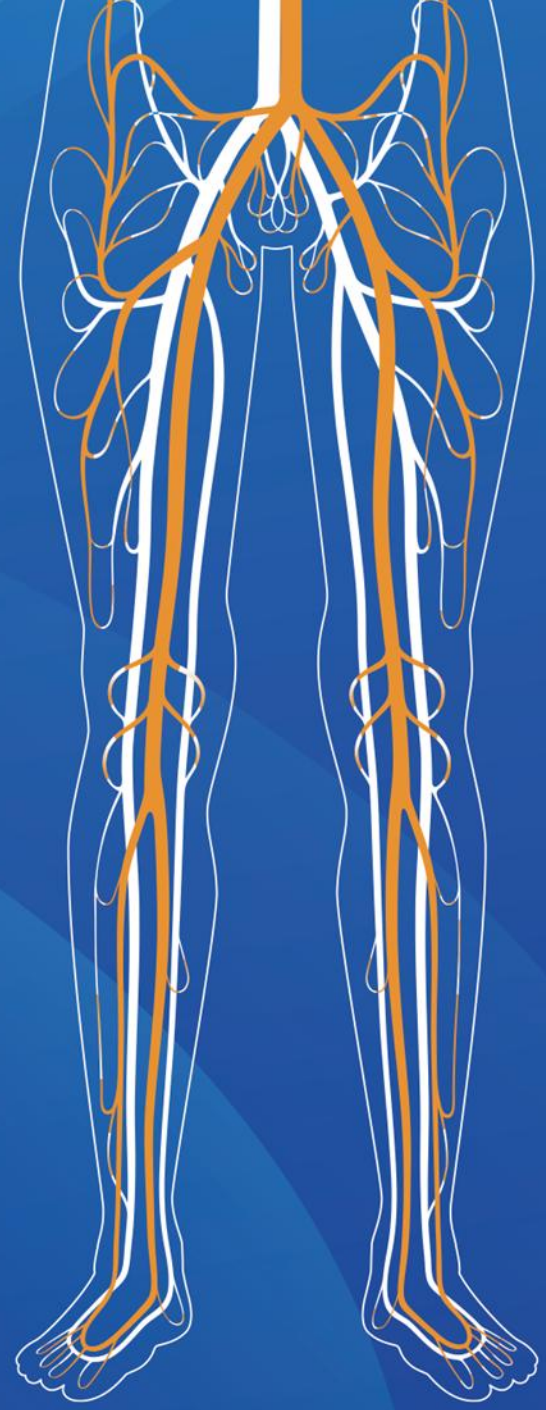
Beyond the Veins:

Harnessing Compression to Heal Skin, Control Swelling, and Improve Flow



Supported by an educational grant from Urgo Medical North America

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Faculty Disclosures

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Disclosures

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Submit Questions via the Question Box at any Time

We look forward to hearing from you!

Learning Objectives

1. Describe the current gold standard for compression therapy in managing venous reflux and evaluate the supporting evidence
2. Explain the relationship between venous disease and lymphedema and assess the effectiveness of compression therapy in managing both conditions concurrently
3. Examine the pathophysiology of venous dermatitis and analyze clinical data and concepts supporting the role of compression therapy in improving skin perfusion and condition
4. Summarize the multi-benefit potential of effective compression therapy in simultaneously managing venous reflux, lymphedema, and venous dermatitis

Venous Leg Ulcers:
Is That the Whole Story?

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Venous Leg Ulcers (VLUs): A Growing Clinical Challenge

- Affect 1%–3% of the adult population, especially over age 65
- Associated with chronic venous insufficiency (CVI)
- Recurrence rate: up to 70% within 3yrs
- Delayed healing: >50% remain unhealed after 6mos
- High symptom burden: pain, drainage, odor, immobility

Cost Burden

- Estimated $14.9 billion/yr in U.S. healthcare costs (Nussbaum, et al. 2018)
- Hospital readmissions and home health visits drive cost
- Significant impact on quality of life and productivity

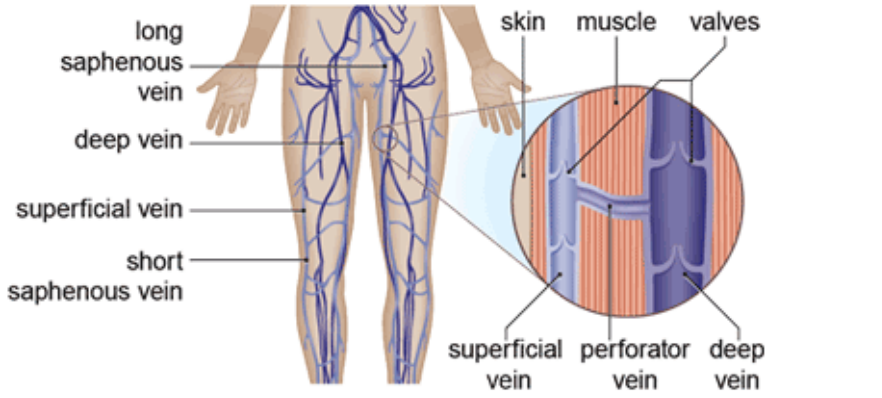
CVI = chronic venous insufficiency.

Nussbaum SR, et al. *Value Health*. 2018;21(1):27-32.

Venous System (Classical Teaching)

- Three working together
  - Veins
  - Valves
  - Calf pump

- Veins
  - Deep veins
  - Superficial veins
  - Perforating veins

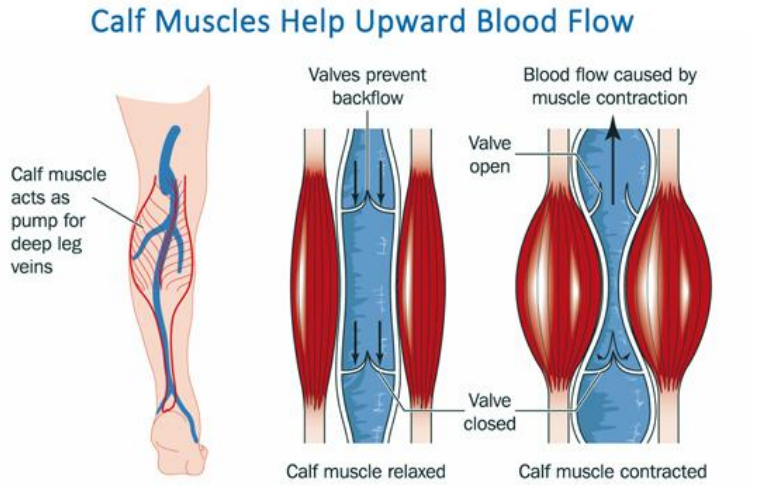


Superficial veins of the leg

Image credit: Bupa

WHAT IS MISSING HERE ?

- SKIN
- LYMPHATICS



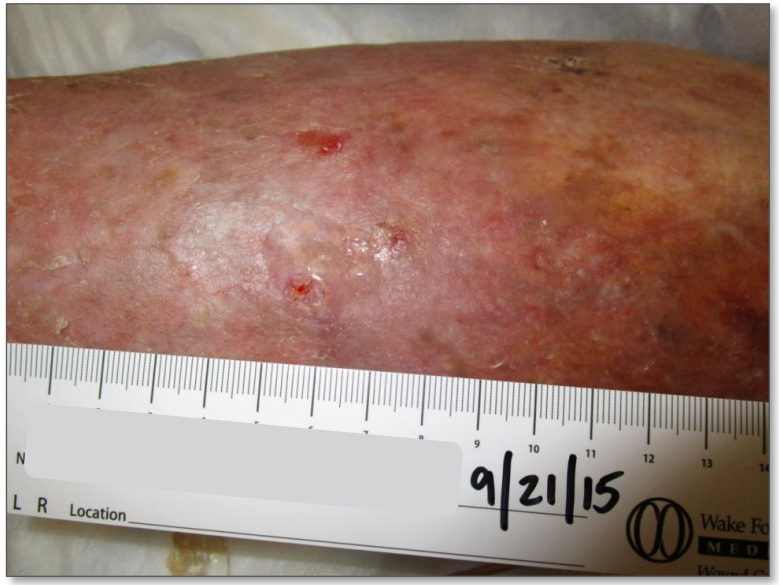
Venous Hypertension

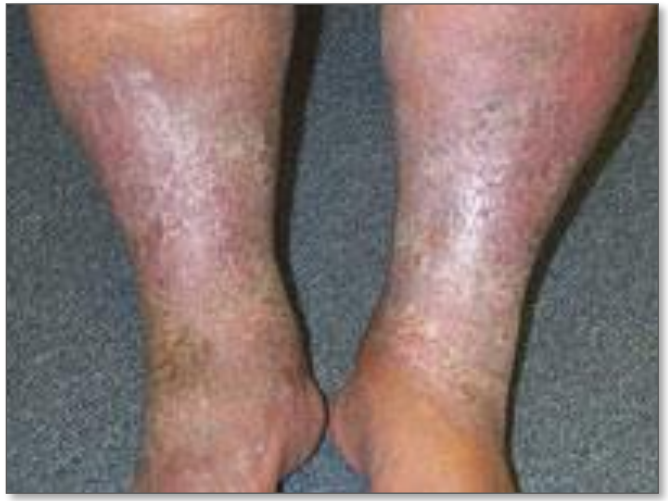
Chronic venous disease (CVD) is a spectrum disorder

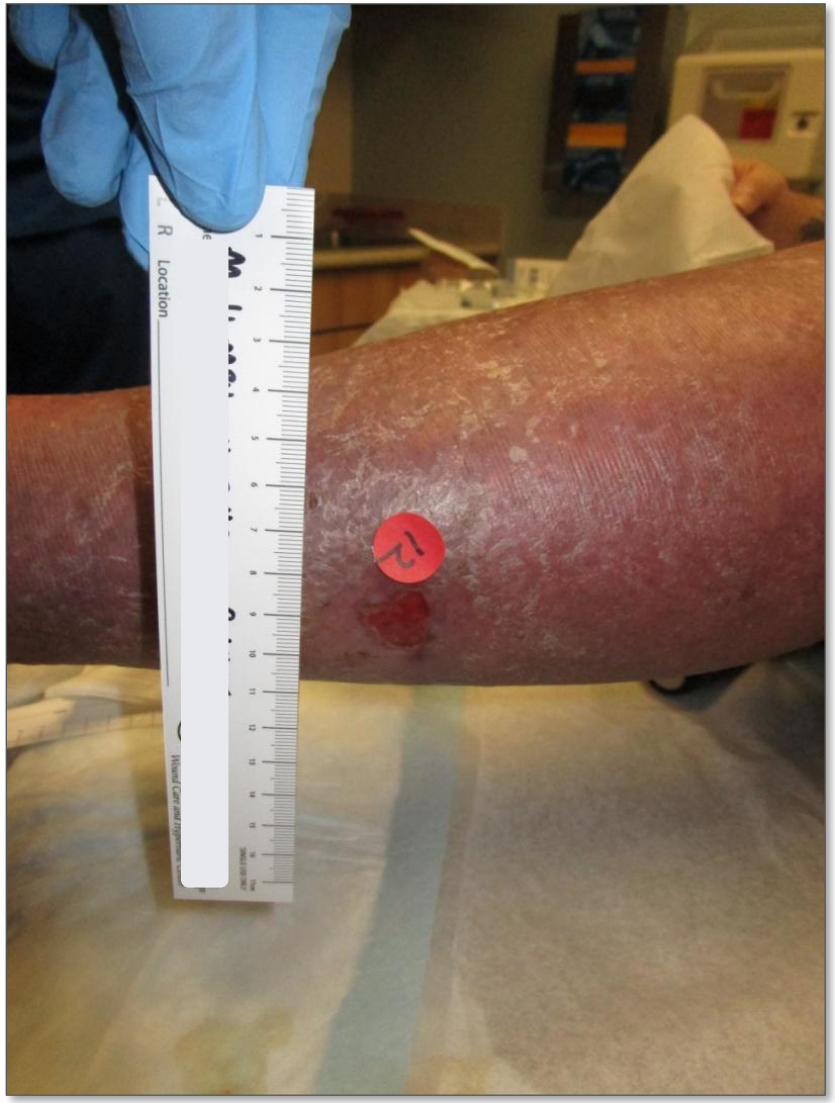
- Leg fatigue, heaviness
- Telangiectasia/reticular veins
- Varicose veins
- Skin pigmentation / dermatitis
- Fibrosing panniculitis (lipodermatosclerosis)
- Atrophie blanche or livedoid vasculopathy
- Venous ulceration

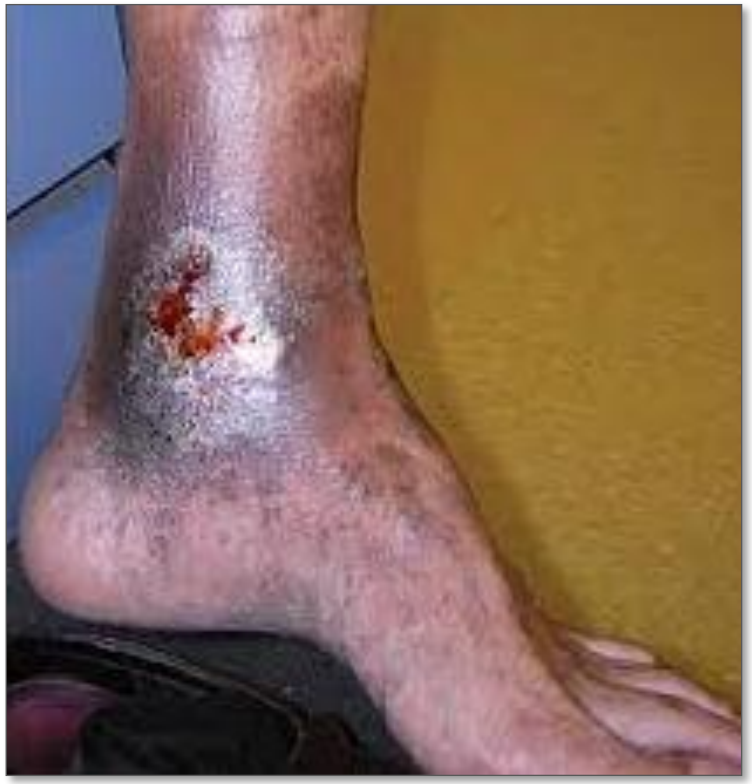
CVD = chronic venous disease.

What Is Going on with This Skin?

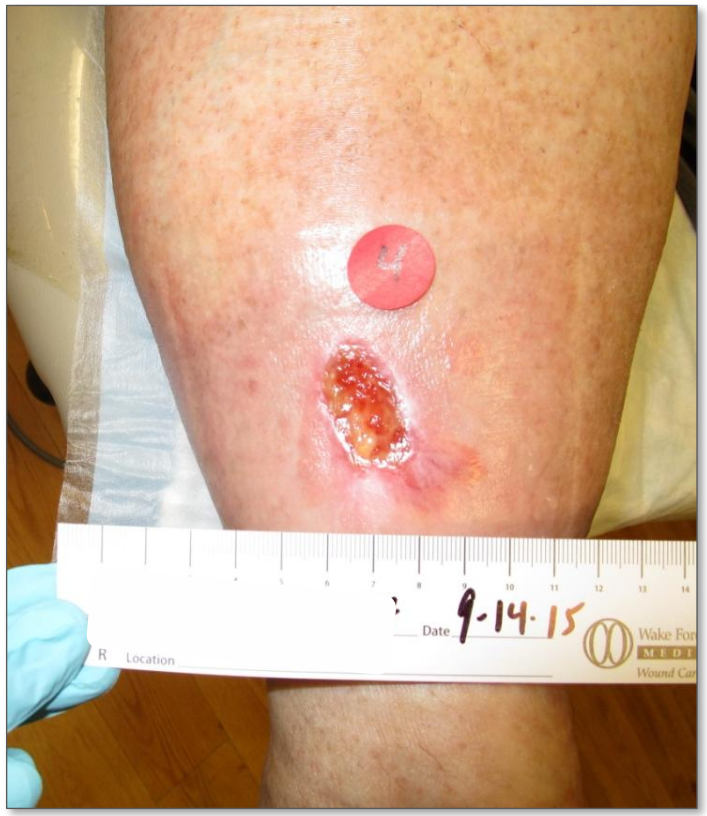


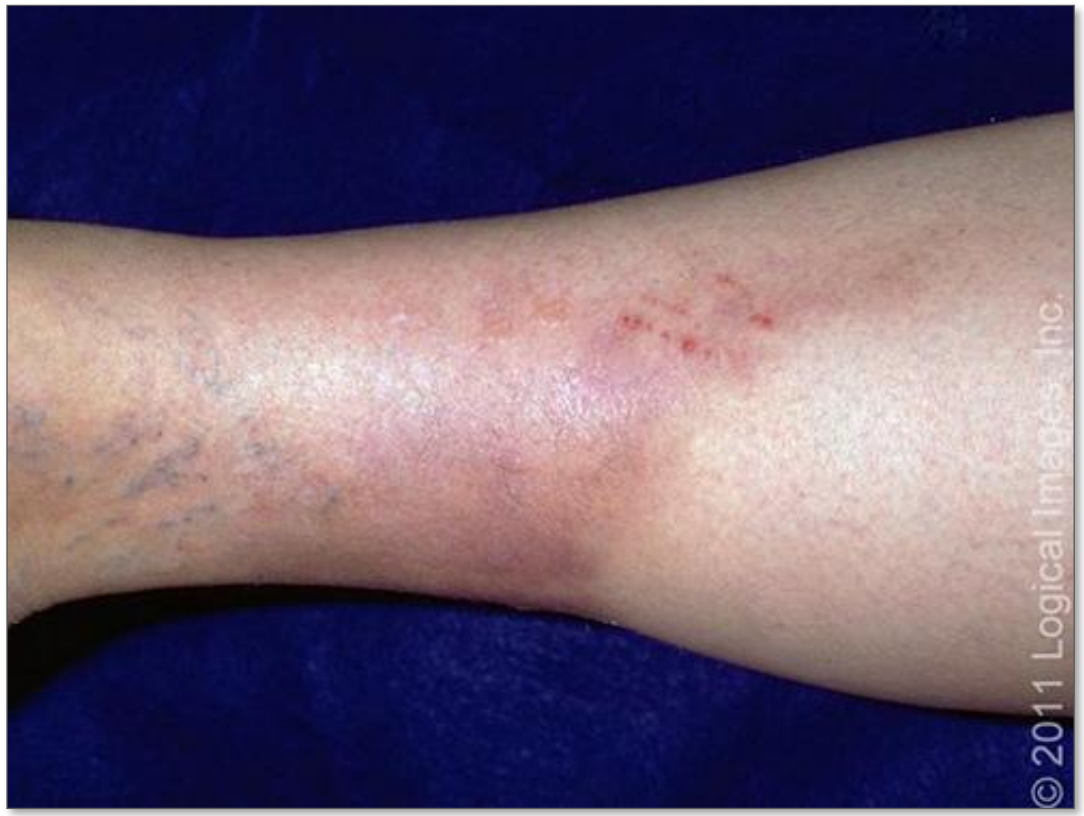






This Skin Is Not Normal

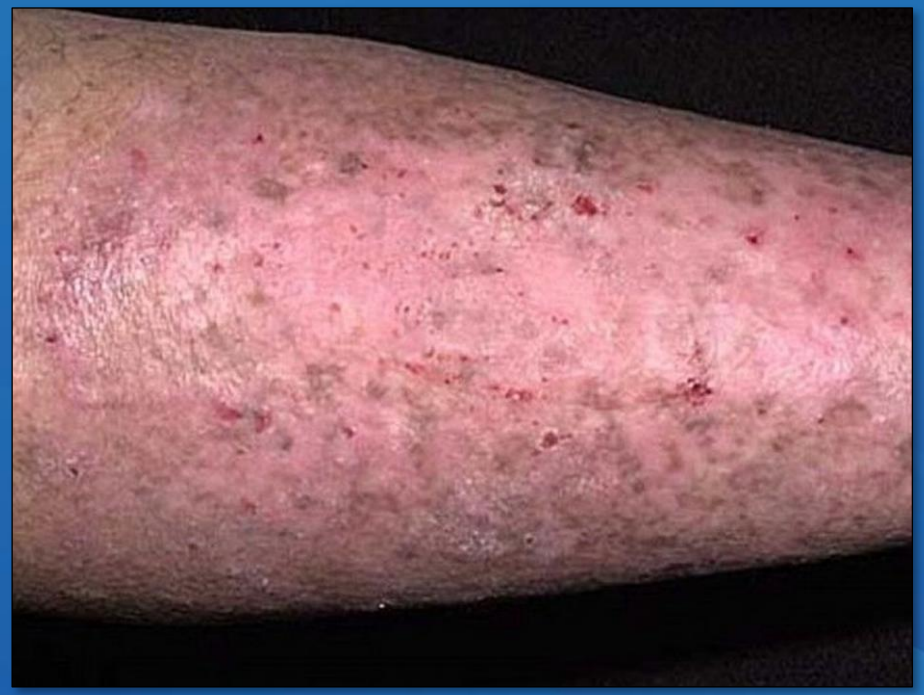




With permission from visualdx.com

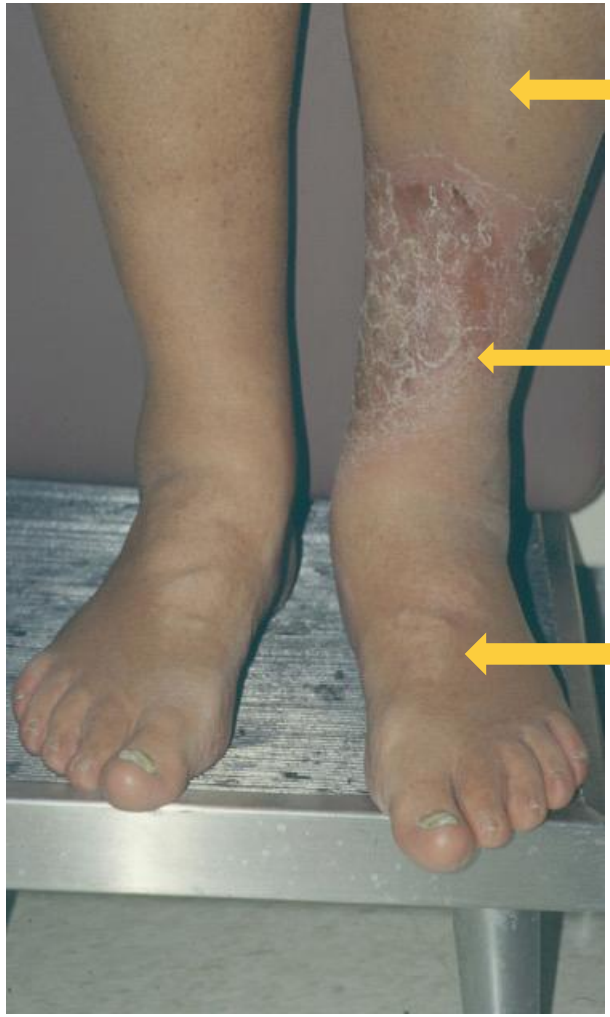
Lipodermatosclerosis

Skin induration, redness, and hyperpigmentation involving the lower third of the leg in a patient who has stasis dermatitis and lipodermatosclerosis.



Stasis Dermatitis and Venous Leg Ulcers

Clinical Appearance / Nomenclature

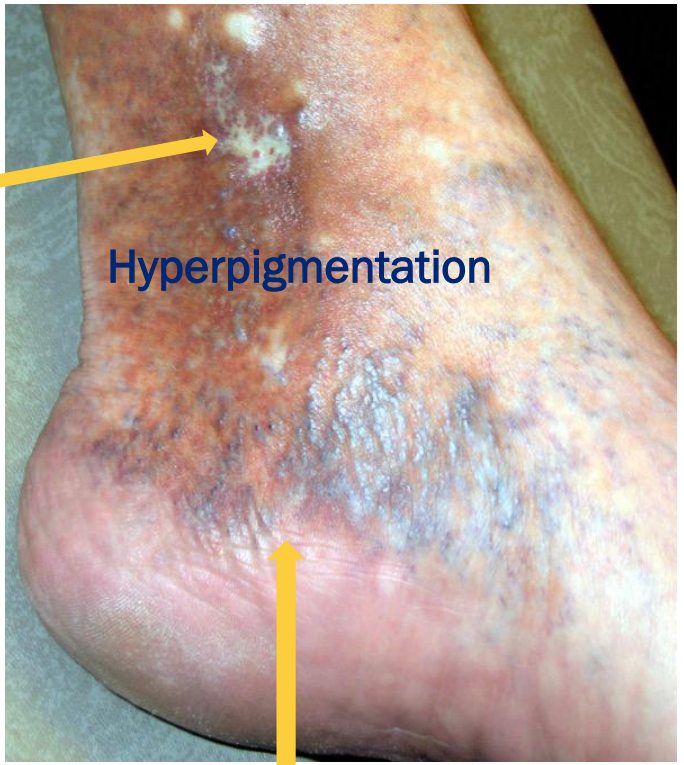


Swelling

Stasis dermatitis

Varicosities

Atrophie Blanche

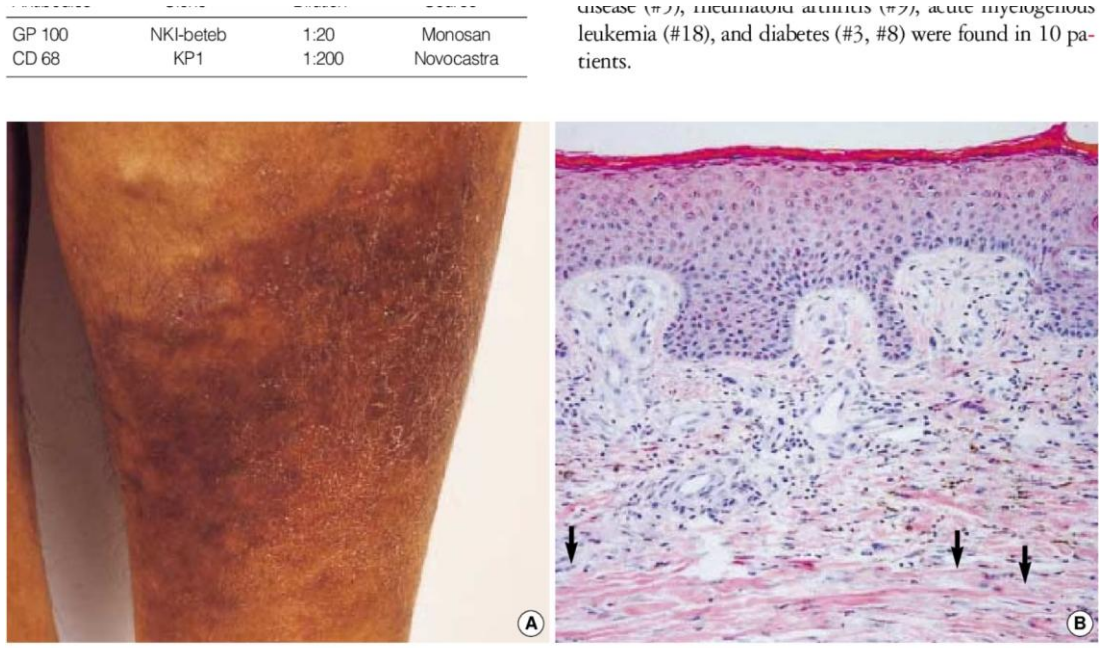


Ankle Flare/ Corona Phlebectatica

Understanding Stasis Dermatitis

Definition and Presentation

- Chronic inflammatory condition of lower extremities caused by venous insufficiency
- Predominantly affects middle-aged to elderly patients with venous disease
  - Erythema and scaling with intense pruritus
  - Hyperpigmentation from hemosiderin deposition
  - Progressive skin thickening (lipodermatosclerosis)



GP 100	NKI-beteb	1:20	Monosan
CD 68	KP1	1:200	Novocastra

leukemia (#18), and diabetes (#3, #8) were found in 10 patients.

