L4

Wound Healing & management

Objectives:

- Basic Principles
- Classification
- Classes Operative wound
- Phases of wound healing
- Collagen types
- Factors affecting wound healing
- Scars
- Pressure sole
- Burn

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Important
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Skin

- **Largest organ** in the body

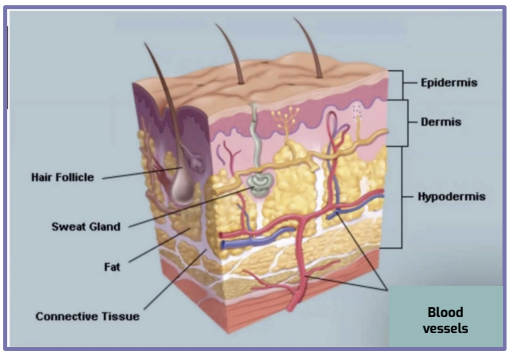
Very Important in tactile (Touch) Function

- **Functions:**
  - Thermoregulation
  - Protection (Immunity)
  - Prevention of water loss
  - Sensation
  - Aesthetic

Depends on the skin site:

1. **Subcutaneous fat**
2. **Fascia**
3. **Dermis**
4. **Border Layers of fatty tissue in the Abdomen & Thigh**
5. **Glands in Breast immediately under the skin**

The deeper structures like muscles and bones

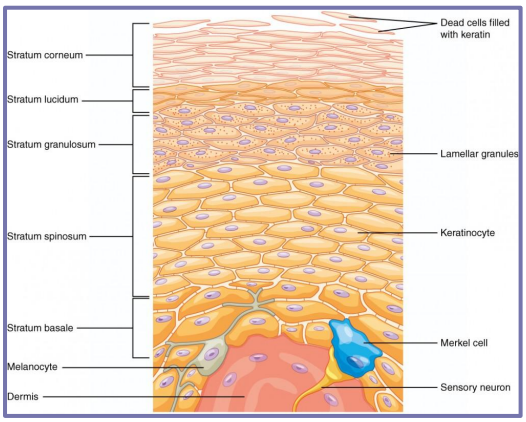


Made by different Layers:
1- Dermis
2-Epidermis
3-Hypodermis

We have multiple layers in the Epidermis:

Starting from the basal layer going up we have the surface:

- **Stratum Basale**
- **Stratum Spinosum**
- **Stratum Granulosum**
- **Stratum lucidum**
- **Stratum Corneum → Very imp to know that the cells in this layer are NOT nucleated, so whenever you see a nuclei in these cells think of: Inflammatory conditions in the skin or psoriasis** (skin condition that causes flaky patches of skin which form scales)**or Malignant changes**



We have 2 Layers of Dermis:

- **Reticular Dermis:** some of them lay deeply
- **Superficial Papillary dermis:** Hair follicles (Majority)

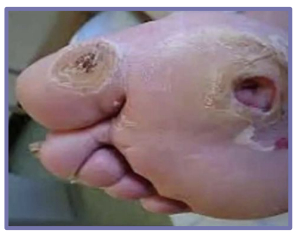
The most Imp layer in the epidermis:

- **Stratum basale or Germinative layer :** Because from this layer the rest of the epidermis layers originate

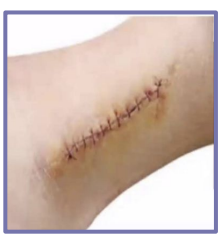
The most Imp cells in this layer is Melanocyte - Nerve ending

The basement Membrane that separate dermis from epidermis (it is very imp landmark) in terms of measuring the depth of wound and burn

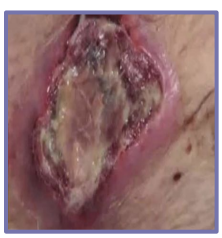
Imp to differentiate which type of wound you are dealing with + identify the cause of the wound → in order to treat it effectively



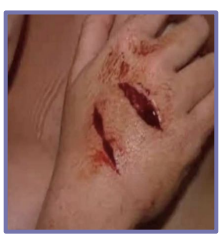
Chronic diabetic wound due to neuropathy + Compromised Vascularity



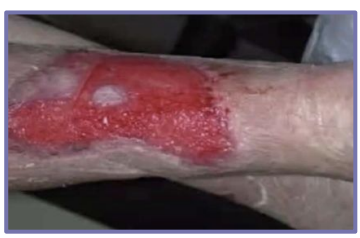
Surgical wound suture



Infected wound Pressure sole



Abrasions due to Tension also called Traumatic Wound



Chronic wound due to infection + Trauma

Vascular insufficiency in the peripheral vessels

Collagen

- Left handed helix involving 3 polypeptides.
- Most abundant family of proteins in the human body (30%).
- Over 19 types of collagen have been identified

Types of collagen:

Type I: is the major structural component of **bones, skin, tendons** Muscles.

Type II: is found predominantly in **cartilage**.

Type III: is found in association with type I collagen in varying ratios depending on the type and maturity of tissue (predominant type in **granulation tissue**) [1]

Type IV: collagen is found in the **basement membrane** of arteries, nerves, & tendons.

