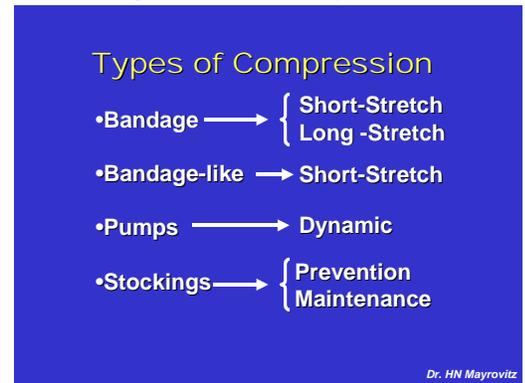


COMPRESSION THERAPY

This material is presented strictly as an educational resource for students and professionals.

Send questions/comments to: Dr. H N Mayrovitz, College of Medical Sciences, Nova Southeastern University, 3200 S. University Drive, Ft. Lauderdale, Florida 33328, 954-262-1313. mayrovit@nsu.nova.edu

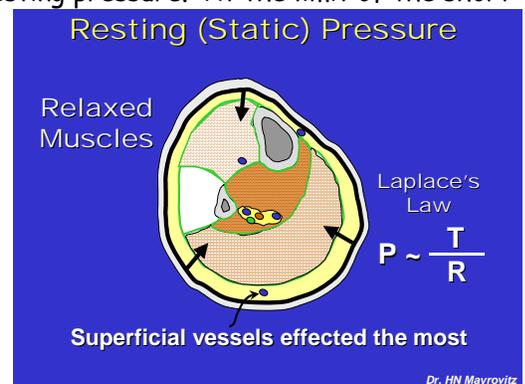
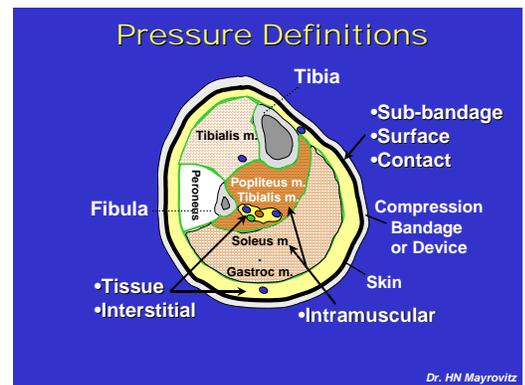
Therapeutic limb compression is used to prevent edema formation, to reduce existing edema, and to prevent re-accumulation of edema once it is reduced. Forms of therapeutic compression for limb edema, lymphedema and venous ulcers include compression bandaging, pneumatic compression and compression garments (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 paper describes major relevant aspects and offers some practical pearls and guidelines.



Compression Bandaging

A. 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 2-3 times the 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 a SBP in a muscularly relaxed limb from one in which muscular contraction is occurring resulting in a dynamic pressure. 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. Since 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 bandage. In the case of short-stretch and zero-stretch bandage, additional layers of bandaging can be used to increase pressures. 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 bandage material that has few 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 (figure 3).

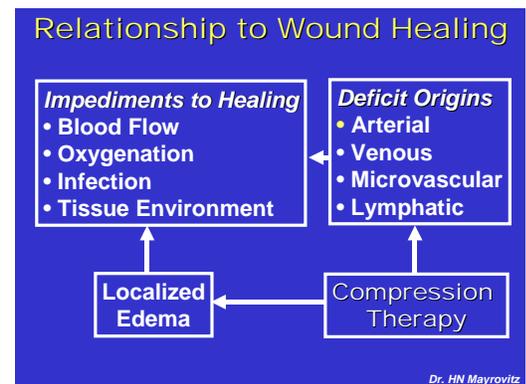
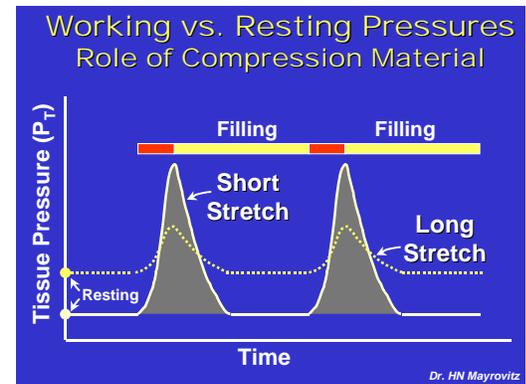
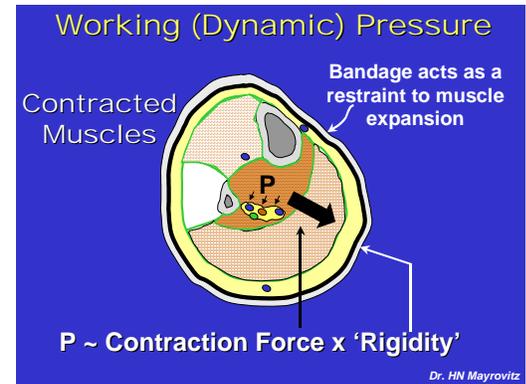
Thus, under static conditions (muscles relaxed) sub-bandage pressures generated by bandage compression depend on bandage structure and manner of wrapping. A low-stretch bandage made to form fit a limb without additional tension, results in a low SBP (low resting pressure). Contrastingly, a high-stretch bandage that is applied with some amount of initial stretch, results in a 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". These dynamic pressures play an important role in controlling edema/lymphedema via their favorable effects on interstitial fluid movement, which, together with lymphatic activation, helps reduce, localized tissue edema.

