Mastering the Foundation of Wound Healing Using ABCESS©
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Goals

- Review the TIME/DIME/TIMERS acronym and understand why a new model is needed.

- Understand how the ABCESS© acronym was developed and know how each element contributes to complete wound care assessment.

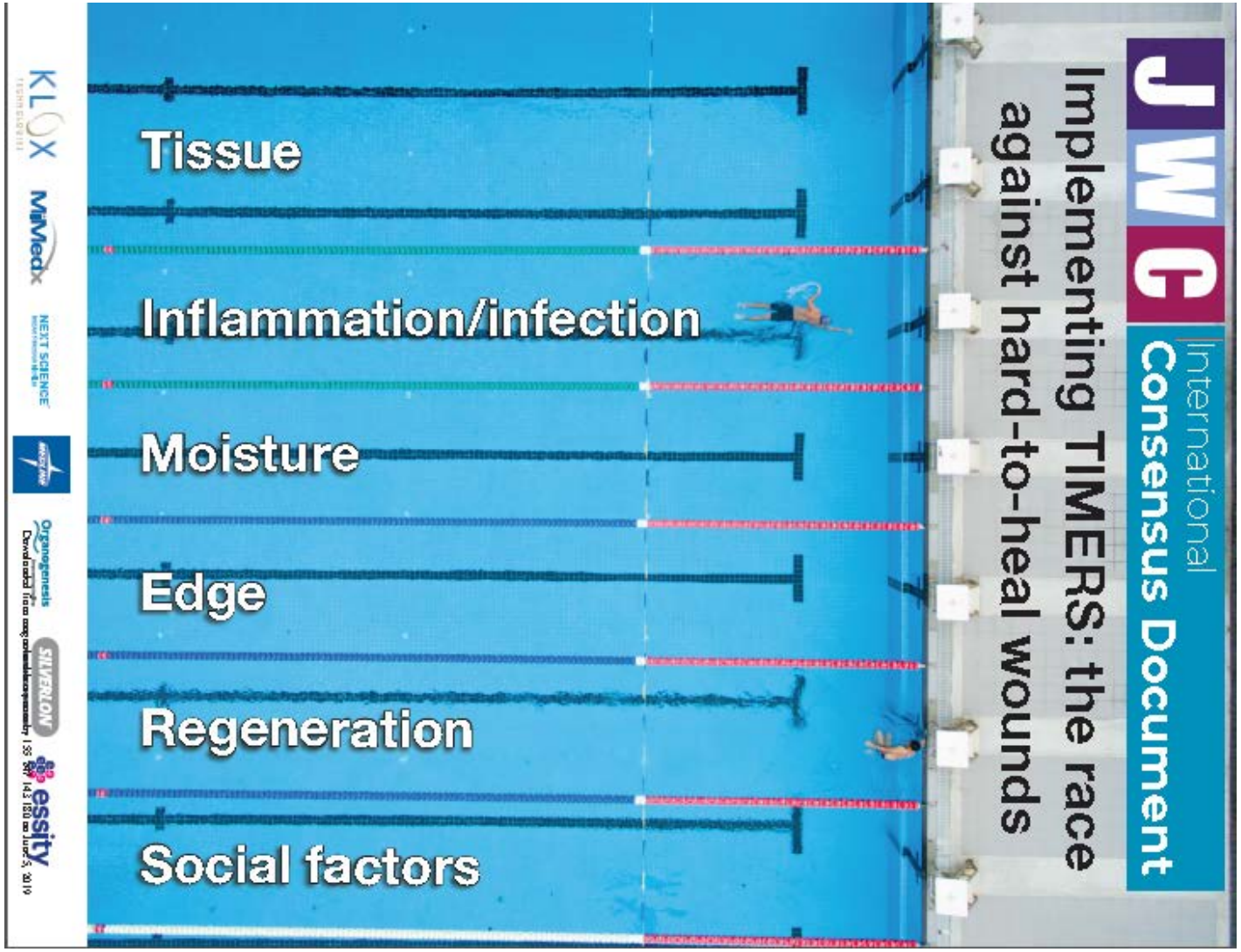
T/DIME Principles of Wound Bed Preparation

Tissue non viable or deficient	Infection or inflammation	Moisture imbalance	Edge of wound non advancing or undermined
Defective matrix and cell debris	High bacterial counts or prolonged inflammation	Desiccation or excess fluid	Non-migrating keratinocytes Non-responsive wound cells
↓	↓	↓	↓
Debridement	Antimicrobials	Dressings compression	Biological agents Adjunct Therapies Debridement
↓	↓	↓	↓
Restore wound base and ECM proteins	Low bacterial counts and controlled inflammation	Restore cell migration, maceration avoided	Stimulate keratinocyte migration

R. Gary Sibbald, MD; Heather Orsted, RN, ET; Gregory S. Schultz, PhD; Patricia Coutts, RN; and David Keast, MD, for the International Wound Bed Preparation Advisory Board and the Canadian Chronic Wound Advisory Board. PREPARING THE WOUND BED 2003: FOCUS ON INFECTION AND INFLAMMATION. Ostomy/Wound Management 2003;49(11):24–51

Now Its TIMERS

Leanne Atkin, Zofia Bućko, Elena Conde Montero, Keith Cutting, Christine Moffatt, Astrid Probst, Marco Romanelli, Gregory S Schultz, William Tettelbach. Implementing TIMERS: the race against hard-to-heal wounds. Published Online:5 Mar. Journal of Wound Care, Vol. 23, No. Sup3a



ABCESS: A New Acronym for Improved Wound Management

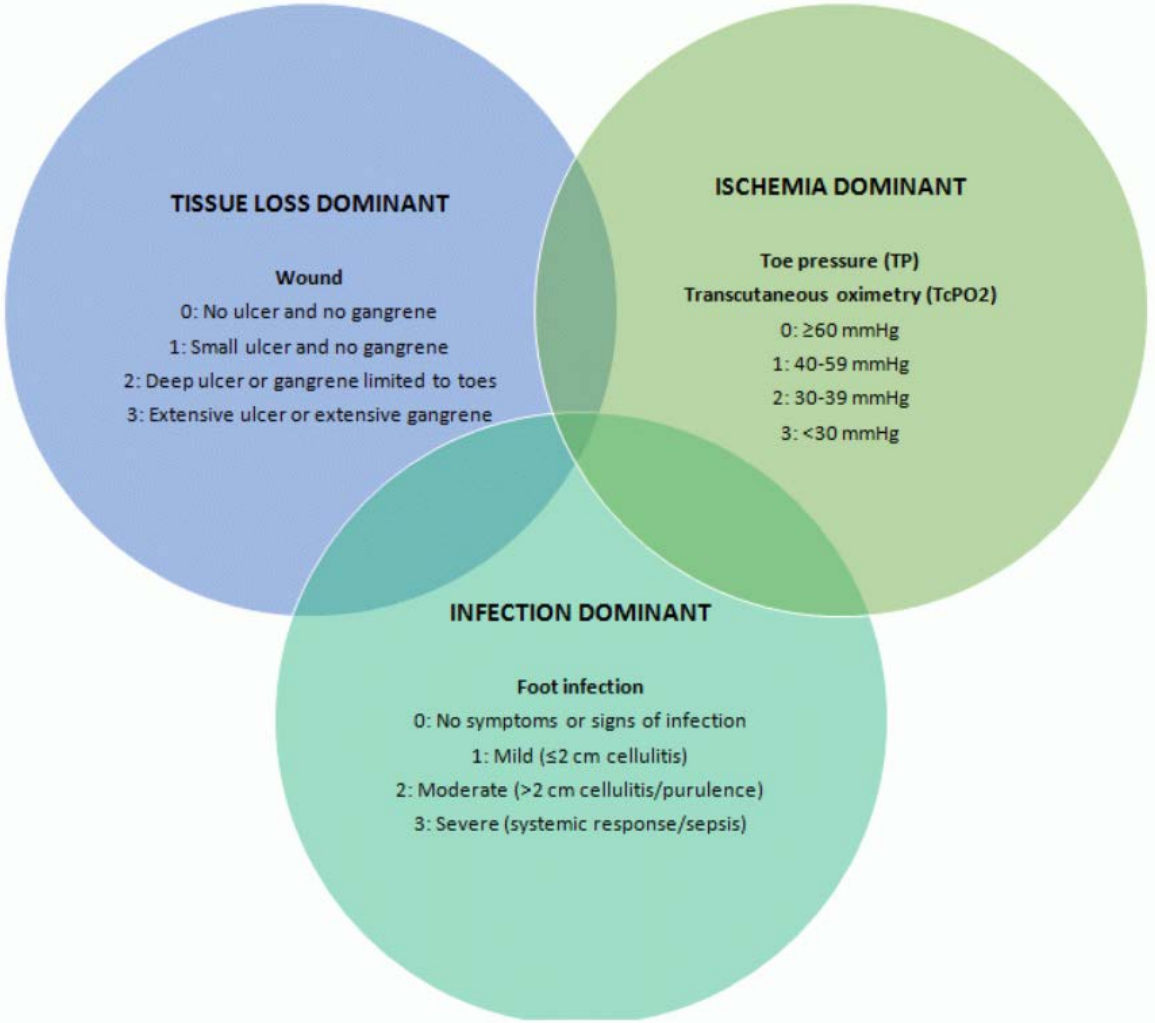
Assess_ Arterial Venous and Lymphatic system function	**B**ioload and Wound **B**ed Management	**C**ellular Activity Assessment	**E**xudate level assessment	**S**ystemic disease diagnosis and management	**S**kin protection and treatment
Normalize to the extent possible with available interventions	Prevent 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

ABCESS

Assess Arterial, Venous, and Lymphatic Circulation

Hicks CW, et al. The Society for Vascular Surgery

Wound, Ischemia, and foot Infection (WIfI) classification independently predicts wound hea or 2



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

- 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 – 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.

Relative Risk Factors for Developing Critical Limb Ischemia

- Diabetics are 4x more likely to develop CLI than non-diabetics
- Smoking 3x
- Pts over 65 2x
- Pts with elevated lipids 2x
- Pts with ABI <0.7 x2
- ABI <0.5 x2.5

Norgren, L. et al. Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC II). Journal of Vascular Surgery , Volume 45 , Issue 1 , 2007, S5 - S67

Arterial Ulcer Assessment

Ankle/Brachial Index

0.91 – 1.3	Normal
0.7 – 0.9	Mild Ischemia
0.4 – 0.69	Moderate Ischemia
< 0.4	Severe Ischemia
> 1.3	(Arterial Calcinosis)
➢ 0.8 – 1.0	Normal Toe – Brachial Index

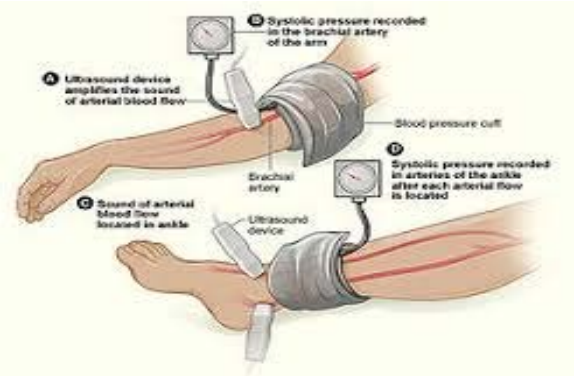
Based on ADA Guidelines

WiFI

- 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

Non-invasive Diagnostic Tests

- Ankle/Brachial Index
- Pulse Volume Recordings (PVR)
- Toe pressures
- SensiLase™ Skin Perfusion Pressure
- Ultrasonic Doppler Waveforms
- Transcutaneous Oxymetry (TCOM)
- Near Infrared Photography



Vascular Findings Indicative of Poor Healing

- Ankle Systolic Pressure <60 mmHg or <90 with DM
- ABI <0.5mmHg
- Toe Systolic Pressure <35 mmHg or <50 mmHg w/ DM
- TBI <0.4 mmHg
- TCPO2 < 20 mmHg
- Skin Perfusion Pressure (Laser Doppler) <30 mmHg CLI

Lo T, et al. Prediction of Wound Healing Outcome Using Skin Perfusion Pressure & Transcutaneous Oximetry. WOUNDS. 2009;21(11):310-316.