Sundaresan S, et al. *Am J Clin Dermatol.* 2021;22(1):66-75.

Inflammatory Cascade in Stasis Dermatitis

Venous Hypertension

Valve incompetence leads to blood pooling and increased hydrostatic pressure

Capillary permeability increases under sustained pressure

Tissue Remodeling

Chronic inflammation leads to fibrosis and tissue hypoxia

Impaired tissue repair mechanisms perpetuate inflammation

Endothelial Activation

Sustained pressure triggers endothelial activation and dysfunction

Fibrinogen and other macromolecules leak into interstitium

Inflammatory Mediator Release

TNF-α, IL-1β, and IL-6 trigger neutrophil and macrophage infiltration

Matrix metalloproteinases degrade extracellular matrix components

Patel SK, et al. *J Vasc Surg Venous Lymphat Disord.* 2020;8(6):1128-1144.

Progression to Venous Ulceration

Sustained Inflammation

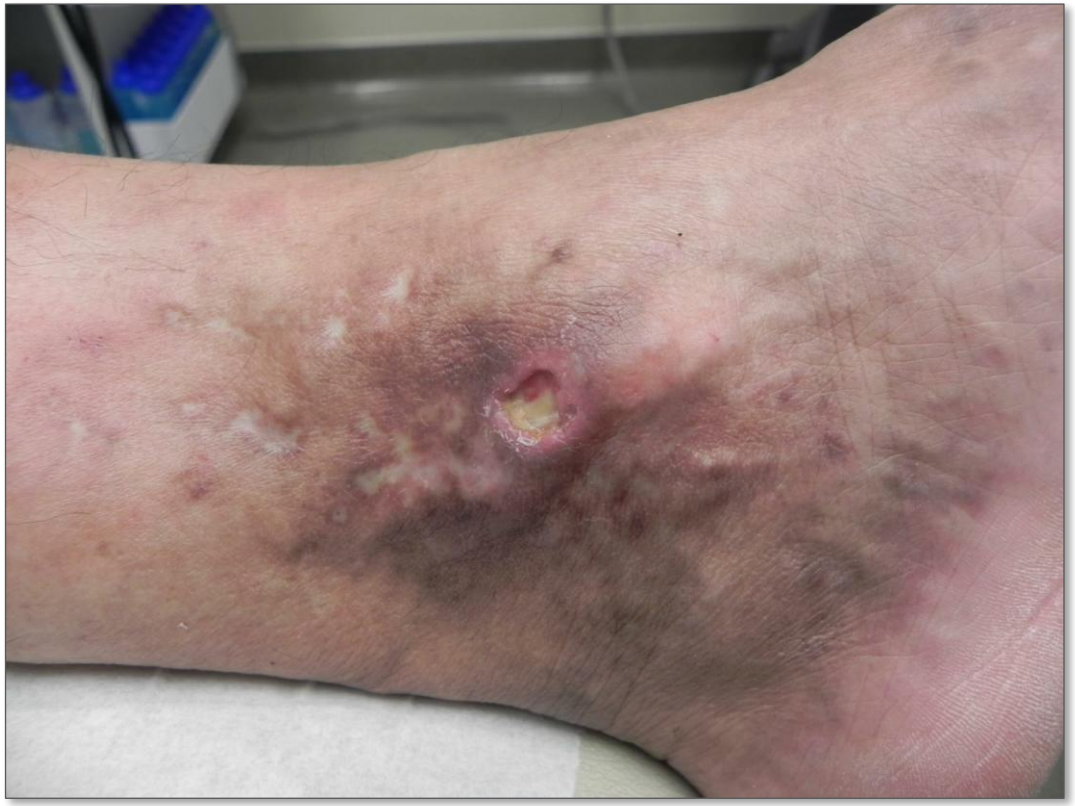
- Persistent neutrophil activation causes tissue damage through protease release
- Reactive oxygen species (ROS) impairs keratinocyte function and migration

Microenvironment Alteration

- Fibrin cuffs form around capillaries, impairing oxygen diffusion
- Cytokine dysregulation creates chronic inflammatory state

Ulcer Formation

- Minor trauma breaches weakened epidermal barrier
- Impaired healing mechanisms prevent resolution



Ulcers typically form at the medial malleolus where venous pressure is highest.

ROS = reactive oxygen species.

Raffetto JD. *J Vasc Surg.* 2018;67(2):564-572.

Management Strategies

1 Control Inflammation

Mid-potency topical steroids for acute flares (7-14 days)

Tacrolimus 0.1% for steroid-sparing maintenance therapy

2 Wound Care Optimization

Moist wound healing with appropriate dressings based on exudate.

Consider cellular and tissue-based products for refractory ulcers.

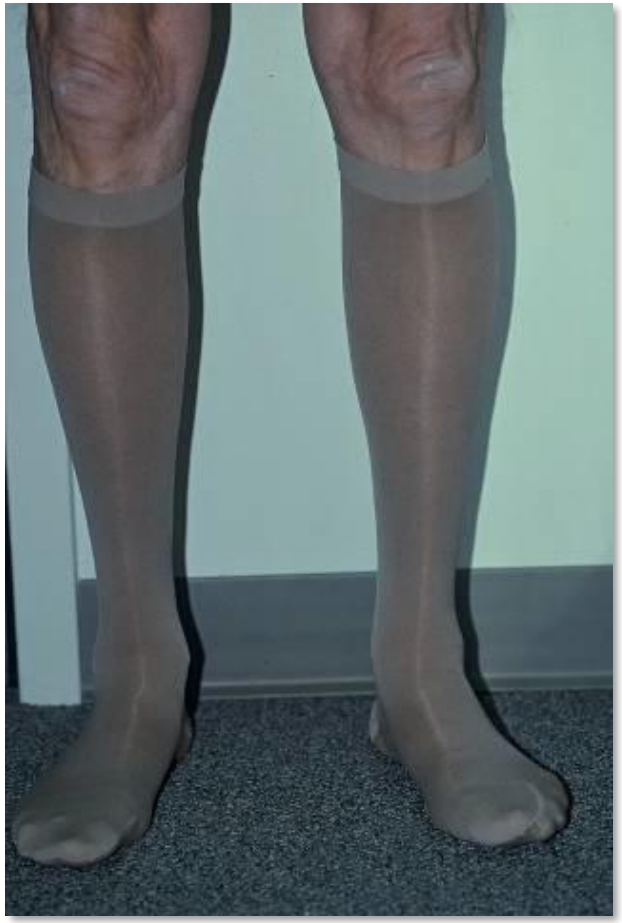
3 Address Venous Hypertension

Compression therapy (30-40 mm Hg) reduces edema and inflammation.

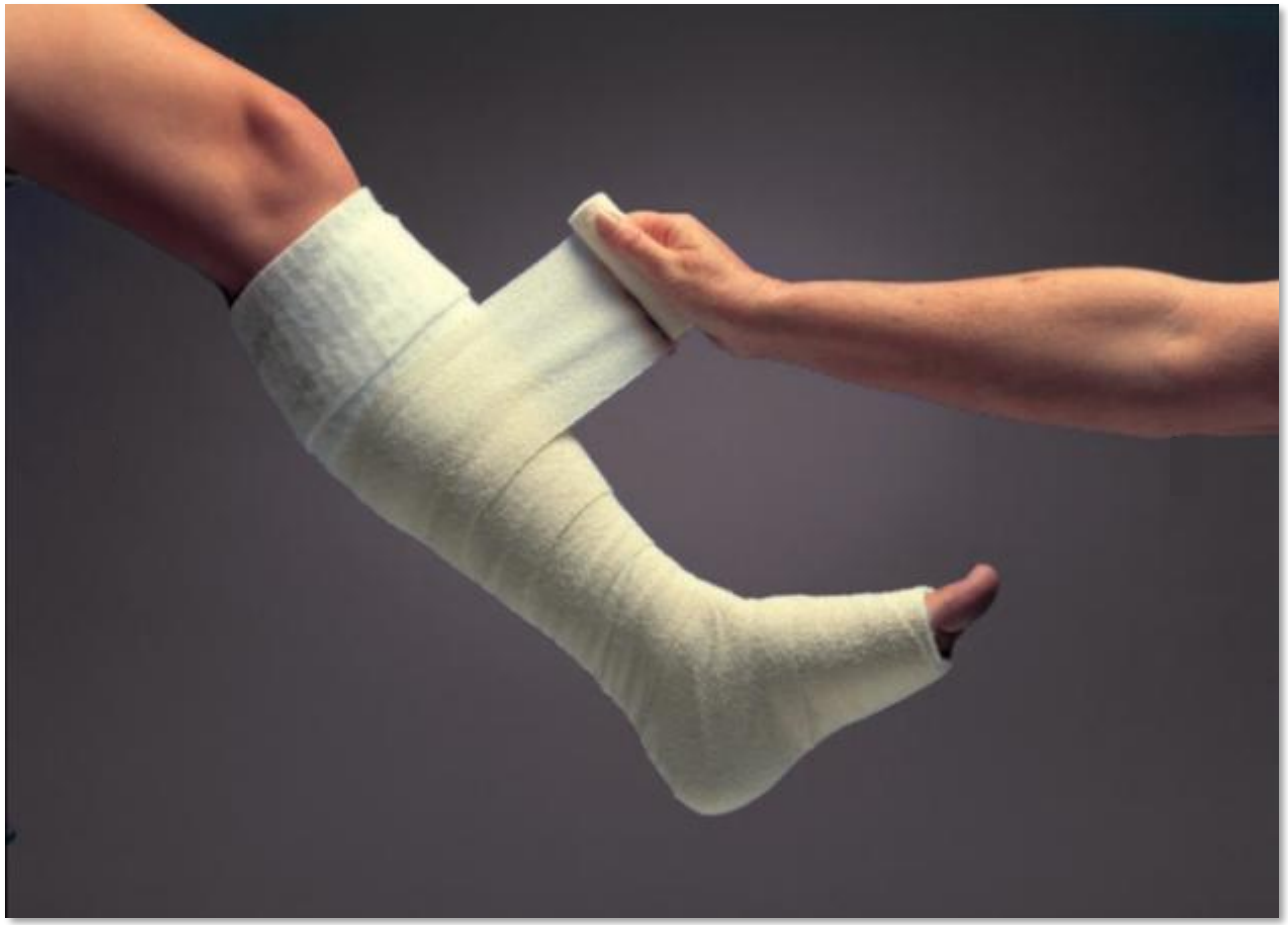
Leg elevation (30 minutes, 3-4 times daily) promotes venous return.

Alavi A, et al. *Int Wound J.* 2022;19(4):935-946. Marston W, et al. *Phlebology.* 2020;35(1):142-151. Kelechi TJ, et al. *Cochrane Database Syst Rev.* 2021;9:CD002303.

Compression Options

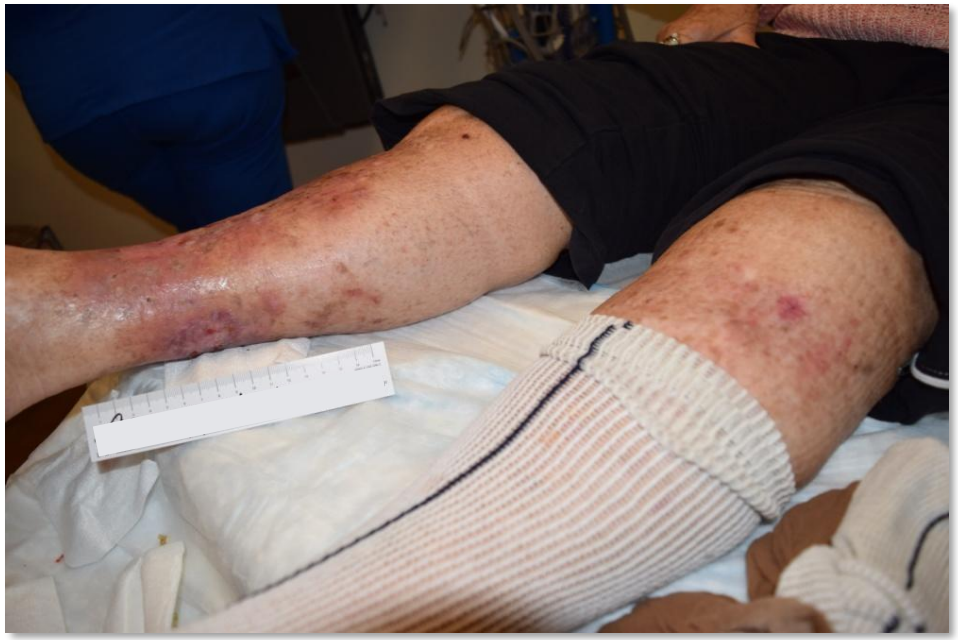


Prevention



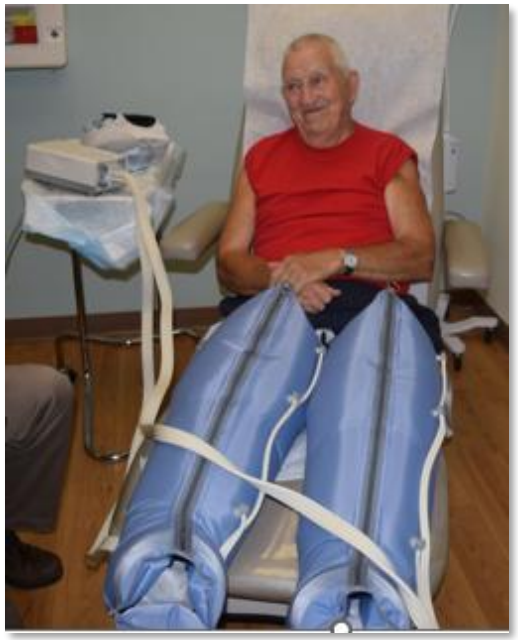
Treatment

Compression Options: Adjuvants



Textured Compression

Athletic Socks



Pumps

Compression Therapy Is the Gold Standard for Lymphedema and Leg Ulcers

- **Compression therapy**
  - Increases ulcer healing rates
  - Reduces venous pooling and venous reflux
  - Improves capillary filtration and lymphatic drainage
  - Improves arterial flow in patients with impairment
  - Decreases inflammatory mediators

O'Meara S, et al. *Cochrane Database Syst Rev*. 2012;11(11):CD000265.

The Objective Truth about Venous Leg Ulcers

- **Compression increases ulcer healing** rates compared with no compression
- **Multi-component systems are more effective** than single-component systems
- Multi-component **systems containing an elastic bandage appear to be more effective** than those composed mainly of inelastic constituents
- 2-component bandage systems appear to perform as well as 4LB
- Patients receiving the 4LB heal faster than those allocated the short stretch bandage (SSB)

SVS Society for Vascular Surgery

THE GOLD STANDARD

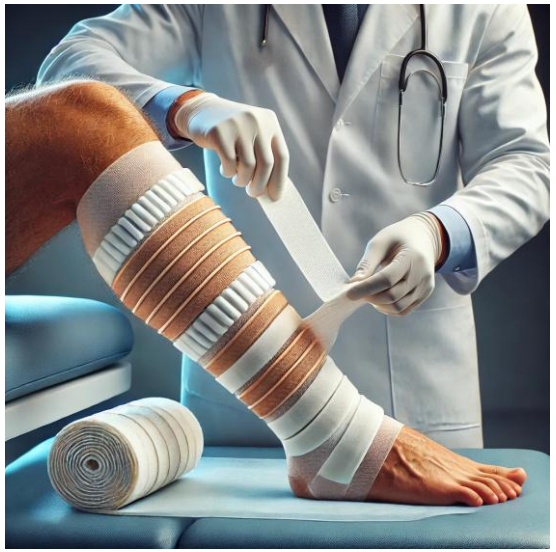
Cochrane

SSB = short stretch bandage.

O'Meara S, et al. *Cochrane Database Syst Rev*. 2012;11(11):CD000265.

Compression: The Ongoing Cornerstone of Treatment Ongoing Proof

- Gold standard: Multilayer compression bandaging
- Reduces ulcer recurrence by up to 50%
- Early compression reduces hospitalization and home care use
- Barriers to access: Cost, lack of insurance coverage, poor provider awareness



Nussbaum SR, et al. *Value Health*. 2018;21(1):27–32. Wounds UK. Best Practice Statement: Compression Hosiery. 3rd ed. Wounds UK; 2021. National Institute for Health and Care Excellence (NICE). Published July 2021. Accessed June 25, 2025. https://www.nice.org.uk/about/what-we-do/research-and-development/research-recommendations/ng199/1.

Why We Do an ABI Before Compression Is Started

The main concern with application of a compression system is perfusion impairment.

Plaque

Risk of ischemia

Plaque forms within the arteries and affects blood supply to the tissues

No Compression Revascularization

Both venous valves and arterial supply are impaired

Reduced Compression (~20 mm Hg*)

Venous valves are impaired, affecting blood return from ankles to the heart

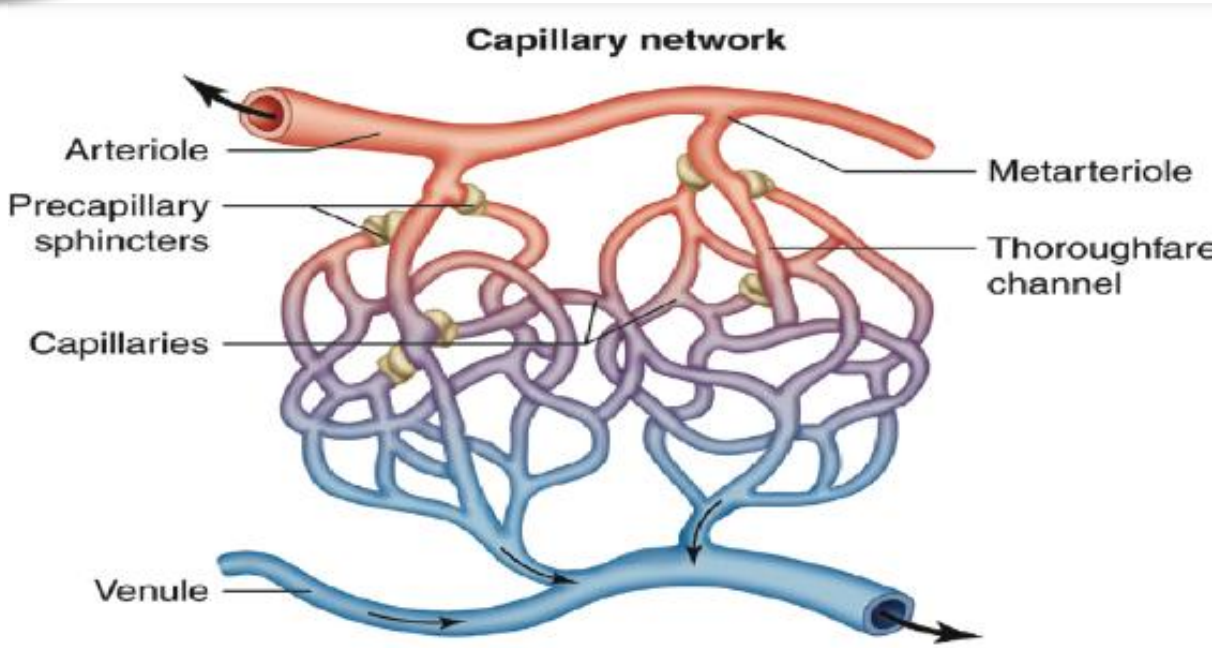
Strong Compression (~40 mm Hg*)

*at the ankle

The Difference Between Arterial and Venous Pressure

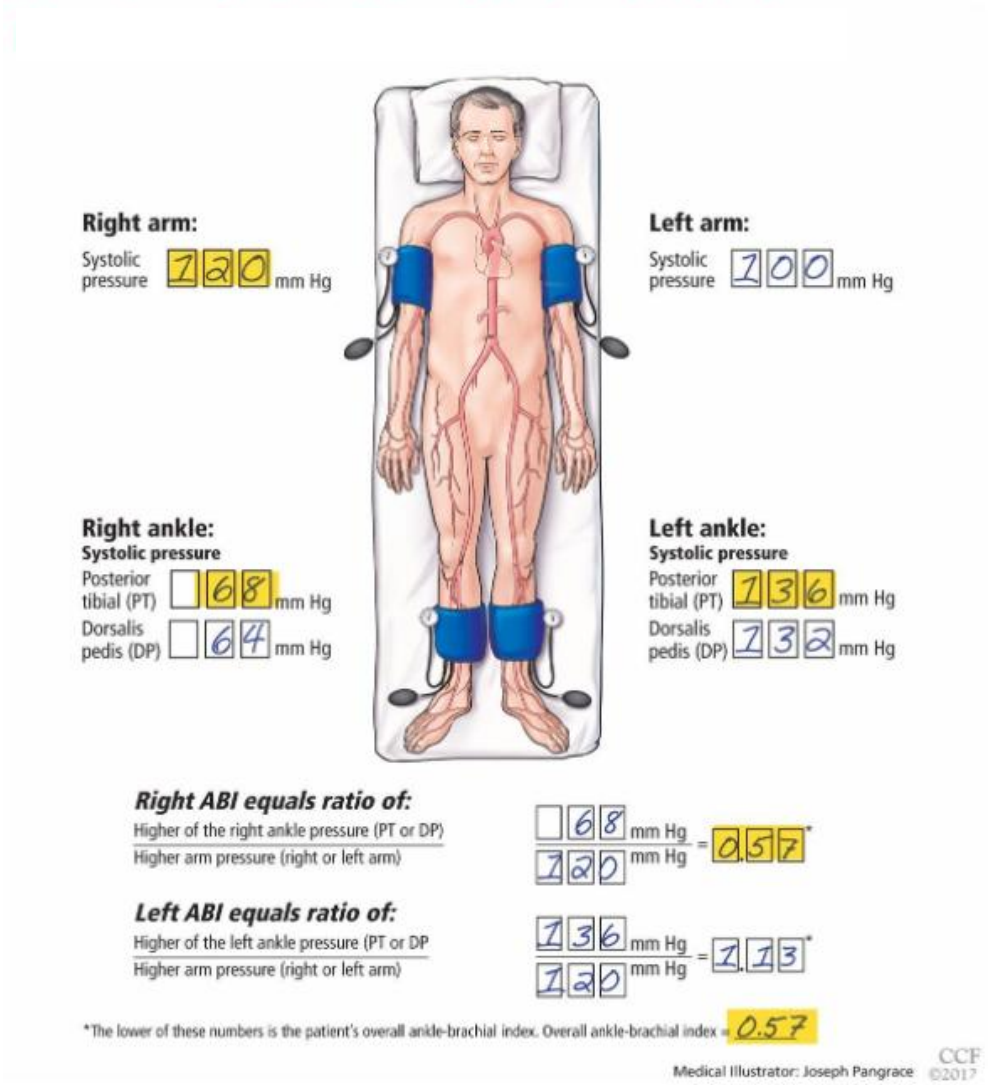
140 mm Hg

Compression
40-45 mm Hg



60 mm Hg

How to Calculate the Ankle-Brachial Index



Interpretation:

- 0.5 or less - severe arterial insufficiency
- 0.6 – 0.7 - arterial insufficiency
- 0.8 – 1.3 - normal
- 1.3 or higher - calcified vessels

Recommended Compression Based on ABI

Table 3 Arterial circulation and Ankle Brachial Pressure Index

Ankle brachial pressure index	Arterial circulation	Compression treatment
ABPI > 1.00–1.3[15,16]	Normal	Apply compression
ABPI = 0.8–1.0[15,16]	Mild peripheral Disease	Apply compression with caution[15]
ABPI ≤ 0.8–0.6[15,16]	Significant arterial disease	Use modified compression with caution[16] – refer to specialist[15]
ABPI < 0.5[15,16]	Critical ischaemia	Do not compress – refer urgently to vascular specialist
ABPI > 1.3	Refer to vascular/diabetic specialist	

ABPI, Ankle Brachial Pressure Index.

Source: Andriessen A, et al. *J Eur Acad Dermatol Venereol*. 2017;31(9):1562-1568.

