THE UNIVERSITY OF JORDAN

Normal Wound Healing

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- Wound closure:

A. Primary closure: Immediate suturing of the wound
B. Delayed primary closure: Leave stitches in the wound and close it after 3-5 days when wound is clean. We do this method for contaminated wounds.
C. Secondary closure: By scar formation and epithelisation.
D. Tertiary: By graft or flap.

- Phase of Wound Healing: Look at the diagram

A. Inflammatory
B. Proliferative phase
C. Remodeling phase

Please refer to these links:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2903966/

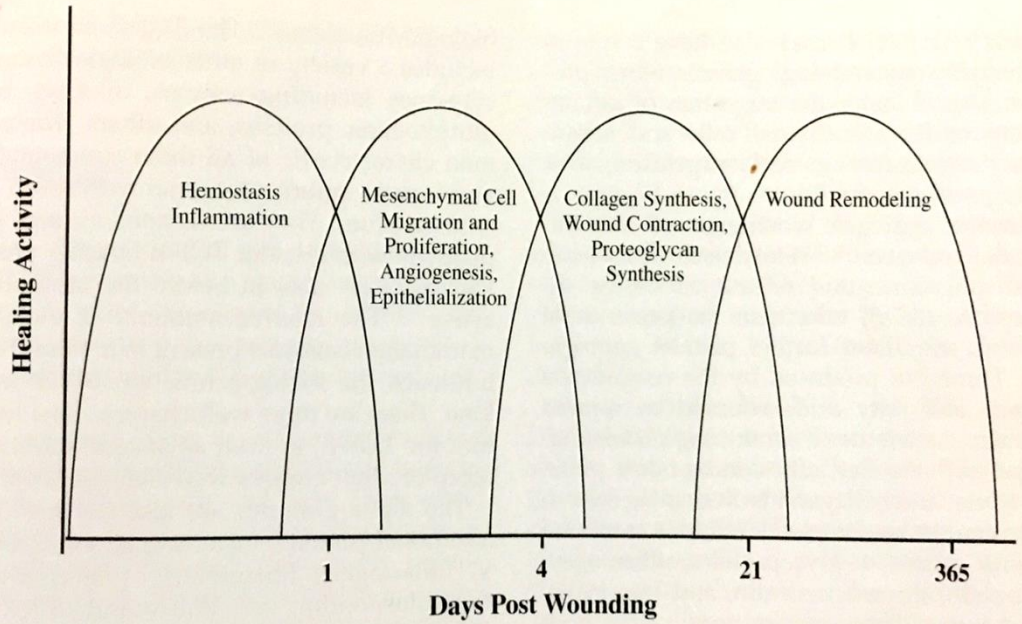


Figure 1. Phases of the healing process.

