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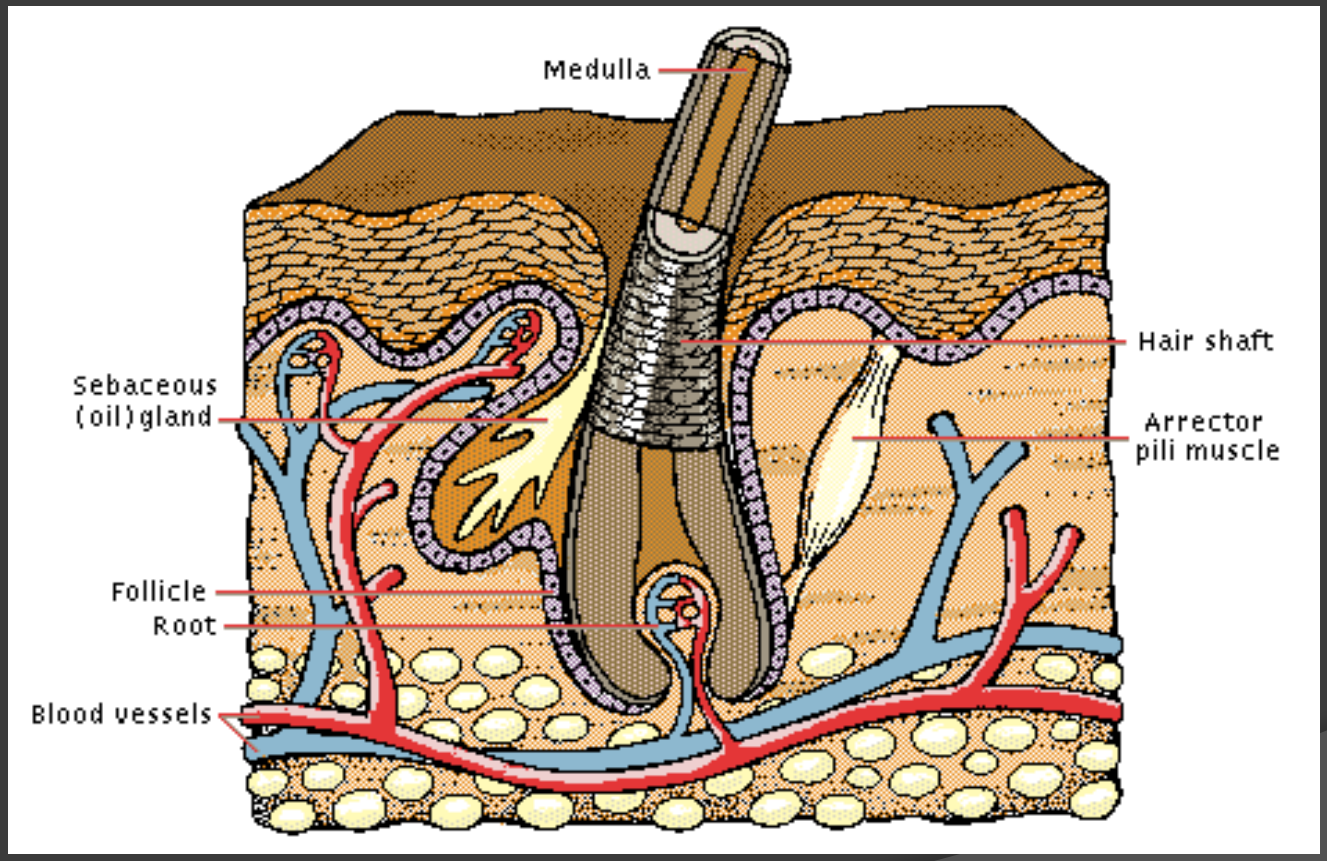
BURN INJURY AND WOUND HEALING

Skin Function

- 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
- Immunoserveillance
  - Langerhaus cells, t-lymphocytes

Skin layers


Medulla
Hair shaft
Sebaceous
(oil) gland
Arrector
pili muscle
Follicle
Root
Blood vessels

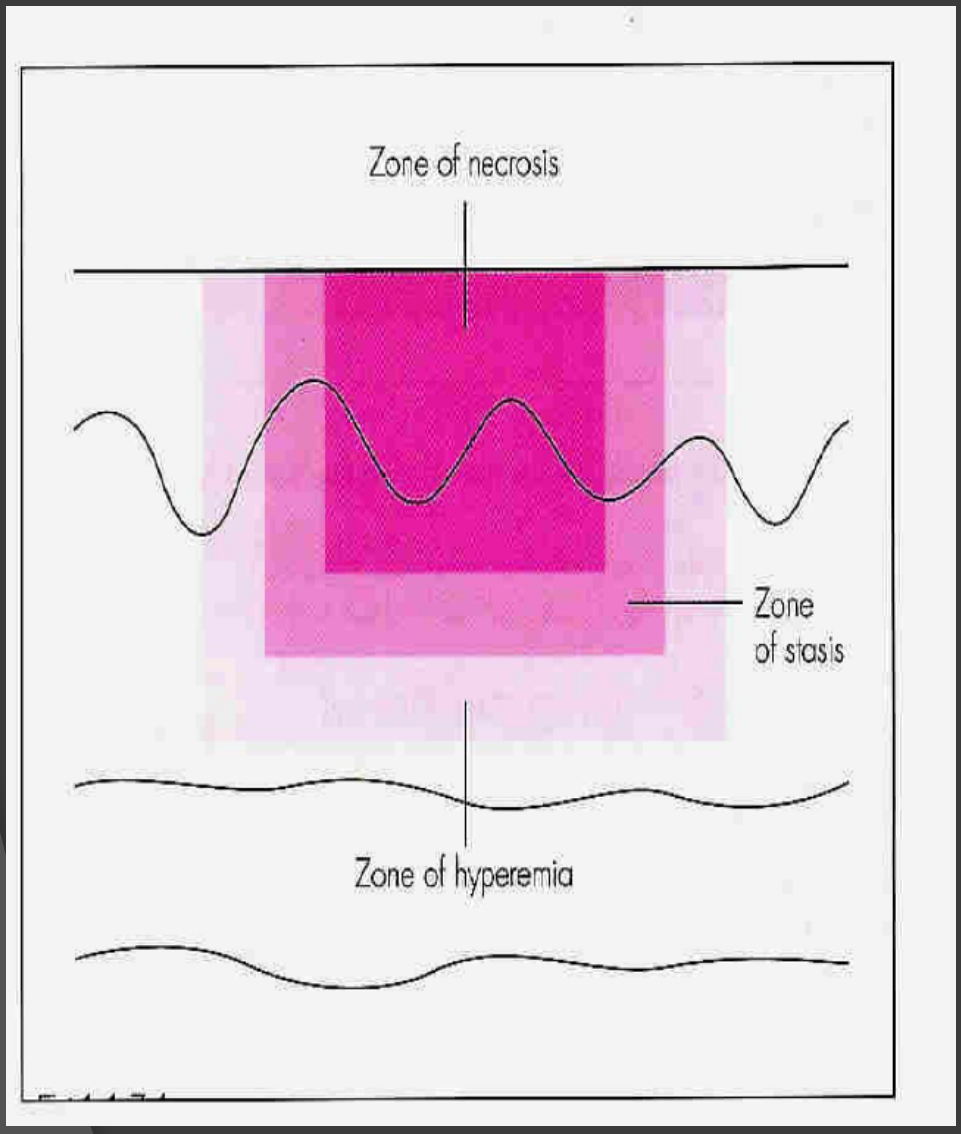
- Epidermis
  - Outer layer contains the stratum corneum
    - The rate limiting step in dermal or percutaneous absorption is diffusion through the epidermis
- Dermis
  - Much thicker than epidermis
  - True skin & is the main natural protection against trauma
  - Contains
    - Sweat glands
    - Sebaceous glands
    - Blood vessels
    - Hair
    - Nails
- Subcutaneous Layer
  - Contains the fatty tissues which cushion & insulate

Burns

- **Causes of death**
  - **– Smoke inhalation, sepsis, pneumonia,shock**
- **More common in elderly**
- **(age+BSA=%mortality), most with>70% die**
- **Risk factors for death:**
  - **– > 40% BSA, > 60 years, inhalation injury**

Pathophysiology of Burns

- Dynamic injuries
- Cellular damage at >45° C
  - – Dependent on temperature and duration
- Three zones of injury
  - – Central necrosis
  - – Zone of stasis (at risk of necrosis)
  - – Zone of hyperemia


Zone of necrosis
Zone
of stasis
Zone of hyperemia

Burn Pathophysiology

- Thermal injury triggers intense inflammatory response SIRS
  - Initial release of histamine, bradykinin
  - Release of prostanoids, free radicals, proteases
- Leading to:
  - Hypermetabolism.
  - Bacterial translocation.
  - MOF.

