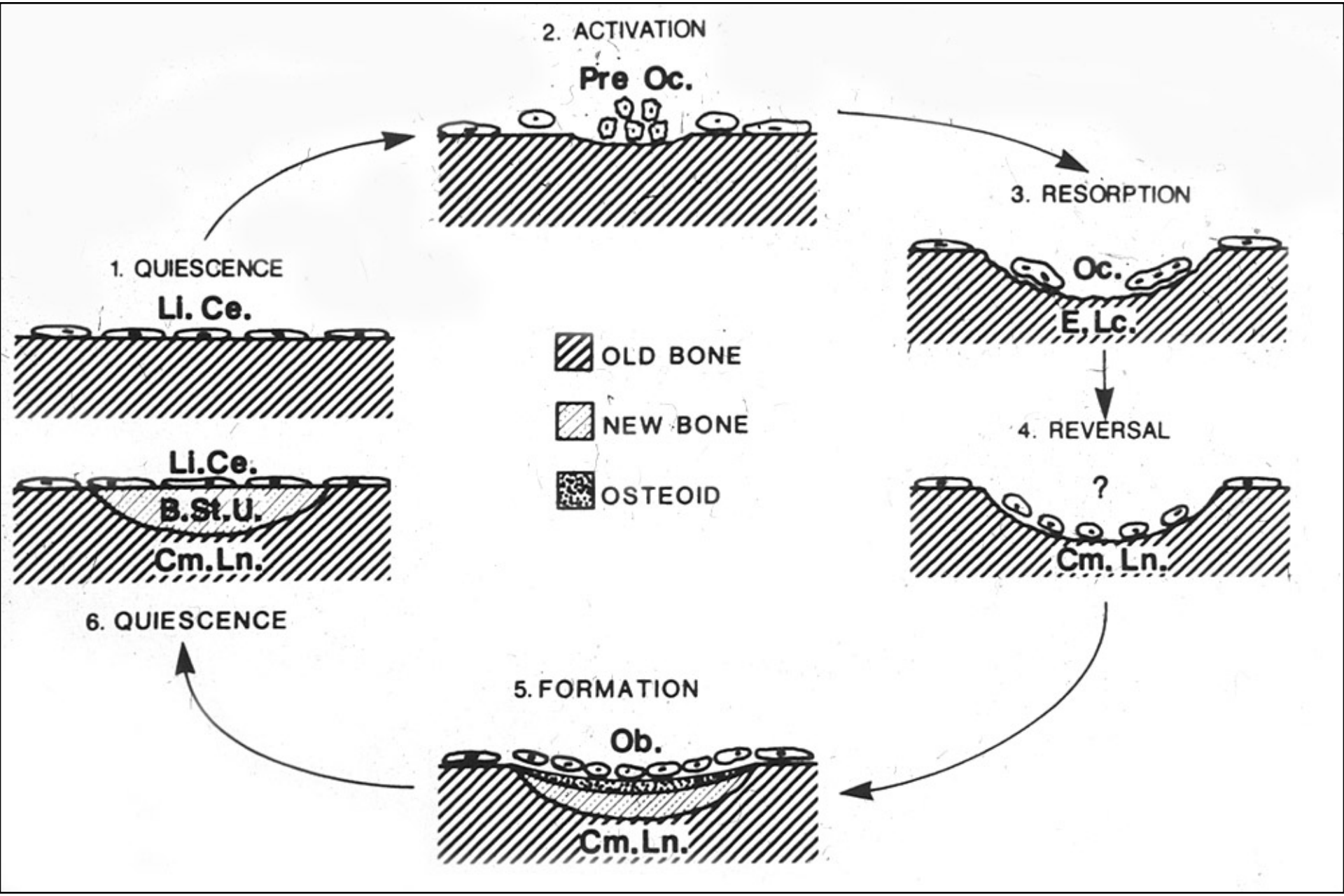
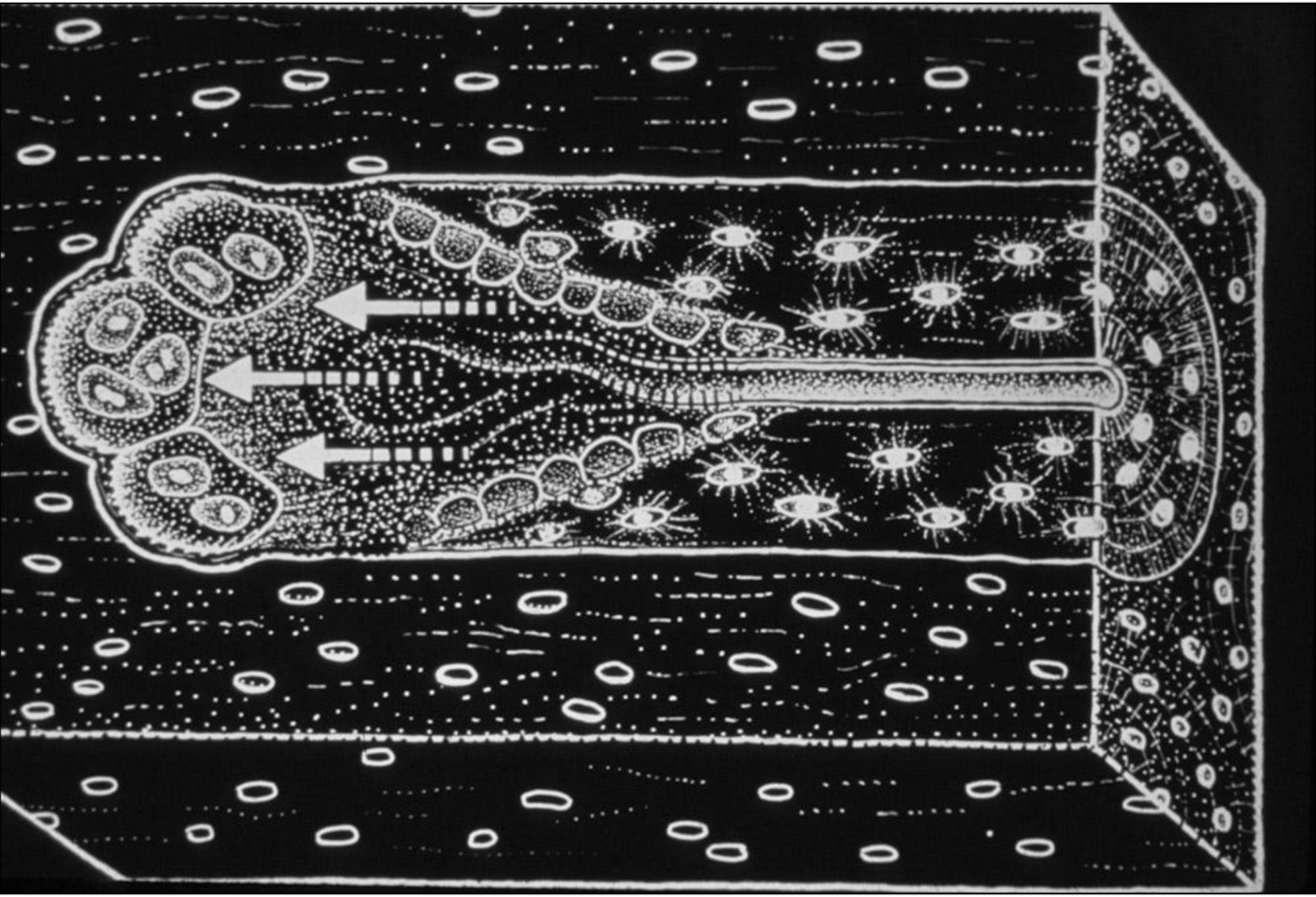
Review of Remodeling

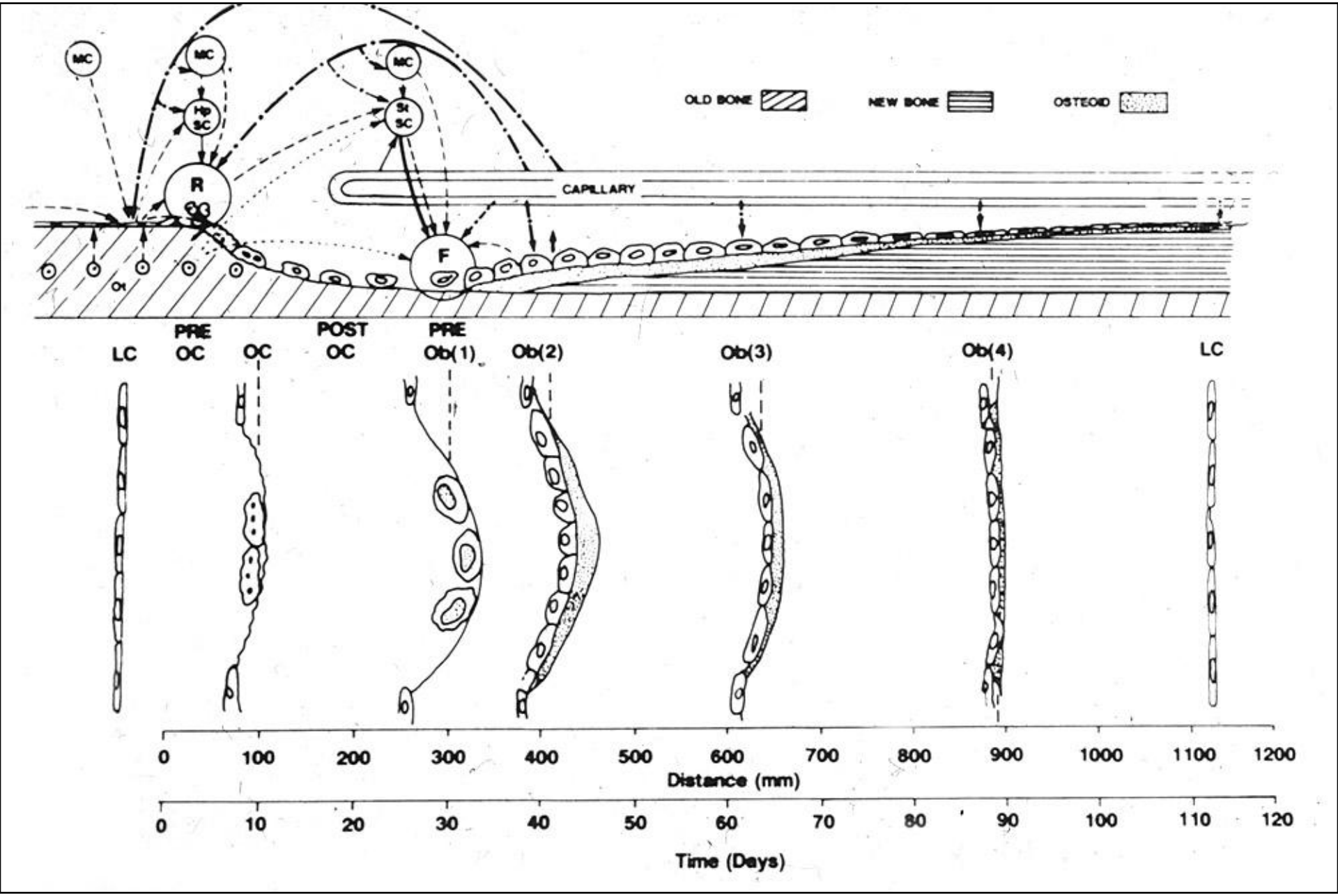
- Activation – signals the osteoblasts to “prime” the surface and stimulate osteoclast recruitment
- Resorb – osteoclasts initiate and macrophages complete resorption
- Reversal – inhibit resorption and stimulate formation likely via actions of cytokines (eg.TGF-β) – leaves cement where reversal occurred
- Formation – new bone synthesis by osteoblasts


2. ACTIVATION
Pre Oc.
3. RESORPTION
Oc.
E, Lc.
4. REVERSAL
?
Cm. Ln.
5. FORMATION
Ob.
Cm.Ln.
6. QUIESCENCE
1. QUIESCENCE
Li. Ce.
Li.Ce.
B.St.U.
Cm.Ln.
OLD BONE
NEW BONE
OSTEOID

Cement Lines

- Depth of resorption (BSU or Osteon)
- Basophilic line in decalcified H&E section
- Collagen poor
- Proteoglycan rich
- Mineral rich
- Irregular – reversal
- Regular – resting
- Allow for slippage between remodeling units
- “Sump” for microcracks



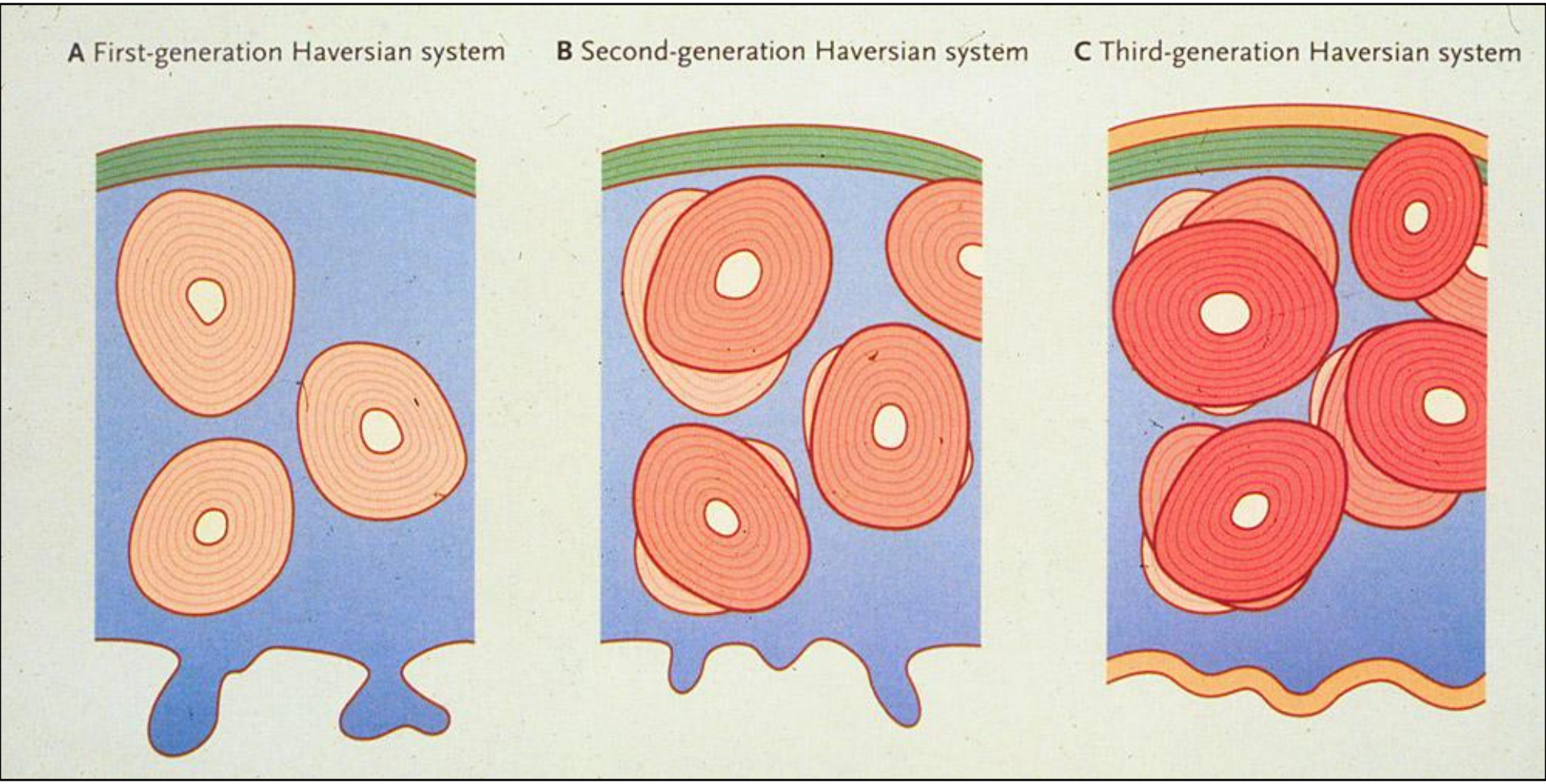

MC
MC
MC
Hp SC
St SC
R
F
CAPILLARY
OLD BONE
NEW BONE
OSTEOID
LC
PRE OC
OC
POST OC
PRE Ob(1)
Ob(2)
Ob(3)
Ob(4)
LC
0 100 200 300 400 500 600 700 800 900 1000 1100 1200
Distance (mm)
0 10 20 30 40 50 60 70 80 90 100 110 120
Time (Days)

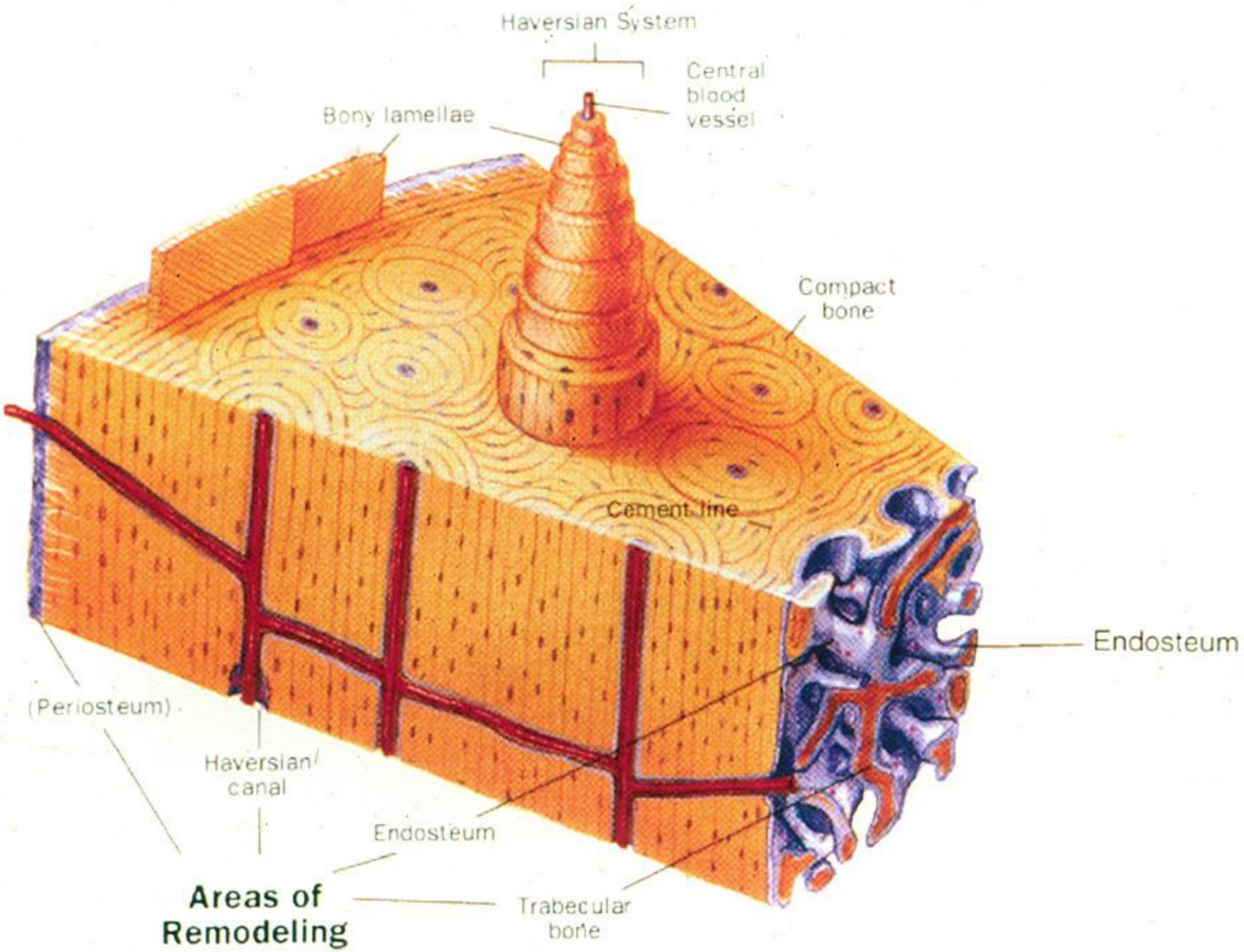
Primary and Secondary Remodeling

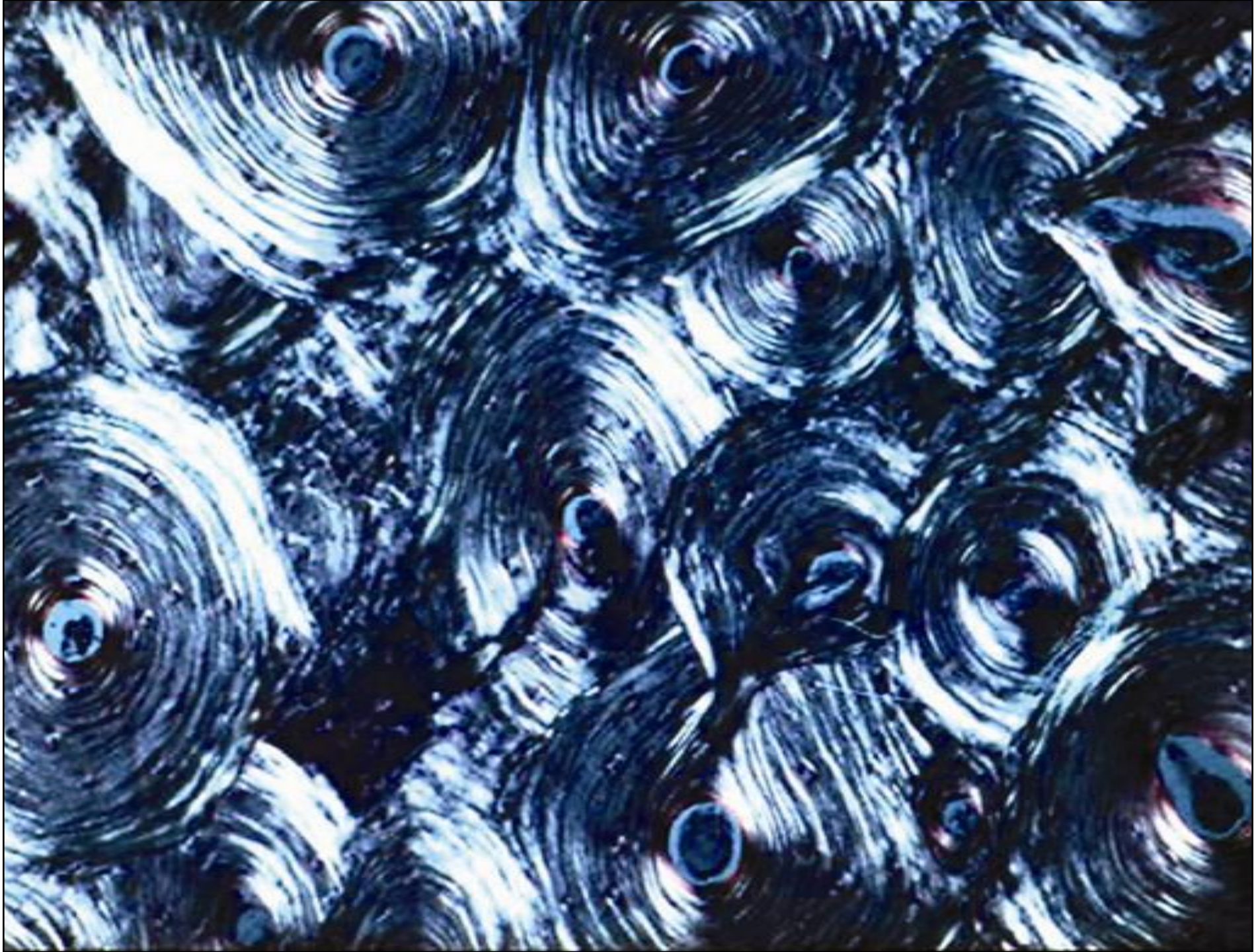
- Primary remodeling - areas that have NEVER been remodeled into osteons or BSUs
- Secondary remodeling- remodeling areas ALREADY in the form of osteons or BSUs

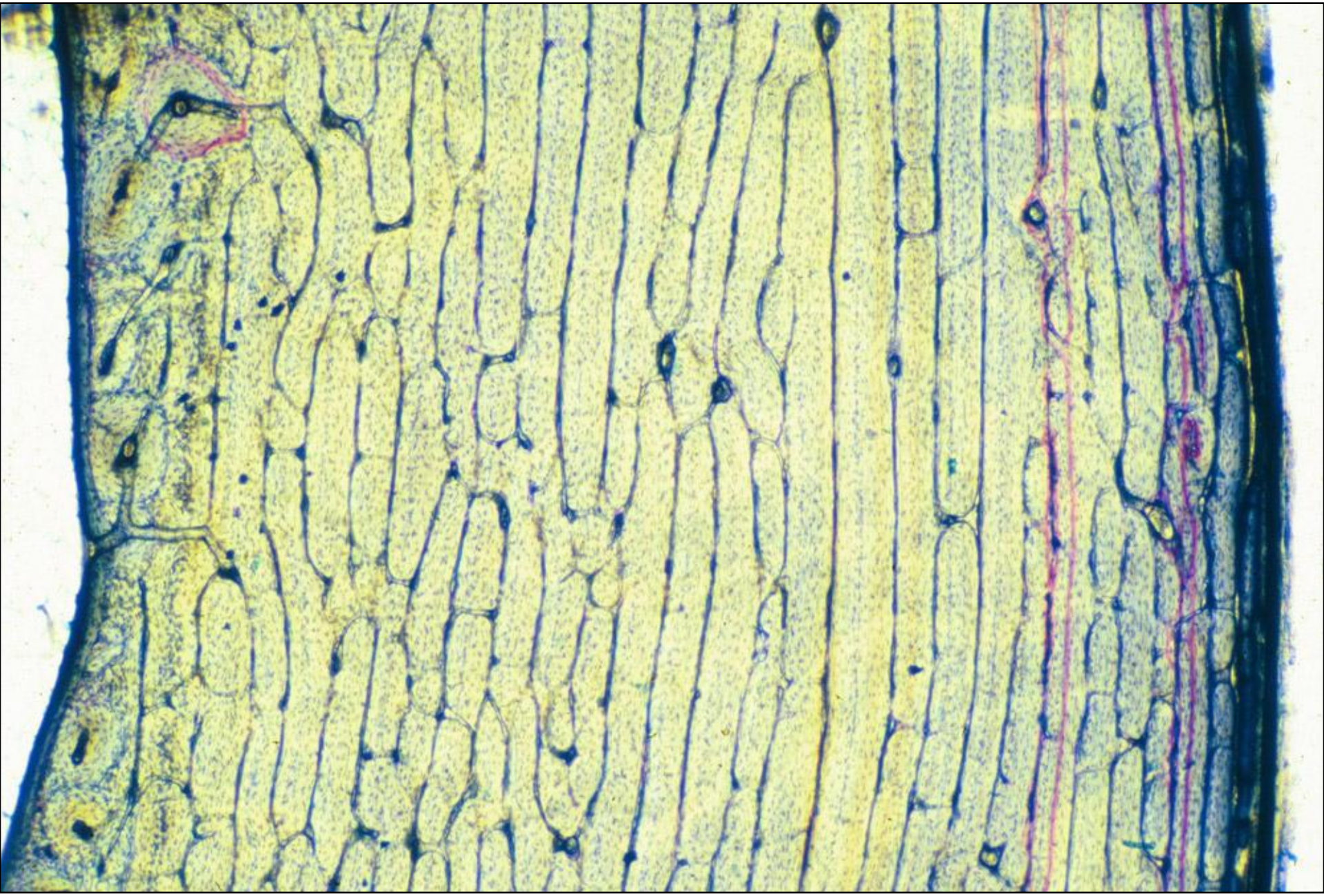
Why Remodel

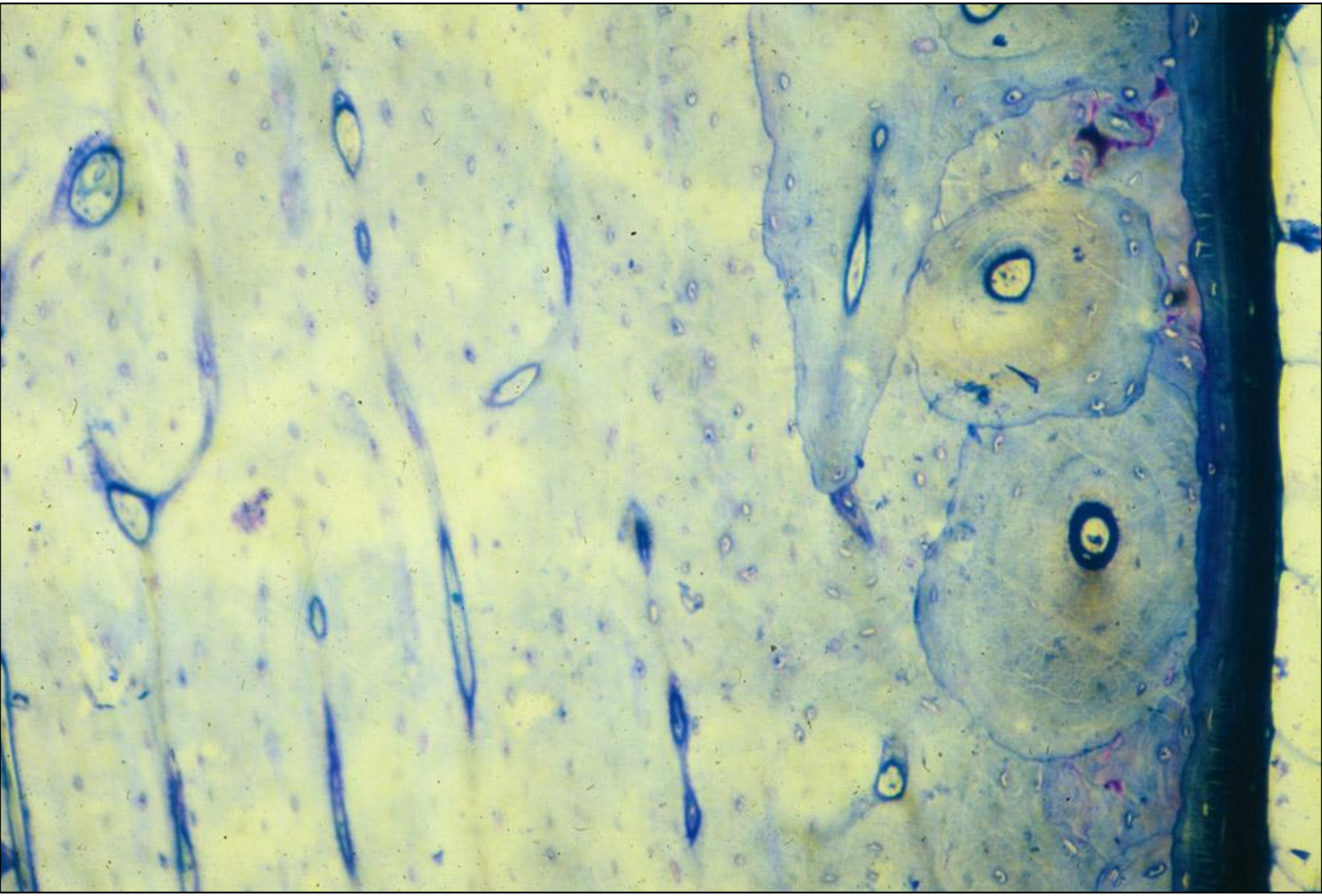
- Primary remodeling – create bone remodeling units with greater flexibility and ability to dissapate microcracks (due to shape and orientation of remodeling units and presence of cement lines); orient vasculature/nerves in cortical bone
- Secondary – repair microdamage

