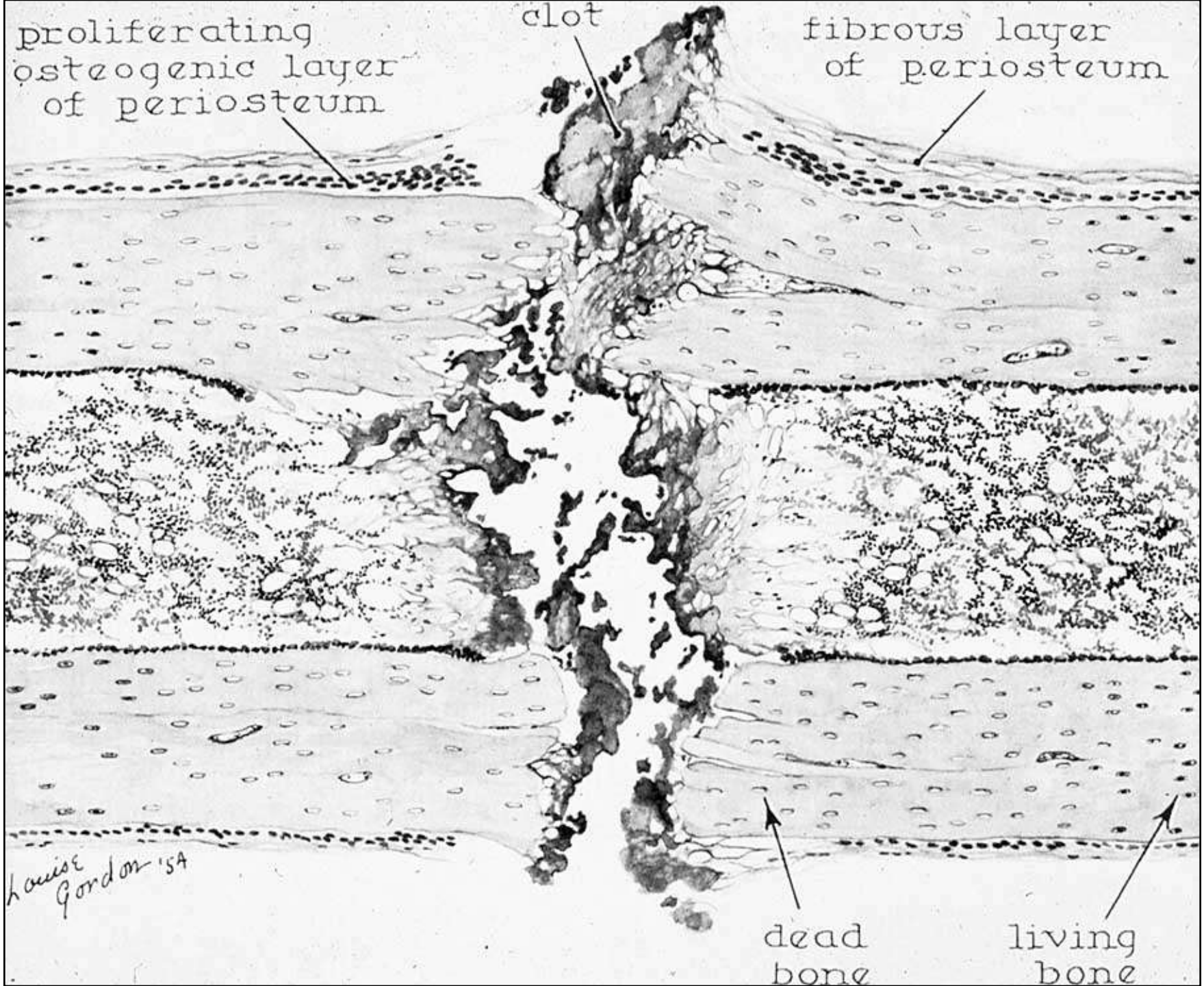
Stable Fracture Healing


proliferating
osteogenic layer
of periosteum
clot
fibrous layer
of periosteum
Louise Gordon '54
dead
bone
living
bone

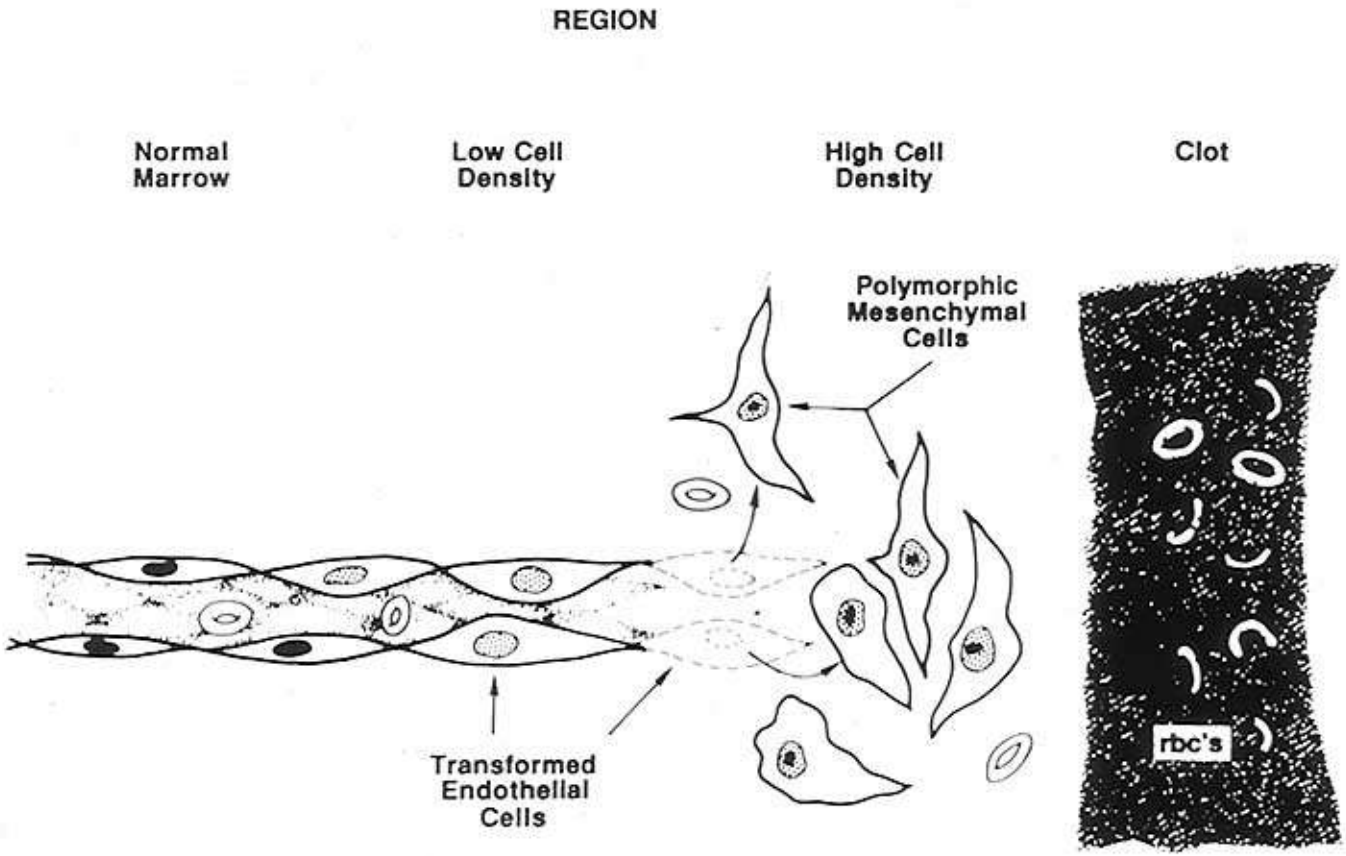


FIG. 20

Drawing summarizing the major findings of this study. In the region of low cellular density in the medullary callus, the endothelial cells of the capillary or small venous vessels undergo enlargement and transformation. In the region of high cellular density adjacent to the fibrin clot, no vessels are seen. Polymorphic mesenchymal cells appear in the regions of both high and low cellular density. In many places, the transformed endothelial cells appear to extravasate from the vessel; these cells could possibly appear as polymorphic mesenchymal cells. Rbc = red blood cell.

