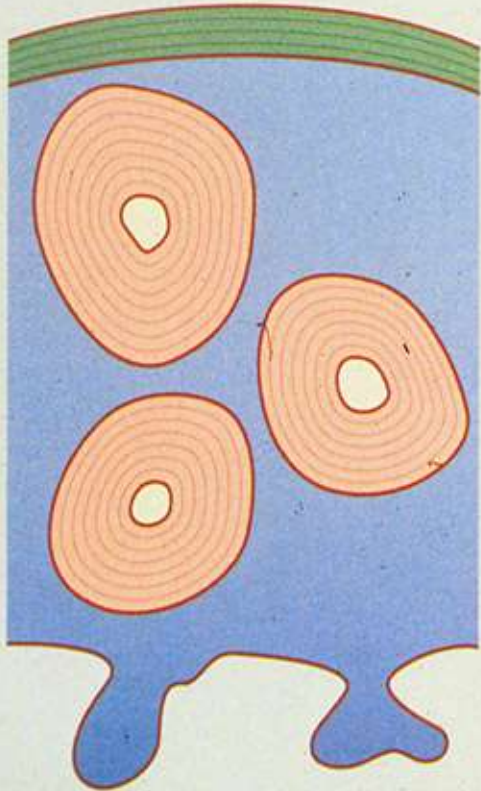


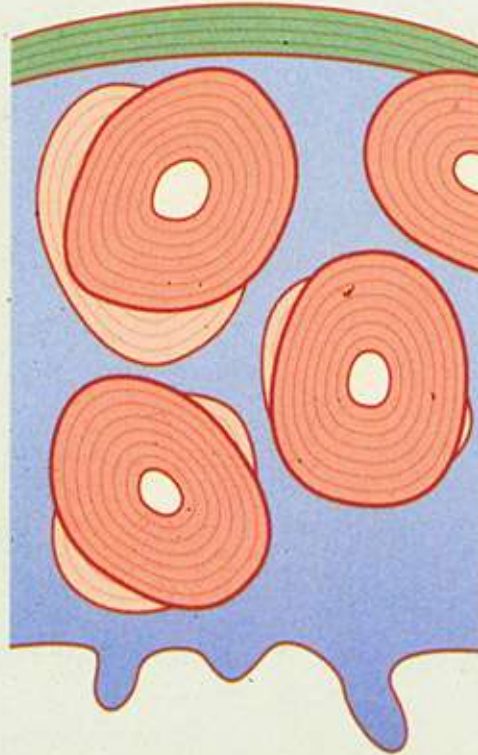


Primary and Secondary Remodeling

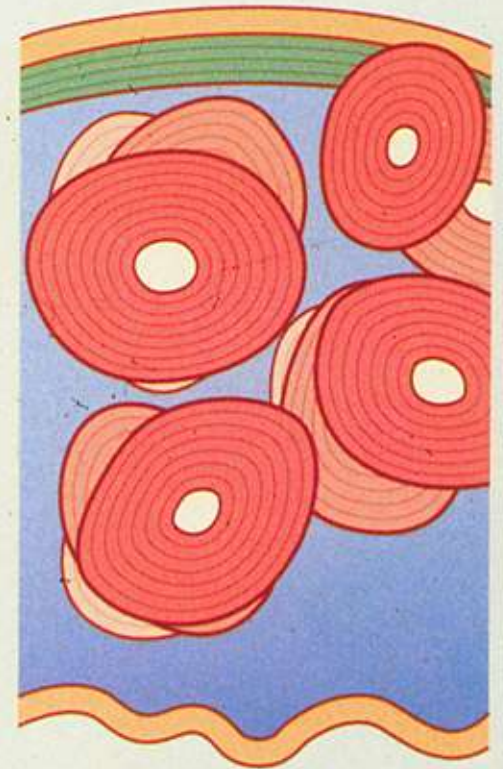
A First-generation Haversian system



B Second-generation Haversian system



C Third-generation Haversian system



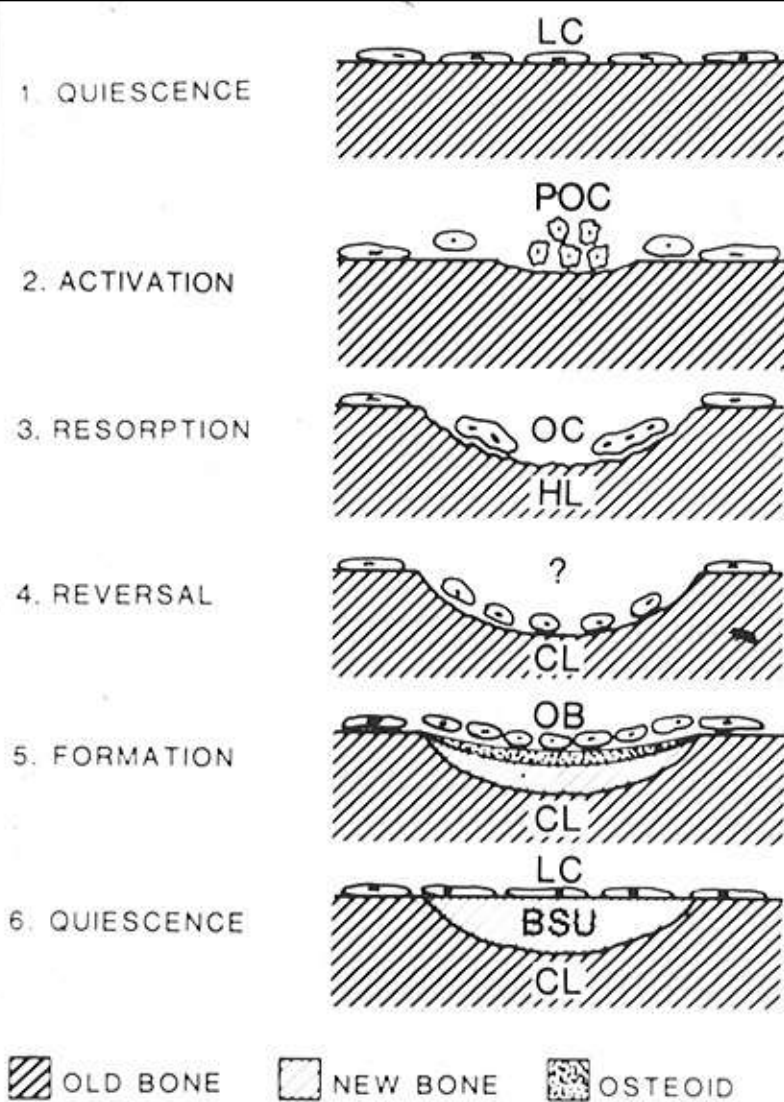


Fig. 1. The normal remodeling sequence in adult bone. Schematic representation of successive events on the endosteal surface. LC—lining cell. POC—preosteoclast (mononuclear). OC—osteoclast (multinucleated). HL—Howship's lacuna. CL—cement line. OB—osteoblast. BSU—bone structural unit. (Modified from Resident and Staff Physician (December 1981, 60–62) with permission.)

BONE RESORBING AGENTS

PTH
1,25D3
PGE2
PGI2
IL-1
TNF- α
TNF- β

EGF, TGF- α
PDGF, TGF- β
IL-1, TNF- α
Complement
Bradykinin
Thrombin
Mechanical stress

Endotoxin
Muramyl dipept.
Vitamin A
Thyroxine
Glucocorticoids
Acidosis

PROGENITORS AND PREOSTEOCLASTS

CT
INF- γ

Glucocorticoids
Oestrogens

'RESTING' OSTEOCLAST

'ACTIVATION FACTOR'
EXPOSURE OF MINERAL

CT
PGE2
PGI2
CGRP

OSTEOBLAST

Cell retraction

Collagenase
Degradation of
Non-mineralized
Collagen

'ACTIVATED' OSTEOCLAST

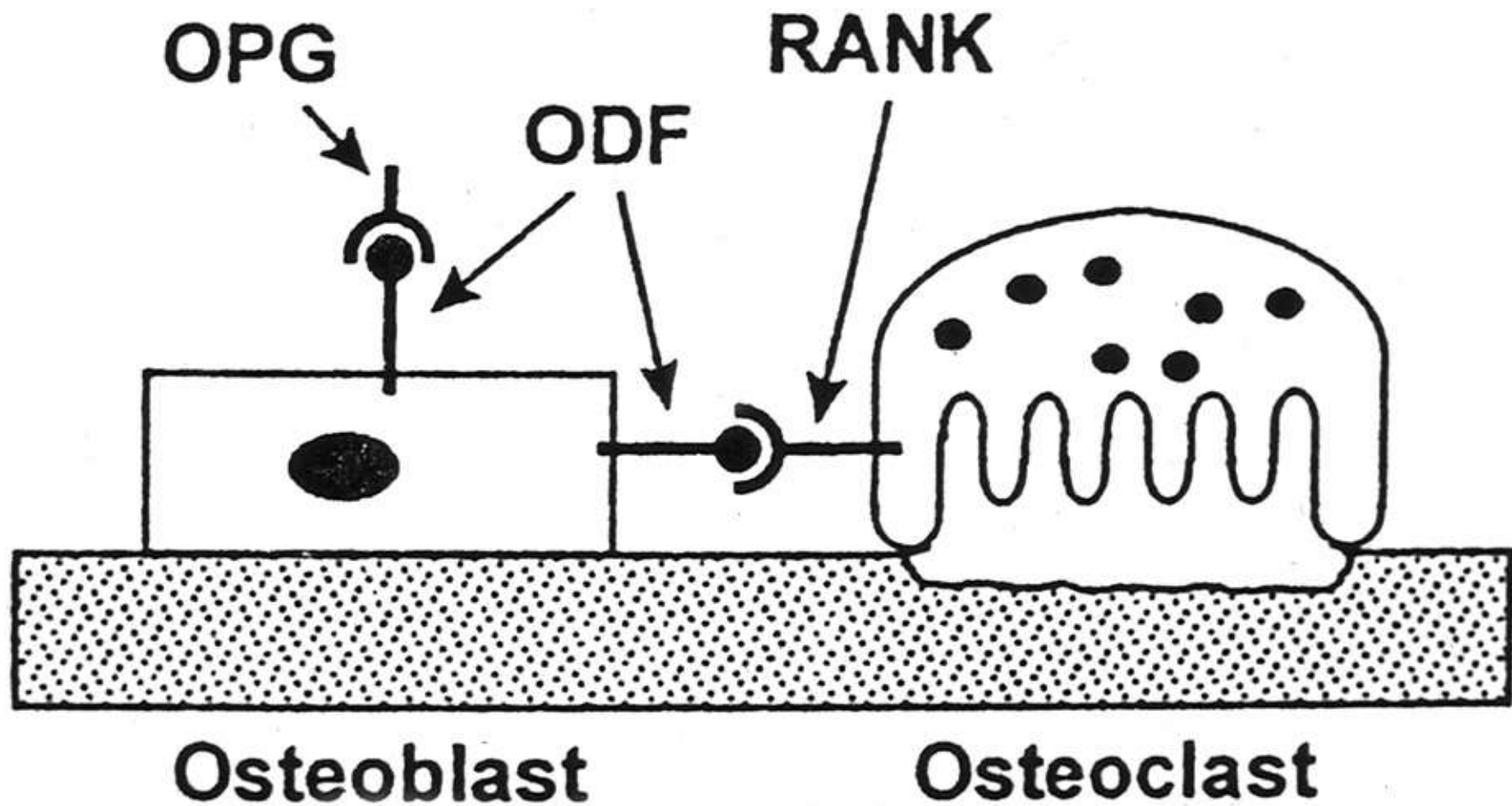
Ruffled Border

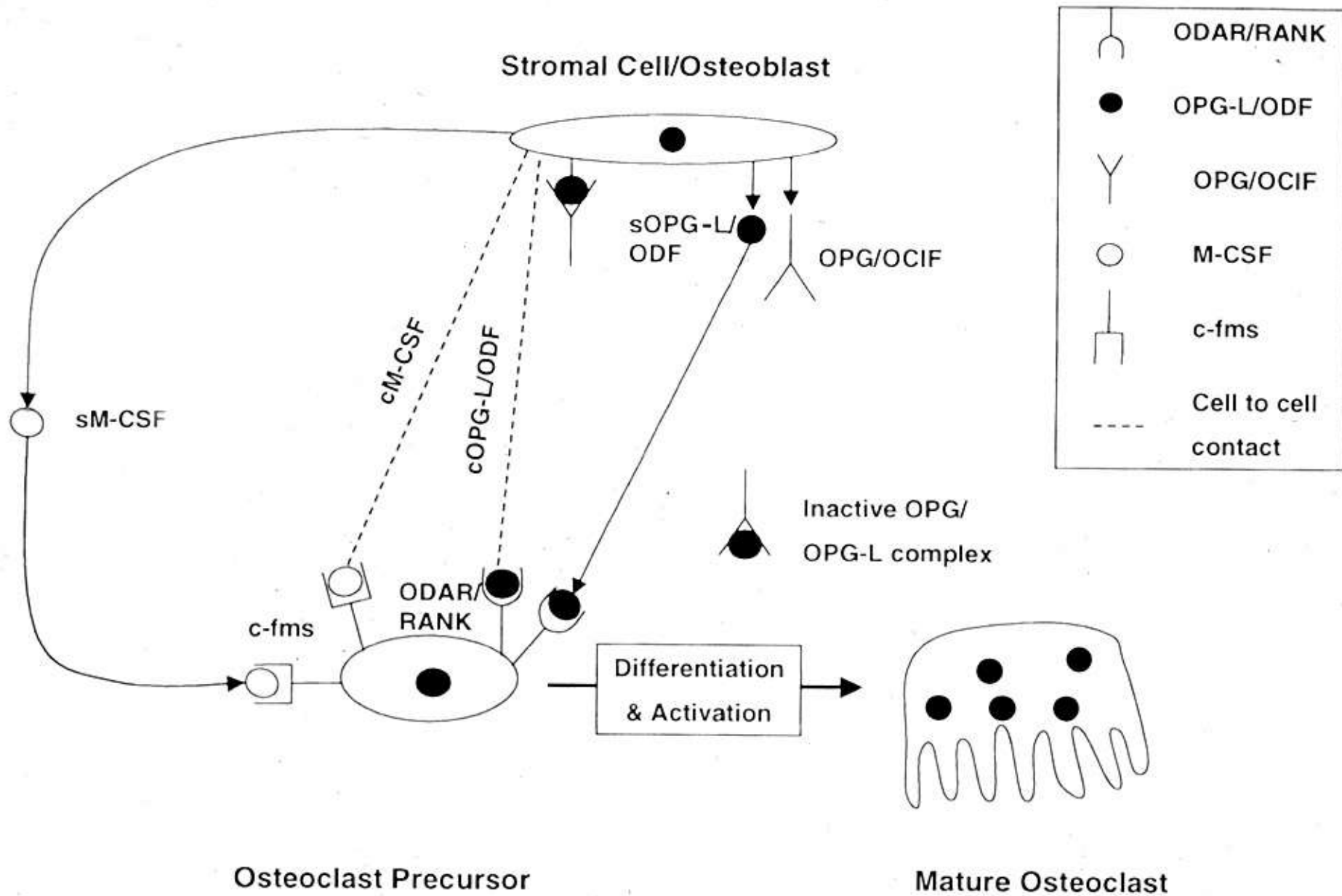
Sealing Zone

Segregated
Extracellular
Resorption Zone

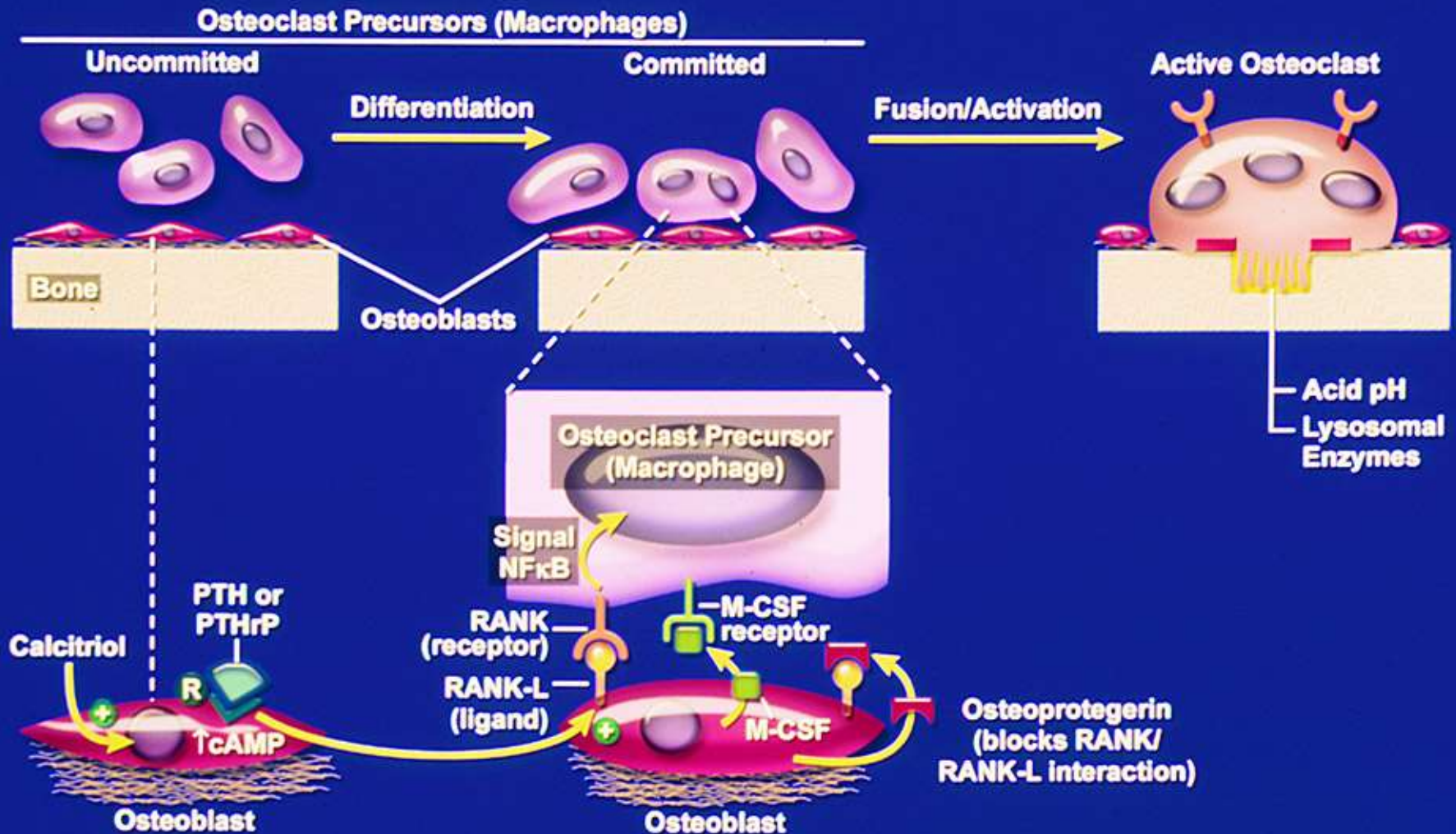
Non-Mineralized Collagen

Mineralized Bone Matrix

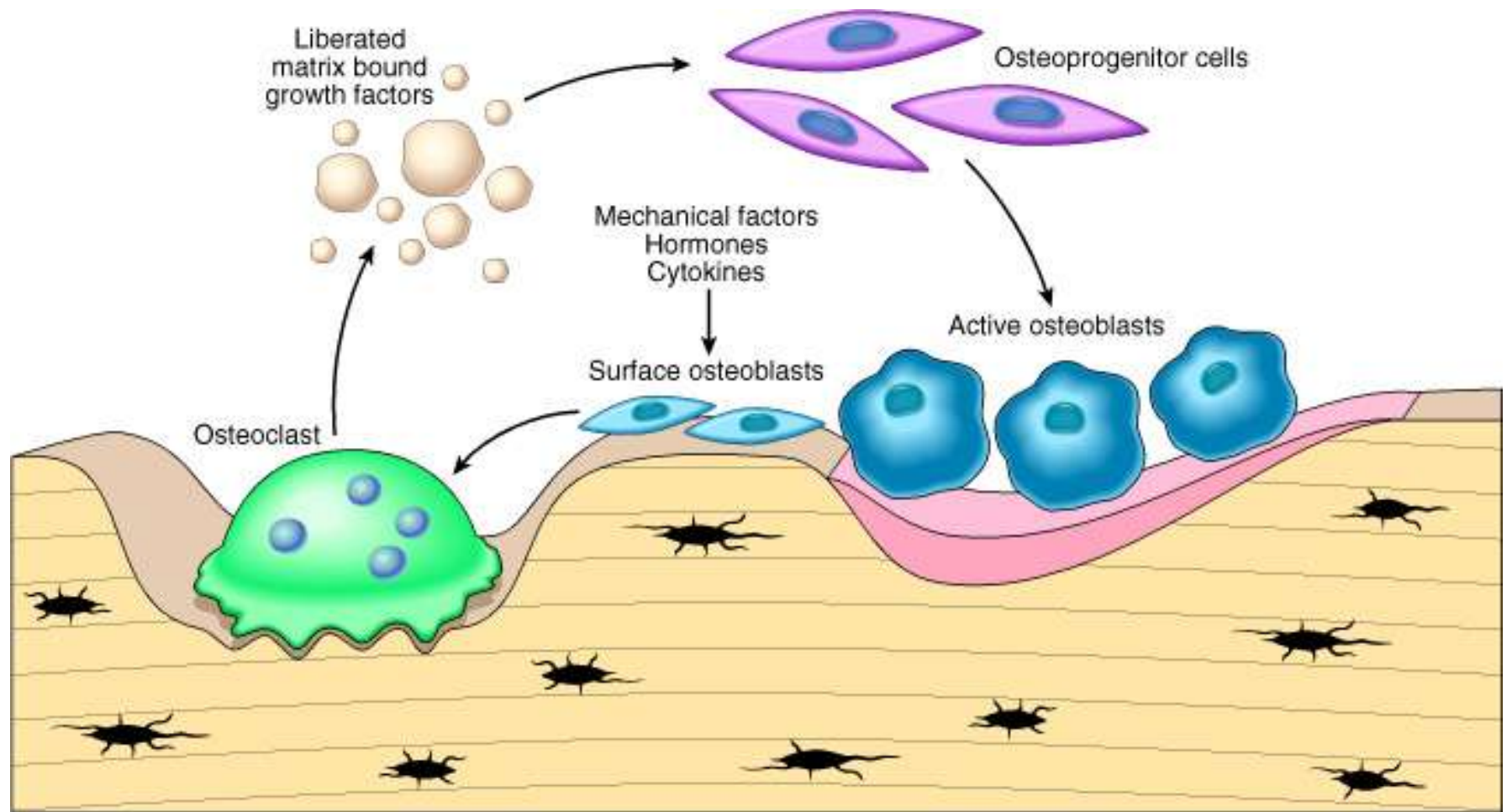




Cellular Control of Bone Resorption



RANK-L = Receptor Activator NF- κ B ligand
M-CSF = Macrophage Colony-Stimulating Factor
R = Receptor for PTH



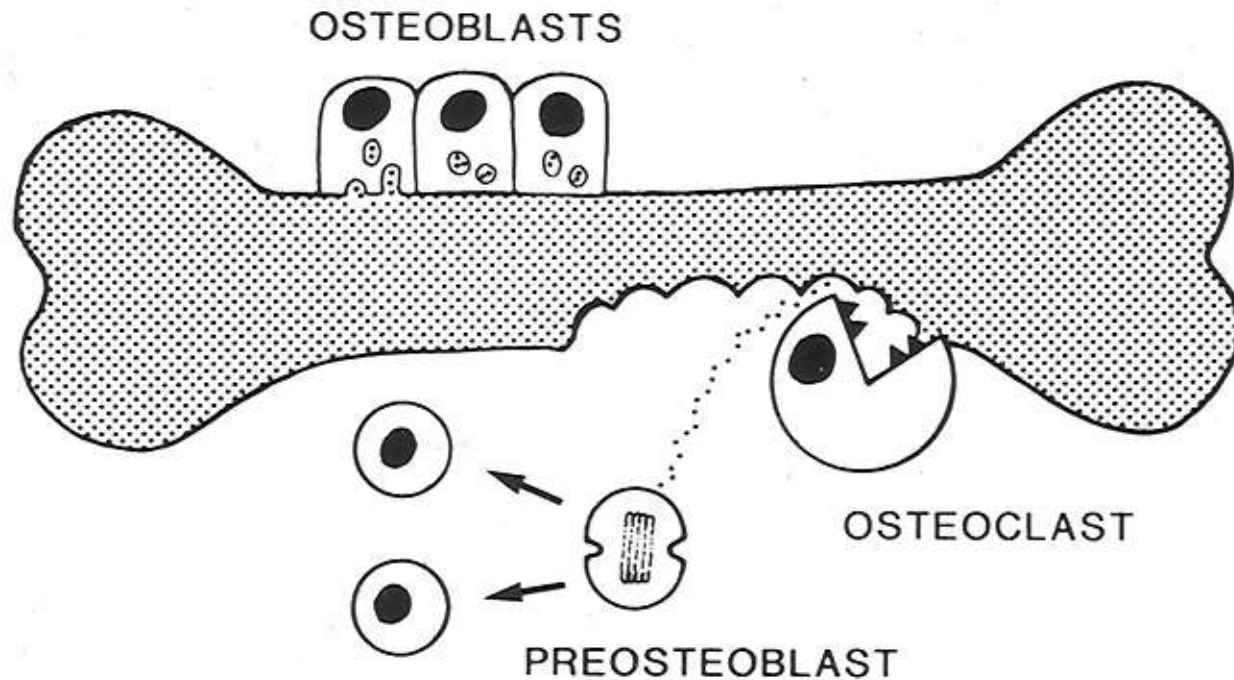


Fig 5. Model of osteoblast-derived mitogenic activity as a delayed paracrine effector of bone volume, illustrating how mitogenic activity might be synthesized by osteoblasts, deposited in bone, and released by osteoclastic resorption to promote the proliferation of osteoprogenitor cells, increasing osteoblast number and thereby effecting site-specific bone replacement.

