Lower Extremity Training Camp: Best Practice Tips for Wounds, Bioburden, Edema, Biomechanics and More

Desert Foot 2019
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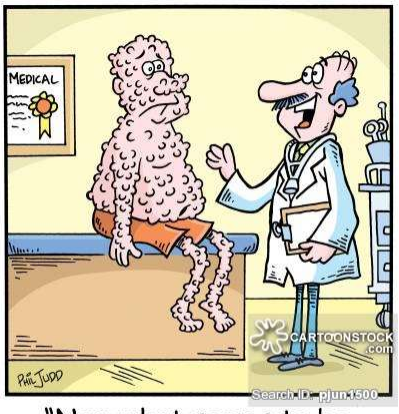
Basic Skin and Wound Assessment

Heather Hettrick

Comprehensive Integument Assessment



"Now what seems to be the problem?"

Faculty:

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Disclosures

Professor at Nova Southeastern University;

KOL- 3M;

Consultant- Cell Constructs;

Advisory Board- Molnlycke;

SME- Healiant;

Faculty/Director of Wound Education- ILWTI

Objectives

At the end of this presentation participants should be able to:

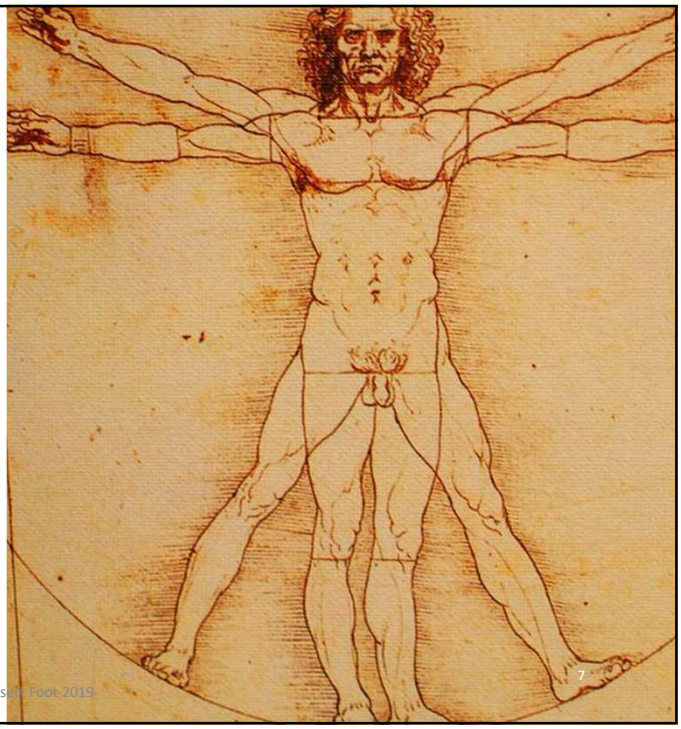
- Recognize normal versus abnormal dermatological variants
- Describe the components of comprehensive skin assessment
- Appreciate the unique presentations of skin conditions with CVI, AI and lymphedema
- Understand the basic elements of wound assessment

Integument Assessment

For effective wound management, one must have:

- Sound clinical knowledge of integumentary anatomy and physiology
- Understand changes associated with aging skin
- Identify threats to the skin (endogenous, exogenous, iatrogenic)
- Recognize patients'/residents' comorbidities and overall health status

- Thorough skin assessment is paramount
- Early intervention is critical
- *Prevention* is key



Skin Assessment Components

From Hettrick H, In Myers B. Wound Management Principles and Practice, 3rd Edition, Pearson, 2012.

Describe integrity
Ethnicity
Review sensory status
Moisture
Atrophic changes
Turgor/texture
Observe nail composition/hair quality
Look/feel edema, color and temperature variations
Observe skin folds
Gerontodermatological changes
Inquire about allergies and PMH
Callus
Assess vascular status
Lesions

"After all these years, I'm finally comfortable in my own skin. Maybe it's because my skin is a lot bigger than it used to be!"

Skin Assessment Components

_D_: describe integrity

- Skin is intact or presents with impairment
  - Describe (derm terms)
  - Classify (etiology)

_E_: ethnicity

- Note skin tone and dermatological variants
  - Caucasian
  - Asian
  - African American
  - Latino/Hispanic
  - Native American
  - Pacific Islander
  - Other
- Fitzpatrick Scale

Skin Assessment Components

R: review sensory status

- Intact or altered
  - Location
  - Specific tests
  - Soft tissue status

M: moisture

- Supple or slightly moist to touch
  - Dry/cracked
  - Wet/macerated

A: atrophic changes

- Shiny, hairless extremities
  - Recommend vascular consult

Skin Assessment Components

"It's not a rash, it's moss. You need to start being more active than a tree."

T: turgor/texture

- Turgor
- Texture
  - Normal
  - Dry, cracked, hyperkeratotic
  - Watery
  - Softly pitting
  - Brawny/fibrotic
  - Hard/non-compressible

O: observe nail composition and hair quality

- Both are extensions of the skin

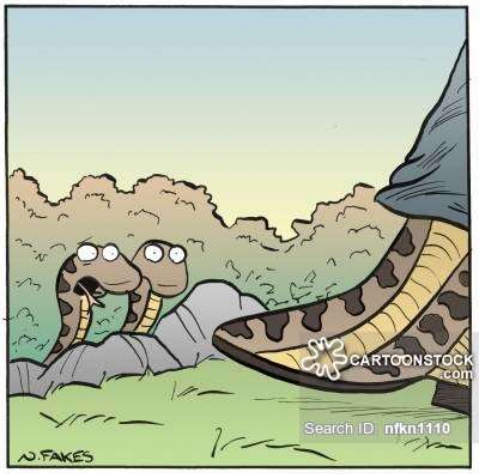
Skin Assessment Components

L: look/feel for edema, color and temperature variations

- Edema
  - Location
  - Pitting or non-pitting
- Color
  - Tone
  - Pigmentation
- Temperature

O: observe skin folds

- Look for moisture lesions
- Pressure necrosis
- Yeast/fungal infections
- Foreign objects



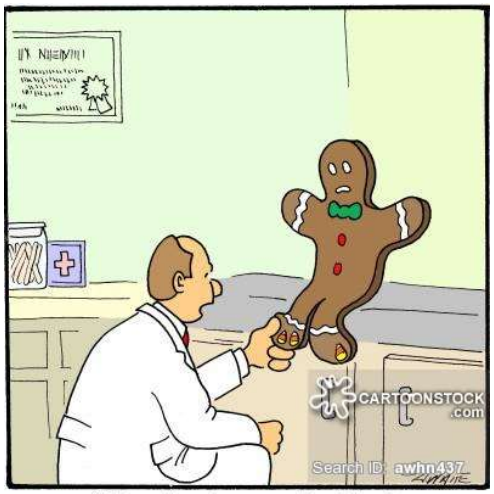
"It's the walking dead!"

Skin Assessment Components

<u>*G*: gerontodermatological changes</u>

- Normal skin changes with aging
- Risks: skin integrity disruption from minor trauma
  - Bruising
  - Skin tears
  - Ulceration

Skin Assessment Components



"There's why your feet hurt: You have candy corns!"

<u>I: inquire about allergies and past medical history</u>

- Are findings exogenous or endogenous in nature

<u>C: callus</u>

- Indicates area(s) of high pressure or repetitive stress/trauma
- Hemorrhagic is indicative of ulceration

"Thumpity-thump-thump, thumpity-thump-thump...."

Skin Assessment Components

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***A*: assess vascular status**

- Look, listen, feel
  - Color changes
  - Doppler
  - Perfusion monitoring
  - Palpate pulses, capillary refill, ABI

***L*: lesions**

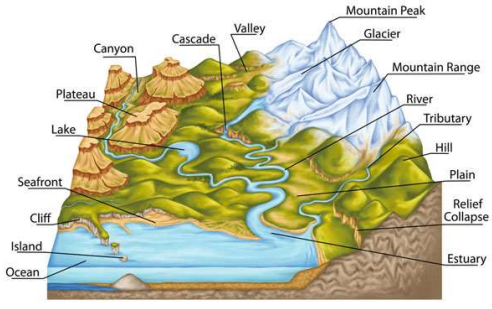
- Document location(s), describe presentation, formulate working clinical diagnosis
- Denote anything unusual or suspicious
- Identify unique/characteristic skin presentations related to disease processes

Wound Geography

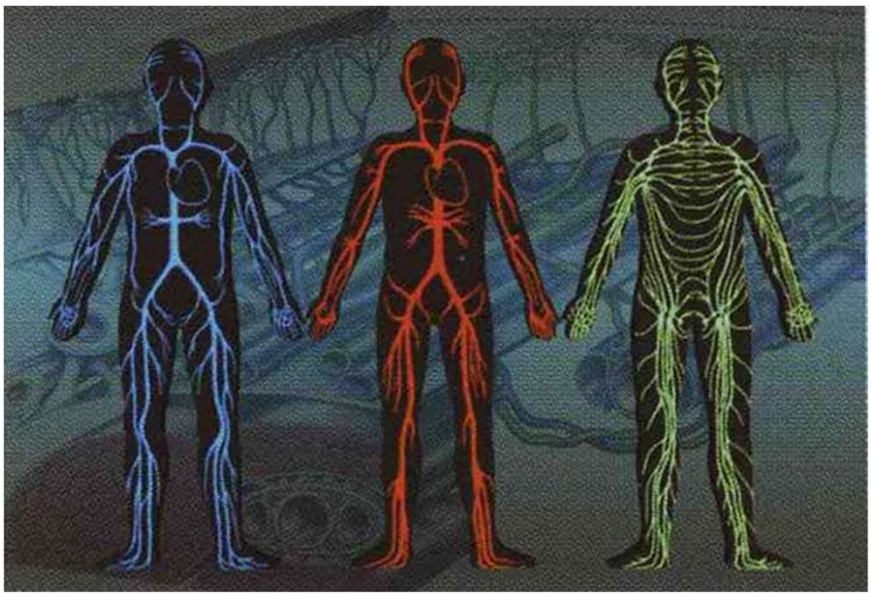
Location!!

Wound Topography

Characteristics!!



Clues to assist with differential diagnosis



Venous Arterial Lymphatic

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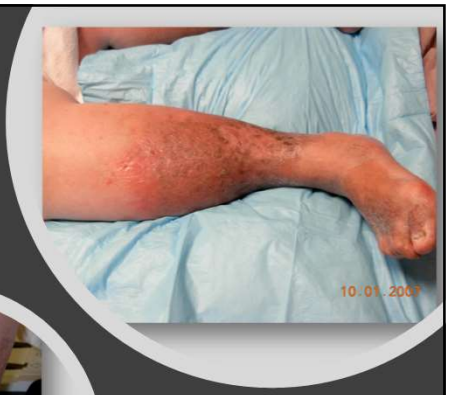
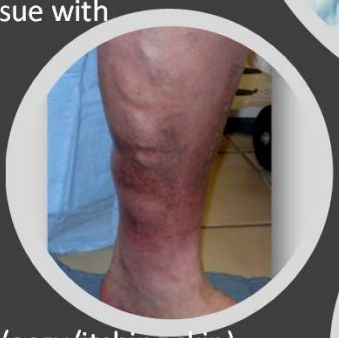
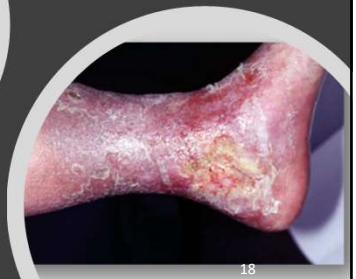
Common Venous Skin Changes

- Varicose veins
- Spider veins/telangiectasia
- Medial ankle flare (spider veins at medial ankle/foot)
- Hemosiderin staining
- Atrophie blanche (white patches of scar tissue with red dilated capillary loops)
- Shiny, taut skin
- Brawny or leathery skin
- Venous rubor (chronic inflammation)
- Lipodermatosclerosis (fibrosis replaces the adipose layer at the ankle)
- Livedo reticularis (mottled discoloration)
- Stasis dermatitis (red/scaly/crusty/cracked/oozy/itching skin)
- Recurrent cellulitis
- Ulceration







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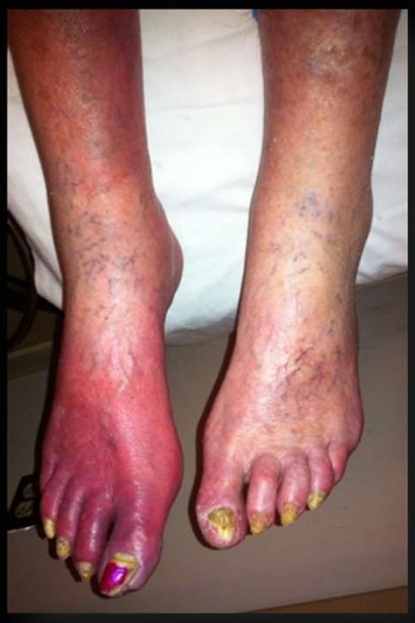

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Common Arterial Skin Changes

- Cool, pale, dry skin
- Mottled and/or cyanotic skin
- Thin, brittle shiny skin (legs and feet)
- Dependent rubor with pallor on elevation
- Diminished pulses
- Lower extremity hair loss
- Thickened or dystrophic nails
- Distal ulceration to gangrene
- Pain at rest (burning, aching)

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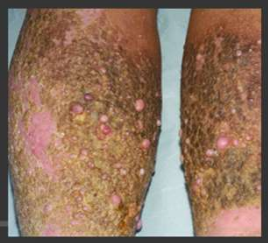
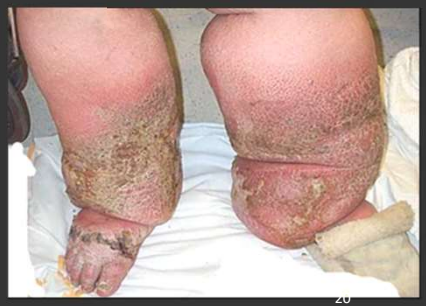
Common Lymphatic Skin Changes

- Thickened, fibrotic skin
- Hyperpigmentation, lymphatic rubor
- Hyperkeratosis (scaly brown or grey patches of over-proliferated keratin layers)
- Papillomatosis or verrucous skin (lumpy, bumpy skin or fibrotic wart-like projections of the skin)
- Rebound > 30 seconds
- Tissue folds/rubber band effect
- Dorsal foot swelling
- Squaring of toes or fingers
- Up-sloped 'ski jump' nail changes
- Positive Stemmer Sign





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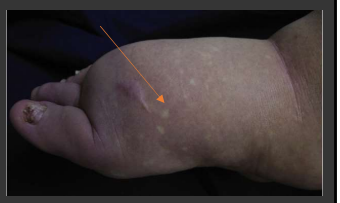
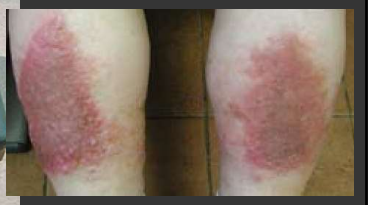
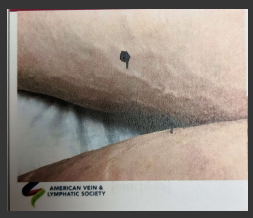
Other Lymphatic Skin Changes

- Licenification
- Cobblestoning
- Lymphorrhea
- Scleroindurative pachydermatitis
- Bier spots (physiologic anemic macules)
- Rivulets (dermal capillary volume excess due to heart failure)
- Acroangiodermatitis
- Carcinogenic changes (verrucous carcinoma, cutaneous horn/SCC, Stewart Treves Syndrome)







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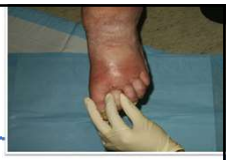
Tissue Texture and Turgor



Texture

- Normal- elastic, supple, pliable
- Watery- palpable pockets of fluid, translucent skin, can manually displace fluid
- Softly pitting- soft, boggy, feels like dough
- Brawny/fibrotic- denser connective tissue, poorly pitting (~>30 sec), skin thickened difficult to pinch/tent
  - less fibrotic = feels like tube of toothpaste
  - more fibrotic = firm, leathery
- Hard/non-compressible- advanced lymphedema, non-pitting, crusty like tree bark or alligator skin, cannot be pinched, not pliable

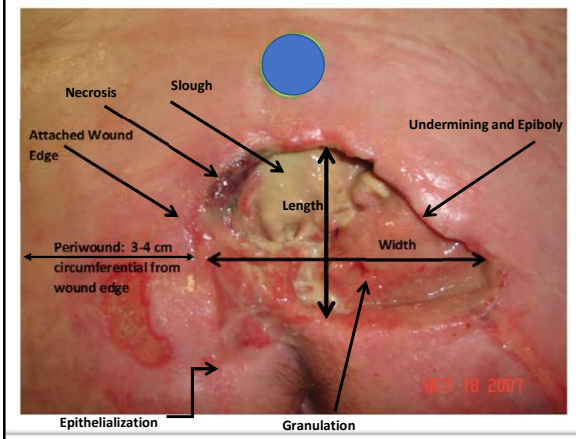
Turgor

- Pitting and Rebound
  - Grade according to depth and time to rebound
  - Rebound > 30 secs indicative of lymphedema
- Non-pitting edema
  - Taut, extremely distended extremity
  - Lymphedema with fibrotic changes
- Stemmer Sign
  - Thickening of skin over proximal phalanges of toes or fingers
    - (+) indicates lymphedema
    - (-) does not exclude lymphedema
      - may be early in disease process and fibrosis has not developed; monitor and if patient does not respond to conventional edema management, it is likely lymphedema

Comprehensive Wound Assessment

This mnemonic will help you to remember the key items needed to review and document comprehensive wound assessment.

Adapted from Baranoski & Ayello Nursing 2005



A-	Anatomic location and age of wound
S-	Size, shape or stage (if pressure)
S-	Sinus tracts and undermining
E-	Exudate (amount, color, consistency)
S-	Sepsis (signs of local or systemic infection)
S-	Surrounding skin (periwound tissue integrity)
M-	Maceration
E-	Edges and epithelialization
N-	Necrotic tissue
T-	Tissue bed (describe tissue types present, quantify amount)
S-	Status (describe plan of care based upon clinical wound presentation)

Deser

Be *Sensible* With Your Integ/Wound Exam

- Use your senses to help with differential diagnosis
  - Visual inspection alone is not sufficient
- Visual identification of skin changes unique to different conditions (venous, arterial and lymphatic) will assist with differential diagnosis
- Touch and palpate the skin for texture and turgor
- Listen to your patients; they will often tell you what is wrong if you pay attention to the clues

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Wound Examination

Pay attention to the clues…

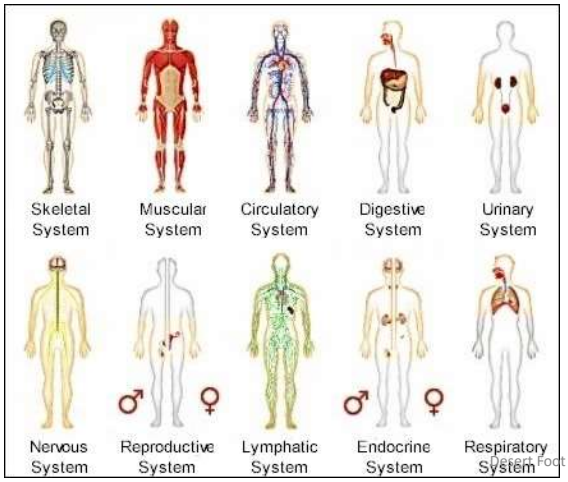
- General appearance of the patient
- Appearance of the extremity/extremities/body region
- Location of the wound (*geography*)
- Appearance of the wound (*topography*)
  - Base
  - Edge
  - Periwound
- Surrounding skin (*topography*)

Look at the WHOLE patient
Not just the HOLE in the patient
- Carrie Sussman, PT, DPT, CWS

Wound Examination

History taking still remains the best and cheapest tool to make a good clinical diagnosis

- Be thorough and review all body systems



Consider how the other systems impact the integumentary system and vice versa?

Wound Examination

During history, note:

- Medications/vitamins
  - steroids
  - immunosuppressive agents
  - heparin/coumadin
  - vitamin supplements
  - herbal supplements
- Allergies
  - latex
  - fiberglass
  - sulfur
  - medications
  - bovine (cow/collagen)
  - food
  - adhesives

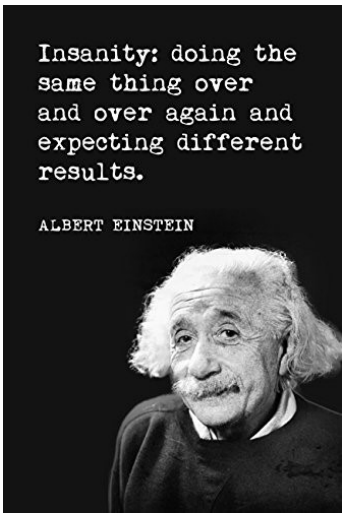
Wound Examination

Social history

- Smoking
- Alcohol
- Drug abuse
- Nutrition/hydration
- Social support
- Work/school/community environment

Previous treatment(s) and results*

- *Has the patient had a wound before?
  - What type (if known)?
  - What interventions were provided?
  - Were they successful?



*You do not want to implement the same treatments if they did not help the patient initially

Wound Examination

Subjective examination

- Ask the patient…
  - What are symptoms?
    - Allow patient to describe in own words
  - Where are symptoms located?
  - How do symptoms behave?
    - What make the symptoms better/worse?
  - How and when did this start?
    - Unknown, rapid, gradual?
    - Acute vs. chronic
- Be thorough with pain assessment
  - Use visual analog scale; try to objectify pain

 Pain is whatever it is to the person experiencing it

Wound Examination- Clinical Measurements

Objective wound examination components

- wound location(s)
- etiology or mechanism of injury
- onset
- size (Length x Width x Depth; undermining, tunneling)
- wound bed (color, quality, quantity of tissue)
- wound margins/edges
- periwound integrity
- exudate quality, quantity, odor
- presence of foreign objects
- edema/extremity girth measurements
- tissue temperature
- loss of protective sensation
- vascular exam

Consider yourself a Wound Scene Investigator- Look for clues during your exam to determine clinical diagnosis and intervention planning or referral

Wound Classification

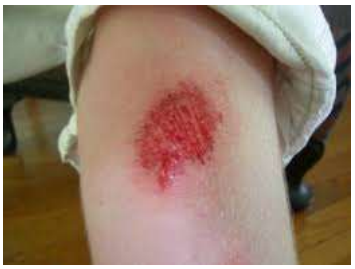
Various methods exist in order to classify wounds; often dependent upon etiology

- Classification by **Depth**
- Classification by **Staging/Category**
- Wagner Ulcer Grade Classification
- CEAP for Venous Ulcer Classification
- Burn Classification

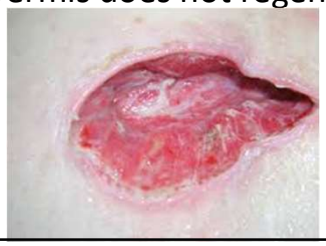
Wound Classification: Depth

Used to classify wounds whose primary cause is something *other* than pressure

Partial-thickness- limited to epidermis and upper portion of dermis. Heals by regeneration. No scar tissue. Healing complete 7-14 days.

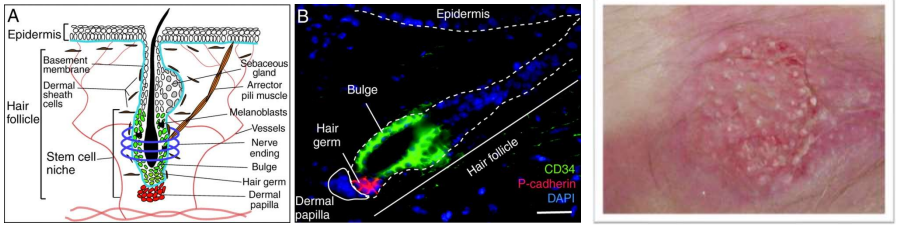


Full thickness- wounds that involve total loss or destruction of the epidermis and dermis, as well as subcutaneous tissue. May involve deep tissue structures. Tissue heals by scar formation (granulation, contraction, epithelialization). May take 3 weeks or longer. Dermis does not regenerate.



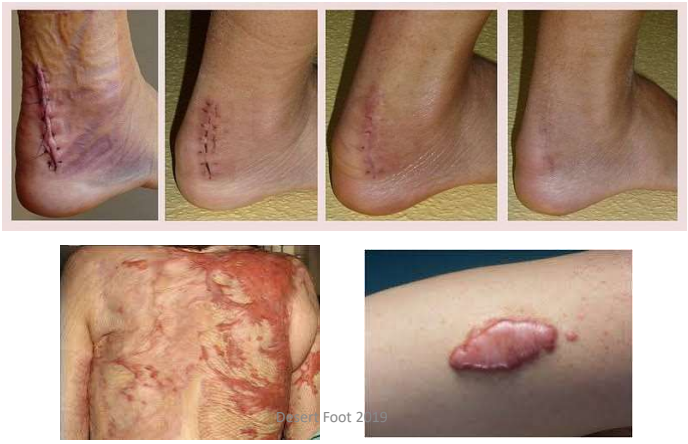
Regeneration

In <u>partial-thickness wounds</u>, where sweat glands and hair follicles remain in the wound bed, islands of new epidermis (epithelial buds) may form around these epidermal appendages to facilitate re-epithelialization



Repair

In <u>full-thickness wounds</u>, the dermis will not regenerate and nerve endings and epidermal appendages do not reproduce…the result is scar tissue without the qualities of uninjured skin



What is the Difference?

