

# Wound Healing

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# Outlines ...

- Introduction
- What we need to know
- **WOUND HEALING STAGES**
- **TYPES OF WOUND HEALING**
- CYTOKINES
- **Factors affect wound healing**
- COMPLICATIONS OF WOUND HEALING
- Summary

# Introduction

It is a series of events that begins at the moment of injury and can continue for months to years.

it is a complex and dynamic process of restoring cellular structures and tissue layers.

Involves multiple cell types, growth factors, and extracellular matrix; Essential for restoring tissue integrity after injury

What we need to know... ✓ ?



Knowing the cell  
types involved



Knowing the order in  
which they appear in  
the wound



The growth factors  
and cytokines  
functions

# WOUND HEALING STAGES

Haemostasis

II . INFLAMMATORY  
PHASE

III . PROLIFERAT  
IVE PHASE

IV . MATURATION  
PHASE

# Hemostasis Phase

- Immediate response after injury

1

Vasoconstriction to reduce blood loss

2

Platelet plug formation

3

Fibrin clot stabilizes wound

# Inflammatory Phase

- Begins within hours, lasts 2-5 days

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a. vasodilatation

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b. Phagocytosis:

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-leucocyte activation

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-lymphocyte activation

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-macrophage role

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- Neutrophils and macrophages remove debris and pathogens

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- Release of cytokines and growth factors

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- Signs: redness, heat, swelling, pain

# Proliferative Phase



**PERIOD : 2 DAYS TO  
3 WEEKS**



**STEPS :**

# A- Angiogenesis:

Formation of granulation tissue which occurs 3-5 days following injury and overlaps with the preceding inflammatory phase

## B- Fibroplasia:

- Fibroblasts are responsible for the production of collagen, elastin, fibronectin, glycosaminoglycans

# C- Epithelization

- Begins within hours of tissue injury.
- Epidermal cells at the wound edges undergo structural changes, allowing them to detach from their connections
- Cell may travel about 3 cm from point of origin in all directions

## d. Wound contraction

- Wound edges pull together to reduce the defect
- It is maximal 5-15 days after injury.
- The maximal rate of contraction is 0.75 mm/day and depends on the degree of tissue laxity.
- Wound contraction depends on the myofibroblast located at the periphery of the wound

# E. Collagen synthesis

- Collagen is rich in hydroxylysine and hydroxyproline moieties, which enable it to form strong cross-links.
- The hydroxylation of proline and lysine residues depends on the presence of oxygen, vitamin C, ferrous iron.

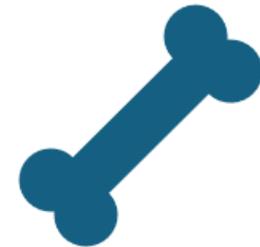
# Remodeling Phase



Period : 3 weeks to 2 years



New collagen forms which  
increases tensile strength to  
wounds



Scar tissue formation ; only  
80 percent as strong as  
original tissue Type III  
collagen replaced by Type I  
collagen



collagen becomes increasingly organized  
Type III collagen is replaced by type I collagen



Water is resorbed from the scar



remodeling begins approximately 21 days after injury

# WOUND HEALING

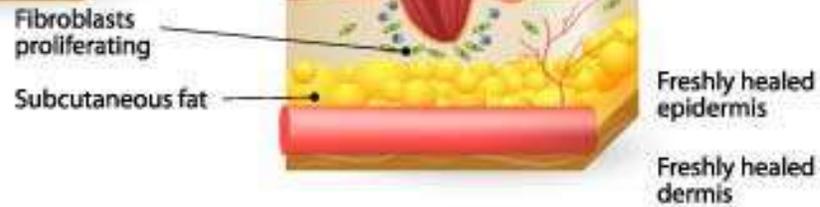
## Bleeding



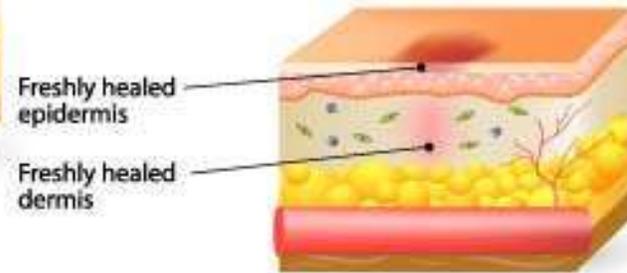
## Inflammatory



## Proliferative



## Remodeling



# TYPES OF WOUND HEALING

## Primary healing "healing by 1<sup>st</sup>. Intension" :

- involves closure of a wound within hours of its creation
- Combination of epithelization and connective tissue formation

## Secondary healing "healing by 2<sup>nd</sup>.intension:

- involves no formal wound closure, the wound closes spontaneously by: contraction, connective formation and re

## Healing by 3<sup>rd</sup>. Intention:

- also known as delayed primary closure, involves initial debridement of the wound for an extended period and then formal closure with suturing

# CYTOKINES IN WOUND HEALING

- a cytokine is a protein mediator, released from various cell sources, which binds to cell surface receptors to stimulate a cell response.



