27 **Describe primary healing ie sutured wound**

Blood clot of fibrin and blood cells fills the gap, surface dries to form a scab

- 24 hours
  - neutrophils appear at wound margin
  - mitosis of basal cells of epidermis at edges of wound
- 24 – 48 hours
  - spurs of epithelial cells from wound edges migrate over cut margins of dermis beneath scab to form a thin epithelial layer
- day 3
  - macrophages replace neutrophils
  - granulation tissue invades the gap
  - epithelial cell proliferation forms a thick epithelial layer
- day5
  - neovascularization is maximal
  - abundant collagen fibrils begin to bridge the gap
  - differentiation of epithelial cells forms a mature keratinized epidermal covering
- second week
  - collagen and fibroblast proliferation prominent
  - regression of vascular channels and disappearance of acute inflammatory changes
- by 4 weeks
  - scar of connective tissue without inflammatory exudate covered by intact epidermis
  - tensile strength takes months to reach maximum
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28 **Describe Healing by secondary intention**

- Where a large defect is present.
- Differences from primary healing;
- inflammatory reaction is more intense
- more granulation tissue is formed
- wound contraction occurs due to myofibroblasts
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29 **Complications of Wound healing**

- Inflammation and repair impaired by;
  - Protein deficiency

- Vitamin C deficiency
- Glucocorticoids
- Infection
- Mechanical factors (tension causing dehiscence)
- Poor blood supply (atherosclerosis, venous abnormalities)
- Foreign bodies

- Abnormalities of growth;
  - Keloid (excessive collagen)
  - Exuberant granulation or proud flesh (excessive granulation tissue)
  - Desmoids or aggressive fibromatoses (excessive fibroblasts)
- Chronic inflammatory fibrosis; (eg. Rheumatoid arthritis, lung fibrosis, cirrhosis)
  - Due to
    - persistence of initial stimulus for fibroplasia
    - immune reactions with lymphocyte-monocyte interactions (and sustained secretion of growth factors, fibrogenic cytokines proteolytic enzymes etc)
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