Transforming Growth Factor Beta

- * produced by osteoblasts**
- * activated by acidification**
- * released by osteoclasts**
- * inhibitory to osteoclasts**
- * stimulatory to osteoblasts**

What cell really completes the
resorption?

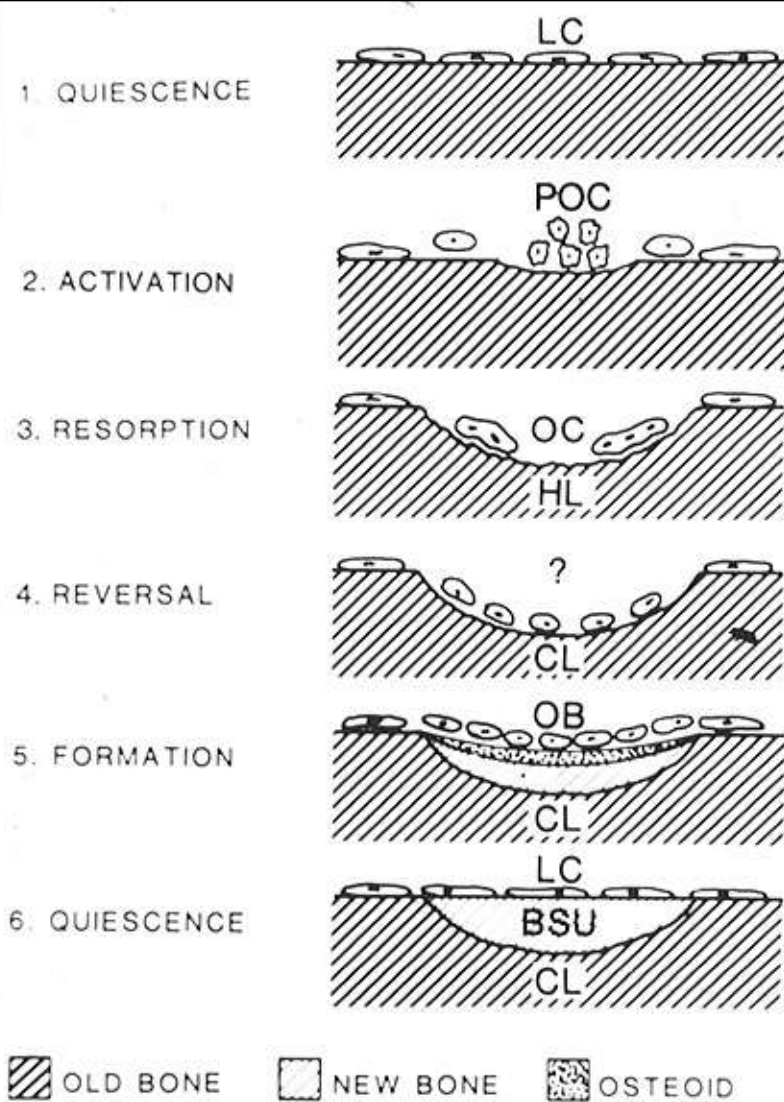


Fig. 1. The normal remodeling sequence in adult bone. Schematic representation of successive events on the endosteal surface. LC—lining cell. POC—preosteoclast (mononuclear). OC—osteoclast (multinucleated). HL—Howship's lacuna. CL—cement line. OB—osteoblast. BSU—bone structural unit. (Modified from Resident and Staff Physician (December 1981, 60–62) with permission.)

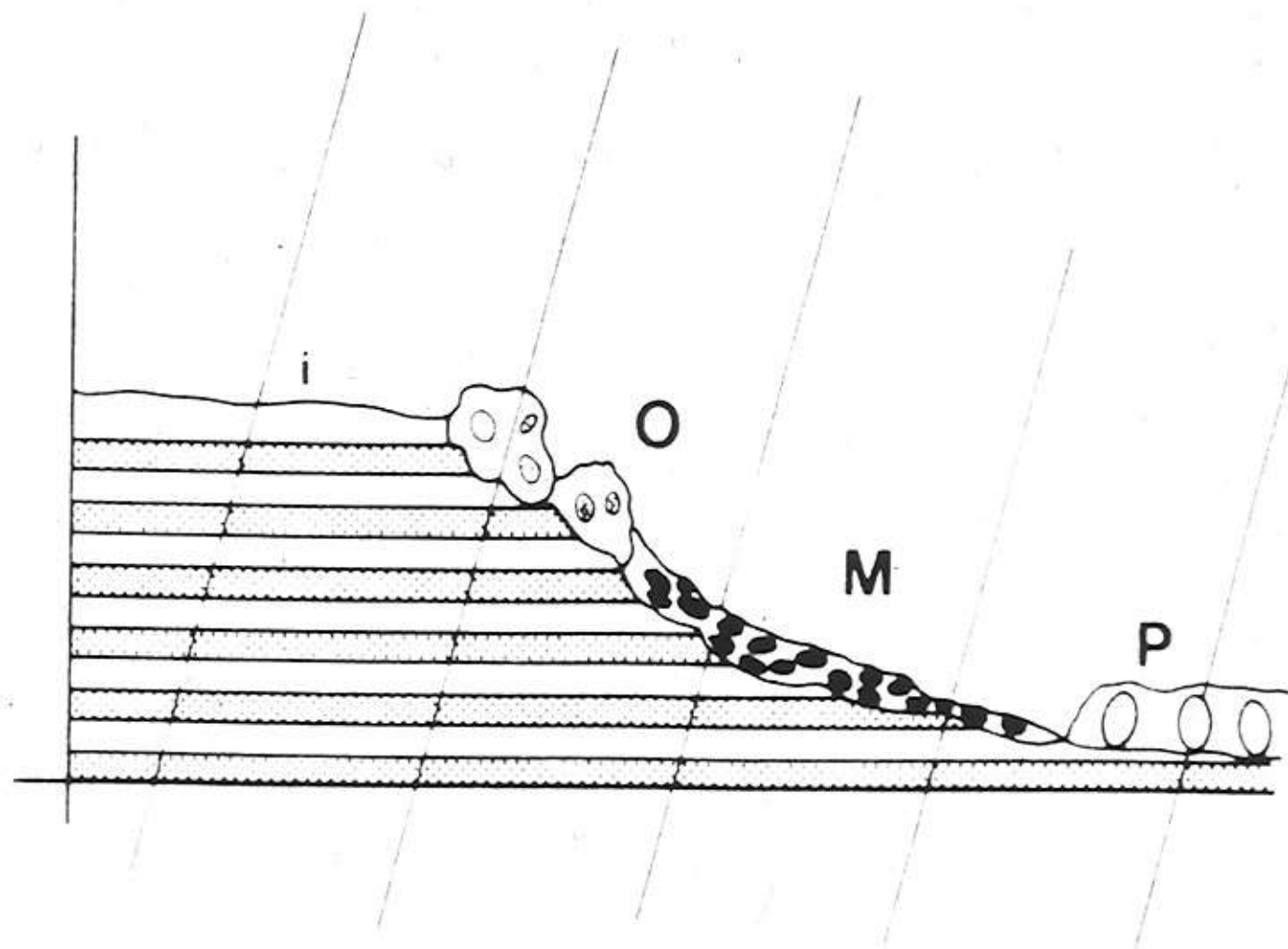


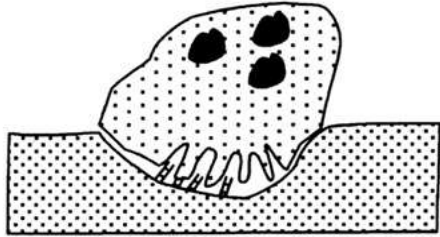
Fig. 1. Sampling design used in this paper. One testline intersects (*i*) with osteoclasts (O) at a depth of 2.5 laminae, two testlines intersect with mononuclear cells (M) at depths of 8 and 10 laminae, respectively, and one testline intersects with preosteoblast-like cells (P) at a depth of 11 laminae.



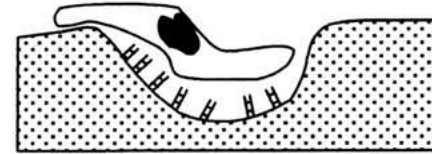
I. digestion of nonmineralized collagen



III. cleaning of the Howship's lacuna



II. osteoclastic bone resorption



IV. deposition of a thin layer of fibrillar collagen

FIG. 10. Schematic presentation of the proposed sequence of events involved in coupling bone resorption and bone formation. (I) Before osteoclastic attachment, bone lining cells “clean” the bone surface from protruding nonmineralized collagen fibrils. (II) Osteoclasts attach to the bone surface, digest bone, and withdraw. (III) Bone lining cells occupy the Howship's lacunae and digest the leftovers. (IV) Bone lining cells form a cement line and deposit a thin layer of collagen at the bottom of the pit. This activity is probably followed by retrieval of the bone lining cell and formation of new bone by osteoblasts.

Cement Lines

- Proteoglycan rich
- Mineral rich
- Collagen poor

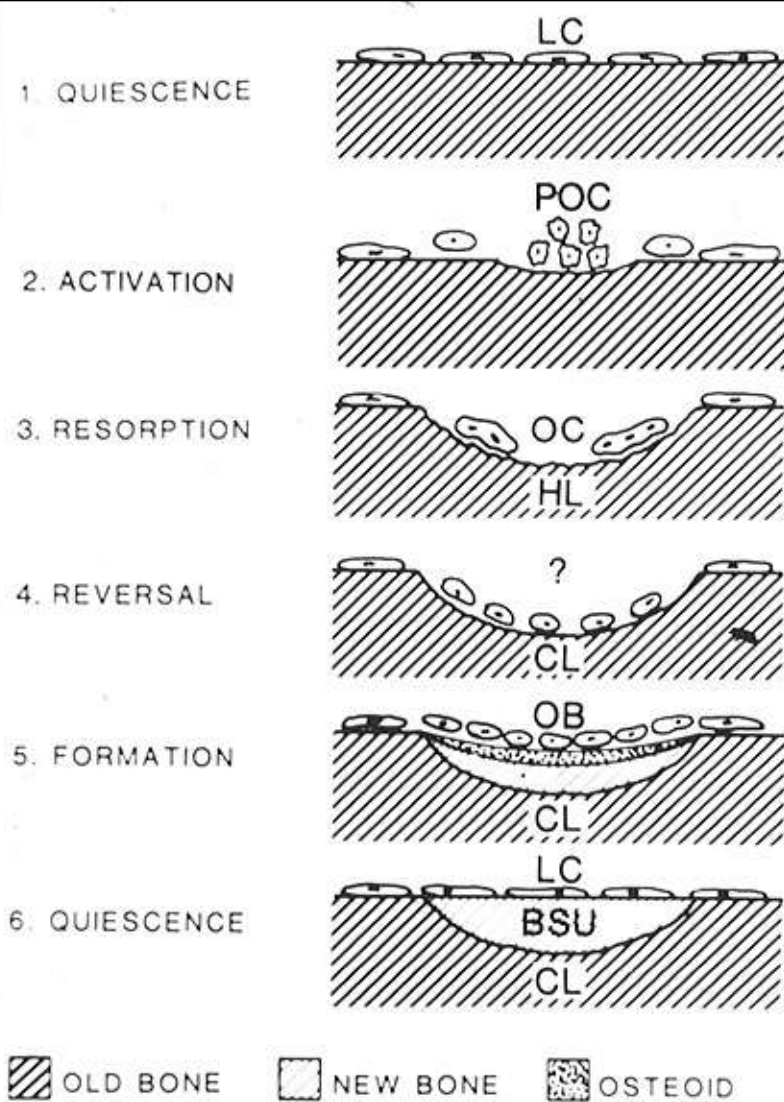
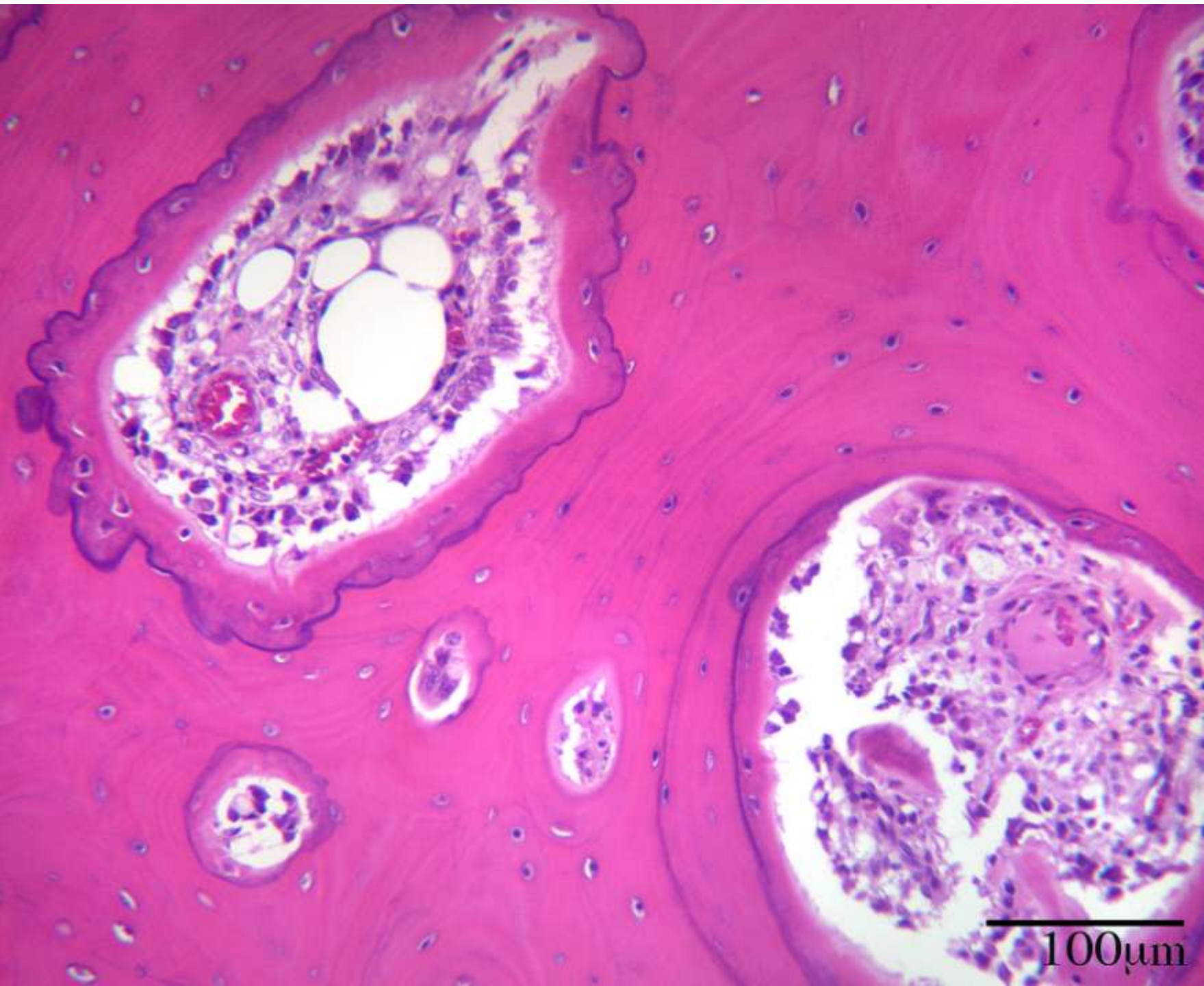
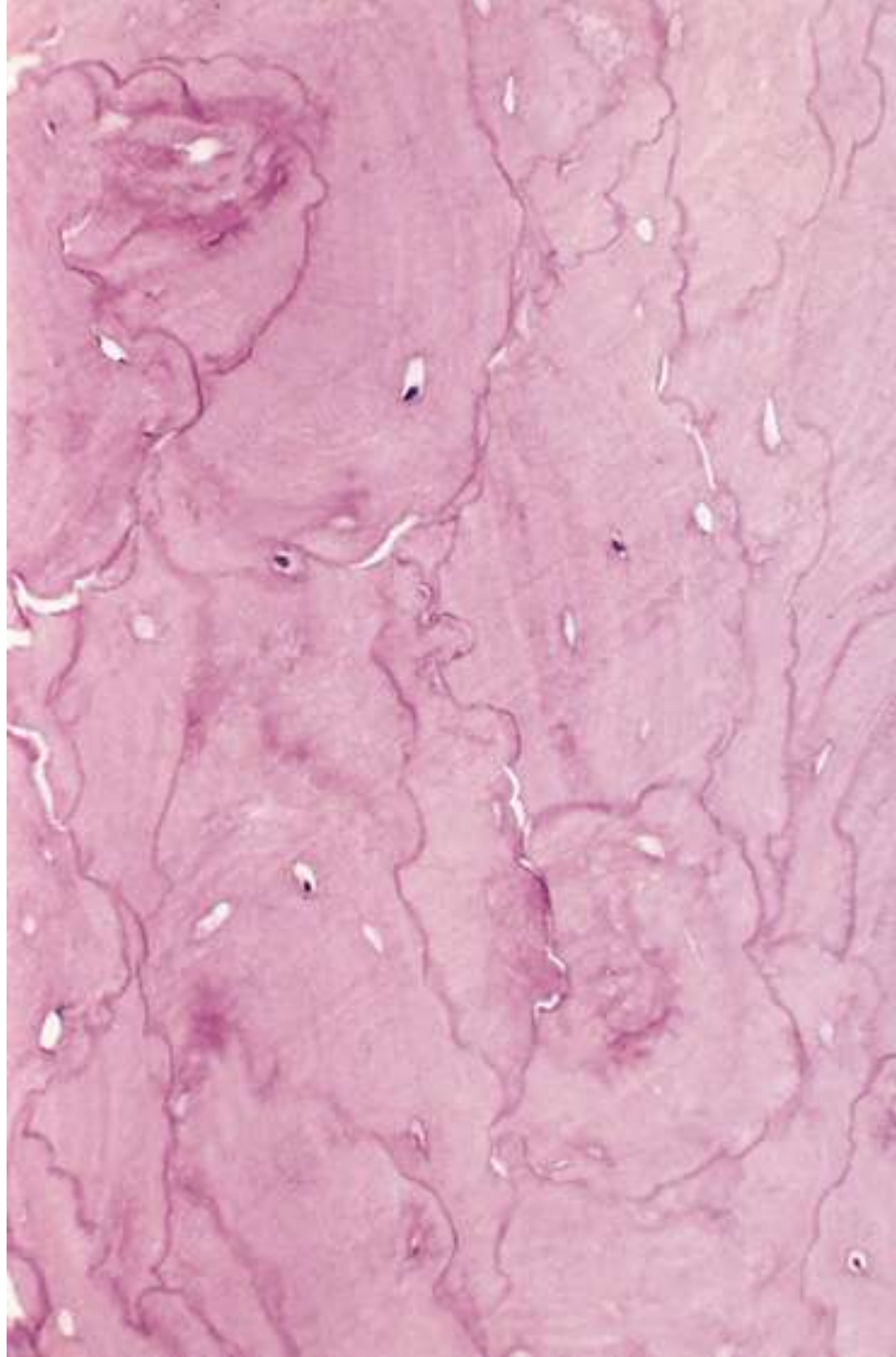


Fig. 1. The normal remodeling sequence in adult bone. Schematic representation of successive events on the endosteal surface. LC—lining cell. POC—preosteoclast (mononuclear). OC—osteoclast (multinucleated). HL—Howship's lacuna. CL—cement line. OB—osteoblast. BSU—bone structural unit. (Modified from Resident and Staff Physician (December 1981, 60–62) with permission.)