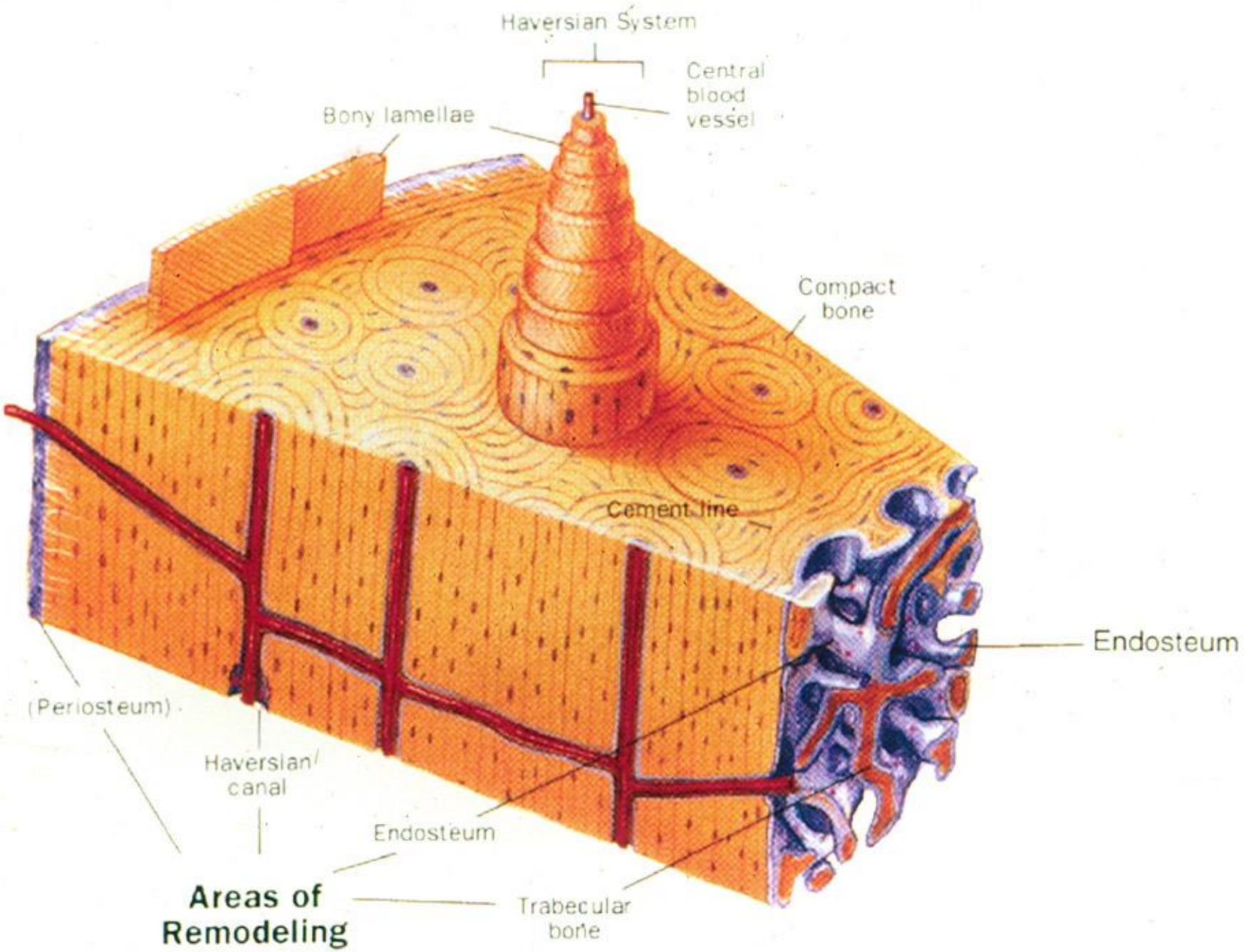

A First-generation Haversian system
B Second-generation Haversian system
C Third-generation Haversian system


Haversian System
Central blood vessel
Bony lamellae
Compact bone
Cement line
Endosteum
(Periosteum)
Haversian canal
Endosteum
Areas of Remodeling
Trabecular bone








Haversian System
Central blood vessel
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Compact bone
Cement line
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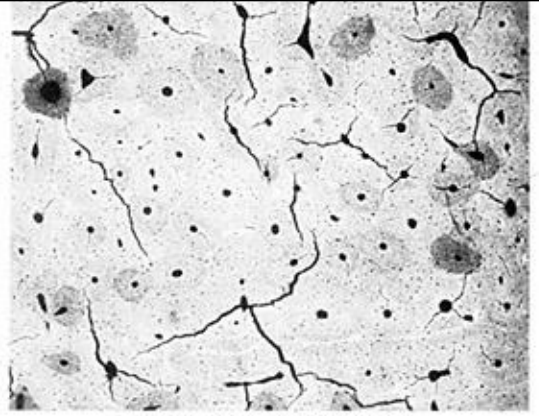
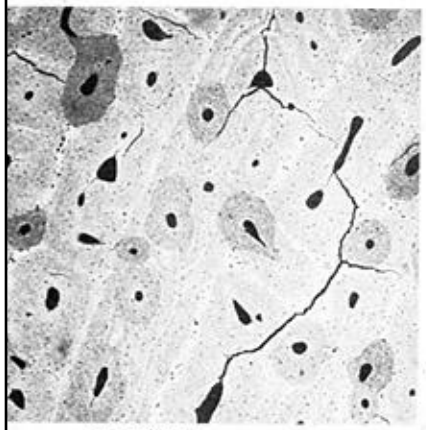
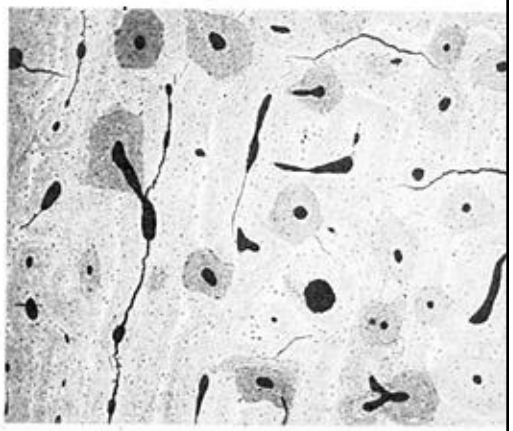


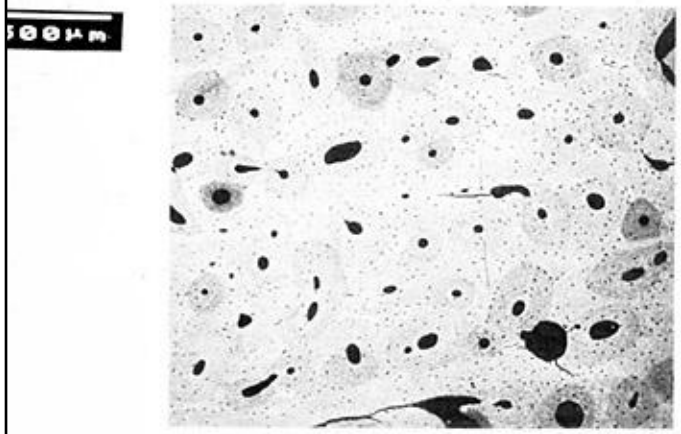
CRANIAL (TENSION)



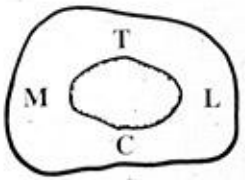
MEDIAL

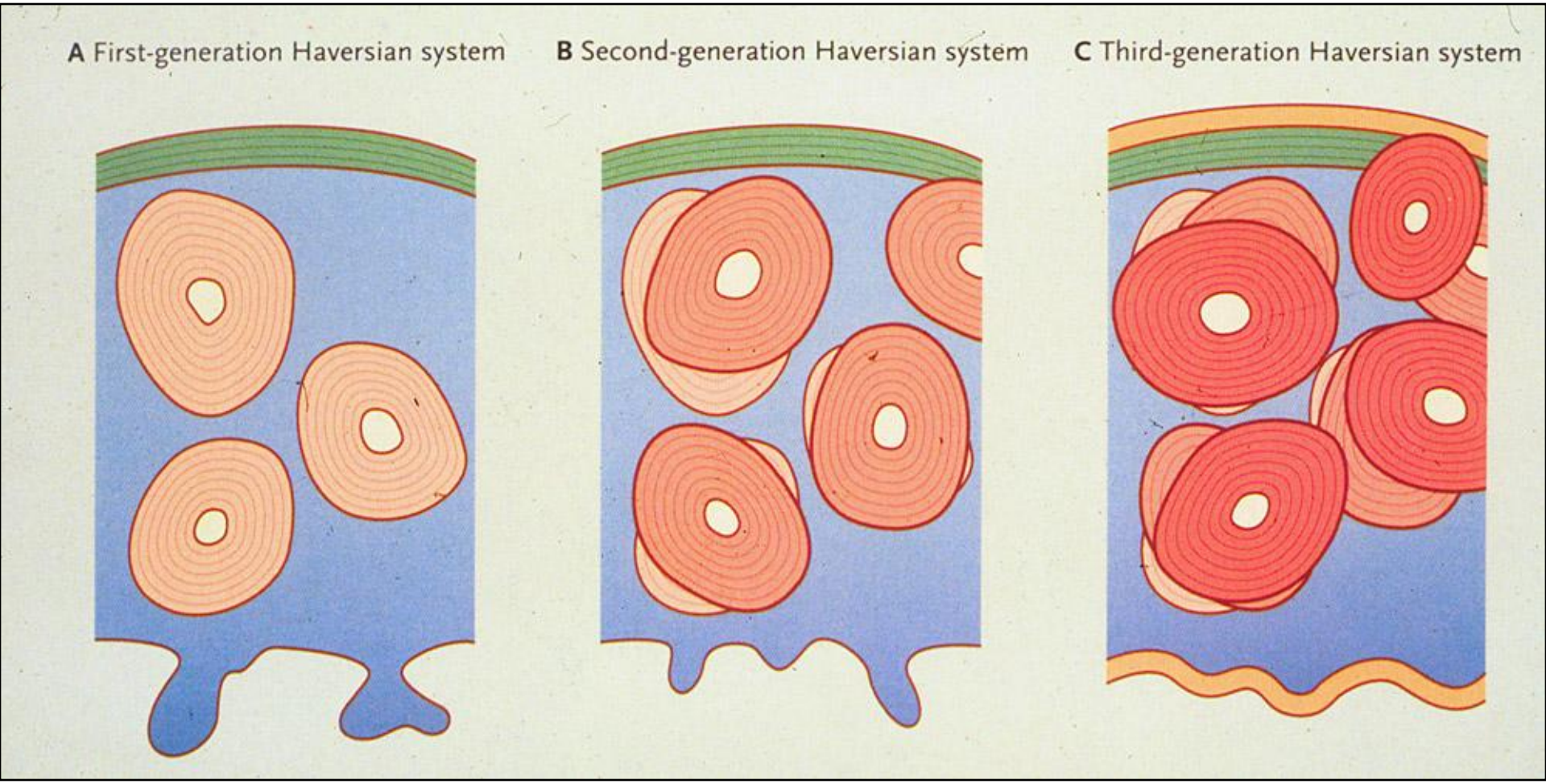


LATERAL



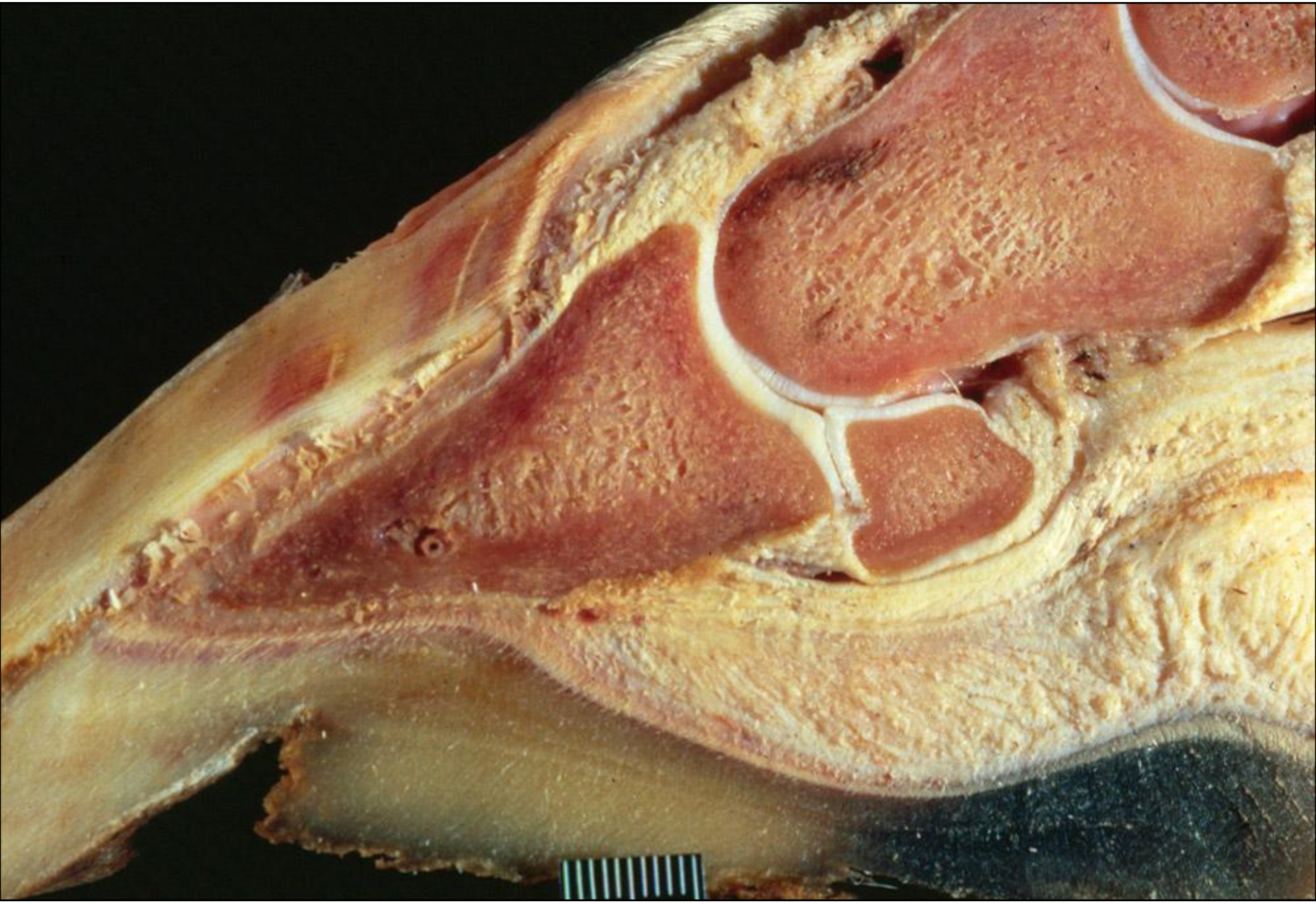
CAUDAL (COMPRESSION)

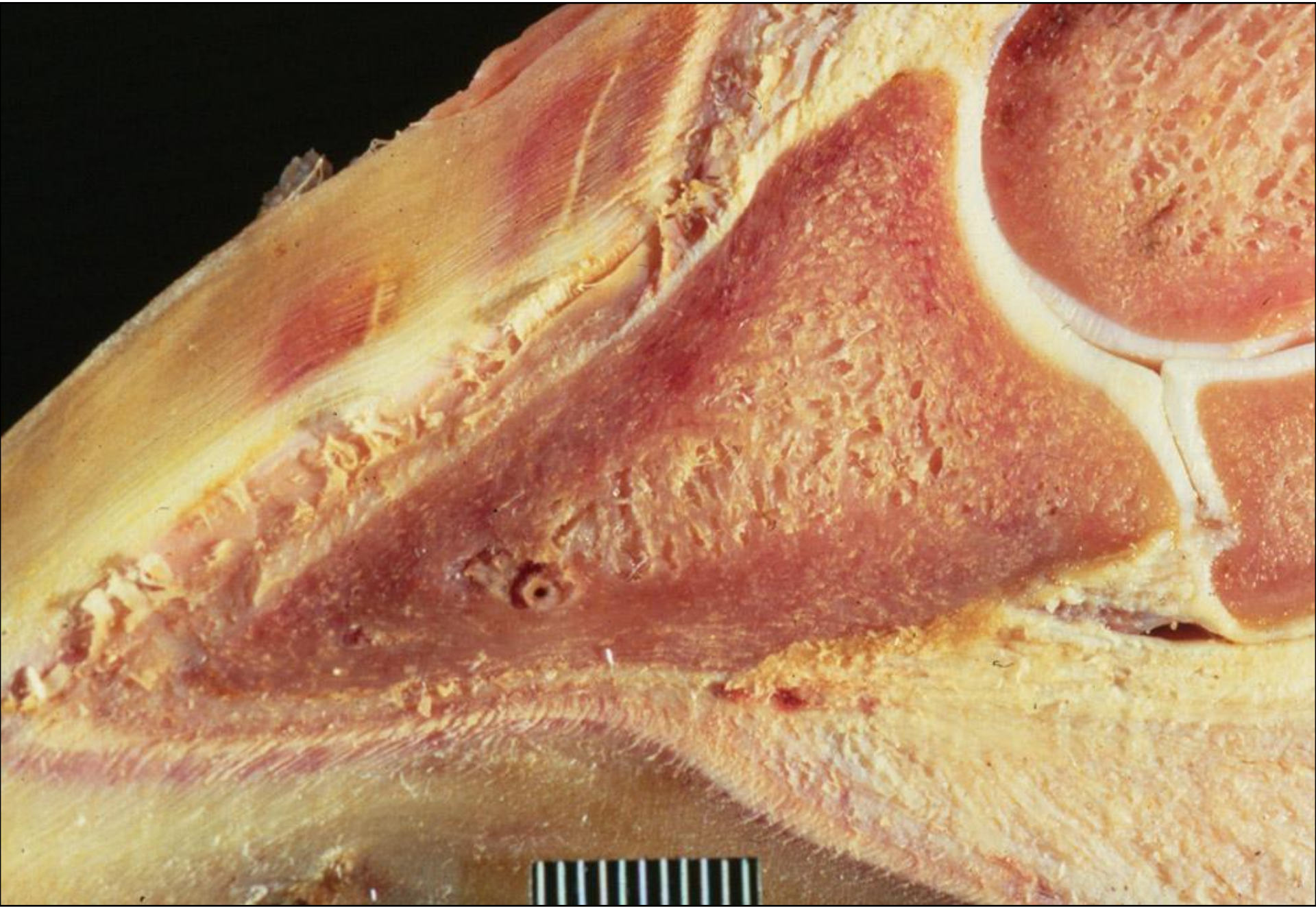


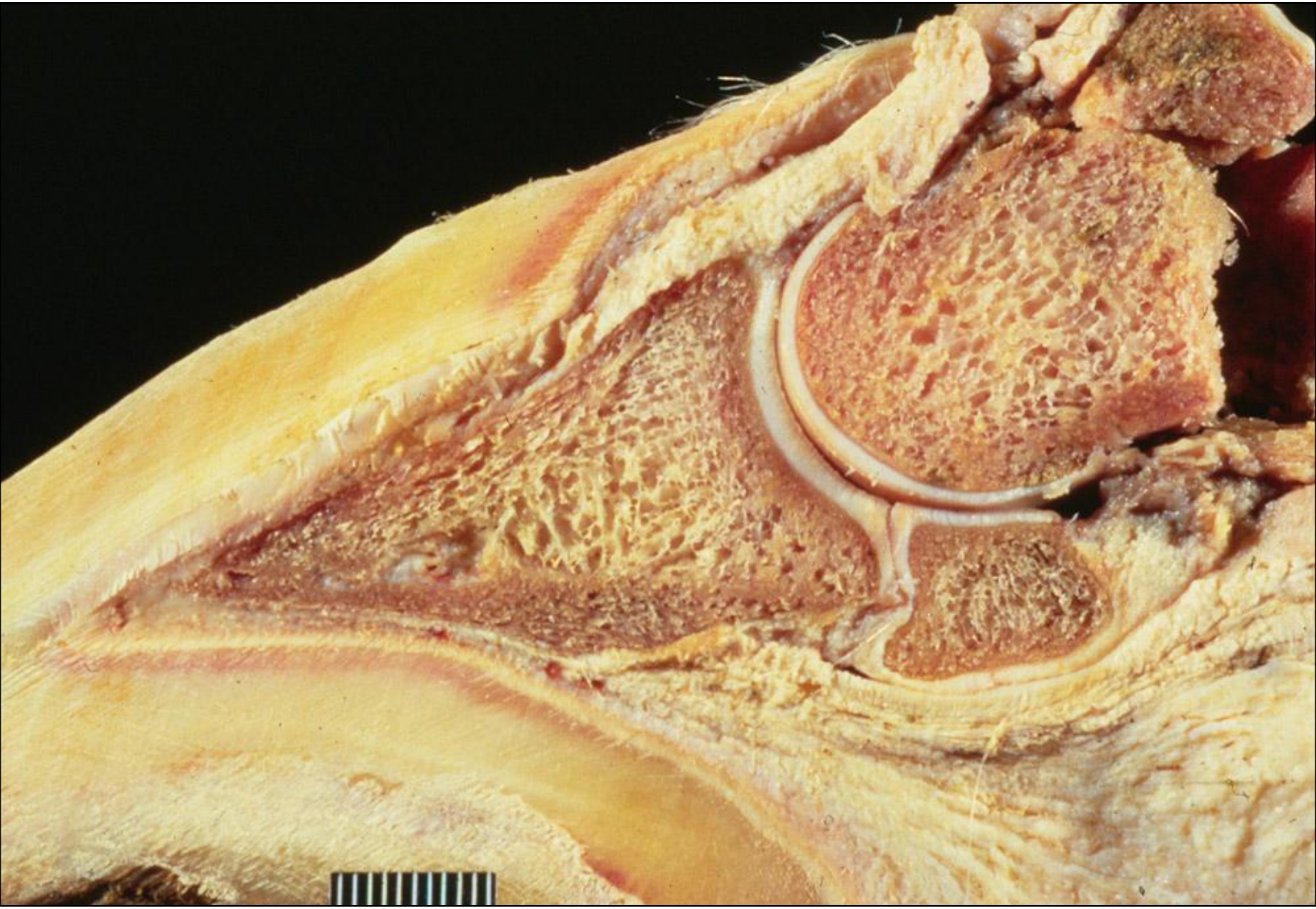

A First-generation Haversian system
B Second-generation Haversian system
C Third-generation Haversian system

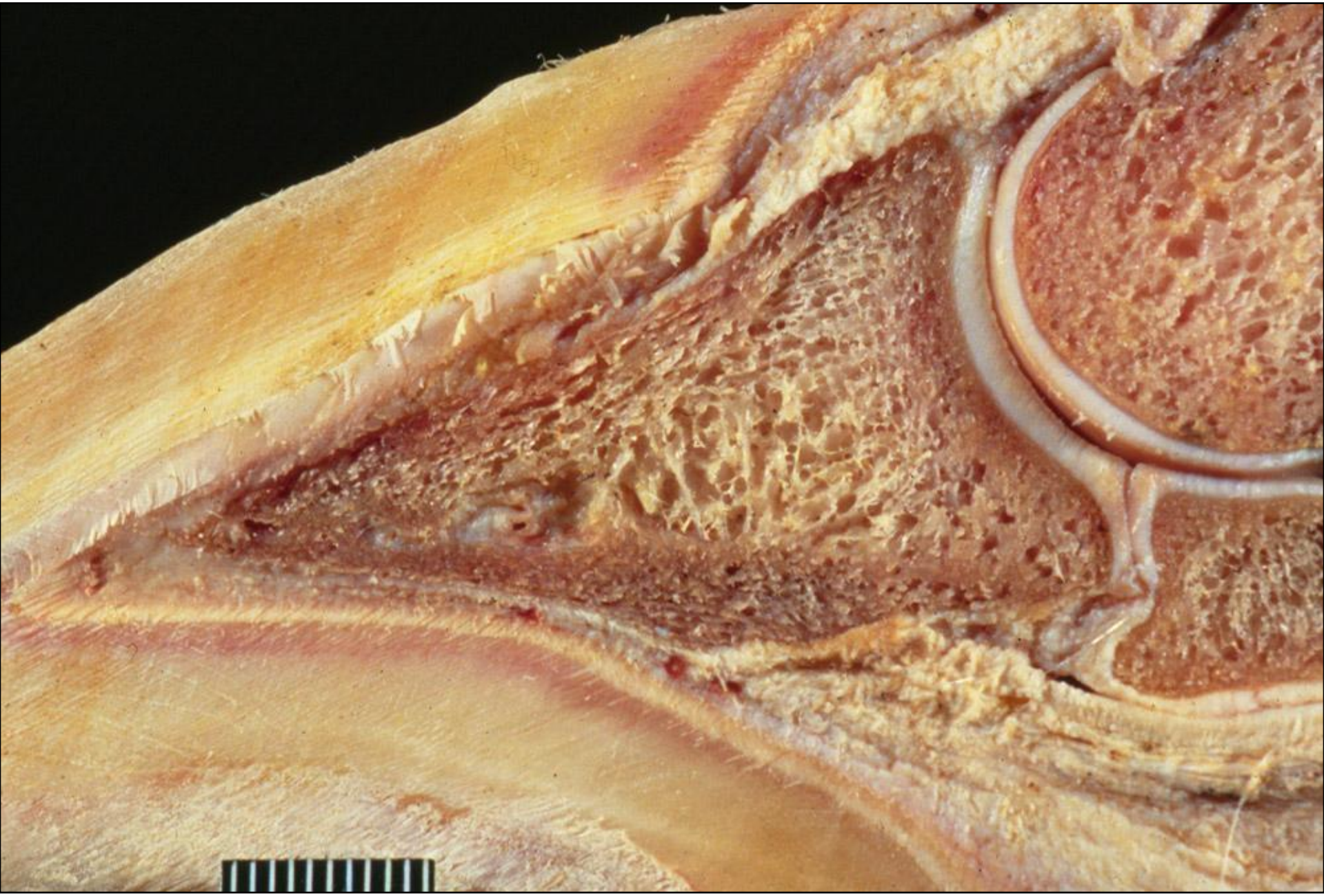
Remodeling in response to abnormal use and systemic disease

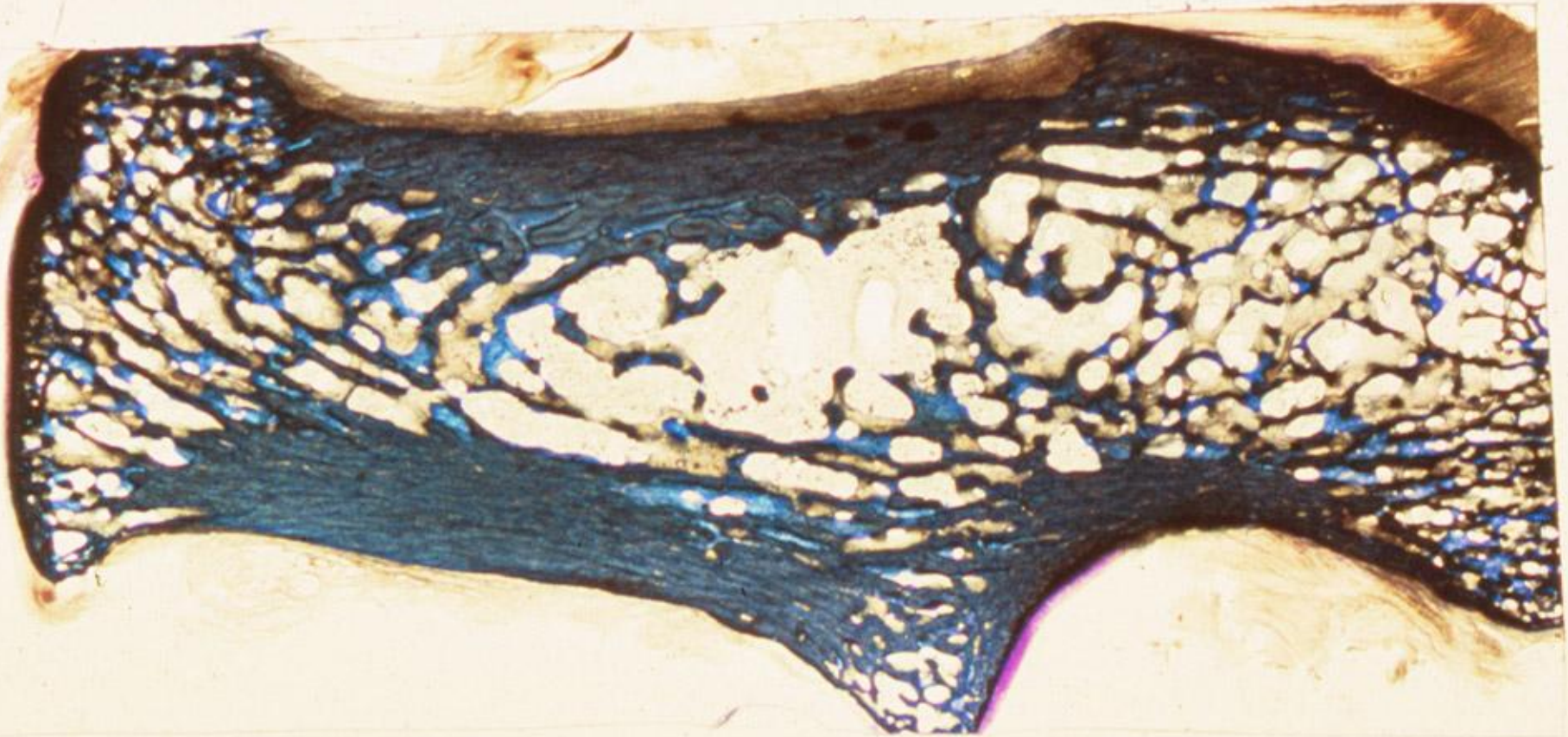
- Decreased mechanical use will activate remodeling and decrease vigor of bone formation – osteopenia
- Increased mechanical use will suppress activation of remodeling - osteosclerosis
- Effects of PTH and CT on bone turnover
- Cachexia and ability to form bone
- Systemic acceleratory phenomenon