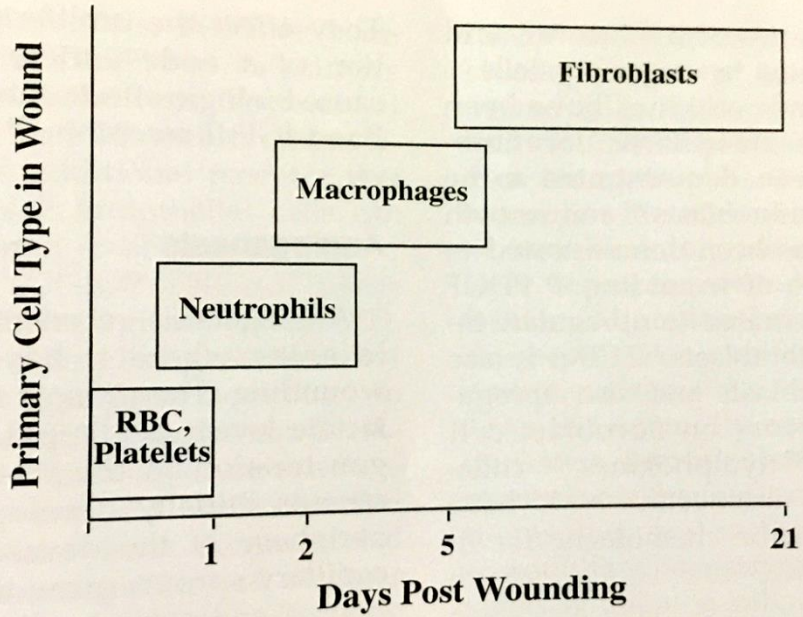


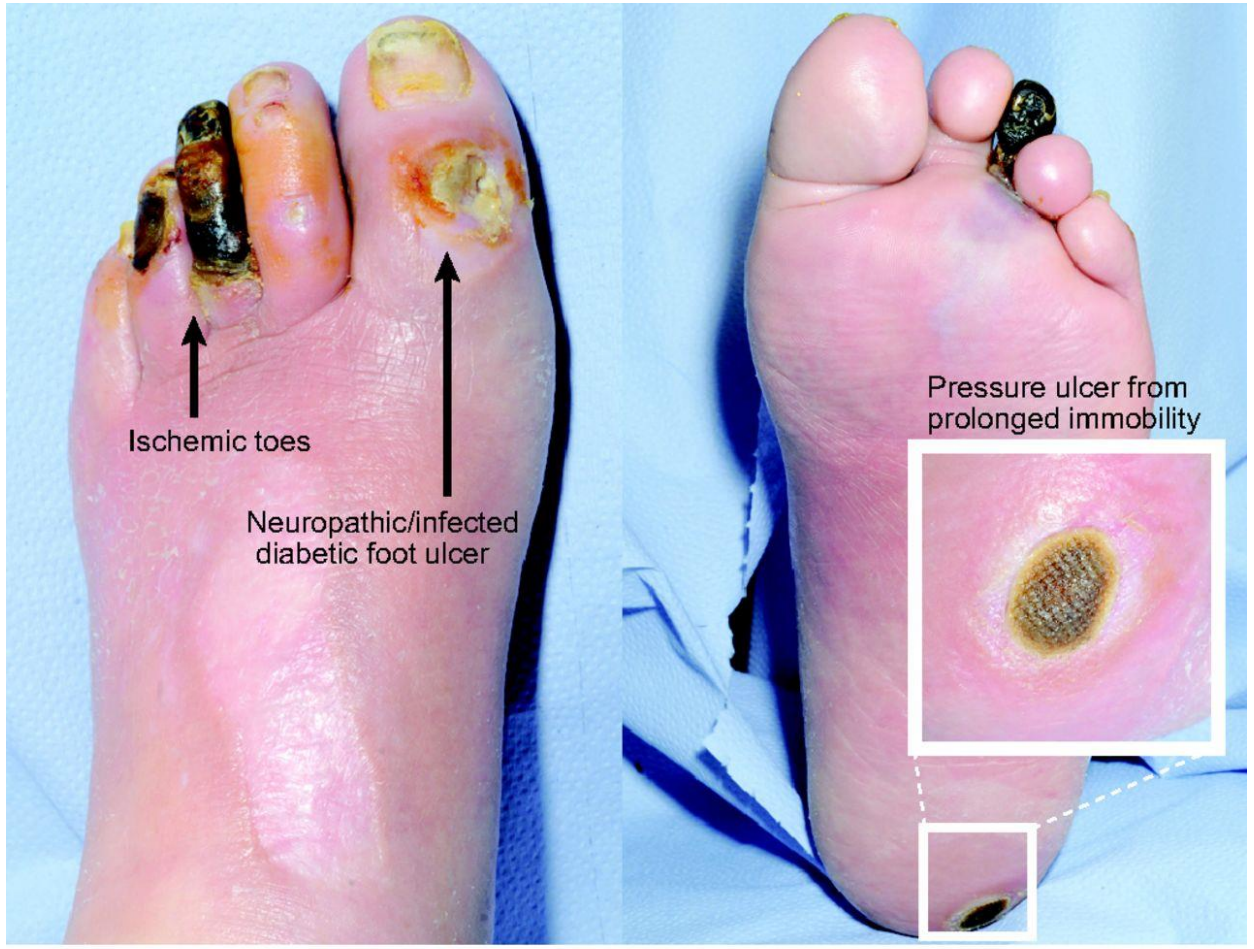
Figure 2. Cellular makeup of the healing wound.

