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FACULTY OF NURSING

Chapter-01

WOUND HEALING

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WOUND

- It is a circumscribed injury which is caused by external force and it can involve any tissue and organ.

- A wound is a break in the integrity of the skin or tissues often which may be associated with disruption of the structure and function.

  (SRB 4th edition)

- A cut or break in the continuity of any tissue, caused by injury or operation.

  (Baillière's 23rd Ed)

CLASSIFICATION OF WOUNDS

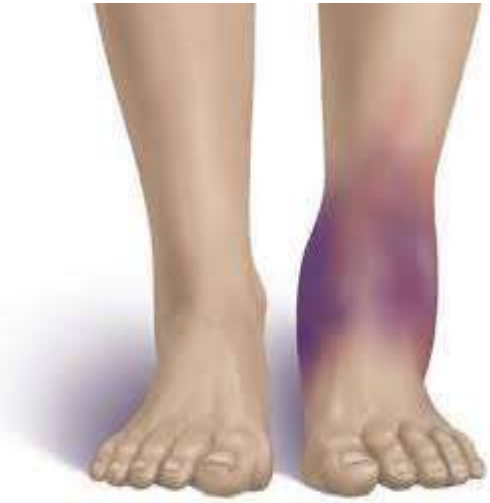
Rank and Wakefield classification

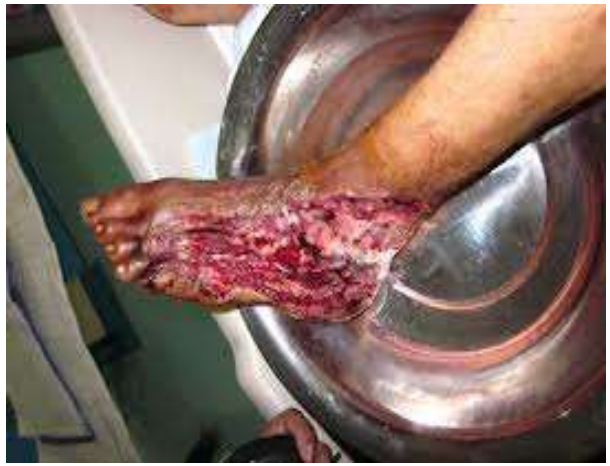
a) Tidy wounds

b) Untidy wounds

Classification based on type of wound

i. Clean incised wound
ii. Lacerated wound
iii. Bruising and contusion
iv. Haematoma
v. Puncture wound
vi. Abrasion
vii. Crush injury
viii. Injuries to bone and joint (maybe open or closed)
ix. Injuries to nerve (either clean cut or crush)
x. Injuries to arteries and veins
xi. Penetrating wounds

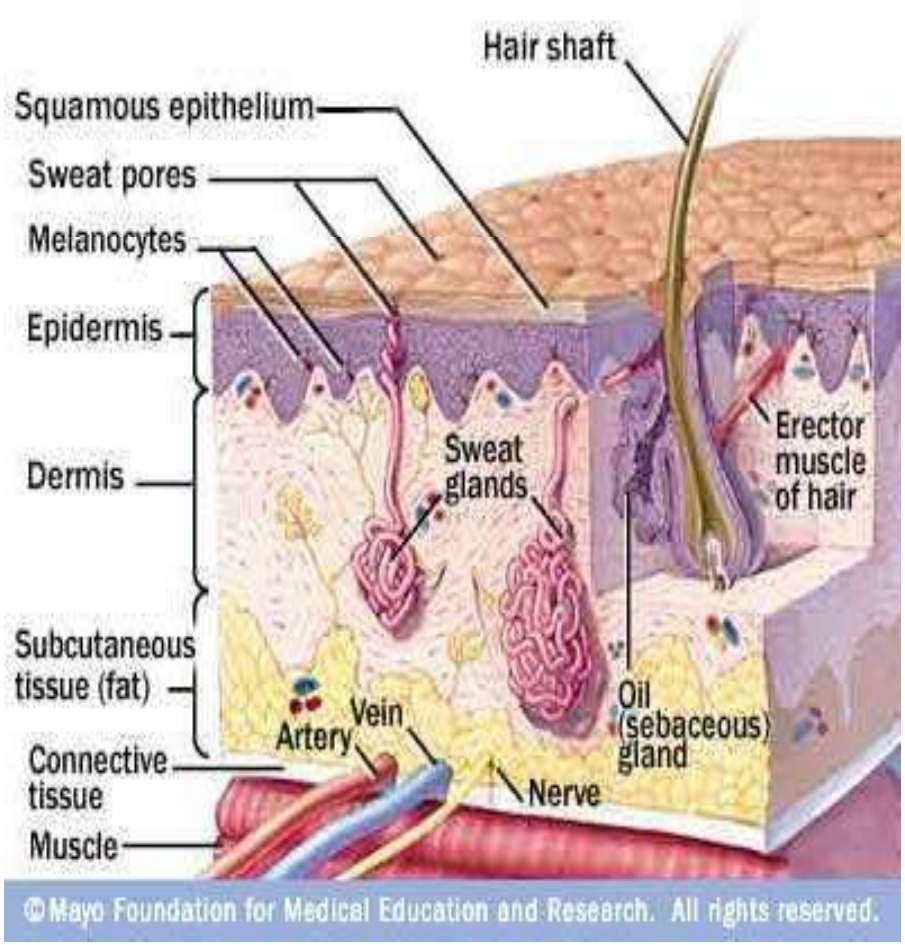




Classification based on thickness of wound

a) Superficial wound
b) Partial thickness
c) Full thickness
d) Deep wounds
e) Complicated wounds
f) Penetrating wound

Superficial
Partial thickness
Full thickness
Deep wound



Classification of surgical wounds

a) Clean wound
b) Clean contaminated wound
c) Contaminated wound
d) Dirty infected wound

HEALING

- Healing is the body's response to injury in an attempt to restore normal structure and function.

The process of healing involves 2 distinct processes:

A. REGENERATION

B. REPAIR

- **Regeneration**: Is when healing takes place by proliferation of parenchymal cells and usually results in complete restoration of the original tissues.

- The goal of all surgical procedures should be regeneration which returns the tissues to their normal microstructure and function.

- **Repair**: It is a healing outcome in which tissues do not return to their normal architecture and function.

- Repair typically results in the formation of scar tissue.

TYPES OF WOUND HEALING

- Healing by first intention (wounds with opposed edges)

- Healing by secondary intention (wounds with separated edges)

Healing by first intention (wounds with opposed edges)

Healing of wound with following characteristics:

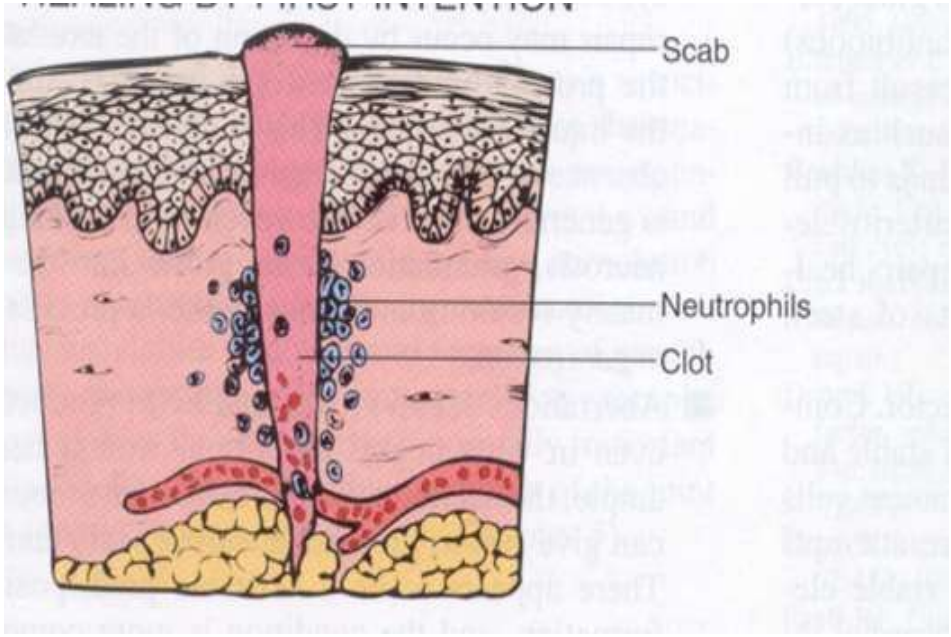
- Clean and uninfected
- Surgically incised
- Without much loss of cells and tissue
- Edges of wound are approximated by surgical sutures.
- Wounds with opposed edges
- Primary union

- The incision causes
  - death of a limited number of epithelial cells and connective tissue cells
  - disruption of epithelial basal membrane continuity

- The narrow incisional space immediately fills with clotted blood containing fibrin and blood cells; dehydration of the surface clot forms the well known scab that covers the wound.

