



## Burn injury and wound healing

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#### Color Index:

-Doctor's Notes -Surgery Recall -Doctor's Slides -Important -Extra

# OBJECTIVES

<b>Skin</b>	Structure and function
<b>Wounds:</b>	<ul style="list-style-type: none"> <li>a. Types of wound</li> <li>b. Principles of wound healing</li> <li>c. Factors influencing wound healing</li> <li>d. Wound infection</li> <li>e. Involvement of other structures</li> <li>f. Devitalized skin flaps</li> <li>g. Wound with skin loss</li> <li>h. Crushing/degloving injuries and gunshot wounds</li> </ul>
<b>Burns:</b>	<ul style="list-style-type: none"> <li>a. Mechanisms</li> <li>b. Local effects of burn injury</li> <li>c. General effects of burn injury</li> <li>d. Classification</li> <li>e. Prognosis</li> <li>f. Management</li> </ul>
<b>Skin and soft tissue lesions</b>  <b>“Not mentioned by the doctor” But you can find it in Davidson’s P( 307 -314)</b>	<ul style="list-style-type: none"> <li>a. Diagnosis of skin swelling</li> <li>b. Cysts</li> <li>c. Tumors of the skin</li> <li>d. Epidermal neoplasms arising from basal germinal cells</li> <li>e. Epidermal neoplasms arising from melanocytes</li> <li>f. Vascular neoplasms (hemangiomas)</li> <li>g. Tumors of nerves</li> <li>h. Tumors of muscles and connective tissue</li> <li>i. Disorders of the nails</li> </ul>

# Skin

## Functions

- Body Covering
- Permit movement of underlying muscles & joint
- Sensors for touch, pain, and temperature
- Vitamin D production
- Temperature regulation
  - sweating, blood flow
- Sun protection
- Detoxification/activation of drugs and chemicals
- Immunosurveillance
  - Langerhaus cells, t-lymphocytes

## Layers

### Epidermis:

- Outer layer contains the stratum corneum
- The rate limiting step in dermal or percutaneous absorption is diffusion through the epidermis

### Dermis:

- Much thicker than epidermis.
- Dermis layer produce the skin
- True skin & is the **main** natural protection against trauma
- Contains (called **Skin appendages**)
  - Sweat glands
  - Sebaceous glands
  - Blood vessels
  - Hair
  - Nails

### Subcutaneous:

- Also called hypodermis.
- Contains the fatty tissues which cushion & insulate



**skin layers:**  
don't miss clicking on mr.Surgeon to watch (9:39)

# Burns

## ❖ Death:

- Most deaths occur at home & more common in elderly.
- **Risk factors of burn death??** mnemonic:BAI..
  - > 40% BSA.
  - Age > 60 years.
  - **inhalation injury.**

{Age+BSA\*=%mortality} → most with >70% die

- Causes of death??

1. Smoke inhalation.

2. Sepsis ⇒ tetanus vaccination is mandatory in all pts.

3. Pneumonia.

4. Shock ⇒ either septic or Hypovolemic shock which is also called "Burn shock", caused by loss of fluid from intravascular space.

### Local Effect Of Burns Injury:

1- Destruction of tissues

2- Inflammation (deeper tissues) .

the damaged capillaries become permeable to protein ⇒ exudate forms ⇒ Lymphatic drainage fails to keep pace with the rate of exudation ⇒ interstitial edema ⇒ reduction in circulating fluid volume.

3- Infection

almost always be infected by microorganisms within 24 to 48 hrs.

### General Effect Of Burns Injury:

Depend upon the burn's size & Depth.

- Large Surface burns:

-water, salt and protein loss ⇒ hypovolemia

-increased catabolism.

-Some red cells are destroyed immediately by a full-thickness burn.

- Red cell loss is small compared to plasma loss in the early period, and haemoconcentration,

Reflected by arising hematocrit. "high proportion of RBCs/Plasma"

\* BSA: Body Surface Area.

## ❖ Three zones of injury:

### Zone of hyperemia

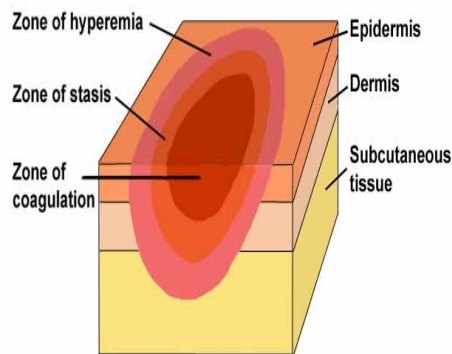
- Outermost area
- no injury , only hyperemia

### Zone of stasis

- (at risk of necrosis)
- Area of reversible damaged cells it is the area we are trying to preserve & convert to an area of hyperemia . by treating the burn.
- If **NOT** treated will turn to area of Necrosis.