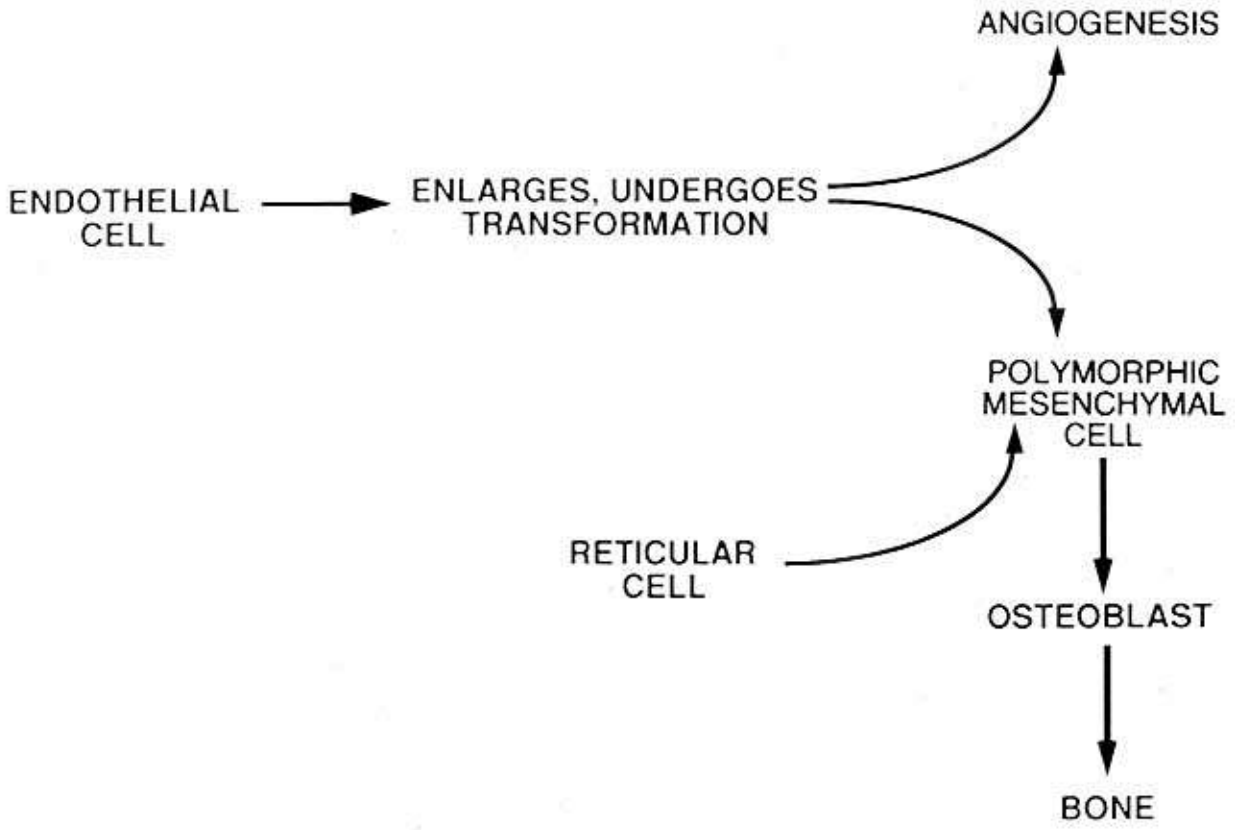
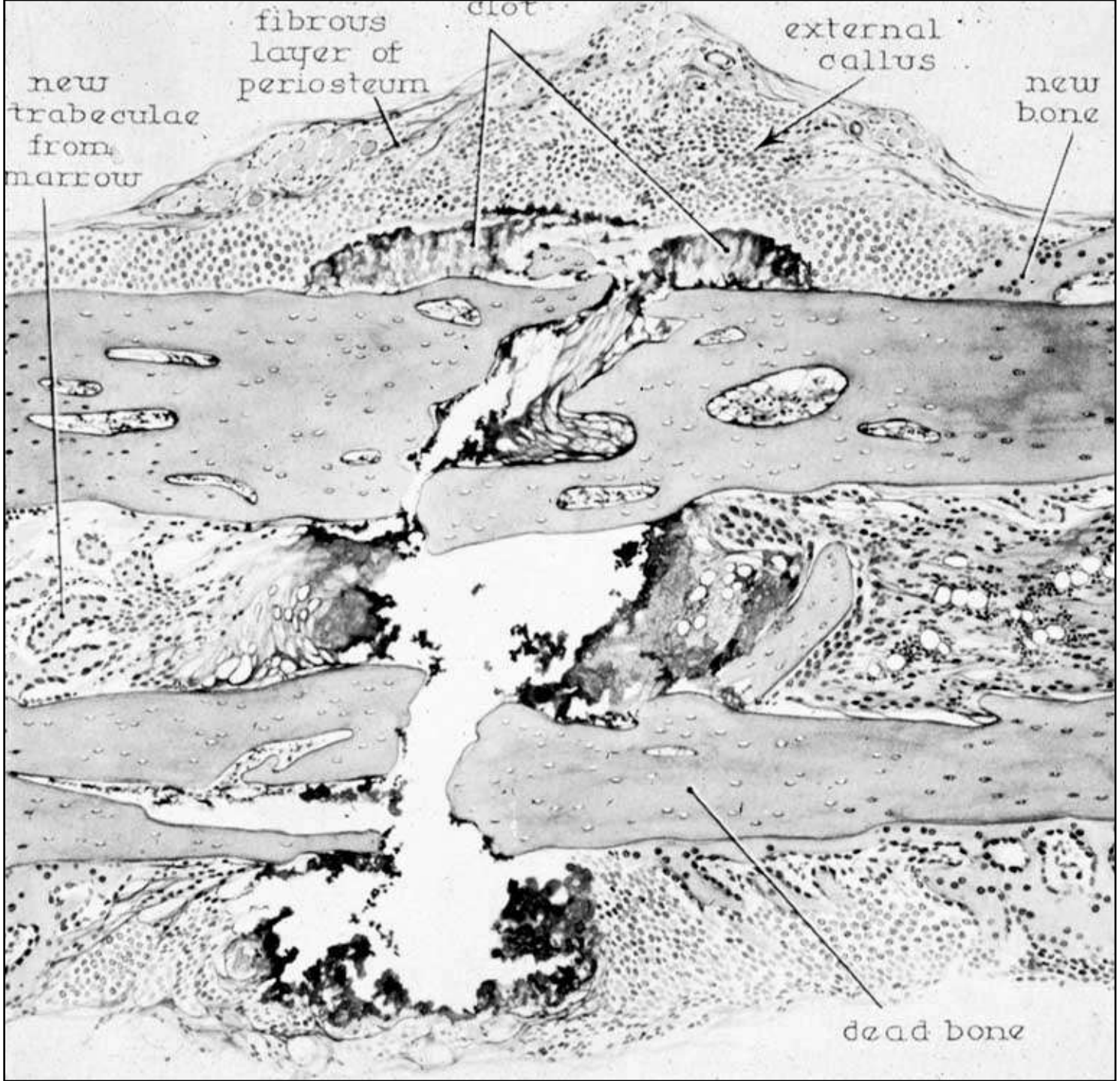
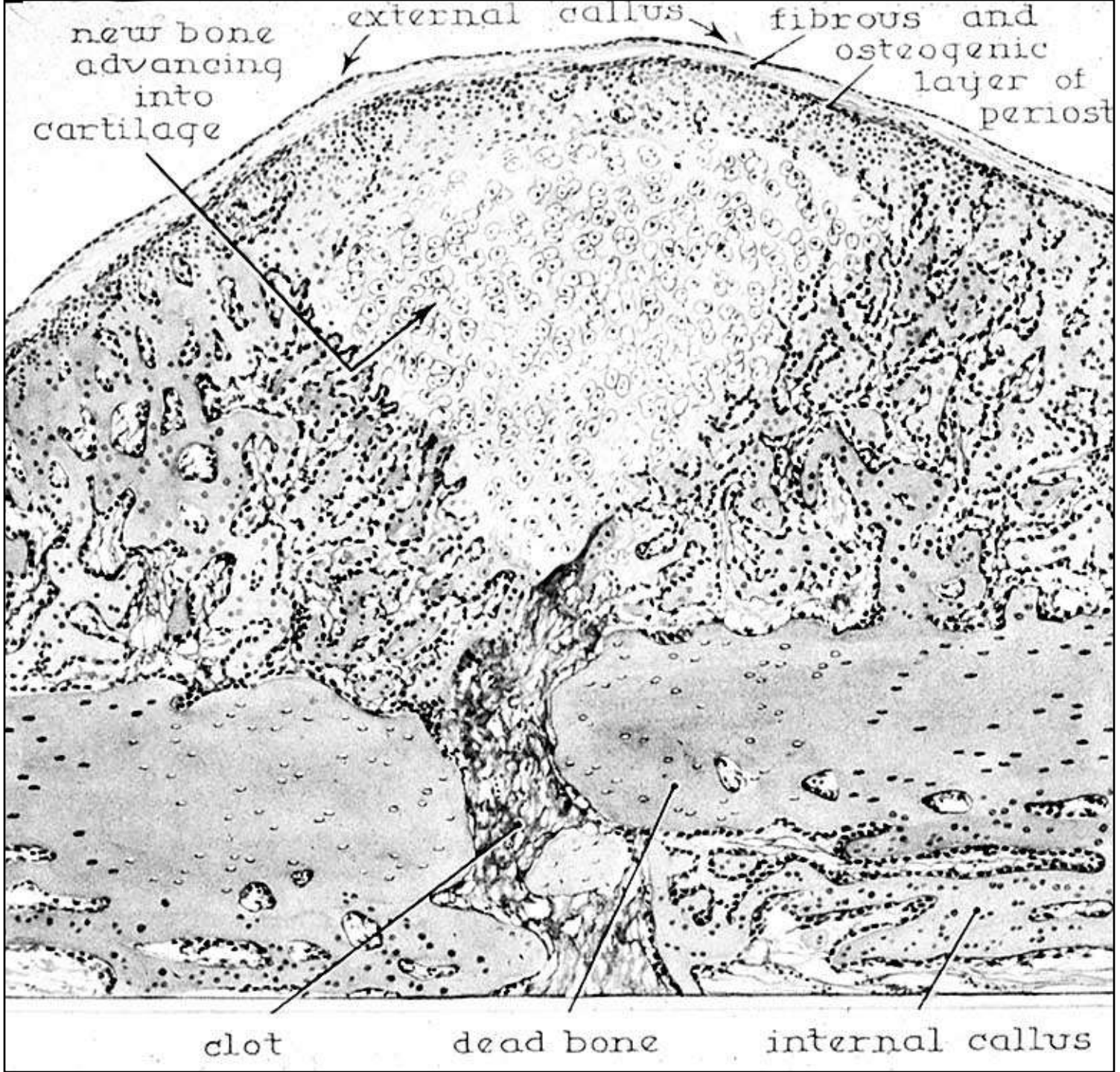
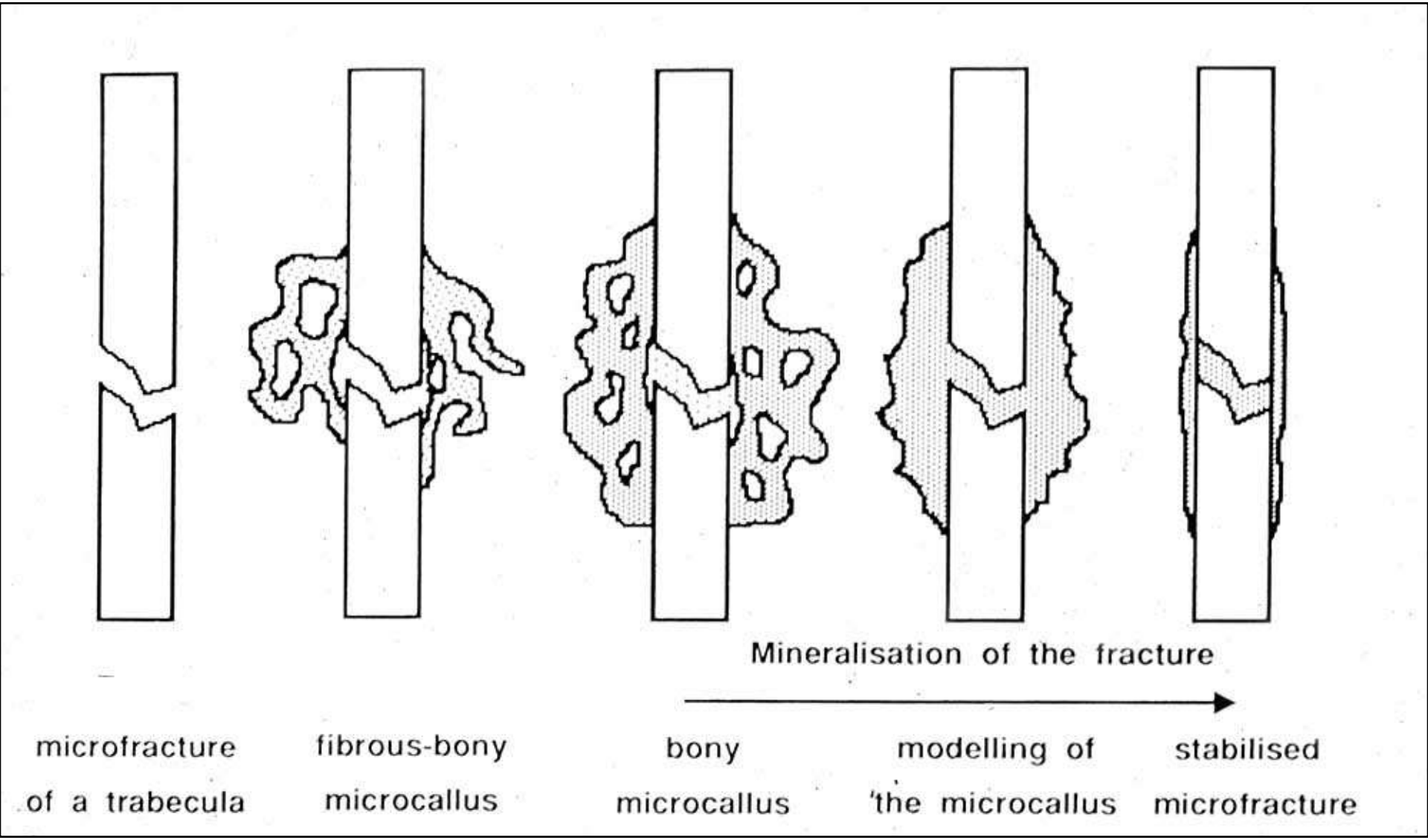


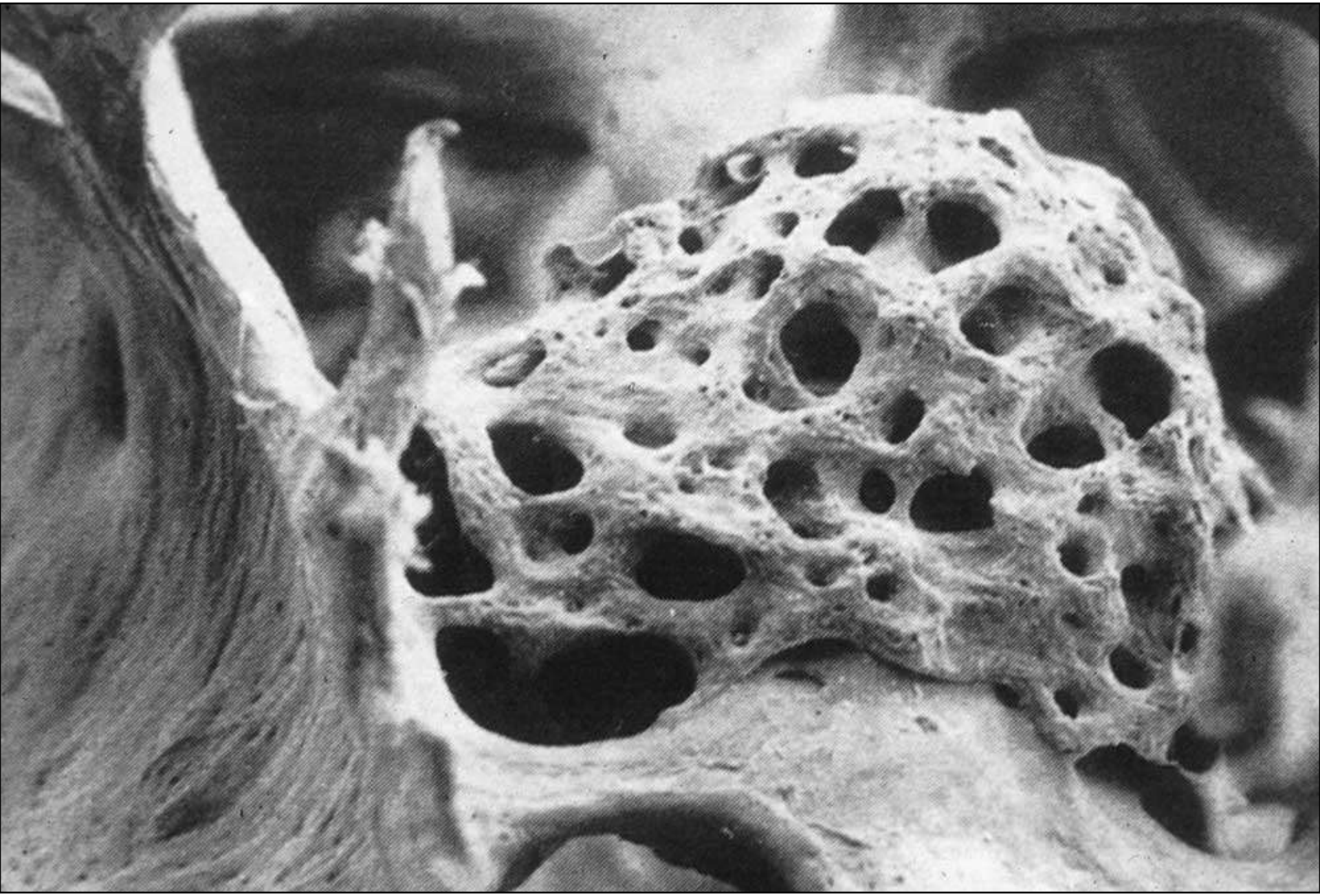
FIG. 21

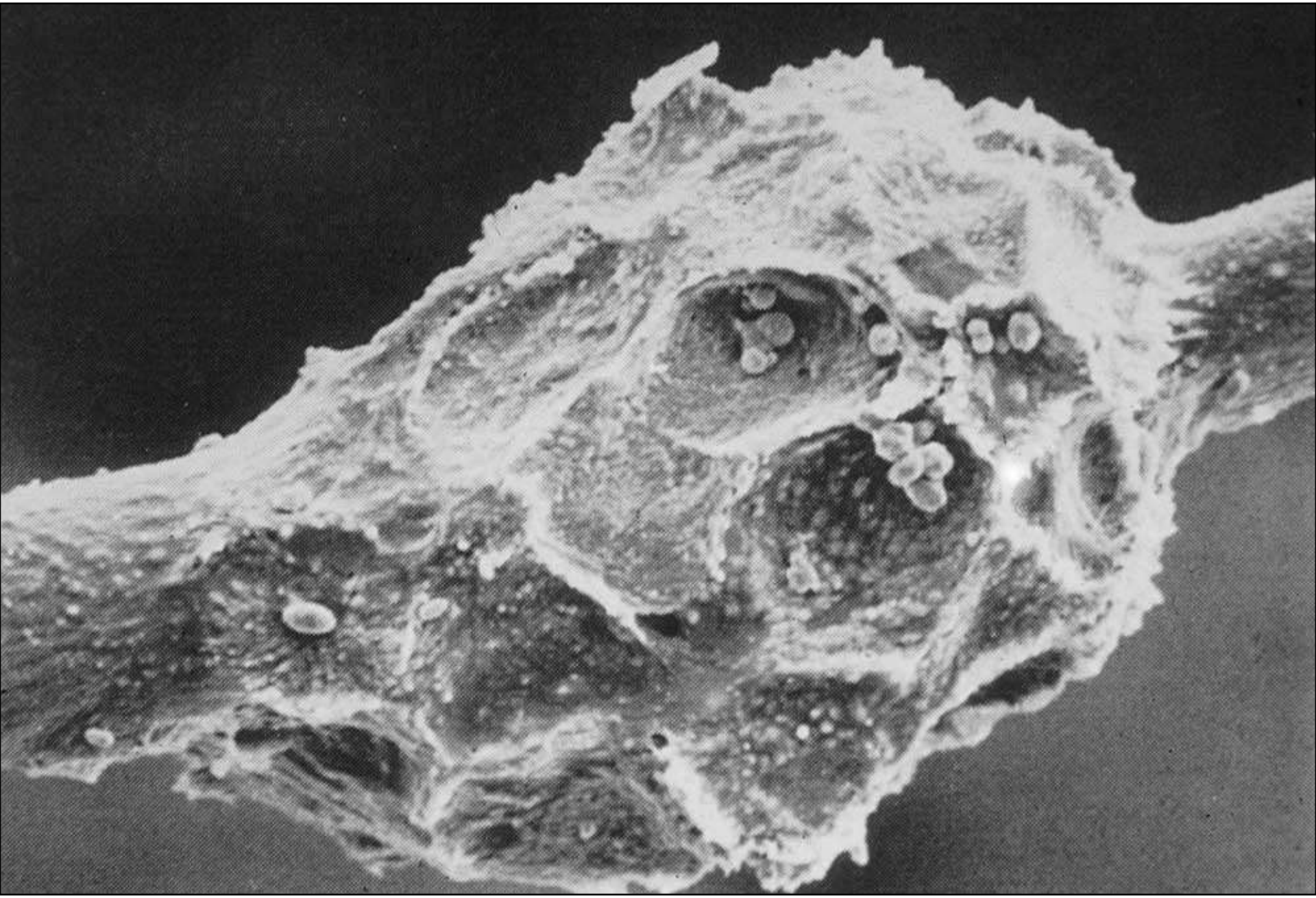
Diagram illustrating the hypothesis that the endothelial cell enlarges and undergoes a transformation to become a polymorphic mesenchymal cell. The reticular cell loses contact with the hematopoietic cells and also becomes a polymorphic mesenchymal cell. The polymorphic mesenchymal cell, in turn, is an osteoblast precursor cell.