Within 24 hours

- Neutrophils appear at margins of incision, moving toward fibrin clot
- Epidermis at its cut edges thickens as a result of mitotic activity of basal cells



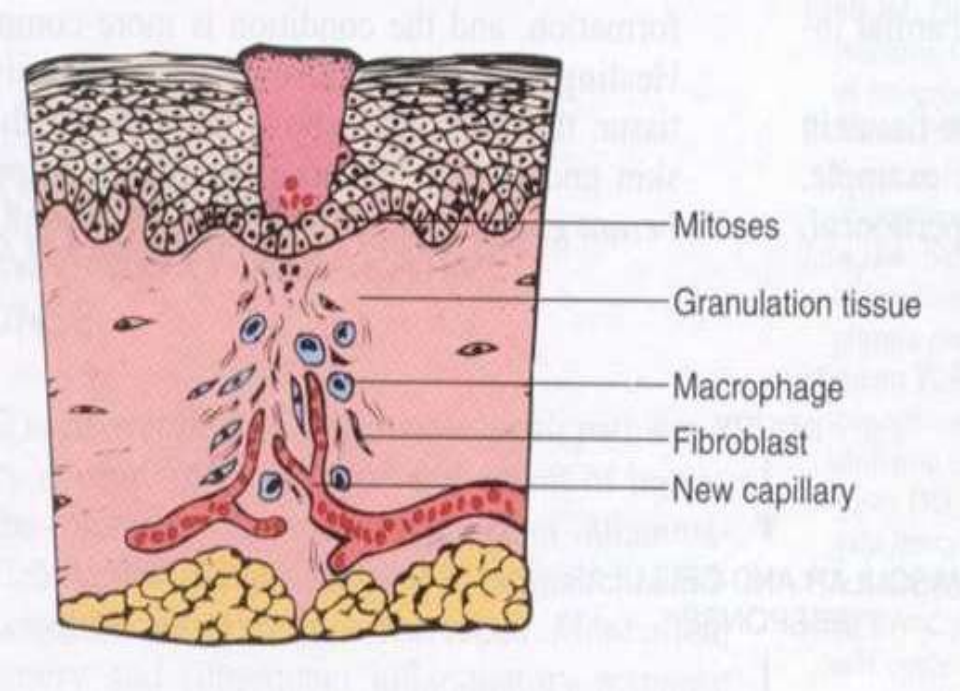
•Within 24 to 48 hours, spurs of epithelial cells from the both edges migrate and grow along the cut margins of the dermis, depositing BM components as they move. They fuse in the midline beneath the surface scab, thus producing a continuous but thin epithelial layer.

By day 3,

- Neutrophils replaced by macrophages
- Granulation tissue progressively invades incision space

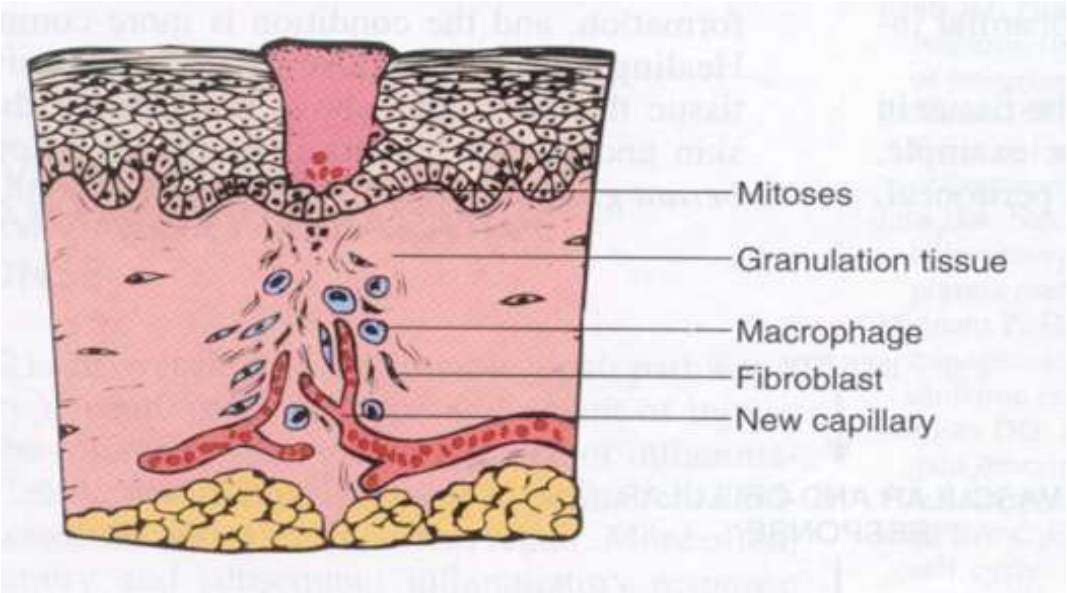
•Collagen fibers are now present in the margins of the incision, but at first these are vertically oriented.

•Epithelial cell proliferation continues, thickening epidermal covering layer



By day 5,

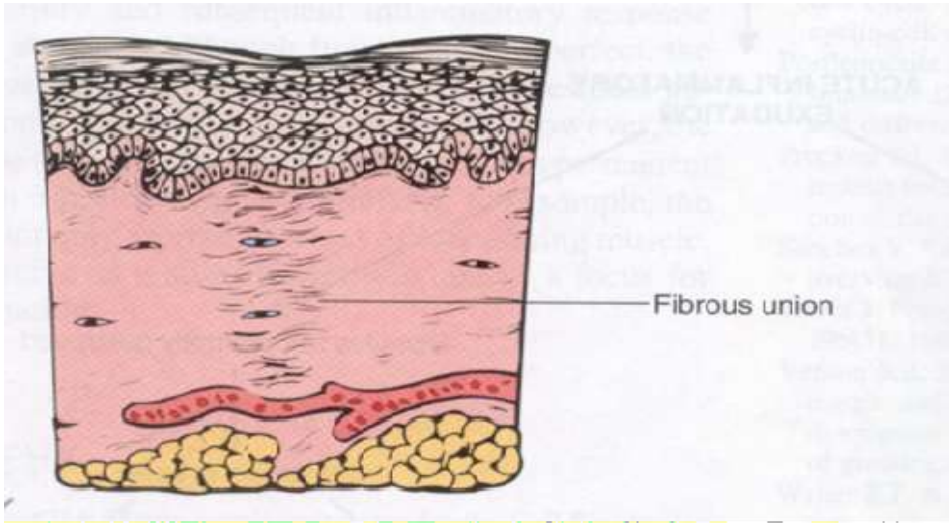
- Incisional space is filled with granulation tissue
- Neovascularisation is maximal
- Collagen fibrils become more abundant and begin to bridge incision
- The epidermis recovers its normal thickness, and differentiation of surface cells yields a mature epidermal architecture with surface keratinization



week

- Continued accumulation of collagen and proliferation of fibroblasts

- Leukocytic infiltrate, edema, and increased vascularity have largely disappeared.

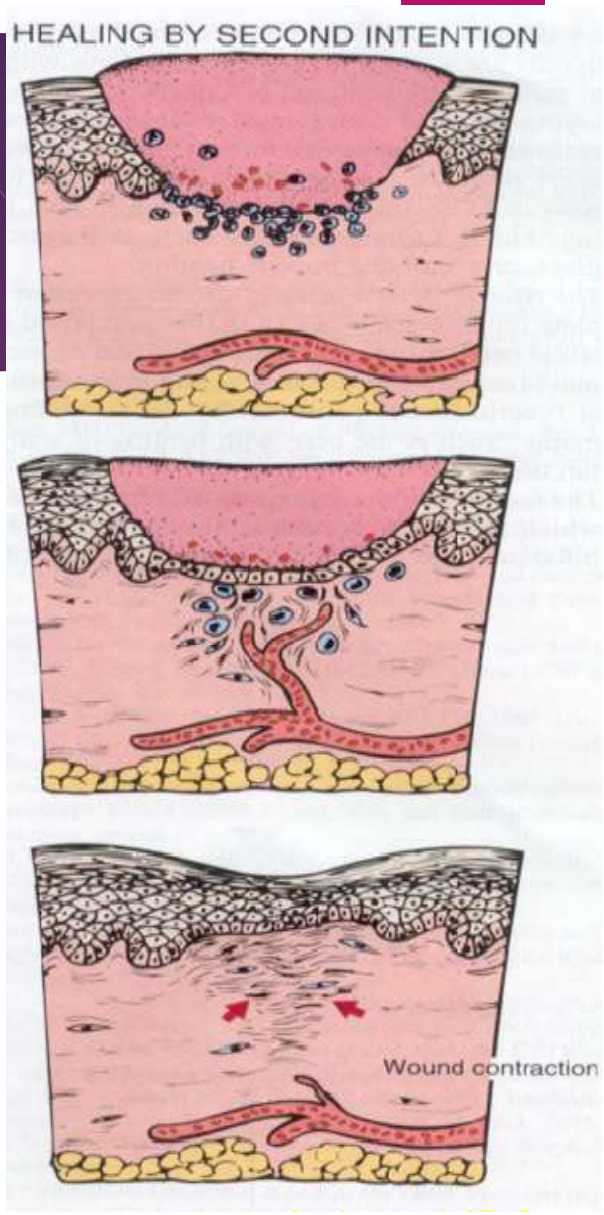


By the end of the first month,

- Scar comprises a cellular connective tissue devoid of inflammatory infiltrate, covered now by intact epidermis.
- Dermal appendages that have been destroyed in the line of the incision are permanently lost.
- Tensile strength of the wound increases thereafter, but it may take months for the wounded area to obtain its maximal strength.

Healing by second intention

- Wounds with separated edges
- Secondary union
- When there is more extensive loss of cells and tissue
- Regeneration of parenchymal cells cannot completely reconstitute the original architecture.
- Abundant granulation tissue grows in from the margin to complete the repair.



