

Burn injury and wound healing

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● Notes

● Important

431

SURGERY TEAM

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Burns

- Most deaths occur at home
- Causes of death: **Smoke inhalation, sepsis**, pneumonia, shock.
- More common **in elderly**: (age+BSA(body surface area) =%mortality), most with >70% die
- Risk factors for death: > 40% BSA, > 60 years, inhalation injury.

Pathophysiology of Burns:

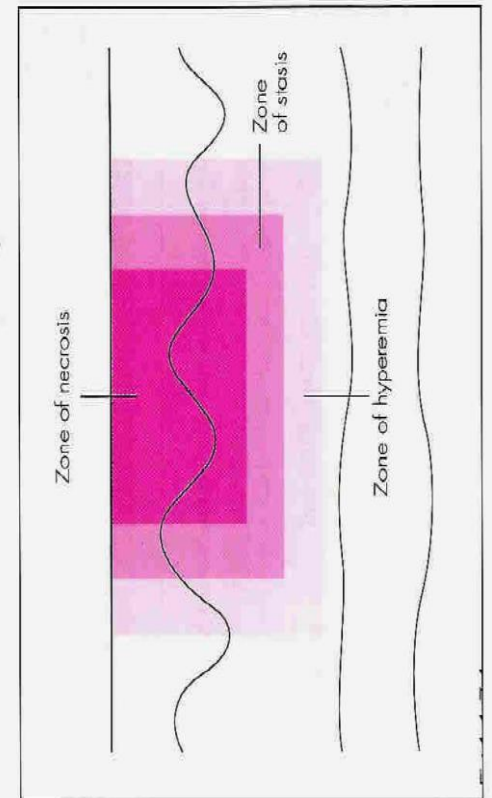
- Dynamic injuries: (Injury doesn't stop by heat removal)
 - Cellular damage at >45° C
 - Dependent on temperature and duration
- Three zones of injury
 - Central necrosis (Irreversible)
 - Zone of **stasis (at risk of necrosis)** (reversible)
 - Zone of hyperemia (inflammatory response , vasodilation)

(our aim is to direct zone of stasis and hyperemia away from necrosis , and special consideration to zone of stasis because it is at risk to develop necrosis)

Thermal injury triggers intense inflammatory response **SIRS (systemic inflammatory response syndrome)**

- Initial release of histamine, bradykinin
- Release of prostanoids, free radicals, proteases

Leading to: Hyper metabolism → Bacterial translocation → MOF (Multi-organ failure).



Burn Depth Classification

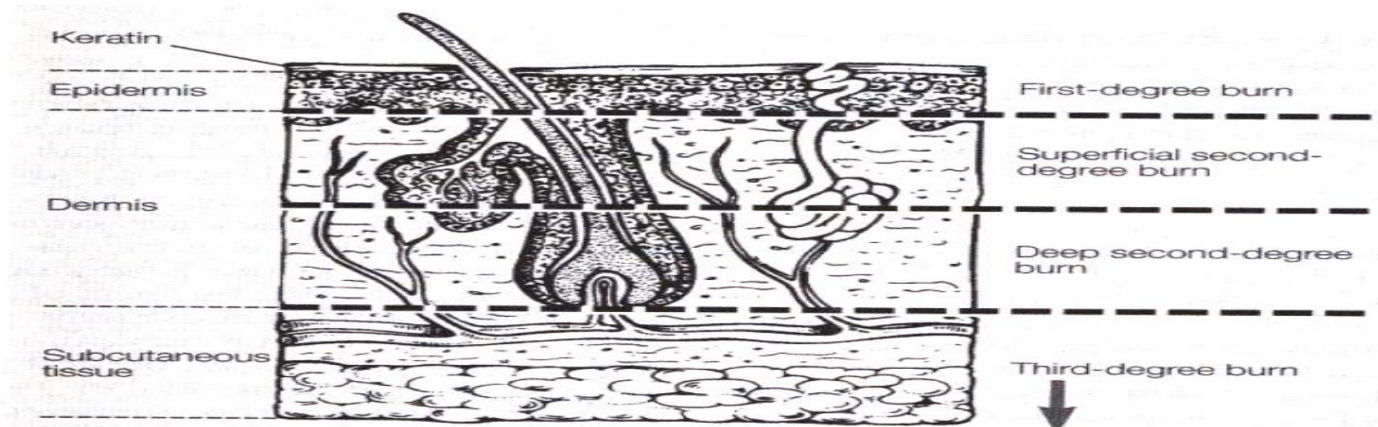


Table 2. Burn Depth Classification.

