



**SNS COLLEGE OF ALLIED HEALTH SCIENCES**  
SNS Kalvi Nagar, Coimbatore - 35  
Affiliated to Dr MGR Medical University, Chennai



**DEPARTMENT OF CARDIOPULMONARY PERFUSION CARE**  
**TECHNOLOGY**

**COURSE NAME: Introduction to Surgery**

**TOPIC : Wound Healing**



# WOUND HEALING

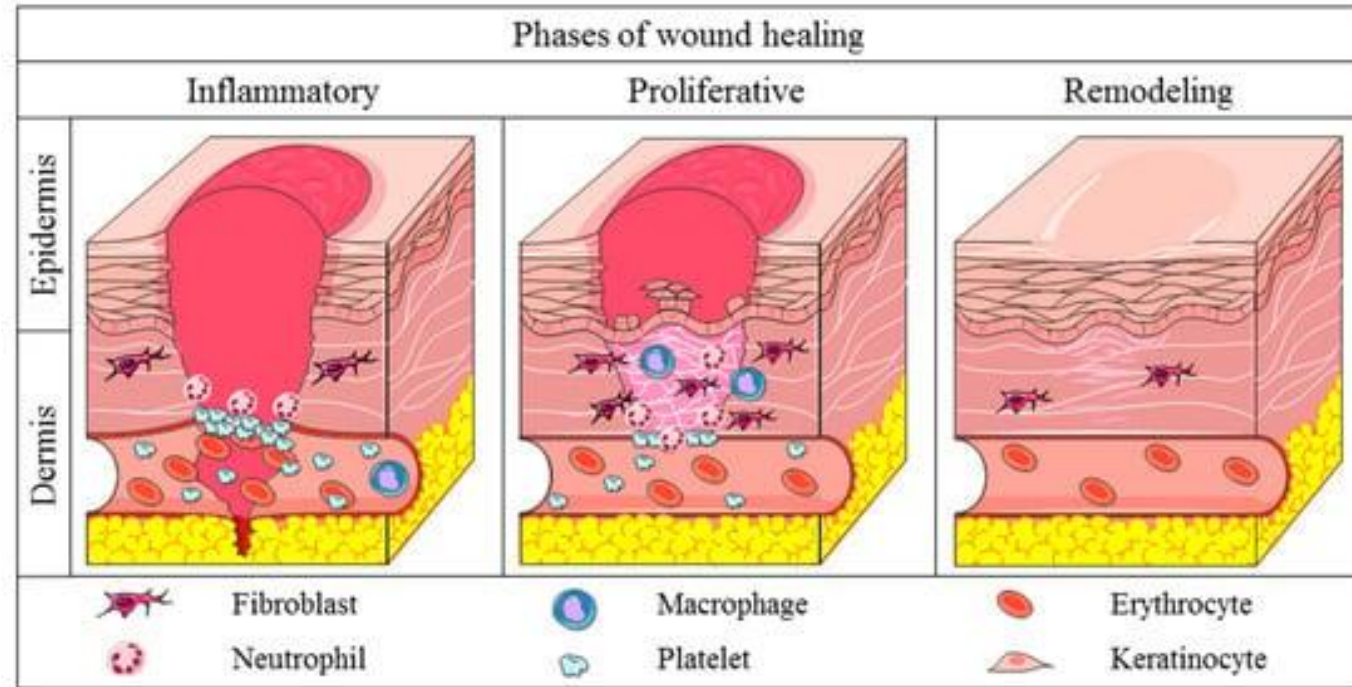


- Wound healing is the **process of repair** that follows injury to the skin and other soft tissues.
- Healing is the interaction of a complex cascade of cellular events that generates resurfacing, reconstitution, and restoration of the tensile strength of injured tissue.
- Under the most ideal circumstances, healing is a systematic process, traditionally explained in terms of 3 classic phases:
  - **Inflammation**
  - **Proliferation**
  - **maturation.**



# PHASES OF WOUND HEALING

- **The inflammatory phase:** a clot forms and cells of inflammation debride injured tissue.
- **The proliferative phase:** epithelialization, fibroplasia, and angiogenesis occur; additionally, granulation tissue forms and the wound begins to contract.
- **The maturation phase / remodeling phase:** Collagen forms tight cross-links to other collagen and with protein molecules, increasing the tensile strength of the scar.