Secondary healing differs from primary healing in several respects:

- ✓ Inflammatory reaction is more intense
- ✓ Much larger amounts of granulation tissue are formed
- ✓ Wound contraction occurs in large surface wounds
- ✓ Substantial scar formation and thinning of the epidermis occurs

Difference between 1° & 2° union of wound

FEATURES	PRIMARY	SECONDARY
CLEANLINESS	CLEAN	NOT CLEAN
INFECTION	NOT INFECTED	INFECTED
MARGINS	SURGICALLY CLEAN	IRREGULAR
SUTURES	USED	NOT USED
HEALING	SMALL GRANULATION TISSUE	LARGE GRANULATION TISSUE
OUT COME	LINEAR SCAR	IRREGULAR WOUND
COMPLICATION	NOT FREQUENT	FREQUENT

STAGES OF WOUND HEALING

1. Stage of inflammation.
2. Stage of granulation tissue formation and organisation.
3. Stage of epithelialisation.
4. Stage of scar formation and resorption.
5. Stage of maturation.

PHASES OF WOUND HEALING

- For soft tissue wound healing:

1. Inflammatory phase: It can be broken down into further
   a) Clot formation
   b) Early inflammation
   c) Late inflammation

2. Prolife

rative

Inflammatory phase

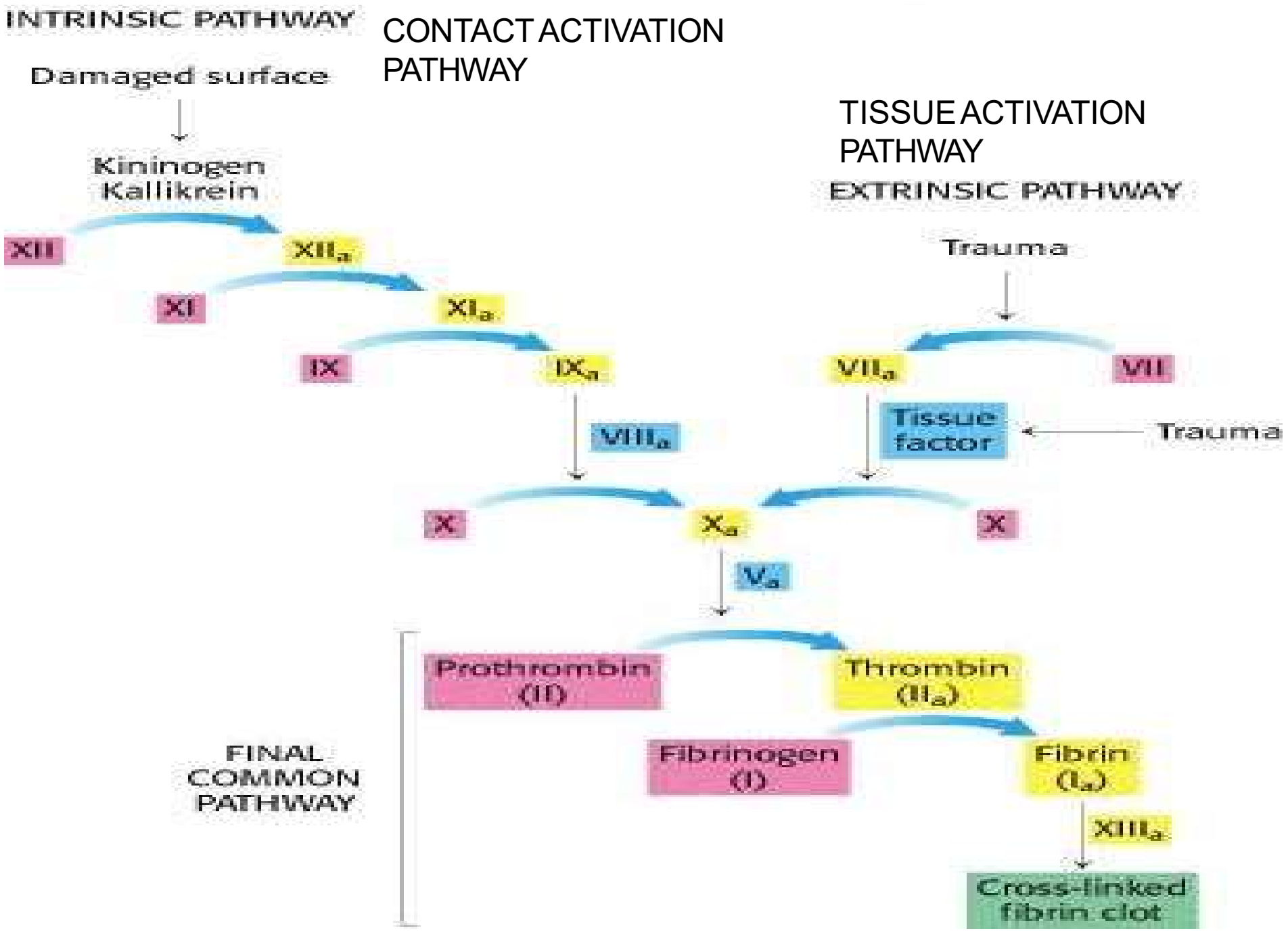
a) Clot formation:

- Begins with three events:

i. Blood vessel contraction initiated by platelet degranulation of serotonin, which acts on endothelial cell and increases the permeability of the vessel, allowing a protein rich exudate to enter the wound site

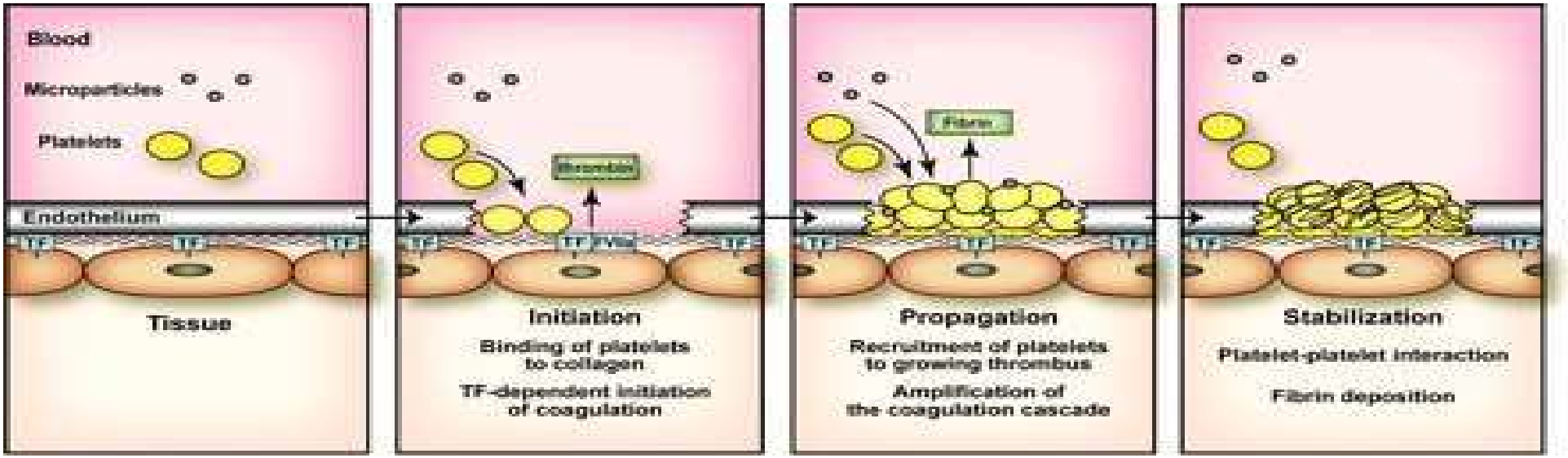
ii. A platelet plug formation

iii. Activation of extrinsic and intrinsic clotting mechanism


INTRINSIC PATHWAY
CONTACT ACTIVATION PATHWAY
Damaged surface
Kininogen
Kallikrein
XII
XIIa
XI
XIa
IX
IXa
VIIIa
X
Xa
Va
Prothrombin (II)
Thrombin (IIa)
Fibrinogen (I)
Fibrin (Ia)
XIIIa
Cross-linked fibrin clot
FINAL COMMON PATHWAY
TISSUE ACTIVATION PATHWAY
EXTRINSIC PATHWAY
Trauma
VIIa
VII
Tissue factor
Trauma
X

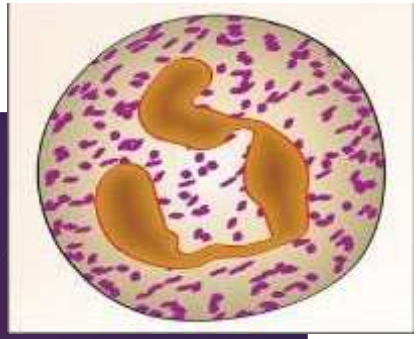
- These events stabilize hemostasis, begins production of chemoattractants and initiate the process of wound decontamination.

- This result in formation of a coagulum.



- Compression of surgical flap with sterile iced gauze immediately after surgery is designed to minimize the thickness of fibrin clot and thereby accelerate optimal wound healing.

b) Early inflammation:



- Characterized by production of polymorphonuclear neutrophils (PMNs)
- Begin to enter the wound site within 6 hours of clot stabilization.
- The number of PMNs increases steadily, peaking at about 24 to 48 hours after the injury.

