HEALING OF BONE FRACTURE

By : Dr Diwakar vasudev
PG 2nd year .

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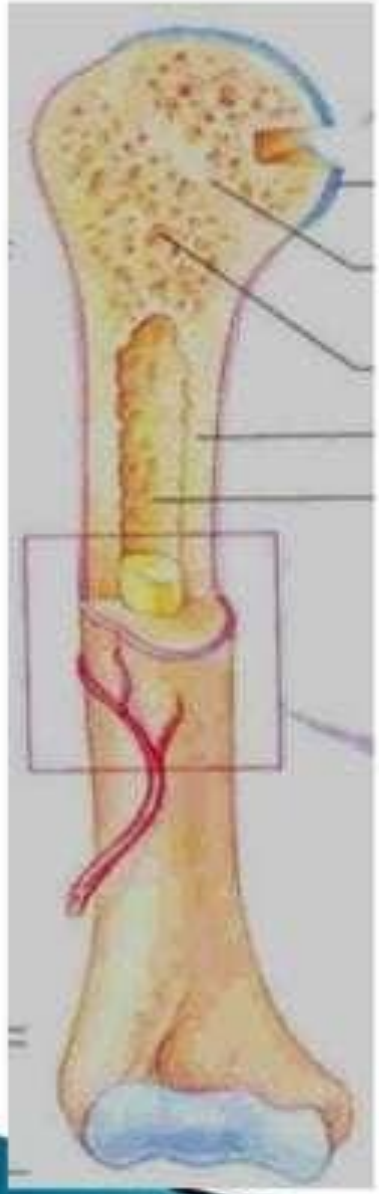
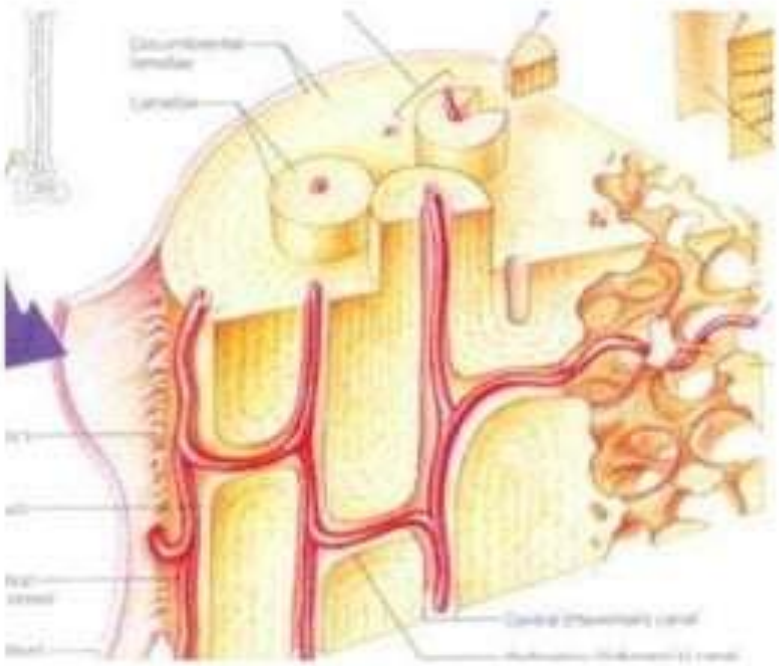
INTRODUCTION

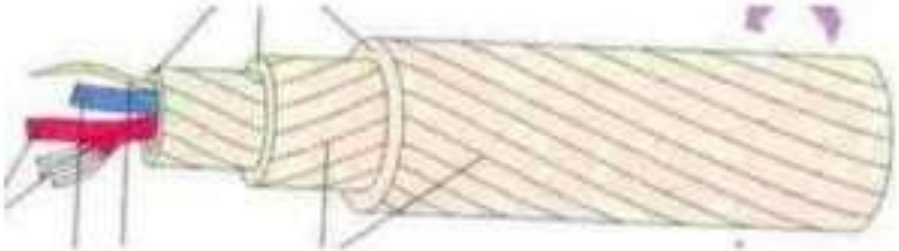
The study of repair of maxillofacial skeleton must begin logically with an understanding of nature of biologic response to osseous injury and repair



- Bone is ***unique*** structure with several specific functions
- Major reservoir of Calcium
- Support to the human frame
- ***Origin*** and ***insertion*** of muscles
- ***Protects*** vital soft tissues.
- Locomotion.