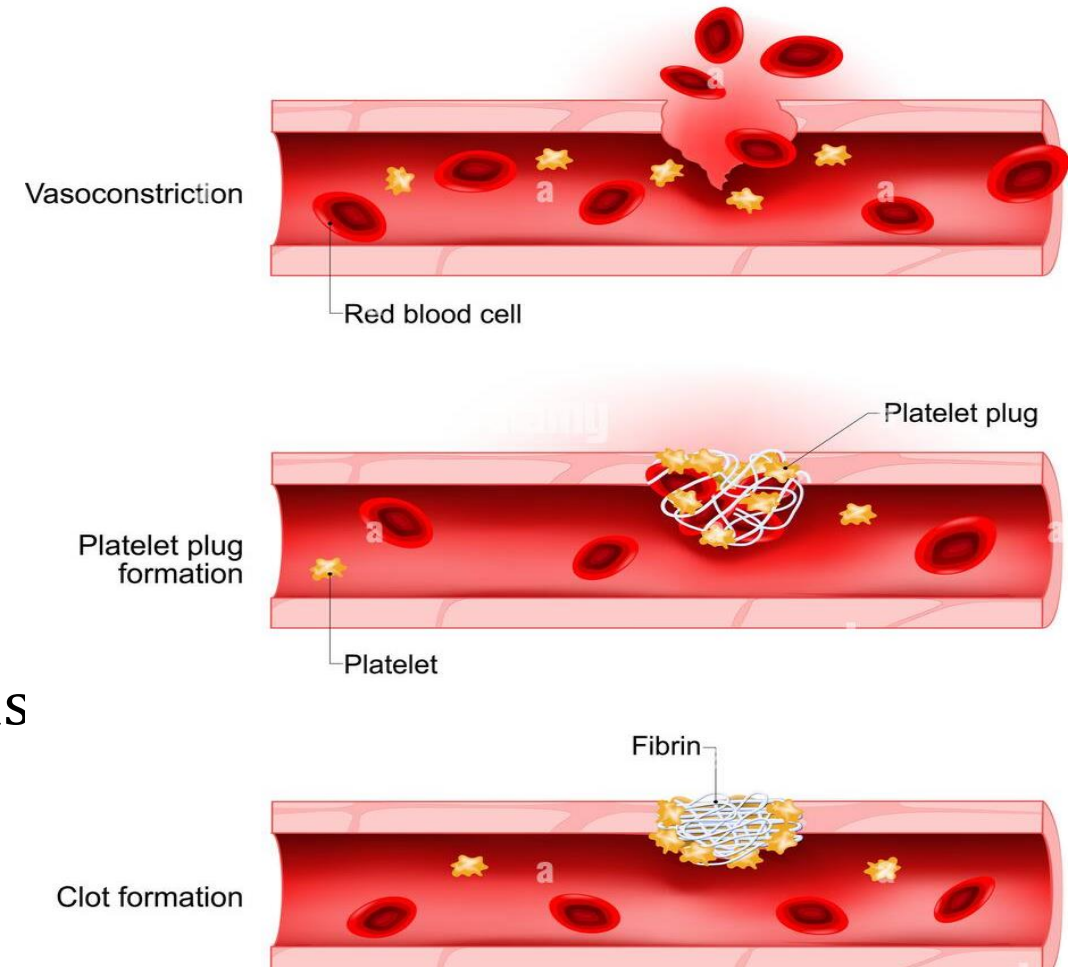
# PHASES OF WOUND HEALING



- **Inflammatory Phase** -----→ Hemostasis & Inflammation  
(Reactive Phase)
- **Proliferative Phase** -----→ Epithelial Migration, Proliferation & Maturation  
(Regenerative / Reparative)
- **Maturation phase** -----→ Contraction of wound, Scarring and Remodelling

# INFLAMMATORY PHASE

- **Immediate to 2-5 days**
- **Hemostasis** – Vasoconstriction, Platelet aggregation, Thromboplastin makes clot
- **Inflammation** – Vasodilation, Phagocytosis





# INFLAMMATORY PHASE

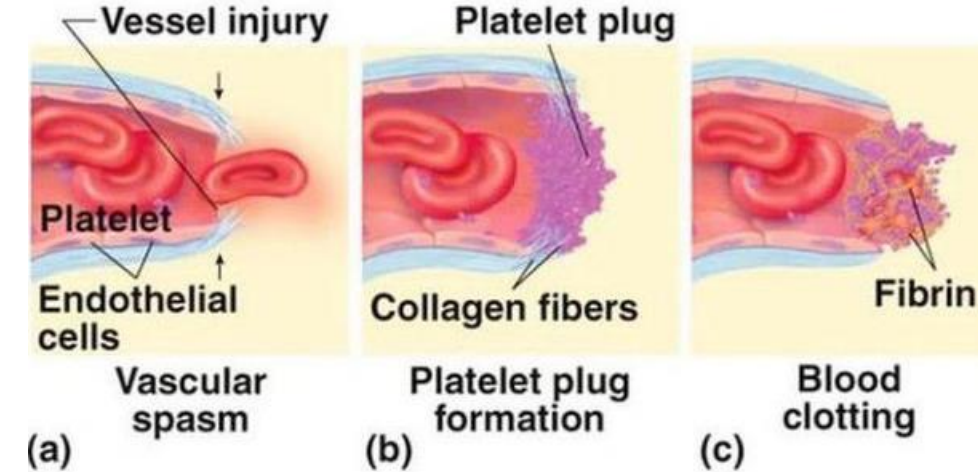


The body responds quickly to any disruption of the skin's surface

The early events of wound healing are characterized by a **vascular and cellular response to injury**