Ulcers Venous

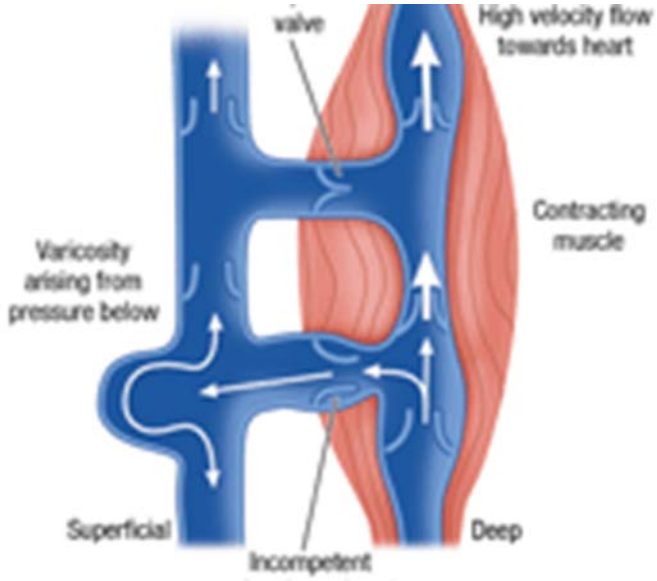
Prevalence of Chronic Venous Insufficiency

1. Incidence of CVI from < 1% to 40% in females and from < 1% to 17% in males.
2. Prevalence estimates for varicose veins are higher, <1% to 73% in females and 2% to 56% in males.
3. Among the 226 screened patients, 138 (61.1%) were diagnosed as having CVI (69% female and 45% male, $p<0.001$).

1,2 Beebe-Dimmer JL, et. al. The epidemiology of chronic venous insufficiency and varicose veins. Ann Epidemiol. 2005 Mar;15(3):175-84
3. Al Shammeri O, et.al. Chronic Venous Insufficiency: prevalence and effect of compression stockings. International Journal of Health Sciences. 2014;8(3):231-236.

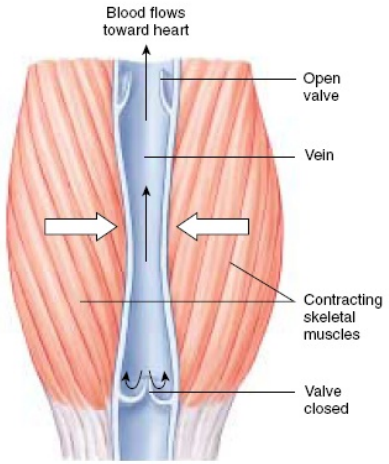
Venous System- Components

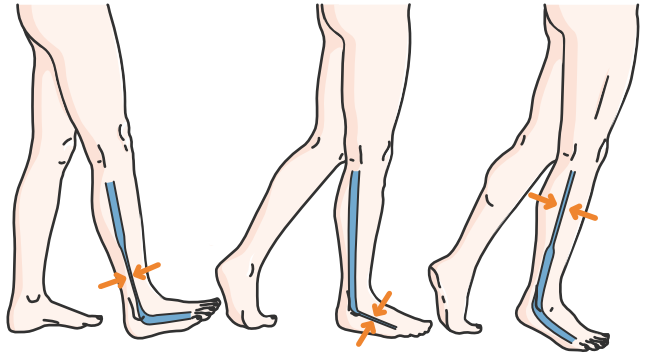
- **Deep system** – encased in muscle, evacuates majority of blood from the extremity.
- **Superficial system** – veins in the subcutaneous to dermal layers – oriented longitudinally
- **Saphenous veins-** between muscle and subcutaneous layers
  - Greater
  - Lesser
- **Perforators**- small veins, that run perpendicular to the deep, saphenous and superficial veins.
- **77% of all venous disease involves perforator veins.**
- Unidirectional flow from superficial to deep veins
- 30 named perforators, hundreds per leg,
- 3 to 5 valves per vein,
- Small in diameter.
- Transport blood to deep veins for evacuation
- Help to equalize pressure through out the system



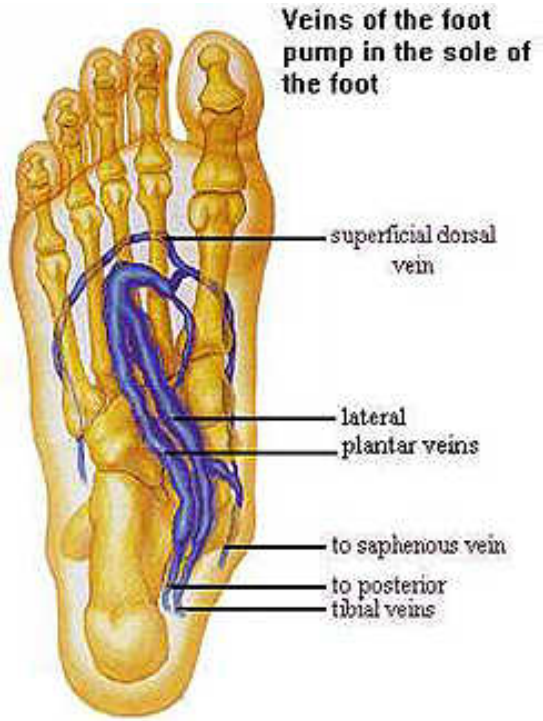
Venous Pumps – Calf and Foot
Ambulation is Essential for Effective Venous Circulation

Calf Pump – The Heart of the Lower Extremity Veins, Pumps the Deep Veins



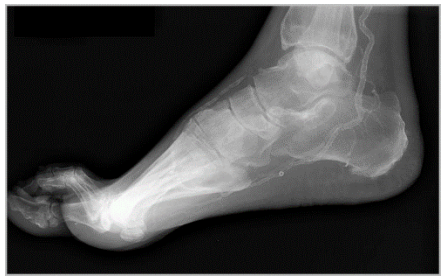


Foot Pump – Empties the foot and ankle area, loads the Saphenous veins



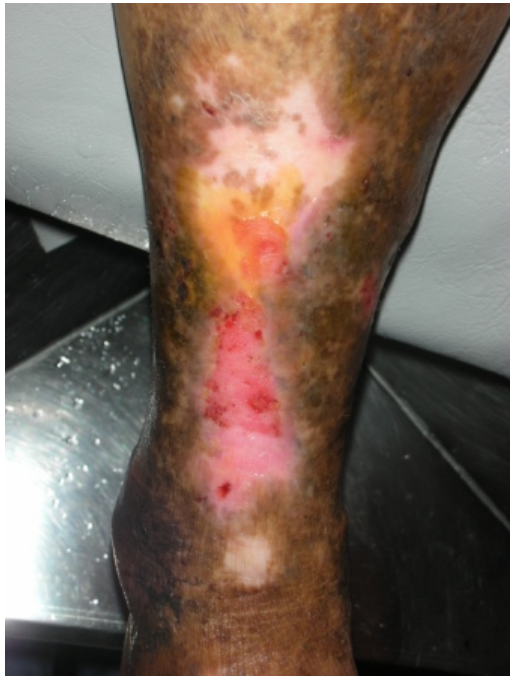
ABI Clinical Significance

- > 1.2
- 0.9 - 1.1
- 0.7 - 0.8
- 0.4 - 0.6
- < 0.4



- **Suspect vessel wall sclerosis or calcification**
- **Normal**
- **Mild occlusive disease. May have intermittent claudication**
- **Moderate to severe occlusive disease - Wound healing impaired**
- **Severe occlusive disease, ischemia**

The problem with lower extremity circulatory disorders



Untreated, the symptoms get progressively worse
Patients are often shuffled around
Reoccurrence is common*

*67% of venous ulcers reoccur within 48 months with elastic compression alone.

2002 J Vascular Surg. Apr;35(4):723-8

Venous disease- CEAP classification

Clinical classification of the progression of venous disease

0: No visible or palpable signs of venous disease

1: Reticular veins (spider veins)

2: Varicose veins

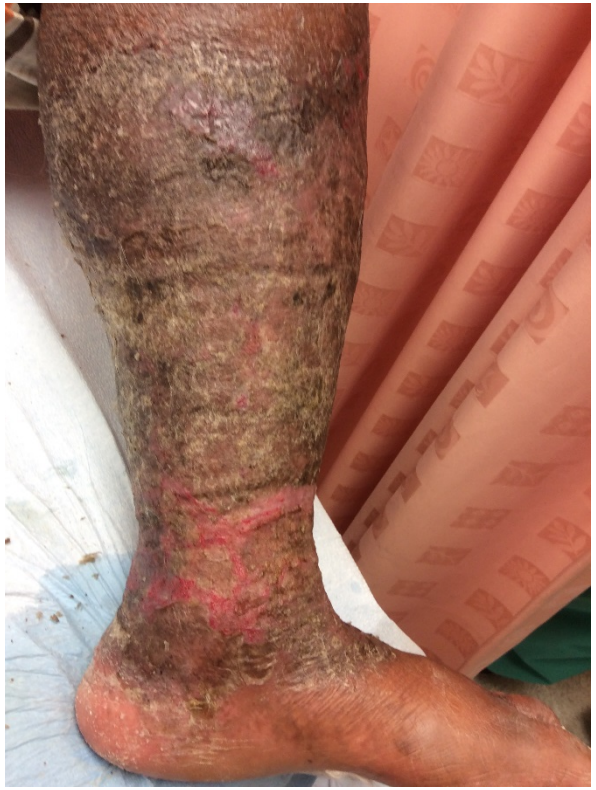
3: Edema (I89.0 – secondary lymphatic swelling)

4a: Pigmentation or eczema (stasis)

4b: Lipodermatosclerosis or atrophy blanche (fibrosis / necrosis)

5: Healed venous ulcer

6: Active venous ulcer



http://www.veinforum.org/medical-and-allied-health-professionals/avf-initiatives/ceap-and-venous-severity-scoring

Margolis DJ, Allen-Taylor L, Hoffstad O, Berlin JA. The accuracy of venous leg ulcer prognostic models in a wound care system. Wound Repair Regen. 2004;12(2):163-168

COMPLICATING FACTORS

- 20,000 patients with VLUs identified patients who were likely to heal by the 24th week of care
- Wounds older than 12 months at the first visit has a 29% chance of not healing by week 24th week of care
- Wounds < 10 cm2 were likely to heal
- Wounds > 10 cm2 **and** > 12 months old have a 78% chance of not healing
- VLUs in the presence of PAD
- Wound beds with >50% Fibrous Tissue (Inflammatory)

What does the literature say?

