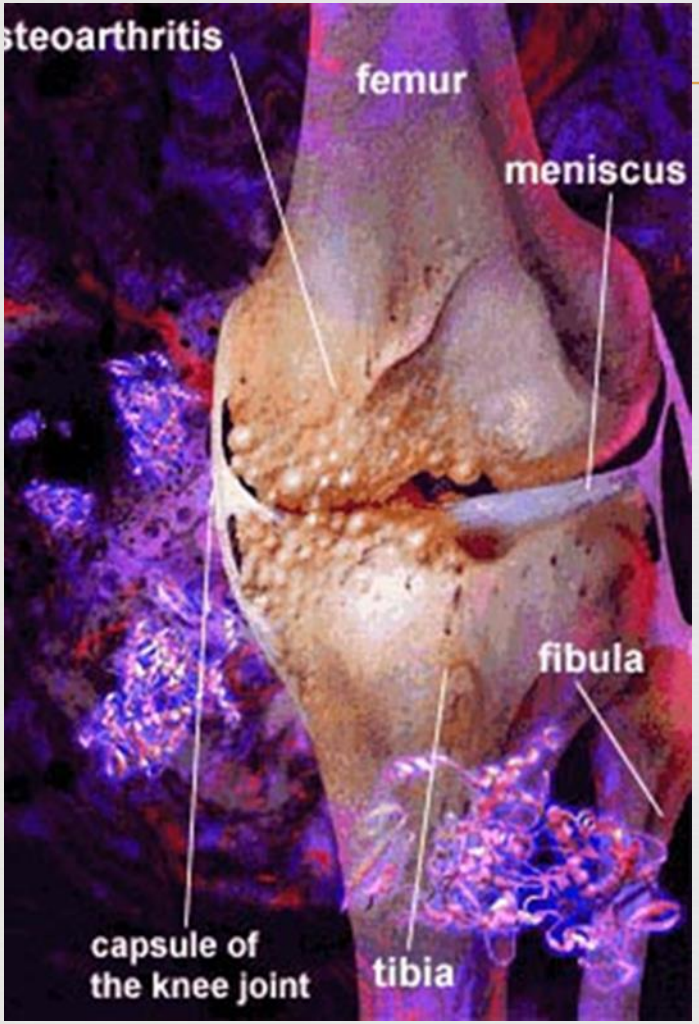
Osteoarthritis

Definition

- Osteoarthritis is the term which comprises an etiologically heterogeneous group of disorders, which have the same anatomo-pathological, radiological and biological characteristics, with similar clinical manifestations evolving due to degeneration of joint cartilage and subsequent subchondral bone changes, with consequences across the entire joint (synovial membrane, ligaments, capsule, periarticular muscles).



- In the advanced phases the articular cartilage disappears partially or totally from the overloaded areas, and consequently the bone is deformed and remodeled by sclerosis and osteophytosis, generating particular biomechanical conditions.
- Also OA is associated with obvious or discrete synovitis

OA - very old disease.

- Paleontological studies evidenced arthritic manifestations, axial and peripheral in Mummies of Dynasty XII aa 2000-1788 BC, soldiers of Alexander the Great - 300 BC
- Thomas Sydenham 1683 - Clinical description of osteoarthritis.
- Hunter 1758 - the first anatomical-pathological presentation of the degenerative process.
- 1800 - Heberden - changes of the distal interphalangeal joints that distinguish them from the gout and call them "arthritis sicca“
- 1849 Redfern reports for the first time the destructive lesions that appear in the cartilage.
- 1908 - Hoffa first describes the radiological signs of osteoarthritis.
- In 1913 F. von Muller differentiates the joint degenerative diseases from the inflammatory ones.

Epidemiology

- One of the most common diseases in the world
- The most common rheumatic disease
- The incidence increases with age.
- Maximal incidence is found at the age of 55 - 75.

Clinical features appear :

- In 0,1% of patients at 25-35 y.o.
- In 10% of patients over 65 y.o. and
- In 30% of patients over 75 y.o.;

X ray changes appear:

- In 1% of people at 25-35 y.o.,
- 30% of people over 65 y.o.
- 80% of people over 75 y.o.

Morphologic changes at the joint cartilage level are found in all the autopsies of people over 65 y.o.

Epidemiology

- there are sex related differences :
  - Before 50 years, men a more frequently affected
  - After 50 years, women suffer more of:
    - hand osteoarthritis
    - knee osteoarthritis
- Hip osteoarthritis is more frequent in men, no matter the age
- In the period 30-65 years, the disease prevalence increases in 2-10 times

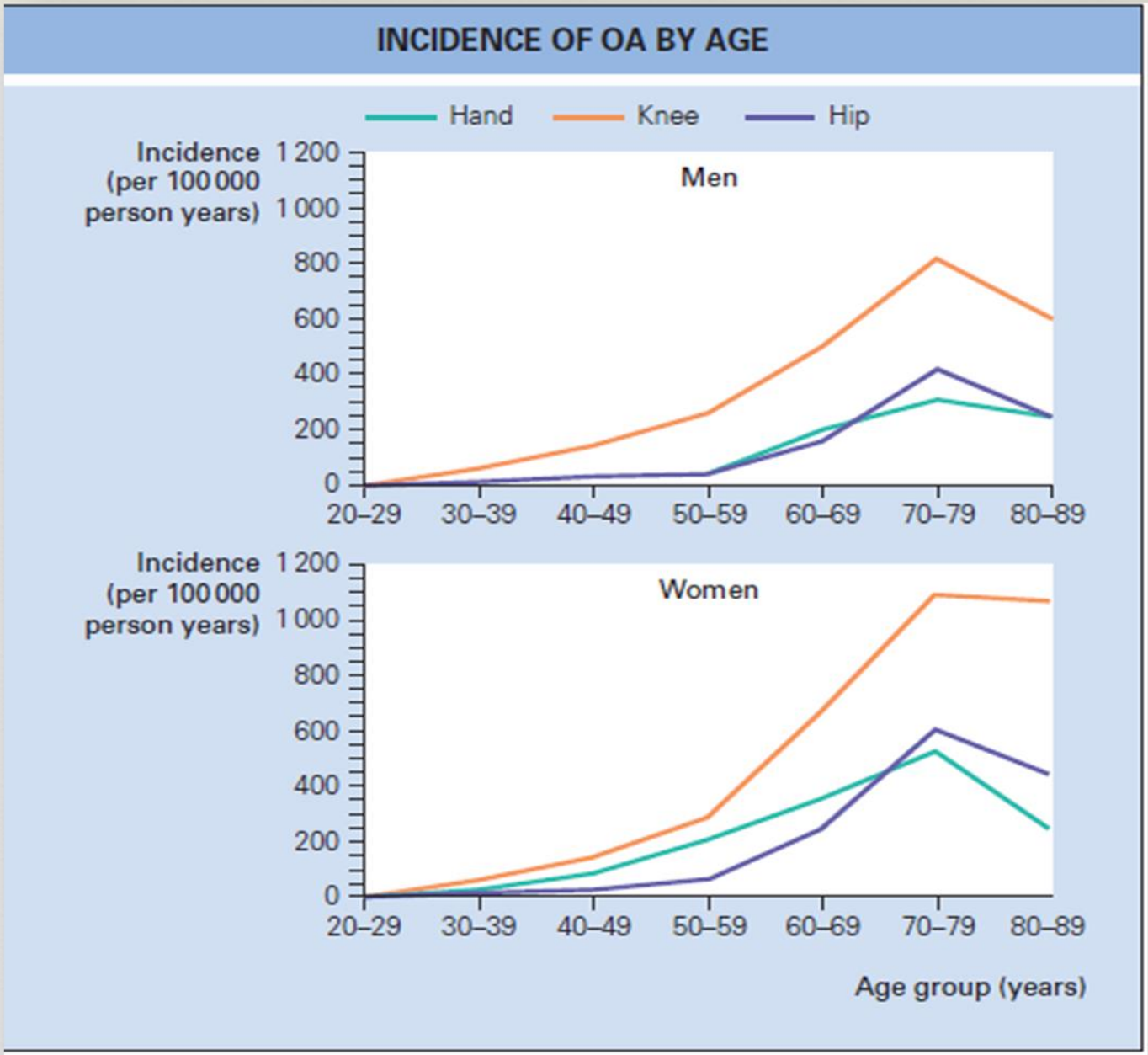
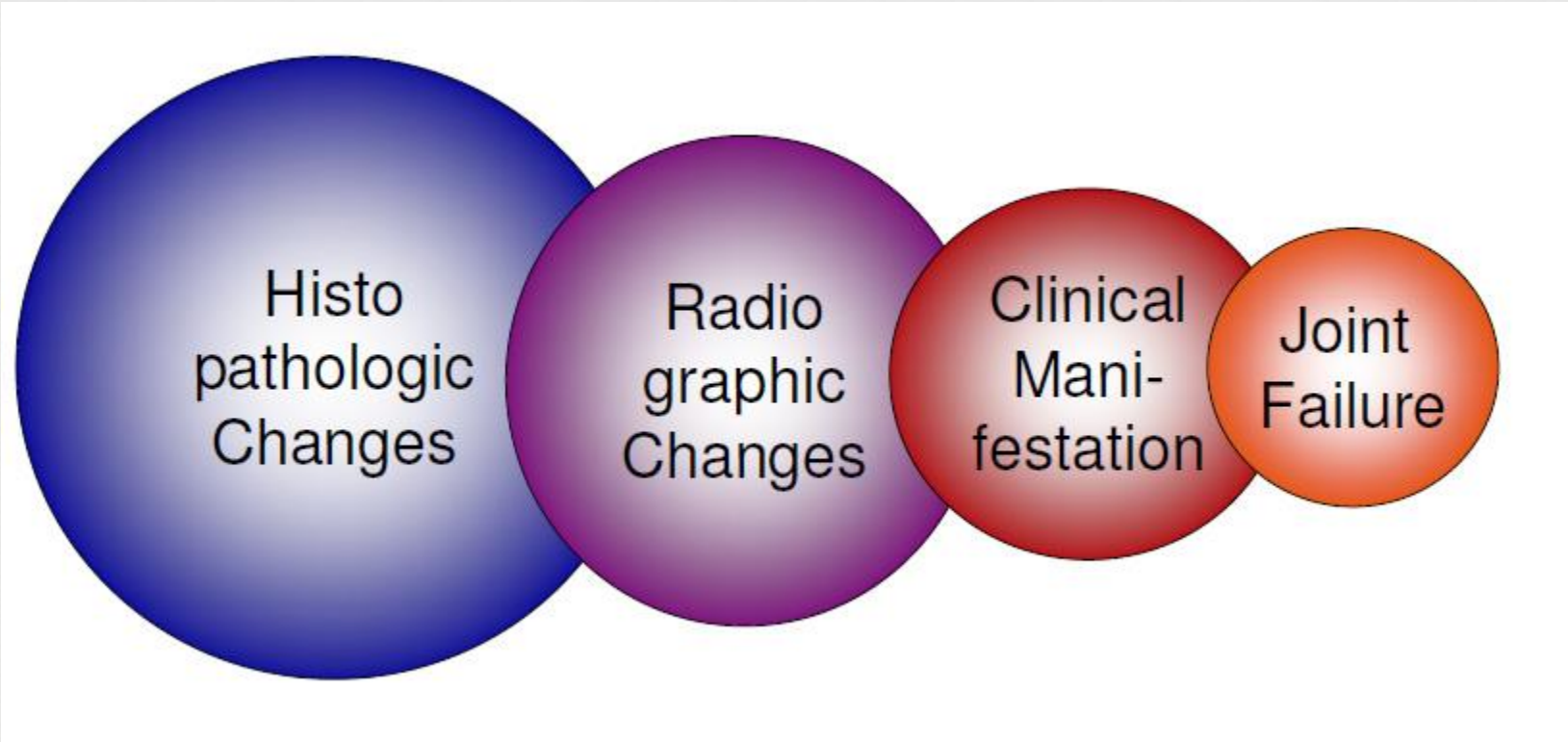


Fig. 169.3 Incidence of symptomatic osteoarthritis of the hand, knee, and hip by age. *(Data from Fallon Community Health Plan; Oliveria SA, Felson DT, Reed JI, et al. Incidence of symptomatic hand, hip, and knee osteoarthritis among patients in a health maintenance organization. Arthritis Rheum 1995;38:1134-1141.)*

Consequence of events in OA



Racial differences

- Coxarthrosis was more common in the than in the Asian (Hong Kong studies)
- Native Americans suffer more from OA
- Increased incidence of coxarthrosis and osteoarthritis of the hand in Caucasian (South Africa).

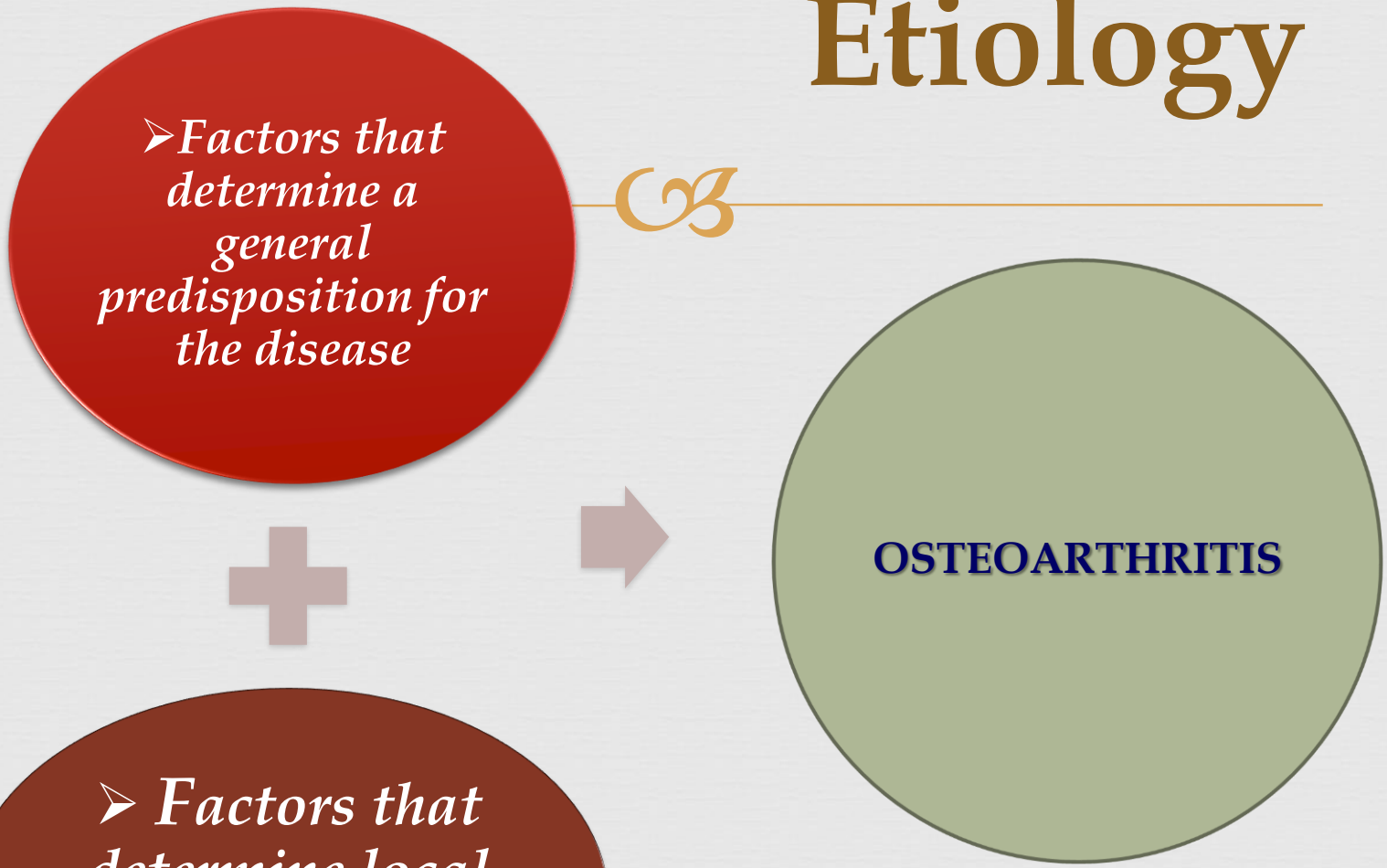
Hereditary predisposition:

- The frequency of OA among relatives of OA patients is 2 times higher,
- In people who have some defects of the locomotor system - the risk of developing OA increases by 7.7 times.

Etiology

- OA - multifactorial disease.
- **The etiology of primitive osteoarthritis is unknown.**
  1. First - Mechanical cartilage overload
- **2. Reduction of cartilage resistance (in physiological "loading" conditions).**
- **In all the forms of the disease there is a joint imbalance induced by several risk factors.**

Etiology


➢Factors that determine a general predisposition for the disease
➢ Factors that determine local biomechanical anomalies
OSTEOARTHRITIS

Osteoarthritis risk factors

A. General *: Systemic factors that determine a general predisposition for the disease*

- **Age**
- **Hereditary**
- **Nutrition**
- **Hormonal status and bone density**
- **Cartilage and bone metabolism**

B. *Local (biomechanical): that determine local biomechanical anomalies.*

- **Overweight**
- **Mechanical milieu of the joint**
- **Cartilage loading**
- **Trauma and joint deformity**
- **Professional requirements**
- **Professional sport**
- **Weakening of the muscles**

Age (prevalence of knee and hip replacement surgery in different age groups)

With aging, it occurs:

- **Reduction of aggregation of proteoglycans**
- **intensification of proteoglycan aggregation defects - the substantial reduction in cartilage damping quality**
- **additional shock absorbers - the peri-articular muscles become more incompetent.**
- **Possibly chondrocytes, with aging, as a result of injury or natural metabolism, lose the ability to accumulate or restore the cartilage matrix.**
- **the cartilage matrix in the aging process becomes more sensitive to cumulative microtrauma.**
- **Dysregulation of type II collagen - caused by repeated trauma of the cartilage (defibrillation of the collagenous carcass).**

Hereditary:

- 70% în hand osteoarthritis and 50% in hip osteoarthritis
- In erosive OA - especially - the distal and proximal interphalangeal joints - genetic determinism is noted.
- This variant is 10 times more common in women - a fact explained by the dominant autosomal transmission in women and autosomal recessive in men.
- Defect of collagen type II gene leads to degeneration of collagen type II
- vitamine D receptors, type II collagen, *insulin growth factor I*

Nutrition:

- Risk factors: hypovitaminosis C and D
- Protective factors: antioxidant nutrients

Hormonal status:

- Osteoarthritis prevalence increases after menopause
- Women with estrogen substitution therapy seem to be relatively protected

- The association of polyarticular OA with some clinical manifestations possibly influenced by endogenous sex hormones, (obesity, BMD, gynecological diseases and gynecological surgery).

Obezity

- There is a direct link between overweight and osteoarthritis of the weight-bearing joints
- It is determined as a risk factor for knee and hand OA, less directly - coxofemoral osteoarthritis.
- In people with excessive BMI, the risk of knee OA over 36 years is 1.5 times higher in men and 2.1 times higher in women.
- reduction of body weight by 5 kilograms reduces the risk of symptomatic gonarthrosis by 50%

External risk factors (local)

1. Microtraumatization:

- trauma
- Multiple stereotype movements.
- Cartilage injury in athletes and in some professions may have special locations (tennis, baseball, ballerina and others).