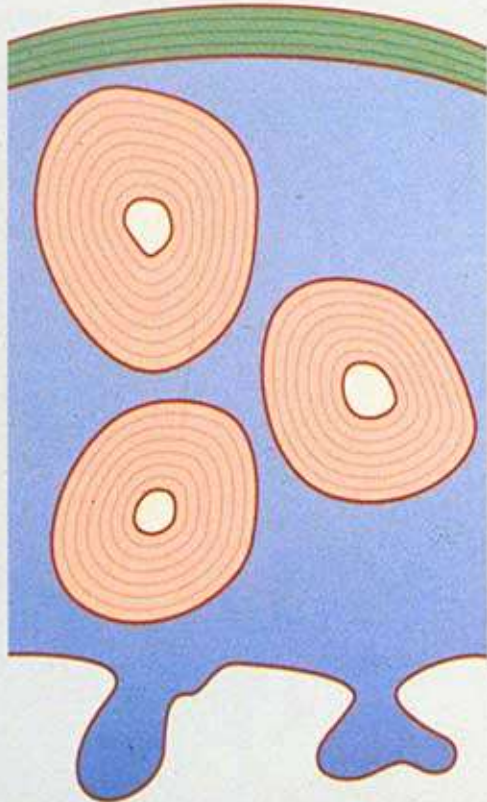
Slide 28.13

Remodeling Units

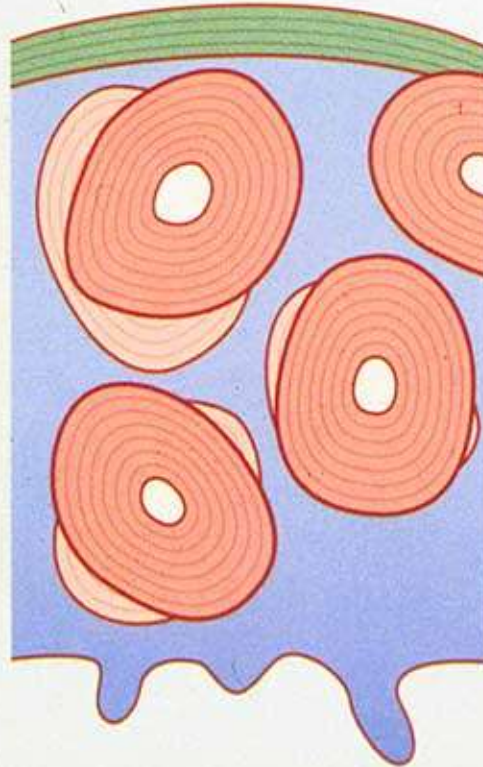
Osteon – cortical bone

Basic Structural Unit (BSU) –
trabecular bone

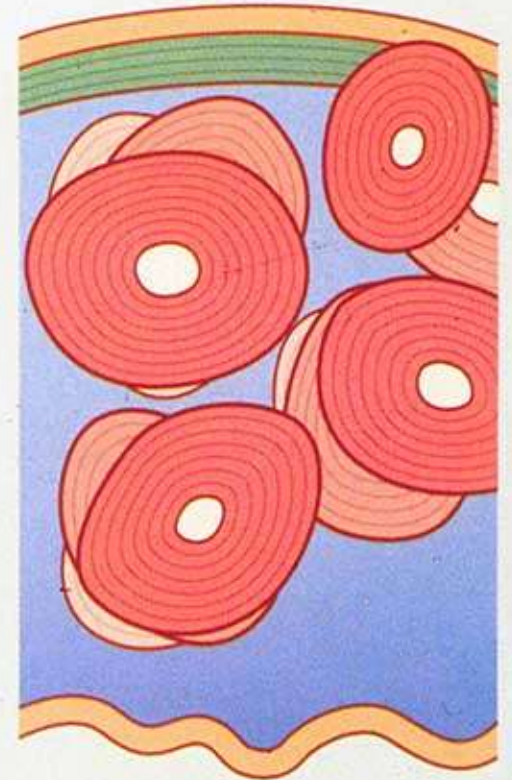
A First-generation Haversian system



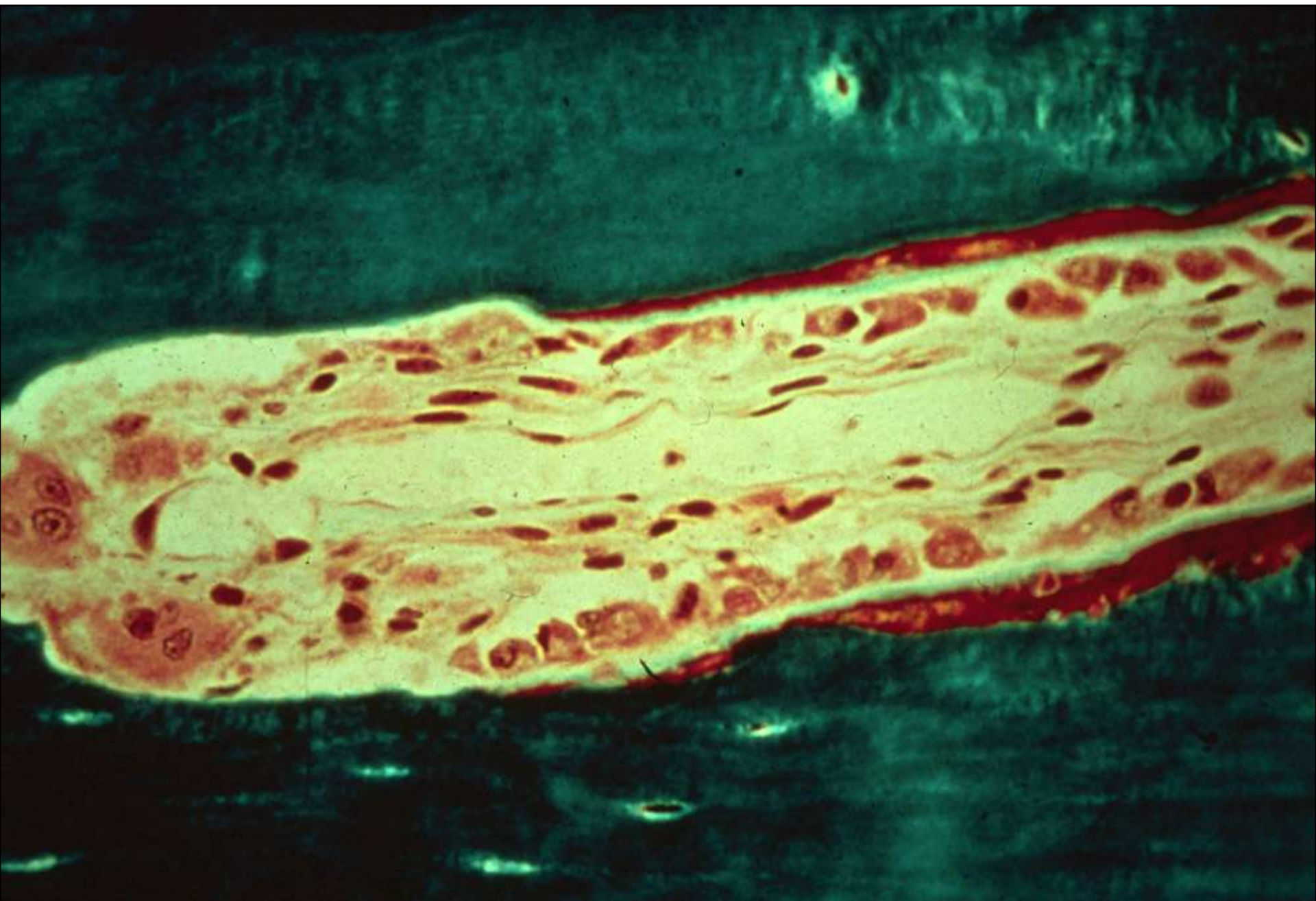
B Second-generation Haversian system

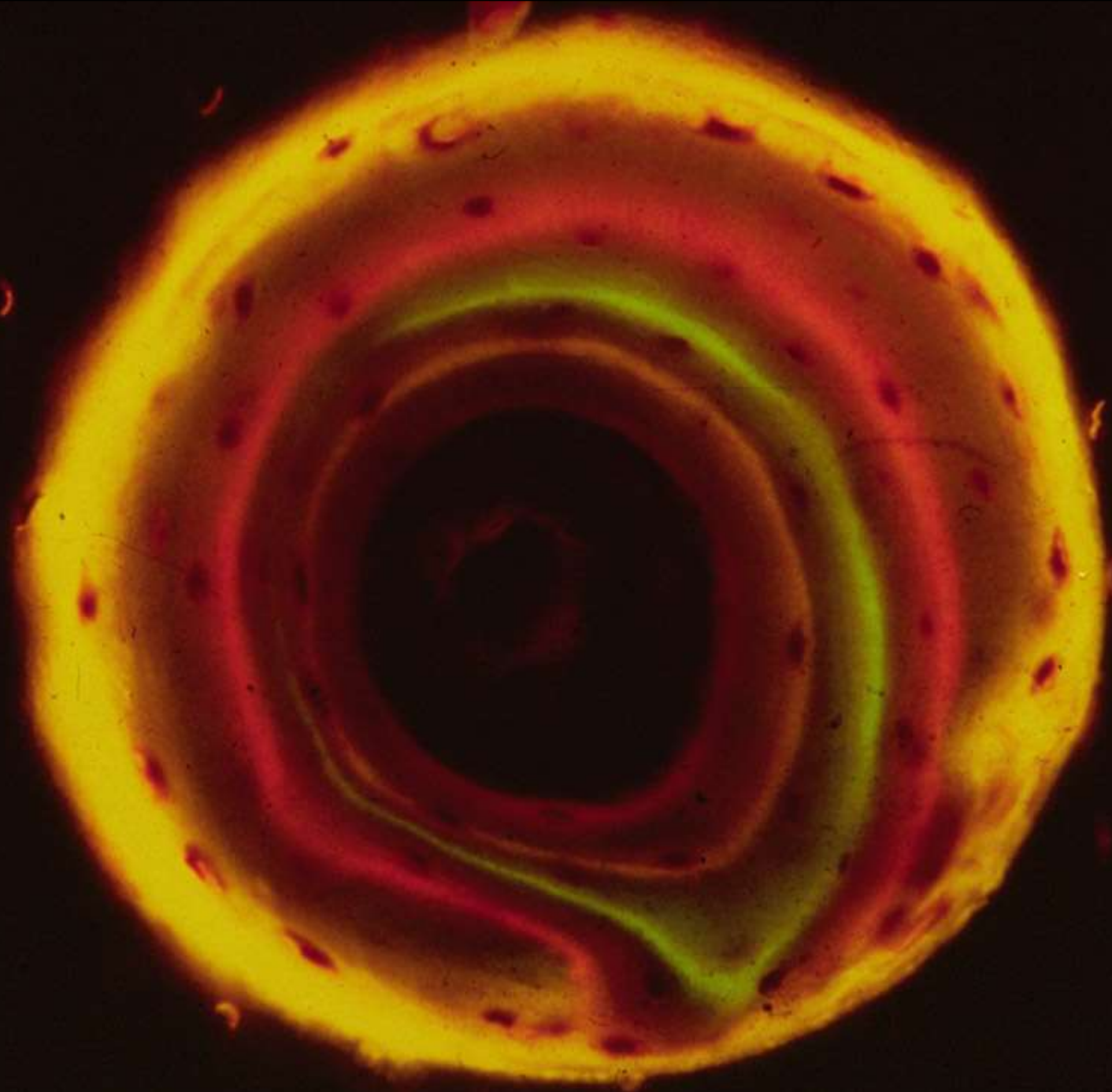


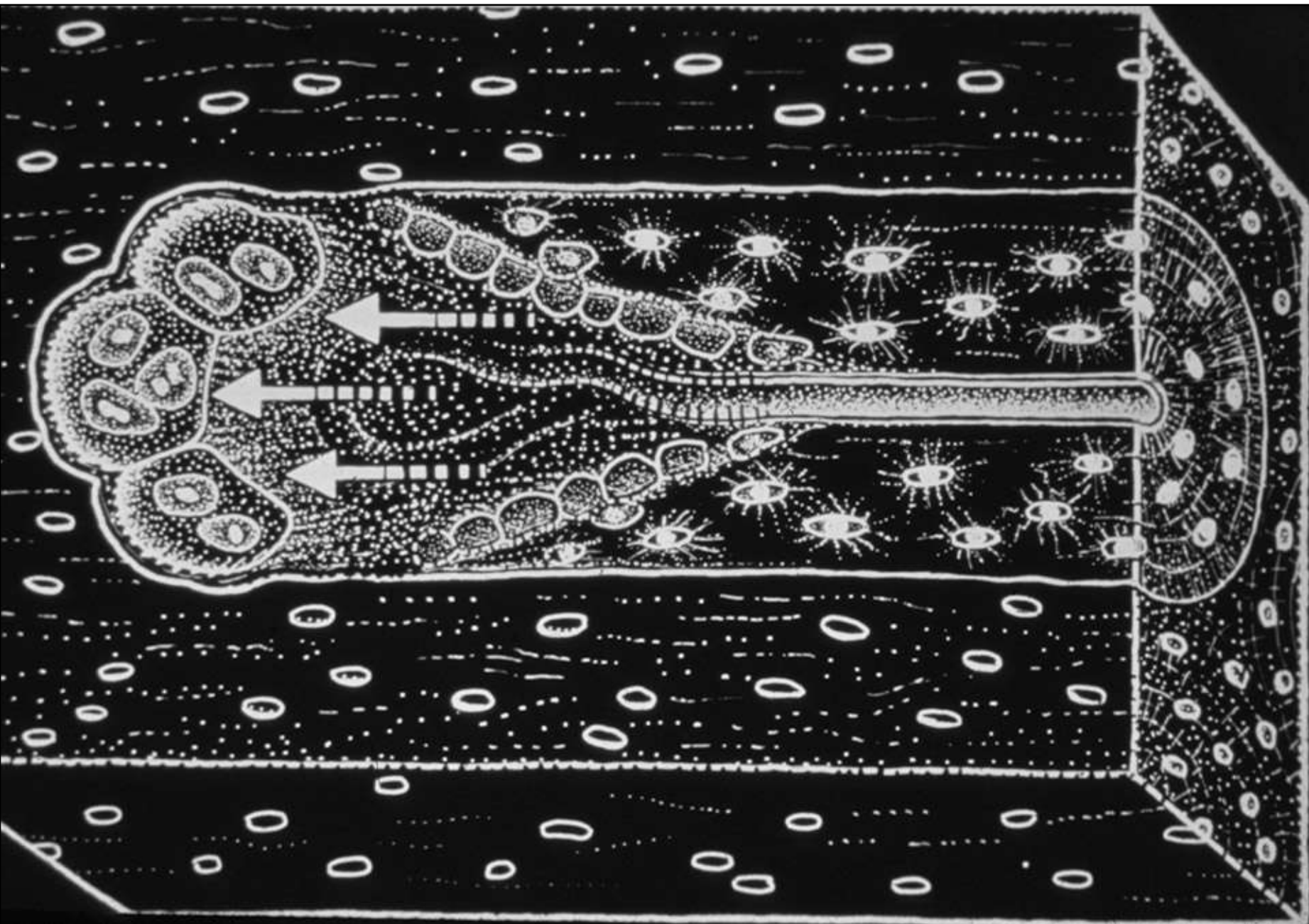
C Third-generation Haversian system

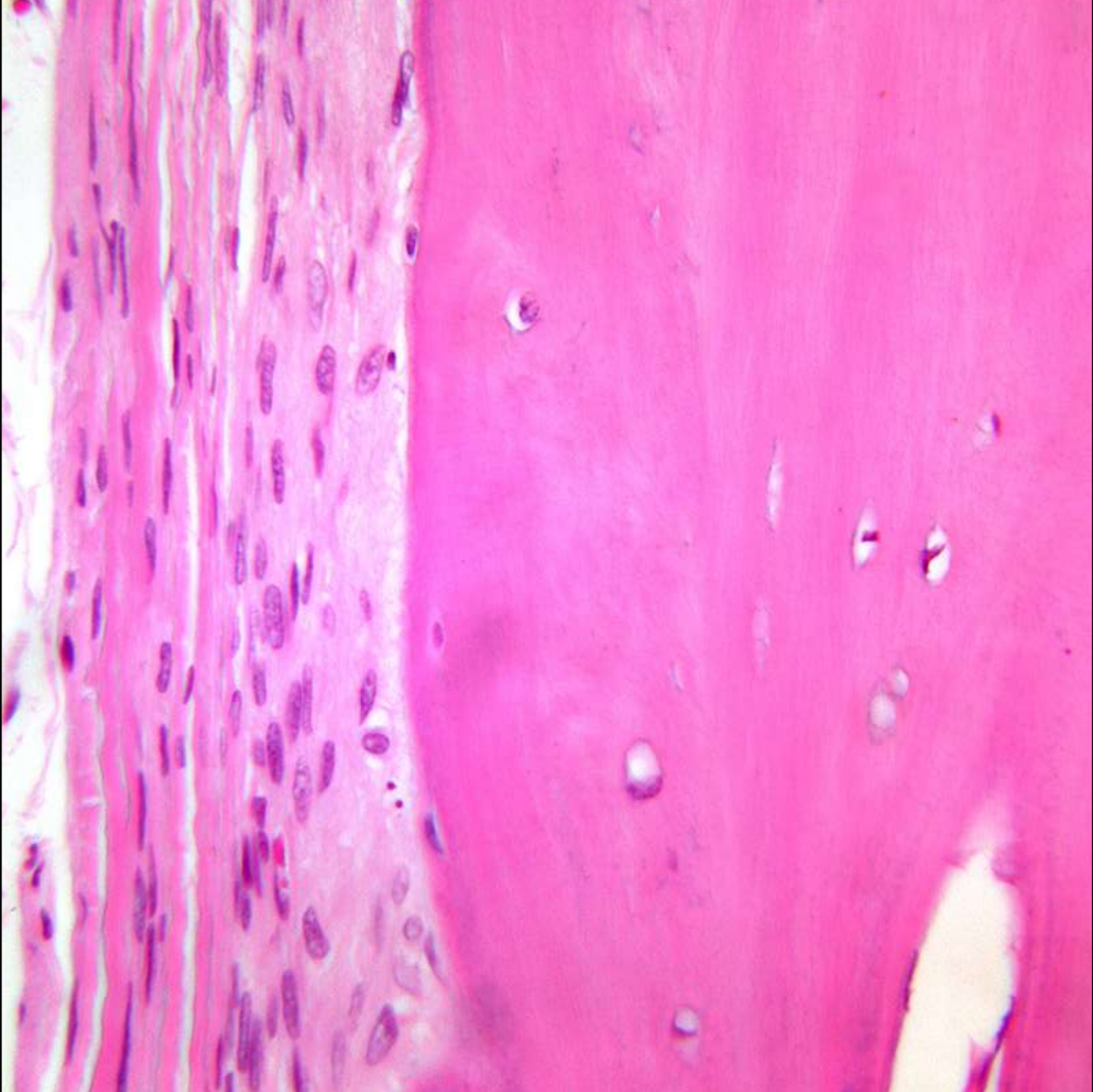


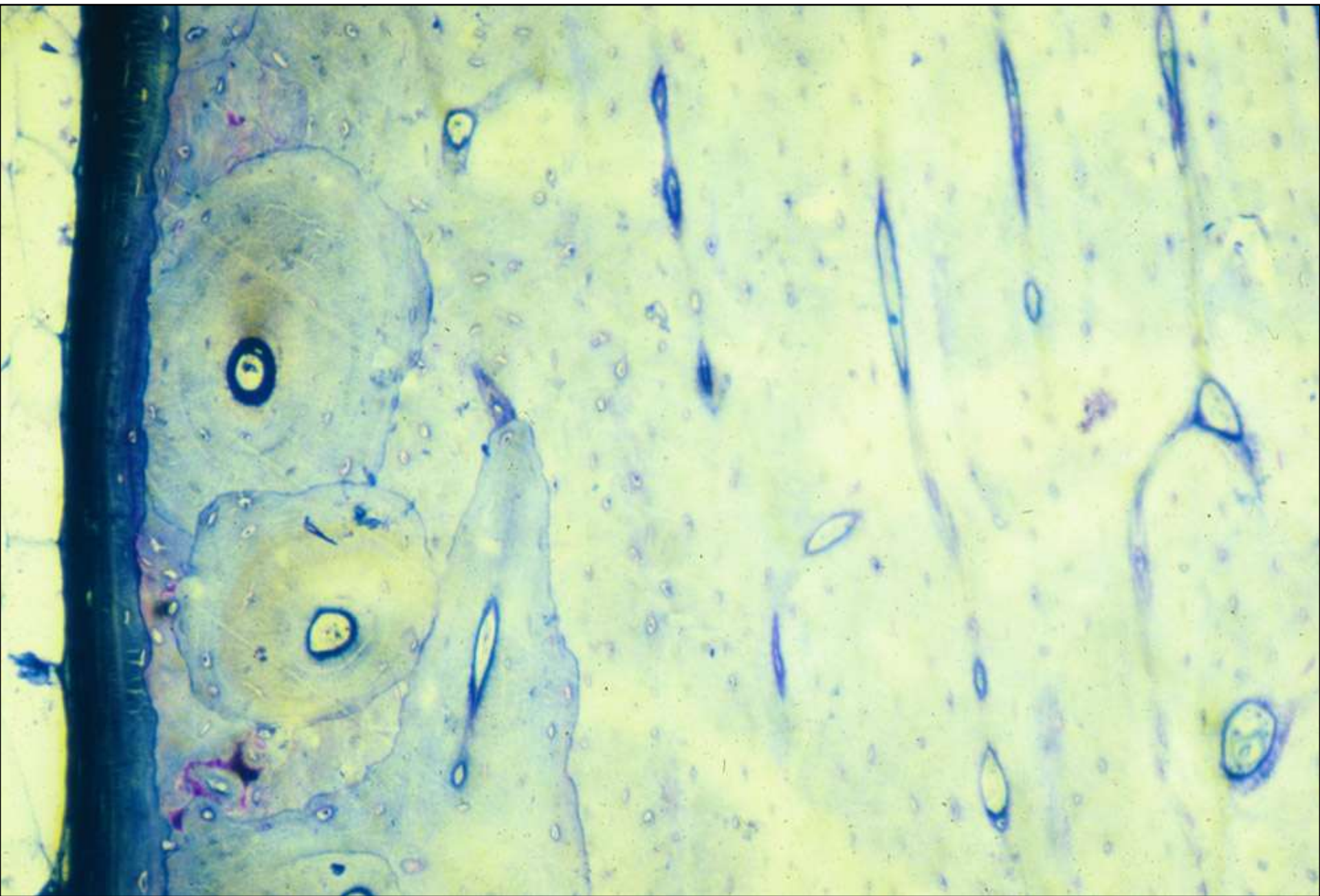