Table 1. CYTOKINE INVOLVEMENT IN WOUND HEALING FUNCTIONS

Healing Function	Cytokines Involved
Inflammatory Cell Migration	PDGF
	TGF-β
	TNF-α
Fibroblast Migration	PDGF
	TGF-β
	EGF
Fibroblast Proliferation	PDGF
	TGF-β
	EGF
	IGF
	TNF-α
	IL-1
Angiogenesis	bFGF (FGF2)
	aFGF (FGF1)
	TGF-β
	TGF-α
	EGF
	TNF-α
	VEGF
	IL-8
	PD-ECGF
Epithelialization	EGF
	TGF-α
	KGF (FGF7)
	bFGF (FGF2)
	IGF
	HB-EGF
Collagen Synthesis	PDGF
	TGF-β
	bFGF (FGF2)
	EGF

PDGF = platelet-derived growth factor; TGF-β = transforming growth factor-β; TNF-α = tumor necrosis factor-α; EGF = epidermal growth factor; IGF = insulin-like growth factor; IL-1 = interleukin-1; bFGF = basic fibroblast growth factor; aFGF = acidic fibroblast growth factor; TGF-α = transforming growth factor-α; VEGF = vascular endothelial growth factor; IL-8 = interleukin-8; PD-ECGF = platelet-derived-endothelial cell growth factor; KGF = keratinocyte growth factor; and HB-EGF = heparin binding epidermal growth factor.

Chronic Wound



Dorsal surface

Plantar surface

Chronic Wound

Factors contributing to impaired wound healing

A. Local factors	B. Systemic factors
❖ Arterial insufficiency ❖ Venus insufficiency ❖ Edema ❖ Infection ❖ Pressure ❖ Radiation ❖ Foreign material ❖ Necrotic tissue	❖ DM ❖ Malnutrition ❖ Vitamin deficiency ❖ Chemotherapy ❖ Smoking ❖ Aging ❖ Steroids

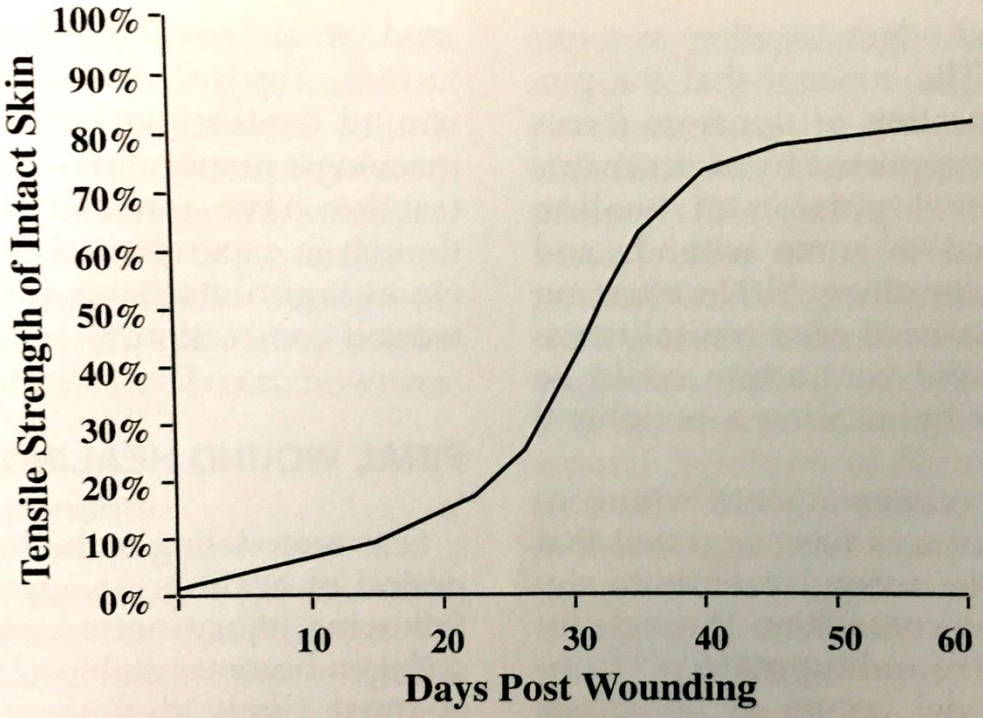


Figure 3. Tensile strength of the healing wound.

