Burn injury and wound healing

Objectives:

1- To know basic principles about wounds.
2- Classification of wounds and classes of operative wounds.
3- Factors affecting wound healing and the phases of it.
4- Collagen types
5- Scars and pressure sores
6- Burns

Resources:

- Davidson's.
- 436 doctors slides.
- Surgical recall.
- 435' team work

Done by: Rana barasain, Rayan alqarni, Qais almuhaideb, Ibrahim fetyani
Leaders: Heba Alnasser, Jawaher Abanumy, Mohammed Habib, Mohammad Al-Mutlaq
Revised by: Maha alghamdi

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Color index:

Notes , Important , Extra , Davidson's

Editing file

Feedback

Wounds, scars and wound healing davidson page 92

Wound: is a disruption of normal anatomical structure, function and relations as a result of intentional or unintentional injuries. Regardless of causation or tissue type, wound healing presents with identical biochemical and physiologic processes, though wound healing may vary in timing and intensity. Classified as Acute vs. Chronic.

- Wound healing main goal is restoration of integrity and continuity of injured tissue to re-establish homeostasis of that tissue and 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

Classification :

By type	By timing	By abnormal healing
1- Primary 2- Delayed primary 3- Secondary 4- Partial thickness wound healing	1-Acute: first 6 weeks 2-Chronic End result: the scar is formed of collagen	1- Overgrowth (Hypertrophic (more synthesis of collagen) vs. Keloid) 2- Undergrowth (Chronic unstable wound (like diabetic wounds)) 3- Abnormal pigmentation 4- Contour abnormality

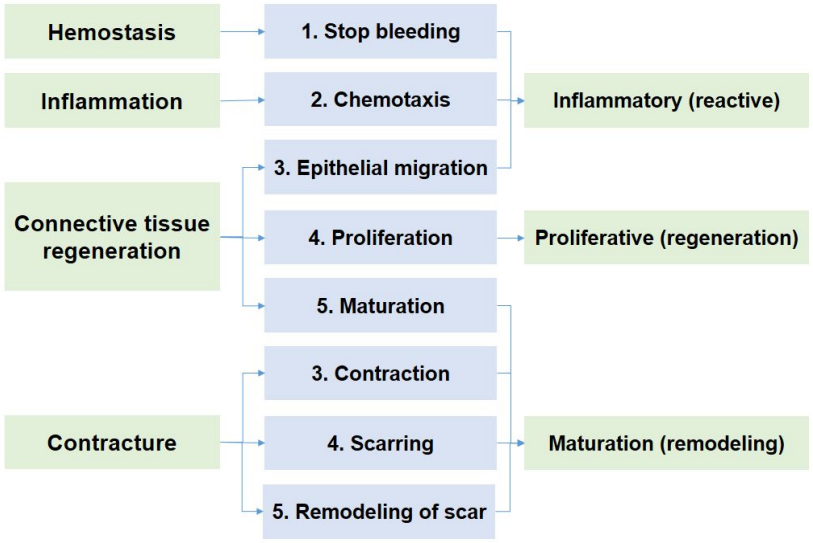
Classification of wound closure :

Primary healing (1st intention)	Secondary healing (2nd intention)	Tertiary healing (3rd intention)
- Primary closure. - Within hours of repairing full thickness surgical incision. (As soon as you suture the wound) - Result in mortality of minimal number of cellular constituents. -Usually there's 2 edges and you suture these edges to approximate them.	-Wound left open to heal by processes of granulation contraction and epithelialization -Results in more intense inflammatory response -Larger quantity of granulation tissue with pronounced contraction of wounds. -You keep the wound open (for example 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.	-"Delayed primary closure" -Desired for contaminated wounds -Phagocytosis of contaminated tissues well underway by 4th day Foreign materials walled off by macrophages. -When the wound is dirty > you clean it, wait for about 3 days and then re-approximate it by suturing (like primary healing).

Epithelial repair :

- Epithelial continuity is re-established across a wound :
  1- Mobilization
  2- Migration (Stimulus is loss of **contact** inhibition -if the cells reach the edges together they stop migration and mitosis starts-)
  3- Mitosis
  4- Cellular differentiation
  Summary: During wound healing, multiple events happen, so for example you have two edges; the cells mobilize (migrates to the middle) and gets in proximity then duplicate by mitosis and differentiate to different cells

Extra picture that shows the order of the events



Factors affecting wound healing :

General	Local
Nutrition (example: patients with pressure sore are usually malnourished so the wound healing would be delayed in such patients) Drugs/Toxins Age DM Smoking (because it reduces oxygen delivery) Vascular diseases Obesity Systemic diseases Idiopathic Inherited diseases	Reduction of delivery of Oxygen Infection Acidity Radiation Loss of growth factors Denervation Iatrogenic Edema Cancer patients who receive chemo and radiotherapy (because they lead to fibrosis of the skin as well as stenosis of the arteries) Foreign body

Phases of wound healing : **You need to know the phases and cellular components of each phase.**

1- Hemostasis:

- Usually considered part of the inflammatory phase in some resources
- Takes 5 - 10 minutes
- Initial response to injury - constriction
- Platelet plug forms 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 :

1. Platelet derived growth factor (PDGF) : Made by macrophages, endothelial cells, fibroblasts (Chemotaxis and Fibroblast stimulation)
2. Transferring growth factor beta (TGF B) : Made by macrophages, platelets, fibroblasts
    a. Fibrinogenesis, angiogenesis, chemotaxis, immune suppression
3. Fibroblast growth factor : Made by macrophages and endothelial cells (Angiogenesis and chemotaxis)

2- Inflammatory (lag) phase: (also called migratory phase)

- Typically starts immediately after hemostasis and takes **1 - 4 days, main cells are macrophages.**
- **Aim**: to stop bleeding.
- Initial intense local vasoconstriction of arterioles and capillaries followed by vasodilation and vascular permeability
- Classically represented by
    - Rubor (Redness)
        a. Caused by vasodilation
        b. Primarily result of prostacyclin (PGL) and histamine, also caused by prostaglandin A, D and E (PGA, PDD, PGE)

