

Wound Care 101 for Primary Care Providers

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Objective

- To identify different types of chronic wounds and the choice of dressings
- To impart knowledge about the equipment/assistive devices that can be ordered at primary care office for wound care
- To understand the role of antibiotics in wound healing
- To give a brief overview of advanced therapies that are available in wound care

A Brief Overview of Wound Healing

WOUND is alteration in Skin Integrity



WOUND is alteration in Skin Integrity

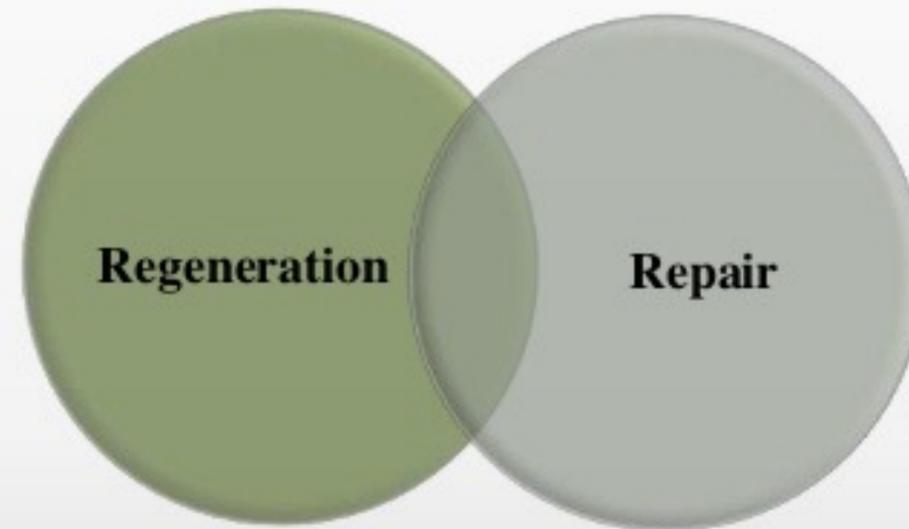
- May be caused by:
 - Disease
 - Injury
 - Surgery
 - Environmental circumstances (i.e., immobility, incontinence, eg pressure ulcer)

Wound Healing

A complex integrated sequence of cellular, physiologic, and biochemical events initiated by the stimulus of injury to tissue

Process of healing

- It involves 2 distinct processes :



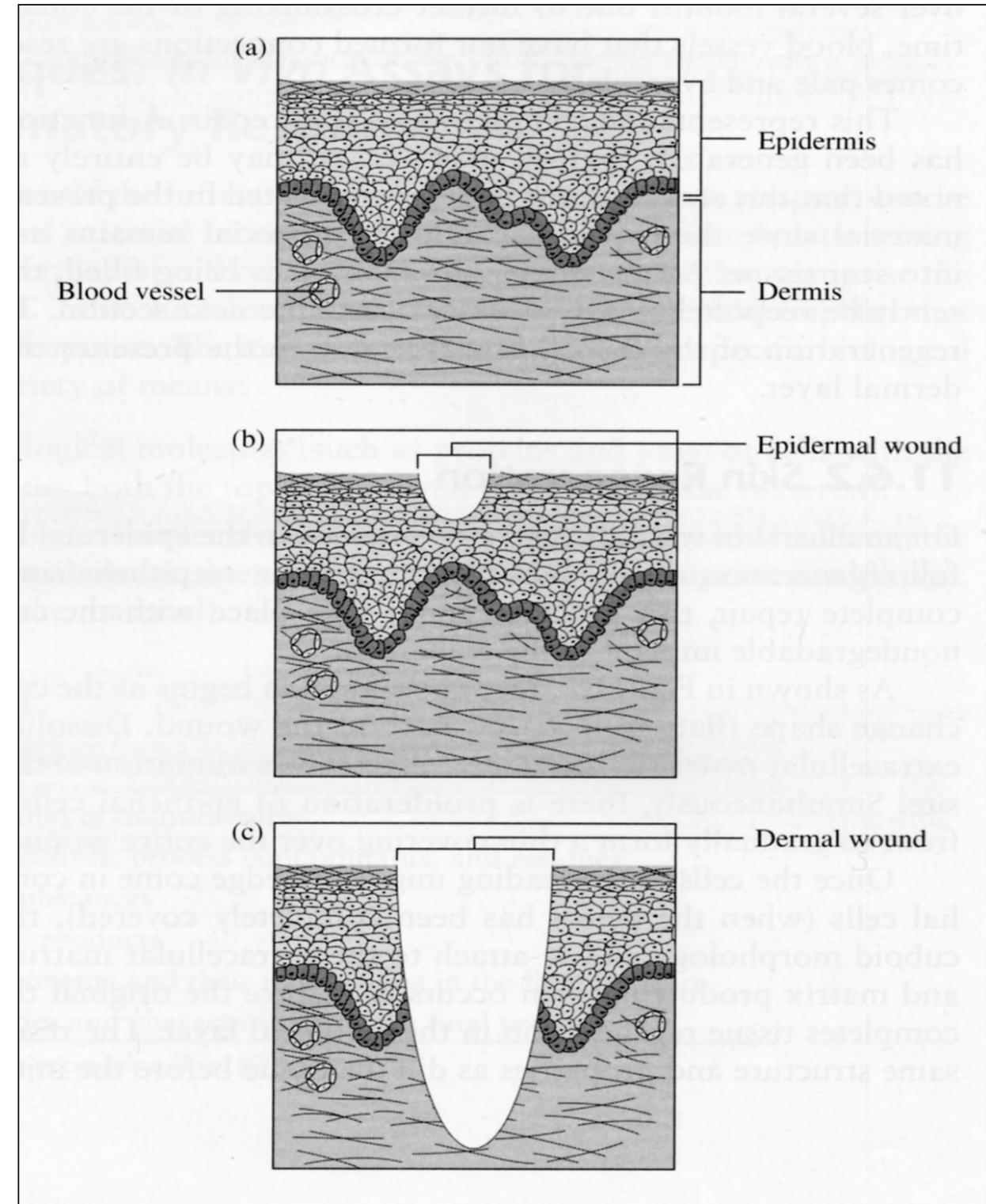
- At times, both the processes take place simultaneously



Repair vs. Regeneration

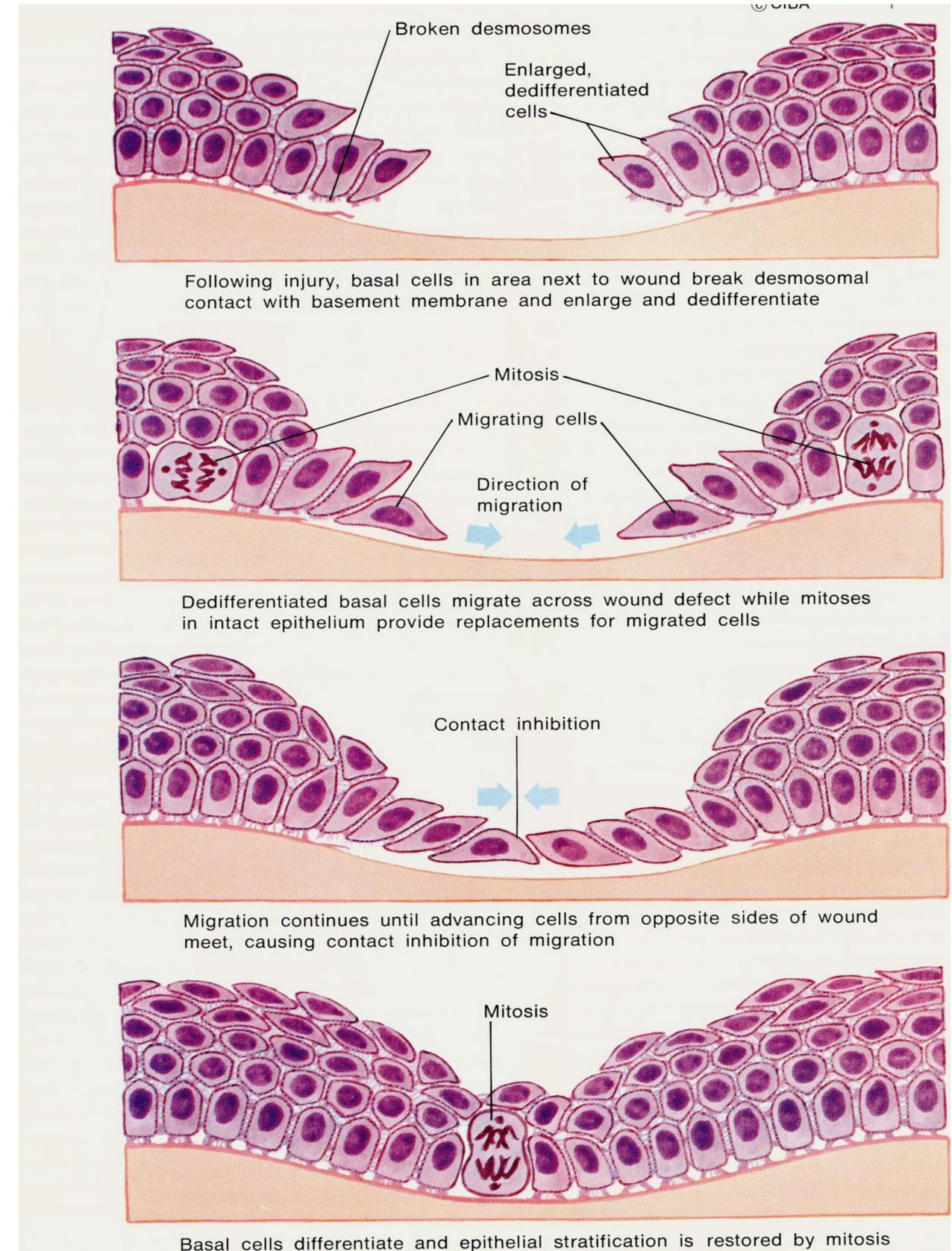
Wound healing in Skin

- Repair involves healing of the internal dermal layer
- Regeneration is regrowth of thin outer epidermal layer

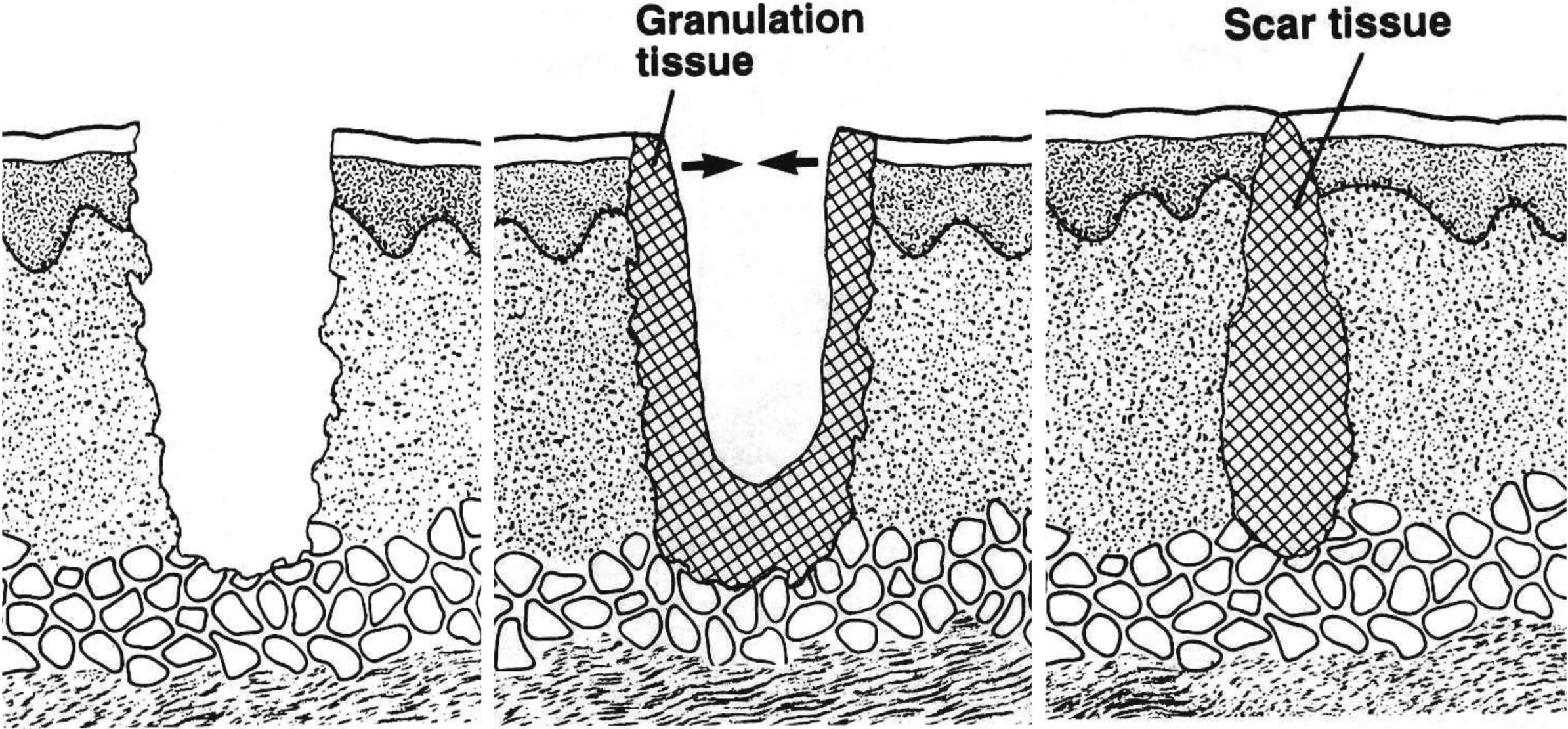


Epithelial Regeneration

- Epithelial cells detach from the basal layer
- Cells migrate towards the wound (contact guidance)
- Migrated cells are replaced by rapid mitosis
- Migration continues until epithelial cells come in contact on all sides with other epithelial cells (contact inhibition)
- Contact inhibition does not occur when epithelial cells encounter cells of other types

