Osteoarthritis

Dr. C. C. Visser
Constituents of hyaline cartilage

- Chondrocytes
- Matrix (extracellular material)
  - Collagen fibres
  - Proteoglycan molecules
Collagen

• Sheets of fibres
• Provides shear resistance
• Orientation:
  – Vertically from bone upward
  – Change direction at surface to run parallel with surface
Proteoglycans

• Large hygroscopic molecules

• Long central chain of hyaluronic acid

• Numerous side chains along its length, each with
  – Central cores of protein
  – Chondroitin sulphate and keratan sulphate side chains

• Pg’s attract water and put collagen under tension
Normal articular cartilage. The swelling pressure of the fully hydrated but compressed aggrecans is counterbalanced by the tight collagen network. Due to this unique composite structure, healthy articular cartilage can resist major pressure and shear forces.
<table>
<thead>
<tr>
<th>No load: low permeability and flow of water</th>
<th>Load: volume decrease, flow of liquid out of the cartilage</th>
<th>Load removed: volume increase, flow of liquid back into the cartilage</th>
</tr>
</thead>
</table>

![Diagram showing water flow within healthy articular cartilage](image_url)
OA AS A BALANCE OF ETIOLOGIC FACTORS AND TISSUE PROCESSES

Abnormal loading of normal tissues or normal loading of abnormal tissues

Ability of tissue to adapt to and respond to insult or repair potential

The OA

Stabilization or repair
Macroscopic changes 1

• Softening and swelling of cartilage
  – Rupture of collagen fibres
  – More water is absorbed by proteoglycans
  – Cartilage is considerably weakened
Osteoarthritic articular cartilage. Damaged or ruptured collagen fibers lead to swelling of the now less compressed aggregans. The cartilage becomes softer, the pressure and shear resistance is diminished, which leads to further damage to the collagen network.
Macroscopic changes 2

• Fibrillation
  – Fine flakes of superficial cartilage become loosened and flake off (and cause mild secondary synovitis which can lead to ‘cold’ effusions)

  – Cracks appear in cartilage: eventually run through full thickness of cartilage
Arthroscopic appearances in OA of the knee joint: fibrillated surface of the cartilage on the medial femoral condyle with minor meniscal damage
Macroscopic changes 3

• Erosion of cartilage
  – Progressive loss of cartilage
  – Ultimate loss of full thickness of cartilage
  – Exposed bone becomes very hard with a polished appearance: ‘eburnation’ of bone (looks like ivory)
Gross superior view of a femoral head from a patient with radiographic stage I OA. This shows an area of complete cartilage loss, with polishing or eburnation of the underlying bone.
A portion of the eburnated surface of an osteoarthritic joint. This demonstrates focal superficial bone and bone marrow necrosis, which is seen macroscopically as the opaque yellow area on the left.
Right: Early OA with area of cartilage loss in the center.

Left: More advanced changes with extensive cartilage loss and exposed underlying bone.
Macroscopic changes 4

• Synovial effusions due to synovitis
  – Joint effusions: usually small and ‘cold’
  – Subchondral cysts: Fluid is forced through clefts in cartilage into the underlying bone, which can be seen on X-ray
An area of cystic degeneration in the subchondral bone of the superior surface of a femoral head. Note the large flat osteophyte on the medial surface.
Macroscopic changes 5

• Osteoblastic stimulation (repair attempt)

  – Underneath the damaged cartilage: subchondral sclerosis on X-ray

  – Around edge of joint forming lip of bone: fibro-osseous osteophytes
A NORMAL VERSUS AN OSTEOPATHRIC SYNOVIAL JOINT

regular normal subchondral bone texture

irregular thickening and remodeling of subchondral bone, with sclerosis and cysts

normal, thick, smooth articular cartilage

thickening, distortion and fibrosis of the capsule

smooth joint margin

fibrillation, loss of volume and degradation of articular cartilage

normal, single cell layered synovium

modest, patchy, chronic synovitis

thin, even capsule

osteoaphytosis and soft tissue growth at joint margin
Macroscopic changes: Summary

• Softening and swelling
• Fibrillation
• Full thickness cracks
• Eburnation
• Subchondral cysts
• Subchondral sclerosis
• Osteophyte formation
## Classification of Osteoarthritis

