Repair, part II

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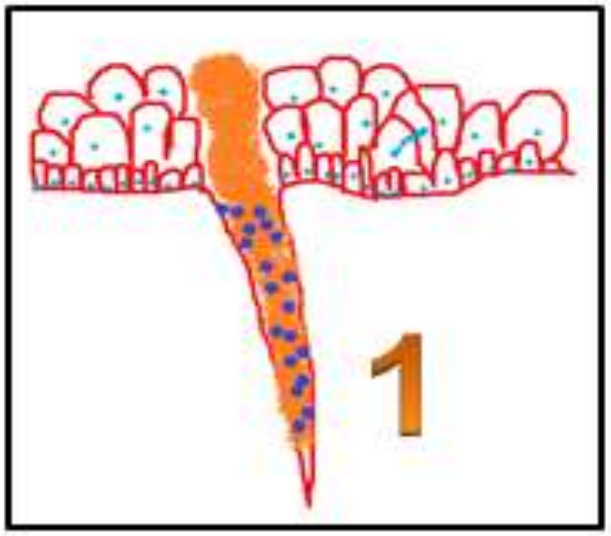
HEALING OF THE WOUNDS

I- Healing by first intension

It occurs in clean incised wound with minimal tissue destruction with approximated edges as in surgical wounds.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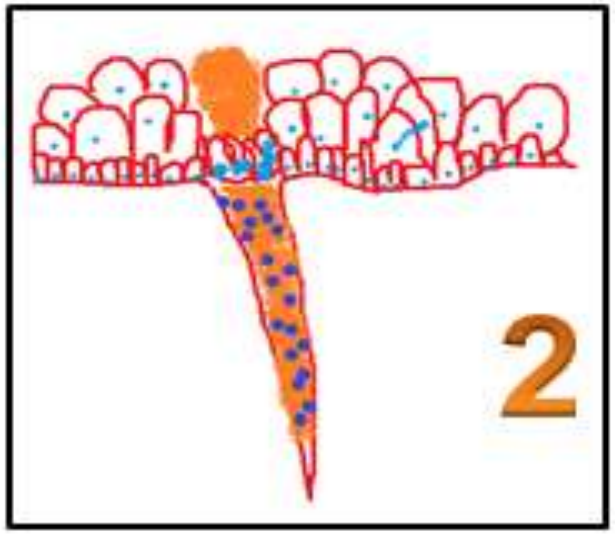