Partial Thickness	Full Thickness

• Only involves epidermis and part of the dermis
  • Papillary layer
• Regenerate
• No scar tissue
	• Involves epidermis and dermis (or beyond)
	  • Papillary and reticular dermal layers
	  • May extend into deep tissue structures
	• Repair
	• Develop scar tissue to replace lost tissue

Rete Ridges and Dermal Layers- The Skin's Continental Divide

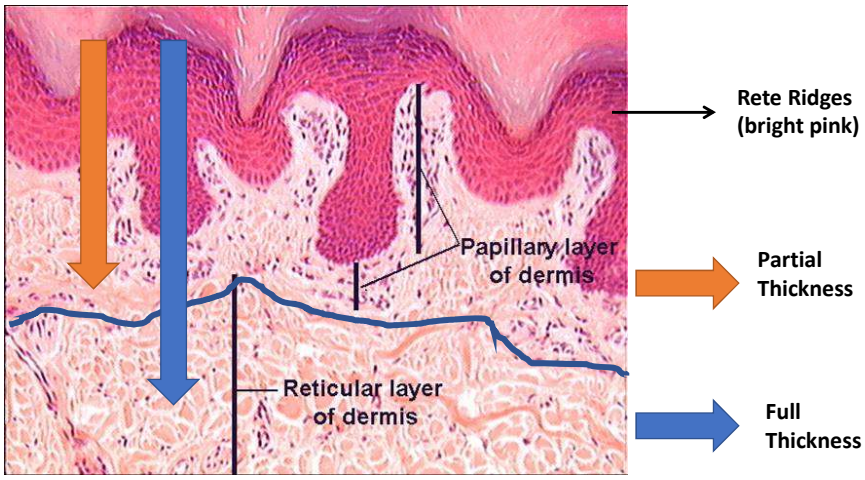
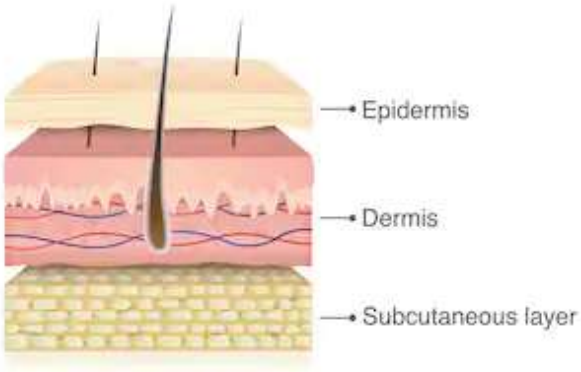


Photo: www.neuromedia.neurobio.ucla.edu



Dermal Distinctions…

- Two layers: Papillary & Reticular
- Houses epidermal appendages
- Vascular
- Cannot regenerate
- When damaged it cannot replace lost tissues or structures; it fills in with granulation tissue (raw scar tissue)
- Over time, scar replaces lost tissues
- Color normalizes due to capillary regression and re-pigmentation

The Skinny on the Skin

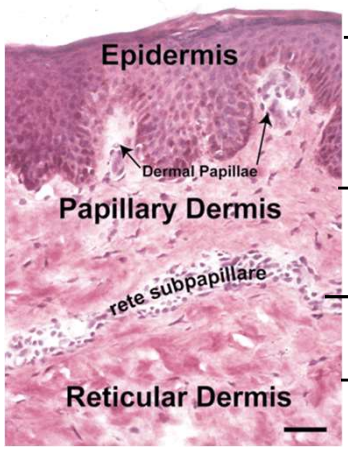
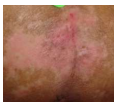
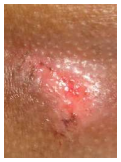


Photo: biology-online.org

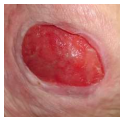


New Epithelium-
Yikes, my epidermis is showing!



Partial Thickness-
Ouch! I skinned my knee.

Separation of papillary and reticular dermal layers



Full Thickness-
Uh oh…better go to the doctor.

Wound Classification: Staging/Category

Classification by Staging/Category

- Identify wounds by the tissue layer involved. Anatomic description of wound depth.
- NPIAP (National Pressure Injury Advisory Panel) created the staging system for pressure ulcers
- Updated 2016
  - From pressure ulcer to pressure injury
  - Staging system definitions were revised
- *NPIAP staging system is only for pressure ulcers/injuries and should not be used to describe other wound types*

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NPIAP Pressure Injury Definition

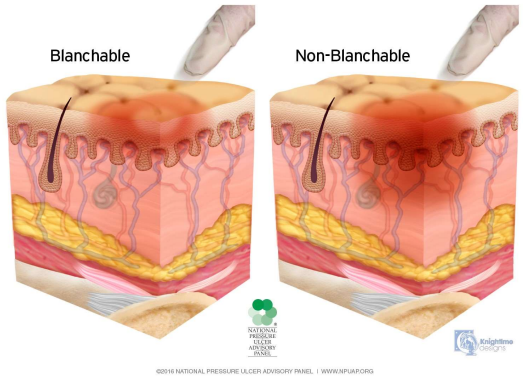
www.nplap.org

A pressure injury is localized damage to the skin and/or underlying soft tissue usually over a bony prominence or related to a medical or other device. The injury can present as intact skin or an open ulcer and may be painful. The injury occurs as a result of intense and/or prolonged pressure or pressure in combination with shear. The tolerance of soft tissue for pressure and shear may also be affected by microclimate, nutrition, perfusion, co-morbidities and condition of the soft tissue.

NPIAP Pressure Injury Stages

Stage 1 Pressure Injury: Non-blanchable erythema of intact skin

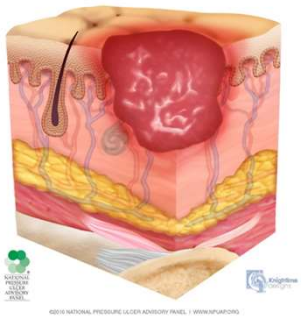
- Intact skin with a localized area of non-blanchable erythema, which may appear differently in darkly pigmented skin. Presence of blanchable erythema or changes in sensation, temperature, or firmness may precede visual changes. Color changes do not include purple or maroon discoloration; these may indicate deep tissue pressure injury.



NPIAP Pressure Injury Stages

Stage 2 Pressure Injury: Partial-thickness skin loss with exposed dermis

- Partial-thickness loss of skin with exposed dermis. The wound bed is viable, pink or red, moist, and may also present as an intact or ruptured serum-filled blister. Adipose (fat) is not visible and deeper tissues are not visible. Granulation tissue, slough and eschar are not present. These injuries commonly result from adverse microclimate and shear in the skin over the pelvis and shear in the heel. This stage should not be used to describe moisture associated skin damage (MASD) including incontinence associated dermatitis (IAD), intertriginous dermatitis (ITD), medical adhesive related skin injury (MARSI), or traumatic wounds (skin tears, burns, abrasions).

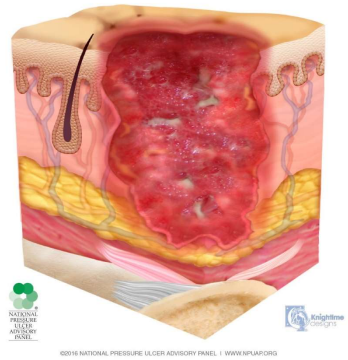


NPIAP Pressure Injury Stages

Stage 3 Pressure Injury:

Full-thickness skin loss

- Full-thickness loss of skin, in which adipose (fat) is visible in the ulcer and granulation tissue and epibole (rolled wound edges) are often present. Slough and/or eschar may be visible. The depth of tissue damage varies by anatomical location; areas of significant adiposity can develop deep wounds. Undermining and tunneling may occur. Fascia, muscle, tendon, ligament, cartilage and/or bone are not exposed. If slough or eschar obscures the extent of tissue loss this is an Unstageable Pressure Injury.

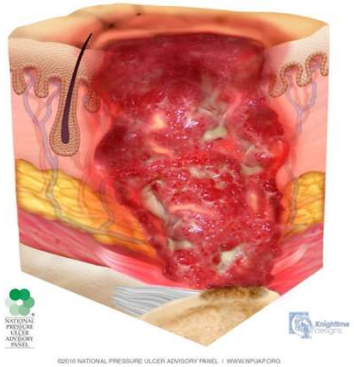


NPIAP Pressure Injury Stages

Stage 4 Pressure Injury:

Full-thickness skin and tissue loss

- Full-thickness skin and tissue loss with exposed or directly palpable fascia, muscle, tendon, ligament, cartilage or bone in the ulcer. Slough and/or eschar may be visible. Epibole (rolled edges), undermining and/or tunneling often occur. Depth varies by anatomical location. If slough or eschar obscures the extent of tissue loss this is an Unstageable Pressure Injury.



NPIAP Pressure Injury Stages

Unstageable Pressure Injury:

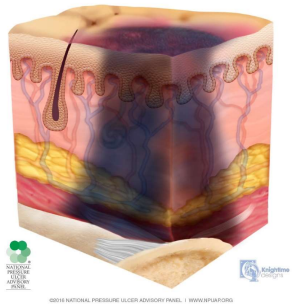
Obscured full-thickness skin and tissue loss

- Full-thickness skin and tissue loss in which the extent of tissue damage within the ulcer cannot be confirmed because it is obscured by slough or eschar. If slough or eschar is removed, a Stage 3 or Stage 4 pressure injury will be revealed. Stable eschar (i.e. dry, adherent, intact without erythema or fluctuance) on the heel or ischemic limb should not be softened or removed.

NPIAP Pressure Injury Stages

Deep Tissue Pressure Injury:

Persistent non-blanchable deep red, maroon or purple discoloration



- Intact or non-intact skin with localized area of persistent non-blanchable deep red, maroon, purple discoloration or epidermal separation revealing a dark wound bed or blood filled blister. Pain and temperature change often precede skin color changes. Discoloration may appear differently in darkly pigmented skin. This injury results from intense and/or prolonged pressure and shear forces at the bone-muscle interface. The wound may evolve rapidly to reveal the actual extent of tissue injury, or may resolve without tissue loss. If necrotic tissue, subcutaneous tissue, granulation tissue, fascia, muscle or other underlying structures are visible, this indicates a full thickness pressure injury (Unstageable, Stage 3 or Stage 4). Do not use DTPI to describe vascular, traumatic, neuropathic, or dermatologic conditions.

NPIAP Pressure Injury Stages

Medical Device Related Pressure Injury:

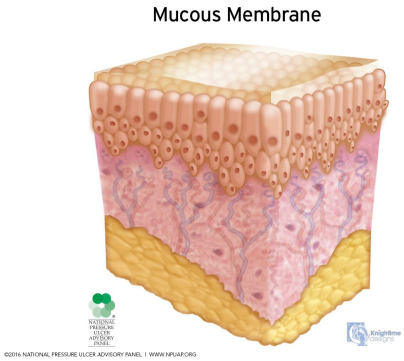
This describes an etiology

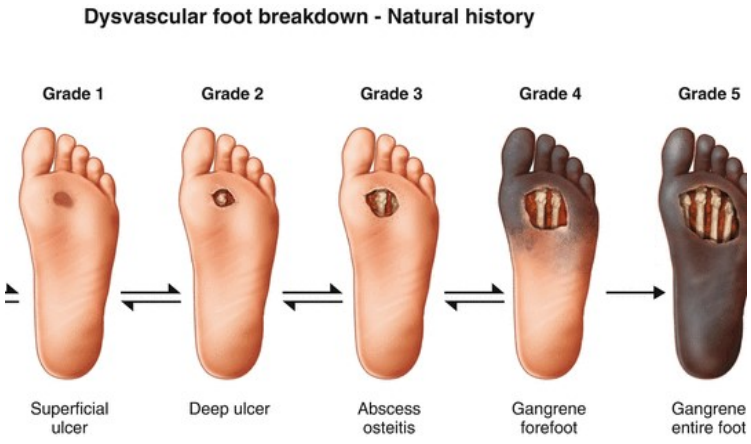
- Medical device related pressure injuries result from the use of devices designed and applied for diagnostic or therapeutic purposes. The resultant pressure injury generally conforms to the pattern or shape of the device. The injury should be staged using the staging system.

NPIAP Pressure Injury Stages

Mucosal Membrane Pressure Injury:

- Found on mucous membranes with a history of a medical device in use at the location of the injury. Due to the anatomy of the tissue these ulcers cannot be staged.
- Locations: Tongue, GI, nasal passage, urinary tract, etc.
- Caused by: Devices, tubing, ET tubes, bite blocks, catheters, etc.
- Characteristics: injury causes bleeding and soft clot (coagulum); appears shiny and not to be confused with slough
- These are not staged nor described as PT or FT as mucosal tissue is different





Wound Classification: Wagner Scale

Wagner Ulcer Grade Classification

- Developed for the diagnosis and treatment of the dysvascular foot.
- Used to establish the presence of depth and infection.
- Six grades of severity:
  - 0: preulcer lesion, healed ulcer, presence of bony deformity
  - 1: superficial ulcer without subcutaneous tissue involvement
  - 2: penetration through subcutaneous tissue; may expose bone, tendon, ligament, or joint capsule
  - 3: osteitis, abscess, or osteomyelitis
  - 4: gangrene of a digit
  - 5: gangrene requiring foot amputation

Wound Classification: CEAP

Classification for Chronic Venous Disorders

- ***C***linical classification
- ***E***tiologic classification
- ***A***natomic classification
- ***P***athophysiologic classification

Wound Classification: CEAP

Classification for Chronic Venous Disorders

Clinical classification

- C0: no visible or palpable signs of venous disease
- C1: telangiectasies or reticular veins
- C2: varicose veins
- C3: edema
- C4a: pigmentation or eczema
- C4b: lipodermatosclerosis or atrophie blanche
- C5: healed venous ulcer
- C6: active venous ulcer
- S: symptomatic, including ache, pain, tightness, skin irritation, heaviness, and muscle cramps, and other complaints attributable to venous dysfunction
- A: asymptomatic

Wound Classification: CEAP

Classification for Chronic Venous Disorders

- *C*linical classification
- ***E***tiologic classification
  - Ec: congenital
  - Ep: primary
  - Es: secondary (postthrombotic)
  - En: no venous cause identified

Wound Classification: CEAP

Classification for Chronic Venous Disorders

- *C*linical classification
- *E*tiologic classification
- *A*natomic classification
  - As: superficial veins
  - Ap: perforator veins
  - Ad: deep veins
  - An: no venous location identified

Wound Classification: CEAP

Classification for Chronic Venous Disorders

- *C*linical classification
- *E*tiologic classification
- *A*natomic classification
- *P*athophysiologic classification
  - Pr: reflux
  - Po: obstruction
  - Pr,o: reflux and obstruction
  - Pn: no venous pathophysiology identifiable

Wound Classification: Burns

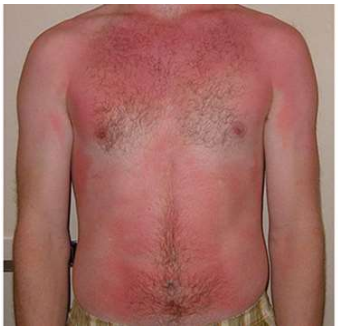
Burn Classifications

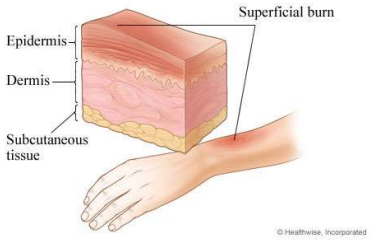
- Traditional
  - First, Second, Third degree
- American Burn Association (ABA) classification
  - Current standard classification
    - Superficial
    - Partial-thickness
      - superficial partial
      - deep partial
    - Full-thickness

Wound Classification: Burns

	Cause	**Appearance**	**Color**	**Sensation**	**Histologic depth**	**Healing**
Superficial	Sunburn	Dry	Pink	Painful	Epidermis	3-7 days
Partial thickness -superficial	Scald Short exposure	Blisters, moist	Red	Very painful	Epidermis and papillary dermis	7-21 days
Partial thickness -deep	Immersion scald, flame	Large blisters, moist, wet	Mottled white, pink to red	Very painful	Epidermis and reticular dermis	21-35 days if no infection; may convert to full thickness
Full thickness	Flame, scald, electrical, chemical	Dry, leathery, nonblanching	black, mixed white, pearly, dark khaki, charred	Little to no pain	Epidermis and all dermis; deep structures may be involved	Requires skin grafting

Wound Classification: Burns

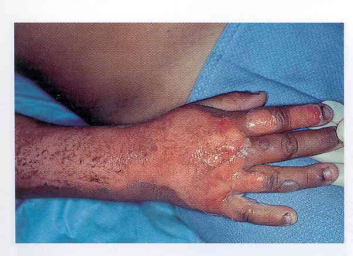


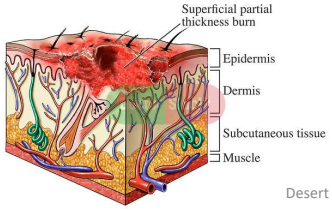


Superficial Burn

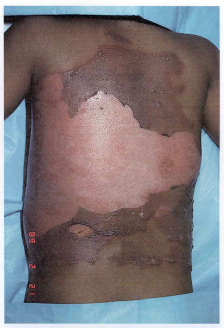
Wound Classification: Burns

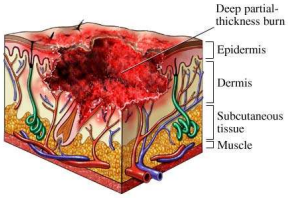
Superficial Partial Thickness Burn





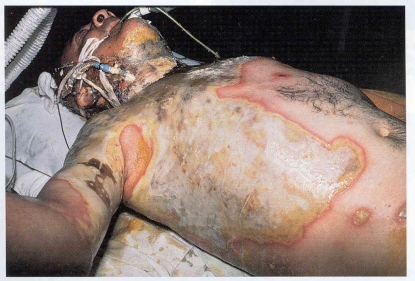
Deep Partial Thickness Burn

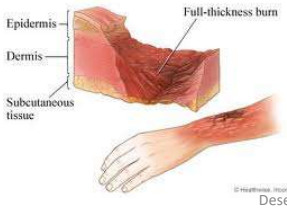


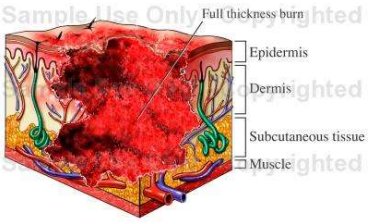


Wound Classification: Burns

Full Thickness Burns







The Missing Link: The Lymphatic System

Heather Hettrick

Disclosures

Professor at Nova Southeastern University;

KOL- 3M;

Consultant- Cell Constructs;

Advisory Board- Molnlycke;

SME- Healiant;

Faculty/Director of Wound Education- ILWTI

Objectives

At the end of this presentation participants should be able to:

- Recognize the role and importance of the lymphatic system with respect to homeostasis
- Relate the impact of lymphatic pathophysiology on the body systems
- Describe the lymphedema continuum and the new understanding of fluid hemodynamics
- Verbalize the inter-relationship and inter-dependence of the VAIL (venous, arterial, integument, lymphatic) systems

What do these diseases have in common?

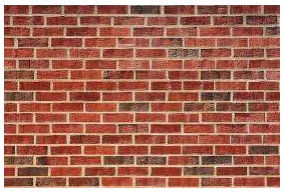
David Zawieja PhD

- Lymphedema
- Lymphatic vascular malformations
- Visceral lymphatic diseases
- GI infections and Clostridium difficile colitis
- Peritonitis
- Cancer and metastasis
- Chronic infections and inflammation
- Organ transplantation
- Autoimmune diseases (IBD, arthritis)
- Neuro-immune disorders
- Metabolic syndrome
- Burn and hemorrhagic shock
- Obesity, fat disorders
- Diabetes

lymphatic dysfunction, inflammation and altered immunity

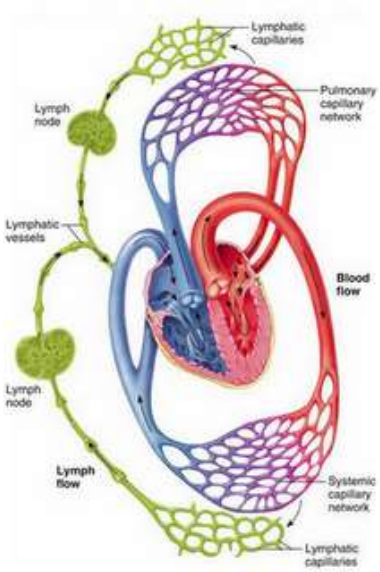
Key point: the lymphatic system mediates Immunity and inflammation!

A nodal-centric immuno-vascular system

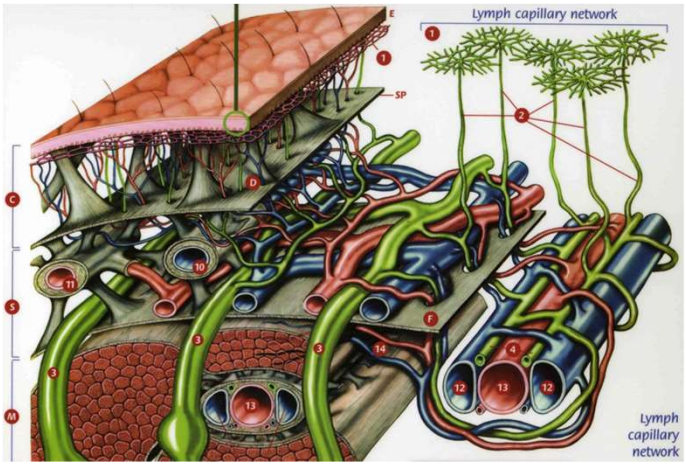


Lymphatics are the mortar

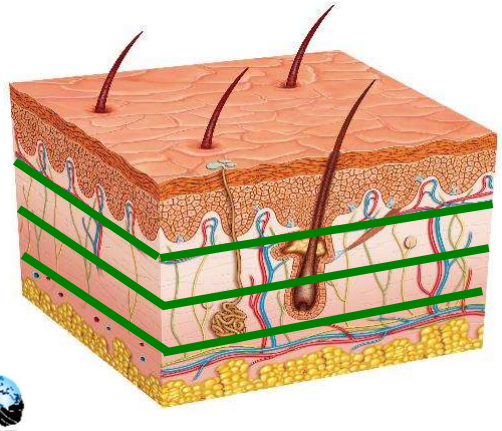
-Stanley Rockson

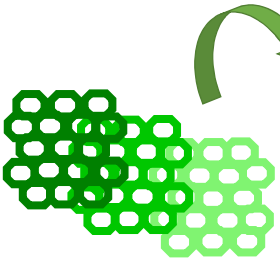


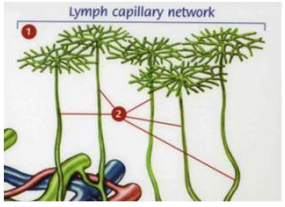
Lymphatics:
Body's Drainage and Waste Management System



Three layers of **lymph capillaries** located in the dermis

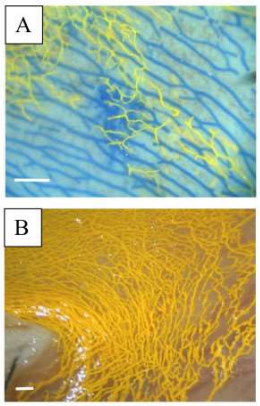


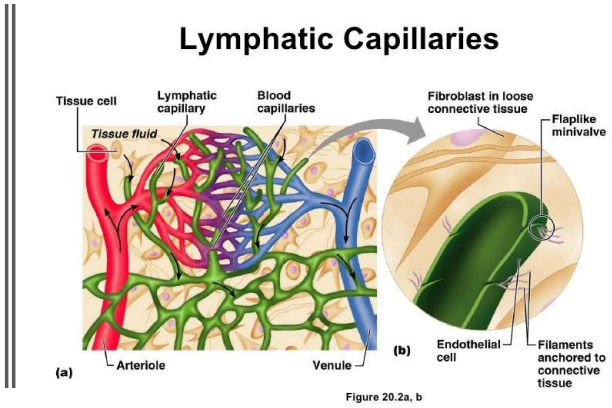




3mm!!

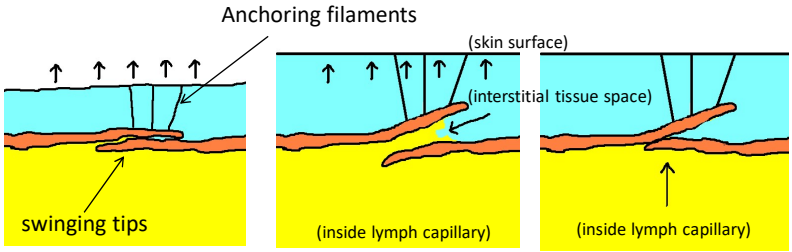
Slide courtesy of ILWTI



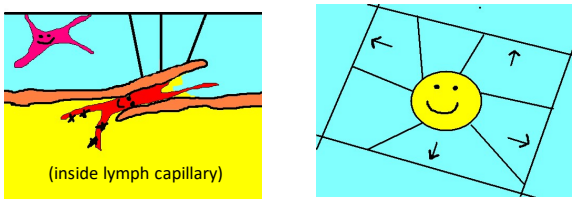


Spider web-like network

Lymphatic Capillaries: Swinging Tips & Anchoring Filaments



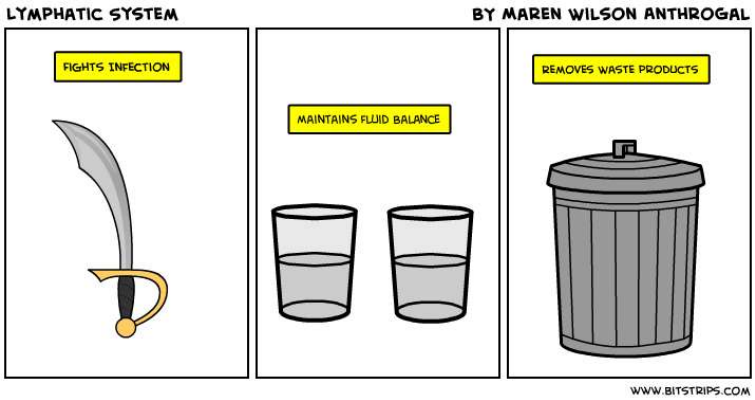
Edema (blue) stretches skin and opens swinging tips



Langerhan's cell enters lymph capillary

Anchoring filaments open lumen

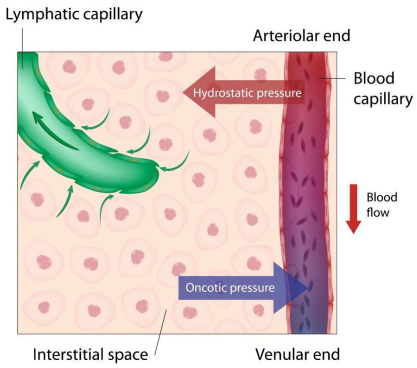
One of the main functions of the lymphatic system is to facilitate fluid movement from the tissues back to the blood circulation….*maintain a normal fluid balance*



Starling's Law Redefined

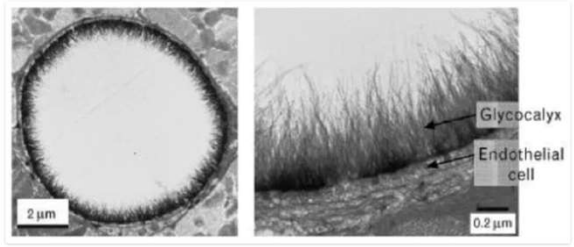
Previous Understanding

Capillary Fluid Exchange



Hydrostatic Pressure:
Favors filtration of plasma <u>out</u> of capillaries
Osmotic Pressure:
Favors osmotic movement of interstitial fluid <u>into</u> capillaries

New Understanding



Reprinted with permission from Biddle, C., 2013. Like a slippery fish, a little slime is a good thing: the glycocalyx revealed. *AANA journal, 81*(6).