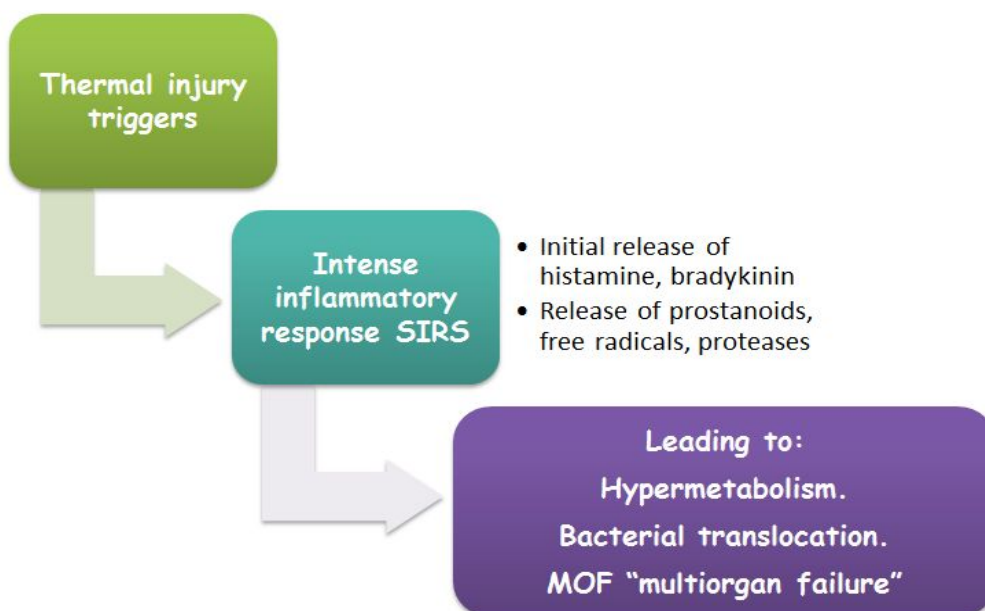
### Central necrosis

- Area of an irreversible damage
- the Thickest part of burn and has more Depth of injury



## ❖ Pathophysiology of Burns:

- Dynamic injuries
- Cellular damage at  $>45^{\circ} C$
- Dependent on temperature and duration.



## ❖ Pathophysiology of Burns cont:

- Any injury in any part of your body will cause inflammation.
- In burns, usually the inflammatory process is limited as long as the surface area is less than 20% .
- If more the body will develop systemic inflammatory response:

⇒ breakdown of body tissues especially proteins

⇒ this will affect the muscles and the lining of the gut leading to losing it

⇒ Thus the bacteria of the GIT system will travel to the blood

⇒ causing sepsis and multi-organ failure and death.

## ❖ What is the 1st thing we need to do for a Burn patients ?

- Apply ATLS protocol "ABCDEs", then urine output;
- check for eschar and compartment syndromes

## ❖ In History taking, what is important to know regarding burn patients is ?

- Type of burn
- Onset
- Closed or an open area (especially the flame burn)
- The exact mechanism (How Did he get the burn?)
- In Examination what is important to know is the:

1-Degree

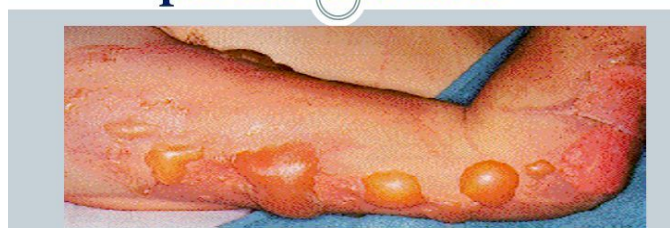
2-Body surface area (how much of the body was affected?)

❖ Level of burn: important..

- For each degree of burn you need to Know > which part of the skin is affected /how does it look / management
- This table from team 433..
- The dr said that he won't ask about differentiation between the 2 types of 2nd degree burns "superficial & deep" in pictures..

Burn Degree	First Degree (Superficial)	Second Degree (Partial Thickness Burns)		Third Degree (Full Thickness)
		Superficial (Mid-dermal)	Deep (Deep-dermal)	
Affected skin layer	Epidermis only	Epidermis + superficial dermis	Epidermis + most of dermis	Epidermis and all of dermis
Involving of appendages	No	No	Most of them	All of them
Sensation	Intact	Intact	Decreased	Anesthetic
Pain	Mild to moderate	Severe pain	Less painful "more nerves are destroyed"	Painless
Healing and scarring	3-6 days with no scar	1-3 weeks , scaring is unusual with perfect cosmetic result	More than 3 weeks with hypertrophic scarring	Does not heal with severe scarring
Appearance	Erythema + blanches with pressure	Blisters + blanches with pressure	Thick blisters + decreased blanching	White and tan thrombosed vessels + <b>Escher</b> "dead tissue" +leather skin + no blanching
Treatment	Analgesics & pain medications	Flamazine	Surgical	Surgery and skin grafting
Examples	Sun burns	Scaled from under boiling water	Scaled from boiling water	Flame burns

**2nd Degree Burn –superficial partial thickness**



Alert: Mr. surgeon is waving his saw telling you to test yourself in this table



<p><b>Burn</b></p>			
<p><b>Degree</b></p>	<p><b>2nd degree with some areas of 1st degree</b></p>	<p><b>A- 3rd degree:</b> <b>B-3d degree and deep partial</b></p>	<p><b>4th degree</b> <b>Burn injury into bone or muscle</b></p>
<p><b>Management</b></p>	<p><b>1st degree:</b> Nothing just give them something for the skin care + painkillers and send them home it will heal</p> <p><b>2nd degree:</b> 1- Superficial partial: Topical treatments (ex : ointments) and painkillers. 2- Deep partial: Surgery</p>	<p><b>3rd degree :</b> Surgery (skin graft)  Deep partial: Surgery</p>	



## ❖ Determining Extent of Injury:

Burn extent determines therapy and prognosis.