    - Tumour (Swelling)
        a. Caused by leakage of plasma proteins through gaps in vascular endothelium
        b. Edema **potentiated by PGE2, Prostaglandin F2 alpha, (PGF2 alpha)**

    - Calour (Heat)
        a. Increased local temperature secondary to both increased blood flow and elevated metabolic rates

    - Dolour (Pain)

3- Proliferative (incremental) phase

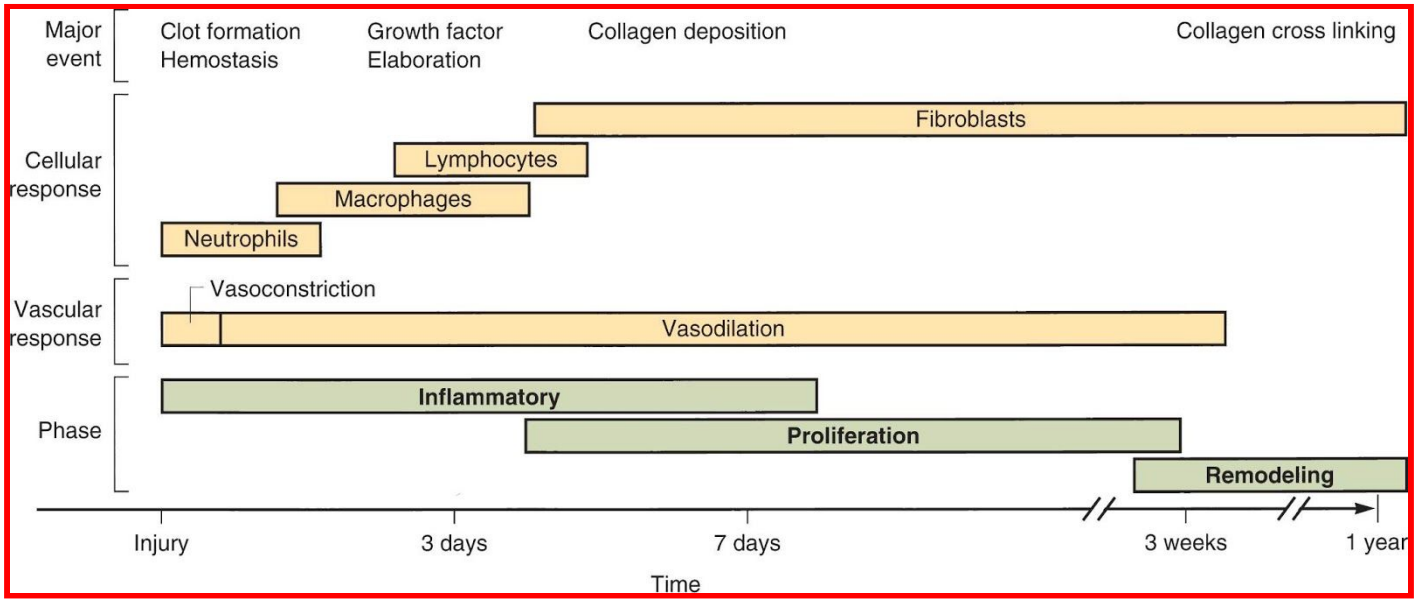
- **Aim:** to start healing.
- Regenerative or Reparative
- **begins 2 - 3 days after wounding and last for 3 weeks** (The Proliferative phase depends on Fibroblasts).
- Signalled by arrival of **fibroblasts (main cells)**
    - Driven by macrophage-derived bFGF, TGF beta, PDGF to proliferate and synthesize glycosaminoglycans (GAGs) and proteoglycans (building blocks of new extracellular matrix of granulation tissue and collagen).
    - Also produce bFGF, TGF beta, PDGF, Keratinocyte growth factor, insulin-like growth factors-1.
    - Dominant cell type peaking at 7-14 days.
- Collagen synthesis (Net production for next 3-6 weeks).
- Elevated (Keratinocyte mitosis, number of endothelial cells, **Angiogenesis** “From vessels at wound

margins").

- Lasts 2-4 weeks depending on site and size of wound with slowing of fibroblast migration and proliferation.
- Starting a chemotherapy after surgical resection of a tumor and wound healing needs to be minimum after 14 days
- Different cells differentiate into different types and new blood vessels are formed (angiogenesis)

4- Maturational - Remodeling (plateau):

- Aim: to increase strength.
- **Type I** replaces **Type III** Collagen.
- start at **3 weeks** after the injury **until 1 year.**
- Collagen synthesis and degradation are accelerated but in equilibrium with collagen breakdown (no net increase in collagen content).
- Collagen deposition peaks by 3rd week.
- Large capillaries growing into wound regress/disappear.
- Indurated, raised, pruritic scar becomes organized.
- Re-established normal 4:1 ratio (I:III) : Duration depends on age, genetics, type of wound, location (1-2 years). Type three collagen **is the main collagen present in the wound healing process.** for example 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.
- Tensile strength increases to 80% of pre injured skin. Depends on the site of injury, for example for tendons it reaches up to 60-80% maximum, but for normal wounds it usually reaches 100%.



- **Collagen:**
- Left handed helix involving 3 polypeptides
- Most abundant family of proteins in the human body (30%)
    - **Types of collagen:** there are nearly 23 types of collagen but you only need to know the first 5 types:
      (the most common collagen type in normal woundless skin is type 1 followed by type 2)
      The most common type in wounded (scarred) skin is type 3 specially in proliferative phase

    a. Type I (80% skin): Most Common: Major structural component in skin, bones and tendons.

Primary type in wound remodeling “latest phase”.

b. Type II: Found predominantly in cartilage.
c. Type III (20 % skin): found in association with type 1 in varying ratios depending on the type and maturity of tissue (predominant type in granulation tissue) Increased ratio in healing wound, also blood vessels and skin.
d. **Type IV:** Basement Membrane.
e. **Type V:** Widespread, particularly in the cornea.