- ***Bone*** and ***liver*** in the body only organs capable of undergoing spontaneous ***regeneration.***
- It is surrounded by a fibrous sheet called as ***periosteum*** outer sheath ***fibrous layer*** and **inner_layer** called as ***cambium*** layer. which is source of ***new bone cells***



- **Endosteum**:Inner portion of bone marrow cavity is lined with ***fibrous sheet*** called as endosteum .
- **Haversian system** or **osteon** is the ***functional unit*** in mature bone

BONE COMPOSITION

- Water 8%
- Soild material 92 %
- Organic phase 21%
- Inorganic phase 71%

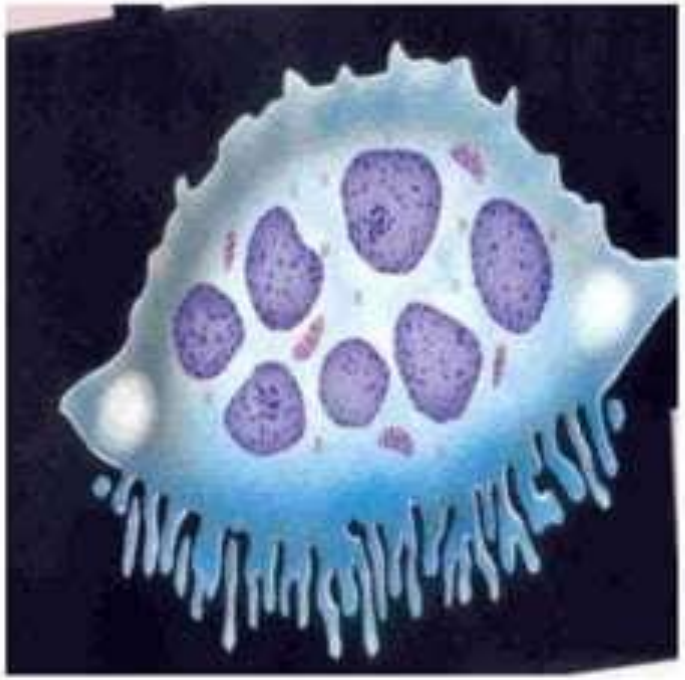
CELLS INVOLVED IN BONE HEALING

- Platelets
- Neutrophils
- Macrophages
- Mast cells
- lymphocytes

- Osteocyte
- Osteoblast
- Osteoclast
- Fibroblast

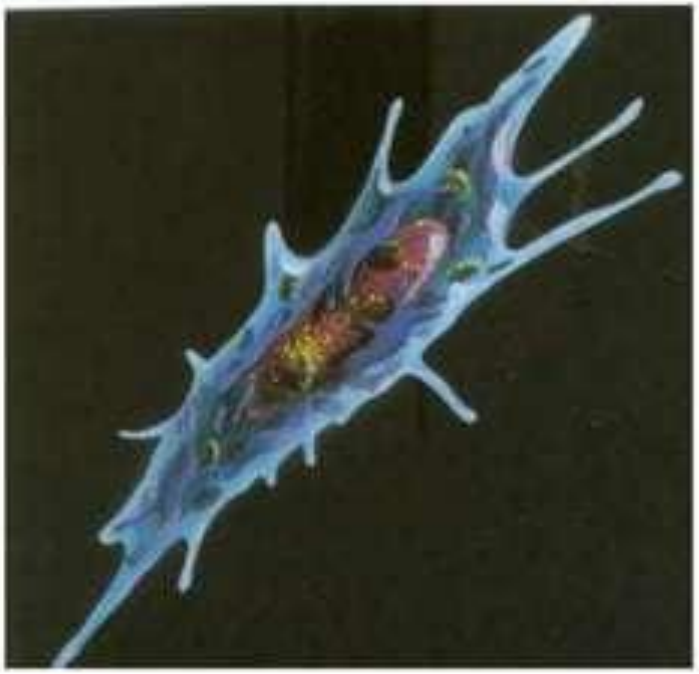
OSTEOBLAST

- Uninucleated cell
- Synthesis of collogeneous[1&5] and non collagenous bone protein
- Modulation of osteoclastic function.
- Osteoblast secretes some BM then get entrapped within lacunae called as Osteocytes.
- No. of osteocytes indicate rapidity of bone formation or repair
- Multipotential Mesenchymal cells



OSTEOCLAST

- Multinucleated giant cell
- Much larger cell
- They are found against the bone surface hallowed out depressions-Howships Lacunae [Themselves have created]
- Resorption of the bone .
- Hemopoeitic in origin.



FIBROBLAST

- Form the extra cellular fibers of CT. i.e. collagen and elastin , ground substance.
- produces motility and contractions in CT.
- Called as Architect ,Builder , and Care Taker of connective tissue

BONE HEALING

- Process of bone healing has many features similar to that of skin healing expect it also involve calcification of connective tissue matrix .
- Bone heals by means of regeneration rather that repair.

