1. Soft Tissue Coverage

Wounds

Definition: Discontinuity of epithelium caused by trauma or pathological causes (ulcers).

Types:

- **Partial Thickness**: Involves epidermis and part of the dermis; heals by **regeneration.** (functional+ cosmetic)
- **Full Thickness**: Involves epidermis and entire dermis; heals by **fibrosis.** (not got the function and from)

Tissue Transfer Criteria:

1. Replace Like with Like
2. Maximize Recipient Benefit
3. Minimize Donor Area Harm
4. Safety

Principles of General Management

1. **Clean Wounds Before Closure**:
   - Clean wounds = can close immediately.
   - Contaminated wounds = delayed closure after cleaning and debridement.

2. **Method Selection for Wound Closure**:
   - **Direct Closure**: Simple wounds without significant tissue loss.

   - **Secondary Intention**: Allow healing naturally for small, non-critical wounds. (no functional or cosmetic value)
   - **Skin Grafting**: Use for defects needing durable coverage, require new blood supply. (using the skin or part of it)
     - ➢ Plasmatic circulation 1-2d and neovascularization 2-3d.

a) **Split Thickness (STSG)**: Epidermis and partial dermis; heals in two weeks; good for large areas. (donor heals by regeneration)

b) **Full Thickness (FTSG)**: Entire skin layer; used for face, hands, and small areas needing high-quality results. (donor→ loose skin→ direct closure) (dermatome)

Signs for take: adherent, pink, blanches with pressure.

   - **Flaps**: For deep or complex defects requiring vascularized tissue.

Types:

1. **Local Flaps**: For nearby defects.
2. **Free Flaps**: Transferred with vascular pedicle and connected via microvascular surgery.

3. **Wound Classification**
   1. Incised Wound: Clean edges; primary closure if <6 hours.
   2. Lacerated Wound: Jagged edges; excision then direct closure.
   3. Crushed Wound: Heavy contamination; repeated cleaning and delayed closure.

2. **Prevent Complications**:
   1. Prevent infection (minimize bacterial load).
   2. Avoid tension on wound edges to prevent poor healing.
   3. Manage underlying causes (e.g., diabetes, ischemia).

2. Burns

1. **Thermal Burns**:

   - Mechanism: Coagulative necrosis due to heat (temperature >45°C).

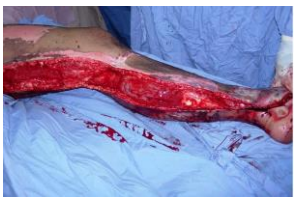
   - **Types:**
     - Dry heat (direct flame).
     - Moist heat (scalds).
     - Contact burns (hot surfaces).
     - Friction burns.

   - **Wound Zones:**
     1. Zone of Coagulation: Dead necrotic tissue.
     2. Zone of Stasis: Injured but salvageable tissue.
     3. Zone of Hyperemia: Peripheral vasodilation, viable tissue.

2. **Chemical Burns**:
   - Acids: Coagulative necrosis, limited penetration.
   - Alkalis: Liquefactive necrosis, deeper tissue damage.
   - Management: Immediate irrigation (2–4 hours for alkalis, 30 minutes for acids).

3. **Electrical Burns**:
   - Mechanism: Damage inversely related to tissue resistance (nerves and muscles most affected). DECEIVING

   - **Complications:**
     - Head injury, PNS damage, arrythmias, bone fractures
     - Compartment syndrome.



compartment syndrome, severe leg pain and numbness, treated but fasciotomy

- **Myoglobinuria leading to renal failure.**

urine bag --> red dark urine bc of myoglobinuria--> AKI. to prevent this: good dehydration, alkalization

General Management of Burns

1. Oxygenation:
   - Ensure adequate oxygenation to support tissue perfusion.
2. Fluid Resuscitation:
   - Vital for maintaining perfusion and addressing electrolyte and acid-base imbalances.
   - Proper fluid management helps prevent shock and organ failure.
3. Anemia Management:
   - Address any blood loss and maintain hemoglobin levels.
4. Nutritional Support:
   - Provide adequate nutrition to support healing and combat the hypermetabolic state induced by burns.
5. Minimize Tissue Edema:
   - Avoid over-resuscitation of fluids to reduce the risk of edema, which can compromise circulation.
   - Elevate injured limbs to reduce swelling.

