Wound Healing, Scar & Pressure Sores

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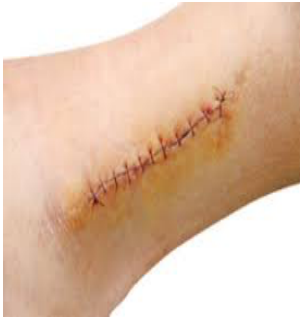
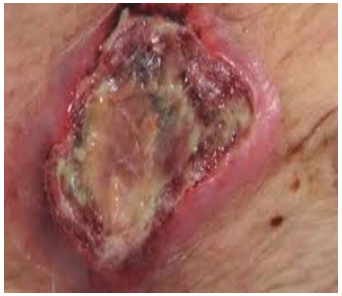
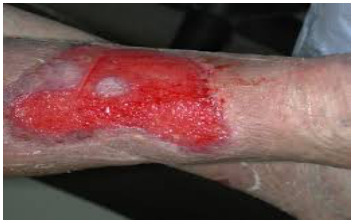
Objectives

- Basic Principles
- Classification
- Classes of operative wound
- Phases of wound healing
- Collagen Types
- Factors affecting wound healing
- Scars
- Pressure sores
- Burn








QUIZ
TIME!

The following are the sequence of events and phases taking place in wound healing?

1- Remodelling, epithalization,& contracture

2- inflammatory, proliferative & remodeling

3- vasocontriction,epithalization & contracture

4- proliferative, remodelling and wound synthesis

The major cell/s involved in the proliferative phase is/are

- 1- PMN
- 2- Fibronectin
- 3- Fibroblast
- 4- collagen

The difference between secondary & partial thickness healing is :

- Contracture only in secondary healing
- Contracture only in partial thickness healing
- Epithilisation only in secondary
- Epithalization only in parial thickness healing

Perforated gastric ulcer or Perforated appendicitis is/are

- Clean
- Clean-contaminated
- Contaminated
- infected

All of the following is/are charachteristic of ideal scar except:

- Painful
- Flat
- No restriction of movement
- Adequate color

All of the following is characteristic of keloid:

- 1- crosses border
- 2- genetic predisposition
- 3- common in earlobes/ chest in African
- 4- All of the above

Preventive measures of HTS is/are:

- 1- adequate suture bite closure
- 2- Free tension closure
- 3- non-strangulated sutures
- 4- minimal undermining/ electroqautery
- 5- all of the above

Wound:

- disruption of normal anatomical structure and function
- Classified as acute vs. chronic

Definition

- Restoration of integrity and continuity of injured tissue to re-establish homeostasis of that tissue and to stabilize the entire organism's physiology
- Wound healing requires the coordinated completion of a variety of cellular activities, including phagocytosis, chemotaxis, mitogenesis, synthesis of collagen and extracellular matrix components

Classification

By type	By timing	By abnormal healing
1- primary 2- delayed primary 3- secondary 4- partial-thickness wound healing	1- Acute 2-Chronic	1. overgrowth (hypertrophic, keloid) 2. undergrowth (chronic unstable wound) 3. abnormal pigmentation 4. contour abnormality

Classification of Wounds Closure

- **•Primary healing (1° intention)**
  - Primary closure
  - Within hours of repairing full-thickness surgical incision
  - Results in mortality of minimal number of cellular constituents
-
- **Secondary healing (2° intention)**
  - Wound left open to heal by processes of granulation, contraction, and epithelialization
  - Results in more intense inflammatory response
  - Larger quantity of granulation tissue with pronounced contraction of wounds
-
- **Tertiary healing (3° intention)**
  - Delay primary closure
  - Desired for contaminated wounds
  - Phagocytosis of contaminated tissues well underway by 4^{th} day
  - Foreign materials walled off by macrophages

Epithelial Repair

- epithelial continuity is reestablished across a wound

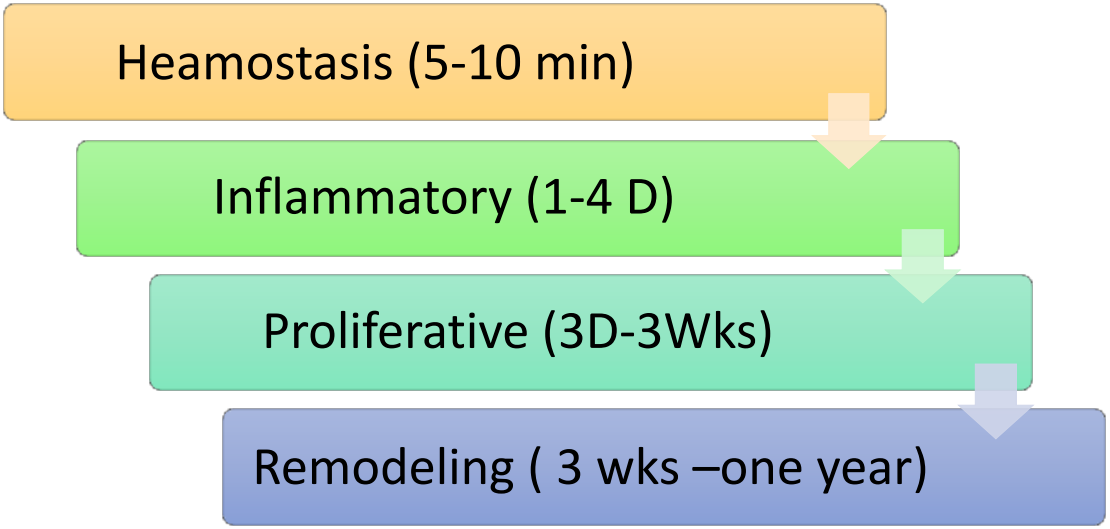
1-mobilization

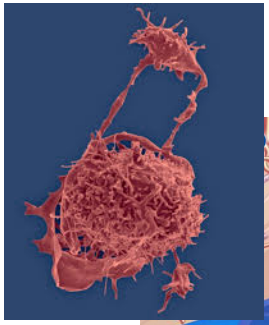
2-migration (stimulus is loss of contact inhibition)