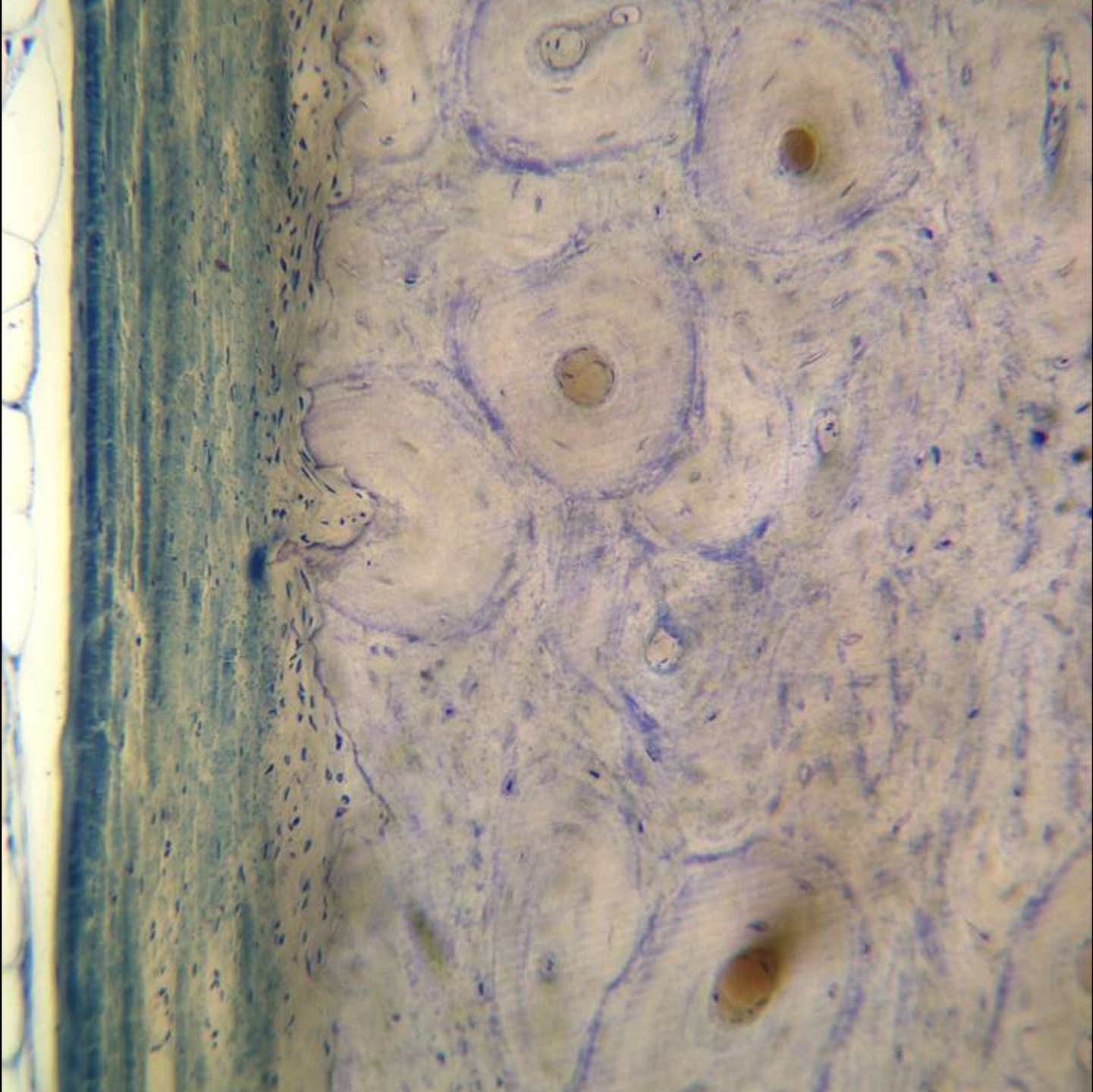


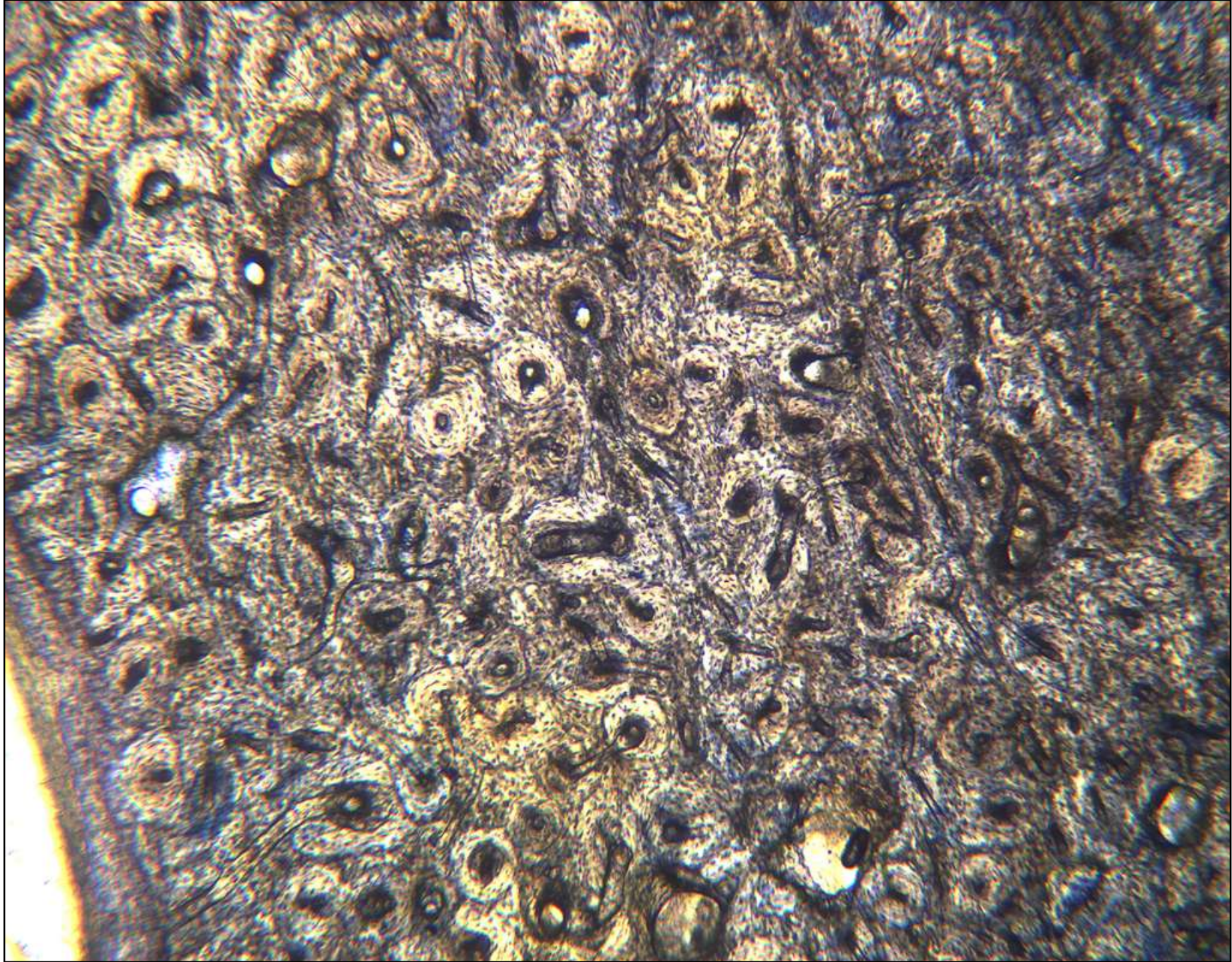


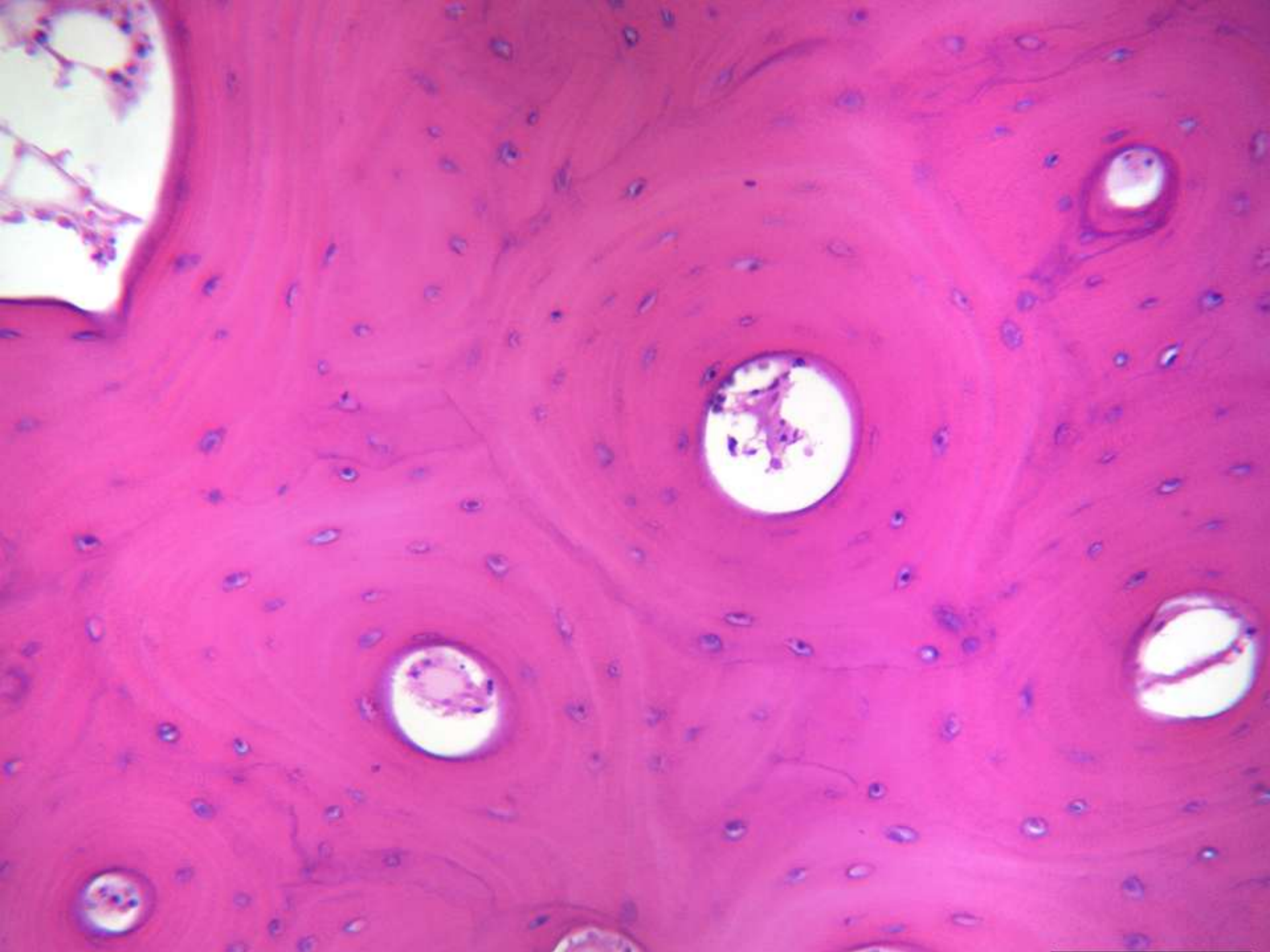




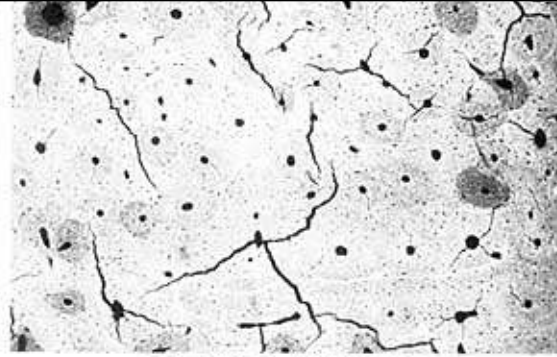




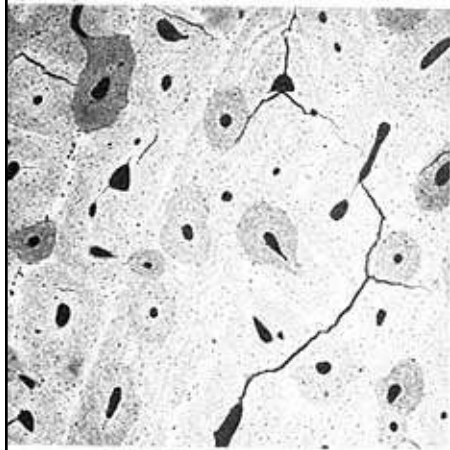




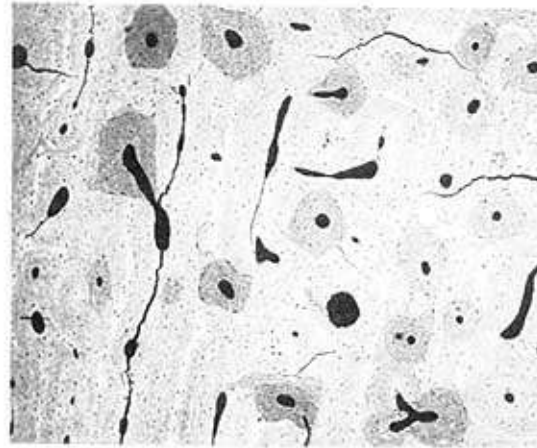




CRANIAL (TENSION)

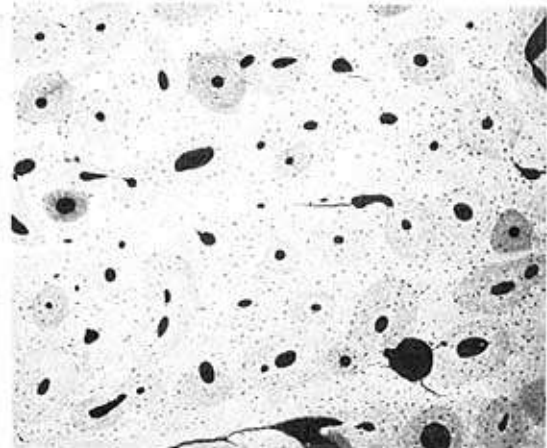


MEDIAL

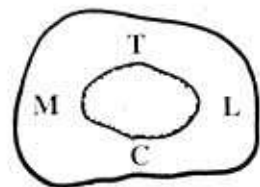


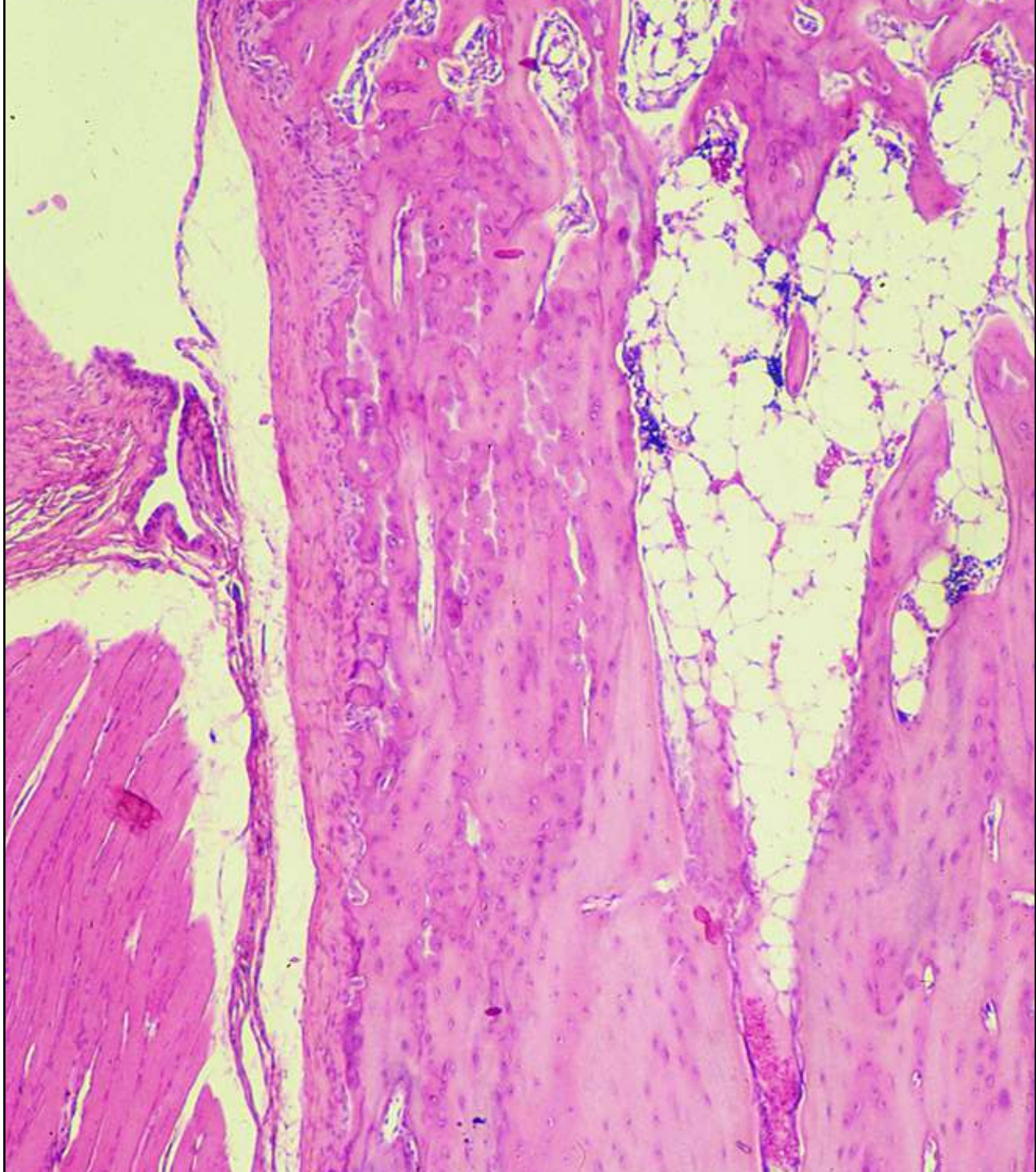
LATERAL

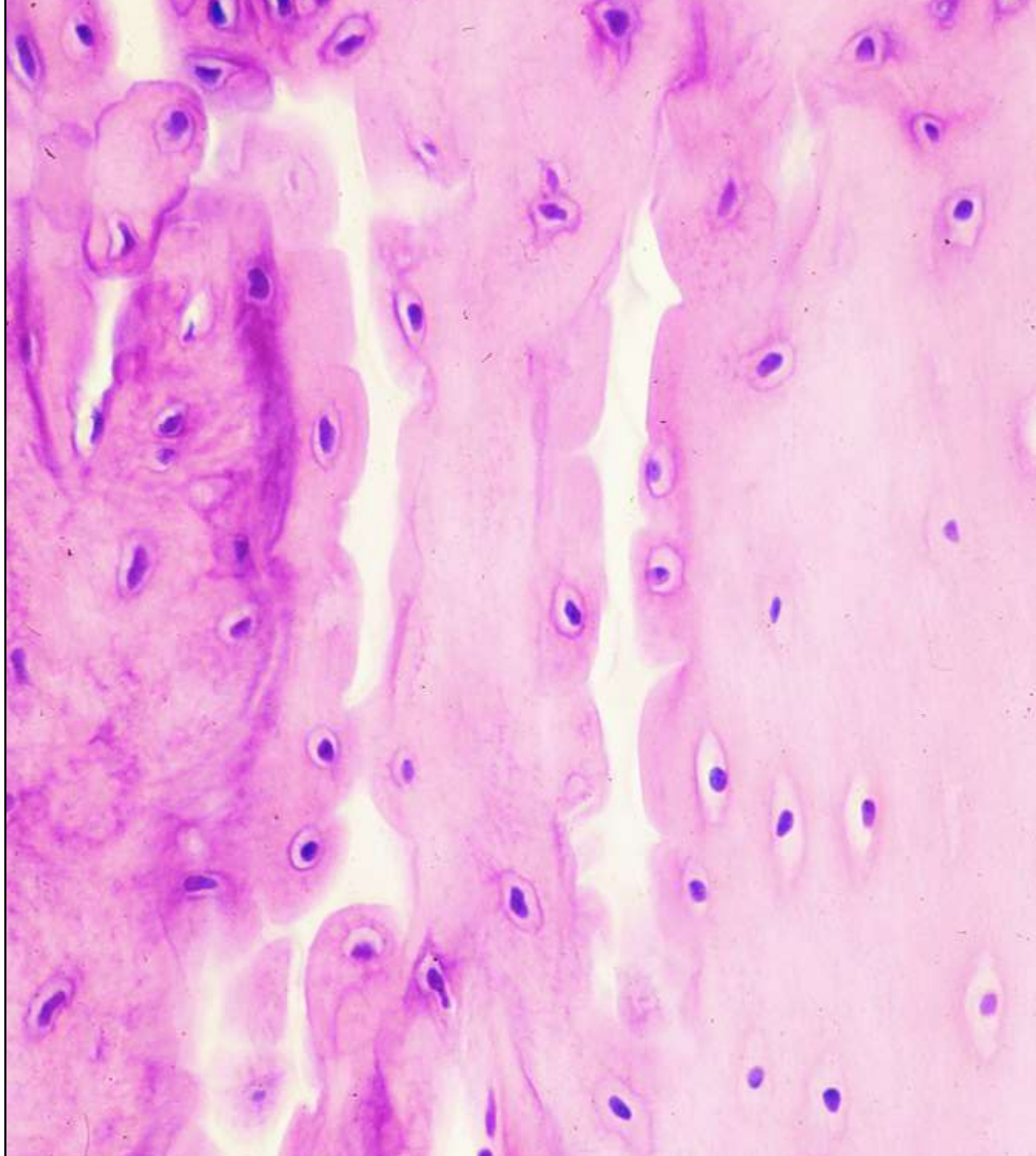
500µm

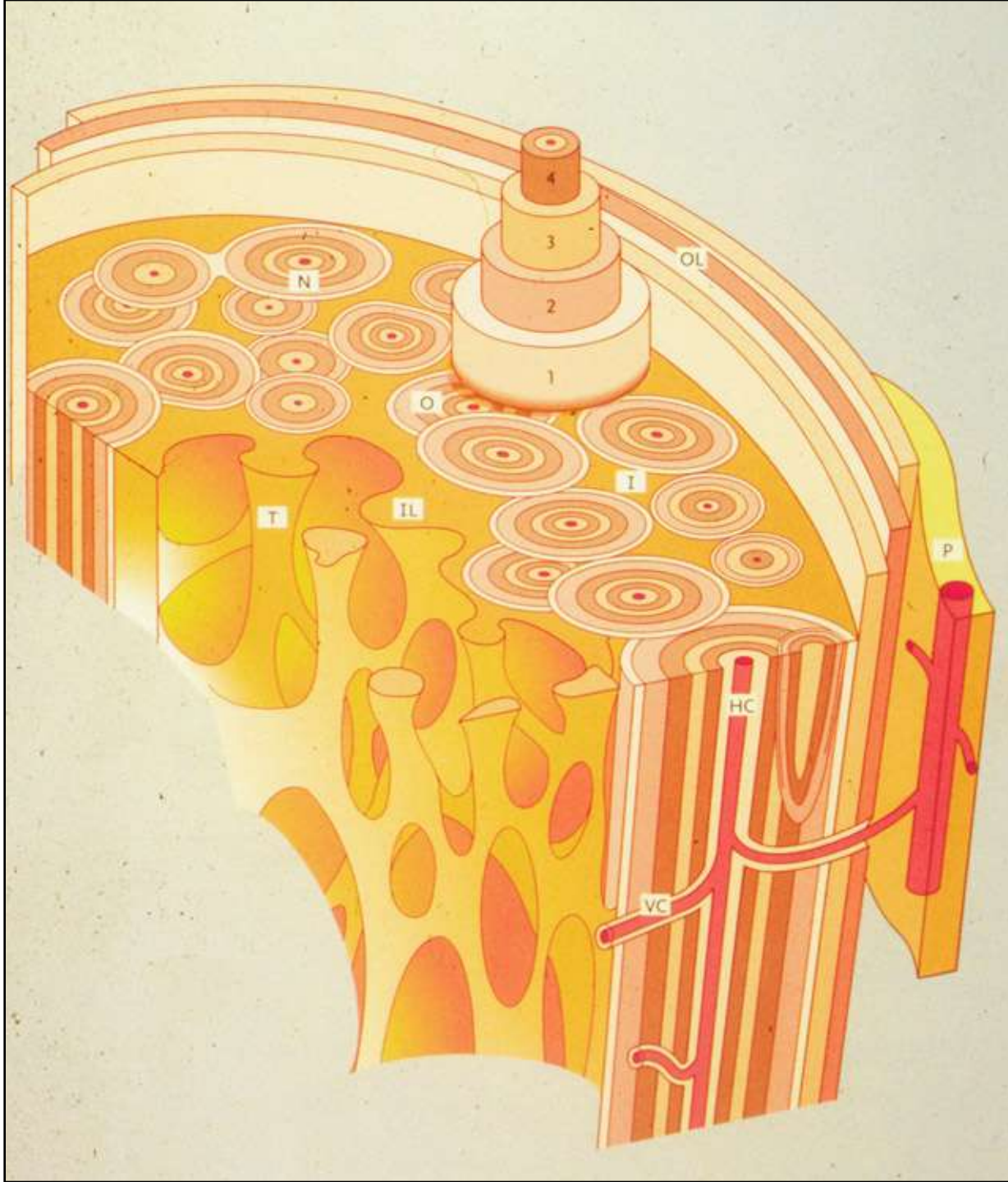


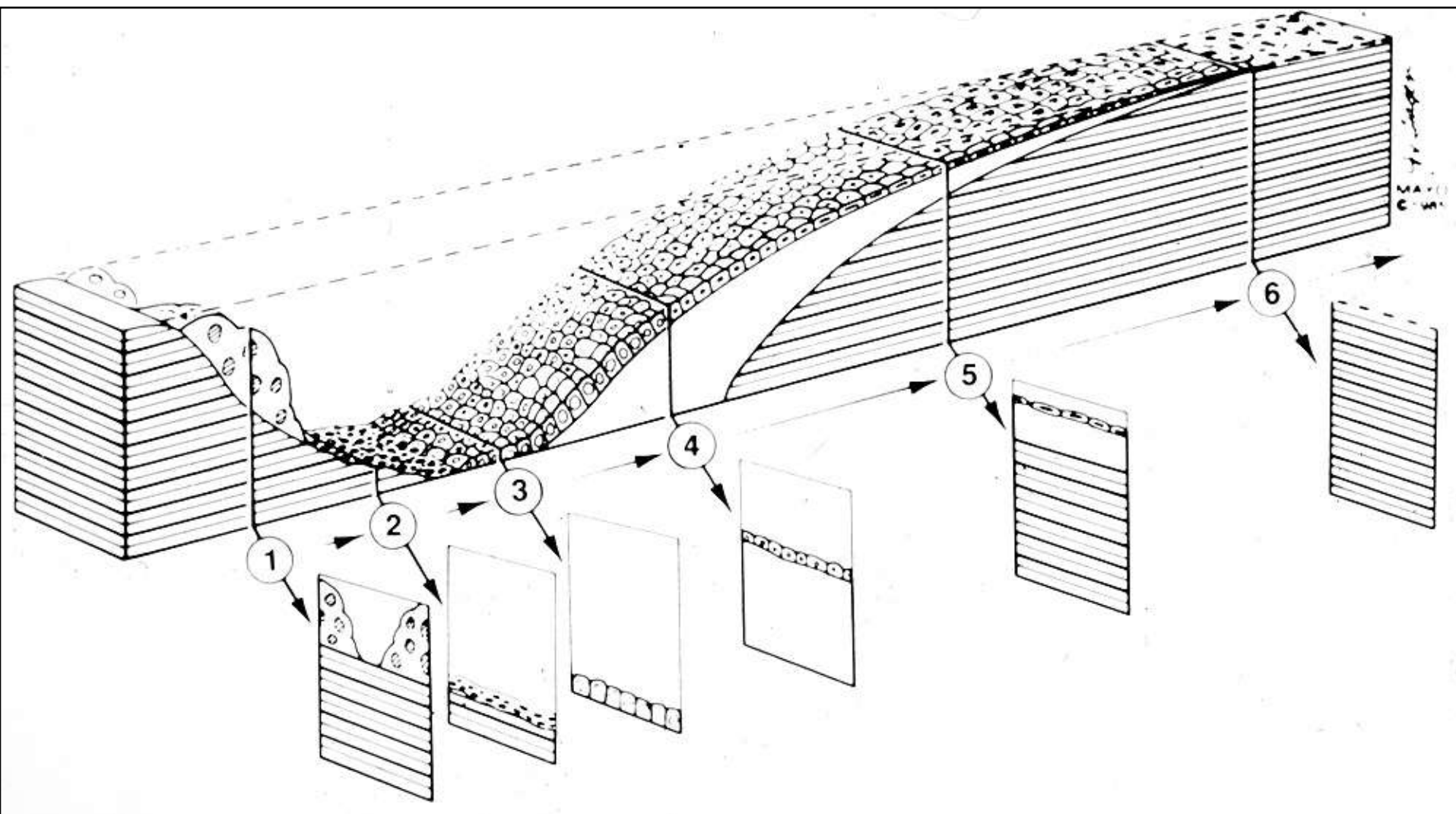
CAUDAL (COMPRESSION)