- Lysine and proline hydroxylation are required for cross-linkage. Biochemistry: collagen synthesis events: procollagen gets converted to collagen by proline and lysine hydroxylation (essential component to have complete collagen synthesis).
- Differs in relative composition of hydroxylysine and hydroxyproline and cross-binding.
  - Type 1 90% of collagen in body
  - Normal skin ratio – Type I/Type III – 4:1
  - hypertrophic / immature scar 2:1 ratio

- Formation inhibited by: they activate collagenase which degrades collagen synthesis and inhibits cross linkage hydroxylation of lysine and proline.

1. Colchicine
2. Penicillamide
3. Steroids
4. Vitamin C deficiency as it has a role in the hydroxylation process
5. Fe deficiency

Types of surgical scars: Why is it important to know these things? to know if you need to cover the patient with antibiotics or not

Clean	Clean-contaminated	Contaminated	Infected
-Non traumatic, non infected wounds & no breach of respiratory, GI, or GU tract. -No spillage of the content of the tract itself (**example**: thyroid and breast surgery).	-Small branch in protocol; respiratory, GI, or GU tract are entered with minimal contamination -Very minor spillage of the content (**example**: uncomplicated appendicitis, intestinal resection ONLY if there was no spillage)	-Fresh traumatic wounds; major break in sterile technique, nonpurulent inflammation in or near contaminated skin -Major spillage (**example**: resection of the intestine with spillege)	-Purulent infection -Traumatic wounds, for example: open fracture (usually covered with triple dose of antibiotics) Not closing the wound or not well - drained wounds creates a suitable environment for infections .

Wound management:

- Cleansing
- Exploration of diagnosis
- Debridement
- Tetanus immunization status
- Replacement of lost tissue where indicated
- Skin cover if required
- Dressing
- Skin closure without tension ..

Extra image recommended by the doctor:

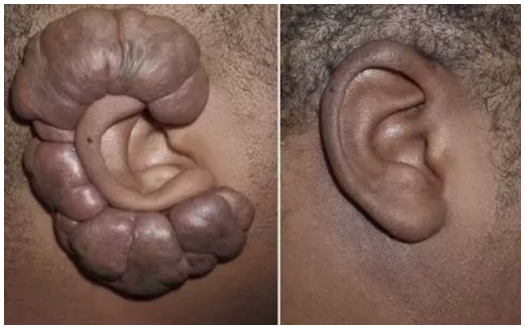
Table 2. Wound management, tetanus prevention and passive immunity administration

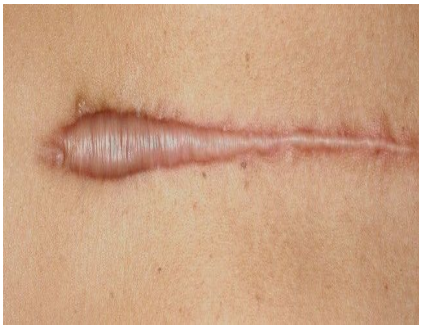
Vaccination status	Clean, minor wounds including infection-related ones	All other wounds including infection-related ones
Unknown or <3 doses of TT-containing vaccine	TT and recommend catch-up vaccination	TT and recommend catch-up vaccination: TIG <5 years 75 IU, 5 - 10 years 125 IU, >10 years 250 IU
≥3 doses of TT-containing vaccine and <5 years since last dose	No indication	No indication
≥3 doses of TT-containing vaccine and >5 years since last dose	TT recommended	TT recommended

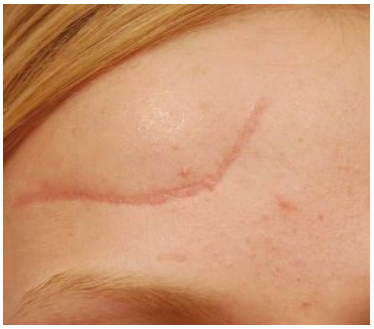
TT = tetanus toxoid; TIG = tetanus immunoglobulin.

you first clean the wound then check for tetanus then take history then check the diagnosis then depritment if needed ..etc (in case of a neck wound, take the pt to the operating room immediately. Fear of vessels injuries)

Keloid Vs Hypertrophic scars Vs Wide scars:







Wide scar: usually happens if you leave the wound open and just apply dressing

Ideal scar:

- Flat, narrow
- Good color & contour match to surrounding skin
- Parallel to or within resting skin tension lines (RSTL)
- Pliable
- Doesn't restrict function or distort normal anatomy, (for example in the axilla or in the joints) because this is how it heals better.
- Matures within 6-18 months
- Asymptomatic (not painful)

Comparison of keloid and hypertrophic scars: VERY IMPORTANT

Table 3.4 Comparison of hypertrophic and keloid scars

Features	*Hypertrophic scar*	*Keloid scar*
Genetic	Not familial	May be familial
Race	Not race related	Black > white
Sex	Female = male	Female > male
Age	Children	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

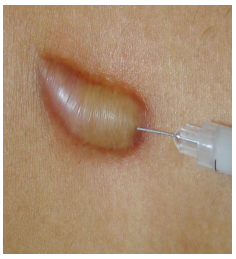
Keloids: familial, females, black people, beyond the boundaries of the scar itself, and there are specific sites for the development of keloids, related to tensions but far less than hypertrophic scars

What do we mean by tension? when you put a lot of tension when you close the wound and you strangulate the skin.