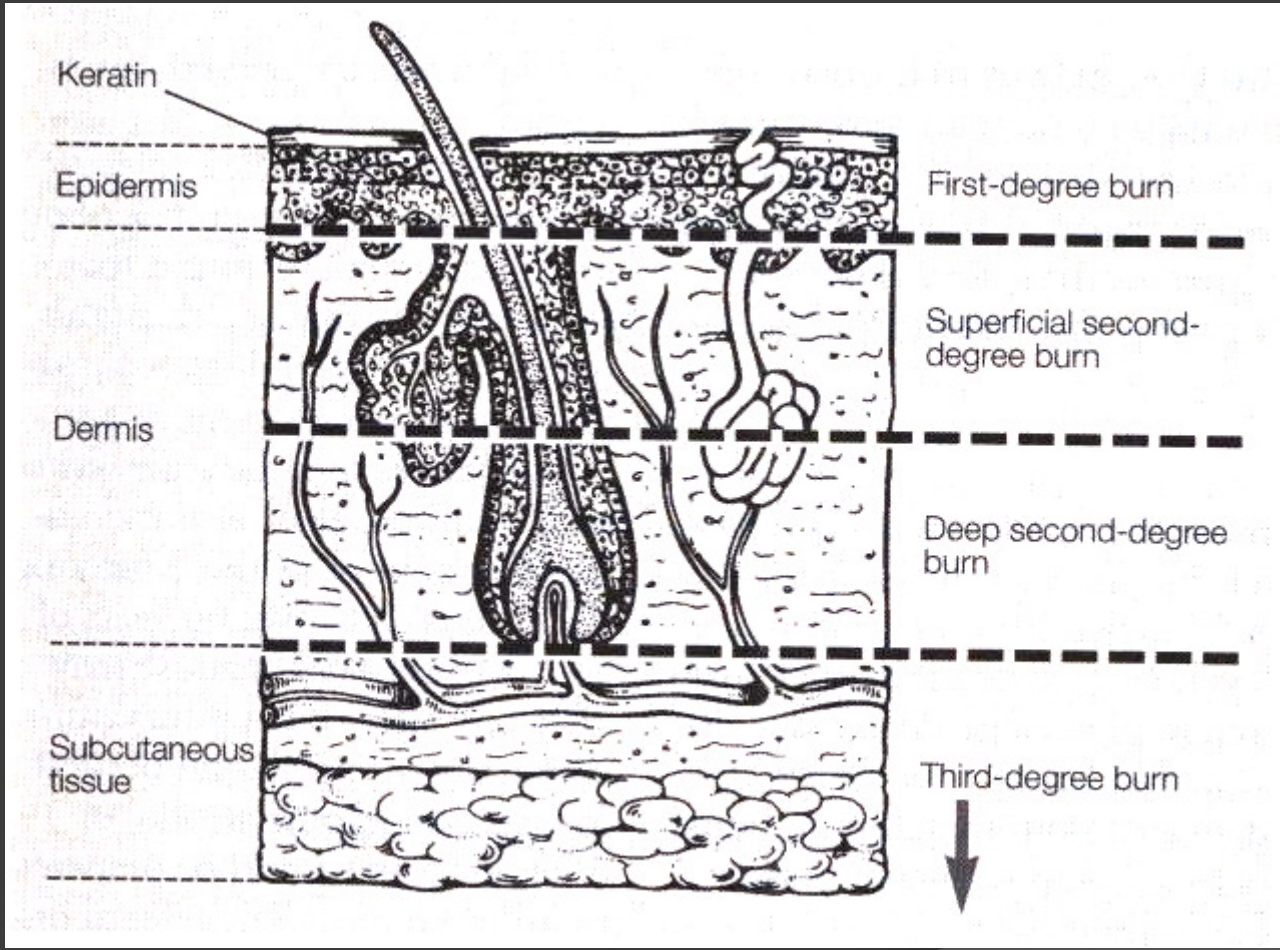
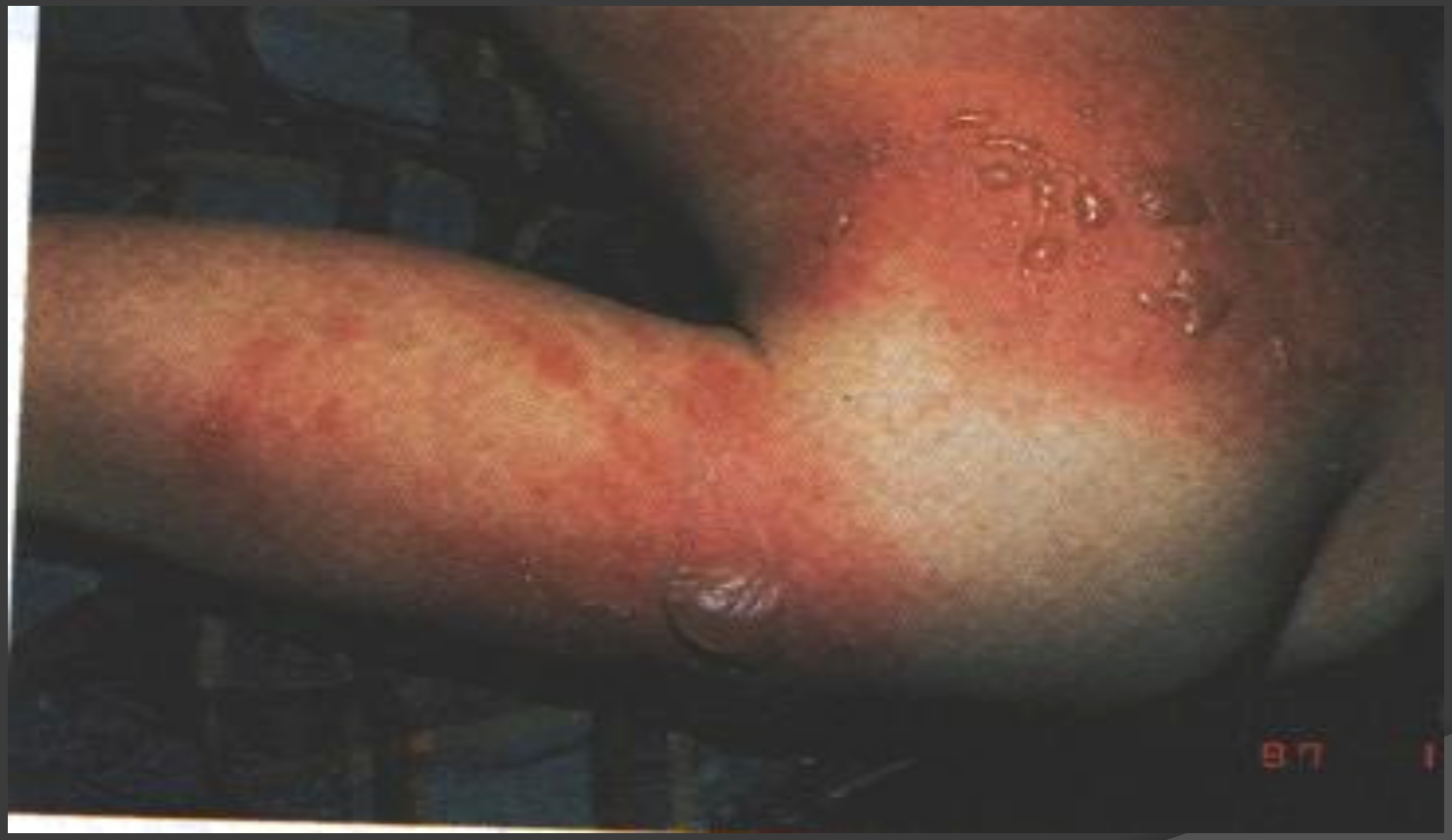
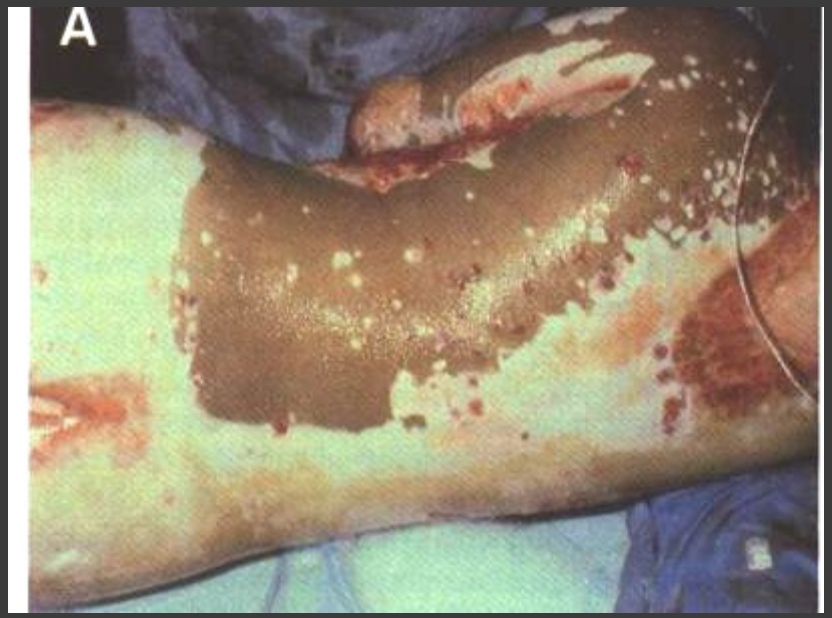

Keratin
Epidermis
Dermis
Subcutaneous tissue
First-degree burn
Superficial second-degree burn
Deep second-degree burn
Third-degree burn

Table 2. Burn Depth Classification.

