TARGET ORTHO

# BASICS OF BONE AND FRACTURE HEALING

BY DR DAIVIK T SHETTY

TARGET ORTHO

# WHAT IS A BONE ?

FRCS Q

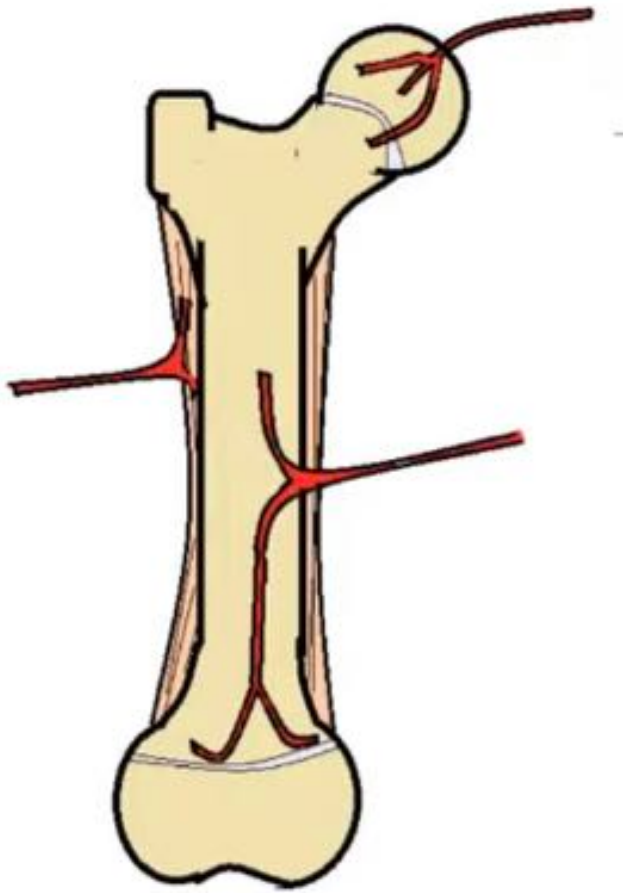
COMPOSITE AND DYNAMIC FORM OF **SPECIALIZED** CONNECTIVE TISSUE WHICH IS **ANISOTROPHIC**

TARGET ORTHO

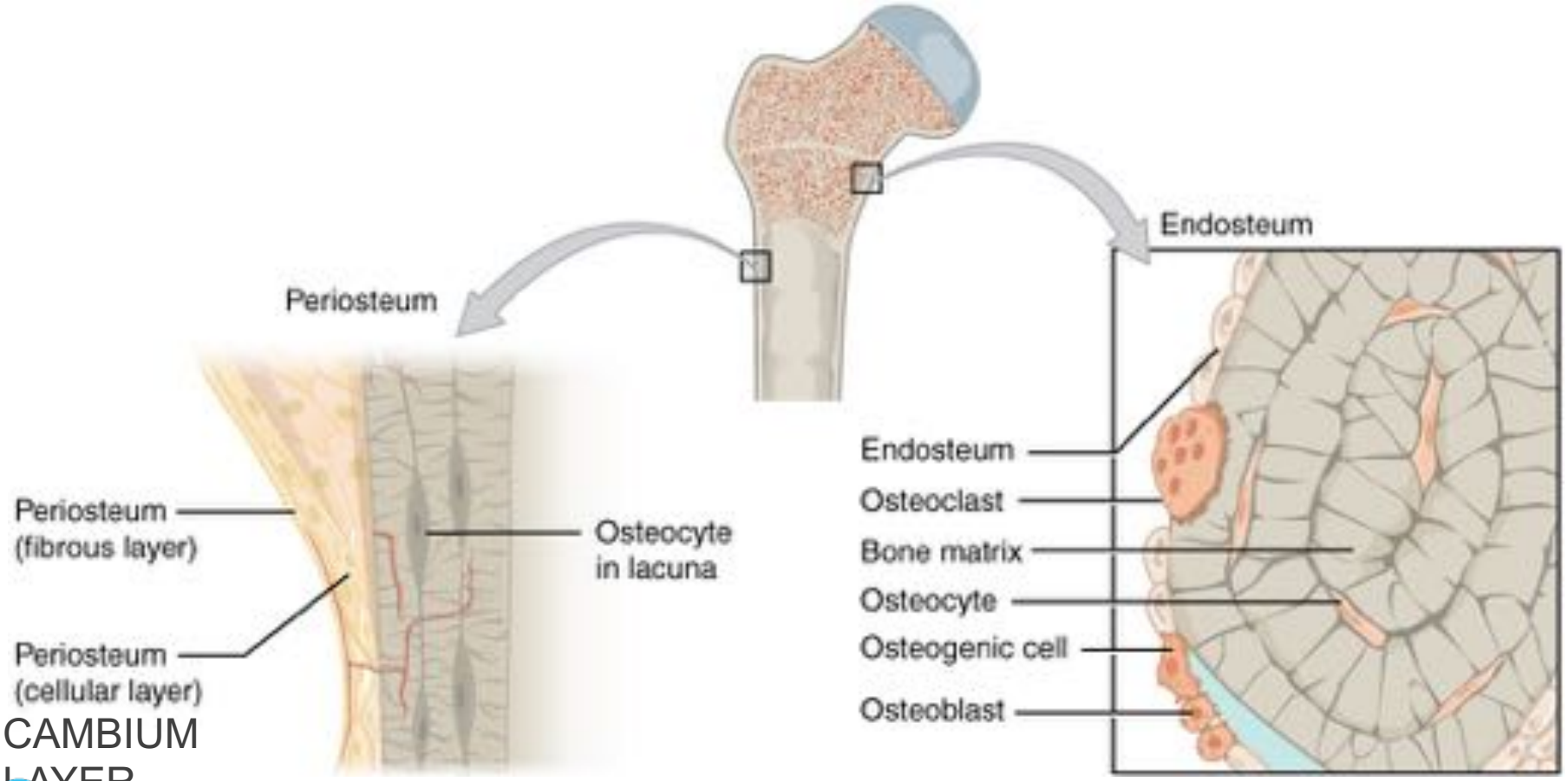
# FUNCTIONS OF BONE

- POSTURE
- PROTECTION
- PROPULSION
- PROPRIOCEPTION
- PRODUCTION

STRONG IN COMPRESSION
WEAK IN TENSION
WEAKEST IN SHEER



# PERIOSTEUM

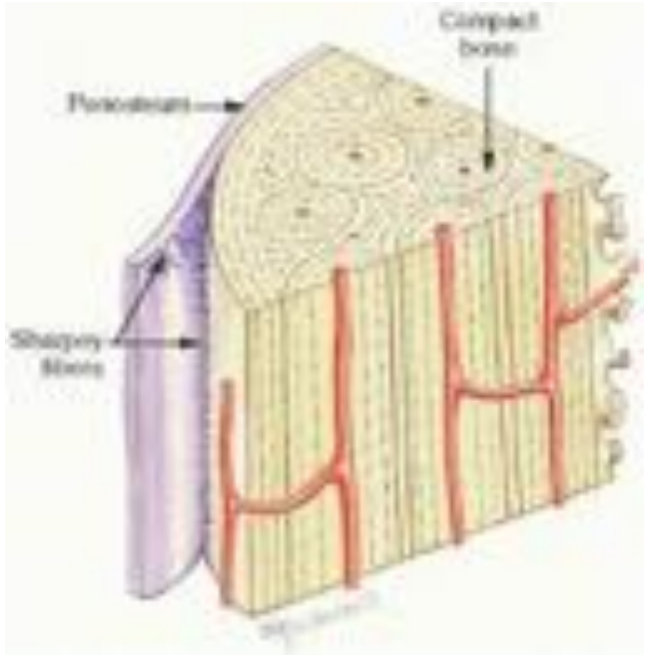



CAMBIUM LAYER

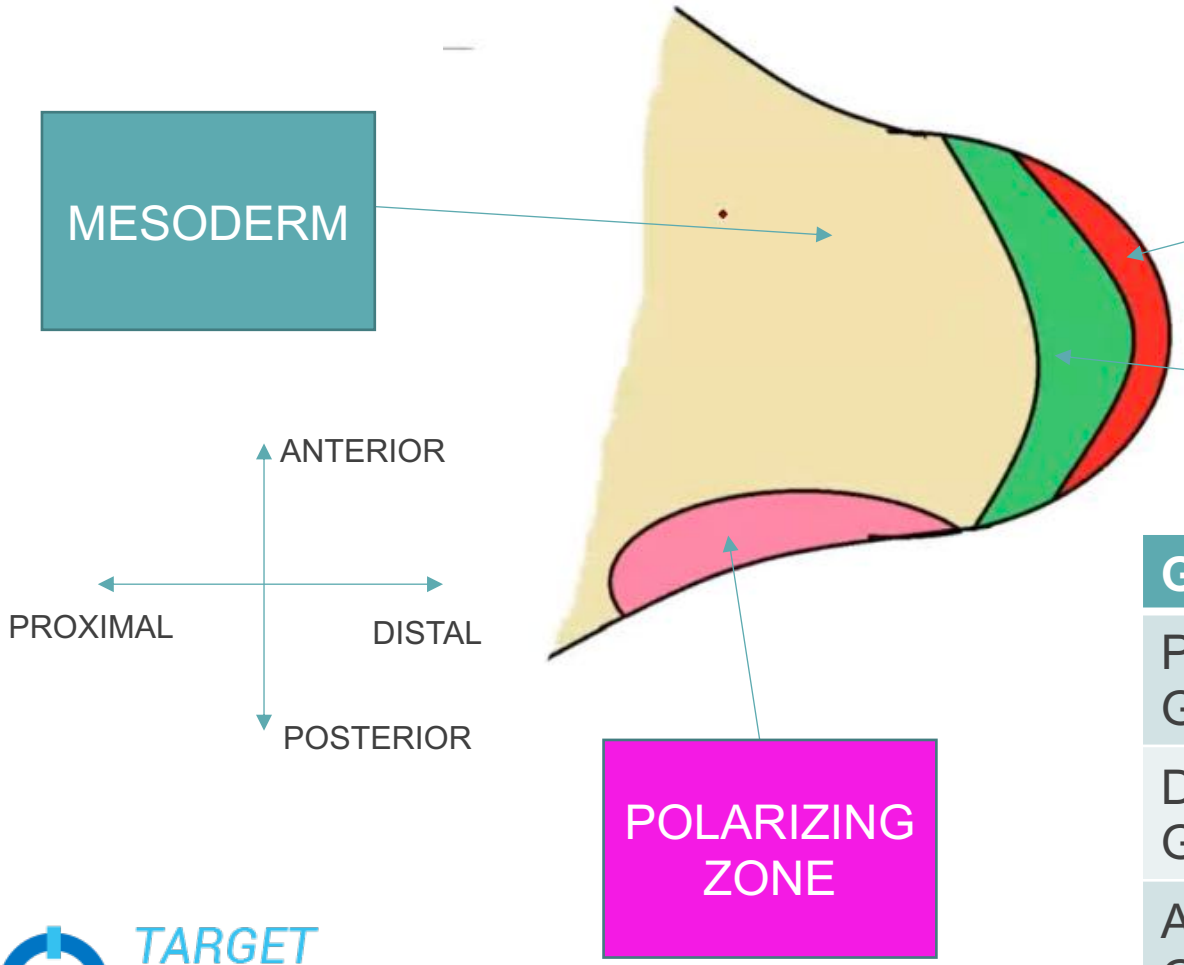
# PERIOSTEUM

- FIBROUS SHEATH PROVIDES NUTRITION AND GROWTH
- COVERS ENTIRE BONE, EXCEPT ARTICULAR SURFACE AND SESAMOID BONES (e g PATELLA)
- SHARPEY FIBRES -

ANCHOR PERIOSTEUM TO BONE



# BONE DEVELOPMENT




APICAL RIDGE

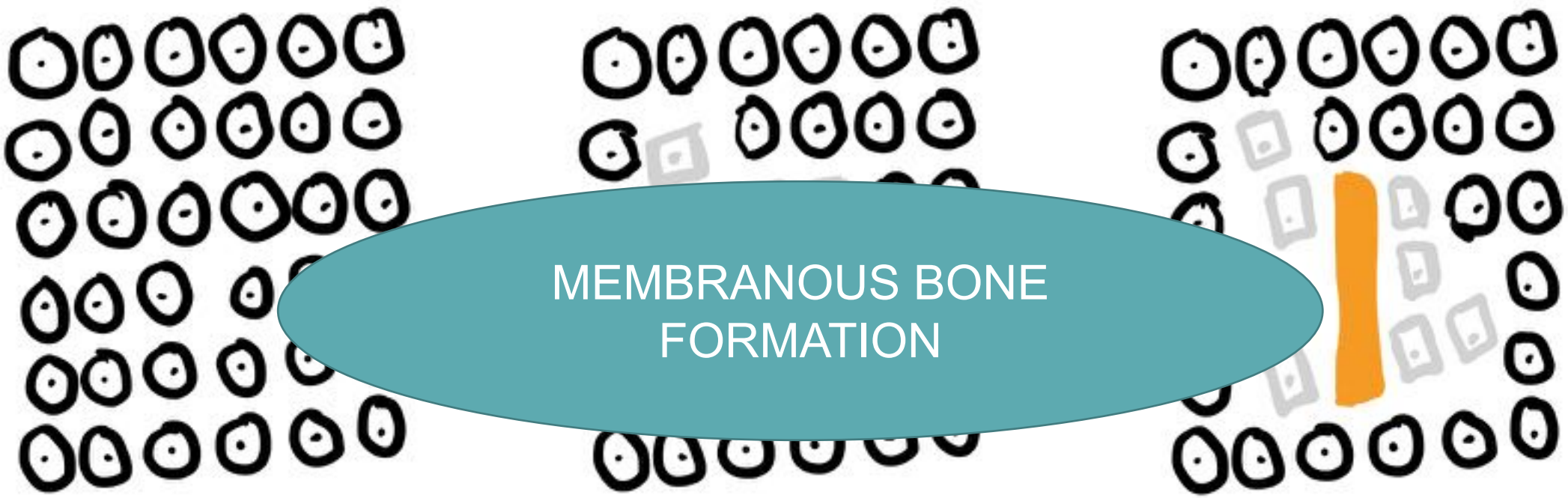
PROGRESS ZONE

| GROWTH | REGULATED BY |
|---|---|
| PROXIMAL DISTAL GROWTH | HOX GENE - HOMEOBOX |
| DORSOVENTRAL GROWTH | WNT GENE – WINGLESS INTEGRATED |
| ANTERO- POSTERIOR GROWTH | SHH GENE SONIC HEDGEHOG |

# OSSIFICATION

- THE PROCESS OF FORMATION OF BONE IS CALLED OSSIFICATION
- OCCURS EITHER BY INTRAMEMBRANOUS OSSIFICATION OR ENDOCHONDRAL OSSIFICATION

# BONE DEVELOPMENT



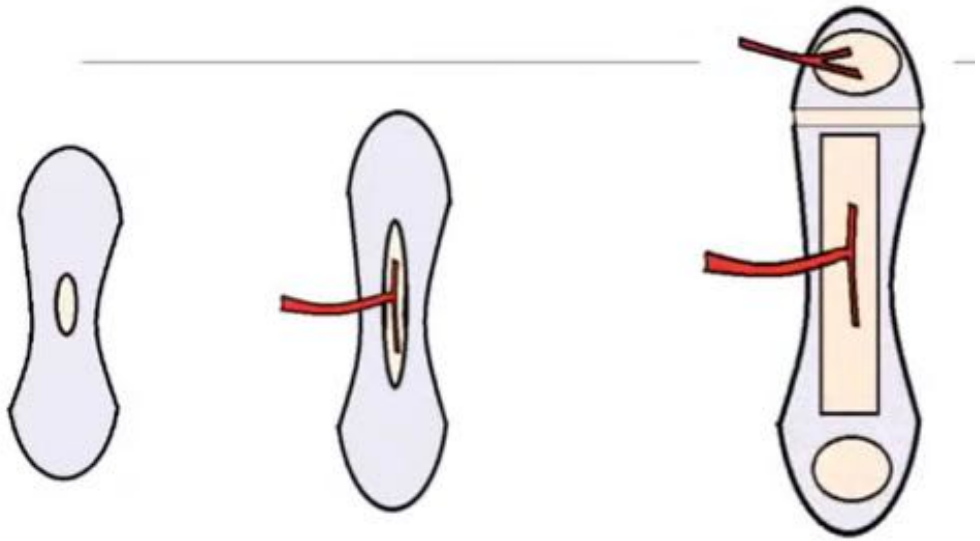

PRIMITIVE MESODERMAL CELLS

SOME TRANSFORM INTO PRE- OSTEOBLASTS

ORGANIZE AND CONVERT INTO BONE

# BONE DEVELOPMENT

ENDOCHONDRAL OSSIFICATION



# LAW OF OSSIFICATION

- AS PER THIS LAW, THE SECONDARY CENTER THAT APPEARS FIRST, FUSES FIRST

- THE BONE THAT DOES NOT OBEY THIS IS **FIBULA**

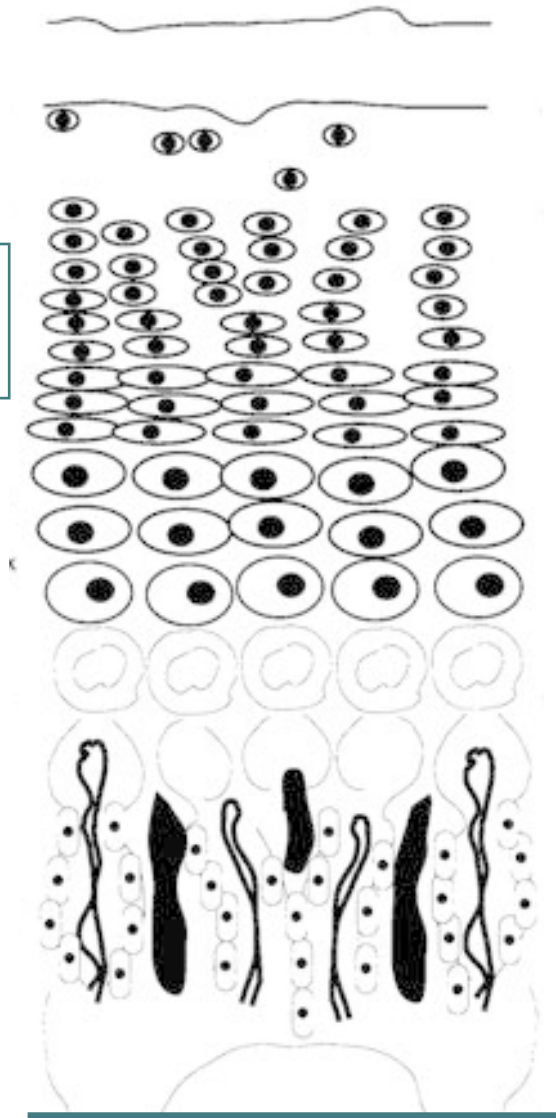
# LAYERS OF GROWTH PLATE

DIRECTION OF GROWTH

EPIPHYSIS

RESERVE/GERMINAL ZONE

PROLIFERATIVE ZONE



HYPERTROPHIC ZONE

DEGENERATIVE ZONE AND ZONE OF PROVISIONAL CALCIFICATION

PRIMARY AND SECONDARY SPONGIOSA

METAPHYSIS

# RESERVE / GERMINAL ZONE

- LIES IMMEDIATELY ADJACENT TO EPIPHYSIS
- CONTAINS RESTING CARTILAGE CELLS

- MOST IMPORTANT LAYER – LONGITUDINAL GROWTH

DISORDERS

INJURY – LONGITUDINAL GROWTH ARREST

LYSOSOMAL STORAGE DISORDER

DIASTROPHIC DWARFISM

# PROLIFERATIVE ZONE

- PROLIFERATING CARTILAGE CELLS ARE SEEN
- NUMBER OF CELLS IN THIS ZONE SHOWS ACTIVITY IN GROWTH PLATE

DISORDERS

GIGANTISM

ACHONDROPLASIA

# HYPERTROPHIC ZONE

- PRESENCE OF HYPERTROPHIC CELLS , UNGERGO VACUOLIZATION
- WEAKEST PART OF PHYSIS

DISORDERS

SCFE

MUCOPOLYSACCHARIDOSIS

# ZONE OF PROVISIONAL CALCIFICATION

- IN THIS AREA , CELLS BECOME IMPREGNATED WITH CALCIUM SALTS
- CALCIFICATION OF MATRIX

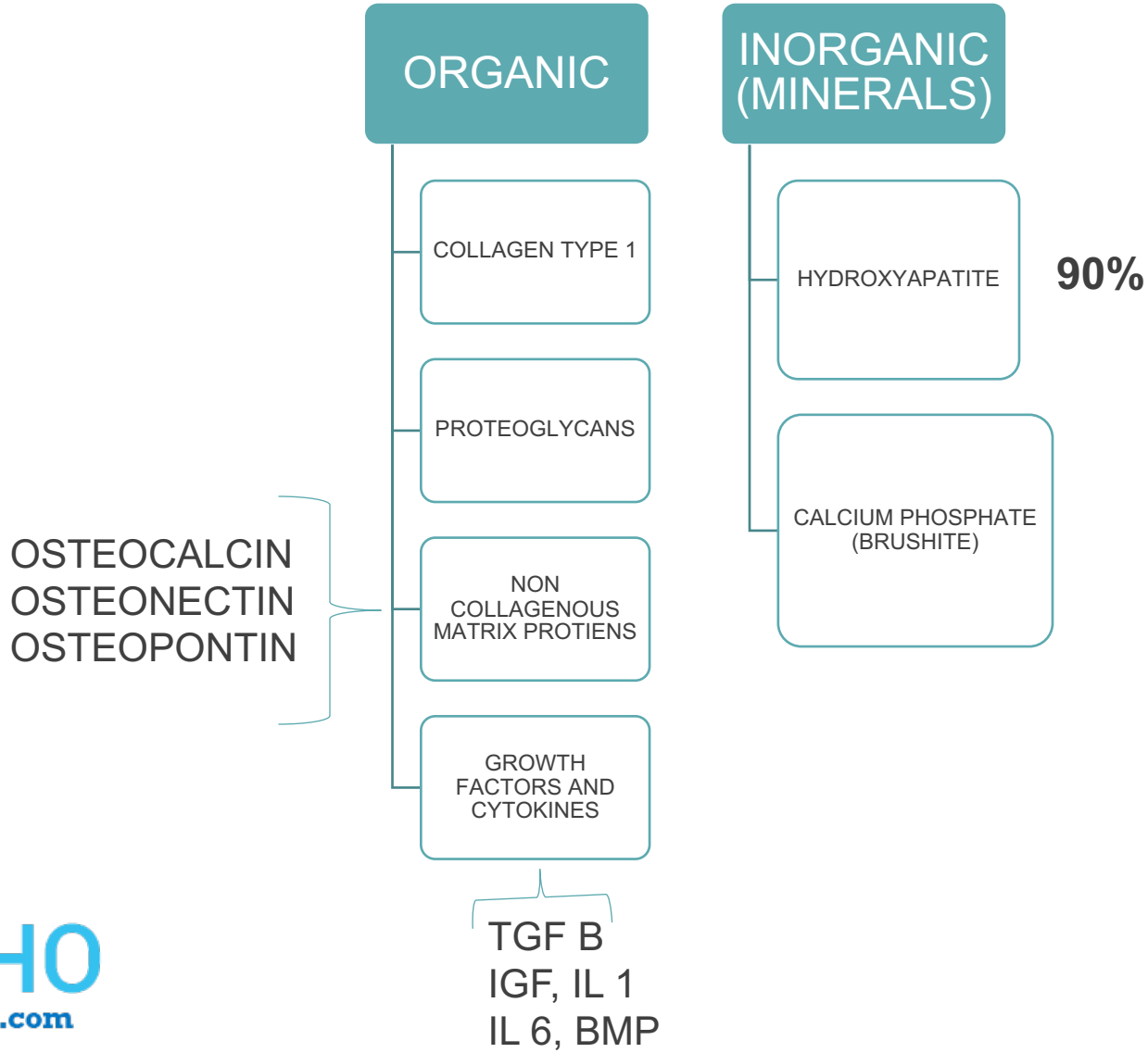
DISORDERS

RICKETS – FAILURE OF CALCIFICATION

# PERCENTAGE OF GROWTH

- SHOULDER – 40 %
- ELBOW – 20 %
- WRIST – 40 %
- HIP – 12 %
- KNEE 70%
- ANKLE – 18 %

# BONE



# BONE MATRIX

| INORGANIC | ORGANIC |
| --- | --- |
| Ca – Hydroxyapatite<br>**Ca10(PO4)6(OH)2** | Type 1 Collagen – 90% of organic matrix |
| **99%** of body Ca2+<br>**85 %** of body PO4+ | Responsible for **tensile** strength of bone |
| **40-60%** of total body Na+ and K+<br>Responsible for **compression** strength of bone | Proteoglycan |

- Most abundant non collagenous protein in bone is?