Fluid Resuscitation for Burns

1. **Parkland Formula**:

Fluid in first 24 hours = **4 mL × Body weight (kg) × % TBSA burned**.

Half of the calculated volume in the first 8 hours, Remaining half over the next 16 hours.

2. **Monitoring Parameters**: **Urine Output/ Hematocrit/ Central Venous Pressure (CVP**.
3. **Type of Fluids**:
   - **First 24 hours**: Crystalloids (e.g., Ringer's lactate).
   - **After 24 hours**: Colloids may be used.

ABCD Approach in Burn Management:

A→ airway:

- **direct thermal injury** → upper air way obstruction due to edema of the oropharynx and vocal cords

Direct inspection by laryngoscopy or bronchoscopy then endotracheal intubation.

Tachycardia, hoarseness and difficulty clearing bronchial secretions.

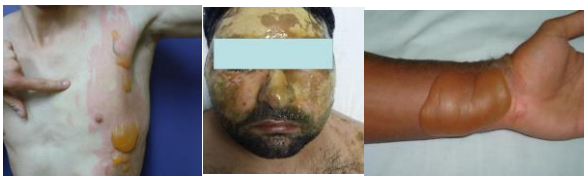
- **Carbon monoxide poisoning**→ diagnosed by estimation of carboxyhemoglobin level in the blood, 100% O_2 for the treatment

---

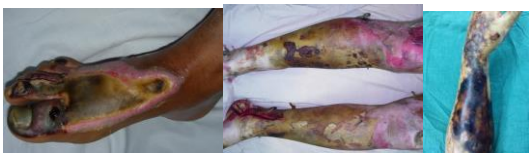
Assessment of Severity

1. **Depth of Burn:**

- **First Degree:** Epidermis only, heals in 1–6 days, no scarring, erythema (sun burn).
- **Second Degree (Partial Thickness)**: Epidermis + dermis, painful, blisters (bullae), wet, exudate, blanching denoting intact dermal vascularity, preserved skin elasticity. heals in 1-4 weeks. (regeneration)



- **Third Degree (Full Thickness):** Entire skin necrosis, lathery, insensitive, eschar, inelastic (gelatine), thrombosed dermal vessels, leaves scar, requires grafting.



2. **Percentage of Burn:**

- Rule of Nines:
  - Head & neck: 9%.
  - Each arm: 9%.
  - Each leg: 18%.
  - Front & back trunk: 18% each.
  - Perineum: 1%.
- Children:
  - head and neck: 20%
  - Lower limbs: 14%
- For small burns: the palm of the patient's hand : 1%

Management

1. **Local Wound Care:**
   - Partial Thickness: Wet dressings, infection prevention.
   - Full Thickness: Early escharotomy and skin grafting.

2. **Complications:**
   - Edema→ tissue ischemia: Requires escharotomy.
   - Infections: No prophylactic antibiotics to avoid resistance only for treatment.
   - Nutritional Support: High-calorie intake to counter hypermetabolism.

INDICATIONS OF ADDMISSION TO HOSPITAL→

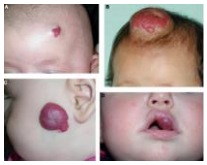
1. burns that need fluid resuscitation: Adults>15%, children>10%).
2. Full-thickness burns> 2%
3. Burns of special areas: face, hands, perineum.
4. Electric and chemical burns.
5. Inhalation injury.
6. Old age and co-morbidity.
7. Suspected child abuse.

3. Vascular Anomalies

1. **Vascular Tumors**:

- **Infantile Hemangiomas (95%)**: strawberry naevus/ mast cells
- "M.C, endothelial proliferation.