- Burn size estimate often inaccurate.
- Extent of injury described using percentage of **total body surface area that is burned (TBSA)**
- For patients > 9 "rule of nines" may be used.
- For small burns, the patient's palm covers 1%. (If patient present with multiple burns we use it to estimate)
- With young children proportions differ ⇒ b/c disproportionate size of head and trunk.

### Rule of nine

Figure 1. Estimating the percent total body surface area of burns in adults.

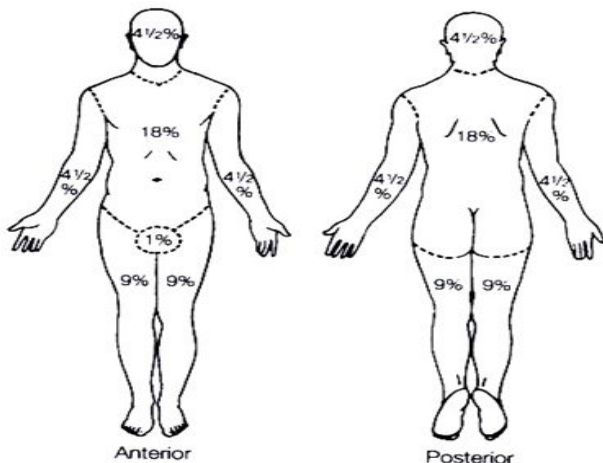
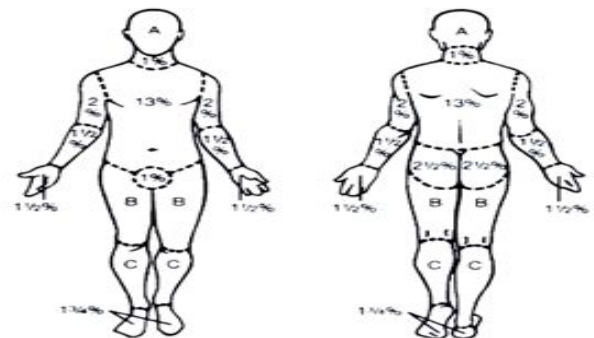


Figure 2. Estimating the percent total body surface area of burns in children.



Relative percentages of areas affected by growth

Age	Half of head (A)	Half of one thigh (B)	Half of one leg (C)
Infant	9 1/2	2 3/4	2 1/2
1 yr	8 1/2	3 1/4	2 1/2
5 yr	6 1/2	4	2 3/4
10 yr	5 1/2	4 1/4	3
15 yr	4 1/2	4 1/2	3 1/4
Adult	3 1/2	4 3/4	3 1/2

In Adults:

- Each hand is 9% "4.5% anteriorly and 4.5% posteriorly"
- Each leg is 18% "9% anteriorly and 9% posteriorly"
  - Head & Neck are 9%
  - Genital area is 1%

$$V = \text{BSA Burn (\%)} \text{ Weight (kg)} \times 4$$

**Parkland formula:** widely used to estimate the volume (V) of crystalloid necessary for the initial resuscitation of the burn patient; half of the calculated volume is given in the first 8 hours, the rest in the next 16 hours.

First half of the amount is given in the first 8 hours if patient came after 4 hours the amount should be given is (only the first half) but if patient came after 8 hours give it all.

## ❖ Inhalation Injury:

- Upper airway obstruction : patient will have a local reaction (edema) which will lead to obstruction although they have nothing in their lungs!
- The problem is in the upper area it is very very critical , when you see a patient with burn in closed area or head & neck and he is coughing Dark sputum = think of upper airway injury ⇒ Prophylactic intubation

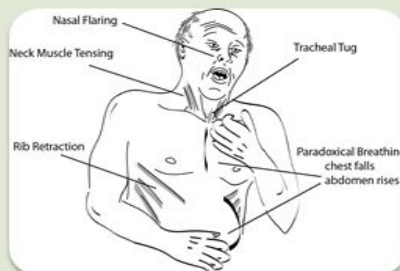
⇒ For intubation; oropharyngeal swelling may occlude the airway so that intubation is impossible; 100% oxygen should be administered **immediately** and continued until significant **carboxyhemoglobin is ruled out..**

⇒ to diagnose smoke inhalation **bronchoscopy** should be done.



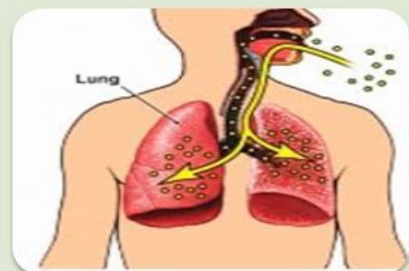
### 1. Carbon Monoxide Poisoning.