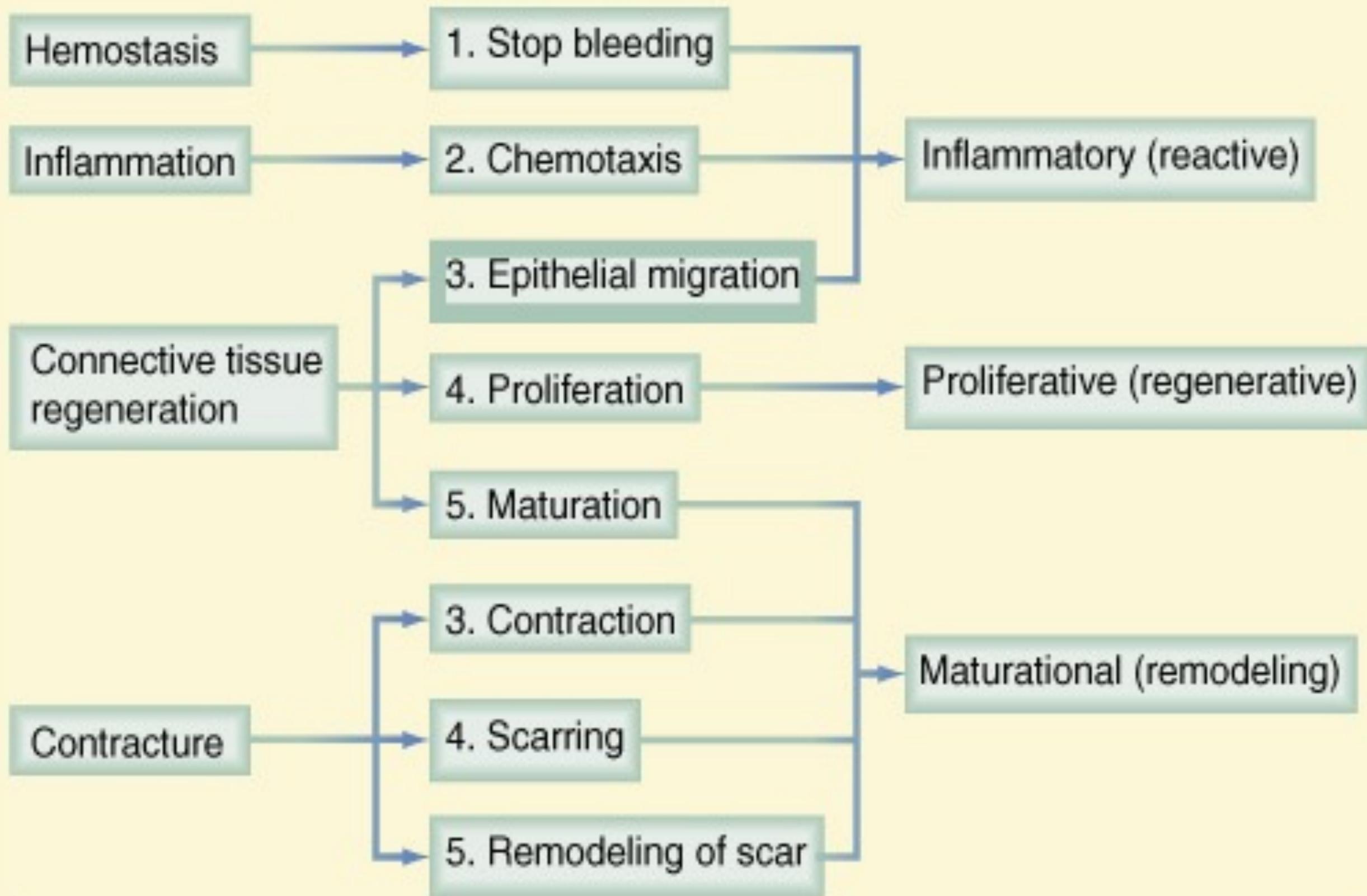


Repair vs Regeneration - Repair



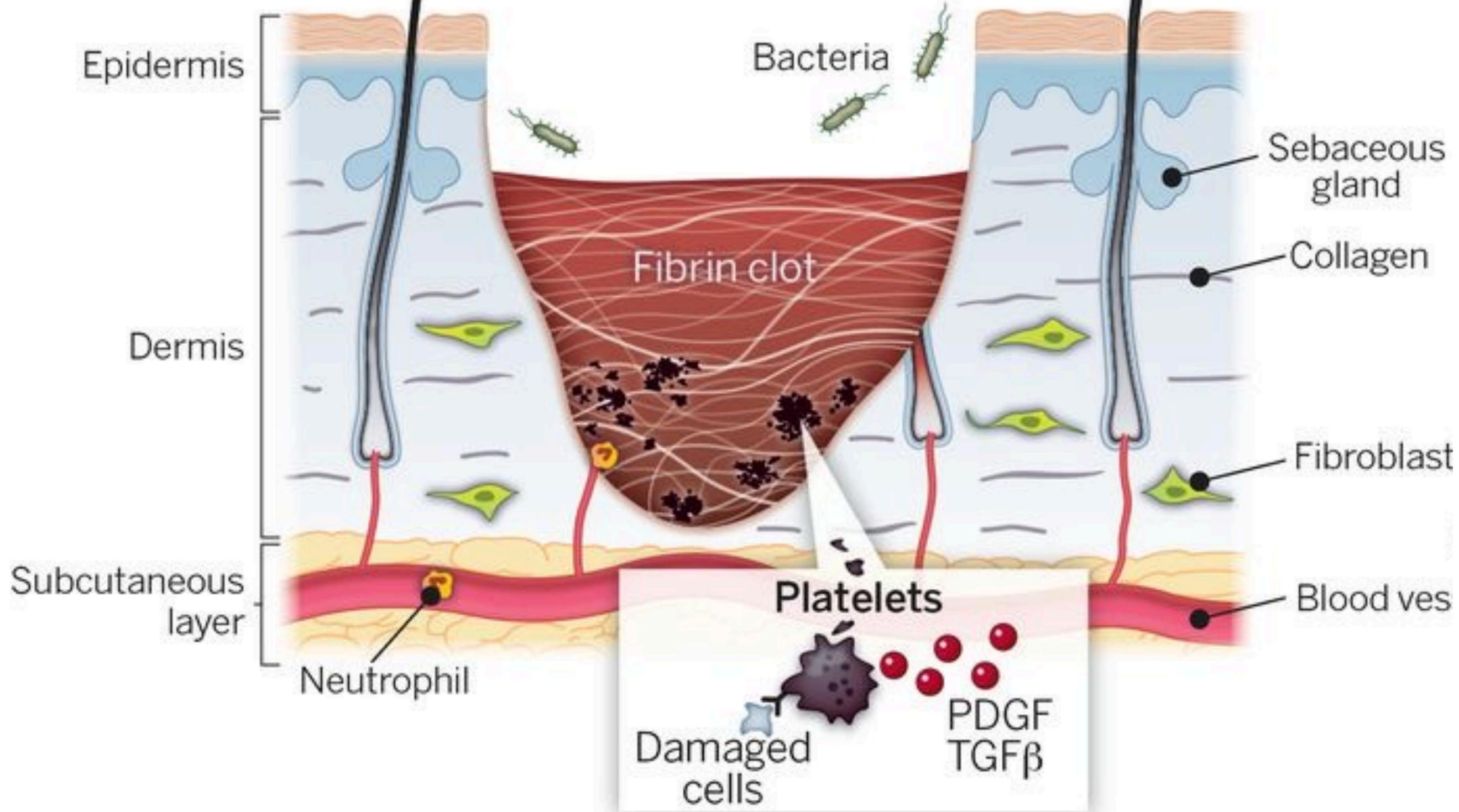
Phases of Wound Healing

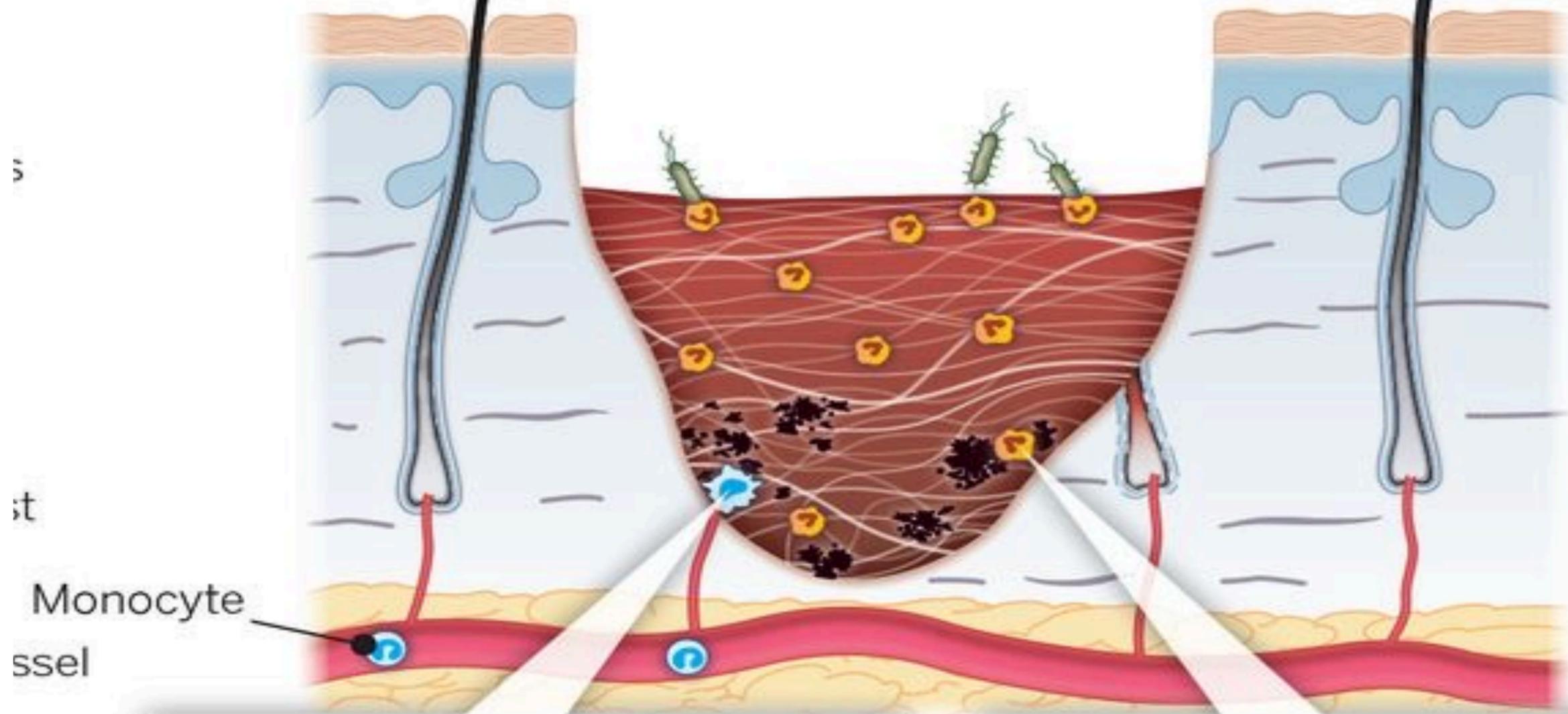
HEALING RESPONSES



A

Hemostasis



B**Inflammation**

Monocyte
vessel

Macrophage

CX3CL1
CCL2
PDGF



VEGF
EGF
IL-1
TNF α
TGF α

Neutrophil

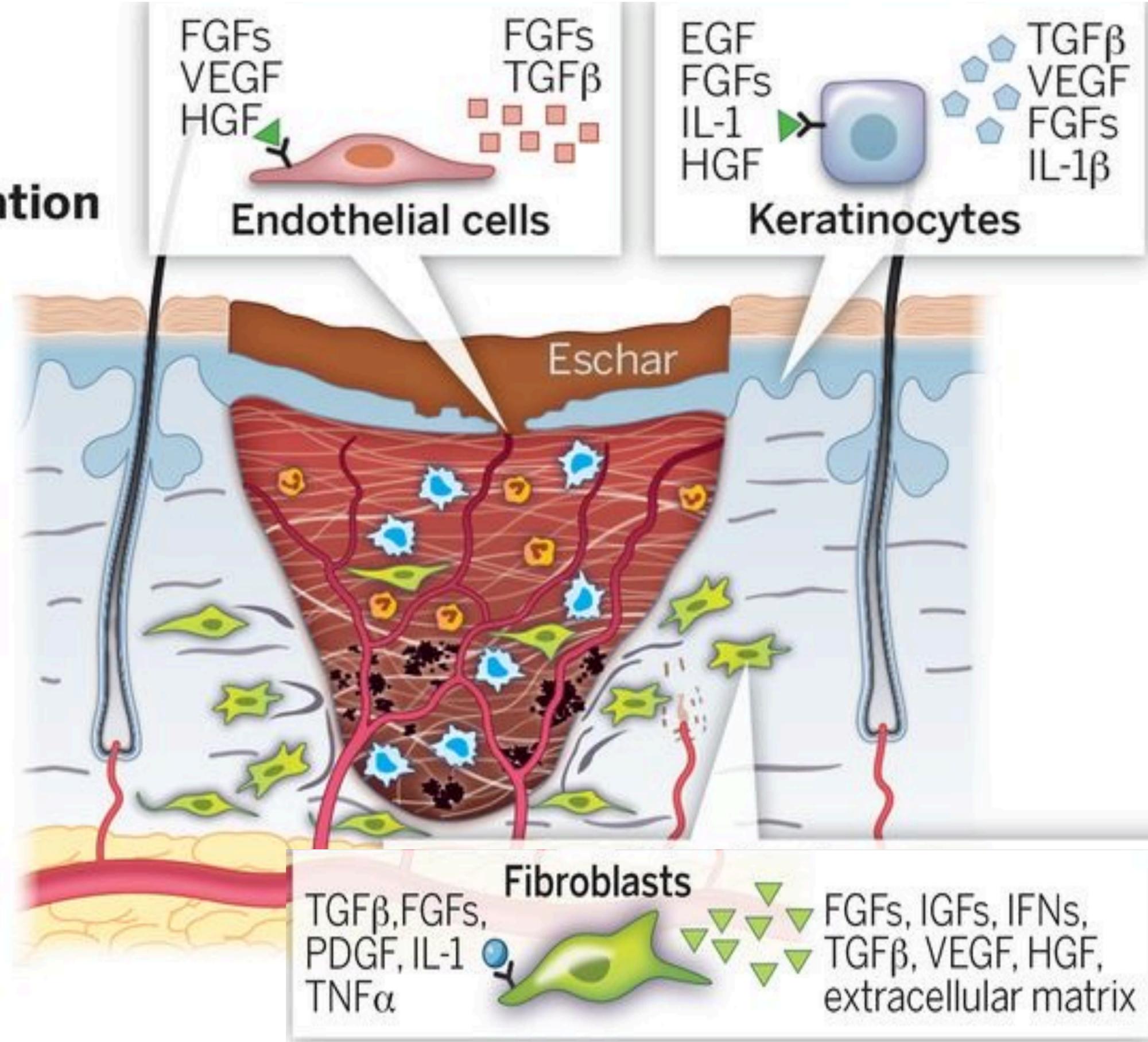
CXCL1
CXCL8
PDGF
TGF β



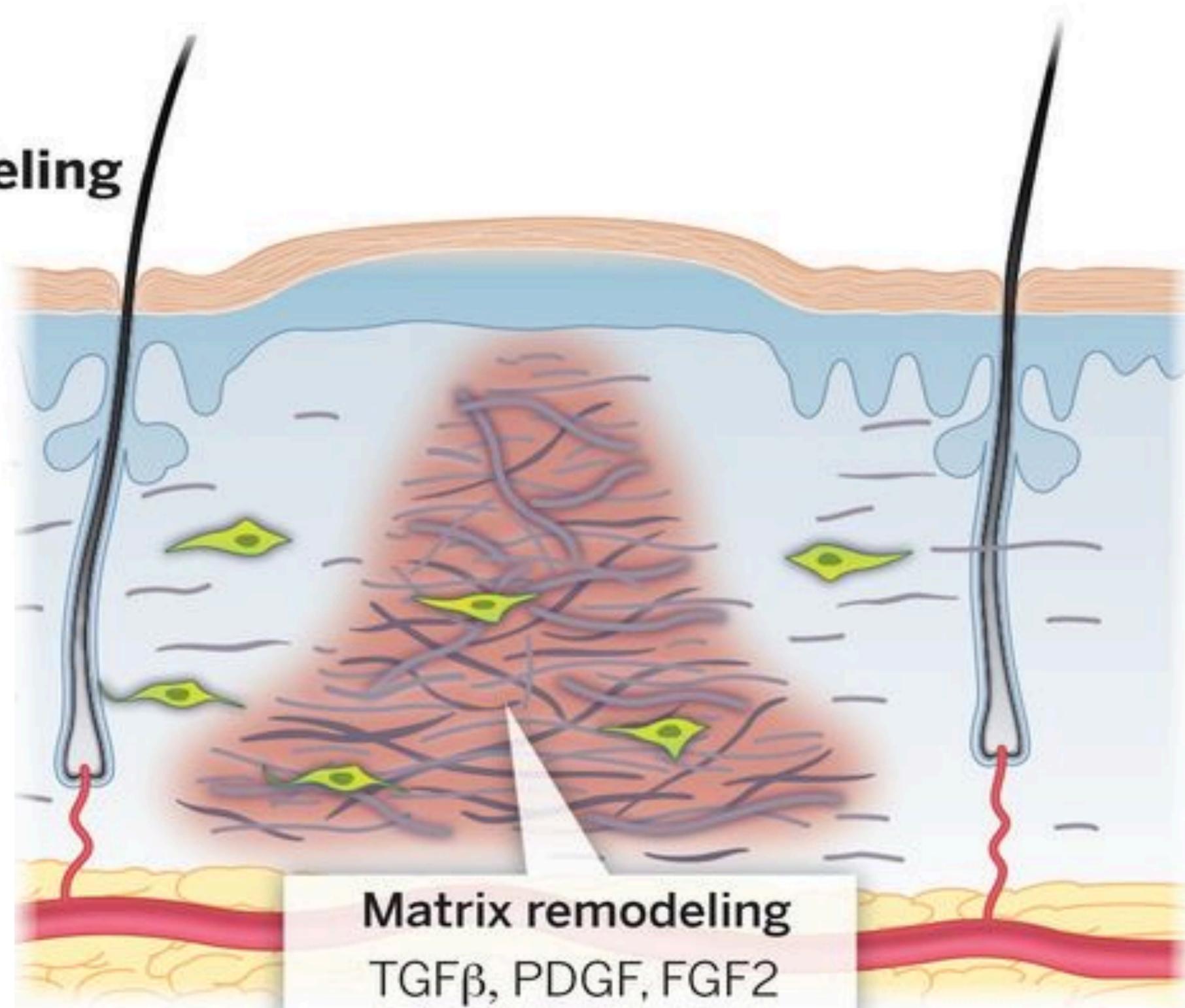
TGF β

C

Proliferation

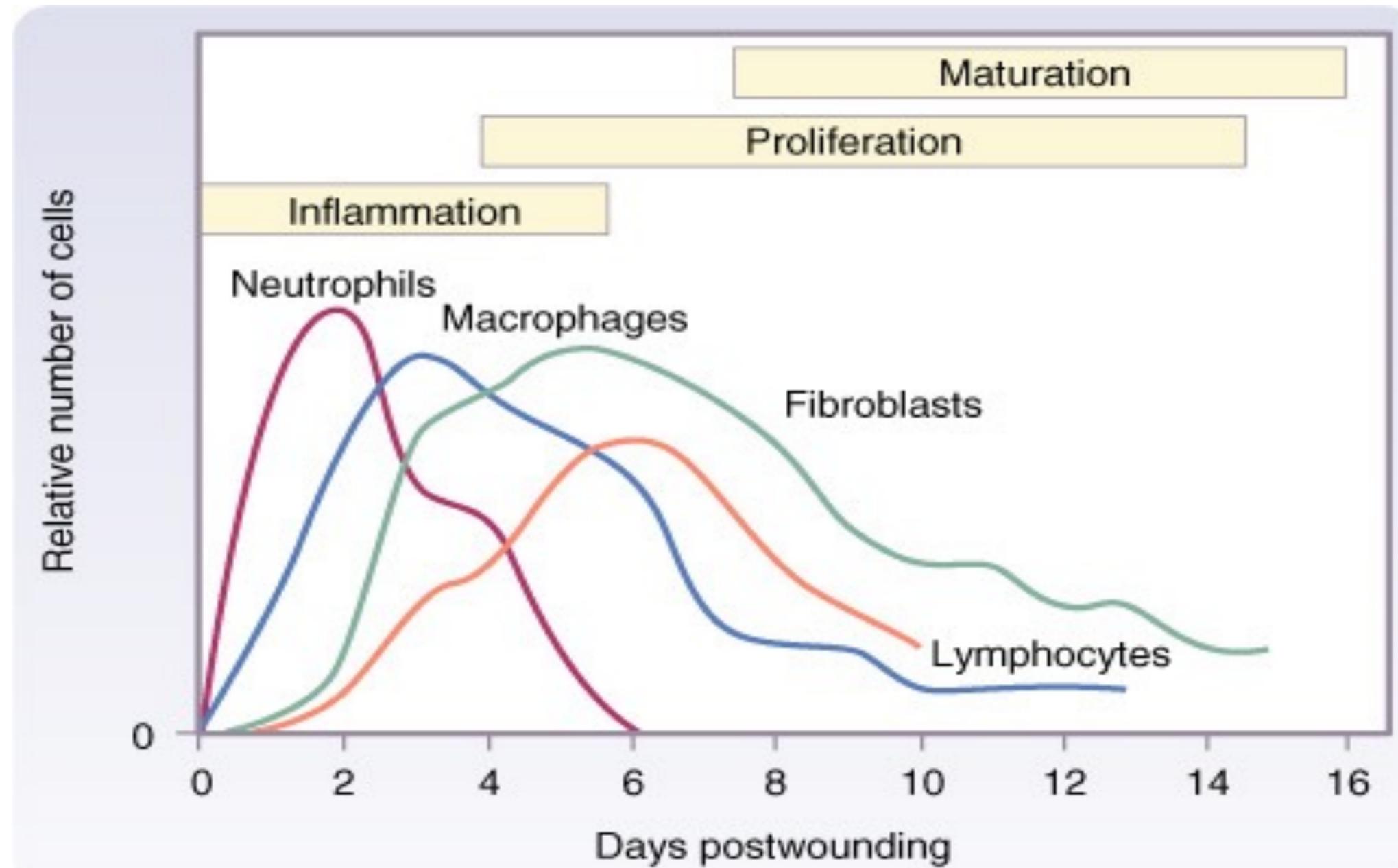


D
Remodeling



Matrix remodeling
TGF β , PDGF, FGF2
MMPs, TIMPs

WOUND HEALING is a highly orchestrated & a predictable process of physiologic events
Cells migrate into the wound site in an orderly fashion