Treatment of hypertrophic scars: Best thing is prevention of HTS (1st), by minimal undermining, closing layer, no strangulation (put spaces between each suture)

- Pressure (by compression garments).
- Silicone
- Prevention
- 5-FU (Fluorouracil) (chemotherapy)
- Intra-lesion Steroids (first line in treatment).
- Radiation
- Laser
- Surgery

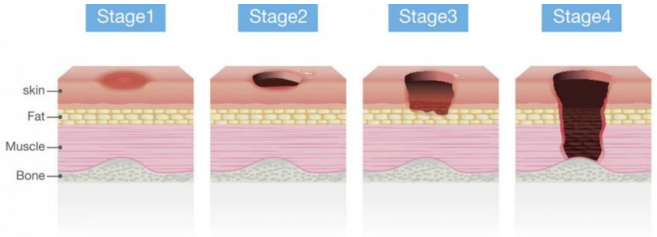
Intra-lesion steroid - silicone - pressure garments



- **You may ask how does radiation and chemotherapy heal HTS while it leads to reduced wound healing?** the unit of given radiation therapy is the guide, so patients that receive radiation therapy for cancer receive high doses compared to HTS which receive minimal doses, the second thing is that HTS has abnormal collagen synthesis (high turnover of collagen) and radiation stops this by leading to fibrosis and less formation of collagen.

Pressure sores AKA Ulcers, bed sores:

Skin layers (check it before proceeding for better understanding)



PRESSURE SORES

Stage 1: developed if the person doesn't move while sleeping for 2 hours (bed sore): mainly involves the epidermis and produces erythema , avoided by changing the position every 30 min.
Stage 2: involves epidermis and upper dermis
Stage 3: reaches up to the fascia, does not go beyond that
Stage 4: beyond the fascia and can reach to the muscle, tendons and bones, and they usually develop osteomyelitis

Vancouver Scar scale: not important.

	Feature	Score
Vascularity	Normal	0
	Pink	1
	Red	2
	Purple	3
Pigmentation	Normal	0
	Hypo-pigmentation	1
	Mixed-pigmentation	2
	Hyper-pigmentation	3
Pliability (Elasticity)	Normal	0
	Supple (flexible with minimal resistance)	1
	Yielding (giving way to pressure)	2
	Firm (inflexible, not easily moved, resistant to manual pressure)	3
	Banding (rope-like tissue that blanches with extension of the scar)	4
	Contracture (permanent shortening of scar, producing deformity or distortion)	5
Height	Flat	0
	< 2 mm	1
	2-5 mm	2
	> 5 mm	3
Pain	None	0
	Occasional	1
	Requires medication	2
Itchiness	None	0
	Occasional	1
	Requires medication	2

Recall:

Primary wound closure:
Suture wound closed immediately (a.k.a. “first intention”).
Secondary wound closure:
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 larger scar).
Delayed primary closure(DPC):
Suture wound closed 3 to 5 days AFTER incision (classically 5 days).
How long a sutured wound epithelialized?
24–48 hours.
What inhibits wound healing?
Infection, ischemia, diabetes mellitus, malnutrition, anemia, steroids, cancer, radiation, smoking.
What reverses the deleterious effects of steroids on wound healing?
Vitamin A.
Diseases Associated With Abnormal Wound Healing:

- Osteogenesis Imperfecta: Type I Collagen defect.
- Ehler-Danlos syndrome: Collagen disorder, 10 types. Group of disorders that affect the connective tissues that support the skin, bones, blood vessels, and many other organs and tissue.
- Marfan Syndrome: fibrillin defect (collagen).
- Epidermolysis Bullosa: Excess fibroblasts Tx: phenytoin. Epidermolysis bullosa (EB) is a group of inherited bullous disorders characterized by blister formation in response to mechanical trauma.
- Scurvy: Vit C is required for proline hydroxylation (involved in collagen synthesis).

Burns and burn management Davisons p303.

Burns: are thermal, chemical or electrical injury sufficient enough to cause tissue disruption, denaturation, or even death.

- Carry the risk of permanent: Disfunction or impairment of function

Epidemiology:

Burns: are thermal, chemical or electrical injury sufficient enough to cause tissue disruption, denaturation, or even death.

- Males > females
- 2 peaks at:
  - 0-5 years and 25-35 years
  - 80% of burns are less than 20% TBSA
- Pediatrics:
  - Scald burns >80%
  - Account for 45% of hospital admission
  - 33% due to **child abuse**
- Elderly:
  - Impaired mobility, poor coordination, decrease awareness to pain
  - Abuse/neglect
- Factors that increase mortality (Each factor increases mortality for about 30%, so three factors would lead to 90% chance of mortality):
  1. Age greater than 60
  2. Greater than 40% TBSA (total body surface area)
  3. Inhalation injury
  4. Alcohol is a common contributing factor

Why is this important to know? because we need to discuss with the patient and his family about the outcomes and prognosis of burns before going into surgery.

Calculation of Total Body Surface Area: IMPORTANT!

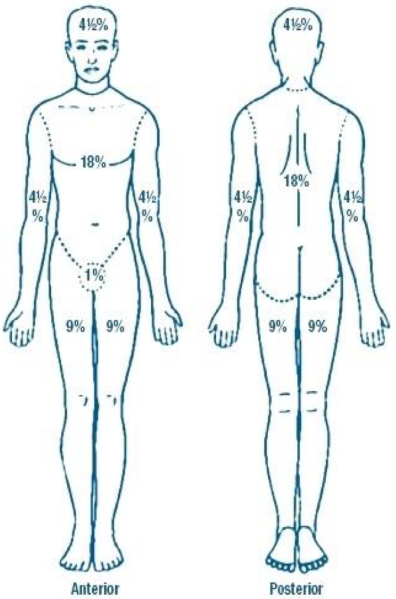
a. Wallace rule of nines.
   (Anterior and posterior separately)
   Ex. The whole right arm = 9% (4.5 anteriorly and 4.5 posteriorly)

b. The rule of palm: used for fast evaluation (emergency) otherwise we use the role of 9

c. Patients palm (each palm) is equal to 1 % of their TBSA: Good for scattered burns

d. Lund browder chart (for paediatrics)

Wallace rule of nines



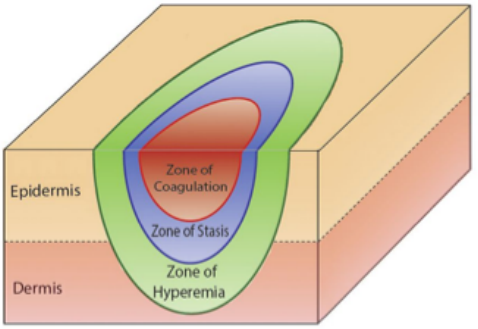
Types of burns:

A. Scalds (hot liquids) most common type in children
B. Flame (thermal) most common in adults
C. Electrical
D. Chemical "mainly causes hypocalcemia"
E. Frictional
F. Flash
G. Contact

Pathophysiology:

- Increased fluid loss from the surface.
    a. Normally 15 ml/m^2 increase to 200 ml/m^2.
- In deep burns, the dermis is converted to coagulum called ESCHAR.
- Inflammatory response ranges from.
    a. Capillary dilation in sunburns.
    b. Damaged capillaries and increase permeability and interstitial edema.
- 2 cm increase in leg diameter represent 2L of increase in fluid.
- Destruction of epidermis removes the protective bacterial barrier > Contamination > Sepsis > Death.

- Has 3 zones.
    a. Zone of coagulation
    b. Zone of stasis
    c. Zone of hyperemia



In burns, there are 3 zones which are called **jacksonian** zones: (like a process of injury to the skin)
-Coagulation: loss of oxygen and proteins
-Stasis: most important zone: why is this important? because it is the key for fluid resuscitation, in which we give fluids to turn into the normal hyperemia zone.
-Hyperaemia: normal skin
So we need to transfer the injury from coagulation to hyperaemia

General effect of burns:

➔ The effect of the burn depends on the size.
➔ Majority of mortality rates: early: poor resuscitation, late: wound sepsis

- The bigger the burn the more the physiological changes:
    - Water loss.
    - Salt loss.
    - Protein loss.
    - Increased catabolism.
    - Edema due to fall in circulating plasma.
    - Damage to RBC's : Immediate or delayed.
    - Increased Hematocrit.

Just go through it

Classification of burn severity

Minor burn
≤15% TBSA in adults ≤10% TBSA in children and the elderly ≤2% TBSA full-thickness burn in children or adults without cosmetic or functional risk to eyes, ear, face, hands, feet, or perineum
Moderate burn
15–25% TBSA in adults with <10% full-thickness burn 10–20% TBSA partial-thickness burn in children under 10 and adults over 40 years of age, with <10% full-thickness burn ≤10% TBSA full-thickness burn in children or adults without cosmetic or functional risk to eyes, ears, face, hands, feet, or perineum
Major burn
≥25% TBSA ≥20% TBSA in children under 10 and adults over 40 years of age ≥10% TBSA full-thickness burn All burns involving eyes, ears, face, hands, feet, or perineum that are likely to result in cosmetic or functional impairment All high-voltage electrical burns All burn injuries complicated by major trauma or inhalation injury All poor-risk patients with burn injury

TBSA, total body surface area.

- Hypovolemic shock.
- Increased metabolic rate: 7000 Kcal expended daily.
- Daily weight loss of 0.5 kg.

Mortality:

- Result mainly from poor resuscitation
- In the early post burn period:
  a. Inadequate hydration.
  b. Respiratory issue (inhalation or infection).

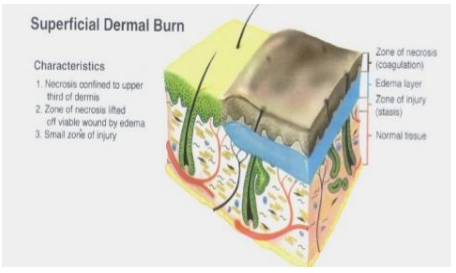
- In the late post burn period:
  a. Wound sepsis.
  b. Delayed healing.
  c. Increased energy expenditure.

Burn classification: A) By degree / Thickness B) By type

A- By Degree / Thickness:

Old	1st degree burn 2nd degree burn 3rd degree burn 4th degree burn
New (better classification)	Superficial burn (1st degree) Superficial partial thickness (2nd degree) Deep partial thickness (2nd degree) Full thickness (3rd & 4th degrees) YOU NEED TO KNOW THE DIFFERENCE BETWEEN THE THREE

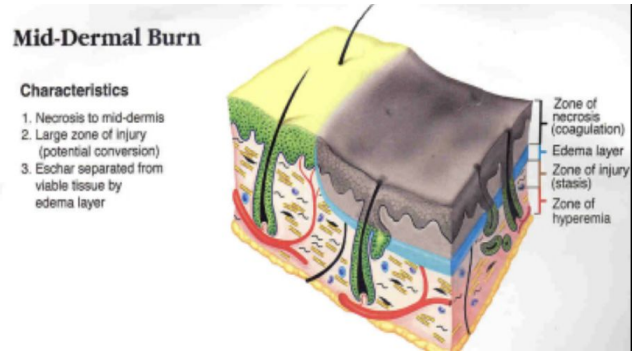
- **Superficial (1st degree):**

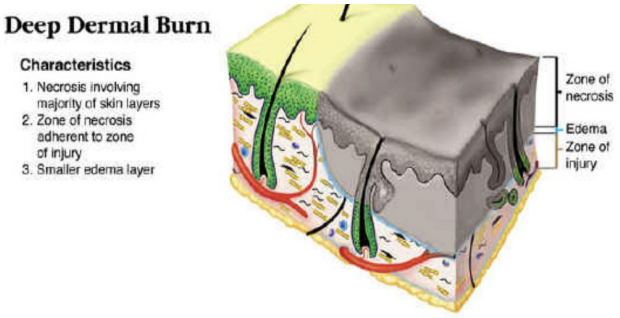


- Damage to the **epidermis** only, you don't need to count TBSA for it because it's a very small injury. (Similar to tanning or sun bathing)
- No need for admission
- Heal within 5 – 7 days by itself.
- No scarring
- Needs only analgesics and hydration.