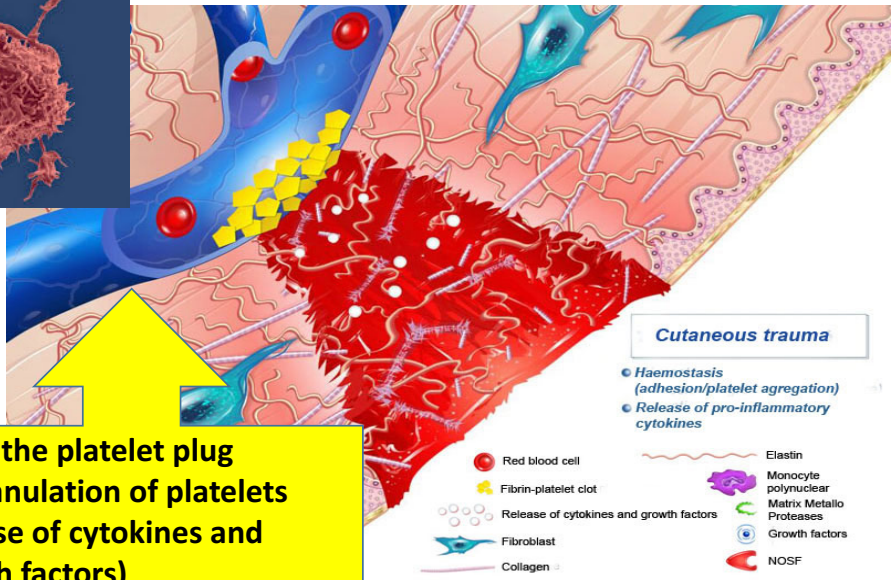
3-mitosis

4-cellular differentiation

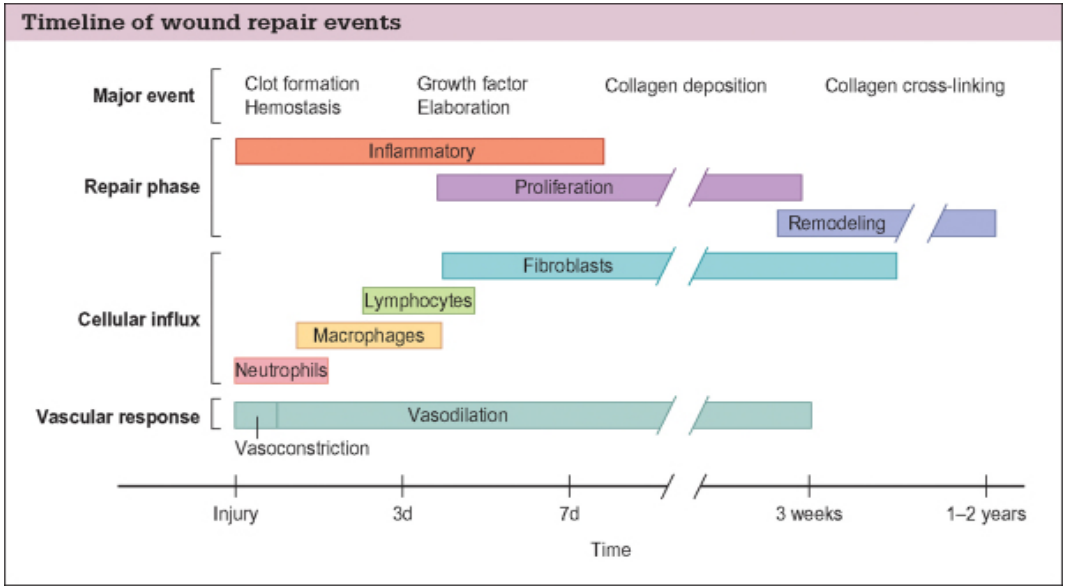
PHASES

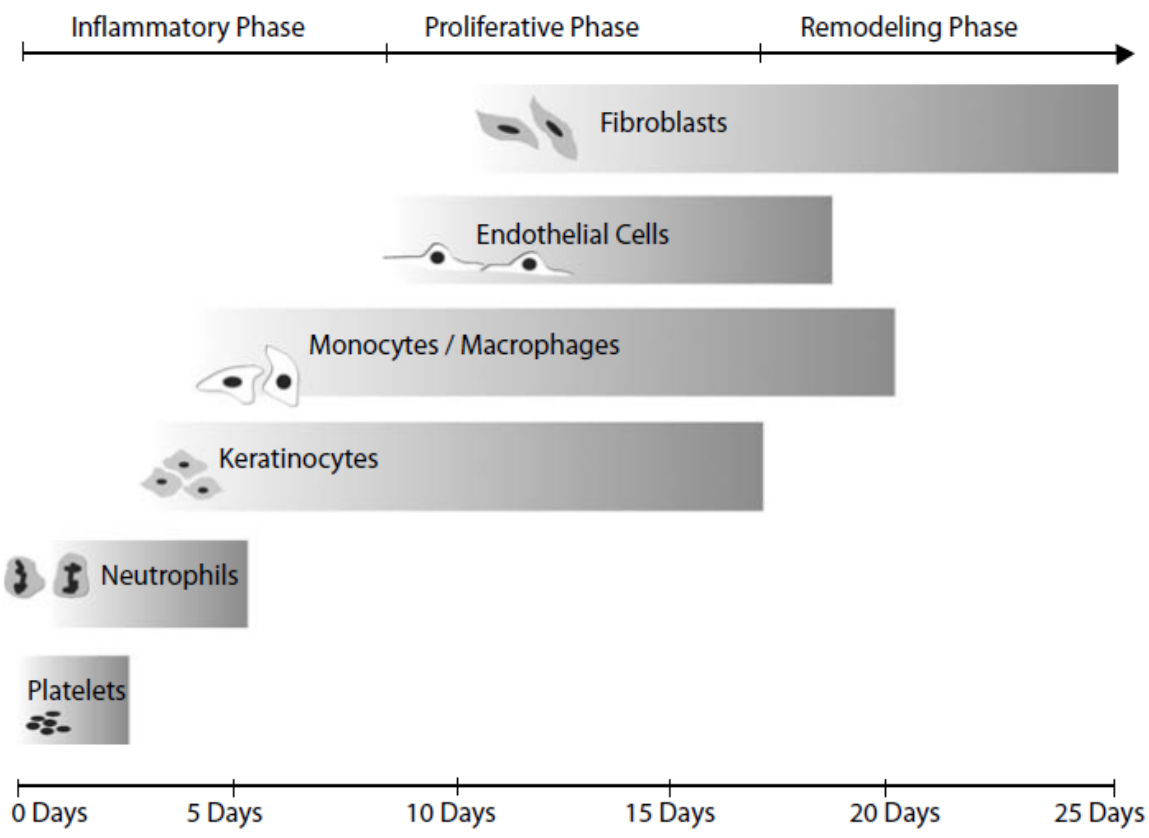

Heamostasis (5-10 min)
Inflammatory (1-4 D)
Proliferative (3D-3Wks)
Remodeling (3 wks –one year)





.Form the platelet plug
.Degranulation of platelets (release of cytokines and growth factors)
.Activation and recruitment of neutrophils

