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
"The physician treats, but nature heals."

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

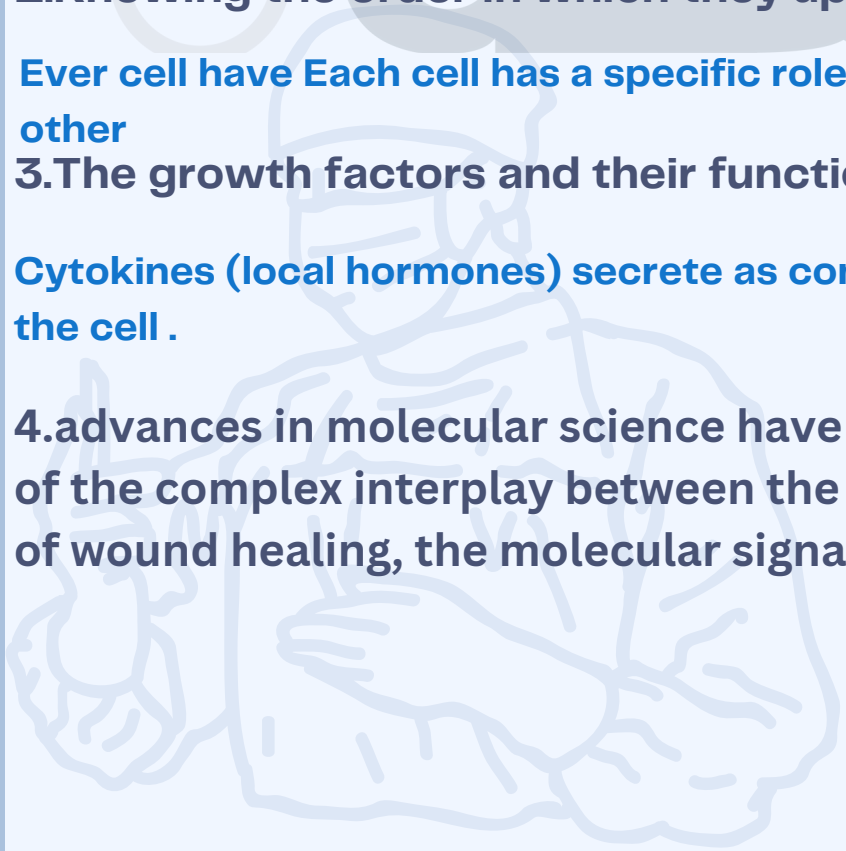
2.Knowing the order in which they appear in the wound

Ever cell have Each cell has a specific role that is different from the other

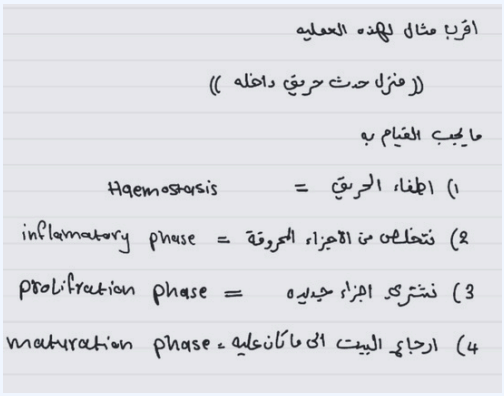
3.The growth factors and their functions have been elucidated.

Cytokines (local hormones) secrete as communication pathways between the cell .

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 :

It Starts immediately after injury & Immediate

vasoconstriction, platelet aggregation, clot.

II. INFLAMATORY PHASE

Period: Immediate to 2-5 days

a. vasodilatation

b. Phagocytosis:

- leucocyte activation

-lymphocyte activation

- macrophage role
to engulf the damaging tissue

III. PROLIFERATIVE PHASE

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

THE PHASES OF PROLIFERATION:

A.Angiogenesis

Formation and new blood vessels

B.Fibroplasia:

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

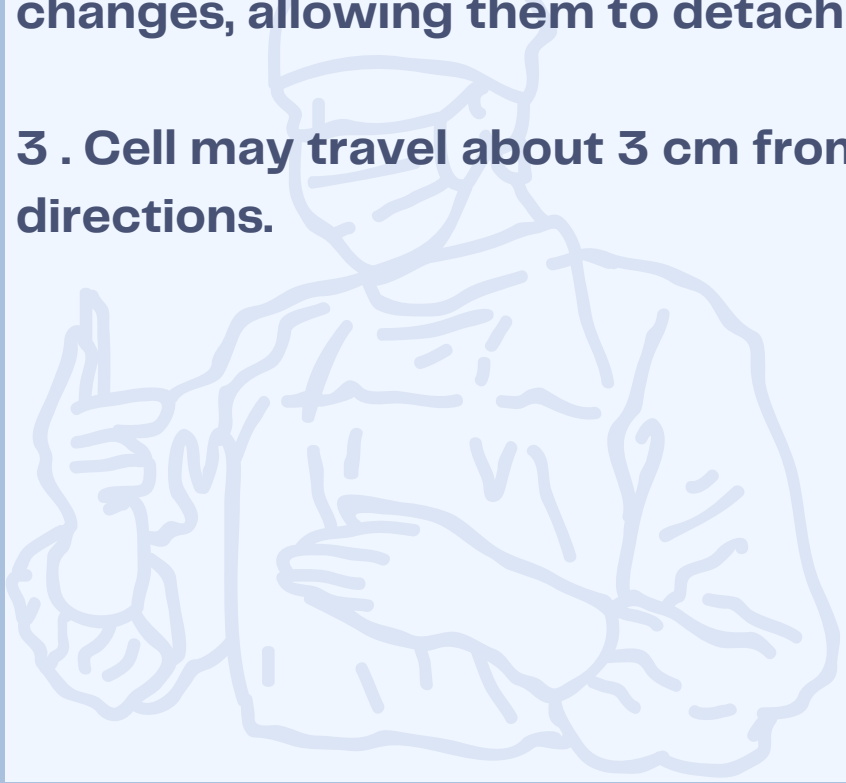
C. Epithelization

Migration of epithelial cells from the edges of the wound and from remaining skin appendages.
Migration of epithelial cells occurs at the rate of 1 mm/day in clean, open wounds.
In surgical wounds, epithelization may be less visible due to factors like wound closure methods(edge to edge), depth of injury, inflammation.

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

The matrix is not sufficient to fill this defect ,We need to contract in order to reduce these imbalances

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

Wound contraction tends to be less noticeable because it occurs gradually and subtly. Factors such as the methods used for wound closure and individual patient differences also influence its visibility. While wound contraction is essential for reducing the size of a wound, its effects may not be immediately apparent compared to other stages of healing that are more prominent.

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.

Hydroxylysine is an oxidized form of lysine, produced through hydroxylation, and plays a vital role in collagen stability and function.

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.

IV. MATURATION PHASE REMODELLING

Remodeling is a critical phase of wound healing characterized by the reorganization and strengthening of collagen fibers, reduction of vascularity, and maturation of the tissue.

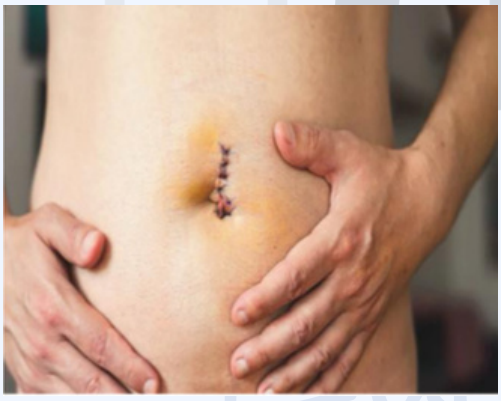
1. New collagen forms which increases tensile strength to wounds

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



- clean surgical incision.
- no significant bacterial contamination.
- The wound heals quickly with complete closure without much Loss of cells and tissue.

- An open wound with a tissue defect, sometimes infected.
- loss of cells and tissues.
- Healing by fibrosis tissue.
- We should clean the wound and give antibiotics.

