

# Burns and Wound Healing



**COLOR GUIDE:** • Females' Notes • Males' Notes • Important • Additional

# <u>Objectives</u>

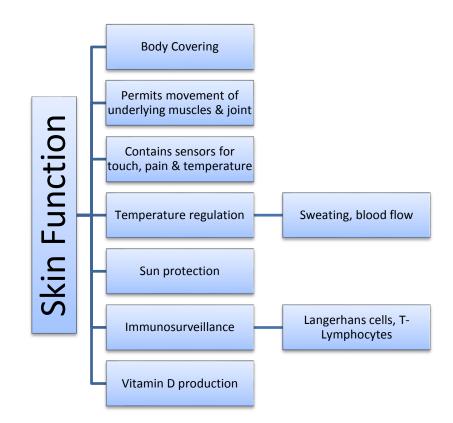


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# Introduction



# <u>Skin Layers:</u>

• <u>Epidermis</u>

Outer layer contains the stratum corneum (outermost layer consist of dead cells

corneocytes composed of 15-20 layer of flattened cells with no nuclei)

- The rate-limiting step in dermal or percutaneous absorption is diffusion through the epidermis.
- <u>Dermis</u>
  - Much <u>thicker</u> than epidermis.
  - True skin & is the main natural protection against trauma.
  - Contains the appendages:
    <u>Sweat Glands, Sebaceous gland, blood vessels, hair and nails</u>.

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#### • <u>Subcutaneous Layer</u>

• Contains the fatty tissues, which cushion & insulate.

# Burns

## **Classification:**

- 1. Thermal (It's further classified to: Scald, Flame or Contact Burn)
- 2. Chemical
- 3. Electrical
- 4. Friction injuries (Mechanical)

## Causes of death:

Smoke inhalation, sepsis (Major cause), pneumonia, and shock (Hypovolemic).

- Most deaths occur at home
- More common in elderly
- (Age + BSA [Burn Surface Area]=%mortality). Most with > 70% die.
- <u>Risk factors for death:</u>
  - > 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
  - <u>Central necrosis (irreversible damage)</u>
  - Zone of stasis (at risk of necrosis)
  - Zone of hyperemia
- Thermal injury triggers intense inflammatory response SIRS (systemic Inflammatory Response Syndrome characterized by bacterial translocation to the blood causing sepsis, hypermetabolism ,break down of muscle, loss of papillae in the intestine)

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- o Initial release of histamine, bradykinin. (Vasodilators)
- Release of prostanoids\*, free radicals, proteases.
  \*It is consisting of prostaglandin, thromboxane and prostacyclin which is essential in the resolution phase of inflammation.

#### Note:

When the Body Surface Area is more than 20%, SIRS (Systemic inflammatory response syndrome) will be activated. This may lead to:

1. Bacterial translocation leading to **septic shock**.

2. Hypoperfusion to vital organs because of **vasodilation** leading to **MOF(multiple organ dysfunction)** 

#### 3. **Death**.

Note:

Central zone of necrosis

zone of necrosis.

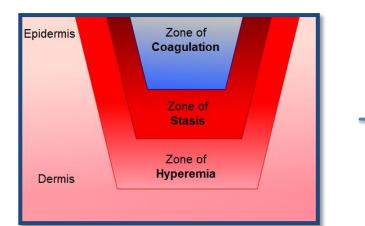
*Outer zone of hyperemia:* 

Intermediate zone of Stasis:

(Coagulation): <u>IRREVERSIBLE</u>

<u>REVERSIBLE.</u> This is the zone that we aim to save by directing it to the zone of hyperemia instead of the

Inflammatory response, vasodilation



# **Burn Depth Classification**

Table 2. Burn Depth Classification.				
Depth First-degree:	<b>Histology</b> Epidermis only	Appearance Erythema; blanches with pressure	Sensation Intact; mild to moderate pain	Healing 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 <u>: not painfu</u> l (although surrounding areas of second-degree burns are painful)	Does not heal; severe scarring and contractures

First-degree burn examples>> sunburn, steam burn usually it will heal by itself. Third-degree burn>> the dr. mentioned that it'll heal but it takes very long time. Forth-degree burn>> involves the muscle and bone.

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#### Note:

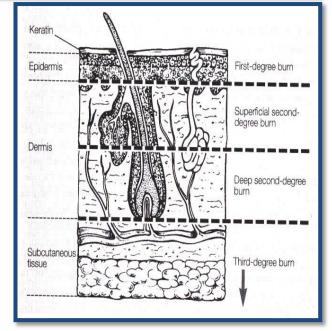
The Dr. mentioned that it's not necessary to know all the characteristics of each degree. In the previous table, the underlined and circled words are the ones the doctor mentioned. Also, to make it easier, he mentioned that if you see a(n):

- 1. Erythema only, this means it's a 1<sup>st</sup> degree burn.
- 2. Blister, this means it's a 2<sup>nd</sup> degree burn.