Type V: is found in the **cornea**.

- Wound Strength is 80% of original after remodelling. (Healing)
- Lysine and proline hydroxylation required for cross linkage. (The main step in collagen synthesis)[2]
- 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 scar 2:1 ratio
- **Formation inhibited by:**

Colchicine(used in hypercalcemia, Gout), penicillamine(immunosuppressant), steroid, Vit.C deficiency and Fe deficiency(affect cross linkage of collagen).

They activate collagenase which degrades collagen synthesis and inhibits cross linkage hydroxylation of lysine and proline.

1. E.g. in traumatic wounded skin (associated with healing). Can also be found in newborns.
2. Biochemistry: collagen synthesis events: procollagen gets converted to collagen by proline and lysine hydroxylation (essential component to have complete collagen synthesis).
3. Vit c deficiency → problems in healing → less collagen in basal membrane

Growth Factors in Wound healing

Growth Factor	Abbreviation	Source	Functions
Platelet-derived growth factor	PDGF	Platelets, keratinocytes, fibroblasts, endothelial cells , perivascular cells	Fibroblast proliferation, chemotaxis &collagen metabolism; angiogenesis
Transforming growth factor-B	TGF-B	Platelets, keratinocytes, fibroblasts, endothelial cells, macrophages	Fibroblast proliferation, chemotaxis &collagen metabolism; angiogenesis; immunomodulation
Transforming growth factor-a	TGF-a	Platelets, keratinocytes	Keratinocyte proliferation &migration
Epidermal growth factor	EGF	Platelets	Keratinocyte proliferation &migration
Interleukins	IL-1, IL-10	Leukocytes, keratinocytes	Fibroblast proliferation; proinflammatory (IL-1); anti-inflammatory (IL-10)
Tumor necrosis factor	TNF	Leukocytes, keratinocytes	Promotes inflammation
Connective tissue growth actor	CTGF	Fibroblasts, endothelial cells	Fibroblast proliferation, chemotaxis &collagen metabolism
Fibroblast growth factor	FGF	Keratinocytes, macrophages	Fibroblast & epithelial cell proliferation; matrix deposition, wound contraction: Angiogenesis
Keratinocyte growth Factor	KGF	Fibroblasts	Keratinocyte proliferation
Insulin-like growth actor 1	IGF-1	Fibroblasts	Keratinocyte proliferation
Hepatocyte growth	HGF	Fibroblasts, macrophages	Keratinocyte proliferation
Vascular endothelial	VEGF	Platelets, keratinoctes , macrophages , neutrphils	Angiogenesis

Wound

Definition

By disruption of normal anatomical structure and function. Wound is classified [1] as acute vs. chronic.

Wound healing:**The aim** is restoration of integrity and continuity of injured tissue to reestablish 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. identify what cells play major role in wound healing, so we can focus on them

Classification of Healing:

By type:

01

1. **Primary:**
   proximation of the edges and migrations of cells from edge to edge (horizontal). **wound heals directly**
2. **Delayed primary:**
   we wait for 2-4 days then we proximate by primary healing.
   wound edges are not approximated but after while we excise the granulation tissues
3. **Secondary:**
   Horizontal contraction by myofibroblast and epithelization.
   not approximated at all and this gap between 2 wounds edge is filled with granulation with scar.
4. **partial-thickness wound healing:**
   No contraction, only vertical epithelization.

By timing:

02

1. **Acute**
   (First week)
   (3 days)
2. **Subacute**
   (Lasts for 1-6 weeks)
   (up to 10 days)
3. **Chronic**
   (Lasts for >6 weeks)
   double time healing (20 days)

By abnormal healing:

03

1. **Overgrowth**[2]:
   (Hypertrophic vs. Keloid).
2. **Undergrowth:**
   (chronic unstable wound[3]).
3. **Abnormal pigmentation.**
4. **Contour abnormality.**

1. Wounds can be classified according to the mechanism of injury:
   Incised wounds: A sharp instrument causes these; if there is associated tearing of tissues, the wound is said to be lacerated
   Abrasions: These result from friction damage and are characterized by superficial bruising and loss of a varying thickness of skin and underlying tissue.
   Degloving injuries: These result from shearing forces that cause parallel tissue planes to move against each other: for example, when a hand is caught between rollers or in moving machinery. And others, Crush injuries, burns and gunshot wounds
2. Due to increased collagen production.
3. E.g. pressure sore, and diabetic wounds.
   - if the distraction of the epithelial continuity of the mucous membrane → **ULCER**
   - if it's occurred somewhere in the body it could be classified a **different pathology** than Wound
   - if there is a disruption in the architecture at the **LIVER** → **we don't call it wound**
   - if the distraction of the epithelial continuity in the ureter → we don't call it wound
   - there is a wrong terminology some people say → **Pressure Ulcer which wrong**! → the **right** terminology is **Pressure sole**

Wound

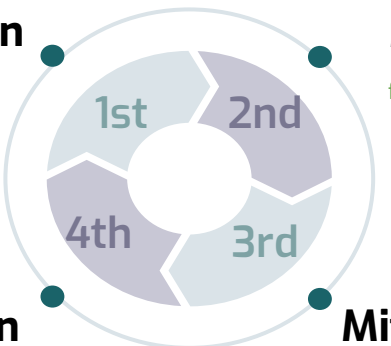
Classification of Wound Closure:

Primary healing (1st intention)	Secondary healing (2° intention)	Tertiary healing (3° intention)
• Primary closure by suturing the edges together. • Within hours of repairing full-thickness surgical incision. • Results in mortality of minimal number of cellular constituents. • Usually done In the first 24 hours • Ex, surgical wounds	• Wound left open to heal[1] by processes of **granulation**, **contraction**[2], and **epithelialization**[3]. • Results in more intense inflammatory response. • Larger quantity of granulation tissue with pronounced contraction of wounds.	• Delayed primary closure (24-48h) • Desired for contaminated wounds (infected wounds)[4] • Phagocytosis of contaminated tissues well underway by 4th day. • Foreign materials walled off by macrophages. • 3° intention healing describes the situation where a wound healing by 2nd intention (neglected traumatic wound/burn) is treated by excising its margins and then opposing them or covering the area with a skin graft.[5]

Epithelial Repair:

- Epithelial continuity is reestablished across a wound: Multiple events happen during the healing process. The cells (from the two edges of the wound) mobilize & migrate to the middle and get in proximity then duplicate by mitosis and differentiate to different cells.

Mobilization
of cells

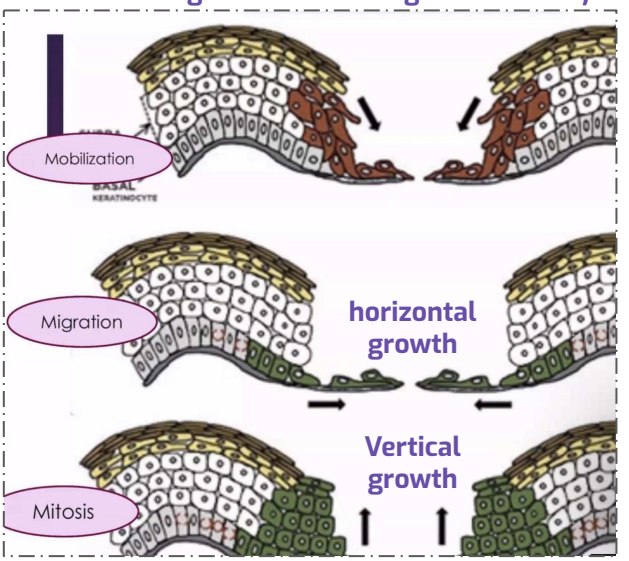


Migration
from edge to edge
(stimulus is loss of contact inhibition)

Cellular differentiation

Mitosis

mobilization of the epithelial cells from the periphery, these cells will migrate along the basement membrane then once these cells get contact with each other they stop horizontal growth and start grow vertically.



1. You keep the wound open (you do dressing and wound care but no suturing) and it heals by itself by contraction of myosin and actin, as well as epithelialization of dermis and epidermis, and it usually takes longer than primary healing.
2. Contraction results in edge to edge (horizontal) repair.
3. Epithelialization results in vertical repair.
4. Tertiary healing is a combination of primary healing and secondary healing. Clean the contaminated wound, wait for about 3 days and then re-approximate it by suturing.
5. The final cosmetic result may be better than if the wound had been left to heal by 2° intention.

Phases of Wound Healing

We talk about the major events occurring in each of the phases

01) Heamostasis[1] (5-10min): Insulting event

★ Main cells in this phase: platelets

- **Initial** response to injury = **constriction** followed by dilation
- Platelet plug forms due to activation of coagulation processes after adherence to exposed subendothelial collagen via vWF
- Platelets degranulate releasing: ADP, thromboxane-A2, bradykinin, and 5-HT → further vasoconstriction and platelet aggregation.
- Platelets stimulated to release :

platelet derived growth factor (PDGF)

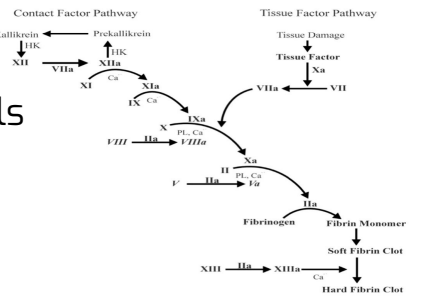
a. made by macrophages, endothelial cells, fibroblasts
b. chemotaxis, fibroblast stimulation after cell migration in the injured zone.

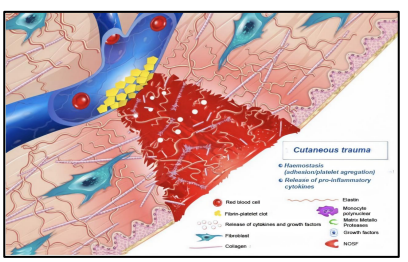
transforming growth factor β (TGFβ)[2]

a. made by macrophages, platelets, fibroblasts
b. fibrinogenesis, angiogenesis, chemotaxis, immune suppression or cellular response after the migration of cell in the injured zone

fibroblast growth factor

a. made by macrophages and endothelial cells
b. angiogenesis and chemotaxis after cell migration in the injured zone.