- CO has stronger affinity for HGB than O<sub>2</sub>
- Signs of CO poisoning:
  - Confusion, dizziness, HA, NV, flushed skin
- Treatment **100% FiO<sub>2</sub>**



### 2. Upper Airway Obstruction.

- Common in head and neck burns and smoke inhalation
- Edema continues at **least 24 hours**
- Protect airway with **intubation**
- Edema usually decreases by post burn day 3



### 3. Pulmonary Injury from Chemical Inhalation.

- Develops ARDS "Acute Respiratory Distress Syndrome" within 24 hours post injury
  - **Pneumonia** may occur as late as post burn day 10

# SMOKE INHALATION

Pulmonary alveolar  
macrophages  
activated

↓  
Chemotactic factors  
released

↓  
Aggregation/activation  
neutrophils

↓  
Release of  
oxygen radicals,  
proteases

↓  
Increased  
pulmonary  
endothelial  
permeability

↓  
Pulmonary oedema

Tracheobronchial  
epithelial injury

↙  
Thromboxane  
released

↓  
Increased  
pulmonary artery  
pressure

↓  
Airway exudate/  
cast formation,  
small airway  
obstruction

↓  
Atelectasis,  
air trapping

↘  
Pneumonia

↓  
Proliferation type 2  
pneumocytes

↓  
Pulmonary fibrosis

## ❖ Determining Extent of Injury

- Look for **circumferential** burns to chest, neck and limbs that may compromise ventilation or circulation.

⇒ *Circumferential deep full thickness burns of an extremity or around the chest or abdomen should be carefully monitored.*

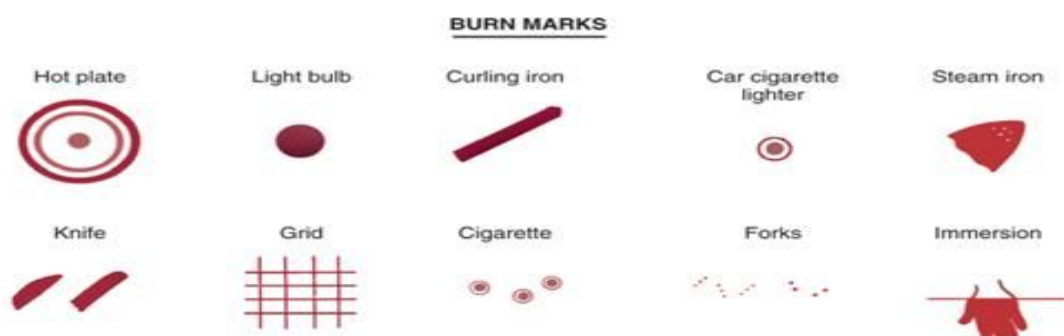
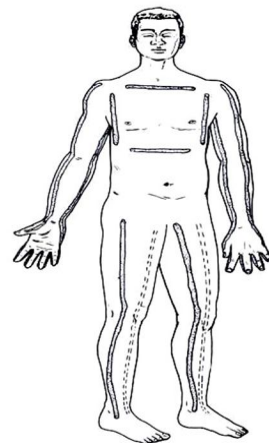
- Oedema and swelling in the tissue deep to the burn cause the unyielding overlying burnt skin ('**eschar**') to act like a tourniquet.
- In a limb this will result in interference with distal blood flow.
- In the chest or abdomen, it can restrict chest expansion and diaphragmatic movement and interfere with ventilation

## ❖ How is it treated?

**Escharotomy**: full-thickness longitudinal incision through the eschar with scalpel or electrocautery

- Loss of distal pulses late:
  - Assess for warmth, sensation, motor, rigidity.
  - Doppler exam helpful.
    - ⇒ This test uses **ultrasound** to look at the blood flow in the large arteries and veins in the arms and legs.
- Identify potential abuse. "**specially in children**"
- Well circumscribed, feet, ankles, buttocks.

Figure 3. Performance of escharotomies.



⇒ Compartment Syndrome is seen in only : Deep partial & 3rd Degree Burns .

⇒ What is the difference between the Compartment Syndrome caused by Burn and the one caused by bleeding inside the compartment (ex: vascular injury) ?

	Burn	Others
Limiting tissue	The skin	The fascia
Management	escharotomy	Fasciectomy

### ❖ Burn wound infection:

- Causative organisms?

Staphylococcus aureus, Pseudomonas, Streptococcus, Candida albicans

- Signs of burn wound infection?

1. Increased WBC with of left shift "high number of immature WBCs ⇒ the bone marrow is fighting the infection by producing new immature cells ,
2. **Discoloration of burn eschar** (most common sign),
3. Green pigment, necrotic skin lesion in unburned skin,
4. Edema, ecchymosis tissue below eschar,
5. Second degree burns that turn into third-degree burns,
6. hypotension

- Why not to use Prophylactic antibiotics in burn pts?