*3. White appearance, this means it's a 3<sup>rd</sup> degree burn.* 

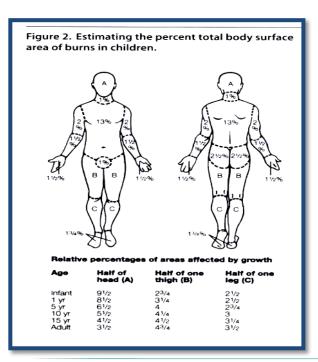
#### **Determining Extent of Injury:**

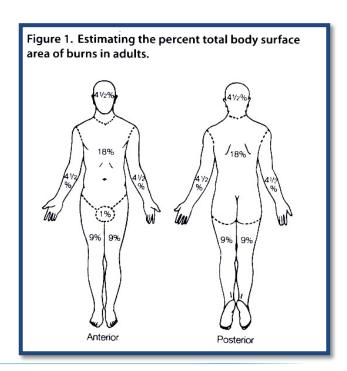
- Burn extent determines therapy and prognosis
- Burn size estimate often inaccurate
- Extent of injury described using percentage of total body surface area that is burned (TBSA).
- For patients > 9 "rule of nines" may be used. (The body's divided into different parts with different percentages as shown below in the <u>Lund and Browder Chart</u>. So, for



example, if a patient comes to you with a burned left arm. His TBSA would be 9%, because it's the whole arm [anterior and posterior] = [4.5+4.5])

- For small or scattered burns, the patient's palm covers 1%. (So you look at the patient's hand and you estimate).
- With young children proportions differ.





## **Compartment Syndrome:**

- It's the compression of nerves, blood vessels, and muscles inside a closed space (compartment) within the body.
- This leads to tissue death from lack of oxygenation due to the blood vessels being compressed by the raised pressure within the compartment.
- You must always look for *circumferential burns* around the chest, abdomen, limbs, etc.... and perform an *Escharotomy* to release the pressure.

#### Note:

An eschar is a slough or piece of dead tissue (skin). When burns occur, eschars will eventually form and compress the surrounding nerves, blood vessels and muscles → Compartment Syndrome. This is why we do an escharotomy.

### **Evaluation of Burns:**

- Look for circumferential (all over the limb or the affected part, like a circle) 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

(Circumferential burns are very imp because the skin becomes leathery in appearance "contacted", loses its' elasticity, in addition to generalized edema which leads to compression of the arteries which causes compartment syndrome).

# Inhalation of Injury

## Smoke Inhalation:

#### Carbon Monoxide Poisoning

- CO has stronger affinity for HGB than O2
- Signs of CO poisoning:
  Confusion, dizziness, HA, NV, flushed skin.
- <u>Treatment: 100% FiO2 (</u>FiO2>> fraction of inspired oxygen).

#### > Upper Airway Obstruction

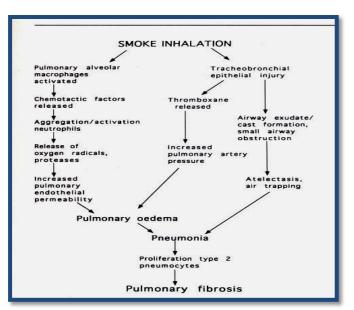
- o <u>Common in head and neck burns</u> and smoke inhalation
- o Edema continues at least 24 hours
- Protect airway with intubation (patient with head and neck burn as a prophylaxis do intubation cause once they develop the edema we can't intubate them)
- 0
- o Edema usually decreases by post burn day 3

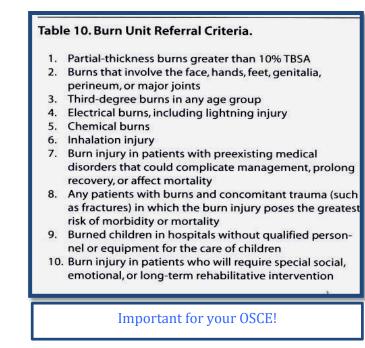
#### > Pulmonary Injury from Chemical Inhalation

- o Develops ARDS within 24 hours post injury
- Pneumonia may occur as late as post burn day 10

#### > Inflammation and systemic reactions

▶ Poisoning: When fire affects furniture (flame burn in closed space)
 → toxins get released into the air → inhaling these toxins affects the lungs directly causing "inflammation pneumonitis" and later pneumonia. There could also be systemic poisoning due to inhaled fumes like cyanide.