A. Alpha 2-HS-glycoprotein
B. TGF-beta
C. Osteocalcin
D. Osteopontin

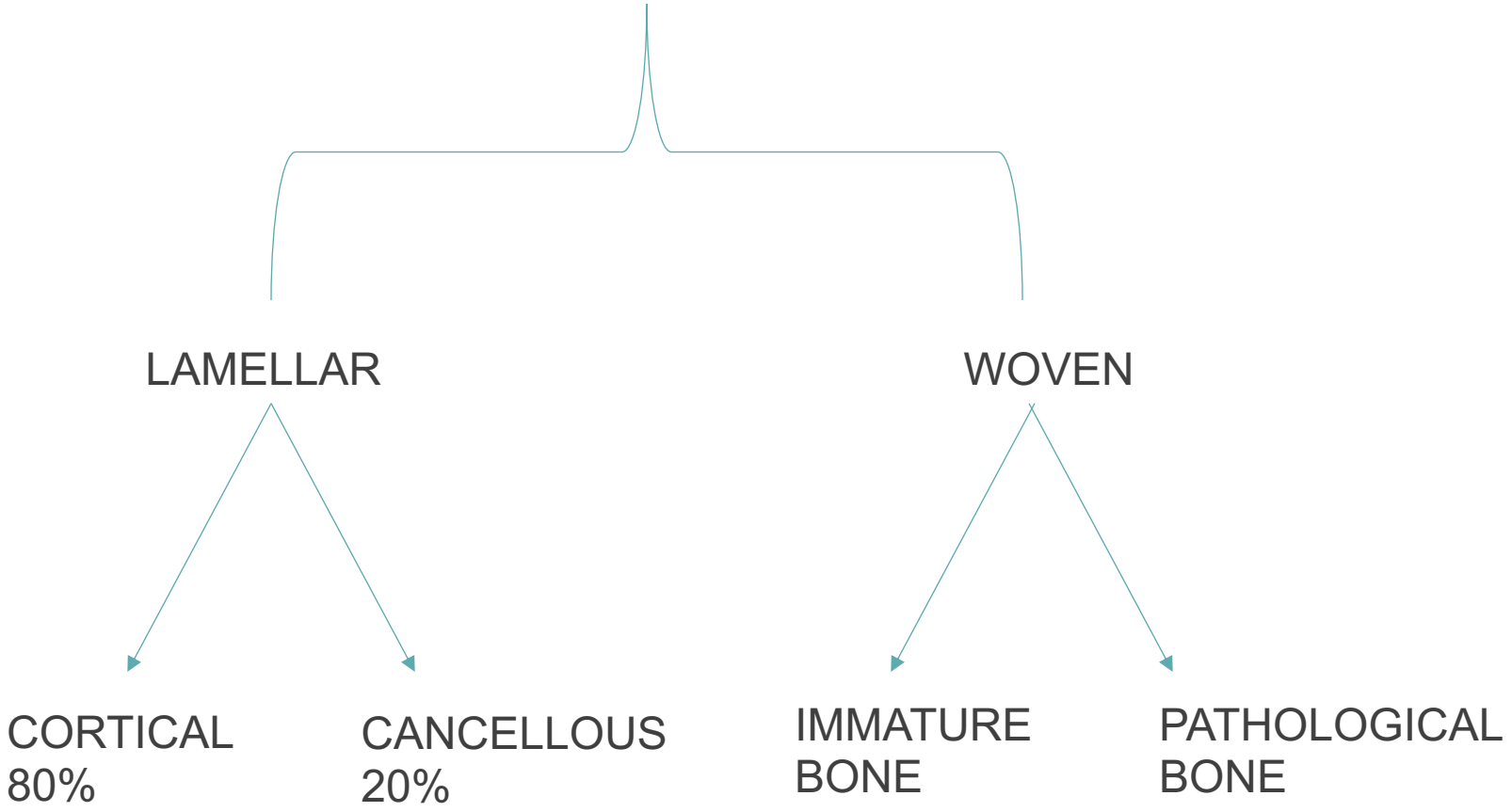
**C – Osteocalcin**
most abundant noncollagenous protein in bone

MCQ

# OSTEOCALCIN

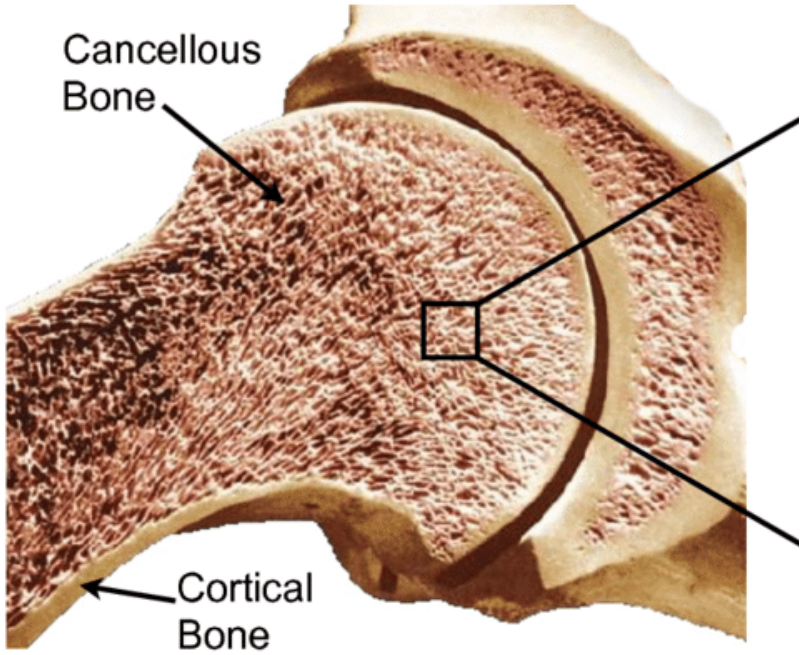
- IT IS A PROTIEN THAT IS EXCLUSIVELY PRODUCED BY OSTEOBLASTS
- HENCE USED AS A MARKER – DETECTION OF WHICH IS SUFFICIENT TO LABEL A STRUCTURE AS BONE
- ITS CONCENTRATION OF BLOOD IS DIRECT MEASURE OF OSTEOBLASTIC ACTIVITY

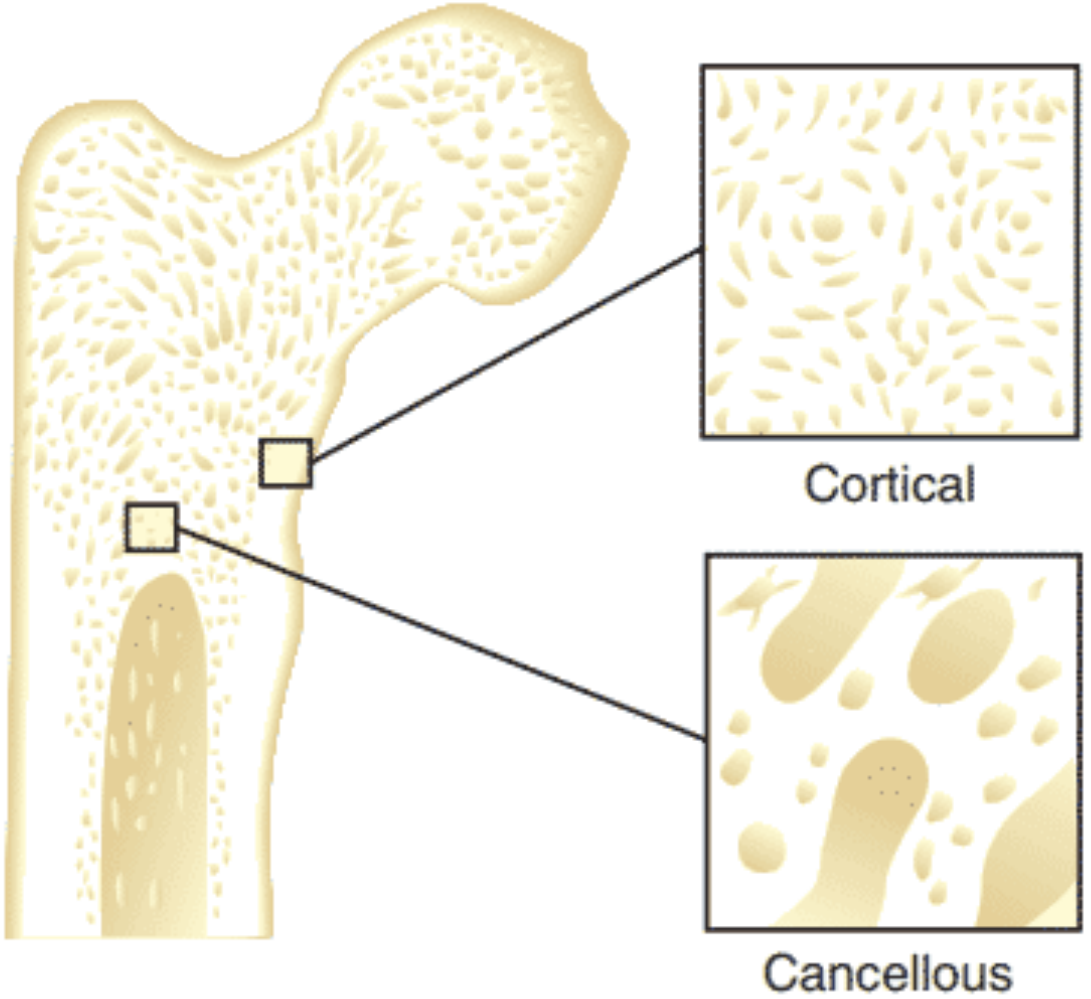
# TYPES OF BONE



| WOVEN BONE (IMMATURE BONE) | LAMELLAR BONE (MATURE BONE) |
| --- | --- |
| COLLAGEN FIBRES ARE ALIGNED RANDOMLY | CORTICAL AND CANCELLOUS BONE |
| NO LAMELLAE | STRESS ORIENTED COLLAGEN FIBER ARRANGEMENT |
| WEAK AND MORE FLEXIBLE | LAMELLAE ARRANGEMENT |
| ISOTROPHIC | |
| EG<br>EMRYONIC AND NEONATAL SKELETON<br>METAPHYSIS OF GROWING BONE<br>FRACTURE CALLUS<br>PATHOLOGICAL – TUMOR, PAGET'S DISEASE, OI | |

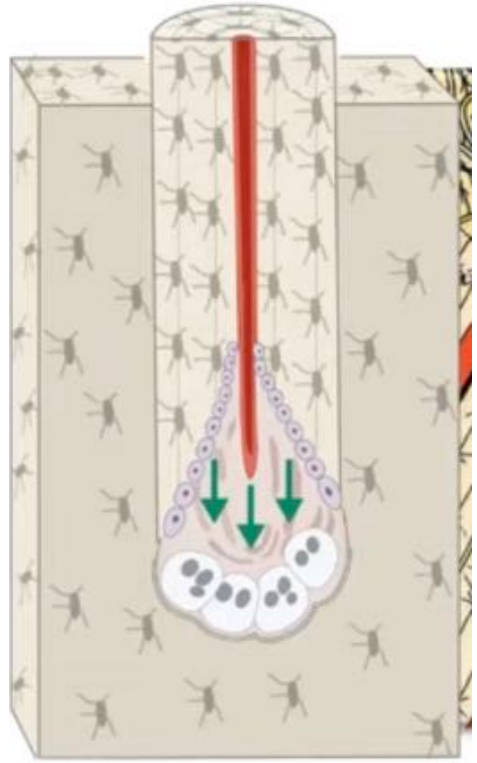
# TYPES OF BONE



# CORTICAL BONE

- MADE UP OF OSTEONS
- CONTINUALLY REMODELLED BY CUTTING CONES
- **POROSITY OF CORTICAL BONES** MOSTLY FALLS IN THE RANGE OF **5-10%**