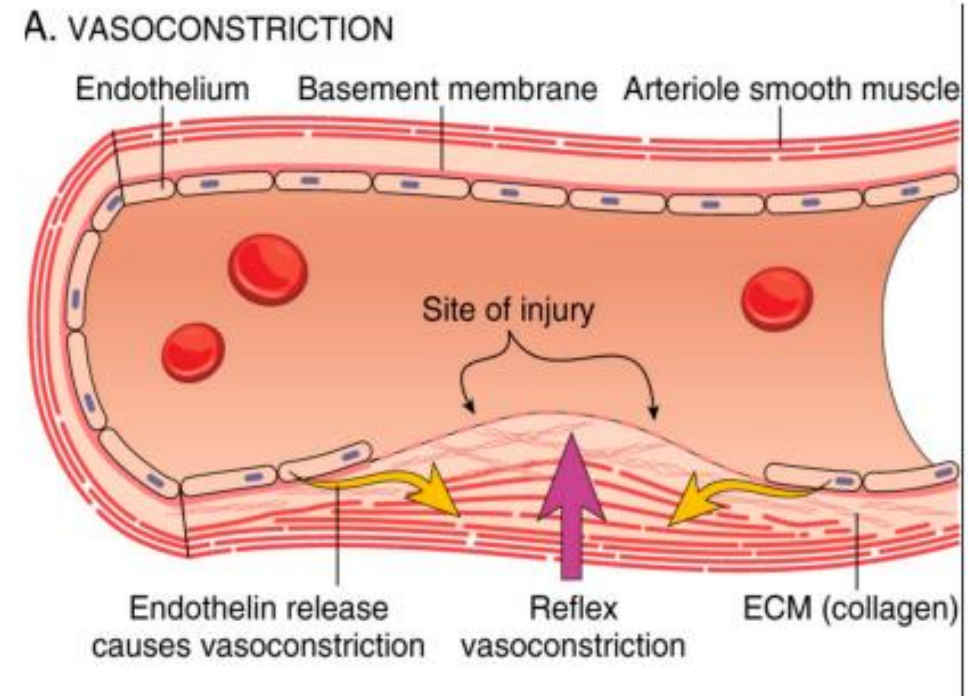
An incision made through a full thickness of a skin causes a disruption of the microvasculature and **immediate hemorrhage**.

Within seconds of the injury, blood vessels **constrict** to control bleeding at the site. **Platelets** coalesce within minutes to stop the bleeding and begin clot formation



# INFLAMMATORY PHASE

- Following incision of the skin, a 5- to 10- minute period of vasoconstriction ensues, mediated by **epinephrine, norepinephrine, prostaglandins, serotonin, and thromboxane.**
- Vasoconstriction causes temporary blanching of the wound and functions to **reduce hemorrhage** immediately following tissue injury, aid in platelet aggregation, and keep healing factors within the wound.
- Endothelial cells retract to expose the subendothelial collagen surfaces; platelets attach to these surfaces.
- Adherence to exposed collagen surfaces and to other platelets occurs through adhesive glycoproteins: **fibrinogen, fibronectin, thrombospondin, and von Willebrand factor.**



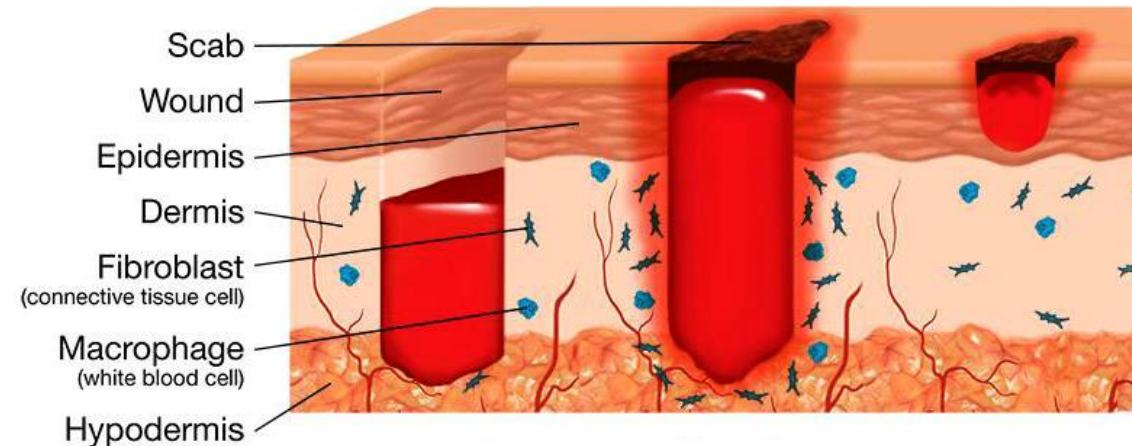


# INFLAMMATORY PHASE



- **Platelets** also release factors that attract other important cells to the injury.
- **Neutrophils** enter the wound to fight infection and to attract macrophages.
- **Macrophages** break down necrotic debris and activate the fibroblast response.
- The inflammatory phase lasts about **24 hours** and leads to the proliferation phase of the healing process.