- Benign, 2f:m, started noticed at week 2



Phases:

1. **Proliferating Phase (1st 5–8 months)**: Rapid growth, bright red, potentially disfigured.
2. **Involution Phase (7–9 years)**: Darker, grey hue, loss of color, fine telangiectasia. (mottling)
3. **Involution Phase**: soft lump, fibro-fatty residue, Regression in 70% by 7 years, 90% by 9 years.

- **Histology**: Placental-like, GLUT-1 positive, PHACE association.
- Predilection: Head and neck (10% of full-term, 20% of preterm infants).

Management: treatment is mostly expectant/ Biopsy, CBC, MRI/US
First Line: Propranolol (1–2 mg/kg/day): Causes vasoconstriction, inhibits growth, and promotes regression.

Second Line:

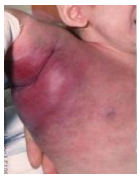
- Intra-lesional Steroids: 2 mg/kg every 4–6 weeks for localized lesions.
- Systemic Therapy: Reserved for refractory cases (monitor for rebound growth).

Surgical: Excision for lesions causing obstruction (airway (in subglossal space→ tracheostomy), vision(amblyopia)) or large disfigurements.
Other Interventions:

- Laser Therapy: telangiectasias after 10y or ulcerated lesions.
- Embolization in high output cardiac failure

- **Kaposiform Hemangioendotheliomas (KHE)**

Rare, aggressive tumor presenting in early infancy.



Often associated with **Kasabach-Merritt Phenomenon (KMP)**, causing high risk of systemic bleeding.

Management: **Sirolimus**: First-line treatment, particularly effective in MTOR-positive tumors.

- **Congenital Hemangiomas:** Fully developed at birth/ negative GLUT-1

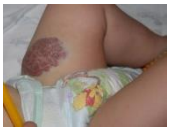
- **Types**:

1. **Rapidly Involuting Congenital Hemangiomas (RICH)**:

- involutes by 1 year.
- Large masses often on leg, firm, leave plaque-like → atrophic patch

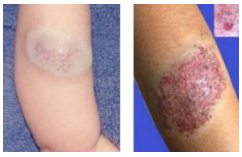
2. **Non-Involuting Congenital Hemangiomas (NICH)**:

- does not regress+ no further growth
- Round or oval masses, flat shape, telangiectasia, halo
- Surgical excision



3. **Partially Involuting Congenital Hemangiomas (PICH)**:

- Similar to NICH but slowly regresses by age 10.
- Management: Observation or surgery depending on symptoms.



- **Pyogenic Granuloma (Lobular Capillary Hemangioma)**

- Rapidly growing (w-m then stabilizes) red papule with a friable surface.
- Bleeds profusely with minor trauma and may ulcerate.
- complete excision.

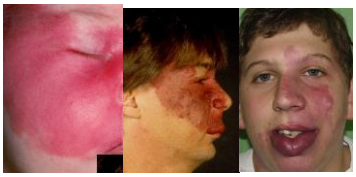
---

2. **Vascular Malformations**: normal turn over rate with abnormal architecture

Can lead to long term muscular and soft tissue hypertrophy, bone fracture, bleeding, distal parts atrophy, entrapment of platelets.

- **Capillary Malformations**:

Includes **Port Wine Stains**:

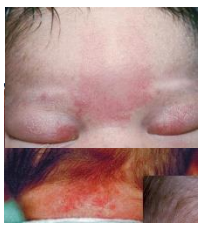


- 0.3% of newborns, often on the face.
- Macular patch→ purplish discoloration
- 2nd hypertrophy, skin nodules, incidence of pyogenic hemangioma, restricted to one or more of 3 trigeminal sensory area
- Sturge- weber syndrome.

Management: clinical psychologist, Laser therapy for lightening color, Surgery for tissue hypertrophy (e.g., lower lip).

