Burn injury and wound healing

Objectives:
Not given

Resources:
- Davidson's.
- Slides
- Surgical recall.
- Raslan's notes.

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Once you stop learning
you start dying.

Burn Injury

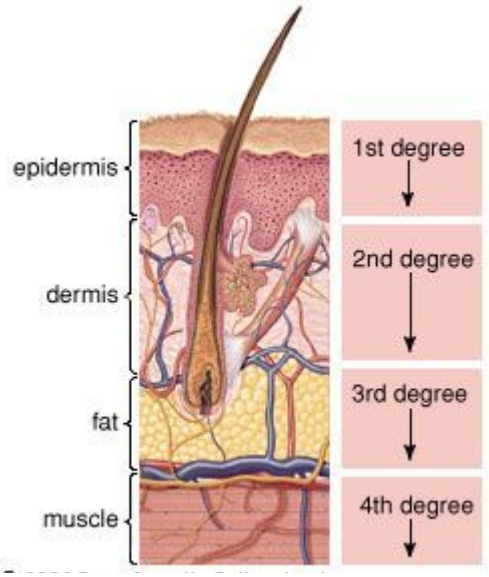
Skin function:

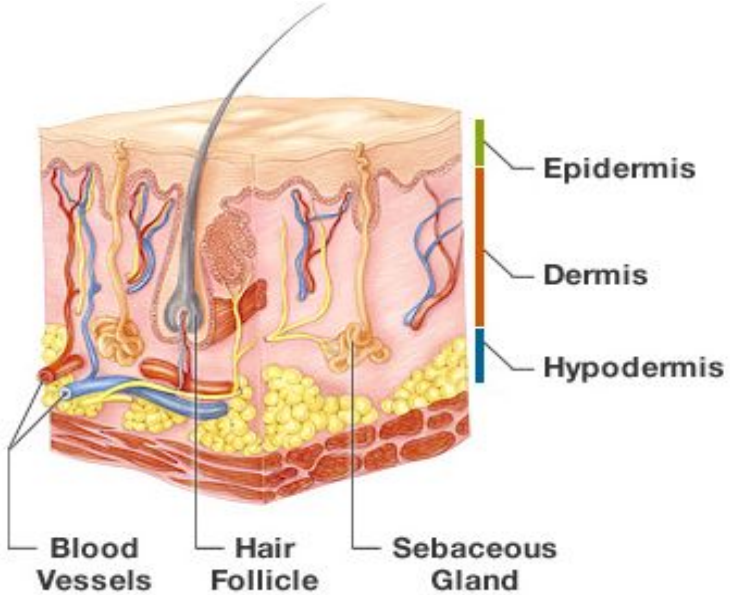
- Body Covering
- Permit movement of underlying muscles & joint
- Sensors for touch, pain, and temperature
- Vitamin D production
- Temperature regulation by sweating and blood flow.
- Sun protection
    - Detoxification/activation of drugs and chemicals
- Immuno-surveillance
    - Langerhans cells,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

Layers of the Skin :

Deep layers injured = less healing

Epidermis	• Thin compared to the dermis • Outer layer contains the stratum corneum[1] • The rate limiting step in dermal or percutaneous absorption is diffusion through the epidermis
Dermis has the skin appendage	• Much thicker than epidermis • True skin & is the main natural protection against trauma and responsible for skin regeneration • Contains skin appendages: ○ .Sweat glands ○ Sebaceous glands ○ Blood vessels ○ Hair ○ Nails
Subcutaneous Layer (fat)	• Contains the fatty tissues which cushion & insulate the skin





[1] outermost layer of the skin, consists of keratinized cells

Burns: Davisons p303.

It is important to know exact mechanism of burn specially in children to rule out child abuse, as well as duration, time of exposure and environment.
Most deaths occur at home are caused by burns.