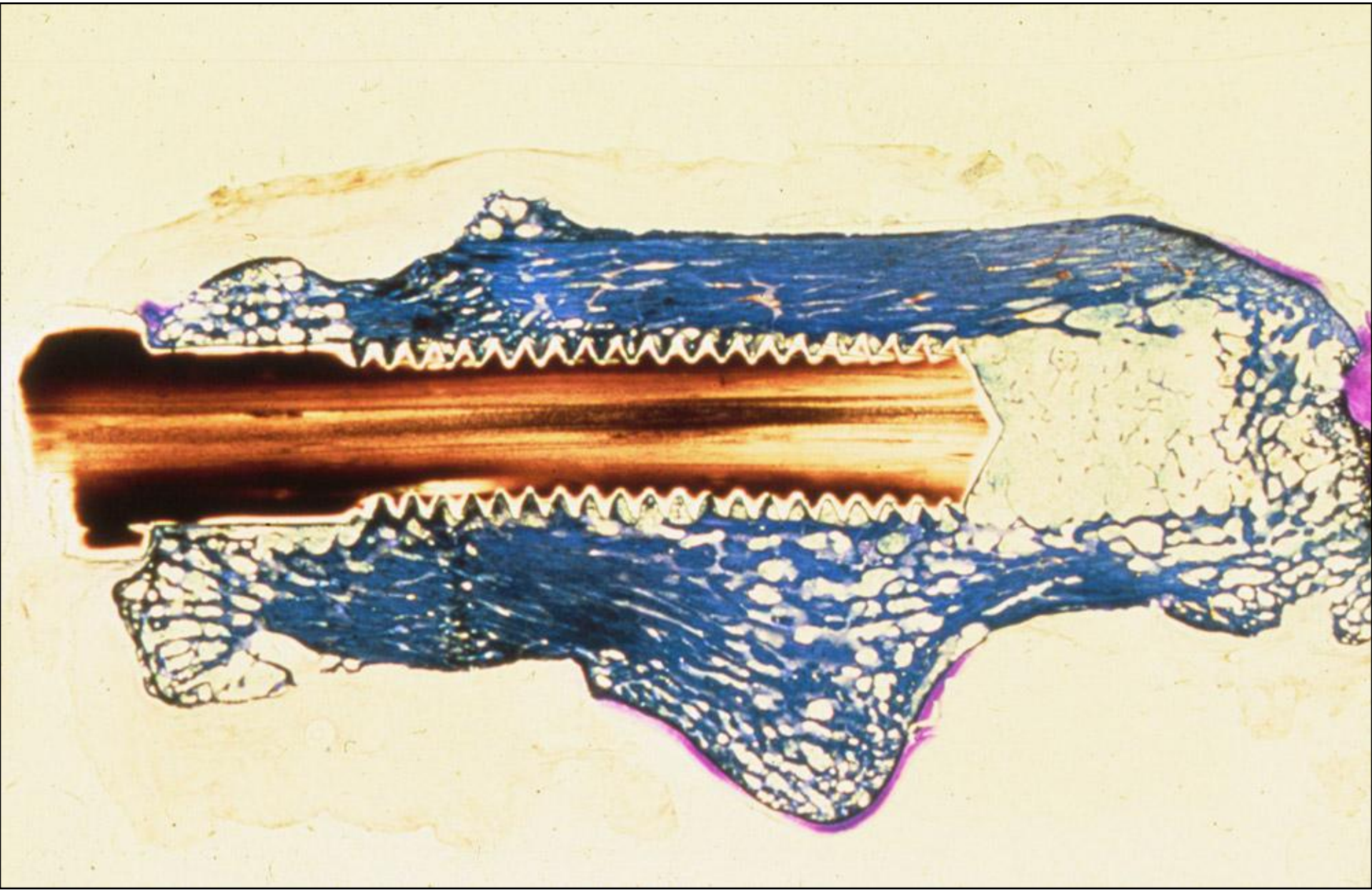


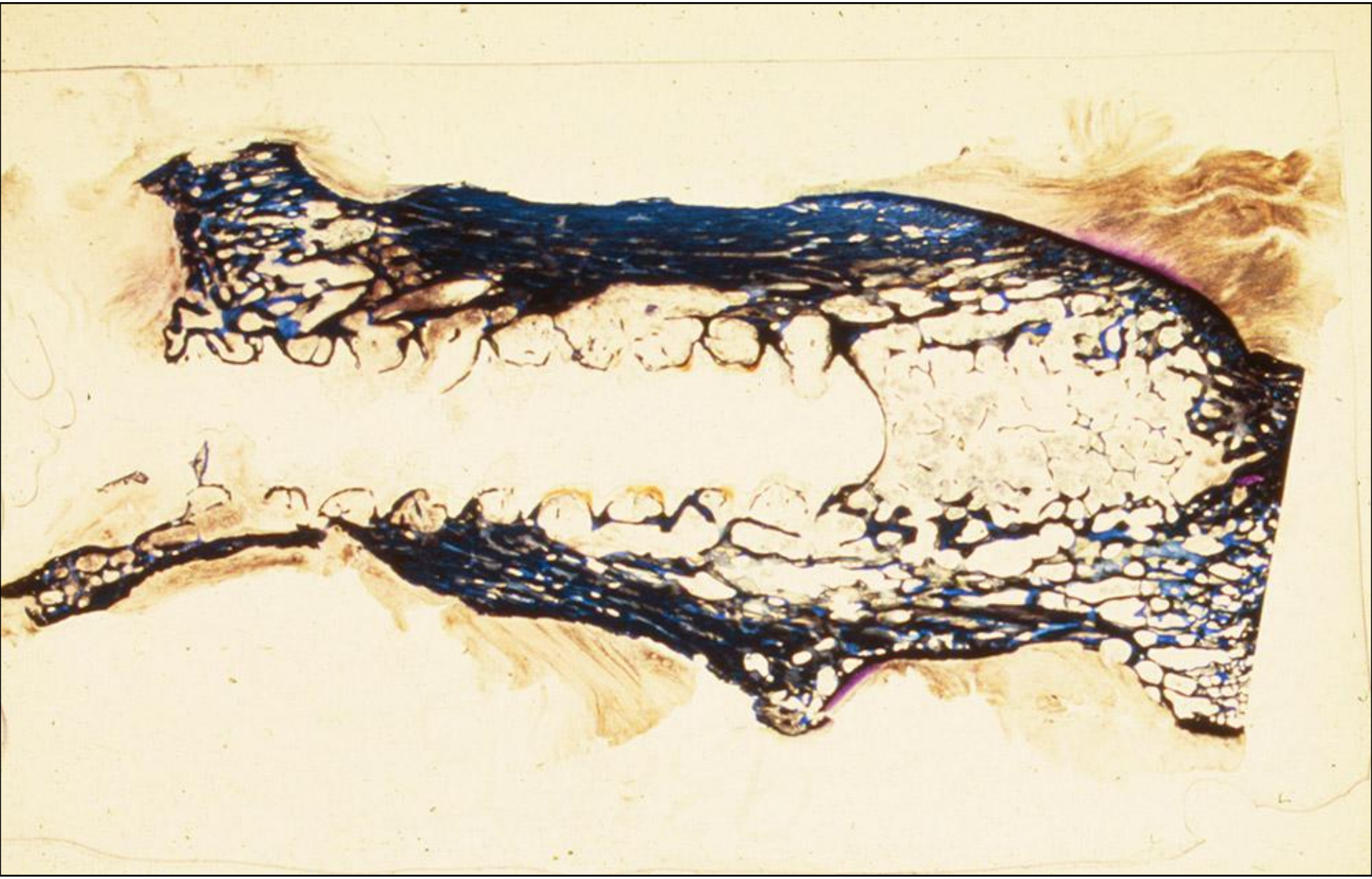












Forty % bone loss with one year disuse mostly at the endocortical and periosteal surfaces

RISEDRONATE ON LONG-TERM DISUSE OSTEOPOROSIS

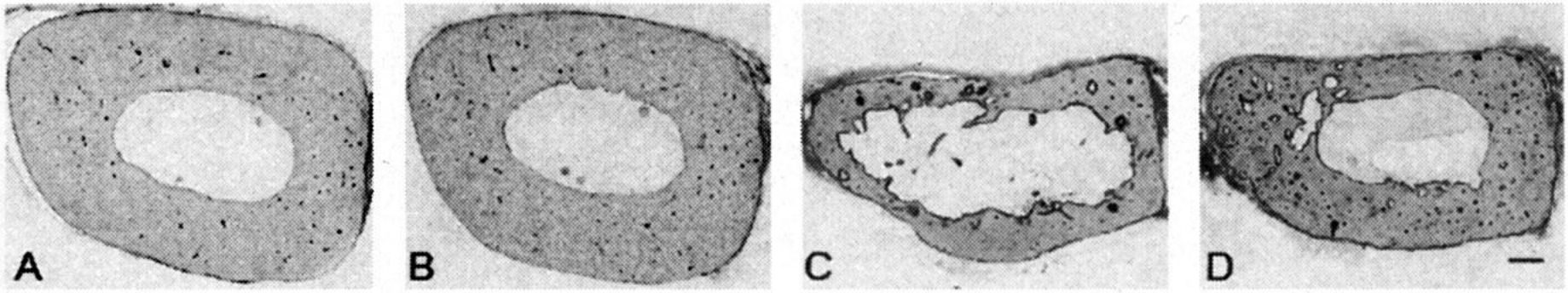
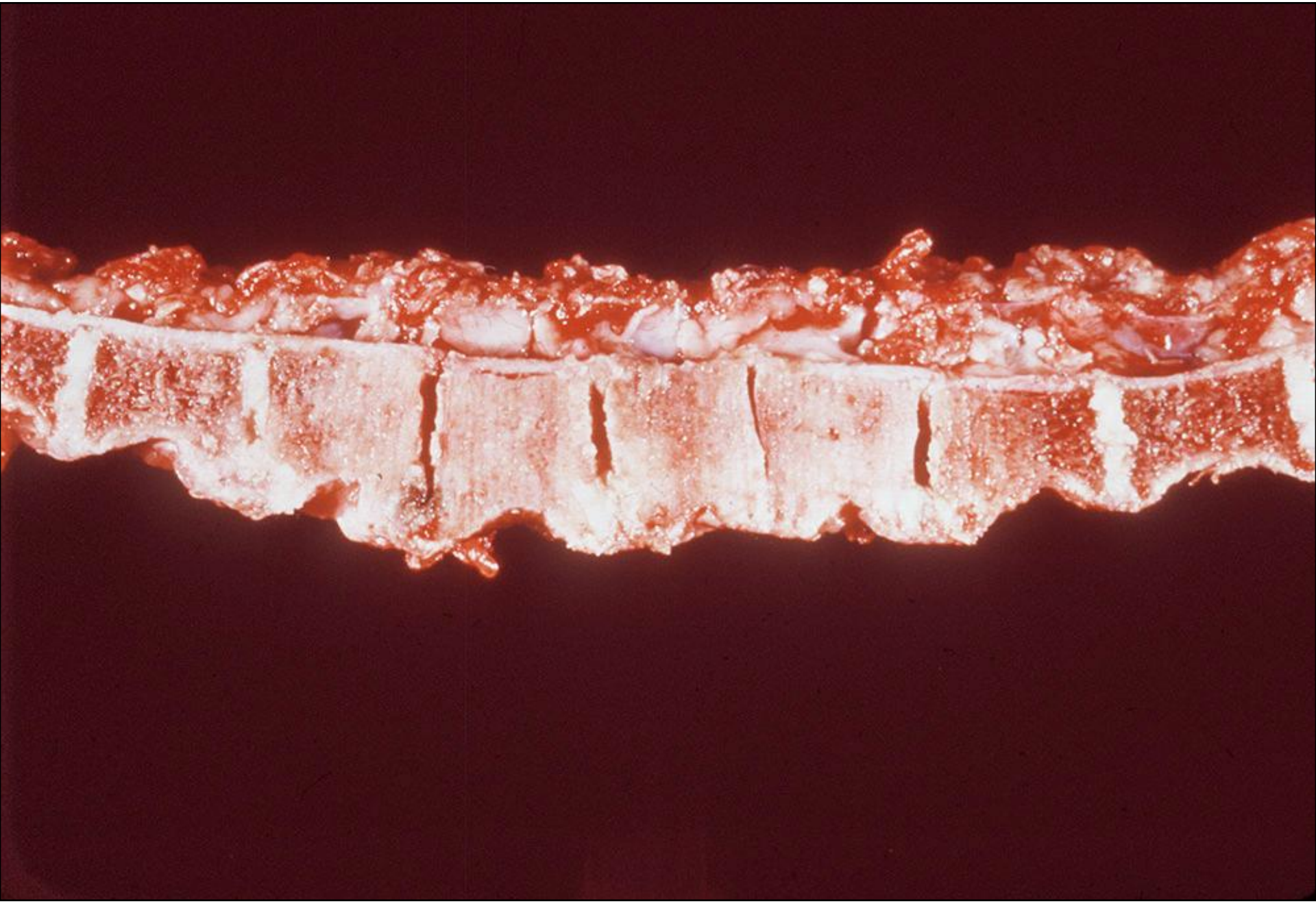


FIG. 1. Photomicrographs of metacarpal midshaft cross sections showing (A) control + vehicle, (B) control + RIS, (C) IM + vehicle, and (D) IM + RIS. (C) IM bone showed a smaller subperiosteal area, larger marrow cavity, thinner cortex, and elevated porosity compared with (A) control bone. (D) RIS-treated IM bone showed evidence of significant bone loss, but to a marked lesser degree than IM alone. Bar = 500 μm.

Remodeling in response to abnormal use and systemic disease

- Decreased mechanical use will activate remodeling and decrease vigor of bone formation – osteopenia
- Increased mechanical use will suppress activation of remodeling - osteosclerosis
- Effects of PTH and CT on bone turnover
- Cachexia and ability to form bone
- Systemic acceleratory phenomenon



Modeling in response to structural damage and abnormal use (Wolff's Law)

Bone will model to accommodate mechanical use

- Formation with compression
- Resorption with tension
- Alignment of trabeculae along lines of stress

Detection of Altered Mechanical Use

- Streaming potentials in the canalicular system
- Piezoelectric forces derived from deformation of the collagen “crystal” lattice
- Stretch receptors on osteoblasts
- Compression/tension on nerves and blood vessels

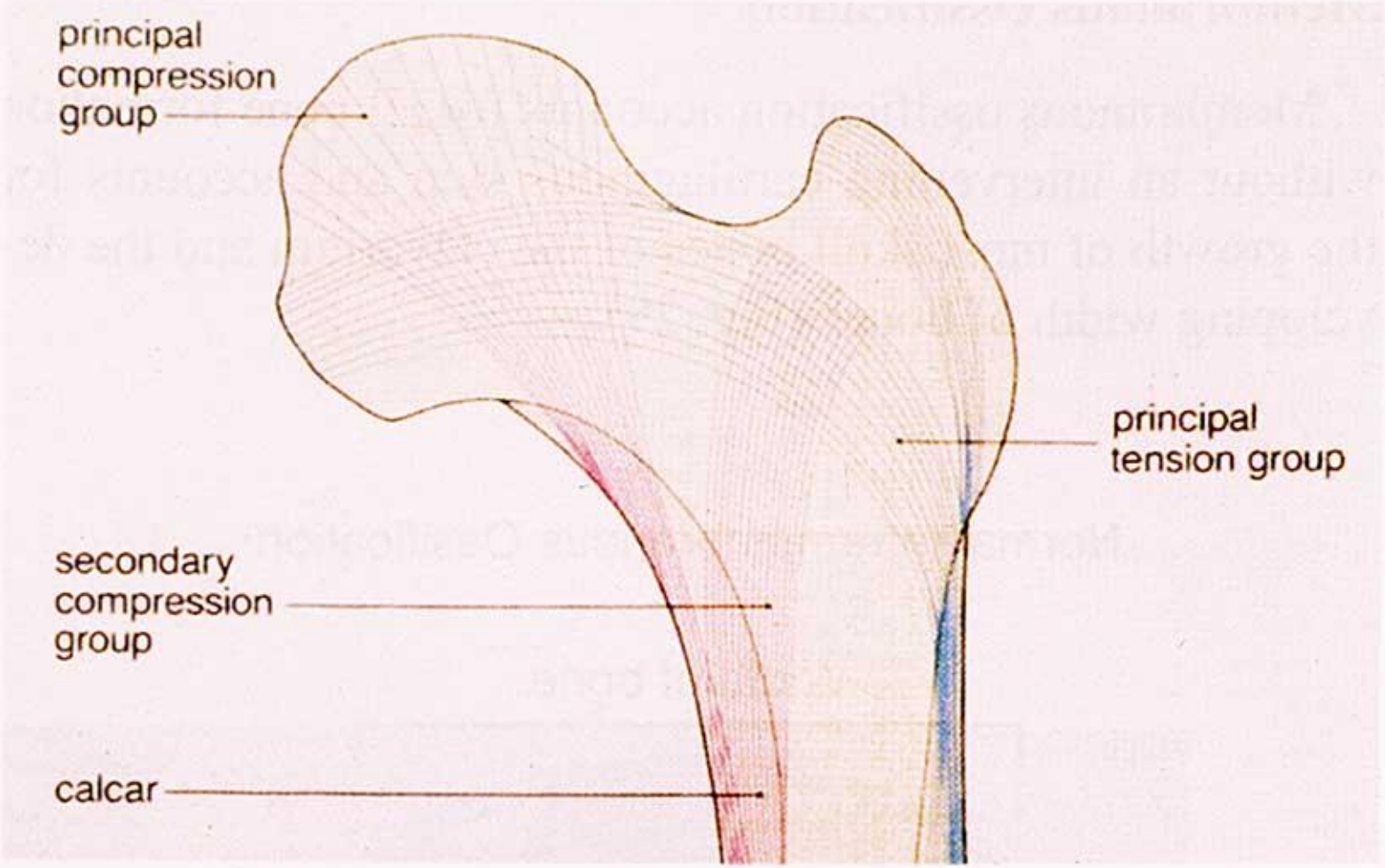
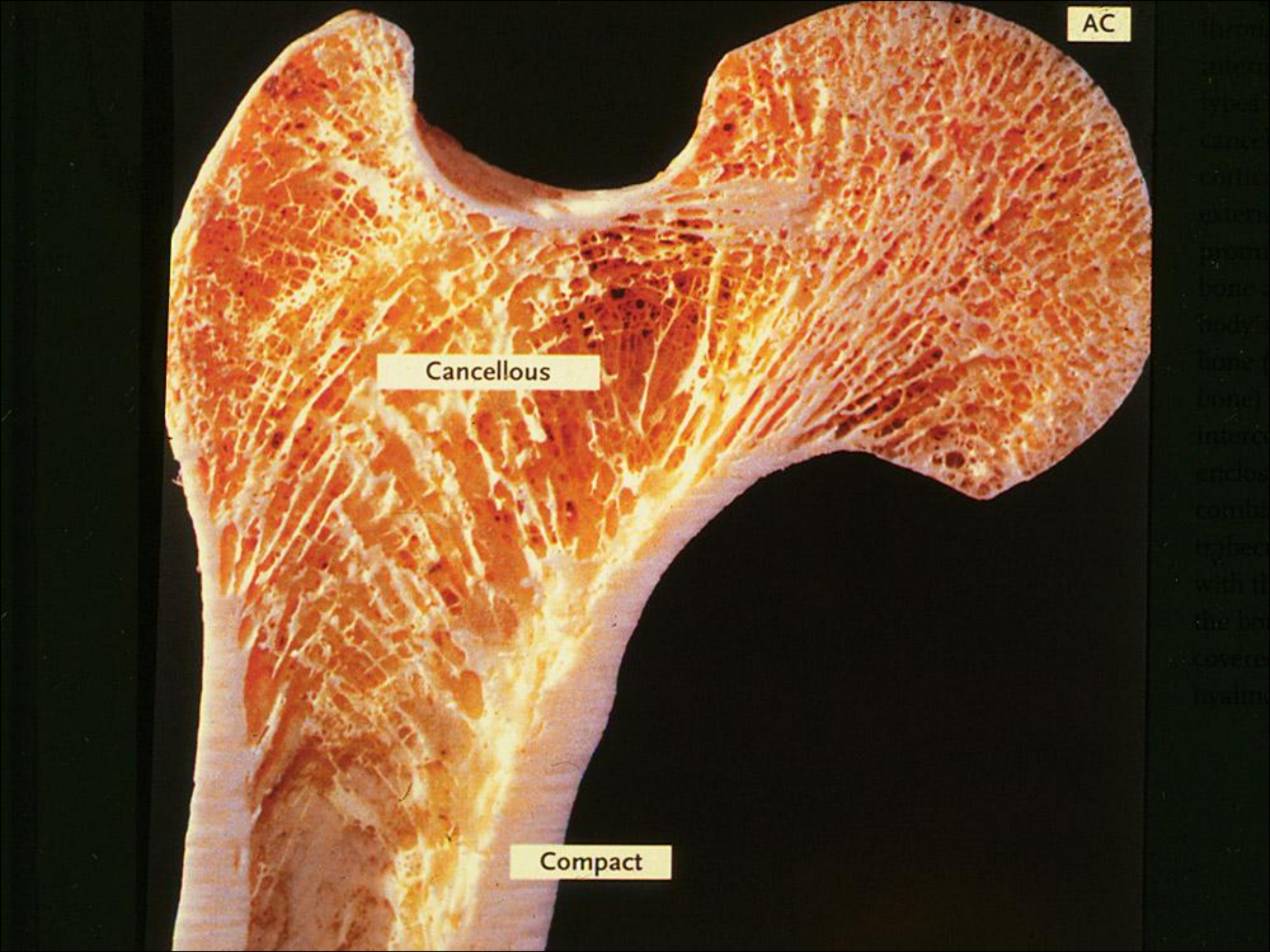
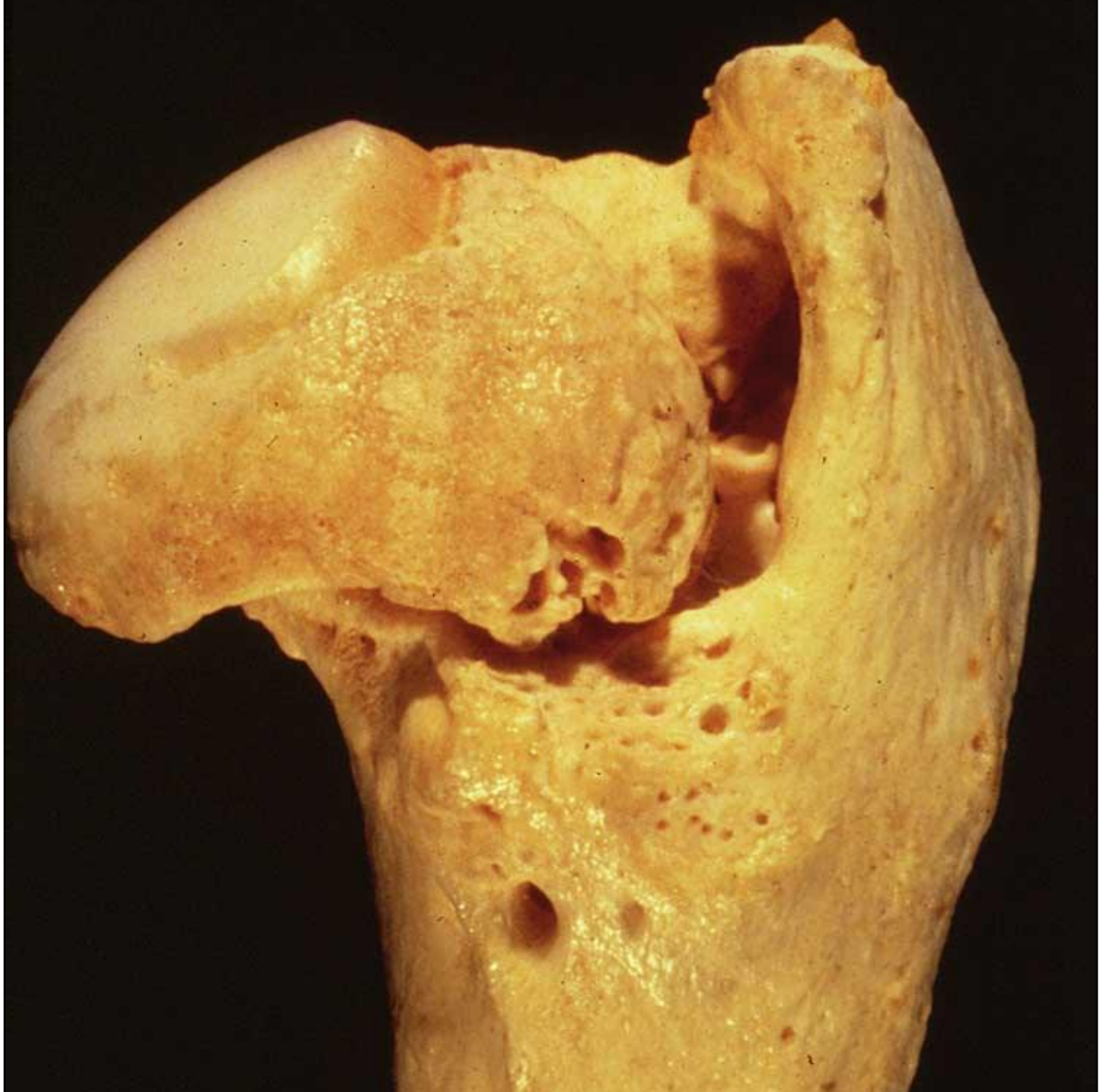
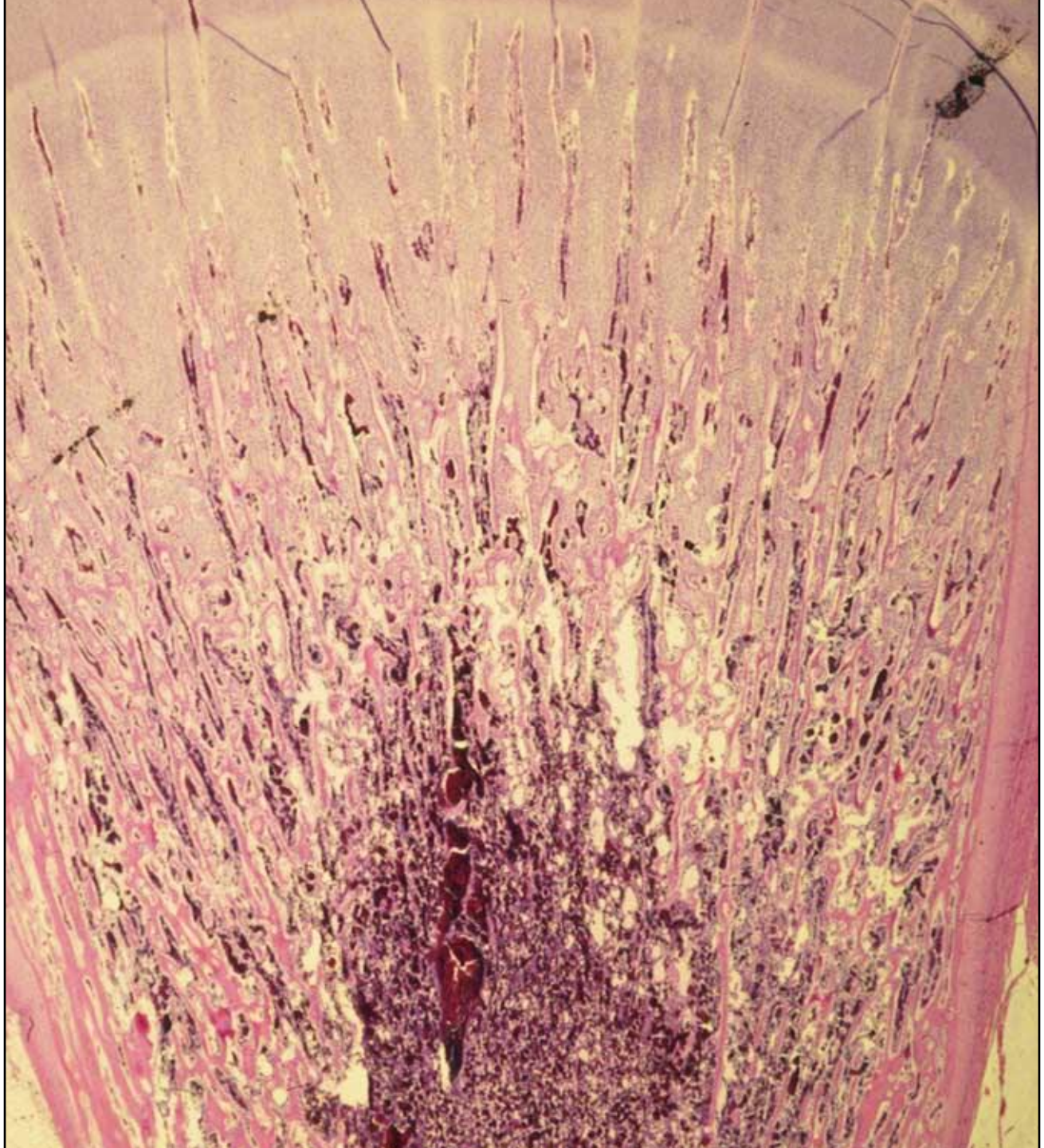
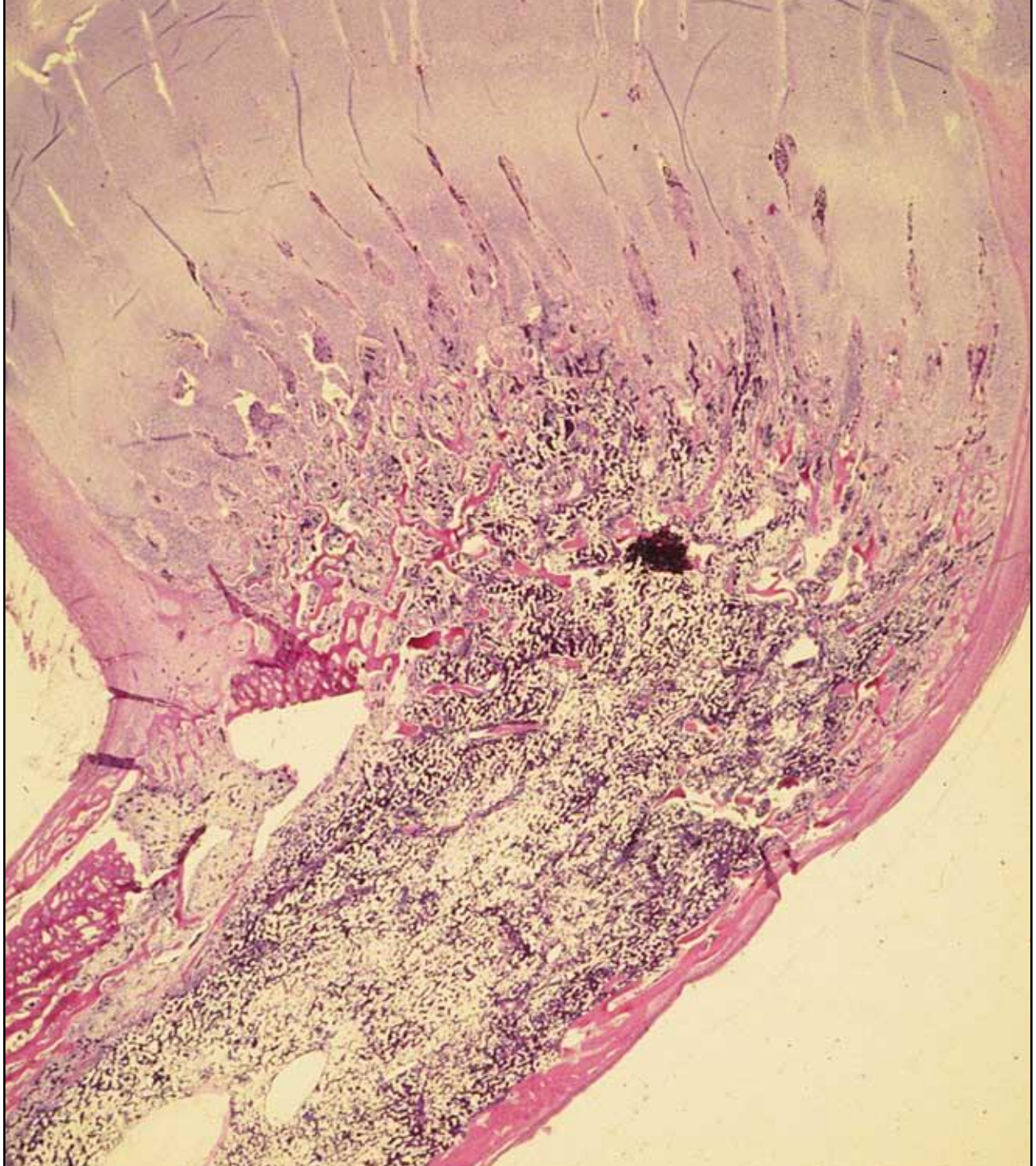


