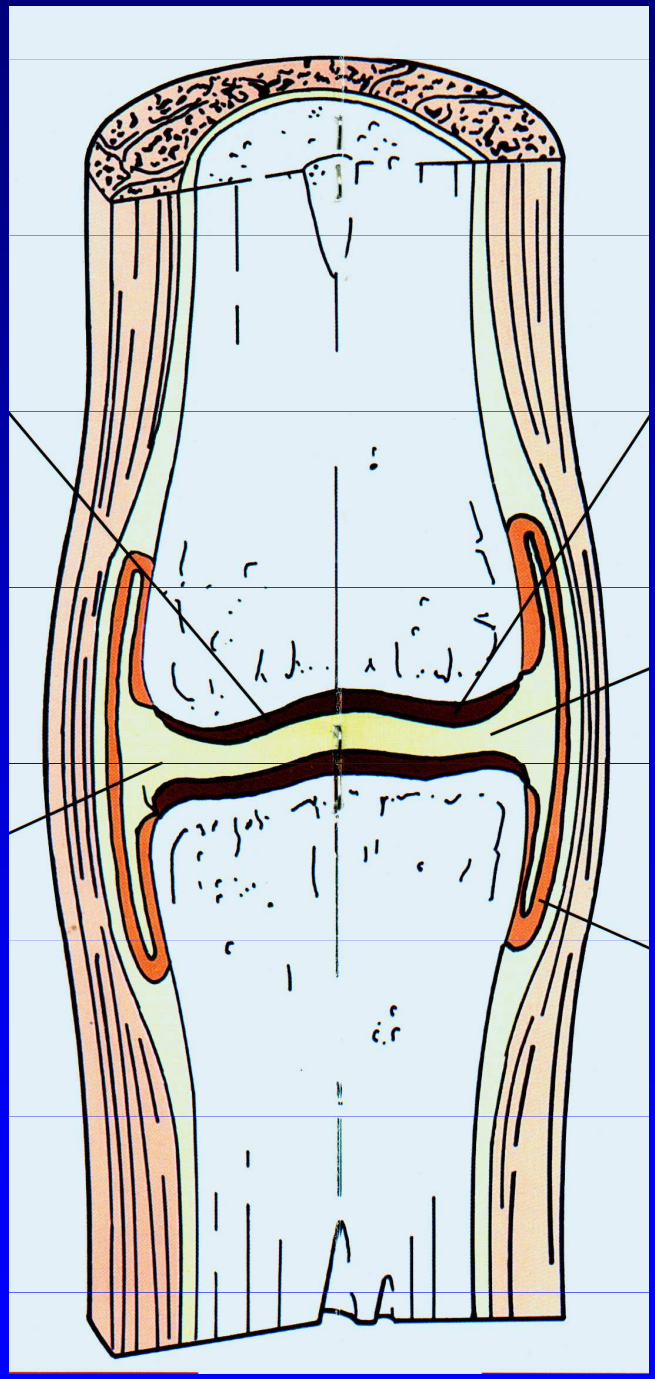
Osteoarthritis

Z. Rozkydal

Synovial joint

The end of bones
Hyaline cartilage
Ligaments
Joint capsule
Synovial membrane
Synovial fluid



Hyaline cartilage

Chondrocytes

Matrix – intercelullar mass:

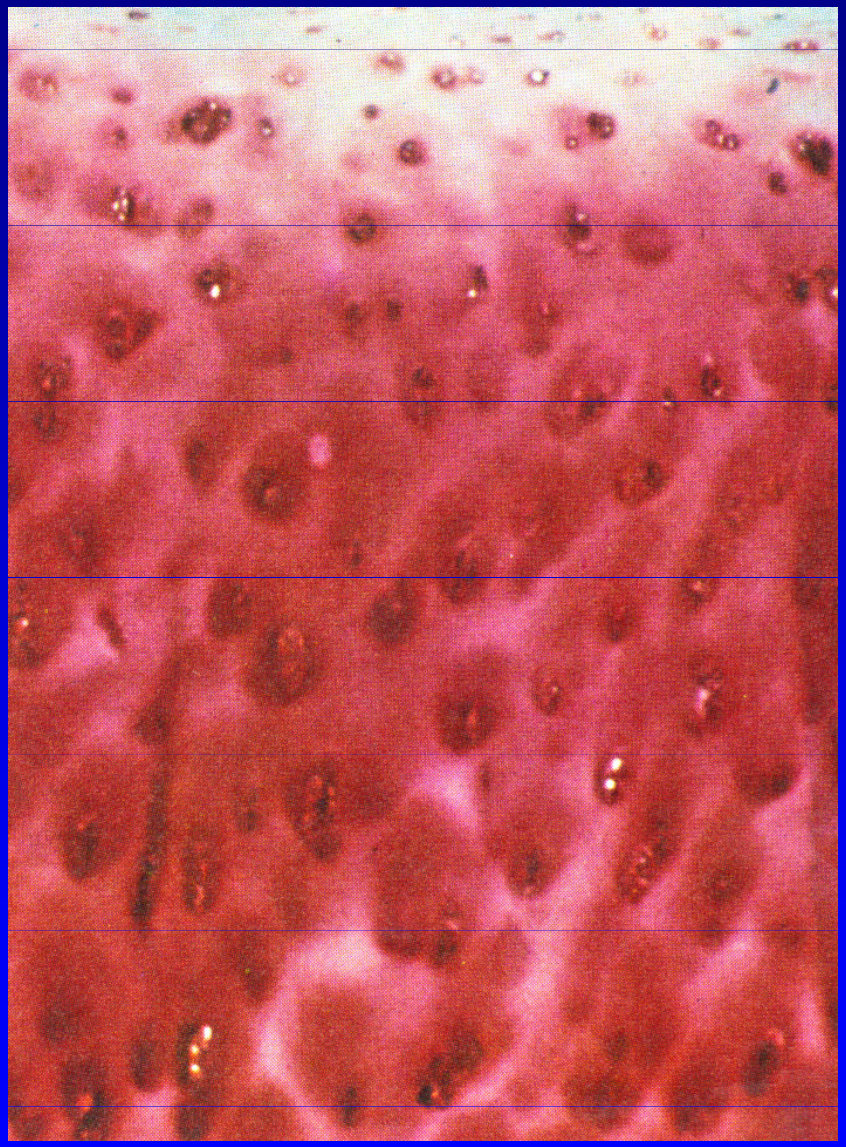
Fibrilar structure - collagen

Proteoglycans

Proteins of noncollagen nature

Hyaluronic acid

Water – 70 volume percent

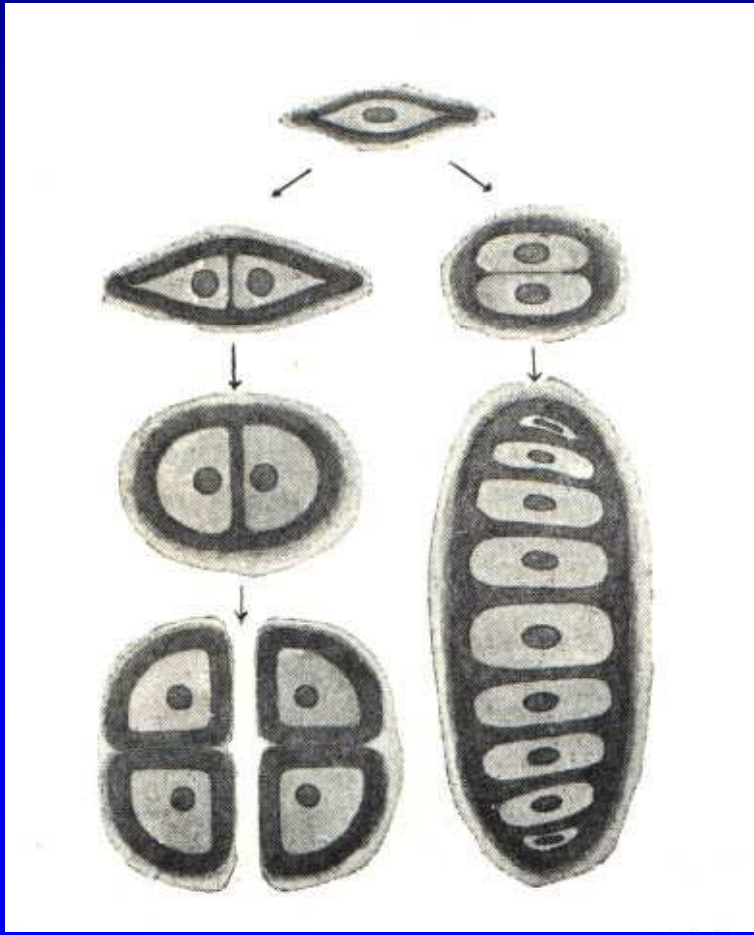


Hyaline cartilage

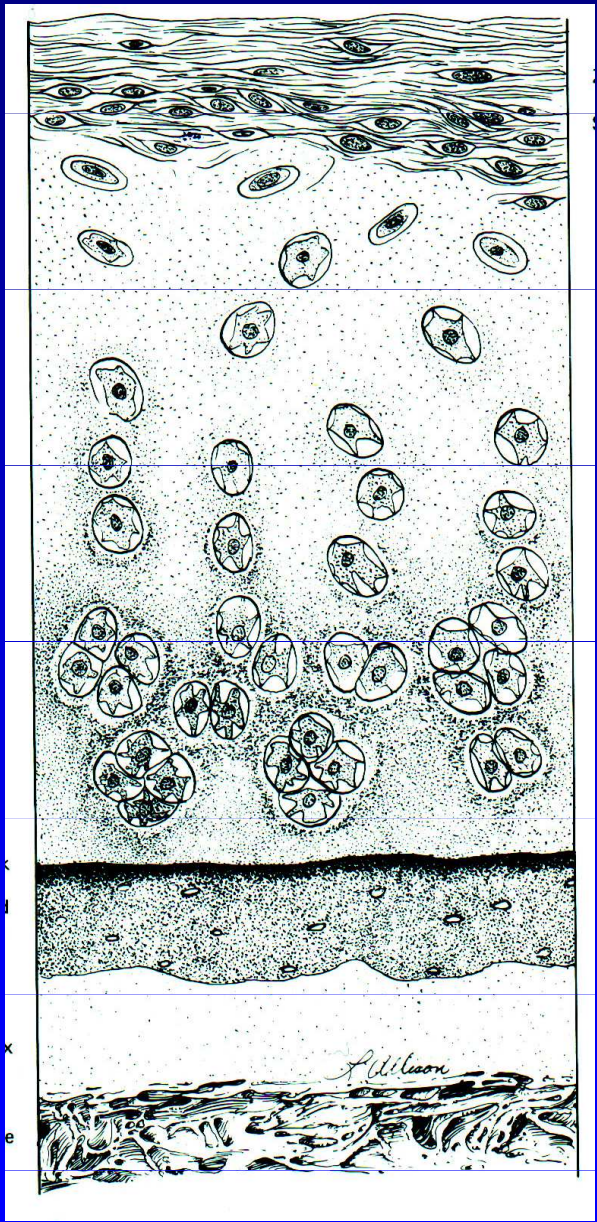
Chondrocytes- 2 percent of volume

Localised in lacunes of matrix

Isogenetic groups 2-8 cells
from one mother cell



Hyaline cartilage - layers



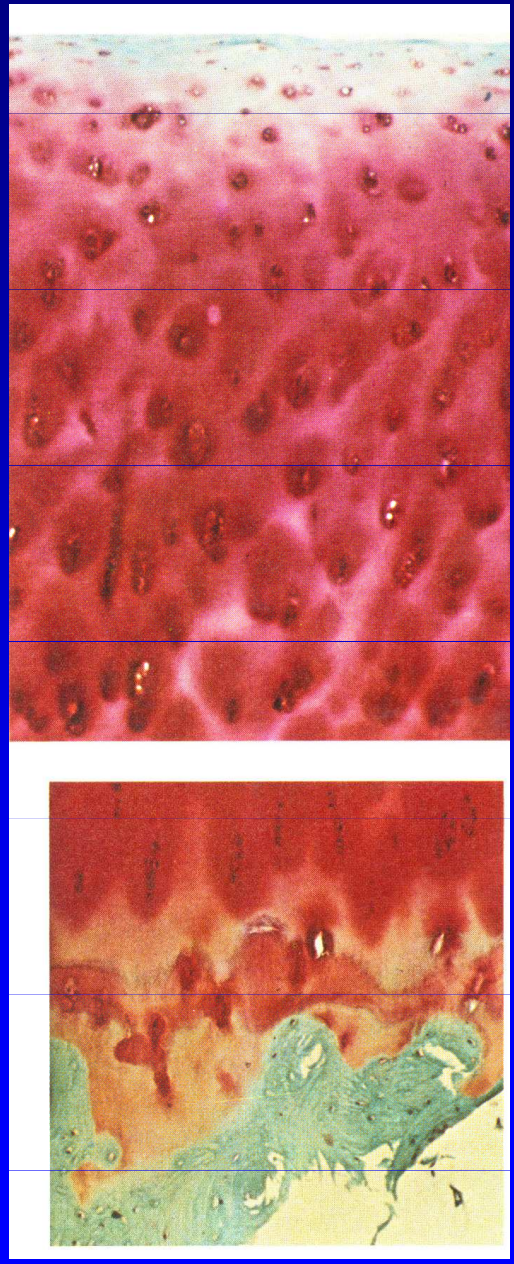
Superficial

Middle

Deep

Zone of calcifying Catilage

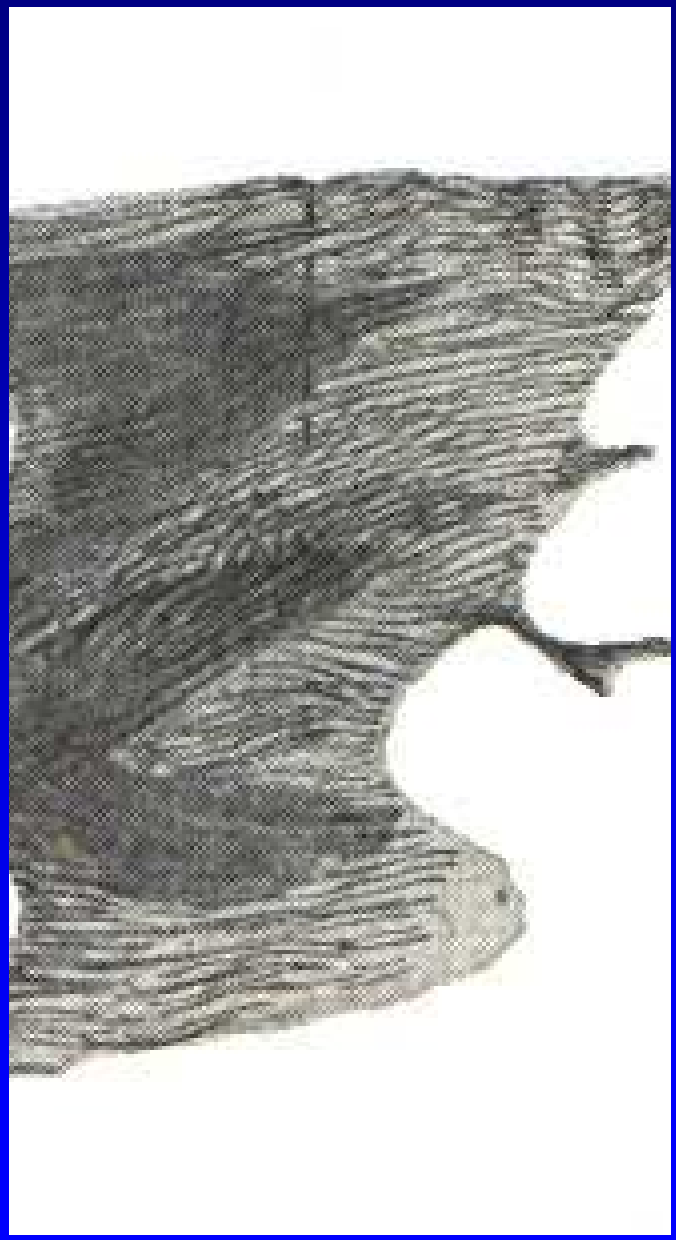
Bone



Collagen

Collagen type II (3 alfa-1 chains- 90 %)