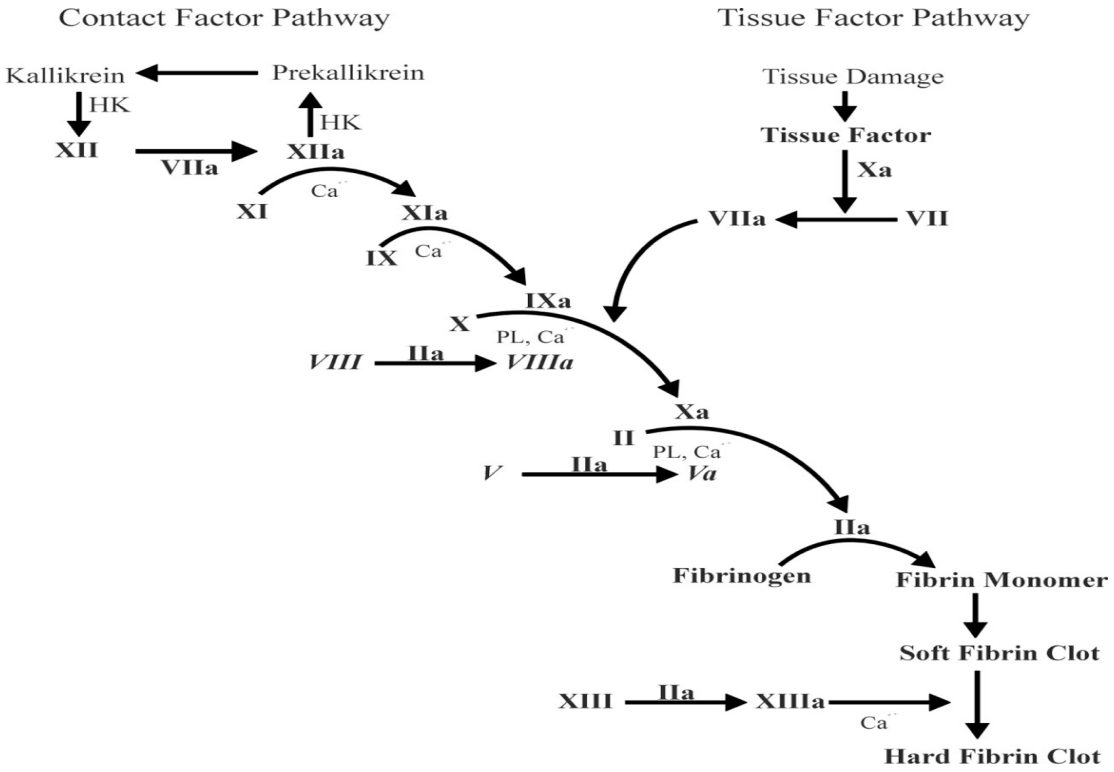

Timeline of wound repair events
Major event
Clot formation
Hemostasis
Growth factor
Elaboration
Collagen deposition
Collagen cross-linking
Repair phase
Inflammatory
Proliferation
Remodeling
Cellular influx
Fibroblasts
Lymphocytes
Macrophages
Neutrophils
Vascular response
Vasodilation
Vasoconstriction
Injury
3d
7d
3 weeks
1–2 years
Time