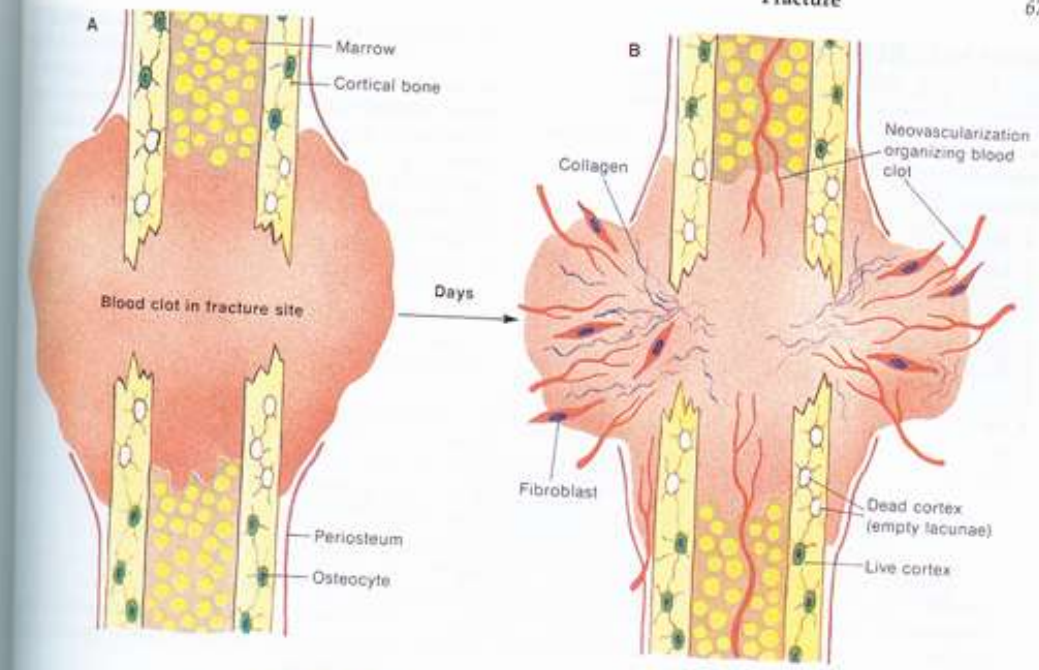

fibrous
layer of
periosteum
clot
external
callus
new
trabeculae
from
marrow
new
bone
dead bone

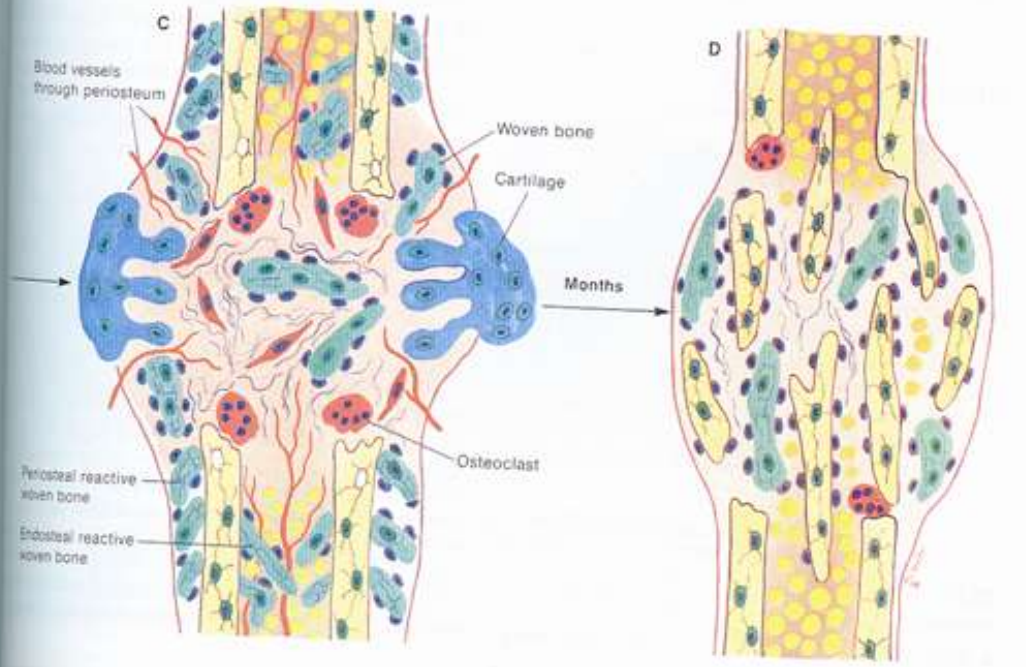

new bone advancing into cartilage
external callus
fibrous and osteogenic layer of periost
clot
dead bone
internal callus


Mineralisation of the fracture
microfracture of a trabecula
fibrous-bony microcallus
bony microcallus
modelling of the microcallus
stabilised microfracture






A
Marrow
Cortical bone
Blood clot in fracture site
Periosteum
Osteocyte
Days
B
Neovascularization
organizing blood
clot
Collagen
Fibroblast
Dead cortex
(empty lacunae)
Live cortex


C
Blood vessels
through periosteum
Woven bone
Cartilage
Osteoclast
Periosteal reactive
woven bone
Endosteal reactive
woven bone
Months
D

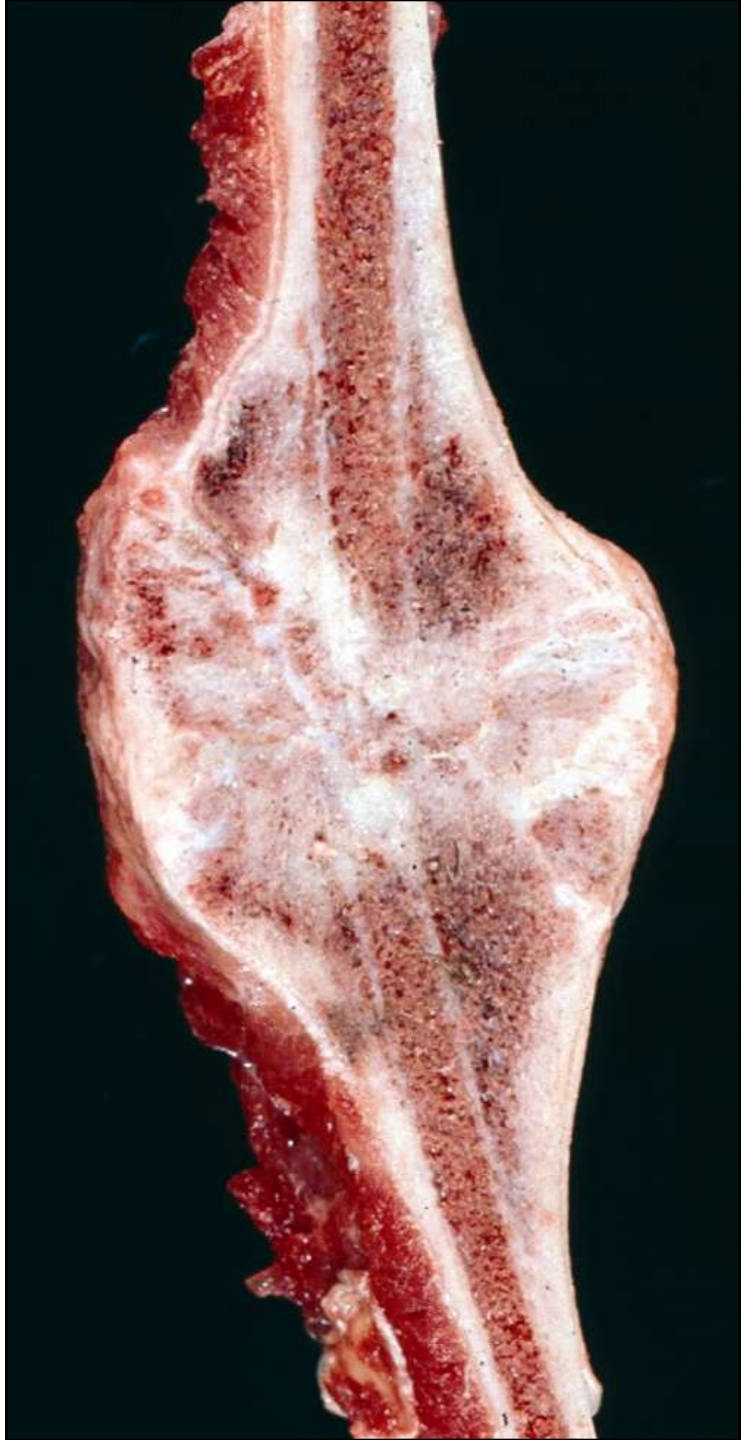
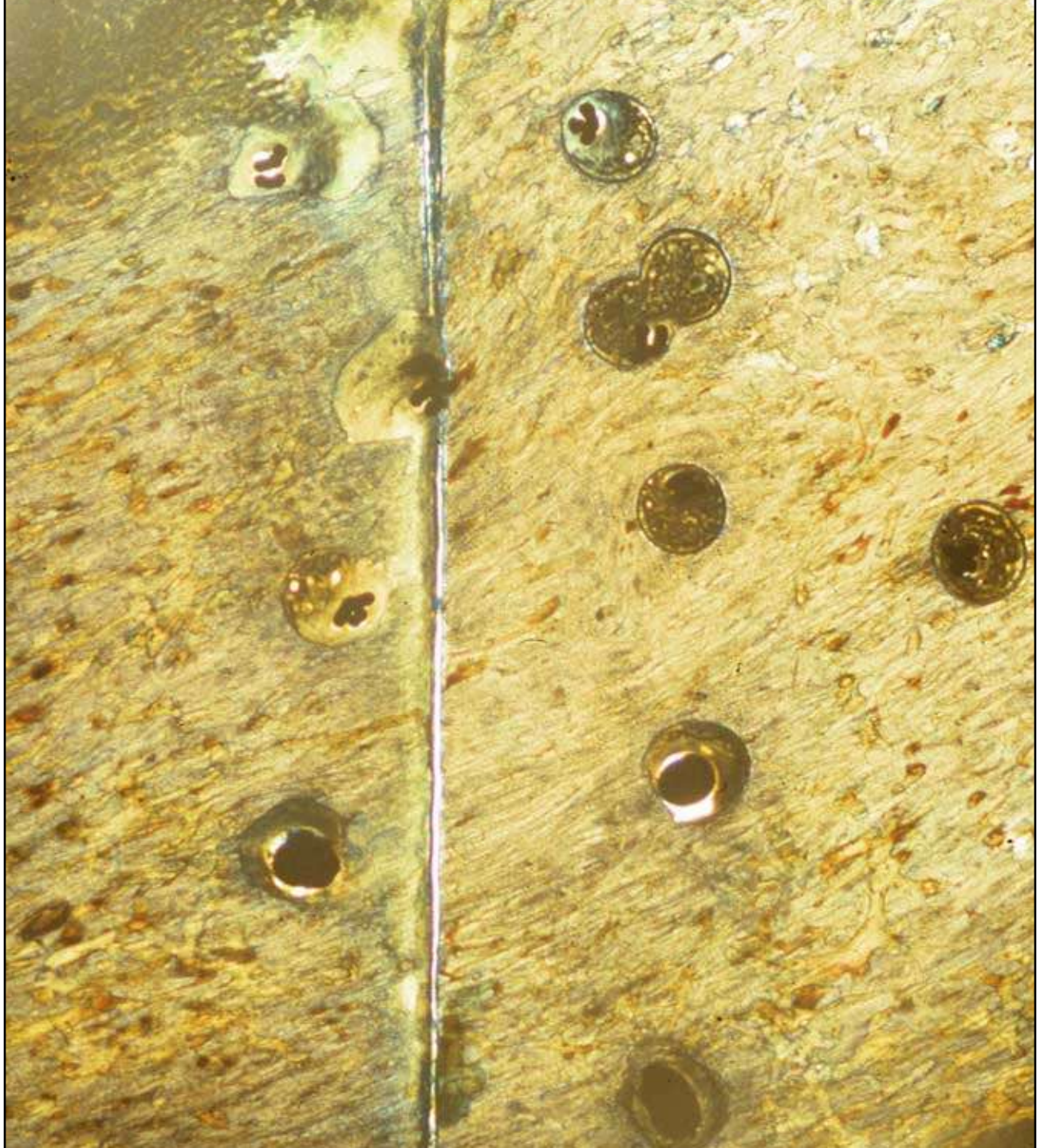


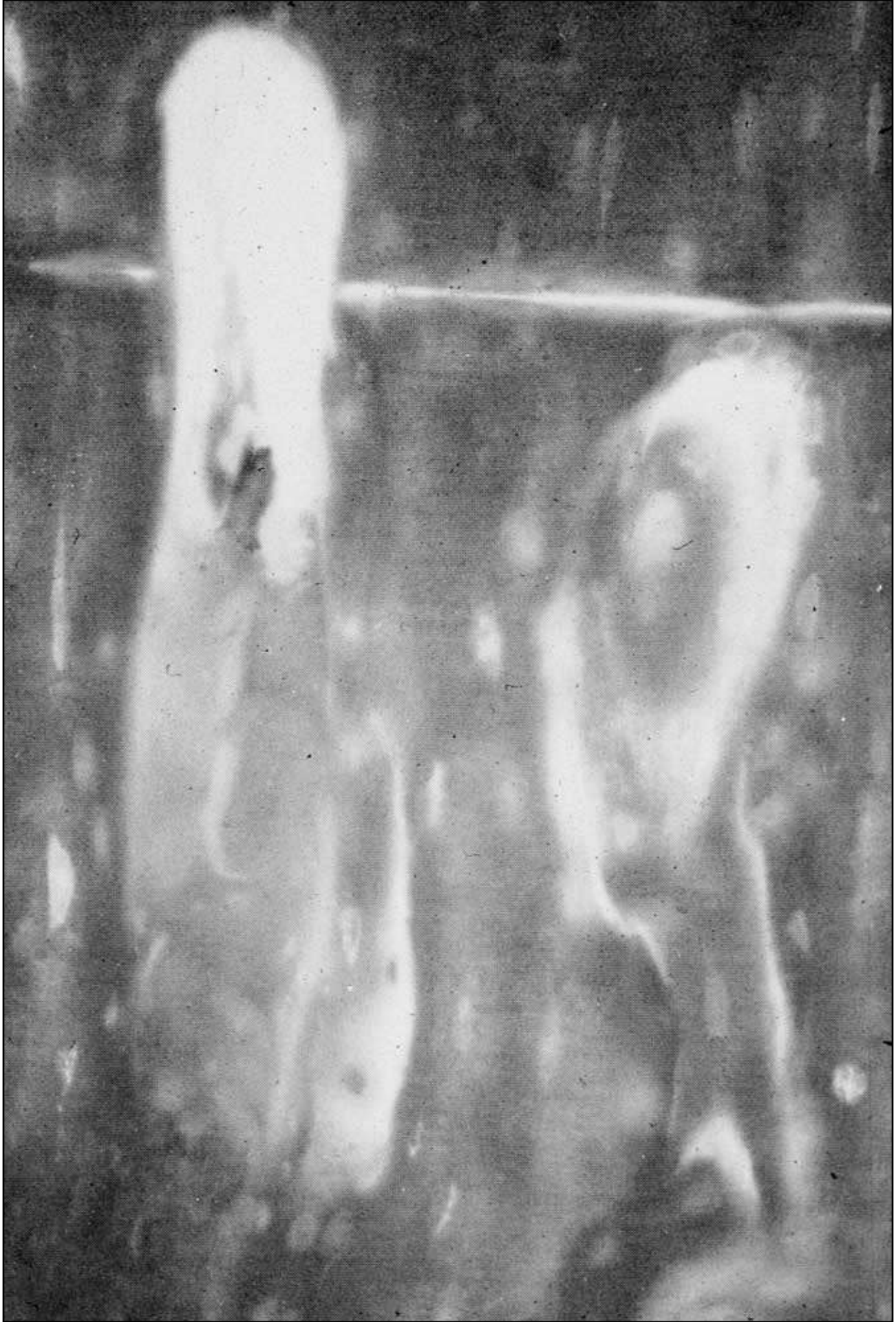
Table 9.1. Timetable for fracture healing (after Collins 1966)

