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Burns

Etiology (Types of Burns)

There are many types of burns depending on the etiology.

1. **Thermal Burns:**

Heat causes **coagulative necrosis** of tissue, by coagulation of the cellular proteins, this type of necrosis is characterized by, preservation of the shape of the tissue involved, the temperature that causes burn is greater than 45 degrees. The depth **(degree of burn)** depends on the quantity of heat (temperature and duration of exposure), so exposure to a relatively lower temperature for long period may cause more damage than exposure to high temperature for a short period.

Thermal burns are classified into,

a. Dry heat (direct flame burn) direct exposure to fire.
b. Moist heat (scald burn), exposure to hot liquids,
c. Contact burn. contact with hot metals.
d. Friction burns.

The burn wound, and surrounding tissues classically have been described as a system of several circumferential zones radiating from primarily burned tissues, as follows:

1. **Zone of coagulation** - A nonviable area of tissue at the "epicenter" of the burn
2. **Zone of ischemia or stasis (Injury zone):** Surrounding tissues (both deep and peripheral to the coagulated necrotic areas), which are not devitalized initially but, due to microvascular insult, can progress irreversibly to necrosis over several days if not resuscitated properly
3. **Zone of hyperemia** - Peripheral tissues that undergo vasodilatory changes due to neighboring inflammatory mediator release but are not injured thermally and remain viable.

The primary aim of management should be to preserve the tissues in <u>ischemic areas</u> (zone of injury) by:

1. **Proper fluid resuscitation to maintain adequate perfusion and proper tissue oxygenation in the initial stages.**
2. **Minimizing tissue edema which has a negative effect on micro-circulation and decreases tissue perfusion, this is achieved by <u>proper</u> fluid resuscitation (avoid over-resuscitation) and by elevation of injured limbs.**
3. **Proper burn wound management later**.

   If the above measures are not performed properly, more necrosis will follow, and the degree of burn would increase, and second-degree burn may become third.

2. <u>Chemical Burns:</u>

Caused by acids or alkalis: characterized by deeper penetration and damage to the tissues, due to the longer duration of action of the chemical agent which continues to damage until it is inactivated by reaction with the tissues.
Contrary to expectations, acids produce less damage, and less penetration, than alkalis, because acids usually produce **coagulative necrosis** (denaturation of cellular proteins) this forms a barrier limiting the destructive effect of acids on tissues, while alkalis produce **liquefactive necrosis**, allowing deeper penetration and destruction.
The primary management of chemical burns is by irrigation of the area affected by water to dilute the chemical agent, this should continue for 2-4 hours in case of alkaline burn, and 30 minutes for burns caused by acids.

3. <u>Electrical Burns:</u>

- The severity of burn depends on the voltage (high voltage is more than 1000 volts), so it is more serious with high voltage current.
- The passage of electric current through different tissues within the body, and hence the burn damaging effect, is inversely related to the **tissue resistance** which varies among different tissues. Nerves, muscles, blood, and blood vessels have low resistance, so they are affected most, while skin, and tendons, have high resistance, hence, they are less burned. Although nervous tissue is the most sensitive to electric injury, the major effect of electric burn involves the muscles due to their bulk, so we can say that electric burn is muscle burn! **It is very important to remember this fact, because a massively electrically burned patient may deceive the casualty officer who finds minimal skin burn while the patient suffers massive hidden muscle burn. ELECTRIC BURN IS DECEIVING!!!**

In managing electrical burns, the following should be noticed:

1. Patients have **cardiac arrythmias,** so they should be monitored for cardiac arrythmias.
2. Due to the muscle damage, myoglobin is released from the damaged muscles leading to myoglobinemia and myoglobinuria that caused **acute renal failure**. good hydration, and alkalization of urine are measures to be used to prevent this renal impairment.
3. Also, due to the muscle damage, patients are liable for **compartment syndrome**, so limb vascularity should be observed, and fasciotomy considered.
4. due to severe muscle contraction patients may have **bone fractures.**
4.The severity of the electrical burn is not evident, and cannot be estimated, as in the case of thermal burn, which depends on the percentage of the burned skin, **so fluid management could not be based on a calculated Parkland's formula as in thermal injury,** but on close clinical observation, urine output, serial hematocrit values, and CVP readings.

Assessment of the Severity of the Burn (Depth and Percentage)

1. **The depth of burn damage (degree):** determines the **local management and outcome of** the burn wound.

2. **The surface area involved in burn**, this is the percentage of the burned area to the total body surface area. **This determines the prognosis (mortality rate) and the systemic management and complications,** initially fluid resuscitation depends on the percentage of burn injury, later the percentage of burn determines the systemic complications as sepsis, catabolism and decreased immunity.

Degree of the Burn

This determines the local management of the burn wounds, in partial thickness burn, part of the dermis containing skin appendages is preserved, from these epithelial elements, the burn wound would heal by **REGENERATION**, within weeks, hence the local treatment of the burn is **conservative (no skin grafts)**. While in full thickness burn all the dermis with the epithelial elements are lost, so the burn wound would naturally heal by **FIBROSIS**, a process usually takes longer period, and leaves an unstable scar, with all its **functional**, and **cosmetic** complications, to avoid this unfavorable fate, full thickness burns, should be treated by **skin grafting**, better sooner than later. According to the previous discussion, the deeper the burn is, the more the scarring would be, and the more time is taken to heal.

Classification of the depth of burn injury:

1. **First degree burn**, thermal necrosis is limited to the epidermis, clinically there is pain, and erythema, **it takes 1-6 days** to heal and leaves no scars.

2. **Second degree burn (partial thickness),** necrosis of the epidermis and varying depth of the dermis, characterized clinically by **pain** (due to irritation of the dermal sensory nerves), **erythema**, **blisters(bullae)**, the burned area is **wet with exudate** (weeping), **blanching** denoting intact dermal vascularity, and **preservation of skin elasticity. It takes 1-4 weeks to heal and leaves variable degrees of scarring.**

3. **Third degree burn (full thickness),** necrosis of the whole skin (epidermis and dermis) and its skin appendages, clinically there is an **eschar** which is simply -the burned necrotic skin -, it is **insensitive**, **leathery**, hard, **inelastic**, **and may show thrombosed dermal vessels. It takes months to heal and leaves significant scarring, to avoid scarring it should be skin grafted.**

Note that the deeper the burn the more dermis is necrotic so:

- **Less pain due to damage of dermal nerves.**
- **Healing is by fibrosis rather than regeneration. So, leaving more post burn contractures**
- **More loss of skin elasticity, so it compresses the limbs that needs escharotomy.**

Estimation of the Percentage of the Burn

This determines the prognosis, and the systemic management of burn, especially fluid management.

There are three methods of TBSA estimation

1. Rule of nines: the body is divided into 11 nines; Head &neck (9%), Upper limbs. (9% each), Anterior trunk (18%), Posterior trunk (18%), Lower limbs (18% each) and the remaining 1% for the genitals.
2. As the rule of nines is not very accurate especially in children, in whom the percentage of the surface area of the head (20% at birth) and decreases with age, while the percentage of the surface area of the lower limbs increases with age, it is more accurate to follow the specially made charts available in the burns units and in the emergency rooms.
3. For small burns, the palm of the hand equals 1% of the body surface area.

MANAGEMENT OF BURN

When the percentage of burn is significant, we should look at the burn injury as a systemic (disease) or syndrome affecting all the systems of the body, at some stage of the hospital stays. Regarding the respiratory system, burn victim may have upper respiratory tract obstruction, in the first 24 hours, smoke inhalation syndrome in the initial 2-3 days, and later may develop respiratory infections, or ARDS. The cardiovascular, renal, gastrointestinal, endocrine, immune, systems may be affected by burn that increases the metabolic rate, and results in negative nitrogen balance and malnutrition.

Management of burn is divided into:

1.Acute or emergency stage.
2.Local management of burn wounds.
3.Treatment of complications.

ACUTE OR EMERGENCY MANAGEMENT

Like in any trauma, we follow the ATLS (Advanced **T**rauma **L**ife **S**upport) rules, which dictates treatment of life threatening conditions in the first minutes, before full screening and diagnosis of the injuries, so we follow the, A, B, C, rules.
AIRWAY: patients involved in flame burns may suffer from upper airway obstruction, due to soft tissue edema of the oropharynx and vocal cords, resulting from direct thermal injury to the upper respiratory tract, by inhalation of flame and hot gases. This obstruction may not be evident initially but appears in the first 24 hours. Direct inspection of the oropharynx and the vocal cords, by either direct laryngoscopy, or better by bronchoscopy is indicated. Endotracheal intubation, to secure a patent airway, should be performed before obstruction is complete. Signs of impending obstruction include:
Tachycardia, progressive hoarseness, and difficulty to clear bronchial secretions.

Carbon Monoxide Poisoning

This is due to occupation of the oxygen carrying sites of hemoglobin by CO, which has 210 times higher affinity to hemoglobin than oxygen. The condition is diagnosed by estimation of carboxyhemoglobin level in the blood, the PO2 level may be normal, as this is an estimation of the oxygen dissolved in the plasma. The treatment is by administration of 100% oxygen to displace the tightly bound CO from hemoglobin.

Fluid Management of Burned Patients

Total Body Water

Water constitutes approximately 50 to 60% of total body weight, Lean tissues such as muscle and solid organs have higher water content than fat and bone. In an average young adult male 60% of total body weight is TBW, whereas in an average young adult female it is 50%. The lower percentage of TBW in females correlates with a higher percentage of adipose tissue and lower percentage of muscle mass in most the highest percentage of TBW is found in newborns, with approximately 80% of their total body weight comprised of water. This decrease to approximately 65% by 1 year of age and thereafter remains constant.

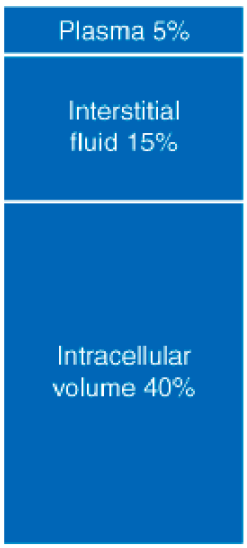
Fluid Compartments

In a male adult with weight=70kgs, 70 KgsX60%= 42 Kgs of water= 42 liters=42000 ml. This TBW is divided into three functional fluid compartments:

1. INTRA-CELLULAR (ICF) = 2/3 of TBW= 28 liters.
2. EXTRA-CELLULAR (ECF) = 1/3 of TBW= 14 liters.

A. INTRA-VASCULAR FLUID (PLASMA) = ¼ of the 1/3= 3.5 liters.

B. EXTRA-VASCULAR INTERSTITIAL= 3/4 of the 1/3= 10.5 liters.



% of Total body weight	Volume of TBW	Male (70 kg)	Female (60 kg)
Plasma 5%	Extracellular volume	14,000 mL	10,000 mL
Interstitial fluid 15%	Plasma	3500 mL	2500 mL
	Interstitial	10,500 mL	7500 mL
Intracellular volume 40%	Intracellular volume	28,000 mL	20,000 mL
		42,000 mL	30,000 mL

Source: Brunicardi FC, Andersen DK, Billiar TR, Dunn DL, Hunter JG, Matthews JB, Pollock RE: *Schwartz's Principles of Surgery, 9th Edition:* http://www.accessmedicine.com

Composition of Fluid Compartments

The main cation of the ECF compartment is **sodium**, and the main anions are **chloride and bicarbonate.** The composition of the plasma and interstitial fluid differs only slightly in ionic composition.

The main cations of the intracellular fluid (ICF) are **potassium and magnesium**, and the main anions are **phosphate and proteins**.

Although the movement of ions and proteins between the various fluid compartments is restricted, **water** is freely diffusible. Water is distributed evenly throughout all fluid compartments of the body; so that a given volume of water increases the volume of any one compartment relatively little. **Sodium**, however, is confined to the ECF compartment, and because of its osmotic and electrical properties, it remains associated with water. Therefore, sodium-containing fluids are distributed throughout the ECF and add to the volume of both the intravascular and interstitial spaces. Although the administration of sodium-containing fluids expands the intravascular volume, it also expands the interstitial space by approximately three times as much as the plasma.

Osmotic Pressure

The movement of water across a cell membrane depends primarily on osmosis. To achieve osmotic equilibrium, water moves across a semipermeable membrane to equalize the concentration on both sides. This movement is determined by the concentration of the solutes on each side of the membrane. Osmotic pressure is measured in units of osmoles (osm) or milliosmoles (mOsm) that refer to the actual number of osmotically active particles. For example, 1 mmol of sodium chloride contributes to 2 mOsm (one from sodium and one from chloride). The principal determinants of osmolality are the concentrations of sodium, glucose, and urea (blood urea nitrogen, or BUN):

$$\text{Calculated serum osmolality} = 2\,\text{sodium} + (\text{glucose}/18) + (\text{BUN}/2.8)$$

The osmolality of the intracellular and extracellular fluids is maintained between 290 and 310 mOsm in each compartment.

Because cell membranes are permeable to water, any change in osmotic pressure in one compartment is accompanied by a redistribution of water until the effective osmotic pressure between compartments is equal. For example, if the ECF concentration of sodium increases, there will be a net movement of water from the intracellular to the extracellular compartment. Conversely, if the ECF concentration of sodium decreases, water will move into the cells. Although the intracellular fluid shares in losses that involve a change in concentration or composition of the ECF, an isotonic change in volume in

either one of the compartments is not accompanied by the net movement of water if the ionic concentration remains the same. For practical clinical purposes, most significant gains and losses of body fluid are directly from the extracellular compartment.

BODY FLUID CHANGES

Normal Exchange of Fluid and Electrolytes

The healthy person consumes an average of 2000 mL of water per day.

INPUT:

SINSIBLE:

1. ORAL INTAKE (75%) = 1500 ml.
2. SOLID FOOD (25%) =500 ml.

INSINSIBLE: WATER OF OXIDATION= 250 ml.

OUTPUT;

SINSIBLE:

1. Urine: 800–1500 ml.
2. STOOL: 0–250 ml.
3. SWEAT.
4. Pathologic GIT losses.

INSINSIBLE:

Lungs and skin: 600 ml.

Classification of Body Fluid Changes

Disorders in fluid balance may be classified into three general categories: disturbances in (a) volume, (b) concentration, and (c) composition. Although each of these may occur simultaneously, each is a separate entity with unique mechanisms demanding individual correction.

Isotonic gain or loss of salt solution (solutions containing sodium) results in **extracellular volume changes**, with little impact on intracellular fluid volume. If free water is added or lost from the ECF, water will pass between the ECF and intracellular fluid until solute concentration or osmolarity is equalized between the compartments.

Unlike with sodium, the concentration of most other ions in the ECF can be altered without significant change in the total number of osmotically active particles, producing only a **compositional change**. For instance, doubling the serum potassium concentration will profoundly alter myocardial function without significantly altering volume or concentration of the fluid spaces because potassium level= 3.5-5 mEq/L in the extra-cellular fluid, so it has little effect on ECF osmolarity and volume.

Disturbances in Fluid Balance (volume)

Extracellular volume deficit is the most common fluid disorder in surgical patients.
It can be divided into either acute or chronic.
Acute (Hypovolemic shock): acute volume deficit is associated with cardiovascular and central nervous system signs.
Chronic (dehydration): chronic deficits display tissue signs of dehydration, such as a decrease in skin turgor and sunken eyes, in addition to cardiovascular and central nervous system signs
Laboratory examination: significant ECF losses may be associated with:

1. Elevated blood urea nitrogen level.
2. Urine osmolality usually will be higher than serum osmolality.
3. Urine sodium will be low, typically <20 mEq/L.

Volume changes may be associated with normal, high or low sodium concentration.

Causes of fluid losses in surgical patient:

1. The most common cause of volume deficit in surgical patients is a loss of GI fluids from nasogastric suction, vomiting, diarrhea, or enterocutaneous fistula.
2. Sequestration (third space losses) secondary to soft tissue injuries and inflammation burns, and intra-abdominal processes such as peritonitis, pancreatitis, intestinal obstruction, or prolonged surgery can also lead to massive volume deficits.
3. Hemorrhage.

Fluid Management of Burned Patients (Resuscitation)

In the last decades, the understanding of the pathophysiology of fluid derangement in burn patients, and their proper management has decreased the mortality of burn related to shock and its sequels as renal failure.
In burns, there is a major shift of fluids from the INTRAVASCULAR compartment, which is responsible for direct tissue perfusion, to the INTERSTITIAL compartment. The cause of this shift is the **increase capillary permeability**, or loss of the capillary integrity, so the capillary membrane which is normally semi-permeable sieving proteins inside the circulation to exert oncotic pressure, becomes fully permeable, so proteins will leak to the interstitium, dragging the intra-vascular water with it.
The shifted fluid called **THIRD SPACE LOSS**, causes edema in the intestitium.

Hemodynamically, the depletion of the intra-vascular compartment will cause hypovolemic shock, and its severity depends on the percentage of burn. **Practically, burn shock is seen in adults with burns greater than 15-20% and in children with burn more than 10-15%.**

The causes of loss of the capillary integrity (increased capillary permeability) in the burned tissues are:

1. Direct thermal damage to the capillaries,
2. And the released vaso-active inflammatory mediators.

However, it is important to note that edema, and third space loss are not limited to the burned areas only, the non-burned tissue is affected as well, the cause in this case is the resulting generalized hypo-proteinemia and possibly the circulating vasoactive mediators.

Fluid management in major burns is critical, the following guidelines are important in planning the fluid replacement:

1 The lost capillary membrane integrity with increased permeability, has two implications:

Firstly: most of the fluids administered, would leak into the interstitium causing more tissue edema, which is harmful to the tissue as it increases tissue hypoxia, and secondly, huge amount of fluids is needed to maintain a functional, perfusing, intra-vascular compartment

2.**Amount of fluid given should be just adequate to perfuse tissue**; over-perfusion is at the expense of edema.

3.Although there are so many formulas, to estimate the amount of fluid to resuscitate a burned patient, there is no ideal, magic one that you apply and go to sleep! This means that the amount of fluid needed vary among patients, and the only way to ensure that the optimal amount of fluid is given is by close monitoring by the following ways:

a. Clinically by observing, the general condition of the patient, and the vital signs.
b. Urine output, which is the most sensitive indicator of tissue perfusion, this should be 0.5-1 ml / kg / hour, higher urine output may indicate that, extra fluid is given, that increases tissue edema.
c. Serial PCV readings, in which, high PCV indicates, hemoconcentration, where more fluid is to be administered, and low PCV means, hemodilution, in which the rate of fluid administration is to be lowered.
d. Swan-Ganz or CVP lines may be indicated in some patients, especially those with border line cardiac reserve, like the elderly.

4. Regarding the type of fluids to be given because the capillaries are leaky initially, it is wise to give crystalloids, in the first 16-24 hours, and to give colloids thereafter.
5. All formulas are based on, burn percentage and patient weight. Parkland formula, for example states that:
   Fluid in the first 24 hours = 4 X Weight. X % of burn

   An adult weighing 70 Kg., with 50% burn, should receive:
   4x 70x 50= 14000 ml of Ringer lactate
   Half of this amount is administered in 8 hours, and the remaining half over the next 16 hours.

Escharotomy

In full thickness burns, the skin which is normally elastic, is transformed into eschar (dead or necrotic skin), with loss of elasticity, so in circumferential full-thickness burns of the limbs, the eschar would act as a tourniquet. when the burned tissues develop edema, the pressure inside the limb increases above the capillary pressure level (32 mm/ Hg) leading to tissue ischemia. The picture is similar to compartment syndrome.

Management of this condition consists of:

1. Elevation of the affected limbs.
2. Observation of the circulation (capillary filling, color, temperature) and sings of ischemia (pain, paresthesia paralysis)

   It is important to mention that: Presence of distal pulses does NOT rule out the condition

   .
3. If ischemia is suspected, **ESCHAROTOMY** is indicated. This means incising the eschar of the affected limbs and fingers, on both, the medial and lateral aspects, to release the high pressure inside the limb.
   Note that escharotomy is different from eschectomy which means excision or debridement of the eschar.
   Escharotomy is not limited to the limbs: we do eschrotomy to release pressure on the neck or chest wall to improve breathing.

Antibiotic Use in Burned Patients

Are used to treat infections, but not prophylactically

Prophylactic antibiotics are contra-indicated in burns, for the following reasons:

1. Studies did not prove that prophylactic antibiotics decrease the incidence of sepsis.
2. Antibiotics increase the incidence of fungal infections.
3. Antibiotics increase the incidence of bacterial resistance.

Analgesia and Sedation

A sort of pain and anxiety relieve, is needed in the burn victim, even in those with full thickness burn, the following guidelines are to be applied:

1.In patients with low tissue perfusion, the drugs should be administered by the intra-venous route to avoid accumulation of the drug.

2. Given in increments of small doses, till the required dose is reached.

3.Head injury, hypoxia, and shock all have the same symptomatology of pain, so these should be ruled out before treating pain.

Indications of Admission to the Hospital

1. burns that need fluid resuscitation: Adults>15%, children>10%).
2. Full-thickness burns> 2%
3. Burns of special areas: face, hands, perineum.
4. Electric and chemical burns.
5. Inhalation injury.
6. Old age and co-morbidity.
7. Suspected child abuse.

Local Management of Burn Wound

Partial Thickness Burn

In partial thickness burns, healing occurs within weeks, by regeneration of the dermal skin appendages, contained in the remaining dermis, the more superficial the burn is, the quicker the healing, and the better the result would be. So basically, the treatment in conservative, aiming at providing the optimal conditions for this regeneration to occur, this is by protecting the wound from dryness, infection, and trauma. Practically, this means keeping the burn wet, covered with local antibiotics, and changing the dressing gently as required.

Infection of partial thickness burn would damage the epithelial elements and change the burn to full thickness one.