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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.
the doctor should clean the wound ,then it healed spontaneously by fibrous tissue

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.
Open Wound**: The wound has a tissue defect and infected.
There is significant loss of cells and tissues around the wound.
The wound should be cleaned thoroughly to remove dirt and debris.
Antibiotics**: Antibiotics should be given to prevent or treat infection.
(the doctor should clean the wound then sutures it ""not spontaneously "")

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.

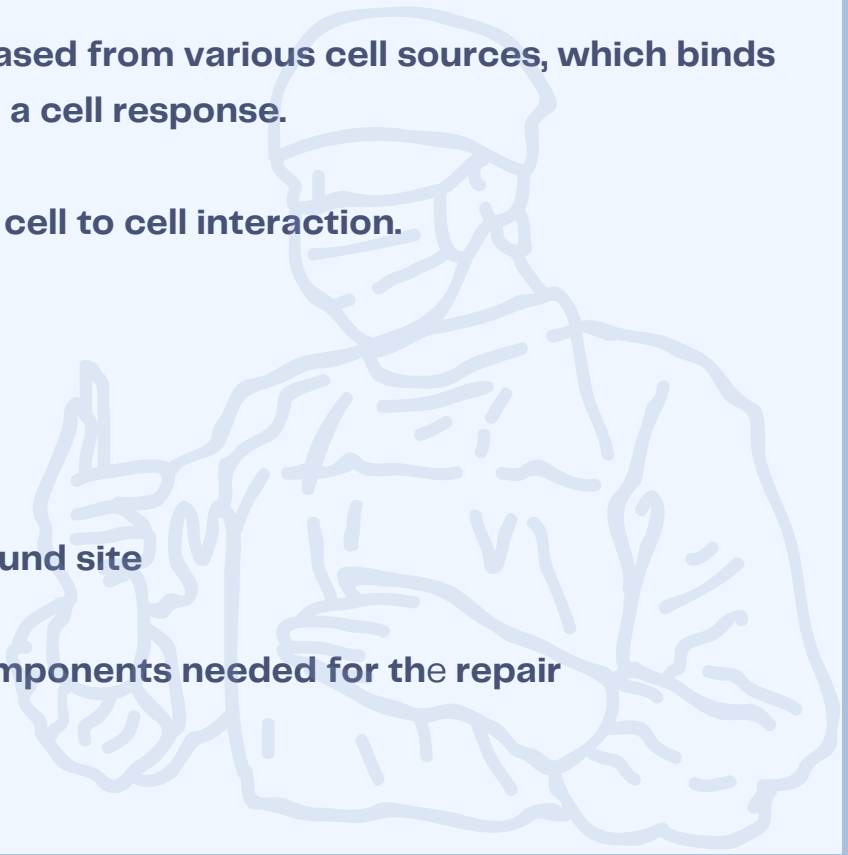
1. it provide all the comunications for cell to cell interaction.

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



CYTOKINE

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

It only affects the time taken, so the older you are, the slower the process becomes .

2. Anaemia

it affect the hydroxlation of lysine .

3. Malnutrition :

- Hypoprotinaemia
- vitamin c
- ca + Mg

4. Drugs:

- Steroid, cytotoxic

5. Malignancy

6. Metabolic disorders: Diabetis, Uraemia text

Diabetes significantly affects wound healing through

1. impaired blood circulation, immune system; alteredinflammatory responses lead increased infection risk
2. have detrimental effects on muscle cells, leading to cellular dysfunction and reduced metabolic activity.

FACTORS AFFECT HEALING

LOCAL FACTORS:

1. Blood supply.

2. Hypoxia.

3. Infection.

4. Haematoma.

5. Mechanical stress.

6.Type of tissue:
The healing process of bones and cartilage is generally slower compared to other tissues.

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

It can result from various factors, including mechanical stress, infection, and patient-related issues.

Keloid:

There is an extreme over grouth of scar tissue beyond the limit of the original wound with no tendency to resolve.