Table 1. THE ESTIMATED PREVALENCE AND HEALTH CARE COSTS OF CHRONIC WOUNDS.

Wound Type	Total Prevalence	Estimated Annual Cost
Pressure Ulcer[1]	0.04–0.08%	$1.3 billion
Venous Ulcer[2]	1–2%	$1 billion
Diabetic Ulcer[3]	Total 0.15–0.3% (Diabetics 5–10%)	$1 billion

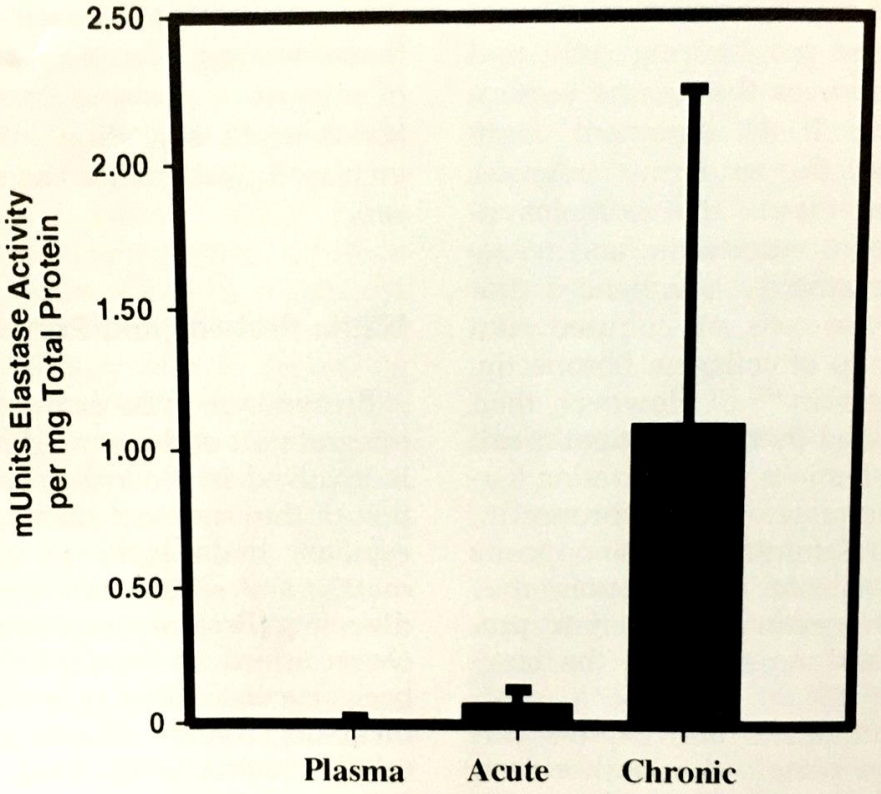


Figure 2. Levels of elastase activity are significantly higher in chronic wound fluid compared with acute wound fluid. Elastase activity was determined by a colorimetric assay using methoxysuccinyl-ala-ala-proval-p-nitoanilide substrate. (*From* Yager DR, Chen SM, Ward BS, et al: Ability of chronic wound fluid to degrade peptide growth factors is associated with increased levels of elastase activity and diminished levels of proteinase inhibitors. Wound Repair and Regeneration 5:23, 1997; with permission.)