# CORTICAL BONE

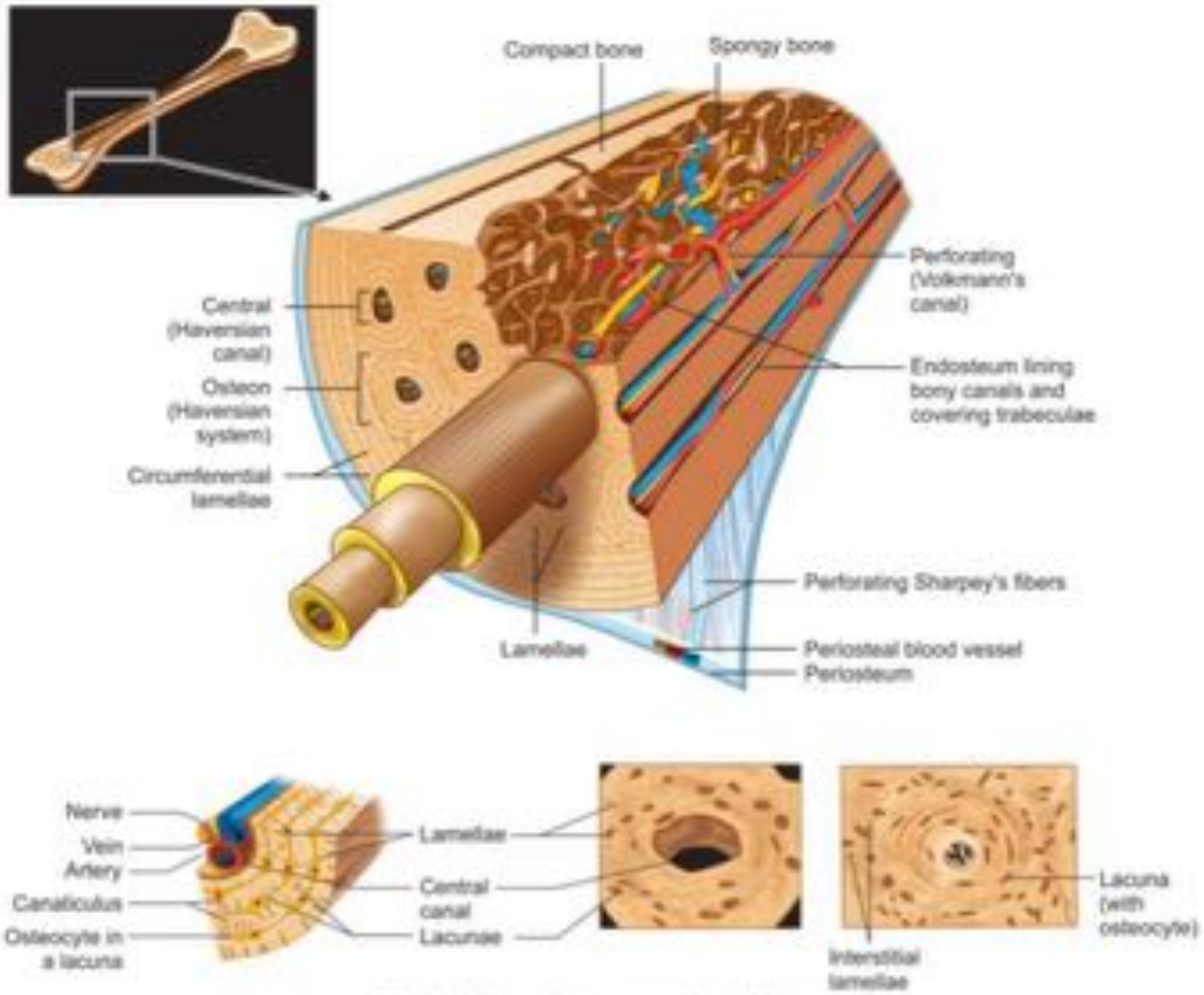



Fig. 2.3: Microscopic structure of cortical bone

# CANCELLOUS BONE

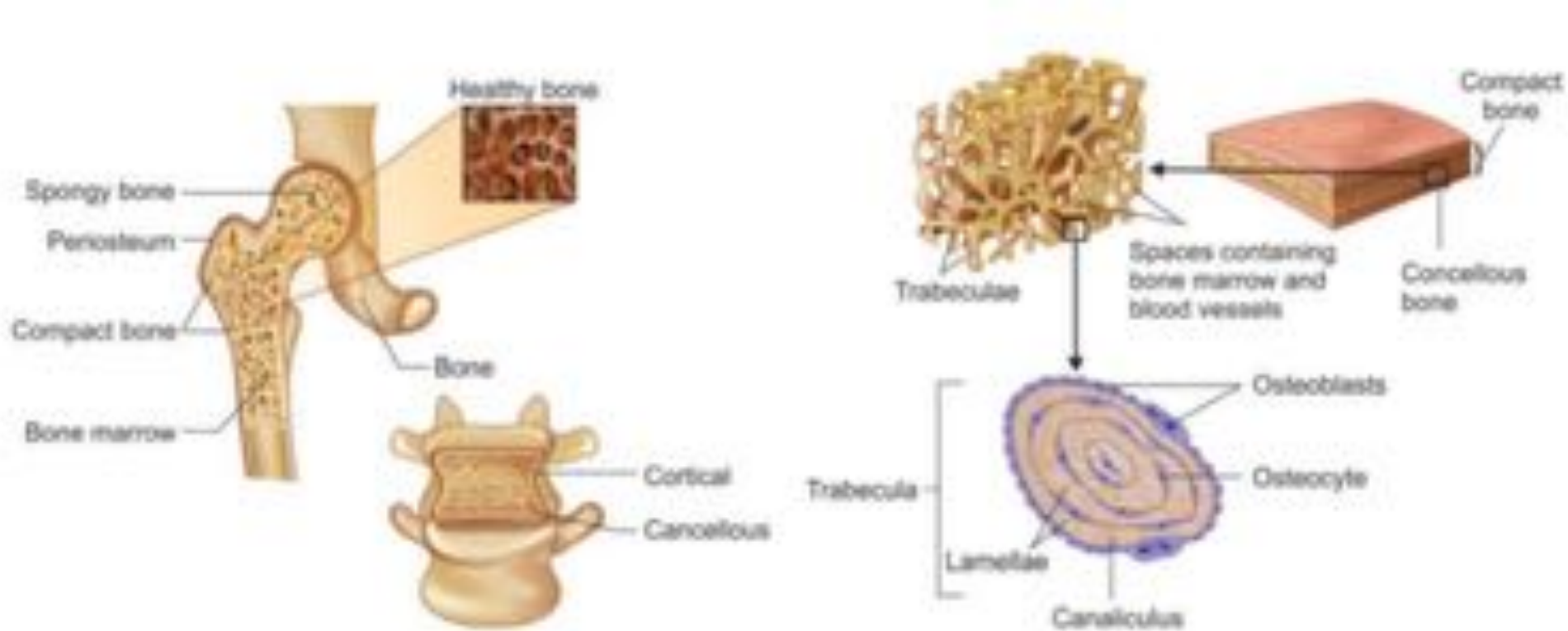



Fig. 2.4: Microscopic structure of cancellous bone

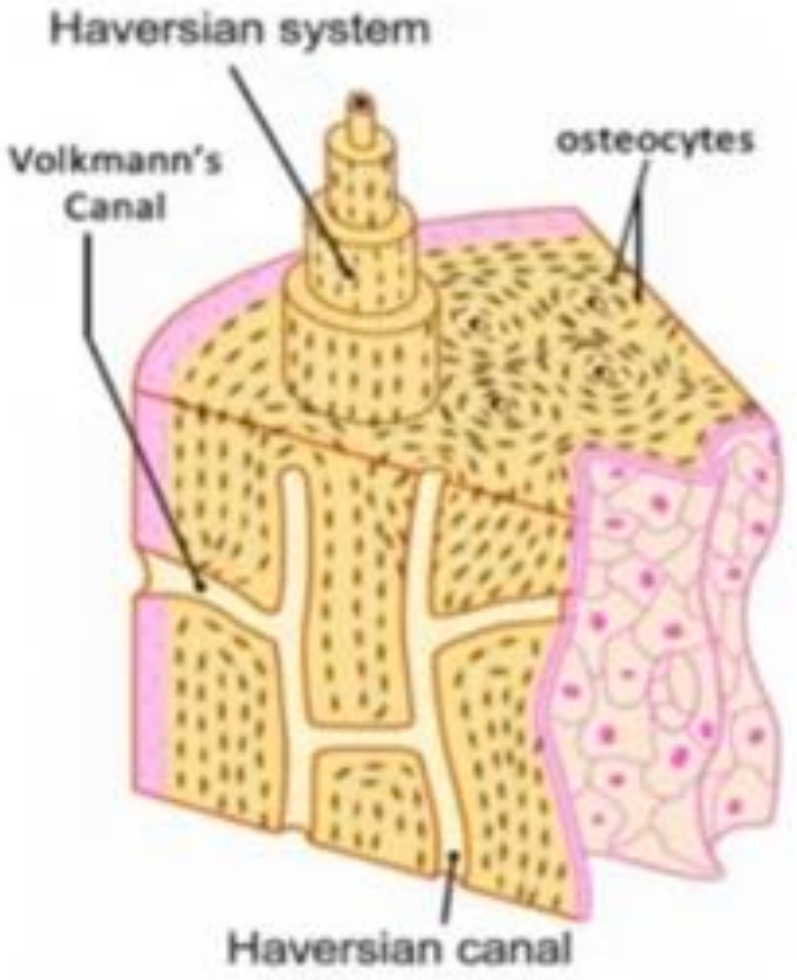
# CANCELLOUS BONE

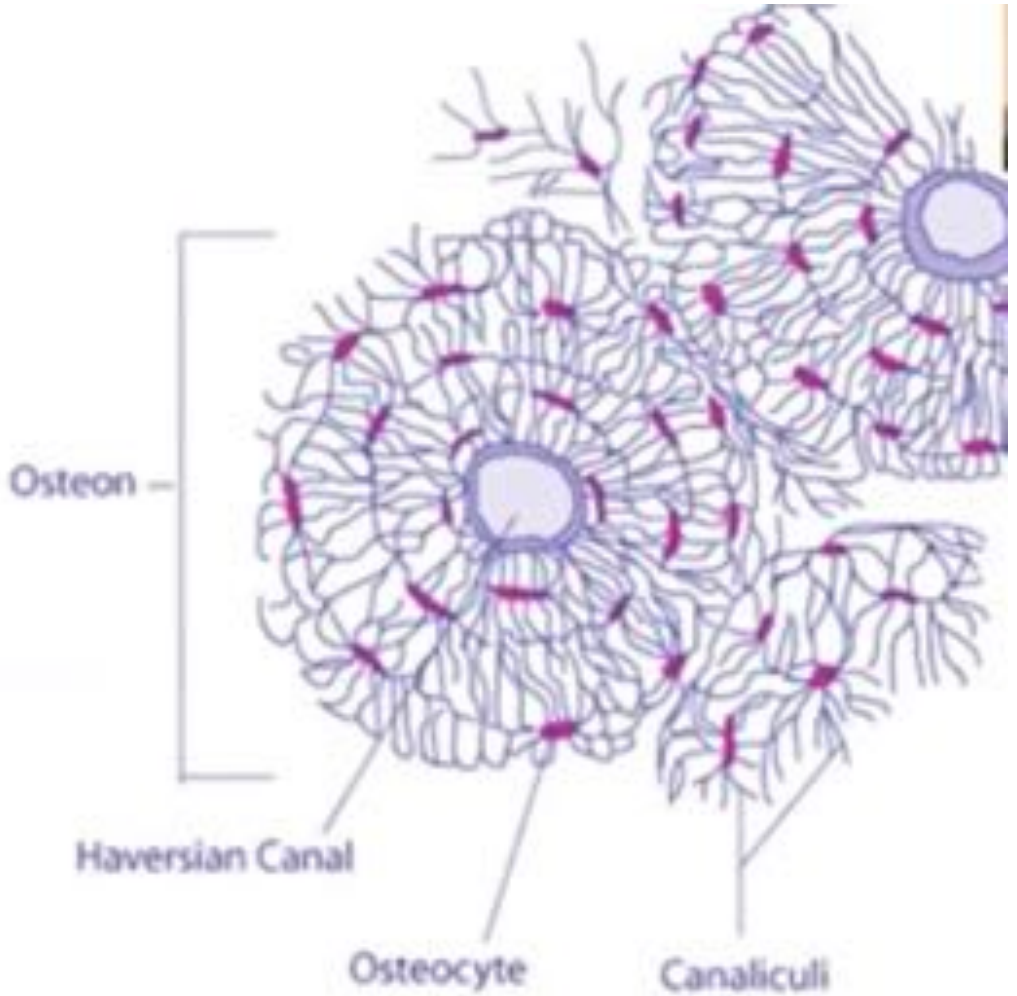
- CANCELLOUS BONE **METABOLIC TURNOVER IS 8 TIMES HIGHER** THAN CORTICAL BONE

- **POROSITY OF CANCELLOUS BONES** RANGE FROM **75-95%**

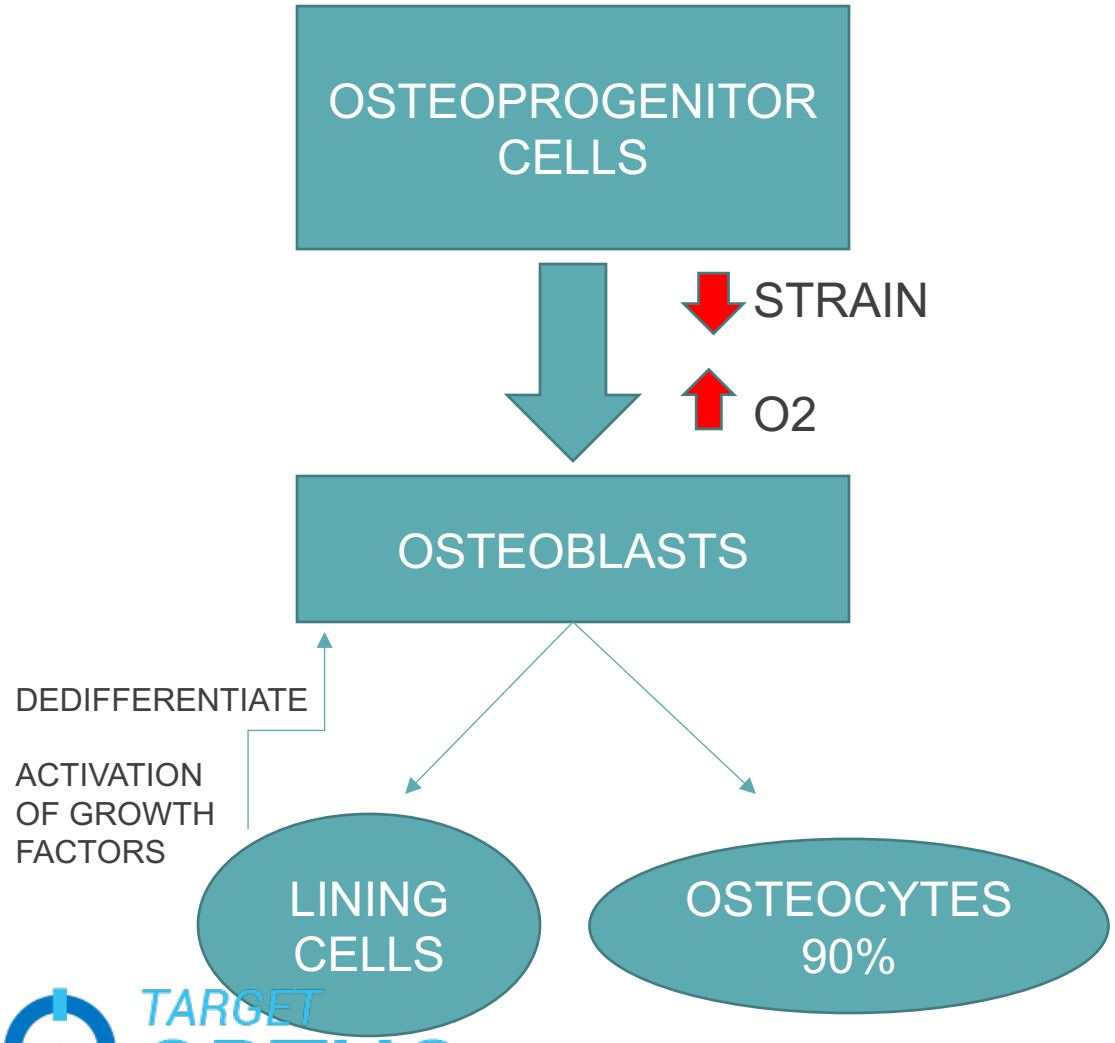
- VERTEBRAE HAVE MAXIMUM RATIO OF CANCELLOUS BONE COMPARED TO ANY OTHER BONE IN THE BODY (CANCELLOUS TO CORTICAL BONE RATIO IS 75:25)

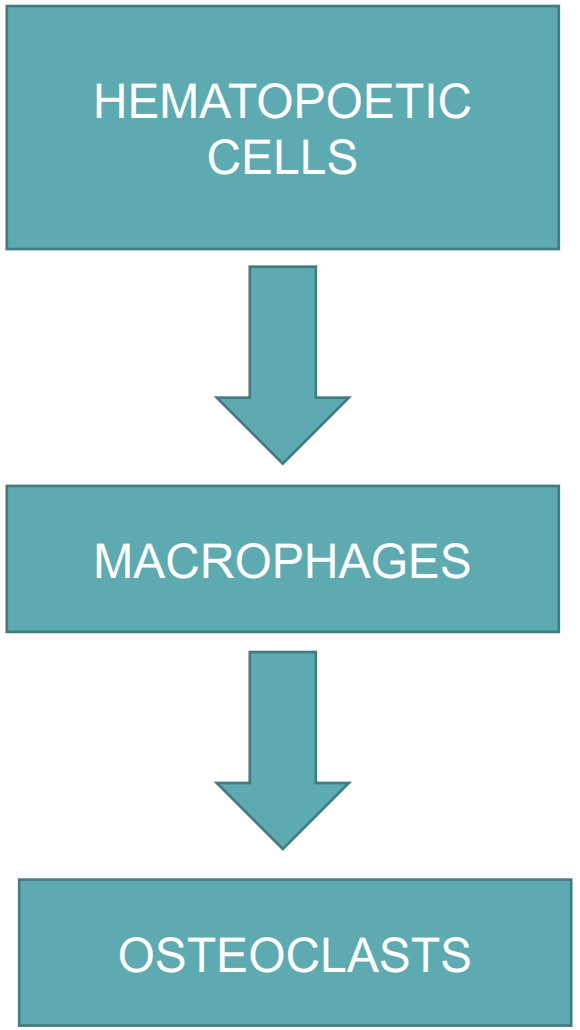
# HAVERSIAN SYSTEM AND VOLKMAN'S CANAL



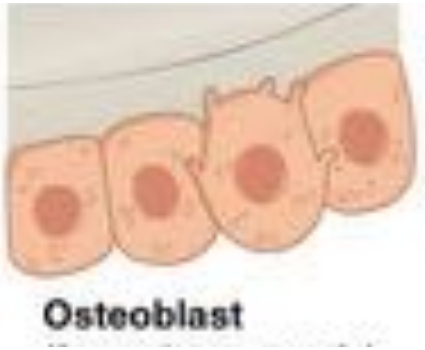



# CELL BIOLOGY



# OSTEOBLASTS




- CUBOID CELLS
- ALIGNED IN LAYERS ALONG IMMATURE OSTOIDS
- DERIVED FROM UNDIFFERENTIATED MESENCHYMAL STEM CELLS

ENDOPLASMIC RETINACULUM
GOLGI APPARATUS
MITOCHONDRIA

THAN OTHER BONE CELLS

CENTER FOR SYNTHESIS AND SECREATION OF MATRIX FOR BONE FORMATION

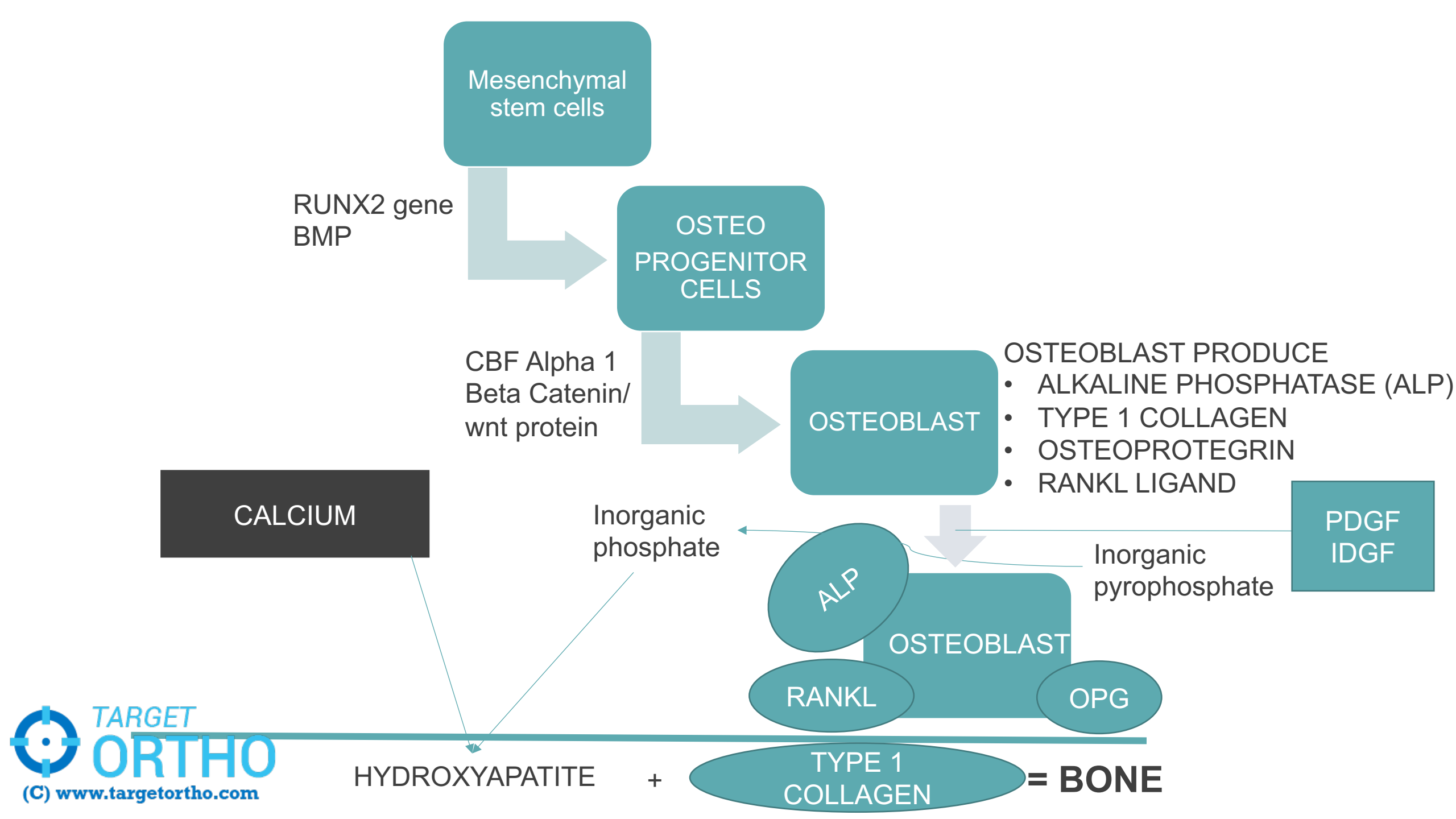
# Which one of the following is not involved in Osteoblast differentiation ?

A) Core bonding protein

B) Platelet Derived growth factor

C) Cathepsin K

D) Beta Catenin


Mesenchymal stem cells
RUNX2 gene
BMP
OSTEO PROGENITOR CELLS
CBF Alpha 1
Beta Catenin/
wnt protein
OSTEOBLAST
OSTEOBLAST PRODUCE
• ALKALINE PHOSPHATASE (ALP)
• TYPE 1 COLLAGEN
• OSTEOPROTEGRIN
• RANKL LIGAND
CALCIUM
Inorganic phosphate
PDGF
IDGF
Inorganic pyrophosphate
ALP
OSTEOBLAST
RANKL
OPG
TARGET ORTHO
(C) www.targetortho.com
HYDROXYAPATITE
+
TYPE 1 COLLAGEN
= BONE

# OSTEOBLAST DIFFERENTIATION

- BONE MORPHOGENIC PROTIEN
- CORE BINDING PROTIEN FACTOR ALPHA 1
- BETA CATENIN / WNT PROTIEN
- PLATELET DERIVED GROWTH FACTOR
- INSULIN LIKE GROWTH FACTOR

# WNT PROTIEN

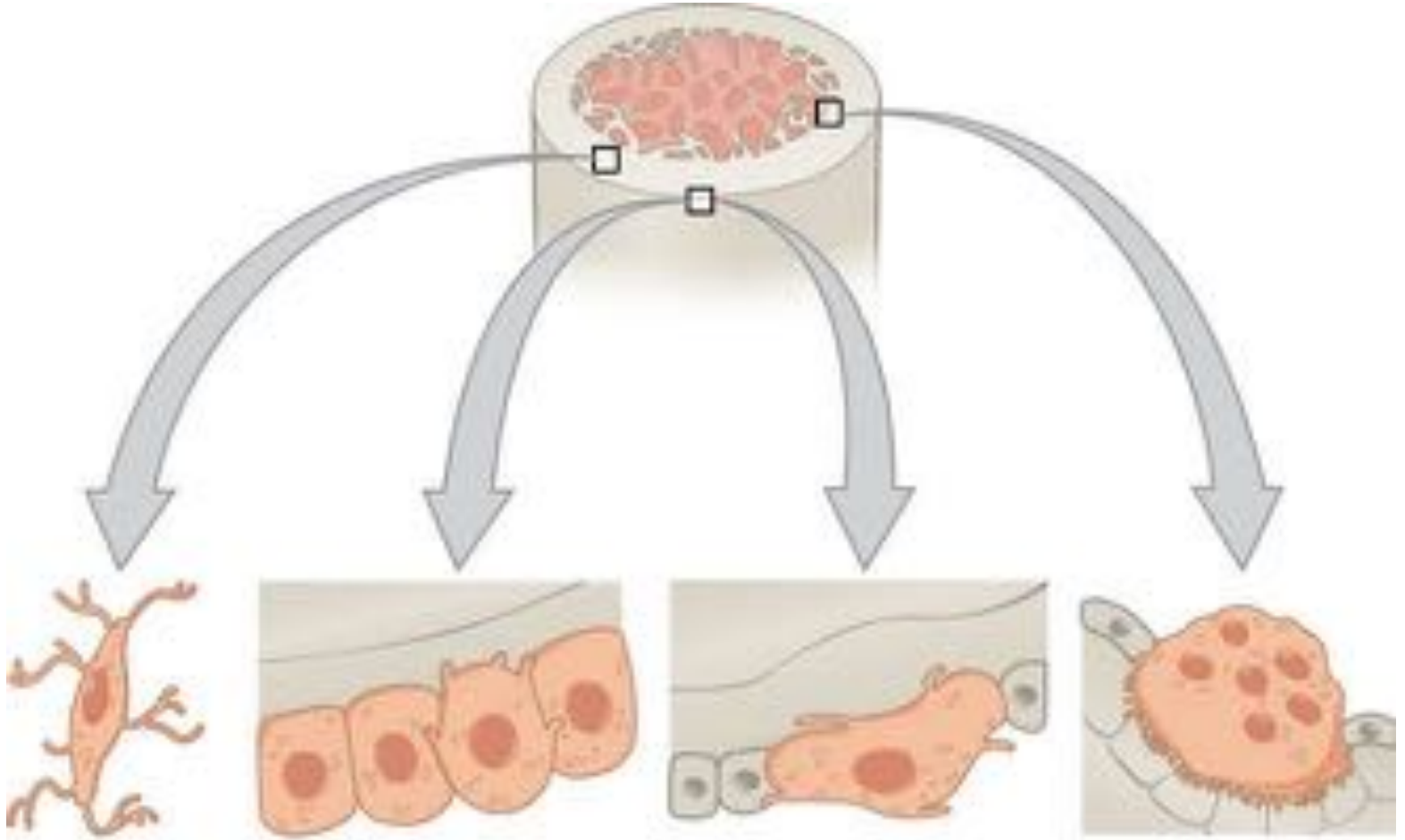
- Promote osteoblast survival and proliferation

Excess wnt expression – **HIGH BONE MASS**

Deficient wnt – **OSTEOPENIA**

# ANTISEPTICS TOXIC TO CULTURED OSTEOBLASTS

- HYDROGEN PEROXIDE
- POVIDONE IODINE (BETADINE)
- BACITRACIN



# ULTIMATE FATE OF OSTEOBLAST

- INACTIVE BONE LINING CELLS
- OSTEOCYTES
- APOPTOSIS

# OSTEOCYTES


- OSTEOBLAST TRAPPED IN CALCIFIED BONE MATRIX
- 90% CELLS IN MATURE SKELETON
- UNLIKE OSTEOBLAST, OSTEOCLAST- THEY HAVE HIGH NUCLEUS:CYTOPLASM RATIO
- INTERCELLULAR CONNECTION BY CANALICULI