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

1. Considered as phase zero or part of the inflammatory phase. This phase is delayed by bleeding disorders.
2. It has a major role in wound healing. The main source for the production of TGFβ is the Alpha granules of platelets. Excess production of TGF-β isoforms causes abnormal scars (Hypertrophic and Keloid scars).
3. If this phase is longer → it indicates platelets related disorders → the patient will have bleeding tendency with healing problems.
4. platelets play a role in hemostasis, so any patients who use Anti-platelets like aspirin will have some source of wound healing disorders like hemophilia (coagulation disorders)must be under consideration

Phases of Wound Healing cont.

02) Inflammatory/Migratory "lag" phase1 (1-4 Days)

- Typically starts immediately after hemostasis.
- **Main cells in the inflammatory phase:** in the first 24 hours → **PMNs (neutrophils)**
- **After 24 hours → Macrophages.**
- **Which one is more important, neutrophils or macrophages?**

A patient who has neutropenia will have normal wound healing process, so the answer will be macrophages since they have role in chemotaxis in the release of interleukins and growth factors.

Classically represented by:

1- Rubor (redness) caused by:

- vasodilation
- primarily result of prostacyclin (PGI_2) and histamine, also caused by prostaglandin A, D, E (PGA, PGD, PGE)

2-Tumour (swelling):

- caused by leakage of plasma proteins through gaps in vascular endothelium
- edema potentiated by PGE_2, prostaglandin $F_{2\alpha}$ ($PGF_{2\alpha}$)

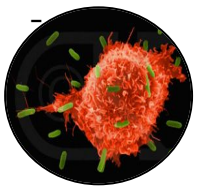
3- Calour (heat)

Increased local temperature secondary to both:

- Increased blood flow
- Elevated metabolic rates

4-Dolour (pain)

Macrophages :



- Phagocytosis
- Wound debridement
- activation of fibroblast
- Angiogenesis
- Matrix synthesis (granulation tissue formation) regulation

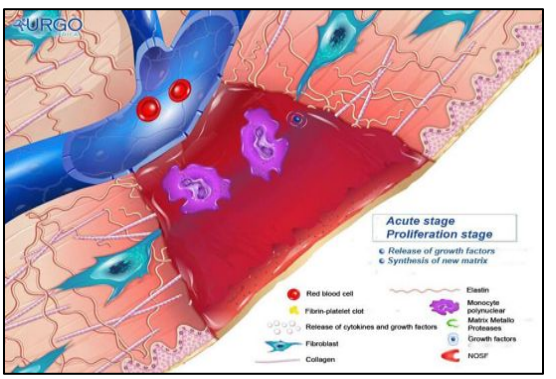
1. Characterized by an inflammatory response to injury, through an increased capillary permeability, proliferation of capillaries at wound edges and accumulation of protein-rich exudate preceding collagen synthesis

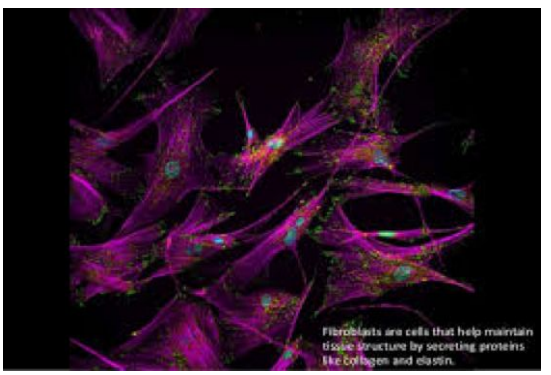
Phases of Wound Healing cont.

03) Proliferative/Fibroplasia "incremental" [1] (3 days - (3 weeks)

- Begins 2-3 days after wounding.
- **Signalled by arrival of fibroblasts (Main cells in the proliferative phase, they play a major role by deposition of extra-cellular matrix, including fibrin, collagen, and hyaluronic acid in collagen synthesis).**
  - Driven by macrophage-derived **bFGF**, **TGFβ**, **PDGF** to proliferate and synthesize glycosaminoglycans (**GAGs**) and **proteoglycans** (building blocks of new extracellular 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[2] (net production for next 3-6 weeks).
- ↑ Keratinocyte mitosis, ↑Endothelial cells and ↑Angiogenesis (from vessels at wound margins).
- Lasts 2-4 weeks depending on site and size of wound with slowing of fibroblast migration and proliferation.
- Different cells differentiate into different types and new blood vessels are formed (angiogenesis).

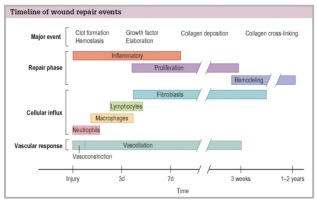




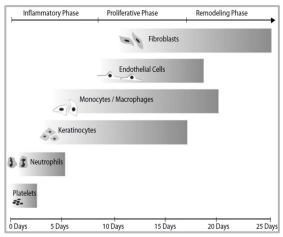
1. In this phase there is progressive collagen synthesis by fibroblasts and a corresponding increase in tensile strength with Increased collagen turnover. This phase is delayed by: Microvascular diseases (Diabetes), Macrovascular (Atherosclerosis).
2. Arterial oxygen tension (PaO2) is a key determinant of the rate of collagen synthesis.
3. Sometimes there is purulent discharge or association with serum discharge in every patient.
4. Clinically represented by a pale scar.