TISSUE RESPONSES TO INJURY

Vascular events

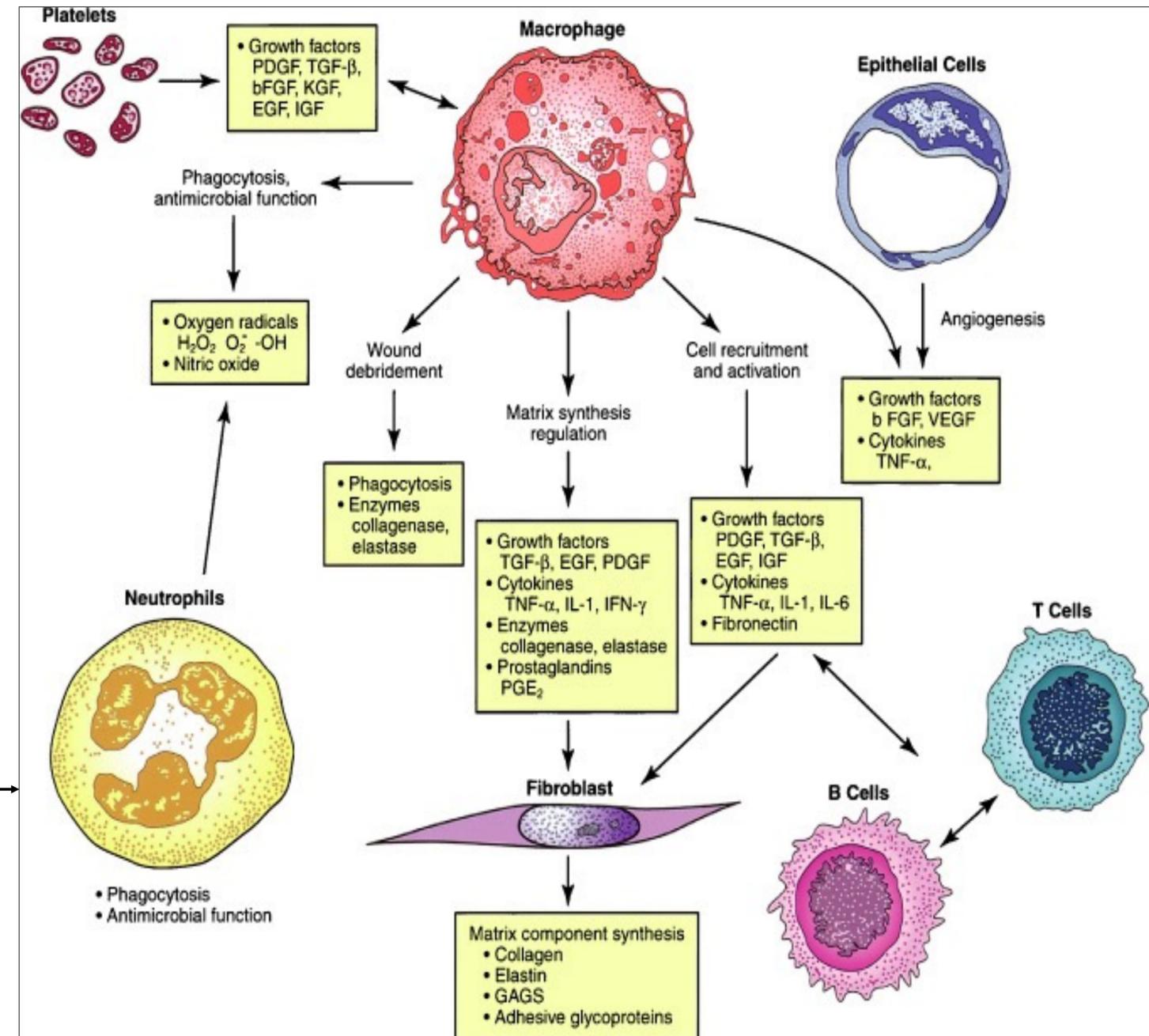
- Immediate transient vasoconstriction
- Active vasodilatation
- Permeability change

Cellular events

- Platelets
- Neutrophils
- Macrophages
- Lymphocytes
- Fibroblasts
- Endothelial cells

Chemical mediators and MMP

- Chemotactic factors
- Cytokines



CLASSIFICATION OF MMPs

24
Identified

Collagenases

- MMP1
- MMP8
- MMP13

Gelatinases

- MMP2
- MMP9

Stromelysins

- MMP3
- MMP10
- MMP11

Membrane-type MMPs

- MMP14
- MMP15
- MMP16
- MMP17
- MMP24
- MMP25

Matrilysin

- MMP7
- MMP26

Enamelysin

- MMP20

Other

- MMP19
- MMP21
- MMP23A
- MMP23B
- MMP27
- MMP28

Metalloelastase

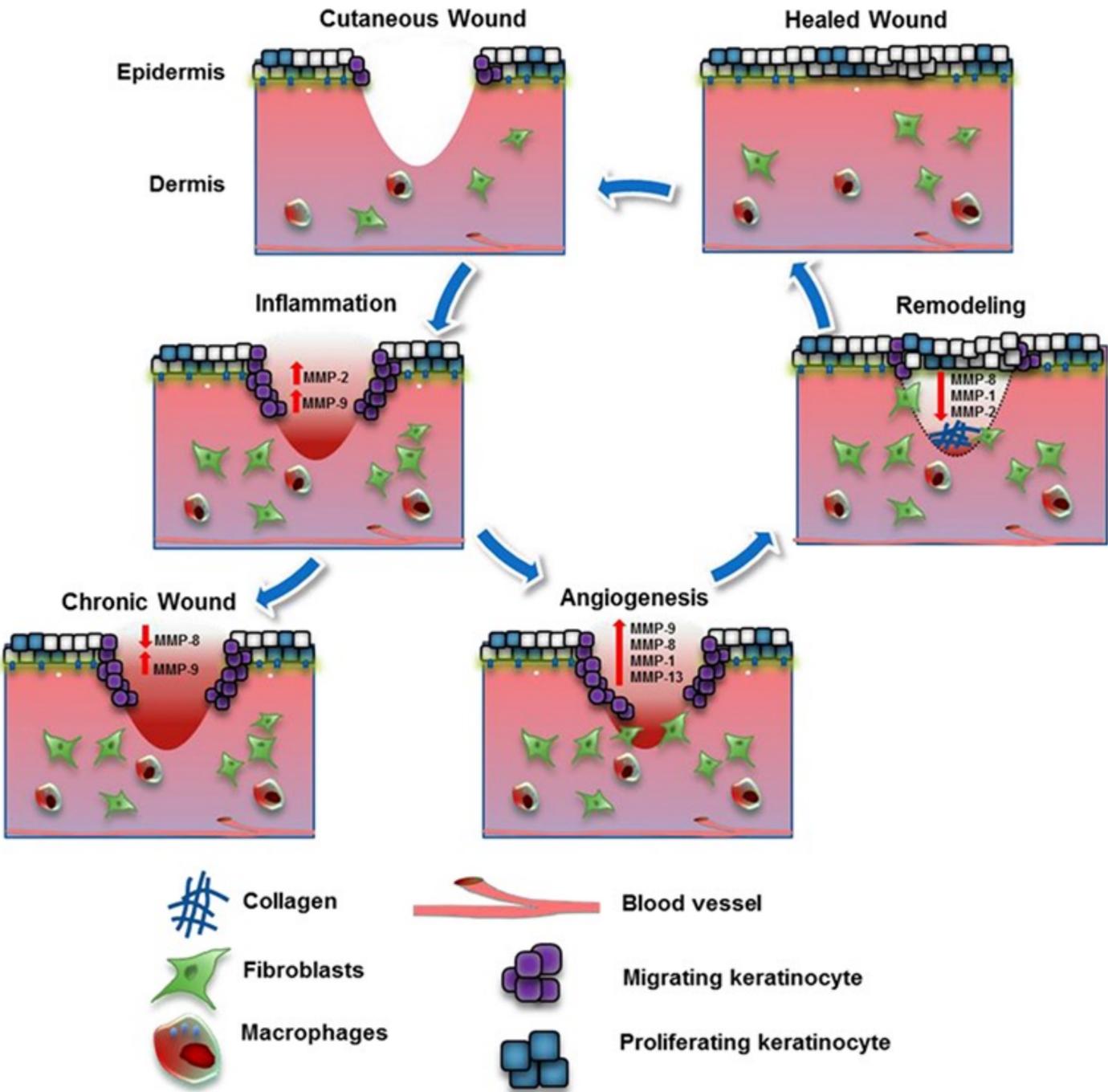
- MMP12

Inhibitors

- TIMP1
- TIMP2
- TIMP3
- TIMP4

Potential Inducers of Transcription

- BSG
- TCF20
- TNF



MATRIX METALLOPROTEINASES (MMPs)

Play a key role in wound healing



Proteinases are protein degrading enzymes that require calcium for conformation and zinc to be active

Act on a limited number of molecules (enzymes' substrates) & physically change them into other substances

MMP ROLE	HEALING PHASE
<ul style="list-style-type: none">• Removal of damaged ECM & bacteria	Inflammatory
<ul style="list-style-type: none">• Degradation of capillary basement membrane for angiogenesis• Migration of epidermal cells	Proliferative
<ul style="list-style-type: none">• Contraction of ECM scar• Remodeling of ECM scar	Remodeling

When Are MMPs a Problem?

- MMPs degrade proteins that are not their normal substrates when in a wound bed:
 - At too high a level
 - For too long a time
 - In the wrong places
- This can result in 'off target' destruction of proteins, such as:
 - Growth factors
 - Growth factor receptors
 - ECM proteins
- Damaging effects compounded by low levels of TIMP
- Multiple studies have shown that exudate of wounds that fail to heal contains more MMP than the exudate of healing wounds
- If indeed excess of MMP in chronic wounds is the cause not a result of protracted healing wound products that absorb or sequester MMP could help chronic wounds to heal.
- Silver containing dressing may effectively do that

MMPs (matrix metalloproteinases)

Level of MMPs in Wound Fluid



Key Points

Chronic wounds generally have:

- Elevated inflammatory markers
- High levels of proteases, including MMPs
- Diminished growth factor activity
- Reduced cell numbers in the wound

MECHANISMS IN WOUND HEALING

Epithelialization

- Epithelial cells detach from the basal layer
- Cells migrate towards the wound (contact guidance)
- Migrated cells are replaced by rapid mitosis
- Migration continues until epithelial cells come in contact on all sides with other epithelial cells (contact inhibition)

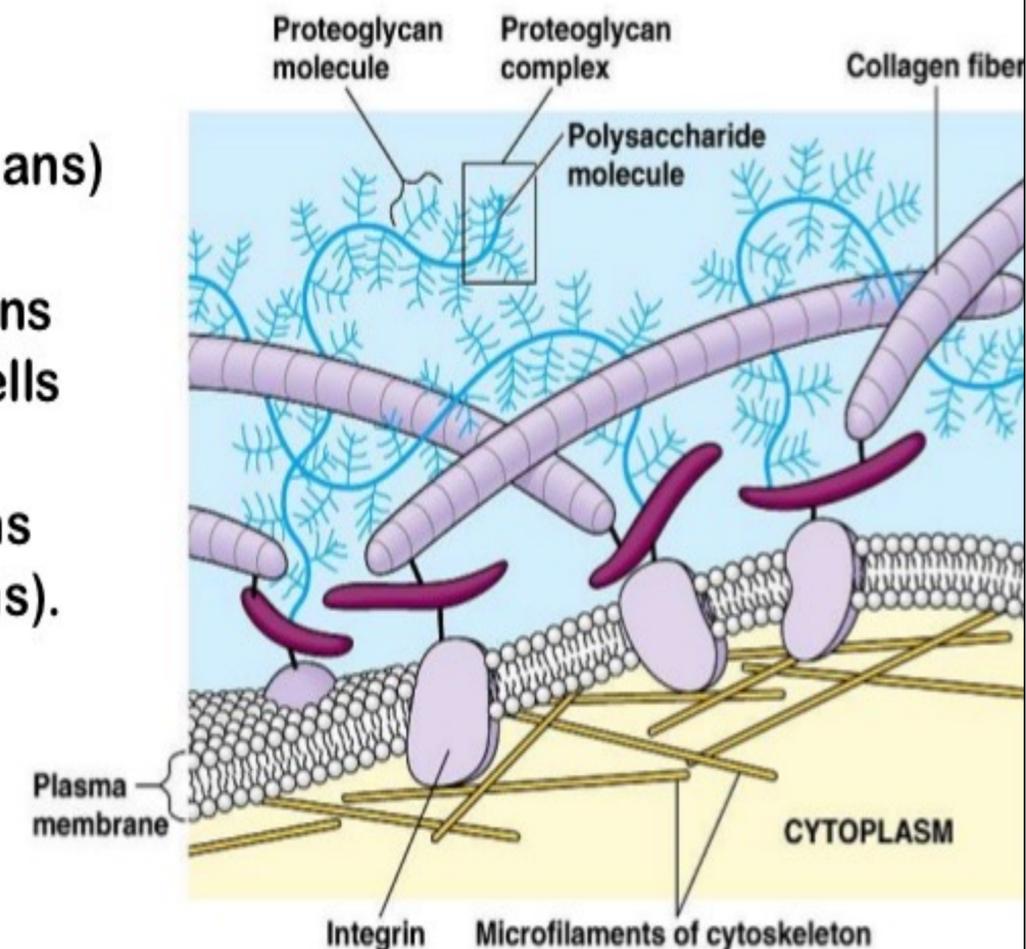
Contraction

- Inward movement of the edges of the injured tissue
- Begins between days 8 and 10 after injury
- Fibroblast and extracellular matrix control the process.

Connective tissue matrix deposition

The ECM contains 3 classes of molecules:

- structural proteins (collagens and elastins)
- protein-polysaccharide complexes to embed the structural proteins (proteoglycans)
- adhesive glycoproteins to attach cells to matrix (fibronectins and laminins).