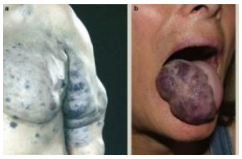
- **Nevus Simplex (Macular Stain)**

- **Features**: Single or multiple blanchable, pink-red patches in newborns. Affects 40–60% of infants.
- sites: Eyelids, glabella, neck ("stork bite" or "angel kiss"). Scalp, nose, lip, back.
- fade within 1–2 years. / Lesions on the back of the neck may persist without significant consequence.

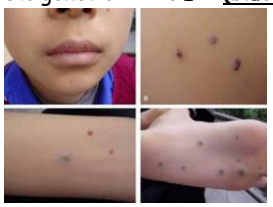


- **Venous Malformations**:

- Low-flow, Compressible blue masses, painful, empty on elevation, may cause coagulopathy.



- Mortality→ thrombi, emboli, bleeding, DIC
- 5% genetic → TIE-2 **(blue-rubber bleb syndrome)**



Management: Compression garments, NSAIDs, sclerotherapy, surgery.

- **Lymphatic Malformations**:
  - **Microcystic:** superficial, well-circumference, small, raise.
  - **Macrocystic:** neck, large, cystic hygroma subcutaneous
  - Histo: dilated lymph channel without connection to the lymphatic system.
  - **Management**: Sclerotherapy (e.g., OK-432), surgery.

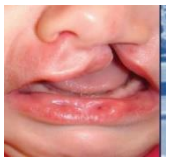
- **Arteriovenous Malformations (AVMs)**:
  - High-flow lesions with arterial feeders, fistulas and enlarged vein.
  - Warm, pulsatile with bruit, Doppler signal, purplish discoloration, larger→ cardiac failure
  - **Management**: radiology→ embolization (e.g., ethanol, coils) and surgical excision and reconstruction.

4. Cleft Lip and Palate

- 1 in 700 live births globally; 1.39 per 1000 in Jordan (2001).
- Cleft lip +/- palate: Male > Female (2:1).
- Cleft palate only: Female > Male (1:2).

Causes: Genetic and environmental interactions.

1. **Familial**: Recurrence risk increases with affected relatives: 1 child affected: ~4%, 1 parent affected: 3.2%, 1 parent + 1 child affected: ~15%.
2. **Nonsyndromic**: Multifactorial inheritance, influenced by genes (e.g., **IRF-6, TGF-B2**).
3. **Syndromic**: Associated with >300 syndromes (e.g., **Van der Woude Syndrome**). →low protein level, lower lip pits.



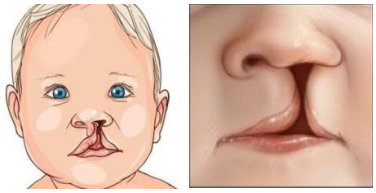
Environmental Factors: Smoking, viral infections, teratogens (e.g., Rubella virus, Cortisone/ steroids, Mercaptopurine, Methotrexate, Valium, Dilantin), maternal diabetes, advanced maternal age, and folic acid deficiency.

Formation:

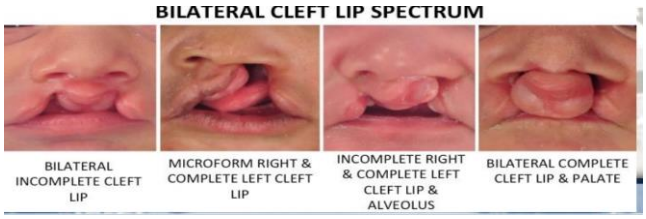
Cleft lip → failure of proliferation of the mesodermal cells in the midline.

Veau Classification:
- **Class I**: Incomplete cleft involving only the soft palate.
- **Class II**: Cleft of hard and soft palate.
- **Class III**: Complete unilateral cleft lip and palate.
- **Class IV**: Complete bilateral cleft lip and palate.

unilateral incomplete --> intact but hypoplastic



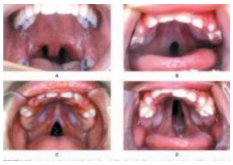
unilateral complete --> columella is displaced to the normal side/ Nasal ala on the cleft side displaced laterally and inferiorly/ Tip of the nose deviates toward the non-cleft side.