Phases of Wound Healing cont.

04) Remodeling /Maturation (3 weeks - one year)



- Begins approx. 3 weeks after injury
- Collagen synthesis and degradation are accelerated but in equilibrium with collagen breakdown (no net increase in collagen content)[1]
- 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** (most abundant) →**cross linkage of collagen fibers** to stabilize the wound.



- **re-establishing normal 4:1 ration (I:III)**:
  Duration depends on age, genetics, type of wound, location (1-2 years)[2].
- **Tensile strength increases to 80% of pre-injured skin[3]**

Factors Affecting Wound Healing: ★

Mnemonic: **DID NOT HEAL**

General (patient):

1. **N**utrition[4]
2. **D**rugs/**T**oxins[5]
3. Age[6]
4. **D**M
5. Smoking[7]
6. Vascular disease
7. **O**besity
8. Systemic diseases
9. **I**diopathic
10. Inherited diseases
11. Surgical technique[8]

Local (wound):

1. Oxygen (**H**ypoxia)
2. **I**nfection
3. **A**cidity
4. Radiation[9]
5. **L**oss of growth factors
6. Denervation (in diabetics)
7. **I**atrogenic
8. **E**dema
9. Cancer[9]
10. Foreign body

1. The major event in the remodeling phase.
2. Type three collagen is the main collagen present in the wound healing process. In abnormal wound healing (such as in hypertrophic scars and keloids) we have a higher ratio that could reach up to 1:30 so you'll have high collagen type 3 compared to the normal ratio, and you'll have abnormal scarring.
3. Especially in healing tendons, muscles, and blood vessels. For example for tendons it reaches up to 60-80%, but for normal wounds it usually reaches 100%.
4. Patients with pressure sore are usually malnourished therefore the healing process is usually delayed in such patients, Malnutrition has to be severe before healing is affected. Protein availability is most important, and wound dehiscence and infection are common when the serum albumin is low. Healing problems should be anticipated if recent weight loss exceeds 20%. **Vitamin C i**s essential for proline hydroxylation and collagen synthesis.
5. E.g. penicillin, steroids , cortisol and chemotherapy.
6. Elderly patients are more prone to have chronic wounds, they heal poorly because of impaired blood supply, poor nutritional status or intercurrent disease.however, they still form 'good' scars.
7. Vasoconstriction →reduced oxygen delivery, by shifting oxygen hemoglobin dissociation curve to the left
8. Dead spaces must be avoided, as the accumulation of blood and exudate encourage infection. Correct suturing of the deeper layers avoids dead space and often allows the skin edges to fall together without tension. 5/0 or 6/0 sutures are appropriate for the face, stronger ones (3/0 or 4/0) are needed for incisions near joints and still stronger ones for the abdominal wall
9. Chemo and radiotherapy lead to **fibrosis** of the skin as well as stenosis of the arteries.
10. **Types of collagen:**
    a. **Type I:** tendon and skin.
    b. **Type II:** cartilage, retina, and cornea.
    c. **Type III:** skin mainly.
    d. **Type IV:** basement membrane, some extend to cartilage.
    e. **Type V:** mainly cornea.
11. **collagen I** is the most abundant.

Classification of contamination in Surgical wounds
(WHO classification of wound)[1]

Clean: (class I)

- nontraumatic (elective), non infected wounds & no breach of Resp, GI, or GU tract.
- No spillage of the content of the tract itself.
- E.g. thyroid and breast surgeries.
- No need for antibiotics.
- Infection rate should be less than 1%.
- Use of Ab prophylaxis isn't
- recommended.

Clean-contaminated: (class II)

- Small breach in protocol; Resp/GI/GU tract are entered with minimal contamination.
- Non-elective surgery w/ very little contamination.
- Very minor spillage of the content.
- E.g. **cholecystectomy**, uncomplicated appendicitis, intestinal resection ONLY If there was no spillage.
- Infection rates in excess of 5% may suggest a breakdown in wards.

Contaminated [2] "Dirty": (class III)

- Fresh traumatic wounds; major break in sterile technique, **nonpurulent inflammation** (serious discharge from the wound); in or near contaminated skin.
- Major spillage.
- E.g. hemicolectomy or resection of the intestine with spillage, emergency surgery for perforated diverticular disease, or drainage of a subphrenic abscess.

Infected: (class IV)

- **Purulent infection**. Traumatic & severe wounds.
- They have positive culture and require broad spectrum antibiotics
- E.g. traumatic open bone fracture, and purulent pyogenic perforated appendicitis.