Depth	Histology	Appearance	Sensation	Healing
First-degree:	Epidermis only	Erythema; blanches with pressure	Intact; mild to moderate pain	3-6 days without scarring
Second degree:				
· Superficial	Epidermis and superficial dermis; skin appendages intact	Erythema, blisters, moist, elastic; blanches with pressure	Intact; severe pain	1-3 weeks; scarring unusual
· Deep	Epidermis and most dermis; most skin appendages destroyed	White appearing with erythematous areas, dry, waxy, less elastic; reduced blanching to pressure	Decreased; may be less painful	>3 weeks; often with scarring and contractures
Third-degree:	Epidermis and all of dermis; destruction of all skin appendages	White, charred, tan, thrombosed vessels; dry and leathery; does not blanch	Anesthetic; not painful (although surrounding areas of second-degree burns are painful)	Does not heal; severe scarring and contractures




A

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%
- With young children proportions differ

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

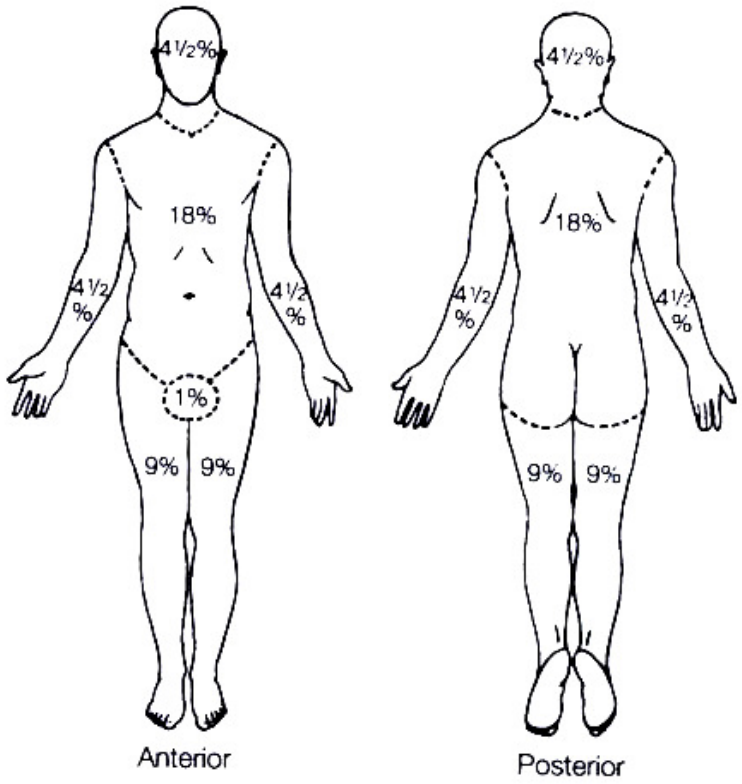
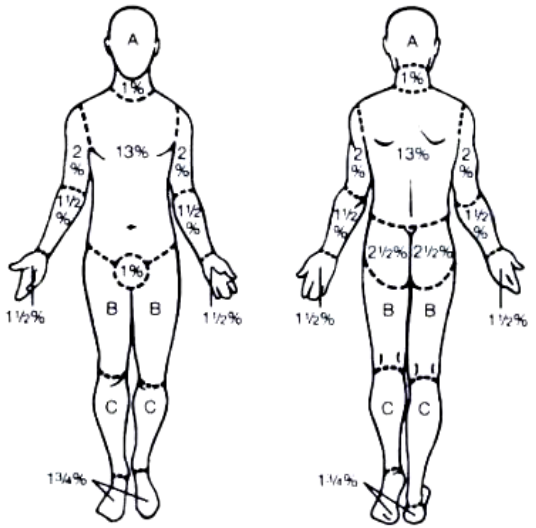


Figure 2. Estimating the percent total body surface area of burns in children.



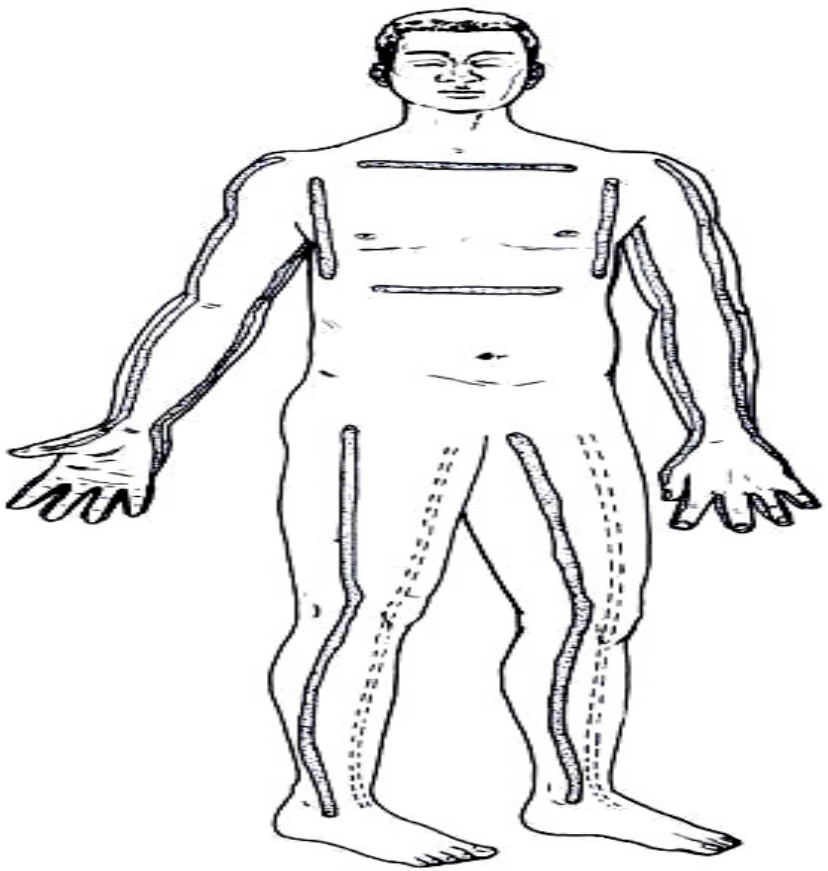
Relative percentages of areas affected by growth

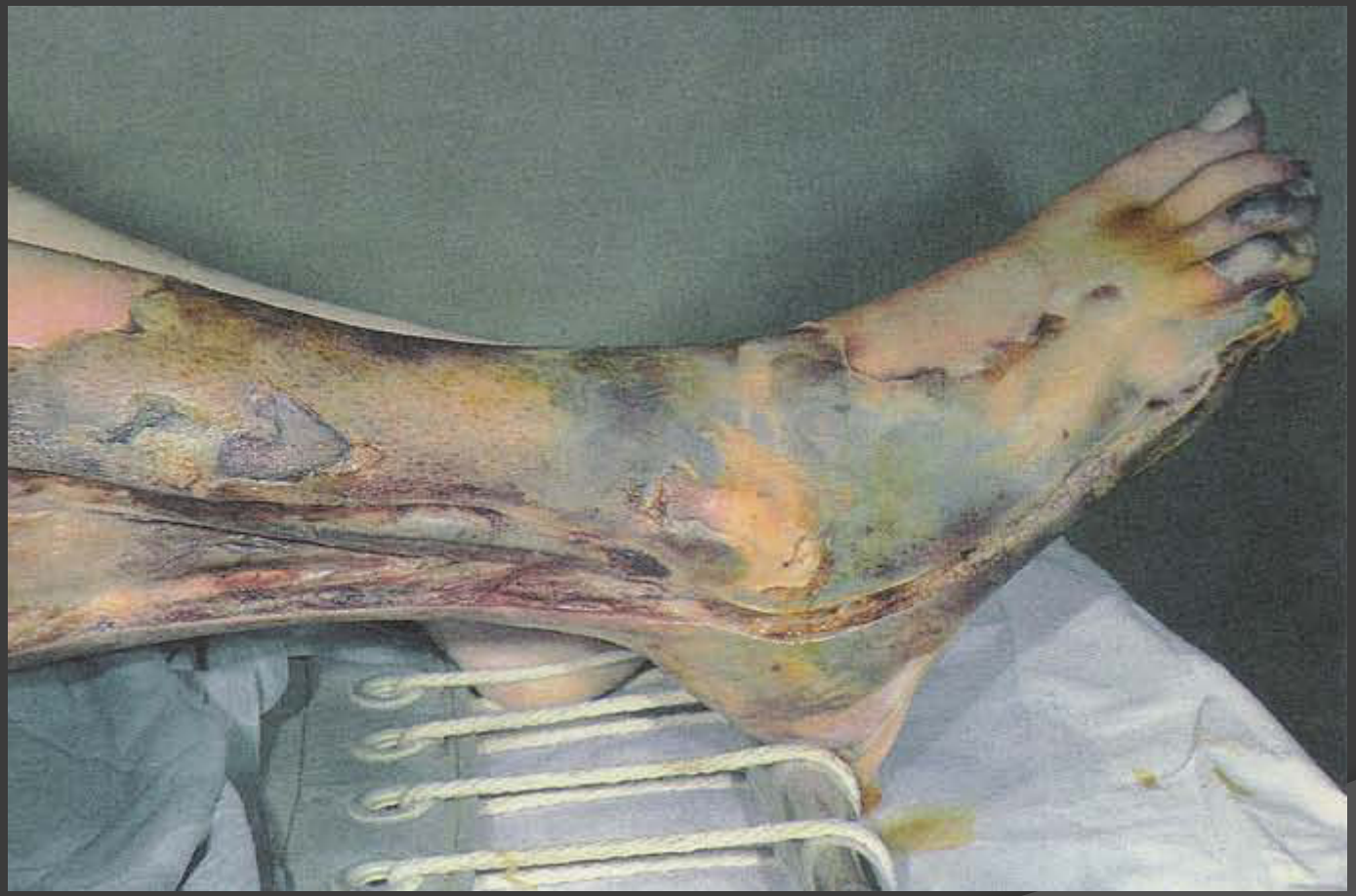
Age	Half of head (A)	Half of one thigh (B)	Half of one leg (C)
Infant	9½	2¾	2½
1 yr	8½	3¼	2½
5 yr	6½	4	2¾
10 yr	5½	4¼	3
15 yr	4½	4½	3¼
Adult	3½	4¾	3½

Evaluation of Burns – cont

- Look for circumferential burns to chest, neck and limbs that may compromise ventilation or circulation
- Loss of distal pulses late
- – Assess for warmth, sensation, motor, rigidity
- – Doppler exam helpful
- Identify potential abuse
- – Well circumscribed, feet, ankles, buttocks

Figure 3. Performance of escharotomies.