2. Pre-existing diseases of the locomotor system, surgery

Pre-existing diseases of the locomotor system, surgeryex. meniscectomie)

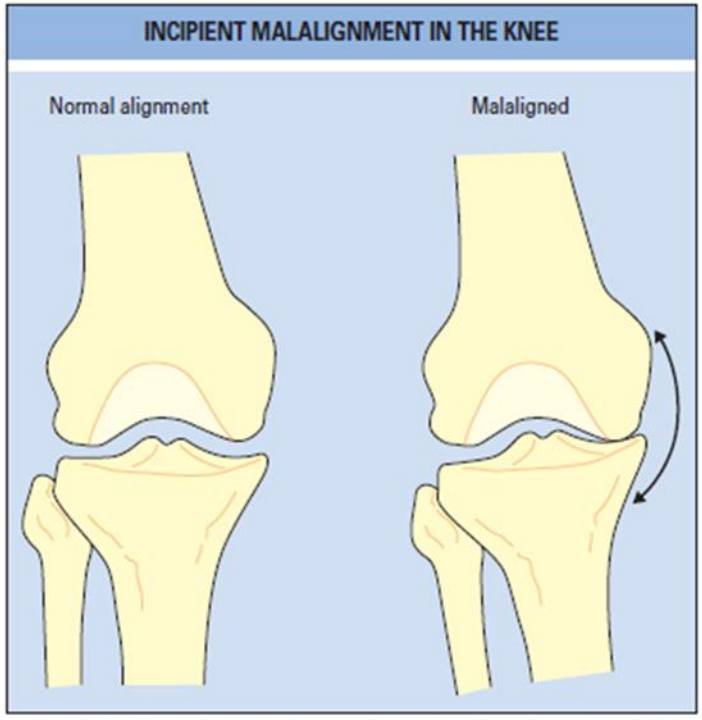


Fig. 170.2 Incipient malalignment in the knee. The left side is normal with loading occurring physiologically. With early medial disease there is a tilting of the joint so that focal load (stress) increases medially (depicted by line with two arrows), leading to further focal medial load. A vicious cycle of increased medial joint damage, more tilting, and further increased medial stress develops.

- Static and joint congruence disorders, congenital anomalies (dysplasia and subluxations in newborns uncorrected, unrecognized - up to 80% of coxarthrosis after some authors).

Joint inflammatory processes

- **The detection of Ig - s and complement that attaches to the surface of the articular cartilage proves the ability of the antigenic components of the degraded cartilage to cause inflammation.**
- **OA may also be a consequence of other rheumatic diseases with inflammatory mechanism.**

Metabolic diseases

- Hemochromatosis
- Alcaptonuria
- Konovalov - Wilsson

Pathogenesis

1). *Progressive degradation of the joint cartilage;*

2). *Subchondral bone reparation.*

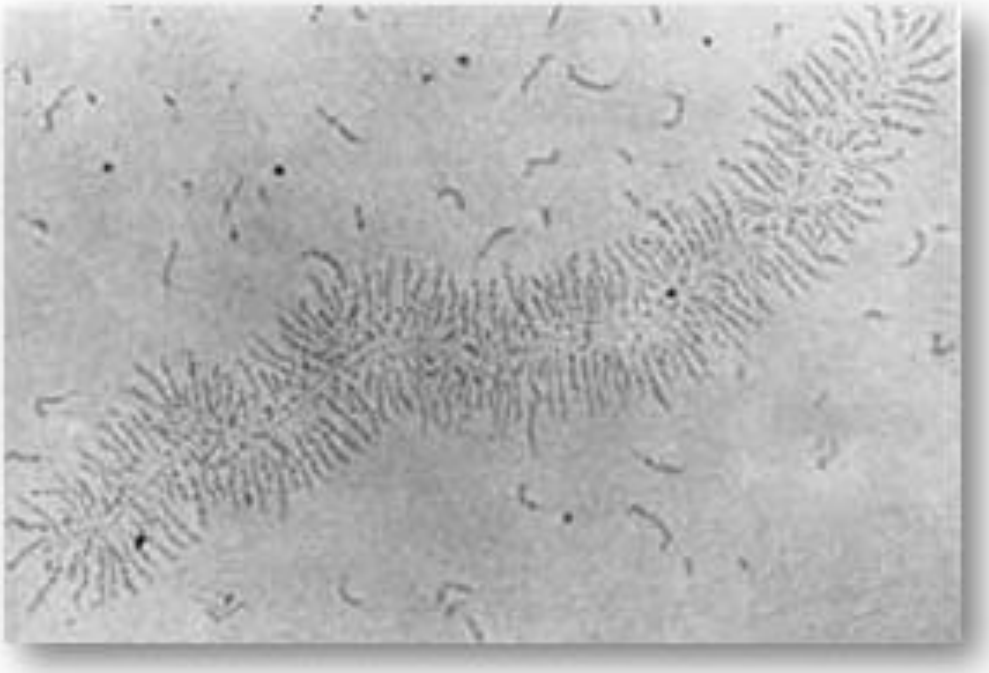
The process begins at the level of the hyaline cartilage, in the superficial layer, extends to the whole cartilage and to the subhondral bone and damages the other joint components.

The structure of articular cartilage matrix - predominantly 2 types of macromolecules:

1. Proteoglycans (90% aggrecan) - synthesized by chondrocyte, provides compression resistance.

   Agrecan consists of chains of chondroitin sulfate and keratan sulfate proteoglycans, molecules closely related to hyaluronic acid.

2. Collagen fibers (predominantly type II) – three - dimensional insoluble network - provides resistance to extension and displacement.



Actual electron photomicrograph of a cartilage molecule (proteoglycan). Note the protein backbone and the sugar sidechains sticking out to the side.

Cartilage

Matrix

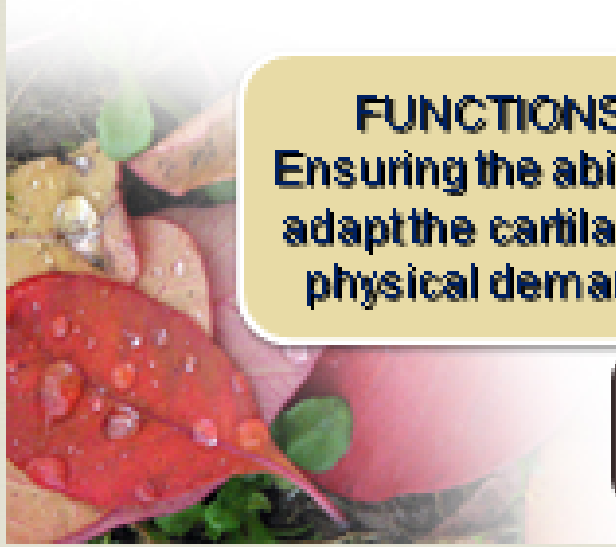
composition:

Collagen molecules of various types. Proteoglycans (90% aggregate)

FUNCTIONS: Ensuring the ability to adapt the cartilage to physical demands

chondrocytes

- **Regulation of cartilage tissue metabolism and degradation of cartilage matrix components**
- **Synthesis of proteoglycans and collagen**
- **Synthesis of substances that destroy proteoglycans and collagen**

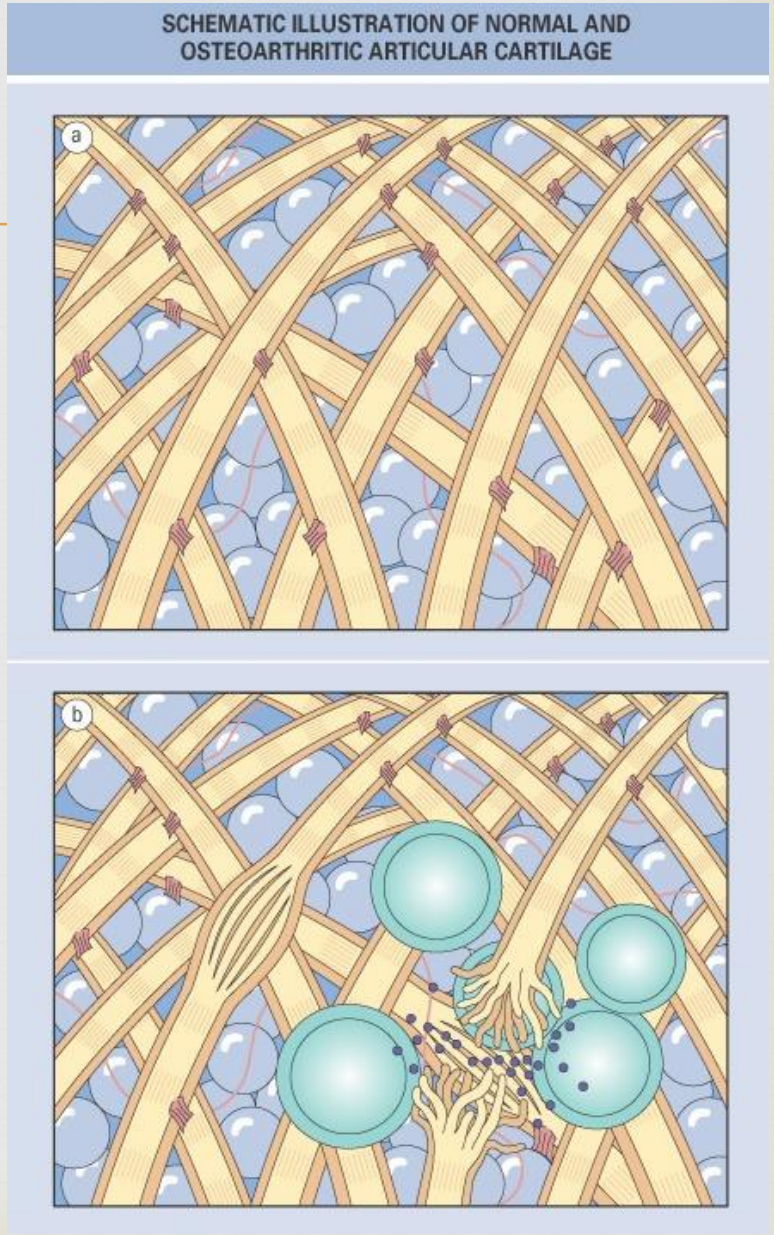


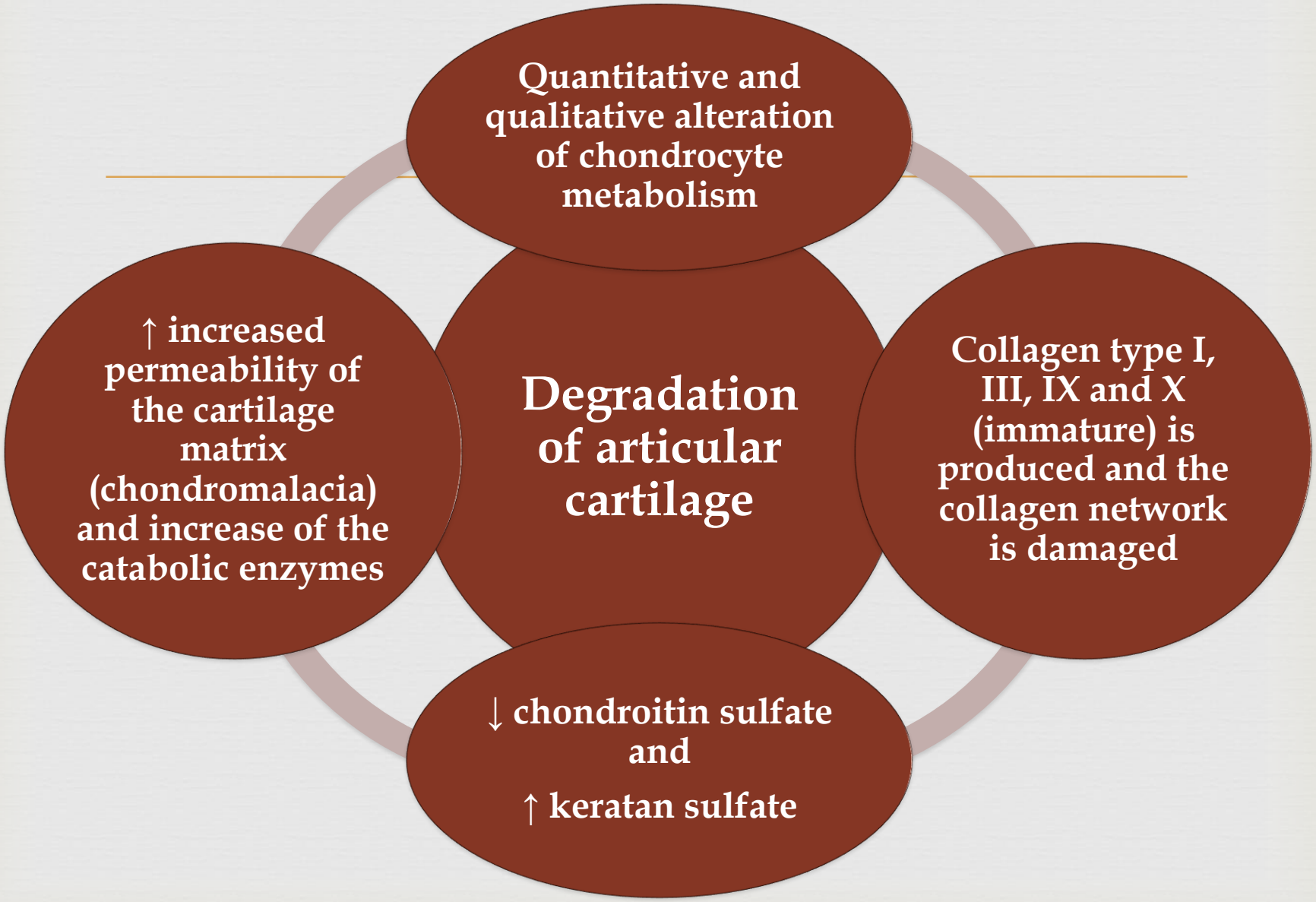
Cartilage structure and function

Cartilage performs two basic functions:

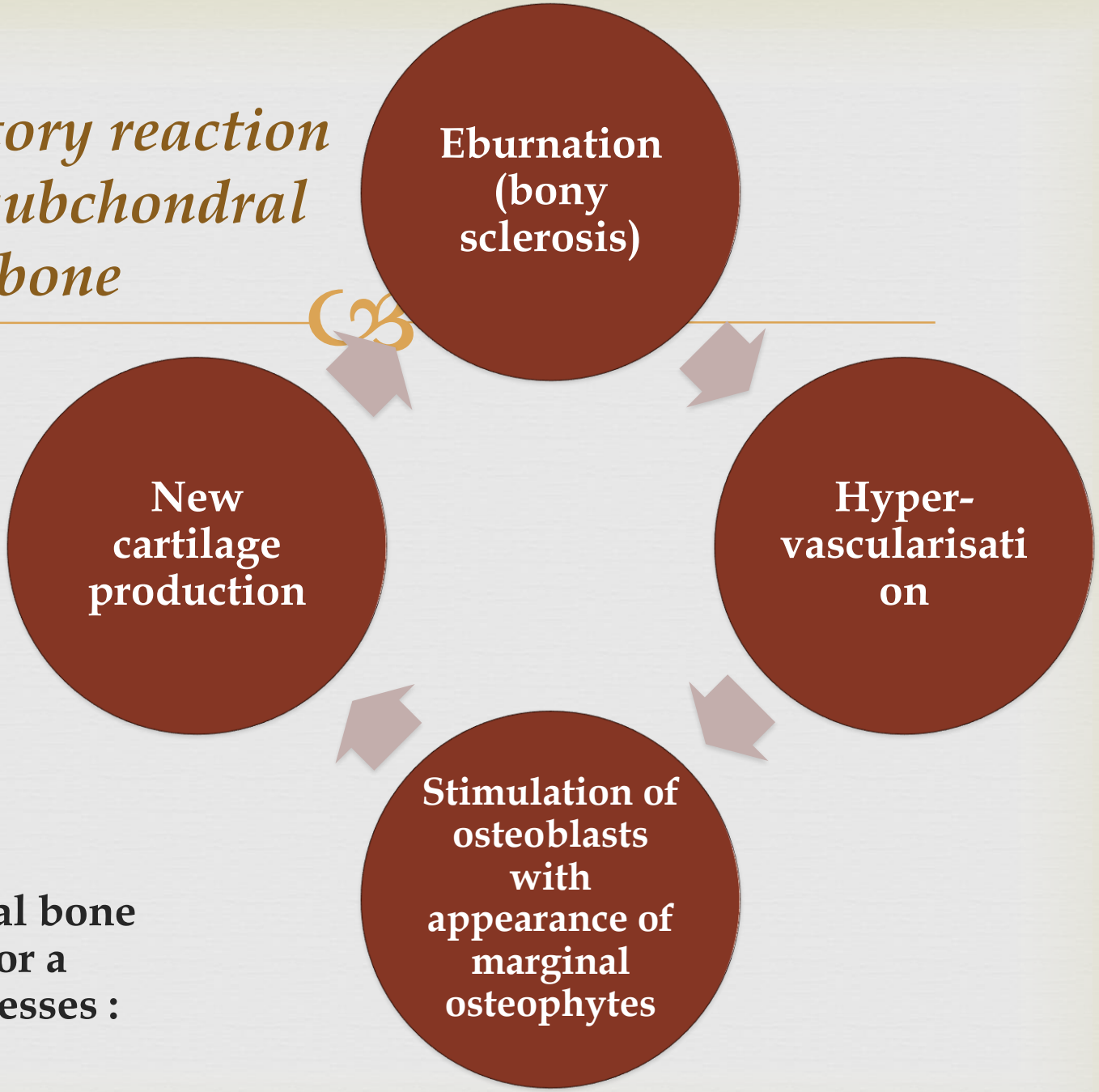
- 1. he has a very smooth surface and ensures the sliding of the articular surfaces when moving
- 2. cartilage ensures uniform distribution of the load on the joint surfaces and protects them from destruction.
- With age, fragmentation of binding proteins, instability of cellular aggregates and their reduction, modification of chondroitin sulfate chains, and growth of keratin sulfate chains occur, which seems to be favorable for the development of osteoarthritis.

- As consequence loss of quality and functions of cartilage appear: elastic / damping and lubrication capacities.
- Thus, loads are transmitted directly and intensely to the subchondral bone, which responds adaptively.




