WOUND HEALING

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WOUND HEALING

* **It is a series of events that begins at the moment of injury and can continue for months to years,**

* **it is a complex and dynamic process of restoring cellular structures and tissue layers,**

with cells Interaction and cytokines working in concert.

Hippocrate : **Assisst Nature**

Richard L. Lammers

“the primary goal of wound care is not the technical repair of the wound

it is providing optimal conditions for the natural reparative processes of the wound to proceed”

What are the advances in wound healing

1. **Knowing the cell types involved**
2. **Knowing the order in which they appear in the wound**
3. **The growth factors and their functions have been elucidated.**
4. **advances in molecular science have allowed a true appreciation of the complex interplay between the cells involved in the phases of wound healing, the molecular signaling**

WOUND HEALING STAGES

I. Haemostasis
II. INFLAMATORY PHASE
III. PROLIFERATIVE PHASE
IV. MATURATION PHASE

I. Haemostasis :

vasoconstriction, platelet aggregation,clot formation

II. INFLAMATORY PHASE

Period : Immediate to 2-5 days

a. vasodilatation

b. Phagocytosis:

- leucocyte activation
- lymphocyte activation
- macrophage role

III. PROLIFERATIVE PHASE

1. **Formation of granulation tissue which occurs 3-5 days following injury and overlaps with the preceding inflammatory phase**

2. **Period : 2 days to 3 weeks**

The phases of proliferation:

a. **Angiogenesis**

b. **Fibroplasia**：

Fibroblasts are responsible for the production of collagen, elastin, fibronectin, glycosaminoglycans,

C. Epithelization

1. begins within hours of tissue injury.

2. Epidermal cells at the wound edges undergo structural changes, allowing them to detach from their connections

3. Cell may travel about 3 cm from point of origin in all directions

d.Wound contraction

1. Wound edges pull together to reduce the defect

2. it is maximal 5-15 days after injury.

3. The maximal rate of contraction is 0.75 mm/day and depends on the degree of tissue laxity.

4. Wound contraction depends on the myofibroblast located at the periphery of the wound

e.Collagen synthesis

1.Collagen is rich in hydroxylysine and hydroxyproline moieties, which enable it to form strong cross-links.

2.The hydroxylation of proline and lysine residues depends on the presence of oxygen, vitamin C, ferrous iron.

IV . MATURATION PHASE

Remodelling

1. New collagen forms which increases tensile strength to wounds

2. Scar tissue is only 80 percent as strong as original tissue

3.collagen becomes increasingly organized Type III collagen is replaced by type I collagen.

4.Water is resorbed from the scar,

remodeling begins approximately 21 days after injury,

Period : 3 weeks to 2 years


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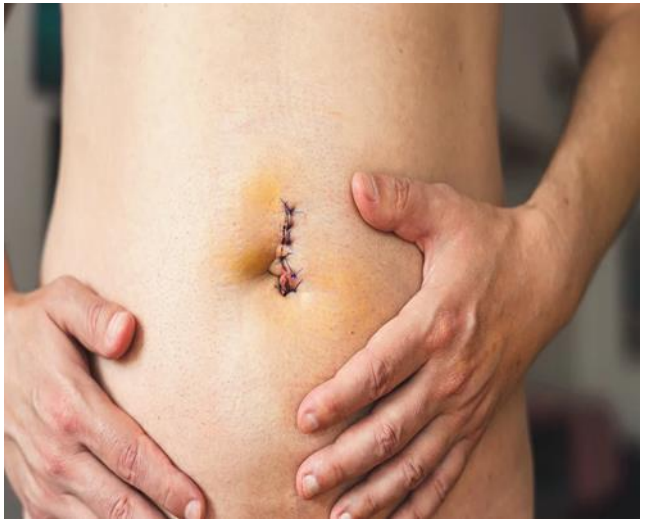
Credit: Lukassek

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TYPES OF WOUND HEALING

1. Primary healing "healing by 1st Intension" :

involves closure of a wound within hours of its creation,acombination of epithelization and c.t. formation

2.Secondary healing "healing by 2nd intension:

involves no formal wound closure, the wound closes spontaneously by: contraction,C.T. formation and re epithelialization

3. Healing by 3rd Intention:

also known as delayed primary closure, involves initial debridement of the wound for an extended period and then formal closure with suturing

CYTOKINES IN WOUND HEALING

a cytokine is a protein mediator, released from various cell sources, which binds to cell surface receptors to stimulate a cell response.