Endothelial Glycocalyx Layer (EGL): gel-like matrix with hair like projections extending into lumen of blood vessels; acts as molecular sieve regulating fluid and macromolecule movement
Only a diminishing net *filtration* across capillary bed

Endothelial Glycocalyx Layer (EGL)

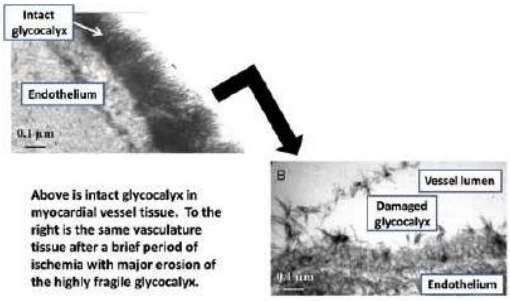
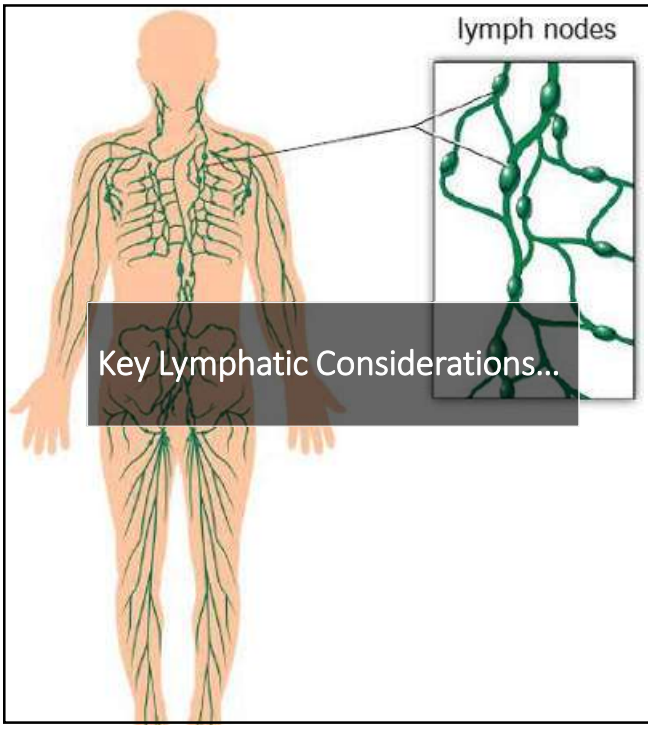


Image from: Biddle, C., 2013. Like a slippery fish, a little slime is a good thing: the glycocalyx revealed. *AANA journal*, *81*(6).

lymphatic system becomes overwhelmed leading to clinical edema

- The EGL is dynamic and can "shed" in response to stimuli, such as during inflammation or disease states
- Shedding is conceptualized as a dog shedding its fur
- During inflammation, this shedding allows more fluid to escape through the EGL
  (Biddle, 2013; Weinbaum, Tarbell, and Damiano, 2007)
- EGL sheds in response to inflammation, ischemia, sepsis, trauma, atherosclerosis, diabetes, intravenous fluid mis-management (Biddel, 2013; Reitsma et al., 2007) as well as prolonged immobility and anti-gravity environments
  (Belgrado lecture Peachtree City, GA Dec 2017)



- The dense, capsular design of the lymph nodes, placement in joint areas that are mechanically compressed by movement, and EGL, all work synergistically to facilitate fluid reabsorption back into the venous system through the lymphatics alone
- Conversely, immobility and decreased joint movement through the full range of motion, lymph node removal, or venous hypertension, can have a significant impact on fluid retention in the dermis and subcutaneous tissues
- All fluid, proteins and macromolecules are *removed from the interstitium by the lymphatics alone* (capillaries and nodes)

Starling's Law Redefined

There is no re-absorption in the blood capillaries; fluid is returned to venous system/vascular compartment by the lymphatics

Paradigm Shift

"Arguably, it may be better to consider the presence of chronic edema as synonymous with the presence of lymphedema, inasmuch as all edema represents relative lymph drainage failure."

–Dr. Stanley Rockson, Stanford Medical Ctr.

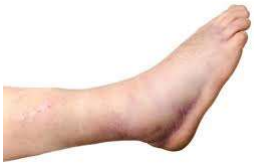
Thinking Point

- "…all edema indicates an inadequacy or failure of lymphatic drainage…"
  - Mortimer, Rockson. New developments in clinical aspects of lymphatic disease. The Journal of Clinical Investigation http://www.jci.org Volume 124 Number 3 March 2014.
- …lymphatic failure is responsible for all forms of peripheral edema…
  - Diagnosis and treatment of primary lymphedema consensus document of the International Union of Phlebology (IUP) 2013.

Edema vs. Lymphedema Recap

Edema (lymphatic insufficiency)

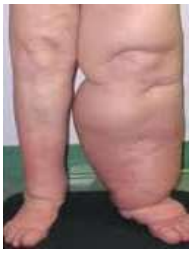
abnormal excess accumulation of serous fluid in connective tissue. Lymphatic system is *temporarily overwhelmed*, but not permanently damaged



Low protein edema

Lymphedema (lymphatic impairment)

accumulation of protein rich fluid in an extremity or body part as a result of *damage or loss* of part of the lymphatic vessel system



Protein rich edema

The Lymphedema Continuum

Lymphatics Temporarily Overwhelmed

Lymphatic Transport Capacity

Overwhelmed…Most Edemas
Transient lymphedema/Lymphatic Insufficiency

Lymphatics Permanently Impaired or Damaged

Permanently Damaged…
The Disease of Lymphedema

Lymphatics: The Undervalued Missing Link

- The lymphatic system is also tasked with the absorption and transportation of lipids and fatty acids to the circulatory system, and transporting antigens, antigen-presenting cells and other immune cells to the lymph nodes where adaptive immunity is stimulated
- Disorders of the lymph system, whether systemic (macro-lymphedema) or localized (micro-lymphedema), produce cutaneous regions susceptible to infection, inflammation and carcinogenesis (skin barrier failure)

  Carlson A. Lymphedema and subclinical lymphostasis (microlymphedema) facilitate cutaneous infection, inflammatory dermatoses, and neoplasia: A locus minoris resistentiae. Clinics in Dermatology. 2014;32: 599-615.

  Ruocco V, Schwartz RA, Ruocco E. Lymphedema: An immunologically vulnerable site for development of neoplasms. J Am Acad Dermatol. 2002;47:124-127.

  Ruocco E, Puca RV, Brunetti G, et al. Lymphedematous areas: Privileged sites for tumors, infections, and immune disorders. Int J Dermatol. 2007;46:662.

- 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

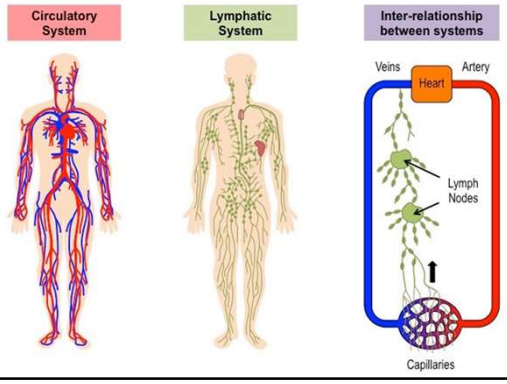
  Ridner SH. Pathophysiology of lymphedema. Semin Oncol Nurs. 2013;29:4-11.

Lymph/Integ Connection!! Contributes to development and/or chronicity of chronic wounds. Impairment in one system can lead to dysfunction in others…

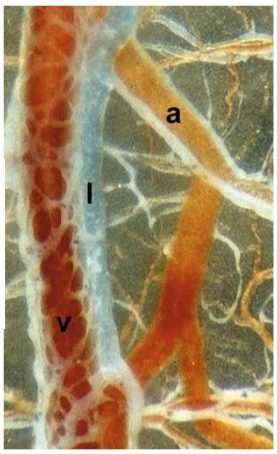
Redefining the Circulatory System

Circulatory System has 3 components:

- Closed blood circulatory system
  - Arteries
  - Veins
- Half-open lymphatic system

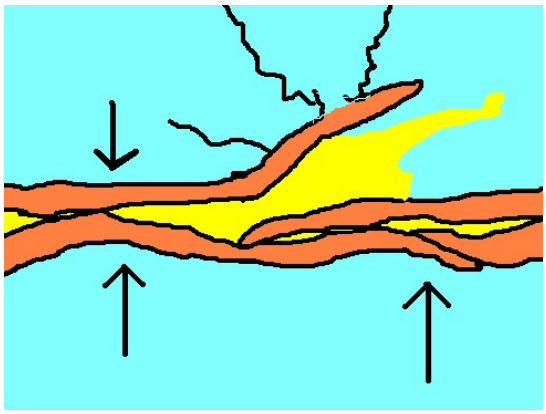


a- artery
v-vein
l- lymph vessel



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

Lymphatic Capillaries: Pathophysiology with CVI

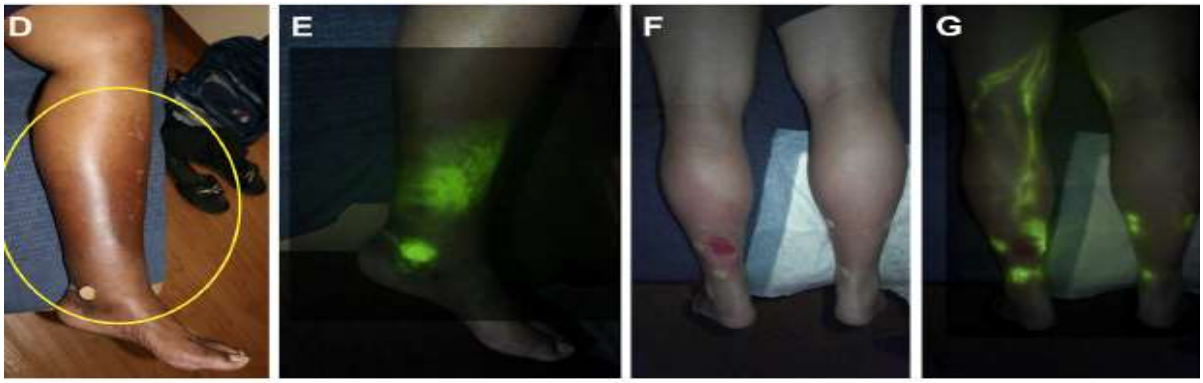


Study by Scelsi et al. 1994: CVI & stasis dermatitis:

1. **lymphatic capillary lumen collapses**
2. **derangement of the anchoring filaments**

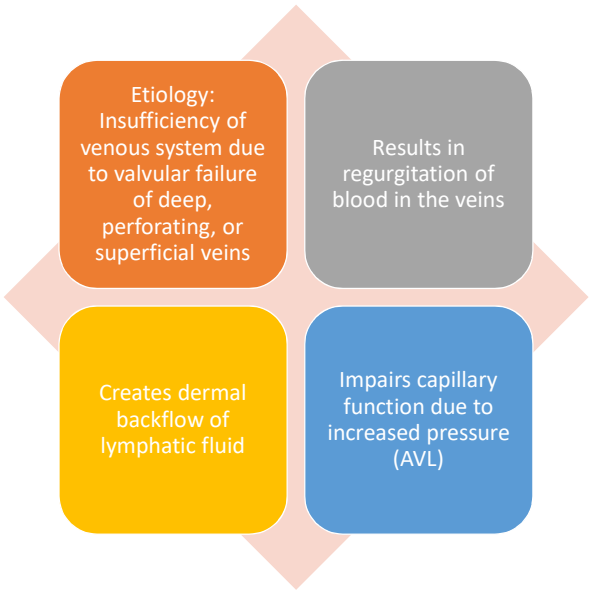
Lymphatics are Damaged From CVI & VLU

Slide courtesy of ILWTI



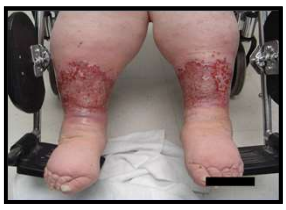
Rasmussen, J et al. Lymphatic transport in patients with chronic venous insufficiency and venous leg ulcers following sequential pneumatic compression. Journal of Vascular Surgery: Venous and Lymphatic Disorders. 2016; 4(1): 9-17.

CVI Pathophysiology



Dual Outflow System Failure

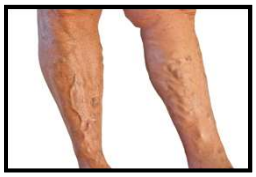
Pathophysiology of Phlebolymphedema

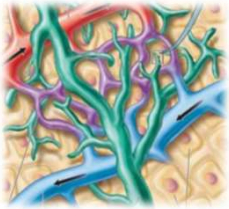


Chronic venous insufficiency

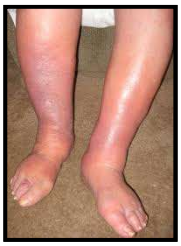
Lymphedema is underlying pathology contributing to formation of venous ulcers

High filtration pressure/increased fluid in tissues

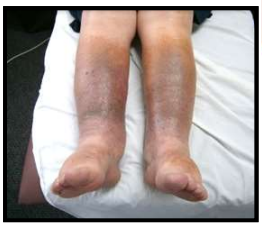




Waterload exceeds lymphatic transport capacity



Lymphatic damage



Lymphatic hypertension leads to fibrosclerosis

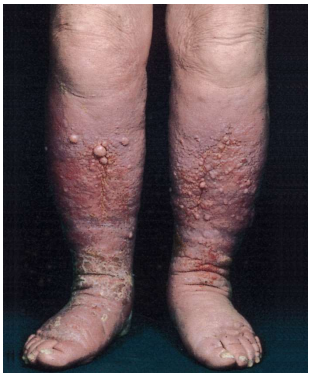
Low protein edema

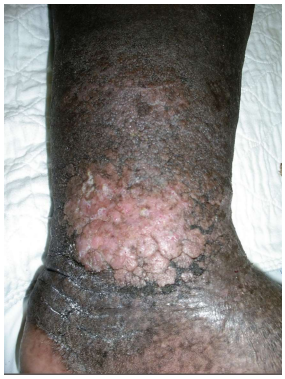
All edema including chronic venous insufficiency is actually 2° lymphedema!

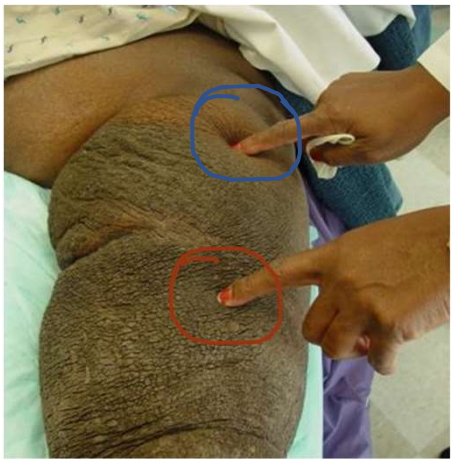
≥ C3 = CVI = 2° LYMPHEDEMA!

Eklöf B et al. Revision of the CEAP classification for chronic venous disorders: consensus statement. J Vasc Surg 2004;40:1248-52.

Why does this happen to the skin?



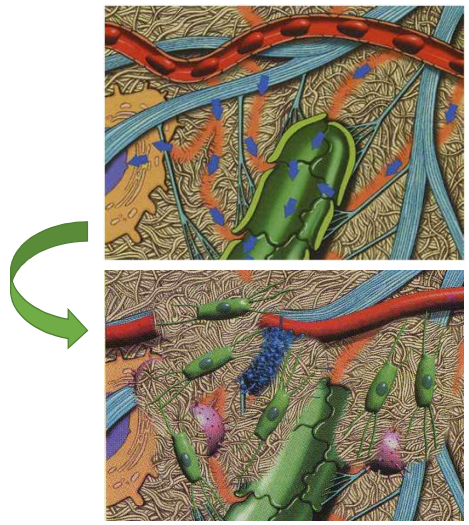




Pathophysiology and Integumentary Manifestations

Disorders of Lymph Drainage

- Protein rich fluid accumulates in the tissue
- Interstitial "ground substance" swells
- Characteristics of protein rich fluid
  - Macrophages (Blue)
  - Fibroblasts (Green) produce collagen
- Connective tissue proliferation
- Fibrosclerosis



Lymphedema Results in Chronic Inflammation & Stagnant Wound Milieu

Slide courtesy of ILWTI

"…stagnating high protein edema develops a patho-histological state of chronic inflammation, with infiltration of the tissue by mononuclear cells, angiogenesis, proliferation of connective tissue, fibrosis and fibrosclerosis…"

"Oxidation & degradation of interstitial proteins attracts monocytes that change into macrophages. Macrophages ingest the proteins and activate fibroblasts that, in turn, form collagen resulting in connective tissue proliferation."

Also triggers adipocytes, leading to fatty tissue proliferation (spongy lymphedema).

Foldi, M. (2006). Foldi's textbook of lymphology. Munchen: Elsevier.

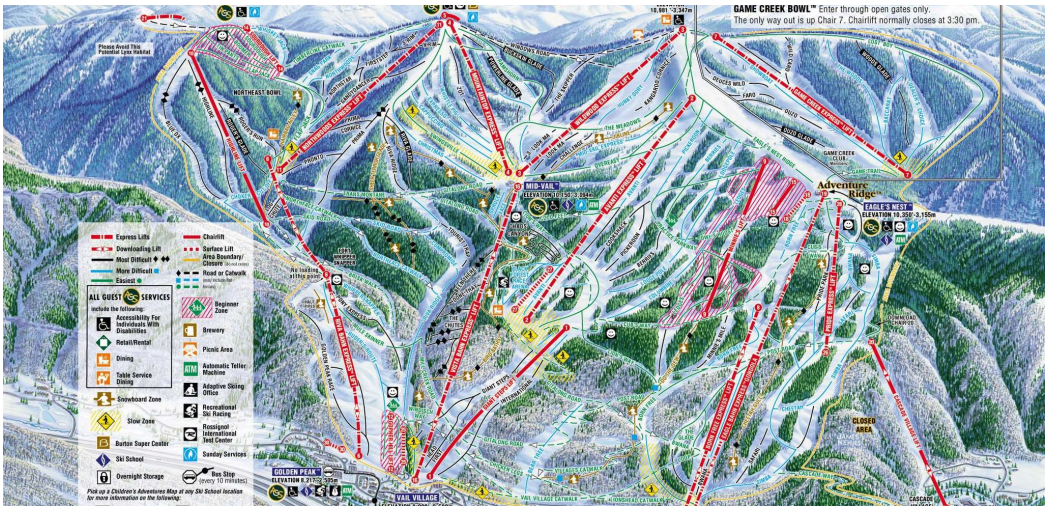
Clinical Pearls

- Edema is the clinical manifestation of either an overwhelmed or damaged lymphatic system; one is transient the other is a disease and both are part of the lymphedema continuum
- The venous, arterial, integumentary, and lymphatic systems (think VAIL) are inter-related; dysfunction in one system will lead to dysfunction in the other systems (may be subclinical or overt)
- Lymphatic impairment leads to local areas of compromised skin barrier function rendering the skin more prone to breakdown and impairments
- Movement enhances VAIL promoting more optimal functioning (muscle pump, nodes near joints, vascular integrity/health)
- Compression is essential with venous and lymphatic disease even before clinical evidence of edema

Clinical Pearls

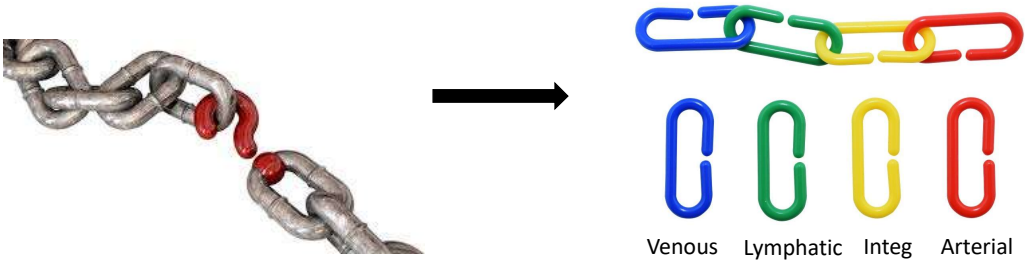
- The venous and lymphatic systems are mutually interdependent
  - When dysfunctional, the result is a dual outflow system failure
- Severe phlebolymphedema is caused by combined high lymphatic *flow* and low lymphatic *drainage*
- Phlebolymphedema (not cancer) is the most common cause of lower extremity secondary lymphedema in Western countries
- The pathophysiology of lymphedema explains the propensity for infections (cellulitis) and hypersensitivity reactions in patients with CVI
- Complete Decongestive Therapy is indicated for the management of Phlebolymphedema along with appropriate skin and wound management

VAIL- do you see the veins, arteries, skin and lymphatics in this picture?



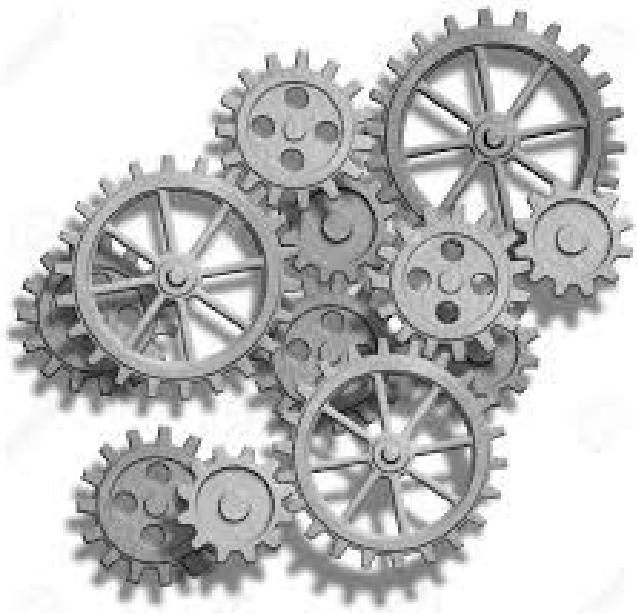
Vail, Colorado

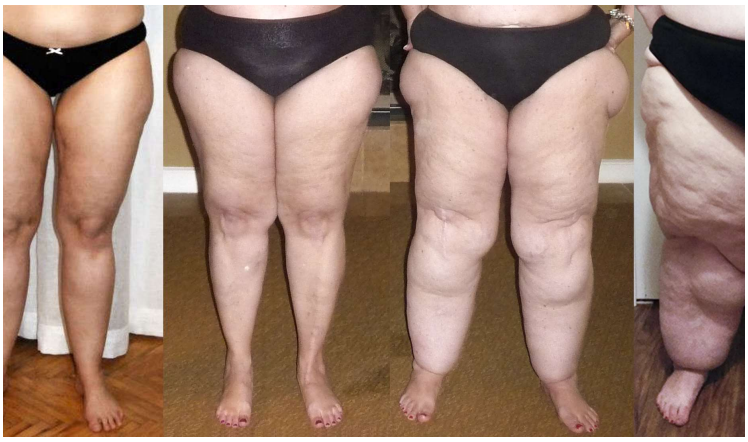
From Missing Link to Inter-dependence



Switching Gears…

Let's briefly discuss Lipedema





The Lipedema Project

Lipedema
Diagnosis is Clinical

- Pathological deposition of fatty tissue, usually below waist, leading to progressive leg enlargement
- Feet spared
- Occurs almost exclusively in women
- May be inherited
- Frequently misdiagnosed as lymphedema
- Disparity, lower body size > upper body size (masked by obesity)
- Increased fat tissue on head, supraclavicular, axilla
- Palpable nodules
- Mattress pattern thigh
- Lobules in adipofascia
- Hypermobility

Lipedema Key Symptoms

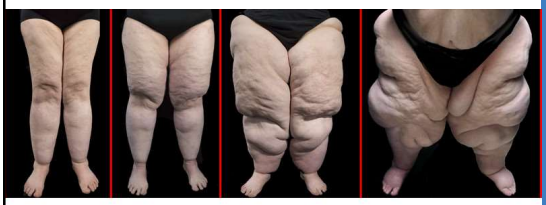
- Feeling of heaviness in the legs (aching dysesthesia)
- Easy bruising due to a lack of anchoring of the small capillaries in the connective tissue, which results in tearing when affected by the pull of gravity
- Sensitivity to touch (painful fat syndrome)
- Orthostatic edema during long periods of standing
- May have "oatmeal changes" to the skin
- Fat pad sign (filling of retro-malleolar sulcus)
- Diet and exercise resistant
- Fluid in the fat

Lipedema Additional Considerations

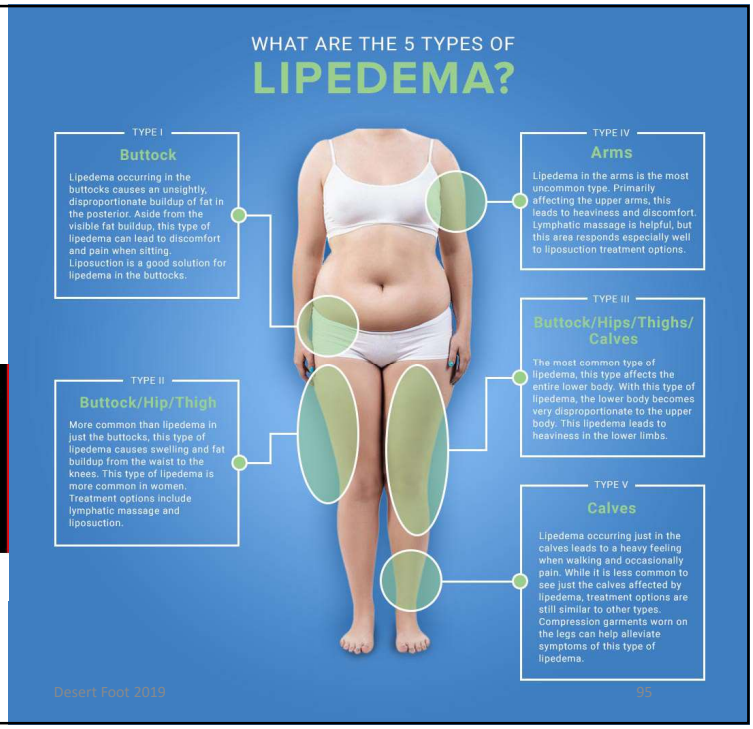
- Knee/joint hyper-mobility
- Knee problems common which may lead to gait impairment
- Nodular fat pads above, inside and below knees and in outer region of upper thighs
- Accumulation of lipedemic fat in the upper arms, sometimes leaving large amounts of arm fat hanging when arm is outstretched
  - Research suggests 80% of people with lipedema are affected in their arms as well

Progressive condition
4 stages and 5 types

www.fatdisorders.org
www.lipedema.net

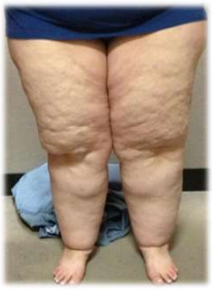


Stage 1 Stage 2 Stage 3 Stage 4

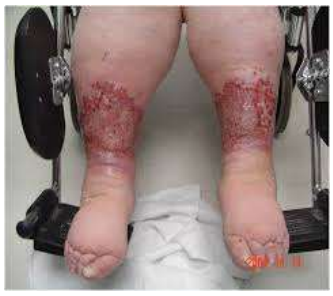


Mixed Presentations

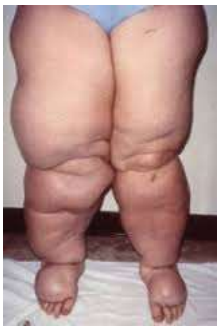
These conditions can present independently or in combination…



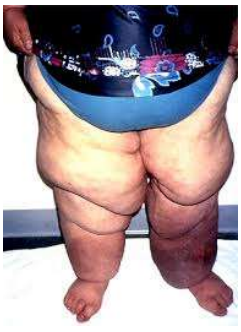
Lipedema



Phlebolymphedema



Lipolymphedema



Phlebolipolymphedema

Note the tell-tale clinical characteristics of these various conditions

Utilization of Compression
Early to Late Stage Manifestations for Edema Control

Marie Clarke, MSN, RN, CWCN

Lower Extremity Boot Camp,
Desert Foot 2019

Disclosure

Marie Clarke is a Clinical Wound Care Specialist
for
3M Medical Solutions Division, 3M Health Care

Objectives

At the completion of this presentation participants should be able to:

- Identify the role of compression in edema management,
- Describe etiologies that require compression intervention,
- Discuss management options available for edema management using compression therapy devices.