12 h	Blood clot and exudate between fragments
24 h	Acute inflammation with migration of polymorphonuclears and macrophages
48 h	Formation of granulation tissue
5 days	Earliest new bone formation
7 days	Empty osteocyte lacunae in remaining dead fragments
3 weeks	Fibrous union; some primary callus
6 weeks	Periosteal shell of external callus; complete meshwork of woven bone
After 6 weeks	Progressive formation of secondary callus and subsequent remodelling

Rigid Fracture Healing

Contact Healing





Small Gap Healing

Less than 1 mm

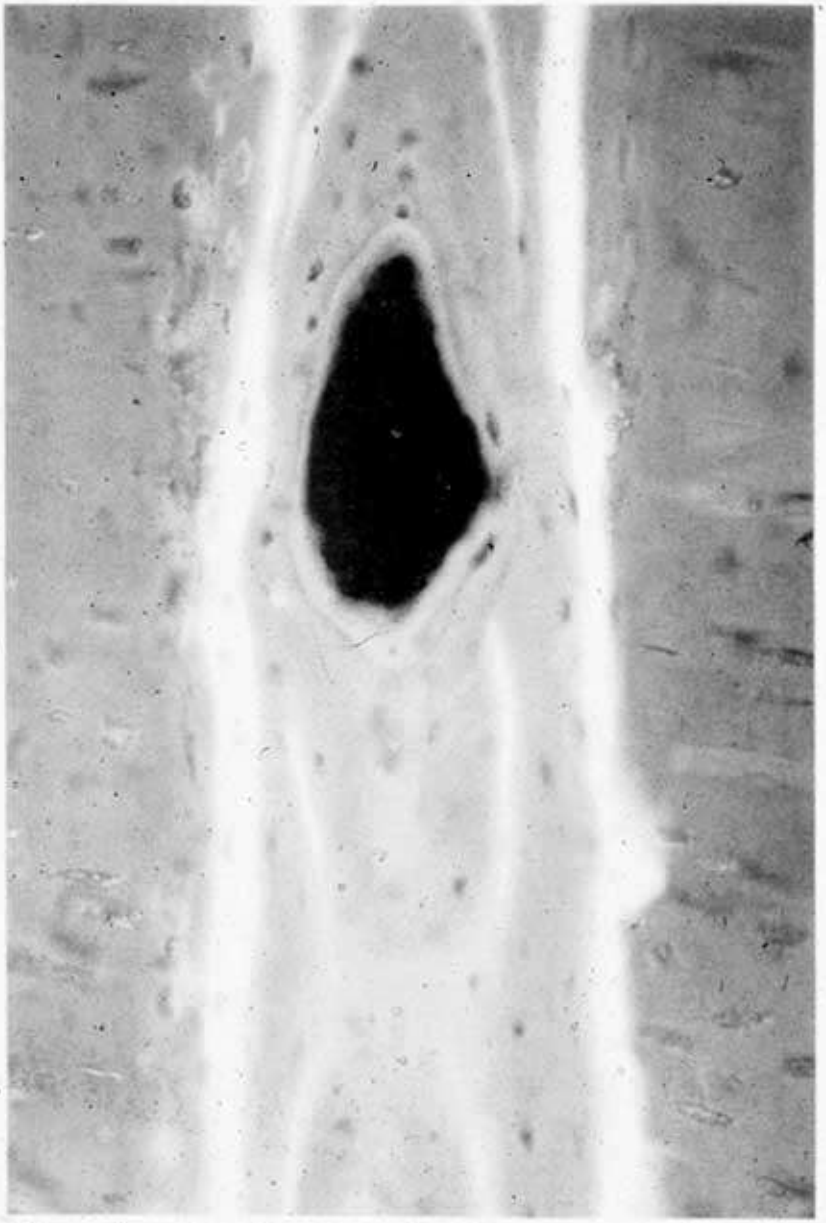
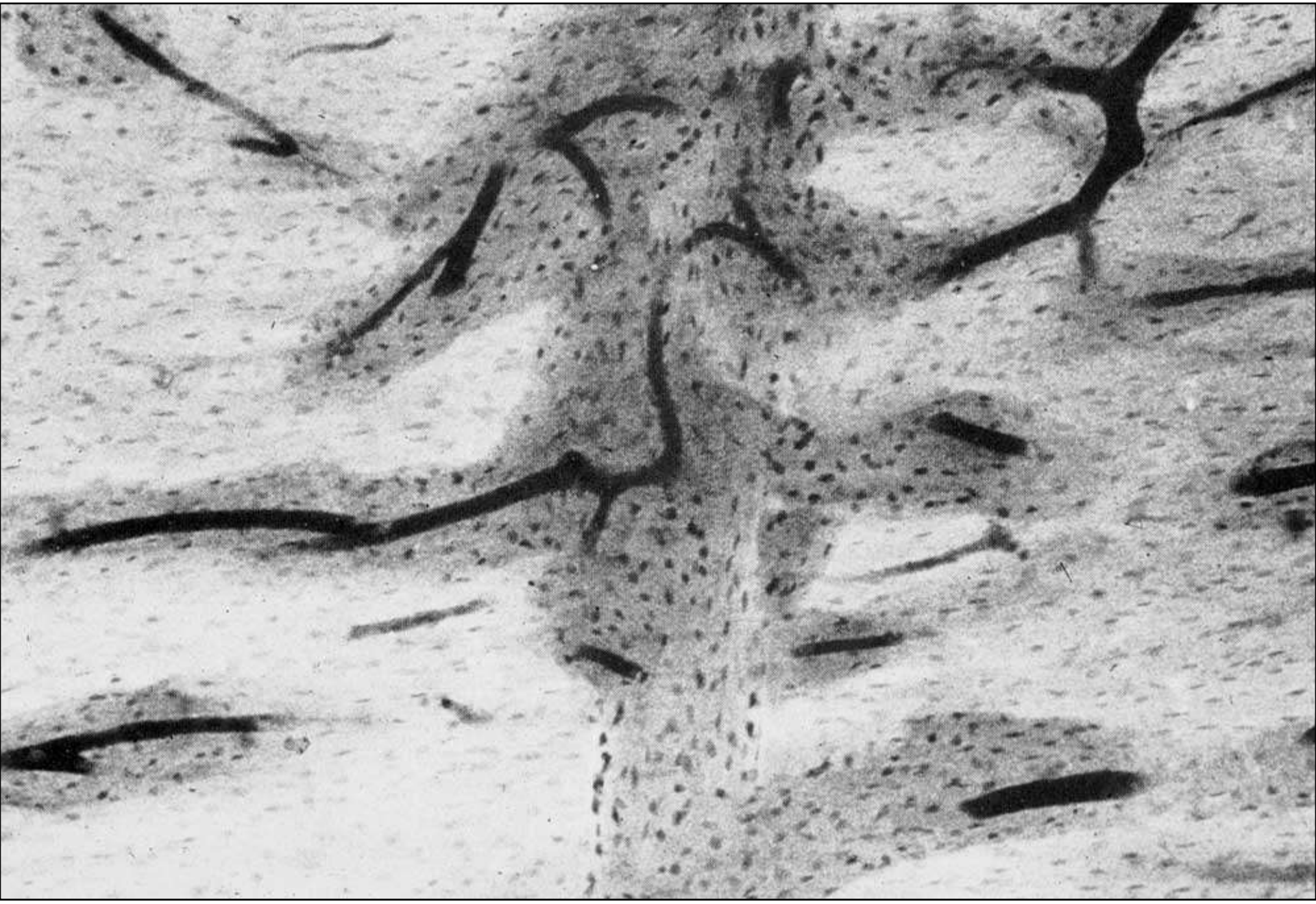


Figure 9–36. Primary bone formation in a stabilized narrow gap. Lamellar bone formation starts during the second week at the walls of the gap and continues to fill the gap at a speed of one to two micrometers per day. Blood vessels (black area) persist in the center of the filled gap. (See also Figure E, p. 385.)



Large Gap Healing

Greater than 1 mm

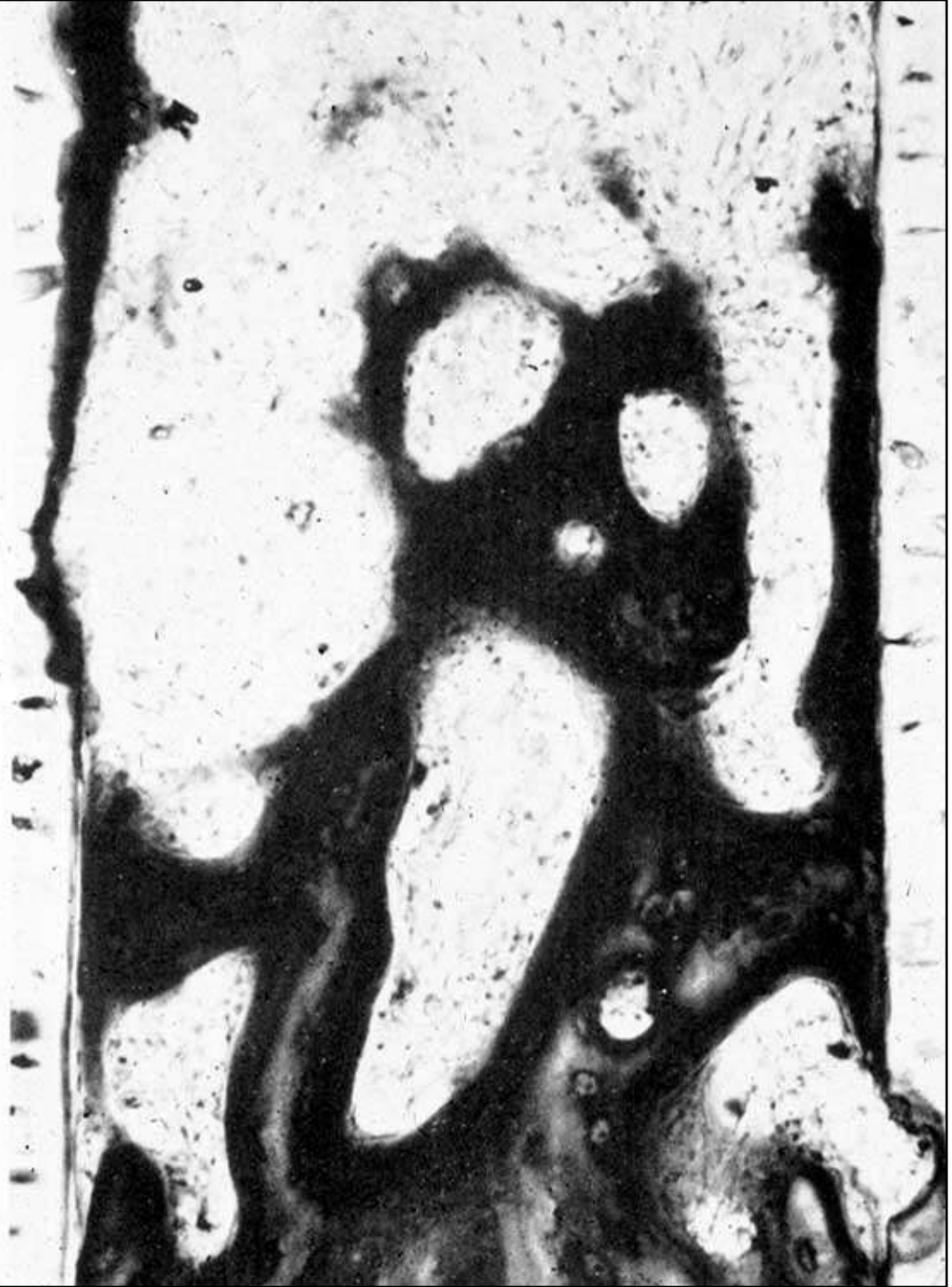
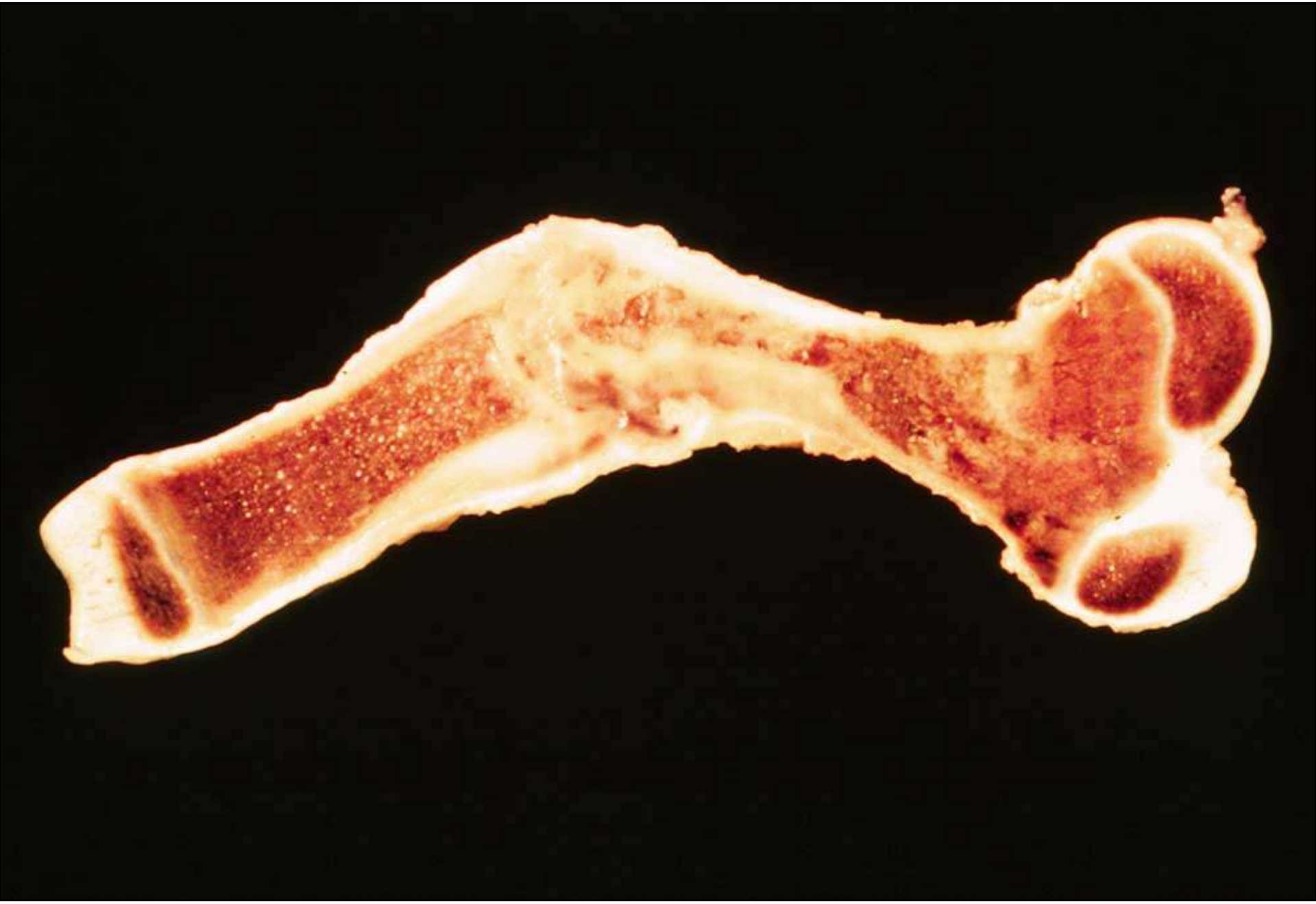