# OSTEOCYTES

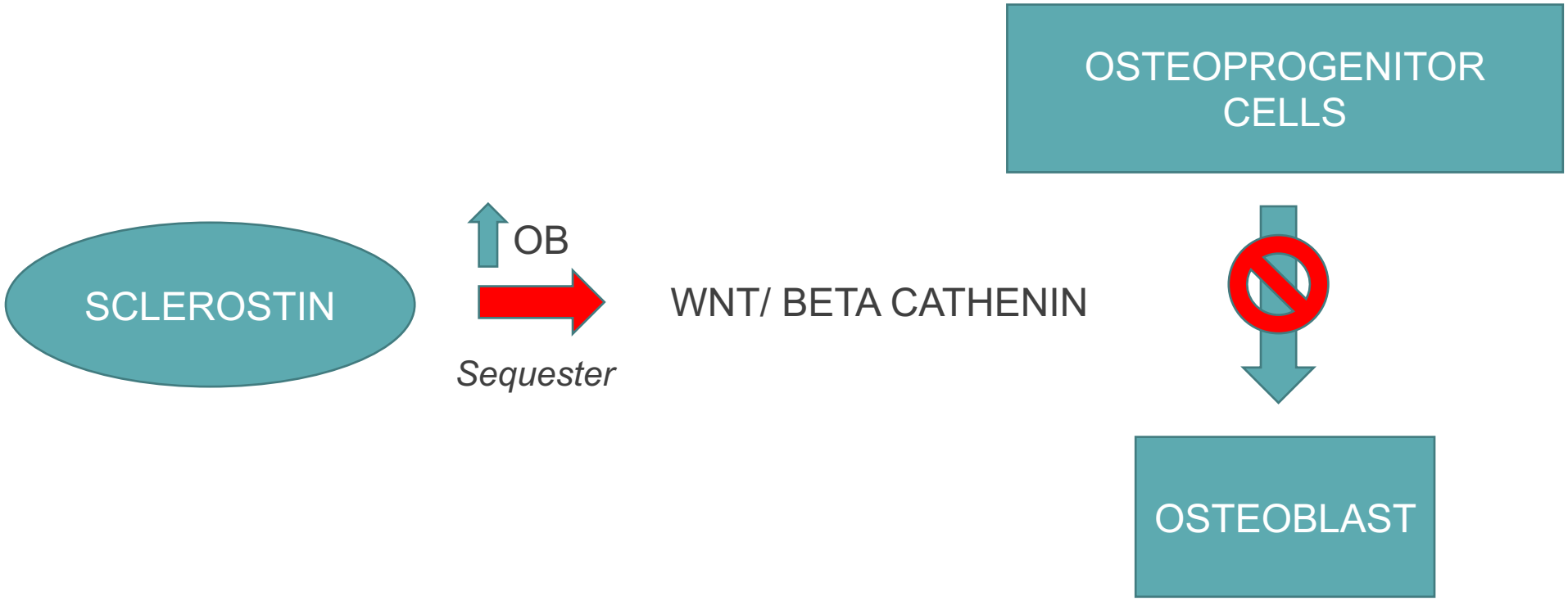

FUNCTIONS

1. MAINTAINS THE BONE

2. IMPORTANT FOR CONTROL OF EXTRACELLULAR CALCIUM AND PHOSPHORUS CONCENTRATION

3. DIRECTLY STIMULATED BY CALCITONIN AND INHIBITED BY PTH

# OSTEOCYTE – OSTEOBLAST




- Negative feedback on Osteoblast bone deposition

# BONE LINING CELLS

- LYING ON THE SURFACE OF THE BONE
- INACTIVE FORM OF OSTEOBLASTS
- MAY REACTIVATE DURING NEW BONE FORMATION

# OSTEOCLAST

- ARISES FROM HAEMATOPOETIC MACROPHAGE AND MONOCYTES STEM CELL LINE

- LARGE MULTINUCLEATED GIANT CELL

HEMATOPOIETIC CELLS

MACROPHAGE LINEAGE

OSTEOCLAST

OPG

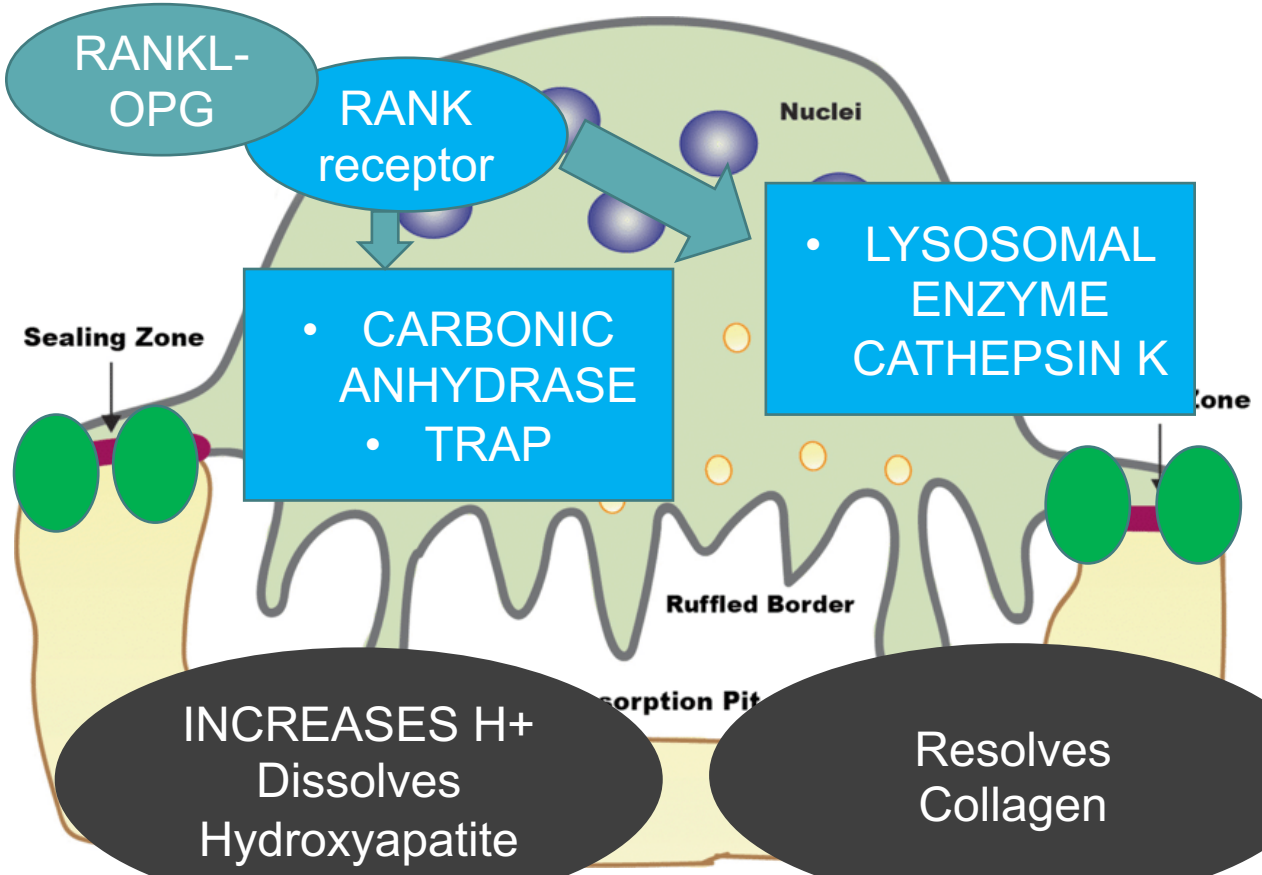
OSTEOBLAST

RANKL

OB-OC COUPLING

RANKL-OPG

RANK receptor



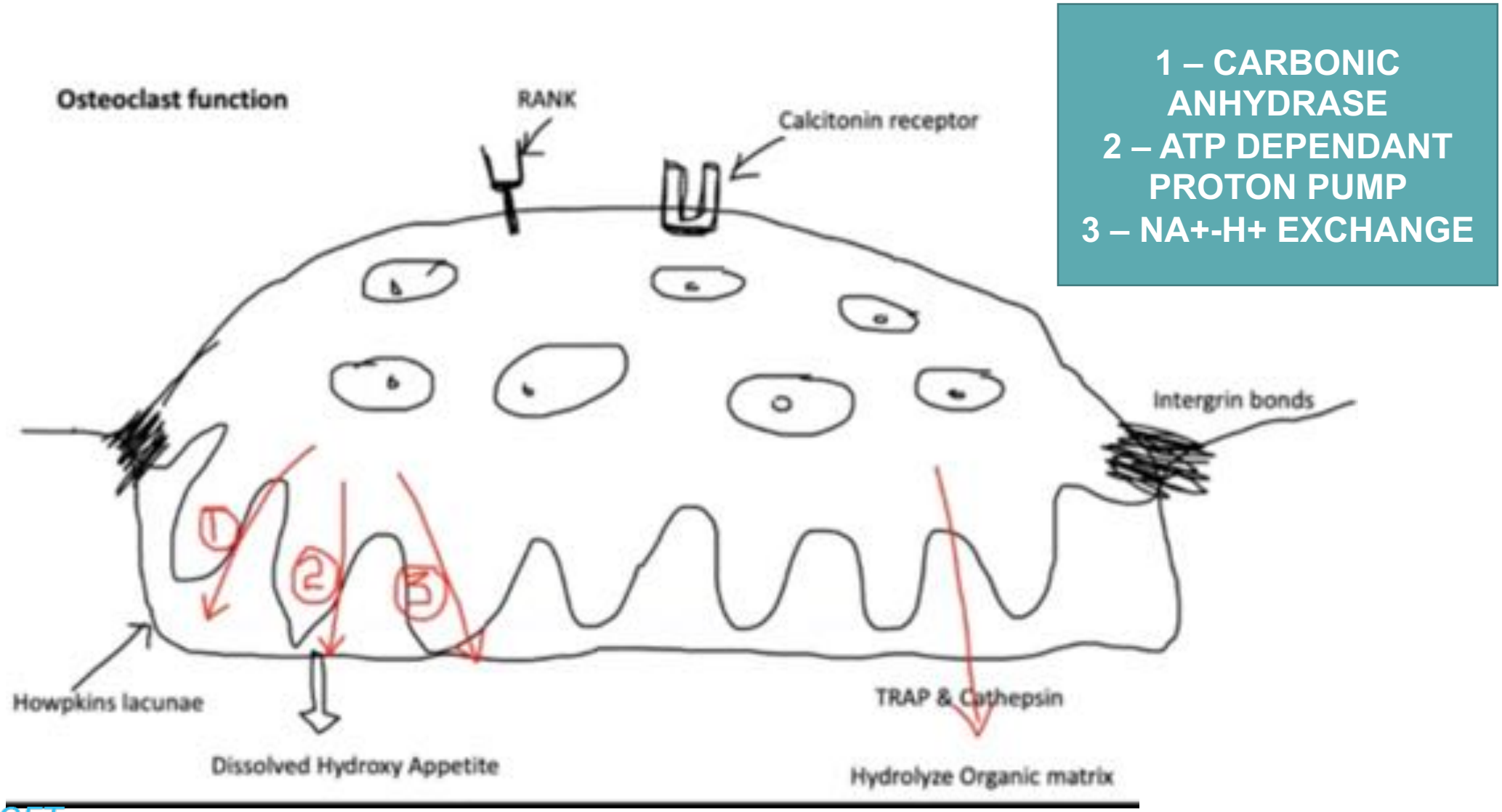

- CARBONIC ANHYDRASE
- TRAP

- LYSOSOMAL ENZYME CATHEPSIN K

Anchoring Protein **INTEGRIN**

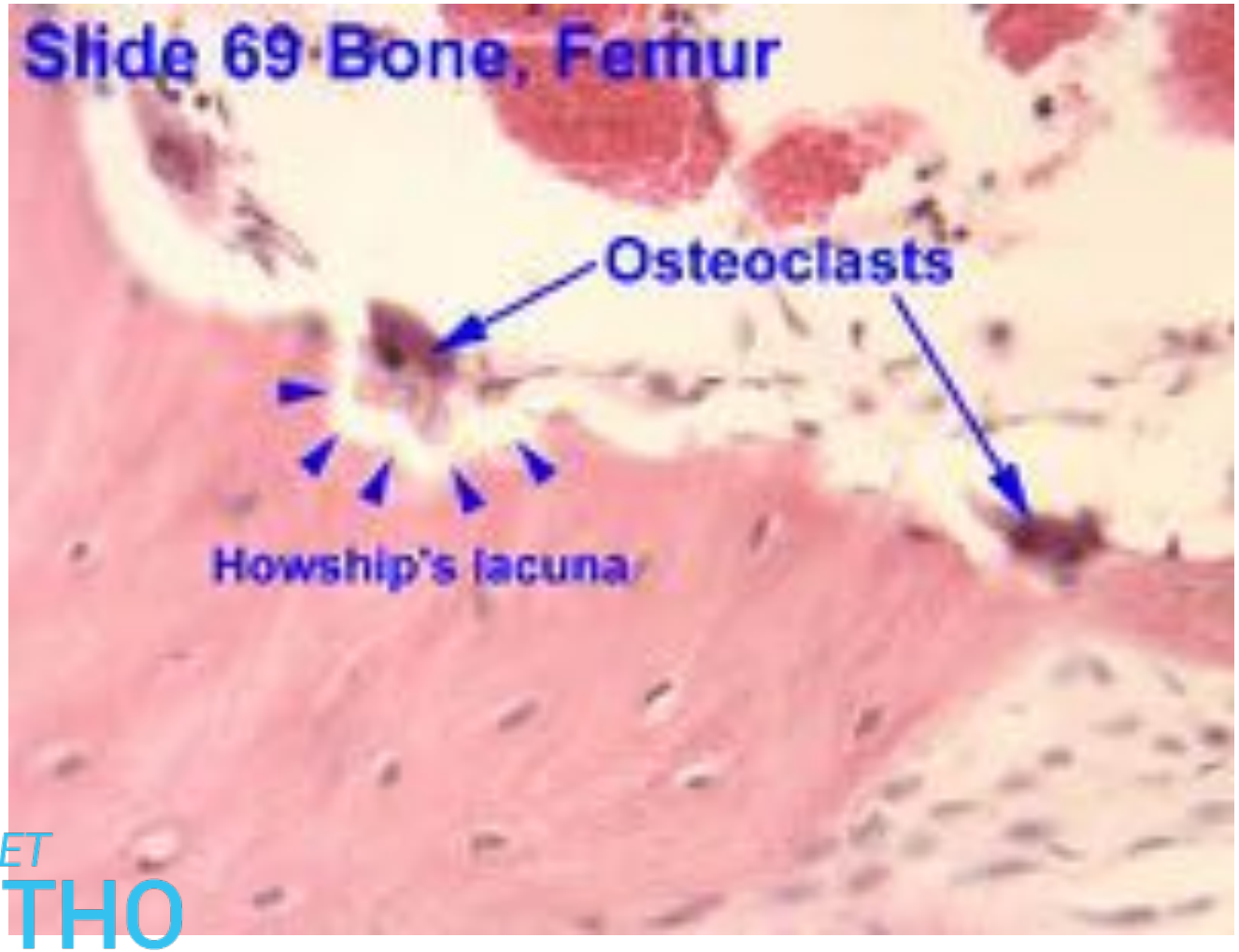
INCREASES H+ Dissolves Hydroxyapatite

Resolves Collagen




1 – CARBONIC ANHYDRASE
2 – ATP DEPENDANT PROTON PUMP
3 – NA+-H+ EXCHANGE

# HOWSHIP'S LACUNAE

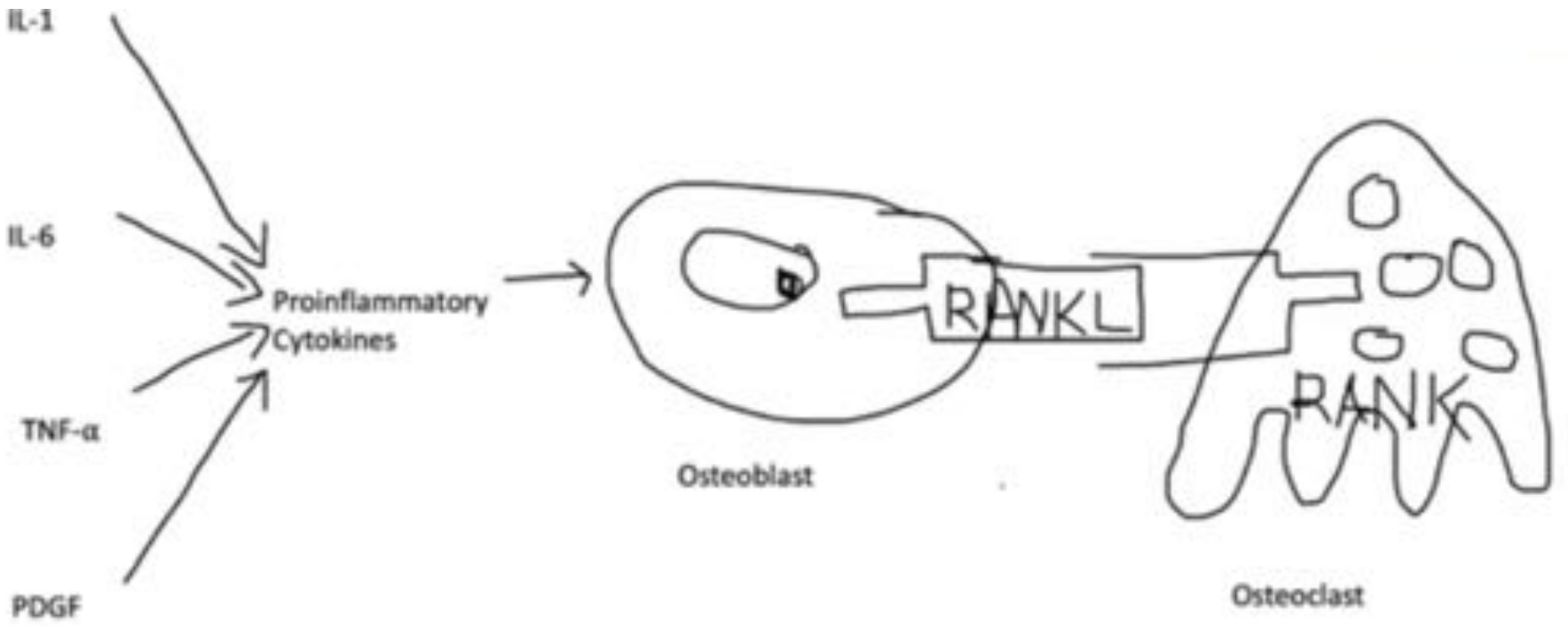



FORMED BY RUFFLED BORDERS

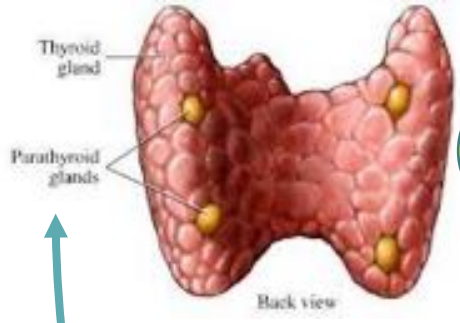
OSTEOCLASTIC ACTIVITY TAKES PLACE HERE

# BONE CYCLE

# IN BONE TURNOVER



# METABOLISM




PARATHYROID HORMONE

+

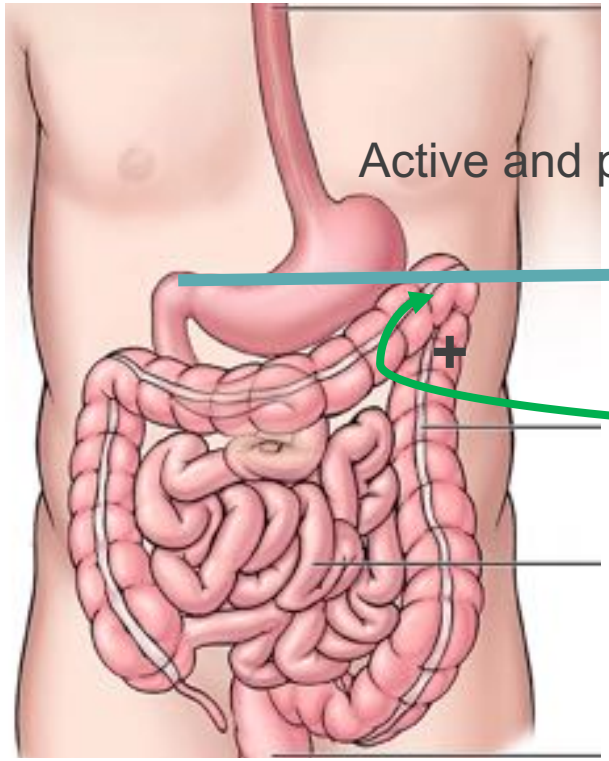
BLOOD

Ca2+

Ca2+

+

INACTIVE VITAMIN D



Active and passive

Ca2+ PO4

+

VIT D

PTH

PTH

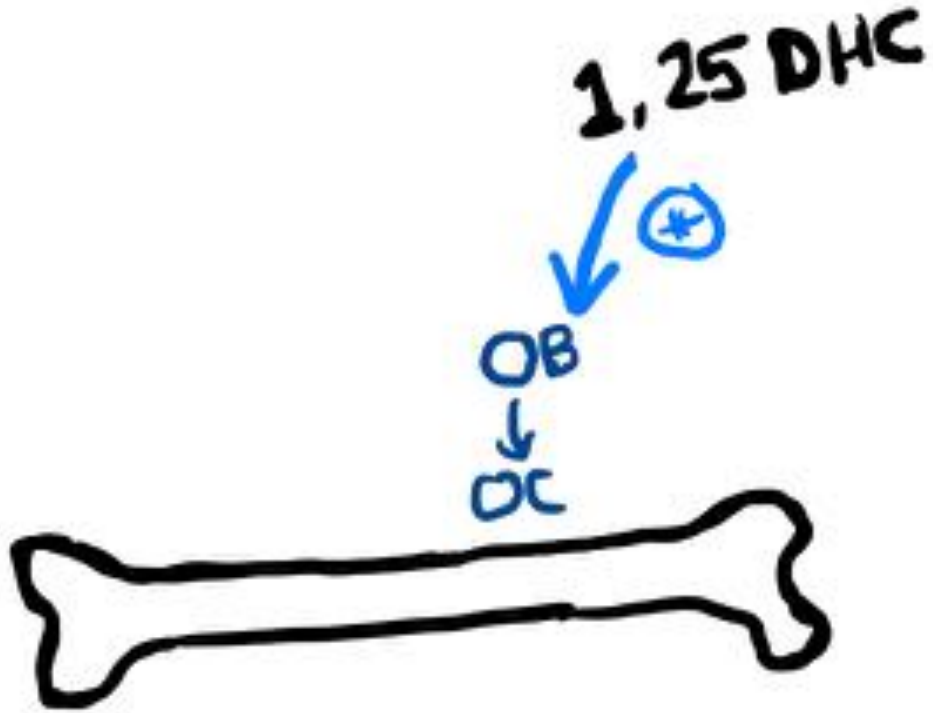
ACTIVE VITAMIN D

Osteoclastic activity

Ca2+ & PO4

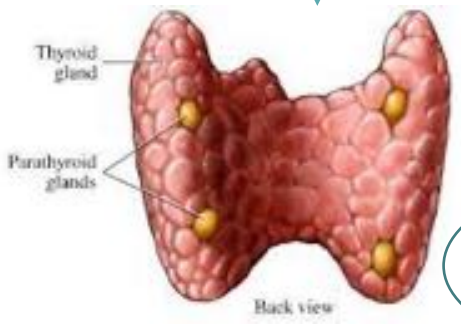
Ca2+ reabsorption

Urine
Low Ca, High PO4



# Calcitonin

Blood Calcium

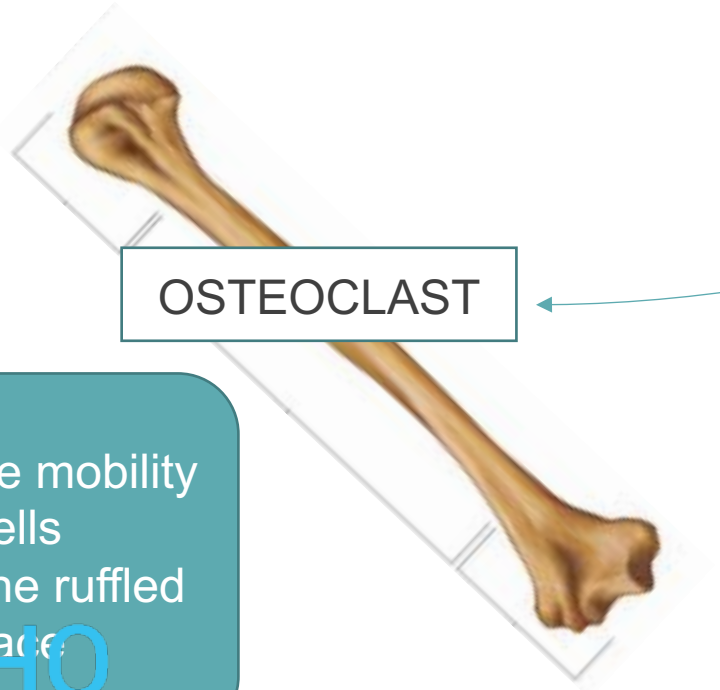



THYROID Parafollicular Cells

CALCITONIN

INHIBIT

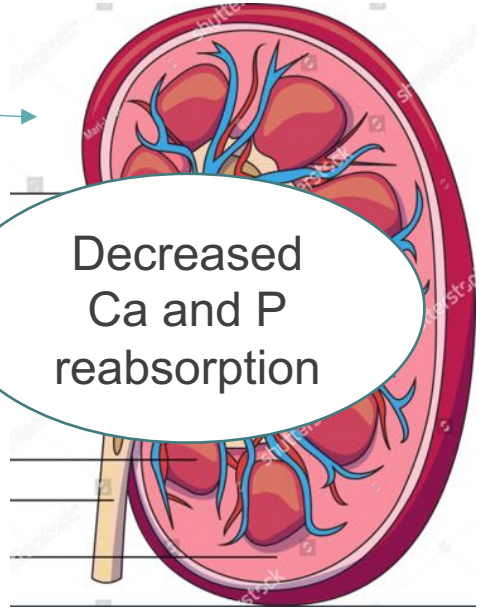
OSTEOCLAST



- Reduce the mobility of cells
- Flattens the ruffled surface

Normal Blood Calcium

Decreased Ca and P reabsorption



# Bones grow both in length and breadth and gain strength as they begin to ossify. The increase in WIDTH of the bone is what type of growth?

