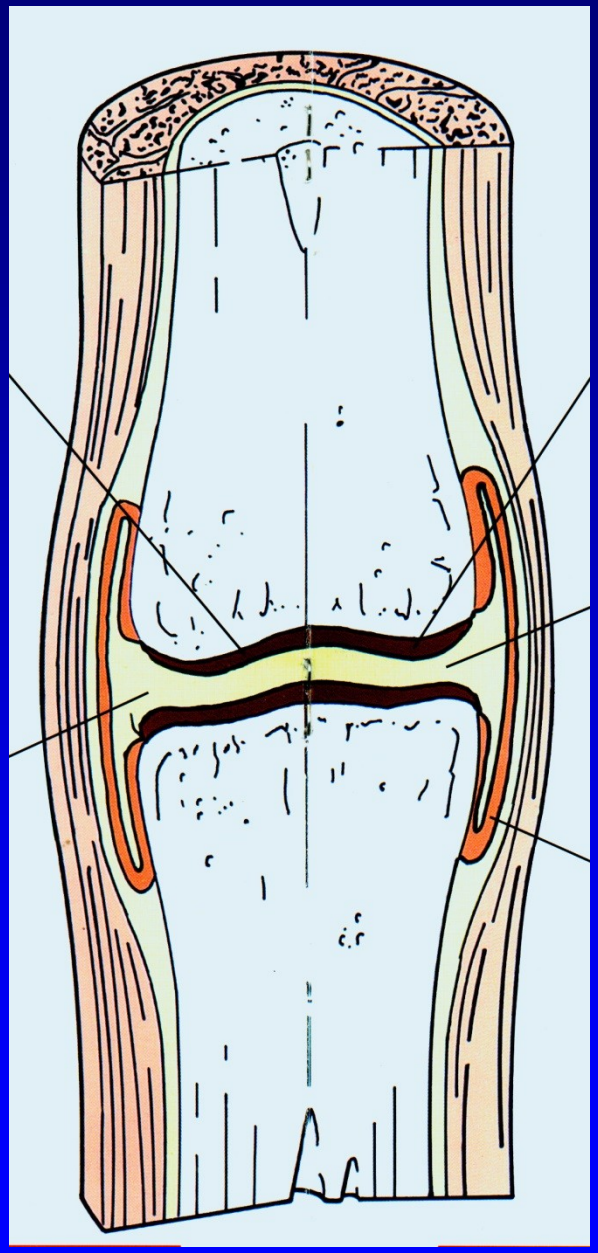
Osteoarthritis

Z. Rozkydal

Synovial joint

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



Hyaline cartilage

Chondrocytes

Matrix – intercelullar mass:

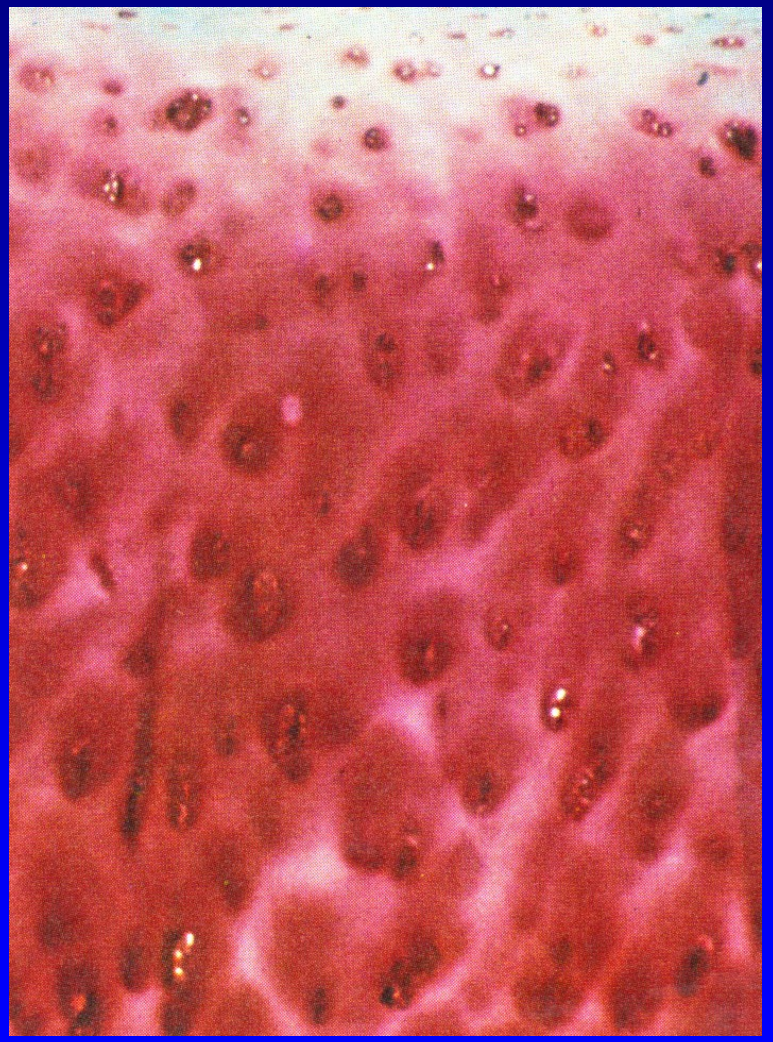
Fibrilar structure - collagen

Proteoglycans

Proteins of noncollagen nature

Hyaluronic acid

Water – 70 volume percent

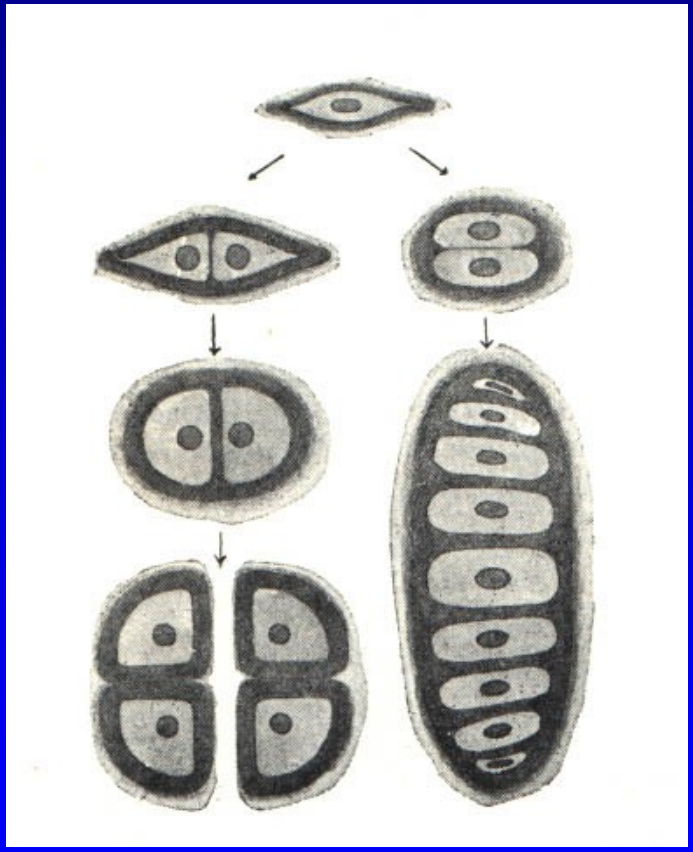


Hyaline cartilage

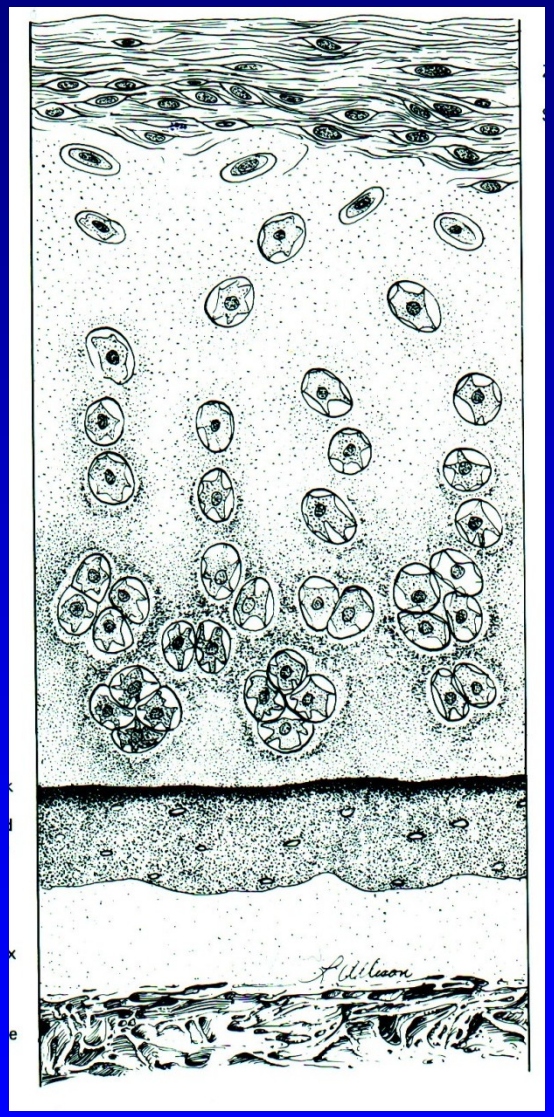
Chondrocytes- 2 percent of volume

Localised in lacunes of matrix

Isogenetic groups 2-8 cells
from one mother cell



Hyaline cartilage - layers



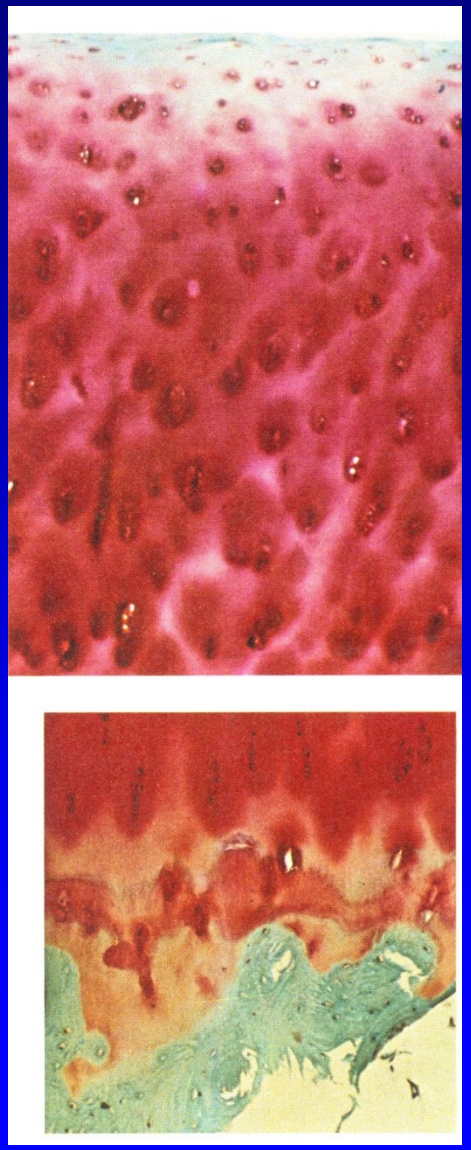
Superficial

Middle

Deep

Zone of calcifying cartilage

Bone



Collagen

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

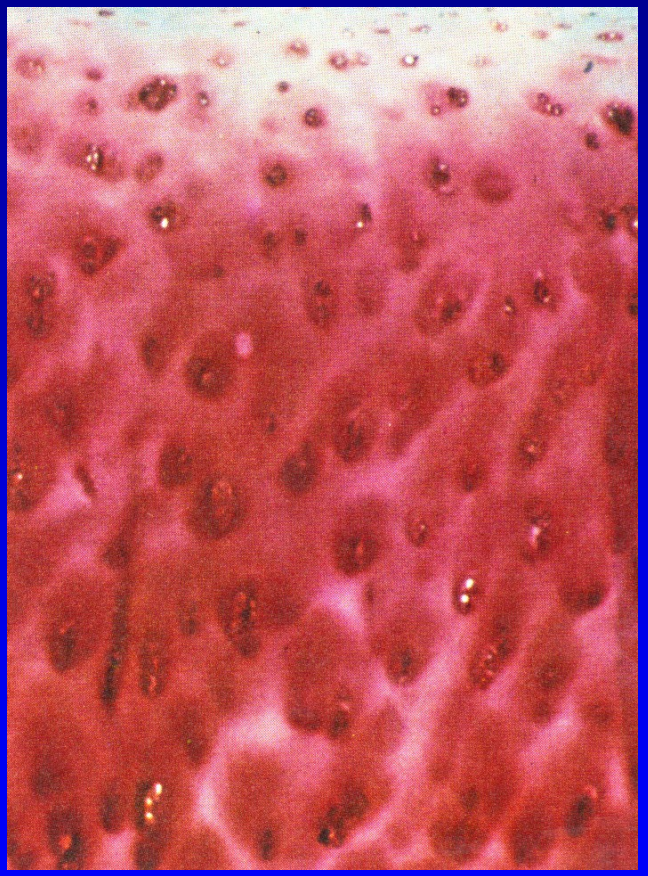
Chains form fibrils
Fibrils form a three dimensional network
Paraler to the surface
In deep layers in columns

Proteoglycans- PG

They are high hydrophylic
- elasticity !!

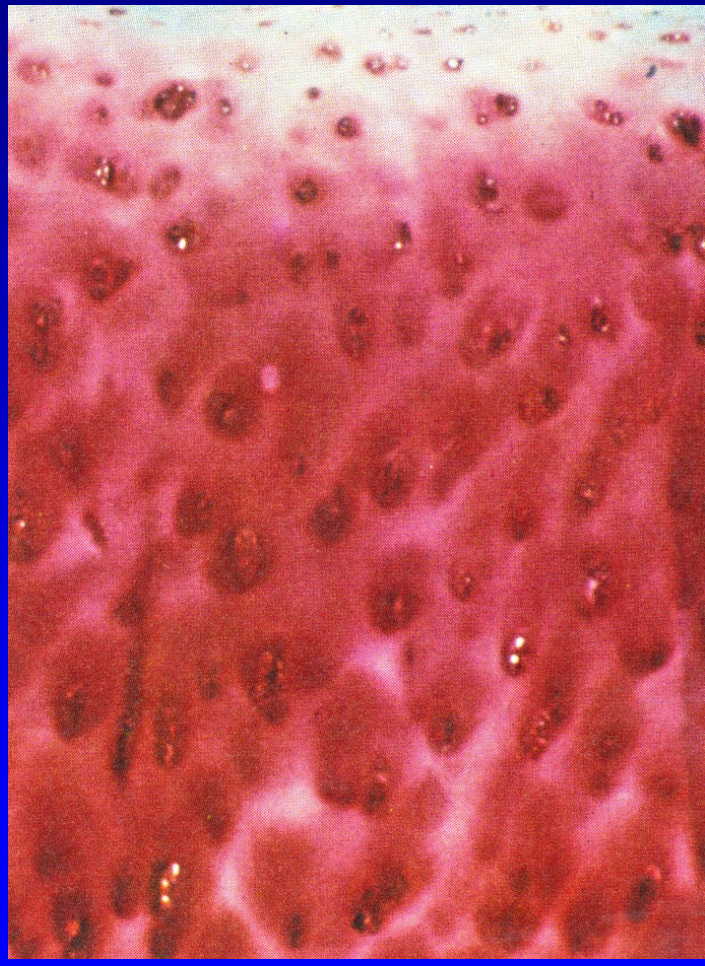
Large PG - glukosaminoglycans:
Chondroitin 6- sulfate
Keratansulfate
Chondroitin 4- sulfate

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- HA

HA + proteoglycans + collagen - intercelullar mass

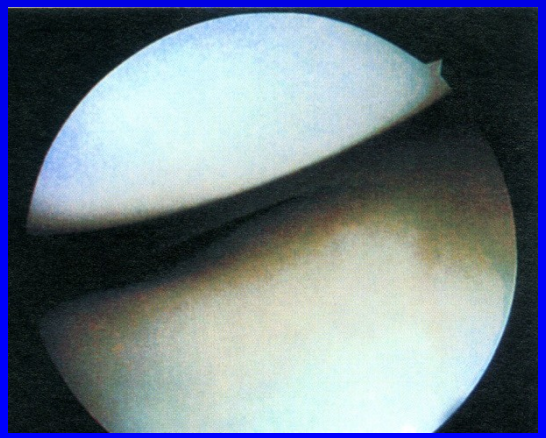
Hydrophylic, maintains homeostasis

Responsible for lubrication of the joint

Promotes transport of nutritiens into the cartilage

Gives the cartilage elastic resistance

Gives rheologic properties to synovial fluid



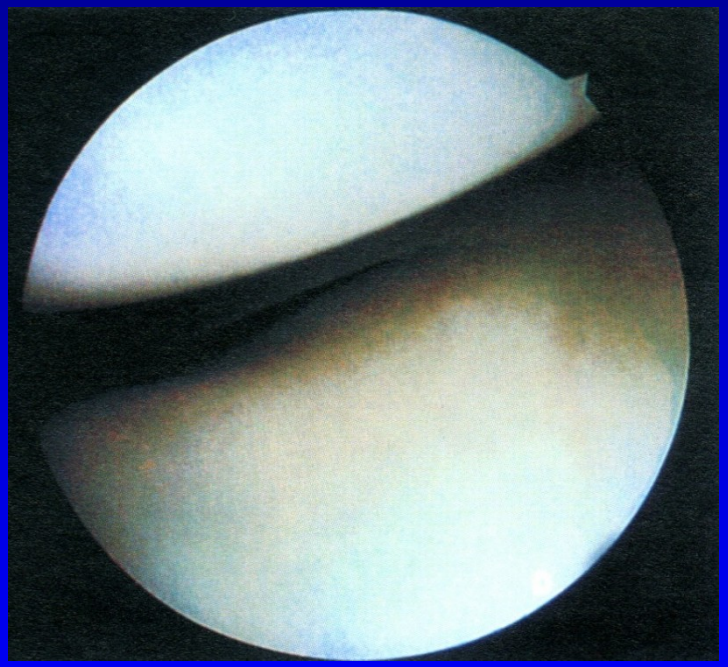
Hyaline cartilage

High volume of water gives resistance in pressure

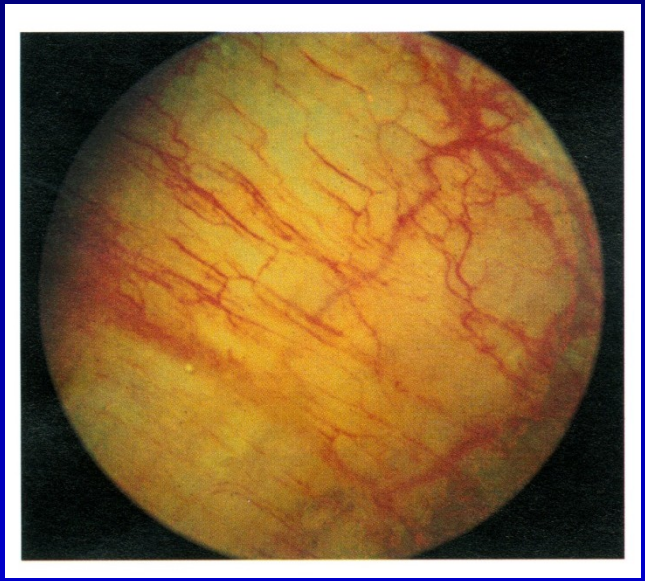
Condrocytes are nourished from synovial fluid

