Physiology of wound healing

wound healing process occurs in sequential yet overlapping phases , 3 or 4 phases.

1-inflammatory stage:

 An incision made through a full thickness of skin causes a disruption of the micro vasculature and immediate hemorrhage following incision ,vasoconstriction of small vessels in the wound , mediated by epinephrine , norepinephrine ,prostaglandins , serotonin and thromboxane .vasoconstriction causes temporary blanching of the wound and functions to reduce hemorrhage immediately following tissue injury. Histamine released from platelets and circulating mast cells increases vascular permeability and indirectly stimulates vasodilatation through production of prostaglandins which causes vasodilatation through the activation of adenylyl cyclase pathway . the inflammatory reaction is localized to an area immediately surrounding the injury , soon after injury , leukocytes in local vessels adhere to the endothelium , within 30 -60 minutes , they begin to move through the gaps in the vessels wall and eventually concentrate at the site of wound. Neutrophils are predominate cell type for the first 48 hours after wounding , they cleanse the wound site of bacteria and necrotic materials . neutrophils release inflammatory mediators , also monocytes appear in the area , neutrophils are short-lived cells compared with monocytes , therefore monocytes predominate in older wounds , monocytes may coalesce to form multinucleated giant cells , which are also phagocytic . macrophages are important cells in the early phase of wound healing , they phagocyte debris and bacteria secrete collagenase which breakdown injured tissue and release cytokines .cytokines released by macrophages stimulate the chemo taxis and proliferation of fibroblasts and smooth muscle cells . lymphocytes also found at site of inflammation (usually in chronic inflammatory reactions).

Duration of inflammation depends on :

1-degree of contamination .

2-degree of tissue damage .

2-Repair stage.

 a-fibroblastic phase:

fibroblasts originate from local mesenchymal cells , particularly those associated with blood vessel adventitia . fibroblasts are critical component of granulation tissue and are responsible for production of collagen, elastin,fibronectin ,glycosaminoglycans and proteases . growth of fibroblasts in the wound occurs as the cells of inflammation decrease in number. Skin fibroblasts and mesenchymal cells differentiate to perform migratory and contractile capabilities . collagen is secreted to the extracellular in the form of procollagen . which then transformed to collagen fibrils , which in turn aggregate to form collagen fibers . elastin is also present in the wound in smaller amounts. Elastin is structural protein with random coils that allow for stretch and recoil properties of the skin.

b-epithelialization phase.

Epithelialization is the formation of epithelium over a denuded surface .it involves migration of cells at the wound edges from one side to the other side ,the epithelial layer provides a seal between the underlying wound and the environment .epidermal basal cells adhere to one another and to the underlying layers of dermis ,intracellular microfilaments are formed ,allowing the epidermal cells to creep across the wound surface , as the cells migrate they dissect the wound and separate the overlying eschar from the underlying viable tissue.

c-contraction .

wound contraction begins almost concurrently with collagen synthesis .contraction is defined as the centripetal movement of wound edges that facilitates closure of a wound defect . wound contraction depends on the myofibroblasts of the extracellular matrix and myofibroblast proliferation .

3-Maturation stage.

During this stage ,collagen fibers becomes mature and more organized .there are gradual reduction of fibroblasts and a corresponding decrease in capillaries with in the wound are the histological features of maturation .maturation stage is characterized by balanced synthesis and degradation of connective tissue components.