Prophylactic antibiotics **have not** been shown to reduce the incidence of sepsis, but rather have been shown to select for resistant organisms; Except for **Tetanus** prophylaxis "**Mandatory**"

## ❖ Prognosis: According to obj..

- 1) Age And General Condition ⇒ Infants, the elderly, alcoholics and those with other co-morbidity fare less well than healthy young adults .
- 2) Extent Of The Burn: Use the Rule of nine.
- 3) Depth Of The Burn :  
Full-thickness burns inevitably become infected unless excised early, and in the case of large burns infection may prove life-threatening.
- 4) Site Of The Burn :  
Burns involving the face, neck, hands, feet or perineum are particularly liable to threaten appearance or function. They require inpatient management.
- 5) Associated Respiratory Injury:  
This is extremely common in house fires and usually results from the inhalation of smoke from burning plastic foam upholstery. It is frequently fatal.

## ❖ Fluid Resuscitation:

- Hypovolemia was major cause of death.
- Massive transudation of fluids from vessels due to increased permeability.
- Edema intensifies over 8-48 hours.
- **Goal:** preservation of organ perfusion and urine output.

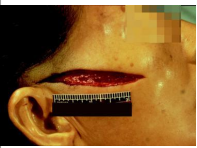





⇒ Foley catheter to measure urine output, blood pressure, heart rate, peripheral perfusion, and mental status are mandatory to know the **volume status**.

Burns	character	Notes
Electrical	<ul style="list-style-type: none"> <li>Caused by passage of electric current</li> <li>Damage increased in small bony areas: Fingers, feet, lower legs, forearm</li> <li>Systemic effects</li> <li>Low voltage (&lt;1000 V)</li> <li>High voltage (&gt;1000): Massive tissue damage, respiratory and cardiac arrest</li> <li>ECG, CPK, UA, monitor</li> <li>Local care often necessitates grafting and amputation</li> </ul>	<p>⇒ Any electrical burns are considered 3rd degree pain</p> <p>⇒ Most of the destruction from electrical burns is internal because the route of least electrical resistance follows nerves, blood vessels, and fascia; injury is usually worse than external burns at entrance and exit sites would indicate; cardiac dysrhythmias, myoglobinuria, acidosis and renal failure are common.</p> <p>- Management by fasciotomy because there is no burnt skin</p>
Chemical	<ul style="list-style-type: none"> <li>Delayed and progressive injury</li> <li>Deceptively superficial at first</li> <li>Acid more limited (coagulation necrosis)</li> <li>Alkalis more destructive (liquefaction)</li> <li>HFI: significant necrosis, arrhythmias,</li> <li>hypoCa</li> <li>Removal of causative agent <ul style="list-style-type: none"> <li>Brush off metals and powders</li> <li>Copious irrigation with water</li> </ul> </li> </ul>	<p>⇒ In general, ALKALI burns are more serious because the body cannot buffer the alkali, thus allowing them to burn for much longer.</p>
Thermal	<p>1- Flame burn : by fire</p> <p>2- Scald burn : caused by hot fluids you should know the type of fluid because you need to know the temperature of the fluid .</p>	<p>-----</p>
Radiation	<p>-----</p>	<p>-----</p>

# Wound healing

## ❖ Wound:

- A disruption of normal anatomic relations as a result of injury intentional or unintentional.
- Regardless of causation or tissue type, wound healing presents with identical biochemical and physiologic processes, though wound healing may vary in timing and intensity.
- The doctor focused on the "Aim, Duration, Time and the cells of each phase..

Types Of Wound (according to the mechanism of injury)	
	<p><b>Incised Wounds :</b> A sharp instrument causes these; if there is associated tearing of tissues, the wound is said to be lacerated</p>
	<p><b>Abrasions:</b> These result from friction damage and are characterized by superficial bruising and loss of a varying thickness of skin and underlying tissue</p>
	<p><b>Crush Injuries:</b> These are due to severe pressure. Even though the skin may not be breached, there can be massive tissue destruction. Edema can make wound closure impossible. It can lead to compartment Syndrome.</p>
	<p><b>Degloving Injuries:</b> These result from shearing forces that cause parallel tissue planes to move against each other. Large areas of apparently intact skin may be deprived of their blood supply by rupture of feeding vessels</p>
	<p><b>Gunshot Wounds:</b> These may be low-velocity (e.g. shotguns) or high-velocity (e.g. military rifles). Bullets fired from high-velocity rifles cause massive tissue destruction after skin penetration</p>
	<p><b>Burns:</b> These are caused not only by heat but also by electricity, irradiation and chemicals</p>



# Phases Of Wound Healing

	1. Inflammatory	2. Proliferative	3. Maturation
Aim	Stop <b>bleeding</b> by provisional matrix + protect the body	<b>Repair</b> the damaged area	<b>Remodeling</b> of the wound
Duration	Elongated if you don't clean the wound.	-----	-----
important cells	Macrophages.	Fibroblasts replaces the provisional matrix formed before by collagen .	-----

## 1. Inflammatory:

- Substrate or reactive phase, immediate
- Typically **days 1-10**
- Response to **limit and prevent further injury, inflammation, hemostasis, sealing surface, removing necrotic tissue and debris, migration of cells** into wound by chemotaxis, cytokines, and growth factors

**A- Initial** intense local **vasoconstriction** of arterioles and capillaries **followed by vasodilation** and vascular permeability