Chains form fibrils
Fibrils form a three dimensional network
Paraler to the surface
In deep layers in columns

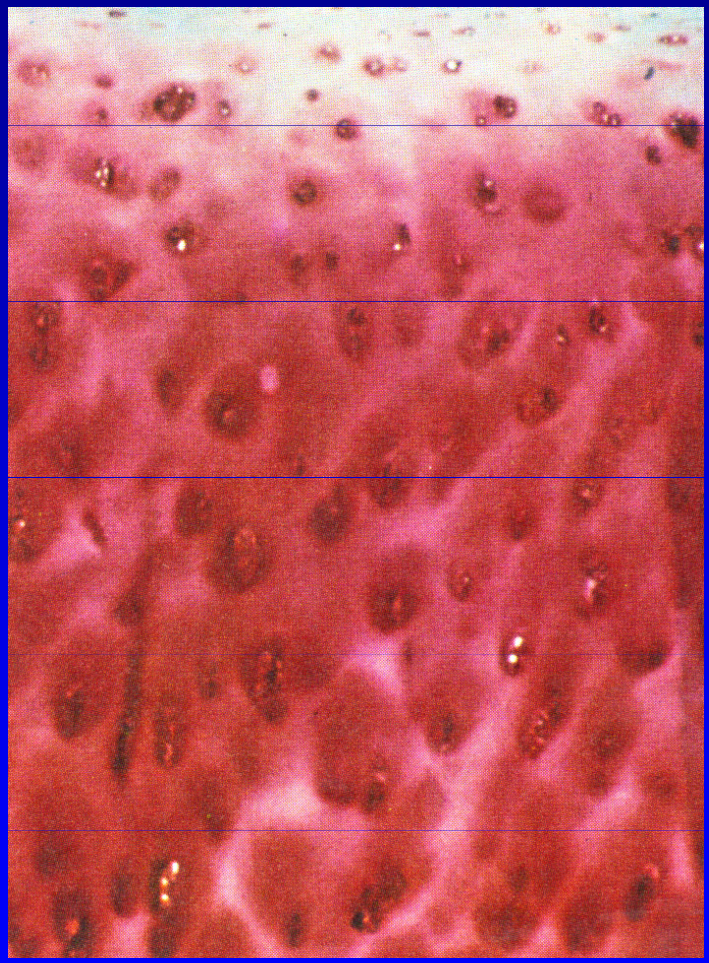


Proteoglycans- PG

They are high hydrofilic- elasticity !!

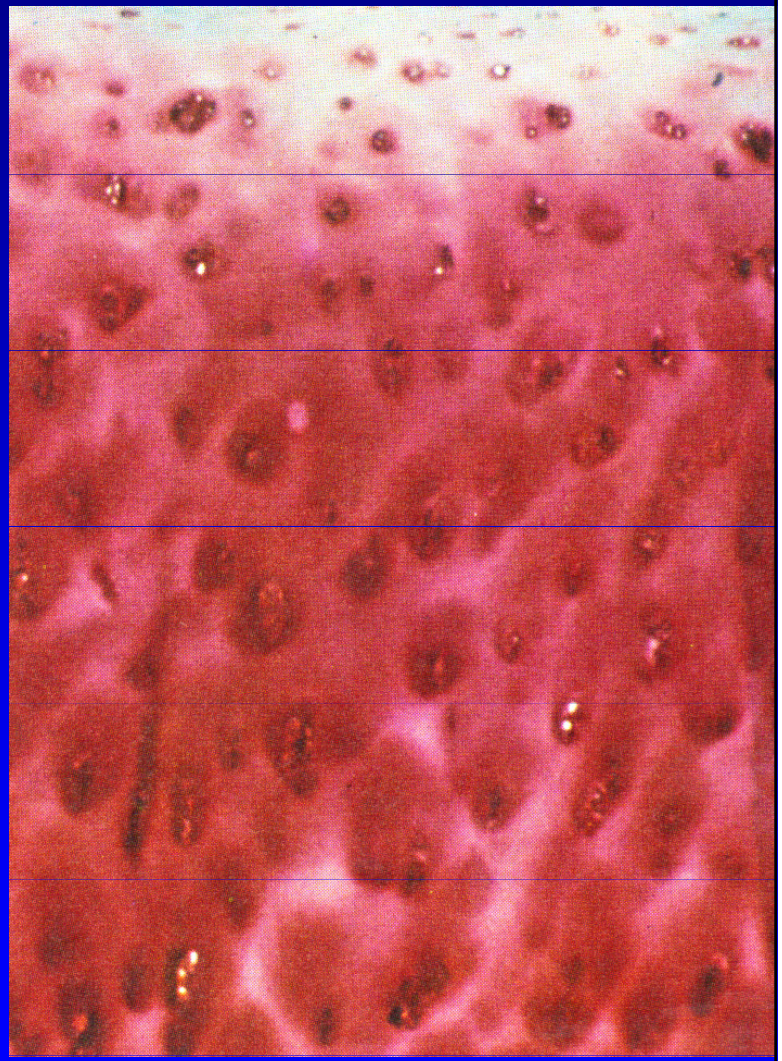
Large PG - glukosaminoglycans:
Chondroitin 6- sulfate
Keratansulfate
Chondroitin 4- sulfate

Small PG:
Decorin, biglycan
Agrecan – binds on hyaluronic acid
Sulfatan glukosaminoglycan



Noncollagen proteins

Fibronectin, chondronectin
Anchorin
Cytocins- interleukin-1, interleukin- 6
Enzymes – metaloproteinase
(kolagenase, gelatinase)
Growth factors
Prostaglandins



Hyaluronic acid

It forms with proteoglycans intercelullar mass

It is hydrophylic, it maintains homeostasis

It is responsible for lubrication of the joint

It promotes transport of nutritiens into the cartilage

It gives the cartilage elastic resistance

It gives rheologic properties to synovial fluid

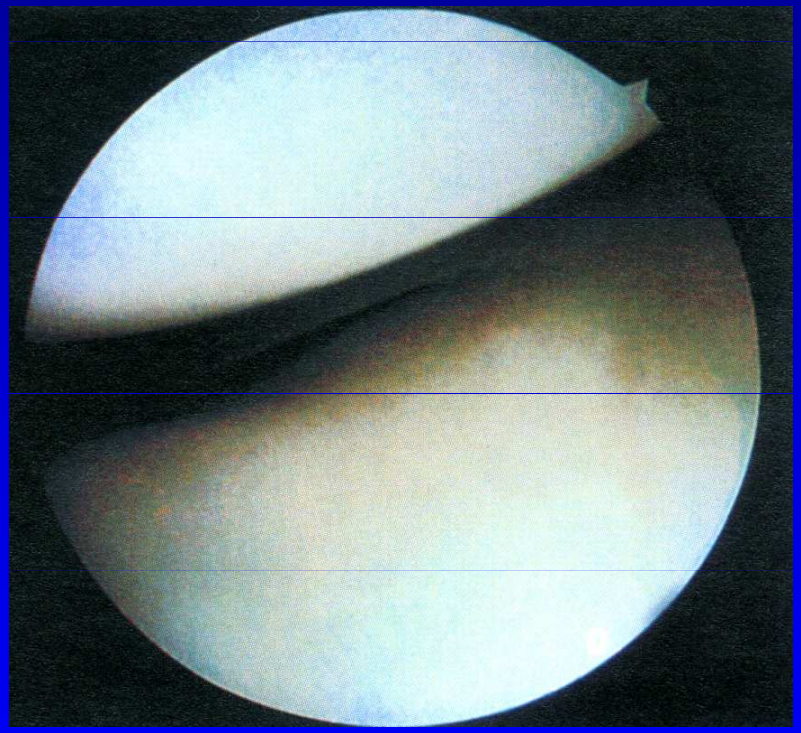
Hyaline cartilage

High volume of water gives resistance in pressure

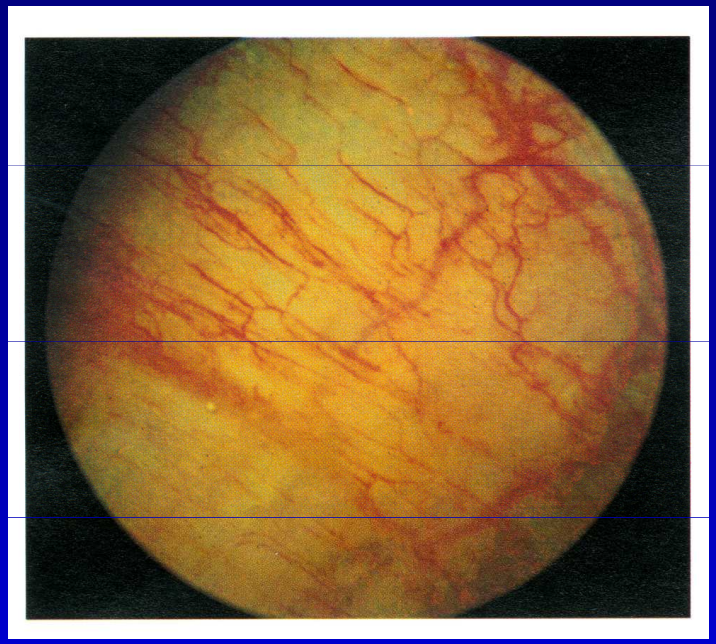
Condrocytes are nourished from synovial fluid

Cartilage has no vessels and nerves - low regeneration

The fluid is pushed by movements into the cartilage



Synovial membrane



Network of vessels

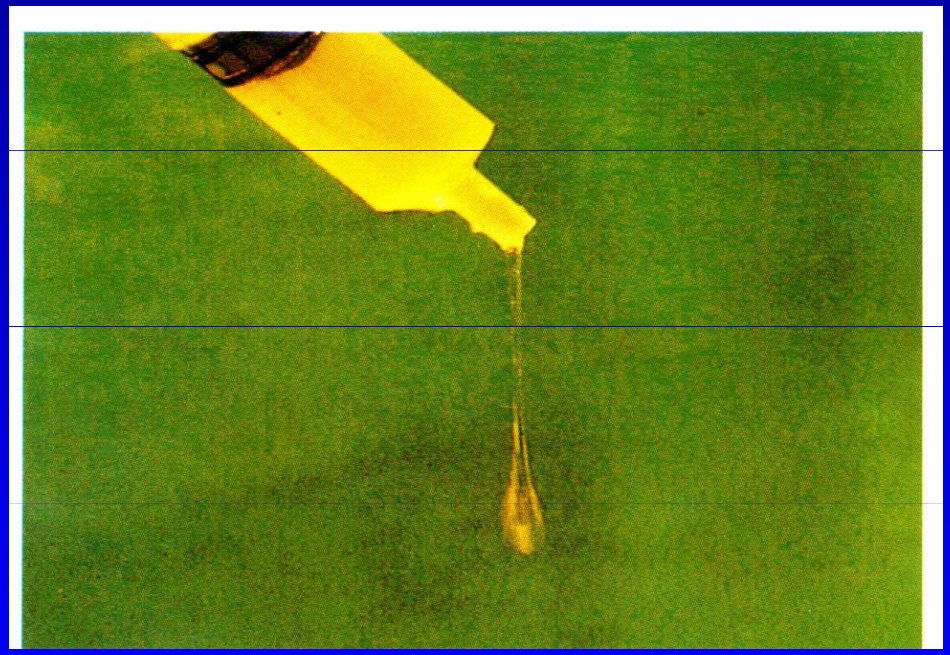
It contains:
Cells A – makrophages
Cells B – produce hyaluronic acid
Cells C – mixed cells – properties of cells A and B

Synovial fluid

Ultrafiltrate of plasma
Clear, slight yellowish
Viscous

The amount of 0,13-3,5 ml
Intracelular pressure:
-8 až - 12 ml H_2O

Proteins- only one third
of concentration in plasma



Synovial membrane

Cytology: 65/mm^3 lymfocytes, monocytes, mononucluears

Mucin = hyaluronic acid and N-acetylglucosamin
- gives viscosity

No fibrinogen

Diseases of joints

- Osteoarthrosis deformans
- Rheumatoid arthritis
- Psoriatic arthritis
- Gout
- Ancylosing spondylitis
- Septic arthritis

Onemocnění kloubů

- Systemic arthritis (Lupus erythematodes)
- Haemofilia
- Aseptic necrosis
- Osteochondritis dissecans
- Chondromatosis
- Neurogenic arthropathy
- Pigmented villonodular synovitis

Osteoarthritis

- Degenerative, slow and progressive disease of hyaline cartilage of synovial joint
- All conditions changing the structure and function of hyaline membrane and surrounding tissues lead to osteoarthritis