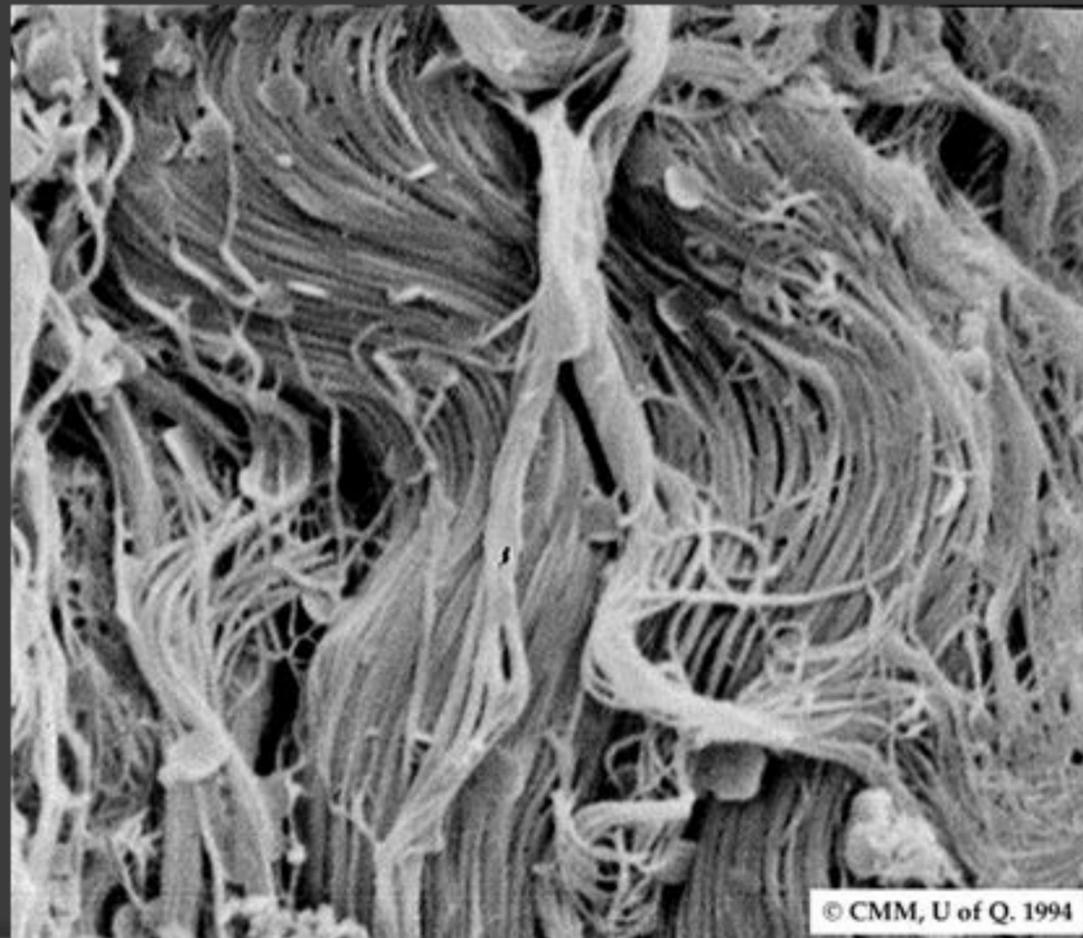


Collagen

Type III predominant collagen synthesis **days 1-2**

Type I days **3-4**

Type III replaced by **Type I** in **3 weeks**



I (80% skin)	Most Common: skin, bone, tendon. Primary type in wound healing.
II	Cartilage
III (20 % skin)	Increased Ratio in healing wound, also blood vessels and skin
IV	Basement Membrane
V	Widespread, particularly in the cornea

FACTORS AFFECTING WOUND HEALING

- Age
- Medications
- Host disease factors
- Technical factors

Host disease factors

- Nutrition
- Infection
- Wound hypoxia
- Diabetes
- Jaundice
- Uremia
- Malignancy
- Irradiation
- Denervation

Nutrition

- Protein
- Vitamin C – decrease in rate and quality of collagen production
- Vitamin K
- Minerals – zinc, copper

Infection

- **Bacteria**

 - prolong the inflammatory phase

 - interfere with epithelization, contraction and

 - collagen deposition

- **Endotoxin**

 - collagen degradation and destruction of

 - surrounding previously normal tissue

Diabetes mellitus

- **Susceptible to infection**
 - attenuated inflammatory response
 - impaired chemotaxis
 - inefficient bacterial killing
- **Impaired lymphocyte and leukocyte function**
- **Increased collagen degradation and decreased deposition**

Wound hypoxia

- Oxygen – necessary for normal metabolic cellular function
- Tissue oxygen level 35mmHg
- Impaired bacterial killing
- Impaired Fibroblasts collagen and ECM production
- Impaired epithelization

Causes of wound ischemia

- Poor arterial flow - atherosclerosis
- Poor venous flow – venous stasis
- Smoking
- Radiation
- Edema
- Diabetes mellitus
- Vasculitis
- Pressure

Most common types of wounds in primary care

- Vascular
- Diabetic
- Pressure
- Atypical

Vascular

Venous

- Venous Leg Ulcers (VLUs) account for 70-80% of lower extremity wounds
- Venous ulcers are open skin lesions that occur in an area affected by venous hypertension. The prevalence of venous ulcers in the United States ranges from 1% to 3%. In the United States, 10% to 35% of adults have chronic venous insufficiency, and 4% of adults 65 years or older have venous ulcers.
- Chronic venous insufficiency is the most common cause of lower extremity ulceration, accounting for up to 80 percent of the approximately 2.5 million leg ulcer cases in the United States. Annual costs in the United States for the treatment of venous ulcers are estimated at more than \$2 billion from costs related to frequent physician visits, care provided by nurses, compression therapy and wound care products, and, potentially, hospitalization

Arterial

In a report from the National Health and Nutrition Examination Survey (NHANES) from the United States, in which PAD was defined as an ankle-brachial index ABI <0.9 in either leg, the prevalence of PAD among adults aged 40 years and over in the US was 4.3 percent, corresponding to approximately 5 million individuals

- **Venous Ulcers: Diagnosis and Treatment**

SUSAN BONKEMEYER MILLAN, MD; RUN GAN, MD; and PETRA E. TOWNSEND, MD, University of Florida Health Wound Care and Hyperbaric Center and the University of Florida College of Medicine, Gainesville, Florida
Am Fam Physician. 2019 Sep 1;100(5):298-305.

Prevalence of and risk factors for peripheral arterial disease in the United States: results from the National Health and Nutrition Examination Survey, 1999-2000. Selvin E, Erlinger TP *Circulation.* 2004;110(6):738. Epub 2004 Jul 19.

- Protocol for the successful treatment of venous ulcers. Brem H, Kirsner RS, Falanga V *Am J Surg.* 2004;188(1A Suppl):1.

Vascular Ulcers

Venous Ulcers

- The first step in the pathophysiology is the development of Venous hypertension
- The venous pressure in the foot of a person in a standing position is proportional to the height of a column of blood from the foot to the right side of the Heart.
- This ranges from 60 to 90 mm Hg.
- With ambulation, muscles in the feet and legs work to expel venous blood from the extremity while venous valves prevent retrograde flow. This thereby works to lower ambulatory pressure.
- As the veins become dilated and venous valves failed to effectively cusp come up the calf pump is unable to effectively move venous blood an ambulatory hypertension results
- Causes
 - Inadequate muscle pump function -stroke, immobility, muscular dystrophy etc
 - Incompetent venous valves (reflux),
 - venous thrombosis, or
 - nonthrombotic venous obstruction
- Initiates a sequence of anatomic, physiologic, and histologic changes leading to vein dilation, skin changes, and/or skin ulceration

Vascular Ulcers

Venous Ulcers Risk factors

- Age 55 years or older
- family history of chronic venous insufficiency
- higher body mass index
- history of pulmonary embolism or superficial/deep venous thrombosis
- lower extremity skeletal or joint disease
- higher number of pregnancies
- parental history of ankle ulcers
- physical inactivity
- history of ulcers
- severe lipodermatosclerosis
- venous reflux in deep veins

Edema

Major causes of edema by primary mechanism

Increased capillary hydraulic pressure

Increased plasma volume due to renal sodium retention

Heart failure, including cor pulmonale

Primary renal sodium retention

- Renal disease, including the nephrotic syndrome

- Drugs:* Nonsteroidal antiinflammatory drugs (NSAIDs), glucocorticoids, fludrocortisone, thiazolidinediones (glitazones), insulins, estrogens, progestins, androgens, testosterone, aromatase inhibitors, tamoxifen; and by multiple mechanisms: vasodilators (hydralazine, minoxidil, diazoxide) and calcium channel blockers (particularly dihydropyridines [ie, amlodipine, nifedipine]); also refer to "Arteriolar vasodilation" below

- Refeeding edema

- Early hepatic cirrhosis

Pregnancy and premenstrual edema

Idiopathic edema, when diuretic induced

Sodium or fluid overload: Parenteral antibiotics or other drugs with large amounts of sodium, sodium bicarbonate, or excessive or overly rapid fluid replacement

Venous obstruction or insufficiency

Cirrhosis or hepatic venous obstruction

Acute pulmonary edema

Local venous obstruction

- Venous thrombosis

- Venous stenosis

Chronic venous insufficiency - Post-thrombotic syndrome

Arteriolar vasodilation

Drugs:* Frequent - Vasodilators (hydralazine, minoxidil, diazoxide), dihydropyridine calcium channel blockers; less frequent - alpha1 blockers, sympatholytics (ie, methyl dopa), nondihydropyridine calcium channel blockers^[1]

Idiopathic edema

Hypoalbuminemia

Protein loss

Nephrotic syndrome

Protein-losing enteropathy

Reduced albumin synthesis

Liver disease

Malnutrition

Increased capillary permeability

Idiopathic edema

Burns

Trauma

Inflammation or sepsis

Allergic reactions, including certain forms of angioedema

Acute respiratory distress syndrome

Diabetes mellitus

Interleukin 2 therapy

Malignant ascites

Lymphatic obstruction or increased interstitial oncotic pressure

Lymph node dissection

Nodal enlargement due to malignancy

Hypothyroidism

Malignant ascites

Other drugs* (uncertain mechanism)

Anticonvulsant: Gabapentin, pregabalin

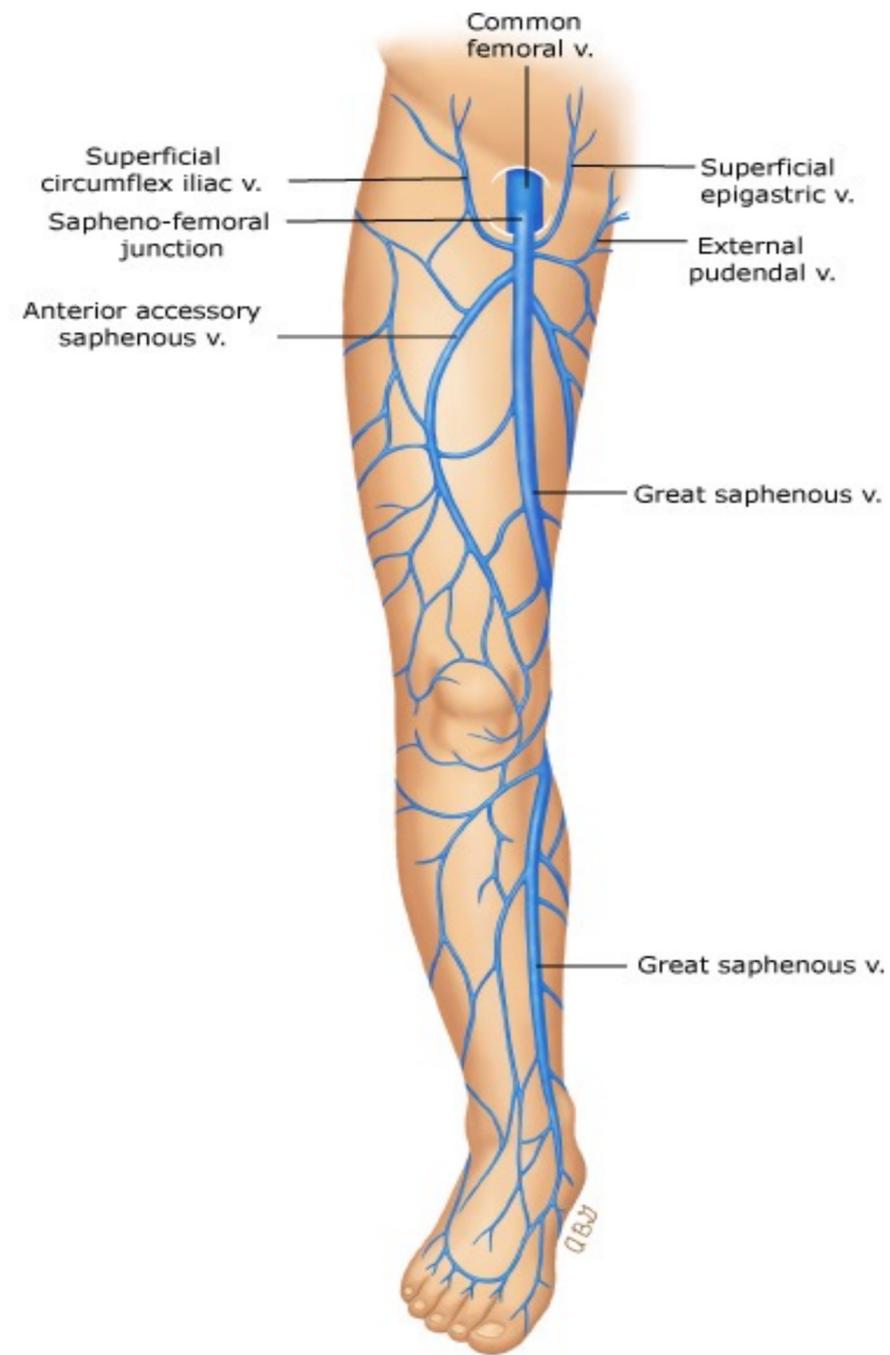
Antineoplastic: Docetaxel, cisplatin

Antiparkinson: Pramipexole, ropinirole

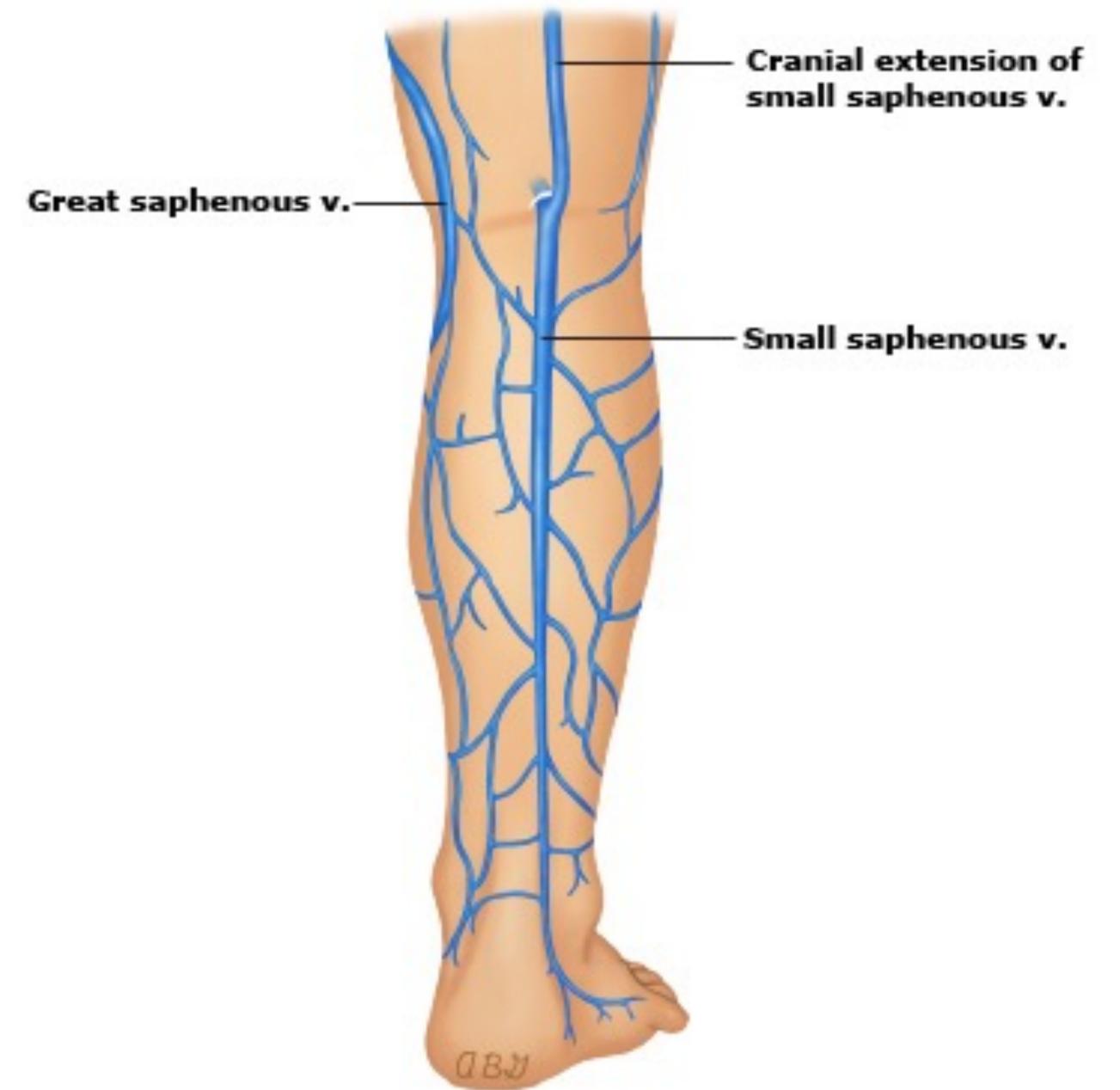
* Patients with decreased cardiac output, preexisting renal insufficiency, and/or receiving higher doses are more likely to experience edema and edema-associated adverse events. This is not a complete list of drugs associated with edema. For additional information, refer to the Lexicomp individual drug monographs included with UpToDate.