## Fluid Resuscitation:

- Hypovolemia is a major cause of death.
- Massive transudation of fluids from vessels due to increased permeability will cause edema which intensifies over 8-48 hours.
- Goal: preservation of organ perfusion and urine output.

#### Note:

How much IV fluids should we give a burned patient? (If needed) Parkland formula (Crystalloid) is the most common one used. Formula: (<u>4cc</u> X <u>(%of burn)</u> X <u>Weight of patient</u>) = total amount of fluid needed in 24 hours. Half of the amount calculated is given in the **first 8 hours**; the other half is to be given in the next 16 hours (start counting from **the time of burn** NOT the time of patient's admission to ER)

So, for example, a patient comes in and tells you he was burned <u>3 hours ago</u>. You give him half of the amount calculated for <u>5 more hours only</u>. Then you give him the other half in the next 16 hours.

# **Electrical Burns**

- 4<sup>th</sup> degree if the current passes through the body
- Caused by passage of electric current
- Damage increased in small bony areas: Fingers, feet, lower legs, forearm
- Systemic effects
  - Low voltage (<1000 V): May cause arrythmias
  - High voltage (>1000): Massive tissue damage, respiratory and cardiac arrest
- ECG, CPK, UA, monitor
- Local care often necessitates grafting and amputation

#### Note:

Electrical burns are the only type of burns that have an entry and an exit point. They may be minimal on the surface; we should check the muscles and bones for any injuries. Damage is due to resistance, which generates heat, that's why it's common in small bones. (Bones have the highest resistance in the body). Here, the treatment is <u>fasciotomy.</u>

# **Chemical Burns**

- 4<sup>th</sup> degree if it reaches the fat and muscle.
- Delayed and progressive injury. Deceptively superficial at first.
- Acid more limited (coagulation necrosis)
- Alkalis more destructive (<u>liquefaction</u>)
- HFl (Hydrofluric acid): Significant necrosis, arrhythmias.(Worst of all, as it contains both Acid and Alkalis).
- Cause Hypo Calcemia
  - Treatment of all chemical burns: Removal of causative agent, brush off metals and powder & 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.

Has 3 phases: Inflammatory, proliferative, and maturational phases. (The important things to know are: stage, aim of stage, duration of stage, & main cells in that stage)

### 1. <u>Inflammatory Phase</u> (Stop bleeding + limit injury)

- Substrate or reactive phase, immediate: typically occurs in 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
- Tissue injury & blood vessel damage exposure of subendothelial
  Collagen (responsible for plaelelets addheions) to platelets and vWF activates the coagulation pathway.
- **Plugging:** Platelet and fibrin
- **Provisional matrix**: platelets, fibrin, and fibronectin
- **Platelet aggregation**: <u>Thromboxane</u> (vasoconstriction), thrombin, platelet factor IV.

## A.<u>Plaetelets</u>

- Alpha granules contain:
  - Platelet factor 4: aggregation
  - $\circ$  Beta-thrombomodulin: binds
  - o Thrombin
  - PDGF: chemoattractant
  - o **<u>TGF-beta</u>**: key component tissue repair
- **Dense granules** contain <u>vasoactive</u> substances: adenosine, serotonin, and calcium.
- Other factors released: TXA, Platelet activate factor, Transform. growth factor alpha, Fibroblast growth factor, Beta lysin (antimicrobial), PGE2 and PGI2 (vasodilator) and PGF2 (vasoconstrictor).

## B. Polymorphonuclear Cells

- Chemotoxins attract after extravasation.
- Neutrophils are the first cells to appear.
- Migrate through the ECM by transient interaction with integrins.
- PMNs scavenge, present antigens, provide cytotoxicity-free radicals (H2O2).
- 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 <u>not essential</u> to wound healing.

# C. <u>Macrophages</u> (Most imp cells in this phase)

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

## 2. Proliferative Phase

- Regenerative or Reparative. <u>Typically occurs in day 5 week 3.</u>
- **Angiogenesis**: Endothelial cells activate & degrade basement membrane, migrate, and divide to form more tubules.
- **Granulation Tissue**: capillary ingrowth, collagen, Macrophages, Fibroblasts, Hyaluronic acid (GAG

## A. 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

## B.<u>Collagen</u>

Ι	80% of the skin. Found mostly in: skin, bone, and tendons.		
	<u>Primary type in wound healing</u> .		
II	Cartilage		
III	20% of the skin. Increased ratio in wound healing. Also found in blood vessels.		
IV	Basement membrane		
V	Widespread, particularly in the cornea		

#### When wounded:

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

#### Further explanation:

In a normal person, type 1 to type 3 ratio is 3:1. When wounded, in the first 1-2 days, the ratio of type 1 to type 3 becomes 1:3. The ratio will eventually go back to normal in 3 weeks.

