Reviewed By
Noura Alturki
Jehad Alorainy

Approach to wound healing & Burn case-scenario

Objectives

Approach to wound healing The students should be able to:

- List and explain the phases of Normal Wound Healing
  - The hemostatic phase,The inflammatory phase, The proliferative phase,The remodeling (maturing) phase
- List and describe the types of Wound Healing
  - Healing by primary intention (first intention),Healing by secondary intention, Healing by tertiary intention (delayed primary closure)
- Identify and differentiate between the factors Affecting Wound Healing
  - Local factors: Wound site, Wound contamination, Infection, Mechanism of wounding, Tissue loss, Hematoma formation in the wound, Vascular insufficiency, Previous radiation of wounded area, A pressure in the wounded area
  - Systemic Factors: Malnutrition, Uncontrolled diabetes, Medications, Chronic diseases, Immunosuppression, Smoking
- List and discuss the types of wounds
  - Acute Wound
  - Chronic
- Classify Wounds According to the Mechanism of Wounding
  - Clean. Avulsion, Abrasion, Puncture,Crushing
- Explain the Management plan of Acute Wound
- Explain the Management plan of Chronic Wound
- Explain the mechanism of Compartment Syndrome
- Describe Degloving Injury
- Demonstrate the following points regarding leg ulcer:
  - List the different types of leg ulcer including: Venous Ulcer, Ischemic Ulcer, Traumatic Ulcer, Chronic Infection, Neoplastic Ulcer, Pressure ulcer, Venous ulcer, Ischemic ulcer,Diabetic Ulcers
  - List and explain the Risk factors, and the prevention mechanisms of Different Leg Ulcer
- Abnormal scars:
  - Explain how scars are formed
  - Explain the different abnormal scars and how they are formed
    - Atrophic scar, Hypertrophic scar, Keloid scar, Contracture scar
  - Explain the Management plan of abnormal scar

Burn- case scenario

- The student is expected to describe and perform the following:
- Calculation
- Management
  - Surgical options
  - Non surgical options

Colour Index

- Main Text
- Males slides
- Females slides
- Doctor notes
- Textbook
- Important
- Golden notes
- Extra

Editing File

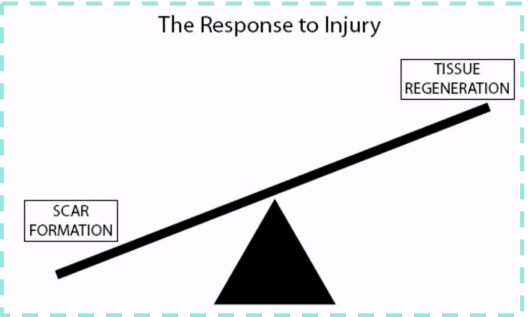
Wound healing

Definitions

- **Wound**: disruption of normal anatomical structure and function on the skin or internal organ
  - **Acute wound**: proceeds through an orderly and timely reparative process to restore function and anatomical integrity (3-4 wk).
  - **Chronic wound**: proceed through a reparative process without establishing a sustained anatomic and functional result (beyond 6 wk)
- **Healing**: response of an organism to a physical disruption in an organ or a tissue to repair a defect.
  - Abnormal healing:
    - Overgrowth: keloid or hypertrophic scar.
    - Undergrowth: wide or thin or chronic unstable.
    - Abnormal pigmentation: hypo/hyper
      Whenever there is injury in the skin the melanin production can be over or under produced

Scar formation VS regeneration:

- **Scar formation**: substitution of a different cellular matrix as a patch to immediately re-establish both a physical and a physiological continuity of the injured organ. Will not behave like normal tissue
- **Regeneration**: recreating the original organ by the developmental process which created it.
- The more the tissue regeneration the better the function of the organ



Wound contraction:

- Active, normal & essential part of healing, that starts from the proliferative phase.
- This is beneficial especially in bigger or chronic wounds where the contracture will bring the cells together at a faster rate which will accelerate the healing process.
- Rate depends on the number of myofibroblasts
  - Myofibroblasts resemble fibroblasts but contain smooth muscle Actin.
  - Responsible for wound contraction.
- From end to end
- Contracture: an undesirable result of healing due to excessive contraction and fibrosis.

Wound:

Collagen

- **Type I ≅ 90% of all collagen in body**
- Type IV collagen found in basement membrane
- Normal skin ratio - **Type I:Type III = 4:1**
- Hypertrophic / immature scar 2:1 ratio
- **Formation inhibited by:**
  - Colchicine (a medication used in Gout), penicillamine (anti tumor), steroid, Vit.C deficiency and Fe deficiency.

★ They activate collagenase which degrades collagen synthesis and inhibits cross linkage hydroxylation of lysine and proline.

Classification of Wound Closure:

Primary healing (1st intention)	Secondary healing (2° intention)	Tertiary healing (3° intention)
• Primary closure by suturing the edges together. • Within hours of repairing full-thickness surgical incision. • Results in mortality of minimal number of cellular constituents. • Considered the most efficient method and results when a clean incised surgical wound is meticulously apposed.	• Wound left open to heal[1] by processes of **granulation**, **contraction**[2], and **epithelialization**[3]. • Results in more intense inflammatory response. • Larger quantity of granulation tissue with pronounced contraction of wounds. • Like chronic wounds	• Delayed primary closure • Desired for contaminated wounds (infected wounds)[4]. • Phagocytosis of contaminated tissues well underway by 4th day. • Foreign materials walled off by macrophages. • 3° intention healing describes the situation where a wound healing by 2nd intention (neglected traumatic wound/burn) is treated by excising its margins and then apposing them or covering the area with a skin graft. • The final cosmetic result may be better than if the wound had been left to heal by 2° intention.

Note

Examples of some cases where we use 2nd wound healing intention are:
1. Perineal abscess 2. Diabetic foot.
If we treat by 1st intention it may lead to Cellulitis

Do we need to give the patient antibiotics? If the wound is fresh and clean, it is not preferred to give prophylactic Abx
If dirty —> always start with oral antibiotics (IV requires admission)

1. You keep the wound open (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.
2. **Contraction** results in edge to edge (**horizontal**) repair.
3. **Epithelialization** results in **vertical** repair.
4. Tertiary healing is a combination of primary healing and secondary healing. Clean the contaminated wound, wait for about 3 days and then re-approximate it by suturing.

Note

If a patient presents with a wound, always ask about Tetanus status:

- Received a booster in the last 5-10 years?
  If not —> give a booster
- If a child is not immunized or missed a dose in childhood —> give passive and active immunizations

Phases of Wound Healing:

01 Heamostasis[1] (5-10min)

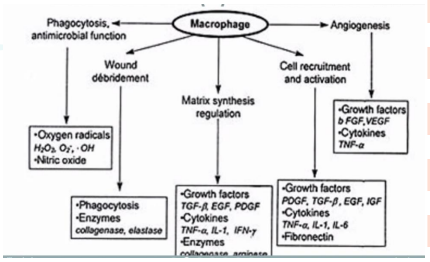
Main cells in this phase: platelets

- **Initial** response to injury = **constriction** followed by dilation. (vasoconstriction is to reduce blood loss and aggregate the platelets)
- 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 :

platelet derived growth factor (PDGF)

a. made by macrophages, endothelial cells, fibroblasts
b. chemotaxis, fibroblast stimulation

Chemotaxis: dealing with debris from the wound

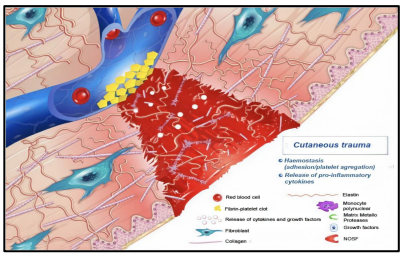


transforming growth factor β (TGFβ)[2]

a. made by macrophages, platelets, fibroblasts
b. fibrinogenesis, angiogenesis, chemotaxis, immune suppression

fibroblast growth factor

a. made by macrophages and endothelial cells
b. angiogenesis and chemotaxis



- Form the platelet plug
- Degranulation of platelets (release of cytokines and growth factors)
- Activation and recruitment of neutrophils

1. Considered as phase zero or part of the inflammatory phase. This phase is delayed by bleeding disorders.
2. It has a **major role** in wound healing. The main source for the production of TGFβ is the Alpha granules of platelets. Excess production of TGF-β isoforms causes abnormal scars (Hypertrophic and Keloid scars).