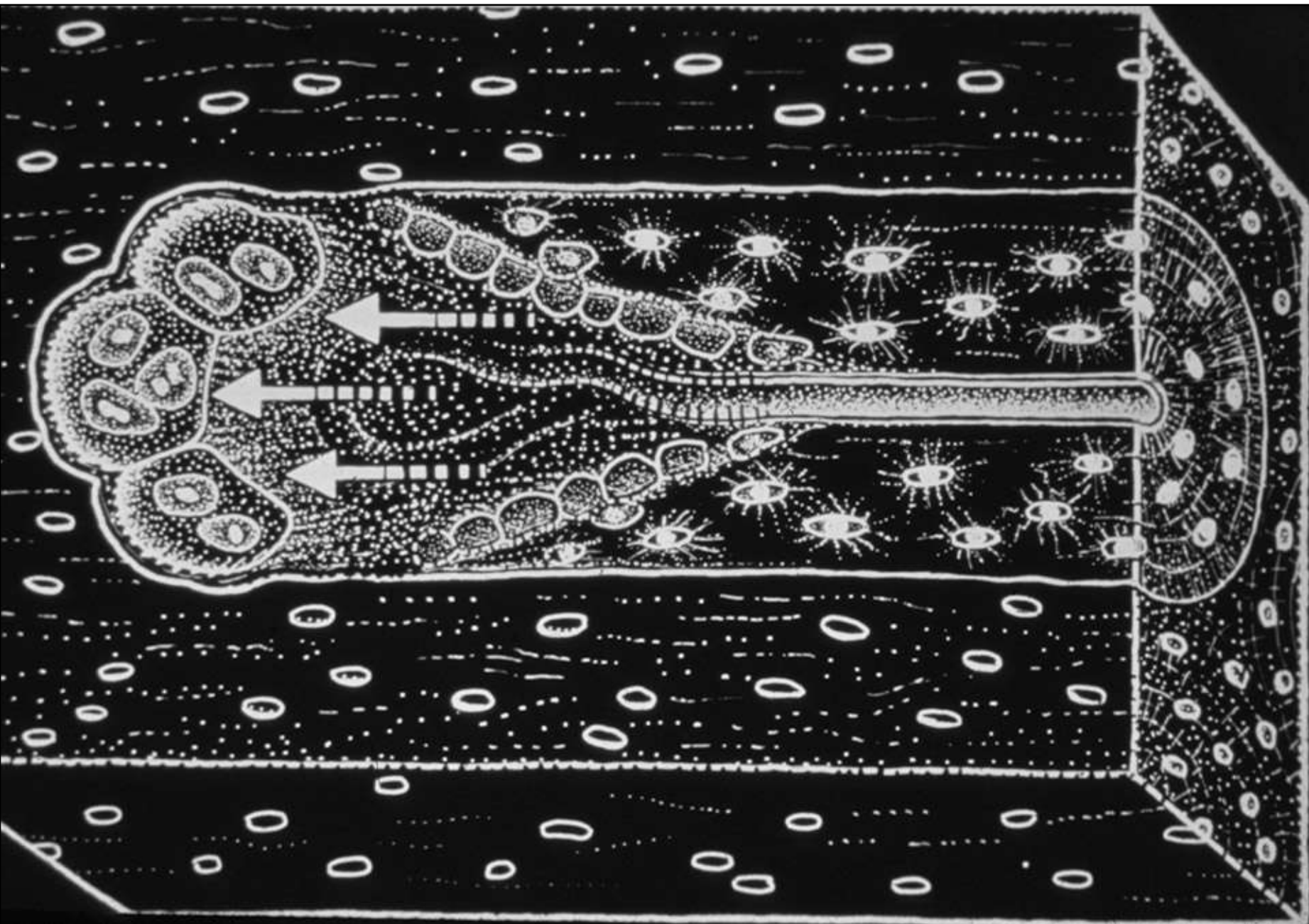


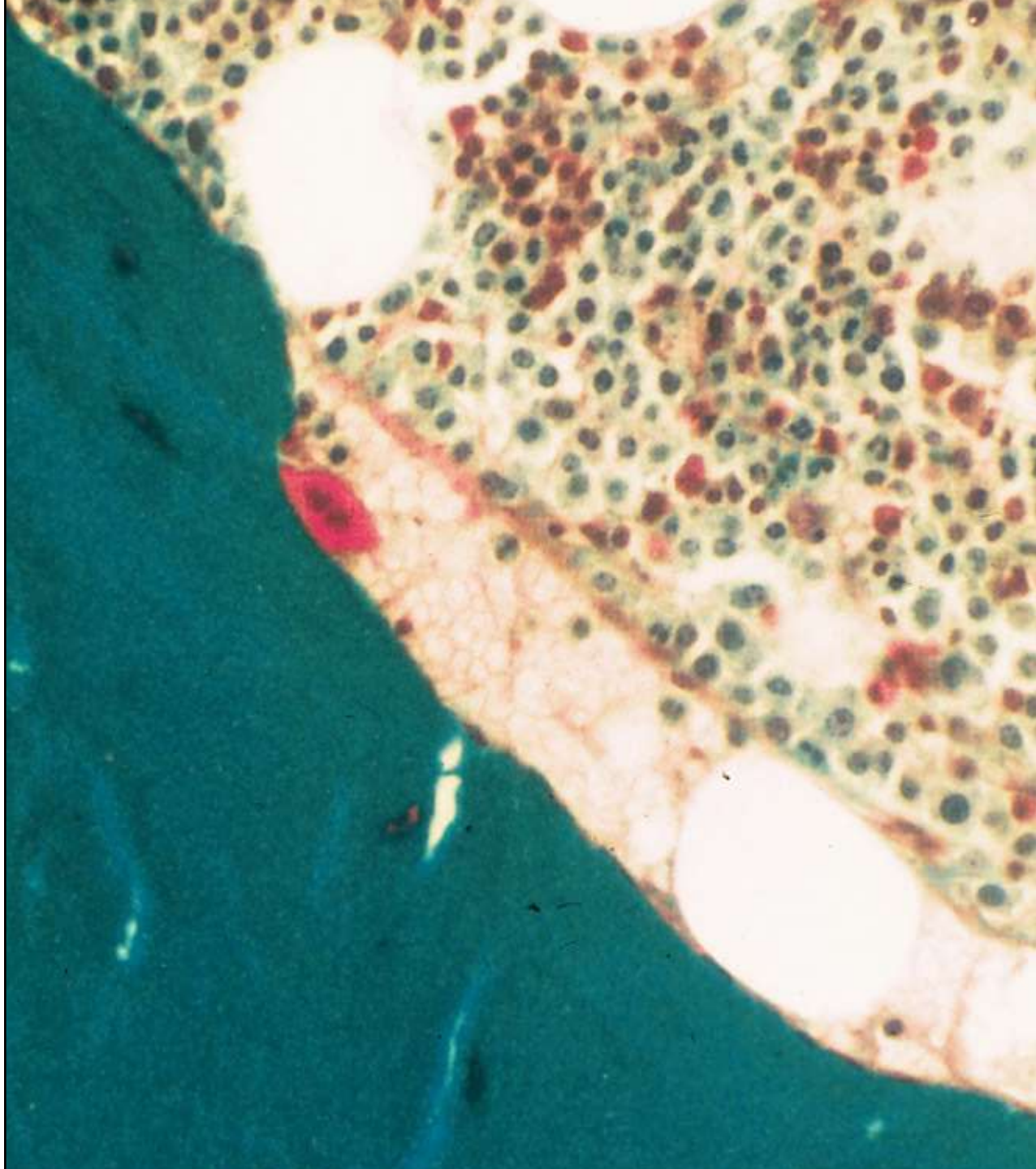


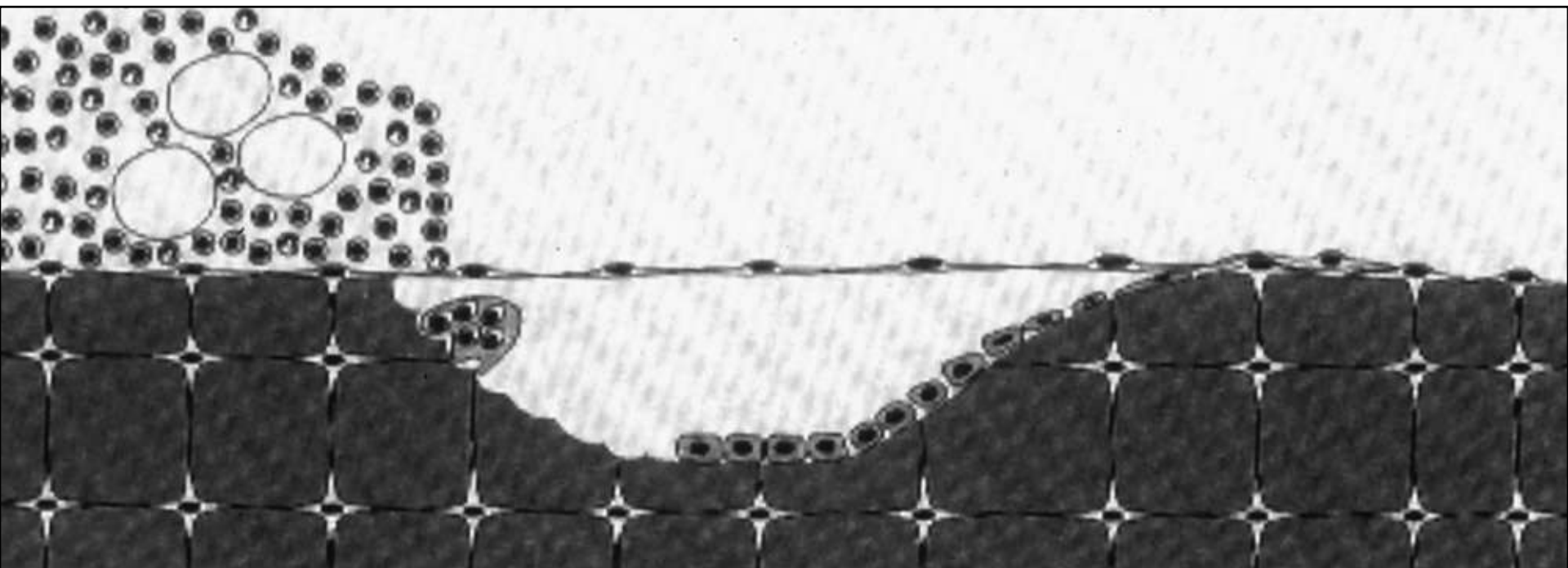


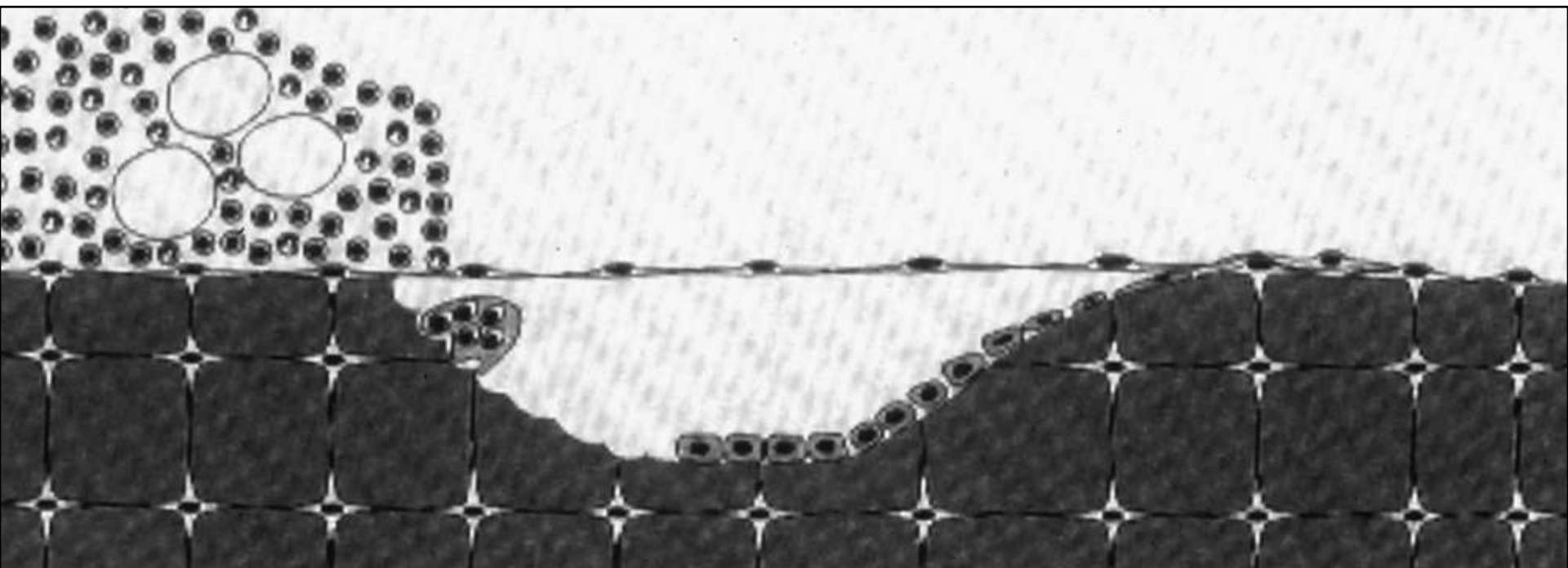








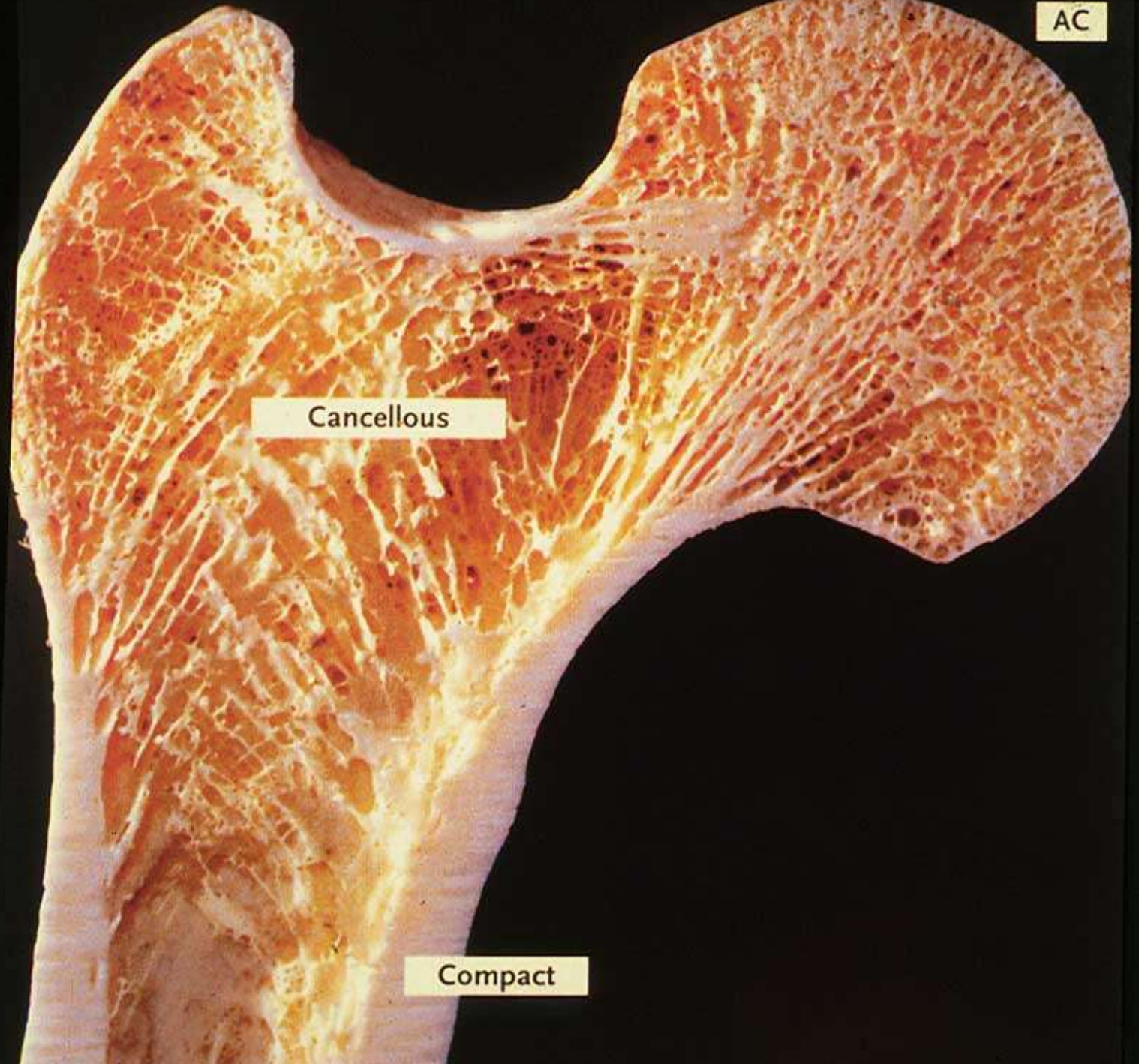




Modeling

Change in shape or size

AC



Cancellous

Compact



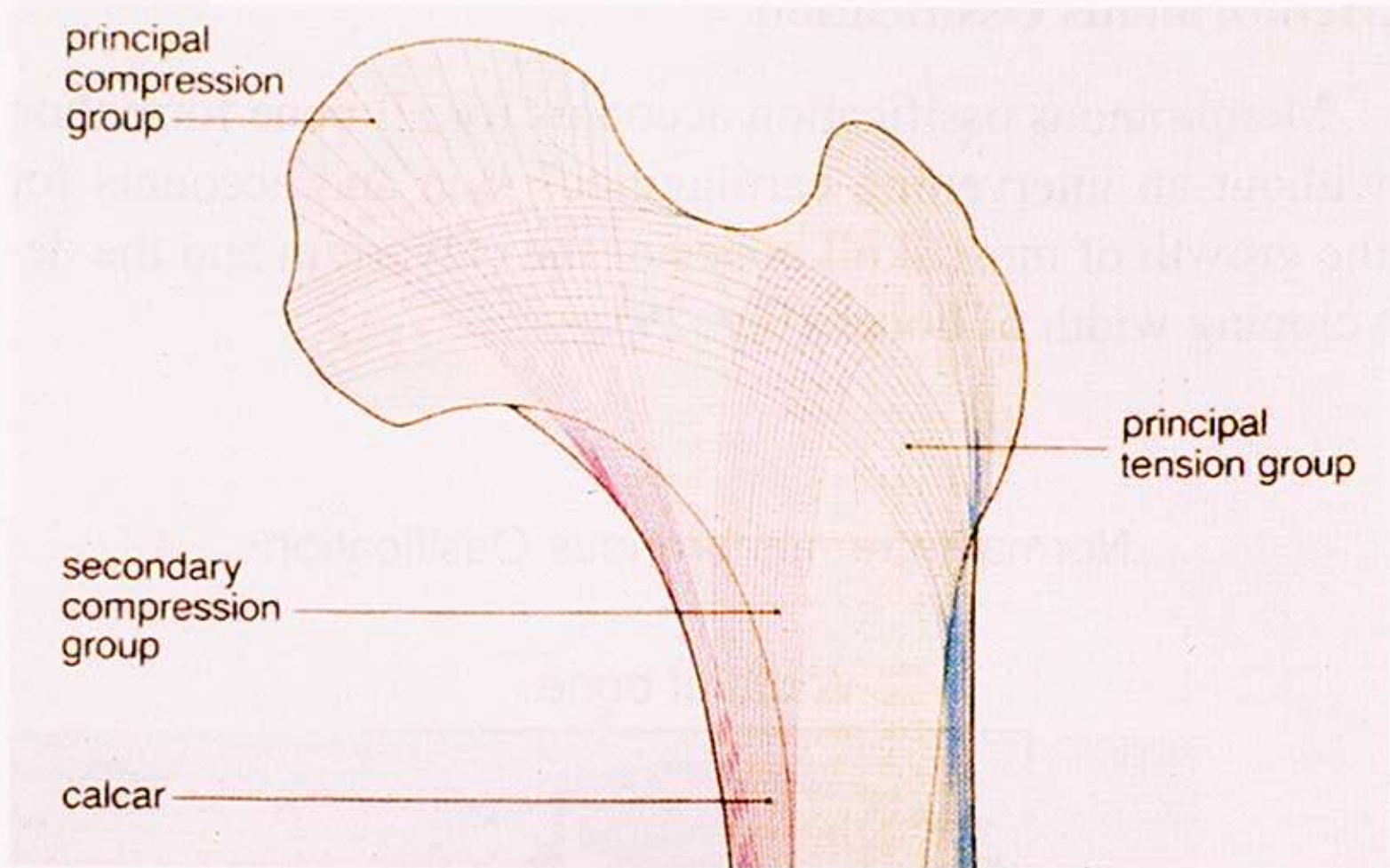
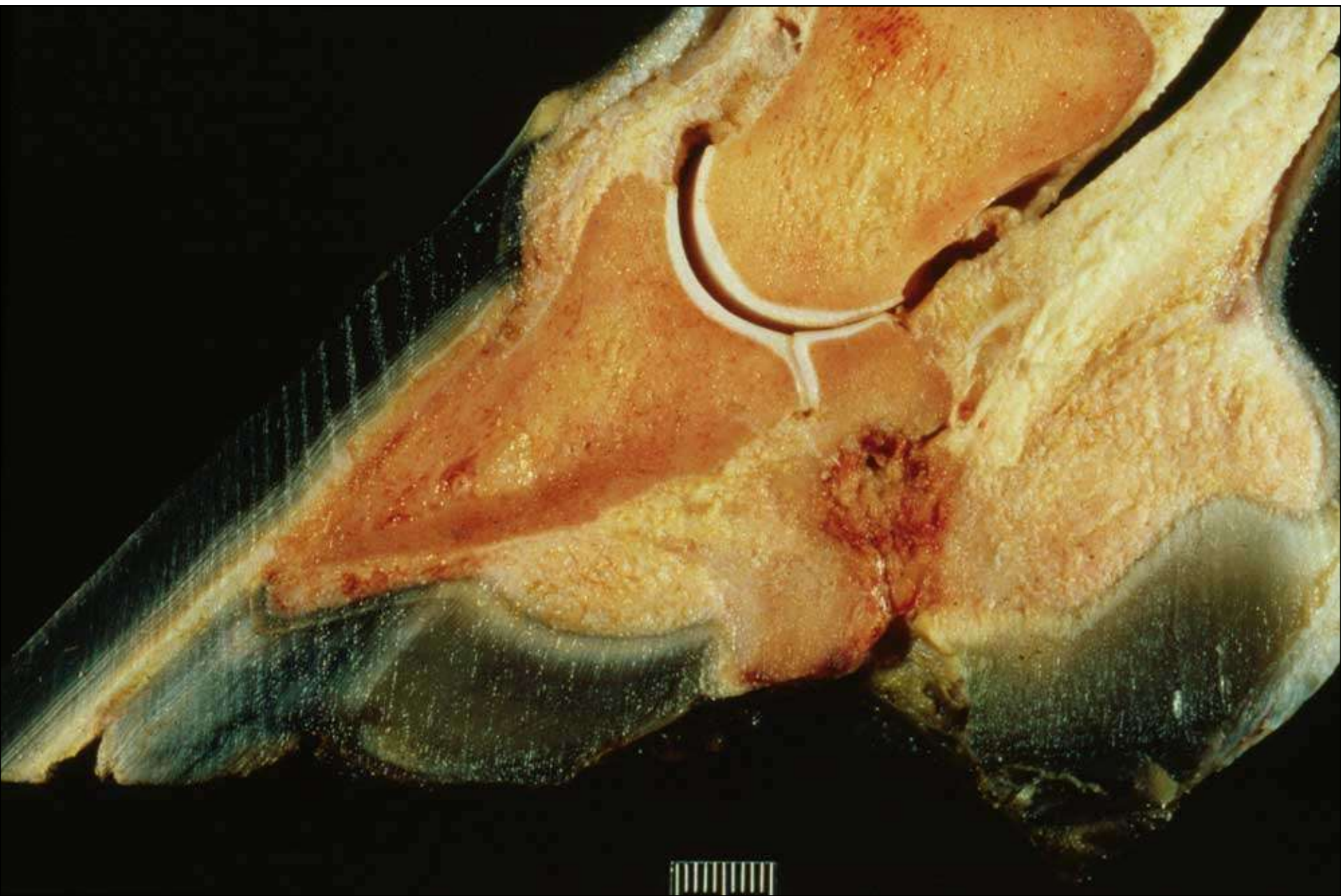
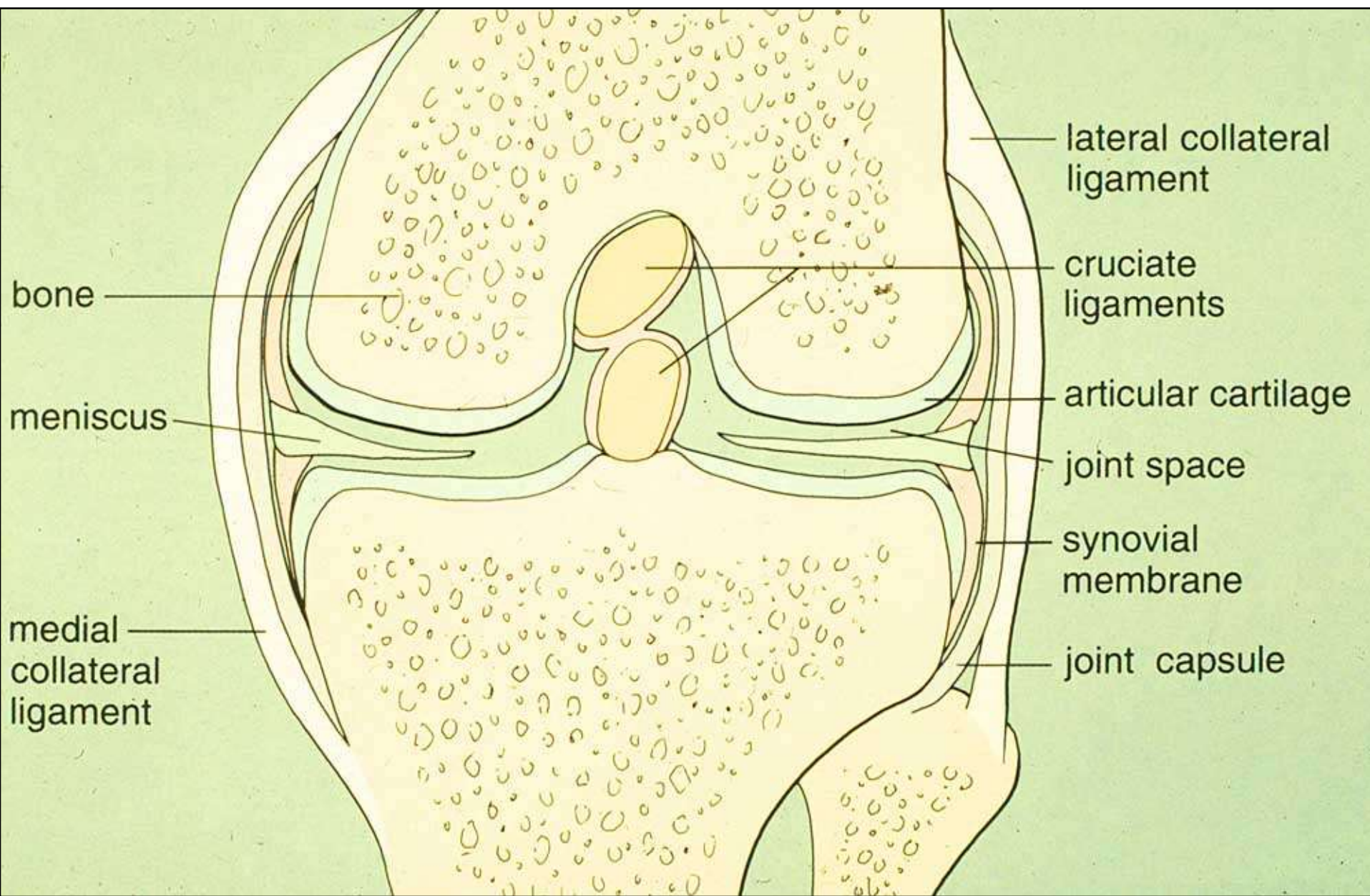


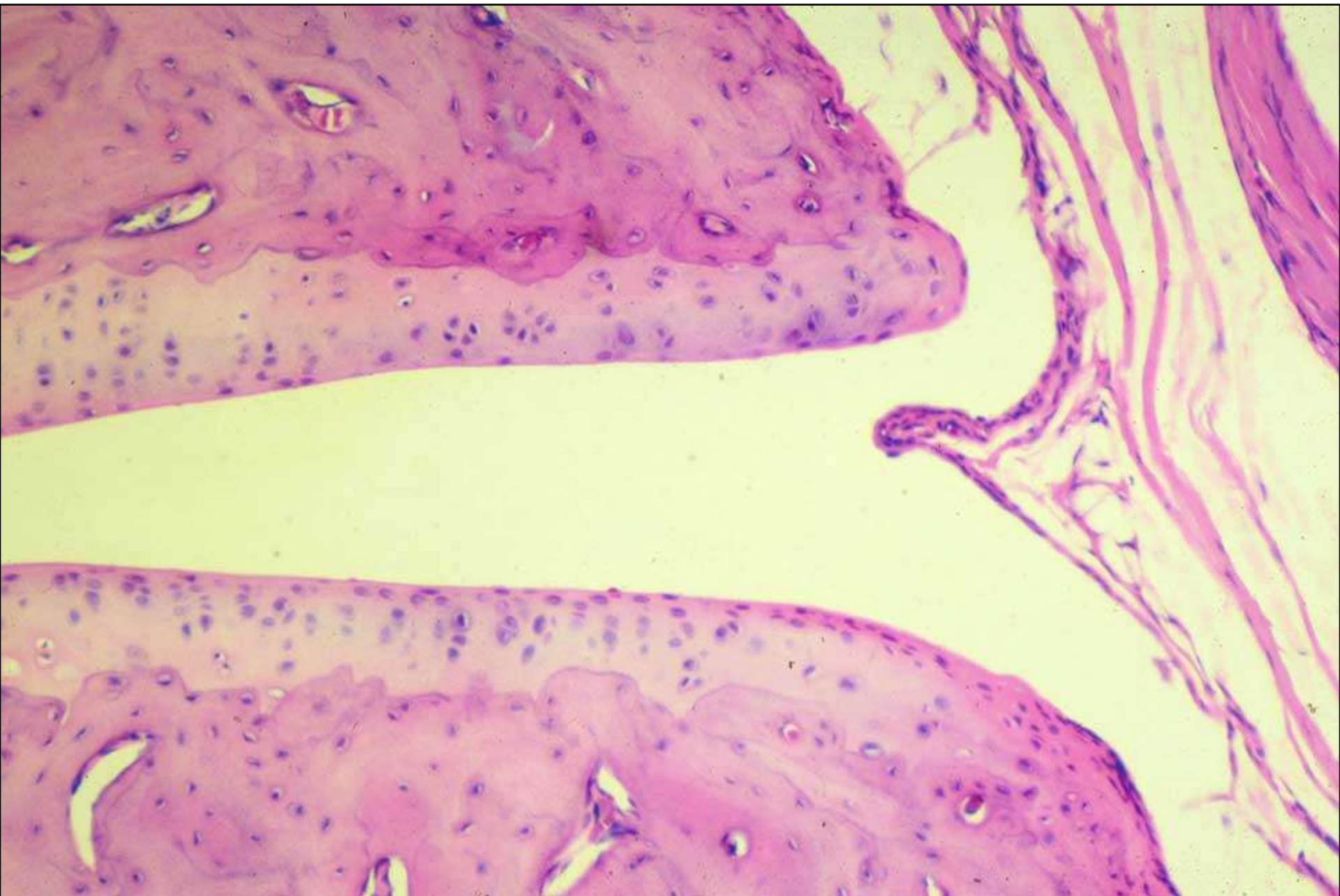
FIG. 31. Bone modeling: the trabecular structure of the proximal femur is a composition of arcades of cancellous bone that “model” or “shape” the internal architecture of bone along compressive and tensile stresses produced during weight bearing.