Osteoarthrosis deformans

- Primary (after 40 years of age)
- Secondary – the cause is known

Osteoartróza

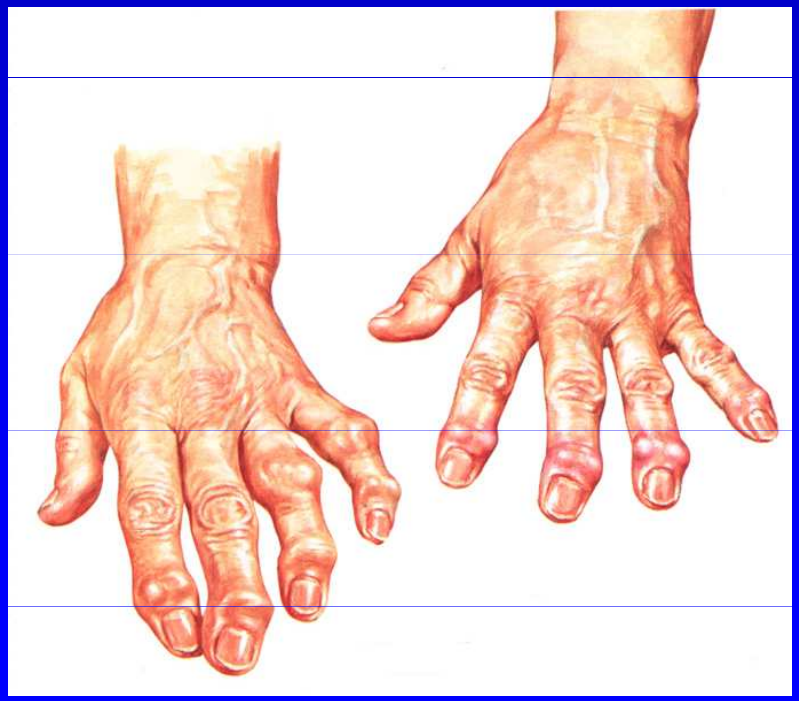
15 % of the population

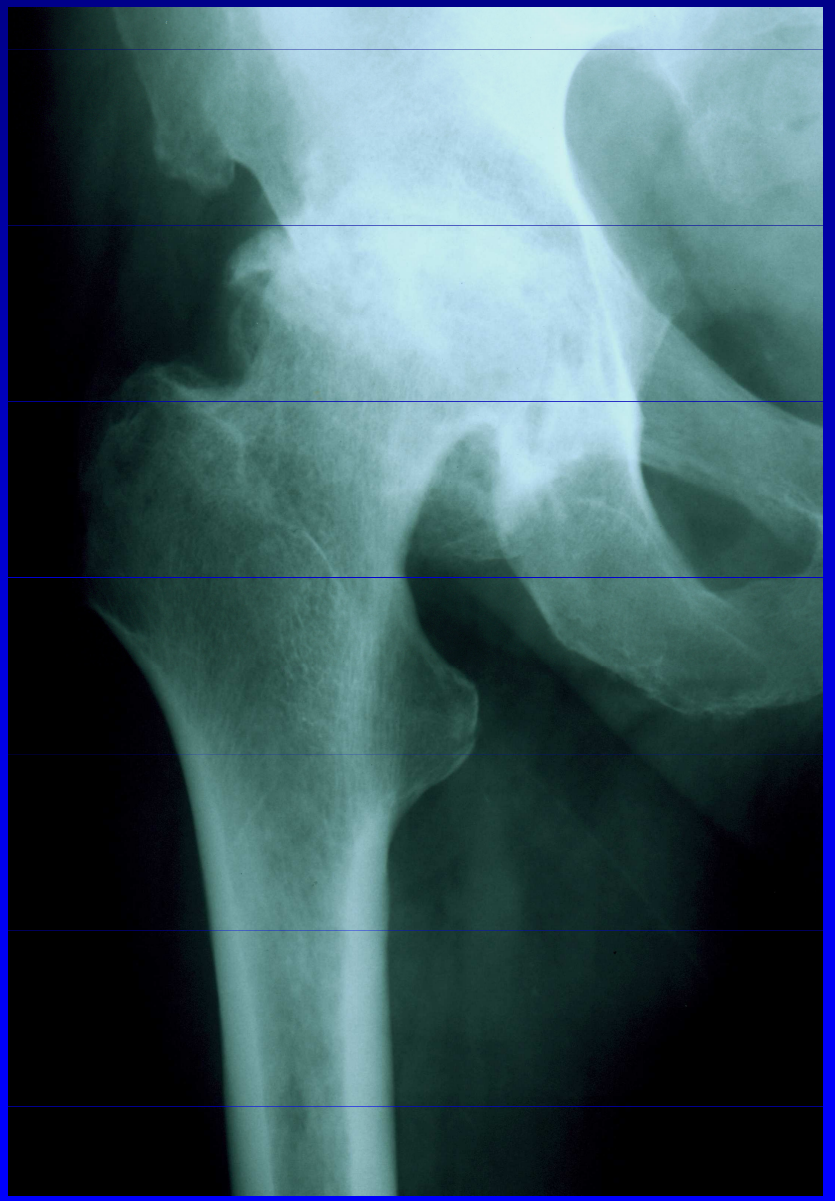
50 percent of people above 65 years

80 percent of people above 75 years

Primary O.A.

Begins over 40 y.
Small joint in hands
Cervical and lumbar spine
Hip and knee joints

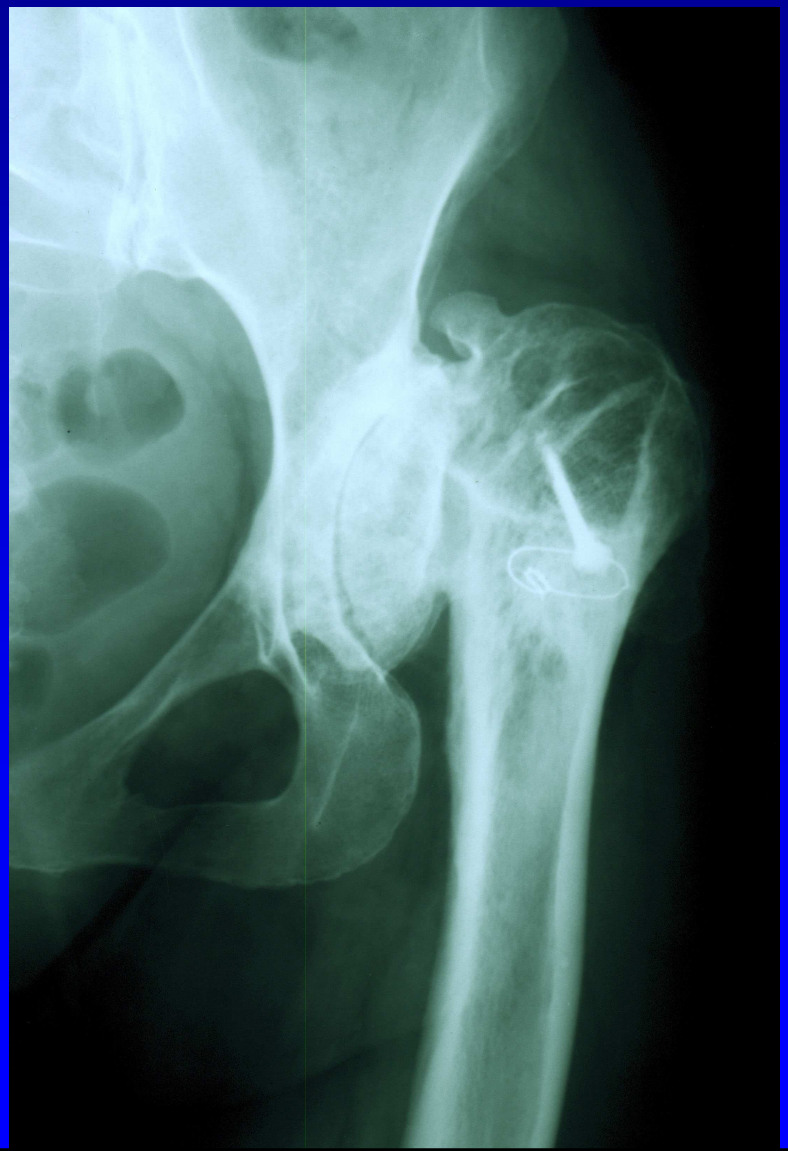




Secondary O.A.

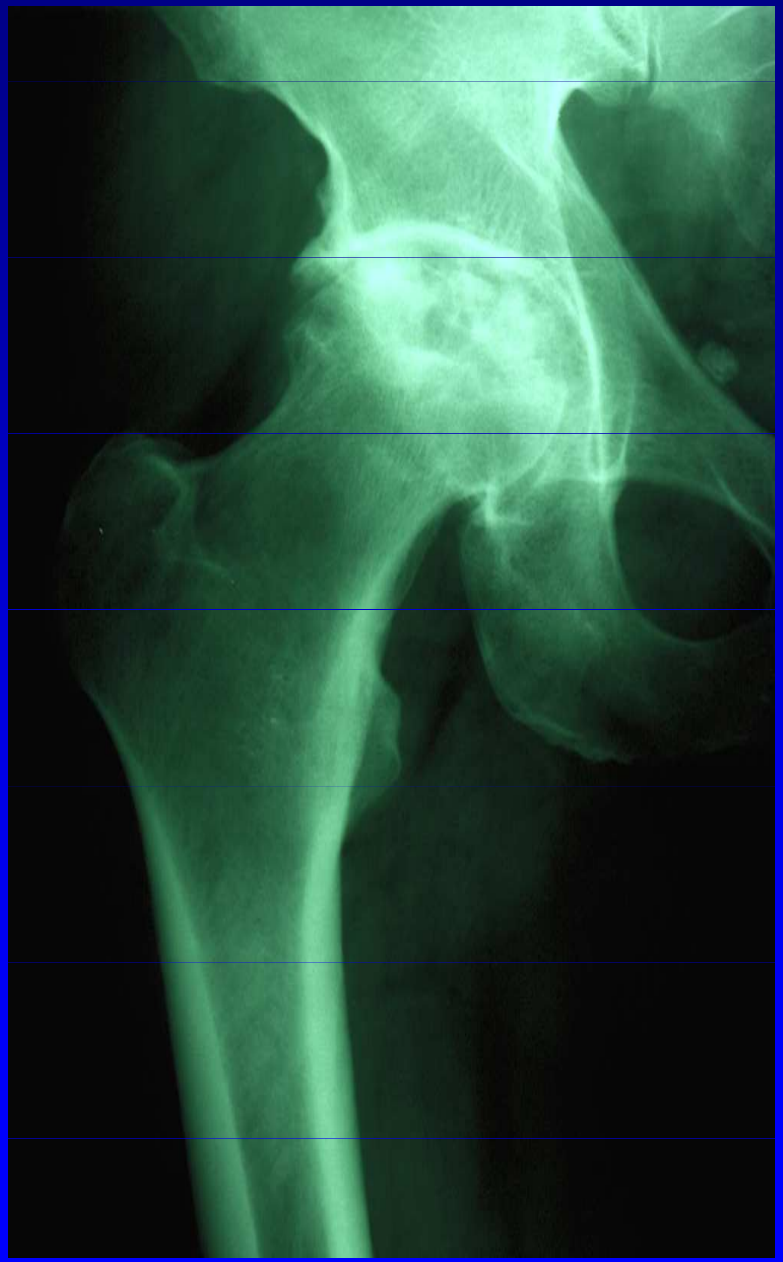
1. Mechanical factors (DDH, Perthes disease, aseptic necrosis, slipped femoral epiphysis, condition after fractures)
2. Metabolic disorders (ochronosis, gout, chondrocalcinosis, Gaucher disease)
3. Hormonal disorders (acromegaly, diabetes m.)
4. Haemofilia
5. Inflamated disorders (septic artritis, R.A.)