TYPES OF BONE HEALING

- Types of bone healing
- Direct bone healing (Primary bone healing , Callus free healing)
- Indirect bone healing (Secondary bone healing , Callus forming healing healing)

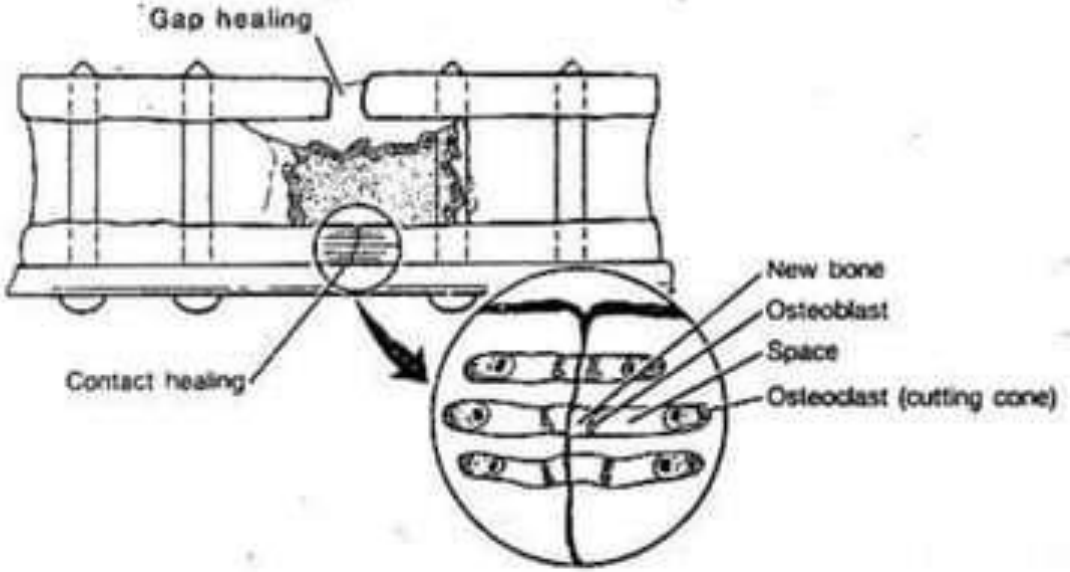
PRIMARY BONE HEALING

- PRIMARY BONE HEALING
- It occurs in following condition
  - Excellent Anatomical reduction.
  - Minimal or no mobility.
  - Good vascular supply.
  - It can also occur without rigid fixation if there is no gross mobility.

PRIMARY BONE HEALING

- **Osteogenic cells and Capillaries** proliferate in the medullary bone on both sides of fracture , forming new bone along the fracture site.

- Primary healing types
- Gap healing
- Contact healing



GAP HEALING

- Even with rigid fixatation ,in some areas of fracture ,small gaps occur between the bone segments.
- Blood vessels from Periosteum, Endosteum or Haversian Canals invade the gaps bringing osteogenic cells into the gap.
- Bone is deposited directly over the fracture ends without resorption or intermediate cartilage formation.

GAP HEALING

- If Gap is < 0.3mm - Lamellar Bone forms directly.
- 0.5-1 mm Woven bone formation and lamellar bone subsequently laid down within the trabecular spaces
- At the end of six weeks lamellar bundles oriented at right angle to the longitudinal axis of remaining bone.Over several months ,remodeling then leads to change in this direction.

CONTACT HEALING

- Interfragmentary gap is essentially zero
- vascular and cellular ingrowth can not proceed as that occurs in gap healing.
- Bone heals by means through the formation of bone metabolizing unit. (BMU)
- Osteoclast begin to cut away cores on either side of the fracture progessing towards the fracture site at a rate of 50- 80 um / day.

CONTACT HEALING

- Core which is 200 um in diameter provide a pathway for vessel ingrowth and osteoblasting proliferation with new bone formation. (Pegging together).
- Osteon forms at a rate of 1-2 um/day.
- This lag between resorption and osteon ingrowth produce transient porosity in the compact bone visible radiographically for 3 months after fracture in humans.

SECONDARY BONE HEALING

Intermediate fibrous tissue is formed

- **INITIAL** STAGE .
- **CARTILAGINOUS** CALLUS.
- **BONY** CALLUS .
- **REMODELING**

INITIAL STAGE

- Inflammatory response
- Hematoma formation
- New vascularity
- Mesenchymal cells that differentiate to form the fibrocartilgenous callus

CARTILAGINOUS CALLUS

- It begins externally as well a internally
- External nodules of cartilage are separated by septa.
- Blood vessels increases, tendency towards hypoxemia is reversed

CARTILAGINOUS CALLUS

- **Calcification** of cartilage
- **Trapping** of chondroblasts and conversion to chondrocyte.
- **Osteoblast** from endosteam