FIG. 31. Bone modeling: the trabecular structure of the proximal femur is a composition of arcades of cancellous bone that "model" or "shape" the internal architecture of bone along compressive and tensile stresses produced during weight bearing.


AC
Cancellous
Compact





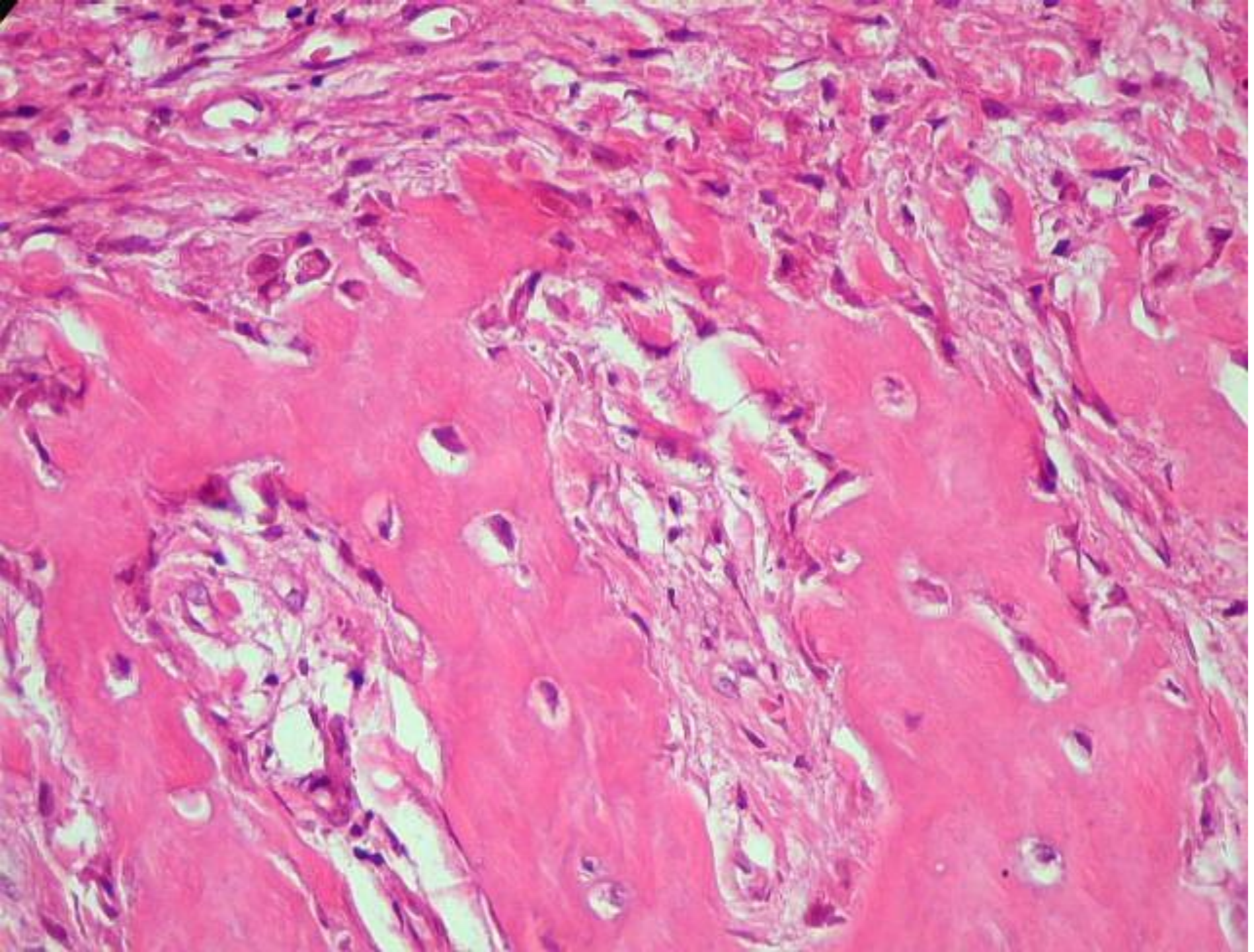


General Reactions of Bone to Injury (Modified from Palmer)

- Disruption of endochondral ossification .
- Modeling in response to structural damage and abnormal use (Wolff's Law).
- Remodeling in response to abnormal use and systemic disease.
- Repair bone/rapidly deposited bone is woven rather than lamellar
- Periosteum responds to injury usually by formation of woven bone.

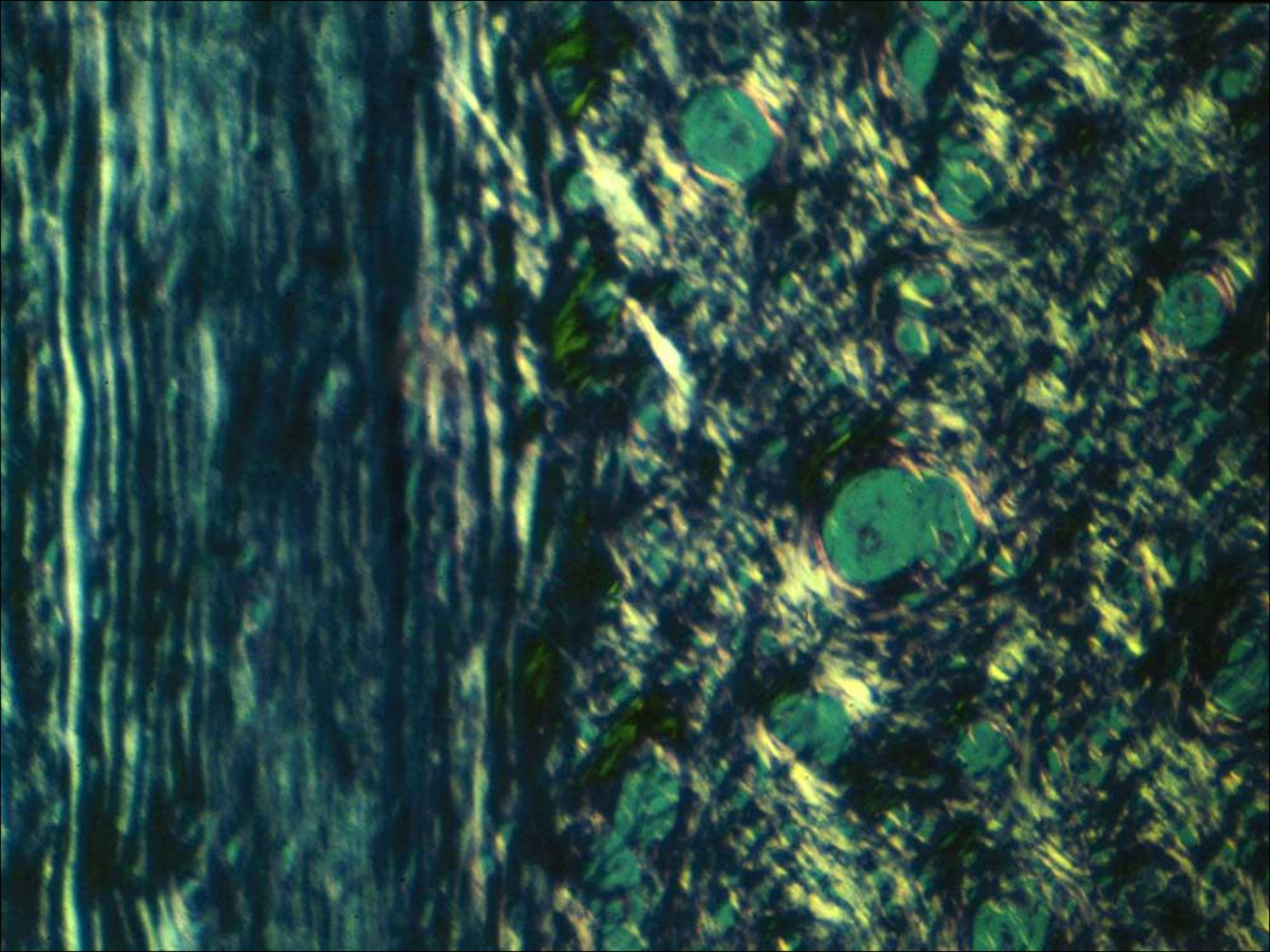
Repair bone/rapidly deposited bone is woven rather than lamellar

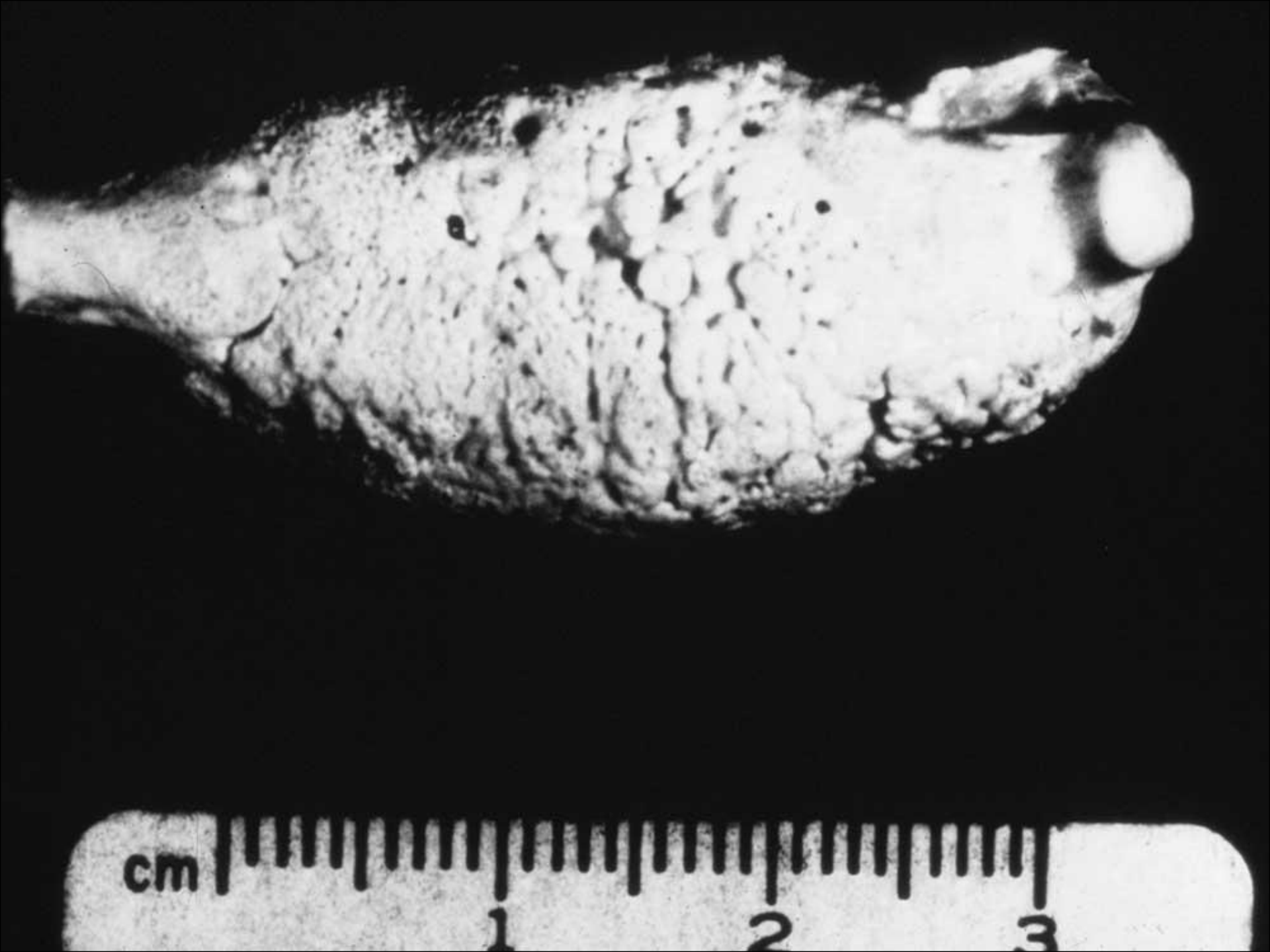
- Woven bone has larger haphazardly arranged osteocytes and haphazardly arranged collagen fibers
- Mineralizes more rapidly than lamellar bone



Periosteum responds to injury usually by formation of woven bone

- Reactive periosteal bone should have gradual transition between fibrous and osteogenic layers of periosteum




cm
1
2
3

Major Bone Disease Categories (from McCarthy and Frassica)

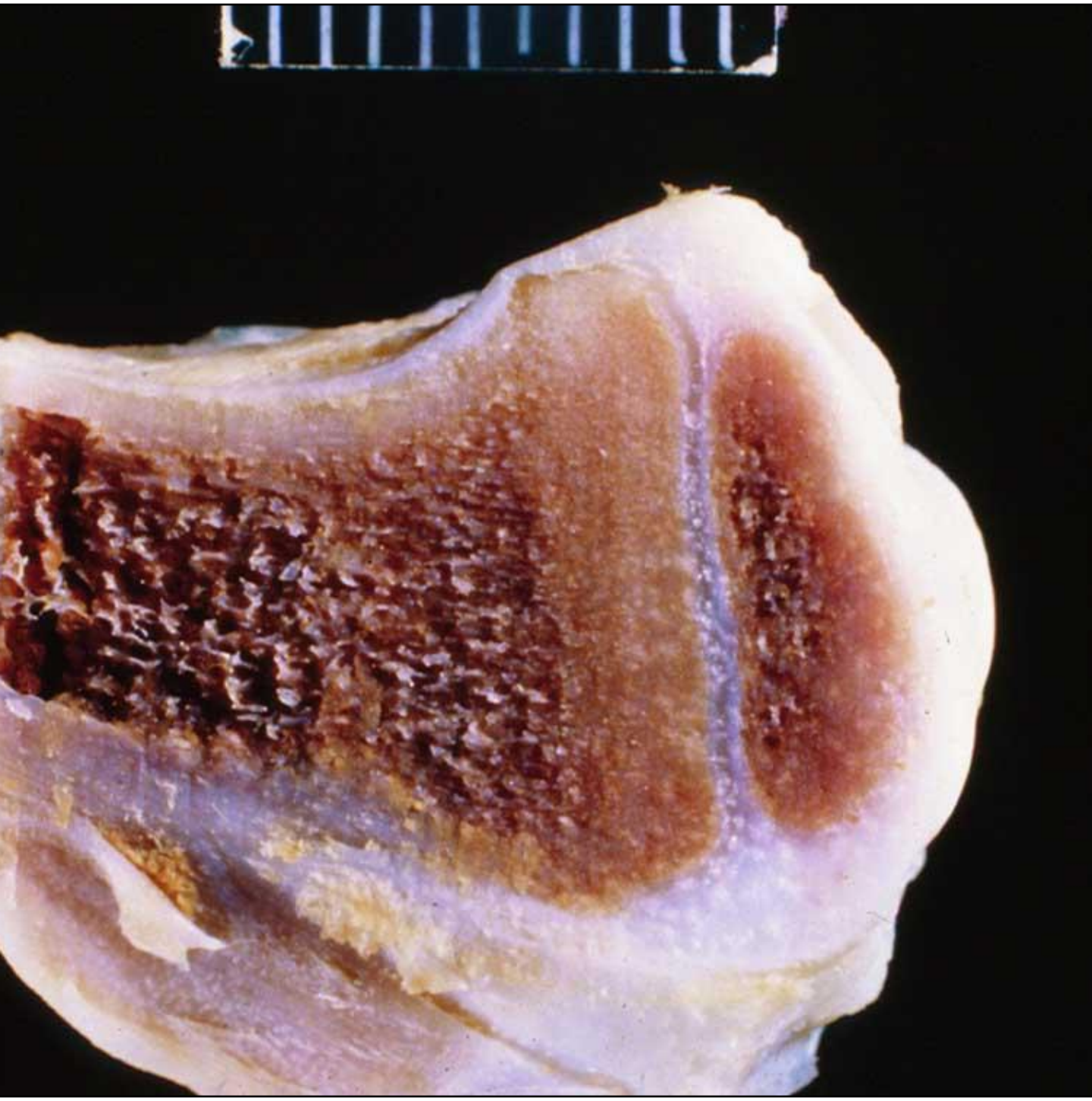
1. Congenital
2. Metabolic
3. Traumatic
4. Circulatory
5. Neoplastic
6. Infectious
7. Responses to systemic disease

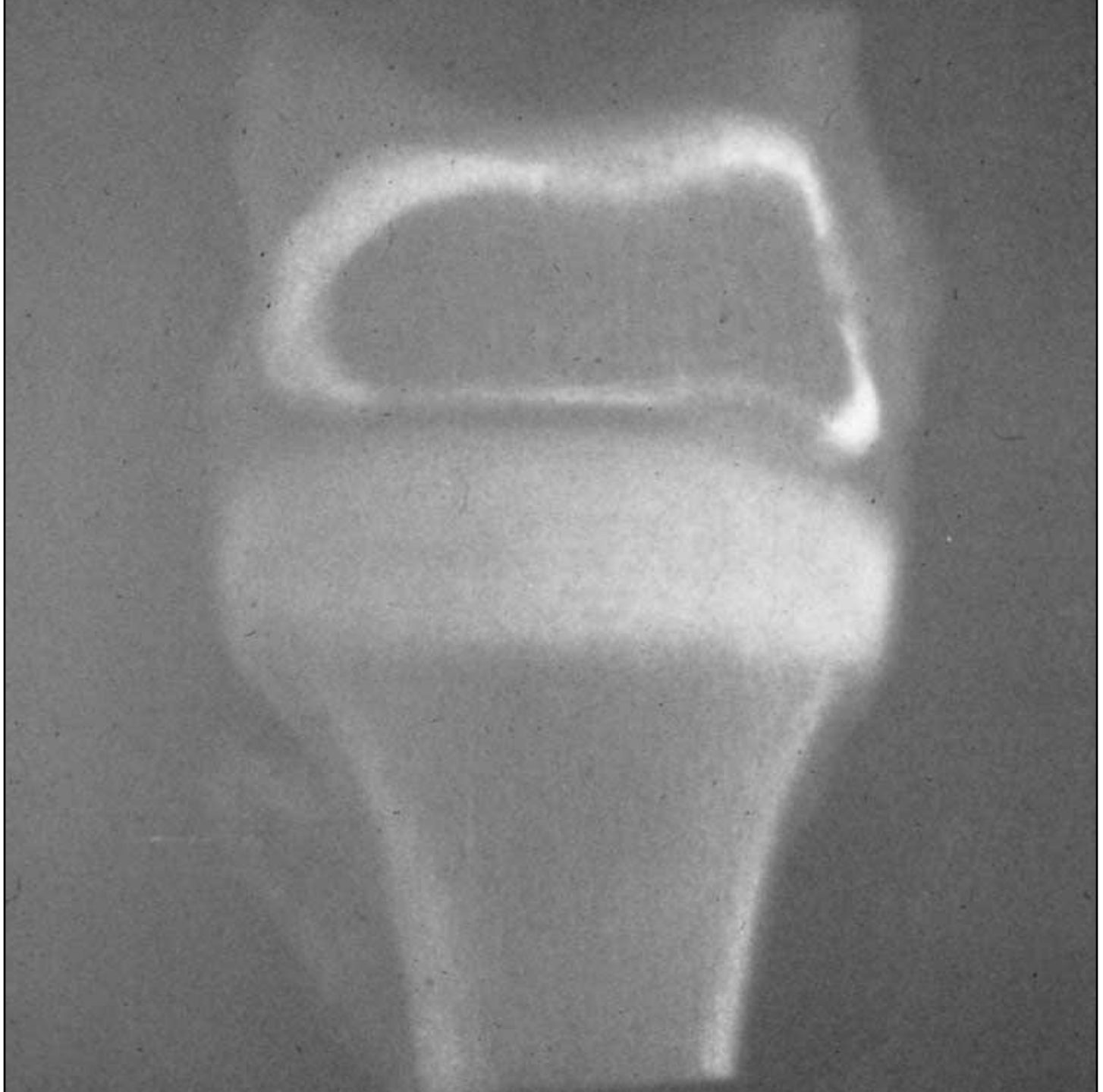
Disruption of endochondral ossification

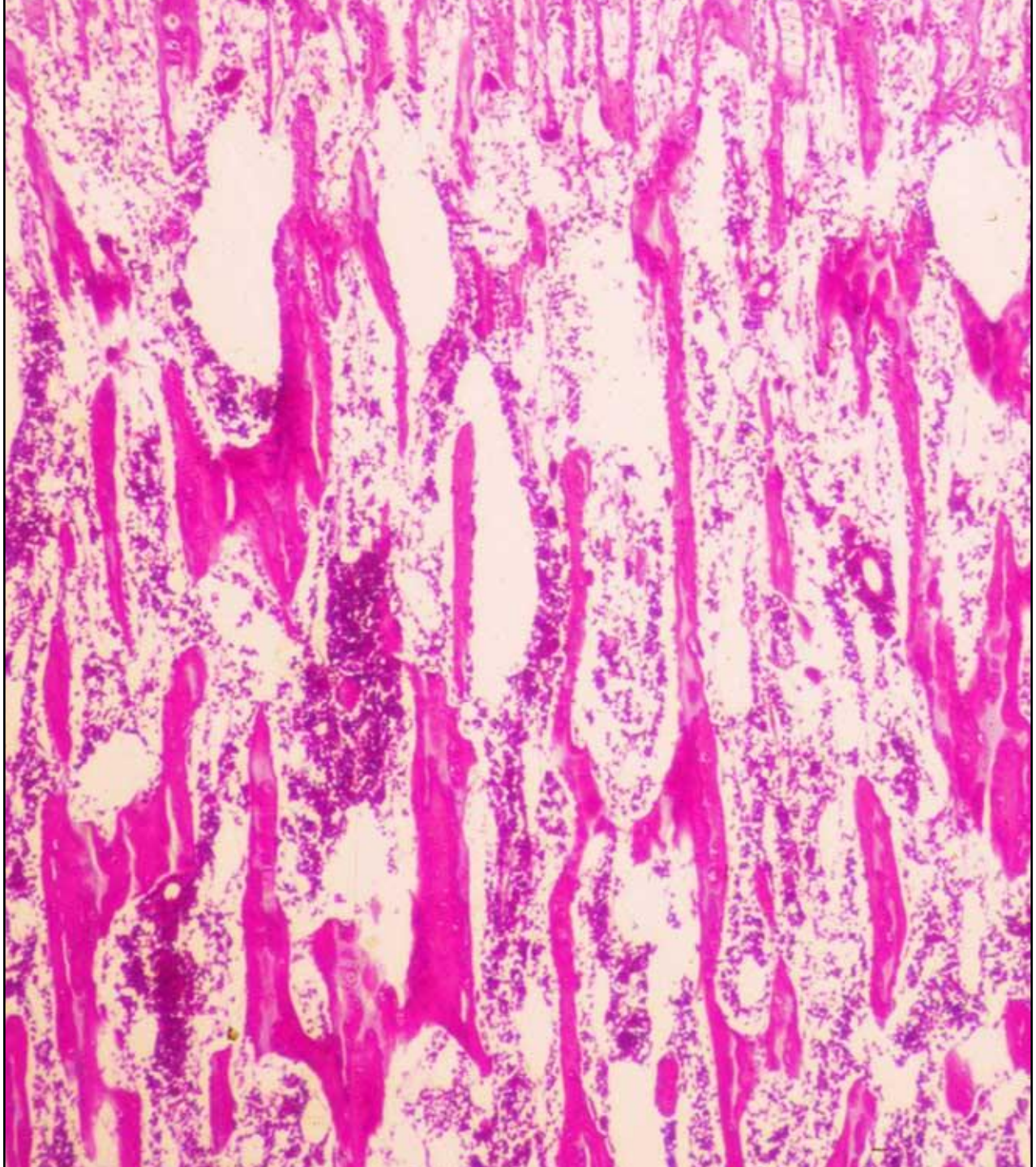
- Altered trabecular bone formation
  - Growth retardation lattice
  - Growth arrest lines
  - Retained growth cartilage
- Cavitation/cyst formation
- Altered bone length/angular deformity