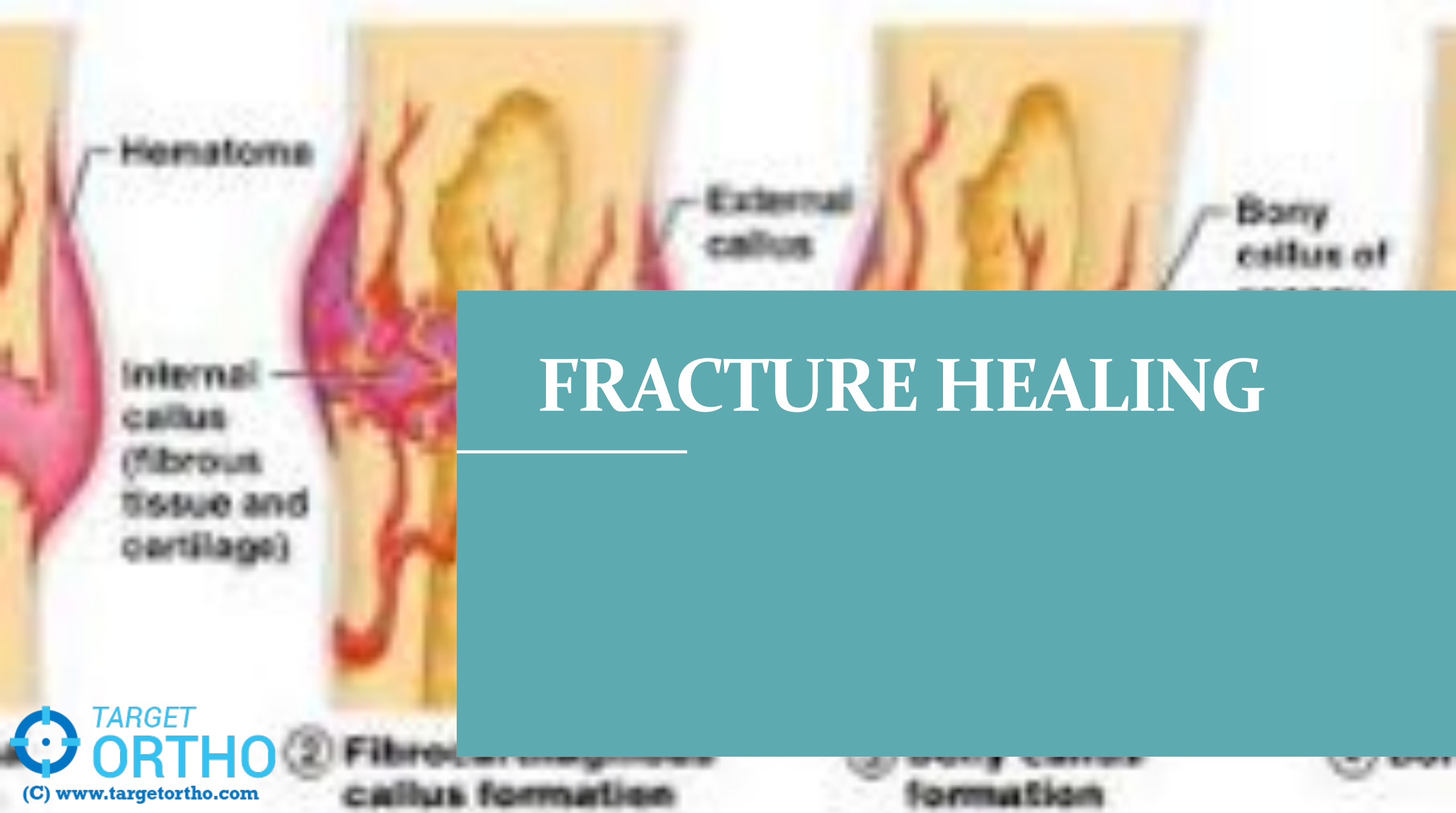
A. Enchondral growth
B. Appositional growth
C. Longitudinal growth
D. Concentric growth

# Growth

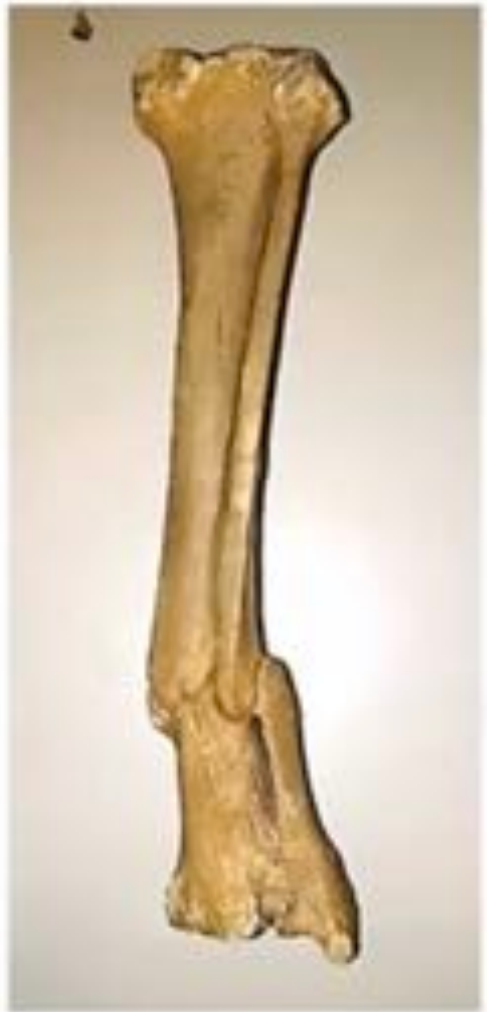
| Type of Ossification | Mechanism | Examples of Normal Mechanisms | Examples of Diseases with Abnormal Ossification |
|---|---|---|---|
| Enchondral | Bone replaces a cartilage model | Embryonic formation of long bones<br>Longitudinal growth (physis)<br>Fracture callus<br>Bone formed with the use of demineralized bone matrix | Achondroplasia |
| Intramembranous | Aggregates of undifferentiated mesenchymal cells differentiate into osteoblasts, which form bone | Embryonic flat bone formation<br>Bone formation during distraction osteogenesis<br>Blastema bone | Cleidocranial dysostosis |
| Appositional | Osteoblasts lay down new bone on existing bone | Periosteal bone enlargement (width)<br>The bone formation phase of bone remodeling | Paget disease of bone<br>Infantile hyperostosis (Caffey disease)<br>Melorheostosis |

Epiphysis

Endosteum, periosteum

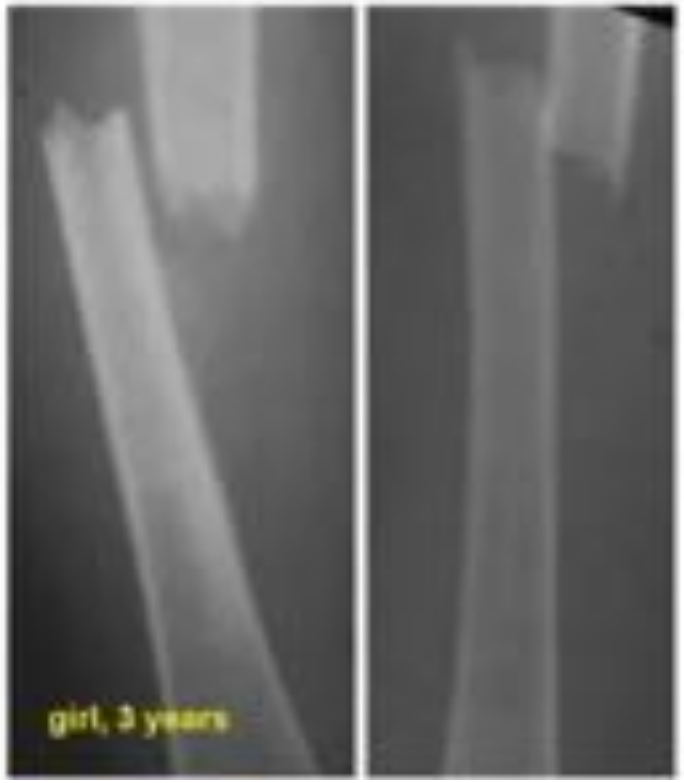


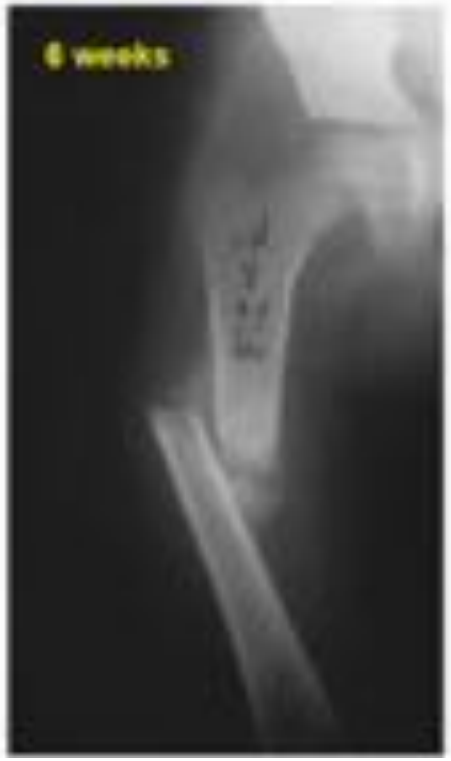

# FRACTURE HEALING

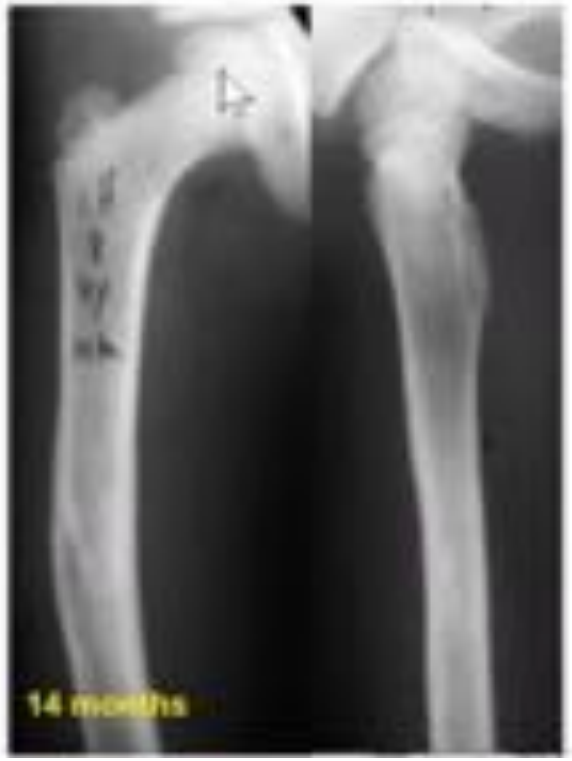


**BONES HEAL THEMSELVES !**

# BONE HEALING !

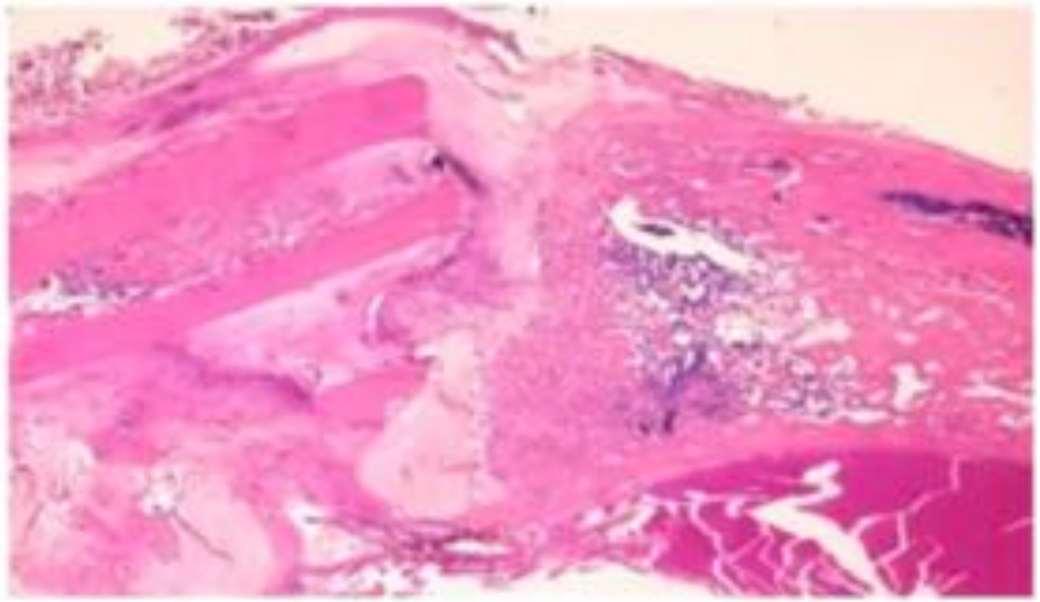


# WHAT DOES NATURE DO WITH A BROKEN BONE ?

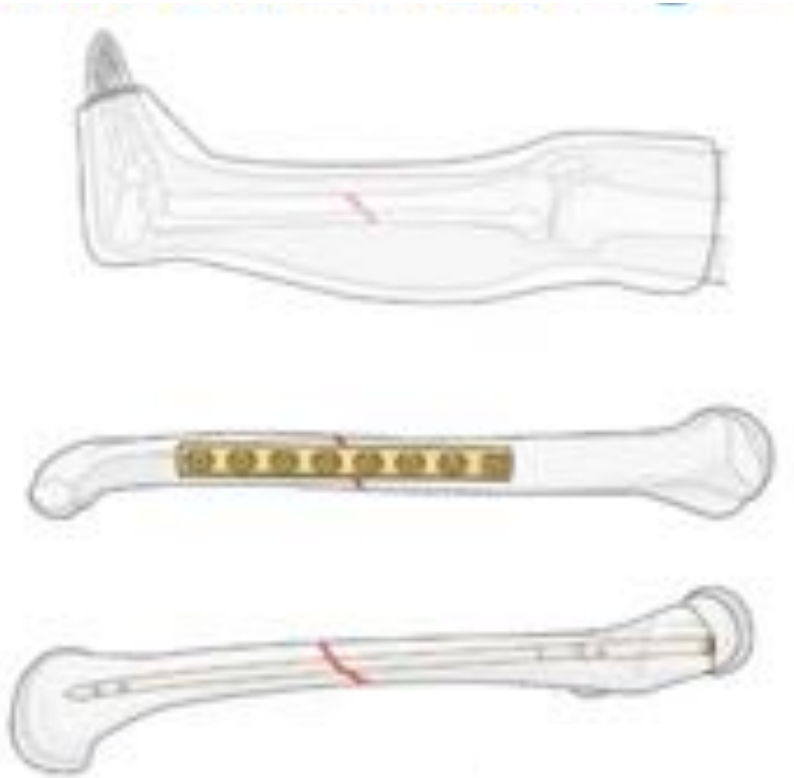
IT HEALS BY CALLUS FORMATION



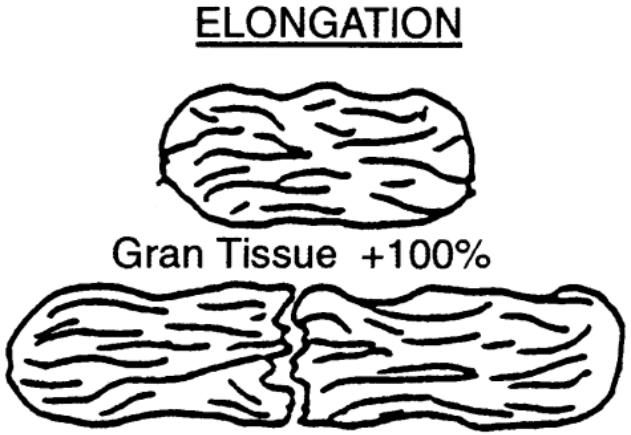
IF THERE IS MOTION BETWEEN THE FRAGMENTS

# MECHANO BIOLOGY OF FRACTURE HEALING

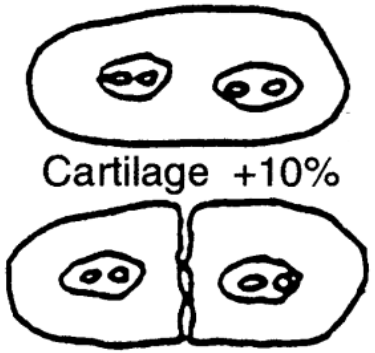
- Strain - deformation of a material under load
- Change in length(ΔI) in comparison to original length(I)
- Strain is inductor of callus formation
- Bony bridging occurs if local strain < than the woven bone can tolerate



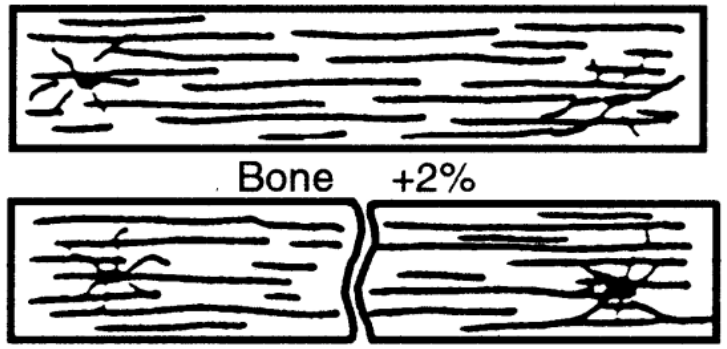
# PERREN STRAIN THEORY




DEPENDS ON MECHANICAL & CHEMICAL ENVIRONMENT




| | |
|---|---|
| STRAIN < 2% | PRIMARY BONE HEALING |
| STRAIN 2-10 % | SECONDARY BONE HEALING - CALLUS |
| STRAIN > 17% | FIBROUS UNION |
| STRAIN UPTO 100% | GRANULATION TISSUE |



# WHICH TYPE OF FRACTURE HEALING OCCURS IN COMPRESSION PLATING ?

A) PRIMARY

B) SECONDARY

C) MIXED

D) INDIRECT HEALING

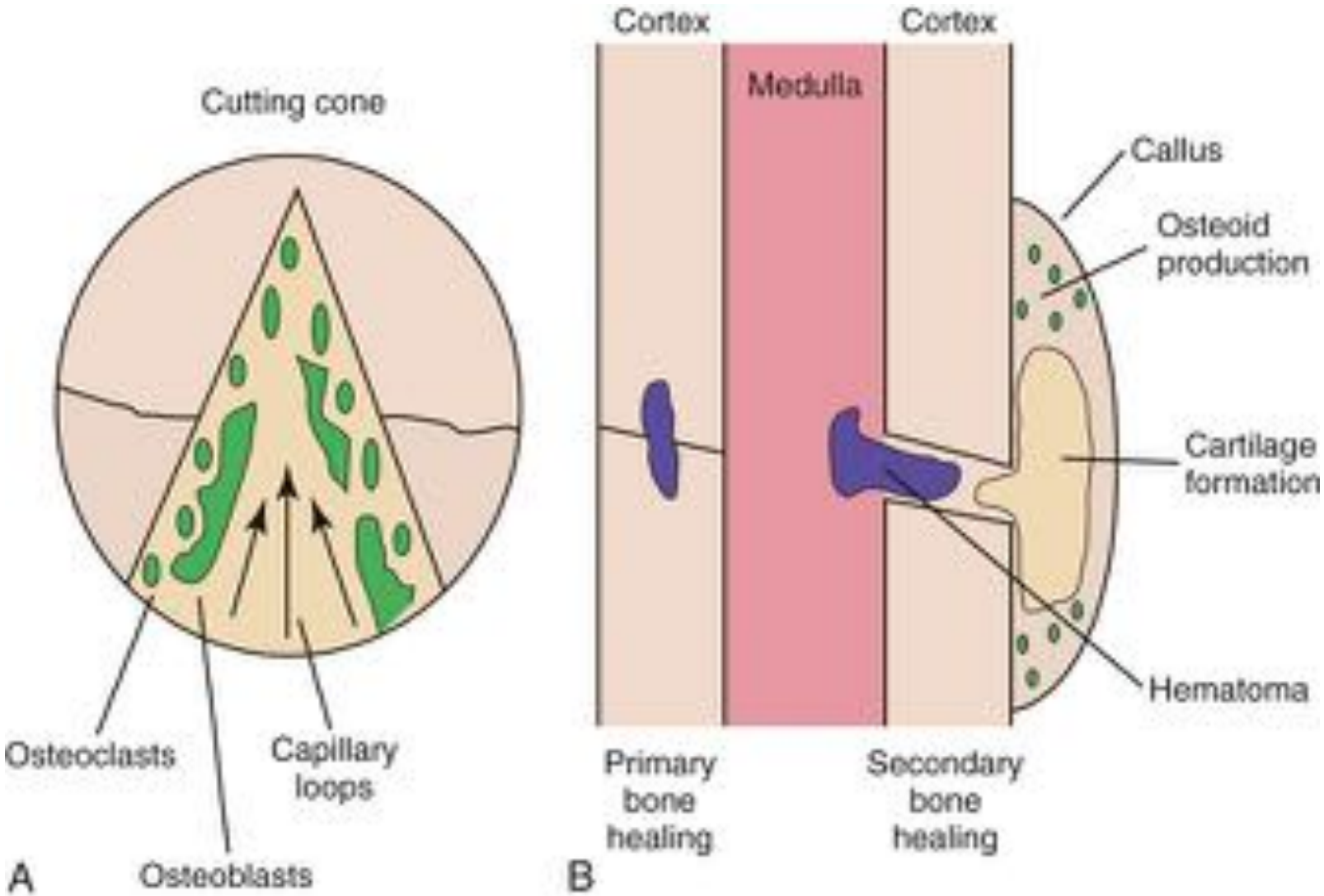
# WHICH TYPE OF FRACTURE HEALING OCCURS IN LOCKED COMPRESSION PLATING ?