Quantitative and qualitative alteration of chondrocyte metabolism
↑ increased permeability of the cartilage matrix (chondromalacia) and increase of the catabolic enzymes
Degradation of articular cartilage
Collagen type I, III, IX and X (immature) is produced and the collagen network is damaged
↓ chondroitin sulfate and
↑ keratan sulfate

Reparatory reaction of the subchondral bone



- The subchondral bone is responsible for a number of processes :

SCHEMATIC VIEW OF THE MAIN STRUCTURES OF A HEALTHY (LEFT) AND DEGENERATE OA (RIGHT) JOINT

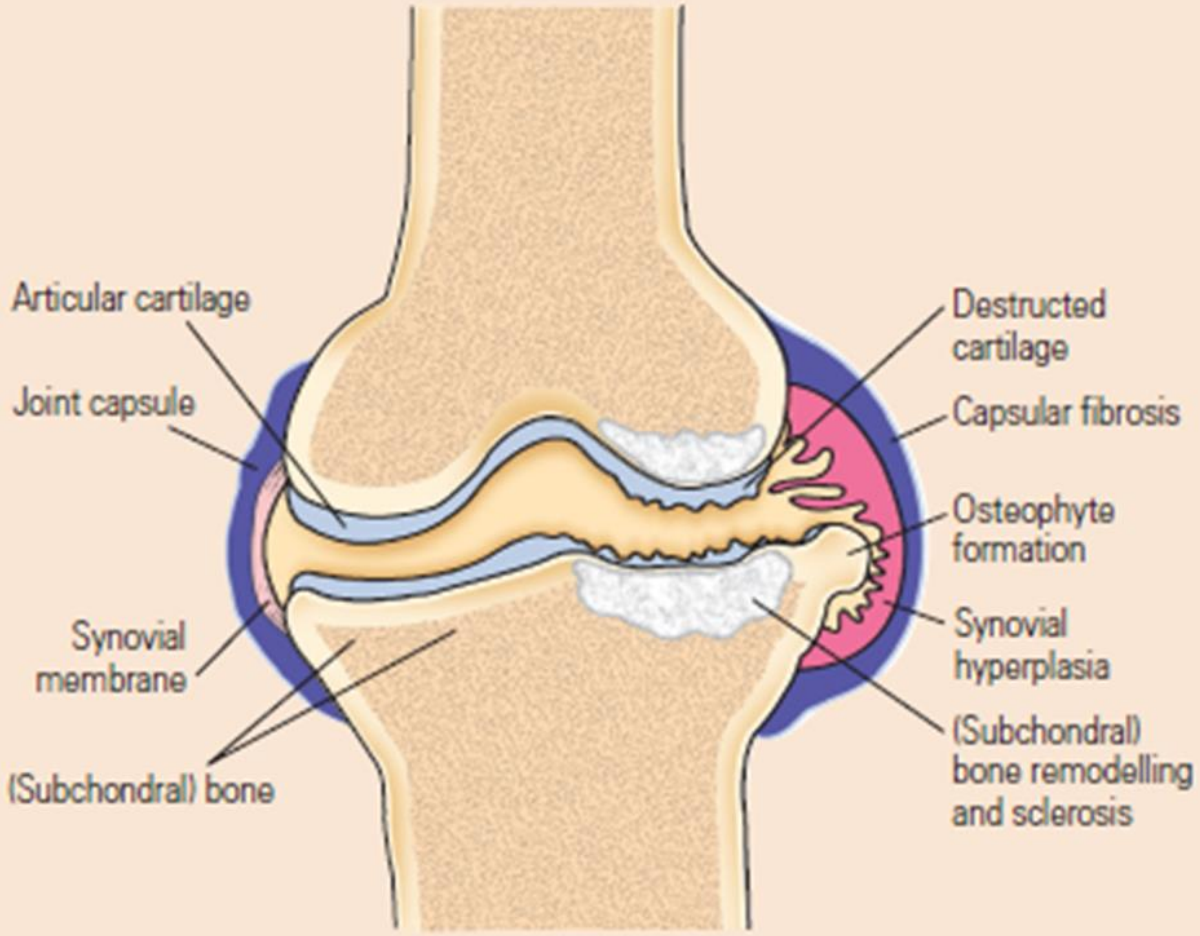
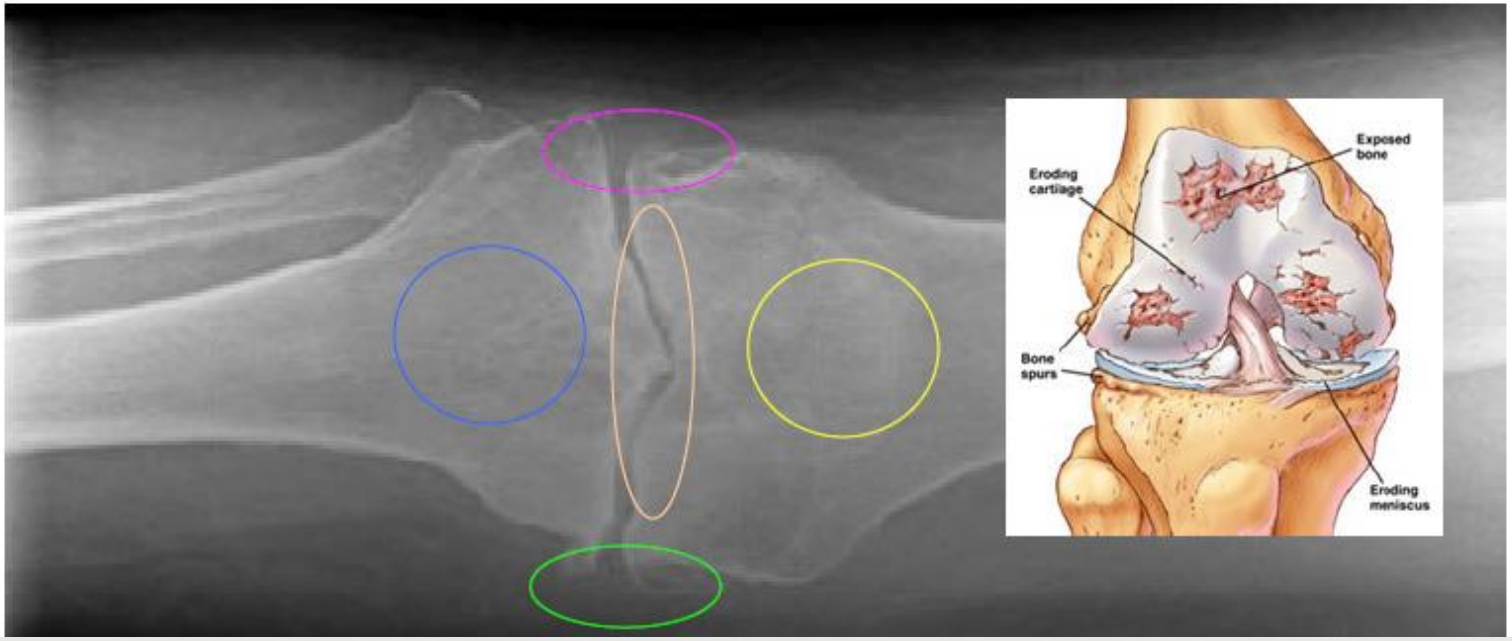


Fig. 173.1 Schematic view of the main structures of a healthy (left) and degenerated OA (right) joint. In OA the articular cartilage is lost or severely thinned, the (subchondral) bone is sclerotic, the joint capsule is thickened, and the synovial membrane is activated. *(Courtesy of E. Bartnik, Frankfurt.)*

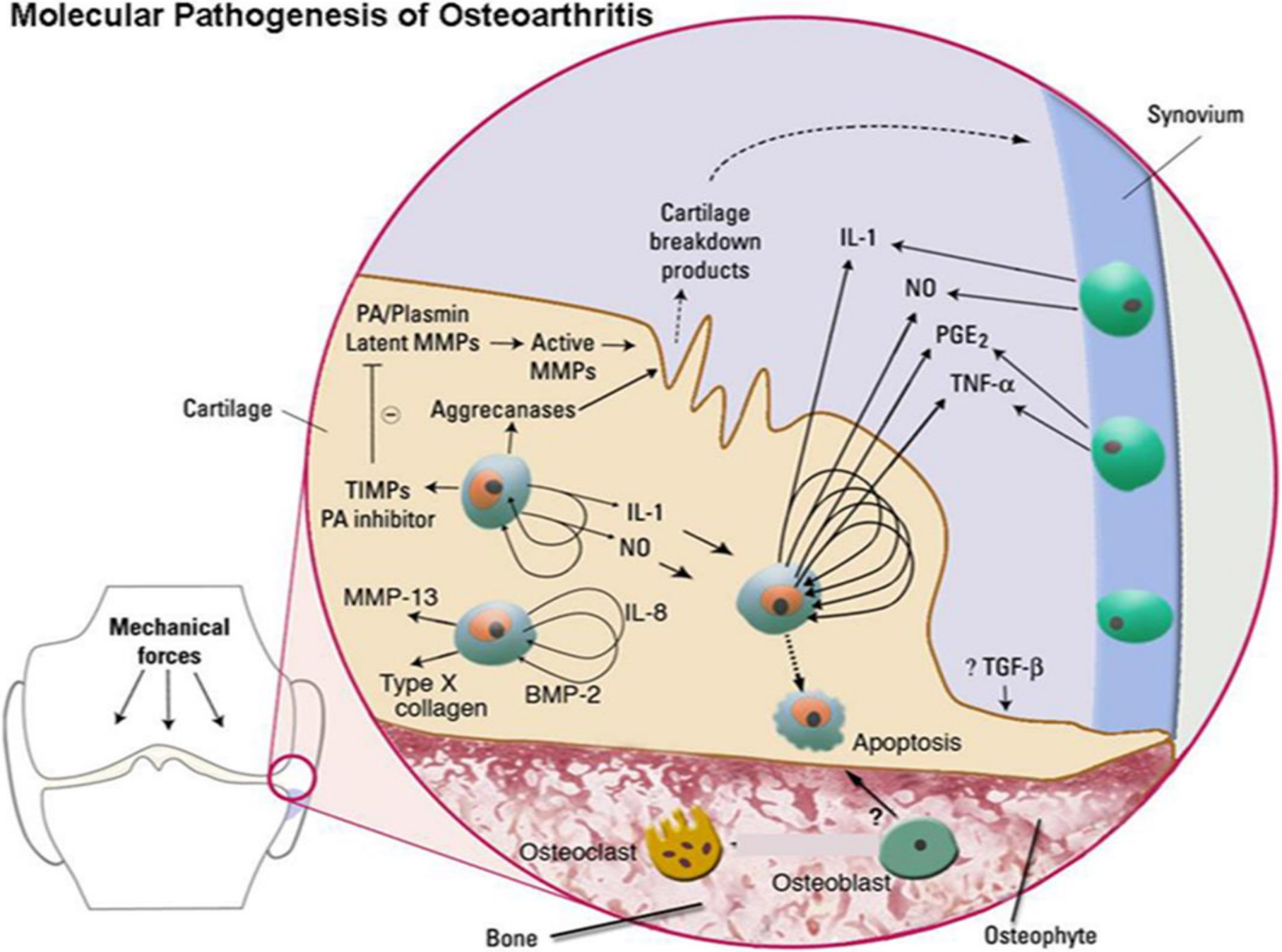
- The synovia also does not passively attend to the a mentioned alterations, but participates in an inflammatory reaction that is initiated and maintained by the particles and chemical elements resulting from cartilage degradation processes.
- And the other capsular, ligamentous, meniscal, periarticular musculature and even nerve endings are damaged by osteoarthritis.

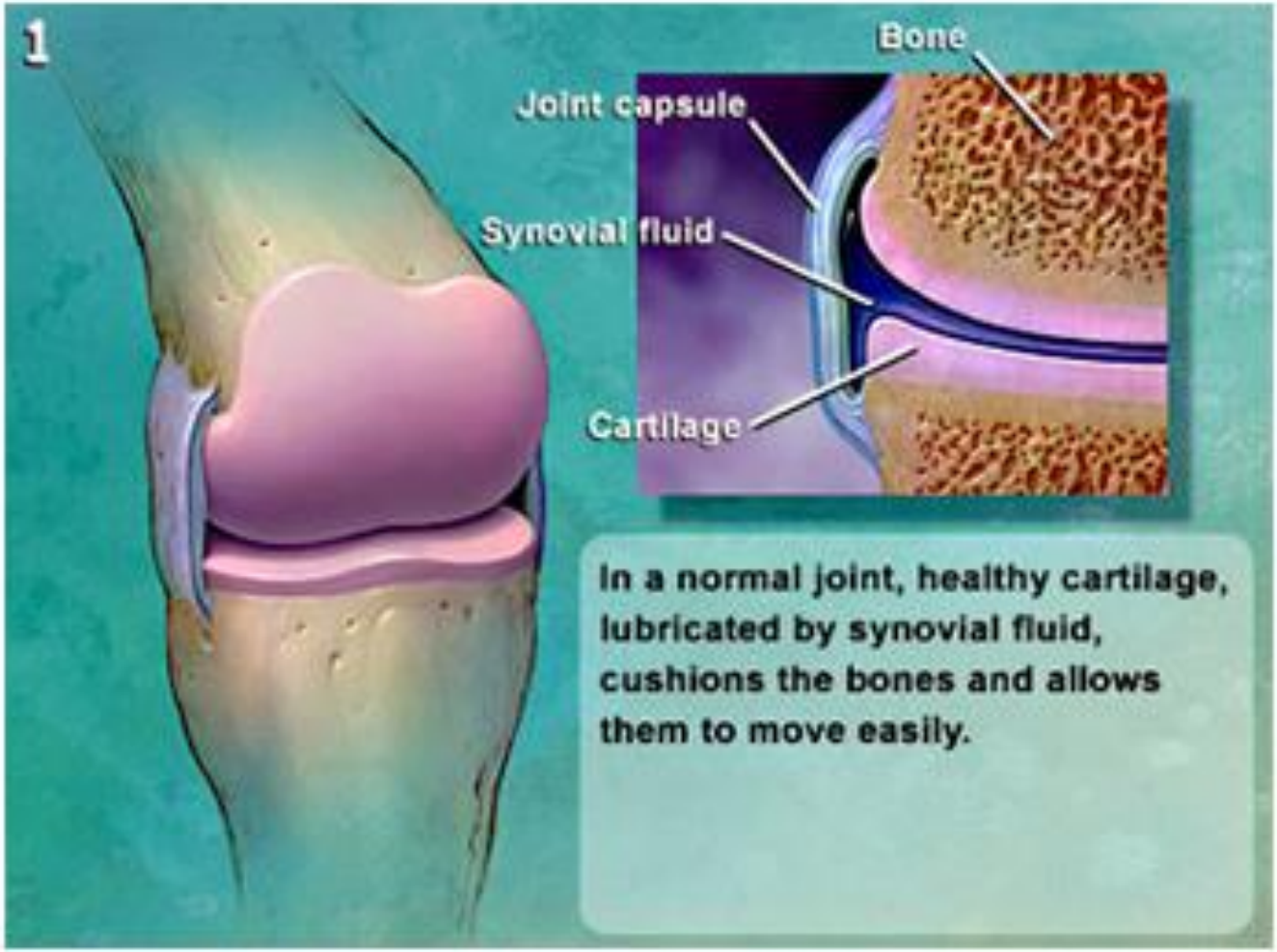
Pathomechaism of OA

Osteoarthritis =
Cartilage Damage + Synovitis +
Osteosclerosis + Osteophyte growth +
Bone Marrow Edema

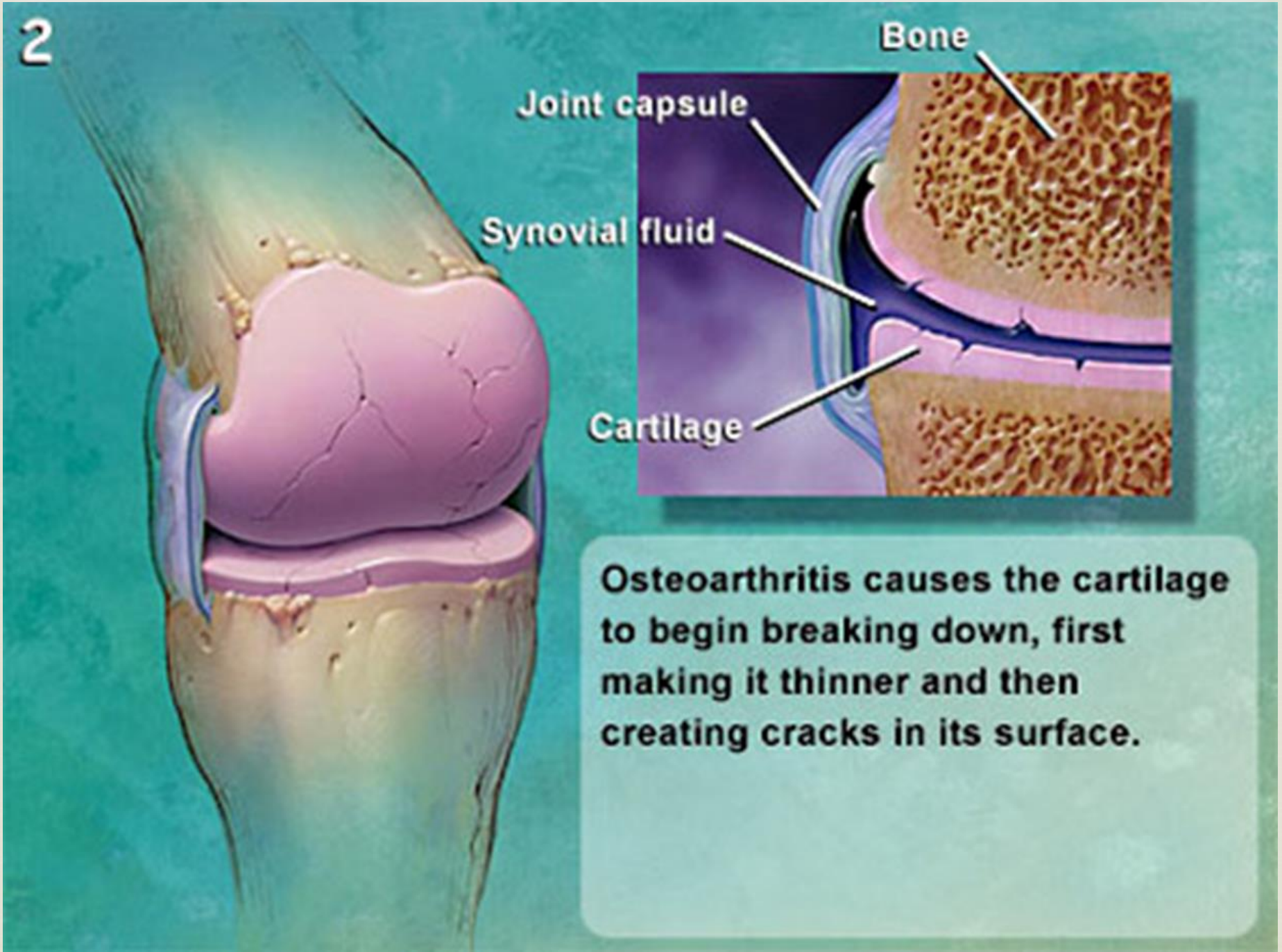


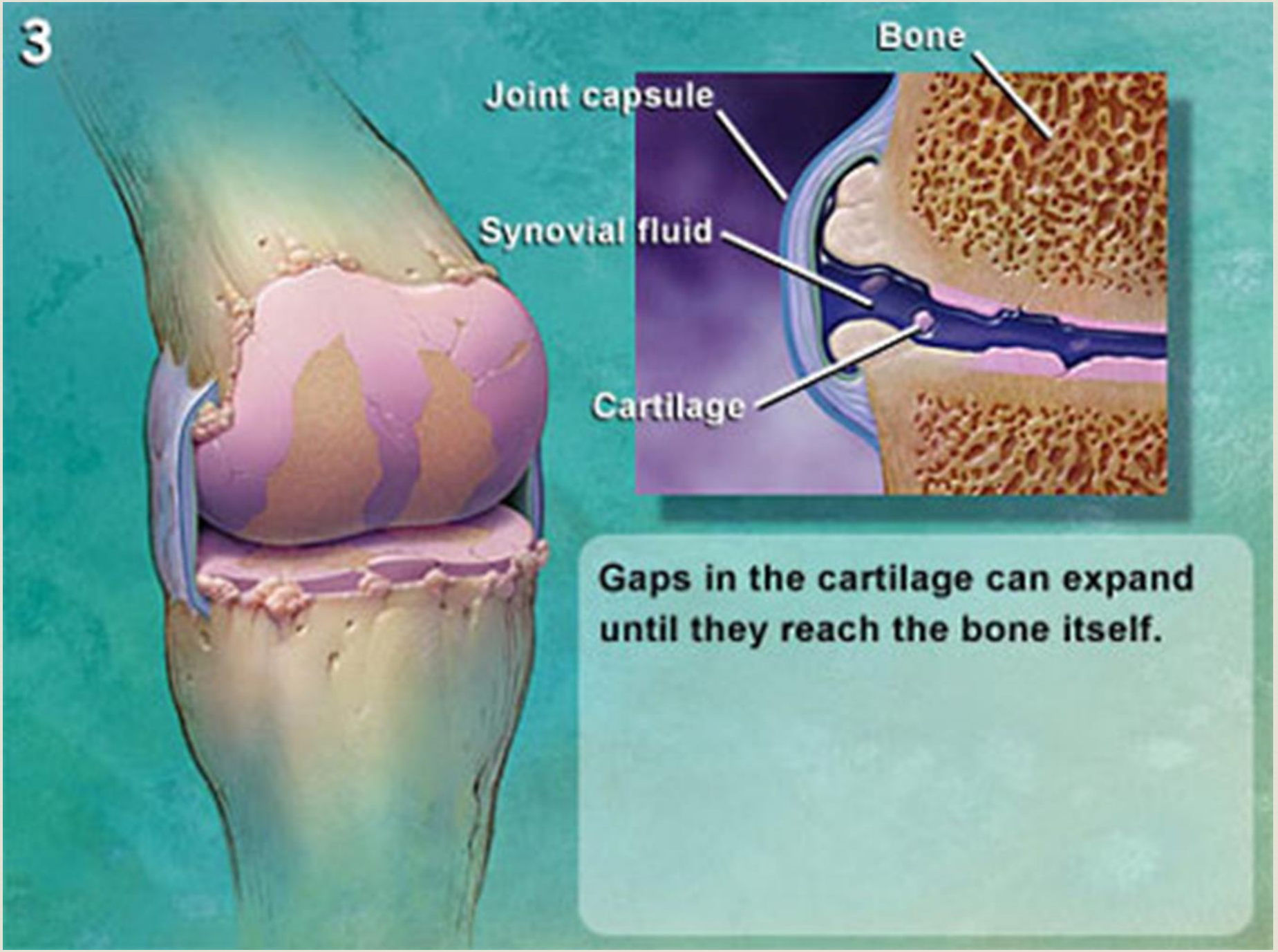
Molecular Pathogenesis of Osteoarthritis