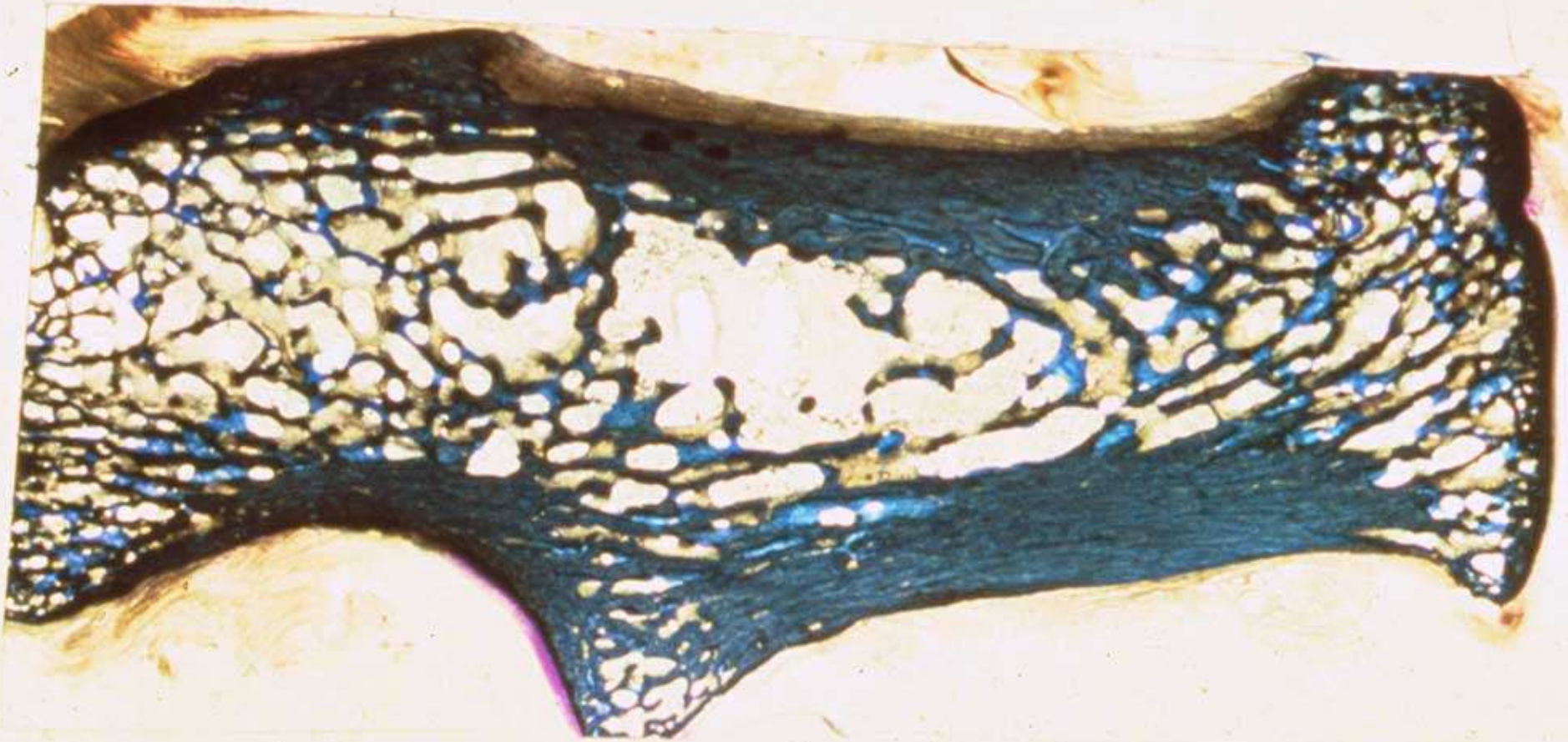
Figure 9–37. Filling of a wide gap, phase I: Fibrous bone formation. In a stabilized wide gap a network of fibrous bone is formed, which subdivides the whole space into smaller compartments. The holes in this network are approximately the size of osteons.