DDH- developmental dysplasia of the hip joint



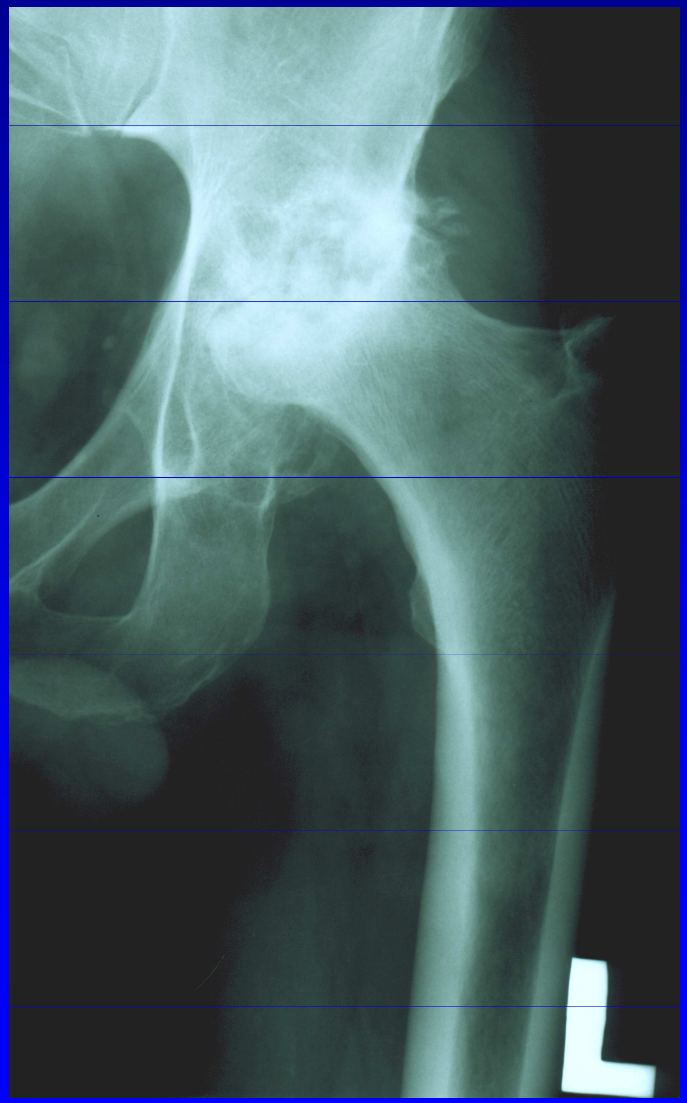
Obr. 6

Idiopatic necrosis of the femoral head



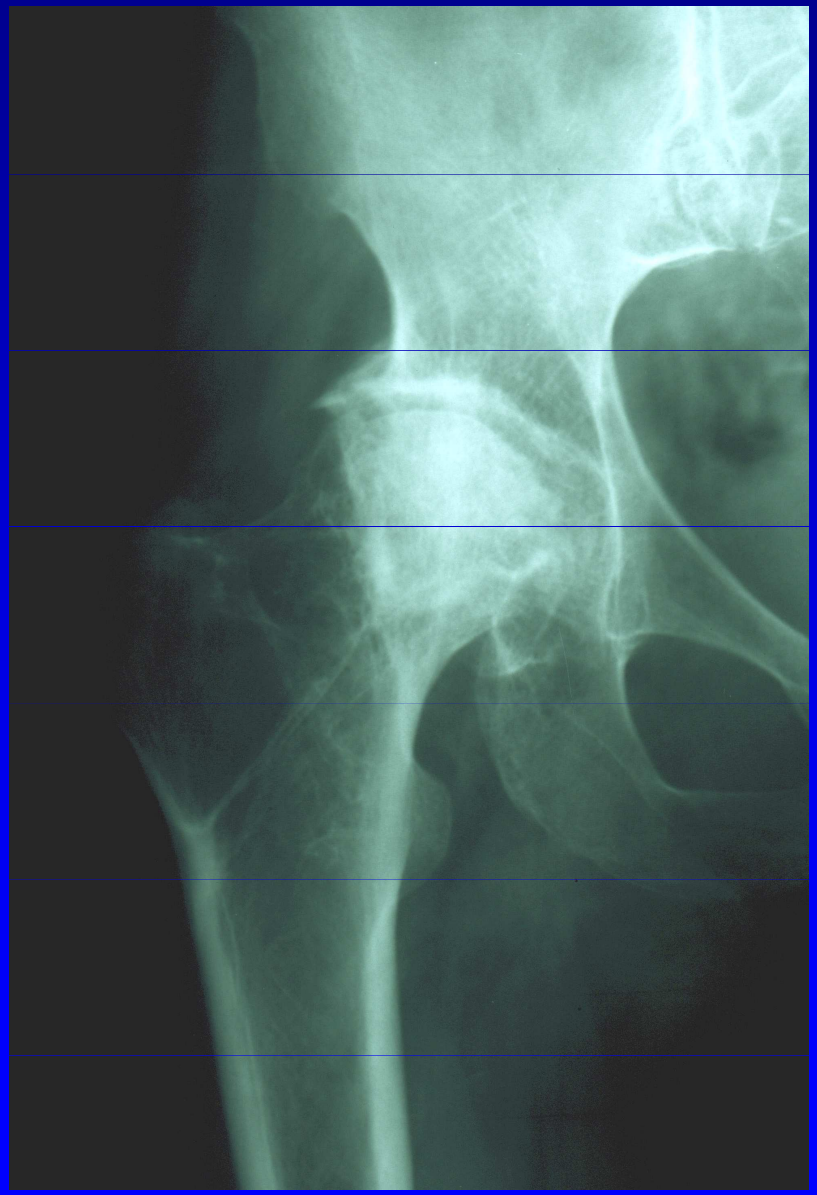
Obr. 7

Condition after Perthes disease



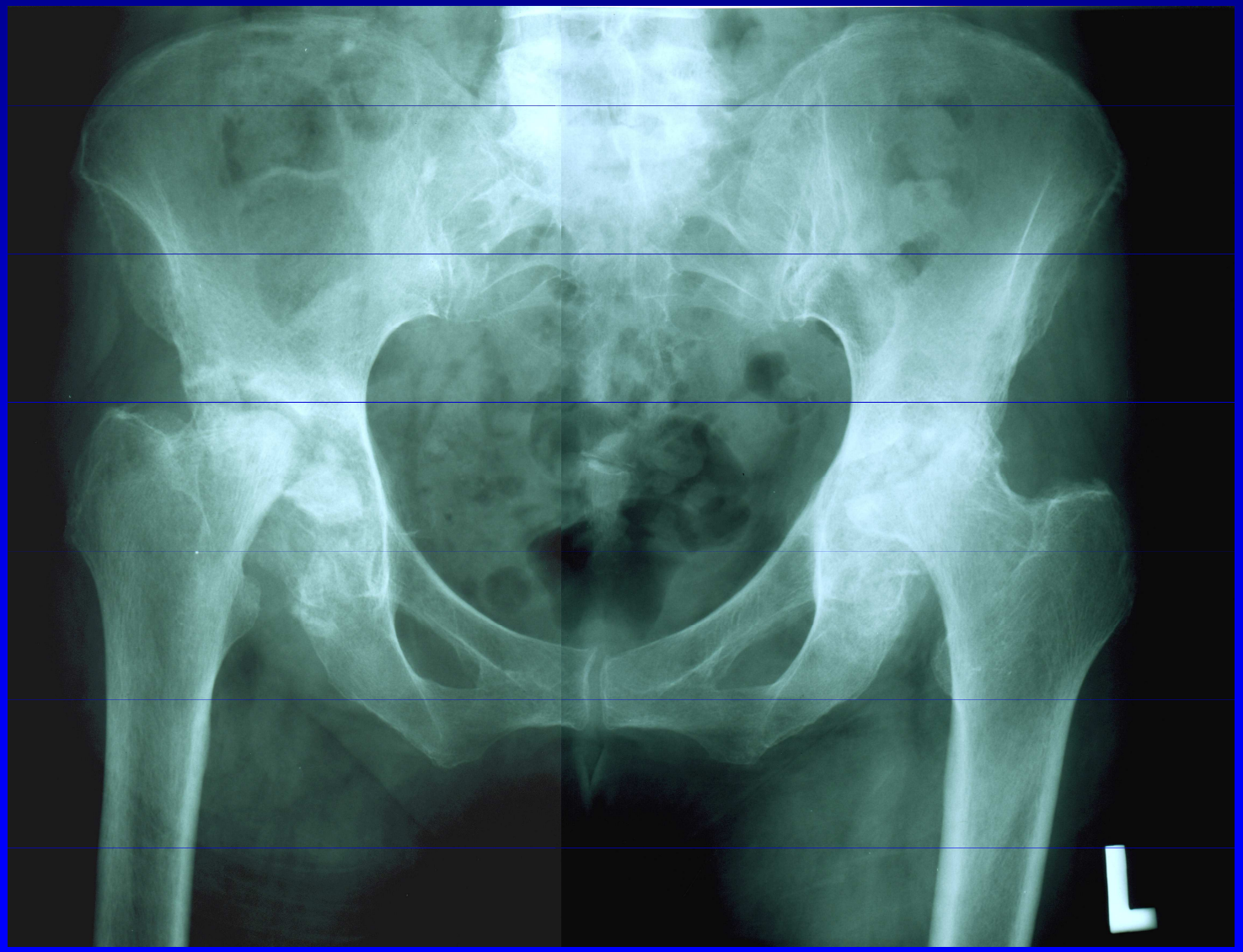
Obr. 8

Necrosis after femoral neck fracture



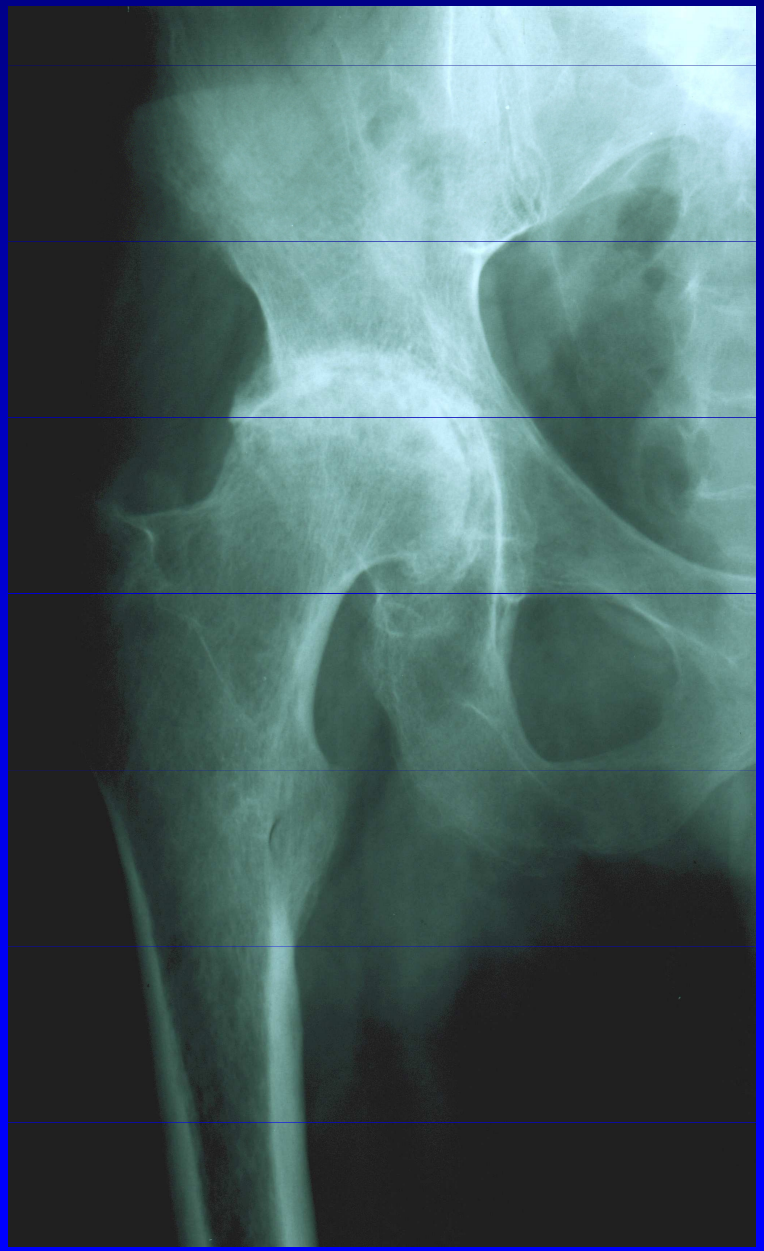
Obr. 9

Rheumatoid artritis



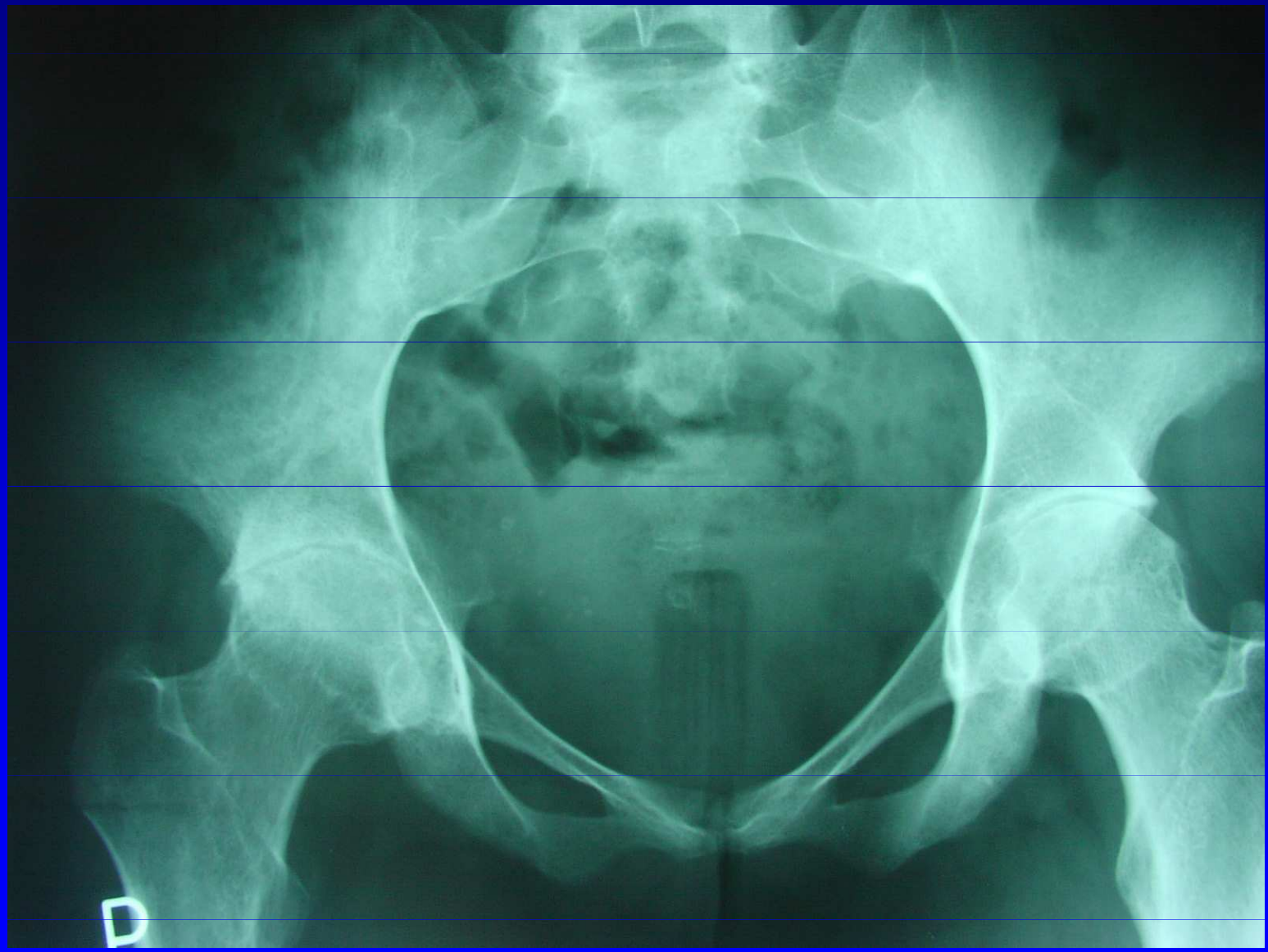
Obr. 10

Ancylosing spondylitis - hip joint



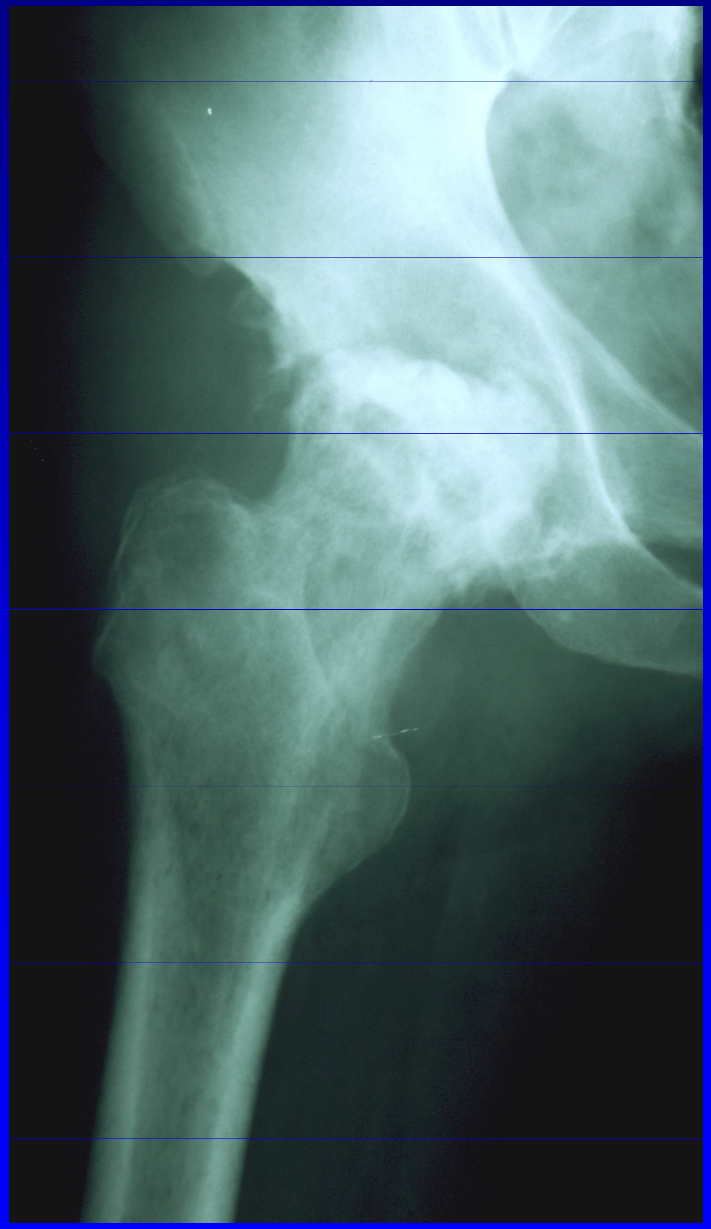
Obr. 11

Ancylosing spondylitis



Obr. 12

Septic arthritis



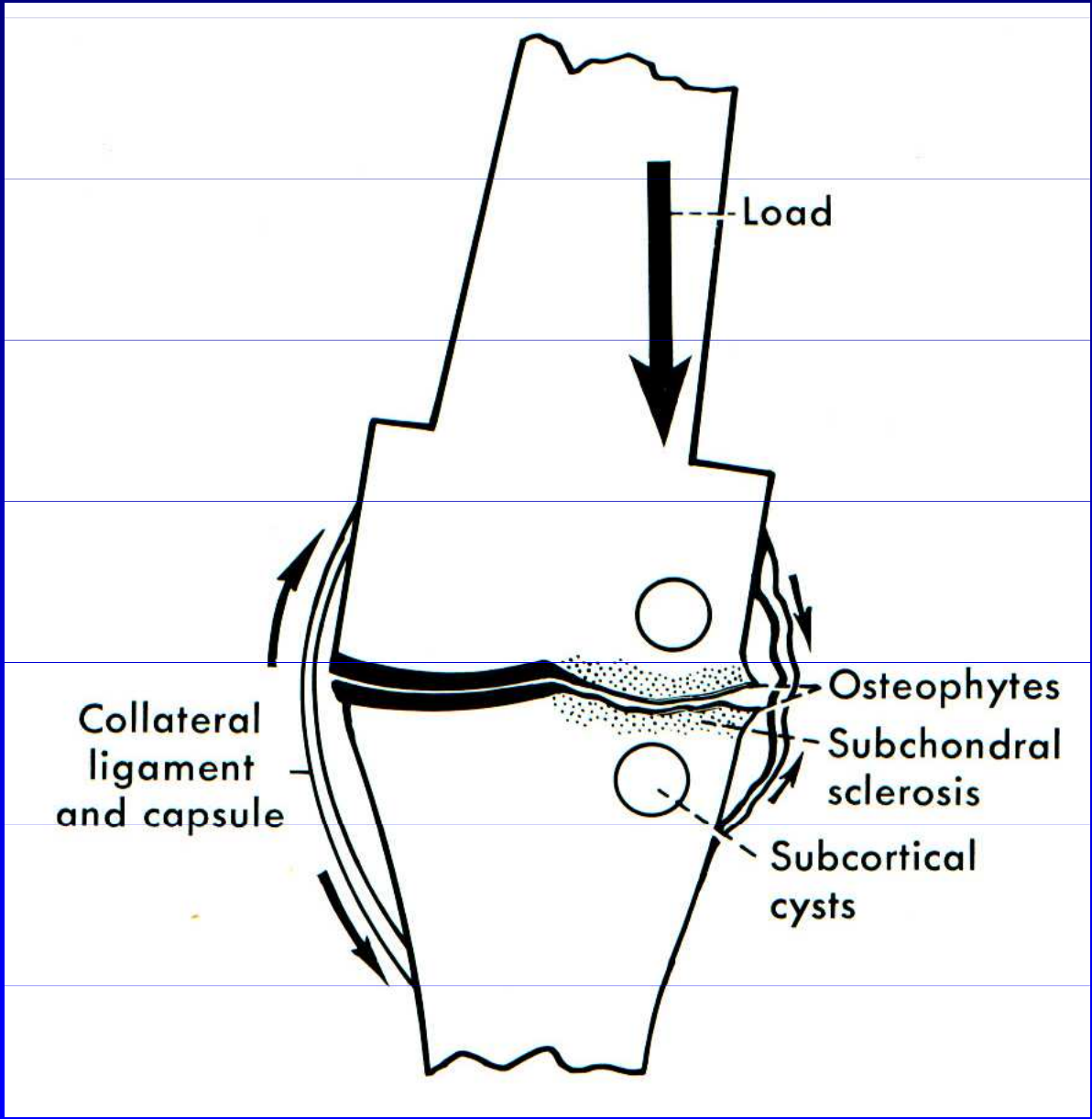