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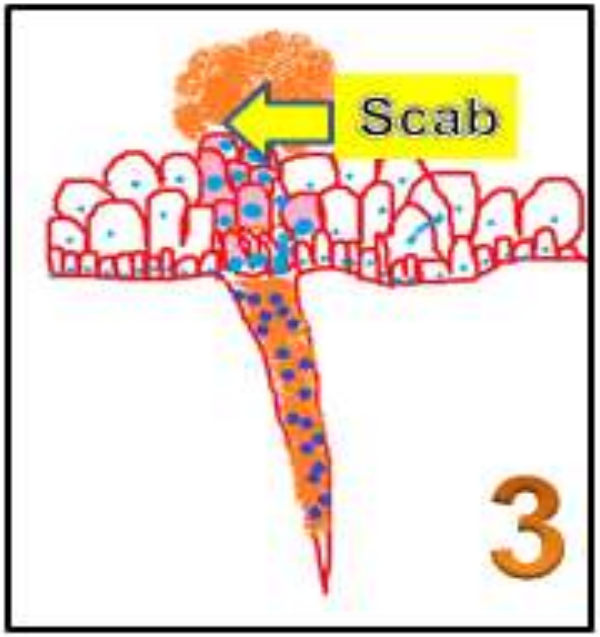
Scab
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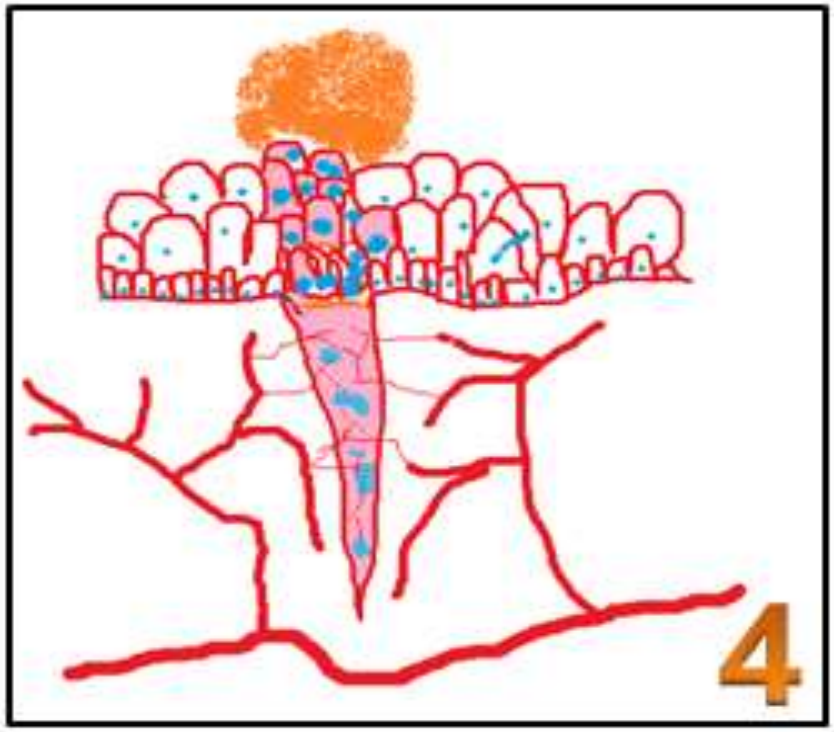
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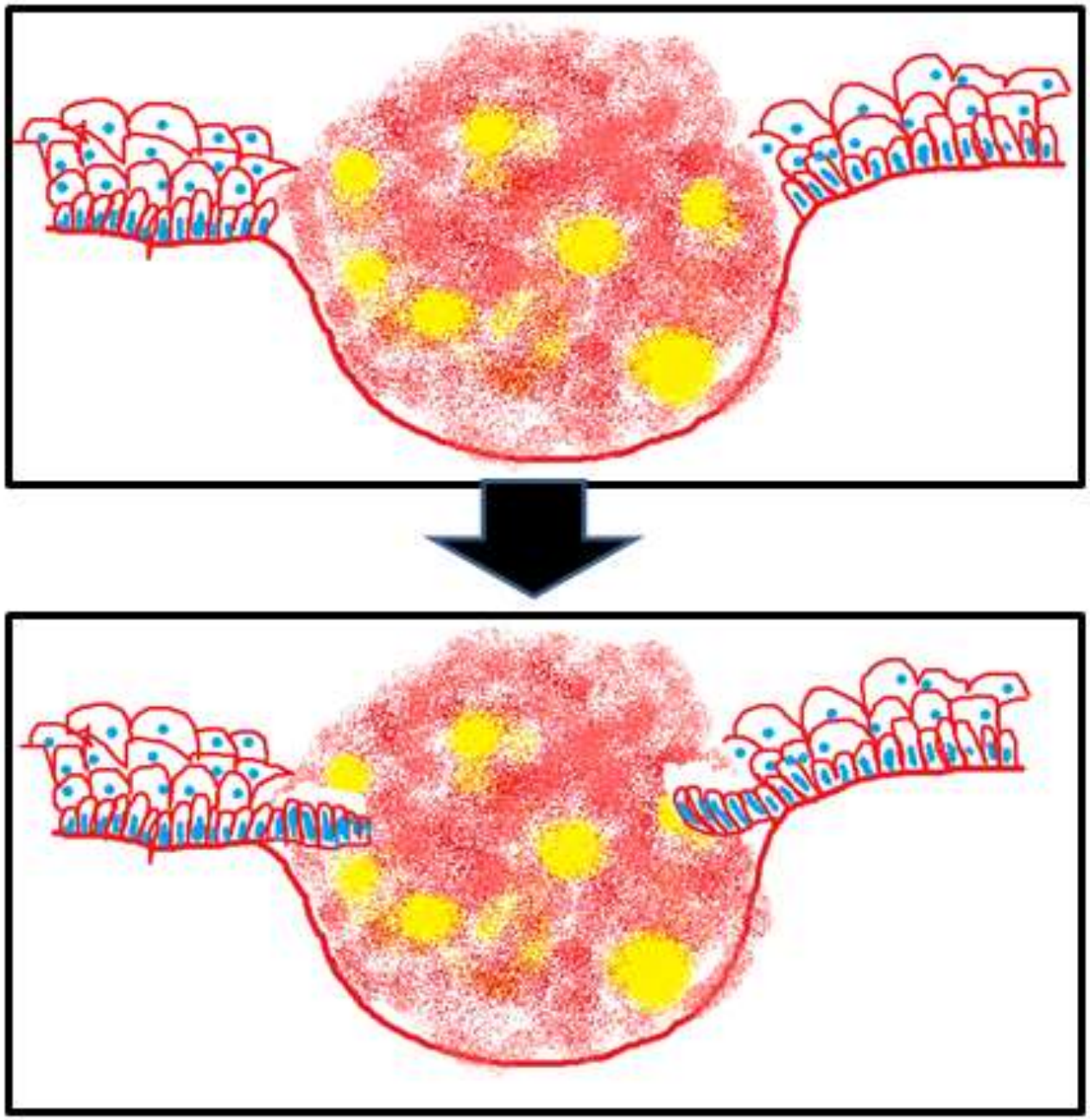
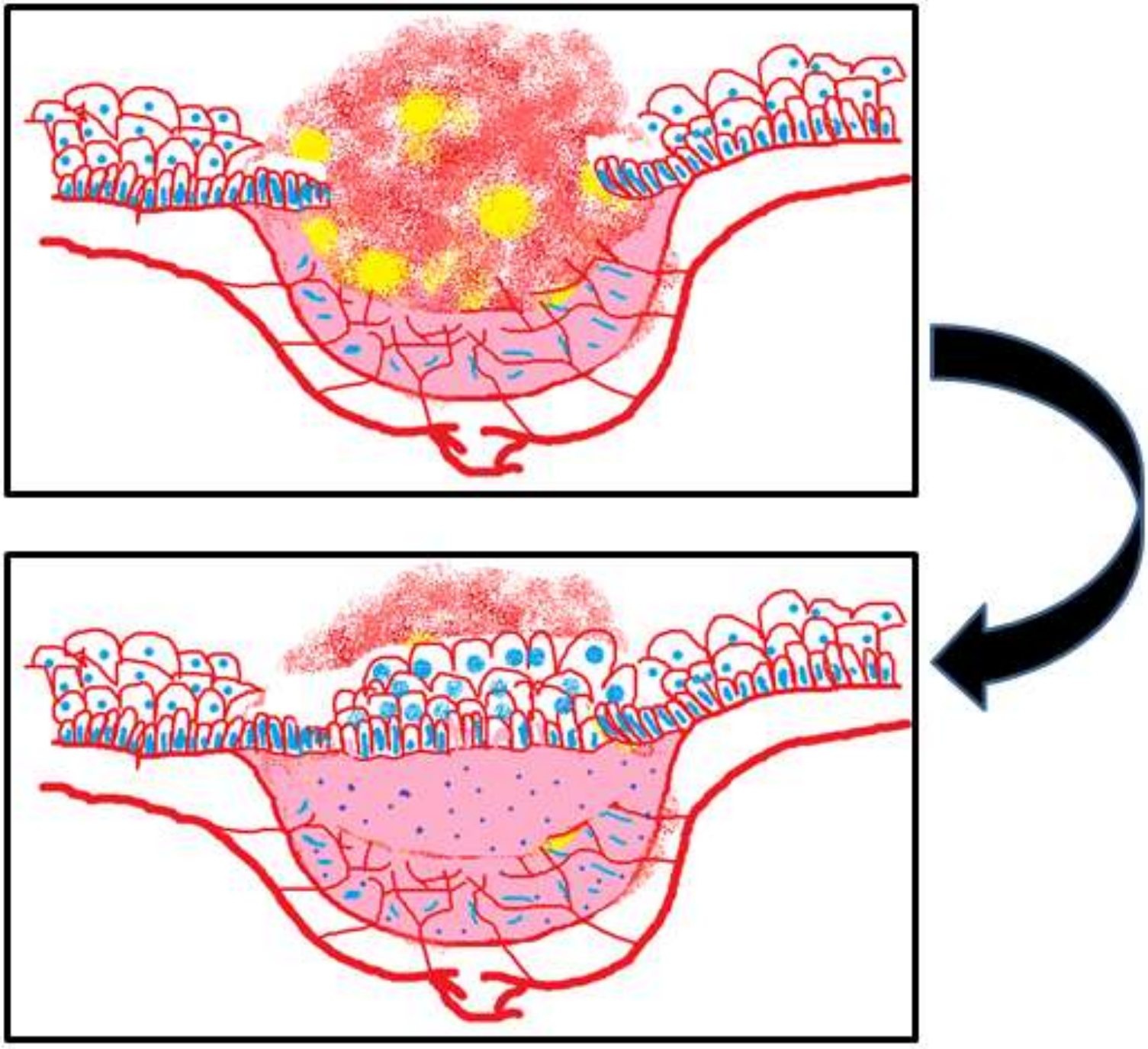