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Understanding Compression

- Application of a mechanical force or external pressure to a limb or body part
- "Gold Standard"
  - Management of edema associated with chronic venous insufficient and lymphedema and various other etiologies
  - Treatment and prevention of venous leg ulcers
- Positive effects on venous and lymphatic function

Bjork R, Ehmann S. S.T.R.I.D.E. Professional guide to compression garment selection for the lower extremity. Journal of Wound Care 2019: 28㉖6 suppl 1):1-44.

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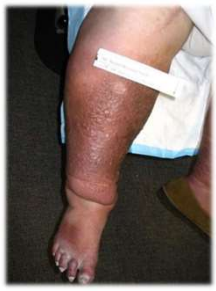
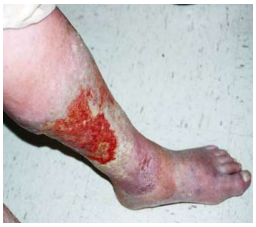
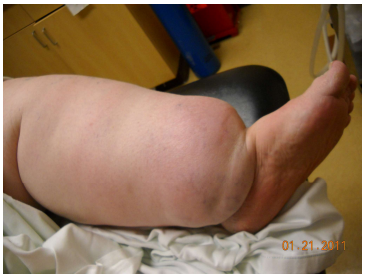
Who needs Compression?





- Patients with known risk factors for Lower Extremity Venous Disease
- Patients with early signs of Chronic Venous Disease
- Edema management
- Prevention of disease progression to ulceration
- Treatment for edema associated with venous ulceration



Carmel JE. Venous Ulcers. In: Bryant BA, Nix DP. In: Acute & Chronic Wounds; Current

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Risk factors for Lower Extremity Venous Disease

Valvular Dysfunction

- Family history venous disease
- Pregnancy (multiple or close together)
- Systemic inflammation
- Venous thromboembolism
- Obesity

Calf Muscle Pump Dysfunction

- Sedentary lifestyle
- Prolonged sitting or standing
- Surgery/trauma top foot/ankle/leg
- Altered gait
- Paralysis
- Restricted range of motion of the ankle
- Advanced age

Carmel JE. Venous Ulcers. In: Bryant BA, Nix DP. In: Acute & Chronic Wounds; Current Management Concepts, 5th ED. St. Louis, MO: Elsevier Mosby; 2016: Chapter 12.

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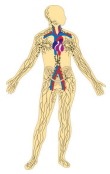
Effects of Compression



Venous Circulation

- Reduced venous reflux and improved venous return
- Reduced venous hypertension
- Maximized calf muscle pump
- Reduced elevated matrix metalloproteinase levels, promoting healing of VLUs

Lymphatic Circulation

- Reduced formation of excess interstitial fluid
- Shifting fluid into areas with functional lymphatics
- Promotes lymphatic drainage
- Improved muscle pump activity
- Reduces inflammation
- Softens fibrotic tissue

Partsch, H., and Mortimer, P. Compression for leg wounds. British Journal of Dermatology, (2015) 173. pp359-369
Partsch, H., & Moffatt, C. (2012). An overview of the science behind compression bandaging for lymphedema and chronic oedema. In Best Practice For the Management of Lymphedema, 2nd Edition. The International Framework in Lymphedema with the World Alliance for Wound and Lymphedema Care. June 2012
Bjork R, Ehmann S. S.T.R.I.D.E. Professional guide to compression garment selection for the lower extremity. Journal of Wound Care 2019: 28☹6 suppl 1):1-44.

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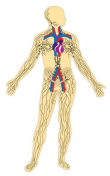
Effects of Compression



Clinical Symptoms

- Reduced pro-inflammatory cytokines
  - Anti-inflammatory effect
  - Reduced pain
  - Promotion of wound healing
- Reduced edema
  - Able to ear own shoes
  - Able to participate in normal Activities of Daily Living
  - Improved Quality of Life

Trophic Changes

- Reduced inflammatory response
- Resolution of fibrotic tissue → softening of skin

Partsch, H., and Mortimer, P. Compression for leg wounds. British Journal of Dermatology, (2015) 173. pp359-369
Partsch, H., & Moffatt, C. (2012). An overview of the science behind compression bandaging for lymphedema and chronic oedema. In Best Practice For the Management of Lymphedema, 2nd Edition. The International Framework in Lymphedema with the World Alliance for Wound and Lymphedema Care. June 2012
Bjork R, Ehmann S. S.T.R.I.D.E. Professional guide to compression garment selection for the lower extremity. Journal of Wound Care 2019: 28☹6 suppl 1):1-44.

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CEAP Classification
C: Clinical
E: Etiology
A: Anatomical
P: Pathophysiology

Clinical Manifestations

- C0 – No visible or palpable signs of venous disease
- C1 – Telangiectasias or reticular veins
- C2 – Varicose veins
- C3 – Edema
- C4 – Changes in skin and subcutaneous tissue
  - Pigmentation or eczema
  - Lipodermatosclerosis or atrophie blanche
- C5 – Healed venous ulcer
- C6 – Active venous ulcer

Eklof B., Rutherford R.B., Bergan J.J. et al. (2004). Revision of the CEAP classification for chronic venous disorder: Consensus statement. Journal of Vascular Surgery, 40(6), 1248-1252.

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Compression options

- Short stretch inelastic bandaging
  - Multilayer compression bandaging
- Zinc-paste wrap with cohesive wrap
- Sequential compression pump therapy (adjunct, not maintenance)
- Therapeutic stockings/garments (prevention)
- Long stretch

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Unraveling the Compression Terminology Types of Compression

Non Stretch	Short Stretch	Long Stretch
Paste bandages	Stretch 30-60% original length	Stretch 140-300% original length
High working, no resting pressure	High working, low resting pressure	Low working pressure, high resting pressure
Excellent containment	Excellent containment, some recoil to accommodate as edema decreases	Poor containment, good recoil
Facilitates calf muscle pump only during ambulation	Facilitates calf muscle pump during ambulation	Substandard care for pts with CVI or lymphedema and poses risk for patient with PAD
May slide/bunch if edema decreases	Safe to apply up to 40 mmHg on pts with ABI > 0.5 and systolic ankle pressure > 60 mmHG	ACE bandage = all cotton elastic
Risk of edging	Increases arterial blood flow by 28% Increases venous return by 103%*	

Hettrick, H. Compression Fundamentals. 2016

*Mosti G, Iabichella ML, Partsch H. Compression therapy in mixed ulcers increases venous output and arterial perfusion. J Vasc Surg. 2012; 55(1):122-8.

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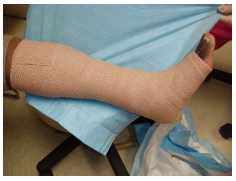
Unraveling the Compression Terminology Types of Compression

Compression Stockings or Garments	Compression Wraps	Intermittent Pneumatic Compression
Used for long term maintenance after optimal reduction with bandages	Alternate device for compression during the day or night	Increases venous return without impairing arterial perfusion
Worn during the day	Easier to don than garments	Reduces watery forms of edema (good for venous edema not for lymphedema w/o CDT)
Types: off the shelf, custom, knee high, thigh high, pantyhose	Often have alternative closure systems (i.e., Velcro)	Air pump intermittently inflates sleeve
Materials: Circular knit or flat knit	Excellent containment, minimal recoil	# of chambers can vary (4-12 for venous; ~30 for lymphedema)
Donning and Doffing Aids can assist with use/adherence	Adjustable	Time intensive

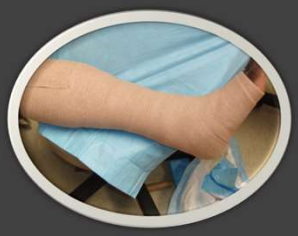
Hettrick, H. Compression Fundamentals. 2016

Interpretation of Compression



ABI > 0.8 and < 1.3	Standard compression (30-40mmHg) recommended Modified or light compression if patient unable to tolerate standard compression
ABI > 0.5 and < 0.8, w/o claudication or rest pain	Modified or light compression (20-30 mmHg) Refer to specialist for additional vascular testing
ABI < 0.5	Compression contraindicated Refer for additional testing
ABI > 1.3	Reading likely unreliable - refer for additional testing Do not initiate compression until arterial blood flow established

Inelastic Compression (Short-Stretch)



Maximum stretch < 100%

Low resting pressure

High working and standing pressure

Materials or textiles may vary:

- Woven material (cotton)
- Paste impregnated
- Short stretch cohesive
- Provide higher stiffness properties
- Applied at full stretch
- Extensibility < 40%
- Reusable or single use
- May or may not have cohesive properties

Mosti, G. Venous ulcer treatment requires inelastic compression. Phlebologie 2018; 47(01): 7-12

Multi-layer bandage systems

- 2, 3, 4 layer systems
- Layers may include:
  - cotton padding
  - crepe (conforming bandage)
  - foam padding
  - elastic bandage
  - self-adherent wrap

Product components differ between manufacturers.
Follow Instructions for Use

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Short stretch woven bandages

- Woven cotton provides minimal stretch, may be referred to as inelastic
- Provides higher stiffness properties
- Applied at full extension
- Extensibility < 40%
- Reusable or single use
- May be cohesive
- High working pressure; low resting pressure (supports lymphatic system)

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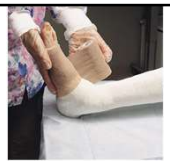
Zinc Paste Bandages\Unna's Boots



- Used for managing VLU since 1854 despite minimal research evidence.
- Does not adjust to changes in leg volume and continue to provide sustained therapeutic compression.
- Messy to apply and wear.
- Potential for allergic reaction or skin reaction to certain paste ingredients.
- Creates occlusive environment
  - Increase risk of maceration
  - Unable to handle high exudate levels
  - Requires more frequent application.
- High skill level for accurate application

No longer considered optimal for most patients.

Johnson, J, Yates, S, Burguss, J. Venous Insufficiency, Venous Ulcers, and Lymphedema, pp 393-94. In Core Curriculum Wound Management. 2016. Wound, Ostomy and Continence Nurses Society. Wolters Kluwer
Carmel, E, Bryant, R.A. Venous Ulcers, pp. 217-18. In Acute & Chronic Wounds: Current Management Concepts. Fifth Edition. 2016. Elsevier

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Adjustable Compression Devices

- Provides inelastic, short stretch compression
- Resistance to the calf muscle pump, increasing venous return
- Provides high working and low resting pressures
- Most have adjustable Velcro closure systems
- Adjustable to provide high or modified compression
- Permits bathing, access to wound for dressing change
- May be easier to don than compression garments

3M

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Intermittent Pneumatic Compression (IPC)

- Pump and sleeve, air pumped intermittently inflates the sleeve, Number of chambers can vary – 4-12 for CVI
- Increases venous return without impairing arterial perfusion
- Consider if arterial revascularization is not possible or for complete immobility
- Used in addition to compression bandage or stockings
- Compression begins at ankle and proceeds up the leg, cycling at different intervals
- Time intensive: 1-2 hours, twice a day
- Contraindicated in patients with uncompensated (symptomatic) heart failure, acute cellulitis, acute venous thrombosis

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Compression for life - Stockings and Garments

- Provide maintenance compression for life
- Generally not used when ulcers present
- Woven to provide a pre-determined level of graduated pressure
- Moderate elasticity to provide combination of moderate resting and working pressure
- Used daily for prevention and management of venous hypertension
  - ✓ Apply in AM before arising
  - ✓ Remove at HS

3M

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Compression Stocking Classification

Benefits:
- Low profile
- Reusable
- Patient independence

Challenges:
- Require fitting, training
- Cost
- Difficult to apply
- Patient adherence

US Class	Descriptor	Ankle Pressure
Class 1	Light support	20-30 mm Hg
Class 2	Medium support	30-40 mm Hg
Class 3	Strong support	40-50 mm Hg
Class 4	Very strong support	50-60 mm Hg

- 20-30 mmHg typically used for scar tissue management, UE edema
- 30-40 mmHg used for edema management in ambulatory patients

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Long-stretch Bandages - elastic bandage

- Woven/knitted materials with elastic fibers
- Highly extensible - stretch up to 3X length (100-300%)
- Application extension variable – commonly applied at 50 -100% stretch with 50% overlap
- Generally applied with around 40-50 mmHg pressure

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Carmel JE. Venous Ulcers. In: Bryant BA, Nix DP. In: Acute & Chronic Wounds; Current Management Concepts, 5th ED. St. Louis, MO: Elsevier Mosby; 2016: Chapter 12.

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Compression selection considerations – *patient* factors

- Precautions and Contraindications:
  - Inadequate arterial flow….
  - Suspected or known thrombus
  - CHF (controversial and lack of evidence)
  - Cellulitis (controversial and lack of evidence)
- Past experience/preference
- Tolerance/adherence to method
- Quality of Life issues

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Other pertinent considerations….

Will the product stay in place to optimize muscle contractions?

Does the bandage support normal mobility?

- Can the patient wear their own shoes?

What is your patient's response?

- Will your patient be comfortable?
- Will your patient be concordant / keep it on?

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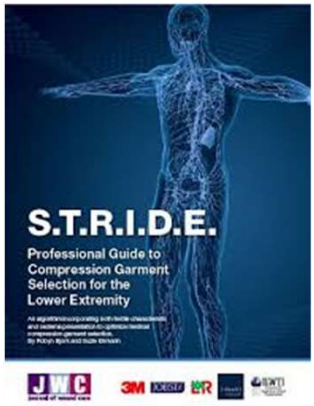

Great Resource

S.T.R.I.D.E.

Professional Guide to Compression Garment Selection for the Lower Extremity

J Wound Care. 2019 Jun 1;28(Sup6a):1-44. doi: 10.12968/jowc.2019.28.Sup6a.S1.

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Thank you!!!

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The Offloading Continuum: How to Improve Your Outcomes

James McGuire DPM, PT, LPed, FAPWHc
Director: Leonard Abrams Center for Advanced Wound Healing
Clinical Professor Temple University School of Podiatric Medicine
Philadelphia, PA

Disclosures

- Speaker: Essity, Smith & Nephew, 3M, Pure & Clean, Osirus
- Research Support:
- Osirus
- Pending Research support:
- RedDress, Reapplix

OBJECTIVES

At the end of this presentation participants should be able to:

- Analyze data that supports the Non-removable Knee High Device as the Gold Standard for offloading, why the TCC is the preferred device, and the reasons for clinician avoidance of the therapy.
- Describe the medical evidence supporting Alternative Offloading strategies and review devices used for wound healing in the diabetic foot.
- Share clinical experience with the use of the strategies and review indications and contraindications with each

Fife CE, et al. A Predictive Model for Diabetic Foot Ulcer Outcome: The Wound Healing Index. Adv Wound Care (New Rochelle). 2016 Jul 1;5(7):279-287

- Variables that significantly predicted healing were:
- Wound age (duration in days)
- Wound size
- Number of concurrent wounds of any etiology
- Evidence of bioburden/infection
- Patient age
- Wagner grade
- Being non-ambulatory
- Renal dialysis
- Renal transplant
- Peripheral vascular disease
- Patient hospitalization for any reason.

- Out of 13,266 DFUs from the original data set, 6,440 were eligible for analysis. The logistic regression model included 5,239 ulcers, of which 3,462 healed (66.1%). The 10% validation sample utilized 555 ulcers, of which 377 healed (67.9%).

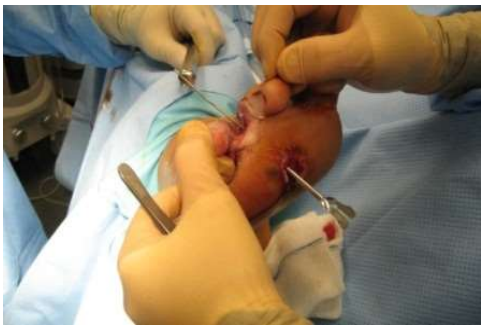
DFU Wound Healing Index is a comprehensive and user-friendly validated predictive model for DFU healing.

ABCESS Principles of Wound Management

Assess Arterial and Venous system function	**B**ioload Management	**C**ellular Activity Assessment	**E**xudate level assessment	**S**ystemic disease diagnosis and management	**S**kin protection and treatment
Normalize to the extent possible with available interventions	Prevent high bacterial counts and prolonged inflammation	Alter therapies based on cells and cell function observed in the wound bed	Maintain a moist wound environment	Control DM, ESRD, Autoimmune Dx, CHF, etc.	Prevent traumatic, inflammatory or iatrogenic damage
Vascular surgery , Compression wraps and Segmental Compression	Debridement, antimicrobials, antiseptics, hydrophobics, bacterial trapping	Wound fluid analysis, PCR/DNA, CTP Intervention Autogrft	Dressing selection or alteration NPWT	BS, BP, Nutrition,	Periwound protection, offloading, edge effect, decrease inflammation
Maximize vascularity and healing potential	Low bacterial counts and controlled inflammation	Restore cell migration, maceration avoided	Assures a healthy environment for growing cells	Systemic interventions to improve local healing capacity	Prevent unintended damage to the wound and nearby tissues

Diabetes

- 85% of all lower limb amputations in Diabetics are preceded by a foot ulcer(1,2,3)
- Diabetics who develop a foot ulcer have a 55x greater risk of infection (4)
- If a DFU is open 30 days or longer it has a 4x greater risk of infection



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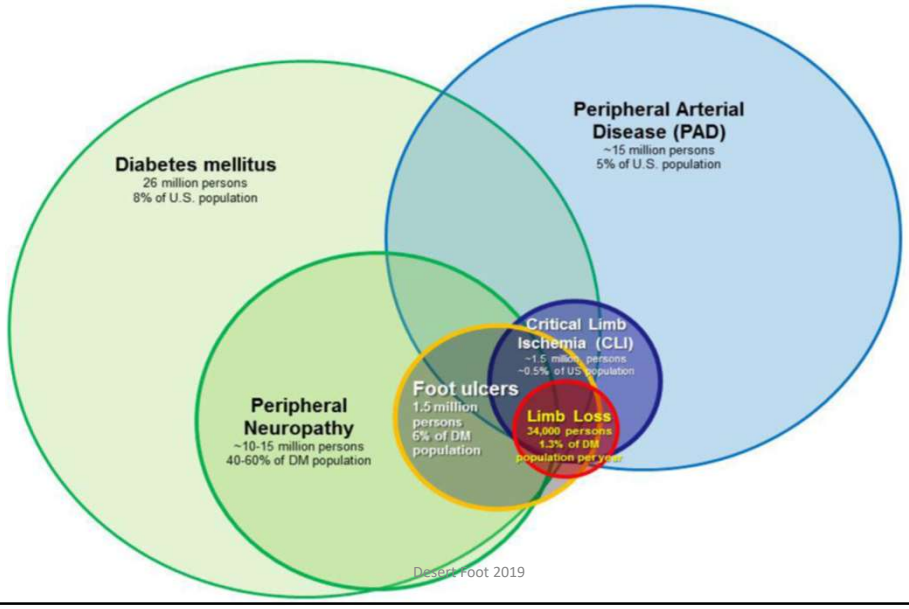
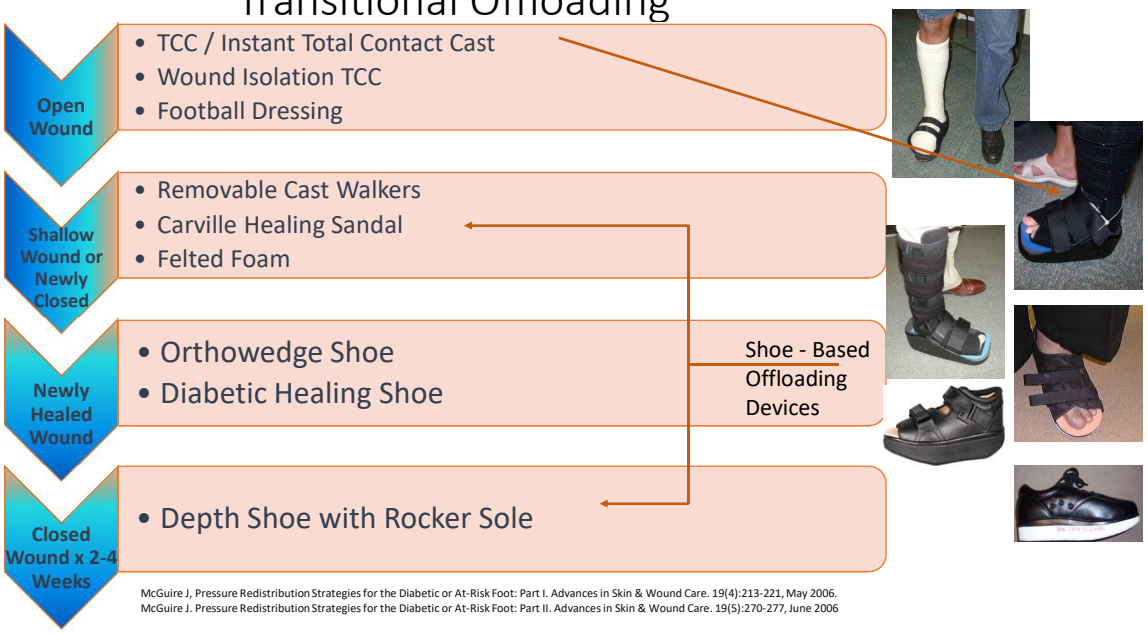
Overlapping risk factors associated with non-traumatic limb loss

Barshes NR, et al. Diabetic Foot & Ankle 2013



Transitional Offloading



McGuire J, Pressure Redistribution Strategies for the Diabetic or At-Risk Foot: Part I. Advances in Skin & Wound Care. 19(4):213-221, May 2006.
McGuire J. Pressure Redistribution Strategies for the Diabetic or At-Risk Foot: Part II. Advances in Skin & Wound Care. 19(5):270-277, June 2006

Forces an Offloading Device Must Address

- Direct Pressure – weight, sagittal deformities
- Anterior Posterior Shear – heel strike, push off
- Transverse Shear – lateral weight shift
- Rotary Shear – twist
- Load distribution – total contact, pressure relief
- Temporal Management – time on limb, speed of gait, distribution of forces across the gait cycle
- Compliance: Pt. willingness to wear the device

2015 Recommendations from the International Working Group on the Diabetic Foot (IWGDF)

- http://www.iwgdf.org/files/2015/website_footwearofloading.pdf

2015 IWGDF Guidelines: Casting and prefabricated healing devices

- To heal a neuropathic plantar forefoot ulcer without ischemia or uncontrolled infection in a patient with diabetes, offload with a non-removable knee-high device (TCC, iTCC) with an appropriate foot-device interface. (GRADE recommendation: strong, Quality of evidence: high)
- When a non-removable knee-high device is contraindicated or not tolerated by the patient, consider offloading with a removable knee-high walker with an appropriate foot-device interface when the patient can be expected to be adherent to wearing the device. (Weak; Moderate)
- There is no particular preference for a TCC or prefabricated non-removable knee-high device as long as an appropriate foot device interface is maintained. We conclude that the quality of (Evidence high)

IWGDF Guidance on footwear and offloading interventions to prevent and heal foot ulcers in patients with diabetes. S. A. Bus; D. G. Armstrong; R. W. van Deursen; J. Lewis; C. F. Caravaggi; and P. R. Cavanagh; on behalf of the International Working Group on the Diabetic Foot (IWGDF), 2015

Strut Height Influences Forefoot Loading

Mean forefoot pressure reduction based on strut height in removable cast walkers.

Gentzkow GD, Iwasaki SD, Hershon KS. Use of Derma-graft, a cultured human dermis, to treat diabetic foot ulcers. Diabetes Care. 1996;19(4):350-354

In Armstrong et al. Offloading Foot Wounds in People with Diabetes. WOUNDS. 2014;26(1):13-20.

Therapeutic Footwear

- When a knee-high device is contraindicated or cannot be tolerated consider offloading with a forefoot offloading shoe, cast shoe, or custom-made temporary shoe but only and when the patient can be expected to be adherent to wearing the shoes. (Weak; Low)
- Do not prescribe, *or encourage the use of*, conventional or standard therapeutic shoes to heal a plantar foot ulcer. (Strong; Low)
- Consider using shoe modifications, temporary footwear, toe spacers or orthoses to offload and heal a non-plantar foot ulcer without ischemia or uncontrolled infection in a patient with diabetes. The specific modality will depend on the type and location of the foot ulcer. (Weak; Low)

IWGDF Guidance on footwear and offloading interventions to prevent and heal foot ulcers in patients with diabetes. S. A. Bus; D. G. Armstrong; R. W. van Deursen; J. Lewis; C. F. Caravaggi; and P. R. Cavanagh; on behalf of the International Working Group on the Diabetic Foot (IWGDF). 2015

Using "Extra-Depth" Diabetic Shoes to Heal Ulcers

- "Extra depth" shoes help prevent ulcers…but were never intended to treat active ulcers.
- After ulcers close, the tissue is highly vulnerable to re-ulceration for weeks/months (30% - 40% recurrence within 1 year).
- Offloading insoles are needed to transition patients from contact cast to extra-depth shoes.

Typical Surgical Shoe Felt Modification

Lehrman 2017

Other offloading interventions

- If other forms of biomechanical relief are not available, consider using felted foam in combination with appropriate footwear to offload and heal a neuropathic foot ulcer without ischemia or uncontrolled infection in a patient with diabetes. (Weak; Low)

IWGDF Guidance on footwear and offloading interventions to prevent and heal foot ulcers in patients with diabetes. S. A. Bus; D. G. Armstrong; R. W. van Deursen; J. Lewis; C. F. Caravaggi; and P. R. Cavanagh; on behalf of the International Working Group on the Diabetic Foot (IWGDF). 2015

Things to Remember About Removable Devices

- Armstrong, et al., reported that patients treated with a removable device wore the device a total of 28% of their daily activity with even the most compliant population not exceeding 60%

Armstrong, D.G., et al., Activity patterns of patients with diabetic foot ulceration: patients with active ulceration may not adhere to a standard pressure off-loading regimen. Diabetes Care, 2003

TCC Systems: Designed to Make It Easier to Use

- Easy to use – no learning curve
- Shortened application time
- Not messy
- Lighter
- Cooler
- Increased patient acceptance
- Functionality of traditional TCC

Reverse Gold Standard

- So if the offloading device used by most practitioners is the shoe or a shoe based system (41.2%, P < 0.03), which has the worst healing data, then…
- It just might be considered the "Reverse Gold Standard"
- "Anything" that demonstrates better healing rates than the diabetic shoe would therefore be preferable with the TCC being the most preferred device.