Obr. 13

Risk factors

Age over 50 years
Obesity
Mutation of gene for procollagen II (COL2A1)
- autosomal gene for Heberden´s nodes
- dominant in female and recessive in male
- female are involved twice oft than male

After 55 years – postmenopausal defecit of estrogens - O.A. is more often

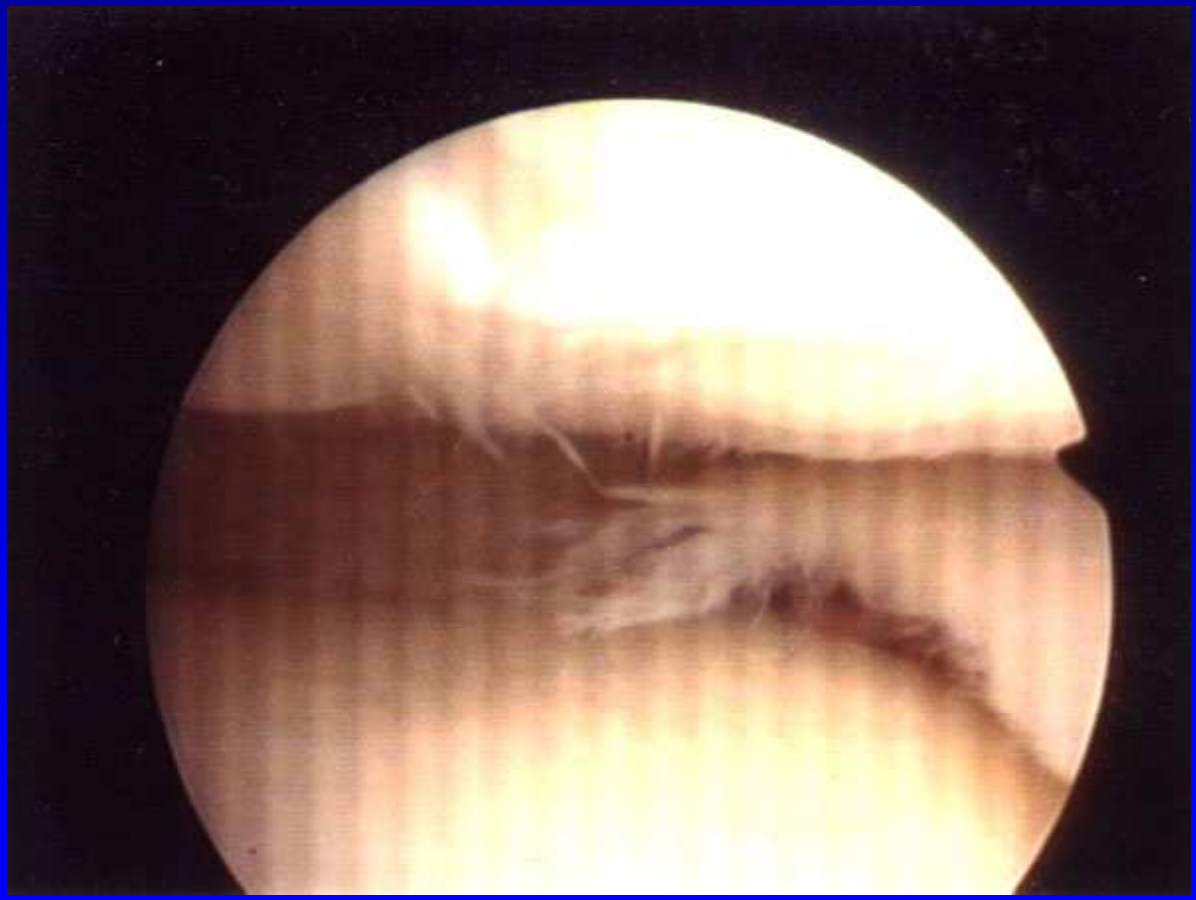
Mechanical O.A.



Obr. 14

Macroscopis changes

Cartilage is matte, soft, yellowish, fibrilations



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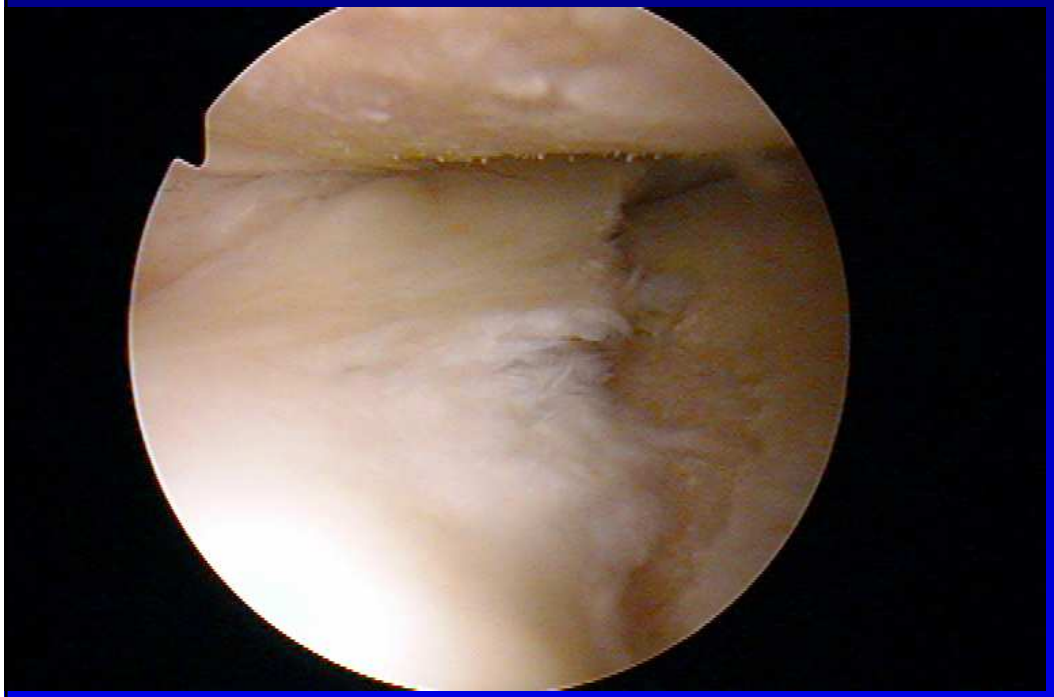
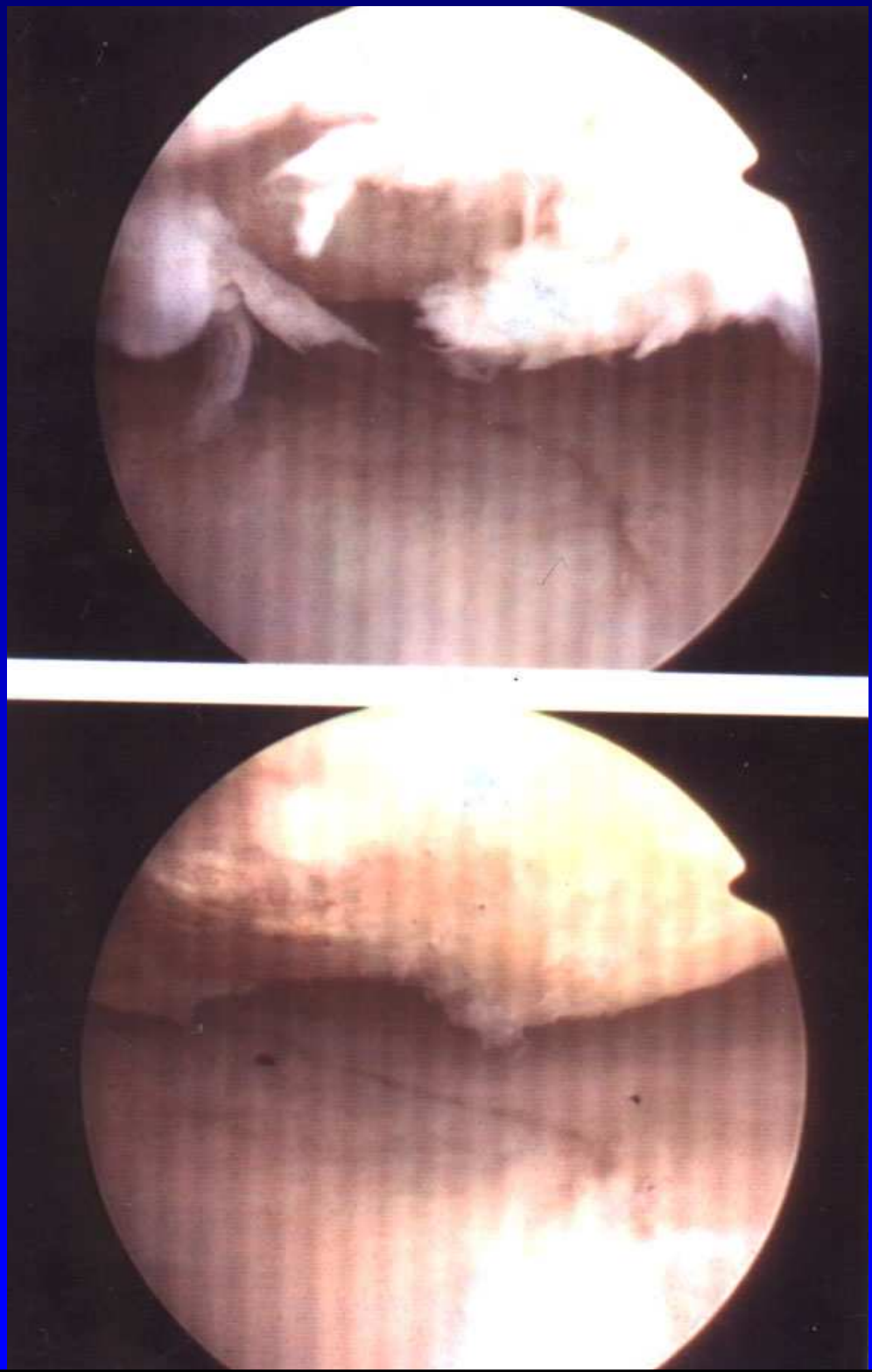
Ulcers, defects