Phases of Wound Healing cont.:

02 Inflammatory/Migratory "lag" phase[1] (1-4 Days)

Typically starts immediately after hemostasis.

Main cells in the inflammatory phase: in the first 24 hours → **PMNs (neutrophils)**

After 24 hours → Macrophages.

Classically represented by:

01 **Rubor (redness) caused by:**

- vasodilation
- primarily result of prostacyclin (PGI_2) and histamine, also caused by prostaglandin A, D, E (PGA, PGD, PGE)

02 **Tumour (swelling):**

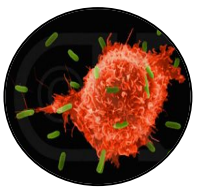
- caused by leakage of plasma proteins through gaps in vascular endothelium
- edema potentiated by PGE_2, prostaglandin $F_{2\alpha}$ ($PGF_{2\alpha}$)

03 **Calour (heat)**

Increased local temperature secondary to both:

- Increased blood flow
- Elevated metabolic rates

04 **Dolour (pain)**



Macrophages :

- Phagocytosis
- Wound debridement
- activation of fibroblast
- Angiogenesis
- Matrix synthesis (granulation tissue formation) regulation

1. Characterized by an inflammatory response to injury, through an increased capillary permeability, proliferation of capillaries at wound edges and accumulation of protein-rich exudate preceding collagen synthesis

Phases of Wound Healing cont.:

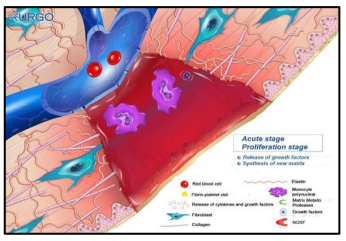
03

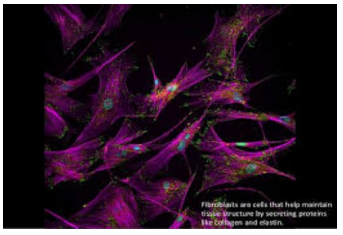
Proliferative/Fibroplasia "incremental" [1] (3 days - 3 weeks)

- Begins 2-3 days after wounding.
- ★ **Signalled by arrival of fibroblasts** (**Main cells in the proliferative phase**, they play a role in collagen synthesis).

  - Driven by macrophage-derived **bFGF**, **TGFβ**, **PDGF** to proliferate and synthesize glycosaminoglycans (**GAGs**) and **proteoglycans** (building blocks of new extracellular matrix of granulation tissue and collagen).
  - Also produce **bFGF**, **TGFβ**, **PDGF**, **keratinocyte growth factor**, **insulin-like growth factors-1**.
  - Dominant cell type peaking at 7-14 days.

- Collagen synthesis[2] (net production for next 3-6 weeks).
- ↑ Keratinocyte mitosis, ↑Endothelial cells and ↑Angiogenesis (from vessels at wound margins).
- Lasts 2-4 weeks depending on site and size of wound with slowing of fibroblast migration and proliferation.
- Different cells differentiate into different types and new blood vessels are formed (angiogenesis).





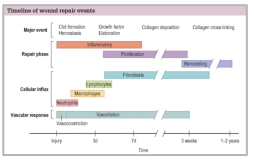
Collagen and elastin synthesis

1. In this phase there is progressive collagen synthesis by fibroblasts and a corresponding increase in tensile strength with Increased collagen turnover. This phase is delayed by: Microvascular diseases (Diabetes), Macrovascular (Atherosclerosis).
2. Arterial oxygen tension (PaO2) is a key determinant of the rate of collagen synthesis.

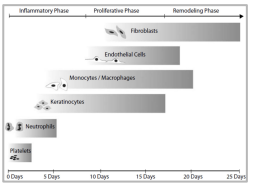
04

Remodeling /Maturation (3 weeks - one year)

- Begins approx. 3 weeks after injury
- Collagen synthesis and degradation are accelerated but in equilibrium with collagen breakdown (no net increase in collagen content)[1]



- Collagen deposition peaks by 3rd week
- Large capillaries growing into wound regress/disappear
- Indurated, raised, pruritic scar becomes mature scar
- Collagen fibers become organized

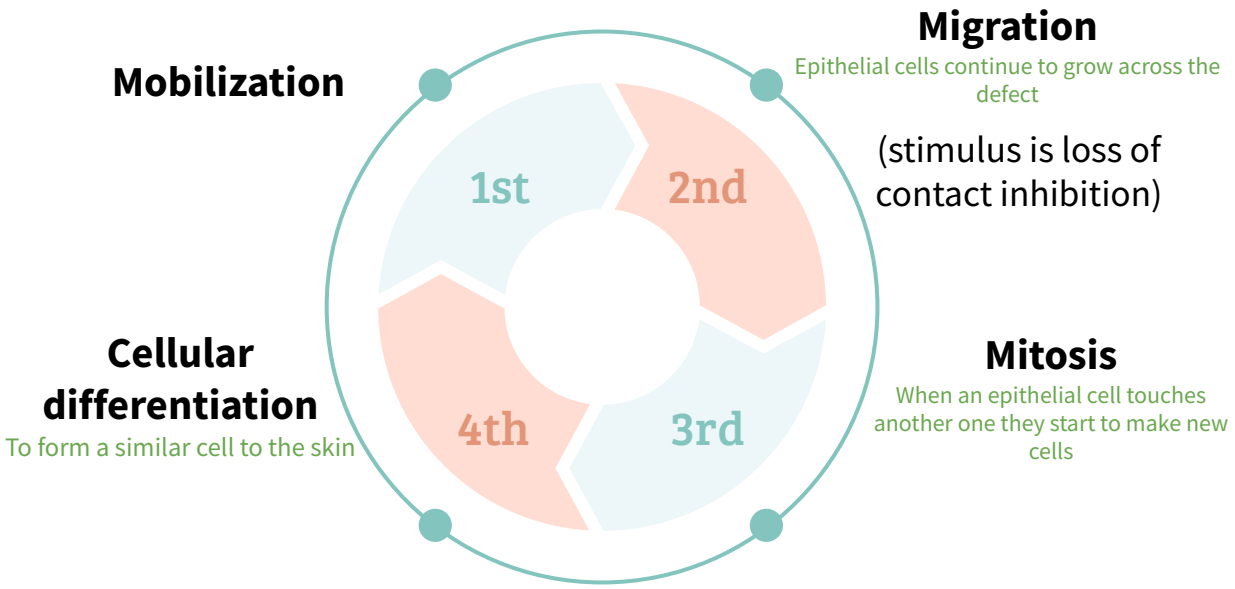


- **Type III collagen replaced by type I collagen**
- **re-establishing normal 4:1 ration (I:III)**:

  Duration depends on age, genetics, type of wound, location (1-2 years)[2].
- Tensile strength increases to 80% of pre-injured skin[3]
- Collagen becomes more organized in shape depending on the location of the wound (6-8 weeks)

Epithelial Repair: outermost portion of the skin that heals from edge to edge

- Epithelial continuity is reestablished across a wound: Multiple events happen during the healing process. The cells (from the two edges of the wound) mobilize & migrate to the middle and get in proximity then duplicate by mitosis and differentiate to different cells.



1. The major event in the remodeling phase.
2. Type three collagen is the main collagen present in the wound healing process. 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.
3. Especially in healing tendons, muscles, and blood vessels. For example for tendons it reaches up to 60-80%, but for normal wounds it usually reaches 100%.

Wound healing

Factors affecting wound healing:

The aim of wound care is to achieve healing by primary intention and thereby reduce the proliferative and inflammatory phases, by correcting any abnormality that can retard or slow the wound healing.
Several local and systemic factors can influence the wound healing, that can result in delay or abnormal healing process which will lead to a poor cosmetic outcome and delay recovery of the function of the wounded part.
These factors should be addressed and controlled by the treating clinician.

Local factors

1

Blood supply

- Decreased tissue perfusion results in decreased wound oxygenation.
- Fibroblasts are oxygen-sensitive and their function is reduced in hypoxic states.
- Poor blood supply also leads to decreased nutrients delivery which will retard the wound healing.