Full Thickness Burn

Naturally, this heals by fibrosis which takes long time and leaves bad scars. To avoid this fate, these wounds should be skin grafted. The eschar (dead skin), is adherent to the underlying subcutaneous tissue. As time goes by, enzymes that separates the eschar gradually from the deeper tissues, within two to three weeks, the eschar is spontaneously separated, at this time the deep tissue is covered by granulation tissue. At this stage **split thickness skin graft** is harvested from unburned areas and applied to the burn wound.

The modern treatment of burn, evolved in the last decades, is to excise the eschar early (early **escharectomy**) and cover the burn wounds by skin graft, rather than waiting for the natural, bacterial assisted spontaneous separation of the eschar, the **Advantages of early escharectomy and grafting are:**

1. Decrease the duration of hospital stay.
2. Decrease the incidence of burn wound sepsis, by elimination of the dead tissue and bacteria.
3. Helps early mobilization of the patient, decreasing joint contractures.
4. Shortens the catabolic state, minimizing the protein breakdown, and malnutrition.
5. Better cosmetic outcome.

However, early burn wound excision is associated with the following problems:

1. The eschar is adherent to the underlying tissues, so surgical excision results in massive blood loss, and hypothermia.
2. When the burned area is large (i.e.>60 %burn), excision would leave a large exposed wound that we cannot cover by the patient's own skin.

The first problem is solved by, better blood banking, better ICU care, hypotensive anesthesia, and staged excision.

The second problem is dealt with by, temporary coverage of the excised areas, with biological dressings: These are either, allografts (homografts), taken from cadavers, or heterografts, taken from animals, they are applied as temporary dressing to the excised burn wounds.

So, after eschar excision, we cover as much as we can of the excised areas with split thickness skin graft (autografts), the remaining areas are covered temporarily with biological dressing. After around two weeks, when the donor areas heal, we take skin grafts again from the same donor areas, (re-harvesting) and apply them to new areas after taking off the biological dressing.

Soft tissue coverage

Plastic surgery is divided into:

1. **Aesthetic surgery: deals with improving the beauty of clients!**
2. **Reconstructive surgery: deals with return of lost tissues and repair of congenital and acquired defects.**

In our course we deal with basic principles of reconstructive surgery.

Soft tissues that are dealt with by the reconstructive surgeon are *skin, subcutaneous tissues, fascia and muscles*.

Wounds are defined by pathologists as: discontinuity of epithelium, this could be due to trauma or to pathological causes, in this case they are called (ulcers). They may be **partial thickness** (involving the epidermis and part of the dermis) or **full thickness** (involving the epidermis and the whole dermis). As partial thickness wounds heal usually by regeneration (as second-degree burns) they are treated conservatively.

Wounds (tissue loss or defects as called by plastic surgeons) vary in their complexity from simple to complex, the **incised skin wound** is the simplest wound. Wounds can however increase in depth and surface area to involve more soft tissues than skin. On the other extreme, is the **compound defect** is three-dimensional defect which results from major loss of many tissues as that resulting from excision of malignant tumors.

Causes of soft tissue defects that are dealt with by plastic surgeon

1. **Congenital:**
   Cleft lip and palate, hypospadias, microtia and anotia, syndactyly, vascular anomalies, and other soft tissue congenital anomalies.

2. **Acquired:**

a) Inflammatory: defects that follow soft tissue infections as abscesses, necrotizing fasciitis.
b) Neoplastic: resulting from tumors as following mastectomy and skin and soft tissue cancers.
c) Metabolic: as in diabetic foot.
d) Ischemic: following peripheral vascular diseases and pressure sores.
e) Traumatic: that follows burns, frost bites, radiation injury and soft tissue injury.
f) Iatrogenic: as in extravasation injury.

WHY DO WE NEED RECONSTRUCTIVE SURGERY?

The body can deal with Defects (tissue loss or discontinuity) by wound healing, which is simply: replacement of lost or discontinued tissue (ROLT). There are two modes of wound healing:

1. **Regeneration:** replacement of lost or discontinued tissue (ROLT) by the same lost tissue: This is the ideal mode of healing with maximal functional and cosmetic recovery.
2. **Fibrosis:** replacement of lost or discontinued tissue(ROLT) by fibrous tissue: the fibrous tissue has not got the function and form of the lost tissue. So, healing with fibrosis is a bad mode of healing.

In human beings unfortunately, the ability to heal by regeneration is limited to simple tissues (epithelium, hepatocytes, and bone). While some lower creatures as salamanders they can heal organs as limbs.

The role of plastic surgeon is to deal with the defects that would heal by fibrosis by tissue transfer trying to avoid the fate of fibrosis.

THE ROLE OF PLASTIC SURGEON:

In wound healing, all lost tissues would heal ultimately, however in human beings' tissues heal mostly by fibrosis with its unfavorable functional and cosmetic outcome. To avoid this fate the plastic surgeon would reconstruct the defects by replacing the lost tissues by transferring tissues from donor sites of the same patient. The donor area would donate tissues to the defected area (donor gives, and recipient takes). By doing this we are looking to achieve a result that is better than fibrosis but less than the ideal regeneration and not as the original lost tissues. To be optimal, the tissue transfer (graft or flap) should achieve the following criteria:

1. The transferred tissues should be as similar as possible to the lost tissues in the defect (replace like with like)
2. The tissue transfer should achieve maximum benefit to the recipient area.
3. The tissue transfer should achieve minimal harmful effect on the donor area, this is referred to as **minimal donor site morbidity.**
4. The tissue transfer should be safe to patient.

YOU RUB PETER TO PAY PAUL, PETER SHOULD BE ABLE TO AFFORD IT.

METHODS OF SOFT TISSUE CLOSURE (RECONSTRUCTION)

There are different methods of closure of wounds, which vary in complexity, depending on the defect, and whether there is tissue loss or not. The hierarchy of methods includes:

1. Direct closure.
2. Healing by secondary intention.
3. Skin grafting; split thickness, or full thickness.
4. Flaps. Local or distant.

WHEN AND HOW TO CLOSE A DEFECT?

When we face a wound, we should answer two questions:
When do we close the wound? The answer to this question is: We close it when it is **clean** to be closed! This means that the wound should be free of contamination. And dead tissue and the second question to be answered is **how to close it?** The answer is that we choose the appropriate method depending on the need and the condition of the defect. When there is no tissue loss, or little skin loss, so that the edges of the wound could be approximated to each other without tension, then we close it by direct closure, but if the tissue loss is beyond the ability to directly approximate the wound edges, then we need to choose another method (i.e. Healing by secondary intention, skin graft, or a flap).

MANAGEMENT OF DEFECTS

1. WHEN **NOW: When the wound is clean.
**LATER OR DELAYED: when the wound is not clean.

** **Clean:** means **minimal bacterial load (contamination and infection)**, and **minimal necrotic tissue**, this depends on two factors:

1. **MECHANISM OF INJURY, AND INSTRUMENT USED**: Crushing injuries, and injuries inflicted by blunt instruments are usually associated with a degree of contamination and tissue damage.
2. **TIME ELAPSED FROM INJURY TO PRESENTATION**: if this time is more than 6 hours, then the wound is considered contaminated, an exception to this rule is the face, in which primary closure could be done within 24 hours, this is due to the excellent vascularity of the face.

HOW: Wounds are closed by one of 5 methods:

1. Direct closure.
2. Healing by secondary intention.
3. Skin grafting; split thickness, or full thickness.
4. Flaps. Local or distant.
5. Prosthesis

Classification of wounds:

Depending on the degree of tissue necrosis and contamination, wounds are classified into:

1. **Incised wound**: caused with sharp, relatively clean instruments, like kitchen knife, these wounds have minimal necrosis and contamination. These wounds are closed primarily if patient arrives within six hours.

2. **Lacerated wounds**: characterized by jagged edges, caused with blunt instruments, they are associated with moderate degree of necrosis and contamination, if patient arrives within six hours, these wounds are managed by wound excision, (to transform it into an incised wound) and then direct closure.

3. **Crushed wounds**: seen in industrial and severe road traffic accidents, associated with heavy contamination and severe tissue revitalization.
   These wounds are managed by wound opening, cleaning, irrigation and adequate debridement, which means excision of the devitalized tissue.
   This procedure is repeated daily till the wound is clean with no dead tissue, when it could be closed.

   Primary closure is contra-indicated in these crushed wounds, as the dead tissue, contamination, and the tissue tension due to inflammatory edema will predispose to infection, especially gas gangrene and tetanus.

Direct closure:
This method is used when there is no or minimal tissue loss, so we can approximate the wound edges without tension.

Edges of the wound are approximated usually with suture materials, however other methods may be used as, staplers, tissue glue, or adhesive tape (Steri-Strip).

Wounds with tissue loss.

When there is tissue loss, so the wound could not be closed directly without tension, other alternatives should be considered to deal with the wound;

- Healing by secondary intention.
- Skin grafting; split thickness, or full thickness.
- Flaps. Local or distant.

Healing by secondary intention.

We may let the wound to heal by secondary intention, this option is good for, small defects, when the area is of no functional or cosmetic value, or when other operative methods like grafts or flaps are not safe.

Skin grafts:

In this method skin (EPIDERMIS AND DERMIS) or part of the skin (Epidermis and part of the Dermis) is harvested from a donor area and applied on the defected area (recipient area).

Flaps:

A FLAP is a piece of tissue carries its own blood supplies that is moved from its original site, to cover a defect.

SKIN GRAFTS

SKIN GRAFTS ARE DIVIDED INTO:

1.Split thickness skin grafts: are thin grafts formed of epidermis and a thin part of the dermis, the donor site heals by regeneration (like the healing of superficial second-degree burn) within two weeks, and the same donor area can be re-harvested after this period. Almost any area of the body may be used as a donor site, so large areas of skin defects may be covered with STSG.

Full thickness skin grafts: consists of the whole skin (epidermis and dermis), it is taken from areas of loose skin as the donor area is closed by approximation of the edges (direct closure), due to this fact, only small areas could be covered by FTSG.

FTSG is superior to STSG from functional and cosmetic aspects: Better texture, better color matching with less pigmentation problems, more durable, less wound contraction; they have better sweat and sebaceous glands function, it grows with the child, and they have better final innervation.

Although FTSG are better they have 2 drawbacks: they are less available to cover large areas, and they are more difficult to take.

Graft take

The process by which the graft is integrated in the recipient site and acquires new blood supply.

HOW DOES (SKIN GRAFT TAKE) OCCURE

Skin graft take passes through two stages:

1. PLASMATIC CIRCULATION: in the first 1-2 days, the graft is nourished from the underlying recipient site by the process of imbibition or diffusion (plasmatic circulation).

2. NEOVASCULARIZATION: within 2-3 days, the graft blood vessels are joined with the recipient site vessels, the latter process is called Neovascularization.

SIGNS OF SKIN GRAFT TAKE

1. The graft is adherent to the recipient site.
2. The graft is pink in color.
3. The graft blanches with pressure, denoting vascularity.

Factors affecting graft take:

1. **Vascularity of the recipient site.**
   This is the most important factor. Skin graft take is poor on avascular areas, such as cortical bone bared of its periosteum, cartilage devoid of its perichondrium, tendons bared of its peritendon, and over irradiated areas, graft take does not take place on prosthesis.
2. **Bacterial load** (contamination and infection), especially that is caused by streptococcus, group A.
3. **Presence of barriers between the graft and the recipient area, as hematoma, seroma, debris, or foreign materials.**
4. **Immobilization**, the graft should be fixed to the recipient site, as graft mobility hinders imbibition and neovascularization.

What type of skin graft to use, STSG or FTSG?

When the area to be covered, needs good quality of skin, i.e. good cosmesis, as on the face, or good durable skin as on the hand, FTSG is used, but if we are to cover large areas, as in major burns, then STSG is the logic choice.

Remember:

The thicker the graft, the better. But: less available, and more difficult to take!!!!!!!

FLAPS

A FLAP is a piece of tissue carries its own blood supplies that is moved from its original site, to cover a defect.

Flap composition

Flaps vary in their composition, to suit the need of the recipient area, it may be composed of; skin and subcutaneous tissue (skin flaps), skin and muscle (myocutaneous flaps), muscle alone (muscle flaps), skin, fascia and bone (Osseofasciocutaneous flaps)

Difference between skin grafts and flaps, and indications of their use:

1.Skin graft is thin as it is composed of skin (FTSG), or part of skin (STSG), while flaps are formed of more bulky tissue as mentioned above, so the complexity of the defect and its requirements dictates whether to use a skin graft or a flap, and also the type of flap to be used, to give an example: A facial defect following excision of basal cell carcinoma, may be closed by FTSG, but the defect that follows excision of infiltrating oral tumor will require a flap.

2. Skin grafts depend on the vascularity of the recipient site for their survival, so they cannot be used on vascular beds or over prosthesis, flaps on the other hand are used in these situations as they bring their blood supply with them.

In summary, flaps rather than grafts, are used when the latter are insufficient to cover the defect, or they would not be taken.

Flaps may be raised locally to cover nearby defects or may be brought from distant sites as free flaps, in the case of free flaps the flap with its vascular pedicle (its supplying artery and vein) are taken from the donor area to the recipient site where the artery and vein are connected by microvascular anastomosis to an artery and vein near the recipient site.

Vascular anomalies

Vascular anomalies: are congenital anomalies caused by abnormal growth of blood vessels, leading to masses originating and consisting of blood vessels with variable shapes. Vascular lesions in the head and neck region can result in significant cosmetic problems for the patient, and some may lead to even serious life-threatening hemorrhage and other complications.

Vascular anomalies should be differentiated from congenital nevi. Vascular anomalies are formed of blood vessels filled with blood so their color is related to blood with different shades, while nevi are formed of melanin cells so their color is shades of melanin. This differentiation is important as congenital nevi are premalignant and may lead to malignant melanoma but vascular anomalies are not premalignant.

Classification of vascular anomalies:

In the past, there has been confusion regarding the proper nomenclature for vascular lesions.

1- According to morphology:
(Salmon patch, strawberry hemangioma, port wine stain, cherry vascular anomalies)

2- According to the diameter of the blood vessel:
Thin small channel: capillaries wide cavernous
Mixed: cavernous and capillaries

3- In 1982, Mulliken and Glowacki "biologically classified" the vascular anomalies based on their clinical behavior and endothelial cell characteristics into two groups: hemangiomas and vascular malformations.

Now we should depend only on THE LAST BIOLOGICAL classification and ignore the previous classifications because in Mulliken biological classification the 2 sub-types of vascular anomalies differ regarding prognosis, outcome and management.

Group 1: Hemangiomas:

- The suffix **(–OMA)** in **(HAEM- ANGI- OMAS)** is due to the tumor like behavior of hemangiomas. **In fact they are not real neoplasms and they are also not pre-malignant lesions.**
- They are the most common tumors of the head and neck in infancy and childhood, comprising approximately 7% of all benign soft tissue tumors.
- The definition of hemangiomas is restricted to vascular anomalies caused by "endothelial proliferation".

Characteristics of endothelial cells in hemangiomas:

- Hemangiomas consist of young endothelial cells: these cells are: (Plump, active cells with high mitotic activity; they have high number of mitotic figures indicating division of endothelial cells.
- They have receptors to mediate cellular proliferation. In between the endothelial cells there are mast cells.
- These cells are considered as embryonic cells with short doubling time.

HEMANGIOMAS HAVE 2 PHASES:

Hemangiomas start de novo or as tiny lesions at the age of 3-4 weeks. This is in contrary to the vascular malformations which appear at birth.

- **Proliferative phase: relatively rapid early growth until approximately 6 to 12 months of age.**
- **Involution phase or regression phase: follows the proliferative phase and lasts till 5 to 9 years of age. It is characterized by a decrease in size and fading of the red color (mottling)**

In the proliferative phase mast cells will increase in number playing a role in neoangiogenesis, so the lesion will expand and grow rapidly till the age of one year, then the lesion will stabilize, then starts to involute characterized by a decrease in size and fading of color (mottling).

Clinical picture of hemangioma

1. Natural history: This is important for diagnosis of hemangiomas

- Appear or start as small lesions at the age of 3-4 weeks.
- Grow rapidly to reach their maximum size at the age of one year.
- They usually involute and disappear either completely or remain as a thin fibro- fatty tissue.
- The process of involution is normally *slow and will not be completed until the age of 5 to 9 years.*

2. They are the most common tumors of infancy and childhood, comprising approximately 7% of all benign soft tissue tumors.

3. Female to male is 3:1.

4. More common in pre-mature babies.

5. 80% are solitary, 20% are multiple.

6. Most of them (60%) are in the head and neck, less in trunk and less in the extremities.

Treatment of hemangiomas

- **As they resolve spontaneously, they are usually managed by expectant observation: follow up to check for involution and to check for possible complications.**
- **If it is hemangioma you reassure the family, it will involute and resolve spontaneously.**
- **Treatment is indicated when they are complicated.**
- **First line of treatment is by systemic steroids or beta blockers (propranolol) which induce involution of the lesion.**
- **Other methods include LASER or surgery.**

In summary, hemangioma is one of the 2 divisions of vascular anomalies, they form blood vessels, multiply, proliferate, involute and disappear spontaneously, treatment is by observation unless they are complicated; in this case the first line of treatment is by systemic steroids or by beta blockers (propranolol).

Complications of hemangiomas:,

1) Obstruction: hemangioma can grow in the eyelid obstructing the vision leading to amblyopia (lazy eye). They may also obstruct airway or auditory canal.
2) Bleeding.
3) Large hemangioms may entrap platelets leading to thrombocytopenia, this is called Kassbach-Merit syndrome.
4) Skeletal distortion.
5) Congestive heart failure due to multiple hemangiomas.
6) Ulceration and infection.

Group 2: Vascular malformation:

- They are structural abnormalities resulting from errors in the morphogenesis of embryonic vessels between weeks 4-10 weeks of gestation.
- Almost always sporadic.
- They appear at birth and their growth is parallel to the growth of the child.
- They are formed of mature endothelial cells which have normal turnover rate throughout their natural history.
- These cells have no receptors and no mast cells between them.
- Vascular malformations can be one of two types: either high flow or low flow (capillary, venous, lymph or combined).
- **Never goes spontaneously as hemangiomas and they do not respond to steroids.** It may need treatment if **complicated** or for **cosmetic reasons.**
- **Female: male is 1:1**

Complications:

1- Erosion of bones leading to fractures.
2- Stealing blood from a limb leading to atrophy of distal parts.
3- Entrapment of platelets.
4- Bleeding.

Treatment:

1-Surgery
2-Laser
3-Embolization: by injecting material embolizing the feeding artery.

Port-wine stains are capillary vascular malformation not (hemangiomas).
They are usually evident at birth.
They are facial lesions restricted to one or more of the three trigeminal sensory areas (ophthalmic, maxillary and mandibular branches).
They are flat and sharply demarcated, grow proportionally with the child.
Their surface is studded with nodules.

Port-wine stains can be part of *Sturge-Weber syndrome:*

1-Vascular malformations of the face with involvement of choroid plexus and meninges.
2- The stain mainly involving the ophthalmic division.
3- Local or generalized seizure.
4- Ipsilateral glaucoma.

Cleft Lip and Palate

Embryology

Frontonasal process which is proliferation of mesenchyme from the ventral surface of the developing brain, forms the, **nose, the central part of the upper lip (the philtrum), and the central part of the alveolar process (the part which carries the central and lateral incisors)**. The first pharyngeal arch gives rise to the **maxillary and the mandibular processes**, from the former, develop the lateral parts of the upper lip, the lateral parts of the alveolus, it also gives rise to the palatine shelves which fuse in the midline to form the palate. The mandibular processes from each side fuse to form the lower lip and the mandible.
So the cleft lip and cleft alveolus result from non-fusion of the frontonasal process with either one or both maxillary processes resulting in left, right, or bilateral cleft.
Non fusion of the palatine shelves, result in cleft palate.

Incidence

- The incidence of Cleft Lip or Cleft Palate is 1:750.
- It constitutes 2/3 of all craniofacial anomalies; the incidence of cleft lip is two times that of cleft palate.
- In cleft lip, 60% of the cases affect the left side, 30% the right side, and 10% bilateral.
- Cleft lip is more common in males, while cleft palate is more common in females.
- Isolated cleft palate is associated with other syndromes in 30% of cases.

Etiology

Not known. While hereditary plays important role, other factors may include:

1. Vitamin deficiency in pregnancy (folic acid).
2. Drugs as steroids.
3. Gestational viral infections or irradiation.
4. Loss of amniotic fluids.

Cleft Lip

Cleft lip, which is usually associated with nasal deformity, is purely aesthetic problem, although it can be corrected at birth or later at any age. Most surgeons prefer to repair it at 3 months of age.

Cleft Alveolus

Cleft alveolus may lead to abnormal teething especially of the lateral incisors and canines. Orthodontic treatment may be needed to correct the alignment of the alveolar arch, and those children need alveolar bone graft at the age of 8-9 years to allow the eruption of the permanent canine.

Cleft Palate

Cleft palate is not an aesthetic problem as cleft lip, but it is associated with many functional problems: **feeding**, **speech, regurgitation of food from nose and may lead to hearing loss due to recurrent ear infections**. Normally Eustachian tube is patent to equalize pressure. Patients with cleft palate have Eustachian tube dysfunction due to abnormal insertion of muscles, so the tube is not patent (obstructed) so fluids will accumulate in the tube leading to **secretory otitis media** which is evident by accumulation of fluids behind the ear drum, this is treated by the ENT specialist by drugs as anti-histamines, or by drainage of the fluids surgically by puncturing the ear drum and putting tubes (Gromet tubes) for continuous drainage. If secretory otitis media is not properly treated it would be complicated by bacterial acute otitis media (recurrent ear infections) that may lead to with and hearing loss. So, remember that hearing loss in cleft palate patients is not congenital by acquired due to repeated ear infections.