Inhalation Injury

- Smoke inhalation
  - – Carbon Monoxide Poisoning
    - • CO has stronger affinity for HGB than O2
    - • Signs of CO poisoning:
      - – Confusion, dizziness, HA, NV, flushed skin
    - • Treatment 100% FiO2
  - – 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
  - – Pulmonary Injury from Chemical Inhalation
    - • Develops ARDS within 24 hours post injury
    - • Pneumonia may occur as late as post burn day 10

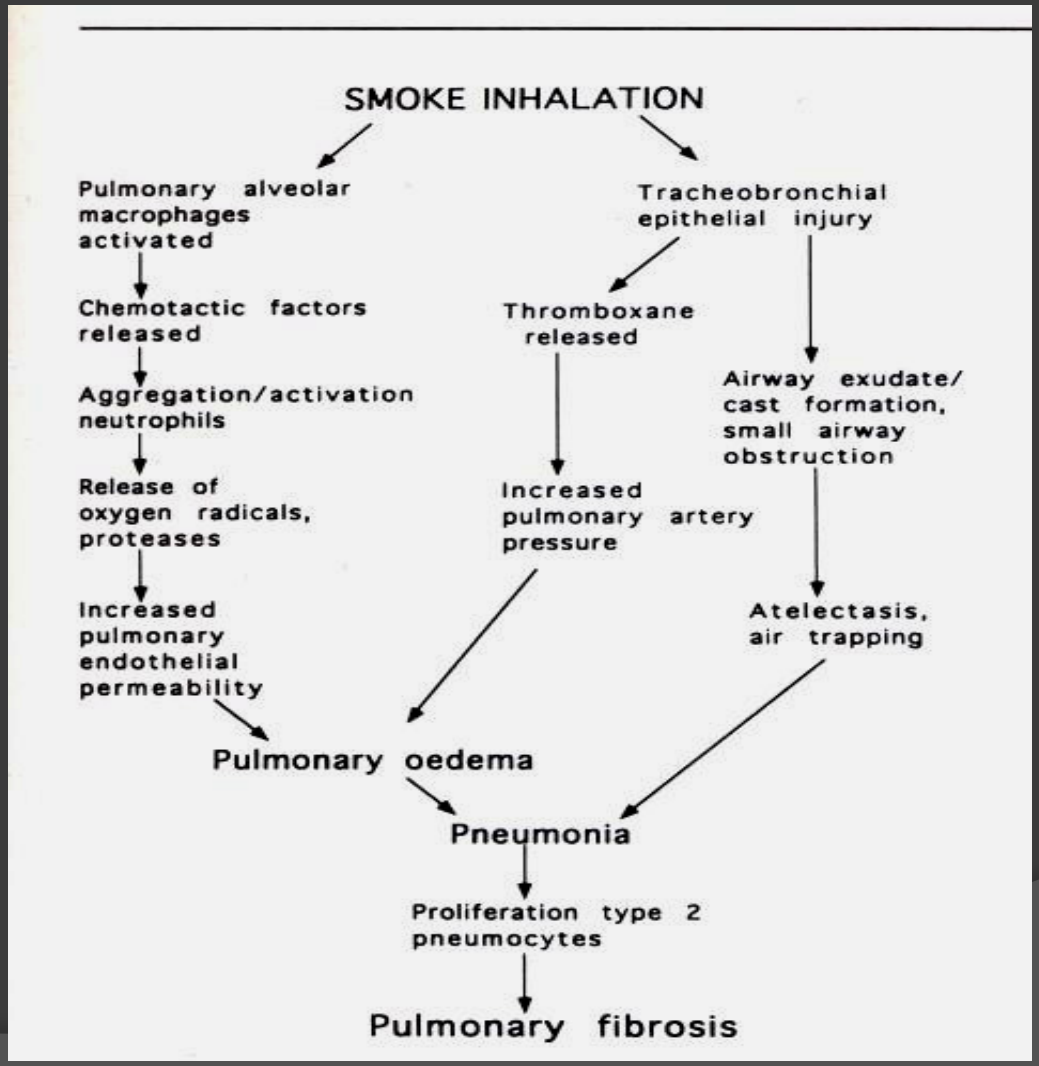

SMOKE INHALATION
Pulmonary alveolar macrophages activated
Chemotactic factors released
Aggregation/activation neutrophils
Release of oxygen radicals, proteases
Increased pulmonary endothelial permeability
Tracheobronchial epithelial injury
Thromboxane released
Increased pulmonary artery pressure
Airway exudate/ cast formation, small airway obstruction
Atelectasis, air trapping
Pulmonary oedema
Pneumonia
Proliferation type 2 pneumocytes
Pulmonary fibrosis

Table 10. Burn Unit Referral Criteria.

1. Partial-thickness burns greater than 10% TBSA
2. Burns that involve the face, hands, feet, genitalia, perineum, or major joints
3. Third-degree burns in any age group
4. Electrical burns, including lightning injury
5. Chemical burns
6. Inhalation injury
7. Burn injury in patients with preexisting medical disorders that could complicate management, prolong recovery, or affect mortality
8. Any patients with burns and concomitant trauma (such as fractures) in which the burn injury poses the greatest risk of morbidity or mortality
9. Burned children in hospitals without qualified personnel or equipment for the care of children
10. Burn injury in patients who will require special social, emotional, or long-term rehabilitative intervention

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

Table 6. Fluid Resuscitation Formulas.

Crystalloid Formulas

Parkland

- Lactated Ringer's 4 mL/%TBSA burn/kg
- Give half of calculated needs in first eight hours, the rest over 16 hours

Modified Brooke

- Lactated Ringer's 2 mL/%TBSA burn/kg

Hypertonic Saline

- Saline solution containing sodium 250 meq/L 0.6 mL/%TBSA burn/kg plus one-third isotonic salt solution orally up to 3,500 mL limit

Colloid Formulas (under recent question: see reference 83)

Brooke

- Lactated Ringer's 1.5 mL/%burn/kg+0.5 mL/kg Colloid+2,000 mL D_5W

Evans

- Normal saline 1.0 mL/%burn/kg + 1.0 ml/%burn/kg Colloid+2,000 mL D_5W

Slater

- Lactated Ringer's 2,000 mL/24 hr + Fresh frozen plasma 75 mL/kg/24 hr

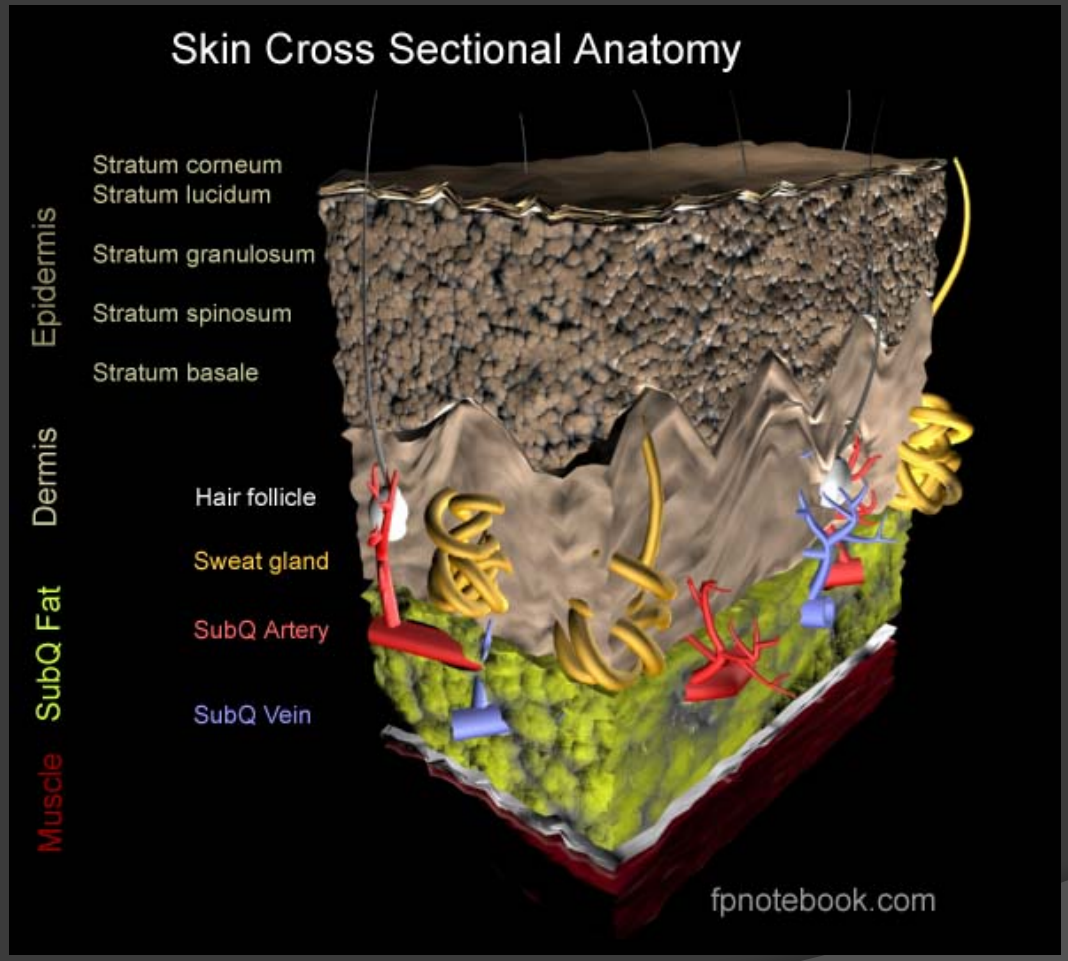
Electrical Burns

- Caused by passage of electric current
- Damage increased in small bony areas
  - – Fingers, feet, lower legs, forearm
- Systemic effects
- – Low voltage (<1000 V)
- – High voltage (>1000): Massive tissue damage, respiratory and cardiac arrest
- ECG, CPK, UA, monitor
- Local care often necessitates grafting and amputation