2

Radiation

- Causes endothelial cell, capillary and arteriole damage.
- Irritated fibroblasts secrete less collagen and extracellular matrix.
- Lymphatics are destroyed resulting in edema and risk of infection.
- Previous radiation of wounded area also results in vasculitis with local hypoxia, ischemia, reducing the amount of oxygen and nutrition to the wound site.

3

Infection

- Wounds with over 105 organisms per gram of tissue are considered infected and are unlikely to heal without further treatment with debridement and Abx
- It reduces the rate of wound healing, granulation tissue formation, oxygen and nutrient delivery.the use of debriding agent to clean the wound and appropriate prophylactic antibiotics is important in preventing wound infection.

Wound healing

Local factors

4 Wound site

The presence of persistent pressure or recurrent trauma at wound site may compromise the healing of the wound.

5 Wound contamination

Such wound require debridement, removal of all dead tissues and foreign bodies, and cleaning.

6 Hematoma formation in the wound

Bleeding must be controlled because hematoma formation in the wound predisposes to wound infection and it separates the wound edges and prevents wound healing.

7 Mechanism of wounding

Incisional wounds will follow the normal phases of wound healing, whereas crushing or avulsion wounds may not heal in normal process and may have a prolonged healing process.

8 Tissue loss

This will delay the slow wound healing and predispose to infection.

9 Pressure in the wound area

Pressure in the wound will reduce the blood supply, which causes hypoxia, ischemia, and retarded wound healing.

In addition to Nerve supply, trauma, hydration and temperature.

Systemic factors

- Congenital
- Acquired

- Pseudoxanthoma elasticum
  - Autosomal dominant
  - Problem with collagen cross linkage → weakness and failure of healing
- Ehler-Danlos syndrome
  - AD, defect in synthesis, structure and cross linkage of collagen
- Cutis laxa
- Progeria
- Werner syndrome
- Epidermolysis bullosa
  - Similar to a burn

Table 1-2 *Diseases and Conditions*

	Defect	Characteristics	Surgical Intervention
Ehlers-Danlos syndrome	Abnormal collagen structure, production of processing	Hyperflexible joints Stretchy, fragile skin Easy bruising Vascular aneurysms	Not recommended
Progeria	Mutation in *LMNA* gene	Limited growth Full body alopecia Wrinkled skin Atherosclerosis Large head, narrow face, beaked nose	Not recommended
Werner syndrome	Mutation in *WRN* gene	Graying of hair Hoarse voice Thickened skin Diabetes mellitus Atherosclerosis Cataracts	Not recommended, but reported for temporary improvements
Pseudoxanthoma elasticum	Fragmentation and mineralization of elastic fibers	Cutaneous laxity Yellow skin papules Vision loss	Redundant skin folds can be treated with surgical excision
Cutis laxa	Mutation in elastic fibers	Loose, wrinkled skin Hypermobile joints	Surgical excision of redundant skin produces temporary benefit but patients do **not** have wound healing problems

Factors affecting wound healing:

Systemic factors

- Congenital
- Acquired

Malnutrition:

Nutrition is extremely important for protein and collagen synthesis, and metabolic energy for wound healing.

Folic acid is important for the synthesis of collagen.
Magnesium is required for the synthesis of protein.

Vitamin K is essential for the process of carboxylation in the synthesis of coagulation factors II, VII, IX, and X. deficiency of vitamin K will cause coagulopathy and hematoma formation in the wound that retards the wound healing.

Vitamin A
Deficiency delays wound healing. Because it's part of the structure of collagen fibers
25,000 IU po OD increases tensile strength.
200,000 IU topical Q8 increases epithelialization.
Increases collagen synthesis, inflammatory response, and recruitment of macrophages into the wound.

Vitamin C
Collagen synthesis.
Scurvy : Immature fibroblasts,deficient collagen synthesis, capillary hemorrhage, decreased tensile strength.

Vitamin E
Antioxidant and a membrane stabilizer.
Large doses may inhibit healing but unproven to reduce scarring.
Too much Vit E will negatively affect wound healing

Zinc is an essential cofactor for many enzymes and the synthesis of RNA and DNA.
Deficiency causes impared epithelial and fibroblast proliferation. Deficiency of zinc can also affect migration and mobilization

Chronic diseases

Diseases such as liver disease, jaundice, malignancy, uremia, will dispose to malnutrition, wound infection, and delay wound healing.

Factors affecting wound healing:

Systemic factors

- Congenital
- Acquired

Pharmacological

- Steroids decrease inflammation and subsequent wound healing.
- NSAIDS decrease collagen synthesis and inhibit platelet aggregation.
- Antineoplastic agents decrease fibroblasts proliferation and wound contraction.
    - Few or no adverse reaction of administered 10-14 days after wound closure.

(Endocrine abnormalities) Uncontrolled diabetes

- Diabetics often have delayed wound healing.
- Neuropathy rather than small vessel occlusive disease may be responsible for delayed healing.
- Hyperglycemia will inhibit fibroblast proliferation, alter collagen formation, and retard wound healing. Hyperglycemia predisposes to wound infection by making good media for bacteria growth, and suppression of phagocytic function of neutrophils.

Smoking

- Nicotine causes vasoconstriction decreasing perfusion. Decreasing cell production
- CO shifts the oxygen dissociation curve to the left and reduces tissue oxygenation.
- It also reduces collagen formation in the wound.

Age

- Rate of cell multiplication decreases with age.
- All stages of healing are protracted in elderly.
- Healed wounds have less tensile strength in elderly.
  Thus their wounds break easily and no problem with hypertrophic scars and keloids

Immunosuppression

Immunosuppression status due to some conditions (leukemia, post-transplant, immunosuppressive drugs, chemotherapy, and AIDS) will predispose to wound infection, reduce the inflammatory response, and retard wound healing.

Decreased oxygen delivery to tissue reduce:

- Collagen formation
- Extracellular matrix deposition
- Angiogenesis
- Epithelialization

Classification of contamination in Surgical wounds

(WHO classification of wound):

1

Clean:

- nontraumatic, non infected wounds & no breach of Resp, GI, or GU tract.
- No spillage of the content of the tract itself.
- E.g. thyroid and breast surgeries.
- No need for antibiotics.
- Infection rate should be less than 1%.
- Use of Ab prophylaxis isn't recommended.

2

Contaminated "Dirty":

- Fresh traumatic wounds; major break in sterile technique, **nonpurulent inflammation**; in or near contaminated skin.
- Major spillage.
- E.g. hemicolectomy or resection of the intestine with spillage, emergency surgery for perforated diverticular disease, or drainage of a subphrenic abscess.
- Give prophylactic Abx

3

Clean-contaminated:

- Small breach in protocol; Resp/GI/GU tract are entered with minimal contamination.
- Very minor spillage of the content.
- E.g. cholecystectomy, uncomplicated appendicitis, intestinal resection ONLY if there was no spillage.
- Infection rates in excess of 5% may suggest a breakdown in wards.
- Give prophylactic Abx

4

Infected:

- **Purulent infection**.
- Traumatic & severe wounds.
- They have positive culture and require broad spectrum antibiotics.
- E.g. traumatic open bone fracture, and purulent pyogenic perforated appendicitis.
- Must go to specialized OR

Types of wounds

Wounds can be classified into:

1. Acute wounds such as clean incised, avulsion, abrasion puncture and crushing.
2. Chronic wounds such as venous, ischemic, pressure and diabetic ulcers.

Acute wounds <24 hours

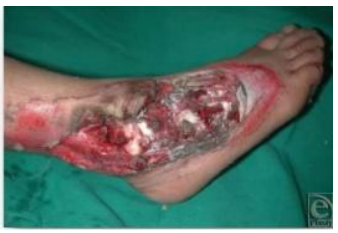
Tidy

- Incised, clean wound in healthy tissue without tissue loss such as a wound from a clean sharp object.
- Can be closed immediately.
- Heals by primary intention.

VS

Untidy

- Crushed or avulsed tissues with contamination, devitalized tissues and tissue loss, such as wounds caused by explosion, missile or bullet injury.
- Requires debridement, excision of devitalized tissues, and cleaning several times before definitive repair is performed
- Usually heals by secondary or tertiary intention.