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



Mixed

1st degree (Erythema) + superficial 2nd degree (small blisters)



3rd Degree



Deep 2nd degree (large blisters)

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

How to calculate the surface area ? (should Know it very very very important)

- By using rule of nine for normal sized adult
- By using Lund- Browder chart mostly in children
- By using hand method in scattered burns

E.g. An adult burned his left leg, what is the BSA? 18% , E.g. 2: child burned his trunk? 18%

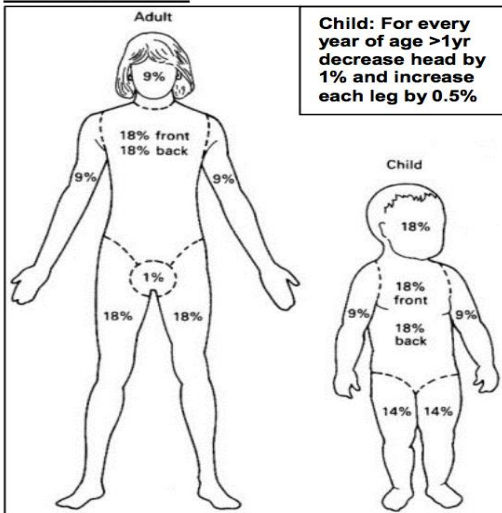
Evaluation of Burns

- 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 (by detail Hx taking)
- Well circumscribed, feet, ankles, buttocks

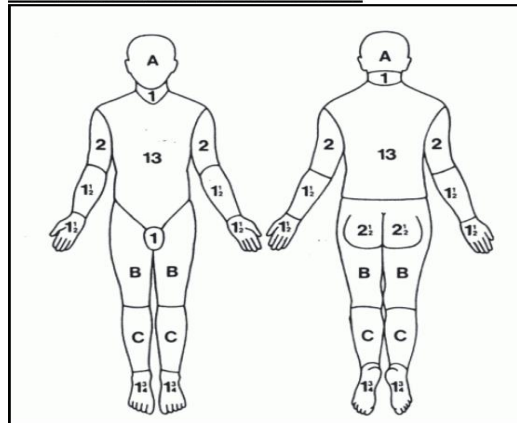
A Doppler ultrasound test uses reflected sound waves to see how blood flows through a blood vessel. It helps doctors evaluate blood flow through major arteries and veins, such as those of the arms, legs, and neck. It can show blocked or reduced blood flow through narrowing in the major arteries of the neck.

Burn Surface Area Estimation

Rule of Nines

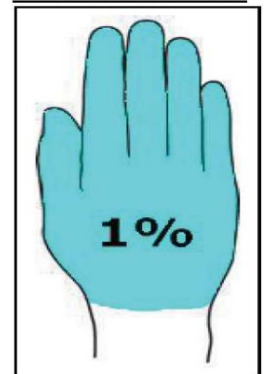


Or Lund & Browder Chart



Age	0	1	5	10	15	Adult
A	9.5	8.5	6.5	5.5	4.5	3.5
B	2.75	3.25	4	4.5	4.5	4.75
C	2.5	2.5	2.75	3	3.25	3.5

Or Hand Method



Palm and fingers of the patient = 1% TBSA

Inhalation Injury**Smoke inhalation**

- **Carbon Monoxide Poisoning**
 - CO has stronger affinity for HGB than O₂
 - Signs of CO poisoning:
 - Confusion, dizziness, HA, NV (**nausea , vomiting**), 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 (Acute respiratory distress syndrome)** within 24 hours post injury
 - **Pneumonia** may occur as late as post burn day 10

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

Parkland formula (most important formula)

- **Initial 24 hours:** Ringer's lactated (RL) solution 4 ml/kg/% burn for adults and 3 ml/kg/% burn for children. RL solution is added for maintenance for children:
 - 4 ml/kg/hour for children weighing 0–10 kg
 - 40 ml/hour +2 ml/hour for children weighing 10–20 kg
 - 60 ml/hour + 1 ml/kg/hour for children weighing 20 kg or higher
 This formula recommends no colloid in the initial 24 hours
- **Next 24 hours:** Colloids given as 20–60% of calculated plasma volume. No crystalloids. Glucose in water is added in amounts required to maintain a urinary output of 0.5–1 ml/hour in adults and 1 ml/hour in children.