Acute Wound[3]:

- Cleansing
- Exploration and diagnosis[4]

- Debridement
- Tetanus immunization status[5]

- Replacement of lost tissues where indicated
- Skin cover if required

- Dressing[6]
- Skin closure without tension

1. To determine whether the administration of antibiotics is needed or not.
2. When wound contamination is anticipated, topical or systemic antibiotics can be used for prophylaxis .For example, systemic antibiotic is normally used to reduce the risk of infection during gastrointestinal surgery and when prosthetic material (hip joint, cardiac valves, arterial bypass) is inserted.
3. If it's a major trauma start with ATLS, if not start with vital signs. Don't jump to the wound directly, the patient should be stable, history needs to be taken, examination needs to be done, and then manage the wound.
4. To identify any risk factors that may interfere with wound healing.
5. In acute traumatic wounds, tetanus prophylaxis is routine.
6. proper protection against water loss and external environment

Ideal Scar:

01 - Flat
- Narrow
- Pliable

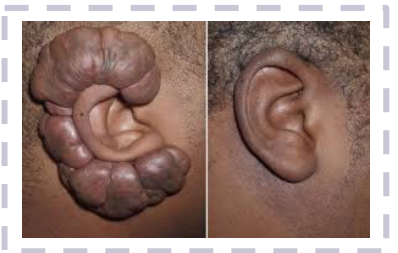
02 good colour and contour match to surrounding skin

03 parallel to or within resting skin tension lines (RSTL)[1]

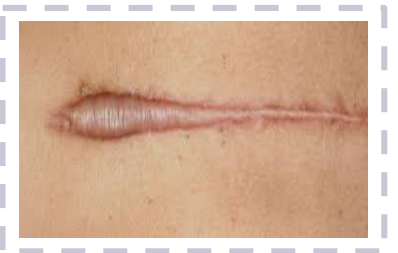
04 does not restrict function or distort normal anatomy [2]

05
- matures within 6-18 months
- Asymptomatic and painless

Abnormal Scars :



Keloid scar
excision can be done as an alternative.



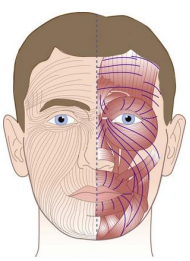
Hypertrophic scars
These scars should not be excised.

Wide scar
Caused by traumatic wounds that are not closed properly.

Important to remember this table

Features	Hypertrophic scar	Keloid scar[3]
Genetic	Not familial	May be familial
Race	Not race related	Black > White
Sex	Female = Male	Female > Male
Age	Children have tight skin	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

1. For example when you operate on the forehead (frontalis muscle), RSTL are perpendicular to the muscle fibers, therefore you should do the scar horizontally rather than vertically to decrease the tension, create less visible scars & decrease the risk of hypertrophic scar.
2. Wounds that cross a joint (hands, fingers, etc) are at high risk for causing functional defects due to contracture (myofibroblasts).
3. Classical presentation: 20 year old African American female, with a scar in the earlobe.
   - Keloid scars have high recurrence rate.



Vancouver Scale:

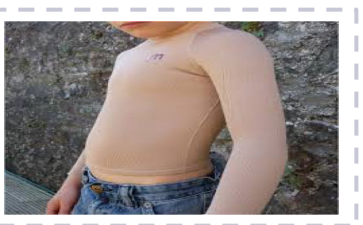
Scar characteristic	Description
Pigmentation	
0	Normal color that closely resembles the color over the rest of one's body
1	Hypopigmentation
2	Hyperpigmentation
Vascularity	
0	Normal color that closely resembles the color over the rest of one's body
1	Pink
2	Red
3	Purple
Pliability	
0	Normal
1	Supple: 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	
0	Normal: flat
1	<2 mm
2	<5 mm
3	>5 mm

Treatment of hypertrophic scar and keloid[1]:

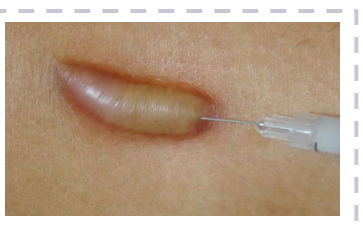
Prevention by: (in surgical wounds)
- No tension closure
- Minimal undermining of skin
- Adequate hemostasis

Non surgical:
- Pressure: compression garment.
- Silicone sheets or gels.

Medical [2]:
- 5-FU (Fluorouracil is a chemotherapy no after 14d).
- Intralesional Steroids (first-line therapy).



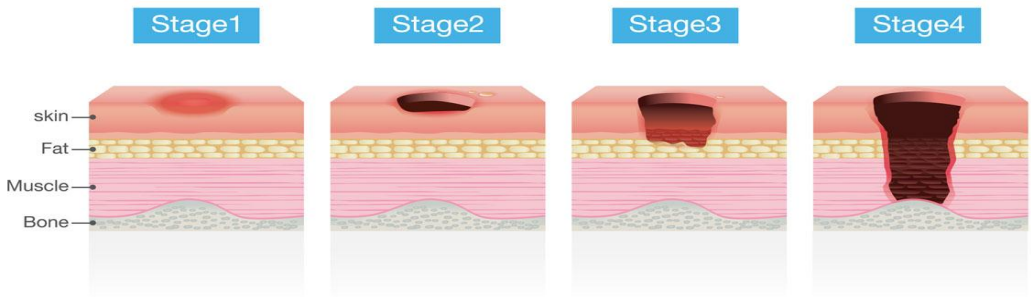
- Laser
- Radiation
- Surgery
- Debridement: remove necrotic, injured tissue. (If a patient comes with a wound that has been there >6 hrs, the wound was highly contaminated, or the surgeon was not able to completely remove the necrotic tissue, closing the wound would create a suitable environment for infection).