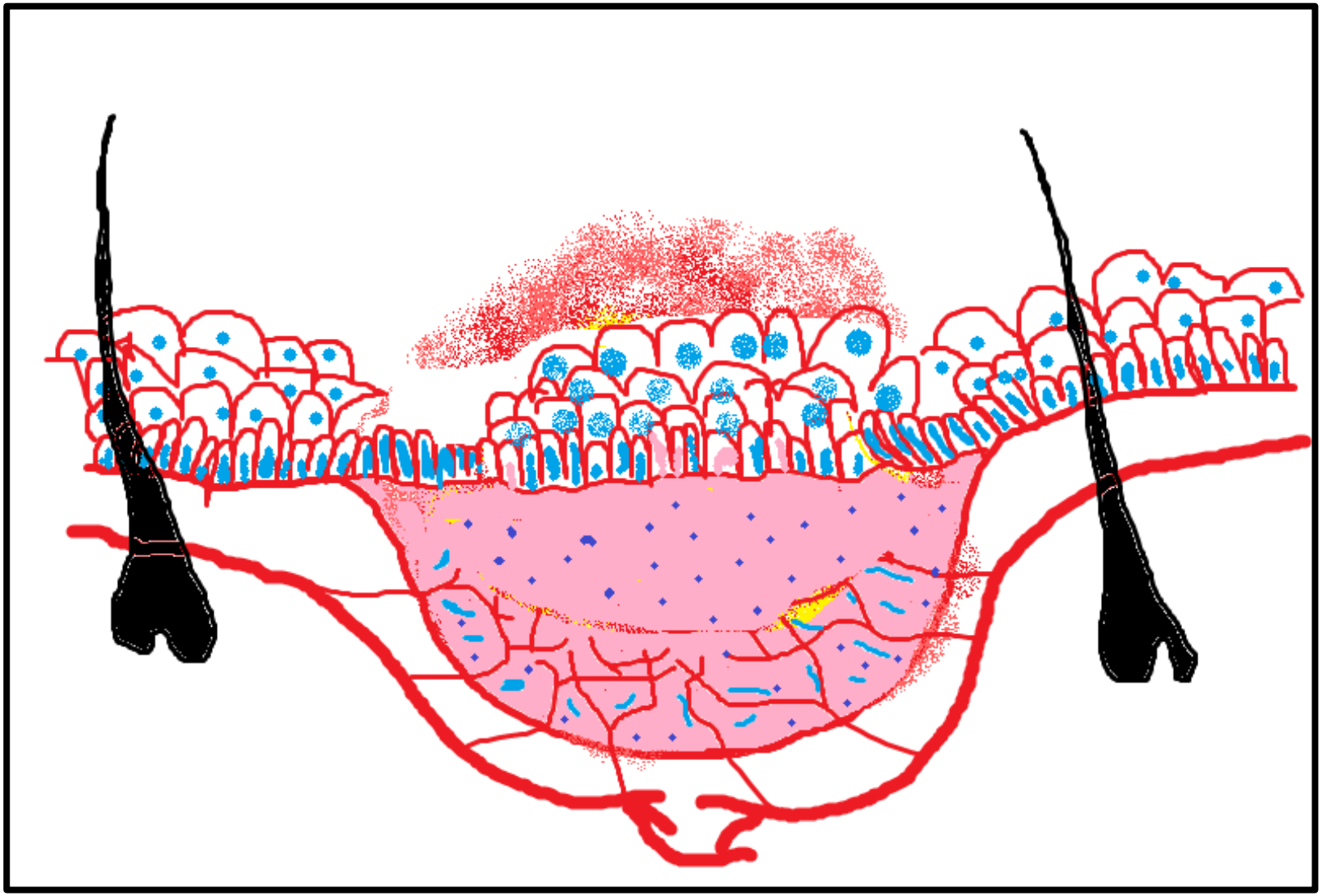
- Blood is clotted between wound edges and on the surface.
- The incision cause mild acute inflammation in the edges of the wound. Products of inflammation are rapidly removed by macrophages.
- The basal cell layer of the epidermis on both edges of the wound proliferate across the clot and meet in the center. Then they divide to form the whole thickness of the epidermis.
- Dermal adenexa don't proliferate. The remnants of clot on the surface called scab. The scab separates within 10-14 days.
- The gap of the wound under the new epithelium get filled by granulation tissue originating from both edges of the wound.
- Maturation of granulation tissue to fibrous tissue occurs.