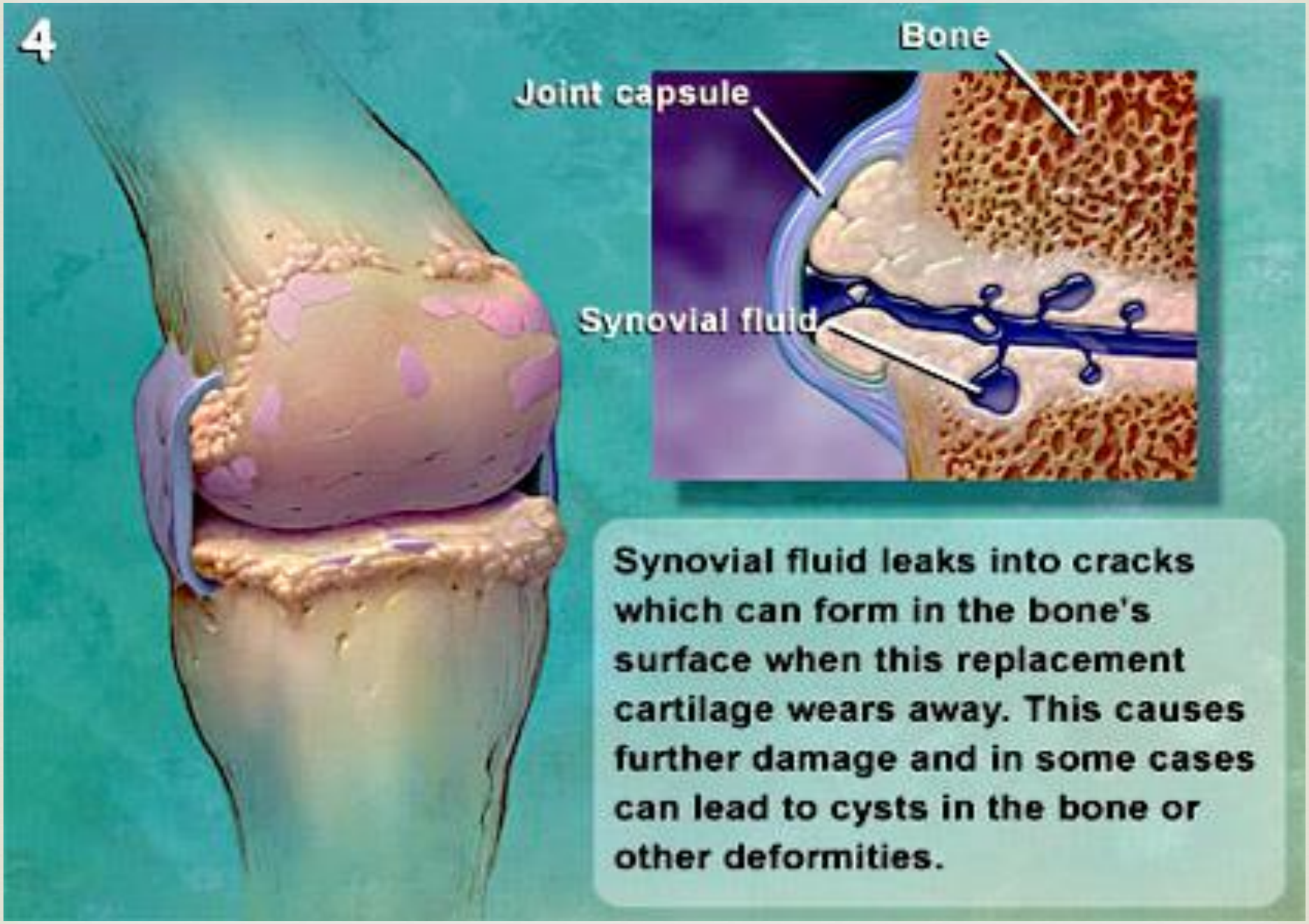


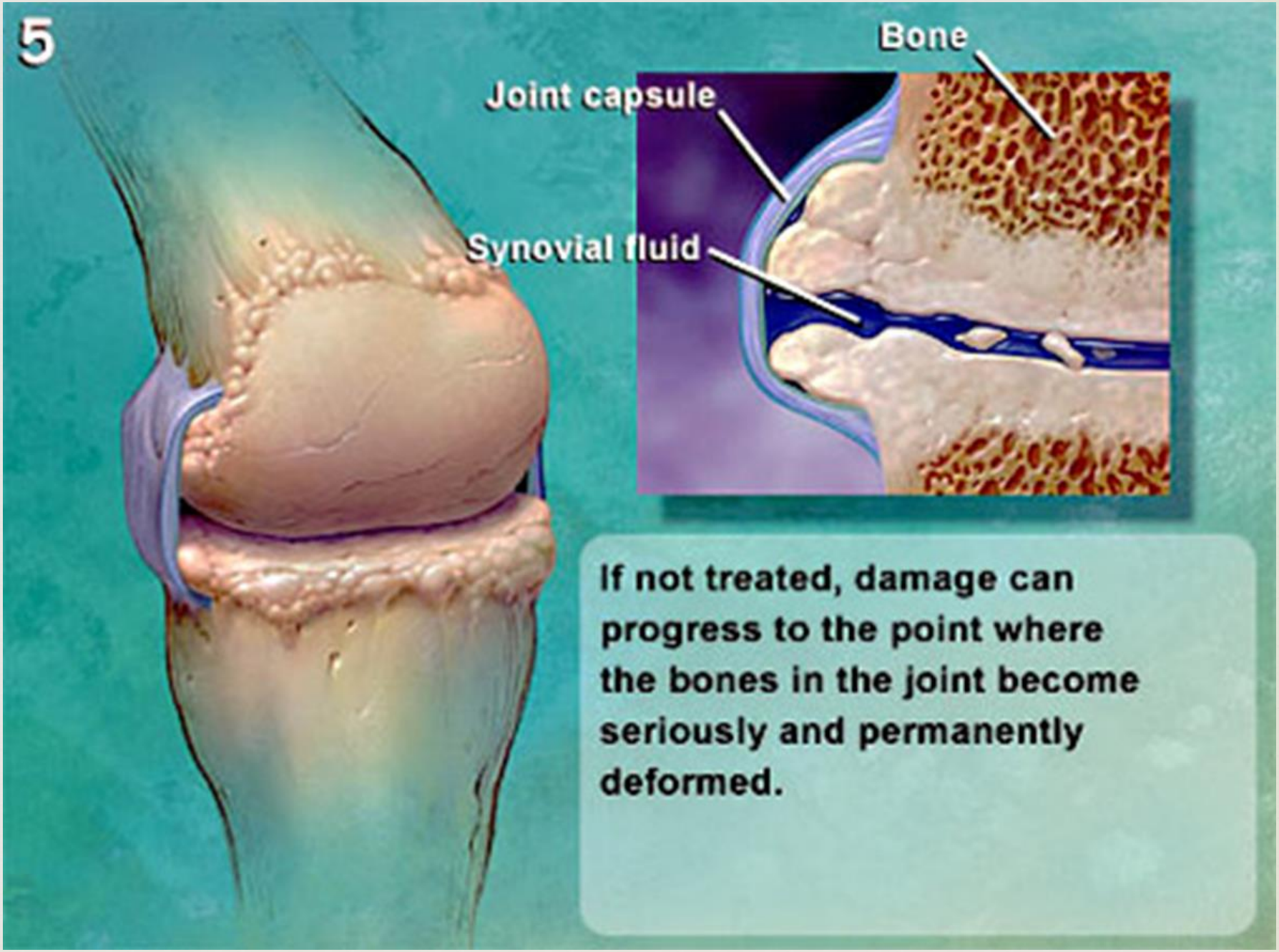


At the earliest stages of osteoarthritis, your joints look like this:


2
Bone
Joint capsule
Synovial fluid
Cartilage
Osteoarthritis causes the cartilage to begin breaking down, first making it thinner and then creating cracks in its surface.


3
Bone
Joint capsule
Synovial fluid
Cartilage
Gaps in the cartilage can expand until they reach the bone itself.


4
Bone
Joint capsule
Synovial fluid
Synovial fluid leaks into cracks which can form in the bone's surface when this replacement cartilage wears away. This causes further damage and in some cases can lead to cysts in the bone or other deformities.


5
Bone
Joint capsule
Synovial fluid
If not treated, damage can progress to the point where the bones in the joint become seriously and permanently deformed.

Classification:

Primary or idiopathic osteoarthritis

- the majority of cases, is associated with age or genetic factors, is generally pluriarticular

Osteoarthritis secondary to other diseases

it is not related to age and manifests itself rapidly clinically.

This type of osteoarthritis is mono - or oligoarticular

Primary OA (idiopathic)

A. Local

- of the hand
- of the leg joint
- osteoarthritis
- osteoarthritis
- spine
- other joints

B. Generalized OA (3 groups of joints and more).

- affecting the distal and proximal interphalangeal joints
- affecting large joints.
- erosive

Secondary OA

A. Posttraumatic

B. Congenital, acquired, or endemic conditions (Pertes disease, hypermobility syndrome)

C. Metabolic diseases:

- **ochronosis**
- **Goche disease**
- **Wilsson's disease**
- **hemochromatosis**
- **D. endocrinopathies**
- **acromegaly**
- **Hypo - and hyperparathyroidism**
- **DZ**

E. Microcrystalline diseases

F. Neuropathies (Shark)

G. Other diseases (Pagett, AR, aseptic necrosis)

Clinical picture

- **Symptoms**

From the point of view of complaints osteoarthritis has four major symptoms, pain being the dominating one.

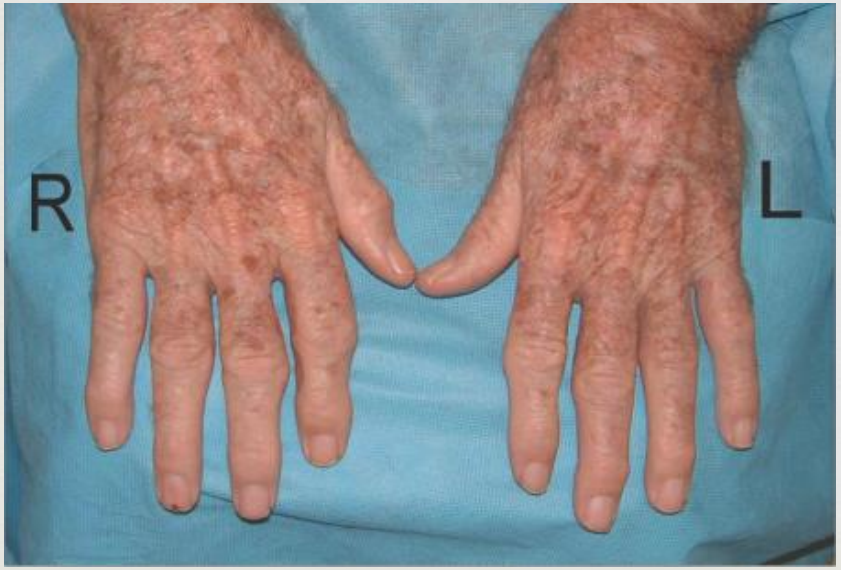
Osteoarthritis symptoms

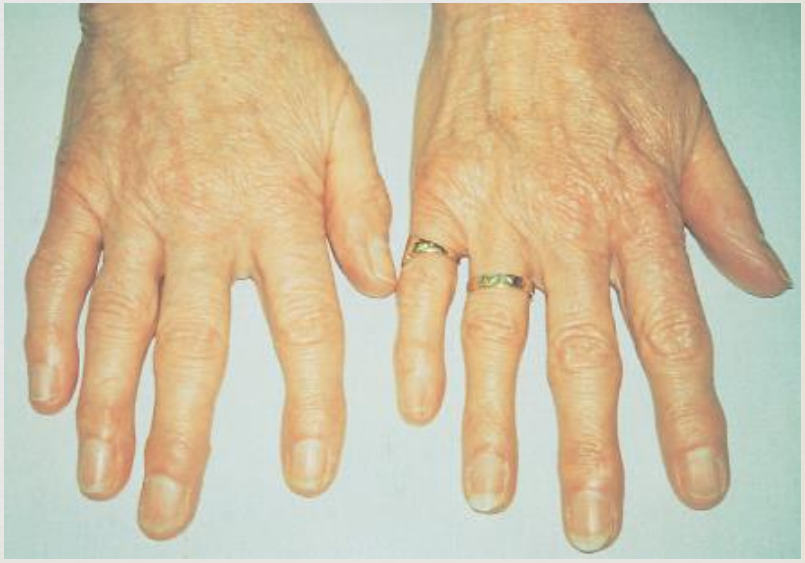
- Pain
- Rest stiffness (gelling), cracls
- Limited ROM (range of motion)
- Instability sensation

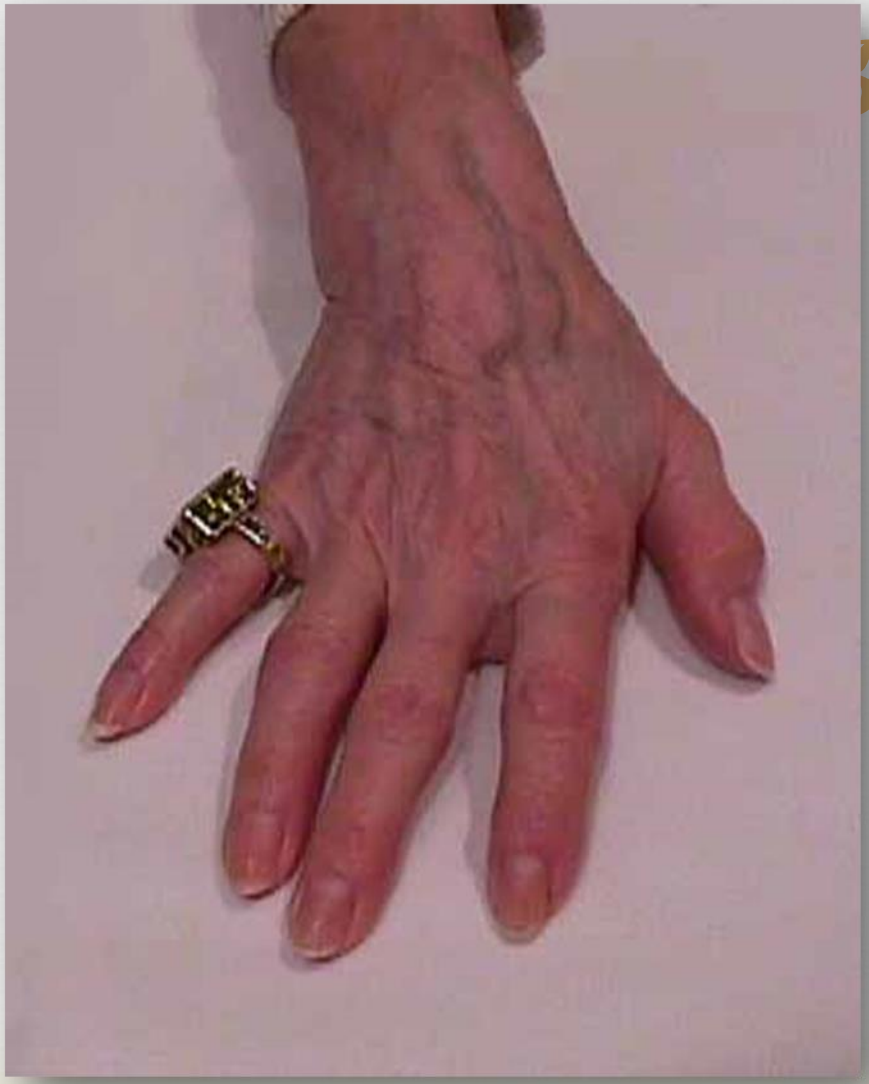
- Osteoarthritic pain - is the prototype of mechanical pain.
- In the initial stages it is a passenger , exacerbated during physical exercise .
- Rest improves it.
- In the advanced stages the pain becomes constant.
- Metheosensitivity

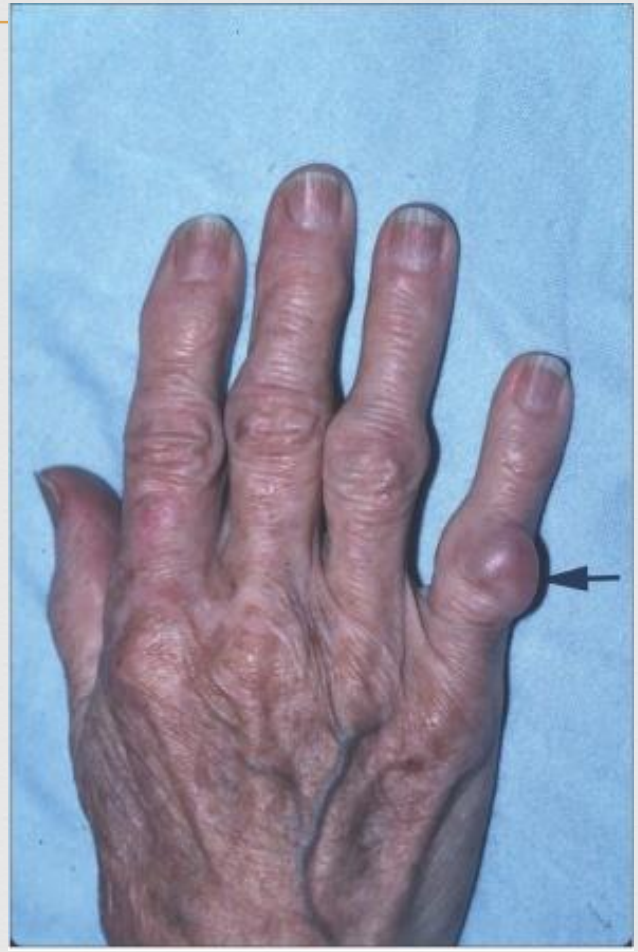
Symptoms of osteoarthritis are:

- Tenderness on the joint line, hard-elastic ("bony") deformity due to hypertrophy of the bony edges and marginal osteophytes (i.e., Heberden's and Bouchard's nodules in the interphalangeal osteoarthritis)









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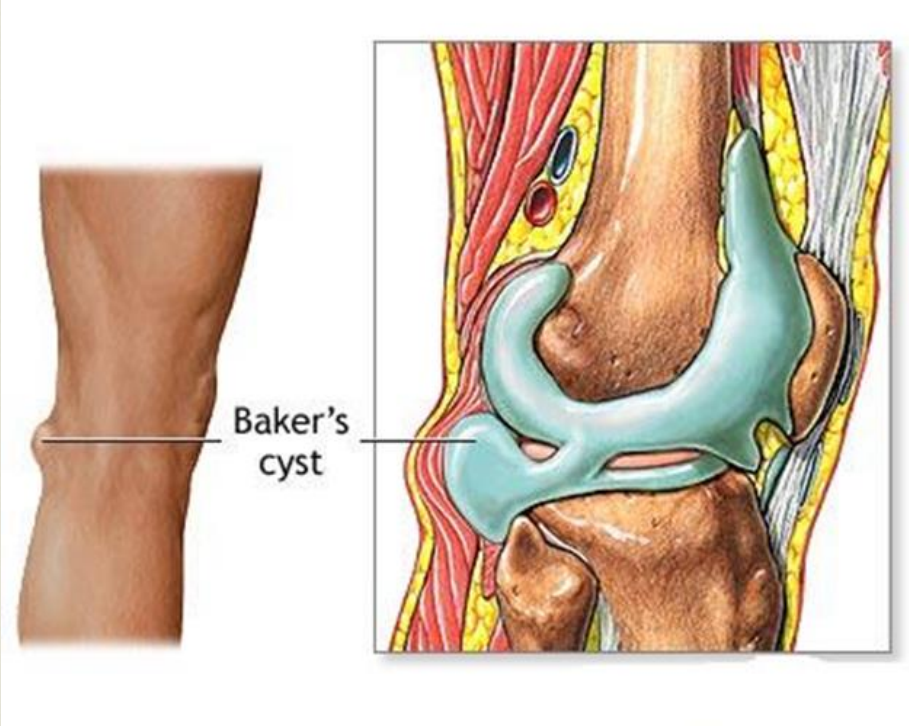
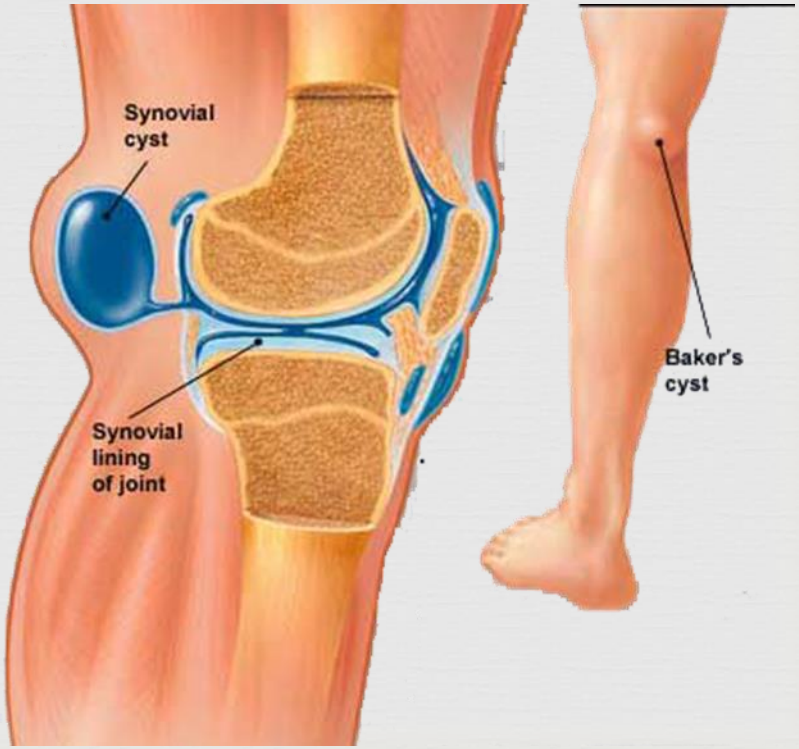

Baker's
cyst


Synovial
cyst
Synovial
lining
of joint
Baker's
cyst

- **Crepitation** is produced by the friction of irregular cartilage or denuded joint surfaces, evidenced by palpation, but also audible during passive or active mobilization
- **Limitation of joint mobility**
- **Blocking movement** by the residual ("joint mice") fragments of meniscus with synovial chondrophyte or chondroid metaplasia interposed between the joint surfaces
- **Misalignment** due to remodeling and bone destruction and loosening of the capsule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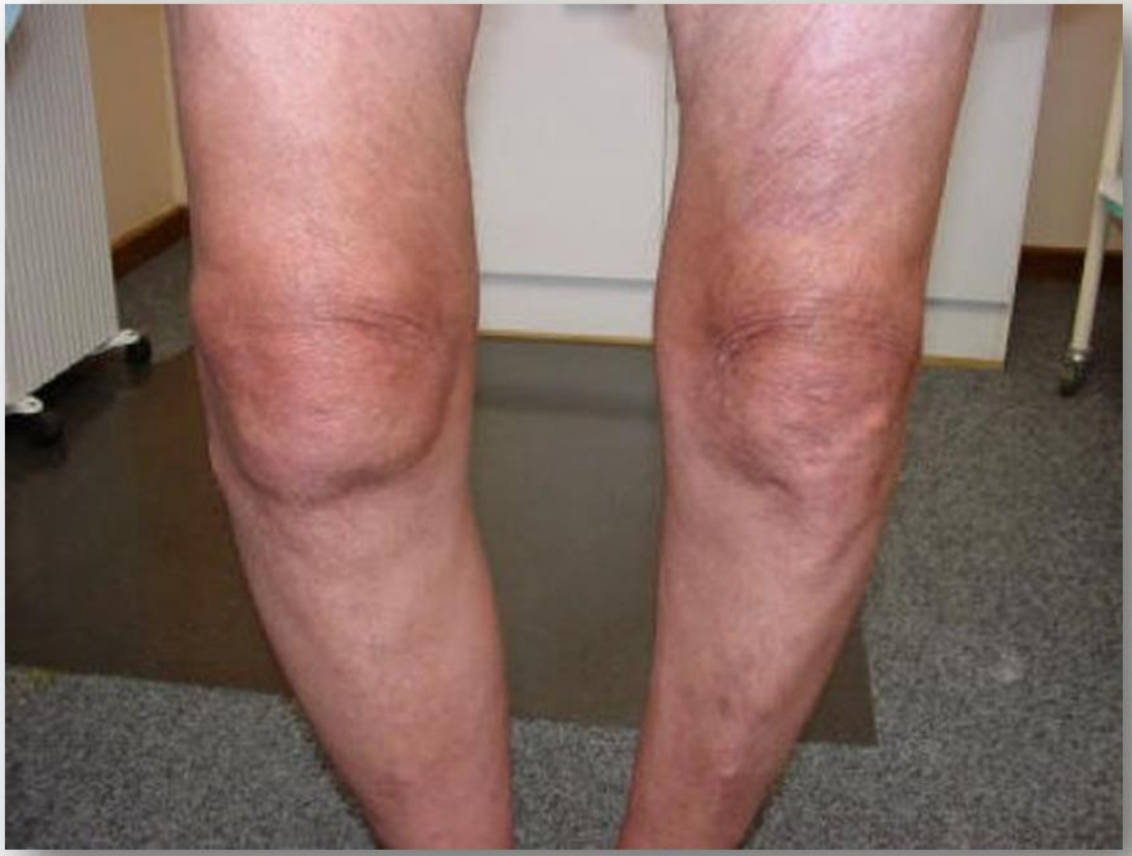


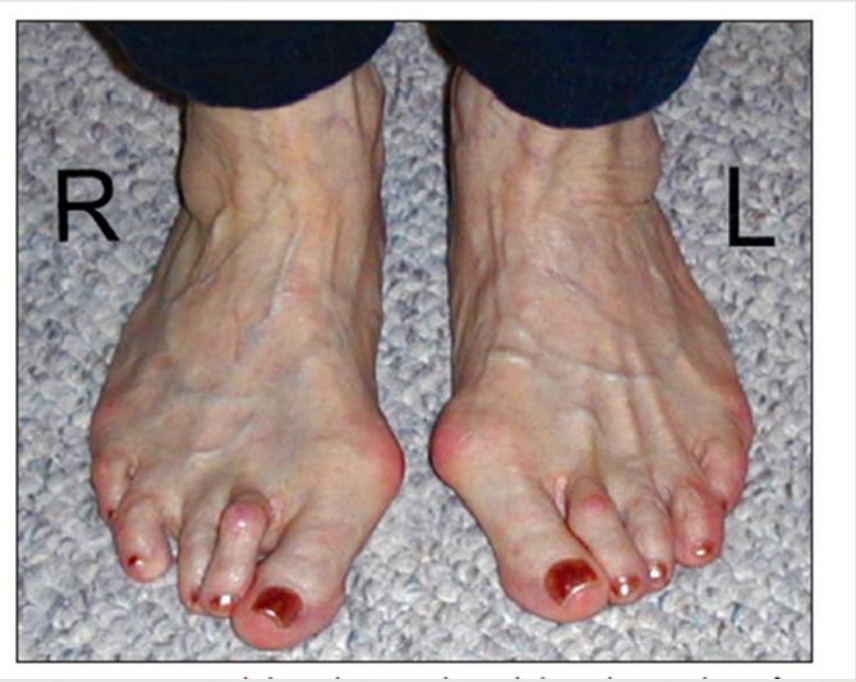
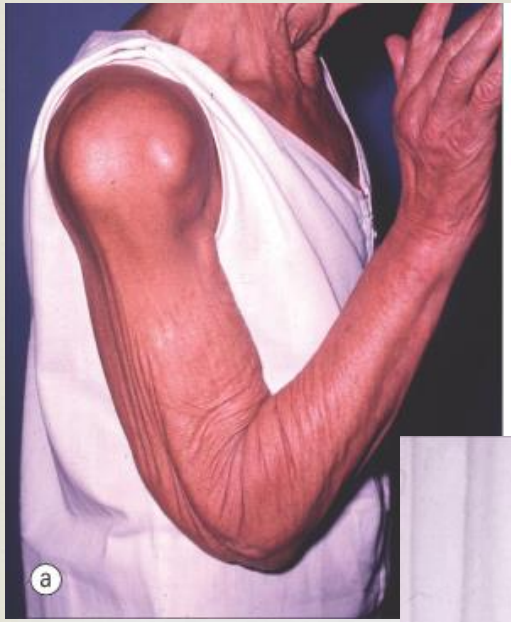


Fig. 171.6 Metatarsophalangeal (MTP) and interphalangeal osteoarthritis of the feet. There is bilateral enlargement of the first MTP joint, with medial subluxation of the phalanx. Flexion contraction of toes (particularly of the right second digit), with subluxation of the MTP, has caused an associated callus over the dorsum of the proximal interphalangeal joint. The right third digit is subluxed under the second digit. There is relaxation of the transtarsal ligament, with medial subluxation of the MTP and bony enlargement of the fifth MTP joints (tailor bunion, bunionette).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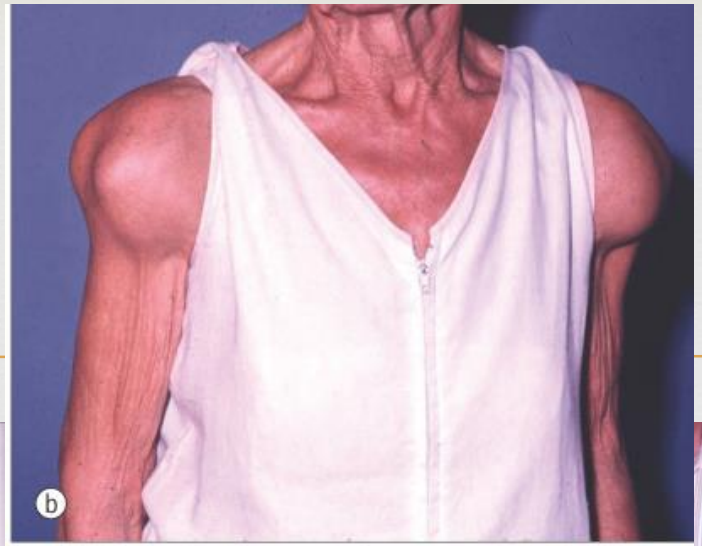
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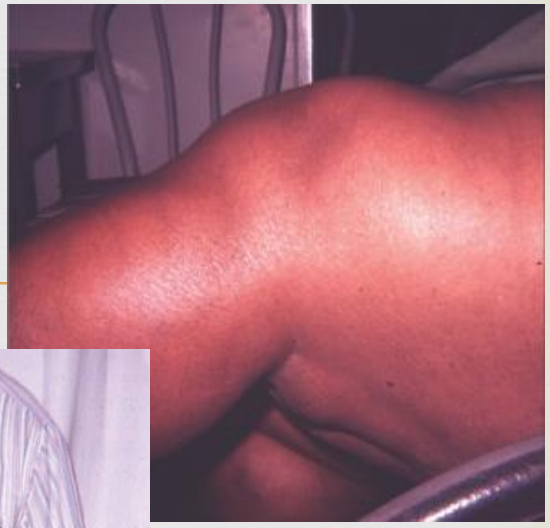

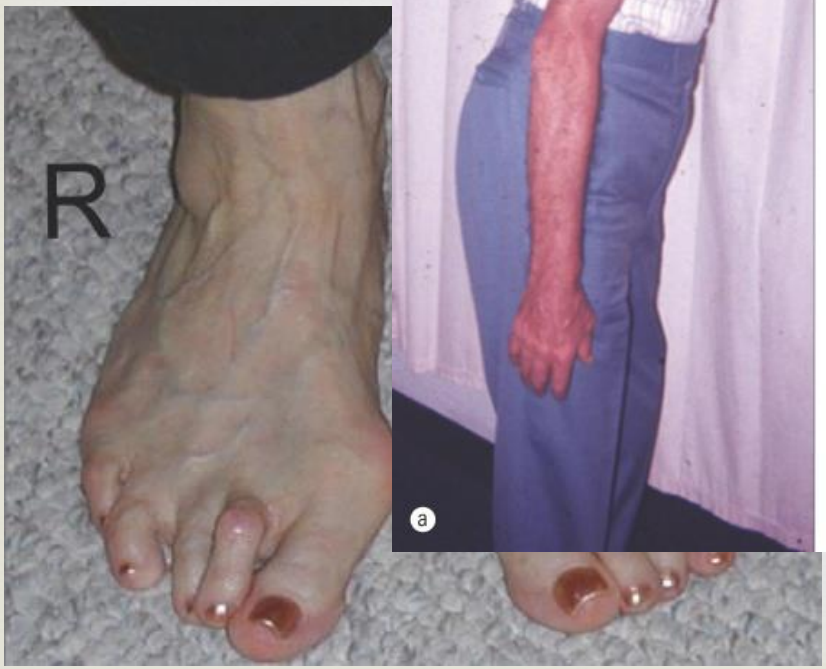
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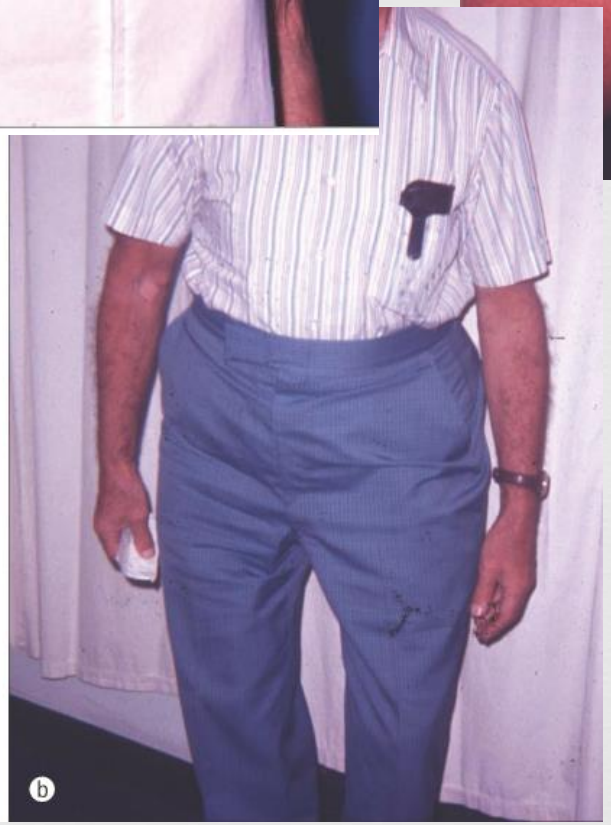

b

OA Diagnostic criteria (ACR)

Osteoarthritis of the Hand

- Hand pain, aching, or stiffness most days of the last month and;
- Hard tissue enlargement of two or more of 10 selected joints and;*
- Fewer than three swollen MCP (metacarpophalangeal) joints and;
- Hard tissue enlargement of two or more DIP (distal interphalangeal) joints;
- Deformity of two or more of 10 selected joints

Points 1, 2, 3 and 4 either 1, 2, 3, and 5 are necessary for the diagnosis. 92% sensitivity, 98% specificity.

Osteoarthritis of the Hip

- Hip pain and most days of the last month;
- Femoral and/or acetabular osteophytes evident on x-ray or
- ESR ≤ 20 mm/hour and;
- Internal hip rotation of ≤ 15 degrees

Points 1 and 2 either 1, 3 and 4 are necessary for the diagnosis. 91% sensitivity, 89% specificity.