1.Reference:Messerli FH. Vasodilatory edema: A common side effect of antihypertensive therapy. Curr Cardiol Rep 2002; 4(6):479.

Superficial veins of lower extremity anterior view



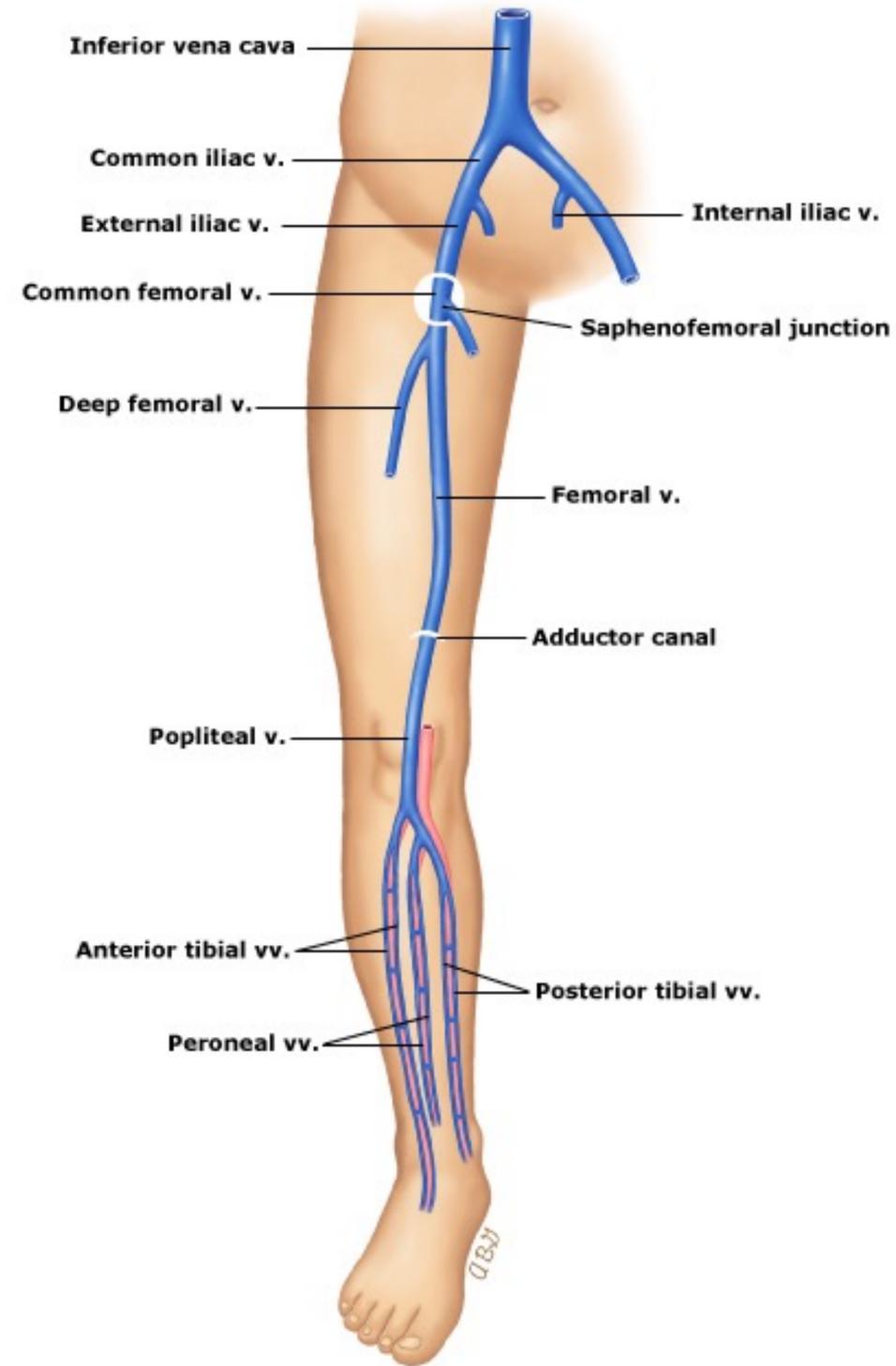
Superficial veins of lower leg, posterior view



The figure shows the superficial veins of the anterior lower extremity. Many of these veins are tributaries to the great saphenous vein, which drains into the main deep vein of the lower extremity, the common femoral vein.

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Deep veins of the lower extremity



Deep veins of the right lower extremity. The paired tibial veins (anterior tibial, peroneal, and posterior tibial) are shown with their adjacent arteries. The bridging veins between the paired veins are also demonstrated. The popliteal and femoral veins are also sometimes duplicated (omitted from the diagram) with one of the duplicated segments frequently larger in caliber than the other.

Revised CEAP Classification

Clinical classification	
C ₀	No visible or palpable signs of venous disease
C ₁	Telangiectasias, reticular veins
C ₂	Varicose veins
C _{2r}	Recurrent varicose veins
C ₃	Edema
C ₄	Changes in skin and subcutaneous tissue secondary to chronic venous disease
C _{4a}	Pigmentation or eczema
C _{4b}	Lipodermatosclerosis or atrophie blanche
C _{4c}	Corona phlebectatica
C ₅	Healed
C ₆	Active venous ulcer
C _{6r}	Recurrent active venous ulcer
S	With symptoms attributable to venous disease
A	Absence of symptoms attributable to venous disease

Etiology classification	
E _p	Primary
E _s	Secondary
E _{si}	Secondary (intravenous)
E _{se}	Secondary (extravenous)
E _c	Congenital
E _n	No cause identified
Anatomy classification*	
A _s	Superficial veins (Tel, Ret, GSV _a , GSV _b , SSV, AASV, NSV)
A _d	Deep veins (IVC, CIV, IIV, EIV, PELV, CFV, DFV, FV, POPV, TIBV, PRV, ATV, PTV, MUSV, GAV, SOV)
A _p	Perforator veins (TPV, CPV)
A _n	No venous anatomic location identified
Pathophysiology classification*	
P _r	Reflux
P _o	Obstruction
P _{r,o}	Reflux and obstruction
P _n	No venous pathophysiology identifiable

Limbs in higher categories have more severe signs of chronic venous disease and may have some or all of the findings defining a less severe clinical category. Each limb is further characterized as asymptomatic (C₀₋₆, A) or symptomatic (C₀₋₆, S).

Symptoms that may be associated with telangiectatic, reticular, or varicose veins include lower extremity aching, pain, and skin irritation. Therapy may alter the clinical category of chronic venous disease. Limbs should therefore be reclassified after any form of medical or surgical treatment.

CEAP: Clinical-Etiology-Anatomy-Pathophysiology; Tel: telangiectasia; Ret: reticular veins; GSV_a: great saphenous vein above the knee; GSV_b: great saphenous vein below the knee; SSV: small saphenous vein; AASV: anterior accessory saphenous vein; NSV: nonsaphenous vein; IVC: inferior vena cava; CIV: common iliac vein; IIV: internal iliac vein; EIV: external iliac vein; PELV: pelvic veins; CFV: common femoral vein; DFV: deep femoral vein; FV: femoral vein; POPV: popliteal vein; TIBV: tibial vein; PRV: peroneal vein; ATV: anterior tibial vein; PTV: posterior tibial vein; MUSV: muscular vein; GAV: gastrocnemius vein; SOV: soleal vein; TPV: thigh perforator vein; CPV: calf perforator vein.

* Specific anatomic location(s) to be reported under each pathophysiologic (P) class to identify anatomic location(s) corresponding to P class.

From: Lurie F, Passman M, Meisner M, et al. The 2020 update of the CEAP classification system and reporting standards. *J Vasc Surg Venous Lymphat Disord* 2020; 8:342. Reproduced with permission from the Society for Vascular Surgery. Copyright © 2020 Society for Vascular Surgery. All rights reserved.

Corona Phlebectatica

- The corona phlebectatica (CP) is classically described as the presence of abnormally visible cutaneous blood vessels at the ankle with four components: "venous cups," blue and red telangiectases, and capillary "stasis spots."
- Recently added in the revised ceap classification



C4cEsApPr

Atrophie blanche

(atrophic, white scarring) in a patient
with a venous ulcer



[Am Fam Physician](#). 2019 Sep 1;100(5):298-305.

Venous Ulcers: Diagnosis and Treatment

[Susan Bonkemeyer Millan¹](#), [Run Gan²](#), [Petra E Townsend¹](#)

Telangiectasias are dilated intradermal venules less than one millimeter in diameter. Synonyms include spider veins, hyphen webs, and thread veins



Courtesy of Patrick C Alguire, MD.

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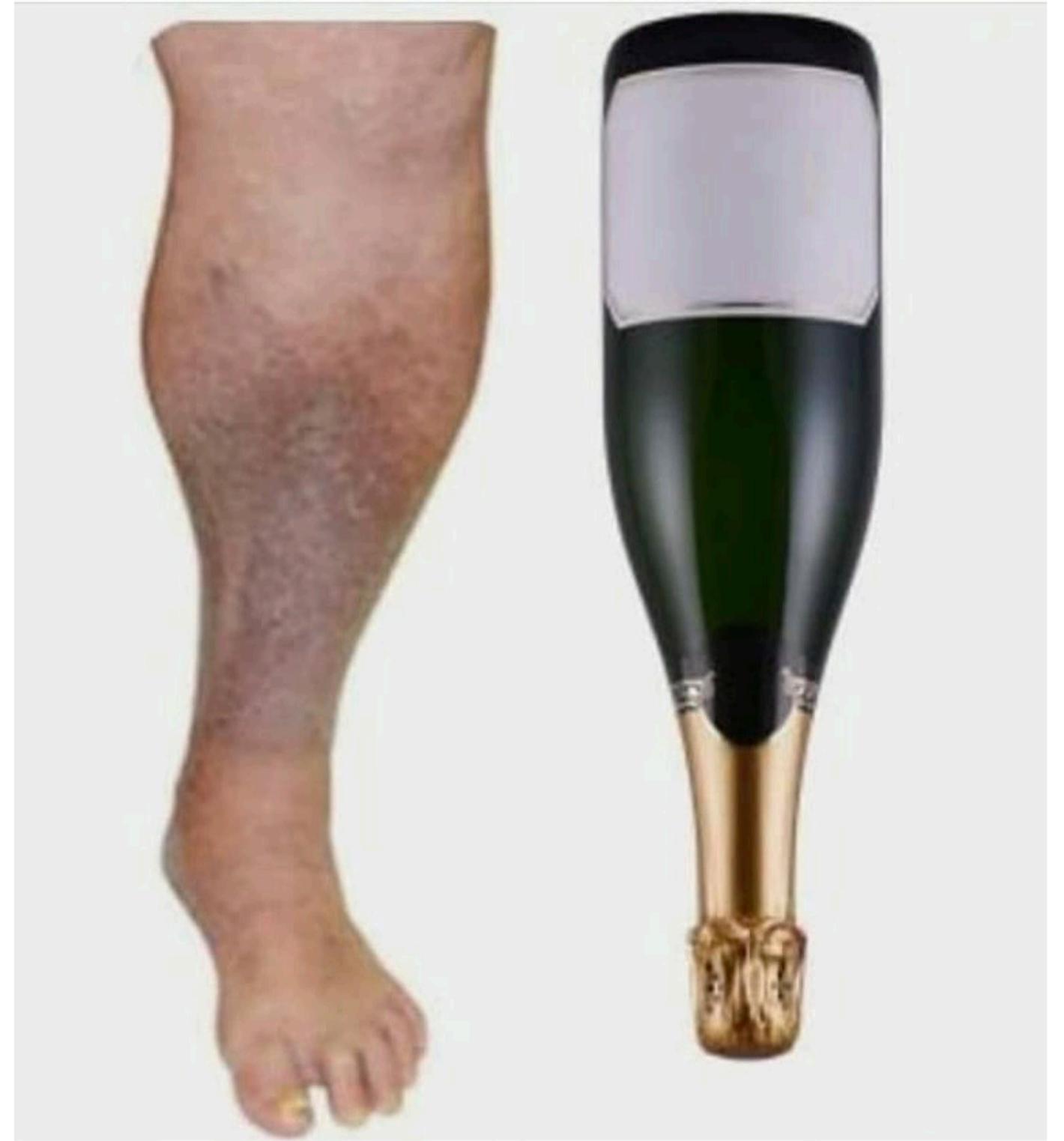
**Longstanding edema in this patient with chronic venous insufficiency led to moderately advanced pigment changes on the medial and lateral ankles, which extend onto the dorsum of the foot. The left medial ankle displays a healed venous ulcer below the malleolus; the right lateral ankle has a small active venous ulcer (arrow).
Courtesy of Patrick C Alguire, MD.
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Lipodermatosclerosis

- Inverted Champagne bottle sign
- Subcutaneous tissue becomes inflamed
- Tissue hypoxia → protein leakage into the interstitium → activation of leukocytes
- Recurrent ulcerations and necrosis of fat



Autoeczematous reaction

Several areas of autoeczematization on the leg and thigh of a patient with venous insufficiency and stasis dermatitis.



Courtesy of Patrick C Alguire, MD.

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**Large venous ulcer
on the medial ankle
in a patient with
chronic venous
hypertension. The
ulcer is shallow with
irregular borders
and has a
granulation tissue
base.**



Courtesy of Patrick C Alguire, MD.
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Venous leg ulcer. Wounds are generally irregular and shallow.

*Thomas Jefferson University Clinical Image Database.



Venous Leg Ulcers

Clinical Diagnosis

- Location- On the lower part of leg usually, rarely on the foot
-61% are on the medial malleolus
- Appearance- irregular, shallow, Hyperpigmentation of surrounding skin, edema (pitting or woody)
- Diagnostic Evaluation:
CBC (anemia), CMP (renal disease, protein), A1C, PT, INR (increased risk of bleeding in ulcer site), Venous duplex scan with ultrasound

Venous Leg Ulcers

Venous duplex ultrasound examination can confirm

- the presence of venous obstruction
- valvular incompetence
- used for planning venous ablation procedures but is not necessary in all cases of suspected venous insufficiency where intervention is not being considered.

Indications:

- If a clinical diagnosis of venous insufficiency or obstruction cannot be established but symptoms are strongly suggestive.
 - In patients with signs of chronic venous disease but whose symptoms are questionably related to the venous disease.
 - In atypical cases, such as an unusually early age of onset (<40 years) of symptoms, or following trauma.
 - In cases of ulceration. Patients with ulceration due to superficial venous reflux may benefit from venous ablation procedures. Significantly decreased ulcer recurrence rates have been found with removal or ablation of greater or small saphenous veins
 - In patients with clinically suspected venous disease who do not respond to standard conservative measures.
-
- Other noninvasive tests -air plethysmography and photoplethysmography
 - ABI if PAD suspected

Venous Leg Ulcers

Treatment

- Conservative management:
 - Compression
 - Choice of dressing
 - Medications- pentoxifylline (Evidence level A), zinc, horse chestnut seed extract (?), phlebotonics
 - Debridement
- Surgical options

- The goals of treatment are
 - primary- to heal the wound and
 - secondary-to reduce edema and prevent recurrence

Venous Leg Ulcers

Compression therapy

- Is the standard of care for venous ulcers and chronic venous insufficiency.
- A recent Cochrane review found that venous ulcers heal more quickly with compression therapy than without.
- Methods include inelastic, elastic, and intermittent pneumatic compression.
- Compression therapy reduces edema, improves venous reflux, enhances healing of ulcers, and reduces pain.
- Success rates range from 30 to 60 percent at 24 weeks, and 70 to 85 percent after one year.
- After an ulcer has healed, lifelong maintenance of compression therapy may reduce the risk of recurrence.
- However, adherence to the therapy may be limited by pain; drainage; application difficulty; and physical limitations, including obesity and contact dermatitis.
- Contraindications to compression therapy include clinically significant arterial disease and uncompensated heart failure

Venous Leg Ulcers

Compression therapy

Elastic:

Elastic compression bandages conform to the size and shape of the leg, accommodate to changes in leg circumference, provide sustained compression during rest and walking, have absorptive capacity, and require infrequent changes (about once a week). There is strong evidence for the use of multiple elastic layers vs. single layers to increase ulcer healing.

Inelastic

Inelastic compression wraps, most commonly zinc oxide–impregnated Unna boots, provide high compression only during ambulation and muscle contraction. They should not be used in nonambulatory patients or in those with arterial compromise. Unna boots have limited capacity for fluid absorption in patients with highly exudative ulcers and are best used for early, small, dry ulcers and for venous dermatitis because of the skin soothing effects of zinc oxide.

- **Venous Ulcers: Diagnosis and Treatment**

SUSAN BONKEMEYER MILLAN, MD; RUN GAN, MD; and PETRA E. TOWNSEND, MD, University of Florida Health Wound Care and Hyperbaric Center and the University of Florida College of Medicine, Gainesville, Florida

Am Fam Physician. 2019 Sep 1;100(5):298-305.