- **Partial thickness (2nd degree):**

Superficial partial thickness	Deep partial thickness
Epidermis and upper dermis Pink, painful and swollen Fluid loss Blisters Heal within 2 weeks From epidermal appendages Requires daily dressing Minimal scarring You don't need to treat it by debridement	**Epidermis and most of the dermis** Treat like 3rd degree burn Less or not painful* Rarely blister Prolonged inflammatory phase cause scarring Leaves an ugly hypertrophic scars NEEDS DEBRIDEMENT and skin grafting





- **Full thickness (3rd degree):** No remaining visible dermis.. Up to muscle.
  - Epidermis and total dermis including the epidermal appendages
  - Destroyed tissue undergoes coagulative necrosis
  - Not painful
  - No blisters
  - Marble or leathery like appearance
  - THROMBOSED VEINS
  - Cause significant scarring due to inward growing and movement of the cells
  - Needs debridement and skin grafting

- **Full thickness (4th degree):**
  - Injury extends to the underlying structures.
    - Muscle, fascia, bone.
    - Charring of the tissue.
    - This type definitely needs debridement, if you don't treat it > HTS and contractions

Determination of burn depth:

- Mechanism
  - Scald: short vs long
  - Flame, chemical, electrical
- Appearance
  - ➔ Erythema, blister, eschar, thrombosed veins
- Sensations
- Time to healing: It's important to wait for 5 days (3 to 7 days) before determining the extent of the thickness, because as we mentioned in the stasis zone, the thickness is determined after fluid resuscitation and if the patient goes to coagulation zone, he will need debridement, but if it goes to hyperaemia then it will heal normally.

Prognosis: Depends on :

A- Age and general condition

B- Extent of the burn: increases mortality rate by 30%
The smaller the TBSA the better the burn prognosis and vice versa

C- Depth of the burn: increases mortality rate by 30%
Superficial burns heal within 2-3 weeks
The deeper the burn the higher the risk of infection

D- Site of the burn: increases mortality rate by 30%
Due to appearance and functional impairment

E- Inhalation injury

Burn management :

In the ER you start with checking (ABC: Airway, breathing, circulation) then:

First Aid			
Stop the burning process	**Chemical burns**	**Electrical burns**	**Avoid wound contamination**
Victim stay flat to avoid inhalation of smoke and fumes. Remove all burned clothes. Apply cold water vs hypothermia.	Copious irrigation (adding tons of water) DON'T ADD ACID IF THE PATIENT IS BURNED WITH ALKALINE OR VICE VERSA, because both of these agents increase the burn.	Switch of the current or by pushing the victim away from the current source with a non-conducting object. Put the patient on a cardiac monitor.	Burn must be covered with clean sheet Avoid using household items as It might convert partial injury to a full thickness one

- **ABC's (airway, breathing, circulation) - Life preservation**

- History:
  - Causative agent of injury
  - Medical co-morbidities

- Physical exam:
  - Inhalational component?
  - Is it still active? You need to stop it! And if the patient is wearing clothes on the burned area take them off.
  - Estimation of depth
  - Estimation of TBSA of burn

- Determination: severity of injury and triage/transfer.

- Irrigation and debridement of wounds.

Transfer criteria : Guidelines for transfer of patients to burn centers

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

There is a specific criteria for transferring and taking good care of this patient (usually more than 20% of partial thickness and more than 5% of full thickness and if it's in a sensitive area such as face, hand, foot and genital areas) electrical and chemical burns (patients who need social support) and patients with co-morbidities, traumatic patients in which the burn is the major problem.

Inhalation injuries : The mechanism of inhalation injury can be divided into three broad areas:

when you're in a **closed area** and there's fire, there will be a lot of gases for example carbon monoxide and cyanide which lead to inhalation injury because the gas goes inside and leads to burn and sometimes desloughing of the mucosa.

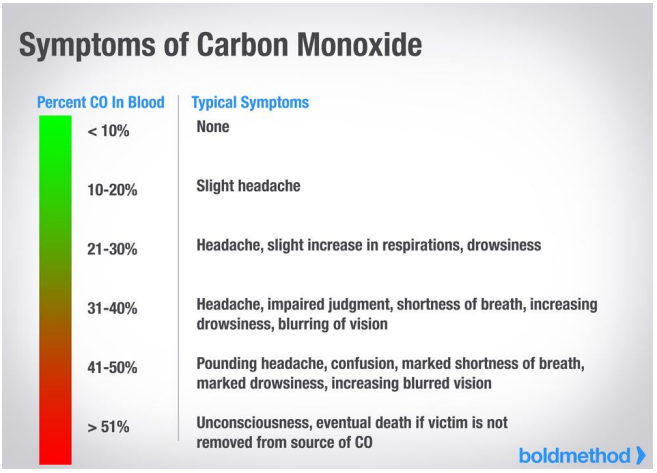
1) Inhalation of products of combustion
2) Carbon monoxide inhalation
3) Direct thermal injury to the upper aero-digestive tract

➔ Suspect inhalation injury if: IMPORTANT: do a bronchoscopy to determine the extent of burn.

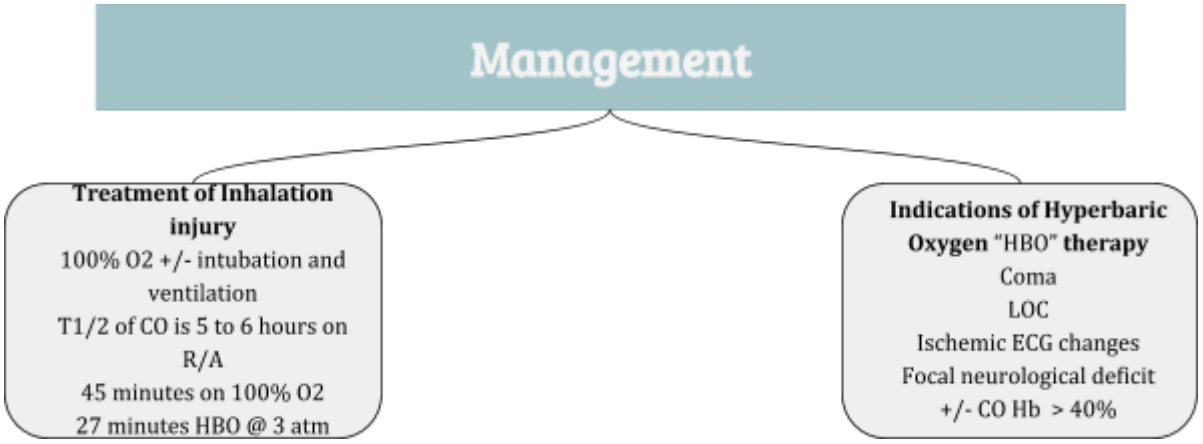
- Flame burn in a closed space
- Singed (burned) nasal hairs
- Facial or oropharyngeal burns
- Expectoration of carbonaceous (blackish) sputum
- Signs of upper respiratory obstruction – such as crowing, dyspnea, cough, stridor, or air hunger
- Symptoms: distress, hoarseness of voice, sputum production.