Veno-Lymphatic Connection and Importance of Compression

Heather Barnhart, PT, PhD, CWS, CLT-LANA, CLWT
Director of Clinical Operations, Koya Medical
St. Augustine, FL

Veno-Lymphatic Connection

- During embryonic development, a subset of blood endothelial cells (BECs) in the cardinal vein become lymphatic endothelial cells (LECs), which form initial lymph structures called lymph sacs
- These sacs give rise to the entire lymphatic network (more than just a vessel system)

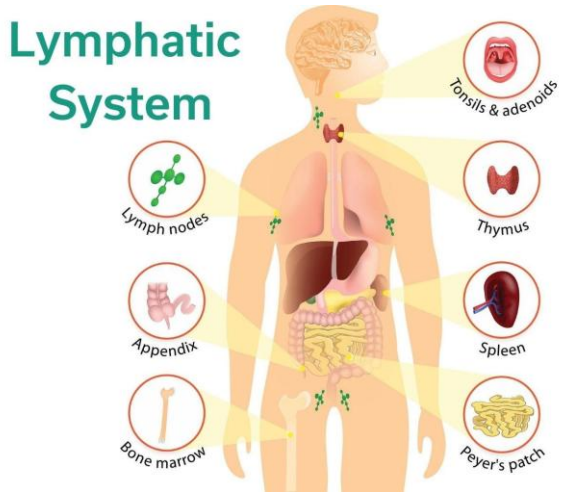
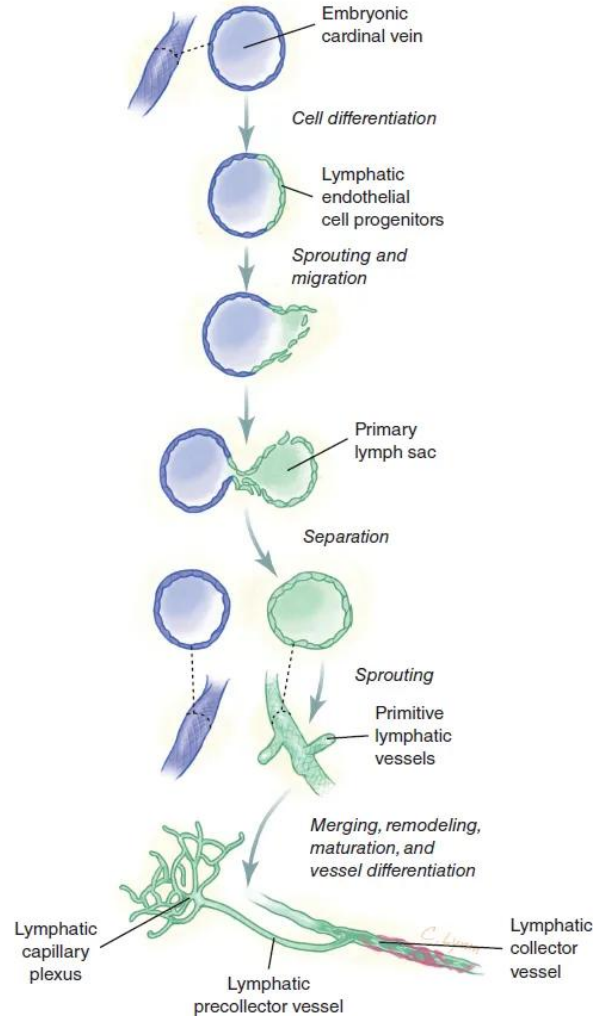


Image: Integris Health



https://plasticsurgerykey.com/chapter-4-embryology/

BEC = blood endothelial cell; LEC = lymphatic endothelial cell.
Yang Y, et al. *Blood*. 2012;120(11):2340-2348.

Venous System and Lymphatic System Overview

Veins: 3 different vessels

Deep- below the fascia

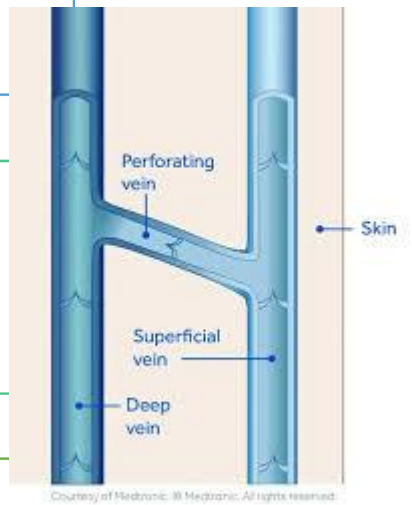
- Popliteal vein most common site to develop a blood clot
- Deep system handles up to 90% of the venous blood volume

Superficial- above the fascia

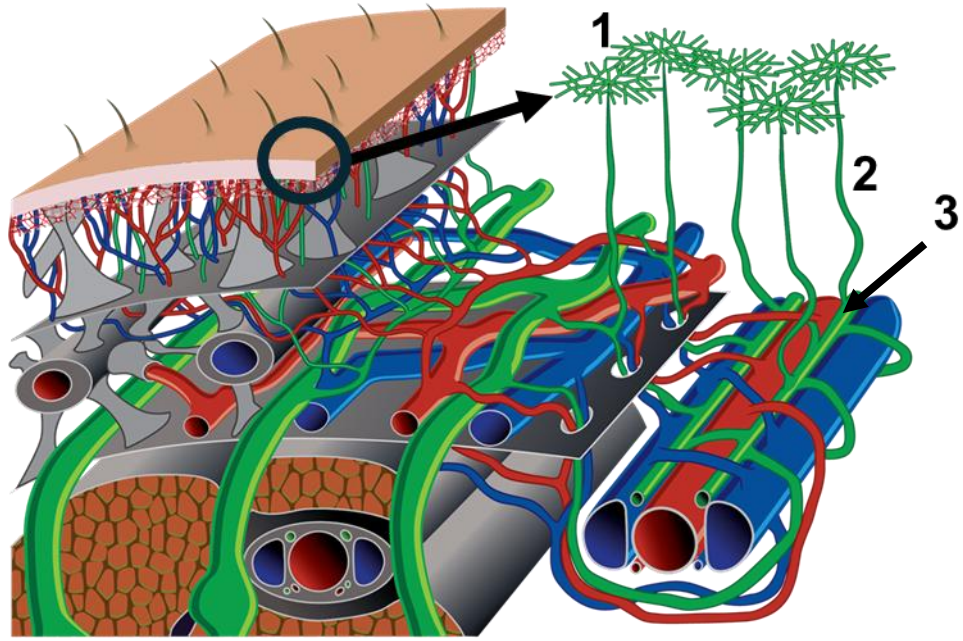
- Handles approximately 10% of the total venous blood volume and is the low-pressure system within the venous circulation

Perforating- perforates the fascia

- Connect superficial and deep veins



Lymphatics: 3 different vessels, lymph nodes, organs

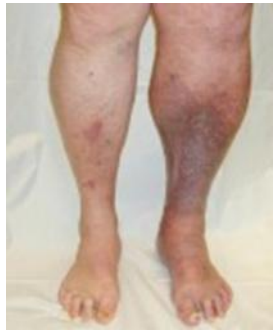


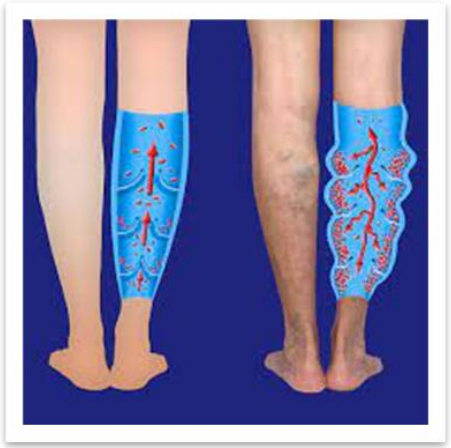
1. Superficial lymphatic capillaries (dermis)
2. Precollectors
3. Deep lymphatic collector vessels (valved, fascial plane)

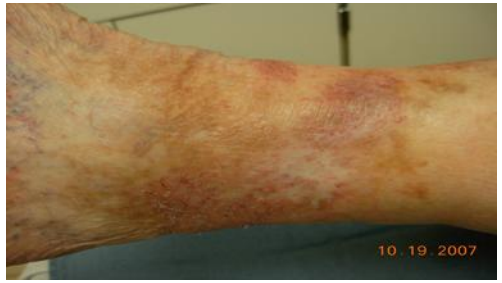
Cleveland Clinic. Accessed June 25, 2025. https://my.clevelandclinic.org/health/body/23360-veins

Pathophysiology of Venous Disease = CVI

- The proper outflow of venous blood from the lower extremities depends on vein patency, valve competency, and proper calf muscle function
- Chronic venous disease (CVD) is the result of continuous venous hypertension caused by venous reflux, venous obstruction, and/or a poorly functioning calf muscle pump
- Any disturbance in these 3 components can result in venous hypertension leading to chronic venous insufficiency (CVI)







CVD = chronic venous disease; CVI = chronic venous insufficiency.

Araki C, et al. *J Vasc Surg*. 1994;20:872-879. Simka M. *Phlebolymphology* 2004;47:299-303. Lurie F, et al. *Phlebology*. 2022;37(4):252-662. Torgbenu E, et al. *Adv Wound Care* (New Rochelle) 2023;12(1):15-27.

Pathophysiology of Venous Disease = CVI

Valvular reflux, obstruction, +/or calf muscle pump failure

↓

Retrograde venous blood flow (reflux)

↓

Venous hypertension (elevated ambulatory pressure)

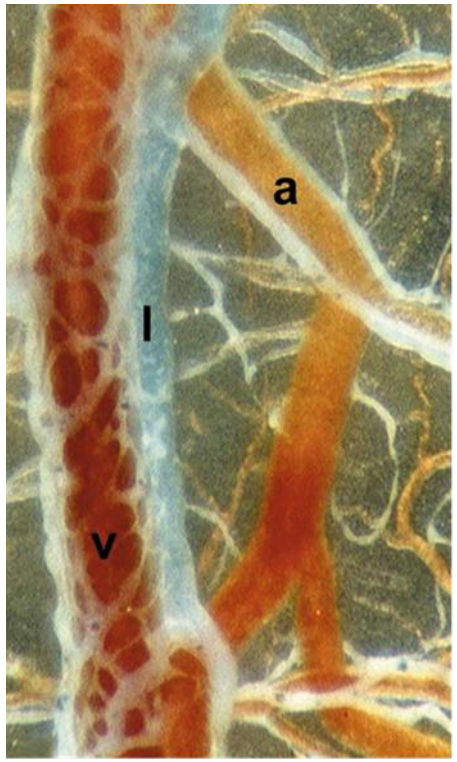
↓

Wide spectrum of venous disease

(telangiectasia, dilated reticular veins, varicose veins, edema/lymphedema, skin changes, venous ulcers, chronic pain, inflammation, altered lifestyle, consequences to quality of life)

Shiman MI, et al. *Wound Rep Reg*. 2009;17(2):147-152. Meissner MH, et al. *J Vasc Surg*. 2007;46(Suppl S):4S-24S.

Veno-Lymphatic Connection



a- artery
v-vein
l- lymph vessel

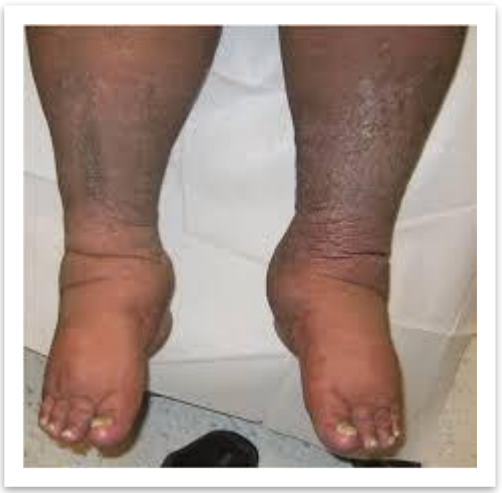
Image from
http://jeltsch.org/static/publications/jeltsch03/index.html

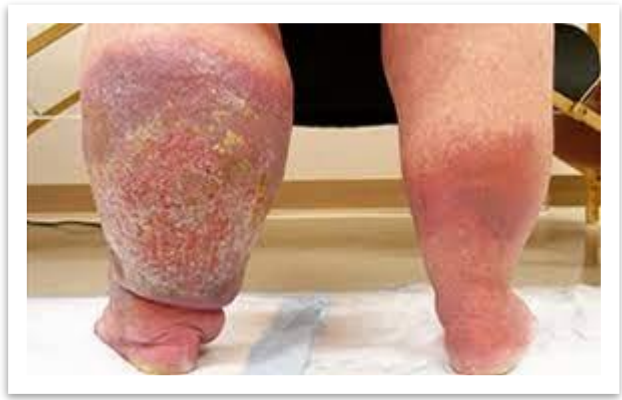
- Venous disease is associated with microangiopathic changes in the vascular and lymphatic networks
- The disruption or loss of lymphatic function results in the development of edema, commonly seen in CVI

Mortimer P, Pearson I. *Phlebolymphology*. 2004;44:253-257.

Phlebolymphedema

- The leading cause of secondary lymphedema in the U.S. is phlebolymphedema, or lymphedema of venous etiology (LOVE)
- Dual system impairment that can lead to multisystem impairment (think VAIL)
  - VAIL = Veins, arteries, integument, and lymphatics are interconnected

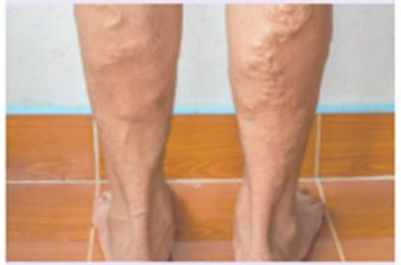
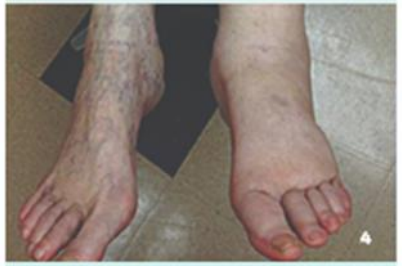
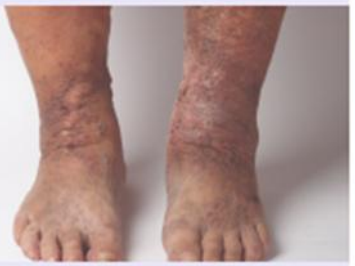
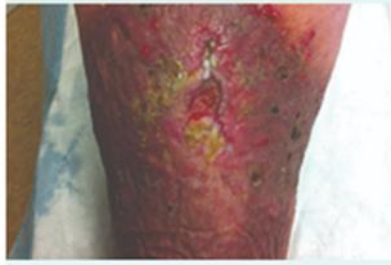
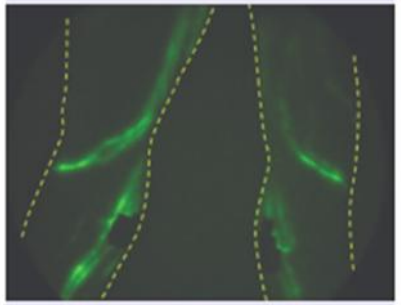
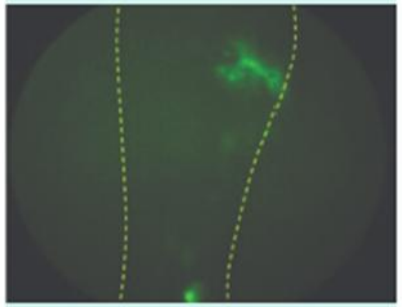
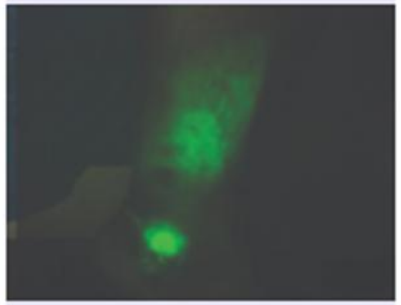
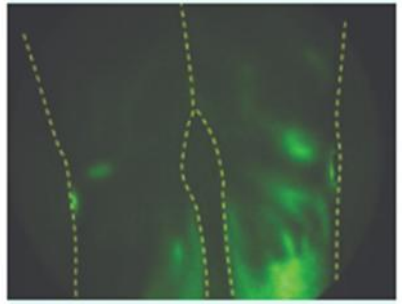




Dean SM, et al. *J Vasc Surg Venous Lymphat Disord.* 2020;8(5):851-859.

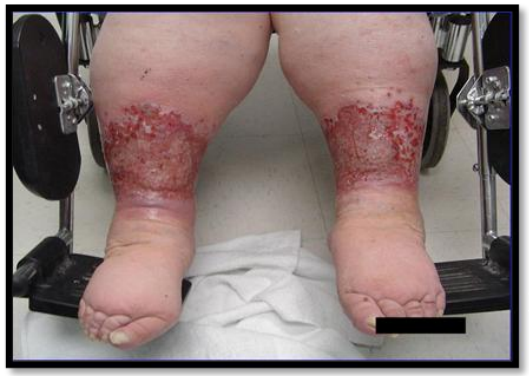
Phlebolymphedema and the Veno-Lymphatic Connection

Prevents irreversible progression by starting treatment in the early stages.

Chronic Venous Disease Staging	C1, C2	C3	C4	C5, C6
				
	C0: No Clinical Signs **C1:** Telangiectasias or Reticular Veins **C2:** Varicose Veins	**C3:** Edema (pitting) Edema (non-pitting)	**C4a:** Pigmentation or Eczema **C4b:** Lipodermatosclerosis or Atrophie Blanche **C4c:** Corona Phlebectatica	**C5:** Healed Venous Ulcer **C6:** Active Venous Ulcer
Near-Infrared Images Show Lymphatic Impairment				
	C0: In early stages of venous disease, the lymphatic capacity is not overloaded and can manage the venous filtrate	**C3:** Swelling develops when the microvascular (capillary and venular) filtration rate exceeds lymph drainage for a sufficient period	**C4:** Altered immune control paves the way for exaggerated immune reactions including Granulomatous reactions and Dysimmune reactions	**C6:** Once formed, an ulcer will not heal (C5) unless venous hypertension and the resulting excess capillary fluid filtration are ameliorated
Lymphedema Key Clinical Characteristics	**Latent** No clinical signs	**Pitting Edema** Changes in solid structures, pitting is manifest **Non-pitting Edema** Excess subcutaneous fat and fibrosis develop	If not treated: **Lymphostatic Elephantiasis** Trophic skin changes such as Acanthosis, alterations in skin character and thickness, further deposition of fat and fibrosis, and warty overgrowths have developed	

Lurie F, et al. *J Vasc Surg Venous Lymphat Disord*. 2020;8(3):342-352. Rasmussen JC, et al. *J Vasc Surg Venous Lymphat Disord*. 2016;6(1):9-17. Executive Committee of the International Society of Lymphology. *Lymphology*. 2020;53(1):8-19. Kamel MK, et al. *Phlebolymphology*. 2020;27(1):1-44. Mortimer PS, et al. *J Clin Invest*. 2014;124(3):915-921. Ruocco E, et al. *Clin Dermatol*. 2012;30(5):541-548. Bornatchez SF, et al. *Adv Wound Care (New Rochelle).* 2022;11(1):28-41. Images reprinted with permission from Elsevier.

Veno-Lymphatic Connection and VAIL

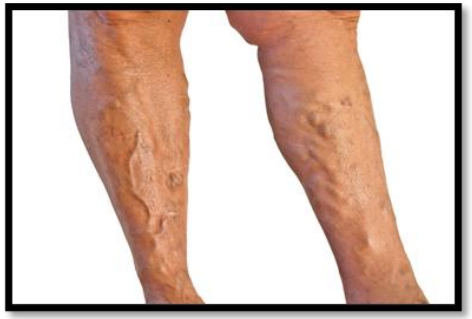


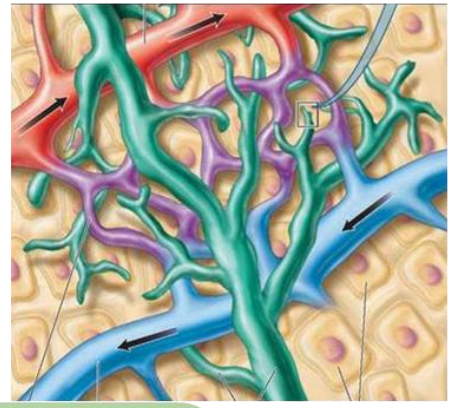
Lymphostatic dermopathy is underlying pathology contributing to formation of venous ulcers

Tri-system failure

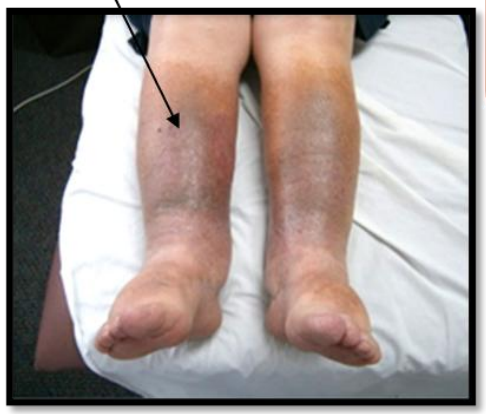
Chronic venous insufficiency

High filtration pressure/increased fluid in tissues





Hemosiderin
Impairs lymph pumping



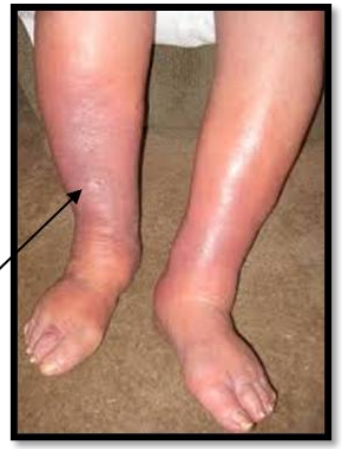
Lymphatic damage = impairment

Lymphatic hypertension leads to fibrosclerosis (stagnation/chronic inflammation)

Low protein edema

Waterload exceeds lymphatic transport capacity = lymphatic insufficiency

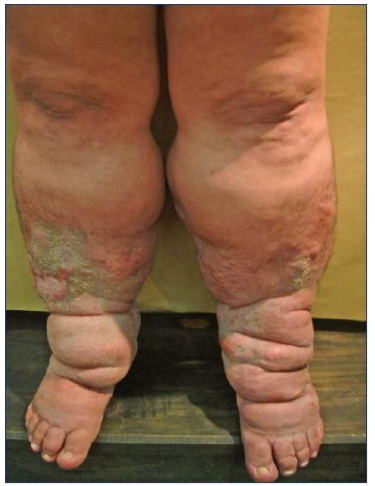
Hemosiderin impairs lymph pumping



Wandolo G, et al. *J Vasc Res*. 1992;29(3):248-255. Slide used with permission from International Lymphedema and Wound Training Institute (ILWTI).

Lymphostatic Dermopathy: Interconnected Systems

- Decongesting and maintaining interstitial fluid mitigates and prevents the deleterious effects of stagnant fluid on tissues and skin
- ***Chronic lymph stasis (lymphostasis) has numerous consequences***, including inflammation, lipogenesis, fibrosis, and immunosuppression
- Lymphostasis leads to accumulation of antigens, foreign material, and immune complexes in the interstitial space, which leads to ***chronic localized inflammation***
- This can ***manifest on the skin*** as disruption of immune cell trafficking leads to localized immune suppression, predisposing the area affected to chronic inflammation, connective tissue proliferation, infection, and malignancy
- In essence, it ***disrupts the skin's barrier function***



Carlson JA. *Clin Dermatol*. 2014;32(5):599-615. Finegold DN, et al. *Lymphat Res Biol*. 2008;6(2):65-68.

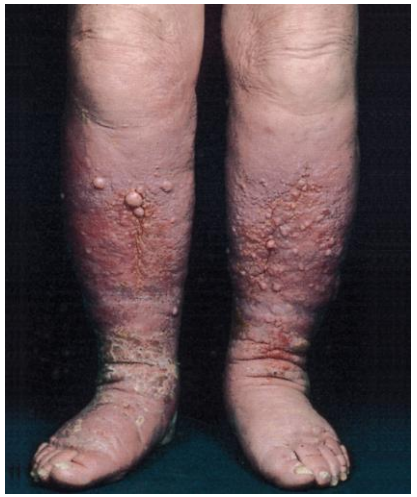
Lymphostatic Dermopathy: Interconnected Systems

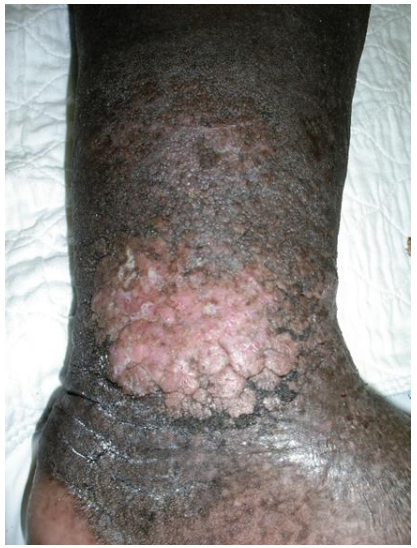
- The venous and lymphatic systems are mutually interdependent; when dysfunctional, the result is a ***dual outflow system failure*** → Tri-system failure with integumentary impairments (VLUs, dermatitis, etc.)
- ***Disorders of the lymph system***, whether systemic (macro-lymphedema) or localized (micro-lymphedema), ***produce cutaneous regions susceptible to infection, inflammation, and carcinogenesis*** (lymphostatic dermopathy = skin barrier failure; tri-system failure)
- The ***pathophysiology of lymphedema explains the propensity for infections*** (cellulitis) and ***hypersensitivity*** reactions in ***patients with chronic venous insufficiency*** (lymphostatic dermopathy)
- A functional lymphatic system is essential to an organism's overall health given its role in fluid homeostasis, removal of cellular debris and mediating immunity and inflammation
- ***Lymph stasis (lymphostasis) is problematic → chronic inflammatory state***

Carlson JA. *Clin Dermatol*. 2014;32(5):599-615. Finegold DN, et al. *Lymphat Res Biol*. 2008;6(2):65-68.

Lymphostatic Dermopathy: Interconnected Systems

- Clinically, fibrosis, papillomatosis, stasis dermatitis, and classic venous and lymphatic skin changes are seen
- ***Chronic inflammation*** also attenuates (reduces) lymphatic contraction, ***hindering lymphatic flow*** to the lymph node
- Hemosiderin, classic with CVI, also suppresses lymphatic contractility
- To trigger an effective immune response, antigen and antigen-presenting cells must travel to the lymph node; thus, lymphostasis disrupts cell-mediated (adaptive) immunity by decreasing or obstructing immune trafficking by antigen, lymphocytes, macrophages, and dendritic/antigen-presenting cells to the lymph node, creating a ***cutaneous region of immunosuppression***
- All these abnormalities lead to a condition termed lymphostatic dermopathy, which is the ***failure of the skin as an immune organ***





Carlson JA. *Clin Dermatol*. 2014;32(5):599-615. Finegold DN, et al. *Lymphat Res Biol*. 2008;6(2):65-68. Wandolo G, et al. *J Vasc Res*. 1992;29(3):248-255.