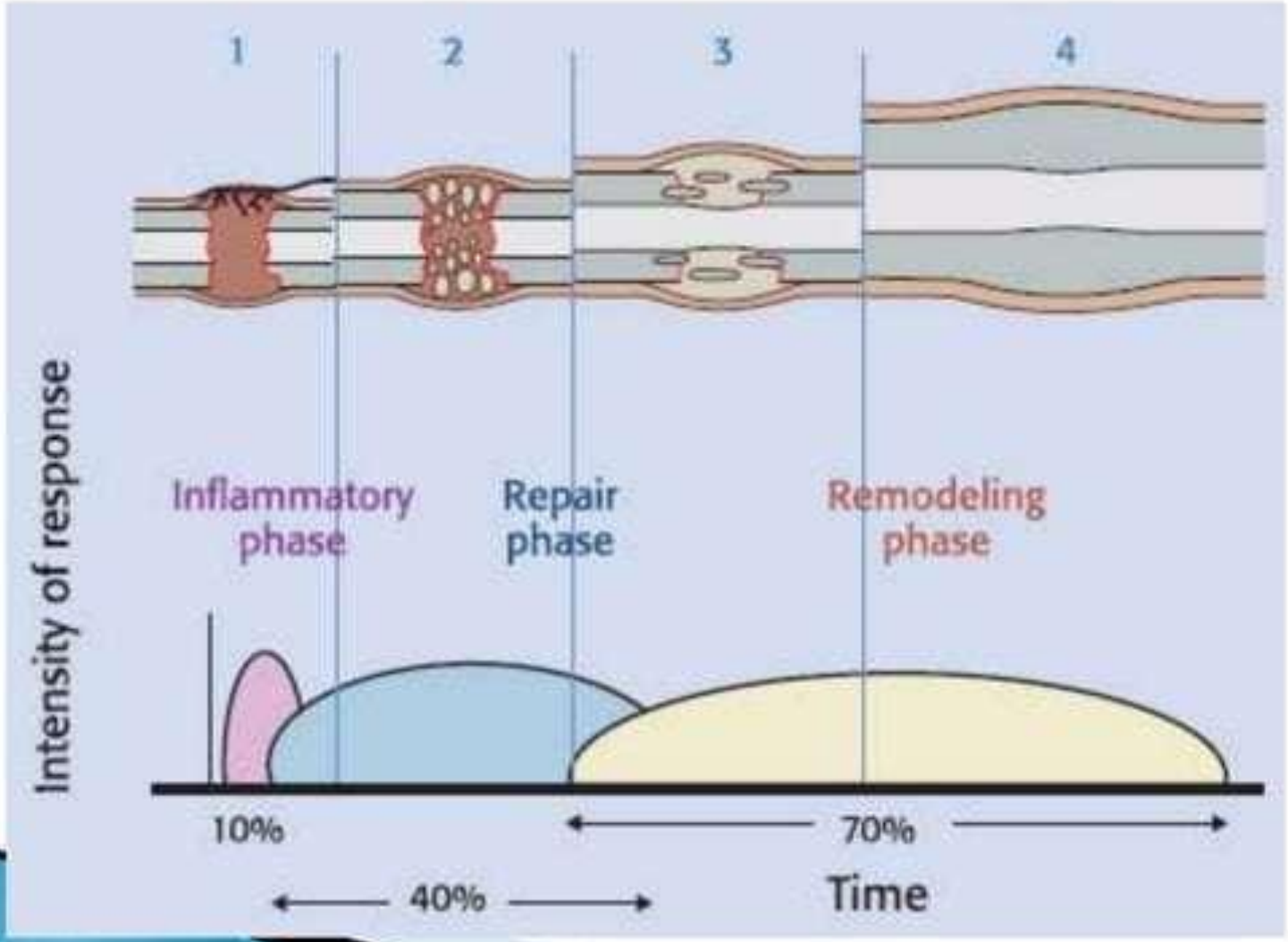
Hard callus formation

- Same as endochondral Bone formation.
- Calcification of cartilage-
  woven bone formation
- Increased vascularity & nutrients-osteoblast-osteoid-bone

Bone remodelling

- Initially bone is randomly arranged
- Woven bone is more familiar pattern of lamellar bone.
- This is slow process progresses in accordance in *wolfs* law.

PHASES OF FRACTURE HEALING



FACTORS AFFECTING(LOCAL)

ADVERSE	FAVORABLE
Infection	Good apposition
Pathological #	Good immobilistion
Poor apposition& alignment	Good blood supply
Continuing movement of bone ends	
Poor blood supply	

FACTORS AFFECTING (SYSTEMIC)