Causes of death: Dehydration is the number one killer in patient with burn

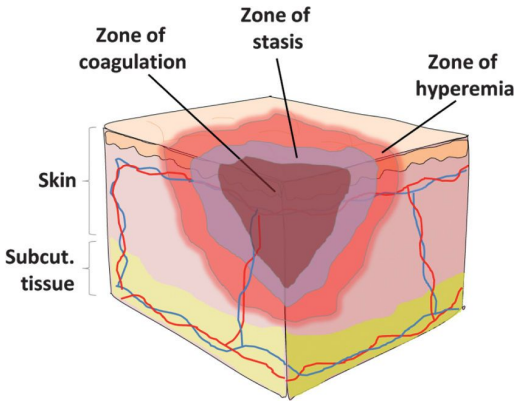
Smoke inhalation, sepsis (major cause), pneumonia (infections), shock (burns more than 20% causes systemic inflammatory response ⇒ 3rd spacing ⇒ hyponatremia ⇒ shock)

- More common in elderly
- (age + affected BSA[2] = %mortality)

most of those with affected BSA > 70% die (the more the burn the higher the mortality rate)

Risk factors for death

- > 40% affected BSA the larger the surface area the higher the mortality.
- > 60 years the older the patient is, the higher the mortality
- inhalation injury



Pathophysiology of Burns:

- **Dynamic injuries**
  - Dependent on temperature and duration
- Cellular damage starts
  - at >45° C (irreversible injury)
- **Three zones of injury:**

Central necrosis irreversible damage	it receives the maximal temperature of the burn which leads to necrosis
Zone of stasis (at risk of necrosis) the middle zone	One of the ways of management is to prevent this zone from converting to the central necrosis, we want it to change to the hyperemia (by good hydration)
Zone of hyperemia the outer zone	inflammatory response, vasodilatation high vascularity due to wound healing

- **Thermal injury** triggers intense inflammatory response SIRS[3]. SIRS causes catabolism (breaking down of all body protein) ⇒ the intestinal lining gets affected ⇒ bacteria in the guts will translocate to the blood vessels ⇒ sepsis ⇒ MOF
  - Initial release of histamine, bradykinin
  - Release of prostanoids, free radicals, proteases.
- **Leading to:**
  - Hypermetabolism.
  - Bacterial translocation.
  - MOF[4]

Once the burn injury is more than 20% of the body surface area the inflammatory response will be systemic
There will be systemic vasodilation
Fluid will shift from the intravascular space to the extravascular space which leads to hypo-perfusion to vital organs such as the kidneys, causing renal failure.
Hypo-perfusion to the intestines may cause intestinal ischemia
Bacteria will shift into the bloodstream (bacterial translocation) → leading to sepsis (may lead to death if left untreated)

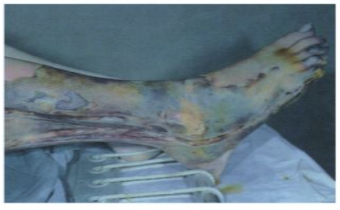
[2] Body Surface Area
[3] Systemic Inflammatory Response Syndrome, (inflammatory process is usually local in burns that are < 20%)
[4] Multiple Organ Failure

Burn patients are usually treated as trauma pts (ATLS protocol), important things to do:

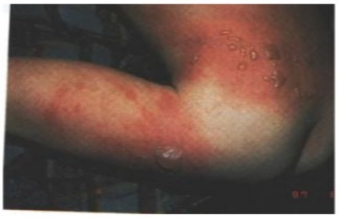
- Make sure he's stable
- Hx [type of burn, when and how it happened (time and exact mechanism of the burn)
- Examination
  - degree of burn
  - total BSA

Degree of burns:

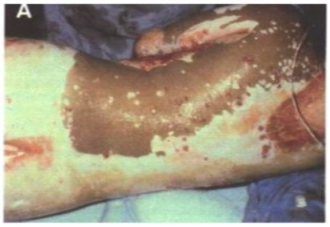
Depth		Histology	Appearance	Sensation	Healing	Example	Manage
1st		Epidermis only ,common in Caucasians who have less melanin Not calculated in TBSA	Erythema, blanches with pressure	Intact; mild to moderate pain	3-6 days No scarring	Sunburn & hot fluid	painkiller + fucidin cream (or fadiazine which is a sulfa broad spectrum antibiotic) + NO admission
2nd	Superficial	Epidermis and the superficial dermis ; skin appendage intact, Pinkish in color	Blisters, Erythema, moist, elastic; Blanches with pressure	intact leading to severe pain (worst pain)	1-2weeks Scarring is unusual	Hot water and soup	topical dressing + ointment NO Surgical intervention flamazine
	Deep	Epidermis and most of the dermis, most skin appendage destroyed.	Thick large blisters, white appearing with erythema, dry, waxy, less elastic, reduced blanch with pressure	nerve damage causing Less pain.	>3 weeks Significant scarring (due to destruction of appendage) and contracture so we do surgery		surgery debridement & skin grafting
3rd		All skin layers (epidermis and all dermis), destruction of all skin appendage, hair will fall out easily	Skin feels like leather due to loss of elasticity, dry Whitish or black in color Eschar formation Thrombosed vessels (No blood flow), does not blanch	Painless (anesthetic) although the surrounding areas of 2nd degree burns are painful.	Doesn't heal, takes very long time. Severe scarring and contracture	Flame burn	
4th		Reaches bones & muscles (you can see the underlying tissue)					