Carbon monoxide :

- Odorless, tasteless gas
- Impairs tissue oxygenation by preferentially binding to Hgb
- Affinity 240 times that of oxygen
- **Shifts the Hgb dissociation curve to the left decreasing O2 delivery**



- Signs include :
  - Headache
  - Cherry red lips
  - Arrhythmias
  - Acidosis
  - Seizures
  - LOC "Lower level of Consciousness"



***HBO: hyperbaric oxygen (for pregnant patients and patients who got into a coma, because the need to get normalized as fast as possible).**

Burn management cont:

1- Non surgical:

- Tetanus vaccine
- Fluid
- Nutrition

- Physiotherapy
- Dressing

2- Surgical: (for deep and full-thickness burns)

- Escharotomy (is a surgical procedure used to treat full-thickness (third-degree) circumferential burns. In full-thickness burns, both the epidermis and the dermis are destroyed along with sensory nerves in the dermis. The tough leathery tissue remaining after a full-thickness burn has been termed eschar.)
- Debridement
- Grafting
- Fasciotomy: for compartment syndrome (it is a surgical procedure where the fascia is cut to relieve tension or pressure commonly to treat the resulting loss of circulation to an area of tissue or muscle)

Non surgical burn management:

A- Fluid resuscitation: very important to know the types of fluids and IVs.

- Burns over 15% TBSA in adults or 10% in children
- Require IVF administration through a peripheral vein or internal jugular or subclavian vein line if peripheral line is not possible
- Withdraw blood for CBC, electrolytes, CO level
- Foley catheter

- **Parkland formula: (crystalloid) →** (most common) **(for the first 24 hours after injury):**

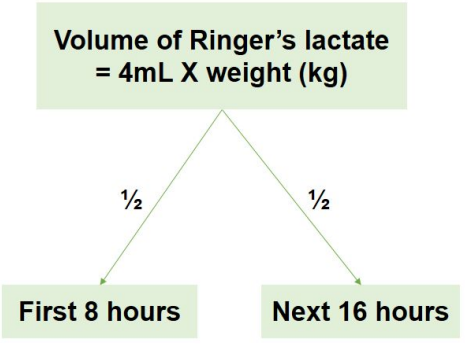
The Parkland formula is a burn formula used to estimate the amount of replacement fluid required for the **first 24 hours** in a burn patient so as to ensure they remain hemodynamically stable.

Lactated **Ringer** solution = 2-4ml x TBSA x body weight (kg)

*(4cc **x** (%of burn) **x** weight of patient = total amount of fluid needed in 24 hrs)*

Half of the calculated volume is given in the first 8 hours after injury and remaining volume is infused over the next 16 hours.

Start counting from time of burn NOT when you see the patient in the ER.



Ringer-lactate, is a solution that is isotonic with blood.

The main goal is to reach 1cc/kg/hour of urine output for adults, for children we give more fluids and so the main goal is to reach 2cc/kg/hour of urine output

More explanation :

مثال: مريض يحتاج ٨ لتر تعطيه نصف الكمية اللي هي ٤ بأول ٨ ساعات وتكمل الباقي اللي هي ٤ لتر في ١٦ ساعة الجاية >> هذا في حالة ان المريض جاك بعد الحرق مباشره ما تأخر .

لكن لو افترضنا ان المريض تأخر ساعتين تقوم تطرح ال٢ ساعتين من ٨ ساعات يعطيك ٦ ساعات, الحين تعطيه نصف الكمية بأول ٦ ساعات و الباقي تعطيه ب ١٦ ساعه الجاية

مثال ثاني لو المريض جاء بعد ٨ ساعات من الحرق، هنا تعطيه نصف الكمية اللي يحتاجها دفعة وحدة و الباقي تعطيه في ١٦ ساعة الجاية .

after 24 hrs you maintain the same IV fluid whether you decrease or increase the amount according to the Urine output

Always ask the patient when did the burn happen.

- **Other formulas and requirements:**
  - **Modified brooke's :** Weight in Kg x TBSA x 2cc
  - **Using hypertonic saline :** 250-300 mEq, Decrease the fluid requirement, Require regular Na monitoring
  - **Colloid replacement formula :** 0.5 ml/kg/ % of burn
  - **Blood transfusions :** After 24 hours, Mainly needed in patients with full thickness burns. Regular hemoglobin, Hematocrit monitoring is mandatory

Children

will need the maintenance IVF add to their fluid resuscitation
100 ml / kg / 24 hours 1st 10 Kg
50 ml / kg / 24 hours 2nd 10 kg
20 ml / kg / 24 hours 3rd on kg
To avoid hypoglycemia
Use D5 ½ NS fluid

- **Adequate fluid resuscitation is monitored by: Urine output**

★ 0.5 – 1 ml/kg/hour for adult
★ 2 ml/kg/hour for children
★ In cases of electrical burns more urine is needed to protect the kidney 2-3 ml/kg/hour
★ Inhalation injury patients also need extra fluids

B- Nutrition: Due to increased energy expenditure following severe burns .