Cartilage has no vessels and nerves - low regeneration

The fluid is pushed by movements into the cartilage



Synovial membrane



Network of vessels

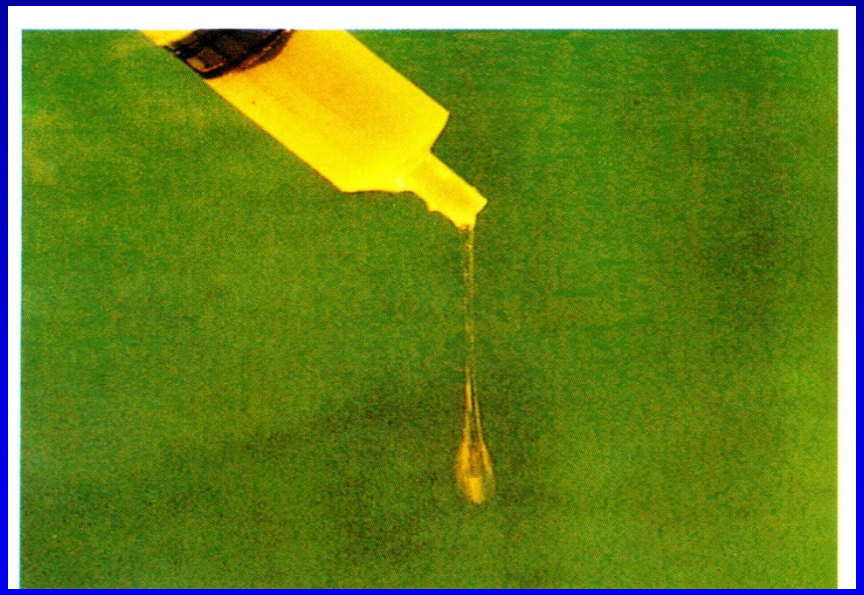
It contains:
Cells A – macrophages
Cells B – produce hyaluronic acid
Cells C – mixed cells – properties of cells A and B

Synovial fluid

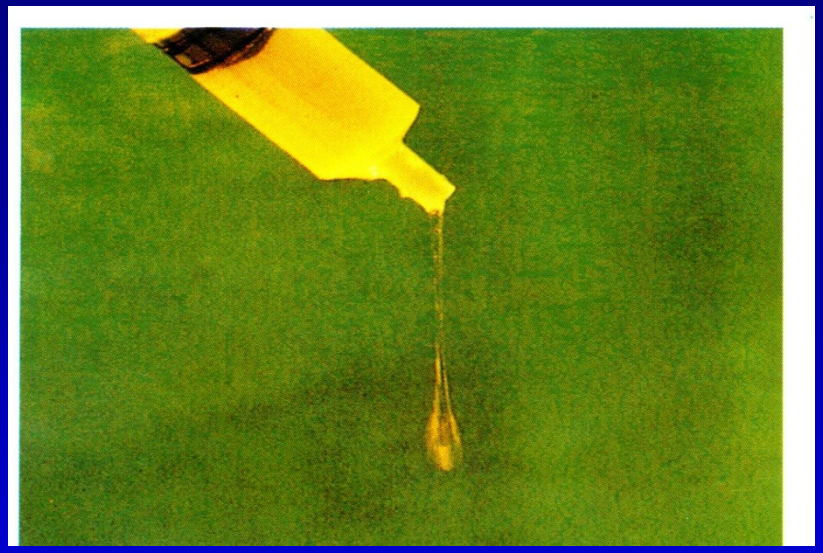
Clear, slight yellowish
Viscous

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 fluid



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

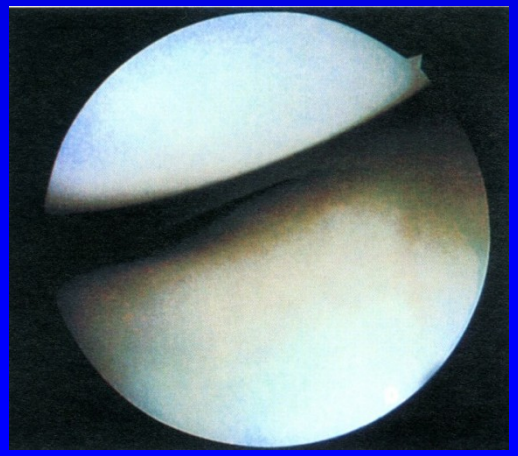
Dieseases of joints

- 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

Osteoarthrosis

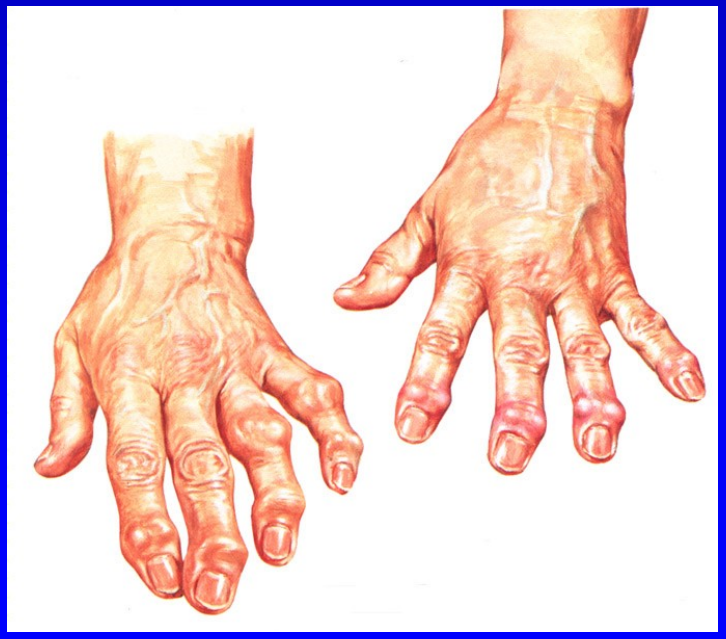
15 percent of the population

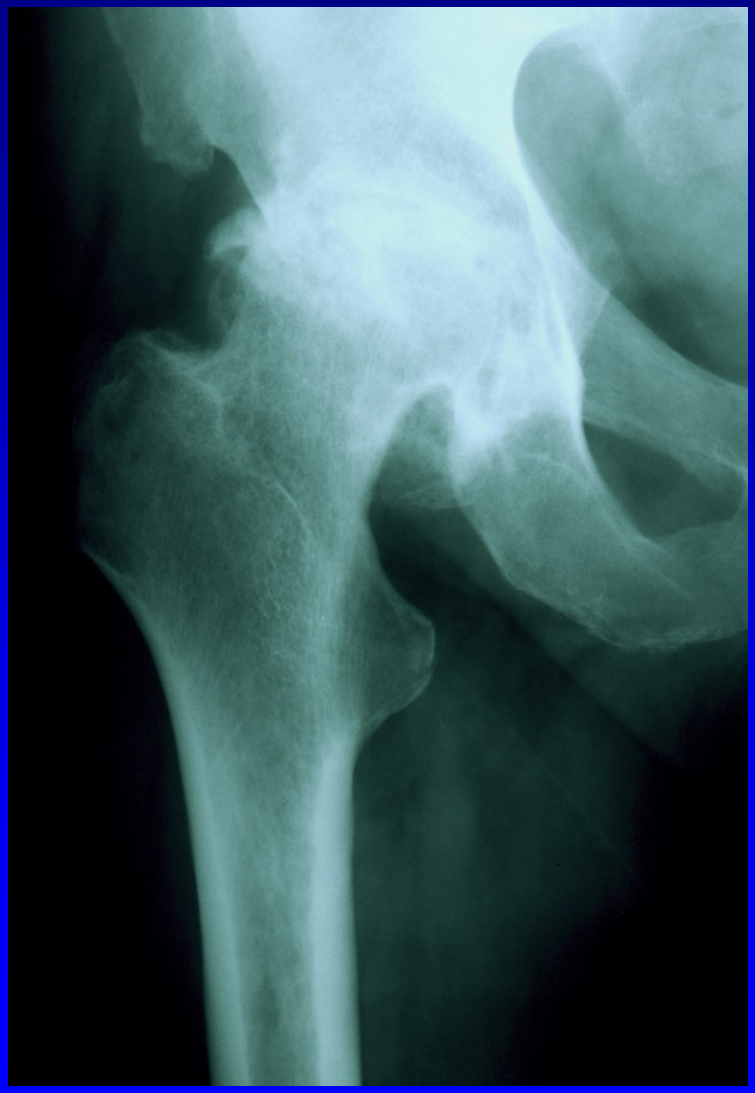
50 percent of people above 65 years

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

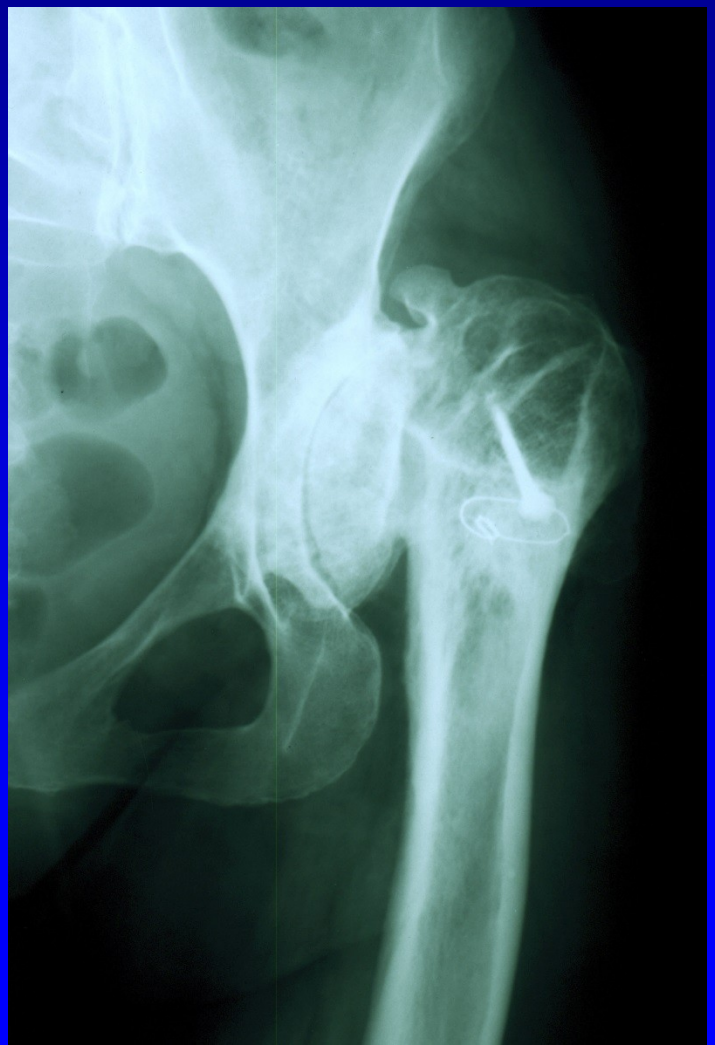




Secondary O.A.

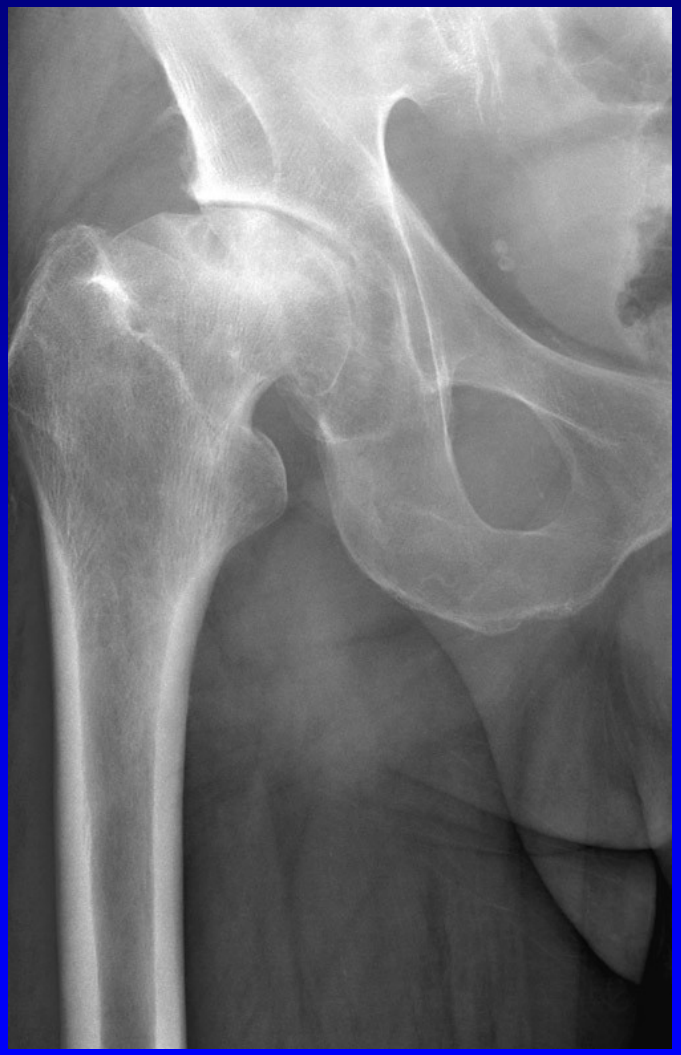
1. Mechanical factors (DDH, Perthes disease, aseptic necrosis, slipped femoral epiphysis, condition after fractures)
2. Metabolic disorders (ochronosis, gout, chondrocalcinosis, Gaucher disease)
3. Hormonal disorders (acromegaly, diabetes m.)
4. Haemofilia
5. Inflamated disorders (septic artritis, R.A.)