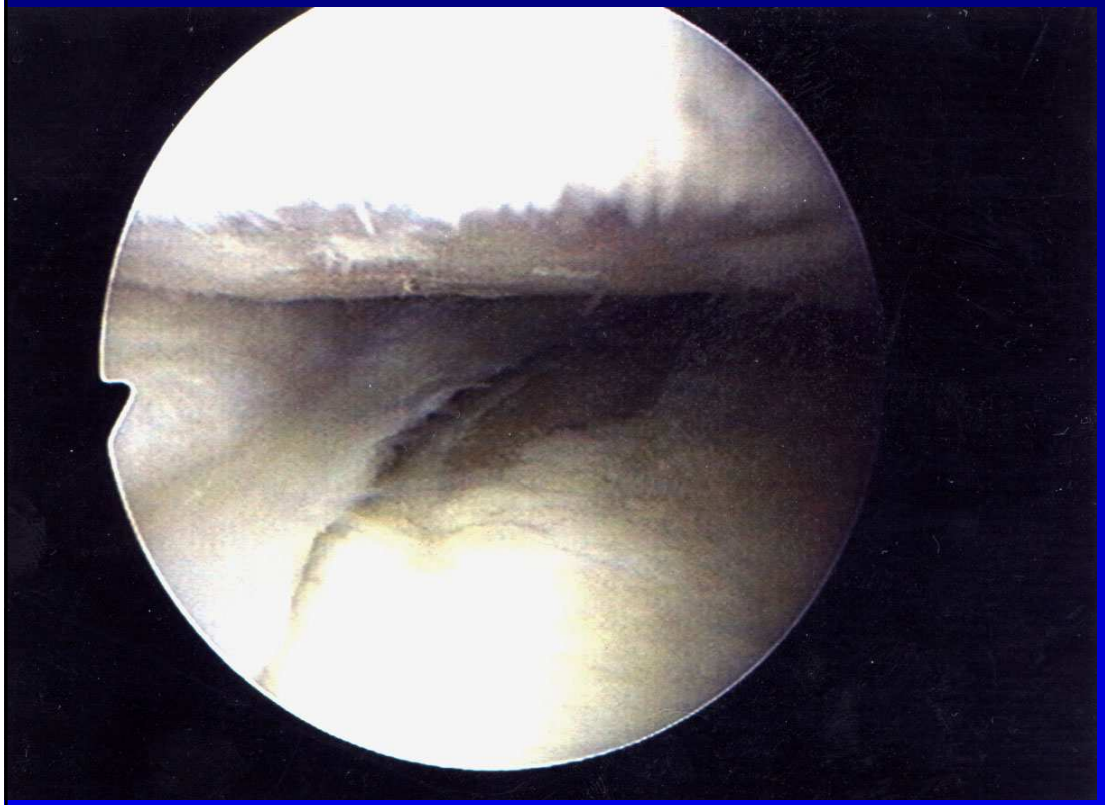


Obr. 16

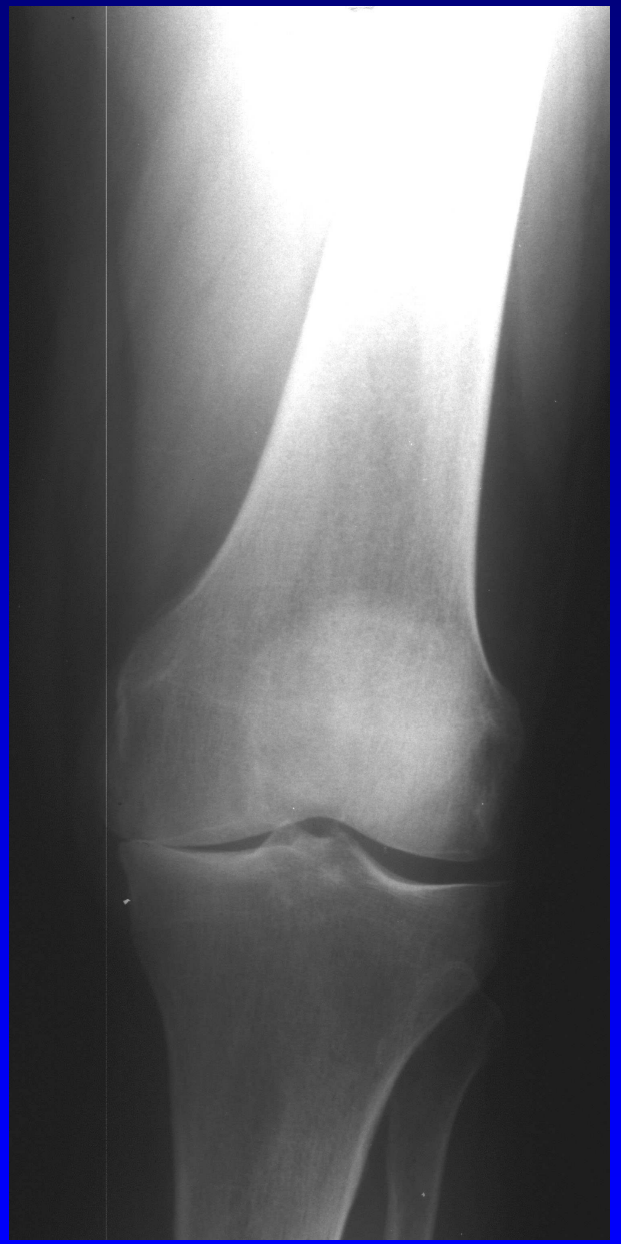


Obr. 17

Subchondral bone is sclerotic



Obr. 18



Obr. 19

Macroscopic changes

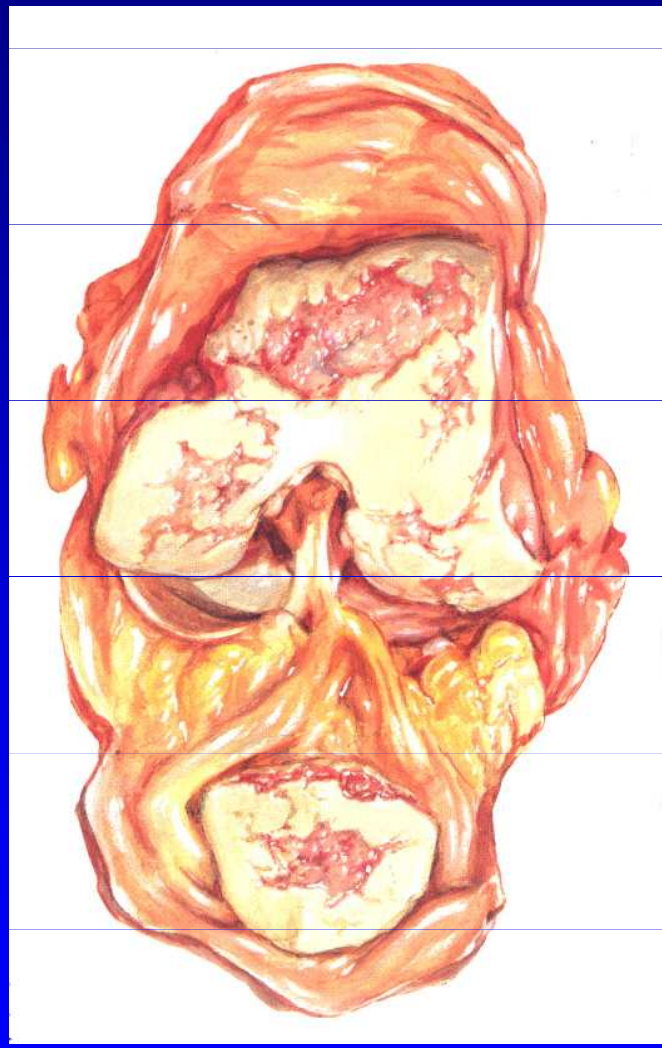
Subchondral cysts

Osteophytes

Narrowing of cartilage

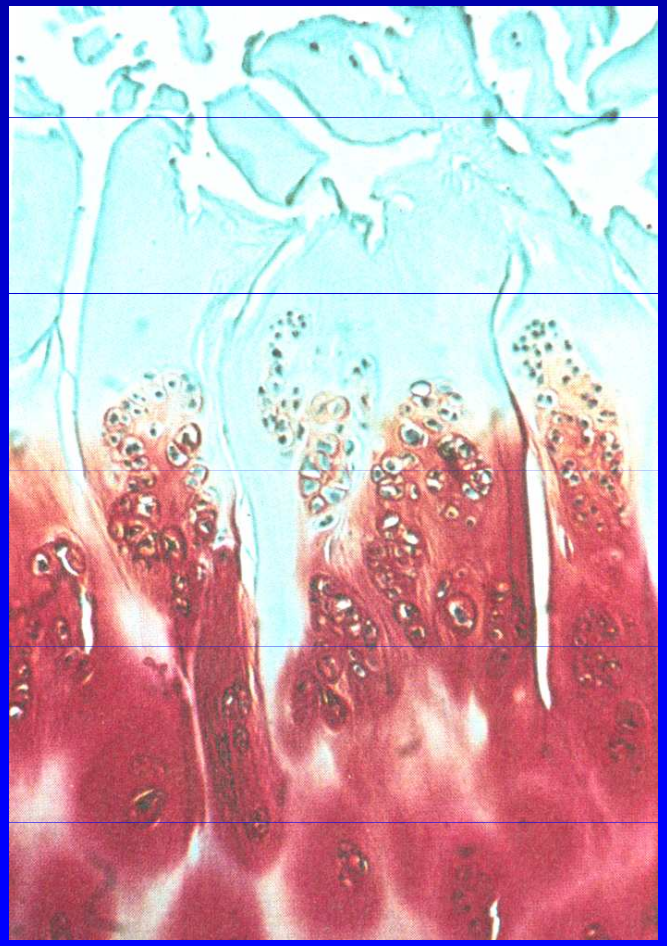
Hypertrophic synovial membrane

Loose bodies



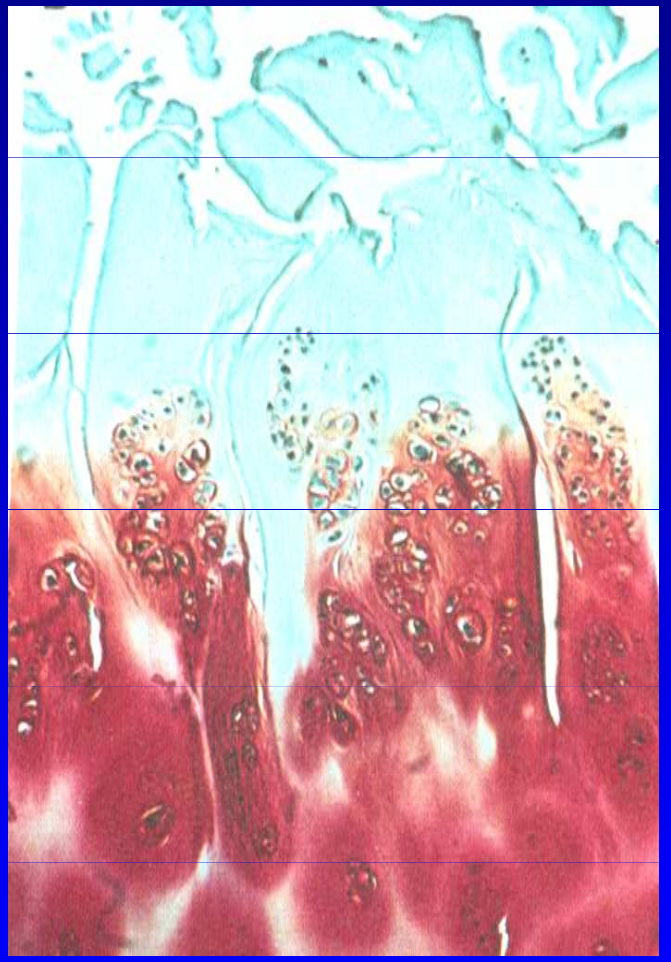
Obr. 20

Condrocytes form clusters in 10-20
Irregularities of the surface
lamina splendens is absent, fibrilations
Fissures, defects of cartilage
Collagen network is disturbed



Biochemical changes

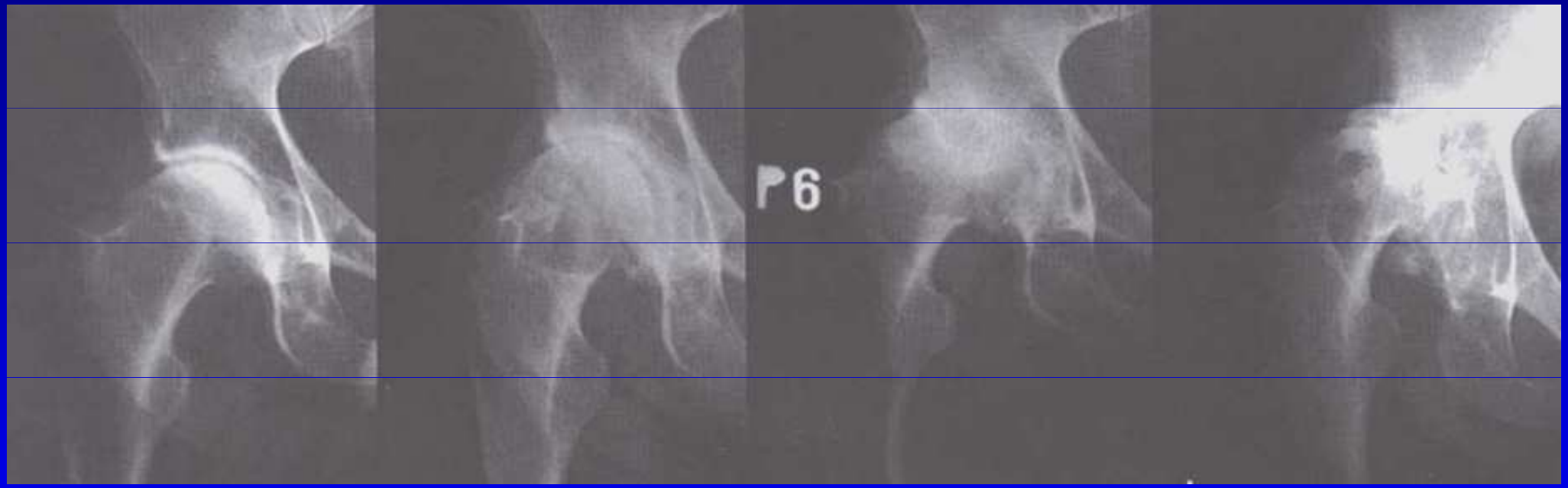
Higher amount of water
Synthesis of PG is higher
Loss of proteoglycans
Chondroitin 6 sulfate is lower
Ketaransulfate is lower
Condroitin 4 sulfate is higher



Clinical symptoms

Pain, mild, in weather changes, later is higher
Stiffness
Effusion, synovitis
Limping, difficultis in standing and walking
Muscle atrophy, joint contracture
Malalignment

I. II. III. IV.



Kellgren- Lawrence clasification I- IV.

Chondromalacia - Outerbridge

1 Softening and swelling
2 Fragmentation and fissures to 1,3 cm
3 Fragmentation and fissures above 1,3 cm
4 Erosions up to subchondral bone

Conservative treatment

Change of life style
Low weightbearing
Loss of overweight
Crutches, sticks
Physioterapy
Physical therapy

Conservative treatment

- Analgetics nonopioid (paracetamol)
- Analgetics opioid (tramadol, Durogesic)
- Nonsteroidal antiinflammatory drugs (NSAID)

NSAID

Inhibitors of cyclooxygenase 1 COX - 1 inhibitors

Acetylsalicylic acid
Ibuprofen
indometacin
piroxicam
naproxen
diclofenac
tiaprofenic acid

NSAID

Inhibitors of cyclooxygenase - 2 COX 2 inhibitors

Preferred: meloxicam (Movalis, Recoxa)
nimesulid (Aulin, Coxtral, Nimesil)

Selective (coxiby): celecoxib (Celebrex)
rofecoxib
valdekoxib (Bextra)
parecoxib
etoricoxib

SYSADOA

- Symptomatic, slow acting, antiinflamatory drugs (chondroprotectives)

Slowly acting
Long lasting efect
Stimulation of PG and collagen
Inhibition of catabolic enzymes

SYSADOA

1. systemic: glucosamin sulfate
   chondroitin sulfate

2. local: hyaluronic acid

SYSADOA systemic

GS Condro GS forte,
Proenzi 3 - glukosamin, methylsulfonylmethan,
Geladrink forte
Mobilin
DONA - glukosaminsulfát
Condrosulf 400 - chondroitinsulfát
Chondroitin 1200 (chondroitin 800 mg +glukosamin 400 mg).
Arthrofit (glukosaminsulfát, chondroitin sulfát)
Artrodar (diacerein, rostlinný původ, tlumí aktivitu interleukinů)

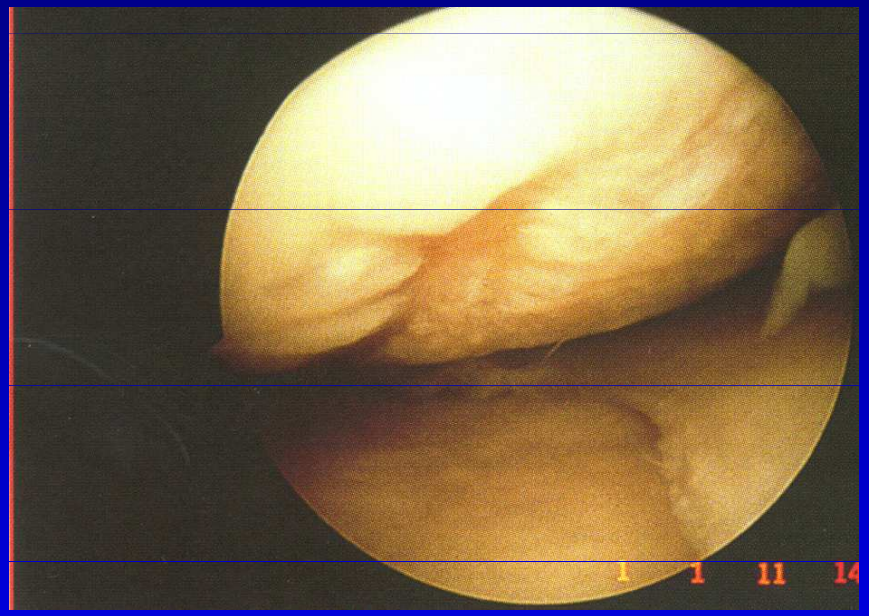
SYSADOA local
- viscosuplementation

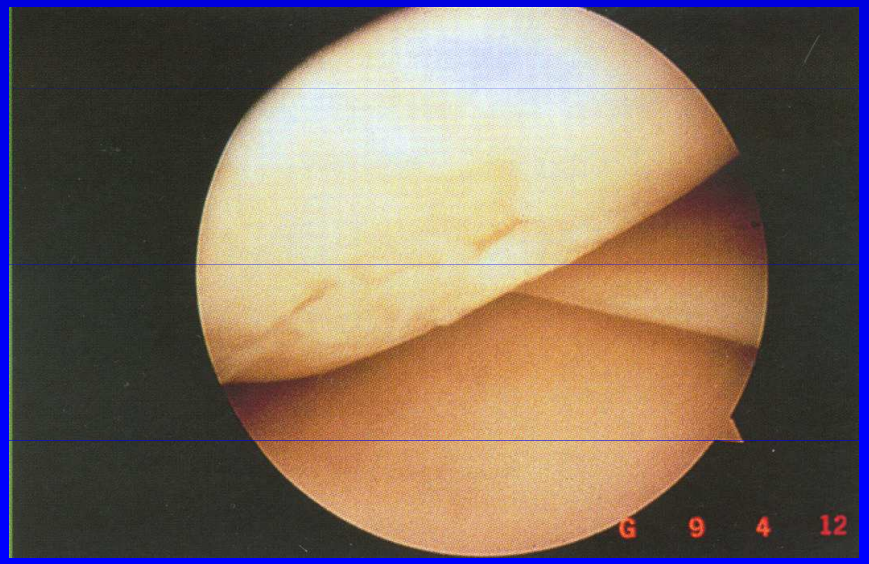
Hyalgan (hyaluronát sodný)