- Dolibog P, Franek A, Taradaj J, et al. A comparative clinical study on five types of compression therapy in patients with venous leg ulcers. *Int J Med Sci*. 2013;11(1):34–43

Venous stasis ulcers

Compression therapy

Classification of compression stockings

- Class I - 20-30 mmHg
- Class II- 30-40 mmHg
- Class III- 40-50 mmHg
- Class IV- >50 mmHg

Intermittent pneumatic compression pumps

Indications

Lymphedema

Contraindications

PAD

Advanced heart failure

Venous stasis ulcers

Compression therapy

Types of Compression

Multilayer – 4 layer, 3 layer, unna's boot

High Compression- 3 or 4 layer, unna's boot, regular stretch ACE

Elastic- 3 or 4 layer, stockings

Inelastic- Unna's boot, Short stretch ACE

Venous stasis ulcers

Ankle Brachial Index

ABI (Determines the degree of compression)

>0.9- 1.2 Normal

0.7- 0.8- Adequate

0.5- 0.6- Reduced

<0.5 Marked Arterial disease

>1.2 Calcified vessels

- Recheck ABI every 3 months for patients with non-healing lower extremity ulcers
- Consider TCOM (Transcutaneous Oxygen Measurement), in patients with calcified blood vessels

Venous stasis ulcers

Compression therapy

Jobst zippered
stocking



Circaid



Venous stasis ulcers

Compression therapy



This tool is intended as a guideline only and should be used in conjunction with existing clinical protocols that consider physiological state and wound and skin condition.

Sizing and Measuring Guide

Circumference measurements required to provide pressures of between 10-15 mm Hg

Art No.	Size	Product Width	Limb Size (cm)
1436	B	6.0 - 6.4	13 - 16
1443	C	6.6 - 7.2	16 - 20
1437	D	7.3 - 7.9	20 - 24
1434	E	8.7 - 9.2	24 - 28
1438	F	9.7 - 10.3	28 - 36
1439	G	11.3 - 12.7	36 - 46
1440	J	17.4 - 19.3	46 - 60

Measure around the widest part of the area
ALWAYS USE AS A DOUBLE LAYER

Directions for use

Warning: Contains natural rubber latex.



1. Cut Tubigrip to twice length required for limb, allowing an extra 2-3 cm for overlap.



2. Pull Tubigrip onto limb like a stocking.



3. Double Tubigrip back over limb. Ensure upper edge is taken 2-3 cm higher up the limb than the first.



Venous Ulcers

Surgical Options

No axial vein reflux — sclerotherapy or excision (phlebectomy)

With superficial venous reflux — superficial venous ablation

With deep venous reflux — For selected patients, endovenous treatment of central venous obstruction (eg, angioplasty/stenting) improves venous outflow and reduces symptoms.

- Patients with primary deep vein valvular incompetence can usually be treated with vein valve repair. It is worth noting that common femoral venous reflux is abolished in most patients who undergo great saphenous venous ablation.
- Patients with secondary deep vein valvular incompetence (ie, following prior deep venous thrombosis) require more extensive transplantation or transposition procedures.

In general, better outcomes are achieved in patients with primary valvular incompetence compared with secondary incompetence with 10 year cumulative clinical success rates of 73 and 43 percent, respectively

With perforator reflux — Symptomatic patients whose physical examination and duplex study are consistent with perforator reflux as a source of venous ulceration may be candidates for perforator ablation with ultrasound-guided sclerotherapy or endovenous methods

Venous stasis ulcers

Venous clinical severity score — The venous clinical severity score (VCSS) is a disease-specific instrument that is complementary to the CEAP classification. It has both physician-determined and patient-reported elements. Ten clinical parameters (pain, varicose veins, venous edema, pigmentation, inflammation, induration, number of active ulcers, duration of active ulcers, size of active ulcers, and compliance with compression therapy) are graded from zero to three depending upon severity (None = 0, Mild = 1, Moderate = 2, Severe = 3)

Venous disability score

Venous segmental disease score (VSDS)

Villalta scale

Aberdeen varicose vein questionnaire

Other QOL scores

Lymphedema

Primary

Secondary

Stages

Subclinical

Stage 1

Stage 2

Stage 3

Vascular Ulcers- Arterial

Vascular

Venous

Arterial

- Venous Leg Ulcers (VLUs) account for 70-80% of lower extremity wounds
- Venous ulcers are open skin lesions that occur in an area affected by venous hypertension. The prevalence of venous ulcers in the United States ranges from 1% to 3%. In the United States, 10% to 35% of adults have chronic venous insufficiency, and 4% of adults 65 years or older have venous ulcers.
- Chronic venous insufficiency is the most common cause of lower extremity ulceration, accounting for up to 80 percent of the approximately 2.5 million leg ulcer cases in the United States. Annual costs in the United States for the treatment of venous ulcers are estimated at more than \$2 billion from costs related to frequent physician visits, care provided by nurses, compression therapy and wound care products, and, potentially, hospitalization

In a report from the National Health and Nutrition Examination Survey (NHANES) from the United States, in which PAD was defined as an ankle-brachial index ABI <0.9 in either leg, the prevalence of PAD among adults aged 40 years and over in the US was 4.3 percent, corresponding to approximately 5 million individuals

- **Venous Ulcers: Diagnosis and Treatment**

SUSAN BONKEMEYER MILLAN, MD; RUN GAN, MD; and PETRA E. TOWNSEND, MD, University of Florida Health Wound Care and Hyperbaric Center and the University of Florida College of Medicine, Gainesville, Florida
Am Fam Physician. 2019 Sep 1;100(5):298-305.

Prevalence of and risk factors for peripheral arterial disease in the United States: results from the National Health and Nutrition Examination Survey, 1999-2000. Selvin E, Erlinger TP *Circulation.* 2004;110(6):738. Epub 2004 Jul 19.

- Protocol for the successful treatment of venous ulcers. Brem H, Kirsner RS, Falanga V *Am J Surg.* 2004;188(1A Suppl):1.

Vascular Ulcers

Ischemic/Arterial Ulcers

Risk Groups

The American College of Cardiology/American Heart Association (ACC/AHA) guidelines on PAD

- Age ≥ 70 years
- Age 50 to 69 years with a history of smoking or diabetes
- Age 40 to 49 with diabetes and at least one other risk factor for atherosclerosis
- Known atherosclerosis at other sites (eg, coronary, carotid, renal artery disease)

Vascular ulcers- arterial/ischemic

PAD risk factors

Smoking number one risk factor

Diabetes

Hypertension

Hyperhomocysteinemia

Hypercholesterolemia

Obesity

HIV

Hypothyroidism

Chronic renal insufficiency

Family history of cardiovascular disease

African American ethnicity

The age groups mentioned in the previous slide

Vascular ulcers- arterial/ischemic

Clinical diagnosis

- Located between toes, tips of toes, lateral malleolus, or where there is trauma and or friction from walking
- Deep punched out hole in appearance
- Poor granulation tissue
- Well defined edges
- Extremely painful, worse at night, improves with dangling
- Dry gangrene versus wet gangrene
- History of any risk factors and physical exam

Vascular ulcer - Arterial

Ischemic wound Lateral foot



The picture depicts the clinical appearance of an ischemic wound on the lateral forefoot, specifically at the plantar aspect of the 5th metatarsal-phalangeal joint. Note the wound appearance of fibrotic tissue mixed with eschar in an area of increased pressure near a bony prominence.

Graphic 101202 Version 2.0

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Blue toe syndrome



Blue toes are a classic manifestation of peripheral embolization of atheromatous material from proximal arterial sources (eg, aorta); the pedal pulses are often normal. This patient, who has a 30-year history of type 1 diabetes and severe peripheral vascular disease, presented with foot pain and discoloration. Cholesterol microemboli from the aorta were suspected to be the cause.

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Graphic 58761 Version 2.0

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Vascular ulcers- arterial/ischemic

Diagnostic Evaluation

- All patients with lower extremity ulcers should be assessed for arterial disease
- Decreased or absent palpable pedal pulses (presence of pulses does not rule out lower extremity arterial disease – LEAD)
- Delayed capillary refill response (Tcom or TcPO₂ is a better test)
- 10 to 15 second delay in return of color when raising the leg 45 degrees for one minute, Dependent rubor (Buerger's Test)
- Assess functional ability (walking aids ,neuropathy)
- Assess for complications (Cellulitis ,gangrene,osteomyelitis)
- Look for contributing factors other than atherosclerosis involving the arterial system (thromboangiitis, vasculitis, reynaud's ,pyoderma gangrenosum, thalassemia, sickle cell disease)

Vascular ulcers- Arterial/ischemic

Diagnostic evaluation

ABI-ankle brachial index

TBI –toe brachial index

TCP O₂ or TCOM-transcutaneous oxygen measurement

Others- culture- biopsy, swab, A1C ,homocysteine levels, lipids

Vascular ulcer- arterial

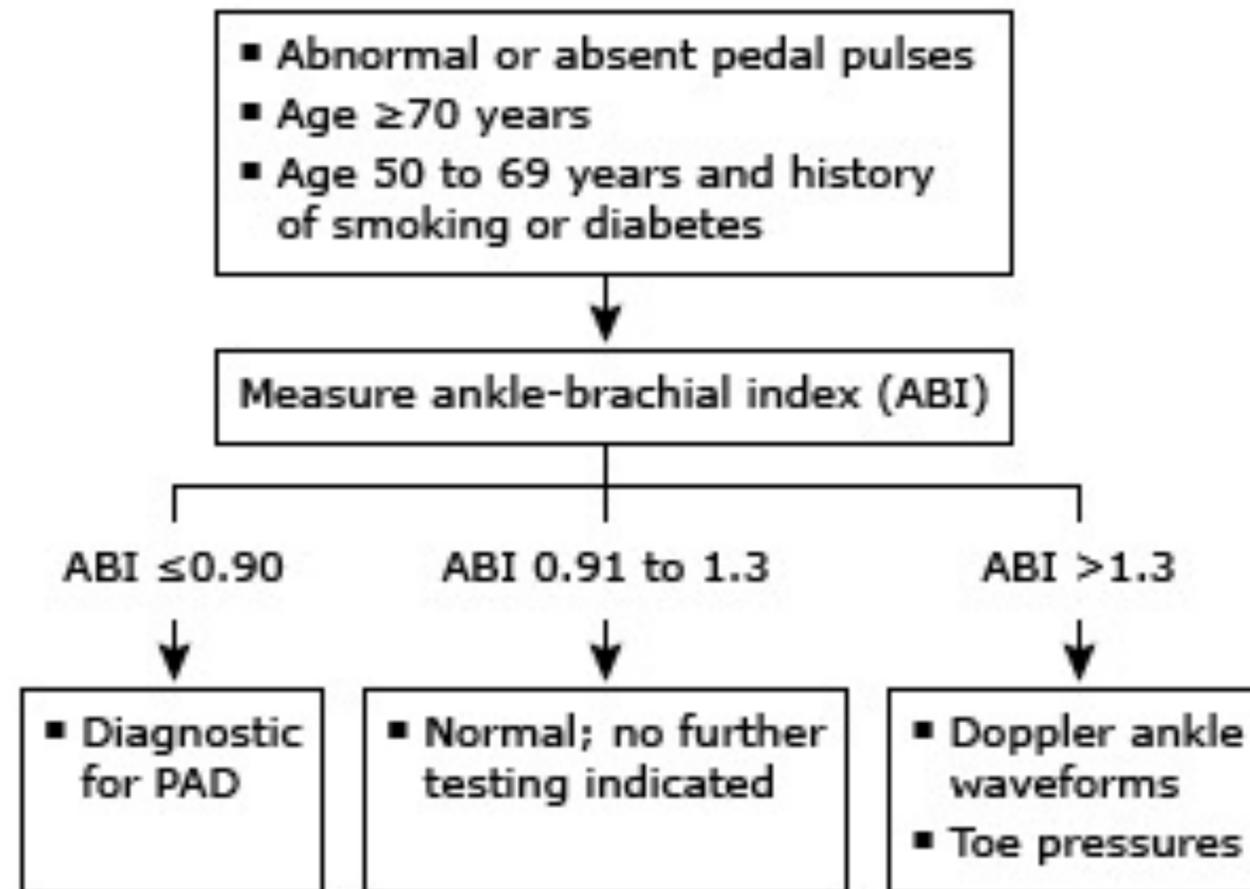
ABI - ankle brachial index

ABI is the ratio of the ankle systolic blood pressure divided by the brachial systolic pressure detected with a Doppler probe. In patients with no or mild to moderate symptoms, an ABI of <0.90 has been reported to have a sensitivity ranging from 79 to 95 percent, with specificity consistently >95 percent for diagnosing PAD

$>0.9- 1.2$	Normal
$0.7- 0.8-$	Adequate
$0.5- 0.6-$	Reduced
<0.5	Marked Arterial disease
>1.2	Calcified vessels

Vascular ulcer- Arterial/ischemic

Guideline for ABI measurement



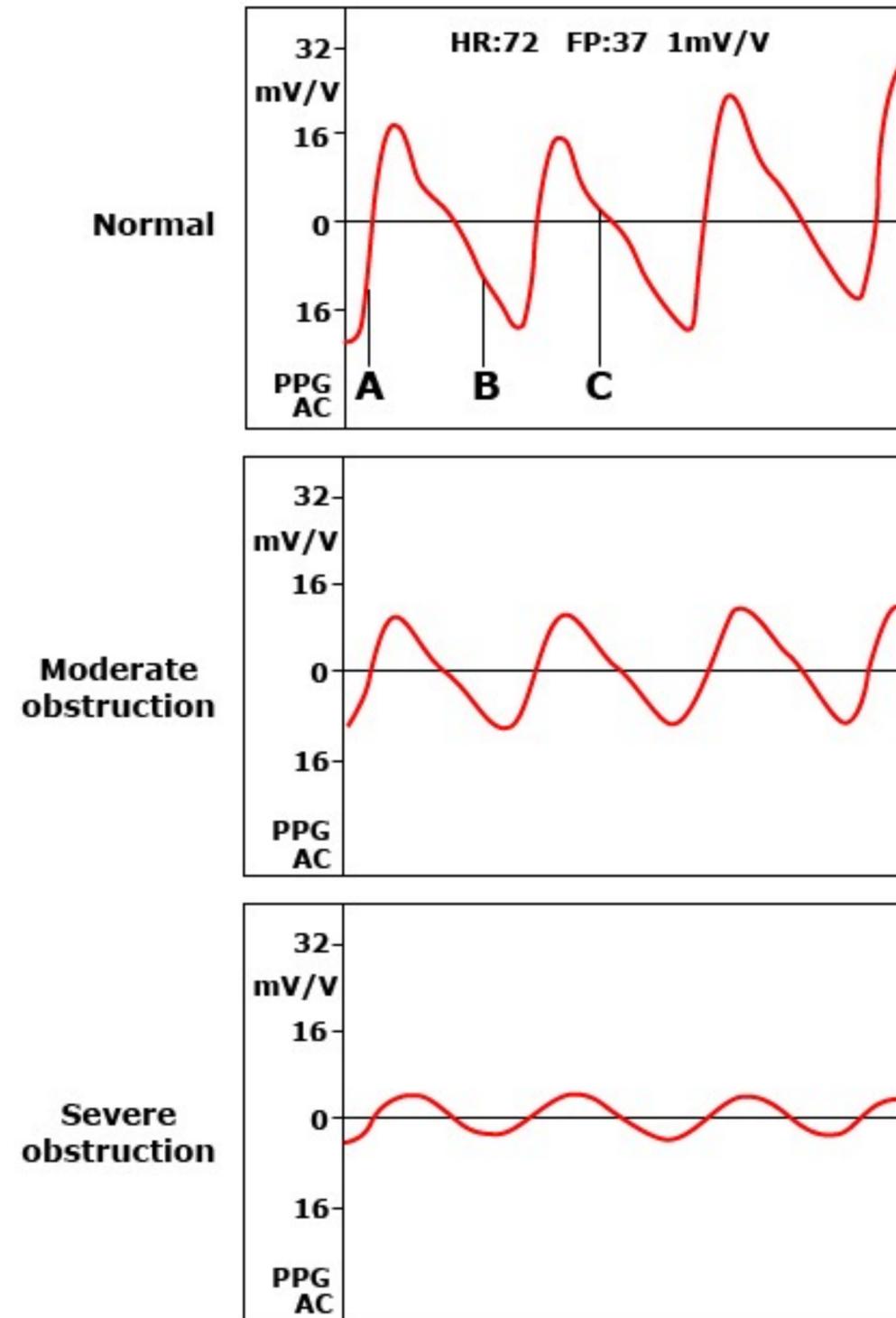
Toe brachial index

A pressure gradient of 20 to 30 mmHg between the ankle and the toe, and thus, a normal TBI is 0.7 to 0.8. An absolute toe pressure >30 mmHg is favorable for wound healing, although toe pressures >45 to 55 mmHg may be required for healing in patients with diabetes

With photoplethysmography (PPG), infrared light is emitted onto a defined area of skin. More or less light is absorbed depending upon changes in blood volume, which is determined by measuring the light reflected from the skin. A normal PPG waveform consists of a short rise in the upstroke during systole (A), a gradual decline during diastole (B), and a dicrotic notch in the diastolic downstroke (C). Moderate arterial obstruction results in loss of the dicrotic notch, flattening of the upstroke and downstroke, and rounding of the peaks of the waveform. Severe obstruction flattens the waveform.

mV: millivolts; V: volts; PPG: photoplethysmography; HR: heartrate; FP: finger pressure.