A) PRIMARY

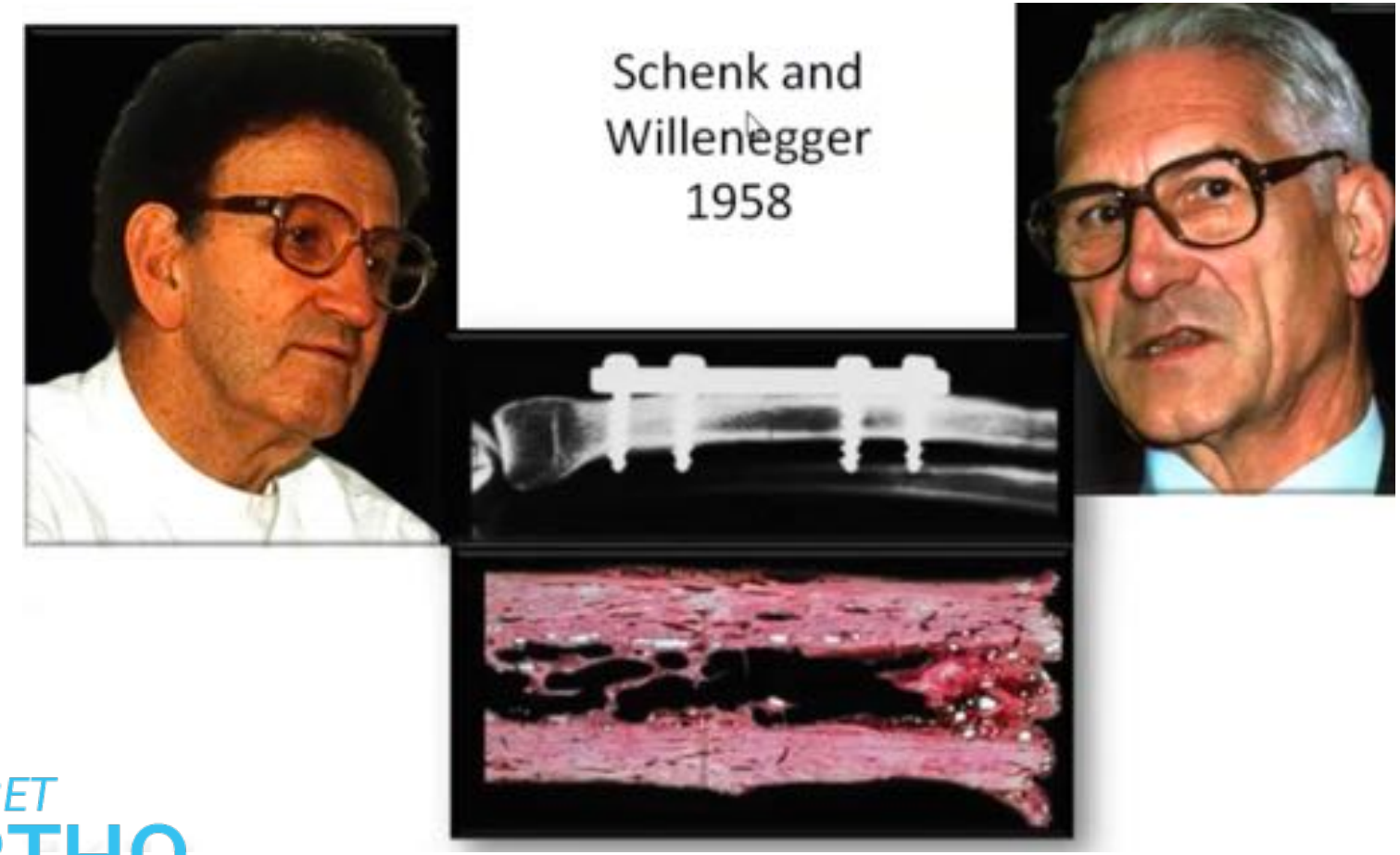
B) SECONDARY

C) MIXED

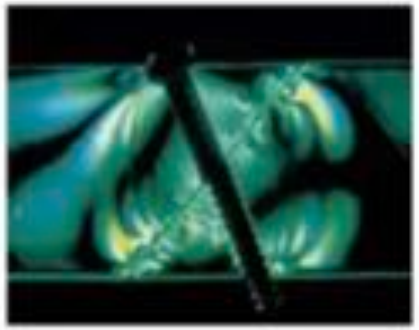
D) CONTACT HEALING



# DIRECT BONE HEALING



# DIRECT / PRIMARY BONE HEALING



Perfect Anatomical Reduction

+

Interfragmentary Compression

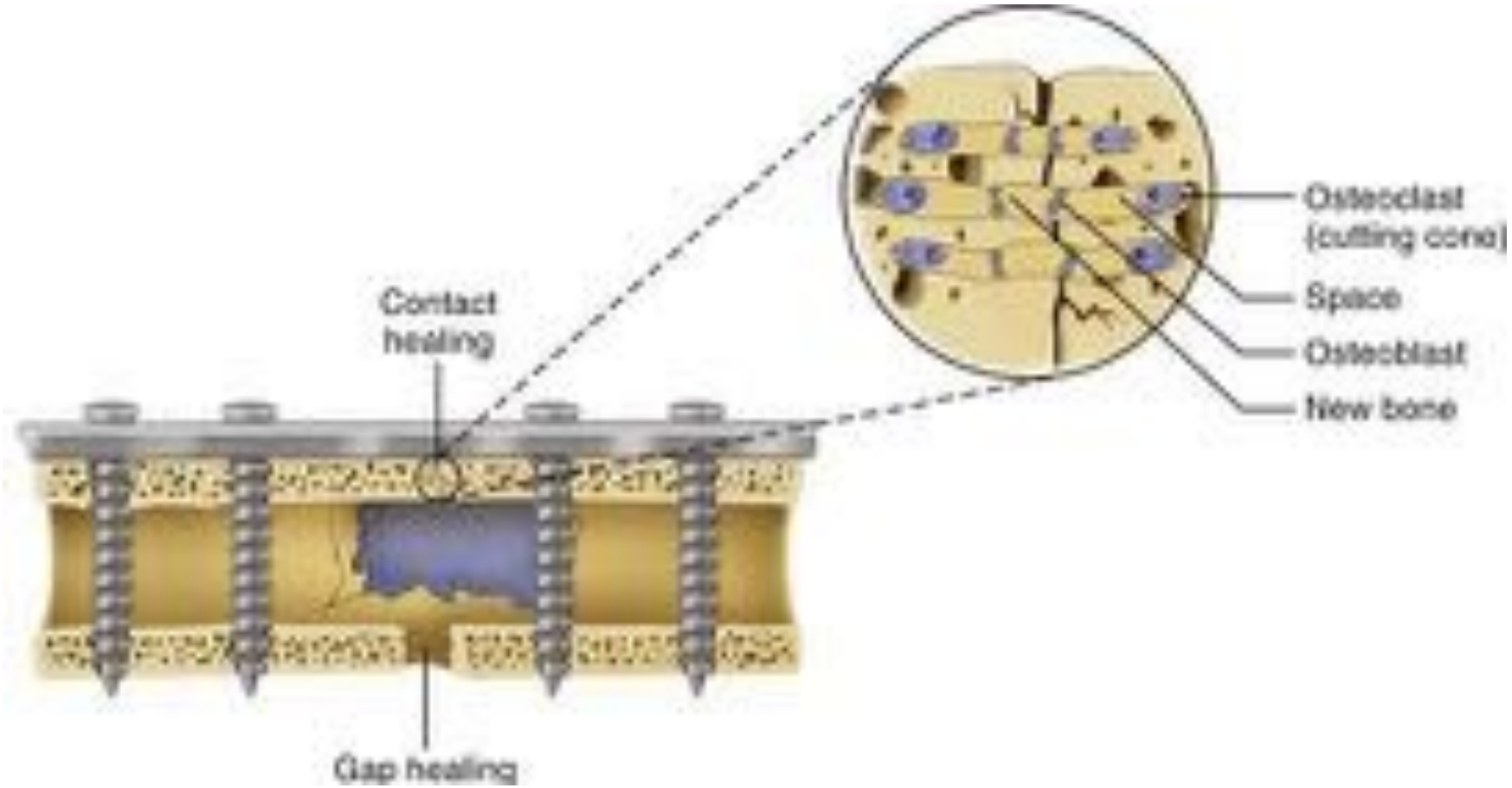
↓

RIGID STABILITY

↓

No stimulus for (peri)osteal

callus formation



# DIRECT / PRIMARY BONE HEALING

- LESS COMMON MODE OF HEALING

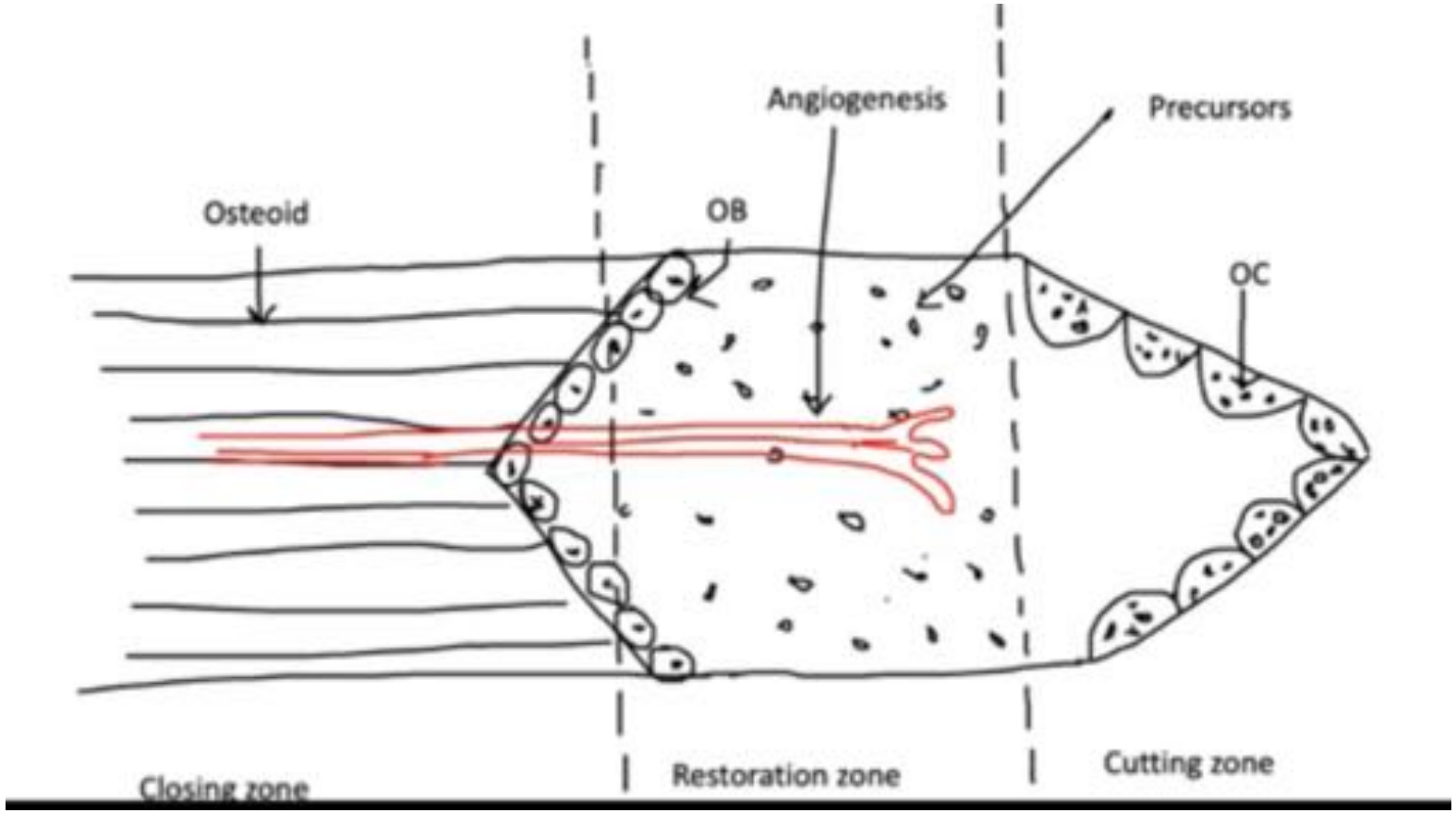
- IT IS A DIRECT ATTEMPT OF BONE TO RESTORE ITS CONTINUITY WITHOUT FORMING FRACTURE CALLUS

EG

**RIGID INTERNAL FIXATION OF FRACTURE (COMPRESSION PLATE)**

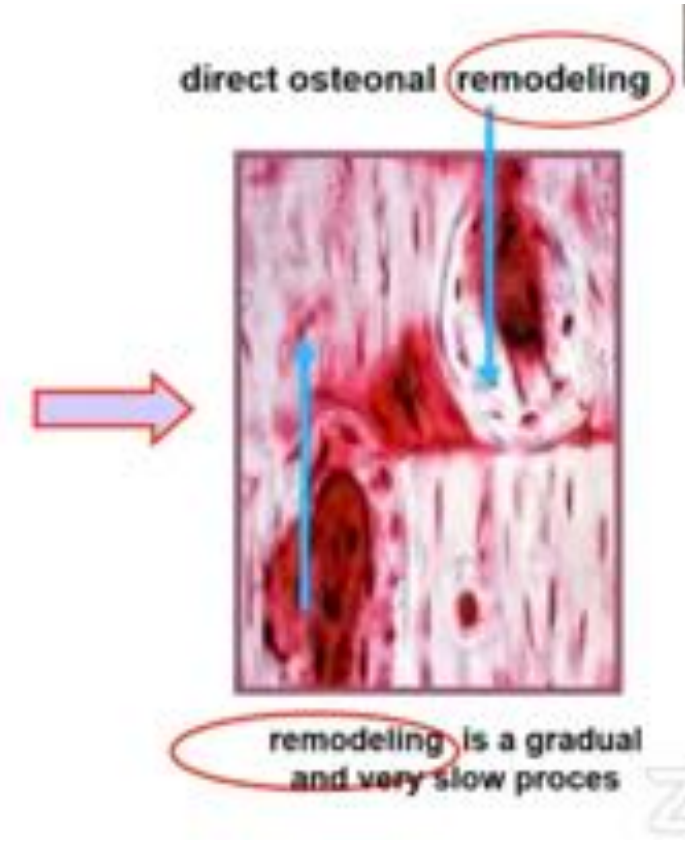
**UNICORTICAL FRACTURE (GREENSTICK FRACTURE)**

# CUTTING CONES

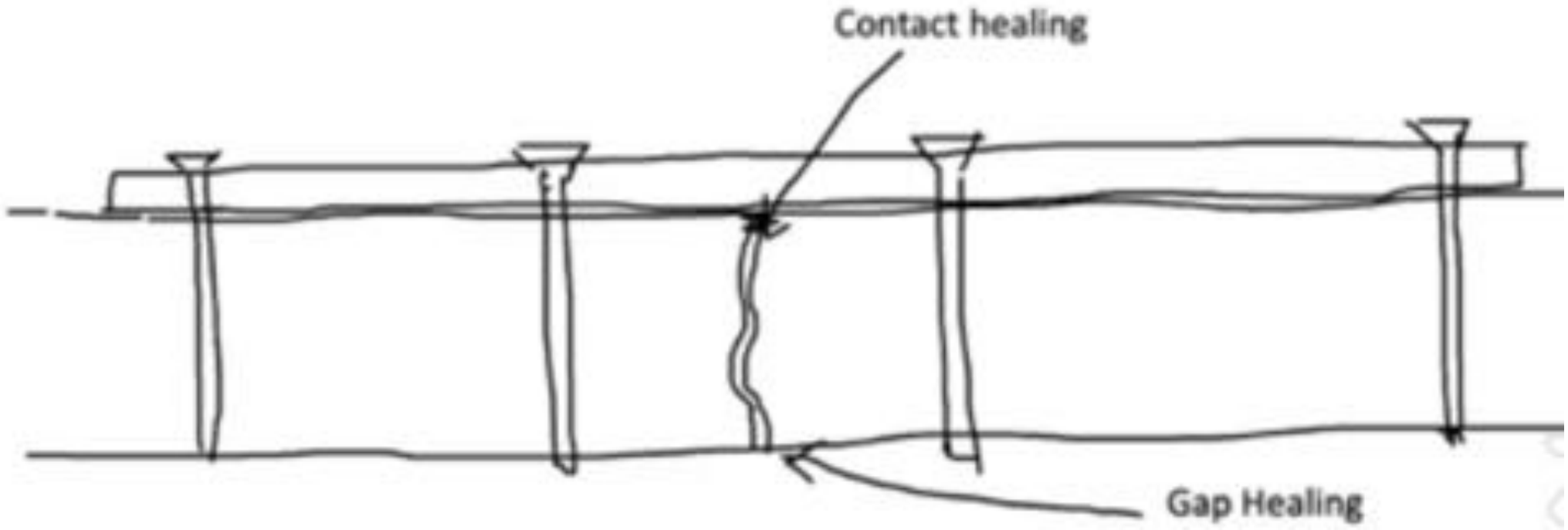


# PRIMARY BONE HEALING

# COMPRESSION PLATING

# TWO TYPES



# GAP HEALING

- OCCURS WHEN THERE IS MINIMAL GAP IN BETWEEN RIGIDLYFIXED FRACTURE ENDS
- WOVEN BONE IS INITIALLY FORMED IN TRANSVERSE ORIENTATION BETWEEN FRACTURE GAPS
- LATER REPLACED BY LAMELLAR BONE

# CONTACT HEALING

- SEEN WHEN FRACTURE ENDS ARE CLOSELY APPROXIMATED TO EACH OTHER WITHOUT ANY GAP

- CUTTING CONES ARE RESPONSIBLE

# WHICH TYPE OF FRACTURE HEALING OCCURS IN COMPRESSION PLATING ?

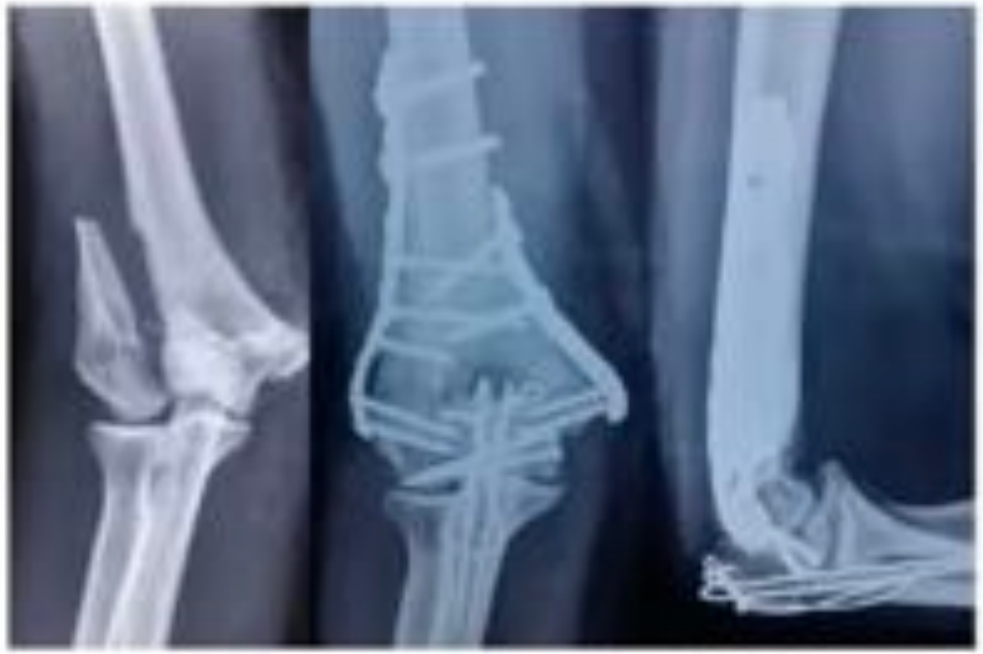
A) PRIMARY

B) SECONDARY

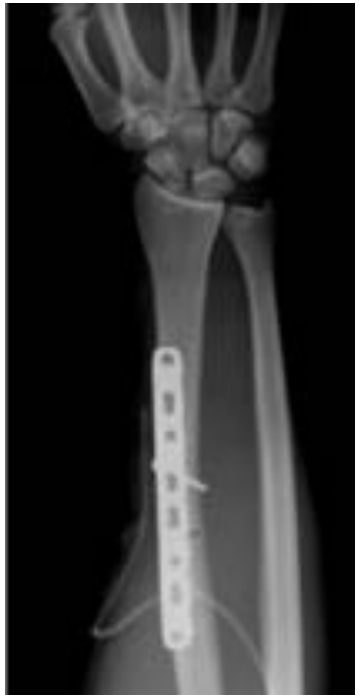
C) MIXED

D) INDIRECT HEALING

# Anatomic reduction and rigid fixation required in



ARTICULAR FRACTURE



FOREARM FRACTURE

# SECONDARY FRACTURE HEALING

- MORE COMMON METHOD OF FRACTURE HEALING

- SEEN IN ABSENCE OF RIGID FIXATION

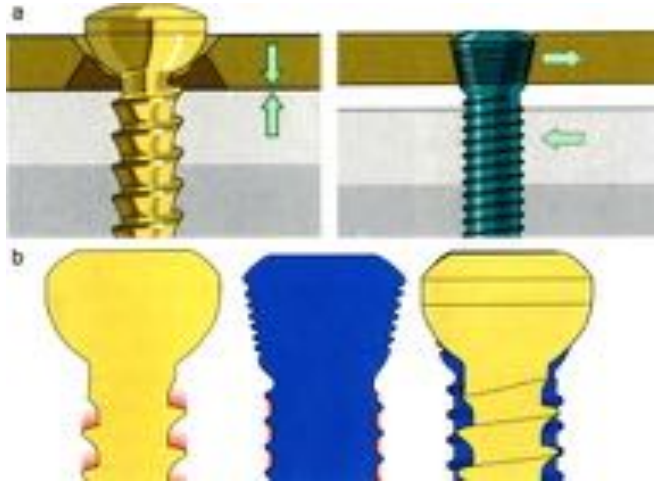
EG

CAST IMMOBILIZATION

INTRAMEDULLARY NAILING

BRIDGE PLATING FOR COMMINUTED METAPHYSEAL FRACTURES

# LOCKING PLATE



USED IN COMMINUTED FRACTURE ,
SEGMENTALLY DEFICIENT OR POROTIC BONE
WHERE BONE QUALITY IS POOR

PLATE IS USED AS A **INTERNAL FIXATOR** WITH MULTIPLE ANCHOR POINTS

ANATOMIC REDUCTION IS NOT REQUIRED AS **FIXED ANGLE CONSTRUCT** CONVERTS AXIAL LOAD TO COMPRESSIVE FORCE ACROSS FRACTURE SITE MINIMISING GAP LENGTH AND STRAIN