DDH- developmental dysplasia of the hip joint



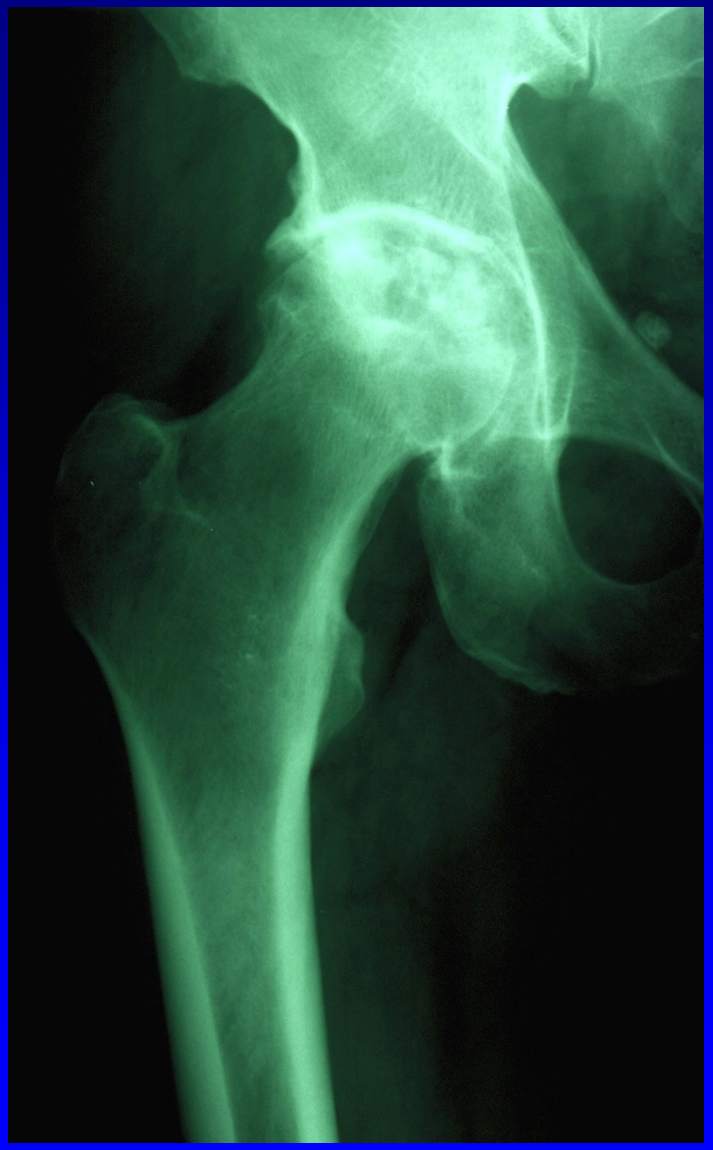
Obr. 6

Condition after Perthes disease



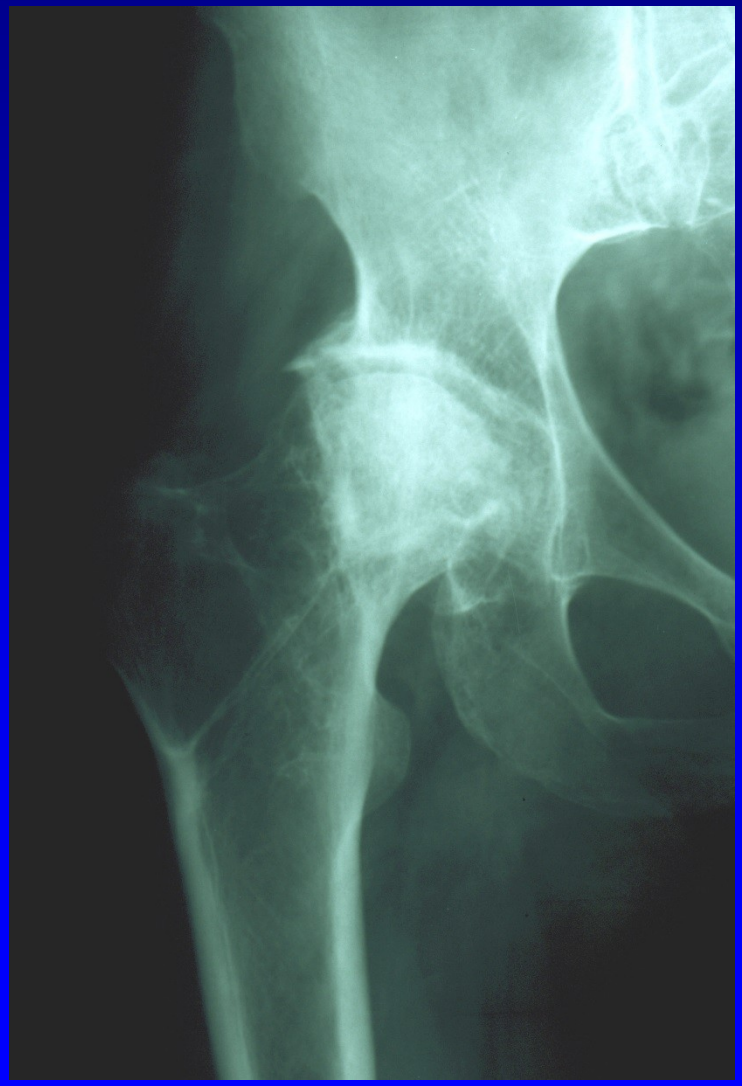
Obr. 8

Idiopatic necrosis of the femoral head



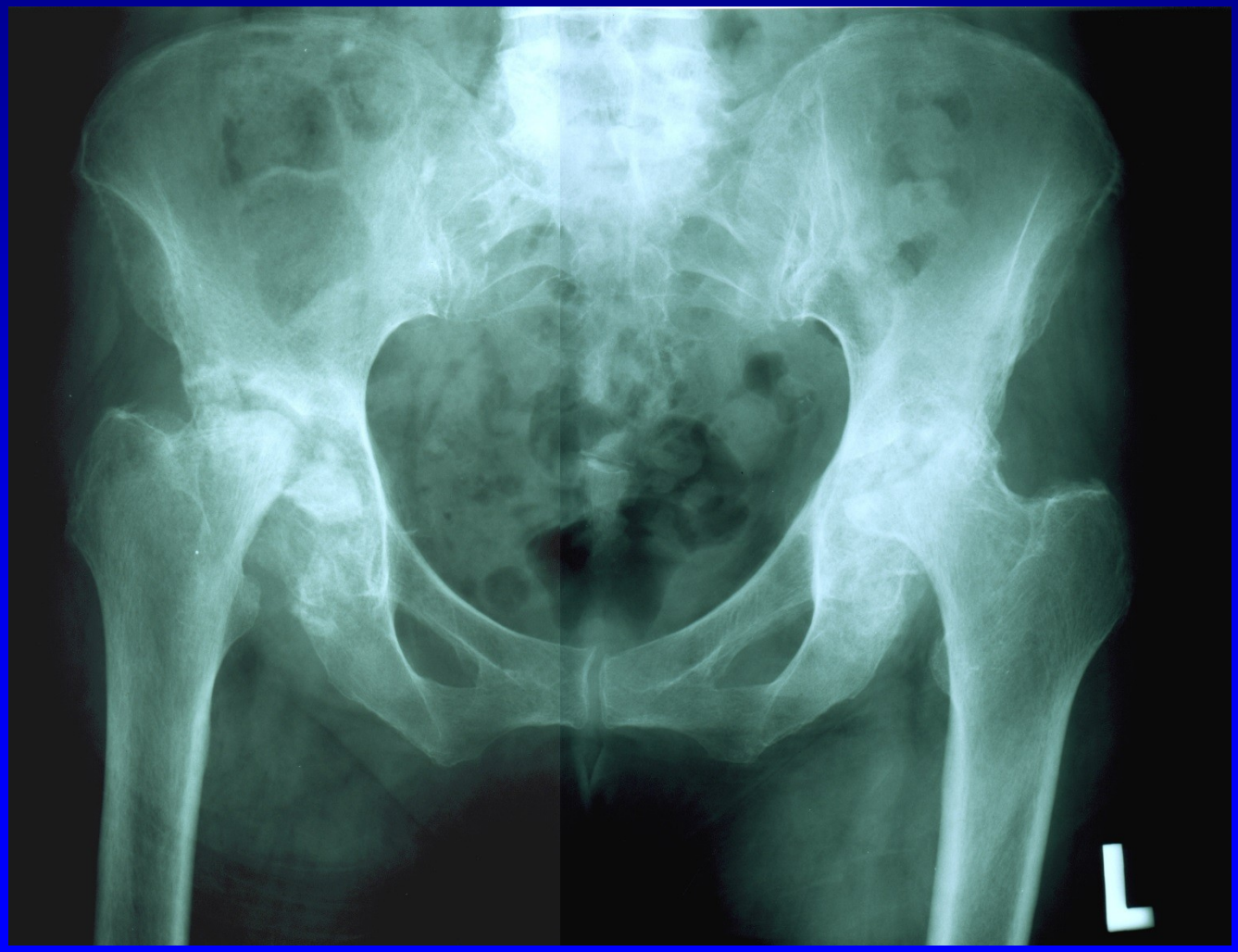
Obr. 7

Necrosis after femoral neck fracture



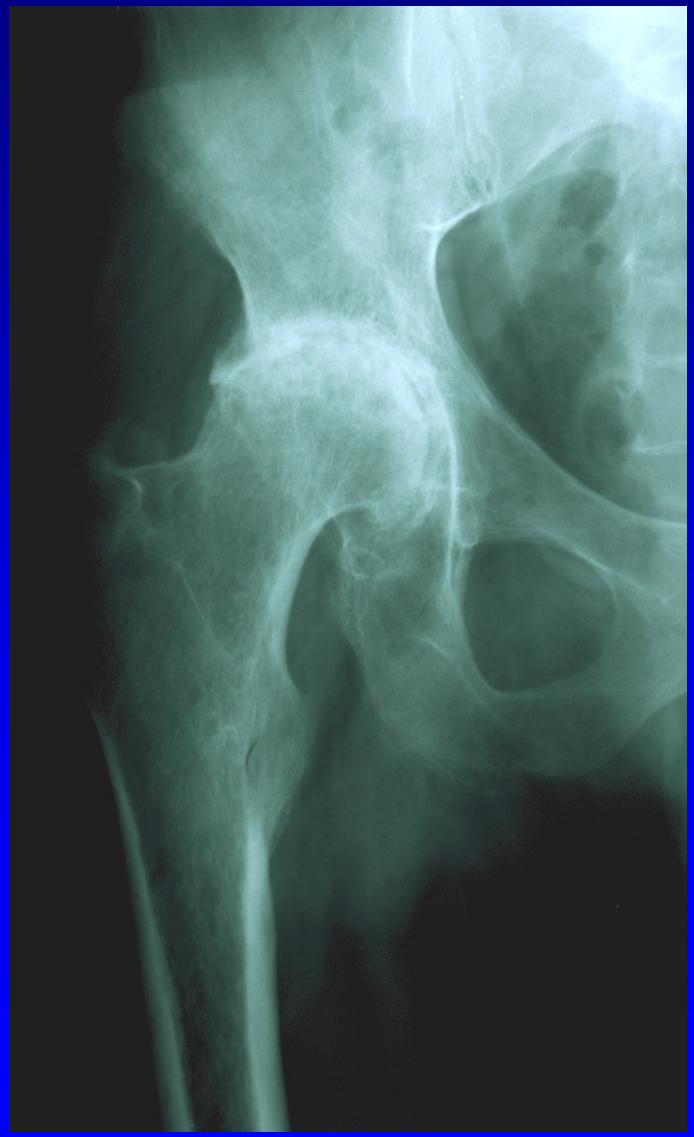
Obr. 9

Rheumatoid artritis



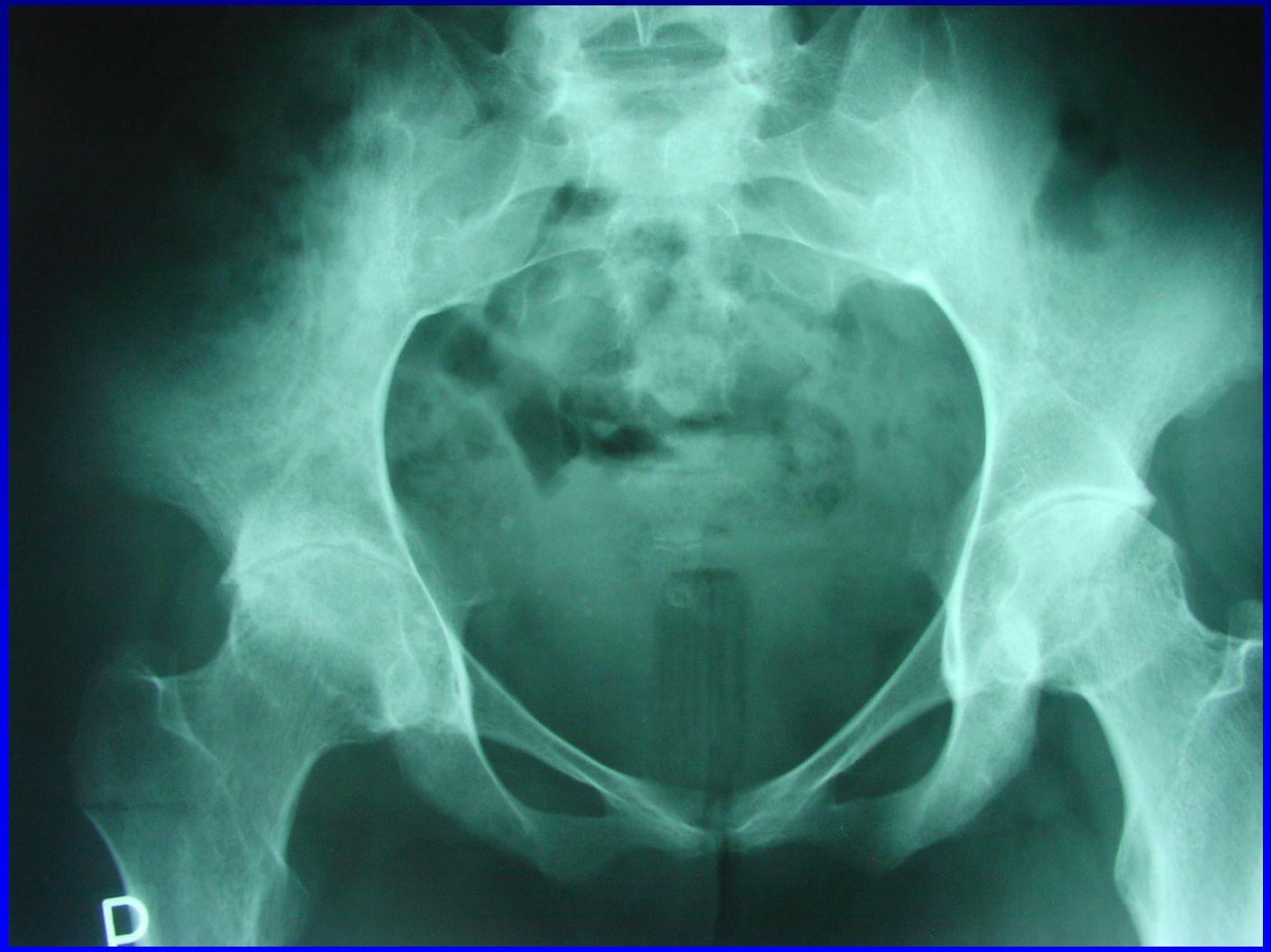
Obr. 10

Ancylosing spondylitis - hip joint



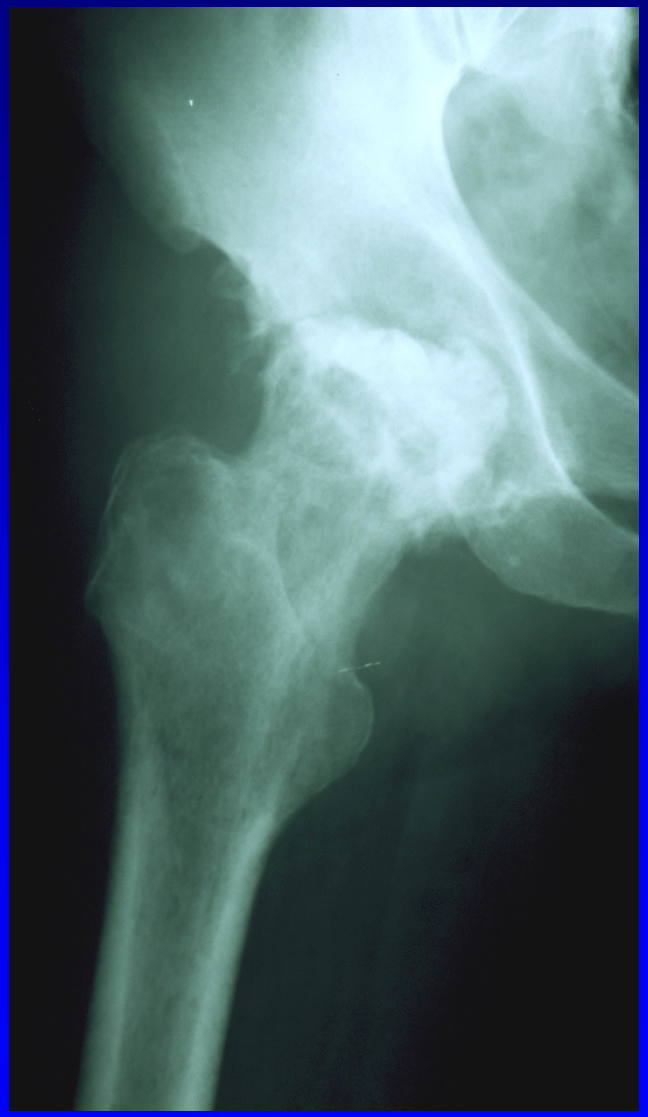
Obr. 11

Ancylosing spondylitis



Obr. 12

Septic arthritis



Obr. 13

Risk factors

Age over 50 years

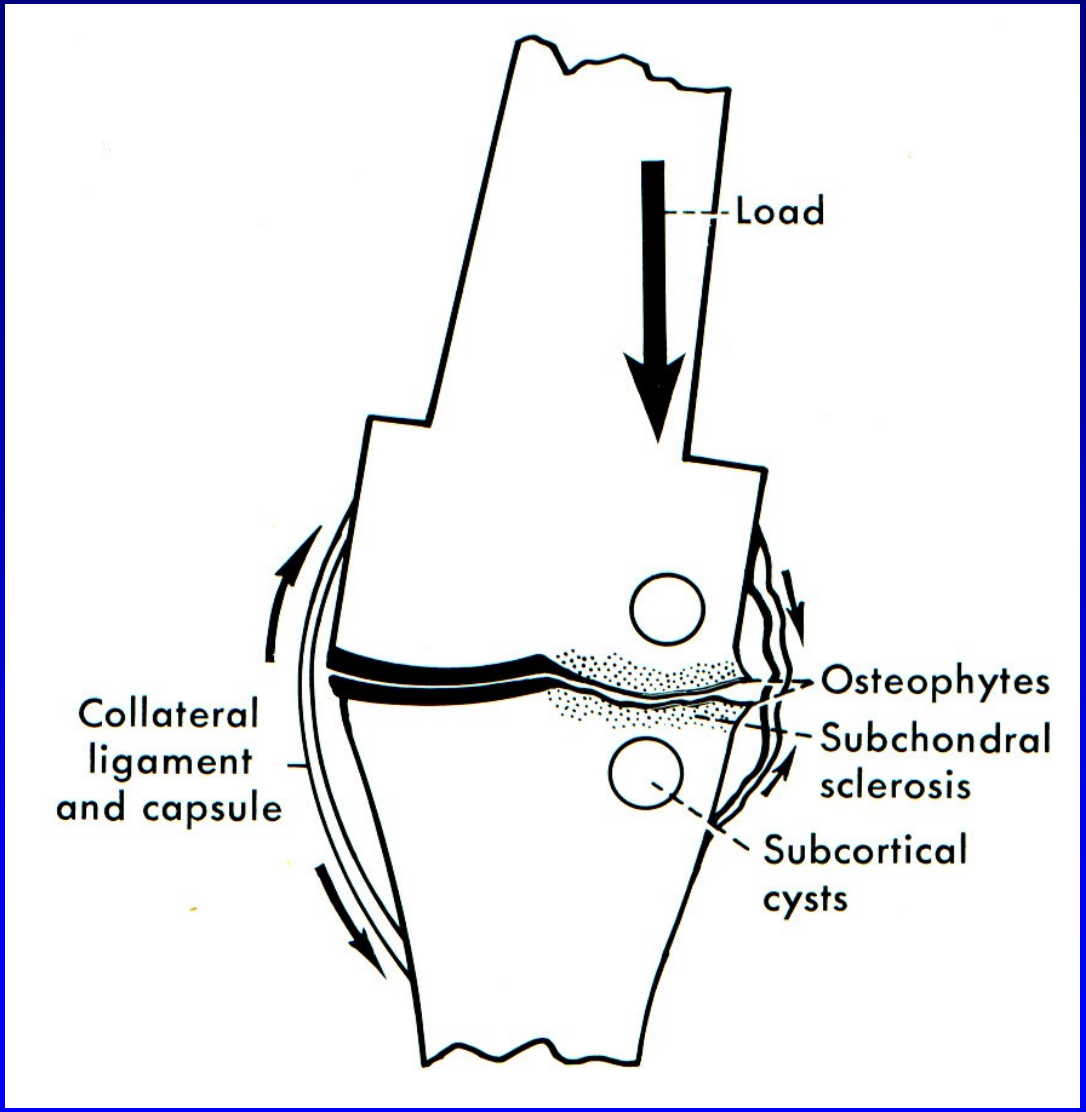
Obesity

Mutation of gene for procollagen II (COL2A1)

Autosomal gene for Heberden´s nodes
is 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

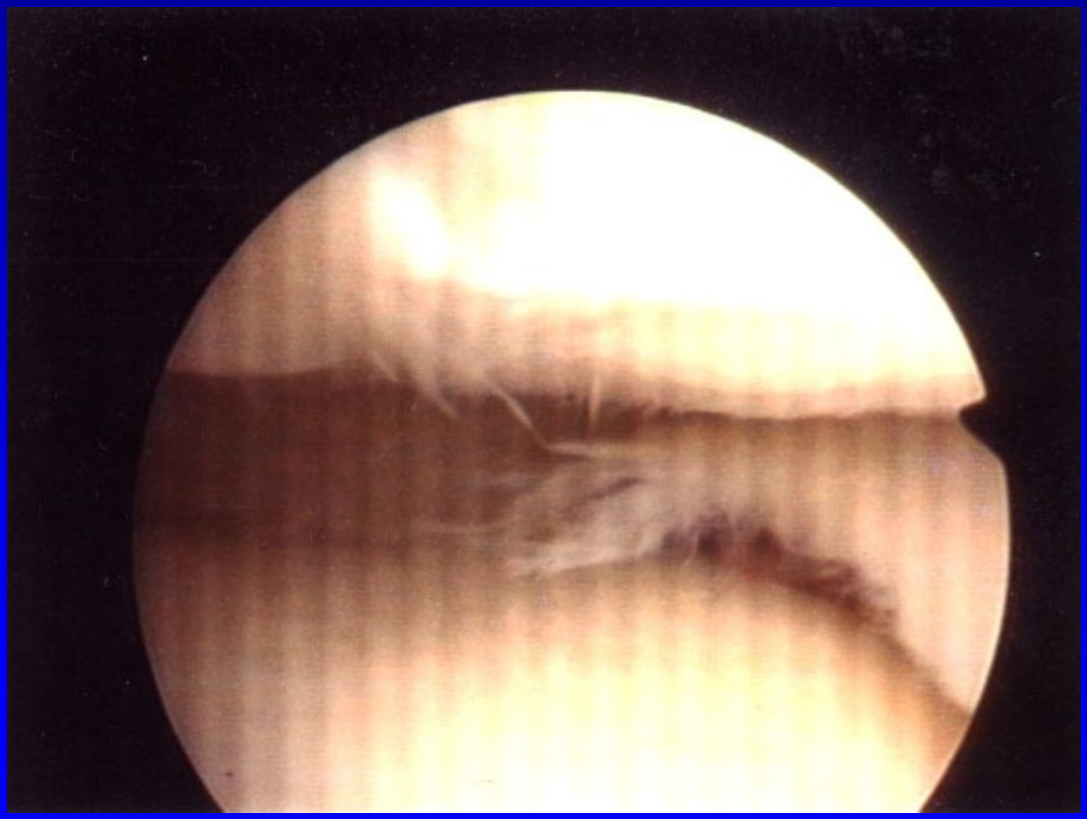
Mechanical O.A.