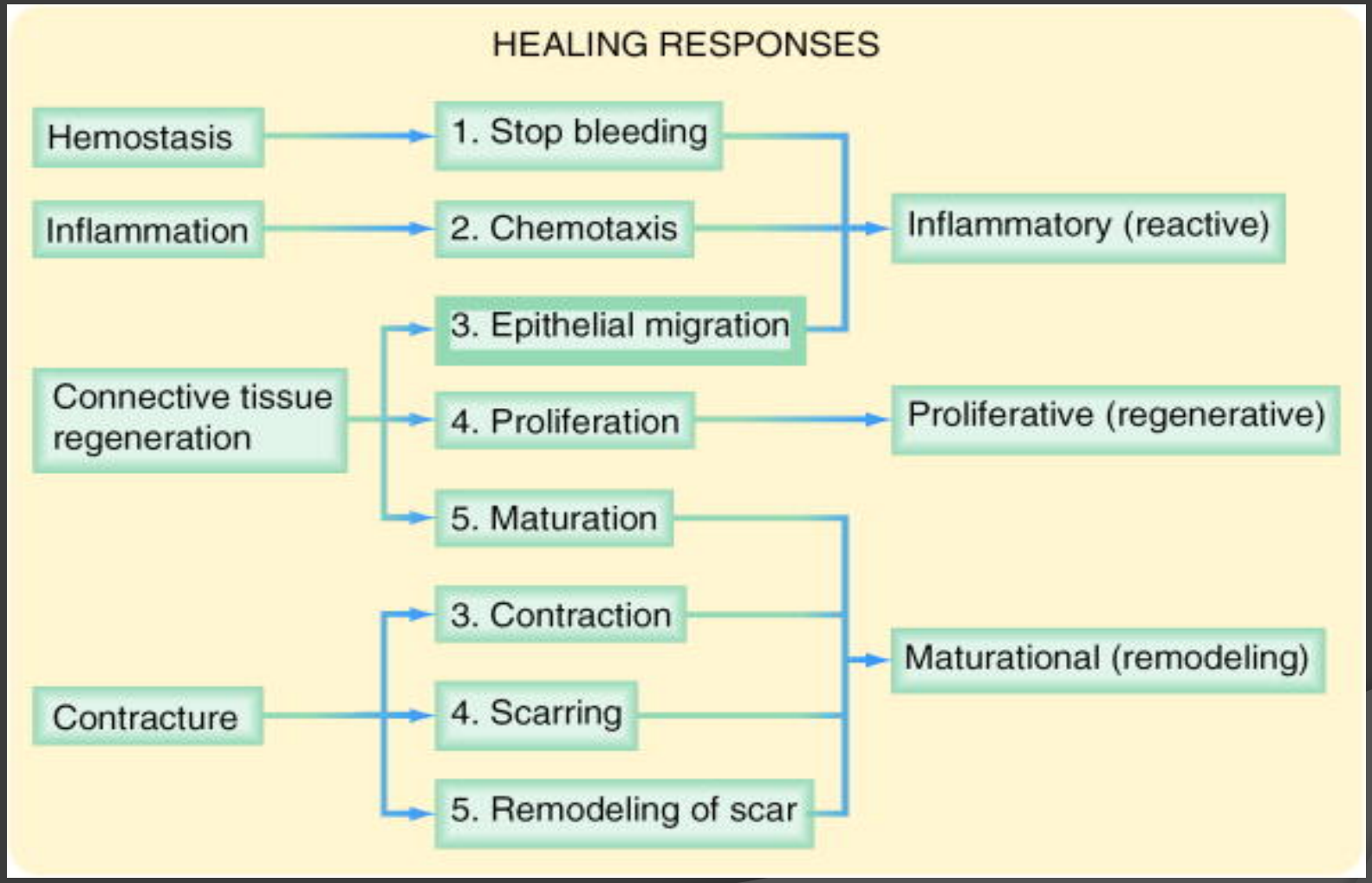
Chemical Burns

- Delayed and progressive injury
- Deceptively superficial at first
- Acid more limited (coagulation necrosis)
- Alkalis more destructive (liquefaction)
- HFI: significant necrosis, arrhythmias, hypoCa
- Removal of causative agent
- – Brush off metals and powders
- – Copious irrigation with water

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.


HEALING RESPONSES
Hemostasis
1. Stop bleeding
Inflammation
2. Chemotaxis
Inflammatory (reactive)
3. Epithelial migration
Connective tissue regeneration
4. Proliferation
Proliferative (regenerative)
5. Maturation
3. Contraction
Maturational (remodeling)
Contracture
4. Scarring
5. Remodeling of scar

Inflammatory

Substrate or reactive phase, immediate
typically **days 1-10**

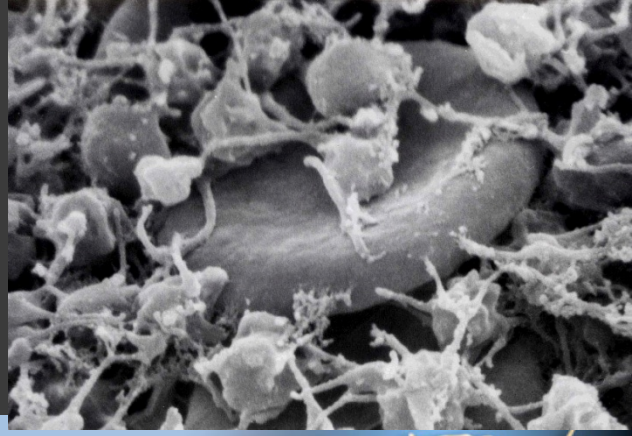
Response to **limit and prevent further injury, inflammation, hemostasis, sealing surface, removing necrotic tissue and debris, migration of cells** into wound by chemotaxis, cytokines, and growth factors

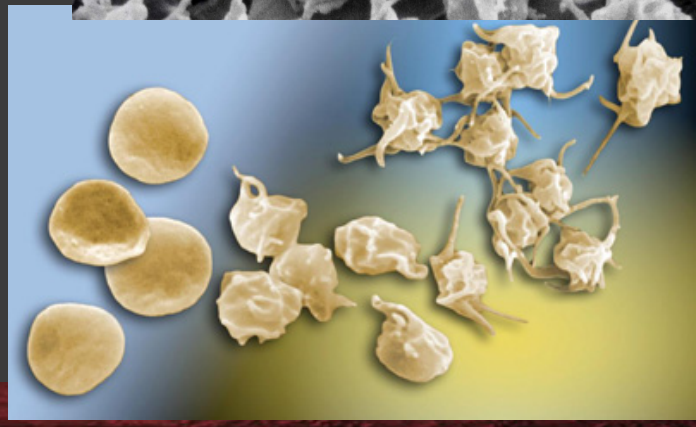
Initial intense local **vasoconstriction** of arterioles and capillaries **followed by vasodilation** and vascular permeability

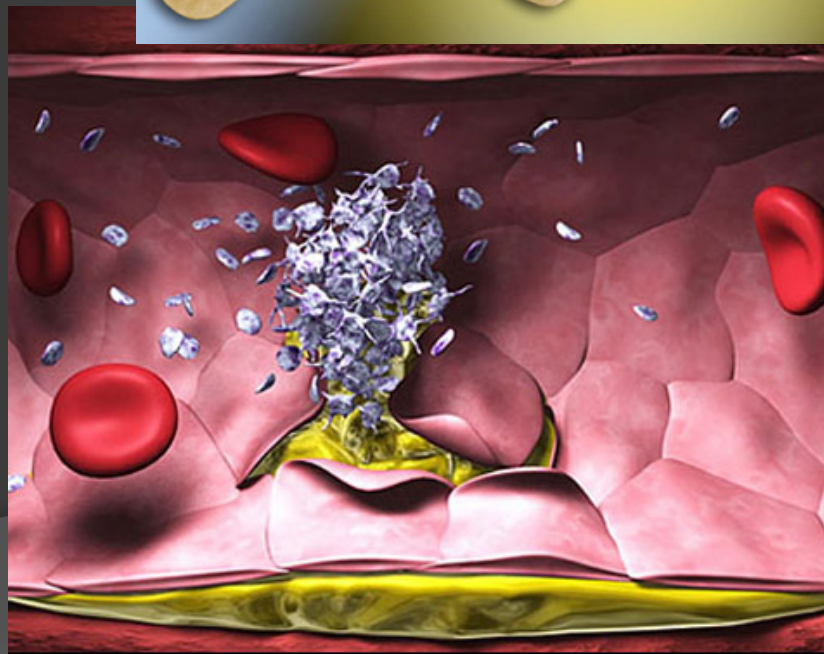
Inflammatory

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

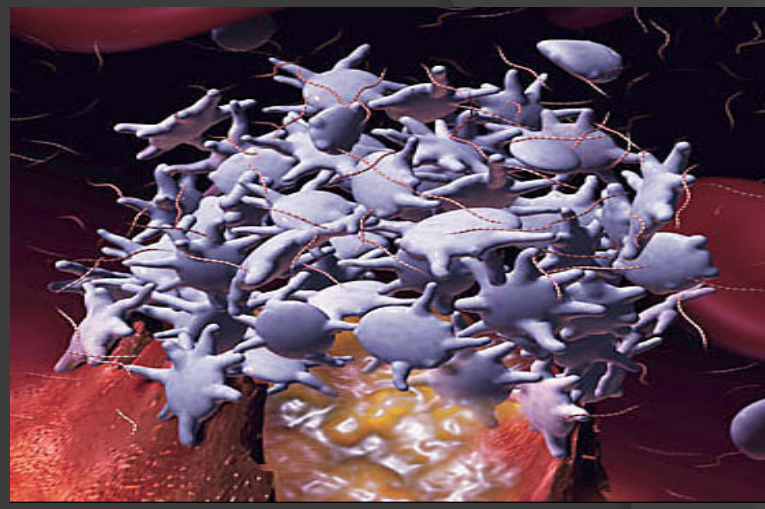
- **Plugging:** Platelet and fibrin
- **Provisional matrix**: platelets, fibrin, and fibronectin
- **Platelet aggregation**: Thromboxane (vasoconstrict), thrombin, platelet factor 4