### Classification by the joints involved

- Monoarticular, oligoarticular or polyarticular (generalized)
- Chief joint site (index joint site) and localization within the joint
  - Hip (superior pole, medial pole or concentric)
  - Knee (medial, lateral, patello-femoral compartments)
  - Hand (interphalangeal joints and/or thumb base)
  - Spine (apophyseal joints or intervertebral disc disease)
  - Others

### Classification into primary and secondary forms of OA

**Primary**

5 idiopathic

**Secondary** indicates that a likely cause can be identified

#### Causes of secondary OA

1. **Metabolic:** examples include,
   - Ochronosis
   - Acromegaly
   - Hemochromatosis
   - Calcium crystal deposition

2. **Anatomic:** examples include,
   - Slipped femoral epiphysis
   - Epiphyseal dysplasias
   - Blount’s disease
   - Perthe’s disease
   - Congenital dislocation of the hip
   - Leg length inequality
   - Hypermobility syndromes

3. **Traumatic:** examples include,
   - Major joint trauma
   - Fracture through a joint or osteonecrosis
   - Joint surgery (e.g. meniscectomy)
   - Chronic injury (occupational arthropathies)

4. **Inflammatory:** examples include,
   - Any inflammatory arthropathy
   - Septic arthritis

### Classification by the presence of specific features

- Inflammatory OA
- Erosive OA
- Atrophic or destructive OA
- OA with chondrocalcinosis
- Others
Epidemiology

- Most common disease of joints over age 65 radiologically (correlates poorly with symptoms)
- Rapid increase in radiologic evidence of OA after age 40
Individual risk factors for development of OA

- Obesity: knee > Hip
- Family history (genetic): polyarticular esp hands
- Trauma
- Hypermobility
- Dysplasia: Hip and knee
- Occupation and sport: excessive and repeated loading of a joint
Clinical features 1

• Pain and tenderness

  – Usually slow onset of discomfort, with gradual and intermitent increase

  – Poor correlation between symptoms and radiologic findings

  – Diffuse/ sharp and stabbing local pain

  – Originates in joint /periarticular soft tissue
POSSIBLE ANATOMIC SITES OF PAIN GENERATION IN OA

- muscle pain
- raised pressure in subchondral bone
- synovitis
- stretching of capsule
- ligament insertion
- tendon insertion
- elevation of periosteum
Clinical features

• Pain and tenderness (cont)

  – Types of pain

    • Mechanical: increases with use of the joint

    • Inflammatory phases

    • Rest pain later on in 50%

    • Night pain in 30% later on
Clinical features 2

• Movement abnormalities

  – ‘Gelling’: stiffness after periods of inactivity, passes over within minutes of using joint again

  – Coarse crepitus: palpate/hear

  – Reduced ROM: capsular thickening and bony changes in joint
Clinical features 3

• Deformities
  – Soft tissue swelling:
    • mild synovitis
    • small effusions
  – Osteophytes
  – Joint laxity
  – Asymmetrical joint destruction leading to angulation
Osteoarthritis of the DIP joints. This patient has the typical clinical findings of advanced OA of the DIP joints, including large firm swellings (Heberden’s nodes), some of which are tender and red due to associated inflammation of the periarticular tissues as well as the joint.
Knee joint effusion
A patient with typical OA of the knees. In the normal standing posture there is a mild varus angulation of the knee joints due to symmetrical OA of the medial tibiofemoral compartments.
Pseudolaxity due to cartilage loss. The joint is not loaded in the first photograph.
Unstable distal interphalangeal joints in OA. The examiner is able to push the joint from side to side due to gross instability, a common finding in late interphalangeal joint OA.
Clinical patterns of involvement
DISTRIBUTION OF OA OF THE HANDS