Synvisc - (hylan G-F 20)

Arthrease

Synovial





Local corticosteroids

Diprophos
Depo-Medrol

They influence synovitis
Do not stop progression of O.A.
Synthetic activity of chondrocytes is lower
The amount of chondrocytes and PG is lower

Operative treatment

Preventive surgery

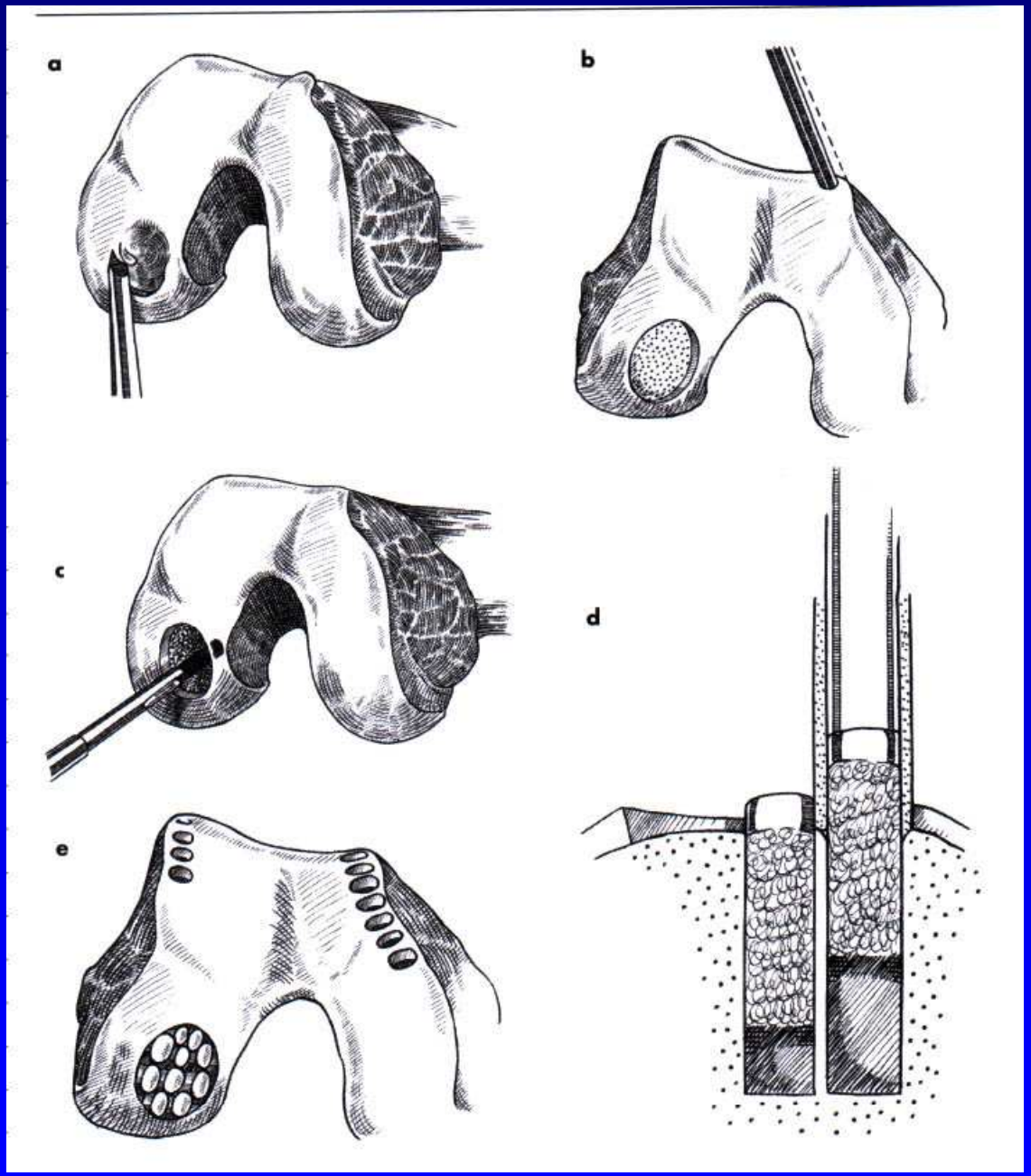
- correct treatment of intraarticular fractures
- correct treatment of ligament injuries
- correct treatment of dislocations
- correct treatment of menical lesions
- treatment of chondromalatia
- removal of loose bodies

Operační léčba

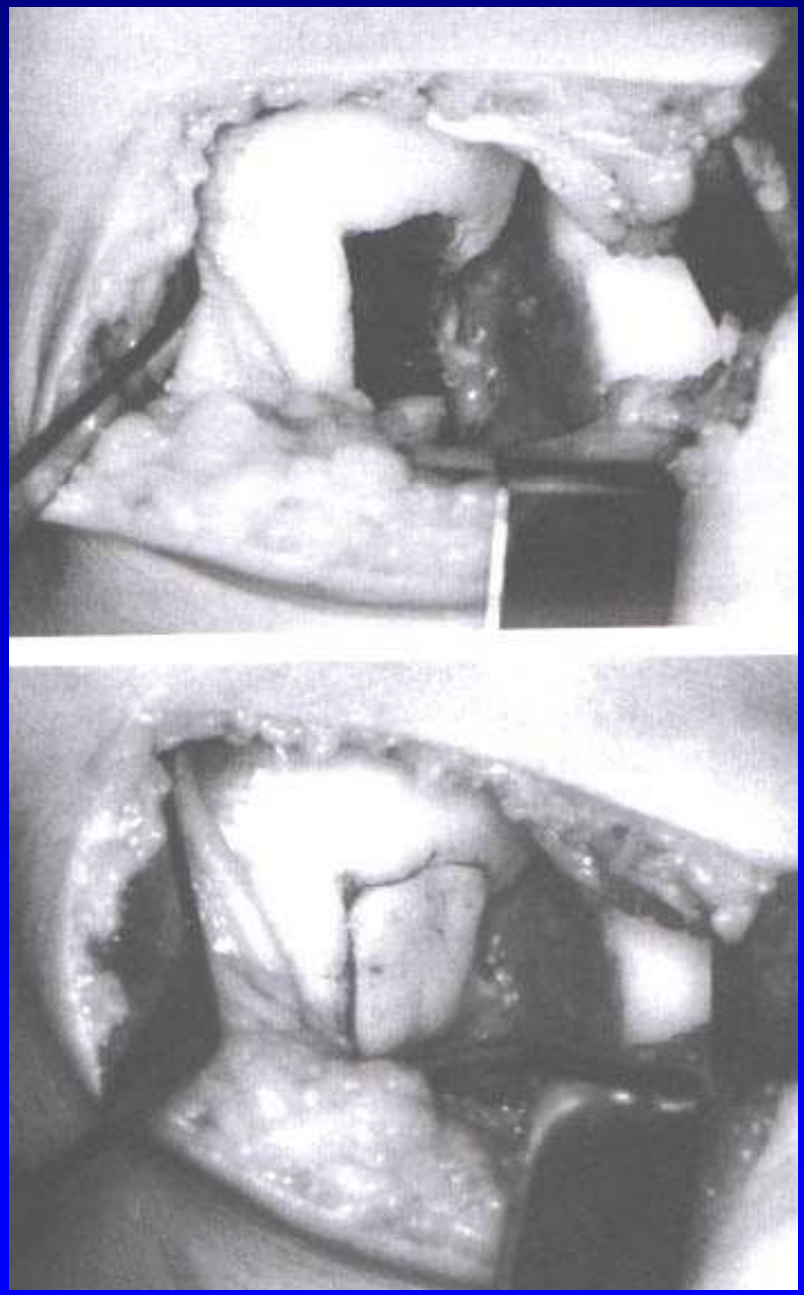
Preventive surgery

- Correction of malalignment- osteotomy
- Acetabuloplasty, shelf plasty
- Replacement of cruciate ligaments
- synovectomy, debridement, shaving

Mosaicplasty



Chondrografts



Operative treatment

Resection arthroplasty – op. sec. Keller
op. sec. Girdlestone

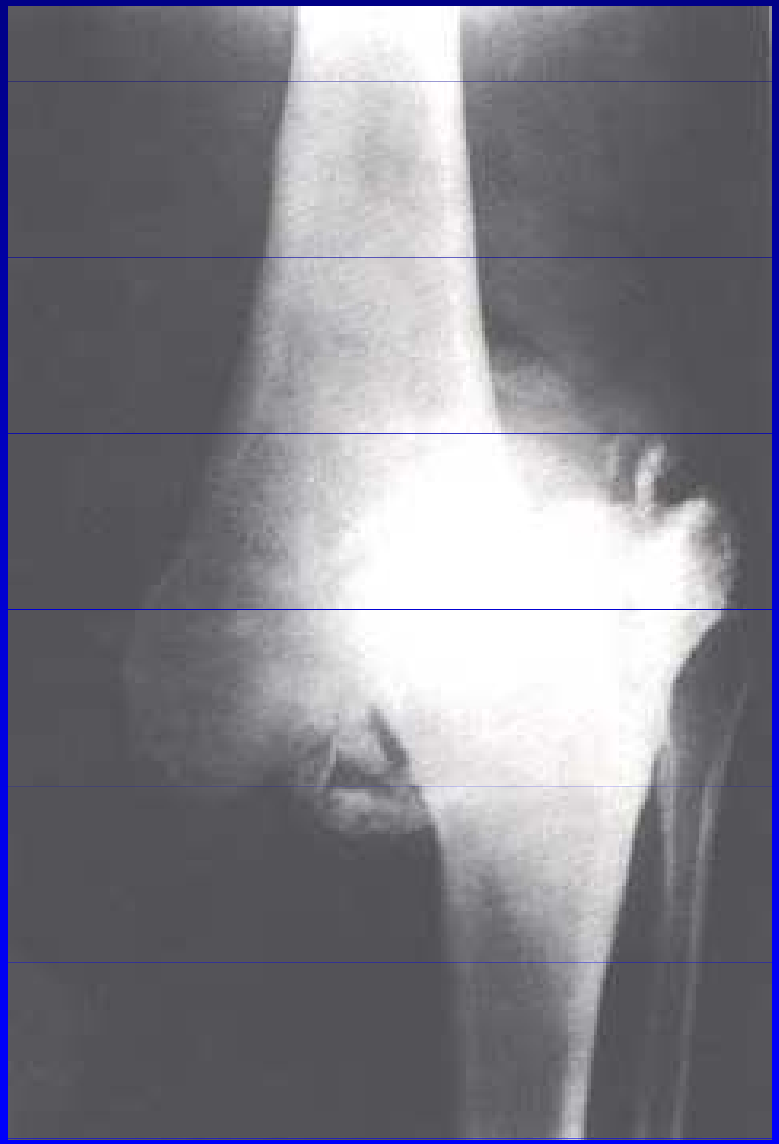
Arthrodesis

Total joint replacement

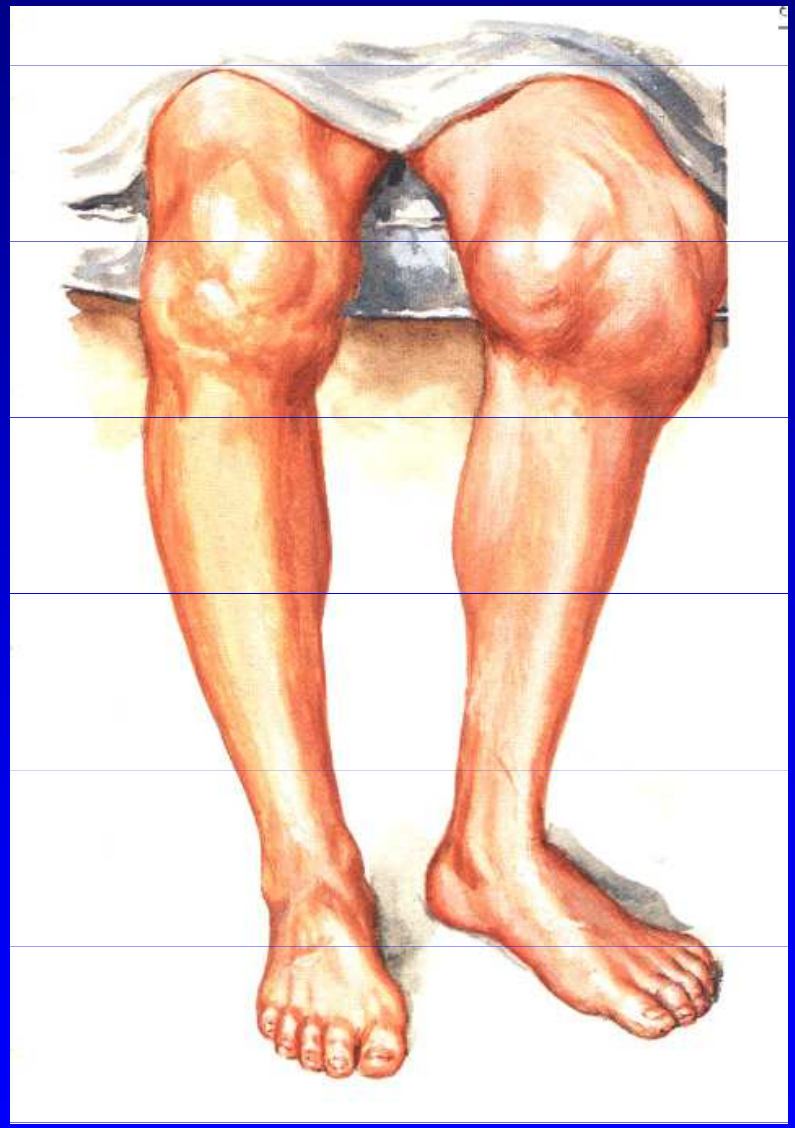
Diferential diagnosis

Rheumatoid arthritis
Ancylosing spondylitis
Psoriatic arthritis
Septic arthritis
Haemofilic arthropathy
Gout
Chondrocalcinosis
Neurogenic arthropathy