Chronic wounds

Ischemic ulcer

This ulcer results from atherosclerosis of the main lower limb arteries or due to thromboembolic occlusion of theses arteries

Ischemic ulcer contains little granulation tissue, indicating impairment of healing process with exposure of deep structures such as bones, tendons, and joints with black mummified (dry gangrene) or black suppurative (wet gangrene) tissues.

Ischemic ulcer most commonly involves the toes but any part of the leg or foot can be affected.

Peripheral pulses (dorsalis pedis and posterior tibial) will be absent, but the evaluation with doppler ultrasound is more accurate.

Ischemia is the harbinger of limb loss due to infection when associated with diabetic ulcers.

Treatment:

1

- Intervention is very important to save the limb and prevent major amputation.

Endovascular balloon angioplasty or stenting is the appropriate intervention for focused obstruction or stenosis

long segment arterial obstruction or stenosis require **bypass surgery with the autologous saphenous vein or synthetic graft.**

- This is to improve tissue perfusion and enhance ulcer healing. Most ischemic ulcer will not without revascularization.

2

- After intervention and improvement of blood supply, wounds debridement, cleaning, excision of all dead tissues, local antibiotics and amputation of dead parts (e.g toes) are essential.

- Systemic antibiotics are required in the presence of infection.
- Incompletely healed clean ulcers with the favorable condition may require skin flap or graft fore coverage.

Acute wounds cont.

Classification of wounds according to the mechanism of wounding:

1

Clean
New wound (<12 hours), minimal contamination, may need debridement, usually closed primarily, the edges approximated with an appropriate method.

2

Avulsion
Shearing force causing such wound, with skin flap or total skin loss, and exposure of underneath structures.
Management includes several debridement sessions, cleaning, with a pressure dressing to prevent accumulation of blood and serum under the skin flap of sometimes a skin graft or flap os required when there is significant blood loss.

3

Abrasion
This is a superficial loss of epithelial cells and portions of the dermis with intact deep structures.
Management includes cleaning to prevent traumatic tattoo and and to allow the wound to heal by regeneration of epithelial cells.

4

Puncture
Such wound does not need closure, but assessment for deep structures injury or deep foreign bodies by imaging and clinical follow up to detect the development of infection is required.

5

Crushing
This injury is associated with significant loss of tissues including different structures that may initially appear viable. Non-viable tissues need debridement, cleaning of the wound, closure with a skin graft or myocutaneous flap depending on the exposed underlying structures.

Acute wounds cont.

Management of acute wounds:

- Management of the patient should follow the acute trauma life support (ATLS) principles.
- The patient should be examined completely and a stab wound in the wound should not be missed.
- The wound should be examined, taking into consideration the site and the possible damaged structures including vascular structures.

1

2

- Assess the severity of the pain and motor and sensation function.
- Tetanus therapy should be assessed and appropriate treatment given.

- Pressure pad or tourniquet should be applied over the bleeding wound. Clamps should not be applied blindly to a bleeding wound because it may cause nerve damage and cause further damage to the vessels which rendered vascular anastomosis impossible.

3

4

- Adequate anesthesia (local, regional or general) is sometimes required to facilitate the examination. The toxic level of local anesthesia should not be exceeded.

- Prophylactic antibiotics for anaerobic bacteria are required in certain cases and wounds in a certain location. The tourniquet should be used in limb injuries in order to facilitate examination and visualization of all structures. During tourniquet application, care should be taken to avoid uneven pressure and the tourniquet time must be noted.

5

6

- An x-ray may be required to rule out deep foreign bodies.
- After complete assessment, a thorough debridement of the wound should be performed. All dead or devitalized tissue should be excised and foregin bodies should be removed.copious irrigation of the wound with saline is helpful. Debridement should be carried out until bleeding occurs. Avoid injuries to the nerves,vessels, and tendons.

- Muscle viability is judged by the color, bleeding pattern, and contractility.
- Repair of damaged structures (nerves, vessels, and tendons) maybe attempted in a tidy wound.
- Large hematoma may require evacuation by incision or aspiration. If the large hematoma is not evacuated, it calcifies with htime, and cause symptoms include local lump or mass.

7

8

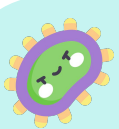
- Skin cover by graft or flap may be required if there is skin loss, as skin closure should always be without tension.
- Needle stick injuries should be managed according to the protocols of hepatitis and HIV risks.

- Preparation of the wound by cleansing and debridement before closure is important. All wounds are contaminated, even the wounds that considered to be clean contain bacterial inoculum. Therefore, lavage and adequate debridement are essential steps in wound care. The decision to close the wound primarily depends on the extent of contamination.for example, the incised clean wound can be closed primarily, but crush wounds, human bite or farm injury should be managed as open wound till the appropriate condition of closure is reached. Delayed closure give time for further debridement, cleaning and reduction of bacterial count in the wound.

9

10

- The dressing of the wound to provide important issues including complete wound cover, protect the wound, immobilize, compress the wounded area evenly, and absorb any secretion. The wound might need to be checked frequently to avoid infections.



- Infection of the closed wound clinically evidenced after few days with pain, redness, swelling, tenderness, and hotness with or without discharge. Such wound should be opened with the evacuation of any collection, lavage, debridement, and left open with daily dressing.

11

12

- The sutures or clips should be removed under sterile condition after they have done the job for which they placed.

Acute wounds cont.

Compartment syndrome

1. This occurs due to increased pressure in the muscles compartment after trauma. It occurs most commonly after closed trauma (crushing injury).
2. The typical clinical feature is severe pain in the affected compartment particularly with passive muscles movement, distal sensory deficit, and the late sign is absent distal pulses.
3. A catheter can be inserted in the muscles compartment and using a pressure monitor to measure the intra-compartmental pressure.
   Presence of clinical features with persistent pressure of 30 mmHg or higher is an indication for fasciotomy.

fasciotomy

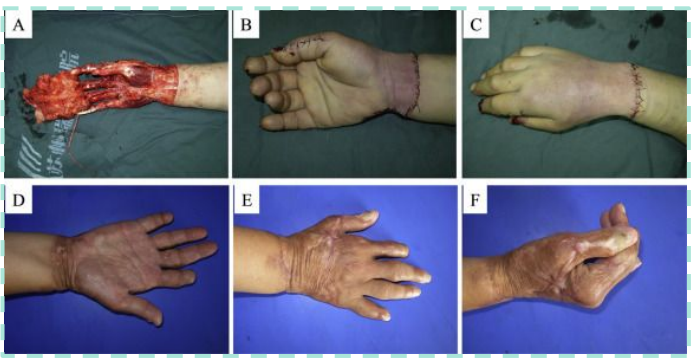
Fasciotomy is performed by skin incisions in specific locations depending on the area involved that includes skin, fat and fascia to decompress the muscle compartments and relieve the pressure.

Late fasciotomy in the presence of dead muscles may lead to a sudden release of a large amount of myoglobin to the bloodstream which may cause myoglobinuria, blockage of glomeruli, and precipitate acute renal failure.
Therefore, severely traumatized limb with extensive dead tissues better to be amputated.

Degloving injury

- This occurs when the skin and subcutaneous tissue are avulsed from the underlying fascia with exposed neurovascular structures, bones, and tendons.
- It can be an open or closed injury.

- The treatment includes antibiotics prophylaxis, saline irrigation, removal of foreign bodies, resection of nonviable skin and subcutaneous tissue with adequate debridement, followed by skin flap or grafting.
- The trial of replantation or revascularization is extremely challenging.



Chronic wounds

01

- Chronic wounds develop when the normal wound healing process fails to repair the tissue injury.

02

- Debridement, cleaning, daily dressing changes, and negative pressure devices have been the main method to enhance the healing process in chronic wounds.

03

- Several local and systemic factors are well known to retard the healing process, and results in a chronic wound.
- Judicious intervention is essential in countering these factors to enhance the normal wound healing. E.g, debridement can change the wound to an acute state or condition that will accelerate normal healing.

Leg ulcer

- An ulcer is a break in the epithelial continuity.
- The persistent inflammatory phase of wound healing results in overgrowth of the granulation tissues and attempt of wound healing by scarring with fibrotic ulcer margin.
- Necrotic tissue is in the ulcer floor is called “Eschar” whereas yellowish soft material are called “Slough”.