Time scale: 25 mm/s



Reproduced from: Kesselman P. Non-Invasive Arterial Vascular Testing: Providing these diagnostic services benefits both the patient and your bottom line. Podiatry Management Nov/Dec 2006. Copyright © 2006.

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Vascular- Arterial/ischemic ulcers

Transcutaneous Oxygen Measurement (TCOM)

- Platinum oxygen electrodes are placed on the chest wall and extremities and on any digits that are being evaluated. The absolute value of the oxygen tension at the limb, foot or hand, or a ratio of the foot or hand value relative to chest wall value, can be used.
- A normal value at the foot is 60 mmHg, and a normal chest/foot ratio is 0.9 . Local edema, skin temperature, emotional state (sympathetic vasoconstriction), inflammation, and pharmacologic agents limit the accuracy of the test.
- The level of TcPO₂ that predicts tissue healing remains controversial. It is generally accepted that in the absence of diabetes and tissue edema, wounds are likely to heal if oxygen tension is greater than 40 mmHg .
- A higher value is needed for healing a foot ulcer in the patient with diabetes. Patients with values of <20 mmHg are severely ischemic and are likely to need revascularization for wound healing.
- A meta-analysis of four studies found a more than threefold risk of developing a chronic wound healing complication in patients with a TcPO₂ below a threshold of 20 to 30 mmHg (odds ratio 3.2, 95% CI 1.1-9.7).

Vascular ulcers- arterial/ischemic

Management

- revascularization or amputation
- Revascularization can be endovascular or open
- Sometimes amputation is the correct surgery
- Adjunctively therapy- hyperbaric oxygen therapy cannot replace revascularisation, debridement (should not be debrided until arterial inflow has been re established)
- choice of dressings -dry gangrene or eschar are best left dry until revascularization is successful
- if that is adequate blood flow then moist wound healing environment should be provided
- topical antimicrobial dressings can be used but not routinely
- compression can be used if there is mixtured ulcer what ABI of greater than 0.5 (use reduced compression)
- for ABI less than 0.5 sustained high compression should not be used

Pressure injuries/ ulcers

**Pressure ulcers cost \$9.1 to \$11.6 billion
per year in the U S**

Pressure ulcer/injury

Staging

Changes were made to the National Pressure Injury Advisory Panel (NPIAP) system favoring the use of the term "pressure injury" instead of "pressure ulcer" to recognize the fact that lesser degrees of skin damage due to pressure may not be associated with skin ulceration (Stage 1) and that deep tissue pressure injury can occur without overlying skin ulceration

Staging of pressure-induced skin and soft tissue injuries^[1]

Stage	Description
1	Skin intact but with non-blanchable redness for >1 hour after relief of pressure.
2	Blister or other break in the dermis with partial thickness loss of dermis, with or without infection.
3	Full-thickness tissue loss. Subcutaneous fat may be visible; destruction extends into muscle with or without infection. Undermining and tunneling may be present.
4	Full-thickness skin loss with involvement of bone, tendon, or joint, with or without infection. Often includes undermining and tunneling.
Unstageable	Full-thickness tissue loss in which the base of the ulcer is covered by slough and/or eschar in the wound bed.
Deep tissue pressure injury	Purple or maroon localized area of discolored intact skin or blood-filled blister due to damage of underlying tissue from pressure and/or shear.

Reference:

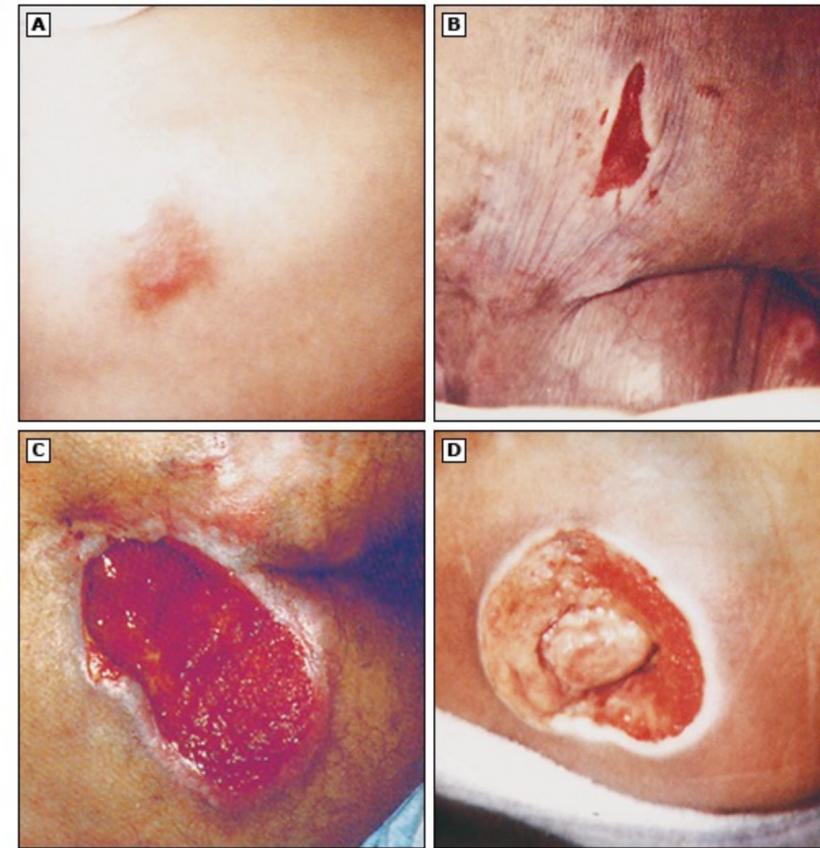
1. National Pressure Injury Advisory Panel. NPIAP Pressure Injury Stages. Available at: <https://npiap.com/page/PressureInjuryStages> (Accessed on January 29, 2020).

Pressure ulcer /injury

Staging

- Unstageable pressure injury is characterized by 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 (ie, dry, adherent, intact without erythema or fluctuance) on the heel or ischemic limb should not be softened or removed
- Documentation matters in discharge summary

Pressure-induced injury by stage



Pressure-induced injury staging^[1].

(A) Stage 1 – Skin intact but with nonblanchable redness.

(B) Stage 2 – Partial-thickness loss of skin with exposed dermis.

(C) Stage 3 – 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.

(D) Stage 4 – Full-thickness skin and tissue loss with exposed or directly palpable fascia, muscle, tendon, ligament, cartilage, or bone in the ulcer.

Not shown:

Unstageable pressure injury – 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.

Deep tissue pressure injury – Deep tissue pressure injury should be suspected whenever there is a localized area of skin (intact or non-intact) with persistent nonblanchable deep red, maroon, or purple discoloration or epidermal separation revealing a dark wound bed or blood-filled blister.

Reference:

1. National Pressure Injury Advisory Panel. www.npiap.com.

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Pressure Ulcers/Injuries

Risk Factors

- Older individuals
- Reduced pain perception
- Poor circulation
- Poor diet
- Altered mental state
- Incontinence

Pressure ulcers

Overall management goals

- Identify at risk patients and initiate early prevention strategies
- Implement strategies /plan to
 - Attain /maintain intact skin
 - prevent complications
 - promptly identify or manage complications
 - optimize potential for wound healing
 - involve caregiver or patient in self management
- Implement cost effective strategies or plans that prevent and treat pressure ulcers

Pressure Ulcers/Injuries

Offloading

- Reactive bed surface
- Active bed surface
 - Group 2
 - Group 3

Gel cushions

Heel protectors

Braden score

Pressure ulcers /injuries

Support Surfaces

- Reactive support surface – A powered or nonpowered support surface with the capability to change its load distribution properties only in response to applied load.
- Active support surfaces – A powered support surface, with the capability to change its load distribution properties, with or without applied load.
- Integrated bed system – A bed frame and support surface that are combined into a single unit whereby the surface is unable to function separately.
- Nonpowered support surface – Any support surface not requiring or using external sources of energy.
- Overlay – An additional support surface designed to be placed directly on the top of an existing surface.
- Mattress – A support surface designed to be placed directly on the existing bed frame.
- Features of a support surface that may assist in prevention include -
 - Air fluidized – Provides pressure redistribution through a fluid-like medium created by forcing air through beads
 - Alternating pressure – Provides pressure redistribution via cyclic changes in loading and unloading
 - Lateral rotation – Provides rotation about a longitudinal axis
 - Low air loss – Provides a flow of air to assist in managing the heat and humidity of the skin
 - Multizoned – Different segments of the support surface have different pressure redistribution characteristics

Pressure ulcers /injuries

Support surfaces

Group 1 mattress overlay



Group 2 Mattress



Group 3/Clinitron



Pressure ulcers/injuries

Offloading equipments

Prevlon Boot for heel protection



Roho Cushion



Braden Score

Braden scale for predicting risk of pressure-induced injury*

Sensory perception	Moisture	Activity	Mobility	Nutrition	Friction & shear
Ability to respond meaningfully to pressure-related discomfort	Degree to which skin is exposed to moisture	Degree of physical activity	Ability to change and control body position	Usual food intake pattern	
1. Completely limited	1. Constantly moist	1. Bedfast	1. Completely immobile	1. Very poor	1. Problem
Unresponsive (does not moan, flinch, or grasp) to painful stimuli, due to diminished level of consciousness or sedation OR Limited ability to feel pain over most of body	Skin is kept moist almost constantly by perspiration, urine, etc. Dampness is detected every time patient is moved or turned.	Confined to bed	Does not make even slight changes in body or extremity position without assistance	Never eats a complete meal. Rarely eats more than 1/3 of any food offered. Eats 2 servings or less of protein (meat or dairy products) per day. Takes fluids poorly. Does not take a liquid dietary supplement. OR Is NPO and/or maintained on clear liquids or IV's for more than 5 days	Requires moderate to maximum assistance in moving. Complete lifting without sliding against sheets is impossible. Frequently slides down in bed or chair, requiring frequent repositioning with maximum assistance. Spasticity, contractures or agitation leads to almost constant friction.
2. Very limited	2. Very moist	2. Chairfast	2. Very limited	2. Probably inadequate	2. Potential problem
Responds only to painful stimuli Cannot communicate discomfort except by moaning or restlessness OR Has a sensory impairment which limits the ability to feel pain or discomfort over 1/2 of body	Skin is often, but not always moist. Linen must be changed at least once a shift.	Ability to walk severely limited or non-existent. Cannot bear own weight and/or must be assisted into chair or wheelchair.	Makes occasional slight changes in body or extremity position but unable to make frequent or significant changes independently	Rarely eats a complete meal and generally eats only about 1/2 of any food offered. Protein intake includes only 3 servings of meat or dairy products per day. Occasionally will take a dietary supplement. OR Receives less than optimum amount of liquid diet or tube feeding	Moves feebly or requires minimum assistance. During a move skin probably slides to some extent against sheets, chair, restraints or good position in chair or bed most of the time but occasionally slides down.
3. Slightly limited	3. Occasionally moist	3. Walks occasionally	3. Slightly limited	3. Adequate	3. No apparent problem
Responds to verbal commands, but cannot always communicate discomfort or the need to be turned OR Has some sensory impairment which limits ability to feel pain or discomfort in 1 or 2 extremities	Skin is occasionally moist, requiring an extra linen change approximately once a day	Walks occasionally during day, but for very short distances, with or without assistance. Spends majority of each shift in bed or chair.	Makes frequent though slight changes in body or extremity position independently	Eats over half of most meals. Eats a total of 4 servings of protein (meat, dairy products) per day. Occasionally will refuse a meal, but will usually take a supplement when offered. OR Is on a tube feeding or TPN regimen which probably meets most of nutritional needs	Moves in bed and in chair independently and has sufficient muscle strength to lift up completely during move. Maintains good position in bed or chair.
4. No impairment	4. Rarely moist	4. Walks frequently	4. No limitation	4. Excellent	
Responds to verbal commands Has no sensory deficit which would limit ability to feel or voice pain or discomfort	Skin is usually dry, linen only requires changing at routine intervals	Walks outside room at least twice a day and inside room at least once every two hours during waking hours	Makes major and frequent changes in position without assistance	Eats most of every meal. Never refuses a meal. Usually eats a total of 4 or more servings of meat and dairy products. Occasionally eats between meals. Does not require supplementation.	
Score: _____	Score: _____	Score: _____	Score: _____	Score: _____	Score: _____

* To calculate the Braden scale score, rank the patient in each of the subscales; sensory perception, mobility, activity, moisture, nutrition and friction and shear. Add the six subscale scores to yield a total Braden scale score. Lower scores are associated with a higher risk of developing pressure scores. A score of 18 or less indicates high risk. See www.bradenscale.com.

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Diabetic Foot Ulcers (DFU)

Diabetic foot Ulcer (DFU)

The Problem

- The lifetime risk of a foot ulcer in patients with diabetes (type 1 or 2) may be as high as 34 percent.
- Diabetic foot ulcer (DFU) prevalence is as high as 25% and 40-80% of DFUs become infected (DFI). About 20% of infected ulcers will spread to bone causing diabetic foot osteomyelitis (DFO).
- Diabetic foot ulcers are a major cause of morbidity , accounting for at least two-thirds of all nontraumatic amputations performed in the United States. DFU patients have a 3-year cumulative mortality rate of 28% and rates approaching 50% in amputated patients.
- DFU costs Medicare \$9-13 billion/year. The most expensive costs associated with DFU are inpatient costs and hospital admissions. DFO costs are driven mostly by surgical procedures.
- Based on data from the World Health Organization, lower extremity complications of diabetes constitute a top ten condition in terms of years lived with disability.
- Patients with diabetes with or without a diabetic foot ulcer have increased rates of depression, and expressing signs of depression is associated with an increased risk of diabetic foot ulcers

• Current health and economic burden of chronic diabetic osteomyelitis [Terese Geraghty](#) & [Guido LaPorta](#)

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• Uptodate

Diabetic foot Ulcer (DFU)

Risk factors

- Neuropathy
- Prior ulcers or amputations
- Foot deformity leading to excess pressure, external trauma, infection
- Effects of chronic ischemia, typically due to peripheral artery disease
- For healing capacity due to high levels of matrix metalloproteinases (MMP 9) come on low levels of growth factors come on for collagen production , impaired resistance and response to local infection come on limited angiogenesis, decreased fibroblasts replication

Diabetic foot Ulcer (DFU)

Classification/Grading

The first step in managing diabetic foot ulcers is assessing, grading, and classifying the ulcer.

Classification is based upon

- the extent and depth of the ulcer
- the presence of infection or ischemia- ankle-brachial index and toe pressure measurements.

Diabetic foot Ulcer (DFU)

Ulcer classification

University of Texas system

Assessed wound depth, the presence of infection, and peripheral arterial occlusive disease for every category of the wound assessment

Grade:

Grade 0: Pre- or postulcerative (Stages A to D)

Grade 1: Full-thickness ulcer not involving tendon, capsule, or bone (Stages A to D)

Grade 2: Tendon or capsular involvement without bone palpable (Stages A to D)

Grade 3: Probes to bone (Stages A to D)

Stage:

A: Noninfected

B: Infected

C: Ischemic

D: Infected and ischemic

Diabetic foot Ulcer (DFU)

Superficial diabetic foot ulcer



Full thickness diabetic foot ulcer



Foot from a diabetic patient with a penetrating neuropathic ulcer that is not associated with abscess formation or bone involvement.

Dorsal diabetic foot wound



Dorsal diabetic foot wound following debridement of necrotizing streptococcal infection with exposed tendon and joint capsule.