## Fibroblasts and Wound Healing

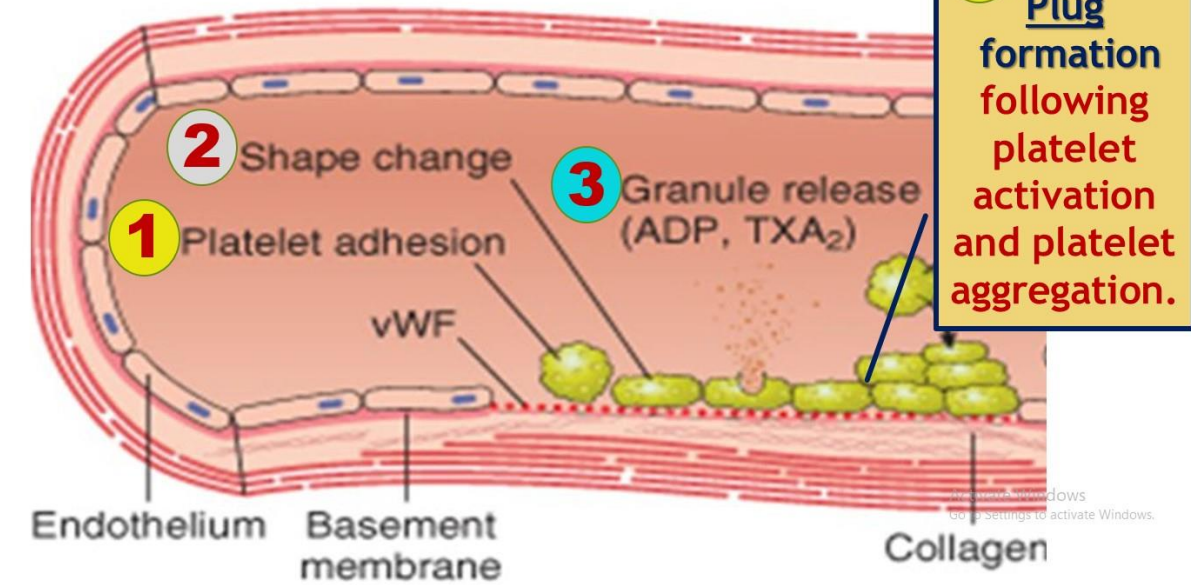




# INFLAMMATORY PHASE

- The aggregation of platelets results in the formation of the **primary platelet plug**.
- Aggregation and attachment to exposed collagen surfaces activates the platelets.
- Activation enables platelets to degranulate and release chemotactic and growth factors, such as platelet-derived growth factor (PDGF), proteases, and vasoactive agents (eg., **serotonin, histamine**).