- Etiologies of leg ulcers:

Venous ulcer

The usual site is above the medial malleolus. It results from venous hypertension due to varicose veins or deep vein thrombosis.

Ischemic ulcer

This is due to arterial insufficiency or vasculitis. it occurs commonly at the tip of the toes or in the shin area of the leg.blood supply insufficiency is obvious, and the ulcer is dry with minimal granulation tissue.

Traumatic ulcer

This is due to repeated trauma and can be self-inflicted.

Infective ulcer

This is due to Tuberculosis or Syphilis

Neoplastic ulcer

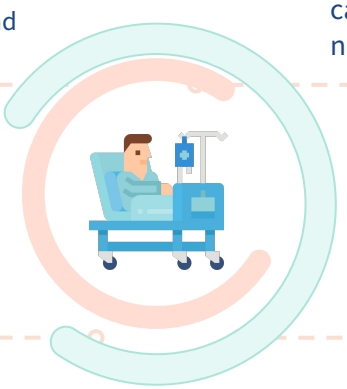
- It could be squamous or basal cell carcinoma or sarcoma.
- Biopsy should be obtained from any chronic ulcer which is persistent and unresponsive to appropriate wound care to rule out malignant changes (squamous cell carcinoma called “Marjolin’s ulcer”).
- It usually affects the area with chronic or prolonged scar (chronic burn), and it has poor prognosis.
- Treatment of chronic leg ulcer includes treatment of underlying cause, appropriate wound care, but skin graft is required in some cases.

Chronic wounds

Pressure ulcer

01
- It is defined as tissue necrosis and ulceration as a result of prolonged pressure.
- Other terms are pressure sore, bed sore, and decubitus ulcer.

02
- It occurs most commonly in bedridden, praplegic or severely ill patients in the intensive care unit.



03
- When the pressure on the skin exceeds the capillary pressure, it causes tissue hypoxia, necrosis, and ulceration.

04
- The most commonly affected sites in the body are sacrum, greater trochanter, heel, ischium, occiput, and lateral malleolus.

Prevention:

1. Good skin care and avoid shearing.
2. Special pressure relief mattresses or dispersion cushions (e.g air-fluidized beds) to minimize pressure.
3. Urinary or fecal diversion in some cases.
4. The bedridden patient should be turned at least every 2 hours.

Staging of pressure ulcers

Extra

- **Stage1.** Intact dermis, no open wounds. The skin may painful, but appears reddened and does not blanch and feel either softer or firmer than the skin area around it.
- **Stage2.** Partial thickness skin loss, with breaks, forms an ulcer which is tender and painful.
- **Stage3**. Full thickness skin loss and the wound extends into the tissues beneath the skin, but not through the underlying fascia.
- **Stage4.** Full thickness skin loss, with very deep pressure sore, reaching into muscle and bone and causing extensive damage to deeper tissues, such as tendons, and joints.

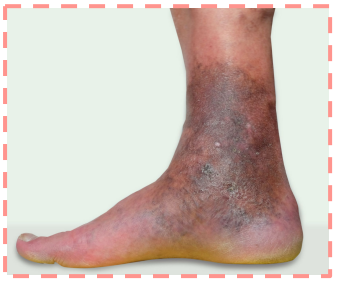
Treatment:

- Nutrition optimization.
- Antibiotics therapy when there is evidence of infection.
- Adequate debridement and excision of devitalized tissues and sloughs.
- Drainage of any collection.
- Negative pressure vacuum-assisted closure (VAC) may enhance the closure of the ulcer (porous sponge packed into the wound connected to negative pressure). The negative pressure will stimulate wound closure, promote drainage, and provide a moist wound environment which is favorable for the growth of granulation tissue.
- Fecal diversion stoma is indicated when the ulcer is recurrently contaminated by fecal materials.
- Large skin flap with intact blood supply and sensation is the best cover when the ulcer is clean and ready for grafting.

Chronic wounds

Venous ulcer

- This is due to venous valvular incompetence of the lower limb, resulting in venous insufficiency.
- the patient usually develops discomfort in the leg ,heaviness, and edema.
- Ulceration may develop in the anteromedial aspect of the leg “gaiter zone”, ulcers are generally superficial.



Pathophysiology:

01 Deep and and superficial venous valve incompetence.

02 venous hypertension

03 The pressure is transmitted to the skin resulting in deposition of plasma proteins and red cells in the skin,

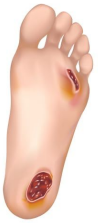
04 Edema and hyperpigmentation of the skin “dermatofibrosis”.

Treatment:

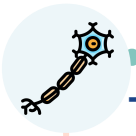
1. Debridement of the ulcer with excision if all dead tissues.
2. Sterile moist dressing.
3. Weekly or biweekly application of compression with paste bandages or multiple layer dry bandages.
4. Antibiotic therapy when infection is present.
5. Teaching the patient to elevate the limb most of the time.
6. Most venous ulcers will heal with compression within a few months, but some refractory ulcer may need surgery for ablation of normal venous perforators and skin grafting of the ulcer.

Chronic wounds

Diabetic ulcer



Diabetic patients are at risk of skin ulcer for the following reasons:



Neuropathy

Diabetic patient develop polyneuropathy of the nerves supplying the lower extremities.

- ➔ Motor neuropathy results in atrophy of the small intrinsic muscles of the foot with derangement of the bony structures with the collapse if the midfoot and plantar subluxation (Charcot's foot). This will result in excessive weight bearing in area at the risk of ulceration.
- ➔ Sensory neuropathy will result in loss of sensation and protective reflexes.

Hyperglycemia

Uncontrolled blood sugar will impair the leukocytes response to infection, create good media for bacterial growth, and retard the wound healing.

Arterial disease

Deficiency of blood supply will cause ischemic ulceration. Poor oxygen and nutrients delivery will retard the wound healing.

The commonest sites of dibetic ulcers are found at the plantar surface of the metatarsal, heels and dorsum of foot but it can occur anywhere in the foot or leg. It can be superficial, clean ulcer or even deep penetrating the deep structures with sloughs, purulent discharge with a limb or even life-threatening sepsis.

Treatment:

01 Tight control of blood sugar.

02 Antibiotics therapy covering aerobic and anaerobic bacteria.

03 Plain x-ray to look for osteomyelitis.

04 Adequate debridement, drainage of any collection, excision of dead tissues.

05 Daily sterile dressing.

06 The negative pressure vacuum-assisted closure may enhance the wound healing and closure when the wound is clean and granulating.

07 Minor amputation is required for dead or severely infected toes.

08 Major amputation is required for limb-threatening or life-threatening infected diabetic foot or in the presence of extensive osteomyelitis.

09 Arterial surgery or endovascular intervention may be required when the ischemia is a prominent factor.

Scar formation

- Scars form in the maturation phase of wound healing, and over a year or more the immature scar becomes a mature scar.
- The collagen matures and becomes aligned along the stress lines of the skin and and their strength increased.
- Although the tensile strength of the wound continues to increase, it would not exceed 80% of the normal skin.

Abnormal scar formation

How can it prevented?

- If acute wound managed correctly, most of the abnormalities in scar formation will be prevented.
- Proper scrubbing and cleansing of the wound with the proper alignment of all deep structures and good apposition of the wound edges are important in preventing the formation of an abnormal scar.

Immature scar	VS	Mature scar
• Pink • Hard • Raised • Itchy		• Paler • Soft • Flattened • Less itchy

- Suture marks can be prevented by the use of fine (5-6/0) monofilaments suture under less tension and can be removed as early as 3-5 days in certain area and replaced by sticky strips, but subcuticular suture will result in a much better cosmetic scar
- When the size of suture increases, it leaves obvious marks with the less cosmetic outcome.

Types of abnormal scars

Atrophic scar

- This is a flat, pale and stretched in appearance.
- It can be traumatized easily as the epidermis and dermis are thinned.
- Excision and resuturing may improve such scar.

Contracture scar

- This is when the scar form across a joint or flexion or extension skin creases.
- It may cause restriction of the movement at the joint , flexion or extension deformity.
- Contracture can be prevented be postoperative splints and intensive physiotherapy.
- Treatment include multiple Z-plasty or skin graft or flap.

