction. This may have at least three major effects: (1) effective ejection fraction for venous return from the deep system is reduced; (2) excessive pressures in the superficial system may further compromise valve competency; and (3) the sustained increase in venous volume effects microcirculation resulting in endothelial cell changes and an increase in outward flux of fluids and materials from capillaries and venules. Resultant changes in capillary hemodynamics, nutritional blood flow, and interstitium content and volume then follow.

Although the precise sequence whereby initiating hemodynamic changes end in skin ulceration is not fully worked out, there is strong evidence implicating a reduction in nutritional capillary density and degradation of capillary function (1). These changes may be due to retrograde dynamic pressures that are transmitted to nutritive capillaries (2), likely causing trauma and inflammatory-like responses (3). The venous hypertension may result in vessel rarefaction in a manner akin to that seen in systemic hypertension. Surprisingly, in spite of increased leg-blood flow in the ulcer region (4) and in periulcer subcutaneous microcirculation (5), transcutaneous oxygen is reduced. Normalization of microcirculatory parameters (6) is a positive feature of compression bandaging. In addition to microcirculatory effects, limb compression augments arterial flow pulsatility (7), which likely stimulates interstitial fluid and lymphatic dynamics and ulcer healing (Fig. 4). Thus, appropriate compression therapy may pre-empt ulcer formation in cases of chronic venous insufficiency and significantly aid in the healing of ulcers in part due to combined hemodynamic effects.

A basic goal of compression in the treatment of edema and lymphedema is to prevent further limb swelling and to facilitate limb-volume reduction. The form of bandaging/compression needed here may differ in detail from that needed for therapy related to venous ulcer treatment. A difference in bandaging approach relates to the importance here of achieving high dynamic pressures during the active treatment phase. Whereas static pressure enhancement is important in venous ulcer therapy to sustain vascular compression during resting conditions, in the case of lymphedema, elevated resting tissue pressures may in fact inhibit lymphatic function and thereby interstitial fluid removal. However, sufficient resting pressure is still needed to sustain gains made in decongestion.

1.3. Compression Bandaging Mechanisms of Action

Compression bandaging causes a "counter pressure" that is directed in such a fashion so as to reduce abnormally elevated transmural pressures of veins that may be caused by combined valve incompetence, gravitational forces, and muscular dynamics.

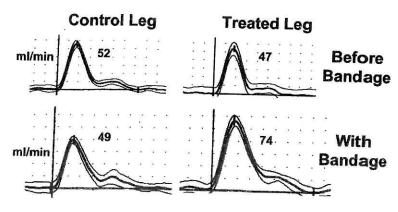


Figure 4 Compression bandaging augments pulsatile blood flow. Nuclear magnetic resonance was used to measure pulse blood flow at mid-calf with and without multilayer compression bandaging of one lower extremity (treated leg). Each flow pulse is the ensemble average of 30 beats and numbers indicate average flow per pulse in mL/min. Scales are the same for all pulses. Before bandaging, flow was similar in both limbs. With the bandage in place, pulsatile flow significantly increased resulting in a greater "dynamic" flow pattern. It is likely that this enhances tissue and lymphatic fluid movements.

The amount of counter pressure that is functionally effective depends on the type of bandage material that is used (highly extensible or relatively inelastic), the manner of wrapping (tightness of wrap), and whether the limb is relaxed (static conditions) or muscle is contracting (dynamic conditions). For a given set of conditions, the counter pressure needs to be sufficient to reduce abnormally elevated superficial venous volume by reducing overload volume entering the superficial system during muscular contraction and to maintain a lessened volume during static conditions. The resultant lessened average venous pressure tends to reduce excessive transcapillary outward filtration into interstitial spaces, which in turn reduces tissue edema. Simultaneously, a greater dynamic tissue pressure promotes greater tissue fluid movement and its uptake and removal by the lymphatic system, which adds to the edema-lessening process. Deep vein volume is also reduced because its effective ejection fraction is returned toward normal concomitant with the reduction in venous reflux.

The literature is replete with reports demonstrating the efficacy of compression bandaging as a main component in the treatment of venous ulcers (8–10). Most small ulcers, present for short durations (<6–9 months), tend to heal with good standard wound care combined with compression bandaging (11–13). However, questions still remain about the optimum approach to compression bandaging and the precise mode of action. One issue concerns the relative efficacies of long-stretch, short-stretch, or multilayer bandaging systems (14). A benefit has been suggested for multilayer when compared with short stretch (15), whereas others have found both to be equally effective (16,17). Still, others have advocated short-stretch bandaging to achieve therapeutically effective graduated compression (18). Other questions relate to achieving verifiable and appropriate compression pressures and gradients. Still, other questions relate to possible direct effects of the compression on the wound bed (19).