Compression is ‘standard of care' in management of edema, CVI and VLU [1-8]

- Improves healing rates in those with existing VLU
- Reduces likelihood of recurrence
- Improves CVI related symptoms
- Reduces swelling

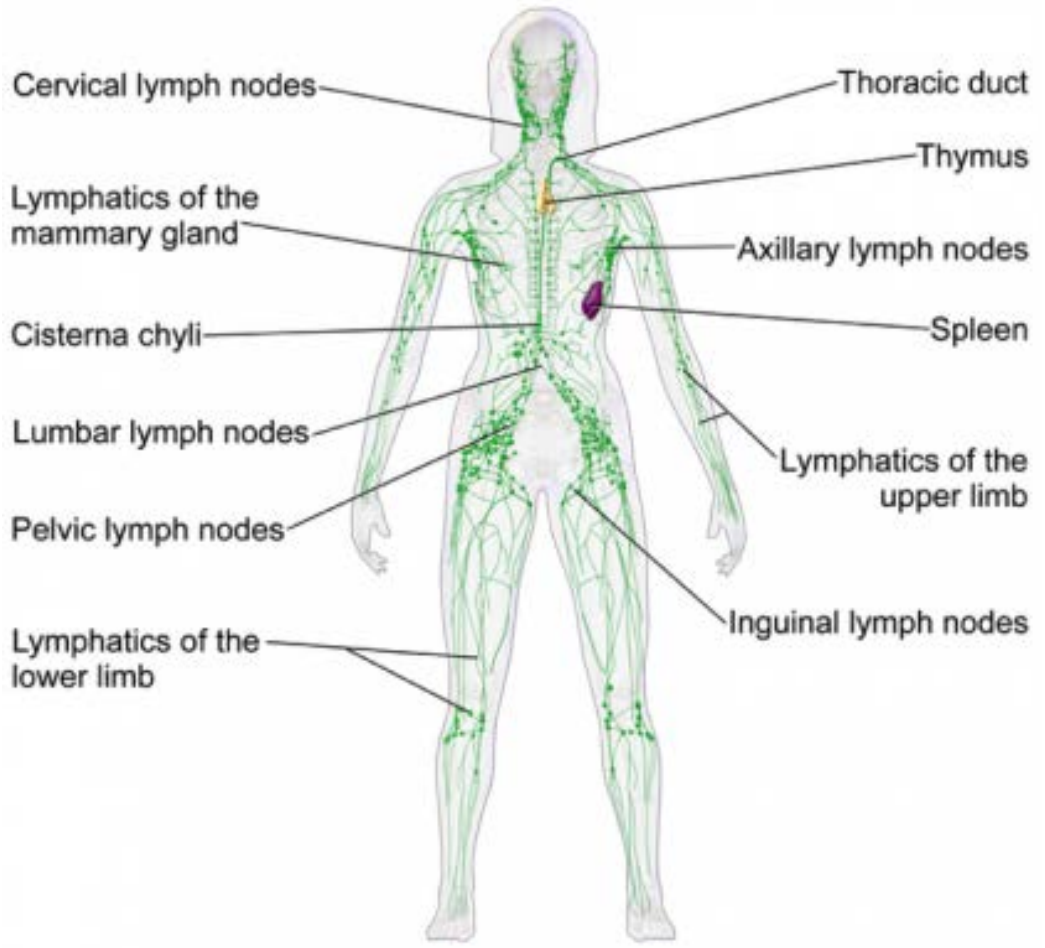
Multi-layer compression is more effective than low compression or single layer compression

Some compression is better than no compression [6]

- Improved healing rates VLU compared to no compression
- Improved CVI related symptoms (itching, stasis) when compared to no compression

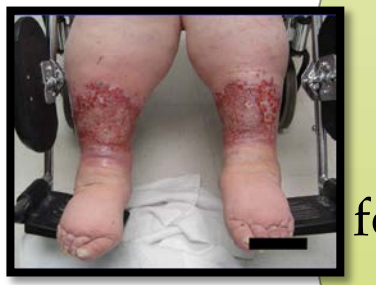
O’Meara S, Cullum N, Nelson EA, Dumville JC. Compression for venous leg ulcers. Cochrane Databse Syst Rev 2012 Nov 14.

Lymphedema The "Other" Circulatory Sytem



https://www.physio-pedia.com/The_role_of_the_Physiotherapist_in_Palliative_Care_for_people_with_Lymphoedema#cite_note-Williams_et_al._2005-16

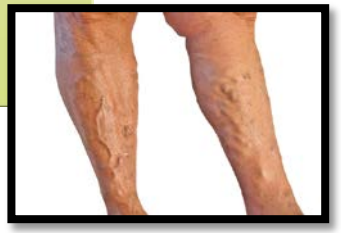
Pathophysiology of Phlebolymphedema



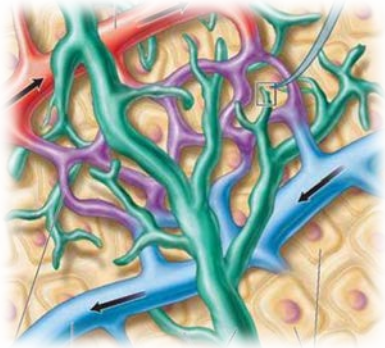
Lymphedema is underlying pathology contributing to formation of venous ulcers

Chronic venous insufficiency

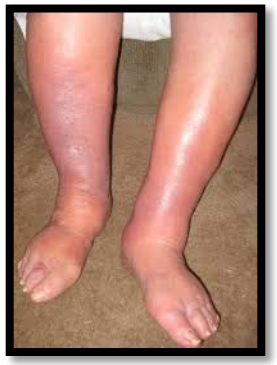
High filtration pressure/increased fluid in tissues



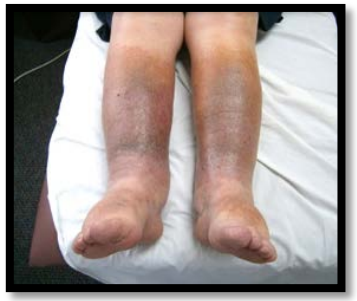
Waterload exceeds lymphatic transport capacity



Low protein edema

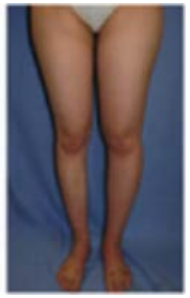
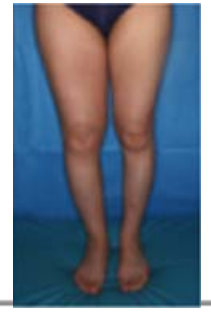
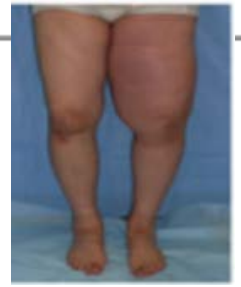
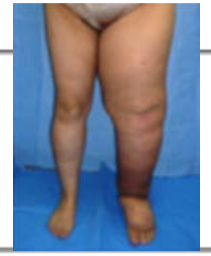


Lymphatic hypertension leads to fibrosclerosis



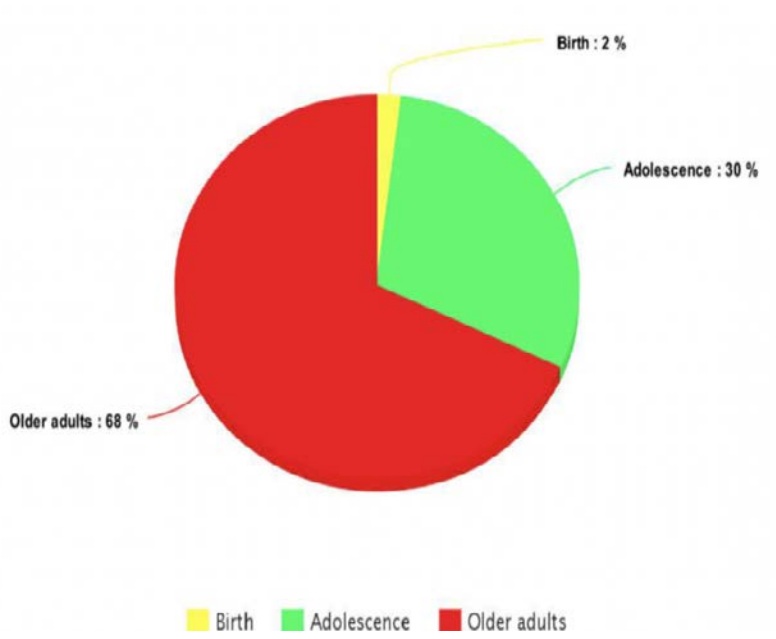
Lymphatic damage

Lymphedema Stages

Stage 0 (Subclinical or latent) 	• No visible changes • Period of time from reduced lymphatic transport to when lymphoedema occurs • Altered sensation, for example, mild heaviness • People can be in this stage for a long period of time before symptoms fully develop
Stage 1 (Mild) 	• Slight swelling in affected area • High-protein fluid builds up in the interstitium • Pitting oedema occurs when skin is pressed • Symptoms can be cured by treatment because skin isn't permanently damaged. For example, elevating the arm
Stage 2 (Moderate) 	•Results from lack of treatment during Stage 1 • Increased swelling and tissue damage present (tissue is hard) • Symptoms can be managed through treatment but not cured
Stage 3 (Severe) 	• Uncommon for people with breast cancer •Final stage, affected limb becomes deformed and skin is badly damaged

Classification of Primary Lymphedema

- Milroy's Disease – Occurs at or soon after birth (2%)
- Lymphedema Praecox – Presents before the age of 35 (30%)
- Lymphedema Tarda – Presents after the age of 35 (68%)



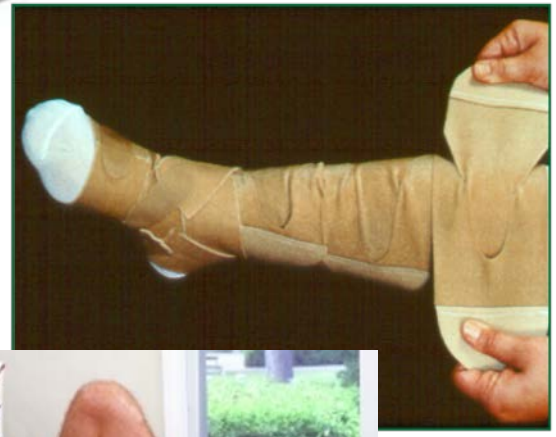
Williams AF, Franks PJ, Moffatt CJ. Lymphoedema: estimating the size of the problem. Palliat Med 2005;19:300-313.

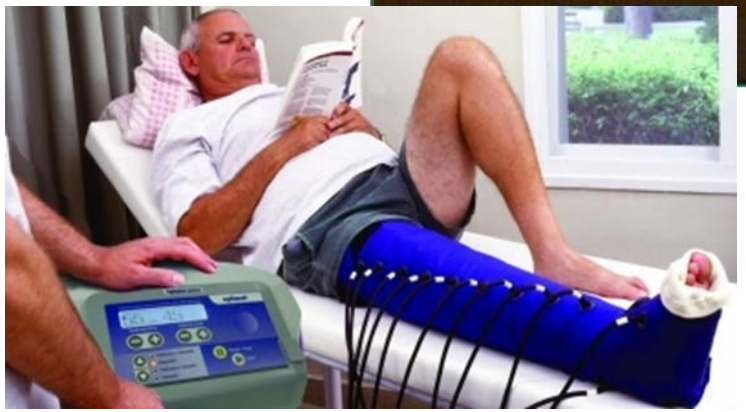
Treatment of Venous Ulcers

Compression

Compression

Compression





ABCESS

Bioload/Biofilm Management

Wound Bed Optimization

Fife, Caroline E. et al. "A Predictive Model for Diabetic Foot Ulcer Outcome: The Wound Healing Index." Advances in Wound Care 5.7 (2016): 279–287. PMC. Web. 15 Sept. 2018

- Results: Variables that significantly predicted healing were as follows:
  - Wound Age (duration in days)
  - Wound Size
  - Number of concurrent wounds of any etiology
  - Evidence of bioburden/infection
  - Patient age
  - Wagner grade
  - Non-ambulatory status
  - renal dialysis or renal transplant
  - peripheral vascular disease
  - patient hospitalization for any reason

Horn SD1, Barrett RS, Fife CE, Thomson B. A predictive model for pressure ulcer outcome: the Wound Healing Index. Adv Skin Wound Care. 2015 Dec;28(12):560-72