B. Rationale for compression in wound healing

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 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 are not fully worked out, there is strong evidence implicating a reduction in nutritional capillary density and degradation of capillary function¹. These changes may be due to retrograde dynamic pressures that are transmitted to nutritive capillaries², likely causing trauma and inflammatory-like responses³. 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 region⁴ and in peri-ulcer subcutaneous microcirculation⁵, transcutaneous oxygen is reduced. Normalization of microcirculatory parameters⁶ is a positive feature of compression bandaging. In addition to microcirculatory effects, limb compression augments arterial flow pulsatility⁷, which likely stimulates interstitial fluid and lymphatic dynamics and ulcer healing. Thus, appropriate compression therapy may preempt 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.

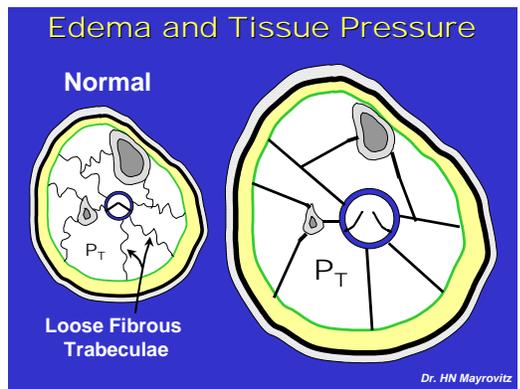
If Net Filtration Exceeds Lymphatic Transport Capacity

Overload = Edema
 + [Protein]
 = Lymphedema

Therapy Options

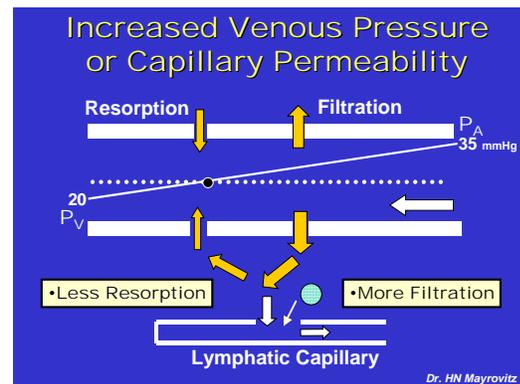
- Reduce Filtration
- Increase Transport

Dr. HN Mayrovitz



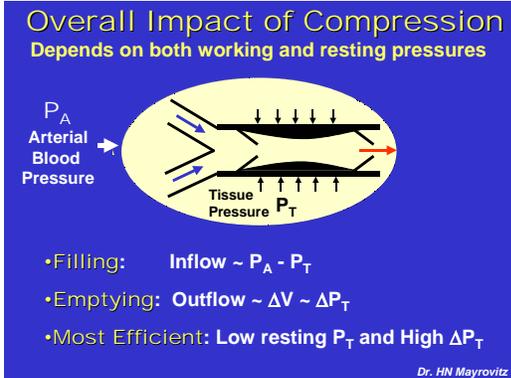
C. Compression Bandaging Mechanisms of Action

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. 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.