Lymphostatic Dermopathy: Interconnected Systems

- Lymphostatic dermopathy in part explains the propensity of skin infections and VLUs in people with CVI
- Therefore, it is imperative to reduce and manage the fluid burden to optimize venous and lymphatic function while simultaneously facilitating wound resolution and tissue remodeling
  - Reducing the fluid burden and diffusion distance also supports better perfusion by optimizing arterial inflow
  - Compression over time restores the skin barrier function and reverses the effects of lymphostatic dermopathy
  - Compression is cornerstone for all forms of edema and essential for venous/lymphatic impairment (phlebolymphedema)

Carlson JA. *Clin Dermatol*. 2014;32(5):599-615. Finegold DN, et al. *Lymphat Res Biol*. 2008;6(2):65-68. Wandolo G, et al. *J Vasc Res*. 1992;29(3):248-255

Compression Is Cornerstone

- It has been well established that externally applied compression aids in providing support to the venous system by approximating the valves to reduce venous backflow and promote blood flow toward the heart
- Additionally, compression provides the necessary containment of the interstitial fluid to prevent and reduce edema formation resulting from an overburdened superficial lymphatic system
- **When compression is properly applied, it has been shown to reduce edema, normalize venous function, optimize healing of VLUs, improve lymphatic function, enhance arterial pulse width, diminish episodes of recurrent cellulitis, promote resolution of trophic changes, decrease the presence of inflammatory mediators and symptoms, and reduce pain**

Stout N, et al. *Int Angiol*. 2012;31(4):316-329. Gianesini S, et al. *Phlebology*. 2019;34(1 Suppl):4-66. Shi C, et al. *Cochrane Database Syst Rev*. 2021;7(7):Cd013397. Dissemond J, et al. *J Dtsch Dermatol Ges*. 2016;14(11):1072-1087. Kitayama S, et al. *Lymphat Res Biol*. 2017;15(1):77-86. Mukherjee A, et al. *J Physiol*. 2021;599(10):2699-2721. Negrini D, Moriondo A. *J Physiol*. 2011;589(Pt 12):2927-2934. Schmid-Schönbein GW. *Lymphat Res Biol*. 2003;1(1):25-29. McGeown JG, et al. *J Physiol*. 1987;387:83-93. Mosti G, Cavezzi A. *Phlebology*. 2019;34(8):515-522. Mosti G. *Phlebology*. 2014;29(1 suppl):13-17. Mayrovitz HN, Macdonald JM. *Int Angiol*. 2010;29(5):436-441. Mayrovitz HN, Larsen PB. *Clin Physiol*. 1997;17(1):105-117. O'Meara S, et al. *Cochrane Database Syst Rev*. 2012;11(11):CD000265. Partsch H. *J Wound Care*. 2019;28(7):427. White-Chu EF, Conner-Kerr TA. *J Multidiscip Healthc*. 2014;7:111-117. Beidler SK, et al. *J Vasc Surg*. 2009;49(4):1013-1020. Beidler SK, et al. *Wound Repair Regen*. 2008;16(5):642-648. Bojesen S, et al. *Eur J Dermatol*. 2019;29(4):396-400. Travis TE, et al. *Eplasty*. 2018;18:e1. Webb E, et al. *N Engl J Med*. 2020;383(7):630-639. Ebell MH. *Am Fam Physician*. 2021;103(4):247. Lerman M, et al. *J Vasc Surg*. 2019;69(2):571-580. Rabe E, et al. *Phlebology*. 2020;35(7):447-460. Kerr A, et al. *Wounds International*. 2020;10:4-32.

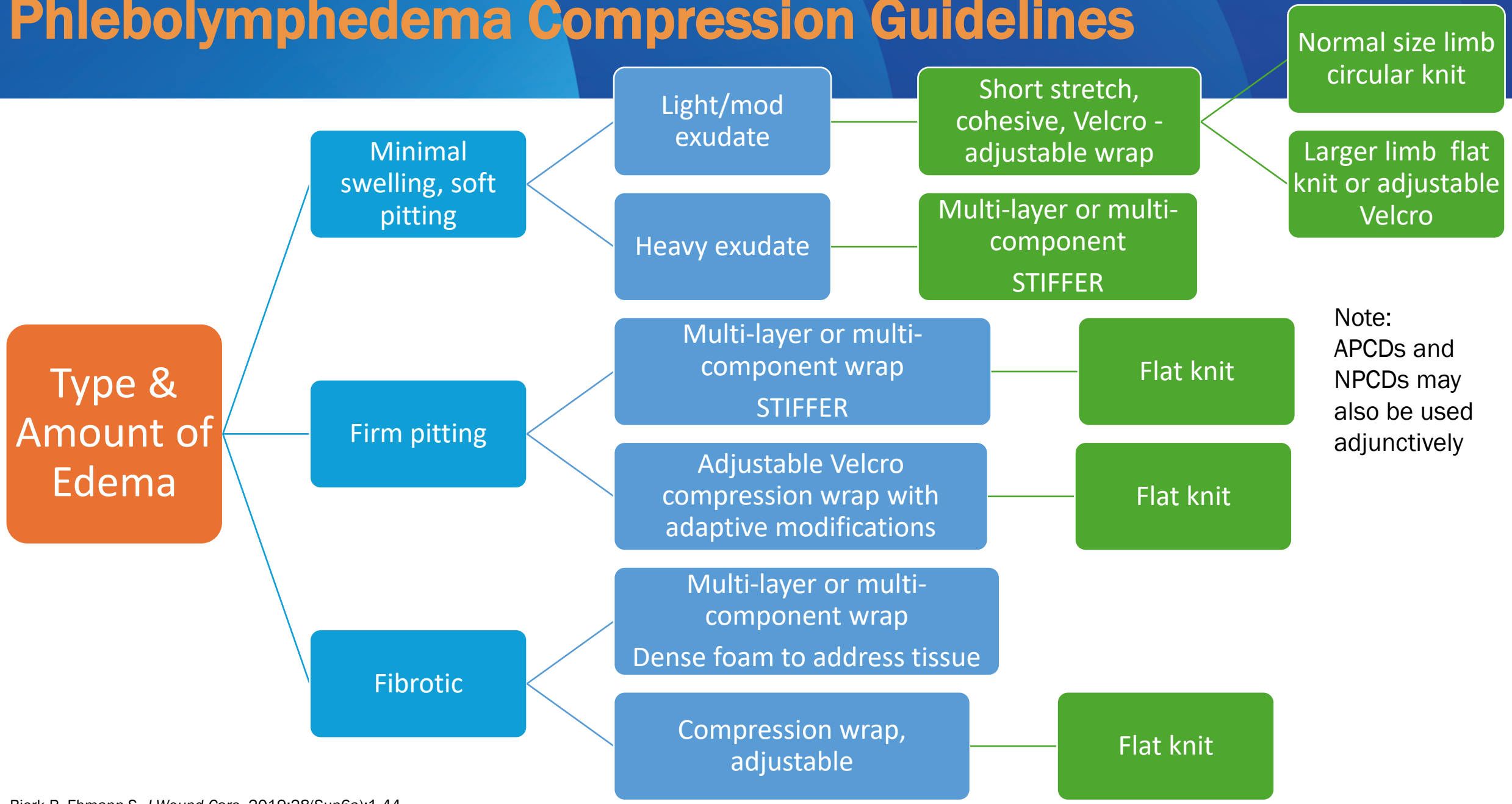
Compression Evidence

- For phlebolymphedema, compression therapy is essential; however, no single consensus exists. Many guidelines recommend wraps and garments in conjunction with other therapies (ie, manual lymphatic drainage, exercise)
  - Compression devices (NPCDs) and pumps (APCDs) are useful adjuncts
- Multilayer and multicomponent bandages are recommended during active reduction of edema and in the presence of skin impairment
- Flat knit garments are recommended over circular knit for patients with more severe swelling and skin changes
- For patients with donning/doffing difficulties or atypical limb geometry, Velcro® adjustable wraps are recommended

NPCD = non-pneumatic compression device; APCD = advanced pneumatic compression device.

Farrow W. *J Am Coll Certif Wound Spec*. 2010;2(1):14-23. Fife CE. Wound Care Practice. 2nd ed. Best; 2007. Position statement of the National Lymphedema Network. Published September 30, 2009. Accessed June 25, 2025. http://www.lymphnet.org/pdfDocs/nlntreatment.pdf.

Phlebolymphedema Compression Guidelines



Note:
APCDs and NPCDs may also be used adjunctively

Bjork R, Ehmann S. *J Wound Care*. 2019;28(Sup6a):1-44.

Before Compression, Consider...

PAD**

- ABI <0.5 or >1.3
  NO compression
  Vascular workup first
- Systolic ankle pressure >50-60 mm Hg,
  toe pressure >30 mm Hg
- Inelastic compression not to exceed 40 mm Hg in supine position and should be changed q1-2 days

Mixed (AI & VI)

- ABI ≥0.8
  Standard compression
- Ankle systolic pressure ≥60 mm Hg, ABI ≥0.5
  modified compression
  **Close monitoring

Immobility

- Short stretch compression system
  (cohesive, multi-component wrap, velcro adjustable wraps, etc)
- NPCD with ankle ROM
  APCD

Proximal Swelling

- Wrap full extremity
- NPCD
  APCD

**use ankle perfusion pressure of 60 mm Hg or greater rather than ABI ≤ \0.5 as cutoff for compression bandages/garments b/c correlate better with tissue perfusion

O'Donnell TF, et al. *J Vasc Surg*. 2014;60(2 Suppl):3S-59S.

How to match Compression to the patient

What will the patient use?

Does it match their functional abilities?

Does it match their lifestyle?

Compression is only effective if it is used!

Know medical history, co-morbid conditions

Do not need overt edema to use compression

Perfusion status

Skin integrity

Limb shape/size

Function (ROM, strength, gait, hand dexterity to don/doff)

Wrap right, not tight

Clinical Pearls

- Veins and lymphatics have similar embryological origin and anatomical structure
- Venous disease is associated with microangiopathic changes in vascular and lymphatic networks
- Disruption or loss of lymphatic function results in development of edema, commonly seen in CVI
- Phlebolymphedema can be due to insufficiency of venous or lymphatic system or both
- Most cases of phlebolymphedema are due to an overload of the venous system that overwhelms the lymphatic system's ability to remove interstitial fluid
- Phlebolymphedema is a leading cause of secondary lymphedema in the U.S.
- A CEAP score of C3 or higher indicates phlebolymphedema, though lymphatic damage exists in C1-2
- Compression is the cornerstone therapy for CVI/edema and is more effective when combined with a walking program

Blood Flow Linkages to Compression Bandaging

Harvey N. Mayrovitz, PhD
Nova Southeastern University,
Dr. Kiran C. Patel College of Allopathic Medicine
Department of Medical Education
Ft. Lauderdale, FL

Three Principles for Good Compression: Continuous, Consistent, Comfortable

The 3 Cs of good compression and the results with DCS

- Continuous pressure, 24/7
  - Interface pressure remained the same for DCS up to 7 days and at different levels of activity
- Consistent pressure and application
  - 87% of nurses achieved therapeutic pressure at first application
- Comfortable compression that patients will wear
  - 95.7% patient compliance in winter months
  - 92.2% patient compliance in summer months

Hanna R, et al. *Br J Nurs*. 2008;17(20):S16-S24. Jünger M, et al. *J Wound Care.* 2009;18(11):474, 476-480. Pilati L. Do the findings in winter of a dual compression system compression bandage's wear compliance with patients replicate if the study is repeated in the summer months? Presented at Symposium on Advanced Wound Care Fall 2021.

Is There a Fourth “C”

Cutis

Compression's Relationship to Wound Healing

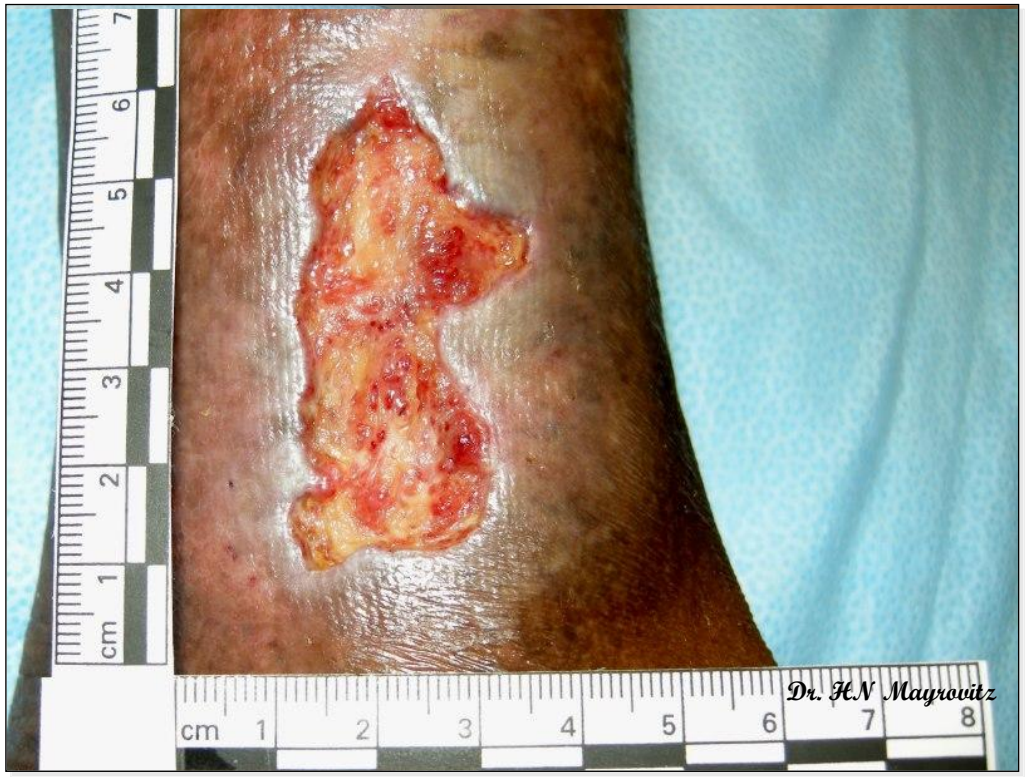
Healing Impediments
- **Blood Flow**
- Oxygenation
- Infection
- Tissue Environment
  - Edema
  - Lymphedema

Therapy Targets
- **Arterial**
- Venous
- **Microvascular**
- Lymphatic
- Interstitial

Compression Therapy

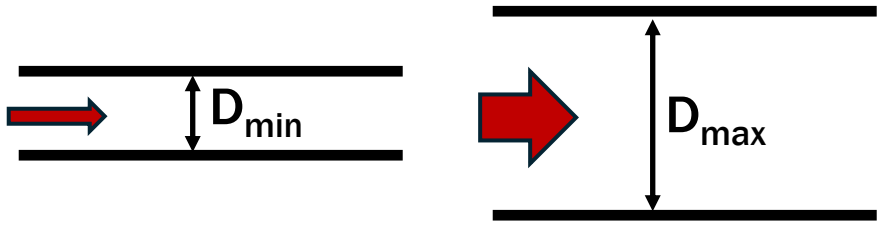
Part 1. Microvascular → Flowmotion

Part 2. Arterial Pulsatile Blood Flow

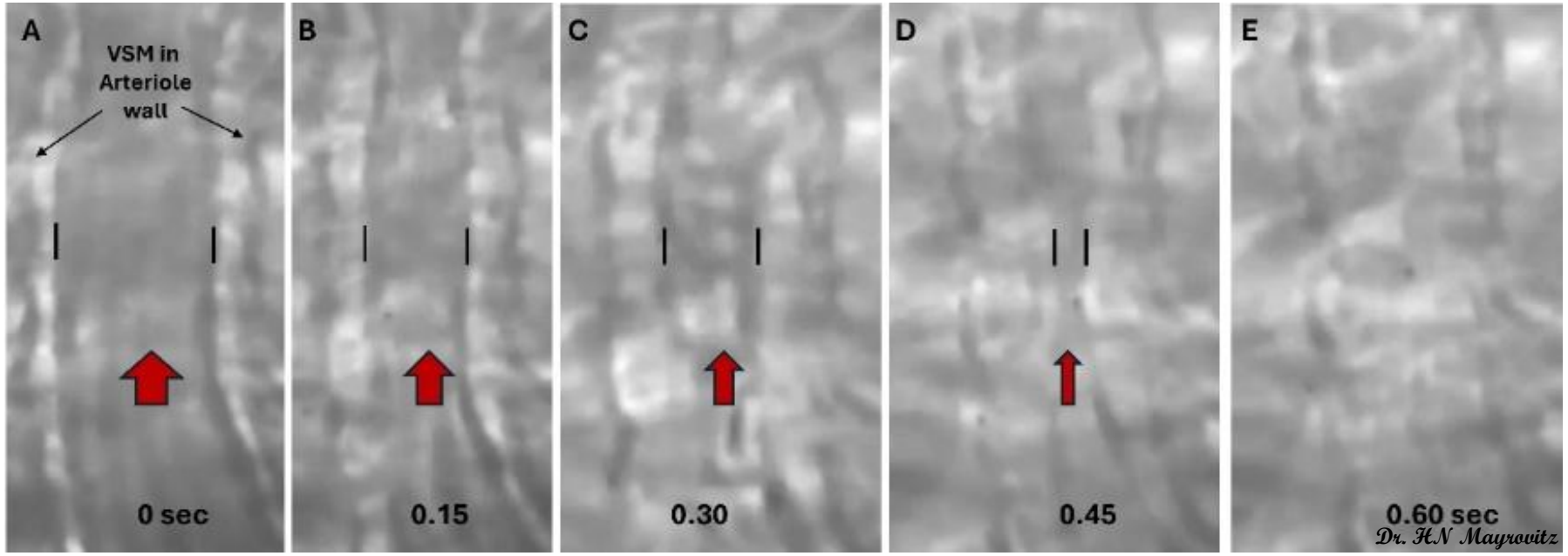


Part 1. Microvascular Flowmotion

Spontaneous Vasomotion as the Source of Flowmotion



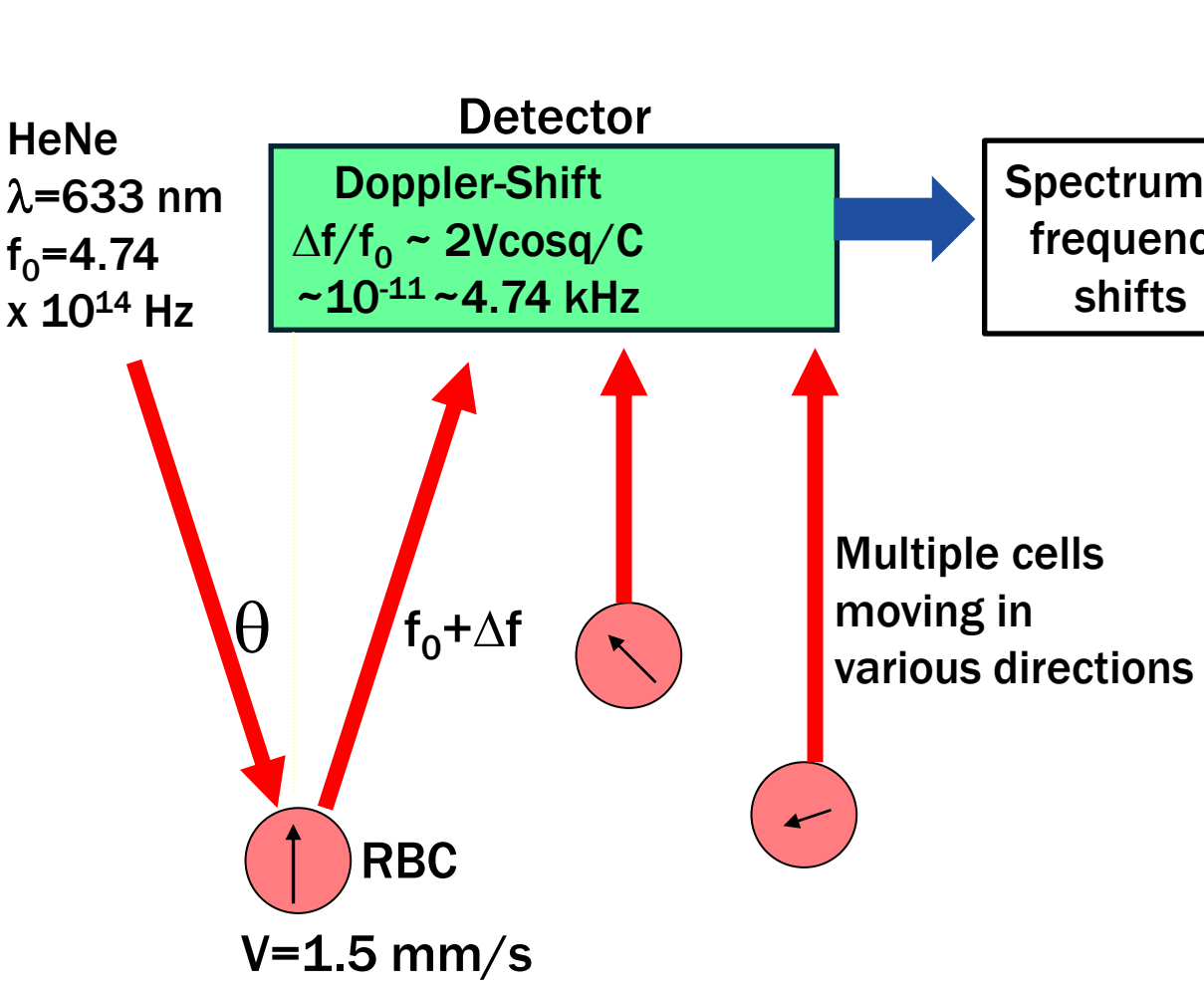
Arteriole displaying spontaneous temporal diameter changes

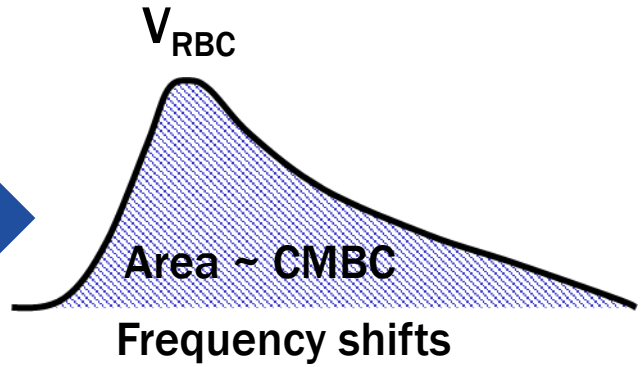


Vasomotion showing arteriole progressive vasoconstriction to 100% closure

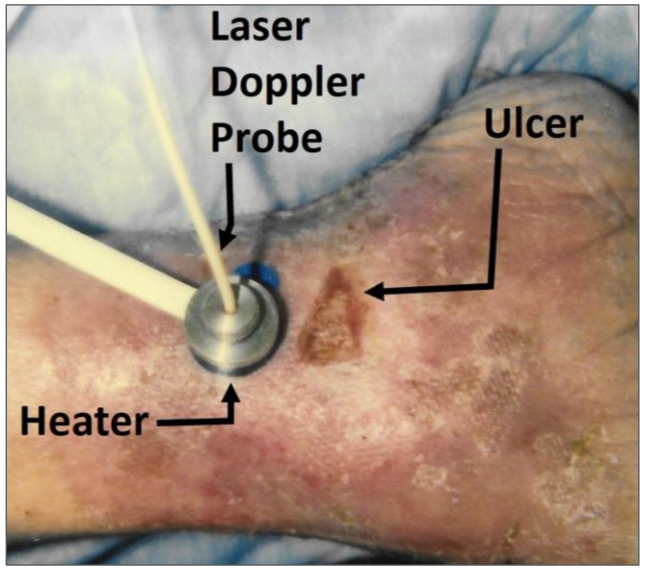
Dr. Mayrovitz. Uploaded 2010. Accessed June 25, 2025. https://www.youtube.com/watch?v=6E_OLwVsf9w

Detecting Flowmotion with Laser Doppler Flowmetry (LDF)



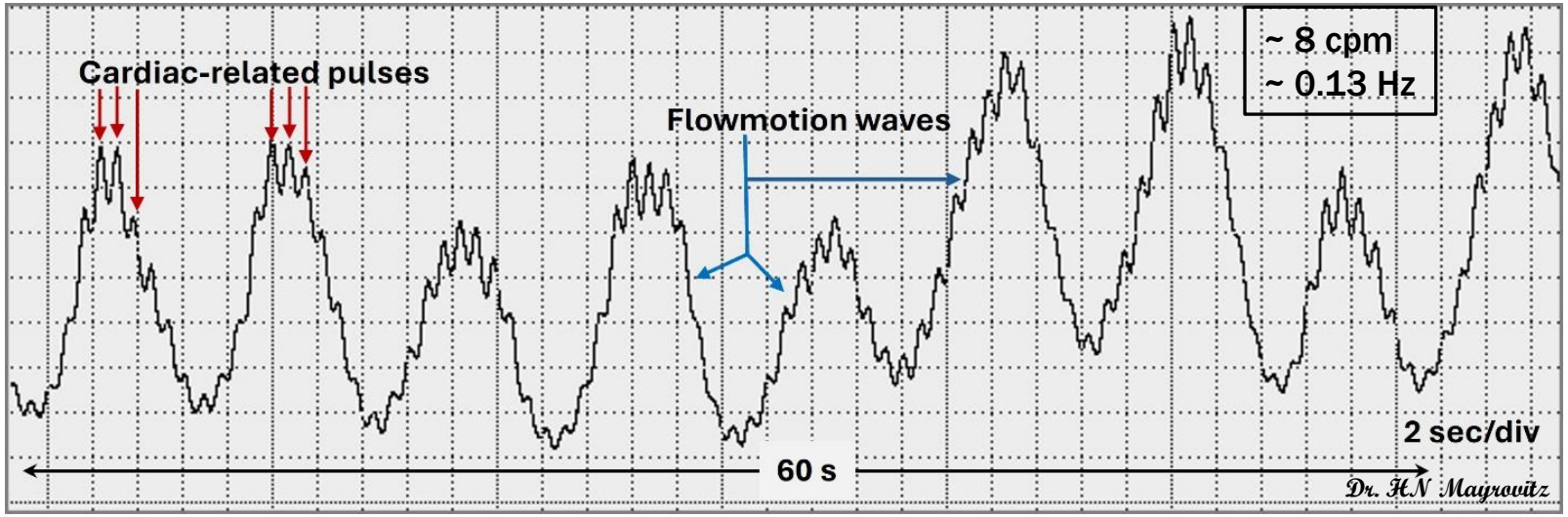


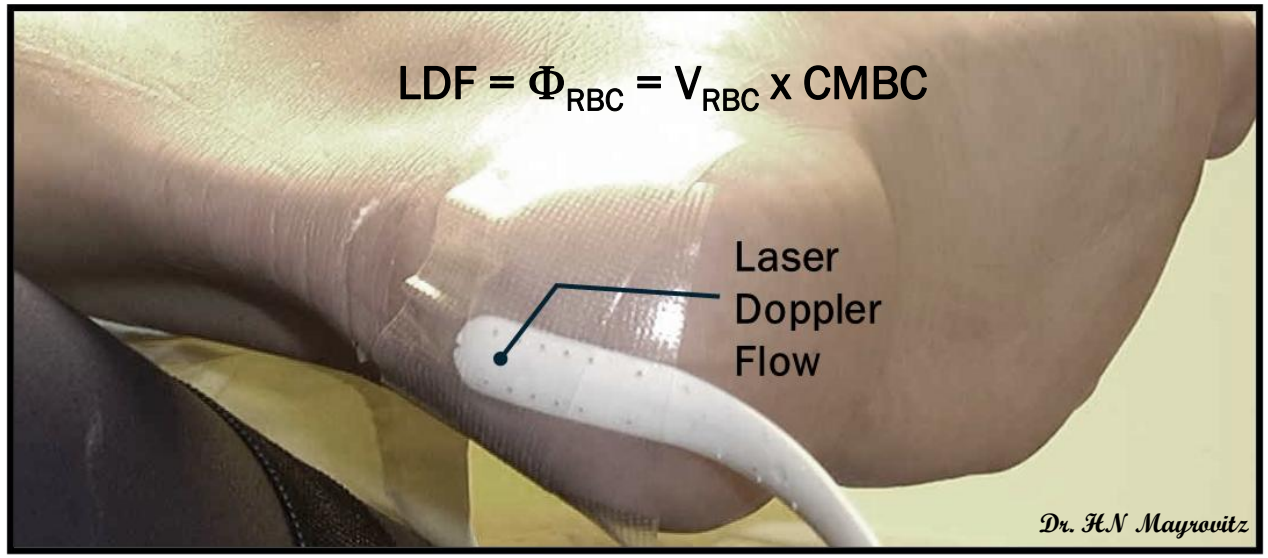
CMBC = concentration of moving blood cells