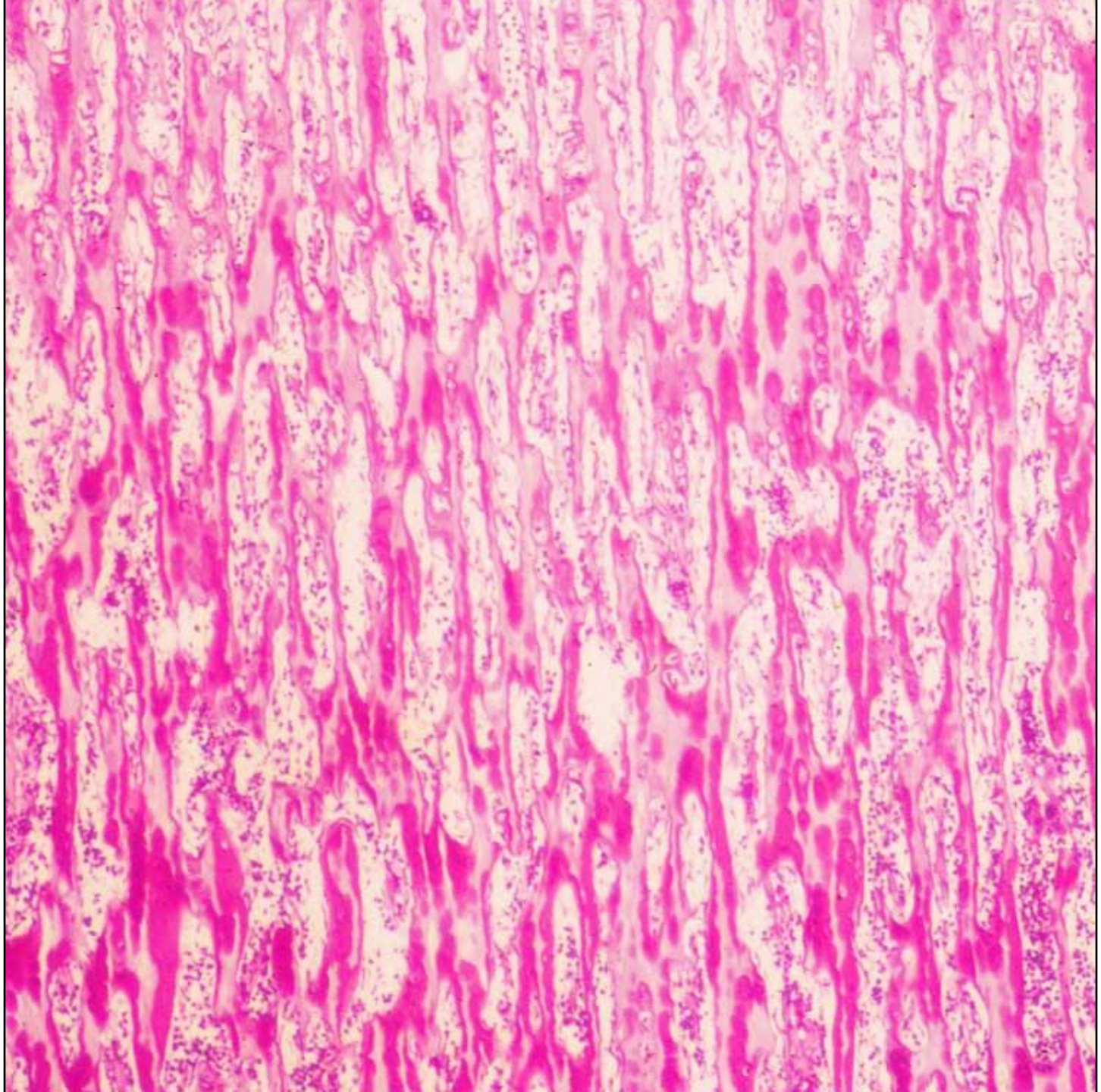
Growth Retardation Lattice

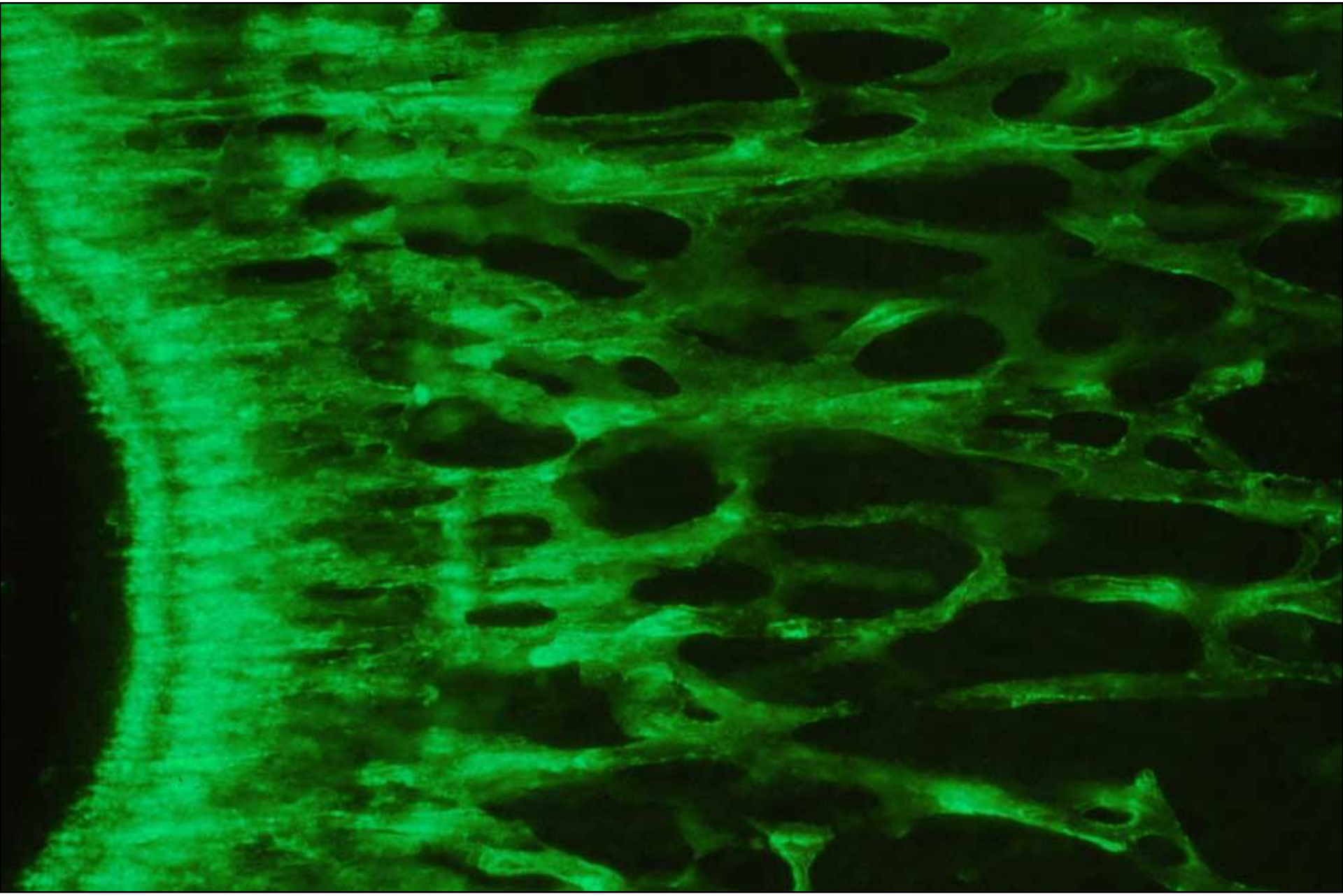
- Failure of osteoclastic modeling at a site of endochondral ossification
- Retention of un-modeled primary trabeculae

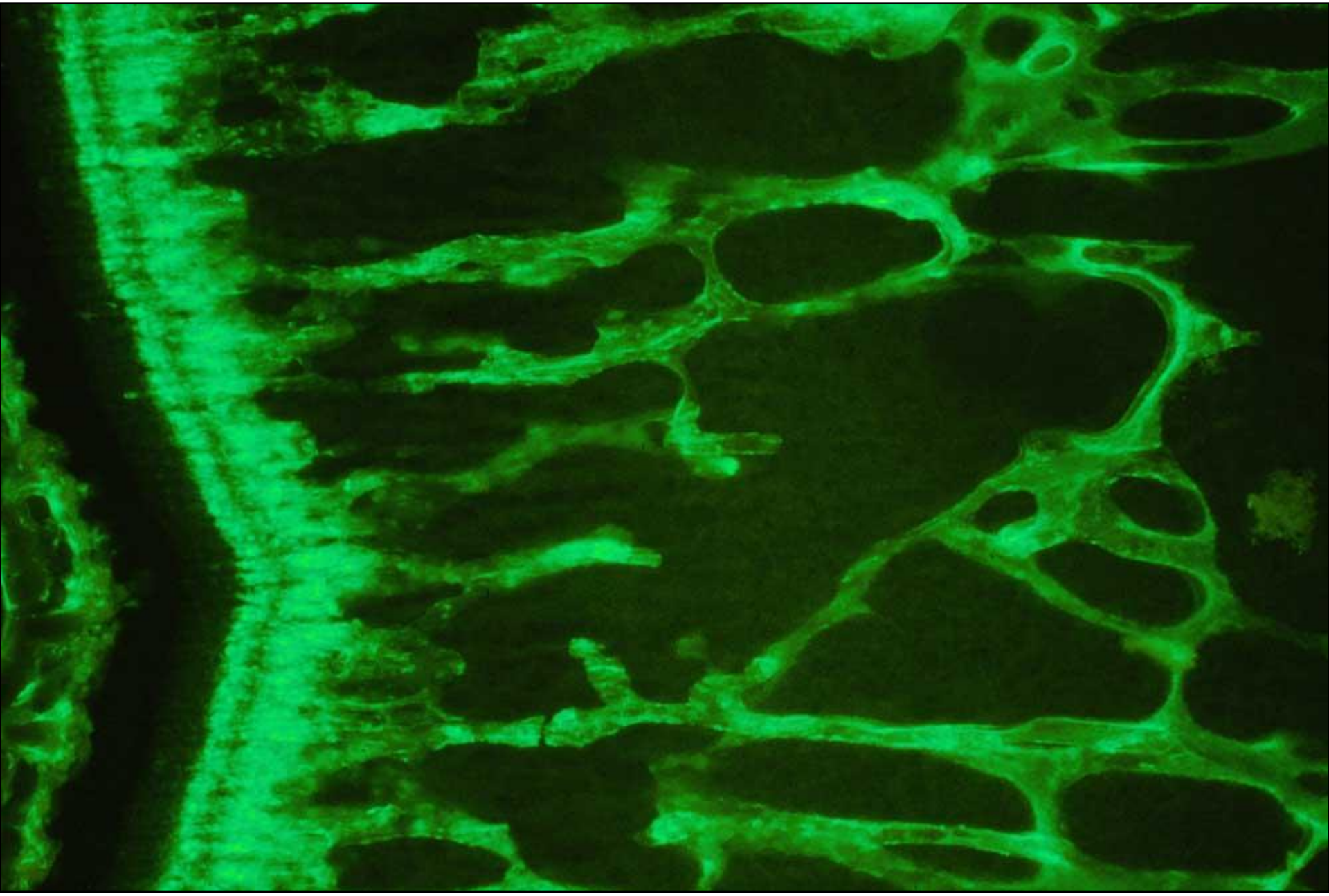


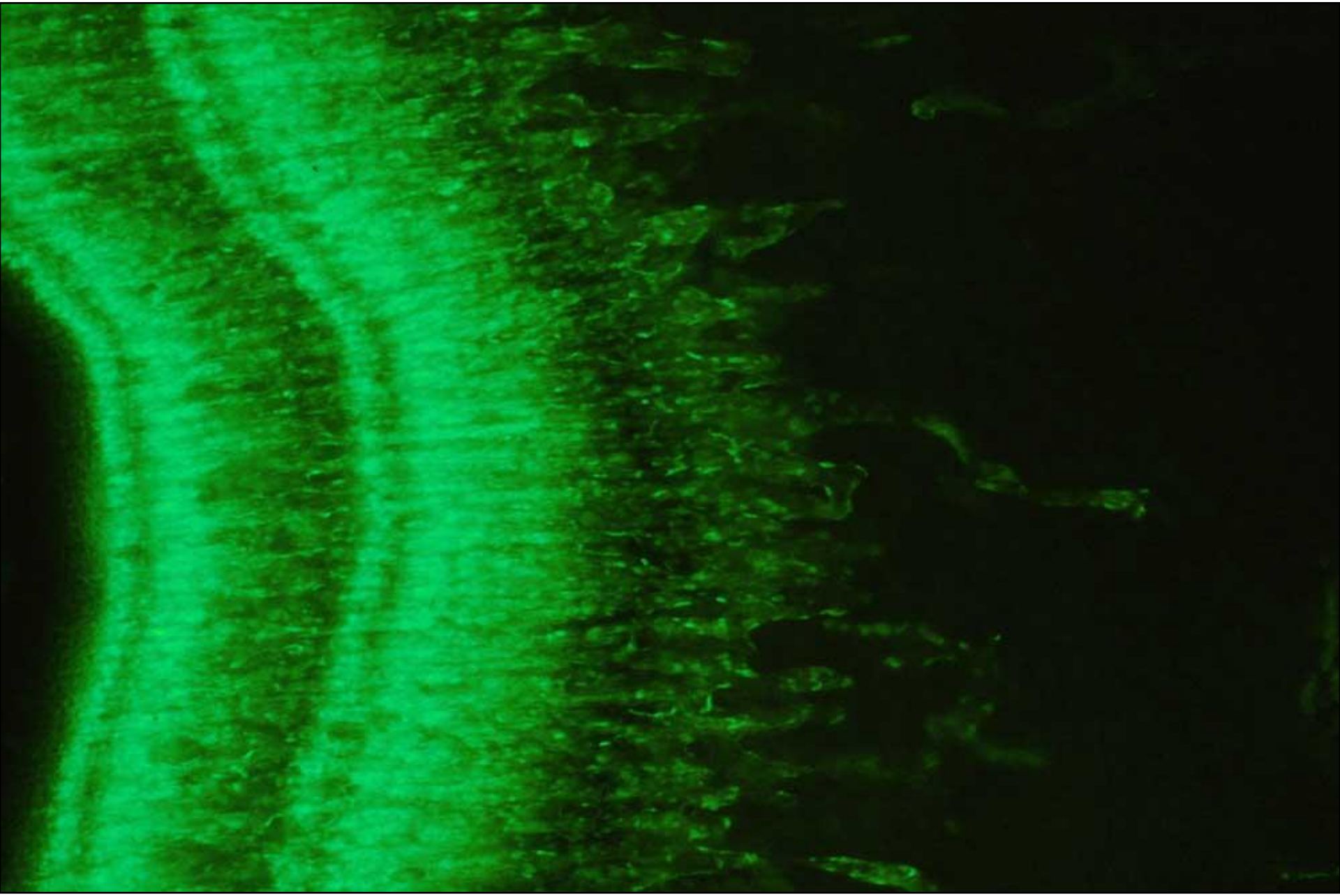


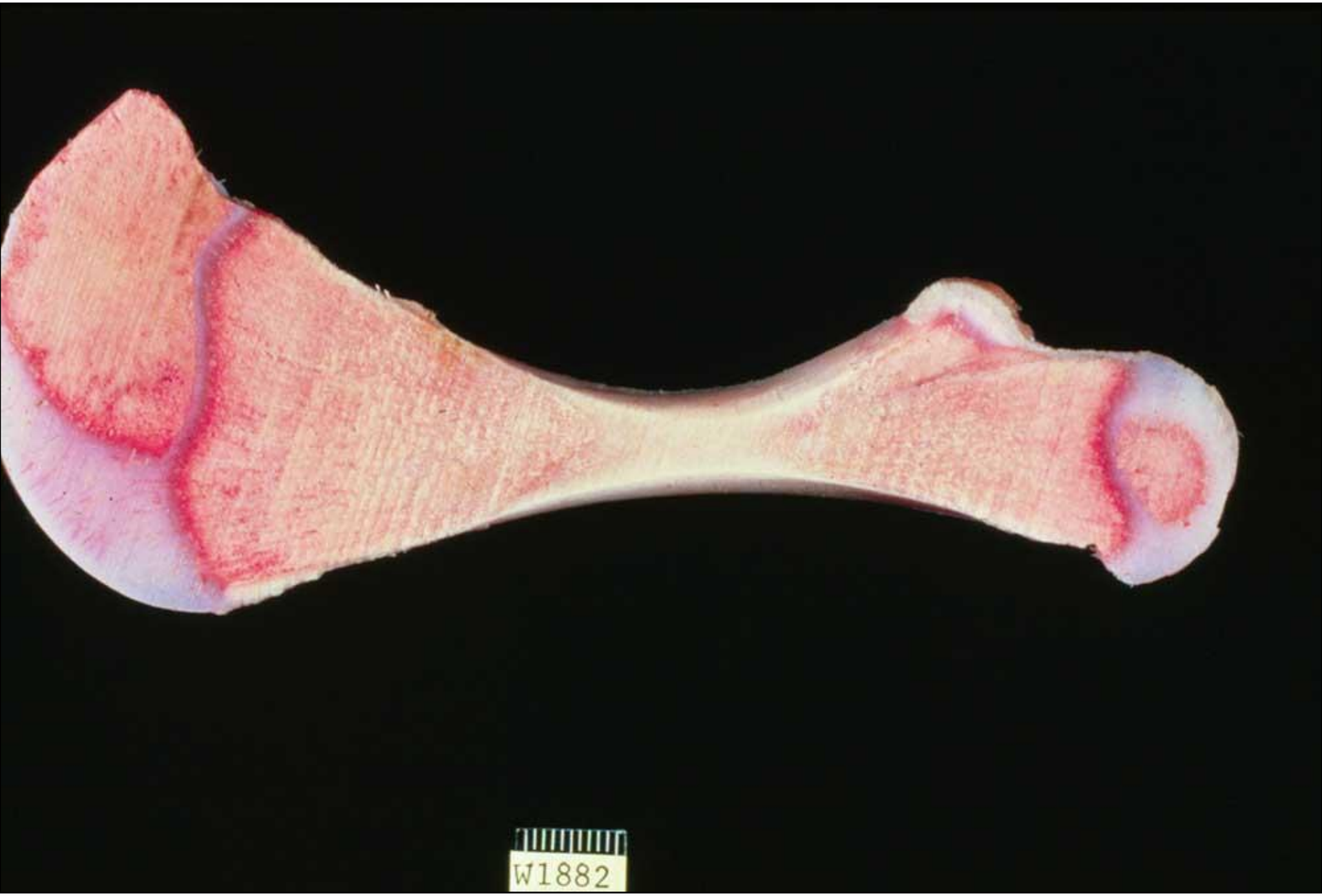








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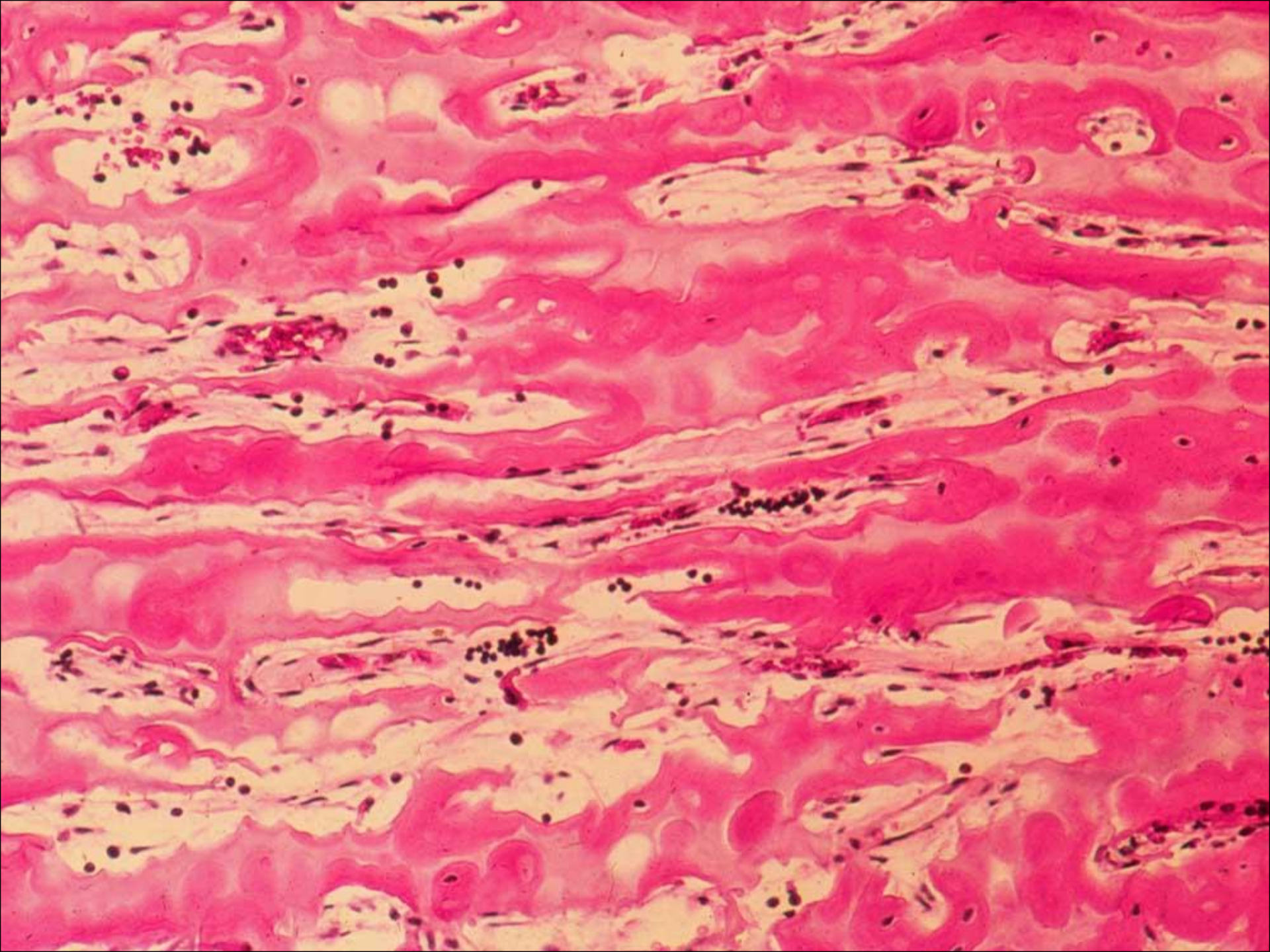
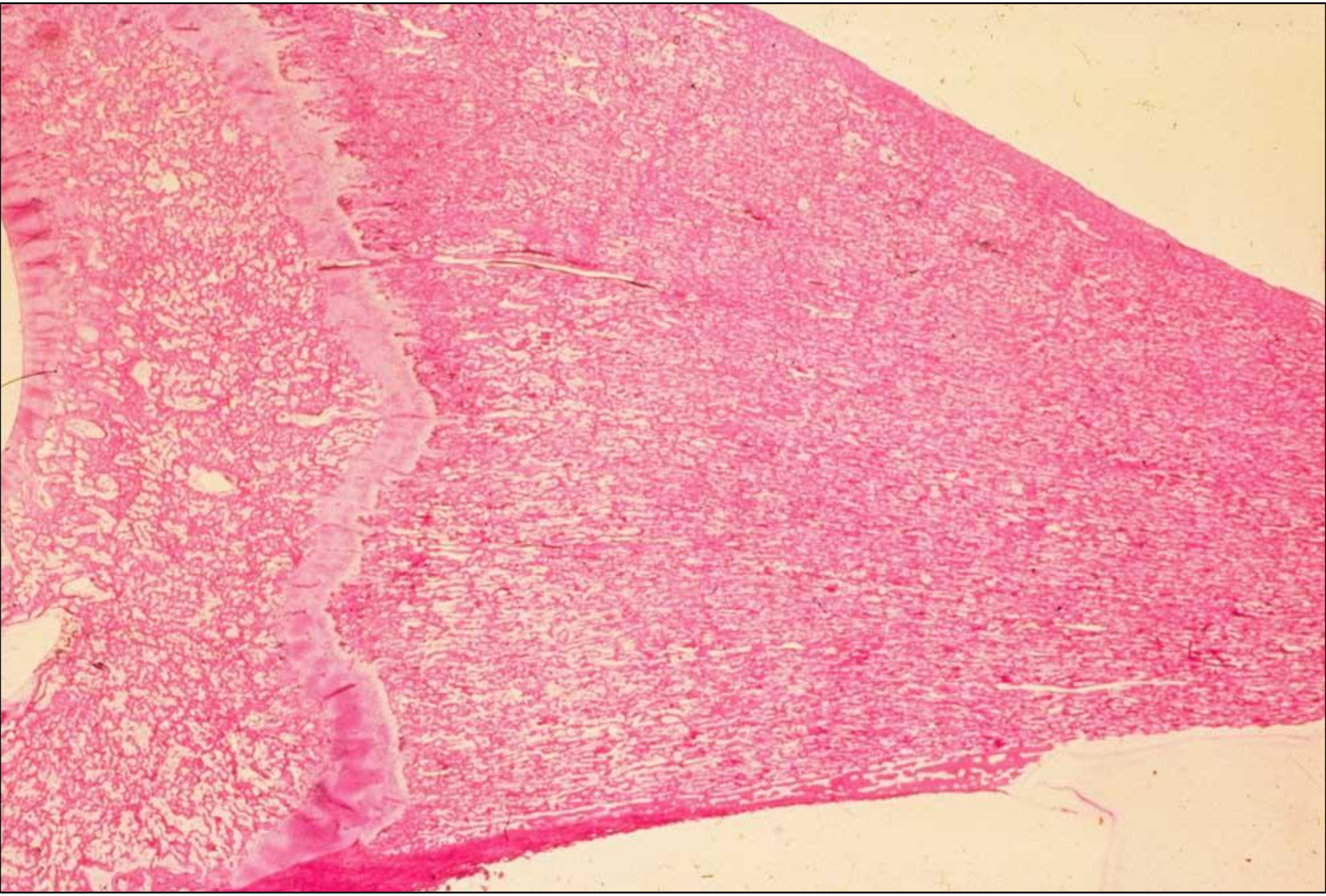
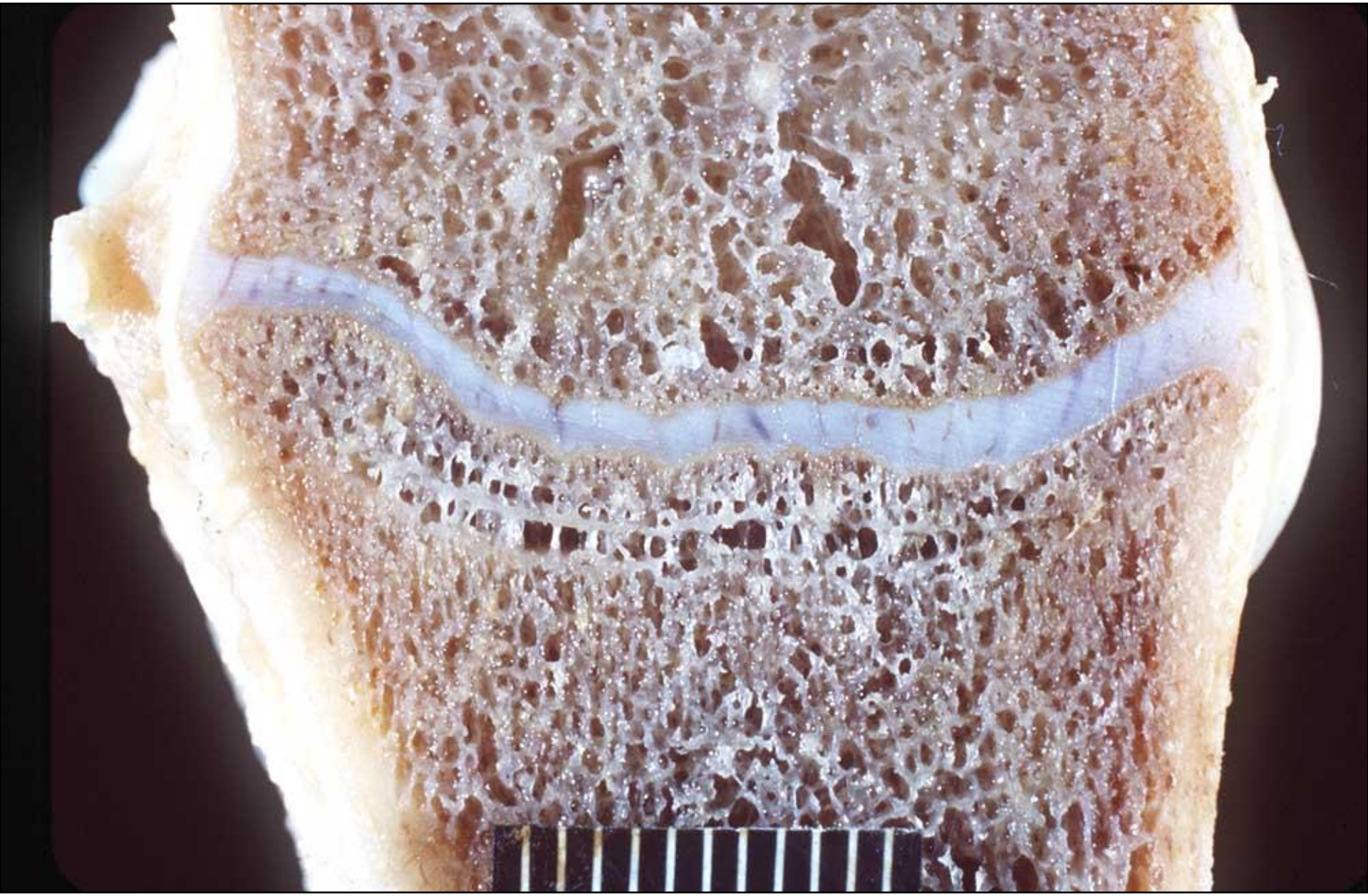
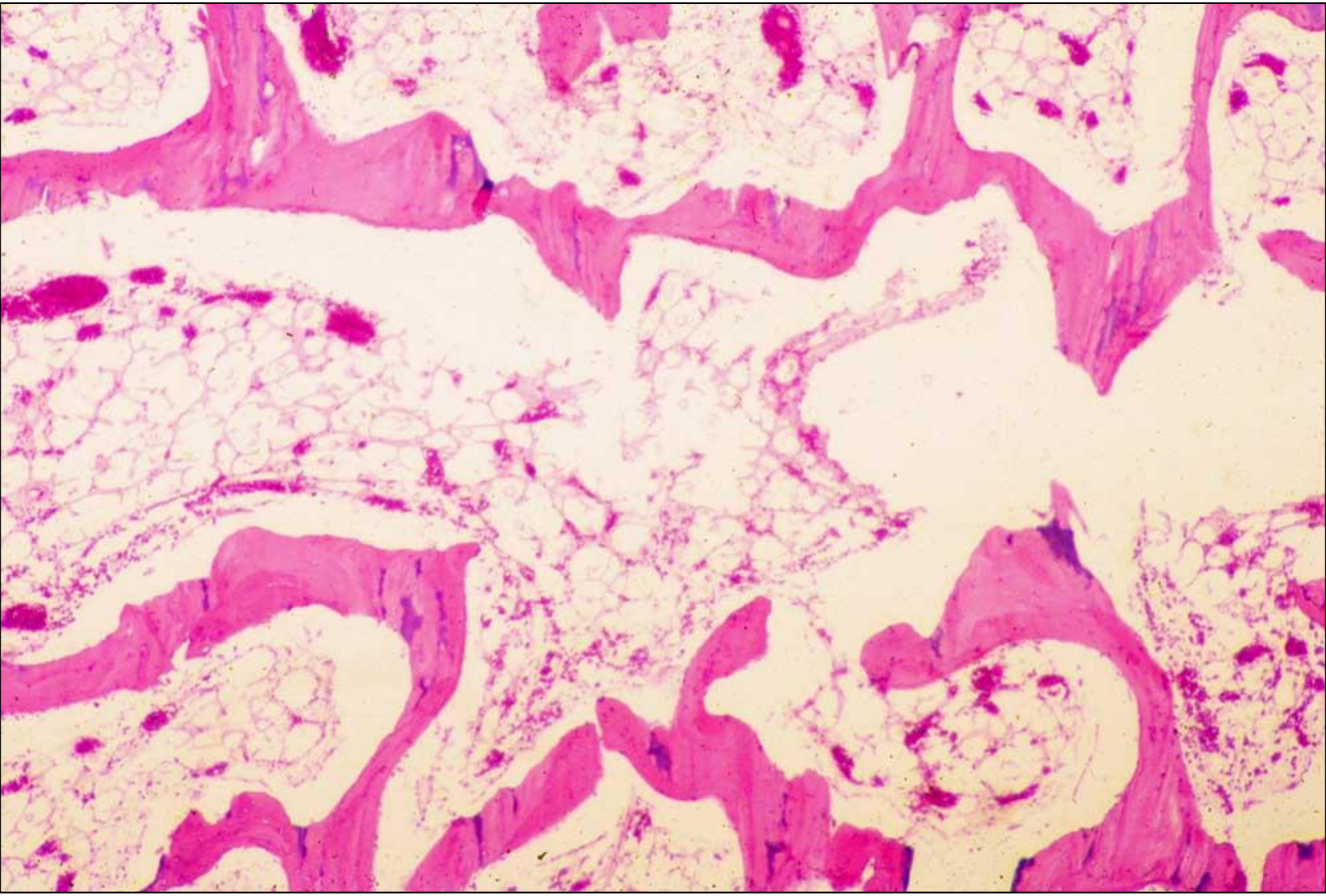