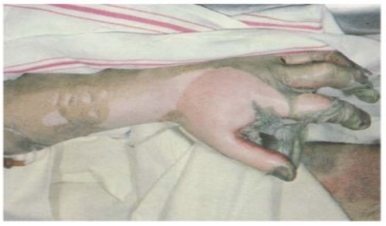
4th degree burn with 3rd degree burns



1st & 2nd degree (redness & blisters)



Third degree



2nd degree (scalded skin from blister) & 3rd degree (white area)

patient usually presents with burn of multiple degrees. (you don't get type 2 or type 3 alone, you get a combination of both) BUT should you consider the most prominent and most severe type.

to differentiate between type 2 and type 3 : type 2 blisters , type 3 : no blisters ⇒ if the patient JUST came with **burn** , but after 3 weeks for example there will be NO blisters, so you will see the **healing** area : if red type 2, if white type 3 .

in the first degree burn we do not calculate the percentage of the burn as it does not count as a real burn even if they told you we have a 90% first degree burn you don't calculate it.

- in MCQ: you don't have to differentiate between the second superficial burn and the second deep burn

Determining Extent of Injury:

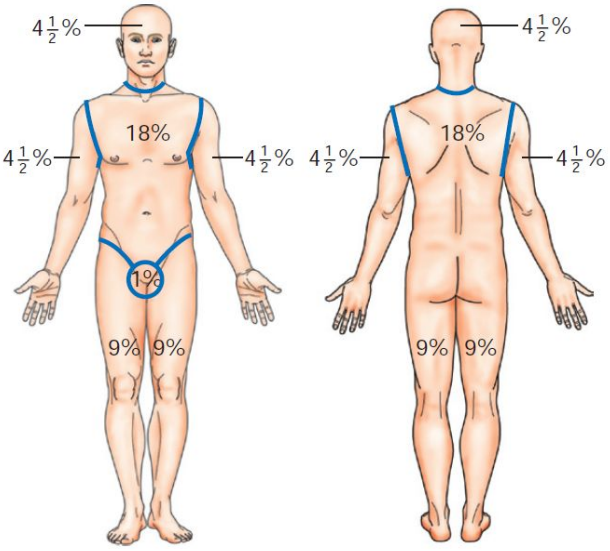
It's important to know the percentage of the surface area; it determines the mortality, SIRS.

- Burn extent determines therapy and prognosis (look in the history for cause & type of burn)
- Burn size estimation is often inaccurate
- Extent of injury described using percentage of total body surface area that is burned (TBSA)
- In adults with large area burns "rule of nines" or "wallace rule of nine" may be used (we divide the body into estimate areas of 9%)

In Small, multiple patchy burns , we use the patient's palm (شبر) as a 1% (0.8%to be exact).
Since children have larger head surface areas, the proportions are not the same, there is a chart found in the ER according to the age No need to memorize it
Memorize the **ADULT** percentages only.

Head and neck **9%** one upper limb **9%**
Anterior trunk **18%** one lower limb **18%**
Posterior trunk **18%** **genital**/ some say neck **1%**

In 1st degree burns TBSA is not calculated



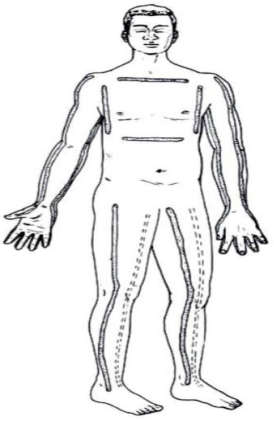
Anterior Posterior

Evaluation of burns

- Look for circumferential burns (eschar) in chest, neck, buttocks and limbs that may compromise ventilation or circulation. Why?in 3rd degree burn skin will loss of elasticity and become leathery, we know is pt has >20% burns he'll have generalized edema, so the whole body will get swollen and the body will be like a band (due to the leathery skin) → increase pressure → compression of organs, vessels & nerves → less blood flow → ischemia and necrosis (compartment syndrome)
- The treatment is release the limiting tissue (the leathery dead skin) escharotomy
- Loss of distal pulses (late)
- Assess for warmth, sensation, motor, rigidity
- Doppler exam is helpful to assess the blood flow
- Localization of the pain.
    - GIT burns → loss of GI layer → sepsis → death.
- **Identify potential abuse** (mostly suspected in children or elderly patients, check if the story matches the burn type and site if burns look the same)