Inflammatory Phase
Proliferative Phase
Remodeling Phase
Fibroblasts
Endothelial Cells
Monocytes / Macrophages
Keratinocytes
Neutrophils
Platelets
0 Days
5 Days
10 Days
15 Days
20 Days
25 Days

Haemostasis

- Initial response to injury = constriction
- Platelet plug forms after adherence to exposed subendothelial collagen via vWF
- Platelets degranulate releasing: ADP, thromboxane-A2, bradykinin, and 5-HT → further vasoconstriction and platelet aggregationPlatelets stimulated to release

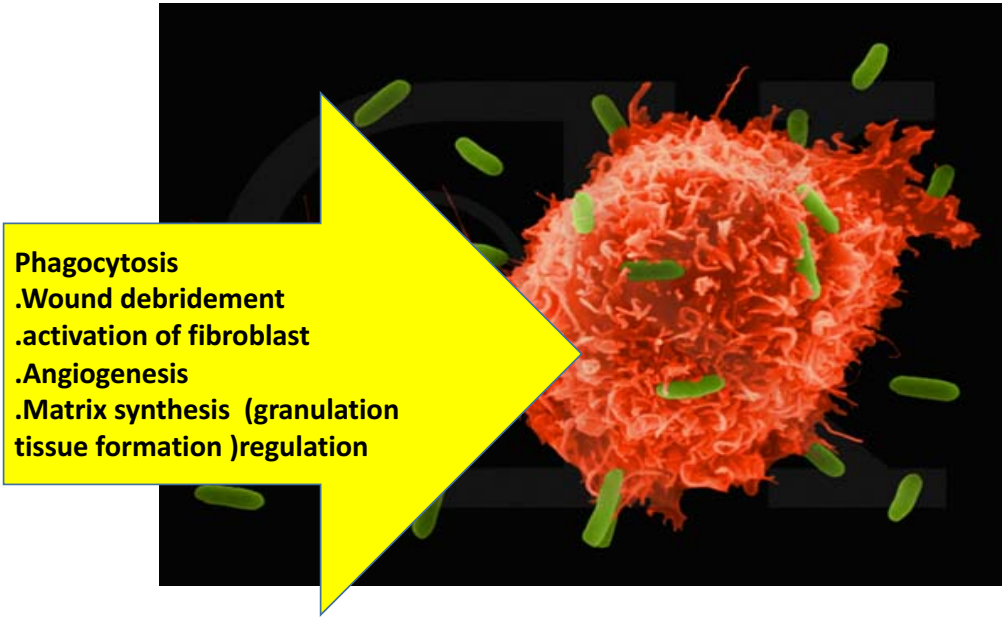
- ○ **platelet derived growth factor (PDGF)**
- -made by macrophages, endothelial cells, fibroblasts
- -chemotaxis, fibroblast stimulation
- ○ t**ransforming growth factor β (TGFβ)**
- -made by macrophages, platelets, fibroblasts
- -fibrinogenesis, angiogenesis, chemotaxis, immune suppression
- ○ **fibroblast growth factor**
- -made by macrophages and endothelial cells
- -angiogenesis and chemotaxis


Contact Factor Pathway
Tissue Factor Pathway
Kallikrein
Prekallikrein
HK
HK
XII
VIIa
XIIa
XI
Ca++
XIa
IX
Ca++
IXa
X
PL, Ca++
VIII
IIa
VIIIa
Tissue Damage
Tissue Factor
Xa
VIIa
VII
Xa
II
PL, Ca++
V
IIa
Va
IIa
Fibrinogen
Fibrin Monomer
Soft Fibrin Clot
XIII
IIa
XIIIa
Ca++
Hard Fibrin Clot

Inflammatory phase/ Migratory phase

- Classically represented by:
  - Rubor (redness)
    - caused by vasodilation
    - primarily result of prostacyclin (PGI_2) and histamine, also caused by prostaglandin A, D, and E (PGA, PGD, PGE)
  - Tumour (swelling)
    - caused by leakage of plasma proteins through gaps in vascular endothelium
    - edema potentiated by PGE_2, prostaglandin $F_{2\alpha}$ ($PGF_{2\alpha}$)
  - Calour (heat)
    - Increased local temperature secondary to both increased blood flow and elevated metabolic rates
  - Dolour (pain)