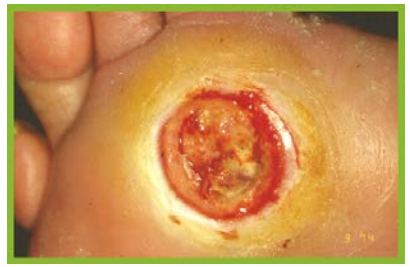
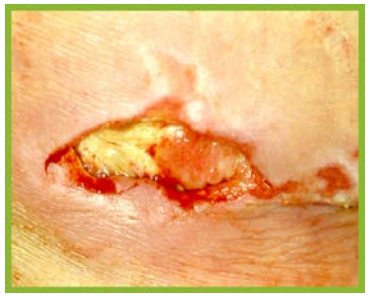
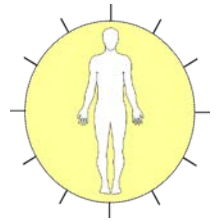
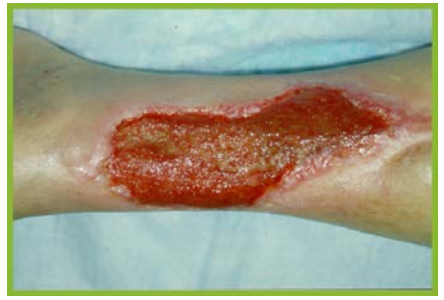
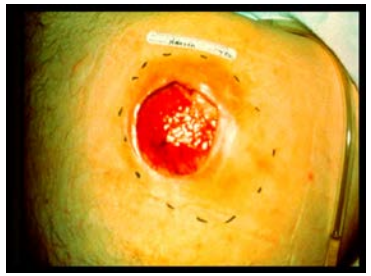
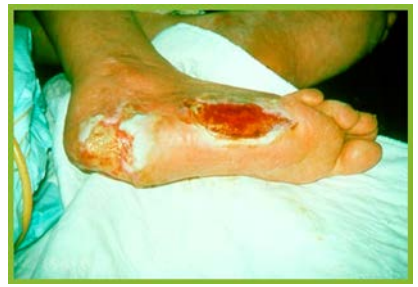
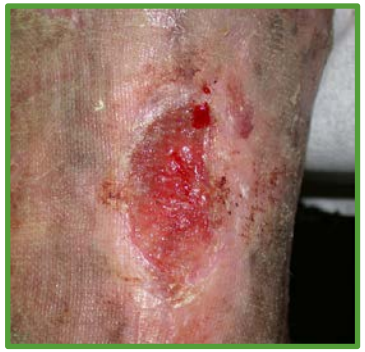
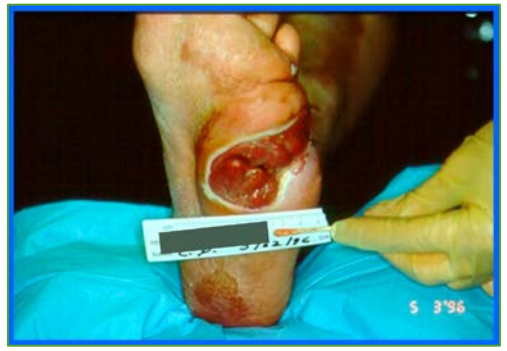
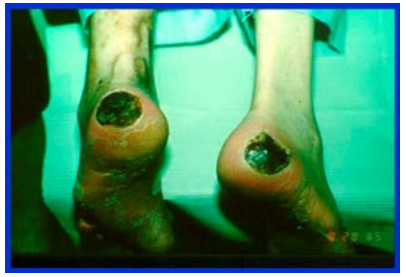
- Variables significantly predicting healing were:
  - PrU size
  - PrU age
  - Number of concurrent wounds of any etiology
  - PrU Stage III or IV
  - Evidence of bioburden/infection
  - Patient age
  - Non-ambulatory status
  - Hx of a Renal transplant
  - Paralysis
  - Malnutrition
  - Patient hospitalization for any reason.

Takahashi P. A predictive model for venous ulceration in older adults: results of a retrospective cohort study. Ostomy/wound management. 2010;56(4):60-66.

- All patients over 60 years in a primary care panel in Olmsted County, MN, on January 1, 2005 were enrolled (N=12,650.)
- 215 (1.7%) subjects developed new venous ulcers in 2 years. Previous venous ulceration was the most significant risk factor (OR 19.4; 95% CI 14.5-25.9); thus, a 19 fold increased risk of future venous ulceration.
- Renal insufficiency, blindness, cataracts and a history of decubitus ulceration were the other significant risk factors identified.
- Patients with prior venous ulceration should be considered high risk for future venous ulceration and should have aggressive edema control.

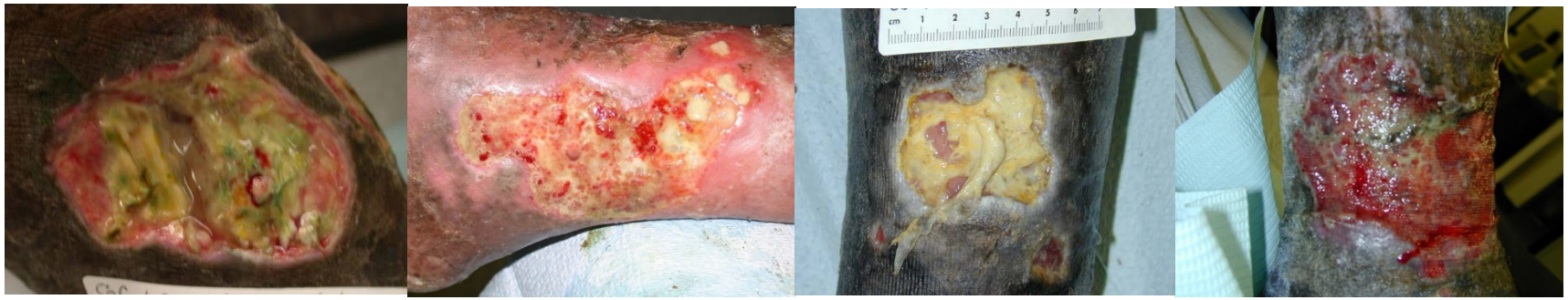
Elements of a Complete Wound Assessment

- Location /Etiology
- Deformity / Mechanical Imbalance
- Dimensions / Size
- Tunneling / Undermining
- Appearance of Wound Base
- Wound Edges
- Periwound Skin
- Exudate / Drainage
- Tissue involvement / Staging
  - UTHSC
  - NPUAP
  - Superficial/Deep

Slough Types

- Fibroslough - primarily fibrous tissue
- Leukoslough - primarily WBC accumulation
- Necroslough - primarily necrotic tissue
- Bioslough - primarily biofilm



Classification Systems

- Meggitt / Wagner - General
- UTHSC - Diabetic Foot Wound Classification
- CEAP – Venous Disease Classification
- NPUAP - Pressure Injury
- Tissue Involvement

The University of Texas Health Science Center Diabetic Foot Wound Classification System

- 4 Grades (depth)
- 4 Stages (associated comorbidity)

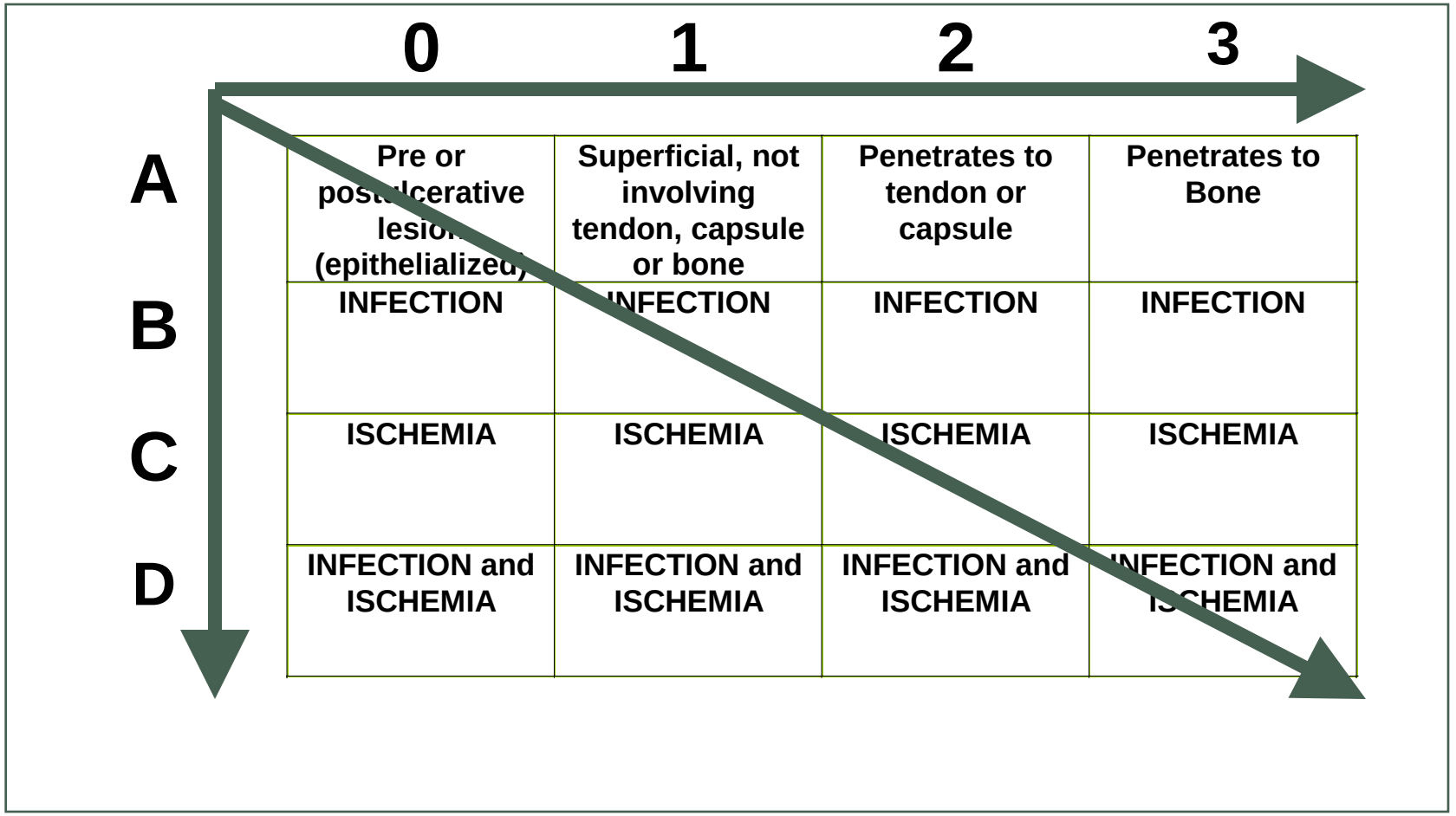
Four Grades of Depth

- Grade 0: Pre or post-ulcerative skin (completely epithelialized)
- Grade 1: Full-thickness skin
- Grade 2: Tendon and/or capsule
- Grade 3: Bone

Four Stages of Comorbidity

- A: non-ischemic clean wound
- B: infected
- C: ischemic
- D: infected & ischemic

UT Diabetic Wound Classification System



	0	1	2	3
A	Pre or post-ulcerative lesion (epithelialized)	Superficial, not involving tendon, capsule or bone	Penetrates to tendon or capsule	Penetrates to Bone
B	INFECTION	INFECTION	INFECTION	INFECTION
C	ISCHEMIA	ISCHEMIA	ISCHEMIA	ISCHEMIA
D	INFECTION and ISCHEMIA	INFECTION and ISCHEMIA	INFECTION and ISCHEMIA	INFECTION and ISCHEMIA

APWH
Academy of Physicians in Wound Healing

Meggitt-Wagner Ulcer Classification System

• 0- Intact Skin • 1- Superficial Ulcer • 2- Deep to Tendon, Bone or Joint	**Depth**
• 3- Deep with abscess-ostitis	**Infection**
• 4- Forefoot Gangrene • 5- Whole Foot Gangrene	**Ischemia**