Function of the soft palate:

The soft palate (velum) is formed of muscles that elevate the soft palate and push it backward to meet the posterior pharyngeal wall to close and separate the nasopharynx from the oropharynx. So, the **velopharyngeal competence** is defined as the ability of the soft palate (velum) and the pharynx to act as a valve between the mouth and the nose. This valve should be open in **breathing** to allow air to get into the airways. Also, it should be closed in **swallowing** to prevent nasal regurgitation of food.
Normal speech also requires closure of the port between the mouth and the nose to create a positive pressure inside the oral cavity to pronounce most of the consonants. **In suckling** the **port between the mouth and the nose should be closed to create negative pressure for suckling.**

Failure of this valve mechanism is called **VELOPHARYNGEAL INCOMPETENCE.**
Among many causes, **cleft palate is the most common cause of this incompetence**, which is attributed to 3 abnormalities in the patient with cleft palate:
1. The mechanical defect of the cleft.
2. Hypoplasia of the palate.
3. Abnormal insertion of the palatal muscles

Surgical correction of cleft palate aims at closure of the cleft palate to restore the velopharyngeal competence.

Family Counseling

The parents of the cleft baby should be counseled, this means reassuring them, relieving their anxiety, discussing with them the problems associated with cleft palate, especially the feeding and its management. Also, they should be informed about the other functional deficits: nasal regurgitation, speech abnormalities (nasal speech), and the importance of follow up by the ENT specialist to manage the ear infections. The family should be introduced to the Cleft Palate Team which consists of: Plastic surgeon, Pediatrician, ENT surgeon, Dentist, Orthodontic surgeon, Speech therapist, Cleft palate nurse, and Social worker.

1. Feeding

For normal feeding the baby should suckle the milk and then swallow it. It is so important to educate the mother that babies with cleft palate have **defective sucking**, simply because they cannot create negative pressure inside their oral cavity to suckle (the mouth is communicating with the atmosphere through the nose). This makes breast feeding difficult. **Although suckling is defective, swallowing is normal.**
With these fruitless efforts the baby will get exhausted and fall asleep with insufficient feeding that leads to weight loss. Although there are special nipples available in the market, that are designed to close the cleft while feeding to help suckling. This special nipple is not mandatory as a simple solution of the problem of ineffective suckling is simply to passively introduce the milk to the mouth by **widening the opening of the bottle nipple,** so as the milk is getting to the mouth passively.
The following roles should be applied:

1. The mother is the best nurse; she is the person who should be involved from the very beginning in the feeding and care of her baby.
2. Nasogastric feeding should not be used for permanent feeding.
3. Babies should be nursed in semi-sitting position, and should be burped well, to get rid of the swallowed air.
4. It should be realized that feeding of cleft babies – at least in the beginning- takes more time than normal babies, the mother must be patient!!!
5. Although breast feeding is difficult some babies can be breast fed as the mother would help this by pressing her breast. So, breast feeding although difficult is not contra-indicated.

2. Speech

Normal speech requires that air coming from the lungs, passing through the vocal cords, is collected in the oral cavity to create positive pressure, before passing through the lips to pronounce most consonants.
Patients with cleft palate, with **velopharyngeal incompetence** are unable to create this positive intra-oral pressure as air leaks through the nose, leading to nasal escape, or abnormal nasal speech. Again, surgical correction of the cleft helps to restore normal speech, in addition they need the help of speech therapist to achieve good speech.

3. Recurrent otitis media and hearing loss

Normally the Eustachian tubes should be patent and aerated to balance the pressure on the two sides of the tympanic membrane.
In cleft palate patients the tubes are not patent due to abnormal insertion of pharyngeal muscles, so fluid accumulates behind the eardrums leading to recurrent otitis media, and if not managed may result in hearing loss.
The ENT surgeon is an important member of the Cleft Palate Team deals with these problems by treating secretory otitis media (by drugs or Gromet tubes) and treating acute otitis media by suitable antibiotics.

4. Nasal Regurgitation

Children with cleft palate have embarrassing nasal regurgitation as swallowed food will escape from nose.

Timing of Surgical Repair of Cleft Palate

Speech therapists believe that, the earlier the cleft palate repair is, the better the outcome of speech would be, so they encourage early repair, but the facial surgeons think that early surgical repair would interfere with the facial bony growth leading to retardation of maxillary growth (dish face). So, **the compromise between these two opinions is to operate at 1 year of age.**

Local Wound Management

Open wounds should be managed locally according to the following principles:

1. **Limb Elevation**

   To decrease edema that increases interstitial pressure and decreases tissue perfusion. Elevation improves vascularity and preserves viability of the injured cells in (the zone of injury) and enhances wound healing.

2. **Decrease Bacterial Load**

Bacteria may be present in the wounds in two forms that should be recognized and differentiated.

A. **Contamination**: **defined as the mere presence of bacteria on the wound environment, it is enhanced by necrotic tissues, external contaminants and foreign bodies; it is a mechanical process** that can be detected by **wound swab culture.**

Management of contamination

1. Wound debridement: means excision of the necrotic tissue.
2. Wound mechanical irrigation with normal saline under appropriate pressure.
3. Topical antimicrobial agents as **silver sulphadiazine**
4. It should not be treated with systemic antibiotics, because the necrotic tissues are not vascular, so systemic agents would not reach the bacteria.

B. **Infection**: Contrary to contamination, infection is an **active biological phenomenon** and involves bacterial invasion of living tissues with the response of the living body to this invasion by **inflammation.** The source infection may come from the **contaminating bacteria** when their number and virulence exceed the immunity or may be (hematogenous) coming through **the blood stream from distant sources**. **Wound infection is manifested by the known signs of inflammation.** Laboratory diagnosis of infection is by **(wound tissue culture):** sending a piece of **living tissue** for culture. In this case wound infection is diagnosed when the bacterial number is equal or more than 100,000 microorganisms per gram of tissue. Wound infection is a serious complication that may lead to sepsis and septic shock, and **should be treated with appropriate systemic antibiotics, NOT by local tropical agents.**

3. Avoid Trauma to the Wound

There are two sources that cause wound trauma: **Mechanical trauma and chemical trauma.**

a. Mechanical trauma: The following causes of mechanical trauma should be avoided:

I. Adherence of dressing to the granulating wound causing damage to the healing tissues when the dressing is removed, so the dressing should be non-adherent by using appropriate agents.

II. Gentle handling of tissue and avoid aggressive manipulation of the wound by rubbing.

III. Avoid tight dressing.

b. Chemical trauma:

Many antiseptics are cytotoxic to the wound tissues, they should not be applied to the granulating wounds, and the rule is: DO NOT PUT ON THE WOUND WHAT YOU CAN NOT PUT IN YOUR EYES. So, irrigation of the wound with normal saline is the safest option.

REMEMBER: Wound dressing should be painless! Pain means that you are harming the wound.

4. Keep the Wound Moist

Keeping the granulating wound (open wound) wet prevent its desiccation and enhance its healing because cellular functions require moist environment. The wound media however should not be over-wet.

Dr. Bareqa Salah

Lectures and Seminars

Contents:

Lectures:

Wound Healing

Keloid and Hypertrophic Scars

Frostbite and Related Injuries

Late Burn Complications

Seminars:

Detailed Pathophysiology of Different Types of Ulcers

Benign and Malignant Skin Conditions

Soft Tissue Sarcomas

Edited and Groomed by:

Fareed Halteh

Wounds and their management

- Types of wounds:
    - Contusion:
        - It is a bruising injury caused by a blunt trauma
        - Contusions are, sometimes, associated with a hematoma, which can be small or large.
        - A small hematoma is resorbed by itself due to the action of macrophages. This means that there is no need to open and evacuate small hematomas. An exception to this rule is small hematomas in the face. These need to be opened and evacuated because leaving them will cause fibrosis and persistent indurations leading to cosmetic problems. Intracranial hematomas should be evacuated, as well.
        - Large hematomas are managed according to the of presentation by one of the following techniques:
            - Aspiration: this is applicable when the hematoma is acute (<24 hours). At that early stage, the hematoma is still liquid.
            - Incision and drainage: after 24 hours of injury, the hematoma starts to undergo clotting, and it is best managed by incision and drainage.
    - Abrasion:
        - It is the loss of epithelial cells (epidermis), and sometimes the upper dermis.
        - They are managed by dressing to prevent secondary bacterial infections.
    - Puncture wound:
        - This is caused by pointed objects (lead pencils), and is sometimes associated with the implantation of foreign bodies (wood or rust). The depth of the wound and the degree of contamination cannot be predicted in this type of wounds.
        - Management:
            - Tetanus vaccine: puncture wounds provide a suitable environment for the growth of the anaerobic Clostridium tetani
            - Excision of the puncture wound
            - Removal of foreign bodies.
    - Simple lacerations:
        - Lacerations are caused by sharp objects like knives.
        - Management:
            - Cleaning
            - Debridement

            - Suturing: this depends on the degree of contamination. If the wound is clean, it can be closed primarily (immediately). However, in the case of heavily contaminated wounds or wounds more than 6 hours old, delayed closure is indicated. However, lacerations in the face, can be closed after 24 hours of injury.
    - Avulsion flap:
        - An undermined laceration in the dermis and subcutaneous tissue
        - Management:
            - Debridement of the edges
            - Excision of the small avulsion flap: this is recommended to prevent the trap door effect. The trap door effect is the formation of nodules and indurations on the flap due to lymphatic and venous obstruction upon suturing the flap.
            - Suturing
- Methods of soft tissue closure:
    - Direct closure
    - Healing by secondary intention
    - Skin grafting: split thickness or full thickness
    - Flaps: local or distant
    - Prosthesis
- When and how to close a defect?
    - When?
        - If the wound is clean, we close it. This means that the wound should b free of contamination, infection, and dead tissue.
    - How?
        - It depends on the need and the condition of the defect. When there is no tissue loss, or minimal skin loss, the edges of wound can be approximated without tension. Here, we use direct closure. If the tissue loss is great (beyond the ability of direct approximation), a different method must be chosen.
- Management of defects:
    - When?
        - Now: when the wound is clean.
        - Later: when the wound is dirty
    - The cleanness of the wound depends on the mechanism of injury and time elapsed from injury to presentation. Crushing injuries with blunt instruments are associated a high degree of tissue damage and contamination. If the time between injury and presentation is more than 6 hours, it is considered as a contaminated wound. Facial wound are an exception to this rule; primary closure can be done after up to 24 hours after injury.

- Different classification for wounds:
    - Incised wound:
        - Caused by a sharp, relatively clean instrument (kitchen knife).
        - These wounds have minimal necrosis and contamination.
        - If the patient arrives within 6 hours of injury, they can be closed primarily.
    - Lacerated wound:
        - Characterized by jagged edges. They are caused by blunt instrument.
        - Associated with a moderate degree of necrosis and contamination.
        - If the patient arrives within six hours of injury, they can be managed via excision. They are transformed into an incised wound that can be closed primarily.
    - Crushed wound:
        - Seen in industrial and severe road traffic accidents.
        - Associated with heavy contamination and severe tissue devitalization.
        - These wounds are managed by opening, cleaning, irrigation, and adequate debridement. The devitalized tissue should be excised. This procedure is repeated daily until the wound is clean with no dead tissue. This is when you close the wound.
- How to close a wound with tissue loss?
    - Secondary intention: this option is good for small defects, when the area is of no functional or cosmetic value, or when other operative methods like flaps or grafts are risky
    - Skin grafts: in this method, a part of the skin is harvested from a donor area and applied on a defected area.
    - Flaps
- Types of wound healing:
    - Primary healing: when the wound is closed within hours of its creation
    - Delayed primary healing: if the wound is contaminated, we leave it open to prevent wound infection. Then, it is closed after 3-5 days.
    - Secondary healing: open, full thickness wounds are allowed to close by both contraction and epithilization. Wound contractions means a decrease in the wound size due to the contraction of myofibroblasts, which contain filaments.
    - Tertiary healing: when we use a skin graft or flap to cover the defect.
- Overview of wound healing:
    - In the process of healing, the injured tissue, depending on its type, is either regenerated or repaired.
    - Regeneration: the process in which the same type of cells regrows without functional effects. Examples include: regeneration of GI mucosa and vascular endothelium

    - Repair: the process in which the injured cells are replaced by fibrous tissue (Scarring). Examples include: repair of neurons and muscle cells.
- Stages of wound healing:
    - Early stage:
        - Tissue injury
        - Coagulation
        - Inflammation: during the inflammatory phase, polymorphs are recruited during the first 24-48 hours. Macrophages play their role in 48-72 hours. Fibroblasts follow. Macrophages function as phagocytic cells. They are the primary source of growth factors (cytokines), which regulate the whole process of wound healing.
    - Intermediate phase:
        - Mesenchymal cell migration and proliferation
        - Epithelization from the edges of the wound and remaining appendages.
        - Angiogenesis: the formation of new blood vessels
    - Late phase:
        - Matrix formation: collagen synthesis by fibroblasts. Collages fives tissues their strength and integrity.
        - Wound contraction
        - Proteoglycan synthesis: this is more important in the healing of fractured bones.
    - Final phase: this is the stage of wound remodeling. This occcurs by the breakdown and resynthesis of collagen. This stage can continue for 12-18 months.
- The stages of wound healing are not neatly arranged into distinct stages; these stages overlap.
- Abnormal wound healing:
    - The process of wound healing might be inadequate or abnormally extensive.
    - Inadequate wound healing: the causes of inadequate wound healing are either local or systemic:
        - Local:
            - Local tissue hypoxia: fibroblasts and macrophages are sensitive to hypoxia
            - Infection: this will perpetuate the inflammatory (early) phase
            - Presence of a foreign body
        - Systemic:
            - DM: it causes microangiopathy and most importantly atherosclerosis. At the molecular level, it affects inflammation through impairing chemotaxis of macrophages, fibroblast function (even if glucose levels are controlled), and inhibits epithelization, angiogenesis, and wound contracture.

        - Vitamin C deficiency: this prevents cross linking of proline and lysine moieties in tissue collagen.
        - Drugs (steroids and chemotherapy) : steroids are anti-inflammatory; therefore, they prevent the release of cytokines. This process is counteracted by vitamin A through an unknown mechanism affecting only wound healing.
        - Malnutrition: protein balance is affected
        - Trace elements deficiency: zinc and copper
        - Renal disease: causes hypoalbuminemia due to proteinurea. In addition, it might lead to uremia with the toxic effects of uric acid on the tissues
        - Liver diseases causing hypoalbuminemia
        - Old age: attributable to slower metabolic rates.
    - Acute wound healing is governed by macrophages. They release cytokines. Cytokines will cause fibroplasia and cellular proliferation. Proteases and their inhibition are controlled processes, so epithelization is followed by matrix deposition, angiogenesis, and tissue remodeling. This leads to normal wound healing.
    - If the wound was affected by repeated trauma due to its location, for example, the acute inflammatory phases changes into a chronic inflammatory phase due to the increased activation of macrophages and neutrophilic infiltration. Inflammatory cytokines are released in copious amounts from macrophages. The neutrophils release hydrogen peroxide, a reactive oxygen species, responsible for bacterial killing. Both cytokines and peroxide lead to the activation of matrix degrading proteases and decrease the activation of protease inhibitors leading to excessive ECM degradation. In addition, peroxide will kill surrounding cells impairing epithelization. This leads to more inflammation; therefore, leading to a vicious cycle of chronic inflammation and ulceration.
- Excessive wound healing:
    - There are two types of abnormally excessive wound healing; hypertrophic scars and keloid scars. These two types are different from normal scars biochemically and histopathologically.
    - Occurs only in humans. No animal models exist
    - During the early stages, these two anomalies cannot be differentiated from each other.
    - Incidence of excessive healing is 5-15% of all wounds.
    - Common features:
        - Raised above the skin
        - Erythematous: ongoing inflammation; always reddish

    - Pruritic
    - Near the wound
    - Common in areas of stress and tension (joint over shoulder, upper back, anterior chest, and ear lobe)
- Hypertrophic scars develop insidiously 6-8 weeks after trauma. They worsen up to 6 months to 2 years. Then, they regress spontaneously or by medications. Keloid scars do not regress
- Both are more common in darkly pigmented races
- When excised, they have a tendency to re-occur months after treatment.
- Keloid scars tend to worsen during puberty and pregnancy.
- Fetal wound healing doesn't leave any scars.
- Keloids and hypertrophic scars do not have an increased number of fibroblasts; they have an increased activity of fibroblasts.

Hypertrophic scar	Keloid scar
Improves with time (within 2 years)	Does not improve with time
No genetic predisposition (can occur in caucasians)	Genetic predisposition (blacks with nigroid features). Autosomal dominant with incomplete penetration
Limited ot the borders of the original wound	Extends beyond the margins of the wound.
Less collagen	More collagen
Distinct bundles with fine fibers	Large collagen fibers with closely packed fibrils
Fibers parallel to the dermis	Fibers random in orientation
Less cytokines	More cytokines
Myofibroplasts present (undergo contraction formation)	Absent myofibroplasts (do not undergo contracture formation)

- Pathogenesis of fibroproliverative scars:
    - Increase in the activity of cytokines, especially TGF-beta from platelets, macrophages, T-cells, and fibroblasts.
    - TGF-beta causes an increase in pro-collagen gene expression and reduction in proteolytic enzyme synthesis.
    - It increases the lay down of extracellular matrix (fibronectin and proteoglycans). Fibronectin is the tissue glue secreted by cells into the ECM. It allows for the chemotaxis of inflammatory cells.
    - Angiogenesis is triggered by other cytokines; therefore, the scar appears red.

        - The itching is caused by elevated levels of mast cells when compared to normal scar tissue. The itching can be relieved by antihistamines (systemic or local).
        - TGF-beta affects apoptosis related genes/ it prolongs the lifespan of cells in these scars when compared to normal scars.
- Histopathological differences between hypertrophic scars and keloid scars cannot be appreciated through light microscopy. They can only be differentiated through immunohistochemistry or electron microscopy.
- Treatment:
    - Surgical:
        - If surgery was not coupled with adjuvant therapy, the scar will reoccur. Recurrence rate is 50-80%. The median recurrence time is 13 months.
        - Multimodal therapy should be applied
        - Z-plasty or W-plasty are used for scars occuring against lines of minimal tension to reorient the scar.
        - Steroids are used to suppress the inflammatory process intraoperatively. This technique is used for keloids.
        - Factors to consider during surgical treatment:
            - Tension free closure to prevent ischemia of the edges of the wound
            - Removal of all old scar tissue
            - Avid trauma to surrounding normal skin and tissue
            - Obliterate dead space to prevent hematoma formation
            - Multimodal therapy.
    - Artificial skin (Integra):
        - It is a form of artificial dermis
        - These are used to supplement skin grafts (mainly epidermis) by filling the defect that was occupied by the debrided dermis over which the skin graft is applied.
    - Steroids:
        - Intralesional Triamcenolone acironide: most effective and young scars.
        - 4-8 weeks between injections to prevent systemic side effects (cushinoid features which are reversible). In children, the dose is 40 mg for a 6x10 lesion. In adults, the dose can reach up to 120 mg for a lesion of the same size. Steroids are not given for children less than one year of age as they may affect bone growth.

    - Side effects: hypopigmentation, skin and subcutaneous tissue atrophy (Adipose tissue does not regenerate; therefore, a depression may form permanently), telangiectasias, necrosis and ulceration of the skin, and cushinoid features.
- Pressure therapy:
    - Utilizes custom made pressure garments.
    - It reduces and softens hypertrophic scars. It also reduces the progression in 60-85% of hypertrophic scars. However, a single study reported no significant benefits of the use of this type of therapy.
    - The mechanism is by creating local tissue ischemia that leads to reduced metabolism. This will lead to reduced cell proliferation, which results in decreased collaged synthesis.
    - 24-30 mmHg is the effective pressure range
    - The garments should be utilized 24 hours a day for 12 months.
- Topical silicon:
    - Available as gel and sheets
    - Application of 12 hours a day for 6 months.
    - Mechanism is mainly unknown; however, it may involve an increase in tissue temperature under the silicon (2 degrees). This increase in temperature inhibits fibroblastic enzymes. Therefore, less collagen is synthesized.
    - The silicon that is applied is not absorbed through the skin. It helps reduce water loss. This leads to an increase in the elasticity and cosmetic appearance of the scar without any change in the history of the scar.
- Low dose radiation: superficial radiation is used. Given to resistant cases.
- Lasers: CO_2 and argon lasers are used
- Calcium channel blockers:
    - Trigger ECM degradation
    - Decrease proline entry into ECM
    - Change fibroblast shape; therefore, they disturb its function.
- Interferons:
    - Alpha, beta, and gamma interferons are applied intralesionally.