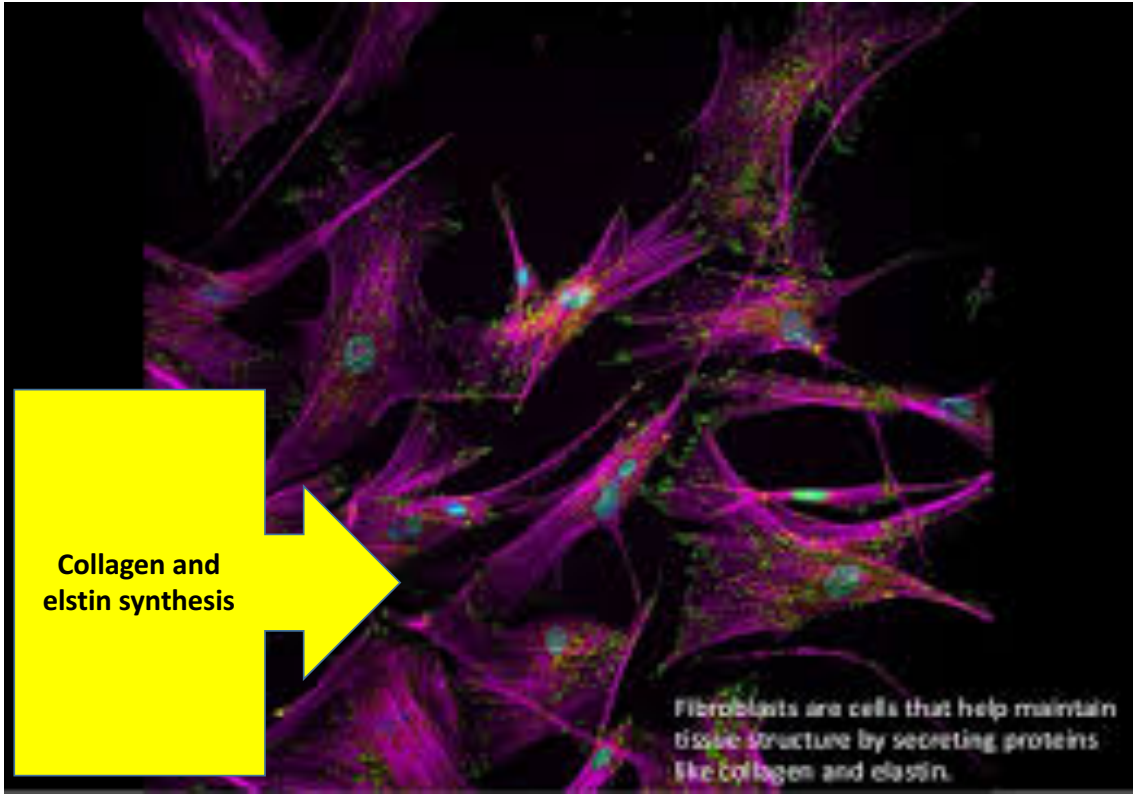
Macrophages



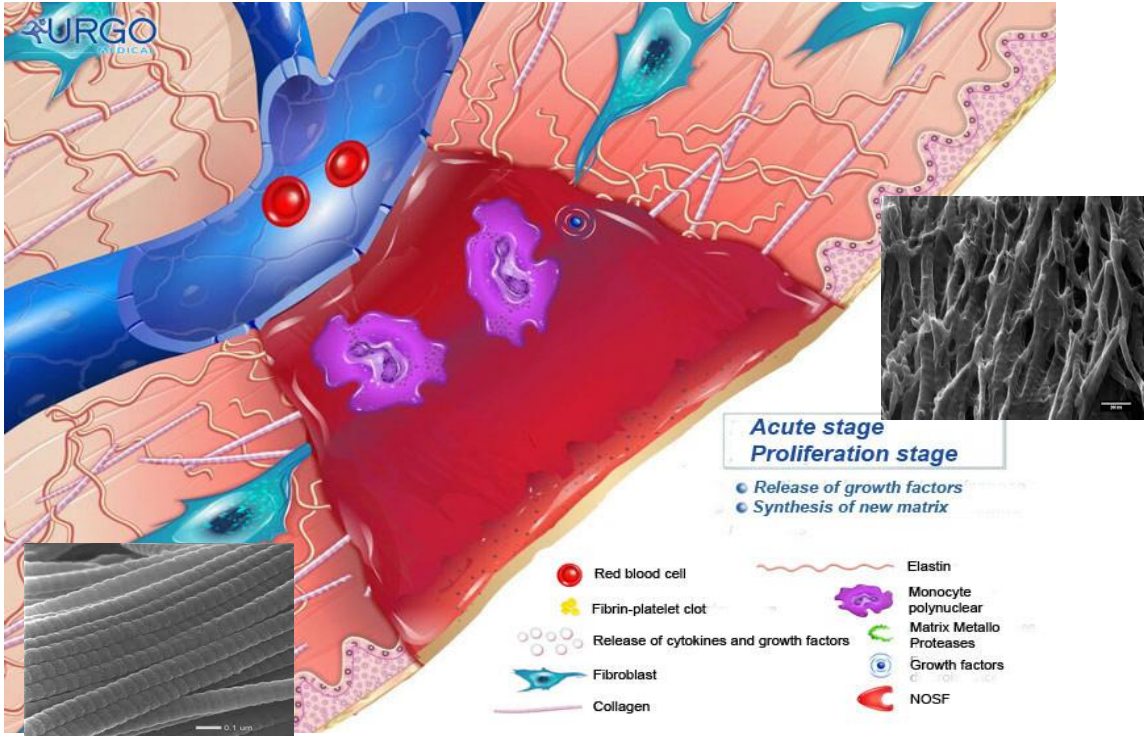
Proliferative Phase (fibroplasia

- Begins 2-3 days after wounding
- Signalled by arrival of **fibroblasts**
  - Driven by macrophage-derived **bFGF**, **TGFβ**, **PDGF** to proliferate and synthesize **glycosaminogylcans (GAGs)** and **proteoglycans** (building blocks of new extracelluar matrix of granulation tissue and collagen)
  - Also produce **bFGF**, **TGFβ**, **PDGF**, **keratinocyte growth factor**, **insulin-like growth factors-1**
  - Dominant cell type peaking at 7-14 days
- Collagen synthesis (net production for next 3-6 weeks)
- ↑ Keratinocyte mitosis
- ↑ Endothelial cells
- ↑ Angiogenesis (from vessels at wound margins)
- Lasts 2-4 weeks depending on site and size of wound with slowing of fibroblast migration and proliferation

Fibroblast


Collagen and elstin synthesis
Fibroblasts are cells that help maintain tissue structure by secreting proteins like collagen and elastin.