Obr. 14

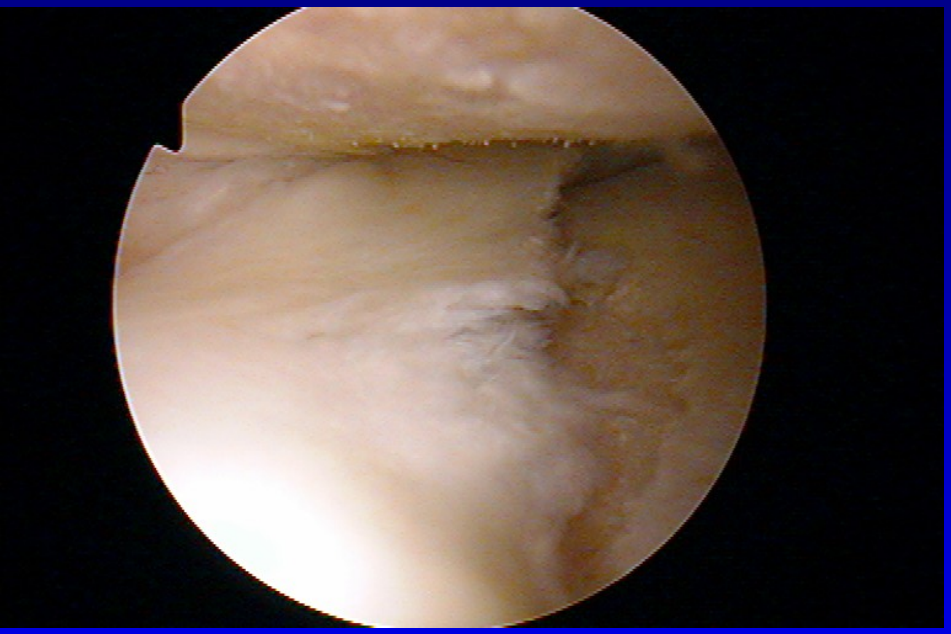
Macroscopis changes

Cartilage is matte, soft, yellowish, fibrilations

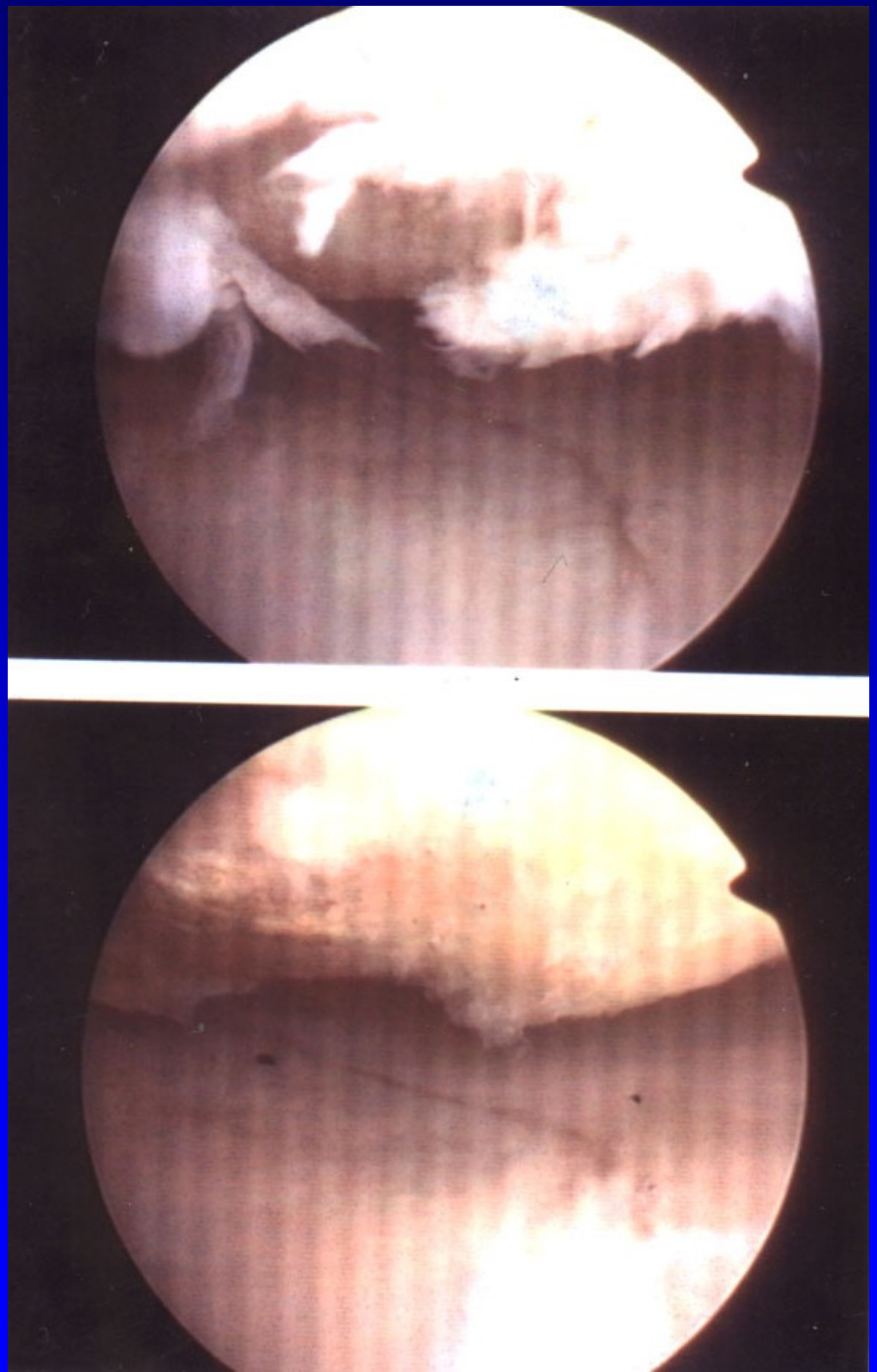


Obr. 15

Ulcers, defects

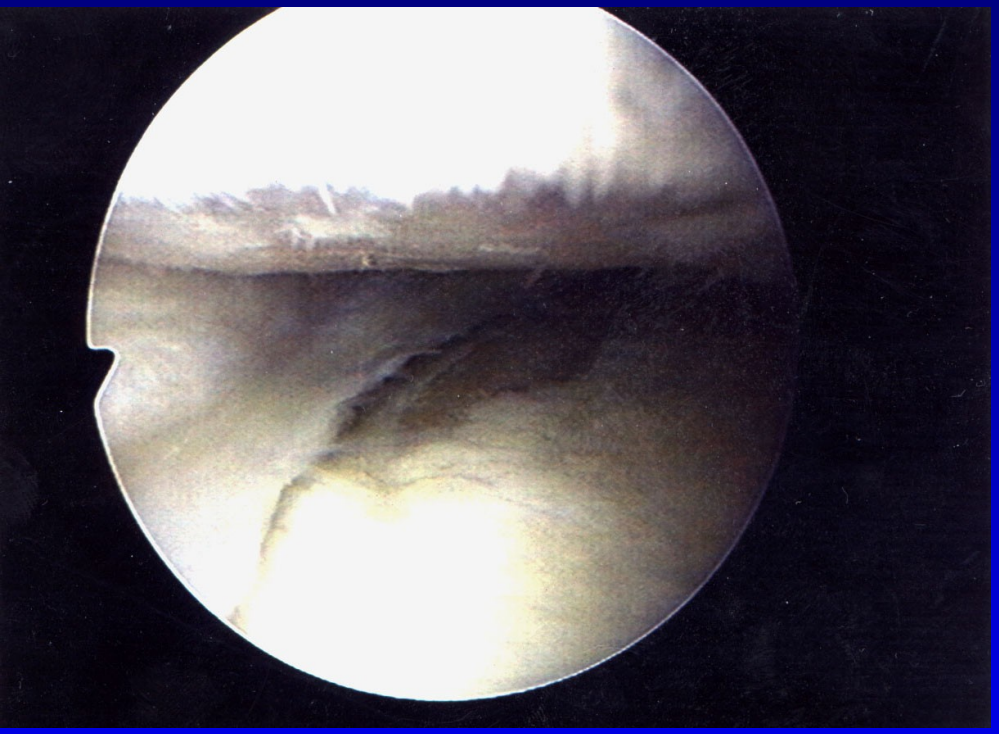


Obr. 16

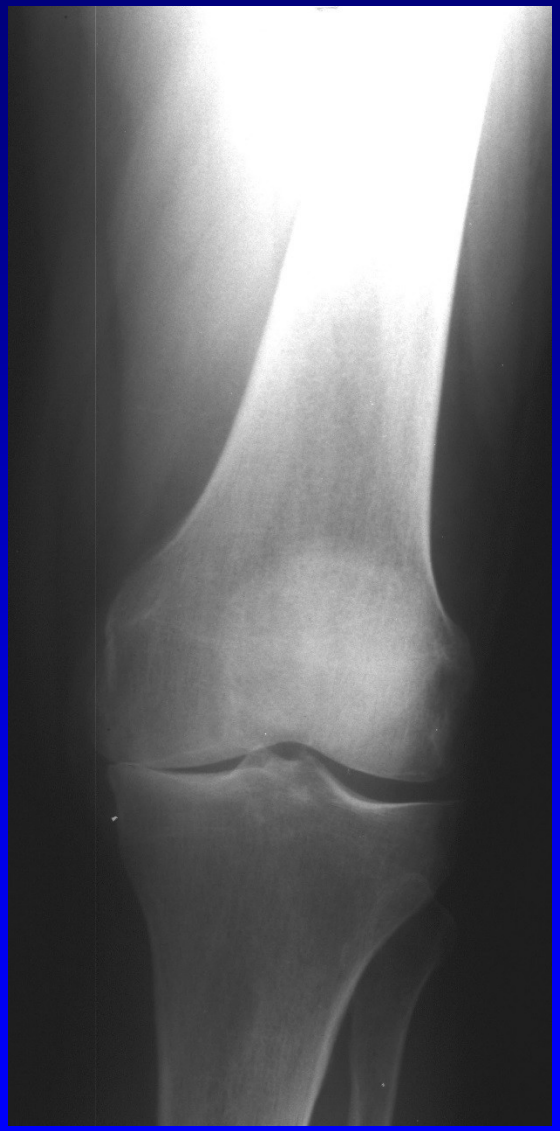


Obr. 17

Subchondral bone is sclerotic



Obr. 18



Obr. 19

Macroscopic changes

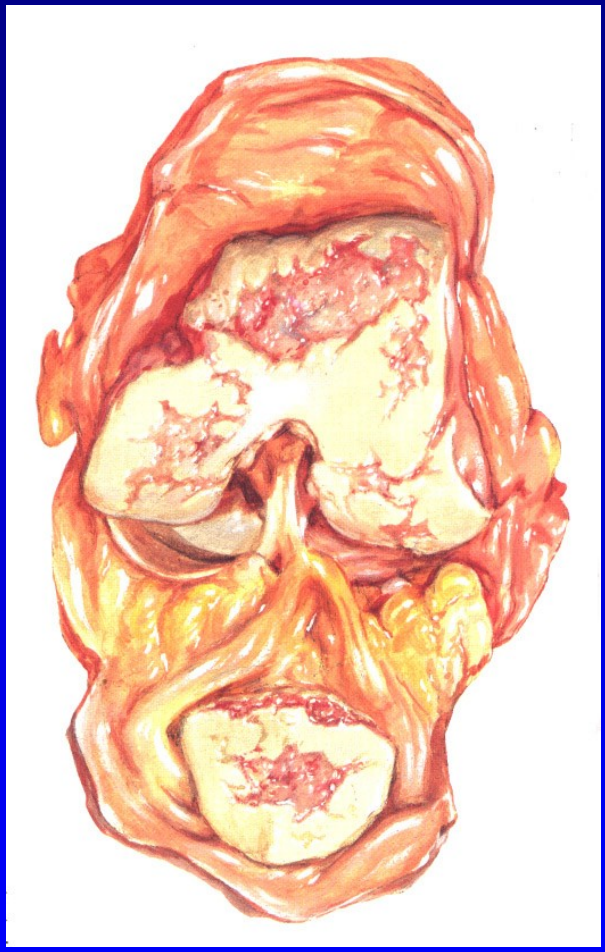
Subchondral cysts

Osteophytes

Narrowing of cartilage

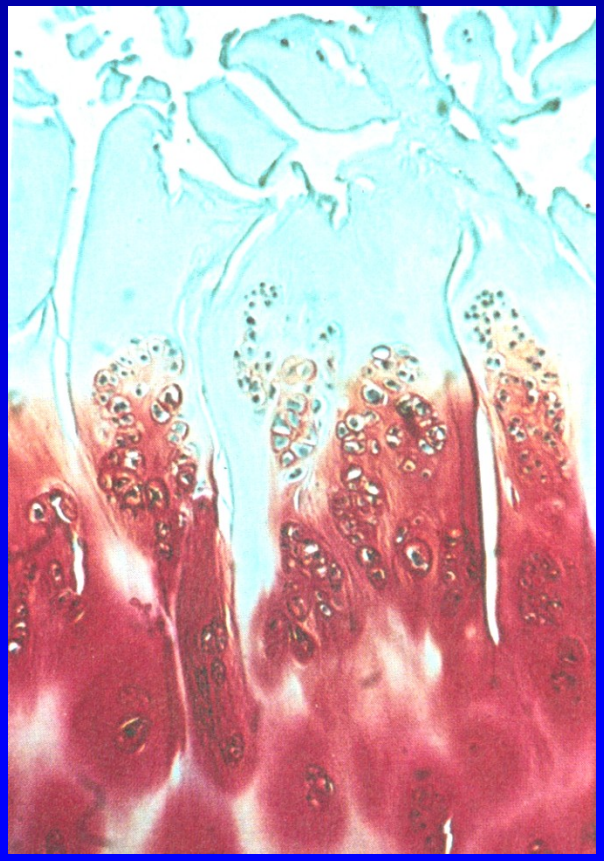
Hypertrophic synovial membrane

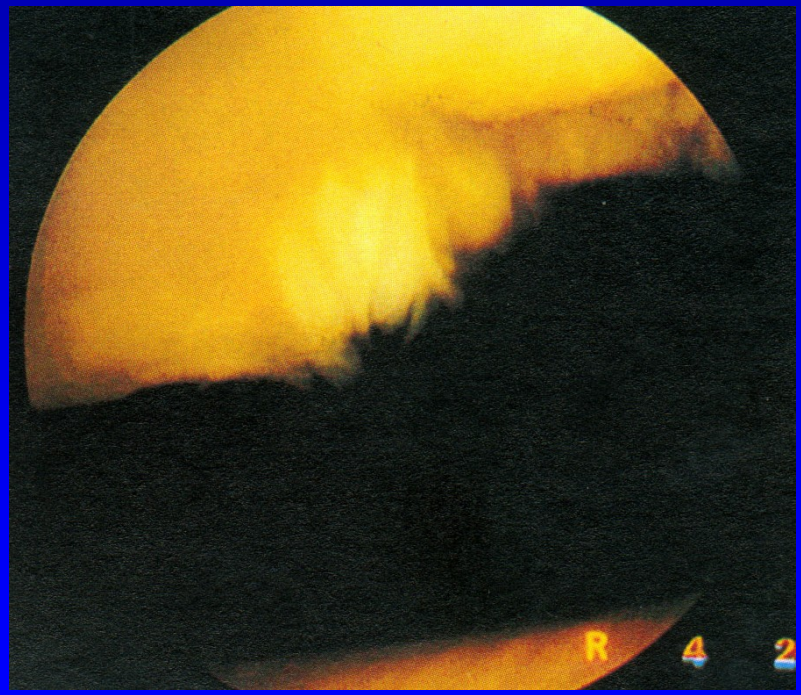
Loose bodies



Obr. 20

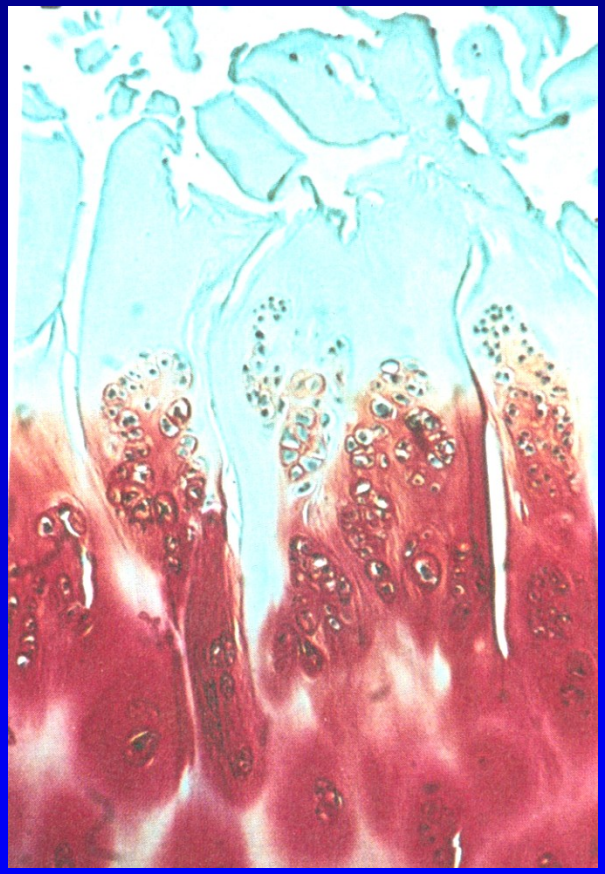
Condrocytes make clusters in 10-20
Irregularities of the surface
Lamina splendens is absent, fibrilations
Fissures, defects of cartilage
Collagen network is disturbed