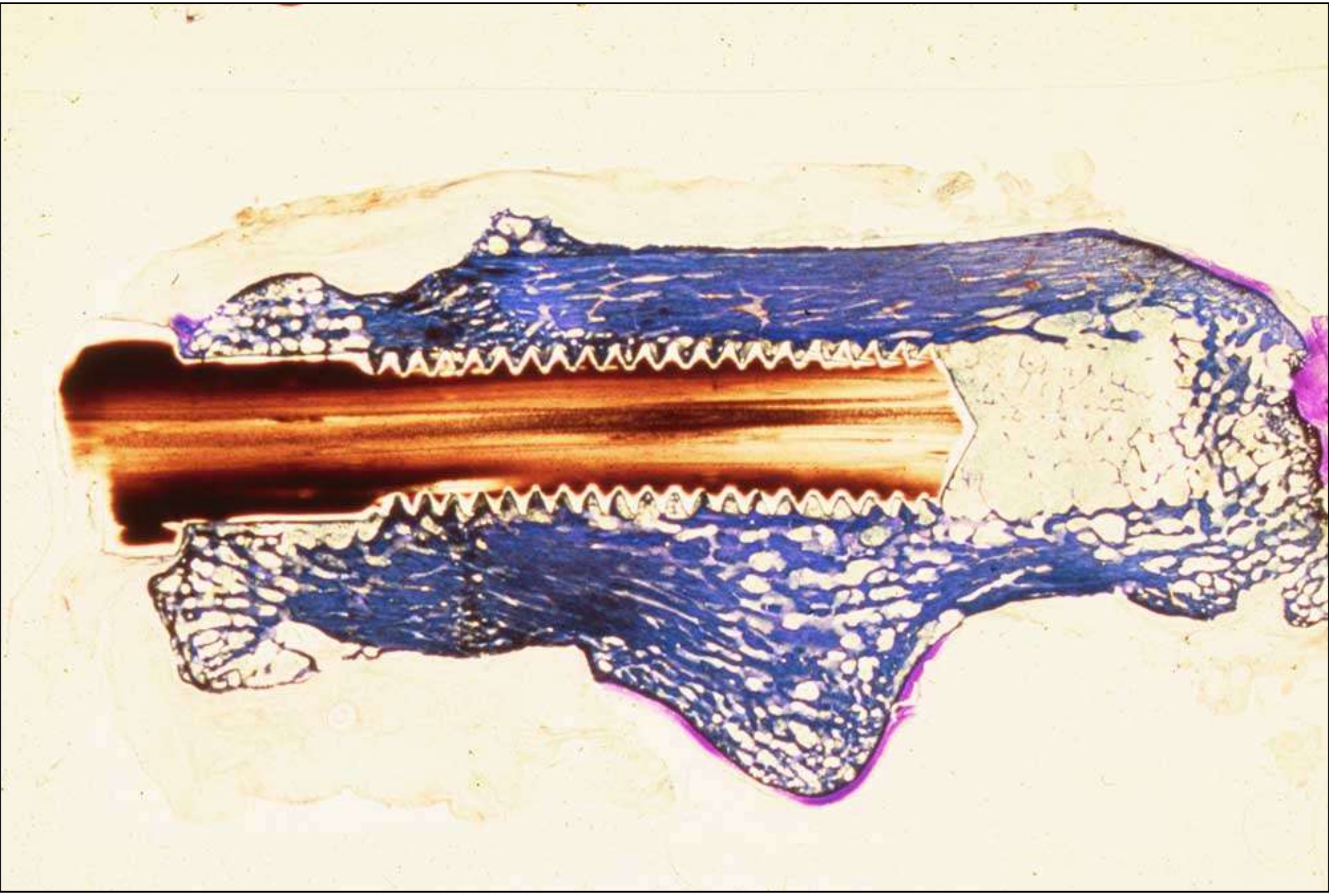
Complications of Fracture Healing

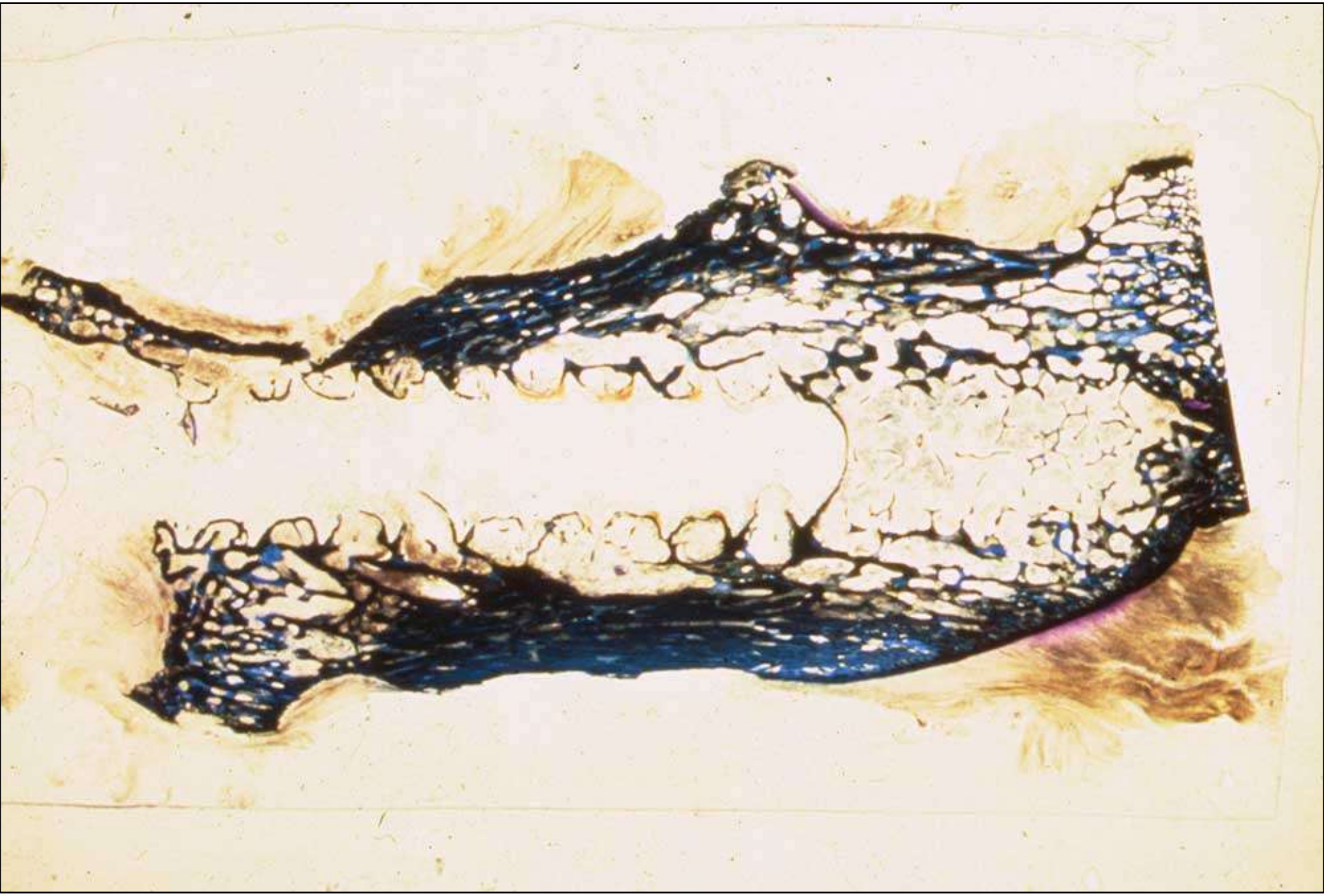
Mal-alignment



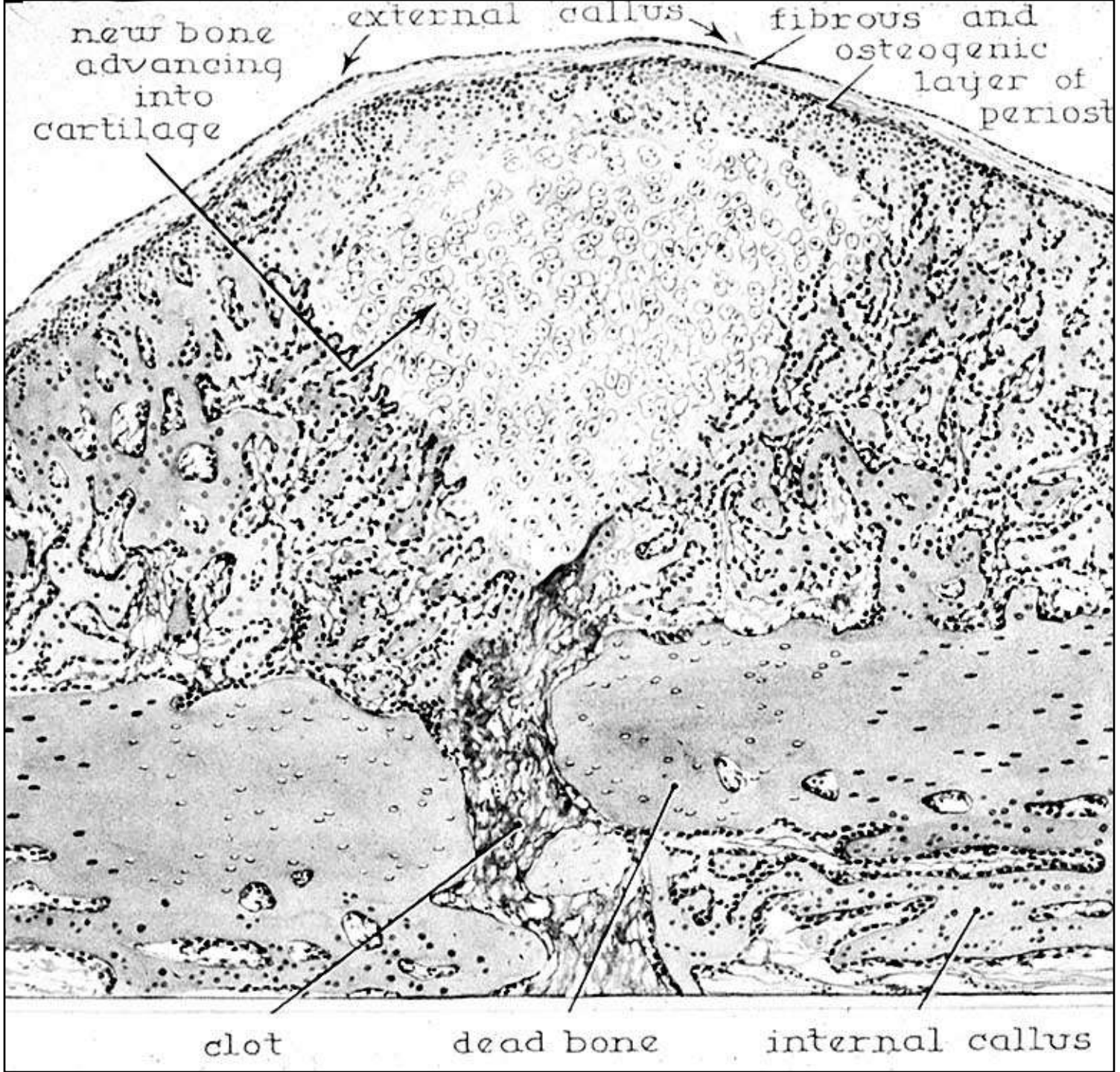
Stress Shielding



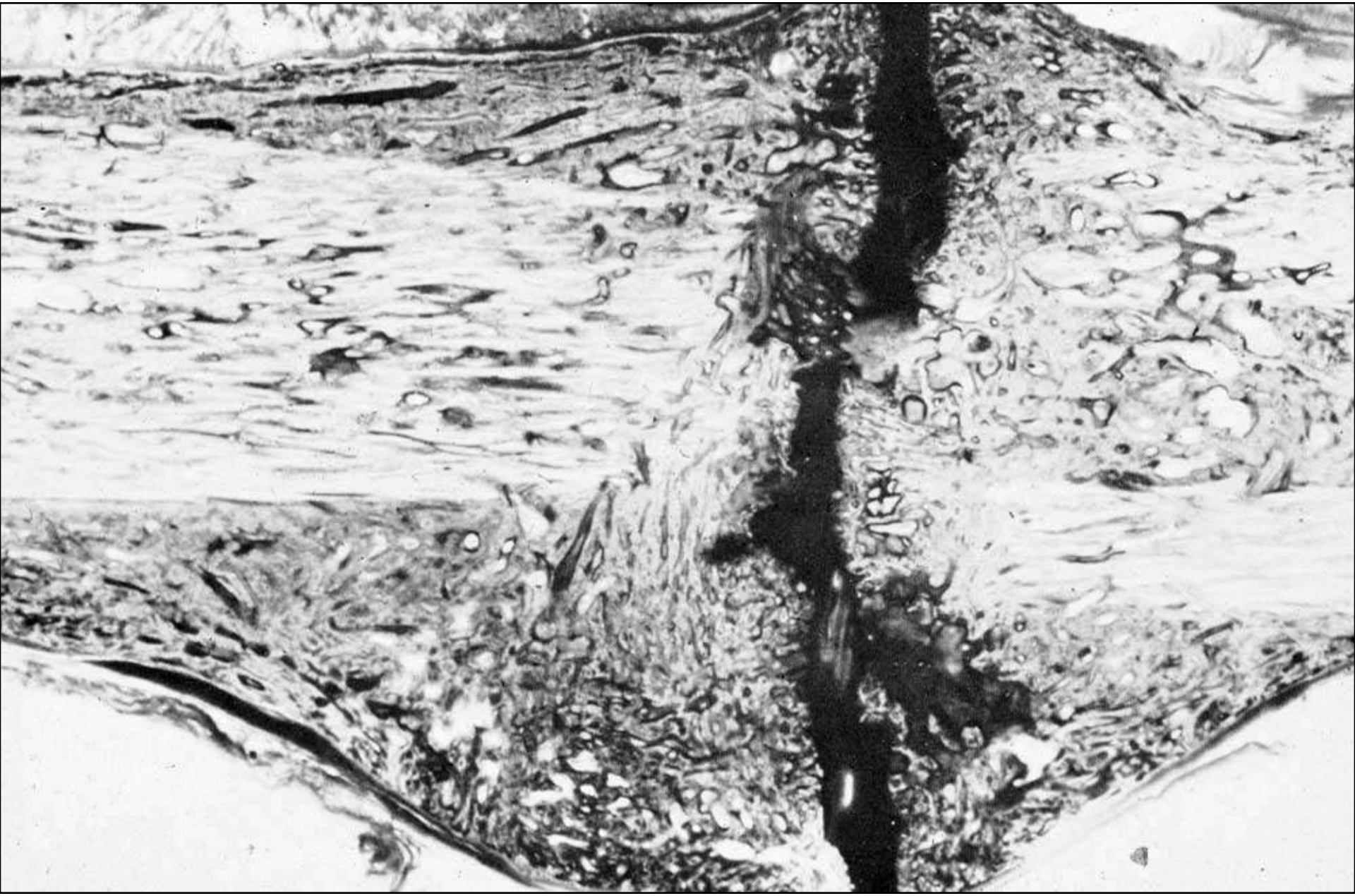




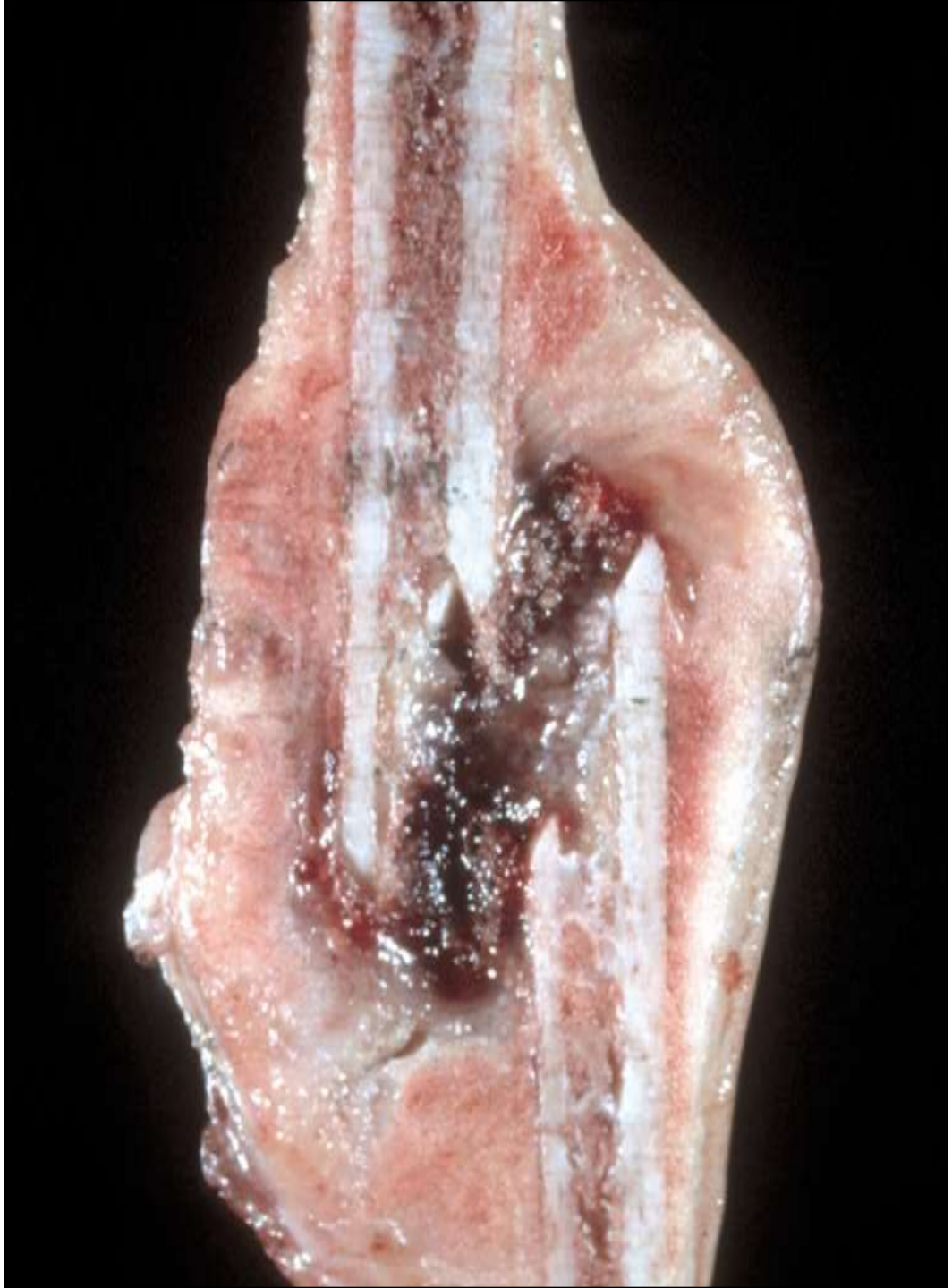
Excessive Cartilage Callus

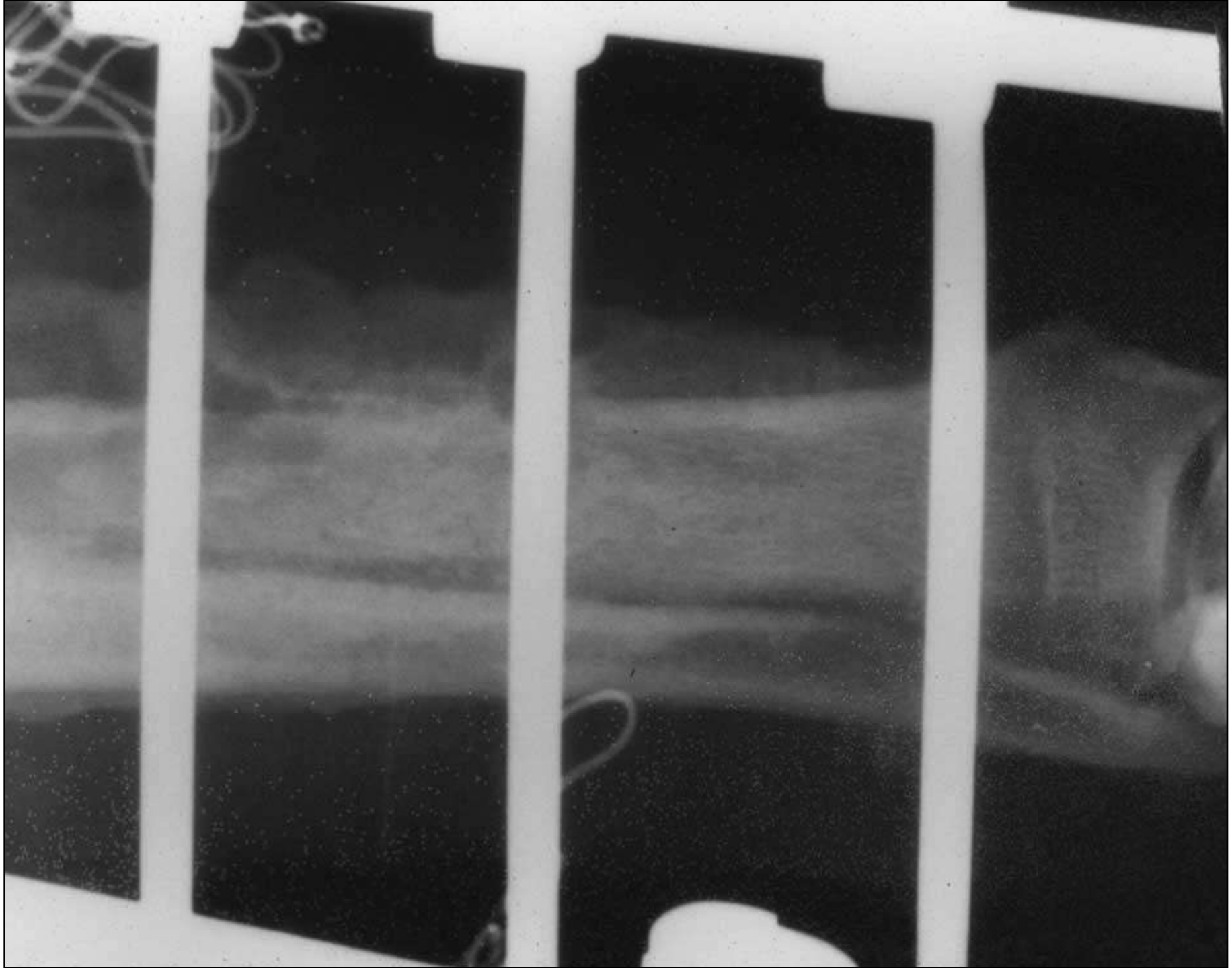

new bone advancing into cartilage
external callus
fibrous and osteogenic layer of periost
clot
dead bone
internal callus