1.4. Bandage Compression Pressures (Sub-bandage and Tissue)

Limb bandaging with either single layer or multilayer combinations achieve SBPs between skin and bandage that depend on bandage material properties, extent of

bandage stretch as applied, number of layers used, wrapping pattern, and the structure, physical features, shape, and dimensions of the limb. As a simplified estimate to describe SBP dependency on limb size, the law of Laplace is often applied. Accordingly, for the same amount of tension in the bandage, SBP is inversely proportional to the circumference of the limb to which it is applied. As a consequence, if a bandage is applied to a lower extremity with equal bandage stretch along the limb length, then in theory, SBP varies along the limb, being greatest at limb sites with the least circumference. Actual pressure dependencies differ from this due to many factors including the fact that a limb is not a true cylinder and it has many nonuniformities in its contour and physical properties. Local regions, with small radii of curvature such as at the malleolus, have larger pressures. Shape distortions, as with significant lymphedema, also lead to unexpected pressure gradients (20). Nonetheless, it is useful to keep in mind that all else being equal, SBP tends to be greater, the smaller the circumference for the same bandage tension.

A direct outcome of this "principle" is that an ankle-to-knee bandage applied at uniform tension will result in an SBP gradient, with pressure being greatest at the ankle and least at the calf site with the maximum circumference. This is a fortuitous result with respect to the ability of a bandage to counteract gravitational dependencies of lower extremity intravascular pressures under resting conditions. However, the effect of such graduated pressures with respect to impacts of dynamic pressures is less clear. In fact, it is unclear which aspect—static or dynamic pressure gradients—is the most relevant parameter vis-à-vis ulcer healing potential. Considerable differences in both pressures have been observed with different bandage systems, as applied ostensibly in the same manner by experienced practitioners (21), although training improves the variability (22).

A part of the effectiveness of compression therapy depends on an increase in compression-related subcutaneous tissue pressure (PT) to diminish transcapillary fluid filtration and promote vascular resorption and removal of interstitial fluids. Local or generalized reduction in such edema or microedema allows better oxygen and nutrient delivery to, and chemical byproduct removal from, skin, subcutaneous tissues, and the wound bed. It should be noted that tissue pressure is already elevated in edematous limbs. For example in untreated lymphedema of the arm, P_T differences between affected and nonaffected arms averaged 4.6 cm H₂O (23) with even larger differences reported for the leg (24). This component of elevated P_T is due to excess tissue fluid and is diminished as compression therapy reduces fluid volume. However, promotion of fluid resorption by the compression-induced increase in P_T is not as great as would be indicated by the amount of P_T increase. This is because capillary pressure simultaneously increases by about 80% of the P_T increase (25). There is also evidence that SBP in the range of 20-40 mmHg may blunt the normal veno-arterial constriction response in dependent limbs (26,27). This effect would tend to maintain capillary pressure higher and thus detract from the desired decrease in capillary filtration.

If compression-induced tissue pressures become too large for too long, there is a negative impact on blood vessels and perfusion (28) and on lymphatic vessels and their lymph flow. Optimal tissue pressures have not been defined and there is little direct information as to relationships between surface SBPs and associated tissue pressures (29). Subdermal pressure measurements under compression garments give some idea of surface-to-subsurface radial pressure gradients to be expected at different sites (30). Absolute pressures are greater at bony prominences, but gradients are larger for soft tissue. For example, at the posterior mid-calf, an SBP of 66 mmHg

resulted in a subdermal pressure of 24 mmHg, whereas at medial mid-calf, an SBP of 36 mmHg resulted in a subdermal pressure of 21 mmHg.