Proliferative phase



Maturation (remodelling)

- Begins approx. 3 weeks after injury
- Collagen synthesis and degradation are accelerated but in equilibrium with collagen breakdown (no net increase in collagen content)
- Collagen deposition peaks by 3rd week
- Large capillaries growing into wound regress/disappear
- Indurated, raised, pruritic scar becomes mature scar
- Collagen fibers become organized
- Type III collagen replaced by type I collagen

- ○ re-establishing normal 4:1 ration (I:III)
  - Duration depends on age, genetics, type of wound, location (1-2 years)
- Tensile strength increases to 80% of pre-injured skin

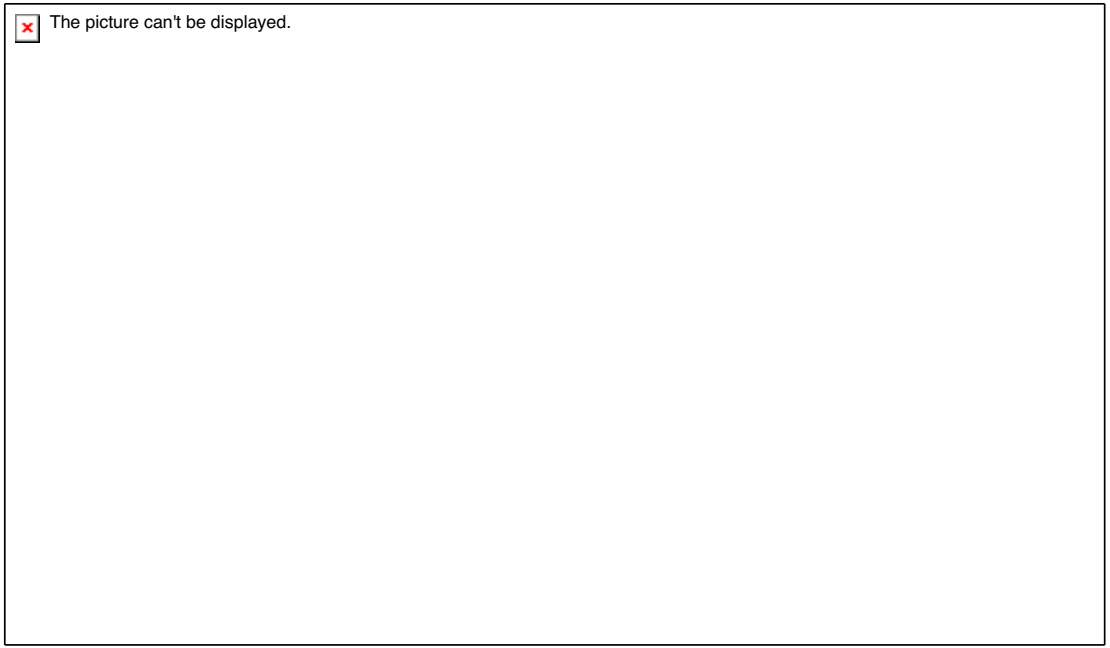
Collagen

- ***Left handed helix involving 3 polypeptides***
- Most abundant family of proteins in the human body (30%)
  - > 19 types of collagen have been identified
  - ***Type I collagen is the major structural component of bones, skin, and tendons***
  - ***Type II collagen is found predominantly in cartilage***
  - ***Type III collagen is found in association with type I collagen in varying ratios depending on the type and maturity of tissue (predominant type in granulation tissue)***
  - ***Type IV collagen is found in the basement membrane***
- ***Type V collagen is found in the cornea***

- ***Wound Strength is 80% of original after remodelling***
- ***Lysine and proline hydroxilation required for cross linkage.***

- Differs in relative composition of **hydroxylysine** and **hydroxyproline** and cross-linking
  - Type I ≅ 90% of all collagen in body
  - Normal skin ratio - Type I:Type III = 4:1
  - Hypertrophic / immature scare 2:1 ratio

- Formation inhibited by:
- Colchicine
- Penicillamide
- Steroids
- Vit. C deficiency
- Fe deficiency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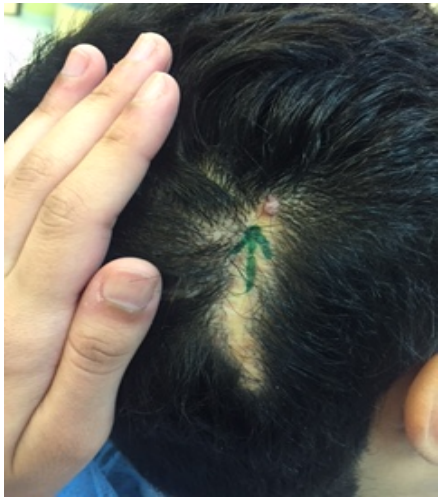
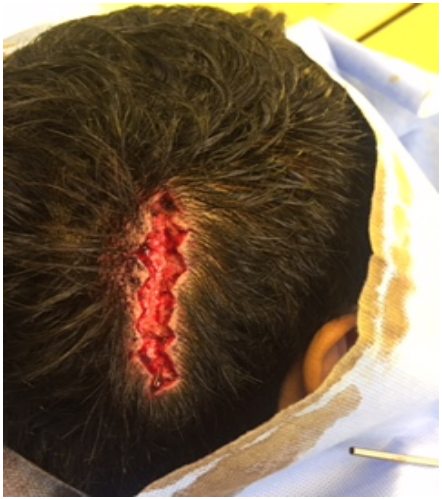
Types of Surgical wounds