    - Decrease cell proliferation, inhibit collagen and fibronectin synthesis, increase collagenase, and decrease glycosaminoglycans synthesis.
    - There are expensive and not used in Jordan.
    - Recurrence rate with interferon therapy is low when compared to steroid therapy.
    - Side effects: fever, chills, fatigue, decrease in WBC
- TGF-beta antagonists:
    - Inhibition of TGF-beta by exogenous administration of receptors to scavenge excess TGF-beta.
    - We can use auto-antibodies against TGF-beta
    - We can also use binding proteins (alpha-2-macroglobulins)

Detailed pathophysiology of different types of ulcers

- Venous ulcer:
    - Chronic venous insufficiency and venous hypertension due to:
        - Incompetent valve
        - DVT
        - Calf muscle dysfunction due to dystrophy or atrophy.
    - Theories that explain venous ulcers:
        - Homan's theory: in 1917, Homan said that venous ulcers result from stasis in the underlying veins.
        - After Homan, they discovered that there is a hyperdynamic circulation in the limb that has a venous ulcer. This means that is a hyperdynamic state rather than stasis that causes the ulceration.
        - Recently, a new theory is becoming wildly accepted. The white-cell theory states that when there is a problem in the veins proteins and cells leak outside the circulation. When these cells (leukocytes) are outside, they will degranulate. Their granules contain enzymes and reactive oxygen species; both of which are damaging materials. Damage to the capillaries will ensue; this will increase the permeability of the capillaries to certain macromolcules (mainly albumin and alpha 2 macroglobulin). These macromolecules will bind the growth factors (cytokines) along with the matrix material making them unavailable for the surrounding tissues. In this case, the cytokines never work because they are trapped in the extravascular space.
        - In the USA, there is a special dressing for those with a venous ulcer. These dressings contain certain digestive (proteolytic) enzymes to digest albumin and alpha-2-macroglobulin.
- Diabetic foot ulcer:
    - Pathogenesis of diabetic ulcer:
        - Neuropathy: it affects the sensory, motor, and autonomic divisions:
            - Sensory: loss of vibrational and proprioceptive sensations.
            - Motor:
                - Flat foot is due to collapse in the medial arch of the foot (muscle weakness)
                - Claw toes (hammer toes): plantar flexion at the distal interphalangial joints of the toes. It follows weakness of both flexors and extensors of the lower limb; however, flexor muscles are stronger than the extensors. This results in plantar flexion
            - Autonomic:

        - Dry skin and poor nutrition
        - AV shunting: in a diabetic patient, you will find weak pulses with pink and warm skin. This is due to AV shunting not because of good circulation.
    - Biochemical bases of diabetic neuropathy:
        - Accumulation of sorbitol inside Schwann cells leads to absorption of water from the extraneuronal tissue to the Schwann cells (hyperosmolar effect). This leads to swelling of Schwann cells, which decreases their conduction velocity. This leads to impaired sensation.
        - Increased concentration of glucose inside the neuron causes inhibition of the entry of myoenositol. Decreased myoenositol entry will lead to abnormal cellular response to receptor stimulation (there is stimulation, but the response is weak), impairment of the Na^+/K^+ ATPase pump which is responsible for nerve conduction, and a decrease in ATP production (decreased energy).
        - Glucose entry to the neurons does not require insulin
- Vascular:
    - In contrast to the classical teaching about microangiopathy of the skin, it has been found out that the only histological change is a thickening of the basement membranes of the muscle of arterioles. This thickening of the basement membrane doesn't impair perfusion of the surrounding tissue. This was proven by measuring the transcutaneous O_2 tension in diabetic foot patients. However, if the patient has peripheral vascular disease in addition to diabetes, it will be a completely different story. This means that there is nothing called microangiopathy of the skin or even of the vasa nervosa in diabetics. There is microangiopathy in the kidneys and retina.
    - Patients with diabetes characteristically have an infrapopliteal macrovascular disease (at the bifurcation of the popliteal artery)
- Infection:
    - There is no evidence that diabetic foot ulcer results primarily from infection; however, it is a contributing factor. If the diabetic ulcer becomes infected, it will make the condition worse. Infection means that there is an abundance of proteolytic enzymes, which will result in more damage to the skin. Moreover, these patients have a decreased immunity.

        - Decreased cellular immunity due to decrease in chemotaxis and phagocytosis
        - Decrease in humoral immunity dye to a decrease in antibody production. This is caused by high levels of glucose (affects lymphocyte function)
    - Note: all chronic wounds are contaminated; however, they are not necessarily infected.
        - Contamination means the presence of bacteria
        - Infection means invasion and multiplication of the microorganism in the bodily tissue with our without systemic manifestations.
    - Each chrnoic ulcer is contaminated. Therefore, if you want to diagnose an infection, you should take a swab of tissue and culture it. A result of more than 10^5 cells per gram of tissue indicates an infection.
    - When taking a swab, always take the sample from a deep part of the ulcer.
- Ischemic ulcer (arterial ulcer):
    - The oxygen molecule is very important for the synthesis and hydroxylation of collagen. Collagen is a weak structure that is strengthened by the hydroxylation of proline and lysine moieties. For this process to happen, O_2 is needed. Therefore, hypoxia is one of the causes of poor wound healing and development of chronic ulcers.
    - Causes of hypoxia:
        - Local causes: peripheral ischemia and vasculitis
        - Systemic cause: heart diseases and chronic pulmonary disease
    - Chronic ulcers are treated using cytokines:
        - You should know that some cytokines are found in acute ulcers more than in chronic ulcers. This is proven by biochemical assays. It was found out that TGF-beta is present in chronic ulcers.
        - Accordingly, TGG-beta has been used to treat chronic ulcers. However, it was found out that some patients improved while others didn't. The reason behind this variation is that cytokines work in a cascade fashion.
        - Nowadays, TGF-beta is given in combination with other cytokines. They are administered in manner similar to the physiological progression of the events.
        - The type of cytokine used to treat a chronic ulcer depends on the phase of wound healing:

- If the chronic wound is caused by a defect in the epithelization (as in venous ulcers), we administer PDGF, which is the most important cytokine in the epithelization process.
- In pressure ulcers, the defect is in fibroplasia (lay down of collagen). In these cases, we give TGF-beta, which is the most important cytokine in the process of fibroplasia.

Frost bite and related injuries

- The injuries caused by cold are divided into:
    - Tissue freezing injury (frost bite)
    - Non-tissue freezing injury (French foot, chilblain, pernio)
    - Hypothermia
- Chilblain: the skin is exposed to chronic high humidity and low temperatures without tissue freezing. The body's core temperature remains normal; it occurs to mountain climbers.
- French foot: the extremities are exposed to a damp environment over long period of time at temperatures of 1-10 degrees. Heat is lost because the extremity is wet. The vascular flow is poor because of vasoconstriction. The clinical picture is numbness, tingling, pain and itching. The skin is initially red and edematous, then it gradually becomes bluish-grayish. After a few days, the syndrome resolves; however, the extremity remains sensitive to cold.
- Cold urticaria: urticara and angioedema due to exposure to cold temperatures (seen mainly in aquatic activities). There are two types; familial and acquired. History of cold stimulation confirms the diagnosis.
- Frost bite:
    - The most common type of cold injury
    - Occurs when the temperature fall to -2C. The tissue freezes resulting in the formation of intracellular ice crystals and microvascular occlusions.
    - Pathophysiology:
        - The damage is either direct damage caused by formation of intracellular crystals or indirect due to ischemia caused by microvascular thrombosis and vasoconstriction. The damage occurs due to rewarming changes. When ice crystals melt, they will cause endothelial damage, which promotes edema. This releases reactive oxygen species, which increases tissue damage. Vasoconstriction occurs due to the release of prostaglandins.
    - Predisposing/risk factors:
        - Substance abuse (30-50%) alcohol
        - Psychiatric illness (10-20%)
        - Environmental factors (lack of appropriate clothing)
        - Peripheral vascular disease
        - Age: elderly and very young
        - Race: black more than white
        - Medications: amirophylline, caffeine, and ergot alkaloids.
    - Classification:
        - 1st degree: while/yellow plaque, hyperemia, edema, causalgia and pain (indicates nerve damage). Here, tissue loss and necrosis are rare

        - 2nd degree: blisters containing clear or milky fluid, erythema and edema are common. Characteristic recovery without tissue loss
        - 3rd degree: deep full-thickness skin necrosis. Tissue loss is common
        - 4th degree: cyanosis, gangrene and necrosis. Underlying muscles and bones are affected.
    - Treatment:
        - The most important step is rapid rewarming. Immersion in water heated to 40-42C
        - Pain killers
        - Massage is contraindicated because it increases the damage.
        - Debridement of clear blisters. Hemorrhagic blisters are left intact and aspired only if infected. You shouldn't attempt to debride aggressively during the early phase of a frostbite. Debride after you have a landmark between dead and viable tissue. The only indication for early operative intervention is to ameliorate a constricting eschar in circumferential 3rd or 4th degree frostbites to prevent the occurrence of compartment syndrome. Another indication is to drain a subeschar infection. If the gangrene is well demarcated, amputation is indicated.
        - Elevation of the affected area to decrease edema
        - Apply topical thrbomoxane inhibitors (aloevera) to injured areas
        - Systemic antiprostaglandin agents (NSAIDs)
        - Physiotherapy and mobilization
    - Adjuvant therapy:
        - Alpha blockers: work as pharmacologic sympatholytics
        - Nifedipine (Adalat)
        - Free radical scavengers (dimethylsulfide, vitamin C and E)
    - Diagnostic modalities:
        - Radio-isotope vascular imaging
        - Radio-isotope bone imaging
        - MRI and MRA
    - Sequels of a frostbite:
        - Arthritis
        - Pain at the site
        - Hyperasthesia
        - Hyperhydrosis (increse in sweating)
        - Pigment changes
        - Growth deformities
        - Cold sensitivity

Post burn complications

- Splanchnic ischemia: ischemia to the splanchnic circulation of 3 major types:
    - Acalculus cholecystitis:
        - It occurs in cases of sepsis, TPN, critically ill patients, and diabetics.
        - It happens in less than 0.5% of burn patients
        - It is more common in patients with major burns (those involving more than 50% of TBSA)
        - It occurs 2-4 weeks post burn
        - If the patient can communicate and talk, he will complain of right upper quadrant pain associated with nausea and vomiting.
        - In the lab investigations, we would have leukocytosis and increase amylase levels.
        - Ultrasound is the diagnostic tool of choice due to its high accuracy (98%).
        - Management: an urgent operation is needed because the gallbladder is distended due to obstruction. The urgent operation may be cholecystectomy or cholecystotomy (drain in the gallbladder to decompress it)
    - Curling ulcer:
        - Gastroduodenal ulcer due to ischemia and hypoperfusion
        - When it occurs in the stomach, they are multiple
        - When it occurs in the duodenum, it is solitary.
        - One third of the patients complain of pain in the form of heart burn and epigastric pain; the rest do not have pain.
        - Patients have hematemesis
        - In 12% of patients, perforation takes place (it is mostly anterior).
        - Prophylaxis is better than treatment. It can be achieved by 3 measures:
            - Resuscitation and fluid management to prevent hypoperfusion.
            - Locally acting drugs:
                - Antacids: like Maalox (magnesium and aluminum hydroxide)
                - PPIs: like omeprazole
                - H_2 blockers: whether the old or the new generations
                - Sucralfate: used for mucosal cytoprotection (since mucosa is the most sensitive to ischemia and ulceration)
                - Bismuth therapy: mechanical barier preventing acid reflux
            - Start enteral nutrition as soon as the clinical status of the patient permits enteral nutrition. (orally or through and NG tube). TPN increases the possibility of curling ulcers, and it is one of the most important causes of bacterial translocation through the GI mucosa

to the circulation. This will lead to sepsis and multi organ failure syndrome.

- Ischemic enterocolitis:
  - Ischemia to the mucosa due to hypoperfusion and hypotension ending in bacterial translocation (mainly gram negative) and it has a very high mortality rate to multiple organ failure syndrome.
  - In order prevent bacterial translocation due to poor circulation, they use a technique called selective digestive tract decompression. The technique is as follows:
    - These patients are given cefotaxime (3^{rd} generation cephalosporin), tobramycin (aminoglycoside), polymyxin, and amphotericin B (since these antibiotics are wide spectrum antibiotics, fungal infections may cause trouble to the patient).
    - This technique prevents bacterial translocation and superimposed fungal infection.
    - This technique could successfully decrease bacteremia and MOFS. However, there is an increase in MRSA infections.

- Hypercatabolic metabolism:
  - Burn patients will pass through 2 phases which are the ebb and the flow phase.
  - What is peculiar to burn patients is that they remain in negative nitrogen balance as long as their burn is not grafted.
  - Once you graft them, or the burn heals with or without scarring, they will approach zero. Then, they proceed into positive nitrogen balance.
  - Since this negative balance and other metabolic changes may persist chronically, these patients will start to lose weight from both, fat and muscle mass. This will have 3 main chronic consequences:
    - Respiratory muscle paralysis; thus, they can't breathe or can't cough. This usually ends with multiple pneumonias.
    - Immune deficiency (lymphocytes and Ig deficiency)
    - Wound healing impairment (fibroblast problems)
  - If these patients have major burns, their B<R will increase by 100-150% the normal BMR.
  - Our job is to minimize the protein deficiency as much as we can. Thus, we give these patients a diet with the following ratios:
    - Carbohydrate: 50%
    - Fat: 30%
    - Protein 20%
  - This diet should be enteral whenever the patient's status permits.

- We have formulas to estimate the energy need in burn patients and these formulas are different between adults and children. Children need more energy for their growth; otherwise, they fail to thrive.
- Setherland's formula:
  - For children: energy = 60 kCal/kg + 35 kCal for % burn
  - For adults: 20 kCal/kg + 70 kCal/kg for % burn
- Now, if after using the formula, the patient was found to need 6000 calories, then we construct a diet in which 3000 calories come from carbohydrates, 1800 calories from fat, and 1200 calories from protein.
- Being the most important part of the diet, we have several formulas for the estimation of protein needs. The most important of which is Davie's formula:
  - For children: 3gm/kg + 1 gm for % burn
  - For adults: 1 gm/kg + 3 gm for % burn
- In general, proteins are not used as a source of energy. This is why we give more carbohydrates and fats. We let the body use them as a source of energy and keep proteins for our vital functions (immunity, wound healing, and muscle mass).
- We prefer to give these patients what they need and only what they need. However, if you give them more than what they need, it will not be a problem except in COPD patients. In these patients, carbohydrates will be metabolized to CO_2 and H_2O. this will increase CO_2 concentration, and these patients end with more CO_2 retention.
- Patients with renal impairment should not receive proteins more than they need. This also applies to hepatic failure patients in whom a higher protein intake is better avoided since amino acids are normally metabolized to urea. These patients have problems in the urea cycle, which takes place in the liver. They might end up with a hepatic coma.

- Suppurative thrombophlebitis:
  - This complication is directly related to the duration of canula usage. In the special units (ICU and CCU), a microfilter is used to filter small bacteria (but not very small viruses).
  - In general, a canula should last no longer than 48 hours except in patients with difficult veins. In these cases, a microfilter must be used.
  - 1/3 of the patients who develop this complication will show clinical signs in the form of fever or tenderness at the insertion site. However, the remaining 2/3 will present with fever of an unknown origin. These usually have a completely normal physical examination. However, if you culture the blood, it will be positive.
  - In non-burn patients, the most common microorganism in canula infections is staph aureus. However, in burn patients the organism is the same as the organism found inside the burn wound.

- Heterotrophic bone formation:
    - This takes place in joints underneath a full thickness burn. If a patient had a full thickness burn over the elbow joint, and the burn was never grafted, abnormal calcifications around the joint will develop and limit the patient's movement.
    - It was found that the aggravating factor for this process is passive aggressive physiotherapy. This form of therapy will cause trauma; this trauma will lead to Ca deposition (deposited as hydroxyapatite).
    - This complication is more common in patients when the burn's surface area exceeds 20%. It occurs 3 weeks – 3 months after the burn injury.
    - In these patients calcium and phosphorus serum levels are normal because it is a local process. However, alkaline phosphatase will be elevated due to new bone formation.
    - Treatment:
        - if patients present early on, NSAIDs will prevent the differentiation of mesenchymal cells into osteoblasts. This will prevent bone formation.
        - If there is early bone lay down, the joint should be closed. This will reverse the process (seen on X-ray).
        - If bone deposition was complete, the wound is left for a year. After a year, an X-ray for the area is taken. If the X-ray shows no "hotspots", this indicates that the process of deposition has stopped. The joint can be operated. However, if bone deposition is still active, you cannot operate. If it was operated, the deposition will recur. A delay in the treatment is associated with a poorer outcome.
        - If treatment was never sought, the joint will become dysfunctional. Moreover, bony alkalosis will develop.
    - This complication has to be differentiated from a similar condition called myositis ossificans. Myositis ossificans is a limb mass that develops secondary to a muscle trauma. After the trauma, a hematoma develops. This hematoma will organize to form a well localized, hard, and rounded mass. An X-ray of this mass will reveal a rounded mass surrounded by a calcified wall. There is no center of inflammation; the disease's name is a misnomer.
- Marjolin's ulcer:
    - SCC that develops over a longstanding chronic ulcer or chronic scar (especially that of a post-burn origin).
    - When compared to SCC de novo, Marjolin's ulcer has a more aggressive systemic and local behaviors.
- Hypopigmentation:
    - Occurs in the head and neck and on the hands of black burned patients.
    - Melanin in the skin is synthesized in melanocytes. Melanocytes are present at the junction between the dermis and epidermis. After its production, melanin is

transported to the keratenocytes in the dermis through the melanosomes that are transported through the dendrites of the melanocytes.
    - Even if the melanocytes are intact, the presence of scar tissue will prevent the transport of meanosomes to the keratenocytes. This will result in permanent hypopigmentation.
    - This phenomenon is known as Koepner's phenomenon. It is different from the Koepner's phenomenon of psoriasis in which psoriatic patches will appear in a previously path free area after an incision.
    - Treatment: dermabrasion (removal of the superficial later which is the rough scar tissue. After the removal of that layer, a skin graft is placed to permit melanosomal transport). Medical tattooing or camouflage are alternatives.
- Post traumatic stress syndrome (PTSS):
    - This is a psychological disorder that burn patients have. It comes in the dorm of depression, insomnia, anxiety, nightmares, and low self esteem.
    - After those patients are discharged from the burn unit, many families report behavioral changes in these patients. Some of these patients will end with psychosis. This is why psychotherapy is an important part of the treatment of such patients.
- Late post-electric burn complications:
    - If elctricity passes through the CNS or the peripheral nerves, it may produce central or peripheral damage.
    - Central damage is apparent after 6 months. It manifests in the form of epilepsy, encephalopathy, and brain stem dysfunction. This might lead to an abnormal breathing pattern, arrhythmias, and vasomotor disturbances. If electricity passes through the spinal cord, it will produce ALS.
    - Peripheral damage is apparent after 6 months of injury. It manifests as peripheral neuropathy that affects the motor part more than any other part. This is due to the heavy myelination of the motor fibers.
    - If electricity passes through the eyes, patients will suffer from permanent cataracts. The lenses will lose their translucency and they will be opaque. This, if you look at the black part of the eye, you will be able to see white spots. The patients will present with a decreased level of vision.

Malignant Skin and Soft Tissue Lesions

Keith G. Wolter

I. SKIN EMBRYOLOGY
- A. Epidermis: Ectoderm
- B. Dermis: Mesoderm
- C. Other cells
  1. Melanocytes: Neural crest
  2. Merkel cells: Neural cells
  3. Langerhans cells: Mesenchymal

II. SKIN HISTOLOGY
- A. Epidermis
  1. **Keratinocytes**
     - a. Primary cell in epidermis
     - b. Start in basal layer (stratum germinativum or basale) and make their way to surface becoming a dead cornified layer (stratum corneum).
  2. **Melanocytes**
     - a. Found in basal layer
     - b. Protect against ultraviolet (UV) radiation
  3. **Merkel cells:** Mechanoreceptors
  4. **Langerhans cells:** Antigen-presenting cells in stratum spinosum
- B. Dermis
  1. **Cell types:** Fibroblast, macrophage, and mast cell
  2. **Papillary dermis**
     - a. Similar thickness to epidermis
     - b. High content of type III collagen, less type I
     - c. Site of collagenase activity.
     - d. Intertwines with the rete ridges of the epidermis.
     - e. Contains terminal networks of Meissner corpuscles and capillaries.
  3. **Reticular dermis**
     - a. Majority of the dermal layer
     - b. Mostly type I collagen bundles with elastic fibers between
     - c. Contains roots of the hair, sebaceous glands, sweat glands, receptors, nails, and blood vessels.
  4. **Tissue components**
     - a. **Collagen**
       - i. Tensile strength
       - ii. Type I to type III—4:1 ratio in adult skin
       - iii. Immature scar type I to type III—2:1 ratio in adult skin.
     - b. **Elastin**
       - i. Interdigitates with collagen
       - ii. Important in skin recoil and decreases with aging
       - iii. Composed of the protein fibrillin
     - c. **Ground substance**
       - i. Noncellular component of extracellular matrix with fibers
       - ii. Composed of glycosaminoglycans (hyaluronic acid and proteoglycans)

*Denotes common in-service examination topics

III. SKIN MALIGNANCIES

- **A. Generally grouped into three types** (listed from most common to least)
- **B. Basal cell carcinoma** (BCC)
- **C. Squamous cell carcinoma** (SCC)
- **D. Melanoma**
  1. The ratio of BCC to SCC to melanoma is ≈40:10:1
  2. Incidence of all three types is increasing; fortunately, the more common types (BCC and SCC) are far less aggressive than melanoma
  3. More than 20% of the US population develops a skin cancer during their lifetime
  4. Each year in the United States, there are more new cases of skin cancer than combined new cases of breast, prostate, lung, and colon cancers

BASAL CELL CARCINOMA (BCC)

I. EPIDEMIOLOGY

- **A. Incidence**
  1. BCC is the most common skin cancer, accounting for ≈80% of all skin cancers.
  2. Roughly 2.8 million new cases per year in the United States.
- **B. Risk factors**
  1. **Sun exposure** (increased with lower latitudes, high altitude): 36% of BCCs originate from the area of previously diagnosed actinic keratosis (AKs), but have distinct cells of origin.
  2. **Advancing age**
  3. **Fair complexion**
  4. **Long-term exposure to psoralens and UVA therapy** (i.e., PUVA therapy for psoriasis)
  5. **Immunosuppression**, most commonly seen in transplant patients
  6. **Nevus sebaceus of Jadassohn**, a superficial skin lesion typically in the head and neck regions, presents as an irregular, raised, yellow to pink, non–hair-bearing raised mass. They are usually present at birth or develop in early childhood, and approximately 15% undergo malignant transformation to BCC.
  7. **Arsenic exposure**
  8. **Syndromes associated with BCC**
     - **a. Basal cell nevus syndrome (Gorlin's syndrome)**
       - **i.** Autosomal dominant inheritance
       - **ii.** Multiple nevi/lesions often seen early in childhood with malignant degeneration more likely by the age of puberty.
       - **iii.** Skin pits on palms and soles, jaw cysts (odontogenic keratocysts), rib abnormalities, mental retardation
     - **b. Xeroderma pigmentosum (XP):** Patients have increased incidence of BCC, SCC, and malignant melanoma (see above in melanoma section)
     - **c. Albinism**

II. BCC DISEASE BIOLOGY AND CHARACTERISTICS

- **A. Basal keratinocytes** are the cell of origin, residing in the basal layer of the epidermis at the dermoepidermal junction.
- **B. No universal clinical precursor lesion**
- **C. BCC is most common** in areas with high concentrations of pilosebaceous follicles and thus >90% are found on the head and neck.
- **D. Metastasis** is rare—termed "barely a cancer" by some researchers
- **E. Morbidity** is caused by invasion of the tumor into underlying structures, including the sinuses, orbit, and brain. Typically, only a problem if neglected for many years.
- **F. Types of BCC**
  1. **Nodular BCC**
     - **a.** The most common type, usually presenting as a single lesion consisting of pearly papules with telangiectasias, pruritus, and occasional bleeding.
     - **b.** Lesion breakdown over time leads to nodulo-ulcerative BCC ("Rodent ulcer").
     - **c.** Histology demonstrates palisading nuclei.