Scar formation

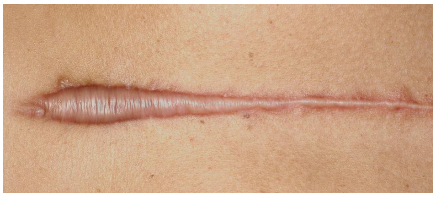
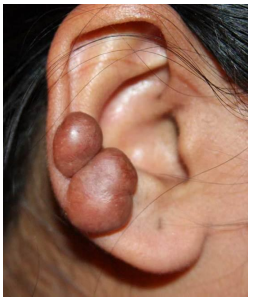
Types of abnormal scars cont.

Hypertrophic scar

- This is an excessive scar tissue that does not extend beyond the original wound boundaries.
- It results from the prolongation of the inflammatory phase of wound healing and from inappropriate scar sitting such as wound across the lines of skin tension.

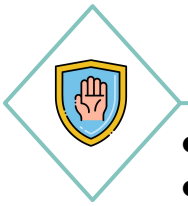
Keloid scar

- Overproduction of collagen and fibroblasts
- This is an excessive scar tissue that extends beyond the original wound boundaries.
- The etiology of keloid scar is unknown, but it is associated with deeply pigmented skin and inherited tendency.
- It is more common in the body triangle between the point of each shoulder tip, xiphisternum, ear, and chin of the tibia.
- Hypertrophic scars improve with time spontaneously, while keloid scars do not. All excisions have high rates of recurrence.

Features	Hypertrophic scar	Keloid scar
Genetic	Low familial incidence	Significant familial incidence
Race	Low racial incidence	Dark > fair skin
Sex	Female = Male	Female > Male
Age	Any age	Most common 10-30 years
Borders **Diagnostic feature**	Remains within wound borders	Outgrows wound area
Natural history	Appears soon after surgery (less than 2 years) and subsides with time	Appears months after surgery and rarely subsides
Site	Flexor surfaces	Mostly in earlobes, anterior chest , deltoid and shin of the tibia
Aetiology	Related to tension at closure	Possible autoimmune phenomenon
		

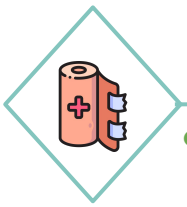
Scar formation

Treatment of hypertrophic and keloid scars:
Hypertrophic scars are treated like keloids with better outcome



Prevention by avoiding

- Unnecessary surgeries
- High risk patients or sites
  People who have multiple piercings
- Closure under tension
- Crossing joint lines when performing surgery
- Rough tissue handling
- Infection and hematoma



Therapeutic

- Excision then a combination of the following
  - Pressure garments
  - Silicone sheets for symptoms like itching
  - Steroid injections (triamcinolone injection a2 macroglobulin inhibitor)
  - Radiation
  - Revision mandatory
- Success rate of triple therapy (surgical revision,radiation and steroids)can exceed 95%

Click here for more on surgical revision

Doctor's questions

- What is the cell that peaks in wound healing at 3 weeks
- What's the main cell in the inflammatory phase
- Medications
- Clinical case will be given during OSCE about keloid and hypertrophic scars
- When to do primary/secondary/tertiary closure

Burn

Pathophysiology of burn:

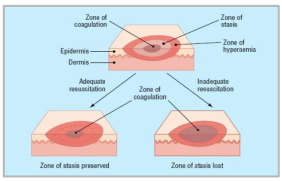
01 The local effects are the result of tissue destruction and inflammatory response.

02 The inflammatory response to injury causes capillary dilation (manifested as erythema) in mild cases, or if there is capillary damage, that leads to protein leakage and edema.

03 Insensible fluid loss can cause severe hypovolemia which might progress to hypovolemic shock (when > 15% of the body surface area is burned).

04 Destruction of the Epidermis causes impairment of the physical barriers and predispose to infections which can delay healing and increase energy demand.

★ 05 Burns have 3 zones : 1-Zone of coagulations 2-Zone of stasis (the area of potential reversible cell damage) 3-Zone of hyperemia.



Calculations:

01 02 03

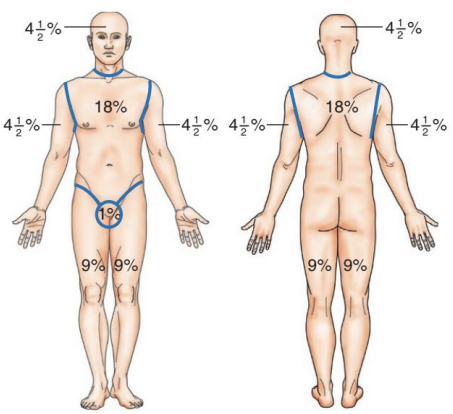
Rule of nines ★

- **Used to approximate burn size.**
- **Divides the body into areas that each represent approximately 9% of the total body surface area (TBSA) of an adult**
- **less useful in children because of the relatively large head size (and the relatively small limbs.**

★ **Palm without fingers = 1%**
★ **Head in kids = 18% (9% in adults)**
★ **Single leg in kids = 9% (18% in adults)**

Mortality

Mortality =
(body surface area % + age)/100



Parkland formula ★

- **Fluid volume of crystalloid administration during the first 24 hours of admission**
- **Volume of lactated Ringer's = 4 mL × weight (kg) × TBSA%**
- **50% given in the first 8 hours**
- **50% given in the next 16 hours**
- **Crystalloid is preferred because of the tendency to hyponatremia after the injury**
- Parkland's Formula only for more than 20 TBSA
- Not used in the case of first degree burns

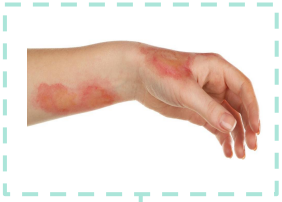
Classification of Burns Based on the Depth of Skin Injury[1] (main clinical classification):

Note for OSCE: in the exam, they will bring a picture and ask you to estimate the degree of burn.

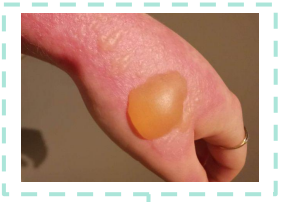
	Degree	Treatment

First Degree (superficial burn)	• Epidermal injury only.
	• Clinically characterized by: **edema** and **erythema**. No fluid collection occur at this degree.
	• Most common presentation is kitchen burns and sunburns.
		• Symptomatic treatment:
		  ○ Mild analgesics/NSAIDs pain is the commonest presentation
		• Local wound care:
		  ○ Daily cleansing.
		  ○ Topical **antibiotics** (**silver sulfadiazine**)[2] if needed.
		• Elevation
		• Occupational therapy
		  ○ Splints in functional position.
		  ○ Early range of motion.[3]

Second Degree (superficial/deep partial thickness)	• Injury to epidermal +/- dermal[4] layers. • Clinically characterized by: painful **blisters**[4] (Hallmark of Second degree burns). • Skin is repopulated by viable germinal cells in follicles.[5]	• Similar to first degree burns. • Leave blisters intact they are the best natural dressing . If debrided, cover with an occlusive dressing. • Compression garment after wound epithelialization.
Third Degree[6] (full thickness)	• Entire dermal layer and subdermal fat injury the entire content of the skin+nerve endings are completely burnt here. • Clinically characterized by: **dry, inelastic and waxy appearing scar** skin is similar to commercial leather.	• Early tangential skin excision and meshed split thickness skin grafting (within 7 days). • We have to surgically debride and skin graft. We remove the eschar (the white patch) we clean the wound until we reach healthy tissue then we remove a skin patch from one area of the body and transplant it to the burned area.
Fourth Degree (full thickness)	• Dermis and deep tissue injury. • Clinically characterized by: **injury to all skin layers**, and **injury to tendon, nerve, bone and joint** in addition to Muscles and subcutaneous fat. • Caused when the patient is unable to move away from the burning agent eg: unconscious patients/disabled patients/infants.	• Skin grafts not adequate for exposed deep structures. Treatment options: • Amputation. • Flap coverage with salvage procedures. Both grafts and flaps will be further explained in the following slides