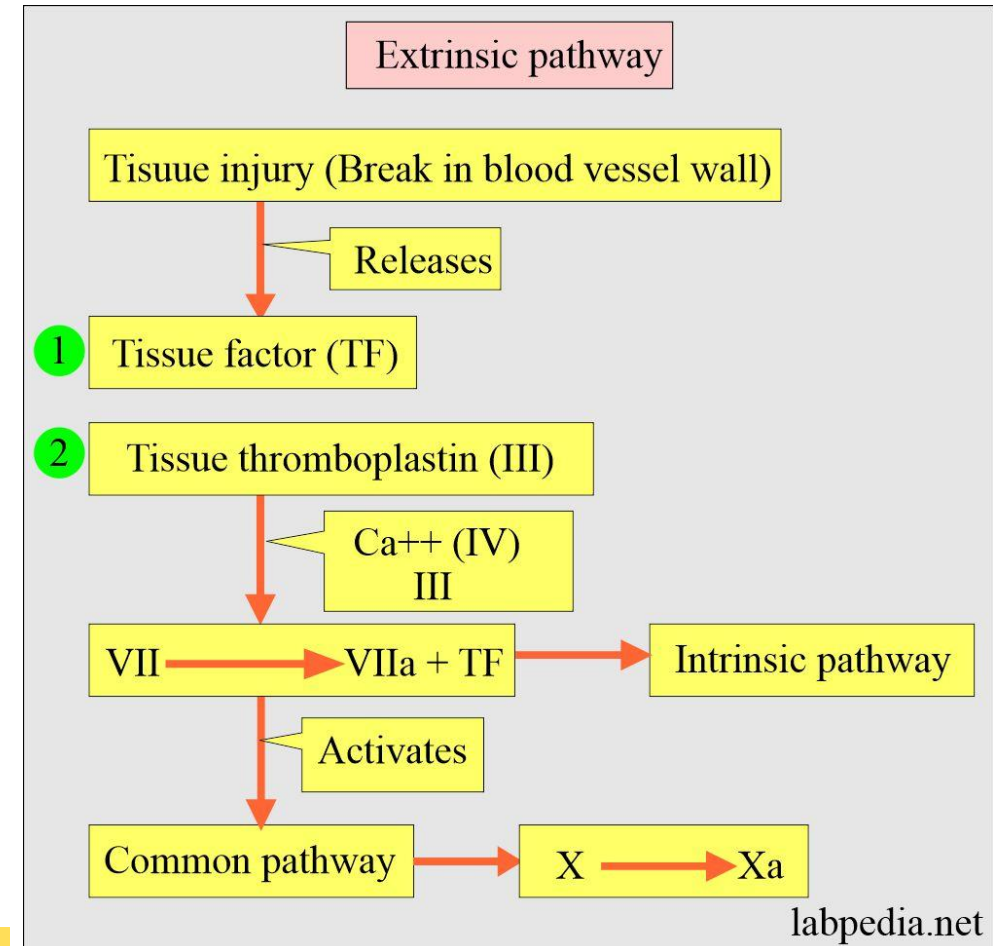
## Primary Hemostasis - Platelets



# INFLAMMATORY PHASE

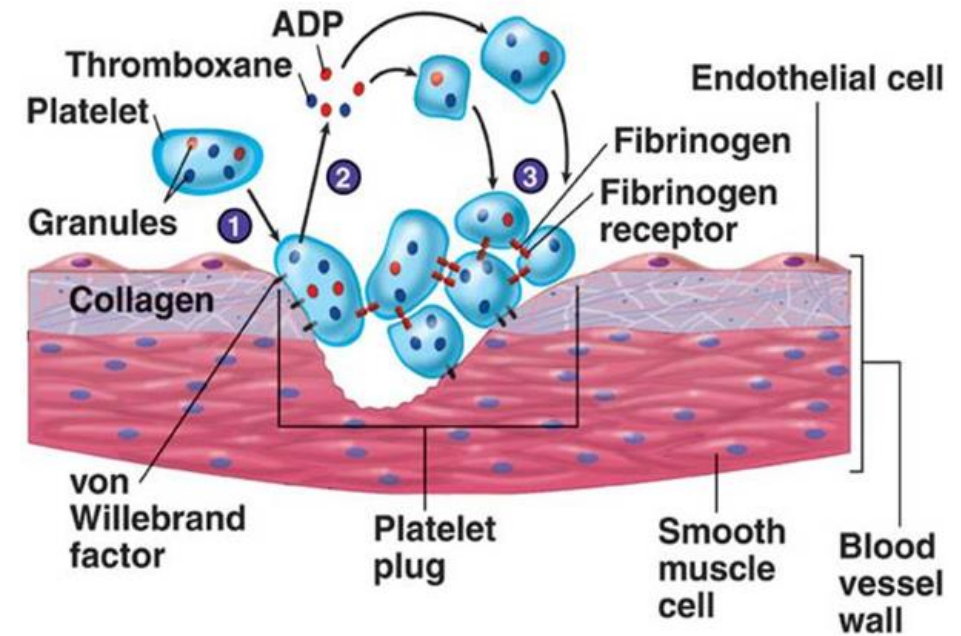
The coagulation cascade occurs by 2 different pathways.

- The intrinsic pathway begins with the activation of factor XII (Hageman factor), when blood is exposed to extravascular surfaces.
- The extrinsic coagulation pathway occurs through the **activation of tissue factor** found in extravascular cells in the presence of factors VII and VIIa



# INFLAMMATORY PHASE

- Both pathways proceed to the activation of thrombin, which converts fibrinogen to fibrin.
- The fibrin product is essential to wound healing and is the primary component of the wound matrix into which inflammatory cells, platelets, and plasma proteins migrate.
- Removal of the fibrin matrix impedes wound healing.
- The result of platelet aggregation and the coagulation cascade is clot formation.





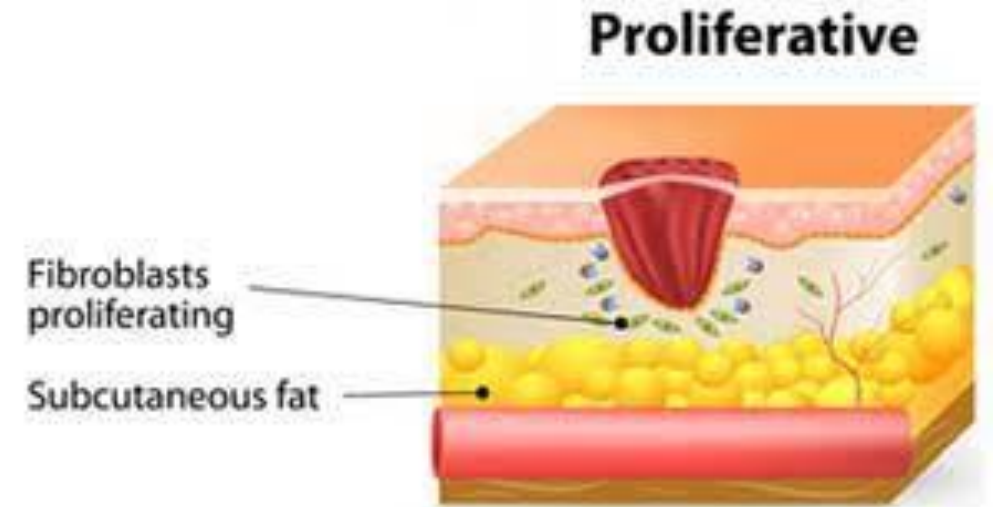
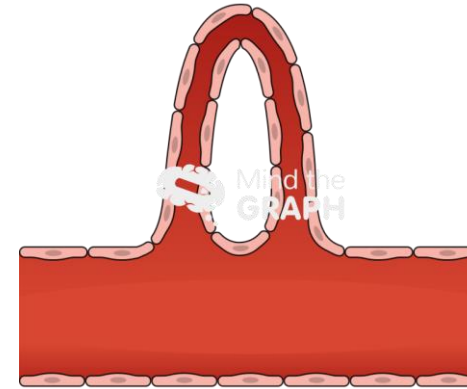
## PROLIFERATION PHASE