2. **Superficial spreading BCC**
   a. Slow-growing, erythematous, with minimal induration, and located primarily on the trunk.
   b. It is easily confused with other scaly, eczematous dermatoses.
   c. The lesions are shallow with a characteristic horizontal growth pattern and often present in multiples.
3. **Morpheaform (sclerosing, fibrosing) BCC**
   a. Flat, often yellowish or hypopigmented, sometimes resembling scars or normal skin.
   b. The true extent of the lesion is usually greater than the clinical appearance.
   c. There is a high incidence of recurrence or incomplete excision due to "finger-like" extensions.
   d. Margins of 1 cm or Mohs extirpation is warranted.
4. **Pigmented BCC:** Similar to nodular BCC; easily confused with melanoma due to its deep pigmentation and nodularity
5. **Adnexal BCC**
   a. Uncommon and found in older individuals.
   b. Tumors arise from sweat glands, and although they exhibit slow growth, they are locally invasive, with a high incidence of local recurrence.

III. TREATMENT OF BCC

A. Standard surgical techniques: ≈95% cure rate

1. **Wide local excision** of BCC: **3- to 5-mm margins** for nonaggressive types and 7-mm margins for morpheaform type.
   a. Frozen sections may be used to confirm negative margins intraoperatively. False negatives are common. Surgeon must have confidence in pathologist/laboratory to use this modality.
2. **Mohs surgery:** Sequential horizontal excision with immediate frozen section testing by dedicated Mohs dermatopathologist
   a. ***Indications include morpheaform BCC and/or lesions in aesthetically sensitive areas (nose, eyelid, lip, etc.)**
   b. Advantages are tissue preservation and confirmation of complete excision.

B. Field therapies

1. Curettage and electrodessication can be used for BCC <1 cm that is NOT a recurrent disease or morpheaform type, but leads to a widened scar.
2. Cryotherapy is effective for small BCC over bone or cartilage, tip of nose, or around the eye.
3. Radiation is effective but requires multiple visits. High cure rates (≈90%), but recurrence is relatively common many years (10 to 15) later.

C. Topical Pharmaceuticals

1. **Imiquimod:** Immune stimulant. FDA-approved only for superficial BCCs, with cure rates between 80% and 90%. The 5% cream is applied 5 times per week for 6 weeks or longer.
2. **5-Fluorouracil (5-FU):** Chemotherapy. FDA-approved for superficial BCCs, with similar cure rates to imiquimod. Five percent liquid or ointment is rubbed onto the tumor 2 times per day for 3 to 6 weeks.

D. Adjuvant radiation therapy (after surgery): Useful for advanced, deeply invasive BCC

SQUAMOUS CELL CARCINOMA

I. EPIDEMIOLOGY

A. Incidence

1. Second most common skin cancer after BCC.
2. Roughly 700,000 new cases annually in the United States.

B. Risk factors

1. **UV radiation:** Sun exposure and tanning booth use; PUVA therapy for psoriasis
2. **Chemical exposure**, including some pesticides, organic hydrocarbons such as coal tar, fuel oil, paraffin oil, and arsenic (in welding materials)

3. **Viral infection:** Some types of human papillomavirus (HPV); herpes simplex virus
4. **Radiation:** Long latency between exposure and disease.
5. **Marjolin's ulcer:** SCC arising in a **chronic wound** (i.e., chronic burn scars and pressure sores) secondary to genetic changes caused by chronic inflammation.
6. **Impaired immunity:** That is, immunosuppression for transplants and AIDS. Ratio of SCC to BCC in these patients is 2:1.
7. **Fitzpatrick skin type**

II. SCC DISEASE BIOLOGY AND CHARACTERISTICS

A. Precursor lesions

1. **Actinic keratoses** (AKs, or solar keratoses)
   - **a.** Erythematous macules and papules with coarse, adherent scale
   - **b.** Histologically resembles SCC in situ (pre-malignant)
   - **c.** AK is considered a precursor lesion; up to 5% progress to SCC; in turn, 65% of all SCC arise from sites of AKs
2. **Bowen's disease** (SCC in situ)
   - **a.** Exhibits full-thickness cytologic atypia of the keratinocytes
   - **b.** Erythroplasia of Queyrat is SCC in situ of the glans penis.
3. **Leukoplakia**
   - **a.** Presents as a white patch on oral or other mucosa.
   - **b.** Malignant transformation occurs in 15%.
4. **Keratoacanthoma**
   - **a.** Benign skin tumor that is composed of squamous cells and keratin; may clinically resemble SCC.
   - **b.** Etiology is unknown but thought to originate from hair follicles.
   - **c.** Typically has a rapid 6-week growth phase followed by involution over the next 6 months. However, can progress to SCC in 5% to 10% of cases.
   - **d.** Excision is the treatment of choice; may be difficult to differentiate from SCC histologically.

B. Types of SCC

1. **Verrucous SCC:** Slow-growing, exophytic, and less likely to metastasize.
2. **Ulcerative SCC:** Grows rapidly and is locally invasive.
   - **a.** Ulcerative SCC has very aggressive growth characteristics, raised borders, and central ulceration.
   - **b.** <50% 5-year survival if spread to lymph nodes in the head and neck.
3. **Majorlin's ulcer**
   - **a.** Arise from chronic wounds (burn, pressure ulcer, fistula, osteomyelitis tracks)
   - **b.** Commonly metastasize to lymph nodes.

III. SCC TREATMENT OPTIONS

A. Standard surgical techniques: 90% to 95% cure rates; similar to BCC options

1. **Wide local excision** of SCC: 5- to 10-mm margins are usually sufficient. Frozen sections may be used to confirm negative margins intraoperatively.
   - **a.** If <2 cm, low grade and extends to dermis, 4-mm margin
   - **b.** If >2 cm, grade 2 to 4, high risk or extension into fat, 6-mm margin
2. **Mohs surgery:** Sequential horizontal excision with frozen section testing. Highest cure rate for SCC: 94% to 99%.
   - **a.** Indications, include recurrent, high-risk SCC, and/or lesions in aesthetically sensitive areas (nose, eyelid, lip, etc.)
   - **b.** Advantages are tissue preservation and confirmation of complete excision.

B. Field therapies

1. **Curettage, electrodessication, and cryotherapy** are used much less in SCC treatment than in BCC treatment, because of higher risk associated with missed deep tumor portions, and the risk of scarring obscuring SCC recurrences.
2. **Radiation** is reserved for unresectable lesions or for the very elderly. Cure rates vary widely. Cosmetic damage and long-term risk of radiation must be considered.
3. **Pharmaceuticals** are being investigated for topical application, but not currently recommended for invasive SCC.

C. Regional lymphadenectomy

1. **Indicated for** clinically positive (palpable) nodes.
2. **FNA:** Confirm spread of SCC to palpable lymph node.
3. **ELND:** Indicated for a tumor extending down to parotid capsule or a large lesion contiguous with a draining nodal basin.
4. **SLN biopsy:** Considered for high-risk SCC without palpable nodes (controversial).

D. Adjuvant radiation therapy: Used postexcision for high-risk cutaneous SCC.

MELANOMA

I. EPIDEMIOLOGY

A. Incidence is increasing, faster than any other cancer in Western world

1. 2% to 3% increase in incidence per year in the United States as of 2009.
2. 75,000 new cases predicted to be diagnosed in the United States in 2012.
3. Lifetime risk in general population is 2% for children born today.
4. Less than 3% of all skin cancers, but cause of 75% of skin cancer-related deaths.
5. Prognosis of metastatic disease has changed little in past 40 years (unlike many other cancers).

B. Risk factors

1. **Phenotypic** include fair skin (Fitzpatrick I and II) (Table 13-1), freckling, light eye color, and light hair color (stronger risk factor than eye color). Darker skin is protective against melanoma.
2. **Geographic:** High altitudes, lower latitudes have increased UV exposure, and therefore increased risk.
3. **Gender:** Females have lower risk and better prognosis; however, gender-based differences in risk are lessenings (Table 13-2). Lower extremity is the most common site in females; males more commonly have lesions on the head and trunk.

TABLE 13-1 Fitzpatrick Classification of Skin Type

	Skin phototype	Unexposed areas	Tanning history
I	Never tan, always burn	Pale/milky white	Red sunburn, painful swelling, skin peels
II	Sometimes tan, usually burn	Very light brown, sometimes freckles	Usually burn, pinkish or red coloring, light brown tan gradually develops
III	Usually tan, sometimes burn	Light tan, brown, and olive	Rarely burn with moderately rapid tanning response
IV	Always tan, rarely burn	Brown, dark brown, or black	Rarely burn with rapid tanning response

TABLE 13-2 Anatomic sites of cutaneous melanoma according to sex

Anatomic Site	Men		Women	
	Percent	Median age, y	Percent	Median age, y
Face	8.2	66	10.1	70
Scalp	5.1	64	2.0	61
Neck	2.2	57	1.6	56
Anterior trunk	16.3	55	7.7	45
Posterior trunk	39.3	55	17.1	48
Genital region	0.2	59	0.8	65
Upper extremity	12.2	58	18.4	59
Lower extremity	16.5	52	42.3	56

4. **Race:** Incidence is lower, but prognosis is worse for African-Americans, due to delayed diagnosis and/or worse disease subtype.
5. **Affluence:** Unlike most cancer types, higher socioeconomic status correlates with higher risk.
6. **History of UV radiation exposure** (both UVA and UVB): Evidence for direct causality is less clear than for other skin cancer types. A history of blistering sunburns, particularly in early life, correlates to increased risk of some melanoma types.
7. **Previous melanoma** is a strong predictive factor and confers a 3% to 5% chance of developing a second melanoma.
8. **Family history:** Vast majority of melanomas are sporadic; however, some hereditary forms exist (see also Genetics section below).
   a. **Familial melanoma** (aka hereditary melanoma): Two or more cases of melanoma in first-degree relatives may indicate familial melanoma, autosomal dominant transference with variable penetrance.
   b. **Dysplastic nevus syndrome** (also known as familial atypical multiple mole and melanoma [**FAMMM**] syndrome): Patients have a first- or second-degree relative with malignant melanoma and typically have at least 50 melanocytic nevi. Mutations in CDKN2A typical. Patients need vigilant screening.
   c. **Xeroderma pigmentosum** (XP)
      i. Heterogeneous group of syndromes; due mutations in various DNA repair genes.
      ii. DNA damage by UV leads to early death secondary to metastatic spread of skin tumors.
      iii. Typically presents in childhood with multiple BCCs; SCCs and melanomas typically cause death.
      iv. Restriction from sunlight exposure is mandatory, with aggressive surveillance/treatment of skin lesions.

II. MELANOMA DISEASE BIOLOGY AND CHARACTERISTICS

A. Precursor lesions

1. **Melanoma** is caused by multiple processes leading to malignant transformation of melanocytes.
2. **Congenital nevi**
   a. Malignant potential is more dependent on histology than on size.
   b. Giant hairy nevi: Confer a 5% to 20% lifetime risk of melanoma (difficult to predict risk accurately due to variability in size/location); prophylactic excision (often serially) is recommended
3. **Acquired melanocytic nevi**
   a. Typically appear at 6 to 12 months of age; usually <5 mm
   b. Increase in number through the fourth decade then slowly regress.
   c. The greater the number of nevi, the greater the chance of melanoma.
4. **Dysplastic or atypical nevi**
   a. Often appear in puberty
   b. Larger than common nevi (5 to 12 mm)
   c. Commonly found in covered areas
   d. May represent a precursor lesion and/or marker for increased risk for melanoma development.
5. **Melanoma in situ / atypical junctional melanocytic hyperplasia (AJMH)**
   Also termed "lentigo maligna"; Hutchinson freckle
   a. Melanoma precursor lesion; no penetration of atypical cells beyond epidermal junction.
   b. May arise within dysplastic nevi
   c. Needs to be fully excised; 5-mm margins are recommended, but re-excision is often needed.
6. **Spitz nevus**
   a. Benign lesion most commonly found in children and young adults (formerly called juvenile melanoma). NOT a melanoma precursor lesion.
   b. Presents as a well-circumscribed, raised lesion with variable pigmentation.

c. Despite the lack of malignant potential, it is very difficult to distinguish histopathologically from melanoma.
d. Recent data indicate that Spitz nevi have mutations in the *HRAS* gene, distinct from the BRAF/NRAS mutations seen in melanoma.

B. Genetic mechanisms

1. *p16/CDKN2A* gene: Tumor suppressor gene that is mutated or deleted in the majority of melanoma cell lines; mutations found in some familial melanomas.
2. *CDK4* gene: Cell cycle regulator-like CDKN2A; plays a role in melanoma progression in a small proportion of familial and sporadic melanomas.
3. *MC1R* gene: Pigmentation gene; certain isoforms correlate with fair skin/poor tanning ability as well as increased risk of melanoma.

C. Classification of melanoma types

1. **Superficial spreading melanoma**
   a. **Most common type,** ≈70% cases
   b. Intermediate in malignant potency
   c. Most likely to arise from a preexisting nevus
   d. Affects both genders equally
   e. Median age at diagnosis is 50 years
   f. Upper back in men and lower legs in women are most common sites
   g. Irregular, asymmetric borders with color variegation
   h. **Radial growth** phase early, vertical growth phase late
2. **Nodular melanoma**
   a. Second most common: 15% to 30% cases
   b. **Most aggressive type**
   c. Typically do not arise from preexisting nevi
   d. Men are affected twice as frequently as women
   e. Median age at diagnosis is 50 years
   f. No clear association with sunlight exposure
   g. Typically bluish-black, with uniform, smooth borders
   h. 5% are amelanotic—associated with a poorer prognosis because of delayed diagnosis
   i. **Vertical growth** phase is a hallmark feature; no radial growth
3. **Lentigo maligna melanoma (LMM)**
   a. 10% to 15% of cutaneous melanomas
   b. **Least aggressive type**
   c. Most clearly associated with sunlight/UV exposure
   d. Head, neck, and arms of elderly (sun-exposed areas) typically affected
   e. Women are affected more frequently than men
   f. The median age at diagnosis is 70 years
   g. Usually greater than 3 cm in diameter; irregular, asymmetric with color variegation, areas of regression may appear hypopigmented.
   h. **Precursor lesion is lentigo maligna** or Hutchinson freckle (histologically equivalent to melanoma in situ, or AJMH): radial growth phase only. Transition to vertical growth phase marks development of LMM.
   i. Malignant degeneration is characterized by nodular development.
4. **Acral lentiginous melanoma**
   a. 2% to 8% of melanomas in Caucasians, 35% to **60% of melanomas in African-Americans, Hispanics, and Asians**
   b. Presents in **palms, soles, and beneath nail plate (subungual)**. Must be distinguished from melanonychia, a benign, linear, pigmented streak in the nail, common in African and Asian populations. Due to the risk of melanoma, biopsy of suspect lesions should be performed.
   c. Median age at diagnosis is ≈60 years
   d. Irregular pigmentation, large size (>3 cm) common
   e. Most common site is great toe or thumb
   f. Long radial growth phase, transition to vertical growth phase occurs with high risk of metastasis.

D. Noncutaneous melanoma

1. **Mucosal melanoma**
   - **a.** Mucosal melanomas represent <2% of melanomas, most commonly presenting within the genital tract, anorectal region, and head and neck mucosal surfaces.
   - **b.** Difficult to detect; typically advanced at the time of diagnosis with poor prognosis.
   - **c.** Radical excision is of questionable benefit.
2. **Ocular melanoma**
   - **a.** Represent 2% to 5% of melanomas (most commonly noncutaneous melanoma)
   - **b.** Interference with vision leads to earlier diagnosis.
   - **c.** Melanomas of iris are similar to cutaneous melanomas in genetics/behavior; melanomas of the posterior uvea act more like mucosal melanomas and have a worse prognosis.
   - **d.** The eye has no lymphatic drainage; therefore, no nodal metastasis is seen
   - **e.** The liver is the main site of metastatic disease
   - **f.** Treatment is by enucleation

E. Melanoma with an unknown primary

1. Represent 3% of melanomas
2. Diagnosis is by exclusion
3. Nodal metastases are the most common presentation
4. Prognosis is similar to metastatic melanomas with a known primary.

III. DIAGNOSIS AND STAGING OF MELANOMA

A. Physical examination is only 60% to 80% sensitive for diagnosing melanoma. Full-body photography to monitor atypical nevi may increase sensitivity.

B. Common clinical features of melanoma lesions: (ABCDE)

1. Asymmetry
2. Border irregularity
3. Color variation
4. Diameter >6 mm
5. Enlarging/evolving lesion

C. Diagnosis of primary melanoma is made by histologic analysis of full-thickness biopsy specimens

1. **Excisional biopsy** is preferred for lesions <1.5 cm in diameter. If possible, excise lesion with 1- to 2-mm margins.
2. **Incisional biopsy** is appropriate when suspicion is low, the lesion is large (>1.5 cm) or is located in a potentially disfiguring area (face, hands, and feet), or when it is impractical to perform complete excision. Incisional biopsy does not increase risk of metastasis or affect patient survival.
3. **Permanent sectioning** is used to determine tumor thickness
4. **Avoid shave biopsies**, since they forfeit the ability to stage the lesion based on thickness.
5. **Do not cauterize or freeze** the specimen: Tissue destruction makes it impossible to evaluate thickness and margins.
6. **Wide local excision** for tissue diagnosis can decrease the efficacy of future lymphatic mapping because of disruption of local lymphatics. Biopsy incisions should result in scars parallel to lymphatic drainage.
7. **Orientation of biopsy** incisions should also take definitive surgical therapy into consideration.
   - **a.** Extremity biopsies should use longitudinal incisions.
   - **b.** Transverse incisions are sometimes preferable for preventing contractures over joints.
   - **c.** Head and neck incisions should be placed within relaxed skin tension lines, keeping facial aesthetic units in mind.

D. Major prognostic factors: Tumor thickness, **N**odal status, and **M**etastases—TNM (Table 13-3)

1. **Breslow thickness** is reported in millimeters; thus, it is more accurate and reproducible than Clark level and is a better prognostic indicator.

TABLE 13-3 Melanoma Thickness Grading

Clark level	Skin layer/depth	5-y survival (%)
I	In situ	100
II	Papillary dermis	88
III	Papillar-reticular dermis	66
IV	Reticular dermis	55
V	Subcutaneous	22
Breslow depth (mm)		**5-y survival (%)**
<0.76		89
0.76–1.49		75
1.5–2.49		58
2.5–3.99		46
>3.99		25

2. **Clark level** is based on invasion through the histologic layers of the skin; more subjective.

E. Other significant prognostic factors

1. **Anatomic location:** Trunk lesions generally carry worse prognosis than those on the extremities.
2. **Sex:** For a given melanoma, women generally have a better prognosis; women are also more likely to have extremity melanomas which carry a better prognosis.
3. **Ulceration** is a poor prognostic sign
4. **Lymph node involvement** or in-transit metastases are more significant than any other prognostic factors.

F. The American Joint Committee on Cancer has developed a staging system based on TNM classification (Table 13-4)

IV. MELANOMA TREATMENT

A. Definitive management of melanoma

1. Wide local excision is the treatment of choice.
2. Recommended surgical margins depend on tumor thickness (Table 13-5)
3. Subungual melanoma requires amputation proximal to the DIPJ for fingers and proximal to IP joint for the thumb.

B. Management of regional lymph nodes

1. **Elective lymph node dissection (ELND)** involves removal of clinically negative lymph nodes from the nodal basin. No prospective survival benefit was seen except for a subgroup with 1- to 2-mm (intermediate thickness) melanomas.
2. **Sentinel lymph node biopsy (SLNB)**
   a. In the sentinel node theory, a sentinel node will be the first lymph node seeded by tumor cells, and therefore, excision of sentinel node(s) alone is adequate to determine nodal status. The morbidity of SLNB is considerably less than ELND. Sentinel node(s) can be detected in >90% to 95% of patients. SLNB is now widely considered the standard of care.
   b. SLNB is performed in conjunction with wide local excision of the primary tumor. Lymphatic mapping is performed to determine the first lymph node that drains the primary tumor site (sentinel node).
   c. SLNB-positive patients undergo staged regional lymphadenectomy and may be candidates for adjuvant therapy.
   d. Preoperative nuclear imaging is performed with radiolabeled colloid solution (technetium-99) injected intradermally at the primary tumor. This can be done on the day of or day prior to surgery. Lymphoscintigraphic imaging localizes the sentinel node basin(s) (some tumor sites can drain to multiple basins).

TABLE 13-4 AJCC Melanoma Staging System (1998)

TNM DEFINITIONS	
Primary Tumor	
Tx	Unknown, cannot be assessed
T0	No evidence of primary tumor
Tis	Melanoma in situ (AJMH, Clark II)
T1	<0.75 mm (Clark II)
T2	0.76–1.50 mm (Clark III)
T3	1.51–4 mm (Clark IV)
T4	>4 mm or satellitosis within 2 cm of primary (Clark V)
Regional Lymph Node Involvement	
NX	Unknown, cannot be assessed
N0	Negative
N1	Metastasis 3 cm or less in greatest dimension in any regional lymph node(s)
N2	Metastasis >3 cm in greatest dimension in any regional lymph node(s) and/or in-transit metastasis
Distant Metastasis	
MX	Unknown, cannot be assessed
M0	No distinct metastasis
M1	Distant metastasis
STAGING	
Stage 0	Tis, N0, M0
Stage I	T1, N0, M0
	T2, N0, M0
Stage II	T3, N0, M0
Stage III	T4, N0, M0
	Any T, N1, M0
	Any T, N2, M0
Stage IV	Any T, Any N, M1

e. In the operating room, a lymphangiography dye (lymphazurin or methylene blue) can be injected intradermally at the periphery of the primary tumor site prior to excision of the primary tumor.

i. Mark edges of the lesion before injection to avoid obscuring them with the dye and take care with the dye because spills are difficult to manage.