**2-10% - SECONDARY HEALING**

# WHICH TYPE OF FRACTURE HEALING OCCURS IN LOCKED COMPRESSION PLATING ?

A) PRIMARY

B) SECONDARY

C) MIXED

D) CONTACT HEALING

| PRIMARY BONE HEALING | SECONDARY BONE HEALING |
| --- | --- |
| NEEDS ABSOLUTE STABILITY WITH ANATOMICAL REDUCTION | RELATIVE STABILITY |
| FRACTURE GAP STRAIN < 2% | STRAIN > 2-10% |
| HEALING BY CUTTING CONES | |
| NO CALLUS FORMATION | CALLUS FORMATION – ENCHONDRAL BONE FORMATION |
| NO REMODELLING | REMODELLING |
| X-RAY – CAN'T SEE CALLUS | X-RAY – CAN SEE HEALING |

# TRUE REGARDING FRACTURE HEALING

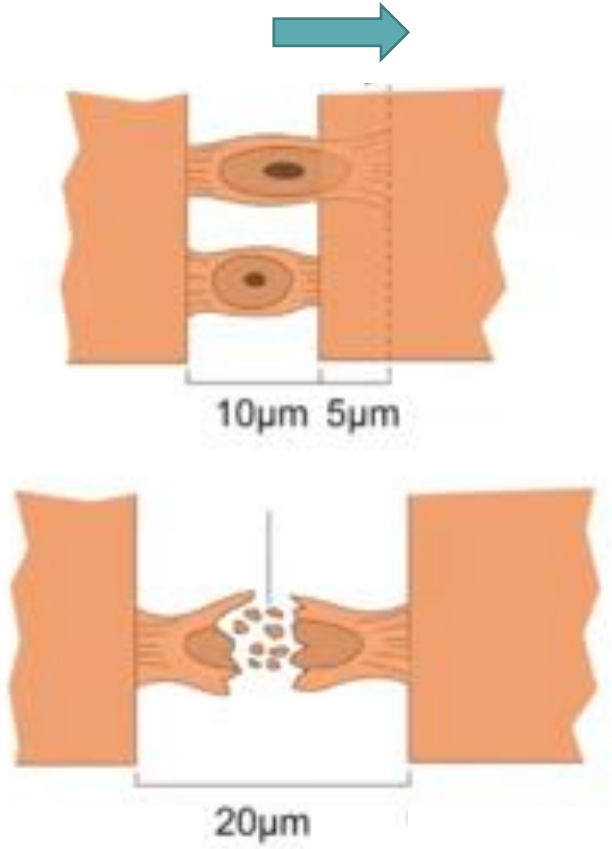
- Rigid internal compression fixation, which minimizes strain, will lead to healing by cutting cones
- Cortical bone can tolerate up to 10% strain
- Lamellar bone can tolerate up to 2 % strain
- Contact healing is seen in locked compression plating

# TRUE REGARDING FRACTURE HEALING

- Rigid internal compression fixation, which minimizes strain, will lead to healing by cutting cones
- Cortical bone can tolerate up to 2% strain
- Lamellar bone can tolerate up to 10% strain
- Secondary healing is seen in locked compression plating

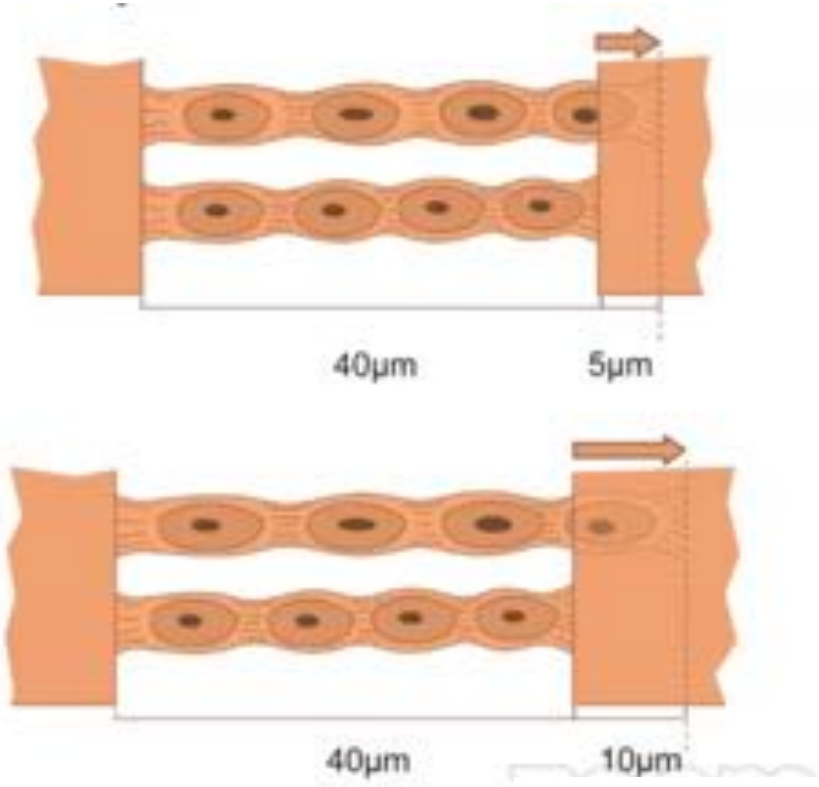
# HIGH STRAIN IN SMALL GAP

- In a minute gap with only few bridging cells
- If motion exceeds strain tolerance of the tissue
- Cell structure is destroyed



# LOW STRAIN IN LARGE GAPS

- If the gap is wider
- Strain is shared by many more bridging soft tissue
- Fragment motion does not cause intolerable strain



# SUMMARY OF HEALING

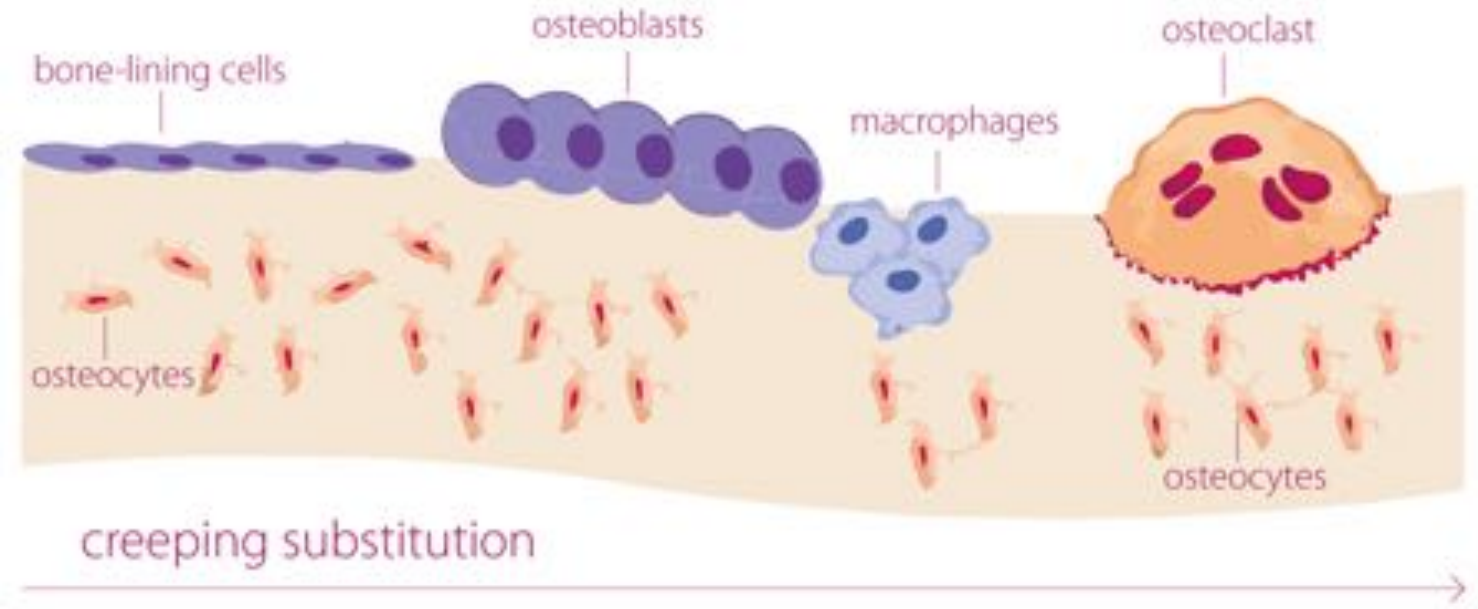

Absolute Stability
No Movement
Good Blood Supply

Relative Stability
Controlled Movement
Good Blood Supply

# CANCELLOUS BONE HEALING

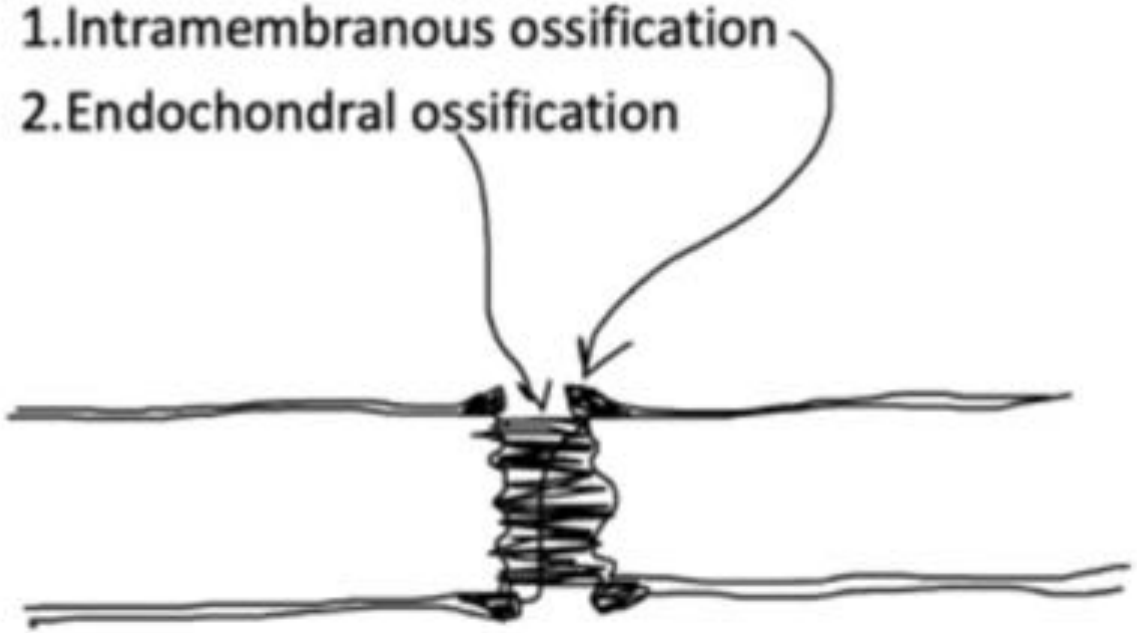
- HEALS WITH LITTLE TO NO CALLUS FORMATION AS LONG AS THE BONE ENDS ARE CLOSE TOGETHER
- DIRECT OSTEOBLASTIC ACTIVITY – **CREEPING SUBSTITUTION**
- IF FRACTURE ENDS ARENT CLOSE TOGETHER, THE BONE WILL HEAL WITH CALLUS FORMATION AS A HEMATOMA WILL FILL IN THE GAP BETWEEN THE BONY ENDS

# CREEPING SUBSTITUTION



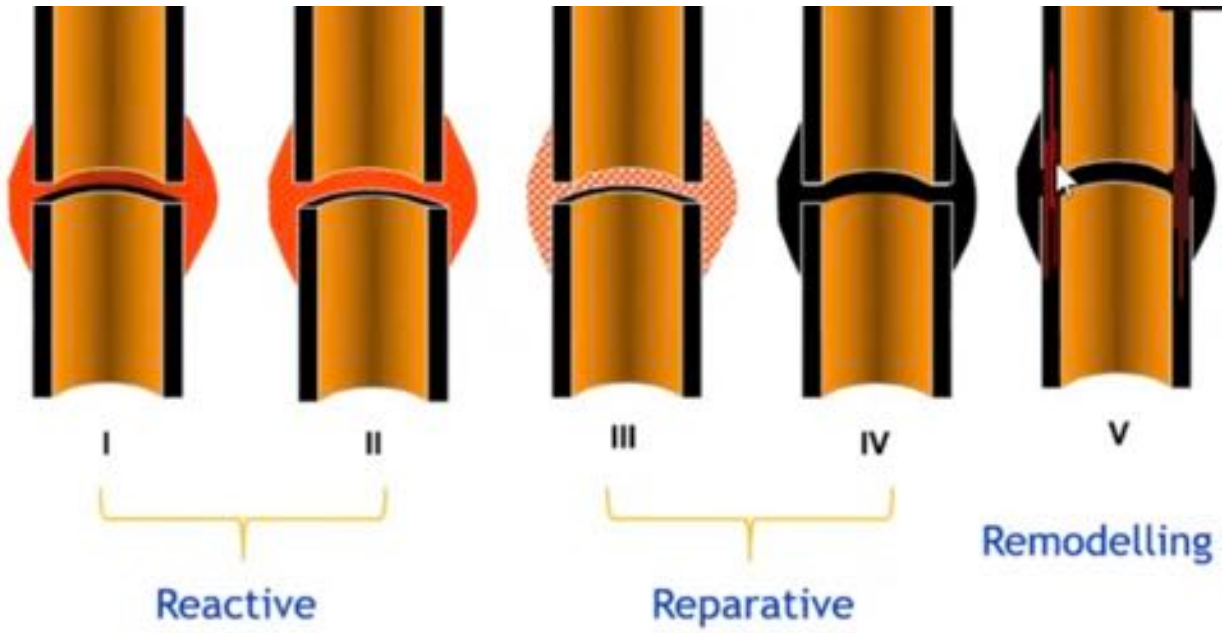
# FRACTURE HEALING

- 2 discrete process
  1. Intramembranous ossification
  2. Endochondral ossification

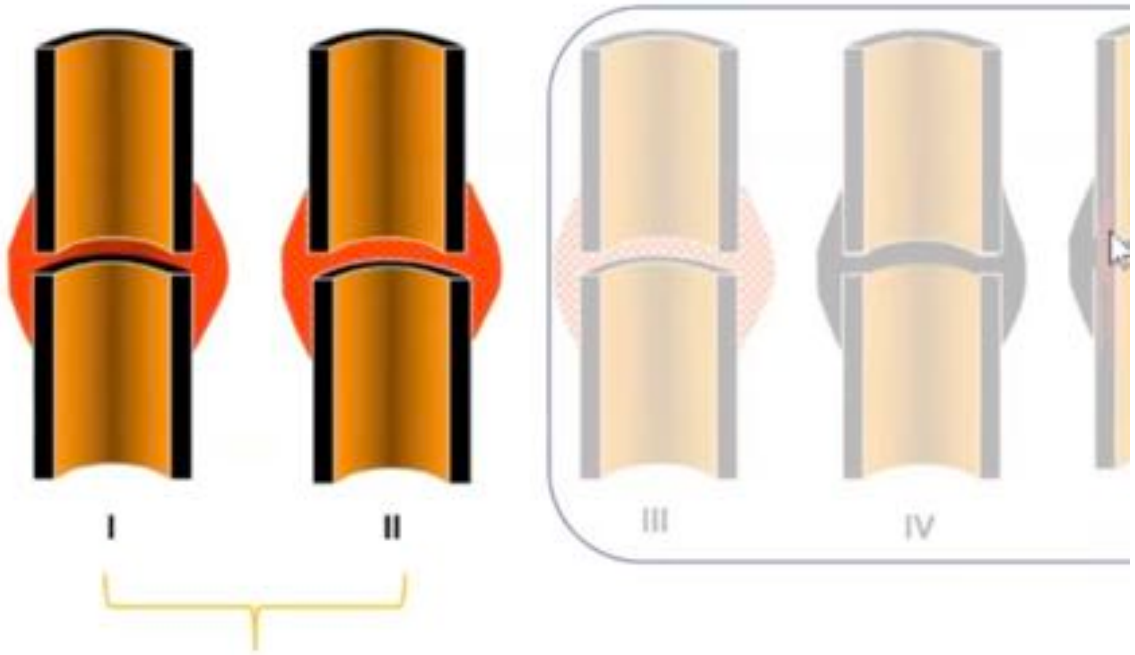


# STAGES OF NATURAL BONE HEALING

1. REACTIVE STAGE
2. REPARATIVE STAGE
3. REMODELLING STAGE



# REACTIVE STAGE




I. Haematoma Formation

II. Inflammation

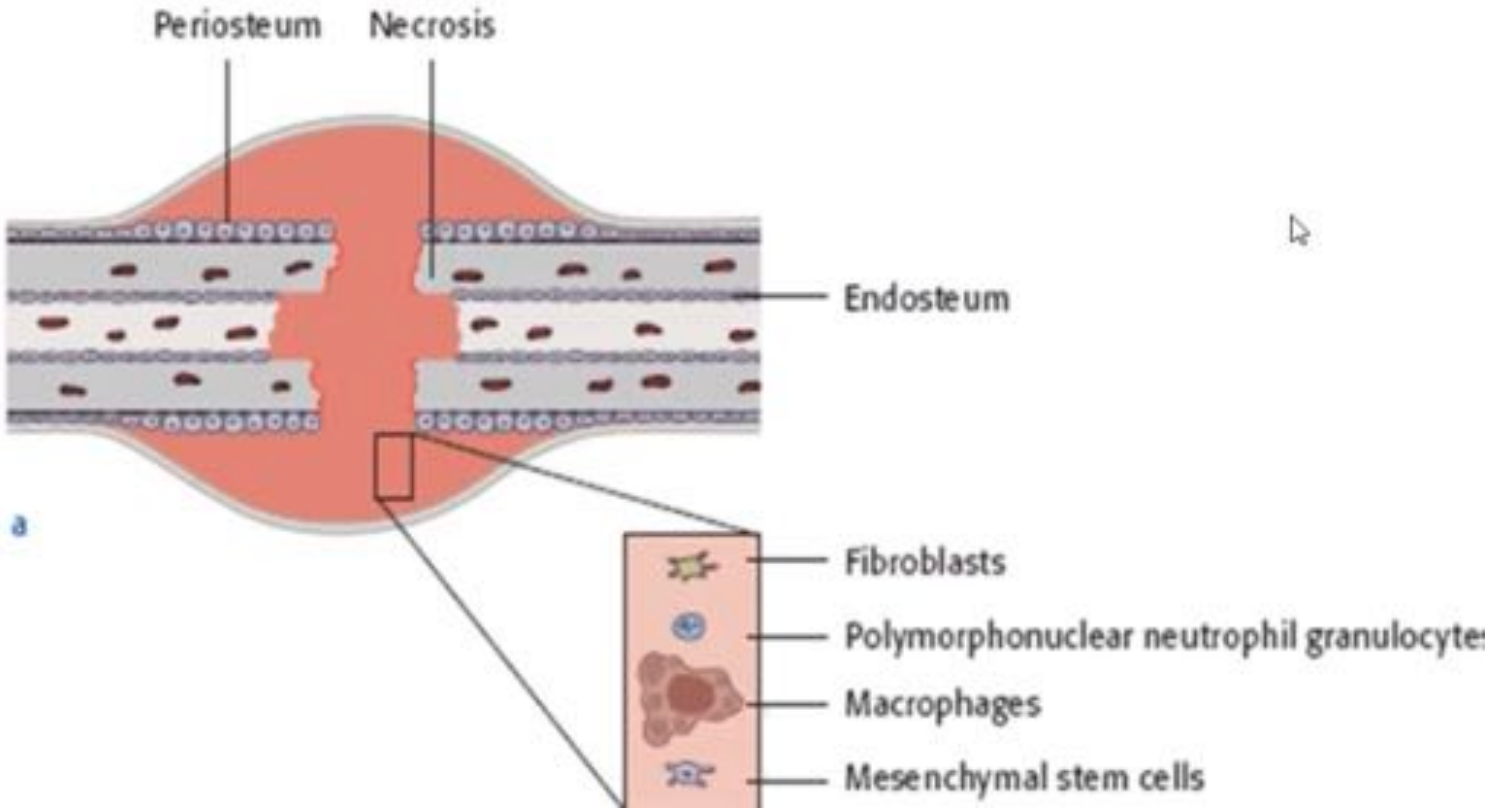
# BLOOD CLOT FORMATION / HEMATOMA FORMATION