Wu SC, Jensen JL, Weber AK, Robinson DE, Armstrong DG. Use of pressure offloading devices in diabetic foot ulcers: do we practice what we preach? Diabetes Care. 2008 Nov;31(11):2118-9

Don't Forget to Use Ambulatory Aides Just Don't Expect Them to Use Them

- Cane - Increases base of support, Minimal off-loading
- Crutches - NWB to PWB
- Walker - Greater stability, NWB to PWB
- Wheel Chair
- Bed Rest/Decreased Activity

Patient Adherence: Factors

- ❖Understanding of the Disease Process
- ❖Footcare Knowledge
- ❖Compliance History
- ❖Complexity of the Treatment or Therapy
- ❖Beliefs and Perceptions of Benefits
- ❖Family Support
- ❖Practitioner Reinforcement

Non-Removable Knee High Walkers or the Instant Total Contact Cast (iTCC)

- Contouring insole
- Easy to apply and modify.
- A single roll of 2” cast tape, self-adhesive wrap or a non-removable plastic cable connector may be applied to prevent removal and insure patient compliance.
- Allows for easy access to the wound

Offloading System

*Alberto Piaggesi MD et Al, Comparison of Removable and Irremovable Walking Boot to Total Contact Casting in Offloading the Neuropathic Diabetic Foot Ulceration. Foot & Ankle International April 15, 2016 (60 Patients)
**Alberto Piaggesi MD et Al, An Off-the-Shelf Instant Contact Casting Device for the Management of Diabetic Foot Ulcers. Diabetes Care 2007 Mar; 30(3): 586-590. (40 Patients)

Our results suggest that a walking boot was as effective and safe as TCC in offloading the neuropathic DFUs, irrespective of removability. The better acceptability and lesser costs of a removable device may actually extend the possibilities of providing adequate offloading.

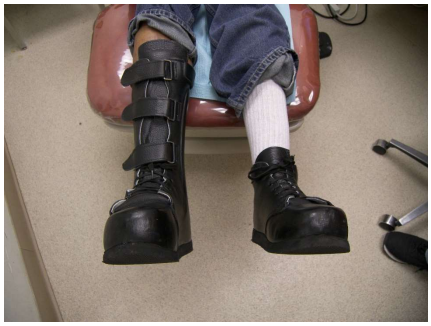
Commercial Wound Care Shoe

- Rocker sole. Free Ankle
- Hook and loop closure
- Sold in pairs to avoid sole height problems
- Permits off-loading of individual ulcers
- Transitional device to precede the use of final footwear
- Balance and Fall Issues

Custom Molded Shoes

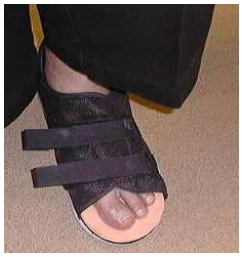
Reserved for patients with:

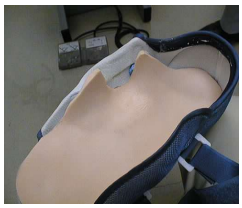
- Moderate to severe foot deformity
- Unstable skeletal structure
- Recurrent ulcerations
- Chronic problems not designed for acute wound management



Modified Surgical Shoe Technique

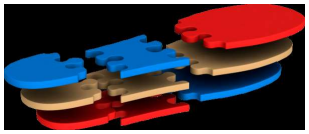
- For the initial "off-loading" of superficial ulcerations or pre-ulcerative lesions in Compliant patients
- Not appropriate for deep Full Thickness Ulcerations in Non-Compliant patients
- A Rocker Bottom Surgical Shoe is Modified with a Heat Molded EVA or Plastizote® insole



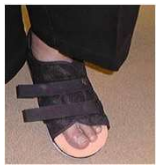


Transitional Shoe-Based Offloading Devices

- **Segmented Insoles**

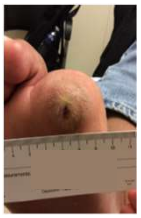


- **Heat Molded Insoles**



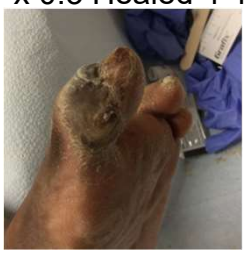
Effectively Off-Loads Select Plantar Areas

- The pixelated units can be removed for selective site off-loading.
- Various shaped units are easily removed from the multilayer insole
- Plantar units do no need to be skived to reduce edge effect



CASE 1:
DFU in a Non-Compliant Ambulator
2-3-16 to 4-11-16 (5 weeks)

CASE 2: DFU with Ischemia: 3/11/16 - Initial Ulcer 1.0 x 1.0 x 0.3 Healed 4-18-16 in 5 Weeks



Current shoe/boot based offloading systems

- Cast walkers are not designed to match the anatomical contours of the foot and/or offload plantar ulcers in specific anatomy. (To improve effectiveness of cam walkers an adjustable insole is preferred.)
- Similarly, "rocker-bottom" shoes help shift some weight, but not appropriate for open ulcers, and does not address specific anatomy. (An molded or adjustable insole is often preferred.)
- "Heel Wedge" or Half shoes can help shift force from the forefoot…but increase likelihood of patients slipping and falling. Also, not useful for heel or midfoot ulcers

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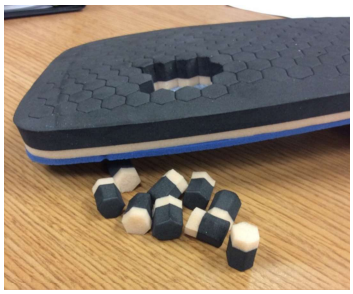
Adjustable Offloading Insoles

- Edge effects
- Segment instability
- Compression
- Lack of compliance


Desert Foot 2019

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Presented at the American Podiatric Medical Conference, Nashville, Tennessee 2017

Evaluation of a Pixelated Innersole Designed to Offload Areas of Elevated Pressure on the Soles of At-Risk Feet

James McGuire DPM, PT, LPed, FAPWHc, James Furmato DPM, PhD., Jesse Borys BS
Temple University School of Podiatric Medicine, Philadelphia, PA.

Background

The Total Contact Cast (TCC) has been recognized as the "gold standard" to treat diabetic foot ulcers due to its superior healing rate and ability to reduce pressures at the site of ulceration (1). Total contact casts have been shown to heal a higher proportion of DFUs and to heal them faster than some types of removable cast boots and shoe-based systems (SBS). Only a very small minority of clinicians who identify themselves as wound experts (1.7%-6%) use total contact casts. Many other clinicians do not have the training or the resources to use total contact casting. In addition many patients have conditions that make the TCC contraindicated or ill-advised. In those cases alternative devices such as the removable cast walker (RCW) or a shoe-based system has to be used. (2),(3)

According to the International Working Group on the Diabetic Foot, when a TCC or other knee-high device is contraindicated or cannot be tolerated, you should consider offloading with a forefoot offloading shoe, cast shoe, or custom-made temporary shoe to heal a neuropathic plantar forefoot ulcer in a patient with diabetes when the patient can be expected to be adherent to wearing them. Because an appropriate foot-device interface is recommended for use in the TCC and knee high devices it is implied that these should also be included in the shoe-based systems (4)

Numerous over the counter and professionally modified off-loading devices have been produced to provide a foot-device interface that would reduce pressure on ulcerated areas of the foot. Few of these have studies with data to show that they demonstrated any degree of pressure reduction or improvements in healing outcomes.(5-8) Only a limited number of studies have been done to look at the offloading capabilities of individual innersoles and less have looked at the comparative effectiveness of foot-device interfaces. (9-13)

Abstract

This study evaluated the effectiveness of the FORS-15 Offloading Innersole® (Saluber, San Zeno, Italy) in reducing pressure under focal areas on the sole of the foot. A simulated pressure site was created under the right first metatarsal head. Normal subjects walked wearing a surgical shoe while pressure was measured under the foot in three conditions: no insole, unmodified insole and insole modified for offloading. The study was able to demonstrate an average 43% reduction in maximum pressure under the first metatarsal head when using the modified insole.

Data

	Peak submetatarsal 1 pressure (kPa)					
	Cond 1	Cond 2	Cond 3	change 1>2	Change 2>3	Change 1>3
S1	165.913	162.7826	130.087	1.9%	20.1%	21.6%
S2	162.7826	128.4638	102.029	21.1%	20.6%	37.3%
S3	121.7391	96.69565	48.34783	20.6%	50.0%	60.3%
S4	74.08696	65.73913	43.13043	11.3%	34.4%	41.8%
S5	55.30435	43.47826	29.56522	21.4%	32.0%	46.5%
S6	59.36232	29.56522	21.91304	50.2%	25.9%	63.1%
S7	65.3913	66.43478	45.21739	-1.6%	31.9%	30.9%
S10	82.43478	18.08696	12.86957	78.1%	28.8%	84.4%
S11	104	99.13043	93.56522	4.7%	5.6%	10.0%
S12	169.3913	142.9565	99.47826	15.6%	30.4%	41.3%
S13	77.91304	34.08696	44.52174	56.3%	-30.6%	42.9%
S14	61.21739	47.65217	38.26087	22.2%	19.7%	37.5%
S15	124.1739	107.1304	66.31884	13.7%	38.1%	46.6%
			mean change	24.3%	23.6%	43.4%
			mean deviation	23%	19%	19%

Results

The average pressure reduction by the insole alone was 24.3% and with the pixels removed 43.4%, reflecting an average additional pressure reduction of 19.1% when the pixels are removed

Procedures and Data Analysis

After giving consent each subject was examined and the first metatarsal head of the right foot identified and a 1/4 inch thick 1.5 inch circle of skived adhesive felt prepared to apply to the plantar skin over the area.
The subjects were then observed in three conditions: barefoot in a standard surgical shoe, barefoot in the surgical shoe with an unmodified insole, and barefoot in the surgical shoe with an insole modified to remove pixels from under the designated areas of high pressure. The subjects were allowed time to acclimate to the off-loading device and then data was collected while subjects walked in the surgical shoes. Dynamic plantar pressures were collected at 100 Hz while subjects walked at self-selected speeds in a straight line. The FScan® in-shoe dynamic pressure measuring system and software (Tekscan, Boston, MA) was used to record pressures ranging from 30 – 1,500 kPa. Pressure insoles were calibrated for each subject to ensure accurate data acquisition.
Three gait trials were performed five mid-gait steps identified from each trial for evaluation. Pressure distributions were calculated for a total of 15 steps for each subject. There were consent issues regarding subjects 8,9 so they were eliminated from the data pool.
Custom written Matlab (Mathworks Inc, Natick, MA) codes will be used to visualize maximum pressures in ten regions of each foot. Descriptive statistics for each condition were calculated and conditions compared using the Student t-test. No funding was received from any outside source for this study.

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12. Lavery LA, Vela SA, Lavery DC, Quebedeaux TL. Reducing dynamic foot pressures in high-risk diabetic subjects with foot ulcerations. A comparison of treatments. Diabetes Care. 19(8):818-21, 1996.
13. Giacalone VF, Armstrong DG, Ashry HR, Lavery DC, Harkless LB, Lavery LA. A quantitative assessment of healing sandals and postoperative shoes in offloading the neuropathic diabetic foot. J Foot Ankle Surg. 36(1):28-30, 1997.

Evaluated Off-Loading Insole

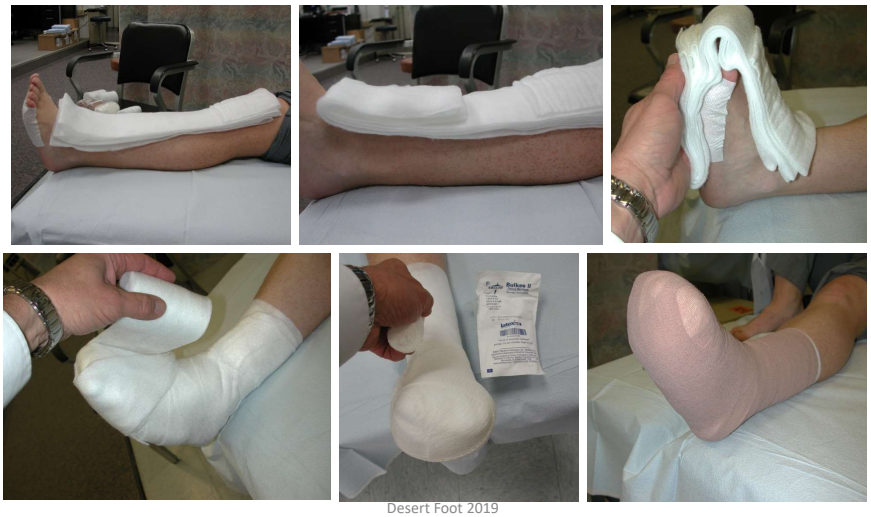
Felted-Foam Dressings

- Felted Foam forefoot ulcer study comparing to TCC, healing shoes, and a walking splint
- Results: 93% of the FF ulcers were healed in 12 weeks as compared to the TCC 92%

Birke JA, Pavich MA, Patout CA, and Horswell R. Advances in Skin and Wound Care, September/October 2002

Football Dressing for Neuropathic Forefoot Ulcerations

Rader AJ, Barry T. Wounds 2006;18(4):85-91



Rader AJ, Barry TP. The football: an intuitive dressing for offloading neuropathic plantar forefoot ulcerations. Int Wound J. 2008 Mar;5(1):69-73.

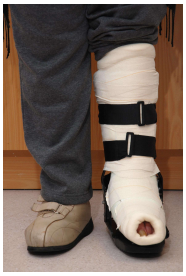
- Wound healing rates were compared with published data on the total contact cast (TCC) and instant total contact cast (iTCC). Overall wound healing rates for University of Texas Health Science Center class 1A, 1B, 1C, 1D, 2A, 2B, 2C and 3B plantar forefoot ulcerations was **2.91 weeks** with a 95% confidence interval of **2.36-3.47 weeks** for complete wound epithelialization.

Patellar Tendon Bearing Braces

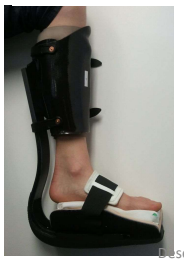
- Increased weight reduction by transfer of weight to the leg cone
- Forefoot pressure reduction with a fixed ankle and a rigid rocker sole

Limb-Load and Other Offloading Braces







Torch Offloading Boot


A Word About CROW Boots

- Charcot Restraint Orthopedic Walker
- Supposed to be a PTBO – Patellar Tendon Bearing Orthosis
- PTB Function rarely works
- $$$$
- Reserve for Unstable Inoperable Charcot Foot
- MUST have a rocker sole

Thank you!!!

- Please do not hesitate to contact me if there is anything at all that that I can do for you:

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215-255-5994

TEMPLE UNIVERSITY

Mobility/Activity Dysfunction Contributing to Lower Extremity Impairments

Faculty:
Pamela Scarborough, PT, DPT, CWS, CEEAA
Director of Public Policy/Education American Medical Technologies
Certified Wound Specialist
Certified Exercise Expert for Aging Adults

Disclosure

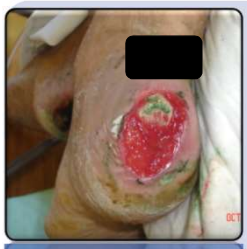
- Employed by American Medical Technologies

Objectives

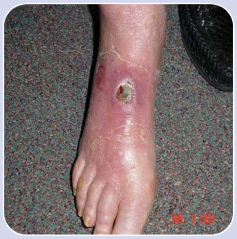
At the end of this presentation participants should be able to:

- Recognize how edema negatively affects normal musculoskeletal function in the lower extremity
- Describe the venous pumps in the lower extremity
- Verbalize what types of exercises or activities would be appropriate for an exercise prescription for lower extremity edema reduction

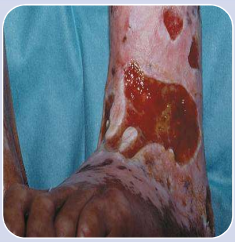
Primary Etiologies Leading to Integumentary Failure in the Lower Extremity



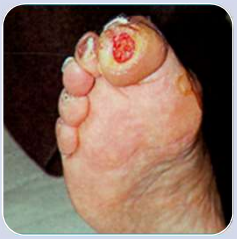
Pressure Injuries



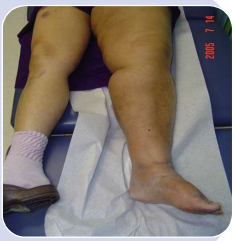
Peripheral Arterial Disease



Venous Insufficiency

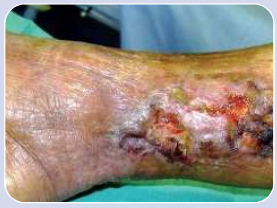


Diabetic Neuropathic Foot Ulcers

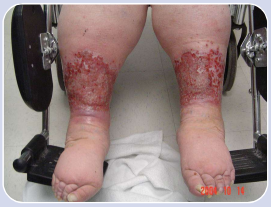


Lymphedema

Mixed Etiologies in Lower Extremity Wounds

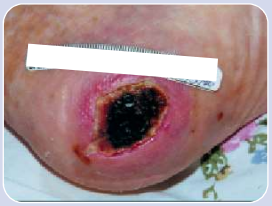


Arterial and Venous



Venous Insufficiency and Lymphedema

AKA Phlebolymphedema



Pressure and Arterial

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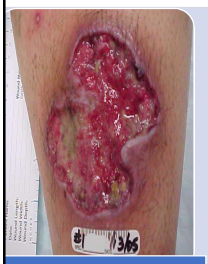
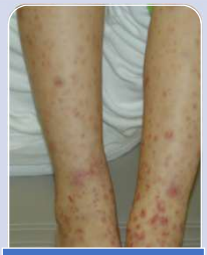
Autoimmune & Atypical Lower Extremity Wounds



Pyoderma Gangrenosum

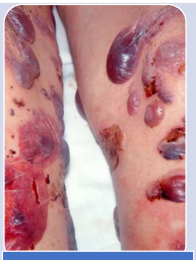
Beware of Pathergy Effect

Associated with ulcerative colitis,



Vasculitis

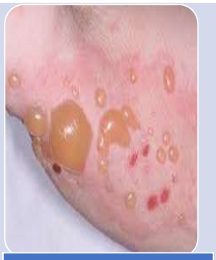
Inflamma-tion and destruction of blood vessels



Pemphigus Vulgaris

Auto-antibodies of epidermis

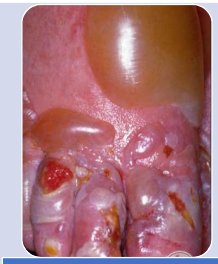
Oral lesions seen 1st



Bullous Pemphigoid

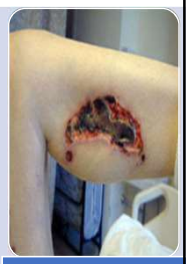
Most common auto-immune dermatosis

(Allergy to one's own



Bullous Diabeticorum

Spontaneous non-inflammatory blistering unique to patients with diabetes



Atypical Wounds

Host of different etiologies often associated with autoimmune diseases

JAMA Dermatology April 2016 Volume 152, Number 4 (Reprinted

Edema and The Musculoskeletal System

- Patients with VLUs have decreased AROM compared with patients diagnosed with venous disease without ulcers
- Some patients diagnosed with VLUs also have undiagnosed neuropathy
- Reduction in leg circumference was associated with improvement in total inversion and eversion AROM
- Improvement in total dorsi/plantar flexion was associated with reduction in leg pain
- Compression increases venous return and lymphatic flow; decreased leg circumference
- Leg circumference to decrease may cause decrease interstitial inflammation
- Decreased inflammation and increased ROM may result in decreased leg and wound pain

The Undervalued Intervention

The Exercise Prescription

Exercise is as powerful an intervention as many drugs…without the side effects

There's a reason it is called Exercise Science

When to Start the Exercise Prescription

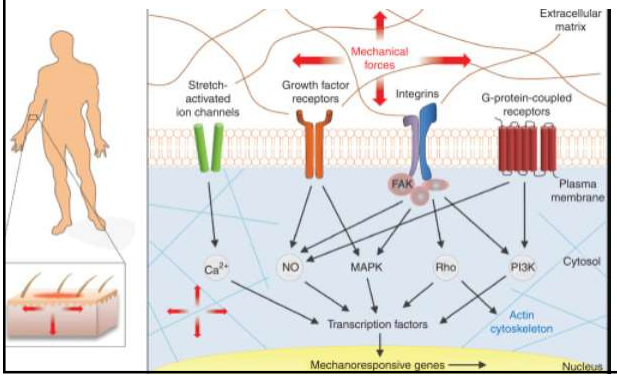
- Before integumentary failure – primary care healthcare providers
  - Prevention of or early diabetes
  - Early venous insufficiency
  - Early lymphedema
- When open wounds present, unless specific exercise contraindicated (e.g. walking for exercise with an DFU)
  - Provide non-weight bearing physical activities
  - Consider: many non or reduced weight-bearing exercises provide meaningful outcomes in sitting and supine positioning

Benefits of Exercise with Wound Healing

- Upregulates blood flow delivering more oxygen to tissues, which is crucial to healing process
- Increases collagen formation; building block of granulation tissue
- Gradient of oxygen from the wound to the periwound area is a strong stimulus for angiogenesis necessary to form new capillaries
- Increased oxygen delivery also increases the killing capacity of white blood cells, essential to handling the wound bioburden
- Increased blood flow also brings about, through mechanotransduction, an increase in anti-inflammatory substances (e.g. NO, prostacyclin, etc) while downregulating inflammatory substances (e.g. tumor necrosis factor, adhesion molecules)

Mechanotransduction Increased with Exercise/Activity

- Conversion of physical stimuli into biochemical responses
- Mechanical force sensed by integumentary systems; activates multiple intracellular signaling pathways important for healing



- Mechanoreceptor Complexes/Cells Affected by Exercise/Activity
  - Ion channels
  - Growth factor receptors
  - Integrins
  - Nitric Oxide
  - Mitogen-associated protein kinases (MAPKs)
  - Rho GTPases
  - Keratinocytes
  - Fibroblasts
  - Nociceptors
  - Etc.

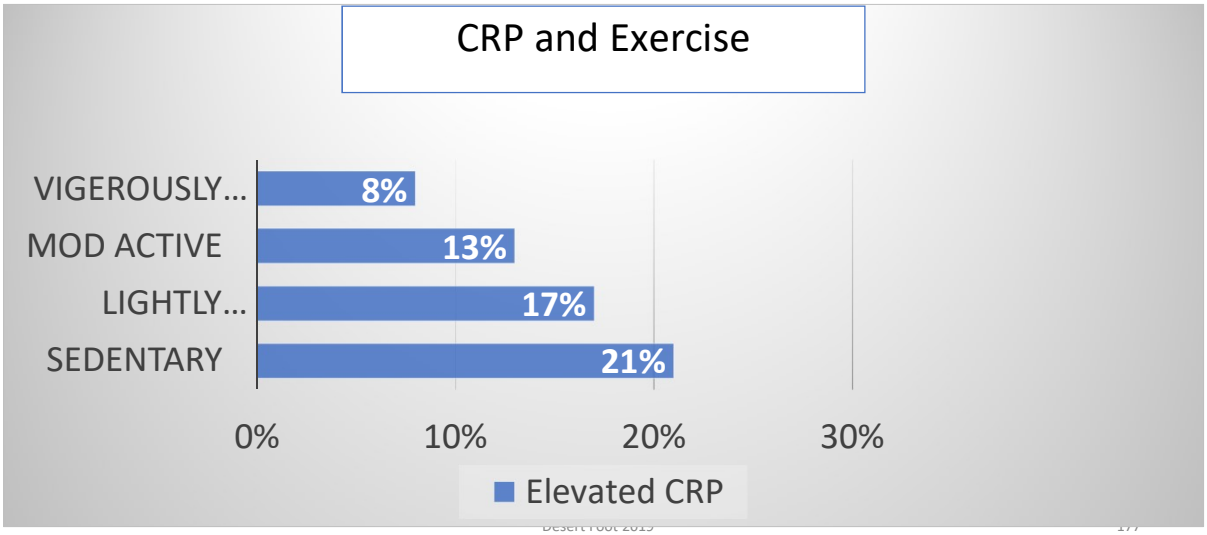
V. W. Wong, S. et. al J. Pushing Back: Wound Mechanotransduction in Repair and Regeneration. Invest. Dermatol.2011, 131, 2186. 175

Exercise Reduces Inflammation

- Chronic Inflammation - major causes of delayed healing
- Research shows that wounds with low levels of inflammation heal more quickly and completely
- Exercise and other physical activity have been shown to reduce the level of inflammatory markers in the blood, thus helping promote healing.
- Reduced inflammation may also provide palliative benefits by decreasing pain and discomfort in wounds.

Keylock K, Young H. Delayed wound healing: can exercise accelerate it?. Int J Exerc Sci. 2010;3(3):70–8. 176

C-Reactive Proteins – Inflammatory Markers



How does exercise physiologically impact Chronic Wound Healing ?

- Evidence that the production of reactive oxygen species and resulting damage is increased in the aged and diabetic populations.
- Strong evidence that suggests moderate aerobic exercise increase anti-oxidant enzyme activities which can prevent damage caused by free radicals. (5)
- Additional research needs to be conducted to confirm the mechanisms responsible for the effects of exercise in obese and diabetic populations.