TABLE 13-5 Recommended Surgical Margins for Melanoma Excision

Melanoma thickness (mm)	Margin (cm)
In situ	0.5
<1	1
1–4	2
>4	2–3 (controversial)

ii. Potential sentinel nodes will appear blue when exploring the nodal basin, giving secondary confirmation to localization with Geiger counter detection of Tc^{99}.
iii. Dye injection may briefly interfere with pulse-oximeter readings; alert anesthesiologist at the time of injection.
iv. Caution: Risk of allergy or anaphylaxis with dye injection

f. Following excision of the primary tumor, drapes, instruments, gowns, and gloves are changed and regional lymph node basin(s) identified by lymphoscintigraphy are explored. All radioactive ("hot") and/or blue nodes are excised.
g. Histologic analysis of sentinel node with immunohistochemical staining identifies micrometastases. Permanent sections are required; frozen sections cannot reliably differentiate normal from neoplastic melanocytes.

C. Surveillance and treatment of melanoma recurrence

1. **Asymptomatic patients** should be seen every 3 to 4 months for 2 years, then every 6 months for 3 years, and then annually. The most accurate way to detect metastatic disease is to take a thorough history.
2. **Chest X-ray and liver function tests** (LDH and alkaline phosphatase) are usually sufficient; more extensive work-ups including CT scans have not altered outcomes.
3. **Local recurrences** typically occur within 5 cm of the original lesion, usually within 3 to 5 years after primary excision; most often this represents incomplete excision of the primary tumor.
4. **The most common sites** of recurrence are the skin, subcutaneous tissues, distant lymph nodes, then other sites (lung, liver, brain, bone, GI tract).
5. **Re-excision** is the primary treatment for local, small, isolated lesions
6. **Surgery is effective for palliation** in patients with isolated recurrences in skin, CNS, lung, or GI tract.
7. **Chemotherapy:** Complete remission is rare. Decarbazine (DTIC), carmustine, cisplatin, and tamoxifen in combination are most frequently used. Isolated hyperthermic limb perfusion for extensive extremity cutaneous disease (melphalan and tumor necrosis factor) is used at some centers.
8. **Cytokine therapy** has been demonstrated to produce relatively high levels of tumor response, albeit transient. FDA-approved regimens include interferon-α (IFN-α) for stage III disease and interleukin-2 (IL-2) for stage IV disease; however, these therapies demonstrate little or no improvement in overall survival.
9. **Immunotherapies** with **monoclonal antibodies, tumor vaccines,** and **modified immune cells** have been the subject of active investigation for several decades. Despite a number of dramatic successes, these modalities have yet to prove applicable.
10. **Selective cell-signaling inhibitors** (e.g., vemurafenib) have recently been developed and can produce dramatic tumor responses in appropriately chosen patients. Increases in survival time, however, do not translate to improve overall survival, as resistant melanomas return with added aggressiveness.
11. **Mean survival with disseminated disease** is 6 months. Respiratory failure and CNS complications are the most common causes of death.

LESS COMMON SKIN CANCERS

A. Merkel cell carcinoma (MCC)

1. Rare, malignant neuroendocrine tumor arising within the dermis from cells of neural crest origin.
2. Incidence is increasing for unknown reasons; 1,500 cases per year in the United States.
3. Risk factors include age over 65; history of extensive sunlight exposure; fair skin; and immunosuppression (HIV; organ transplants).
4. Recent research has implicated Merkel cell polyomavirus in 80% of MCC cases.

5. Presents as a purple to red papulonodule or indurated plaque; 50% involve the head and neck, 40% the extremities, and 10% the trunk.
6. MCC is aggressive, with radial spread, high local recurrence, and regional and systemic metastasis.
7. Treatment involves local excision with wide (up to 3 cm) margins; SLN biopsy, and postoperative radiation started several weeks later.
8. Poor prognosis; 50% survival at five years.

B. Microcystic adnexal carcinoma

1. Pathophysiology is subject of debate, but many authors support dual follicular and eccrine differentiation.
2. Tumor is invasive and locally destructive.
3. Presents as a white to pink papule–plaque primarily on the head and neck.

C. Sebaceous gland carcinoma

1. Malignant tumor derived from adnexal epithelium of sebaceous glands.
2. Most are periocular; sebaceous gland carcinomas elsewhere are vanishingly rare.
3. Yellowish to pink, slowly growing papulonodule on eyelid (resembles chalazion).

SOFT TISSUE SARCOMAS

I. EPIDEMIOLOGY

A. 6,000 to 7,000 new cases are diagnosed annually in the United States

B. 1% of all malignancies in adults and 15% of those in children

C. 50% are located in the extremities

D. Risk factors

1. **The majority of sarcomas** have no identifiable predisposing genetic or environmental cause.
2. **Radiation exposure**
   a. Associated with osteosarcomas and malignant fibrous histiocytomas.
   b. Typically, there is a 10- to 20-year latency period after exposure.
   c. Thorium dioxide (Thorotrast) is a contrast agent used in 1940 to 1950s for radiologic procedures; linked with a high incidence of hepatic angiosarcoma.
3. **Chemical exposure:** Arsenic, vinyl chloride, and dioxin (contained in the Vietnam War era defoliant Agent Orange)
4. **Genetic factors**
   a. Neurofibromatosis (von Recklinghausen syndrome): 5% lifetime risk of developing neurofibroma or neurofibrosarcoma
   b. Mutation in *Rb1* tumor suppressor gene: Retinoblastoma (sarcoma of the eye)
   c. Mutation in *p53* tumor suppressor gene: Li–Fraumeni syndrome (variety of sarcomas)
5. **Lymphedema**
   a. Following surgical procedures, radiation therapy, or parasitic infection; may also arise idiopathically.
   b. 10- to 20-year latency for the development of lymphangiosarcoma.
6. **Kaposi's sarcoma:** Strongly associated with HIV infection.

II. DIAGNOSIS

A. In contrast to ectoderm-derived carcinomas, sarcomas behave in a similar fashion regardless of the cell of origin.

B. Paucity of local symptoms often leads to advanced disease at diagnosis.

C. A pseudocapsule forms as the tumor expands and compresses adjacent tissue.

D. Major fascial planes typically act as barriers to local invasion.

E. Extremity sarcoma: Generally painless. Delay in diagnosis is common and patients are often erroneously treated for a hematoma or "pulled muscle"

1. Suspicious findings include: Mass >5 cm, enlarging or symptomatic mass, mass present for >4 weeks.
2. MRI is the preferred imaging modality.
3. Pulmonary metastases are the most common location for metastatic disease.
4. Approximately 75% 5-year survival rate

F. Sarcoma of the abdomen or retroperitoneum

1. **Can present** with vague abdominal complaints: Fullness, early satiety, pain, weight loss, nausea, and vomiting
2. **Metastatic disease:** Most commonly to liver
3. **Palpable mass** in 80% of patients at the time of presentation
4. **Median survival**
   a. Primary disease—72 months
   b. Recurrent disease—28 months
   c. Metastatic disease—10 months
5. **Imaging**
   a. MRI with gadolinium contrast: Best technique for visualizing tumor and relationship to adjacent structures.
   b. CT scan: Valuable for evaluating chest/abdomen/pelvis for metastatic disease and as a staging tool.
   c. Angiography: For surgical planning
   d. Chest X-ray: Evaluates for pulmonary metastasis
6. **Biopsy** of sarcomas: Performed for extremity lesions <5 cm

III. CLASSIFICATION AND STAGING

A. Subtypes are named for the cell of origin (Table 13-6). Fibrosarcoma is the most common sarcoma in adults and the second most common in children.

B. Histologic type has little prognostic significance; histologic grade (including frequency of mitotic figures, cellular atypia, and presence or absence of tumor necrosis) is the best for prognosis and therapy.

C. Staging criteria (Table 13-7)

1. **Histologic grade is the most important prognostic indicator** (see above). Low-grade tumors have less than a 15% chance of metastasis; high-grade tumors metastasize in >50%
2. **Tumors of larger size are more difficult to grade** and have a greater chance of recurrence and dedifferentiation.
3. **Nodal and distant metastases** are associated with a similar prognosis and are classified as stage IV disease.
4. **Five-year survival** is on the order of 80% for stage I disease, 60% for stage II, 35% for stage III, and <10% for stage IV

IV. SARCOMA MANAGEMENT

A. Extremities (especially the thighs) are the most common sites for sarcoma

1. **Surgery**
   a. Complete resection with negative margins is the mainstay of treatment.
   b. The pseudocapsule should not be entered.
   c. Wide local excision (WLE) is the standard of care, with **3- to 5-cm margins** of normal tissue proximally and distally. En bloc resection of uninvolved fascial plane with tumor is performed for control of the other margins.
   d. WLE is performed after excisional biopsy even if the margins are clear.

TABLE 13-6 Tissue Classification of Soft Tissue Sarcomas

Tissue of origin	Benign soft tissue tumor	Malignant soft tissue tumor
Fat	Lipoma	Liposarcoma
Fibrous tissue	Fibroma	Fibrosarcoma
Smooth muscle	Leiomyoma	Leiomyosarcoma
Skeletal muscle	Rhabdomyoma	Rhabdomyosarcoma
Cartilage	Chondroma	Chondrosarcoma
Bone	Osteoma	Osteosarcoma
Blood vessel	Hemangioma	Angiosarcoma

TABLE 13-7 AJCC "GTNM" Classification and Stage Groupings of Soft Tissue Sarcomas

Stage	Description
Histologic grade	
G1	Well-differentiated
G2	Moderately well-differentiated
G3	Poorly differentiated
G4	Undifferentiated
Primary tumor size	
T1	Tumor ≤5 cm in greatest diameter
T2	Tumor >5 cm in greatest diameter
Regional lymphatic involvement	
N0	No known metastases to lymph nodes
N1	Verified metastases to lymph nodes
Distant metastasis	
M0	No known distant metastases
M1	Known distant metastases

Stage	Groupings	5-y survival (%)
IA	G1, T1, N0, M0	80
IB	G1, T2, N0, M0	
IIA	G2, T1, N0, M0	60
IIB	G2, T2, N0, M0	
IIIA	G3-4, T1, N0, M0	35
IIIB	G3-4, T2, N0, M0	
IVA	Any G, any T, N1, M0	<10
IVB	Any G, any T, N1, M1	

e. Major neurovascular structures are generally preserved for low-grade lesions, but are sacrificed and reconstructed as needed for high-grade tumors.

f. There is no survival benefit of amputation compared to limb-sparing procedure.

2. **Radiation therapy** is not indicated for small (<5 cm) low-grade tumors due to excellent prognosis with WLE alone. It can be used as primary therapy for patients who cannot tolerate or refuse surgery and is also useful as combination therapy for sarcomas up to 10 cm.
3. **Chemotherapy** is of undetermined benefit in soft tissue sarcoma.

B. Retroperitoneal and intra-abdominal sarcomas have a uniformly poor prognosis. Excision with tumor-free margins is curative, but difficult to achieve. Radiation is rarely used because surrounding organs cannot tolerate therapeutic doses.

PEARLS

1. SCC commonly affects the lower lip and upper eyelid; BCC characteristically affects the upper lip and lower eyelid.
2. Spitz nevus: Looks like melanoma (even under a microscope) but does not metastasize; if it spreads, then it is not a Spitz nevus after all!
3. Perform full-thickness biopsies of pigmented lesions (i.e., punch or excisional) rather than shave biopsy or curettage so that the depth of the lesion can be determined whether it is a melanoma.
4. Merkel cell carcinoma has increasing incidence and behaves much like an aggressive melanoma; unlike melanoma, Merkel cell tumors respond to radiotherapy.
5. Fibrosarcoma is generally not sensitive to chemotherapy or radiation therapy.

QUESTIONS YOU WILL BE ASKED

1. Should you undermine the wound arising from excision of a suspected malignant skin lesion to facilitate wound closure?

   No. It will permit spread of malignancy.

2. A 35-year-old breast augmentation patient also mentions a 5-mm, nonhealing wound on the face that has been present for two months. What do you recommend?

   Immediate biopsy.

Recommended Readings

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Netscher DT, Leong M, Orengo I, Yang D, Berg C, Krishnan B. Cutaneous malignancies: melanoma and nonmelanoma types. *Plast Recon Surg*. 2011;127:37E.

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Benign Skin Lesions

Shailesh Agarwal

I. EMBRYOLOGY

- **A. Ectoderm:** Epidermis, pilosebaceous glands, apocrine glands, eccrine sweat glands, nails
- **B. Mesoderm:** Langerhans cells, macrophages, mast cells, Merkel cells, fibroblasts, blood vessels, lymph vessels, fat cells
- **C. Neuro-ectoderm:** ***Melanocytes, nerves, specialized sensory receptors**

II. ANATOMY

- **A. Epidermis—outer layer**
  1. **Cell types:** Keratinocytes, melanocytes, Langerhans cells, Merkel cells
  2. **Superficial to deep:** Stratum corneum, lucidum, granulosum, spinosum, basale
- **B. Dermis**
  1. **Cell types:** Collagen, elastin, ground substance
  2. **Nerves,** blood vessels, lymphatics, muscle fibers, pilosebaceous/apocrine/eccrine glands
  3. **Two layers superficial to deep**
     - **a. Papillary**—fibroblasts, mast cells, histiocytes, Langerhans cells, lymphocytes
     - **b. Reticular**—thicker than papillary dermis
       - **i.** Extends to underlying fat
       - **ii.** Contains elastin with interspersed large collagen fibers.

III. BENIGN LESIONS

- **A. Epidermal lesions**
  1. **Epidermal nevus** (linear nevus)
     - **a.** May be associated with developmental abnormalities. Ocular, central nervous, skeletal cardiovascular, urogenital systems
     - **b.** Present at birth or early childhood.
     - **c.** Clinical presentation: Tan or brown warty papules
     - **d.** Anatomic location: Extremities
     - **e.** Treatment: Excision, laser therapy(CO_2), dermabrasion, or cryotherapy
  2. **Inflammatory linear verrucous epidermal nevus**
     - **a.** Present at birth or early childhood.
     - **b.** Clinical presentation: Erythematous, rough, scaly papules in linear array, extremely pruritic
     - **c.** Anatomic location: Extremities
     - **d.** Treatment: Excision or laser therapy (intense pulsed light)
  3. **Seborrheic keratosis**
     - **a.** Derived from basal layer of epidermis. Cystic inclusions of keratinous material
     - **b.** Present in middle age around fifth decade.
     - **c.** Clinical presentation: Waxy, stuck-on appearance; warty papule or plaque May be yellow, light brown, dark brown, or black in color
     - **d.** Anatomic location: Head, neck, and trunk
     - **e.** Treatment: Dermabrasion, cryotherapy, shaving, and excision

*Denotes common in-service examination topics

4. **Actinic keratosis**
   - **a.** Occurs on sunlight-exposed skin
   - **b.** Most common pre-malignant skin lesion
     - **i.** ***Approximately 5% to 20% will develop into squamous cell carcinoma**
     - **ii.** May be present in transplant patients
     - **iii.** Require aggressive treatment due to high risk of malignant transformation.
   - **c.** Clinical presentation: Erythematous, rough, or scaly macules or papules
   - **d.** Anatomic location: Most commonly located on sunlight-exposed areas (scalp, ears, face, and hands)
     - **i.** Actinic chelitis (aggressive form involving lips)
     - **ii.** Histologically characterized by dyskeratosis, atypia in basal layer of epidermis
   - **e.** Treatment: Imiquimod 5% (Aldara) or 5-fluorouracil, cryotherapy, topical tretinoin
5. **Verruca vulgaris**
   - **a.** Common wart: Caused by human papillomavirus (HPV)
   - **b.** Clinical presentation: Scaly, rough appearance with a cap of friable keratotic material
   - **c.** Anatomic location: Variable. Lesions arise from stratum granulosum
   - **d.** Treatment: Cryotherapy, chemical ablation, or excision
6. **Cutaneous horn**
   - **a.** Clinical presentation: Well-circumscribed cone with hyperkeratotic features.
     - **i.** Resemble actinic keratoses
     - **ii.** Must be distinguished from squamous cell carcinoma
   - **b.** Anatomic location: Variable
   - **c.** Treatment: Excisional biopsy with careful evaluation of lesion base
7. **Leukoplakia**
   - **a.** Associated with chronic inflammation/irritation (Alcohol or tobacco)
   - **b. *May degenerate into SCC**
   - **c.** Clinical presentation: Mucosal lesion
     - **i.** White plaque exists on stratified squamous epithelium
     - **ii.** Cannot be wiped away
   - **d.** Anatomic location: Mucosal surface
   - **e.** Treatment: Removal of irritant, biopsy may be warranted
8. **Keratoacanthoma**
   - **a. *Rapid growth phase followed by spontaneous regression**
   - **b.** Clinical presentation: Firm, dome-shaped nodule
     - **i.** Prominent horn-filled central depression
     - **ii.** Keratin with thick epidermis
   - **c.** Difficult to distinguish from SCC
   - **d.** Treatment: Simple excision; may consider 5-fluorouracil for patients with multiple lesions.

B. Melanocytic lesions

1. **Nevus of Ota**
   - **a.** Found in patients with Asian ancestry
   - **b.** Appears at birth
   - **c.** Clinical presentation: Appears as large, blue-gray patch
   - **d.** Anatomic location: Areas innervated by first and second branches of trigeminal nerve
   - **e.** Treatment: Laser therapy (Q-switched Nd:YAG)
2. **Nevus of Ito**
   - **a.** Found in patients with Asian ancestry
   - **b.** Appears at birth
   - **c.** Clinical presentation: Appears as large, blue-gray patch
   - **d.** Anatomic location: Posterior shoulder and areas innervated by posterior supraclavicular and lateral cutaneous brachial nerves
   - **e.** Treatment: Laser therapy (Q-switched Nd:YAG)

3. **Nevus spilus**
   - **a.** Appears at birth
   - **b.** Clinical presentation: Tan patch with speckled hyperpigmented macules and papules
   - **c.** Anatomic location: Commonly on trunk
   - **d.** Treatment: Observation, laser therapy (intense pulsed light), or simple excision
4. **Spitz nevus** (benign juvenile melanoma)
   - **a.** Appears in childhood or early adulthood
   - **b.** Clinical presentation: Pink or tan, dome-shaped, smooth plaque
   - **c.** Anatomic location: Commonly located on face
   - **d.** Treatment: Excision with margins to decrease recurrence risk (range from 1 to 2 mm to 1 to 2 cm depending on concern for melanoma)
   - **e.** May be difficult to distinguish histologically from malignant melanoma
5. **Junctional nevus**
   - **a.** Nevus cells located at epidermal-dermal junction
   - **b.** Appears in childhood or early adulthood
   - **c.** Clinical presentation: Brown, evenly pigmented macule with well-defined borders
   - **d.** May be difficult to differentiate from melanoma
   - **e.** Anatomic location: Most commonly on trunk
   - **f.** Treatment: Simple excision
6. **Compound nevus**
   - **a.** Contains both junctional and intradermal components.
   - **b.** Appears in childhood or early adulthood.
   - **c.** Clinical presentation: Appears a dark-brown papule with regular borders
   - **d.** Anatomic location: Most commonly on trunk
   - **e.** Treatment: Simple excision
7. **Intradermal nevus**
   - **a.** Located entirely within the dermis.
   - **b.** Appears in the second or third decade of life
   - **c.** Clinical presentation: Appears as a flesh-colored or light tan papule
   - **d.** Anatomic location: Face or neck
   - **e.** Treatment: Simple excision
8. **Common blue nevus**
   - **a.** Appears during adolescence
   - **b.** Clinical presentation: Blue or blue-black papule
   - **c.** Anatomic location: Head, neck, and dorsum of hands/feet
   - **d.** Treatment: Simple excision
   - **e.** Cutaneous metastasis of malignant melanoma can resemble blue nevus
9. **Cellular blue nevus**
   - **a.** Appears after second decade of life
   - **b.** Clinical presentation: Blue-black papule
   - **c.** Anatomic location: Most commonly on buttocks
   - **d.** Treatment: Simple excision
10. **Atypical (dysplastic) nevus**
    - **a.** Patients with dysplastic nevi and a family history of melanoma in a first-degree relative are at a high risk of melanoma.
    - **b.** Regular skin examination
    - **c.** Appear after puberty
    - **d.** Clinical presentation: Appears as a central brown macule with irregular pink rim.
    - **e.** More irregular pigmentation and borders compared with typical nevi.
    - **f.** Anatomic location: Trunk
    - **g.** Treatment: Excision with margins to prevent recurrence
      - **i.** Total body skin examination to rule out other lesions
      - **ii.** Sunscreen and avoidance of sunburning/tanning

C. Adnexal tumors

1. **Background**
   - **a.** Excised for aesthetic reasons
   - **b.** Normal relationship between epithelial and stromal components of skin altered

- **c.** May be classified as nevus, adenoma, or epithelioma
- **d.** May include sebaceous glands, hair follicles, apocrine, or eccrine sweat glands