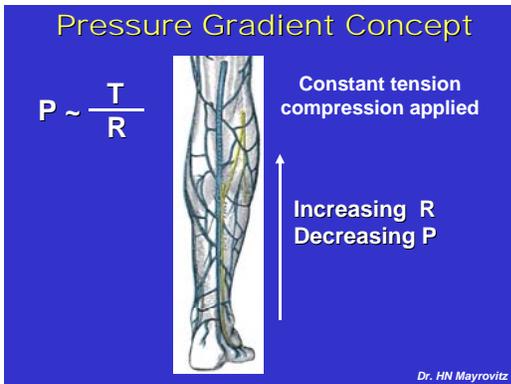
The literature is replete with reports demonstrating the efficacy of compression bandaging as a main component in the treatment of venous ulcers⁸⁻¹⁰. Most small ulcers, present for short durations (<6-9 months) tend

to heal with good standard wound care combined with compression bandaging¹¹⁻¹³. 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 systems¹⁴. A benefit has been suggested for multilayer as compared to short stretch¹⁵ whereas others have found both to be equally effective^{16,17}. Still others have advocated short stretch bandaging to achieve therapeutically effective graduated compression¹⁸. 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 bed¹⁹



D. Bandage Compression Pressures (sub-bandage and tissue)

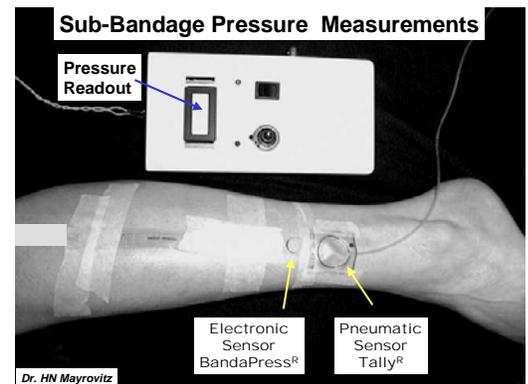
Limb bandaging with either single layer or multi-layer combinations achieve sub-bandage pressures between skin and bandage that depend on bandage material properties, extent of bandage stretch as applied, number of layers used, wrapping pattern and the structure, physical features, shape and dimensions 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 non-uniformities 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 gradients²⁰. None-the-less, 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.



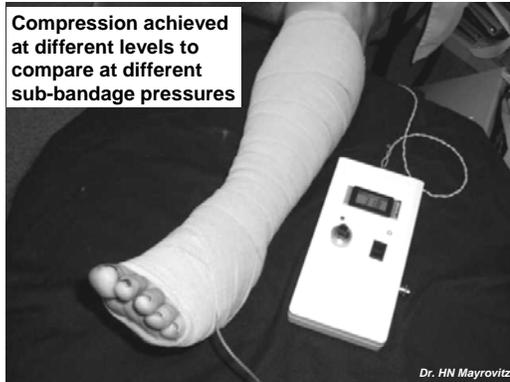
A direct outcome of this "principle" is that an ankle-to-knee bandage applied at uniform tension will result in a sub-bandage pressure gradient, with pressure being greatest at the ankle and least at the calf site with the maximum circumference. This is a fortuitous result with respect to the ability 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 are 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 in ostensibly the same manner by experienced practitioners²¹, although training has been reported to improve this variability²².

E. Measuring Sub-bandage pressures

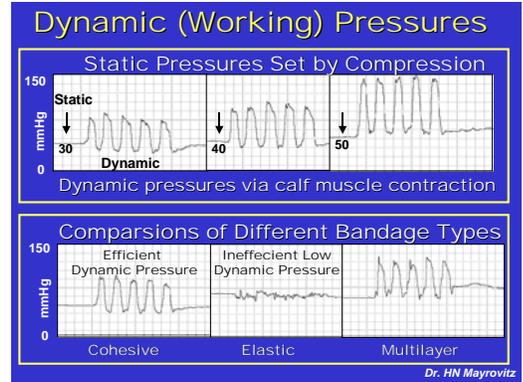
The accurate measurement of sub-bandage pressures has many technical pitfalls and interpretation of such measurements requires great care. This is true in part due to possible perturbing effects of pressure sensors placed to make the measurements and in part due to variability in limb contour and physical properties that contribute to significant variability in recorded pressures. However, using proper technique it is possible to obtain relative indices of the amount of sub-bandage pressures achieved by different bandages and bandage-systems for different application methods and wrapping tensions. The adjacent figure illustrates one such procedure using both a pneumatic sensor and a thin solid-state sensor. Such methods may be used to assess both static and dynamic pressures.



