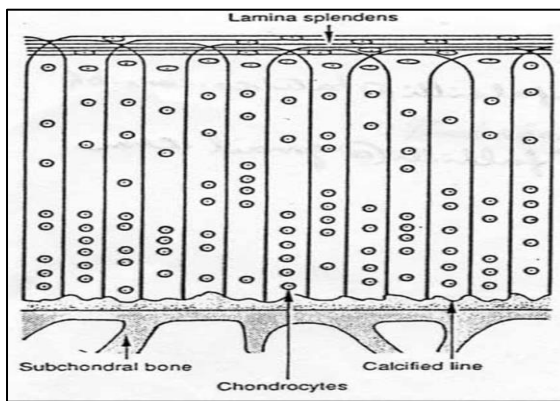




- Articular cartilage:

- **Thickness:** 6 mm (life-long: you have to preserve it because it is avascular and once it is damaged it is difficult for it to be regenerated).
- **Avascular** (not supplied with blood vessels), **alymphatic** (lymphatic vessels are not present), **hyaline** cartilage.
- Resist compressive forces and pressure by **changing its shape**, but it returns to its regular shape when the force/load is removed.
- **Articular cartilage is composed of:**
 - ✓ Chondrocytes:
 - ❖ They synthesize matrix components (collagen and proteoglycans).
 - ❖ Synthesize enzymes (collagenase).
 - ❖ Capable of phagocytosis.
 - ✓ Intercellular matrix: which is composed of collagen (mostly type-I), and proteoglycans (they bind large amount of water and link proteins, hyaluronic acid and glycosaminoglycans).



Normal articular cartilage. In the superficial zone, collagen fibers are oriented parallel to the surface of the cartilage forming the lamina splendens. Beneath this, the collagen is arranged perpendicular to the surface. A calcified line marks the boundary between articular cartilage and a thin zone of calcified cartilage which abuts on the subchondral bone plate.

- Osteoarthritis:

- **Definition:** chronic, progressive, non-inflammatory (but there is minimal infiltration by lymphocytes and plasma cells), degenerative destruction of the articular cartilage of weight-bearing joints leading to:
 - ✓ Joint narrowing.
 - ✓ Subchondral bone thickening.
 - ✓ And eventually, non-functioning painful joint.
- **Etiology:**
 - ✓ Primary osteoarthritis:
 - ❖ Degeneration of articular cartilage of unknown etiology but can be due to:
 - ✚ Increased unit load on the chondrocyte.
 - ✚ Decreased flexibility of the articular cartilage.
 - ✚ Increased rigidity of subchondral rough cartilage (calcified line) and cancellous bone (spongy bone) of the epiphysis.
 - ✚ Biochemical abnormalities.
 - ✚ Genetic influences.
 - ❖ Early changes may be arrested or delayed:
 - ✚ Metabolic alteration of chondrocytes.
 - ✚ Alteration in the matrix components of the articular cartilage (increased water and decreased proteoglycans).
 - ✚ Thickening of the subchondral bone plate thus preventing dissipation (توزيع) of compressive forces from the articular cartilage to the underlying bone.



✚ Chemical mediators (such as prostaglandins and IL-1) suppress chondrocytes proteoglycans synthesis and metabolism.