Meggitt, Br J Hosp Med, 1975

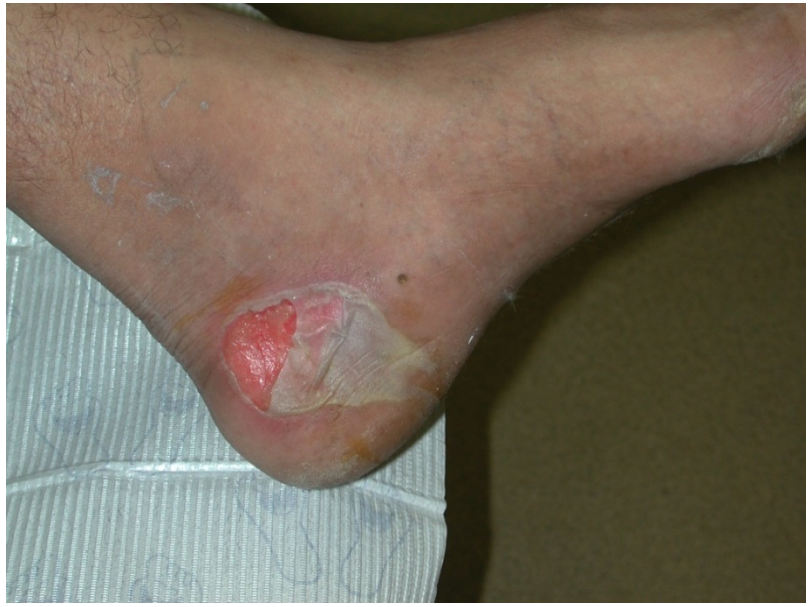
Wagner, Foot Ankle, 1981

Preulcer

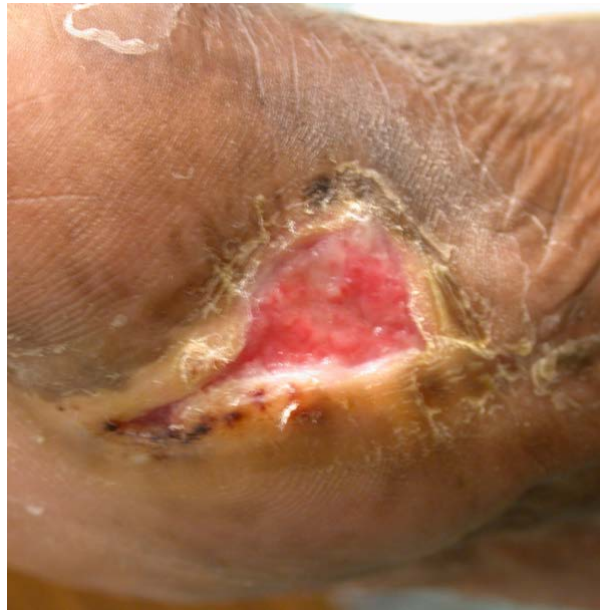
- Temp↑ >2°F when compared to the same site on the opposite foot
- Intradermal maceration
- Callus with underlying hematoma

Tissue Involvement Classification

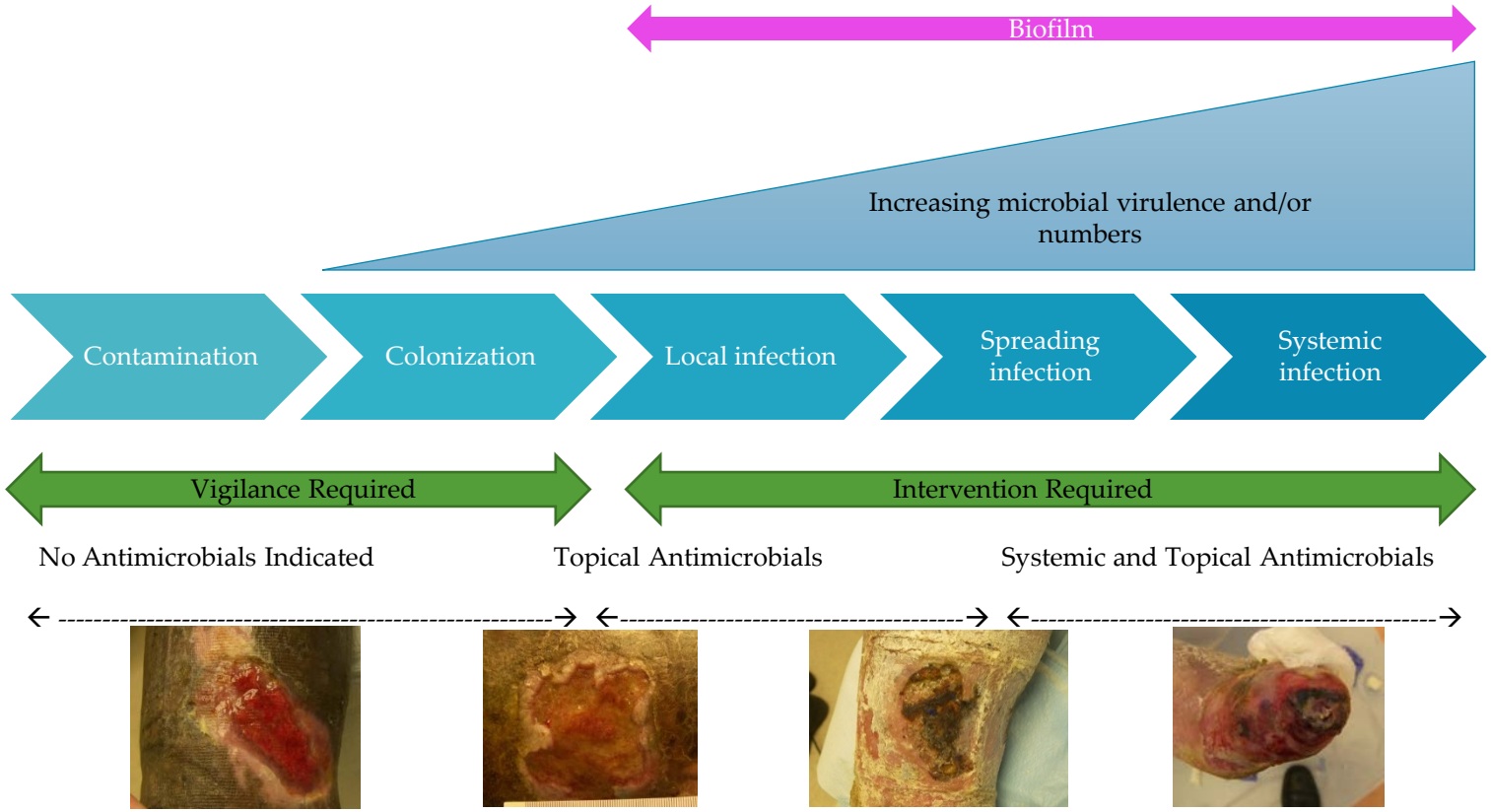
Partial Thickness



Full Thickness



The New IWII Wound Infection Continuum

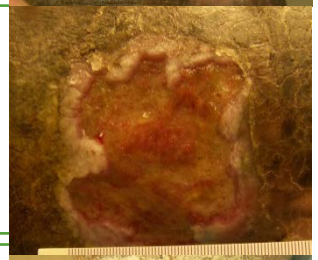


International Wound Infection Institute (IWII) *Wound infection in clinical practice.* Wounds International 2016

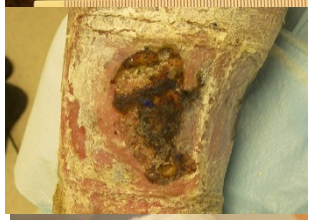
The Biofilm Continuum

- Planktonic

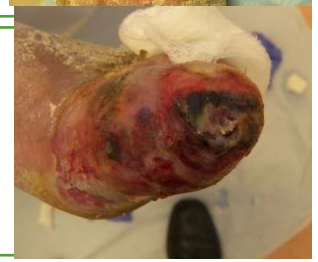
- Early Biofilm Formation



- Mature Biofilm Formation

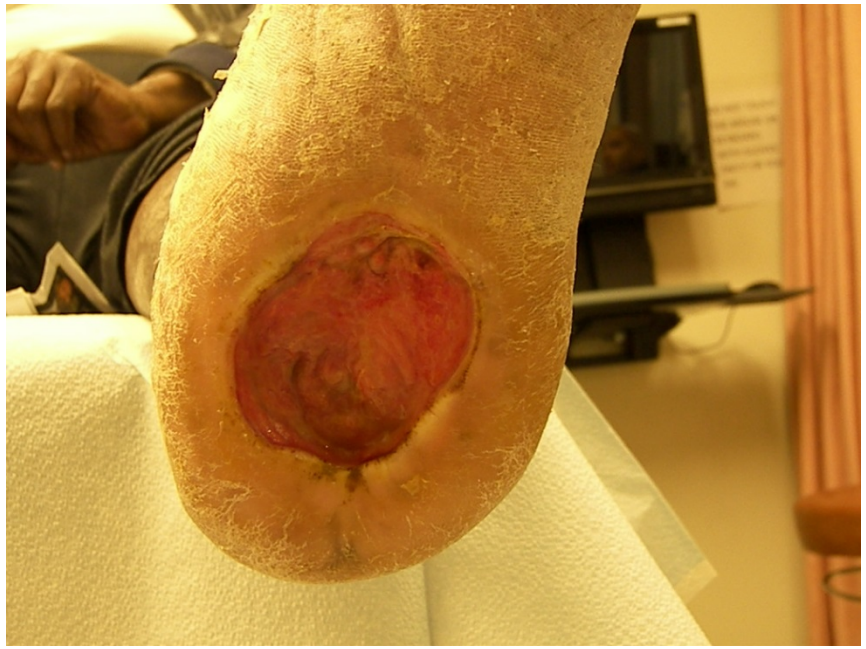


- Local to Systemic Infection
- Biofilm Seeding



Secondary Signs of Infection = 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