Using such a procedure it is possible to characterize various features of both static and dynamic pressures. In the



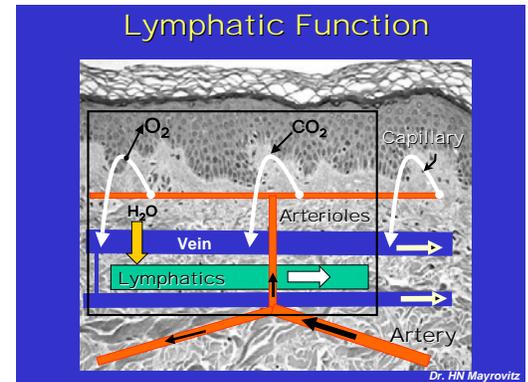
examples shown in the adjacent right figure, dramatic differences in dynamic pressures achieved with different bandage materials are clearly observed. The top panels shows the effect of wrapping tension that produces different levels of static pressure and the



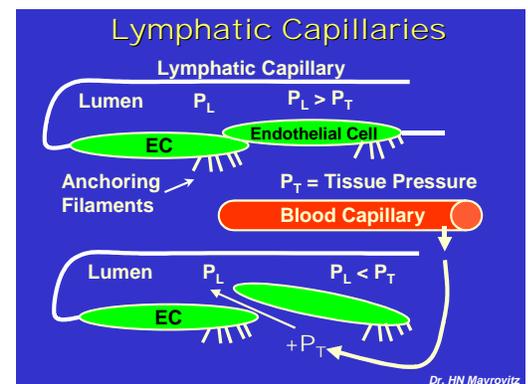
bottom panel shows a comparison of different dynamic pressures achieved with different bandage materials.

F. Tissue Pressure Issues

As noted previously, part of the effectiveness of compression therapy depends on an increase in compression-related subcutaneous tissue pressure (P_T) 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 non-affected arms averaged 4.6 cm H_2O ²³ with even larger differences reported for the leg²⁴. 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 increase²⁵. There is also evidence that sub-bandage pressure 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 perfusion²⁸ 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 sub-bandage pressures and associated tissue pressures²⁹. Subdermal pressure measurements under compression garments give some idea of surface-to-subsurface radial pressure gradients to be expected at different sites³⁰. Absolute pressures are greater at bony prominences but gradients are larger for soft tissue. For example, at the posterior mid-calf a sub-bandage pressure of 66 mmHg resulted in a subdermal pressure of 24 mmHg whereas at medial mid-calf, a SBP of 36 mmHg resulted in a subdermal pressure of 21 mmHg.



II. 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 reported³¹. Regarding IPC applications to venous ulcer treatment, a recent review of the literature³², 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 ulcerations³³. 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 established³⁴. 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 studies³⁵, 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 velocity³⁶, so variability in effectiveness may be due to differences in IPC device parameters. Also, there is evidence that IPC-related limb edema reduction in venous ulcer patients causes an increase in periwound oxygen tension³⁷. 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 alone³⁸. 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 spaces³⁹. Sophisticated dynamic lymphoscintigraphy tests suggest an immediate IPC effect that facilitates tracer transport within edematous tissue in patients with lymphedema⁴⁰. This would be consistent with known IPC-related lymph flow augmentation in experimental animals⁴¹. Use of IPC therapy in patients with filarial lymphedema has also been reported⁴². However, IPC per se has not shown itself to improve limb volume reductions as compared to complete decongestive physiotherapy⁴³ nor has a beneficial effect of IPC always been demonstrated⁴⁴. 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 lymphedema⁴⁷. 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 suggest 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 reviewed⁵².

III. Compression Garments

Maintenance of limb volume reductions achieved during decongestive phases and prevention of reoccurrence of healed ulcers is 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 deep-to-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 three years⁵³. With respect to recurrence, patient compliance in using the compression stockings is an important factor; better compliance - less recurrence. In another study⁵⁴, of 62 patients initially treated with compression bandaging and subsequent maintenance compression hosiery, about 2/3 experienced venous ulcer recurrence at four years post healing. 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 a foam padding inner layer, while 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 re-accumulation of edema in patients who require constant compression

IV. 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 since 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 compression⁵⁷ show significant reductions in both at therapeutically used pressures (figure 5).

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. Contrastingly, intravascular pressures in arteries are reduced in proportion to the amount of leg elevation and perfusion pressure is further reduced with a greater potential for blood flow compromise. Such tendencies have been experimentally demonstrated⁵⁸.

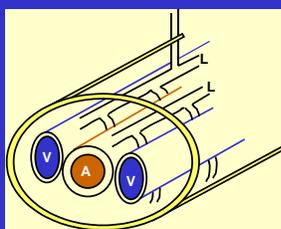
In contrast to limb elevation, limb dependency causes a blood flow reduction, partly due to the increased venous volume, which reflexively induces an arteriolar vasoconstriction²⁷. Compression bandaging, even in normal subjects, helps reverse this blood flow reduction and the associated tissue oxygen decrease⁵⁵. The bandage effect is due in part to unloading of venous stretch receptors, and in part due to reduced arteriolar transmural pressure results in a myogenic vasodilation in persons with normal vasodilatory capacity⁵⁹. However, similar compression pressures reduce blood flow when a person is supine⁵⁵ in both subcutaneous and skeletal muscle blood flow⁶⁰.