II- Healing by secondary intension

It occurs in gaping wounds, septic wounds or abscesses.







- The gab of the wound get filled by blood clots, necrotic material and/or pus.
- The epidermal cells at the margins proliferate across the blood clot but they don't cover the wound until the gap get filled by granulation tissue up to the level of the basal cell layer of the epidermis.
- The gap get filled from below upwards and from sides.
- Neutrophils, macrophages and inflammatory fluid exudate appear in the meshes of the granulation tissue to deal with any remaining infection.
- The basal cell layer around the cavity proliferates to cover the formed granulation tissue and divide to form the whole epidermal thickness.
- Dermal adenexa don't regenerate.
- The granulation tissue mature to fibrous tissue from the periphery to the center.

Control mechanisms in repair:

- **Growth factors:** which promote cell division; they include: platelet derived growth factors, epidermal growth factors, macrophage derived growth factors and fibroblast growth factors.

- **Removal of chemical factors inhibiting mitosis:** these factors are called Chalones. They are formed by living cells and inhibit mitosis in the neighboring cells of the same type. Tissue destruction results in lack of Chalones locally, so adjacent living cells can proliferate.

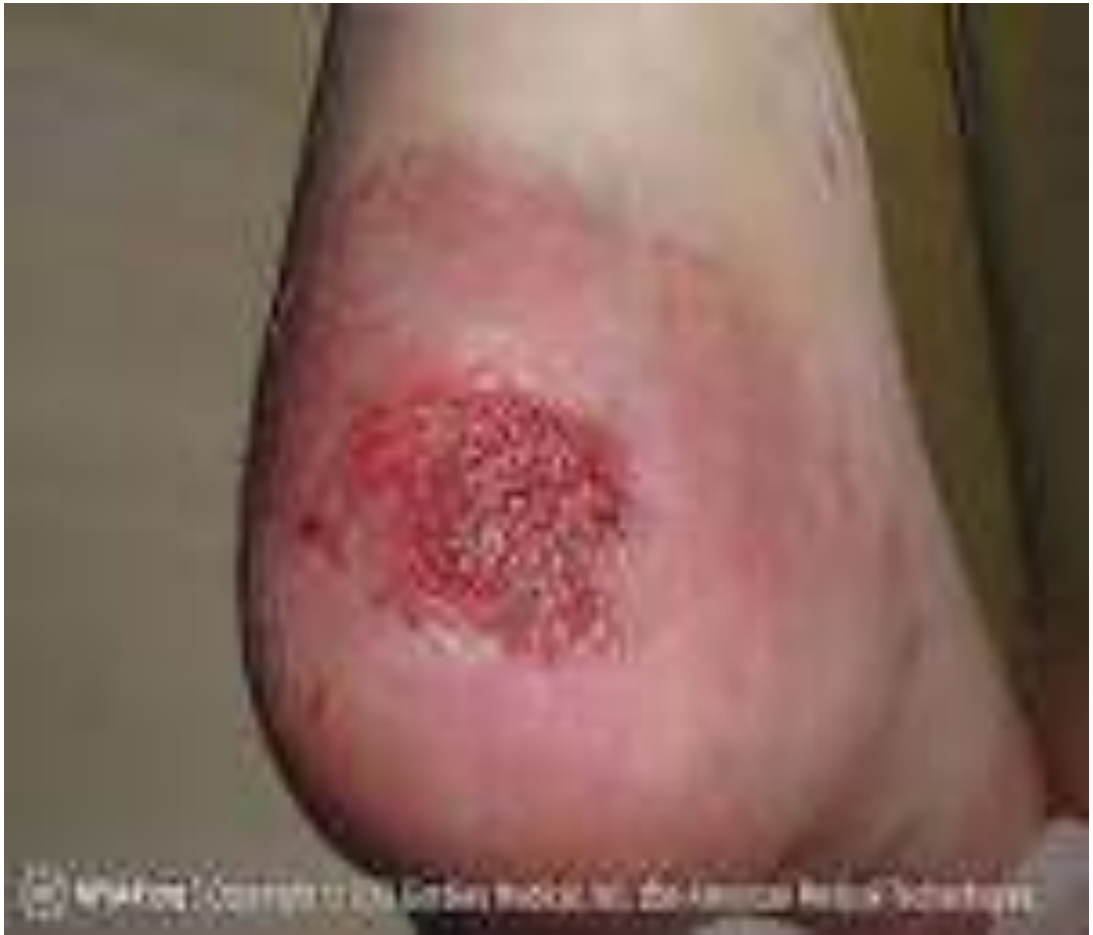
- **Removal of contact inhibition:** massages passing between cells in close contact inhibit cell motility. In wounds, due to absence of contact inhibition, cells can migrate to cover the wound surface.