Biochemical changes

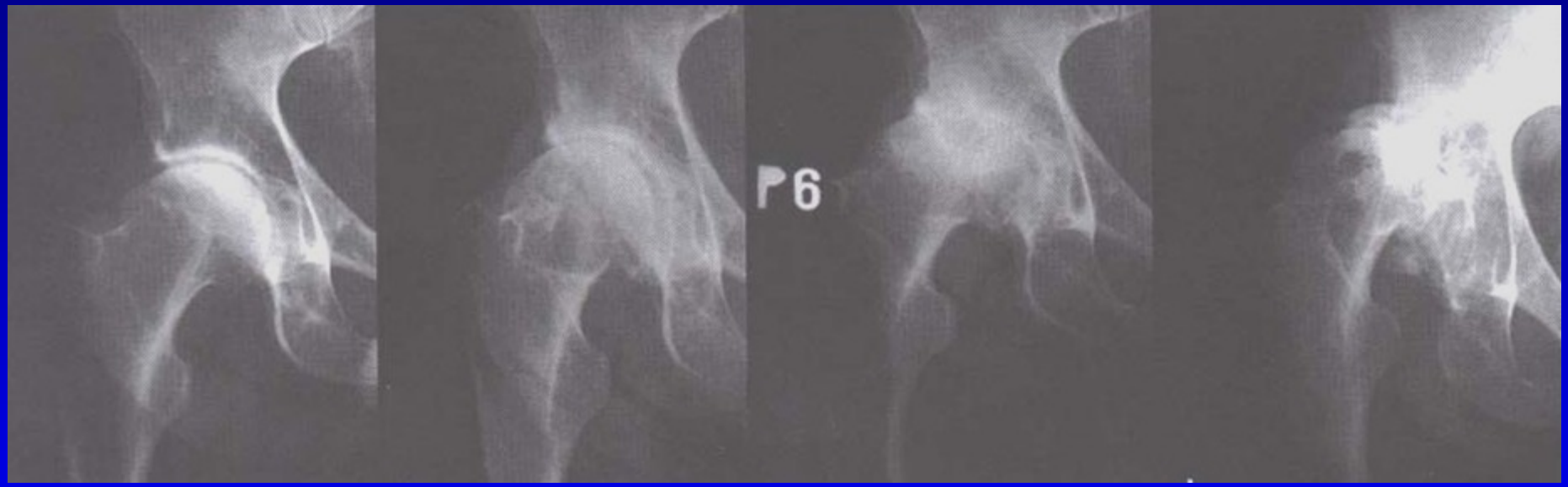
Higher amount of water
Synthesis of PG is higher
Loss of proteoglycans is high
Chondroitin 6 sulfate - less
Ketaransulfate- less
Condroitin 4 sulfate is higher



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.

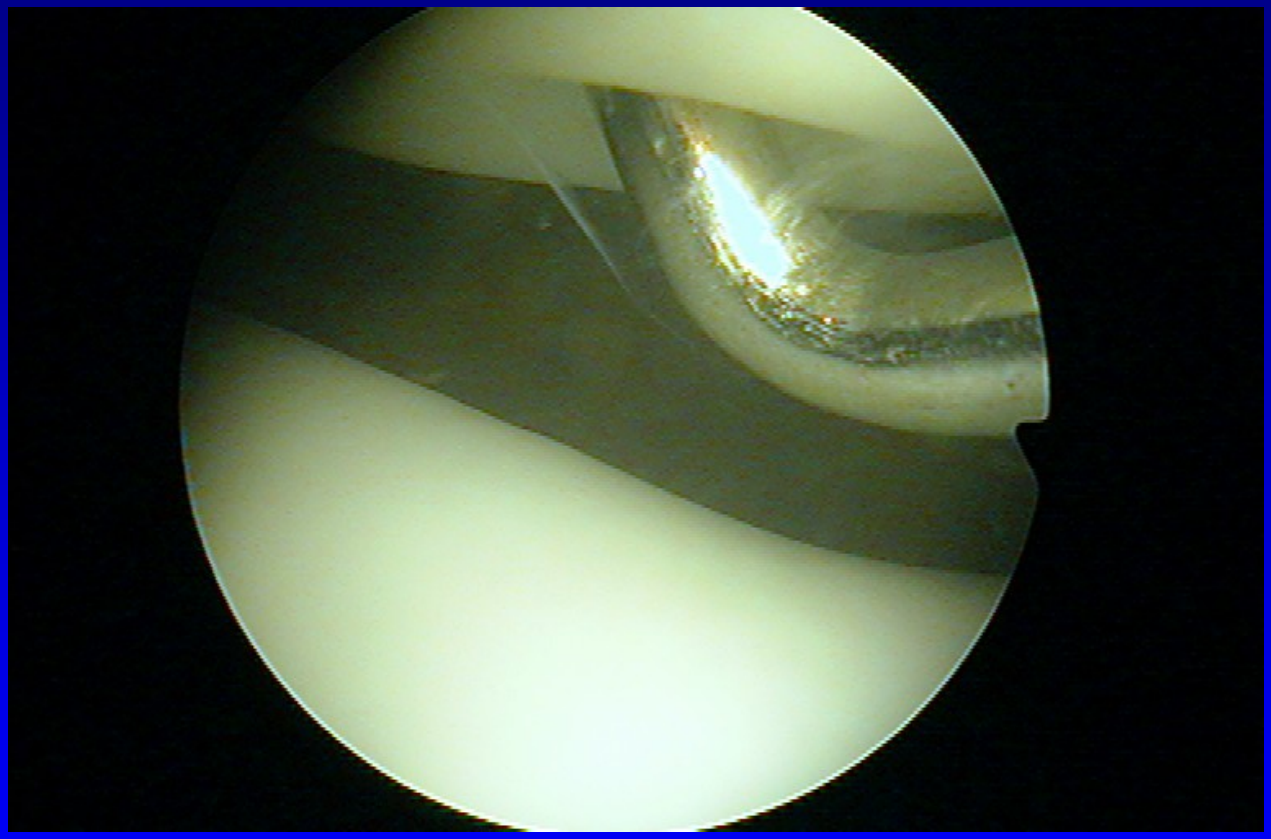


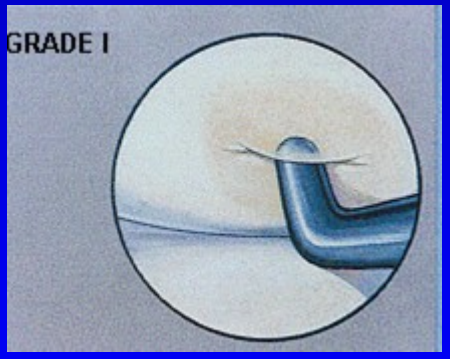
Kellgren- Lawrence classification I- IV.

Chondropathy

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

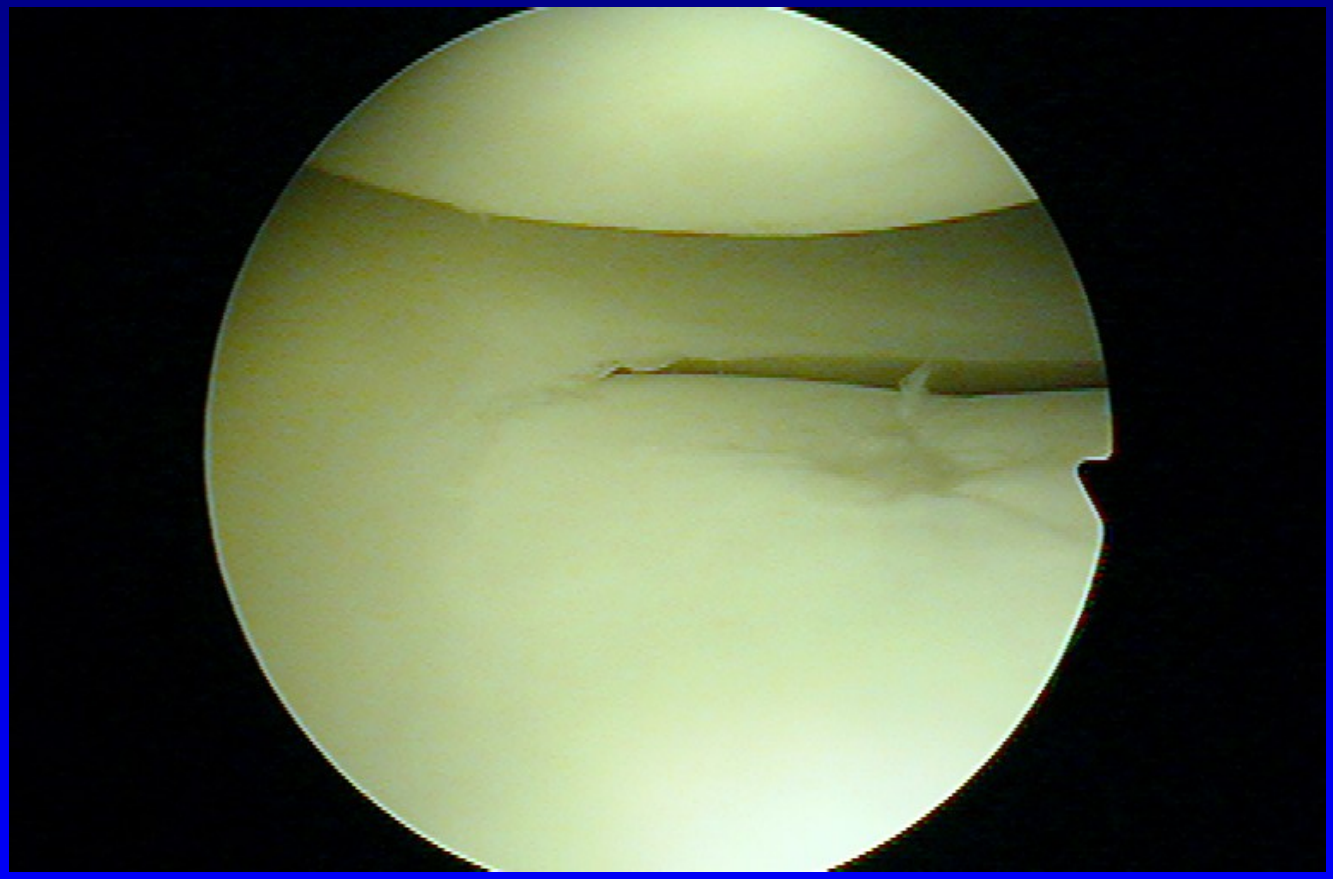
Chondropathy I. st.

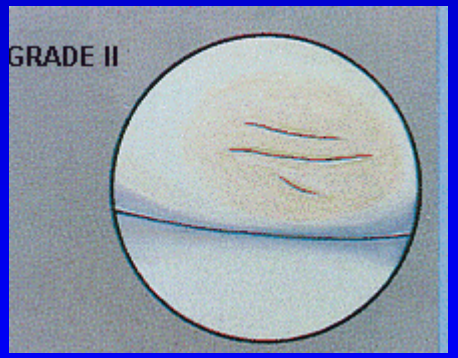




Chondromalatia- soft cartilage

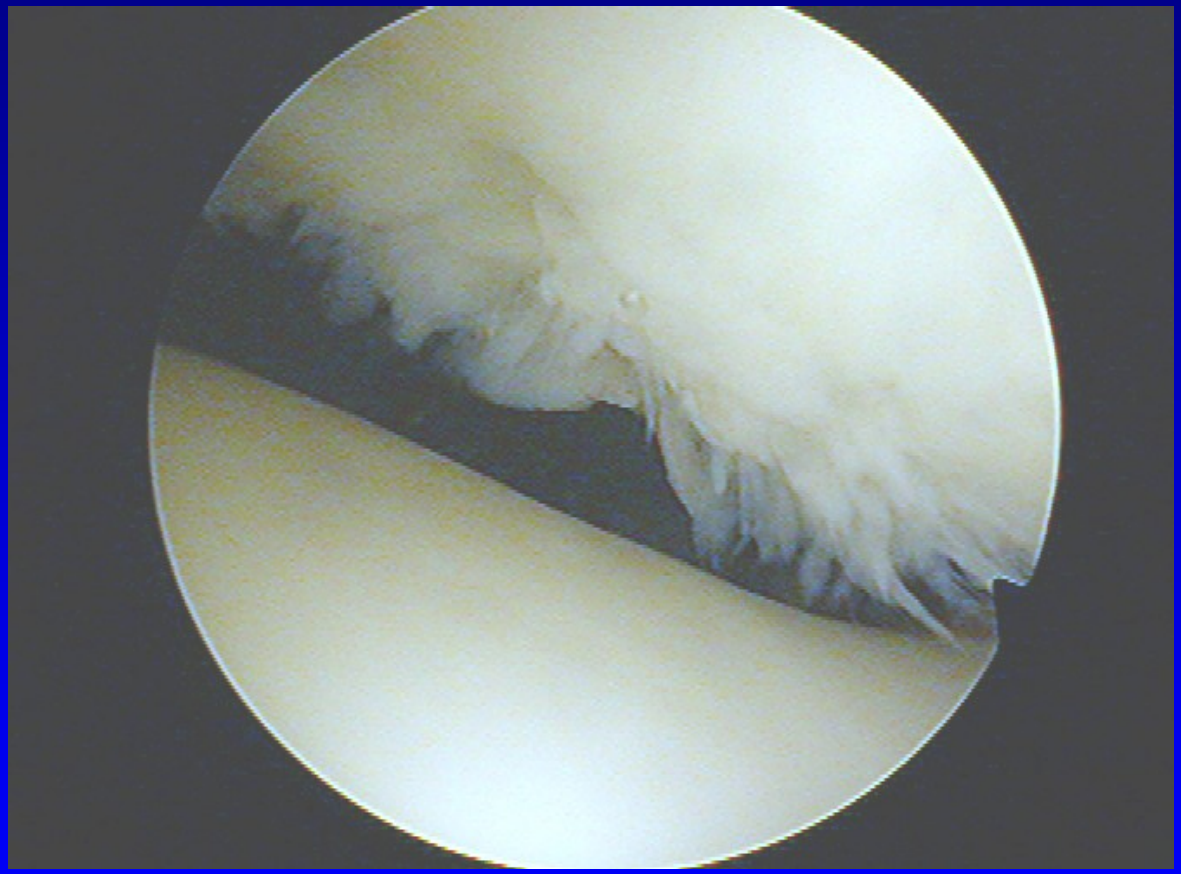
Chondropathy II. st.

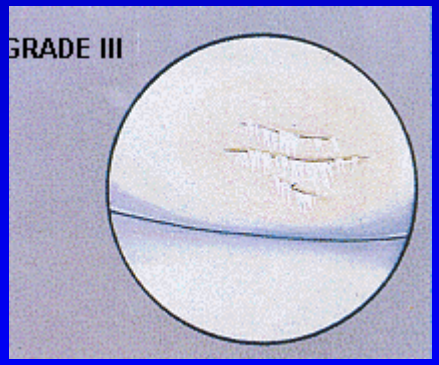




Fissures in the cartilage

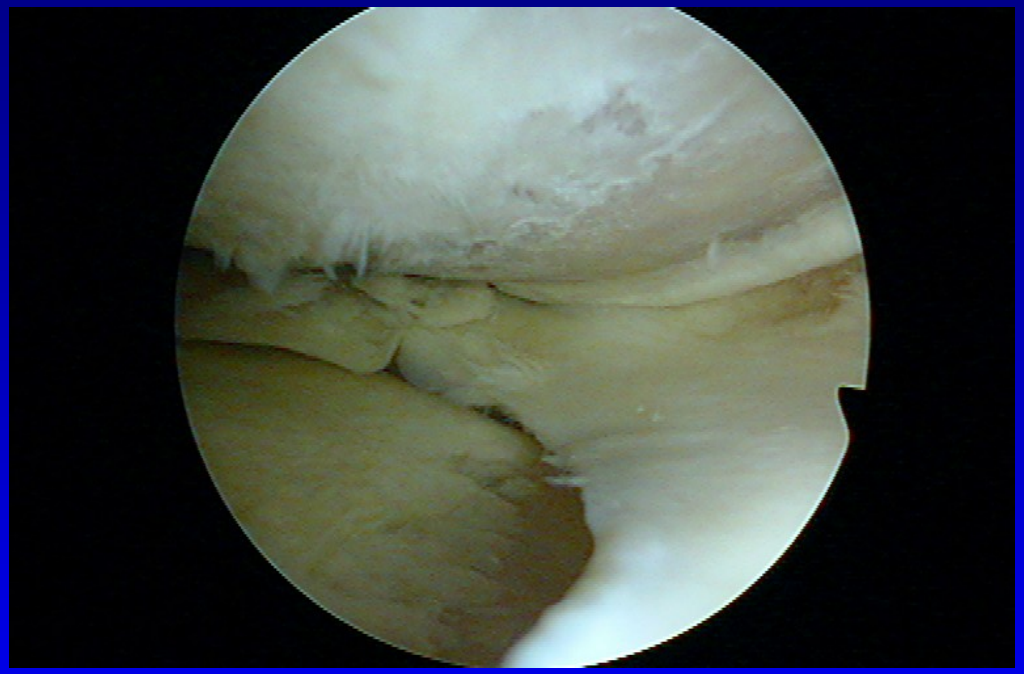
Chondropathy III. st.