- Three key steps mark PMN migration into the wound site: a) Pavementing

  b) Emigration

  c) Migration

- Main role of PMNs is wound decontamination by phagocytosis of bacteria.
- The number of PMNs *drop rapidly after the third day*.

c) Late inflammation:

- Presence of macrophages.

- Reaches peak concentration by approximately third or fourth day.

- They have longer life than PMNs and remain in wound till healing is completed.

- Are more bioactive then PMNs – secrete a vast array of cytokines – leads to initiation of proliferative phase of wound healing.
- Major functions of macrophages:
  - ✓ wound decontamination through phagocytosis and digestion of microorganisms and tissue debris.
  - ✓ ingestion and processing of antigens for presentation to T lymphocytes
  - ✓ regulation of wound healing

Proliferative Phase

- Characterized by formation of granulation tissue in the wound.
- 2 key cell types are present in this phase:

a) fibroblasts

b) endothelial cells

- *Granulation tissue:*

  It is a fragile structure composed of an extracellular matrix of fibrin, fibronectin, glycosaminoglycans, proliferating endothelial cells, new capillaries, and fibroblasts mixed with inflammatory macrophages and lymphocytes.

- Epithelial cells are active during this phage and are responsible for initial wound closure.

- GTR procedures are based on control of the epithelial cell growth rate during this phase.

a) Fibroblasts: Fibroplasia

- They migrate into the wound site on the 3rd day after injury and achieve their peak numbers by approximately 7th day.

- This action is stimulated by combination of cytokines produced initially by platelets and subsequently by macrophages and lymphocytes.

- As the number of macrophages declines and fibroblasts population increases the wound transforms from *granulomatous tissue* to *granulation tissue*.

- *Fibroblasts produces collagen (*first detected in the wound about the third day of injury) .

- Fibroblasts produce type III collagen initially and as the wound matures type I collagen is formed.

- As wound healing progresses, the collagen fibers become organized by cross-linking.
- A focused type of fibroblast known as a *myofibroblast plays* a significant role in wound contraction, particularly in incisional-type wounds.
- Myofibroblasts align themselves parallel with the wound surface and then contract, drawing the wound edges together.
- These cells are eliminated by apoptosis after wound closure.

b)Endothelial cells: Angiogenesis

–Formation of new blood vessels at the site of injury takes place by proliferation of endothelial cells from the margins of severed blood vessels.

–The newly formed blood vessels are more leaky accounting for the more edematous appearance of new granulation tissue.

Epithelium:

- The first step is formation of an epithelial seal on the surface of the fibrin clot. It begins at the edge of the wound, where the basal and suprabasal prickle cells rapidly undergo mitosis.
- Migration stops as a result of contact inhibition of the epithelial cells from the opposing wound edge.
- In wounds healing by primary intention, formation of an epithelial seal typically takes 21 to 28 hours after reapproximation of the wound margins.

Maturation Phase

- Begins 5 to 7 days after injury.

- There is conversion of granulation tissue to fibrous connective tissue and decrease parallelism of collagen to the plane of the wound.

- Maturation of the epithelial layer quickly follows formation of the epithelial seal.

- Scar strength is:
  - 3% in 1 week
  - 20% in 3 weeks
  - 80% in 12 weeks

Growth Factors and Cytokines Affecting Various Steps in Wound Healing	
Monocyte chemotaxis	PDGF, FGF, TGF-ß
Fibroblast migration	PDGF, EGF, FGF, TGF-ß, TNF, IL-1
Fibroblast proliferation	PDGF, EGF, FGF, TNF
Angiogenesis	VEGF, Ang, FGF
Collagen synthesis	TGF-ß, PDGF
Collagenase secretion	PDGF, EGF, FGF, TNF, TGF-ß inhibits

PDGF- platelet-derived growth factor
FGF- fibroblast growth factor
TGF- transforming growth factor
EGF- epidermal growth factor
IL- interleukin
TNF- tumor necrosis factor
VEGF- vascular endothelial growth factor

HARD TISSUE HEALING

- The inflammatory and proliferative phases are similar to those for soft tissue.

- The maturation phase differs markedly from that for soft tissues because of the tissue involved.

Osteoblasts: Osteogenesis

- A major difference lies in the role of the osteoclast.
- Osteoclasts acts as an organizational unit to debride necrotic bone from the wound margin similarly as macrophages remove tissue debris from the clot.
- New bone formation is apparent about 6 days after surgery.

- Bone formation is categorized into two types:
  a) Matrix vesicle – based process
  b) Osteoid secretion

a) Matrix vesicle – based process

- Woven bone formation occurs by this process.
- In this process osteoblasts produce matrix vesicles through exocytosis of their plasma membrane.
- It begins with the deposition and growth of hydroxyapatite crystals in the pore regions.
- The crystals amalgamate to form spherulites whose union results in mineralization.

b)Osteoid secretion

- Lamellar bone formation occurs by this process.

- Osteoblasts secrete an organic matrix composed of longitudinally arranged collagen matrix fibrils.

- Mineralization occurs by mineral deposition directly along the collagen fibrils.

- In this stage alkaline phosphatase plays an important role in mineralization.
- Several growth factors identified as a key components in the production of osseous tissue are:
  a. TGF- ß
  b. BMP
  c. PDGF
  d. FGF
  e. IGF

- The osseous defect is filled with bone tissue by 16 weeks after surgery.

Cementoblasts: Cementogenesis:

- Begins 10 to 12 days after root end resection.
- The exact sequence leading to the formation of new cementum remain unidentified.
- Cementum covers the resected root end in approximately 28 days.

FACTORS AFFECTING WOUND HEALING:

1) **Local factors**:
   i. Infection
   ii. Presence of necrotic tissue and foreign body
   iii. Poor blood supply
   iv. Venous or lymph stasis
   v. Tissue tension
   vi. Hematoma
   vii. Large defect or poor apposition

viii. Recurrent trauma

ix. X-ray irradiated area

x. Site of wound, eg.wound over the joints and back has poor healing

xi. Underlying diseases like osteomyelitis and malignancy

factors:

i. Age, obesity,smoking
ii. Malnutrition, zinc, copper
iii. Vitamin deficiency (vit C, vit A)
iv. Anemia
v. Malignancy
vi. Jaundice
vii. Diabetes
viii. HIV and immunosupressive diseases
ix. Steroids and cytotoxic drugs

COMPLICATION:

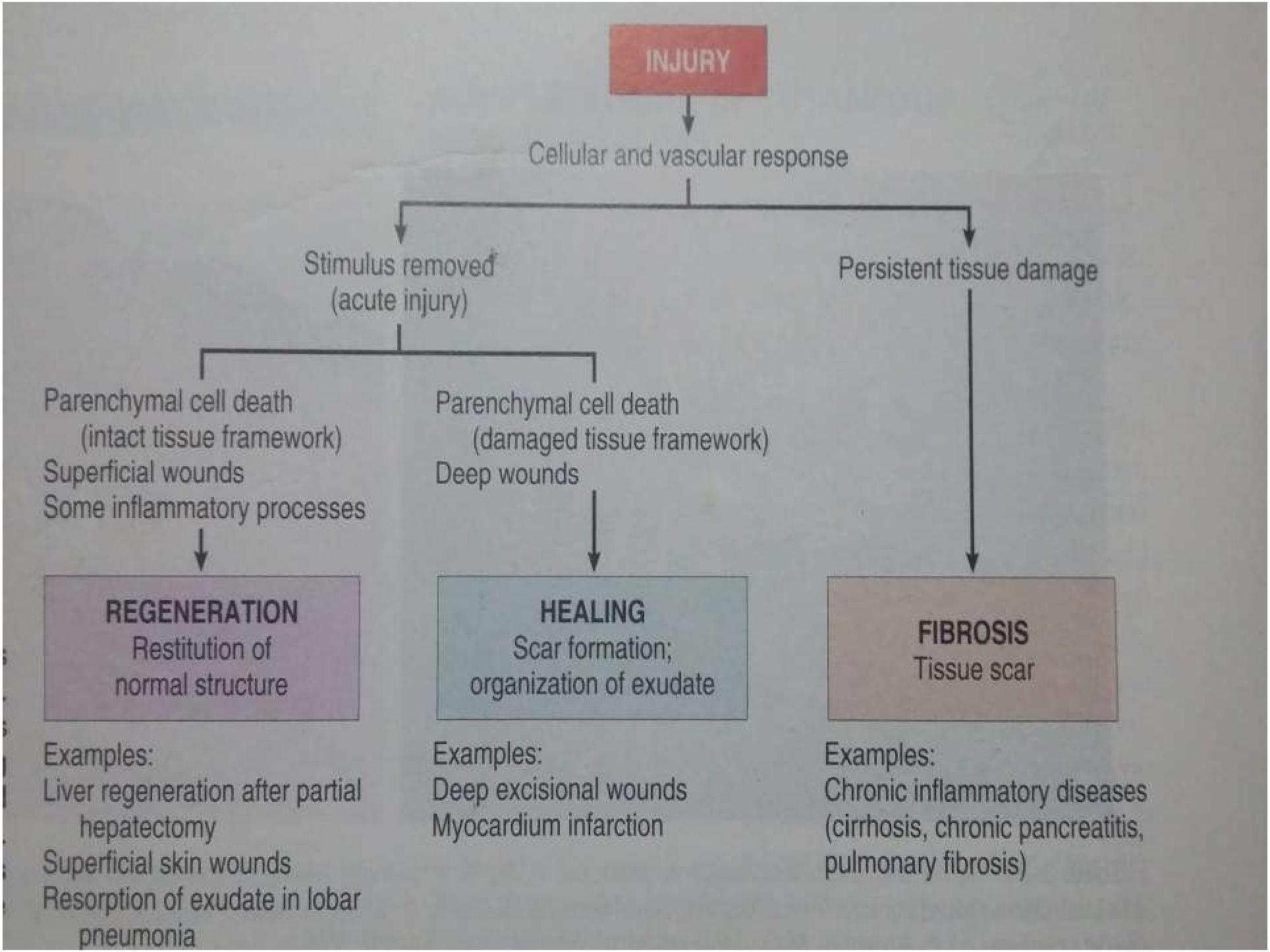
1. Deficient scar formation:
   a) Wound dehiscence
   b) Ulceration
2. Excessive formation of the repair components:
   a) Aberrations of growth: -hypertrophic scar
      -keloid
   b) Excessive amount of granulation tissue formation

c) Exuberant proliferation of fibroblasts and other connective tissue elements: Desmoids or Aggressive fibromatoses