Osteoarthritis of the Knee

- **Knee pain most days of the last month and;**
- **Marginal osteophytes**
- **Synovial fluid examination showing clear, viscous fluid with a white blood cell count less than 2,000/mm3.**
- **Age ≥40 years**
- **Morning stiffness ≤ 30 min.**
- **Crepitus at active movements**

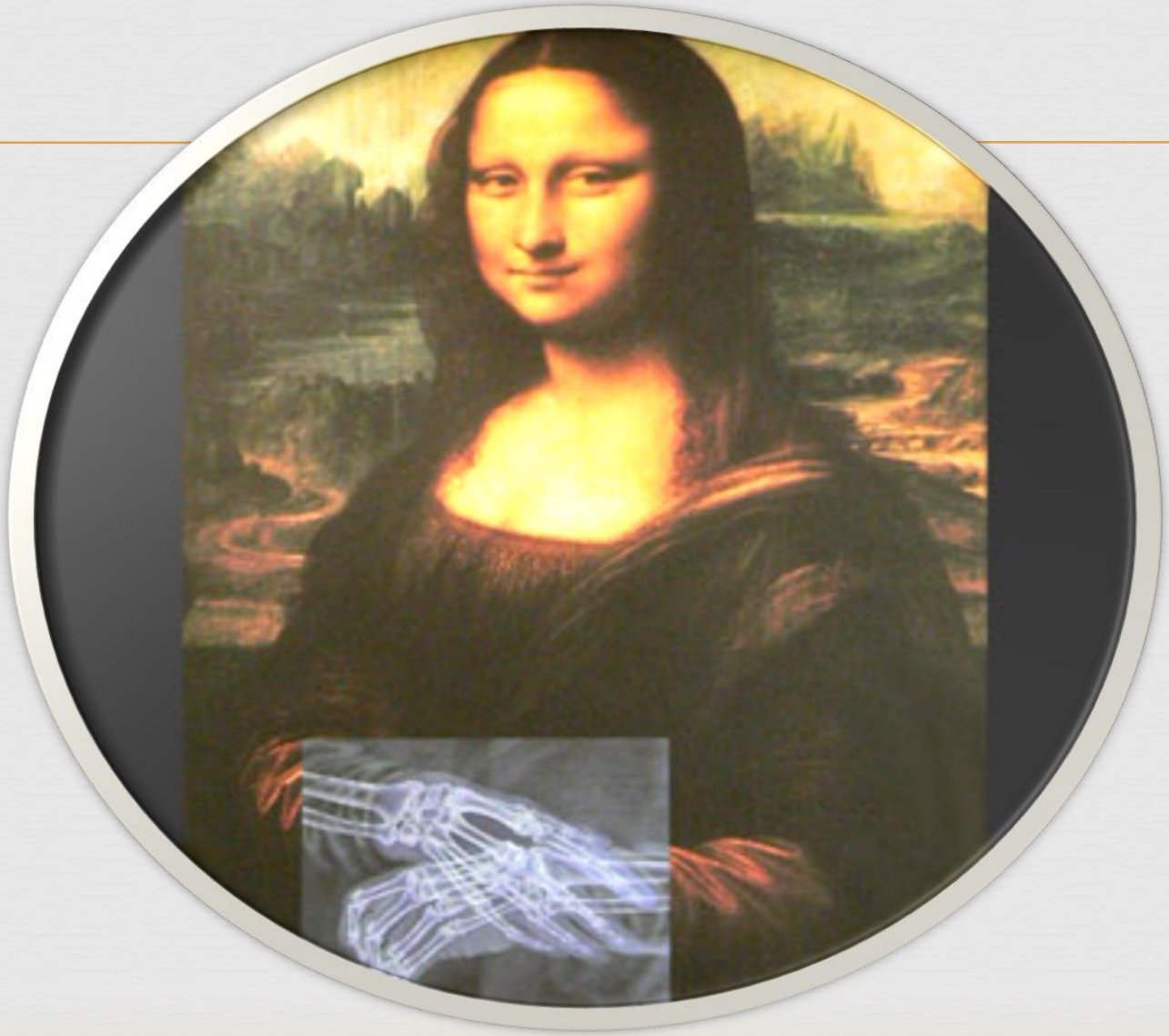
Points 1 and 2 or 1, 3, 5 and 6 either 1, 4, 5 and 6 are necessary for the diagnosis. 94% sensitivity, 88% specificity.

Complementary investigations

Laboratory changes are generally not significant in osteoarthritis and contribute to the diagnosis especially when they are negative.

- Non-specific inflammation (ESR, C-reactive protein) can be slightly elevated in acute periods.
- Synovial fluid is
  - “non-inflammatory" or "mechanical”
  - Viscous, with dense mucin clot in the Ropes test
  - <25% polynuclear
  - Eventually, crystals of hydroxyapatite or calcium pyrophosphate

X-ray

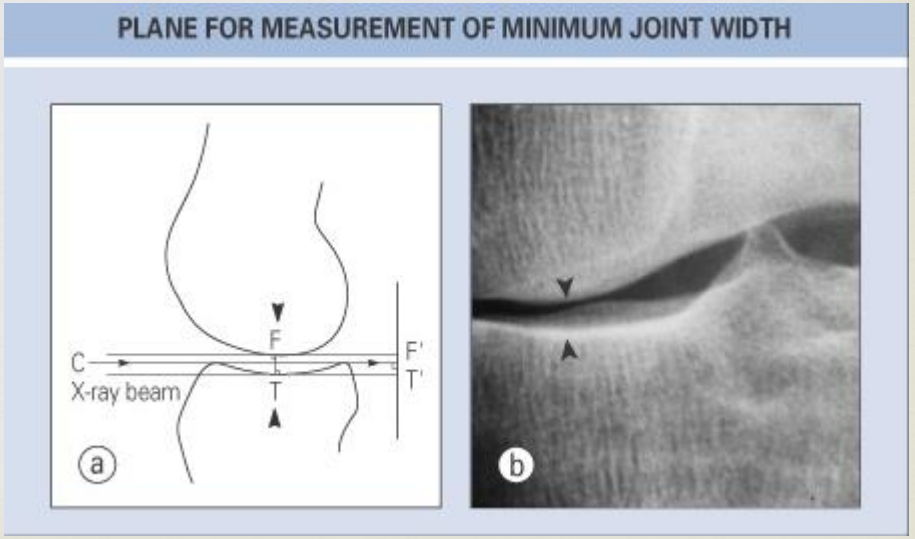


X-ray

- Plain X-ray imaging is the most useful tool in osteoarthritis, both for diagnosis and for monitoring.

View

- Narrowing of the joint space
- Subchondral bone sclerosis
- Marginal ostephytes
- Pseudocysts or geodes



Kellgren and Lawrence Radiographic Criteria for Assessment of OA*

Radiographic grade	0	I	II	III	IV
Classification	Normal	Doubtful	Mild	Moderate	Severe
Description	No features of OA	Minute osteophyte; doubtful significance	Definite osteophyte; normal joint space	Moderate joint-space reduction	Joint space greatly reduced; subchondral sclerosis

*Radiography does not reliably correlate with symptoms.

Cooper C et al. In: Brandt KD, Doherty M, Lohmander LS, eds. *Osteoarthritis*. Oxford, NY: Oxford University Press; 1998:237-249.

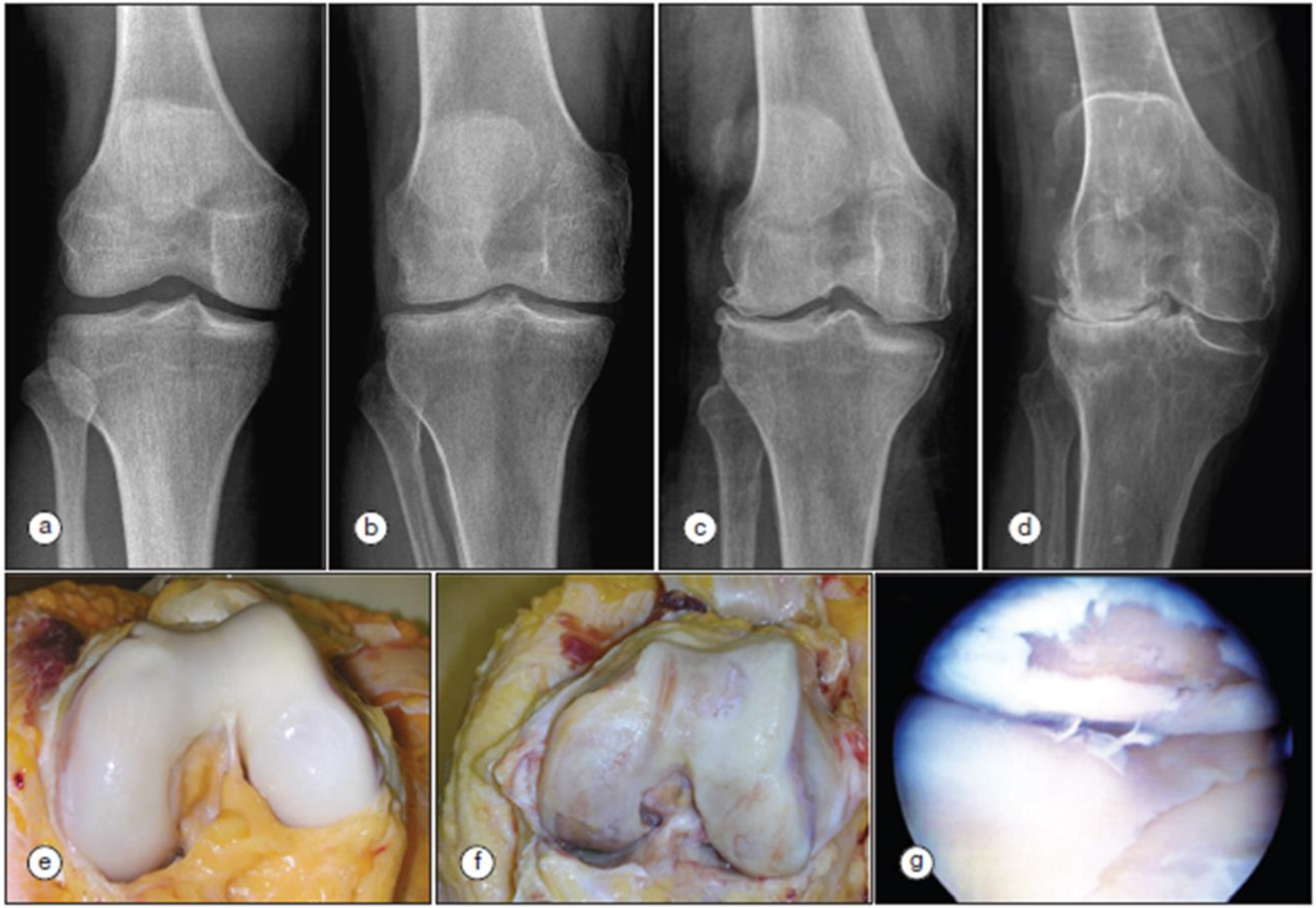
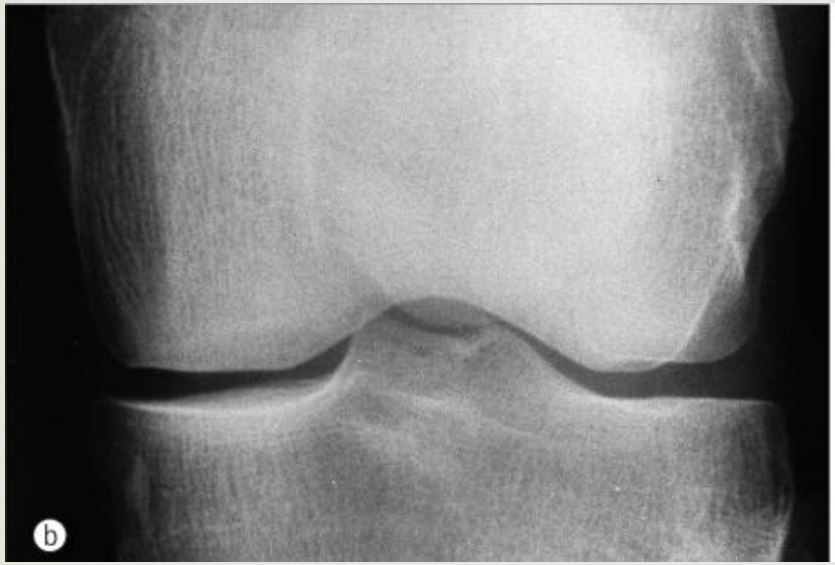


Fig. 173.3 Knee: (a) grade 0 normal, (b) grade 1 lateral tibiofemoral narrowing, (c) grade 3 lateral tibiofemoral narrowing, and (d) grade 3 lateral tibiofemoral narrowing. (e, f) Macroscopic appearance of femoral condyles of a normal (e) and severely damaged (f) knee. (g) Arthroscopic image of a cartilage defect of the femoral condyle within the knee joint. *(a-d, from Altman RD, Gold GE. Atlas of individual radiographic features in osteoarthritis, revised. Osteoarthritis Cartilage 2007;15[Suppl A]:A1-A56; g, courtesy of Dr. W. Eger, Rummelsberg.)*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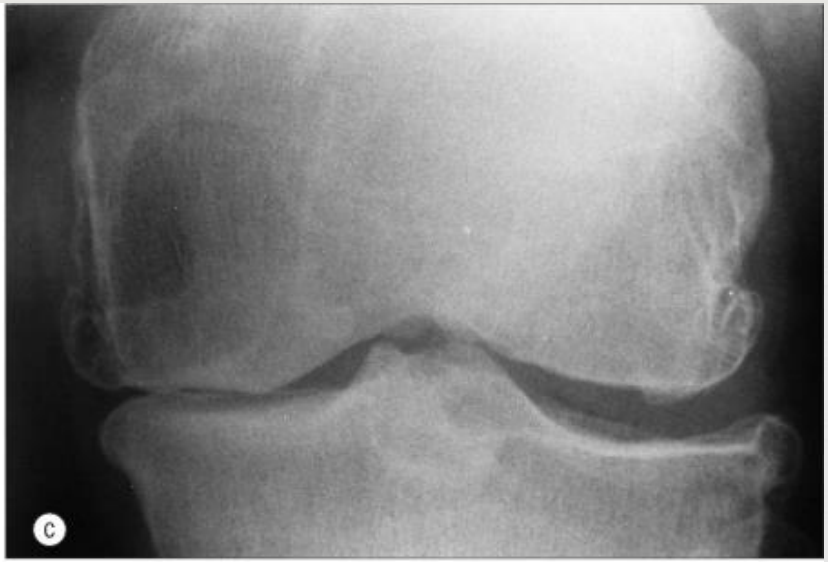
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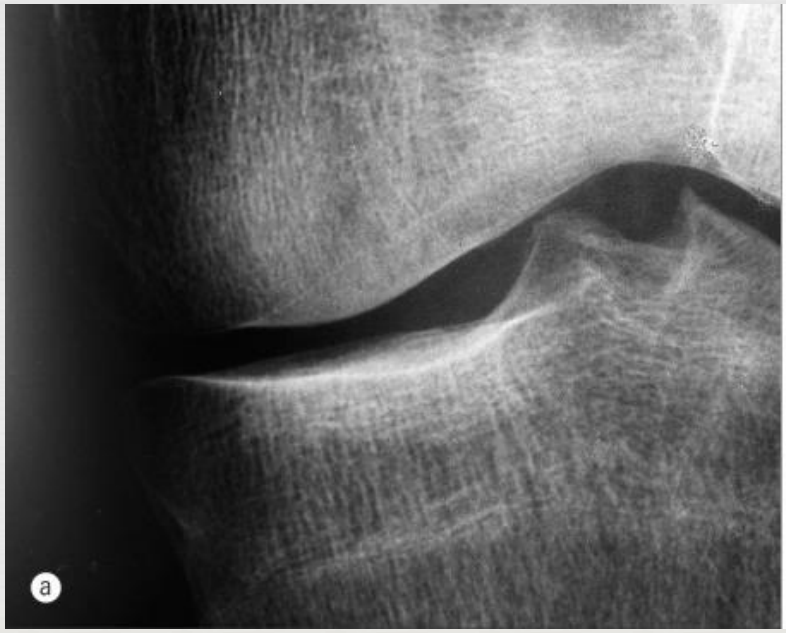
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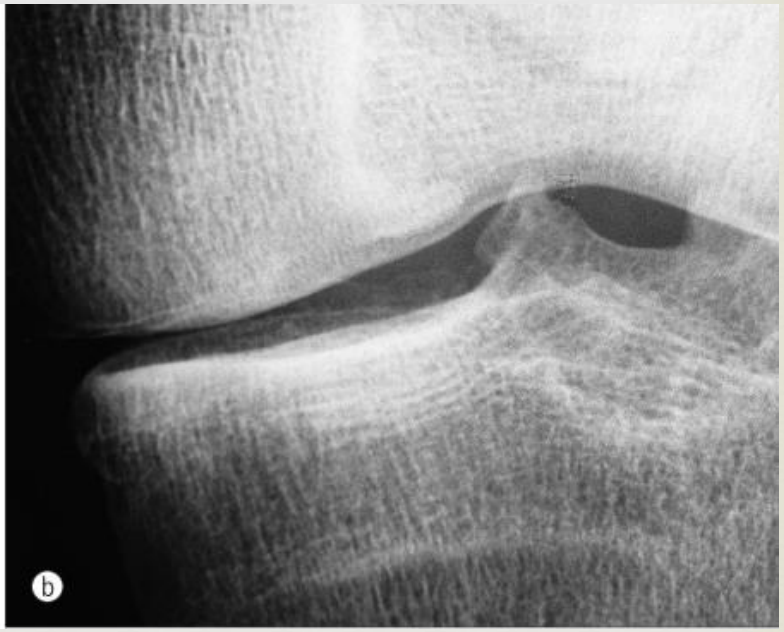
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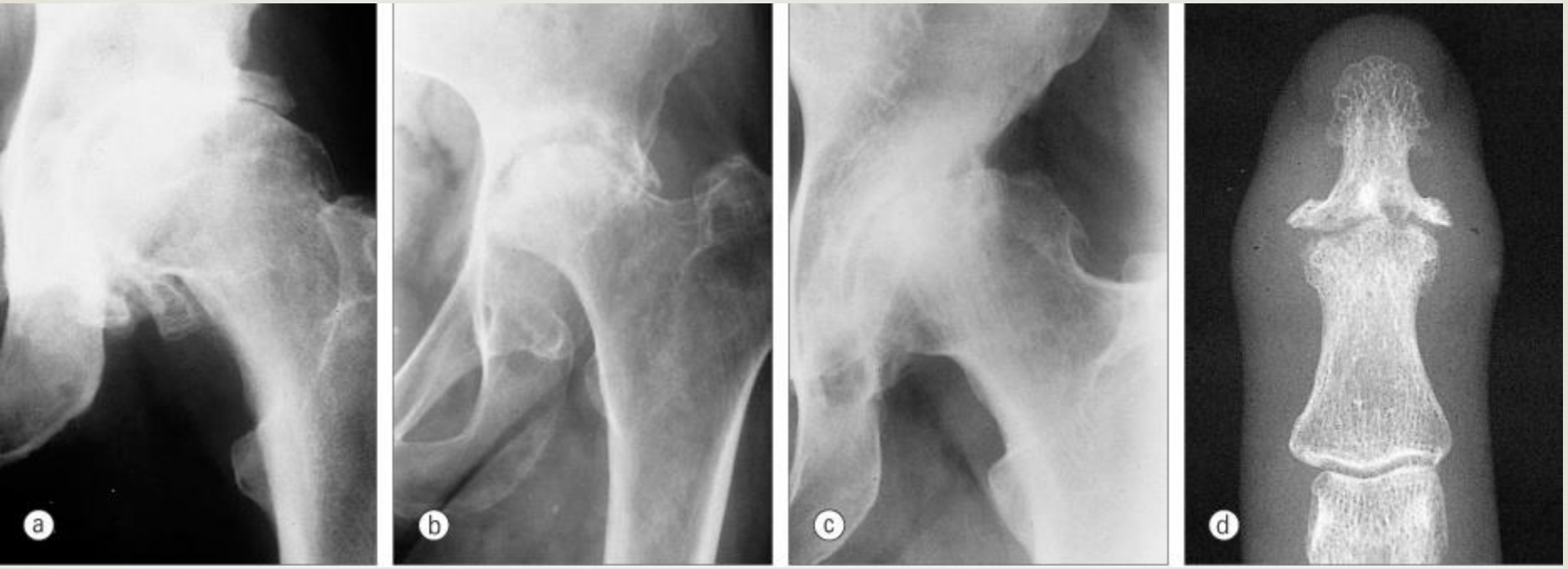
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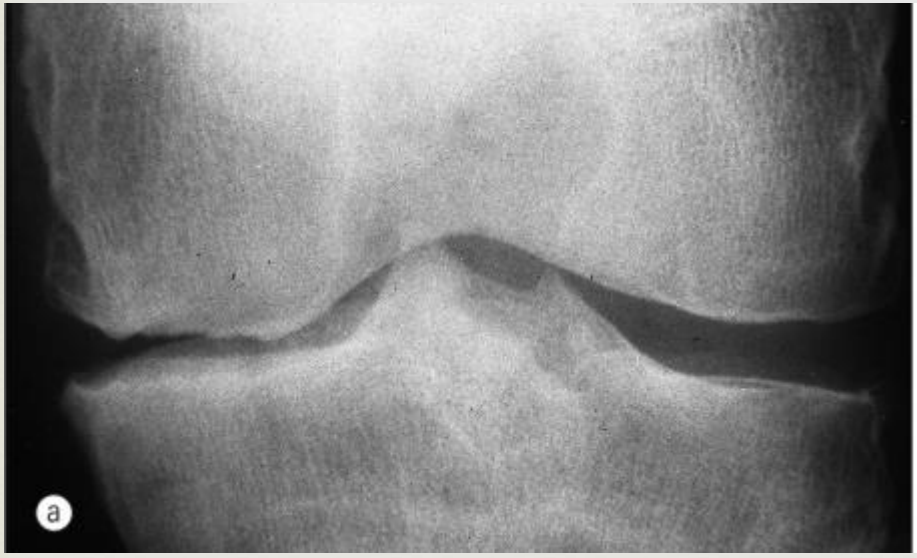
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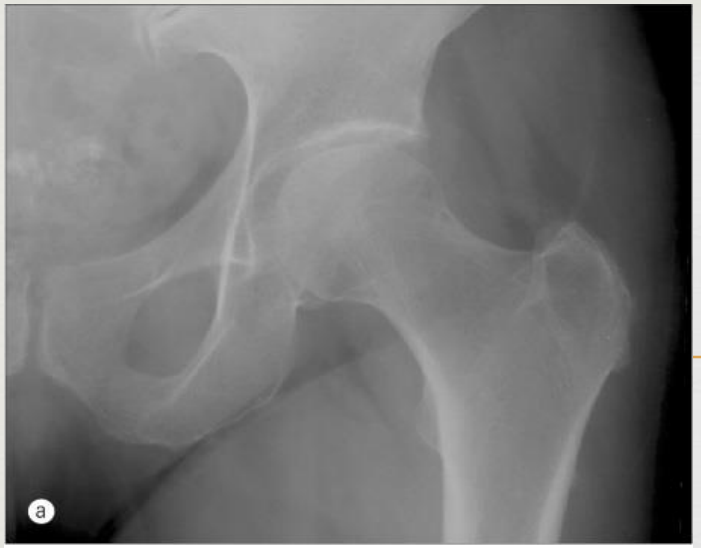