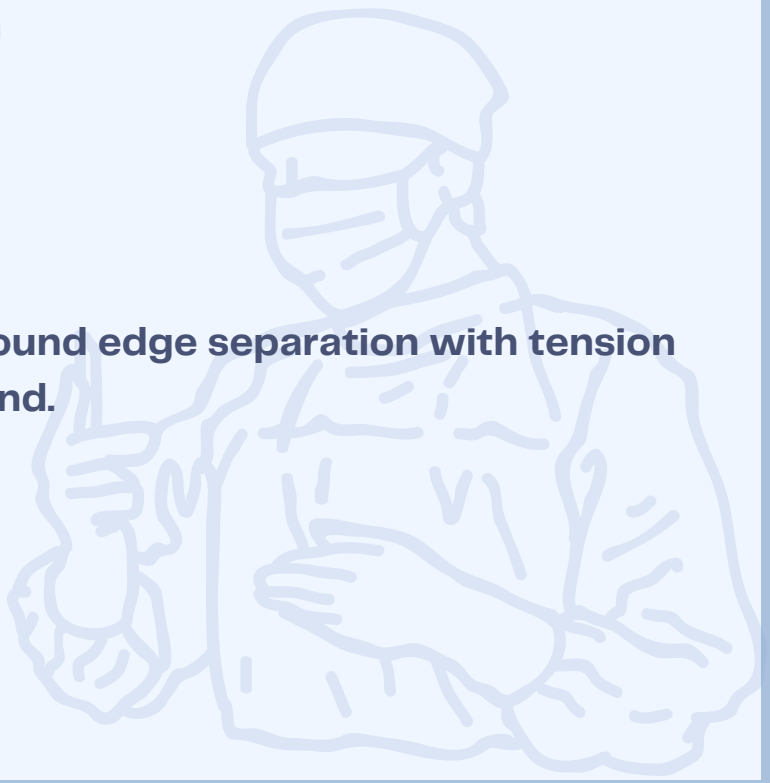
There is a decrease in collagen degradation.

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

Keloids are caused by a malfunction in the enzymes responsible for collagen breakdown, leading to excessive tissue proliferation. While proliferation is a necessary part of wound healing, it can also result in complications such as keloid formation, obstruction, and increased risk of infection

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

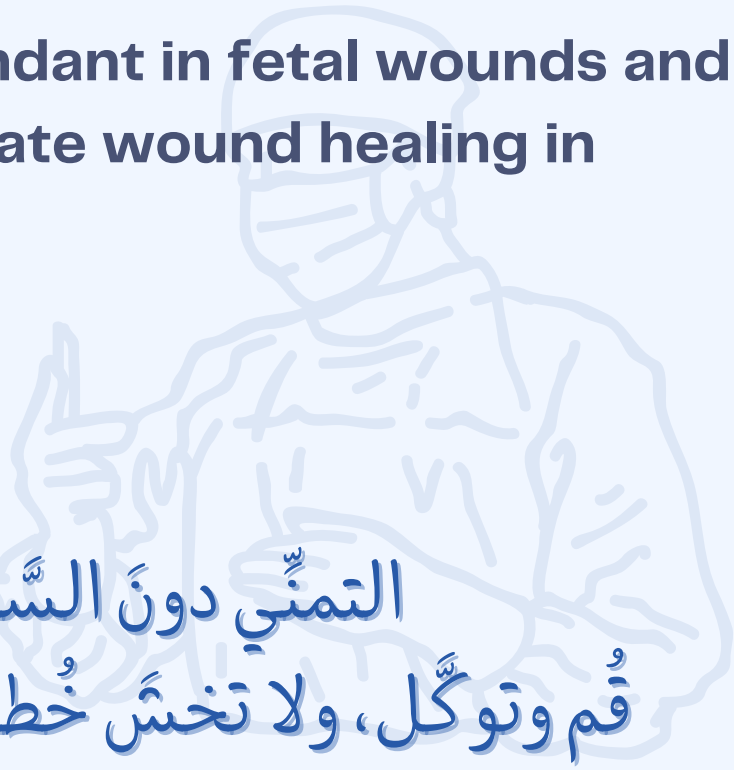
2. Presence of fewer neutrophils and more monocytes during the inflammatory period,

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.



التمنِّي دونَ السَّعي مضيعةً للوقت.
قُم وتوكَّل، ولا تخشَ خُطوةً فقد تلمسُ الغيمَ أو تكاد!