Articular Cartilage









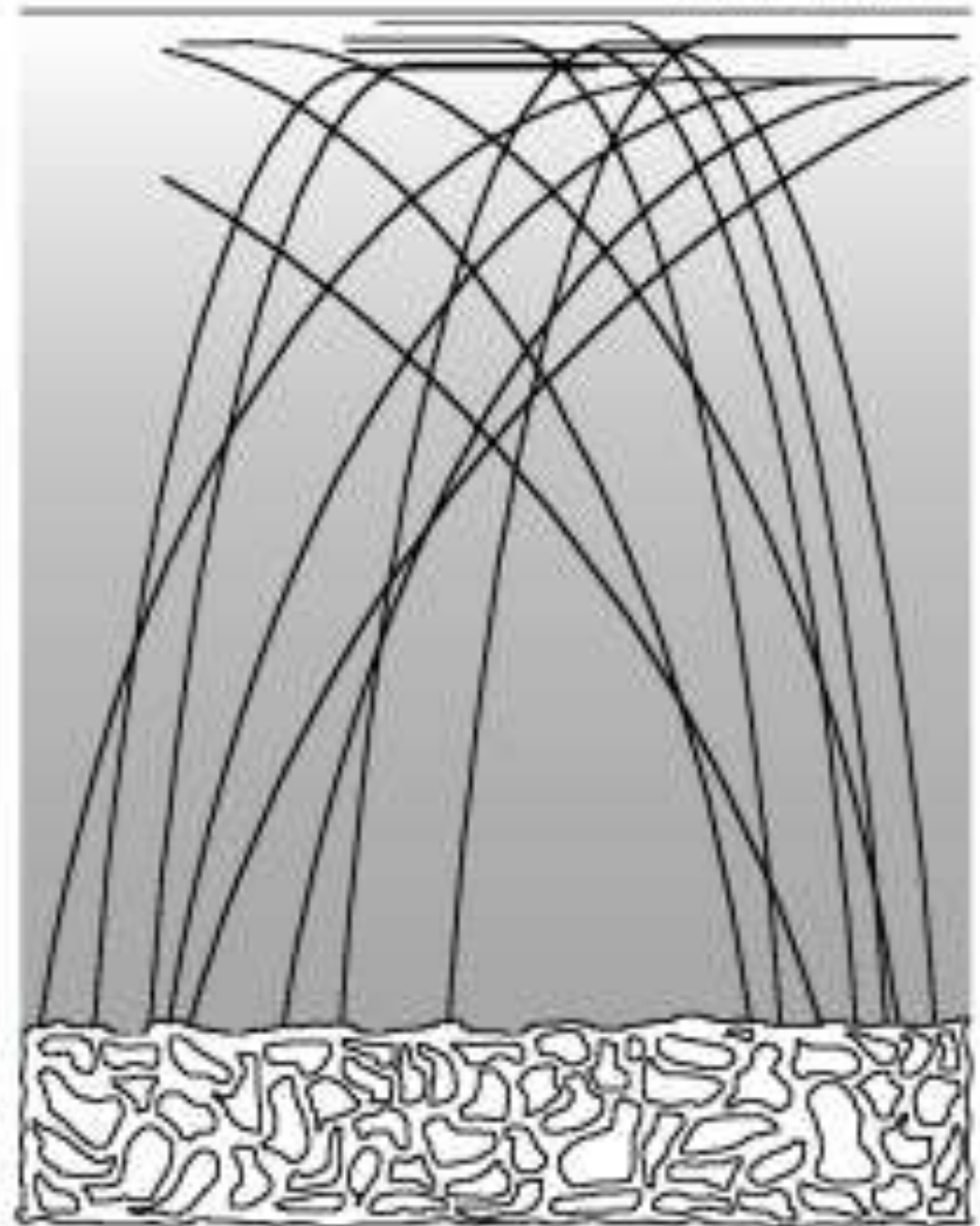
articular surface

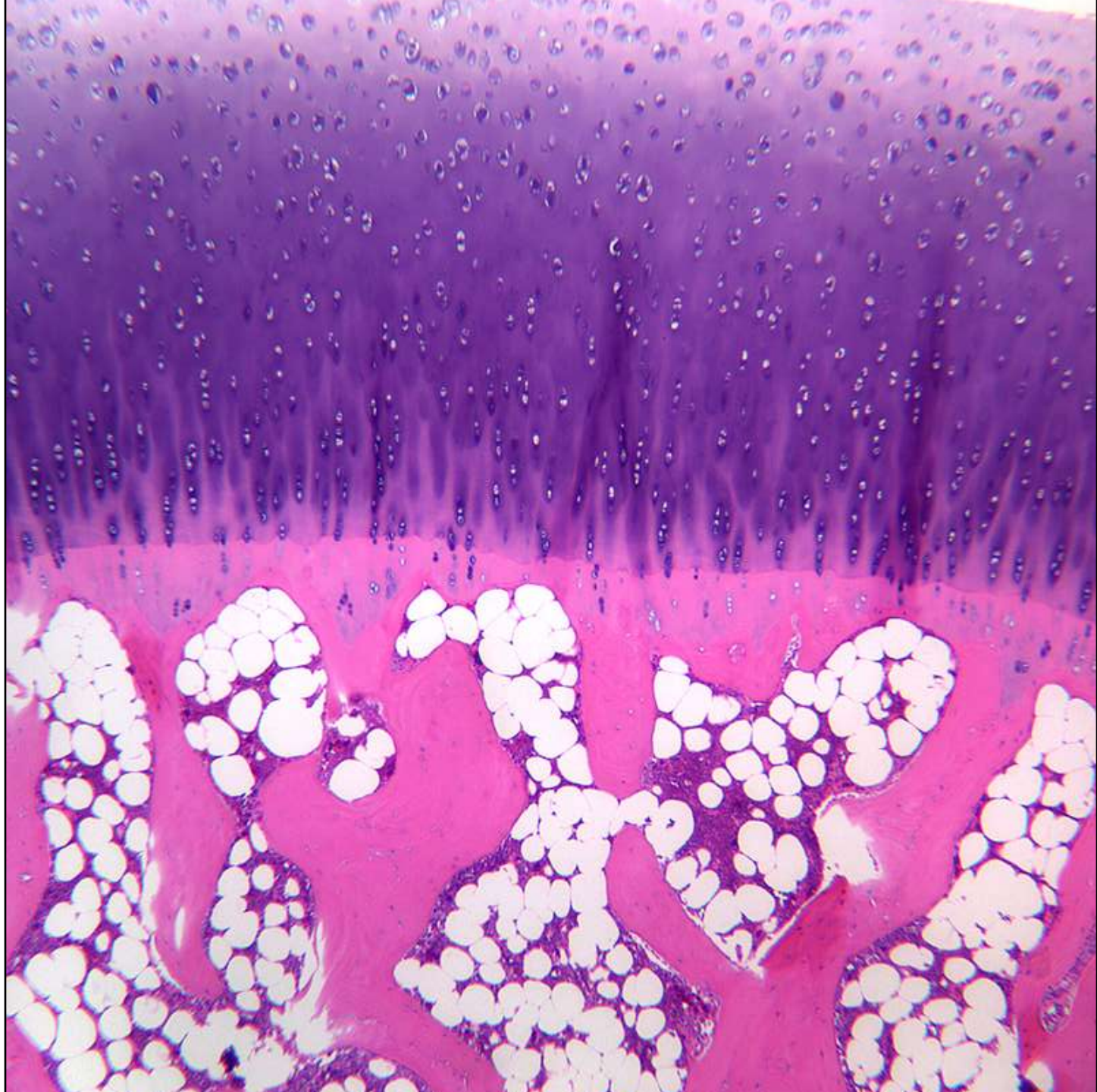
tangential zone

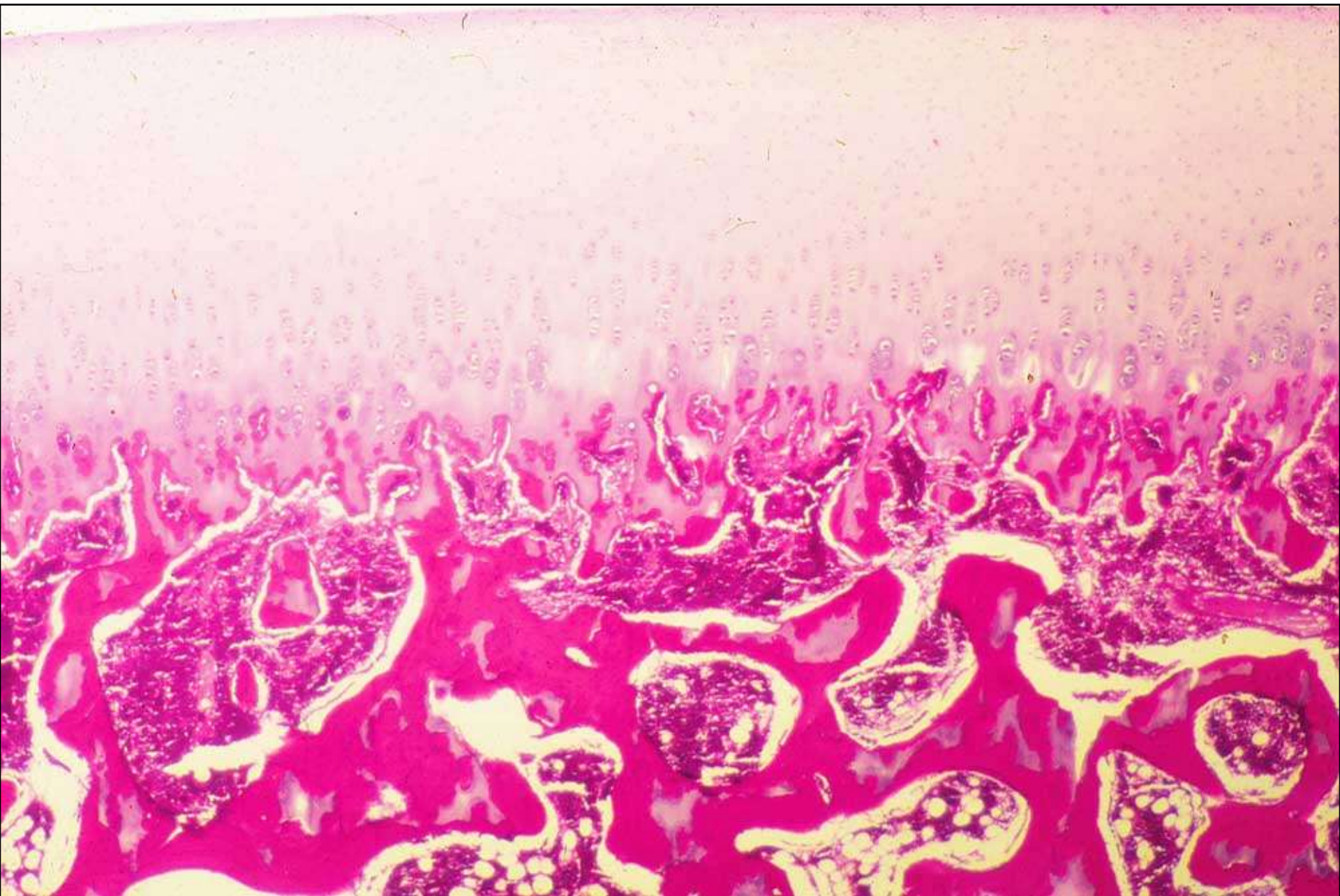
transitional zone

radial zone

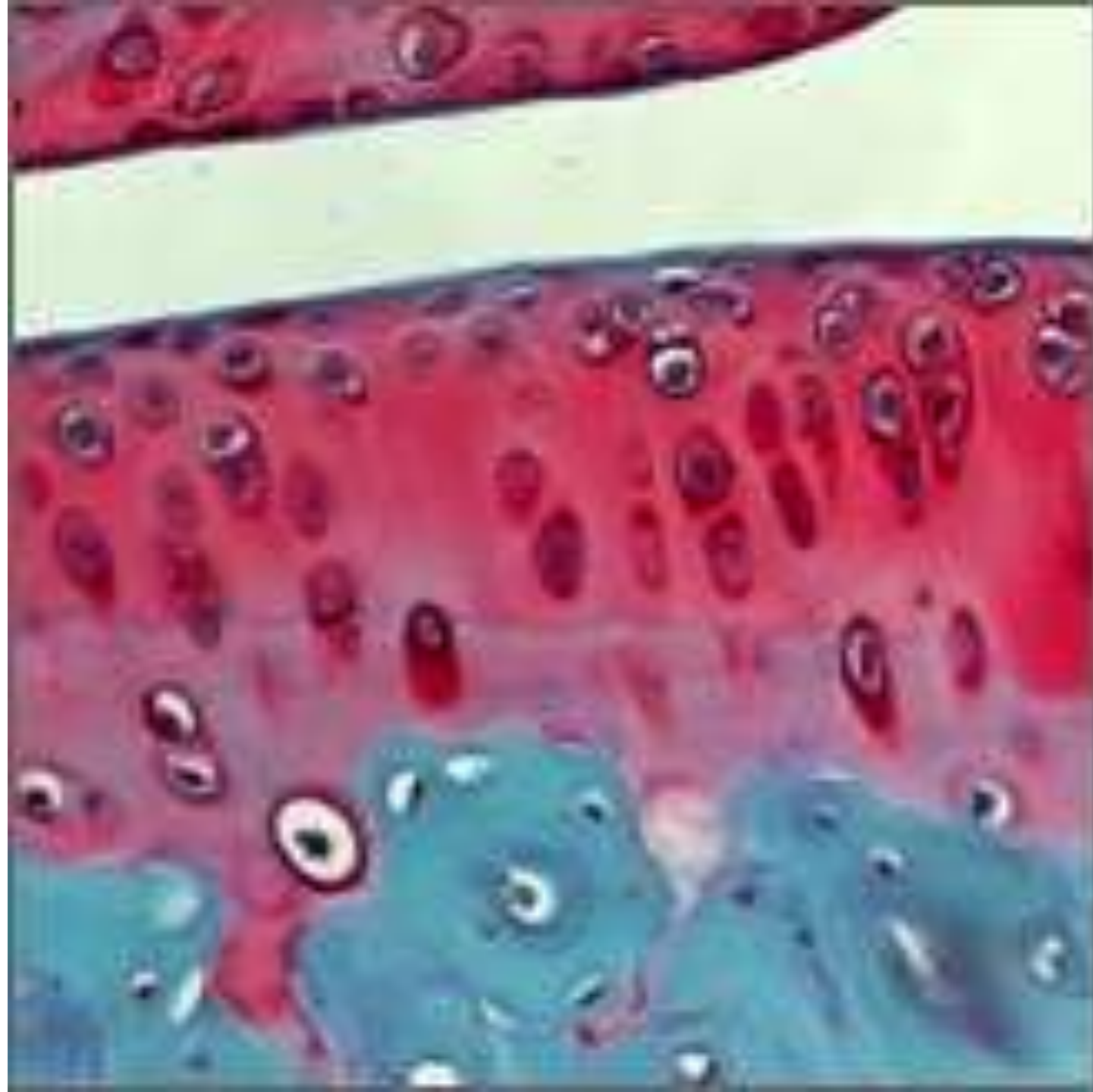
subchondral bone

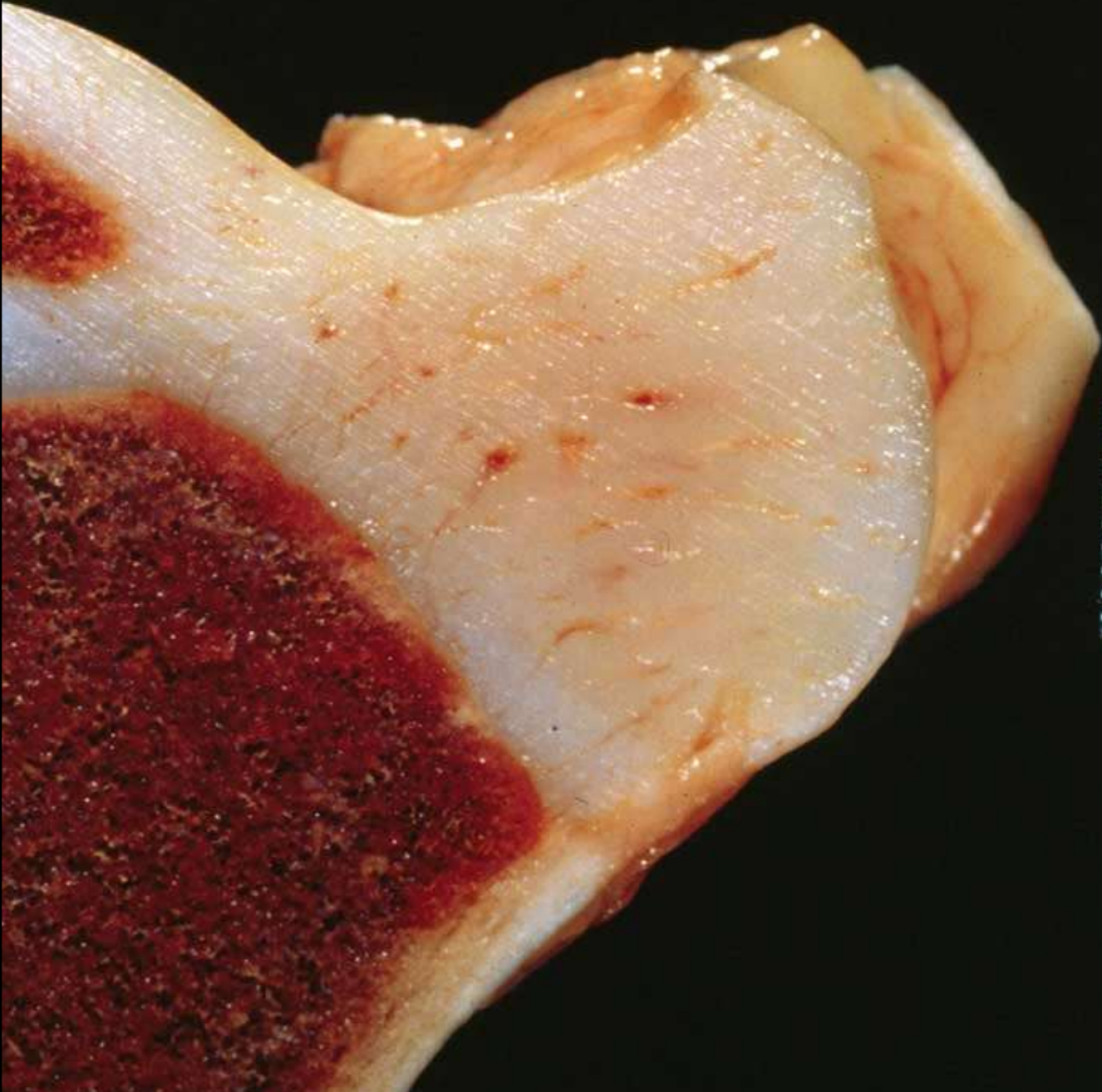




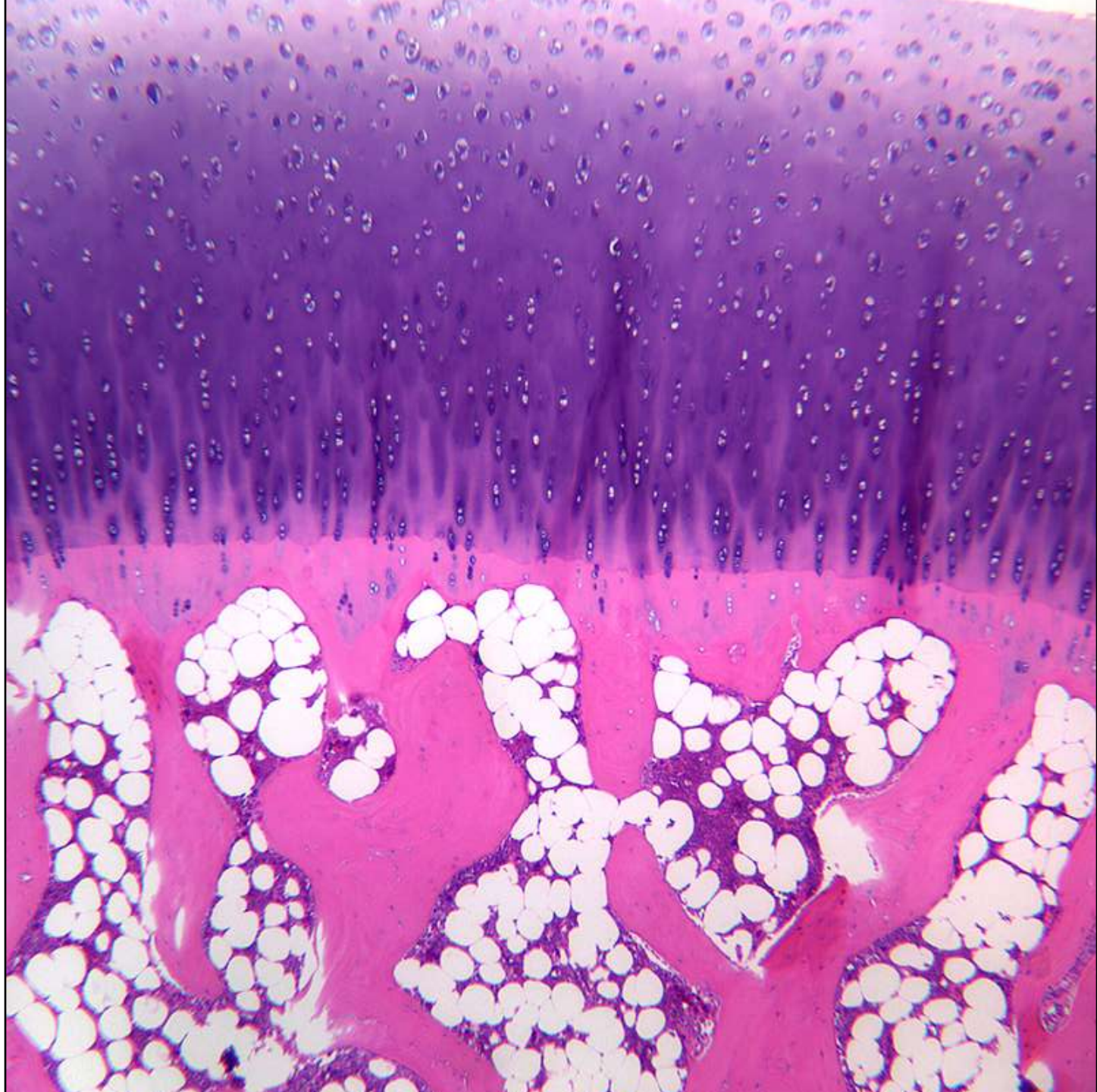


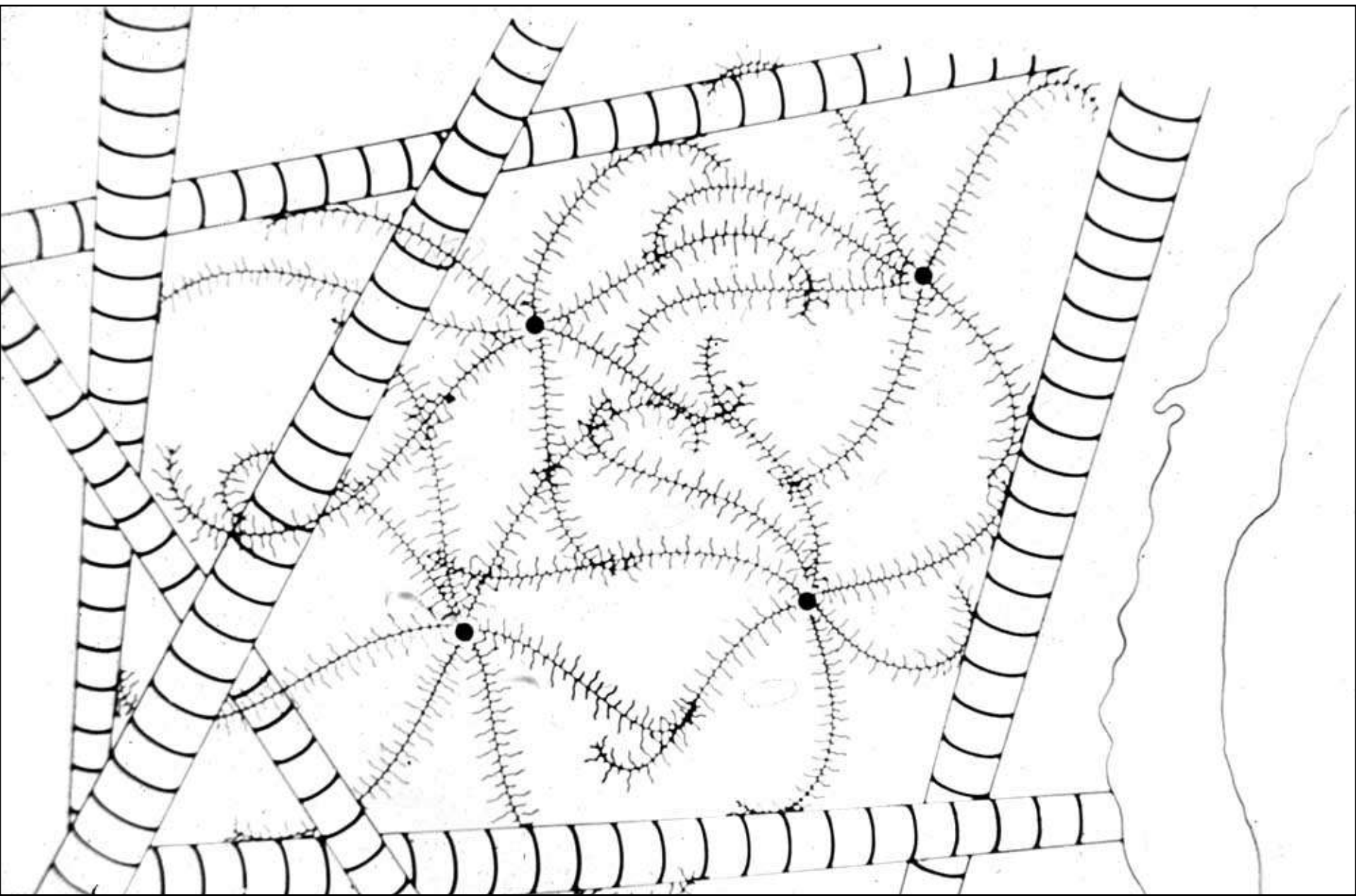


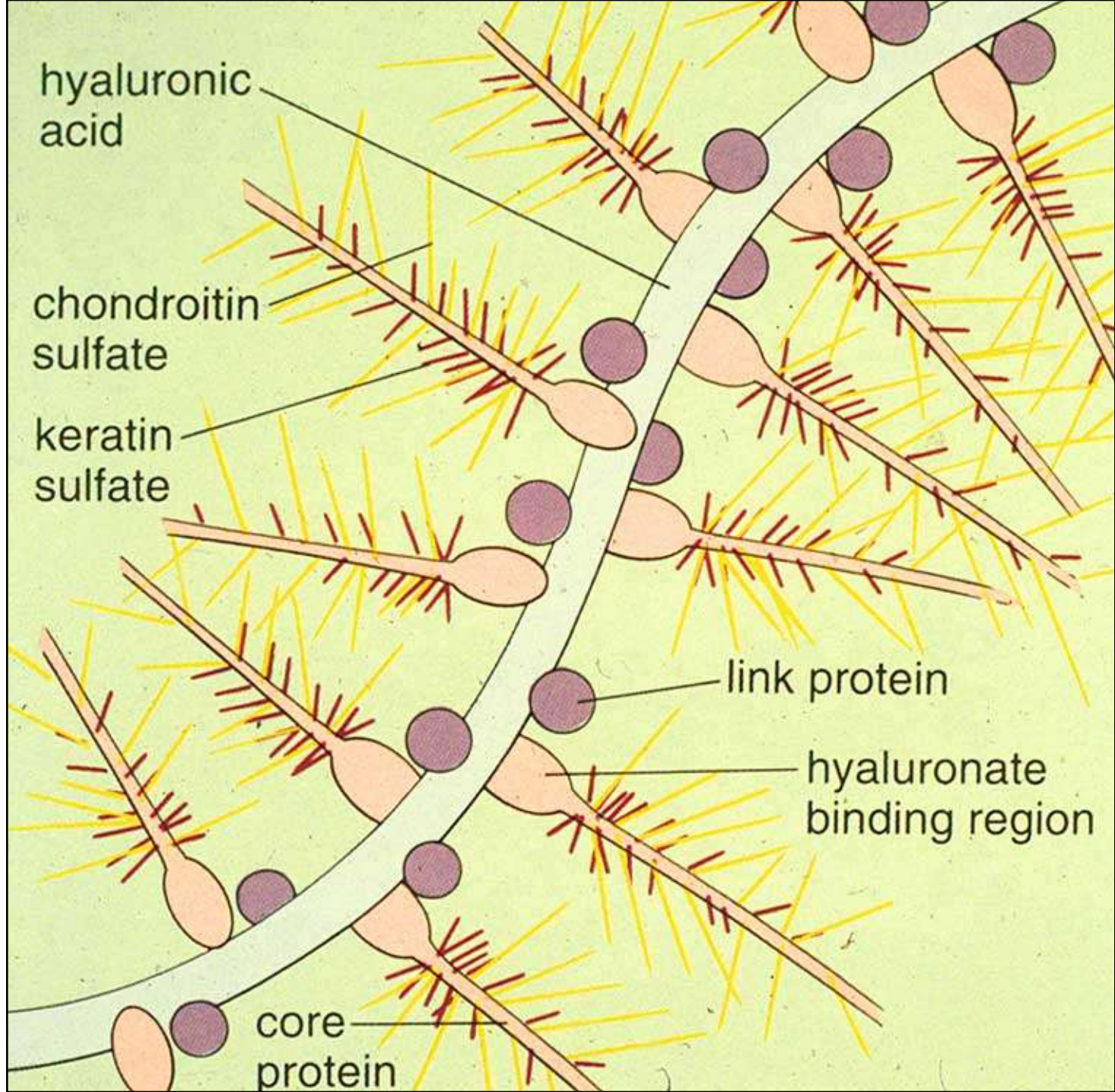