LDF = Φ_{RBC} = V_{RBC} x CMBC

Example Skin Blood Flowmotion Pattern Measured by LDF

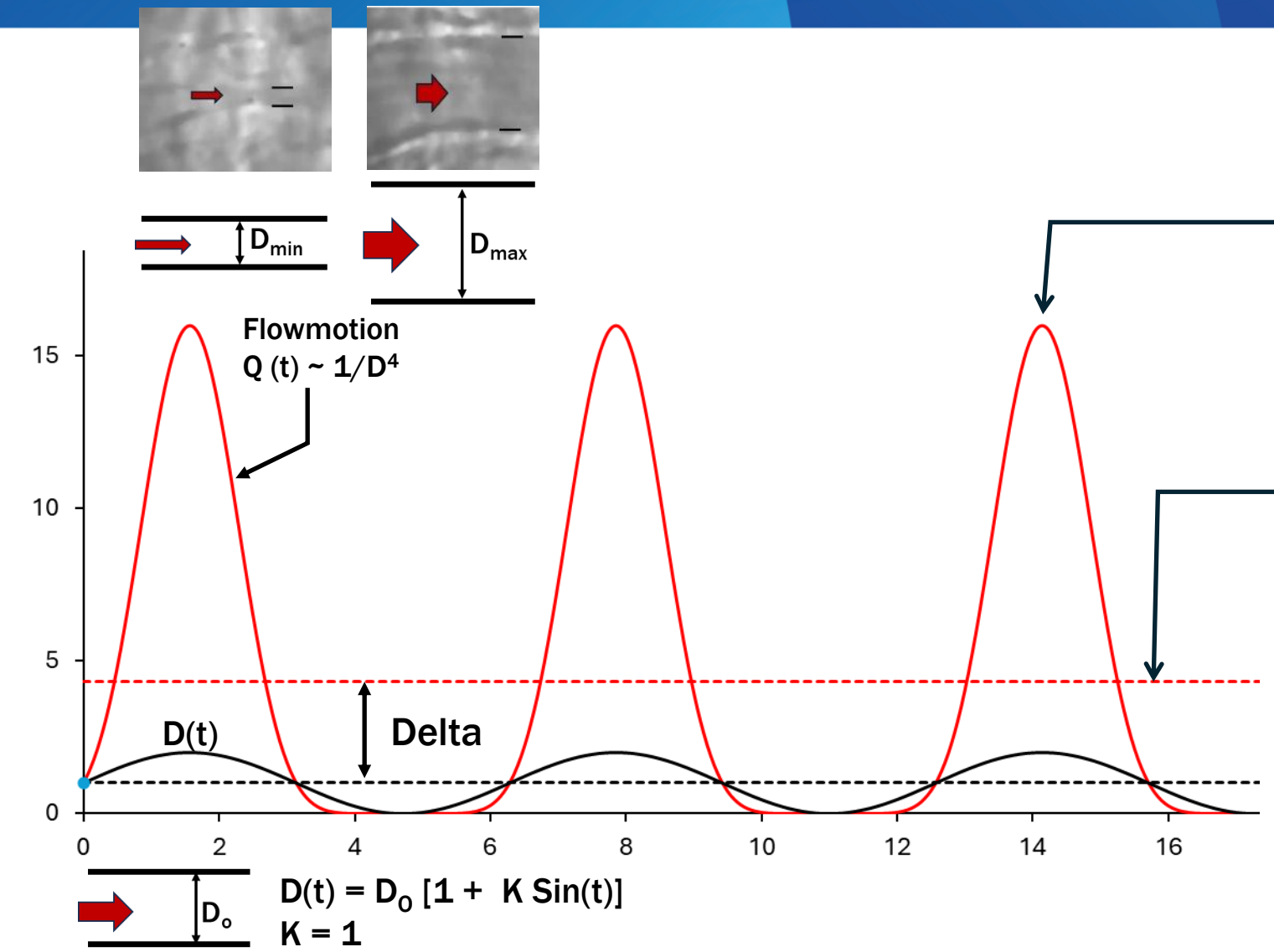




LDF = laser doppler flowmetry.

Mayrovitz HN, Sims N. *Adv Skin Wound Care.* 2002;15(4):158-164. Mayrovitz HN, et al. *Adv Skin Wound Care.* 2003;16:141-145.

Relevant Blood Flow Features of Flowmotion



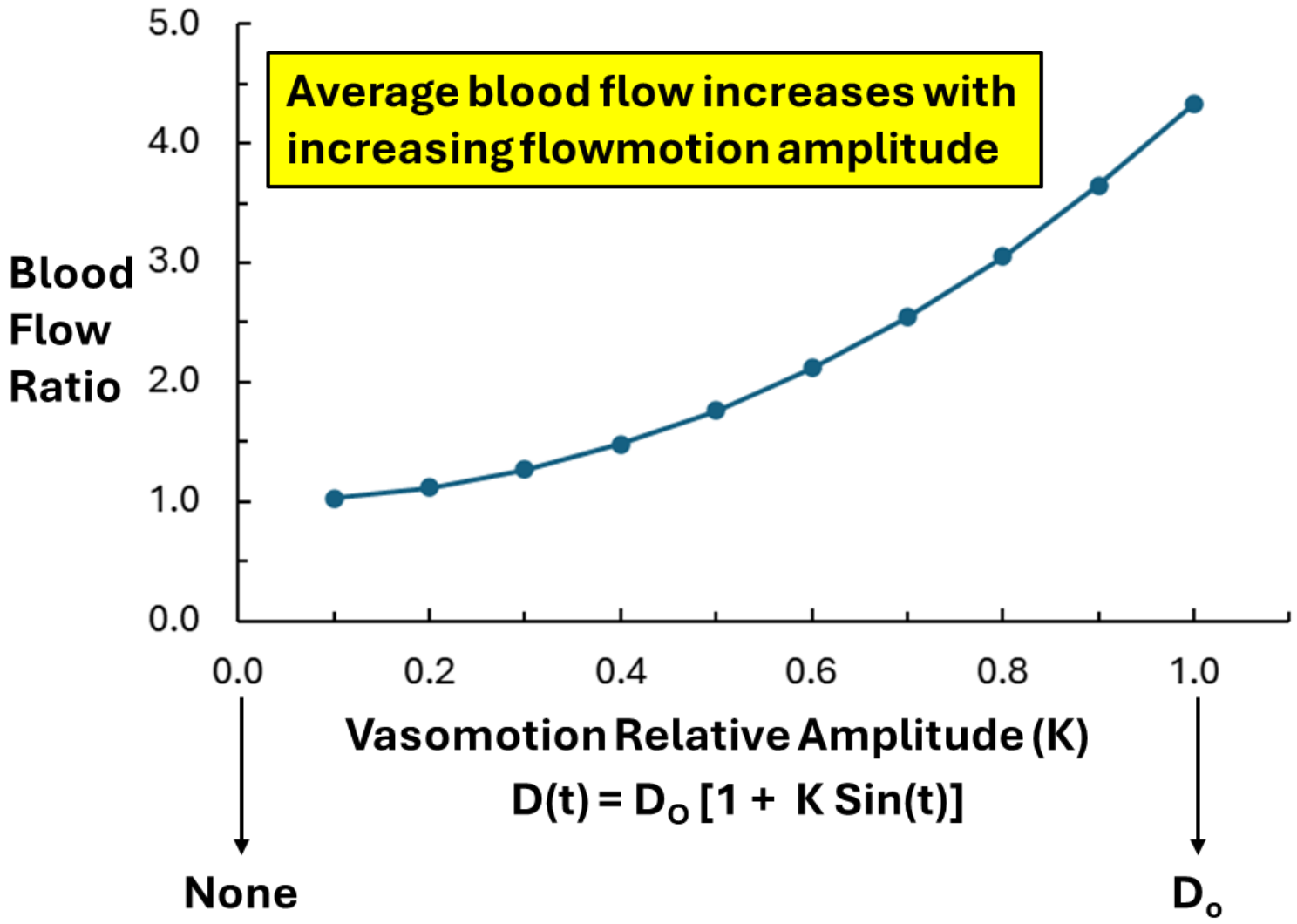
Important Features

- Peak blood flow is associated with an increased pressure gradient and increased shear rate
- Average blood flow is greater with flowmotion than would be in an arteriole with a fixed diameter (Do)

Dr. HN Mayrovitz

Mayrovitz HN, et al. *Wounds*. 1993;5(1):47-55.

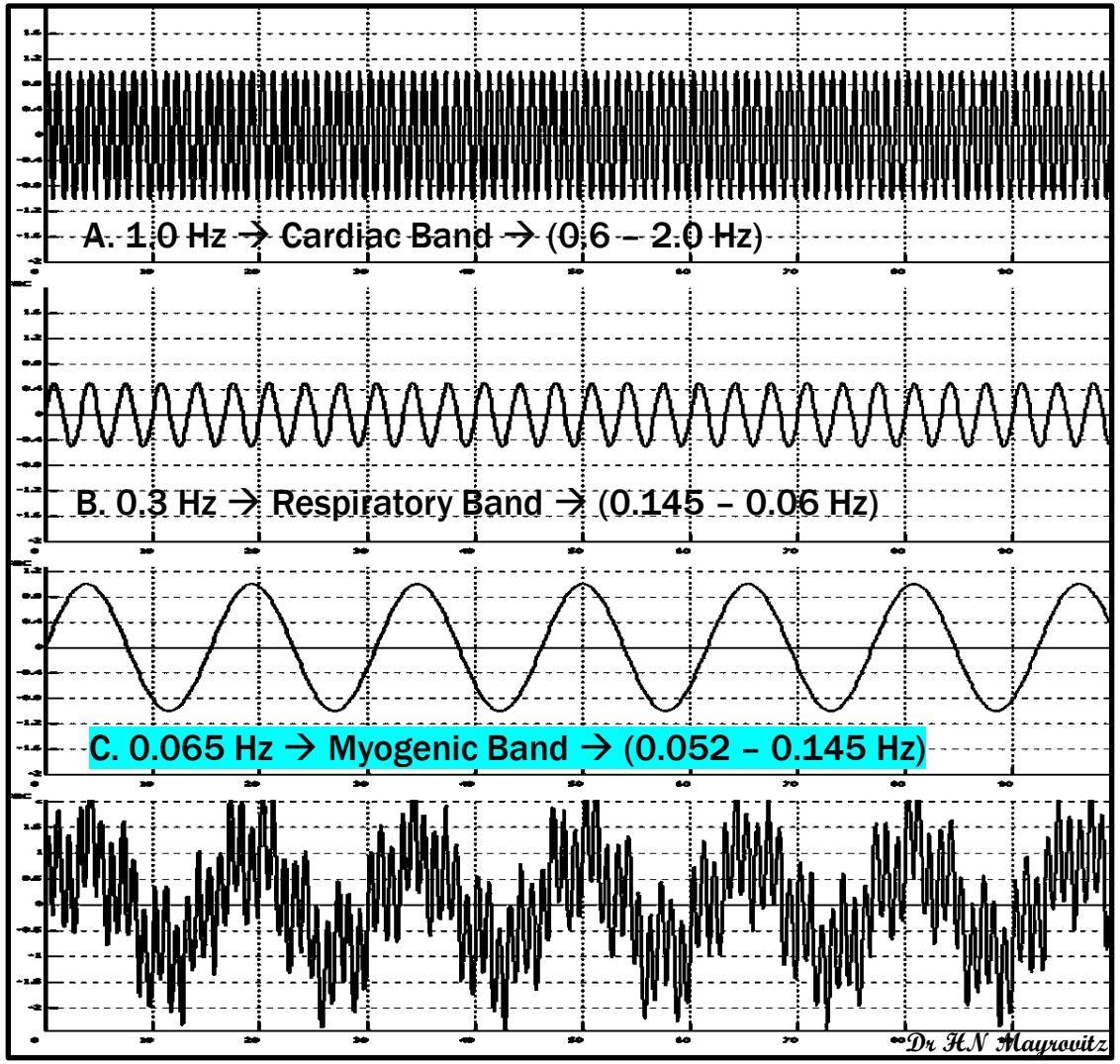
Blood Flow Augmentation Depends On Vasomotion Amplitude



A question for later

What processes or conditions increase or decrease flowmotion amplitude and thereby affect flow?

Spectral Analysis of Flowmotion Signals

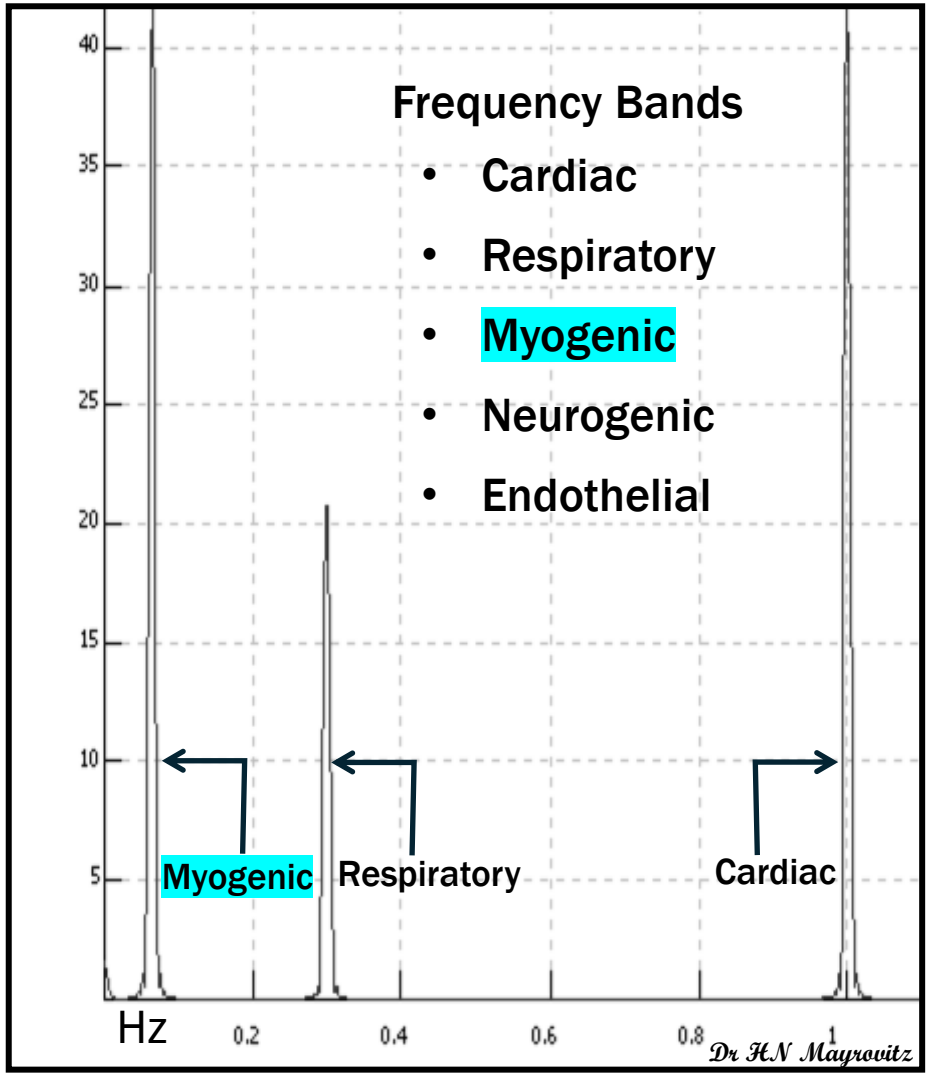


Composite = A + B + C

Multiple Physiological Processes

Myogenic is of current interest

Spectral Analysis
- Fourier
- Wavelet
- Other

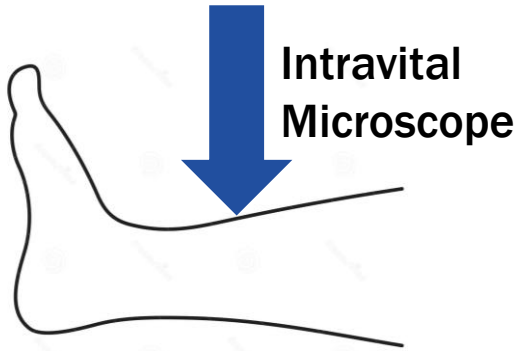


Frequency Spectrum

Compression in Chronic Venous Insufficiency

Methodology

Images visualized and recorded



12 women with CVI

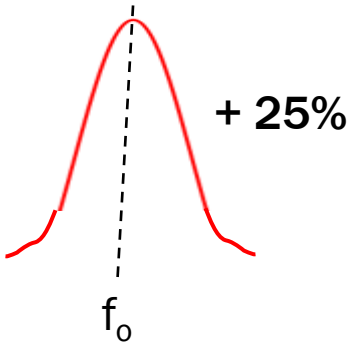
Compression for 30 days

Bandage → Class II stockings

Observations

- Perfused nodal points +17%
- Venular blood flow +22%
- RBC aggregation –20%
- WBC vein adherence –61%

Flowmotion (FM) via Spectral Analysis



Explanation?

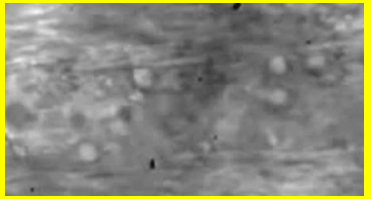
- High peak FM gradients promote flow in capillaries anatomically present but not yet having flow

  Capillary recruitment

- High peak FM shear helps remove WBCs adherent to venular endothelium

  Reduced vascular resistance

  Reduces WBC cytotoxic effects



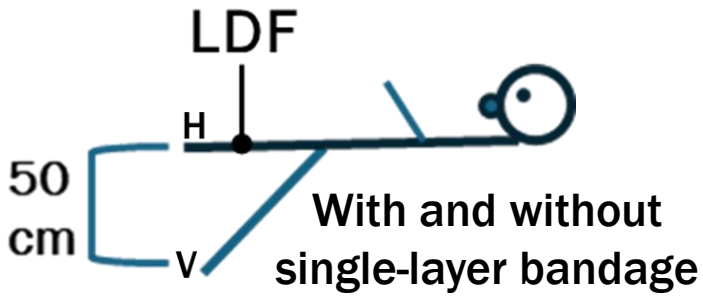
- Improve rheological properties of blood via a "shake up" process

Dr. HN Mayrovitz

Kloop, et.al. *Phlebology.* 1996;11:19-25

Compression Bandage Mitigates Flowmotion Reduction

- 19 Healthy adults



With and without single-layer bandage

- Observed Flowmotion (FM)

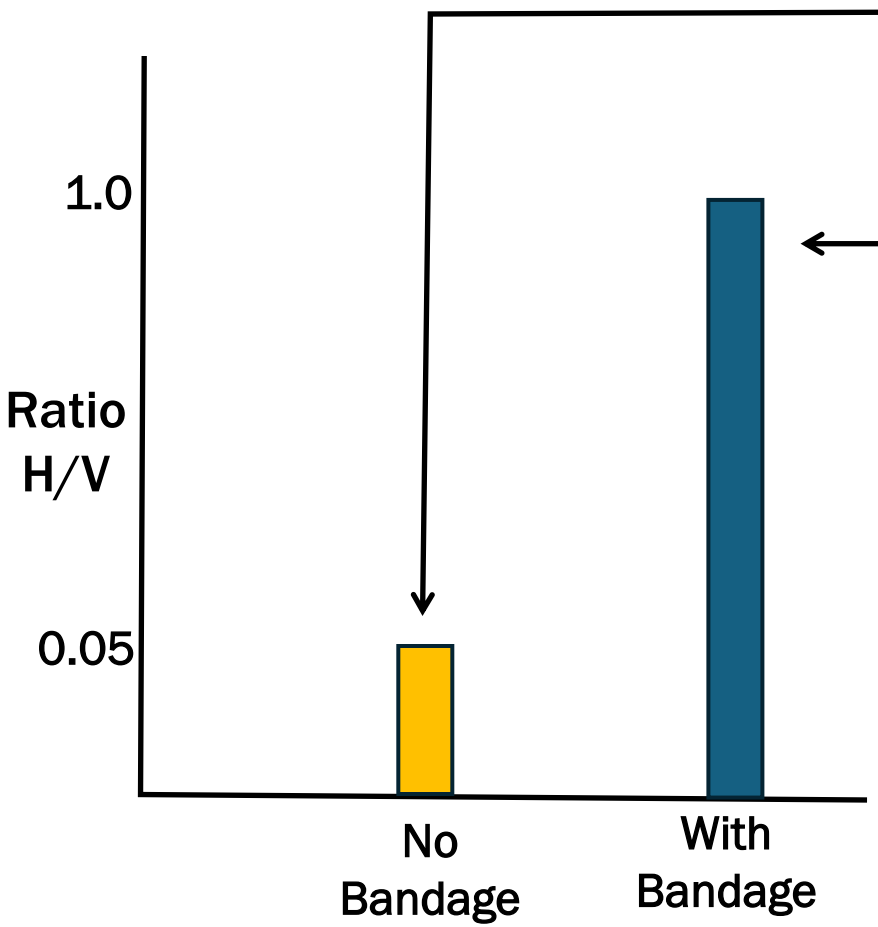
  7.5 ± 0.6 cpm

  0.125 ± 0.01 Hz

- Compared H vs V with ankle pressure = 25 mm Hg

- Flowmotion amplitude ratio

  Leg H / Leg V = Ratio



- FM dramatically decreased in the dependent position when not bandaged
- FM amplitude reduction essentially prevented with compression bandage
- Positive aspects of FM are preserved with bandaging
- Counteracting the gravitational pressure-induced increase in transmural pressure?

Dr. H.N Mayrovitz

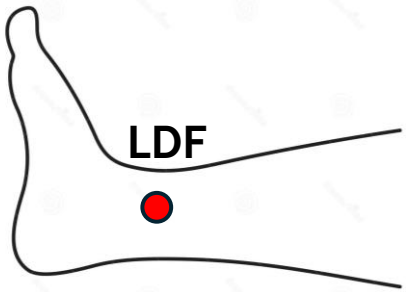
Gniadecki, et al. *Acta Derm Venereol* (Stockh) 1992;72:259-260.