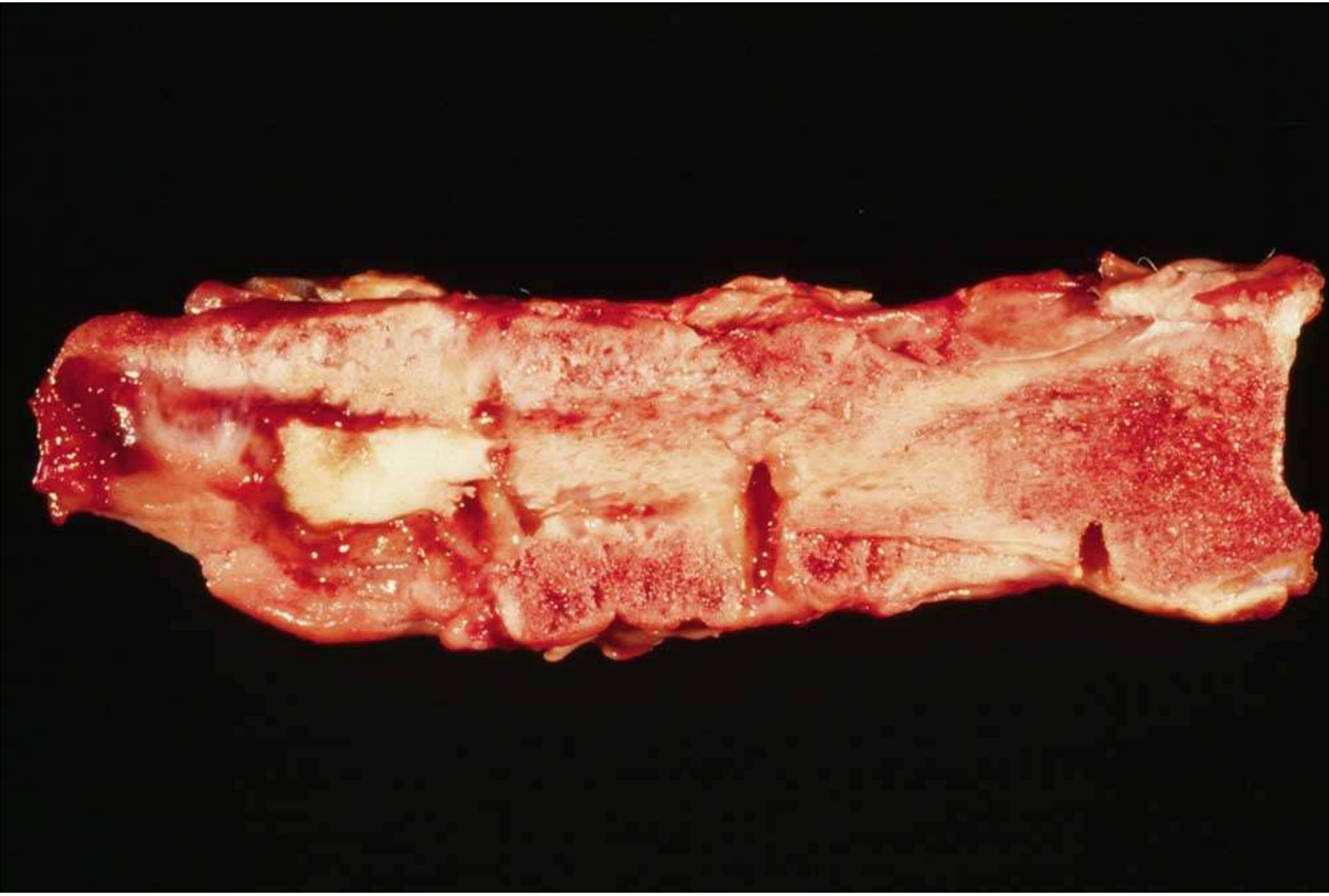
Fibrous Non-Union



Infection







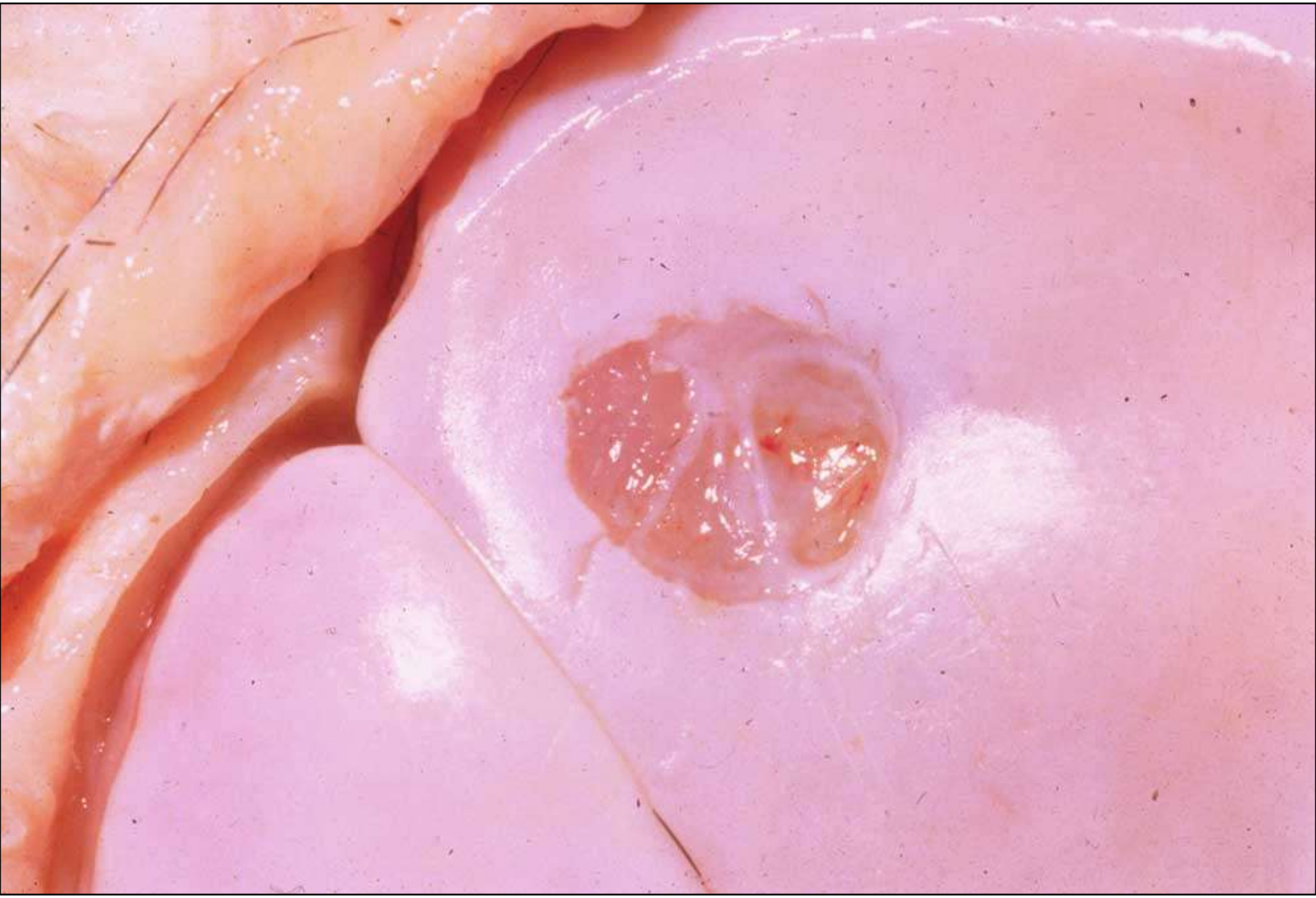
Cartilage Healing

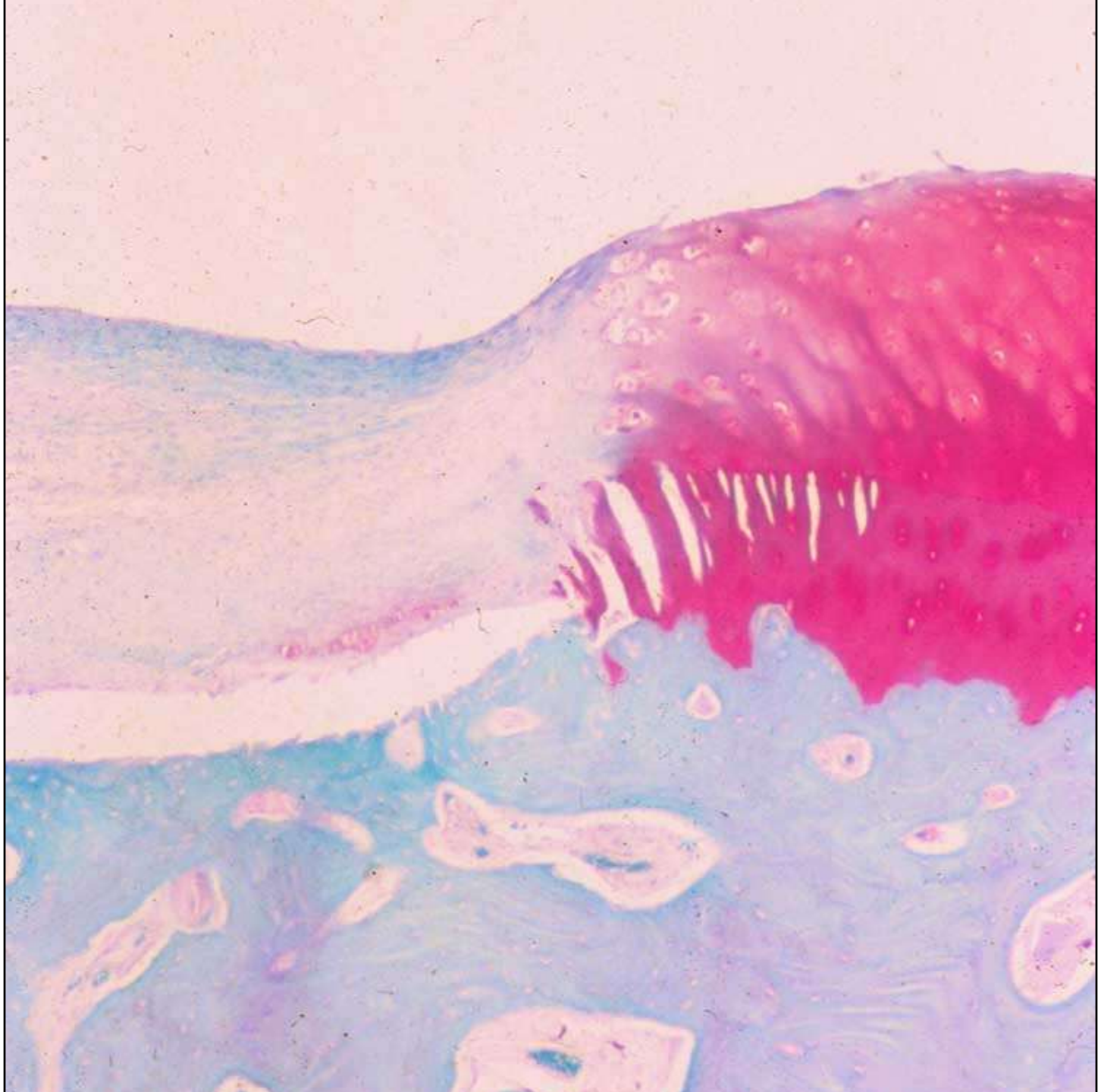
Partial thickness Defects

analagous to degenerative joint disease

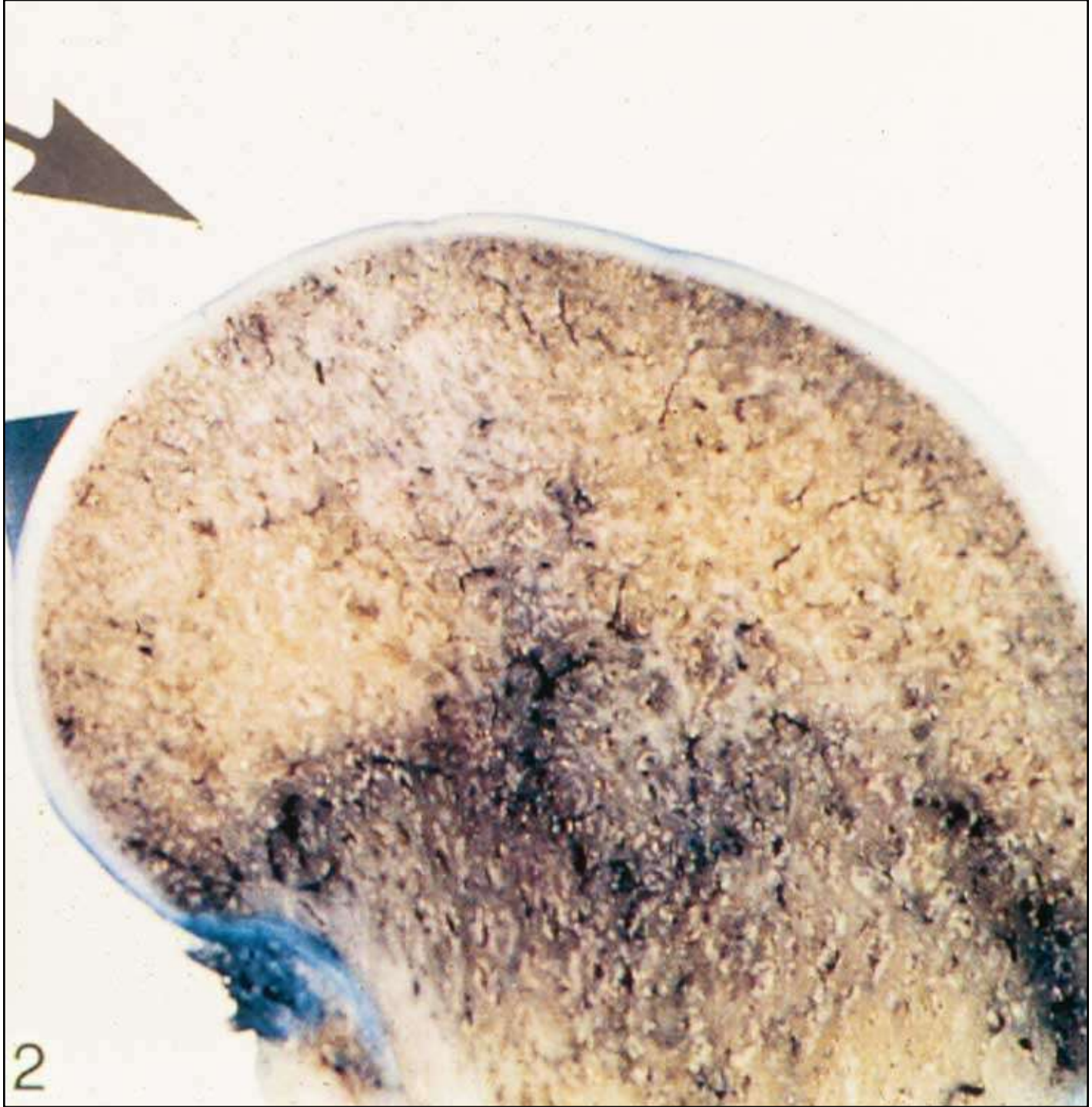
Full Thickness

(extends to bone with blood vessels)