1. **it provide all the comunications for cell to cell interaction.**
2. **Play role in regulating fibrosis.**
3. **They regulate cell proliferation**
4. **Stimulate cell to migrate to the wound site**
5. **Direct cells to produce specific components needed for the repair**

CYTOKINES

cytokin	Cell of origin	function
PDGF **"Platelet derived grouth factor"**	**-platelet** **-macrophage** **-endoth. cell**	**-cell chemotaxis** **-stim. Angiogenesis** **-stim. Wound contraction** **Mitogenic for fibroblast**
TGF **"Transforming grouth factor"**	**- Macrophage** **- T lymphocyte** **-Platelet**	**Cell chemotaxis** **-mitogenic for fibroblast**
EGF **"Epith. Grouth factor"**	**Platelet** **Macrophage.**	**-stim. Angiogenesis** **-Mitogenic for fibroblasdt**

Factors affect wound healing

General factors :

1. Age
2. Anaemia
3. Malnutrition : - Hypoprotinaemia
   - vitamin c
   - ca + Mg
4. Drugs : Steroid,cytotoxic
5. Malignancy
6. Metabolic disorders : Diabetis ,Uraemia

FACTORS AFFECT HEALING

LOCAL FACTORS:

1. **Blood supply**
2. **Hypoxia**
3. **Infection**
4. **Haematoma**
5. **Mechanical stress**
6. **Type of tissue**
7. **Surgical technique**
8. **Suture material**

COMPLICATIONS OF WOUND HEALING

1. **Infection : delays healing by causing T.damage**
2. **Wound Dehiscence**
3. **Pathological Fibrosis**

result from increased collagen production and decreased collagen degradation.

a. **Hypertrophic scar**
b. **Keloid**
c. **Widened scar**

Wound dehiscence

*It is partial or complete seperation of previously approximated wound edges due to failure of proper wound healing

*Occurs 3- 10 days after surgery due to poor b. supply or infection or malnutrition

Keloid :

there is an extreme over grouth of scar tissue beyond the limit of the original wound with no tendency

to resolve

There is a decrease in collagen degradation

The levels of the collagenase inhibitor alpha-2 macroglobulin have been shown to be decreased in keloid lesions

Widened scar :

formation is thought to result from wound edge separation with tension perpendicular to the healing skin wound.

Hypertrophied scar

1. It occures in wounds whose healing was delayed

2.The scar remain in the remodelling phase for longer than usual,

its more cellular &more vascular than mature scar.

3. Clinicly the scar is red raised itchy and tender

Comparision of Hypertrophic & keloid scars

Features	**Hypertrophic scsr**	**Keloid scar**
Genetic	Not familial	May be familial
Race	Not race related	Black>white
Sex	F = M	F > M
Age	children	10-30 y
Borders	Remains within wound	Out grow wound area
Natural history	Subsides with time	Rarely subside
site`	Flexor surfaces`	Sternum,shoulder
Aetiology	Related to tension	unknown

Fetal Wound Healing Studies

1. Wounds occurring in fetuses of early gestational age can heal without any scar formation
2. presence of fewer neutrophils and more monocytes during the inflammatory period,
3. different concentrations of cytokines,
4. a greater proportion of type III collagen in contrast to adult wounds.
5. collagen deposition in fetal wounds displays a fine reticular pattern
6. fibronectin is more abundant in fetal wounds and has been noted to accelerate wound healing in fetal rat models.