Neurogenic arthropathy

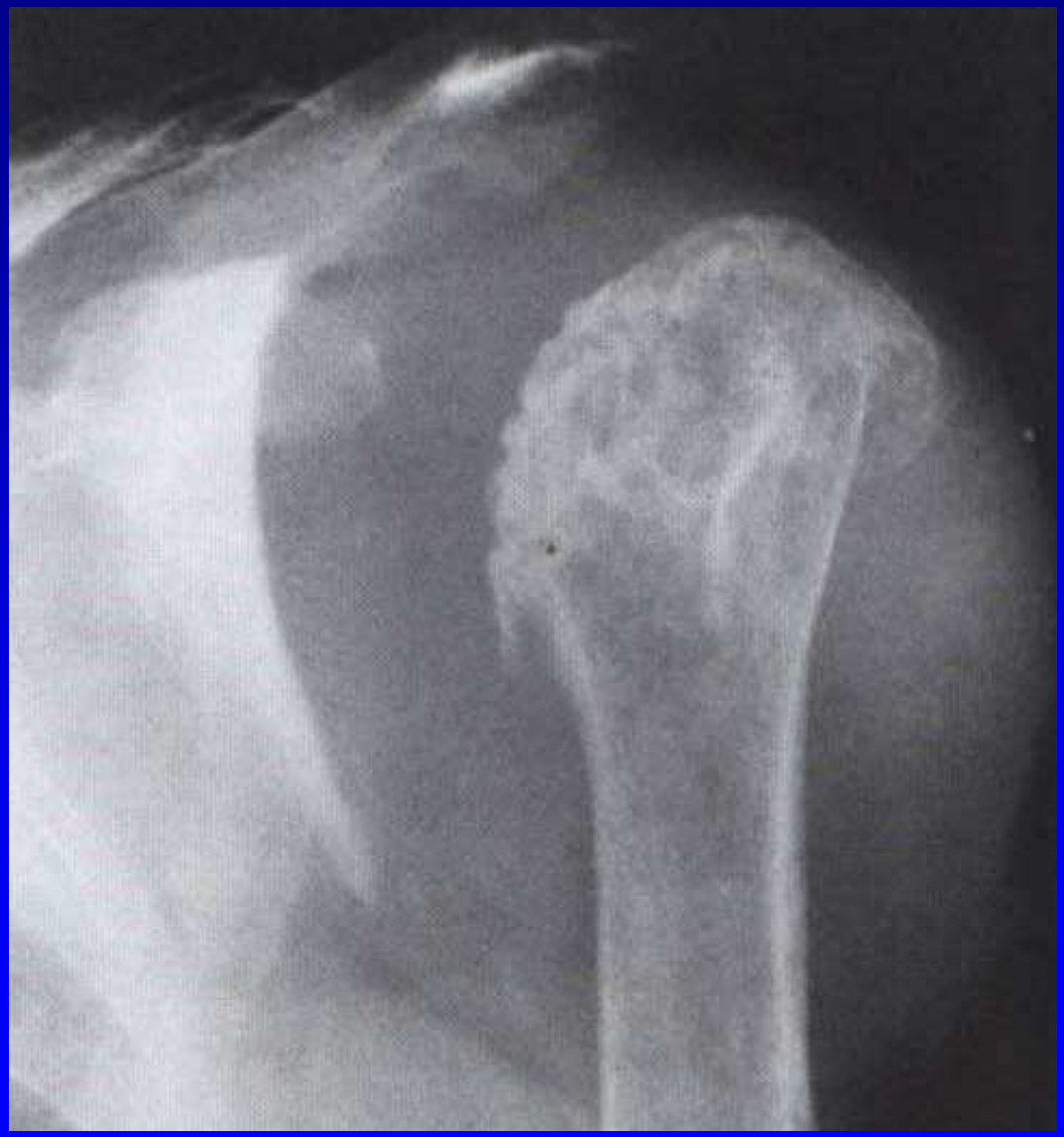


Obr. 30



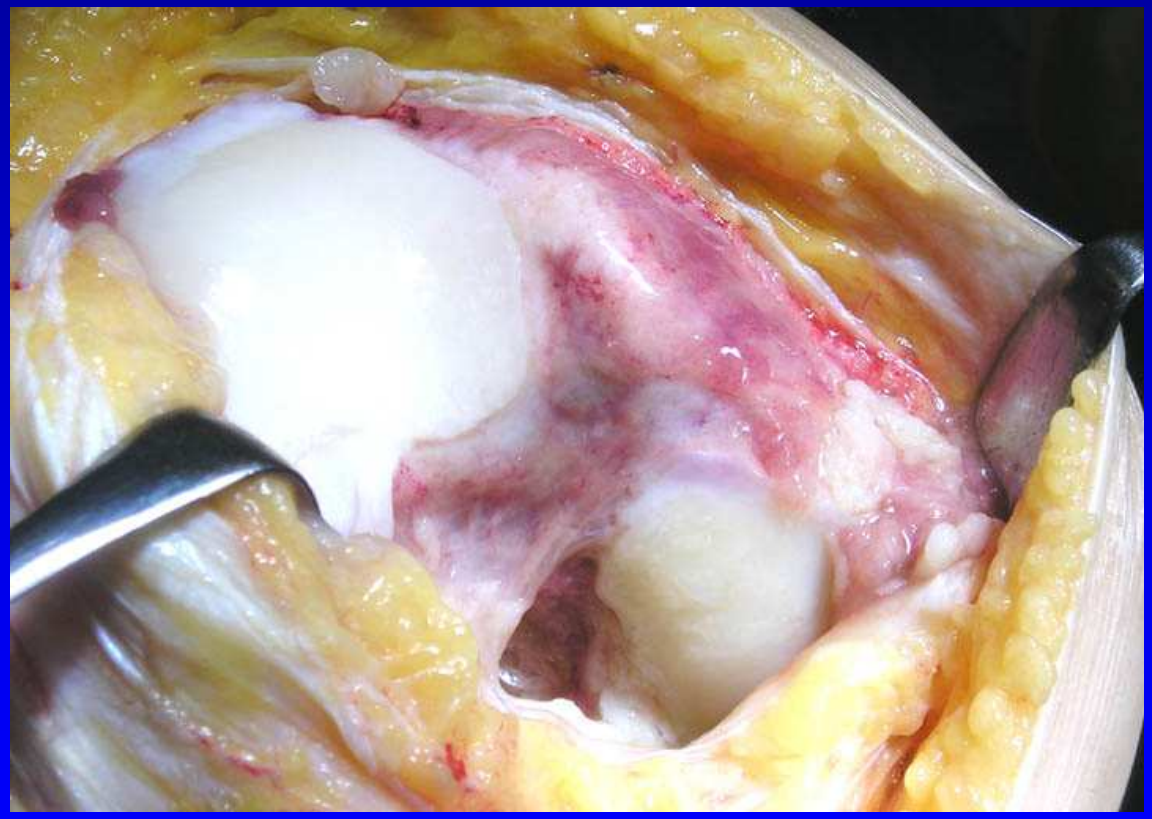
Obr. 31

Neurogenic arthropathy

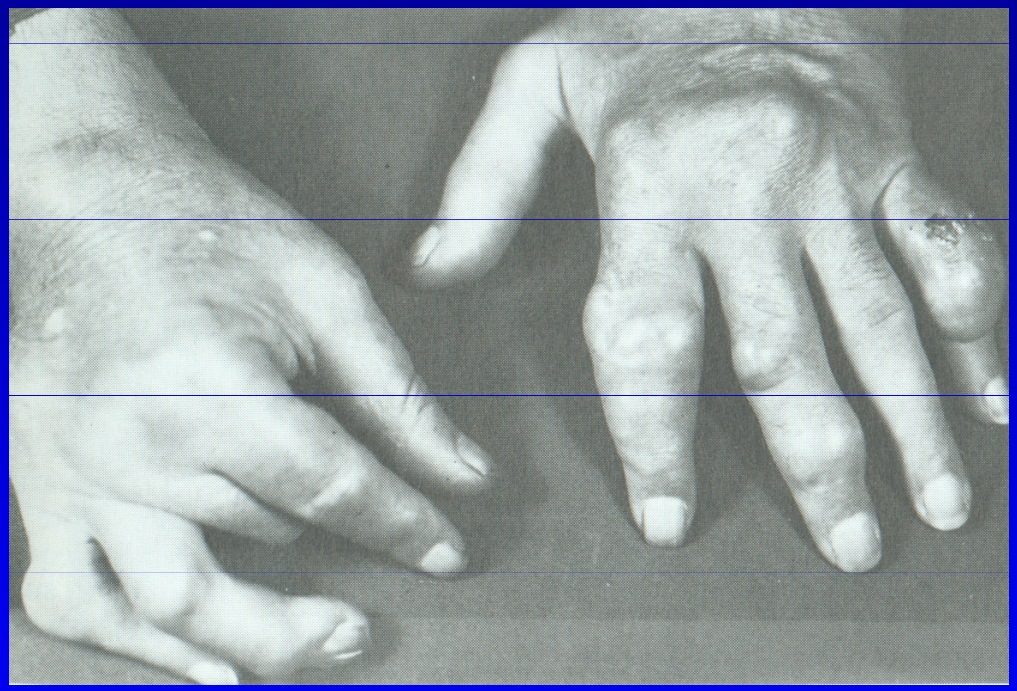


R.A.

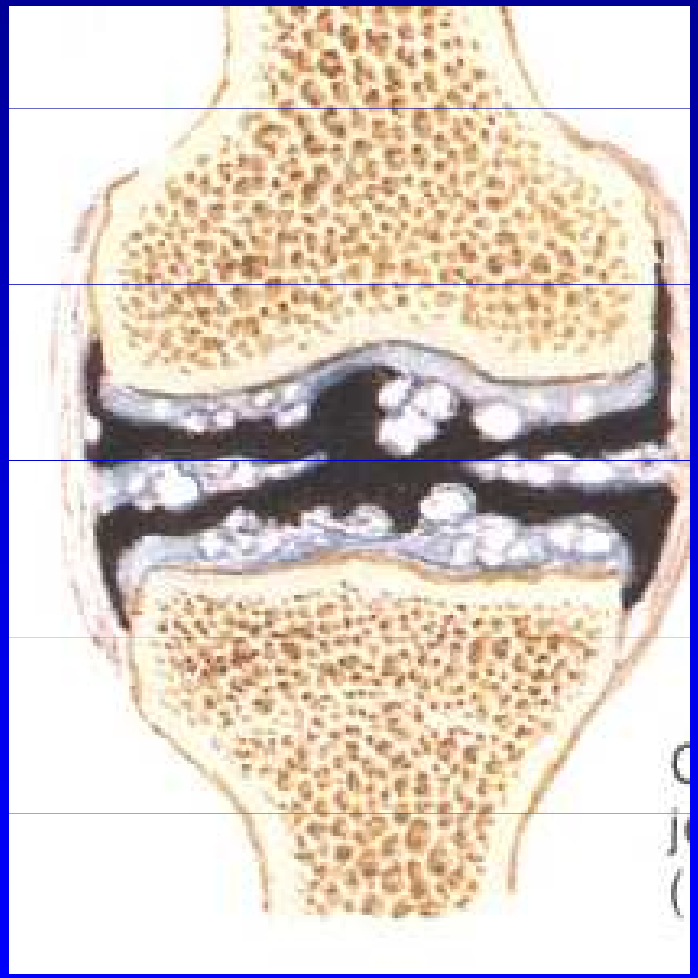
- R.A.
- Juvenilní R.A.
  - Still´s disease



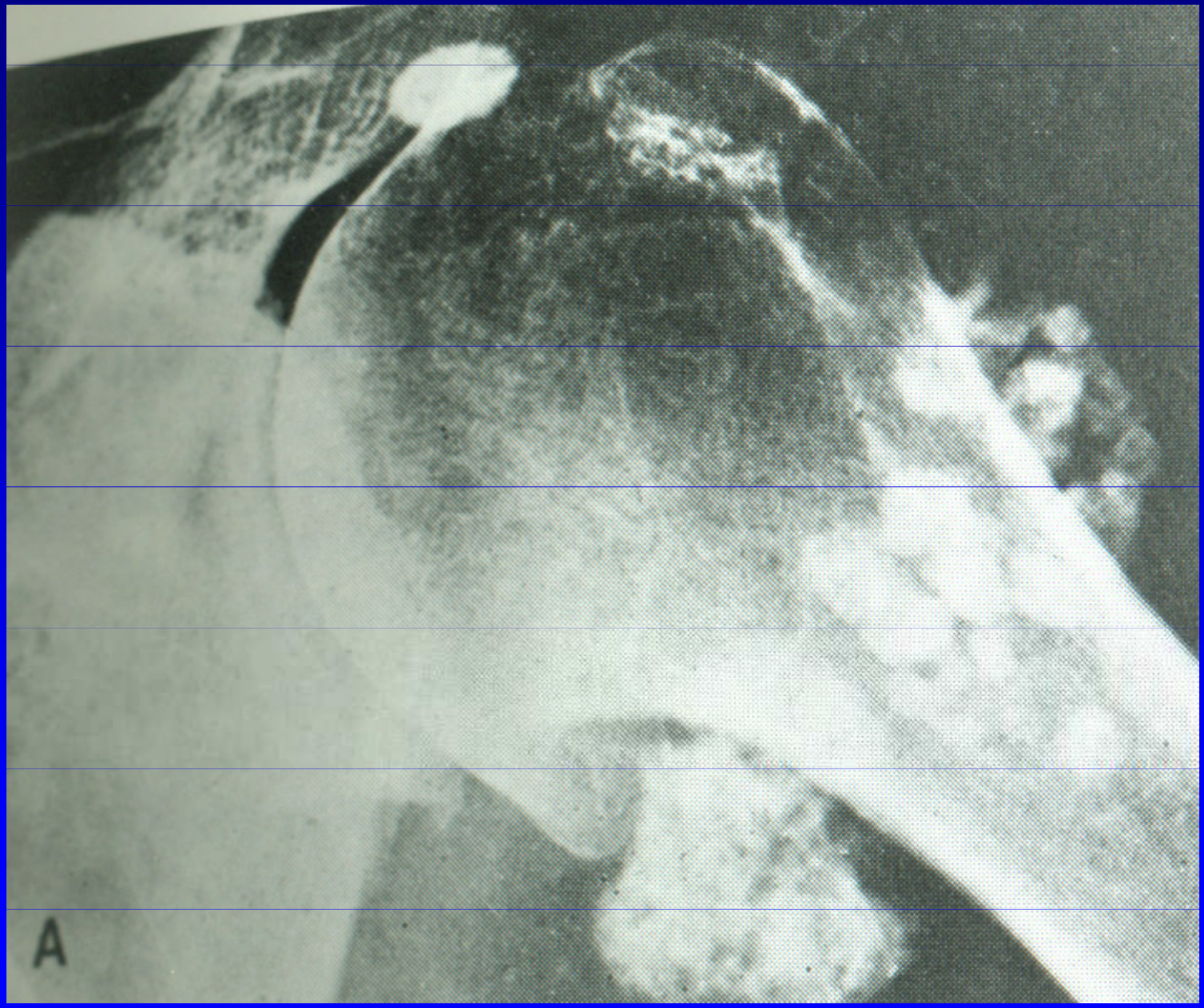
Gout



Chondrocalcinosis



Synovial chondromatosis



Septic arthritis