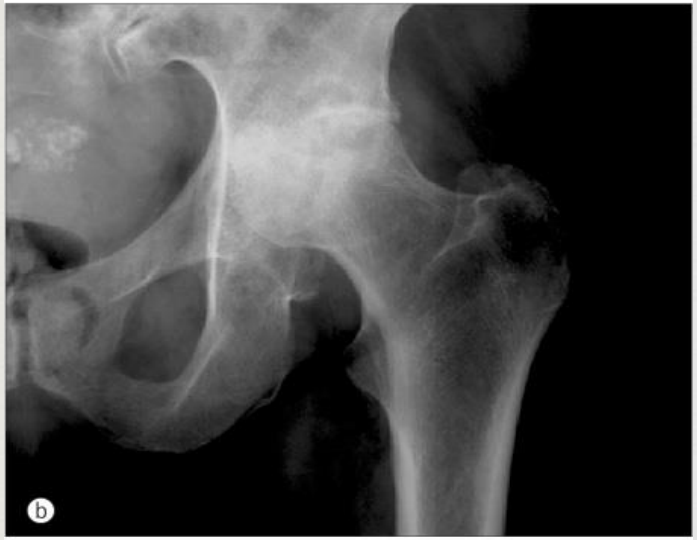

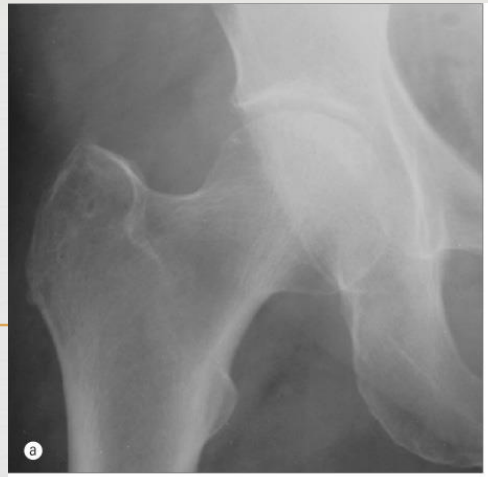

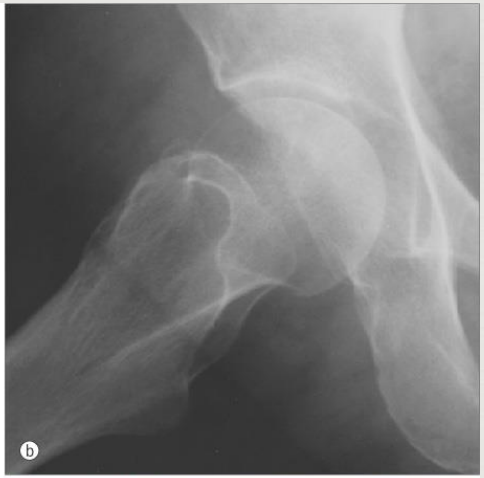

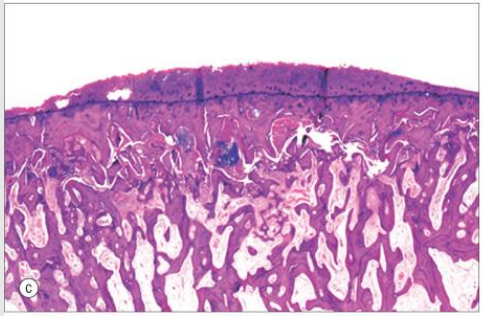

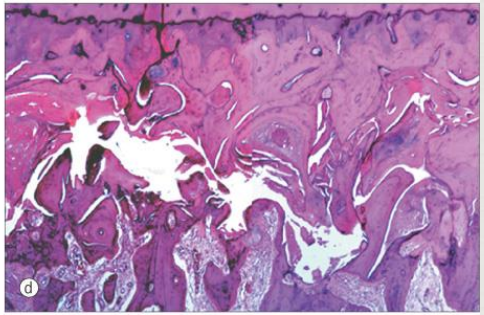

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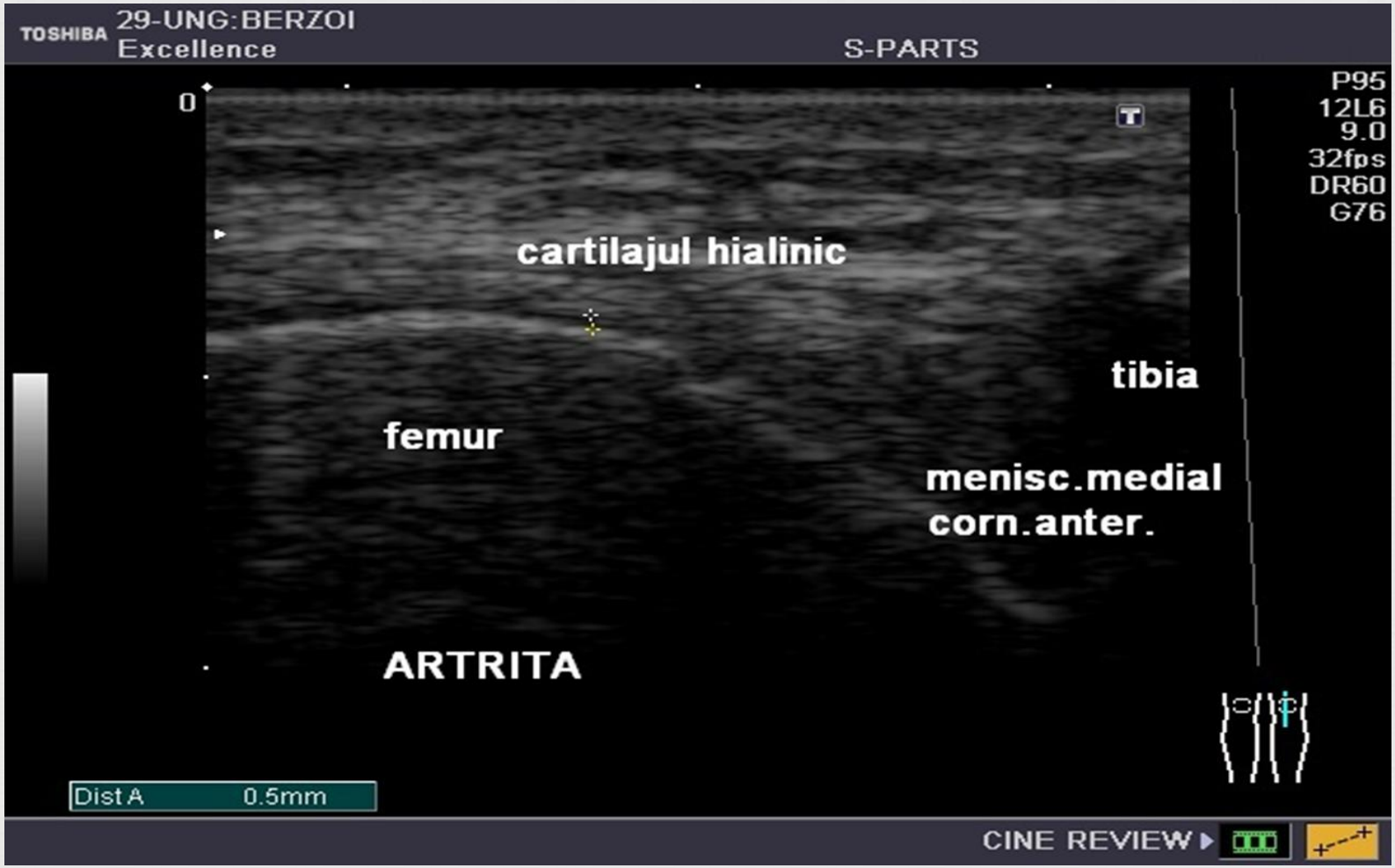
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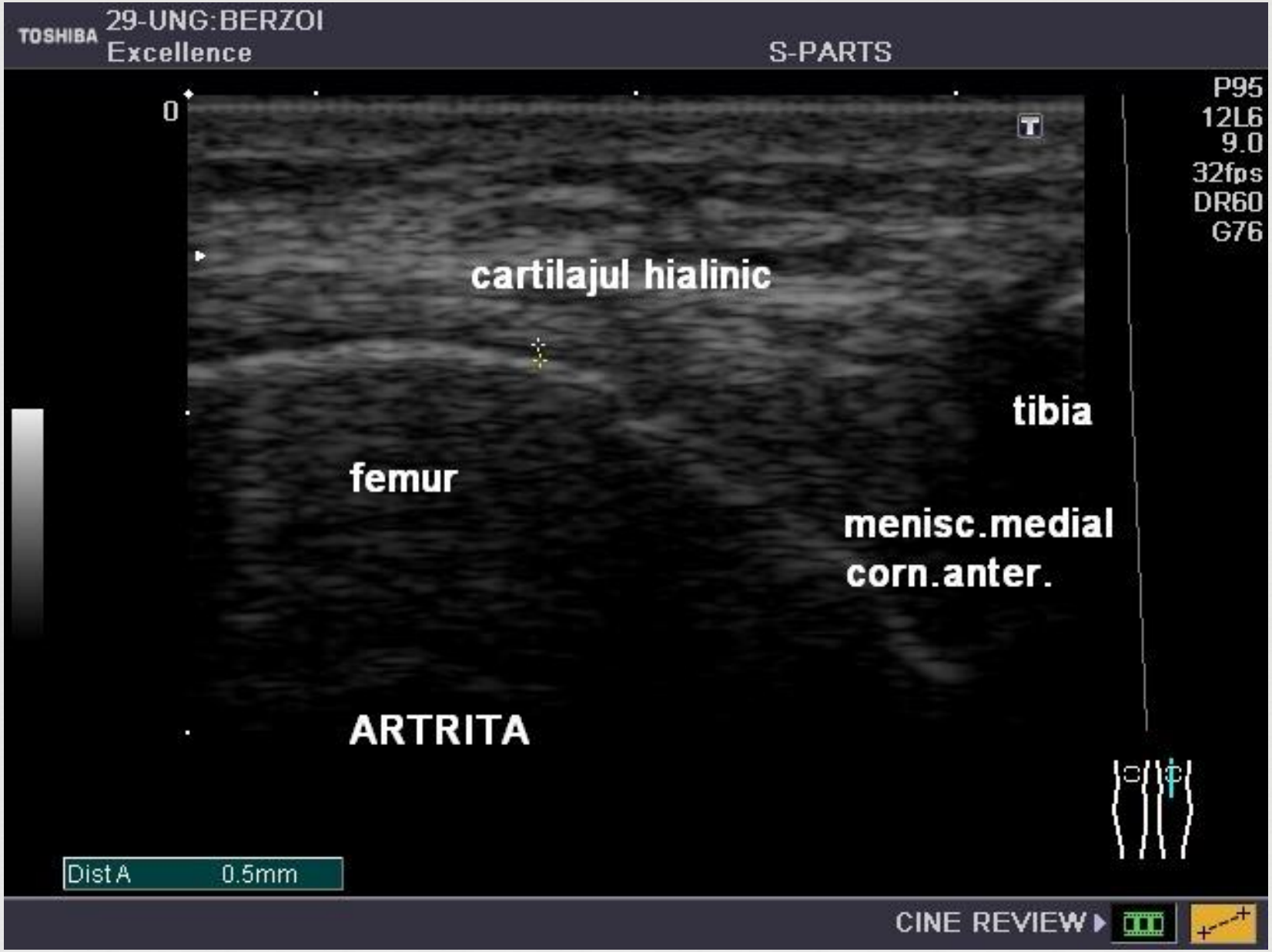
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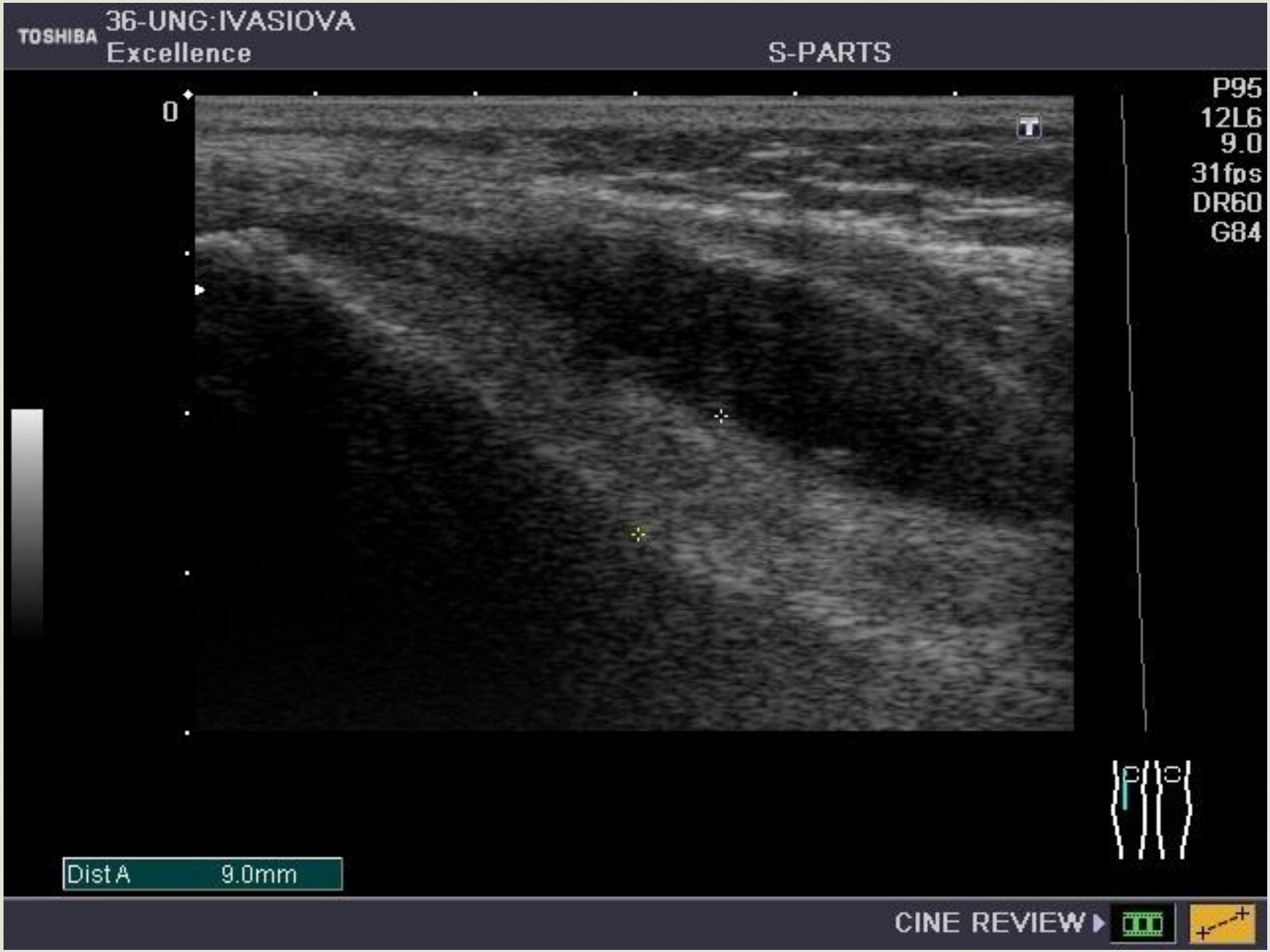
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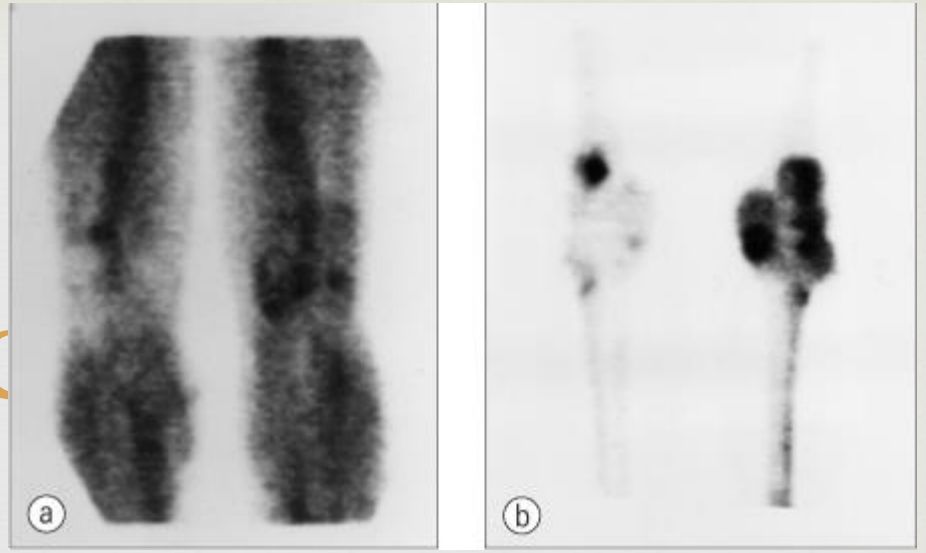
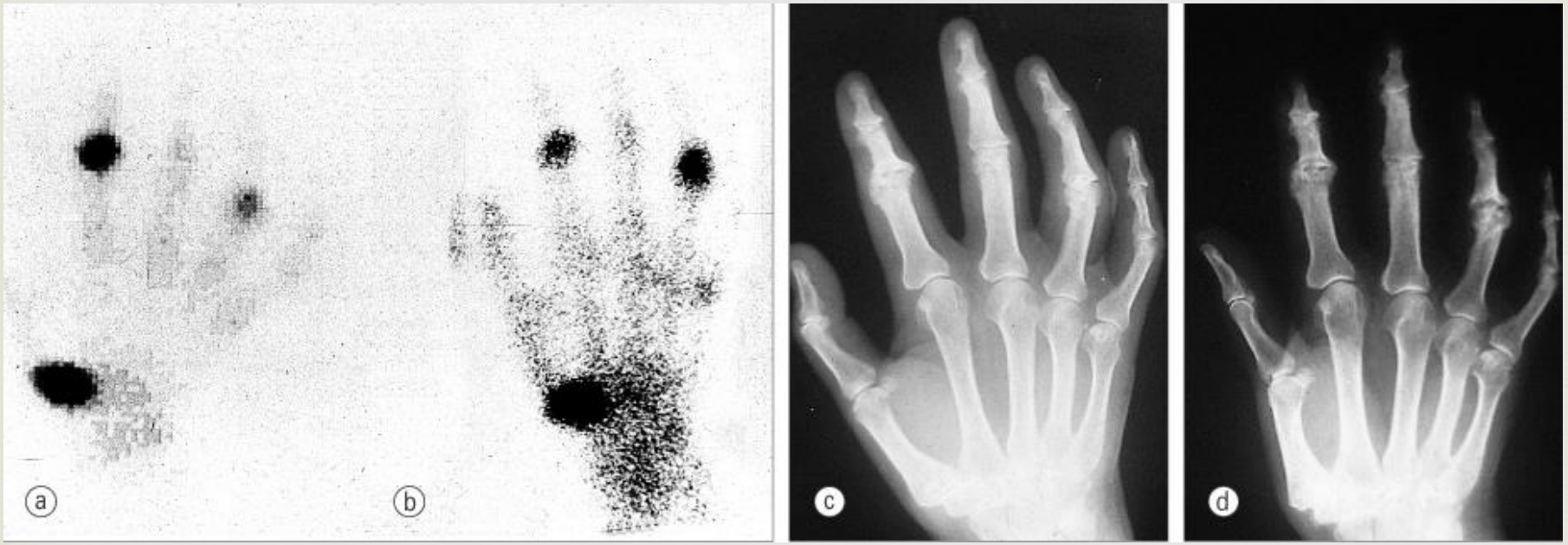
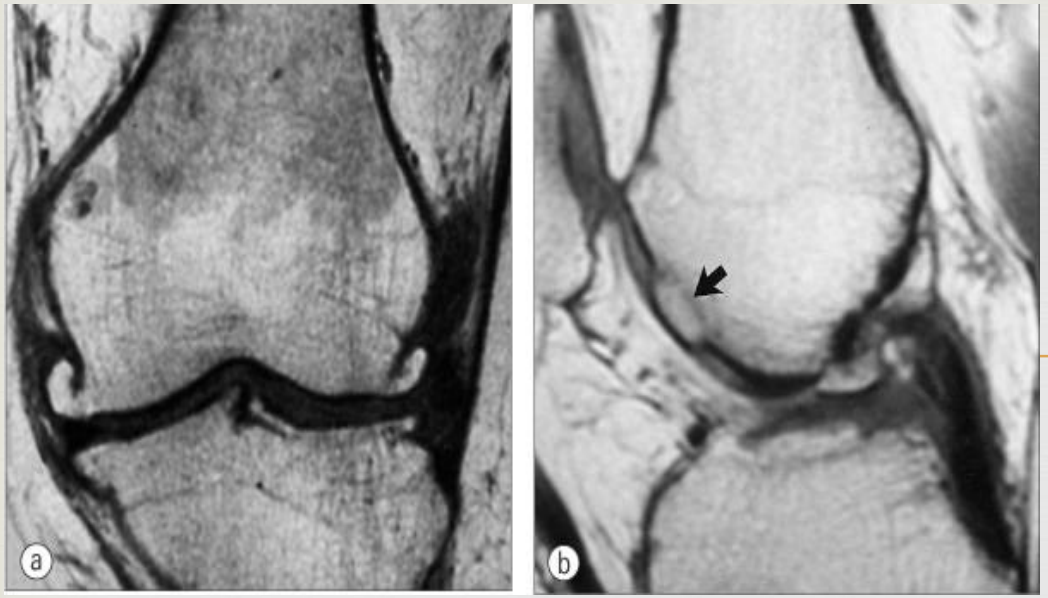
Scintigraphy