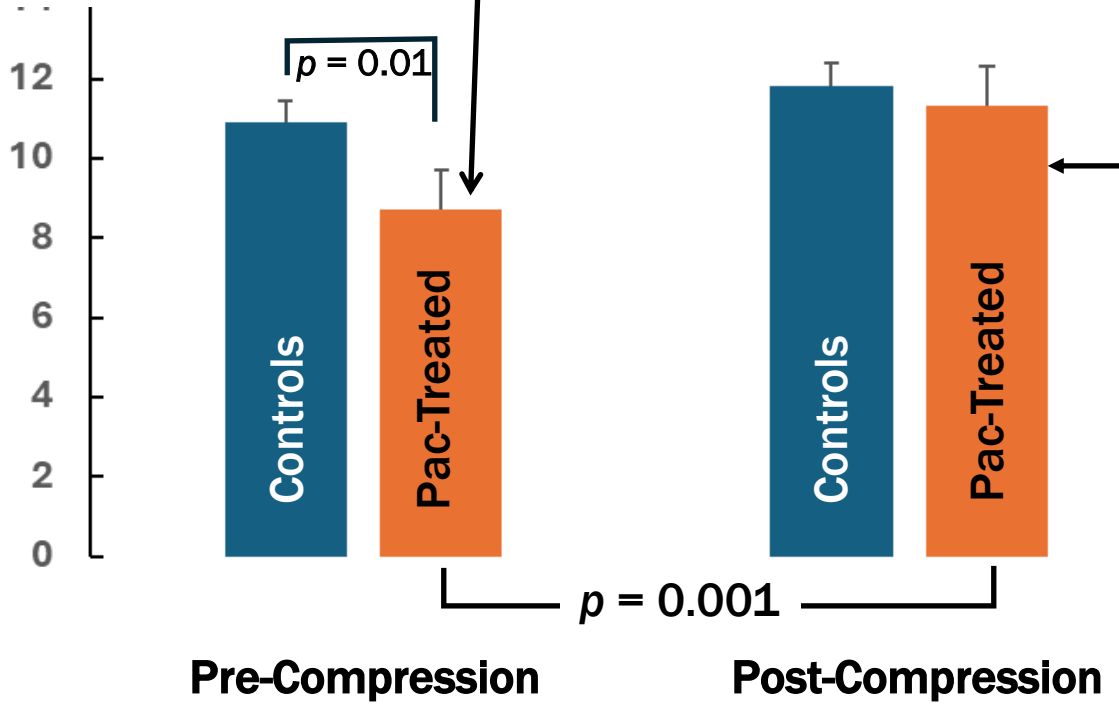
Compression-Induced Microcirculatory Blood Flow Increase

50 breast cancer patients with chemotherapy-induced peripheral neuropathy

50 healthy controls

Lower extremity LDF pre-post stocking compression



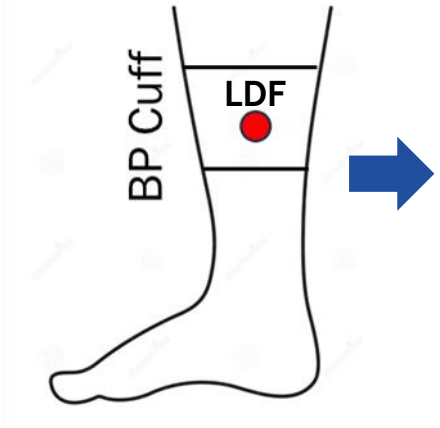


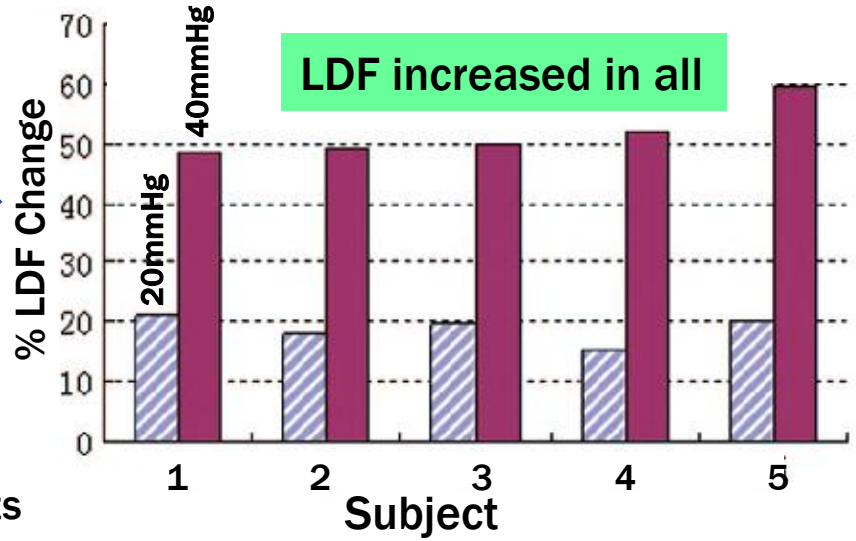
- Skin LDF blood perfusion less in patients treated with paclitaxel
- Compression significantly increased LDF in treated patients but had little effect on controls
- Compression pressure and duration not given

Dr. HN Mayrovitz

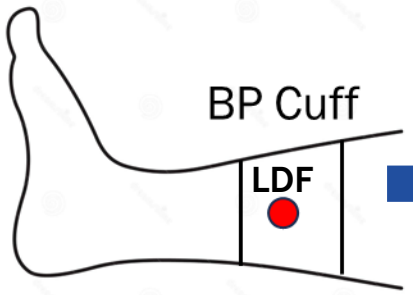
Ohno et al. *J Clin Oncol.* 2014;32 (15) https://doi.org/10.1200/jco.2014.32.15_suppl.e20677

Flowmotion vs Physical Compression Effects

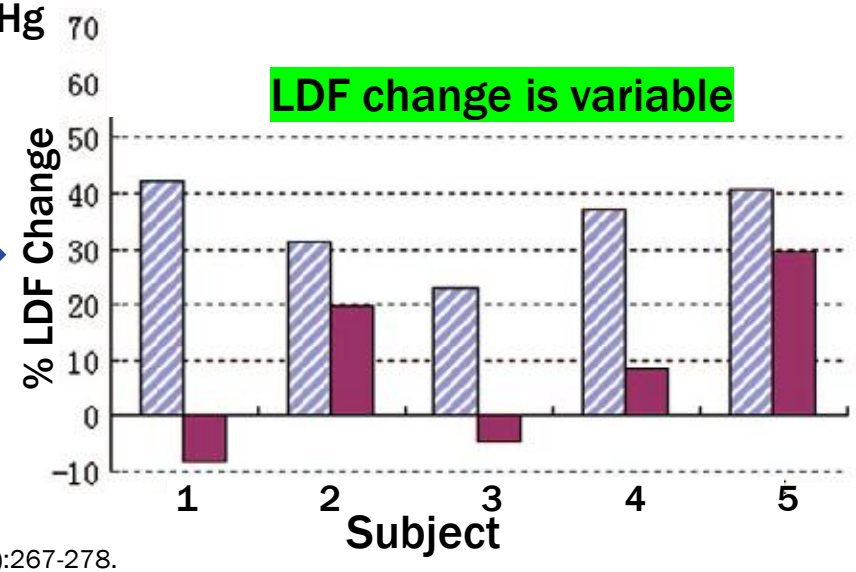




- N = five healthy subjects
- Cuff to 20 and 40 mm Hg



Not full bandage



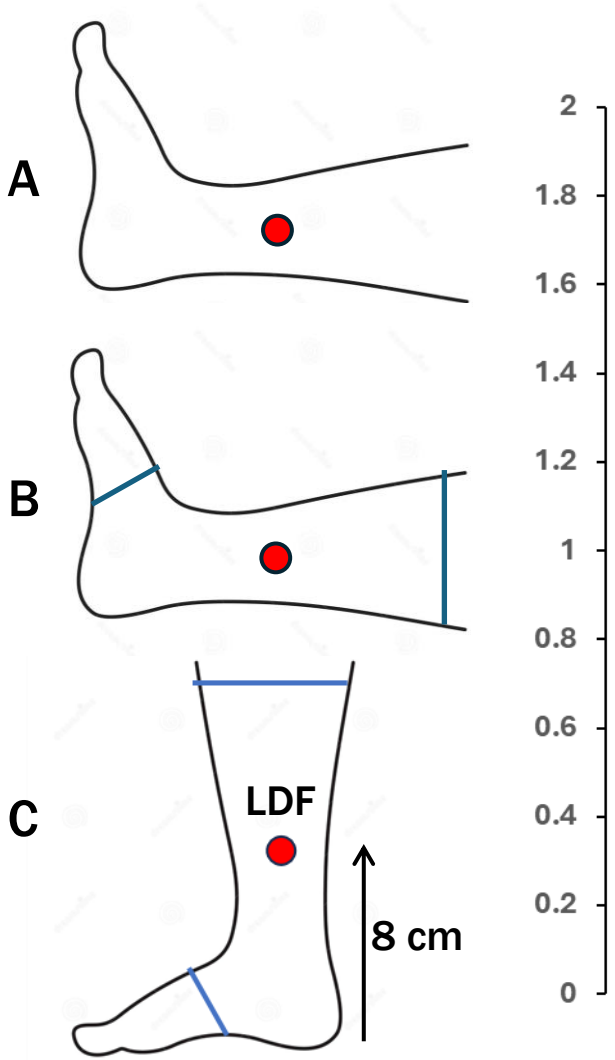
Dia, et al. *Biol Rhythm Res.* 2012;43(3):267-278.

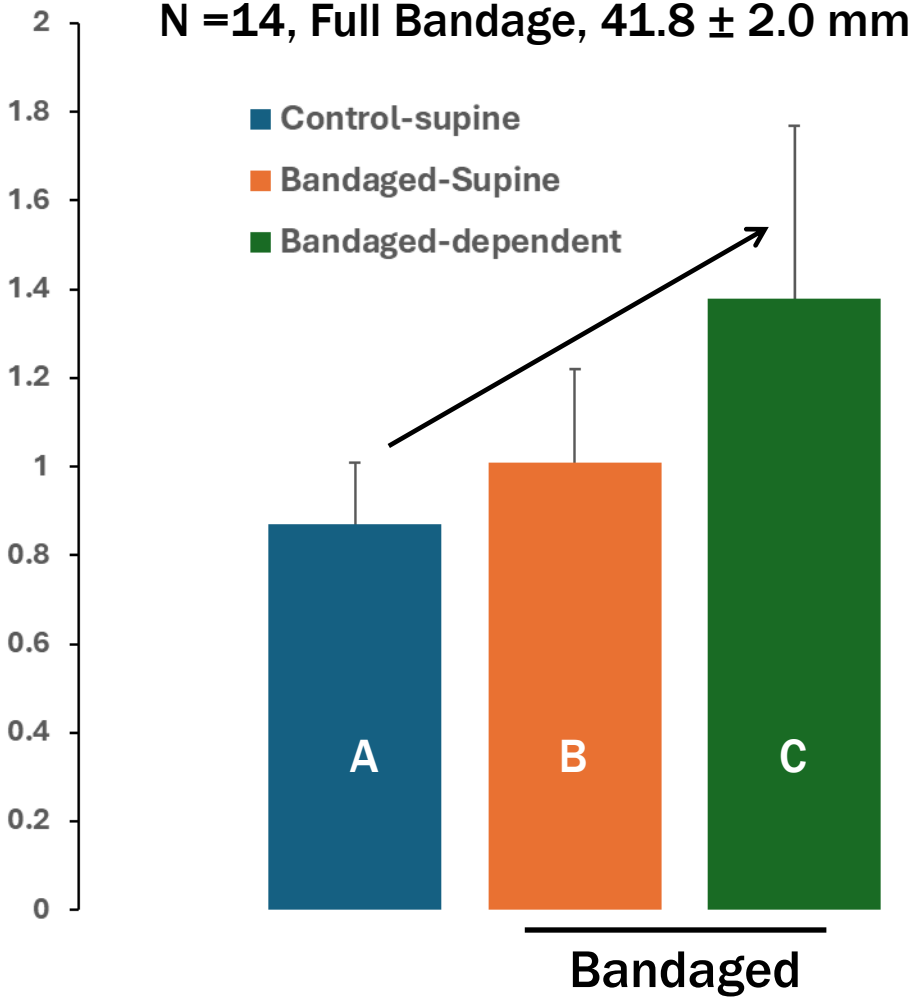
Interpretation

- Compression → + myogenic spectral amplitude → + LDF
- LDF increase with compression depends on the intravascular pressure (IVP)
- Outcome depends on the balance between:
  A. capillary recruitment by pressure-induced myogenic flowmotion and
  B. direct vessel physical compression effects
- Threshold 35-40 mm Hg?

Dr HN Mayrovitz

Variable Perfusion Response to Compression Bandaging





Interpretation

- Compression-related LDF increase
- Consistent with individual variability in in intravascular pressure (IVP)

  4/14 → - LDF → -35 ± 9.9%

  6/14 → + LDF → 106 ± 34%

- Higher IVP (within limits) achieves greater perfusion increase

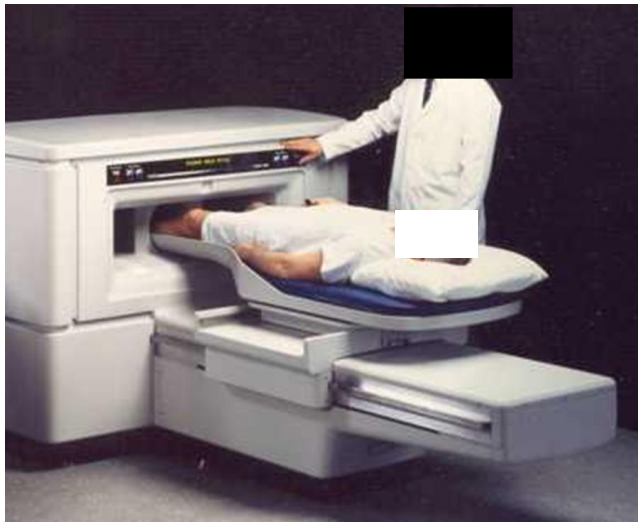
Dr. HN Mayrovitz

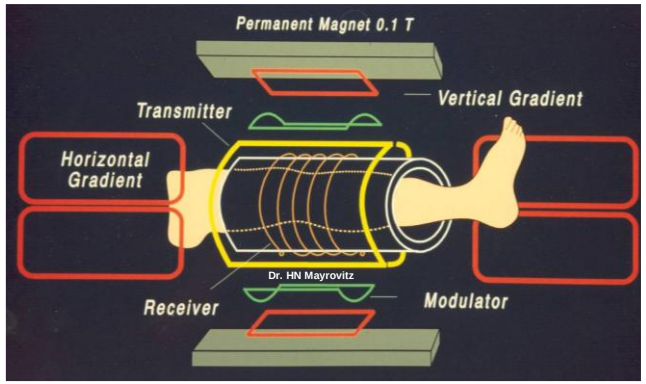
Mayrovitz HN, et al. *WOUNDS*. 1996;8(6):200-207.

Part 2. Pulsatile Arterial Blood Flow

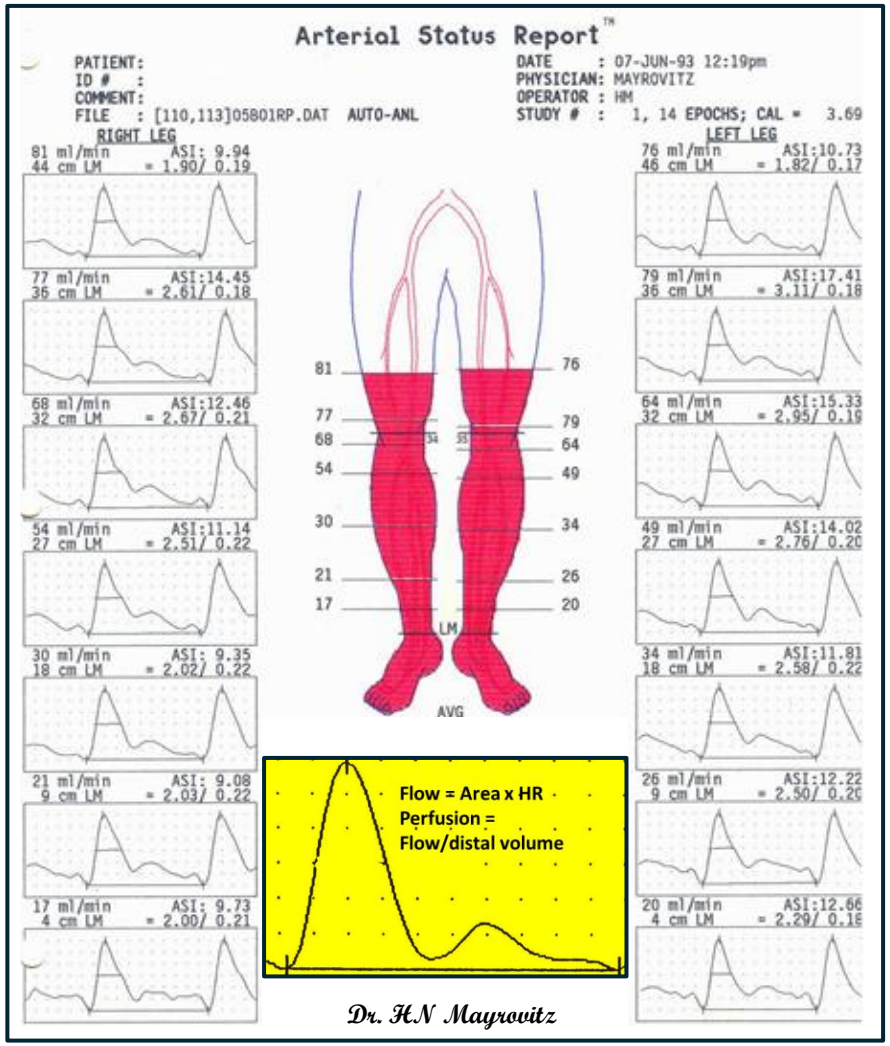
LV

Assessing Arterial Pulsatile Blood Flow via NMR



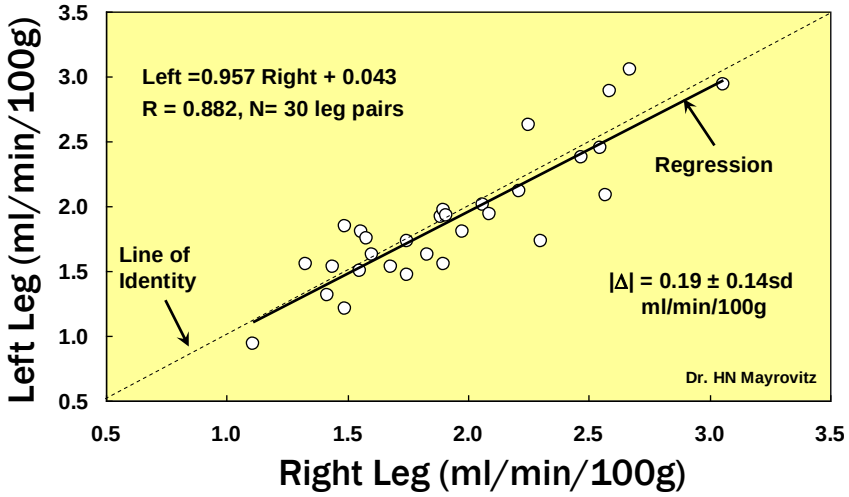


Nuclear Magnetic Resonance



Pulse flow at multiple cross-sections

Normally, flow values are very similar bilaterally at corresponding sections

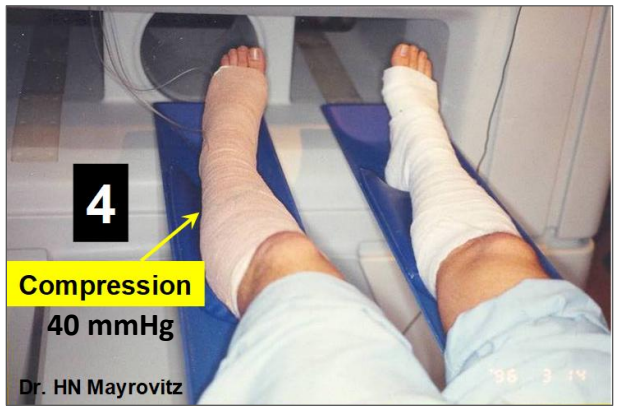


Would generally expect the flow Ratio (Left Leg/Right Leg) ~ 1

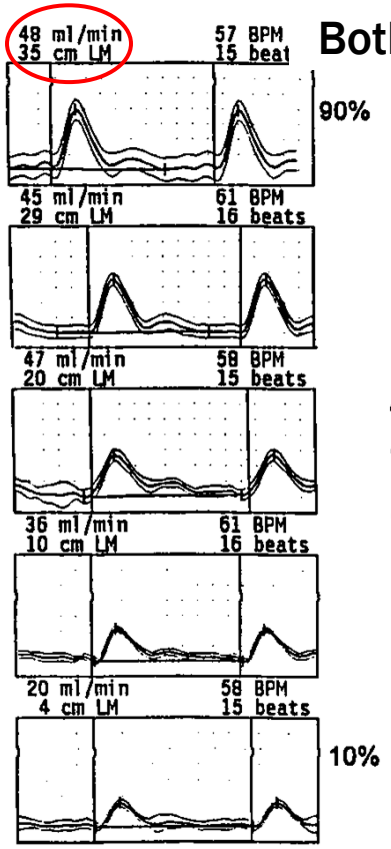
NMR = nuclear magnetic resonance.

Mayrovitz HN, Larsen PB. *Clin Physiol.* 1996;16(5):495-505.

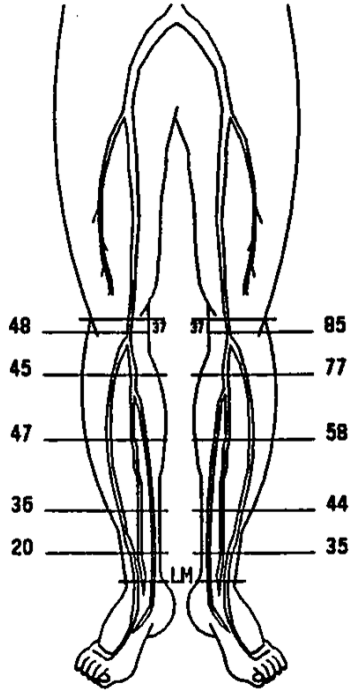
Assessing Compression Bandaging Effects

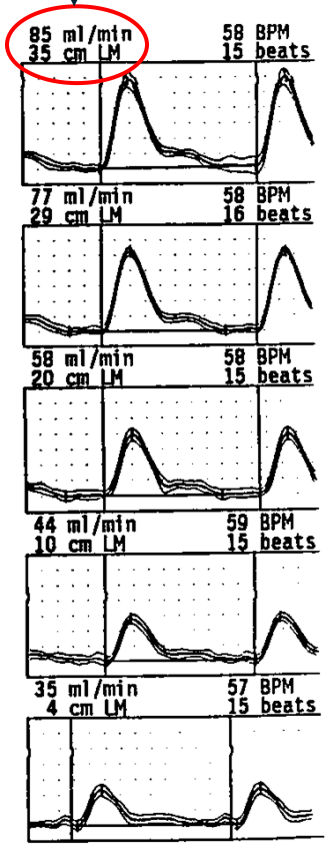


Both show the left leg



No Compression





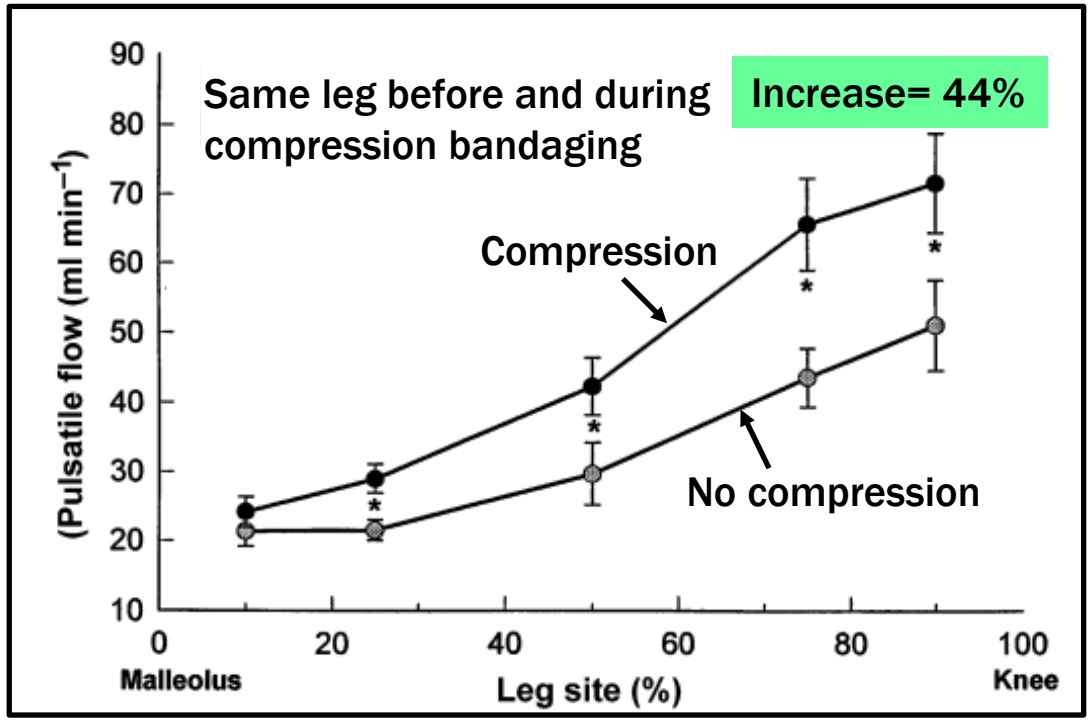
Compression Bandage

- Leg with compression bandaging had increased pulsatile blood flow at every cross-section
- Flow ratios for this subject were 1.77 at 90% distance and 1.75 at 10% distance
- Increased pulsatility likely helps to promote increased lymphatic / venous flow

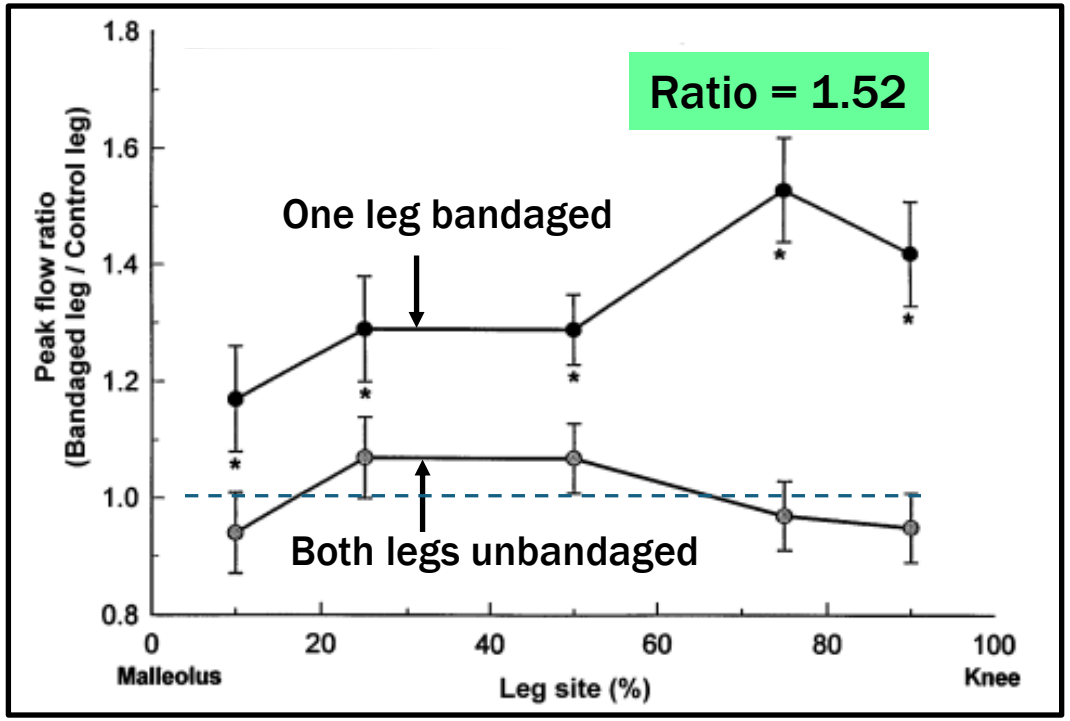
Dr. HN Mayrovitz

Mayrovitz HN, Larsen PB. *Clin Physiol.* 1997; 17(1):105-117.