- **Availability of good blood supply.**

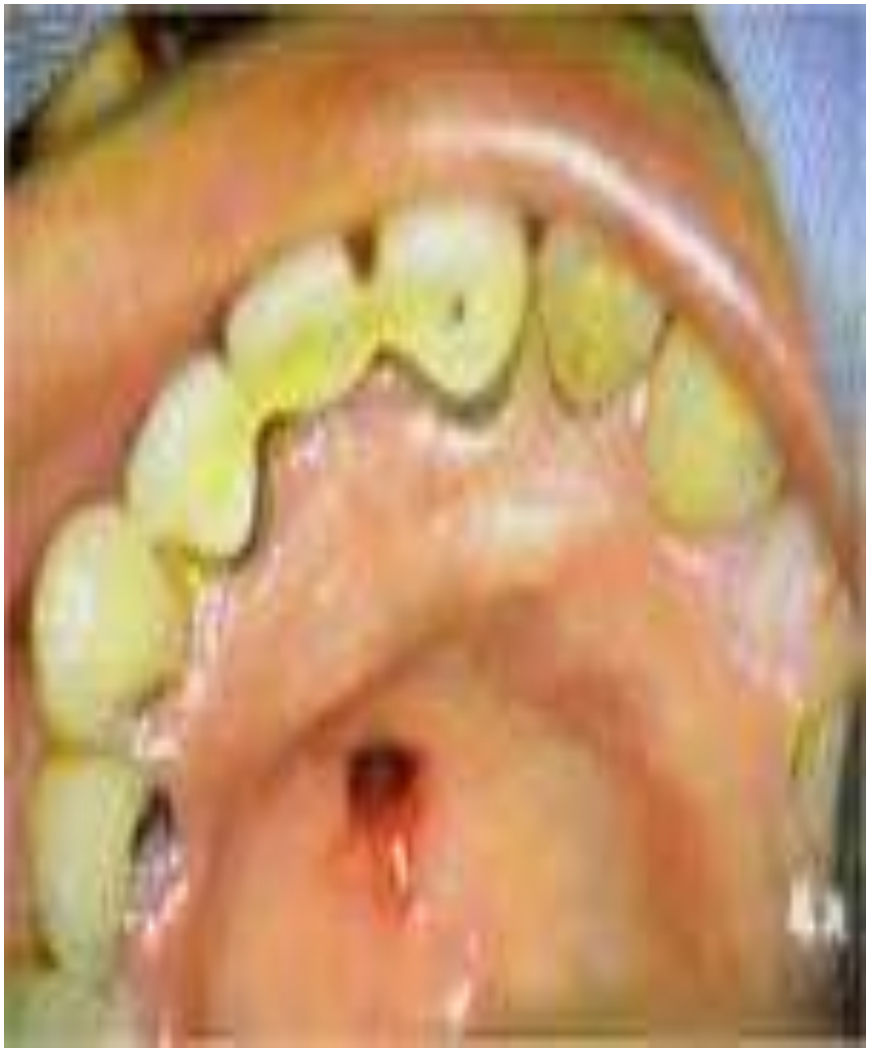
- **Presence of supporting framework for the regenerating cells.**