2. Hair follicle tumors

- **a.** Located in lower dermis and subcutaneous fat
- **b. Pilomatrixoma** (calcifying epithelioma of Malherbe)
  - **i.** Typically seen in younger patients (<20 years old)
  - **ii.** Clinical presentation: Single, solid subdermal nodule
    - **a)** Positive tent sign—stretching of overlying skin yields multiple peaks
    - **b)** Difficult to distinguish from calcified masses or carcinoma.
    - **c)** On pathology shows epidermoid cells with basophilic and eosinophilic cells
  - **iii.** Anatomic location: Involves face and upper extremities
  - **iv.** Treatment: Excision (with up to 1 to 2 cm margins) with up to 10% recurrence rate
- **c. Trichofolliculoma** (hair follicle nevus)
  - **i.** Clinical presentation: <1 cm and skin-colored
  - **ii.** Anatomic location: On face with thin pale hairs
  - **iii.** Treatment: Excisional biopsy for management
- **d. Trichoepithelioma**
  - **i.** Involves patients after puberty
  - **ii.** Rasmussen syndrome is an autosomal dominant disorder that is a triad of multiple trichoepitheliomas, cylindromas, and milia.
  - **iii.** Clinical presentation: Appears pink or flesh-colored
  - **iv.** May be difficult to distinguish clinically and histologically from basal cell carcinoma
  - **v.** Anatomic location: Multiple trichoepitheliomas may have symmetric distribution around face and eyes
  - **vi.** Treatment: Electrodesiccation
- **e. Trichilemmoma**
  - **i.** Cowden disease (multiple hamartoma syndrome) should be suspected if patients have multiple such tumors
  - **ii.** Glycogen-rich epithelial cells surrounded by sheaths of cells resembling hair follicles on histology
  - **iii.** Clinical presentation: Smooth papule
  - **iv.** Anatomic location: Found on scalp or other hair-bearing regions. Association with nevus of Jadassohn if on scalp. Warrants biopsy
  - **v.** Treatment: Laser therapy (CO_2), electrodessication with curettage, or simple excision due to similar appearance with BCC and trichilemmal carcinoma

3. Eccrine tumors

- **a.** Cylindroma (turban tumor or tomato tumor)
  - **i.** Appears in early adulthood
  - **ii.** Multiple cylindromas may indicate autosomal dominant cylindroma syndrome.
  - **iii.** Clinical presentation: Appears as firm, smooth pink nodules
  - **iv.** Anatomic location: Often located on scalp
  - **v.** Treatment: Laser therapy (CO_2), electrodessication/curettage, cryotherapy, or simple excision
- **b. Eccrine poroma**
  - **i.** Clinical presentation: Firm, papular or nodular lesions surrounded by rim of hyperkeratosis; may appear pedunculated
  - **ii.** May resemble amelanotic melanoma and pyogenic granuloma
  - **iii.** Anatomic location: Found on palms and soles of feet
  - **iv.** Treatment: Simple excision
- **c. Syringoma**
  - **i.** Appears in early adulthood
  - **ii.** May have increased incidence with Down's syndrome
  - **iii.** Clinical presentation: Small papules ranging from yellow to pink in color. May be confused with xanthelasma or trichoepithelioma

iv. Anatomic location: Most commonly appears in the periocular region (eyelids, upper cheek) but may involve trunk, neck, or extremities
v. Treatment: Laser (CO_2) or electrodesiccation

d. Eccrine spiradenoma
i. Appears in young adults
ii. Clinical presentation: Tenderness or pain with manipulation
iii. May be mistaken for glomus tumor
iv. Anatomic location: Appears as a single nodule on ventral upper half of body
v. Treatment: Simple excision if symptomatic

e. Eccrine hidrocystoma
i. Dilated and obstructed sweat ducts histologically
ii. Clinical presentation: Translucent vesicles
iii. Swell in heat/humidity; regress in cooler/dry climate
iv. Anatomic location: Appears on lower eyelids and upper cheeks
v. Treatment: Puncture to release pressure

4. Sebaceous tumors
a. Sebaceous nevus of Jadassohn
i. Appears at birth
ii. ***After puberty, 10% to 15% degenerate into BCC.** May also develop SCC or keratoacanthoma
iii. Clinical presentation: Appears as yellow/orange, waxy, smooth plaques prior to puberty
iv. Appear as rough, verrucous, orange plaques after puberty
v. Anatomic location: Most commonly found on scalp
vi. Treatment: Excision

b. Sebaceous hyperplasia
i. Appears in middle or late age
ii. Clinical presentation: Appears as shiny, small umbilicated, yellow-white papules
iii. May be covered with telengiectasia
iv. Anatomic location: Most common on face
v. Treatment: Cryotherapy, electrodesiccation, or laser (intense pulsed light or CO_2)
vi. May be excised due to similar appearance with BCC.

c. Sebaceous adenoma
i. Appears in middle age
ii. ***May be associated with Muir-Torre syndrome—an autosomal dominant syndrome associated with multiple keratoacanthomas, marked increase in visceral neoplasm**
iii. Clinical presentation: Yellow nodules
iv. Anatomic location: Located primarily in head and neck
v. Treatment: Simple excision

5. Apocrine tumors
a. Apocrine cystadenoma
i. Contains brown or blue tinged fluid
ii. Clinical presentation: Appears as a single translucent nodule
iii. Anatomic location: Most common on face

b. Chondroid syringoma
i. Composed of sweat gland (epithelial) and cartilaginous elements (mesenchymal) on histology
ii. Treatment: Excisional biopsy

c. Syringocystadenoma papilliferum
i. Appears during childhood
ii. Clinical presentation: May be associated with nevus sebaceous
iii. Nearly 10% will harbor BCC
iv. Anatomic location: Most commonly found on scalp
v. Treatment: Excision

D. Smooth muscle tumor

1. **Leiomyoma**
   - **a.** Abnormal proliferation of smooth muscle
   - **b.** May become symptomatic with pain on exposure to cold/pressure
   - **c.** Clinical presentation: Appears as firm, pale intradermal nodules with brown hue
   - **d.** Treatment: Excisional biopsy
   - **e.** Local recurrence may occur
   - **f.** Malignant degeneration to leiomyosarcoma is rare

E. Cysts

1. **Epidermal inclusion cyst** (epidermoid cyst)
   - **a.** May be incorrectly called a sebaceous cyst; however, not sebaceous in origin
   - **b.** Appears in adulthood
   - **c.** Clinical presentation: Fluctuant, flesh-colored, well-circumscribed nodules
     - **i.** Punctum may be visible
     - **ii.** Contains foul-smelling keratinous debris
   - **d.** Anatomic location: Commonly found on face, neck, and trunk
   - **e.** Treatment: Simple excision if uninfected; if infected, perform incision and drainage with interval excision
2. **Dermoid cyst**
   - **a.** Appears at birth or early childhood
   - **b.** Clinical presentation: Similar to epidermal inclusion cysts. Lined with epidermal skin appendages
   - **c. *Anatomic location: Most commonly found along supraorbital ridge, lateral brow, or nasal midline**
   - **d.** Treatment: Excision
   - **e. *Midline nasal mass differential diagnosis**
     - **i. Dermoid cyst, glioma, meningocele/encephalocele**
     - **ii. CT or MRI prior to excision to determine intracranial extension**
3. **Trichilemmal cyst**
   - **a.** Pilar cyst
   - **b.** Appear in adulthood
   - **c.** Clinical presentation: Similar to an epidermal inclusion cyst
   - **d.** Anatomic location: Most commonly found on scalp
   - **e.** Treatment: Excision if uninfected; if infected, incision and drainage with interval excision

F. Fibrous Lesions

1. **Dermatofibroma**
   - **a.** Appears in adulthood
   - **b.** Clinical presentation: Brown-red indurated papule or nodule Positive dimple sign—when squeezed it sinks
   - **c.** Anatomic location: Most common lower extremities
   - **d.** Treatment: Simple excision
2. **Angiofibroma**
   - **a.** Clinical presentation: Pale, firm papule. May have telengiectasia or erythema
   - **b.** Anatomic location: Most commonly on lower third of face
   - **c.** Treatment: Simple excision for cosmesis
   - **d.** May be associated with tuberous sclerosis if multiple
3. **Lipoma**
   - **a.** May be present at any age
   - **b.** Clinical presentation: Painless, soft, flesh-colored nodule
   - **c.** Anatomic location: Commonly found in the trunk and extremities
   - **d.** Treatment: Simple excision
4. **Dermatofibrosarcoma protuberans**
   - **a.** Appears in middle age
   - **b.** Clinical presentation: As a reddish-brown, firm, nodular plaque
   - **c.** Anatomic location: More commonly found on trunk, extremities

d. *Treatment is radical excision due to locally aggressive behavior. Margins >3 cm if possible
e. Local recurrence is common; however, metastasis is rare.

5. Neurofibroma
a. Composed of Schwann cells and endoneurial fibroblasts.
b. May appear at any age
c. Clinical presentation: Soft, compressible, flesh-colored or pink nodules; button-hole sign (can be pushed deeper into dermis)
d. Anatomic location: More common on the trunk and extremities
e. Treatment: Excision
f. Multiple neurofibromas may be associated with neurofibromatosis type I or II
i. *Type I—Cafe-au-lait spots, Lisch nodules (iris hamartomas), and optic nerve glioma
ii. Type II—bilateral acoustic neuroma

IV. OTHER DISORDERS

A. Calciphylaxis
1. Metastatic calcification resulting in calcification of blood vessels and necrosis of surrounding tissue.
2. Associated with renal failure
3. May appear at any age; more common in women
4. Clinical presentation: As necrotic ulcerations with red-blue mottling of skin (livedo reticularis)
5. Anatomic location: Common trunk and extremities
6. Treatment: Supportive care, phosphate-binding agents, parathyroidectomy, intravenous sodium thiosulfate, or excision
7. Excision often results in progressive calcification

B. Hidradenitis suppuritiva
1. Clinical presentation: Chronic inflammation and infection of the apocrine sweat glands. May result in chronic draining sinus tracts and abscesses.
2. Anatomic location: Most commonly affects axillae, breasts, perineum, and buttocks.
3. Treatment: Topical clindamycin, oral or IV antibiotics, excision of involved tissue. Following excision, reconstruction can occur via healing by secondary intention or by placement of a skin graft.

C. Xeroderma pigmentosum
1. Autosomal recessive disorder affecting DNA repair
2. High risk for SCC, BCC, and melanoma
3. Treatment: Avoidance of sunlight, isotretinoin, 5-fluorouracil, or excision of malignant lesions

D. Dystrophic epidermolysis bullosa
1. Hereditary disease with bulla formation of skin/mucosa following minor trauma
2. May result in encasement of digits with scar tissue
3. Treatment: Scar release/Z-plasty, topical steroids, avoidance of trauma

E. Cutis laxa
1. Defect in elastic fibers
2. Skin hangs loose from folds
3. Premature aging
4. Wound healing unaffected
5. Blepharoplasty and face lift can be beneficial

F. Pseudoxanthoma elasticum
1. Affects elastic fibers and collagen
2. Skin thickens and appears cobblestoned with mechanical stress
3. Wound healing is normal

G. Ehlers-Danlos syndrome (cutis hyperelastica)
1. Autosomal recessive or x-linked
2. Hyperextensible skin, severe joint laxity
3. Blood vessels are delicate
4. ***Wound healing is abnormal: Approach surgery with caution**

H. Acne vulgaris
1. Appears in younger patients
2. Clinical presentation: Comedones, inflammatory cysts, seborrheic plaques
3. Anatomic location: Face
4. Treatment: Topical retinoic acid, oral antibiotics, antibiotic pads, and oral isotretinoin (accutane). Isotretinoin—risk of birth defects; patients must have two forms of contraception. Avoid if planning aesthetic facial rejuvenation with lasers or peels.

I. Acne rosacea
1. Clinical presentation
   a. Facial flushing (increased vascularity)
   b. Thickened skin erythema, telangiectasia
   c. Acne rosacea (papules and pustules)
   d. Rhinophyma—nasal skin becomes erythematous with telangiectatic changes
2. Anatomic location: Affects forehead glabella, malar region, nose, chin
3. Treatment: Oral antibiotics, retinoic acid, dermabrasion, cryotherapy, laser (CO_2), tangential excision (for Rhinophyma)
4. Reconstruct with secondary contraction versus skin graft

J. Pyoderma gangrenosum
1. Clinical presentation: Multiple superficial abscesses with significant ulceration and skin necrosis.
2. Should get a dermatology consult and biopsy to evaluate, though this is a diagnosis of exclusion.
3. Treatment: Broad spectrum antibiotics
4. Approach surgery with caution given chances that this could create a flare and spreading of the disease.
5. Harvesting STSG can also create a flare of the disease at the donor site.
6. Associated with ulcerative colitis

PEARLS

1. Keratoacanthomas are characterized by rapid growth and spontaneous regression; they are difficult to distinguish from squamous cell carcinomas although they are not malignant.
2. Nevus sebaceous has a 10% to 15% risk of malignant degeneration into BCC. Excision is warranted.
3. Patients with cutis laxa do not have impaired wound healing and can be candidates for surgery, while those with cutis hyperelastica have wound healing difficulties and may not tolerate surgery.
4. 5-Fluorouracil is a DNA synthesis inhibitor; imiquimod (Aldara) is an immunomodulator.
5. Excision is generally appropriate when definitive pathology and/or margins are required.
6. Keep in mind the amount of local anesthetic which can be used (e.g., 1% lidocaine vs. 1% lidocaine with epinephrine).
7. When drawing up lidocaine for excision, avoid displaying needle.
8. Identify lesion and its observable borders prior to injection with lidocaine to avoid obscuring the lesion boundaries.
9. Design excisions as ellipses with sharp corners to facilitate closure and avoid dog-earing.
10. Nylon stitches from the face should be removed in 5 days to avoid railroad tracks. Stitches on back should be left in for 2 weeks to allow for more robust healing and to avoid splitting apart.

QUESTIONS YOU WILL BE ASKED

1. What is the recommended treatment of a nevus sebaceous?

   Excision with clear margins.

2. What is the risk of malignant transformation of an actinic keratosis, and what type of skin cancer can it progress to?

   Rates of AK progression to SCC were calculated at 0.6 percent at one year and 2.6 percent at four years. Of these SCCs, 75 percent were considered invasive and the remainder were in situ.

3. Patients with which of the following disorders should be approached with caution when considering surgical intervention: Cutis laxa? Pseudoxanthoma elasticum? Ehlers-Danlos?

   Patients with Ehlers-Danlos syndrome may suffer from excessive postoperative bleeding and poor wound healing and therefore surgery is generally contraindicated.

4. What is the most common location of dermoid cysts?

   Periocular region.

5. What malignancy can Spitz nevi appear histologically similar to?

   Melanoma.

Recommended Readings

Lee EH, Nehal KS, Disa JJ. Benign and premalignant skin lesions. *Plast Reconstr Surg.* 2010;125:188e–198e. PMID: 20440130.

Dr. Marzouq Amarin Lectures + Other Subjects

Contents:

Pressure Sore

Edited and Groomed by:

Fareed Halteh

Pressure sores

- A pressure sore is defined as an ulcer that develops over a bony prominence due to prolonged pressure. They can be called decubitus ulcers or bedsores; however, it is preferable to call them pressure sores as they may develop in a paraplegic patient due to prolonged sitting in a wheelchair.
- Despite countless advances during the last decade, the treatment of pressure sores remains a significant challenge to the medical community. What is particularly disturbing is the enormous number of affected patients.
- Epidemiology:
    - The incidence of pressure sore formation is highly variable; it depends on the population of patients under question
    - Several studies have been performed to determine the incidence of pressure sores in different settings:
        - In general, approximately, 9% of all hospitalized patients develop pressure sores.
        - In acute care settings, 11% (CVD 41%, acute neurological disease 27%, orthopedic injury 15%)
        - In chronic care facilities 30%
        - Acute settings include: ICU, CCU, and NICU.
        - Acute neurological disease includes: spinal cord injury or head injury
- The more significant ulcerations (deeper, more severe, and more in number) tend to occur in acute settings. In these patients, the primary disease process may overshadow other concerns leading to pressure sores to progress unnoticed for greater periods of time.
- In chronic care facilities, the most common sites for pressure sores are ischial (because of sitting on a wheelchair, and sitting on the ischial tuberosity), trochaneric (because of sleeping on one side more than the other, greater troachnter), and sacral regions (because the elderly sleep on their backs).
- Patients at high risk: these patients are at a high risk due to incontinence (stool, urine, or double), bed or wheelchair bound, unable to ambulate without assistance, loss of sensation (DM or multiple sclerosis).
    - Elderly: according to the national pressure ulcer prevalence survey (1994), it was found out that 62% of patients with ulcers were more than 70 years of age.
    - Malnourished (poor wound healing)
    - Alterations in mental abilities (the patient is semi conscious or unconscious; thus, you can't ask the patient to change position or corporate)
    - Paraplegics and tetraplegics
    - Spastic disorders (multiple sclerosis)
- Contributing factors and pathophysiology:
    - Pressure:
        - It is the single most important etiological factor.

- 96% of all pressure sores occur below the level of the umbilicus.
- 75% of all pressure sores are located around the pelvic girdle.
- In early studies, it was noticed that the pressure caused by an externally applied pressure reaches a maximum in deep tissues at the point of osseous protrusions (occiput, heel, knees, malleoli, sacral areas).
- Landis, in 1930, using a microinjection system, determined that capillary blood pressure in a single capillary ranged from 12 mmHg on the venous end to 32 mmHg on the arterial end.
- The external pressure at which blood flow stops is known as the capillary closing pressure.
- Complete cessation of flow, even in the face of positive arterial pressure, was found to occur at an external pressure of 70 mmHg less than the mean arterial pressure. The difference, known as transmural arterial pressure = MAP-70. However, this effect is not instantaneous; it needs time.
- There is an inverse relationship between the amount of pressure and the length of time required to cause ulceration:
    - Hussain et. Al studied the effects of pressure and time to determine which had the greater impact on ulcer formation. He believed that low pressures maintained for long time induced more tissue damage than did high pressure for short periods of time (proven in animal models); thus, the time factor is more important.
    - It was found that relieving the pressure for as little as five minutes can lessen the damage caused by pressures as high as 450 mmHg.
    - Based on many studies, it was proven that muscle is more susceptible to ischemia than skin. The initial pathology of ulcers occurs in muscle. With increasing magnitude or duration of pressure, the pathology progresses towards the skin. Muscle is especially sensitive, and it begins to degenerate after 4 hours of ischemia. In contrast, skin can withstand ischemia due to its lesser metabolic demands.
    - Due to this sensitivity, pressure sores in areas with a greater deal of muscle tend to take the shape of an inverted cone or flask. What we see is the “tip of the iceberg”.
    - Efforts to map the distribution of pressure on the bodies of supine and seated subjects suggest that pressures experience by the body in supine and prone positions are between 10-50 mmHg. However, on the ischial tuberosity, it ranges from 10-100 mmHg.

- Immobility:
    - It was found that patients with even some ambulation are less likely to develop ulcers. The protective mechanism of ambulation does not only

include relief of pressure, it includes an increase in minute ventilation, cardiac output, venous return, and maintenance of muscle mass.

- Shear (tangential pressure)
  - It is the second ischemic force in pressure ulcer formation. The theory states that a shearing force will weaken the superficial fascial fibers that attach skin to deep underlying fascia. This will lead to deformation and destruction of blood vessels leading to vascular occlusion and tissue destruction
  - In other words, the skin's blood supply comes from a dermal plexus which comes from perforators, which in turn, originate from the fascia of muscles. These perforators perforate fascia and ascend perpendicularly, then they start to branch giving the dermal plexus. Fascia is immobile relative to the skin, so when we apply a shearing force on the skin, it moves. However, the fascia doesn't. This will cause blood vessels to undergo angulations; thus, it augments ischemia.
  - To avoid shearing forces:
    - Mobilize patients as one piece; skin and fascia together.
    - Elevate the patient in linen
  - Although both shear and pressure act to produce stasis, pressure is the primary force in terms of occluding the arteriolar blood flow. Shear's greatest effect is on the reduction of the pressure value needed to stop arteriolar blood flow.
  - Friction is a factor that can contribute to skin ulceration through a non-ischemic mechanism. Friction increases mechanical forces on the epidermis, this leads to loss of stratum (friction is an issue in those patients who cannot lift themselves to change position). Friction occurs between the skin and the underlying liner, but shearing occurs between the skin and fascia.
- Moisture:
  - It leads to skin maceration, which leads to cellulitis (skin napkin rash). This will lead to infections, which will accelerate tissue breakdown increasing friction.
  - Sources of moisture include: perspiration, urine, feces, fistulae, and wound drainage. This is common in patients with incontinence.
- Nutrition:
  - Hypoproteinemia: a drop in either serum albumin or hemoglobin indicates anemia or hypoalbuminurea. Albumin is important for wound healing as it serves as a precursor for cytokines.
  - Ascorbic acid deficiency (vitamin C)
  - Decrease in trace elements: zinc

        - People with malnutrition are more liable for bed sores due to:
            - Decreased cushioning somatic fat mass
            - Decreased mobility
            - Decreased wound healing
- Staging and grading: this system is a clinical system, and it has its limitations. Erythema, for example, is harder to detect in darker people
    - Stage I: skin is intact, but reddened for more than 1 hour after relief of pressure. The muscle is involved, but it is not apparent clinically
    - Stage II (ischemia): blister or other break in dermis with or without an infection. It develops if pressure was continuous for 2-6 hours. In contrast to hyperemia, redness from ischemia requires at least 36 hours to disappear.
    - Stage III (necrosis): subcutaneous destruction into the muscle with or without an infection. Occurs if the pressure last for more than 6 hours. Usually, the skin is blue and firm
    - Stage IV (ulceration): involvement of bone and joint with or without an infection.
- Preoperative care:
    - The treatment of pressure sores is one of the most challenging areas in plastic surgery. Recurrence rates as high as 95% have been reported. Preparing a patient and the family for the long road ahead requires a team approach. The team should include: an internist, endocrinologist, nutritionist, neurologist, urologist, physiotherapist, psychiatrists, and a wound care nurse specialist.
    - All the components of the patient's overall care must be optimized prior to surgery. This increases the chances for a successful closure. These components include:
        - Nutrition:
            - Keeping serum albumin >2 g/dL
            - Protein intake: 1.5-3 g/kg: depends on the size of the ulcer
            - Caloric intake: 25-35 kCal/kg of non-protein calories
            - Vitamins: A, C, Zinc, Ca, Ferrous, copper
            - Patients may need a supplemental diet
        - Infection:
            - You should eradicate any UTI whether it is catheter related or not. Infected urine is a source of bacteremia and septicemia; thus, if you have recent wounds and there are bacteria in the blood, the bacteria will always settle on this fresh wound because it is an area rich in blood supply. Therefore, prior to any surgery, it is important to make sure that the patient doesn't have a septic focus.
            - Decrease the bacterial load inside the pressure sore itself. This is done through systemic and local antibiotics depending on the

tissue culture. Contamination of the pressure sore cannot be totally eliminated because it is a deep contamination.
        - The most common organisms that infect pressure sores include skin flora and enteric bacteria.
    - Pressure relief:
        - Turning the patient at intervals (5 minutes every two hours)
        - Special mattresses: to relieve pressure (foam, static floating, alternating air, low air loss, and air fluidized beds. The purpose of these beds is to evenly distribute the patient's weight. This will minimize the pressure in pressure areas.
    - Spasm relief:
        - Common in patients with spinal cord injury. The more proximal the lesion, the higher the incidence of spasm
        - The patient will be in flexion deformity due to head injury or multiple sclerosis.
        - If the spasm is not eliminated prior to any surgical procedure, the pressure sore will inevitably recur. In addition, it will be difficult for the nurse to take care of the reconstructed area.
        - To relieve the spasm:
            - Medical: diazepam
            - Surgical: this method is used in longstanding flexion deformities in which fibrosis of the tendons occur. Here, we perform tenotomy (cutting the spastic tendon).
    - Contracture (of a joint):
        - Longstanding denervation will lead to joint contracture.
        - Early cases are treated with physiotherapy
        - Late cases are treated with tenotomies.
        - If they are not treated, pressure sores will recur.
- Different methods for treating an ulcer:
    - Non-surgical treatment:
        - It is always prudent to attempt ulcer closure without surgical means.
        - If proper preoperative assessment and preparation are performed, there will be a period of time during which the ulcer can be observed. If the ulcer appears to be healing significantly, continuation of non operative treatment is indicated.
        - Some patients may never be candidates for surgical correction because of significant medical problems. In these patients, conservative treatment may lead to successful closure or at least it may allow for a stable wound that doesn't progress.