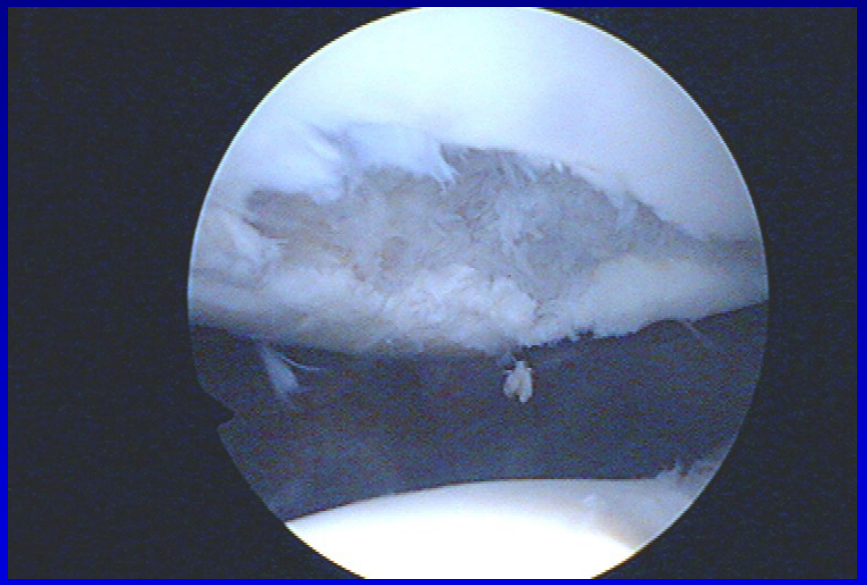




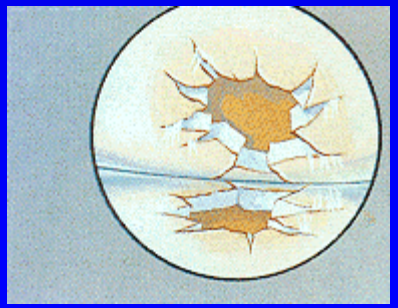
Fibrilation- „ crab meet“

Chondropathy IV. st.





Defects 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, codein,)

Nonsteroidal antiinflammatory drugs (NSAID)

NSAID

Inhibitors of cyclooxygenase 1 COX - 1 inhibitors

Ibuprofen
indometacin
piroxicam
naproxen
diclofenac
tiaprofenic acid

NSAID

Inhibitors of cyclooxygenase - 2 COX 2 inhibitors

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

Selective : celecoxib (Aclexa)
rofecoxib

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
   diacerein
   ASU piascledine

2. local: hyaluronic acid

Combined drugs + collagen

SYSADOA local
- viscosuplementation

Hyalgan

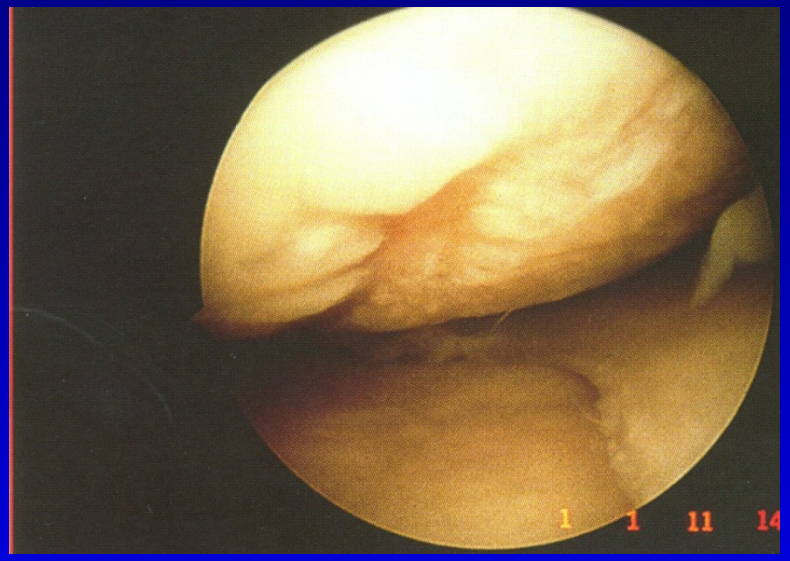
Synvisc

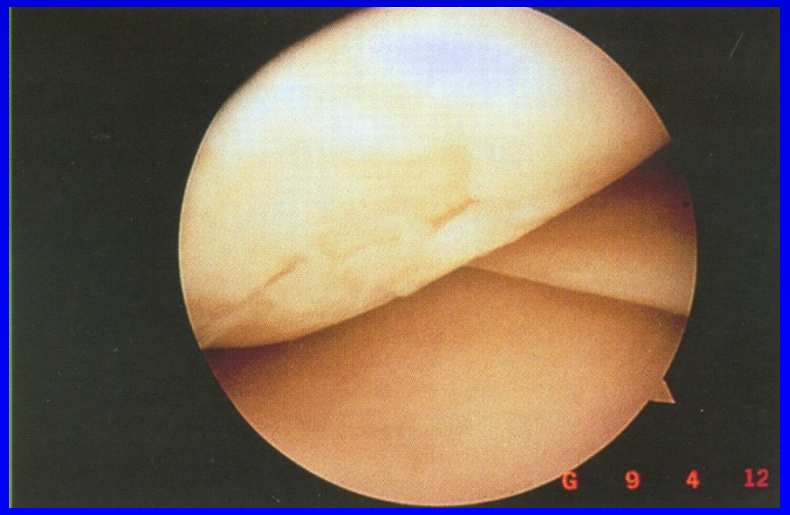
Synovial

Monovisc

Hyaline

Renehavis





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

Recommended treatment

Paracetamol- up to 4 g per day

NSA - + inhibitors of proton pump (omeprazol)

Chondroprotectives

Hyaluronic acid

Local corticosteroids

Pain department- in a case we can not do surgery

Other options

PRP- platelets rich plasma

ACP- autologous conditioned serum- Orthokine

Mesenchymal stem cells ?

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

Operative treatment

Preventive surgery

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

Operative treatment

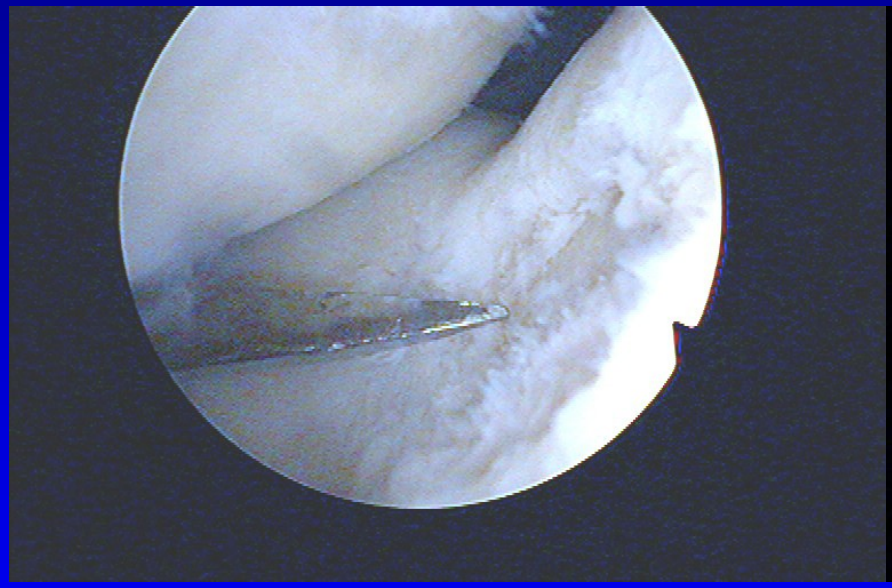
Resection arthroplasty – op. sec. Keller
op. sec. Girdlestone

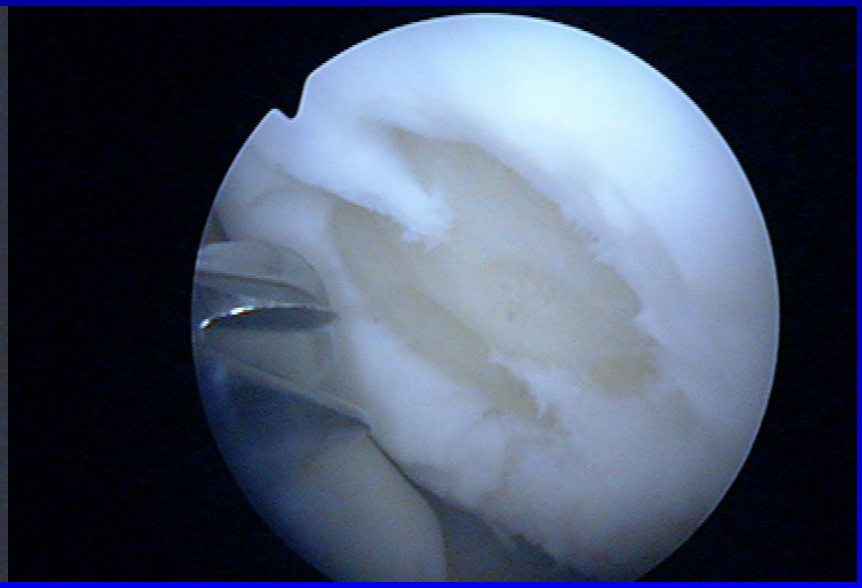
Arthrodesis

Total joint replacement

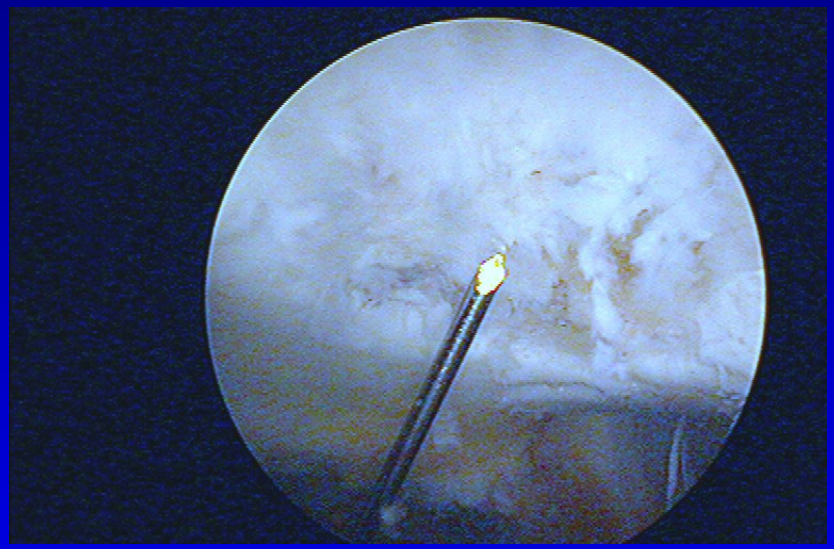
Options for localised chondral defects

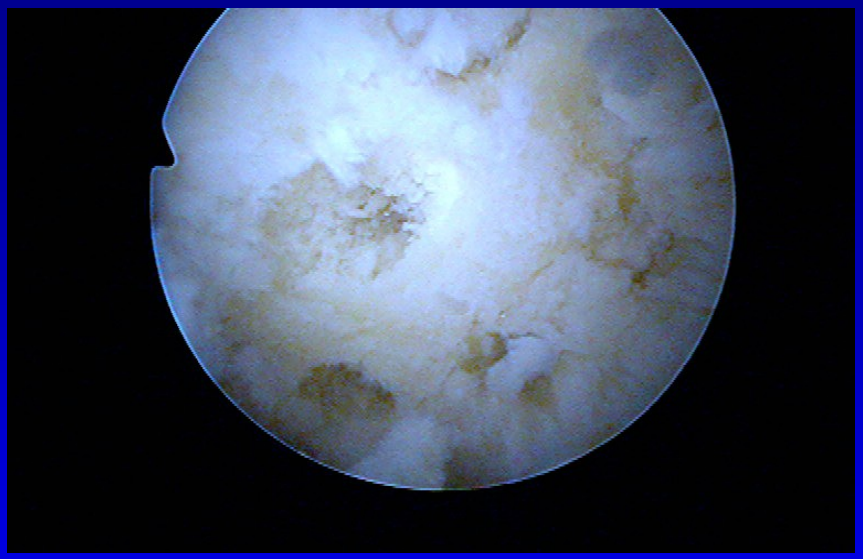
Shaving and drilling





Drilling





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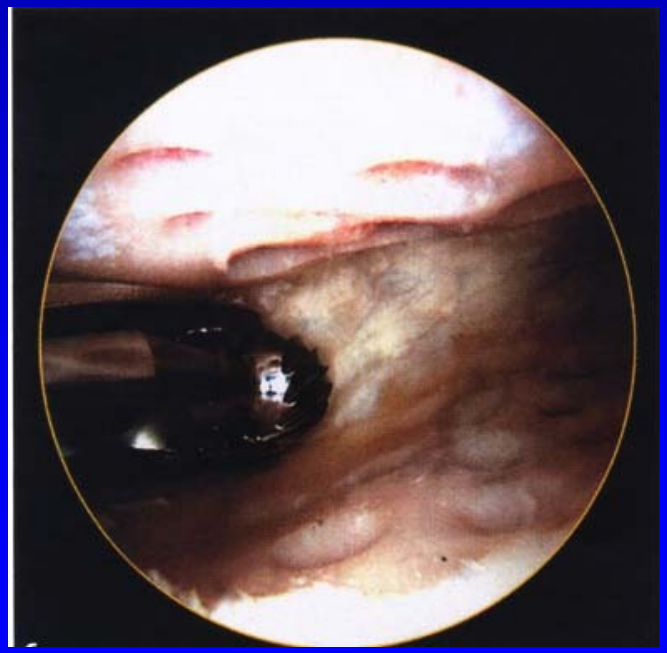
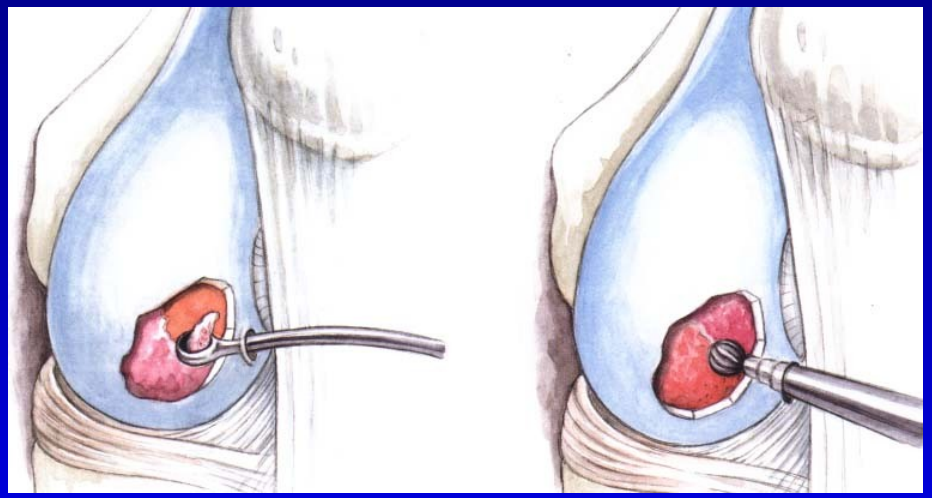
Abrasion chondroplasty