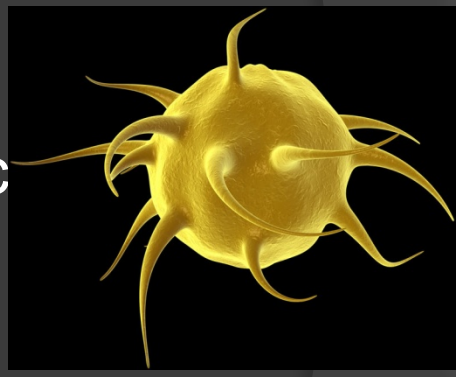


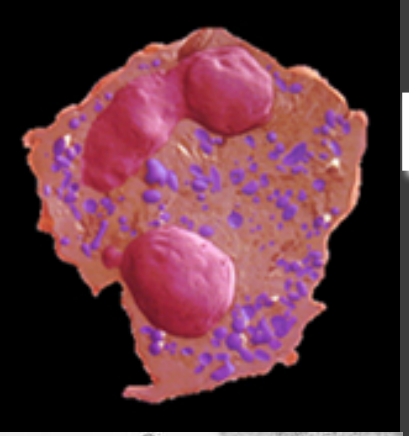


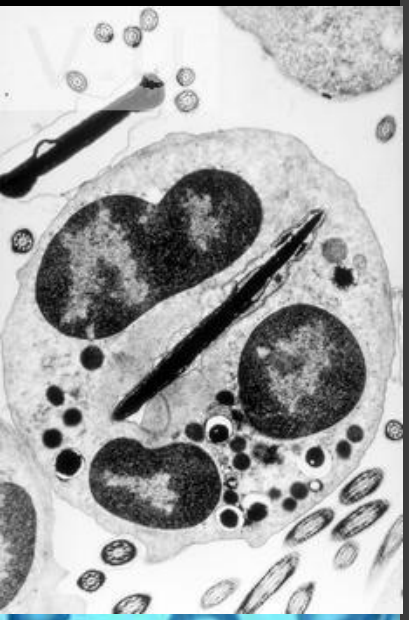
Platelets

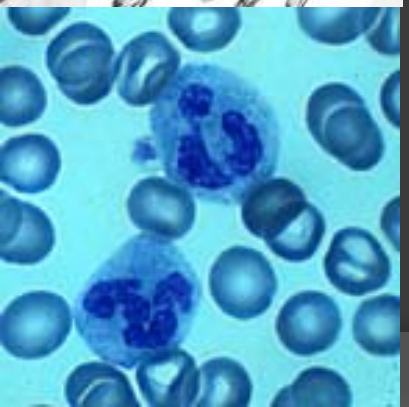


- **Alpha granules** contain:
  -platelet factor 4: aggregation
  -Beta-thrombomodulin: binds thrombin
  -PDGF: chemoattractant
  -TGF-beta: key component tissue repair
- **Dense granules** contain **vasoactive** substanc adenosine, serotonin, and calcium
- Other factors released: TXA, Platelet activate factor, Transform. growth factor alpha, Fibroblast growth factor, Beta lysin (antimicrobial), PGE2 and PGI2 (vasodilate) and PGF2 (vasoconstrict).







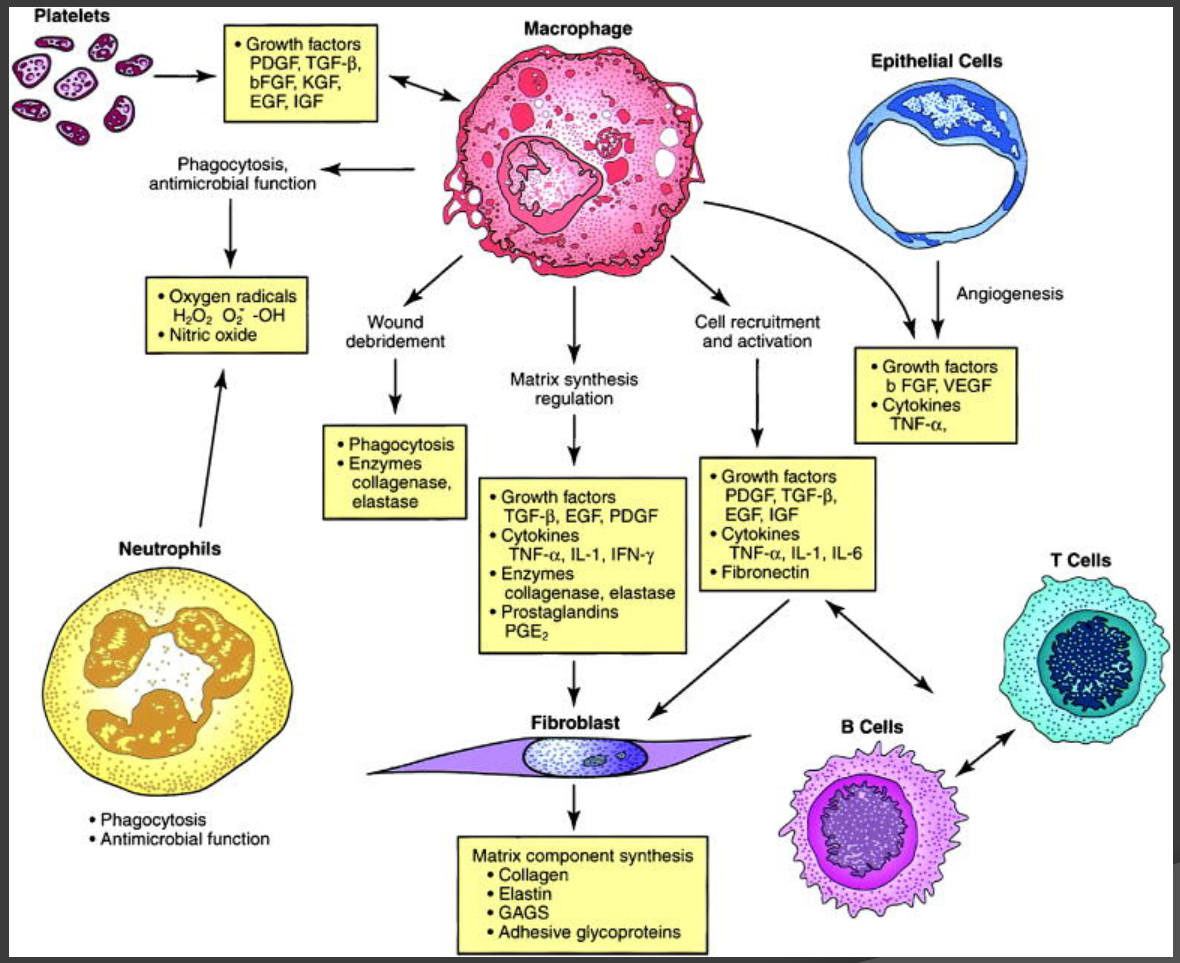


Polymorphonuclear Cells

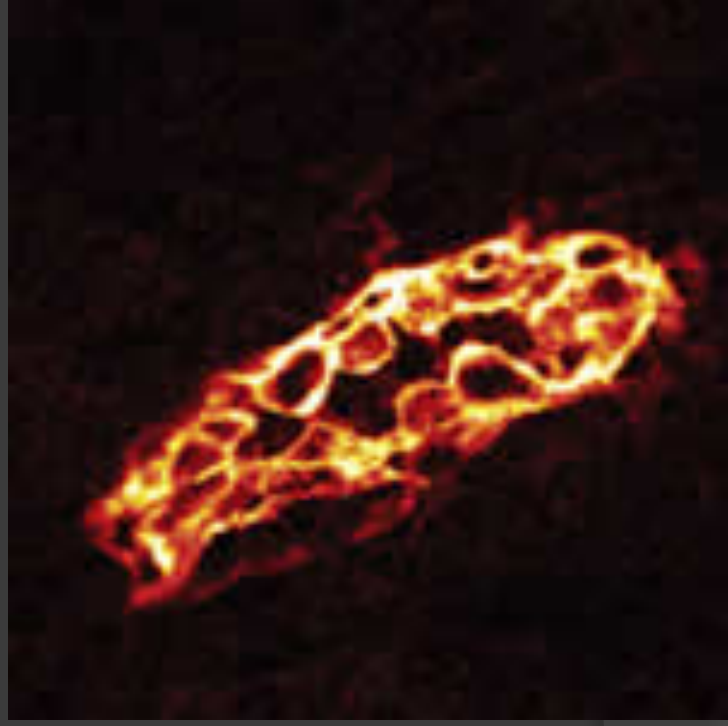
- **Chemotaxins** attract after extravasation
- Migrate through the ECM by transient interaction with **integrins**
- PMNs **scavenge**, present **antigens**, provide cytotoxicity-**free radicals** (H_2O_2)
- Migration PMNs **stops** with wound **contamination control** usually a few days
- **Persistant contaminant**: continuous influx PMN's and tissue destruction, necrosis, abscess, & systemic infection
- PMNs are **not essential** to wound healing

Macrophages Necessary

- Monocytes migrate & activate: Macrophages
- Appear when PMN's 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 Gran. Tissue