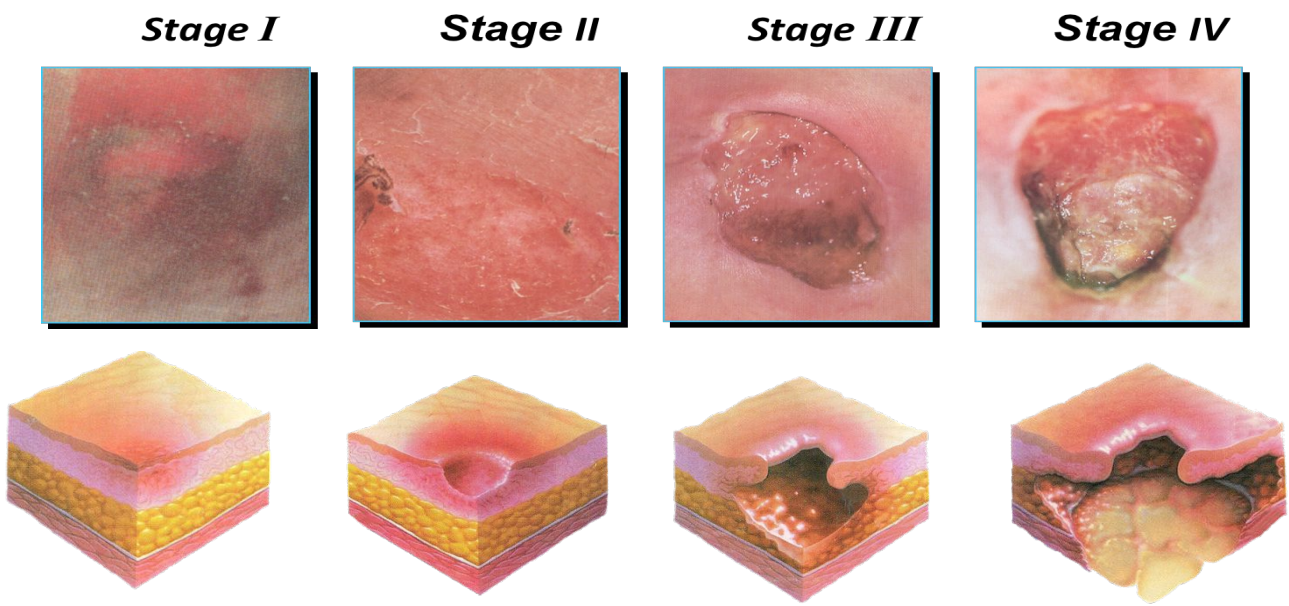


1. We should take measures to prevent it from occurring in the first place. You prevent HTS, by avoiding tension in creating a scar parallel to the RSTL, minimal undermining (raising the skin and going underneath it), minimal electrocauter (leads to seroma formation → high tension) closing layer, no strangulation (put spaces between each suture).
2. How does radiation and chemotherapy heal HTS if it leads to reduced wound healing? the unit of given radiation therapy is the guide. Patients that receive radiation therapy for cancer receive high amounts of radiation compared to HTS. The second thing is that HTS has abnormal collagen synthesis (high turnover of collagen) and radiation stops this by causing fibrosis and less formation of collagen.

- There's still a 10% chance of recurrence after treatment.

Pressure Sores, A.K.A Bed Ulcers:





- develops if the patient doesn't move while sleeping for 2 hours (bed sore).
- **mainly involves the epidermis.**
- produces erythema, avoided by changing the position every 30 min.

Involves the **epidermis and the upper dermis.**

Involves the **epidermis, dermis and upper fascia.**

Involves the fascia and can reach to the **muscle, tendons and bones** and they usually develop osteomyelitis.

- Stage 1 and 2 require non-medical intervention.

Summary 439

Recall

Q1: What inhibits wound healing?
Answer: Infection, ischemia, DM, malnutrition, anemia, steroids, cancer, radiation, and smoking.

Q2: How long a sutured wound epitheliazes?
Answer: 24 to 48 hours.

Q3: Define the following terms:

- Primary Wound Closure:

Answer: Suture wound closed immediately (a.k.a "first intention").

- Secondary Wound Closure:

Answer: Wound is left open and heals over time **without sutures** (a.k.a "secondary intention"); it heals by granulation, contraction, and epithelialization over weeks (leaves a large scar).

- Delayed Primary Closure

Answer: Suture wound closed 3-5 days **AFTER** incision (classically 5 days).