- Nutrition(protein,vitamins)
- Age
- Irradiation
- Immunosuppression
- Steroids

BIOCHEMICAL FACTORS

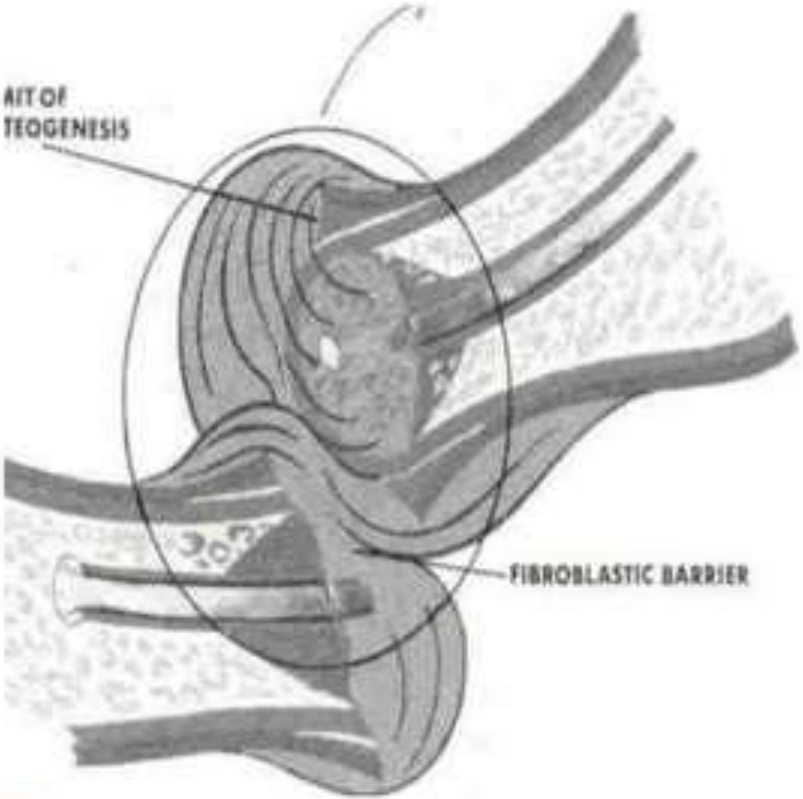
Resorbing

- PTH
- Vit D3
- Interleukin 1
- TNF
- Prostaglandin

Inhibitors

1. Calcitonin
2. Glucocoticoids
3. Bisphospnates
4. Indomethacin &aspirin
5. TGF-b

COMPLICATIONS



- Malunion
- Nonunion
- Delayed union

DELAYED UNION

Fracture that has not healed in the expected time for type of fracture, patient and method of repair.

Causes

- Inadequate blood supply
- Severe soft tissue damage
- Periosteal stripping
- Excessive traction
- Insufficient splintage
- Infection

- **Clinical features**

Persistant pain at fracture site
Instability of fracture site

X-RAY
Visible fracture line
Very little callus formation or periosteal reaction

- **TREATMENT**
- Conservative
- To eliminate any possible cause
- Immobilization

- Operative

Indication

Union is delayed more than 6 months

No signs of callus formation

Internal fixation and bone grafting

MALUNION

Condition when the fragments join in an unsatisfactory position (unaccepted angulation, rotation).

Causes

Failure to reduce a fracture adequately.

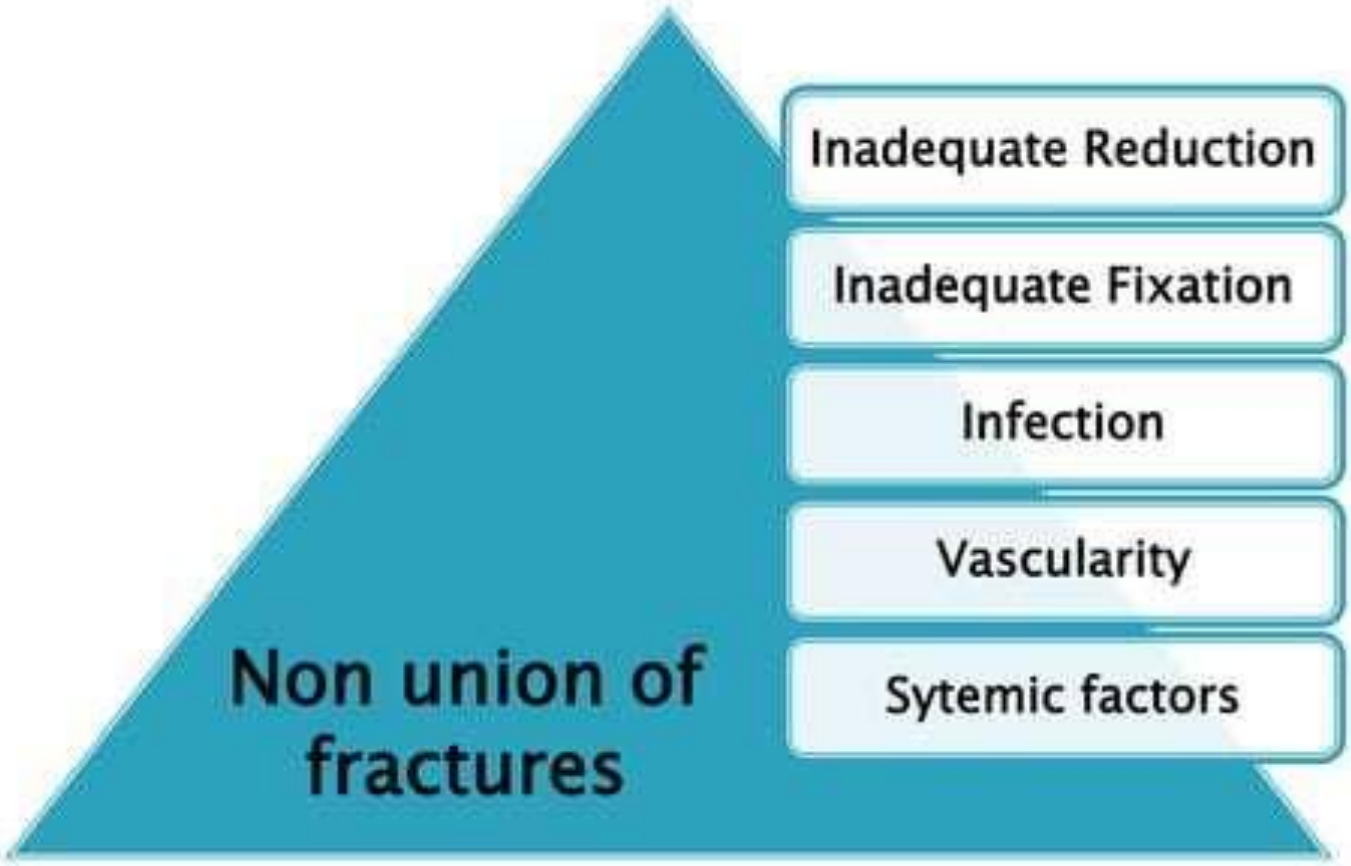
Failure to hold reduction while healing proceeds.