Compartment Syndrome: extra

Compartment: any closed space in the body that is closed by limiting tissue e.g. fascia or skin. Increase in the pressure in that compartment may result in the syndrome. It Is characterized by compression of nerves, blood vessels and muscle in that compartment.This leads to tissue death from lack of oxygenation due to the blood vessels being compressed by the raised pressure within the compartment.



Two types:

- Compartment syndrome caused by fracture/bleeding, which increases the pressure inside the compartment (due to crushing injury): perform fasciotomy to relieve the pressure [because the limiting tissue here is fascia]
- Compartment syndrome caused by full thickness (dermis & epidermis> Type3) circumferential burns: perform an Escharotomy to release the pressure [because the limiting tissue here is skin].

An **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.

Inhalation Injury:

When do we suspect inhalation injury?

1- If the patient had a **flame burn** + closed space + loss of consciousness

2- Large area + flame burn + burned facial hair or facial burn

3-hoarseness of voice + carbonaceous sputum

Very common injury in a closed smoke retention environment (common in winter)

Smoke inhalation: divided into 3 types

1. **Carbon Monoxide Poisoning**
   - CO has stronger affinity for HGB than O_2
   - Signs of CO poisoning: Confusion, dizziness, Headache, N/V, flushed skin
   - Treatment 100% FiO2[5].
2. **Upper Airway Obstruction**
   - Common in head and neck burns and smoke inhalation
   - Edema continues for at least 24 hours, obstruction can occur after 8-48 hours.
   - Protect airway with prophylactic intubation (you intubate the patient before the obstruction occurs).
   - Edema usually decreases by the third day
3. **Pulmonary Injury from Chemical Inhalation**
   - Develops ARDS within 24 hours post injury
   - Pneumonia may occur as late as in the 10th day → sepsis
   - Treatment: 100% oxygen.

Fluid Resuscitation

- Hypovolemia was a 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
- Fluids are administered intravenously when TBSA of burns is >15% in adults and >10% in Children.

How much IV fluids should we give to a burn patient? (if needed)

PARKLAND Consensus 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.

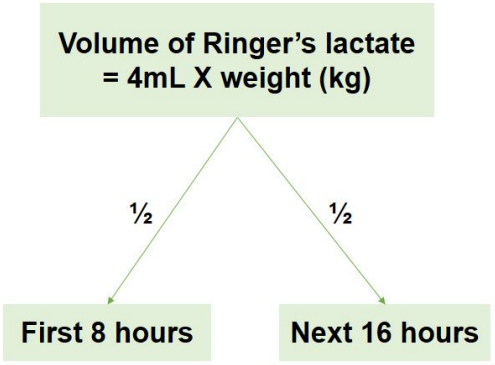
Lactated **Ringer** solution = 2-4ml **x** %BSA **x** body weight (kg)

*(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.



More explanation :

مثال: مريض يحتاج ٨ لتر تعطيه نصف الكمية اللي هي ٤ بأول ٨ ساعات وتكمل الباقي اللي هي ٤ لتر في 16 ساعة الجايه >> هذا في حالة ان المريض جاك بعد الحرق مباشره ما تأخر .

لكن لو افترضنا ان المريض تأخر ساعتين تقوم تطرح ال ٢ ساعتين من ٨ ساعات يعطيك ٦ ساعات, الحين تعطيه نصف الكمية بأول ٦ ساعات و الباقي تعطيه ب ١٦ ساعه الجايه

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

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.