Exercise – A Wound Care Intervention

- Low risk
  - Avoid side effects of medications, surgical procedures
- Cost effective
- Exercise program must be tailored to individual
  - Frequent review and modification maybe necessary
- Strong evidence that exercise can improve healing time

Exercises for Chronic Wound Etiologies

Diabetes Exercise/Activities

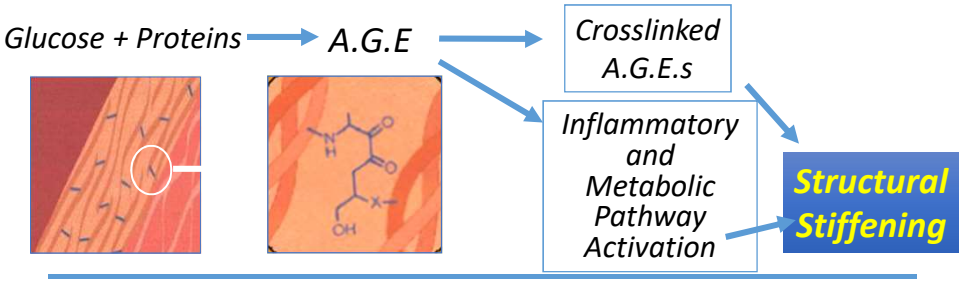
- Issues ROM of foot/ankle – patient prone to equinus and shortening of Achilles tendon due to A.G.E.s
- Cardiovascular precautions necessary
- Assist blood glucose control
- Decreases depression
- Increases ADL abilities

Diabetes and Physical Limitations

Affects the body systemically due, in part, to A.G.E.s

- Joints
- Blood vessels
- Tendons
- Muscles
- Organs
- Brain – an organ that gets extra mention
- Gait and balance

A.G.E. Pathway in Ageing & Diabetes



An Inevitable Consequence of Ageing, **Accelerated by Diabetes**

- Glucose binds to proteins [non-enzymatic glycation] subsequently form irreversible crosslinks between proteins
- A.G.E.s stimulate multiple inflammatory & metabolic pathways
- **Tissues/organs progressively lose flexibility & function**

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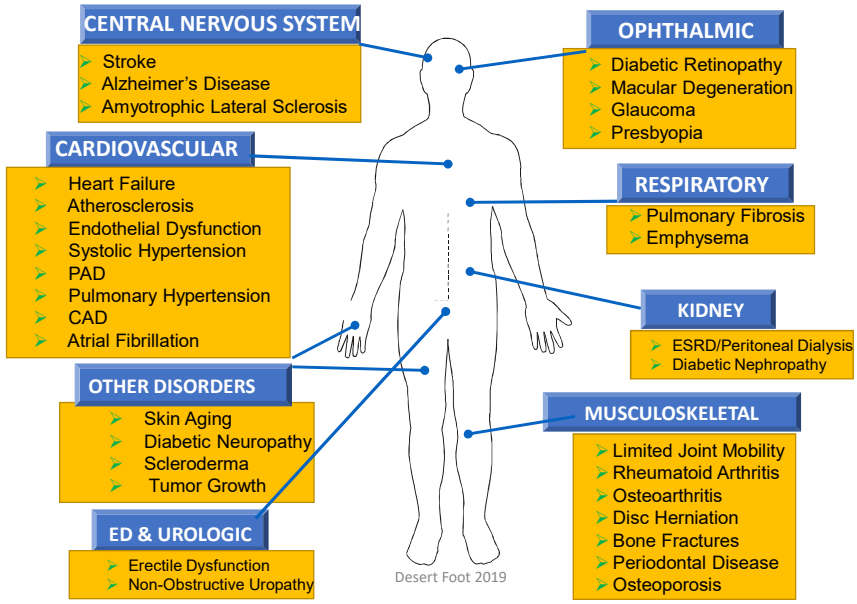
A.G.E.s Cause Or Contribute To Many Diseases



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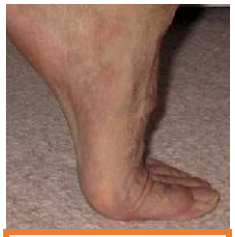
A.G.E. and the Musculoskeletal System

- DM exaggerates the normal aging processes
- Adds to impairments & functional limitations associated with the aging musculoskeletal system
- **AGEs' effects on joint mobility and ROM**, especially in the foot/ankle
- Limited joint mobility of the MTP considered to be a clinical marker of DM-related complications



Normal ROM of MTP Jt

Limited MTP & IP Jt. ROM

Considerations Prior to Exercise Prescription

- ROM
- Strength
- Balance during dynamic and static activities
- Shoe gear
- Level of fitness
- Motivation
- Ability of health care provider design individualized exercise prescription
- Ability of health care provider to monitor exercise prescription

Exercise Considerations for People with Diabetic Foot Complications

- There is always some type of exercise people with DM complications can do
- Not remaining active can lead to developing additional complications and loss of functional capacity
- Charcot Foot or DFU
  - Ankle ROM-stretching
  - Non-weight bearing muscle group strengthening of lower extremity
  - Stationary arm ergometer (arm cycle)
  - Chair exercises

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Important to Know When to NOT Exercise when a Person has Diabetes

- Blood glucose values that are either:
  - too high

  - too low

- Both situations put the individual's well-being at risk
- After exercise muscles are replacing glycogen stores which causes blood glucose to fall further in the hours after exercising

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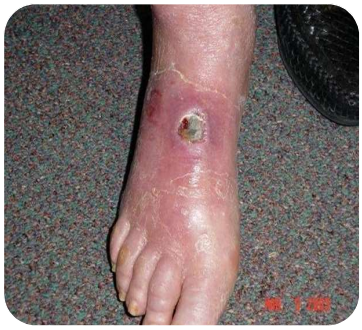
Key Study Results of Walking in People with DM

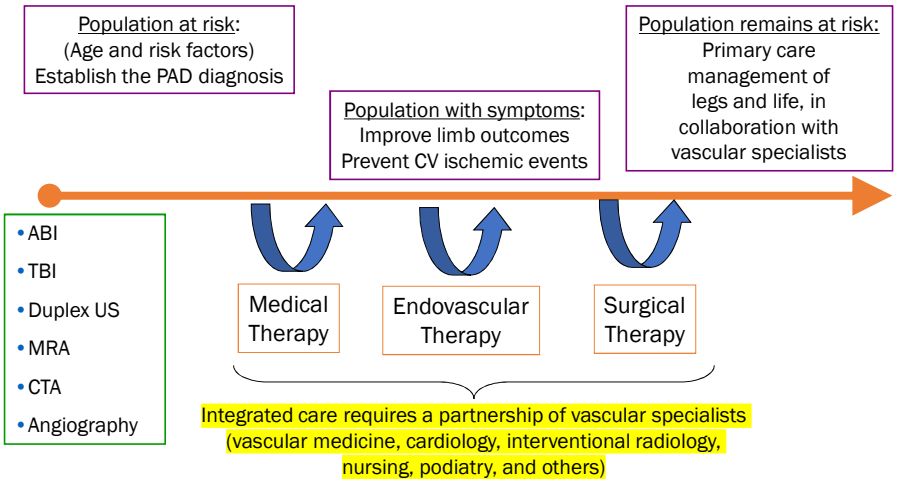
- Lengthened life of people with DM regardless of age, sex, race, body mass index, length of time since diagnosis, and presence of complications or functional limitations.
- People with DM who walked for exercise at least 2 hours a week = ~ **18 minutes/day**-lowered their mortality rate from all causes by 39 %.
- Risk of death from heart disease could by reduced by 34 % by walking at least 2 hours per week.
- Those engage in at least 2 hours of any leisure-time physical activity a week had a 29 % lowered mortality risk compared with people who are inactive.
- Walking 3-hours = **26 minutes/day** to 4 hours= **35 minutes/day**…a week reduced mortality 54 %.
- While the study found that walking at **moderate-intensity** levels reduced mortality, **no reduction in mortality was associated with more intense levels of walking**, indicating that vigorous levels of exercise are not as beneficial for people with type 2 diabetes.

CDC: Relationship of Walking to Mortality among U.S. Adults with Diabetes

Peripheral Arterial Disease and Exercise Prescription



The PAD Guideline is Intended to Guide Lifelong Primary to Specialty PAD Care



ABI=ankle-brachial index; CTA=computed tomographic angiography; CV=cardiovascular; MRA=magnetic resonance angiography; TBI=toe-brachial index; US= ultrasound.

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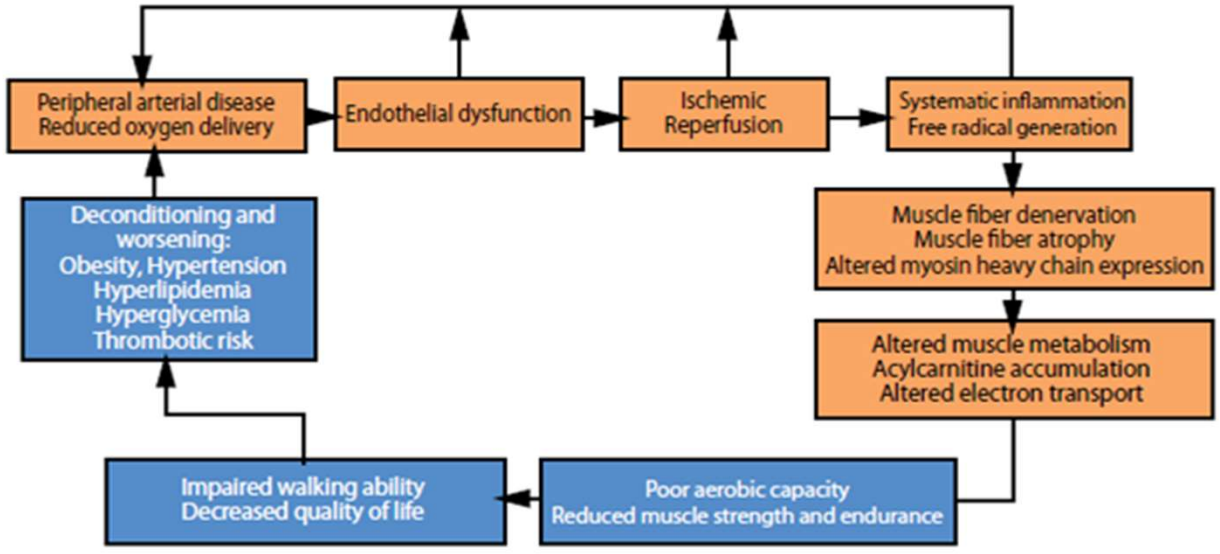
Cycle of Disability with Intermittent Claudication



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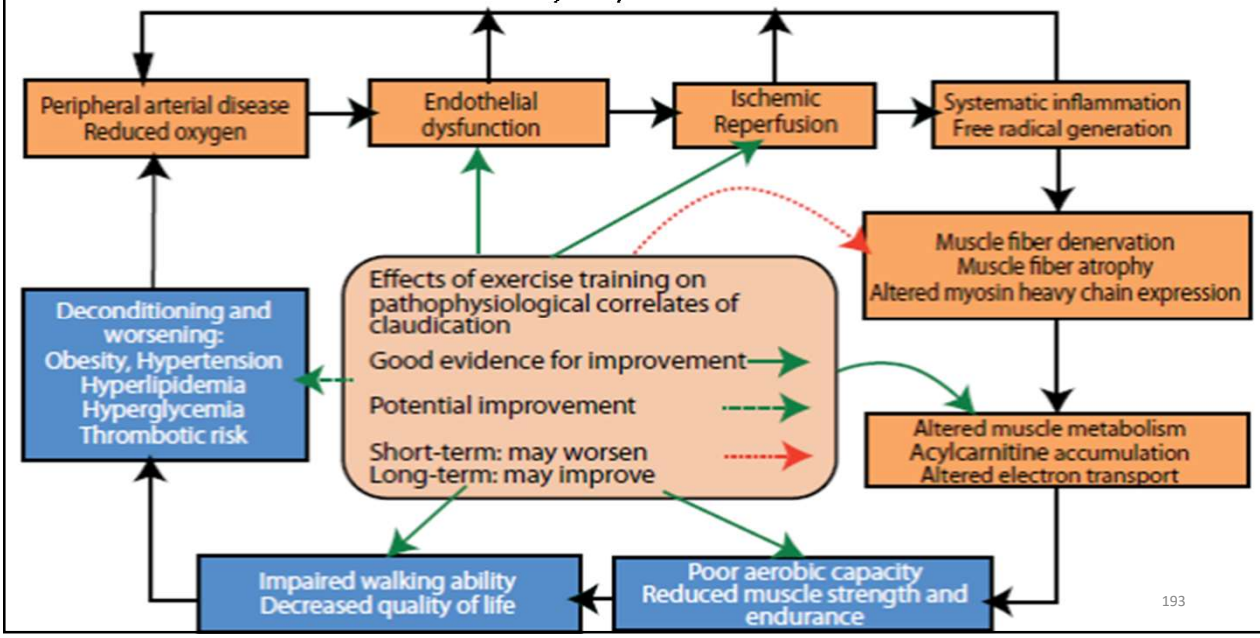
Mechanisms by Which Exercise May Improve Function and Symptoms in Claudication



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Structured PAD Walking Programs

- Patients should be under the care and guidance of a clinician/professional trained and competent in general exercise prescriptions
- Due to correlation of CAD and PAD a cardiovascular assessment is advised prior to beginning an exercise program if not active or used to exercising
- E.g. Gardner-Skinner Protocol
- Treadmill based testing and treatment with other exercise modalities - stationary bikes, UBE's, recumbent steppers etc.
- Average visits 15-20; *until* goal is achieved (50 minutes at 2.0 MPH, 0% incline)
  - Typically recommended in literature is 3x/wk for 12 weeks
- Outcomes measured by Focus on Therapeutic Outcomes (FOTO) or vascular specific questionnaire VascuQOL-6

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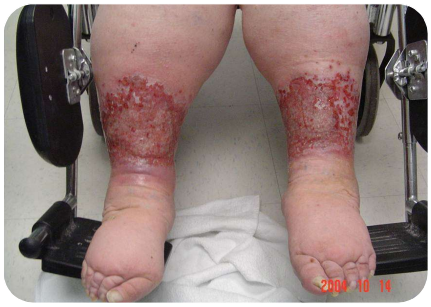
Chronic Venous Insufficiency, Lymphedema and Phlebolymphedema Exercises for the Lower Extremities



Phlebolymphedema

Attribution: Dr. John McDonald

Benefits of Exercise for Lower Extremity Swelling related to CVI, Lymphedema and Phlebolymphedema

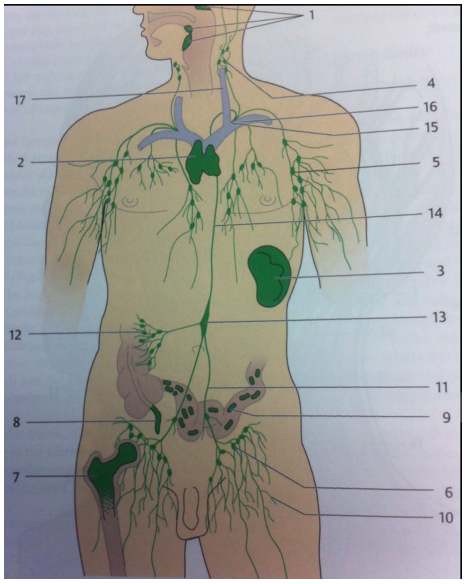
- Why exercise in general
  - Positive impact on a healthy lifestyle
  - Improvement of general well being
  - Increased energy level
  - Stress and weight management
- Additional benefits of exercises for individuals at risk of, or have lymphedema
  - Improved limb flexibility
  - Improved range of movement
  - Increased lymphatic drainage and venous return from edematous areas
    - Contributes to reduction of limb size and subjective limb symptoms

Specific Exercise Considerations for Lymphatic System

- Muscle activity and diaphragmatic breathing - considerable impact on venous blood returning from extremities back to central system;
- Which positively affects fluid management within interstitial spaces;
- Increased venous return is of particular importance for those individuals affected by lower extremity lymphedema.
- Research indicates that transport of lymph fluid and proteins from edematous areas increases during and after exercises.

Abdominal/Diaphragmatic Breathing

https://www.youtube.com/watch?v=hp-gCvW8PRY



https://www.youtube.com/watch?annotation_id=annotation_352662&feature=iv&src_vid=ZLyT_0Tr40M&v=rMljOuz_l_Q

Lymph fluid from the lower extremities passes through these deep lymphatic structures and an increased flow of lymph, particularly in the thoracic duct, results in improved lymphatic drainage from the lower extremities.

Individuals affected by lymphedema of the leg greatly benefit from diaphragmatic breathing exercises, especially when combined with a comprehensive decongestive exercise regimen.

Effects of Diaphragmatic Breathing

- Downward and upward movement of diaphragm in deep abdominal breathing is essential for the sufficient return of venous and lymphatic fluid back to the central system;
- Movement of diaphragm, combined with the outward and inward movements of the abdomen, ribcage, and lower back, promotes general well-being, relaxation, peristalsis and return of venous blood back to the heart
- Considerable decongestive effects on the lymphatic and venous systems in combination with these additional benefits make abdominal breathing exercises also a valuable tool for the treatment of upper extremity lymphedema.
- Diaphragmatic breathing exercises with the primary goal of decongestion is best performed lying on back (supine) with the knees bent and head supported with a pillow; however, they may also be performed sitting on a stool or chair **without leaning back.**

How to perform abdominal breathing in the supine position: (Link to a Video: https://www.youtube.com/watch?v=kgTL5G1ibIo

1. Lie on your back on a flat surface (or in bed), with your knees bent and your head supported. You may use a pillow under your knees to support your legs. Place one hand on your upper chest and the other on your belly. This will allow you to feel your diaphragm move as you breathe.

2. Breathe in slowly through your nose so that you feel your stomach moving out against your hand. The hand on your chest should remain as still as possible.

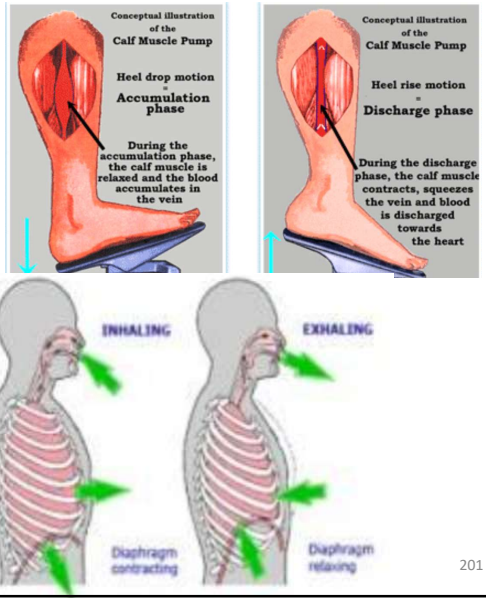
3. Tighten your stomach muscles, letting them fall inward as you exhale. The hand on your upper chest must remain as still as possible. To pronounce the exhaling you may let the exhaled air flow through pursed lips.

It is recommended to first practice abdominal breathing exercises 5-10 minutes about 3-4 times per day. If no dizziness or discomfort is noted, you may gradually increase the amount of time you spend doing this exercise.

Here is another link to video tutorial providing a 3-D view of the diaphragm during abdominal/diaphragmatic breathing: https://www.youtube.com/watch?v=hp-gCvW8PRY

Venous Blood Return to Heart/Central System by Two Mechanism

1. Peripheral venous pump mechanism (calf pump)
2. Pressure changes within abdomen
   - Diaphragm drops, rib cage expands up and out creating negative pressure in the central cavity sucking blood back to the central system
   - Why diaphragmatic breathing an important exercise



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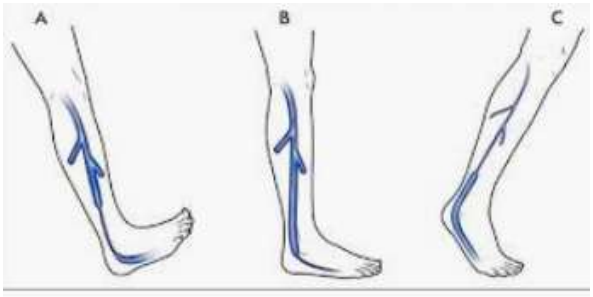
Venous Pumps in the Lower Extremity

- Proximal calf muscle pump = A
- Distal calf "piston" pump = B
- Venous foot pump = C
  - Activated by compression of body weight or planter muscle contractions during gait

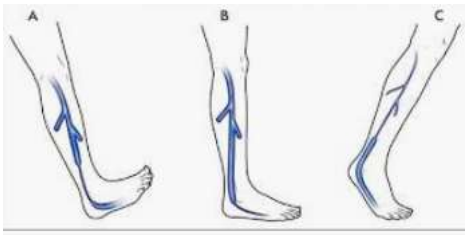


The venous system of the foot: anatomy ...
phlebolymphology.org

Phlebolymphology – Vol 22. 2. 2015

Musculoskeletal Movement for Volume Return

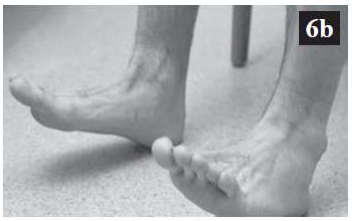
- During ambulation these three vein-pumping systems are synchronized to form a complete network for volume return to central system



The venous system of the foot: anatomy ...
phlebolymphology.org

Phlebolymphology – Vol 22. 2. 2015

- Moderate muscular movement of legs in **seated position** also activates the pumping mechanisms and reduces mean distal vein pressure

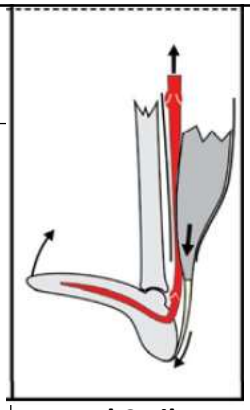


What Does Gait Have to do With It?

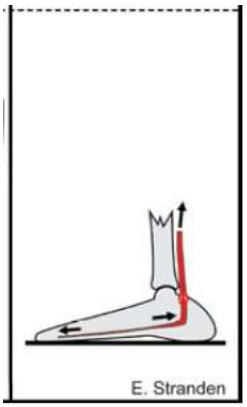
Heel strike – dorsiflexion (passive or active): bulk of calf muscle descends within facial sheath, expels blood into distal veins like a piston

Foot flat/stance – weight bearing:-tarso-metatararsal joints extended; tarsal arch flattens, veins stretch ejecting blood content

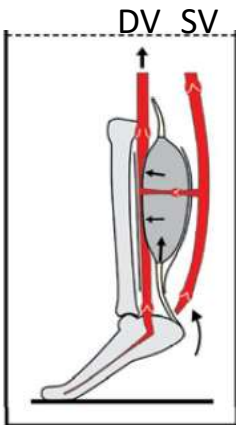
Toe-off/plantar flexion- gastroc/soleus muscles contract strongly during push-off; ejects blood into the proximal collecting vein



Heel Strike
Activates distal calf pump



Foot Flat/Mid-Stance
Activates venous foot pump

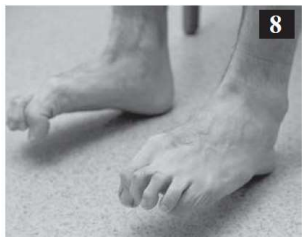


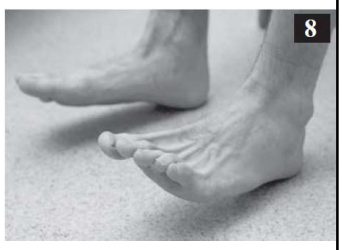
Push/Toe-Off
Activates calf msl Pump

StrandenE. Edema in venous insufficiency. In: Wittens C, ed. *Best Practice in Venous Procedures*. Turin, Italy: Edizioni Minerva Medica; 2010:131-140.

The efficacy of forceful ankle and toe exercises to increase venous return: A comprehensive Doppler ultrasound study.

- Conclusion
- All exercises (in this study) achieved significant increases in peak systolic velocity compared to baseline.
- Ranking showed that forceful ankle dorsiflexion, plantarflexion and forceful flexion of all toes yielded the highest mean peak systolic velocity values





Practice Point: Include strong toe flexion with ankle ROM exercises

Phlebology. 2018 Jun;33(5):330-337. doi: 10.1177/0268355517706042. Epub 2017 May 7. Accessed 11/29/19

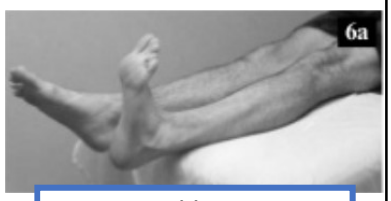
Ankle exercise and venous blood velocity.

Paul D. Stein, Abdo Y. Yaekoub, James E. Denier

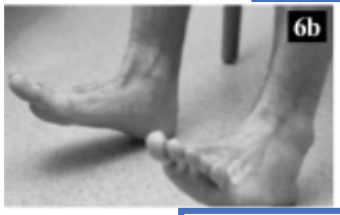
Published in Thrombosis and haemostasis 2009

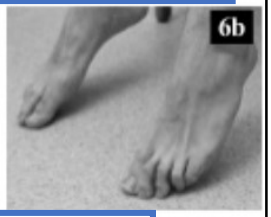
DOI:10.1160/TH08-09-0615

- Ankle exercise increase venous blood velocity in both supine and sitting positions, thereby transiently reducing a tendency toward venous insufficiency
- Repetitive dorsiflexion while sitting predominantly increases blood velocity in the popliteal and common femoral veins



Supine Ankle Exercises





Sitting Ankle Exercises

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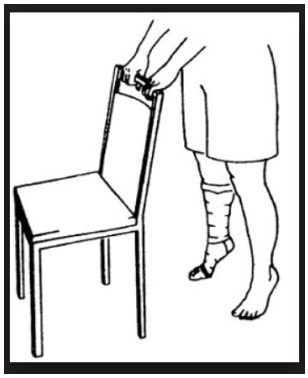
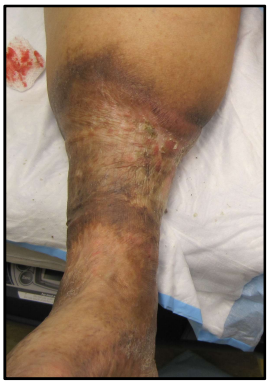
Summary: Muscle Movement/Exercise for CVI/Lymphedema/Phlebolymphedema

- Lymphedema system does not have an active pump to propel lymphatic fluid back to the bloodstream
- Effective lymph flow depends on sufficient muscles activity and joint range of motion
- Decongestive exercises are most effective if performed with patient wearing compression garments or bandages
- Non or minimally ambulatory patients **CAN** do these movements (sitting/lying)
- If not including exercise as part of the treatment plan you are leaving out ½ of the effectiveness of your compression interventions…actively engaging the calf/foot pumps



Chronic Venous Insufficiency (CVI) and Venous Hypertension

- The moving ankle joint and calf/foot pump in the lower extremity veins work together to "pump" venous blood back up to the central system



What Needs to be in Place for the Lower Extremity to Function Optimally?

- Range of Motion – specifically ankle
- Muscle strength and endurance
- Balance when standing, walking

Exercise with Chronic Venous Insufficiency (CVI) and Lower Extremity Lymphedema

- **Key is to engage calf muscle pump**
- Assess does patient have adequate ROM/strength/phases of gait
  - ankle/great toe range of motion (> 90 degrees ankle dorsiflexion)
  - Lower extremity strength : ankle (DF/PF), knee (flex/extension), hip(extension)
- Assess gait, foot wear and need for assistive device

Exercise with Chronic Venous Insufficiency (CVI)

- **Recommended exercises:**
  - ROM/stretching
  - Lower extremity strengthening program (*TheraBand ™*)
  - Ambulation
  - Ankle pumps
- Diaphragmatic breathing exercises in conjunction with above
  - Negative pressure increase volume return
- Difficult population to motivate BUT encourage group exercise, walking programs

Limited Mobility – No Problem!!!