Diabetic foot ulcer (DFU)

Deep diabetic foot ulcer involving the bone



Infected and ischemic diabetic foot ulcer



Courtesy of Paul Thottingal, MD.
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Courtesy of David McCulloch, MD.
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Diabetic foot ulcer (DFU)

Ulcer classification

Threatened limb classification: WIfI

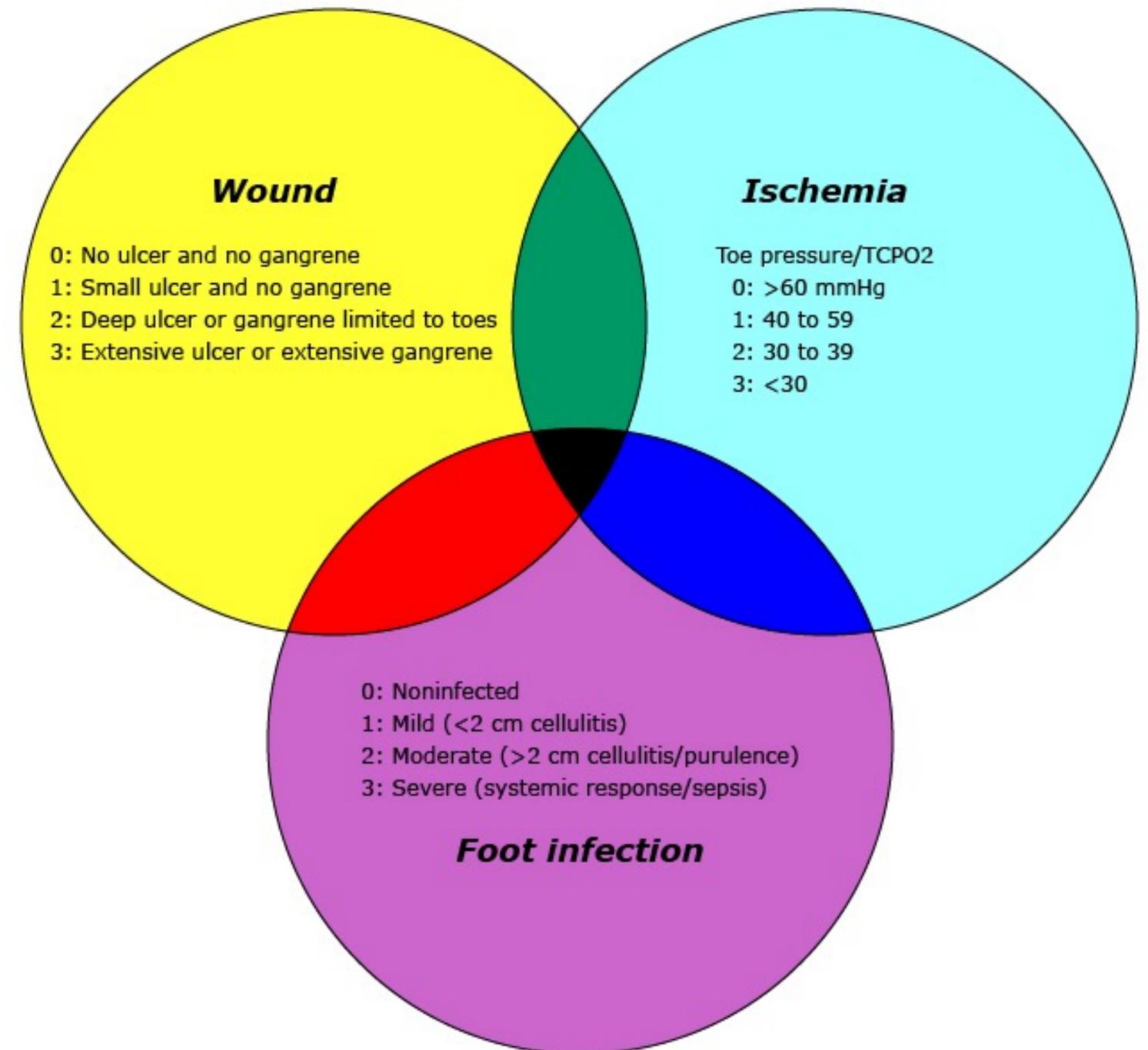
To provide a more quantitative assessment of peripheral artery disease (PAD) as a predictor and contributor to lower extremity pathology, the Society for Vascular Surgery has proposed the structure of the Wound/Ischemia/Foot Infection (WIfI) system

Scoring- none/mild/moderate/severe basis
(0/1/2/3)

Threatened limb classification: WIfI

Pathophysiologic assessment in chronic foot ulcers

The conceptual diagram illustrates the interaction between the main factors that contribute to tissue loss. This scheme is appropriate for any patient with a chronic wound/tissue loss. The clinician should ask, "Which factor or combination of factors contributes the most to the pathophysiology of the wound? Ischemia? Infection? Wound extent?" Early assessment helps determine initial wound management priorities, but frequent reassessment is important since the wound environment is dynamic, and the balance toward one or another factor can change.



Diabetic foot ulcer (DFU)

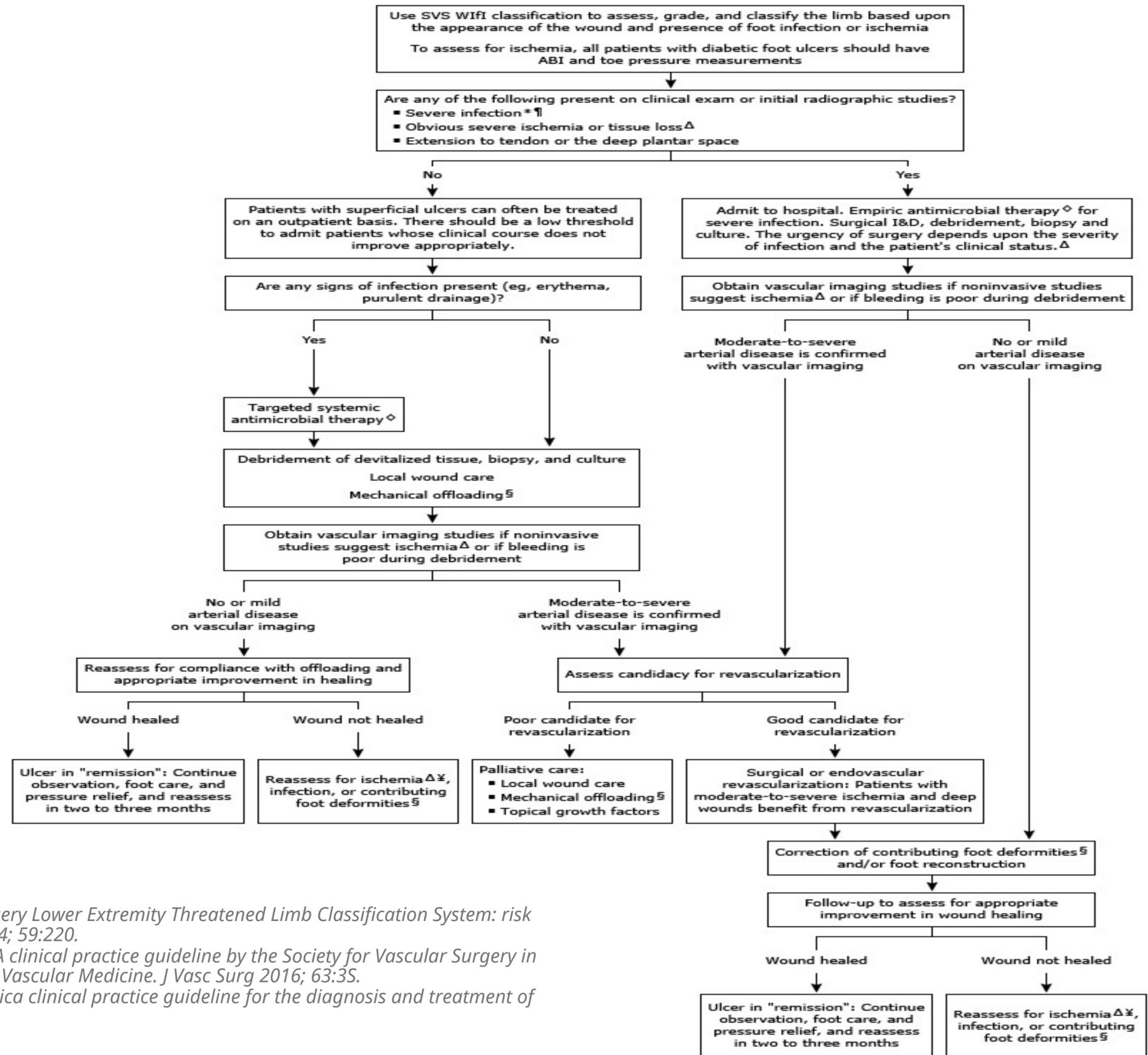
Ulcer classification

Wagner grade

- Grade 1: Superficial ulcer – Skin and subcutaneous tissue only
- Grade 2: Deep ulcer to tendon, muscle, joint capsule, or bone
- Grade 3: Deep ulcer with abscess, osteomyelitis, or tendinitis
- Grade 4: Partial foot gangrene
- Grade 5: Whole foot gangrene

Doesn't account for the vascular status of the foot

Management of diabetic foot wounds



1. References: Mills JL, Conte MS, Armstrong DG, et al. The Society for Vascular Surgery Lower Extremity Threatened Limb Classification System: risk stratification based on wound, ischemia, and foot infection (WifI). *J Vasc Surg* 2014; 59:220.

2. Hingorani A, LaMuraglia GM, Henke P, et al. The management of diabetic foot: A clinical practice guideline by the Society for Vascular Surgery in collaboration with the American Podiatric Medical Association and the Society for Vascular Medicine. *J Vasc Surg* 2016; 63:3S.

3. Lipsky BA, Berendt AR, Cornia PB, et al. 2012 Infectious Diseases Society of America clinical practice guideline for the diagnosis and treatment of diabetic foot infections. *Clin Infect Dis* 2012; 54:e132.

Diabetic foot ulcers (DFU)

Management:

- Sugar control – Unclear as to aggressive control makes any difference in DFU (yes in amputations)
- protein intake
- L-arginine intake
- Local wound care includes sharp debridement and proper own coverage with proper choice of dressing
- Infection
- Peripheral vascular disease
- Edema (venous insufficiency)
- Offloading
- Surgical correction

Diabetic foot Ulcer (DFU)

Adjunctive therapy

- Offloading
- Role of hyperbaric oxygen therapy
- Pressure redistribution (offloading)
- Advanced tissue therapies

Diabetic foot Ulcer (DFU)

Offloading

The use of total contact casts and nonremovable cast walkers for relief of pressure to improve healing of diabetic foot ulcers.

Total contact cast —

Takes pressure off the heel or elsewhere on the foot by averaging the pressure across the sole of the foot
is advantage- need expertise to apply

-inability to frequently inspect the foot

-risk of developing the secondary ulcer in an ill fitting cast

Total contact casts - Contraindications

- infected ulcers or wounds
- osteomyelitis
- peripheral ischemia (ankle-brachial index <0.6)
- bilateral ulceration
- lower extremity amputation
- heel ulceration

Diabetic foot Ulcer (DFU)

Offloading

Removable cast Walker



Rocker sole shoe



Wedge shoes



Total contact cast system



Knee Walker



Diabetic foot Ulcer (DFU)

The role of hyperbaric oxygen therapy

Recommendation 1:

In patients with Wagner Grade 2 or lower diabetic foot ulcers, we suggest against using hyperbaric oxygen therapy (very low-level evidence in support of HBO2, conditional recommendation).

Recommendation 2:

In patients with Wagner Grade 3 or higher diabetic foot ulcers that have not shown significant improvement after 30 days of treatment, we suggest adding hyperbaric oxygen therapy to the standard of care to reduce the risk of major amputation and incomplete healing (moderate-level evidence, conditional recommendation).

Recommendation 3:

In patients with Wagner Grade 3 or higher diabetic foot ulcers who have just had a surgical debridement of an infected foot (e.g., partial toe or ray amputation; debridement of ulcer with underlying bursa, cicatrix or bone; foot amputation; incision and drainage [I&D] of deep space abscess; or necrotizing soft tissue infection), we suggest adding acute post-operative hyperbaric oxygen therapy to the standard of care to reduce the risk of major amputation and incomplete healing (moderatelevel evidence, conditional recommendation)

Properties of topical agents and dressing materials

Properties of topical agents and dressing materials

Type	Actions	Indications/use	Precautions/contraindications
Alginates/CMC*	<ul style="list-style-type: none"> Absorb fluid. Promote autolytic debridement. Moisture control. Conformability to wound bed. 	<ul style="list-style-type: none"> Moderate to high exuding wounds. Special cavity presentations in the form of rope or ribbon. Combined presentation with silver for antimicrobial activity. 	<ul style="list-style-type: none"> Do not use on dry/necrotic wounds. Use with caution on friable tissue (may cause bleeding). Do not pack cavity wounds tightly.
Foams	<ul style="list-style-type: none"> Absorb fluid. Moisture control. Conformability to wound bed. 	<ul style="list-style-type: none"> Moderate to high exuding wounds. Special cavity presentations in the form of strips or ribbon. Low-adherent versions available for patients with fragile skin. Combined presentation with silver or PHMB for antimicrobial activity. 	<ul style="list-style-type: none"> Do not use on dry/necrotic wounds or those with minimal exudate.
Honey	<ul style="list-style-type: none"> Rehydrate wound bed. Promote autolytic debridement. Antimicrobial action. 	<ul style="list-style-type: none"> Sloughy, low to moderate exuding wounds. Critically colonized wounds or clinical signs of infection. 	<ul style="list-style-type: none"> May cause "drawing" pain (osmotic effect). Known sensitivity.
Hydrocolloids	<ul style="list-style-type: none"> Absorb fluid. Promote autolytic debridement. 	<ul style="list-style-type: none"> Clean, low to moderate exuding wounds. Combined presentation with silver for antimicrobial activity. 	<ul style="list-style-type: none"> Do not use on dry/necrotic wounds or high exuding wounds. May encourage overgranulation. May cause maceration.
Hydrogels	<ul style="list-style-type: none"> Rehydrate wound bed. Moisture control. Promote autolytic debridement. Cooling. 	<ul style="list-style-type: none"> Dry/low to moderate exuding wounds. Combined presentation with silver for antimicrobial activity. 	<ul style="list-style-type: none"> Do not use on highly exuding wounds or where anaerobic infection is suspected. May cause maceration.
Iodine	<ul style="list-style-type: none"> Antimicrobial action. 	<ul style="list-style-type: none"> Critically colonized wounds or clinical signs of infection. Low to high exuding wounds. 	<ul style="list-style-type: none"> Do not use on dry necrotic tissue. Known sensitivity to iodine. Short-term use recommended (risk of systemic absorption).
Low-adherent wound contact layer (silicone)	<ul style="list-style-type: none"> Protect new tissue growth. Atraumatic to periwound skin. Conformable to body contours. 	<ul style="list-style-type: none"> Low to high exuding wounds. Use as contact layer on superficial low exuding wounds. 	<ul style="list-style-type: none"> May dry out if left in place for too long. Known sensitivity to silicone.
PHMB	<ul style="list-style-type: none"> Antimicrobial action. 	<ul style="list-style-type: none"> Low to high exuding wounds. Critically colonized wounds or clinical signs of infection. May require secondary dressing. 	<ul style="list-style-type: none"> Do not use on dry/necrotic wounds. Known sensitivity.
Odor control (eg, activated charcoal)	<ul style="list-style-type: none"> Odor absorption. 	<ul style="list-style-type: none"> Malodorous wounds (due to excess exudate). May require antimicrobial if due to increased bioburden. 	<ul style="list-style-type: none"> Do not use on dry wounds.
Protease modulating	<ul style="list-style-type: none"> Active or passive control of wound protease levels. 	<ul style="list-style-type: none"> Clean wounds that are not progressing despite correction of underlying causes, exclusion of infection, and optimal wound care. 	<ul style="list-style-type: none"> Do not use on dry wounds or those with leathery eschar.
Silver	<ul style="list-style-type: none"> Antimicrobial action. 	<ul style="list-style-type: none"> Critically colonized wounds or clinical signs of infection. Low to high exuding wounds. Combined presentation with foam and alginates/CMC for increased absorbency. Also in paste form. 	<ul style="list-style-type: none"> Some may cause discoloration. Known sensitivity. Discontinue after 2 weeks if no improvement and reevaluate.
Polyurethane film	<ul style="list-style-type: none"> Moisture control. Breathable bacterial barrier. Transparent (allow visualization of wound). 	<ul style="list-style-type: none"> Primary dressing over superficial low exuding wounds. Secondary dressing over alginate or hydrogel for rehydration of wound bed. 	<ul style="list-style-type: none"> Do not use on patients with fragile/compromised periwound skin. Do not use on moderate to high exuding wounds.

Other more advanced dressings (eg, collagen and bioengineered tissue products) may be considered for wounds that are hard to heal^[1].

CMC: carboxymethylcellulose; PHMB: polyhexamethylene biguanide.

* Wound dressings may contain alginates or CMC only; alginates may also be combined with CMC.

Reference:

1. International Consensus. Acellular matrices for the treatment of wounds. An expert working group review. Wounds International 2010. Available at <http://woundsinternational.com> (Accessed on March 2013).

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Skin substitute classification as described by Kumar^[1]

Class	Description	Subcategory	Subdivision	Product example
I	Skin substitutes with mechanical traits of the epidermis; lack keratinocytes	Single-layered materials	Naturally occurring membrane or cover as biological dressing substitute	Biomembrane Biocompatible vegetal membranes derived from the <i>Hevea brasiliensis</i> rubber tree
			Single-layer synthetic skin dressing material substitute	Tegaderm, Opsite, Dermafilm, Nexfill, Gelapin
		Bi-layered tissue-engineered materials	–	TransCyte
II	Epidermal or dermal skin substitutes	Epidermal substitutes – similar to human epidermis; prone to breakdown; poor healing outcomes	–	Epicel, EpiDex, Laserskin, MySkin, BioSeed, CellSpray, cultured epithelial allograft (CEA)
		Dermal substitutes – composition that includes proteins found in the dermal matrix	–	Kollagen, Permacol, Matriderm, Alloderm
III	Materials that replace both the dermal and epidermal layer	Skin graft	–	Allograft from cadaver, Xenograft from porcine origin
		Tissue-engineered skin	–	Apligraf, Integra, Biobrane

Reference:

1. Kumar P. Classification of skin substitutes. *Burns* 2008; 34:148.

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Atypical Wounds

- Malignancy
- Dermatological
- Autoimmune
- Infectious
- Bites
- Pyoderma Gangrenosum

Summary

In Primary Care Setting

- History, History, History
- Physical Exam- Location
- Culture –Levine technique- roll the Qtip for 1 cm² and give enough pressure to bleed
- Etiology can be overlapping
- ABI on all patients with DFU
- ABI on patients at risk for PAD before applying compression therapy in VLU
- VLU- COMPRESSION is KEY
- DFU- OFFLOADING is KEY
- Pressure ulcer/injury- OFFLOADING is KEY

Questions?