[5] Fraction of inspired oxygen

Burns types:

	Chemical	Electrical	Thermal
causes	-Delayed & progressive injury -Deceptively superficial at first	Passage of electric current	Types : 1-Flame burns (fire) 2-Scald burns (caused by liquids ex: hot water and soups, solutions like soups usually have higher temperature than water.) 3-Contact burn (touching hot objects e.g. Iron)
Damage	-4th degree if it reaches the fat and muscle. -By acids or alkalis (see the notes below) -HFI[6]: significant necrosis, arrhythmias (worst chemical burn because it has both acid and alkaline properties); causes decalcification leading to hypocalcaemia and arrhythmias)	-4th degree if the current passes through the bod -Wound appears small but it's very bad. -Seen in pts who works in factories. -Damage increases in small bony areas e.g. Fingers, feet, lower legs, forearm -Only burns with entry point and exit point.	
Systemic effects	Hypocalcemia	-Most commonly it affect the heart patient can have MI and arrhythmias, can cause a compartment syndrome -Low voltage (<1000 V) (may cause arrhythmias) -High voltage (>1000 V): Massive tissue damage, respiratory and cardiac arrest	
Manage	Removal of causative agent Brush off metals and powders Copious irrigation with water	-ECG, CPK, UA, monitor -Local care often necessitates grafting and amputation	
Notes	Types of chemical burns: **1-Acids**: cause **coagulation** and regular burn necrosis and will stop at that level (limited). **2-Alkalis**: "worse" cause **liquefaction** that may continue for hours after the injury (deep)	-High voltage burns are the only type of burns that have an entry and exit point. -They may be minimal on the surface; we should check the muscles and and bones for any injuries. -Damage mostly affects the small bones (feet, hands, and forearms) -Damage is due to resistance which generates heat, that's why it's common in small bones (because bones have the highest resistance in the body) -electrical burn most common complication is cardiac injury. second most common is compartment syndrome how ? the electrical current will pass to the bone ⇒ the bone will be very hot ⇒ damage the surrounding muscles ⇒ swollen muscles ⇒ compartment syndrome ⇒ treated by fasciotomy	

[6] hydrofluoric acid

Recall:

Which is more serious acid or alkali burns?
In general, ALKALI burns are more serious because the body cannot buffer the alkali, thus allowing them to burn for much longer
Why are electrical burns so dangerous?
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 the level of burn injury:
First-degree burns:
Epidermis 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 burns present?
Painful, hypersensitive, swollen, mottled areas with blisters and open weeping surfaces.
How do third-degree burns present?
Painless, insensitive, 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.
By which measure is burn severity determined?
Depth of burn and TBSA [7]affected by second- and third-degree burns
TBSA is calculated by the “rule of nines” in adults and by a modified rule in children to account for the disproportionate size of the head and trunk.
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**
  - >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 electrical burns

What principles guide the initial assessment and resuscitation of the burn patient?
ABCDEs, then urine output; check for eschar and compartment syndromes.
What are the signs of smoke inhalation?
Smoke and soot in sputum/mouth/nose, nasal/facial hair burns, carboxyhemoglobin, throat/mouth erythema, history of loss of consciousness/explosion/ fire in small enclosed area, dyspnea, low O2 saturation, confusion, headache, coma
How should the airway be managed in the burn patient with an inhalational injury?
With a low threshold for intubation; oropharyngeal swelling may occlude the airway so that intubation is impossible; 100% oxygen should be administered immediately and continued until significant carboxyhemoglobin is ruled out.
What is “burn shock”?
Burn shock describes the loss of fluid from the intravascular space as a result of burn injury, which causes “leaking capillaries” that require crystalloid infusion