Osteochondral Graft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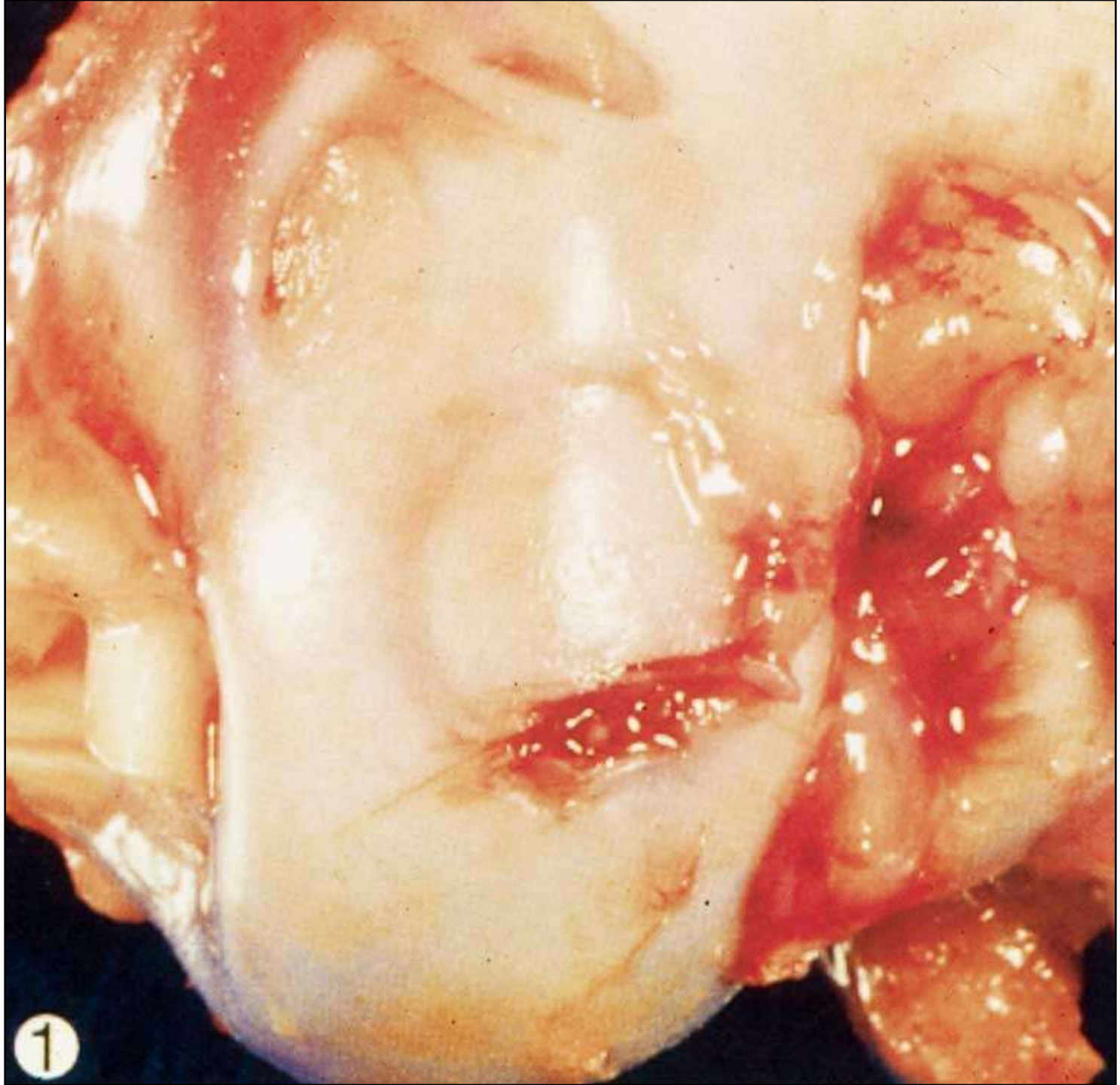
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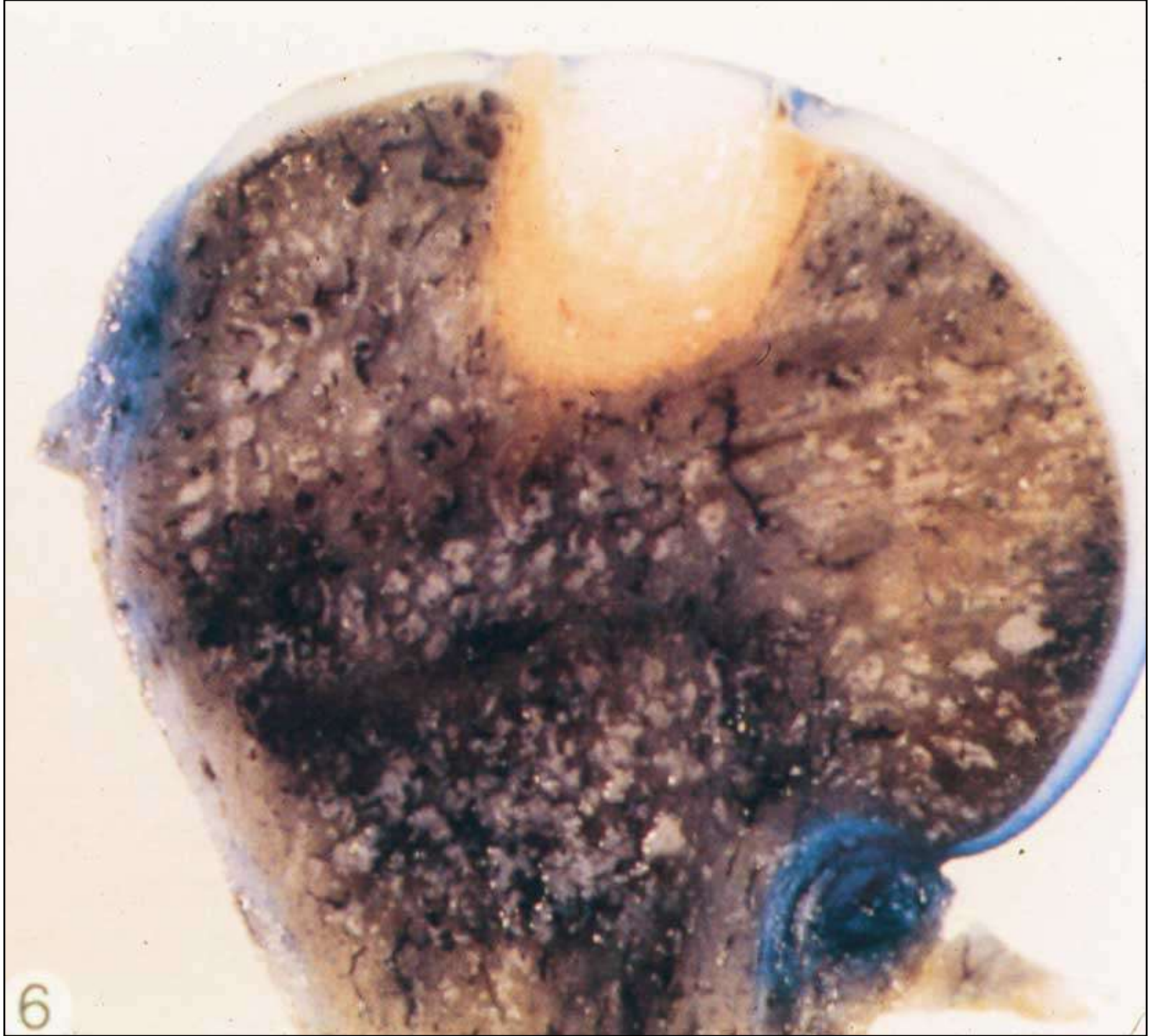

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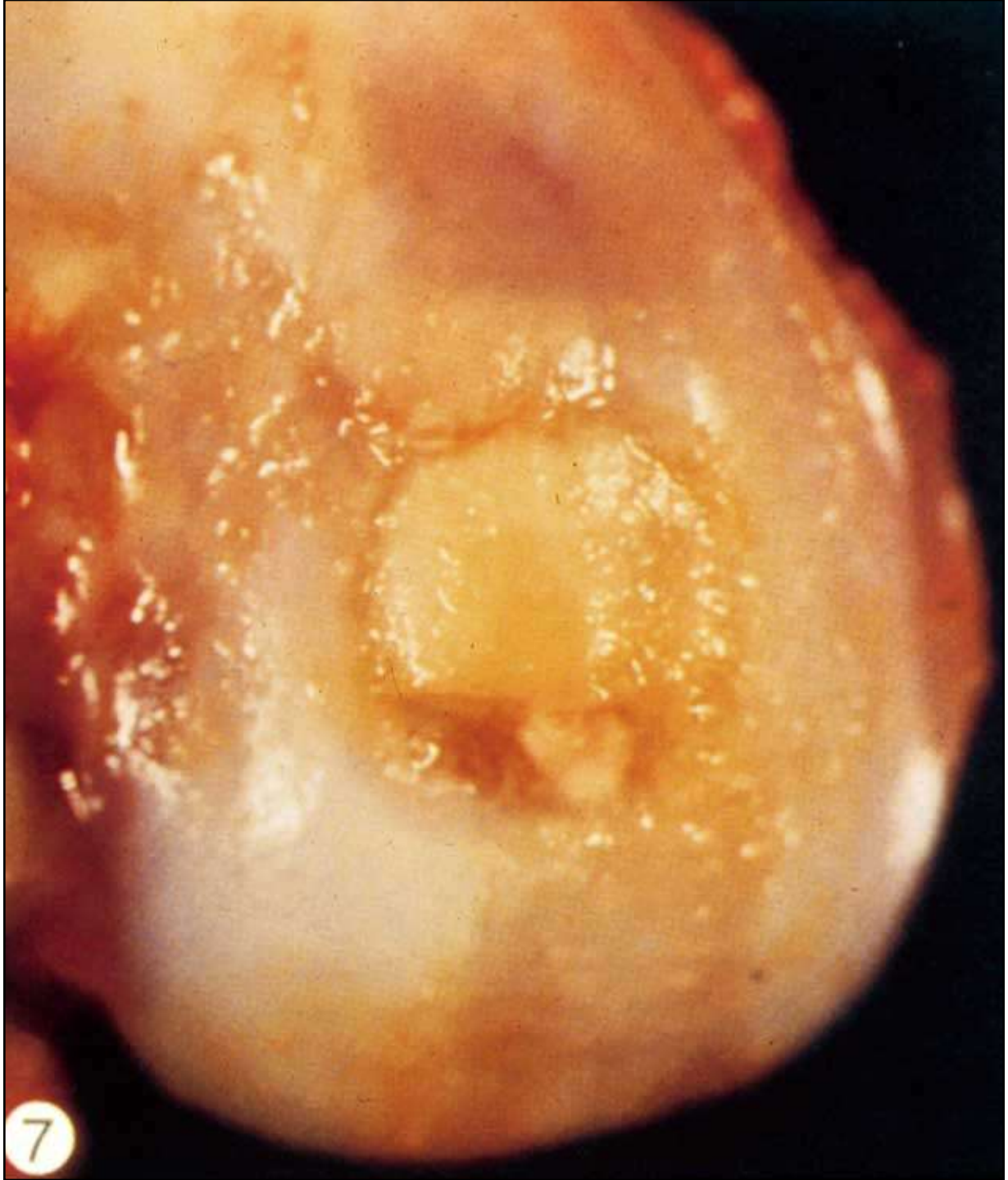
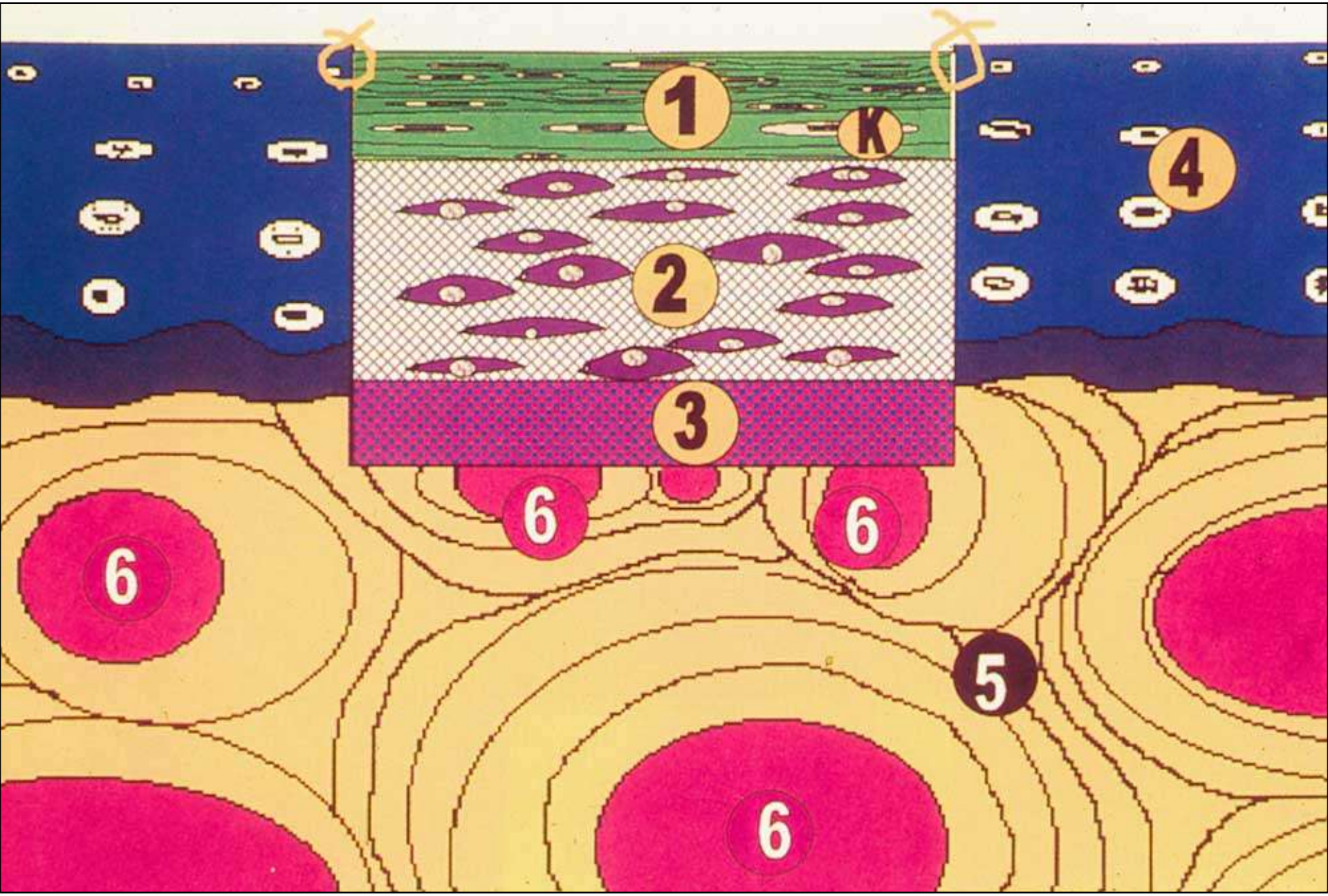

7

Implanatation of Cultured Chondrocytes


1
K
2
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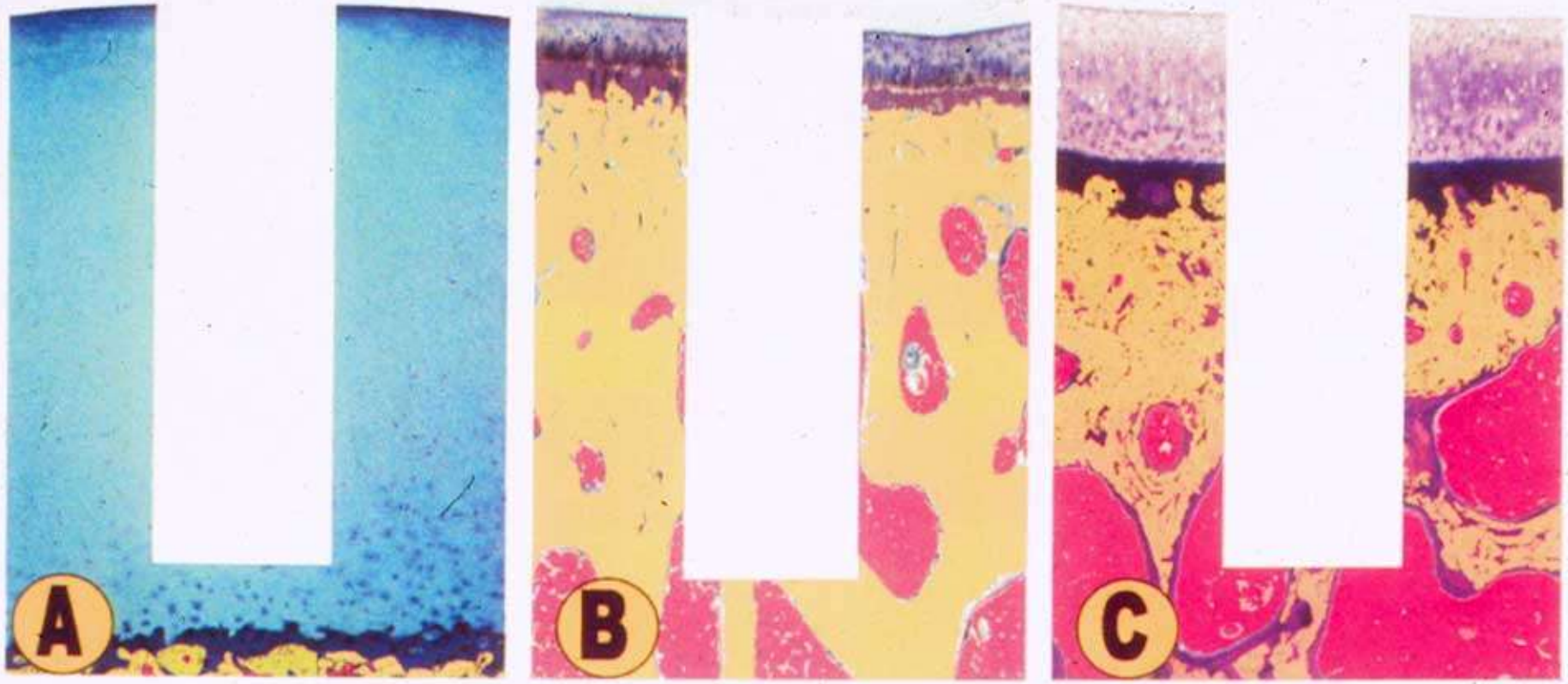

Fig 5A–C. Schematic representations of similar sized defects superimposed on light micrographs of (A) human, (B) rabbit and (C) goat articular cartilage (with varying portions of the underlying subchondral bone), represented at the same magnification. Because the thickness of the articular cartilage 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