A. Requires high caloric protein diet once the patient can take orally or via NGT
B. Daily requirement is 20 Kcal + 70 Kcal/kg/ % of burn (1g/kg + 3g/kg/ % of burn of protein)
C. Vitamins supplement : Vitamin C, Zinc & general multi-vitamins.

C- Physiotherapy: Splints to prevent contractures, Range of motion exercise to prevent stiffness.

D- Dressing:

Local wound management	Cover burn regularly to avoid infection which is the main threat to life once the 1st 24 hours have passed
Initial cleansing and debridement	Cleanse wound with antiseptic and saline Blister are punctured
No role for IV or oral Antibiotics	
Do not forget tetanus	

- ➔ Destruction of the epidermis removes the normal barrier to infection (Full thickness burns impair the normal response to infection.
- ➔ The organism colonize the burn wound (staphylococci is the most common, pseudomonas remains troublesome in most burn units)
- ➔ Flamazine (silver sulfadiazine) the most common burn dressing used
  - ◆ Must be applied daily
  - ◆ S/E neutropenia
  - ◆ Can not be used in infants under 6/12 of age
- ➔ **Open vs closed dressing**
  - ◆ Fucidine or bacitracin ointment
  - ◆ To prevent superinfection of the burn wound from the colonized organisms

Surgical burn management:

- **Escharotomy :**
- Mid – Axial incision in the eschar only of full thickness burn
- To relive the tourniquet effect of the circumferential full thickness burns on extremity,
- trunk if breathing was an issue. If increase in intra-abdominal pressure
- Due to interstitial fluid accumulation

Summary note: the summary does not contain all the info

Wound: is a disruption of normal anatomical structure, function and relations as a result of intentional or unintentional injuries and it is Classified as Acute vs. Chronic.

Primary wound healing	- Primary closure. - Within hours of repairing full thickness surgical incision. - Result in mortality of minimal number of cellular constituents.
Secondary healing	- Wound left open to heal by processes of granulation contraction and epithelialization - Results in more intense inflammatory response - Larger quantity of granulation tissue with pronounced contraction of wounds
Tertiary healing	-"Delayed primary closure" - Desired for contaminated wounds - Phagocytosis of contaminated tissues well underway by 4th day ,Foreign materials walled off by macrophages

Phases of wound healing

Hemostasis	-Takes 5 - 10 minutes -Initial response to injury there will be constriction -Platelet plug forms after adherence to exposed subendothelial collagen via vWF -Platelets degranulate releasing: ADP, Thromboxane A2, Bradykinin, and 5-HT > -Further vasoconstriction and platelet aggregation. Platelets release:**PDGF**, **TGF B** and **fibroblast growth factor**
Inflammatory phase	Typically takes **1 - 4 days,** **main cells are macrophages.** Classically represented by : 1-Rubor (Redness): vasodilation, and release of prostacyclin , histamine and prostaglandins 2-Tumour (Swelling) : leakage of plasma proteins and Edema **potentiated by PGE2, Prostaglandin F2 alpha** 3-Calour (Heat); duo to high blood flow and metabolic rate 4-Dolour (Pain)
Proliferative phase	**- begins 2 - 3 days after wounding and last for 3 weeks** - Signalled by arrival of **fibroblasts** **(main cells)** - Collagen synthesis (Net production for next 3-6 weeks). - Elevated (Keratinocyte mitosis, number of endothelial cells, **Angiogenesis** "From vessels at wound margins"). - Lasts 2-4 weeks depending on site and size of wound with slowing of fibroblast migration and proliferation.
Remodeling	**- Type I** replaces **Type III** Collagen. - start at 3 weeks after the injury until 1 year. - Collagen synthesis and degradation are accelerated but in equilibrium with collagen breakdown (no net increase in collagen content). - Collagen deposition peaks by 3rd week. - Large capillaries growing into wound regress/disappear. - Indurated, raised, pruritic scar becomes organized.

Burns

1st degree	2nd degree		3rd degree	4th degree
-Damage to the **epidermis** only, -No need for admission -Heal within 5 – 7 days -No scarring -Needs only analgesics	**1-Epidermis and upper dermis** -Pink, painful and swollen Fluid loss Blisters -Heal within 2 weeks -From epidermal appendages -Requires daily dressing Minimal scarring	**2-Epidermis and most of the dermis** -Treat like 3rd degree burn -Less or not painful* Rarely blister -Prolonged inflammatory phase -cause scarring - Leaves an ugly hypertrophic scars NEEDS DEBRIDEMENT and skin grafting	- Epidermis and total dermis including the epidermal appendages. - Destroyed tissue undergoes coagulative necrosis -THROMBOSED VEINS - Cause significant scarring - Needs debridement and skin grafting	Injury extend into underlying structures: Muscle fascia bone and there is charring of tissue

Questions

1. The following are the sequence of events and phases taking place in wound healing :
A. Remodeling, epithelialization & contracture .
B. Inflammatory, proliferative & remodeling .
C. Vasoconstriction, epithelialization & contracture .
D. Proliferative, remodeling & wound synthesis .

2. The major cell/s involved in the proliferative phase is/are :
A. PMN .
B. Fibronectin .
C. Fibroblast .
D. Collagen

3. The difference between secondary & partial thickness healing is :
A. Contracture only in secondary healing .
B. Contracture only in partial thickness healing .
C. Epithelialization only in secondary healing .
D. Epithelialization only in partial thickness healing .

4.Perforated gastric ulcer or perforated appendicitis is/are :
A. Clean.
B. Clean-contaminated.
C. Contaminated.
D. Infected.

5. All of the following is/are characteristic of ideal scar except :
A. Painful
B. Flat
C. No restriction of movement
D. Adequate color

6. All of the following is characteristic of keloid :
A. Crosses border
B. Genetic predisposition
C. Common in earlobes / chest in africans
D. All of the above

7. Preventive measures of hypertrophic scars is/are :
A. Adequate suture bite closure

B. Free tension closure
C. Non strangulated sutures
D. All of the above

Answers:
1: B 2: C 3: A 4: C 5:A 6:D 7:D