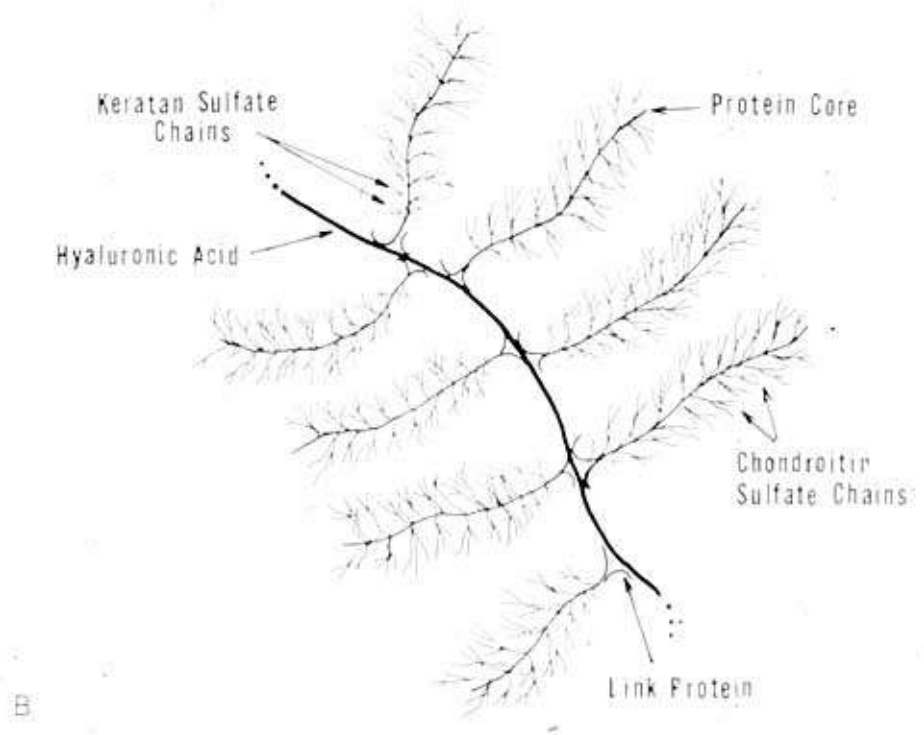
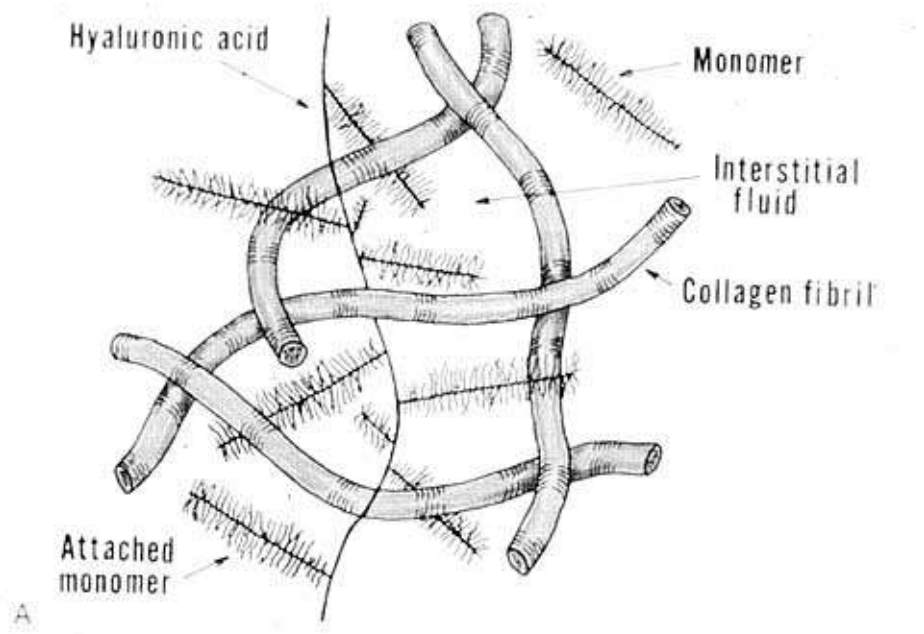


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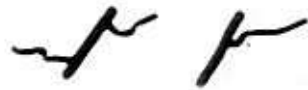




MATRIX

PROTEOGLYCANS

Bone



Cartilage

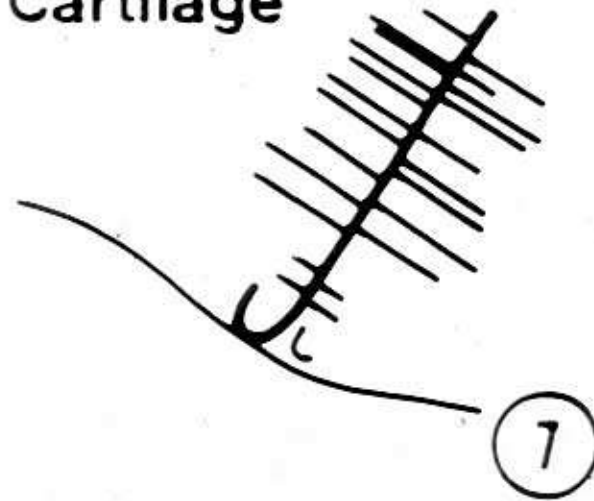
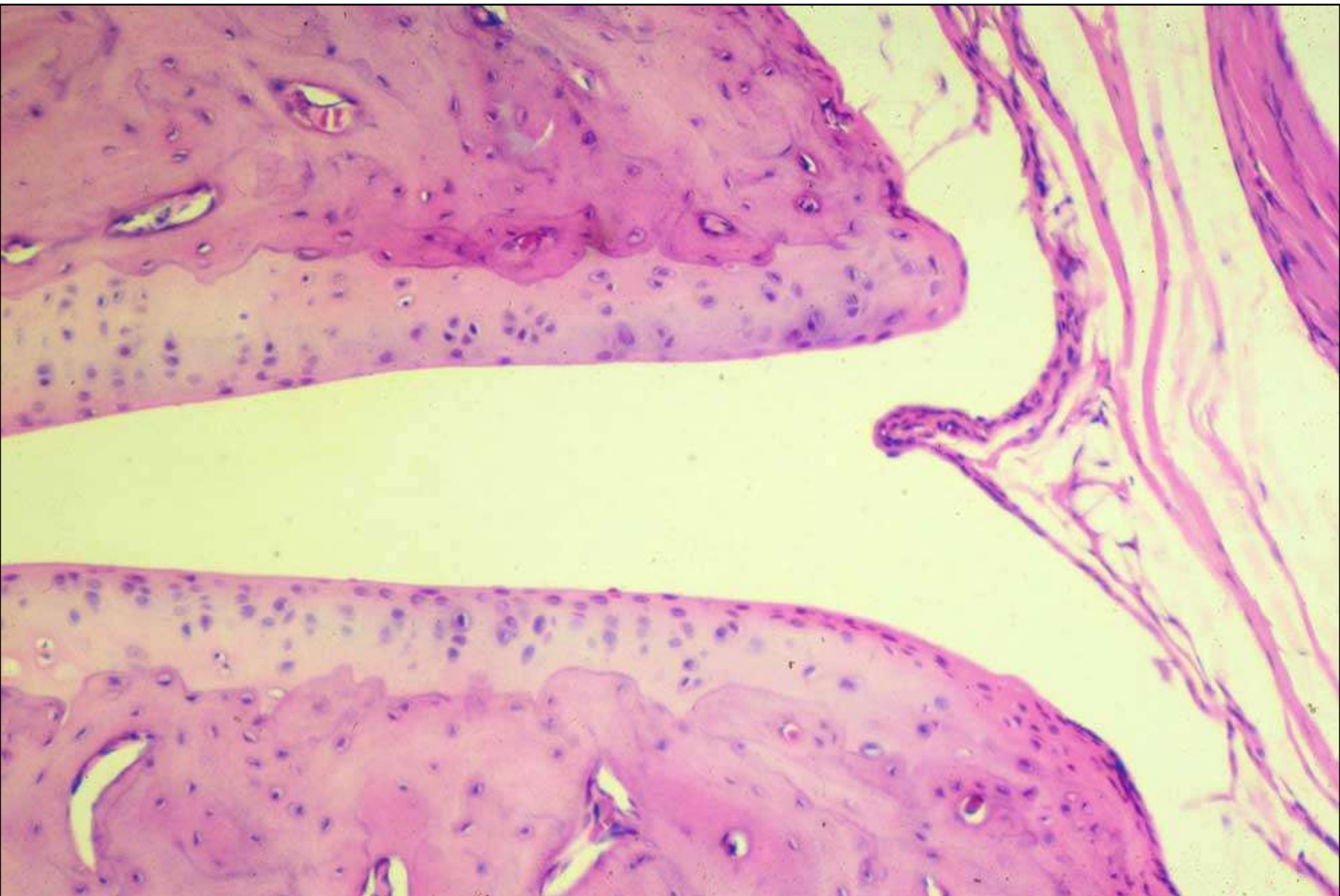
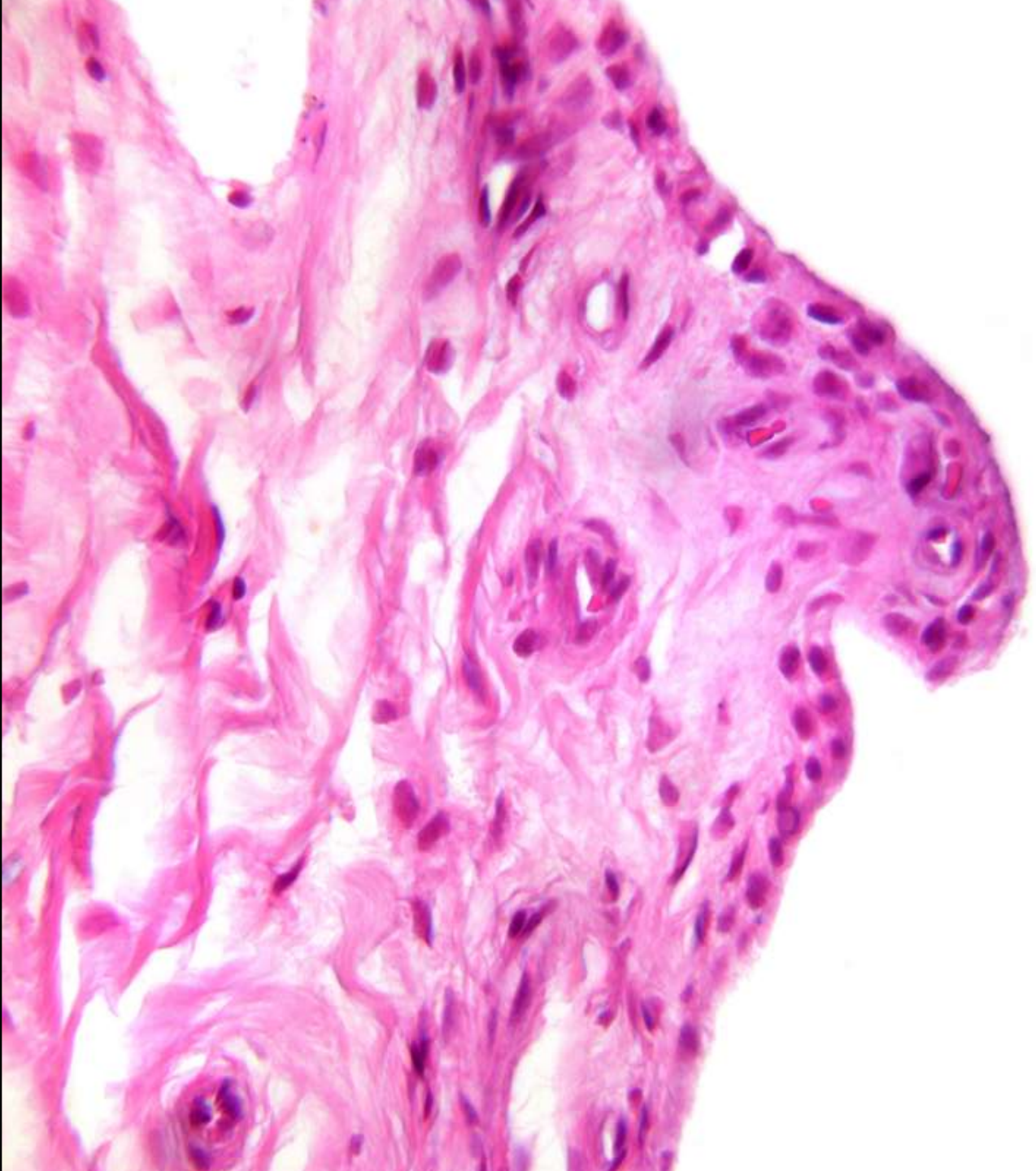
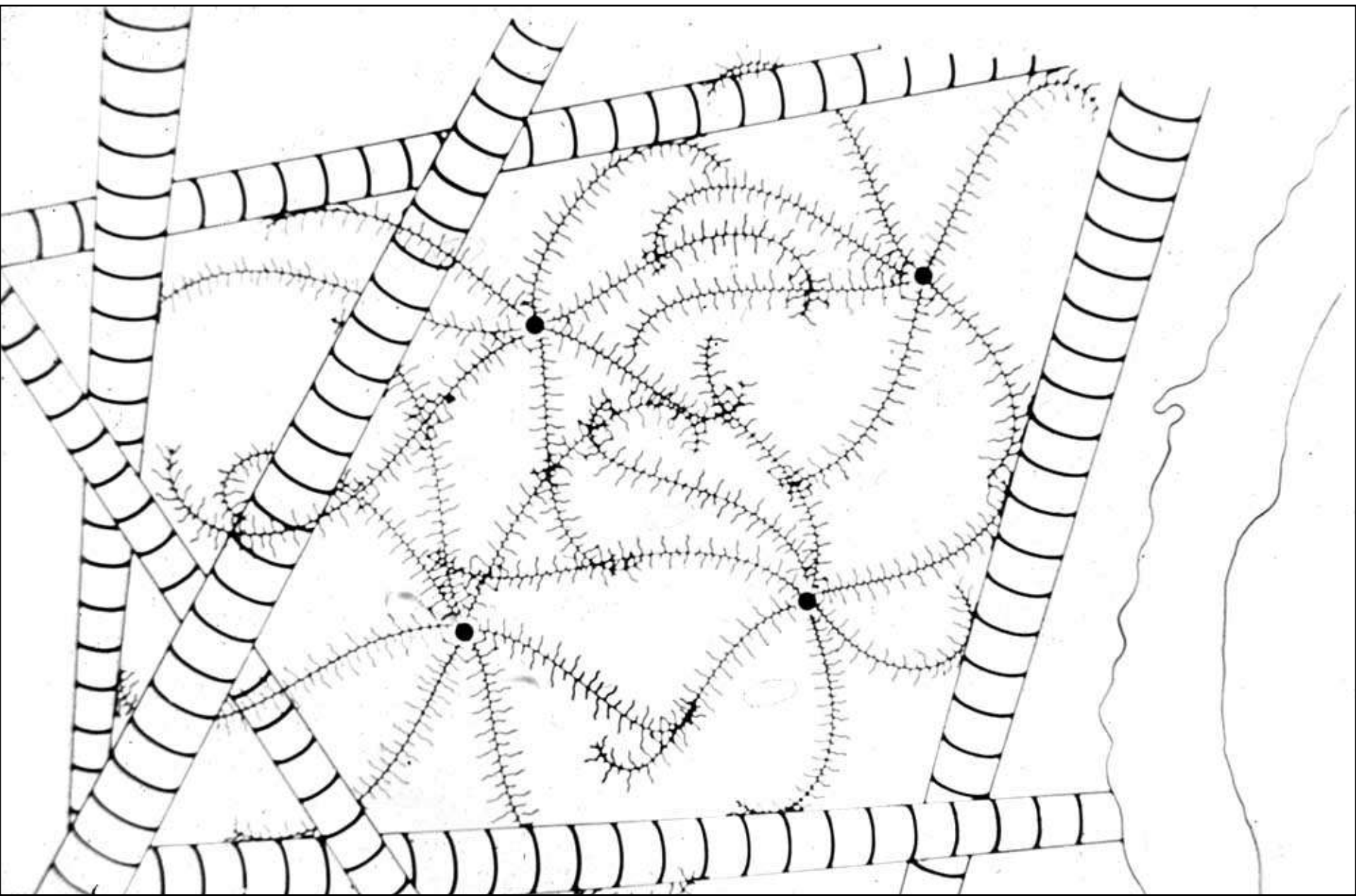
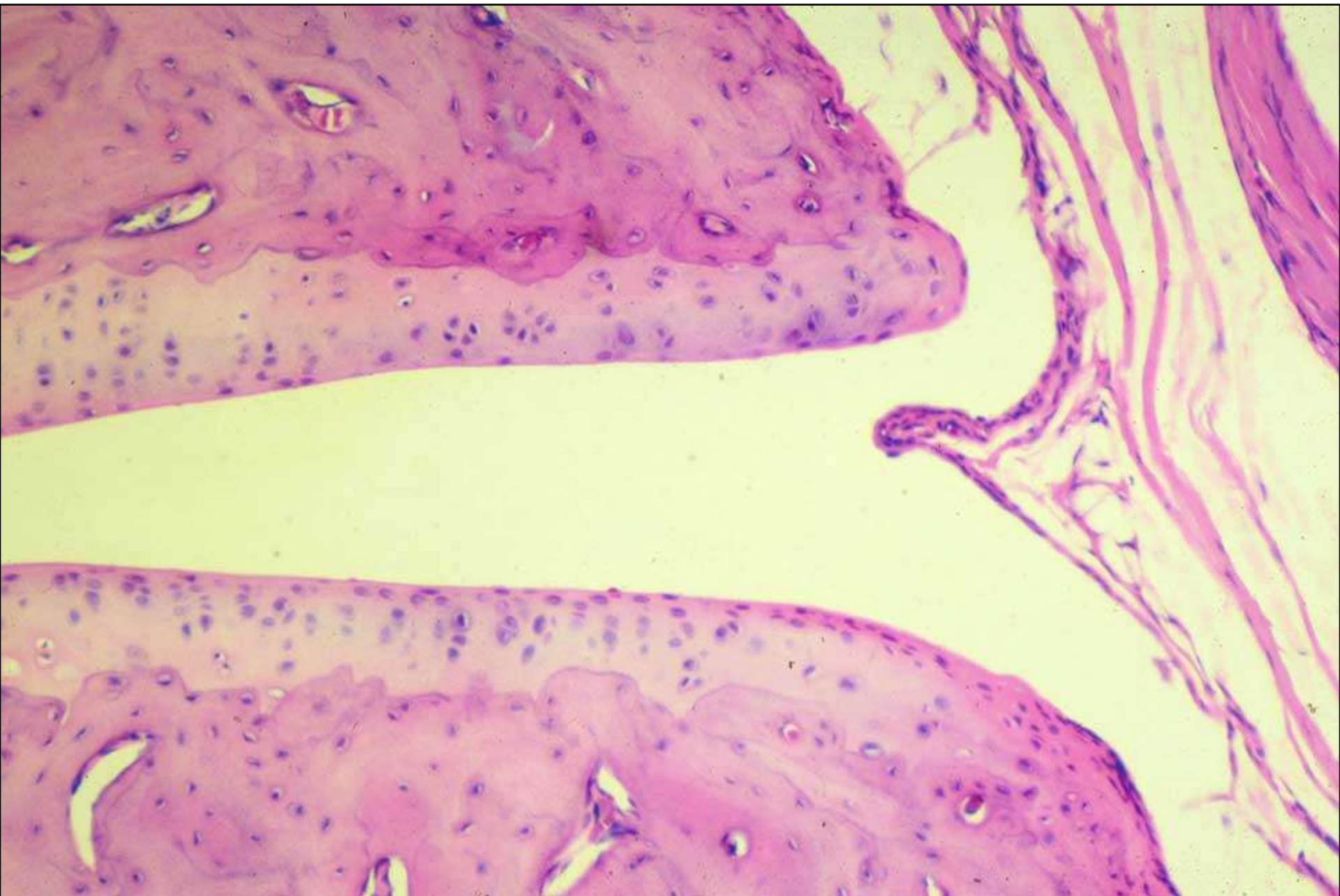