Gradual collapse of comminuted or osteoporotic bone

Treatment : Osteotomy and internal fixation.

NON UNION OF FRACTURES

- Result of an impairment or delay in the natural healing process of bone.
- Implies a failure of fracture hematoma to become transformed into an osteogenic matrix and ultimately converted into non osteogenic fibrous tissue
- Mobility of the bone ends in all planes after an interval of time 10 weeks


Non union of fractures
Inadequate Reduction
Inadequate Fixation
Infection
Vascularity
Sytemic factors

INADEQUATE REDUCTION

- Inadequate reduction can result in marked distraction of fracture fragments
- Excessive traction from muscles insertion onto the fracture fragments or may be secondary to interposition of soft tissue in between both the ends.

INADEQUATE FIXATION

- It can result in excessive motion at the fracture site producing delayed or malunion .
- Motion can lead to formation of external callus and secondary bone healing.
- Excessive motion can lead to disruption of fragile capillaries that migrate into fracture hematoma resulting in delay or lack of maturation of hematoma.

- Limited but not excessive movement at the fracture site may be beneficial
- Motion at the fracture fragments stimulate the stabilization of the callus.
- The callus tissue response is felt to be function of bioelectric potentials that are generated with in the bone stimulate osteoblastic production and activity

INFECTION

- Actue or chronic osteomyelitis at the fracture site can lead to delayed or non union.
- The decrease in the local PH, Coupled with excessive mobility during the early phase of osteogenesis, influences the piezoelectric aspect of bone formation in turn affecting the orientation of the fibroblast migrating across the fracture line.

VASCULARITY

- The decreased vascularity causes reduction in oxygen tension at fracture tension
- It leads to differential survival of fibroblast over more specialized cells producing a fibrous union or nonunion.
- With old age blood supply to mandible changes from centrifugal to centripetal force
- Principle source of blood supply is from the periosteium thus excessive periosteal stripping should be avoided in atrophic mandible.

SYSTEMIC FACTORS

- Vitamin C and D deficency have effects on collagen metabolism and mineralization
- Anemia effects on tissue oxygenation
- Long term steroids causes osteoporotic changes in bones
- Aging
- Diabetes

DIFFERENCE BETWEEN CALLUS AND BONE

CALLUS	BONE
Chondrocytes	Osteocyte
Fibroblast	Osteoblast
	Osteoclast
Soft	Hard
Molding within weeks	Several months
Hydroxyapatite-less	More

HEALING IN BONE GRAFTS

- Classical healing occurs with inflammatory cells followed by ingrowth of new vessels and replacements of necrotic tissues

OSTEOCONDUCTION

- PROCESS of capillary and perivascular tissues in growth from the host recipient bed into the graft.