- ***Clean:*** nontraumatic, noninfected wounds & no breach of Resp, GI, or GU tract
- ***Clean-contaminated***: small breach in protocol; Resp/GI/GU tract are entered with minimal contamination
- ***Contaminated:*** fresh traumatic wounds; major break in sterile technique, nonpurulent inflammation; in or near contaminated skin
- ***Infected:*** purulent infection

Factors affecting wound healing

General	Local
Nutrition	Oxygen
Drugs/Toxins	Infection
Age	Acidity
DM	Radiation
Smoking	Loss of growth factors
Vascular disease	Denervation
Obesity	Iatrogenic
Systemic diseases	Edema
Idiopathic	Cancer
Inherited diseases	Foreign body



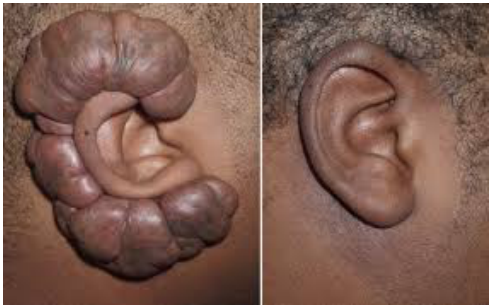


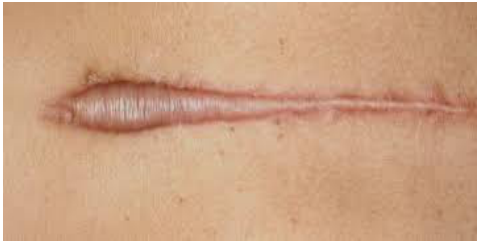
Acute wound

- Cleansing
- Exploration and diagnosis
- Debridement
- Tetanus immunization status
- Replacement of lost tissues where indicated
- Skin cover if required
- Dressing
- Skin closure without tension

Abnormal Scars

- Keliod Vs. Hypertrophic scars Vs. Wide scar





Scar

- ***ideal scar:***
- • Flat, Narrow
- • good colour & contour match to surrounding skin
- • parallel to *or within resting skin tension lines (RSTL)*
- • pliable
- • does *not restrict function or distort normal anatomy*
- • matures within 6-18 months
- • asymptomatic

Vancouver Scale

Characteristic	Score	Description
Pigmentation (P)	0	Normal: color that closely resembles the color over the rest of one's body
	1	Hypopigmentation
	2	Hyperpigmentation
Vascularity (V)	0	Normal: color that closely resembles the color over the rest of one's body
	1	Pink
	2	Red
	3	Purple
Pliability (P)	0	Normal
	1	Supple: flexible with minimal resistance
	2	Yielding: giving way to pressure
	3	Firm: inflexible, not easily moved, resistant to manual pressure
	4	Banding: rope-like tissue that blanches with extension of the scar
	5	Contracture: permanent shortening of scar producing deformity or distortion
Height (H)	0	Normal: flat
	1	<2 mm
	2	<5 mm
	3	>5 mm

*Each scar characteristic is assessed and assigned a score as shown; the summation of these scores then forms the overall scar score.

Comparison of Keloid and Hypertrophic scars

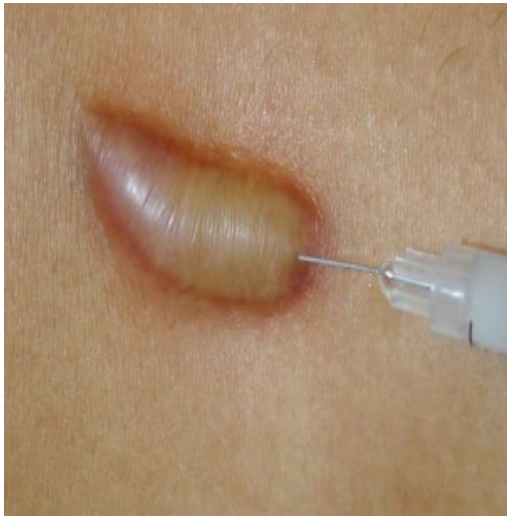
Table 3.4 Comparison of hypertrophic and keloid scars

Features	*Hypertrophic scar*	*Keloid scar*
Genetic	Not familial	May be familial
Race	Not race related	Black > white
Sex	Female = male	Female > male
Age	Children	10–30 years
Borders	Remains within wound	Outgrows wound area
Natural history	Subsides with time	Rarely subsides
Site	Flexor surfaces	Sternum, shoulder, face
Aetiology	Related to tension	Unknown