Curretage
Shaver

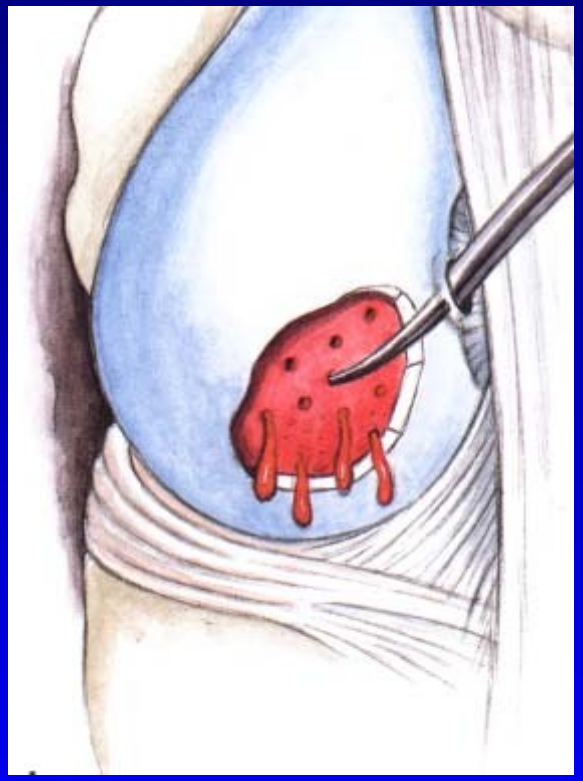




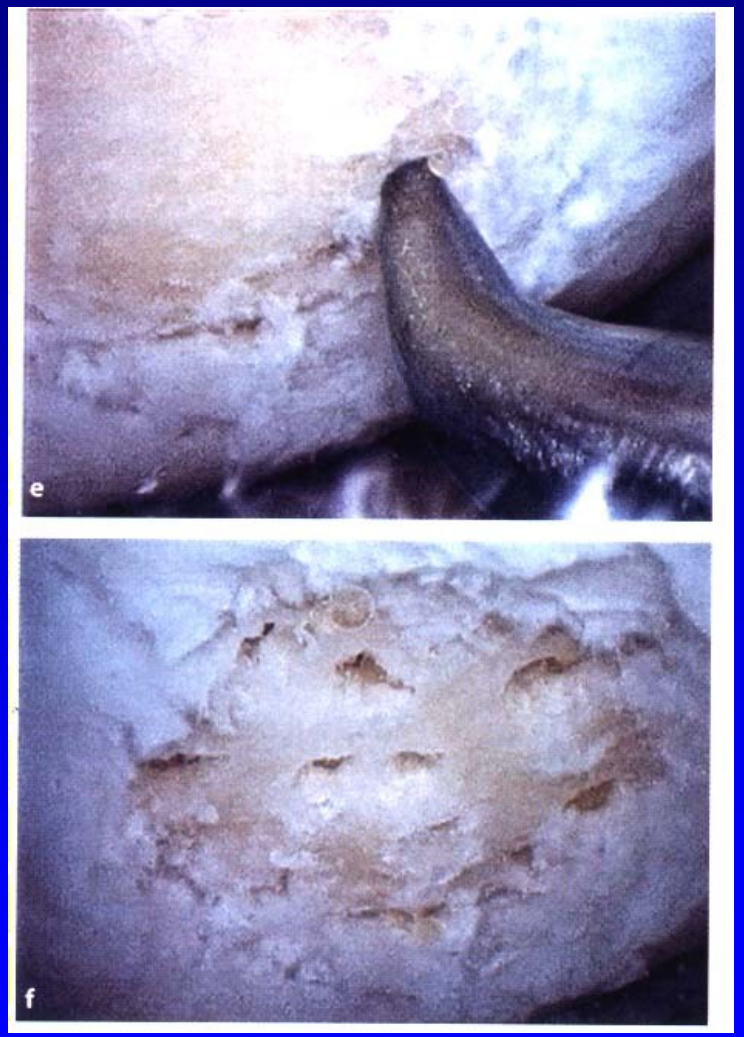
Microfractures

Perforation of subchondral bone
- slight bleeding
Steadman, J.R., 1999

Multipotent stem cells into
the defecfts
The aim- to create fibrocartilago

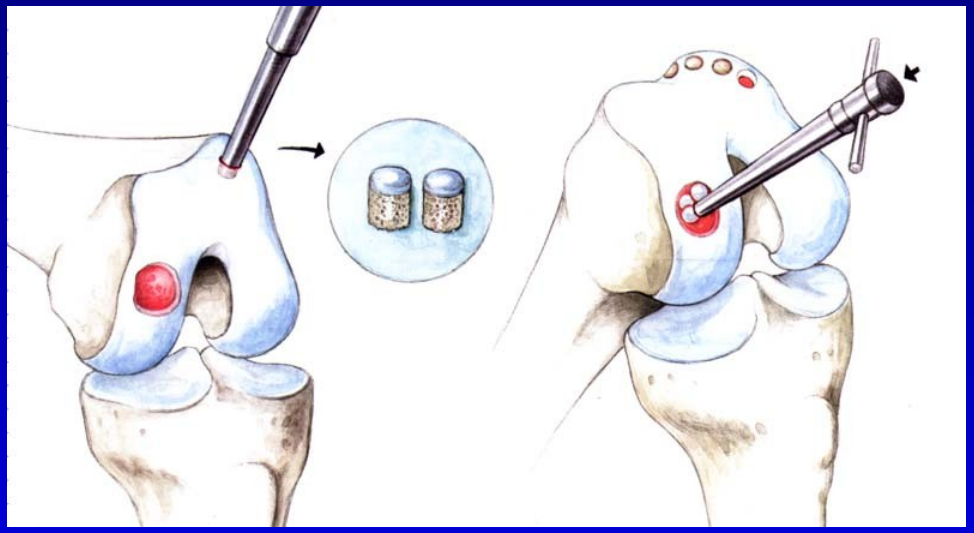


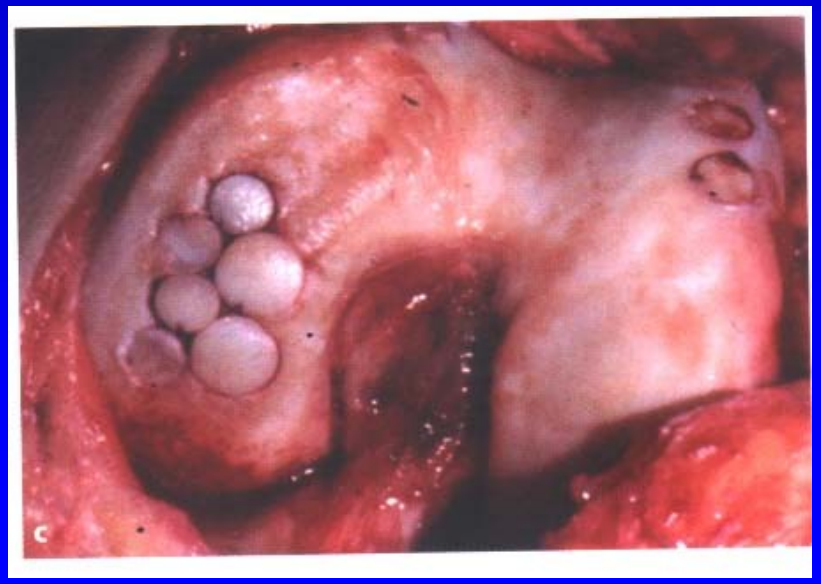
Microfractures



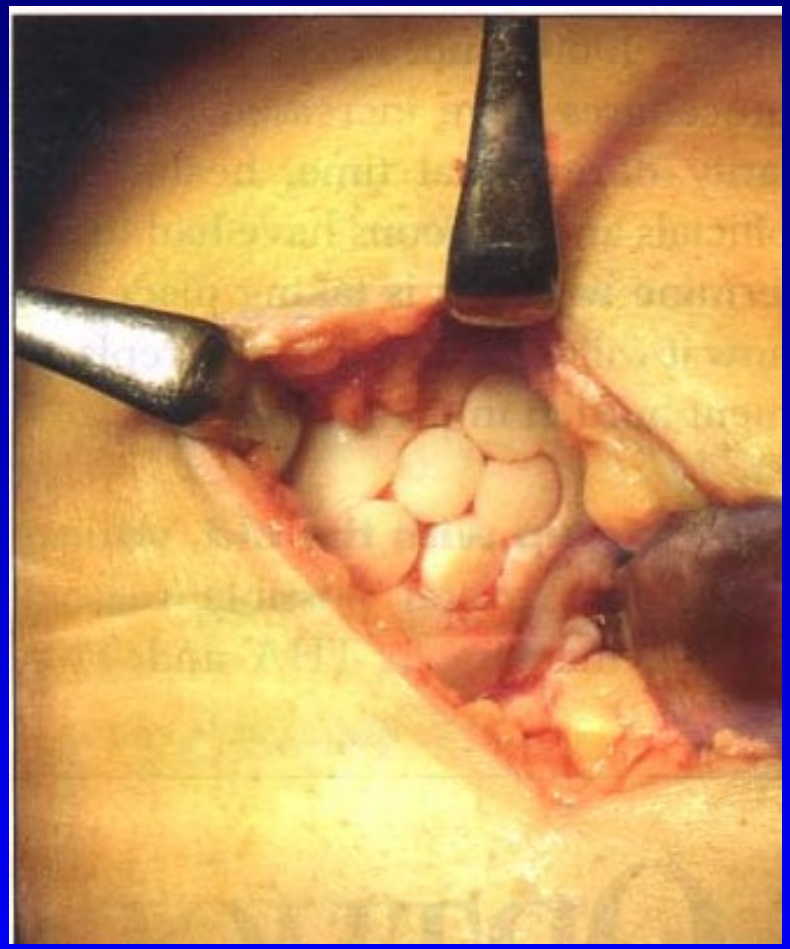
Osteochondral autograft transfer- OAT Mosaicplasty

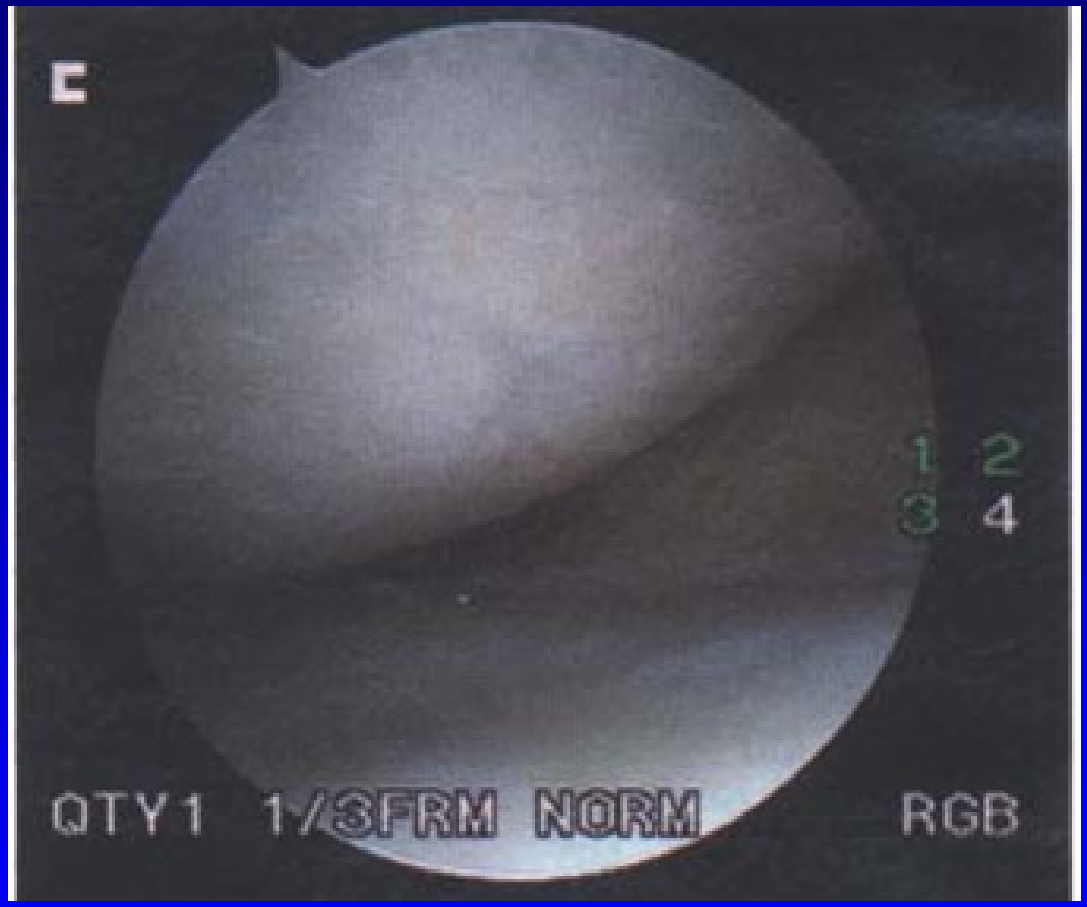
Hangody, L., 1992
Defects up to 2 - 4 cm^2





OAT

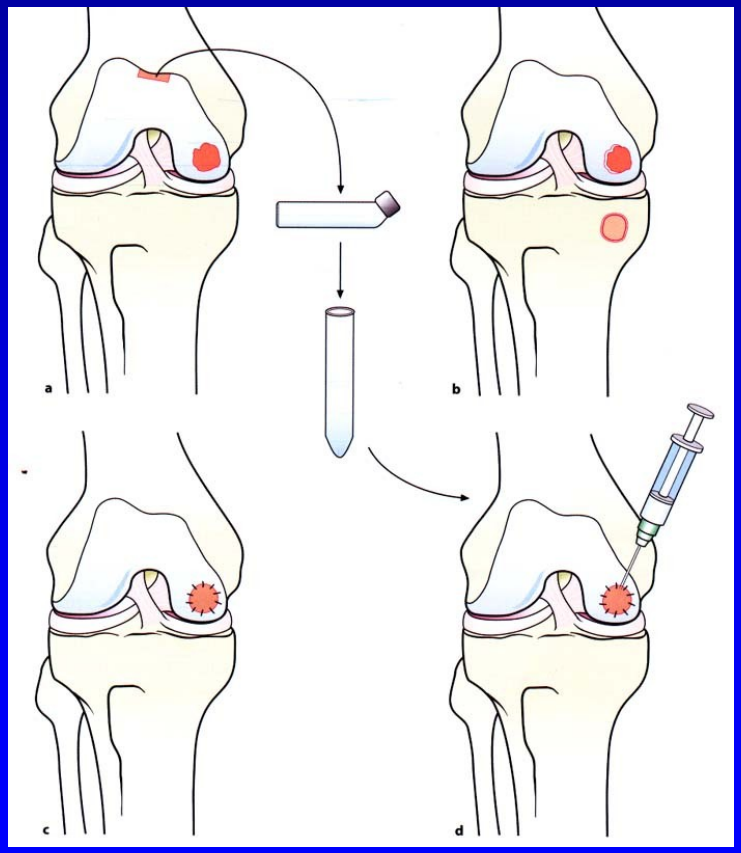


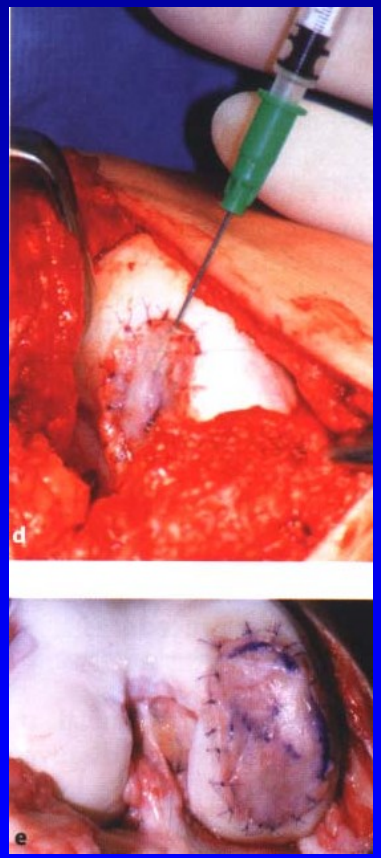


4 years after surgery

ACI – autologous chondrocyte implantation

Transplantation of autologous chondrocytes
into defects of cartilage
Chondrocytes in suspension under periostal layer