- On the surface of the wound, epidermal cells burst into mitotic activity within **24 to 72 hours.**
- These cells begin their **migration across the surface of the wound.**
- **Fibroblasts** proliferate in the deeper parts of the wound.
- These fibroblasts begin to synthesize small amounts of collagen which acts as a scaffold for migration and further fibroblast proliferation.

## PROLIFERATION PHASE

- **Granulation tissue**, which consists of capillary loops supported in this developing collagen matrix, also appears in the deeper layers of the wound.
- Four to five days after the injury occurs, fibroblasts begin producing large amounts of **collagen and proteoglycans**.
- Proteoglycans appear to enhance the formation of collagen fibers

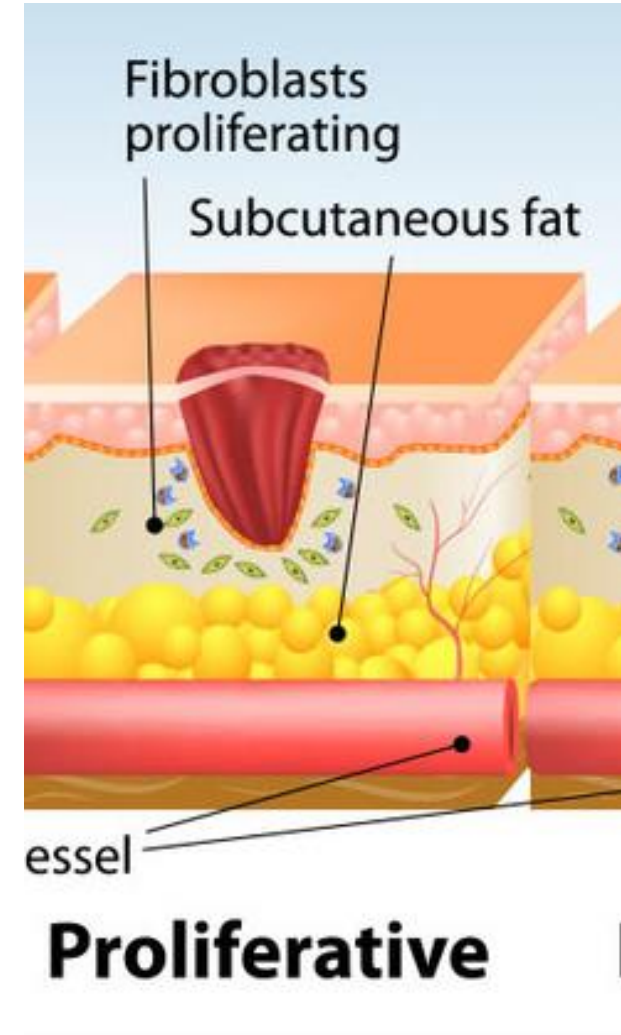




## PROLIFERATION PHASE



- Within two to three weeks, the wound can resist normal stresses, but wound strength continues to build for several months.
- The fibroblastic phase lasts from **15 to 20 days** and then wound healing enters the maturation phase.





## MATURATION PHASE



- This phase lasts from 3 weeks to 2 years
- During the maturation phase, fibroblasts leave the wound and collagen is remodeled into a more **organized matrix**.
- Tensile strength increases for up to one year following the injury.
- While healed wounds never regain the full strength of uninjured skin, they can regain up to 70 to 80% of its original strength.