OSTEOINDUCTION

- Process by which one tissue acts on the another tissues to induce cellular differentiation
- Bone has a this ability through component known as BMP

BONE MORPHOGENIC PROTEIN

- Polypeptide protein 17500 M Wt
- Irreversibly induces the conversion of pericytes ⇒ 0steoprogenitor cells, chondrogenesis and bone formtion

IMBIBATION

- Transferred graft initially derives the nutrition via serum from recipient site

INOSUCULATION

The graft slowly gains blood supply from the recipient site by in growth of blood vessels.
It begins within 48 hrs

NON VASCULAR BONE GRAFTS

- Undergo slight necrosis
- Osteyocyte on the surface reestablish blood supply and survives
- The remaining graft is infiltrated by blood vessels from recipient site & repopulated by recipient mesenchymal stem cells

VASCULAR BONE GRAFTS

- Cells of this type graft fully survives
- Their blood supply is only temporarily interrupted
- There is no dead bone matrix formation that must go revasculeristion,osteoindution
- This healing process is similar like fracture Healing

CANCELLOUS BONE GRAFTS

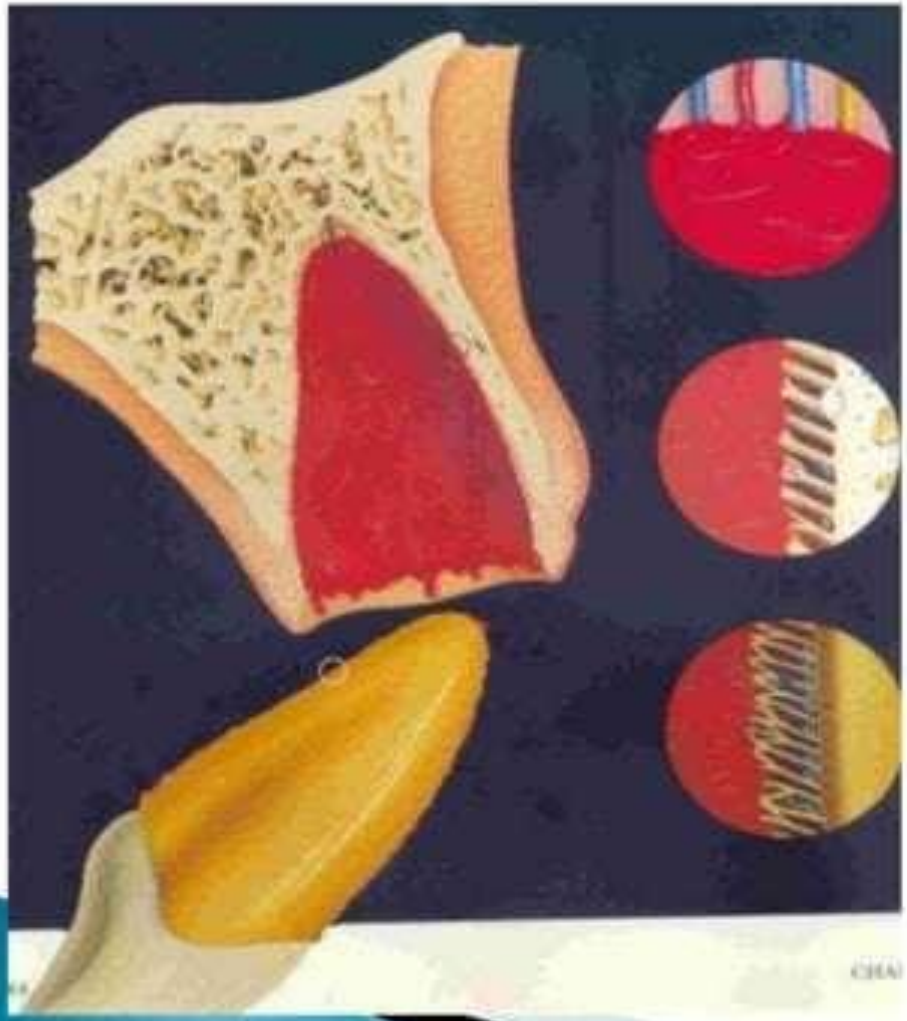
- Revascularization starts within hours &complete by first 2 weeks
- Primitive mesenchymal cells(from host and graft)– osteoblasts osteoid around nonviable bone.
- nonviable bone gradually resorbed and replaced with new bone

CORTICAL BONE GRAFTS

- Revascularization much slower rate.(1-8 weeks)
- Osteoclastic function starts first
- Osteoblstic activity after 2 weeks & continue until 6 months
- Bone that has not under gone resorption can be sealed off by new bone
- At the end cortical bone grafts tend remain admixtures of necrotic and viable bone.