Growth Arrest Line

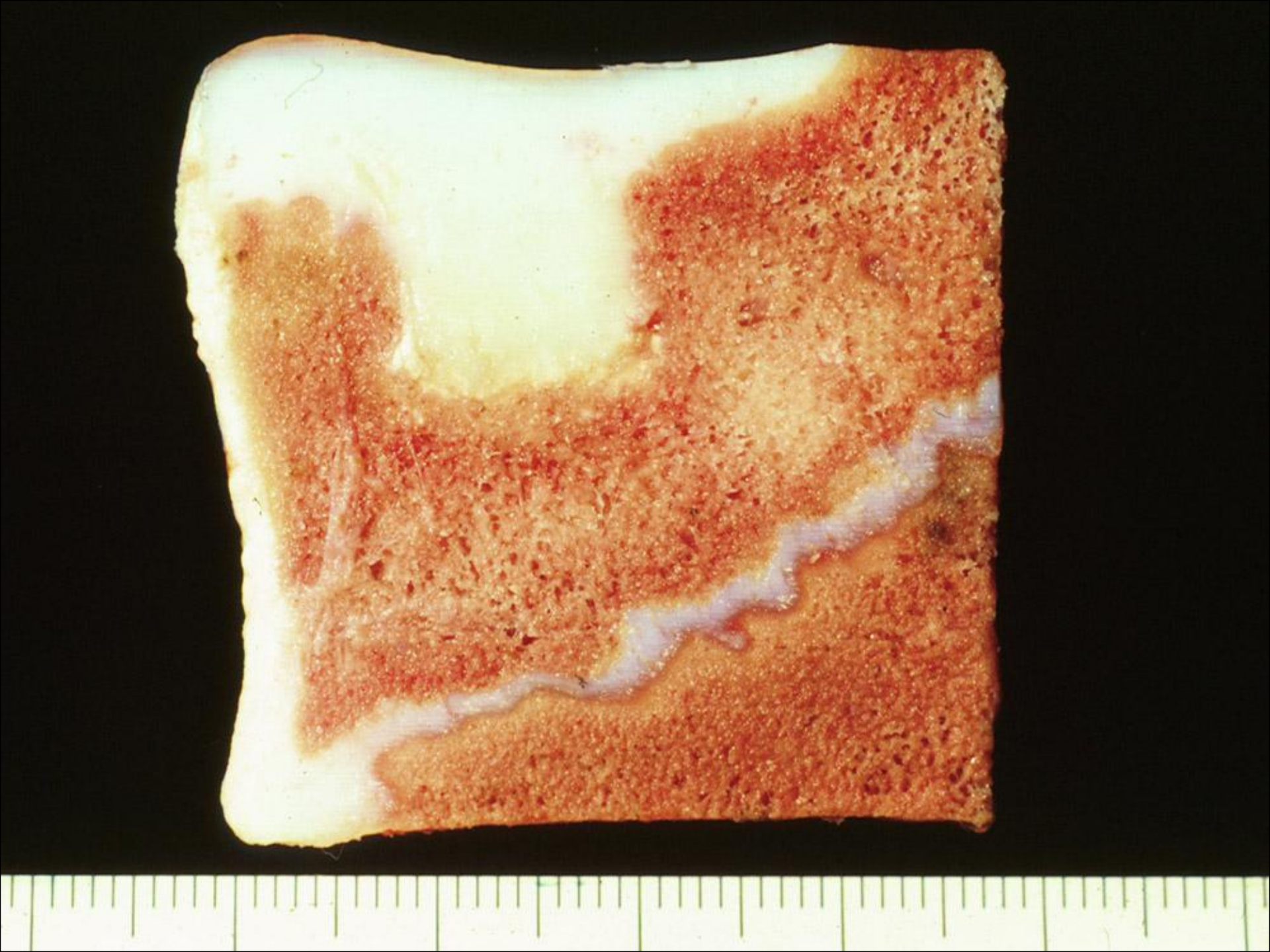
- Transverse trabeculation between trabeculae secondary to slowed longitudinal growth
- Mechanism = ???

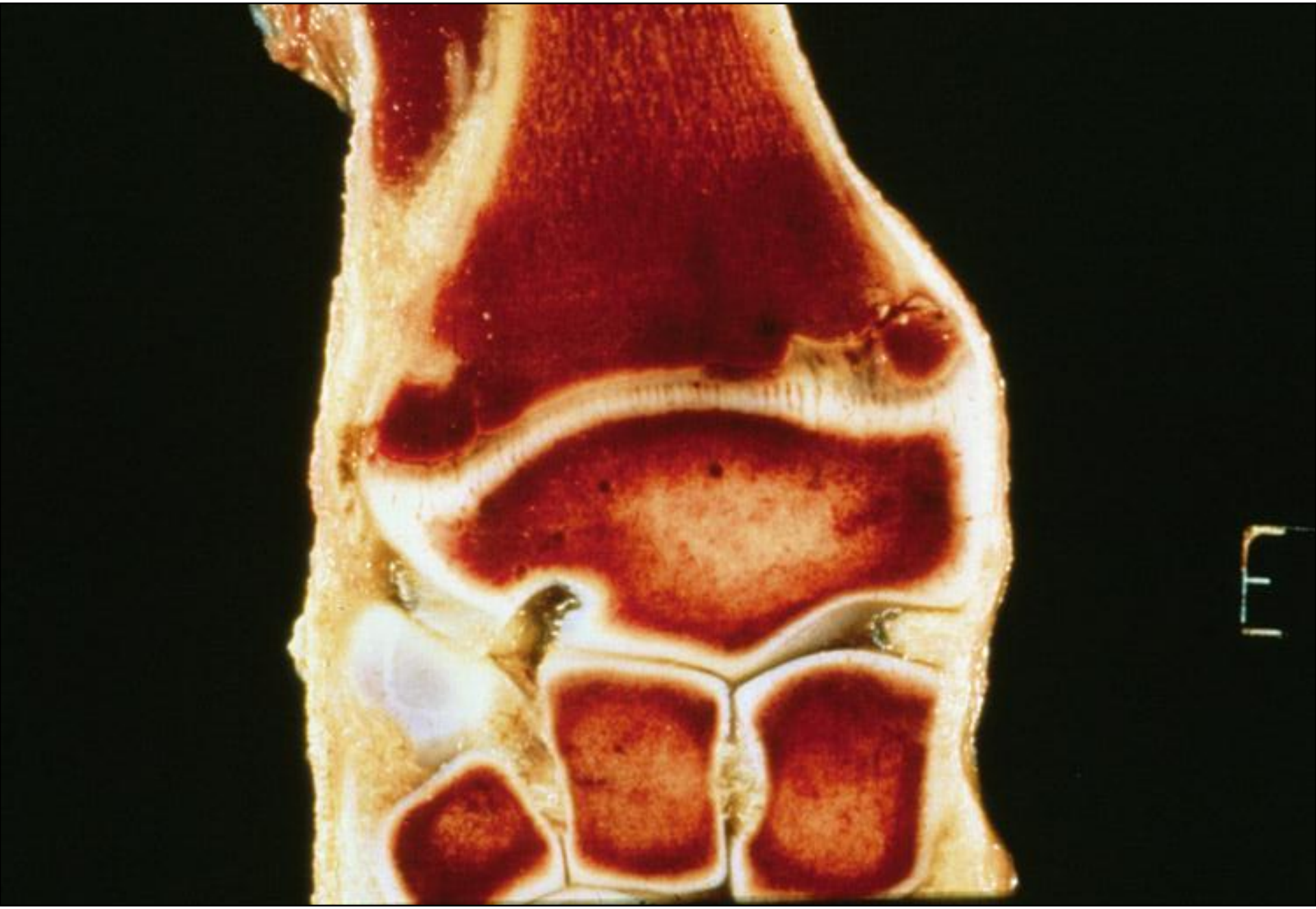


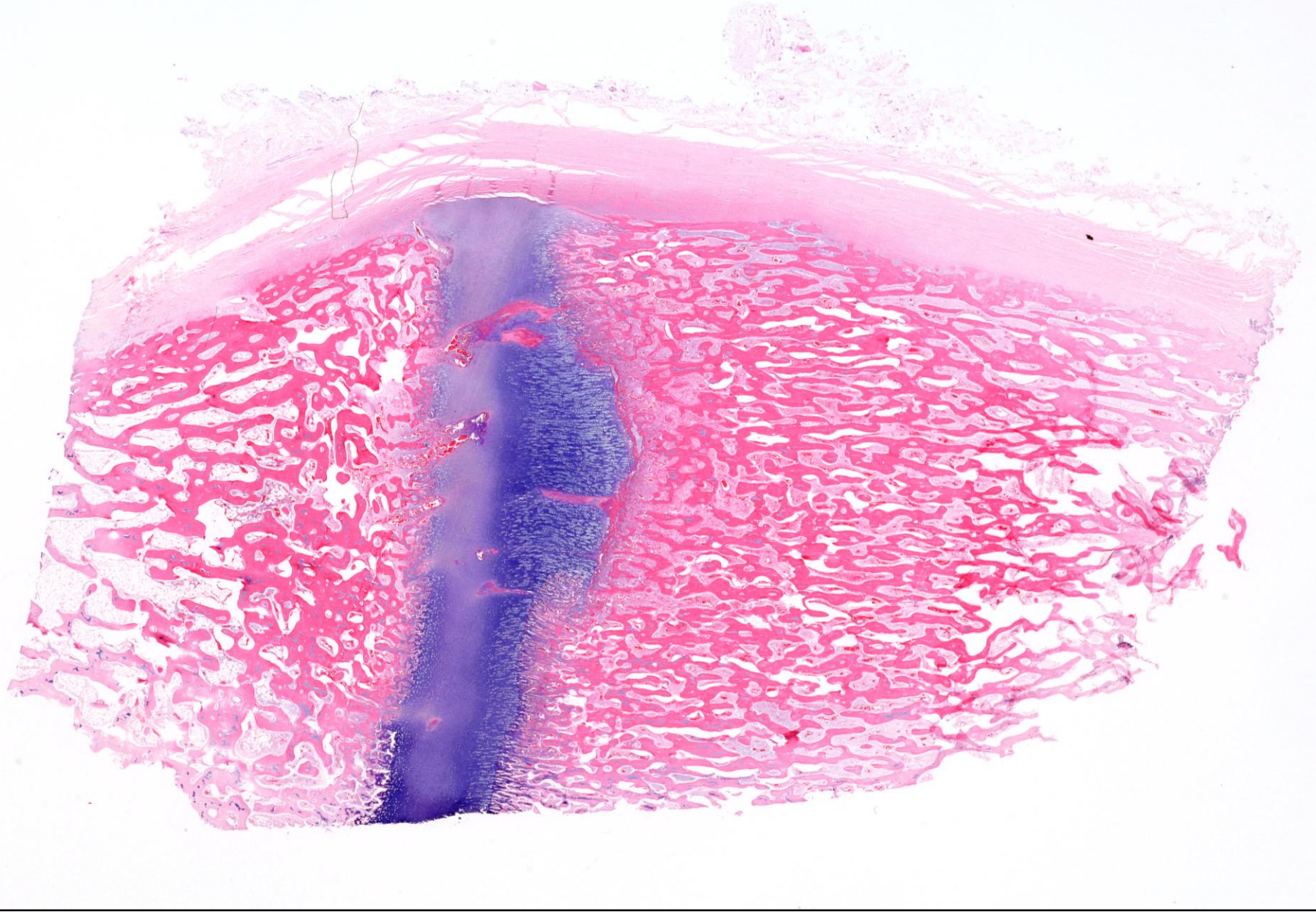


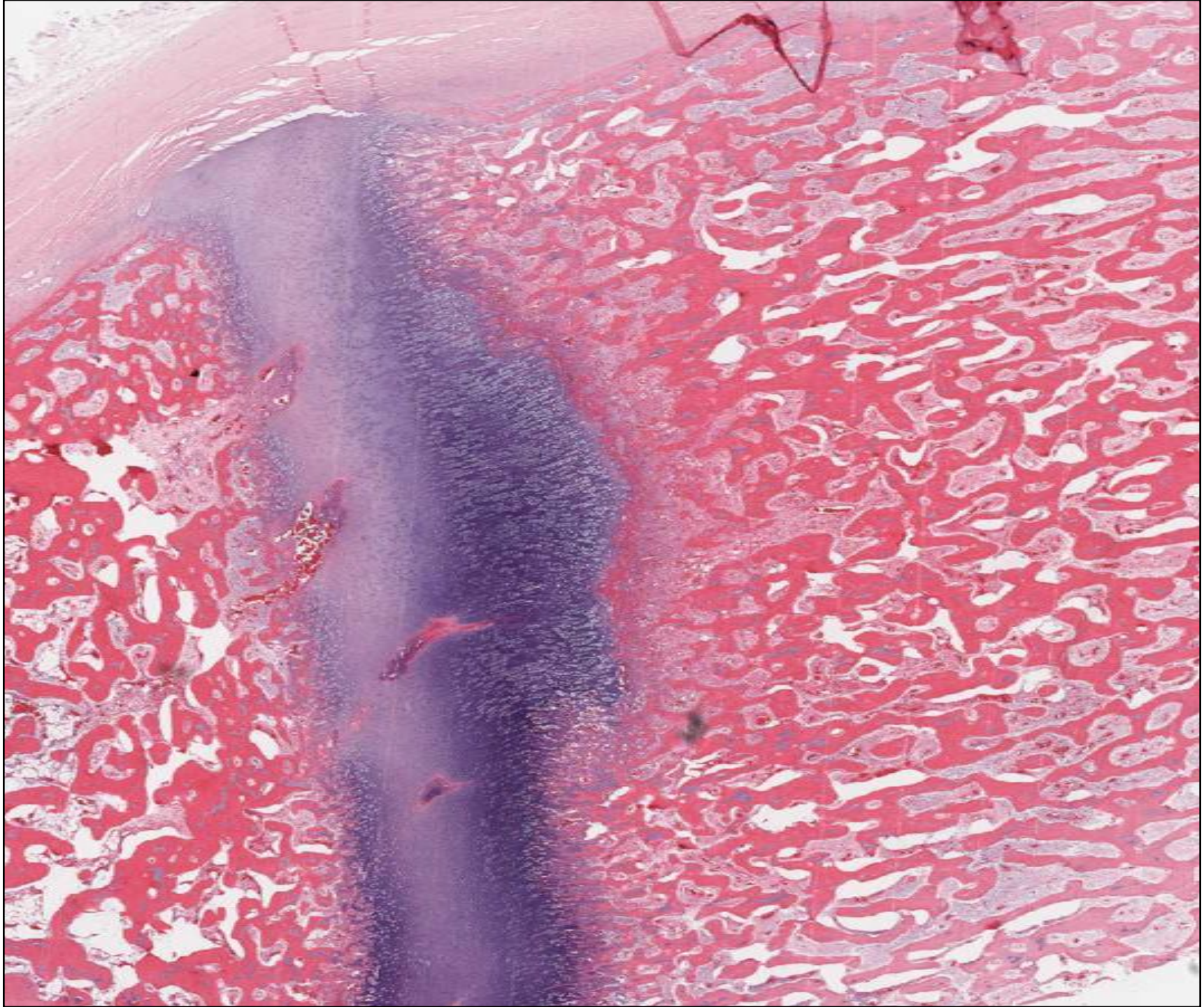
Retained Growth Cartilage

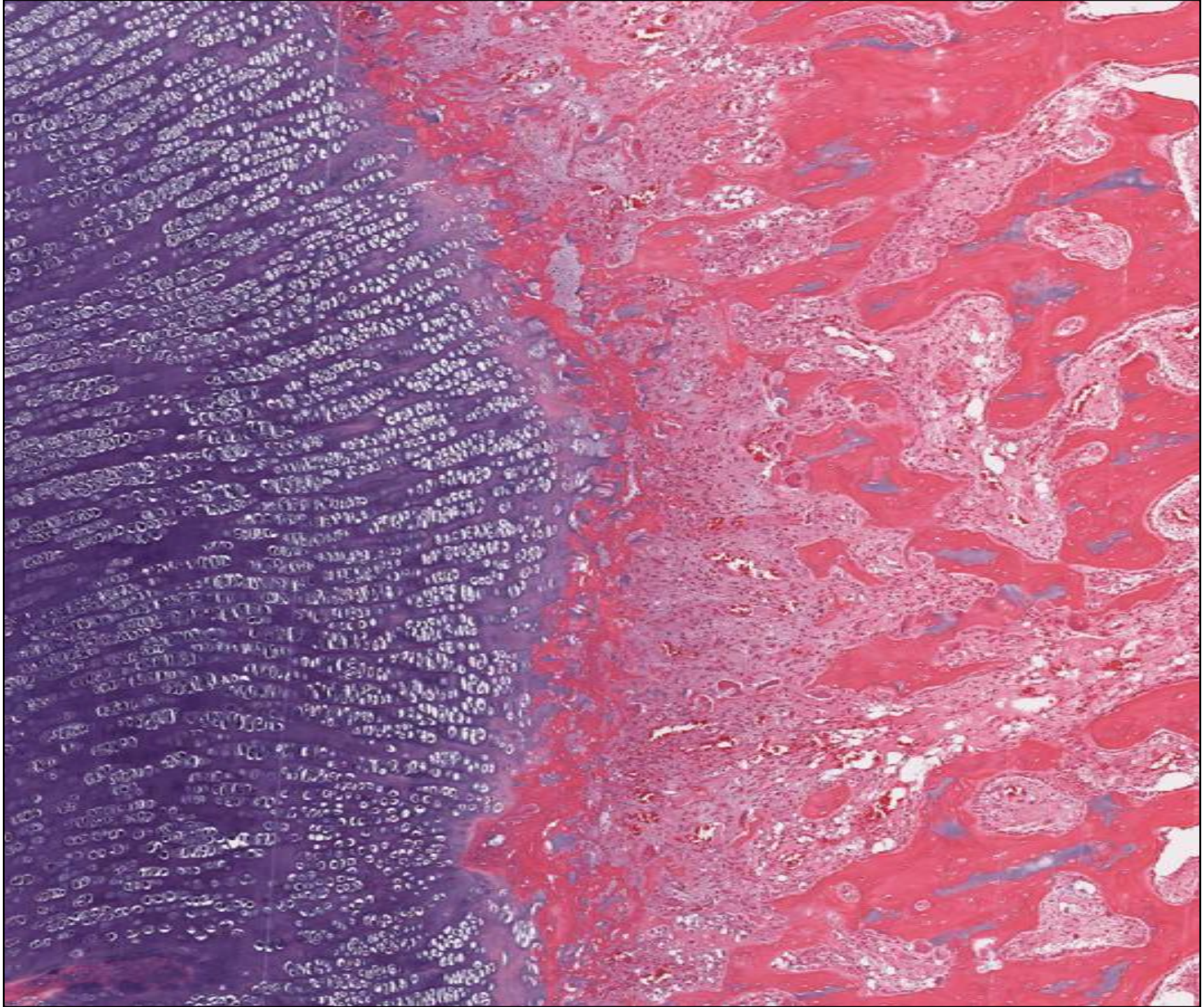
- Primary and idiopathic – osteochondrosis
- Secondary impaired vascular invasion of plate due to inflammation
- Secondary impaired vascular invasion of plate due to trauma

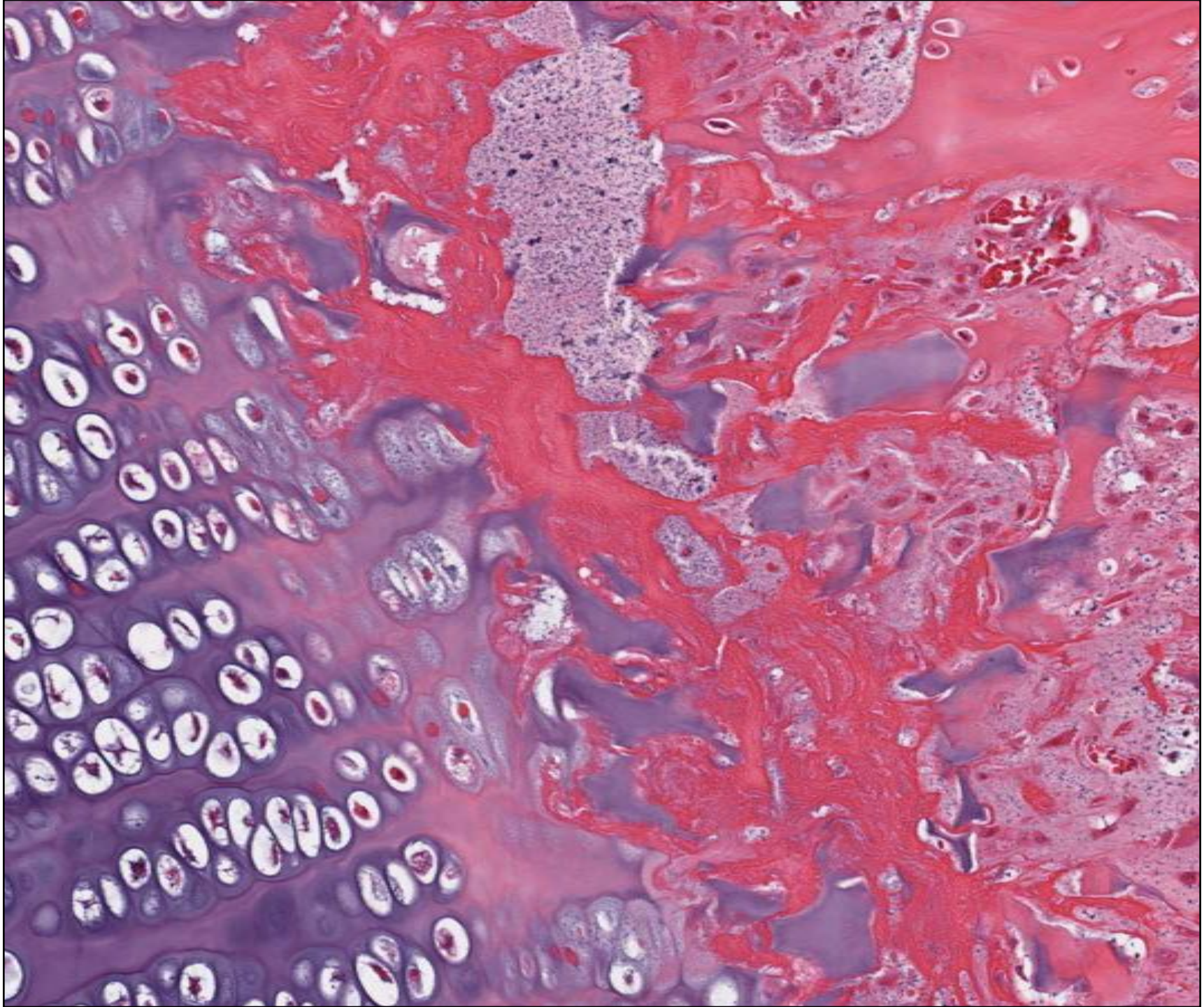






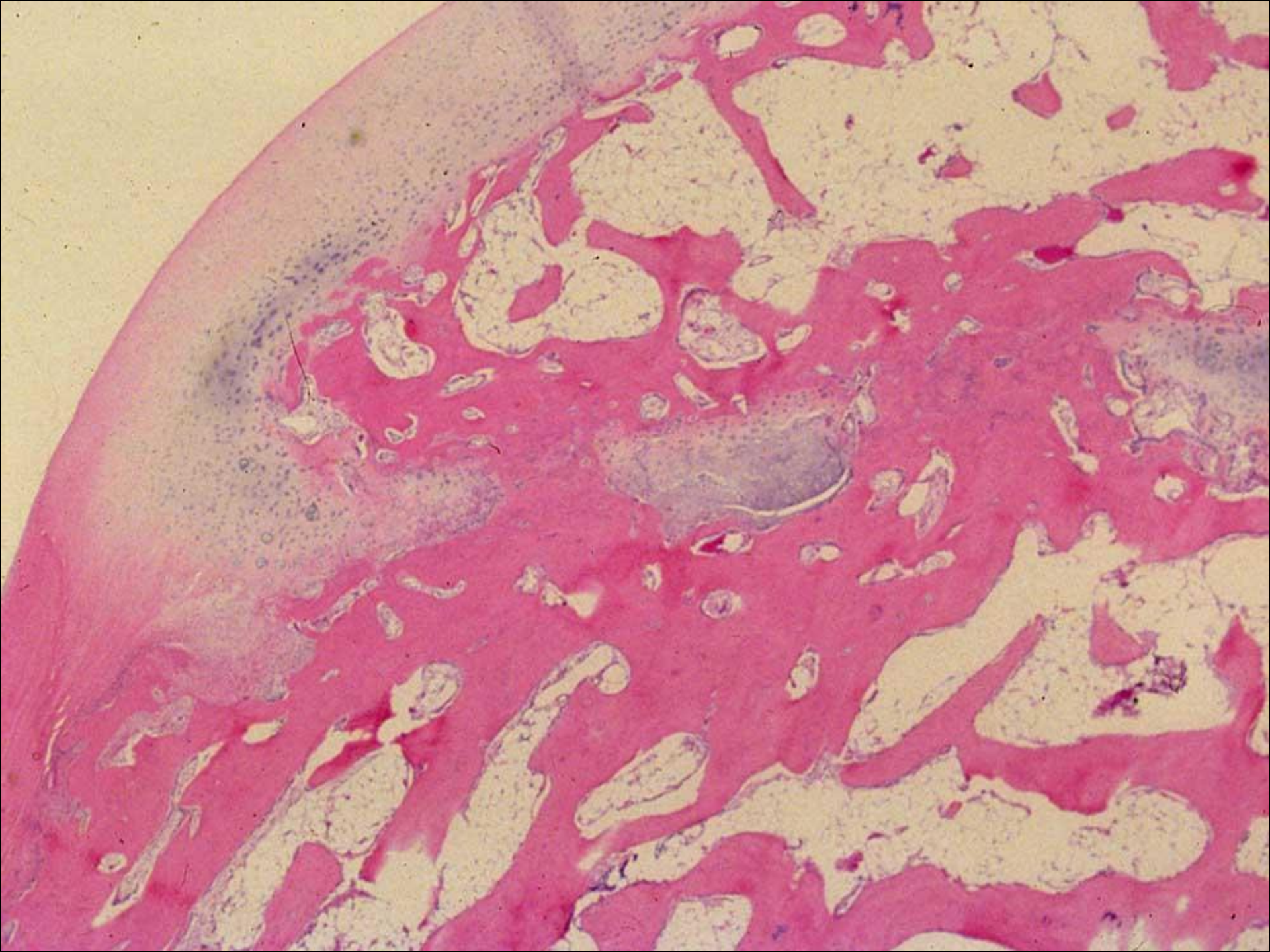


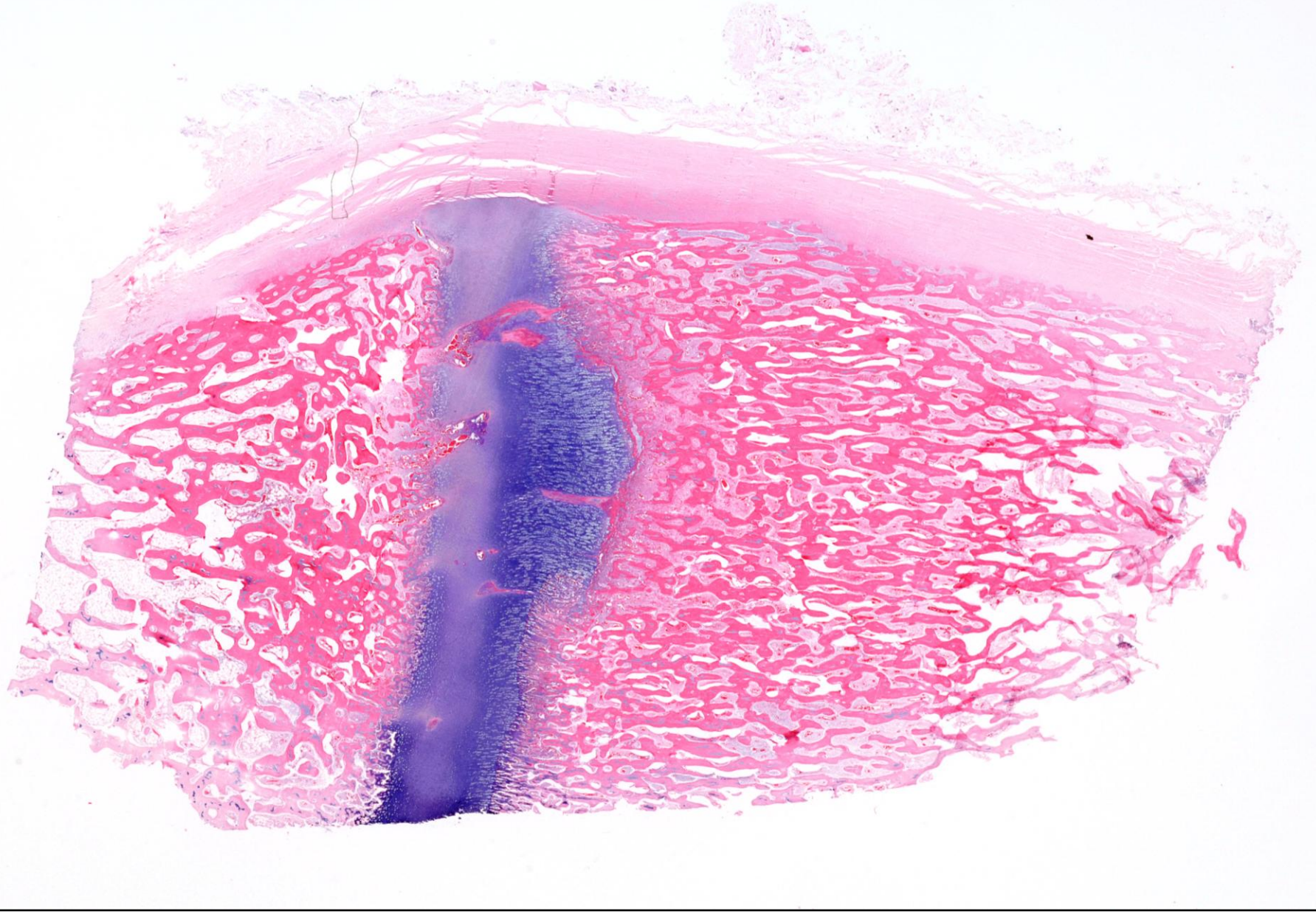




Angular Limb Deformity

- Secondary to trauma or inflammation at the physis (physitis in horses)
- Many cases due to confirmational abnormalities of complex cause/pathogenesis