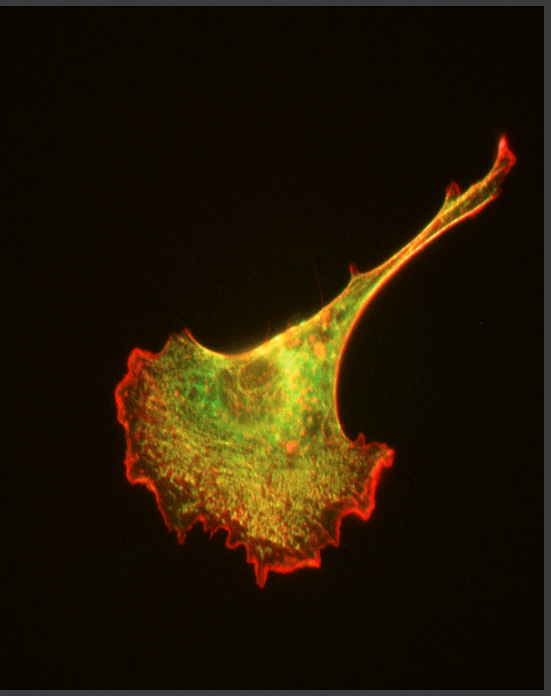

Platelets
• Growth factors PDGF, TGF-β, bFGF, KGF, EGF, IGF
Macrophage
Epithelial Cells
Phagocytosis, antimicrobial function
• Oxygen radicals H_2O_2 O_2^- -OH
• Nitric oxide
Wound debridement
Matrix synthesis regulation
Cell recruitment and activation
Angiogenesis
• Growth factors b FGF, VEGF
• Cytokines TNF-α,
• Phagocytosis
• Enzymes collagenase, elastase
• Growth factors TGF-β, EGF, PDGF
• Cytokines TNF-α, IL-1, IFN-γ
• Enzymes collagenase, elastase
• Prostaglandins PGE_2
• Growth factors PDGF, TGF-β, EGF, IGF
• Cytokines TNF-α, IL-1, IL-6
• Fibronectin
Neutrophils
T Cells
Fibroblast
B Cells
• Phagocytosis
• Antimicrobial function
Matrix component synthesis
• Collagen
• Elastin
• GAGS
• Adhesive glycoproteins

Proliferative



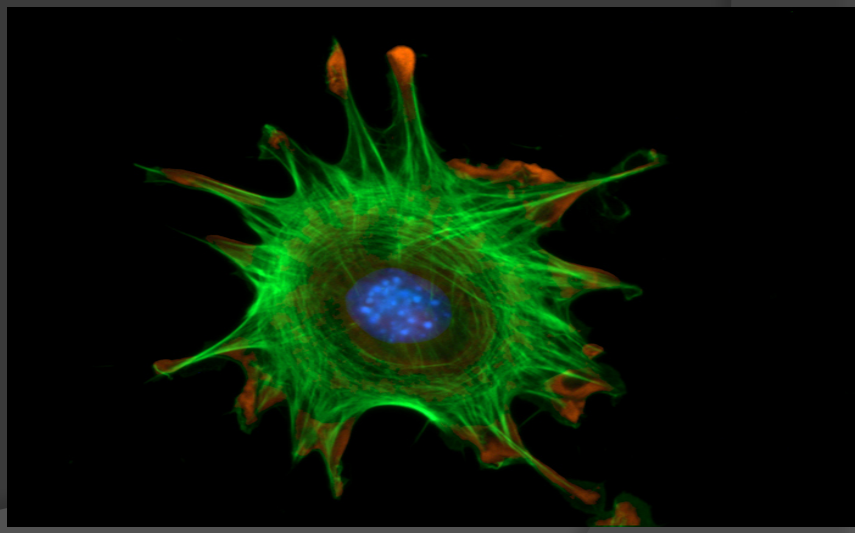
- Regenerative or Reparative
  day 5- 3 weeks
- **Angiogenesis:** endothelial cells activate & degrade Basement membrane, migrate, and divide to form more tubules
- **Granulation Tissue:** capillary ingrowth, collagen, Macrophages, Fibroblasts, Hyaluronic acid (GAG)

Proliferative Cont…



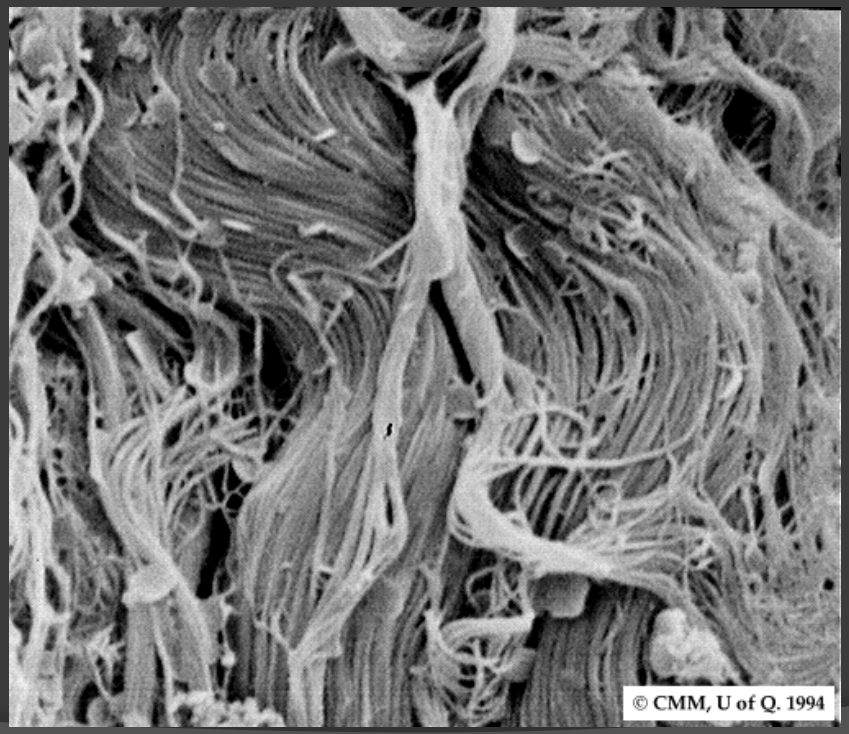
Fibroblasts
differentiate from resting mesenchymal cells in connective tissue
3-5 days migrate from ***wound edge***

- **Fibroplasia**: Fibroblasts proliferate replace **fibronectin-fibrin** with **collagen** contribute ECM



Collagen

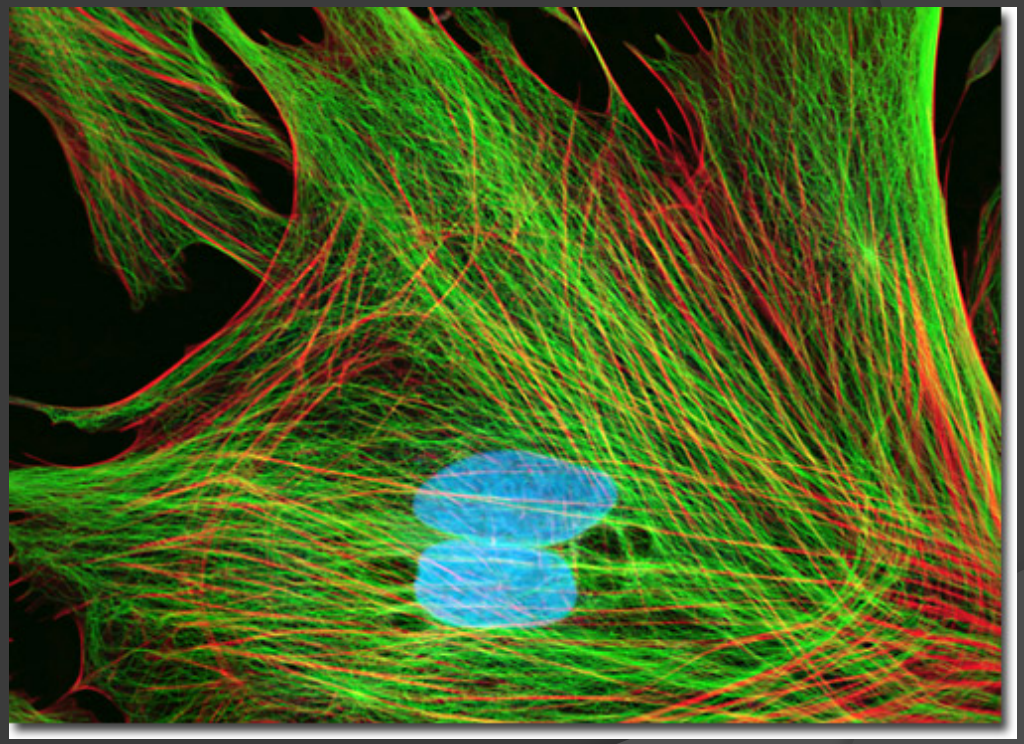
Type III predominant collagen synthesis **days 1-2**
Type I days **3-4**
Type III replaced by Type I in **3 weeks**



I **(80% skin)**	Most Common: skin, bone, tendon. **Primary type in wound healing.**
II	Cartilage
III **(20 % skin)**	**Increased Ratio** in healing wound, also blood vessels and skin
IV	Basement Membrane
V	Widespread, particularly in the cornea

Wound strength

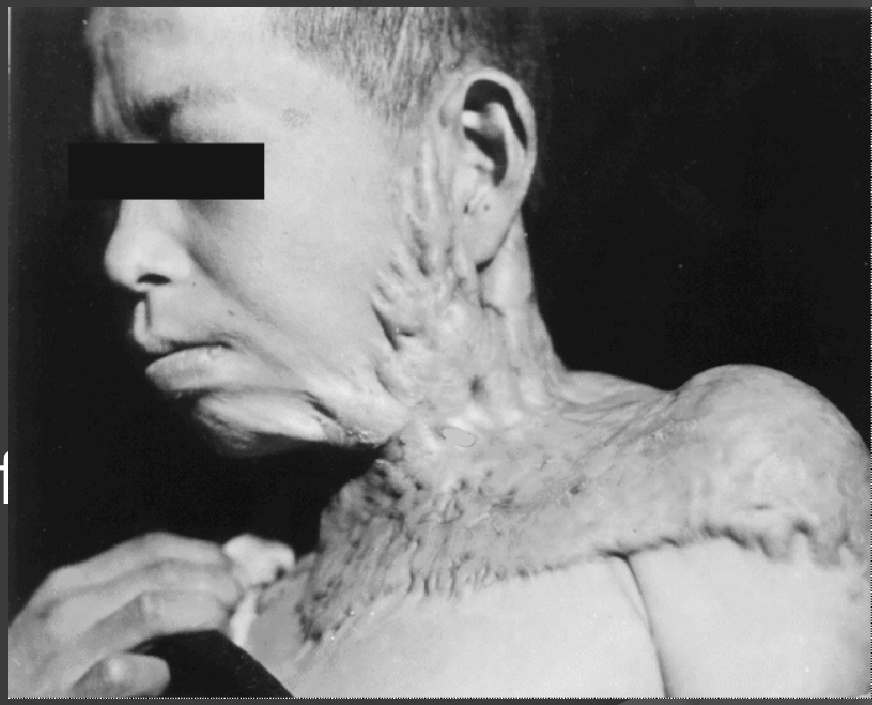
- 6 Week = 60% original, 80% final strength
- 8 Week-1 year ≈ 80% original (Max)
- Net Collagen = 6 weeks amount stays the same but cont. crosslink increase strength = maturation