ABCESS

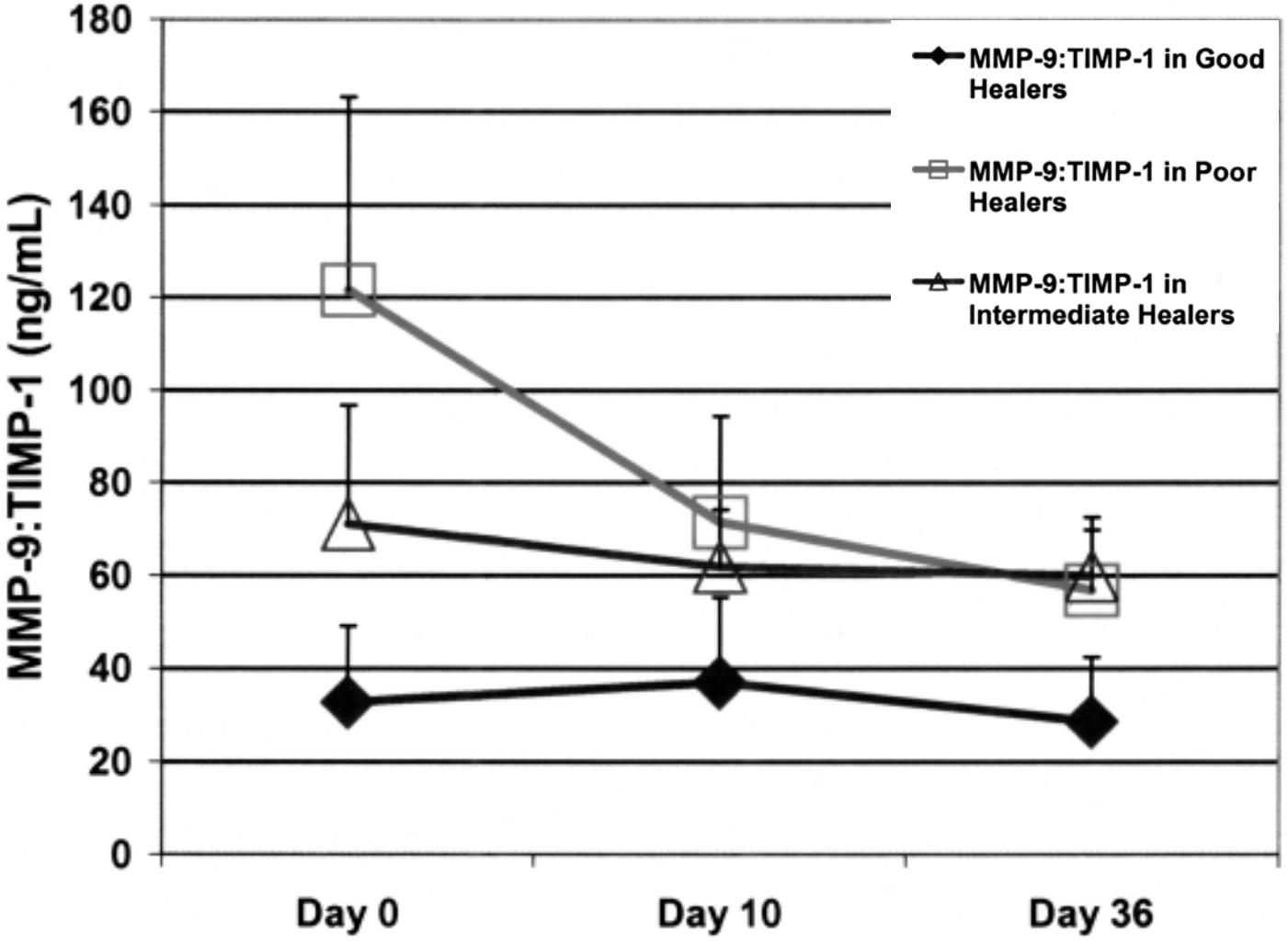
Cellular Assessment

Proteases

Proteases in Normal Wound Healing

- Inflammation n Removal of damaged ECM (aids autolytic debridement)
- Proliferation n Degradation of capillary basement membrane for angiogenesis
- Aiding detachment and migration of cells
- Remodelling n Contraction of scar ECM
- Remodelling of scar ECM

Elevated MMP Activity is a Predictor of Healing



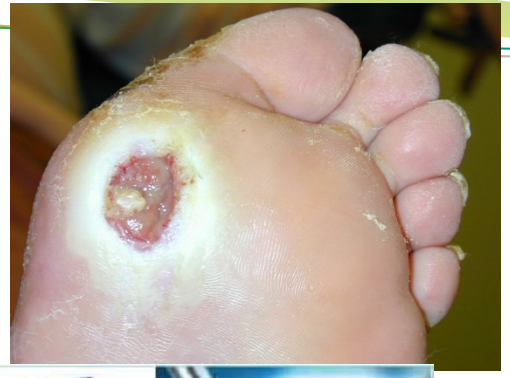
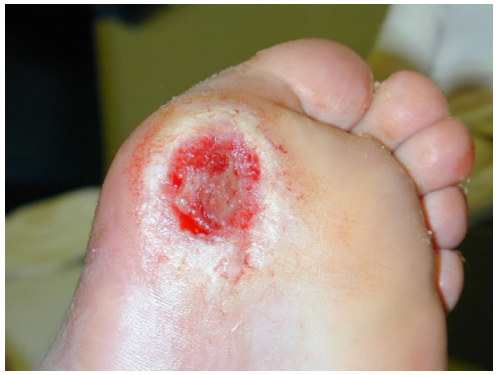
Ladwig, Robson, Liu, Kuhn, Muir, Schultz. Ratios of activated matrix metalloproteinase-9 to tissue inhibitor of matrix metalloproteinase-1 in wound fluids are inversely correlated with healing of pressure ulcers. Wound Repair Reg 10:26-37, 2002

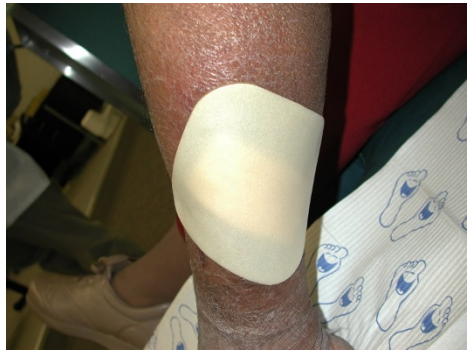
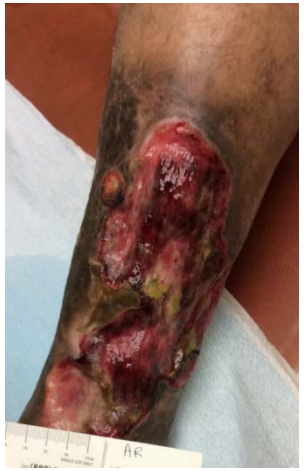
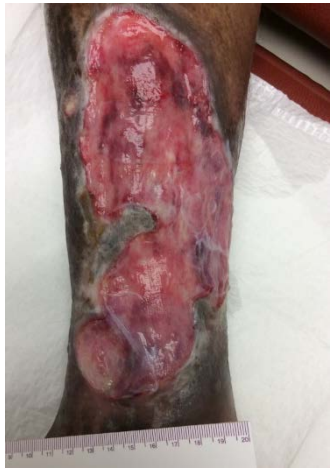
Interventions That Reduce Proteases

- Cleansing
- Debridement
- Protease inactivators: protease-modulating
- Antiseptic dressings
- Anti-inflammatories
- Dressings and devices that absorb/remove wound exudate

Debridement

- Macro
  - Surgical
  - Mechanical
- Micro
  - Autolytic
  - Enzymatic
  - Biosurgial


Sequential Biomarker Profiles May Soon be Available

- Identify Tissue Gene Transcription Biomarkers
- Healed (Antiinflammatory): IFN-ϒ, IL-4, IL-5, IL-10, IL-13, Interferon Protein -10, IL-3, M2
- Dehisced (Inflammatory): TNF-α, IL-1α, IL-1β, IL-2, Monocyte Chemotactic Protein-1, Macrophage Inflammatory Protein-1$^{\alpha}$, Granulocyte-macrophage colony-stimulating factor, M1

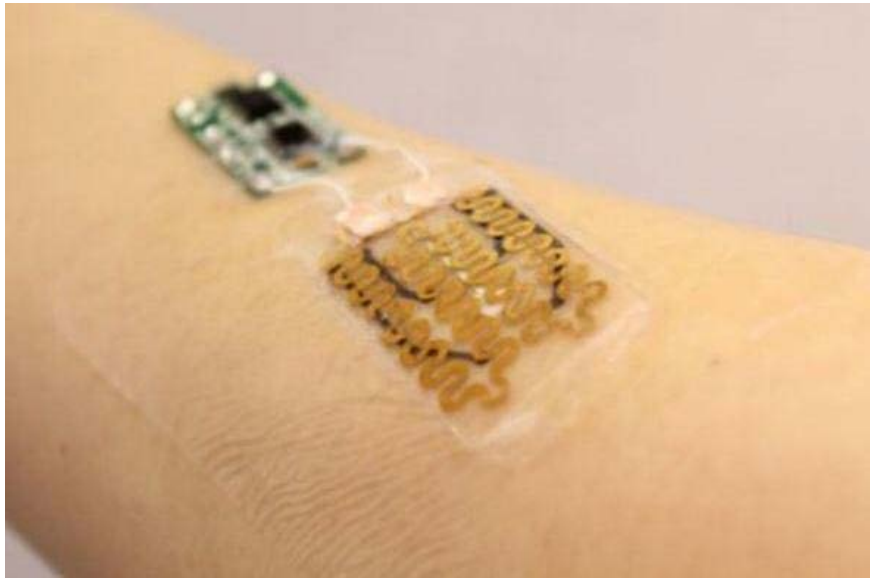
Hawksworth JS, Stojadinovic A, Gage FA, et al. Inflammatory Biomarkers in Combat wound Healing. Annals of Surgery, Vol. 250, No. 6, Dec 2009

M1, M2 Macrophages

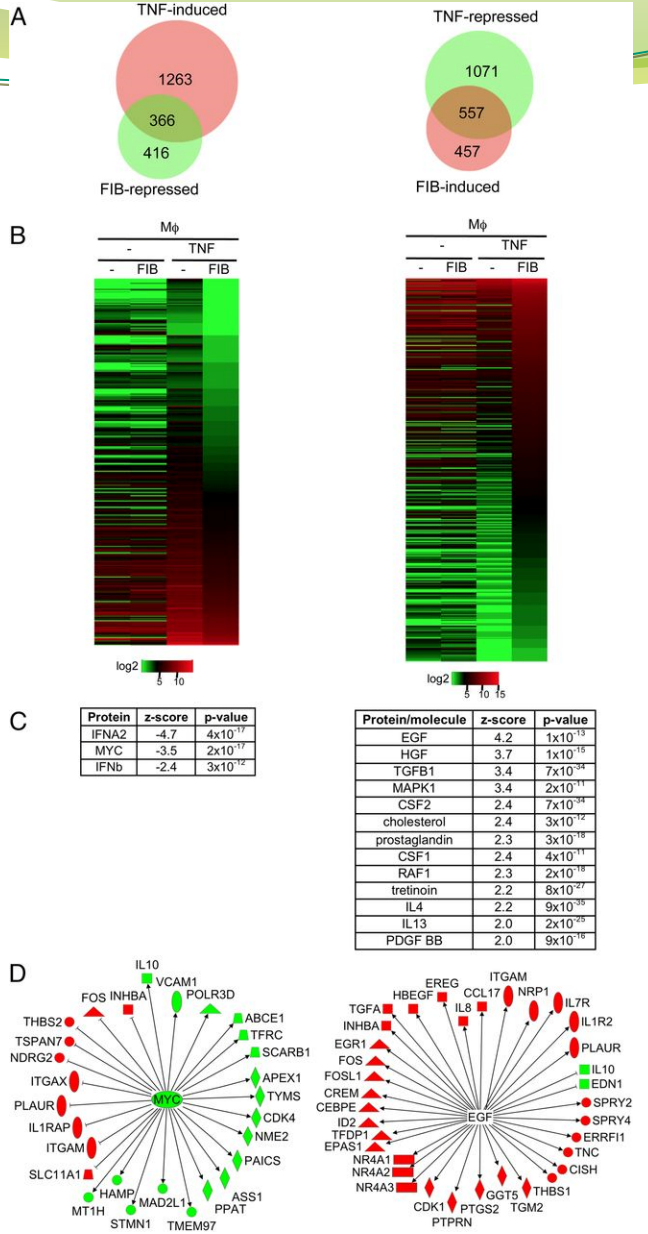
- M1/M2 describes the two major and opposing phenotype activities of macrophages
- M1 (inflammatory) their presence in the wound indicates a highly inflammatory wound with removal of necrotic debris and bacteria
- M2 (proliferative) cells indicates an environment of cell proliferation and tissue repair

Mills CD. M1 and M2 Macrophages: Oracles of Health and Disease. Crit Rev Immunol. 2012;32(6):463-88.