COMPLICATIONS OF REPAIR

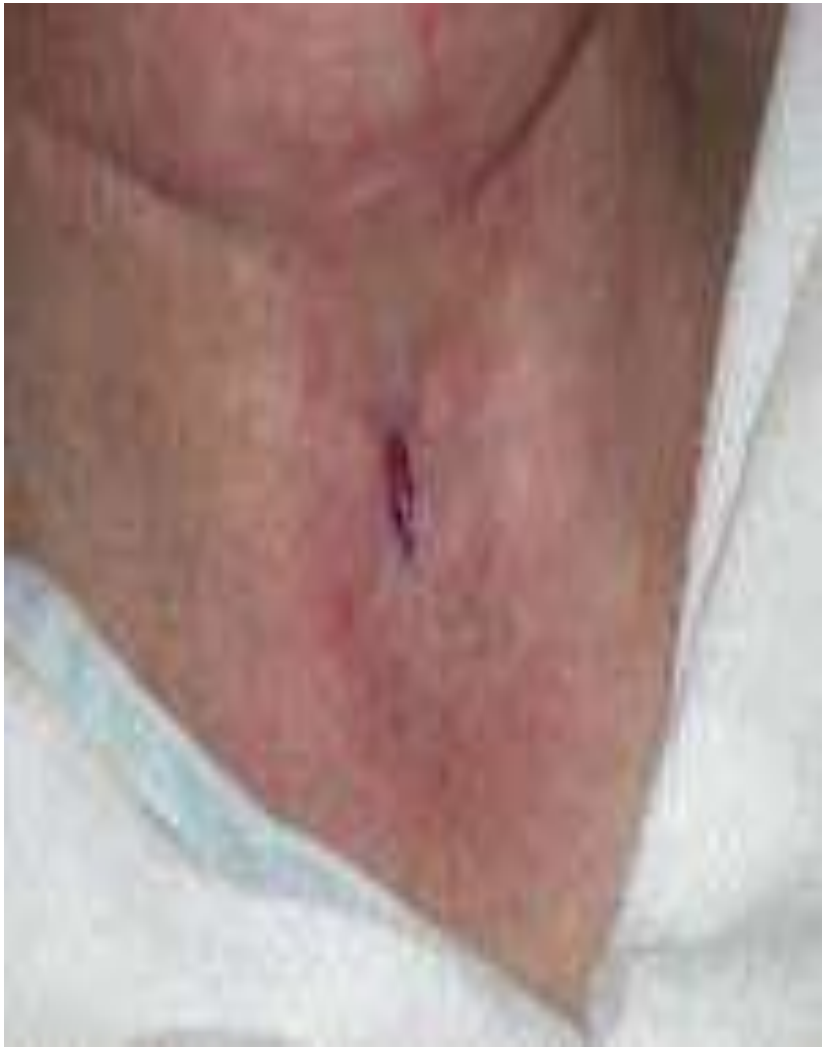
•Ulcer: means permanent loss of surface epithelium of skin or mucous membrane.



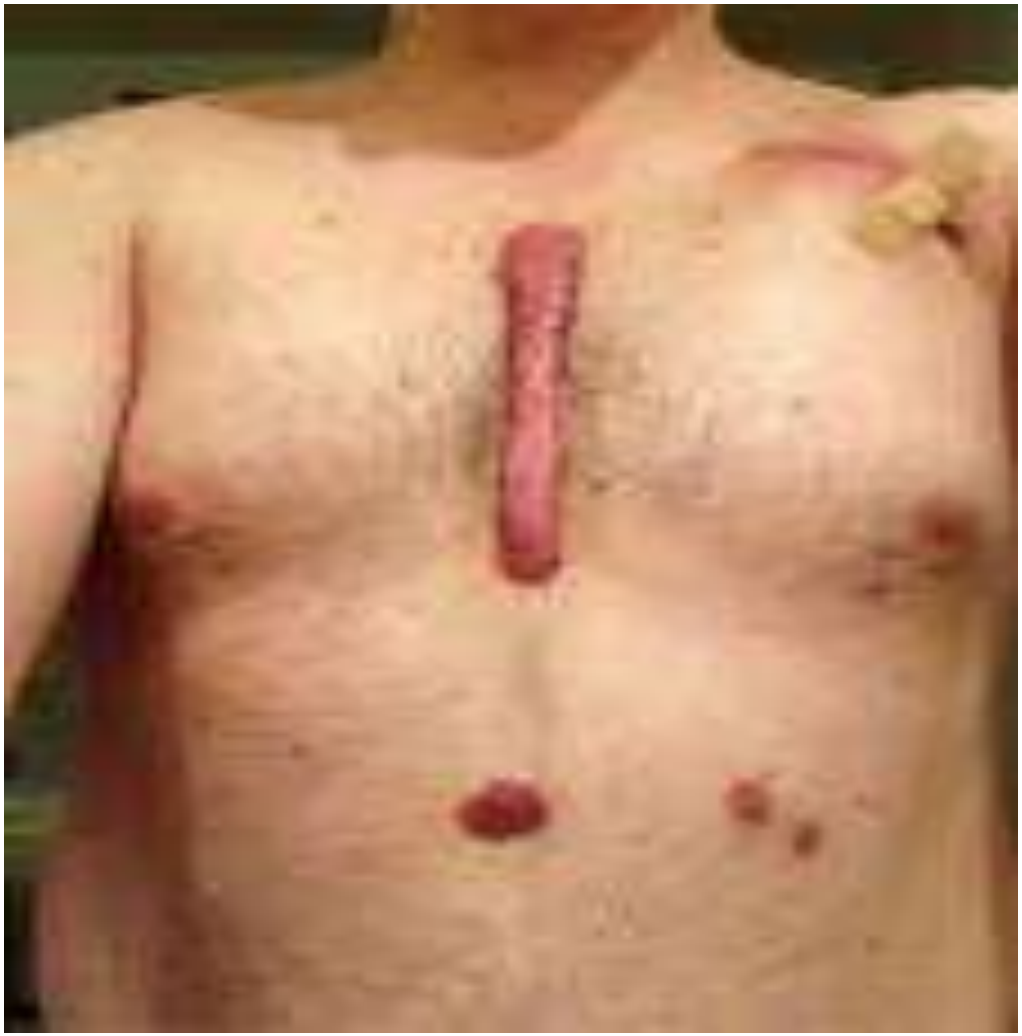
- Sinus: blind-ended tract between depth of wound or abscess cavity and skin surface.