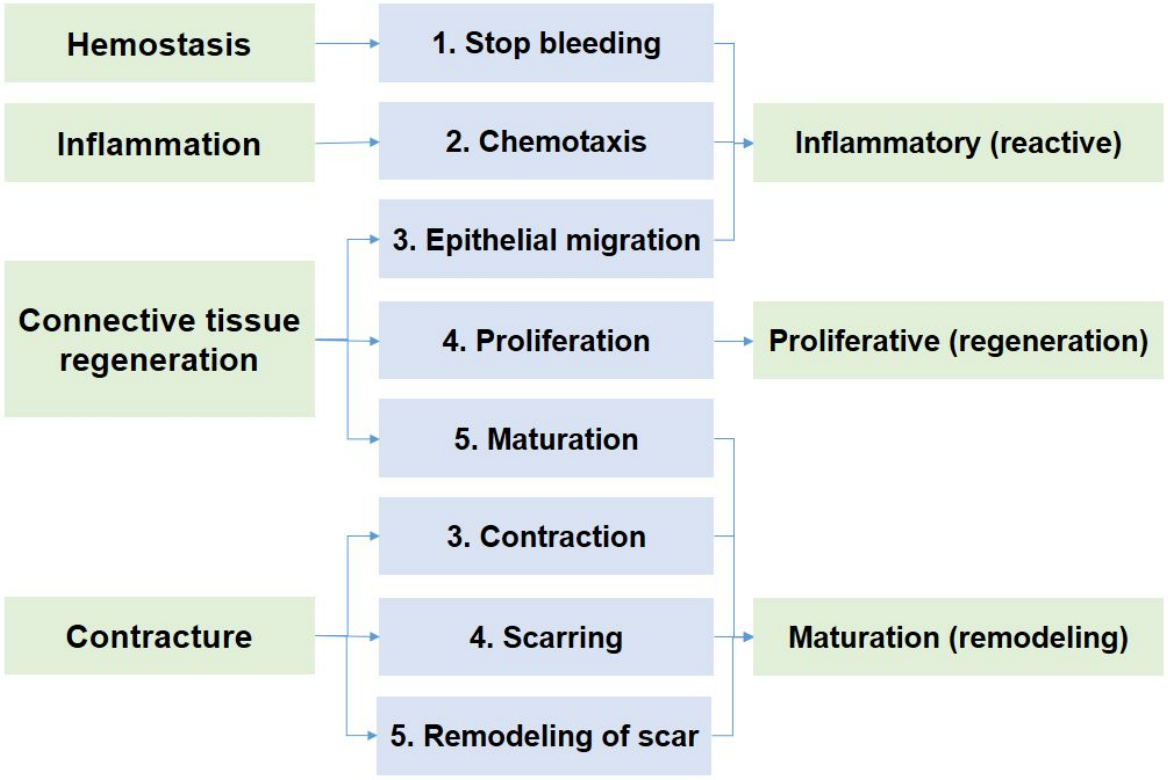
[7] Total Body Surface Area

What is the "Parkland formula"?
V =TBSA Burn (%) X Weight (kg) X 4 Formula widely used to estimate the volume (V) of crystalloid necessary for the initial resuscitation of the burn patient; half of the calculated volume is given in the First 8 hours, the rest in the next 16 hours
What burns qualify for the Parkland formula?
≥ 20% TBSA second- and third-degree burns only.
What is glucose-containing IVF contraindicated in burn patients in the first 24 hours postburn?
Patient's serum glucose will be elevated on its own because of the stress response
What fluid is used after the first 24 hours postburn?
Colloid; use D5W[8] and 5% albumin at 0.5 cc/kg/% burn surface area
How is volume status monitored in the burn patient?
Urine output, blood pressure, heart rate, peripheral perfusion, and mental status; Foley catheter is mandatory and may be supplemented by central venous pressure and pulmonary capillary wedge pressure monitoring
Why do most severely burned patients require nasogastric decompression?
Patients with greater than 20% TBSA burns usually develop a paralytic ileus → vomiting → aspiration risk → pneumonia
What stress prophylaxis must be given to the burn patient?
PPI to prevent burn stress ulcer (Curling's ulcer)
What are the signs of burn wound infection?
Increased WBC with left shift , discoloration of burn eschar (most common sign), green pigment, necrotic skin lesion in unburned skin, edema, ecchymosis tissue below eschar, second- degree burns that turn into third-degree burns, hypoten.
Why are systemic IV antibiotics contraindicated in fresh burns?
Bacteria live in the eschar, which is avascular (the systemic antibiotic will not be delivered to the eschar); thus, apply topical antimicrobial agents
Are prophylactic systemic antibiotics administered to burn patients?
No—prophylactic antibiotics have not been shown to reduce the incidence of sepsis, but rather have been shown to select for resistant organisms; IV antibiotics are reserved for established wound infections, pneumonia, urinary tract infections, etc.
Circumferential, full-thickness burns to the extremities are at risk for what complication?
Distal neurovascular impairment
How is it treated?
Escharotomy: full-thickness longitudinal incision through the eschar with scalpel or electrocautery
What is the major infection (other than wound infection) in burn Patients?
Pneumonia, central line infection (change central lines prophylactically every 3 to 4 days
From which burn wound is water evaporation highest?
Third degree
Can infection convert a partial thickness injury into a full thickness injury?
Yes!
How is carbon monoxide inhalation overdose treated?
100% O2 (± hyperbaric O2)
Which electrolyte must be closely followed acutely after a burn?
Na (sodium)

[8] D5W (5% dextrose in water)

Wound healing davidson page 92

Wound: is a disruption of normal anatomical 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.



Phases of wound healing (Three phases):

It is important to know what happens in each stage, how long it takes, what's the main cell what's the aim, and what're the symptoms?