# C. Wound Strength

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

## 3. Maturational Phase

- 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

# Abnormalities

• **Contracture**: the physical constriction or limitation of function as the result of Contraction (scars across joints, mouth, eyelid)

#### • Keloids: Beyond the Borders

- Excess deposition of collagen causes scar growth beyond the border of the original wound .
- Tx: XRT, steroids, silicone sheeting, pressure, excise. Often Refractory to Tx & not preventable
- Autosomal Dominant, affects darker pigmented skin. Often above clavicle but not always.

#### • Hypertrophic Scar: <u>Confined Within</u> 2 (Differs from Keloids)

• Excess collagen deposit causing raised scar remains within the original wound <u>confined.</u>

#### • Darker pigmented skin & flexor surfaces of upper torso

• Often occurs in burns or wounds <u>that take a long time to heal</u>, sometimes <u>preventable</u>

- o Can regress spontaneously
- Tx: steroids, silicone, pressure garments
- Surgical excision makes it worse.

# Impediments -Things that delay- wound healing

- **Bacteria**>105/cm2: Decreased O2 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
- **Diabetes**: impedes the early phase response
- Malnurishment: Albumin<3.0, Vit-C
- **Smoking**: vasoconstriction, atherosclerosis, carboxyhemoglobin, decreased O2 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 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
- $\ast$

#### SUMMARY (quick hits =))

- Burns classified into thermal, chemical, electrical and friction injuries.
- Zones of injury central necrosis, zones of stasis, zone of hyperemia.
- Sepsis major cause of death in burn patients

• Compartment syndrome: compression of nerves blood vessels, and muscles inside a closed space within the body.

- Blisters always seen in 2 degree burn.
- Scattered burns calculated by the palm of the patient is 1 %.
- Types of chemical burns acids and alkaline.
- Neutrophils are the first cells to arrive at the site of injury.
- Macrophages are the most imp. Cells in the inflammatory phase of wound healing.
- The proliferative phase depend on fibroblasts.

# Surgical Recall

# <u>Chapter</u> 39

#### Burns

Define: TBSA

STSG

Are acid or alkali chemical burns more serious?

Why are electrical burns so dangerous? Total Body Surface Area

Split Thickness Skin Graft

In general, **ALKALI** burns are more serious because the body cannot buffer the alkali, thus allowing them to burn for much longer

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

Define level of burn injury	re.		
First-degree burns	Epidennis only		
Second-degree burns	Epidermis and varying levels of dermis		
Third-degree burns	A.k.a. "full thickness"; all layers of the skin including the entire dermis (Think: "getting the third degree")		
Fourth-degree burns	Burn injury into bone or muscle		
How do first-degree burns present?	Painful, dry, red areas that do not form blisters (think of sunburn)		
How do second-degree bur present?	ns Painful, hypersensitive, swollen, mottled areas with <b>blisters</b> and open weeping surfaces		
How do third-degree burn present?	s Painless, insensate, swollen, dry, mottled white, and charred areas; often described as dried leather		
What is the major clinical difference between second and third-degree burns?	Third-degree burns are painless, and second-degree burns are painful 76		
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What is the "rule of nines"?	In an adult, the total body surface area that is burned can be estimated by the following: Each upper limb = $9\%$ Each lower limb = $18\%$ Anterior and posterior trunk = $18\%$ each Head and neck = $9\%$ Perineum and genitalia = $1\%$		
What is the "rule of the palm"?	Surface area of the patient's palm is ≈1% of the TBSA used for estimating size of small burns		
What is the burn center referral criteria for the following? Second-degree burns	>20% TBSA		
Third-degree burns	d-degree burns >5% TBSA Second degree >10% TBSA in children and the elderly Any burns involving the face, hands, feet, or perineum Any burns with inhalation injury Any burns with associated trauma Any clectrical burns		
What is the treatment of first-degree burns?	Keep clean, $\pm$ Neosporin <sup>®</sup> , pain meds		
What is the treatment of second-degree burns?	Remove blisters; apply antibiotic ointment (usually Silvadene <sup>®</sup> ) and dressing; pain meds Most second-degree burns do not require skin grafting (epidermis grows from hair follicles and from margins)		
What is the treatment of third-degree burns?	<b>f</b> Early excision of eschar (within first week postburn) and STSG		
Why is glucose-containin IVF contraindicated in b patients in the first 24 he postburn?	urn on its own because of the stress response		
What are the common organisms found in burn wound infections?	Staphylococcus aureus, Pseudomonas, Streptococcus, Candida albicans		

## 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