Modeling in response to structural damage and abnormal use (Wolff's Law)

Bone will model to accommodate mechanical use

- Formation with compression
- Resorption with tension
- Alignment of trabeculae along lines of stress

Detection of Altered Mechanical Use

- Streaming potentials in the canalicular system
- Piezoelectric forces derived from deformation of the collagen “crystal” lattice
- Stretch receptors on osteoblasts
- Compression/tension on nerves and blood vessels

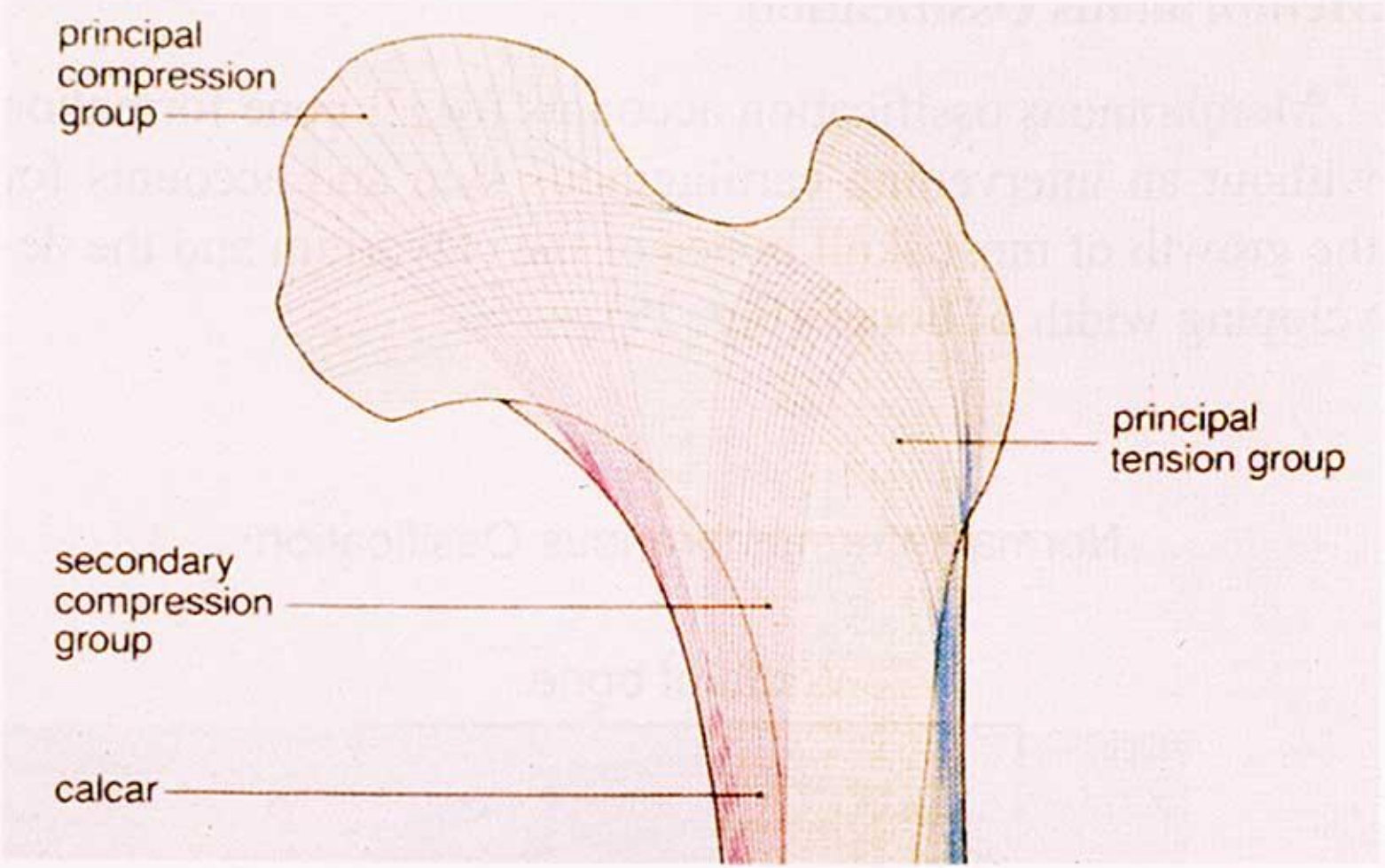
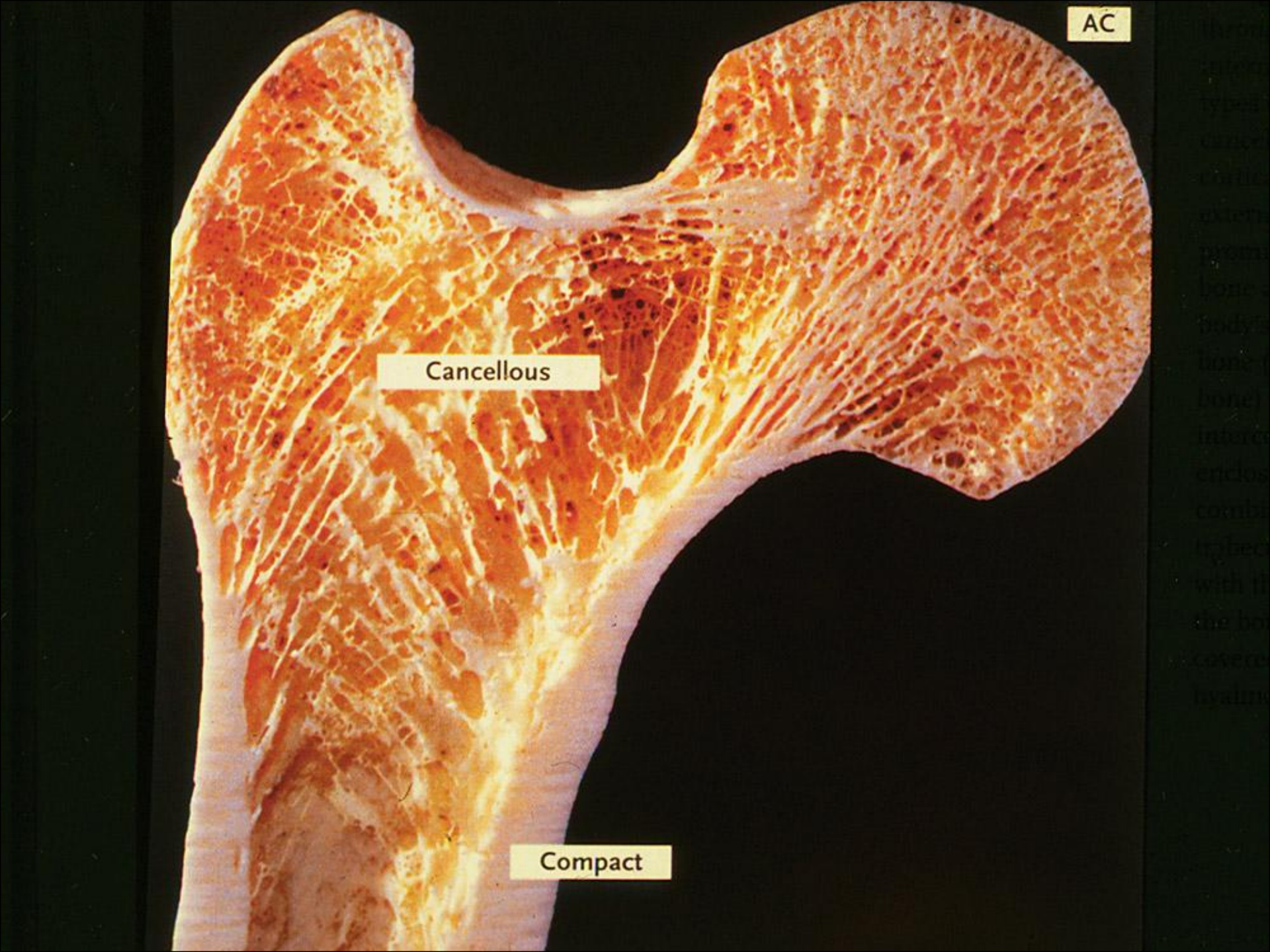


FIG. 31. Bone modeling: the trabecular structure of the proximal femur is a composition of arcades of cancellous bone that "model" or "shape" the internal architecture of bone along compressive and tensile stresses produced during weight bearing.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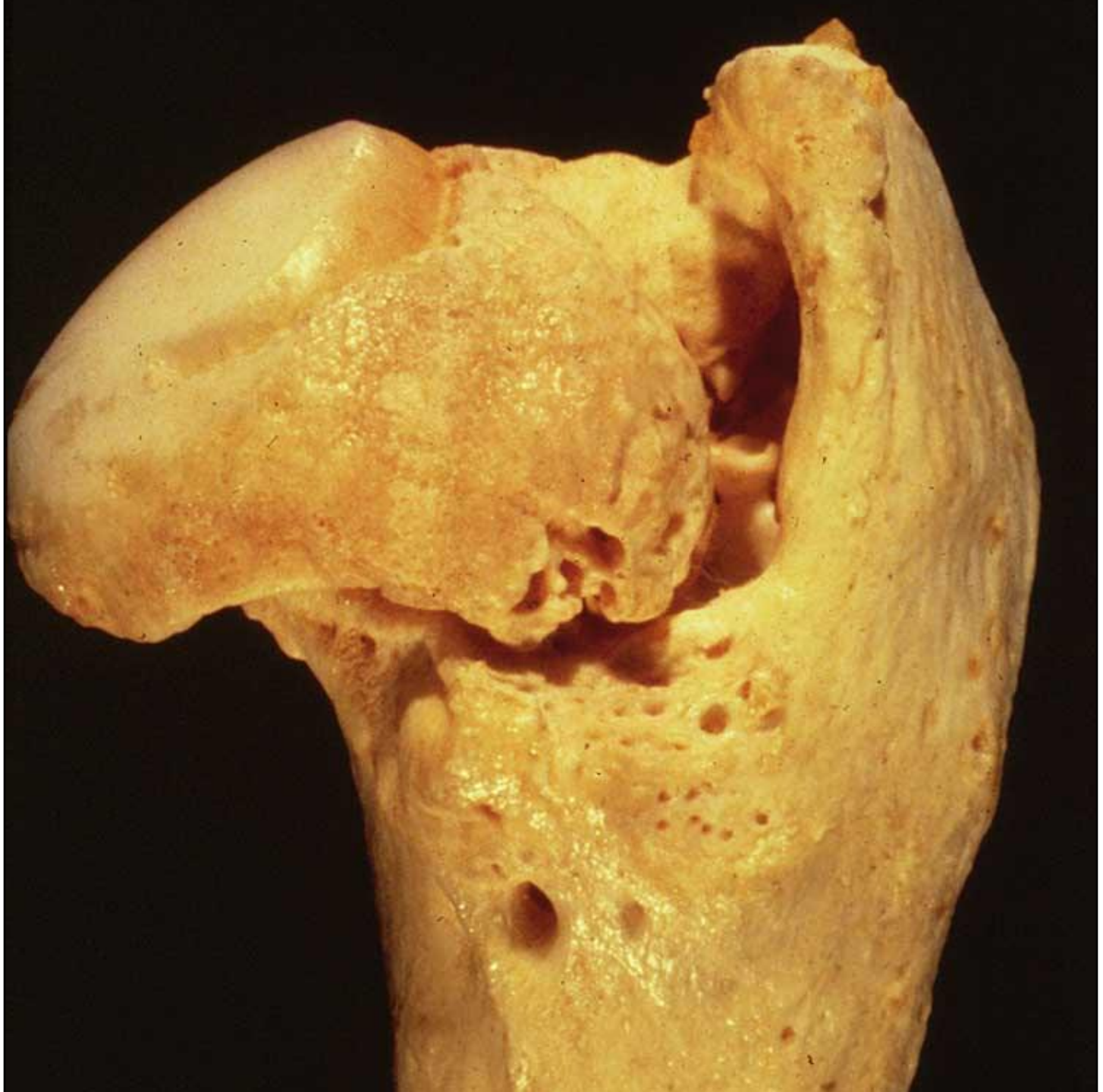