3. Formation of contractures

SYSTEMIC MEDICATION AFFECTING WOUND HEALING:

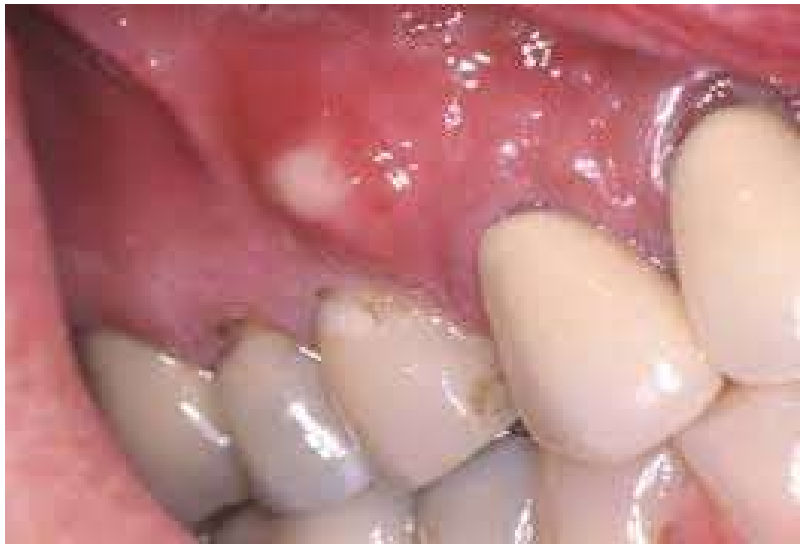
I. Bisphosphonates
II. Glucocorticoids
III. NSAIDS
IV. Cyclooxygenase -2 inhibitors


INJURY
Cellular and vascular response
Stimulus removed (acute injury)
Persistent tissue damage
Parenchymal cell death (intact tissue framework)
Superficial wounds
Some inflammatory processes
Parenchymal cell death (damaged tissue framework)
Deep wounds
REGENERATION
Restitution of normal structure
HEALING
Scar formation; organization of exudate
FIBROSIS
Tissue scar
Examples:
Liver regeneration after partial hepatectomy
Superficial skin wounds
Resorption of exudate in lobar pneumonia
Examples:
Deep excisional wounds
Myocardium infarction
Examples:
Chronic inflammatory diseases (cirrhosis, chronic pancreatitis, pulmonary fibrosis)

HEALING OF SINUS TRACT:

- Sinus is a tract leading from an enclosed area of inflammation to an epithelial surface, and is one of the sequelae of inflammatory disease.

- A sinus tract is a drainage duct for the suppuration produced by abscesses.



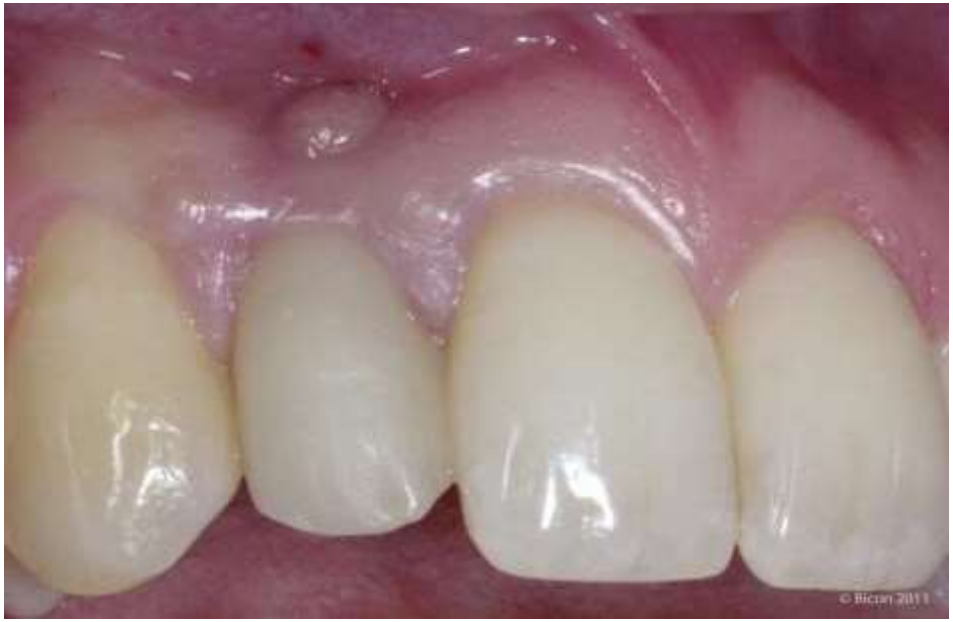
- Suppuration from the periapical inflammatory process may be resorbed by the host organism, otherwise it will flow through the less resistant tissue area.

- It drains onto the epithelial tissue through either a mucosal, or occasionally, a cutaneous sinus tract.

- Sinus tract adjacent to teeth or near the apex of the tooth is usually considered to be of endodontic origin and root canal therapy is the primary treatment to achieve its healing.

- The presence of a sinus tract in the oral cavity is usually considered of pulpal origin, but it can also be caused by periodontal disease.

Draining sinus	Healed sinus
Sinus is active with pus drainage	Sinus has healed with pus discharge absent
Surrounding mucosa reddish pink in color	Surrounding mucosa presents normal tissue coloration
Gutta- percha point can be inserted	Gutta- percha cannot be inserted



Treatment :

- Treatment is directed towards elimination of the source of infection.
- The offending tooth is removed if it is too badly decayed, or if there is extensive loss of the surrounding alveolar bone.
- In most cases, the sinus tract heals spontaneously if the infected pulp is removed, and the root canal debrided and filled.

- Some chronic sinus tracts heal leaving behind a small residual scar, excision may not be required unless its appearance is of concern to the patient.

- If there is fibrosis of the sinus tract trajectory then surgical removal is necessary.

WOUND HEALING AFTER PULPECTOMY

- The healing pattern following pulpectomy is characterized by an initial inflammatory reaction in the apical tissue due to the trauma induced by the cutting procedure.
- In the absence of wound infection, reorganization soon occurs. This involves replacement of the injured tissue by connective tissue derived from the perigingival region

- Materials used to fill root canals may compromise the normal healing pattern, owing to their irritating capacity, and result in a longstanding inflammatory lesion.

- Inflammatory cells accumulate close to the root filling material and remain for as long as toxic components are released. Eventually the material will be lined off by fibrous connective tissue.

- When overfilling occurs, the process of phagocytosis may eliminate the excess root filling material and occasionally also material inside the canal.

WOUND HEALING OF APICAL PERIODONTITIS

- Follows the general principle of wound healing of connective tissues elsewhere in the body, with the formation of fibrovascular granulation tissue, removal of necrotic tissue and dead bacteria by activated macrophages, and finally repair and/or regeneration of the wounded tissue.

- Healing is largely accomplished by regeneration and to some degree by fibrosis.

- Local tissue resident cells involved in periapical wound healing are osteoblasts and bone marrow stromal cells in alveolar bone and multipotent stem cells in periodontal ligament.

- The extracellular matrix and growth factors of cementum (i.e., IGF-1, FGFs, EGF, BMP, TGF-β, PDGF) are capable of inducing proliferation, migration, attachment, and differentiation of multipotent stem cells in the periodontal ligament into cementoblast-like cells and produce cementoid tissue on the root surface denuded of periodontal ligament.

During periapical wound healing, the osteoblasts or mesenchymal cells lining the surfaces of endosteum

Stimulated by TGF-β, BMPs, IGFs, PDGF, VEGF, and cytokines released by stromal cells, osteoblasts, platelets, and bone matrix after bone resorption

Undergo proliferation and differentiation into osteoblasts

Produce bone matrix

- When one of the cortical bone plates (buccal or lingual/palatal) is destroyed

- Mesenchymal cells in the inner layer of periosteum beneath the oral mucosa – stimulated by TGF-β, BMPs,IGFs, PDGF, and VEGF

- Undergoes proliferation and differentiation into osteoblasts and produces bone matrix

- If both cortical bone plates are destroyed by large apical periodontitis lesions, it is possible that the lesion may be repaired with fibrous scar tissue because of extensive destruction of the periosteum beneath the oral mucosa.