Maturational

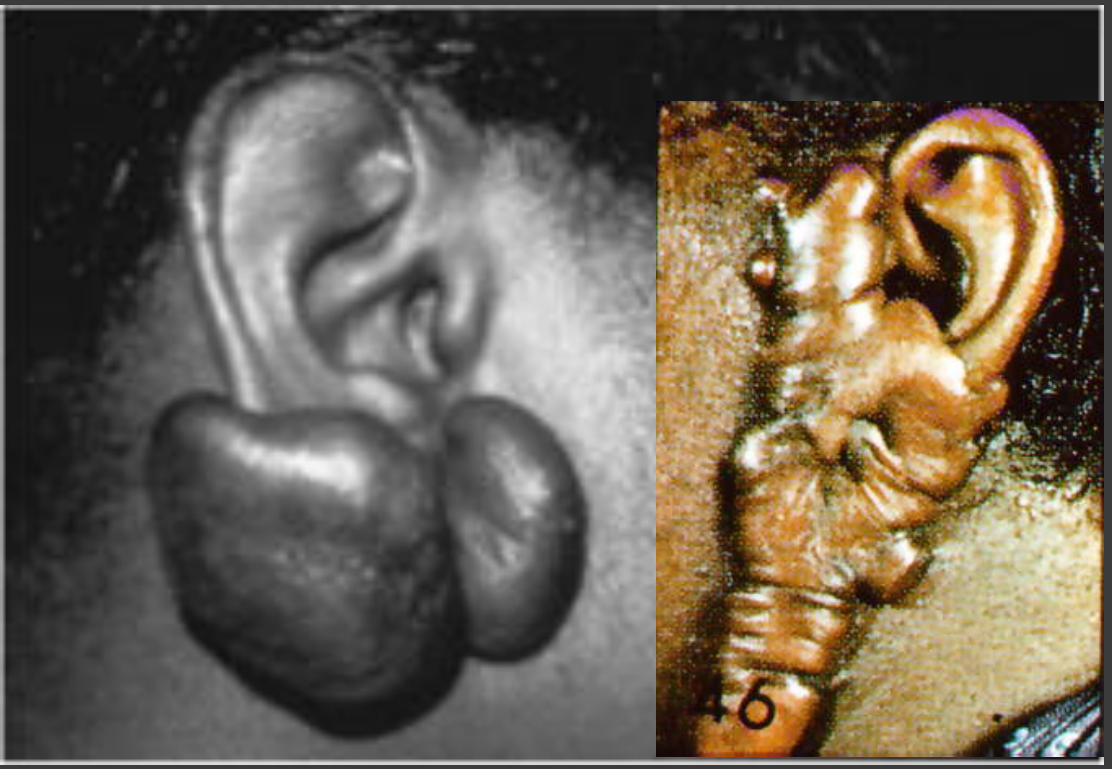
- Remodeling of wound **3 week-1+year**
- Type I replaces Type III Collagen: net amount doesn't change after 6 weeks, **organization** & crosslinking
- **Decreased vascularity**, less fibroblasts & hyaluronic acid
- Peripheral **nerves** regenerate @ **1mm/day**
- **Accelerated Wound Healing**: reopening results in **quicker healing 2nd time around**

- **Contraction**: centripetal movement of the whole thickness of surrounding skin reducing scar
- **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**: the physical constriction or limitation of function as the result of Contraction (scars across joints, mouth, eyelid)



Burn/Keloid causing contracture

Keloids: Beyond the Borders



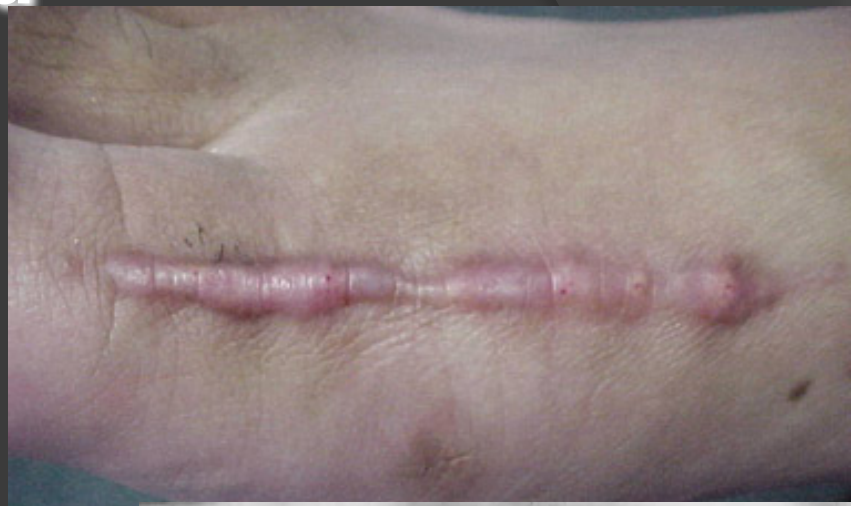
Tx: XRT, steroids, silicone sheeting, pressure, excise. **often Refractory to Tx & not preventable**

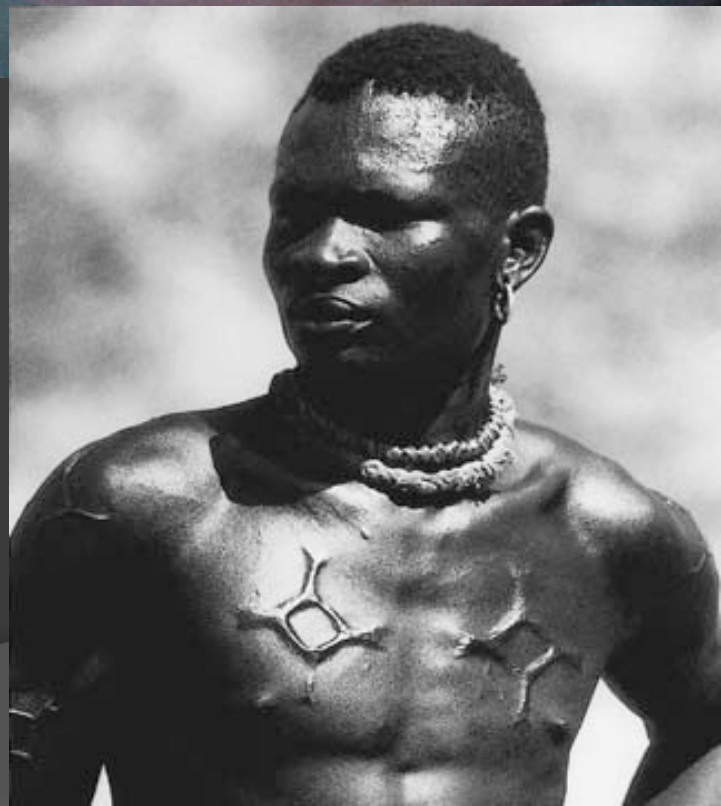
- Excess Deposition of Collagen Causes Scar Growth Beyond the Border of the Original wound

Autosomal Dominant, Darker Pigment, Often above clavicle but not always

Hypertrophic Scar: confined within

- Excess collagen deposit causing raised scar remains within the original wound confines
- Darker pigmented skin & flexc surfaces of upper torso
- Often occurs in burns or wounds that take a long time to heal, sometimes preventable
- Can regress spontaneously
- Tx: steroids, silicone, pressure garments





Impediments to Wound Healing

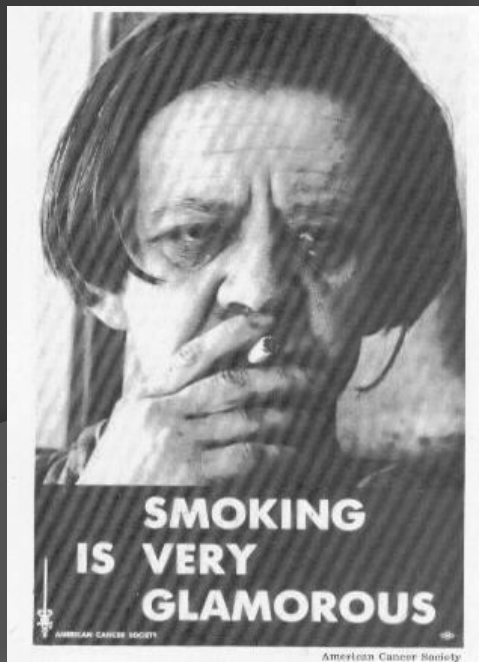
- **Bacteria**>$10^5/cm^2$: Decreased O_2 content, collagen lysis, prolonged inflammation
- **Devitalized Tissue & Foreign Body**: Retards Granulation Tissue formation and healing
- **Cytotoxic drugs**: **5FU, MTX, Cyclosporine**, **FK-506** can impair wound healing. **D-Penicillamine- inhibit collagen x-linking**
- **Chemotherapy:** no effect after 14 days
- **Radiation:** Collagen synthesis abnormal, fibrosis of vessel

More Impediments

- **Diabetes**: impedes the **early phase** response
- Malnurishment: **Albumin<3.0**, **Vit-C**
- **Smoking:** vasoconstriction, atherosclerosis, carboxyhemoglobin, decreased 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

  DENERVATION has **NO EFFECT** on Wound Healing



Diseases Assoc 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

- Thank you