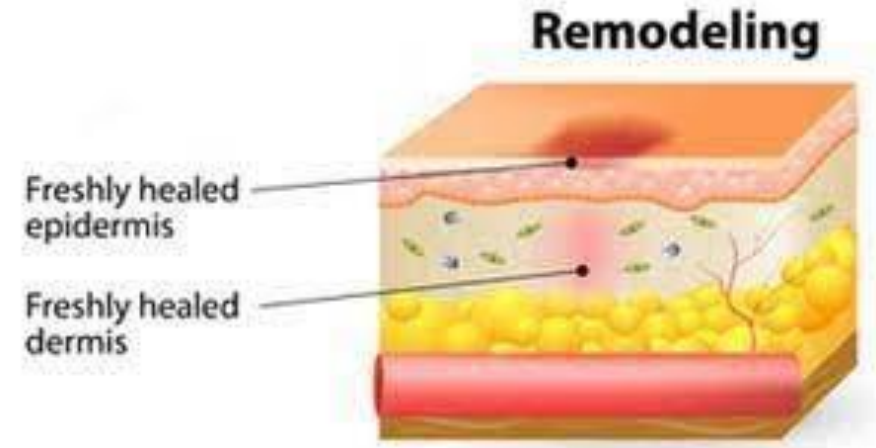




## MATURATION PHASE



- The final product of the healing process is a **scar**.
- This relatively avascular and acellular mass of collagen serves to restore tissue continuity, strength and function.
- **Delays** in the healing process cause the prolonged presence of wounds, while.
- **Abnormalities** of the healing process may lead to abnormal scar formation.