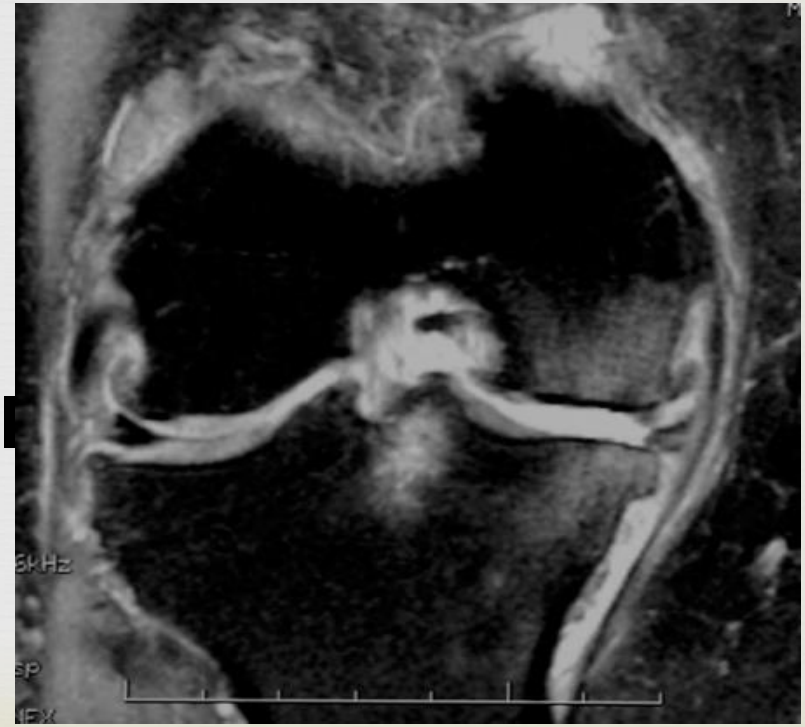


- **Scintigraphy may “predict” radiological changes**

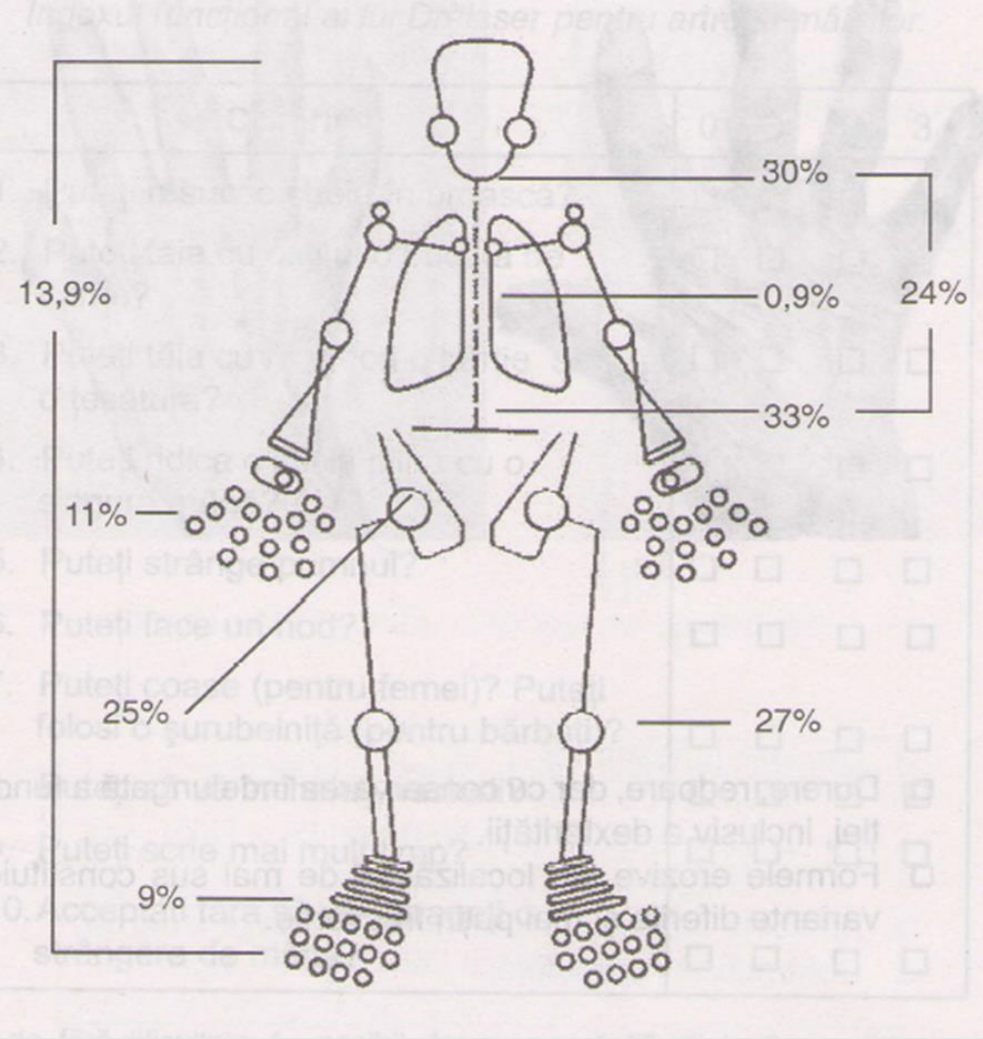


Identification of osteophytes on MRI

Swelling of the subchondral bone marrow depicted onMRI



Clinical topographic forms of peripheral osteoarthritis



The topographic forms rates differ depending on different populations and time periods.

The next picture represents some of the generally recognized rates.

PREVENTION

Healthy lifestyle

Patient education

Physical activity

OSTEOARTHRITIS PREVENTION

Correct position during sleep

Menopause management

Correct positioning during physical activity

Weight loss

Utilizarea cârjei
Orteze,
Suporturi

Therapy

- The goals of treatment in osteoarthritis are as follows

Short-term objectives	Long-term objectives
Antalgic drugs	Delay development
Antiinflammatory drugs	Prevent functional deficit
Chondroproctective drugs	Maintain the quality of life

General treatment

- General treatment is mandatory in all cases and consists of interventions designed to modify the patient's lifestyle:
- Information and Education
- Self-care program
- Coaching (eg telephone contact)
- Weight loss (in obese)
- Physical Therapy: aerobic exercises, muscle strengthening and mobilization
- Occupational Therapy
- Adapting footwear
- Joint Protection (ortheses)
- Walking aids

Symptomatic fast acting drugs - SyFADOA

Are applied in short courses for the painful congestive flares:

- Analgesics (acetaminophen) 3-4 g/day
- Weak opioids (codeine, tramadol)
- More potent opioids are not usually prescribed with the exception of extremely severe pain and inoperable disease

NSAIDs – are applied in courses of 1-1.5 months

- **Non-selective NSAIDs**
  - ✓ ibuprofen
  - ✓ ketoprofen
  - ✓ diclofenac
  - ✓ aceclofenac 100mg bid
- Selective NSAIDs:
  - ✓ COX 2 inhibitors

Symptomatic slow-acting drugs - SySADOA

- Preparations leading to symptomatic slow-acting relief and effective retardation of 2-3 months, are characterized by good tolerance and merely no side effects that prevent the deterioration of joints (chondroprotective) (structure modifying drugs -SMDs)

- **Chondroitine sulphate, glucosamine sulphate/clorhydrate** : these preparations should be administered at least 2-3 months to 8-12 months to assess the effect and improve joint function. Decreased use of pain-killers and NSAIDs intake is a good criterion of effectiveness.

Intraarticular therapy

- **Intra-articular corticosteroids**: are especially useful in synovitis and inflammation. The exact mechanism by which corticosteroids act is unknown, but it has been shown that in syinoviocites from the osteoarthritic joint inhibit the synthesis of IL-1 and TNF-α. However, repeated injection causes chondroresorbtion and additional cartilage deterioration. For these reasons, intra-articular therapy with corticosteroids should be limited to no more than 3-4 times a year.

- Hyaluronic acid derivatives: it is known that in the osteoarthritic joints, hyaluronate concentration is reduced. Therefore, intra-articular administration of its derivatives is warranted, clinical trials showing a significant reduction in pain particularly for osteoarthritis of the knee.

Treatment in discussion

- Synthetic antimalarials (chloroquine)
- Diacerein
- Pentosan polysulphate
- Avocado
- systemic enzimes (Wobenzim)
- Anti-cytokine therapy

Additional therapy

- Antidepressant (paroxetin)
- Myorelaxants (thiocolchicoside, tolperisone, tizanidin)
- Antiaggregant (pentoxifylline)

The role of cytokines:

- The mainly explored is IL-1beta which has the capacity to stimulate stromelizin secretion, the only chondrocyte enzyme, which, being produced locally, is involved in the degradation and depletion of matrix aggrecan. IL-1beta receptor antagonists could interfere with this process
- It is being studied the role of *TNF alfa* in the liposomes that induce the reparatory activity in the injured cartilage.

Surgery

- Osteotomy
- Arthroscopic debridement
- Arthrodesis
- Arthroplasty

« Biological » surgery

- osteochondral graft
- "Tissue Engineering" by autologous chondrocyte transplantation of undifferentiated mesenchymal cells

Non-pharmacological modalities of treatment

2. All patients with hip and knee OA should be given information access and education about the objectives of treatment and the importance of changes in lifestyle, exercise, pacing of activities, weight reduction, and other measures to unload the damaged joint(s).
3. The clinical status of patients with hip or knee OA can be improved if patients are contacted regularly by phone

Non-pharmacological modalities of treatment

4. Patients with symptomatic OA may benefit from referral to a physical therapist for evaluation and instruction in appropriate exercises to reduce pain and improve functional capacity. This evaluation may result in provision of assistive devices such as canes and walkers, as appropriate.
5. Patients with hip and knee OA should be encouraged to undertake, and continue to undertake, regular aerobic, muscle strengthening and range of motion exercises. For patients with symptomatic hip OA, exercises in water can be effective.

Non-pharmacological modalities of treatment

6. Patients with hip and knee OA, who are overweight, should be encouraged to lose weight and maintain their weight at a lower level.
7. Walking aids can reduce pain in patients with hip and knee OA. Patients should be given instruction in the optimal use of a cane or crutch in the contralateral hand. Frames or wheeled walkers are often preferable for those with bilateral disease.

Non-pharmacological modalities of treatment

8. In patients with knee OA and mild/moderate varus or valgus instability, a knee brace can reduce pain, improve stability and diminish the risk of falling.
9. Every patient with hip or knee OA should receive advice concerning appropriate footwear. In patients with knee OA insoles can reduce pain and improve ambulation. Lateral wedged insoles can be of symptomatic benefit for some patients with medial tibio-femoral compartment OA.

Pharmacological modalities of treatment

10. Acetaminophen (up to 4 g/day) can be an effective initial oral analgesic for treatment of mild to moderate pain in patients with knee or hip OA. In the absence of an adequate response, or in the presence of severe pain and/or inflammation, alternative pharmacologic therapy should be considered based on relative efficacy and safety, as well as concomitant medications and co-morbidities.

Pharmacological modalities of treatment

11. In patients with symptomatic hip or knee OA, **NSAIDs** should be used at the lowest effective dose but their long-term use should be avoided if possible. In patients with increased GI risk, either a COX-2 selective agent or a non-selective NSAID with co-prescription of a PPI or misoprostol for gastroprotection may be considered, but NSAIDs, including both non-selective and COX-2 selective agents, should be used with caution in patients with CV risk factors.

Pharmacological modalities of treatment

12. **Topical NSAIDs and capsaicin** can be effective as adjunctives and alternatives to oral analgesic/anti-inflammatory agents in knee OA.
13. **IA injections with corticosteroids** can be used in the treatment of hip or knee OA, and should be considered particularly when patients have moderate to severe pain not responding satisfactorily to oral nalgesic/anti-inflammatory agents and in patients with symptomatic knee OA with effusions or other physical signs of local inflammation.
14. **IA Injections of hyaluronate** may be useful in patients with knee or hip OA.

Pharmacological modalities of treatment

15. **Treatment with glucosamine and/or chondroitin sulphate** may provide symptomatic benefit in patients with knee OA. If no response is apparent within 6 months treatment should be discontinued.
16. In patients with symptomatic knee OA glucosamine sulphate and chondroitin sulphate may have structure-modifying effects while **diacerein** may have structure-modifying effects in patients with symptomatic OA of the hip.

Pharmacological modalities of treatment

17. The use of **weak opioids and narcotic analgesics can be considered** for the treatment of refractory pain in patients with hip or knee OA, where other pharmacological agents have been ineffective, or are contraindicated. Stronger opioids should only be used for the management of severe pain in exceptional circumstances. Non-pharmacological therapies should be continued in such patients and surgical treatments should be considered.

Surgical modalities of treatment

18. **Joint replacement surgery.**
19. Replacement arthroplasties are effective, and cost-effective interventions for patients with significant symptoms, and/or functional limitations associated with a reduced health-related quality of life, despite conservative therapy.

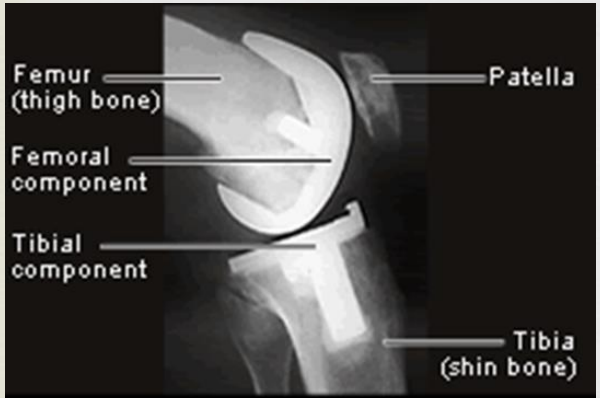


Figure 11. A replacement knee joint, viewed from the side, showing the two artificial parts (components) securely attached to the bones