Smart Bandage



https://www.upi.com/Health_News/2018/07/09/Smart-bandage-can-monitor-wounds-trigger-drug-release/5711531142351/



Protein	z-score	p-value
IFNA2	-4.7	$4x10^{-17}$
MYC	-3.5	$2x10^{-17}$
IFNb	-2.4	$3x10^{-12}$

Protein/molecule	z-score	p-value
EGF	4.2	$1x10^{-13}$
HGF	3.7	$1x10^{-15}$
TGFB1	3.4	$7x10^{-34}$
MAPK1	3.4	$2x10^{-11}$
CSF2	2.4	$7x10^{-34}$
cholesterol	2.4	$3x10^{-12}$
prostaglandin	2.3	$3x10^{-18}$
CSF1	2.4	$4x10^{-11}$
RAF1	2.3	$2x10^{-18}$
tretinoin	2.2	$8x10^{-27}$
IL4	2.2	$9x10^{-35}$
IL13	2.0	$2x10^{-25}$
PDGF BB	2.0	$9x10^{-16}$

Donlin LT, et al. Modulation of TNF-Induced Macrophage Polarization by Synovial Fibroblasts. The Journal of Immunology September 1, 2014, 193 (5) 2373-2383

Cellular Tissue-Based Products

Excerpts from: Skin Substitutes for Treating Chronic Wounds Technology Assessment Report, December 18, 2012 https://www.ncbi.nlm.nih.gov/books/NBK248357/

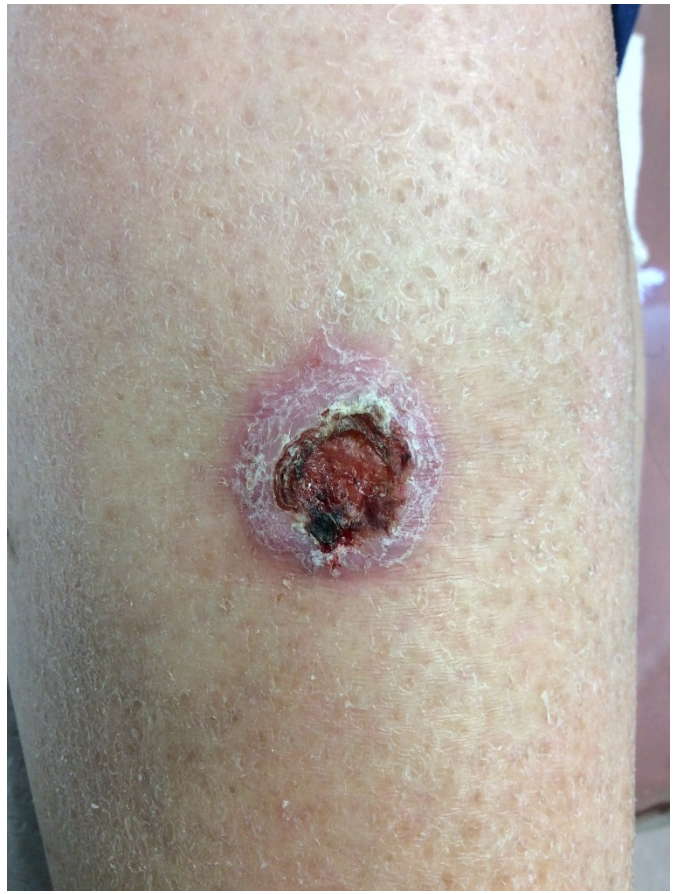
Comparison	# Studies (Total N)	Overall Risk of Bias	Precision
Apligraf vs. TheraSkin[64]	1 (28)	MOD	I % healed at 12 weeks: 41% vs. 67%, NS
Oasis Wound Matrix vs. Dermagraft[54]	1 (26)	MOD	I % healed at 12 weeks 77% vs. 85%, NS
Graftjacket acellular matrix vs. Moist wound therapy[66]	1 (85)[a]	LOW	P % healed at 12 weeks: 70% vs. 46%, p=0.03
Graftjacket acellular matrix vs. weekly debridement, Curasol wound hydrogel and gauze dressing[59]	1 (28)	MOD	P % healed at 12 weeks: 57% vs. 7%, p=0.001
Oasis Wound Matrix vs. Regranex Gel[70]	1 (98)	MOD	I % healed at 12 weeks: 49% vs. 28%, NS
Apligraf vs. nonadherent dressing[53]	1 (72)[a]	LOW	P % healed at 12 weeks: 52% vs. 26%, p=0.03
Dermagraft vs. saline-moistened gauze[56,62,65]	3 (530)	MOD	P OR 1.64 (95% CI 1.10 to 2.43)
Graftskin vs. saline-moistened gauze[67]	1 (208)[a]	LOW	P % healed at 12 weeks 56%vs. 38%, p=0.01
Hyalograft 3D autograft/LaserSkin vs. nonadherent paraffin gauze[58,68]	2 (239)	LOW	I OR 1.43 (95% CI 0.80 to 2.54)

Excerps from : Skin Substitutes for Treating Chronic Wounds Technology Assessment Report, December 18, 2012 https://www.ncbi.nlm.nih.gov/books/NBK248357/

Study	Wound Type	Skin Substitute	Comparison	Number of Patients in Study	Difference in Rate of Wounds Healed (Skin Substitute – Comparator)	p-Value[a]	Relative Risk for Complete Wound Healing (95% CI) for Skin Substitute vs. Comparator[a]
DiDomenico et al. 2011[64]	DFU	Apligraf	TheraSkin	28	Healed at 12 weeks 41% - 67% = -26%	NS (p=0.21)	0.66 (0.33 to 1.30)
Landsman et al. 2008[54]	DFU	Oasis Wound Matrix	Dermagraft	26	Healed at 12 weeks 77% - 85% = -8%	NS (p=0.62)	0.91 (0.62 to 1.33)
Reyzelman et al. 2009[66]	DFU	Graftjacket acellular matrix	Moist wound therapy with alginates, foams, hydrocolloids, or hydrogels	85	Healed at 12 weeks 70% - 46% = 24%	0.03	1.51 (1.02 to 2.22)
Brigido 2006[59]	DFU	Graftjacket acellular matrix	Weekly debridement, Curasol wound hydrogel and gauze dressing	28	Healed at 12 weeks 57% - 7% = 50%	0.001	8.00 (1.15 to 55.80)
Niezgoda et al. 2005[70]	DFU	Oasis Wound Matrix	Regranex Gel (contains platelet-derived growth factor)	98	Healed at 12 weeks 49% - 28% = 21%	NS (p=0.06)	1.75 (0.94 to 3.26)
Edmonds 2009[53]	DFU	Apligraf	Nonadherent dressing	72	Healed at 12 weeks 52% - 26% = 26%	0.03	1.96 (1.05 to 3.66)
Marston et al. 2003[56]	DFU	Dermagraft	Saline-moistened gauze	245	Healed at 12 weeks 30% - 18% = 12%	0.03	1.64 (1.03 to 2.62)
Naughton et al. 1997[62]	DFU	Dermagraft	Saline-moistened gauze	109	Healed at 12 weeks 39% - 32% = 7%	NS (p=0.28)	1.21 (0.86 to 1.72)
Gentzkow et al. 1996[65]	DFU	Dermagraft	Saline-moistened gauze	50	Healed at 12 weeks[b] 30% - 8% = 22%	0.04	1.93 (0.49 to 7.59)
Veves et al. 2001[67]	DFU	Graftskin	Saline-moistened gauze	208	Healed at 12 weeks 56% - 38% = 18%	0.01	1.50 (1.11 to 2.04)

When do I biopsy?

- If there is clinical suspicion of skin cancers (raised boarders, crusting, changes in shape or color, any change at all)
- Previous history or family history of skin cancers
- If a wound is not responding to traditional diagnosis specific treatment protocols
- Re-biopsy if wound continues to “ languish”



O'Donnell, Thomas F. et al. Management of venous leg ulcers: Clinical practice guidelines of the Society for Vascular Surgery® and the American Venous Forum. Journal of Vascular Surgery , Volume 60 , Issue 2 , 3S - 59S
Guidelines for the Management of Venous Ulcer

- Guideline 3.5: Wound Biopsy
- We recommend wound biopsy for leg ulcers that do not improve with standard wound and compression therapy after 4 to 6 weeks of treatment and for all ulcers with atypical features. [GRADE - 1; LEVEL OF EVIDENCE - C]

ABCESS

Exudate Management

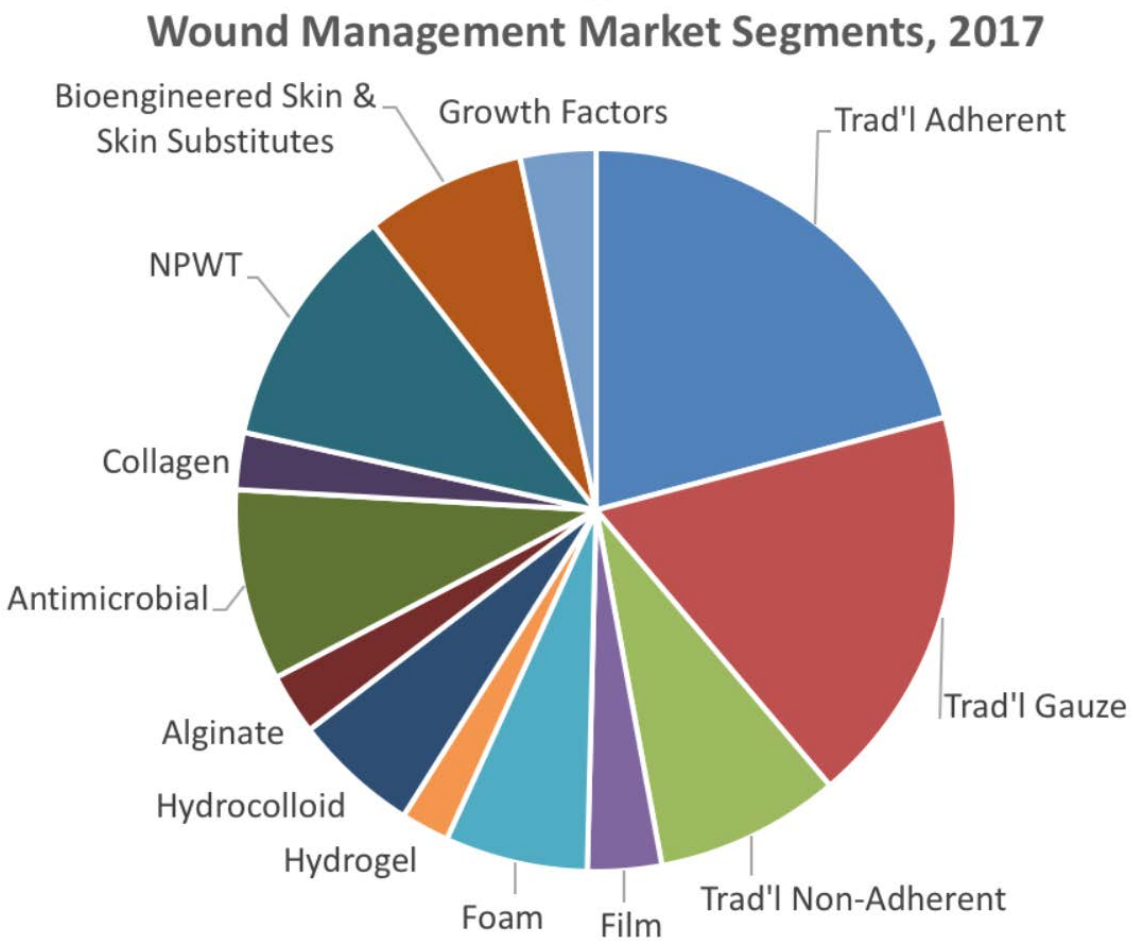
Dressing selection

Characteristics of an Ideal Wound Dressing

- **Removes excess exudate and toxins**
- **High humidity at the dressing wound interface**
- **Allows for gaseous exchange**
- **Provides thermal insulation**
- **Protects against secondary infection**
- **Free from particulate and toxic components**
- **No trauma with removal**

Turner TD. Hospital usage of absorbent dressings. *Pharma J* 1979;222:421-426

Dressing Use



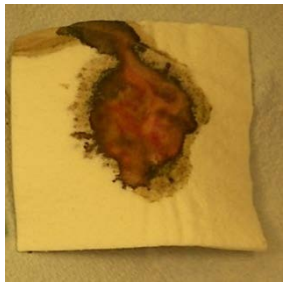
https://blog.mediligence.com/2018/03/22/the-physiology-of-wound-healing/