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 may introduce further blood flow reductions.

V. Arterial Blood Flow Pulsations and Lymphatic Transport

A variety of mechanical events and processes have an impact on lymph flow and thereby on the efficacy of

Vascular Sheath

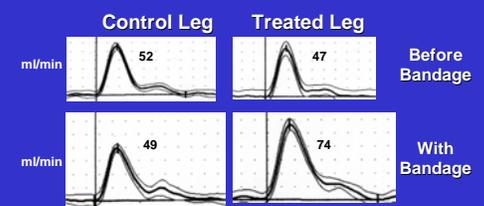


Arterial Pulsations Mechanically Augment Lymph Transport

Dr. HN Mayrovitz

the lymphatic system to maintain normal interstitial fluid volumes, or in the case of edema to reduce such edema. These processes include stimulation of lymphangion contraction, the calf muscle pump, various body movements and respiration. Because of the anatomical arrangements between arteries and lymphatic

Arterial Flow Pulses



Below Knee Blood Flow Determined by Nuclear Magnetic Resonance

Dr. HN Mayrovitz

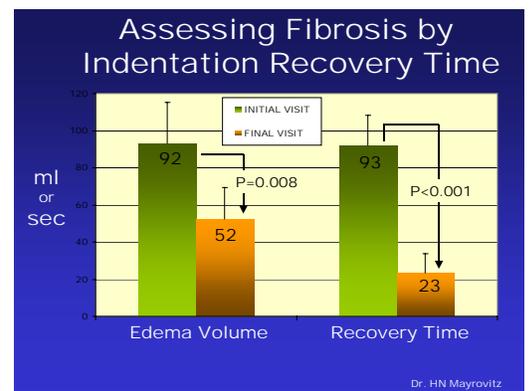
and venous vessels, arterial pulsations can have an important effect. Thus factors that increase such arterial pulsations might be expected to be beneficial in the sense of augmenting lymph movement. Such augmentations in lower extremity arterial flow pulses have in fact been shown using nuclear magnetic resonance flowmetry^{7,61} when lower extremities are evaluated with compression bandaging in place.

VI. 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 of the therapy to reduce the edema component. The effectiveness of compression therapy in lymphedema is in part measured by the lymphedema/edema reduction, in part by changes in tissue properties and in part by recovery of function. Applying a fixed amount of force for a specific amount of time and determining the depth of penetration and/or the rate of tissue recovery can measure the deformability of the tissue. In very fibrotic tissues, even large or prolonged force applications may produce only minimal indentation. In normal tissues, the elasticity of the skin produces recoil and there is no prolongation of recovery to the indetation. Changes in the rate of recovery are indicators of the amount of fibrosis and edema in tissues. A novel and economical method to assess and document such changes in tissue properties relies on the recovery time to a standardized indentation⁶².



Calculating and tracking limb volume changes can document edema reduction. The size of a limb at selected points (i.e. ankle, calf) may change as compression is applied to those areas, while the total volume of the limb remains constant. When compression of the lower part of the limb produces a movement of edema fluid into the upper part of the limb, more extensive compression and decongestive therapy may be necessary. Therefore it is important to determine the total limb volume. Tracking limb volume changes can also monitor the effectiveness of home maintenance programs and patient compliance.



Several methods are available for determining limb volume including water displacement, mathematical models based on circumferential measurements, and photoelectric instruments⁶³. Of these, the circumferential measurements are the easiest to use, and require minimal equipment and staff time. Deviations in limb contour from circularity have a minimal effect on such determinations unless there are significant differences between the maximum and minimum diameter at a given limb location.⁶⁴ Computer programs that automatically calculate, track, and graph limb volumes from circumferential measurements are available and are very useful.⁶⁵

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. Special attention needs to be paid to measurements of hand and foot volumes since their shapes deviate significantly from circularity. Special algorithms based on metric measurements and confirmed by water displacement methods have been developed and are available.⁶⁶ Since non-inclusion of hand or foot volumes in the assessment of therapy may lead to erroneous conclusions it is recommended that when possible these be included in overall limb volume assessments.