1- Inflammatory (lag) phase:

- **Aim**: to stop bleeding.
- Substrate or reactive phase, immediate
- Typically takes **days 1-10** This is in case of closed or clean wound, but contaminated wounds will take longer.
- aims 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 (our aim is to stop bleeding).
- Tissue injury & blood vessel damage → Exposure of **subendothelial collagen** to **platelets** and **vWF** activates the **coagulation** pathway.
- **Plugging:** Platelet and fibrin
- **Provisional matrix** (all the tissue cells in skin are lined within the connective tissue, and when it's damaged it is replaced immediately by the provisional matrix): (Is the primitive tissue formed after platelet plug later on replaced by collagen) platelets, fibrin, and fibronectin.
- **Platelet aggregation will releas** Thromboxane (vasoconstrictor), thrombin, platelet factor 4.

Platelets	Polymorphonuclear Cells -hyperemia	Macrophages
Have Alpha granules that contain: - platelet factor 4: aggregation - Beta-thrombomodulin: binds thrombin - PDGF: chemoattractant - TGF-beta: key component for tissue repair. Transforming growth factor Beta produced by platelets is important for wound healing, if not present; wound healing doesn't occur. - Also have Dense granules contain vasoactive substances: adenosine, serotonin, and calcium. - Other factors released: TXA^{9}, Platelet activating factor, Transforming growth factor alpha, Fibroblast growth factor, Beta lysin (antimicrobial), PGE2 and PGI2 (vasodilate) and PGF2 (vasoconstrict).	-(it's the second step, which involves cleaning of the injured tissue) -Chemotaxins attract PMN 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 usually within a few days. (Sign of wound contamination control) -Persistent contaminant: continuous influx of PMNs and tissue destruction, necrosis, abscess, & systemic infection. -PMNs are not essential to wound healing. -Even though they are very important in wound healing but they are not the key cells of the inflammatory phase of wound healing; macrophages are.	-Necessary (Macrophages are the most important cells in the inflammatory phase of wound healing, they release the mediators and control the work of all other cells) -Monocytes migrate & activate: Macrophages. -Appear when PMNs disappear 24-48 hrs. -Do the same activities as PMNs -Plus orchestrate release of enzymes (collagenase, elastase), PGE's, cytokines (IL-1, TNF alpha, IFN), growth factors (TGF & PDGF), and fibronectin (scaffold/anchor for fibroblasts). -Activate Fibroblasts, endothelial and epithelial cells to form Granulation Tissue. -Macrophages induce PMN apoptosis -Migrate similar to PMN's secrete enzyme to degrade and alter ECM instigate fibroblast -Fibronectin attract more phages

2- Proliferative (incremental)

- **Aim:** to start healing.
- Regenerative or Reparative
- **day 5 to 3 weeks** (The Proliferative phase depends on Fibroblasts).
- **Angiogenesis** (formation of new blood vessels for good blood supply): endothelial cells activate & degrade basement membrane, migrate, and divide to form more tubules.
- **Granulation Tissue** (if you see any person at day 5 or 6 with an open, clean and red wound that easily bleeds when you touch it, this is granulation tissue, it's a good sign that there is no more insult to the wound, and it's starting to heal): capillary ingrowth, collagen, Macrophages, Fibroblasts, Hyaluronic acid (GAG).
- **Fibroblasts:**
  - Differentiate from resting mesenchymal cells in connective tissue **3-5 days** migrate from wound edge.
  - **Fibroplasia**: Fibroblasts proliferate replace fibronectin-fibrin with collagen contribute ECM. Goal of fibroblast is to replace the provisional matrix with collagen.

Provisional matrix will be replaced by hyaluronic acid and then it'll be replaced by collagen!

[9] Thromboxane

- **Collagen:**
  Types of collagen:
  - **Type I (80% skin):** Most Common: skin, bone, tendon. **Primary type in wound healing.** synthesis begins at days 1-2.
  - **Type II:** Cartilage.
  - **Type III (20 % skin): Increased Ratio** in healing wound, also blood vessels and skin. Fibroblasts will start by secreting this type at 1-2 days then replaced by type I in 3 weeks.
  - **Type IV:** Basement Membrane.
  - **Type V:** Widespread, particularly in the cornea.