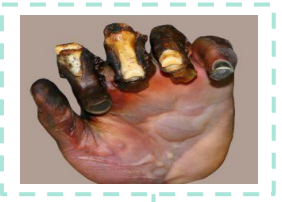


First degree burn



Second degree burn

Third degree burn



Fourth degree burn

1- In clinical practice, most burns are a mix of types. Any burn is surrounded by lighter zones eg: 2nd degree burns are surrounded by 1st degree burns and 3rd degree burns are surrounded by an engorge erythematous area (2nd degree burns). First site to come in contact with the burning agent is the deepest site of the burn. Retainment of sensations at the site of the burn suggest more superficial injury.
2-AKA: Flamazine is the **gold standard** in preventing infections due to its broad spectrum activity.
3-Start early physical therapy if burns and edema are near a joint.
4-Blistering occur when the burn exceed the basement membrane of the epidermis going down to the dermis.
5-In partial thickness burns the epithelium lining the skin appendages is preserved and heals the wound by creating new epithelium in a process called (**epithelization**), unlike full thickness burn where there's no remaining appendages to heal the skin eg: Deep 2nd degree burn > "the burn has exceeded reticular layer of the dermis" leading to delayed healing and scarring and surgical intervention is indicated.
6-Whitish in color; Skin has 5 vascular plexuses, the most important are the Sub-dermal and Sub-epidermal plexuses, these Plexuses provide blood to the skin (give the reddish appearance of the skin).In 3rd ° burn These plexus are gone = no blood supply = skin looks white.
white skin = no skin content to re-Heal = Surgery is required.

Management of Burns:

Surgical

- Escharotomy
- Skin grafting

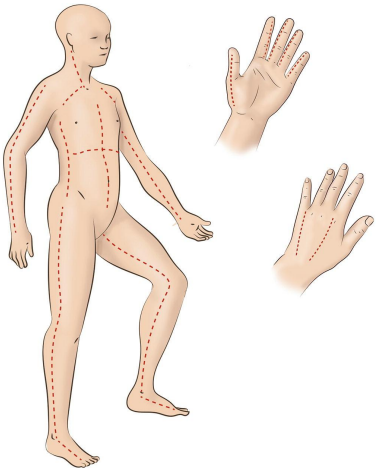
Non surgical

- Tetanus prophylaxis
- Analgesia
- Dressing
- Nutrition
- Fluid
- Foley Catheter

01

Escharotomy[1]:

- Indication:
  - Poor tissue perfusion.
  - Threat to perfusion after volume resuscitation.
  - ★ Deep Circumferential burns.
- Use mid-axial incisions.



Digital Escharotomy:

- Use mid-axial incisions:
  - Index, long → ulnar incision.
  - Ring → radial or ulnar incision.
  - Little → radial incision.
- Leave wounds open
- Consider carpal tunnel release.
- Consider intrinsic muscle release.

Skin Grafting & Flap:

- Skin grafting is a surgical procedure that involves removing skin from one area of the body and moving it, or transplanting it, to a different area of the body.
- Grafts are similar to first degree burns (First layer is removed and appendages are preserved in both of them so healing and epithelialization is possible) the difference is that grafts occur in a controlled environment unlike burns.
- The main differences between grafts and flap:
  - Grafts do not contain blood vessels but flaps do
  - Grafts are thin sheet of skin and is used more with 3rd degree. Flap is a bulky tissue (e.g. muscle flap, subcutaneous tissue flap) used when there's a deep burn (4th degree burns) or when a deep reconstruction following cancer ablation is needed.

★ 1- The most important indication of escharotomy is circumferential burn (which causes hypoperfusion due to edema).
Circumferential burns: are seen in cases where a full thickness burn affects the entire circumference of a digit, extremity, or even the torso, this is called a circumferential burn. as oedema forms the inelastic eschar can cause a buildup of pressure and act like a tourniquet (impairs blood flow). This pressure can lead to significant complications such as respiratory compromisation and loss of tissue perfusion requiring a surgical procedure known as an 'escharotomy'. An escharotomy is performed by making an incision through the eschar to release the pressure.

02

Antibiotics:

- Intravenous or oral antibiotics should cover skin flora for initial treatment.
- Topical antibiotics (silver sulfadiazine) for prevention of infection.
- Topical application of (mafenide acetate) penetrates through eschar and may be effective against a wider variety of organisms.

03

Non-Surgical Management:

- The first Priority would be **maintaining an adequate airway** and first Aid (ABCDE) especially in case of risk of inhalation injury, with continuous observation for signs of respiratory failure.
- **IV fluid resuscitation** if > 15% of BSA is affected (Parkland formula).
- **Analgesic** (eg: opioids).
- placing the patient in a warm room (to reduce energy expenditure) and **enteral feeding using nasogastric tube** with **vitamin** supplements and **iron** (Better to eat normally after 48 hours).
- **Prophylaxis against Tetanus** (eg: Clostridium Tetani) by Tetanus Immunoglobulins (TIG)
- Foley catheter to monitor urine output
- **Dressings**, essential to protect from contamination and for promotion of healing
  Types:
  - Evaporative dressings: eg: paraffin, gauze
  - Semi occlusive and occlusive: eg: hydrogel, hydrocolloid
- **Topical antibiotics**, like Silver Sulfadiazine (Flamazine) and Povidone Iodine (Betadine) (Not advised in the first 48 hours as they can make the determination of the depth more difficult).
- special Cases
  - preexisting Renal disease or Impaired renal function → diuretics.
  - Only in positive blood culture and septicemia → Systemic antibiotics.
  - Proton pump inhibitors (PPI) eg: Omeprazole → prophylaxis for curling's ulcer.

When to transfer to a burn center (transfer criteria): ★

- >25% body surface area (BSA).
- >20% BSA in children.
- High voltage burns.
- Inhalation injuries.
- Burns in the genital area, face, neck, feet and hands in addition to full thickness burns.
- Chemical burns.

Burn Center Referral Criteria

1. Partial-thickness and full-thickness burns totaling greater than 10% TBSA in patients under 10 or over 50 years of age.
2. Partial-thickness and full-thickness burns totaling greater than 20% TBSA in other age groups.
3. Partial-thickness and full-thickness burns involving the face, hands, feet, genitalia, perineum, or major joints.
4. Full-thickness burns greater than 5% TBSA in any age group.
5. Electrical burns, including lightning injury.
6. Chemical burns.
7. Burn injury in patients with preexisting medical disorders that could complicate management, prolong the recovery period, or affect mortality.
9. Any burn with concomitant trauma (e.g., fractures) in which the burn injury poses the greatest risk of morbidity or mortality. If the trauma poses the greater immediate risk, the patient may be treated initially in a trauma center until stable, before being transferred to a burn center. The physician's decisions should be made with the regional medical control plan and triage protocols in mind.
10. Burn injury in children admitted to a hospital without qualified personnel or equipment for pediatric care.
11. Burn injury in patients requiring special social, emotional, and/or long-term rehabilitative support, including cases involving suspected child abuse.

Inhalation injury

- The mechanisms of inhalation injury can be divided into three broad areas
  - Inhalation of products of combustion
  - Carbon monoxide inhalation
  - Direct thermal injury to the upper aero-digestive tract (inhaling hot air)

- Features of inhalation injury (physical/mechanical components)
  - Flame burn in a closed space
  - Singed (burned) nasal hair
  - Facial or oropharyngeal burns
  - Expectoration of carbonaceous (blackish) sputum
  - Loss of facial hair (eyebrows,mustache..etc)
  - On intraoral examination: burns on the soft and hard palate
  - Does Not occur with burns caused by hot water

- Acutely the patient is fine, the problems begins when the patient starts receiving fluids.

This fluid will go to third spacing (interstitial edema) and compromises the airway.
Patient will start developing signs of upper respiratory obstruction—such as crowing, dyspnoea, cough, stridor, or air hunger.

- Intubating in this stage is semi-impossible due to swelling of the vocal cords and trachea, tracheostomy or cricothyroidotomy which is not always feasible, that's why you should intubate the patient as soon possible until this phase passes. (don't miss inhalation component)

Carbon monoxide chemical component of inhalation injuries

Why is it bad?