Orbicularis oris attaches at the lateral cleft margin bilaterally at the nasal alla, laterally displaced alla and extremely short columella, symmetrical nasal deformaties.

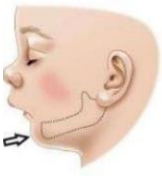
Cleft palate → primary→ failure of fusion of maxillary and medial nasal processes/ anterior to incisive foramen

→ secondary→ failure of fusion of palatine shelves/ posterior to incisive foramen.



Associated Conditions and Complications

- **Otological**: Persistent otitis media with effusion; 80–95% require myringotomy (grommet) tubes.
- **Speech**: velopharyngeal incompetence, Hypernasality, articulation errors; may require pharyngoplasty or dental prosthesis.
- **Airway**: Rarely in isolated cleft palate, seen in Pierre Robin sequence. (Micrognathia, glossoptosis, and cleft palate) Management – prone positioning)/ Mandibular distraction.



- **Dental**: Malocclusion, missing teeth.
- **Psychosocial**: Social stigma and self-esteem issues.
- **Feeding difficulties**: limit ability to suck due to common cavity/ special bottles/ not in cleft lip alone.

Management

1. **Multidisciplinary Team**: Involves plastic surgeons, orthodontists, audiologists, speech pathologists, otolaryngologists, geneticists, pediatricians, psychologists, and oral maxillofacial surgeons.

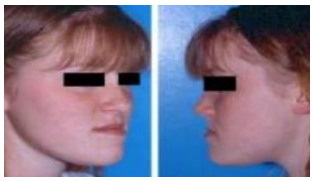
2. **Surgical Repair Timeline**:

- **Birth**: Address airway and feeding issues.
- **Age 1–3 months**: Lip taping and nasoalveolar molding.
- **Age 3 months**: Repair cleft lip; ventilation tubes placed.
- **Age 9–12 months**: Repair cleft palate.
- **Age 7–8 years**: Alveolar bone grafting.
- **Age 18+ years**: Midface advancement and orthodontic treatment.

3. **Techniques**:

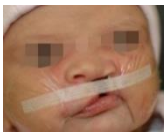
- **Lip Repair**: typically at 3 months of age/ rule of 10 → 10w, 10 lbs, Hemoglobin 10
  Milliard rotation-advancement technique.
  Goals of the bilateral→ Symmetry, orbicularis oris closure, nasal tip alignment, proper philtral and tubercle formation.
- **Palate Repair**:

Primary goals→ Create velopharyngeal valve for swallowing and speech/ Separate nasal and oral cavities/ Preserve midface growth. (dish face deformity).

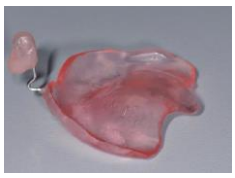


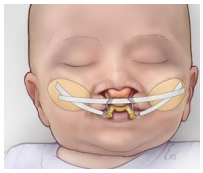
Bardach two-flap palatoplasty/ Furlow double-opposing Z-plasty.

Special Devices and Preoperative Care



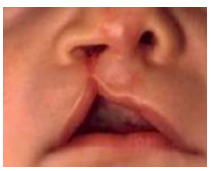
- **Lip Taping**: Reduces cleft width; worn 24 hours/day.
- **Nasoalveolar Molding (NAM)**: Custom devices with nasal stents for shaping cartilage and alveolar ridge.





- **Lip adhesion**: Performed at 2–4 weeks of age to reduce tension and convert to incomplete clefts/ leaves ascar.

  Indicated for: Wide bilateral clefts, Severe premaxilla protrusion.

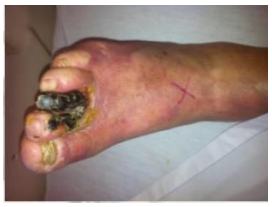


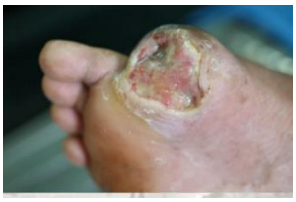
5. Chronic Wounds

- Wounds that fail to heal in the expected time frame, usually more than 6 weeks.