How to Exercise with Limited Mobility ...

Conclusion

- Exercise should be considered a core component of treatment for many chronic wound etiologies
- Without the exercise prescription the plan of care for several of these etiologies (i.e. venous insufficiency, lymphedema, arterial insufficiency, diabetes related musculoskeletal impairments, and in some cases pressure ulcers, **is incomplete**
- Consider adding exercise to your plans of care and track outcomes
- There is significant evidence for exercise for several etiologies particularity venous insufficiency, PAD, diabetes in general and in the presence of DM complications
- Exercise will also help prevent recurrence of these wounds

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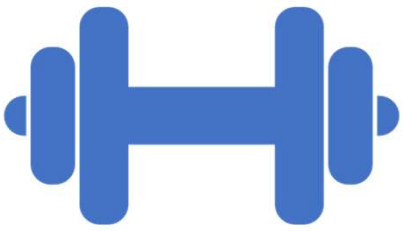

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Please raise your hand if you think exercise/activity prescriptions should be considered a component of the standard of practice for wound management for people who qualify physically, cognitively, and by ability and motivation.

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The Exercise Prescription-Who

- Who should recognize the need for the exercise/activity prescription for patients with chronic wounds?
  - The practitioner
- Who should order the evaluation and treatment for an exercise/activity program to improve wound healing?
  - The practitioner
- Who should design the exercise/activity program?
  - Someone educated and trained in the exercise prescription with knowledge of indication, contraindications, precautions, and modification of movements to keep the patient safe AND who is trained in chronic wound management.
    - Physical therapists
    - Clinical exercise physiologist ??? Seeing CEP more frequently working with patients with DM and cardiac rehab

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The Journey of a Thousand Miles Begins with a Single Step
Lao Tzu

Take the first step towards improved function using the exercise prescription for your patients.

Take the first step to fitness for yourself.

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Thank you!!!

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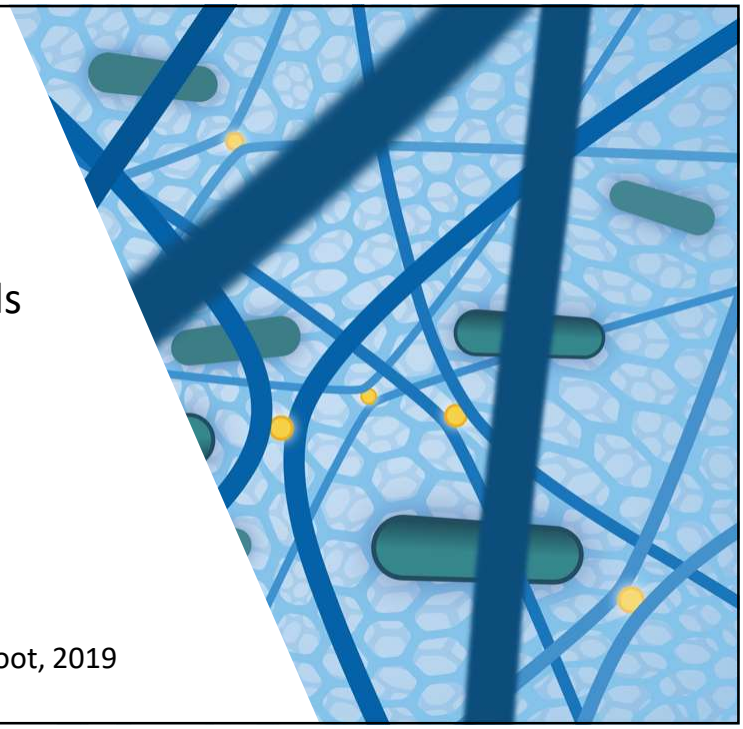
Biofilm and Chronic Wounds
The problem. The solution. The evidence.



Marie Clarke, MSN, RN, CWCN
Lower Extremity Boot Camp, Desert Foot, 2019

Disclosure

Marie Clarke is a Clinical Wound Care Specialist
for
3M Medical Solutions Division, 3M Health Care

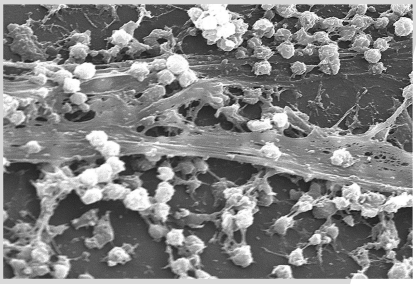
Objectives

At the end of this presentation participants should be able to:

- Describe the constituents of biofilm makeup
- Verbalize how biofilms form
- Recall why bacteria in biofilm are difficult to eliminate

What is biofilm?

Biofilm is a community of pathogens enveloped within a complex structure of entangled polymers strengthened with metallic bonds.[1,2]



Source image: https://phil.cdc.gov/Details.aspx?pid=7488

Image courtesy of CDC/Rodney M. Dolan, PhD. and Janice Haney Carr

Community of pathogens

Multiple species of bacteria and fungi living together.

Entangled polymers

Microbes secrete a protective matrix called EPS (extracellular polymeric substance) made from polymers including proteins, glycolipids, polysaccharides and DNA.

Metallic bonds

Metallic ions bind polymers of the EPS together forming a resilient-barrier.

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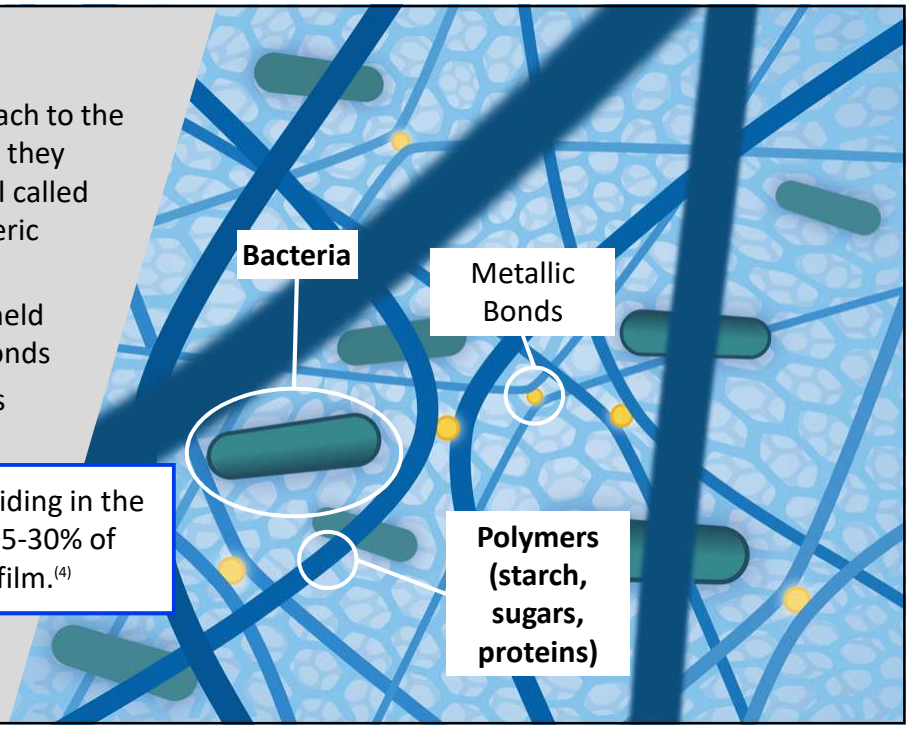
A closer look

As bacteria and fungi attach to the wound bed and multiply, they secrete a sticky, moist gel called EPS (extracellular polymeric substance).[3]

- Made up of polymers held together by metallic bonds
- Envelopes and protects the microorganism

Microbial cells residing in the EPS make up only 5-30% of the volume of biofilm.[4]



Most bacteria exist within biofilms

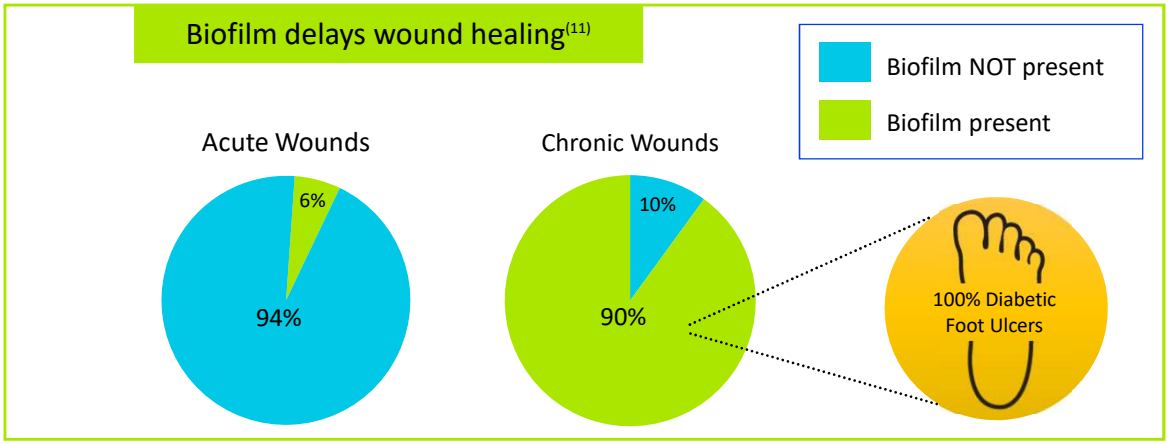
10%
OF BACTERIA are PLANKTONIC or FREE-FLOATING

90%[5]
of BACTERIA exists in BIOFILMS

Bacteria protected by biofilm EPS (extracellular polymeric substance) can be 1000x more tolerant to antibiotics than planktonic bacteria.[8,9]

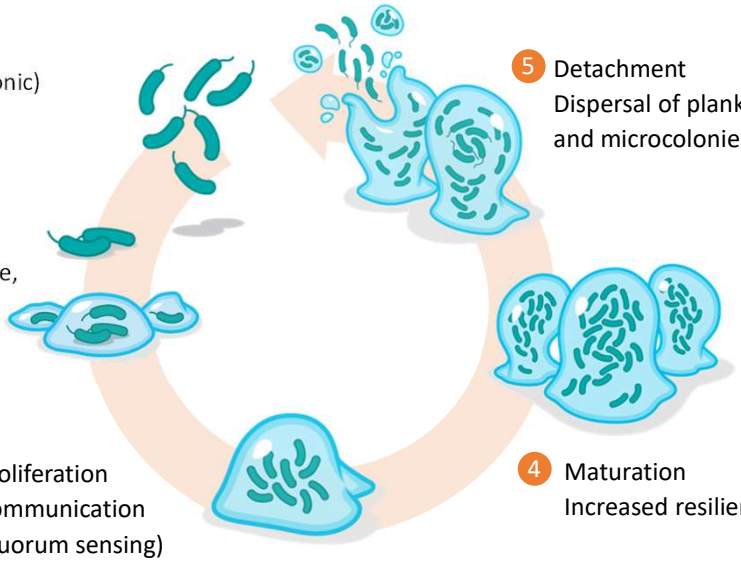
Biofilm is present in most chronic wounds

- 6% of acute wounds and up to 90% of chronic wounds[8]
- 100% of diabetic foot ulcers[10]



How do biofilms form?

1. Colonization
   Free-floating (planktonic) pathogens
2. Attachment
   Permanent adherence, secretion of EPS
3. Proliferation
   Communication (quorum sensing)
4. Maturation
   Increased resilience
5. Detachment
   Dispersal of planktonic bacteria and microcolonies



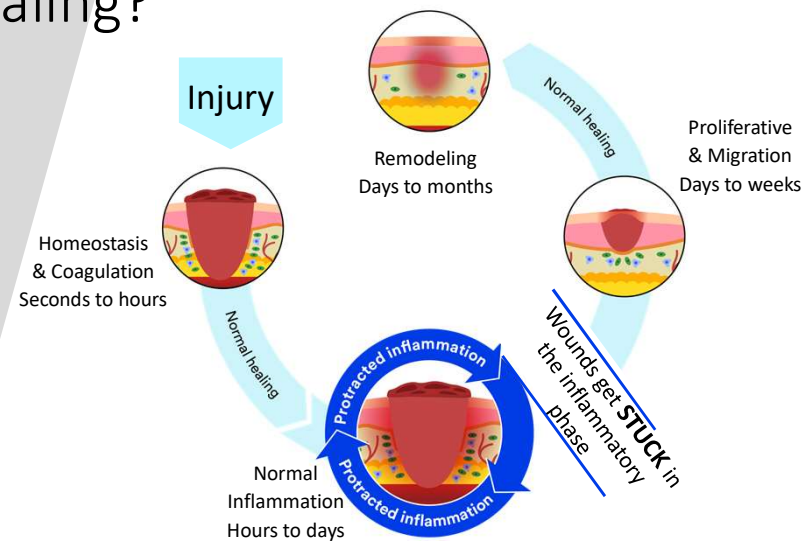
Adapted from: International Wound Infection Institute (IWII) *Wound infection in clinical practice*. Wounds International 2016

How does biofilm inhibit wound healing?

During the inflammatory phase of wound healing, neutrophils and macrophages release proteases (MMPs and elastase) in attempt to remove biofilm.

Ultimately, the ineffective inflammatory response prevents wounds from healing.[3,4]

The body is susceptible to spreading biofilm-based infection.



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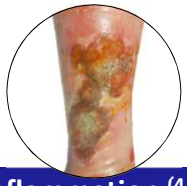
Inflammation and wound healing

vs.



Normal Inflammation

- Increased vasodilation and vasopermeability causing increased exudate
- Immune cell recruitment
- Release of enzymes, cytokines and growth factors
- Bacterial clearance

The result is normal wound healing

Protracted Inflammation [4,12]

- Immune cells continuously stimulated
- Prolonged release of proteases and ROS (reactive oxygen species) which have "off-target effects":
  - Degradation of growth factors, receptors, extracellular matrix
  - Impaired cell proliferation and migration
  - Increased exudate — providing nutrition for biofilm

The result is impaired healing

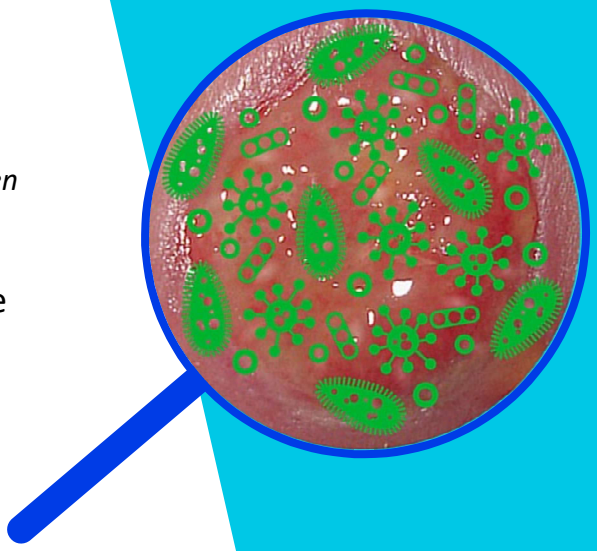
Identifying biofilm in a wound

FACT: You can't necessarily see biofilm.[13]

Biofilm are microscopic structures, often undetectable to the naked eye.

FACT: Routine wound cultures are not an effective method of identifying biofilm.[11]

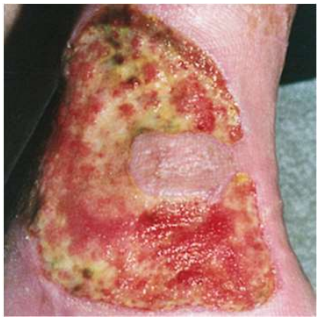
Cultures identify free-floating (planktonic) bacteria, not biofilm bacteria.



Identifying biofilm in a wound

FACT: Classic (overt) signs of a wound infection may not be present when a wound has biofilm.

A comprehensive physical assessment is necessary.

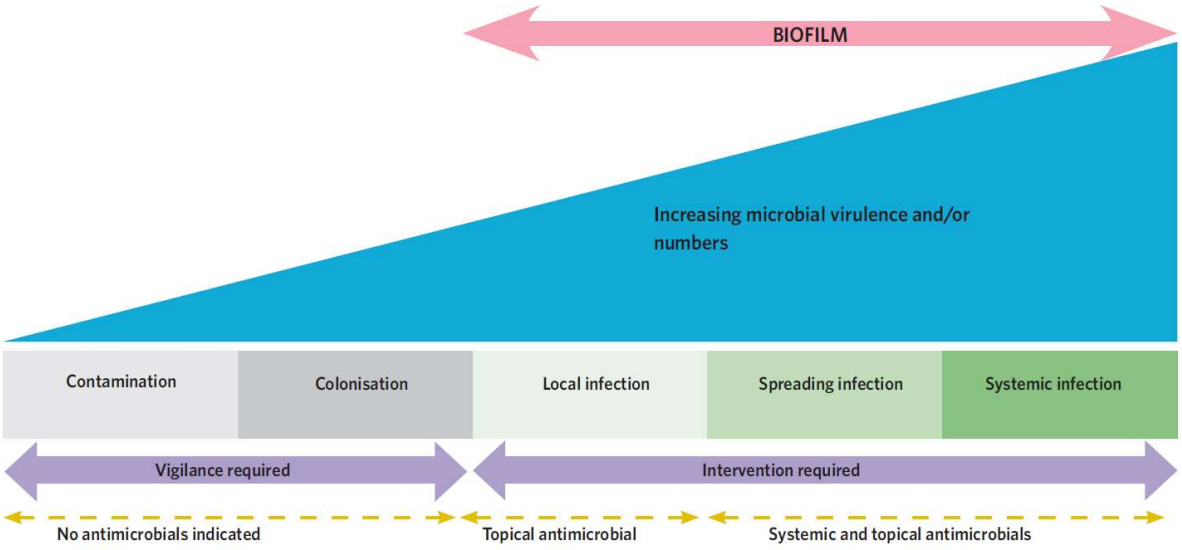


Signs and symptoms of biofilm may include:[13,16]

- Poor quality/friable granulation tissue
- Low-level chronic inflammation (erythema and induration)
- Increased exudate/moisture
- Failure of appropriate antibiotic treatment
- Recurrent slough formation

Delayed healing despite optimal wound management and supportive measures

Wound Infection Continuum



International Wound Infection Institute (IWII) Wound infection in clinical practice. Wounds International 2016

Why are bacteria within biofilm hard to kill?

EPS — Extracellular Polymeric Substance

Blocks effects of white blood cells and antimicrobials agents[3]

Mutual Protection

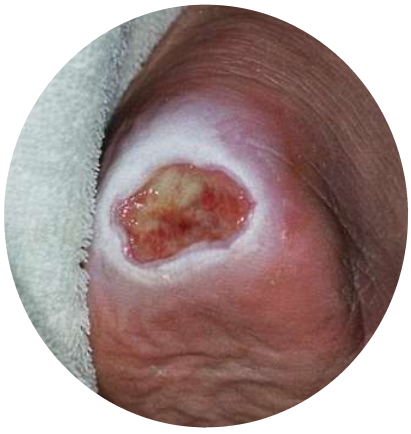
Different species of bacteria communicate and change gene expression to promote survival (quorum sensing)[3]

Hibernation (Quiescence)

Metabolically inactive — antibiotics unable to kill hibernating bacteria[3,14]

Rapid Reformation

Mature biofilm reforms within 24–72 hours after mechanical disruption such as debridement[8,11]



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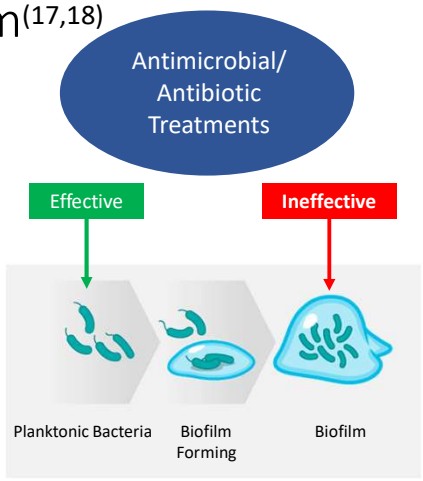
Most topical antimicrobial treatments are not effective in eliminating biofilm[17,18]

- Unable to penetrate into the biofilm EPS matrix
- May be damaging to healing tissue when used at strengths necessary to kill bacteria within a biofilm
- Non-selective — can affect human cells, not just pathogens
- Pathogens within biofilm are metabolically inactive (quiescent) so antibiotics ineffective
- Antibiotics lose effectiveness as bacteria change gene expression (quorum sensing) — can lead to bacterial resistance



Once biofilm has formed, antimicrobial/antibiotic treatments become ineffective

Illustration adapted from: Richards JJ, et al. Controlling bacterial biofilms. Chem Bio Chem. 2009;10(14):2287-2294.

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Optimal biofilm treatment requires:[21]

- The ability to dismantle the EPS structure
- Broad antimicrobial spectrum
- No microbial resistance
- High tissue compatibility
- Prevents biofilm reformation

Dismantle the EPS structure

Destroy the EPS structure

Defends from recolonization

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Thank you!!!

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18. Hall-Stoodley L, Stoodley P. Evolving concepts in biofilm infections. *Cell Microbiol*. 2009 Jul;11(7):1034-43.
19. Miller KG, Tran PL, Haley CL, Kruzek C, Colmer-Hamood JA, Myntti M, Hamooda HM. Next Science Wound Gel Technology, a Novel Agent That Inhibits Biofilm Development by Gram-Positive and Gram-Negative Wound Pathogens. *Antimicrob Agents Chemother*. (2014) Jun;58(6):3060-72.
20. Wolcott R. Disrupting the biofilm matrix improves wound healing outcomes. *J Wound Care.* 2015; 24: 366-371.
21. Niezgoda JA, Stevenson P. Shifting the Spotlight to the Biofilm Structure: Why Only Targeting the Bacteria in Wounds is Not Enough. (2018) *Ostomy Wound Management*. 2018;64(9):8,10.

Local Infection to SIRS: When Does the Local Become Systemic? Bioload Management

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Objectives

- Recognize the progression of biofilm development and damage
- Identify symptoms of Systemic Inflammatory Response Syndrome (SIRS)
- Verbalize treatments for preventing and treating chronic wound biofilms

Disclosures

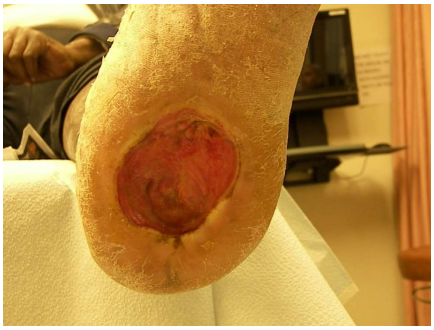
- Speaker: Essity, Smith & Nephew, 3M, Pure & Clean, Osirus
- Research Support:
- Osirus
- Pending Research support:
- RedDress, Reapplix

Signs and Symptoms

- Classic Signs: Redness, Warmth, Swelling, Pain or Tenderness
- Secondary Signs: increased drainage, friable or discolored granulation tissue, undermining of wound edges, odor

Secondary Signs of Biofilm on the Wound

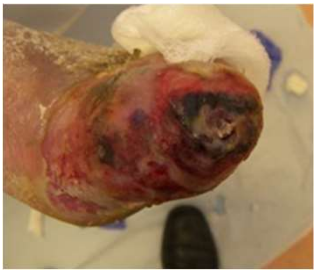
- Increased Serous exudate
- Discoloration of wound bed
- Friable granulation tissue
- Pocketing of the wound
- Increasing odor
- Delayed Healing



Gardner et al WRR Vol 9, 2001

Definitions

- **Wound Infection:** the presence of replicating microorganisms within a multispecies biofilm in the wound that cause host injury/response, initially local then, with seeding of fragments, systemic. Multispecies biofilms with seeding
- Primarily pathogens of concern:
  - *Staphylococcus aureus,* Beta-hemolytic *Streptococcus (S. pyogenes, S. agalactiae), E. coli, Proteus, Klebsiella,* anaerobes, *Pseudomonas, Acinetobacter, Stenotrophomonas (Xanthomonas).*



Progression of Bacterial Damage

- Low Risk
- Contaminated or Colonized
- Bacteria present on the wound surface
- Replicating organisms attaching to the wound surface but not associated with tissue damage or delayed healing (colonization)

Progression of Bacterial Damage

- **Medium Risk**
- Local infection, increased bioburden
- Initiates the body's immune response (inflammation)
- Wound healing delayed, closure stalled
- **NERDS** Non-healing, Exudative wound, Red and bleeding, Debris in wound, Smell

Increased Bacterial Burden and Infection: The Story of NERDS and STONES
Sibbald RG, Woo K, Ayello E. Advances in Skin & Wound Care: October 2006 - Volume 19 - Issue 8 - p 462-463

Increased Bacterial Burden and Infection: The Story of NERDS and STONES
Sibbald RG, Woo K, Ayello E. Advances in Skin & Wound Care: October 2006 - Volume 19 - Issue 8 - p 462-463

STONEEES: Signs of Severe Wound Infection

- SIZE: Increased wound Size
- TEMPERATURE: Increased local wound Temperature
- OS: Extension of the wound to bone
- NEW wound breakdown
- Exudate/ Edema/ Erythema
- SMELL: increasing odor

Systemic Inflammatory Response Syndrome (SIRS)

- Two or more symptoms including fever or hypothermia, tachycardia, tachypnoea and change in blood leucocyte count.

  - Temperature >38° C or <36° C
  - Heart rate >90 beats/min
  - Respiratory rate >20 breaths/min or $PaCO_2$<32 torr
  - WBC >12,000 cell/mm^3, <4,000 cells/mm^3, or >10% immature (band) forms

Systemic Inflammatory Response Syndrome (SIRS)

- The use of SIRS is a valid method of classifying infection severity in hospitalized patients with DFI.
- Recently, the Infectious Disease Society of America (IDSA) updated their guidelines (204) and recommended using systemic inflammatory response syndrome (SIRS) as a method for distinguishing between moderate and severe DFI.
- Hospitalized patients with DFI who presented with SIRS had higher rates of major amputation, had longer hospital stays, required more surgery, required more subsequent admissions, and grew more organisms on wound culture than patients who did not manifest SIRS.