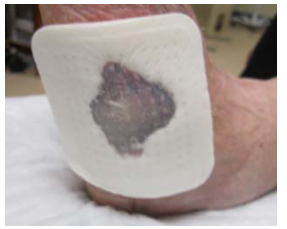
Expanded Categories of Wound Dressings

- **Enzymatic Debriding Agents-Collagenase**
- **Surfactant-based debriding Agents**
- **Antibiofilm Agents- HOCl, Silver gels, I2, Xylitol, Lactoferrin, EDTA**
- **Collagens- gels, dressings**
- **Nanoparticle dressings**
- **Antimicrobial Dressings - Silver, Cadexomer Iodine, PHMBG, Gentian Violet/Methylene Blue, I2**
- **Negative Pressure Therapy**
- **Cellular and Acellular Tissue-Based Products**
- **Extracellular matrix components-HLA**
- **Growth factor applications**
- **Gauze**

Advanced Dressings Designed to Address Fluid Management

- Alginates
- Hydrofibers
- Foams
- Hydroconductives
- Active Fluid Management
- Super Absorbant Polymers
- NPWT



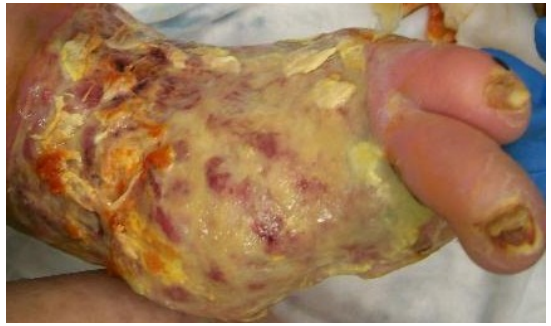


ABCESs

Systemic Illness

Common Barriers to healing

- Tissue Oxygen < 30 mm Hg (Hypoxia)
- Blood Sugars > 350 (Systemic Disease)
- Albumin less than 2.7 (Malnutrition)
- Repetitive Trauma (Pressure)
- Increased “Bioburden” (“Infection”)



Systemic Factors Affecting Wound Healing

- Nutrition and hydration
- Medications
  - Steroids
  - ASA
  - Anticoagulants
  - Chemotherapy
- Infection
- Incontinence
- Immobility
  - Use of calf muscles
- Co-morbid disease states
  - Diabetes
  - PAD
  - Inflammatory diseases
  - Anemia
  - Hyper- or hypotension
  - Hyper or hypothyroid
  - COPD
  - Sickle cell
  - CVA
  - Renal disease
  - Age
  - Past radiation

Common Systemic Conditions Affecting Wound Chronicity

- Endocrine diseases (eg, diabetes, hypothyroidism)
- Hematologic conditions (eg, anemia, SS, polycythemia, myeloproliferative disorders)
- Cardiopulmonary problems (eg, chronic obstructive pulmonary disease, congestive heart failure)
- GI problems that cause malnutrition and vitamin deficiencies, obesity
- Autoimmune disorders (eg, RA, SLE, Colitis)

Laboratory Workup for the Wound Care Patient

- Albumin
- Prealbumin
- Transferrin
- Total lymphocyte count
- Vitamins: A,E,C
- Minerals: Zn, Fe
- Electrolytes
- Glucose, Hemoglobin A_{1c}
- Hepatic:ALT, AST, GGT, Alk Phos, PT, PTT, INR
- Lipids: Total Cholesterol, HDL, LDL, VDL
- Amylase/lipase
- Iron/ferritin
- Parathyroid hormone
- Complete blood count with differential
- Sedimentation rate
- Glucose 6 phosphate dehydrogenase
- Protein C/S
- Fibrinogen/FDP/D-DIMERS
- Prothrombin time/partial thromboplastin time
- Cryoglobulins/cryofibrinogens
- Antiphospholipid antibodies
- Sickle cell
- Serum protein electrophoresis
- Antithrombin 3

Nutritional Assessment

- Chronic malnutrition can be diagnosed by using anthropometric data to compare actual and ideal body weights and by observing low serum albumin levels.
- Serum prealbumin is sensitive for relatively acute malnutrition because its half-life is 2-3 days (vs 21 d for albumin).
- A serum prealbumin level of less than 7 g/dL suggests severe protein-calorie malnutrition.
- Vitamin A deficiency reduces fibronectin on the wound surface, cell chemotaxis, adhesion, and tissue repair
- Vitamin C is required for the hydroxylation of proline and subsequent collagen synthesis.

Daley BJ, Geibel J. Wound Care Treatment & Management.
http://emedicine.medscape.com/article/194018-treatment

ABCESS
Skin Protection and Treatment

Affects of Aging

- thinning the epidermis, dermis, and subcutaneous layers
- reducing epidermal turnover, increasing time to healing, decreasing the injury response
- compromising barrier function as the rete pegs lose their depth resulting in a loosening of the normal anchoring of the epidermis to the dermis through the basement membrane
- delaying chemical clearance rate and reduced sensory perception
- decreasing immune function, vascular responsiveness, thermoregulation, and the production of sweat, sebum, and vitamin D1,5,6

Woo K, Sibbald G, Ayello E. Dermatology: The skin and periwound skin disorders and management. Wound Healing Southern Africa 2009 Volume 2 No 2 . Pp. 1-6

Glycosolation

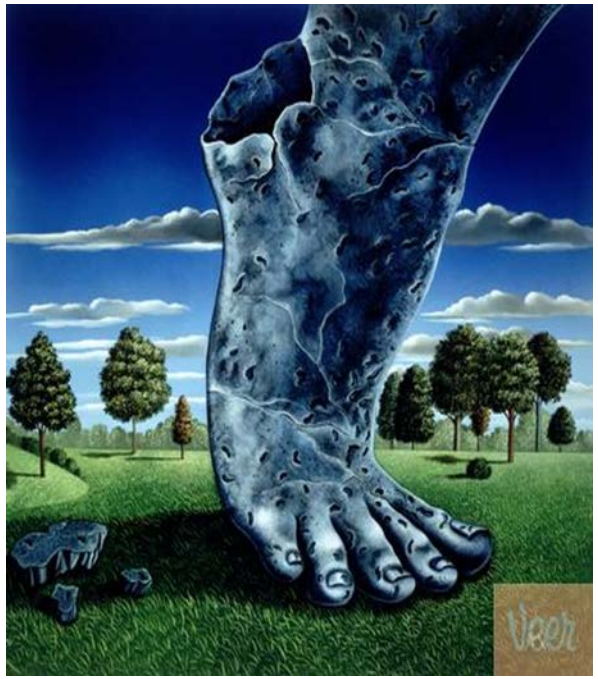
- Loss of elasticity
- Thinning of plantar fat pad
- Stiffening of the plantar fat pad
- Loss of connective tissue flexibility (eg, equinus, hammertoes)
- Loss of protective sensation
- Diminished healing response

Moisture associated skin damage-MASD

- wound exudate leading to periwound maceration - dressings
- incontinence from urine, stool or both leading to incontinence associated dermatitis (IAD)
- effluent from a stoma or fistula
- perspiration including for example excessive moisture in skin folds

Pressure Offloading

- Foot protection boots/orthoses - PRAFOs
- Seat Cushions
- Mattresses



Transitional Approach to Off-Loading

Open Wound

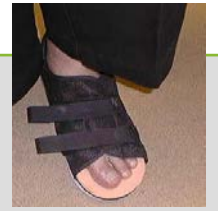
- TCC / Instant Total Contact Cast
- Wound Isolation TCC
- Football Dressing

Shallow Wound or Newly Closed

- Removable Cast Walkers
- Carville Healing Sandal
- Felted Foam

Newly Healed Wound

- Orthowedge Shoe
- Diabetic Healing Shoe



Closed Wound x 2-4 Weeks

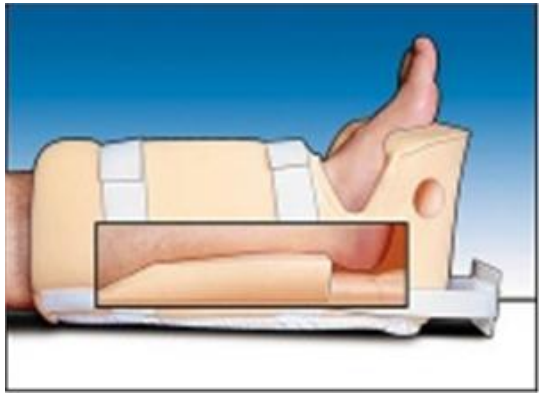
- Depth Shoe with Rocker Sole

McGuire, J. Transitional Off-loading: An Evidence-Based Approach to Pressure Redistribution in the Diabetic Foot. Advances in Skin & Wound Care. April 2010 - Volume 23 - Issue 4 - pp 175-188

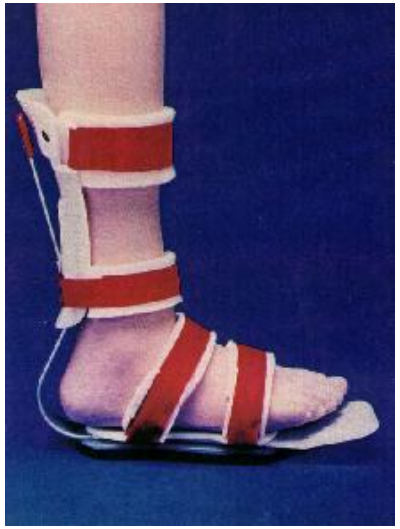
Offloading Devices






Offloading Devices



Pressure Reducing AFO

Classifications

- Group 1: Static devices/mattresses or overlays
- Group 2: Dynamic
  - Low Air Loss
  - Alternating Air

- Group 3: Air-fluidized
  - Original Model

  - New Model

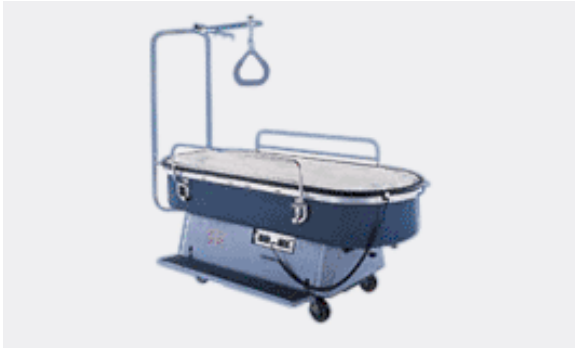
Group I: Static devices/mattresses or mattress overlays

- Air
- Foam
- Gel
- Water Overlay

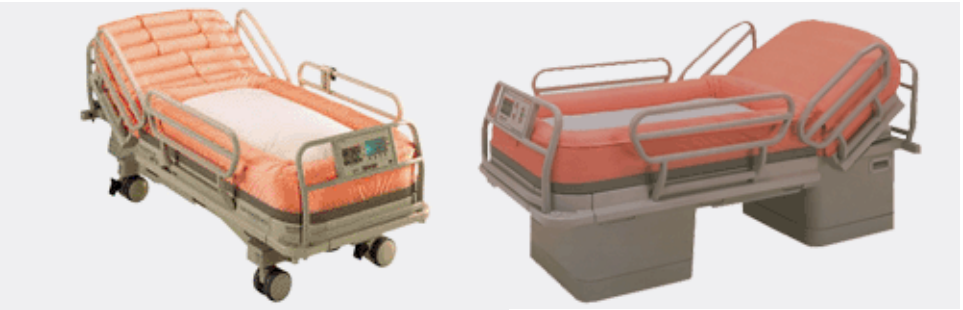
Group 2- Low air loss Alternating pre

Group 3: Air Fluidized Bed

The “Original Model”



New & Improved



QUESTIONS?
THANK YOU

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