---

1. Ischaemic Arterial Ulcers



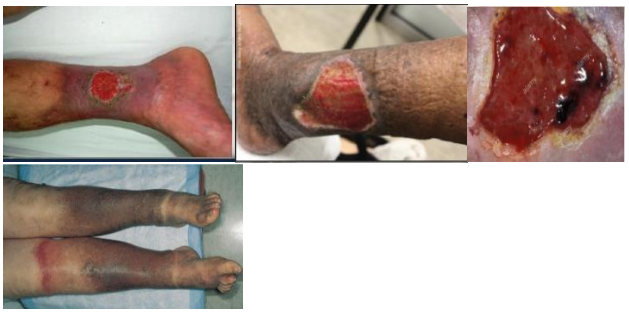


- **Cause**: Lack of blood supply; associated with peripheral vascular disease (e.g., intermittent claudication, rest pain, color changes, night pain).
- **Symptoms**: Painful ulcers, diminished/absent pulses, decreased ankle-brachial index, poor granulation tissue.
- **Appearance**: Shallow, smooth margins, pale base, dry surrounding skin.
- **On examination**: diminished or absent pulse, decreased ABI, poor granulation
- **Management**:

1. Revascularization (bypass/angioplasty).
2. Wound care: Debridement, rest, antibiotics for infection.
3. Address comorbidities: Glycemic control, smoking cessation.

---

2. Venous Stasis Ulcers

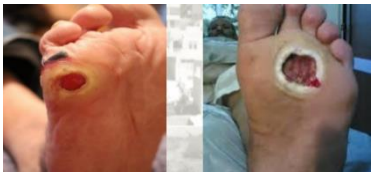


- **Cause**: Venous insufficiency; commonly due to deep venous system incompetence.
- **Symptoms**: Painless ulcers, pigmented surrounding skin.
- **Appearance**: Shallow, irregular margins, granulated; commonly above the medial malleolus (cockett's perforator)
- **Management**:

1. Compression therapy (e.g., stockings).
2. Address venous hypertension.
3. Prevent recurrence with ongoing compression therapy.

---

3. Diabetic Foot Ulcers

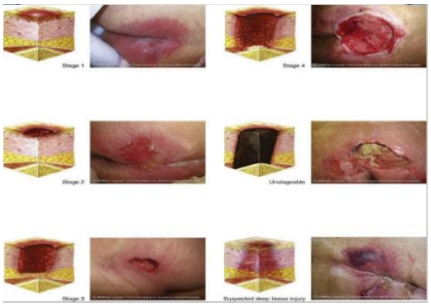


- **Cause**: Prolonged inflammatory phase, neuropathy, immune compromise, microvascular damage.
- **Symptoms**: Painless initially due to neuropathy, poor healing due to ischemia and infection.
- **Management**:

1. Blood sugar control.
2. Multidisciplinary care (podiatrists, surgeons).
3. Infection treatment (antibiotics, debridement).
4. Advanced therapies: Arterial revascularization, platelet-rich fibrin.

---

4. Pressure Ulcers



- **Cause**: Tissue necrosis from pressure over bony prominences.
- **Risk Factors**: Immobility, altered mental/nutritional status, friction, shear forces.
- **Stages**:
  - **I**: Non-blanchable redness, intact skin.
  - **II**: Partial dermis loss, pink wound bed.
  - **III**: Full-thickness loss; subcutaneous fat visible.
  - **IV**: Full-thickness loss; bone, tendon, or muscle exposed.
- **Management**:

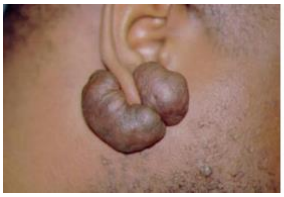
1. Pressure redistribution.
2. Debridement & dressing based on stage.