**B-** Tissue injury & blood vessel damage ⇒ exposure of subendothelial collagen to platelets and vWF activates ⇒ the coagulation pathway

★ <b>Plugging:</b>	★ <b>Provisional matrix:</b>	★ <b>Platelet aggregation:</b>
Platelet and fibrin	Platelets, fibrin and fibronectin	Thromboxane (vasoconstrictor), thrombin, platelet factor 4

## ➤ Platelets:

- Alpha granules contain:

1 -platelet factor 4 ⇒ aggregation

2-Beta-thrombomodulin ⇒ binds thrombin

3-PDGF: chemoattractant

4-TGF-beta ⇒ **key component tissue repair**

(Transforming growth factor beta if not present, no wound healing)

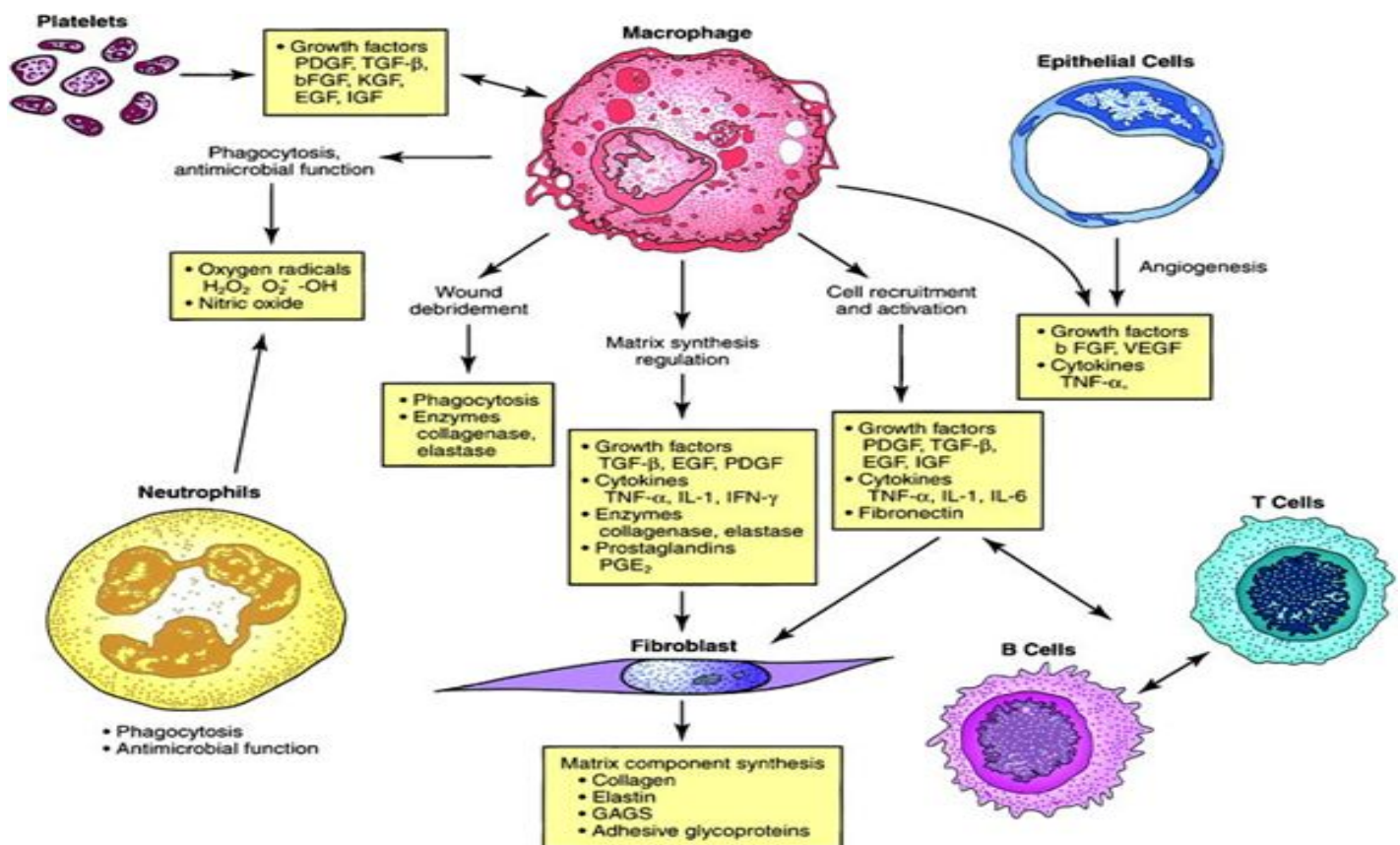
- Dense granules contain vasoactive substances: adenosine, serotonin and Ca
- Other factors released: TXA, Platelet activation factor, Transform. growth factor alpha, Fibroblast growth factor, Beta lysin (antimicrobial), PGE2 and PGI2 (vasodilate) and PGF2 (vasoconstrict).