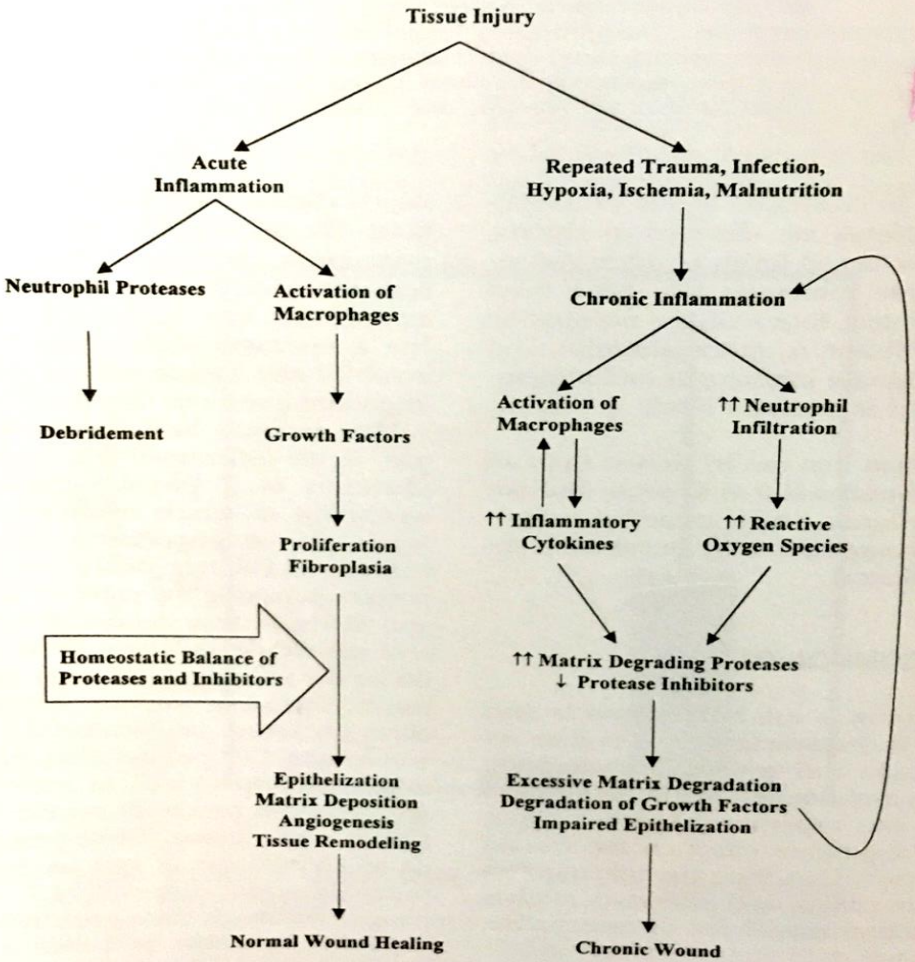


Figure 4. The final common pathway in the pathophysiology of chronic wounds.

Excessive Wound Healing

1. Keloids
2. Hypertrophic scars

Please refer to this link:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4129552/

- Etiology
- Histology
- Treatment

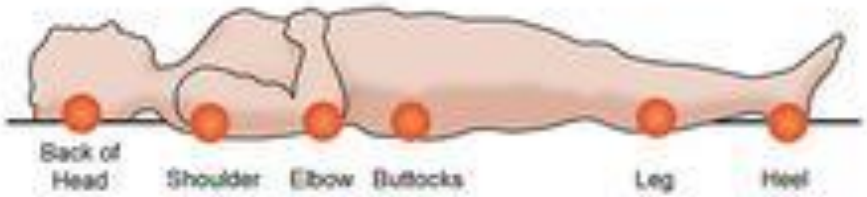
❖ Surgical excision
❖ Z-Plasty
❖ W-Plasty
❖ Steroids
❖ Silicon
❖ Pressure garment
❖ Laser
❖ Interferon

Pressure Ulcers
Bed sores

- Definition
- Etiology
- Pre-disposing factors
- Locations
- Prevention
- Work up
- Treatment : Medical surgical
- Complications of surgery

Please refer to the following links:

https://www.researchgate.net/publication/257777910_Bedsores_Top_to_bottom_and_bottom_to_top


Back of Head
Shoulder
Elbow
Buttocks
Leg
Heel


Ear
Shoulder
Elbow
Hip
Thigh
Leg
Heel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