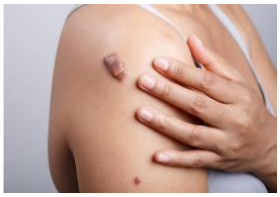
Malignant Transformation (Marjolin Ulcer)

- **Cause**: Chronic wounds can transform into malignancies (squamous/basal cell carcinoma).
- **Signs**: Overturned wound edges.
- **Action**: Biopsy for suspected cases.

Excessive Wound Healing

1. Keloids



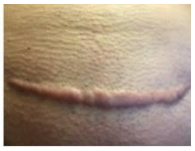


- **Definition**: Overgrowth of scar tissue extending beyond the original wound margin.
- **Etiology**:
  - Genetic predisposition.
  - More common in individuals with darker skin tones.
  - Triggers: Minor injuries, surgeries, burns.
- **Histology**:
  - Thickened dermis with disorganized collagen bundles. (circles)
  - Increased fibroblast activity.
  - Increased number of mast cells → itching
- **Treatment**:

1. **Steroids (triamcinolone)**: Intralesional corticosteroids reduce fibroblast activity and collagen synthesis.
2. **Silicone Gel/Sheets**
3. **Pressure Garments**: Reduce scar volume through compression.
4. **Laser Therapy**: Targets vascular components to flatten scars.
5. **Surgical Excision**: Typically combined with adjuvant therapies to prevent recurrence.
6. **Interferon Injections**

---

2. Hypertrophic Scars



- **Definition**: Raised scar tissue confined to the wound margin.
- **Etiology**:
  - Excessive mechanical tension on wound edges.
  - Often develops within 4-8 weeks of injury.
- **Histology**:
  - Collagen bundles in a parallel arrangement.
  - Increased vascularity.
  - Increased number of mast cells → itching
- **Treatment**:

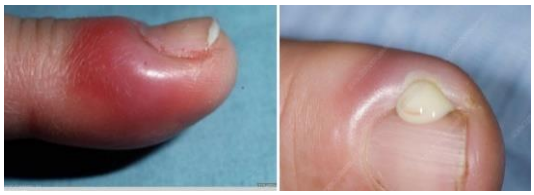
1. **Steroids**: Similar to keloid treatment.
2. **Silicone Gel/Sheets**
3. **Pressure Garments**
4. **Laser Therapy**: Fractional CO2 and pulsed dye lasers are most effective.
5. **Surgical Revision**: Often used for long-standing scars.

6. Common Hand Conditions

1. Paronychia

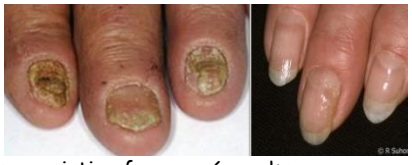
Acute Paronychia

- **Definition**: Sudden infection of the lateral or proximal nail folds.



- **Etiology**:
  - Most caused by *Staphylococcus aureus* or *Streptococcus pyogenes*
  - Triggered by trauma (e.g., nail biting, hangnail removal).
- **Presentation**:
  - Pain, redness, warmth, and swelling near the nail fold.
  - Pus may accumulate under the nail fold.
- **Pathophysiology**:
  - Bacterial entry via minor trauma leads to localized infection.
- **Treatment**:
  1. Warm soaks to reduce pain and swelling.
  2. Incision and drainage if abscess forms. (partial nail removal may be needed)

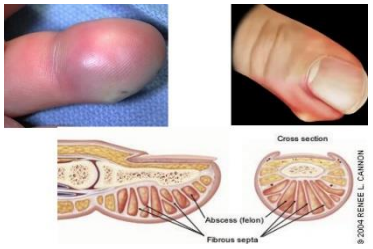
Chronic Paronychia



- **Definition**: Gradual onset of inflammation involving the nail folds, persisting for over 6 weeks.
- **Etiology**:
  - Often caused by fungal infections (*Candida albicans*).
  - Exacerbated by repeated exposure to moisture and irritants.
- **Presentation**:
  - Mild pain, swelling, and redness.
  - Nail may become thickened or discolored.
- **Pathophysiology**:
  - Persistent moisture softens the nail fold, allowing fungal colonization.
- **Treatment**:
  1. Avoidance of prolonged moisture exposure.
  2. Topical antifungals (*e.g.*, clotrimazole).
  3. Steroids for inflammation in combination with antifungal therapy.