It provide all the comunications for cell to cell interaction.



Play role in regulating fibrosis.



They regulate cell proliferation



Stimulate cell to migrate to the wound site



Direct cells to produce specific components needed for the repair

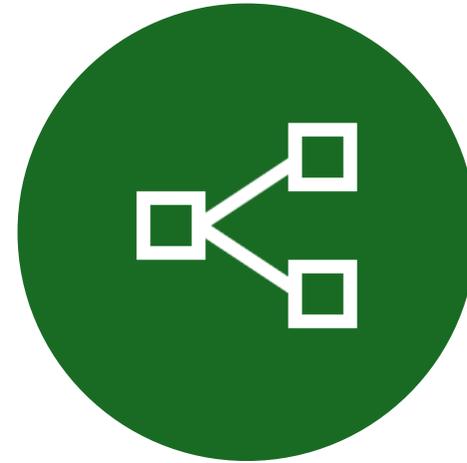
# CYTOKINES

cytokin	Cell of origin	function
<b>PDGF</b> "Platelet derived growth factor"	-platelet -macrophage -endoth. cell	-cell chemotaxis -stim. Angiogenesis -stim. Wound contraction Mitogenic for fibroblast
<b>TGF</b> "Transforming growth factor"	- Macrophage - T lymphocyte -Platelet	Cell chemotaxis -mitogenic for fibroblast
<b>EGF</b> "Epith. Growth factor"	Platelet Macrophage.	-stim. Angiogenesis -Mitogenic for fibroblast

# Factors Affecting Wound Healing



LOCAL FACTORS



GENERAL FACTORS  
(SYSTEMIC )

# General factors :

Age

Anaemia

Malnutrition: -Hypoprotinaemia

vitamin c

Ca + Mg

Drugs: Steroid , cytotoxic

Malignancy

Metabolic disorders :Diabetis  
,Uraemia

# LOCAL FACTORS :

Blood supply

Hypoxia

Infection

Hematoma

Mechanical  
stress

Type of tissue

Surgical  
technique

Suture material

# COMPLICATIONS OF WOUND HEALING



Infection : delays healing by causing Tissue damage



Wound Dehiscence



Pathological Fibrosis, result from increased collagen production and decreased collagen degradation

- a. Hypertrophic scar
- b. Keloid
- c. Widened scar

# Wound dehiscence



\*It is partial or complete separation of previously approximated wound edges due to failure of proper wound healing



\*Occurs 3-10 days after surgery due to poor blood supply or infection or malnutrition...



# Hypertrophied scar



It occurs in wounds whose healing was delayed



The scar remains in the remodeling phase for longer than usual



It is more cellular & more vascular than mature scar



Clinically the scar is red, raised, itchy, and tender





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# Keloid :



There is an extreme overgrowth of scar tissue beyond the limit of the original wound with no tendency to resolve



There is a decrease in collagen degradation



The levels of the collagenase inhibitor alpha-2 macroglobulin have been shown to be decreased in keloid lesions





## Comparision of Hypertrophic & keloid scars

<b>Features</b>	<b>Hypertrophic scsr</b>	<b>Keloid scar</b>
<b><i>Genetic</i></b>	Not familial	May be familial
Race	Not race related	Black>white
Sex	F = M	F > M
Age	children	10-30 y
Borders	Remains within wound	Out grow wound area
Natural history	Subsides with time	Rarely subside
site`	Flexor surfaces`	Sternum, shoulder
Aetiology	Related to tension	unknown

## Widened scar :

- Possibly result from wound edge separation with tension perpendicular to the healing skin wound.



## Notes ..Wounds healing in fetuses

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Wounds occurring in fetuses of early gestational age can heal without any scar formation

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presence of fewer neutrophils and more monocytes during the inflammatory period



a greater proportion of  
type III collagen in  
contrast to adult wounds



Collagen deposition in  
fetal wounds displays a  
fine reticular pattern



fibronectin is more  
abundant in fetal wounds  
and has been noted to  
accelerate wound healing in  
fetal rat models

# Summary



- WOUND HEALING OCCURS IN FOUR OVERLAPPING PHASES



- MULTIPLE CELLS, CYTOKINES, AND GROWTH FACTORS INVOLVED



- MANY SYSTEMIC AND LOCAL FACTORS CAN IMPAIR HEALING



- CLINICAL KNOWLEDGE ESSENTIAL FOR PATIENT CARE

Thank you