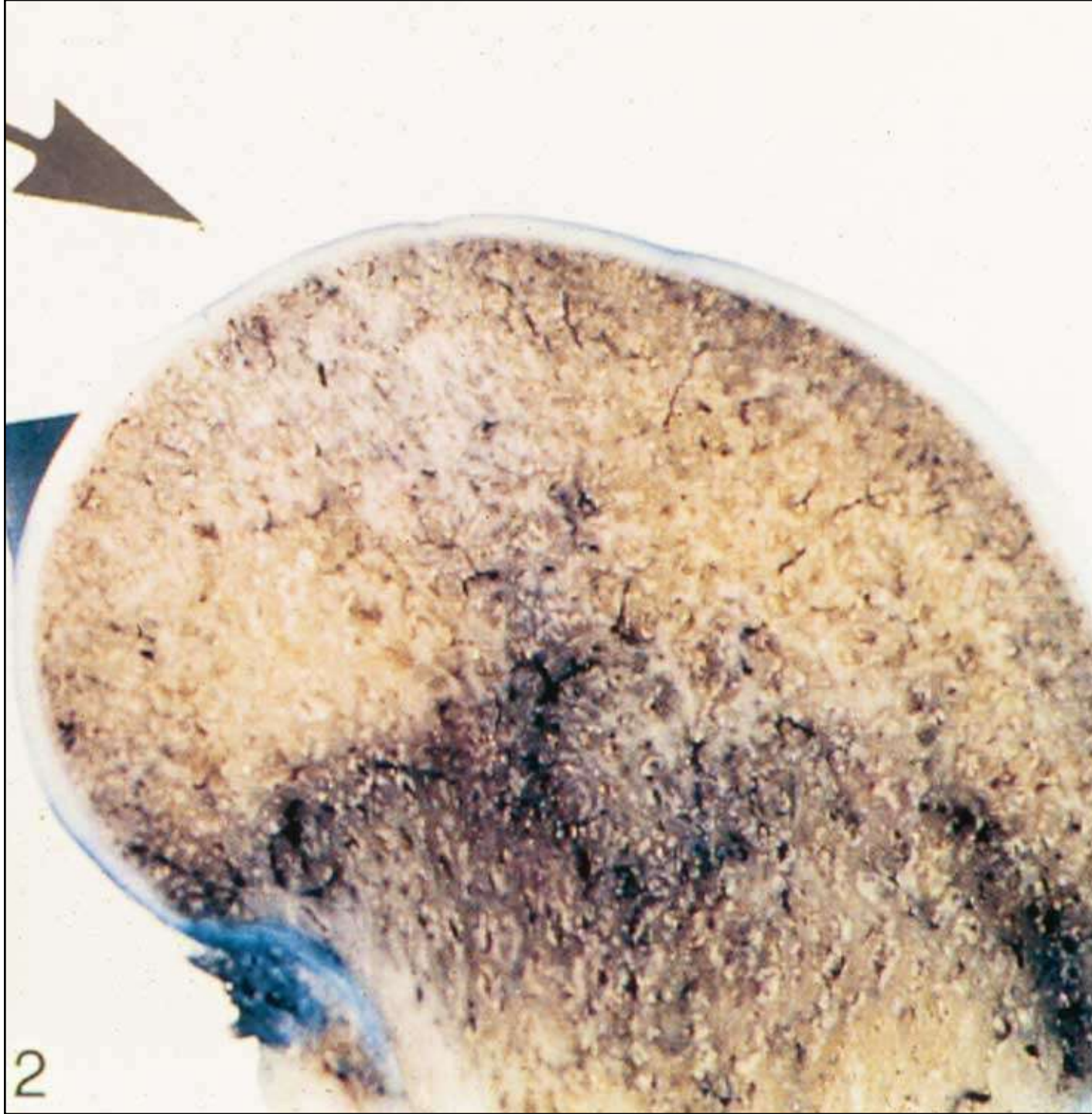
Fig. 7. Proteoglycans in cartilage and bone differ significantly in both size and components. The bone variety has a smaller core protein with one or two glycosaminoglycan (GAG) side chains. The cartilage variety is heterogeneous with respect to both number and length of GAG side chains.



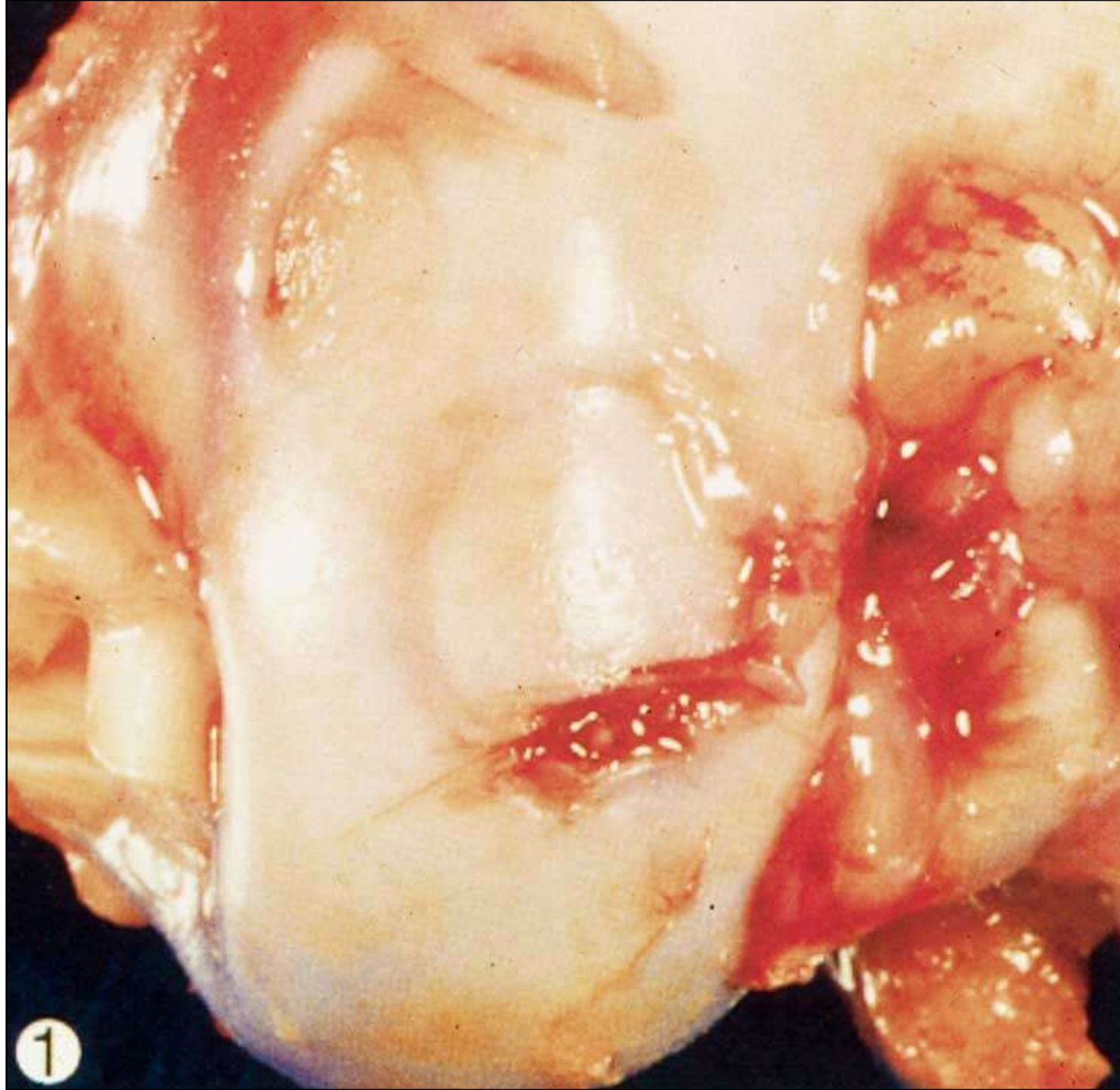




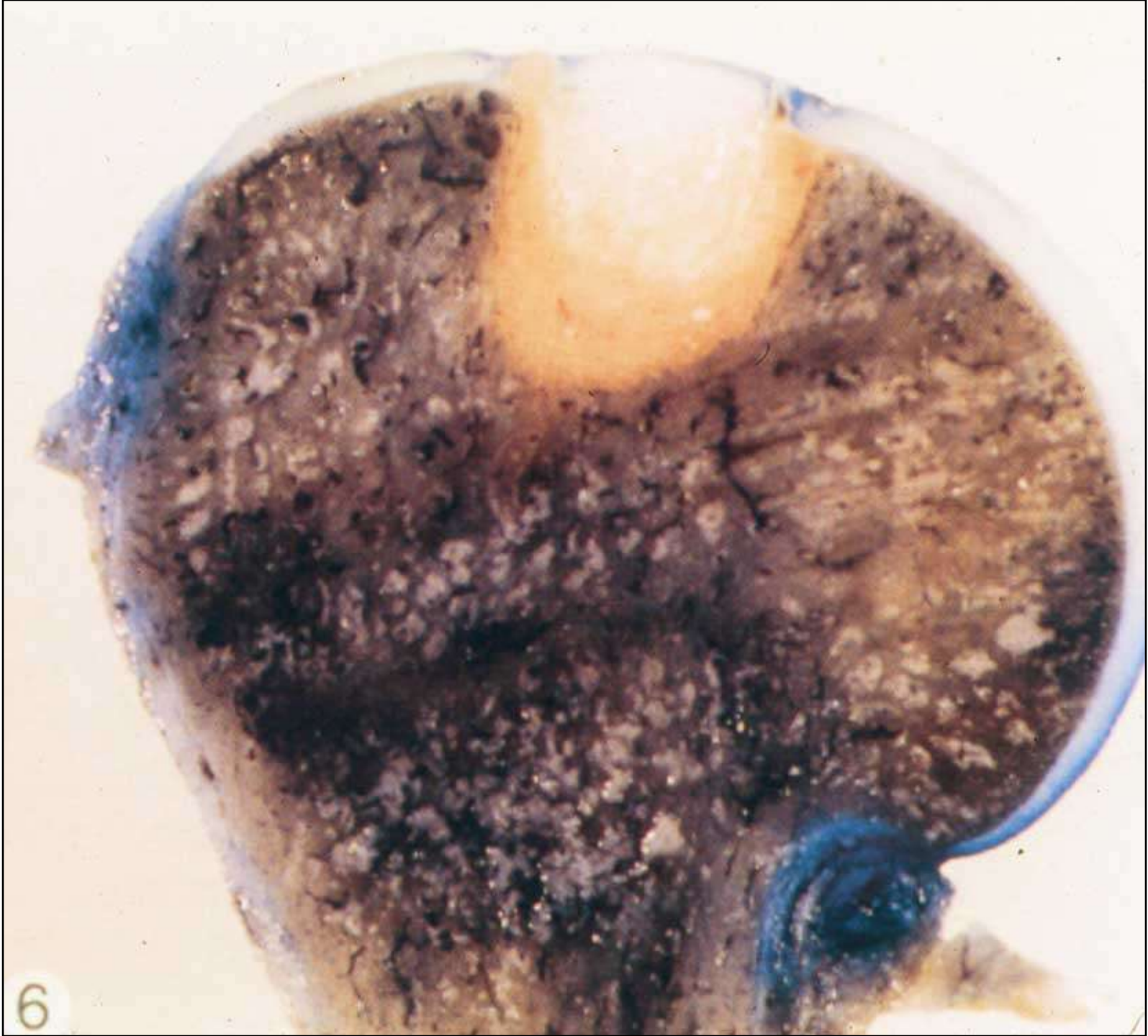




2



1



6



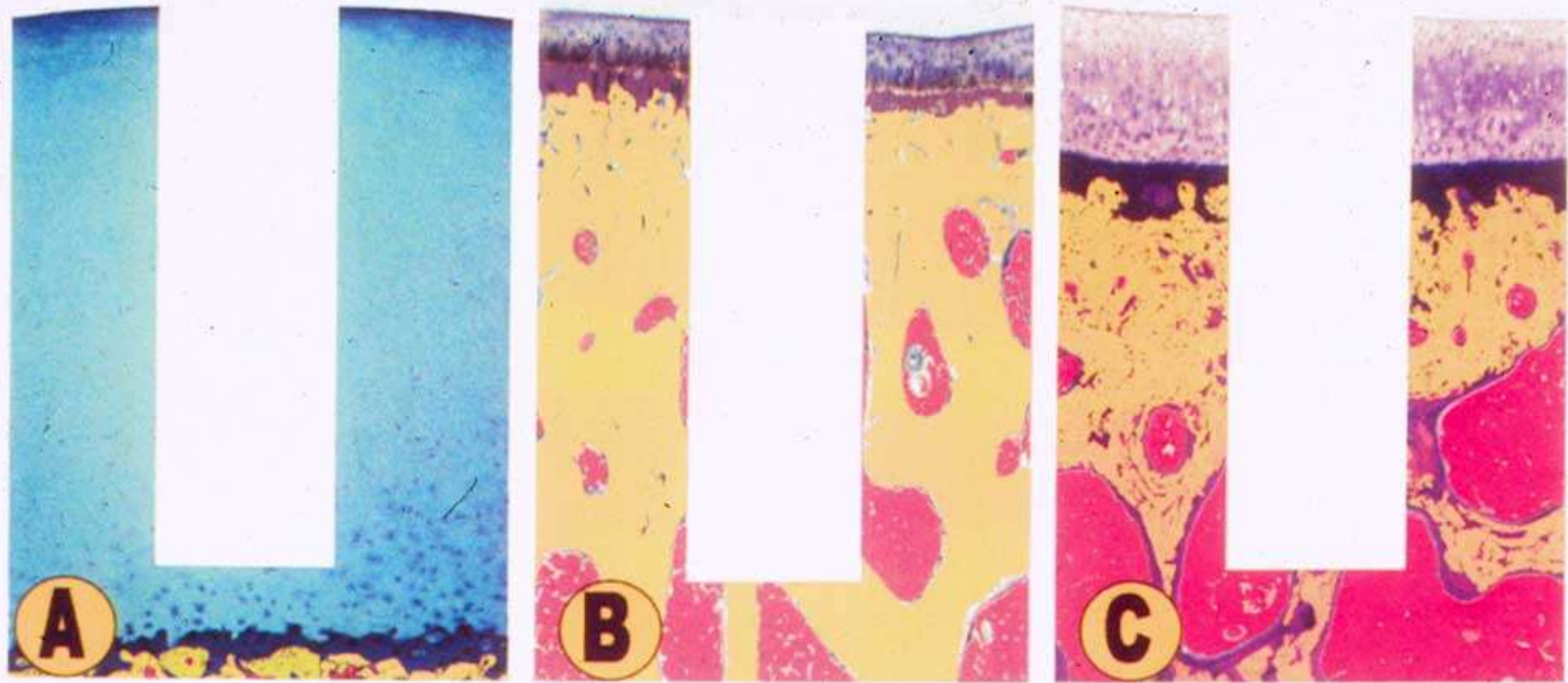
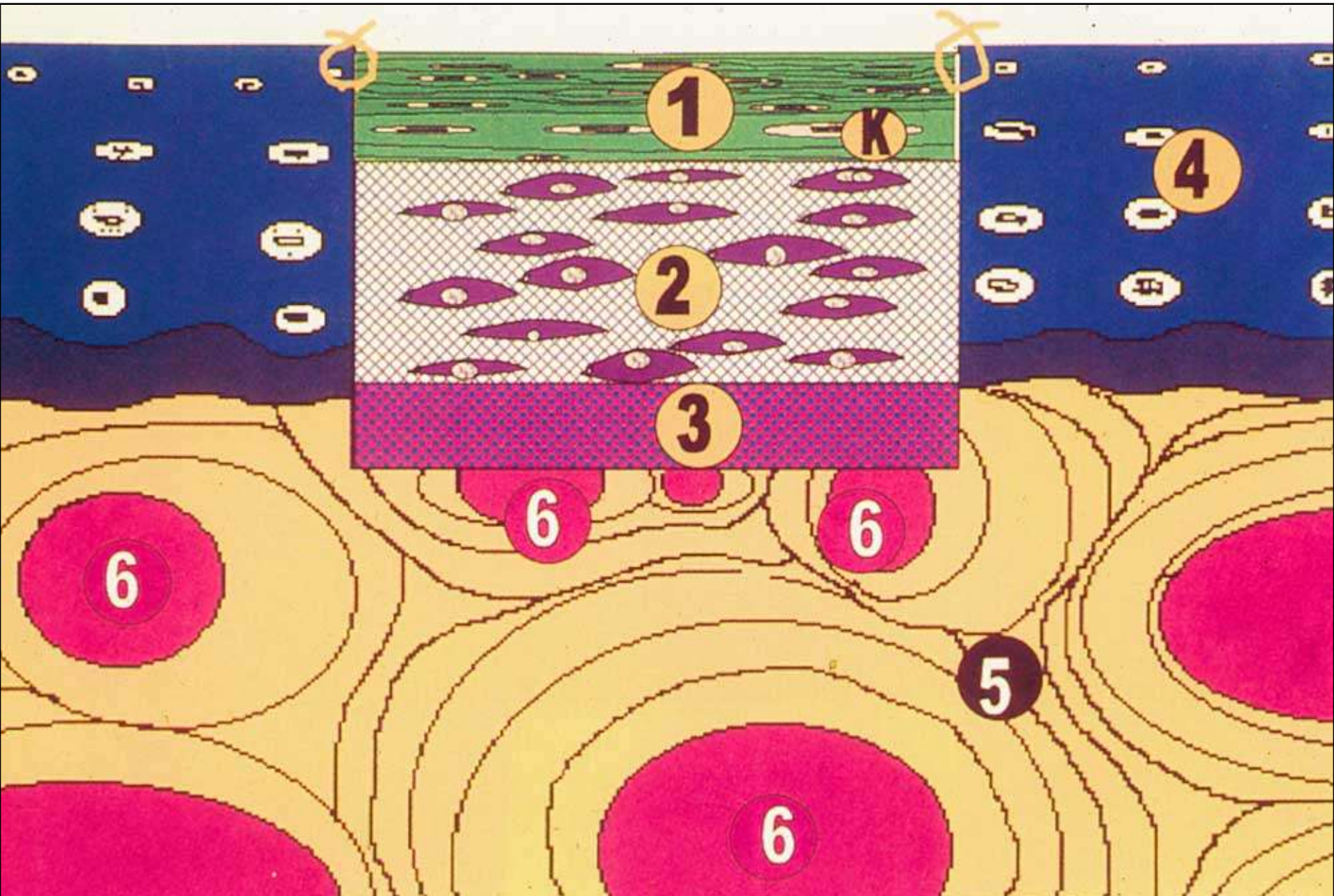


Fig 5A–C. Schematic representations of similar sized defects superimposed on light micrographs of (A) human, (B) rabbit and (C) goat articular cartilage (with varying portions of the underlying subchondral bone), represented at the same magnification. Because the thickness of the articular cartilage layer in humans is several times greater than that in goats, and many times greater than that in rabbits, the biologic environment surrounding each defect differs. In the human, the defect is a partial thickness defect, and, as such, it is surrounded exclusively by cartilage tissue. In the rabbit and goat, lesions of the same dimensions are full thickness defects; approximately 95% and 85% of their volumes, respectively, are surrounded by bone and bone marrow tissue. In these two latter cases, bleeding from the bone marrow vascular spaces will furnish the defects with an abundant supply of signaling substances and cells which the partial thickness human defect will not be accessible to.

Cartilage and Subchondral Bone Thickness (mm)

McIlwraith VCOT 19:142, 2006

| • Species | AC | TMAC | SB |
|-----------|-----|------|-----|
| • Human | 2.5 | 0.2 | 0.4 |
| • Equine | 2.0 | 0.2 | 0.5 |
| • Dog | 0.6 | 0.1 | 0.3 |
| • Rabbit | 0.3 | 0.1 | 0.3 |
| • Sheep | 0.5 | 0.2 | 0.3 |



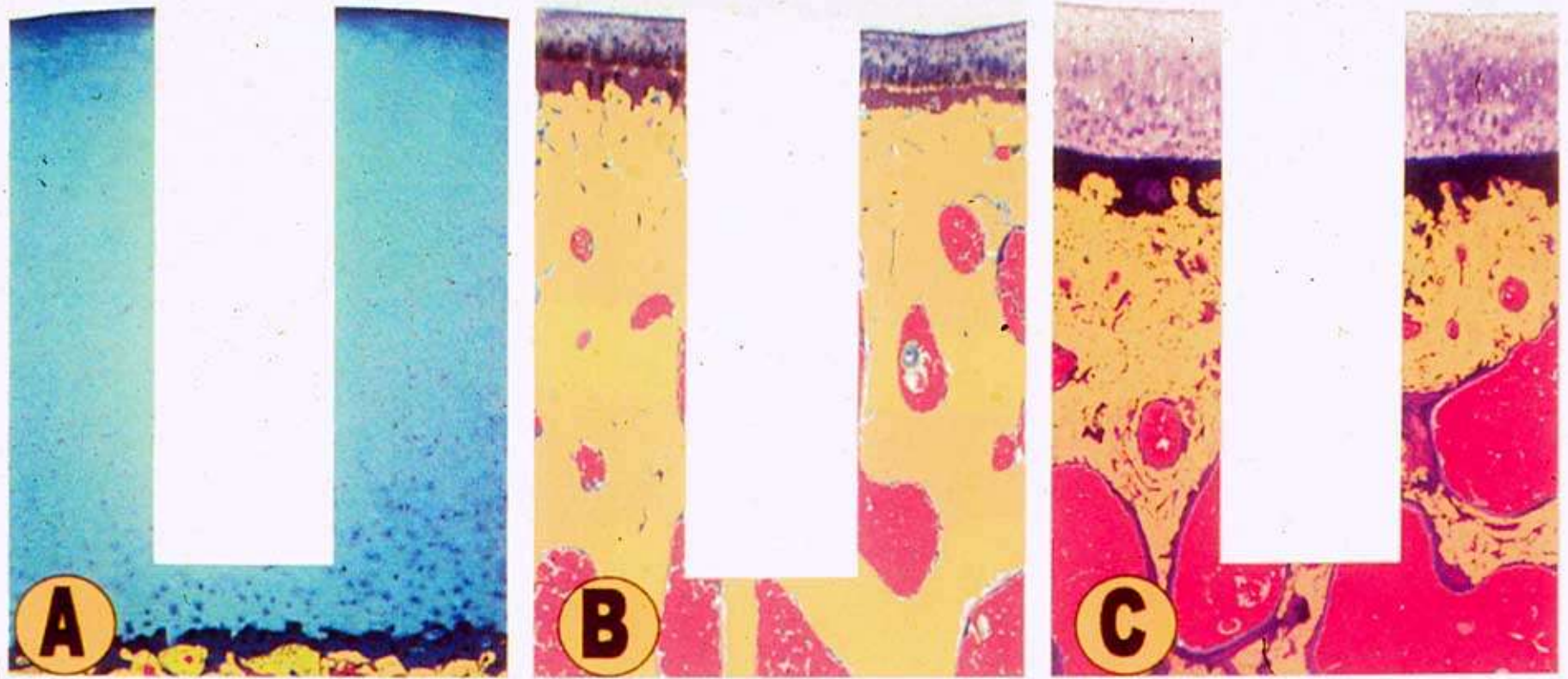


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Synovial Fossa