        - Local treatment with antibiotics (ointments), recombinant human PDGF and basic FGF (costly, limited, and not applicable for a huge wound), and wound care products.
    - Surgical treatment:
        - Excisional debridement of the ulcer, buria, heterotropic calcification, and the necrotic bone (removal of the whole iceberg)
        - Partial or complete osteoctomy to reduce the bony prominence
        - Closure of the wound with healthy, durable tissue. This depends on the nature of the patient, ulcer, and the need for subsequent procedures. Closure can be:
            - Direct closure: used in small pressure sores
            - Skin grafts: used in superficial ulceration with a success rate of 30% due to the presence of unhealthy underlying tissue
            - Flapps: local tissue flap, fascio-cutaneous, or myocutaneous.
    - Choice of flap: depends on the location, size, depth, previous surgery, and ambulation.
        - Myocutaneous flaps (superior to fascio-cutaneous flaps):
            - Excellent blood supply
            - Provision of bulky padding (cushioning)
            - Ability to readvance or reroatate to treat recurrence
            - Good for infected wounds
            - Can atrophic in elderly and patients with a spinal cord injury
            - It causes functional deformity in ambulatory patients.
            - Sensitive to external pressure
        - Fascio-cutaneous flaps:
            - Adequate blood supply
            - Durable coverage
            - Minimal functional deformity
            - Limited bulk (disadvantage)
    - Examples of flaps:
        - Flaps used to close a pressure sore in the gluteal region:
            - Medially based thigh flap: fascio-cutaneous flap
            - Gluteus maximus muscle flap: cover the pressure sore using this flap and cover the muscle with a skin graft.
            - V-Y hamstring advancement flap
            - Gluteal island flap
            - Gluteus maximus myocutaneous flap: muscle and skin
            - Tensor fascia lata flap: it is a good flap; but you must graft the donor area.

                - Gracilis flap: skin grafting for the donor area.
            - Flaps used to close a sacral pressure sore:
                - Bilateral gluteus maximus myocutaneous flap: it is called a rotation flap because there is an arch of rotation during the procedure.
                - Local flaps (fascio-cutaneous): these are called transposition flaps
                - Gluteus maximus muscle flap: you turn part of the gluteus maximus over the defect, and skin graft the donor area.
                - Myocutaneous flaps.
- Postoperative care:
    - Continuation of preoperative care
    - Leave the drains for a longer period of time.
    - No pressure on the operation site for 2-3 weeks, followed by gradual weight bearing.
    - Complications:
        - Acute, especially when taking a huge flap:
            - Hemorrhage
            - pulmonary and cardiac complications
            - infections.
        - Long term complications:
            - Recurrence (most common complication):
                - Causes of recurrence include underlying medical problems, changed mentation, improper nursing care, presence of spasm and contractures.
            - Marjolin's ulcer: SCC that develops in chronic ulcers (pressure sore is a chronic ulcer). It is aggressive with a poor overall survival (2 year survival rate is 66%). It can develop after as short as 3 years and as long as 25 years. Treated by chemotherapy, radiotherapy, and surgery.

Ulcers, sinuses, and fistulae

- The body is covered and lined by epithelium. The skin is covered by epidermis formed by keratinized stratified squamous epithelium, which is continuous with the epithelium lining the different internal organs. The epidermis is continuous with the GIT through the mouth and the anal canal, with the respiratory system through the nose, with the genital organs through the vaginal and with the urological system through the urethral meatus.
- The dermis of the skin is made of:
    - Fibers, cells, and ground substances
    - Blood, vessels, nerves, and lymphatics
    - Hair follicles, sebaceous glands, and sweat glands
- The epithelium stops diving via a mechanism called contact inhibition; division stops when the cell is surrounded by epithelial cells from all directions.
- Ulcer:
    - Defined as a pathological discontinuity of an epithelial surface. There are skin ulcers, gastric ulcers, and duodenal ulcers. The difference between a wound and an ulcer is that the ulcer is a wound, but of a pathological origin. The wound is of a traumatic origin.
    - Erosions of the mucosa and abrasions of the skin: Discontinuity of epithelium. Healed by regeneration.
    - Clinical examination: ulcers should be examined systematically. First, by inspection, then by palpation. The medical student should memorize the points to be examined, and record them:
        - Site, size and shape: it is more informative to join these items in one statement: there is an ulcer situated on the (specify the area), circular/oval/irregular in shape, and measures 5x3 cm.
        - Margin: describe the surrounding skin
        - Edge: the transition between the margin and the floor.
            - Sloping: denotes healing
            - Undermined: tuberculous or pressure ulcer
            - Everted: malignant (SCC)
            - Rolled or raised: rodent ulcer (BCC)
        - Floor: what the examiner sees; you should comment on the following:
            - Granulation tissue: healthy or not healthy
            - Necrotic tissue
            - Exposed structures as bone, tendon, or prosthesis
            - Islands of healing or skin grafts if present
            - Discharge.
        - Base: what can be palpated
        - Lymph nodes: lymph nodes draining the ulcer area should be examined.

- Lower limb ulcers:
  - The leg is a common site for different types of ulcers:
    - Arterial (ischemic)
    - Venous
    - Neuropathic
    - Malignant
    - Traumatic
    - Inflammatory
    - Diabetic
  - Every type of these aforementioned ulcers is usually associated with a specific clinical environment or background in the legs. The ulcer is a result of this environment, so upon physical examination, it is important to look for the signs of these environments or chronic diseases. This will help you in diagnosing what type of ulcer there is.
  - Arterial ulcers:
    - Occur on top of chronic arterial insufficiency due to atherosclerosis of the lower limb. The lower limb should be examined for signs of chronic ischemia: coldness, pallor, dryness, venous gutters, and absent pulses.
    - The arterial ulcer itself occurs at the distal parts of the foot or on the pressure areas. It usually lacks the signs of healing (sloping edges and presence of granulation tissue on the floor)
  - Venous ulcers:
    - Occur on top of chronic venous insufficiency. Other names are chronic venous hypertension or post-phlebetic syndrome.
    - This entity is characterized by increased venous pressure of the legs due to destruction of the veins following DVT. The leg is swollen due to venous edema. It is accompanied with induration and hyperpigmentation around the ankle. In addition, varicosity of the veins might be noted.
    - The venous ulcer is characteristically located on the lower third of the leg above the ankle. The most common site is above the medial malleolus. These ulcers usually show signs of healing.
  - Neuropathic or trophic ulcers:
    - Occur on top of an area with a disturbed sensation. The disturbance in sensation might be due to peripheral neuropathy or due to spinal cord pathology (spina bifida). The foot may be deformed due to loss of proprioception. Loss of proprioception leads to continuous trauma that ends as a Charcot joint. Moreover, muscle atrophy might be noted.

- Mechanism: it was thought that nerves have a nutritional role to tissue. This is why these ulcers were called trophic ulcers. However, it was discovered that sensory nerves have a protective role to the foot. They protect the foot from recurrent trauma. These ulcers occur over pressure areas (heads of the metatarsals or the heels). These areas are areas where soft tissue suffers from repeated pressure and trauma. This results in ischemia, scattered necrosis, and healing by fibrosis. This vicious cycle continues until the area finally breaks down and ulcerates

- Malignant ulcers:
  - Develop on top of chronic unstable scars that were not allowed to fully heal due to repeated trauma (burn scars), pr due to the presence of dead bones (chronic osteomyelitis).
  - These ulcers are squamous cell carcinomas called Marjolin ulcers; they usually need 20-30 years to develop.
- Diabetic ulcers:
  - Diabetic foot is defined as the pathological results of diabetes on the foot of a diabetic patient, namely ischemia, tissue loss, and infection. These changes are the result of three factors:
    - Diabetic peripheral neuropathy: this leads to the loss of or disturbance in the sensation of the feet. The feet will be vulnerable to injury and pressure ischemia.
    - Angiopathy: diabetic patients have both micro-angiopathy and macro-angiopathy.
    - Decreased immunity: different aspects of immunity are affected.
  - These factors contribute to the production of two elements of the diabetic foot: ischemia and tissue loss and infection. In fact, one of these elements leads to the other as follows:
    - Ischemia decreases the immunity of the affected part. This makes it more vulnerable to infection. Dead tissue is a good medium for contamination. On the other hand, infection contributes to ischemia and tissue loss by the following mechanism:
      - Infection cases edema which causes ischemia
      - Infection causes thrombosis of the micro-circulation
      - Infection causes tissue damage.
      - Infection increases the metabolic rate and oxygen requirement of the affected area. This results in relative ischemia.

- Fistula:
    - defined as an abnormal tract connecting two epithelial surfaces. It could be a communication between the epithelium of one viscus to another, or between the epithelium of a viscus and that of the skin.
    - Etiology: fistulae may be congenital or acquired. The latter could be inflammatory, malignant, iatrogenic, or traumatic.
    - Congenital fistulae:
        - Tracheo-esophageal fistula: many types of abnormal communications between the trachea and the esophagus with different clinical presentations.
        - Branchial fistula: it is a pharyngeo-cutaneous fistula formed by the second branchial cleft and pouch due to failure of obliteration of the cervical sinus. The cervical sinus is formed by the down growth of the second pharyngeal arch over the second, third, and fourth pharyngeal clefts. The tract is lined by ciliated columnar epithelium. Its discharge is mucus or muco-pus. The tract passes between the external and internal carotid arteries and opens internally into the pharynx posterior to the tonsil. It opens externally on the skin of the lower neck anterior to the sternocliedomastoid muscle.
        - Umbilical fistula: it is formed due to the presence of a patent vitello-intestinal duct. It communicates the midgut with the umbilicus. The vitello-intestinal duct normally disappears, but it may persist as an umbilical fistula, umbilical sinus, vitelline cyst, a fibrous band, or Meckel's diverticulum.
        - Fistulae associated with anorectal agenesis: with the high type of anorectal agenesis, the anus, the anal canal, and the lower part of the rectum are absent. The rectum ends with a pouch; a fistula connects this pouch with the bladder or urethra in males, and the vagina in females.
        - Differential diagnosis of umbilical discharge:
            - Pilonidal sinus
            - Umbilical fistula
            - Umbilical sinus
            - Patent urachus
            - Sister Mary-Joseph's nodule: a nodule of metastatic carcinoma bulging through the umbilicus causing a seroanguinous discharge.
    - Inflammatory fistulae:
        - Inflammatory bowel disease and diverticultis may be complicated by the formation of cutaneous or internal fistulae. These fistuale connect the colon to the urinary bladder or the vagina

        - In acute cholecystitis, rupture of the inflamed gallbladder into a part of the GIT produces a fistula into that part.
        - Perianal fistulae are the result of infection of the anal glands.
    - Malignant fistulae: malignant disease of one viscus may invade another one and commnicate with it. Examples include: tracheo-esophageal, gastro-colic, and colo-vesical fistulae. Cancers may open to the skin producing a cutaneous fistula.
    - Iatrogenic fistulae: due to dehiscence and leakage of bowel anastomosis.
    - Traumatic fistulae: due to penetrating abdominal trauma with visceral injury.
    - Clinical presentation of a fistula:
        - The bacterial effect: when a viscus with a high bacterial load (colon) communicates with a normally sterile viscus (bladder), it causes an infection. A colo-vesical fistula produces a UTI.
        - The effect of bypassing functional parts of the GIT: in a gastro-colic fistula, the small bowel is bypassed. This produces diarrhea and malabsorption.
        - Loss of water, electrolytes, enzymes and nutrients. Entero-butaneous fistulae are classified into high and low output fistulae depending on their output volume. The more the amount, the more the loss. The more proximal the fistula is, the greater the loss. This means that a duodenal fistula is more dangerous than an ileal one.
        - Effect of fistula content on the skin: upper GI fistulae contain a high amount of digestive enzymes. This produces skin damage and maceration.
        - Vesico-vaginal fistulae are associated with urinary incontinence. Colo-urinary fistulae are associated with passage of feces with urine.

- Sinus:
    - Defined as a blind tract lined by granulation tissue and opens into an epithelial surface.
    - Most sinuses are congenital:
        - Auricular and pre-auricular sinuses: due to incomplete fusion of the nodules originating from the first and second pharyngeal arches. These form the pinna of the ear.
        - Branchial sinus: failure of obliteration of the cervical sinus or due to infection of a branchial cyst.
        - Thyroglossal sinus: due to infection of thyroglossal duct cyst.
        - Umbilical sinus: a remnant of the vitello-intestinal duct
        - The pilonidal sinuses are believed to be of an acquired nature. The most common site is over the coccyx in the anal cleft. They may be seen in the umbilicus, as well.

- Clinical picture of a sinus: sinuses are prone to recurrent infections, so they should be excised completely before an infection that produces fibrosis, adhesions, and secondary tracts. At this stage, complete removal is a difficult task.
- Clinical case: a patient presents with umbilical discharge; what is your differential diagnosis?
    - Vitello-intestinal fistula: the content is feculent
    - Vitello-intestinal sinus: contains mucus
    - Patent urachus: smell of urine
    - Pilonidal sinus: purulent discharge, hair is seen
    - Sister Mary-Joseph's nodule: serosanguinous discharge; rare.

Postoperative pain control

- Postoperative pain control is inadequate for the following reasons:
    - Misbelieves:
        - Addiction
        - Overdose of narcotics which might lead to cardiac and respiratory arrest
    - Psychological causes: the patient thinks that postoperative pain is normal. Most of the time, patients will not admit to pain. Sometimes, the nurses don't provide enough care for such patients
- Answers to the aforementioned problems:
    - Narcotics can cause addiction. However, the dose at which addiction happens is much higher than the dose needed for postoperative pain relief. In burn patients, analgesia can be continued for 2 months without addiction.
    - To give analgesia without complications and to avoid overdosing, it should be given in a controlled manner.
- Why should we provide adequate postoperative analgesia?
    - Humane cause: doctors should control pain and stop the suffering of their patients.
    - Physical causes: inadequate postoperative pain will increase morbidity and mortality.
- Effect on pain on the body:
    - Increase in heart rate, blood pressure and oxygen demand. If the patient has borderline coronary circulation with a decreased cardiac reserve, increased pain can lead to ischemia
    - Decreased GI motility: paralytic ileus.
    - Atelactasis: if the patient has pain, their chest won't be able to move.
    - Pneumonia: inability to expectorate
    - Decreased mobility: increased risk for DVT and PE
- Certain advances that helped ease pain:
    - Laparoscopic surgery: decreased size of incision will decrease postoperative pain
    - Medical treatment for previously surgically treated diseases.
- Route of admission:
    - Minor surgery: oral
    - Major surgery: IM or IV. Due to the unreliable absorption of IM doses, IV is used most of the time.
- Frequency of analgesia:
    - Regular (eg Q4hours): here, doctor guarantees that the pain is controlled. The dose is given every 4 hours regardless of the patient's pain
    - PRN: (eg. Q4hours PRN): this means that the doses are given at least 4 hours apart. It doesn't mean that the patient is given a dose every 4 hours. If the patient doesn't need analgesia after the passage of 4 hours, the dose can be avoided.

- PCA (patient controlled analgesia): you install a pump that gives a baseline dose of analgesia. If the patient is in pain, he/she can press a button that will give an increased dose. Here, the patient doesn't have absolute control as the doctor sets a safety interval. It was found out that patients who had control over their analgesia needed lesser doses

Hand infections and trauma

- We are going to deal with hand problems as a separate entity because Man is very dependent on his hand. Without hands, Man would be disabled. In addition, the hand is formed from compartments. This means that hands can suffer from compartment syndrome.
- Hand infections:
    - Paronychia:
        - Infection of the nail fold.
        - The most common infection in the hand
        - It happens due to bad maneuvering of hangnails or due to bad manicure.
        - In paronychia, there is redness around the nail on either or both sides of the nail.
    - Felon
        - It is the layman term for distal pulp space infection
        - The proximal, middle, and distal pulp spaces lie on the palmar aspects of the proximal, middle, and distal phalanges.
        - The pulp space is lined by skin, subcutaneous tissue, and a deep fascia connected to the bone. Skin of the palms and soles is thick due to its adherence to the underlying tissue by fibrous septa. The skin of the dorsum of the hand or foot, on the other hand, slides freely over the underlying tissue.
        - The distal pulp space is formed of fibrofatty tissue and it is considered as a compartment. Infection in that area will remain contained.
        - Each finger receives 2 digital arteries (one at each side of the finger) as part of the neurovascular bundle.
        - The 2 digital arteries anastamose with each other. Upon reaching the distal phalanges, they are considered as end arteries with no anastomosis between them. Thrombosis of the end arteries due to infection and increased pressure can lead to gangrene of the distal pulp space and bone necrosis. This leads to osteomyelitis. In other words, a felon can lead to osteomyelitis.
        - Felon is usually caused by pricking. This means that it affects the index and thumb fingers the most.
    - Tenosynovitis:
        - Anatomy:
            - Each finger receives two tendons; one for flexor digitorum superficialis and the other one for flexor digitorum profundus. FDS is inserted on the middle phalanx and flexes the finger at the proximal interphalangeal joint. FDP is inserted on the proximal

part of the distal phalanx. So it flexes the finer at the distal interphalagneal joint.

- DIP is located at the distal finger crease; the PIP is located at the next finger crease. The MCP is not located at the third crease; it is located more proximally in the palm (at the distal palmar crease)
- Each tendon is surrounded by a synovial shear. Infection in this sheath is called tenosynovitis.
- Since the tendon of FDP is inserted on the proximal part of the distal phalanx, tenosynovitis will not involve the distal pulp space.

▪ Treatment of hand infections:

- The most common causative organism of hand infections is staph aureus. The second most common is streptococcus.
- Strep and staph are gram positive cocci. Staph usually produces abscesses and contained infection due to the production of coagulase. Strep infections usually cause a spreading infection due to the production of hyaluronidase and streptokinase.
- The initial treatment of hand infections is according to the "good guess" principle. We use oxacillin or amplicillin to combat staph aureus. Them, we take sample and culture them. The treatment is then determined according to the culture.
- The initial phase of any subcutaneous infection is the cellulitic phase. In staph infections, the infection will be localized to form an abscess. The abscess is treated by incision and drainage.
- If a patient presents with a staphylococcal abscess and we don't treat it, the abscess will transform into a sterile abscess. This abscess is treated b incision and drainage; there is no need for antibiotics. Usually, patients present at earlier stages where an abscess and a cellulitic focus are both present. These patients are given systemic antibiotics. In other words, if a patient presents with signs of infection he/she is treated with antibiotics. If there was no response, this indicates the presence of a sterile abscess.
- General rules of management:
    - Antibiotics with incision and drainage
    - Elevation of the upper limb to decrease edema, which improves the circulation. This decreases the pain.
    - Resting the organ to decrease its pain.

o Trauma of the hand:

▪ This is complicated issue because we have tendons, soft tissues, blood vessels, and nerves.

        - The most important thing to know is the concept of revascularization of a completely removed finger. We replant the finger by joining its artery to the corresponding artery in the hand. This amputated finger has to be dealt with carefully. While being transported to the hospital, it should not be placed in water nor frozen. On the other hand, has to be kept at 4 degrees in a dry environment.
    - Notes:
        - Acute inflammation Vs. chronic inflammation:
            - The insult forces the body to form an inflammatory reaction, which will subside either when the insult disappears or the body overcomes it by resolution and repair.
            - In the case of chronic inflammation, tissue destruction coincides with tissue repair. This leads to a viscous cycle of insult, inflammation, and healing.
        - Antibioma:
            - It is a hard, edematous swelling containing sterile pus. It is a result of the treatment of an abscess with long term antibiotics rather than by incision and drainage.
            - Treatment involves exploration and drainage of the lump (if it looks suspicious) or by careful observation until spontaneous resolution