Blood Flow Increase Associated with Compression Bandaging

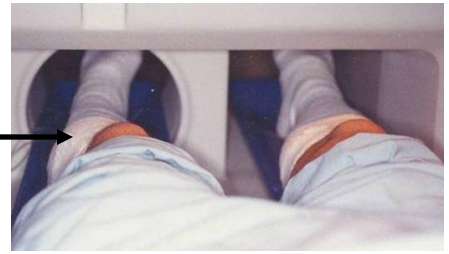


Pulsatile Blood Flow by Leg Site (N = 14)

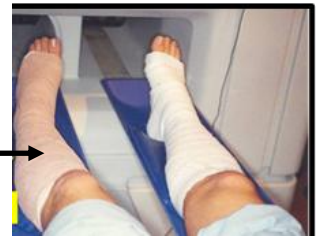


Peak Blood Flow Ratio (Bandaged Leg/Control Leg)

Left leg 1st with no compression then with compression

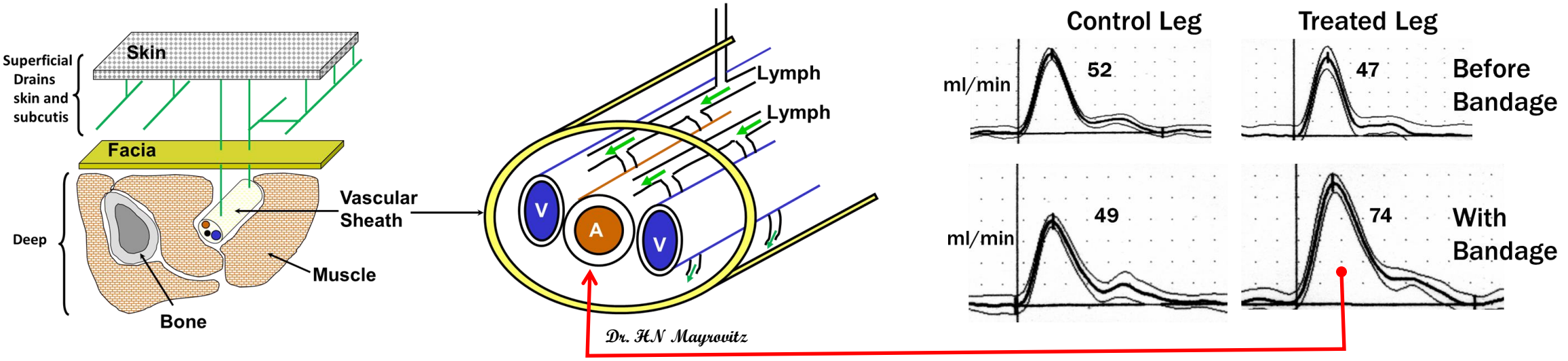


Both legs had no compression then the left leg was bandaged



Mayrovitz HN, Larsen PB. *Clin Physiol.* 1997; 17(1):105-117.

Example of Increased Pulsation's Impact Lymph/Venous Transport



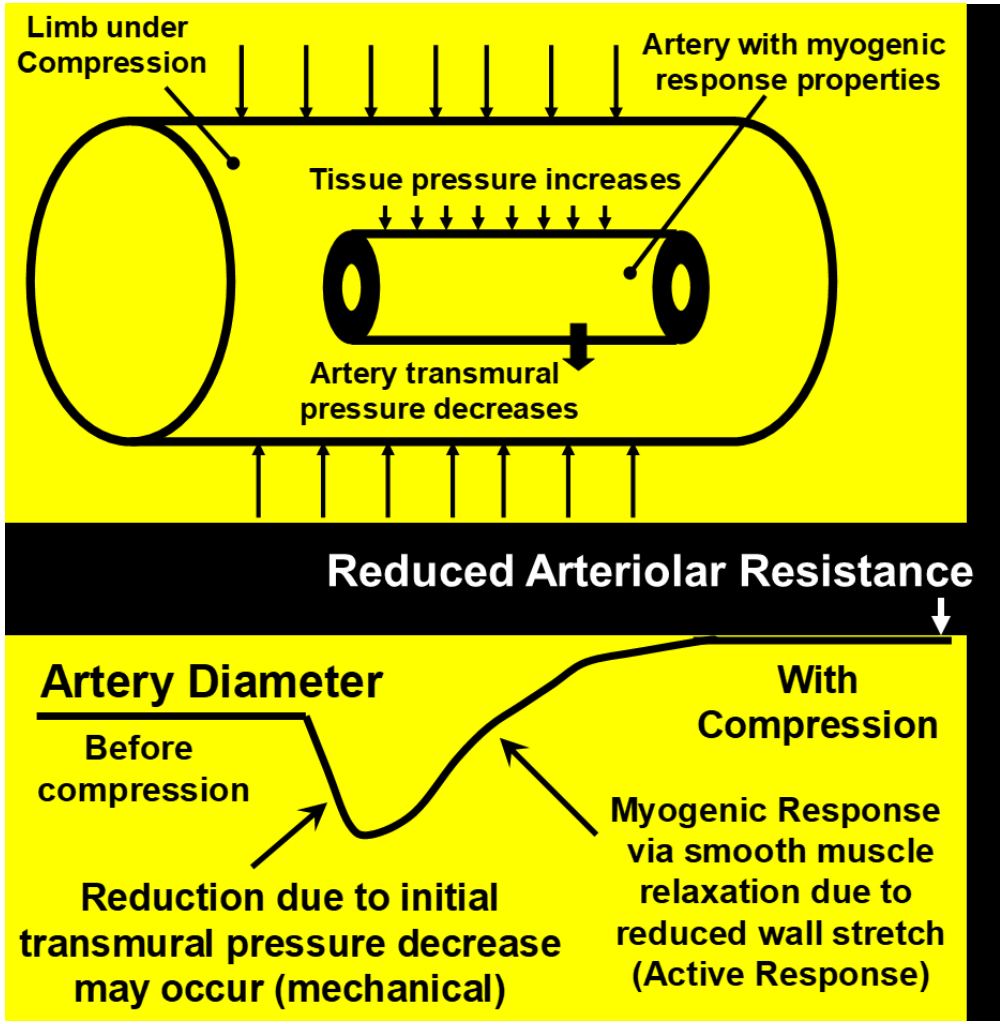
Increased pulse amplitude likely mechanically augments lymph/venous transport

Increased interstitial pulse pressure at 60-80 times/min → Mechanical coupling to both lymphatic and venous vessels → + Lymphatic stimulation → + Lymph transport
+ Venous volume reduction → + Fluid reuptake
+ Venous shear rate → Adherent cell clearance

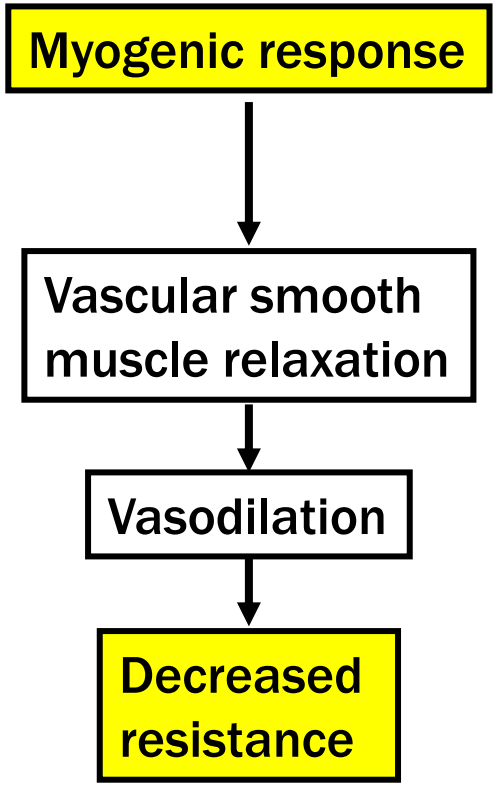
Bottom line: take home

Potential Compression Related Mechanisms

Arteriole Myogenic Related



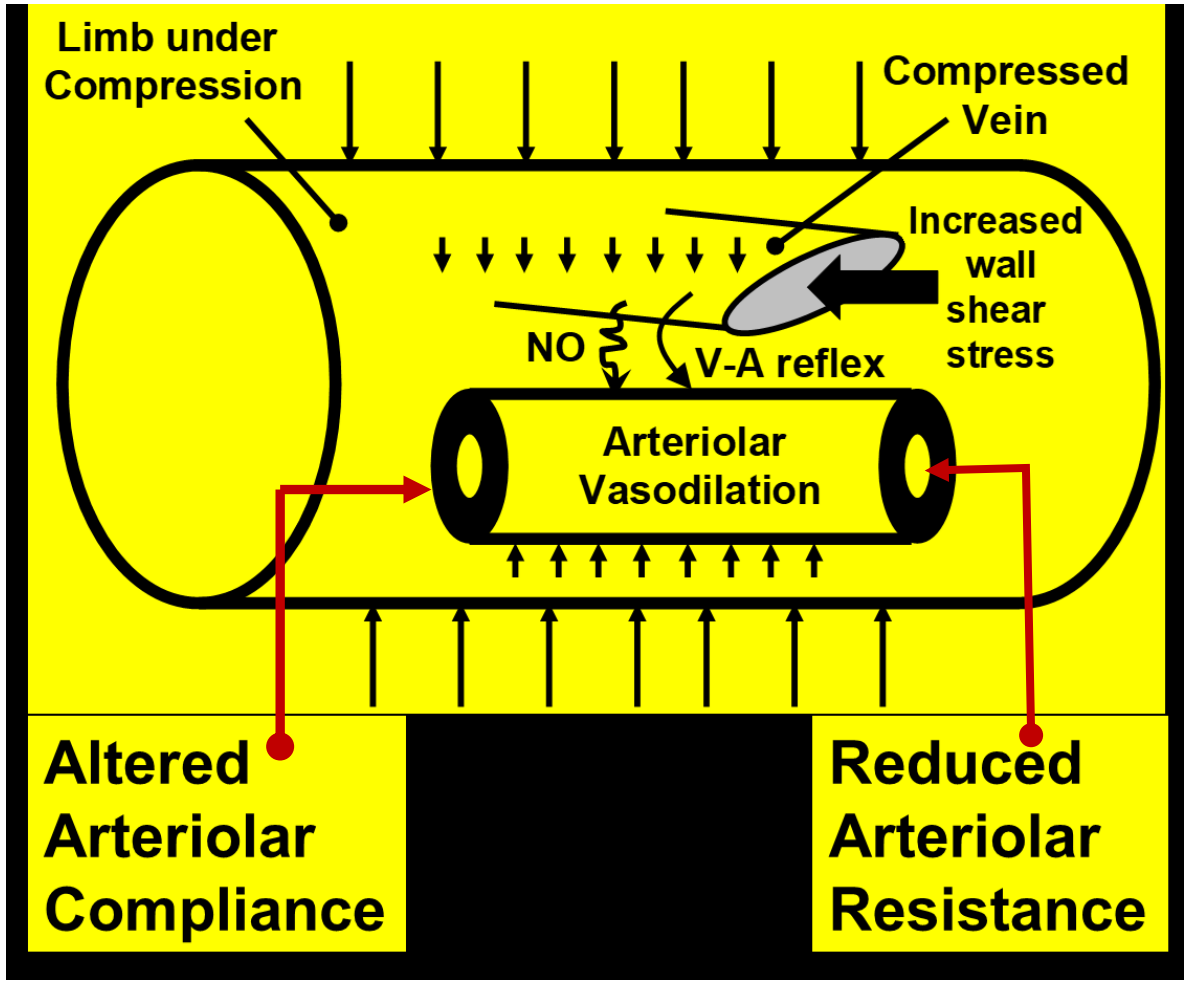
Arteriolar transmural pressure decreases



Myrovitz HN, Macdonald JM. *Int Angiol.* 2010;29(5):436-441,

Potential Compression Related Mechanisms

Venous Compression Related



Venous compression

+ venous velocity

↓

+ wall shear rate

↓

+ Nitric oxide
V-A reflex

↓

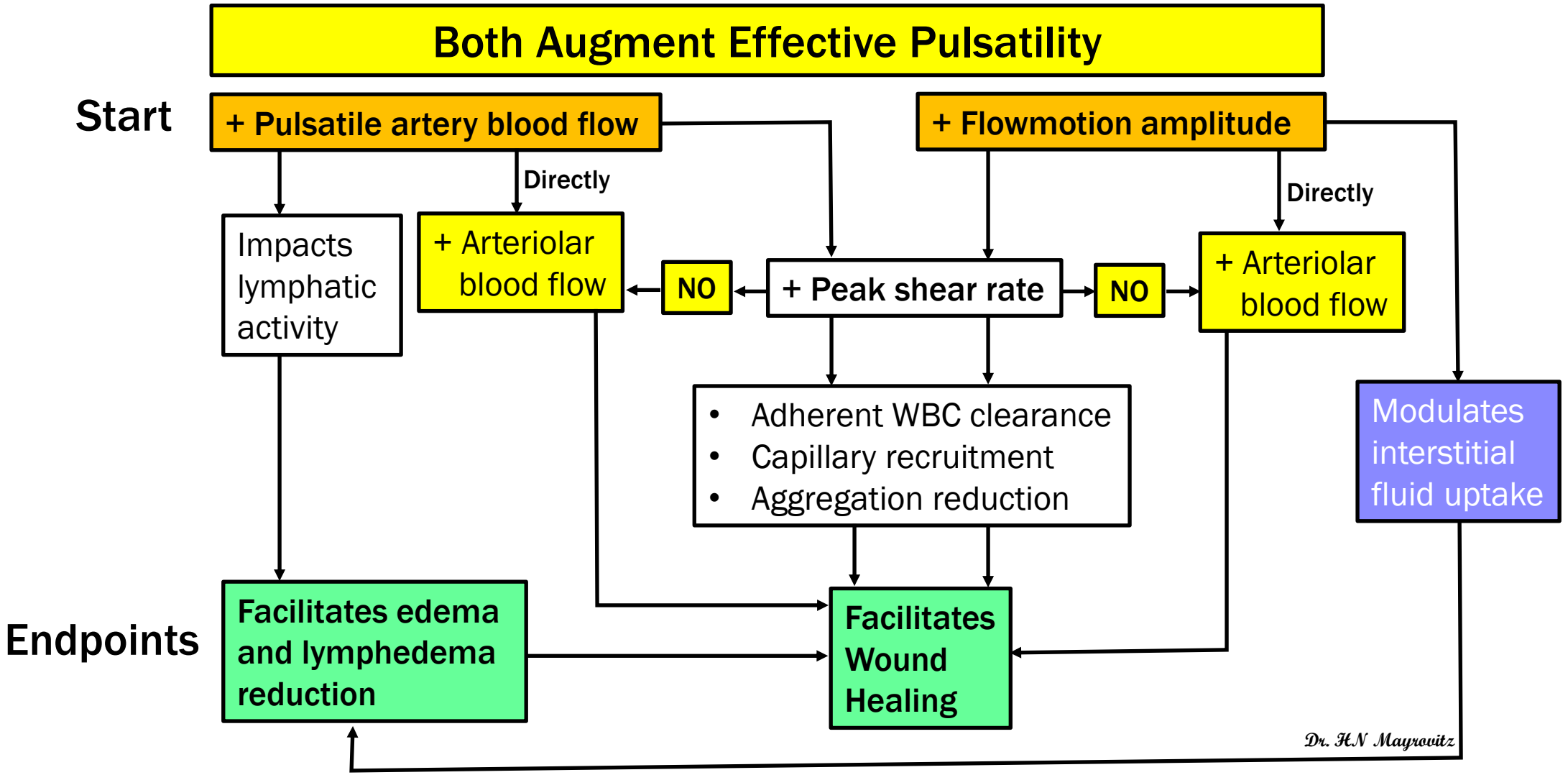
Vasodilation

↓

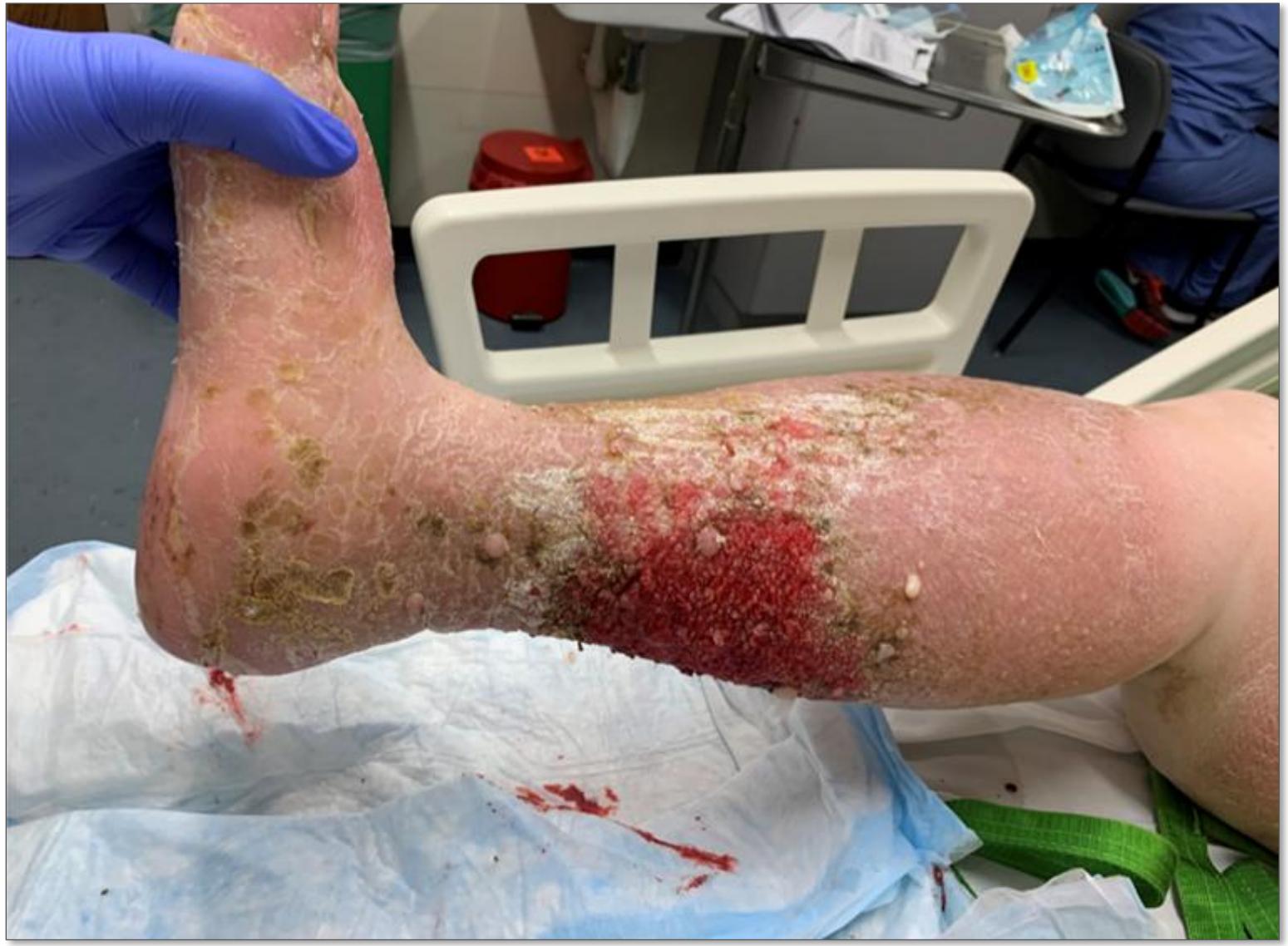
Decreased resistance

Mayrovitz HN, Macdonald JM. *Int Angiol.* 2010;29(5):436-441,

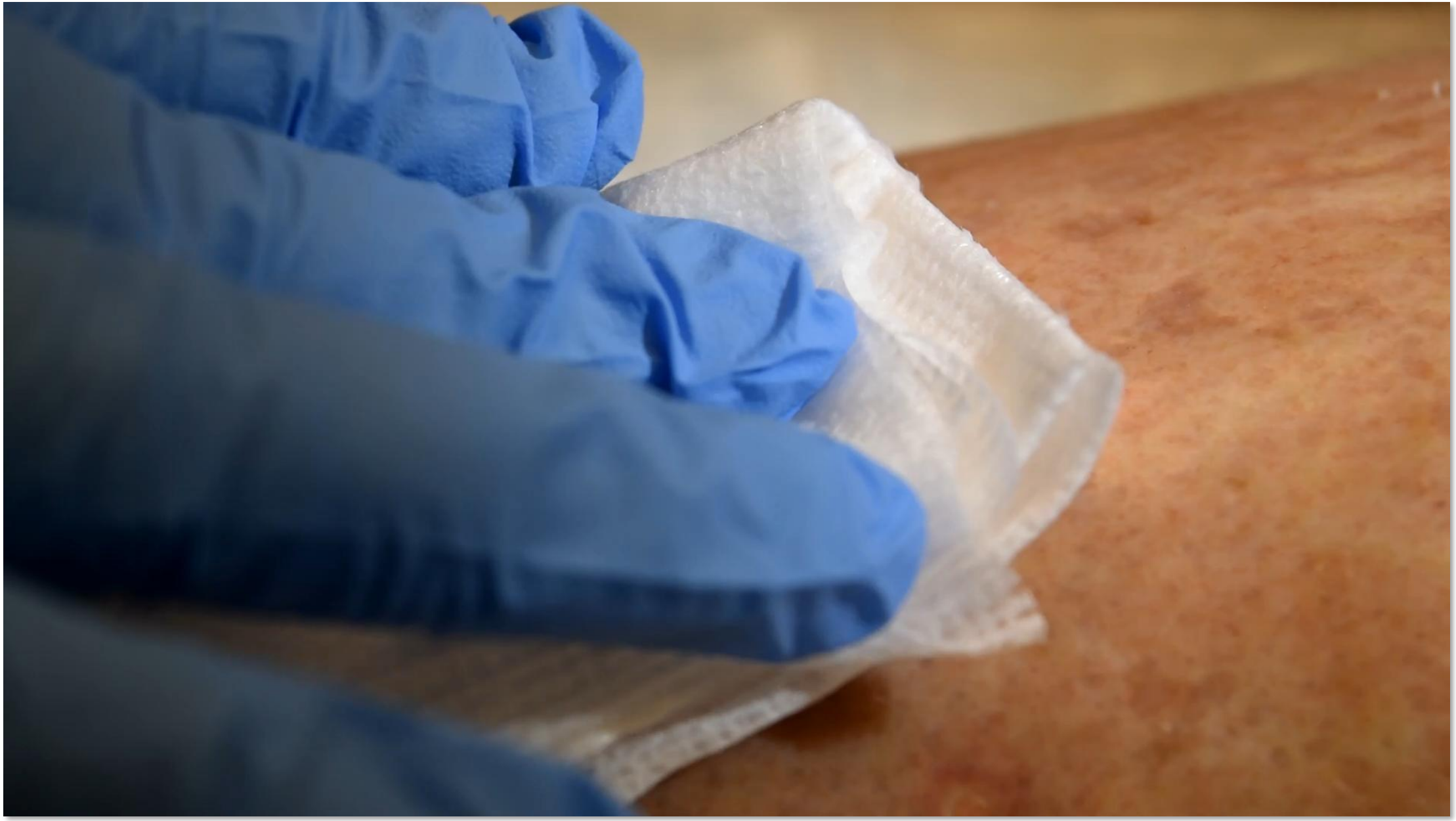
Summary of Some Blood Flow Impacts of Compression Bandaging