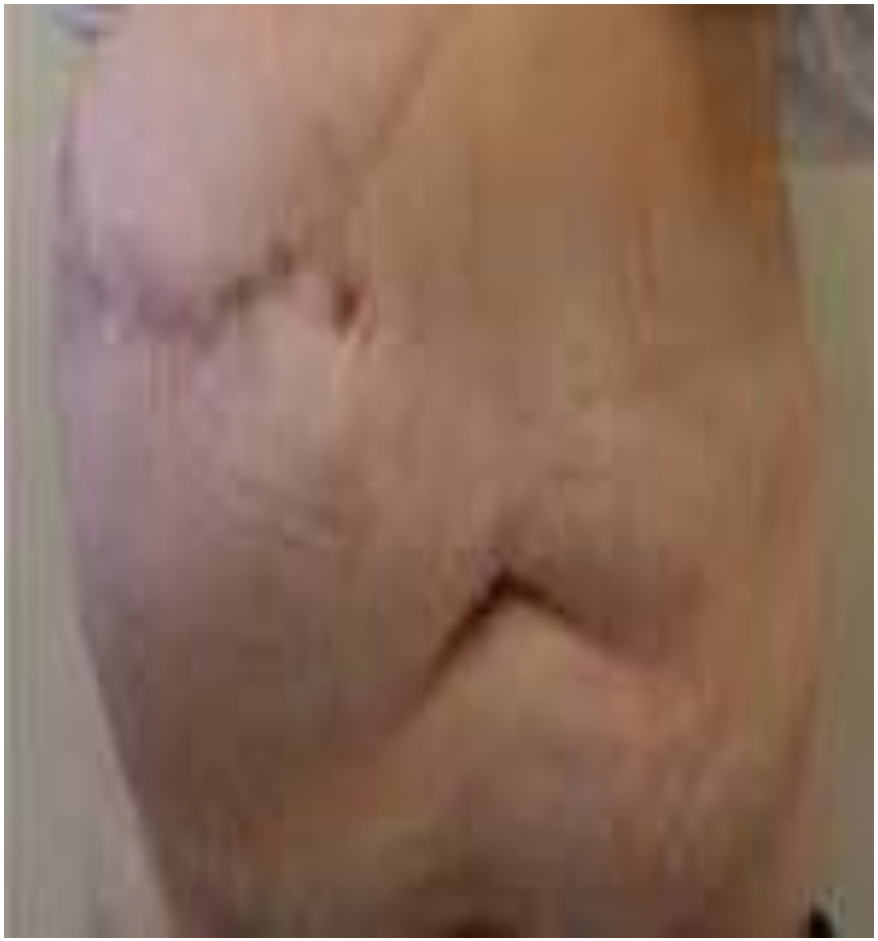
- Fistula: a tract between abscess cavity and hollow organ or two hollow organs.



- Keloid: large scar projecting on the surface due to overdone repair.



- Weak scar leading to incisional hernia.



- Delayed healing.
- Implantation (epidermoid) cyst: epithelial cells trapped in the wound during healing and then proliferate forming an epidermoid cyst.
- Squamous cell carcinoma.


THANK YOU