Clinical types of wounds & wound healing

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Clinical types:

1– Incised wounds:
- Produced by sharp objects: knives, glasses. Or by hitting between two hard objects. They have healthy clean cut edges.

2– Stab (punctured) wounds:
- Produced by pointed instruments such as knives they are small & deep it may extend to involve important organs (liver – intestine – heart etc…….)
3– Contused & Lacerated wounds
Caused by blunt instruments, car accident, machines etc..........
They have irregular outlines e devitalized edges; important vessels & nerves could be injured.

4– Gunshot wounds
They are lacerated, penetrating wounds e entry & exit.
These wounds are small but with extensive destruction around the track.
The amount of tissue damage depends on the missile velocity.
1– Healing by first intention:
Occurs in sutured wounds in absence of infection.
The wound heals in 1–2 W. Leaving a linear scar with minimum contraction & disfigurement.

2– Healing by secondary intention:
Occurs in gapped & lacerated wounds.
The healing process is slow (3 – 4 W) e scarring, contraction & deformity.
A highly integrated series of vascular, cellular, biochemical and physical events constitutes to

The 3 main steps of wound healing:
- Lag phase
- Proliferation phase
- Remodeling phase
(1) Lag phase:

- It takes 2 – 5 day’s in clean wounds & may extend up to 3 weeks in septic wounds.
- In this period, the wound has no tensile strength & the integrity depends entirely on the sutures.
- There is an acute inflammatory reactions which leads to: “hyperaemia with infiltration with polymorphs & Macrophages”
- Removal of debris
- Sealing the wound e plasma clot & fibrin strands.
The inflammatory reaction is initiated by **substances** released from damaged tissues such as:

- Histamine
- Serotonin
- Prostaglandin
- Kinins (Leukotoxine – bradykinin)

* Epitheliazation occurs by:

- Migration of the marginal **basal cells** of epidermis across the defect.
- After bridging the defect with basal cells, **other layers** of epidermis occurs & the surface cells **Keratinize**.
(2) Proliferation phase:

- lasts up to **3 weeks** in clean incised wounds.
- During this phase, **the tensile strength** is less than 10% of normal.
- This phase is **characterized by:**
  - Capillary budding
  - Fibroblastic proliferation
  - Deposition of collagen
The capillary budding & fibroblastic proliferation forms a **new framework** of young vascular tissue known as “Granulation tissue”

By the **4th or 5th week**, the granulation tissue is replaced by deposition of “tropocollagen” in a ground substance of *mucopolysaccharides, mucoproteins & glycoproteins*.

Finally, Maturation of collagen is completed by **aggregation & polymerization of tropocollagen molecules**.
The collagen molecule has abundant qualities of two unique amino acids, hydroxyproline and hydroxylysine. The hydroxylation process that forms these amino acids requires ascorbic acid (vitamin C) and is necessary for the subsequent stabilization and cross-linkage of collagen. Procollagen is formed within the fibroblasts as a long linear amino acid segment with regular repeats of hydroxyproline every third amino acid and with terminal extension peptides. Procollagen molecules aggregate in the case of type 1 collagen (the most common) as three a chains to form a triple-helical complex. The triple helix is maintained by intramolecular disulfide bonds between specific cystine residues. Procollagen is secreted in its triple-helical form; extracellular peptidases cleave the extension peptides at the amino and carboxy termini, leaving the central collagen molecule. Collagen cross-linking then occurs in the extracellular space as the collagen molecules aggregate into larger structures.
Although there are many types of collagen, type I predominates throughout the body. The principal collagen in scar is type I, with lesser amounts of type III collagen present.

Other collagen types make important contributions to the basement membrane, cartilage, and other structures.
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(3) Remodelling phase:

- Starts after the 3rd week & continue for several months.
- The wound gradually regains its tensile strength (*by 9th month, 75% of tensile strength is regained*).
- The final scar is much smaller than the original wound (wound contraction).
- During this phase, there will be proper orientation & weaving of the individual fibers of the collagen bundles.
Wounding time 0 hour

- Fibrin
- Platelets
  - ADP
  - TXA$_2$
- Disrupted collagen
- Red blood cells
- Disrupted blood vessel
Healing of bone occurs in the same three phases.

The duration of healing differs according to the location of the fracture and its nature.