Documenting and Reporting Limb Volume Changes

| Visit | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 |
|-----------------------|------|------|------|-------|-------|-------|-------|-------|-------|------|----|----|
| Ta Limb Volume (ml) | 2969 | 2979 | 2746 | 2674 | 2599 | 2599 | 2579 | 2575 | 2492 | 2471 | 0 | 0 |
| Norm Limb Volume (ml) | 1961 | 1961 | 1961 | 1961 | 1961 | 1961 | 1961 | 1961 | 1961 | 1961 | 0 | 0 |
| %Edema | 51.4 | 43.8 | 48.1 | 36.4 | 32.1 | 38.5 | 29.8 | 29.3 | 27.1 | 26.1 | | |
| Ta: % Vol change | -5.0 | -7.5 | -9.9 | -12.8 | -13.8 | -14.8 | -15.3 | -16.1 | -16.1 | | | |
| Norm: % Vol change | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | -1.6 | | |

Note: In the above graphics, Visit refers to patient visits during which limb volume measurements were made and recorded.

Comments

This patient is part of the Lymphedema Fibrosis-Oxygen study, 58 year old with initial lymphedema starting 2 years after primary surgery and has been present since July 2000.

Initial whole arm percentage edema was determined to be 51.4% which was reduced to 26.1% with CDP.

Limb Volumes Professional V 3.0 Copyright 2004 Bioscience Research Institute

References

1. Junger M, Hahn M, Klyszcz T, Steins A. Microangiopathy in the pathogenesis of chronic venous insufficiency. *Curr Probl Dermatol* 1999;27:124-9.
2. Steins A, Hahn M, Junger M. Venous leg ulcers and microcirculation. *Clin Hemorheol Microcirc* 2001;24(3):147-53.
3. Hahn J, Junger M, Friedrich B, et al. Cutaneous inflammation limited to the region of the ulcer in chronic venous insufficiency. *Vasa* 1997;26(4):277-81.
4. Mayrovitz HN, Larsen PB. Leg blood flow in patients with venous ulcers: Relationship to site and ulcer area. *Wounds* 1994;6:195-200.
5. Mayrovitz HN, Larsen PB. Periwound skin microcirculation of venous leg ulcers. *Microvasc Res* 1994;48(1):114-23.
6. Junger M, Steins A, Hahn M, Hafner HM. Microcirculatory dysfunction in chronic venous insufficiency (CVI). *Microcirculation* 2000;7(6):S3-12.
7. Mayrovitz HN, Larsen PB. Effects of compression bandaging on leg pulsatile blood flow. *Clin Physiol* 1997;17(1):105-17.
8. Moffatt CJ, O'Hare L. Venous leg ulceration: treatment by high compression bandaging. *Ostomy Wound Manage* 1995;41(4):16-8, 20, 22-5.
9. Fletcher A, Cullum N, Sheldon TA. A systematic review of compression treatment for venous leg ulcers. *Bmj* 1997;315(7108):576-80.
10. Palfreyman SJ, Lochiel R, Michaels JA. A systematic review of compression therapy for venous leg ulcers. *Vasc Med* 1998;3(4):301-13.
11. Mayberry JC, Moneta GL, Taylor LM, Jr., Porter JM. Fifteen-year results of ambulatory compression therapy for chronic venous ulcers. *Surgery* 1991;109(5):575-81.
12. Margolis DJ, Berlin JA, Strom BL. Which venous leg ulcers will heal with limb compression bandages? *Am J Med* 2000;109(1):15-9.
13. Phillips TJ. Current approaches to venous ulcers and compression. *Dermatol Surg* 2001;27(7):611-21.
14. Macdonald J, Sims N, Mayrovitz HN. Lymphedema, lipedema, and the open wound: The role of compression therapy. *Surg Clin N Am* 2003;83:639-658.
15. Ukat A, Konig M, Vanscheidt W, Munter KC. Short-stretch versus multilayer compression for venous leg ulcers: a comparison of healing rates. *J Wound Care* 2003;12(4):139-43.
16. Scriven JM, Taylor LE, Wood AJ, Bell PR, Naylor AR, London NJ. A prospective randomised trial of four-layer versus short stretch compression bandages for the treatment of venous leg ulcers. *Ann R Coll Surg Engl* 1998;80(3):215-20.
17. Partsch H, Menzinger G, Mostbeck A. Inelastic leg compression is more effective to reduce deep venous refluxes than elastic bandages. *Dermatol Surg* 1999;25(9):695-700.
18. Hampton S. Venous leg ulcers: short-stretch bandage compression therapy. *Br J Nurs* 1997;6(17):990-2, 994, 996-8.
19. Roberts G, Hammad L, Collins C, Shearman C, Mani R. Some effects of sustained compression on ulcerated tissues. *Angiology* 2002;53(4):451-6.
20. Williams AF, Williams AE. 'Putting the pressure on': a study of compression sleeves used in breast cancer-related lymphoedema. *J Tissue Viability* 1999;9(3):89-94.
21. Wertheim D, Melhuish J, Williams R, Harding K. Measurement of forces associated with compression therapy. *Med Biol Eng Comput* 1999;37(1):31-4.
22. Hafner J, Luthi W, Hanssle H, Kammerlander G, Burg G. Instruction of compression therapy by means of interface pressure measurement. *Dermatol Surg* 2000;26(5):481-6; discussion 487.
23. Bates DO, Levick JR, Mortimer PS. Subcutaneous interstitial fluid pressure and arm volume in lymphoedema. *Int J Microcirc Clin Exp* 1992;11(4):359-73.