PLATELET AGGREGATION + CLOT FORMATION

PDGF → Activate clotting cascade + Complement cascade

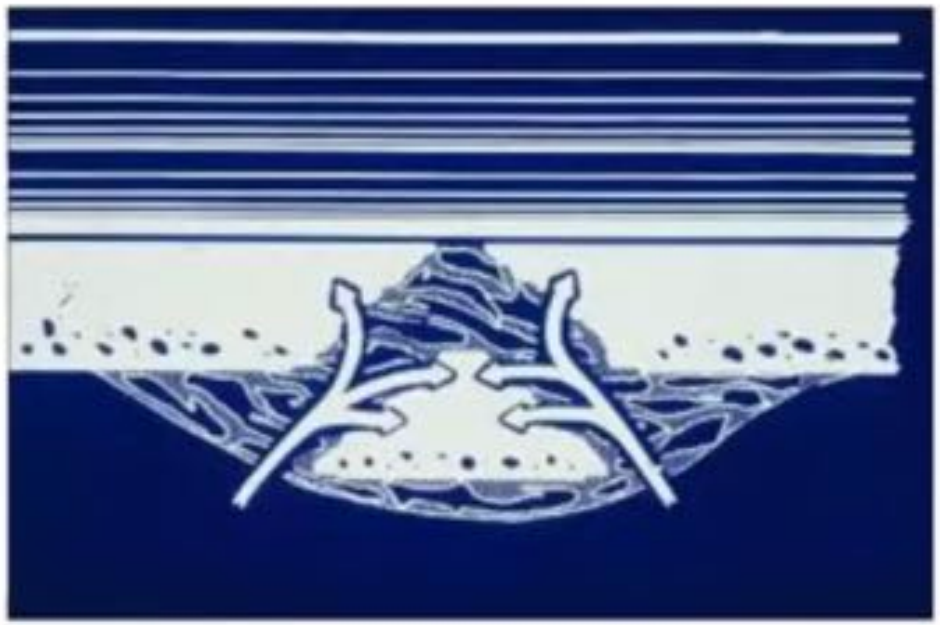
**STEP WISE AMPLIFICATION**

# INFLAMMATION



# INFLAMMATION

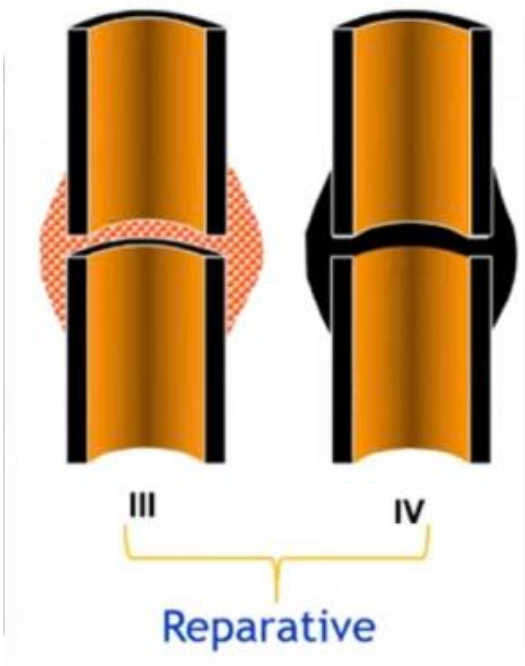
- CHEMOTACTIC – NEUTROPHIL FOLLOWED BY MACROPHAGE
- ANGIOGENIC
- OSTEO-INDUCTIVE



Acute inflammatory reaction

Ingrowth of blood vessels + Pluripotent Mesenchymal stem cells appear

# REPARATIVE PHASE




III. Callus formation

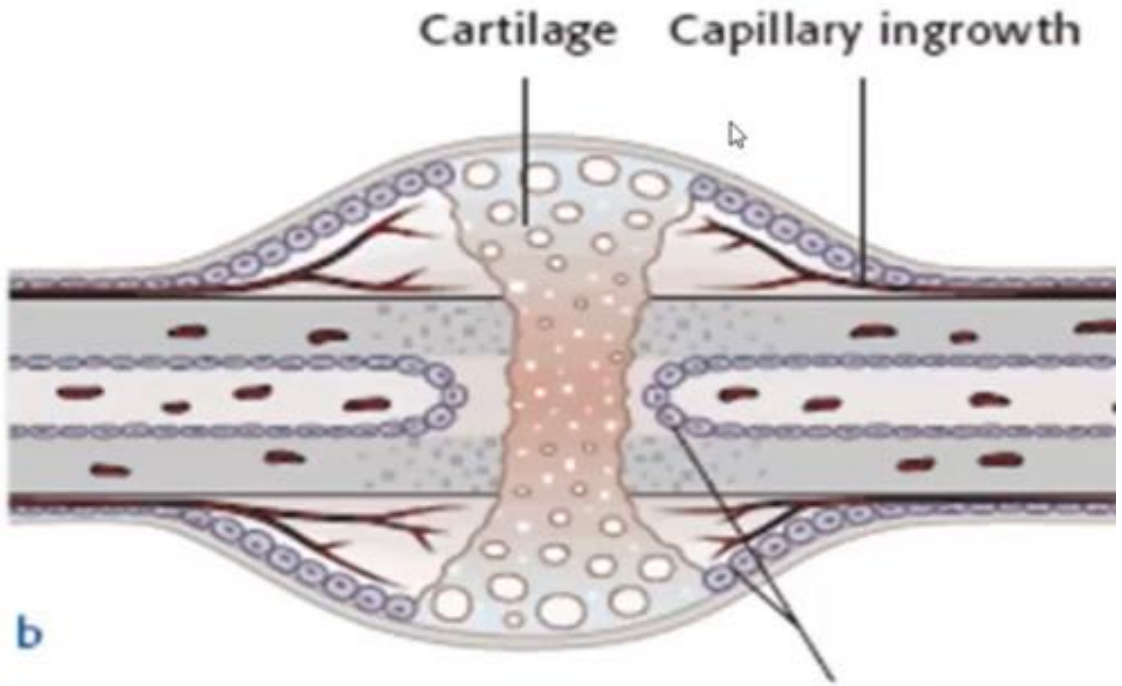
IV. Consolidation

# REPAIR PHASE

SOFT CALLUS → HARD CALLUS

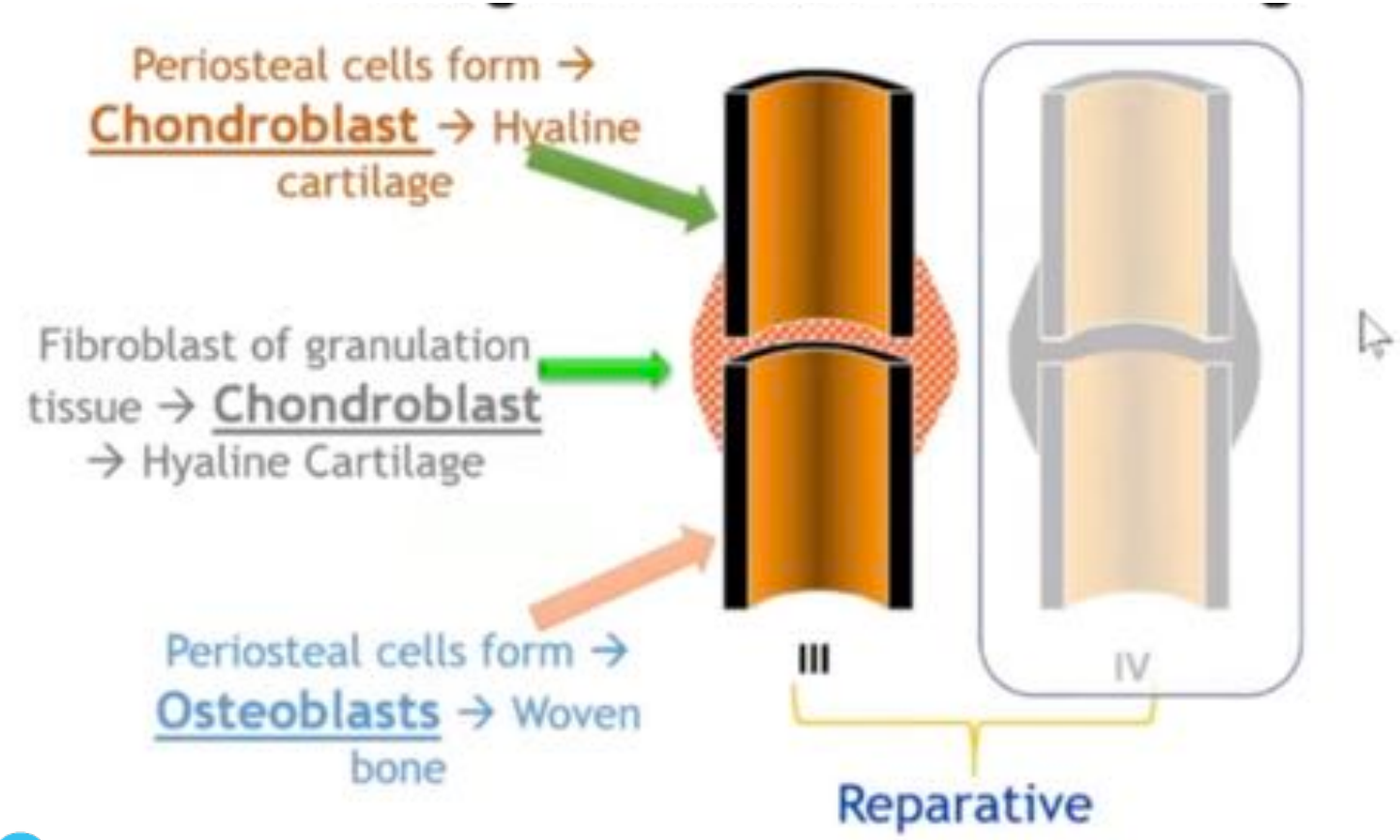
DEPENDS ON **PERREN'S STRAIN THEORY**

# SOFT CALLUS



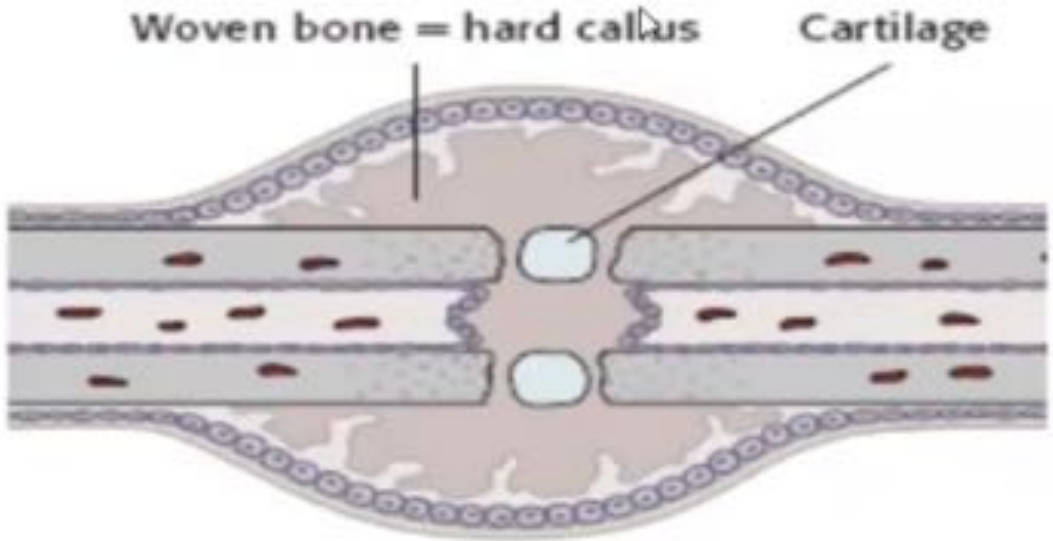

OSTEOBLASTS

# CALLUS FORMATION

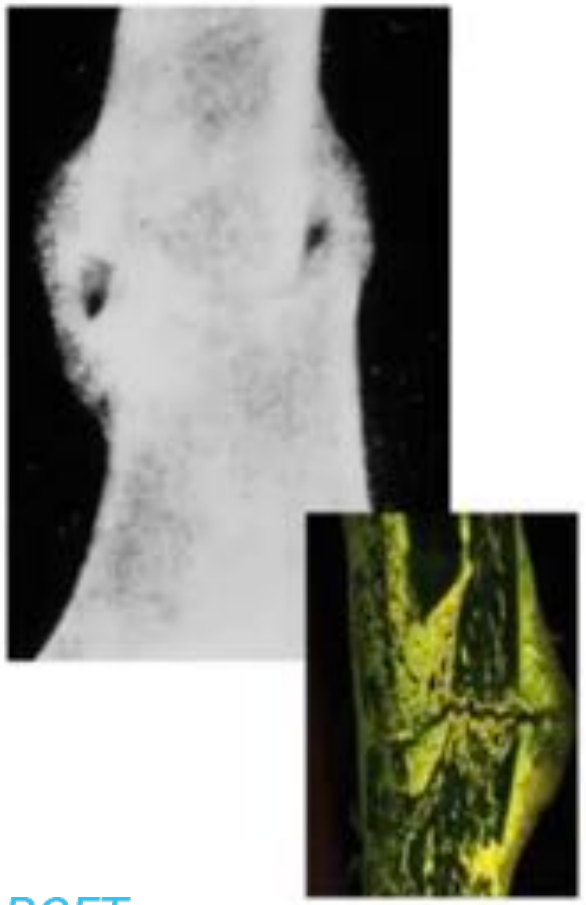


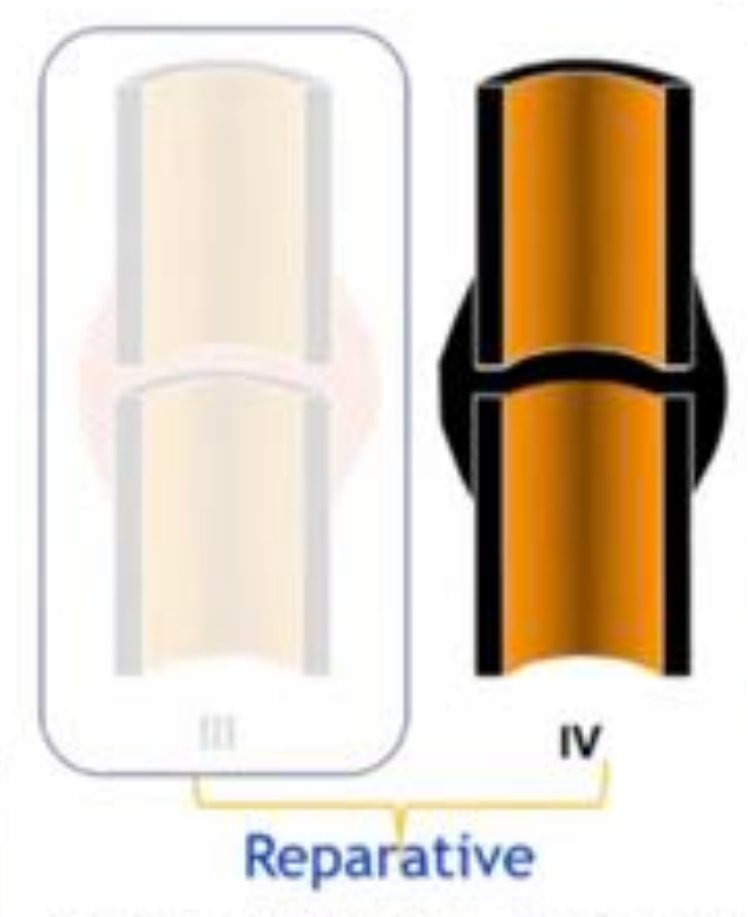
# Hard Callus Formation

## Intramembranous and endochondral ossification



# CONSOLIDATION

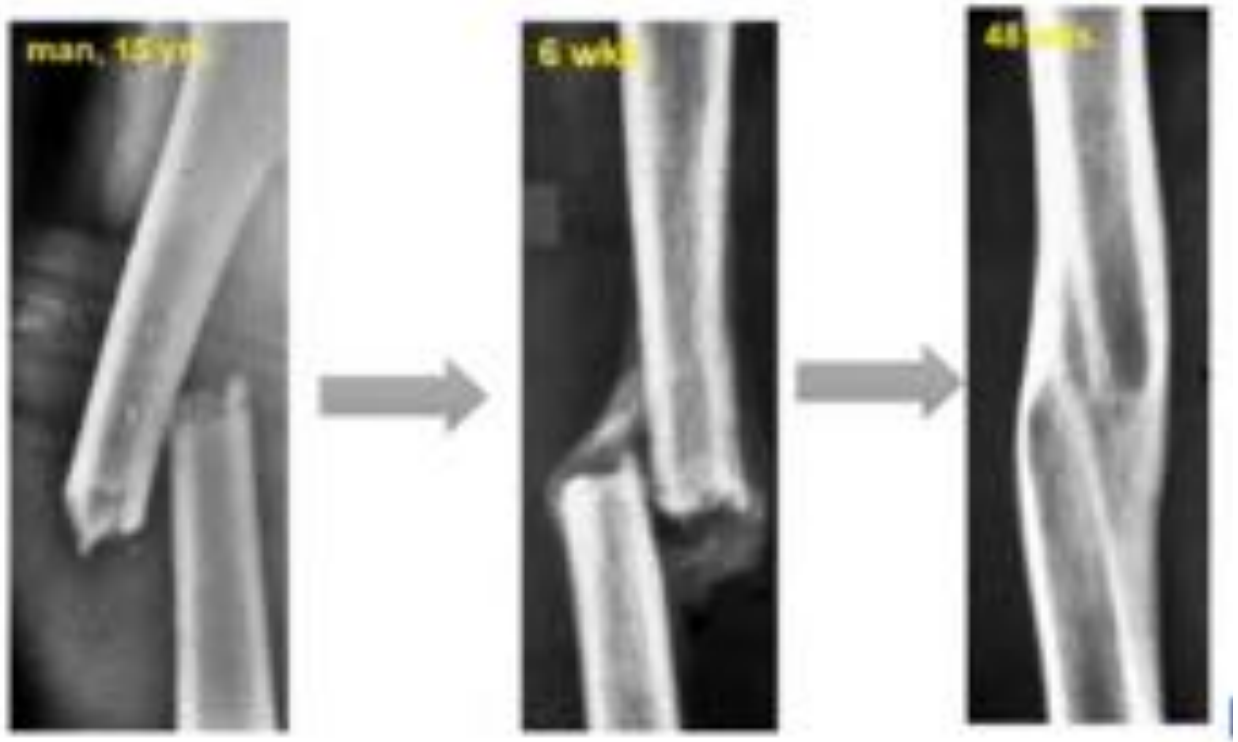





- RESORPTION OF BONE ENDS
- FRACTURE LINE VAGUE
- BRIDGING CALLUS

**CONSOLIDATION – SOFT CALLUS CONVERTED TO RIGID CALCIFIED TISSUE BY ENCHONDRAL OSSIFICATION**

# REMODELLING



TRABECULAR BONE → COMPACT BONE

Osteoclastic resorption and osteoblastic new bone formation

# REMODELLING PHASE

- OVER YEARS
- IT OBEYS **WOLFFS LAW**, BALANCE BETWEEN ANABOLIC AND CATABOLIC PHASE
- REMODEL ALONG THE STRESS LINES

# WOLFF'S LAW

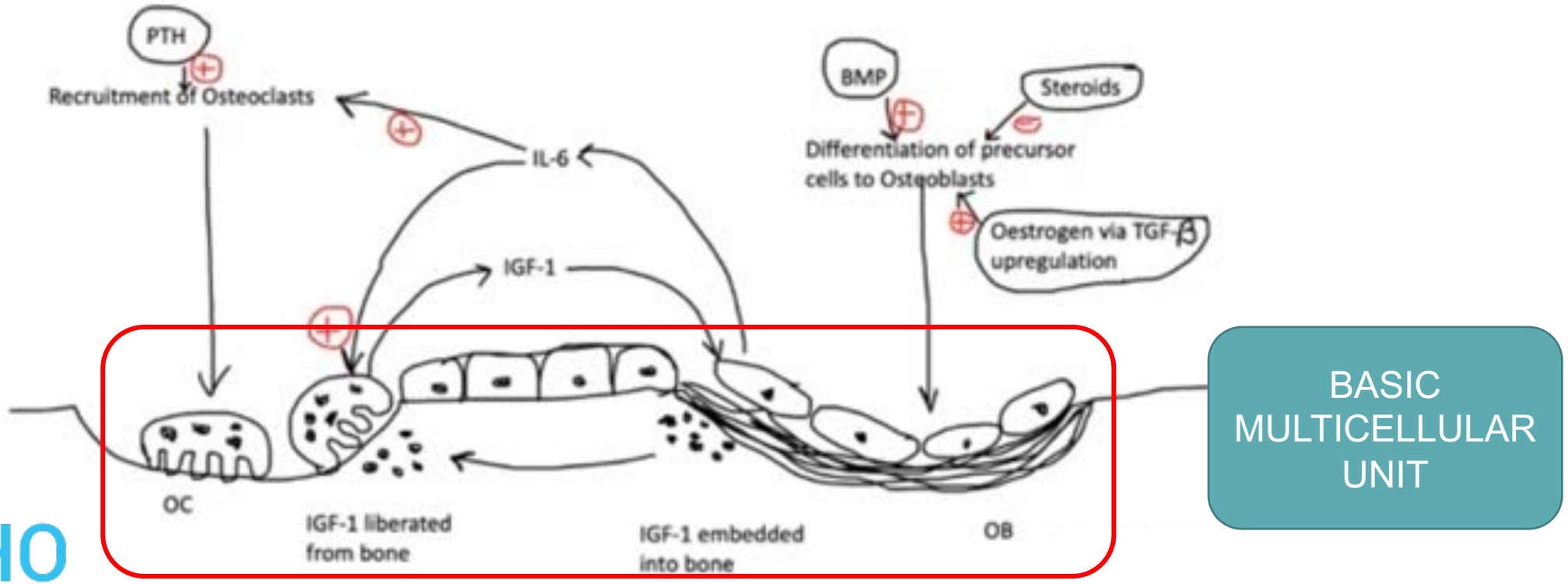
- BONE ASSUMES CONFIGURATION AND SHAPE BASED ON STRESSES ACTING ON IT
- OSTEOCLASTIC ACTIVITY BEING PREDOMINANT ON ELECTROPOSITIVE TENSION SIDE
- OSTEOBLASTIC ACTIVITY ON ELECTRONEGATIVE COMPRESSION SIDE

# BONE REMODELLING ALWAYS HAPPENS IN THE BONE

ALL BONES PARTICIPATE IN REMODELLING

BONE RESORPTION –--- BONE FORMATION

OSTEOCLAST OSTEOBLAST




BASIC MULTICELLULAR UNIT

# WHY REMODELLING ?

ALLOWS SKELETON TO

- RESPOND TO MECHANICAL LOADING (WOLFFS LAW)
- REPAIR AND PREVENT MICRO DAMAGE ( WEAR AND TEAR)
- RELEASE GROWTH FACTORS AND MINERALS (Ca,Ph)

# FACTORS AFFECTING BONE HEALING

- LOCAL FACTORS

Degree of soft tissue trauma

Open fracture

Infection

NV injury

Degree of bone loss

Degree of immobilization

Tumor

Site of bone/ type of bone

- SYSTEMIC FACTORS

Smoking

DM

Age

Nutrition level

Drugs

Hormones

Head injury