2. PNEUMATIC COMPRESSION

Intermittent pneumatic compression (IPC) devices deliver controlled sequential pressures to a limb. Parameters of such systems include absolute pressures achieved, rate of pressure development, and for sequential systems, the timing. Considerable variation in parameters has been reported (31). Regarding IPC applications to venous ulcer treatment, a recent review of the literature (32) indicates that available evidence for IPC effectiveness is inconclusive. Thus, although some studies showed no benefit, others showed benefit if IPC was used in patients with long-standing, previously intractable ulcerations (33). As of this writing, IPC is covered only for patients with sustained edema and significant remaining ulceration after a 6-month trial of standard therapy. The potential therapeutic value of IPC for venous ulcers in immobile patients has been suggested but not established (34). An aspect not previously considered in this connection is the possible effects of IPC on promoting wound healing via direct nitric oxide pathways or related vasodilatory processes. Experimental studies (35) have shown that IPC upregulates eNOS mRNA and induces vasodilation of arterioles in noncompressed tissues. Variability in effectiveness of IPC therapy in venous ulcer patients may have roots in differences in these aspects among patients. In addition, greater IPC-induced vasodilatory effectiveness has been shown to be related to greater inflation velocity (36); so, variability in effectiveness may be due to differences in IPC-device parameters. In addition, there is evidence that IPC-related limb edema reduction in venous ulcer patients causes an increase in periwound oxygen tension (37). This may be unrelated to how the edema is reduced.

The possible utility of IPC as an adjunctive therapy for breast cancer treatment-related lymphedema is suggested by recent work in which IPC was added to standard decongestive lymphedema therapy and compared to standard therapy alone (38). Effectiveness of initial and maintenance phases of therapy improved with respect to limb-volume reductions in this small group of patients. It is unclear whether the mechanism of action is related to improved lymph fluid transport or reduction in capillary filtration into interstitial spaces (39). Sophisticated dynamic lymphoscintigraphy tests suggest an immediate IPC effect that facilitates tracer transport within edematous tissue in patients with lymphedema (40). This would be consistent with known IPC-related lymph flow augmentation in experimental animals (41). Use of IPC therapy in patients with filarial lymphedema has also been reported (42). However, IPC per se has not shown itself to improve limb-volume reductions when compared with complete decongestive physiotherapy (43) nor has a beneficial effect of IPC always been demonstrated (44). The IPC effectiveness for limb-volume reduction is inversely related to the extent of fibrosis present (45,46). There are also significant concerns about using pump therapy to treat lower extremity lymphedema, especially with regard to risk of causing genital lymphedema (47). Such risks do not often justify pump use for lower extremity lymphedema.

A retrospective study of patients with lower extremity critical ischemia and nonhealing ulcers suggests a positive benefit as measured by wound healing and limb salvage (48,49). These effects may be related to the improvement in arterial blood

flow associated with a very rapid rise in compression pressure (50,51). Other aspects of IPC clinical applications have been recently reviewed (52).

3. COMPRESSION GARMENTS

Maintenance of limb-volume reductions achieved during decongestive phases and prevention of reoccurrence of healed ulcers are aided or dependent on the use of compression garments. Elastic stockings provide an elevated resting pressure that mainly acts on superficial veins to help prevent gravitational overload and deepto-superficial reflux. Both standard and custom stockings are specified by class/grade corresponding to calf-ankle pressures (20–30, 30–40, 40–50 mmHg) they produce. Larger pressures project pressures to a greater depth and are associated with greater dynamic pressures.

However, even with the use of compression hosiery after healing, there may be a significant recurrence of venous ulceration. For example, of 502 ulcer legs initially treated with compression bandaging, 75% healed by 24 weeks but 44% recurred within 3 years (53). With respect to recurrence, patient compliance in using the compression stockings is an important factor; better compliance—less recurrence. In another study (54), of 62 patients initially treated with compression bandaging and subsequent maintenance compression hosiery, about two-third experienced venous ulcer recurrence at 4 years posthealing. Careful attention to stocking fit, absolute pressure levels achieved, ankle-to-calf pressure gradient, and patient compliance may improve this situation.

In addition to elastic-graded compression garments, inelastic sleeves and leg compression devices are available. They use inelastic fabric straps secured with Velcro to produce a graded compression. The amount of SBP produced is determined by how tightly the straps are pulled. Some types incorporate foam padding inner layer, whereas others can be worn over a separate padding layer or over compression garments to provide additional compression and support. Because of their inelasticity and adjustability, these devices have the benefits of short-stretch bandages and the convenience of easy application and removal. Their low resting pressures allow them to be worn at night to prevent reaccumulation of edema in patients who require constant compression.

4. ARTERIAL AND MICROCIRCULATORY BLOOD-FLOW CONSIDERATIONS

The potential impact of all forms of limb compression on arterial and microcirculatory flow needs to be considered from both functional and safety perspectives. Compression-induced pressures if too large, or in some cases even at levels therapeutically needed, may compromise blood circulation. This possibility is of particular concern if long-stretch compression is used, as elastic restoring forces are sustained under resting conditions. This is an issue in persons with normal limb circulation but of greater concern in persons with compromised circulation. In supine persons, skin-blood perfusion decreases with increasing levels of limb compression (55,56). Comparisons of the effects of various leg compression pressures on blood perfusion in skin overlying bone and in skin distal to leg compression (57) show significant reductions in both at therapeutically used pressures (Fig. 5).