24. Christenson J, Shawa N, Hamad M, Al-Hassan H. The relationship between subcutaneous tissue pressures and intramuscular pressures in normal and edematous legs. *Microcirculation, Endothelium and Lymphatics* 1985;2:367-384.
25. Mellander S, Albert U. Effects of increased and decreased tissue pressure on haemodynamic and capillary events in cat skeletal muscle. *J Physiol* 1994;481(Pt 1):163-75.
26. Nielsen HV. Effects of externally applied compression on blood flow in the human dependent leg. *Clin Physiol* 1983;3(2):131-40.
27. Mayrovitz HN. Posturally induced leg vasoconstrictive responses: relationship to standing duration, impedance and volume changes. *Clin Physiol* 1998;18(4):311-9.
28. Matsen FA, 3rd, Krugmire RB, Jr., King RV. Nicolas Andry Award. Increased tissue pressure and its effects on muscle oxygenation in level and elevated human limbs. *Clin Orthop* 1979(144):311-20.
29. Hargens AR, McClure AG, Skyhar MJ, Lieber RL, Gershuni DH, Akeson WH. Local compression patterns beneath pneumatic tourniquets applied to arms and thighs of human cadavera. *J Orthop Res* 1987;5(2):247-52.
30. Giele HP, Liddiard K, Currie K, Wood FM. Direct measurement of cutaneous pressures generated by pressure garments. *Burns* 1997;23(2):137-41.
31. Rithalia SV, Heath GH, Gonsalkorale M. Evaluation of intermittent pneumatic compression systems. *J Tissue Viability* 2002;12(2):52-7.
32. Berliner E, Ozbilgin B, Zarin DA. A systematic review of pneumatic compression for treatment of chronic venous insufficiency and venous ulcers. *J Vasc Surg* 2003;37(3):539-44.
33. Smith PC, Sarin S, Hasty J, Scurr JH. Sequential gradient pneumatic compression enhances venous ulcer healing: a randomized trial. *Surgery* 1990;108(5):871-5.
34. Vowden K. The use of intermittent pneumatic compression in venous ulceration. *Br J Nurs* 2001;10(8):491-509.
35. Chen LE, Liu K, Qi WN, et al. Role of nitric oxide in vasodilation in upstream muscle during intermittent pneumatic compression. *J Appl Physiol* 2002;92(2):559-66.
36. Liu K, Chen LE, Seaber AV, Urbaniak JR. Influences of inflation rate and duration on vasodilatory effect by intermittent pneumatic compression in distant skeletal muscle. *J Orthop Res* 1999;17(3):415-20.
37. Kolari PJ, Pekanmaki K, Pohjola RT. Transcutaneous oxygen tension in patients with post-thrombotic leg ulcers: treatment with intermittent pneumatic compression. *Cardiovasc Res* 1988;22(2):138-41.
38. Szuba A, Achalu R, Rockson SG. Decongestive lymphatic therapy for patients with breast carcinoma-associated lymphedema. A randomized, prospective study of a role for adjunctive intermittent pneumatic compression. *Cancer* 2002;95(11):2260-7.
39. Miranda F, Jr., Perez MC, Castiglioni ML, et al. Effect of sequential intermittent pneumatic compression on both leg lymphedema volume and on lymph transport as semi-quantitatively evaluated by lymphoscintigraphy. *Lymphology* 2001;34(3):135-41.
40. Baulieu F, Baulieu JL, Vaillant L, Secchi V, Barsotti J. Factorial analysis in radionuclide lymphography: assessment of the effects of sequential pneumatic compression. *Lymphology* 1989;22(4):178-85.
41. McGeown JG, McHale NG, Thornbury KD. Effects of varying patterns of external compression on lymph flow in the hindlimb of the anaesthetized sheep. *J Physiol* 1988;397:449-57.
42. Manjula Y, Kate V, Ananthakrishnan N. Evaluation of sequential intermittent pneumatic compression for filarial lymphoedema. *Natl Med J India* 2002;15(4):192-4.
43. Johansson K, Lie E, Ekdahl C, Lindfeldt J. A randomized study comparing manual lymph drainage with sequential pneumatic compression for treatment of postoperative arm lymphedema. *Lymphology* 1998;31(2):56-64.
44. Dini D, Del Mastro L, Gozza A, et al. The role of pneumatic compression in the treatment of postmastectomy lymphedema. A randomized phase III study. *Ann Oncol* 1998;9(2):187-90.
45. Raines JK, O'Donnell TF, Jr., Kalisher L, Darling RC. Selection of patients with lymphedema for compression therapy. *Am J Surg* 1977;133(4):430-7.
46. Pappas CJ, O'Donnell TF, Jr. Long-term results of compression treatment for lymphedema. *J Vasc Surg* 1992;16(4):555-62.