- Guided tissue regeneration procedure and bone grafts is recommended to prevent ingrowth of fibroblasts from periosteum or submucosa into the bony defect and to enhance periapical wound healing if periapical surgery is necessary.

ENDODONTIC IMPLICATIONS (PATHOGENESIS OF APICAL PERIODONTITIS AS EXPLAINED BY FISH)

- Described the reaction of the periradicular tissues to bacterial products, noxious products of tissue necrosis, and antigenic agents from the root canal

- FISH in 1939 theorized that the zones of infection are not an infection by themselves but the reaction of the body to infection. Thus he concluded that the removal of this nidus of infection will result in resolution of infection.

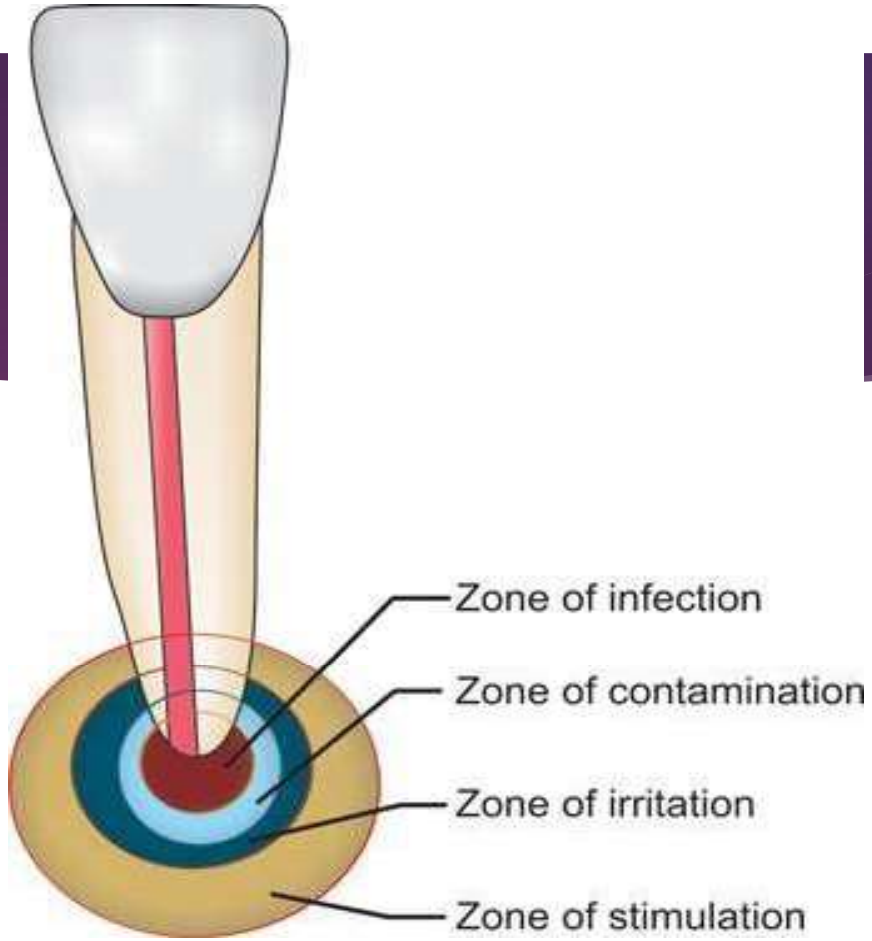
- Four well defined zones of reaction were found during the experiment:

a. Zone of infection or necrosis (*PMNLs)*

b. Zone of contamination *(Round cell infiltrate – lymphocytes)*

c. Zone of irritation *(Histiocytes and osteoclasts)*

d. Zone of stimulation *(Fibroblasts, capillary buds and Osteoblasts)*


Zone of infection
Zone of contamination
Zone of irritation
Zone of stimulation

FISH Zone

KRONFELD'S MOUNTAIN PASS THEORY

Kronfeld had explained that the granuloma does not provide a favorable environment for the survival of the bacteria. He employed the Fish concept so as to explain the tissue reaction in and around the granulomatous area.

Zone A

He compared the bacteria in the infected root canal with the invaders entrenched behind 'high and inaccessible mountains', the foramina serving as mountain passes.

Zone B

The exudative and granulomatous (proliferative) tissue of the granuloma represents a mobilized army defending the plains (periapex) from the invaders (bacteria). When a few invaders enter the plain through the mountain pass, they are destroyed by the defenders (leukocytes). A mass attack of invaders results in a major battle, analogous to acute inflammation.

Zone C

Only complete elimination of the invaders from their mountainous entrenchment will eliminate the need for a defense forces in the 'plains'. Once this is accomplished, the defending army of leukocytes withdraws, the local destruction created by the battle is repaired (granulation tissue) and the environment returns to its normal pattern.

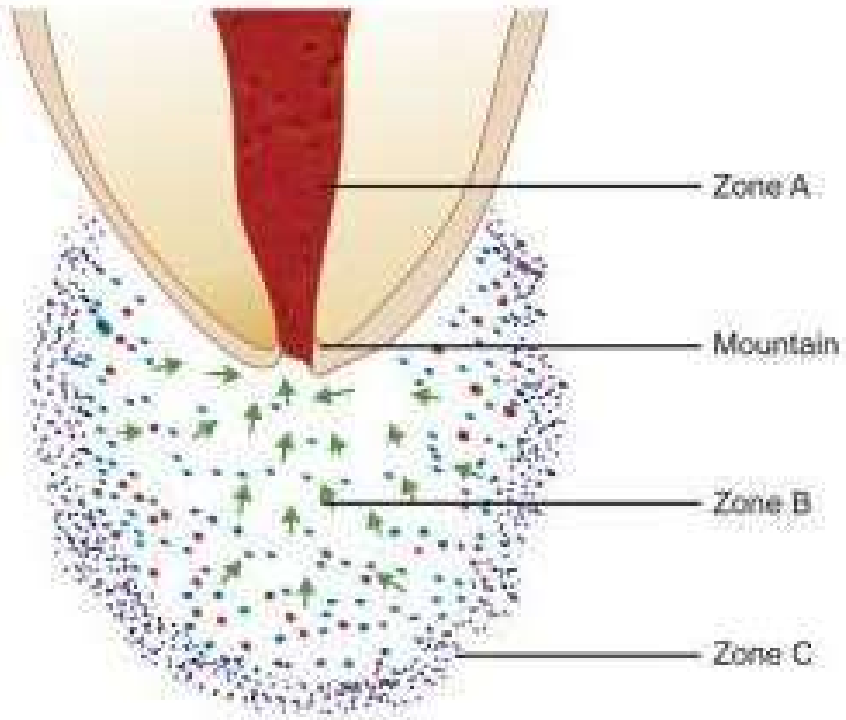


Fig. 6.11 Kronfeld's mountain pass theory

PERIAPICAL WOUND HEALING AFTER SURGICAL ENDODONTIC THERAPY

- Faster than nonsurgical endodontic therapy.
- Surgical debridement is done.
- Goal of surgical endodontic therapy is to seal microbial etiology within the root canal system by root- end filling in most cases.

Periapical Index (PAI)