Q4: Define the following terms:

- Clean Wound:

Answer: Elective, non traumatic wound without acute inflammation; usually closed primarily.
Infection rate: <1.5%

- Clean-contaminated Wound:

Answer: Operation on the GI or respiratory tract without unusual contamination or entry into the biliary or urinary tract. Infection rate: <3%

- Contaminated Wound:

Answer: Acute inflammation, traumatic wound, GI tract spillage, or a major break in sterile technique.
Infection rate: ≈5%

- Dirty Wound:

Answer: Presence of pus, perforated viscus, or dirty traumatic wound.
Infection rate: ≈33%

Wound healing:

Classification		
By type	**By timing**	**By abnormal healing**
1. Primary 2. Delayed primary 3. Secondary Partial-thickness wound healing	1. Acute 2. Chronic	1. Overgrowth -hypertrophic - keloid 2. Undergrowth (chronic unstable wound) Contour abnormality)

Summary 439

Classification of Wound closure:

Primary 1st intention
Suture

- Primary closure
- Within hours
- Mortality of minimal number of cellular constituent

Secondary 2nd intention
No suture

- wound left open to heal by processes (granulation, contraction, epithelization)

Tertiary 3rd intention
Suture after 3 days

- delayed primary closure
- Contaminated wounds

Phases of wound healing:

Haemostasis 5-20 mins
aim to stop bleeding

Most important : Platelets

-Vascular constriction
-Platelet aggregation
-Degranulation
-Thrombus formation

-platelet stimulus to release: PDGF, TGF B, FGF

Inflammatory 1-4 d

-Neutrophil infiltration
-Monocyte infiltration and differentiation to macrophage
-Lymphocyte infiltration

- Clinically represented by :Rubor, Tumour, Calour, Dolour

Proliferative 3d-3wks

1- Re-Epithelization
2- Angiogenesis
3- Collagen Synthesis
4- ECM formation

Epithelial repair: mobilization, migration, mitosis, cellular differentiation

Remodeling 3wks-1y

1- Collagen Remodeling (collagen type lll replaced by type l until ratio of 1:4)

2- vascular maturation and regression

Scars:

01 Ideal scars:

- flat narrow
- Good color
- Parallel to RSTL
- Doesn't restrict function
- Asymptomatic
- Mature 6-18m

02 Abnormal scars:

- **Hypertrophic:** Common in children, remain with in wound, subside, flexor surface, related to TENSION.
- **Keloid:** Common in black, 10-30 yrs females, outgrow wound area; sternum, shoulder, face.
- **Wide:** Traumatic wounds that are not closed properly

Quiz!

Q1:25-year-old woman presents with a benign nevus on the right upper arm. She desires removal and undergoes a clean incision and then closure of the incision without complication. With regard to the healing process, which of the following cell types are the first infiltrating cells to enter the wound site, peaking at 24 to 48 hours?

A) Macrophages
B) Neutrophils
C) Fibroblasts

Q2: A 35-year-old woman undergoes an elective laparoscopic cholecystectomy for symptomatic cholelithiasis. Which of the following wound classes best describes her procedure?.

A) Class I, Clean
B) Class II, Clean/contaminated
C) Class III, Contaminated

Q3:A 25-year-old man is brought to the emergency room after sustaining burns during a fire in his apartment. He has blistering and erythema of his face, left upper extremity, and chest. He also has circumferential frank charring of his right upper extremity with decreased capillary refill. He is agitated, hypotensive, and tachycardic. Which of the following is the most appropriate initial management of his wounds?

A) Topical antibiotics should be applied to the burn wounds.
B) Excision of all third-degree burns.
C) Escharotomy of the right upper extremity.

Q4: A 60-year-old diabetic man undergoes incision and drainage of an infected boil on his back. The wound is left open and packed daily. Week by week, the wound grows smaller and eventually heals. Which of the following terms describes the method of wound closure by the patient?

A) Primary intention
B) Secondary intention
C) Tertiary intention

B) 4 / C) 3 / B) 2 / B) 1

Quiz!

Q5: A 22-yo African-American woman presents with a recurrent growth on her right thigh. She has a childhood history of a third-degree scald burn to the same area that did not require skin grafting. The growth was completely removed 2 years ago. On physical examination there is a, irregularly shaped purple lesion with a smooth top. Which of the following is the most likely diagnosis?

A) Keloid
B) Hypertrophic scars
C) Angiosarcoma

Q6: The following are the sequence of events and phases taking place in wound healing?

A) Remodelling, epithelization,& contracture
B) inflammatory, proliferative & remodeling
C) vasoconstriction, epithelization & contracture
D) proliferative, remodelling and wound synthesis

Q7: The difference between secondary & partial thickness healing is :

A) Contracture only in secondary healing
B) Contracture only in partial thickness healing
C) Epithelization only in secondary
D) Epithelization only in partial thickness healing

Q8: Perforated gastric ulcer or Perforated appendicitis is/are:

A) Clean
B) Clean- contaminated
C) Contaminated
D) Infected

5) A / **6**) B / **7**) A / **8**) D

القادة

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الأعضاء

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حسبي الله لا إله إلا هو عليه توكلت وهو رب العرش العظيم.
اللهم إني أستودعك ما قرأت وما حفظت وما تعلمت فرده لي عند حاجتي إليه إنك على كل شيء قدير.

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