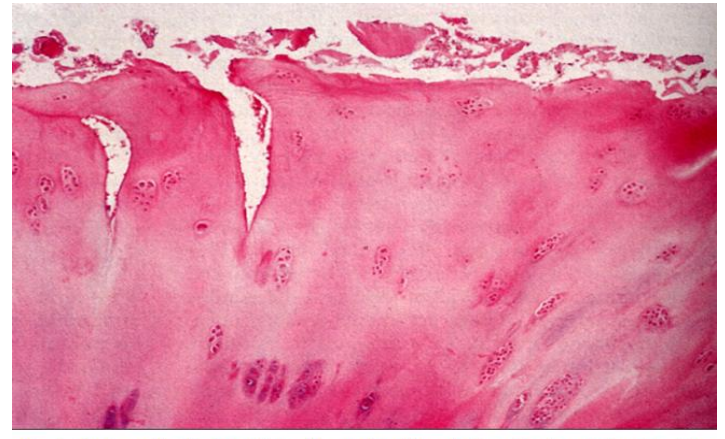
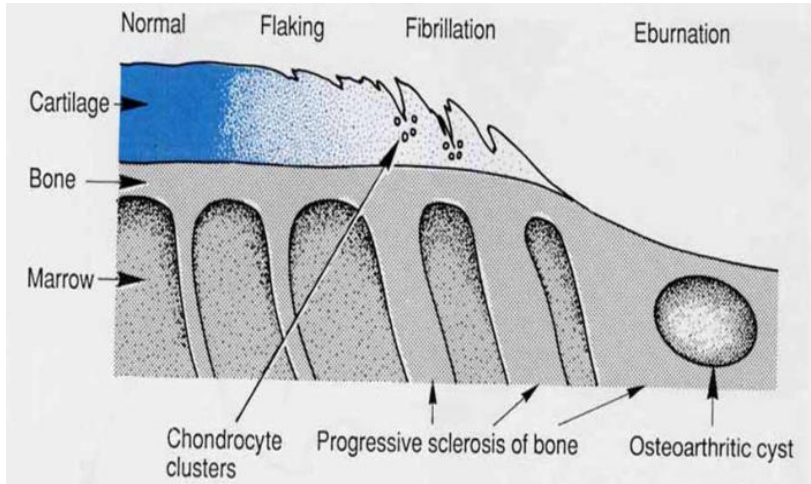
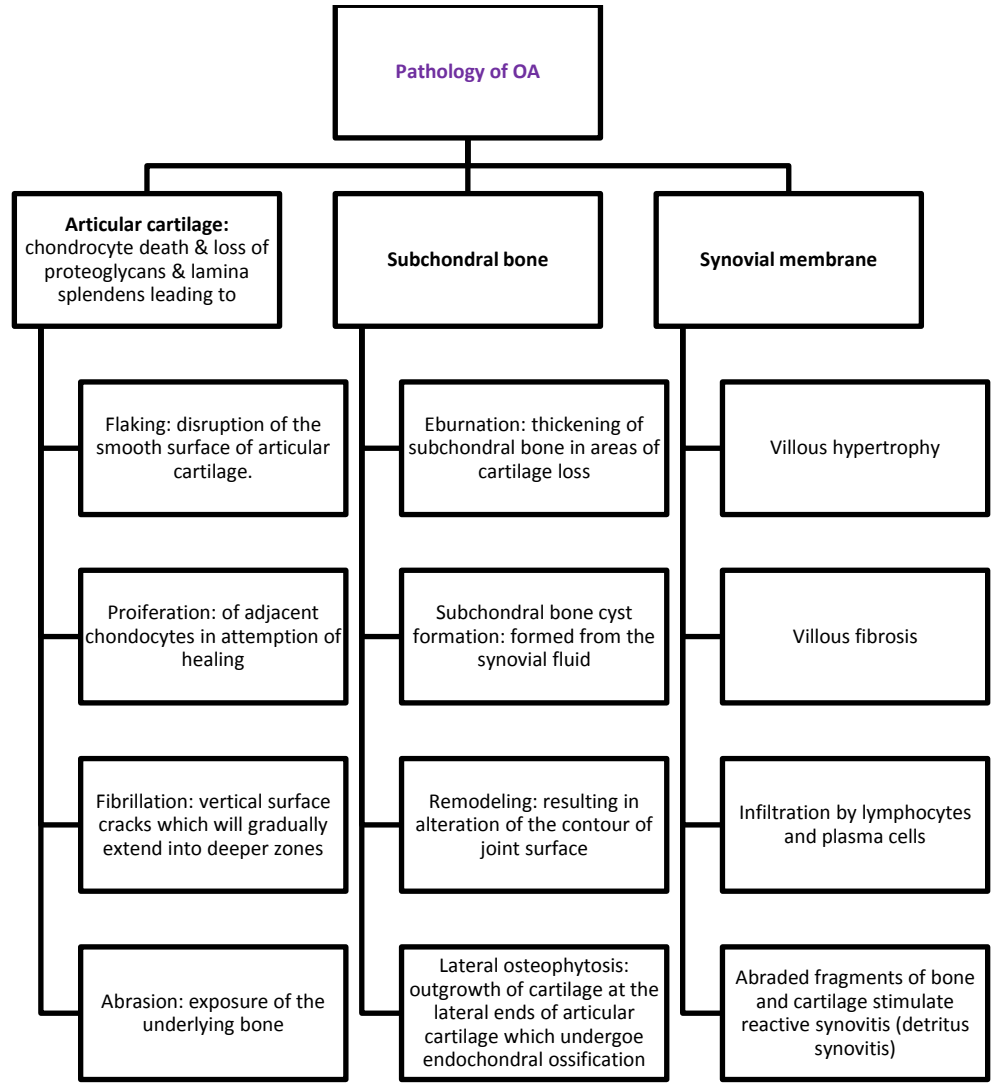
❖ Sequences of primary osteoarthritis:

- Damage: increased destruction of articular cartilage.
- Repair: focal replication of chondrocytes and increased synthesis of matrix.
- Failure of repair: due to continuing stress factors.

✓ Secondary osteoarthritis:

- ❖ Underlying joint disorders.
- ❖ Abnormal stress.
- ❖ Metabolic/endocrine.
- ❖ Neuropathic disorders.

• Pathogenesis:



Early Osteoarthritis. Histology of a section through the articular surface showing "fibrillation" and "fissuring".



Osteoarthritis. Articular surfaces of the femoral condyles shows advanced osteoarthritis. There are areas of complete cartilage loss with eburnated subchondral bone.

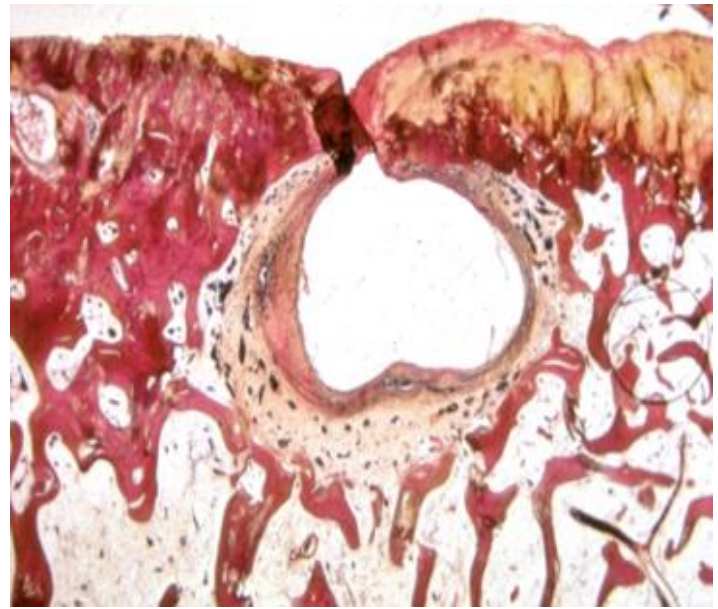


Table 20.10 A comparison of rheumatoid arthritis and osteoarthritis

	Rheumatoid arthritis	Osteoarthritis
Age	Any age, mainly 25–55	Predominantly in elderly
Affected joints	Symmetrical arthritis Metacarpophalangeal, proximal interphalangeal, wrists, shoulders, knees	Often one affected joint – hip, knee, ankle Hereditary form – proximal interphalangeal and distal interphalangeal joints
Synovium	Hyperplastic, dense chronic inflammation	Mild 'secondary' inflammation
Articular cartilage	Eroded by pannus from periphery	Flaking, fibrillation and loss on weight bearing surface
Subchondral bone	Osteoporotic Marginal erosions	Sclerotic Cysts
Osteophytes	Usually absent	Present
Systemic disease	Yes – see text for extra-articular manifestations	No
Pathogenesis	Autoimmune disease	Degenerative