<table>
<thead>
<tr>
<th>Joint site</th>
<th>Female</th>
<th>4:1</th>
<th>Male</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distal interphalangeal joints</td>
<td>70</td>
<td>70</td>
<td>20</td>
</tr>
<tr>
<td>Proximal interphalangeal joints</td>
<td>35</td>
<td>35</td>
<td>20</td>
</tr>
<tr>
<td>Metacarpophalangeal joints</td>
<td>60</td>
<td>40</td>
<td>20</td>
</tr>
<tr>
<td>Thumb base</td>
<td>5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Wrist</td>
<td>10</td>
<td>10</td>
<td>10</td>
</tr>
</tbody>
</table>
OA of the knees can affect any combination of the three main compartments of each knee. It is usually symmetrical, and the compartments most frequently involved are the medial tibiofemoral and patellofemoral compartments.
Distribution of OA of the hip joint. OA can maximally affect the superior pole, inferior pole, posterior part or other segments of the hip joint. Superior pole involvement, with a tendency for the head of the femur to sublux superolaterally, is the commonest pattern. Involvement of the whole joint (concentric OA) is relatively uncommon.

<table>
<thead>
<tr>
<th>Distribution of OA of the Hip Joint</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Supercloateral (~61%; M &gt; F)</td>
<td>Medial pole (~25%; F &gt; M)</td>
</tr>
<tr>
<td>Young man with OA of one hip joint</td>
<td>Middle aged man with post-traumatic OA of a single lower limb joint</td>
</tr>
<tr>
<td>-----------------------------------</td>
<td>------------------------------------------------------------------</td>
</tr>
<tr>
<td>![Image of young man with OA]</td>
<td>![Image of middle aged man with OA]</td>
</tr>
<tr>
<td>Middle aged lady with OA of the hands and knees</td>
<td>Elderly lady with widespread, destructive OA</td>
</tr>
<tr>
<td>![Image of middle aged lady with OA]</td>
<td>![Image of elderly lady with OA]</td>
</tr>
</tbody>
</table>
Special Investigations

• Blood tests: Normal

• Radiological features:
  – Cartilage loss
  – Subchondral sclerosis
  – Cysts
  – Osteophytes
<table>
<thead>
<tr>
<th>Cause of secondary OA</th>
<th>Laboratory tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>Underlying joint disease</td>
<td>ESR, CRP raised in inflammatory disease; autoantibodies such as RF and ANA in connective tissue disease</td>
</tr>
<tr>
<td>Ochronosis</td>
<td>Presence of homogentisic acid in urine which turns black on exposure to light or on alkalization</td>
</tr>
<tr>
<td>Wilson's disease</td>
<td>Reduced serum caeruloplasmin; increased urinary excretion of copper</td>
</tr>
<tr>
<td>Hemochromatosis</td>
<td>Raised serum iron; raised serum ferritin</td>
</tr>
<tr>
<td>Acromegaly</td>
<td>Raised growth hormone with lack of suppression with a glucose tolerance test</td>
</tr>
<tr>
<td>Hyperparathyroidism</td>
<td>Raised serum calcium; low phosphate; raised parathyroid hormone levels</td>
</tr>
<tr>
<td>Hypothyroidism</td>
<td>Low serum thyroxine; raised thyroid-stimulating hormone</td>
</tr>
<tr>
<td>Gout</td>
<td>Raised serum urate</td>
</tr>
<tr>
<td>Neutropenic disorders</td>
<td></td>
</tr>
<tr>
<td>Tabes dorsalis</td>
<td>Positive VDRL; positive TPHA and FTA-ABS test</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>Abnormal glucose tolerance test</td>
</tr>
</tbody>
</table>
The pathology of osteoarthritis determines the characteristic radiographic features.

Pathology

- Focal loss of articular cartilage
  - If severe enough and sufficiently widespread leads to
  - Radiology
    - Joint space

- Increased activity of subchondral bone
  - When severe results in
  - Subchondral bone sclerosis and cysts

- Marginal lipping and outgrowths of bone
  - When visualized on the unidimensional radiograph
  - Osteophytosis

- Remodeling of joint surfaces and alterations in joint shape
  - Altered long contour