## ➤ Polymorphonuclear Cells:

- Chemotaxins attract after extravasation
- Migrate through the ECM by transient interaction with integrins
- PMNs scavenge, present antigens, provide cytotoxicity-free radicals (H<sub>2</sub>O<sub>2</sub>)
- Migration PMNs stops with wound contamination control usually a few days
- Persistent contaminant: continuous influx PMNs and tissue destruction, necrosis, abscess & systemic infection
- PMNs are **not essential** to wound healing

## ➤ Macrophages Necessary:

- Monocytes migrate & activate: Macrophages
- Appear when PMNs disappear 24-48 hr Do the same activities as PMN's
- Plus orchestrate release of enzymes (collagenase, elastase), PGE's, cytokines (IL-1, TNF alpha, IFN ), growth factors (TGF & PDGF), and fibronectin (scaffold/anchor for fibroblasts)
- Activate Fibroblasts, endothelial and epithelial cells to form **Granulation Tissue**



## 2. Proliferative:

- Regenerative or Reparative day 5- 3 weeks
- In the beginning of the phase type 3 collagen Is the more abundant.
- **Angiogenesis:** endothelial cells activate & degrade Basement membrane, migrate and divide to form more tubules
- **Granulation Tissue:** capillary ingrowth, collagen, Macrophages, Fibroblasts, Hyaluronic acid (GAG)
- ★ Fibroblasts differentiate from resting mesenchymal cells in connective tissue 3-5 days migrate from *wound edge*
- ★ **Fibroplasia:** Fibroblasts proliferate replace fibronectin-fibrin with collagen contribute ECM

### ➤ Collagen:

- Type III predominant collagen synthesis days 1-2
  - Type I days 3-4
- ⇒ Type III **replaced** by Type I in 3 weeks

## Collagen Types

I	II	III	IV	V
<ul style="list-style-type: none"> <li>- (80% skin)</li> <li>- Most Common: skin, bone, tendon.</li> </ul> <p><b>Primary type in wound healing.</b></p>	Cartilage	<ul style="list-style-type: none"> <li>- (20 % skin)</li> <li>- <b>Increased Ratio</b> in healing wound, also blood vessels and skin</li> </ul>	Basement Membrane	Widespread, particularly in the cornea

- Wound strength:

- 6 Week = 60% original, 80% final strength
- 8 Week-1 year  $\approx$  80% original (Max)
- Net Collagen = 6 weeks amount stays the same but cont. crosslink increase strength = maturation

### 3. Maturation:

- Remodeling of wound **3 week-1+year**
- Type I replaces Type III Collagen: net amount doesn't change after 6 weeks, organization & crosslinking (No increased amount of collagen only replacing type 3 by type1).
- Decreased vascularity, less fibroblasts & hyaluronic acid.
- Peripheral nerves regenerate @ 1mm/day.
- Accelerated Wound Healing: reopening results in quicker healing 2nd time around.



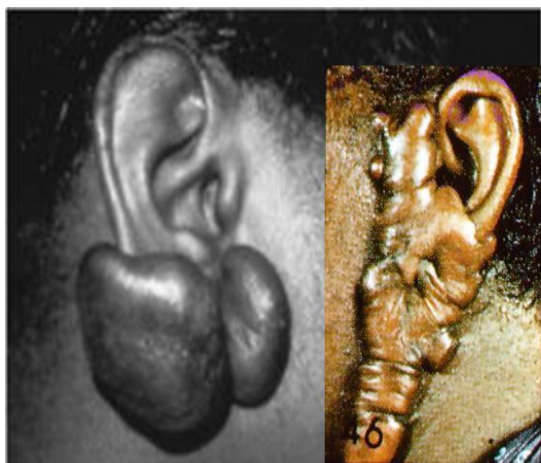
*Burn/Keloid causing contracture*

- ★ **Contraction:** (physiological process) centripetal movement of the whole thickness of surrounding skin reducing scar Contraction.
- ★ **Myofibroblasts:** special Fibroblasts express *smooth muscle* and bundles of actin connected through cellular fibronexus to ECM fibronectin, communicate via *gap junctions* to pull edges of the wound .
- ★ **Contracture:** (pathological process) the physical constriction or limitation of function as the result of Contraction (scars across joints, mouth, eyelid).

# Difference between Keloids and Hypertrophic Scars

## Keloids

- **Beyond the Borders**
- Excess Deposition of Collagen Causes Scar Growth Beyond the Border of the Original wound
- Unknown cause
- **Autosomal Dominant, Darker Pigment, Often above clavicle but not always**
- Tx: XRT, steroids, silicone sheeting, pressure, excise. often Refractory to
- Tx & not preventable
- **Treatment : gets worse by time , we need to treat them like cancer. Surgical excision only is not adequate we do intralesional excision within the borders of keloid + steroids & radiation thereby .**



## Hypertrophic Scars

- **Confined within**
- Excess collagen deposit causing raised scar remains within the original wound confines
- Darker pigmented skin & flexor surfaces of upper torso
- Often occurs in burns or wounds that take a long time to heal, sometimes preventable or healing under tension
- **Can regress spontaneously and we can fasten the process by surgery**
- Tx: steroids, silicone, pressure garments