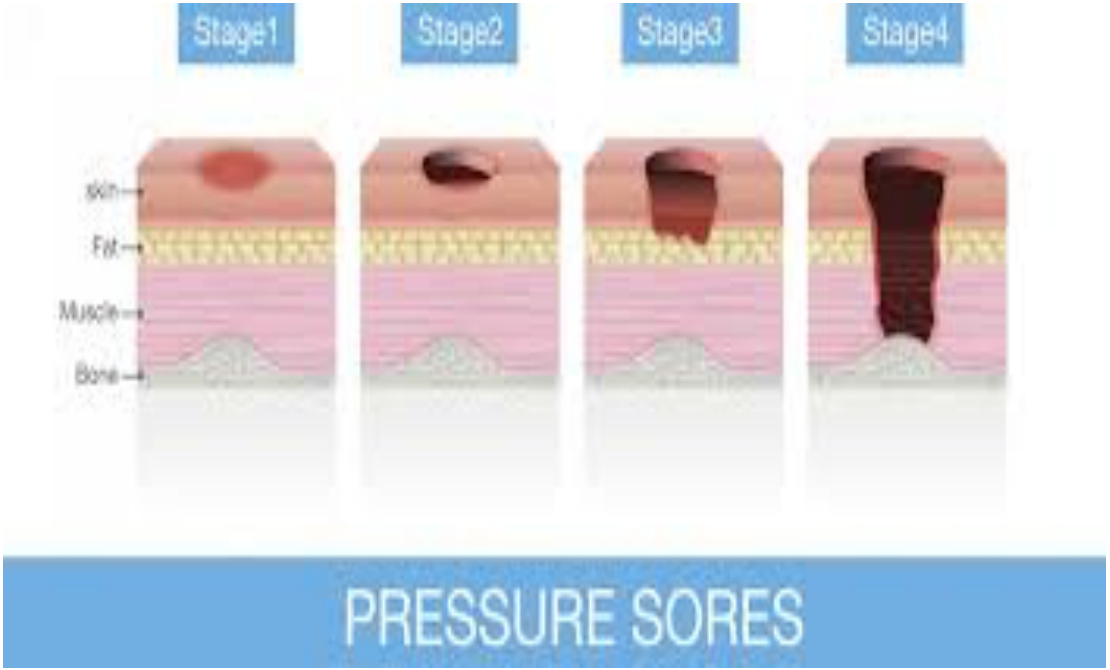
Medical Quick Review of Basics
http://pgmcqs.com/tag/all-india-mdms-entrance-exams-mcqs/page/7/

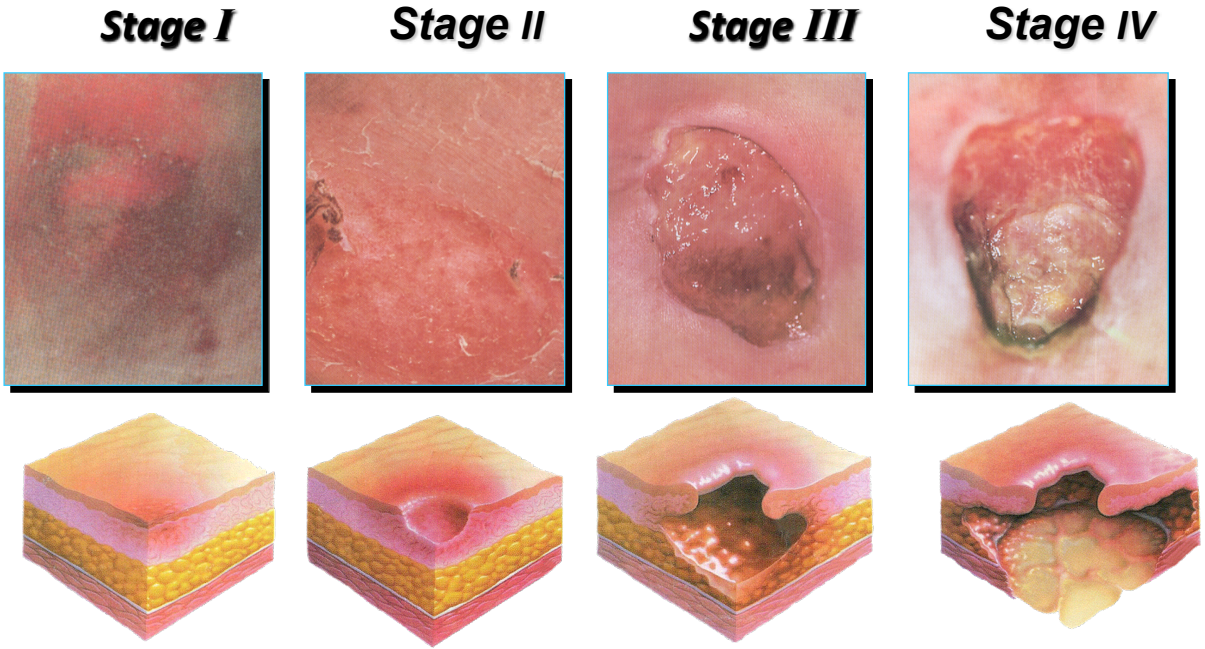
Treatment of HTS !!

- Pressure
- Silicone
- Preventation
- 5-FU
- Steroids
- Radiation
- Laser
- Surgery



Pressure Sores




Stage I
Stage II
Stage III
Stage IV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köszönöm תודה! dėkuji
mahalo 고맙습니다
thank you
merci 谢谢 danke
Ευχαριστώ شكرا
どうもありがとう gracias

QUESTIONS ?

Case Module Discussion