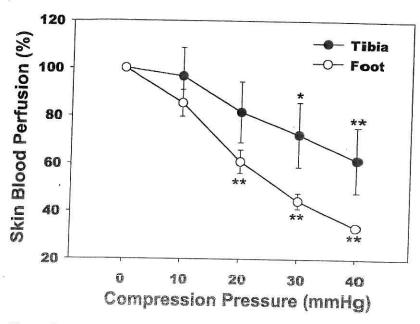


Figure 5 Compression-induced skin-blood perfusion reductions. Skin-blood perfusion using laser-Doppler was simultaneously measured overlying the anterior tibia and foot dorsum, as compression was applied from ankle-to-knee. Relative flow reduction was greater at the foot, but both regions demonstrated significant effects (*p < 0.05, **p < 0.01, when compared with no compression).

It is important to note that increases in tissue pressure, whether edema-induced or compression-related, are transmitted to veins and arteries, increasing their intravascular pressures. Because the pressure increase is greater in veins, the effective perfusion pressure (arteriovenous pressure difference) that drives blood flow through the compressed limb region is thus reduced. When edematous legs are elevated, venous pressure tends to decrease, but it cannot go below the abnormally increased tissue pressure. In contrast, intravascular pressures in arteries are reduced in proportion to the amount of leg elevation. Thus, under these circumstances, perfusion pressure is further reduced with a greater potential for blood-flow compromise. Such tendencies have been experimentally demonstrated (58).

In contrast to limb elevation, limb dependency causes a blood-flow reduction that is partly due to the increased venous volume, which reflexively induces an arteriolar vasoconstriction (27). Compression bandaging, even in normal subjects, helps reverse this blood-flow reduction and the associated tissue oxygen decrease (55). The bandage effect is due in part to unloading of venous stretch receptors and in part due to reduced arteriolar transmural pressure that results in a myogenic vasodilation in persons with normal vasodilatory capacity (59). However, similar compression pressures reduce blood flow when a person is supine (55) in both subcutaneous and skeletal muscle blood flow (60).

It is expected that in persons with micro- or macrocirculatory dysfunction, including blunted or absent vasodilatory reserve, compression-related flow decrements would be greater. These facts have led to the adage that compression bandaging should generally be withheld in patients with ABIs <0.80 and used with extreme care in patients with possible microcirculatory deficits such as in diabetes. It should also be recognized that the combination of edema, limb compression, and limb elevation might introduce further blood-flow reductions.

5. ASSESSING OUTCOMES

For venous ulcers, the effectiveness of compression therapy is measured by the rate and extent of healing, which is linked to the effectiveness in reducing the edema component. The effectiveness of compression therapy in lymphedema is measured by the lymphedema/edema reduction and by changes in tissue properties. The deformability of the tissue can be measured by applying a fixed amount of pressure for a specific amount of time and by determining the depth of penetration and/or the rate of tissue recovery. In very fibrotic tissues, even prolonged pressure will produce only minimal indentation. In normal tissues, the elasticity of the skin produces recoil and there is no prolonged indentation. Changes in depth and rate of recoil are indicators of the amount of fibrosis and edema in tissues. Edema reduction can be documented by calculating and tracking limb-volume changes. The size of a limb at selected points (i.e., ankle, calf) may change as compression is applied to those areas, whereas the total volume of the limb remains constant. When compression of the lower part of the limb produces a movement of edema into the upper part of the limb, more extensive compression and decongestive therapy is necessary. Therefore, it is important to determine the total limb volume. The effectiveness of home maintenance programs and patient compliance can also be monitored by tracking limb-volume changes.

Several methods are available for determining limb volume, such as water displacement, mathematical models based on circumferential measurements, and photoelectric instruments (61). Of these, the circumferential measurements are the easiest to use and require minimal equipment and staff time. Computer programs that automatically calculate, track, and graph limb volumes from circumferential measurements are available and are very useful (62). Professional reports and graphs provide a visual record of treatment progress and are valuable tools for communicating the effectiveness of treatment to referring physicians and other members of the medical team, to patients to enhance compliance with treatment, and to insurance companies to help assure reimbursement for treatment.

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