(Half of the amount calculated is given in first 8 hours , other half is to be given in the next 16 hours , the counting of time start from the time of burns) E.g. patient came after 4h? Give half in the next 4h, E.g. 2 patient came after 8h? Give the entire first half.

Electrical Burns

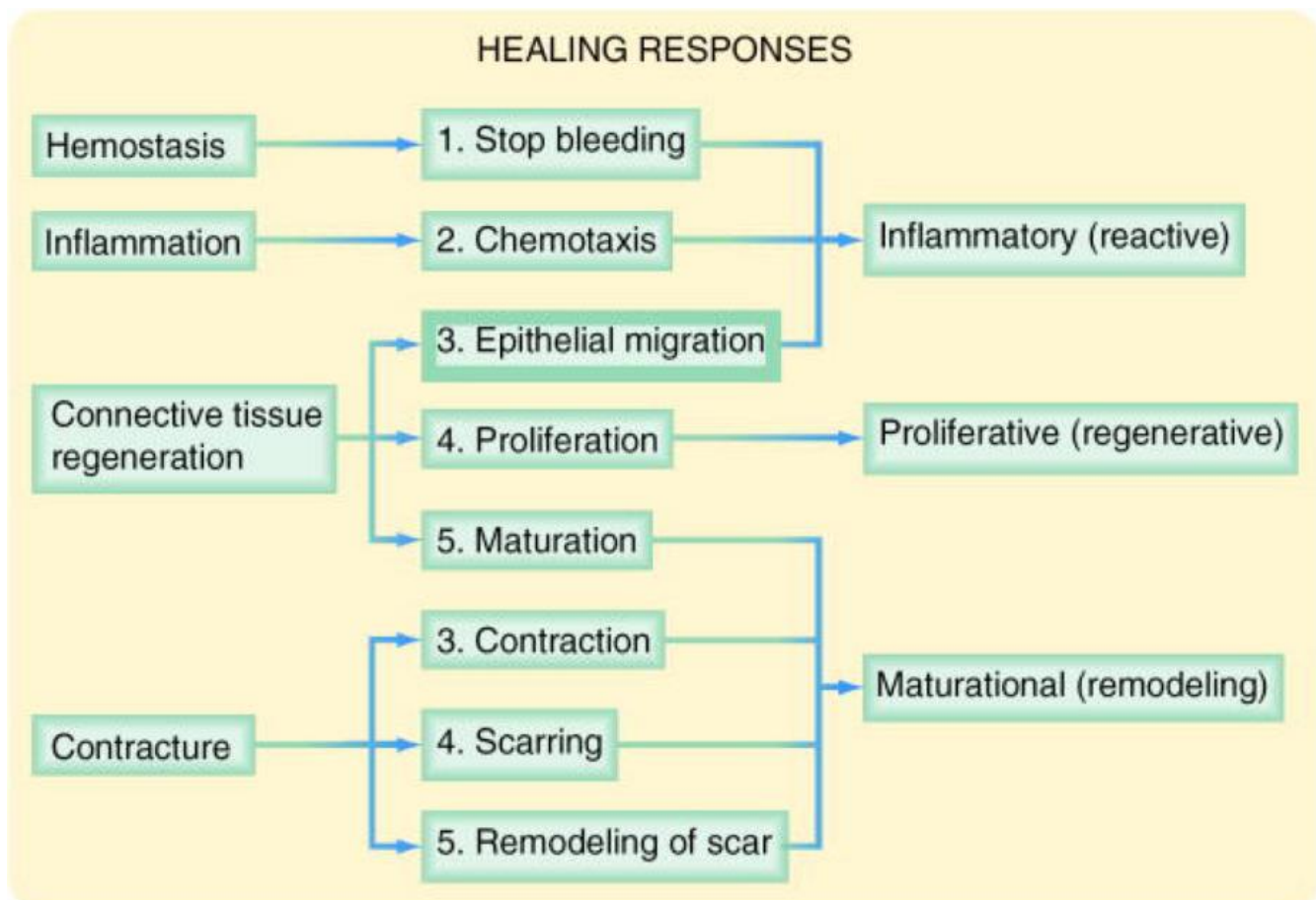
- Caused by passage of electric current
- Damage increased in small bony areas :
Fingers, feet, lower legs, forearm
- Systemic effects
 - Low voltage (<1000 V)
 - High voltage (>1000): Massive tissue damage, respiratory and cardiac arrest
- ECG, CPK, UA, monitor
- Local care often necessitates grafting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