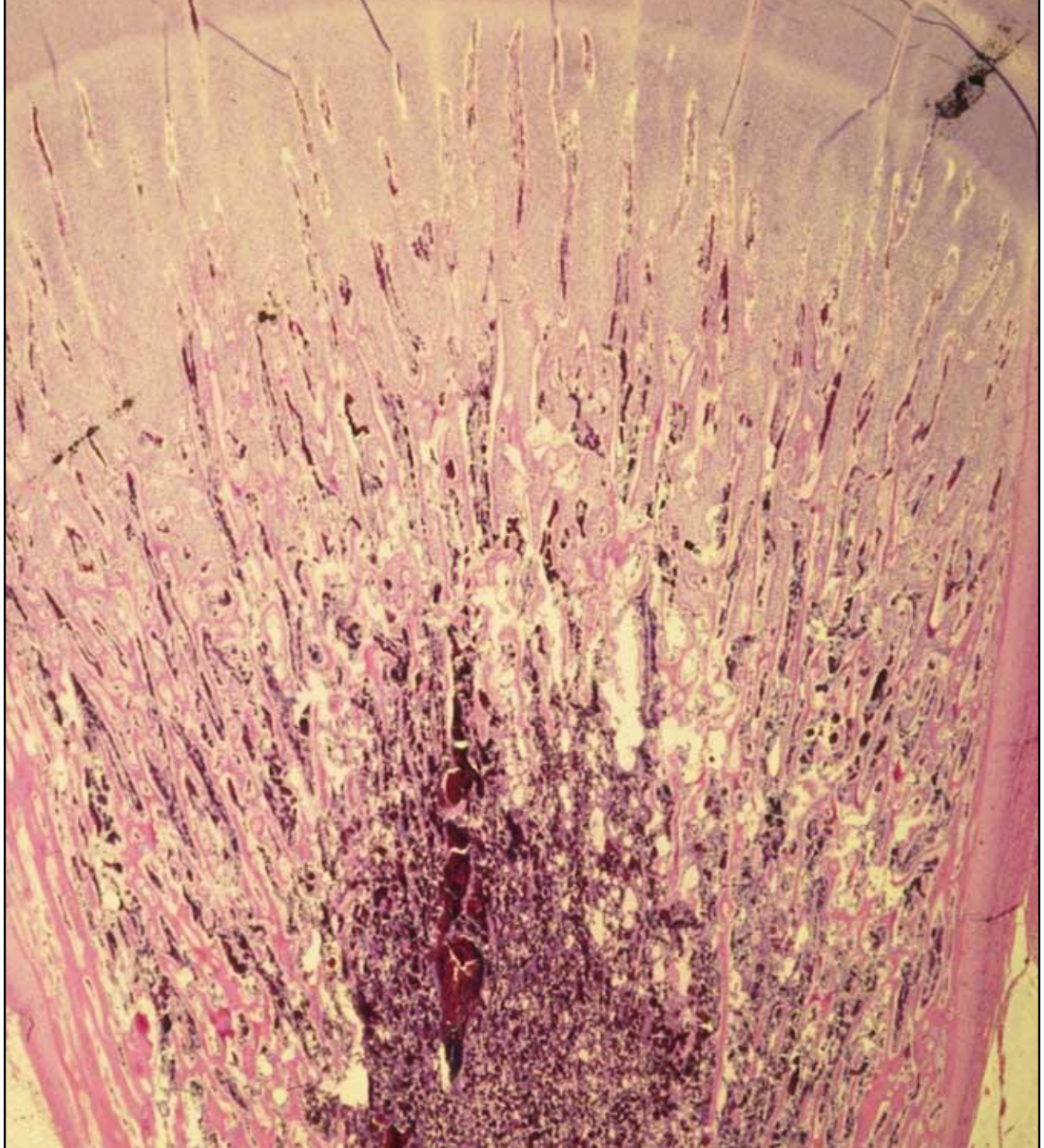

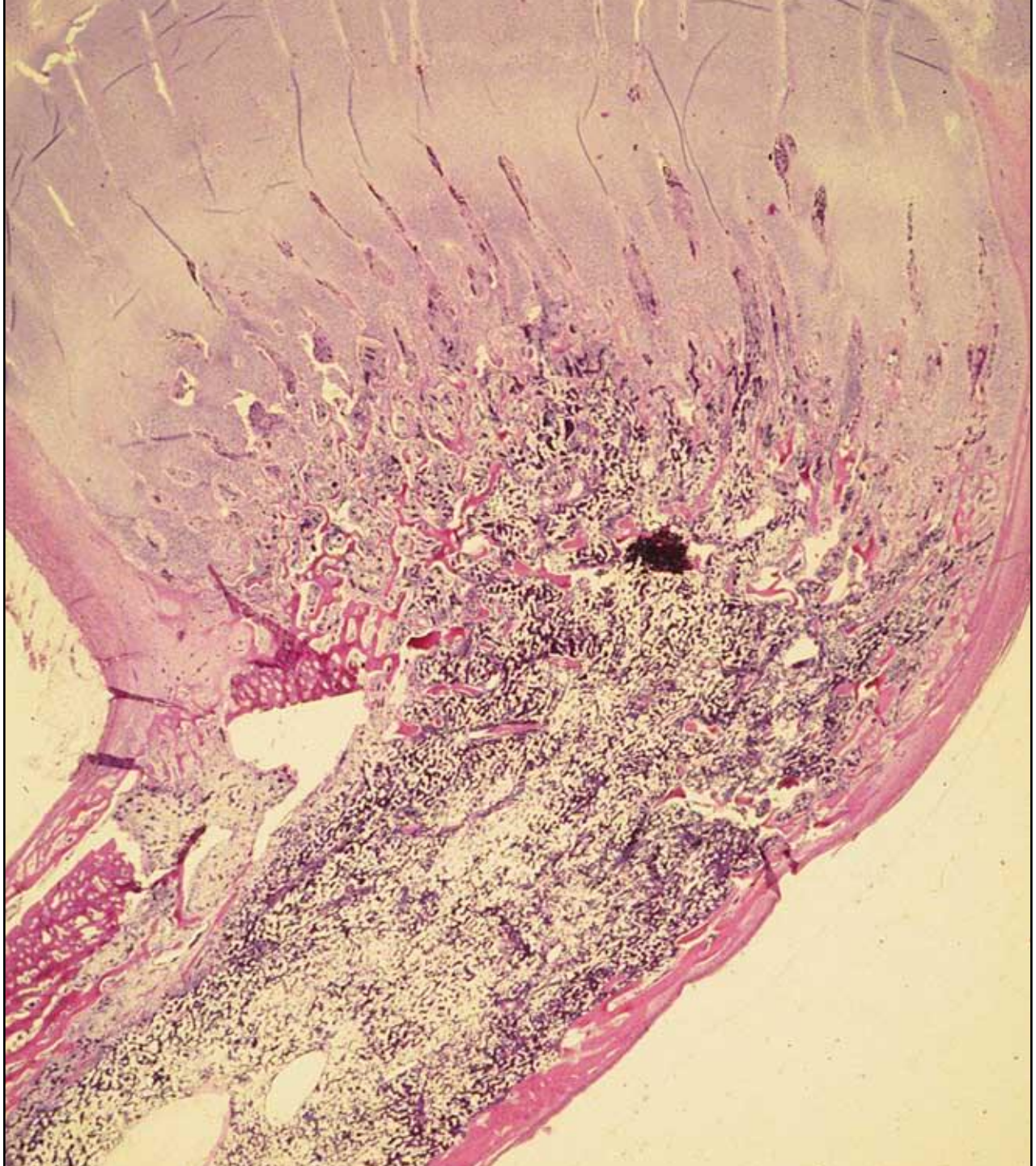

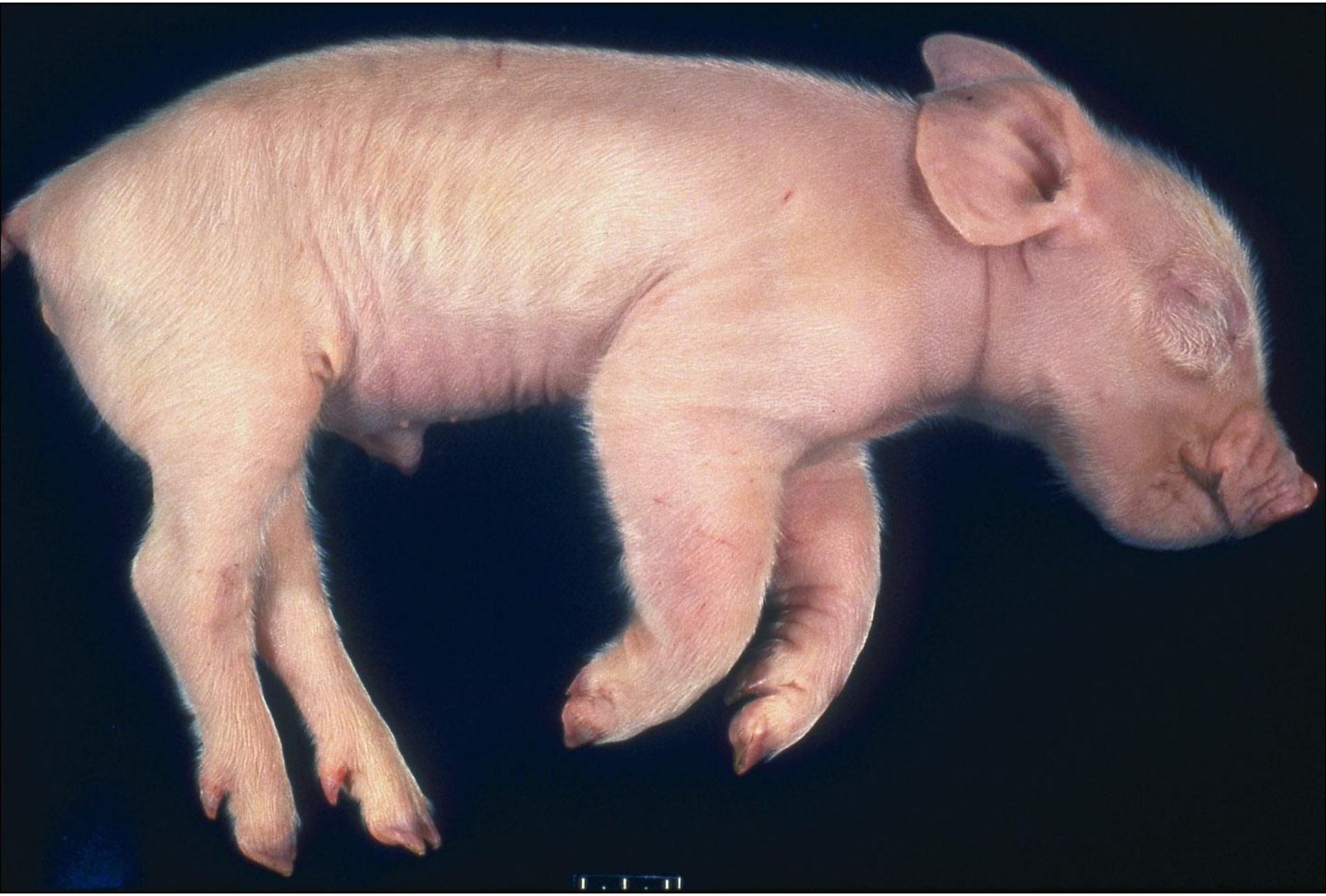

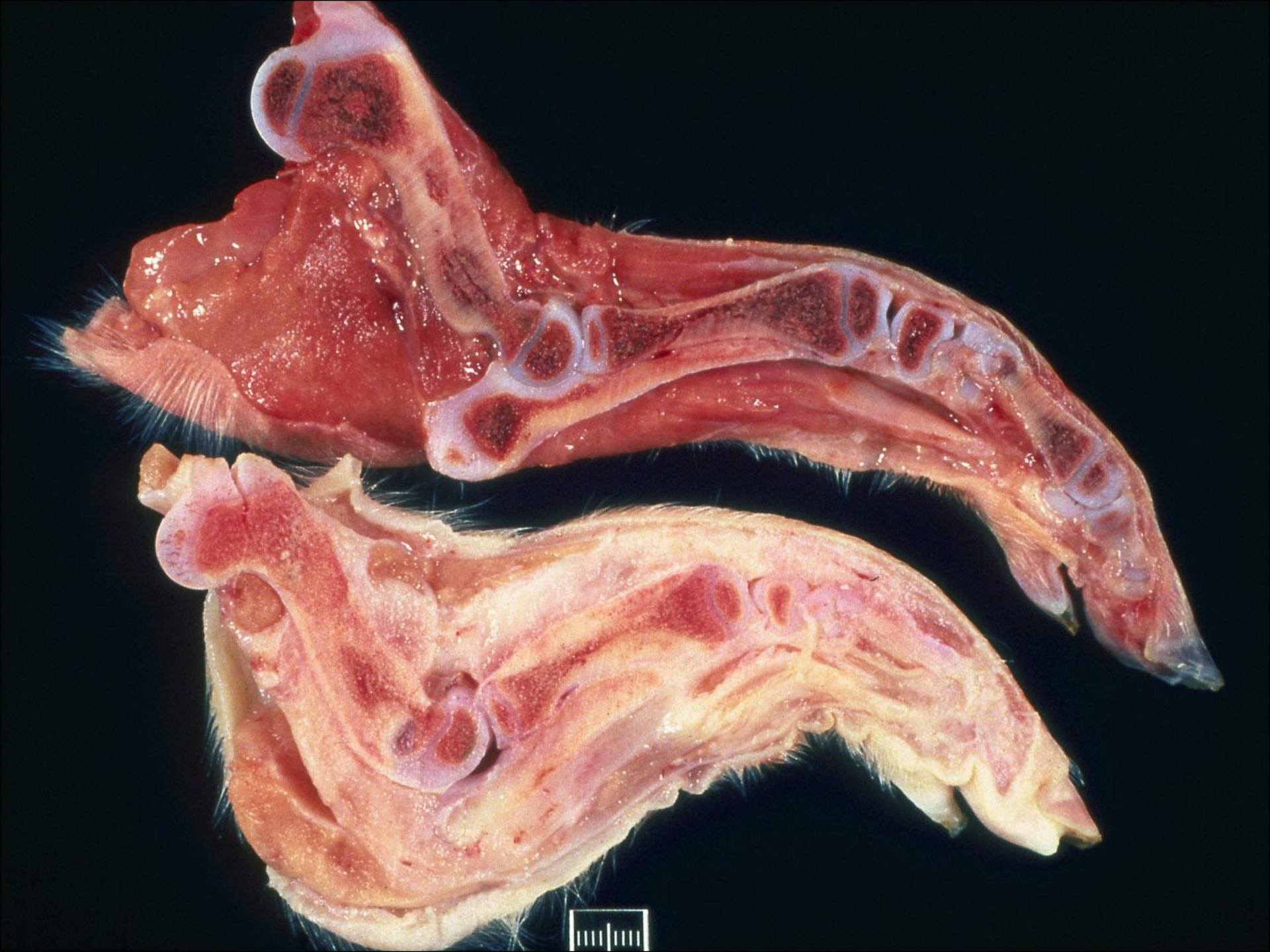

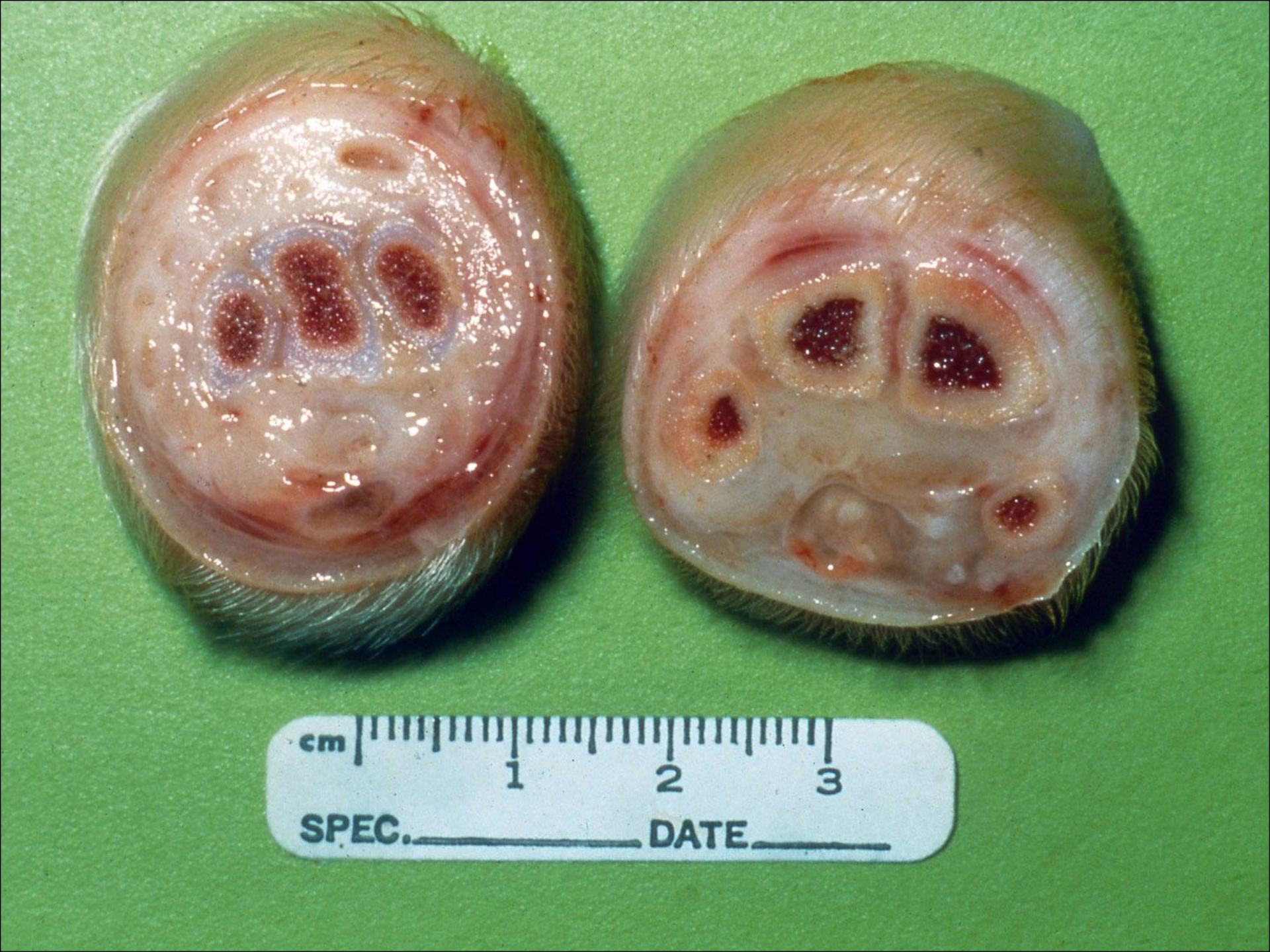
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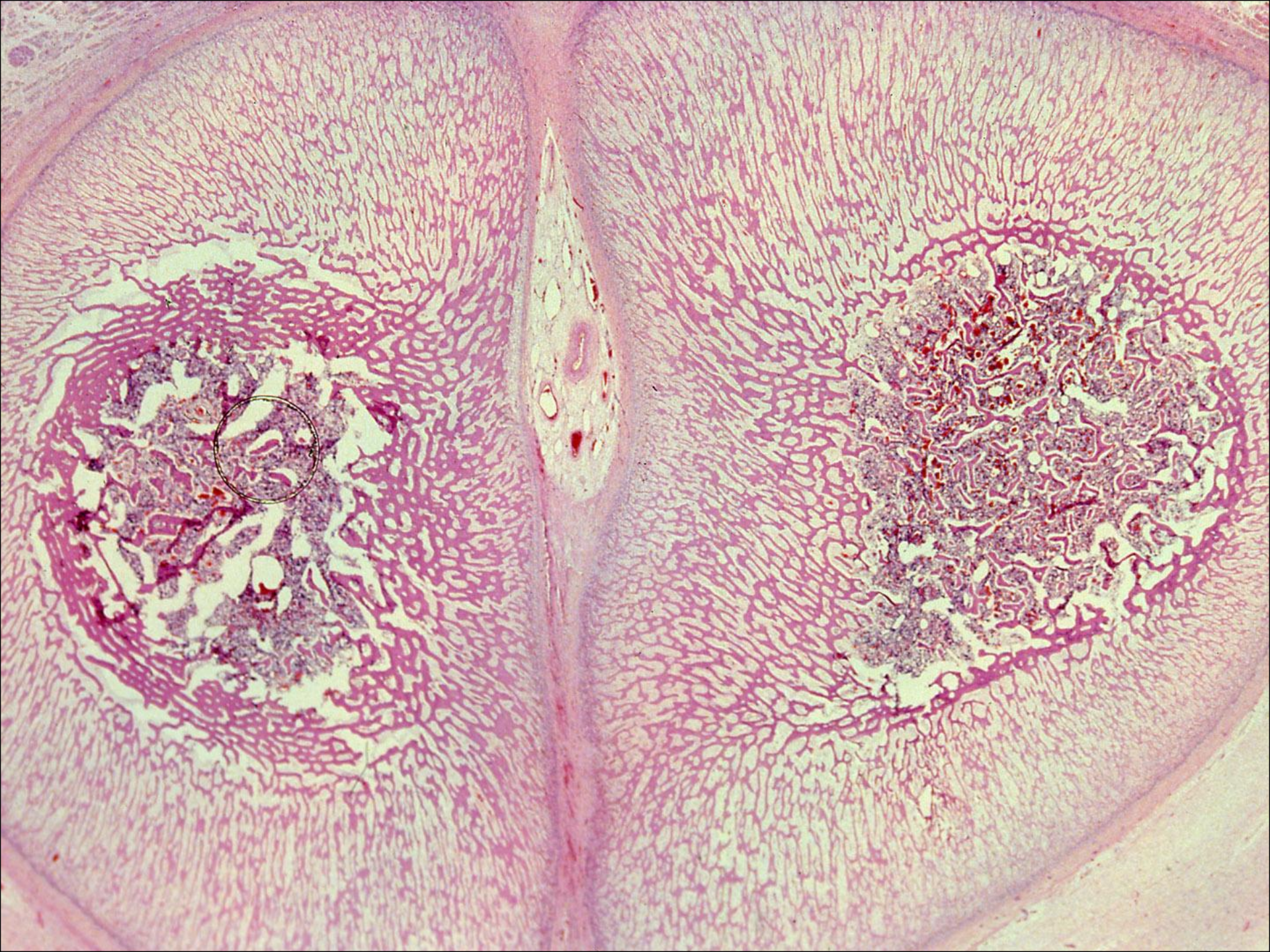
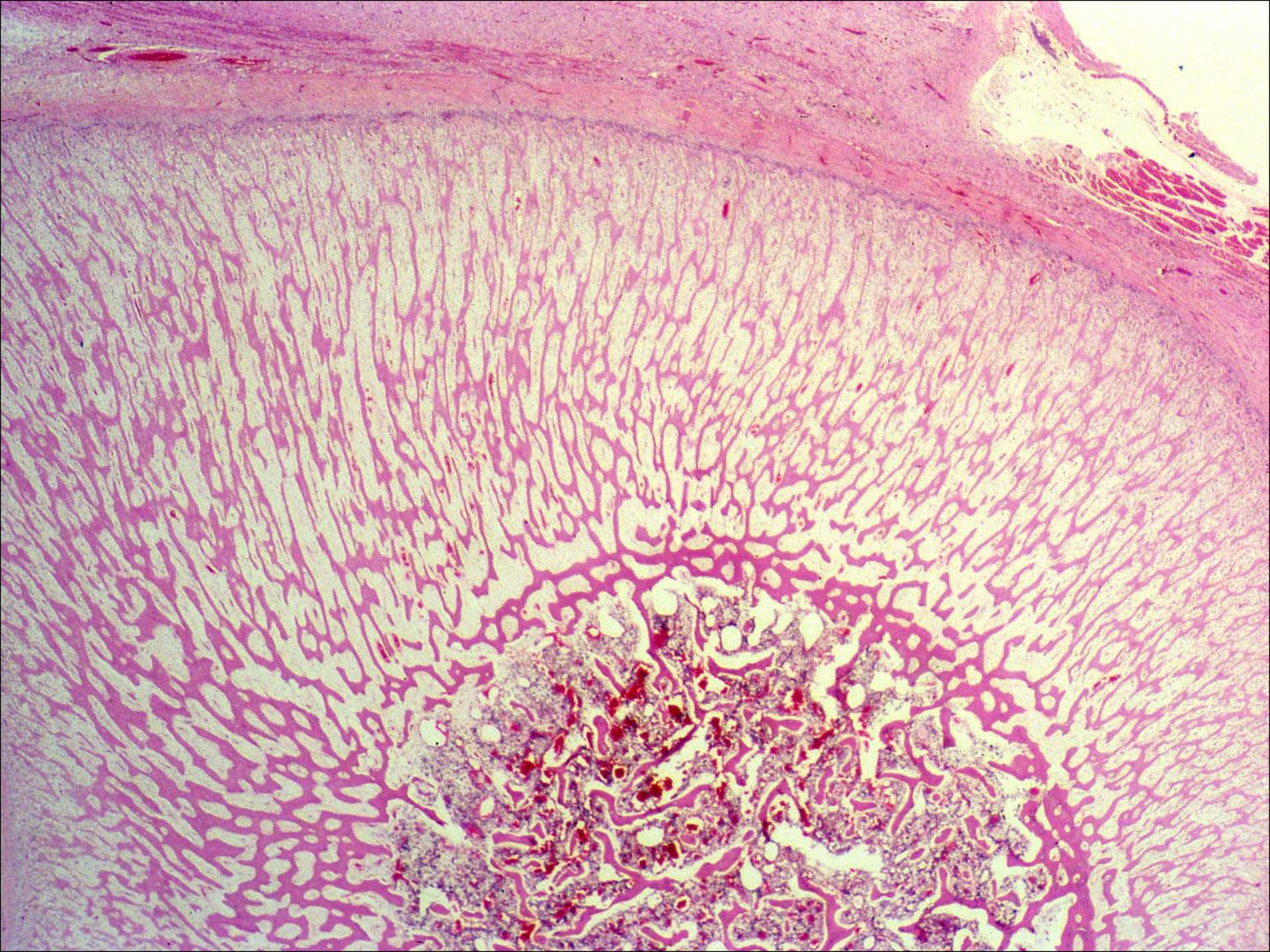
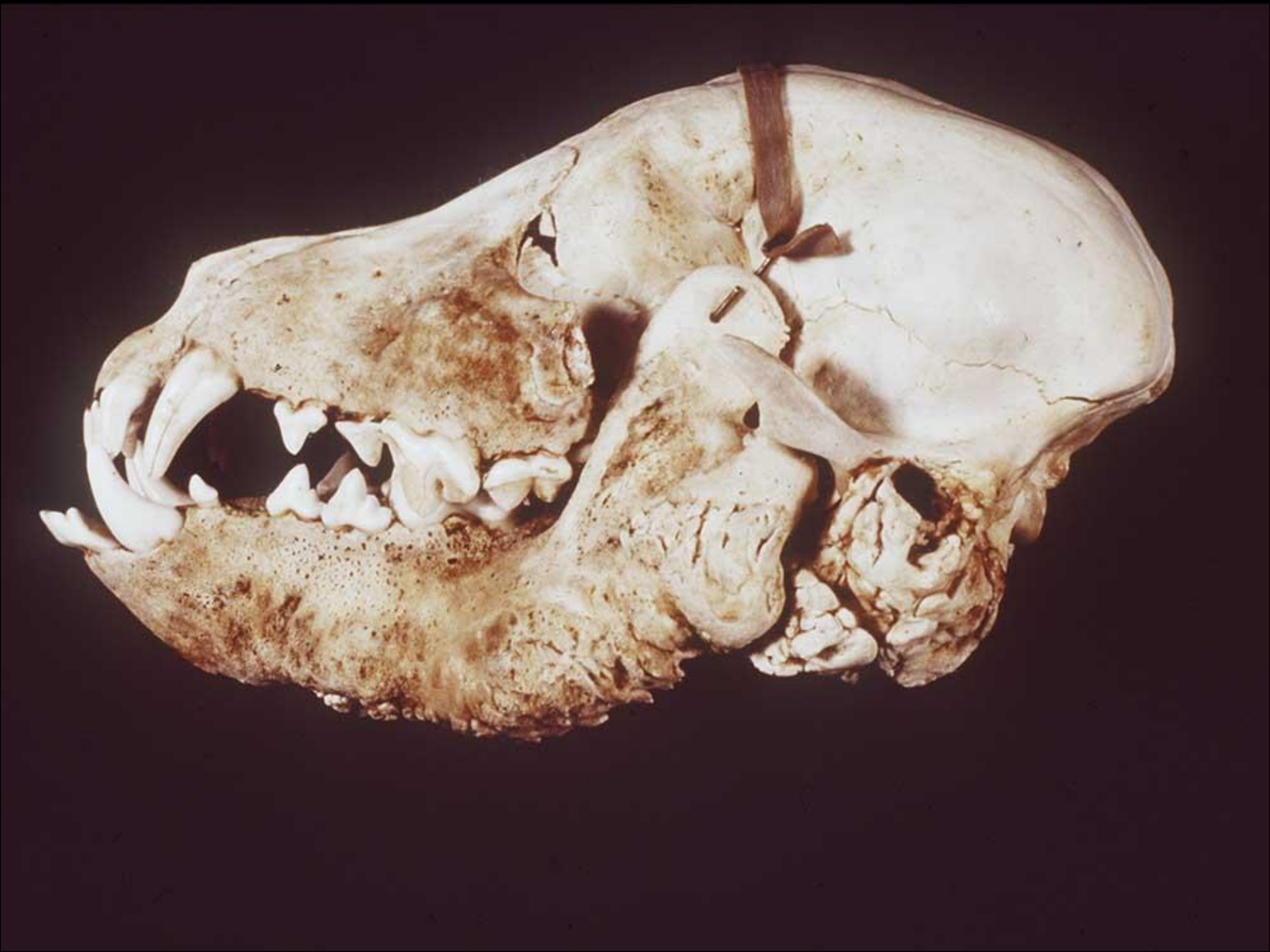
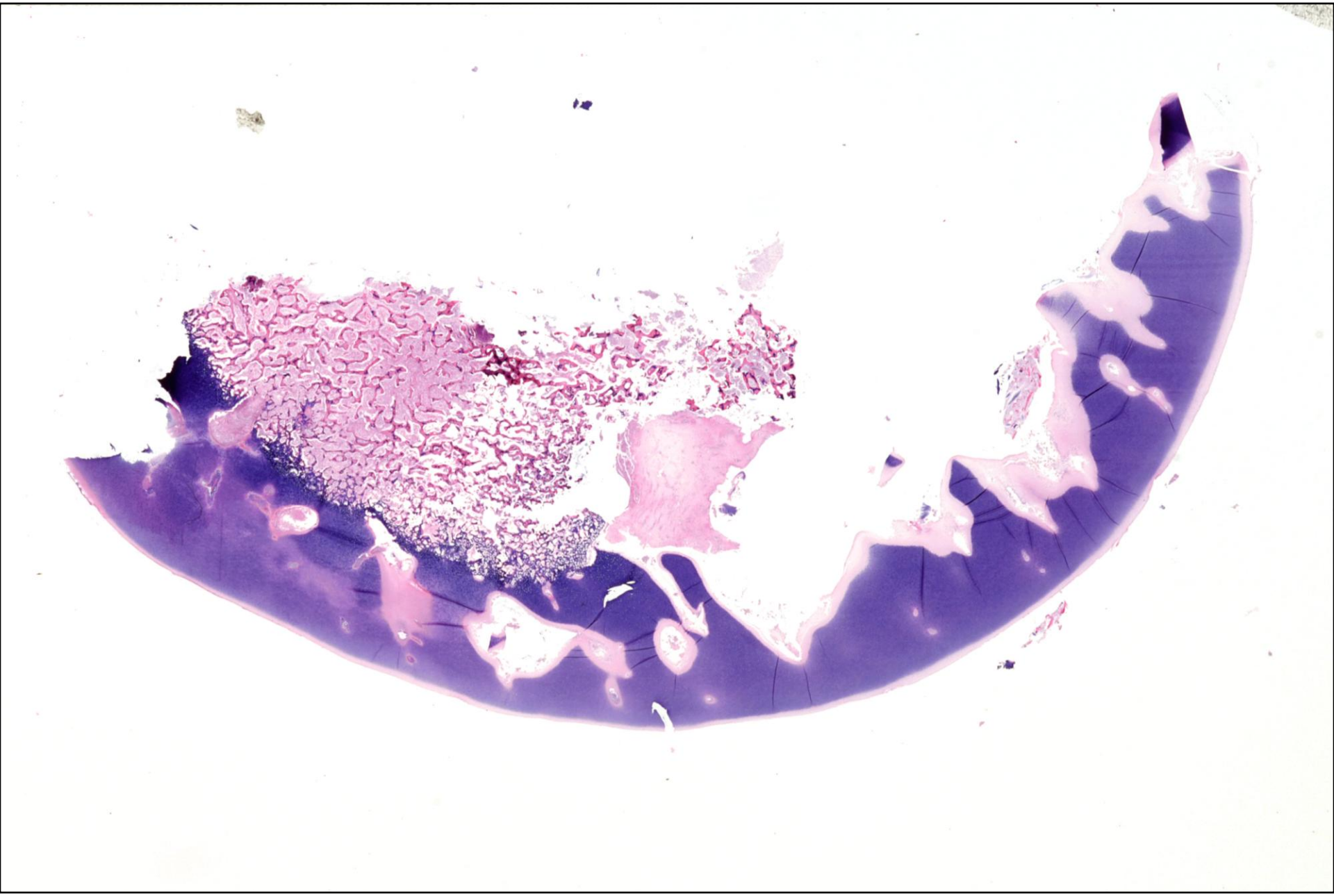


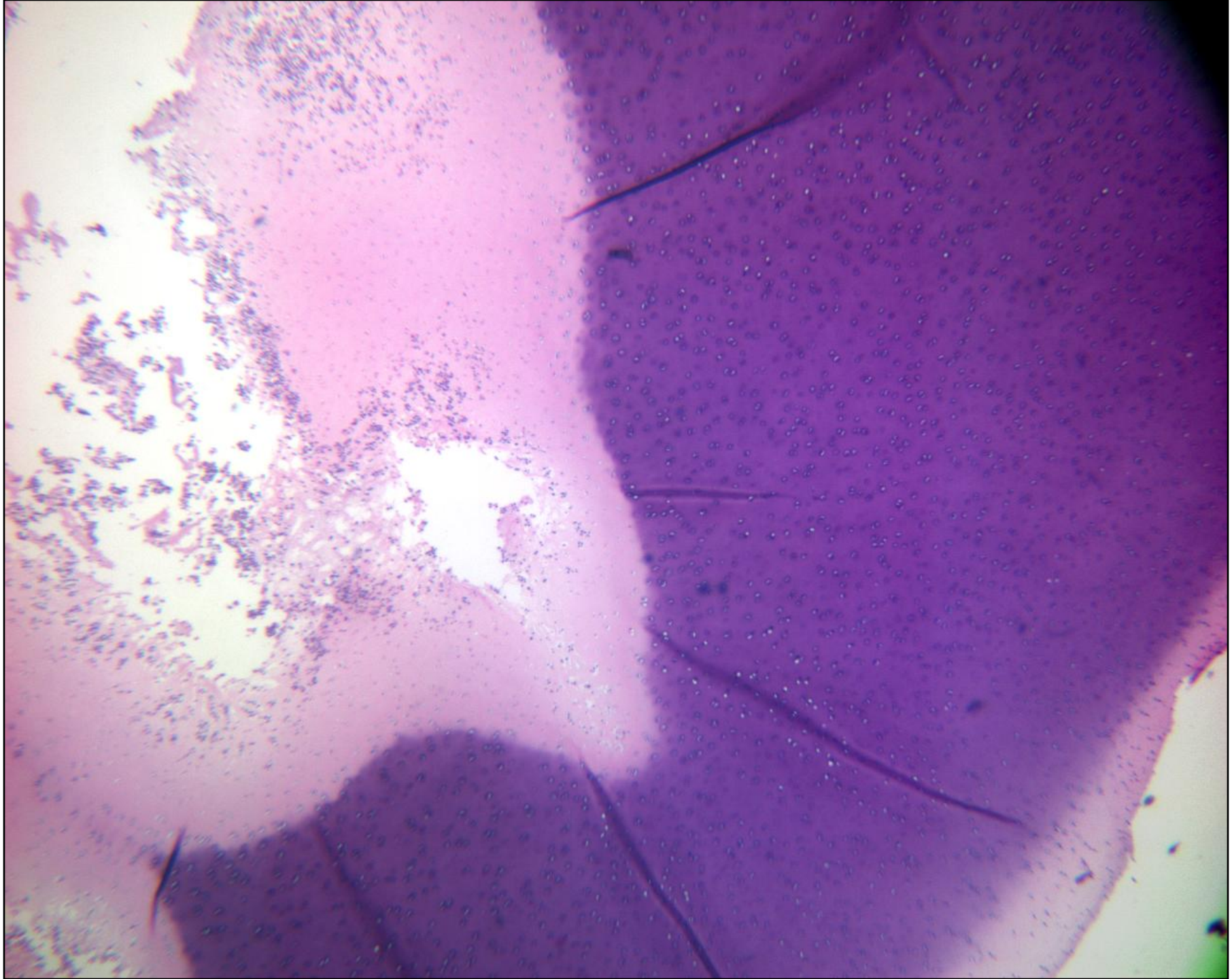


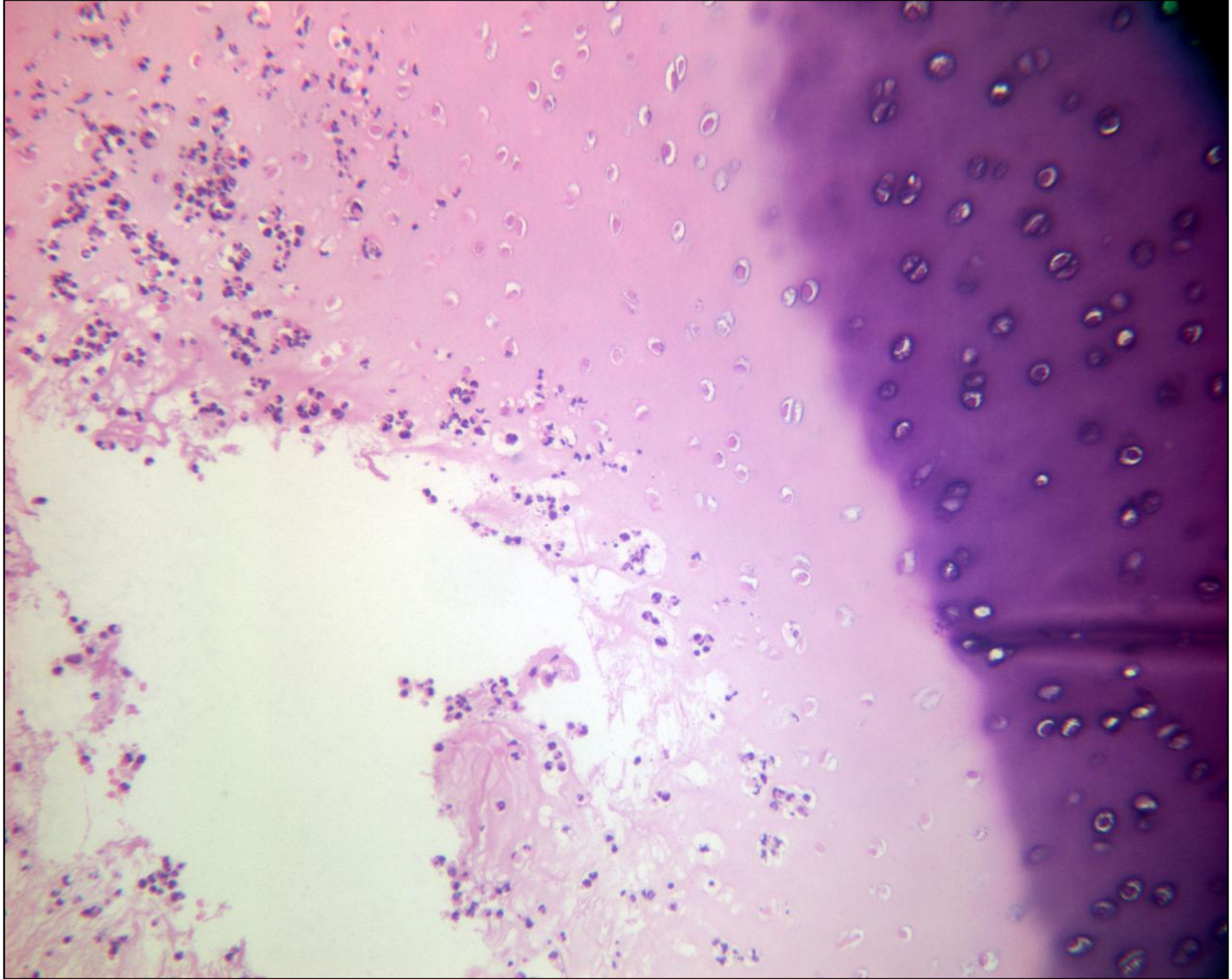


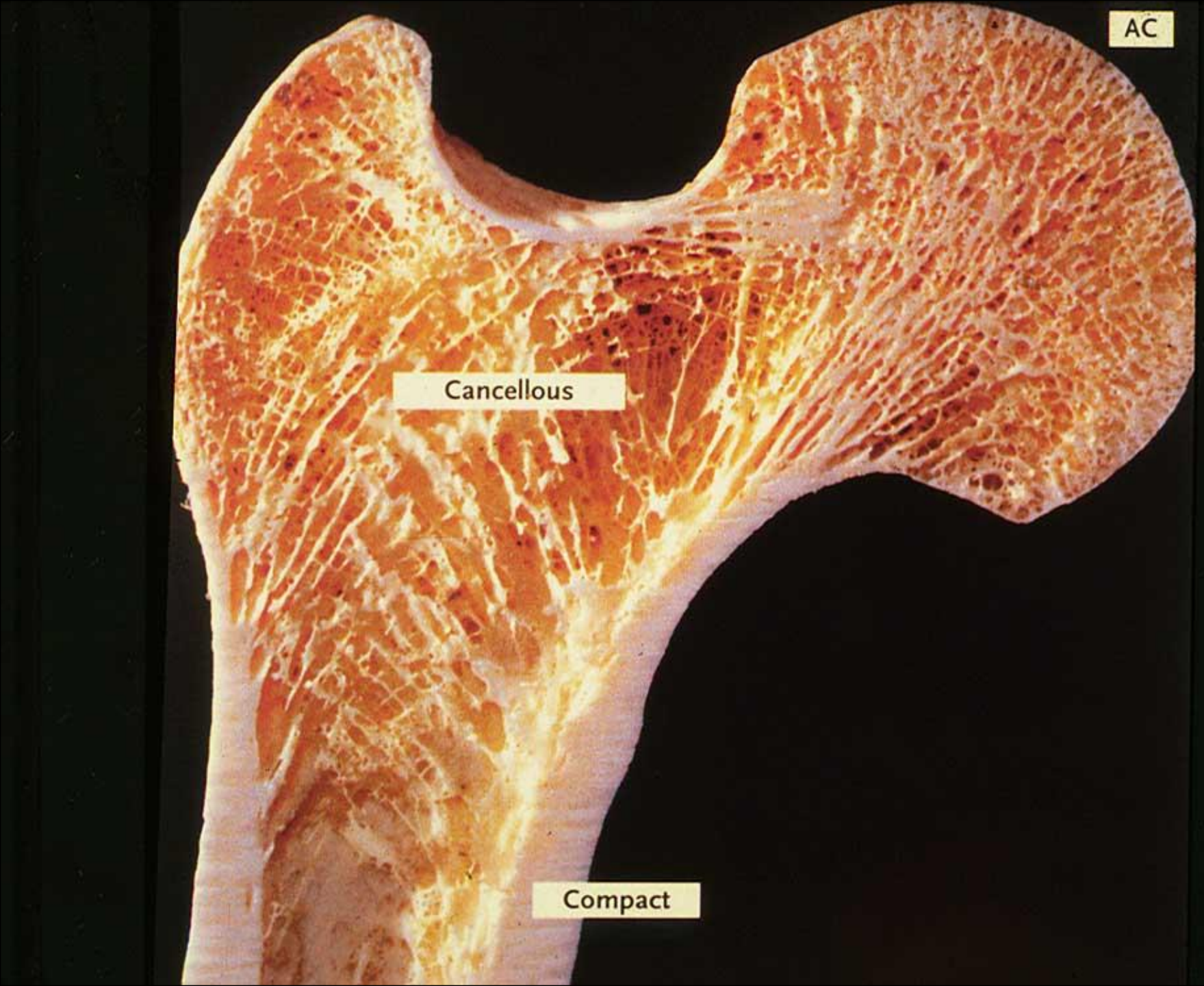
Major Bone Disease Categories (from McCarthy and Frassica)

1. Congenital
2. Metabolic
3. Traumatic
4. Circulatory
5. Neoplastic
6. Infectious
7. Responses to systemic disease








AC
Cancellous
Compact