Dermal lymphatic stasis



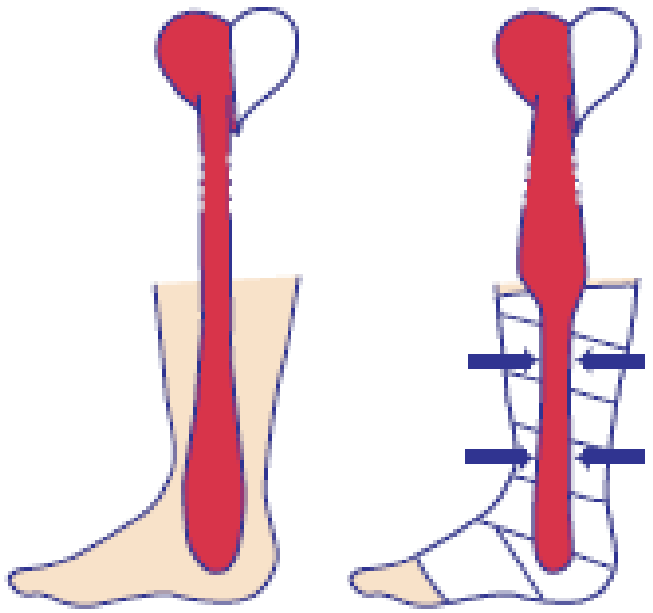
Lymphorrhea



Effect of Stretching

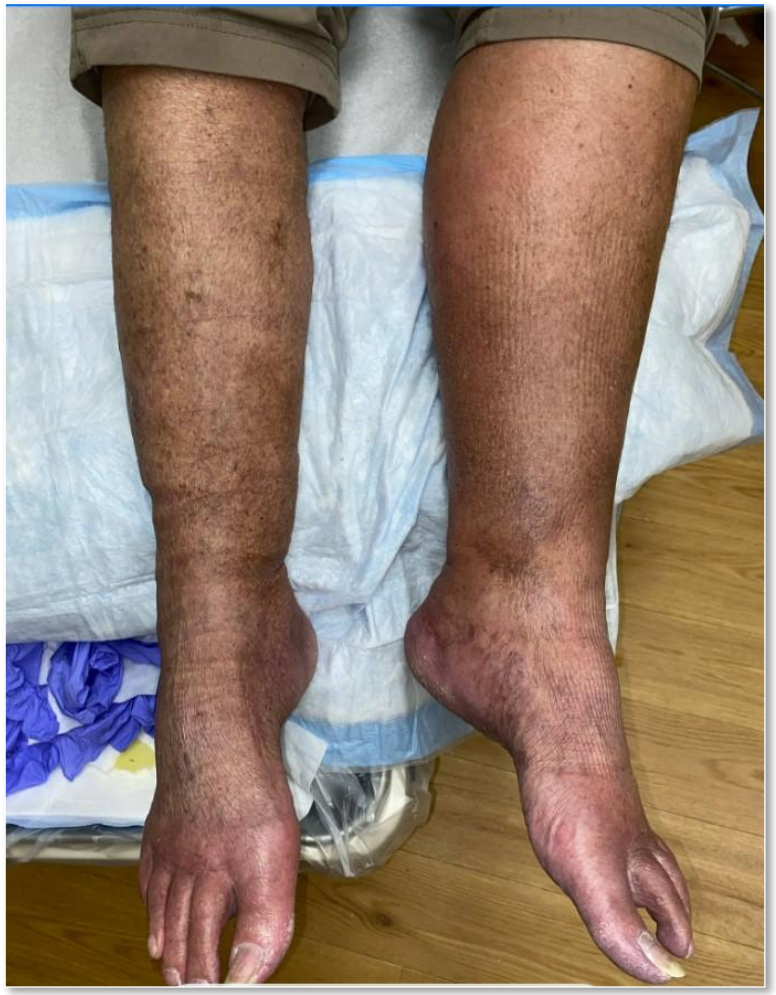
Rationale for Compression

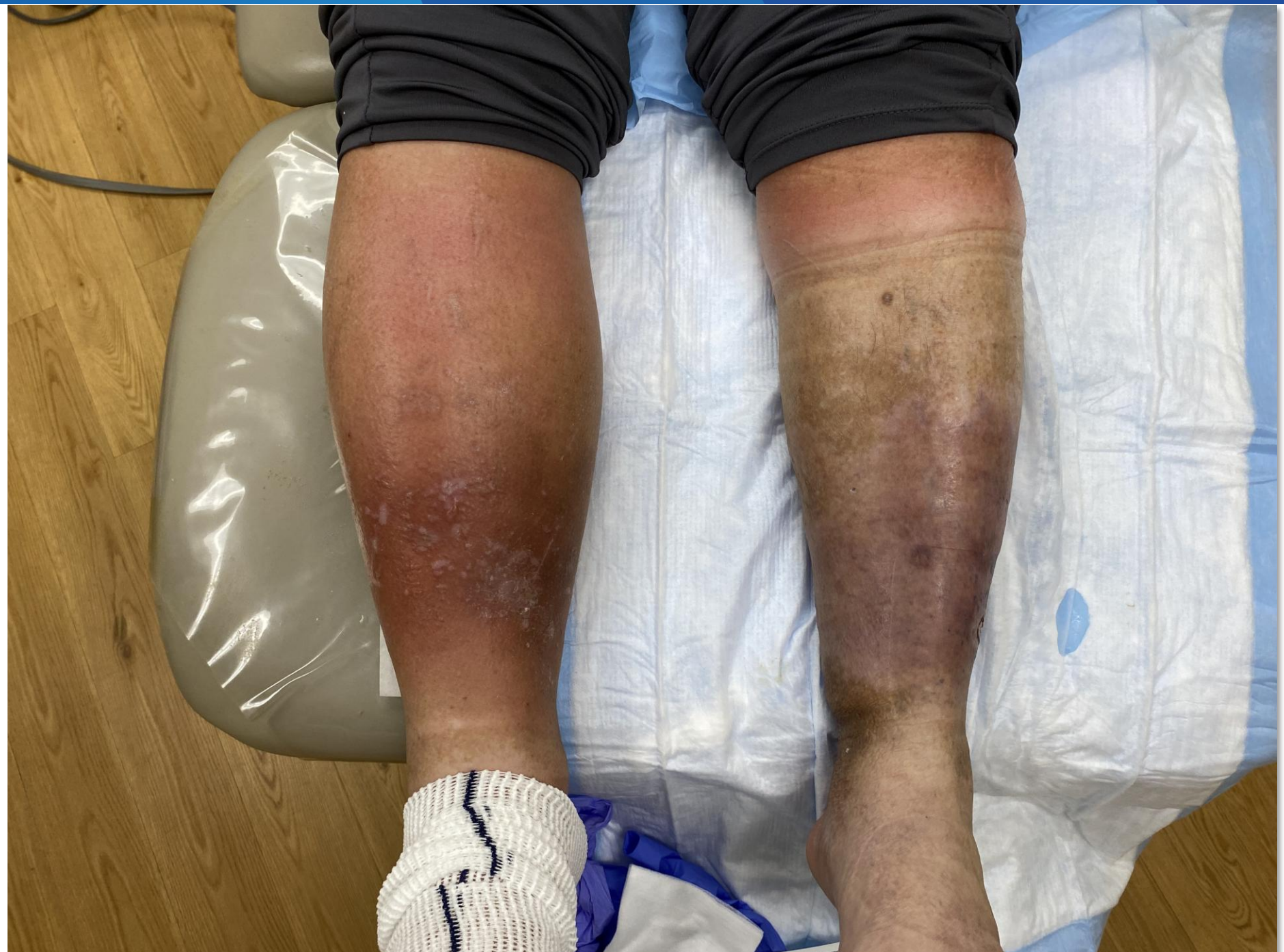
- Application of external pressure counteracts loss of capillary fluid by increasing local tissue pressure
- Reduces diameter of major leg veins by:
  - Reducing local blood volume by squeezing fluid into veins and lymph vessels
  - Redistributing blood towards central parts of the body to enhance venous return



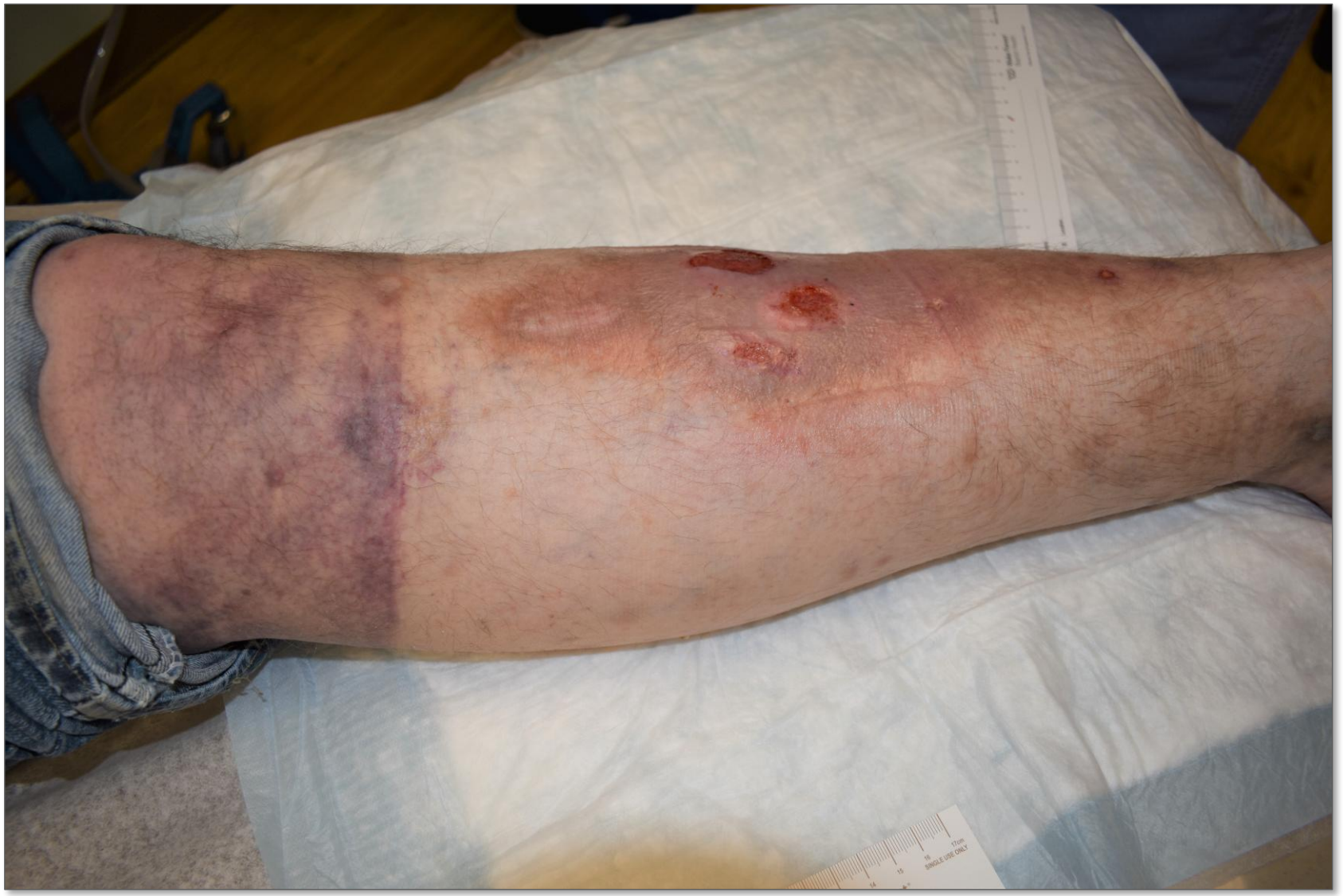
European Wound Management Association (EWMA). Understanding Compression Therapy. 1st ed. Medical Education Partnership; 2003.

Congested vs Decongested Leg

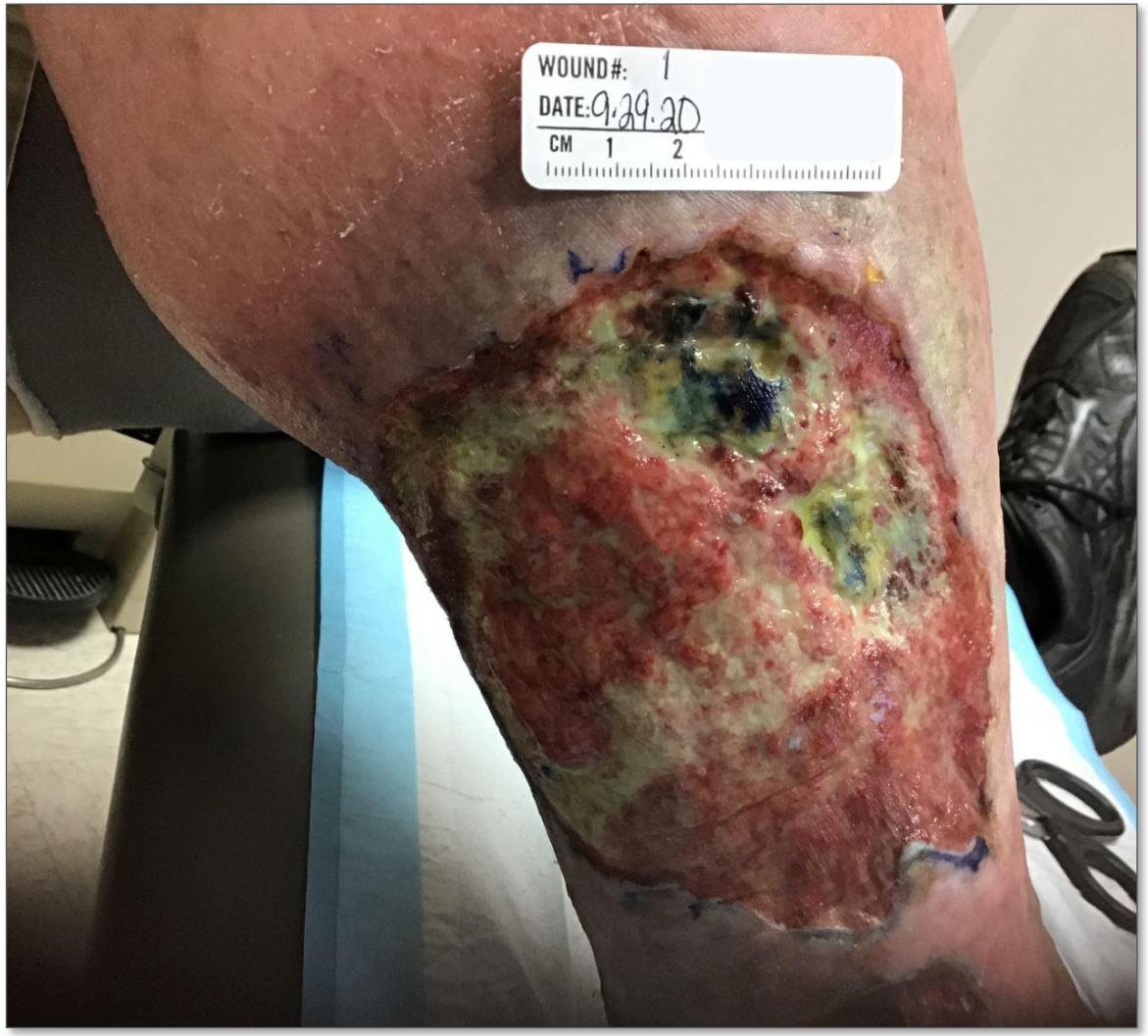


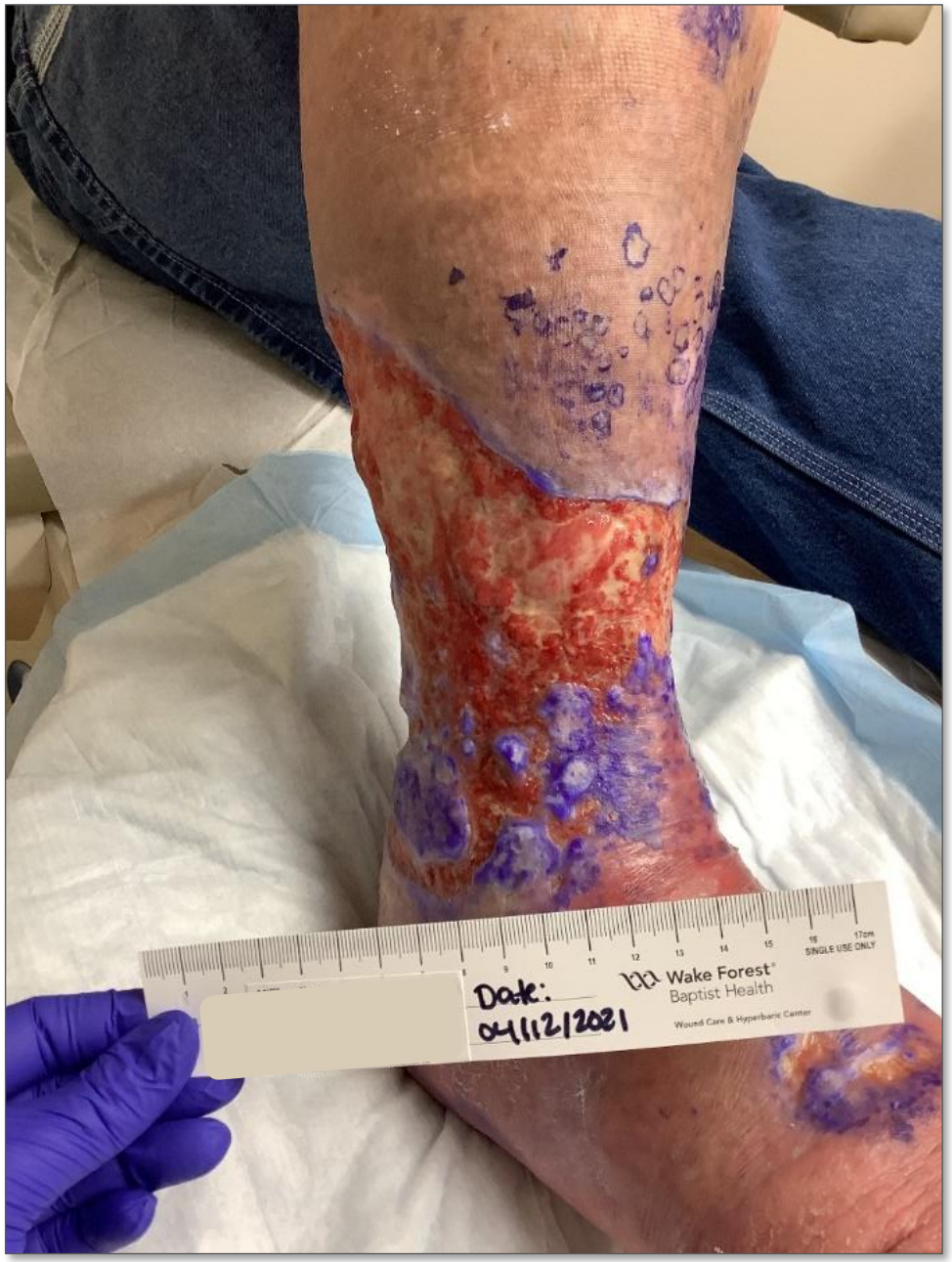


The “Aha!” Moment

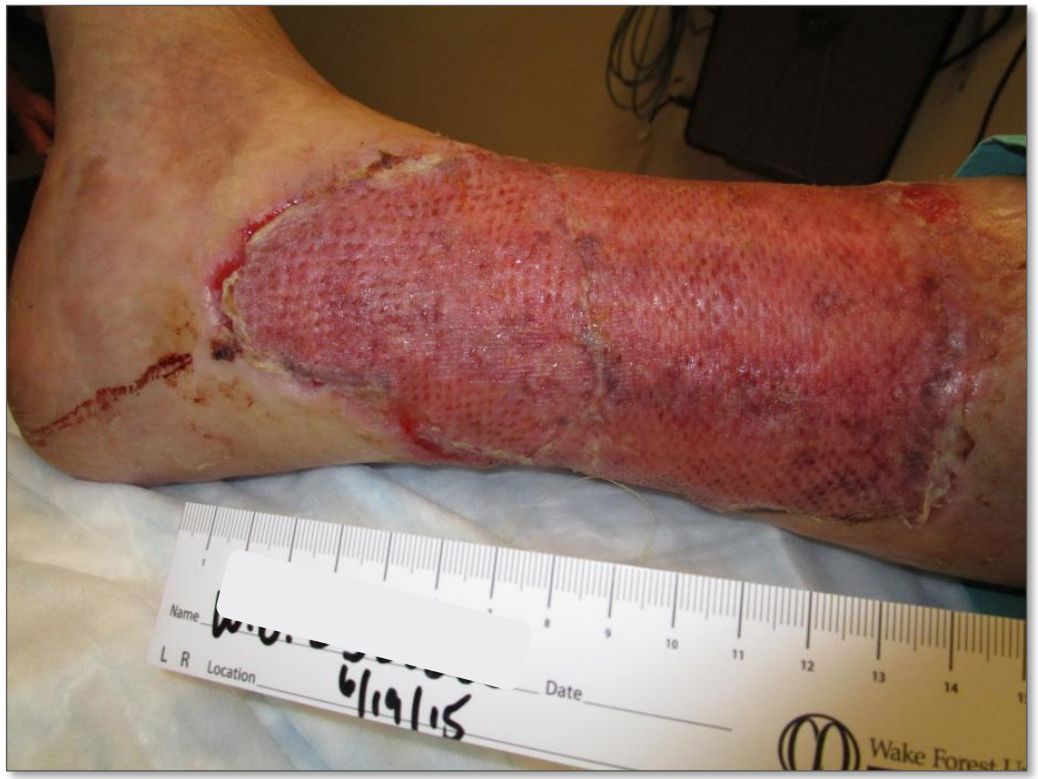


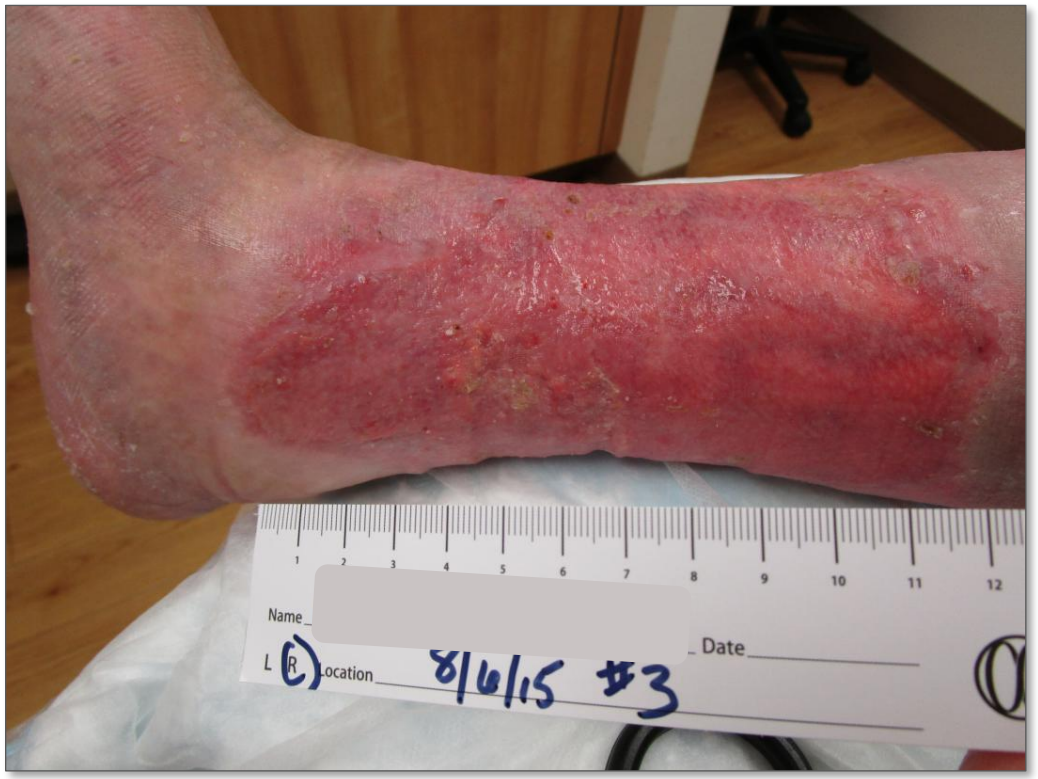
Pyoderma Gangrenosum Improves with Compression

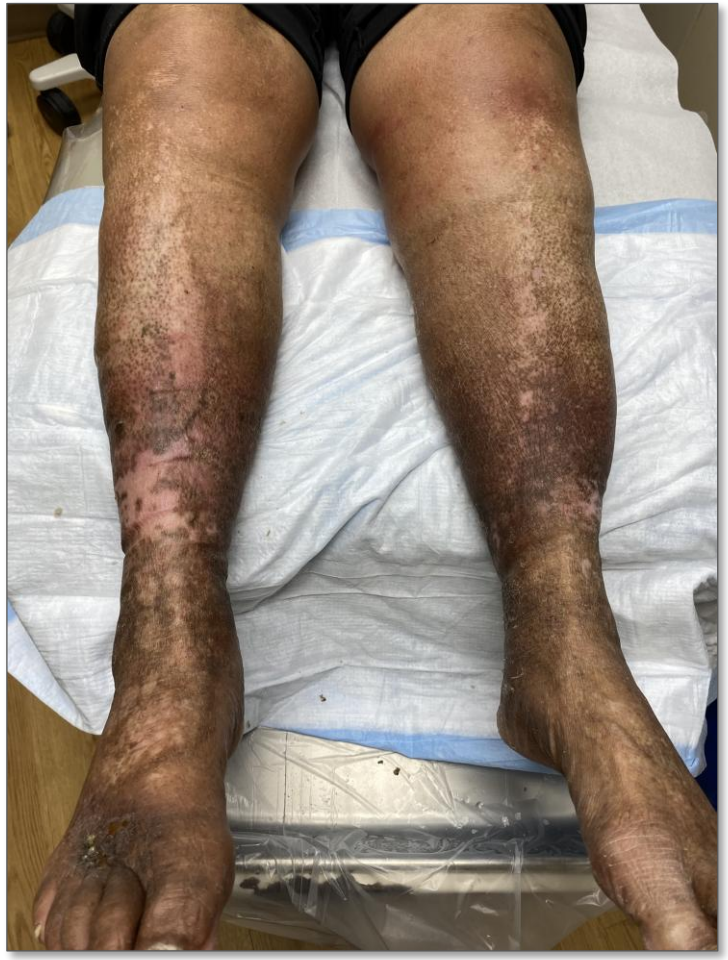


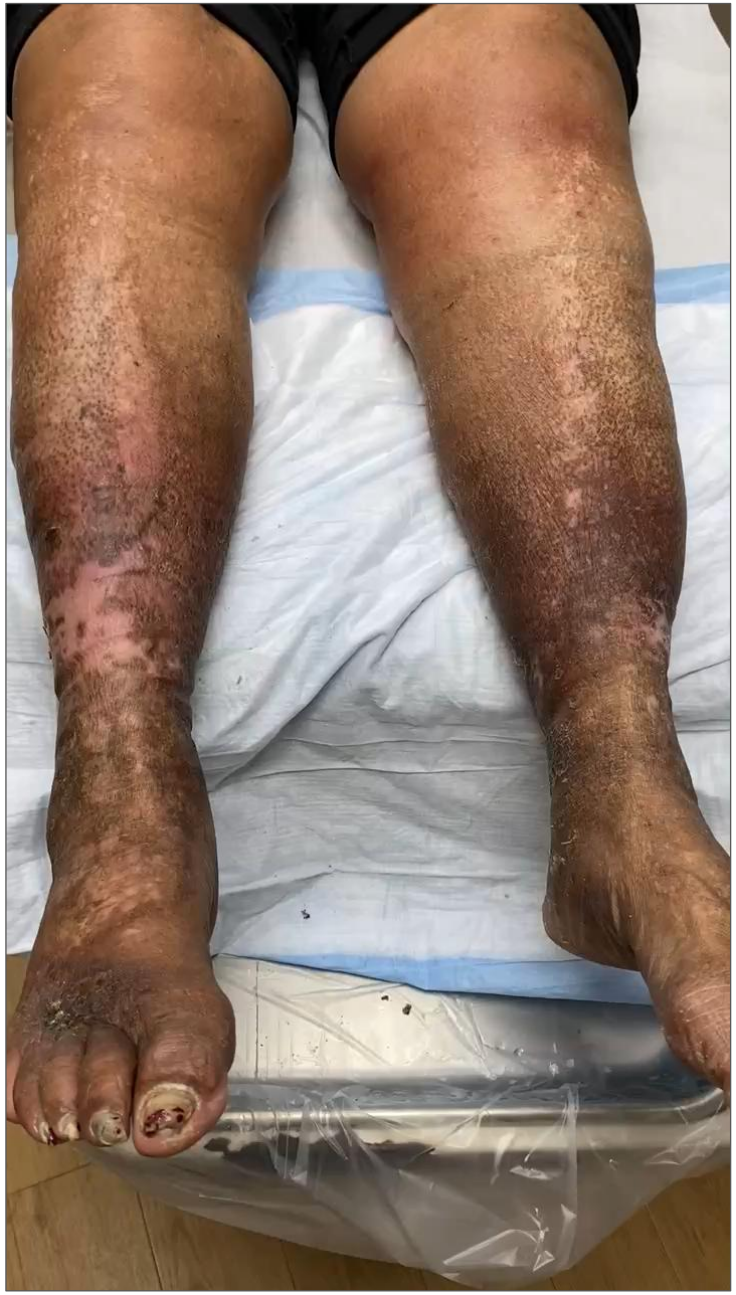


A congested leg cannot take a split-thickness skin graft









Thank You

Submit Questions via the Question Box