---

2. Felon (Pulp Abscess)

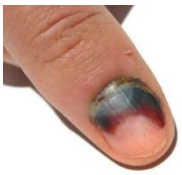


- **Definition**: Abscess in the pulp space of the fingertip.
- **Etiology**:
  - Bacterial infection, often *Staphylococcus aureus*.
- **Presentation**: Severe throbbing pain, swelling, erythema, and warmth localized to the fingertip.
- **Pathophysiology**: Compartmentalized infection in the fibrous septa of the fingertip pulp, can lead to necrosis, osteomyelitis, tenosynovitis, septic arthritis
- **Treatment**:

1. **Incision and drainage**: Careful approach to avoid neurovascular damage. (emergent)
2. Antibiotics for cellulitis or systemic involvement.

---

3. Subungual Hematoma

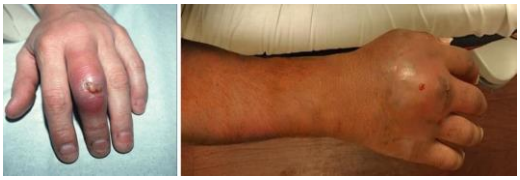


- **Definition**: Collection of blood under the nail plate due to trauma.
- **Presentation**: Painful, discolored nail (red, purple, or black).
- **Treatment**:

1. **Simple Cases**: Nail trephination (electrocautery or needle) for pressure relief.
2. **Complex Cases**: (more than 2/3) Nail removal and nail bed repair for severe injuries or fractures.

---

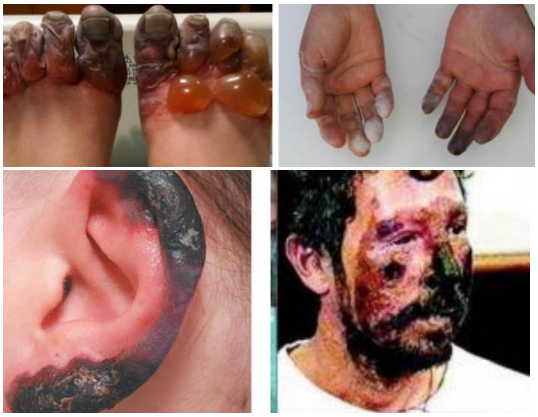
4. Human Fight Bite (Fist Injury)



- **Definition**: Penetrating injury over the knuckles caused by punching another person.
- **Etiology**: Contamination with oral flora (e.g., *Eikenella corrodens*, anaerobes).
- **Presentation**: Pain, swelling, erythema, and puncture wound over knuckles.

---

5. Frostbite



- **Definition**: Tissue injury caused by freezing temperatures (subzero), leading to ischemia and necrosis.
- **Presentation**: Numbness, pale skin, and blistering in severe cases.
- **Pathophysiology**:
  1. **Ambient Temperature:** Ice crystals form, causing cell damage and ischemia.
  2. **Rewarming Phase:** Rewarming restores circulation but causes reperfusion injury.
  3. **Complete Rewarming:** Reperfusion leads to oxidative stress and thrombosis.
     Thromboxane A2: exacerbating damage.
     Presentation: Swelling, blisters.
     Post-Rewarming: Tissue repair or necrosis.
- **Treatment**:
  1. **Rewarming**: Warm water immersion (42°C >).
  2. Analgesics for pain.
  3. Debridement or Amputation → only if demarcated
  4. Thrombolytics, free radical scavengers for severe cases, topical thromboxane inhibitor

---

6. Trench Foot

- **Definition**: Non-freezing cold injury caused by prolonged exposure to wet, cold conditions. (> 2°C)

DONE BY: Mariam Qussay