## ❖ Impediments to Wound Healing: (Factors Affecting Wound Healing)

- **Bacteria**  $>10^5/\text{cm}^2$  : Decreased  $\text{O}_2$  content, collagen lysis, prolonged inflammation
- **Devitalized Tissue & Foreign Body**: Retards Granulation Tissue formation and healing
- **Cytotoxic drugs**: 5FU, MTX, Cyclosporine, FK-506 can impair wound healing.  
D-Penicillamine- inhibit collagen x-linking
- **Chemotherapy**: no effect after 14 days
- **Radiation**: Collagen synthesis abnormal, fibrosis of vessel
- **DENERVATION** has NO EFFECT on Wound Healing
- **Diabetes**: impedes the early phase response
- **Malnourishment**: Albumin  $<3.0$ , Vit-C
- **Smoking**: vasoconstriction, atherosclerosis, carboxyhemoglobin, decreased  $\text{O}_2$  delivery
- **Steroids**: inhibit macrophages, PMNs, Fibroblast collagen synthesis, cytokines, and decreased wound tensile strength
- **Vit A** (25,000 IU QD) counteracts effect of steroids

## ❖ Diseases Associated With Abnormal Wound Healing:

- **Osteogenesis Imperfecta**: Type I Collagen defect
- **Ehler-Danlos syndrome**: Collagen disorder, 10 types
- **Marfan Syndrome**: fibrillin defect (collagen)
- **Epidermolysis Bullosa**: Excess fibroblasts Tx: phenytoin
- **Scurvy**: Vit C req. for proline hydroxylation

## ❖ Wound Infection:

Surgical procedures can be classified according to the likelihood of contamination and wound infection to:

### 1) Clean Procedures:

are those in which wound contamination should not occur.

- **Example:** An incision for a clean elective procedure.
- In clean operations, the wound infection rate should be less than 1%.

### 2) Clean-contaminated Procedures:

are those in which no frank focus of infection is encountered but where a significant risk of infection is nevertheless present, perhaps because of the opening of a viscus, such as the colon.

- Infection rates in excess of 5% may suggest a breakdown in ward and operating theatre routine.

### 3) Contaminated Or 'Dirty' Wounds:

are those in which gross contamination is inevitable and the risk of wound infection is high.

- **Example:** emergency surgery for perforated diverticular disease, or drainage of a subphrenic abscess.

(Antibiotic prophylaxis is appropriate for the first two types of operation. Dirty wounds require therapeutic antibiotics.)

## ❖ Involvement Of Other Structures

- Some wound may conceal extensive damage to deeper structures such as body cavities, tendons, nerves or blood vessels.
- **Damage To Muscles, Tendons Or Nerves:** assessed by checking relevant motor and sensory function.
- **If the injury involves a limb,** the distal circulation must be checked.
- **To Check for injury to peritoneal, pericardial or pleural cavities & underlying bony injury:** X-rays



## ❖ Devitalized Skin Flaps

- In some cases, the flap is blue-black in color and obviously non-viable, but in most cases viability is uncertain.
- The wound must be cleansed and non-viable tissue excised.
- No attempt should be made to suture the flap back into place (because of the post-traumatic edema)

## ❖ Crushing/Degloving Injuries And Gunshot Wounds

Wounds of this type should never be closed primarily due to the extensive tissue destruction. After thorough irrigation and the removal of any obviously dead tissue and foreign material, such wounds should be lightly packed and dressed.

## ❖ Wounds With Skin Loss

- A small skin defect at a functionally or aesthetically unimportant site may be allowed to heal by secondary intention.
- However, it is often better to speed healing by importing skin to close the wound by means of a skin graft, which requires a vascular bed as it has no blood supply of its own, or a flap.

## ❖ Skin Grafts: 2 types:

**1) Split-thickness Skin Graft (STSG):** is a skin graft including the epidermis and part of the dermis. It can be processed through a skin mesher which makes apertures onto the graft, allowing it to expand up to nine times its size.

**2) Full-thickness Graft:** leaves a donor defect (which needs to be sutured or grafted) as large as the one to be filled and requires a well-vascularized bed to survive.

- They are strong, do not shrink, and look better than a split-skin graft.
- They are commonly used in reconstructive surgery: on the lower eyelid (where a good functional and/or cosmetic result is important).

## ❖ Flaps

- Flaps bring their own blood supply to the new site.
- They are thicker and stronger than grafts and can be applied to avascular areas such as exposed bone, tendon or joints.

**Flaps can be divided into:**

1. **Local Flaps:** The simplest flaps, uses local skin and fat.
2. **Distant Flap:** A flap may have to be brought from a distance and remain attached temporarily to its original blood supply until it has picked up a new one locally. This usually takes 2-3 weeks.

### surgical recall

★ Phases of wound healing: mnemonic: **In Every Fresh Cut**

1. **In**flammation

2. **E**pithelialization = epithelial coverage of the wound

3. **F**ibroplasia

4. **C**ontraction

★ Conditions that inhibit wound healing ⇒

- Anemia
- Malnutrition
- Cancer
- Radiation
- Hypoxia
- Sepsis
- DM

★ Contusion ⇒ bruise without a break in the skin..

**DIFFICULT ROADS  
OFTEN LEAD TO  
BEAUTIFUL  
DESTINATIONS.**