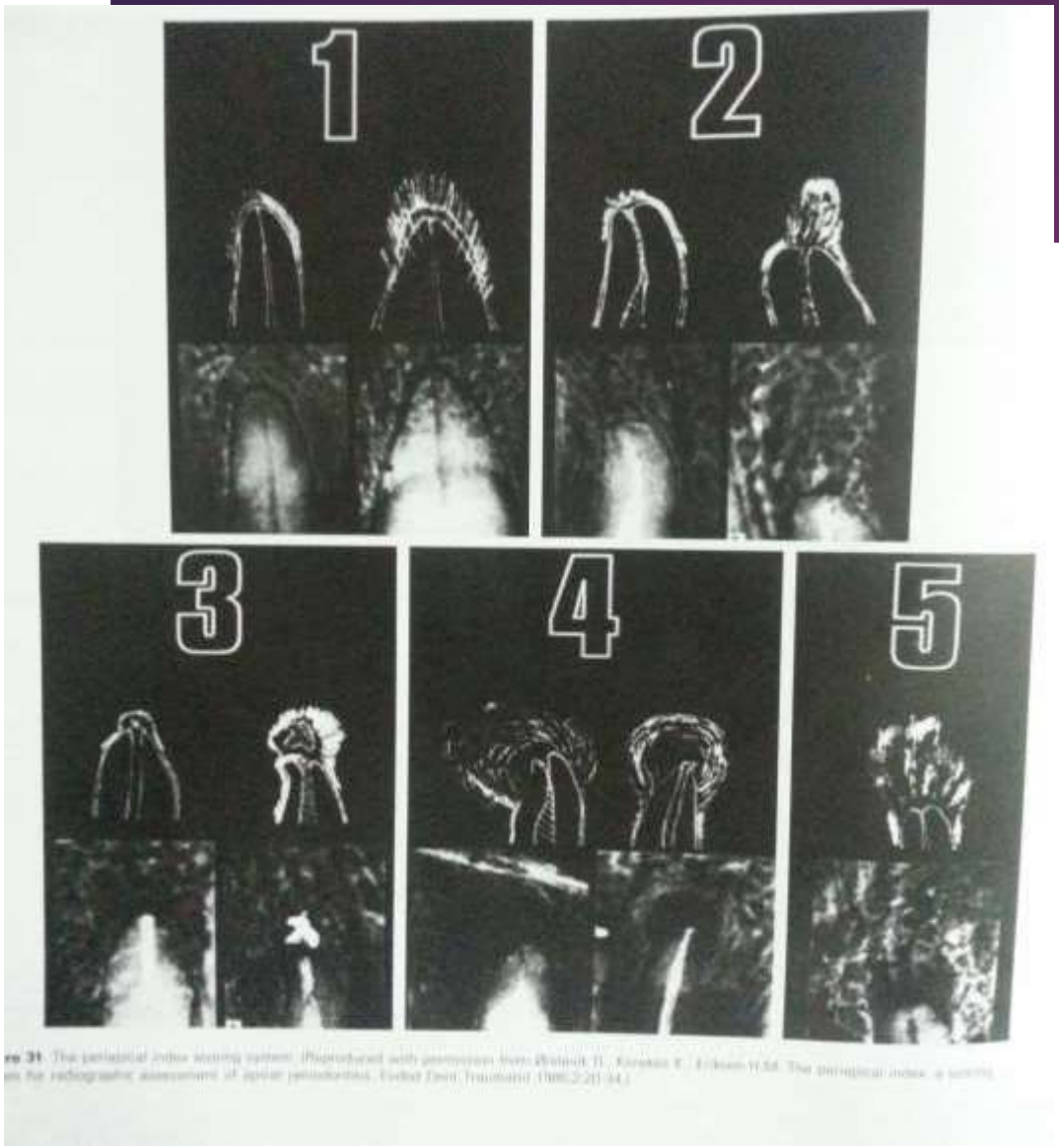


Fig. 9 The periapical index[6,7]

PAI 1 - Normal periapical structure

PAI 2 - Small changes in bone structure not pathognomic for apical periodontitis

PAI 3 - Changes in bone structure with some mineral loss characteristic of apical periodontitis

PAI 4 - Periodontitis with well defined radiolucent area

PAI 5 - Severe periodontitis with exacerbating features and bone expansion

HEALING OF ROOT FRACTURE

- Healing of root fracture depends upon:
  a) Site of the fracture
  b) Status of the pulp

- Two directions of wound healing response are expected:
  a) At the pulpal side
  b) At periodontal ligament side

- If pulp is intact at the fracture site, the odontoblastic progenitor cells will create a hard tissue bridge uniting the fractures fragments.

- During initial stage of wound healing, traumatized tissue stimulate an inflammatory response and trigger the release of a series of osteoclastic activity ,subsequently obliterating the fracture site.

Any of the following types of resorption may occur:

i. External surface resorption surrounding the proximal fracture edges at the periodontal side of fracture.

ii. Internal surface resorption surrounding the fracture edges centrally at the pulpal side of the fracture.

iii. Internal tunneling resorption burrows behind the pre-dentin layer and along the root canal walls of the coronal fragment.

The pattern of healing of root fracture are:

a) *Healing with calcified tissue*
b) *Healing with interproximal calcified tissue*
c) *Healing with interposition of bone and connective tissue*
d) *Interposition of granulation tissue*

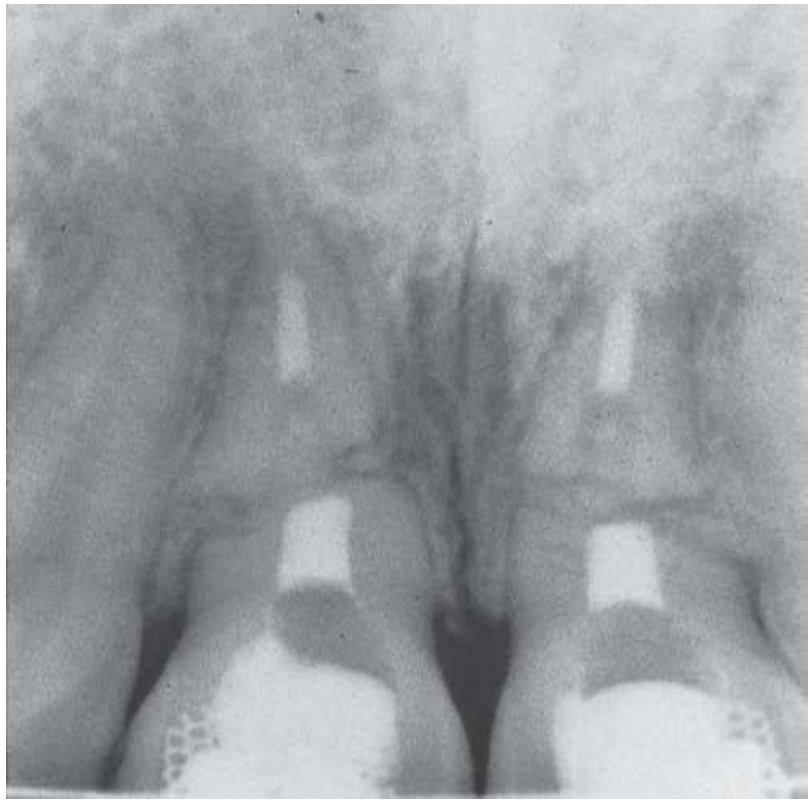
Healing With Calcified Tissue

- The calcified tissue is formed at the fracture site.

- The innermost layer of repair maybe of dentin while the more peripheral portion of the fracture is incompletely repaired with cementum.

- *Clinically* the teeth appears normal.

- *Radiographically*, the fracture line is discernible, but the fragments are in close contact.

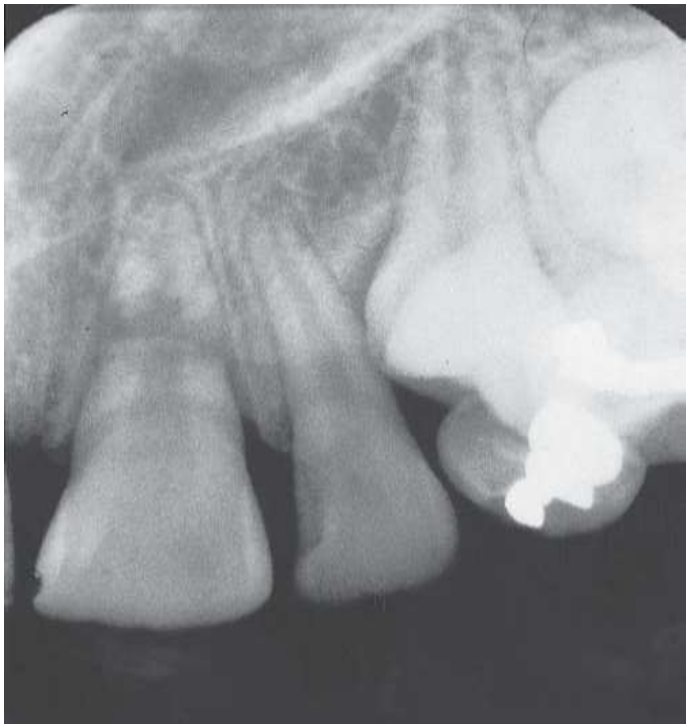


Healing With Interproximal Calcified Tissue

- Characterized by presence of connective tissue between the fragments.

- The fracture surfaces are covered by cementum along with connective tissue fibres running parallel to the fracture surface or from one fragment to the another.

- *Clinically* the teeth are firm or may be slightly mobile.
- *Radiographically*, the fragments appear separated by a narrow radiolucent line, and the fractured edges appear rounded.

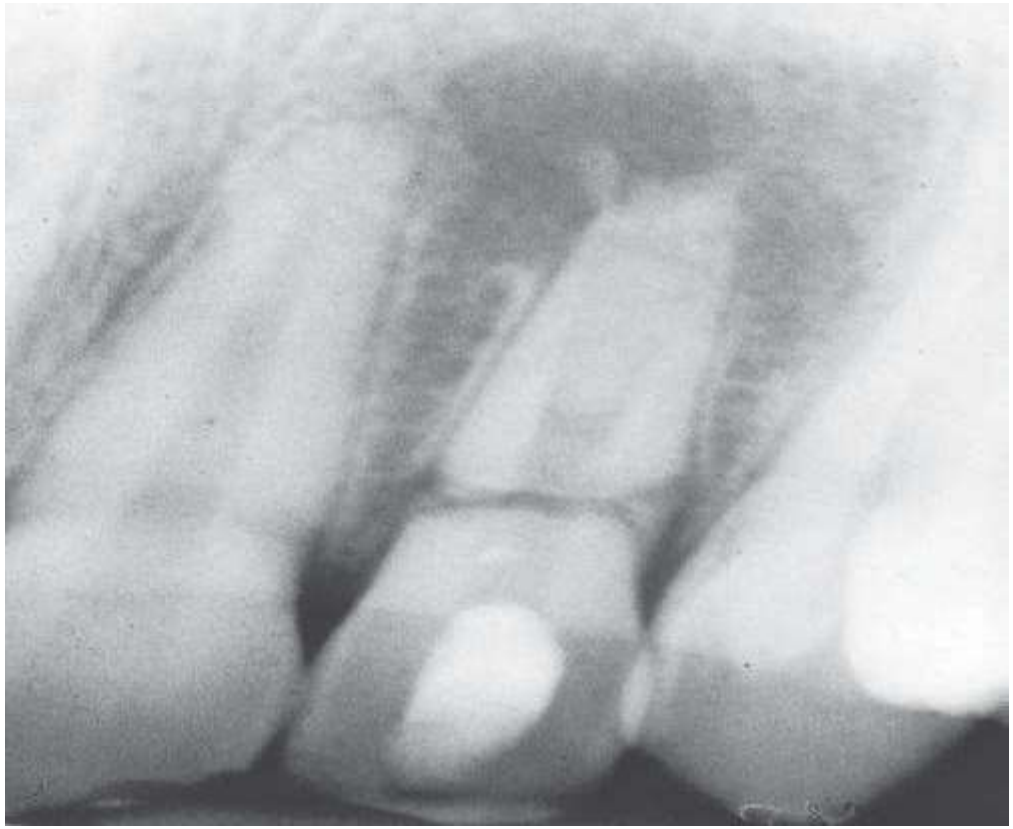


Interposition Of Granulation Tissue

- The fracture site is obliterated with granulation tissue.