Radiograph of normal knee

Radiograph of osteoarthritic knee
<table>
<thead>
<tr>
<th>Grade</th>
<th>Classification</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Normal</td>
<td>No features of OA</td>
</tr>
<tr>
<td>1</td>
<td>Doubtful</td>
<td>Minute osteophyte, doubtful significance</td>
</tr>
<tr>
<td>2</td>
<td>Minimal</td>
<td>Definite osteophyte, unimpaired joint space</td>
</tr>
<tr>
<td>3</td>
<td>Moderate</td>
<td>Moderate diminution of joint space</td>
</tr>
<tr>
<td>4</td>
<td>Severe</td>
<td>Joint space greatly impaired with sclerosis of subchondral bone</td>
</tr>
</tbody>
</table>
Extensive OA with complete loss of the joint space in a concentric pattern and subchondral bone destruction.
Evolution of erosive OA of the IP. Paired X-rays 3 years apart. One joint has progressed, one has shown evidence of healing and one has fused. This indicates the whole range of changes that can occur.
DIAGNOSIS OF OA AS A CAUSE OF JOINT PAIN

A single painful joint in an older individual

- Use-related pain
  - Inactivity stiffness
- Bony swelling
  - Crepitus
  - Reduced range, with pain at end of range
  - Severe OA on radiograph
    - Normal ESR/CRP
  - Features most likely that the joint pain is due to OA
- Mild early morning stiffness
- Small effusion
  - Tenderness
  - Mild OA on radiograph
    - Chondrocalcinosis
    - Low titer rheumatoid factor or other autoantibodies
  - Features of little help in distinguishing the cause of pain
- Constant pain, prolonged early morning stiffness
- Tense large effusion
  - Hot joint
  - Normal radiograph
    - High ESR/CRP
    - High titer autoantibodies
  - Features suggesting that the pain is unlikely to be due to OA
Management
## Management of Osteoarthritis

### Objectives in management of osteoarthritis

<table>
<thead>
<tr>
<th>Objective</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Education</td>
<td>The patient, relatives and carers should understand the condition and know what they themselves can do to help</td>
</tr>
<tr>
<td>Relieve symptoms</td>
<td>Pain, stiffness and other symptoms of the condition should be controlled as well as possible with minimum risk to the patient</td>
</tr>
<tr>
<td>Minimize handicap</td>
<td>Any consequences on function, and any disability or handicap, should be minimized through appropriate rehabilitative techniques</td>
</tr>
<tr>
<td>Limit progression</td>
<td>Any factors known to be likely to worsen the condition should be avoided, and any practices likely to reduce the risk of progression instituted, with minimum risk, if they do not conflict with the other objectives</td>
</tr>
</tbody>
</table>

### Problems in realization of management objectives

Widespread ignorance and misconceptions about the nature of OA
Lack of understanding of the cause of symptoms
Lack of understanding of disability and handicap in OA
Inability to understand or control disease progression
Treatment Principles

- Education
- Physiotherapy
  - Exercise program
  - Pain relief modalities
- Aids and appliances
- Medical Treatment
- Surgical Treatment
Education

• Nonsystemic nature of disease
• Prevent overloading of joint. Obesity!!
• Appropriate use of treatment modalities
  – Importance of exercise program
  – Aids, appliances, braces
  – Medial treatments
  – Surgical treatments
## PHYSICAL THERAPY AND HYDROTHERAPY IN THE MANAGEMENT OF OA

### Indications for use

- Loss of joint motion without severe joint destruction
- Muscle weakness/wasting, and instability of joint(s)
- Malalignment of joint(s) and/or abnormal joint use (an abnormal gait for example)
- Severe symptoms not relieved by other measures

### Objectives and role of treatment

- Maintain/improve the range of joint motion
- Maintain/increase the strength of muscles acting on the affected joint(s), which also improves stability
- Optimize joint biomechanics to maintain/improve alignment, and reduce any abnormal or excess loading of the joint(s)
- Relieve pain, stiffness and other symptoms
Exercise

• Will not ‘wear the joint out’

• Important for cartilage nutrition

• Some evidence that lack of exercise leads to progression of OA
Exercise

• Encourage full range low impact movements eg swimming, cycling

• Avoid
  – Prolonged loading
  – Activities that cause pain
  – Contact sports
  – High impact sports eg running
Quadriceps exercises for knee OA. Quadriceps exercises are of proven value for pain relief and improving function, and everyone with knee OA should be taught the correct techniques and encouraged to make these exercises a lifetime habit. There is a weight on the ankle.
Use of transcutaneous nerve stimulation (TENS) as an adjunct to other therapy for pain relief at the knee joint. The use of acupuncture, TENS and other local techniques to aid pain relief in difficult cases of OA is often worthwhile.
Aids and appliances