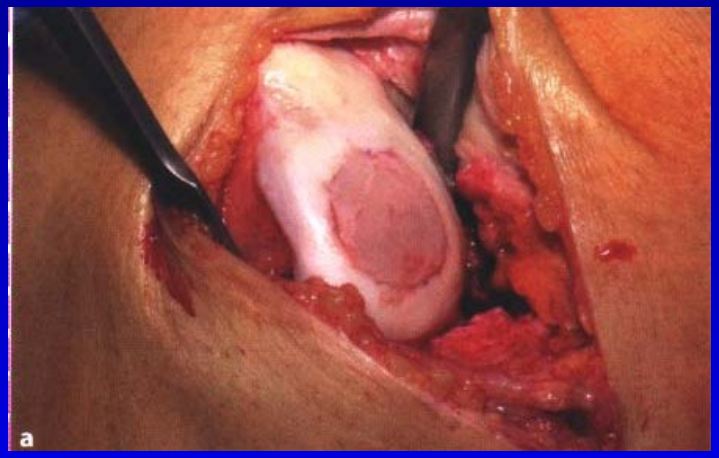


Hyalografts and chondrografts

Scaffolds- HyaloFast, Chondrotissue…

Biodegradable

Matrix for stem cells from bone marrow after drilling or from serum



Collagen scaffolds

HyaloFast- scaffold

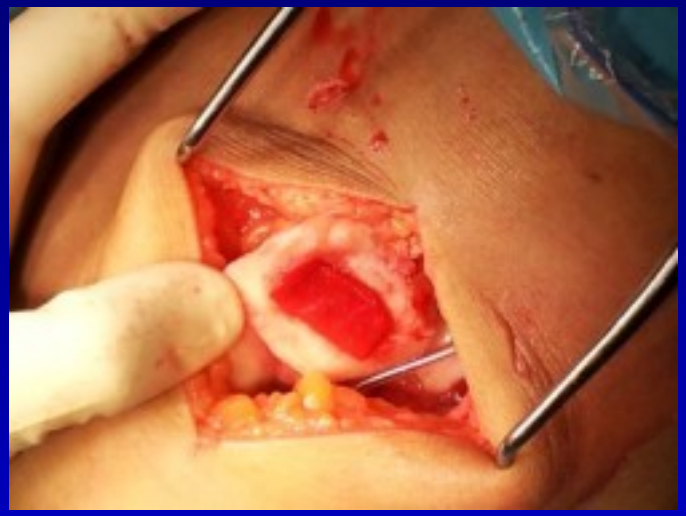
Polymer of HA

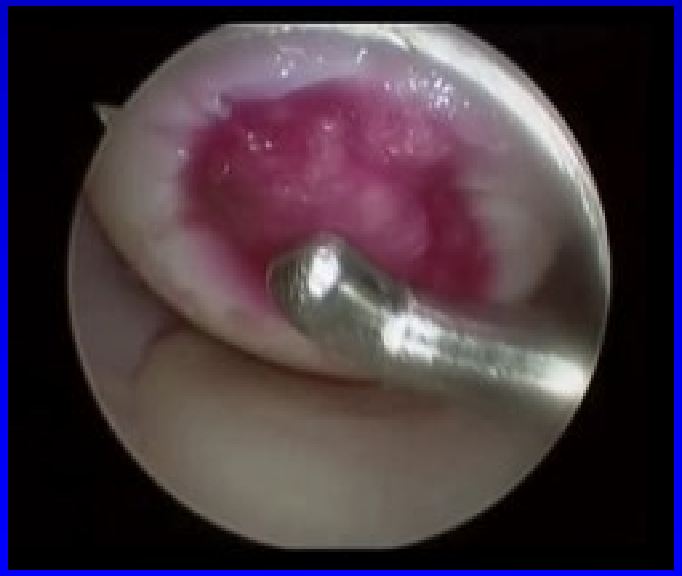
No special fixation

Scaffold serves for maintaining of stem cells from bone marrow

Supports viable cells

Fills the defects of hyaline cartilage

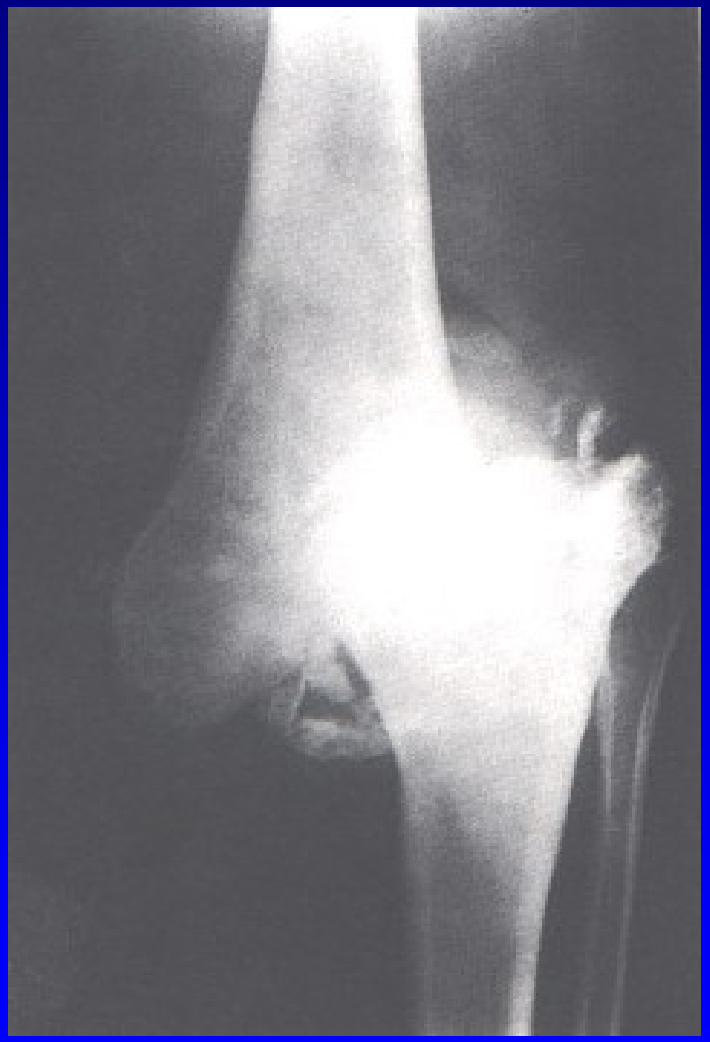




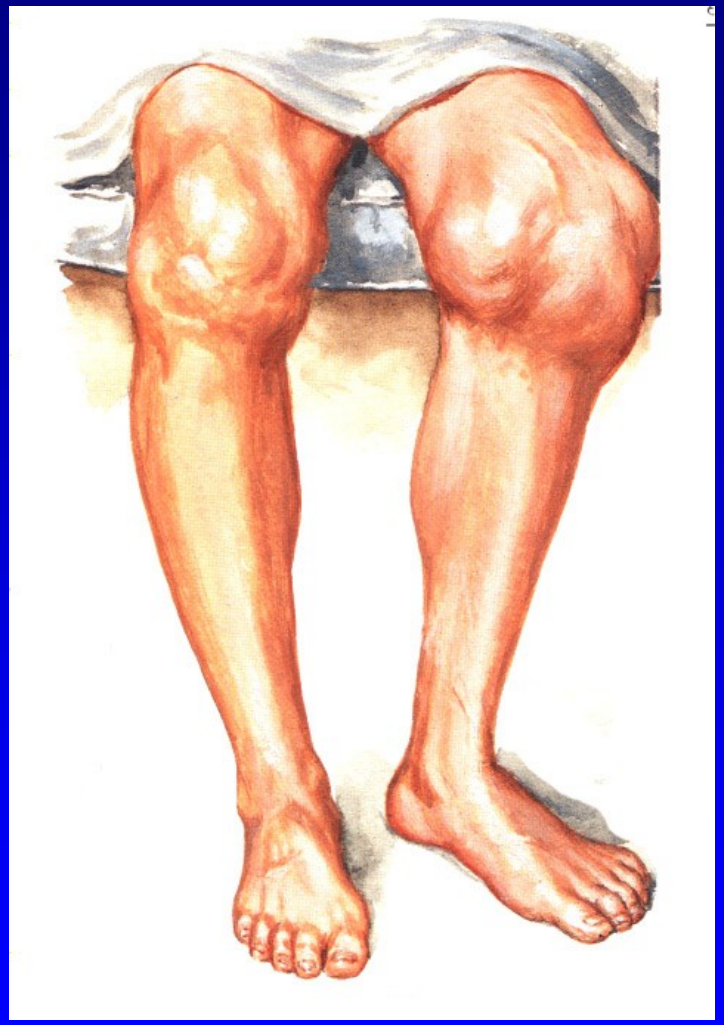
Diferential diagnosis

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

Neurogenic arthropathy



Obr. 30



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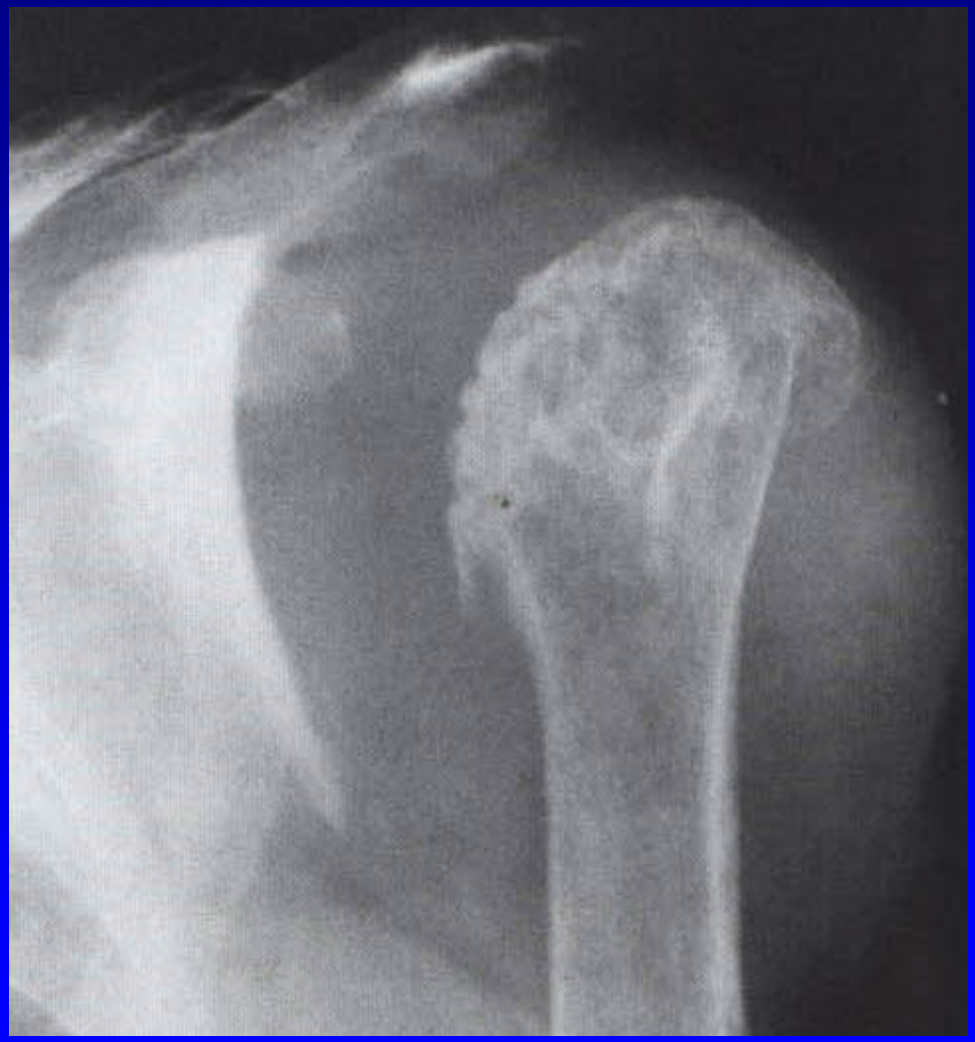
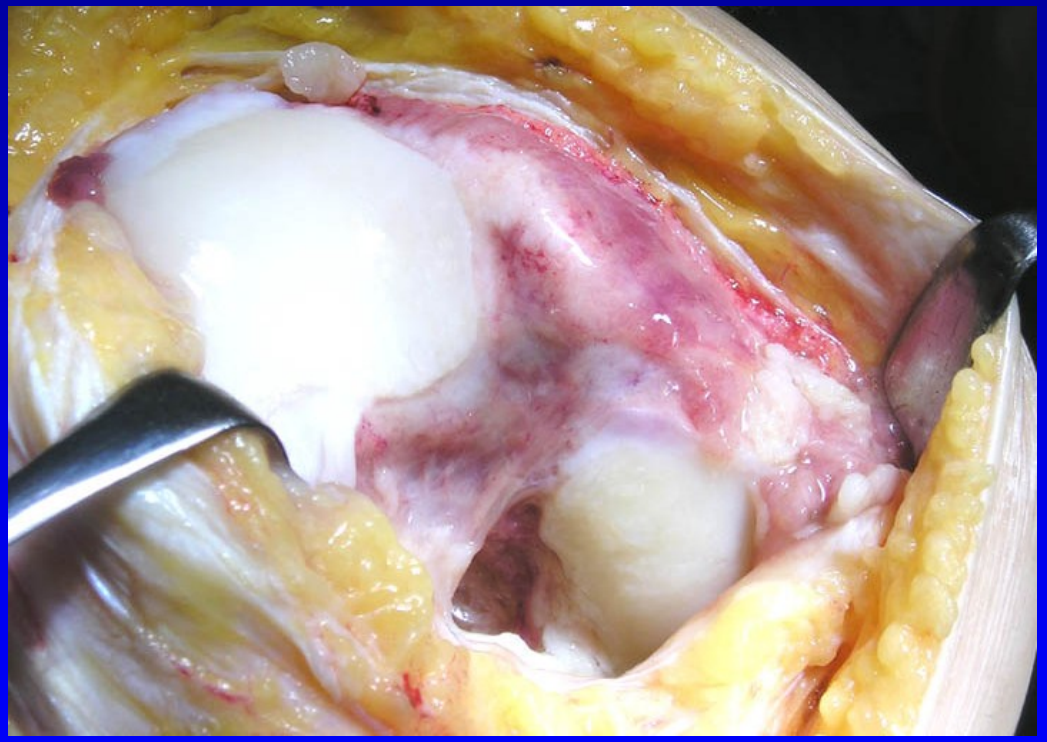
Neurogenic arthropathy

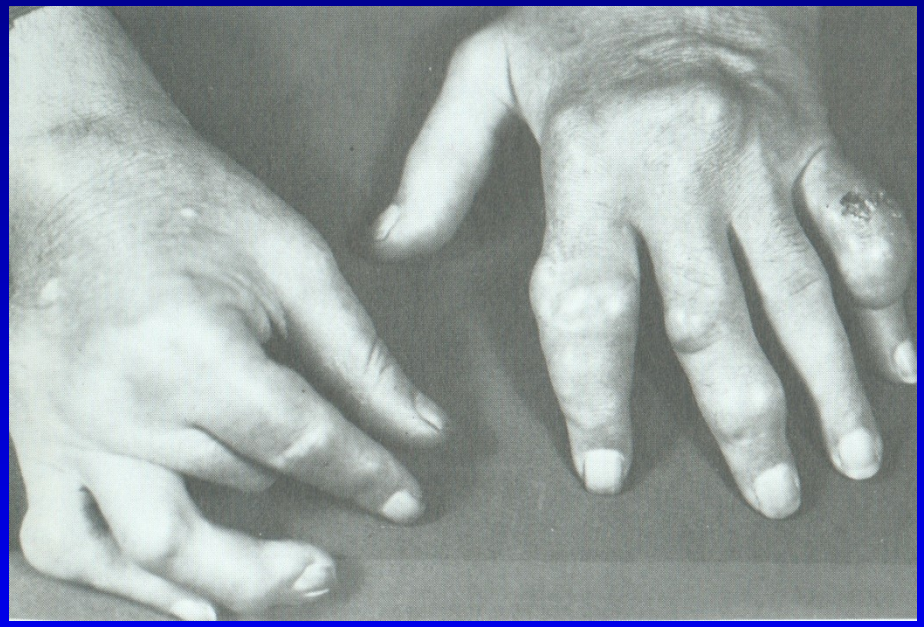


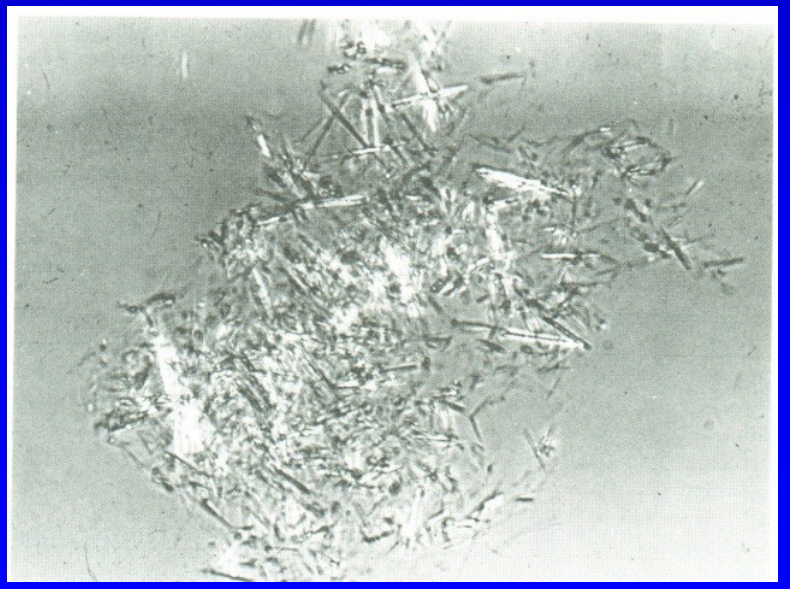
R.A.

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

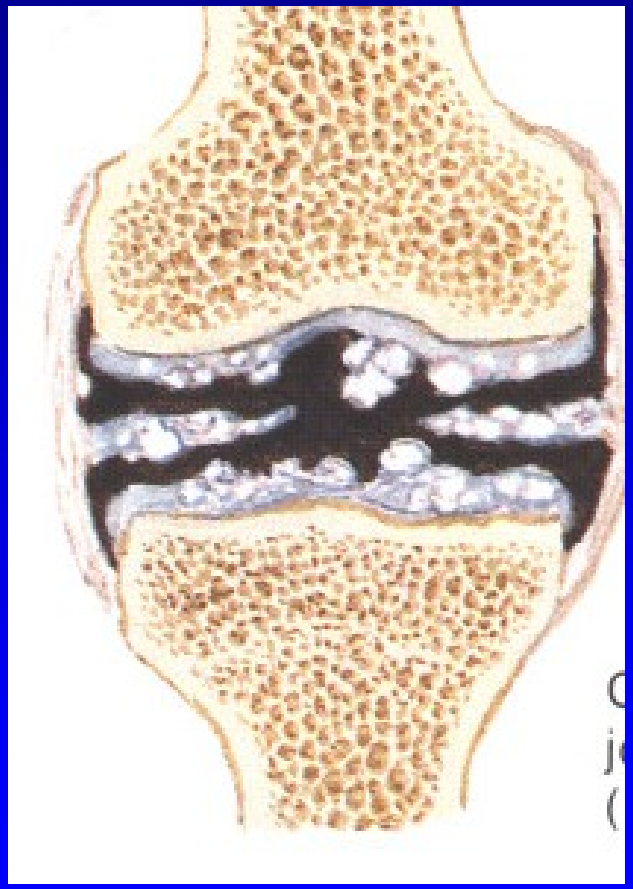


Gout

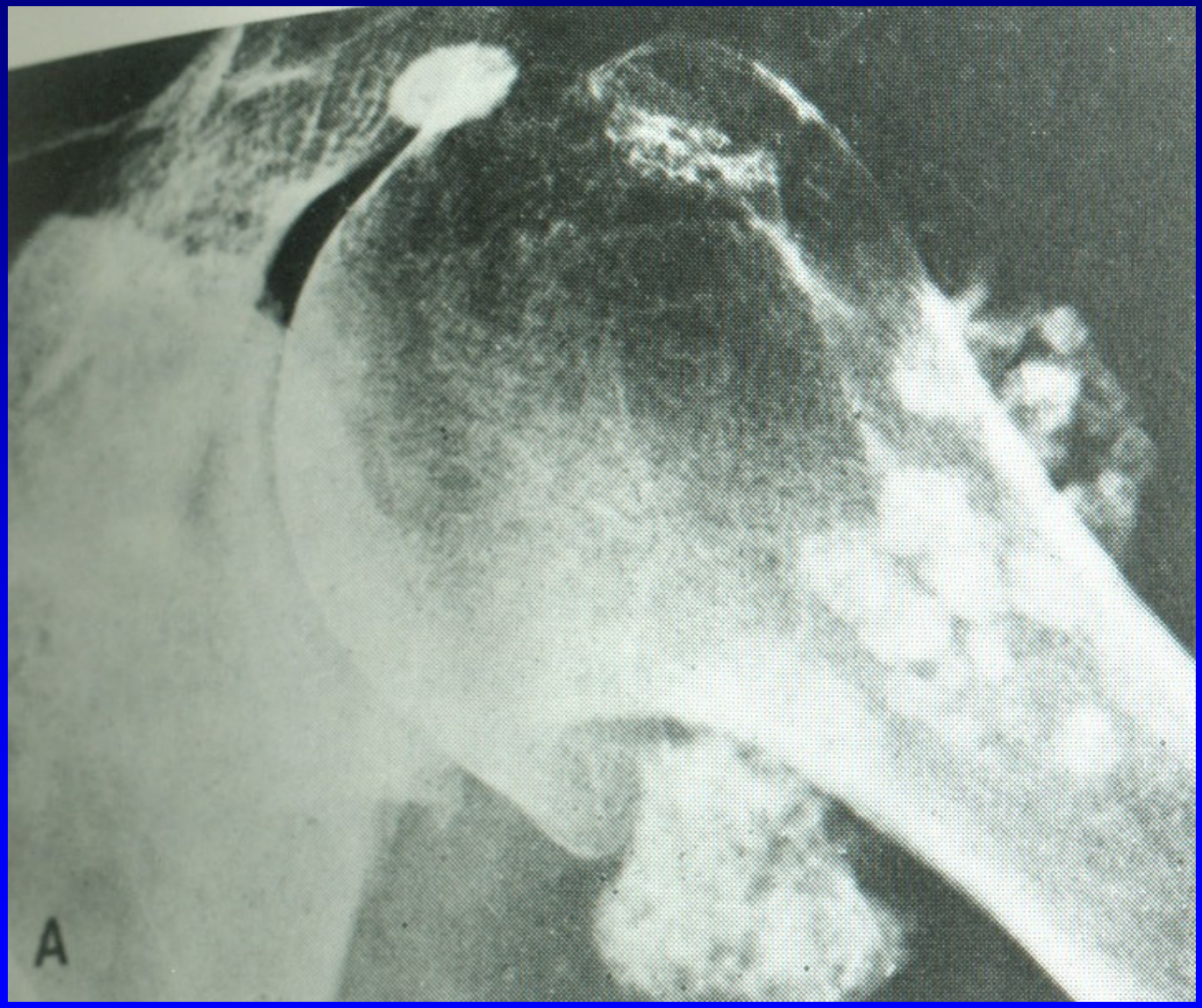




Chondrocalcinosis



Synovial chondromatosis



Septic arthritis