- The coronal portion is necrotic while the apical portion is vital.

- Radiographically, widening of the fracture line and/or a developing radiolucency corresponding to the fracture line becomes apparent.

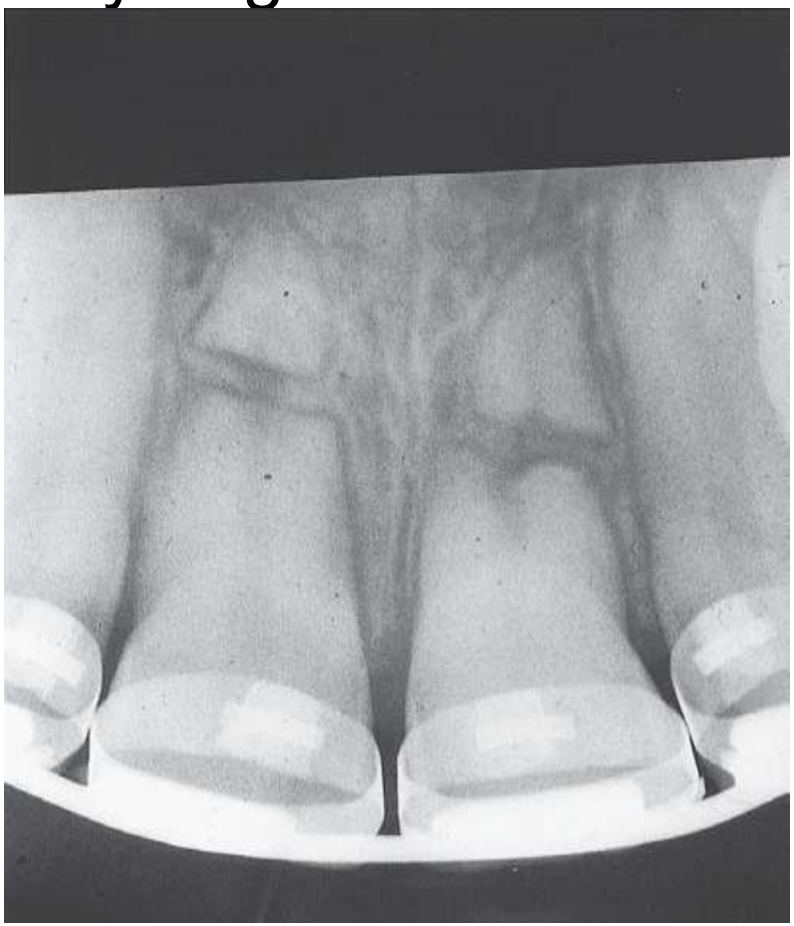


Healing with interposition of bone and connective tissue

- This mode of healing is a sequelae of trauma prior to completed growth of the alveolar process
- Thus, the coronal fragment continues to erupt while the apical fragment remains stationary.
- Interposition of bone and connective tissues is seen along the fracture site.

- *Clinically* the teeth are firm and react normal to pulp tests.

- *Radiographically*, the fragments are seperated by a distinct bony ridge.



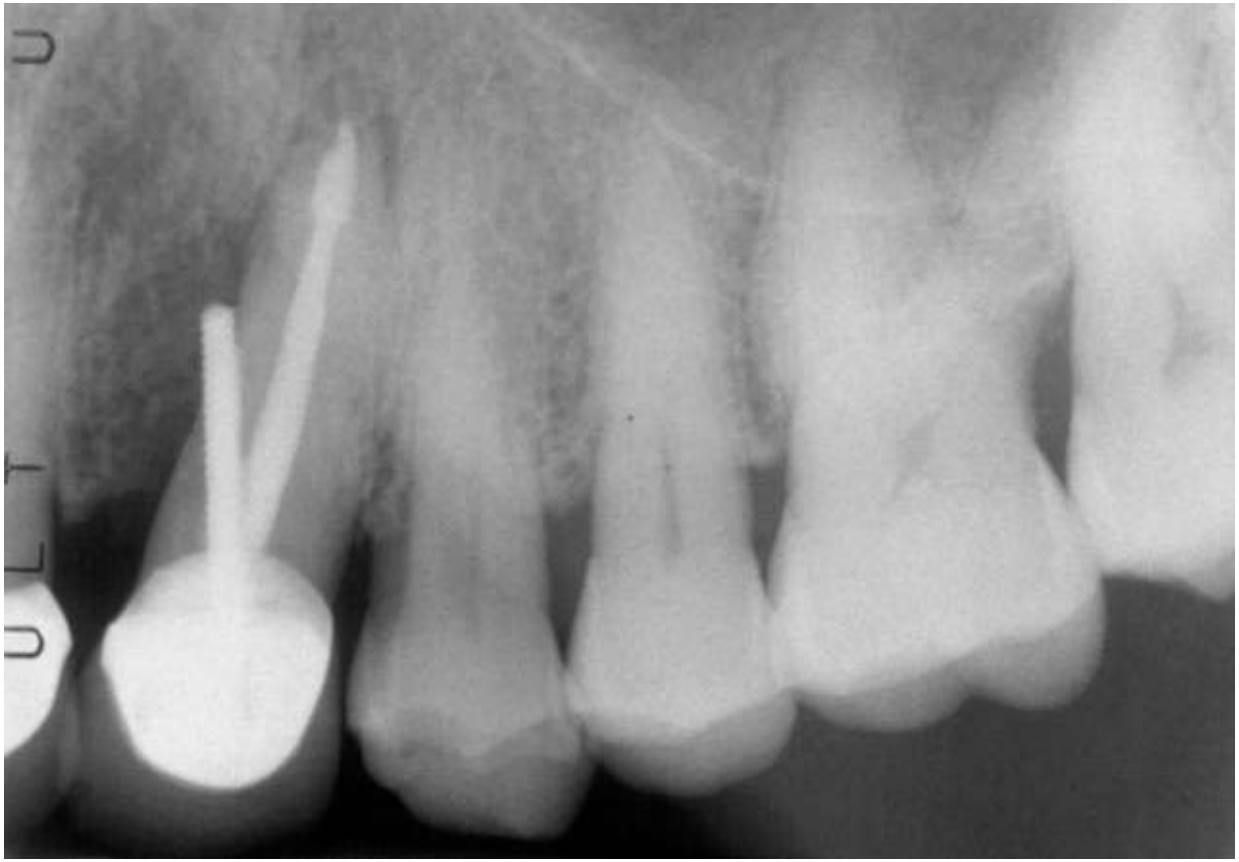
HEALING IN PATIENTS ON CHEMOTHERAPY/ RADIATION THERAPY

- These patients have impaired healing responses.
- Pulp may become necrotic during radiation therapy.
- Symptomatic nonvital teeth should be endodontically treated 1 week before initiating radiation or chemotherapy whereas asymptomatic nonvital teeth may be delayed.

PERFORATION AND PROGNOSIS FOR HEALING

- An endodontic perforation is an artificial opening in the tooth or its root, created by the clinician during entry to the canal system or by a biologic event such as pathologic resorption or caries that results in a communication between the root canal and the periodontal tissues.
- A furcation perforation refers to a mid-curvature opening into the periodontal ligament space and is a worst possible outcome in root canal treatment.

- A post space perforation is defined as a communication between the lateral root surface and the surrounding periodontal structures due to misdirection or an excessively large post enlargement.



Timing Of Perforation Repair

- The main complication arising from perforation is the potential for secondary inflammation of the periodontal attachment with eventual infection and ultimately tooth loss if untreatable.

- The timing of repair categorized into immediate or delayed.

- Immediately closing the communication between the periodontal tissues and the root canal system promotes a superior healing potential.

Location Of Perforation

- Perforations can be categorized by location:
  a) Subgingival
  b) Midroot
  c) Apical
- Once perforations exhibit the formation of osseous defects, the prognosis is compromised significantly.

- Any perforation located near the gingival sulcus promoted inflammation and loss of epithelial attachment, resulting in periodontal pocket formation.

- Apical and midroot perforations without communication to the oral cavity has a good prognosis provided an immediate seal was obtained .

Size Of Perforation

- Small perforations of the canal space promote a direct and immediate restoration of the defect.

- It offers fewer chances for periodontal breakdown and epithelial proliferation within the perforation site.

Conclusion:

- Understanding of wound healing is as important as knowing the pathogenesis of disease, because satisfactory wound healing is the ultimate goal of treatment.

- If we are able to understand the mechanism of periapical wound healing, we can design treatment approaches that maximize favorable conditions for wound healing to occur.

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Thank You