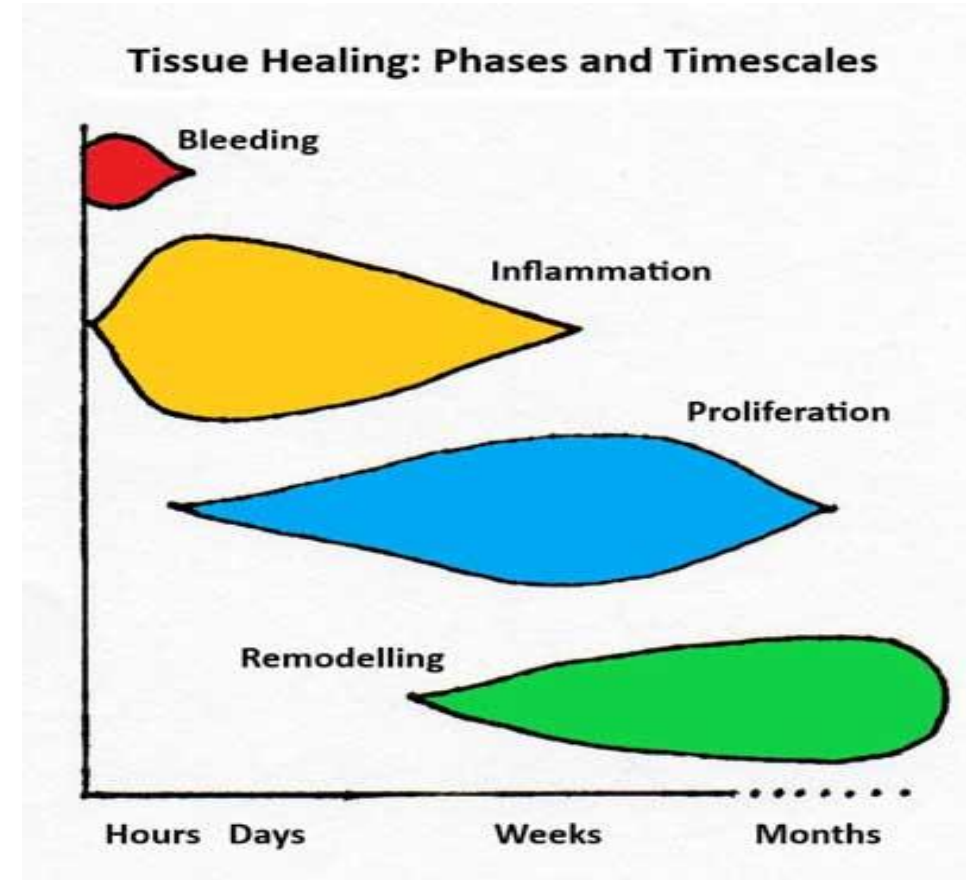




# HEALING RETARDED BY SEVERAL FACTORS & TIME SCALE

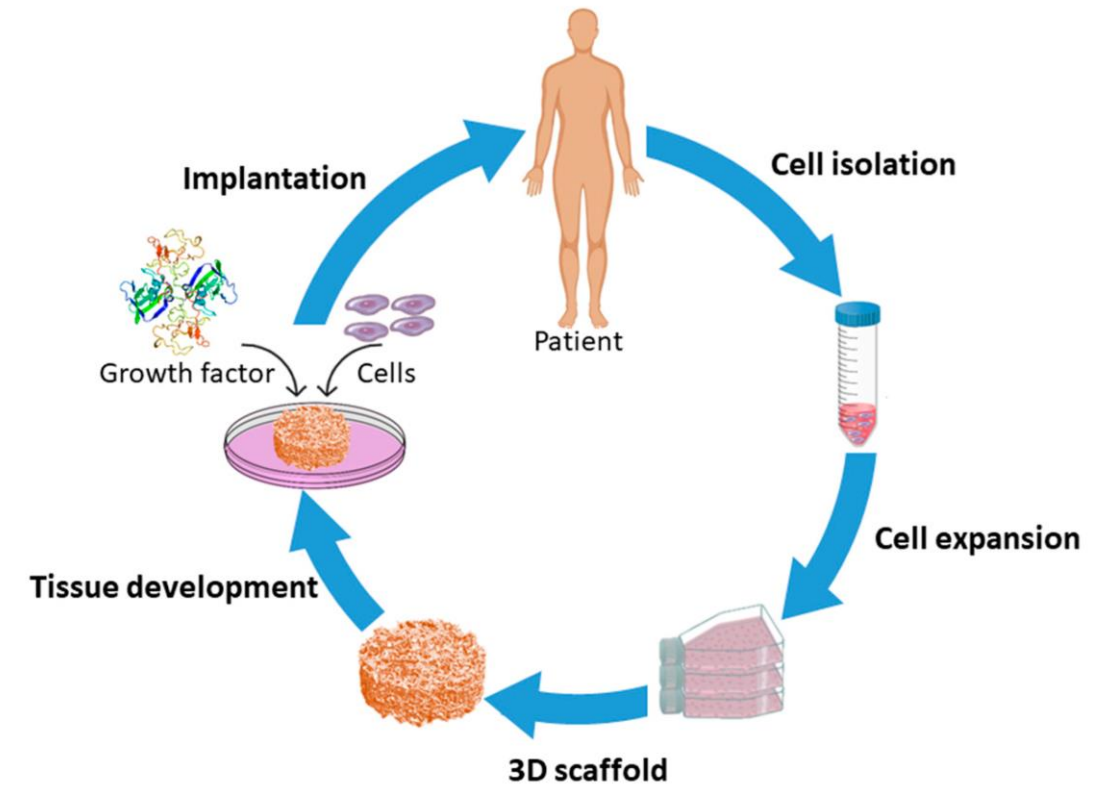
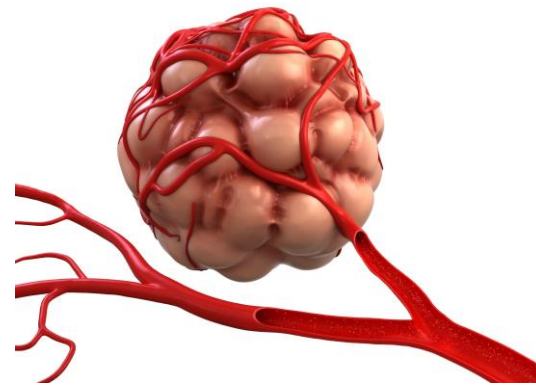
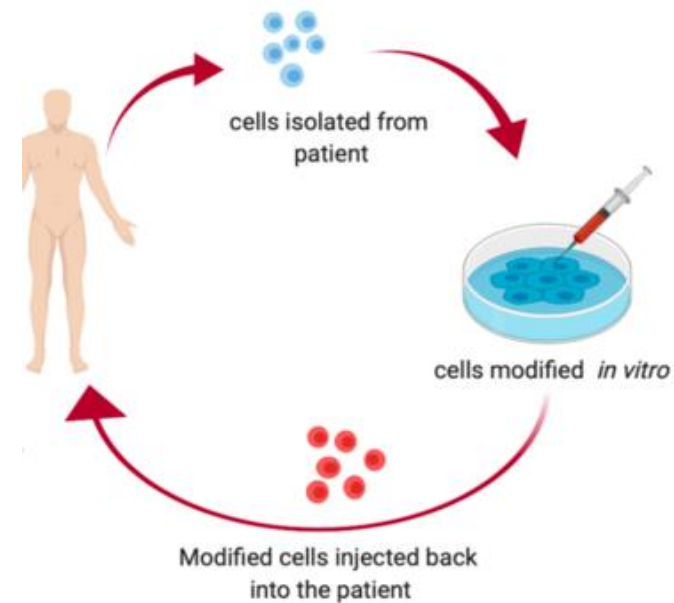


- Ischemia
- Dry environment
- Infection
- Foreign Bodies
- Anti-inflammatory therapy
- Nutritional Deficiency



# FUTURE OF WOUND HEALING

- Epithelization
- Wound contraction and scarring
- Angiogenesis
- Tissue engineering
- Gene Therapy





## ASSESSMENT



- Phases of wound healing
- Maturation phase is also called as -----
- Hemorrhage due to injury is arrested by -----
- Scar formation occurs during -----
- -----proliferate in the deeper parts of the wound.



# THANK YOU



## Reference:

- <https://www.mdpi.com/2075-1729/11/7/665>
- <https://step1.medbullets.com/hematology/111005/thrombogenesis>
- [https://www.researchgate.net/figure/Steps-of-primary-hemostasis-platelet-adhesion-activation-and-aggregation-Image-from\\_fig1\\_334593905](https://www.researchgate.net/figure/Steps-of-primary-hemostasis-platelet-adhesion-activation-and-aggregation-Image-from_fig1_334593905)