Inflammatory Phase	Proliferative Phase	Maturation Phase
<p>Substrate or reactive phase, immediate typically days 1-10</p> <p>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</p> <p>Initial intense local vasoconstriction of arterioles and capillaries followed by vasodilation and vascular permeability.</p> <p>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</p> <ul style="list-style-type: none"> • Platelets: Alpha granules contain: <ul style="list-style-type: none"> ◦ TGF-beta: key component tissue repair • Polymorphonuclear Cells <ul style="list-style-type: none"> ◦ PMNs are <u>not</u> essential to wound healing. • Macrophages: <ul style="list-style-type: none"> ◦ Essential to wound healing. 	<p>Regenerative or Reparative day 5- 3 weeks</p> <p>Angiogenesis: endothelial cells activate & degrade Basement membrane, migrate, and divide to form more tubules</p> <p>Granulation Tissue: capillary ingrowth, collagen, Macrophages, Fibroblasts, Hyaluronic acid (GAG)</p> <p>Fibroblasts differentiate from resting mesenchymal cells in connective tissue 3-5 days migrate from wound edge</p> <p>Fibroplasia: Fibroblasts proliferate replace fibronectin-fibrin with collagen contribute ECM</p> <p>Collagen:</p> <ul style="list-style-type: none"> - Type III predominant collagen synthesis days 1-2 - Type I days 3-4 - Type III replaced by Type I in 3 weeks <p>Type I:</p> <ul style="list-style-type: none"> - (80% skin) - Most Common: skin, bone, tendon. Primary type in wound healing. <p>Type III:</p> <ul style="list-style-type: none"> - (20 % skin) - Increased Ratio in healing wound, also blood vessels and skin 	<p>Remodeling of wound 3 week-1+year</p> <p>Type I replaces Type III Collagen: net amount doesn't change after 6 weeks, organization & crosslinking</p> <p>Decreased vascularity, less fibroblasts & hyaluronic acid</p> <p>Peripheral nerves regenerate @ 1mm/day</p> <p>Accelerated Wound Healing: reopening results in quicker healing 2nd time around</p>

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

Wound Healing Disorder

(we mention here only what doctor focus on , go back to the lecture to read other types)

Keloids (Beyond the Border)	Hypertrophic Scar (confined within)
<p>Excess Deposition of Collagen Causes Scar Growth Beyond the Border of the Original wound</p> <p>Autosomal Dominant, Darker Pigment, Often above clavicle but not always</p> <p>Tx: XRT, steroids, silicone sheeting, pressure, excise. often Refractory to Tx & not preventable</p>	<p>Excess collagen deposit causing raised scar remains within the original wound confines</p> <p>Darker pigmented skin & flexor surfaces of upper torso</p> <p>Often occurs in burns or wounds that take a long time to heal, sometimes preventable</p> <p>Can regress spontaneously</p> <p>Tx: steroids, silicone, pressure garments</p>

Questions:

- 1- A patient came to the ER after sustaining a flame burn to his entire left upper arm, the burn area on this patient will be estimated as:
 - a. 4.5%
 - b. 9%
 - c. 15%
 - d. 18%
- 2- In a burn injury, which one of the following describes an area of potentially reversible cell damage:
 - a. Zone of necrosis
 - b. Zone of stasis
 - c. Zone of hyperemia
 - d. Zone of calcification
- 3- In wound healing, the key cell in inflammatory phase is:
 - a. Fibroblast
 - b. Macrophages
 - c. PMN cells
 - d. T-lymphocytes

Answers:

- b
- b
- b