(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

- **Wound strength**
  - (Granulation tissue contains: Capillary ingrowths, Collagen, Macrophages, Fibroblasts, Hyaluronic acid (GAG))
  - Week 6 (increase in collagen secretion, starting the maturation phase) = 60% original, 80% final strength.
  - Week 8 -1 year ≈ 80% original (Max). it will never go back to normal except for the bones they can go back to 100%!
  - Net Collagen = 6 weeks amount stays the same but cont. crosslink increase strength = maturation.

3- Maturational (plateau):

- Aim: to increase strength.
- Remodeling of wound
- start at 3 week until 1 year.
- **Type I** replaces **Type III** Collagen.
- Decreased vascularity, less fibroblasts & hyaluronic acid.
- Peripheral nerves regenerate at 1mm/day.
- Accelerated Wound Healing: reopening results in quicker healing 2nd time around.

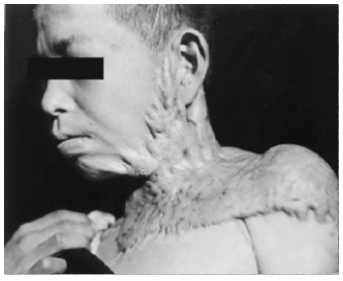
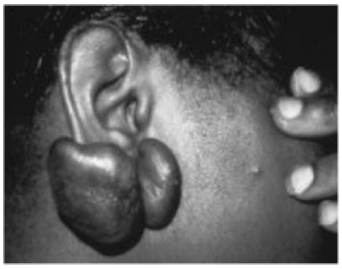
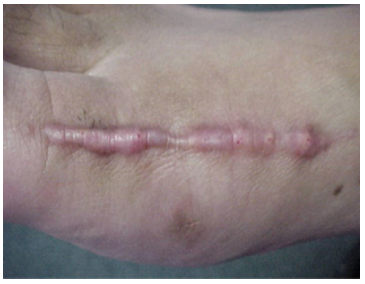
Contraction:

- Centripetal movement of the whole thickness of surrounding skin reducing scar. (a normal physiological process to minimize wound size)
- **Contracture** is basically **contraction** gone wrong→ keloid,hypertrophic 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	Keloids: Beyond the Borders	Hypertrophic Scar: confined within wound area
Contraction: centripetal movement of the whole thickness of surrounding skin reducing scar (a normal physiological process to minimize wound size). **Myofibroblasts** (helps in shrinking the size of the wound): 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:** (A minimization for wound's size due to myofibroblasts) The physical constriction or limitation of function as the result of Contraction (scars across joints, mouth, eyelid). (pathological process that affect joints and bones) When burn causes an over contractions and start to affect the function of a joint. it is called a **Contracture**. Most common sites: Perineum and Trunk, then Head and neck, then Extremities. Burn/Keloid causing Contracture It took a very long time in proliferative phase, a lot of collagen, and it will affect the movement of the neck. 	Beyond the borders: excess Deposition of Collagen Causes Scar Growth Beyond the Border of the Original wound. (grows out of the borders of the initial injury) Caused by specific genetic predisposition: Autosomal Dominant, Darker Pigment, Often above clavicle like sternum and shoulders but not always. Happens in areas with no tension. Tx: Intralesional excision, XRT[10], steroids, silicone sheeting, pressure, excise. **often Refractory to Tx & not preventable.** Excision alone makes it worse Occur in specific areas such: earlobes and sternum Here we try everything before we do surgery, because if we remove it by surgery, it might recur, and get worse! So, first we give injection steroids and silicon. If that didn't help, we treat it like we're treating a tumor: intralesional excision and radiotherapy, and this is the last treatment choice. 	Limited to the site of injury: excess collagen deposit causing raised scar remains within the original wound confined.(limited to the site of wound) Genetic but to a lesser extent. Happens in areas with tension like joints. Darker pigmented skin & flexor surfaces of upper torso. Often occurs in burns or wounds that take a long time to heal, sometimes preventable.(occurs in any place in the body) Can regress spontaneously. Tx: steroids, silicone, pressure garments. Surgical excision makes it worse Treated by injection steroids or silicon. In excessive tension: we do a surgery. 

[10] Radiotherapy

Impediments to Wound Healing:

- **Bacteria**>$10^5/cm^2$: causes 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.
- **Diabetes**: impedes the early phase response.
- Malnourishment: **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.
- Most dangerous burn complication is **SEPSIS**.
- Most common organisms before antimicrobials: **staph aureus** and group A streptococcus.
- Most common organisms after antimicrobials: **pseudomonas**

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).

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.