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

Signs

- Headache
- cherry red lips
- arrhythmias
- acidosis
- seizures
- LOC

CO level	Symptoms
0-5	Normal Value Normal people don't have any carbon monoxide
15-20	Headache, Confusion
20-40	Disorientation, Fatigue, Nausea< Visual Changes
40-60	Hallucinations, Coma< Shock
60 or above	Mortality >50%

Not done routinely, you have to call the lab and ask for it with the regular Arterial Blood Gas analysis (ABG)

Treatment:

- 100 % O2 +/- intubation and ventilation
- t1/2 of CO is 5 to 6 hours on R/A
- 45 min on 100% O2
- 27 min HBO @ 3 atm

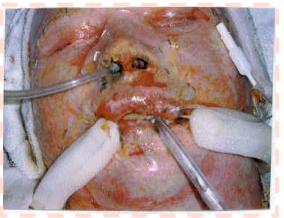
Indications for hyperbaric oxygen therapy HBO

- Coma
- LOC
- ischemic ECG changes
- focal neurological deficits
- +/- CO Hb >40%

Questions /cases from the doctors

1

A 29 years old male, sustained a burn 1 hour ago, cam to the ER with these features.
What will be your main concern?
Inhalation injury
What are the features of this injury?

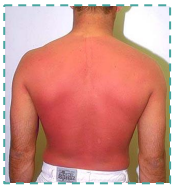


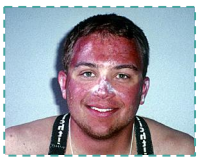
<u>Calculation of inhalation burns</u>
Add 25% to TBSA
In this case the patient is suffering burns on his head,neck and right arm =18%
18+25= 43%
If we only calculate the external TBSA the patient will be under resuscitated.

Most common cause of morbidity and mortality in burn patients acutely is
under resuscitation
Most common cause of morbidity and mortality in burn patients a week after the injury is infection

2

What is the degree of the burn?
First degree
What is diagnostic feature?
Erythema
Only requires analgesia because it only affects the epidermis

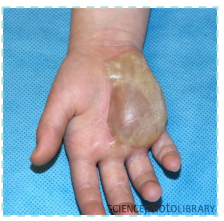




3

What is the degree of the burn?
Second degree
Why?
Presence of blisters, there is loss of fluid underneath the skin and the burn extends to the dermis
Second degree burns are divided into

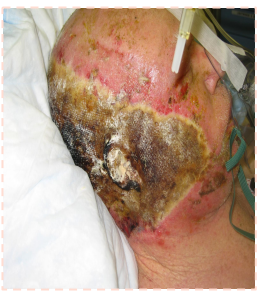
- Superficial partial thickness (upper layer of the dermis)
- Deep partial thickness (middle layer of the dermis) facial burns heal on their on because the face has a big blood supply

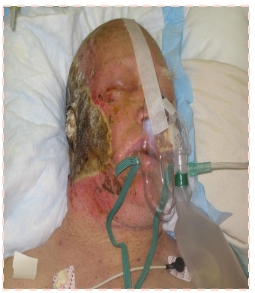


4

What is the degree?
Third degree burns involve the whole dermis
What is the diagnostic feature ?
The presence of an eschar

Fourth degree burns are rare and require surgery and skin grafts





Questions /cases from the doctors

5

A patient presents to the ER suffering from a cut wound in the right hand, the wound has been 2 hours and is superficial , how would you approach this pt ?

- Hx & Px
- Ask about Tetanus Vaccine
- Do you need to give ABx ? if the wound dirty and old > YES
- Start with oral ABx > after 48h , progression of clinical infection ? shift to IV

6

Patient sustained a burn at 4 o'clock, presented to ER at 8 , when would you give the first half amount ?
You estimate time from the time of injury NOT admission

7

History:
CC: Difficulty breathing; Machine (in wheat house--closed area) exploded in face

Comments:
This patient most likely have inhalation injury, so ask yourself:

- Can the patient see?
- Can the patient hear?

Since its a burn bare in mind the following:

- Vaccination and degree of pain
- Was the patient wearing clothes or not?
- If so, did the clothes burn or not?
- Covered after removal of clothes?—to avoid dehydration and hypothermia. —in case of continuous contact
- Assess breathing, assess mucosal lining,
- Transfers to burn unit (more than 30% and inhalation injury)
- The burn will demarcate after 3-5 days

Management:

- Maintain Airway, breathing and circulation (make sure patient is stable)
- Make sure patient is pain free and the wound is covered
- if chemical then irrigate with NS or water
- Decide on surgery after 3-5 days
- If circumstantial burn → escharotomy
- If compartment → fasciotomy

Questions /cases from the doctors

8

History

Personal: Khalid , 65 yo from SA, retired policemen - no allergies

CC: Came to ER

- large wound with **pain** in right upper buttocks and **scarring** X 2 years ago
- Wound has discharge now (note: Painful wound on Right lower buttocks for two years. With discharge so ask about: color, smell, how many times do you change dressing (continuous or not), amount)
- Assos.: fever, loss of weight, redness around wound

PMx:

- Admitted 5 times to hospital 5/7 past years for this issue, improved with AxB
- Paraplegic for the last 10 years due to RTA
- Diabetic since long time and takes insulin and glucophage
- HTN, dyslipidemia
- Recurrent UTI (note:can't control bowel and urine output so ask: is there a need for a catheter? Colostomy?)

PSx:

- Peripheral vascular disease with clopidogrel (Plavix) for stent

Sx:

- Smokes 10 cigs for 20 years

Clinical exam:

(inspect: site, size, depth of floor, granulation tissue of unhealthy tissue

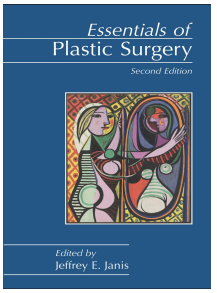
Palpate: Discharge (pus, yellow, green, blood) and (continuous-then its a sinus continuously draining), Around the wound (raised edges, laceration (with an M), muscle wasting, frail skin, redness), Pulses (Femoral, popliteal, dorsalis pedis) incase its vascular or hematoma, Percuss (around for abscess)**)**

- Site: On the right ischium
- Single wound
- Measure size
- Irregular edges
- With areas of necrosis
- Redness in surrounding skin
- The edges are darker
- Purulent discharge
- Measure depth of wound
- Assess base
- Palpate for tenderness and
- Asses temperature of surrounds area and floor
- Check for disagree or bleeding
- Percuss around loculated abscess

- Random comment: Usually the cause of sepsis is urosepsis

Quiz

The doctor recommended this book (click on it)



MCQ

Q1:A 22-year-old healthy African-American woman presents with a recurrent growth on her right thigh.She has a childhood history of a third-degree scald burn to the same area that did not require skin grafting.The growth was completely removed 2 years ago. On physical examination there is a 5 cm× 2 cm, raised,irregularly shaped purple lesion with a smooth top. Which of the following is the most likely diagnosis?

A. Angiosarcoma
B. Malignant melanoma
C. Squamous cell carcinoma
D. Kaposi sarcoma
E. Keloid

Q2:A 65-year-old man sustains a 50% TBSA burn while burning trash in the backyard. The patient is resuscitated with lactated Ringer (LR) solution using the Parkland formula and a weight of 80 kg. What is the rate of LR given in the first 8 hours?

A. 100 mL/h
B. 500 mL/h
C. 1000 mL/h
D. 5000 mL/h
E. 10,000 mL/h

Q3:What reverses the deleterious effects of steroids on wound healing?

Vitamin A

Q4:What are the signs of smoke inhalation?

Smoke and soot in sputum/mouth/nose, nasal/facial hair burns, throat/mouth erythema, history of loss of consciousness/explosion/fire in small enclosed area, dyspnea, low O2 saturation, confusion, headache, coma

Q5:What lab value assesses smoke inhalation?

Carboxyhemoglobin level (carboxyhemoglobin level >60% is associated with a 50% mortality); treat with 100% O2 and time

Q6: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

Answers
Click here for explanation

Q1	E	Q4	
Q2	C	Q5	
Q3		Q6	

Extra Questions

Good Luck!

Team leaders:

Nouf Alshammari

Haneen Somily

Naif Alsulais

Mohammed Alshoieer

This lecture was done by:

- Haneen Somily
- Nouf Alshammari
- Fawaz Alotaibi
- Mohammed Alhamad
- Nujud Alabdullatif
- Lama Alassiri
- Razan Alrabah
- Ibrahim Aldakhil

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Note taker Reviewer

Feedback