Wukich DK, Hobizal KB, Raspovic KM, Rosario BL. SIRS is valid in discriminating between severe and moderate diabetic foot infections. Diabetes Care. 2013;36(11):3706–3711. doi:10.2337/dc13-1083

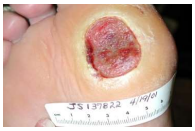
Classification of Diabetic Foot Infections Mild/Severe

Feature	Mild infection	Severe infection
Presentation	Slowly progressive	Acute or rapidly progressive
Ulceration	Involves only skin	Penetrates to subcutaneous tissues
Tissues involved	Epidermis, dermis	Fascia, muscle, joint, bone
Cellulitis	Minimal (<2 cm around ulcer rim)	Extensive, or distant from ulceration
Local signs	Limited inflammation	Severe inflammation, crepitus, bullae, necrosis or gangrene
Systemic signs	None or minimal	Fever, chills, hypotension, confusion, volume depletion, leukocytosis
Metabolic control	Mildly abnormal (hyperglycemia)	Severe hyperglycemia, acidosis, azotemia, electrolyte abnormalities
Foot vasculature	Minimally impaired (normal/reduced pulses)	Absent pulses, reduced ankle or toe blood pressure
Complicating features	None or minimal (callus, ulcer)	Eschar, foreign body, puncture wound, abscess, marked edema, implanted metalwork or other prostheses

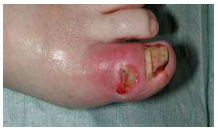
Benjamin A. Lipsky. Medical Treatment of Diabetic Foot Infections.
Clin Infect Dis. (2004) 39 (Supplement 2): S104-S114. doi: 10.1086/383271

Clinical Classification of Diabetic Foot Infection PEDIS Grades (Perfusion, Extent, Depth, Infection, Sensation)

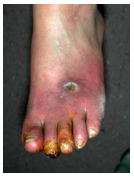
1 (None) Wound without purulence or other evidence of inflammation



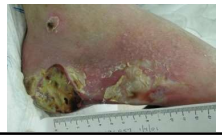
2 (Mild) ≥More than 2 of purulence, erythema, pain, tenderness, warmth or induration. Any cellulitis/erythema extends ≤2 cm around ulcer and infection is limited to skin/superficial subcut tissues. No local complications or systemic illness



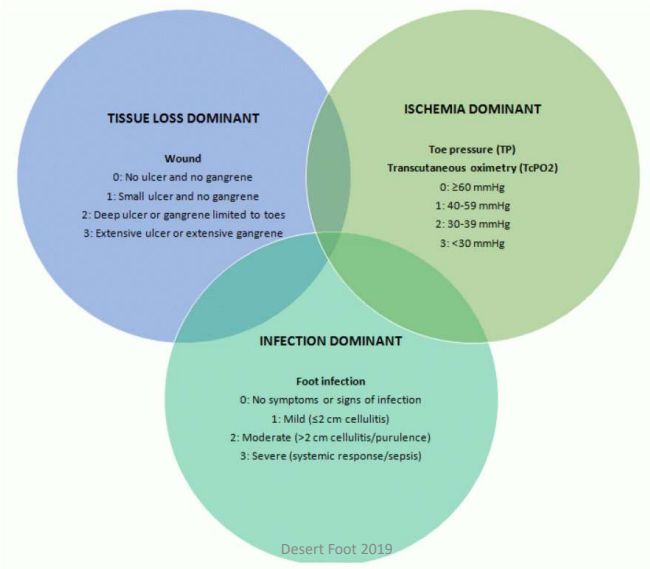
3 (Moderate) Infection in patient who is systemically well & metabolically stable but has any of: cellulitis extending >2 cm; lymphangitis; spread beneath fascia; deep tissue abscess; gangrene; muscle, tendon, joint or bone involved



4 (Severe) Infection in a patient with systemic toxicity or metabolic instability



Hicks CW, et al. The Society for Vascular Surgery Wound, Ischemia, and foot Infection (WIfI) classification independently predicts wound healing in diabetic foot ulcers. J Vasc Surg. 2018 Apr 2



Tissue Loss Dominant - Wound

- 0 - No Ulcer No Gangrene
- 1 - Shallow Ulcer, No Gangrene
- 2 – Deeper ulcer w/ exposed bone or shallow heel ulcer w/ no calcaneal involvement, Gangrene limited to digits
- 3 – Extensive deep ulcer or heel ulcer involving the calcaneus, Extensive gangrene forefoot =/or midfoot

Ischemia Dominant

- 0 – ABI ≥ 0.8, Ankle pressure > 100 mmHg, Toe pressure TP or TCPO2 ≥ 60 mmHg
- 1 - ABI ≥ 0.6-0.79, Ankle 70-100 mmHg. TP or TCPO2 40-59 mmHg
- 2 – ABI ≥ 0.4-0.59, Ankle 50-70, TP or TCPO2 30-39 mmHg
- 3 – ABI < 0.39, Ankle <50 mmHg. TP or TCPO2 <30 mmHg

Infection Dominant – IDSA/PEDIS

- 0 – No symptoms or signs of infection, uninfected
- 1 – Local Infection skin and subQ, mild
- 2 – Local infection, >2 cm erythema, involved deeper structures, no systemic response, moderate
- 3 – Local infection with signs of SIRS (2 or more of: ↑ Temp, HR, Resp, WBC), severe

Estimate Amputation Risk

a, Estimate risk of amputation at 1 year for each combination

	Ischemia – 0				Ischemia – 1				Ischemia – 2				Ischemia – 3			
W-0	VL	VL	L	M	VL	L	M	H	L	L	M	H	L	M	M	H
W-1	VL	VL	L	M	VL	L	M	H	L	M	H	H	M	M	H	H
W-2	L	L	M	H	M	M	H	H	M	H	H	H	H	H	H	H
W-3	M	M	H	H	H	H	H	H	H	H	H	H	H	H	H	H
	fI-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3

b, Estimate likelihood of benefit of/requirement for revascularization (assuming infection can be controlled first)

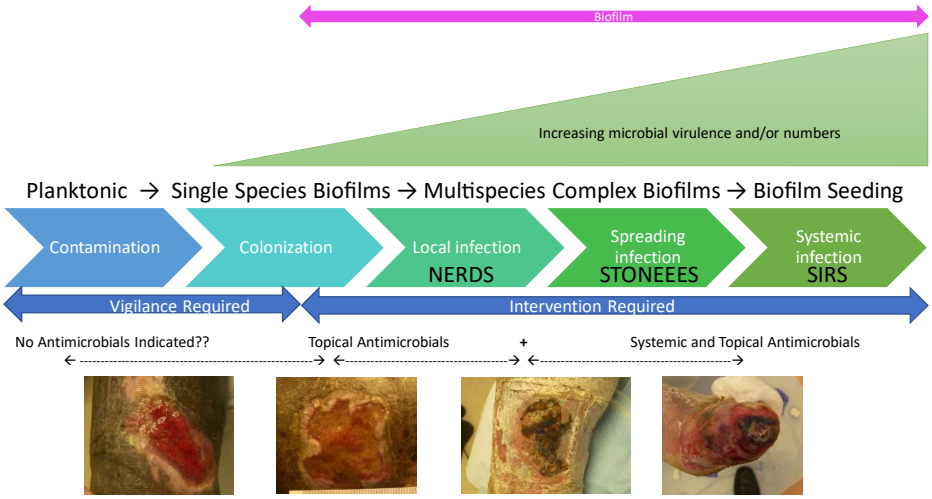
	Ischemia – 0				Ischemia – 1				Ischemia – 2				Ischemia – 3			
W-0	VL	VL	VL	VL	VL	L	L	M	L	L	M	M	M	H	H	H
W-1	VL	VL	VL	VL	L	M	M	M	M	H	H	H	H	H	H	H
W-2	VL	VL	VL	VL	M	M	H	H	H	H	H	H	H	H	H	H
W-3	VL	VL	VL	VL	M	M	M	H	H	H	H	H	H	H	H	H
	f-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3

fI, foot Infection; I, Ischemia; W, Wound.

ABCESS Approach to Chronic Wound Assessment and Management

Assess_ Arterial Venous and Lymphatic system function	**B**ioload and Wound Bed Management	**C**ellular Activity Assessment	**E**xudate level assessment	**S**ystemic disease diagnosis and management	**S**kin protection and treatment
Normalize to the extent possible with available interventions	Prevent high bacterial counts, remove and prevent biofilm formation and prolonged inflammation	Alter therapies based on cell function observed in the wound bed, identify malignancies	Maintain moisture balance in the wound bed and prevent periwound maceration	Control DM, ESRD, Autoimmune Dx, CHF, CKD, etc. to maintain local and systemic homeostasis	Prevent traumatic, inflammatory or iatrogenic damage
Vascular surgery , Compression wraps and Segmental Compression, CDT/MLD	Debridement, antimicrobials, antiseptics, biofilm disrupters, dressing technologies	Wound fluid analysis, PCR/DNA, Biopsy, CTPs, Growth Factors, Autografts	Topical applications, Dressing selection or alteration, NPWT	BS, BP, Nutrition, Renal Function	Periwound protection, offloading, reduce edge effect, decrease inflammation
Maximize vascularity and healing potential	Lower bacterial counts, remove necrotic debris, convert chronic to acute wound bed, and reduce inflammation	Maximize cell function and identify active cell types and adjust therapies to respond to cell activity	Assure a healthy environment for growing cells	Systemic interventions to maximize local healing capacity	Prevent unintended damage to the wound and nearby tissues

The New IWII Wound Infection Continuum - Modified



International Wound Infection Institute (IWII) *Wound infection in clinical practice.* Wounds International 2016

WHAT LEADS TO CHRONICITY?

- **Recent studies have shown that the bacterial bioburden significantly affects healing rates of wounds.[1]**
- **Wound infection is the most common cause for poorly healing wounds and complicates the healing process.[1]**

1. Granick MS, Tenehause M, Knox KR, Ulm JP. Comparison of wound irrigation and tangential hydrodissection in bacterial clearance of contaminated wounds: Results of a randomized clinical study. *Ostomy Wound Management.* 2007; 53(4): 64-72.

Biofilms Have Been Identified in >80% of Biopsies of Chronic Wounds but in Only 6% of Acute Wounds*

- Biofilm development is one of the leading causes of relapsing/decreased healing in our high-risk patients. *(Wolcott RD, J WdCare 2008)*
- HypochlorousAcid is an extremely safe/inexpensive means of destroying the matrix of biofilm without harming the essential wound healing cells. *(SakaryaS, et al. Wounds, Dec 2014)*

* M. Malone, T. Barjnsholt, A. McBain, G. James, P. Stoodley, D. Leaper, M. Tachi, G. Schultz, T. Swanson, R. Wolcott. Prevalence of biofilms in chronic wounds: a systematic review and meta-analysis of published data, J wound Care, 2017

Biofilm Seeding

- Bone biofilms are the result of surface resistant bacteria fracturing and seeding deeper structures
- Control the surface and you prevent the extension of infection to deeper structures

Waldvogel FA, Medoff G, Swartz MN. Osteomyelitis: a review of clinical features, therapeutic considerations and unusual aspects (first of three parts). N Engl J Med 1970;282:198–206.) (Lew DP, Waldvogel FA. Osteomyelitis. Lancet. 2004 Jul 24-30;364(9431):369-79

- Despite recent advances in antibiotic therapies, the incidence of chronic osteomyelitis has steadily risen because of our inability to address the biofilm component of the infection leading to radical debridement as the preferred method of treatment as compared to medical management of the disease.

Zhongguo Xiu Fu Chong Jian Wai Ke Za Zhi. Bacterial biofilms and chronic osteomyelitis. 2010 Jan;24(1):108-11.

- RESULTS:
  - Biofilms form on the surface of necrotic soft tissue and, if exposed, dead bone……. Due to the protective mechanisms of the bacterium they were far more resistant to antimicrobial agents, which led to the development of chronic osteomyelitis…….
- Designing *treatment programs to address biofilms is the most effective way to prevent osteomyelitis*
- CONCLUSION:
  - The most effective way to prevent biofilms is still debridement.

The Role of Regular Wound Cleansing

- Studies have shown a **direct correlation** between ongoing wound cleansing and debridement and increased healing rates.[6]
- **Frequent cleansing and debridement improves clinical outcomes and may reduce amputation rates.[6]**

Falanga V, Brem H, Ennis WJ, Wolcott R. Maintenance debridement in the treatment of difficult-to-heal chronic wounds. *Ostomy Wound Management.* 2008 Supplement: 1-15.

De A, Raj HJ, Maiti PK. Biofilm in Osteomyelitis caused by a Rare Pathogen, Morganella morganii : A Case Report. J Clin Diagn Res. 2016;10(6):DD06–DD8. doi:10.7860/JCDR/2016/18666.7990

- Morganella morganii is a member of Enterobacteriaceae family …..rarely causes infection alone and is generally encountered in immunosuppressed patients.
- Isolation of uncommon pathogens from chronic wounds should prompt us to suspect biofilm producing capability of the isolated pathogens
- The clinical goal should be to cure the case radically instead of performing occasional investigations and altering antimicrobial therapy in response to those changes
- **The main principles of osteomyelitis treatment are radical debridement of necrotic tissues, eliminating dead spaces, and targeting long-term antibiotic therapy to isolated pathogens in the biofilm.**
- Antimicrobial treatment should not be empirical rather directed towards isolated pathogens

WOUND CLEANSING IN WOUND BED PREPARATION

- **Wound cleansing, along with thorough debridement, is essential for bacterial clearance and a prerequisite for proper wound healing.[1,2]**
- **THE USE OF WOUND IRRIGATION SOLUTIONS**[3]
  - Rapidly decontaminates the wound
  - Removes necrotic tissue
  - Enhances the effectiveness of other advanced healing modalities, reducing the interference of competing biofilm

1. Schultz GS, Sibald RG, Flanaga V, Ayello EA. Wound bed preparation: a systematic approach to wound management. *Wound Rep Reg.* 2003; 11: 1–28.
2. Granick MS, Tenehause M, Knox KR, Ulm JP. Comparison of wound irrigation and tangential hydrodissection in bacterial clearance of contaminated wounds: Results of a randomized clinical study. *Ostomy Wound Management.* 2007; 53(4): 64-72
3. 18. Klein S, Schreml S, Dolderer J, Gehmert S. Evidence-based topical management of chronic wounds according to the T.I.M.E. principle. *Journal of the German Society of Dermatology.* 2013; 819-829

Ideal Topical Agent

- STAR
- not used Systemically
- low Tissue Toxicity
- non – Allergic
- low incidence of bacterial Resistance

Topical Antiseptics

• Chlorhexidine	Broad spectrum/low toxicity
• Povodine-iodine	Broad spectrum/low toxicity
• Acetic acid	Pseudomonas
• Dyes: Scarlet red	Gram + bacteria alone
	Proflavine
• Na Hypochlorite	Toxic to granulation tissue
	Dakin's, Eusol
• Hypochlorous Acid	Non-toxic to tissue
• Hydrogen peroxide	Effective only when effervescent
• Quaternary ammonia:	Cetrimide - Very toxic

Adjunctive Topical Antibiofilm Agents are the Key to Successful Wound Debridement

- Disrupt and Prevent
- Systemic antibiotics are only 25-32% effective against biofilms
- Ischemic wounds are less susceptible to systemic antibiotics
- Topical antibiotics, antibiofilm, antimicrobial applications must be coupled with frequent debridements to allow the agents to come in contact with exposed planktonic bacteria

Rhoads D, Wolcott R, Percival S. Biofilms in Wounds: Management Strategies. Journal of Wound Care. Vol 17. no. 11, Nov 2008

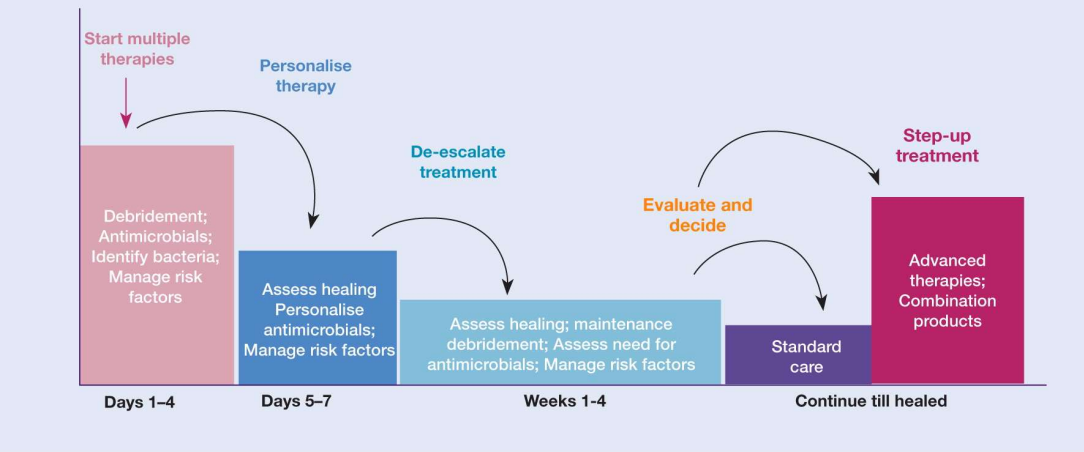
BIOFILM-BASED WOUND CARE™

- The proliferation of uncontrolled biofilm inhibits wound healing.1
- "When a wound is strongly suspected of containing a biofilm, there is no one-step solution for treatment. A proactive approach using a combination strategy based on elements of wound bed preparation may be helpful and aims to: reduce the biofilm burden, and prevent reconstitution of the biofilm.

  This approach is sometimes called 'biofilm-based wound care'.2

1. Bowler PG, Duerden DI, Armstrong DG. Wound microbiology and associated approaches to wound management. *Clinical Microbiology Reviews*. 2001; 14(2): 244–269.
2. Phillips PL, Wolcott RD, Fletcher J, Schultz GS. Biofilms Made Easy. *Wounds International* 2010; 1(3): Available from http://www.woundsinternational.com

Proposed step-down and then step-up biofilm pathway. Adapted from Schultz et al.



Schultz G, Bjarnsholt T, James GA et al.. Consensus guidelines for the identification and treatment of biofilms in chronic nonhealing wounds. **Wound Repair Regen** 2017; 25(5):744–757. https://doi.org/10.1111/wrr.12590

Osteomyelitis

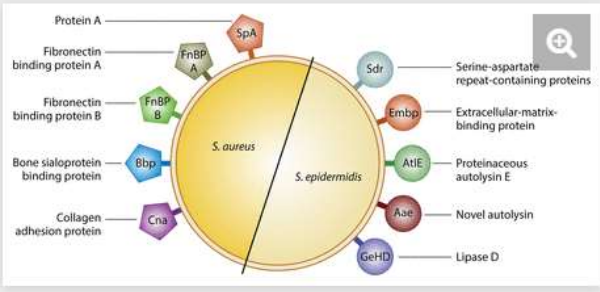
- Chronic osteomyelitis, has been defined as a "bone-biofilm"[1]
- The resistance of osteomyelitis to antibiotic therapy is largely attributed to the ability of its causative organisms to form a biofilm on bone surfaces when the infection becomes chronic in nature[2]

1. Brady, R. A., Leid, J. G., Calhoun, J. H., Costerton, J. W., & Shirtliff, M. E. (2008). Osteomyelitis and the role of biofilms in chronic infection. FEMS Immunology & Medical Microbiology FEMS Immunol Med Microbiol, 52(1), 13-22. doi:10.1111/j.1574-695x.2007.00357.x
2. Waldvogel FA, Medoff G, Swartz MN. Osteomyelitis: a review of clinical features, therapeutic considerations and unusual aspects (first of three parts). N Engl J Med 1970;282:198–206.) (Lew DP, Waldvogel FA. Osteomyelitis. Lancet. 2004 Jul 24-30;364(9431):369-79.

Brady RA, Leid JG, Calhoun JH, Costerton JW, Shirtliff ME. Osteomyelitis and the role of biofilms in chronic infection. FEMS Immunology & Medical Microbiology, January 2008. DOI 10.1111/j.1574-695x.2007.00357.x

- Contiguous focus osteomyelitis is commonly seen in patients with diabetes mellitus, and often involves multi-species infections in the feet
- **The most important factor in the development of chronic osteomyelitis, is its ability to form a biofilm.**
- Because (Staphylococcus aureus) relies on biofilm formation for persistent infection, therapeutics targeting biofilm could prove to be promising in the treatment of osteomyelitis.

Multiple Adhesive Molecules
biofilm formation facilitates persistence of the infection



Staphylococcus aureus and *Staphylococcus epidermidis* cell surface proteins, known as microbial surface components recognizing adhesive matrix molecules (MSCRAMMs), that are involved in interacting with bone and the bone ECM.

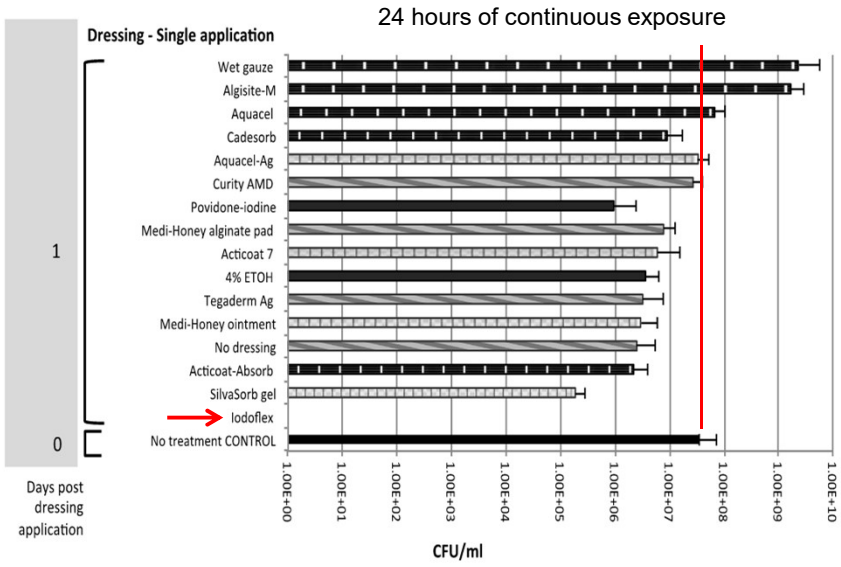
Kavanagh N, Ryan EJ, Widaa A, et al. Staphylococcal Osteomyelitis: Disease Progression, Treatment Challenges, and Future Directions. Clinical Microbiology Reviews. April 2018 Volume 31 Issue 2 e00084-17

Antiseptics and Antimicrobials

- Many are able to penetrate biofilms but often harmful to growing cells
- Effective within the dressing but not on the wound surface
  - Silver – the concentration of silver present in most dressings is too low to effect chronic wound biofilms. Silver resistance has begun to develop in response to heavy usage of silver. High concentrations inhibit fibroblast activity in vitro. Intimate contact low dose silver may be an answer
  - Iodine – slow release cadexomer iodine, iodine foams have less host damage
  - Honey – antimicrobial with osmotic effects on bacteria and enhanced autolytic debridement
  - Methylene blue and gentian violet and PHMB – prevents biofilm within the dressing
  - Hypochlorous acid (HOCl) – kills most wound pathogens within 12 seconds, dose dependent favorable effects on fibroblast and keratinocyte migration, effective against bacteria within the biofilm

Rhoads D, Wolcott R, Percival S. Biofilms in Wounds: Management Strategies. Journal of Wound Care. Vol 17. no. 11, Nov 2008
Sakarya S. et al. Hypochlorous acid: An ideal wound care agent with powerful microbiocidal, antibiofilm, and wound heling potency. Wounds vol.26, no. 12 Dec 2014 276

Effects of Antimicrobial Agents on Mature Biofilms on Pig Skin Explants



P.L. Phillips, Q. Yang, E. Sampson, G. Schultz. Effects of Antimicrobial Agents on an In Vitro Biofilm Model of Skin Wounds, **Advances Wound Care**, 1: 299-304, 2010.

Kramer A, et al. Consensus on Wound Antisepsis: Update 2018. Skin Pharmacol Physiol 2018;31:28–58, DOI: 10.1159/000481545

Table 13. Orientating recommendation for the indication-based selection of wound antiseptics

Indication	Antiseptic compound	
	1st choice	2nd choice
Critically colonized wounds, wounds at risk of infection	PHMB	OCT, hypochlorite, silver
Burns	PHMB	OCT, hypochlorite
Bite, stab, and gunshot wounds	PVP-I	Hypochlorite
MDRO-colonized or infected wounds	OCT/PE	OCT, PHMB, silver
Prevention of SSI	PHMB	OCT/PE
Decontamination of acute and chronic wounds	Hypochlorite, PHMB	–
Peritoneal lavage	Hypochlorite	–
Risk of CNS tissue exposure	Hypochlorite	PVP-I
Wounds with lack of drainage	Hypochlorite	PHMB

PHMB Polyhexamethylene biguanide
OCT Octenesin OCT/PE Polyethylene with Octenesin
Hypochlorite Dakins
PVP-I Povidone iodine

EXPERT RECOMMENDATIONS FOR THE USE OF HYPOCHLOROUS SOLUTION: SCIENCE AND CLINICAL APPLICATION. Armstrong et al.

- Based on *in vitro* studies, the antimicrobial activity of HOCl appears to be comparable to other antiseptics but without cytotoxicity
- Strong evidence was found for use in diabetic foot wounds
- The panel recommended HOCl should be used in addition to tissue management, infection, moisture imbalance, edge of the wound and aggressive debridement.

Armstrong DG, Bohn G, Glat P, Kavros SJ, Kirsner R, Snyder R, Tettelbach W.
Expert Recommendations for the Use of Hypochlorous Solution: Science and Clinical Application. Ostomy Wound Manage. 2015 May;61(5):S2-S19.

Wound Bed: Benefits of Topical HOCl

- Reduction of microbial load/disruption of bacterial biofilms
- Inhibition of the inflammatory response from mast cells
- Increase of the periwound Transcutaneous Pressure of Oxygen (TcPO2)
- Promotion of wound healing
- Reduction of odor
- Promotes Demarcation Without Infection

Armstrong DG, Glatt P, et.al. Expert Recommendations for the Use of Hypochlorous Solution: Science and Clinical Application. Ostomy Wound Manage. 2015 May;61(5):S2-S19.

Ragab II, et.al. The Effectiveness of Hypochlorous Acid Solution on Healing of Infected Diabetic Foot Ulcers. Journal of Education and Practice, Vol.8, No.8, 2017

- N=60 DFUs
- Hypochlorous acid controls the tissue bacterial bio-burden without inhibiting the wound healing process, rapidly relieves of pain, short hospital stay and the ulcer well prepared to natural healing or skin flap or graft.
- Hypochlorous acid is an ideal wound care solution in cleansing infected diabetic foot ulcers.
- HOCL as a cleanser agent appears to be effective on infected diabetic foot ulcers.

Bongiovanni CM, Effects of Hypochlorous Acid Solutions on Venous Leg Ulcers (VLU): Experience With 1249 VLUs in 897 Patients. Journal of the American College of Clinical Wound Specialists. Volume 6, Issue 3, December 2014, Pages 32-37

- Following 15–30 s of exposure to HCA, all patient categories exhibited increased tcpO2 except for those with significant PAD and/or smoking
- At 72 h, those patients who exhibited increased tcpO2 in response to initial wound bed exposure to HCA also demonstrated persistent elevation of tcpO2 above baseline.
- Aqueous solutions of hypochlorous acid, even in trace amounts, will kill most pathogens within 30 seconds of exposure.
- HCA also reduces mast cell degranulation and encourages active capillary dilation
- We conclude that venous leg ulcer care protocols that clean, debride, pack and dress with hypochlorous acid solutions can reduce the effects of some comorbidities while accelerating healing times.