- Braces / splints
- Special shoes/insoles
- Mobility aids
- Aids: dressing, reaching, tap openers, kitchen aids
- Taping of patella in patello femoral OA
Use of a cane, stick or other walking aid. This patient, who has hip OA, has found that she can reduce the pain in her damaged left hip by leaning on the stick in the right hand as she walks. The reduction in loading can be huge, and the effect on symptoms and confidence with walking very beneficial.
The use of shoes and insoles to reduce impact loading on lower limb joints. Modern sports shoes (‘trainers’) often have appropriate insoles. Alternatively, special heel or shoe insoles of sorbithane or viscoelastic materials can be used. They may help relieve pain as well as reducing the peak impact load on the joints during walking.
Medical Treatment

- Simple analgesics: paracetamol, low dose ibuprofen: PRN or regular
- NSAID’s/Coxibs PRN regular
- Intra-articular corticosteroids
- Topical treatment eg NSAID creams, capsaicin
- ‘Chondroprotective agents’
A patient with OA of the carpometacarpal joint of the left thumb undergoing arthrocentesis for injection of a depot corticosteroid preparation. The operator is distracting the patient’s thumb to open up the joint space.
<table>
<thead>
<tr>
<th>Procedure</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arthroscopic washout or ‘tidal irrigation’</td>
<td>Moderate to severe symptoms without severe radiographic changes, useful in diagnosis, mainly used for the knee</td>
</tr>
<tr>
<td>Joint debridement</td>
<td>Moderate to severe symptoms, especially mechanical type, without advanced loss of articular cartilage</td>
</tr>
<tr>
<td>Bony decompression</td>
<td>Occasionally used in early osteonecrosis or to relieve severe pain</td>
</tr>
<tr>
<td>Osteotomy</td>
<td>Pain relief and realignment of joints with severe symptoms and focal damage, but without complete loss of cartilage</td>
</tr>
<tr>
<td>Arthroplasty</td>
<td>Severe symptoms with severe joint damage as well</td>
</tr>
</tbody>
</table>
Joint replacement surgery

• Indications: pain affecting work, sleep, walking and leisure activities

• Complications
  – sepsis
  – loosening
  – lifespan of materials (mechanical failure)
THE PYRAMIDAL APPROACH TO THE TREATMENT OF OA

- **Everyone**
  - Education, counselling, dietary advice if overweight, reassurance
  - Teach appropriate exercises to maintain joint mobility and muscle strength

- **Some patients/more severe cases**
  - Teach joint protection techniques; review function/handicap
  - Assess biomechanics, need for shoe alterations, walking aids, etc.
  - Simple analgesics (regular or ‘on demand’) for pain
  - Short courses of NSAIDs for symptoms
  - Other and physical techniques for symptoms relief
  - Intra-articular steroids
  - Joint lavage, debridement, ‘medical synovectomy’

- **Minority of patients/most severe cases**
  - Major surgical procedures
A healthy joint:

- Bursa
- Muscle
- Cartilage
- Tendon
- Bone
- Joint capsule
- Synovial membrane
- Synovial fluid

Changes in a joint:

- Cartilage destruction
- Loose cartilage particles
- Boney growth (bone spur)
Normal joint

Osteoarthritis

Rheumatoid arthritis

Muscle

Bursa

Synovial membrane

Synovial fluid

Joint capsule

Bone

Tendon

Cartilage

Eroded cartilage

Bone ends rub together

Swollen inflamed Synovial membrane
Osteoarthritic joint

- Inflamed Synovium
- Distorted damaged capsule
- Thick abnormal bone with no remaining cartilage cover
- Loss of remaining cartilage
Osteoarthritis (late stage)

- Fusiform swelling of joints
- Heberden's nodes
Joint affected by osteoarthritis

- Muscle
- Bone
- Tendon
- Capsule (reinforced by ligaments) is thickened
- Synovial fluid
- Cartilage becomes rough and thinner
- Synovial membrane (or synovium) is thickened and inflamed
- Osteophyte
- Bone

Joint expanded to show detail
Osteoarthritis

Healthy knee joint

Hypertrophy and spurring of bone and erosion of cartilage