47. Boris M, Weindorf S, Lasinski BB. The risk of genital edema after external pump compression for lower limb lymphedema. *Lymphology* 1998;31(1):15-20.
48. Montori VM, Kavros SJ, Walsh EE, Rooke TW. Intermittent compression pump for nonhealing wounds in patients with limb ischemia. The Mayo Clinic experience (1998-2000). *Int Angiol* 2002;21(4):360-6.
49. van Bemmelen PS, Gitlitz DB, Faruqi RM, et al. Limb salvage using high-pressure intermittent compression arterial assist device in cases unsuitable for surgical revascularization. *Arch Surg* 2001;136(11):1280-5; discussion 1286.
50. Eze AR, Cisek PL, Holland BS, Comerota AJ, Jr., Verramasuneni R, Comerota AJ. The contributions of arterial and venous volumes to increased cutaneous blood flow during leg compression. *Ann Vasc Surg* 1998;12(2):182-6.
51. Eze AR, Comerota AJ, Cisek PL, et al. Intermittent calf and foot compression increases lower extremity blood flow. *Am J Surg* 1996;172(2):130-4; discussion 135.
52. Chen AH, Frangos SG, Kilaru S, Sumpio BE. Intermittent pneumatic compression devices -- physiological mechanisms of action. *Eur J Vasc Endovasc Surg* 2001;21(5):383-92.
53. Barwell JR, Taylor M, Deacon J, et al. Surgical correction of isolated superficial venous reflux reduces long-term recurrence rate in chronic venous leg ulcers. *Eur J Vasc Endovasc Surg* 2000;20(4):363-8.
54. McDaniel HB, Marston WA, Farber MA, et al. Recurrence of chronic venous ulcers on the basis of clinical, etiologic, anatomic, and pathophysiologic criteria and air plethysmography. *J Vasc Surg* 2002;35(4):723-8.
55. Gaylarde PM, Sarkany I, Dodd HJ. The effect of compression on venous stasis. *Br J Dermatol* 1993;128(3):255-8.
56. Mayrovitz HN, Delgado M, Smith J. Compression bandaging effects on lower extremity peripheral and sub-bandage skin blood perfusion. *Ostomy Wound Manage* 1998;44(3):56-65.
57. Mayrovitz HN, Sims N. Effects of ankle-to-knee external pressures on skin blood perfusion under and distal to compression. *Adv Skin Wound Care* 2003;(in press).
58. Matsen FA, 3rd, Wyss CR, Krugmire RB, Jr., Simmons CW, King RV. The effects of limb elevation and dependency on local arteriovenous gradients in normal human limbs with particular reference to limbs with increased tissue pressure. *Clin Orthop* 1980(150):187-95.
59. Nielsen HV. External pressure--blood flow relations during limb compression in man. *Acta Physiol Scand* 1983;119(3):253-60.
60. Nielsen HV. Effects of externally applied compression on blood flow in subcutaneous and muscle tissue in the human supine leg. *Clin Physiol* 1982;2(6):447-57.
61. Mayrovitz HN. Compression-induced pulsatile blood flow changes in human legs. *Clin Physiol* 1998;18:117-24.
62. Bioscience Research Institute. <http://bioscience-research.net/tissupress.html>. 2004.
63. Mayrovitz HN, Sims N, Macdonald J. Limb volume measurement in patients with edema. *Adv Skin Wound Care* 2000;113:272-276.
64. Mayrovitz HN. Limb volume estimates based on limb elliptical vs. circular cross section models. *Lymphology* 2003;36(3):140-3.
65. Bioscience Research Institute. <http://bioscience-research.net/lymphedema.html>. 2004.
66. Bioscience Research Institute. <http://bioscience-research.net/footvolumes.html>. 2004.