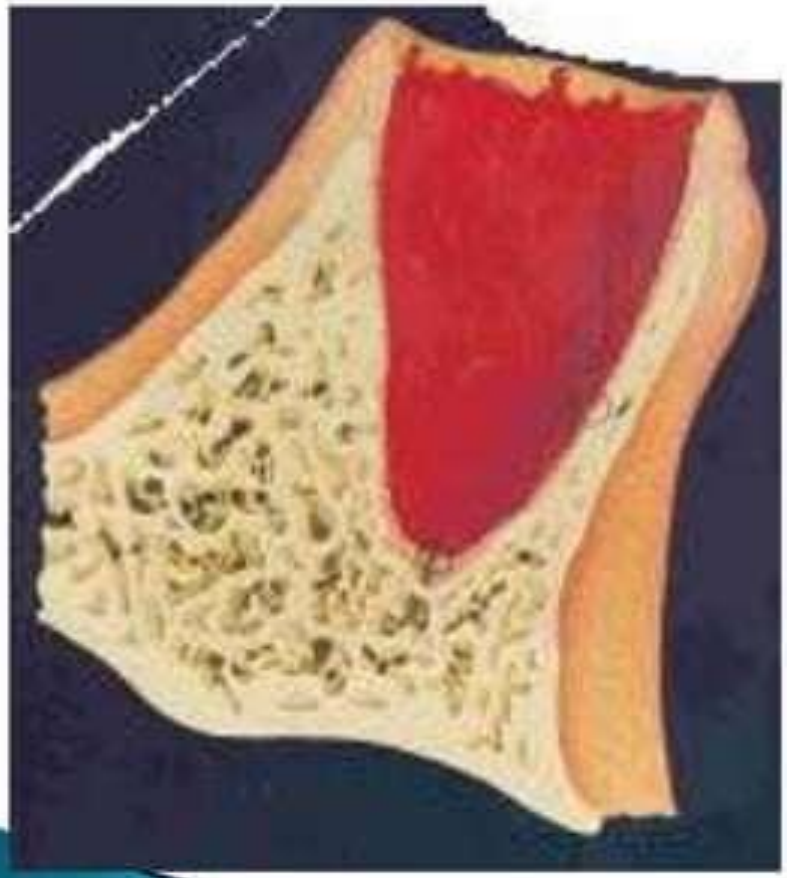
	Cancellous	Cortical
Vascularization	Hours-2 weeks	1-8weeks
1 st function	Osteoblastic	Osteoclastic
Resorption	Complete	Incomplete
End	viable bone	Admixture of necrotic bone& viable new bone

SOCKET HEALING



- Stage 1- Coagulum
- Stage 2- Granulation tissue formation.
- Stage 3-Connective tissue formation.
- Bone 4-Bone development begins.

STAGE 1- COAGULUM FORMATION



- Coagulum is formed once hemostasis has been established it consists of RBC,WBC in same ratio of circulating blood,entrapped within fibrin

STAGE 2-GRANULATION TISSUE FORMATION

Tissue is formed along the socket walls **2-3 days** post operatively & is characterized by proliferating **endothelial** cells, **capillaries** and many leucocytes .**within 7days** ,granulation tissue has usually replaced the coagulum

STAGE 3-CONNECTIVE TISSUE FORMATION

- Connective tissue formation begins peripherally and , within 20 days post operatively ,replaces the granulation tissue this newly formed CT is comprised of cells, collagen and reticular fibers dispersed in a metachromatic ground substance

STAGE 4-BONE DEVELOPMENT

- Bone development starts within 7days . By 38 days the socket is full of immature bone.
- Complete healing of socket is usually by 6 months (Aust. Dental Journal)

COMPLICATIONS

Dry socket [Crawford 1896]

- Alveolitis sicca dolorosa.
- Alveolar osteitis.
- Localized osteitis.

Failure in stage1 and 2 in socket healing

PREDISPOSING FACTORS

- Extraction site
- Gender
- Trauma
- Smoking
- Vasoconstrictors
- Microorganisms
- Oral contraceptives
- Radiotherapy

PATHOGENESIS OF DRY SOCKET

BIRN HYPOTHESIS 1973 (Insitu fibrinolysis)

- Increased in fibrinolytic activity in dry socket.
- Plasminogen (activated) Plasmin.
- Plasmin like activity in dry socket was not present in normal extraction socket.
- Both Physiologic and non physiologic activity can activate the plasminogen.

ACTIVATORS OF PLASMINOGEN

Physiologic (Direct)

Released to alveolar bone after cell trauma

Extrinsic
tPA
ePA

Intrinsic
Factor XII
Urokinase

Non physiologic (Indirect)

Bacteria

Streotokinase

Stpylokinase

Chemical

Glycerol

chloroform

CLINICAL FEATURES

- ✓ Severe **pain, halitosis**
- ✓ Empty socket,very sensitive bone surfaces and covered by a layer of necrotic tissues and food particle. (Roger.E.Alexander, JOMS 2000)

TREATMENT OF DRY SOCKET

IOSR-JDMS May 2014 (S.preetha)

- Irrigation
- Medical dressing
- Analgesics
- Surgical intervention

CONCLUSION

Both hard&soft tissue could be injured in truama,hence surgeons must have an under standing of basic fracture healing and its management

- THANK YOU.....