During the proliferation or reparative phase, the hematoma is replaced by special granulation tissue called the callus, which is composed of fibroblasts, endothelial cells, and bone forming cells (chondroblasts and osteoblasts).

Fibroblasts deposit a collagen matrix, and chondroblasts deposit proteoglycans in a process called chondral bone formation.
Osteoblasts converts chondral bone into bone by condensation of hydroxyapatite on specific points of the collagen fibres.

Bone repair occurs through primary or secondary intention.

Primary intention occurs only with rigid plate fixation.

Unlike soft tissue, bone heals without leaving a scar.
(*) Factors affecting wound healing:

1. Local Factors:
   - Poor vascularity.
   - Impaired venous or lymphatic drainage.
   - Movement of the injured limb
   - Presence of necrotic tissue
   - Infection
(2) General factors

- Hypoproteinaemia
- Ascorbic acid ↓↓ (Vit C) essential for maturation of collagen
- Cortisone administration delays the early cellular maturation.
(1) Haematoma:

Wound Haematoma means collection of blood & clots in the wound.

Causes:
1. It is almost caused by imperfect haemostasis.
2. Also patient receiving aspirin or small doses heparin have a high risk of developing haematoma.
3. Patients with hypertension.

Haematomas produce elevation & discoloration of wound edges, discomfort & swelling.

Blood may leak through sutures.
Wound haematomas of particular importance that occurs after thyroidectomy with pressure on carotid A and larynx.

Blood collects under the deep cervical fascia, compress the trachea → suffocation.

In this case, blood should be evacuated immediately even without anesthesia.
Small haematoma may be reabsorbed but it ↑ the incidence of infection.

Treatment of **early** (24 h.) wound haematoma is **evacuation** under sterile conditions, ligation of the bleeding vessels & re–closure of the wound.

**Late** haematoma is evacuated by opening the wound & compression by packing of wound edges.
(2) Seroma:

- Seroma is a fluid collection in the wound other than pus or blood.
- Seroma fluid has a higher H+ & CO2 conc. and lower O2 & globulin conc. than serum.
- Seroma usually follow operation involving elevation of skin flaps, with transection of numerous lymphatic channels as in mastectomy – repair of large hernias …..etc.
Seroma if collected should be evacuated by needle aspiration.

Seroma if not evacuated will delay healing & ↑ the risk of wound infection.

After aspiration, tetracycline 1g/150ml saline can be injected into the space for 45 min as sclerotherapy & disinfection, then it is re-aspirated followed by pressure.

Large serum may need surgical drainage and compression to prevent re accumulation.
(3) Wound Dehiscence

- It means partial or total disruption of any or all layers of the operative wound.
- In abd. wounds, it is called Burst abdomen.
- Incidence 1–3% of all abd. wounds.
- Mortality rate 10%.
- Peak incidence: 6–8 th postop. day.
Predisposing factors:

1) **Preop. causes (patient’s fault):**
II) Operative causes: surgeon’s fault:

- Aseptic surgery
- Operations for peritonitis
- Poor surgical technique: e.g.
  - To tight or too loose sutures.
  - Bad selection of the sutures.
• III) Post operative (nurses & surgeon’s fault)

• Abd. Distension (gastric – paralytic ileus)
• Wound infection & haematoma formation.
• Premature removal of deep tension sutures.
Types & Treatment of Wound Dehiscence

1– Superficial burst:
Gaping of skin & subcut. tissue only after removal of sutures (2 weeks after surgery).

Treatment:
- Evacuation of blood clots
- Let the wound open to heal by 2nd intention.
- Give antibiotics.
2– Deep burst:

- Separation of deep layers of the ant. abd. wall except the skin.
- Incisional hernia will be the result.
- If the condition is recognized in the hospital, delay removal of sutures + abd. Corset; then treat the incisional H as usual.
3– Complete (Burst abd.)

- Occurs gradually → serosanguinois discharge from wound edges at 7–10 th. days.
- Or suddenly with protrusion of a loop of intestine or omentum through the wound.
- It needs urgent re-operation under general anesthesia with non–absorbable nylon sutures with interrupted tension sutures.
- Skin may be included or left open & heal with 2nd intention.
4– Wound infection:

Wound infection remains the commonest of all post operative complications.

It is best defined as the presence of pus in the wound which discharges spontaneously or following opening of the wound.
Late Complications

- Incisional hernia
- Contracted scar
- Hypertrophic scar
- Keloid